## Question #:269

**CLINICAL SCENERIO**: A 72-year-old male with a 4-day history of shortness of breath, productive cough, and a history of type 2 diabetes, hypertension, and hypercholesterolaemia presents drowsy and confused. Examination reveals dry mucous membranes, hypotension, tachycardia, a temperature of 37.6°C, and respiratory rate of 20/min. Course crepitations were found at the right base and his pulse was thready with a capillary refill of 3 seconds. Capillary blood glucose was HI. Venous blood sample results are provided.

**QUESTION LINE**: What treatment would you initiate first?

**OPTIONS**: - a. 0.9% normal saline - b. 0.45 % normal saline - c. Hartmann’s solution - d. Intravenous insulin - e. 5% dextrose

**CORRECT-CHOICE LINE**: A.

**REASONING**: The patient likely has hyperosmolar hyperglycaemic state (HHS) due to hyperglycemia and hypovolemia. Initial treatment is 0.9% normal saline to address hypovolemia, as patients in HHS are sodium depleted. Insulin is not started immediately in the absence of significant ketonemia.

**>>DESCRIPTION**: A 72-year-old male with a 4-day history of shortness of breath, cough, and history of type 2 diabetes presents drowsy, confused, and with relevant examination findings. Laboratory results are provided. What is the initial treatment?

**>>OPTIONS**: a) 0.45 % normal saline b) 0.9% normal saline c) 5% dextrose d) Hartmann’s solution e) Intravenous insulin

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: 0.9% normal saline

**>>REASONING**: 0.9% normal saline is the first-line treatment for hyperosmolar hyperglycemic state (HHS) to address hypovolemia. Other options are incorrect because they are not the initial treatment of choice for HHS.

## Question #:22

**CLINICAL SCENERIO**: A 75-year-old man with a history of high blood pressure, type 2 diabetes and hypercholesterolaemia was admitted to the emergency department with confusion. His daughter states that this has come on slowly over the last week and prior to this he had no memory problems. He currently takes metformin, ramipril, amlodipine and atorvastatin.

On examination, he smells strongly of urine and his mucous membranes appear dry. His abbreviated mental test score is 7 out of 10 and he is oriented in person but not in place or time. His heart rate is 95 per minute and his blood pressure is 105/62 mmHg. His chest is clear and has a soft ejection systolic murmur which does not radiate. His jugular venous pressure is not visible and he has mild ankle oedema. He has diffuse tenderness in the lower abdomen with no peritonism and normal bowel sounds. He has no focal neurology.

Investigation results are as follows:

Chest x-ray: Clear lung fields.

Urine dip:

| Glucose | +++ |
| --- | --- |
| Blood | + |
| Protein | + |
| Leucocyte | + |

| s |
| --- |
| Nitrites |
| Ketones |

## Venous blood gas:

| pH | 7.43 |
| --- | --- |
| BE | - 1.5 mmol/l |
| HCO3 | 23 mmol/l |
| Glucose | 34 mmol/l |
| Lactate | 2.5 mmol/l |

## Full blood count:

| Hb | 120 g/l |
| --- | --- |
| Platelet s | 445 \* 10 9 /l |
| WBC | 13 \* 10 9 /l |

Renal function:

Na +

151 mmol/l

K +

5 mmol/l

Urea

10 mmol/l

Creatinin e

137 µmol/l

Glucose

32 mmol/l

Ketones

2 mmol/l

**QUESTION LINE**: Which would be the most appropriate initial resuscitation measure?

**OPTIONS**: - a) 0.45% saline - b) 0.9% saline - c) Fixed rate insulin and 0.9% saline - d) Hartmann’s - e) Sliding scale insulin and 0.9% saline

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This gentleman has hyperosmolar hyperglycaemic state (HHS), likely precipitated by urinary tract infection and his pre-existing diabetes.

According to the Joint British Diabetes Society Guidelines for HHS, 0.9% saline is the recommended initial resuscitation fluid, aiming for 3-6 litres positive at 12 hours. This should only be switched to 0.45% saline if osmolality is not declining despite positive fluid balance. Fixed rate insulin should only be added if glucose fails to fall with fluid.

Reference: Joint British Diabetes Societies Inpatient Care Group. The management of hyperosmolar hyperglycaemic state (HHS) in adults with diabetes. 2012.

**>>DESCRIPTION**: A 75-year-old man with hypertension, type 2 diabetes, and hypercholesterolemia presents with a week-long history of confusion. He takes metformin, ramipril, amlodipine, and atorvastatin. Examination reveals dry mucous membranes, disorientation, a heart rate of 95, blood pressure of 105/62 mmHg, mild ankle edema, and diffuse lower abdominal tenderness. Labs show elevated glucose (34 mmol/l), ketones (2 mmol/l), Na+ (151 mmol/l) and WBC (13 \* 10 9 /l), along with glucosuria, ketonuria, and proteinuria.

**>>OPTIONS**: a) 0.45% saline b) 0.9% saline c) Fixed rate insulin and 0.9% saline d) Hartmann’s e) Sliding scale insulin and 0.9% saline

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: 0.9% saline

**>>REASONING**: The patient likely has hyperosmolar hyperglycaemic state (HHS) precipitated by a urinary tract infection. Initial resuscitation should be with 0.9% saline per Joint British Diabetes Society Guidelines. Fixed rate insulin should only be added if glucose fails to fall with fluid.

## Question #:28

**CLINICAL SCENERIO**: A 75-year-old man with a history of high blood pressure, type 2 diabetes and hypercholesterolaemia was admitted to the emergency department with confusion. His daughter states that this has come on slowly over the last week and prior to this he had no memory problems. He currently takes metformin, ramipril, amlodipine and atorvastatin.

On examination, he smells strongly of urine and his mucous membranes appear dry. His abbreviated mental test score is 7 out of 10 and he is oriented in person but not in place or time. His heart rate is 95 per minute and his blood pressure is 105/62 mmHg. His chest is clear and has a soft ejection systolic murmur which does not radiate. His jugular venous pressure is not visible and he has mild ankle oedema. He has diffuse tenderness in the lower abdomen with no peritonism and normal bowel sounds. He has no focal neurology.

Investigation results are as follows:

Chest x-ray: Clear lung fields.

Urine dip:

| Glucose | +++ |
| --- | --- |
| Blood | + |
| Protein | + |
| Leucocyte | + |

| s |
| --- |
| Nitrites |
| Ketones |

## Venous blood gas:

| pH | 7.43 |
| --- | --- |
| BE | - 1.5 mmol/l |
| HCO3 | 23 mmol/l |
| Glucose | 34 mmol/l |
| Lactate | 2.5 mmol/l |

## Full blood count:

| Hb | 120 g/l |
| --- | --- |
| Platelet s | 445 \* 10 9 /l |
| WBC | 13 \* 10 9 /l |

Renal function:

Na +

151 mmol/l

K +

5 mmol/l

Urea

10 mmol/l

Creatinin e

137 µmol/l

Glucose

32 mmol/l

Ketones

2 mmol/l

**QUESTION LINE**: Which would be the most appropriate initial resuscitation measure?

**OPTIONS**: - a) 0.45% saline - b) 0.9% saline - c) Fixed rate insulin and 0.9% saline - d) Hartmann’s - e) Sliding scale insulin and 0.9% saline

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This gentleman has hyperosmolar hyperglycaemic state (HHS), likely precipitated by urinary tract infection and his pre-existing diabetes.

According to the Joint British Diabetes Society Guidelines for HHS, 0.9% saline is the recommended initial resuscitation fluid, aiming for 3-6 litres positive at 12 hours. This should only be switched to 0.45% saline if osmolality is not declining despite positive fluid balance. Fixed rate insulin should only be added if glucose fails to fall with fluid.

Reference: Joint British Diabetes Societies Inpatient Care Group. The management of hy

**>>DESCRIPTION**: A 75-year-old man with hypertension, type 2 diabetes, and hypercholesterolemia presents with a week-long history of confusion. Examination reveals dry mucous membranes, disorientation, tachycardia (95 bpm), hypotension (105/62 mmHg), mild ankle edema, abdominal tenderness, elevated glucose (32-34 mmol/l), hypernatremia (151 mmol/l), and leukocytosis (13 \* 10^9 /l). Urine dip shows glucosuria, blood, protein and leucocytes.

**>>OPTIONS**: a) 0.45% saline b) 0.9% saline c) Fixed rate insulin and 0.9% saline d) Hartmann’s e) Sliding scale insulin and 0.9% saline

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: 0.9% saline

**>>REASONING**: The patient likely has hyperosmolar hyperglycemic state (HHS) precipitated by a urinary tract infection. Initial resuscitation should be with 0.9% saline as per the Joint British Diabetes Society Guidelines. Insulin should be added only if glucose fails to fall with fluid resuscitation.

## Question #:341

**CLINICAL SCENERIO**: A 75-year-old man with a history of high blood pressure, type 2 diabetes and hypercholesterolaemia was admitted to the emergency department with confusion. His daughter states that this has come on slowly over the last week and prior to this he had no memory problems. He currently takes metformin, ramipril, amlodipine and atorvastatin.

On examination, he smells strongly of urine and his mucous membranes appear dry. His abbreviated mental test score is 7 out of 10 and he is oriented in person but not in place or time. His heart rate is 95 per minute and his blood pressure is 105/62 mmHg. His chest is clear and has a soft ejection systolic murmur which does not radiate. His jugular venous pressure is not visible and he has mild ankle oedema. He has diffuse tenderness in the lower abdomen with no peritonism and normal bowel sounds. He has no focal neurology.

Investigation results are as follows:

Chest x-ray: Clear lung fields.

Urine dip:

| Glucose | +++ |
| --- | --- |
| Blood | + |
| Protein | + |
| Leucocyte s | + |
| Nitrites | + |
| Ketones | + |

Venous blood gas:

| pH | 7.43 |
| --- | --- |
| BE | - 1.5 mmol/l |
| HCO3 | 23 mmol/l |
| Glucose | 34 mmol/l |
| Lactate | 2.5 mmol/l |

## Full blood count:

| Hb | 120 g/l |
| --- | --- |
| Platelet s | 445 \* 10 9 /l |
| WBC | 13 \* 10 9 /l |

## Renal function:

| Na + | 151 mmol/l |
| --- | --- |
| K + | 5 mmol/l |
| Urea | 10 mmol/l |
| Creatinin e | 137 µmol/l |
| Glucose | 32 mmol/l |
| Ketones | 2 mmol/l |

**QUESTION LINE**: Which would be the most appropriate initial resuscitation measure?

**OPTIONS**: - f) 0.45% saline - g) 0.9% saline - h) Fixed rate insulin and 0.9% saline - i) Hartmann’s - j) Sliding scale insulin and 0.9% saline

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This gentleman has hyperosmolar hyperglycaemic state (HHS), likely precipitated by urinary tract infection and his pre-existing diabetes.

According to the Joint British Diabetes Society Guidelines for HHS, 0.9% saline is the recommended initial resuscitation fluid, aiming for 3-6 litres positive at 12 hours. This should only be switched to 0.45% saline if osmolality is not declining despite positive fluid balance. Fixed rate insulin should only be added if glucose fails to fall with fluid.

Reference: Joint British Diabetes Societies Inpatient Care Group. The management of hyperosmolar hyperglycaemic state (HHS) in adults with diabetes. 2012.

**>>DESCRIPTION**: A 75-year-old man with type 2 diabetes, hypertension, and hypercholesterolemia presents with a week-long history of confusion. Medications include metformin, ramipril, amlodipine, and atorvastatin. Examination shows signs of dehydration (dry mucous membranes, HR 95, BP 105/62 mmHg, AMTS 7/10, mild ankle oedema, diffuse lower abdominal tenderness) and strong urine smell. Urine dip is positive for glucose, blood, protein, leukocytes, nitrites, and ketones. Lab results reveal: Na+ 151 mmol/l, K+ 5 mmol/l, Urea 10 mmol/l, Creatinine 137 µmol/l, Glucose 32-34 mmol/l, Ketones 2 mmol/l, pH 7.43, HCO3 23 mmol/l, WBC 13 x 10^9/l. The clinical picture indicates hyperosmolar hyperglycaemic state (HHS) precipitated by likely UTI.

**>>OPTIONS**: a) 0.45% saline b) 0.9% saline c) Fixed rate insulin and 0.9% saline d) Hartmann’s e) Sliding scale insulin and 0.9% saline

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: 0.9% saline

**>>REASONING**: The patient presents with hyperosmolar hyperglycaemic state (HHS), likely triggered by a urinary tract infection. Per Joint British Diabetes Society Guidelines for HHS, 0.9% saline is the recommended initial resuscitation fluid. 0.45% saline is considered only if osmolality does not decline with 0.9% saline. Insulin (fixed-rate or sliding scale) is not the initial treatment; it is added only if glucose levels remain high after fluid resuscitation.

## Question #:192

**CLINICAL SCENERIO**: An 18-year-old man with congenital hypoparathyroidism, treated with vitamin D and calcium supplementation, presents with elevated creatinine and a history of renal stones.

**QUESTION LINE**: Which of the following is the most appropriate target with respect to serum calcium?

**OPTIONS**: - a) 1.85 mmol/l - b) 2.10 mmol/l - c) 2.25 mmol/l - d) 2.60 mmol/l - e) 2.85 mmol/l

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This 18-year-old man is at risk of symptomatic hypocalcaemia because of congenital hypoparathyroidism, although elevating his calcium too much with vitamin D supplementation runs the risk of symptomatic renal stones. As such guidelines recommend aiming towards a calcium just below the lower end of the normal range and 2.10 is an appropriate target for serum calcium.

1.85 mmol/l is considered too low and puts the patient at risk of muscle weakness, paresthesias, tetany and cardiac arrhythmia. Maintenance of calcium either at the upper end of the normal range, (2.60 mmol/l), or 2.85 mmol/l (above the normal range), is associated with symptomatic renal stones and progressive deterioration in renal function.

**>>DESCRIPTION**: An 18-year-old man with congenital hypoparathyroidism and a history of renal stones requires a target serum calcium level.

**>>OPTIONS**: a) 1.85 mmol/l b) 2.10 mmol/l c) 2.25 mmol/l d) 2.60 mmol/l e) 2.85 mmol/l

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: 2.10 mmol/l

**>>REASONING**: The appropriate target serum calcium is 2.10 mmol/l, balancing the risk of hypocalcemia against the risk of renal stones. Levels too low (1.85 mmol/l) risk hypocalcemic symptoms, while levels too high (2.60 or 2.85 mmol/l) promote renal stones and renal function decline.

## Question #:33

**CLINICAL SCENERIO**: A 19-year-old man with vomiting and abdominal pain was diagnosed with diabetic ketoacidosis and type 1 diabetes mellitus. He was managed as an inpatient for five days and educated on managing his diabetes and insulin at home. He is concerned about his target plasma glucose after eating.

**QUESTION LINE**: What is the recommended target after eating to be achieved by home monitoring?

**OPTIONS**: a) 3-6mmol/litr b) 5-10mmol/litre c) 5-9mmol/litre d) 7-12mmol/litre e) 2-9 mmol/litre

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The correct answer is 5-9mmol/litre. NICE recommends that people with type 1 diabetes should aim for 5-7mmol/litre on waking, 4-7mmol/litre before meals and 5-9mmol/litre 90 minutes after eating. Frequent testing is very important in patients starting with insulin therapy to avoid both high and low sugar levels.

**>>DESCRIPTION**: A 19-year-old man diagnosed with DKA and type 1 DM is concerned about his target plasma glucose after eating, despite education on diabetes management.

**>>OPTIONS**: a) 2-9 mmol/litre b) 3-6mmol/litr c) 5-10mmol/litre d) 5-9mmol/litre e) 7-12mmol/litre

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: 5-9mmol/litre

**>>REASONING**: NICE recommends a target glucose of 5-9mmol/litre 90 minutes after eating for individuals with type 1 diabetes. Other options represent incorrect target ranges for post-meal glucose.

## Question #:10

**CLINICAL SCENERIO**: A 47-year-old woman with a history of paroxysmal atrial fibrillation and Graves’ disease, undergoes thyroidectomy, but residual thyroid tissue and symptoms recur, including palpitations from atrial fibrillation. She is referred for radioiodine therapy. Three days before the therapy, she is admitted with acute abdominal pain, and an urgent CT scan rules out any acute pathology. When is the earliest radioiodine therapy can take place?

**QUESTION LINE**: With regards to the radioiodine therapy, when is the earliest that this can take place?

**OPTIONS**: a) 3 days b) 2 weeks c) 8 weeks d) 16 weeks e) 26 weeks

**CORRECT-CHOICE LINE**: c

**REASONING**: Radioiodine therapy should be avoided until 8 weeks following CT contrast administration

Iodine in intravenous contrast interferes with the administration of radioiodine. As the levels of iodine in contrast are several hundred-fold higher than the recommended daily allowance, this may be retained by the body for months. As such, it can interfere with therapeutic uptake of radioiodine by the thyroid gland. Various studies have demonstrated a 6-8 week time period is the minimum to wait before administering radioiodine, following CT contrast. 8 weeks is therefore the correct answer.

Going ahead with the procedure as planned, 3 days after this CT scan with contrast is therefore too soon. The contrast would interfere with the radioiodine.

Similarly, 2 weeks is still too soon to go ahead with the administration.

16 weeks is an unnecessarily long amount of time to wait - 8 weeks is sufficient according to current studies.

Similarly, 26 weeks is longer than needed - the question asks what the earliest time of administration should be.

**>>DESCRIPTION**: A woman with atrial fibrillation and Graves’ disease, post-thyroidectomy, requires radioiodine therapy. A CT scan with contrast is performed. When is the earliest radioiodine therapy can be administered?

**>>OPTIONS**: a) 3 days b) 16 weeks c) 2 weeks d) 8 weeks e) 26 weeks

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: 8 weeks

**>>REASONING**: Radioiodine therapy should be delayed for 8 weeks after CT contrast due to iodine interference. 3 days and 2 weeks are too soon. 16 and 26 weeks are unnecessarily long.

## Question #:234

**CLINICAL SCENERIO**: A 9-year-old boy with secondary sexual characteristics since age 7, coarse voice, facial hair, enlarged testicles, acne, and adult body odor presents with hypertension and hypokalemia. Blood tests reveal elevated testosterone, and normal LH/FSH.

**QUESTION LINE**: From the listed options, which is the most likely diagnosis?

**OPTIONS**: a. Brain tumour b. Hypothyroidism c. Liddle’s syndrome d. McCune-Albright syndrome e. 11-beta hydroxylase deficiency

**CORRECT-CHOICE LINE**: e

**REASONING**: CAH due to 11-beta hydroxylase deficiency can cause apparent mineralocorticoid excess syndrome (AMES) resulting in hypertension and hypokalemia. The elevated serum testosterone but normal LH and FSH suggest a peripheral cause of precocious puberty.

**>>DESCRIPTION**: A 9-year-old boy presents with precocious puberty, hypertension, hypokalemia, elevated testosterone, and normal LH/FSH.

**>>OPTIONS**: a. Brain tumour b. 11-beta hydroxylase deficiency c. Hypothyroidism d. Liddle’s syndrome e. McCune-Albright syndrome

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: 11-beta hydroxylase deficiency

**>>REASONING**: The most likely diagnosis is 11-beta hydroxylase deficiency due to precocious puberty, hypertension, and hypokalemia. Other options are less likely due to the presenting signs/symptoms and laboratory findings.

## Question #:154

**CLINICAL SCENERIO**: A 19-year-old woman with 11beta-hydroxylase deficiency and hypertension, treated with ramipril and indapamide, presents for review. The deficiency was identified at birth due to clitoromegaly.

**QUESTION LINE**: Which of the following is likely to be raised most markedly?

**OPTIONS**: a) 17-OH pregnenolone b) Oestradio c) 11-deoxycortisol d) 17-OH progesterone e) Oestrone

**CORRECT-CHOICE LINE**: c

**REASONING**: 11 beta-hydroxylase converts 11-deoxycorticosterone and 11-deoxycortisol to corticosterone and cortisol. Deficiency leads to accumulation of these steroids. 11-deoxycortisol is most significantly raised.

**>>DESCRIPTION**: A 19-year-old woman with 11beta-hydroxylase deficiency and hypertension presents for review. Which hormone is most markedly elevated?

**>>OPTIONS**: a) 11-deoxycortisol b) 17-OH progesterone c) 17-OH pregnenolone d) Oestradio e) Oestrone

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: 11-deoxycortisol

**>>REASONING**: In 11-beta-hydroxylase deficiency, the conversion of 11-deoxycortisol to cortisol is impaired, causing 11-deoxycortisol levels to increase significantly.

## Question #:123

**CLINICAL SCENERIO**: A 38-year-old woman, 10 weeks pregnant, presents with increasing fatigue, weight gain, feeling cold, and constipation only responsive to high-dose laxatives. She has a history of well-controlled hypothyroidism on 50 micrograms of levothyroxine. Examination reveals thin hair, waxy skin, mild peripheral oedema, and a larger body habitus. Blood tests show TSH of 5.0 mU/L and free T4 of 9.8 pmol/L.

**QUESTION LINE**: What is the best next management option?

**OPTIONS**: - a) 100 micrograms levothyroxine - b) 25 micrograms levothyroxine - c) 75 micrograms levothyroxine - d) Continue 50 micrograms levothyroxine - e) Stop the levothyroxine

**CORRECT-CHOICE LINE**: 75 micrograms levothyroxine is the correct answer.

**REASONING**: Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

75 micrograms levothyroxine is the correct answer. This is because during pregnancy (as early as 4-6 weeks) the amount of thyroglobulin binding protein increases, thus there is less free thyroid hormone in the bloodstream exacerbating this woman’s state of hypothyroidism. Increasing the dose of 50 by 50% to 75 is usually sufficient as a measure to induce a state of euthyroid.

25 micrograms levothyroxine is incorrect as this would worsen the state of hypothyroidism.

Continue 50 micrograms levothyroxine is incorrect as this would not address the issue of the state of hypothyroidism on the current dose.

Stop the levothyroxine is incorrect as this would almost certainly exacerbate the state of hypothyroidism yet further.

100 micrograms levothyroxine is incorrect as this would be an increase on the dose by 100%; at this stage most likely too much and may cause a state of hyperthyroidism.

**>>DESCRIPTION**: A 38-year-old woman, 10 weeks pregnant, presents with fatigue, weight gain, cold intolerance and constipation. She has hypothyroidism, managed with 50 mcg levothyroxine. Exam: thin hair, waxy skin, mild oedema. Labs: TSH 5.0 mU/L, free T4 9.8 pmol/L.

**>>OPTIONS**: a) 100 micrograms levothyroxine b) 25 micrograms levothyroxine c) 50 micrograms levothyroxine d) 75 micrograms levothyroxine e) Stop the levothyroxine

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: 75 micrograms levothyroxine

**>>REASONING**: During pregnancy, increased thyroglobulin binding protein reduces free thyroid hormone, exacerbating hypothyroidism. A 50% dose increase to 75 mcg levothyroxine is appropriate. Lower doses or stopping medication would worsen hypothyroidism, while 100 mcg may cause hyperthyroidism.

## Question #:295

**CLINICAL SCENERIO**: A 19-year-old man presents to the emergency department via ambulance with a 2-day history of loss of appetite, abdominal pain, and significant weight loss over six months. He is tachypnoeic, tachycardic, and has a capillary refill time of six seconds. Initial investigations reveal elevated urea, creatinine, sodium, potassium, and blood glucose/ketones. Venous blood gas shows severe acidosis. ECG shows sinus tachycardia. What is the correct immediate management?

**QUESTION LINE**: What is the correct immediate management for this patient?

**OPTIONS**: a) 1000 mg calcium gluconate b) 10 mL 8.4 % sodium bicarbonate c) 1000 mL 0.9 % saline d) 1000 ml 0.45 % saline e) Intravenous insulin bolus

**CORRECT-CHOICE LINE**: c

**REASONING**: The patient has clear biochemical evidence of diabetic keto-acidosis. The correct immediate management is to give a litre of 0.9 % saline to restore intravascular volume. An initial bolus dose of intravenous insulin is not recommended. Bicarbonate infusion are not routinely recommended in the treatment of DKA. Given the absence of ECG changes of hyperkalaemia in this patient, an infusion of calcium gluconate is not required.

**>>DESCRIPTION**: A 19-year-old man presents to the emergency department with a 2-day history of loss of appetite, abdominal pain, and significant weight loss. Examination reveals tachycardia, and prolonged capillary refill. Investigations show elevated glucose and ketones, and severe acidosis. What is the correct immediate management?

**>>OPTIONS**: a) 1000 mg calcium gluconate b) 10 mL 8.4 % sodium bicarbonate c) 1000 mL 0.9 % saline d) 1000 ml 0.45 % saline e) Intravenous insulin bolus

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: 1000 mL 0.9 % saline

**>>REASONING**: The correct immediate management is 1000 mL 0.9% saline to restore intravascular volume in the context of diabetic ketoacidosis. Insulin bolus and bicarbonate are not the initial steps. Calcium gluconate is not indicated.

## Question #:111

**CLINICAL SCENERIO**: A 22-year-old woman attends the clinic with a 12-month history of infertility and irregular periods. She also complains of excess hair growth over her face and chest. Observations are as follows: heart rate 82 beats per minute, blood pressure 115/75 mmHg, respiratory rate 16 breaths per minute, SpO2 98% (on air), temperature 37.2ºC. Blood results are as follows:

| Hb | 125 g/L | Male: (135-180) Female: (115 - 160) |
| --- | --- | --- |
| Platelets | 228 \* 10 9 /L | (150 - 400) |
| WBC | 8.2 \* 10 9 /L | (4.0 - 11.0) |
| Na + | 132 mmol/L | (135 - 145) |
| K + | 5.1 mmol/L | (3.5 - 5.0) |
| Urea | 6.2 mmol/L | (2.0 - 7.0) |
| Creatinine | 74 µmol/L | (55 - 120) |
| CRP | 2 mg/L | (< 5) |
| Glucose | 5.2 mmol/L | (4-7) |
| Early morning cortisol | 140 nmol/l | (>350) |

**QUESTION LINE**: What investigation is required?

**OPTIONS**: - a) ACTH stimulation testing - b) Dexamethasone suppression test - c) Karyotyping - d) Serum prolactin - e) Transvaginal ultrasound

**CORRECT-CHOICE LINE**: Correct answer Is a.

**REASONING**: ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

ACTH stimulation testing is correct. The clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH). This form is milder, more common, and often diagnosed in later childhood or early adulthood. Usually, women with non-classic CAH present with signs of excess androgens (i.e., excess body hair, early puberty, irregular periods). Hypoaldosteronism (e.g. hyponatraemia, hyperkalaemia, metabolic acidosis) can also occur. The biochemical features and low early morning cortisol in this case are highly suggestive of hypoaldosteronism.

Dexamethasone suppression test is incorrect. Although Cushing’s disease remains within the differential diagnosis, there are no other clinical (e.g. hypertension, proximal myopathy, weight gain, easy bruising) or biochemical features (e.g. hyperglycaemia, hypernatraemia, hypokalaemia) to support this diagnosis. Furthermore, the low early morning cortisol makes this an unlikely diagnosis.

Karyotyping is incorrect. Although Turner’s syndrome can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Serum prolactin is incorrect. Although hyperprolactinaemia can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Transvaginal ultrasound is incorrect. Although ovarian tumours can result in androgen excess, this usually results in virilization which is not present in this case. Furthermore, hypoaldosteronism would not be expected to occur with ovarian pathology.

**>>DESCRIPTION**: A 22-year-old woman with infertility, irregular periods, and excess hair growth. Examination: HR 82, BP 115/75, RR 16, SpO2 98%, T 37.2ºC. Labs: Na+ 132 mmol/L, K+ 5.1 mmol/L, Cortisol 140 nmol/l.

**>>OPTIONS**: a) ACTH stimulation testing b) Dexamethasone suppression test c) Karyotyping d) Serum prolactin e) Transvaginal ultrasound

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: ACTH stimulation testing

**>>REASONING**: ACTH stimulation testing is correct because clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH), with signs of excess androgens and hypoaldosteronism. Dexamethasone suppression is incorrect as clinical/biochemical features don’t support Cushing’s. Karyotyping and serum prolactin are less likely given hypoaldosteronism points to CAH. Transvaginal ultrasound is less likely as virilization is absent.

## Question #:115

**CLINICAL SCENERIO**: A 22-year-old woman attends the clinic with a 12-month history of infertility and irregular periods. She also complains of excess hair growth over her face and chest. Observations are as follows: heart rate 82 beats per minute, blood pressure 115/75 mmHg, respiratory rate 16 breaths per minute, SpO2 98% (on air), temperature 37.2ºC.

Blood results are as follows:

| Hb | 125 g/L | Male: (135-180) Female: (115 - 160) |
| --- | --- | --- |
| Platelets | 228 \* 10 9 /L | (150 - 400) |

| WBC | 8.2 \* 10 9 /L | (4.0 - 11.0) |
| --- | --- | --- |
| Na + | 132 mmol/L | (135 - 145) |
| K + | 5.1 mmol/L | (3.5 - 5.0) |
| Urea | 6.2 mmol/L | (2.0 - 7.0) |
| Creatinine | 74 µmol/L | (55 - 120) |
| CRP | 2 mg/L | (< 5) |
| Glucose | 5.2 mmol/L | (4-7) |
| Early morning cortisol | 140 nmol/l | (>350) |

**QUESTION LINE**: What investigation is required?

**OPTIONS**: - a) ACTH stimulation testing - b) Dexamethasone suppression test - c) Karyotyping - d) Serum prolactin - e) Transvaginal ultrasound

**CORRECT-CHOICE LINE**: ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

ACTH stimulation testing is correct. The clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH). This form is milder, more common, and often diagnosed in later childhood or early adulthood. Usually, women with non-classic CAH present with signs of excess androgens (i.e., excess body hair, early puberty, irregular periods). Hypoaldosteronism (e.g.

hyponatraemia, hyperkalaemia, metabolic acidosis) can also occur. The biochemical features and low early morning cortisol in this case are highly suggestive of hypoaldosteronism.

**REASONING**: Dexamethasone suppression test is incorrect. Although Cushing’s disease remains within the differential diagnosis, there are no other clinical (e.g. hypertension, proximal myopathy, weight gain, easy bruising) or biochemical features (e.g. hyperglycaemia, hypernatraemia, hypokalaemia) to support this diagnosis. Furthermore, the low early morning cortisol makes this an unlikely diagnosis.

Karyotyping is incorrect. Although Turner’s syndrome can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Serum prolactin is incorrect. Although hyperprolactinaemia can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Transvaginal ultrasound is incorrect. Although ovarian tumours can result in androgen excess, this usually results in virilization which is not present in this case. Furthermore, hypoaldosteronism would not be expected to occur with ovarian pathology.

**>>DESCRIPTION**: A 22-year-old woman with infertility, irregular periods, and excess hair presents. Examination reveals: HR 82, BP 115/75, RR 16, SpO2 98%, T 37.2ºC. Labs: Hb 125 g/L, Platelets 228 \* 10^9 /L, WBC 8.2 \* 10^9 /L, Na+ 132 mmol/L, K+ 5.1 mmol/L, Urea 6.2 mmol/L, Creatinine 74 µmol/L, CRP 2 mg/L, Glucose 5.2 mmol/L, Early morning cortisol 140 nmol/l.

**>>OPTIONS**: a) ACTH stimulation testing b) Dexamethasone suppression test c) Karyotyping d) Serum prolactin e) Transvaginal ultrasound

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: ACTH stimulation testing

**>>REASONING**: The clinical and biochemical findings (hirsutism, irregular menses, low sodium, high potassium, low cortisol) suggest non-classic congenital adrenal hyperplasia (CAH), making ACTH stimulation testing the correct choice. Dexamethasone suppression test is less likely due to the absence of Cushing’s features. Karyotyping, serum prolactin, and transvaginal ultrasound are less likely given the biochemical features favoring CAH.

## Question #:336

**CLINICAL SCENERIO**: A 22-year-old woman presents with a 12-month history of infertility, irregular periods, and excess hair growth. Observations: heart rate 82 bpm, BP 115/75 mmHg, RR 16 breaths/min, SpO2 98%, temperature 37.2ºC. Blood results are provided, including Hb, Platelets, WBC, Na+, K+, Urea, Creatinine, CRP, Glucose, and Early morning cortisol.

**QUESTION LINE**: What investigation is required?

**OPTIONS**: f) ACTH stimulation testing g) Dexamethasone suppression test h) Karyotyping i) Serum prolactin j) Transvaginal ultrasound

**CORRECT-CHOICE LINE**: Correct answer Is a.

**REASONING**: ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

ACTH stimulation testing is correct. The clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH). This form is milder, more common, and often diagnosed in later childhood or early adulthood. Usually, women with non-classic CAH present with signs of excess androgens (i.e., excess body hair, early puberty, irregular periods). Hypoaldosteronism (e.g. hyponatraemia, hyperkalaemia, metabolic acidosis) can also occur. The biochemical features and low early morning cortisol in this case are highly suggestive of hypoaldosteronism.

Dexamethasone suppression test is incorrect. Although Cushing’s disease remains within the differential diagnosis, there are no other clinical (e.g. hypertension, proximal myopathy, weight gain, easy bruising) or biochemical features (e.g. hyperglycaemia, hypernatraemia, hypokalaemia) to support this diagnosis. Furthermore, the low early morning cortisol makes this an unlikely diagnosis.

Karyotyping is incorrect. Although Turner’s syndrome can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Serum prolactin is incorrect. Although hyperprolactinaemia can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Transvaginal ultrasound is incorrect. Although ovarian tumours can result in androgen excess, this usually results in virilization which is not present in this case. Furthermore, hypoaldosteronism would not be expected to occur with ovarian pathology.

**>>DESCRIPTION**: A 22-year-old woman with infertility, irregular periods, and excess hair growth has specific blood test results. Which investigation is most appropriate?

**>>OPTIONS**: a) ACTH stimulation testing b) Dexamethasone suppression test c) Karyotyping d) Serum prolactin e) Transvaginal ultrasound

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: ACTH stimulation testing

**>>REASONING**: ACTH stimulation testing is correct because the clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH), which is supported by the symptoms and low early morning cortisol. Other tests are less likely as they do not explain the specific blood test results.

## Question #:85

**CLINICAL SCENERIO**: A 64-year-old man is reviewed in clinic. He has a history of ischaemic heart disease and was diagnosed with type 2 diabetes mellitus around 12 months ago. At this time of diagnosis his HbA1c was 7.6% (60 mmol/mol) and he was started on metformin which was titrated up to a dose of 1g bd. The most recent bloods show a HbA1c of 6.8% (51 mmol/mol). He has just retired from working in the IT industry and his body mass index (BMI) today is 28 kg/m². His other medication is as follows:

Atorvastatin 80mg on Aspirin 75mg od Bisoprolol 2.5 mg od Ramipril 5mg od

**QUESTION LINE**: What is the most appropriate next step?

**OPTIONS**: a) Add sitagliptin b) Make no changes to his medication c) Add empagliflozin d) Add pioglitazone e) Add exenatide

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Normally NICE recommend we only add another drug if the HbA1c has risen to >= 58 mmol/mol (7.5%) at this stage. However, as this patient has established cardiovascular disease he should also be given an SGLT-2 inhibitor (e.g. empagliflozin) in addition to metformin.

**>>DESCRIPTION**: A 64-year-old man with ischaemic heart disease and type 2 diabetes (diagnosed 12 months ago, HbA1c 6.8% on metformin 1g BD) presents with a BMI of 28 kg/m². Current medications include atorvastatin, aspirin, bisoprolol, and ramipril.

**>>OPTIONS**: a) Add empagliflozin b) Add exenatide c) Add pioglitazone d) Add sitagliptin e) Make no changes to his medication

**>>CORRECT-CHOICE LINE**: Correct answer is a.

**>>CORRECT-CHOICE\_TEXT**: Add empagliflozin

**>>REASONING**: The patient has established cardiovascular disease. Therefore, an SGLT-2 inhibitor (empagliflozin) should be added to metformin, even though HbA1c is below 7.5%.

## Question #:98

**CLINICAL SCENERIO**: A 62-year-old taxi driver with a history of inferior myocardial infarction and stenting is reviewed in the diabetes clinic. He is currently on metformin 1g twice a day, ramipril, and indapamide. His blood pressure is 139/85 mmHg, pulse is 84 bpm and regular. There are bilateral basal crackles consistent with cardiac failure. Labs: Na+ 140 mmol/l, K+ 4.5 mmol/l, Urea 7.2 mmol/l, Creatinine 112 µmol/l, HbA1c 64 mmol/mol.

**QUESTION LINE**: Which of the following is the most appropriate intervention with respect to his glycaemic control?

**OPTIONS**: - a) Add empagliflozin - b) Add gliclazide - c) Add insulin glargine - d) Add pioglitazone - e) Add saxagliptin

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: SGLT-2 inhibition is the best option for glucose control in a patient with IHD and heart failure failing glycaemic control on metformin because there is good evidence for outcome benefit across the class

This patient has a history of diabetes and cardiac failure after a myocardial infarction. Evidence in this patient group is strong for a putative benefit of SGLT-2 inhibitors. In particular both empagliflozin and canagliflozin have reported outcomes data which demonstrates a reduction in mortality of around 1/3rd. Their outcome trials also demonstrate a reduction in episodes of symptomatic cardiac failure and reduced progression of microvascular complications of diabetes.

The other options are all inappropriate. Adding gliclazide is associated with increased risk of hypoglycaemia, weight gain, and is not associated with positive cardiovascular outcomes. In particular, given his occupation as a taxi driver, hypoglycaemia should be avoided. Insulin glargine may promote significant weight gain, can worsen cardiac failure and does also cause hypoglycaemia. Pioglitazone promotes weight gain and fluid retention and is therefore contraindicated in cardiac failure. Saxagliptin was associated with an increase in cardiac failure in the SAVOR-TIMI cardiac outcome trial.

**>>DESCRIPTION**: A 62-year-old taxi driver post-MI with stent, diabetes (metformin), hypertension (ramipril, indapamide), and cardiac failure (crackles). HbA1c is 64 mmol/mol. What is the most appropriate intervention for glycaemic control?

**>>OPTIONS**: a) Add empagliflozin b) Add gliclazide c) Add insulin glargine d) Add pioglitazone e) Add saxagliptin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Add empagliflozin

**>>REASONING**: Empagliflozin (SGLT-2 inhibitor) is the best option due to outcome benefits in patients with IHD and heart failure. Gliclazide increases hypoglycemia risk, glargine promotes weight gain and worsens heart failure, pioglitazone causes fluid retention, and saxagliptin is linked to increased heart failure.

## Question #:191

**CLINICAL SCENERIO**: A 45-year-old man with type 2 diabetes on metformin, post-NSTEMI (3 months ago) on aspirin, clopidogrel, bisoprolol, and ramipril, presents for an annual review. HbA1c was 51 mmol/mol (6 months ago) and 47 mmol/mol (today).

**QUESTION LINE**: What is the correct management of type 2 diabetes in this patient at this point?

**OPTIONS**: a) Add empagliflozin b) Add gliclazide c) Add pioglitazone d) Add sitagliptin e) No changes to current treatment

**CORRECT-CHOICE LINE**: a

**REASONING**: In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

Add empagliflozin is correct. While this patient has well-controlled diabetes on metformin, SGLT-2 inhibitors should be introduced if at any point they developed cardiovascular disease (CVD). As he has had a recent NSTEMI, empagliflozin should be commenced.

Add gliclazide is incorrect. The patient has well-controlled diabetes and so the addition of gliclazide is not warranted.

Add pioglitazone is incorrect. The patient has well-controlled diabetes and so the addition of pioglitazone is not warranted. Additionally, you may be cautious about the possibility of heart failure in this patient.

Add sitagliptin is incorrect. The patient has well-controlled diabetes and so the addition of sitagliptin is not warranted.

No changes to current treatment is incorrect. While the HbA1c is well controlled, if at any point the patient develops CVD, an SGLT-2 inhibitor should be added.

**>>DESCRIPTION**: A 45-year-old man with type 2 diabetes (on metformin) and a history of NSTEMI (3 months prior) is undergoing an annual review. His current medications include aspirin, clopidogrel, bisoprolol, and ramipril. His HbA1c is currently 47 mmol/mol.

**>>OPTIONS**: a) Add empagliflozin b) Add gliclazide c) Add pioglitazone d) Add sitagliptin e) No changes to current treatment

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Add empagliflozin

**>>REASONING**: The correct management is to add empagliflozin because the patient has a history of cardiovascular disease (NSTEMI). The other options are incorrect as the patient’s diabetes is well-controlled, and those medications aren’t indicated in this scenario.

## Question #:319

**CLINICAL SCENERIO**: A 32-year-old man with heterozygous familial hypercholesterolaemia, post inferior myocardial infarction, is on 80mg atorvastatin with LDL cholesterol of 3.5 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate next intervention?

**OPTIONS**: - a) Add cholestyramine - b) Add evolocuma - c) Add fenofibrate - d) Add nicotinic acid - e) Change atorvastatin to rosuvastatin

**CORRECT-CHOICE LINE**: b

**REASONING**: Although the 80mg of atorvastatin has clearly brought the LDL cholesterol much closer to target, but LDL of 3.5 mmol/l is still suboptimal given the history of an inferior myocardial infarction. Evolocumab, a PCSK9 inhibitor which interferes with degradation of the LDL receptor is the most appropriate intervention and can reduce LDL by a further 50%. Given his relatively young age and high lifetime risk of cardiovascular disease, he is exactly the type of patient likely to benefit from PCSK9 inhibition.

Cholestyramine was formerly used in the treatment of hypercholesterolaemia, it is a cholesterol binding resin, but is less effective in lowering cholesterol than a statin. Both fenofibrate and nicotinic acid have most effect in lowering triglyerides, and changing atorvastatin to rosuvastatin is likely to have limited benefit in further lowering LDL.

**>>DESCRIPTION**: A 32-year-old man with heterozygous familial hypercholesterolaemia, post inferior myocardial infarction, has an LDL cholesterol of 3.5 mmol/l on 80mg atorvastatin.

**>>OPTIONS**: a) Add cholestyramine b) Add evolocuma c) Add fenofibrate d) Add nicotinic acid e) Change atorvastatin to rosuvastatin

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Add evolocuma

**>>REASONING**: Evolocumab, a PCSK9 inhibitor, is the most appropriate intervention to further reduce LDL cholesterol. Other options are less effective for lowering LDL or primarily target triglycerides.

## Question #:194

**CLINICAL SCENERIO**: A 47-year-old man with type 2 diabetes (HbA1c 48mmol/mol) and heart failure (preserved ejection fraction) on metformin, apixaban, bisoprolol, atorvastatin, and ramipril.

**QUESTION LINE**: What would be the most appropriate course of action regarding his diabetic treatment?

**OPTIONS**: - a) Add in dapagliflozin - b) Add in glimepiride - c) Add in sitagliptin - d) No changes to current medications - e) Reduce metformin dose

**CORRECT-CHOICE LINE**: a

**REASONING**: In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

The stem describes a patient with well-controlled type 2 diabetes mellitus and a background of heart failure. He is also on apixaban for atrial fibrillation and so at a higher risk of cardiovascular disease. All patients with type 2 diabetes and concomitant high risk of cardiovascular disease, chronic heart failure, or new cardiovascular disease, should be commenced on an SGLT-2 inhibitor once established on metformin if there are no contraindications.

Add in dapagliflozin is correct. Patients with a high risk of cardiovascular disease should be started on an SGLT-2 inhibitor such as dapagliflozin.

Add in glimepiride is incorrect. Glimepiride is a sulfonylurea, these drugs work by increasing insulin secretion and can be helpful for patients with symptomatic, recurrent hyperglycaemia. It can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Add in sitagliptin is incorrect. Sitagliptin is a DDP-4 inhibitor and can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Reduce metformin dose is incorrect, the current metformin dose is maintaining a good HbA1c and the patient is not troubled by hypo or hyperglycaemia. There would be no indication to reduce the dose in this case.

No changes to current medications would be appropriate if this patient had not developed atrial fibrillation. Due to the development of atrial fibrillation, his cardiovascular risk is much increased and as a result, the current best practice is to add in an SGLT-2 inhibitor such as dapagliflozin.

**>>DESCRIPTION**: A 47-year-old man with type 2 diabetes (HbA1c 48mmol/mol), heart failure, and atrial fibrillation, is on metformin, apixaban, bisoprolol, atorvastatin, and ramipril. What’s the best diabetic treatment?

**>>OPTIONS**: a) Add in dapagliflozin b) Add in glimepiride c) Add in sitagliptin d) No changes to current medications e) Reduce metformin dose

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Add in dapagliflozin

**>>REASONING**: The best action is to add dapagliflozin because of the patient’s high cardiovascular risk due to heart failure and atrial fibrillation. Other options (glimepiride, sitagliptin, and no change) are less appropriate as the patient is meeting HbA1c targets or not addressing the cardiovascular risk. Reducing metformin isn’t indicated as HbA1c is controlled.

## Question #:145

**CLINICAL SCENERIO**: A 50-year-old man with a history of weight loss has thyroid function tests: TSH 0.01 mIU/L, T4 8.5 ug/dL. No levothyroxine use.

**QUESTION LINE**: You should advise which of the following:

**OPTIONS**: a) Admit for urgent MRI head b) Repeat the bloods and include parathyroid hormone (PTH) c) Add on T3 as this may represent T3 toxicosis d) Start radio-iodine treatment immediately e) Start thyroxine replacement

**CORRECT-CHOICE LINE**: c

**REASONING**: In approximately 5% of patients with clinical and biochemical hyperthyroidism T3 may be elevated prior to T4. This is known as T3 toxicosis.

When both free hormones are normal but TSH is low, the term subclinical thyrotoxicosis can be applied.

Radio-iodine treatment should never be started without discussion with the patient and is only used prior to carbimazole in certain circumstances. An MRI head would be part of the work up for secondary hyperthyroidism. PTH would not be useful in this circumstance and starting thyroxine would not be a good idea! T3 toxicosis is treated in the same manner as T4 hyperthyroidism.

**>>DESCRIPTION**: A 50-year-old man with weight loss has thyroid function tests showing TSH 0.01 mIU/L, T4 8.5 ug/dL. No levothyroxine use.

**>>OPTIONS**: a) Admit for urgent MRI head b) Add on T3 as this may represent T3 toxicosis c) Repeat the bloods and include parathyroid hormone (PTH) d) Start radio-iodine treatment immediately e) Start thyroxine replacement

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Add on T3 as this may represent T3 toxicosis

**>>REASONING**: The patient’s presentation could indicate T3 toxicosis, where T3 is elevated before T4. This requires further investigation and management similar to T4 hyperthyroidism. The other options are less appropriate given the presentation.

## Question #:102

**CLINICAL SCENERIO**: A 78-year-old man with metastatic lung carcinoma presents with increasing lethargy and a number of falls. He describes feeling very unsteady on standing from his bed. His appetite has been poor and he has vomited two times each day for the past three days. There is no diarrhoea or abdominal pain and he denies any shortness of breath. He takes regular paracetamol for pain and omeprazole for dyspepsia. On examination, he is a tanned gentleman with cachexia. He appears pale with dry mucosa and his abdomen is soft and non-tender. There is reduced air entry at the right base with bronchial breathing overlying. His heart sounds are normal and his capillary refill time is prolonged to 4 seconds. Bloods pressure is 85/65mmHg, heart rate 86/min, respiratory rate 23/min

| Hb | 102 g/l | Na + | 129 mmol/l |
| --- | --- | --- | --- |
| Platelets | 189 \* 10 9 /l | K + | 5.0 mmol/l |
| WBC | 5.6\* 10 9 /l | Urea | 7.2 mmol/l |
| Neuts | 4.2 \* 10 9 /l | Creatinine | 87 µmol/l |
| Lymphs | 0.7 \* 10 9 /l | CRP | 32 mg/l |
| Eosin | 0.1 \* 10 9 /l |  |  |

CT-chestabdomenpelvis

There is a 4cm mass in the right lower lobe with extension to adjacent pleura. Bilateral pleural effusions are present. There is mediastinal lymphadenopathy and enlargement of para-aortic nodes with masses seen in both adrenals. Findings are in keeping with a primary lung malignancy with metastatic spread

**QUESTION LINE**: What is the likeliest explanation of this presentation?

**OPTIONS**: - a) SIADH - b) Terminal decline - c) Addisonian crisis - d) Proton pump inhibitor side effects - e) Pneumonia

**CORRECT-CHOICE LINE**: Correct anser is c.

**REASONING**: Metastatic malignancy can cause Addison’s disease

This gentleman presents in a shock like state with poor peripheral perfusion. This could be caused by pneumonia but there are no signs of sepsis and no consolidation on CT. SIADH and PPIs can cause hyponatraemia but not cardiovascular collapse. Terminal decline is the diagnosis of exclusion. This gentleman does have metastatic deposits in both adrenal glands which can impair function and cause Addison’s. Furthermore, he has hyponatraemia, hyperkalaemia and hyperpigmentation all which are features seen in Addison’s.

**>>DESCRIPTION**: A 78-year-old man with metastatic lung carcinoma presents with lethargy, falls, and unsteadiness. He has poor appetite and has vomited twice daily for three days. Examination reveals cachexia, pallor, dry mucosa, reduced air entry at the right base, prolonged capillary refill, and hypotension (85/65mmHg). Labs show hyponatremia (Na+ 129 mmol/l) and hyperkalemia (K+ 5.0 mmol/l). CT shows a right lower lobe mass with pleural extension, bilateral effusions, mediastinal lymphadenopathy, and adrenal masses.

**>>OPTIONS**: a) Addisonian crisis b) Pneumonia c) Proton pump inhibitor side effects d) SIADH e) Terminal decline

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Addisonian crisis

**>>REASONING**: The patient’s presentation (shock, hyponatremia, hyperkalemia, hyperpigmentation) and CT findings (metastatic deposits in adrenals) suggest Addisonian crisis. Pneumonia is less likely due to absence of sepsis signs and consolidation on CT. SIADH and PPIs don’t cause cardiovascular collapse. Terminal decline is a diagnosis of exclusion.

## Question #:327

**CLINICAL SCENERIO**: A 78-year-old man with metastatic lung carcinoma presents with increasing lethargy, falls, poor appetite, and vomiting. Examination reveals cachexia, pallor, dry mucosa, reduced air entry at the right base, and prolonged capillary refill. Blood pressure is low, and laboratory results show hyponatremia. CT scan confirms a lung mass with metastatic spread to the adrenals.

**QUESTION LINE**: What is the likeliest explanation of this presentation?

**OPTIONS**: f) SIADH g) Terminal decline h) Addisonian crisis i) Proton pump inhibitor side effects j) Pneumonia

**CORRECT-CHOICE LINE**: c

**REASONING**: Metastatic malignancy can cause Addison’s disease. This gentleman presents in a shock like state with poor peripheral perfusion. This could be caused by pneumonia but there are no signs of sepsis and no consolidation on CT. SIADH and PPIs can cause hyponatraemia but not cardiovascular collapse. Terminal decline is the diagnosis of exclusion. This gentleman does have metastatic deposits in both adrenal glands which can impair function and cause Addison’s. Furthermore, he has hyponatraemia, hyperkalaemia and hyperpigmentation all which are features seen in Addison’s.

**>>DESCRIPTION**: A 78-year-old man with metastatic lung carcinoma presents with lethargy, falls, poor appetite, vomiting, and hyponatremia, along with low blood pressure and prolonged capillary refill. CT scan shows metastatic spread to the adrenals.

**>>OPTIONS**: a) Addisonian crisis b) Pneumonia c) Proton pump inhibitor side effects d) SIADH e) Terminal decline

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Addisonian crisis

**>>REASONING**: The most likely explanation is Addisonian crisis due to adrenal metastasis. Pneumonia is less likely due to lack of signs. SIADH and PPI side effects cannot explain cardiovascular collapse. Terminal decline is a diagnosis of exclusion.

## Question #:107

**CLINICAL SCENERIO**: A 28-year-old woman with Addison’s disease, diagnosed after a Synacthen test, is being discharged from the hospital. She’s on hydrocortisone three times a day and fludrocortisone daily. She sometimes works night shifts.

**QUESTION LINE**: What is the most appropriate advise to give regarding steroid dosing for night shifts?

**OPTIONS**: - a) Take doses at 09:00, 12:00 and 15:00 regardless of shift patterns - b) Adjust to take first dose at waking, then doses at three hours and six hours from starting - c) On shift days take doses at 09:00, 12:00 and 15:00, but also take an additional 10mg at starting shift - d) On shift days take doses at 09:00, but then take second dose at starting shift and third dose six hours into shift - e) On shift days omit taking steroids

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: For patients on steroid replacement when working shift work, doses should be taken from when waking

Patients with Addison’s disease need both glucocorticoid and mineralocorticoid replacement. Cortisol is highly linked to diurnal rhythm - which is why cortisol naturally peaks in the morning and is at its nadir between midnight and 04:00. Replacement should aim to match the natural rhythm as much as possible; dosing is normally split between three doses, first on waking, then at midday and early afternoon. When a patient shifts their daytime routine, such as working on night shifts or travelling, the patient should be advised to take their morning dose on waking and maintain the timing from there. Patients should not be advised to omit doses, and encouraged to always ensure they are well supplied.

**>>DESCRIPTION**: A 28-year-old woman with Addison’s disease, managed with hydrocortisone and fludrocortisone, is being discharged. She works night shifts sometimes.

**>>OPTIONS**: a) Adjust to take first dose at waking, then doses at three hours and six hours from starting b) On shift days omit taking steroids c) On shift days take doses at 09:00, 12:00 and 15:00, but also take an additional 10mg at starting shift d) On shift days take doses at 09:00, but then take second dose at starting shift and third dose six hours into shift e) Take doses at 09:00, 12:00 and 15:00 regardless of shift patterns

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Adjust to take first dose at waking, then doses at three hours and six hours from starting

**>>REASONING**: Steroid replacement should mimic the diurnal rhythm. Patients on shift work should take their first dose upon waking and maintain the subsequent dose intervals. Omitting doses is not advised.

## Question #:332

**CLINICAL SCENERIO**: A 28-year-old woman with Addison’s disease, treated with hydrocortisone and fludrocortisone, is being discharged. She reports occasional shift work. What’s the best steroid dosing advice for night shifts?

**QUESTION LINE**: What is the most appropriate advise to give regarding steroid dosing for night shifts?

**OPTIONS**: - f) Take doses at 09:00, 12:00 and 15:00 regardless of shift patterns - g) Adjust to take first dose at waking, then doses at three hours and six hours from starting - h) On shift days take doses at 09:00, 12:00 and 15:00, but also take an additional 10mg at starting shift - i) On shift days take doses at 09:00, but then take second dose at starting shift and third dose six hours into shift - j) On shift days omit taking steroids

**CORRECT-CHOICE LINE**: b

**REASONING**: For patients on steroid replacement when working shift work, doses should be taken from when waking

Patients with Addison’s disease need both glucocorticoid and mineralocorticoid replacement. Cortisol is highly linked to diurnal rhythm - which is why cortisol naturally peaks in the morning and is at its nadir between midnight and 04:00. Replacement should aim to match the natural rhythm as much as possible; dosing is normally split between three doses, first on waking, then at midday and early afternoon. When a patient shifts their daytime routine, such as working on night shifts or travelling, the patient should be advised to take their morning dose on waking and maintain the timing from there. Patients should not be advised to omit doses, and encouraged to always ensure they are well supplied.

**>>DESCRIPTION**: A 28-year-old woman with Addison’s disease on hydrocortisone and fludrocortisone needs advice on steroid dosing for night shifts.

**>>OPTIONS**: a) Adjust to take first dose at waking, then doses at three hours and six hours from starting b) On shift days omit taking steroids c) On shift days take doses at 09:00, but then take second dose at starting shift and third dose six hours into shift d) On shift days take doses at 09:00, 12:00 and 15:00, but also take an additional 10mg at starting shift e) Take doses at 09:00, 12:00 and 15:00 regardless of shift patterns

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Adjust to take first dose at waking, then doses at three hours and six hours from starting

**>>REASONING**: The best advice is to take the first dose upon waking and maintain the timing of subsequent doses to match the natural diurnal rhythm. Incorrect options suggest fixed schedules, additional doses, or omitting doses, which are not appropriate for shift workers.

## Question #:65

**CLINICAL SCENERIO**: A 53-year-old female presents with 48 hours of general malaise. 30 years ago, she underwent a resection of a pituitary mass and has since been compliant on desmopressin, levothyroxine and hydrocortisone, up until her last dose earlier in the morning. She has no other past medical history. Her husband reports the patient to have had reduced oral intake for the past 2 days while she has been unwell. She has no reported head injuries, rigors or pyrexia. On examination, her GCS is E3 V2 M5. She is cool peripherally and a temperature demonstrates 33.4 degrees under her tongue. Her spot blood glucose is 2.2 mmol/l. Her blood pressure is 86/50 mmhg heart rate 110/min and sinus rhythm. Blood tests demonstrate a sodium of 158 mmol/l and potassium of 4.2 mmol/l.

**QUESTION LINE**: What is your first action(s)?

**OPTIONS**: - a) Send thyroid function test - b) Administer IV liothyronine - c) Send random cortisol - d) Administer IV hydrocortisone - e) CT head, blood culture, urine dip, HbA1c

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: In the context of a sudden decline in any patient with long-term steroid use with hypotension, hypothermia and hypoglycaemia, you must treat presumptively for an Addisonian crisis. It must be remembered while the classic biochemical presentation of hyponatraemia and hyperkalaemia is frequently quoted in textbooks, this is frequently not the case in real life. While she may indeed be thyroid deficient, neither thyroid function tests nor intravenous free T3 is the most immediate treatment. A random cortisol, in the context of recent hydrocortisone in the community, is uninterpretable.

**>>DESCRIPTION**: A 53-year-old female, s/p resection of a pituitary mass 30 years ago, presents with 48 hours of malaise. She is compliant on desmopressin, levothyroxine, and hydrocortisone but missed her morning dose. She reports reduced oral intake, GCS is E3 V2 M5, temperature is 33.4 degrees, blood glucose is 2.2 mmol/l, and blood pressure is 86/50 mmHg. Sodium is 158 mmol/l and potassium is 4.2 mmol/l.

**>>OPTIONS**: a) Administer IV hydrocortisone b) Administer IV liothyronine c) CT head, blood culture, urine dip, HbA1c d) Send random cortisol e) Send thyroid function test

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Administer IV hydrocortisone

**>>REASONING**: Given the patient’s history of pituitary mass resection, long-term steroid use, hypotension, hypothermia, and hypoglycemia, the most immediate action is to treat for an Addisonian crisis with IV hydrocortisone. Thyroid function tests and random cortisol are not the most immediate treatments.

## Question #:174

**CLINICAL SCENERIO**: A 70-year-old woman presents to the ED with confusion, dizziness, and weakness. She has a history of type 2 diabetes managed with metformin and glimepiride, hypertension, and hyperlipidemia. She has recurrent hypoglycemic episodes. Initial treatment with glucagon and dextrose is ineffective.

**QUESTION LINE**: Which of the following is the next best step in the management of this patient?

**OPTIONS**: 1. Recheck insulin and C-peptide levels 2. Spiral computed tomography (CT) of the abdomen 3. Repeat glucagon IM 4. Observe for 30 minutes and recheck finger-stick glucose 5. Administer octreotide IM

**CORRECT-CHOICE LINE**: e

**REASONING**: In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride. This patient has symptomatic hypoglycaemia and elevated insulin and C-peptide levels, suggesting that the cause of her hypoglycaemia is excessive insulin secretion by her pancreas. Since she is on glimepiride, the most likely cause is sulphonylurea overdose, which should be treated with octreotide if the patient remains hypoglycemic after dextrose administration.

**>>DESCRIPTION**: A 70-year-old woman with type 2 diabetes, managed with metformin and glimepiride, presents with recurrent hypoglycemic episodes unresponsive to glucagon and dextrose.

**>>OPTIONS**: 1. Administer octreotide IM 2. Observe for 30 minutes and recheck finger-stick glucose 3. Recheck insulin and C-peptide levels 4. Repeat glucagon IM 5. Spiral computed tomography (CT) of the abdomen

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Administer octreotide IM

**>>REASONING**: The patient’s persistent hypoglycemia, despite dextrose, and elevated insulin/C-peptide levels suggest a sulfonylurea overdose. Octreotide is the correct next step to inhibit insulin release.

## Question #:1

**CLINICAL SCENERIO**: A 74-year-old woman with a history of diverticular disease and Graves’ disease presents with lower abdominal pain, fever and bloody diarrhoea. She has a CT of the abdomen with contrast, which confirms diverticulitis. This responds well to treatment, and she is discharged. The patient is readmitted three weeks later in fast atrial fibrillation. On assessment, she reports a one-week history of palpitations, non-bloody diarrhoea and feeling hot all the time. She continues to smoke 5 cigarettes per day and denies recent coryzal symptoms. On examination, she has an irregular pulse of 134 bpm and a BP of 132/88 mmHg. Her abdomen is soft and non-tender, with active bowel sounds. A non-tender goitre is present. Her investigations:

| Thyroid stimulating hormone (TSH) | < 0.05 mU/L | (0.5-5.5) |
| --- | --- | --- |
| Free thyroxine (T4) | 24 pmol/L | (9.0 - 18) |

**QUESTION LINE**: What is the most likely cause of her presentation?

**OPTIONS**: a) Administration of contrast b) De Quervain’s thyroiditis c) Further episode of diverticulitis d) Riedel’s thyroiditis e) Smoking

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Thyrotoxicosis may develop in patients following the administration of contrast, particularly if a background of thyroid disease

Administration of contrast is the correct answer here. This can precipitate thyrotoxicosis in patients with a history of previous thyroid disease. IV contrast results in a load of iodine to the thyroid, resulting in increased secretion of thyroid hormones and hyperthyroidism, which can develop over 2-12 weeks.

De Quervain’s thyroiditis often follows an upper respiratory viral illness and usually presents with a painful goitre. In this case, the patient denies coryzal symptoms and has a non-tender goitre, making this diagnosis less likely.

Though a further episode of diverticulitis is possible, the vignette suggests a clinical resolution, and this diagnosis would be unlikely with a soft, non-tender abdomen.

Patients with Riedel’s thyroiditis classically present with clinical and biochemical features consistent with hypothyroidism, which is not the case here.

While the patient has a history of smoking , her intake has not drastically increased. Furthermore, smoking induces mild hyperthyroidism, so this is unlikely to be the cause of her presentation in this case.

**>>DESCRIPTION**: A 74-year-old woman with diverticular disease and Graves’ disease presents with lower abdominal pain, fever, bloody diarrhea, and atrial fibrillation. A CT scan confirmed diverticulitis, which resolved. Three weeks later, she was readmitted with palpitations, non-bloody diarrhea, and feeling hot. She smokes 5 cigarettes daily and denies recent colds. Exam shows an irregular pulse (134 bpm), BP 132/88 mmHg, soft non-tender abdomen, and a non-tender goiter. TSH <0.05 mU/L (0.5-5.5), Free T4 24 pmol/L (9.0-18).

**>>OPTIONS**: a) Administration of contrast b) De Quervain’s thyroiditis c) Further episode of diverticulitis d) Riedel’s thyroiditis e) Smoking

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Administration of contrast

**>>REASONING**: Contrast administration is the most likely cause of thyrotoxicosis given her history of thyroid disease. Iodine from the contrast increases thyroid hormone secretion. Other options are unlikely: De Quervain’s thyroiditis usually presents with a painful goiter and preceding illness; diverticulitis resolved; Riedel’s thyroiditis causes hypothyroidism; and smoking-induced hyperthyroidism is usually mild.

## Question #:219

**CLINICAL SCENERIO**: A 58-year-old man with resistant hypertension despite multiple medications presents with specific observations and investigations.

**QUESTION LINE**: What is the single most likely diagnosis?

**OPTIONS**: - a) Adrenal hyperplasia - b) Conn’s adenoma - c) Malignant hypertension - d) Phaeochromocytoma - e) Renal artery stenosis

**CORRECT-CHOICE LINE**: a

**REASONING**: All of the above diagnoses could cause hypertension. This patient has a hypokalaemic alkalosis and low renin. The differentials are therefore Conn’s adenoma and adrenal hyperplasia. To differentiate between the two look at the aldosterone on standing. In this case, the aldosterone increases on prolonged standing, therefore, the diagnosis is adrenal hyperplasia. In Conn’s adenoma, the aldosterone would stay the same or drop on standing.

In malignant hypertension and renal artery stenosis, the renin would be high.

**>>DESCRIPTION**: A 58-year-old man with resistant hypertension, hypokalemic alkalosis, and low renin presents with specific investigation results.

**>>OPTIONS**: a) Adrenal hyperplasia b) Conn’s adenoma c) Malignant hypertension d) Phaeochromocytoma e) Renal artery stenosis

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Adrenal hyperplasia

**>>REASONING**: The most likely diagnosis is adrenal hyperplasia due to hypokalemic alkalosis, low renin, and an increase in aldosterone upon standing. Conn’s adenoma is also a differential, but can be ruled out by increase in aldosterone on standing. Malignant hypertension and renal artery stenosis are unlikely due to the low renin levels.

## Question #:209

**CLINICAL SCENERIO**: A 24-year-old lady presents with increasing confusion, weight loss, lethargy, and abdominal cramping. Examination reveals thinness, cool skin, sunken eyes, and a capillary refill time of 3 seconds. Investigations show hyponatremia, hyperkalemia, and normocytic anemia.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Adrenal insufficiency - b) Laxative abuse - c) Hypothyroidism - d) Diuretic use - e) SiADH

**CORRECT-CHOICE LINE**: Corectr answer is a.

**REASONING**: This lady has hypovolaemic hyponatraemia. Sodium can be lost through the gastrointestinal tract, skin or urinary tract. This patient is likely to have renal loss secondary to adrenal insufficiency. The urinary sodium would be reduced if there were gastrointestinal or skin losses. Also, the patient would be hypokalaemic if there were laxative abuse. The patient would be expected to have hypokalaemia if there were abuse of bendrofumethiazide. The patient is hypovolaemic and therefore SiADH is excluded, also, hypothyroidism and adrenal insufficiency have not been excluded which would be necessary to make the diagnosis of SiADH. Hypothyroidism classically presents with weight gain and euvolaemic hyponatraemia. This patient has adrenal insufficiency. It is possibly of an autoimmune nature with a probable autoimmune hypothyroidism in the immediate family. She has a gradual onset of non-specific symptoms lethargy, weight loss and abdominal symptoms. The hyponatraemia has caused confusion. The blood tests reveal hypoglycaemia, hyponatraemia with hyperkalaemia and normocytic anaemia. Examination revealed signs of hypovolaemia, it also could show skin or mucus membrane pigmentation.

**>>DESCRIPTION**: A 24-year-old woman presents with confusion, weight loss, lethargy, and abdominal cramps. Examination reveals signs of hypovolemia and investigations show hyponatremia, hyperkalemia, and normocytic anemia.

**>>OPTIONS**: a) Adrenal insufficiency b) Diuretic use c) Hypothyroidism d) Laxative abuse e) SiADH

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Adrenal insufficiency

**>>REASONING**: The most likely diagnosis is adrenal insufficiency due to hypovolemic hyponatremia, hyperkalemia, and associated symptoms. Other options are less likely because they do not explain the electrolyte abnormalities or clinical presentation. SiADH is excluded due to hypovolemia, and hypothyroidism typically presents with weight gain.

## Question #:96

**CLINICAL SCENERIO**: A 45-year-old female with a 2-year history of headache and visual blurring presents with poorly controlled hypertension (235/160mmHg) despite maximal doses of four antihypertensives, including 50mg spironolactone. Blood tests show: Na+ 140 mmol/l, K+ 2.9 mmol/l, Urea 5.8 mmol/l, Creatinine 78 µmol/l, CRP 2 mg/l, Serum ambulatory renin activity 0.34 pmol/L, Serum ambulatory aldosterone 2052 pmol/L. A CT adrenal reveals a right adrenal mass of 2.5cm diameter. The patient wants to address the underlying problem.

**QUESTION LINE**: What is the most appropriate next management step?

**OPTIONS**: - a) Add amiloride - b) Add eplerenone - c) Increase spironolactone to 100mg OD - d) Adrenal vein sampling - e) Right adrenalectomy

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism

The patient presents with poorly controlled hypertension, is hypokalaemic, hypertensive, metabolically alkalotic, has increased aldosterone and reduced renin. The underlying diagnosis is Conn’s disease. The treatment involves removal of the aldosterone secreting tumour. However, those pursuing adrenalectomy must undergo adrenal vein sampling first, CT imaging alone is not diagnostic of the symptomatic side responsible for aldosterone secretion.

**>>DESCRIPTION**: 45-year-old female with headache, visual blurring, and poorly controlled hypertension (235/160mmHg) despite maximal antihypertensives and 50mg spironolactone. Labs: K+ 2.9 mmol/l, renin 0.34 pmol/L, aldosterone 2052 pmol/L. CT shows a 2.5cm right adrenal mass. Patient seeks definitive treatment.

**>>OPTIONS**: a) Add amiloride b) Add eplerenone c) Adrenal vein sampling d) Increase spironolactone to 100mg OD e) Right adrenalectomy

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Adrenal vein sampling

**>>REASONING**: Adrenal venous sampling (AVS) is needed to differentiate between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism (Conn’s disease) before adrenalectomy. CT imaging alone isn’t sufficient to determine the source of aldosterone secretion.

## Question #:238

**CLINICAL SCENERIO**: A 54-year-old man with persistent hypertension and type 2 diabetes (on metformin) presents with laboratory findings suggestive of primary hyperaldosteronism. He is keen to undergo surgical management.

**QUESTION LINE**: What investigation would be the most reliable in confirming the location of the lesion in this case?

**OPTIONS**: - a. Adrenal venous sampling - b. Delayed contrast-enhanced CT adrenals - c. Fine-needle aspiration adrenal biopsy - d. MRI adrenals - e. MRI brain

**CORRECT-CHOICE LINE**: A

**REASONING**: Adrenal venous sampling (AVS) is the gold standard for distinguishing between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism. The other options are less reliable or not appropriate in this context.

**>>DESCRIPTION**: A 54-year-old man with hypertension and type 2 diabetes (on metformin) presents with primary hyperaldosteronism and wants surgical management. Which investigation is most reliable to locate the lesion?

**>>OPTIONS**: a. Adrenal venous sampling b. Delayed contrast-enhanced CT adrenals c. Fine-needle aspiration adrenal biopsy d. MRI adrenals e. MRI brain

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Adrenal venous sampling

**>>REASONING**: Adrenal venous sampling (AVS) is the most reliable to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism, which is the likely diagnosis. CT, MRI and biopsy are less reliable. MRI brain is irrelevant.

## Question #:129

**CLINICAL SCENERIO**: A 34-year-old woman with Addison’s disease calls for advice. She has been vomiting for 24 hours and unable to take her oral hydrocortisone and fludrocortisone. She denies dizziness, blackouts, or diarrhea and has a normal temperature. She has IM hydrocortisone at home.

**QUESTION LINE**: What is the most appropriate advice to give in regards to her hydrocortisone?

**OPTIONS**: - a) Advise her to come to the emergency department for IV hydrocortisone - b) Advise her to take the IM hydrocortisone at home whilst vomiting - c) Advise her to not take any hydrocortisone until the vomiting settles - d) Adviser her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms - e) Advise her to try and take both IM and oral hydrocortisone

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: A person with Addisons’ who vomits should take IM hydrocortisone until vomiting stops

This is a patient with Addison’s disease who is unable to take her regular oral hydrocortisone and is therefore at risk of developing an Addisonian crisis. If unable to take the normal oral hydrocortisone then the patient should be advised to take IM hydrocortisone to avoid this. This is why all patients with Addison’s disease should have IM hydrocortisone for these situations. If the patient was unwell with systemic involvement then admission for IV fluids and IV hydrocortisone would be appropriate.

**>>DESCRIPTION**: A 34-year-old woman with Addison’s disease, unable to take oral hydrocortisone due to vomiting, seeks advice. She has no dizziness, blackouts, or diarrhea, and has a normal temperature. She has IM hydrocortisone at home.

**>>OPTIONS**: a) Advise her to come to the emergency department for IV hydrocortisone b) Advise her to not take any hydrocortisone until the vomiting settles c) Advise her to take IM hydrocortisone at home whilst vomiting d) Advise her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms e) Advise her to try and take both IM and oral hydrocortisone

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Advise her to take the IM hydrocortisone at home whilst vomiting

**>>REASONING**: Patients with Addison’s disease who are vomiting and cannot take oral hydrocortisone should take IM hydrocortisone to avoid Addisonian crisis. IV hydrocortisone is only needed if systemically unwell.

## Question #:137

**CLINICAL SCENERIO**: A 34-year-old woman with Addison’s disease, taking oral hydrocortisone and fludrocortisone, has been vomiting for 24 hours. She denies other symptoms and has IM hydrocortisone at home.

**QUESTION LINE**: What is the most appropriate advice to give in regards to her hydrocortisone?

**OPTIONS**: a) Advise her to come to the emergency department for IV hydrocortisone b) Advise her to take the IM hydrocortisone at home whilst vomiting c) Advise her to not take any hydrocortisone until the vomiting settles d) Adviser her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms e) Advise her to try and take both IM and oral hydrocortisone

**CORRECT-CHOICE LINE**: b

**REASONING**: A person with Addisons’ who vomits should take IM hydrocortisone until vomiting stops

This is a patient with Addison’s disease who is unable to take her regular oral hydrocortisone and is therefore at risk of developing an Addisonian crisis. If unable to take the normal oral hydrocortisone then the patient should be advised to take IM hydrocortisone to avoid this. This is why all patients with Addison’s disease should have IM hydrocortisone for these situations. If the patient was unwell with systemic involvement then admission for IV fluids and IV hydrocortisone would be appropriate.

**>>DESCRIPTION**: A 34-year-old woman with Addison’s disease, on oral hydrocortisone and fludrocortisone, is vomiting and unable to take her medications. She has IM hydrocortisone at home.

**>>OPTIONS**: a) Advise her to come to the emergency department for IV hydrocortisone b) Advise her to take the IM hydrocortisone at home whilst vomiting c) Advise her to not take any hydrocortisone until the vomiting settles d) Adviser her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms e) Advise her to try and take both IM and oral hydrocortisone

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Advise her to take the IM hydrocortisone at home whilst vomiting

**>>REASONING**: The patient should take IM hydrocortisone because she cannot take her oral medication due to vomiting, preventing an Addisonian crisis.

## Question #:272

**CLINICAL SCENERIO**: A patient with type 1 diabetes mellitus, recently completed a DAFNE course, has experienced three hypoglycemic episodes requiring assistance. He uses a basal-bolus insulin regimen and works in a restaurant. What is the most appropriate action?

**QUESTION LINE**: Apart from adjusting the insulin dose, what is the most appropriate action?

**OPTIONS**: a) Advise the patient to inform the DVLA and to not drive b) The consultant should inform the DVLA c) Advise the patient to start checking blood glucose prior to driving d) Advise the patient to always carry a snack when driving e) Advise the patient to attend retinal screening prior to driving again

**CORRECT-CHOICE LINE**: a

**REASONING**: Patient with diabetes who have had two hypoglycaemic episodes requiring help needs to surrender their driving licence

This patient has had hypoglycaemic episodes needing help multiple times. If a patient needs help to correct the hypoglycaemic episode then it is termed severe hypoglycaemia and is of concern to the DVLA. If a patient has two or more episodes of severe hypoglycaemia then they need to inform the DVLA and not drive.

Patients who take insulin should have their licence for driving assessed on an annual basis. For this they will be required to submit three months of blood glucose readings. Patients are advised to always check blood glucose before driving and to always have a snack in their vehicle as well, not just if having hypoglycaemic episodes.

When the DVLA is informed, the patient should be advised to inform the DVLA themselves rather than breaking patient confidentiality, but if the patient repeatedly fails to follow this advise then the doctor should inform the DVLA after telling the patient that he or she is doing so

**>>DESCRIPTION**: A type 1 diabetic on a basal-bolus insulin regimen, who recently completed a DAFNE course, has experienced three hypoglycemic episodes requiring assistance. What is the most appropriate next step?

**>>OPTIONS**: a) Advise the patient to inform the DVLA and to not drive b) Advise the patient to always carry a snack when driving c) Advise the patient to attend retinal screening prior to driving again d) Advise the patient to start checking blood glucose prior to driving e) The consultant should inform the DVLA

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Advise the patient to inform the DVLA and to not drive

**>>REASONING**: The patient needs to inform the DVLA and stop driving due to recurrent severe hypoglycemia (requiring assistance). Other options are secondary preventative measures, not the immediate priority.

## Question #:117

**CLINICAL SCENERIO**: A 32 year-old man collapsed at work. He doesn’t remember the episode, and there was no incontinence or fitting. He has no prior medical history and takes no regular medication. Examination reveals a blood pressure of 162/95 mmHg, a pulse of 74 bpm, a respiratory rate of 16, and a temperature of 37.4ºC. Heart and lung examination are normal, and his abdomen is soft and non-tender. Blood tests show Na+ 143 mmol/l, K+ 3.0 mmol/l, Urea 5.6 mmol/l, Creatinine 76 µmol/l, Bicarbonate 31 mmol/l, low Renin, and low Aldosterone.

**QUESTION LINE**: Which of the following is the best treatment?

**OPTIONS**: a) Amiloride b) Bumetanide c) Spironolactone d) ACE inhibitor e) Angiotensin II receptor blocker

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This man has Liddle’s syndrome, an autosomal dominant disorder characterised by hypertension associated with hypokalaemic metabolic alkalosis, low plasma renin activity, and suppressed aldosterone secretion. Amiloride is the best treatment for the hypertension and hypokalaemia as it acts on the sodium channels directly, as opposed to spironolactone, which acts on mineralocorticoid receptors.

**>>DESCRIPTION**: A 32-year-old man collapsed at work with hypertension (162/95 mmHg), hypokalemia (K+ 3.0 mmol/l), elevated bicarbonate (31 mmol/l), low renin, and low aldosterone, suggesting Liddle’s syndrome.

**>>OPTIONS**: a) ACE inhibitor b) Amiloride c) Angiotensin II receptor blocker d) Bumetanide e) Spironolactone

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Amiloride

**>>REASONING**: The patient has Liddle’s syndrome. Amiloride is the best treatment as it directly acts on sodium channels, addressing both hypertension and hypokalemia. Spironolactone acts on mineralocorticoid receptors and is less effective in this condition.

## Question #:243

**CLINICAL SCENERIO**: A 16-year-old male with 3 months of chronic headaches and visual blurring presents with papilloedema, hard exudates, flame haemorrhage, and a blood pressure of 226/160mmHg. Laboratory findings include hypokalemia, metabolic alkalosis, decreased renin and aldosterone.

**QUESTION LINE**: What is the optimal long-term treatment?

**OPTIONS**: - a) Amlodipine - b) Ramipril - c) Atenolol - d) Doxazosin - e) Amiloride

**CORRECT-CHOICE LINE**: E

**REASONING**: The patient is young and presents with grade 4 hypertensive retinopathic changes associated with a systolic of over 200mmHg, associated with hypokalaemia and a metabolic alkalosis. Importantly, both renin and aldosterone are decreased, ruling out primary hyperaldosteronism. The diagnosis is thus likely to be Liddle’s syndrome, a genetic disorder of ENaC channels in the collecting duct, leading to increased sodium reabsorption and increased potassium excretion. The treatment is with amiloride, a potassium sparing diuretic that directly blocks collecting tubule sodium channels and resolves hypertension.

**>>DESCRIPTION**: A 16-year-old male with chronic headaches, visual blurring, papilledema, and hypertension (226/160 mmHg) presents with hypokalemia, metabolic alkalosis, and suppressed renin/aldosterone.

**>>OPTIONS**: a) Amlodipine b) Amiloride c) Atenolol d) Doxazosin e) Ramipril

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Amiloride

**>>REASONING**: The patient likely has Liddle’s syndrome. Amiloride, a potassium-sparing diuretic, is the optimal long-term treatment because it directly blocks sodium channels in the collecting tubule, resolving the hypertension associated with the condition. Other options are not appropriate for the underlying pathophysiology.

## Question #:287

**CLINICAL SCENERIO**: A 24-year-old woman collapses while shopping. Observations: temperature 37.2°C, pulse 78/min (regular), BP 164/92 mmHg. Heart sounds normal. Abdomen soft. Neurological exam unremarkable. No significant medical history or medications. Blood tests: low renin/aldosterone, hypokalemia, bicarbonate 30 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate treatment for her condition?

**OPTIONS**: - a. Angiotensin converting enzyme inhibitor therapy - b. Bumetanide - c. Potassium replacement - d. Spironolactone - e. Amiloride

**CORRECT-CHOICE LINE**: e

**REASONING**: This woman has presented with the combination of hypokalaemic alkalosis, suppressed renin and aldosterone levels in the presence of hypertension indicates a diagnosis of Liddle syndrome. Hypertension and hypokalaemia respond well to amiloride. Spironolactone is not as effective as this medication acts on the mineralocorticoid receptor, as opposed to amiloride, which acts directly on the sodium channel.

**>>DESCRIPTION**: A 24-year-old woman collapses while shopping, presenting with hypertension, hypokalemia, low renin/aldosterone, and elevated bicarbonate. Which treatment is most appropriate?

**>>OPTIONS**: a) Amiloride b) Angiotensin converting enzyme inhibitor therapy c) Bumetanide d) Potassium replacement e) Spironolactone

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Amiloride

**>>REASONING**: Amiloride is the most appropriate treatment for Liddle syndrome, which is suggested by the patient’s presentation of hypertension, hypokalemia and metabolic alkalosis. Spironolactone is less effective. The other choices are not first-line treatments and/or are not directly relevant to the underlying mechanism of the disease.

## Question #:271

**CLINICAL SCENERIO**: A 17-year-old woman with primary amenorrhoea presents with no axillary or pubic hair, bilateral groin swellings, and normal breast development.

**QUESTION LINE**: What is the likely diagnosis?

**OPTIONS**: - a. 17-alpha-hydroxylase deficiency - b. Androgen insensitivity syndrome - c. Klinefelter syndrome - d. Pituitary tumour - e. Turner syndrome

**CORRECT-CHOICE LINE**: b

**REASONING**: Primary amenorrhoea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome

Androgen insensitivity syndrome is the correct answer. This is an X-linked recessive condition caused by end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Patients often present with primary amenorrhoea along with absent pubic/axillary hair. Bilateral groin swellings may reflect undescended testes and breast development may occur as a result of the conversion of testosterone to oestradiol. Testosterone is typically in the upper normal range for a post-pubertal male.

17-alpha-hydroxylase deficiency is incorrect. This is an uncommon form of congenital adrenal hyperplasia that may also present with primary amenorrhoea and external female genitalia. However, in this condition, testosterone will be low as a lack of androgens are characteristic of this disease.

Klinefelter syndrome is incorrect. This is a syndrome where a patient that is phenotypically male has an extra X chromosome (i.e. 47 XXY). Patients may have small testicles and infertility as predominant features. The patient in this scenario is phenotypically female.

Pituitary tumour is incorrect. This may cause delayed puberty if it causes impaired secretion of gonadotrophins (LH and FSH) i.e. hypogonadotropic hypogonadism. However, breast development may also be impaired with a pituitary tumour and testosterone will be in the normal range for a female, in contrast to this case where it is in the high-normal range for a male.

Turner syndrome is incorrect. This can cause primary amenorrhoea as a consequence of primary ovarian insufficiency. Breast development will be abnormal but pubic and axillary hair development will typically be normal. Unlike this case, testosterone will be in the normal range for a female. Patients have a 45XO karyotype

**>>DESCRIPTION**: A 17-year-old woman with primary amenorrhea presents with absent axillary and pubic hair, bilateral groin swellings, and normal breast development.

**>>OPTIONS**: a. 17-alpha-hydroxylase deficiency b. Androgen insensitivity syndrome c. Klinefelter syndrome d. Pituitary tumour e. Turner syndrome

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Androgen insensitivity syndrome

**>>REASONING**: Androgen insensitivity syndrome is the correct answer due to primary amenorrhea, absent pubic/axillary hair, and normal breast development. Other options are incorrect because they don’t present with all the features of the case, especially the lack of androgen effect.

## Question #:268

**CLINICAL SCENERIO**: A 35-year-old woman with a family history of MEN 2a and a positive RET oncogene test is reluctant to undergo prophylactic thyroidectomy.

**QUESTION LINE**: Which of the following management options would be appropriate for her case?

**OPTIONS**: - a) Annual monitoring of thyroid peroxidase (TPO) - b) Annual monitoring of thyroglobulin - c) Annual 5 hydroxyindoleacetic acid levels (5-HIAA) - d) Annual monitoring of calcitonin - e) Annual monitoring of chromogranin A

**CORRECT-CHOICE LINE**: D

**REASONING**: Calcitonin can be used as a marker to monitor for Medullary thyroid carcinoma in MEN type II in those unwilling to undergo prophylactic thyroidectomy

Calcitonin is a marker for medullary thyroid carcinoma which is associated with MEN type II.

Chromogranin A and 5-HIAA can be used as markers for neuroendocrine tumours and carcinoid syndrome respectively.

Thyroid peroxidase antibody titre can be used as a marker in autoimmune thyroid conditions such as Hashimoto’s thyroiditis and Graves disease.

Thyroglobulin can be used as a marker for papillary or follicular thyroid carcinoma. For example, a rise in this marker following a thyroidectomy can indicate the recurrence of these particular cancers

**>>DESCRIPTION**: A 35-year-old woman with a family history of MEN 2a and a positive RET oncogene is reluctant to undergo prophylactic thyroidectomy.

**>>OPTIONS**: a) Annual 5 hydroxyindoleacetic acid levels (5-HIAA) b) Annual monitoring of calcitonin c) Annual monitoring of chromogranin A d) Annual monitoring of thyroglobulin e) Annual monitoring of thyroid peroxidase (TPO)

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Annual monitoring of calcitonin

**>>REASONING**: Annual calcitonin monitoring is appropriate to monitor for medullary thyroid carcinoma in MEN type II. Other options are markers for different conditions (e.g., chromogranin A for neuroendocrine tumors, thyroglobulin for papillary/follicular thyroid carcinoma, and TPO for autoimmune thyroid diseases).

## Question #:200

**CLINICAL SCENERIO**: A 39-year-old woman had a total thyroidectomy for a 3.2cm papillary thyroid tumor with no lymph node involvement or metastasis. She is in endocrinology clinic and wants to know the best way to monitor for recurrence.

**QUESTION LINE**: What is the most appropriate monitoring?

**OPTIONS**: - a) Annual CT neck - b) Annual US neck - c) Annual MRI neck - d) Annual thyroglobulin - e) Annual TSH and free T

**CORRECT-CHOICE LINE**: d

**REASONING**: Following surgery and radioiodine therapy, patients with papillary thyroid cancer should be monitored with thyroglobulin annually

This patient has a very good prognosis as she has had papillary thyroid cancer which is less than 4cm in size without lymph node or other organ involvement. The most appropriate way to monitor for cancer recurrence is annual thyroglobulin. Imaging is unlikely to detect a recurrence early and is therefore inappropriate. TSH and free T4 levels should be measured frequently as this patient will need thyroxine replacement, but this is not to detect recurrence of malignancy.

**>>DESCRIPTION**: A 39-year-old woman post-total thyroidectomy for a 3.2cm papillary thyroid tumor, without lymph node involvement or metastasis, asks about the best monitoring for recurrence.

**>>OPTIONS**: a) Annual CT neck b) Annual MRI neck c) Annual TSH and free T d) Annual thyroglobulin e) Annual US neck

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Annual thyroglobulin

**>>REASONING**: Annual thyroglobulin is the most appropriate monitoring for recurrence in this patient with papillary thyroid cancer due to her good prognosis. Imaging is less sensitive for early recurrence detection. TSH and free T4 monitoring is important for thyroid hormone replacement but not for detecting recurrence.

## Question #:132

**CLINICAL SCENERIO**: A 40-year-old school teacher presented with a 2-month history of polyuria and elevated blood glucose levels. No family history of diabetes, not on medication. BMI was 22 kg/m 2 with normal examination. Urine negative for ketones. Blood glucose 16.5 mmol/l, pH 7.40, HCO3 25 mmol/l, Na+ 140 mmol/l, K+ 3.7 mmol/l.

**QUESTION LINE**: Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

**OPTIONS**: - a) Oral glucose tolerance test - b) Serum ferritin and total iron binding capacity - c) Mitochondrial gene mutation (A3243G) - d) Anti-GAD antibody - e) Toxicology screen

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of T2DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies)

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of type 2 DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies). Most of these patients are in the age group of 30-50 years, may or may not have a normal BMI and may present with elevated blood glucose levels. These patients can be managed initially with oral hypoglycaemic agents, although the β-cell function may decline over months to a few years, necessitating a relatively early requirement for insulin use.

**>>DESCRIPTION**: 40-year-old with 2-month history of polyuria, elevated blood glucose. No family history of diabetes, normal BMI, negative urine ketones. Blood glucose 16.5 mmol/l. Which test is useful for diagnosis?

**>>OPTIONS**: a) Anti-GAD antibody b) Mitochondrial gene mutation (A3243G) c) Oral glucose tolerance test d) Serum ferritin and total iron binding capacity e) Toxicology screen

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Anti-GAD antibody

**>>REASONING**: The patient’s presentation suggests latent autoimmune diabetes of adulthood (LADA). Anti-GAD antibodies are markers of autoimmunity seen in LADA. Other options are less likely given the clinical picture.

## Question #:140

**CLINICAL SCENERIO**: A 40-year-old teacher with a 2-month history of polyuria and elevated blood glucose, no family history of diabetes, and no current medications. Examination revealed a BMI of 22 kg/m2 and normal findings. Urine was negative for ketones, and blood work showed elevated glucose (16.5 mmol/l), normal pH, HCO3, Na+, and K+.

**QUESTION LINE**: Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

**OPTIONS**: a) Oral glucose tolerance test b) Serum ferritin and total iron binding capacity c) Mitochondrial gene mutation (A3243G) d) Anti-GAD antibody e) Toxicology screen

**CORRECT-CHOICE LINE**: d

**REASONING**: Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of T2DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies)

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of type 2 DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies). Most of these patients are in the age group of 30-50 years, may or may not have a normal BMI and may present with elevated blood glucose levels. These patients can be managed initially with oral hypoglycaemic agents, although the β-cell function may decline over months to a few years, necessitating a relatively early requirement for insulin use.

**>>DESCRIPTION**: A 40-year-old teacher presents with polyuria, elevated blood glucose, normal BMI, and normal examination findings, with negative ketones in urine.

**>>OPTIONS**: a) Anti-GAD antibody b) Mitochondrial gene mutation (A3243G) c) Oral glucose tolerance test d) Serum ferritin and total iron binding capacity e) Toxicology screen

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Anti-GAD antibody

**>>REASONING**: Anti-GAD antibody testing is useful for diagnosing latent autoimmune diabetes of adulthood (LADA), which presents with features similar to type 2 diabetes but with autoimmune markers.

## Question #:20

**CLINICAL SCENERIO**: A 56-year-old man with a history of hypertension presents for review. As part of his annual health check he has a U&E, HbA1c and cholesterol check done. The following results are obtained:

His blood pressure today is 128/78 mmHg. His only regular medication is ramipril 5mg od.

Na +

142 mmol/l

K +

4.6 mmol/l

Urea

5.2 mmol/l

Creatinine

88 µmol/l

Total cholesterol

5.2 mmol/l

HbA1c

45 mmol/mol (6.3%)

His 10-year QRISK2 score is 7%.

**QUESTION LINE**: What is the most appropriate action following these results?

**OPTIONS**: a) Start atorvastatin 20mg on b) Arrange a fasting glucose sample c) Diagnose type 2 diabetes mellitus d) Increase the dose of ramipril e) Add amlodipine 5mg od

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: His QRISK2 score is < 10% so no action needs taking about his cholesterol. His blood pressure is also well controlled

**>>DESCRIPTION**: A 56-year-old hypertensive man has an annual health check with these results: BP 128/78 mmHg, Na+ 142, K+ 4.6, Urea 5.2, Creatinine 88, Total cholesterol 5.2, HbA1c 45 mmol/mol (6.3%). His QRISK2 score is 7%. He takes ramipril 5mg daily.

**>>OPTIONS**: a) Add amlodipine 5mg od b) Arrange a fasting glucose sample c) Diagnose type 2 diabetes mellitus d) Increase the dose of ramipril e) Start atorvastatin 20mg on

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Arrange a fasting glucose sample

**>>REASONING**: The patient’s HbA1c is 6.3%, indicating pre-diabetes, so a fasting glucose sample is the most appropriate next step. His QRISK2 score is low and blood pressure is controlled.

## Question #:339

**CLINICAL SCENERIO**: A 56-year-old man with a history of hypertension presents for review. As part of his annual health check he has a U&E, HbA1c and cholesterol check done. The following results are obtained:

His blood pressure today is 128/78 mmHg. His only regular medication is ramipril 5mg od.

| Na + | 142 mmol/l |
| --- | --- |
| K + | 4.6 mmol/l |
| Urea | 5.2 mmol/l |
| Creatinine | 88 µmol/l |
| Total cholesterol | 5.2 mmol/l |
| HbA1c | 45 mmol/mol (6.3%) |

His 10-year QRISK2 score is 7%.

**QUESTION LINE**: What is the most appropriate action following these results?

**OPTIONS**: - f) Start atorvastatin 20mg on - g) Arrange a fasting glucose sample - h) Diagnose type 2 diabetes mellitus - i) Increase the dose of ramipril - j) Add amlodipine 5mg od

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: His QRISK2 score is < 10% so no action needs taking about his cholesterol. His blood pressure is also well controlled

**>>DESCRIPTION**: A 56-year-old hypertensive man on ramipril 5mg od presents for review with BP 128/78 mmHg. Labs: Na+ 142, K+ 4.6, Urea 5.2, Creatinine 88, Total cholesterol 5.2, HbA1c 45 mmol/mol (6.3%). 10-year QRISK2 score is 7%.

**>>OPTIONS**: a) Add amlodipine 5mg od b) Arrange a fasting glucose sample c) Diagnose type 2 diabetes mellitus d) Increase the dose of ramipril e) Start atorvastatin 20mg on

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Arrange a fasting glucose sample

**>>REASONING**: QRISK2 score is < 10% and blood pressure is well controlled, so no immediate action is needed regarding cholesterol or blood pressure management.

## Question #:84

**CLINICAL SCENERIO**: A 48-year-old man with type 2 diabetes mellitus presents for review. His results are: Total cholesterol 5.3 mmol/l, HDL cholesterol 1.0 mmol/l, LDL cholesterol 3.1 mmol/l, Triglyceride 1.7 mmol/l, HbA1c 6.4%. QRISK2 score shows a 12% 10-year risk of cardiovascular disease. Current medication is metformin 500mg tds.

**QUESTION LINE**: According to recent NICE guidelines, what is the most appropriate action?

**OPTIONS**: a) Simvastatin 40mg on b) Lifestyle advice, repeat lipid profile in 3 months c) Atorvastatin 40mg on d) Atorvastatin 20mg on e) Increase his metformin slowly to 1g tds

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: NICE recommend the following when considering the use of statins in patients with type 2 diabetes mellitus:

**>>DESCRIPTION**: A 48-year-old man with type 2 diabetes mellitus has the following results: Total cholesterol 5.3 mmol/l, HDL 1.0 mmol/l, LDL 3.1 mmol/l, Triglyceride 1.7 mmol/l, HbA1c 6.4%. QRISK2 score: 12%. He is on metformin 500mg tds.

**>>OPTIONS**: a) Atorvastatin 20mg on b) Atorvastatin 40mg on c) Increase his metformin slowly to 1g tds d) Lifestyle advice, repeat lipid profile in 3 months e) Simvastatin 40mg on

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Atorvastatin 20mg on

**>>REASONING**: NICE guidelines should be consulted for statin use in type 2 diabetes.

## Question #:167

**CLINICAL SCENERIO**: A 24-year-old female presents with worsening fatigue, constipation, cold intolerance, and dry skin. Examination reveals postural hypotension and abnormal lab results.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: 1. Autoimmune polyendocrinopathy syndrome (APS) - type 1 2. Autoimmune polyendocrinopathy syndrome (APS) - type 2 3. Addison’s disease 4. Type 1 diabetes mellitus 5. Type 2 diabetes mellitu

**CORRECT-CHOICE LINE**: b

**REASONING**: APS type 2 patients have Addison’s disease plus either T1DM or autoimmune thyroid diseas

A fasting glucose of 7.5 mmol/l suggests a diagnosis of diabetes. In the absence of diabetic symptoms (e.g. polyuria, and polydipsia), this would need to be repeated to confirm the diagnosis. Alternatively, other tests such as HbA1c, or an oral glucose tolerance test could be used.

The clinical features of constipation, cold intolerance and dry skin are suggestive of hypothyroidism. Thyroid function tests should be performed.

The biochemistry (hyponatraemia, hyperkalemia, and acidosis) and postural hypotension are suggestive of hypoaldosteronism.

The unifying diagnosis is autoimmune polyendocrinopathy syndrome (APS) type 2. APS type 2 patients have Addison’s disease plus either T1DM and/or autoimmune

**>>DESCRIPTION**: A 24-year-old female presents with fatigue, constipation, cold intolerance, dry skin, and postural hypotension. Lab results are provided.

**>>OPTIONS**: 1. Addison’s disease 2. Autoimmune polyendocrinopathy syndrome (APS) - type 1 3. Autoimmune polyendocrinopathy syndrome (APS) - type 2 4. Type 1 diabetes mellitus 5. Type 2 diabetes mellitu

**>>CORRECT-CHOICE LINE**: 3

**>>CORRECT-CHOICE\_TEXT**: Autoimmune polyendocrinopathy syndrome (APS) - type 2

**>>REASONING**: The most likely diagnosis is autoimmune polyendocrinopathy syndrome (APS) type 2, given the combination of findings including suggestive of diabetes (fasting glucose), hypothyroidism (constipation, cold intolerance, dry skin) and hypoaldosteronism (hyponatremia, hyperkalemia, and acidosis).

## Question #:166

**CLINICAL SCENERIO**: A 23-year-old man is diagnosed with type 1 diabetes mellitus after presenting with diabetic ketoacidosis. His blood sugars are now stable.

**QUESTION LINE**: What is the first-line insulin regime he should be offered?

**OPTIONS**: 1. Twice-daily mixed insulin 2. Once-daily mixed insulin 3. Basal-bolus insulin regimen with twice-daily insulin detemir 4. Basal-bolus insulin regimen with once-daily insulin glargine 5. Rapid-acting insulin analogue before each meal with no longer acting insulin

**CORRECT-CHOICE LINE**: c

**REASONING**: In patients with type 1 diabetes, a basal-bolus insulin regimen is the first-line treatment option. This involves using a long-acting insulin to cover the basal insulin requirements and rapid-acting insulin before meals to cover the bolus requirements. Options A and B (mixed insulin) are not the optimal first-line choice as they do not provide the flexibility to adjust insulin doses according to carbohydrate intake and blood glucose levels. Options D (once-daily insulin glargine) and E (rapid-acting insulin analogue before each meal with no longer acting insulin)are not as effective in mimicking the physiological insulin secretion.

**>>DESCRIPTION**: A 23-year-old man with type 1 diabetes mellitus, stable after diabetic ketoacidosis.

**>>OPTIONS**: 1. Basal-bolus insulin regimen with once-daily insulin glargine 2. Basal-bolus insulin regimen with twice-daily insulin detemir 3. Once-daily mixed insulin 4. Rapid-acting insulin analogue before each meal with no longer acting insulin 5. Twice-daily mixed insulin

**>>CORRECT-CHOICE LINE**: 2

**>>CORRECT-CHOICE\_TEXT**: Basal-bolus insulin regimen with twice-daily insulin detemir

**>>REASONING**: Basal-bolus insulin regimen is first-line for type 1 diabetes. Mixed insulin regimens (options 1 & 2) lack flexibility. Options 4 and 5 are not as effective.

## Question #:208

**CLINICAL SCENERIO**: A 40-year-old woman presents with headaches, milky nipple discharge, and absent menstrual periods. Laboratory tests show elevated prolactin and a pituitary adenoma on MRI.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

**OPTIONS**: - a) Bromocriptine - b) Dexamethasone - c) Domperidone - d) Stereotactic radiotherapy - e) Trans-sphenoidal surgery

**CORRECT-CHOICE LINE**: a

**REASONING**: Dopamine agonists (e.g., bromocriptine) are first-line for prolactinomas, even with neurological complications. This patient has a prolactinoma (amenorrhea, galactorrhea, elevated prolactin, pituitary mass). Dexamethasone is not first-line. Domperidone is contraindicated. Radiotherapy and surgery are secondary options.

**>>DESCRIPTION**: A 40-year-old woman with headaches, milky nipple discharge, absent menses, elevated prolactin, and a pituitary adenoma.

**>>OPTIONS**: a) Bromocriptine b) Dexamethasone c) Domperidone d) Stereotactic radiotherapy e) Trans-sphenoidal surgery

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Bromocriptine

**>>REASONING**: Bromocriptine (a dopamine agonist) is the first-line treatment for prolactinomas. Other options are incorrect: Dexamethasone is not first-line, Domperidone is contraindicated, and radiotherapy/surgery are second-line.

## Question #:265

**CLINICAL SCENERIO**: A 23-year-old woman attends a fertility clinic with her partner due to oligomenorrhoea, galactorrhoea, and inability to conceive after 18 months. Blood tests show elevated prolactin (6000 mIU/l) and a pituitary MRI reveals a microprolactinoma.

**QUESTION LINE**: Which of the following is the best initial treatment?

**OPTIONS**: - a. Octreotide - b. Bromocriptine - c. Trans-sphenoidal hypophysectomy - d. Pituitary radiotherapy - e. Transfrontal hypophysectomy

**CORRECT-CHOICE LINE**: B

**REASONING**: Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

This patient has a prolactinoma. In the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. bromocriptine) which inhibit the release of prolactin from the pituitary gland. Surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension. Radiotherapy is rarely performed and octreotide is a somatostatin analogue used in the treatment of acromegaly.

**>>DESCRIPTION**: A 23-year-old woman with oligomenorrhoea, galactorrhoea, and infertility has an elevated prolactin level and a microprolactinoma.

**>>OPTIONS**: a. Bromocriptine b. Octreotide c. Pituitary radiotherapy d. Trans-frontal hypophysectomy e. Trans-sphenoidal hypophysectomy

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Bromocriptine

**>>REASONING**: Bromocriptine, a dopamine agonist, is the first-line treatment for prolactinomas. Other options like surgery or radiotherapy are considered if medical therapy fails, and octreotide is used for acromegaly.

## Question #:235

**CLINICAL SCENERIO**: A 64-year-old man with metastatic prostate cancer and hypercalcaemia, treated with IV fluids, pamidronate, and denosumab, presents with persistent elevated calcium levels and renal function parameters.

**QUESTION LINE**: What is the most appropriate next step in management?

**OPTIONS**: - a) Calcitonin - b) Further dose of denosumab - c) Further dose of pamidronate - d) Indapamide - e) Prednisolone

**CORRECT-CHOICE LINE**: a

**REASONING**: Refractory hypercalcaemia of malignancy may be treated with calcitonin. Calcitonin acts by increasing renal calcium excretion and decreasing bone resorption. Denosumab was given recently, and pamidronate’s effect takes time. Indapamide is contraindicated, and prednisolone is not beneficial for hypercalcaemia.

**>>DESCRIPTION**: A 64-year-old man with metastatic prostate cancer and hypercalcaemia, treated with IV fluids, pamidronate, and denosumab, presents with persistent elevated calcium levels.

**>>OPTIONS**: a) Calcitonin b) Further dose of denosumab c) Further dose of pamidronate d) Indapamide e) Prednisolone

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Calcitonin

**>>REASONING**: Calcitonin is the most appropriate next step for refractory hypercalcemia of malignancy. Denosumab and pamidronate have been recently administered or are not indicated. Indapamide and prednisolone are not appropriate.

## Question #:275

**CLINICAL SCENERIO**: A 50-year-old woman with a history of a thyroid nodule, 6-month history of worsening diarrhea and occasional flushing, RET proto-oncogene mutation, thyroidectomy, and radiotherapy is being followed up in an endocrinology clinic. She is currently on levothyroxine.

**QUESTION LINE**: Which of the following is the most useful test in monitoring for recurrence of this woman’s disease?

**OPTIONS**: - a. Alfa fetoprotein - b. Calcitonin - c. Thyroglobulin - d. Thyroid stimulating hormone - e. Thyroxine

**CORRECT-CHOICE LINE**: b

**REASONING**: Medullary thyroid cancer - calcitonin is used for screening, prognosis and monitoring

Medullary thyroid carcinoma originates from the C cells, or parafollicular cells, which secrete calcitonin. It makes up approximately 5% of thyroid cancers. It may be sporadic, but in 25% of cases, it is associated with RET oncogene, as part of multiple endocrine neoplasia type 2 (MEN2). Despite testing positive for RET oncogene it is possible that other features of MEN2, such as hyperparathyroidism or phaeochromocytoma, do not manifest. Such is the case with this woman.

Also, note that radioiodine was not used in the treatment of her disease. Unlike other thyroid cancers, it is of little benefit in medullary thyroid cancer. Her symptoms of flushing and diarrhoea, are typical of medullary cancer and are caused by calcitonin and calcitonin gene-related peptide. Papillary and follicular cancers are more commonly asymptomatic.

Calcitonin is used for screening, prognosis and monitoring in medullary thyroid cancer. CEA is often used in conjunction with calcitonin but is not an option here.

Thyroglobulin is used in monitoring follicular thyroid cancer.

Thyroxine (T4) and thyroid-stimulating hormone (TSH) are both typically normal in thyroid cancer. This woman has had her thyroid gland removed, and is taking levothyroxine. As such, she would likely be having her TSH monitored. Unlike in other thyroid cancers, TSH suppression is not appropriate for the treatment of medullary thyroid cancer and so thyroxine replacement is monitored similarly to that of hypothyroidism, with a target TSH within the normal range. Her TSH may be monitored, but not to detect recurrence of medullary thyroid cancer.

Alfa fetoprotein is not of use in monitoring thyroid cancer.

**>>DESCRIPTION**: A 50-year-old woman with medullary thyroid cancer (RET mutation, thyroidectomy, radiotherapy) and current levothyroxine is being followed up. She presented with a thyroid nodule and symptoms of diarrhea and flushing.

**>>OPTIONS**: a. Alfa fetoprotein b. Calcitonin c. Thyroid stimulating hormone d. Thyroglobulin e. Thyroxine

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Calcitonin

**>>REASONING**: Calcitonin is the most useful test for monitoring the recurrence of medullary thyroid cancer. Thyroglobulin is used to monitor follicular thyroid cancer. TSH and Thyroxine are monitored for levothyroxine management, not cancer recurrence. Alfa-fetoprotein is not used in monitoring thyroid cancer.

## Question #:297

**CLINICAL SCENERIO**: A 51-year-old woman, two months post-op for medullary thyroid cancer (RET oncogene mutation) with no recurrence signs, is reviewed in clinic.

**QUESTION LINE**: What is the most appropriate test to monitor for recurrence?

**OPTIONS**: - a) Thyroglobulin - b) Thyroid transcription factor- - c) Chromogranin - d) Calcitonin - e) S100 protein

**CORRECT-CHOICE LINE**: Answer is D.

**REASONING**: Medullary thyroid cancer - calcitonin is used for screening, prognosis and monitoring

S100 protein is used in patients with melanoma. Thyroglobulin is used in other types of thyroid malignancy but not medullary thyroid cancer.

**>>DESCRIPTION**: A 51-year-old woman, two months post-op for medullary thyroid cancer (RET mutation) with no recurrence signs.

**>>OPTIONS**: a) Calcitonin b) Chromogranin c) S100 protein d) Thyroid transcription factor- e) Thyroglobulin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Calcitonin

**>>REASONING**: Calcitonin is the primary marker for monitoring medullary thyroid cancer recurrence. Thyroglobulin is used for other thyroid cancers, and S100 protein is for melanoma. Chromogranin is not mentioned.

## Question #:264

**CLINICAL SCENERIO**: A middle-aged woman with symptomatic hypercalcemia due to squamous cell lung cancer (serum calcium 3.60 mmol/L) is slow to respond to saline hydration and intravenous pamidronate. What is the next best step while awaiting surgical resection?

**QUESTION LINE**: What is the most appropriate management?

**OPTIONS**: - a) High dose loop diuretics - b) Calcitonin 4 units/kg - c) Insulin actrapid 50 units in50% dextrose - d) IV colloid administration instead of crystalloid - e) Plasma exchange

**CORRECT-CHOICE LINE**: Answer is B.

**REASONING**: General symptoms of hypercalcaemia may include malaise, lethargy, depression, dehydration and can lead to depressed consciousness. Bone pain and abdominal pain may feature and can be summarised by the classic ‘bones, stones, moans and abdominal groans’.

Alongside searching for the underlying cause, management initially involves aggressive rehydration, typically 4-6 L saline on the first day. Bisphosphonates act by interfering with osteoclastic bone resorption and typically IV pamidronate is used at a dose of 60-90mg over 2-4 hours. Calcitonin (extracted from salmon) also interferes with osteoclast activity as well as increasing renal calcium excretion.

Diuretics may lead to further dehydration. Dialysis may be a last line treatment for life threatening hypercalacemia, but not plasma exchange

**>>DESCRIPTION**: A woman with symptomatic hypercalcemia (3.60 mmol/L) from squamous cell lung cancer has a poor response to hydration and pamidronate. What’s the next best management step pending surgery?

**>>OPTIONS**: a) Calcitonin 4 units/kg b) High dose loop diuretics c) Insulin actrapid 50 units in50% dextrose d) IV colloid administration instead of crystalloid e) Plasma exchange

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Calcitonin 4 units/kg

**>>REASONING**: Calcitonin is the next best step because it inhibits osteoclast activity and increases renal calcium excretion. Loop diuretics could worsen dehydration. Dialysis or plasma exchange are not the first-line treatments.

## Question #:49

**CLINICAL SCENERIO**: A 40-year-old woman presents with a 4-day history of lethargy and weakness, along with intermittent cramps. She had a recent gastrointestinal infection and has coeliac disease and vitiligo. Her medications include folic acid and ferrous sulphate. Observations, cardiorespiratory, and abdominal examinations are unremarkable. Blood tests are requested and an ECG is taken.

**QUESTION LINE**: What is the most appropriate intervention to improve this patient’s symptoms?

**OPTIONS**: - a) 0.9% sodium chloride with potassium supplementation - b) Calcium chlorid - c) Hartmann’s solution - d) Magnesium sulphate - e) Sodium bicarbonate

**CORRECT-CHOICE LINE**: Calcium chloride is correct. This patient has a history of lethargy and muscle cramping along with an ECG that demonstrates a prolonged QT interval. A

combination of these features is highly suspicious for hypocalcaemia. Given the past medical history of coeliac disease, it is quite possibly a malabsorptive cause of hypocalcaemia contributing to this patient’s symptoms. In the context of significant hypocalcaemia, intravenous replacement should be given with either calcium chloride or calcium gluconate.

**REASONING**: 0.9% sodium chloride with potassium supplementation is incorrect. There are no features of hypokalaemia (e.g. U waves) on this patient’s ECG.

Hartmann’s solution is incorrect. There is nothing to suggest that this patient would benefit from intravenous fluid resuscitation.

Magnesium sulphate is incorrect. This is classically used in the management of Torsades de Pointes. However, it is also commonly given in the treatment of other arrhythmias (e.g. atrial fibrillation). Given that this patient has a prolonged QTc interval, and not yet an arrhythmia (such as Torsades de Pointes that is associated with QTc interval prolongation), the priority should be to reduce the QTc interval which is with the administration of intravenous calcium. Furthermore, calcium would also help to improve the patient’s physical symptoms.

Sodium bicarbonate is incorrect. This is not indicated in the management of hypocalcaemia or QTc interval prolongation.

**>>DESCRIPTION**: A 40-year-old woman presents with lethargy, weakness, and intermittent cramps after a recent GI infection. She has coeliac disease, vitiligo, and is on folic acid and ferrous sulphate. Examination is unremarkable. An ECG was taken.

**>>OPTIONS**: a) 0.9% sodium chloride with potassium supplementation b) Calcium chlorid c) Hartmann’s solution d) Magnesium sulphate e) Sodium bicarbonate

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Calcium chlorid

**>>REASONING**: The patient’s lethargy, muscle cramping, and prolonged QT interval on ECG are suspicious for hypocalcaemia, likely due to malabsorption from coeliac disease. IV calcium is indicated. Other options are incorrect because there’s no evidence of hypokalemia, need for fluid resuscitation, or Torsades de Pointes.

## Question #:283

**CLINICAL SCENERIO**: A 53-year-old obese HGV driver with type 2 diabetes, insulin-dependent for 2 years, presents with concerns about driving regulations following a hypoglycemic episode 18 months prior.

**QUESTION LINE**: What is the advice you give him regarding driving?

**OPTIONS**: a. Can continue driving, review in 1 year b. Can continue driving, no further reviews required c. Must stop driving and give up license permanently d. Must stop driving temporarily and review in 6 months e. Patient can drive type 1 vehicles (cars, motorcycles) but not type 2 vehicles (lorries, HGV) and should reconsider his profession

**CORRECT-CHOICE LINE**: A

**REASONING**: The patient, although insulin dependent, retains hypoglycaemia awareness and hasn’t experienced a severe hypoglycemic episode requiring assistance in the past 12 months. The DVLA guidelines (May 2012) allow HGV drivers to retain their license under these conditions, with annual reviews by a diabetes consultant and blood glucose monitoring data.

**>>DESCRIPTION**: A 53-year-old obese HGV driver with type 2 diabetes on insulin presents with concerns about driving regulations following a hypoglycemic episode.

**>>OPTIONS**: a. Can continue driving, review in 1 year b. Can continue driving, no further reviews required c. Must stop driving and give up license permanently d. Must stop driving temporarily and review in 6 months e. Patient can drive type 1 vehicles (cars, motorcycles) but not type 2 vehicles (lorries, HGV) and should reconsider his profession

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Can continue driving, review in 1 year

**>>REASONING**: The patient can continue driving and be reviewed in 1 year because they have hypoglycemia awareness and haven’t had a severe hypoglycemic episode in the last year, according to DVLA guidelines.

## Question #:189

**CLINICAL SCENERIO**: A 65-year-old man presents with a hot, swollen right toe, indicating a possible gout attack. He has a history of alcohol excess and is intolerant of NSAIDs and colchicine. Prednisolone was initiated but led to steroid-induced psychosis.

**QUESTION LINE**: What agent will you consider prescribing?

**OPTIONS**: a) Canakinumab b) Ibuprofen c) Naproxen d) Paracetamol e) Allopurinol

**CORRECT-CHOICE LINE**: a

**REASONING**: Canakinumab is a human monoclonal antibody that selectively inhibits interleukin-1 beta receptor binding. It can be used for treatment of acute gout has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them

The patient is intolerant of NSAIDs therefore ibuprofen and naproxen would be inappropriate.

Paracetamol is a simple analgesic and unlikely to control the patients pain. In addition, the inflammatory nature of gout warrants the use of a drug which reduces inflammation.

Allopurinol is indicated for recurrent gout. It should not be initiated in the acute setting as doing so can prolong the duration and severity of the gout.

**>>DESCRIPTION**: A 65-year-old man with gout (hot, swollen right toe), alcohol history, and intolerance to NSAIDs and colchicine, developed steroid-induced psychosis on prednisolone.

**>>OPTIONS**: a) Allopurinol b) Canakinumab c) Ibuprofen d) Naproxen e) Paracetamol

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Canakinumab

**>>REASONING**: Canakinumab is the correct choice as it’s a treatment option for acute gout when NSAIDs and colchicine are not tolerated. Ibuprofen, and Naproxen are contraindicated due to patient intolerance. Paracetamol is insufficient for inflammation. Allopurinol is for recurrent gout, not acute attacks.

## Question #:216

**CLINICAL SCENERIO**: A 39-year-old woman with recurrent urinary tract infections (UTIs) over six months, treated unsuccessfully with trimethoprim and nitrofurantoin, presents with painful urination and difficulty urinating over the last 2 days. She has a history of type 2 diabetes mellitus, and pain persists after amoxicillin treatment. The HbA1c is 82 mmol/mol.

**QUESTION LINE**: What is the most likely organism causing the recurrent urinary tract infections?

**OPTIONS**: a) Candida b) Neisseria gonorrhoea c) Chlamydia trachomatis d) Trichomonas vaginalis e) Mycoplasma genitalium

**CORRECT-CHOICE LINE**: a

**REASONING**: The most likely organism in this scenario is candida - in those individuals who do have symptomatic Candida infections, the symptoms are indistinguishable from those caused by bacterial infections. Cystitis is associated with dysuria, urgency, suprapubic discomfort. This patients recent symptoms of oliguria, difficultly passing urine and painful urination suggest a complication such as the presence of a fungus ball.

**>>DESCRIPTION**: A 39-year-old woman with recurrent UTIs, treated unsuccessfully and now presents with painful and difficult urination. She has a history of type 2 diabetes mellitus with HbA1c of 82 mmol/mol.

**>>OPTIONS**: a) Candida b) Chlamydia trachomatis c) Mycoplasma genitalium d) Neisseria gonorrhoea e) Trichomonas vaginalis

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Candida

**>>REASONING**: Candida is the most likely cause given the patient’s symptoms and history of diabetes. The symptoms suggest a potential complication like a fungal ball.

## Question #:246

**CLINICAL SCENERIO**: A 42-year-old man presents with a 3-month history of increasing anxiety, 6 kg weight loss in 2 months, increased bowel movements, and diarrhea. Blood tests show abnormal thyroid function.

**QUESTION LINE**: What is the most appropriate treatment?

**OPTIONS**: - a. Reassurance - b. Carbimazole - c. Radio-iodine - d. Surgery - e. Propranolol

**CORRECT-CHOICE LINE**: B

**REASONING**: The diagnosis in this scenario is triiodothyronine thyrotoxicosis. A small subset of those patients experiencing thyrotoxicosis (roughly 5%) have isolated triiodothyronine thyrotoxicosis. As with other types of thyrotoxicosis, carbimazole is the main initial treatment for the condition

**>>DESCRIPTION**: A 42-year-old man presents with anxiety, weight loss, increased bowel movements, and diarrhea. Blood tests indicate abnormal thyroid function.

**>>OPTIONS**: a. Carbimazole b. Propranolol c. Radio-iodine d. Reassurance e. Surgery

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Carbimazole

**>>REASONING**: The patient has triiodothyronine thyrotoxicosis. Carbimazole is the primary initial treatment.

## Question #:50

**CLINICAL SCENERIO**: A 80-year-old patient was referred to Accident and Emergency after being found unresponsive in his home. He had just completed a course of antibiotics for a chest infection. He had not been seen for the preceding 36 hours. He had a past medical history of hypertension and type two diabetes.

His medication included Metformin, Gliclazide, Humulin M3 insulin twice a day, Ramipril and Bendroflumethiazide.

His initial examination revealed. Blood pressure 104/53, heart rate 103 beats per minute, respiratory rate 24 and oxygen saturations 90% on air. He had inspiratory crackles on his left lower lung zone. He had sunken eyes, capillary refill time of four seconds and no lower limb swelling. GCS 13 out of 15.

## Initial blood tests;

| Hb | 11.0 g/dL |
| --- | --- |
| WCC | 21.4 \*10^9/l |
| Platelets | 189 \*10^9/l |
| CRP | 340 mg/L |
| Na+ | 149 mmol/l |
| K+ | 4.4mmol/l |
| Ur | 28 mmol/l |
| Cr | 180 µmol/l |
| Glucose | 54mmol/l |

ABG on air

| pH | 7.32 |
| --- | --- |
| pCO2 | 3.7kPa |
| pO2 | 9kPa |
| HCO3 | 18 mmol/l |

Urine dipstick analysis - ++ glucose, - WCC, - leucocytes, + ketones

The patient was treated with oxygen, intravenous antibiotics for a chest infection and prophylactic low molecular weight heparin. They were treated with the local diabetic ketoacidosis protocol with IV inulin sliding scale and IV fluids 5500ml in 24 hours.

His repeat bloods 12 hours later were;

| Na+ | 132 mmol/l |
| --- | --- |
| K+ | 3.9 mmol/l |
| Ur | 12 mmol/l |
| Cr | 110 µmmol/l |
| Glucose | 5 mmol/l |
| HCO3 | 24 mmol/l |
| Lactate | 1.7 mmol/l |
| CRP | 270mg/l |

The patient developed a grand mal seizure. His Glasgow coma scale remained 10 an hour after the seizure.

**QUESTION LINE**: What is the most likely cause of his neurological deterioration?

**OPTIONS**: - a) Hypoglycaemia - b) Intracranial venous sinus thrombosis - c) Cerebral oedema9 - d) Sepsis - e) Renal failure

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: All the answers are potentially associated with hyperosmolar hyperglycaemic coma. The current guidance advises treatment initially with normal saline intravenous rehydration. The target reduction in osmolality is 3-8 mosmol/kg an hour. Only if this target is not being met and the glucose level not reducing sufficiently is insulin to be started.

This patient developed cerebral oedema secondary to rapid reduction in serum osmolality.

**>>DESCRIPTION**: 80-year-old with hypertension and type 2 diabetes, found unresponsive after completing antibiotics for a chest infection. Medications include Metformin, Gliclazide, Humulin M3, Ramipril, and Bendroflumethiazide. Examination: BP 104/53, HR 103, RR 24, SpO2 90%, inspiratory crackles, sunken eyes, 4-second capillary refill, GCS 13/15. Labs: Hyperglycemia, hypernatremia, elevated WCC and CRP, ketones in urine. Treated for DKA. Later developed a seizure, GCS 10.

**>>OPTIONS**: a) Cerebral oedema b) Hypoglycaemia c) Intracranial venous sinus thrombosis d) Renal failure e) Sepsis

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cerebral oedema

**>>REASONING**: Cerebral edema is the most likely cause due to the rapid reduction in serum osmolality during treatment for hyperosmolar hyperglycemic coma. The other options are less likely given the clinical context and the treatment received.

## Question #:227

**CLINICAL SCENERIO**: A 72-year-old woman is recovering on the neurosurgical unit following a subdural haemorrhage and Burr hole surgery. She has persistently low sodium levels post-surgery.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) SIADH - b) Diabetes insipidus - c) Cerebral salt wasting syndrome - d) Renal tubular acidosis type IV - e) Sheehan’s syndrome

**CORRECT-CHOICE LINE**: c

**REASONING**: Diabetes insipidus is classically associated with hypernatraemia. Sheehan’s syndrome refers to the specific situation of pituitary necrosis following childbirth. The cardinal feature of renal tubular acidosis type IV is hyperkalaemia.

This leaves SIADH and cerebral salt wasting syndrome. The hydration status in this patient can be considered hypovolaemic making SIADH unlikely (typically euvolaemic). Additionally, this diagnosis should only be made in the absence of hypothyroidism and adrenal dysfunction.

Cerebral salt wasting syndrome can occur following neurosurgery. It occurs due to sodium wasting in the urine. Comparatively, it is treated with replacing fluid and sodium losses.

**>>DESCRIPTION**: A 72-year-old woman post-subdural haemorrhage and Burr hole surgery presents with persistent hyponatremia.

**>>OPTIONS**: a) Cerebral salt wasting syndrome b) Diabetes insipidus c) Renal tubular acidosis type IV d) SIADH e) Sheehan’s syndrome

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cerebral salt wasting syndrome

**>>REASONING**: The most likely diagnosis is cerebral salt wasting syndrome, which can occur after neurosurgery, due to sodium wasting in the urine, and resulting hypovolemia and hyponatremia. SIADH is less likely due to the patient’s hypovolemic state. Diabetes insipidus is associated with hypernatremia, Sheehan’s syndrome is postpartum pituitary necrosis, and renal tubular acidosis type IV is characterized by hyperkalemia.

## Question #:178

**CLINICAL SCENERIO**: A 78-year-old female with type 2 diabetes, gastroparesis, and chronic kidney disease presents with recurrent nausea, vomiting, recent weight loss, and morning falls. Her HbA1c is 6.2%, and her blood sugar readings vary, including both low and high values. She is on multiple medications, including Humulin M3 and metformin.

**QUESTION LINE**: What is the correct step in the management of her diabetes?

**OPTIONS**: a) Change Humulin M3 to a glucagon-like-peptide 1 receptor agonist b) Stop metformin c) Add sitagliptin d) Change Humulin M3 to 20 units in the morning and 10 units in the evening e) Change Humulin M3 to 30 units once daily

**CORRECT-CHOICE LINE**: e

**REASONING**: The patient’s fasting blood sugars are too low, with some high readings suggesting potential overnight hypoglycemia. Due to recent weight loss and a lower HbA1c, her insulin resistance has decreased. Reducing the total daily insulin dose from 44 to 30 units helps prevent hypoglycemia. There’s no indication for a GLP1 agonist. Metformin should continue assuming eGFR remains above 30.

**>>DESCRIPTION**: A 78-year-old female with type 2 diabetes, gastroparesis, CKD, weight loss, and variable blood sugars presents with recurrent nausea and morning falls. HbA1c is 6.2% on multiple medications.

**>>OPTIONS**: a) Add sitagliptin b) Change Humulin M3 to 20 units in the morning and 10 units in the evening c) Change Humulin M3 to 30 units once daily d) Change Humulin M3 to a glucagon-like-peptide 1 receptor agonist e) Stop metformin

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Change Humulin M3 to 30 units once daily

**>>REASONING**: The patient’s variable blood sugars and weight loss indicate a need to adjust insulin dosage. Reducing Humulin M3 to 30 units once daily is the most appropriate initial step to address potential hypoglycemia and decreased insulin resistance. Other options are not appropriate.

## Question #:325

**CLINICAL SCENERIO**: A 32-year-old patient, diagnosed with type 1 diabetes mellitus, is being discharged after his third DKA admission in two years. The patient has resumed his insulin regimen (detemir and aspart) after recovering. DKA was likely triggered by alcohol consumption and missed insulin doses. The patient drinks moderately but occasionally binge drinks. No other health issues or medications are noted. What is the best recommendation to prevent future DKA admissions?

**QUESTION LINE**: What is the most appropriate suggestion to avoid further admissions with DKA?

**OPTIONS**: - f) Start metformin - g) Change insulin detemir (Levemir) to insulin degludec (Tresiba) - h) Change insulin detemir (Levemir) to insulin aspart biphasic (Novomix 30) - i) Increase dose in insulin aspart (Novorapid) by 20% - j) Advise reduction in carbohydrates in diet

**CORRECT-CHOICE LINE**: b

**REASONING**: A patient with recurrent admissions for DKA can be started on degludec to reduce readmission rate

This is a patient with type 1 diabetes with recurrent DKA secondary to a combination of missing doses of insulin and binge drinking alcohol. The most appropriate and likely to be successful strategy is to change Levemir to Degludec. Degludec has a much higher half-life than Levemir and therefore maintains a basal insulin level when the patient omits or forgets doses. This can prevent DKA. Metformin is not indicated for this patient. Insulin aspart biphasic (Novomix 30) would allow reduction in the number of insulin injections. Increasing the dose of insulin aspart (Novorapid) is unlikely to prvent DKA for this patient as the fundamental issue is missing doses, and the short-term nature of the insulin aspart (Novorapid) is unlikely to provide any insulin cover for further omitted doses. Patients with type 1 diabetes can also benefit from DAFNE, a course which helps patients adjust their insulin doses to what they are eating and the patient should be offered this as an educational tool as well to improve control.

**>>DESCRIPTION**: A 32-year-old type 1 diabetic patient is being discharged after a third DKA admission, likely due to missed insulin doses and alcohol. The patient is on detemir and aspart insulin. What is the best approach to prevent future DKA admissions?

**>>OPTIONS**: a) Advise reduction in carbohydrates in diet b) Change insulin aspart biphasic (Novomix 30) c) Change insulin detemir (Levemir) to insulin degludec (Tresiba) d) Increase dose in insulin aspart (Novorapid) by 20% e) Start metformin

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Change insulin detemir (Levemir) to insulin degludec (Tresiba)

**>>REASONING**: Switching to insulin degludec (Tresiba) is the best option because it provides a longer half-life, offering more consistent basal insulin coverage and reducing the risk of DKA from missed doses. Other options are less effective or not appropriate for this patient.

## Question #:19

**CLINICAL SCENERIO**: A 62-year-old man with a 7-year history of Type 2 diabetes, treated with metformin, sitagliptin, and empagliflozin, presents to the Emergency department with worsening nausea and vomiting over 2-3 weeks and a 5kg weight loss in the past month. Examination reveals BP 110/65 mmHg, pulse 85 bpm, elevated ketones, and glucose of 12.2 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate way to manage his glucose control?

**OPTIONS**: - a) Add liraglutide - b) Add long-acting insulin - c) Change the empagliflozin for liraglutide - d) Change the empagliflozin for long-acting insulin - e) Stop the metformin

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Given the duration of Type 2 diabetes and the fact that patient has lost weight in the past month, the possibility that he is insulinopenic is raised. In this situation, calorie loss and metabolic disturbance can be exacerbated by the use of SGLT-2 inhibitors and patients may present as here, with euglycaemic ketoacidosis. The SGLT-2 inhibitor should be withdrawn, and given he is insulinopaenic, long-acting insulin added.

In this situation the empagliflozin must be withdrawn, therefore options including adding liraglutide and long-acting insulin are incorrect. GLP-1 agonists such as liraglutide work less well in patients who are relatively insulinopenic, so liraglutide is incorrect. Stopping the metformin won’t remove the cause of ketosis, the empagliflozin.

**>>DESCRIPTION**: A 62-year-old man with T2DM on metformin, sitagliptin, and empagliflozin presents with worsening nausea/vomiting and 5kg weight loss. Exam: BP 110/65, HR 85, elevated ketones, glucose 12.2 mmol/l.

**>>OPTIONS**: a) Add liraglutide b) Change the empagliflozin for liraglutide c) Change the empagliflozin for long-acting insulin d) Stop the metformin e) Add long-acting insulin

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Change the empagliflozin for long-acting insulin

**>>REASONING**: The patient is likely insulinopenic. SGLT-2 inhibitors exacerbate calorie loss and metabolic disturbance, leading to euglycaemic ketoacidosis. Empagliflozin should be stopped and long-acting insulin added. Liraglutide is less effective in insulinopenic patients, and stopping metformin alone won’t address the ketosis caused by empagliflozin.

## Question #:338

**CLINICAL SCENERIO**: A 62-year-old man presents to the Emergency department with worsening nausea, vomiting, and 5kg weight loss over 2-3 weeks. He has Type 2 diabetes treated with metformin, sitagliptin, and empagliflozin. Examination reveals blood pressure 110/65 mmHg, pulse 85 bpm, elevated ketones, and glucose of 12.2 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate way to manage his glucose control?

**OPTIONS**: - f) Add liraglutide - g) Add long-acting insulin - h) Change the empagliflozin for liraglutide - i) Change the empagliflozin for long-acting insulin - j) Stop the metformin

**CORRECT-CHOICE LINE**: d

**REASONING**: Given the duration of Type 2 diabetes and the fact that patient has lost weight in the past month, the possibility that he is insulinopenic is raised. In this situation, calorie loss and metabolic disturbance can be exacerbated by the use of SGLT-2 inhibitors and patients may present as here, with euglycaemic ketoacidosis. The SGLT-2 inhibitor should be withdrawn, and given he is insulinopaenic, long-acting insulin added.

In this situation the empagliflozin must be withdrawn, therefore options including adding liraglutide and long-acting insulin are incorrect. GLP-1 agonists such as liraglutide work less well in patients who are relatively insulinopenic, so liraglutide is incorrect. Stopping the metformin won’t remove the cause of ketosis, the empagliflozin.

**>>DESCRIPTION**: A 62-year-old man with Type 2 diabetes (metformin, sitagliptin, empagliflozin) presents with worsening nausea, vomiting, 5kg weight loss, elevated ketones, and a glucose of 12.2 mmol/l.

**>>OPTIONS**: a) Add liraglutide b) Add long-acting insulin c) Change the empagliflozin for liraglutide d) Change the empagliflozin for long-acting insulin e) Stop the metformin

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Change the empagliflozin for long-acting insulin

**>>REASONING**: The patient likely has euglycemic ketoacidosis due to insulinopenia and SGLT-2 inhibitor use. The correct approach is to change the empagliflozin for long-acting insulin. Other options are incorrect because they do not address the underlying cause or are less effective in insulinopenic states.

## Question #:196

**CLINICAL SCENERIO**: A 75-year-old lady presents to the acute medical unit after exposure to chicken pox. She has heart failure, giant cell arteritis, and takes prednisolone, ramipril and bisoprolol. She has been taking 40mg prednisolone for two weeks.

**QUESTION LINE**: How will you manage this patient?

**OPTIONS**: a) Intravenous ganciclovir b) Intravenous acyclovir c) Oral acyclovir d) Check Varicella Zoster antibodies e) Varicella Zoster immunoglobulin

**CORRECT-CHOICE LINE**: d

**REASONING**: This patient, on high-dose prednisolone, is immunocompromised and exposed to varicella. Checking for Varicella Zoster antibodies is crucial to determine the need for VZIG. VZIG prophylaxis is recommended for immunosuppressed patients exposed to the virus who lack antibodies. Intravenous ganciclovir and acyclovir are not the primary management strategies in this scenario and oral acyclovir is not the immediate step.

**>>DESCRIPTION**: A 75-year-old lady presents to the acute medical unit after exposure to chicken pox and is on 40mg prednisolone for two weeks.

**>>OPTIONS**: a) Check Varicella Zoster antibodies b) Intravenous acyclovir c) Intravenous ganciclovir d) Oral acyclovir e) Varicella Zoster immunoglobulin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Check Varicella Zoster antibodies

**>>REASONING**: The patient is immunocompromised due to high-dose steroids and exposed to varicella. The initial step is to check for Varicella Zoster antibodies to guide the need for VZIG. Incorrect choices offer other treatment options but not the immediate diagnostic strategy.

## Question #:205

**CLINICAL SCENERIO**: A 47-year-old female presents with recurrent palpitations, sweating, blurring of vision, and generalized weakness, with episodes occurring in the morning or before meals, relieved by eating. She has gained weight, has a history of anxiety but is not on medications, and her current blood glucose is 4.9 mmol. Insulinoma is suspected.

**QUESTION LINE**: What is the next step in the evaluation of this patient?

**OPTIONS**: - a) Check insulin level - b) Check pro-insulin level - c) CT abdomen - d) Screening for sulphonylurea drugs - e) Check blood glucose during or after the episode to confirm hypoglycemia Correct answer is e.

**CORRECT-CHOICE LINE**: e

**REASONING**: Completion of Whipple’s triad is required before further investigations for insulinoma. The best next step is to confirm hypoglycemia by checking blood glucose. All other options are relevant but not the immediate next step.

**>>DESCRIPTION**: A 47-year-old female presents with recurrent palpitations, sweating, vision blurring, and weakness, with episodes before meals relieved by eating. Insulinoma is suspected.

**>>OPTIONS**: a) Check blood glucose during or after the episode to confirm hypoglycemia b) Check insulin level c) Check pro-insulin level d) CT abdomen e) Screening for sulphonylurea drugs

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Check blood glucose during or after the episode to confirm hypoglycemia

**>>REASONING**: The next step is to confirm hypoglycemia by checking blood glucose to complete Whipple’s triad. The other options are relevant to diagnosis but not the initial step.

## Question #:228

**CLINICAL SCENERIO**: A 38-year-old woman with Graves’ disease (goitre, suppressed TSH, thyroid antibodies), no significant past medical history, drinks 10 units alcohol/week and smokes 20 cigarettes/day. Examination: BP 112/88 mmHg, pulse 89 bpm, fine tremor, smooth goitre, marked proptosis.

**QUESTION LINE**: Which of the following has the greatest negative impact on prognosis of her thyroid eye disease?

**OPTIONS**: a) Alcohol consumption b) Cigarette smoking c) DR4 HLA type d) LATS titre e) Use of block replace therapy

**CORRECT-CHOICE LINE**: b

**REASONING**: Smoking is the most important modifiable risk factor for the development of thyroid eye disease

A systematic review published in 2006 has confirmed the strong link between cigarette smoking and thyroid eye disease. Across 15 studies a strong association between thyroid eye disease in patients with Graves’ disease and smoking was established, with an odds ratio of up to 20 for thyroid eye disease in current smokers vs non-smokers who have Graves’.

http://www.ncbi.nlm.nih.gov/pubmed/16980921

Block replace therapy establishes stable control of thyroid function, and is actually associated with reduced incidence of thyroid eye disease because thyroxine is consistently in the normal range. Alcohol consumption within the recommended safe limits may actually reduce the severity of thyroid eye disease in Graves’. Thyroid eye disease is primarily driven by pathogenic T cells, as such it isn’t closely related to LATS titre. HLA DR4 is more strongly associated with Type 1 diabetes, rheumatoid arthritis and autoimmune hepatitis than with thyroid disease.

**>>DESCRIPTION**: A 38-year-old woman with Graves’ disease (goitre, suppressed TSH, thyroid antibodies) and smoking history presents with thyroid eye disease. Which factor has the greatest negative impact on prognosis?

**>>OPTIONS**: a) Alcohol consumption b) Cigarette smoking c) DR4 HLA type d) LATS titre e) Use of block replace therapy

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Cigarette smoking

**>>REASONING**: Cigarette smoking is the most significant modifiable risk factor, strongly linked to the development and progression of thyroid eye disease. Other options are incorrect: Block replace therapy is associated with reduced incidence. Alcohol consumption within recommended limits may reduce severity. LATS titre and HLA DR4 have less impact.

## Question #:177

**CLINICAL SCENERIO**: A 32-year-old overweight female presents to the infertility clinic with an inability to conceive, irregular periods, hirsutism, and a deepening voice. Pelvic ultrasound reveals multiple ovarian cysts. Examination shows a cushingoid appearance and hypertension. Laboratory investigations reveal elevated cortisol, LH, DHEAS, and prolactin levels. The patient wishes to conceive.

**QUESTION LINE**: Which of the following treatment options would be most appropriate for the treatment of infertility?

**OPTIONS**: 1. Metformin 2. Spironolactone 3. Reverse circadian rhythm steroids 4. Clomiphene citrate 5. Cabergoline

**CORRECT-CHOICE LINE**: 4

**REASONING**: Clomiphene citrate is superior to metformin in achieving live birth in infertile women with polycystic ovary syndrome. Metformin is an option, but Clomiphene is more effective. Spironolactone is for hirsutism, reverse circadian rhythm steroids are for congenital adrenal hyperplasia, and cabergoline is for hyperprolactinemia.

**>>DESCRIPTION**: A 32-year-old overweight female presents to an infertility clinic with infertility, irregular periods, hirsutism, deepening voice, and multiple ovarian cysts. Lab results show elevated cortisol, LH, DHEAS, and prolactin. She wishes to conceive.

**>>OPTIONS**: 1. Cabergoline 2. Clomiphene citrate 3. Metformin 4. Reverse circadian rhythm steroids 5. Spironolactone

**>>CORRECT-CHOICE LINE**: 2

**>>CORRECT-CHOICE\_TEXT**: Clomiphene citrate

**>>REASONING**: Clomiphene citrate is the most effective treatment for infertility in women with PCOS. Metformin is an option, but less effective. Other options are not relevant to treating infertility in this context.

## Question #:67

**CLINICAL SCENERIO**: A 19-year-old woman presents with hirsutism, irregular periods, and weight gain. Her GP advised weight loss without medication. She is a law student with a BP of 135/85 mmHg, pulse 65 bpm, and BMI 32kg/m². Examination reveals hirsutism (beard line, upper lip, nipples) and acne on the face and upper chest. Labs: testosterone 4.8 nmol/l (ULN 2.1 nmol/l), LH:FSH ratio 2.1, fasting glucose 5.0 mmol/l. Her main concern is hirsutism.

**QUESTION LINE**: Which of the following is the most appropriate intervention?

**OPTIONS**: a) Co-cyprindiol b) Clomiphene c) Levonorgestrel d) Metformin e) Pioglitazone

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Co-cyprindiol contains cyproterone (anti-androgen) and ethinylestradiol (synthetic oestrogen). It is used for polycystic ovarian syndrome, reducing hirsutism and acne caused by androgen excess. Clomiphene is used for inducing ovulation, sometimes with metformin in obese patients. Pioglitazone reduces ovarian insulin resistance but has adverse effects. Levonorgestrel is ineffective for hirsutism.

**>>DESCRIPTION**: 19-year-old female with hirsutism, irregular periods, weight gain, BP 135/85, BMI 32. Hirsutism on face/chest, testosterone 4.8 nmol/l (ULN 2.1), LH:FSH 2.1. Main concern: hirsutism.

**>>OPTIONS**: a) Clomiphene b) Co-cyprindiol c) Levonorgestrel d) Metformin e) Pioglitazone

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Co-cyprindiol

**>>REASONING**: Co-cyprindiol, containing an anti-androgen, is most appropriate for managing hirsutism and acne in PCOS due to androgen excess. Clomiphene induces ovulation; pioglitazone has adverse effects; levonorgestrel is ineffective.

## Question #:81

**CLINICAL SCENERIO**: You order an oral glucose tolerance test which is as follows:

| Time (mins) | 0 | 30 | 60 | 90 | 120 |
| --- | --- | --- | --- | --- | --- |
| Growth hormone (ng/ml) | 2.2 | 2.4 | 3.1 | 3.6 | 3.7 |

**QUESTION LINE**: Given the likely diagnosis, what other investigation will be required in the future?

**OPTIONS**: - a) Transesophageal echocardiography - b) Colonoscopy - c) Flexible sigmoidoscopy - d) CT colonography - e) No further investigations required

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly

The clinical features and results of the oral glucose tolerance test confirm a diagnosis of acromegaly. In normal a rise in glucose should cause suppression of growth hormone (GH) to undetectable values.

Cardiovascular disease is highly prevalent in Acromegaly due to a combination of risk factors associated with this disease, in particular, secondary diabetes mellitus, hypertension and hyperlipidaemia. Patients should therefore be screened for cardiovascular risk factors and these should be managed aggressively. A transthoracic echo would be prudent to perform, however transesophageal echocardiography is unnecessary.

Patients with acromegaly have high levels of insulin like growth factor which predisposes to malignancy, in particularly colorectal carcinoma. Colonoscopy, flexible sigmoidoscopy and CT colonography can all be used to screen for colorectal carcinoma, however colonoscopy is the most sensitive test making this the best answer.

Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly.

**>>DESCRIPTION**: Oral glucose tolerance test shows elevated growth hormone levels. Likely diagnosis?

**>>OPTIONS**: a) Colonoscopy b) CT colonography c) Flexible sigmoidoscopy d) No further investigations required e) Transesophageal echocardiography

**>>CORRECT-CHOICE LINE**: Correct answer is a.

**>>CORRECT-CHOICE\_TEXT**: Colonoscopy

**>>REASONING**: The clinical features and OGTT results suggest acromegaly. Patients with acromegaly are at increased risk of colorectal carcinoma due to elevated insulin-like growth factor. Colonoscopy is the most sensitive screening test for colorectal cancer. Other options are less sensitive or not indicated.

## Question #:213

**CLINICAL SCENERIO**: A 17-year-old woman presents with amenorrhea and has never had a menstrual cycle. Examination reveals little axillary and pubic hair, bilateral groin swellings, and a normal vagina and labia. Blood results show elevated testosterone, FSH, and LH.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Complete androgen insensitivity syndrome (CAIS) - b) Kallmann’s syndrome - c) Mullerian agenesis - d) Partial androgen insensitivity syndrome (PAIS) - e) Turner’s syndrome

**CORRECT-CHOICE LINE**: a

**REASONING**: Primary amenorrhea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome. Complete androgen insensitivity syndrome (CAIS) is correct. The clinical features of primary amenorrhea, lack of secondary sexual characteristics, and groin swellings (suggestive of undescended testes) are suggestive of androgen insensitivity syndrome. The hormonal profile findings of very high testosterone, elevated LH, and a relatively normal FSH are classical findings of partial androgen insensitivity syndrome. The presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome.

Kallmann’s syndrome is incorrect. Kallmann’s syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. Cryptorchidism can also occur. However, the biochemical profile, in this case, is not in keeping with Kallmann’s syndrome - which would present with low sex hormone levels, and inappropriately low/normal LH, and FSH levels.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Both Mullerian agenesis and androgen insensitivity syndrome can present with primary amenorrhea, shortened vagina, and absent cervix. However, in patients with androgen insensitivity, the gonads are testes, which produce normal androgens. The presence of bilateral groin swellings (indicative of undescended testes) makes Mullerian agenesis a less likely diagnosis.

Partial androgen insensitivity syndrome (PAIS) is incorrect. The clinical and biochemical features are suggestive of androgen insensitivity syndrome. However the presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome. PAIS results in a spectrum of disorders that ranges from a female phenotype with clitoromegaly and/or minimal posterior labial fusion (Lub syndrome) to genital ambiguity to an unequivocally male phenotype with a minor defect such as isolated hypospadias or azoospermia.

Turner’s syndrome is incorrect. Turner’s syndrome can cause primary ovarian failure due to gonadal dysgenesis. However, the biochemical profile, in this case, is not in keeping with Turner’s syndrome - which would present with low sex hormone levels, and markedly raised LH and FSH levels.

**>>DESCRIPTION**: A 17-year-old woman with primary amenorrhea, little axillary/pubic hair, bilateral groin swellings, and a normal vagina/labia has elevated testosterone, FSH, and LH.

**>>OPTIONS**: a) Complete androgen insensitivity syndrome (CAIS) b) Kallmann’s syndrome c) Mullerian agenesis d) Partial androgen insensitivity syndrome (PAIS) e) Turner’s syndrome

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Complete androgen insensitivity syndrome (CAIS)

**>>REASONING**: The most likely diagnosis is Complete androgen insensitivity syndrome (CAIS) due to primary amenorrhea, lack of secondary sexual characteristics, groin swellings, and the hormonal profile. Other options are less likely due to differences in clinical presentation or hormonal findings.

## Question #:106

**CLINICAL SCENERIO**: A 52-year-old man presents with a 1-month history of polyuria and polydipsia. He has no significant past medical history and takes no regular medications. Blood results show elevated Na, Urea, and Creatinine. Osmolarity studies after water deprivation show low urine osmolarity and high plasma osmolarity. After desmopressin, urine osmolarity increases, and plasma osmolarity decreases.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Complete cranial diabetes insipidus - b) Nephrogenic diabetes insipidus - c) Partial cranial diabetes insipidus - d) Primary polydipsia - e) Syndrome of inappropriate antidiuretic hormone release

**CORRECT-CHOICE LINE**: Complete cranial diabetes insipidus is correct.

**REASONING**: Complete cranial diabetes insipidus is correct. The water deprivation test confirms a diagnosis of diabetes insipidus (DI) due to a failure of the urine to concentrate with dehydration (urine osmolarity <300 mmol/L). Following administration of desmopressin there is a >50% rise in urine osmolarity confirming the diagnosis of cranial DI.

Partial cranial diabetes insipidus is incorrect. This subtype of cranial DI is confirmed when the patient’s symptoms resolve with desmopressin however the urine osmolarity fails to meet the diagnostic criteria (e.g. >50% increase).

Nephrogenic diabetes insipidus is incorrect. This condition is characterised by the failure of the urine to concentrate with desmopressin.

Primary polydipsia is incorrect. This condition is characterised by a normal physiological response to water deprivation e.g. the urine will concentrate (>600

mmol/L).

Syndrome of inappropriate antidiuretic hormone release is incorrect. Syndrome of inappropriate antidiuretic hormone release (SIADH) is associated with hyponatraemia, a decreased plasma osmolarity (<275 mOsm/kg), a relatively low urine osmolarity (< 100 mOsm/kg), and a high urinary sodium (> 40 mmol/L).

**>>DESCRIPTION**: A 52-year-old male with polyuria and polydipsia. Labs show elevated Na, Urea, and Creatinine. Water deprivation leads to low urine osmolarity and high plasma osmolarity, which improves with desmopressin.

**>>OPTIONS**: a) Complete cranial diabetes insipidus b) Nephrogenic diabetes insipidus c) Partial cranial diabetes insipidus d) Primary polydipsia e) Syndrome of inappropriate antidiuretic hormone release

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Complete cranial diabetes insipidus

**>>REASONING**: Complete cranial DI is correct because desmopressin administration leads to a significant (>50%) rise in urine osmolarity, indicating a response to ADH. Nephrogenic DI would not respond to desmopressin. SIADH would present with hyponatremia.

## Question #:331

**CLINICAL SCENERIO**: A 52-year-old man presents to the endocrine clinic with a 1-month history of polyuria and polydipsia. He has no significant past medical history and takes no regular medications. Blood results are as follows:

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: f) Complete cranial diabetes insipidus g) Nephrogenic diabetes insipidus h) Partial cranial diabetes insipidus i) Primary polydipsia j) Syndrome of inappropriate antidiuretic hormone release

**CORRECT-CHOICE LINE**: f

**REASONING**: Complete cranial diabetes insipidus is correct. The water deprivation test confirms a diagnosis of diabetes insipidus (DI) due to a failure of the urine to concentrate with dehydration (urine osmolarity <300 mmol/L). Following administration of desmopressin there is a >50% rise in urine osmolarity confirming the diagnosis of cranial DI. Partial cranial diabetes insipidus is incorrect. This subtype of cranial DI is confirmed when the patient’s symptoms resolve with desmopressin however the urine osmolarity fails to meet the diagnostic criteria (e.g. >50% increase). Nephrogenic diabetes insipidus is incorrect. This condition is characterised by the failure of the urine to concentrate with desmopressin. Primary polydipsia is incorrect. This condition is characterised by a normal physiological response to water deprivation e.g. the urine will concentrate (>600 mmol/L). Syndrome of inappropriate antidiuretic hormone release is incorrect. Syndrome of inappropriate antidiuretic hormone release (SIADH) is associated with hyponatraemia, a decreased plasma osmolarity (<275 mOsm/kg), a relatively low urine osmolarity (< 100 mOsm/kg), and a high urinary sodium (> 40 mmol/L).

**>>DESCRIPTION**: A 52-year-old man presents with polyuria and polydipsia. Blood and osmolarity results after water deprivation are provided.

**>>OPTIONS**: a) Complete cranial diabetes insipidus b) Nephrogenic diabetes insipidus c) Partial cranial diabetes insipidus d) Primary polydipsia e) Syndrome of inappropriate antidiuretic hormone release

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Complete cranial diabetes insipidus

**>>REASONING**: The correct diagnosis is complete cranial diabetes insipidus because of the low urine osmolarity after water deprivation and the increase in urine osmolarity following desmopressin administration. Other options are incorrect because they don’t align with these findings. Partial cranial DI shows an incomplete response to desmopressin. Nephrogenic DI doesn’t respond to desmopressin. Primary polydipsia shows appropriate urine concentration during water deprivation. SIADH presents with hyponatremia.

## Question #:222

**CLINICAL SCENERIO**: A 28-year-old woman with refractory hypertension despite combination therapy. Blood pressure in clinic is 181/105 mmHg. She admits to passing urine more than 10 times per day. Blood results are provided.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Phaeochromocytoma b) Coarctation of the aorta c) Renal artery stenosis d) 21-hydroxylase deficiency e) Conn’s syndrome

**CORRECT-CHOICE LINE**: e.

**REASONING**: All of the answers above are causes of secondary hypertension, except for 21hydroxylase deficiency which accounts for over 90% of congenital adrenal hyperplasia.

The high sodium and low potassium in the bloods reflects the increased levels of aldosterone produced in Conn’s syndrome. Aldosterone stimulates the Na+/K+ATPase membrane transporter in the distal convoluted tubule resulting in increased sodium reabsorption and potassium excretion. Polyuria and polydipsia can result from the kidneys inability to concentrate urine.

The diagnosis is usually confirmed using plasma aldosterone:renin ratio

**>>DESCRIPTION**: A 28-year-old woman with refractory hypertension and polyuria, despite combination therapy. Blood pressure is 181/105 mmHg. Blood results are provided.

**>>OPTIONS**: a) Coarctation of the aorta b) Conn’s syndrome c) Phaeochromocytoma d) Renal artery stenosis e) 21-hydroxylase deficiency

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Conn’s syndrome

**>>REASONING**: Conn’s syndrome is the most likely diagnosis due to refractory hypertension, polyuria, hypernatremia, and hypokalemia. The high aldosterone levels in Conn’s syndrome cause sodium retention and potassium excretion. Other options are causes of secondary hypertension, but less likely based on the given data.

## Question #:51

**CLINICAL SCENERIO**: A 42-year-old lady, post-resection for thyroid cancer, on levothyroxine 100mcg daily, presents with TSH < 0.1 mU/l. She is asymptomatic.

**QUESTION LINE**: What is the best course of action?

**OPTIONS**: a) Stop levothyroxine b) Continue at 100mcg per day c) Repeat thyroid function in 6 weeks d) Change to liothyronine equivalent dose e) Reduce the levothyroxine as she is over treated

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: As TSH is a growth factor for many thyroid cancers it is routinely suppressed with levothyroxine. TSH and thyroglobulin should be monitored in patients with a history of thyroid cancer and decisions on the level required are usually specialist led.

High and intermediate risk patients should have their TSH suppressed bellow 0.1 mU/l and low-risk patients TSH should be 0.1-0.5 mU/l.

As she is asymptomatic and her TSH is adequately suppressed this lady should be maintained on her current dose pending specialist review.

British Thyroid Association, guidelines for the management of thyroid cancer (2014) and The American Thyroid Associations Guidelines (2009).

**>>DESCRIPTION**: 42-year-old female, post-thyroid cancer resection, on 100mcg levothyroxine daily, TSH < 0.1 mU/l, asymptomatic.

**>>OPTIONS**: a) Change to liothyronine equivalent dose b) Continue at 100mcg per day c) Reduce the levothyroxine as she is over treated d) Repeat thyroid function in 6 weeks e) Stop levothyroxine

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Continue at 100mcg per day

**>>REASONING**: TSH is suppressed in thyroid cancer patients. TSH < 0.1 mU/l is appropriate for high/intermediate risk patients. As the patient is asymptomatic and TSH is adequately suppressed, current dose should be maintained. Stopping or reducing levothyroxine, or changing to liothyronine would be inappropriate.

## Question #:313

**CLINICAL SCENERIO**: A 35-year-old woman with type 1 diabetes, non-compliant with insulin, presents with shortness of breath. Initial and follow-up VBG results are provided, along with current treatment of IV fluids and fixed-rate insulin infusion.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate management choice at this point?

**OPTIONS**: a. Continue current management and add IV dextrose b. No changes to current management c. Stop FRII and continue d. sodium chloride with added potassium e. Stop current management and switch to IV dextrose f. Switch to VRII, continue current IV fluids and add IV dextrose

**CORRECT-CHOICE LINE**: a

**REASONING**: Continue FRII and add IV dextrose is correct. The patient has not yet had a resolution of ketonaemia or acidosis. The underlying pathophysiology of DKA is insulin deficiency. Without insulin, the body cannot use glucose for energy and therefore fat sources are broken down to use as fuel, prompting the generation of ketones, which in turn cause acidosis. To reverse this process and resolve ketonemia and acidosis, we need to continue insulin. Insulin therapy causes a lowering of blood glucose levels and therefore we should administer IV dextrose alongside insulin in order to prevent hypoglycaemia while allowing continued insulin administration.

No changes to current management is incorrect. Insulin drives glucose into cells and lowers blood glucose levels. We need to continue insulin until the ketonemia has resolved. However, this will cause hypoglycaemia unless we add IV dextrose.

Stop FRII and continue 0.9% sodium chloride with added potassium is incorrect. While this will result in the resolution of dehydration and avoid precipitating hypoglycaemia (as insulin has been stopped), it will not correct the underlying pathophysiological process (insulin deficiency) and the DKA will worsen.

Stop current management and switch to IV dextrose is not the right answer. Stopping insulin will resulting in worsening DKA, compounded by exogenous glucose administration.

Switch to VRII, continue current IV fluids and add IV dextrose is incorrect. It is standard practice now to administer a fixed rate insulin infusion in DKA rather than a variable rate as an FRII has been proven to be superior as it has been shown to have the following benefits:

*  Faster resolution of DKA
*  Avoids titration against a poor surrogate marker of blood glucose
*  Confirms resolution of DKA if the FRII is stopped when blood ketone levels are < 0.6mmol/L.

**>>DESCRIPTION**: A 35-year-old woman with type 1 diabetes, poorly compliant with insulin, presents with shortness of breath and is diagnosed with DKA. Given initial and follow-up VBG results and current treatment with IV fluids and a fixed-rate insulin infusion, what is the best next step?

**>>OPTIONS**: a. Continue current management and add IV dextrose b. No changes to current management c. Stop FRII and continue sodium chloride with added potassium d. Stop current management and switch to IV dextrose e. Switch to VRII, continue current IV fluids and add IV dextrose

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Continue current management and add IV dextrose

**>>REASONING**: The correct action is to continue the fixed-rate insulin infusion and add IV dextrose because the patient still has acidosis and ketonemia. Stopping insulin would worsen the DKA. Adding dextrose prevents hypoglycemia from the insulin.

## Question #:225

**CLINICAL SCENERIO**: A 32-year-old patient with Addison’s disease presents for review. He’s on hydrocortisone (20mg in divided doses), fludrocortisone (50 micrograms), and levothyroxine (100 micrograms for hypothyroidism). He reports intermittent fatigue. His thyroid function is well-controlled. Which investigation assesses the accuracy of hydrocortisone replacement?

**QUESTION LINE**: What investigation could be offered to assess if the dose of hydrocortisone represents accurate glucocorticoid replacement?

**OPTIONS**: a) Dexamethasone suppression test b) Cortisol day curve c) Synacthen test d) Morning serum cortisol e) Urinary electrolytes

**CORRECT-CHOICE LINE**: b

**REASONING**: A cortisol curve can be used to assess how appropriate dosing of glucocorticoid steroids in Addison’s disease patients is

A cortisol day curve allows serial measurements to be taken to monitor how well cortisol is being replaced in patient with Addison’s disease as well other causes of adrenal insufficiency such as panhypopituitarism. Morning serum cortisol is an appropriate investigation as first line testing for Addisons’s disease, with Synacthen testing to confirm that diagnosis. Dexamethasone suppression testing can confirm Cushing’s syndrome. Urinary electrolytes would be appropriate in the diagnosis of SIADH.

**>>DESCRIPTION**: A 32-year-old with Addison’s disease, treated with hydrocortisone, fludrocortisone, and levothyroxine, reports fatigue. Which investigation best assesses hydrocortisone replacement adequacy?

**>>OPTIONS**: a) Cortisol day curve b) Dexamethasone suppression test c) Morning serum cortisol d) Synacthen test e) Urinary electrolytes

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cortisol day curve

**>>REASONING**: A cortisol day curve assesses glucocorticoid replacement in Addison’s disease by monitoring cortisol levels over time. Morning serum cortisol is a first-line test for diagnosis; Synacthen tests confirm the diagnosis; Dexamethasone suppression tests evaluate for Cushing’s; urinary electrolytes are irrelevant.

## Question #:63

**CLINICAL SCENERIO**: A 22-year-old woman, 16 weeks pregnant, presents with persistent nausea and vomiting. She has no significant past medical history and takes no regular medicines. Observations are within normal limits. Blood results show normal Hb, platelets, WBC, Na+, K+, urea, creatinine, and CRP levels.

**QUESTION LINE**: What treatment is indicated?

**OPTIONS**: - a) Cyclizine - b) Domperidone - c) Metoclopramide - d) Ondansetron - e) Pyridoxine

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Antihistamines are first-line in the management of nausea & vomiting in pregnancy/hyperemesis gravidarum

Cyclizine is correct. The royal college of Obstetrics and Gynaecology (RCOG) guidelines recommend antihistamines as the first-line agents for nausea and vomiting in pregnancy.

Domperidone is incorrect. This drug is a second-line agent. The risk of extrapyramidal side effects is less compared to metoclopramide.

Metoclopramide is incorrect. This drug is a second-line agent. Metoclopramide can cause extrapyramidal side effects, particularly in young females (<25 years old).

Ondansetron is incorrect. This drug is a second-line agent. The use of ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate.

Pyridoxine is incorrect. Pyridoxine can be used in conjunction with doxylamine, however, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines.

**>>DESCRIPTION**: A 22-year-old woman, 16 weeks pregnant, presents with persistent nausea and vomiting. Her blood results are normal.

**>>OPTIONS**: a) Cyclizine b) Domperidone c) Metoclopramide d) Ondansetron e) Pyridoxine

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cyclizine

**>>REASONING**: Cyclizine (antihistamine) is the first-line treatment for nausea and vomiting in pregnancy according to RCOG guidelines. Domperidone, metoclopramide and ondansetron are second-line agents. Pyridoxine monotherapy is not recommended.

## Question #:312

**CLINICAL SCENERIO**: A 31-year-old, 10 weeks pregnant woman is admitted with nausea and vomiting, unable to tolerate oral medications or fluids for over 24 hours. Intravenous fluids are commenced due to suspected hyperemesis gravidarum.

**QUESTION LINE**: What treatment is recommended first-line?

**OPTIONS**: a. Cyclizine b. Haloperidol c. Metoclopramide d. Ondansetron e. Pyridoxine

**CORRECT-CHOICE LINE**: cyclizine

**REASONING**: Antihistamines are first-line for hyperemesis gravidarum. Cyclizine is an antihistamine, recommended first-line. Haloperidol is an antipsychotic, not first-line. Metoclopramide carries risks and is not first-line. Ondansetron is used if first-line treatments fail, with potential risks. Pyridoxine is not yet recommended as first-line.

**>>DESCRIPTION**: A 31-year-old, 10 weeks pregnant woman presents with hyperemesis gravidarum, requiring intravenous fluids.

**>>OPTIONS**: a. Cyclizine b. Haloperidol c. Metoclopramide d. Ondansetron e. Pyridoxine

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Cyclizine

**>>REASONING**: Cyclizine (an antihistamine) is the recommended first-line treatment for hyperemesis gravidarum. Other options like Haloperidol, Metoclopramide, Ondansetron and Pyridoxine are not first-line due to various factors such as side effects or lack of strong guideline support.

## Question #:188

**CLINICAL SCENERIO**: A 60-year-old woman with mild lethargy had a suppressed TSH (0.25 microU/L) and normal free T4 (14.1 pmol/L). She has a history of hysterectomy without oophorectomy, no significant family history, and is a retired school teacher. Examination showed no goitre, tremor, or eye symptoms. Further tests, including repeat thyroid function tests (TSH 0.21 microU/L and T4 13.8 pmol/L), were performed. The patient’s blood pressure and ECG were within normal limits. Laboratory values were provided, including HbA1c, cholesterol, and electrolytes. What is the most appropriate management?

**QUESTION LINE**: What is the most appropriate management of the deranged thyroid function tests?

**OPTIONS**: - a) DEXA scan - b) Thyroid ultrasound - c) Start treatment with simvastatin - d) Radioiodine therapy - e) Treat with propylthiouracil

**CORRECT-CHOICE LINE**: a

**REASONING**: The patient has subclinical hyperthyroidism with suppressed TSH and normal thyroid hormones. The risk is increased risk of atrial fibrillation and hip fractures. The patient has a low level of cardiac risk factors with a low-risk lipid profile. Therefore, a DEXA scan is appropriate next line management to quantify her osteoporosis risk and inform the decision as to whether or not to treat the sub-clinical hyperthyroidism. Thyroid ultrasound, simvastatin, radioiodine therapy, and propylthiouracil are less appropriate at this stage.

**>>DESCRIPTION**: A 60-year-old woman with mild lethargy presents with suppressed TSH and normal free T4 levels. She has a history of hysterectomy and normal examination findings. Subsequent tests and lab values were provided. What is the most appropriate management?

**>>OPTIONS**: a) DEXA scan b) Radioiodine therapy c) Start treatment with simvastatin d) Thyroid ultrasound e) Treat with propylthiouracil

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: DEXA scan

**>>REASONING**: The patient has subclinical hyperthyroidism. A DEXA scan is the appropriate next step to assess osteoporosis risk. Other options are not indicated at this stage.

## Question #:125

**CLINICAL SCENERIO**: A 44-year-old woman with no medical problems is admitted for a swollen breast. Diagnosed with a breast abscess, she’s treated with antibiotics. Elevated corrected calcium (2.79 mmol/L) and parathyroid hormone (9.5 pmol/L) are noted. Further tests show normal vitamin D, 24-hour urine calcium, and DEXA scan. Diagnosed with primary hyperparathyroidism.

**QUESTION LINE**: What additional investigation should be used to monitor her?

**OPTIONS**: - a) 24-hour urine calcium annually - b) Breast ultrasound annually - c) Abdominal X-ray annually - d) Abdominal ultrasound every three years - e) DEXA scan every one to two years

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The correct answer is a DEXA scan. This patient has been incidentally found to have primary hyperparathyroidism and has no evidence of indications for parathyroidectomy. Monitoring should include renal function and DEXA scanning to identify any decline in renal function, worsening hypercalcaemia or osteoporosis. Any of these changes would be indications for surgery. Abdominal Xrays and ultrasound scanning may be useful in the acute setting to detect renal stones but are not recommended as monitoring. Urinary calcium useful at diagnosis to exclude hypocalciuric hypercalcaemia.

**>>DESCRIPTION**: A 44-year-old woman is diagnosed with primary hyperparathyroidism after presenting with a breast abscess and elevated calcium/PTH. Vitamin D, urine calcium, and DEXA are normal.

**>>OPTIONS**: a) 24-hour urine calcium annually b) Abdominal ultrasound every three years c) Abdominal X-ray annually d) Breast ultrasound annually e) DEXA scan every one to two years

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: DEXA scan every one to two years

**>>REASONING**: DEXA scan is the correct monitoring choice for primary hyperparathyroidism to detect osteoporosis. Renal function should also be monitored. Abdominal imaging and urine calcium are not for routine monitoring.

## Question #:276

**CLINICAL SCENERIO**: A 28-year-old woman presents with abnormal thyroid function tests, neck pain, and a recent coryzal illness six weeks postpartum. She takes lithium and smokes. Examination reveals a tender neck swelling, mild tremor, and tachycardia. Blood tests show an elevated CRP and suppressed TSH. A radioactive iodine uptake scan shows globally reduced uptake.

**QUESTION LINE**: What is the likely diagnosis?

**OPTIONS**: - a) De Quervain’s thyroiditis - b) Factitious hyperthyroidism - c) Grave’s disease - d) Lithium associated thyroiditis - e) Post-partum thyroiditis

**CORRECT-CHOICE LINE**: Answer is A

**REASONING**: De Quervain’s thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

De Quervain’s thyroiditis is the correct answer. This woman presents with painful swelling in her neck (a likely goitre in the context of hyperthyroidism) post-coryzal illness with raised inflammatory markers (CRP), in association with globally reduced uptake of iodine-131. These findings are consistent with viral sub-acute thyroiditis.

Factitious hyperthyroidism is incorrect. Although she has a history of depression (and this is a risk factor for thyroid hormone misuse), this would not explain the raised CRP or neck pain.

Grave’s disease is incorrect. This is associated with increased uptake of iodine-131 on a radioactive iodine scan.

Lithium associated thyroiditis is incorrect. This is classically ‘painless’ thyroiditis.

Post-partum thyroiditis is incorrect. While she is post-partum and this is a plausible differential, this condition is not associated with neck pain or raised inflammatory markers, making this diagnosis unlikely.

**>>DESCRIPTION**: A 28-year-old postpartum woman with a history of bipolar disorder on lithium presents with neck pain, tenderness, tachycardia, and a suppressed TSH, elevated CRP, and reduced iodine uptake.

**>>OPTIONS**: a) De Quervain’s thyroiditis b) Factitious hyperthyroidism c) Grave’s disease d) Lithium associated thyroiditis e) Post-partum thyroiditis

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: De Quervain’s thyroiditis

**>>REASONING**: The diagnosis is De Quervain’s thyroiditis due to the presentation of neck pain, a tender goitre, elevated inflammatory markers (CRP), and reduced iodine uptake. Factitious hyperthyroidism, Grave’s disease, lithium-associated thyroiditis and post-partum thyroiditis are less likely due to the absence of the characteristic signs and symptoms associated with them, such as absent neck pain, increased iodine uptake, or elevated inflammatory markers.

## Question #:263

**CLINICAL SCENERIO**: A 77-year-old female with COPD and heart failure, admitted with a chest infection, presents with hyponatremia (Na+ 120 mmol/l) and is water restricted. Subsequent bloodwork on day 4 shows worsening hyponatremia (Na+ 118 mmol/l).

**QUESTION LINE**: How would you manage this patient?

**OPTIONS**: a. Furosemide b. Levothyroxine c. Demeclocycline d. Hypertonic saline (3%) e. Hypotonic saline (0.45%)

**CORRECT-CHOICE LINE**: c.

**REASONING**: The patient’s worsening hyponatremia despite fluid restriction, in the context of a likely diagnosis of SIADH (supported by the investigations), indicates the need for demeclocycline. Furosemide is indicated for hypervolemic hyponatremia. Levothyroxine is not indicated as thyroid function tests suggest euthyroid sick syndrome and not primary hypothyroidism. Hypertonic saline is reserved for severe hyponatremia. Hypotonic saline would worsen the hyponatremia.

**>>DESCRIPTION**: A 77-year-old female with a chest infection, COPD, and heart failure, presents with worsening hyponatremia despite fluid restriction. How should this patient be managed?

**>>OPTIONS**: a. Demeclocycline b. Furosemide c. Hypertonic saline (3%) d. Hypotonic saline (0.45%) e. Levothyroxine

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Demeclocycline

**>>REASONING**: Demeclocycline is the correct choice because it is indicated for SIADH when fluid restriction is ineffective. Furosemide is for hypervolemic hyponatremia, and levothyroxine is not indicated given the thyroid function test results (suggesting euthyroid sick syndrome not primary hypothyroidism). Hypertonic and hypotonic saline are not appropriate for this patient’s condition.

## Question #:226

**CLINICAL SCENERIO**: A 72-year-old female with a history of type 2 diabetes presents with a 5-day decline following a UTI treated with oral antibiotics. She is insulin-dependent and disoriented (GCS 14/15). Examination reveals dehydration, suprapubic tenderness, and a blood sugar of 31 mmol/L. Venous blood gas shows pH 7.22, lactate 2 mmol/l, and ketones 5 mmol/l.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Hyperglycaemic hyperketotic state b) Hyperglycaemia secondary to poor medical compliance during recent acute illness c) Urosepsis secondary to inadequately treated UTI d) Diabetic ketoacidosis e) Dehydration secondary to poor oral intake

**CORRECT-CHOICE LINE**: d

**REASONING**: The patient is acidotic with ketones >3 mmol/l demonstrated, on a background of known insulin dependence. Although she is known to be a type 2 diabetic, it should be remembered that both types of diabetics can present as DKA, particularly advanced T2 DM who produce little to no endogenous insulin and are hence unable to shut down ketogenesis. Treatment should be as per DKA protocols, with intravenous fluids, fixed rate insulin infusion @ 0.1 unit/kg/hour, thromboprophylaxis, broad spectrum antibiotics and appropriate K+ replacement with insulin1.

**>>DESCRIPTION**: A 72-year-old insulin-dependent type 2 diabetic presents with 5-day decline after UTI treated with antibiotics, is disoriented, dehydrated, with suprapubic tenderness, blood sugar 31 mmol/L, pH 7.22, ketones 5 mmol/L.

**>>OPTIONS**: a) Diabetic ketoacidosis b) Dehydration secondary to poor oral intake c) Hyperglycaemia secondary to poor medical compliance during recent acute illness d) Hyperglycaemic hyperketotic state e) Urosepsis secondary to inadequately treated UTI

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Diabetic ketoacidosis

**>>REASONING**: The most likely diagnosis is diabetic ketoacidosis (DKA) due to acidosis and ketones in an insulin-dependent diabetic. Other options are less likely because of the presence of ketones and acidosis. The patient’s history of UTI and dehydration could be contributing factors, but the primary issue is DKA.

## Question #:169

**CLINICAL SCENERIO**: A 16-year-old male attends the clinic with steatorrhoea, poor growth, ataxia, and reduced visual acuity. He has a family history of abetalipoproteinemia.

**QUESTION LINE**: What management will you advise?

**OPTIONS**: 1. Reassurance 2. Simvastatin 3. Dietary restriction of fats 4. Ezetimibe 5. Increase fat intake

**CORRECT-CHOICE LINE**: c

**REASONING**: Treatment of abetalipoproteinemia involves dietary restriction of fats, and highdose vitamin E therapy

Abetalipoproteinemia is a rare genetic condition caused by a mutation in the microsomal triglyceride transfer protein resulting in deficiencies in apolipoproteins. Apolipoproteins are essential in the synthesis and exportation of chylomicrons and VLDL. The end results is malabsorption of dietary fats, cholesterol, and fat soluble vitamin (e.g. vitamins K, A, D and E).

Clinical features include steatorrhoea, poor growth, neurological dysfunction, and impaired vision.

Treatment is with dietary restriction of fats, and high-dose vitamin E therapy.

**>>DESCRIPTION**: A 16-year-old male with steatorrhoea, poor growth, ataxia, reduced visual acuity, and a family history of abetalipoproteinemia.

**>>OPTIONS**: 1. Dietary restriction of fats 2. Ezetimibe 3. Increase fat intake 4. Reassurance 5. Simvastatin

**>>CORRECT-CHOICE LINE**: 1

**>>CORRECT-CHOICE\_TEXT**: Dietary restriction of fats

**>>REASONING**: The correct management is dietary restriction of fats and high-dose vitamin E therapy. This addresses the malabsorption of fats and fat-soluble vitamins, which is the core issue in abetalipoproteinemia. Other options are incorrect because reassurance is insufficient, Simvastatin and Ezetimibe are not indicated, and increasing fat intake would worsen the condition.

## Question #:88

**CLINICAL SCENERIO**: A 35-year-old female presented with severe right flank pain radiating to her groin. She was being investigated for joint pains, dry eyes, and dry mouth. On examination, BP was 132/68 mmHg. Abdominal examination revealed right flank tenderness. Blood tests showed Na+ 136 mmol/L, K+ 2.8 mmol/L, Urea 3.6 mmol/L, Creatinine 70 µmol/L, Bicarbonate 9 mmol/L, Chloride 116 mmol/L, Calcium 2.3 mmol/L, Phosphate 1.1 mmol/L.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Bartter syndrome b) Fanconi syndrome c) Gitelman syndrome d) Lactic acidosis e) Distal renal tubular acidosis

**CORRECT-CHOICE LINE**: Distal renal tubular acidosis can cause calcium phosphate renal stones and is linked to Sjogrens syndrome

**REASONING**: This patient presents with renal colic. Blood tests show severe metabolic acidosis and hypokalaemia which fits with a distal renal tubular acidosis (RTA). The history of dry eyes and mouth is suggestive of Sjogrens syndrome, which is frequently associated with distal RTA. Patients with distal RTA are at risk of developing calcium phosphate stones.

Fanconi syndrome causes a proximal RTA which tends to cause milder acidosis and hypokalaemia, along with other electrolyte disturbances such as hypophosphataemia.

Gitelman and Bartter syndromes cause a hypokalaemic alkalosis and are therefore incorrect.

Lactic acidosis would cause a raised anion gap acidosis, whereas the anion gap in this case is normal (12).

**>>DESCRIPTION**: A 35-year-old female with right flank pain, dry eyes, and dry mouth presents with BP 132/68 mmHg and right flank tenderness. Labs: Na+ 136, K+ 2.8, Bicarbonate 9, Cl- 116.

**>>OPTIONS**: a) Bartter syndrome b) Distal renal tubular acidosis c) Fanconi syndrome d) Gitelman syndrome e) Lactic acidosis

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Distal renal tubular acidosis

**>>REASONING**: Severe metabolic acidosis and hypokalemia, along with dry eyes and mouth, suggest distal renal tubular acidosis (RTA) linked to Sjogren’s. Fanconi syndrome causes proximal RTA with milder symptoms. Gitelman and Bartter cause hypokalemic alkalosis. Lactic acidosis causes a raised anion gap.

## Question #:91

**CLINICAL SCENERIO**: A 59-year-old man with type 1 diabetes mellitus for 40 years presents with peripheral diabetic sensory neuropathy, impotence, bilateral diabetic retinopathy (treated with laser therapy), and unpredictable vomiting of undigested food. Dietary modification is ineffective. Condition responded to erythromycin during a previous hospital admission. Barium swallow shows significantly prolonged gastric emptying.

**QUESTION LINE**: Which of the following is the most appropriate long-term intervention?

**OPTIONS**: a) Ondansetron b) Gastric pacemaker c) Chlorpheniramine d) Domperidone e) Erythromycin

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Diabetic gastroparesis is best managed long term with domperidone

Pro-kinetic anti-emetics are the intervention of choice for the treatment of diabetic gastroparesis, the diagnosis here. Vomiting is often unpredictable and doesn’t respond to dietary modification, making the condition extremely debilitating for sufferers. Domperidone, a dopamine D2 receptor antagonist is the main prospect for long-term intervention, as it doesn’t have the neurological liabilities which are associated with metoclopramide use, it may be associated with galactorrhoea due to a rise in prolactin levels in some patients who take it.

Ondansetron, a centrally acting anti-emetic is largely ineffective for gastroparesis, erythromycin is used for acute episodes of diabetic gastroparesis, and gastric pacemaker therapy is designated a research therapy of last resort by NICE. Chlorpheniramine is an anti-histamine that has some anti-emetic properties, although it is predominantly a centrally acting agent.

**>>DESCRIPTION**: A 59-year-old man with 40-year history of type 1 DM presents with diabetic neuropathy, impotence, retinopathy, and unpredictable vomiting of undigested food. Barium swallow shows delayed gastric emptying.

**>>OPTIONS**: a) Chlorpheniramine b) Domperidone c) Erythromycin d) Gastric pacemaker e) Ondansetron

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Domperidone

**>>REASONING**: Domperidone, a pro-kinetic antiemetic and dopamine D2 receptor antagonist, is the best long-term management for diabetic gastroparesis. Ondansetron and chlorpheniramine are largely ineffective. Erythromycin is for acute episodes, and gastric pacemaker therapy is a last resort.

## Question #:255

**CLINICAL SCENERIO**: A 29-year-old man with Addison’s disease planning to run a marathon asks his endocrinologist about managing his hydrocortisone and fludrocortisone.

**QUESTION LINE**: What is the most appropriate advice to give?

**OPTIONS**: a) Take doses as normal for the marathon b) Double the hydrocortisone dose but not the fludrocortisone dose for the marathon c) Double the fludrocortisone dose but not hydrocortisone dose for the marathon d) Double both the fludrocortisone dose and the hydrocortisone dose for the marathon e) Omit both medications for the marathon

**CORRECT-CHOICE LINE**: B.

**REASONING**: A patient with Addison’s disease undertaking strenuous activity should double both glucocorticoid and mineralocorticoid doses, with advice to increase fluid intake. Omitting medication could cause an Addisonian crisis.

**>>DESCRIPTION**: A 29-year-old man with Addison’s disease preparing for a marathon needs advice on managing hydrocortisone and fludrocortisone.

**>>OPTIONS**: a) Take doses as normal for the marathon b) Double the hydrocortisone dose but not the fludrocortisone dose for the marathon c) Double the fludrocortisone dose but not hydrocortisone dose for the marathon d) Double both the fludrocortisone dose and the hydrocortisone dose for the marathon e) Omit both medications for the marathon

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Double the hydrocortisone dose but not the fludrocortisone dose for the marathon

**>>REASONING**: The patient needs to double the hydrocortisone dose, but not the fludrocortisone dose for the marathon to counter the increased stress on the body. Option a) is incorrect as normal doses would be inadequate. Options c), d) and e) are incorrect as they suggest incorrect medication management or omission, which can lead to an Addisonian crisis.

## Question #:41

**CLINICAL SCENERIO**: A 62-year-old man with type 2 diabetes mellitus, osteoarthritis, steatohepatitis, and stable angina presents with vomiting, abdominal pain, and drowsiness after eating undercooked chicken four days prior. He takes metformin, dapagliflozin, aspirin, omeprazole, paracetamol, and bisoprolol. Investigations reveal a serum glucose of 12.1 mmol/L, a pH of 6.9, and +++ ketones in urine, leading to a diagnosis of euglycaemic diabetic ketoacidosis.

**QUESTION LINE**: What factor has most likely contributed to the development of diabetic ketoacidosis?

**OPTIONS**: a) Drug history of dapagliflozin b) Drug history of metformin c) Drug history of bisoprolol d) Medical history of steatohepatitis e) Medical history of angina

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: SGLT2 inhibitors can cause normoglycaemic ketosis in type 2 diabetes

The most likely explanation is the drug history of dapagliflozin. Dapagliflozin is a SGLT2 inhibitor which reduces glucose reabsorption in the urine and increases urinary glucose excretion. This patient has developed one of the side effects of dapagliflozin; DKA. DKA secondary to SGLT2 dapagliflozin is more likely to be euglycaemic than DKA in general. Metformin is associated with metabolic acidosis, especially in the context of reduced renal function or alcohol, and bisoprolol can cause hypoglycaemia. Steatohepatitis can eventually result in hepatic failure causing impaired gluconeogenesis, but this is more likely to result in hypoglycaemia rather than DKA. Angina is unlikely to be relevant.

**>>DESCRIPTION**: A 62-year-old man with type 2 diabetes, osteoarthritis, steatohepatitis, and angina presents with vomiting, abdominal pain, and drowsiness after suspected undercooked chicken. He takes metformin, dapagliflozin, aspirin, omeprazole, paracetamol, and bisoprolol. He is diagnosed with euglycaemic DKA (glucose 12.1 mmol/L, pH 6.9, +++ ketones).

**>>OPTIONS**: a) Drug history of bisoprolol b) Drug history of dapagliflozin c) Drug history of metformin d) Medical history of angina e) Medical history of steatohepatitis

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Drug history of dapagliflozin

**>>REASONING**: Dapagliflozin, an SGLT2 inhibitor, likely caused euglycemic DKA by reducing glucose reabsorption. Metformin can cause metabolic acidosis, bisoprolol can cause hypoglycemia, and steatohepatitis is more likely to cause hypoglycemia than DKA. Angina is unlikely to be relevant.

## Question #:293

**CLINICAL SCENERIO**: A 68-year-old gentleman with a history of depression and type 2 diabetes presents with severe abdominal pain. His medications include levemir, sitagliptin, and gliclazide. Examination reveals dry mucus membranes, generalized abdominal pain, a blood pressure of 101/76 mmHg, a pulse rate of 113 beats per minute, and a temperature of 37.8ºC. Investigations include a clear chest X-ray, an abdominal ultrasound showing a normal gallbladder and liver changes, and blood work indicating elevated amylase (1378 IU/L) and glucose (38 mmol/l), with ketones + in the urinalysis.

**QUESTION LINE**: What is the likely underlying diagnosis?

**OPTIONS**: - a) Diabetic ketoacidosis - b) Perforated small bowel - c) Drug induced pancreatitis - d) Acute hepatitis - e) Alcohol induced pancreatitis

**CORRECT-CHOICE LINE**: Sitagliptiin induced pancreatitis.

**REASONING**: Patients with perforated small bowel or DKA can present with a raised amylase. The level suggests the cause is most likely pancreatitis. A perforated small bowel would more likely show free gas on a Chest X ray, diminished bowel sounds and guarding. Diabetic ketoacidosis would show more ketones on urinalysis and is rare, although still possible, in type 2 diabetics.

The gentleman has no evidence of gallstones or biliary obstruction. This combined with a normal liver function tests makes gallstone unlikely. The patient denies alcohol intake and non-alcoholic fatty liver can produce the appearances on ultrasound scan.

Both dipeptidyl peptidase 4 inhibitors (sitagliptin) and glucagon like peptide-1 agonists have been linked to pancreatitis. They are still both under investigation but is to be discontinued in the event of an episode of pancreatitis.

**>>DESCRIPTION**: A 68-year-old man with type 2 diabetes presents with severe abdominal pain. Examination reveals dry mucus membranes, generalized abdominal pain, elevated heart rate, and elevated temperature. Investigations show elevated amylase, elevated glucose, and ketones in the urine.

**>>OPTIONS**: a) Acute hepatitis b) Alcohol induced pancreatitis c) Diabetic ketoacidosis d) Drug induced pancreatitis e) Perforated small bowel

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Drug induced pancreatitis

**>>REASONING**: The patient’s presentation of severe abdominal pain, elevated amylase and the use of sitagliptin strongly suggest drug-induced pancreatitis. Perforated bowel and DKA are less likely due to the presentation and specific lab findings. There is no evidence to support acute hepatitis or alcohol induced pancreatitis.

## Question #:237

**CLINICAL SCENERIO**: A 58-year-old man presents with a two-month history of weight loss, increasing confusion, and proximal weakness. He was recently treated for an islet cell carcinoma and has no other significant medical history. Examination reveals cognitive impairment, proximal muscle weakness, and elevated blood pressure. Laboratory findings show hypokalemia, hyperglycemia, and hypernatremia.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Paraneoplastic encephalitis - b) Cerebral metastases - c) Post chemotherapy Cushing’s syndrome - d) Post chemotherapy hypothyroidism - e) Ectopic ACTH secretion

**CORRECT-CHOICE LINE**: E

**REASONING**: The confusion, hypertension, proximal myopathy, hypernatraemia, hypokalaemia, and hyperglycaemia suggest Cushing’s syndrome. Given the patient’s recent islet cell carcinoma treatment, ectopic ACTH secretion by the tumor is the most likely cause. Post-chemotherapy Cushing’s is less likely, and the observed electrolyte imbalances are more pronounced in ectopic ACTH secretion.

**>>DESCRIPTION**: A 58-year-old man with a history of islet cell carcinoma presents with weight loss, confusion, proximal weakness, hypertension, hypokalemia, hyperglycemia, and hypernatremia. What is the most likely diagnosis?

**>>OPTIONS**: a) Cerebral metastases b) Ectopic ACTH secretion c) Paraneoplastic encephalitis d) Post chemotherapy Cushing’s syndrome e) Post chemotherapy hypothyroidism

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Ectopic ACTH secretion

**>>REASONING**: The combination of confusion, hypertension, proximal myopathy, and electrolyte imbalances (hypernatremia, hypokalemia, hyperglycemia) strongly suggests Cushing’s syndrome, most likely caused by ectopic ACTH secretion from the islet cell carcinoma. Other options are less likely given the clinical and laboratory findings.

## Question #:146

**CLINICAL SCENERIO**: A 24-year-old nurse collapses after a night shift and is found to have low blood glucose. A CT scan reveals a hypervascular pancreatic lesion.

**QUESTION LINE**: What further finding would be most supportive of the likely diagnosis?

**OPTIONS**: a) Elevated C-peptide following administration of IV insulin b) Low serum C-peptide level c) Normal serum proinsulin level d) Positive anti-GAD antibodies e) Sulphonylurea detectable in urine

**CORRECT-CHOICE LINE**: a

**REASONING**: An elevated C-peptide level following administration of IV insulin can confirm insulinoma The correct answer is elevated C-peptide following administration of IV insulin . IV insulin administration should generally suppress endogenous insulin secretion and therefore C-peptide levels should fall. An inappropriate elevation in C-peptide levels should raise the suspicion of an endogenous source of insulin secretion. The CT findings are suggestive of an insulinoma. Low serum C-peptide level is incorrect. This would be expected in cases of factitious hypoglycaemia related to insulin administration. While this patient’s employment would give her access to insulin and may raise suspicion of this, the CT findings suggest an insulinoma is more likely. Normal serum proinsulin level is incorrect. This would also be expected in a patient with factitious hypoglycaemia, whether related to insulin or sulphonylurea administration. In an insulinoma, we would expect elevated proinsulin levels, especially after a prolonged fast. Positive anti-GAD antibodies is incorrect. These would be expected in a patient with type 1 diabetes mellitus. In the absence of treatment, this would present with hyper- rather than hypoglycaemia. Sulphonylurea detectable in urine is incorrect. In this context, this would be suggestive of factitious hypoglycaemia related to sulphonylurea use. Again, this patient would have access through her employment but the CT findings suggest insulinoma is more likely.

**>>DESCRIPTION**: A 24-year-old nurse with low blood glucose collapses. CT scan shows a hypervascular pancreatic lesion.

**>>OPTIONS**: a) Elevated C-peptide following administration of IV insulin b) Low serum C-peptide level c) Normal serum proinsulin level d) Positive anti-GAD antibodies e) Sulphonylurea detectable in urine

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Elevated C-peptide following administration of IV insulin

**>>REASONING**: Elevated C-peptide after IV insulin supports insulinoma; IV insulin should suppress endogenous insulin, but an increase suggests inappropriate insulin secretion. Other options are incorrect as they are more consistent with factitious hypoglycemia (insulin or sulfonylurea use) or type 1 diabetes, not insulinoma.

## Question #:124

**CLINICAL SCENERIO**: A 62-year-old woman with type 2 diabetes and mild cardiac failure presents for review. She is managed with ramipril, bisoprolol, and metformin 1g BD. Examination reveals BP 122/82 mmHg, pulse 80 bpm, bilateral basal crackles, and pitting oedema. BMI is 33 kg/m². HbA1c is 73 mmol/mol and creatinine is 82 µmol/l.

**QUESTION LINE**: Which of the following is the most appropriate next step for managing glucose control?

**OPTIONS**: a) Empagliflozin b) Glipizide c) Liraglutide d) Pioglitazone e) Saxagliptin

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This patient has moderate obesity, cardiac failure, and a poorly controlled HbA1c. Empagliflozin, an SGLT-2 inhibitor, is most appropriate due to its glucose-lowering and diuretic effects, which lower blood pressure and reduce fluid retention. Empagliflozin also reduces cardiovascular mortality. Glipizide may cause weight gain, saxagliptin can cause heart failure, and pioglitazone causes fluid retention. Liraglutide is indicated at a BMI of 35 and above.

**>>DESCRIPTION**: 62F, T2DM, cardiac failure, on ramipril, bisoprolol, metformin 1g BD. BP 122/82, HR 80, crackles, edema, BMI 33. HbA1c 73 mmol/mol, creatinine 82 µmol/l.

**>>OPTIONS**: a) Empagliflozin b) Glipizide c) Liraglutide d) Pioglitazone e) Saxagliptin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Empagliflozin

**>>REASONING**: Empagliflozin is the best choice due to its glucose-lowering, diuretic, and cardioprotective effects. Glipizide causes weight gain, saxagliptin causes heart failure, and pioglitazone causes fluid retention. Liraglutide’s indication is BMI > 35.

## Question #:70

**CLINICAL SCENERIO**: You are seeing a 50-year-old lady with type 2 diabetes mellitus in the outpatient clinic. She has a past medical history of gastritis, moderate left ventricular dysfunction and chronic obstructive pulmonary disease. She is currently on metformin and gliclazide. Since last review she has gained 5kg in weight and her HbA1c has deteriorated to 70 mmol/mol from 62 mmol/mol. Body mass index today in clinic is 33 kg/m². Recent blood tests are as follows: Na + 141 mmol/l, K + 3.9 mmol/l, Urea 6 mmol/l, Creatinine 140 µmol/l. She was unable to previously tolerate liraglutide due to nausea and vomiting.

**QUESTION LINE**: What would be the best alteration to her therapy?

**OPTIONS**: a) Empagliflozin (SGLT-2 inhibitor) b) Add insulin c) Add pioglitazone d) Increase dose of metformin e) Increase dose of gliclazide

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: SGLT inhibitors have the advantage of improving glycaemic control/HbA1c and having beneficial effects on weight. This is because their mode of action is independent of insulin release. They act upon the SGLT-2 receptors in the kidney and lead to increased loss of glucose in the urine.

**>>DESCRIPTION**: 50F, T2DM, gastritis, moderate LV dysfunction, COPD, on metformin & gliclazide. Gained 5kg, HbA1c 70 mmol/mol (from 62). BMI 33 kg/m². Liraglutide intolerant due to nausea/vomiting. Na 141, K 3.9, Urea 6, Creatinine 140.

**>>OPTIONS**: a) Add insulin b) Add pioglitazone c) Empagliflozin (SGLT-2 inhibitor) d) Increase dose of gliclazide e) Increase dose of metformin

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Empagliflozin (SGLT-2 inhibitor)

**>>REASONING**: Empagliflozin (SGLT-2 inhibitor) improves glycemic control and promotes weight loss due to its insulin-independent mechanism, increasing glucose excretion via the kidneys. Other options do not address weight gain effectively or have tolerability issues.

## Question #:249

**CLINICAL SCENERIO**: A 28-year-old man with familial hypercholesterolaemia on 80mg atorvastatin has elevated cholesterol levels.

**QUESTION LINE**: What treatment would you begin?

**OPTIONS**: a) Fenofibrate b) Nicotinic acid c) Ezetimibe d) Evolocumab e) Cholestyramine

**CORRECT-CHOICE LINE**: C

**REASONING**: First line management for familial hypercholesterolaemia is high dose statins. Ezetimibe is used as a second line agent

High dose statin therapy is first line for familial hypercholesterolaemia.

If serum total or low-density lipoprotein (LDL) cholesterol concentration is not appropriately controlled on statin therapy, then the second line agent ezetimibe can be co-administered with the statin. Ezetimibe acts by decreasing cholesterol absorption in the small intestine

**>>DESCRIPTION**: A 28-year-old man with familial hypercholesterolaemia on atorvastatin has elevated cholesterol levels.

**>>OPTIONS**: a) Cholestyramine b) Ezetimibe c) Evolocumab d) Fenofibrate e) Nicotinic acid

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Ezetimibe

**>>REASONING**: Ezetimibe is the second-line agent to be used when statin therapy does not appropriately control cholesterol levels. Statins are first-line, and other options are not indicated in this scenario.

## Question #:68

**CLINICAL SCENERIO**: A 43-year-old woman is referred to the endocrinology outpatient service after a finding of incidental hypercalcaemia on a routine blood test. She has no past medical history. The examination is unremarkable. Blood tests show elevated parathyroid hormone (7.9 pmol/L) and calcium (2.74 mmol/L), with normal vitamin D (75 nmol/L), urea (4.2 mmol/L), and creatinine (66 µmol/L).

**QUESTION LINE**: What is the most likely explanation for the findings from the list below?

**OPTIONS**: - a) Familial benign hypocalciuric hypercalcaemia - b) Multiple myeloma - c) Sarcoidosis - d) Secondary hyperparathyroidism - e) Tertiary hyperparathyroidism

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

Familial benign hypocalciuric hypercalcaemia is the correct answer. The patient has hypercalcaemia with a raised PTH level. Plausible differentials include primary hyperparathyroidism, tertiary hyperparathyroidism and familial benign hypocalciuric hypercalcaemia. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Primary hyperparathyroidism is not listed as an option. Therefore, familial benign hypocalciuric hypercalcaemia is the correct answer.

Multiple myeloma is incorrect. Classically, malignancies cause a suppressed PTH level if they are the cause of hypercalcemia. This is true of multiple myeloma.

Sarcoidosis is incorrect. Hypercalcemia in sarcoidosis is due to the uncontrolled production of 1,25-dihydroxy vitamin D3 by macrophages. This causes hypercalcemia and a suppressed PTH. unlike in some forms of hyperparathyroidism, the negative feedback mechanism whereby high calcium causes a low PTH is maintained.

Secondary hyperparathyroidism is incorrect. This form of hyperparathyroidism occurs as an appropriate reaction to low vitamin D or calcium. In this case, the vitamin is normal and the calcium is high.

Tertiary hyperparathyroidism is incorrect. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Therefore this is not likely.

**>>DESCRIPTION**: A 43-year-old woman has incidental hypercalcemia (2.74 mmol/L) and elevated PTH (7.9 pmol/L). Vitamin D, urea, and creatinine are normal. What is the most likely diagnosis?

**>>OPTIONS**: a) Familial benign hypocalciuric hypercalcaemia b) Multiple myeloma c) Sarcoidosis d) Secondary hyperparathyroidism e) Tertiary hyperparathyroidism

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Familial benign hypocalciuric hypercalcaemia

**>>REASONING**: Familial benign hypocalciuric hypercalcemia (FBHH) is the most likely diagnosis given hypercalcemia with normal to elevated PTH. Multiple myeloma and sarcoidosis typically suppress PTH. Secondary hyperparathyroidism is a response to low vitamin D or calcium, which are normal here. Tertiary hyperparathyroidism occurs in longstanding secondary hyperparathyroidism, which is not indicated.

## Question #:302

**CLINICAL SCENERIO**: A 60-year-old man attends a medical health check-up with a past medical history of childhood asthma and osteoarthritis. His observations included a blood pressure of 129/80 mmHg, pulse of 82 bpm, and oxygen sats of 97%. Blood tests and a 24-hour urinary calcium test were performed with the following results (table provided).

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a. Primary hyperparathyroidism b. Secondary hyperparathyroidism c. Vitamin D toxicity d. Multiple endocrine neoplasia type I e. Familial benign hypocalciuric hypercalcaemia

**CORRECT-CHOICE LINE**: e

**REASONING**: Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

The most likely diagnosis in this scenario is familial benign hypocalciuric hypercalcaemia. Most cases are asymptomatic and blood test reveals hypercalcaemia with a reduced calcium urinary excretion rate (of under 0.02 mmol/L). There may also be normal to high parathyroid hormone, despite the elevated serum calcium levels.

**>>DESCRIPTION**: A 60-year-old man with a history of asthma and osteoarthritis presents for a check-up. Investigations reveal specific blood test results, including elevated serum corrected calcium and a low 24-hour urinary calcium excretion.

**>>OPTIONS**: a. Familial benign hypocalciuric hypercalcaemia b. Multiple endocrine neoplasia type I c. Primary hyperparathyroidism d. Secondary hyperparathyroidism e. Vitamin D toxicity

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Familial benign hypocalciuric hypercalcaemia

**>>REASONING**: The most likely diagnosis is familial benign hypocalciuric hypercalcemia, characterized by hypercalcemia and a reduced urinary calcium excretion rate, potentially with normal or elevated PTH. Other options are less likely given the clinical and biochemical findings.

## Question #:58

**CLINICAL SCENERIO**: A 46-year-old man presents with asymptomatic hypercalcaemia discovered on routine blood tests. His past medical history is significant for hypertension, for which he is taking amlodipine, ramipril and chlorthalidone. Investigation results show: Na+ 141 mmol/L, K+ 4.4 mmol/L, Calcium 2.85 mmol/L, Urea 6.6 mmol/L, Creatinine 98 µmol/L, Parathyroid hormone 5.5 pmol/L.

**QUESTION LINE**: What is the most likely explanation for this patient’s hypercalcaemia?

**OPTIONS**: a) Chlorthalidone use b) Familial hypocalciuric hypercalcaemia c) Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion d) Primary hyperparathyroidism e) Secondary hyperparathyroidism

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Low urinary calcium in the presence of hypercalcaemia is suggestive of either familial hypocalciuric hypercalcaemia or thiazide diuretic use

The correct answer is familial hypocalciuric hypercalcaemia . This patient has hypercalcemia with low urinary calcium, which narrows the diagnosis down to familial hypocalciuric hypercalcemia (FHH) or thiazide diuretic usage (other causes of hypercalcemia tend to cause elevated urinary calcium). The diagnosis of FHH is clinched by the inappropriately normal parathyroid hormone. This is because, in FHH, loss of function mutations in the CASR calcium-sensing receptor decrease sensitivity to calcium, meaning PTH remains unsuppressed at higher-than-normal serum calcium levels. Hypocalciuria results from a loss of CASR-mediated negative feedback of tubular reabsorption/ excretion of calcium.

Chlorthalidone use is incorrect, as this would give hypocalciuric hypercalcaemia with a suppressed PTH.

Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion is incorrect. This would classically lead to hypercalciuria. In addition, serum PTH may be suppressed rather than inappropriately normal in this scenario, as the PTHrp peptide is detected by a different assay from the conventional PTH assay.

Primary hyperparathyroidism is incorrect. Although this could explain the hypercalcaemia and inappropriately normal PTH, this would lead to hypercalciuria rather than hypocalciuria.

Secondary hyperparathyroidism is incorrect. Here, PTH is released in response to hypocalcaemia, often in the context of renal disease. The elevated serum calcium and normal renal function seen in this patient do not support this diagnosis.

**>>DESCRIPTION**: A 46-year-old man with hypertension on amlodipine, ramipril, and chlorthalidone presents with asymptomatic hypercalcaemia (Ca 2.85 mmol/L, PTH 5.5 pmol/L) found on routine blood tests.

**>>OPTIONS**: a) Chlorthalidone use b) Familial hypocalciuric hypercalcaemia c) Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion d) Primary hyperparathyroidism e) Secondary hyperparathyroidism

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Familial hypocalciuric hypercalcaemia

**>>REASONING**: The patient has hypercalcemia with inappropriately normal PTH and likely hypocalciuria. This points to FHH due to CASR mutations causing decreased sensitivity to calcium. Chlorthalidone would suppress PTH. Hypercalcemia of malignancy and primary hyperparathyroidism typically cause hypercalciuria. Secondary hyperparathyroidism occurs in the context of hypocalcemia.

## Question #:267

**CLINICAL SCENERIO**: A 45-year-old lady presents with abdominal pain and malaise. Blood tests show elevated calcium, normal renal function, and a normal chest X-ray. She has a history of slightly raised calcium 8 years prior. What is the most likely diagnosis?

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a. Secondary hyperparathyroidism b. Malignancy with bony metastasis c. Primary hyperparathyroidism d. Familial hypocalciuric hypercalcaemia e. Sarcoidosis

**CORRECT-CHOICE LINE**: Answer is D.

**REASONING**: PO4 would normally be low in primary hyperparathyroidism. Her renal function is normal excluding secondary hyperparathyroidism. Sarcoidosis is unlikely with a normal CXR. This leaves malignancy or familial hypocalciuric hypercalcaemia. Although malignancy is possible her raised Ca2+ 8 years makes familial hypocalciuric hypercalcaemia more likely

**>>DESCRIPTION**: A 45-year-old woman presents with abdominal pain, malaise, elevated calcium, normal renal function, and a normal chest X-ray. She had slightly elevated calcium 8 years ago. What is the most likely diagnosis?

**>>OPTIONS**: a. Familial hypocalciuric hypercalcaemia b. Malignancy with bony metastasis c. Primary hyperparathyroidism d. Sarcoidosis e. Secondary hyperparathyroidism

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Familial hypocalciuric hypercalcaemia

**>>REASONING**: The most likely diagnosis is familial hypocalciuric hypercalcemia (FHH) given the patient’s history of elevated calcium and normal renal function. Primary hyperparathyroidism is less likely due to the normal renal function and the lack of a significantly elevated phosphate level. Secondary hyperparathyroidism is excluded due to the normal renal function. Sarcoidosis is less likely given the normal chest X-ray. While malignancy is possible, the patient’s history favors FHH.

## Question #:71

**CLINICAL SCENERIO**: A 36-year-old woman, diagnosed with gestational diabetes during pregnancy, now has type 2 diabetes mellitus post-partum. She is slightly overweight (BMI 27.1 kg/m²) and there’s concern for maturity onset diabetes of the young (MODY) or type 1 diabetes.

**QUESTION LINE**: Which one of the following is most suggestive of MODY?

**OPTIONS**: - a) Ketosis during periods of hyperglycaemi - b) Family history of early onset diabetes mellitus - c) A history of polycystic ovarian syndrome - d) Lack of response to sulphonylureas - e) A history of autoimmune disease

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: MODY is inherited in an autosomal dominant fashion so a family history is often present

The correct answer is a family history of early onset diabetes mellitus . Maturity onset diabetes of the young (MODY) is a group of monogenic disorders characterized by autosomal dominant inheritance, early onset of hyperglycemia (usually before 25 years of age), and a primary defect in pancreatic beta-cell function. A family history of early onset diabetes mellitus is most suggestive of MODY because it indicates that there may be a genetic predisposition to the condition, which is consistent with the autosomal dominant inheritance pattern seen in MODY.

Ketosis during periods of hyperglycaemia is more suggestive of type 1 diabetes rather than MODY. Type 1 diabetes results from autoimmune destruction of pancreatic beta cells, leading to an absolute insulin deficiency. In the absence of sufficient insulin, the body relies on breaking down fats for energy, resulting in ketone production and ketosis. In contrast, patients with MODY typically have a defect in pancreatic beta-cell function but still produce some insulin, so they are less likely to develop ketosis.

A history of polycystic ovarian syndrome (PCOS) is not particularly suggestive of MODY. PCOS is associated with insulin resistance and an increased risk for developing type 2 diabetes mellitus. While some forms of MODY can be associated with insulin resistance, it would not be considered the most characteristic feature when trying to differentiate between types of diabetes.

Lack of response to sulphonylureas can be seen in some subtypes of MODY (e.g., MODY 1 and MODY 3). However, it does not necessarily rule out other types or subtypes since there are several different genetic defects that can cause MODY. Furthermore, lack of response to sulphonylureas could also occur in type 2 diabetics who have progressed to needing insulin therapy.

A history of autoimmune disease is more suggestive of type 1 diabetes, as it results from an autoimmune process that destroys pancreatic beta cells. While there is no strong association between MODY and autoimmune diseases, a history of autoimmune disease could increase the likelihood of developing other types of diabetes, such as type 1 or Latent Autoimmune Diabetes in Adults (LADA).

**>>DESCRIPTION**: A 36-year-old woman with post-partum type 2 diabetes (BMI 27.1 kg/m²) raises suspicion for MODY or type 1 diabetes.

**>>OPTIONS**: a) A history of autoimmune disease b) A history of polycystic ovarian syndrome c) Family history of early onset diabetes mellitus d) Ketosis during periods of hyperglycaemi e) Lack of response to sulphonylureas

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Family history of early onset diabetes mellitus

**>>REASONING**: Family history of early onset diabetes is most suggestive of MODY due to its autosomal dominant inheritance. Ketosis suggests type 1 diabetes. PCOS, lack of response to sulfonylureas, and autoimmune disease are less specific or more indicative of other diabetes types.

## Question #:254

**CLINICAL SCENERIO**: A 29-year-old female presents to the Emergency Department following collapse. She reports generalized weakness, palpitations, and dizziness. Her past medical history includes hypothyroidism, rheumatoid arthritis (treated with infliximab, complicated by TB 3 months prior), and current medications: methotrexate, folic acid, levothyroxine, artificial tears, rifampicin, and isoniazid. Examination reveals broad-complex tachycardia and stable blood pressure. Venous blood gas analysis and urine dipstick results are provided.

**QUESTION LINE**: What is the most likely cause of the metabolic abnormalities described?

**OPTIONS**: a. Addison’s disease b. Fanconi syndrome c. Rheumatoid arthritis d. Sjogren’s syndrome e. Liver cirrhosis

**CORRECT-CHOICE LINE**: B

**REASONING**: The patient exhibits normal anion gap hyperchloremic metabolic acidosis with hypokalemia, suggesting renal tubular acidosis. Fanconi syndrome, associated with proximal tubular dysfunction, is the most likely cause given the patient’s recent rifampicin use. Rheumatoid arthritis and Sjogren’s syndrome are associated with distal renal tubular acidosis, which is inconsistent with the lack of renal calcification. Addison’s disease typically presents with hyperkalemia. Liver cirrhosis is unlikely without evidence of Wilson’s disease.

**>>DESCRIPTION**: A 29-year-old female with a history of hypothyroidism, rheumatoid arthritis (treated with infliximab, complicated by TB), presents with collapse, generalized weakness, palpitations, and dizziness. The patient is taking methotrexate, folic acid, levothyroxine, artificial tears, rifampicin, and isoniazid. Blood gas and urine analysis results are provided.

**>>OPTIONS**: a. Addison’s disease b. Fanconi syndrome c. Liver cirrhosis d. Rheumatoid arthritis e. Sjogren’s syndrome

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Fanconi syndrome

**>>REASONING**: Fanconi syndrome is the most likely cause, characterized by proximal renal tubular dysfunction and potentially caused by rifampicin, presenting with normal anion gap hyperchloremic metabolic acidosis with hypokalemia. Rheumatoid arthritis and Sjogren’s syndrome are less likely. Addison’s disease is inconsistent, and liver cirrhosis is unlikely without Wilson’s disease.

## Question #:309

**CLINICAL SCENERIO**: A 28-year-old lady diagnosed with gestational diabetes in her first pregnancy achieved blood glucose control with diet, exercise, and metformin. She delivered a healthy child at 39 weeks. Her fasting blood glucose at day 1 post-partum was 5.2mmol/l.

**QUESTION LINE**: Which of the following statements is correct with respect to follow-up monitoring for diabetes?

**OPTIONS**: a) OGTT 6-13 weeks postpartum b) Fasting blood glucose test 6-13 weeks postpartum c) No routine follow up unless further pregnancy d) HbA1c 6-13 weeks postpartum e) Annual fasting blood glucose checks only

**CORRECT-CHOICE LINE**: B

**REASONING**: Women with gestational diabetes whose glucose returns to normal after birth need a postnatal glucose check 6-13 weeks postpartum to stratify their risk of developing diabetes in the future. NICE recommends that this is a fasting blood glucose. Further follow up will depend on the result of this postnatal check. Even if postnatal glucose is less than 6mmol/l, annual fasting glucose checks are still recommended thereafter.

**>>DESCRIPTION**: A 28-year-old woman with gestational diabetes controlled by diet, exercise, and metformin delivered a healthy child. Her post-partum fasting blood glucose was 5.2mmol/l. What’s the correct follow-up monitoring for diabetes?

**>>OPTIONS**: a) Annual fasting blood glucose checks only b) Fasting blood glucose test 6-13 weeks postpartum c) HbA1c 6-13 weeks postpartum d) No routine follow up unless further pregnancy e) OGTT 6-13 weeks postpartum

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Fasting blood glucose test 6-13 weeks postpartum

**>>REASONING**: The correct follow-up is a fasting blood glucose test 6-13 weeks postpartum. This helps assess the risk of future diabetes. Annual fasting glucose checks are recommended even if the initial check is normal.

## Question #:16

**CLINICAL SCENERIO**: A 50-year-old female is admitted with epigastric pain radiating to her back. She has no past medical history and takes no regular medications. Her alcohol history includes 4 glasses of wine per week.

Blood results are as follows:

| Hb | 115 g/l | Na + | 138 mmol/l |
| --- | --- | --- | --- |
| Platelets | 288 \* 10 9 /l | K + | 3.8 mmol/l |
| WBC | 10.8 \* 10 9 /l | Urea | 6.8 mmol/l |
| Neuts | 8.8 \* 10 9 /l | Creatinine | 72 µmol/l |
| Lymphs | 1.8 \* 10 9 /l | CRP | 84 mg/l |
| Triglyceride | 12.3 mmol/l (normal range < 2.3 mmol/L) | Bilirubin | 16 µmol/l |
| ALT | 24 u/l | Amylase | 924 U/L |
| ALP | 76 u/l | Albumin | 38 g/l |

An abdominal ultrasound is performed:

Abdominal ultrasound report

Np gall stones identified. No intra or extra hepatic duct dilation evident

**QUESTION LINE**: What treatment will reduce the risk of pancreatitis reoccurrence?

**OPTIONS**: - a) Ursodeoxycholic acid - b) Fenofibrate - c) Simvastatin - d) Nicotinic acid - e) Laparoscopic cholecystectomy

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Fibrates are the most effective drug for treating hypertriglyceridaemia

The patient has clinical features of pancreatitis. The raised amylase confirms the diagnosis. The two most common causes of pancreatitis in the UK are excess alcohol and gallstones.

The normal liver function tests and ultrasound rule out gallstone disease as the cause. Therefore a laparoscopic cholecystectomy or treatment with ursodeoxycholic acid is not indicated.

The patient drinks 4 glasses of wine per week. A 250ml glass of wine at 12% alcohol contains 250 \* 0.12 = 30 ml of pure alcohol. 10 ml of pure alcohol = 1 unit. Therefore each glass of wine contains 3 unit. Her alcohol intake per week is therefore 12 units which is within the recommended intake. It is unlikely that alcohol is the cause.

A patient has a significantly high triglyceride level. While gallstones and alcohol account for roughly 80% of cases of pancreatitis, hypertriglyceridemia is the next most common aetiology, accounting for roughly 1-4% of cases. The exact mechanism is unclear but it is thought to involve increased concentrations of chylomicrons in the blood. These low-density particles are very large and may obstruct capillaries leading to local ischemia and acidaemia. This local damage can expose triglycerides to pancreatic lipases.

Fibrates, statins, and nicotinic acid will all lower triglyceride levels, however, fibrates are the most effective drug in doing so.

Discuss (2)Improve

**>>DESCRIPTION**: A 50-year-old female presents with epigastric pain radiating to the back. No significant past medical history or medications. Alcohol consumption: 4 glasses of wine per week. Labs show elevated triglycerides (12.3 mmol/L) and amylase (924 U/L). Abdominal ultrasound reveals no gallstones or duct dilation.

**>>OPTIONS**: a) Fenofibrate b) Laparoscopic cholecystectomy c) Nicotinic acid d) Simvastatin e) Ursodeoxycholic acid

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Fenofibrate

**>>REASONING**: The patient has pancreatitis likely due to hypertriglyceridemia, given the elevated triglyceride level and the absence of gallstones on ultrasound. While statins and nicotinic acid can lower triglycerides, fibrates are the most effective. Ursodeoxycholic acid and laparoscopic cholecystectomy are not indicated as there is no evidence of gallstone-related disease. The given alcohol intake is not high enough to be the cause of pancreatitis.

## Question #:183

**CLINICAL SCENERIO**: A 41-year-old woman is admitted to hospital with acute epigastric abdominal pain radiating to her back. She has nausea but has not vomited. Acute pancreatitis is suspected and intravenous fluids are commenced. Observations: BP 129/72 mmHg, pulse 88 bpm, oxygen sats 97%. Blood tests reveal Hb 13.9 g/l, Platelets 194 \* 10 9 /l, WBC 8.6 \* 10 9 /l, Na + 139 mmol/l, K + 4.2 mmol/l, Urea 4.1 mmol/l, Creatinine 92 µmol/l, Bilirubin 10 µmol/l, ALP 39 u/l, ALT 34 u/l, γGT 44 u/l, Albumin 48 g/l, Triglycerides 12.1 mmol/l, HDL cholesterol 1.1 mmol/l, LDL cholesterol 3.5 mmol/l.

**QUESTION LINE**: What is the most appropriate treatment for this patients condition?

**OPTIONS**: b) Fenofibrate c) Lovastati d) Ezetimibe e) Alirocumab

**CORRECT-CHOICE LINE**: b

**REASONING**: Fibrates are the most effective drug for treating hypertriglyceridaemia

The diagnosis is hypertriglyceridaemia, which has caused this patients acute pancreatitis. Fibrates are the treatment for hypertriglyceridaemia at high enough levels to cause acute pancreatitis.

**>>DESCRIPTION**: A 41-year-old woman presents with acute epigastric pain radiating to the back, nausea (no vomiting), and suspected acute pancreatitis. Blood tests reveal elevated triglycerides.

**>>OPTIONS**: a) Alirocumab b) Ezetimibe c) Fenofibrate d) Lovastati

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Fenofibrate

**>>REASONING**: Fenofibrate (a fibrate) is the most appropriate treatment for hypertriglyceridemia, the likely cause of the patient’s acute pancreatitis.

## Question #:121

**CLINICAL SCENERIO**: A 40-year-old woman found a neck lump that was palpable in the left lobe of her thyroid gland. She was clinically euthyroid.

TSH

3.6 (NR 0.4-5.0)

free T4

15.1 (NR 10-25)

corrected calcium

2.41 (NR 2.2-2.6)

USS: 1.7 x 1.6cm solid lesion in left lobe of thyroid with microcalcification.

**QUESTION LINE**: What is the next step in management?

**OPTIONS**: a) Fine needles aspiration cytology b) Radio-iodine uptake scan c) Left lobectomy d) Calcitonin level e) Thyroglobulin level

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This nodule Is suspicious for malignancy because: microcalcifications, solid lesion, and no evidence of a hot lesion (as euthyroid). The next step is therefore FNA of the nodule.

Work up for thyroid nodules is as follows:

1. Check TSH
2. TSH suppressed → Thyroid uptake scan → Cold/iso nodule → FNA cytology
3. TSH suppressed → Thyroid uptake scan →Hot nodule → No FNA required
4. TSH normal/elevated → Thyroid USS → Suspicious features → FNA cytology
5. FNA cytology is then graded using the Royal College of Pathologist classification on a spectrum from benign → malignant.

**>>DESCRIPTION**: A 40-year-old woman presents with a palpable neck lump in the left thyroid lobe and is clinically euthyroid. TSH: 3.6, free T4: 15.1, corrected calcium: 2.41. Ultrasound shows a 1.7 x 1.6cm solid lesion with microcalcifications.

**>>OPTIONS**: a) Calcitonin level b) Fine needles aspiration cytology c) Left lobectomy d) Radio-iodine uptake scan e) Thyroglobulin level

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Fine needles aspiration cytology

**>>REASONING**: The thyroid nodule is suspicious for malignancy due to microcalcifications and a solid lesion. The next step is Fine Needle Aspiration (FNA). Radioiodine uptake scan is not indicated as the patient is euthyroid. Lobectomy is premature before cytology. Calcitonin and thyroglobulin levels are not the initial step.

## Question #:42

**CLINICAL SCENERIO**: A 23-year-old man with type 1 diabetes presents to the emergency department with vomiting and abdominal pain. He has been suffering from diarrhea for two days and then became severely dehydrated and started to vomit. He has not had his insulin for the last 24 hours. He normally has a basal bolus regime with Levemir as a long-acting insulin, and Humalog as short-acting. He has no other medical problems or regular prescription. On admission he is found to have acidosis, elevated serum ketones and elevated blood glucose, and was diagnosed with diabetic ketoacidosis. He is given rapid fluid infusion.

**QUESTION LINE**: What insulin should he be prescribed?

**OPTIONS**: - a) Fixed rate IV insulin only - b) Fixed rate IV insulin as well as long-acting insulin - c) Fixed rate IV insulin, long-acting insulin and short-acting insulin - d) Sliding scale IV insulin only - e) Sliding scale IV insulin as well as long-acting insulin

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: In the acute management of DKA, insulin should be fixed rate whilst continuing re

This is a typical presentation for diabetic ketoacidosis in a patient with type 1 diabetes and requires urgent management which should be supported by local guidelines. It is important that IV insulin is started, but any long-acting insulin, Levemir for this patient, should also be continued. This will aid transition back to normal when the DKA resolves and the patient starts to eat and drink again. Giving him short acting insulin is not appropriate during IV insulin therapy which is also a short-acting insulin. Sliding scale IV insulin is no longer recommended in favour of fixed rate insulin.

**>>DESCRIPTION**: A 23-year-old man with type 1 diabetes presents with vomiting, abdominal pain, and dehydration after missing insulin for 24 hours. He has acidosis, elevated serum ketones and glucose, and is diagnosed with DKA. He is given rapid fluid infusion.

**>>OPTIONS**: a) Fixed rate IV insulin as well as long-acting insulin b) Fixed rate IV insulin only c) Fixed rate IV insulin, long-acting insulin and short-acting insulin d) Sliding scale IV insulin as well as long-acting insulin e) Sliding scale IV insulin only

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Fixed rate IV insulin as well as long-acting insulin

**>>REASONING**: In DKA, fixed-rate IV insulin should be started, and long-acting insulin (Levemir) should be continued to aid transition back to normal. Short-acting insulin is not appropriate during IV insulin therapy. Sliding scale IV insulin is not recommended.

## Question #:93

**CLINICAL SCENERIO**: A 31-year-old female with type 1 diabetes, background diabetic retinopathy, and stage 3 chronic kidney disease presents with abdominal pain, diarrhoea, vomiting, and reduced appetite for several days. She hasn’t taken her usual insulin (32 units Lantus at night and variable Novomix with meals) for 2 days due to poor appetite. Examination reveals she is unwell, with a respiratory rate of 26/min, pulse of 120/min, BP of 103/45 mmHg, and temperature of 36.7 oC. Abdomen is tender. Capillary blood glucose is 26 and ketones are 4.9. Arterial blood gas shows pH 7.32, pO2 11.6 kPa, pCO2 3.32 kPa, Bicarbonate 14 mmol/l, Base Excess -6.5 mmol/l, and Lactate 2.1 mmol/l. She is being fluid resuscitated. What insulin therapy is most appropriate?

**QUESTION LINE**: What form of insulin therapy would you advise?

**OPTIONS**: a) Dose of novomix now and increase usual lantus by 8 units b) Variable rate i.v insulin, continue all s/c insulins c) Recommence normal s/c insulin regimen d) Fixed rate i.v insulin, continue lantus e) Fixed rate i.v insulin, stop all s/c insulin

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: This patient has a compensated metabolic acidosis and despite her reasonably preserved pH, a bicarbonate of 14 suggests significant metabolic acidosis. In the presence of high blood ketones and hyperglycaemia, the diagnosis is diabetic ketoacidosis (DKA). Fluid therapy and potassium supplementation is a key part of DKA management but will not be dealt with in this question. Current recommendations advise fixed rate insulin with the addition of 10 % dextrose when capillary blood glucose drops below 14. The aim is to correct the underlying ketoacidosis brought about by a hypoinsulinaemic state. Patients should be continued on their long acting insulin but short/medium term insulins should be held. This ensures a smooth transfer from fixed rate to usual insulin regimen when the patient has clinically and biochemically improved.

**>>DESCRIPTION**: 31F, T1DM, diabetic retinopathy, CKD stage 3, presents with abdominal pain, diarrhea, vomiting, and poor appetite for 5 days. She missed her usual insulin. Exam: RR 26, HR 120, BP 103/45, T 36.7. Glucose 26, ketones 4.9. ABG: pH 7.32, HCO3 14. Fluid resuscitation ongoing. What insulin therapy is appropriate?

**>>OPTIONS**: a) Dose of novomix now and increase usual lantus by 8 units b) Fixed rate i.v insulin, continue lantus c) Fixed rate i.v insulin, stop all s/c insulin d) Recommence normal s/c insulin regimen e) Variable rate i.v insulin, continue all s/c insulins

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Fixed rate i.v insulin, continue lantus

**>>REASONING**: The patient is in DKA. Current guidelines recommend fixed-rate IV insulin while continuing long-acting insulin (Lantus). Short/medium-acting insulins should be held to allow for smooth transition to the usual insulin regimen after clinical and biochemical improvement.

## Question #:286

**CLINICAL SCENERIO**: A 25-year-old woman presents to the emergency department via ambulance after being found unwell. She has a 3-day history of vomiting and diarrhea. She is drowsy, dehydrated, and has abdominal tenderness. Investigations reveal a fingerpick blood glucose of 38.2 mmol/L, blood ketones of 8.7 mmol/L, and arterial blood gas indicating metabolic acidosis. The patient has a history of not taking her insulin.

**QUESTION LINE**: What is the appropriate strategy for intravenous insulin treatment in this patient?

**OPTIONS**: - a) Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once acidosis resolved - b) Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normall - c) Fixed rate insulin infusion following initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally - d) Variable rate insulin infusion with initial insulin bolus, converting to subcutaneous insulin once acidosis resolved - e) Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once ketonaemia resolved

**CORRECT-CHOICE LINE**: B

**REASONING**: The patient is in diabetic ketoacidosis due to vomiting, dehydration, and missed insulin doses. The Joint British Diabetes Society recommends a fixed-rate insulin infusion without an initial bolus, switching to subcutaneous insulin when the patient can eat and drink normally. An initial bolus of insulin is not advised. Fixed-rate infusions are preferred over sliding scales to ensure adequate insulin to resolve ketosis.

**>>DESCRIPTION**: A 25-year-old woman with a 3-day history of vomiting, diarrhea, and missed insulin doses presents with diabetic ketoacidosis. What is the appropriate intravenous insulin treatment strategy?

**>>OPTIONS**: a) Fixed rate insulin infusion following initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally b) Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normall c) Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once acidosis resolved d) Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once ketonaemia resolved e) Variable rate insulin infusion with initial insulin bolus, converting to subcutaneous insulin once acidosis resolved

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normall

**>>REASONING**: The correct approach is a fixed-rate insulin infusion without an initial bolus, transitioning to subcutaneous insulin once the patient can eat and drink. This approach is supported by guidelines and avoids initial bolus to prevent rapid glucose correction and ensure ketone clearance.

## Question #:76

**CLINICAL SCENERIO**: An 82-year-old man admitted for recurrent falls is found to have symptomatic postural hypotension (50mmHg drop in systolic BP on standing). His history includes hypertension, osteoporosis, radial fracture, and COPD. He was taking ramipril, amlodipine, inhalers, and calcium supplements. Despite stopping antihypertensives, physiotherapy is hindered by his symptoms. At rest, his systolic BP is 150mmHg.

**QUESTION LINE**: What is the most appropriate plan?

**OPTIONS**: - a) Fludrocortisone - b) Hydrocortisone - c) Discharge bedbound and continue with physiotherapy at home - d) Discharge bedbound with restarting antihypertensives - e) Inpatient rehabilitation

**CORRECT-CHOICE LINE**: The correct answer is fludrocortisone.

**REASONING**: Fludrocortisone is a corticosteroid with minimal glucocorticoid activity but very high mineralocorticoid activity which can aid in symptomatic postural hypotension and can significantly reduce symptoms. It may take several days to have an effect but can restore mobility in some patients. Continuing with rehabilitation without addressing his treatable cause would not be optimal, whilst re-introducing his antihypertensives would reduce the risk of stroke and cardiovascular disease but would also reduce his independence significantly.

**>>DESCRIPTION**: 82-year-old with recurrent falls and postural hypotension (50mmHg drop in systolic BP). History: hypertension, osteoporosis, COPD. Medications stopped on admission. Systolic BP at rest: 150mmHg.

**>>OPTIONS**: a) Discharge bedbound and continue with physiotherapy at home b) Discharge bedbound with restarting antihypertensives c) Fludrocortisone d) Hydrocortisone e) Inpatient rehabilitation

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Fludrocortisone

**>>REASONING**: Fludrocortisone’s mineralocorticoid activity helps postural hypotension, restoring mobility. Rehabilitation alone is suboptimal. Restarting antihypertensives reduces stroke risk but impairs independence.

## Question #:176

**CLINICAL SCENERIO**: A 28-year-old male with a history of epilepsy on carbamazepine presents with irritability, nausea, and confusion for 2 weeks. Examination reveals eczema and normal lab values except for hyponatremia.

**QUESTION LINE**: Which of the following would be the most appropriate initial management option?

**OPTIONS**: 1) Fluid restriction to 500 - 1000 ml daily 2) Demeclocycline 600 - 1200 mg daily 3) IV hypertonic saline 4) Intranasal desmopressin twice daily 5) Hydrochlorothiazide 12.5 mg daily

**CORRECT-CHOICE LINE**: a.

**REASONING**: The diagnosis is SIADH secondary to carbamazepine. Initial management involves fluid restriction. Demeclocycline is used if fluid restriction is ineffective. Hypertonic saline is for severe, acute cases. Desmopressin is for diabetes insipidus. Thiazide diuretics are rarely used.

**>>DESCRIPTION**: A 28-year-old male on carbamazepine presents with irritability, nausea, confusion, and hyponatremia. What is the most appropriate initial management?

**>>OPTIONS**: 1) Fluid restriction to 500 - 1000 ml daily 2) Demeclocycline 600 - 1200 mg daily 3) Hydrochlorothiazide 12.5 mg daily 4) Intranasal desmopressin twice daily 5) IV hypertonic saline

**>>CORRECT-CHOICE LINE**: 1

**>>CORRECT-CHOICE\_TEXT**: Fluid restriction to 500 - 1000 ml daily

**>>REASONING**: The correct answer is fluid restriction, the initial treatment for SIADH. Other options are incorrect because demeclocycline is used if fluid restriction fails, hypertonic saline is for severe cases, desmopressin is for diabetes insipidus, and thiazide diuretics are rarely used.

## Question #:230

**CLINICAL SCENERIO**: A 24-year-old woman with Addison’s disease presents to the ED with vomiting and lightheadedness, unable to take her hydrocortisone and fludrocortisone for five days. She has been given IV hydrocortisone, fluids, and antiemetics. Her systolic blood pressure improved, and glucose is normal.

**QUESTION LINE**: What further prescription would be appropriate at this stage?

**OPTIONS**: a) Start oral hydrocortisone b) Immediate fludrocortisone c) Start insulin on a sliding scale d) Further IV fluids e) Start IV antibiotics

**CORRECT-CHOICE LINE**: d

**REASONING**: In an adrenal crisis, hydrocortisone is needed at high dose and fludrocortisone can be omitted as hydrocortisone has mineralocorticoid activity

This is a patient with known Addison’s disease who presented unwell having been unable to take regular hydrocortisone doses. The key management is immediate IV hydrocortisone with plenty of IV fluids. When the patient is out of the immediate management stage, feeling better, and able to tolerate oral intake then hydrocortisone can be restarted orally. As this patient has not managed oral intake yet, prescribing oral hydrocortisone may lead to further missed doses. When the patient is having high dose IV hydrocortisone, there is significant mineralocorticoid activity, meaning that fludrocortisone is not needed. She is unlikely to need insulin at all, unless there is evidence of type 1 diabetes mellitus, which can be associated with Addison’s disease.

**>>DESCRIPTION**: A 24-year-old woman with Addison’s disease presents to the ED with vomiting, lightheadedness, and has not taken her hydrocortisone/fludrocortisone for five days. She received IV hydrocortisone, fluids, and antiemetics. Blood pressure improved, glucose is normal.

**>>OPTIONS**: a) Further IV fluids b) Immediate fludrocortisone c) Start IV antibiotics d) Start oral hydrocortisone e) Start insulin on a sliding scale

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Further IV fluids

**>>REASONING**: The correct answer is further IV fluids, as the patient is still unable to take oral intake. Hydrocortisone is provided through IV, and fludrocortisone isn’t immediately required. Insulin is unlikely, and antibiotics are not indicated.

## Question #:187

**CLINICAL SCENERIO**: A 57-year-old female presents with a non-tender neck lump, hypertension, and constipation. She undergoes ultrasound and fine needle aspiration. Following the procedure, she develops a headache and urinary metanephrine collection is positive.

**QUESTION LINE**: Which investigation is likely to produce the underlying diagnosis?

**OPTIONS**: - a) Genetic testing for RET mutation - b) Genetic testing for germline VHL mutation - c) Genetic testing for NF2 mutation on chromosome - d) CT chest/abdomen/pelvis with contrast - e) Urinary calcium collection

**CORRECT-CHOICE LINE**: a

**REASONING**: The clinical presentation suggests phaeochromocytoma and hyperparathyroidism, raising suspicion of multiple endocrine neoplasia type 2a, caused by a RET oncogene mutation. VHL and NF2 mutations are less likely, as they present differently.

**>>DESCRIPTION**: A 57-year-old female presents with a non-tender neck lump, hypertension, and positive urinary metanephrine. What is the likely diagnosis?

**>>OPTIONS**: a) CT chest/abdomen/pelvis with contrast b) Genetic testing for NF2 mutation on chromosome c) Genetic testing for RET mutation d) Genetic testing for germline VHL mutation e) Urinary calcium collection

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Genetic testing for RET mutation

**>>REASONING**: The correct answer is genetic testing for RET mutation because the clinical presentation is most suggestive of Multiple Endocrine Neoplasia type 2a (MEN2a), which is caused by a mutation of the RET oncogene. Other options are less likely based on the presenting symptoms.

## Question #:204

**CLINICAL SCENERIO**: A 57-year-old female presents with a non-tender neck lump, reporting only cosmetic concerns. Her history includes hypertension and constipation, managed with ramipril, irbesartan, amlodipine, and furosemide. Examination reveals a 2cm x 1cm hard, non-tender neck lump that moves with swallowing. Blood tests show elevated adjusted calcium. After fine needle aspiration and persistent hypertension, she develops a headache. Urinary metanephrine collection was positive.

**QUESTION LINE**: Which investigation is likely to produce the underlying diagnosis?

**OPTIONS**: - a) Genetic testing for RET mutation - b) Genetic testing for germline VHL mutation - c) Genetic testing for NF2 mutation on chromosome 22 - d) CT chest/abdomen/pelvis with contrast - e) Urinary calcium collection

**CORRECT-CHOICE LINE**: a

**REASONING**: The clinical presentation of phaeochromocytoma and hyperparathyroidism, along with a thyroid lump, suggests multiple endocrine neoplasia type 2a (MEN 2a), caused by a RET oncogene mutation. VHL and NF2 mutations are less likely given the clinical picture.

**>>DESCRIPTION**: A 57-year-old female presents with a non-tender neck lump, hypertension, and elevated adjusted calcium. After fine needle aspiration and persistent hypertension, she develops a headache. Urinary metanephrine collection was positive.

**>>OPTIONS**: a) CT chest/abdomen/pelvis with contrast b) Genetic testing for germline VHL mutation c) Genetic testing for NF2 mutation on chromosome 22 d) Genetic testing for RET mutation e) Urinary calcium collection

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Genetic testing for RET mutation

**>>REASONING**: The most likely diagnosis is Multiple Endocrine Neoplasia type 2a (MEN 2a), which is associated with a RET oncogene mutation. The clinical presentation of phaeochromocytoma and hyperparathyroidism with a thyroid lump suggests this. Other options are less likely as they relate to different syndromes.

## Question #:317

**CLINICAL SCENERIO**: A 69-year-old lady with a history of headaches, sweating, abdominal pain, and fluctuating blood pressure, now presents with a neck lump and elevated parathyroid hormone levels. Her daughter is concerned about developing similar symptoms.

**QUESTION LINE**: What should you offer the daughter?

**OPTIONS**: 1. Reassurance 2. Genetic testing for patient and daughter 3. Offer annual follow up for surveillance 4. CT abdomen/pelvis with contrast 5. Serum bone and calcium homeostasis profile

**CORRECT-CHOICE LINE**: b.

**REASONING**: The patient describes features consistent with phaeochromocytoma, the appropriate surgical team would be endocrine surgery, who would arrange for a resection. The new blood tests are suggestive of primary hyperparathyroidism, the neck lumps possible medullary thyroid tumour, resulting in a unifying diagnosis of MEN 2a. The daughter can undergo genetic testing for RET-mutation.

**>>DESCRIPTION**: A 69-year-old patient presents with a neck lump, elevated parathyroid hormone, and a history suggestive of phaeochromocytoma. What should be offered to her daughter?

**>>OPTIONS**: 1. Reassurance 2. Genetic testing for patient and daughter 3. Offer annual follow up for surveillance 4. CT abdomen/pelvis with contrast 5. Serum bone and calcium homeostasis profile

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Genetic testing for patient and daughter

**>>REASONING**: Genetic testing for the RET-mutation is indicated for the daughter due to the potential for Multiple Endocrine Neoplasia type 2a (MEN 2a) based on the mother’s symptoms and findings.

## Question #:79

**CLINICAL SCENERIO**: A 16-year-old boy presents with generalised weakness, muscle cramps, polydipsia and cravings for salty foods. On examination: Respiratory rate of 18/min, Blood pressure of 116/78mmHg, Heart rate of 78/min. Blood tests: Na+ 129 mmol/L (135 - 145), K+ 2.8 mmol/L (3.5 - 5.0). Urine dipstick shows no glucose or protein.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Bartter syndrome b) Dent disease c) Fanconi syndrome d) Gitelman syndrome e) Liddle syndrome

**CORRECT-CHOICE LINE**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

**REASONING**: The syndrome presented here is that of Gitelman syndrome . This is a rare genetic disorder caused by mutations in the thiazide-sensitive sodium-chloride symporter in the distal convoluted tubule. Symptomatic patients present with symptoms identical to those taking excessive thiazide diuretics - hypokalaemia, hypomagnesaemia, and hyponatraemia. Patients are normotensive. Importantly, patients also display hypocalciuria, which differentiates the condition from Bartter syndrome. Treatment involves salt replacement.

Bartter syndrome is a similar condition, except that it involves a mutation in the Na/K/2Cl cotransporter in the loop of Henle. As such, the condition mimics loop diuretic abuse. Whilst also presenting with hypokalaemia, hypomagnesaemia, and normotension, the condition crucially presents with hypercalciuria, rather than hypocalciuria as seen in Gitelman syndrome.

Dent disease is a rare X-linked recessive condition that affects the proximal tubules. It is characterised by tubular proteinuria, hypercalciuria, and glycosuria. It is one of the causes of Fanconi syndrome. The scenario above is more closely aligned with Gitelman syndrome.

Fanconi syndrome is a syndrome of proximal tubule dysfunction. It presents with polyuria and polydipsia, like in this scenario, but patients would also have proteinuria and glycosuria, unlike the patient here. Hypokalaemia would also be noted, as well as hypophosphataemia. It may be genetic, such as being caused by Dent disease or acquired.

Liddle syndrome is an autosomal dominant condition - a mutation in the epithelial sodium channels (ENaC) of the kidneys, lungs, and sweat glands. The condition causes hypertension, which does not respond to medications, and hypokalaemia. Typically, children are asymptomatic at presentation. The patient in this scenario is normotensive

**>>DESCRIPTION**: A 16-year-old boy has weakness, muscle cramps, polydipsia and salt cravings. Exam: RR 18/min, BP 116/78mmHg, HR 78/min. Labs: Na+ 129 mmol/L, K+ 2.8 mmol/L. Urine: no glucose or protein.

**>>OPTIONS**: a) Bartter syndrome b) Dent disease c) Fanconi syndrome d) Gitelman syndrome e) Liddle syndrome

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Gitelman syndrome

**>>REASONING**: Gitelman syndrome is most likely due to normotension, hypokalemia, hyponatremia and hypocalciuria. Bartter syndrome presents with hypercalciuria. Dent and Fanconi syndromes typically have proteinuria and glycosuria. Liddle syndrome presents with hypertension.

## Question #:21

**CLINICAL SCENERIO**: A 21-year-old woman presents to the Emergency department following a collapse at the local supermarket. She feels weak and washed out, and hardly ever has any energy. She takes no regular medication. On examination her blood pressure is 100/70 mmHg, pulse is 80 beats per minute and regular. She is slim with a body mass index of 21 kg/m², no abnormal physical signs are noted. Investigations show: Na + 140 mmol/l, K + 3.1 mmol/l, HCO3 - 32 mmol/l, Urea 5.9 mmol/l, Creatinine 85 µmol/l.

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: - a) Conn’s syndrome - b) Cushing’s syndrome - c) Gitelman’s syndrome - d) Liddle’s syndrome - e) Renal tubular acidosis type

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

This patient has hypokalaemic metabolic alkalosis with normal / low blood pressure. This fits best with a diagnosis of Gitelman’s syndrome, Bartter’s syndrome, or diuretic abuse. Bartter’s syndrome often presents in infancy as failure to thrive, and isn’t listed as a distractor. Diuretic abuse presents with a similar clinical picture although it too isn’t listed here as an option. Gitelman’s is caused by a mutation in the gene coding for the thiazide-sensitive sodiumchloride co-transporter, and results in a clinical picture similar to that expected with thiazide diuretic use.

Conn’s syndrome is associated with hypertension, as is Liddle’s syndrome, effectively ruling them out as diagnoses here. Renal tubular acidosis type 1 is associated with hypokalaemia and metabolic acidosis, not with a bicarbonate of 32.

**>>DESCRIPTION**: A 21-year-old woman presents with weakness and collapse. BP 100/70 mmHg, pulse 80 bpm. BMI 21 kg/m², no abnormal signs. Labs: Na+ 140 mmol/l, K+ 3.1 mmol/l, HCO3- 32 mmol/l, Urea 5.9 mmol/l, Creatinine 85 µmol/l.

**>>OPTIONS**: a) Conn’s syndrome b) Cushing’s syndrome c) Gitelman’s syndrome d) Liddle’s syndrome e) Renal tubular acidosis type

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Gitelman’s syndrome

**>>REASONING**: The patient presents with hypokalemic metabolic alkalosis and normal/low blood pressure, suggestive of Gitelman’s syndrome, Bartter’s syndrome, or diuretic abuse. Gitelman’s is the most likely. Conn’s and Liddle’s syndromes are associated with hypertension. Renal tubular acidosis type 1 is associated with metabolic acidosis, not alkalosis.

## Question #:27

**CLINICAL SCENERIO**: A 21-year-old woman presents to the Emergency department following a collapse at the local supermarket. She reports weakness, fatigue, and lack of energy. She takes no regular medication and rarely sees the doctor. Examination reveals a blood pressure of 100/70 mmHg, pulse of 80 bpm, and a BMI of 21 kg/m². No abnormal physical signs are noted. Labs: Na+ 140 mmol/l, K+ 3.1 mmol/l, HCO3- 32 mmol/l, Urea 5.9 mmol/l, Creatinine 85 µmol/l.

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: - a) Conn’s syndrome - b) Cushing’s syndrome - c) Gitelman’s syndrome - d) Liddle’s syndrome - e) Renal tubular acidosis type

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

This patient has hypokalaemic metabolic alkalosis with normal / low blood pressure. This fits best with a diagnosis of Gitelman’s syndrome, Bartter’s syndrome, or diuretic abuse. Bartter’s syndrome often presents in infancy as failure to thrive, and isn’t listed as a distractor. Diuretic abuse presents with a similar clinical picture although it too isn’t listed here as an option. Gitelman’s is caused by a mutation in the gene coding for the thiazide-sensitive sodiumchloride co-transporter, and results in a clinical picture similar to that expected with thiazide diuretic use.

Conn’s syndrome is associated with hypertension, as is Liddle’s syndrome, effectively ruling them out as diagnoses here. Renal tubular acidosis type 1 is associated with hypokalaemia and metabolic acidosis, not with a bicarbonate of 32.

**>>DESCRIPTION**: A 21-year-old woman presents with collapse, weakness, and fatigue. BP is 100/70 mmHg, pulse 80 bpm, BMI 21 kg/m². Labs: Na+ 140, K+ 3.1, HCO3- 32, Urea 5.9, Creatinine 85.

**>>OPTIONS**: a) Conn’s syndrome b) Cushing’s syndrome c) Gitelman’s syndrome d) Liddle’s syndrome e) Renal tubular acidosis type

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Gitelman’s syndrome

**>>REASONING**: The patient presents with hypokalemic metabolic alkalosis and normal/low blood pressure, suggestive of Gitelman’s syndrome. Conn’s and Liddle’s syndromes are associated with hypertension. Renal tubular acidosis type 1 presents with metabolic acidosis, not alkalosis.

## Question #:128

**CLINICAL SCENERIO**: A 27-year-old female presents with fatigue and hypokalemia (2.8 mmol/l, then 2.6 mmol/l). Examination is unremarkable, with BP 108/78 mmHg and BMI 23kg/m². Investigations show Na+ 132 mmol/l, K+ 2.5 mmol/l, Bicarbonate 37 mmol/l, renin 824 pmol/l (NR 100-500), aldosterone 82 pmol/l (NR 55-250). 24-hour urine: Na+ 28 mmol/L, K+ 45 mmol/l, Calcium 0.8 mmol/24hrs.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Addison’s disease b) Gitelman’s syndrome c) Conn’s disease d) Bartter’s syndrome e) Laxative abuse

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

Gitelman’s syndrome is an autosomal recessive disorder resulting in a normotensive hypokalaemic metabolic alkalosis with hypocalciuria and is often accompanied with hypomagnesaemia. The defect is in the thiazide-sensitive sodium chloride symporter within the distal convoluted tubules, in contrast to Bartter’s syndrome which presents in the same way but with hypercalciuria owing to a defect within the ascending loop of Henle. Patients with both conditions are often asymptomatic or may complain of fatigue, cramps and weakness. Conn’s disease is associated with hypertension and in this instance the aldosterone level is normal with an elevated renin, making this diagnosis unlikely. Both Addison’s disease and laxative abuse are associated with a metabolic acidosis; in Addison’s serum potassium also tends to be elevated. The best answer is, therefore, Gitelman’s syndrome.

**>>DESCRIPTION**: 27F with fatigue, hypokalemia, normal BP, elevated renin, normal aldosterone, and hypocalciuria. What is the likely diagnosis?

**>>OPTIONS**: a) Addison’s disease b) Bartter’s syndrome c) Conn’s disease d) Gitelman’s syndrome e) Laxative abuse

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Gitelman’s syndrome

**>>REASONING**: Gitelman’s syndrome is the correct diagnosis due to normotension, hypokalemia, metabolic alkalosis, and hypocalciuria. Bartter’s syndrome presents similarly but with hypercalciuria. Conn’s disease is associated with hypertension. Addison’s and laxative abuse cause metabolic acidosis.

## Question #:136

**CLINICAL SCENERIO**: A 27-year-old female presents with fatigue and persistent hypokalemia. Investigations reveal normal blood pressure, elevated renin and aldosterone, and hypokaluria.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Addison’s disease b) Gitelman’s syndrome c) Conn’s disease d) Bartter’s syndrome e) Laxative abuse

**CORRECT-CHOICE LINE**: b.

**REASONING**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

Gitelman’s syndrome is an autosomal recessive disorder resulting in a normotensive hypokalaemic metabolic alkalosis with hypocalciuria and is often accompanied with hypomagnesaemia. The defect is in the thiazide-sensitive sodium chloride symporter within the distal convoluted tubules, in contrast to Bartter’s syndrome which presents in the same way but with hypercalciuria owing to a defect within the ascending loop of Henle. Patients with both conditions are often asymptomatic or may complain of fatigue, cramps and weakness. Conn’s disease is associated with hypertension and in this instance the aldosterone level is normal with an elevated renin, making this diagnosis unlikely. Both Addison’s disease and laxative abuse are associated with a metabolic acidosis; in Addison’s serum potassium also tends to be elevated. The best answer is, therefore, Gitelman’s syndrome.

**>>DESCRIPTION**: A 27-year-old female presents with fatigue, persistent hypokalemia, normotension, elevated renin and aldosterone, and hypokaluria.

**>>OPTIONS**: a) Addison’s disease b) Bartter’s syndrome c) Conn’s disease d) Gitelman’s syndrome e) Laxative abuse

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Gitelman’s syndrome

**>>REASONING**: Gitelman’s syndrome is the most likely diagnosis due to normotension, hypokalemia, and hypocalciuria. Bartter’s syndrome is less likely due to the absence of hypercalciuria. Conn’s disease is unlikely due to normal blood pressure and elevated renin. Addison’s and laxative abuse are not consistent with the electrolyte and acid-base findings.

## Question #:346

**CLINICAL SCENERIO**: A 21-year-old woman presents to the Emergency department following a collapse at the local supermarket. She tells you that she always feels weak and washed out, and hardly ever has any energy. She takes no regular medication and virtually never sees the doctor. On examination her blood pressure is 100/70 mmHg, pulse is 80 beats per minute and regular. She is slim with a body mass index of 21 kg/m², no abnormal physical signs are noted.

## Investigations

| Na + | 140 mmol/l |
| --- | --- |
| K + | 3.1 mmol/l |
| HCO3 - | 32 mmol/l |
| Urea | 5.9 mmol/l |
| Creatinin e | 85 µmol/l |

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: f) Conn’s syndrome g) Cushing’s syndrome h) Gitelman’s syndrome i) Liddle’s syndrome j) Renal tubular acidosis type

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Gitelman’s syndrome: normotension, hypokalaemia + hypocalciuria

This patient has hypokalaemic metabolic alkalosis with normal / low blood pressure. This fits best with a diagnosis of Gitelman’s syndrome, Bartter’s syndrome, or diuretic abuse. Bartter’s syndrome often presents in infancy as failure to thrive, and isn’t listed as a distractor. Diuretic abuse presents with a similar clinical picture although it too isn’t listed here as an option. Gitelman’s is caused by a mutation in the gene coding for the thiazide-sensitive sodiumchloride co-transporter, and results in a clinical picture similar to that expected with thiazide diuretic use.

Conn’s syndrome is associated with hypertension, as is Liddle’s syndrome, effectively ruling them out as diagnoses here. Renal tubular acidosis type 1 is associated with hypokalaemia and metabolic acidosis, not with a bicarbonate of 32.

**>>DESCRIPTION**: A 21-year-old woman presents with collapse, chronic weakness, and fatigue. Her blood pressure is 100/70 mmHg, pulse 80 bpm. Labs show Na+ 140 mmol/l, K+ 3.1 mmol/l, HCO3- 32 mmol/l, Urea 5.9 mmol/l, Creatinine 85 µmol/l.

**>>OPTIONS**: a) Conn’s syndrome b) Cushing’s syndrome c) Gitelman’s syndrome d) Liddle’s syndrome e) Renal tubular acidosis type

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Gitelman’s syndrome

**>>REASONING**: The patient’s hypokalemic metabolic alkalosis with normotension points to Gitelman’s syndrome, which mimics thiazide diuretic effects due to a co-transporter mutation. Conn’s and Liddle’s syndromes are ruled out by associated hypertension. Renal tubular acidosis type 1 is incorrect due to the presence of metabolic acidosis, not alkalosis.

## Question #:291

**CLINICAL SCENERIO**: A 24-year-old man presents with a five-week history of increasing thirst and frequent urination. The GP suspects diabetes and performs two fasting blood tests showing blood glucose levels of 8.7 mmol/l and 9.2 mmol/l. Urinalysis detects no ketones or protein. The patient’s mother, maternal grandfather, and aunt have type 1 diabetes. C-peptide is consistently high.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate first treatment for managing this condition?

**OPTIONS**: a) Gliclazide b) Metformin c) Pioglitazone d) Insulin e) Sitagliptin

**CORRECT-CHOICE LINE**: A

**REASONING**: The most likely diagnosis in this scenario is maturity onset diabetes of youth, due to the persistently raised c-peptide and strong family history. It normally presents in early adulthood and 1/3 of cases can be treated with oral hypoglycaemics, such as sulphonylureas. It is therefore important to see the response to sulphonylureas first before commencing insulin therapy

**>>DESCRIPTION**: A 24-year-old man with a 5-week history of increased thirst and urination has blood glucose levels of 8.7 and 9.2 mmol/l. Urinalysis is negative for ketones/protein. Family history includes type 1 diabetes in the mother, grandfather, and aunt. C-peptide is consistently high.

**>>OPTIONS**: a) Gliclazide b) Insulin c) Metformin d) Pioglitazone e) Sitagliptin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Gliclazide

**>>REASONING**: The diagnosis is likely maturity-onset diabetes of the young (MODY). Oral hypoglycemics, specifically sulfonylureas like Gliclazide, are appropriate first-line treatment in about one-third of cases.

## Question #:130

**CLINICAL SCENERIO**: A 42-year-old woman presents with feeling warm and more anxious than usual. She had an upper respiratory tract infection three weeks ago. On examination, you find a new tender goitre. Thyroid function tests show TSH 0.2 mU/L (0.5-5.5) and Free T4 25 pmol/L (9.0 - 18).

**QUESTION LINE**: Given the likely underlying diagnosis, what results do you expect from thyroid scintigraphy?

**OPTIONS**: - a) Globally normal uptake - b) Globally increased uptake - c) Globally reduced uptake - d) Well defined area of decreased uptake - e) Well defined area of increased uptake

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: De Quervain’s thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

Subacute thyroiditis is thought to occur following viral infection and typically presents with hyperthyroidism and a new painful goitre. The investigation of choice is thyroid scintigraphy which reveals globally reduced uptake of iodine-131. Most cases are self-limiting and do not require treatment. The other answers provided are not the most common expected findings for De Quervain’s thyroiditis.

Global normal uptake with uniform uptake of iodine-131 is most associated with a normal thyroid gland. Given the abnormal thyroid function and painful goitre, this would be an unexpected result in this case.

Globally increased uptake is most commonly associated with Grave’s disease.

A well-defined area of decreased uptake would be most consistent with a cold (hypofunctioning) thyroid nodule. This finding would also not be consistent with the painful goitre or hyperthyroidism noted on blood tests in this case.

**>>DESCRIPTION**: A 42-year-old woman with recent URI presents with anxiety, warmth, and a tender goitre. TSH is 0.2 mU/L (low), and Free T4 is 25 pmol/L (high).

**>>OPTIONS**: a) Globally increased uptake b) Globally normal uptake c) Globally reduced uptake d) Well defined area of decreased uptake e) Well defined area of increased uptake

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Globally reduced uptake

**>>REASONING**: De Quervain’s thyroiditis (subacute thyroiditis) presents with hyperthyroidism and a painful goitre, typically showing globally reduced uptake on thyroid scintigraphy. Normal uptake is associated with a normal thyroid, increased uptake with Grave’s disease, and a well-defined area of decreased uptake with a cold nodule.

## Question #:138

**CLINICAL SCENERIO**: A 42-year-old woman presents with feeling warm and more anxious than usual. She had an upper respiratory tract infection three weeks ago. On examination, you find a new tender goitre.

**QUESTION LINE**: Given the likely underlying diagnosis, what results do you expect from thyroid scintigraphy?

**OPTIONS**: a) Globally normal uptake b) Globally increased uptake c) Globally reduced uptake d) Well defined area of decreased uptake e) Well defined area of increased uptake

**CORRECT-CHOICE LINE**: c

**REASONING**: De Quervain’s thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

Subacute thyroiditis is thought to occur following viral infection and typically presents with hyperthyroidism and a new painful goitre. The investigation of choice is thyroid scintigraphy which reveals globally reduced uptake of iodine-131. Most cases are self-limiting and do not require treatment. The other answers provided are not the most common expected findings for De Quervain’s thyroiditis.

Global normal uptake with uniform uptake of iodine-131 is most associated with a normal thyroid gland. Given the abnormal thyroid function and painful goitre, this would be an unexpected result in this case.

Globally increased uptake is most commonly associated with Grave’s disease.

A well-defined area of decreased uptake would be most consistent with a cold (hypofunctioning) thyroid nodule. This finding would also not be consistent with the painful goitre or hyperthyroidism noted on blood tests in this case.

A well-defined area of increased uptake would be most consistent with a hot (hyperfunctioning) thyroid nodule.

**>>DESCRIPTION**: A 42-year-old woman presents with warmth, anxiety, and a tender goitre after a recent upper respiratory tract infection. Given the lab results (TSH 0.2 mU/L, Free T4 25 pmol/L), what are the expected thyroid scintigraphy findings?

**>>OPTIONS**: a) Globally increased uptake b) Globally normal uptake c) Globally reduced uptake d) Well defined area of decreased uptake e) Well defined area of increased uptake

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Globally reduced uptake

**>>REASONING**: De Quervain’s thyroiditis is the likely diagnosis, indicated by hyperthyroidism and a painful goitre. Thyroid scintigraphy would reveal globally reduced uptake of iodine-131. Other options are less likely: normal uptake suggests a normal thyroid, increased uptake suggests Grave’s, and focal uptake changes suggest thyroid nodules.

## Question #:18

**CLINICAL SCENERIO**: A 75-year-old man presents to the diabetic clinic with newly identified hyperglycaemia. He has a two-month history of polyuria, polydipsia, and diarrhoea, with a blood sugar of 18.4 mmol/L. He lost 6kg, now weighing 61kg. His past medical history includes hypertension and a DVT diagnosed three months ago. He takes amlodipine 10mg and warfarin.

**QUESTION LINE**: What is the diagnosis?

**OPTIONS**: - a) Glucagonoma - b) Type one diabetes mellitus - c) Cushing’s syndrome - d) Drug induced diabetes - e) Type two diabetes mellitus

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Glucagonoma is an uncommon tumour of the pancreatic alpha cells. It can present with new or worsening diabetes mellitus, venous thromboembolism, the classic rash of necrolytic migratory erythema (a painful, pruritic maculopapular rash occuring typically at sites of friction with clothing…) and other symptoms of hyperglucagonaemia (diarrhoea, weight loss, anaemia). Type one diabetes mellitus can of course present with the osmotic symptoms of hyperglycaemia and weight loss but would be unlikely in a patient of this age with no history of autoimmune disease. Type two diabetes mellitus would be uncommon in a non-obese older patient and is less likely to present with osmotic symptoms.

**>>DESCRIPTION**: A 75-year-old man with new hyperglycemia, polyuria, polydipsia, and diarrhea. Blood sugar is 18.4 mmol/L. He lost 6kg. PMH: HTN and DVT. Medications: amlodipine and warfarin.

**>>OPTIONS**: a) Cushing’s syndrome b) Drug induced diabetes c) Glucagonoma d) Type one diabetes mellitus e) Type two diabetes mellitus

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Glucagonoma

**>>REASONING**: Glucagonoma presents with diabetes, venous thromboembolism, necrolytic migratory erythema, diarrhea, and weight loss. Type 1 DM is less likely in an older patient without autoimmune history. Type 2 DM is less likely in a non-obese patient with osmotic symptoms.

## Question #:165

**CLINICAL SCENERIO**: A 19-year-old pharmacy student collapses at work and is admitted to the hospital. She was alert after the collapse, and her blood glucose was 1.5 mmol/l. She had two prior collapse episodes. Her observations include a blood pressure of 127/77 mmHg, pulse of 81 bpm, and oxygen saturation of 97%.

**QUESTION LINE**: What is the best first-line investigation?

**OPTIONS**: 1. Glucose, c-peptide and insulin 2. Morning c-peptide 3. Evening c-peptid 4. Computed tomography (CT) scan of the abdomen 5. Oral glucose tolerance test

**CORRECT-CHOICE LINE**: a

**REASONING**: Measuring blood glucose, insulin and c-peptide allows the differentiation between the causes of hypoglycaemic attacks, including insulinoma, insulin or sulphonylurea misuse.

**>>DESCRIPTION**: A 19-year-old pharmacy student collapses at work with a blood glucose of 1.5 mmol/l. She had two prior collapse episodes. What is the best first-line investigation?

**>>OPTIONS**: 1. Computed tomography (CT) scan of the abdomen 2. Evening c-peptid 3. Glucose, c-peptide and insulin 4. Morning c-peptide 5. Oral glucose tolerance test

**>>CORRECT-CHOICE LINE**: 3

**>>CORRECT-CHOICE\_TEXT**: Glucose, c-peptide and insulin

**>>REASONING**: Measuring blood glucose, insulin, and c-peptide helps differentiate the causes of hypoglycemic attacks, such as insulinoma, insulin misuse, or sulfonylurea use.

## Question #:308

**CLINICAL SCENERIO**: A 43-year-old woman presents to the endocrinology clinic with a 2-month history of weight loss, sweating, and heat intolerance. Her blood tests and thyroid scintigraphy are provided.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a. Grave’s disease b. Hashimoto’s thyroiditis c. Subacute thyroiditis d. Thyroid cancer e. Toxic multinodular goitre

**CORRECT-CHOICE LINE**: a.

**REASONING**: This woman’s clinical presentation and thyroid function tests are consistent with a diagnosis of hyperthyroidism. The thyroid scintigraphy shows global uptake which is consistent with Grave’s disease. Grave’s disease is therefore the correct answer.

Hashimoto’s thyroiditis is a cause of hypothyroidism rather than hyperthyroidism. This cannot be the case as confirmed by the thyroid function tests and is therefore incorrect.

Subacute thyroiditis is a possibility due to this woman’s recent illness. This often results in subacute hyperthyroidism followed by a period of hypothyroidism. Scintigraphy shows decreased uptake in this condition which is not the case in this instance. This is therefore not the correct answer.

Thyroid cancers can cause hyperthyroidism however scintigraphy would show a discrete mass with increased uptake rather than homogenous uptake. This is not the case here and therefore Grave’s disease is a more likely diagnosis.

Toxic multinodular goitre is another cause of hyperthyroidism however is associated with patchy uptake on thyroid scintigraphy. As the uptake in this instance is homogenous, this is unlikely to be the underlying cause.

**>>DESCRIPTION**: A 43-year-old woman presents to endocrinology with weight loss, sweating, and heat intolerance. Blood tests and thyroid scintigraphy are provided.

**>>OPTIONS**: a. Grave’s disease b. Hashimoto’s thyroiditis c. Subacute thyroiditis d. Thyroid cancer e. Toxic multinodular goitre

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Grave’s disease

**>>REASONING**: The patient’s presentation and tests suggest hyperthyroidism, with global uptake on scintigraphy consistent with Grave’s disease. Hashimoto’s thyroiditis causes hypothyroidism, subacute thyroiditis has decreased uptake, thyroid cancer has focal uptake, and toxic multinodular goitre has patchy uptake; therefore, they are less likely.

## Question #:311

**CLINICAL SCENERIO**: A 54-year-old man with previous non-functioning pituitary adenoma (NFPA) resection, on thyroxine and hydrocortisone, presents with worsening low mood, malaise, and reduced exercise capacity. He has central adiposity, dyslipidaemia, epilepsy, and IHD. Investigations show low IGF-1, normal FT4, and suppressed TSH. An insulin tolerance test is contraindicated.

**QUESTION LINE**: Which one of the following tests is most appropriate in his case to confirm the diagnosis of adult GH deficiency?

**OPTIONS**: - a) Domperidone test - b) Growth hormone levels - c) Growth hormone releasing hormone (GHRH)-arginine stimulation test - d) Insulin-like growth factor- binding protein measurement - e) Glucose tolerance test

**CORRECT-CHOICE LINE**: c

**REASONING**: Arginine-GHRH stimulation test is employed to assess GH secretion in patients with ischemic heart disease or seizures where insulin tolerance test will be inappropriate. The clinical features of growth hormone deficiency (GHD) are non-specific and include lethargy, low mood, poor quality of life, loss of muscle mass, and central adiposity. A low IGF-1 in such a clinical context may point towards GHD, which needs to be confirmed with dynamic tests for GH secretion. Insulin tolerance test (ITT) is considered the gold standard for assessing GH secretion, although it needs to be conducted carefully in a closely-monitored space due to the risks associated with hypoglycaemia. ITT is contra-indicated in patients with seizures or ischaemic heart disease. As a result, an alternative test such as arginine-GHRH stimulation test is employed (as in this clinical scenario where the patient has IHD). About 30-40% of patients with GHD may have a normal IGF-1 level. IGF-1 levels are influenced by age, time of onset of GHD, and degree of hypopituitarism. Domperidone test is used in the evaluation of hyperprolactinemia and macroprolactinaemia associated disorders while glucose tolerance test with measurement of IGF-1 levels is used in the evaluation of suspected acromegaly.

**>>DESCRIPTION**: A 54-year-old man post-NFPA resection, on thyroxine and hydrocortisone, presents with worsening low mood, malaise, and reduced exercise. He has central adiposity, dyslipidaemia, epilepsy, and IHD, and a low IGF-1. Insulin tolerance test is contraindicated.

**>>OPTIONS**: a) Domperidone test b) Glucose tolerance test c) Growth hormone releasing hormone (GHRH)-arginine stimulation test d) Growth hormone levels e) Insulin-like growth factor- binding protein measurement

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Growth hormone releasing hormone (GHRH)-arginine stimulation test

**>>REASONING**: The arginine-GHRH stimulation test is the most appropriate to assess GH secretion in this patient with IHD and contraindication to ITT. Low IGF-1, although suggestive, needs confirmation with dynamic testing. Other options are incorrect because they assess for different conditions or are not suitable in this patient’s case.

## Question #:55

**CLINICAL SCENERIO**: A 22-year-old woman with a history of partial Kallmann syndrome comes to the fertility clinic for review. She got married some 6 months earlier and wants to start a family. She has normal external genitalia and sparse pubic and axillary hair and has a normal body mass index of 23kg/m².

**QUESTION LINE**: Which of the following is the most appropriate intervention?

**OPTIONS**: a) Clomiphene b) HCG and FSH then IVF c) Metformin d) Oestrogen e) Referral for adoption

**CORRECT-CHOICE LINE**: The correct answer is HCG and FSH then IVF.

**REASONING**: Restoration of ovulation in females with Kallmann syndrome is complex and often requires HCG to drive production of gonadal steroid hormones, FSH to drive ovulation, harvesting of eggs, and IVF. This process is most effective in achieving successful pregnancy.

Clomiphene does induce ovulation and is useful in patients with other conditions such as polycystic ovarian syndrome. It is however very unlikely to be effective in patients with gonadotrophin failure like Kallmann syndrome. Metformin has previously been used in the management of PCOS. Oestrogen doesn’t restore ovulation in patients with Kallmann syndrome but may be considered for patients who don’t want to get pregnant. In the event that fertility treatment isn’t successful, adoption can be considered.

**>>DESCRIPTION**: A 22-year-old woman with partial Kallmann syndrome seeks fertility treatment, having normal external genitalia, sparse pubic/axillary hair, and a BMI of 23kg/m².

**>>OPTIONS**: a) Clomiphene b) HCG and FSH then IVF c) Metformin d) Oestrogen e) Referral for adoption

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: HCG and FSH then IVF

**>>REASONING**: HCG and FSH, followed by IVF, are most effective for restoring ovulation and achieving pregnancy in Kallmann syndrome due to the need for hormonal stimulation and assisted reproduction. Clomiphene, Metformin and Oestrogen are not effective in Kallmann syndrome.

## Question #:197

**CLINICAL SCENERIO**: A 22-year-old man with excessive thirst, urination, and a family history of diabetes presents with a BMI of 20.5 kg/m². Lab results show elevated random glucose.

**QUESTION LINE**: Which mutation is associated with the best response to low-dose sulphonylureas?

**OPTIONS**: - a) Glucokinase - b) HNF1A mutation - c) HNF1B mutation - d) HNF4A mutation - e) PDX1 mutation

**CORRECT-CHOICE LINE**: b

**REASONING**: MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

The patient most likely has a diagnosis of maturity-onset diabetes of the young (MODY) given his raised blood glucose, young age, strong family history of diabetes, and normal BMI.

HNF1A mutation is correct. MODY associated with HNF1A often responds well to treatment with low-dose sulfonylureas.

Glucokinase is incorrect. This mutation is associated with MODY 2. This subtype has a low rate of complications and often does not need treatment.

HNF1B mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

HNF4A mutation is incorrect. Although patients with this subtype may also respond favourably to sulphonylureas, the response is usually less marked compared to HNF1A mutations.

PDX1 mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

**>>DESCRIPTION**: A 22-year-old man with polyuria, polydipsia, a family history of diabetes, and elevated random glucose levels. Which mutation shows the best response to low-dose sulfonylureas?

**>>OPTIONS**: a) Glucokinase b) HNF1A mutation c) HNF1B mutation d) HNF4A mutation e) PDX1 mutation

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: HNF1A mutation

**>>REASONING**: HNF1A mutation is the best choice because MODY associated with HNF1A often responds well to low-dose sulfonylureas. Other options are incorrect because either they do not respond to sulfonylureas or respond less effectively.

## Question #:251

**CLINICAL SCENERIO**: A 38-year-old woman presents with calf pain on walking 50 meters, with a history of migraine. She smokes heavily, drinks wine daily, and has a family history of diabetes and early heart attack. Examination reveals tendon xanthomas, xanthelasma, and corneal arcs.

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: a) Tangier disease b) Homozygous familial hypercholesterolaemia c) Heterozygous familial hypercholesterolaemia d) Familial hypertriglyceridaemia e) Apo CII deficiency

**CORRECT-CHOICE LINE**: C

**REASONING**: This patient’s symptoms suggest intermittent claudication and combined with the signs of hypercholesterolaemia especially the tendon xanthomas would suggest one of the familial hypercholesterolaemias. Patients with homozygous familial hypercholesterolaemia, present with early cardiovascular disease, sometimes as early as the second decade of life, whereas, patients with heterozygous familial hypercholesterolaemia rarely present before the age 30. Thus, this case is more suggestive of heterozygous familial hypercholesterolaemia.

In heterozygous familial hypercholesterolaemia, total cholesterol would typically be above 7.9 mmol/l, with normal triglyceride levels. In homozygotes it is typically above 15 mmol/l

**>>DESCRIPTION**: A 38-year-old woman with calf pain on walking, a history of migraine, heavy smoking, and a family history of diabetes and early heart attack, presents with tendon xanthomas, xanthelasma, and corneal arcs.

**>>OPTIONS**: a) Apo CII deficiency b) Familial hypertriglyceridaemia c) Heterozygous familial hypercholesterolaemia d) Homozygous familial hypercholesterolaemia e) Tangier disease

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Heterozygous familial hypercholesterolaemia

**>>REASONING**: The most likely diagnosis is heterozygous familial hypercholesterolaemia, given the patient’s symptoms of claudication and signs of hypercholesterolaemia (xanthomas). The patient’s age makes homozygous familial hypercholesterolaemia less likely. Other options are less probable given the clinical presentation.

## Question #:25

**CLINICAL SCENERIO**: A 48-year-old woman is referred to the endocrinology clinic with a 6-week history of weight gain and fatigue.

Her observations are as follows:

*  Temperature 36.5ºC
*  Heart rate 77 beats/min
*  Blood pressure 171/97mmHg
*  Respiratory rate 16 breaths/min
*  Oxygen saturations 98% on air

Laboratory tests:

Hb

136 g/L

(115 - 160)

| Platelets | 288 \* 10 9 /L | (150 - 400) |
| --- | --- | --- |
| WBC | 9.4 \* 10 9 /L | (4.0 - 11.0) |
| Na + | 142 mmol/L | (135 - 145) |
| K + | 3.4 mmol/L | (3.5 - 5.0) |
| Urea | 4.1 mmol/L | (2.0 - 7.0) |
| Creatinine | 112 µmol/L | (55 - 120) |
| Glucose 16 mmol/L | (4 - 7) |  |

Low-dose dexamethasone suppression test:

**QUESTION LINE**: What is the next best investigation?

**OPTIONS**: - a) High-dose dexamethasone suppression test - b) Insulin tolerance test - c) Petrosal sinus ACTH sampling - d) Pituitary MRI - e) Urinary cortisol

**CORRECT-CHOICE LINE**: Correct anser is a.

**REASONING**: The high-dose dexamethasone suppression test is useful for distinguishing between ACTH dependent (e.g. pituitary source) and non-ACTH dependent (e.g. ectopic and adrenal source) causes of Cushings’s syndrome

This patient has Cushing’s syndrome. Due to increased glucocorticoid production, symptoms include weight gain and hyperglycaemia as seen in this patient. Hypertension is another common finding also seen here. Laboratory tests may reveal hypokalaemic metabolic acidosis. The best test to diagnose Cushing’s syndrome is the low-dose dexamethasone test. This has been done for this patient and shows a cortisol level that has not been suppressed despite exogenous steroid use. Once Cushing’s syndrome has been confirmed, additional localisation tests need to be completed. One of these includes the high-dose dexamethasone suppression test which is used to discriminate between Cushing’s disease (i.e. pituitary adenoma) and ectopic ACTH production. Suppressed cortisol levels following high doses of glucocorticoids confirms a pituitary cause, as opposed to normal levels of cortisol that suggest an adrenal cause.

The insulin stress test is used to differentiate Cushing’s syndrome from pseudoCushing’s. Pseudo-Cushing’s presents similarly to Cushing’s syndrome; however, rather than being caused by excessive corticosteroid levels, it is due to alcohol excess or severe depression. There is nothing in this patient’s history to suggest depression or alcohol excess. Furthermore, the positive finding following the lowdose dexamethasone suppression test points away from a diagnosis of pseudoCushing’s.

Petrosal sinus ACTH sampling is another investigation that can be used to help distinguish between a pituitary or adrenal cause of Cushing’s syndrome. However, it is an invasive investigation and easier tests such as the high-dose dexamethasone suppression test should be considered first.

A pituitary MRI can be used to diagnose a pituitary cause of Cushing’s syndrome. However, it is not the next recommended investigation. Rather, a high-dose dexamethasone suppression test is easier and cheaper to complete and should be completed next.

Urinary cortisol is used as an initial investigation in the diagnosis of Cushing’s syndrome. It is not a specific localisation test.

**>>DESCRIPTION**: A 48-year-old woman presents with weight gain, fatigue, BP 171/97mmHg, and glucose 16 mmol/L. Low-dose dexamethasone suppression test was performed.

**>>OPTIONS**: a) High-dose dexamethasone suppression test b) Insulin tolerance test c) Petrosal sinus ACTH sampling d) Pituitary MRI e) Urinary cortisol

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: High-dose dexamethasone suppression test

**>>REASONING**: The patient has Cushing’s syndrome. After confirmation with a low-dose dexamethasone test, the next step is to differentiate between ACTH-dependent and non-ACTH-dependent causes. A high-dose dexamethasone suppression test helps in this differentiation. Insulin stress test differentiates Cushing’s from pseudo-Cushing’s, petrosal sinus ACTH sampling is invasive, pituitary MRI is not the next recommended step, and urinary cortisol is an initial, not localizing, test.

## Question #:30

**CLINICAL SCENERIO**: A 48-year-old woman is referred to the endocrinology clinic with a 6-week history of weight gain and fatigue.

Her observations are as follows:

*  Temperature 36.5ºC
*  Heart rate 77 beats/min
*  Blood pressure 171/97mmHg
*  Respiratory rate 16 breaths/min
*  Oxygen saturations 98% on air

## Laboratory tests:

| Hb | 136 g/L | (115 - 160) |
| --- | --- | --- |
| Platelets | 288 \* 10 9 /L | (150 - 400) |
| WBC | 9.4 \* 10 9 /L | (4.0 - 11.0) |
| Na + | 142 mmol/L | (135 - 145) |
| K + | 3.4 mmol/L | (3.5 - 5.0) |
| Urea | 4.1 mmol/L | (2.0 - 7.0) |
| Creatinine | 112 µmol/L | (55 - 120) |
| Glucose 16 mmol/L | (4 - 7) |  |

## Low-dose dexamethasone suppression test:

**QUESTION LINE**: What is the next best investigation?

**OPTIONS**: - a) High-dose dexamethasone suppression test - b) Insulin tolerance test - c) Petrosal sinus ACTH sampling - d) Pituitary MRI - e) Urinary cortisol

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: The high-dose dexamethasone suppression test is useful for distinguishing between ACTH dependent (e.g. pituitary source) and non-ACTH dependent (e.g. ectopic and adrenal source) causes of Cushings’s syndrome

This patient has Cushing’s syndrome. Due to increased glucocorticoid production, symptoms include weight gain and hyperglycaemia as seen in this patient. Hypertension is another common finding also seen here. Laboratory tests may reveal hypokalaemic metabolic acidosis. The best test to diagnose Cushing’s syndrome is the low-dose dexamethasone test. This has been done for this patient and shows a cortisol level that has not been suppressed despite exogenous steroid use. Once Cushing’s syndrome has been confirmed, additional localisation tests need to be completed. One of these includes the high-dose dexamethasone suppression test which is used to discriminate between Cushing’s disease (i.e. pituitary adenoma) and ectopic ACTH production. Suppressed cortisol levels following high doses of glucocorticoids confirms a pituitary cause, as opposed to normal levels of cortisol that suggest an adrenal cause.

The insulin stress test is used to differentiate Cushing’s syndrome from pseudoCushing’s. Pseudo-Cushing’s presents similarly to Cushing’s syndrome; however, rather than being caused by excessive corticosteroid levels, it is due to alcohol excess or severe depression. There is nothing in this patient’s history to suggest depression or alcohol excess. Furthermore, the positive finding following the lowdose dexamethasone suppression test points away from a diagnosis of pseudoCushing’s.

Petrosal sinus ACTH sampling is another investigation that can be used to help distinguish between a pituitary or adrenal cause of Cushing’s syndrome. However, it is an invasive investigation and easier tests such as the high-dose dexamethasone suppression test should be considered first.

A pituitary MRI can be used to diagnose a pituitary cause of Cushing’s syndrome. However, it is not the next recommended investigation. Rather, a high-dose dexamethasone suppression test is easier and cheaper to complete and should be completed next.

Urinary cortisol is used as an initial investigation in the diagnosis of Cushing’s syndrome. It is not a specific localisation test.

**>>DESCRIPTION**: A 48-year-old woman presents with weight gain, fatigue, hypertension (171/97mmHg), and hyperglycemia (glucose 16 mmol/L) after a 6-week history. Low-dose dexamethasone suppression test was performed.

**>>OPTIONS**: a) High-dose dexamethasone suppression test b) Insulin tolerance test c) Petrosal sinus ACTH sampling d) Pituitary MRI e) Urinary cortisol

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: High-dose dexamethasone suppression test

**>>REASONING**: The patient has Cushing’s syndrome confirmed by a low-dose dexamethasone suppression test. The next step is a high-dose dexamethasone suppression test to differentiate between ACTH-dependent (pituitary) and ACTH-independent (ectopic/adrenal) causes. Insulin stress test differentiates Cushing’s from pseudo-Cushing’s which is less likely here, Petrosal sinus ACTH sampling is invasive, Pituitary MRI and urinary cortisol are not the next best steps.

## Question #:344

**CLINICAL SCENERIO**: A 48-year-old woman is referred to the endocrinology clinic with a 6-week history of weight gain and fatigue.

Her observations are as follows:

* Temperature 36.5ºC
* Heart rate 77 beats/min
* Blood pressure 171/97mmHg
* Respiratory rate 16 breaths/min
* Oxygen saturations 98% on air

## Laboratory tests:

| Hb | 136 g/L | (115 - 160) |
| --- | --- | --- |
| Platelets | 288 \* 10 9 /L | (150 - 400) |
| WBC | 9.4 \* 10 9 /L | (4.0 - 11.0) |
| Na + | 142 mmol/L | (135 - 145) |
| K + | 3.4 mmol/L | (3.5 - 5.0) |
| Urea | 4.1 mmol/L | (2.0 - 7.0) |
| Creatinine | 112 µmol/L | (55 - 120) |
| Glucose 16 mmol/L | (4 - 7) |  |

## Low-dose dexamethasone suppression test:

**QUESTION LINE**: What is the next best investigation?

**OPTIONS**: f) High-dose dexamethasone suppression test g) Insulin tolerance test h) Petrosal sinus ACTH sampling i) Pituitary MRI j) Urinary cortisol

**CORRECT-CHOICE LINE**: Correct anser is a.

**REASONING**: The high-dose dexamethasone suppression test is useful for distinguishing between ACTH dependent (e.g. pituitary source) and non-ACTH dependent (e.g. ectopic and adrenal source) causes of Cushings’s syndrome

This patient has Cushing’s syndrome. Due to increased glucocorticoid production, symptoms include weight gain and hyperglycaemia as seen in this patient. Hypertension is another common finding also seen here. Laboratory tests may reveal hypokalaemic metabolic acidosis. The best test to diagnose Cushing’s syndrome is the low-dose dexamethasone test. This has been done for this patient and shows a cortisol level that has not been suppressed despite exogenous steroid use. Once Cushing’s syndrome has been confirmed, additional localisation tests need to be completed. One of these includes the high-dose dexamethasone suppression test which is used to discriminate between Cushing’s disease (i.e. pituitary adenoma) and ectopic ACTH production. Suppressed cortisol levels following high doses of glucocorticoids confirms a pituitary cause, as opposed to normal levels of cortisol that suggest an adrenal cause.

The insulin stress test is used to differentiate Cushing’s syndrome from pseudoCushing’s. Pseudo-Cushing’s presents similarly to Cushing’s syndrome; however, rather than being caused by excessive corticosteroid levels, it is due to alcohol excess or severe depression. There is nothing in this patient’s history to suggest depression or alcohol excess. Furthermore, the positive finding following the lowdose dexamethasone suppression test points away from a diagnosis of pseudoCushing’s.

Petrosal sinus ACTH sampling is another investigation that can be used to help distinguish between a pituitary or adrenal cause of Cushing’s syndrome. However, it is an invasive investigation and easier tests such as the high-dose dexamethasone suppression test should be considered first.

A pituitary MRI can be used to diagnose a pituitary cause of Cushing’s syndrome. However, it is not the next recommended investigation. Rather, a high-dose dexamethasone suppression test is easier and cheaper to complete and should be completed next.

Urinary cortisol is used as an initial investigation in the diagnosis of Cushing’s syndrome. It is not a specific localisation test.

**>>DESCRIPTION**: A 48-year-old woman presents with 6 weeks of weight gain, fatigue, hypertension (171/97mmHg), hyperglycemia (glucose 16 mmol/L), and hypokalemia (K+ 3.4 mmol/L). A low-dose dexamethasone suppression test was performed.

**>>OPTIONS**: a) High-dose dexamethasone suppression test b) Insulin tolerance test c) Petrosal sinus ACTH sampling d) Pituitary MRI e) Urinary cortisol

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: High-dose dexamethasone suppression test

**>>REASONING**: The patient’s presentation (weight gain, fatigue, hypertension, hyperglycemia, hypokalemia) and the positive low-dose dexamethasone suppression test confirm Cushing’s syndrome. The next best investigation is the high-dose dexamethasone suppression test, which helps distinguish between ACTH-dependent (e.g., pituitary) and non-ACTH-dependent (e.g., adrenal or ectopic) causes. Suppressed cortisol levels after a high dose suggest a pituitary origin (Cushing’s disease), while non-suppression points towards an adrenal or ectopic source. Other options like the insulin tolerance test (for pseudo-Cushing’s), pituitary MRI, or petrosal sinus sampling are either less appropriate as the immediate next step (MRI and petrosal sampling are typically subsequent or more invasive) or initial diagnostic tests (urinary cortisol) not localization tests.

## Question #:60

**CLINICAL SCENERIO**: A 67-year-old man, inpatient after elective parathyroidectomy for hyperparathyroidism and hypercalcaemia, now presents with finger paraesthesia and severe ankle pain. X-rays reveal osteolytic lesions suspicious for metastatic disease. Medications include paracetamol, tramadol, and dalteparin. Adjusted Calcium is 1.84 mmol/L and Magnesium is 0.7 mmol/L.

**QUESTION LINE**: What is the most likely explanation for his current symptoms?

**OPTIONS**: - a) Metastatic parathyroid cancer - b) Secondary hyperparathyroidism - c) Hypomagnesaemia - d) Hungry bone syndrome - e) Secondary hypoparathyroidism

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Hypocalcaemia after parathyroid surgery is relatively common and usually ‘benign’ and associated with a transient hypoparathyroidism. However, it can sometimes be more marked and give rise to symptoms such as perioral or finger paraesthesia. This state alone would not, however, explain his ankle pain or x-ray findings.

Although hypomagnesaemia may also be present and should be treated, it does not explain the symptoms. Metastatic parathyroid cancer is a possibility given the x-ray findings, but is very uncommon and is less likely given that his hyperparathyroidism and hypercalcaemia was long standing (i.e. indolent). Secondary hyperparathyroidism is the syndrome of appropriately raised parathyroid hormone in response to hypocalcaemia, usually secondary to chronic kidney disease. Secondary hypoparathyroidism describes the normal parathyroid hormone suppression that occurs in hypercalcaemia secondary to non-parathyroid causes, such as malignanc

**>>DESCRIPTION**: 67-year-old male post-parathyroidectomy presents with finger paresthesia, severe ankle pain, and osteolytic lesions. Calcium 1.84 mmol/L, Magnesium 0.7 mmol/L. What is the most likely diagnosis?

**>>OPTIONS**: a) Hungry bone syndrome b) Hypomagnesaemia c) Metastatic parathyroid cancer d) Secondary hyperparathyroidism e) Secondary hypoparathyroidism

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Hungry bone syndrome

**>>REASONING**: Hungry bone syndrome is the most likely explanation, given the recent parathyroidectomy, hypocalcemia, and osteolytic lesions. Hypomagnesemia may be present but doesn’t explain all symptoms. Metastatic parathyroid cancer is less likely due to the long-standing indolent nature of hyperparathyroidism.

## Question #:296

**CLINICAL SCENERIO**: A 62-year-old male from India presents with a 5-day history of feeling unwell, with no recent cough, diarrhea, vomiting, or dysuria. The patient has dry mucous membranes, cool peripheries, and a JVP of +1cm. Blood tests show elevated WBC, neutrophils, Na+, urea, creatinine, CRP, glucose, and ketones, along with arterial blood gas results.

**QUESTION LINE**: What is the unifying diagnosis?

**OPTIONS**: a. Diabetic ketoacidosis (DKA) b. Lactic acidosis c. Hyperosmolar hyperglycaemic state (HHS) d. Urinary tract sepsis e. Chest sepsis

**CORRECT-CHOICE LINE**: C

**REASONING**: This patient has presented with dehydration and non-specific symptoms and a diagnosis difficult to diagnose clinically. However, his biochemistry is diagnostic: calculation of his osmolality, (2[Na + K] + urea+ glucose) reveals an osmolality greater 371.4mosmol/kg. He is likely to present acutely with undiagnosed type 2 diabetes mellitus and a diagnosis of HHS, previously known as HONK.

There is no evidence to suggest uro or chest sepsis but an infectious underlying decompensating trigger should be considered with a prescription of broad spectrum antibiotics. Although lactate is mildly raised, this is likely secondary to intravascular dehydration and hypoperfusion of internal organs. Lactic acidosis alone does not account for the full biochemical picture. Ketones are present and the patient is mild acidotic. However, be aware that neither is sufficiently significant for a diagnosis of DKA.

**>>DESCRIPTION**: A 62-year-old male from India presents with a 5-day history of malaise, dehydration, and abnormal blood test results, including elevated glucose and osmolality.

**>>OPTIONS**: a. Chest sepsis b. Diabetic ketoacidosis (DKA) c. Hyperosmolar hyperglycaemic state (HHS) d. Lactic acidosis e. Urinary tract sepsis

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Hyperosmolar hyperglycaemic state (HHS)

**>>REASONING**: The unifying diagnosis is Hyperosmolar hyperglycemic state (HHS) due to elevated osmolality and glucose. Other options are less likely due to the overall biochemical picture. Mildly elevated lactate is likely secondary to dehydration.

## Question #:92

**CLINICAL SCENERIO**: A 62-year-old woman presents with agitation, nausea, vomiting, productive cough with green sputum, and chest pain. She is confused and agitated. Examination reveals a heart rate of 150bpm, blood pressure of 103/65mmHg, temperature 40.1º and oxygen saturation of 94%. Past medical history includes myocardial infarction, polycystic ovaries, hypertension, and hyperthyroidism (diagnosed four months ago) with poor treatment compliance. Medications include aspirin, atorvastatin, amlodipine, and carbimazole. Blood tests are pending.

**QUESTION LINE**: What feature of her presentation is most suggestive of thyroid storm?

**OPTIONS**: - a) Productive cough - b) History of aspirin use - c) Chest pain - d) Confusion - e) Hyperpyrexia

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: In thyroid storm, hyperpyrexia, tachycardia, cardiac failure and agitation are the key diagnostic elements

This patient has features of thyroid storm. The most important features are high fever and tachycardia, with temperatures becoming very high, even above 40ºC, and heart rate can be as high as 180bpm. Thyroid storm can be triggered by infection, and therefore the productive cough is relevant but not a distinguishing feature. The use of aspirin can worsen thyrotoxicosis as well by displacing T4 from thyroid binding globulin but is not a main feature. Agitation and confusion are both features but are less discriminating than hyperpyrexia.

**>>DESCRIPTION**: A 62-year-old woman presents with agitation, nausea, productive cough, and chest pain. She is confused with HR 150bpm, BP 103/65mmHg, temperature 40.1º, and SpO2 94%. PMH includes MI, PCOS, hypertension, and poorly controlled hyperthyroidism. Medications: aspirin, atorvastatin, amlodipine, and carbimazole.

**>>OPTIONS**: a) Chest pain b) Confusion c) History of aspirin use d) Hyperpyrexia e) Productive cough

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Hyperpyrexia

**>>REASONING**: Hyperpyrexia is the most suggestive feature of thyroid storm, with temperatures often exceeding 40ºC. While productive cough can be a trigger, aspirin use can worsen thyrotoxicosis, and confusion/agitation are features, they are less specific than hyperpyrexia.

## Question #:122

**CLINICAL SCENERIO**: A 67-year-old lady presents a week after Campylobacter gastroenteritis with increased thirst and polyuria. She drinks 9-10 glasses of water/tea a day (twice her normal intake) and her urine is clear. She feels low in energy with easy fatigue and leg cramps after standing. She takes indapamide for hypertension. On examination, she appears euvolaemic, slow, overweight, and lethargic. Blood pressure is 105/90mmHg and heart rate is 67/min. Labs show K+ 2.6 mmol/l, normal TSH and Cortisol.

**QUESTION LINE**: What is the likeliest cause of these symptoms?

**OPTIONS**: a) Addison’s disease b) Hypothyroidism c) Syndrome of inappropriate ADH secretion d) Psychogenic polydipsia e) Hypokalaemia

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Hypokalaemia is a rare cause of polyuria and polydipsia. This lady has polydipsia and polyuria. Of the above options, only hypokalaemia and psychogenic polydipsia are recognised causes of these symptoms. Hyperthyroidism can rarely cause polydipsia but not hypothyroidism. Addison’s disease can cause hypercalcaemia which can cause these symptoms but her calcium is normal. Psychogenic polydipsia can occur but this is usually a chronic problem. This lady has presented very soon after her gastroenteritis and has evidence of hypokalaemia making this a rare but the likeliest cause of her symptoms. Her indapamide is probably contributing to the persistent hypokalaemia and should be stopp

**>>DESCRIPTION**: 67F presents 1 week after Campylobacter gastroenteritis with increased thirst, polyuria, fatigue, and leg cramps. Drinks twice her normal fluid intake. Takes indapamide. Exam: euvolaemic, slow, lethargic. BP 105/90, HR 67. K+ 2.6, normal TSH/Cortisol.

**>>OPTIONS**: a) Addison’s disease b) Hypokalaemia c) Hypothyroidism d) Psychogenic polydipsia e) Syndrome of inappropriate ADH secretion

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Hypokalaemia

**>>REASONING**: Hypokalemia can cause polyuria and polydipsia. While psychogenic polydipsia is a possibility, the recent gastroenteritis and hypokalemia make hypokalemia the most likely cause. Addison’s would cause hypercalcemia. Hypothyroidism is not associated with polydipsia.

## Question #:277

**CLINICAL SCENERIO**: A 17-year-old girl presents to the emergency department with recurrent episodes of waking paralysis lasting 2 hours, with preserved consciousness and no significant medical history. Examination reveals normal findings except for a jerky baseline on ECG with flat T waves.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a. Partial or absence seizures - b. Guillain-Barre syndrome - c. Botulinum toxicity - d. Myasthenia gravis - e. Hypokalaemia

**CORRECT-CHOICE LINE**: E

**REASONING**: The patient describes episodes of periodic paralysis and the ECG characteristics are consistent with that of hypokalaemia. The underlying diagnosis is a rare familial condition of skeletal muscle ion channels called hypokalaemic periodic paralysis, which tends to develop in childhood and adolescence. Attacks last hours and the neurological examination is usually unremarkable in between attacks. The average potassium on diagnosis is 2.4 mmol/L1. Diagnosis is often made clinically in association with low potassium but genetic testing can help if known mutations are present.

**>>DESCRIPTION**: A 17-year-old girl presents with recurrent episodes of paralysis upon waking, lasting 2 hours with preserved consciousness, and a jerky baseline with flat T waves on ECG.

**>>OPTIONS**: a. Botulinum toxicity b. Guillain-Barre syndrome c. Hypokalaemia d. Myasthenia gravis e. Partial or absence seizures

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Hypokalaemia

**>>REASONING**: The most likely diagnosis is hypokalemia due to the presentation of periodic paralysis and the ECG findings. Other options are less likely because they don’t explain the paralysis and the ECG changes associated with hypokalemia.

## Question #:324

**CLINICAL SCENERIO**: A 17-year-old girl presents to the emergency department with recurrent episodes of waking paralysis lasting 2 hours, without loss of consciousness. She has no significant medical history, unremarkable examination findings, normal BMI, and a jerky baseline ECG with flat T waves.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - f. Partial or absence seizures - g. Guillain-Barre syndrome - h. Botulinum toxicity - i. Myasthenia gravis - j. Hypokalaemia

**CORRECT-CHOICE LINE**: E

**REASONING**: The patient describes episodes of periodic paralysis and the ECG characteristics are consistent with that of hypokalaemia. The underlying diagnosis is a rare familial condition of skeletal muscle ion channels called hypokalaemic periodic paralysis, which tends to develop in childhood and adolescence. Attacks last hours and the neurological examination is usually unremarkable in between attacks. The average potassium on diagnosis is 2.4 mmol/L1. Diagnosis is often made clinically in association with low potassium but genetic testing can help if known mutations are present.

1. Miller TM, Dias da Silva MR, Miller HA et al. Correlating phenotype and genotype in the periodic paralyses. Neurology. 2004;63(9):1647.

**>>DESCRIPTION**: A 17-year-old girl presents with recurrent episodes of waking paralysis lasting 2 hours, with no loss of consciousness, unremarkable examination findings, and a jerky baseline ECG with flat T waves.

**>>OPTIONS**: a. Botulinum toxicity b. Guillain-Barre syndrome c. Hypokalaemia d. Myasthenia gravis e. Partial or absence seizures

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Hypokalaemia

**>>REASONING**: The most likely diagnosis is hypokalemia due to the patient’s periodic paralysis episodes and the ECG findings. The other options are incorrect because they do not align with the clinical presentation and ECG findings of hypokalemic periodic paralysis.

## Question #:39

**CLINICAL SCENERIO**: A 68-year-old Indian patient with diffuse cutaneous systemic sclerosis and vitamin D deficiency presents with facial tetany, muscle cramps and paraesthesia. She receives regular vitamin D supplements. Blood tests show corrected calcium of 1.68 mmol/l, phosphate of 1.4 mmol/l, magnesium of 0.28 mmol/l, and PTH of 2 pmol/L.

**QUESTION LINE**: What is the underlying cause for these metabolic disturbances in this patient?

**OPTIONS**: a) Hypomagnesaemia b) Primary hypoparathyroidism c) Insufficient vitamin D supplementation d) Chronic kidney failure e) Chronic pancreatitis

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This complex picture investigates the underlying cause of hypomagnesaemia and hypocalcaemia in a patient with significant GI disease. With regular vitamin D supplementation, it is unlikely this is the cause. Her renal function is also within normal range. Although her parathyroid hormone levels are low, the likely underlying cause is due to insufficient magnesium absorption due to GI systemic sclerosis, which results in reduces parathyroid hormone release. There is nothing in the history to suggest a primary hypoparathyroidism or chronic pancreatitis.

**>>DESCRIPTION**: 68-year-old with systemic sclerosis and vitamin D deficiency presents with tetany, cramps, and paresthesia. Calcium is 1.68 mmol/l, phosphate 1.4 mmol/l, magnesium 0.28 mmol/l, and PTH 2 pmol/L.

**>>OPTIONS**: a) Chronic kidney failure b) Chronic pancreatitis c) Hypomagnesaemia d) Insufficient vitamin D supplementation e) Primary hypoparathyroidism

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Hypomagnesaemia

**>>REASONING**: Hypomagnesemia due to GI involvement from systemic sclerosis leads to reduced PTH release and hypocalcemia. Vitamin D supplementation makes deficiency unlikely. Renal function is normal. No evidence supports primary hypoparathyroidism or chronic pancreatitis.

## Question #:245

**CLINICAL SCENERIO**: A 45-year-old woman with chronic alcohol abuse, admitted 3 days ago for nausea and severe diarrhea, now reports peri-oral and finger tingling. She was admitted for hydration after a week of severe watery diarrhea and is receiving IV fluids but unable to eat due to nausea. Her blood pressure is 130/74 mmHg, pulse 68/min, respiratory rate 16/min, and she is afebrile. Physical exam reveals facial twitching on percussion and carpal spasm after cuff inflation.

**QUESTION LINE**: Which of the following is most likely to have caused these findings?

**OPTIONS**: - a) Hyperuricaemia - b) Hypernatraemia - c) Hypomagnesaemia - d) Hypophosphataemia - e) Hypouricaemia

**CORRECT-CHOICE LINE**: c

**REASONING**: Magnesium deficiency causes hypocalcaemia

This patient is displaying classic signs of hypocalcaemia, including hyperexcitability of her facial nerve (Chvostek’s sign), induced carpal spasm (Trousseau’s sign), and tingling of the extremities and lips. Calcium homeostasis is a complicated process involving PTH, vitamin D, albumin and numerous electrolytes. Acquired hypoparathyroidism is the most common form of true hypocalcaemia, most often occurring transiently after thyroid surgery or after the removal of a parathyroid adenoma. Occasionally, hypomagnesaemia can produce hypocalcaemia by decreasing both the body’s production of PTH and its sensitivity to the hormone. In this case, it is likely that the patient became magnesium depleted from her course of watery diarrhoea, likely baseline poor nutritional status and alcohol abuse.

Choice 1: Hyperuricaemia is not a cause of hypocalcaemia. Chronic kidney disease, however, can lead to hypocalcaemia in the setting of secondary hyperparathyroidism, but there is no evidence of renal failure in this patient.

Choice 2: Fluid balance (hyper- or hyponatraemia) does not play a role in calcium homeostasis.

Choice 4: Hypophosphataemia is not a cause of hypocalcaemia. Actually, hypocalcaemia often leads to hyperphosphataemia secondary to increased PTHmediated bone resorption. Elevations in phosphate may also contribute to hypocalcaemia by complexing with circulating calcium and suppressing conversion of 25-OH to 1, 25-OH vitamin D.

Choice 5: Urate levels do not affect calcium homeostasis

**>>DESCRIPTION**: A 45-year-old woman with chronic alcohol abuse and recent severe diarrhea presents with peri-oral tingling, finger tingling, facial twitching, and carpal spasm. She has been receiving IV hydration and is unable to eat due to nausea.

**>>OPTIONS**: a) Hypernatraemia b) Hyperuricaemia c) Hypomagnesaemia d) Hypophosphataemia e) Hypouricaemia

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Hypomagnesaemia

**>>REASONING**: Hypomagnesemia is the most likely cause due to its potential to cause hypocalcemia, indicated by the patient’s signs (Chvostek’s and Trousseau’s signs). Other options like hyperuricemia, hypernatremia, hypophosphatemia, and hypouricemia are not directly linked to hypocalcemia.

## Question #:11

**CLINICAL SCENERIO**: A 35-year-old man on the Acute Medical Unit, with a 10-year history of alcohol overuse, presents with a seizure during alcohol withdrawal after being admitted for detox with prescribed chlordiazepoxide, pabrinex and fluids. He has no other past medical history.

**QUESTION LINE**: Which electrolyte abnormality is most likely to have caused his seizure?

**OPTIONS**: a) Hypocalcaemia b) Hypoglycaemia c) Hypokalaemia d) Hypomagnesaemia e) Hypophosphataemia

**CORRECT-CHOICE LINE**: e

**REASONING**: Hypophosphataemia is the most common electrolyte abnormality during alcohol withdrawal and is a recognised cause of seizures. It is often present at baseline in alcoholic patients and can further decrease in withdrawal. It is thought that this is due to effects on the proximal renal tubule. It can cause muscle pain and disorientation at very low levels ( < 0.3 mmol/l). Hypoglycaemia would also be possible but in the absence of diabetes, and with the patient eating and drinking, this is less likely. Hypoglycaemia is common in patients intoxicated with alcohol and these patients should be monitored with regular capillary blood glucose testing. Low magnesium and low calcium both cause seizures but are unlikely to be lowered sufficiently in this patient to result in seizures in the absence of other clinical signs. Low potassium is unlikely to result in seizures.

**>>DESCRIPTION**: A 35-year-old man with a history of alcohol overuse presents with a seizure during alcohol withdrawal after being admitted for detox.

**>>OPTIONS**: a) Hypocalcaemia b) Hypoglycaemia c) Hypokalaemia d) Hypomagnesaemia e) Hypophosphataemia

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Hypophosphataemia

**>>REASONING**: Hypophosphatemia is the most likely cause of the seizure due to its association with alcohol withdrawal. Other electrolyte imbalances are less likely. Hypoglycemia is less likely given the patient’s recent oral intake.

## Question #:37

**CLINICAL SCENERIO**: A 18-year-old man with type 1 diabetes and poor compliance attends hospital with shortness of breath, vomiting and feeling unwell. He is found to be in diabetic ketoacidosis and transferred to the high dependency unit. He has recovered well and is asymptomatic for a period but on day 3 of his treatment starts to feel unwell. He is tired and lethargic. His arms and legs ache and there is cramping at times. He walks to the bathroom and his legs collapse beneath him.On examination, he is alert and has moist mucosa. There are no fasciculations or myoclonus. He has 4/5 power in all muscle groups with retained sensation. His abdomen is soft and his chest clear. A set of observations show that he is tachypnoeic at 24 breaths/min. Bloods show improvement in Na+, K+, Urea, Creatinine, Glucose, Ketones and pH.

**QUESTION LINE**: What is the likely course of his deterioration?

**OPTIONS**: - a) Anxiety - b) Hypophosphataemia - c) Cerebral pontine myelinolysis - d) Sepsis - e) Disequilibration syndrome

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Recovering DKA are at risk of hypophosphataemia

This gentleman has developed weakness following treatment for DKA. This is unlikely cerebral pontine myelinolysis as the sodium has been corrected by only 5mmol/l in 2-3 days. There are no focal signs suggesting sepsis though it is likely on the differential. Anxiety should only be made once other diagnoses are excluded. Disequilibration syndrome occurs in dialysis patients and is not relevant here. Therefore the answer is hypophosphataemia that often arises as a side effect of insulin with cells forming ATP and taking up free phosphate to achieve this.

**>>DESCRIPTION**: A 18-year-old man with type 1 diabetes and poor compliance presents with DKA, improving initially but deteriorating on day 3 with weakness and cramping. Examination shows alert patient with 4/5 power in all muscle groups. Bloods show improvement in DKA markers.

**>>OPTIONS**: a) Anxiety b) Cerebral pontine myelinolysis c) Disequilibration syndrome d) Hypophosphataemia e) Sepsis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Hypophosphataemia

**>>REASONING**: Hypophosphataemia is a risk in recovering DKA due to insulin causing cells to take up phosphate. Cerebral pontine myelinolysis is less likely due to the small sodium correction. Sepsis is possible but less likely without focal signs. Disequilibration syndrome is irrelevant in this context.

## Question #:161

**CLINICAL SCENERIO**: A 24-year-old woman with type 1 diabetes, poorly compliant with insulin, presents with dyspnoea. Observations: heart rate 90 bpm, respiratory rate 24/min, BP 120/77 mmHg, temperature 36.7ºC. Arterial blood gas shows metabolic acidosis and hyperglycemia. Treated with IV fluids and insulin infusion. What electrolyte abnormality risk during resolution?

**QUESTION LINE**: What electrolyte abnormality is she at risk of developing during the resolution of

## this condition?

**OPTIONS**: a) Hypercalcaemia b) Hyperkalaemia c) Hypernatraemia d) Hypermagnesemi e) Hypophosphataemia

**CORRECT-CHOICE LINE**: e

**REASONING**: Recovering DKA are at risk of hypophosphataemia

Hypophosphataemia is the correct answer. This patient has diabetic ketoacidosis (elevated blood glucose level, acidaemia and ketonemia). A rise in insulin due to exogenous insulin administration causes an intracellular shift of phosphate. There is an element of phosphaturia in addition.

Hypercalcaemia is incorrect. This has been noted to occur in the development of DKA, likely secondary to severe metabolic acidosis and insulin deficiency but would therefore be not expected to occur as DKA resolves.

Hyperkalaemia is incorrect. Most patients with DKA are potassium deficient and the risk is of hypokalaemia, not hyperkalaemia. Furthermore drives potassium into cells, resulting in hypokalaemia.

Hypermagnesemia is incorrect. Insulin administration causes uptake of magnesium into cells and may cause hypomagnesemia rather than hypermagnesemia.

Hyperphosphataemia is incorrect. It is low rather than high phosphate that is the risk in recovering DKA patients.

**>>DESCRIPTION**: A 24-year-old woman with type 1 diabetes and dyspnea, non-compliant with insulin, presents with metabolic acidosis and hyperglycemia. She’s treated with IV fluids and insulin. What electrolyte abnormality is she at risk of developing during resolution?

**>>OPTIONS**: a) Hypercalcaemia b) Hyperkalaemia c) Hypermagnesemi d) Hypernatraemia e) Hypophosphataemia

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Hypophosphataemia

**>>REASONING**: The correct answer is hypophosphatemia. Insulin treatment in resolving diabetic ketoacidosis (DKA) causes an intracellular shift of phosphate and phosphaturia, leading to hypophosphatemia. Hypercalcemia, hyperkalemia, hypermagnesemia, and hypernatremia are less likely or are not the primary electrolyte imbalances associated with DKA resolution.

## Question #:72

**CLINICAL SCENERIO**: A 40 year old man presents to the Emergency Department with tiredness and dizziness (worse on standing) which has been ongoing for the past few months. He had a past medical history of epilepsy and mentions that he has had ‘brain surgery’ in the past. He is on some medications but cannot remember the names. He has no allergies.

On assessment, he has no focal neurological deficit and cardiovascular/respiratory examination is normal. Observations show a blood pressure of 135/90 mmHg (dropping to 105/82 mmHg on standing), a heart rate of 67 beats per minute, a temperature of 36.2 degrees, oxygen saturations of 94% on air and a respiratory rate of 18/min. Given his medical history, you opt to keep this gentleman in the short stay unit for observation overnight.

Baseline blood tests are as follows:

| Hb | 125 g/l |
| --- | --- |
| WCC | 9.2 x109/l |
| Plt | 290 x109/l |
| CRP | 10 mg/l |
| Gluc | 3.9 mmol/l |
| Na+ | 138 mmol/l |
| K+ | 5.8 mmol/l |
| Ur | 7.2 mmol/l |
| Cr | 100 µmol/l |
| TSH | 0.4 mU/l |

T4

**QUESTION LINE**: Given the above, what is the most likely underlying diagnosis?

**OPTIONS**: - a) Hypopituitarism - b) Hypothyroidism - c) Acromegaly - d) Pheochromocytoma - e) Medication side effects

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This gentleman has hypopituitarism following ‘brain surgery’. Though the details of this are obscured in the question, it is likely that removal of a pituitary mass with trans-sphenoidal surgery. This is exhibited by fairly non-descript symptoms coupled with some underlying evidence of lack of anterior pituitary hormones: low BP/dizziness/postural hypotension, high/normal K+, low/normal Na+ and low/normal blood glucose all indicate lack of cortisol due to low ACTH; the low/normal temperature and heart rate and the tiredness steer you towards low thyroxine level due to lack of TSH.

This gentleman needs assessment of his pituitary function. This can be done in many ways. A baseline pituitary hormone profile can be quite useful; however the most definitive tests involve assessing dynamic pituitary function. The insulin stress test (coupled with TRH and GnRH tests) creates a hypoglycaemic effect in the body and the response of the pituitary (cortisol surge) is measured. However, inducing hypoglycaemia in epileptics, such as this gentleman, is contraindicated. Therefore the next best investigation is the glucagon stimulation test which mimics hypoglycaemia in the body and causes a fake stress on the pituitary, therefore being safe to use in epileptics.

**>>DESCRIPTION**: A 40-year-old man with a history of epilepsy and prior ‘brain surgery’ presents with tiredness and dizziness. Examination reveals postural hypotension. Labs show normal Na+, high/normal K+, and low glucose. TSH is 0.4 mU/l.

**>>OPTIONS**: a) Acromegaly b) Hypopituitarism c) Hypothyroidism d) Medication side effects e) Pheochromocytoma

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Hypopituitarism

**>>REASONING**: The patient’s history of brain surgery, postural hypotension, near normal electrolytes, and low TSH are suggestive of hypopituitarism. These findings indicate a deficiency in anterior pituitary hormones, particularly ACTH and TSH.

## Question #:202

**CLINICAL SCENERIO**: A 19-year-old with type 1 diabetes presents to the Emergency Department with vomiting, diarrhoea, and omitted insulin doses, presenting with a capillary glucose of 37 mmol/l and 4+ ketones on urinalysis. Arterial blood gas results: pH, pO2 13 kPa, pCO2 3.5 kPa, HCO3 13, Na 129 mmol/l, and K 6.1 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate initial management?

**OPTIONS**: - a) IV 0.9% NaCl bolus - b) IV 10 units actrapid + 50ml 50% dextrose - c) IV 8.4% sodium bicarbonate - d) Empirical IV antibiotics - e) Insulin sliding scale

**CORRECT-CHOICE LINE**: a

**REASONING**: This is a classical presentation of diabetic ketoacidosis. While precise protocols vary, the key principals are initial fluid resuscitation with normal saline prior to starting an IV insulin infusion, and careful potassium replacement.

Low sodium is often seen and is a pseudohyponatraemia secondary to the high serum glucose.

Serum potassium derangements are common and need careful management. Potassium is driven into cells by insulin. Serum potassium levels are therefore often high on presentation while blood insulin levels are depleted. Despite this, total body potassium is low due to fluid losses and requires careful monitoring and replacement during treatment.

**>>DESCRIPTION**: A 19-year-old with type 1 diabetes presents with vomiting, diarrhea, omitted insulin, hyperglycemia, ketonuria, and arterial blood gas findings indicative of diabetic ketoacidosis.

**>>OPTIONS**: a) Empirical IV antibiotics b) Insulin sliding scale c) IV 0.9% NaCl bolus d) IV 10 units actrapid + 50ml 50% dextrose e) IV 8.4% sodium bicarbonate

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: IV 0.9% NaCl bolus

**>>REASONING**: The most appropriate initial management is IV 0.9% NaCl bolus for fluid resuscitation. This is the first step in managing diabetic ketoacidosis (DKA). Insulin administration and potassium replacement follow fluid resuscitation. The patient’s acid-base status does not necessitate bicarbonate administration, and antibiotics are not indicated.

## Question #:190

**CLINICAL SCENERIO**: A 19-year-old woman with type 1 diabetes, coeliac disease (gluten-free diet, HbA1c 53 mmol/mol) presents with nausea, vomiting, dehydration, and postural hypotension. Investigations reveal hyponatremia, elevated creatinine and CRP, and a slightly elevated eosinophil count.

**QUESTION LINE**: Which of the following is the most important intervention with respect to her management?

**OPTIONS**: - a) Fluid restriction - b) IV anti-emetic - c) IV hydrocortisone - d) IV normal saline - e) NG feeding

**CORRECT-CHOICE LINE**: c

**REASONING**: Given the clinical presentation (nausea, vomiting, postural drop, tanning) and lab findings (hyponatremia, elevated eosinophils), the patient is likely experiencing Addison’s disease. Therefore, IV hydrocortisone is the most crucial intervention.

**>>DESCRIPTION**: A 19-year-old woman with type 1 diabetes and coeliac disease presents with nausea, vomiting, dehydration, postural hypotension, and relevant lab findings.

**>>OPTIONS**: a) Fluid restriction b) IV anti-emetic c) IV hydrocortisone d) IV normal saline e) NG feeding

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: IV hydrocortisone

**>>REASONING**: The patient likely has Addison’s disease. IV hydrocortisone is the most important intervention to address adrenal insufficiency. Other options are less critical to the underlying cause.

## Question #:306

**CLINICAL SCENERIO**: A 45-year-old woman is brought to the ER with a GCS of 11. The neighbor states the patient seemed low and wore more layers of clothes. Exam: cool to touch, bradycardic (HR 38), RR 8, sats 91% on 15L O2, temp 33ºC, BM 2.7. Blood tests show: Hb 130 g/L, Platelets 220 \* 10 9 /L, WBC 9 \* 10 9 /L, Calcium 2.5 mmol/L, TSH 25 mU/L, Free T4 0.4 pmol/L, Creatine kinase 6000 U/L, Na+ 130 mmol/L, K+ 4 mmol/L, Bicarbonate 22 mmol/L, Urea 8 mmol/L, Creatinine 130 µmol/L.

**QUESTION LINE**: What is the most appropriate management of this patient?

**OPTIONS**: - a. Intubation and urgent CT head - b. IV hydrocortisone, IV dextrose and rewarming - c. IV T4, IV fluids, IV dextrose and intubation - d. IV fluid resuscitation, IV dextrose, intubation and IV T4 - e. IV hydrocortisone, IV T4, IV dextrose and IV fluids answer is E

**CORRECT-CHOICE LINE**: e. IV hydrocortisone, IV T4, IV dextrose and IV fluids answer is E

**REASONING**: If myxedema coma is suspected, IV corticosteroids should be given alongside IV thyroid replacement until coexisting adrenal insufficiency has been excluded. The most appropriate answer is 5 as they need treatment of the underlying cause as well as correction of life-threatening abnormalities. This patient is suffering from severe hypothyroidism and is in a myxedema coma. This is best treated with IV hydrocortisone and thyroxine in case there is co-existing adrenal insufficiency. Their blood sugars are low necessitating correction and there is a raised creatine kinase requiring management with IV fluids. CT head and intubation is appropriate but is not the best option of those above. The other answers do no include thyroxine and hydrocortisone as a combined answer so, although an aspect of the overall management of the patient, are not the most pressing intervention.

**>>DESCRIPTION**: A 45-year-old woman presents with a GCS of 11, bradycardia, hypothermia, and a blood glucose of 2.7. Labs reveal elevated TSH, low free T4, and markedly elevated creatine kinase. What is the most appropriate management?

**>>OPTIONS**: a. Intubation and urgent CT head b. IV fluid resuscitation, IV dextrose, intubation and IV T4 c. IV hydrocortisone, IV dextrose and rewarming d. IV hydrocortisone, IV T4, IV dextrose and IV fluids e. IV T4, IV fluids, IV dextrose and intubation

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: IV hydrocortisone, IV T4, IV dextrose and IV fluids

**>>REASONING**: The correct answer is IV hydrocortisone, IV T4, IV dextrose, and IV fluids. This patient is in myxedema coma. Management includes addressing the underlying cause (hypothyroidism) and correcting life-threatening abnormalities (hypoglycemia, elevated CK). Other options are not the most comprehensive treatment.

## Question #:294

**CLINICAL SCENERIO**: A 42-year-old alcoholic presents with vomiting, muscle weakness, and palpitations. Despite calcium infusions, calcium remains low. Examination reveals hypotension, tachycardia, and SVT. Electrolytes are provided.

**QUESTION LINE**: Which of the following is the most appropriate next step?

**OPTIONS**: a. IV calcium b. IV magnesium c. IV potassium d. IV phosphate e. IV glucose

**CORRECT-CHOICE LINE**: B

**REASONING**: Magnesium deficiency causes hypocalcaemia

Long term alcoholism as well as leading to falls in serum calcium, can also lead to significant falls in magnesium, which can account both for the persistently decreased calcium despite replacement, and for the runs of SVT seen here. The most appropriate next step is magnesium replacement, which is likely to facilitate both an improvement in calcium and resolution of SVT.

Further IV calcium will be ineffective without first replacing magnesium, and potassium is within the normal range, as is glucose. Phosphate replacement may be required in the treatment of alcoholism, but this is usually in the context of refeeding syndrome.

**>>DESCRIPTION**: A 42-year-old alcoholic with vomiting, muscle weakness, palpitations, hypocalcemia, hypotension, tachycardia, and SVT. Electrolyte levels are provided.

**>>OPTIONS**: a. IV calcium b. IV glucose c. IV magnesium d. IV phosphate e. IV potassium

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: IV magnesium

**>>REASONING**: Magnesium deficiency, common in alcoholics, likely underlies the persistent hypocalcemia and SVT. Magnesium replacement is the most appropriate next step to improve calcium levels and resolve SVT. Other options are less likely to address the underlying cause.

## Question #:44

**CLINICAL SCENERIO**: A 51-year-old lady librarian attends outpatient clinic with painful eyes. She reports that her vision has deteriorated over the past four weeks. On examination, she has proptosis, periorbital oedema and a painful complex ophthalmoplegia. She appears anxious and is worried about not coping at work. At present she smokes ten cigarettes daily.

**QUESTION LINE**: What would be the most appropriate next step in managing this patient?

**OPTIONS**: - a) IV methylprednisolone - b) Surgical decompression - c) Smoking cessation advice - d) Total thyroidectomy - e) Artificial tear drops

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: IV methylprednisolone is the treatment of choice for moderately severe active Graves’ ophthalmopathy. IV steroids have fewer side effects than oral steroids. If symptoms or vision do not improve then urgent surgical decompression should be considered.

Artificial tear drops are useful for symptomatic relief.

Total thyroidectomy has shown no benefit in the treatment of thyroid eye disease.

Outcomes have been shown to be worse in those patients who smoke, therefore smoking cessation advice should be given.

**>>DESCRIPTION**: A 51-year-old woman with painful eyes and deteriorating vision over four weeks presents with proptosis, periorbital oedema, and ophthalmoplegia. She is anxious and smokes ten cigarettes daily.

**>>OPTIONS**: a) Artificial tear drops b) IV methylprednisolone c) Smoking cessation advice d) Surgical decompression e) Total thyroidectomy

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: IV methylprednisolone

**>>REASONING**: IV methylprednisolone is the treatment of choice for moderately severe active Graves’ ophthalmopathy. Surgical decompression is considered if symptoms don’t improve. Total thyroidectomy has no benefit, and while smoking cessation is important, it is not the most immediate next step.

## Question #:59

**CLINICAL SCENERIO**: A 35-year-old man with known type 1 diabetes presents with a 24-hour history of nausea, vomiting, and increasing lethargy. He reports non-adherence to insulin. Examination reveals dehydration, Kussmaul breathing, and a weight of 63 kg. Blood pressure is 70/45 mmHg, pulse is 116 bpm, and respiratory rate is 26/min. Laboratory investigations show elevated WBC and random blood glucose, acidosis, low pCO2 and bicarbonate, normal electrolytes, and blood ketones of 3 mmol/L.

**QUESTION LINE**: What is the most appropriate management plan for this patient?

**OPTIONS**: a) IV normal saline, 5.0 units/h insulin infusion and 40 mmol/L potassium b) IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium c) IV normal saline, 63 units/h insulin infusion and 40 mmol/L potassium d) IV normal saline, IV antibiotics, 5.0 units/h insulin infusion and 40 mmol/L potassium e) IV normal saline, IV antibiotics, 6.3 units/h insulin infusion and 40 mmol/L potassium

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Diabetic ketoacidosis: the IV insulin infusion should be started at 0.1 unit/kg/hour

IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium is the correct answer. The clinical presentation is highly suggestive of diabetic ketoacidosis (DKA), characterised by metabolic acidosis, hyperglycaemia, and ketonaemia. Management involves intravenous normal saline for rehydration, insulin therapy at a rate of 6.3 units/h (based on the patient’s body weight of 63 kg), and potassium replacement at 40 mmol/L because the patient’s serum potassium is within the range of 3.5-5.5 mmol/L.

IV normal saline, 5.0 units/h insulin infusion, and 40 mmol/L potassium are incorrect because the appropriate insulin infusion rate should be calculated as 0.1 unit/kg/hour; for a patient weighing 63 kg, this equates to an infusion rate of 6.3 units/hour.

## IV normal saline, 63 units/h insulin infusion, and 40 mmol/L

potassium represents a significant overdose of insulin. The correct dosage should be based on the standard DKA management protocol of administering insulin at a dose of 0.1 unit/kg/hour, resulting in an infusion rate of 6.3 units/hour for this patient.

IV normal saline, IV antibiotics, 5.0 units/h insulin infusion and 40 mmol/L potassium is not the appropriate choice here as it underestimates the required rate of insulin infusion (should be set at 6.3 units/h instead). Moreover, leukocytosis can occur associated with hyperglycaemia and DKA; thus, it should not automatically prompt antibiotic administration without further evidence suggestive of infection.

IV normal saline, IV antibiotics, 6.3 units/h insulin infusion and 40 mmol/L potassium is incorrect due to the unnecessary inclusion of antibiotics in the treatment regimen. As previously stated, leukocytosis can be associated with both hyperglycaemia and DKA; therefore, it does not justify empirical antibiotic therapy without other clinical indications pointing towards an infectious process.

**>>DESCRIPTION**: A 35-year-old man with type 1 diabetes presents with nausea, vomiting, and lethargy after insulin non-adherence. He is dehydrated, with Kussmaul breathing, and weighs 63 kg. BP is 70/45 mmHg, pulse 116 bpm, RR 26/min. Labs show elevated WBC and glucose, acidosis, low pCO2 and bicarbonate, normal electrolytes, and blood ketones of 3 mmol/L.

**>>OPTIONS**: a) IV normal saline, 5.0 units/h insulin infusion and 40 mmol/L potassium b) IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium c) IV normal saline, 63 units/h insulin infusion and 40 mmol/L potassium d) IV normal saline, IV antibiotics, 5.0 units/h insulin infusion and 40 mmol/L potassium e) IV normal saline, IV antibiotics, 6.3 units/h insulin infusion and 40 mmol/L potassium

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium

**>>REASONING**: The patient presents with DKA, requiring IV fluids, insulin, and potassium. The correct insulin infusion rate is 0.1 unit/kg/hour, which is 6.3 units/hour for this patient. Using 5.0 units/h is too low, and 63 units/h is an overdose. Antibiotics are not indicated without signs of infection.

## Question #:150

**CLINICAL SCENERIO**: A 35-year-old patient presents to the emergency department after being brought in by ambulance. She had recently moved locally but had not yet registered with a GP, and therefore had an interruption in her supply of carbimazole for hyperthyroidism, diagnosed three months earlier. She is currently very unwell, complaining of palpitations. Clinically she has a raised JVP, bilateral crepitations on auscultation and severe peripheral oedema, and has become very breathless, and a temperature of 40.1 degrees C. She is started on oxygen. An ECG demonstrates a heart rate of 170bpm in AF, whilst a chest X-ray shows pulmonary oedema.

**QUESTION LINE**: What is the most appropriate immediate management?

**OPTIONS**: a) IV propranolol b) Propylthiouracil c) Aspirin d) Refer for emergency thyroid surgery e) Potassium iodide

**CORRECT-CHOICE LINE**: a

**REASONING**: In thyroid storm with IV beta-blockers are a important first-line treatment

This is a patient with likely thyroid storm. The presentation suggests abrupt withdrawal of carbimazole has been the provoking factor, and now she has developed AF and heart failure. The most appropriate action is to rapidly control the heart rate by the use of IV beta-blockers. Propylthiouracil should also be started urgently, but the more critical action is to manage her heart rate. Aspirin may worsen the situation by displacing T4 from thyroid-binding globulin, causing an increase in free T4. Whilst the patient will likely need both potassium iodide and thyroid surgery, neither are as urgent as controlling the heart rate.

**>>DESCRIPTION**: A 35-year-old patient presents with palpitations, raised JVP, bilateral crepitations, severe peripheral edema, breathlessness, and a temperature of 40.1°C after carbimazole interruption for hyperthyroidism. ECG shows AF with a heart rate of 170 bpm, and chest X-ray reveals pulmonary edema.

**>>OPTIONS**: a) Aspirin b) IV propranolol c) Potassium iodide d) Propylthiouracil e) Refer for emergency thyroid surgery

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: IV propranolol

**>>REASONING**: IV propranolol is the most appropriate immediate management to rapidly control the heart rate in a likely thyroid storm. Propylthiouracil should also be started urgently. Aspirin is incorrect because it may worsen the condition. Potassium iodide and thyroid surgery are not as urgent as controlling the heart rate.

## Question #:141

**CLINICAL SCENERIO**: A 55-year-old male presents to an endocrine clinic for gynaecomastia investigation. Examination reveals bilateral breast tissue growth. His medical history includes hypertension, hypothyroidism, and congestive cardiac failure. He consumes 30 units of alcohol weekly and takes levothyroxine, amlodipine, bisoprolol, lisinopril, and spironolactone.

**QUESTION LINE**: What is the most likely explanation of his gynaecomastia?

**OPTIONS**: a) Cirrhosis b) Hypopituitarism c) Iatrogenic d) Klinefelter’s syndrome e) Idiopathic

**CORRECT-CHOICE LINE**: c

**REASONING**: The most likely explanation for this gentlemans gynaecomastia given the negative examination findings is a side effect of his current regular medications. There are many drugs that can cause gynaecomastia; in this cause the culprit could be either amlodipine or spironolactone.

**>>DESCRIPTION**: A 55-year-old male presents with gynaecomastia. He has hypertension, hypothyroidism, congestive cardiac failure, and consumes 30 units of alcohol weekly. He takes levothyroxine, amlodipine, bisoprolol, lisinopril, and spironolactone.

**>>OPTIONS**: a) Cirrhosis b) Hypopituitarism c) Iatrogenic d) Klinefelter’s syndrome e) Idiopathic

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Iatrogenic

**>>REASONING**: The most likely cause is iatrogenic, stemming from medications like amlodipine or spironolactone. Other options are less likely given the clinical context.

## Question #:282

**CLINICAL SCENERIO**: A 29-year-old pregnant woman (34 weeks) presents with heat intolerance, diarrhea, and anxiety. She has a history of hyperthyroidism treated with carbimazole, and her mother had hyperthyroidism. Examination reveals a pulse of 98 bpm, BP of 124/82 mmHg, and a temperature of 37.5ºC. Blood tests show a suppressed TSH and elevated T4 levels.

**QUESTION LINE**: What is the most appropriate management?

**OPTIONS**: - a) Refer patient for immediate caesarean section - b) Increase carbimazole dose to 20mg once daily - c) Commence radioiodine treatment - d) Switch carbimazole to propylthiouracil - e) Refer for a thyroidectomy

**CORRECT-CHOICE LINE**: B

**REASONING**: The carbimazole can be increased to up to 20mg once daily during pregnancy. Propylthiouracil can be started instead of the carbimazole if the increased carbimazole dose does not adequately control the patients hyperthyroidism.

**>>DESCRIPTION**: A 29-year-old pregnant woman (34 weeks) with hyperthyroidism (on carbimazole) presents with symptoms of hyperthyroidism and abnormal thyroid function tests.

**>>OPTIONS**: a) Commence radioiodine treatment b) Increase carbimazole dose to 20mg once daily c) Refer for a thyroidectomy d) Refer patient for immediate caesarean section e) Switch carbimazole to propylthiouracil

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Increase carbimazole dose to 20mg once daily

**>>REASONING**: The most appropriate management is to increase the carbimazole dose to 20mg once daily. Other options are not appropriate for initial management during pregnancy. Radioiodine is contraindicated in pregnancy, and thyroidectomy is reserved for severe cases unresponsive to medication. Switching to propylthiouracil is an alternative, but increasing the dose of carbimazole is a reasonable first step.

## Question #:151

**CLINICAL SCENERIO**: A 34-year-old woman, 12 weeks pregnant, with Hashimoto’s thyroiditis, is taking levothyroxine 100 mcg. Her TSH is 1.0 mU/l.

**QUESTION LINE**: What is the most appropriate management with regards to her levothyroxine treatment, given her recent diagnosis of pregnancy?

**OPTIONS**: a) Reduce her levothyroxine dose by an average of 25 mcg b) Increase her levothyroxine dose by an average of 100 mcg c) Increase her levothyroxine dose by an average of 25-50 mcg d) Reduce her levothyroxine dose by an average of 50 mcg e) Keep her levothyroxine dose unchanged

**CORRECT-CHOICE LINE**: c

**REASONING**: In patients currently on levothyroxine who become pregnant, thyroid function tests should be assessed at 6-8 weeks gestation, 16-20 and at 28-32 weeks. During pregnancy, the average thyroxine requirements typically increase by 25-50 mcg. The patient normally returns to their original dose of levothyroxine straight after delivery.

**>>DESCRIPTION**: A 34-year-old pregnant woman (12 weeks) with Hashimoto’s thyroiditis, on levothyroxine 100 mcg (TSH 1.0 mU/l).

**>>OPTIONS**: a) Increase her levothyroxine dose by an average of 25-50 mcg b) Increase her levothyroxine dose by an average of 100 mcg c) Keep her levothyroxine dose unchanged d) Reduce her levothyroxine dose by an average of 25 mcg e) Reduce her levothyroxine dose by an average of 50 mcg

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Increase her levothyroxine dose by an average of 25-50 mcg

**>>REASONING**: Levothyroxine dose should be increased by 25-50 mcg during pregnancy. Incorrect options include reducing or maintaining the dose as thyroid hormone requirements increase during pregnancy.

## Question #:215

**CLINICAL SCENERIO**: A 28-year-old woman with hypothyroidism on levothyroxine (100 mcg daily) is 6 weeks pregnant. Her TSH is 5.0 mU/L and free T4 is 10.0 pmol/L. What is the appropriate levothyroxine dose adjustment?

**QUESTION LINE**: What would you recommend regarding her levothyroxine dose?

**OPTIONS**: a) Continue on current levothyroxine dose, no further monitoring required during pregnancy - b) Increase levothyroxine dose to 150 micrograms daily - c) Increase levothyroxine dose to 200 micrograms once daily immediately - d) Reduce levothyroxine dose to 75 micrograms once daily - e) Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose

**CORRECT-CHOICE LINE**: b

**REASONING**: Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

During pregnancy, the thyroid gland increases in size by 10% and the production of thyroid hormones, T3 and T4, increases by 50%.

If a patient has pre-existing hypothyroidism, we usually need to increase the dose of levothyroxine to mimic these changes.

Increase levothyroxine dose to 150 micrograms daily : an increase of levothyroxine supplementation of up to 50% mimics the physiological thyroid response to pregnancy and is the correct option for this patient.

Continue on current levothyroxine dose, no further monitoring required during pregnancy : this is incorrect. The requirement for T3 and T4 increases during pregnancy, therefore the levothyroxine dose must be increased.

Increase levothyroxine dose to 200 micrograms once daily immediately : this increase is too high, the patient does not require such an aggressive dosage adjustment.

Reduce levothyroxine dose to 75 micrograms once daily : incorrect, levothyroxine dose should not routinely be reduced during pregnancy, the requirement generally increases.

Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose : this would be inappropriate, thyroid abnormalities should be treated as soon as possible to minimise the risk to the foetus. Ideally, the patient should be counselled before conception. Maternal hypothyroidism is associated with low birth weight, premature labour, and neonatal respiratory problems.

**>>DESCRIPTION**: A 28-year-old woman with hypothyroidism on 100 mcg levothyroxine is 6 weeks pregnant, with TSH of 5.0 mU/L and free T4 of 10.0 pmol/L. What is the appropriate levothyroxine dose adjustment?

**>>OPTIONS**: a) Continue on current levothyroxine dose, no further monitoring required during pregnancy b) Increase levothyroxine dose to 150 micrograms daily c) Increase levothyroxine dose to 200 micrograms once daily immediately d) Reduce levothyroxine dose to 75 micrograms once daily e) Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Increase levothyroxine dose to 150 micrograms daily

**>>REASONING**: The correct answer is to increase the levothyroxine dose to 150 mcg daily, as the requirement for thyroid hormone increases during pregnancy. The other options are incorrect because they either maintain the current dose (insufficient), increase the dose too much, or reduce the dose (inappropriate). Repeating thyroid function tests later would delay necessary treatment.

## Question #:279

**CLINICAL SCENERIO**: A 29-year-old pregnant woman with hypothyroidism on levothyroxine 75mcg od and folic acid 400mcg od presents to the Endocrinology clinic. What is the next step?

**QUESTION LINE**: You request a repeat TSH and free T4 measurement. What is the most appropriate next step?

**OPTIONS**: - a. Decrease levothyroxine to 50mcg od - b. Keep levothyroxine at 75mcg od - c. Increase levothyroxine to 100mcg od - d. Keep levothyroxine at 75mcg od + increase folic acid to 5mg od - e. Stop levothyroxine until TSH known

**CORRECT-CHOICE LINE**: C

**REASONING**: Pregnant with hypothyroidism → immediately increase levothyroxine and monitor TSH closely

The correct answer is to increase levothyroxine to 100mcg od . In pregnant women with hypothyroidism, it is essential to maintain adequate thyroid hormone levels for the developing fetus. The requirement for levothyroxine often increases during pregnancy due to increased thyroxine-binding globulin (TBG) and placental degradation of maternal T4. According to UK guidelines, pregnant women with pre-existing hypothyroidism should have their levothyroxine dose increased by approximately 30% as soon as pregnancy is confirmed, even before obtaining repeat TSH and free T4 measurements. This ensures that both mother and baby have sufficient thyroid hormone levels during the critical early stages of fetal development.

The option to decrease levothyroxine to 50mcg od would not be appropriate, as reducing the dose could lead to inadequate thyroid hormone levels in the mother and potentially harm fetal development.

Keeping the levothyroxine at 75mcg od might seem reasonable if her TSH was well-controlled prior to pregnancy; however, this does not account for the increased requirements during pregnancy. It would be safer and more appropriate to increase the dose proactively.

As for keeping levothyroxine at 75mcg od + increasing folic acid to 5mg od , while it is important for pregnant women to take folic acid supplements, increasing the dose of folic acid alone without adjusting the levothyroxine dosage does not address her increased thyroid hormone needs during pregnancy.

Finally, choosing to stop levothyroxine until TSH is known would put both mother and fetus at risk of complications related to untreated or inadequately treated hypothyroidism. It is crucial that patients with pre-existing hypothyroidism continue their levothyroxine therapy during pregnancy, with appropriate dose adjustments as needed.

**>>DESCRIPTION**: A pregnant 29-year-old woman with hypothyroidism on levothyroxine 75mcg od presents to the clinic. What is the most appropriate next step?

**>>OPTIONS**: a. Decrease levothyroxine to 50mcg od b. Keep levothyroxine at 75mcg od c. Keep levothyroxine at 75mcg od + increase folic acid to 5mg od d. Increase levothyroxine to 100mcg od e. Stop levothyroxine until TSH known

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Increase levothyroxine to 100mcg od

**>>REASONING**: Increase levothyroxine to 100mcg od due to increased requirements during pregnancy. Decreasing or maintaining the current dose is inappropriate. Increasing folic acid alone is insufficient. Stopping levothyroxine is dangerous.

## Question #:193

**CLINICAL SCENERIO**: A 22-year-old student presents to the ED with a cough, rusty sputum, increased shortness of breath, night sweats, and fevers. She takes hydrocortisone for congenital adrenal hyperplasia and the oral contraceptive pill. Bloodwork is provided, including Hb, platelets, WBC, neutrophils, lymphocytes, eosinophils, Na+, K+, urea, creatinine, and CRP.

**QUESTION LINE**: Which of the following is the most appropriate way to manage her steroid hormone replacement?

**OPTIONS**: a) Convert to 200mg hydrocortisone IV BD b) Increase the daily dose by 50% c) Increase the daily dose by 100% d) Reduce the daily dose by 50% e) Keep the daily dose the same

**CORRECT-CHOICE LINE**: c.

**REASONING**: Patients with congenital adrenal hyperplasia on steroid replacement should have their corticosteroid dose doubled during acute infections like pneumonia. Switching to IV hydrocortisone is excessive. Small increases, reductions, or maintaining the current dose risk adrenal crisis.

**>>DESCRIPTION**: A 22-year-old student with congenital adrenal hyperplasia, taking hydrocortisone, presents with pneumonia. Which is the best way to manage her steroid replacement?

**>>OPTIONS**: a) Convert to 200mg hydrocortisone IV BD b) Increase the daily dose by 50% c) Increase the daily dose by 100% d) Keep the daily dose the same e) Reduce the daily dose by 50%

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Increase the daily dose by 100%

**>>REASONING**: The steroid dose should be doubled during acute infections (like pneumonia) in patients with congenital adrenal hyperplasia on steroid replacement to avoid adrenal crisis.

## Question #:104

**CLINICAL SCENERIO**: A 24-year-old nurse collapses on a night shift with a blood glucose of 1.4 mmol/l and blood pressure of 115/82 mmHg. He denies palpitations, tongue biting, or incontinence. He reports five similar episodes in the last two weeks. Blood tests show low-normal C-peptide and markedly raised insulin.

**QUESTION LINE**: Which of the following is the most likely diagnosis of his multiple episodes of collapse?

**OPTIONS**: - a) Sulphonylurea misuse - b) Insulin misuse - c) Alcohol misuse - d) Retroperitoneal sarcoma - e) Insulinoma

**CORRECT-CHOICE LINE**: Correct answedr is b.

**REASONING**: Hyperinsulinaemia in the absence of raised C-peptide points towards the diagnosis of insulin abuse. Elevation of C-peptide, when combined with hyperinsulinaemia suggests sulphonylurea abuse. To rule this out it may be appropriate to assay levels of commonly used sulphonylureas in urine. Insulinomas are a more rare cause of repeated hypoglycaemic episodes.

**>>DESCRIPTION**: A 24-year-old nurse collapses with hypoglycemia (1.4 mmol/l). He has had five similar episodes in two weeks. Labs show low-normal C-peptide and markedly elevated insulin.

**>>OPTIONS**: a) Alcohol misuse b) Insulin misuse c) Insulinoma d) Retroperitoneal sarcoma e) Sulphonylurea misuse

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Insulin misuse

**>>REASONING**: Elevated insulin with low C-peptide indicates insulin abuse. Elevated C-peptide would suggest sulfonylurea abuse. Insulinoma is a less likely cause of recurrent hypoglycemia.

## Question #:329

**CLINICAL SCENERIO**: A 24-year-old nurse collapses on a night shift with a blood glucose of 1.4 mmol/l. His blood pressure was 115/82 mmHg. He reports five similar episodes in two weeks. Blood tests show a low-normal C-peptide and a markedly raised insulin level.

**QUESTION LINE**: Which of the following is the most likely diagnosis of his multiple episodes of collapse?

**OPTIONS**: f) Sulphonylurea misuse g) Insulin misuse h) Alcohol misuse i) Retroperitoneal sarcoma j) Insulinoma

**CORRECT-CHOICE LINE**: Correct answedr is b.

**REASONING**: Hyperinsulinaemia in the absence of raised C-peptide points towards the diagnosis of insulin abuse. Elevation of C-peptide, when combined with hyperinsulinaemia suggests sulphonylurea abuse. To rule this out it may be appropriate to assay levels of commonly used sulphonylureas in urine. Insulinomas are a more rare cause of repeated hypoglycaemic episodes.

**>>DESCRIPTION**: A 24-year-old nurse presents with recurrent collapse and hypoglycemia. Blood tests reveal a low-normal C-peptide and elevated insulin.

**>>OPTIONS**: a) Alcohol misuse b) Insulin misuse c) Insulinoma d) Retroperitoneal sarcoma e) Sulphonylurea misuse

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Insulin misuse

**>>REASONING**: Insulin misuse is the most likely diagnosis due to hyperinsulinemia and a normal C-peptide level. Sulphonylurea abuse would present with elevated C-peptide. Insulinomas are a less common cause.

## Question #:258

**CLINICAL SCENERIO**: A 35-year-old woman with weight gain, irregular periods, and type 2 diabetes presents with violaceous striae and proximal muscle weakness. She consumes 15 units of alcohol daily. Blood tests reveal hypokalemia. The team suspects alcohol use might be the cause of her presentation and wants to differentiate it from an endogenous cause.

**QUESTION LINE**: What is the most appropriate investigation?

**OPTIONS**: - a. High-dose dexamethasone suppression test - b. Insulin stress test - c. Low-dose dexamethasone suppression test - d. Petrosal sinus sampling - e. Short synacthen test

**CORRECT-CHOICE LINE**: b.

**REASONING**: This patient may have pseudo-Cushing’s syndrome due to alcohol use. The insulin stress test is used to differentiate between true Cushing’s and pseudo-Cushing’s. The high-dose dexamethasone suppression test differentiates pituitary from ectopic sources. The low-dose dexamethasone suppression test is a first-line test for Cushing’s but doesn’t distinguish between Cushing’s and pseudo-Cushing’s. Petrosal sinus sampling is for ACTH-dependent Cushing’s without a clear pituitary lesion. The short synacthen test is for Addison’s disease.

**>>DESCRIPTION**: A 35-year-old woman with weight gain, irregular periods, type 2 diabetes, violaceous striae, proximal muscle weakness, and alcohol use, presents with hypokalemia. The team suspects alcohol as a possible cause of the presentation and wants to differentiate it from an endogenous cause.

**>>OPTIONS**: a. High-dose dexamethasone suppression test b. Insulin stress test c. Low-dose dexamethasone suppression test d. Petrosal sinus sampling e. Short synacthen test

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Insulin stress test

**>>REASONING**: The insulin stress test is the most appropriate investigation to differentiate between true Cushing’s syndrome and pseudo-Cushing’s syndrome, which is suspected in this patient due to alcohol use. Other tests are used for different purposes, such as differentiating the sources of Cushing’s syndrome or diagnosing Addison’s disease.

## Question #:315

**CLINICAL SCENERIO**: A 35-year-old woman presents with weight gain, particularly on her face and abdomen, and irregular periods. She has type 2 diabetes and drinks 15 units of alcohol daily. Examination reveals violaceous striae and proximal muscle weakness. Blood tests show low potassium. The team suspects alcohol use as the underlying cause and wants to differentiate it from an endogenous cause.

**QUESTION LINE**: What is the most appropriate investigation?

**OPTIONS**: 1. High-dose dexamethasone suppression test 2. Insulin stress test 3. Low-dose dexamethasone suppression test 4. Petrosal sinus sampling 5. Short synacthen test

**CORRECT-CHOICE LINE**: b

**REASONING**: The insulin tolerance test can be used to distinguish Cushing’s syndrome from pseudo-Cushing’s. This patient may have pseudo-Cushing’s syndrome due to alcohol use, presenting with similar features to Cushing’s syndrome. The insulin stress test helps differentiate between true Cushing’s and pseudo-Cushing’s. The other tests are not appropriate for distinguishing between Cushing’s syndrome and pseudo-Cushing’s.

**>>DESCRIPTION**: A 35-year-old woman presents with weight gain, menstrual irregularities, type 2 diabetes, alcohol use, violaceous striae, proximal muscle weakness, and hypokalemia. The team suspects alcohol-induced pseudo-Cushing’s syndrome and wants to investigate.

**>>OPTIONS**: 1. High-dose dexamethasone suppression test 2. Insulin stress test 3. Low-dose dexamethasone suppression test 4. Petrosal sinus sampling 5. Short synacthen test

**>>CORRECT-CHOICE LINE**: 2

**>>CORRECT-CHOICE\_TEXT**: Insulin stress test

**>>REASONING**: The insulin stress test is the most appropriate investigation to differentiate between Cushing’s syndrome and pseudo-Cushing’s syndrome. Other tests are used for different purposes, such as differentiating the source of Cushing’s or diagnosing Addison’s disease.

## Question #:195

**CLINICAL SCENERIO**: A 55-year-old male with a 4-month history of sweating, fatigue, and daytime tiredness, attributed to fluid retention, presented with worsening headaches and vision deterioration. Diagnosed with acromegaly, he underwent surgery a month ago and is feeling well.

**QUESTION LINE**: Which of the following investigations would be most useful for monitoring the effect of his therapy?

**OPTIONS**: - a) MRI pituitary - b) Echocardiography - c) Growth hormone levels - d) Insulin-like growth factor levels - e) Oral glucose tolerance test

**CORRECT-CHOICE LINE**: d

**REASONING**: Insulin-like growth factors (IGF-1) have a long half-life and so is a useful measurement to assess growth hormone secretion and therefore screen for acromegaly and monitor the response to therapy. Serum IGF-1 is the most feasible parameter to assess clinical disease activity, in everyday practice in the outpatient clinic setting. It is monitored every 6 months, and growth hormone (GH) levels are done yearly.

Note: Oral glucose tolerance test (plus GH levels) is not helpful for patients receiving somatostatin analogues, and for patients receiving GH receptor antagonist therapy, only IGF-1 should be measured.

**>>DESCRIPTION**: A 55-year-old male, post-surgery for acromegaly, requires monitoring of therapy effectiveness.

**>>OPTIONS**: a) Echocardiography b) Growth hormone levels c) Insulin-like growth factor levels d) MRI pituitary e) Oral glucose tolerance test

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Insulin-like growth factor levels

**>>REASONING**: IGF-1 levels are the most useful for monitoring therapy response due to their long half-life. Other options are less suitable or not as practical in routine clinical settings.

## Question #:142

**CLINICAL SCENERIO**: A 43-year-old woman with a 1-year history of recurrent lethargy, shakiness, slurred speech, and diplopia, with symptoms developing in the mornings and resolving after breakfast. She has type 2 diabetes mellitus, alcohol excess, and a functional neurological disorder. During review, she becomes shaky, with a capillary blood glucose of 2.4 and blood tests as follows: Insulin 43 pmol/L (<25), C-peptide 114 pmol/L (<75), Pro-insulin 23 pmol/L (3.6-22).

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Alcohol-related hypoglycaemia b) Exogenous insulin administration c) Glimepiride use d) Insulinoma e) Non-insulinoma pancreatogenous hypoglycaemia syndrome

**CORRECT-CHOICE LINE**: d

**REASONING**: The most likely diagnosis is insulinoma, suggested by hypoglycaemia symptoms improving after eating and elevated insulin, pro-insulin, and C-peptide levels. Exogenous insulin administration is incorrect as C-peptide would not be elevated. Glimepiride use is less likely due to the degree of insulin elevation. Alcohol-related hypoglycaemia would show low insulin and C-peptide. Non-insulinoma pancreatogenous hypoglycaemia syndrome is less likely as episodes commonly occur after meals.

**>>DESCRIPTION**: A 43-year-old woman presents with recurrent lethargy, shakiness, slurred speech, and diplopia, with morning onset and post-breakfast resolution, along with type 2 diabetes mellitus, alcohol excess, and functional neurological disorder. She becomes shaky during review with a blood glucose of 2.4 and elevated insulin, C-peptide, and Pro-insulin levels.

**>>OPTIONS**: a) Alcohol-related hypoglycaemia b) Exogenous insulin administration c) Glimepiride use d) Insulinoma e) Non-insulinoma pancreatogenous hypoglycaemia syndrome

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Insulinoma

**>>REASONING**: Insulinoma is the most likely diagnosis due to symptoms improving after eating and elevated insulin, pro-insulin, and C-peptide levels. Other options are incorrect because of various reasons: exogenous insulin administration would show no C-peptide, Glimepiride use would not generally increase insulin levels to this degree, alcohol-related hypoglycemia would show low insulin and C-peptide and Non-insulinoma pancreatogenous hypoglycaemia syndrome has post-prandial episodes.

## Question #:90

**CLINICAL SCENERIO**: A 66-year-old male was admitted with agitation and confusion, worsening over the past 1 week. His past medical history includes hypertension, ischaemic heart disease and chronic back pain. His daughter noticed that he had lost about 1 stone in weight (currently weighs 71 kg), has been more tired over the last month and that he has been drinking a lot more water. This was associated with the development of urinary incontinence.

On examination, his heart rate was 108 beats/min, blood pressure was 95/42 mmHg, saturations were 94% on air and respiratory rate was 20/min. He is confused, with a Glasgow Coma Scale of 14 and appeared dehydrated.

Blood results are as follows:

Na +

125 mmol/l

K +

5.0 mmol/l

Urea

18 mmol/l

Creatinine

180 µmol/l

Blood glucose

34 mmol/l

## Venous blood gas was done and showed the following:

| pH | 7.32 |
| --- | --- |
| pCO2 | 4.6 kPa |
| pO2 | 6.1 kPa |
| HCO3 | 17mmol/l |
| BE | -3.6 mmol/l |

**QUESTION LINE**: Which is the most important treatment?

**OPTIONS**: - a) Intravenous 0.9% sodium chloride - b) 10 units of human actrapid stat - c) Start insulin sliding scale at 6 units/hr - d) Calcium gluconate - e) Intravenous 1.8% sodium chloride

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Characteristic features of hyperosmolar hyperglycaemic state (HHS) includes:

*  high osmolality, often 320 mosmol/kg or more
*  high blood glucose, usually 30 mmol/L or more
*  severely dehydrated and unwell.
*  without significant hyperketonaemia or acidosis

HHS typically occurs in the elderly and is often the first presentation of Type 2 Diabetes Mellitus.

Using the blood results, osmolality can be calculated with the formula 2(Na+K) +glucose+urea

Goals of treatment include:

*  Normalise the osmolality
*  Replace fluid and electrolyte losses
*  Normalise blood glucose
*  Prevention of complications: Arterial or venous thrombosis/cerebral oedema

Fluid replacement must commence first; an initial insulin bolus of 0.15 U per kg may be given once infusions are underway. Fluid replacement alone with 0.9% sodium chloride solution will result in falling blood glucose. Insulin treatment prior to adequate fluid replacement may result in cardiovascular collapse as water moves out of the intravascular space, with a resulting decline in intravascular volume.

Patients with HHS are often exquisitely sensitive to insulin and require much lower doses than in diabetic ketoacidosis (DKA).The recommended insulin dose is a fixed rate intravenous insulin infusion (FRIII) given at 0.05 units per kg per hour (e.g. 4 units/hr in an 80 kg man) is used

Beware of rapid correction of hyponatraemia, may lead to cerebral pontine myelinolysis

**>>DESCRIPTION**: A 66-year-old male presents with worsening agitation and confusion over 1 week. History includes hypertension, ischaemic heart disease, and chronic back pain. He has lost weight (71 kg), is more tired, drinks more water, and has urinary incontinence. Examination: HR 108, BP 95/42, SpO2 94%, RR 20, GCS 14, dehydrated. Labs: Na+ 125, K+ 5.0, Urea 18, Creatinine 180, Glucose 34. VBG: pH 7.32, pCO2 4.6, pO2 6.1, HCO3 17, BE -3.6.

**>>OPTIONS**: a) 10 units of human actrapid stat b) Calcium gluconate c) Intravenous 0.9% sodium chloride d) Intravenous 1.8% sodium chloride e) Start insulin sliding scale at 6 units/hr

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Intravenous 0.9% sodium chloride

**>>REASONING**: The patient is in a hyperosmolar hyperglycemic state (HHS). Fluid replacement with 0.9% sodium chloride is the most important initial treatment. Insulin should be administered after fluid replacement to avoid cardiovascular collapse. Rapid correction of hyponatremia should be avoided.

## Question #:318

**CLINICAL SCENERIO**: A 32-year-old alcoholic with acute pancreatitis, abdominal pain, and vomiting presents with restlessness and perioral numbness. Blood results show adjusted calcium of 1.8 mmol/l.

**QUESTION LINE**: What’s the next step in management?

**OPTIONS**: 1. Parathyroid hormone 2. Oral calcium supplementation 3. Calcitonin assay 4. Intravenous 10% calcium gluconate 5. Serum magnesium

**CORRECT-CHOICE LINE**: 4

**REASONING**: Intravenous calcium gluconate is used for the acute management of hypocalcaemia

In the acute setting of a patient with symptomatic hypocalcaemia intravenous replacement with 10% calcium gluconate would be the most sensible next step. Typically this is given over 10-30 minutes ideally with cardiac monitoring.

Checking the serum magnesium would be very useful as hypomagnesaemia would require replacement to also correct hypocalcaemia.

Oral calcium replacement would be an option in asymptomatic hypocalcaemic patients.

**>>DESCRIPTION**: A 32-year-old alcoholic with acute pancreatitis presents with restlessness, perioral numbness, and hypocalcemia (1.8 mmol/l).

**>>OPTIONS**: 1. Calcitonin assay 2. Intravenous 10% calcium gluconate 3. Oral calcium supplementation 4. Parathyroid hormone 5. Serum magnesium

**>>CORRECT-CHOICE LINE**: 2

**>>CORRECT-CHOICE\_TEXT**: Intravenous 10% calcium gluconate

**>>REASONING**: The next step is intravenous 10% calcium gluconate to acutely manage symptomatic hypocalcemia. Checking serum magnesium is also useful. Oral calcium is for asymptomatic patients. Parathyroid hormone and calcitonin assay are not the immediate management steps.

## Question #:179

**CLINICAL SCENERIO**: A 50-year-old woman with Grave’s disease is reviewed 12 hours post-parathyroidectomy. She experiences carpopedal spasm, pins and needles, and a serum calcium of 1.85 mmol/l.

**QUESTION LINE**: Which of the following is the most appropriate intervention?

**OPTIONS**: a) Intravenous diazepam b) Intravenous calcium c) Intravenous magnesium d) Oral calcium e) Oral vitamin D

**CORRECT-CHOICE LINE**: b

**REASONING**: Intravenous calcium gluconate is used for acute hypocalcaemia. This patient has symptomatic hypocalcaemia post-surgery, necessitating IV calcium. Diazepam is inappropriate; IV magnesium is for refractory cases, and oral interventions are too slow.

**>>DESCRIPTION**: A 50-year-old woman post-parathyroidectomy presents with carpopedal spasm, pins and needles, and hypocalcemia.

**>>OPTIONS**: a) Intravenous calcium b) Intravenous diazepam c) Intravenous magnesium d) Oral calcium e) Oral vitamin D

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Intravenous calcium

**>>REASONING**: The correct answer is intravenous calcium because the patient has symptomatic hypocalcemia which is a medical emergency and requires immediate IV calcium replacement. Other options are not appropriate for acute management.

## Question #:94

**CLINICAL SCENERIO**: A 19-year-old woman presents to the emergency department drowsy and vomiting. She is accompanied by a friend who tells you she has been out drinking all day and has been vomiting for the last few hours.

Her speech is slurred and confused, she opens her eyes in response to her name and pushes you away in response to a painful stimulus. Heart rate is 100 beats per minute and regular, blood pressure is 100/60 mmHg, capillary glucose is 18 mmol/L, and a urine dip shows pH: 4, blood: trace, ketones: +++, protein: trace, nitrites: negative and leukocytes: negative.

Chest x-ray: Normal

Venous blood gas:

| pH | 7.27 (7.35-7.45) |
| --- | --- |
| Bicarbonate | 10mmol/L (22-26) |
| Base excess | -10 (-2 to +2) |
| Sodium | 135 mmol/L (137-144) |
| Potassium | 2.9 mmol/L (3.5-4.9) |
| Chloride | 99 mmol/L (95-107) |

## Serum Glucose: 21 mmol/L

**QUESTION LINE**: What is the most important initial intervention?

**OPTIONS**: - a) Fixed rate intravenous insulin infusion (FRIII) - b) Intravenous calcium gluconate - c) Intravenous fluids - d) Sliding scale insulin - e) Urgent CT head

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: This woman fits the diagnostic criteria for diabetic ketoacidosis (DKA):

*  Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
*  Blood glucose > 11.0mmol/L or known diabetes mellitus
*  Bicarbonate (HCO3- ) < 15.0mmol/L and/or venous pH < 7.3

Diabetes UK guidance states that the most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by insulin administration.

The key benefits of fluid resuscitation in this context include:

*  Recovery of circulatory volume
*  Clearance of ketones and therefore improvement of acidosis
*  Correction of electrolyte imbalance

Weight based fixed rate intravenous insulin infusion (FRIII) is now the recommended mode of insulin administration in DKA, over sliding scale.

DKA is a complication of type i diabetes (but can rarely complicate type ii diabetes). It can be the first presentation of type i diabetes, result from poor diabe

**>>DESCRIPTION**: A 19-year-old woman presents to the ED drowsy and vomiting after drinking. She is confused and opens her eyes to her name but withdraws to pain. HR 100, BP 100/60, capillary glucose 18 mmol/L, urine ketones +++, pH 7.27, bicarbonate 10mmol/L, base excess -10, K+ 2.9 mmol/L, serum glucose 21 mmol/L. Chest x-ray is normal.

**>>OPTIONS**: a) Fixed rate intravenous insulin infusion (FRIII) b) Intravenous calcium gluconate c) Intravenous fluids d) Sliding scale insulin e) Urgent CT head

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Intravenous fluids

**>>REASONING**: The patient meets DKA criteria. Initial management per Diabetes UK guidelines prioritizes fluid replacement to restore circulatory volume, clear ketones, and correct electrolyte imbalances, followed by insulin. While FRIII is the recommended insulin administration method, fluids come first.

## Question #:24

**CLINICAL SCENERIO**: A 20-year-old female presented with severe abdominal pain, vomiting and lethargy. She reported being unwell for four months, losing 10 Kg in weight and experiencing fatigue. Diagnosed with hypothyroidism last month, she was prescribed levothyroxine 50 mcg daily. Her mother and sister have hypothyroidism. Examination revealed dehydration, a pulse of 105 bpm, blood pressure of 70/40 mmHg, temperature of 37.6ºC and BMI of 19 kg/m². Cardiovascular, respiratory, and abdominal examinations were normal. Investigations showed low Hb, high MCV, and slightly abnormal thyroid function tests.

**QUESTION LINE**: While awaiting new investigations, what is the most appropriate immediate treatment for this patient?

**OPTIONS**: a) Intravenous glucose 10% b) Intravenous normal saline c) Intravenous normal saline and antibiotics d) Intravenous normal saline and hydrocortisone e) Intravenous thyroxine

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: This patient presented with Addisonian crisis (abdominal pain, vomiting, dehydration and hypotension). She has been complaining of tiredness and weight loss (which are features of Addisons disease) for four months but what precipitated the crisis is the thyroxine given for the presumed hypothyroidism.

Actually, a slightly raised TSH and a decreased T4 are features of primary hypoadrenalism and do not necessarily indicate frank hypothyroidism.

This is a medical emergency and should be treated immediately with intravenous normal saline and hydrocortisone. Thyroxine should not be given as it will exacerbate the condition.

Her low haemoglobin and high MCV may point towards pernicious anaemia which is an autoimmune disease seen sometimes in association with Addisons disease.

**>>DESCRIPTION**: A 20-year-old female presents with abdominal pain, vomiting, and lethargy. She reports 4 months of fatigue and 10 kg weight loss. Diagnosed with hypothyroidism and started on levothyroxine. Family history of hypothyroidism. Examination shows dehydration and hypotension. Labs show anemia and slightly abnormal thyroid function tests.

**>>OPTIONS**: a) Intravenous glucose 10% b) Intravenous normal saline c) Intravenous normal saline and antibiotics d) Intravenous normal saline and hydrocortisone e) Intravenous thyroxine

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Intravenous normal saline and hydrocortisone

**>>REASONING**: The patient is likely experiencing an Addisonian crisis, precipitated by thyroxine administration for presumed hypothyroidism, given the symptoms and slightly abnormal thyroid function tests which could indicate hypoadrenalism. Immediate treatment requires intravenous normal saline and hydrocortisone. Thyroxine is contraindicated.

## Question #:343

**CLINICAL SCENERIO**: A 20-year-old female presented to the accident and emergency department with severe abdominal pain, vomiting and lethargy. On further questioning she stated that she had been generally unwell for the last four months during which time she lost 10 Kg in weight and had been tired all the time.

Last month she has been diagnosed with hypothyroidism and was prescribed levothyroxine 50 mcg daily.

Her mother and sister have hypothyroidism and take thyroxine. On examination, she looks unwell and dehydrated.

Her pulse is 105 beats per minute and blood pressure is 70/40 mmHg

Her temperature is 37.6ºC and BMI is 19 kg/m². Cardiovascular, respiratory and abdominal examination were normal. Investigations done last month showed:

| Hb | 9.5 g/dl |
| --- | --- |
| MCV | 105 fl |
| Platelet s | 190 \* 10 9 /l |
| WBC | 4.5 \* 10 9 /l |

Serum free T4

8.5 pmol/l

Serum TSH

5.5 mU/l

**QUESTION LINE**: While awaiting new investigations, what is the most appropriate immediate treatment for this patient?

**OPTIONS**: - f) Intravenous glucose 10% - g) Intravenous normal saline - h) Intravenous normal saline and antibiotics - i) Intravenous normal saline and hydrocortisone - j) Intravenous thyroxine

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: This patient presented with Addisonian crisis (abdominal pain, vomiting, dehydration and hypotension). She has been complaining of tiredness and weight loss (which are features of Addisons disease) for four months but what precipitated the crisis is the thyroxine given for the presumed hypothyroidism.

Actually, a slightly raised TSH and a decreased T4 are features of primary hypoadrenalism and do not necessarily indicate frank hypothyroidism.

This is a medical emergency and should be treated immediately with intravenous normal saline and hydrocortisone. Thyroxine should not be given as it will exacerbate the condition.

Her low haemoglobin and high MCV may point towards pernicious anaemia which is an autoimmune disease seen sometimes in association with Addisons disease.

**>>DESCRIPTION**: A 20-year-old female presents to the ER with severe abdominal pain, vomiting, lethargy, and hypotension (BP 70/40 mmHg, HR 105 bpm). She reports 4 months of general unwellness, 10 kg weight loss, and fatigue. Diagnosed with hypothyroidism last month, she was started on levothyroxine. Family history includes hypothyroidism. Investigations show Hb 9.5 g/dl, MCV 105 fl, Platelets 190x10^9/l, WBC 4.5x10^9/l, free T4 8.5 pmol/l, TSH 5.5 mU/l.

**>>OPTIONS**: a) Intravenous glucose 10% b) Intravenous normal saline c) Intravenous normal saline and antibiotics d) Intravenous normal saline and hydrocortisone e) Intravenous thyroxine

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Intravenous normal saline and hydrocortisone

**>>REASONING**: The patient presents with an Addisonian crisis, indicated by abdominal pain, vomiting, dehydration, and hypotension, exacerbated by recent levothyroxine initiation. The slightly elevated TSH and low T4 are consistent with primary hypoadrenalism, not necessarily primary hypothyroidism. Immediate treatment for this medical emergency is intravenous normal saline and hydrocortisone. Thyroxine is contraindicated as it worsens the condition. Associated findings like macrocytic anemia (low Hb, high MCV) suggest pernicious anemia, which can co-exist with Addison’s disease.

## Question #:83

**CLINICAL SCENERIO**: A 72-year-old Japanese female presents with sudden onset shortness of breath and palpitations. She has a history of similar palpitations. Examination reveals pyrexia, tachycardia, gallop rhythm, bibasal inspiratory crackles, and mild lower limb pitting oedema. Chest radiograph shows bibasal alveolar shadowing and mild pleural effusions. ECG shows sinus tachycardia. Labs reveal suppressed TSH and elevated Free T4 and T3.

**QUESTION LINE**: What is the next most appropriate immediate treatment?

**OPTIONS**: - a) Intravenous propranolol - b) Lugol’s iodine - c) Oral carbimazole - d) Oral propylthiouracil - e) Oral prednisolone

**CORRECT-CHOICE LINE**: Correct anwer is a.

**REASONING**: In thyroid storm with IV beta-blockers are a important first-line treatment

The patient has presented with sudden onset heart failure associated with sinus tachycardia, pyrexia and thyrotoxicosis: this represents a thyroid storm and is an endocrinological emergency. The treatment comprises of four aims: resuscitation, treat the sympathetic consequences of thyrotoxicosis, block underlying hyperthyroidism and treat any heart failure present. The first step involved intravenous followed by oral beta-blockade. Diltiazem is an appropriate alternative if the patient cannot tolerate beta blockers. In cases of simultaneous poor ventricular function and thyroid storm, intravenous infusions of short-acting beta blockers such as esmolol are also appropriate, which can be switched off immediately at the earliest sign of worsening cardiac function secondary to beta blockade. Thyroid blockers can be instituted after this immediate therapy. Oral corticosteroids are also important to reduce peripheral T4 to T3 conversion. However, both treatments can be instituted after achieving haemodynamic stability.

**>>DESCRIPTION**: A 72-year-old female presents with sudden onset shortness of breath, palpitations, pyrexia, and tachycardia. Exam: bibasal crackles and edema. ECG: sinus tachycardia. Labs: suppressed TSH, elevated T4/T3. Suspect thyroid storm.

**>>OPTIONS**: a) Intravenous propranolol b) Lugol’s iodine c) Oral carbimazole d) Oral prednisolone e) Oral propylthiouracil

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Intravenous propranolol

**>>REASONING**: The patient presents with thyroid storm. The immediate treatment should address the sympathetic consequences with IV beta-blockers. Thyroid blockers and corticosteroids can follow after stabilization. Other options like Lugol’s iodine, carbimazole and propylthiouracil are not first-line, immediate treatments. Prednisolone is also important to reduce peripheral T4 to T3 conversion; however, it is not the immediate line of management.

## Question #:303

**CLINICAL SCENERIO**: A 27-year-old woman presents with palpitations, sinus tachycardia, and a non-tender goitre. Investigations show undetectable TSH, elevated free T4, and positive thyroid-stimulating hormone receptor antibodies, indicating Graves’ disease. She opts for carbimazole treatment. What other symptoms should she be warned about?

**QUESTION LINE**: What other symptoms is it important to warn her about?

**OPTIONS**: a) Loss of vision - b) Loss of peripheral sensation - c) Jaundice - d) Worsening palpitations - e) Insomnia

**CORRECT-CHOICE LINE**: C

**REASONING**: Patients taking carbimazole need to be warned about potential hepatic impairment and the relevant symptoms

This is a patient with clinical and biochemical evidence of Graves’ disease. As this has been confirmed with positive thyroid-stimulating hormone receptor antibodies it would be entirely appropriate to offer carbimazole, radioiodine or surgery. When starting carbimazole, it is very important to ensure the patient is aware of the risk of agranulocytosis, but it is also important to warn about liver dysfunction which may develop and may necessitate stopping the treatment. Rash, headache, fever and malaise commonly occur as well, but can usually be managed with analgesia and antihistamines. Loss of vision could occur in the case of moderate to severe Graves’ disease with orbitopathy and starting on radioiodine, which can worsen thyroid eye disease. Palpitations and insomnia should improve with treatment as these are symptoms of hyperthyroidism.

**>>DESCRIPTION**: A 27-year-old woman with Graves’ disease, confirmed by labs and goitre, starts carbimazole. What other symptoms should she be warned about?

**>>OPTIONS**: a) Insomnia b) Jaundice c) Loss of peripheral sensation d) Loss of vision e) Worsening palpitations

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Jaundice

**>>REASONING**: Patients on carbimazole should be warned about jaundice, as it indicates potential liver dysfunction. Other options are less relevant to carbimazole side effects or Graves’ disease symptoms.

## Question #:270

**CLINICAL SCENERIO**: An 18-year-old man presents to the clinic with poor development of secondary sexual characteristics, lack of testicle development, and sparse axillary/pubic hair.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Androgen insensitivity syndrome - b) Kallmann’s syndrome - c) Klinefelter syndrome - d) Mullerian agenesis - e) Turner’s syndrome

**CORRECT-CHOICE LINE**: b

**REASONING**: Kallman’s syndrome - LH & FSH low-normal and testosterone is low

Kallmann’s syndrome is correct. Kallmann’s syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. The biochemical profile is in keeping with hypogonadotropic hypogonadism due to the presence of low testosterone and inappropriately normal LH and FSH. Kallmann’s syndrome is the only listed condition which results in hypogonadotropic hypogonadism.

Androgen insensitivity syndrome is correct. Androgen insensitivity syndrome in its complete form is a disorder of hormone resistance characterised by a female phenotype in a genetic male due to the resistance to the effector functions of testosterone. The hormonal profile findings include very high testosterone, elevated LH, and a relatively normal FSH. Thus the patient’s phenotypical sex and hormonal profile exclude this diagnosis.

Klinefelter syndrome is incorrect. Klinefelter syndrome is an aneuploid genetic condition where a male has an additional copy of the X chromosome. The primary features are infertility and small, poorly functioning testicles. This condition results in primary gonadal failure, and thus a compensatory elevation in LH and FSH.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Given that the patient is phenotypically male, this diagnosis is unlikely.

Turner’s syndrome is incorrect. Turner’s syndrome can cause primary ovarian failure due to gonadal dysgenesis. This would present with low sex hormone levels, and markedly raised LH and FSH levels. It only affects females. Thus the patient’s phenotypic sex, and biochemical results are not in keeping with this diagnosis.

**>>DESCRIPTION**: An 18-year-old male presents with delayed development of secondary sexual characteristics, small testicles, and sparse hair. Blood tests show low testosterone with normal/low FSH.

**>>OPTIONS**: a) Androgen insensitivity syndrome b) Kallmann’s syndrome c) Klinefelter syndrome d) Mullerian agenesis e) Turner’s syndrome

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Kallmann’s syndrome

**>>REASONING**: Kallmann’s syndrome is the most likely diagnosis, presenting with low testosterone and inappropriately normal FSH/LH, consistent with hypogonadotropic hypogonadism. Other options are excluded because of hormonal profiles or patient’s sex.

## Question #:305

**CLINICAL SCENERIO**: A 22-year-old man presents to endocrinology clinic with low energy, low morning testosterone, and low sexual desire. He is tall, slim, and has gynaecomastia. What is the most appropriate investigation?

**QUESTION LINE**: What is the most appropriate investigation after confirming the low morning testosterone?

**OPTIONS**: a) Prolactin b) LH and FSH c) MRI pituitary d) Morning cortisol e) Synacthen test

**CORRECT-CHOICE LINE**: B

**REASONING**: A patient with low testosterone should have LH and FSH tested as first line investigation to separate primary and secondary hypogonadism

This is a patient with biochemical and clinical evidence of low testosterone. He is symptomatic with low energy and low sexual desire, and most likely would have erectile problems if questioned. The history of immigration is important as it raises the possibility of missed childhood diagnoses, in this case implying a missed chromosomal disorder. The most appropriate investigation to differentiate primary from secondary hypogonadism is LH and FSH. If LH and FSH are elevated this would suggest a primary hypogonadism such as Klinefelter’s syndrome, as is suggested by the description of this patient, but if it is not then a secondary cause should be found. If that were the case, then MRI of pituitary can be necessary, as well as morning cortisol and prolactin. Synacthen testing should be done if morning cortisol is low to confirm adrenal insufficiency.

**>>DESCRIPTION**: A 22-year-old man presents with low energy, low testosterone, and low sexual desire, with gynaecomastia. What is the most appropriate investigation?

**>>OPTIONS**: a) LH and FSH b) Morning cortisol c) MRI pituitary d) Prolactin e) Synacthen test

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: LH and FSH

**>>REASONING**: LH and FSH are the first-line investigations to differentiate between primary and secondary hypogonadism in a patient with low testosterone. Elevated LH/FSH suggests primary hypogonadism. Other tests (prolactin, MRI, cortisol, Synacthen) are considered based on initial findings.

## Question #:266

**CLINICAL SCENERIO**: A 45-year-old man presents with headaches, low energy, weight loss, low libido, and postural dizziness. Investigations reveal low free T4, testosterone, morning cortisol, LH, FSH, and TSH, along with a pituitary adenoma on MRI. The patient is keen to start treatment. Which hormonal replacement is contraindicated immediately?

**QUESTION LINE**: Which hormonal replacement would be contra-indicated in the immediate setting?

**OPTIONS**: a) Hydrocortisone b) Prednisolone c) Dexamethasone d) Testosterone e) Levothyroxine

**CORRECT-CHOICE LINE**: E

**REASONING**: In hypothyroidism with adrenal failure do not start thyroid hormone replacement before glucocorticoid replacement as otherwise an adrenal crisis can be precipitated

This patient has panhypopituitarism secondary to a pituitary adenoma. It is critical for patients with adrenal and thyroid insufficiency to replace steroid function prior to replacing thyroid function as otherwise an adrenal crisis can be precipitated. Therefore levothyroxine is contra-indicated in the immediate setting, even though the patient will need replacement after he has started steroid replacement. Typically he would need hydrocortisone split into three doses to replace his steroid needs, and this is preferred over dexamethasone and prednisolone.

Ultimately the treatment may be trans-sphenoidal surgery

**>>DESCRIPTION**: A 45-year-old man with panhypopituitarism (pituitary adenoma) presents with headaches, low energy, weight loss, low libido, and postural dizziness. Which immediate hormonal replacement is contraindicated?

**>>OPTIONS**: a) Dexamethasone b) Hydrocortisone c) Levothyroxine d) Prednisolone e) Testosterone

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Levothyroxine

**>>REASONING**: Levothyroxine is contraindicated initially due to the risk of precipitating adrenal crisis in the presence of adrenal insufficiency, which must be addressed with glucocorticoid replacement (e.g., hydrocortisone) first.

## Question #:13

**CLINICAL SCENERIO**: An 85-year-old woman is brought into A+E with hypothermia, sinus bradycardia and unresponsive. A CT head reveals no acute intra-cranial pathology. Passive warming and intravenous fluids are commenced. Subsequent blood tests reveal the panel below. A collateral history from family members reveals symptoms of lethargy, cold intolerance and weight gain over the last few months.

TS H

30.0 mU/L

T3

<0.05 mU/L

**QUESTION LINE**: What is the most appropriate initial treatment?

**OPTIONS**: a) Levothyroxine and Liothyronine b) Levothyroxine c) Lugol’s iodine d) Liothyronine e) Carbimazole

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: This is myxedema coma, which is rare but has a high mortality rate. Administration of both liothyronine (T3) and levothyroxine (T4) is the most appropriate treatment. Liothyronine (T3) has a greater biologic activity and faster onset than levothyroxine (T4) and should be continued until there is clinical improvement. The administration of hydrocortisone is also important as patients there may be coexisting adrenal insufficiency (primary or secondary). Supportive measures are important such as re-warming, fluid resuscitation, electrolyte correction and vasopressors if required.

Carbimazole is used for hyperthyroidism.

Lugols iodine is used for rapid control of hyperthyroidism such as a thyroid storm or prior to a total thyroidectomy for poor medical compliance.

**>>DESCRIPTION**: 85-year-old woman presents with hypothermia, sinus bradycardia, and unresponsiveness. CT head is unremarkable. History includes lethargy, cold intolerance, and weight gain. TSH >30.0 mU/L, T3 <0.05 mU/L.

**>>OPTIONS**: a) Carbimazole b) Levothyroxine c) Levothyroxine and Liothyronine d) Liothyronine e) Lugol’s iodine

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Levothyroxine and Liothyronine

**>>REASONING**: The patient is in myxedema coma, requiring both liothyronine (T3) and levothyroxine (T4). T3 has faster onset. Carbimazole and Lugol’s iodine are for hyperthyroidism.

## Question #:64

**CLINICAL SCENERIO**: A 32-year-old woman presents with recurrent headaches, blurred vision, fatigue, and constipation. She has a history of hypertension and is on amlodipine, ramipril, and bendroflumethiazide. Examination reveals a blood pressure of 178/102 mmHg, hypervolemia with peripheral oedema, and grade IV hypertensive retinopathy with papilloedema. ECG shows left ventricular hypertrophy. Urine dip is negative. CT head reveals no acute bleed. Blood results: Na+ 154 mmol/L, K+ 2.9 mmol/L, Bicarbonate 23 mmol/L, Aldosterone 70 pmol/L, Renin 2 mU/L.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Bartter’s syndrome b) Excess salt in diet c) Gitelman’s syndrome d) Liddle syndrome e) Renal artery stenosis

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Hypernatraemia associated with hypervolaemia can occur due to hypertonic saline, hypertonic sodium bicarbonate, excess salt in diet, or hyperaldosteronism

Liddle syndrome is the correct option. This rare autosomal dominant syndrome is due to dysregulation of the epithelial sodium channel (ENaC) due to a genetic mutation at the 16p13-p12 locus, leading to the Na channel not being degraded, thus leading to chronic reabsorption of sodium and water in the nephron. This mimics a state of hyperaldosteronism as aldosterone is usually the hormone implicated in inserting the ENaCs into the nephron. Therefore, this patient has a state of hypervolaemic hypernatraemia with chronically high blood pressure creating the end-organ damage to the optic nerve and heart described above.

The feedback loop in the kidneys has meant that both renin and aldosterone are low for her body to attempt to lower the sodium and water retention. This patient is fatigued and constipated due to hypokalaemia. Liddle’s is treated with a combination of a low sodium diet and potassium-sparing diuretics (e.g. amiloride).

Liddle’s has a classic triad in the textbooks of hypertension, hypokalemia and metabolic alkalosis. However, hypernatraemia is also a feature due to the above processes and this should not be forgotten in the clinical work-up.

Bartter’s syndrome is an incorrect option. This is an autosomal recessive syndrome which causes salt-wasting from the kidney and subsequently a state of hyperaldosteronism and usually low blood pressure, not high blood pressure as seen in this case.

Gitelman’s syndrome is an incorrect option as it is another autosomal recessive syndrome similar to Bartter’s, which causes low blood pressure usually.

Excess salt in the diet is incorrect. Even though it would cause hypernatraemia and chronic excess would cause states of hypertension; you would not find hypokalaemia. Furthermore, it would be a more unlikely cause at the young age of this patient with the anti-hypertensive therapy resistance.

Renal artery stenosis is an incorrect answer because this condition causes difficulty to control hypertension with a high plasma renin activity found, instead of low, as more renin is produced in response to the low pressure sensed by the constricted renal artery. The kidneys think that the blood pressure is systemically low when it is a locally mediated effect from the stenosis.

**>>DESCRIPTION**: A 32-year-old woman presents with recurrent headaches, blurred vision, fatigue, and constipation. She has a history of hypertension and is on multiple antihypertensives. Examination: BP 178/102 mmHg, hypervolemia, peripheral oedema, grade IV hypertensive retinopathy with papilloedema, and LVH on ECG. Labs: Na+ 154 mmol/L, K+ 2.9 mmol/L, Aldosterone 70 pmol/L, Renin 2 mU/L.

**>>OPTIONS**: a) Bartter’s syndrome b) Excess salt in diet c) Gitelman’s syndrome d) Liddle syndrome e) Renal artery stenosis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Liddle syndrome

**>>REASONING**: Liddle syndrome is caused by dysregulation of ENaC, leading to hypervolemic hypernatremia and hypertension. Renin and aldosterone are low. The patient also has hypokalemia. Bartter’s and Gitelman’s syndromes cause low blood pressure. Excess salt would not cause hypokalemia. Renal artery stenosis would cause high renin.

## Question #:181

**CLINICAL SCENERIO**: A 45-year-old woman with a BMI of 34 kg/m 2, type 2 diabetes, and hypertension, treated for stable angina, seeks obesity treatment options on discharge.

**QUESTION LINE**: Which of the following is the best treatment option for her obesity at this stage?

**OPTIONS**: a) Gastric banding b) Surgical gastric bypass c) Sibutramine d) Lifestyle measures (dietary, exercise, and behavioural interventions) e) Orlistat

**CORRECT-CHOICE LINE**: d

**REASONING**: According to NICE guidelines, the first-line treatment for obesity is a combination of dietary, exercise, and lifestyle interventions

Obesity and being overweight are defined as abnormal or excessive fat accumulation that may impair health. Health risks include cardiovascular disease, hypertension, hypercholesterolaemia, diabetes, musculoskeletal problems, respiratory problems (obstructive sleep apnoea and asthma), fatty liver, gallstones, and increased risk of some cancers (colon, breast, and endometrial).

According to NICE guidelines, the first-line treatment is a combination of dietary, exercise, and behavioural interventions; pharmacological therapy is recommended if there is failure of first-line interventions. Orlistat should only be used when more than 3 months’ use of dietary and lifestyle measures has failed.

## Surgery is recommended if:

*  There is a failure of non-surgical measures (no clinically beneficial weight loss over 6 months).
*  The patient has a BMI of 40 kg/m 2 or more, or a BMI of 35-40 kg/m 2 and other significant disease that could be improved if they lost weight.
*  The patient has a BMI greater than 50 kg/m 2 .
*  The patient commits to long-term follow-up which needs to be done under the supervision of a specialist obesity team.

A 19-year-old woman presents to her GP with a 7 month history of weight loss, diarrhoea and palpitations. The diarrhoea is normal colour and over the last three months she has had roughly 2-3 bowel motions per day. The heart palpitations occur randomly throughout the day and night. She has also noticed that she has recently been getting episodes of feeling very hot and sweaty. She has no other past medical history and her only family history is a mother who has Hashimotos thyroiditis.

On examination, the patient is sweaty and her blood pressure is 130/80 mmHg, pulse is 102 bpm and regular, respiratory rate is 16/min and her oxygen SATs are 98% on air.

Blood tests are performed and reveal:

| Hb | 135 g/l |
| --- | --- |
| Platelets | 220 \* 10 9 /l |
| WBC | 7.1 \* 10 9 /l |
| Na + | 139 mmol/l |
| K + | 3.9 mmol/l |
| Urea | 5.1 mmol/l |
| Creatinine | 60 µmol/l |
| Free thyroxine (T4) | 28 pmol/l |
| Thyroid stimulating hormone (TSH) | 0.08 mu/l |

A thyroid radioisotope scan is performed and reveals a globally reduced uptake.

What is the most likely diagnosis?

* 1. Graves disease
  2. Thyrotoxicosis factitial
  3. Hashimotos disease
  4. De Quervains thyroiditis
  5. Atrophic thyroiditis

Correct answer is b.

The most like likely diagnosis in this case is thyrotoxicosis factitia. This is evidenced by the reduced thyroid uptake on radioisotope scanning, along with the fact she may have easy access to thyroxine he mother would be taking it for her Hashimotos thyroiditis. De Quervains thyroiditis can present with symptoms similar to factitious thyroiditis and a decreased uptake on radioisotope scan, however, the fact the symptom shave lasted for 7 months makes this diagnosis unlikely.

**>>DESCRIPTION**: A 45-year-old woman with a BMI of 34 kg/m2, type 2 diabetes, and hypertension, treated for stable angina, seeks obesity treatment options.

**>>OPTIONS**: a) Gastric banding b) Lifestyle measures (dietary, exercise, and behavioural interventions) c) Orlistat d) Surgical gastric bypass e) Sibutramine

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Lifestyle measures (dietary, exercise, and behavioural interventions)

**>>REASONING**: The best initial treatment for obesity is lifestyle measures, including diet, exercise, and behavioral changes, as per NICE guidelines. Other options are considered after lifestyle measures have failed.

## Question #:301

**CLINICAL SCENERIO**: A 54-year-old woman is referred to cardiology clinic due to echocardiogram findings consistent with cardiomyopathy. She underwent the echocardiogram after developing shortness of breath and there were concerns about underlying heart failure. She has a past medical history of hypertension, palpitations and anxiety. She has been troubled by tremors and increasing weight loss, sweating and heat intolerance over the last three months and has been diagnosed with hyperthyroidism and started on carbimazole. What is the likely outcome of her cardiac condition?

**QUESTION LINE**: What is the likely outcome of her cardiac condition?

**OPTIONS**: a) Likely to develop permanent heart failure - b) Likely to develop pulmonary hypertension - c) Likely to develop aortic regurgitation - d) Likely to have resolution of symptoms but not of cardiomyopathy - e) Likely to have resolution of symptoms and of cardiomyopathy

**CORRECT-CHOICE LINE**: Answer is E.

**REASONING**: Thyrotoxicosis is associated with reversible cardiomyopathy

The patient has thyrotoxicosis. The hyperthyroidism can cause cardiac complication by a rate-related mechanism. Due to this, patients can develop tachycardia, palpitations, AF and rate-related cardiac failure, as well as cardiomyopathy. All of these problems are likely to resolve when the thyrotoxicosis resolves, and this patient has been diagnosed and treatment has been started with carbimazole.

**>>DESCRIPTION**: A 54-year-old woman with hyperthyroidism, hypertension, palpitations, anxiety, and cardiomyopathy presents with shortness of breath. She is started on carbimazole. What is the likely outcome of her cardiac condition?

**>>OPTIONS**: a) Likely to develop aortic regurgitation b) Likely to develop permanent heart failure c) Likely to develop pulmonary hypertension d) Likely to have resolution of symptoms but not of cardiomyopathy e) Likely to have resolution of symptoms and of cardiomyopathy

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Likely to have resolution of symptoms and of cardiomyopathy

**>>REASONING**: The cardiomyopathy is likely reversible due to the underlying thyrotoxicosis. Treatment with carbimazole will resolve the hyperthyroidism and subsequently improve the cardiac condition.

## Question #:244

**CLINICAL SCENERIO**: A 54-year-old woman with metastatic pancreatic cancer undergoing palliative chemotherapy (FOLFIRINOX) presents with diarrhea, vomiting, nausea, and fatigue. She has a history of hypothyroidism, epilepsy, and bipolar disorder, and takes various medications including levothyroxine, levetiracetam, lithium, loperamide, paracetamol, oramorph, zomorph, movicol and ondansetron. Lab results show corrected calcium of 3.3µmol/l. She is started on IV 0.9% saline and IV alendronate. Which medication should not be prescribed on admission?

**QUESTION LINE**: Which of her regular medications should not be prescribed on admission?

**OPTIONS**: - a. Zomorph - b. Levetiracetam - c. Loperamide - d. Levothyroxine - e. Lithium

**CORRECT-CHOICE LINE**: E

**REASONING**: This patient has developed hypercalcaemia in the context of cancer but whilst also taking lithium. Lithium can be a cause of elevated calcium and should, therefore, be held in the acute context until the cause of hypercalcaemia is established. Note that the serum concentration of lithium does not correlate with the likelihood of it being a cause of hypercalcaemia. In the context of new confusion or dehydration, withholding the opiate medications would have been reasonable.

**>>DESCRIPTION**: A 54-year-old woman with metastatic pancreatic cancer on FOLFIRINOX presents with diarrhea, vomiting, nausea, and fatigue. She has a history of hypothyroidism, epilepsy, and bipolar disorder and takes multiple medications. Lab results show corrected calcium is elevated. The patient is started on IV saline and alendronate. Which medication should be held?

**>>OPTIONS**: a. Levetiracetam b. Levothyroxine c. Lithium d. Loperamide e. Zomorph

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Lithium

**>>REASONING**: Lithium should be held due to hypercalcaemia. Lithium can elevate calcium levels. Opiates could be considered for holding if confusion or dehydration were present.

## Question #:62

**CLINICAL SCENERIO**: A 48-year-old woman presents with a 2-month history of fatigue, anorexia and nausea. She has also noticed some darkening of the skin on her hands. Prior to this, she was fit and well, with no significant past medical history. She has a strong family history of Hashimoto’s thyroiditis.

On examination, there is some evidence of wasting of the face, and noticeable hyperpigmentation of the palmar creases. Her blood pressure is 105/75 mmHg. Blood tests are taken:

| Na + | 134 mmol/L | (135 - 145) |
| --- | --- | --- |
| K + | 5.3 mmol/L | (3.5 - 5.0) |

| Urea | 6.8 mmol/L | (2.0 - 7.0) |
| --- | --- | --- |
| Creatinine | 76 µmol/L | (55 - 120) |
| Calcium | 2.43 mmol/L | (2.1-2.6) |
| Thyroid stimulating hormone (TSH) | 1.2 mU/L | (0.5-5.5) |
| Free thyroxine (T4) | 13.3 pmol/L | (9.0 - 18) |

An initial test is conducted to confirm the likely diagnosis.

**QUESTION LINE**: Which subsequent test can be performed to distinguish a primary cause from a secondary cause?

**OPTIONS**: - a) High-dose dexamethasone suppression test - b) Long Synacthen test - c) Low-dose dexamethasone suppression test - d) Short Synacthen test - e) Urinary metanephrines

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

The diagnosis here is that of adrenal insufficiency. The history of fatigue, anorexia and nausea points towards this, along with evidence of hyperpigmentation of the palmar creases, hypotension, hyponatraemia and hyperkalaemia. A primary failure is known as Addison’s disease. To make the initial diagnosis of adrenal failure, a short Synacthen test should be used - cortisol will fail to rise. However, the question is asking specifically how to differentiate primary from secondary adrenal failure, secondary being a failure of the pituitary to make sufficient ACTH. For this, the long Synacthen test should be used - a higher dose is given and then cortisol levels measured over a longer time period. With primary failure, the adrenals will still be unable to produce cortisol, but with secondary failure, the exogenous ACTH will eventually push the adrenals to produce cortisol.

The high-dose dexamethasone suppression test is used to differentiate primary and secondary causes of Cushing’s syndrome, not adrenal insufficiency. This would have instead presented with weight gain and hypokalemia, not the other way around.

The low-dose dexamethasone suppression test is used first-line to make the initial diagnosis of Cushing’s. It does not play a role here.

The short Synacthen test is used to initially diagnose adrenal insufficiency. It does not, however, differentiate between primary and secondary causes - the long test does this.

Urinary metanephrines are used to diagnose pheochromocytoma. In this condition, there is increased secretion of catecholamines and patients present with hypertension, headaches, sweating and palpitations.

**>>DESCRIPTION**: A 48-year-old woman presents with fatigue, anorexia, nausea, and skin darkening. Examination reveals facial wasting and hyperpigmentation of palmar creases. BP is 105/75 mmHg. Labs show Na+ 134 mmol/L and K+ 5.3 mmol/L. Likely diagnosis needs confirmation.

**>>OPTIONS**: a) High-dose dexamethasone suppression test b) Long Synacthen test c) Low-dose dexamethasone suppression test d) Short Synacthen test e) Urinary metanephrines

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Long Synacthen test

**>>REASONING**: The long Synacthen test distinguishes between primary and secondary adrenal failure. In primary failure, adrenals won’t produce cortisol even with exogenous ACTH, while in secondary failure, exogenous ACTH will stimulate cortisol production. High and Low-dose dexamethasone suppression tests are for Cushing’s syndrome. Short Synacthen test is for initial adrenal insufficiency diagnosis. Urinary metanephrines are for pheochromocytoma.

## Question #:300

**CLINICAL SCENERIO**: A 22-year-old woman presents with dysuria, increased frequency, and positive urine glucose and nitrites. Capillary blood glucose is elevated. Two weeks later, urine glucose is still positive, and blood glucose remains elevated. Family history includes hypertension and an HNF1A genetic mutation in her father.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate initial treatment for the likely diagnosis?

**OPTIONS**: - a. Dietary and lifestyle change - b. Insulin - c. Low-dose gliclazide - d. Metformin - e. No treatment required

**CORRECT-CHOICE LINE**: c

**REASONING**: MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

Genetic testing would be required to confirm, but the likely diagnosis here is MODY3 which is associated with HNF1A mutations. These mutations are usually inherited in an autosomal dominant pattern. The presentation is often incidental, with recurrent hyperglycaemia without ketosis.

Low dose gliclazide is the correct answer. HNF1A mutations lead to a reduction in the amount of insulin secreted by the pancreas.

Metformin is incorrect. The main mechanism of action of metformin is increased sensitivity to insulin. Gliclazide is the preferred treatment as the HNF1A mutation reduces insulin production by the pancreas. It responds better to insulin secretagogue treatment than an insulin-sensitising agent.

Insulin is incorrect. Most patients with MODY do not require insulin as there is generally some insulin secretion from the pancreas particularly in early diagnosis. Treatment with a secretagogue such as gliclazide is therefore preferable.

No treatment required is incorrect. All forms of MODY apart from glucokinase MODY increase the risk of long-term complications of diabetes. Treatment to stabilise and control blood glucose levels is therefore advisable.

Dietary advice and lifestyle changes is an incorrect answer. Although this advice would form part of the management, the main treatment would be pharmacological with a low dose of gliclazide. Unlike in early type-2 diabetes or pre-diabetes where there is a possibility of improving insulin sensitivity by decreasing glucose spikes, MODY is not reversible.

**>>DESCRIPTION**: A 22-year-old woman presents with dysuria, increased frequency, positive urine glucose and nitrites, and elevated capillary blood glucose. She has persistent hyperglycemia and a family history of hypertension and an HNF1A genetic mutation.

**>>OPTIONS**: a. Dietary and lifestyle change b. Insulin c. Low-dose gliclazide d. Metformin e. No treatment required

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Low-dose gliclazide

**>>REASONING**: The correct answer is low-dose gliclazide because HNF1A mutations reduce insulin secretion, and sulfonylureas are effective. Metformin and insulin are less appropriate. Dietary changes alone are insufficient.

## Question #:4

**CLINICAL SCENERIO**: A 40-year-old female presents to Endocrinology Clinic with a 3-month history of weight gain, fatigue, headaches, galactorrhoea, and reduced libido. She has type 2 diabetes, hypertension, hirsutism, acne, a cervical fat pad, striae, proximal myopathy, and hyperpigmentation.

**QUESTION LINE**: Which of the following investigations will reveal the diagnosis?

**OPTIONS**: - a) Low dose dexamethasone suppression test - b) Prolactin levels - c) Urinary cortisol - d) CT brain - e) MRI pituitary

**CORRECT-CHOICE LINE**: e

**REASONING**: The history suggests Cushing’s syndrome and hyperprolactinaemia, with headaches potentially indicating intracranial pathology. A secreting pituitary tumour causing raised prolactin and ACTH should be suspected. Low dose dexamethasone suppression test and 24-hour urinary cortisol confirm Cushing’s, but MRI pituitary leads to the diagnosis of a pituitary tumour.

**>>DESCRIPTION**: A 40-year-old female presents with weight gain, fatigue, headaches, galactorrhoea, reduced libido, type 2 diabetes, hypertension, hirsutism, acne, a cervical fat pad, striae, proximal myopathy, and hyperpigmentation. Which investigation reveals the diagnosis?

**>>OPTIONS**: a) CT brain b) Low dose dexamethasone suppression test c) MRI pituitary d) Prolactin levels e) Urinary cortisol

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: MRI pituitary

**>>REASONING**: MRI pituitary is the key investigation to diagnose the suspected pituitary tumor. Other tests (dexamethasone suppression test, urinary cortisol, prolactin levels, and CT brain) are not as specific for the primary diagnosis in this presentation.

## Question #:97

**CLINICAL SCENERIO**: A 62-year-old woman attends her GP complaining of weight gain, lethargy and hair loss. She denies any intercurrent illness. Thyroid function tests are performed and the results are as follows:

| Thyroid stimulating hormone (TSH) | 0.3 mu/l |
| --- | --- |
| Free T4 | 8 pmol/l |

**QUESTION LINE**: Which investigation is most likely to be diagnostic?

**OPTIONS**: - a) Thyroid ultrasound - b) Radio-iodine uptake scan - c) Anti-thyroid peroxidase (TPO) antibodies - d) Fine-needle aspiration of thyroid - e) MRI pituitary glan

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: This patient has hypothyroidism. The vast majority of cases are primary hypothyroidism with a high TSH and low T4. The common causes are:

*  Autoimmune (Hashimoto’s disease, atrophic)
*  Iodine deficiency
*  Thyroiditis (post-viral, post-partum)
*  Iatrogenic (thyroidectomy, radioiodine, drugs)

Secondary hypothyroidism is very rare and results in a low TSH and low T4. In these cases, pituitary insufficiency is most likely and therefore an MRI of the gland should be performed.

**>>DESCRIPTION**: 62-year-old woman presents with weight gain, lethargy, and hair loss. TSH is 0.3 mu/l, Free T4 is 8 pmol/l.

**>>OPTIONS**: a) Anti-thyroid peroxidase (TPO) antibodies b) Fine-needle aspiration of thyroid c) MRI pituitary glan d) Radio-iodine uptake scan e) Thyroid ultrasound

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: MRI pituitary glan

**>>REASONING**: Low TSH and low T4 indicate secondary hypothyroidism, likely due to pituitary insufficiency. Therefore, MRI of the pituitary gland is the most appropriate diagnostic investigation. Other options target the thyroid gland itself, which is less likely in secondary hypothyroidism.

## Question #:87

**CLINICAL SCENERIO**: A 78-year-old man with type 2 diabetes mellitus, hypertension, and hypothyroidism is reviewed. He takes metformin 1g twice daily. His HbA1c increased from 44 mmol/mol (6.2%) to 46 mmol/mol (6.4%) in one year.

**QUESTION LINE**: What is the most appropriate next step in management?

**OPTIONS**: a) Increase dose of metformin b) Add glimepiride c) Add sitagliptin d) Add pioglitazone e) Make no changes

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: This man has acceptable glycaemic control, both in terms of NICE guidance and more recent evidence looking at the harms of overzealous glycaemic control. No changes should therefore be made for now.

**>>DESCRIPTION**: 78M with T2DM, HTN, and hypothyroidism, on metformin 1g BID. HbA1c increased from 6.2% to 6.4% in one year.

**>>OPTIONS**: a) Add glimepiride b) Add pioglitazone c) Add sitagliptin d) Increase dose of metformin e) Make no changes

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Make no changes

**>>REASONING**: The patient has acceptable glycemic control according to NICE guidelines. Overzealous control can be harmful, so no changes are needed.

## Question #:108

**CLINICAL SCENERIO**: A 33-year-old woman with a history of Graves’ disease, treated with radioiodine one year prior, presents to the endocrinology clinic. She is clinically and biochemically euthyroid with TSH 2.6 and free T4 of 8.2mg/dl. She stopped oral contraceptive tablets and is planning to become pregnant.

**QUESTION LINE**: What is the most appropriate plan?

**OPTIONS**: - a) Advise to avoid pregnancy - b) Measure serum thyroid-stimulating hormone receptor antibodies - c) Arrange for US scan of neck - d) Repeat TSH and free T4 in one year’s time - e) Measure thyrotrophin receptor stimulating antibodies

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Pregnant woman with a history of Grave’s disease should have thyroid stimulating hormone binding antibody titres measured even if euthyroid as the antibodies can cross the placental barrier

This is a patient with a past medical history of Graves’ disease who is clinically and biochemically euthyroid who is planning pregnancy. It is important, even with no biochemical evidence of hyperthyroidism, to exclude the serum presence of thyroid-stimulating hormone receptor antibodies as these can cross the placenta and cause foetal problems. If they were positive, then treatment should be initiated to control the antibody levels, despite the normal TSH and T4. Thyrotrophin receptor stimulating antibodies should be checked in hyperthyroidism at 30-36 weeks gestation to help assess the risk of neonatal thyroid problems. There is no reason she should not become pregnant.

**>>DESCRIPTION**: A 33-year-old woman, post-radioiodine treatment for Graves’ disease and currently euthyroid (TSH 2.6, free T4 8.2mg/dl), plans to become pregnant after stopping oral contraceptives.

**>>OPTIONS**: a) Advise to avoid pregnancy b) Arrange for US scan of neck c) Measure serum thyroid-stimulating hormone receptor antibodies d) Measure thyrotrophin receptor stimulating antibodies e) Repeat TSH and free T4 in one year’s time

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Measure serum thyroid-stimulating hormone receptor antibodies

**>>REASONING**: Even in euthyroid patients with a history of Graves’ disease, thyroid-stimulating hormone receptor antibodies should be measured before pregnancy, as they can cross the placenta. Measuring thyrotrophin receptor stimulating antibodies is more appropriate in hyperthyroidism, and there’s no indication to advise against pregnancy.

## Question #:333

**CLINICAL SCENERIO**: A 33-year-old woman with a history of Graves’ disease treated with radioiodine, now euthyroid after stopping oral contraceptives, plans to become pregnant. Her TSH is 2.6 and free T4 is 8.2mg/dl.

**QUESTION LINE**: What is the most appropriate plan?

**OPTIONS**: f) Advise to avoid pregnancy g) Measure serum thyroid-stimulating hormone receptor antibodies h) Arrange for US scan of neck i) Repeat TSH and free T4 in one year’s time j) Measure thyrotrophin receptor stimulating antibodies

**CORRECT-CHOICE LINE**: b

**REASONING**: Pregnant woman with a history of Grave’s disease should have thyroid stimulating hormone binding antibody titres measured even if euthyroid as the antibodies can cross the placental barrier

This is a patient with a past medical history of Graves’ disease who is clinically and biochemically euthyroid who is planning pregnancy. It is important, even with no biochemical evidence of hyperthyroidism, to exclude the serum presence of thyroid-stimulating hormone receptor antibodies as these can cross the placenta and cause foetal problems. If they were positive, then treatment should be initiated to control the antibody levels, despite the normal TSH and T4. Thyrotrophin receptor stimulating antibodies should be checked in hyperthyroidism at 30-36 weeks gestation to help assess the risk of neonatal thyroid problems. There is no reason she should not become pregnant.

**>>DESCRIPTION**: A 33-year-old woman with a history of Graves’ disease, now euthyroid and planning pregnancy, has a TSH of 2.6 and free T4 of 8.2mg/dl. What’s the best course of action?

**>>OPTIONS**: a) Advise to avoid pregnancy b) Arrange for US scan of neck c) Measure serum thyroid-stimulating hormone receptor antibodies d) Measure thyrotrophin receptor stimulating antibodies e) Repeat TSH and free T4 in one year’s time

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Measure serum thyroid-stimulating hormone receptor antibodies

**>>REASONING**: Measure thyroid-stimulating hormone receptor antibodies to assess for potential fetal issues due to antibody transfer across the placenta. Other options are not the priority.

## Question #:61

**CLINICAL SCENERIO**: A 47-year-old man with poorly controlled hypertension despite multiple medications was found to have a phaeochromocytoma and a cold thyroid nodule. He was treated with alpha and beta blockers in preparation for surgery.

**QUESTION LINE**: Which type of thyroid cancer would you expect this to be histologically?

**OPTIONS**: - a) Papillary - b) Follicular - c) Anaplastic - d) Lymphoma - e) Medullary

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: This question tests your knowledge of multiple endocrine neoplasias (MEN). This patient could have MEN Type IIa or IIb, which both include phaeochromocytoma and medullary thyroid cancer.

Of all thyroid cancers, medullary thyroid cancer accounts for approximately 5% (and of these, 80% will be sporadic rather than associated with MEN).

**>>DESCRIPTION**: 47-year-old man with hypertension and a thyroid nodule is diagnosed with phaeochromocytoma. Which thyroid cancer type is most likely in this patient?

**>>OPTIONS**: a) Anaplastic b) Follicular c) Lymphoma d) Medullary e) Papillary

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Medullary

**>>REASONING**: The patient’s phaeochromocytoma suggests a possible MEN IIa or IIb syndrome, which are associated with medullary thyroid cancer. Therefore, medullary thyroid cancer is the most likely histological type.

## Question #:120

**CLINICAL SCENERIO**: A 54-year-old man presents with polyuria, polydipsia, and lethargy. Fasting glucose is 7.6 mmol/l. No family history of diabetes. Treated for hypertension and dyslipidaemia. BP is 155/90 mmHg, pulse is 70 bpm, BMI is 34 kg/m². GAD+ antibodies, normal renal function.

**QUESTION LINE**: Which of the following is most appropriate with respect to managing his glucose control?

**OPTIONS**: - a) Gliclazide - b) Liraglutide - c) Metformin - d) Sitagliptin - e) Basal bolus insulin

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Up to 10% of patients who are thought to have type 2 diabetes are found to also be GAD autoantibody positive. These individuals are thought to progress faster to insulin start than patients with autoantibody negative type 2 diabetes, (within 3-5 years vs 7 years on average for ‘standard’ patients).

The management of these patients is the same as for patients with autoantibody negative disease, metformin as the initial therapy of choice. Weight reduction may delay progression to insulin, therefore insulin sparing strategies, at least during the first few years after diagnosis are the preferred intervention. These may include use of SGLT-2 inhibitors or GLP-1 agonists.

**>>DESCRIPTION**: 54-year-old with polyuria, polydipsia, lethargy, fasting glucose 7.6 mmol/l, GAD+ antibodies, BMI 34. Treated for hypertension and dyslipidaemia. No family history of diabetes.

**>>OPTIONS**: a) Basal bolus insulin b) Gliclazide c) Liraglutide d) Metformin e) Sitagliptin

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Metformin

**>>REASONING**: Metformin is the initial therapy of choice for managing glucose control, even in GAD autoantibody positive patients, similar to autoantibody negative type 2 diabetes.

## Question #:170

**CLINICAL SCENERIO**: A 34-year-old Indian man with type 1 diabetes has poor control with a twice-daily insulin regime, elevated HbA1c, and high blood glucose. He’s overweight (BMI 29 kg/m2) and concerned about increasing insulin.

**QUESTION LINE**: Apart from increasing insulin, are there any other medical management options to better control his diabetes?

**OPTIONS**: 1. No further medical treatment 2. Metformin 3. Gliclazide 4. Acarbose 5. Pioglitazone

**CORRECT-CHOICE LINE**: b

**REASONING**: The correct answer is metformin. This is a patient with poorly controlled type 1 diabetes. Of importance to this case, he is also overweight and is of ethnic Indian origin. Increasing insulin doses is seldom popular due to the side effects. NICE recommends that in overweight people of Indian origin metformin is an alternative to increasing insulin and may be helpful, especially as it would not lead to further weight increase.

**>>DESCRIPTION**: A 34-year-old Indian man with type 1 diabetes, poor control on a twice-daily insulin regime, and overweight (BMI 29 kg/m2) seeks alternative management options.

**>>OPTIONS**: 1. Acarbose 2. Gliclazide 3. Metformin 4. No further medical treatment 5. Pioglitazone

**>>CORRECT-CHOICE LINE**: 3

**>>CORRECT-CHOICE\_TEXT**: Metformin

**>>REASONING**: Metformin is the best option, especially in an overweight patient of Indian origin with type 1 diabetes, as it can improve control without increasing weight, as recommended by NICE.

## Question #:253

**CLINICAL SCENERIO**: A 65-year-old Muslim man with type 2 diabetes (controlled by diet and metformin 500mg tds, HbA1c 6.4%) seeks advice about fasting for Ramadan.

**QUESTION LINE**: If he decides to fast during Ramadan, what is the most appropriate advice to give regarding his metformin?

**OPTIONS**: a) Metformin should be stopped b) Metformin 1.5g after sunset c) Metformin 500mg before sunrise, 1g after sunset d) Metformin 500mg after sunset e) Metformin 1g before sunrise, 500mg after sunset

**CORRECT-CHOICE LINE**: c

**REASONING**: During Ramadan, one-third of the normal metformin dose should be taken before sunrise and two-thirds should be taken after sunset

**>>DESCRIPTION**: A 65-year-old Muslim man with type 2 diabetes (controlled by diet and metformin, HbA1c 6.4%) seeks advice about fasting for Ramadan.

**>>OPTIONS**: a) Metformin 1.5g after sunset b) Metformin 500mg after sunset c) Metformin 500mg before sunrise, 1g after sunset d) Metformin 1g before sunrise, 500mg after sunset e) Metformin should be stopped

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Metformin 500mg before sunrise, 1g after sunset

**>>REASONING**: The correct approach is to divide the metformin dose: a smaller dose before sunrise and a larger dose after sunset to manage blood sugar effectively during fasting. Incorrect options involve stopping metformin, taking the entire dose at once, or incorrectly dividing the dose, potentially leading to poor glucose control or increased side effects.

## Question #:210

**CLINICAL SCENERIO**: A 62-year-old Caucasian male inpatient with community-acquired pneumonia, a history of excessive alcohol consumption (abstinent for four days), elevated blood glucose, and a new diagnosis of type 2 diabetes, presents with Cushingoid features. An overnight low-dose dexamethasone suppression test showed an 8 am cortisol of 438 nmol/L (reference range 170-540 nmol/L).

**QUESTION LINE**: What is most appropriate next step in the investigation of this gentleman?

**OPTIONS**: a) Serum ACTH measurement b) Midnight serum cortisol c) Inferior petrosal sinus sampling post CRH administration d) High dose dexamethasone suppression test e) MRI pituitary

**CORRECT-CHOICE LINE**: b

**REASONING**: Pseudo-Cushing’s syndrome, common in those with excessive alcohol consumption, mimics Cushing’s syndrome. The correct approach involves measuring midnight cortisol to assess for diurnal variation, which is usually maintained in pseudo-Cushing’s.

**>>DESCRIPTION**: A 62-year-old male with a history of alcohol abuse, now abstinent for four days, presents with Cushingoid features, elevated blood glucose, and a dexamethasone suppression test result. What is the next best step?

**>>OPTIONS**: a) High dose dexamethasone suppression test b) Inferior petrosal sinus sampling post CRH administration c) MRI pituitary d) Midnight serum cortisol e) Serum ACTH measurement

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Midnight serum cortisol

**>>REASONING**: Measure midnight cortisol to assess for diurnal variation, which is usually maintained in pseudo-Cushing’s syndrome, common in patients with alcohol abuse.

## Question #:34

**CLINICAL SCENERIO**: An 89-year-old woman with a history of delirium, triggered by a change in housing and constipation, is reviewed in the frailty clinic. She was treated for a suspected UTI with trimethoprim, but urine culture showed no growth. She now has urge incontinence impacting her quality of life and has not responded to bladder training.

**QUESTION LINE**: What is the most appropriate medication to use in this case?

**OPTIONS**: a) Darifenacin b) Duloxetine c) Mirabegron d) Oxybutynin e) Tolterodine

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Anticholinergics for urge incontinence are associated with confusion in elderly people - mirabegron is a preferable alternative

Urge incontinence can generally be managed with bladder training, but if this is unsuccessful, a trial of medication is recommended in the NICE guidelines.

The correct answer, in this case, is mirabegron . Although anticholinergic drugs are first-line, this is recommended as an acceptable alternative when they are contra- indicated.

Anticholinergic drugs (such as darifenacin, oxybutynin and tolterodine ) are first line, but there are some safety concerns, particularly in elderly patients at risk of delirium. This makes these options incorrect choices for this patient, who has been admitted recently with delirium.

Duloxetine is incorrect. It is not used in the management of urge incontinence but is a second-line treatment for stress incontinence when other measures have failed.

**>>DESCRIPTION**: 89-year-old woman with delirium history, triggered by housing change and constipation, now has persistent urge incontinence despite bladder training.

**>>OPTIONS**: a) Darifenacin b) Duloxetine c) Mirabegron d) Oxybutynin e) Tolterodine

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Mirabegron

**>>REASONING**: Mirabegron is preferred over anticholinergics (darifenacin, oxybutynin, tolterodine) due to the risk of confusion in elderly patients with delirium history. Duloxetine is for stress incontinence, not urge incontinence.

## Question #:261

**CLINICAL SCENERIO**: A 34-year-old woman with type 1 diabetes experiences nocturnal hypoglycemia (2-4 am) and subsequent morning hyperglycemia while on a twice-daily mixed insulin regimen.

**QUESTION LINE**: What is the most appropriate change to her current insulin treatment?

**OPTIONS**: - a. Move to a basal bolus of insulin - b. Take 1 dextrose tablet at 9pm - c. Reduce nocturnal insulin dose - d. Add gliclazide

**CORRECT-CHOICE LINE**: Answer is A .

**REASONING**: This patient is experiencing severe nocturnal hypoglycaemic episodes followed by rises in blood glucose by breakfast time. The most appropriate change to the insulin treatment regimen is changing to a basal bolus regime, with one daily injection of background long-acting insulin and three short acting injections of insulin

**>>DESCRIPTION**: A 34-year-old woman with type 1 diabetes experiences nocturnal hypoglycemia and morning hyperglycemia on a twice-daily mixed insulin regimen.

**>>OPTIONS**: a. Add gliclazide b. Move to a basal bolus of insulin c. Reduce nocturnal insulin dose d. Take 1 dextrose tablet at 9pm

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Move to a basal bolus of insulin

**>>REASONING**: Switching to a basal-bolus regimen is the most suitable approach to address nocturnal hypoglycemia and morning hyperglycemia. It allows for better control by providing a background insulin dose and mealtime insulin adjustments, which is not provided by other alternatives.

## Question #:143

**CLINICAL SCENERIO**: A 66-year-old man presents with increasing lethargy and confusion. His medical history includes depression, Barrett’s oesophagus, and benign prostatic hyperplasia. Current medications are sertraline, lansoprazole, and tamsulosin. Blood tests reveal hyponatraemia, hypercalcemia, and elevated total protein.

**QUESTION LINE**: What is the most likely cause of this patient’s hyponatraemia?

**OPTIONS**: a) Syndrome of inappropriate ADH secretion due to underlying malignancy b) Multiple myeloma c) Psychogenic polydipsia d) Adrenal insufficiency e) Drug-induced syndrome of inappropriate ADH secretion

**CORRECT-CHOICE LINE**: b

**REASONING**: This patient has pseudohyponatraemia secondary to raised protein levels from an underlying myeloma. Raised protein levels and hyperlipidaemia are both important causes to consider when investigating hyponatraemia. These patients have a normal total sodium but concentrations are skewed due to the high volume of lipids or protein.

Syndrome of inappropriate ADH secretion causes a euvolemic hyponatraemia. Patients have a urinary sodium >20mmol/l with raised urinary osmolality and normal/low plasma osmolality. Psychogenic polydipsia is rare and both urinary and plasma osmolalities are low in these patients. Adrenal insufficiency can cause hyponatraemia but the presence of hypercalcaemia, renal impairment, and raised total in this patient make myeloma the most likely underlying cause.

**>>DESCRIPTION**: A 66-year-old man presents with lethargy, confusion, hyponatraemia, hypercalcemia, and elevated total protein. He is taking sertraline, lansoprazole, and tamsulosin.

**>>OPTIONS**: a) Adrenal insufficiency b) Drug-induced syndrome of inappropriate ADH secretion c) Multiple myeloma d) Psychogenic polydipsia e) Syndrome of inappropriate ADH secretion due to underlying malignancy

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Multiple myeloma

**>>REASONING**: The most likely cause is multiple myeloma due to pseudohyponatraemia caused by elevated protein levels. SIADH, psychogenic polydipsia and adrenal insufficiency are less likely due to the specific lab findings (hypercalcemia and renal impairment).

## Question #:236

**CLINICAL SCENERIO**: A 56-year-old man with type 1 diabetes presents with one day of diarrhea, vomiting, and chest pain, leading to rising blood glucose despite insulin. Examination reveals tachycardia, tachypnea, and dry mucosa. Lab results show hyperglycemia, ketosis, and ECG changes. The HbA1c is elevated.

**QUESTION LINE**: What is the likeliest cause that precipitated this presentation?

**OPTIONS**: a. Missed insulin dose b. Faulty blood glucose monitor c. Lower respiratory tract infection d. Myocardial infarction e. Gastritis

**CORRECT-CHOICE LINE**: D

**REASONING**: Myocardial infarction can precipitate diabetic ketoacidosis

This patient is a well controlled type 1 diabetic presenting with diabetic ketoacidosis (DKA) with no clear precipitant. Given a normal HbA1c and denial of any lifestyle changes, we should assume that he has not just missed his insulin. The blood glucose monitors are very reliable these days and rarely are wrong. Gastritis would not cause DKA. Myocardial infarction (MI) and lower respiratory tract infections can both precipitate DKA and given the burning chest pain and ECG changes, it would be warranted to do a troponin immediately as an MI this is the likeliest cause of DKA. To remember the causes of DKA, think 3 ’I’s which are insulin (missed), infection or infarction (i.e. MI).

**>>DESCRIPTION**: A 56-year-old type 1 diabetic presents with diarrhea, vomiting, chest pain, hyperglycemia, ketosis, tachycardia, and ECG changes. What is the most likely precipitating cause?

**>>OPTIONS**: a. Faulty blood glucose monitor b. Gastritis c. Lower respiratory tract infection d. Missed insulin dose e. Myocardial infarction

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Myocardial infarction

**>>REASONING**: Myocardial infarction (MI) is the most likely cause of diabetic ketoacidosis (DKA) in this patient, supported by chest pain and ECG changes. Missed insulin is less likely due to normal HbA1c and denial of missed doses. A faulty blood glucose monitor and gastritis are less plausible.

## Question #:257

**CLINICAL SCENERIO**: An 18-year-old male with no prior medical history is admitted to the resuscitation room with hypotension, tachycardia, fever, and a widespread, non-blanching, purple rash. Diagnosed with meningococcal septicaemia and subsequently develops Waterhouse-Friderichsen syndrome.

**QUESTION LINE**: Which of the following sets of blood results would be most consistent with his condition at this point?

**OPTIONS**: - a) Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 7.5mmol/L

1. Na+ 147mmol/L, K+ 3.0mmol/L, Glucose 2.0mmol/L
   1. Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L
   2. Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L
   3. Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L

**CORRECT-CHOICE LINE**: c

**REASONING**: Waterhouse-Friderichsen syndrome is defined as adrenal failure due to bleeding into the adrenal glands (otherwise referred to as haemorrhagic adrenalitis) and is most commonly caused by meningococcal septicaemia. The biochemical pattern which results is analogous to that seen in an Addisonian crisis, and so you would expect to see hyponatraemia, hyperkalaemia and hypoglycaemia

**>>DESCRIPTION**: An 18-year-old male with meningococcal septicaemia develops Waterhouse-Friderichsen syndrome. What blood results are most consistent?

**>>OPTIONS**: a) Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 7.5mmol/L b) Na+ 147mmol/L, K+ 3.0mmol/L, Glucose 2.0mmol/L c) Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L d) Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L e) Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L

**>>REASONING**: The correct answer is hyponatremia, hyperkalemia, and hypoglycemia. This is due to adrenal failure and is analogous to an Addisonian crisis. Other options are incorrect because they don’t reflect this electrolyte and glucose disturbances.

## Question #:32

**CLINICAL SCENERIO**: A 45-year-old lorry driver presents with reduced libido and lack of energy, 5 years after a traumatic head injury. His BMI is 42 kg/m 2. Investigations show FT4 8.1pmol/l (11.5-22.7), TSH 0.4mU/l (0.35-5.5), FSH 2.2U/l (1.4-18.1), LH 3.5U/l (3.0-8.0), Testosterone 6.8nmol/l (8.4-28.7), IGF-1 35nmol/l (16-118), and Prolactin 880mU/l (45-375).

**QUESTION LINE**: Which one of the following is the most likely diagnosis, based on his clinical profile?

**OPTIONS**: a) Microprolactinoma b) Morbid obesity c) Non-functioning pituitary adenoma d) Post traumatic pituitary apoplexy e) Primary hypothyroidism

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The presence of an elevated prolactin level along with secondary hypothyroidism and hypogonadism is indicative of stalk compression is consistent with a nonfunctioning pituitary adenoma

This gentleman has secondary hypothyroidism and hypogonadism in the presence of elevated prolactin levels, which are indicative of stalk compression due to a pituitary adenoma. A prolactinoma is generally associated with prolactin levels >2000 mU/L. Morbid obesity may be associated with hypogonadotrophic hypogonadism, although secondary hypothyroidism is unlikely. Pituitary apoplexy is characterized by a sudden onset of headache, visual symptoms, altered mental status, and hormonal dysfunction due to acute hemorrhage or infarction of a pituitary gland. It usually presents with biochemical features of hypopituitarism which is not presented in this scenario. Primary hypothyroidism is characterized by low FT4, low FT3 and high TSH.

**>>DESCRIPTION**: A 45-year-old male with prior head trauma presents with low libido and fatigue. BMI 42. Labs: Low FT4, normal TSH, low FSH/LH/Testosterone, elevated Prolactin.

**>>OPTIONS**: a) Microprolactinoma b) Morbid obesity c) Non-functioning pituitary adenoma d) Post traumatic pituitary apoplexy e) Primary hypothyroidism

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Non-functioning pituitary adenoma

**>>REASONING**: Elevated prolactin with secondary hypothyroidism and hypogonadism suggests stalk compression, consistent with a non-functioning pituitary adenoma. Prolactinomas typically have much higher prolactin levels. Pituitary apoplexy presents acutely with hypopituitarism. Primary hypothyroidism would have high TSH.

## Question #:127

**CLINICAL SCENERIO**: A 32-year-old pregnant woman (26 weeks) presents to her GP because she has been feeling increasingly tired. She is normally fit and well and has had one previous pregnancy which was uneventful. A fasting blood glucose is 5.4 mmol/L. Urine dip is negative and her blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Blood results are as follows:

| Hb | 116 g/L | Male: (135-180) Female: (115 - 160) |
| --- | --- | --- |
| Platelets | 270 \* 10 9 /L | (150 - 400) |
| WBC | 7.3 \* 10 9 /L | (4.0 - 11.0) |

| Na + | 136 mmol/L | (135 - 145) |
| --- | --- | --- |
| K + | 3.9 mmol/L | (3.5 - 5.0) |
| Bicarbonat e | 25 mmol/L | (22 - 29) |
| Urea | 6.5 mmol/L | (2.0 - 7.0) |
| Creatinine | 89 µmol/L | (55 - 120) |

| Bilirubin | 4 µmol/L | (3 - 17) |
| --- | --- | --- |
| ALP | 170 u/L | (30 - 100) |
| ALT | 20 u/L | (3 - 40) |
| Î³GT | 55 u/L | (8 - 60) |
| Albumi n | 37 g/L | (35 - 50) |

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: - a) Acute cholecystitis - b) Gestational diabetes - c) Intrahepatic cholestasis of pregnancy - d) Non-alcoholic fatty liver diseas - e) Normal pregnancy

**CORRECT-CHOICE LINE**: Normal pregnancy can cause a raised ALP - it doesn’t necessarily imply liver problems

**REASONING**: The only abnormal finding is a slightly raised ALP. ALP is found in bone, liver, kidney, and intestinal and placental tissue, so ALP tends to rise during pregnancy. In pregnant women levels of up to twice that upper limit can still be normal.

Acute cholecystitis would usually present with abdominal symptoms and right upper quadrant tenderness. It is important to bear in mind that the risk of gallstones increases during pregnancy, as oestrogen can lead to higher cholesterol levels in bile.

Diagnosis (according to NICE) can be made based on either a fasting plasma glucose level of 5.6 mmol/L or above, or a 2-hour plasma glucose level of 7.8 mmol/L or above. Her fasting plasma glucose is below the threshold.

Intrahepatic cholestasis of pregnancy typically presents with itching (particularly in palms and soles) and can lead to complications for both mother and baby if untreated. LFTs are usually deranged but may be normal on some occasions.

Non-alcoholic fatty liver disease (NAFLD) is excessive fat build-up in the liver without another clear cause. NAFLD can progress to non-alcoholic steatohepatitis (NASH). Elevated liver enzymes are common in both but would not typically present with just an isolated rise in ALP. There is also no reason for this young fit woman to have NAFLD.

**>>DESCRIPTION**: A 32-year-old pregnant woman (26 weeks) presents with increasing tiredness. Fasting blood glucose is 5.4 mmol/L, urine dip is negative, and blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Labs show slightly elevated ALP (170 u/L), with all other values within normal limits.

**>>OPTIONS**: a) Acute cholecystitis b) Gestational diabetes c) Intrahepatic cholestasis of pregnancy d) Non-alcoholic fatty liver disease e) Normal pregnancy

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Normal pregnancy

**>>REASONING**: The most likely diagnosis is normal pregnancy, as ALP can rise during pregnancy up to twice the upper limit. Acute cholecystitis presents with abdominal pain, gestational diabetes requires a fasting glucose ≥5.6 mmol/L, intrahepatic cholestasis presents with itching, and NAFLD typically has more significantly elevated liver enzymes.

## Question #:135

**CLINICAL SCENERIO**: A 32-year-old pregnant woman (26 weeks) presents to her GP because she has been feeling increasingly tired. She is normally fit and well and has had one previous pregnancy which was uneventful. A fasting blood glucose is 5.4 mmol/L. Urine dip is negative and her blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Blood results are as follows:

| Hb | 116 g/L | Male: (135-180) Female: (115 - 160) |
| --- | --- | --- |
| Platelets | 270 \* 10 9 /L | (150 - 400) |
| WBC | 7.3 \* 10 9 /L | (4.0 - 11.0) |

| Na + | 136 mmol/L | (135 - 145) |
| --- | --- | --- |
| K + | 3.9 mmol/L | (3.5 - 5.0) |
| Bicarbonat e | 25 mmol/L | (22 - 29) |
| Urea | 6.5 mmol/L | (2.0 - 7.0) |
| Creatinine | 89 µmol/L | (55 - 120) |

| ALP | 170 u/L | (30 - 100) |
| --- | --- | --- |
| ALT | 20 u/L | (3 - 40) |
| Î³GT | 55 u/L | (8 - 60) |
| Albumi n | 37 g/L | (35 - 50) |

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: - a) Acute cholecystitis - b) Gestational diabetes - c) Intrahepatic cholestasis of pregnancy - d) Non-alcoholic fatty liver disease - e) Normal pregnancy

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Normal pregnancy can cause a raised ALP - it doesn’t necessarily imply liver problems

The only abnormal finding is a slightly raised ALP. ALP is found in bone, liver, kidney, and intestinal and placental tissue, so ALP tends to rise during pregnancy. In pregnant women levels of up to twice that upper limit can still be normal.

Acute cholecystitis would usually present with abdominal symptoms and right upper quadrant tenderness. It is important to bear in mind that the risk of gallstones increases during pregnancy, as oestrogen can lead to higher cholesterol levels in bile.

Diagnosis (according to NICE) can be made based on either a fasting plasma glucose level of 5.6 mmol/L or above, or a 2-hour plasma glucose level of 7.8 mmol/L or above. Her fasting plasma glucose is below the threshold.

Intrahepatic cholestasis of pregnancy typically presents with itching (particularly in palms and soles) and can lead to complications for both mother and baby if untreated. LFTs are usually deranged but may be normal on some occasions.

Non-alcoholic fatty liver disease (NAFLD) is excessive fat build-up in the liver without another clear cause. NAFLD can progress to non-alcoholic steatohepatitis (NASH). Elevated liver enzymes are common in both but would not typically present with just an isolated rise in ALP. There is also no reason for this young fit woman to have NAFLD.

**>>DESCRIPTION**: A 32-year-old pregnant woman (26 weeks) presents with increasing tiredness. She has a normal uneventful previous pregnancy. Fasting blood glucose is 5.4 mmol/L, urine dip is negative, and blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Labs show slightly raised ALP of 170 u/L, with all other values within normal limits.

**>>OPTIONS**: a) Acute cholecystitis b) Gestational diabetes c) Intrahepatic cholestasis of pregnancy d) Non-alcoholic fatty liver disease e) Normal pregnancy

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Normal pregnancy

**>>REASONING**: The most likely diagnosis is normal pregnancy as slightly raised ALP is common due to placental production. Acute cholecystitis presents with abdominal pain. Gestational diabetes requires a fasting glucose >= 5.6 mmol/L. Intrahepatic cholestasis of pregnancy presents with itching. NAFLD presents with more significantly elevated liver enzymes.

## Question #:148

**CLINICAL SCENERIO**: A 32-year-old, 30-weeks pregnant woman presents with fatigue. Examination reveals a distended abdomen consistent with gestational age and fetal movements. Blood tests are provided.

**QUESTION LINE**: What is the most likely explanation for her symptoms?

**OPTIONS**: - a) Acute fatty liver of pregnancy - b) HELLP syndrome - c) Intrahepatic cholestasis of pregnancy - d) Normal pregnancy - e) Primary biliary cirrhosis

**CORRECT-CHOICE LINE**: d

**REASONING**: Normal pregnancy can cause a raised ALP - it doesn’t necessarily imply liver problems

Normal pregnancy is correct. This woman is 30 weeks pregnant and therefore fatigue is not an unexpected complaint. A raised ALP is common in late pregnancy and is usually of placental origin. In the absence of any other signs or symptoms suggesting underlying liver disease, the most likely explanation is that these findings are consistent with normal pregnancy.

HELLP syndrome is incorrect. This stands for haemolysis, elevated liver enzymes and low platelets. She does not have evidence of either haemolysis or low platelets. While fatigue is a possible symptom, typically other symptoms are also present including abdominal pain, nausea, vomiting and backache.

Intrahepatic cholestasis of pregnancy is incorrect. This is typically characterized by itch, which is absent in this case.

Primary biliary cirrhosis is incorrect. While this can cause fatigue and a raised ALP, the median age of diagnosis is 65 and it is relatively uncommon in young people. A placental cause of the raised ALP is far more likely.

Acute fatty liver of pregnancy is incorrect. This is typically characterized by nausea, vomiting and abdominal pain. The ALT level is typically in the 300-500 IU/L range rather than an isolated rise in ALP. The patient will appear unwell, jaundice is common and there may be disseminated intravascular coagulation.

**>>DESCRIPTION**: A 32-year-old, 30-weeks pregnant woman presents with fatigue and a distended abdomen. Relevant blood test results are provided.

**>>OPTIONS**: a) Acute fatty liver of pregnancy b) HELLP syndrome c) Intrahepatic cholestasis of pregnancy d) Normal pregnancy e) Primary biliary cirrhosis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Normal pregnancy

**>>REASONING**: The most likely explanation is normal pregnancy, as fatigue is common, and the raised ALP is likely of placental origin. HELLP syndrome, intrahepatic cholestasis, primary biliary cirrhosis, and acute fatty liver of pregnancy are less likely due to the absence of key clinical or laboratory findings.

## Question #:144

**CLINICAL SCENERIO**: A 65-year-old woman presents with enlarging hands and feet, facial changes, sweating, and arthralgia for 12 months. Examination reveals coarsened facial features, macroglossia, bitemporal hemianopia, and large hands/feet. Blood tests are within normal limits except for an elevated Hb. MRI reveals a pituitary macroadenoma, and an oral glucose tolerance test shows failure to suppress growth hormone. Post-surgery, symptoms persist with elevated IGF-1 and persistent growth hormone suppression failure.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate management at this point?

**OPTIONS**: a) Bromocriptine b) Cabergoline c) Octreotide d) Pegvisomant e) Radiotherapy

**CORRECT-CHOICE LINE**: c

**REASONING**: Octreotide is the correct answer. This patient has acromegaly, and octreotide may be used in cases that are not suitable for trans-sphenoidal surgery. Radiotherapy is incorrect, It is reserved for cases that are refractory to medical management. Cabergoline and Bromocriptine is incorrect since it is less effective than octreotide. Pegvisomant is a second line option.

**>>DESCRIPTION**: A 65-year-old woman with acromegaly, treated with surgery, presents with persistent symptoms and elevated IGF-1. What is the most appropriate management?

**>>OPTIONS**: a) Bromocriptine b) Cabergoline c) Octreotide d) Pegvisomant e) Radiotherapy

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Octreotide

**>>REASONING**: Octreotide is the first-line medical treatment for persistent acromegaly after surgery. Other options are less effective or reserved for later-line treatment or specific circumstances.

## Question #:133

**CLINICAL SCENERIO**: A 40-year-old school teacher presented with a 2-month history of polyuria and elevated blood glucose levels. No family history of diabetes, not on medication. BMI 22 kg/m2, normal examination. Urine negative for ketones. Blood glucose 16.5 mmol/l, pH 7.40, HCO3 25 mmol/l, Na+ 140 mmol/l, K+ 3.7 mmol/l.

**QUESTION LINE**: Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

**OPTIONS**: a) Oral glucose tolerance test b) Serum ferritin and total iron binding capacity c) Mitochondrial gene mutation (A3243G) d) Anti-GAD antibody e) Toxicology screen

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of T2DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies)

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of type 2 DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies). Most of these patients are in the age group of 30-50 years, may or may not have a normal BMI and may present with elevated blood glucose levels. These patients can be managed initially with oral hypoglycaemic agents, although the β-cell function may decline over months to a few years, necessitating a relatively early requirement for insulin use.

A 25-year-old woman is referred urgently to the endocrinology clinic. She became pregnant three weeks ago. She had been suffering from diarrhea for the last month, and despite taking the oral contraceptive pill, became pregnant. She went to see her GP who requested blood tests as well as organizing an antenatal referral. Her full blood count, renal profile and liver function tests are all normal, but her TSH is undetectable and her free T4 levels are 52pmol/l. By the time of referral she has also had thyroid-stimulating hormone receptor antibodies which are positive. She has no past medical history. What is the most appropriate management for her hyperthyroidism?

* 1. Hemithyroidectomy
  2. Carbimazole
  3. Propylthiouracil
  4. Propranolol
  5. Total thyroidectomy

## Correct answer is c.

In pregnant woman who develop hyperthyroidism in the first trimester, propylthiouracil is preferred over carbimazole due to lower risk of foetal malformation

This patient has a new diagnosis of Graves’ disease with positive thyroidstimulating hormone receptor antibodies whilst pregnant in the first trimester. The most appropriate medical treatment is propylthiouracil. This has a lower risk of foetal malformation than carbimazole in the first trimester and would, therefore, be first-line treatment. Radioiodine is contra-indicated in pregnancy, whilst propranolol would not address the underlying problem. Surgery can be an option to patients who are resistant to propylthiouracil, and would need to happy at an urgent basis. Of operations, a hemithyroidectomy is more appropriate in trying to obtain histological samples in suspicious histology, whilst a total thyroidectomy would be a definitive management. Urgent treatment is necessary as thyroid-stimulating hormone receptor antibodies cross the placenta and can cause foetal problems.

**>>DESCRIPTION**: 40-year-old with 2-month history of polyuria, elevated blood glucose (16.5 mmol/l), normal BMI, negative urine ketones. No family history or medications. pH 7.40, HCO3 25 mmol/l, Na+ 140 mmol/l, K+ 3.7 mmol/l.

**>>OPTIONS**: a) Anti-GAD antibody b) Mitochondrial gene mutation (A3243G) c) Oral glucose tolerance test d) Serum ferritin and total iron binding capacity e) Toxicology screen

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Oral glucose tolerance test

**>>REASONING**: The patient presents with Graves’ disease and hyperthyroidism in the first trimester of pregnancy. Propylthiouracil is preferred over carbimazole due to a lower risk of fetal malformation. Propranolol doesn’t address the underlying issue, and surgery is reserved for propylthiouracil-resistant cases.

## Question #:175

**CLINICAL SCENERIO**: A 31-year-old white woman, 10 weeks pregnant with a BMI of 28.7, seeks advice on gestational diabetes. Her history includes a miscarriage, a previous term pregnancy with a 4.6kg birth weight, and a family history of type 1 diabetes. What is the most appropriate testing regime?

**QUESTION LINE**: What is the most appropriate testing regime for ruling out gestational diabetes in this woman?

**OPTIONS**: 1. Oral glucose tolerance test at 24-28 weeks pregnant5 2. None - as she has no risk factors for gestational diabetes 3. Oral glucose tolerance test at 12-14 weeks pregnant 4. Self-monitoring of sugars and repeat appointment in 2 weeks 5. HBa1c

**CORRECT-CHOICE LINE**: a.

**REASONING**: This question requires knowledge on the risk factors for developing gestational diabetes and appropriate testing based on risk:

Risk factors for gestational diabetes include:

*  BMI >30kg/m²
*  Previous delivery of a baby over 4.5kg - which qualifies this patient
*  Previous gestational diabetes
*  Family history of diabetes (1st degree relative)
*  Minority ethnic family origin with a high prevalence of diabetes

If any one of these risk factors is present then one should offer testing for gestational diabetes. The gold standard testing for patients with risk factors is 2hour 75g oral glucose tolerance test (OGTT) at 24-28 weeks gestation. If the patient has had gestational diabetes in a previous pregnancy then early-self monitoring of blood glucose or OGTT as soon as possible after booking could also be used for diagnosis.

A diagnosis of gestational diabetes is made if the patient has either:

*  Fasting glucose of 5.6 mmol/L or above OR
*  A 2-h plasma glucose of 7.8 mmol/L or above

**>>DESCRIPTION**: A 31-year-old pregnant woman with a BMI of 28.7, a history of a previous macrosomic infant (4.6kg), and a family history of diabetes presents for advice on gestational diabetes. Which is the best testing strategy?

**>>OPTIONS**: 1. HBa1c 2. None - as she has no risk factors for gestational diabetes 3. Oral glucose tolerance test at 12-14 weeks pregnant 4. Oral glucose tolerance test at 24-28 weeks pregnant5 5. Self-monitoring of sugars and repeat appointment in 2 weeks

**>>CORRECT-CHOICE LINE**: 4

**>>CORRECT-CHOICE\_TEXT**: Oral glucose tolerance test at 24-28 weeks pregnant5

**>>REASONING**: The correct answer is the oral glucose tolerance test (OGTT) at 24-28 weeks. This is the standard test for patients with risk factors for gestational diabetes, such as a history of macrosomia and a family history of diabetes. Other options are incorrect as they are not the standard of care for this clinical scenario.

## Question #:323

**CLINICAL SCENERIO**: A 47-year-old builder presents with bilateral hand paraesthesia, worse at night, and hand swelling. He reports hand weakness at work, urinary frequency, fatigue, and increased thirst over six months. He has a history of obesity, hypertension, and a family history of type II diabetes. Examination reveals thumb abduction weakness, diminished sensation in the radial digits, and positive wrist percussion for paraesthesia. He has axillary pigmentation, abdominal striae, a protuberant abdomen, and an elevated BMI.

**QUESTION LINE**: Which of the following investigations is most likely to be diagnostic?

**OPTIONS**: a) Magnetic Resonance Imaging of the Pituitary and visual field testing b) Fasting glucose on three occasions, glycosylated haemoglobin (HbA1c) and a 9am cortisol measurement c) Growth hormone measurement and dexamethasone suppression test d) Nerve conduction studies and electromyogram (EMG) e) Oral glucose tolerance test with serum glucose, IGF-1 and growth hormone measurements

**CORRECT-CHOICE LINE**: e

**REASONING**: The features in the question which point to the diagnosis of acromegaly include the presence of a recent increase in hand size and known complications of acromegaly such as diabetes mellitus (polyuria and polydipsia), acanthosis nigricans (pigmentation in the axillae) and carpal tunnel syndrome. The diagnosis of acromegaly is made when there is a failure to suppress the release of growth hormone during an oral glucose tolerance test. A fasting glucose and HbA1c measurement may aid the diagnosis of diabetes mellitus but not acromegaly.

**>>DESCRIPTION**: A 47-year-old builder presents with bilateral hand paraesthesia, worse at night, hand swelling, hand weakness at work, urinary frequency, fatigue, increased thirst, axillary pigmentation, abdominal striae, protuberant abdomen, and elevated BMI. He has a history of obesity, hypertension, and a family history of type II diabetes.

**>>OPTIONS**: a) Fasting glucose on three occasions, glycosylated haemoglobin (HbA1c) and a 9am cortisol measurement b) Growth hormone measurement and dexamethasone suppression test c) Magnetic Resonance Imaging of the Pituitary and visual field testing d) Nerve conduction studies and electromyogram (EMG) e) Oral glucose tolerance test with serum glucose, IGF-1 and growth hormone measurements

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Oral glucose tolerance test with serum glucose, IGF-1 and growth hormone measurements

**>>REASONING**: The most likely diagnostic investigation is an oral glucose tolerance test with serum glucose, IGF-1, and growth hormone measurements, as it assesses for acromegaly (failure to suppress growth hormone). Other options are less relevant, as they either do not diagnose acromegaly or do not address the underlying cause.

## Question #:240

**CLINICAL SCENERIO**: A 19-year-old with asthma presents to the ED with leg weakness and inability to walk after a marathon. Exam shows bilateral leg extensor weakness, normal tone/reflexes/coordination, and downgoing plantars. Straight leg raise and sensation are normal. Blood tests show hypokalemia (K+ 2.9mmol/l).

**QUESTION LINE**: What is the most appropriate treatment in this case?

**OPTIONS**: a. Oral potassium and encourage bed rest b. Hourly forced vital capacity measurements and plasma exchange c. Oral potassium and encourage gentle exercise d. Hourly forced vital capacity measurements and IV immunoglobulin e. Plasma exchange and oral potassium supplementation

**CORRECT-CHOICE LINE**: C

**REASONING**: Hypokalemic periodic paralysis, often triggered by exercise and high carbohydrate intake, presents with weakness and hypokalemia. Treatment involves oral potassium supplementation and gentle exercise. Intravenous potassium is for severe cases with arrhythmias or inability to swallow. Acetazolamide or dichlorphenamide are used prophylactically. Incorrect options: Bed rest is not the primary treatment; forced vital capacity measurements and plasma exchange/IVIG are not indicated; plasma exchange is not used.

**>>DESCRIPTION**: A 19-year-old asthmatic presents with post-marathon leg weakness and hypokalemia (K+ 2.9mmol/l).

**>>OPTIONS**: a. Hourly forced vital capacity measurements and IV immunoglobulin b. Hourly forced vital capacity measurements and plasma exchange c. Oral potassium and encourage bed rest d. Oral potassium and encourage gentle exercise e. Plasma exchange and oral potassium supplementation

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Oral potassium and encourage gentle exercise

**>>REASONING**: The best treatment for hypokalemic periodic paralysis, given the patient’s presentation and potassium level, is oral potassium and gentle exercise. Intravenous potassium is used for serious cases and is not preferred over oral potassium if the patient can swallow and doesn’t have arrhythmias.

## Question #:80

**CLINICAL SCENERIO**: A 65-year-old retired nurse was referred to the endocrine clinic by her primary care physician in view of incidentally-detected hypercalcaemia. She had no significant past medical history and was not taking any regular medications. Her general physical and systemic examination was unremarkable.

Urea

7.5 mg/dl

Calcium

2.8 mmol/l

Phosphate

0.74mmol/l

Creatinine

98µmol/l

Alkaline

450IU/l

phosphatase

**QUESTION LINE**: Which one of the following investigations is most likely to help establish the diagnosis?

**OPTIONS**: a) 1,25-OH vitamin D levels b) PTH level c) 25-OH vitamin D levels d) Myeloma screen e) Sestamibi scan

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Measuring PTH level is the first step in investigating hypercalcaemia

The most likely cause of hypercalcaemia in this case is primary hyperparathyroidism. The annual incidence is approximately 1 in 1000 and is commonly seen in post-menopausal women. The underlying aetiology of primary hyperparathyroidism is parathyroid adenoma (80-85%) followed by parathyroid hyperplasia (~10-14%). Measuring PTH level is the first step in investigating hypercalcaemia.

**>>DESCRIPTION**: A 65-year-old woman with incidentally-detected hypercalcaemia, normal exam, and no significant history/medications. Labs show elevated calcium, normal creatinine, and elevated alkaline phosphatase.

**>>OPTIONS**: a) 1,25-OH vitamin D levels b) 25-OH vitamin D levels c) Myeloma screen d) PTH level e) Sestamibi scan

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: PTH level

**>>REASONING**: Measuring PTH level is the first step to investigate hypercalcemia, as primary hyperparathyroidism is the most likely cause.

## Question #:233

**CLINICAL SCENERIO**: A 25-year-old woman is concerned about her father’s history of tumors and potential genetic risk for herself, prompting a referral to the endocrinology clinic.

**QUESTION LINE**: Given the likely underlying diagnosis, which of the following is most likely to be abnormal?

**OPTIONS**: a) Cortisol - b) Fasting glucose - c) Parathyroid hormone - d) Prolactin - e) Thyroid stimulating hormone

**CORRECT-CHOICE LINE**: c

**REASONING**: This lady’s father is likely to have multiple endocrine neoplasia type 1 (MEN1) as evidenced by 2 MEN1 associated tumours, a pituitary adenoma and a gastrinoma.

Although this lady is asymptomatic, it is important she is offered genetic screening, as the condition is autosomal dominant.

Fasting glucose, parathyroid hormone and prolactin are all biochemical screening tests used for MEN1 associated tumours (insulinoma, parathyroid adenoma and pituitary adenoma respectively). However, hyperparathyroidism is by far the most common initial manifestation and will eventually develop in 90% of MEN1 patients.

ReferencThakker et al. Clinical guidelines for multiple endocrine neoplasia type 1 (MEN1). J Clin Endocrinol Metab. 2012:97(9)2990-3011)

**>>DESCRIPTION**: A 25-year-old woman presents to the endocrinology clinic due to her father’s history of tumors and potential genetic risk for herself.

**>>OPTIONS**: a) Cortisol b) Fasting glucose c) Parathyroid hormone d) Prolactin e) Thyroid stimulating hormone

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Parathyroid hormone

**>>REASONING**: The most likely abnormality in this case of suspected Multiple Endocrine Neoplasia type 1 (MEN1) is elevated parathyroid hormone, as hyperparathyroidism is the most common initial manifestation.

## Question #:241

**CLINICAL SCENERIO**: A 56-year-old lady presents with a 3 month history of abdominal pains, low mood and constipation. Past medical history includes hypertension and depression following the death of her husband 2 years ago. Routine blood tests are performed by the GP and upon review the patient is referred into hospital. Blood tests are as below: Hb 100 g/l, Platelets 230 \* 109/l, WBC 10 \* 109/l, Calcium (adjusted) 2.96 mmol/l, Phosphate 1.35 mmol/l, Albumin 35 g/L, Na+ 135 mmol/l, K+ 4.7 mmol/l, Urea 6 mmol/l, Creatinine 110 µmol/l, CRP 30 mg/l.

**QUESTION LINE**: Which diagnostic test should be performed first?

**OPTIONS**: - a) Parathyroid hormone level - b) Myeloma screen - c) CT chest, abdomen and pelvis - d) Urinary calcium levels - e) Skeletal X-ray

**CORRECT-CHOICE LINE**: A

**REASONING**: The two main causes of hypercalcaemia are primary hyperparathyroidism and malignancy. Parathyroid hormone level will help to differentiate between these two main differentials and help guide further investigations

**>>DESCRIPTION**: A 56-year-old woman with a 3-month history of abdominal pain, low mood, and constipation, with a history of hypertension and depression, presents with blood test results: Calcium (adjusted) 2.96 mmol/l. Which diagnostic test should be performed first?

**>>OPTIONS**: a) CT chest, abdomen and pelvis b) Myeloma screen c) Parathyroid hormone level d) Skeletal X-ray e) Urinary calcium levels

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Parathyroid hormone level

**>>REASONING**: The primary causes of hypercalcemia are hyperparathyroidism and malignancy. Parathyroid hormone level helps differentiate these, guiding further investigations.

## Question #:217

**CLINICAL SCENERIO**: A 45-year-old man presents for review after renal colic due to nephrolithiasis. Initial management included IV fluids, analgesia, and an alpha-blocker. Relevant blood tests are provided, including elevated corrected calcium and parathyroid hormone.

**QUESTION LINE**: How should he be further managed?

**OPTIONS**: - a) Annual monitoring of calcium and renal function - b) Encourage oral fluids - c) Bisphosphonates - d) Vitamin D supplementation - e) Parathyroidectomy

**CORRECT-CHOICE LINE**: e

**REASONING**: The correct answer is parathyroidectomy. This is a patient who has developed renal colic secondary to likely primary hyperparathyroidism, as is suggested by his hypercalcaemia and elevated parathyroid hormone. The mainstay of management of primary hyperparathyroidism is parathyroidectomy, but cases have to be appropriately identified as surgical candidates. This patient developed renal stones as a likely complication and therefore would benefit from surgery. If the blood tests been an incidental finding, then monitoring and oral fluids both would have been more appropriate.

**>>DESCRIPTION**: A 45-year-old man with a history of nephrolithiasis and hypercalcemia with elevated PTH requires further management.

**>>OPTIONS**: a) Annual monitoring of calcium and renal function b) Bisphosphonates c) Encourage oral fluids d) Parathyroidectomy e) Vitamin D supplementation

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Parathyroidectomy

**>>REASONING**: The correct answer is parathyroidectomy. The patient has primary hyperparathyroidism (high calcium and PTH) with renal stones. Parathyroidectomy is the primary treatment for hyperparathyroidism, especially in patients with complications such as kidney stones. Annual monitoring and oral fluids are less appropriate.

## Question #:147

**CLINICAL SCENERIO**: An 82-year-old female with a 4-month history of urinary incontinence, previously continent, presents with incontinence when laughing/coughing and sudden urges to urinate, impacting sleep. She has hypertension and angina. She has reduced caffeine intake and started bladder training.

**QUESTION LINE**: What additional management would you commence?

**OPTIONS**: a) Pelvic floor exercises only b) Pelvic floor exercises and tolterodine c) Pelvic floor exercises and duloxetine d) Pelvic floor exercises and desmopressin e) Long term urinary catheter

**CORRECT-CHOICE LINE**: b

**REASONING**: The question concentrates on the management of an elderly patient with mixed stress incontinence and overactive bladder syndrome. In the context of mixed urinary incontinence, pharmacological therapies should be offered in addition to conservative therapies such as pelvic floor exercises. A long-term catheter in whom persistent incontinence causes skin wounds, the patient requires such significant care that continuous changing of bed linen and clothes would be required, chronic retention with a risk of renal impairment (and inability to selfcatheterise) or if the patient opts for the catheterisation. The 2013 NICE guidelines recommends first line pharmacological treatment for mixed incontinence or overactive bladder syndrome alone is either tolterodine, oxybutynin or darifenacin1. Duloxetine should only be used if the patient does not tolerate first line therapy and is not a candidate for surgery. Desmopressin can reduce nocturia. However, its effects in platelet activation contradicts its use in ischaemic heart disease.

**>>DESCRIPTION**: An 82-year-old female presents with mixed urinary incontinence (stress and urge) for four months, impacting sleep. She has hypertension and angina and has tried conservative management.

**>>OPTIONS**: a) Pelvic floor exercises and desmopressin b) Pelvic floor exercises and duloxetine c) Pelvic floor exercises and tolterodine d) Pelvic floor exercises only e) Long term urinary catheter

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Pelvic floor exercises and tolterodine

**>>REASONING**: The best management includes pelvic floor exercises and tolterodine, according to NICE guidelines, for mixed urinary incontinence or overactive bladder syndrome. Other options are less appropriate: catheters are for specific severe cases; duloxetine is a second-line therapy; and desmopressin is inappropriate given the patient’s angina.

## Question #:289

**CLINICAL SCENERIO**: A 69-year-old woman reports involuntary urine leakage throughout the day while dressing, without sudden urgency.

**QUESTION LINE**: What is the most appropriate first-line management?

**OPTIONS**: a) Bladder retraining b) Mirabegron c) Oxybutynin d) Pelvic floor muscle training e) Tamsulosin

**CORRECT-CHOICE LINE**: D

**REASONING**: Urinary incontinence - first-line treatment:

*  urge incontinence: bladder retraining
*  stress incontinence: pelvic floor muscle training

This woman is presenting with stress incontinence, a leaking of urine normally triggered by coughing, laughing, or sneezing. Although often associated with pelvic floor damage for example during childbirth, it can also present as a result of age-related changes. The first-line management for stress incontinence is pelvic floor muscle training which is the correct answer here.

Bladder retraining is useful in cases of urge incontinence however this patient denies any history of urinary urgency. Urge incontinence occurs due to an overactive bladder. Bladder retraining aims to regain control over the function of the bladder. As stress incontinence is related to weakened pelvic floor muscles, bladder retraining would not be as effective as pelvic floor muscle training in this case.

Mirabegron is a beta-3 agonist that relaxes the detrusor muscle of the bladder, increasing bladder capacity. As the primary problem with stress incontinence is related to the pelvic floor muscles, mirabegron would not be the first-line treatment in this case.

Oxybutynin is an antimuscarinic that is also prescribed for an overactive bladder. Again, this relaxes the detrusor muscles but as the primary pathology in stress incontinence is related to the pelvic floor muscles, this would not be the first-line management step here.

Tamsulosin is an alpha-1 antagonist which relaxes the smooth muscle in the bladder neck and urethra. This is useful in the treatment of overflow incontinence as a result of urethral obstruction for example from benign prostatic hyperplasia. It has no effect on the pelvic floor muscles which are weakened in stress incontinence and so has no role here.

**>>DESCRIPTION**: A 69-year-old woman presents with involuntary urine leakage throughout the day while dressing, without urinary urgency.

**>>OPTIONS**: a) Bladder retraining b) Mirabegron c) Oxybutynin d) Pelvic floor muscle training e) Tamsulosin

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Pelvic floor muscle training

**>>REASONING**: The correct answer is pelvic floor muscle training, which is the first-line treatment for stress incontinence. The patient’s symptoms align with stress incontinence. Other options like bladder retraining, mirabegron, oxybutynin, and tamsulosin are inappropriate as they address other types of incontinence or have no effect on the pelvic floor.

## Question #:185

**CLINICAL SCENERIO**: A 27-year-old woman, 11 weeks pregnant, is reviewed. This is her second pregnancy. She had gestational diabetes in her first pregnancy, which resolved postpartum.

**QUESTION LINE**: What is the most appropriate management at this stage?

**OPTIONS**: - a) Perform an oral glucose tolerance test - b) Advise on a diabetic diet and start metformin at 20 weeks - c) Arrange a fasting glucose - d) Arrange a HbA1c test - e) Advise on a diabetic diet and start insulin at 20 weeks

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: The most appropriate management at this stage is to perform an oral glucose tolerance test (OGTT). Women with a history of gestational diabetes are at increased risk of developing diabetes in subsequent pregnancies. Early screening with an OGTT allows for early diagnosis and management. Fasting glucose and HbA1c may be used for screening, but OGTT is the gold standard. Dietary advice and medication are indicated if the OGTT is abnormal.

**>>DESCRIPTION**: A 27-year-old, 11 weeks pregnant woman with a history of gestational diabetes in a prior pregnancy presents for review. What is the best next step?

**>>OPTIONS**: a) Advise on a diabetic diet and start insulin at 20 weeks b) Advise on a diabetic diet and start metformin at 20 weeks c) Arrange a fasting glucose d) Arrange a HbA1c test e) Perform an oral glucose tolerance test

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Perform an oral glucose tolerance test

**>>REASONING**: An oral glucose tolerance test (OGTT) is the most appropriate initial step for women with a history of gestational diabetes in a prior pregnancy. This allows for early diagnosis. Fasting glucose, HbA1c, diet, and medication are appropriate subsequent interventions if the OGTT is abnormal.

## Question #:52

**CLINICAL SCENERIO**: A 35-year-old woman presents with a three-month history of headaches, palpitations, mild constipation, and urinary frequency. She is persistently hypertensive despite treatment with lisinopril, amlodipine, and indapamide at maximum doses. Respiratory rate is 18/min, oxygen saturation is 99% on room air, heart rate is 115bpm, blood pressure is 187/98mmHg, temperature is 36.2 o C and GCS is 15/15. ECG shows sinus tachycardia. Blood tests reveal Hb 140 g/L, platelets 387 \* 10 9 /L, WBC 9.0 \* 10 9 /L, Calcium 2.80 mmol/L, Phosphate 1.2 mmol/L, Magnesium 0.8 mmol/L, TSH 5.0 mU/L, Free T4 12 pmol/L, and Parathyroid hormone 52 pg/mL.

**QUESTION LINE**: Given the likely diagnosis, what is the most appropriate blood pressure treatment?

**OPTIONS**: a) Atenolol - b) Bisoprolol - c) Nicorandil - d) Phenoxybenzamine - e) Spironolactone

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: PH aeochromocytoma - give PH enoxybenzamine before beta-blockers

This patient has symptoms of hypercalcaemia (constipation, urinary frequency), coupled with a mildly raised serum calcium level. The serum parathyroid hormone (PTH) level is at the upper end of normal - this is suggestive of primary hyperparathyroidism. This patient has several cardinal symptoms of a phaeochromocytoma (headache, palpitations, hypertension). This patient is also likely to have a phaeochromocytoma, as the combination of primary hyperparathyroidism (secondary to a parathyroid adenoma or parathyroid hyperplasia) and a phaeochromocytoma is in keeping with multiple endocrine neoplasia type 2 (MEN-2A). The patient will require cross-sectional imaging to localise the phaeochromocytoma. Treatment should be initiated in the interim to control symptoms.

Phenoxybenzamine is the correct answer. Alpha-blockers should be initiated firstline. Initiation of beta-blockers before alpha-blockade will cause unopposed stimulation of alpha-receptors, causing vasoconstriction and hypertensive crisis.

Atenolol is the incorrect answer. Atenolol is a beta-blocker and will cause unopposed stimulation of alpha receptors.

Bisoprolol is the incorrect answer. Bisoprolol is another beta-blocker - like atenolol, it can risk causing a hypertensive crisis.

Nicorandil is the incorrect answer. Nicorandil is a treatment option for lowering blood pressure but is not specifically used to treat hypertension secondary to a phaeochromocytoma.

Spironolactone is the incorrect answer. Spironolactone is used to treat hypertension but as above, it is not a targeted treatment for hypertension due to a phaeochromocytoma.

**>>DESCRIPTION**: A 35-year-old woman with headaches, palpitations, constipation, and urinary frequency is persistently hypertensive despite maximum doses of lisinopril, amlodipine, and indapamide. BP is 187/98mmHg, HR is 115bpm and ECG shows sinus tachycardia. Labs: Calcium 2.80 mmol/L, PTH 52 pg/mL.

**>>OPTIONS**: a) Atenolol b) Bisoprolol c) Nicorandil d) Phenoxybenzamine e) Spironolactone

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Phenoxybenzamine

**>>REASONING**: The patient’s symptoms and lab results suggest hyperparathyroidism and possible pheochromocytoma (MEN-2A). Phenoxybenzamine (alpha-blocker) is the correct first-line treatment to prevent hypertensive crisis. Beta-blockers (Atenolol, Bisoprolol) are contraindicated as initial therapy and Nicorandil/Spironolactone are not targeted treatments for pheochromocytoma-induced hypertension.

## Question #:203

**CLINICAL SCENERIO**: A 35-year-old woman presents to the emergency department with headaches, sweating, and palpitations, triggered by coffee, alongside elevated heart rate and blood pressure.

**QUESTION LINE**: What is the most appropriate management?

**OPTIONS**: a) Glyceryl trinitrate intravenous infusion b) Labetalol intravenous bolus injection c) Labetalol intravenous infusion d) Phenoxybenzamine intravenous infusion e) Phenoxybenzamine orally

**CORRECT-CHOICE LINE**: e

**REASONING**: The combination of headache, tachycardia, and diaphoresis with hypertension suggests phaeochromocytoma. Phenoxybenzamine, an alpha-blocker, should be administered orally as the initial step. Beta-blockers are secondary, and intravenous options are less appropriate. Glyceryl trinitrate is for managing hypertension, but not the primary approach. Phenoxybenzamine is available only in oral form.

**>>DESCRIPTION**: A 35-year-old woman presents with headaches, sweating, palpitations, triggered by coffee, with elevated heart rate and blood pressure.

**>>OPTIONS**: a) Glyceryl trinitrate intravenous infusion b) Labetalol intravenous bolus injection c) Labetalol intravenous infusion d) Phenoxybenzamine intravenous infusion e) Phenoxybenzamine orally

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Phenoxybenzamine orally

**>>REASONING**: The clinical presentation suggests phaeochromocytoma, and the initial management involves an oral alpha-blocker, phenoxybenzamine. Beta-blockers are secondary. Intravenous options are less preferred, and glyceryl trinitrate is not the primary approach.

## Question #:6

**CLINICAL SCENERIO**: A 68-year-old patient with type 2 diabetes for 28 years reports painless macroscopic haematuria and is concerned about bladder cancer from his medications.

**QUESTION LINE**: Which of the following antiglycemic agents can cause bladder cancer?

**OPTIONS**: - a) Gliclazide - b) Tolbutamide - c) Pioglitazone - d) Insulin detemir - e) Sitagliptin

**CORRECT-CHOICE LINE**: Answer: Pioglitazone

**REASONING**: Thiazolidinediones are associated with an increased risk of bladder cancer Answer: Pioglitazone

Pioglitazone has been associated with an increased risk of bladder cancer. The greatest risk was shown in those patients who have used pioglitazone long term.

Risk of Bladder Cancer Among Diabetic Patients Treated With Pioglitazone Interim report of a longitudinal cohort study http://care.diabetesjournals.org/content/34/4/916

**>>DESCRIPTION**: A 68-year-old diabetic patient reports painless macroscopic haematuria and is concerned about bladder cancer related to his medications. Which of the following antiglycemic agents can cause bladder cancer?

**>>OPTIONS**: a) Gliclazide b) Insulin detemir c) Pioglitazone d) Sitagliptin e) Tolbutamide

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Pioglitazone

**>>REASONING**: Pioglitazone, a thiazolidinedione, is associated with an increased risk of bladder cancer, particularly with long-term use. The other options are not significantly linked to bladder cancer.

## Question #:74

**CLINICAL SCENERIO**: A 50-year-old obese gentleman presented to the emergency department with sweating, pallor, shortness of breath and central chest pain. He was diagnosed with a STEMI and underwent primary PCI. An ECHO after the event showed an EF of 35%. Following work up for his coronary artery disease he was diagnosed with type 2 diabetes. He was commenced on iv insulin which controlled his blood glucose in the interim.

**QUESTION LINE**: In the long term treatment of his diabetes which hypoglycaemic agent is best avoided?

**OPTIONS**: - a) Metformin - b) Pioglitazone - c) Gliclazide - d) Acarbose - e) Insulin glargine

**CORRECT-CHOICE LINE**: Pioglitazone - contraindicated by: heart failure

**REASONING**: One of the side effects of pioglitazone is fluid retention, therefore it is best avoided in a patient with a risk of heart failure like the above patient as it can precipitate or worsen heart failure due to fluid overload.

**>>DESCRIPTION**: A 50-year-old obese male with a history of STEMI, PCI, and an EF of 35% was diagnosed with type 2 diabetes and is currently managed with IV insulin.

**>>OPTIONS**: a) Acarbose b) Gliclazide c) Insulin glargine d) Metformin e) Pioglitazone

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Pioglitazone

**>>REASONING**: Pioglitazone is best avoided due to its side effect of fluid retention, which can worsen heart failure in patients with pre-existing cardiac conditions. Other options do not have such a direct contraindication.

## Question #:252

**CLINICAL SCENERIO**: A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus presents with non-visible haematuria during an annual review. Urine dipstick shows blood ++, no protein, and no leucocytes. Repeated one week later. Current medications: aspirin, bisoprolol, atorvastatin, ramipril, metformin, and pioglitazone.

**QUESTION LINE**: Which one of the following drugs should be stopped whilst awaiting further investigations?

**OPTIONS**: a. Aspirin b. Ramipril c. Atorvastatin d. Metformin e. Pioglitazone

**CORRECT-CHOICE LINE**: Pioglitazone

**REASONING**: Thiazolidinediones are associated with an increased risk of bladder cancer

The correct answer is Pioglitazone . Pioglitazone, an oral antidiabetic agent used in the management of type 2 diabetes mellitus, has been associated with an increased risk of bladder cancer. In the presence of non-visible haematuria (blood in urine), this medication should be discontinued until further investigations are completed.

Aspirin is not typically associated with haematuria and its use as antiplatelet therapy in a patient with ischemic heart disease should not be interrupted without a compelling reason.

Ramipril , an angiotensin-converting enzyme (ACE) inhibitor, is used for hypertension and cardiac conditions. While it can occasionally cause renal impairment leading to proteinuria, it does not typically cause haematuria.

Atorvastatin , a statin used for hyperlipidaemia and cardiovascular risk reduction, may have some side effects such as myopathy or liver dysfunction but it is not commonly associated with haematuria.

Finally, Metformin , another oral antidiabetic agent, can lead to lactic acidosis in cases of renal insufficiency but it does not usually cause haematuria. Therefore, there would be no need to stop metformin based on the given clinical scenario

**>>DESCRIPTION**: A 67-year-old man with ischemic heart disease and type 2 diabetes presents with non-visible haematuria. Current medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin, and pioglitazone.

**>>OPTIONS**: a. Aspirin b. Atorvastatin c. Metformin d. Pioglitazone e. Ramipril

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Pioglitazone

**>>REASONING**: Pioglitazone should be stopped due to its association with an increased risk of bladder cancer, especially in the context of haematuria. Aspirin, ramipril, atorvastatin, and metformin are not typically linked to haematuria.

## Question #:304

**CLINICAL SCENERIO**: A 65-year-old man with type 2 diabetes presents with macroscopic haematuria and an exophytic bladder lesion.

**QUESTION LINE**: Given the likely diagnosis, what is the patient’s most significant risk factor for the development of this condition?

**OPTIONS**: a. Ethnicity b. Threadworm infection c. Metformin d. Occupation e. Pioglitazone

**CORRECT-CHOICE LINE**: E

**REASONING**: Thiazolidinediones are associated with an increased risk of bladder cancer. Pioglitazone is correct. Macroscopic haematuria and an exophytic lesion identified on cystoscopy suggest bladder cancer. Thiazolidinediones are a well-recognized risk factor for the development of bladder cancer. Ethnicity is incorrect. Bladder cancer more commonly develops in those of white ethnicity rather than black. However, black patients are twice as likely to die from bladder cancer. Occupation is incorrect. Typically, those employed in industries such as textile, rubber, leather, dye, paint, and print are at increased risk of bladder cancer due to chemical exposures. Dairy farming is not a typical risk factor. Metformin is incorrect. The use of this medication is not associated with the development of bladder cancer. Threadworm infection is incorrect. This parasite is not associated with bladder cancer. The parasitic infection associated with the development of bladder cancer is schistosomiasis. This is endemic to various tropical and subtropical regions. It is not typically found in Jamaica.

**>>DESCRIPTION**: A 65-year-old man with type 2 diabetes presents with macroscopic haematuria and an exophytic bladder lesion.

**>>OPTIONS**: a. Ethnicity b. Metformin c. Occupation d. Pioglitazone e. Threadworm infection

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Pioglitazone

**>>REASONING**: Pioglitazone, a thiazolidinedione, is the most significant risk factor due to its association with bladder cancer. Other options are incorrect as they are not recognized risk factors for bladder cancer.

## Question #:314

**CLINICAL SCENERIO**: A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus has non-visible haematuria. Urine dipstick shows blood ++, no protein, and no leucocytes. Medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin, and pioglitazone.

**QUESTION LINE**: Which one of the following drugs should be stopped whilst awaiting further investigations?

**OPTIONS**: 1. Aspirin 2. Ramipril 3. Atorvastatin 4. Metformin 5. Pioglitazone

**CORRECT-CHOICE LINE**: Pioglitazone

**REASONING**: Pioglitazone is associated with an increased risk of bladder cancer. In the presence of haematuria, it should be stopped until further investigations. Aspirin, Ramipril, Atorvastatin and Metformin are not typically associated with haematuria.

**>>DESCRIPTION**: A 67-year-old man with a history of ischemic heart disease and type 2 diabetes mellitus presents with non-visible haematuria. His medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin, and pioglitazone.

**>>OPTIONS**: 1. Aspirin 2. Atorvastatin 3. Metformin 4. Pioglitazone 5. Ramipril

**>>CORRECT-CHOICE LINE**: 4

**>>CORRECT-CHOICE\_TEXT**: Pioglitazone

**>>REASONING**: Pioglitazone is associated with an increased risk of bladder cancer and should be discontinued with haematuria. Aspirin, ramipril, atorvastatin, and metformin are not directly linked to this presentation.

## Question #:110

**CLINICAL SCENERIO**: A 49-year-old man presents with feeling under the weather. On further questioning he reports that he has been feeling tired and weak for the past few weeks and his wife has noticed that he as lost some weight, although he states that his appetite has not decreased and if anything he is feeling more thirsty and going to the toilet several times a night. Over the last couple of days he has noticed a rash develop around his groin, which is now present on his buttocks. On examination, there are patches of red with irregular borders and crusting.

A fasting blood test is arranged and reveals a blood glucose of 9.2 mm/l.

**QUESTION LINE**: What is the next most appropriate investigation?

**OPTIONS**: a) Plasma insulin level b) Tissue transglutaminase antibody (TTA) test c) Plasma glucagon level d) Skin biopsy e) Plasma zinc level

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: This patient has a diagnosis of a glucagonoma. The polydipsia and polyuria, coupled with the fasting blood glucose result suggest diabetes and the rash is necrolytic migratory erythema a symptom which is the presenting problem in up to 70% of cases of glucagonoma.

**>>DESCRIPTION**: A 49-year-old man presents with fatigue, weakness, weight loss, increased thirst and frequent urination. He has a rash around his groin and buttocks with red, irregular, crusted patches. Fasting blood glucose is 9.2 mmol/l.

**>>OPTIONS**: a) Plasma glucagon level b) Plasma insulin level c) Plasma zinc level d) Skin biopsy e) Tissue transglutaminase antibody (TTA) test

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Plasma glucagon level

**>>REASONING**: The patient’s symptoms (polydipsia, polyuria, elevated blood glucose) and necrolytic migratory erythema suggest a glucagonoma. Therefore, the next appropriate investigation is Plasma glucagon level. Other options are not directly related to the suspected diagnosis.

## Question #:114

**CLINICAL SCENERIO**: A 49-year-old man presents with feeling under the weather. On further questioning he reports that he has been feeling tired and weak for the past few weeks and his wife has noticed that he as lost some weight, although he states that his appetite has not decreased and if anything he is feeling more thirsty and going to the toilet several times a night. Over the last couple of days he has noticed a rash develop around his groin, which is now present on his buttocks. On examination, there are patches of red with irregular borders and crusting.

A fasting blood test is arranged and reveals a blood glucose of 9.2 mm/l.

**QUESTION LINE**: What is the next most appropriate investigation?

**OPTIONS**: - a) Plasma insulin level - b) Tissue transglutaminase antibody (TTA) test - c) Plasma glucagon level - d) Skin biops - e) Plasma zinc level

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: This patient has a diagnosis of a glucagonoma. The polydipsia and polyuria, coupled with the fasting blood glucose result suggest diabetes and the rash is necrolytic migratory erythema a symptom which is the presenting problem in up to 70% of cases of glucagonoma.

**>>DESCRIPTION**: A 49-year-old man presents with fatigue, weakness, weight loss, increased thirst and frequent urination. He also has a rash around his groin and buttocks with red patches, irregular borders, and crusting. Fasting blood glucose is 9.2 mm/l.

**>>OPTIONS**: a) Plasma glucagon level b) Plasma insulin level c) Plasma zinc level d) Skin biops e) Tissue transglutaminase antibody (TTA) test

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Plasma glucagon level

**>>REASONING**: The patient’s symptoms (polydipsia, polyuria, elevated glucose) and necrolytic migratory erythema suggest a glucagonoma. Therefore, the next step is to check plasma glucagon level. The rash is the key symptom.

## Question #:168

**CLINICAL SCENERIO**: A 55-year-old male presents with a 2-month history of sweating, fatigue, and daytime tiredness. He attributes his tight wedding ring to fluid retention, experiencing worsening headaches and vision deterioration in the last 2 weeks. He has no past medical history and takes no medications.

**QUESTION LINE**: Which of the following findings would be in keeping with the above?

**OPTIONS**: 1. Homonymous hemianopia 2. Overbite 3. Positive Tinel’s sign 4. Reduced FVC on spirometry 5. Dysdiadochokinesia

**CORRECT-CHOICE LINE**: c.

**REASONING**: This is a case of acromegaly which is caused by excessive growth hormone secretion. In adults, the excess growth hormone secretion is usually caused by a benign growth hormone-secreting pituitary adenoma.

It is often associated with hypertension, diabetes, obstructive sleep apnoea, carpal tunnel syndrome, colonic polyps/malignancies, cardiomyopathies, and arrhythmias.

1. Homonymous hemianopia: this is false because the visual field disturbance associated with a pituitary tumour is bitemporal hemianopia
2. Overbite: patients with present with underbite, or prognathism
3. Positive Tinel’s sign: this is true in carpal tunnel syndrome
4. 4 and 5: these features are not characteristic of acromegaly

**>>DESCRIPTION**: A 55-year-old male presents with sweating, fatigue, and vision deterioration, with a tight wedding ring. What finding is consistent with this presentation?

**>>OPTIONS**: 1. Dysdiadochokinesia 2. Homonymous hemianopia 3. Overbite 4. Positive Tinel’s sign 5. Reduced FVC on spirometry

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Positive Tinel’s sign

**>>REASONING**: The presentation suggests acromegaly. The correct answer is positive Tinel’s sign, indicating carpal tunnel syndrome, a common finding in acromegaly. Homonymous hemianopia is incorrect, the vision disturbances are bitemporal. Overbite is also incorrect because patients present with underbite. Reduced FVC and dysdiadochokinesia are not characteristic.

## Question #:184

**CLINICAL SCENERIO**: A 34-year-old woman presents with heat intolerance, tremors, and diarrhea, with blood tests indicating thyrotoxicosis. Symptoms started 4 weeks ago, initially attributed to the stress of caring for her new baby, born 6 weeks prior. Examination showed a small, diffuse, mildly tender goiter and fine tremor, with lab results: TSH 0.1, T4 19.5, T3 8.1, thyroid peroxidase antibodies 250, and ESR 21. Thyroid scintiscanning showed no significant thyroid uptake.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Graves’ disease b) Toxic thyroid nodule c) Viral thyroiditis d) Toxic multinodular goitre e) Post-partum thyroiditis

**CORRECT-CHOICE LINE**: e

**REASONING**: Post-partum thyroiditis is associated with elevated thyroid peroxidase antibodies but no significant uptake on thyroid scintiscanning and is, therefore, the correct answer in this case given that symptoms began approximately two weeks postpartum. Viral thyroiditis is usually associated with an elevated ESR and negative antithyroid antibodies. Graves’ disease, toxic multinodular goitre and a toxic thyroid nodule would be associated with different patterns of uptake on thyroid scintiscanning.

**>>DESCRIPTION**: A 34-year-old woman, 6 weeks postpartum, presents with heat intolerance, tremors, and diarrhea, and thyrotoxicosis. Examination reveals a small goiter, tremor, and elevated thyroid peroxidase antibodies, with no thyroid uptake on scintiscanning.

**>>OPTIONS**: a) Graves’ disease b) Post-partum thyroiditis c) Toxic multinodular goitre d) Toxic thyroid nodule e) Viral thyroiditis

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Post-partum thyroiditis

**>>REASONING**: The most likely diagnosis is postpartum thyroiditis due to elevated thyroid peroxidase antibodies and absent thyroid uptake on scintiscanning, consistent with the postpartum onset. Other diagnoses are less likely due to differing scintigraphy findings or antibody profiles.

## Question #:109

**CLINICAL SCENERIO**: A 30-year-old male with background of type one diabetes mellitus presents with abdominal pain and shortness of breath. Investigations confirm he has diabetic ketoacidosis.

**QUESTION LINE**: Which one of the following investigations would suggest a discussion for possible intensive care admission?

**OPTIONS**: - a) Lactate 3 mmol/L - b) Bicarbonate level 19 mmol/L - c) pH 7.27 - d) White cell count 30 x 10^9/L - e) Potassium 3.4 mmol/L

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Parameters indicate severe diabetic ketoacidosis:

*  pH < 7
*  Blood ketone > 6 mmol/L
*  Bicarbonate < 5 mmol/L
*  Anion gap >16 mmol/l
*  Potassium < 3.5 mmol/L on admission
*  Tachycardia or bradycardia
*  Systolic blood pressure <90 mmHg
*  Oxygen saturation <92% on air
*  GCS < 12

**>>DESCRIPTION**: A 30-year-old male with type one diabetes presents with abdominal pain, shortness of breath, and confirmed diabetic ketoacidosis.

**>>OPTIONS**: a) Bicarbonate level 19 mmol/L b) Lactate 3 mmol/L c) pH 7.27 d) Potassium 3.4 mmol/L e) White cell count 30 x 10^9/L

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Potassium 3.4 mmol/L

**>>REASONING**: Severe DKA parameters include potassium < 3.5 mmol/L, pH < 7, blood ketone > 6 mmol/L, bicarbonate < 5 mmol/L, anion gap >16 mmol/l. Thus, potassium of 3.4 mmol/L is the correct answer.

## Question #:334

**CLINICAL SCENERIO**: A 30-year-old male with type 1 diabetes mellitus presents with abdominal pain, shortness of breath, and diabetic ketoacidosis. Which investigation suggests potential intensive care admission?

**QUESTION LINE**: Which one of the following investigations would suggest a discussion for possible intensive care admission?

**OPTIONS**: - f) Lactate 3 mmol/L - g) Bicarbonate level 19 mmol/L - h) pH 7.27 - i) White cell count 30 x 10^9/L - j) Potassium 3.4 mmol/L

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Parameters indicate severe diabetic ketoacidosis:

*  pH < 7
*  Blood ketone > 6 mmol/L
*  Bicarbonate < 5 mmol/L
*  Anion gap >16 mmol/l
*  Potassium < 3.5 mmol/L on admission
*  Tachycardia or bradycardia
*  Systolic blood pressure <90 mmHg
*  Oxygen saturation <92% on air
*  GCS < 12

**>>DESCRIPTION**: A 30-year-old male with type 1 diabetes mellitus presents with DKA. Which investigation indicates a possible need for intensive care admission?

**>>OPTIONS**: a) Bicarbonate level 19 mmol/L b) Lactate 3 mmol/L c) pH 7.27 d) Potassium 3.4 mmol/L e) White cell count 30 x 10^9/L

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Potassium 3.4 mmol/L

**>>REASONING**: Admission to ICU is suggested by several parameters, including pH < 7, Bicarbonate < 5, and potassium < 3.5. Option e (Potassium 3.4 mmol/L) is a key indicator.

## Question #:149

**CLINICAL SCENERIO**: A 45-year-old gentleman with a history of renal transplant due to membranous glomerulonephritis, on immunosuppressants, presents with visual hallucinations.

**QUESTION LINE**: Which medication may be implicated as a cause of this patient’s presentation?

**OPTIONS**: - a) Mycophenolate mofetil - b) Azathioprine - c) Prednisolone - d) Tacrolimus - e) Ciclosporin

**CORRECT-CHOICE LINE**: c

**REASONING**: This patient is likely to be experiencing steroid-induced psychosis, and therefore, prednisolone is the correct answer. Most immunosuppressants have side-effects, and it is important to be aware of common associations. In addition to psychosis, steroids such as prednisolone may cause weight gain, secondary diabetes, osteoporosis and myopathy.

Ciclosporin and tacrolimus are both calcineurin inhibitors. Ciclosporin is associated with nephrotoxicity, gum hypertrophy and hirsutism. Tacrolimus may cause tremor and secondary diabetes.

Mycophenolate mofetil and azathioprine are both anti-metabolites. Mycophenolate mofetil is associated with mucositis.

Azathioprine is steroid-sparing but can induce rashes, hepatitis and pancreatitis.

**>>DESCRIPTION**: A 45-year-old renal transplant recipient on immunosuppressants presents with visual hallucinations.

**>>OPTIONS**: a) Azathioprine b) Ciclosporin c) Mycophenolate mofetil d) Prednisolone e) Tacrolimus

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Prednisolone

**>>REASONING**: Prednisolone is the likely cause of the patient’s psychosis. Other immunosuppressants have different side effects: ciclosporin and tacrolimus (nephrotoxicity, tremor), mycophenolate mofetil (mucositis), and azathioprine (rashes, hepatitis, pancreatitis).

## Question #:47

**CLINICAL SCENERIO**: A 22-year-old female presents with nausea for 10 weeks, constipation, and fatigue. She has no past medical history and takes no regular medicines. Blood results, including thyroid function tests, show raised total T3 and T4 but normal fT3 and fT4.

**QUESTION LINE**: What investigation will you perform next?

**OPTIONS**: - a) Thyroid ultrasound - b) Thyroid peroxidase antibody levels - c) Thyroglobulin levels - d) Pituitary function tests - e) Pregnancy test

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin, which can be seen during pregnancy

The patient has raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin. This pattern of results is commonly seen during pregnancy. Her symptoms could be attributed to hypothyroidism, however equally they are common symptoms in pregnancy. She is a female of child rearing age and therefore a pregnancy test should be performed.

Thyroid peroxidase antibody and thyroglobulin levels should be measures in suspected autoimmune hypothyroidism. The normal TSH, fT3 and fT4 makes autoimmune hypothyroidism very unlikely.

There is nothing biochemically or clinically to suggest pituitary disease.

A thyroid ultrasound would be indicated in the presence of a thyroid nodule.

**>>DESCRIPTION**: A 22-year-old female presents with 10 weeks of nausea, constipation, and fatigue. Total T3 and T4 are elevated, but fT3 and fT4 are normal.

**>>OPTIONS**: a) Pituitary function tests b) Pregnancy test c) Thyroglobulin levels d) Thyroid peroxidase antibody levels e) Thyroid ultrasound

**>>CORRECT-CHOICE LINE**: Correct answer is b.

**>>CORRECT-CHOICE\_TEXT**: Pregnancy test

**>>REASONING**: Elevated total T3/T4 with normal free T3/T4 suggests increased thyroid binding globulin, common in pregnancy. Given the patient’s age and symptoms, a pregnancy test is the most appropriate next step. Autoimmune hypothyroidism and pituitary disease are unlikely based on the TSH and other thyroid hormone levels. A thyroid ultrasound is not indicated without a nodule.

## Question #:280

**CLINICAL SCENERIO**: A 25-year-old female presents with galactorrhoea. She reports no other symptoms on systematic enquiry. She has no past medical history and takes no regular medicines.

## Blood results are as follows:

| FSH | 0.2 mIU/ml (normal 1 - 8) | LH | 0.5 mIU/ml (normal 1-18) |
| --- | --- | --- | --- |
| Oestradiol | 482 pg/ml (normal 27 - 123) | Progesterone | 46 ng/mL (normal 5 - 20) |
| Prolactin | 82 ng/dL (normal 5 - 40) |  |  |

**QUESTION LINE**: What investigation will you perform?

**OPTIONS**: - a) MRI pituitary - b) Thyroid function tests - c) Pregnancy test - d) Pelvic ultrasound - e) No further investigations

**CORRECT-CHOICE LINE**: c

**REASONING**: Pregnancy is an important differential to consider when assessing a female patient with hyperprolactinaemia

The hormone results are entirely consistent with pregnancy. Oestradiol and progesterone are secreted from the corpus luteum in early pregnancy. At approximately the third month of gestation, the placenta takes over as the source of oestrogen and progesterone. High levels of these hormones results in suppression of LH and FSH. During pregnancy, prolactin levels also increase. Prolactin causes enlargement of the mammary glands and prepares for milk production.

**>>DESCRIPTION**: A 25-year-old female presents with galactorrhoea and the following blood results: FSH 0.2 mIU/ml, LH 0.5 mIU/ml, Oestradiol 482 pg/ml, Progesterone 46 ng/mL, and Prolactin 82 ng/dL.

**>>OPTIONS**: a) MRI pituitary b) No further investigations c) Pelvic ultrasound d) Pregnancy test e) Thyroid function tests

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Pregnancy test

**>>REASONING**: The patient’s hormone profile, including elevated prolactin, oestradiol, and progesterone, strongly suggests pregnancy, making a pregnancy test the appropriate initial investigation. Other options are less likely given the clinical presentation and hormone levels.

## Question #:7

**CLINICAL SCENERIO**: A 47-year-old woman is admitted to the surgical ward with severe loin to groin abdominal pain. A CT-KUB reveals a right-sided renal calculus. She reports polyuria, polydipsia, constipation, and altered mood over the past few weeks. Blood tests show: Elevated calcium (3.1 mmol/l), normal phosphate, and normal PTH.

**QUESTION LINE**: Which of the following is the most likely cause for her symptoms?

**OPTIONS**: a) Primary hyperparathyroidism b) Secondary hyperparathyroidism c) Sarcoidosis d) Tertiary hyperparathyroidism e) Type 1 renal tubular acidosis

**CORRECT-CHOICE LINE**: a

**REASONING**: The most likely diagnosis is primary hyperparathyroidism due to a parathyroid adenoma or hyperplasia. This is supported by the high serum calcium and normal phosphate levels. The PTH level may be normal.

**>>DESCRIPTION**: A 47-year-old woman presents with loin to groin pain, a right-sided renal calculus, polyuria, polydipsia, constipation, and altered mood. Blood tests reveal elevated calcium (3.1 mmol/l), normal phosphate, and normal PTH.

**>>OPTIONS**: a) Primary hyperparathyroidism b) Sarcoidosis c) Secondary hyperparathyroidism d) Tertiary hyperparathyroidism e) Type 1 renal tubular acidosis

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Primary hyperparathyroidism

**>>REASONING**: The correct answer is primary hyperparathyroidism, indicated by elevated calcium with normal phosphate and normal PTH. Secondary and tertiary hyperparathyroidism are less likely, and sarcoidosis or type 1 renal tubular acidosis are rare causes of hypercalcemia.

## Question #:207

**CLINICAL SCENERIO**: A 42-year-old woman presented with palpitation, tremor, and weight loss. Examination revealed a palpable goitre, exophthalmos, and tremor. Thyroid function tests showed elevated T4 and T3, and suppressed TSH.

**QUESTION LINE**: Which of the following treatments should be prescribed initially to improve symptoms?

**OPTIONS**: - a) Thyroidectomy - b) Propanolol - c) Radioiodine ablation - d) Carbimazole - e) Propylthiouracil

**CORRECT-CHOICE LINE**: b

**REASONING**: Propranolol should be used in new cases of Graves’ disease to help control symptoms

There is a high suspicion of Grave’s disease in this patient, and further investigation with a thyroid autoantibody profile is warranted.

Beta blockers are used to treat the symptoms of increased beta-adrenergic tone that are seen in Grave’s disease. Other such symptoms include anxiety and heat intolerance.

Thioamides are used to treat Grave’s hyperthyroidism, but not specifically the symptoms of increased beta-adrenergic tone. While the anti-thyroid effect of these drugs has a rapid onset, their clinical effect is more delayed because the pre-formed store of hormone in the thyroid gland and bound to thyroid-binding globulin must first be exhausted.

**>>DESCRIPTION**: A 42-year-old woman with palpitation, tremor, weight loss, goitre, exophthalmos, and tremor has elevated T4/T3 and suppressed TSH. Which initial treatment improves symptoms?

**>>OPTIONS**: a) Carbimazole b) Propanolol c) Propylthiouracil d) Radioiodine ablation e) Thyroidectomy

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Propanolol

**>>REASONING**: Propranolol is the initial treatment to control symptoms of Graves’ disease, like tremor, through beta-blockade. Other options are for long term management of thyrotoxicosis.

## Question #:281

**CLINICAL SCENERIO**: A 43-year-old man in theatre recovery presents with fever and tachycardia post-operatively after open reduction and internal fixation for a distal radius fracture. He received ondansetron, dexamethasone, and morphine during anesthesia. His vital signs show a heart rate of 130 beats/min (irregular), blood pressure of 135/74 mmHg, and a temperature of 39.4ºC. Investigations reveal elevated platelets, a slightly elevated WBC, suppressed TSH, and an elevated cortisol level.

**QUESTION LINE**: What is the most appropriate initial treatment?

**OPTIONS**: a. Carbimazole b. Hydrocortisone c. Propranolol d. Broad spectrum antibiotics e. Crystalloid infusion

**CORRECT-CHOICE LINE**: C

**REASONING**: The diagnosis is thyrotoxicosis. Initial treatment should focus on sympathetic storm suppression using beta blockade. Anti-thyroid medications and steroids are not the immediate priority.

**>>DESCRIPTION**: A 43-year-old man in theatre recovery presents with fever and tachycardia post-operatively after fracture fixation. He received ondansetron, dexamethasone, and morphine during anesthesia. His heart rate is 130/min (irregular), blood pressure is 135/74 mmHg, and temperature is 39.4ºC. Investigations show elevated platelets and suppressed TSH.

**>>OPTIONS**: a. Broad spectrum antibiotics b. Carbimazole c. Crystalloid infusion d. Propranolol e. Hydrocortisone

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Propranolol

**>>REASONING**: The most appropriate initial treatment is propranolol (beta-blocker) to address the sympathetic storm associated with thyrotoxicosis. Other options are not the initial priority, as infection is less likely given the clinical picture. Anti-thyroid medications (carbimazole) take weeks to act. Hydrocortisone is not indicated as the patient has normal electrolytes.

## Question #:86

**CLINICAL SCENERIO**: A 43-year-old woman with a history of type 1 diabetes mellitus and Grave’s disease (in remission) presents with confusion, visual hallucinations, aggressive behavior, diarrhea, and abdominal discomfort after 48 hours of treatment for diabetic ketoacidosis. Observations: SpO2 97% room air, respiratory rate 18 breaths per minute, heart rate 145 beats per minute, blood pressure 170/110 mmHg, temperature 40.2ºC. Examination reveals generalized abdominal pain, mild pitting oedema, and an irregularly irregular pulse. Labs: elevated FT4, suppressed TSH. CRP 24 mg/L

**QUESTION LINE**: What is the most appropriate acute treatment for the likely underlying cause of this patient’s current presentation?

**OPTIONS**: - a) 300mg aspirin - b) Carbimazole - c) IV antibiotics - d) Propylthiouracil - e) Thyroidectomy

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: In thyroid storm, treat acutely with propylthiouracil rather than carbimazole or surgery

The stem describes a classic case of thyroid storm. It is most commonly seen in patients with a background of Grave’s disease but can occur in other scenarios too. There is usually a precipitating insult such as diabetic ketoacidosis or a heart attack.

The likelihood of a thyroid storm can be calculated using the Burch and Wartofksy thyroid storm diagnostic criteria. This attributes points to the presence of pyrexia, tachycardia, signs of heart failure, confusion, coma, seizures, gastrointestinal disturbance etc.

In this case, the patient scores 90. A score of >45 indicates a likely thyroid storm. The patient also has a suppressed TSH level with raised T4, this cements the diagnosis.

In an ideal situation treatment for presumed thyroid storm should not be delayed whilst awaiting blood results as this condition has significant mortality (>10%). Blood tests may be in keeping with hyperthyroidism, but there is no specific cut- off to diagnose thyroid storm, the diagnosis is made on clinical findings.

Propylthiouracil is the correct answer. Patients should be assessed with an ABCDE approach and stabilised. IV fluids and cooling are important aspects of treatment. Tachyarrhythmias should also be managed according to local guidelines. The most appropriate treatment for the underlying thyroid storm is propylthiouracil. This inhibits thyroid hormone synthesis and also inhibits the conversion of T4 to T3.

Carbimazole is incorrect. Although carbimazole can be used, propylthiouracil is the traditionally preferred treatment as it has a faster onset of action and also inhibits the conversion of fT4 to T3.

Thyroidectomy is incorrect. This can be used as a definitive treatment for Grave’s disease and can also be used in the acute setting if a patient has had a previous poor reaction to thionamides such as propylthiouracil. Usually, the first-line treatment would be stabilisation with beta-blockers, supportive treatment and oral thionamides.

IV antibiotics is incorrect. In this case, the patient is pyrexial and has new-onset atrial fibrillation with a rapid ventricular response. Diarrhoea and abdominal pain could indicate an intra-abdominal infection. It would not be unusual for the patient to receive IV antibiotics in this scenario to cover for potential infection, but given the blood results here - normal white cell count, normal CRP, and deranged thyroid function - we can be quite sure of a diagnosis of thyroid storm which wouldn’t respond to IV antibiotics. The concomitant atrial fibrillation with signs of heart failure (pedal oedema), extreme pyrexia, and delirium, are better explained by thyroid storm.

300mg aspirin is incorrect. This would be the treatment of stroke. There is an altered conscious level with behavioural changes and pyrexia, both of which can be seen in acute stroke. There are, however, no focal neurological findings described and a stroke would not explain the change in bowel habit and abdominal pain.

**>>DESCRIPTION**: 43-year-old woman with type 1 DM and Graves’s disease (remission) presents with confusion, visual hallucinations, aggression, diarrhea, and abdominal discomfort after DKA treatment. Vitals: T 40.2ºC, HR 145, BP 170/110. Exam: abdominal pain, pitting oedema, irregular pulse. Labs: elevated FT4, suppressed TSH. CRP 24 mg/L.

**>>OPTIONS**: a) 300mg aspirin b) Carbimazole c) IV antibiotics d) Propylthiouracil e) Thyroidectomy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil

**>>REASONING**: The patient presents with thyroid storm. Propylthiouracil is the preferred acute treatment as it inhibits thyroid hormone synthesis and T4 to T3 conversion. Carbimazole is slower. Thyroidectomy is not first-line. Antibiotics are not indicated given normal WBC and CRP with deranged thyroid function. Aspirin is for stroke, but no focal findings are present.

## Question #:105

**CLINICAL SCENERIO**: A 28-year-old pregnant lady presents to the Emergency Department with palpitations and sweating. She mentions that she has had these symptoms on and off for the past 4 months but that they have worsened over the past few weeks. Now she is feeling worried and wanted to be assessed medically due to her concern she was having a miscarriage. She looks particularly anxious to be in hospital. This is her first pregnancy. She is 7 weeks pregnant. She has had no vaginal bleeding or discharge during the course of her pregnancy. She is normally fit and well.

Initial observations reveal a blood pressure of 130/85 mmHg, a heart rate of 110 beats per minute, a respiratory rate of 19/min, oxygen saturations of 99% on air and a temperature of 37.5ºC. Examination findings reveal a resting tachycardia and a subtle goitre is noted.

Blood test results are as follows:

Hb

110 g/l

| Wcc | 12 x109/l |
| --- | --- |
| Plt | 245 x109/l |
| CRP | 12 mg/l |
| Na+ | 140 mmol/l |
| K+ | 5.0 mmol/l |
| Ur | 5.7 mmol/l |
| Cr | 110 µmol/l |
| D- dimer | 490 ng/ml |
| T4 | 21 mU/l |
| TSH | <0.05 pmol/l |

**QUESTION LINE**: Given the most likely diagnosis, how should this lady be managed?

**OPTIONS**: - a) Watch and wait/symptomatic control with beta blockade - b) Radioactive iodine therapy - c) Subtotal thyroidectomy - d) Propylthiouracil - e) Block and replace carbimazole + thyroxine

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: This lady has symptoms and biochemical evidence of hyperthyroidism. This lady’s symptoms predate her pregnancy, therefore it is not pregnancy induced thyrotoxicosis and will not self-limit - she will need treatment to prevent complications to her and the foetus. Radioactive iodine is contraindicated.

Subtotal thyroidectomy is a little risky and extreme during pregnancy. Carbimazole, whilst normally first line, has been associated with neonatal aplasia cutis before 12 weeks gestation and is therefore usually avoided. This leaves propylthiouracil as the current safest option. During the second trimester, propylthiouracil should be changed to carbimazole due to the potential risk of hepatotoxicity with propylthiouracil. The lowest dose that controls the hyperthyroid state should be used as both medications can cross the placenta.

**>>DESCRIPTION**: A 28-year-old, 7-week pregnant female presents with palpitations and sweating for 4 months, worsening recently. She is anxious about a possible miscarriage. Exam: BP 130/85 mmHg, HR 110 bpm, goitre. Labs: T4 21 mU/l, TSH <0.05 pmol/l.

**>>OPTIONS**: a) Block and replace carbimazole + thyroxine b) Propylthiouracil c) Radioactive iodine therapy d) Subtotal thyroidectomy e) Watch and wait/symptomatic control with beta blockade

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil

**>>REASONING**: The patient has hyperthyroidism predating the pregnancy, requiring treatment. Radioactive iodine and subtotal thyroidectomy are contraindicated in pregnancy. Carbimazole is avoided in the first trimester due to aplasia cutis risk. Propylthiouracil is the safest first-line option.

## Question #:126

**CLINICAL SCENERIO**: A 25-year-old woman, 3 weeks pregnant, is referred to endocrinology with a month of diarrhea and a recent pregnancy despite oral contraceptives. Blood tests show undetectable TSH and free T4 levels of 52pmol/l. Thyroid-stimulating hormone receptor antibodies are positive. She has no past medical history.

**QUESTION LINE**: What is the most appropriate management for her hyperthyroidism?

**OPTIONS**: - a) Hemithyroidectomy - b) Carbimazole - c) Propylthiouracil - d) Propranolol - e) Total thyroidectomy

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: In pregnant woman who develop hyperthyroidism in the first trimester, propylthiouracil is preferred over carbimazole due to lower risk of foetal malformation

This patient has a new diagnosis of Graves’ disease with positive thyroidstimulating hormone receptor antibodies whilst pregnant in the first trimester. The most appropriate medical treatment is propylthiouracil. This has a lower risk of foetal malformation than carbimazole in the first trimester and would, therefore, be first-line treatment. Radioiodine is contra-indicated in pregnancy, whilst propranolol would not address the underlying problem. Surgery can be an option to patients who are resistant to propylthiouracil, and would need to happy at an urgent basis. Of operations, a hemithyroidectomy is more appropriate in trying to obtain histological samples in suspicious histology, whilst a total thyroidectomy would be a definitive management. Urgent treatment is necessary as thyroid-stimulating hormone receptor antibodies cross the placenta and can cause foetal problems.

**>>DESCRIPTION**: A 25-year-old woman, 3 weeks pregnant, presents with diarrhea, undetectable TSH, elevated free T4 (52pmol/l), and positive thyroid-stimulating hormone receptor antibodies. No significant past medical history.

**>>OPTIONS**: a) Carbimazole b) Hemithyroidectomy c) Propranolol d) Propylthiouracil e) Total thyroidectomy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil

**>>REASONING**: Propylthiouracil is preferred over carbimazole in the first trimester of pregnancy due to a lower risk of fetal malformation. Propranolol doesn’t address the underlying issue, and surgery is reserved for propylthiouracil-resistant cases. Radioiodine is contraindicated.

## Question #:310

**CLINICAL SCENERIO**: A 35-year-old woman with a history of Graves’ disease, non-compliant with medication, presents with fever, sweating, tachycardia, hypertension, and confusion. Examination reveals proptosis, chemosis, crackles, and an elevated JVP. Blood tests show elevated bilirubin, ALP, ALT, Î³GT, and significantly elevated free T4 with a suppressed TSH.

**QUESTION LINE**: Based on the likely diagnosis, what treatment is most appropriate from the listed options?

**OPTIONS**: - a. Antibiotics - b. Carbimazole - c. Propylthiouracil - d. Radioactive iodine therapy - e. Surgery

**CORRECT-CHOICE LINE**: C

**REASONING**: Propylthiouracil is the correct answer. The patient presents with ophthalmological features of Graves’ disease in association with fever, confusion, tachycardia, tremor, hypertension and clinical evidence of heart failure. In the setting of significant hyperthyroidism this constitutes a ‘thyroid storm’. Propylthiouracil has traditionally been the preferred anti-thyroid drug to be used in this setting due to its more rapid onset of action when compared with carbimazole and its additional ability to inhibit peripheral conversion of T4 to T3.

Antibiotics is not the right answer. Although this patient has a fever, she has a normal CRP and WCC and infection is less likely than thyroid storm given the clinical context.

Carbimazole is incorrect. This can be used in thyroid storm but is less preferred due to its slower onset of action.

Radioactive iodine therapy is incorrect. This can be used to manage Graves’ disease but is not an option acutely for treating thyroid storm.

Surgery is incorrect. This is an option for the treatment of Graves disease but is not an option for the acute treatment of thyroid storm.

**>>DESCRIPTION**: A 35-year-old woman with Graves’ disease, non-compliant with medication, presents with fever, tachycardia, hypertension, and confusion, concerning for thyroid storm. Blood tests show elevated free T4 and suppressed TSH.

**>>OPTIONS**: a. Antibiotics b. Carbimazole c. Propylthiouracil d. Radioactive iodine therapy e. Surgery

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil

**>>REASONING**: Propylthiouracil is the preferred treatment for thyroid storm due to its rapid action and ability to inhibit T4 to T3 conversion. Antibiotics are inappropriate as infection is less likely. Carbimazole is an alternative, but less preferred. Radioactive iodine therapy and surgery are not acute treatments.

## Question #:330

**CLINICAL SCENERIO**: A 28-year-old pregnant woman at 7 weeks gestation presents with palpitations, sweating, and anxiety. She reports these symptoms for 4 months, worsening recently, and is concerned about miscarriage. Initial observations: BP 130/85 mmHg, HR 110 bpm, RR 19/min, SpO2 99%, temp 37.5ºC. Examination reveals tachycardia and a subtle goitre. Blood tests show elevated T4 and suppressed TSH.

**QUESTION LINE**: Given the most likely diagnosis, how should this lady be managed?

**OPTIONS**: - f) Watch and wait/symptomatic control with beta blockade - g) Radioactive iodine therapy - h) Subtotal thyroidectomy - i) Propylthiouracil - j) Block and replace carbimazole + thyroxine

**CORRECT-CHOICE LINE**: d

**REASONING**: This lady has symptoms and biochemical evidence of hyperthyroidism. This lady’s symptoms predate her pregnancy, therefore it is not pregnancy induced thyrotoxicosis and will not self-limit - she will need treatment to prevent complications to her and the foetus. Radioactive iodine is contraindicated. Subtotal thyroidectomy is a little risky and extreme during pregnancy. Carbimazole, whilst normally first line, has been associated with neonatal aplasia cutis before 12 weeks gestation and is therefore usually avoided. This leaves propylthiouracil as the current safest option. During the second trimester, propylthiouracil should be changed to carbimazole due to the potential risk of hepatotoxicity with propylthiouracil. The lowest dose that controls the hyperthyroid state should be used as both medications can cross the placenta.

**>>DESCRIPTION**: A 28-year-old, 7-week pregnant woman presents with palpitations, sweating, anxiety, tachycardia, goitre, and elevated T4 with suppressed TSH. How should she be managed?

**>>OPTIONS**: a) Block and replace carbimazole + thyroxine b) Propylthiouracil c) Radioactive iodine therapy d) Subtotal thyroidectomy e) Watch and wait/symptomatic control with beta blockade

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil

**>>REASONING**: The patient likely has hyperthyroidism. Propylthiouracil is the safest initial treatment during the first trimester due to potential fetal risks with other options (radioactive iodine, surgery, carbimazole).

## Question #:57

**CLINICAL SCENERIO**: A 52 year-old woman presents with nausea, fever, and confusion. Her husband states she was recovering from a recent upper respiratory tract infection and sore throat. He also mentions she has previously been experiencing episodes of diarrhoea and palpitations over the last three months. Examination reveals a temperature of 40.6ºC, pulse rate of 160 beats per minute and blood pressure of 110/70 mmHg. Her pulse is irregularly irregular. Blood tests show: TSH 0.03 mu/l, Free T4 31 pmol/l, Total T4 220 nmol/l.

**QUESTION LINE**: What is the most appropriate immediate treatment?

**OPTIONS**: - a) Propylthiouracil, corticosteroids and propranolol - b) Propylthiouracil and propranolol - c) Radio-iodine, corticosteroids and propranolol - d) Propylthiouracil and corticosteroids - e) Radio-iodine, propranolol and carbimazole

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Thyrotoxic storm is treated with beta blockers, propylthiouracil and hydrocortisone

This patient is having a thyrotoxic storm (hyperthyroid crisis) a rare medical emergency that is caused by an exacerbation of hyperthyroidism and characterised by decompensation of one or more organ systems in people with untreated or poorly treated hyperthyroidism. The precipitating cause is most commonly infection, as with this case, although it is important to check for other causes. The patient above is in atrial fibrillation and shows signs of renal impairment due to dehydration. First line treatment for this medical emergency is propylthiouracil, corticosteroids and propranolol, although chlorpromazine can be added for severe anxiety.

**>>DESCRIPTION**: A 52-year-old woman presents with nausea, fever, and confusion, recovering from an upper respiratory infection. She has a history of diarrhea and palpitations. Examination shows a temperature of 40.6ºC, pulse of 160, BP 110/70, and irregular pulse. Labs: TSH 0.03, Free T4 31, Total T4 220.

**>>OPTIONS**: a) Propylthiouracil and corticosteroids b) Propylthiouracil and propranolol c) Propylthiouracil, corticosteroids and propranolol d) Radio-iodine, corticosteroids and propranolol e) Radio-iodine, propranolol and carbimazole

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Propylthiouracil, corticosteroids and propranolol

**>>REASONING**: The patient is experiencing a thyrotoxic storm, likely triggered by an infection. First-line treatment includes propylthiouracil, corticosteroids, and propranolol. Other options are incorrect as they do not comprehensively address the hyperthyroid crisis and its symptoms.

## Question #:180

**CLINICAL SCENERIO**: A 34-year-old woman presents with weight gain, irregular periods, hypertension, and laboratory findings including elevated liver enzymes and urine free cortisol.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Cushing’s disease b) Cushing’s syndrome c) Exogenous steroid use d) Hypothyroidism e) Pseudo-Cushing’s

**CORRECT-CHOICE LINE**: e

**REASONING**: The insulin tolerance test can be used to distinguish Cushing’s syndrome from pseudo-Cushing’s. The patient’s presentation, including weight gain, hypertension, irregular periods, and abnormal liver function tests, along with imaging findings of fatty liver changes, points towards Pseudo-Cushing’s. Pseudo-Cushing’s is also known as ‘physiological hypercortisolism’.

**>>DESCRIPTION**: A 34-year-old woman with weight gain, irregular periods, hypertension, elevated liver enzymes, and high urine free cortisol. What is the most likely diagnosis?

**>>OPTIONS**: a) Cushing’s disease b) Cushing’s syndrome c) Exogenous steroid use d) Hypothyroidism e) Pseudo-Cushing’s

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Pseudo-Cushing’s

**>>REASONING**: The most likely diagnosis is Pseudo-Cushing’s, supported by clinical features (weight gain, irregular periods, hypertension), lab findings (elevated liver enzymes, high urine free cortisol) and imaging (fatty liver). Other options are less likely due to inconsistent lab values or imaging findings.

## Question #:155

**CLINICAL SCENERIO**: A 65-year-old man with a history of NSTEMI, hypercholesterolaemia, hypertension, and depression presents with hyponatraemia. His medications include sertraline, bisoprolol, ramipril, and furosemide. Blood and urine investigations are provided.

**QUESTION LINE**: What is the most likely cause of the hyponatraemia?

**OPTIONS**: a) SIADH b) Dilutional hyponatraemia secondary to heart failure c) Pseudohyponatraemia d) Furosemid e) Hypothyroidism

**CORRECT-CHOICE LINE**: c.

**REASONING**: Pseudohyponatraemia is characterised by a normal measured serum osmolarity, however the calculated osmolarity (based on an erroneously low plasma sodium result) is reduced. This results in a raised osmolar gap

Identifying the cause of hyponatraemia can be challenging. The first step is to confirm that it is a true hypotonic hyponatraemia. This is done by measuring the plasma osmolarity.

* If the measured plasma osmolarity is low this confirms true hypotonic hyponatraemia.
* If the plasma osmolarity is normal then this is suggestive of pseudohyponatraemia and should prompt you to measure proteins and lipids, which if present in high levels, can cause a pseudohyponatraemia due to the measuring technique.
* If the serum osmolarity is high then this confirms hypertonic hyponatraemia and should prompt you to check for high levels of solutes in the plasma, hyperglycaemia being the most common.

In this case, the normal measured serum osmolarity suggests pseudohyponatraemia. Pseudohyponatraemia occurs due to a measuring technique defect. Using standard techniques, serum sodium is measured as a ratio of sodium to plasma volume. If the patients plasma has high amounts of proteins or lipids, the plasma volume will be increased resulting in a measured hyponatraemia. This is not a true hyponatraemia as the actual ratio of sodium to plasma fluid will be normal. Common causes include hyperproteinaemia (e.g. TPN, IVIG) and hyperlipidaemia (in particular hypertriglyceridemia).

In this case, the measured serum osmolarity can be compared with the calculated serum osmolarity. The calculated serum osmolarity = 2Na + 2K + glucose + urea = 273.3 mOsmol/kg. The osmolar gap = measured osmolarity - calculated osmolarity = 290 - 273.3 = 16.7 (normal <10). A raised osmolar gap is suggestive of the presence of other osmotically active particles. In this case it is due to the presence of a significantly raised triglyceride level.

**>>DESCRIPTION**: A 65-year-old man with a history of NSTEMI, hypercholesterolaemia, hypertension, and depression presents with hyponatraemia and is on sertraline, bisoprolol, ramipril, and furosemide. Lab results include a normal serum osmolarity and elevated triglycerides.

**>>OPTIONS**: a) Dilutional hyponatraemia secondary to heart failure b) Furosemid c) Hypothyroidism d) Pseudohyponatraemia e) SIADH

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Pseudohyponatraemia

**>>REASONING**: The most likely cause of hyponatremia is pseudohyponatremia due to the normal serum osmolarity and elevated triglycerides. Other options are less likely as the normal serum osmolarity rules out SIADH, heart failure, hypothyroidism and diuretic use as the primary cause.

## Question #:242

**CLINICAL SCENERIO**: A 19-year-old patient admitted with suspected appendicitis shows prolonged QT on ECG and adjusted calcium of 2.02 mmol/l. The patient has dimples on outer knuckles, BMI of 29 kg/m², and blood tests show: Adjusted calcium 2.02 mmol/l, PTH 69 pmol/L, Phosphate 2.0 mmol/l, and ALP 130 u/l.

**QUESTION LINE**: What is the most likely underlying cause for this patient’s hypocalcaemia?

**OPTIONS**: a. Hypoparathyroidism b. Pseudohypoparathyroidism type 1a c. Pseudohypoparathyroidism type 1b d. Pseudopseudohypoparathyroidism e. Secondary hyperparathyroidism

**CORRECT-CHOICE LINE**: B

**REASONING**: Short fifth metacarpals, short stature, learning difficulties + ↑ PTH, ↓ calcium, ↑ phosphate → pseudohypoparathyroidism

This patient has a high PTH, a low calcium, a high phosphate and a normal ALP. The patient is also obese and the dimples on the outer two knuckles are likely to represent shortening of the 4th and 5th metacarpals. This biochemistry in combination of these clinical features is characteristic of pseudohypoparathyroidism Type 1a (Albright’s Hereditary Osteodystrophy).

Pseudopseudohypoparathyroidism would have the same clinical features but would have normal biochemistry. Pseudohypoparathyroidism Type 1b would have the same biochemistry but lack the clinical features.

This patient has a high PTH, therefore this immediately excludes hypoparathyroidism. In secondary hyperparathyroidism the ALP would be elevated therefore this is incorrect

**>>DESCRIPTION**: A 19-year-old patient with suspected appendicitis has prolonged QT, dimples on outer knuckles, and blood tests reveal hypocalcemia, elevated PTH, elevated phosphate, and normal ALP. What is the most likely cause of hypocalcemia?

**>>OPTIONS**: a. Hypoparathyroidism b. Pseudohypoparathyroidism type 1a c. Pseudohypoparathyroidism type 1b d. Pseudopseudohypoparathyroidism e. Secondary hyperparathyroidism

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Pseudohypoparathyroidism type 1a

**>>REASONING**: The patient presents with hypocalcemia, elevated PTH and phosphate, along with clinical features of Albright’s hereditary osteodystrophy (dimples on outer knuckles), which strongly suggests pseudohypoparathyroidism type 1a. Hypoparathyroidism and secondary hyperparathyroidism are excluded by the elevated PTH. Pseudopseudohypoparathyroidism lacks biochemical abnormalities. Pseudohypoparathyroidism Type 1b lacks the clinical features.

## Question #:131

**CLINICAL SCENERIO**: A 52 year old lady presents complaining of polydipsia and polyuria. She has a background of hypertension, hypercholesterolaemia and bipolar affective disorder and a strong family history of diabetes - she is unsure which type.

Results show the following:

| Na+ | 131mmol/l |
| --- | --- |
| urine osmolality | 287mOsmol/kg (300 - 900mOsmol/kg) |
| plasma osmolality | 287mOsmol/kg (285 - 295mOsmol/kg) |

**QUESTION LINE**: Which of the following is the most likely explanation for this lady’s symptoms?

**OPTIONS**: a) Psychogenic polydipsia b) Syndrome of inappropriate anti-diuretic hormone (SIADH) c) Diabetes insipidus d) Diabetes mellitus type e) Hyponatraemia

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Although this lady is biochemically hyponatraemic, this is unlikely to be the cause of her symptoms.

With a past medical history of bipolar affective disorder, although not stated in the question, there is a good chance she may be on lithium which predisposes her to developing nephrogenic diabetes insipidus. However with this diagnosis, we would expect a much lower urine osmolality and a higher plasma osmolality. She would also have a normal to high serum sodium. The opposite would indicate a diagnosis of SIADH (serum hypo-osmolality and high urine osmolality).

The osmolality results here reflect a diagnosis of psychogenic polydipsia with a low urine osmolality and a low end of normal plasma osmolality.

**>>DESCRIPTION**: A 52-year-old woman with hypertension, hypercholesterolemia, bipolar disorder, and a family history of diabetes presents with polydipsia and polyuria. Na+ 131mmol/l, urine osmolality 287mOsmol/kg, plasma osmolality 287mOsmol/kg.

**>>OPTIONS**: a) Diabetes insipidus b) Diabetes mellitus type c) Hyponatraemia d) Psychogenic polydipsia e) Syndrome of inappropriate anti-diuretic hormone (SIADH)

**>>CORRECT-CHOICE LINE**: Correct answer is d.

**>>CORRECT-CHOICE\_TEXT**: Psychogenic polydipsia

**>>REASONING**: The osmolality results (low urine osmolality and low-normal plasma osmolality) point to psychogenic polydipsia. Nephrogenic diabetes insipidus would show lower urine and higher plasma osmolality, while SIADH presents with serum hypo-osmolality and high urine osmolality. Hyponatremia unlikely cause of her symptoms.

## Question #:139

**CLINICAL SCENERIO**: A 52-year-old lady with hypertension, hypercholesterolaemia, bipolar affective disorder, and a family history of diabetes presents with polydipsia and polyuria. Laboratory results are provided.

**QUESTION LINE**: Which of the following is the most likely explanation for this lady’s symptoms?

**OPTIONS**: - a) Psychogenic polydipsia - b) Syndrome of inappropriate anti-diuretic hormone (SIADH) - c) Diabetes insipidus - d) Diabetes mellitus type - e) Hyponatraemia

**CORRECT-CHOICE LINE**: a

**REASONING**: Although this lady is biochemically hyponatraemic, this is unlikely to be the cause of her symptoms.

With a past medical history of bipolar affective disorder, although not stated in the question, there is a good chance she may be on lithium which predisposes her to developing nephrogenic diabetes insipidus. However with this diagnosis, we would expect a much lower urine osmolality and a higher plasma osmolality. She would also have a normal to high serum sodium. The opposite would indicate a diagnosis of SIADH (serum hypo-osmolality and high urine osmolality).

The osmolality results here reflect a diagnosis of psychogenic polydipsia with a low urine osmolality and a low end of normal plasma osmolality.

**>>DESCRIPTION**: A 52-year-old woman with a history of hypertension, hypercholesterolaemia, bipolar disorder, and a family history of diabetes presents with polydipsia and polyuria. Relevant laboratory results are provided.

**>>OPTIONS**: a) Diabetes insipidus b) Diabetes mellitus type c) Hyponatraemia d) Psychogenic polydipsia e) Syndrome of inappropriate anti-diuretic hormone (SIADH)

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Psychogenic polydipsia

**>>REASONING**: Psychogenic polydipsia is the most likely explanation, given the patient’s low urine osmolality and low-normal plasma osmolality. Hyponatremia is unlikely to be the primary cause. Diabetes insipidus and SIADH are less likely due to the osmolality findings. Lithium use, common in bipolar disorder, can cause nephrogenic diabetes insipidus, but is not consistent with the lab results.

## Question #:38

**CLINICAL SCENERIO**: A 62-year-old man presents with tiredness, muscle aches, fever, and neck pain after a recent upper respiratory infection treated with paracetamol and decongestants. Examination reveals anxiety, tremor, fever, tachycardia, and a tender, enlarged thyroid gland. Blood tests show elevated ESR, high free T4 and T3, and suppressed TSH.

**QUESTION LINE**: Which of the following investigations is likely to be most helpful in establishing the diagnosis?

**OPTIONS**: a) Blood cultures b) Serum anti-thyroid antibodies c) Ultrasound scan of neck d) Radioactive iodine uptake scan e) Fine needle aspiration

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: In De Quervain’s thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy The patients presenting symptoms and signs are typical of a diagnosis of subacute thyroiditis, of which the most likely cause of this is De Quervains thyroiditis.

Radioactive iodine uptake scan is the most suitable investigation for confirming subacute thyroiditis which demonstrates reduced intake.

**>>DESCRIPTION**: 62-year-old man presents with fatigue, myalgia, fever, and anterior neck pain following a recent URI. Exam: anxiety, tremor, fever, tachycardia, tender enlarged thyroid. Labs: elevated ESR, high T4/T3, suppressed TSH.

**>>OPTIONS**: a) Blood cultures b) Fine needle aspiration c) Radioactive iodine uptake scan d) Serum anti-thyroid antibodies e) Ultrasound scan of neck

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Radioactive iodine uptake scan

**>>REASONING**: Radioactive iodine uptake scan is the most helpful investigation, as De Quervain’s thyroiditis (subacute thyroiditis) typically shows globally reduced iodine-131 uptake.

## Question #:164

**CLINICAL SCENERIO**: A 54-year-old woman with a history of Graves’ disease, treated with carbimazole six months prior, presents to the ED with palpitations and sweating. She has type 2 diabetes, hypertension, ischaemic heart disease, and morbid obesity. Observations: HR 111, BP 124/72, RR 21, O2 sats 96%, temp 37.1C. Examination reveals diaphoresis, anxiety, tremor, normal ophthalmic exam, small goitre, and BMI of 53. Labs: TSH 0.0, Free T4 42.

**QUESTION LINE**: Given the likely diagnosis, what is the optimal treatment approach to address the underlying cause?

**OPTIONS**: 1. Propranolol 2. Propylthiouracil 3. Radioiodine therapy 4. Repeat carbimazole treatment 5. Surgery

**CORRECT-CHOICE LINE**: 3

**REASONING**: Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Radioiodine therapy is the correct answer. This patient has a relapse of Graves’ disease. Guidelines recommend that in those patients who have a relapse of disease despite a course of antithyroid drug treatment, definitive management should be instituted in the form of radioiodine treatment or surgery. This patient’s many medical problems including ischaemic heart disease and morbid obesity make her a significant anaesthetic risk and therefore radioiodine therapy would be preferable.

Surgery is incorrect. This is an option for the treatment of relapsed Graves’ disease. However, she would be a high-risk surgical candidate and therefore radioiodine therapy would be preferable.

Repeat carbimazole treatment is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves’ disease.

Propylthiouracil is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves’ disease.

Propranolol is incorrect. This would control some of the symptoms of hyperthyroidism without treating the underlying cause.

**>>DESCRIPTION**: A 54-year-old woman with a history of Graves’ disease, previously treated with carbimazole, presents with palpitations, sweating, and elevated thyroid hormones. She has multiple comorbidities including ischaemic heart disease and morbid obesity.

**>>OPTIONS**: 1. Propranolol 2. Propylthiouracil 3. Radioiodine therapy 4. Repeat carbimazole treatment 5. Surgery

**>>CORRECT-CHOICE LINE**: 3

**>>CORRECT-CHOICE\_TEXT**: Radioiodine therapy

**>>REASONING**: Radioiodine therapy is the preferred treatment for relapsed Graves’ disease, especially given the patient’s comorbidities. The other options are incorrect because they are either not definitive treatments (Propranolol), not first-line in relapse (Propylthiouracil, Repeat carbimazole), or carry higher risk (Surgery).

## Question #:262

**CLINICAL SCENERIO**: A 53-year-old woman with a history of Grave’s disease presents with diarrhea, palpitations, and heat intolerance. She is post-menopausal with no recent illness. Thyroid function tests show a TSH of 0.2. On examination, she is warm, tachycardic, and has a smooth, non-tender goitre, without eye signs.

**QUESTION LINE**: What is the most appropriate treatment?

**OPTIONS**: - a) Advice on medication compliance - b) Prednisolone - c) Propylthiouracil - d) Radioiodine therapy - e) Thyroidectomy

**CORRECT-CHOICE LINE**: Answer is D.

**REASONING**: Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Given this woman’s medical history, findings of a smooth non-tender goitre, and laboratory results, the most likely diagnosis here is a relapse of her Grave’s disease. In these cases and the absence of any contraindications, radioiodine therapy is the preferred treatment.

Prednisolone can be used in subacute thyroiditis however this is not the diagnosis here as evidenced by the lack of recent illness and non-tender goitre.

Propylthiouracil is an alternative antithyroid drug to carbimazole and is preferred in early pregnancy. As this woman is presenting with a relapse of Grave’s disease, radioiodine therapy is preferred. Also, note she is post-menopausal, and therefore pregnancy is not a contraindication here to either radioiodine therapy or carbimazole.

Thyroidectomy is not the first-line treatment in a relapse of previously wellcontrolled Grave’s disease.

**>>DESCRIPTION**: A 53-year-old postmenopausal woman with a history of Graves’ disease presents with diarrhea, palpitations, and heat intolerance. Her TSH is 0.2, and she has a smooth, non-tender goitre and tachycardia. What is the best treatment?

**>>OPTIONS**: a) Advice on medication compliance b) Prednisolone c) Propylthiouracil d) Radioiodine therapy e) Thyroidectomy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Radioiodine therapy

**>>REASONING**: Radioiodine therapy is the preferred treatment for relapsed Graves’ disease in the absence of contraindications. Other options are not first-line treatments for this condition. Prednisolone is used for thyroiditis, Propylthiouracil is used in early pregnancy and Thyroidectomy is not the first-line treatment.

## Question #:186

**CLINICAL SCENERIO**: A 22-year-old Asian woman with a BMI of 24 kg/m² presents with new acne, hirsutism, irregular periods, and weight gain. Examination reveals mild acne and thick hair growth. Abdominal exam is unremarkable.

**QUESTION LINE**: What are the most likely biochemical results given the clinical findings?

**OPTIONS**: - a) Raised testosterone, low LH/FSH ratio, insulin resistance - b) Low testosterone, low LH/FSH ratio, insulin resistanc - c) Low testosterone, raised LH/FSH ratio, insulin resistance - d) Raised testosterone, raised LH/FSH ratio, increased insulin sensitivity - e) Raised testosterone, raised LH/FSH ratio, insulin resistance

**CORRECT-CHOICE LINE**: e

**REASONING**: The clinical description is consistent with polycystic ovary syndrome (PCOS). This presents with hirsutism, acne, oligo/amenorrhoea and subfertility. Biochemical findings in PCOS include insulin resistance, raised testosterone, raised LH/FSH ratio, raised prolactin and low HDL.

**>>DESCRIPTION**: A 22-year-old Asian woman with a BMI of 24 kg/m² presents with acne, hirsutism, irregular periods, and weight gain. Physical exam reveals mild acne and thick hair growth.

**>>OPTIONS**: a) Low testosterone, low LH/FSH ratio, insulin resistanc b) Low testosterone, raised LH/FSH ratio, insulin resistance c) Raised testosterone, low LH/FSH ratio, insulin resistance d) Raised testosterone, raised LH/FSH ratio, increased insulin sensitivity e) Raised testosterone, raised LH/FSH ratio, insulin resistance

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Raised testosterone, raised LH/FSH ratio, insulin resistance

**>>REASONING**: The most likely diagnosis is PCOS. Key biochemical findings include elevated testosterone, elevated LH/FSH ratio, and insulin resistance. Other choices are incorrect because they do not align with the typical hormonal profile of PCOS.

## Question #:9

**CLINICAL SCENERIO**: You are asked to review a 58-year-old female type 1 diabetic with hyperglycaemia. Her capillary blood glucose is 12.8 mmol/l, and she’s on Humulin M3. She took her morning insulin at 07:30. At 11:30, the patient denies polyuria/polydipsia, is euvolaemic, stable, and awaiting discharge. Plasma ketones are 0.4mmol/l. Her blood glucose is usually well-controlled.

**QUESTION LINE**: How will you manage this patient?

**OPTIONS**: a) 6 units of actrapi b) Reassurance to the patient and nurse and continue monitoring c) Intravenous insulin sliding scale d) Diabetic ketoacidosis protocol e) Increase Humulin M3 to 32 units before breakfast starting the following day

**CORRECT-CHOICE LINE**: b.

**REASONING**: When managing hyperglycaemia in an insulin dependent diabetic, avoid the use of stat insulin, unless the patient is symptomatic or has high ketones (>0.6 mmol/l). It is safer to increase the ‘usual’ insulin if persistently hyperglycaemic. The patient has had a once off ‘blip’ in blood glucose control. The patient is medically stable, asymptomatic, and the ketones are at a satisfactory level. Therefore the patient and nurse should be reassured, and the blood glucose level monitored. If the patient had frequent high blood sugars then it would be appropriate to titrate the normal insulin up. An intravenous insulin sliding scale is indicated when the patient is undergoing fasting and will be missing more than one meal. The diabetic ketoacidosis (DKA) protocol should be implemented in patients with DKA which is diagnosed when ketones are significantly elevated (> 3 mmol/l) and the patient is acidotic (HCO3 < 15).

**>>DESCRIPTION**: A 58-year-old female with type 1 diabetes and a blood glucose of 12.8 mmol/l is reviewed. She is asymptomatic, euvolaemic, with ketones at 0.4mmol/l, and on Humulin M3. Her blood glucose is usually well-controlled.

**>>OPTIONS**: a) 6 units of actrapi b) Diabetic ketoacidosis protocol c) Increase Humulin M3 to 32 units before breakfast starting the following day d) Intravenous insulin sliding scale e) Reassurance to the patient and nurse and continue monitoring

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Reassurance to the patient and nurse and continue monitoring

**>>REASONING**: The correct answer is to reassure and monitor because the patient is stable, asymptomatic, and has acceptable ketone levels. Stat insulin and DKA protocols are unnecessary, and increasing insulin is not appropriate for a single high reading. Sliding scales are for fasting patients.

## Question #:14

**CLINICAL SCENERIO**: A 37-year-old female presents with 4 days of generally unwell and a recent dysuria. Her urine is foul smelling and dark. She is a known type 1 diabetic with a long-standing subcutaneous insulin regime. Her pH on admission was 7.24, bicarbonate 8 mmol/l and blood glucose 32 mmol/l. Urinary dip leucocytes 2+, nitrites2+ and 4+ ketones. She was started on treatment for diabetic ketoacidosis with intravenous fluids and fixed rate insulin. She also has intravenous antibiotics for a urinary source of sepsis. You are asked to review her blood sugars at 4 hours after treatment was initiated.

**QUESTION LINE**: What should be the aim in managing hyperglycaemia in a diabetic ketoacidosis patient?

**OPTIONS**: a) Reduce blood glucose to under 14 mmol/l as quickly as possible b) Reduce blood glucose by 3mmol/l per hour c) Reduce blood glucose by 6 mmol/l per hour d) Aim blood glucose above 18 mmol/l e) Blood glucose does not require monitoring if insulin infusion is running3

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: The most recent guidelines by the Joint British Diabetes Societies Inpatient Care Group in September 2012 recommends a reduction in blood glucose of 3 mmol/l per hour until BM reaches 14 mmol/l, at which point 5% dextrose should be considered as the intravenous fluid of choice. Rapid glucose lowering should be avoided: the rapid flux in osmolality can result in significant cerebral oedema and resultant cerebral damage.

**>>DESCRIPTION**: A 37-year-old female, known type 1 diabetic, presents with dysuria, foul-smelling dark urine, pH 7.24, bicarbonate 8 mmol/l, and blood glucose 32 mmol/l. Urinary dip shows leucocytes 2+, nitrites 2+, and ketones 4+. She is being treated for DKA and sepsis. You are asked to review her blood sugars after 4 hours of treatment.

**>>OPTIONS**: a) Aim blood glucose above 18 mmol/l b) Blood glucose does not require monitoring if insulin infusion is running c) Reduce blood glucose by 3mmol/l per hour d) Reduce blood glucose by 6 mmol/l per hour e) Reduce blood glucose to under 14 mmol/l as quickly as possible

**>>CORRECT-CHOICE LINE**: Correct answer is c.

**>>CORRECT-CHOICE\_TEXT**: Reduce blood glucose by 3mmol/l per hour

**>>REASONING**: Guidelines recommend reducing blood glucose by 3 mmol/l per hour until it reaches 14 mmol/l. Rapid glucose lowering can cause cerebral edema.

## Question #:201

**CLINICAL SCENERIO**: An 82-year-old gentleman found on the floor, dehydrated with rhabdomyolysis and acute kidney injury. Blood tests show hypernatremia. Intravenous fluids are started.

**QUESTION LINE**: What is the aim of correcting the patient’s hypernatraemia?

**OPTIONS**: a) Reduce blood sodium to under 145 mmol/l as quickly as possible - b) Reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l - c) Reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l - d) Aim for blood sodium above 145 mmol/l - e) Blood sodium does not require monitoring if intravenous fluids is running, CK falling and renal function improving

**CORRECT-CHOICE LINE**: b.

**REASONING**: The correct answer is to reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l . In cases of hypernatraemia, it is important to correct the patient’s sodium levels gradually to avoid complications such as cerebral oedema and central pontine myelinolysis.

The first option is incorrect because rapidly correcting hypernatraemia can lead to severe neurological complications. The second incorrect option suggests a faster rate of correction than recommended and increases the risk of complications. The fourth option contradicts the goal of treating hypernatraemia. Finally, the last option is incorrect because regular monitoring of blood sodium levels during treatment is essential.

**>>DESCRIPTION**: An 82-year-old gentleman with dehydration, rhabdomyolysis, and acute kidney injury presents with hypernatremia. Intravenous fluids have been initiated.

**>>OPTIONS**: a) Aim for blood sodium above 145 mmol/l b) Blood sodium does not require monitoring if intravenous fluids is running, CK falling and renal function improving c) Reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l d) Reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l e) Reduce blood sodium to under 145 mmol/l as quickly as possible

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l

**>>REASONING**: The aim is to gradually reduce sodium levels by 0.5 mmol/hr (no more than 6 mmol/l in 12 hours) to prevent complications. Rapid correction or no monitoring are incorrect and can lead to neurological issues or failure to address the underlying problem.

## Question #:273

**CLINICAL SCENERIO**: A 37-year-old man on hydrocortisone for Addison’s disease presents with ankle swelling, hypertension, and a history of postural dizziness and vomiting. The patient’s hydrocortisone dose was increased, and he reports resolution of original symptoms. What is the most appropriate action?

**QUESTION LINE**: What is the most appropriate action?

**OPTIONS**: a. Start amlodipine b. Start ACE inhibitor c. Reduce dose of hydrocortisone d. Increase dose of hydrocortisone e. Start beta-blocker

**CORRECT-CHOICE LINE**: c

**REASONING**: The patient exhibits signs of over-replacement of corticosteroids, including hypertension and edema, indicating the hydrocortisone dose should be reduced. The reasoning also elaborates on signs of over/under-replacement.

**>>DESCRIPTION**: A 37-year-old man on hydrocortisone for Addison’s disease presents with ankle swelling and hypertension. What is the most appropriate action?

**>>OPTIONS**: a. Start ACE inhibitor b. Start amlodipine c. Reduce dose of hydrocortisone d. Start beta-blocker e. Increase dose of hydrocortisone

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Reduce dose of hydrocortisone

**>>REASONING**: Reduce the hydrocortisone dose due to signs of over-replacement, such as hypertension. Other options are not the primary approach to manage this complication of steroid use.

## Question #:214

**CLINICAL SCENERIO**: A 28-year-old woman presents with flu-like symptoms, palpitations, anterior neck pain, rapid weight loss, and anxiety over 2-3 weeks. Her TSH is <0.05 IU. Examination reveals BP 128/82 mmHg, pulse 95 bpm, fine tremor, and mild anterior neck tenderness. BMI is 22 kg/m².

**QUESTION LINE**: Which of the following would you also expect to find?

**OPTIONS**: a) Erythema nodosum b) Exophthalmos c) Multiple small thyroid nodules on ultrasound scan d) Positive anti-thyroid antibodies e) Reduced uptake on thyroid scintigraphy

**CORRECT-CHOICE LINE**: e

**REASONING**: In De Quervain’s thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy

The most likely diagnosis, given the history of 2-3 weeks of flu-like symptoms and suppressed TSH, is subacute thyroiditis where thyroid inflammation drives increased release of stored thyroid hormone, rather than the clinical picture being due to overproduction of T3 and T4. Symptoms of hyperthyroidism should be managed with beta blockade as required, and there is no role for thioamides. Pain over the thyroid can be managed with non-steroidal anti-inflammatory drugs. After a period of hyperthyroidism, rebound hypothyroidism may be seen, followed by a recovery to euthyroidism.

Erythema nodosum is not associated with subacute thyroiditis. Exophthalmos and positive anti-thyroid antibodies are associated with autoimmune thyroid disease. Multiple small thyroid nodules are a feature of multinodular goitre.

**>>DESCRIPTION**: A 28-year-old woman presents with flu-like symptoms, palpitations, anterior neck pain, rapid weight loss, and anxiety. Her TSH is <0.05 IU. Examination findings include BP 128/82 mmHg, pulse 95 bpm, fine tremor, and mild anterior neck tenderness.

**>>OPTIONS**: a) Erythema nodosum b) Exophthalmos c) Multiple small thyroid nodules on ultrasound scan d) Positive anti-thyroid antibodies e) Reduced uptake on thyroid scintigraphy

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Reduced uptake on thyroid scintigraphy

**>>REASONING**: The correct answer is reduced uptake on thyroid scintigraphy, which is characteristic of subacute thyroiditis. Other options are incorrect: Erythema nodosum is not associated with subacute thyroiditis. Exophthalmos and positive anti-thyroid antibodies suggest autoimmune thyroid disease. Multiple small thyroid nodules are a feature of multinodular goitre.

## Question #:56

**CLINICAL SCENERIO**: A 35-year-old with type 1 diabetes, well-controlled with insulin, is pregnant. She’s concerned about harming the fetus. She discovered the pregnancy one week ago and stopped atorvastatin due to pregnancy concerns. Her HbA1c was 39mmol/mol three months ago. She has been advised to continue with her insulin.

**QUESTION LINE**: What is the most appropriate action to avoid complications of diabetes?

**OPTIONS**: - a) Restart atorvastatin - b) Increase short-acting insulin dose - c) Advise termination of pregnancy - d) Refer for retinal screening - e) Increase basal insulin dose

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Patients with diabetes should have increased frequency of retinal screening during pregnancy due to increased risk of retinopathy

The most appropriate plan is to repeat the retinal screening and test the urine. In diabetes, patients should achieve good diabetic control prior to planning for pregnancy. If this has not been achieved, then NICE advises contraception and to offer termination if pregnancy does occur due to increased risks in pregnancy. This patient has good diabetic control as evidenced by the lack of complications and HbA1c. When anticipating pregnancy, contraindicated drugs such as atorvastatin and ACE inhibitors should be stopped. Labetalol is an appropriate option for blood pressure control. The dose of insulin does not automatically need to be adjusted up or down but blood glucose would need close monitoring during pregnancy. It is advised, however, if the patient has not had retinal screening within the last six months to offer this urgently as there can be rapid development of diabetic retinopathy in pregnancy.

**>>DESCRIPTION**: A 35-year-old with well-controlled type 1 diabetes is pregnant. She stopped atorvastatin. Her HbA1c was 39mmol/mol three months ago. What action is most appropriate to avoid diabetic complications?

**>>OPTIONS**: a) Advise termination of pregnancy b) Increase basal insulin dose c) Increase short-acting insulin dose d) Refer for retinal screening e) Restart atorvastatin

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Refer for retinal screening

**>>REASONING**: Retinal screening is crucial during pregnancy for diabetic patients due to the increased risk of retinopathy. Good diabetic control doesn’t negate this need. Atorvastatin is contraindicated, and insulin adjustments aren’t automatically required, but close glucose monitoring is needed. Termination is not indicated given the patient’s good diabetic control.

## Question #:321

**CLINICAL SCENERIO**: A 45-year-old woman presents to the Emergency Department with abdominal pain, lethargy, and weakness. She smokes and drinks alcohol. Urine dipstick shows protein, blood, and pH 5.5-6.0. Blood tests reveal Hb 13.6 g/dl, Platelet 225 \* 10 9 /l, WBC 8.4 \* 10 9 /l, Neutrophils 6.0 \* 10 9 /l, Lymphocytes 1.9 \* 10 9 /l, Eosinophils 0.3 \* 10 9 /l, Na+ 143 mmol/l, K+ 2.3 mmol/l, Urea 6.1 mmol/l, Creatinine 81 µmol/l, Bicarbonate 7 mmol/l, and Chloride 124 mmol/l. An abdominal film is requested.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Renal tubular acidosis type 1 b) Renal tubular acidosis type 2 c) Renal tubular acidosis type d) Conn’s syndrome e) Bulimia

**CORRECT-CHOICE LINE**: a

**REASONING**: Hypokalaemia, nephrocalcinosis - type 1 renal tubular acidosis

This is probably a useful ‘spot’ diagnosis to learn for the exam: nephrocalcinosis on AXR → renal tubular acidosis (RTA) type 1. As well as nephrocalcinosis, the other pointers to RTA type 1 include hypokalaemia and a normal anion gap or hyperchloraemic metabolic acidosis.

If you weren’t aware of the link between RTA type 1 and nephrocalcinosis another approach would be to work through the causes of 1. metabolic acidosis and 2. hypokalaemia.

## 1. Metabolic acidosis

It is useful to first calculate the anion gap:

Anion gap = (sodium + potassium) - (bicarbonate + chloride)

= (143 + 2.3) - (7 + 124) = 12.3 mmol/l

A normal anion gap is 8-14 mmol/l

**>>DESCRIPTION**: A 45-year-old woman presents with abdominal pain, weakness, and blood test results including hypokalemia (K+ 2.3 mmol/l) and metabolic acidosis (bicarbonate 7 mmol/l). An abdominal film is requested.

**>>OPTIONS**: a) Bulimia b) Conn’s syndrome c) Renal tubular acidosis type 1 d) Renal tubular acidosis type 2 e) Renal tubular acidosis type

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Renal tubular acidosis type 1

**>>REASONING**: The most likely diagnosis is Renal Tubular Acidosis type 1 due to the presence of hypokalemia and the likelihood of nephrocalcinosis on imaging. The other options are not supported by the provided clinical and laboratory data.

## Question #:8

**CLINICAL SCENERIO**: A 76-year-old woman was admitted with shortness of breath, cough, and palpitations, diagnosed with pneumonia and atrial fibrillation. Initial treatment included antibiotics, fluids, and digoxin. Two days later, the patient improved, cardioverted to sinus rhythm, and digoxin was stopped. Thyroid function tests were performed with abnormal results. What is the next investigation?

**QUESTION LINE**: What is the most appropriate next investigation to assess deranged thyroid function tests?

**OPTIONS**: a) Thyroid ultrasound b) Thyroid peroxidase antibody levels c) Repeat TFT in 6 weeks d) Thyroid scintiscanning e) Thyroglobulin antibody levels

**CORRECT-CHOICE LINE**: .c

**REASONING**: Sick euthyroid is common in unwell, elderly patients and often needs no treatment. Acute illnesses can alter thyroid hormone levels. Therefore, it is best to repeat TFTs after recovery from acute illness before further investigation.

**>>DESCRIPTION**: A 76-year-old woman with pneumonia and atrial fibrillation showed abnormal thyroid function tests. What’s the next investigation?

**>>OPTIONS**: a) Thyroid peroxidase antibody levels b) Thyroid scintiscanning c) Repeat TFT in 6 weeks d) Thyroglobulin antibody levels e) Thyroid ultrasound

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Repeat TFT in 6 weeks

**>>REASONING**: The most appropriate next step is to repeat the thyroid function tests (TFTs) in six weeks, as acute illness can transiently affect thyroid hormone levels. Other options are premature without ruling out sick euthyroid state.

## Question #:199

**CLINICAL SCENERIO**: A 34-year-old alcoholic man, found at home covered in vomit and excrement after two weeks, presents with drowsiness, aches, and abnormal ECG findings (prolonged PR and QTc). Examination reveals dry mucous membranes, sunken eyes, and moisture damage. Blood tests show multiple electrolyte imbalances, including low sodium, potassium, calcium, phosphate, and magnesium.

**QUESTION LINE**: What is the priority in the management of this man’s electrolyte disturbances?

**OPTIONS**: a) Replace magnesium intravenously b) Replace potassium intravenously c) Start insulin-dex infusion d) Start oral magnesium, potassium, phosphate, and calcium e) Start telemetry

**CORRECT-CHOICE LINE**: a

**REASONING**: Replace magnesium before correcting hypokalaemia. Hypomagnesemia prevents potassium absorption

In this question, the patient has multiple electrolyte abnormalities due to poor oral intake over the preceding 2 weeks.

Intravenous magnesium replacement would be recommended in this case due to the severity of magnesium deficiency. Severe hypomagnesaemia is defined as levels <0.4 mmol/L.

Although we do need to replace potassium intravenously , hypomagnesemia prevents potassium absorption, so we need to correct this before supplementing potassium.

Dex-saline can be used as part of the treatment of hyperkalaemia. It does not necessarily need to be used in hypokalaemia.

Start oral magnesium, potassium, phosphate, and calcium : this man has severe hypomagnesaemia (<0.4 mmol/L) and severe hypokalaemia (<2.5 mmol/L). He requires intravenous replacement and oral supplementation is not appropriate.

Telemetry would be required, but it is not the main priority here, electrolyte replacement should not be delayed in this case whilst awaiting telemetry.

**>>DESCRIPTION**: A 34-year-old alcoholic man, found at home after two weeks, presents with drowsiness, aches, prolonged PR and QTc on ECG, and multiple electrolyte imbalances (low sodium, potassium, calcium, phosphate, and magnesium).

**>>OPTIONS**: a) Replace magnesium intravenously b) Replace potassium intravenously c) Start insulin-dex infusion d) Start oral magnesium, potassium, phosphate, and calcium e) Start telemetry

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Replace magnesium intravenously

**>>REASONING**: The priority is intravenous magnesium replacement due to severe hypomagnesemia, as it impairs potassium absorption. Potassium replacement is also needed, but magnesium must be corrected first. Insulin-dex infusion is for hyperkalemia. Oral replacement is not appropriate for severe deficiencies, and telemetry, while needed, is not the primary initial intervention.

## Question #:69

**CLINICAL SCENERIO**: A 23-year-old female presents with worsening acne and a marked increase in the development of body and facial hair which she finds very distressing. She is also overweight and is markedly stressed by her physical appearance and the development of stretch marks over her abdomen. She has tried multiple hair removal techniques with only mild success.

On examination, she has a body mass index of 28 kg/m², coarse hair over the anterior and posterior part of her chest and under her chin. Her Blood Pressure is 135/90mmHg.

Her lab results are as follows:

| 9:00 am Cortisol | 345 nmol/l (170 700 nmol/l) |
| --- | --- |
| LH | 17 iU/l (1 20 iU/l) |
| Basal FSH | 7.1 iU/l (1.0 8.8 iU/l) |

| DHEAS | 545 µg/dl (31 228 µg/dl) |
| --- | --- |
| Prolactin | 160 mU/l (<360 mU/l) |
| 17 OH Progesterone | 1025 ng/dl (<80 ng/dl) |
| Testosterone | 3.9 nmol/l (0.9 3.1 nmol/l) |

Ultrasound abdomen and pelvis reveals two cysts in the right ovary.

**QUESTION LINE**: Which of the following is the most appropriate treatment option for her condition?

**OPTIONS**: - a) Combined oral contraceptive pill - b) Finasteride - c) Surgical resection of the ovarian cysts - d) Reverse circadian rhythm steroids - e) Metformin in combination with spironolactone

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: The diagnosis in this scenario is non-classical congenital adrenal hyperplasia which manifests in adolescence/adulthood. It is caused by a deficiency of the enzyme 21 hydroxylase in the steroid biosynthetic pathway. The result is a shift in the production of steroid hormones towards the androgenic pathway. Since cortisol secretion is reduced, feedback leads to increased ACTH production and resultant hyperplasia of the adrenals. The level of the compounds that are formed prior to the action of 21 hydroxylase is increased, therefore levels of 17 hydroxyprogesterone are elevated. Due to excessive androgen production, there is virilization and hirsutism.

Treatment involves steroids given in reverse circadian rhythm, i.e. a higher dosage at night and a lower dose in the morning.

The rationale behind this approach is due to the pathophysiology of CAH. The adrenal hyperplasia and the over-secretion of adrenal androgens are due to excessive ACTH production. When steroids are given in higher doses at night, ACTH is suppressed and the normal physiological steroid peak in the morning is also reduced.

Cysts in the ovaries are a common finding on routine ultrasound and do not necessarily represent polycystic ovarian syndrome.

**>>DESCRIPTION**: A 23-year-old female presents with acne, increased body/facial hair, and stretch marks. BMI is 28 kg/m², with coarse hair on chest/chin and BP 135/90mmHg. Labs: elevated DHEAS, 17-OH progesterone, and testosterone. Ultrasound shows ovarian cysts.

**>>OPTIONS**: a) Combined oral contraceptive pill b) Finasteride c) Metformin in combination with spironolactone d) Reverse circadian rhythm steroids e) Surgical resection of the ovarian cysts

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Reverse circadian rhythm steroids

**>>REASONING**: The patient has non-classical congenital adrenal hyperplasia due to 21-hydroxylase deficiency, leading to increased androgen production. Treatment involves reverse circadian rhythm steroids to suppress ACTH production. Ovarian cysts are a common incidental finding.

## Question #:232

**CLINICAL SCENERIO**: A 55-year-old female with asthma presents with light-headedness and abdominal pain. Examination reveals hypotension and tachycardia. Blood results show hyponatremia and hyperkalemia. Short and long Synacthen tests are performed.

**QUESTION LINE**: What is the most likely cause?

**OPTIONS**: 1) Adrenal Cushing’s syndrome3% 2) Cushing’s disease5% 3) Iatrogenic adrenal insufficiency23% 4) Primary adrenal insufficiency (Addison’s disease)21% 5) Secondary adrenal insufficiency48%

**CORRECT-CHOICE LINE**: 5

**REASONING**: The patient’s presentation and test results are most consistent with secondary adrenal insufficiency. The long Synacthen test showing a gradual rise in cortisol to a peak at 24 hours supports this diagnosis. The patient is on inhaled steroids, but this is less likely to cause adrenal insufficiency unless on a liver enzyme inhibitor. The other options are less likely based on the presented data.

**>>DESCRIPTION**: A 55-year-old female with asthma presents with light-headedness, abdominal pain, hypotension, tachycardia, hyponatremia, and hyperkalemia. Short and long Synacthen tests are performed.

**>>OPTIONS**: 1) Adrenal Cushing’s syndrome 2) Cushing’s disease 3) Iatrogenic adrenal insufficiency 4) Primary adrenal insufficiency (Addison’s disease) 5) Secondary adrenal insufficiency

**>>CORRECT-CHOICE LINE**: 5

**>>CORRECT-CHOICE\_TEXT**: Secondary adrenal insufficiency

**>>REASONING**: Secondary adrenal insufficiency is most likely due to the clinical presentation, laboratory findings, and the long Synacthen test results (gradual cortisol rise). The patient’s use of inhaled steroids is considered, but other causes are deemed less likely.

## Question #:77

**CLINICAL SCENERIO**: A 58-year-old patient with hypertension undergoes neurosurgery for an intracranial haemorrhage. Over the next few days, his serum sodium level progressively declines, reaching 118 mmol/l by the third day despite fluid restriction to 1L per day. Urine osmolarity is 700 mOsmo/l and urinary sodium is raised at 80 mmol/l.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Addisonian crisis b) Secretion of inappropriate antidiuretic hormone c) Cranial diabetes insipidus d) Hypovolaemi e) Fluid overload

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: The hyponatraemia and hypotonic blood plasma, coupled with the raised urine osmolality and raised urinary sodium excretion indicates a diagnosis of syndrome of inappropriate ADH secretion. This is a condition that can occur after head trauma, a central nervous system infection and intracranial haemorrhage.

**>>DESCRIPTION**: A 58-year-old hypertensive patient post-neurosurgery for intracranial hemorrhage develops progressive hyponatremia (118 mmol/l) despite fluid restriction. Urine osmolarity is 700 mOsmo/l, and urinary sodium is 80 mmol/l.

**>>OPTIONS**: a) Addisonian crisis b) Cranial diabetes insipidus c) Fluid overload d) Hypovolaemi e) Secretion of inappropriate antidiuretic hormone

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Secretion of inappropriate antidiuretic hormone

**>>REASONING**: The combination of hyponatremia, hypotonic blood plasma, high urine osmolality, and elevated urinary sodium points to SIADH, which is associated with head trauma and intracranial hemorrhage. Addisonian crisis, diabetes insipidus, hypovolemia and fluid overload do not fit the clinical and lab findings.

## Question #:158

**CLINICAL SCENERIO**: A 52-year-old patient presents with excessive sweating, headaches, and low energy over several months. Examination reveals a large face and tongue. The patient has a history of hypertension and type 2 diabetes.

**QUESTION LINE**: What is the most appropriate first-line investigation

**OPTIONS**: - a) Serum growth hormone - b) Serum IGF-1 - c) Visual field testing - d) Oral glucose tolerance test measuring serum glucose - e) Pituitary MRI

**CORRECT-CHOICE LINE**: b

**REASONING**: Serum IGF-1 levels are now the first-line test for acromegaly

The most appropriate first-line investigation for this patient is serum IGF-1 . The patient’s symptoms and physical examination findings are suggestive of acromegaly, which is caused by excessive secretion of growth hormone (GH) from the pituitary gland. Acromegaly can lead to hypertension, type 2 diabetes, and other complications. Serum IGF-1 (insulin-like growth factor 1) levels are a reliable indicator of GH activity and are more stable than serum GH levels throughout the day. An elevated serum IGF-1 level would support the diagnosis of acromegaly.

Serum growth hormone measurement is not the best first-line investigation because GH secretion is pulsatile, with peaks and troughs throughout the day. This can lead to false-negative results if the blood sample is taken during a period when GH levels are low. Serum IGF-1 levels are less variable and provide a more accurate assessment of overall GH activity.

Visual field testing could be useful in assessing potential compression of the optic chiasm by a pituitary tumour causing acromegaly; however, it is not an appropriate first-line diagnostic test for confirming the presence of acromegaly itself. It would be more relevant once biochemical evidence has been obtained to support the diagnosis.

An oral glucose tolerance test measuring serum glucose may be helpful in evaluating a patient’s diabetes status but does not directly address the suspicion of acromegaly. In patients with confirmed acromegaly, an oral glucose tolerance test measuring serum growth hormone may be used to assess disease activity, as normal individuals should have suppressed GH levels after glucose ingestion, while those with acromegaly will not suppress their GH adequately.

A pituitary MRI could potentially identify a pituitary adenoma as the cause of acromegaly; however, it is not the best first-line investigation. Biochemical confirmation of GH excess should be obtained before performing imaging studies.

Once a diagnosis of acromegaly has been established through elevated serum IGF-1 levels and/or an abnormal oral glucose tolerance test measuring serum growth hormone, a pituitary MRI would then be appropriate to identify the underlying cause and guide further management.

**>>DESCRIPTION**: A 52-year-old patient with excessive sweating, headaches, low energy, a large face, and tongue, and a history of hypertension and type 2 diabetes requires investigation.

**>>OPTIONS**: a) Oral glucose tolerance test measuring serum glucose b) Pituitary MRI c) Serum growth hormone d) Serum IGF-1 e) Visual field testing

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Serum IGF-1

**>>REASONING**: Serum IGF-1 is the first-line test for suspected acromegaly due to its reliability. Serum GH is pulsatile, making it less reliable. Visual field testing and oral glucose tolerance tests are not first-line. Pituitary MRI is indicated after biochemical confirmation.

## Question #:78

**CLINICAL SCENERIO**: A 46-year-old woman presents with polyuria, neck swelling, headaches, sweating, and palpitations. Her heart rate is 115 bpm, BP is 125/85 mmHg. Blood results show hypercalcemia (2.9 mmol/L), normal PTH (4.8 pmol/L), TSH (4.2 mU/L), and free T4 (14.2 pmol/L).

**QUESTION LINE**: What investigation is required?

**OPTIONS**: - a) Radioactive iodine uptake scan - b) Serum calcitonin - c) Serum thyroglobulin - d) Thyroid peroxidase antibodies - e) Thyroid stimulating hormone receptor antibodies

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer

Serum calcitonin is correct. The patient most likely has multiple endocrine neoplasia (MEN) type 2. The presence of hypercalcemia with an ‘inappropriately normal’ PTH is suggestive of primary hyperparathyroidism. The patient also likely has a phaeochromocytoma due to the classic triad of episodic headache, sweating, and tachycardia. The neck swelling is concerning for medullary thyroid cancer which should be investigated with a thyroid ultrasound and serum calcitonin.

Radioactive iodine uptake scan is incorrect. Medullary thyroid cancer cells do not absorb iodine, and therefore radioiodine scans are not used to diagnose this type of cancer.

Serum thyroglobulin is incorrect. Papillary and follicular thyroid cancer release thyroglobulin which can therefore be used as a tumour marker for these types of cancer. However medullary cell cancer does not produce thyroglobulin. Medullary cell cancer cells produce calcitonin which can therefore be used as a tumour marker.

Thyroid peroxidase antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

Thyroid stimulating hormone receptor antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

**>>DESCRIPTION**: A 46-year-old woman has polyuria, neck swelling, headaches, sweating, and palpitations. HR 115, BP 125/85. Labs: hypercalcemia, normal PTH, TSH, and free T4.

**>>OPTIONS**: a) Radioactive iodine uptake scan b) Serum calcitonin c) Serum thyroglobulin d) Thyroid peroxidase antibodies e) Thyroid stimulating hormone receptor antibodies

**>>CORRECT-CHOICE LINE**: b

**>>CORRECT-CHOICE\_TEXT**: Serum calcitonin

**>>REASONING**: Serum calcitonin is correct because the patient likely has MEN type 2, suggested by hypercalcemia with normal PTH and symptoms of pheochromocytoma. Neck swelling raises concern for medullary thyroid cancer, for which serum calcitonin is a tumor marker. Radioactive iodine uptake scan and serum thyroglobulin are incorrect because they are not used for medullary thyroid cancer. Thyroid antibodies are not indicated as patient is euthyroid.

## Question #:229

**CLINICAL SCENERIO**: A 54-year-old woman with recurrent headaches precipitated by exercise or coffee, heat intolerance, and nausea presents with a history of anxiety, constipation, and renal stones. Examination reveals diaphoresis, tachycardia, palpable cervical lymphadenopathy, and elevated blood pressure. Laboratory findings include hypercalcemia.

**QUESTION LINE**: Given the likely diagnosis, which of the following options should form part of the workup for this patient?

**OPTIONS**: - a) MRI head - b) MRI pancreas - c) Serum calcitonin - d) Skeletal survey - e) Ultrasound adrenals

**CORRECT-CHOICE LINE**: c

**REASONING**: MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer. The patient’s presentation, including hypertension, headaches, sweating, and hypercalcemia, is suggestive of MEN type 2A. Serum calcitonin is crucial for detecting medullary thyroid cancer, a common feature of MEN type 2A. Other options are incorrect because they are not relevant to the most likely diagnosis.

**>>DESCRIPTION**: A 54-year-old woman with recurrent headaches (exercise/coffee-related), heat intolerance, nausea, history of anxiety, constipation, and renal stones, presents with diaphoresis, tachycardia, palpable cervical lymphadenopathy, elevated blood pressure and hypercalcemia.

**>>OPTIONS**: a) MRI head b) MRI pancreas c) Serum calcitonin d) Skeletal survey e) Ultrasound adrenals

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Serum calcitonin

**>>REASONING**: The correct answer is serum calcitonin because it’s essential for workup of MEN type II to exclude medullary thyroid cancer. Incorrect options are not relevant to the likely diagnosis or utilize the wrong imaging modality.

## Question #:3

**CLINICAL SCENERIO**: A 29-year-old nulligravida woman presents with 6 months of amenorrhea. She has no significant medical history, normal vital signs, and a negative pregnancy test. Physical exam is unremarkable.

**QUESTION LINE**: Which of the following is the most appropriate screening test for this patient?

**OPTIONS**: a) Karyotype b) MRI of the pituitary c) Serum 17-hydroxyprogesterone d) Serum prolactin e) Ultrasound of the pelvis

**CORRECT-CHOICE LINE**: d

**REASONING**: The patient presents with secondary amenorrhea. After a negative pregnancy test, the next step is to check prolactin, TSH and FSH. Serum prolactin is indicated to rule out hyperprolactinemia. Karyotype is considered in cases of suspected genetic syndromes. MRI of the pituitary is indicated with persistently elevated prolactin and neurological symptoms. 17-hydroxyprogesterone is checked when congenital adrenal hyperplasia is suspected. Ultrasound is useful for pelvic abnormalities.

**>>DESCRIPTION**: A 29-year-old woman presents with 6 months of amenorrhea and a negative pregnancy test. Physical exam is unremarkable.

**>>OPTIONS**: a) Karyotype b) MRI of the pituitary c) Serum 17-hydroxyprogesterone d) Serum prolactin e) Ultrasound of the pelvis

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Serum prolactin

**>>REASONING**: The most appropriate initial screening test is serum prolactin to evaluate for hyperprolactinemia, a common cause of secondary amenorrhea. Other options are less appropriate as initial screening tests: Karyotype is for suspected genetic syndromes; MRI for pituitary abnormalities; 17-hydroxyprogesterone for suspected congenital adrenal hyperplasia; and ultrasound for pelvic abnormalities.

## Question #:89

**CLINICAL SCENERIO**: A 42-year-old man presents to endocrinology clinic. He has been seeing his GP due to developing gynaecomastia. At first, this was thought to be due to alcohol excess and possible liver involvement, but after stopping alcohol completely and normal liver function tests this diagnosis was excluded. He has no previous medical history apart from having had a tibial fracture a year ago, and having been diagnosed with migraines two months ago. He takes paracetamol for the migraines but finds this to be not very helpful, especially with the headaches at night. On more direct questioning he has also developed problems maintaining an erection. On examination he does have gynaecomastia, but apart from this he appears normal. On repeating blood tests in the clinic his liver function tests are normal, but morning serum testosterone is low, with normal FSH and LH. What further investigation is likely to be diagnostic?

**QUESTION LINE**: What further investigation is likely to be diagnostic?

**OPTIONS**: - a) Liver biopsy - b) Transferrin saturations - c) Serum prolactin - d) CT brain - e) Thyroid function tests

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Hypogonadism secondary to prolactinaemia would be associated with low testosterone and low or normal FSH and LH

This patient has symptoms of hypogonadism with erectile dysfunction and gynaecomastia with a suggested evidence of a headache suspicious for an intracranial lesion, especially with a new diagnosis of headaches at night. The most likely explanation is a prolactinoma, which can be detected by very high prolactin levels. After this, an MRI of the pituitary would be appropriate. A CT brain is unlikely to be able to demonstrate any lesions. A liver biopsy could be considered if there was liver failure and it was unexplained. Transferrin saturation could be considered if haemochromatosis was considered.

**>>DESCRIPTION**: A 42-year-old man with gynaecomastia, erectile dysfunction, and new nocturnal headaches presents with low testosterone and normal FSH/LH. Liver function tests are normal. What is the next most appropriate diagnostic investigation?

**>>OPTIONS**: a) CT brain b) Liver biopsy c) Serum prolactin d) Thyroid function tests e) Transferrin saturations

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Serum prolactin

**>>REASONING**: The patient’s symptoms (gynaecomastia, erectile dysfunction, nocturnal headaches) and lab results (low testosterone, normal FSH/LH) suggest hypogonadism secondary to a prolactinoma. Serum prolactin is the most appropriate next step. CT brain is less sensitive than MRI for pituitary lesions. Liver biopsy and transferrin saturation are not indicated based on the provided information.

## Question #:322

**CLINICAL SCENERIO**: A 29-year-old woman with amenorrhea, negative pregnancy test, undetectable TSH, elevated free T4 (48ng/dl), negative thyroid-stimulating hormone receptor antibodies, and diffuse thyroid uptake on scan, diagnosed with Graves’ disease, opts for radioiodine treatment due to a history of hepatitis C. What is the most appropriate advice regarding conception post-treatment?

**QUESTION LINE**: What is the most appropriate advise?

**OPTIONS**: a) Any future pregnancy will have increased risk of foetal malformation b) She should avoid becoming pregnant for six months after treatment c) She should avoid becoming pregnant for four months after treatment d) Fertility may be adversely affected due to treatment e) She should always avoid breastfeeding

**CORRECT-CHOICE LINE**: b

**REASONING**: Following radioiodine treatment, patients should avoid becoming pregnant for at least 6 months. This patient has a diagnosis of Graves’ disease based on the biochemical evidence of hyperthyroidism and the diffuse radioiodine uptake scan. Radioiodine is an appropriate treatment choice, but in the absence of severe hepatic dysfunction carbimazole can also be used. During radioiodine treatment and for three weeks following the treatment, she should avoid contact with small children and pregnant women. She should avoid becoming pregnant for six months following treatment. Following this, the radioiodine carries no risk for her foetus or children. A man on radioiodine treatment should avoid fathering a child for up to four months following treatment. It is important that Graves’ disease is biochemically controlled during pregnancy as thyroid-stimulating hormone receptor antibodies can cross the placenta.

**>>DESCRIPTION**: A 29-year-old woman with Graves’ disease, treated with radioiodine due to hepatitis C history, is concerned about conception after treatment. What is the most appropriate advice?

**>>OPTIONS**: a) Any future pregnancy will have increased risk of foetal malformation b) Fertility may be adversely affected due to treatment c) She should always avoid breastfeeding d) She should avoid becoming pregnant for four months after treatment e) She should avoid becoming pregnant for six months after treatment

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: She should avoid becoming pregnant for six months after treatment

**>>REASONING**: Patients should avoid pregnancy for six months after radioiodine treatment. The treatment carries no risk to the fetus or children after this period.

## Question #:163

**CLINICAL SCENERIO**: A 28-year-old lady with cold intolerance, fatigue, and low mood has type 1 diabetes and coeliac disease. ECG shows bradycardia. Blood tests reveal elevated TSH, low T3/T4, and macrocytosis.

**QUESTION LINE**: What is the next best step in her management?

**OPTIONS**: a) Commence levothyroxine b) Commence carbimazole c) Ultrasound scan thyroid d) MRI pituitary e) Short synacthen test

**CORRECT-CHOICE LINE**: e

**REASONING**: The short synacthen test is the best test to diagnose Addison’s disease. Given the patient’s autoimmune history (type 1 diabetes, coeliac disease) and hypothyroid findings, Addison’s should be ruled out before treating the hypothyroidism to avoid adrenal crisis.

**>>DESCRIPTION**: A 28-year-old woman with fatigue, cold intolerance, type 1 diabetes, and coeliac disease presents with bradycardia, elevated TSH, low T3/T4, and macrocytosis.

**>>OPTIONS**: a) Commence carbimazole b) Commence levothyroxine c) MRI pituitary d) Short synacthen test e) Ultrasound scan thyroid

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Short synacthen test

**>>REASONING**: The best next step is a short synacthen test to rule out Addison’s disease, which can coexist with autoimmune hypothyroidism. Incorrect choices involve treating hypothyroidism before ruling out adrenal insufficiency.

## Question #:198

**CLINICAL SCENERIO**: A 22-year-old female with type 1 diabetes presents with weight loss, anorexia, and fatigue over six months. She experienced decreased insulin needs, hypoglycemic attacks, weight loss, and amenorrhea. Examination reveals a thin build with a pulse of 70 bpm and a blood pressure of 110/70 mmHg with postural drop. Investigations show abnormal electrolyte levels, elevated prolactin, and other hormonal imbalances.

**QUESTION LINE**: What is the most appropriate investigation for this patient?

**OPTIONS**: a) Thyroid autoantibodies b) PTH concentration c) Short synacthen test d) Pregnancy test e) Random cortisol concentration

**CORRECT-CHOICE LINE**: c

**REASONING**: This known type 1 diabetic female has developed Addisons disease on top of her diabetes. This explains the hypoglycaemic attacks, the decrease in her insulin requirement, the fatigue and weight loss.

Both type 1 DM and Addisons disease are features of Schmidt’s disease (type 2 autoimmune polyendocrine syndrome) which is the diagnosis in this case.

The low T4, raised TSH, high calcium, low FSH, low LH, low oestradiol (hypogonadotrophic hypogonadism) are all features of Addisons disease.

The best investigation to diagnose Addisons disease is the short synacthen test. It is of paramount importance when treating these patients is not to replace thyroxine before hydrocortisone because this will induce Addisonian crisis.

**>>DESCRIPTION**: A 22-year-old female with type 1 diabetes presents with weight loss, anorexia, fatigue, decreased insulin needs, hypoglycemic attacks, and amenorrhea. Examination and investigations reveal hormonal imbalances.

**>>OPTIONS**: a) PTH concentration b) Pregnancy test c) Random cortisol concentration d) Short synacthen test e) Thyroid autoantibodies

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Short synacthen test

**>>REASONING**: The patient likely has Addison’s disease, and the most appropriate investigation is a short synacthen test. Other options are less relevant to the suspected diagnosis of adrenal insufficiency.

## Question #:298

**CLINICAL SCENERIO**: A 23-year-old female presents with worsening acne, hirsutism, and irregular periods over 3 years. Examination reveals a BMI of 29kg/m², BP 135/85 mmHg, hirsutism, acanthosis nigricans, and mild clitoromegaly. Blood tests show elevated 17OH-progesterone and free testosterone. What is the most useful next test?

**QUESTION LINE**: What is the single most useful test?

**OPTIONS**: - a. CT adrenals - b. Karyotype - c. Pelvic USS - d. Short synacthen test - e. MRI pituitary

**CORRECT-CHOICE LINE**: d.

**REASONING**: The patient presents with signs and symptoms of hyperandrogenism and elevated 17OH-progesterone levels, which suggests the possibility of non-classical congenital adrenal hyperplasia (CAH). The short synacthen test is used to diagnose CAH. It involves measuring 17OH-progesterone before and after ACTH stimulation. Other options are less directly relevant to the suspected diagnosis.

**>>DESCRIPTION**: A 23-year-old female with acne, hirsutism, irregular periods, elevated 17OH-progesterone, and free testosterone. What’s the best next test?

**>>OPTIONS**: a. CT adrenals b. Karyotype c. Pelvic USS d. Short synacthen test e. MRI pituitary

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Short synacthen test

**>>REASONING**: The short synacthen test is the most helpful test for the diagnosis of congenital adrenal hyperplasia (CAH), particularly when 17OH-progesterone is only modestly elevated. The test measures 17OH-progesterone levels before and after ACTH stimulation. Other tests address different diagnoses and are less appropriate in this case.

## Question #:153

**CLINICAL SCENERIO**: A 65-year-old man presents with polyuria and polydipsia. He has a history of surgically treated bladder cancer, hypertension, and chronic kidney disease. His medications include amlodipine, furosemide, atenolol, and ramipril. Blood tests show elevated HbA1c and impaired renal function.

**QUESTION LINE**: What is the most appropriate medication to commence?

**OPTIONS**: - a) Empagliflozin - b) Insulin - c) Metformin - d) Pioglitazone - e) Sitagliptin

**CORRECT-CHOICE LINE**: e

**REASONING**: Sitagliptin is correct because the patient has type 2 diabetes with an HbA1c > 48 mmol/mol, and Metformin is contraindicated due to severe renal impairment. Sitagliptin can be used first-line when Metformin is contraindicated, and its dosage can be adjusted for reduced eGFR. Metformin is incorrect due to renal impairment. Empagliflozin is not typically initiated if eGFR is < 45ml/minute. Insulin is not typically first-line. Pioglitazone is contraindicated with a history of bladder cancer.

**>>DESCRIPTION**: A 65-year-old man with polyuria, polydipsia, a history of bladder cancer, hypertension, chronic kidney disease, and elevated HbA1c, requires new medication.

**>>OPTIONS**: a) Empagliflozin b) Insulin c) Metformin d) Pioglitazone e) Sitagliptin

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Sitagliptin

**>>REASONING**: Sitagliptin is the correct choice because Metformin is contraindicated due to renal impairment, and Sitagliptin can be initiated first-line and adjusted for kidney function. Other options are contraindicated or not first-line choices.

## Question #:247

**CLINICAL SCENERIO**: A 55-year-old woman admitted after a fall while ice-skating, with a scaphoid fracture. She reported feeling unwell for months and was hospitalized a month prior for nephrolithiasis. Laboratory results are provided, including blood gas and urine dip analysis.

**QUESTION LINE**: From the following options, what would be the most likely cause of her biochemical abnormalities?

**OPTIONS**: a) Carbonic anhydrase II deficiency b) Fanconi syndrome c) Hypoaldosteronism d) Sjogren’s syndrome e) Topiramate use

**CORRECT-CHOICE LINE**: d

**REASONING**: Sjogren’s syndrome is the most likely cause, as it can lead to type 1 renal tubular acidosis (RTA), presenting with normal anion gap metabolic acidosis (NAGMA) and hypokalemia, matching the patient’s findings. The other options are incorrect because they don’t align with the patient’s presentation of NAGMA and hypokalemia, or are less likely.

**>>DESCRIPTION**: A 55-year-old woman presents with a scaphoid fracture after a fall and a history of nephrolithiasis, with lab results including blood gas and urine dip analysis. What’s the most likely cause of her biochemical abnormalities?

**>>OPTIONS**: a) Carbonic anhydrase II deficiency b) Fanconi syndrome c) Hypoaldosteronism d) Sjogren’s syndrome e) Topiramate use

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Sjogren’s syndrome

**>>REASONING**: Sjogren’s syndrome is correct as it causes type 1 renal tubular acidosis (NAGMA and hypokalemia). The other options are less likely or don’t fit the presentation.

## Question #:335

**CLINICAL SCENERIO**: A 49-year-old man presents with fatigue, weakness, and weight loss. He reports increased thirst, frequent urination, and a rash around the groin and buttocks. Fasting blood glucose is 9.2 mm/l.

**QUESTION LINE**: What is the next most appropriate investigation?

**OPTIONS**: f) Plasma insulin level g) Tissue transglutaminase antibody (TTA) test h) Plasma glucagon level i) Skin biopsy j) Plasma zinc level

**CORRECT-CHOICE LINE**: c

**REASONING**: This patient has a diagnosis of a glucagonoma. The polydipsia and polyuria, coupled with the fasting blood glucose result suggest diabetes and the rash is necrolytic migratory erythema a symptom which is the presenting problem in up to 70% of cases of glucagonoma.

**>>DESCRIPTION**: A 49-year-old man presents with fatigue, weight loss, increased thirst, frequent urination, a rash, and a fasting blood glucose of 9.2 mm/l. What is the next most appropriate investigation?

**>>OPTIONS**: a) Plasma glucagon level b) Plasma insulin level c) Plasma zinc level d) Skin biopsy e) Tissue transglutaminase antibody (TTA) test

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Skin biopsy

**>>REASONING**: The patient’s presentation, including diabetes, rash, and history suggests glucagonoma. A skin biopsy is the next most appropriate investigation to confirm the diagnosis.

## Question #:103

**CLINICAL SCENERIO**: A 70-year-old man with a history of smoking 15 cigarettes/day presents with drowsiness, weight loss and a persistent cough. His investigations show:

| Na + | 115 mmol/l | 135-145 mmol/l |
| --- | --- | --- |
| K + | 5.1 mmol/l | 3.5 - 5.0 mmol/l |
| Urea | 3 mmol/l | 2.0-7 mmol/l |
| Creatinine | 74 µmol/l | 55-120 µmol/l |

| Plasma osmolality | 270 mOsm/kg | 285-295 mOsm/kg |
| --- | --- | --- |
| Urine osmolality | 1210 | 500 - 800 mOsm/kg |

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Small cell lung cancer - b) Hypothyroidism - c) Encephalitis - d) Congestive cardiac failure - e) Squamous cell carcinoma

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: A common endocrine complication of small cell lung cancer is SIADH

Hyponatraemia, reduced plasma osmolality and increased urine osmolality are suggestive of syndrome of inappropriate ADH secretion (SIADH).

The increase in ADH causes more aquaporin utilisation in the collecting duct system of the kidney. This causes more water to be retained, diluting the electrolytes in the blood and making the electrolytes in the urine more concentrated.

Small cell lung cancer is a common cause of SIADH and is the most likely diagnosis in this man with an extensive smoking history, cough and weight loss.

**>>DESCRIPTION**: A 70-year-old man, smoker (15 cigarettes/day), presents with drowsiness, weight loss, and cough. Labs: Na+ 115 mmol/l, Plasma osmolality 270 mOsm/kg, Urine osmolality 1210 mOsm/kg.

**>>OPTIONS**: a) Congestive cardiac failure b) Encephalitis c) Hypothyroidism d) Small cell lung cancer e) Squamous cell carcinoma

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Small cell lung cancer

**>>REASONING**: Hyponatremia, low plasma osmolality, and high urine osmolality suggest SIADH. Small cell lung cancer is a common cause of SIADH, making it the most likely diagnosis given the patient’s history.

## Question #:328

**CLINICAL SCENERIO**: A 70-year-old man with a history of smoking 15 cigarettes/day presents with drowsiness, weight loss and a persistent cough. His investigations show:

| Na + | 115 mmol/l | 135-145 mmol/l |
| --- | --- | --- |
| K + | 5.1 mmol/l | 3.5 - 5.0 mmol/l |
| Urea | 3 mmol/l | 2.0-7 mmol/l |
| Creatinine | 74 µmol/l | 55-120 µmol/l |

| Plasma osmolality | 270 mOsm/kg | 285-295 mOsm/kg |
| --- | --- | --- |
| Urine osmolality | 1210 | 500 - 800 mOsm/kg |

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - f) Small cell lung cancer - g) Hypothyroidism - h) Encephalitis - i) Congestive cardiac failure - j) Squamous cell carcinoma

**CORRECT-CHOICE LINE**: e

**REASONING**: A common endocrine complication of small cell lung cancer is SIADH

Hyponatraemia, reduced plasma osmolality and increased urine osmolality are suggestive of syndrome of inappropriate ADH secretion (SIADH).

The increase in ADH causes more aquaporin utilisation in the collecting duct system of the kidney. This causes more water to be retained, diluting the electrolytes in the blood and making the electrolytes in the urine more concentrated.

Small cell lung cancer is a common cause of SIADH and is the most likely diagnosis in this man with an extensive smoking history, cough and weight loss.

**>>DESCRIPTION**: A 70-year-old man with a history of smoking presents with drowsiness, weight loss, persistent cough, and the following lab results: Hyponatremia, reduced plasma osmolality and high urine osmolality. What is the most likely diagnosis?

**>>OPTIONS**: a) Congestive cardiac failure b) Encephalitis c) Hypothyroidism d) Small cell lung cancer e) Squamous cell carcinoma

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Small cell lung cancer

**>>REASONING**: The most likely diagnosis is small cell lung cancer due to the presence of SIADH (syndrome of inappropriate ADH secretion), which is associated with hyponatremia, low plasma osmolality, and high urine osmolality. This is a common endocrine complication of Small Cell Lung Cancer. Other options are less likely given the clinical presentation and lab findings.

## Question #:82

**CLINICAL SCENERIO**: A 45-year-old female with a history of asthma, rheumatoid arthritis, and Sjogren’s syndrome presents with right-sided flank pain. A CT KUB confirms a right-sided kidney stone. Her medications include salbutamol, methotrexate, and hydroxychloroquine. Blood results show: Hb 115 g/l, Platelets 460 \* 10 9 /l, Bicarbonate 16 mEq/L, Chloride 115mmol/l, Na+ 136 mmol/l, K+ 3.1 mmol/l, Urea 7.8 mmol/l, Creatinine 76 µmol/l.

**QUESTION LINE**: How would you prevent further kidney stones?

**OPTIONS**: - a) Thiazide diuretics - b) Loop diuretics - c) Penicillamine - d) Encourage increased fluid intake - e) Sodium bicarbonate3

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate

The patient has a low bicarbonate which confirms a metabolic acidosis. This should prompt you to work out the anion gap which can be extremely useful diagnostically.

The anion gap in this example = sum of positive cations - sum of negative anions = (Na + K) - (HCO3 + Cl) = 9.1 We are therefore dealing with a normal anion gap metabolic acidosis (NAGMA).

High anion gap metabolic acidosis (HAGMA) is generally caused by the following conditions which can be remembered with the mnemonic KULT

*  Ketoacidosis: diabetic, starvation or alcoholic
*  Uraemia
*  Lactic acidosis
*  Toxins e.g. salicylates, ethylene glycol, methanol

Normal anion gap metabolic acidosis (NAGMA) is generally caused by the following conditions which can be remembered with the mnemonic ABCD

*  Addison’s disease
*  Bicarbonate loss via GI tract (e.g. diarrhoea) or renal tract (e.g. renal tubular acidosis)
*  Chloride excess - hyperchloraemic acidosis
*  Drugs e.g. acetazolamide

In this case, the NAGMA, hypokalaemia, and renal stones should prompt you to consider renal tubular acidosis (RTA). The presence of kidney stones, and the past medical history of Sjogren’s syndrome and rheumatoid arthritis make RTA type 1 (distal) the most likely diagnosis. Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate.

**>>DESCRIPTION**: A 45-year-old female with asthma, rheumatoid arthritis, and Sjogren’s presents with right flank pain and a right kidney stone on CT KUB. Medications include salbutamol, methotrexate, and hydroxychloroquine. Labs: Bicarbonate 16 mEq/L, K+ 3.1 mmol/l, Na+ 136 mmol/l, Cl 115mmol/l.

**>>OPTIONS**: a) Encourage increased fluid intake b) Loop diuretics c) Penicillamine d) Sodium bicarbonate e) Thiazide diuretics

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Sodium bicarbonate

**>>REASONING**: The patient’s normal anion gap metabolic acidosis (NAGMA), hypokalemia, and kidney stones, along with a history of Sjogren’s and rheumatoid arthritis, suggest renal tubular acidosis (RTA) type 1. Treatment involves correcting the acidaemia with oral sodium bicarbonate.

## Question #:278

**CLINICAL SCENERIO**: A 66-year-old man with resistant hypertension and hypokalaemia, currently on multiple medications, presents with bilateral adrenal enlargement and elevated aldosterone: renin ratio. Laboratory findings are provided.

**QUESTION LINE**: What is the most appropriate treatment?

**OPTIONS**: a) Chemotherapy b) IV hydrocortisone c) Radiotherapy d) Spironolactone e) Surgery

**CORRECT-CHOICE LINE**: D

**REASONING**: Spironolactone is the correct answer. Primary hyperaldosteronism caused by bilateral adrenal hyperplasia is treated by a mineralocorticoid receptor antagonist such as spironolactone. Chemotherapy is the wrong answer. The first-line treatment for bilateral adrenal hyperplasia causing primary hyperaldosteronism is a mineralocorticoid receptor antagonist such as spironolactone. If a patient had disseminated malignancy caused by an adrenal carcinoma, then this may be an option. IV hydrocortisone is incorrect. This is the treatment for acute hypoadrenalism i.e. an Addisonian crisis. The patient’s cortisol is within normal limits and he has no clinical features of hypoadrenalism. Radiotherapy is incorrect. This type of treatment may be indicated for adrenal carcinoma. However, cancer is more likely to be a unilateral mass-like lesion, rather than bilateral diffuse enlargement. Surgery is incorrect. This would be the correct option if the patient had a unilateral adrenal adenoma secreting excess aldosterone. This is not the case here.

**>>DESCRIPTION**: A 66-year-old man with resistant hypertension, hypokalemia, and bilateral adrenal enlargement has an elevated aldosterone:renin ratio. What is the most appropriate treatment?

**>>OPTIONS**: a) Chemotherapy b) IV hydrocortisone c) Radiotherapy d) Spironolactone e) Surgery

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Spironolactone

**>>REASONING**: Spironolactone is the correct treatment for primary hyperaldosteronism caused by bilateral adrenal hyperplasia. Incorrect choices include chemotherapy, IV hydrocortisone, radiotherapy, and surgery, each addressing different conditions or scenarios.

## Question #:316

**CLINICAL SCENERIO**: A 36-year-old woman with difficult-to-control hypertension, occasional aches and pains in arms and legs, and increased urinary frequency/urgency, presents with a blood pressure of 175/95 mmHg. She is on amlodipine and lisinopril. Blood tests show normal electrolytes, urea, and creatinine. An aldosterone/renin test reveals an increased ratio, and a CT abdomen shows bilateral adrenal hyperplasia.

**QUESTION LINE**: What is the most appropriate management of this?

**OPTIONS**: 1. Bilateral adrenalectomy 2. Unilateral adrenalectomy 3. Watch and wait 4. Low sodium diet 5. Spironolactone

**CORRECT-CHOICE LINE**: 5

**REASONING**: Primary hyperaldosteronism: manage with spironolactone The correct answer is spironolactone. Bilateral adrenal hyperplasia can be safely and successfully managed with an aldosterone antagonist (either spironolactone or eplerenone) as this will reduce the effects of aldosterone. There is no indication for surgical management in the treatment of bilateral adrenal hyperplasia so there is no rationale for an adrenalectomy. A low sodium diet is a reasonable lifestyle modification for hypertensive patients, but there is an underlying cause so medical management is more appropriate in this case. Watch and wait would be inappropriate as there is a clear diagnosis and a treatment.

**>>DESCRIPTION**: A 36-year-old woman with uncontrolled hypertension, aches, increased urinary frequency, and bilateral adrenal hyperplasia (on CT) has an increased aldosterone to renin ratio. What’s the best management?

**>>OPTIONS**: 1. Bilateral adrenalectomy 2. Low sodium diet 3. Spironolactone 4. Unilateral adrenalectomy 5. Watch and wait

**>>CORRECT-CHOICE LINE**: 3

**>>CORRECT-CHOICE\_TEXT**: Spironolactone

**>>REASONING**: The best management is spironolactone, an aldosterone antagonist, to counteract the effects of excess aldosterone in bilateral adrenal hyperplasia. Adrenalectomy is not indicated. A low sodium diet is insufficient. ‘Watch and wait’ is inappropriate given the clear diagnosis.

## Question #:248

**CLINICAL SCENERIO**: A 45-year-old woman presents to the ED with fever, productive cough, confusion, and agitation. She has community-acquired pneumonia and untreated hyperthyroidism (diagnosed recently). Examination reveals tachycardia (170 bpm), hypotension, fever (39.2°C), and left-sided crepitations. Outpatient blood tests show undetectable TSH and elevated free T4. The patient has not improved with IV antibiotics and fluids.

**QUESTION LINE**: What is the most appropriate immediate treatment?

**OPTIONS**: a. Change to IV meropenem and monitor b. Start carbimazole c. Urgent referral for thyroidectomy d. Start IV propranolol e. IV adenosine

**CORRECT-CHOICE LINE**: D

**REASONING**: In thyroid storm with IV beta-blockers are a important first-line treatment

This is a patient with severe confusion, fever and evidence of chest infection and untreated hyperthyroidism. The clinical history suggests that she has developed a thyroid storm as she has fever, severe tachycardia, confusion and agitation with hyperthyroidism. This is a medical emergency, and the most urgent matter is to control her heart rate which is likely the cause for her low blood pressure. IV beta-blocker should be started for this purpose, and propranolol would further help reduce the conversion of T3 into T4. In addition, fluids and dexamethasone should be given, as well as anti-thyroid medications.

Changing to meropenem is unlikely to add any further benefit as the effects of the co-amoxiclav probably have not been seen yet. As the patient is not stable it is not appropriate to refer for surgery as management, but may be a viable longterm strategy to treat her hyperthyroidism. Starting carbimazole now is not the most appropriate action as the patient is unstable and this needs to be corrected first. Adenosine is would be appropriate for a patient with SVT without infection and thyrotoxicosis

**>>DESCRIPTION**: A 45-year-old woman with community-acquired pneumonia and untreated hyperthyroidism presents with fever, productive cough, confusion, agitation, tachycardia, hypotension, and elevated free T4. She has not improved with initial treatment.

**>>OPTIONS**: a. Change to IV meropenem and monitor b. Start carbimazole c. Urgent referral for thyroidectomy d. Start IV propranolol e. IV adenosine

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Start IV propranolol

**>>REASONING**: The most appropriate immediate treatment is IV propranolol, a beta-blocker, to control the patient’s heart rate in thyroid storm, which is causing her instability. Other options are less appropriate: meropenem is unlikely to help immediately, carbimazole is for long-term management, thyroidectomy is not immediate, and adenosine is for SVT, not thyrotoxicosis.

## Question #:12

**CLINICAL SCENERIO**: A 62-year-old patient with type 2 diabetes for 10 years, previous appendectomy, kidney stones, tibial fracture, and depression (remission) presents to the diabetes clinic. He takes metformin and gliclazide, has no allergies, an HbA1c of 40mmol/mol, negative urine dip, and a blood pressure of 151/96mmHg. He previously tried diet and exercise for blood pressure control. What is the most appropriate blood pressure management plan?

**QUESTION LINE**: What is the most appropriate plan in regards to blood pressure management?

**OPTIONS**: - a) Start a beta-blocke - b) Start a thiazide-like diuretic - c) Start an ACE inhibitor - d) Start an angiotensin II receptor blocker - e) Start a calcium channel blocker

**CORRECT-CHOICE LINE**: c

**REASONING**: A patient with T2DM with a new diagnosis of hypertension should be treated with an ACE inhibitor as first line treatment, regardless of age

The correct answer is to start a ACE inhibitor. This is because he has diabetes. A patient with diabetes should have an ACE inhibitor as first line treatment for diabetes regardless of their age. If the patient did not have diabetes, then a calcium channel blocker would be the most appropriate option. If the patient could not tolerate a ACE inhibitor then an angiotensin II receptor blocker should be tried. ACE inhibitors should also start for patients with diabetes if there is proteinuria, and therefore should have at least annual urinary albumin to creatinine ratios measured. Beta-blockers are only used after ACE inhibitors, calcium channel blockers, diuretics and alpha blockers have failed to controlled blood pressure. However, IV labetalol can be used in hypertensive emergencies to reduce blood pressure.

**>>DESCRIPTION**: A 62-year-old patient with type 2 diabetes presents with hypertension (151/96 mmHg). He is on metformin and gliclazide. What is the best blood pressure management?

**>>OPTIONS**: a) Start an ACE inhibitor b) Start an angiotensin II receptor blocker c) Start a beta-blocke d) Start a calcium channel blocker e) Start a thiazide-like diuretic

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Start an ACE inhibitor

**>>REASONING**: An ACE inhibitor is the first-line treatment for hypertensive patients with type 2 diabetes, regardless of age. Other options are considered after ACE inhibitors.

## Question #:224

**CLINICAL SCENERIO**: A 59-year-old man with type 2 diabetes, gout, obesity, and cholecystectomy, taking metformin and allopurinol, presents with concern about his brother’s heart attack. He smokes five cigarettes daily and has a blood pressure of 132/71mmHg.

**QUESTION LINE**: What is the most appropriate action?

**OPTIONS**: - a) Start atorvastatin 20mg ON - b) Start atorvastatin 80mg ON - c) Start aspirin 75mg OD - d) Start clopidogrel 75mg OD - e) Start ezetimibe 10mg OD

**CORRECT-CHOICE LINE**: a

**REASONING**: Patients with T2DM should not be routinely started on statins; their QRISK2 score should be calculated

This is a patient with high risk in terms of cardiovascular disease due to the fact that he is male, has obesity, is a current smoker and has a strong family history of heart disease. Therefore it is very important to manage his risk factors. In patients with type 2 diabetes, risk should be assessed using the QRISK2 method. This formula uses cardiovascular risk factors and estimates risk of cardiovascular ischaemia within the next 10 years. If you were to assess this patient, his risk would be far greater than 25%. NICE guidelines advises that if the risk is greater than 10% then Atorvastatin 20mg at night should be started, and increased if the cholesterol does not fall. Starting 80mg of Atorvastatin would be appropriate in secondary prevention, such as following a myocardial infarction. NICE does not advise starting aspirin or clopidogrel as primary prevention unless there is a high risk for an ischaemic event, as determined by QRISK scoring, and the patient is hypertensive or being treated for hypertension. This is because the risk of gastrointestinal bleeding is thought to outweigh the benefit of reduction in cardiovascular events in such patients.

**>>DESCRIPTION**: A 59-year-old man with type 2 diabetes, gout, obesity, and a family history of heart attack presents, smokes five cigarettes daily, and has a blood pressure of 132/71mmHg. What is the best course of action?

**>>OPTIONS**: a) Start atorvastatin 20mg ON b) Start atorvastatin 80mg ON c) Start aspirin 75mg OD d) Start clopidogrel 75mg OD e) Start ezetimibe 10mg OD

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Start atorvastatin 20mg ON

**>>REASONING**: Initiate atorvastatin 20mg ON due to high cardiovascular risk (male, obesity, smoking, family history) and diabetes. QRISK2 score should be assessed. Atorvastatin 80mg is for secondary prevention. Aspirin and clopidogrel are not first-line for primary prevention in this scenario due to bleeding risk.

## Question #:5

**CLINICAL SCENERIO**: A 38-year-old woman with type 1 diabetes mellitus, controlled with a basal-bolus regime and citalopram 20mg od for depression, presents to clinic. She was diagnosed with type 1 diabetes at age 13. Recent bloods and urine dip are provided.

**QUESTION LINE**: What is the most appropriate management with regards to lipid modification?

**OPTIONS**: - a) Start atorvastatin 10mg on - b) Start atorvastatin 20mg on - c) Start atorvastatin 40mg on - d) Perform a QRISK2 assessment - e) Reassure her that lipid modification therapy is not required at this stage

**CORRECT-CHOICE LINE**: b

**REASONING**: NICE guidelines recommend atorvastatin 20mg for type 1 diabetics who meet certain criteria, which this patient does. QRISK2 is not recommended for type 1 diabetics. The patient has had diabetes for over 10 years.

**>>DESCRIPTION**: A 38-year-old woman with type 1 diabetes, controlled with a basal-bolus regime and citalopram, presents to clinic. She was diagnosed with type 1 diabetes at age 13. Recent bloods and urine dip are provided.

**>>OPTIONS**: a) Perform a QRISK2 assessment b) Reassure her that lipid modification therapy is not required at this stage c) Start atorvastatin 10mg on d) Start atorvastatin 20mg on e) Start atorvastatin 40mg on

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Start atorvastatin 20mg on

**>>REASONING**: The patient meets the criteria for statin therapy (diabetes duration > 10 years). NICE guidelines recommend atorvastatin 20mg. QRISK2 is not appropriate for type 1 diabetics.

## Question #:152

**CLINICAL SCENERIO**: A 70-year-old woman with hypertension (amlodipine 5mg od and ramipril 10mg od) is reviewed in the chronic kidney disease clinic. Recent blood pressure is 128/74 mmHg. Laboratory results are provided, including Na+, K+, Urea, Creatinine, and eGFR.

**QUESTION LINE**: What is the most appropriate next step in management?

**OPTIONS**: - a) Start atorvastatin 20mg on - b) Reduce ramipril to 5mg od and recheck U&Es in 4 weeks - c) Start simvastatin 40mg on - d) Increase amlodipine to 10mg od - e) Check her QRISK2 score

**CORRECT-CHOICE LINE**: a

**REASONING**: QRISK2 should not be used in patients with chronic kidney diseas (CKD). NICE now recommends that all patients with CKD should take a statin.

Offer atorvastatin 20 mg for the primary or secondary prevention of CVD to people with CKD

*  Increase thedose if a greater than 40% reduction in nonHDL cholesterol is not achieved and eGFR is 30 ml/min
*  Agree the use of higher doses with a renal specialist if eGFR is less than 30 ml/min

**>>DESCRIPTION**: A 70-year-old woman with hypertension and chronic kidney disease is reviewed. Her blood pressure and recent laboratory results (Na+, K+, Urea, Creatinine, eGFR) are provided.

**>>OPTIONS**: a) Start atorvastatin 20mg on b) Check her QRISK2 score c) Increase amlodipine to 10mg od d) Reduce ramipril to 5mg od and recheck U&Es in 4 weeks e) Start simvastatin 40mg on

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Start atorvastatin 20mg on

**>>REASONING**: The most appropriate next step is to start atorvastatin 20mg. NICE guidelines recommend statin therapy for all patients with CKD. QRISK2 is not recommended for patients with CKD. The other options are not the primary management approach based on the provided information.

## Question #:256

**CLINICAL SCENERIO**: A 28-year-old, 26-week pregnant woman with a BMI of 34 kg/m² undergoes an oral glucose tolerance test. Results: 0 hours - 7.4 mmol/l, 2 hours - 11.2 mmol/l. No other antenatal issues or scan abnormalities are noted. What is the most appropriate action?

**QUESTION LINE**: What is the most appropriate action?

**OPTIONS**: a. Repeat oral glucose tolerance test in 4 weeks b. Start metformin + advice about diet / exercise + self-monitor glucose levels c. Advice about diet / exercise + self-monitor glucose levels d. Start insulin + advice about diet / exercise + self-monitor glucose levels e. Reassure results within normal limits

**CORRECT-CHOICE LINE**: D

**REASONING**: NICE have recently changed their gestational diabetes guidelines. Insulin should be started in the fasting glucose is >= 7 mmol/l. Aspirin should also be considered given the increased risk of pre-eclampsia

**>>DESCRIPTION**: A 28-year-old, 26-week pregnant woman (BMI 34 kg/m²) has an oral glucose tolerance test: 0 hours - 7.4 mmol/l, 2 hours - 11.2 mmol/l. No other issues. What is the best next step?

**>>OPTIONS**: a. Advice about diet / exercise + self-monitor glucose levels b. Repeat oral glucose tolerance test in 4 weeks c. Reassure results within normal limits d. Start insulin + advice about diet / exercise + self-monitor glucose levels e. Start metformin + advice about diet / exercise + self-monitor glucose levels

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Start insulin + advice about diet / exercise + self-monitor glucose levels

**>>REASONING**: Based on current guidelines, insulin is indicated when fasting glucose is ≥ 7 mmol/l. Metformin is not the first-line treatment, and diet/exercise alone are insufficient. The results are not within normal limits. Aspirin should also be considered.

## Question #:284

**CLINICAL SCENERIO**: A 59-year-old man with uncontrolled hypertension and facial changes over 6 months, including coarse features, swollen fingers, and thickened skin, is diagnosed with acromegaly due to an elevated IGF-1 level and a pituitary adenoma. He underwent trans-sphenoidal surgery, but 3 months later, IGF-1 levels remain elevated, and symptoms persist.

**QUESTION LINE**: Which of the following is the best next step in the management of this patient?

**OPTIONS**: - a) Refer for stereotactic radiotherapy - b) Repeat transsphenoidal surger - c) Start octreotide - d) Repeat IGF-1 levels in 12 weeks - e) Repeat IGF-1 levels in six months

**CORRECT-CHOICE LINE**: C

**REASONING**: Acromegaly: if patients are not suitable for trans-sphenoidal surgery, or have residual symptoms, then octreotide may be used

This patient’s facial changes over time, coarse facial features on examination and elevated IGF-1 levels point towards a diagnosis of acromegaly. Since his MRI showed a pituitary adenoma, he was treated with transsphenoidal surgery. However, he remains symptomatic, and his IGF-1 levels have not reduced even after 3 months. Patients with residual symptoms need to repeat transsphenoidal surgery or start medical therapy with octreotide.

Since this patient has no residual tumour on his repeat MRI, he is not a candidate for repeat transsphenoidal surgery. Therefore, the most appropriate step for this patient is to start medical therapy with octreotide. Octreotide is a somatostatin analogue. It tends to decrease the size of the adenoma and its secretion of growth hormone.

Stereotactic radiotherapy is recommended for patients who fail medical therapy and is therefore not the best next step in the management of this patient.

Repeating IGF-1 levels again in 12 weeks is inappropriate as the IGF-1 level should have normalised at this follow-up 12 weeks after the transsphenoidal surgery. His repeat IGF-1 levels at 12 weeks are raised. He requires further medical or surgical management instead of follow-up in 12 weeks.

Patients with normal IGF-1 levels on a 12-week follow-up should have repeat IGF-1 levels at six months and then, annually.

**>>DESCRIPTION**: A 59-year-old man with acromegaly, treated with trans-sphenoidal surgery, has persistent elevated IGF-1 levels and symptoms 3 months post-surgery despite no residual tumor on MRI.

**>>OPTIONS**: a) Refer for stereotactic radiotherapy b) Repeat transsphenoidal surger c) Repeat IGF-1 levels in six months d) Repeat IGF-1 levels in 12 weeks e) Start octreotide

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Start octreotide

**>>REASONING**: The best next step is to start octreotide, a somatostatin analogue, since the patient has persistent symptoms and elevated IGF-1 after surgery, and repeat surgery is not an option due to the absence of residual tumor. Radiotherapy is reserved for those failing medical therapy. Repeating IGF-1 levels in the short term is not appropriate.

## Question #:171

**CLINICAL SCENERIO**: A 26-year-old woman with Graves’ disease, previously controlled on carbimazole, presents with tremor, palpitations, and weight loss, confirming a hyperthyroid state. She is on apixaban for atrial fibrillation, pregnancy test is negative, and has a mild goitre.

**QUESTION LINE**: What is the best course of action?

**OPTIONS**: 1. Add Lugol’s iodin 2. Add high dose oral prednisolone 3. Organise thyroidectomy 4. Start radioiodine treatment 5. Switch to propylthiouracil

**CORRECT-CHOICE LINE**: d

**REASONING**: Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

The correct answer is start radioiodine treatment . Radioiodine is the treatment of choice for relapse of Graves’ disease. The negative pregnancy test and lack of significant ophthalmopathy rule out the most important contraindications.

Add Lugol’s iodine is incorrect. Iodine-containing solutions can block the release of T3 and T4. However, the main place is in the treatment of thyroid storms, generally in combination with other agents such as beta blockers and thionamides.

Add high dose oral prednisolone is incorrect. Steroids are mainly used in severe cases such as those with thyroid storm, although the evidence is limited. This patient is symptomatic but with normal observations and no evidence of thyroid storm.

Organise thyroidectomy is incorrect. Thyroidectomy is a very effective treatment option and may be used to treat relapse in some patients but is generally unpopular. However, it is favoured in those with large goitre or contraindications to radioiodine. These include pregnancy or severe eye disease. In the absence of such contraindications, radioiodine would be favoured here. Her anticoagulation would also increase the complexity and risk of surgery here.

Switch to propylthiouracil is incorrect. This patient has relapsed despite appropriate first-line therapy with one thionamide - switching to another drug of the same class is unlikely to offer any benefits.

**>>DESCRIPTION**: A 26-year-old woman with Graves’ disease, previously controlled on carbimazole, presents with tremor, palpitations, and weight loss, confirming a hyperthyroid state. She is on apixaban, pregnancy test is negative, and has a mild goitre.

**>>OPTIONS**: 1. Add Lugol’s iodin 2. Add high dose oral prednisolone 3. Organise thyroidectomy 4. Start radioiodine treatment 5. Switch to propylthiouracil

**>>CORRECT-CHOICE LINE**: 4

**>>CORRECT-CHOICE\_TEXT**: Start radioiodine treatment

**>>REASONING**: Radioiodine therapy is the first-line treatment for Graves’ disease relapse in the absence of contraindications like pregnancy or severe ophthalmopathy. Incorrect options include Lugol’s iodine (for thyroid storm), high-dose prednisolone (severe cases), thyroidectomy (contraindications to radioiodine), and switching to propylthiouracil (no benefit).

## Question #:231

**CLINICAL SCENERIO**: A 42-year-old woman on lithium for bipolar disorder presents with weight gain, lethargy, a dry cough, and a hoarse voice. Examination reveals patchy hair loss, a smooth goitre, and a BMI of 32 kg/m². Her blood pressure is 122/82 mmHg, pulse is 60 bpm. Labs show Na+ 130 mmol/l and TSH 14.2 mIU/l.

**QUESTION LINE**: Which of the following is the most appropriate way to manage her?

**OPTIONS**: a) Iodine supplementation - b) Start prednisolone - c) Stop lithium - d) Surgical thyroidectomy - e) Start thyroxine

**CORRECT-CHOICE LINE**: e

**REASONING**: Lithium increases intrathyroidal iodine content, inhibits the coupling of iodotyrosine residues to form iodothyronines (thyroxine and triiodothyronine), and it also inhibits release of T4 and T3 leading to hypothyroidism, the clinical picture seen here. The most appropriate intervention is thyroxine supplementation.

There is no need acutely to stop lithium therapy, although substitution may eventually be considered after discussion with the patient’s psychiatrist. Due to the fact that lithium prevents coupling of iodotyrosine, iodine supplementation is ineffective. Prednisolone is also not of value because thyroiditis isn’t the cause of the hypothyroidism seen here. Surgical thyroidectomy isn’t indicated because there are no symptoms of extrinsic airway obstruction.

**>>DESCRIPTION**: A 42-year-old woman on lithium presents with weight gain, lethargy, dry cough, hoarse voice, patchy hair loss, a smooth goitre, and a BMI of 32 kg/m². Labs show Na+ 130 mmol/l and TSH 14.2 mIU/l.

**>>OPTIONS**: a) Iodine supplementation b) Start prednisolone c) Start thyroxine d) Stop lithium e) Surgical thyroidectomy

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Start thyroxine

**>>REASONING**: The patient has lithium-induced hypothyroidism. The most appropriate management is thyroxine supplementation. Iodine supplementation and prednisolone are not effective. Surgical thyroidectomy is not indicated.

## Question #:23

**CLINICAL SCENERIO**: A 45-year-old woman with cardiomyopathy (ICD, amiodarone for ventricular tachycardia), rheumatoid arthritis, and no coronary artery disease presents with symptoms suggestive of thyrotoxicosis (feeling very hot, weight loss, nausea, undetectable TSH). Her medications include methotrexate, aspirin, paracetamol, omeprazole, warfarin, and bisoprolol.

**QUESTION LINE**: She is suspected of having acute thyrotoxicosis. What is the most appropriate action in regards to her current medication in addition to stopping amiodarone?

**OPTIONS**: a) Stop aspirin b) Stop omeprazole c) Stop bisoprolol d) Stop warfarin e) Stop paracetamol

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: In acute thyrotoxicosis, stop aspirin as it can worsen the storm by displacing T4 from thyroid binding globulin

This patient with acute thyrotoxicosis should have amiodarone, the likely cause, of her hyperthyroidism stopped. In addition, aspirin should be stopped. Aspirin binds to thyroxine-binding globulin and displaces bound T4, thereby increasing the levels of free T4.

**>>DESCRIPTION**: 45-year-old female with cardiomyopathy (on amiodarone) and rheumatoid arthritis presents with symptoms of thyrotoxicosis (undetectable TSH). Medications include methotrexate, aspirin, paracetamol, omeprazole, warfarin, and bisoprolol.

**>>OPTIONS**: a) Stop aspirin b) Stop bisoprolol c) Stop omeprazole d) Stop paracetamol e) Stop warfarin

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Stop aspirin

**>>REASONING**: Aspirin should be stopped in acute thyrotoxicosis because it worsens the condition by displacing T4 from thyroid binding globulin, increasing free T4 levels.

## Question #:29

**CLINICAL SCENERIO**: A 45-year-old woman with cardiomyopathy, an ICD, and rheumatoid arthritis presents to the emergency department with feeling very hot, weight loss and nausea. She takes amiodarone for recurrent ventricular tachycardia. Blood tests show an undetectable thyroid stimulating hormone. She has previously had an angiogram showing no evidence of coronary artery disease. She takes methotrexate, aspirin, paracetamol, omeprazole, warfarin and bisoprolol. She is suspected of having acute thyrotoxicosis.

**QUESTION LINE**: What is the most appropriate action in regards to her current medication in addition to stopping amiodarone?

**OPTIONS**: a) Stop aspirin b) Stop omeprazole c) Stop bisoprolol d) Stop warfarin e) Stop paracetamol

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: In acute thyrotoxicosis, stop aspirin as it can worsen the storm by displacing T4 from thyroid binding globulin

This patient with acute thyrotoxicosis should have amiodarone, the likely cause, of her hyperthyroidism stopped. In addition, aspirin should be stopped. Aspirin binds to thyroxine-binding globulin and displaces bound T4, thereby increasing the levels of free T4.

**>>DESCRIPTION**: A 45-year-old woman on amiodarone for ventricular tachycardia, with a history of cardiomyopathy, ICD, and rheumatoid arthritis, presents with symptoms suggestive of thyrotoxicosis (undetectable TSH). She is also taking methotrexate, aspirin, paracetamol, omeprazole, warfarin, and bisoprolol.

**>>OPTIONS**: a) Bisoprolol b) Omeprazole c) Paracetamol d) Stop aspirin e) Warfarin

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Stop aspirin

**>>REASONING**: In acute thyrotoxicosis, aspirin should be stopped because it can worsen the condition by displacing T4 from thyroid-binding globulin, increasing free T4 levels. Other medications listed do not directly exacerbate thyrotoxicosis.

## Question #:36

**CLINICAL SCENERIO**: You are reviewing a 57 year-old gentleman in the diabetes outpatient clinic. He has type 2 diabetes mellitus and is currently taking metformin 850mg three times a day and gliclazide 80mg once daily.

On further questioning he admits having frequent hypoglycaemic episodes at night that distress him as he lives alone. His BMI is calculated at 30.3 kg/m², HbA1c 7.8% (62 mmol/mol) and his co-morbidities include congestive cardiac failure.

**QUESTION LINE**: How would you change his diabetic treatment?

**OPTIONS**: a) Stop gliclazide, start insulin b) Add exenatide c) Add sitagliptin to current regimen d) Stop gliclazide, start pioglitazone e) Stop gliclazide, start sitagliptin

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: The NICE guidance on the management of type 2 diabetes mellitus:

*  This gentleman has been started on metformin and a sulphonyurea as first line therapy.
*  He is having frequent hypoglycaemic episodes secondary to his sulphonylurea and yet control remains poor, HbA1c 7.8% (62 mmol/mol)
*  Pioglitazone is contraindicated due to his congestive cardiac failure.
*  A DPP-4 inhibitor such as sitagliptin would be a sensible option, the sulphonylurea should be stopped to prevent hypoglycaemia.

**>>DESCRIPTION**: A 57-year-old male with type 2 diabetes, on metformin and gliclazide, reports frequent nocturnal hypoglycaemic episodes. BMI is 30.3 kg/m², HbA1c is 7.8% (62 mmol/mol), and he has congestive cardiac failure.

**>>OPTIONS**: a) Add exenatide b) Add sitagliptin to current regimen c) Stop gliclazide, start insulin d) Stop gliclazide, start pioglitazone e) Stop gliclazide, start sitagliptin

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Stop gliclazide, start sitagliptin

**>>REASONING**: The patient experiences hypoglycemia due to gliclazide. Pioglitazone is contraindicated due to heart failure. Switching from gliclazide to sitagliptin (a DPP-4 inhibitor) is appropriate to reduce hypoglycemia risk while maintaining glycemic control.

## Question #:157

**CLINICAL SCENERIO**: A 55-year-old man with type 2 diabetes, currently on metformin, glicazide, and sitagliptin, struggles with weight control and has elevated HbA1c. His BMI is 34 kg/m².

**QUESTION LINE**: What would be the most appropriate next step?

**OPTIONS**: a) Canagliflozin b) Glibenclamide c) Increase metformin d) Stop sitagliptin and add insulin e) Stop sitagliptin and add exenatide

**CORRECT-CHOICE LINE**: e

**REASONING**: Given the NICE guidance the most appropriate step would be to start this patient on exenatide. This patient is already of metformin, glicazide and sitagliptin and the blood sugar levels are not under control. His BMI is under 35 but insulin would make ‘it much more difficult for you to do your job’.

**>>DESCRIPTION**: A 55-year-old man with uncontrolled type 2 diabetes on metformin, glicazide, and sitagliptin, with a BMI of 34 kg/m².

**>>OPTIONS**: a) Canagliflozin b) Glibenclamide c) Increase metformin d) Stop sitagliptin and add exenatide e) Stop sitagliptin and add insulin

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Stop sitagliptin and add exenatide

**>>REASONING**: Exenatide is the most appropriate next step according to NICE guidelines, considering the patient’s uncontrolled blood sugar despite existing medications and the suitability for his job.

## Question #:54

**CLINICAL SCENERIO**: A 34-year-old lady with no previous medical history presents to her GP. She has felt anxious and on edge for the past month. She also reports feeling sweaty and developing shakes in her hand that are impairing her work as a teacher. She has occasional abdominal bloating but manages this with herbal tea.

On examination, you notice she is flushed and tremulous but alert. She has no goitre and no rashes across her skin. There is a small mass palpable in the left side of the pelvis that is non tender and there are normal bowel sounds overlying. Her visual fields and cranial nerve exams are normal. A thyroid scintigraphy shows normal uptake. An MRI scan is awaited.

TSH

5.0 mU/L (range 0.4-4.0)

T4

28 pmol/l (range 9-24)

Urine

HCG

negative

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Jod-Basedow phenomenon - b) Exogenous iodine intake - c) Pituitary adenoma - d) Molar pregnanc - e) Struma ovarii

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Ovarian teratomas can produce exogenous TSH

This lady has clear secondary hyperthyroidism. There are no signs of pituitary disease so we have to consider other sources of TSH production. In pregnancy, HCG can act as an agonist at the TSH receptor but the test here is negative. The pelvic mass and TSH rise can be explained by ovarian teratomas that rarely can secrete TSH.

**>>DESCRIPTION**: A 34-year-old woman presents with anxiety, sweats, and hand tremors. Examination reveals flushing, tremors, and a non-tender pelvic mass. TSH is 5.0 mU/L (0.4-4.0), T4 is 28 pmol/l (9-24), and urine HCG is negative. Thyroid scintigraphy shows normal uptake.

**>>OPTIONS**: a) Exogenous iodine intake b) Jod-Basedow phenomenon c) Molar pregnancy d) Pituitary adenoma e) Struma ovarii

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Struma ovarii

**>>REASONING**: The patient presents with hyperthyroidism and a pelvic mass. Negative HCG rules out pregnancy-related causes. Ovarian teratomas can secrete TSH, causing hyperthyroidism and explaining the pelvic mass. Pituitary adenoma is less likely given the absence of specific pituitary signs.

## Question #:116

**CLINICAL SCENERIO**: A 42-year-old woman presents with a three-week history of neck pain and swelling that worsens upon swallowing. She also reports fatigue, tremors, nervousness, occasional palpitations, and unintentional weight loss. Examination reveals blood pressure of 125/80 mmHg, heart rate of 104 beats per minute, and temperature of 37.8 ºC. The thyroid gland is tender on palpation and enlarged without any palpable nodules. Laboratory results are as follows:

TSH

0.1 mIU/L ng/mL

(0.4 - 4.0)

| Free T4 | 8 ng/dL | (0.7 - 1.9) |
| --- | --- | --- |
| ESR | 101 mm/hr |  |

Anti-thyroglobulin and anti-thyroperoxidase antibodies are mildly elevated, while radioactive iodine uptake is low.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Acute infectious thyroiditis - b) Graves disease - c) Hashimoto thyroiditis - d) Solitary toxic nodule - e) Subacute granulomatous thyroiditis

**CORRECT-CHOICE LINE**: Thyrotoxicosis with tender goitre = subacute (De Quervain’s) thyroiditis

**REASONING**: Subacute granulomatous thyroiditis , also known as de Quervain’s thyroiditis, is the correct diagnosis. The patient presents with clinical and biochemical signs of thyrotoxicosis, as indicated by tachycardia, tremors, nervousness, low-grade fever, fatigue, weight loss, palpitations, suppressed TSH, and elevated free T4 levels. These symptoms, in conjunction with an enlarged tender thyroid gland and high ESR level, are indicative of de Quervain’s thyroiditis. Typically following a viral infection (not specified in this scenario), this condition is characterised by an initial phase of thyrotoxicosis followed by hypothyroidism. Management often involves symptomatic relief with non-steroidal anti-inflammatory drugs (NSAIDs) and betablockers.

Acute infectious thyroiditis , or suppurative thyroiditis, is a rare and potentially serious condition usually seen in the elderly and immunocompromised individuals. Gram-positive bacterial infections mainly cause it. Symptoms include sudden-onset fever, chills, malaise, a tender, painful thyroid gland, and signs of hyperthyroidism. In severe cases, the infection causes compression on adjacent structures, leading to dysphagia and hoarseness of voice. It is important to note that de Quervain’s thyroiditis has a gradual onset of symptoms and does not cause compression symptoms, making acute infectious thyroiditis less likely.

Graves’ disease is not the appropriate diagnosis here. Despite the biochemical and clinical features of thyrotoxicosis, the absence of Graves’ ophthalmopathy and dermopathy, specific anti-thyroid stimulating hormone receptor antibodies, and a low radioactive iodine uptake are inconsistent with this condition.

Hashimoto’s thyroiditis is a relevant differential diagnosis; however, it typically presents with a painless goitre. In de Quervain’s thyroiditis, mild elevations in antithyroglobulin and anti-thyroperoxidase antibodies may be observed in fewer than 25% of cases. By contrast, these antibody levels are significantly higher in Hashimoto’s thyroiditis.

Solitary toxic nodule is not consistent with this case presentation. Such nodules are generally single, palpable and non-tender associated with thyrotoxicosis. In contrast to this condition, the thyroid gland is diffusely enlarged and tender with no palpable; the findings align with de Quervain’s thyroiditis rather than a solitary toxic nodule.

**>>DESCRIPTION**: A 42-year-old woman has neck pain/swelling (worsens with swallowing), fatigue, tremors, nervousness, palpitations, and weight loss over 3 weeks. Exam: BP 125/80 mmHg, HR 104 bpm, T 37.8 ºC, tender/enlarged thyroid (no nodules). Labs: TSH 0.1 mIU/L, Free T4 8 ng/dL, ESR 101 mm/hr. Anti-thyroglobulin and anti-thyroperoxidase antibodies mildly elevated, low radioactive iodine uptake.

**>>OPTIONS**: a) Acute infectious thyroiditis b) Graves disease c) Hashimoto thyroiditis d) Solitary toxic nodule e) Subacute granulomatous thyroiditis

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Subacute granulomatous thyroiditis

**>>REASONING**: The patient’s thyrotoxicosis, tender goiter, and elevated ESR suggest subacute granulomatous thyroiditis (De Quervain’s). Graves’ disease is less likely due to the low radioactive iodine uptake and lack of ophthalmopathy. Hashimoto’s typically presents with a painless goiter. Solitary toxic nodules are typically single, palpable, and non-tender.

## Question #:337

**CLINICAL SCENERIO**: A 42-year-old woman presents with a three-week history of neck pain and swelling that worsens upon swallowing, fatigue, tremors, nervousness, occasional palpitations, and unintentional weight loss. Examination reveals a blood pressure of 125/80 mmHg, heart rate of 104 bpm, and temperature of 37.8 ºC. The thyroid gland is tender and enlarged without palpable nodules. Lab results show suppressed TSH, elevated free T4, and an ESR of 101 mm/hr, with mildly elevated anti-thyroid antibodies and low radioactive iodine uptake.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Acute infectious thyroiditis b) Graves disease c) Hashimoto thyroiditis d) Solitary toxic nodule e) Subacute granulomatous thyroiditis

**CORRECT-CHOICE LINE**: e

**REASONING**: Thyrotoxicosis with tender goitre = subacute (De Quervain’s) thyroiditis

Subacute granulomatous thyroiditis , also known as de Quervain’s thyroiditis, is the correct diagnosis. The patient presents with clinical and biochemical signs of thyrotoxicosis, as indicated by tachycardia, tremors, nervousness, low-grade fever, fatigue, weight loss, palpitations, suppressed TSH, and elevated free T4 levels. These symptoms, in conjunction with an enlarged tender thyroid gland and high ESR level, are indicative of de Quervain’s thyroiditis. Typically following a viral infection (not specified in this scenario), this condition is characterised by an initial phase of thyrotoxicosis followed by hypothyroidism. Management often involves symptomatic relief with non-steroidal anti-inflammatory drugs (NSAIDs) and betablockers.

Acute infectious thyroiditis , or suppurative thyroiditis, is a rare and potentially serious condition usually seen in the elderly and immunocompromised individuals. Gram-positive bacterial infections mainly cause it. Symptoms include sudden-onset fever, chills, malaise, a tender, painful thyroid gland, and signs of hyperthyroidism. In severe cases, the infection causes compression on adjacent structures, leading to dysphagia and hoarseness of voice. It is important to note that de Quervain’s thyroiditis has a gradual onset of symptoms and does not cause compression symptoms, making acute infectious thyroiditis less likely.

Graves’ disease is not the appropriate diagnosis here. Despite the biochemical and clinical features of thyrotoxicosis, the absence of Graves’ ophthalmopathy and dermopathy, specific anti-thyroid stimulating hormone receptor antibodies, and a low radioactive iodine uptake are inconsistent with this condition.

Hashimoto’s thyroiditis is a relevant differential diagnosis; however, it typically presents with a painless goitre. In de Quervain’s thyroiditis, mild elevations in antithyroglobulin and anti-thyroperoxidase antibodies may be observed in fewer than 25% of cases. By contrast, these antibody levels are significantly higher in Hashimoto’s thyroiditis.

Solitary toxic nodule is not consistent with this case presentation. Such nodules are generally single, palpable and non-tender associated with thyrotoxicosis. In contrast to this condition, the thyroid gland is diffusely enlarged and tender with no palpable; the findings align with de Quervain’s thyroiditis rather than a solitary toxic nodule.

**>>DESCRIPTION**: A 42-year-old woman with a three-week history of neck pain, swelling, fatigue, tremors, nervousness, palpitations, and weight loss. Examination reveals tachycardia, elevated free T4, suppressed TSH, elevated ESR, tender and enlarged thyroid, mildly elevated anti-thyroid antibodies and low radioactive iodine uptake.

**>>OPTIONS**: a) Acute infectious thyroiditis b) Graves disease c) Hashimoto thyroiditis d) Solitary toxic nodule e) Subacute granulomatous thyroiditis

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Subacute granulomatous thyroiditis

**>>REASONING**: The most likely diagnosis is subacute granulomatous thyroiditis (De Quervain’s) due to thyrotoxicosis symptoms, tender goitre, and elevated ESR. Graves’ disease and solitary toxic nodule are less likely because of the low radioactive iodine uptake and the diffuse nature of the goitre. Hashimoto’s typically presents with a painless goitre, and acute infectious thyroiditis has a more acute onset with different features.

## Question #:2

**CLINICAL SCENERIO**: A 71-year-old gentleman with newly diagnosed type 2 diabetes, unresponsive to dietary changes, presents with a history of bladder cancer (chemotherapy), hypertension, macular degeneration, eczema, and chronic kidney disease (eGFR 28ml/min/1.73m2).

**QUESTION LINE**: What is the most appropriate medication to start?

**OPTIONS**: - a) Metformin - b) Sulfonylurea - c) Pioglitazone - d) Insulin - e) GLP-1 mimetic

**CORRECT-CHOICE LINE**: b

**REASONING**: The correct answer is a sulfonylurea. This is a patient with new type two diabetes and initial treatment would normally be metformin, but since the eGFR is <30ml/minute/1.73m2 metformin is contraindicated. The next step would be either a a dipeptidyl peptidase4 inhibitor, pioglitazone or a sulfonylurea. However, pioglitazone should not be offered to a patient with a history of bladder cancer. Therefore that leaves sulfonylurea as the correct answer. Insulin and GLP-1 mimetic could be used further down in the treatment algorithm.

**>>DESCRIPTION**: A 71-year-old gentleman with newly diagnosed type 2 diabetes, unresponsive to diet and with CKD (eGFR 28) and a history of bladder cancer.

**>>OPTIONS**: a) GLP-1 mimetic b) Insulin c) Metformin d) Pioglitazone e) Sulfonylurea

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Sulfonylurea

**>>REASONING**: Sulfonylurea is the most appropriate initial medication because metformin is contraindicated due to the eGFR <30, and pioglitazone is contraindicated due to a history of bladder cancer. Insulin and GLP-1 mimetics are later-line options.

## Question #:292

**CLINICAL SCENERIO**: A 19-year-old woman presents with recurrent weakness relieved by sweet drinks. Her sister has type 1 diabetes. Capillary glucose was found to be as low as 2mmol/l. The parents are concerned about insulinoma.

**QUESTION LINE**: Which investigation is best suited to exclude insulinoma?

**OPTIONS**: a. CT abdomen b. Capillary blood glucose monitoring over 48 hours c. Post-prandial serum insulin and C-peptide d. Supervised fasting with serum insulin and C-peptide e. Synacthen test

**CORRECT-CHOICE LINE**: D

**REASONING**: Elevated C-peptide in fasting hypoglycaemia is suggestive of insulinoma

This patient has episodes of hypoglycaemia. There are several causes for hypoglycaemia, including insulinoma, liver failure, Addison’s disease, alcohol and use of oral hypoglycaemic agents and insulin. Whilst insulinomas can cause episodes of hypoglycaemia, commonly associated with rapid weight gain and MEN-1, other explanations should also be considered. As this patient has a relative with type 1 diabetes she may have an opportunity to acquire insulin and use this herself. The most appropriate investigation to differentiate between the two is supervised fasting with monitoring of blood glucose, insulin and C-peptide can demonstrate or exclude insulinoma. If the patient were to have an insulinoma, then during fasting the blood glucose would become low, with elevated serum insulin and C-peptide, as C-peptide corresponds to endogenous rather than exogenous insulin. If the patient had hypoglycaemia during a fast with elevated insulin but low C-peptide the diagnosis would more likely be exogenous insulin use. A CT scan of the abdomen can be used if insulinoma is suspected following supervised fasting. Synacthen testing should be used to suspected Addison’s disease.

**>>DESCRIPTION**: A 19-year-old woman presents with recurrent weakness relieved by sweet drinks, and low capillary glucose readings. Her sister has type 1 diabetes. The parents are concerned about insulinoma.

**>>OPTIONS**: a. Capillary blood glucose monitoring over 48 hours b. CT abdomen c. Post-prandial serum insulin and C-peptide d. Supervised fasting with serum insulin and C-peptide e. Synacthen test

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Supervised fasting with serum insulin and C-peptide

**>>REASONING**: Supervised fasting with serum insulin and C-peptide is the best investigation to differentiate between insulinoma and exogenous insulin use by measuring blood glucose, insulin, and C-peptide levels. Elevated C-peptide during hypoglycemia suggests insulinoma. Other options are less helpful or used for different diagnoses.

## Question #:259

**CLINICAL SCENERIO**: A 22-year-old woman presents with intermittent abdominal pain, low mood, and amenorrhea. She has a history of renal calculi and a recent car accident. She experiences headaches, white nipple discharge, low blood pressure, faints, and glycosuria. Examination reveals a blistering rash and reduced visual fields. She is on hydrocortisone. What is the most effective treatment?

**QUESTION LINE**: Which of the following is likely to treat the underlying condition most effectively?

**OPTIONS**: a) Bisphosphonates b) Surgery c) Cabergoline d) Octreotide e) Insulin

**CORRECT-CHOICE LINE**: b

**REASONING**: The patient’s symptoms suggest multiple endocrine neoplasia (MEN) type 1, involving tumors of the parathyroid, pituitary, and pancreas. Surgery is the most comprehensive approach to address these diverse issues, unlike options targeting a single aspect.

**>>DESCRIPTION**: A 22-year-old woman presents with abdominal pain, mood changes, amenorrhea, history of renal calculi, headaches, nipple discharge, low blood pressure, glycosuria, and a blistering rash. She is on hydrocortisone. What’s the best treatment?

**>>OPTIONS**: a) Bisphosphonates b) Cabergoline c) Insulin d) Octreotide e) Surgery

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Surgery

**>>REASONING**: Surgery is the best treatment for Multiple Endocrine Neoplasia (MEN) type 1, because it addresses the tumors affecting multiple endocrine glands. Other choices treat individual symptoms, not the underlying cause.

## Question #:40

**CLINICAL SCENERIO**: A 50-year-old man, a heavy goods vehicle driver, presents to the diabetes clinic. He is on metformin 1g BD and gliclazide 160mg BD. His HbA1c has increased to 72 mmol/mol. He has gained 5kg in 12 weeks. His blood pressure is 155/88 mmHg, pulse is 75 bpm, and BMI is 37 kg/m². Investigations show Na+ 140 mmol/l, K+ 5.0 mmol/l, Urea 7.1 mmol/l, and Creatinine 110 µmol/l.

**QUESTION LINE**: Which of the following is the most appropriate next step?

**OPTIONS**: - a) Add basal insulin - b) Add mixed insulin - c) Add pioglitazone - d) Switch gliclazide to linagliptin - e) Switch gliclazide to liraglutide

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: A number of factors come into play with respect to management of this patient’s blood glucose. His occupation as a lorry driver precludes medication which may significantly increase his risk of hypoglycaemia. This effectively rules out insulin initiation unless he is prepared to accept any impact on his job. Insulin would also promote further weight gain and salt and water retention, and is therefore less desirable as an option. Pioglitazone promotes weight gain and fluid retention, so again is not really an option.

This leaves us with the two switching scenarios. Switching to liraglutide is preferred, because in trials GLP-1 receptor agonists have been shown to reduce HbA1c by similar levels to basal insulin, without increasing the risk of hypoglycaemia, and promote approximately 3% weight loss over a 6 month period. DPPIV inhibitors such as linagliptin are potentially less effective in reducing HbA1c than sulphonylureas and linagliptin is therefore not an option here.

**>>DESCRIPTION**: 50-year-old male, a heavy goods vehicle driver with diabetes, on metformin and gliclazide, presents with increased HbA1c (72 mmol/mol), weight gain (5kg), BP 155/88 mmHg, and BMI 37 kg/m².

**>>OPTIONS**: a) Add basal insulin b) Add mixed insulin c) Add pioglitazone d) Switch gliclazide to linagliptin e) Switch gliclazide to liraglutide

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Switch gliclazide to liraglutide

**>>REASONING**: Lorry driver occupation increases hypoglycemia risk, ruling out insulin. Insulin and pioglitazone promote weight gain. Liraglutide reduces HbA1c without hypoglycemia risk and promotes weight loss. Linagliptin is less effective than sulfonylureas.

## Question #:99

**CLINICAL SCENERIO**: A 56-year-old man with type 2 diabetes managed with Humalog mix 30 and metformin 1g BD presents with HbA1c of 57. He reports troublesome hypoglycaemia episodes in the late afternoon and early mornings. His blood pressure is 132/82 mmHg, pulse is 72 bpm, and BMI is 32 kg/m².

**QUESTION LINE**: Which of the following is the most appropriate next step in his management?

**OPTIONS**: a) Continue current regimen, eat a snack mid afternoon and before bed time b) Reduce the dose of metformin c) Reduce the dose of mixed insulin d) Switch to a basal bolus regimen e) Switch to Humalog mix 20

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: This person’s HbA1c is above target at 57 mmol/mol or 7.4%. In spite of this, he is still suffering from hypoglycaemia episodes as a result of delivering his insulin as a twice a day regimen. Moving him to basal bolus will allow splitting up of the short acting component of his insulin requirements, and therefore reduce the risk of hypoglycaemia at the end of each dosing period.

Reducing the dose of his mixed insulin may drive a further rise in his HbA1c, as would reducing the dose of metformin. Switching to Humalog mix 20 will increase the proportion of long-acting insulin and worsen the risk of hypoglycaemia. Eating a snack will drive weight gain and worsen his HbA1c.

**>>DESCRIPTION**: A 56-year-old man with type 2 diabetes (Humalog mix 30 and metformin) has HbA1c of 57 and experiences hypoglycaemia. BP is 132/82 mmHg, pulse 72 bpm, BMI 32 kg/m².

**>>OPTIONS**: a) Continue current regimen, eat a snack mid afternoon and before bed time b) Reduce the dose of metformin c) Reduce the dose of mixed insulin d) Switch to a basal bolus regimen e) Switch to Humalog mix 20

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Switch to a basal bolus regimen

**>>REASONING**: Basal-bolus regimen is most appropriate to reduce hypoglycaemia risk by splitting the short-acting insulin component. Reducing insulin or metformin doses may worsen HbA1c. Switching to Humalog mix 20 or eating snacks can exacerbate hypoglycaemia or weight gain.

## Question #:172

**CLINICAL SCENERIO**: An elderly male presents with a 2 week history of breathlessness. His past medical history includes diet-controlled type 2 diabetes, ischaemic heart disease, hypothyroidism and depression. His medication list includes levothyroxine, aspirin, simvastatin, ramipril, bisoprolol and citalopram. Observations on presentation to Emergency Department are as follows: respiratory rate 26/min, saturations 94% (on 4 litres oxygen via Venturi), heart rate 80 beats per minute, blood pressure 156/82 mmHg. Auscultation demonstrates crackles at the left base with no wheeze. The abdomen is soft and non-tender. There is no oedema peripherally. Blood results on admission are provided below: Hb 134 g/l, Platelets 172 \* 10 9 /l, WBC 13.3 \* 10 9 /l, Na + 128 mmol/l, K + 5.1 mmol/l, Urea 13 mmol/l, Creatinine 178 µmol/l, Serum osmolality 220 mosm/kg, Urinary sodium 50 mEq//l

**QUESTION LINE**: What is the most likely cause of hyponatraemia?

**OPTIONS**: 1. Hypothyroidism 2. Chronic kidney disease 3. Addison’s disease 4. Salt-losing nephropathy 5. Syndrome of inappropriate antidiuretic hormone (SIADH)

**CORRECT-CHOICE LINE**: e

**REASONING**: This question demonstrates a common scenario in clinical practice. Management of hyponatraemia first requires clarification of fluid status (clinical hypovolaemia, euvolaemia or hypervolaemia), as differentials are influenced by this. This patient’s history, examination findings and haemodynamic parameters are consistent with clinical euvolaemia. Differentials for euvolaemic hyponatraemia would include hypothyroidism and SiADH. There are no clinical features suggestive of the former. Findings are consistent with community-acquired pneumonia with associated SiADH. This is confirmed by the presence of reduced serum osmolality and high urinary sodium. Measurement of urinary sodium concentration is an useful adjunct in helping to differentiate between hyponatraemia secondary to hypovolaemia and SiADH. With SiADH (and salt-wasting syndrome), the urinary sodium is high. With hypovolaemia, the urinary sodium is typically low.

**>>DESCRIPTION**: An elderly male presents with breathlessness, relevant medical history, and medication list. Observations include respiratory rate, oxygen saturation, heart rate, blood pressure, auscultation findings, and blood/urine results indicating hyponatremia.

**>>OPTIONS**: 1. Addison’s disease 2. Chronic kidney disease 3. Hypothyroidism 4. Salt-losing nephropathy 5. Syndrome of inappropriate antidiuretic hormone (SIADH)

**>>CORRECT-CHOICE LINE**: 5

**>>CORRECT-CHOICE\_TEXT**: Syndrome of inappropriate antidiuretic hormone (SIADH)

**>>REASONING**: The most likely cause of hyponatremia is SIADH, given the clinical presentation, euvolemic state, reduced serum osmolality, and high urinary sodium. Hypothyroidism is a differential but less likely given the presentation. The patient’s clinical features and labs do not support other causes such as CKD, Addison’s disease or salt-losing nephropathy.

## Question #:285

**CLINICAL SCENERIO**: A 64-year-old man with a history of bipolar disorder on lithium presents with confusion. He drinks approximately 6 pints of beer daily. Blood results show hyponatremia, low plasma osmolarity, and elevated urine osmolarity.

**QUESTION LINE**: What is the most likely explanation?

**OPTIONS**: a. Beer potomania b. Cerebral salt wasting c. Diabetes insipidus d. Primary polydipsia e. Syndrome of inappropriate antidiuretic hormone ADH release (SIADH)

**CORRECT-CHOICE LINE**: e

**REASONING**: SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient. The patient fulfils the diagnostic criteria of SIADH making this the most likely diagnosis. Beer potomania, cerebral salt wasting, diabetes insipidus, and primary polydipsia are incorrect because they do not fit the patient’s presentation of hyponatremia with elevated urine osmolarity and euvolemia.

**>>DESCRIPTION**: A 64-year-old man on lithium with a history of bipolar disorder presents with confusion. He consumes a large amount of beer daily. Labs show hyponatremia, low plasma osmolarity, and elevated urine osmolarity.

**>>OPTIONS**: a. Beer potomania b. Cerebral salt wasting c. Diabetes insipidus d. Primary polydipsia e. Syndrome of inappropriate antidiuretic hormone ADH release (SIADH)

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Syndrome of inappropriate antidiuretic hormone ADH release (SIADH)

**>>REASONING**: The most likely explanation is SIADH because the patient presents with hyponatremia, low serum osmolarity, and elevated urine osmolarity. Beer potomania, cerebral salt wasting, diabetes insipidus, and primary polydipsia are less likely due to differing electrolyte and fluid balance profiles.

## Question #:320

**CLINICAL SCENERIO**: A 35-year-old female presents with weight loss, palpitations, and fatigue. TSH and T4 levels are provided.

**QUESTION LINE**: What is the next most appropriate investigation to perform in this patient?

**OPTIONS**: a) Urinary pregnancy test b) Plasma metanephrine c) T3 levels d) Thyroid antibodies e) 24 hour ECG

**CORRECT-CHOICE LINE**: c

**REASONING**: T3 levels should be performed where tests show normal T4 with suppressed TSH T3 thyrotoxicosis should always be considered in patients with suppressed TSH and normal T4 levels, especially when patients are symptomatic.

Pregnancy can cause raised total thyroxine but normal free T4 and T3 and would not suppress TSH. Thyroid antibodies would be useful to consider once the diagnosis has been confirmed. Plasma metanephrines should be performed to investigate if an underlying pheochromocytoma is suspected and ECG may be indicated to investigate palpitations but would not explain the whole presentation.

**>>DESCRIPTION**: A 35-year-old female presents with weight loss, palpitations, and fatigue and has abnormal thyroid function tests.

**>>OPTIONS**: a) 24 hour ECG b) Plasma metanephrine c) T3 levels d) Thyroid antibodies e) Urinary pregnancy test

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: T3 levels

**>>REASONING**: T3 levels are the next most appropriate investigation because they help diagnose T3 thyrotoxicosis, which is suggested by the patient’s suppressed TSH and normal T4. Other options are less likely to explain the presentation.

## Question #:119

**CLINICAL SCENERIO**: A 55 year-old female presents with fatigue, increased sweating, and weight loss over four months, along with a loss of sex drive. Examination reveals pallor, a pulse rate of 121 per minute with a bounding pulse, and a smooth goitre without thyroid eye disease. Blood tests show elevated TSH, Free T4, and Free T3, as well as hyponatremia.

**QUESTION LINE**: Which of the following is the most likely diagnosis?

**OPTIONS**: - a) Grave’s disease - b) Thyroid cancer - c) Surreptitious thyroxine ingestion - d) De Quervain’s thyroiditis - e) TSH secreting pituitary tumour

**CORRECT-CHOICE LINE**: Correct answer is e.

**REASONING**: Biochemistry reveals elevated thyroid-stimulating hormone (TSH) with concurrent elevated thyroxine (T4) and tri-iodothyronine (T3). An elevated alkaline phosphatase (ALP) is consistent with thyrotoxicosis. Hyponatraemia suggests hypoadrenalism.

Taken with the symptoms, this patient has a likely diagnosis of a thyrotropinoma, which is a rare type of pituitary tumour accounting for approximately less than 1% of cases of pituitary tumours. 90% are macroadenomas.

Presentation is typically with features of thyrotoxicosis and include weight loss, sweating, fatigue and tachycardia. There may also be signs of hypopituitarism.

**>>DESCRIPTION**: A 55-year-old female presents with fatigue, sweating, weight loss, and decreased libido. Examination shows pallor, tachycardia with bounding pulse, and smooth goitre. Labs reveal elevated TSH, Free T4, Free T3, and hyponatremia.

**>>OPTIONS**: a) De Quervain’s thyroiditis b) Grave’s disease c) Surreptitious thyroxine ingestion d) Thyroid cancer e) TSH secreting pituitary tumour

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: TSH secreting pituitary tumour

**>>REASONING**: Elevated TSH, T4, and T3 indicate a central cause of hyperthyroidism. The most likely diagnosis is TSH-secreting pituitary tumor (thyrotropinoma). Grave’s disease, thyroid cancer, surreptitious thyroxine ingestion and De Quervain’s thyroiditis would not present with elevated TSH.

## Question #:159

**CLINICAL SCENERIO**: A 28-year-old pregnant woman with intractable vomiting, 3+ ketones in urine, and failure of cyclizine.

**QUESTION LINE**: Apart from rehydration and anti-emetics, what also should be prescribed?

**OPTIONS**: - a) Thiamine - b) Carbohydrate replacement with 5% dextrose - c) Niacin - d) Vitamin B1 - e) Folate

**CORRECT-CHOICE LINE**: a

**REASONING**: Nausea and vomiting are common in pregnancy. Hyperemesis gravidarum requires thiamine supplementation, especially before dextrose or parenteral nutrition. A 5% dextrose infusion may precipitate Wernicke’s encephalopathy. Folic acid is for neural tube defects, not hyperemesis. Niacin deficiency is not a primary concern.

**>>DESCRIPTION**: A 28-year-old pregnant woman presents with intractable vomiting and 3+ ketones in her urine, failing cyclizine. What should be prescribed in addition to rehydration and anti-emetics?

**>>OPTIONS**: a) Thiamine b) Carbohydrate replacement with 5% dextrose c) Folate d) Niacin e) Vitamin B1

**>>CORRECT-CHOICE LINE**: a

**>>CORRECT-CHOICE\_TEXT**: Thiamine

**>>REASONING**: Thiamine supplementation is crucial in hyperemesis gravidarum, especially before dextrose administration, to prevent Wernicke’s encephalopathy. Other options are either incorrect or less relevant in this context.

## Question #:75

**CLINICAL SCENERIO**: A 45-year-old woman comes to the clinic some 6 months after thyroid resection for differentiated thyroid cancer. She is well, has recovered from her surgery and has a neatly healed scar across her anterior neck. Blood pressure is normal at 110/80 mmHg, and her pulse is 60 and regular. Her body mass index is unchanged at 25 kg/m². Only medication is thyroid hormone replacement.

**QUESTION LINE**: Which of the following is the most appropriate way to monitor for a recurrence?

**OPTIONS**: a) MRI neck b) Technetium scanning c) Thyroglobulin d) Thyroid ultrasound scan e) T3 levels

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Given this patient is prescribed thyroid hormone replacement, monitoring of T3 or T4 is not useful in monitoring for cancer recurrence. On the other hand the presence of thyroglobulin does indicate thyroid gland activity which isn’t suppressed by thyroid hormone replacement and is therefore potentially cancerous in origin. Levels should be undetectable.

Radiological investigations such as MRI neck, technetium scanning or thyroid ultrasound scanning are potentially less sensitive than monitoring for thyroglobulin and are therefore not a preferred first line investigation. T3 levels are unsuitable as a marker of recurrence given that conversion of exogenously administered T4 to T3 confounds any measurement.

**>>DESCRIPTION**: A 45-year-old woman, 6 months post thyroid resection for differentiated thyroid cancer, is on thyroid hormone replacement. How should recurrence be monitored?

**>>OPTIONS**: a) MRI neck b) T3 levels c) Technetium scanning d) Thyroglobulin e) Thyroid ultrasound scan

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Thyroglobulin

**>>REASONING**: Thyroglobulin levels are the most appropriate way to monitor for recurrence because their presence indicates thyroid gland activity not suppressed by hormone replacement, suggesting potential cancerous origin. Radiological investigations are less sensitive, and T3 levels are confounded by exogenous T4 conversion.

## Question #:118

**CLINICAL SCENERIO**: A 24-year-old female presents with one week of progressive double vision, increasing tiredness over 2 months, and occasional chest tightness with palpitations. She has no past medical history or family history. Examination reveals loss of left eye abduction, right eye upwards gaze, and right eye adduction. Systemic examination shows bilateral clammy hands and a heart rate of 120 per minute, irregular.

**QUESTION LINE**: Which test is most likely to be diagnostic?

**OPTIONS**: - a) Autoimmune screen - b) Thyroid function tests - c) CT thorax - d) Anti-acetylcholine receptor antibodie - e) 12 lead ECG

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This patient presents with systemic symptoms and a complex ophthalmoplegia, the diagnosis of thyroid eye disease, secondary to Graves disease, is most likely. The important test would be thyroid function tests and also MRI of her orbits, which would almost certainly demonstrate retro-orbital and extraocular muscle inflammation. The severity of the patient’s eye disease needs to be assessed: the most frequently used criteria was developed by the American thyroid association, which spells out helpfully NO SPECS

Class 0 No symptoms or signs

Class I Only signs, no symptoms (lid retraction, stare, lid lag)

Class II Soft tissue involvement

Class III Proptosis

Class IV Extraocular muscle involvement

Class V Corneal involvement

Class VI Sight loss (optic nerve involvement)

Any patient presenting with eye movement weaknesses that cannot be explained by isolated or multiple cranial nerve palsies is called complex ophthalmoplegia. The differentials include myasthenia gravis, mononeuritis multiplex, thyroid eye disease, Kearns-Sayre syndrome, complex progressive external ophthalmoplegia, Miller-Fisher syndrome and botulinum poisoning.

**>>DESCRIPTION**: A 24-year-old female presents with progressive double vision, tiredness, chest tightness, and palpitations. Examination reveals ophthalmoplegia, clammy hands, and irregular heart rate of 120 bpm.

**>>OPTIONS**: a) 12 lead ECG b) Anti-acetylcholine receptor antibodie c) Autoimmune screen d) CT thorax e) Thyroid function tests

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Thyroid function tests

**>>REASONING**: Thyroid function tests are most likely diagnostic due to the systemic symptoms and complex ophthalmoplegia suggestive of thyroid eye disease secondary to Graves disease.

## Question #:173

**CLINICAL SCENERIO**: A 52-year-old female presents with galactorrhoea and fatigue. Blood results show elevated prolactin.

**QUESTION LINE**: What investigation will you perform?

**OPTIONS**: 1. MRI pituitary 2. Synacthen test 3. Thyroid function tests 4. Toxicology screen 5. No further investigations required

**CORRECT-CHOICE LINE**: c

**REASONING**: Primary hypothyroidism can cause hyperprolactinaemia. High leveles of thyrotrophin releasing hormone (TRH) stimulate prolactin release. The patient has galactorrhoea secondary to hyperprolactinaemia. Hypothyroidism is a common cause and can cause hyponatraemia. Thyroid function tests are therefore the best answer. The Synacthen test is used to assess for hypoadrenalism. A toxicology screen would not detect the causes of hyperprolactinaemia.

**>>DESCRIPTION**: A 52-year-old female with galactorrhoea and elevated prolactin levels presents with fatigue.

**>>OPTIONS**: 1. MRI pituitary 2. No further investigations required 3. Synacthen test 4. Thyroid function tests 5. Toxicology screen

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Thyroid function tests

**>>REASONING**: The most appropriate investigation is thyroid function tests because primary hypothyroidism can cause hyperprolactinemia. High TRH levels stimulate prolactin release. Other options are less likely; Synacthen assesses adrenal function, and a toxicology screen wouldn’t identify the cause.

## Question #:66

**CLINICAL SCENERIO**: A 45-year-old woman develops agitation, high fever, sweating and atrial fibrillation with a heart rate of 160bpm following a CT pulmonary angiogram. She was recently seen in the emergency department and given a provisional diagnosis of pulmonary embolism and discharged with an urgent CT pulmonary angiogram and treatment dose low molecular weight heparin two days ago. She has a past medical history of breast cancer which has recently recurred with metastases to the liver, depression and COPD. She normally takes sertraline, letrozole, carbimazole and as needed salbutamol. Her original presentation to the emergency department was with shortness of breath which has now resolved. The CT scan has demonstrated no evidence of pulmonary embolism and clear lung fields. On examination, she is very agitated, has tremors, is very hot to touch and has ankle oedema. She has bilateral crepitations on chest auscultation. Her temperature is 40.1ºC. Blood tests are pending.

**QUESTION LINE**: What is the most likely cause of her presentation?

**OPTIONS**: - a) Delayed autoimmune reaction secondary to contrast medium - b) Atypical pneumonia - c) Missed pulmonary embolism with right heart strain - d) Thyroid storm - e) Heparin induced thrombocytopenia

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: Iodine in CT contrast media can precipitate thyrotoxicosis or thyroid storm

The correct answer is thyroid storm. The iodine in the CT contrast medium can trigger thyrotoxicosis and thyroid storm. The features of hyperpyrexia, agitation, confusion, AF with a fast ventricular rate in a patient with known hyperthyroidism following a triggering event, such as CT contrast, infection or surgery can thyrotoxicosis or thyroid storm. An autoimmune reaction is unlikely given the absence of rash and presence of fever and agitation. Pneumonia is unlikely without any radiological features on the CT scan and also with the absence of a cough, and missing pulmonary embolism is unlikely, but it is also unlikely to miss right heart strain on CT

**>>DESCRIPTION**: A 45-year-old woman presents with agitation, high fever, sweating, and atrial fibrillation (HR 160 bpm) after a CT pulmonary angiogram. History includes breast cancer with liver metastases, depression, and COPD. Medications: sertraline, letrozole, carbimazole, and salbutamol. CT shows no pulmonary embolism. Exam: agitated, tremors, hot, ankle edema, bilateral crepitations. Temperature is 40.1ºC.

**>>OPTIONS**: a) Atypical pneumonia b) Delayed autoimmune reaction secondary to contrast medium c) Heparin induced thrombocytopenia d) Missed pulmonary embolism with right heart strain e) Thyroid storm

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Thyroid storm

**>>REASONING**: The most likely cause is thyroid storm, triggered by iodine in the CT contrast medium. Features include hyperpyrexia, agitation, confusion, and AF in a patient with known hyperthyroidism. Autoimmune reaction is less likely without a rash. Pneumonia is unlikely without radiological findings or cough. Pulmonary embolism was ruled out by CT.

## Question #:15

**CLINICAL SCENERIO**: A 35-year-old woman presents with weight loss, feeling warmer, insomnia, and hand tremor. She has a history of depression treated with sertraline. Examination reveals a BMI of 24 kg/m², tremor, and a large, nontender goitre without palpable nodules or lymphoadenopathy. Blood tests show undetectable TSH and elevated free T4. She is diagnosed with hyperthyroidism.

**QUESTION LINE**: What further investigation is most appropriate to determine the type of her hyperthyroidism?

**OPTIONS**: - a) Thyroid-stimulating hormone receptor antibodies - b) Liver function tests - c) CT neck - d) Scan of radionuclide thyroid uptake - e) US-guided fine needle aspiration cytology

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: TSH antibodies are found in 90% of patients with Graves’ disease and can help distinguish from other forms of hyperthyroidism

This patient clinically and biochemically has hyperthyroidism. To help determine the cause of hyperthyroidism, and distinguish between causes such as Graves’ disease, toxic adenoma and toxic multinodular goitre. Thyroid-stimulating hormone receptor antibodies (TSH-RAb, commonly shortened to TRAb) has an up to 98% sensitivity and 99% specificity for Graves’ disease. If this test is negative, then a radionuclide thyroid uptake scan can help determine the cause. In Graves’ disease there is diffuse high uptake, in thyroiditis there is low uptake, and if there are nodules then there is uneven uptake. US-guided fine needle aspiration cytology is recommended in the presence of features suggestive of malignancy. Liver function tests are important if there is a history of liver disease as carbimazole can cause further liver injury, but it would not help in determining the cause of hyperthyroidism

**>>DESCRIPTION**: A 35-year-old woman with weight loss, heat intolerance, insomnia, and tremor has undetectable TSH and high free T4. She has a large goiter. What test will determine the cause of hyperthyroidism?

**>>OPTIONS**: a) CT neck b) Liver function tests c) Scan of radionuclide thyroid uptake d) Thyroid-stimulating hormone receptor antibodies e) US-guided fine needle aspiration cytology

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Thyroid-stimulating hormone receptor antibodies

**>>REASONING**: TSH receptor antibodies are the most appropriate initial test, as they are highly sensitive and specific for Graves’ disease, a common cause of hyperthyroidism. Radionuclide thyroid uptake scan is useful if TSH receptor antibodies are negative. Liver function tests are not helpful to determine the cause of hyperthyroidism.

## Question #:45

**CLINICAL SCENERIO**: A 43-year-old female presents with neck discomfort worsening over the past 2 months. Examination reveals a firm neck lump moving with swallowing but not with tongue protrusion. Ultrasound reveals a 2.5cm papillary thyroid carcinoma. A CT neck reveals one single lymph node in her left anterior cervical chain.

**QUESTION LINE**: What is the optimum treatment?

**OPTIONS**: - a) Thyroidectomy and neck dissection with postoperative radioiodine ablation - b) Thyroidectomy and neck dissection without postoperative radioiodine ablation - c) Lobectomy and neck dissection with postoperative radioiodine ablation - d) Lobectomy and neck dissection without postoperative radioiodine ablation - e) Monitor annually

**CORRECT-CHOICE LINE**: Correct answer is a.

**REASONING**: Diagnosis of thyroid tumours are frequently made after the patient has selfpalpated a neck lump or after an incidental finding following unrelated neck imaging. Most prevalent in young females, diagnosis is clinched on fine needle aspiration. The key considerations for treatment are the size of the thyroid mass and the presence of lymph node involvement: any lump greater than 1cm in size or has any signs of metastatic spread should undergo thyroidectomy instead of a lobectomy. The postoperative use of concurrent radioiodine ablation enhances survival benefits in patients at high risk of disease recurrence and is recommended by both the American Thyroid association and a European consensus group. Patients with thyroid lumps greater than 4cm in diameter regardless of extrathyroid disease; those with lumps between 1 and 4cm in diameter and extra-thyroid disease; and all with high-risk histology such as aggressive histological subtypes benefit from radioiodine ablation. In the case of this patient with a 2.5cm mass and lymph node involvement, thyroidectomy, lymph node clearance with neck dissection and postoperative radioiodine ablation is appropriate. Annual monitoring is thus an unsafe option.

**>>DESCRIPTION**: A 43-year-old female has a 2-month history of worsening neck discomfort. Examination shows a firm neck lump moving with swallowing. Ultrasound reveals a 2.5cm papillary thyroid carcinoma, and CT shows a single lymph node in the left anterior cervical chain.

**>>OPTIONS**: a) Lobectomy and neck dissection with postoperative radioiodine ablation b) Lobectomy and neck dissection without postoperative radioiodine ablation c) Monitor annually d) Thyroidectomy and neck dissection with postoperative radioiodine ablation e) Thyroidectomy and neck dissection without postoperative radioiodine ablation

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Thyroidectomy and neck dissection with postoperative radioiodine ablation

**>>REASONING**: Given the 2.5cm mass and lymph node involvement, thyroidectomy with neck dissection and postoperative radioiodine ablation is the appropriate treatment. Lobectomy is insufficient for masses greater than 1cm or with metastatic spread, and annual monitoring is an unsafe option.

## Question #:307

**CLINICAL SCENERIO**: A 37-year-old woman presents with a 5 cm thyroid nodule, moves on swallowing. History of anxiety/depression, taking sertraline. Mother with hypothyroidism. Fine needle aspiration reveals papillary thyroid cancer, no metastases.

**QUESTION LINE**: What is the most appropriate treatment?

**OPTIONS**: a) Total thyroidectomy followed by radioiodine-131 b) Localised radiotherapy c) Total thyroidectomy d) Total thyroidectomy followed by localised radiotherapy e) Radioiodine-13

**CORRECT-CHOICE LINE**: a

**REASONING**: In papillary thyroid cancer, the recommended treatment is total thyroidectomy followed by radioiodine-131 therapy. Total thyroidectomy removes the entire gland, and radioiodine-131 ablates residual tissue and treats potential metastases. Localized radiotherapy and radioiodine-131 alone are less effective. Total thyroidectomy without adjuvant therapy may not be enough.

**>>DESCRIPTION**: A 37-year-old woman with a 5 cm thyroid nodule (papillary thyroid cancer, no metastases).

**>>OPTIONS**: a) Localised radiotherapy b) Radioiodine-13 c) Total thyroidectomy d) Total thyroidectomy followed by localised radiotherapy e) Total thyroidectomy followed by radioiodine-131

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Total thyroidectomy followed by radioiodine-131

**>>REASONING**: The best treatment is total thyroidectomy followed by radioiodine-131 to remove the tumor and treat potential metastases. Other options are less effective or not appropriate.

## Question #:239

**CLINICAL SCENERIO**: A 54-year-old woman attends a follow-up endocrinology clinic after investigations for hyperthyroidism. She presents with a 4-month history of palpitations, sweating, fatigue, tremor, and 6kg weight loss. Thyroid function tests show TSH of 0.01 mU/L and free T4 of 28.3 pmol/L. Nuclear scintigraphy images are also available.

**QUESTION LINE**: Which of the following is the most likely cause for this woman’s presentation?

**OPTIONS**: a) Grave’s disease b) Hashimoto’s thyroiditis c) Solitary toxic nodule d) Follicular thyroid cancer e) Toxic multinodular goitre

**CORRECT-CHOICE LINE**: E

**REASONING**: The nuclear scintigraphy images show multiple localised areas of increased uptake throughout both lobes of a normal-sized thyroid gland, consistent with toxic multinodular goitre. Grave’s disease shows homogenous uptake in an enlarged gland. Hashimoto’s thyroiditis often presents with hypothyroidism. A solitary toxic nodule shows a single area of increased uptake. Follicular thyroid cancer rarely shows increased iodine uptake and is less likely to cause the described symptoms.

**>>DESCRIPTION**: A 54-year-old woman with hyperthyroidism presents with palpitations, sweating, fatigue, tremor, weight loss, and abnormal thyroid function tests. Nuclear scintigraphy images are available.

**>>OPTIONS**: a) Follicular thyroid cancer b) Grave’s disease c) Hashimoto’s thyroiditis d) Solitary toxic nodule e) Toxic multinodular goitre

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Toxic multinodular goitre

**>>REASONING**: Toxic multinodular goitre is the most likely cause due to the imaging findings of multiple areas of increased uptake and the patient’s symptoms and thyroid function tests. Other options are incorrect because Grave’s disease has homogenous uptake; Hashimoto’s thyroiditis usually presents with hypothyroidism; a solitary toxic nodule shows a single area of uptake; and follicular thyroid cancer does not usually have increased uptake and rarely causes these symptoms.

## Question #:211

**CLINICAL SCENERIO**: A 27-year-old female presents with secondary amenorrhoea after stopping the oral contraceptive pill 6 months ago, headaches, and difficulty standing/climbing stairs. Examination reveals breast milk expression and visual field defects. Lab results show elevated prolactin, suppressed FSH/LH, suppressed TSH, low T4, and low cortisol. MRI shows a 3cm pituitary mass.

**QUESTION LINE**: What is the next step in management?

**OPTIONS**: a) Bromocriptine b) Octreotide c) Stereotactic radiotherapy d) Trans-sphenoidal surgery e) Transcranial hypophysectomy

**CORRECT-CHOICE LINE**: d

**REASONING**: This patient has a macroadenoma causing visual field defects. Transsphenoidal surgery is the first step in management. Raised prolactin can be secondary to blockage of the pituitary stalk. Prolactinomas are treated with dopamine agonists. Octreotide is for acromegaly. Transcranial hypophysectomy is for very large tumors.

**>>DESCRIPTION**: A 27-year-old female presents with secondary amenorrhoea, headaches, and difficulty with mobility, with breast milk expression, visual field defects, and a 3cm pituitary mass on MRI.

**>>OPTIONS**: a) Bromocriptine b) Octreotide c) Stereotactic radiotherapy d) Trans-sphenoidal surgery e) Transcranial hypophysectomy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Trans-sphenoidal surgery

**>>REASONING**: Transsphenoidal surgery is the next step for macroadenoma management with visual field defects. Other options are incorrect as they are used for different conditions or situations.

## Question #:95

**CLINICAL SCENERIO**: A 37-year-old female presents with progressive loss of libido, persistent diarrhea (6 months), 16kg weight loss, and fatigue. She has protuberant eyes with double vision on lateral gaze and painful, watery eyes. Examination reveals hand tremor, irregularly irregular heart rate, marked exophthalmos, and thyroid bruit. Labs: TSH 0.03 mU/l, Total T4 302 nmol/l. CT orbits show taut optic nerves and retro-orbital edema.

**QUESTION LINE**: Which of the following would be the most appropriate management of her eye condition?

**OPTIONS**: a) Treatment with a block and replace regimen b) Treatment with radioactive iodine c) Treatment with IV methylprednisolone d) Surgical removal of the thyroid gland e) Orbital decompression surgery

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The question aims to address the ophthalmopathy associated with Graves disease and tests the candidates understanding of the correct approach to a patient with painful and significant eye disease. The treatment of choice is the administration of systemic steroids to lessen the inflammation and provide symptomatic relief. Treatment of the underlying thyrotoxicosis is essential, but it will not directly result in an improvement in the ophthalmopathy.

Radioactive iodine therapy may worsen Graves ophthalmopathy and should not be the initial treatment option. Treatment-induced hypothyroidism must be avoided as this may also worsen the eye problems.

**>>DESCRIPTION**: 37F with diarrhea, weight loss, fatigue, exophthalmos, double vision, and painful eyes. Exam: tremor, irregular heart rate, exophthalmos, thyroid bruit. Labs: low TSH, high T4. CT: optic nerve tautness, retro-orbital edema.

**>>OPTIONS**: a) Orbital decompression surgery b) Surgical removal of the thyroid gland c) Treatment with a block and replace regimen d) Treatment with IV methylprednisolone e) Treatment with radioactive iodine

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Treatment with IV methylprednisolone

**>>REASONING**: IV methylprednisolone is the treatment of choice for Graves’ ophthalmopathy to reduce inflammation and provide symptomatic relief. Treating thyrotoxicosis alone won’t directly improve the eye condition. Radioactive iodine can worsen ophthalmopathy.

## Question #:46

**CLINICAL SCENERIO**: A 53-year-old woman diagnosed with type 2 diabetes mellitus six months ago presents with fatigue and polyuria. She also has hypothyroidism. She was started on metformin 500mg twice daily but experienced gastrointestinal side effects like diarrhoea.

**QUESTION LINE**: What is the most appropriate action?

**OPTIONS**: a) Reduce to metformin 500mg once daily b) Change to dipeptidyl peptidase-4 inhibitor c) Trial of modified release metformin9 d) Change to sulfonylurea e) Change to pioglitazone

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The correct answer trial of modified release metformin. NICE guidelines advise to offer standard release metformin as the first-line treatment for type 2 diabetes and to gradually increase the dose to minimise the risk of gastrointestinal side effects. If gastrointestinal side effects are not tolerated, then a trial of modified release metformin would be appropriate. If metformin is not tolerated at all then a dipeptidyl peptidase-4 inhibitor, sulfonylurea or pioglitazone would be indicated.

**>>DESCRIPTION**: A 53-year-old woman with type 2 diabetes and hypothyroidism, initially treated with metformin 500mg twice daily, experiences significant gastrointestinal side effects.

**>>OPTIONS**: a) Change to dipeptidyl peptidase-4 inhibitor b) Change to pioglitazone c) Change to sulfonylurea d) Reduce to metformin 500mg once daily e) Trial of modified release metformin

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Trial of modified release metformin

**>>REASONING**: Modified-release metformin is appropriate for managing gastrointestinal side effects from standard metformin, according to NICE guidelines. Other options like DPP-4 inhibitors, sulfonylureas, or pioglitazone are considered if metformin is not tolerated at all.

## Question #:223

**CLINICAL SCENERIO**: A 29-year-old woman presents to the ENT clinic with hoarseness and a painless goitre for three weeks, without other thyroid symptoms. What is the most appropriate course of action?

**QUESTION LINE**: What is the most appropriate course of action?

**OPTIONS**: a) Immediate hospital admission under endocrinology b) Two-week wait appointment with an endocrinologist c) Routine appointment with an endocrinologist d) Reassure and discharge back to GP e) Arrange for bronchoscopy

**CORRECT-CHOICE LINE**: b

**REASONING**: Goitre with unexplained hoarseness should be referred urgently. The combination of thyroid swelling with hoarseness or change in voice which is unexplained needs urgent referral to endocrinology for likely thyroid cancer.

**>>DESCRIPTION**: A 29-year-old woman presents with hoarseness and a painless goitre. What is the most appropriate course of action?

**>>OPTIONS**: a) Immediate hospital admission under endocrinology b) Reassure and discharge back to GP c) Routine appointment with an endocrinologist d) Two-week wait appointment with an endocrinologist e) Arrange for bronchoscopy

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Two-week wait appointment with an endocrinologist

**>>REASONING**: Unexplained hoarseness with goitre warrants urgent referral to endocrinology to rule out thyroid cancer.

## Question #:101

**CLINICAL SCENERIO**: A 19 year-old man is referred by his GP to the outpatient department after having several episodes of collapse at college. He reports that during these episodes he feels tired and ‘blacks out’. Afterwards, he feels shaky and weak. There is no tongue biting or incontinence during these episodes and the patient reports that he often feels dizzy after standing up too quickly from a chair. The only other symptoms he reports is a sore throat that has persisted for a few weeks and lethargy.

On examination of the patient’s mouth and throat, there are some white plaques located at the back of the tongue and throat. His sitting blood pressure is 130/80 mmHg and his standing blood pressure is 95/70 mmHg. He is otherwise well.

Blood tests are performed and reveal:

Hb 13.9 g/dL Platelets 200 \* 10 9 /l WBC 6.2 \* 10 9 /l Na + 132 mmol/l K + 5.1 mmol/l Urea 4.7 mmol/l Creatinine 81 µmol/l Calcium 1.9 mmol/l Random glucose 3.9 mmol/l

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Type II polyglandular autoimmune syndrome - b) Thymoma - c) Type 1 polyglandular autoimmune syndrome - d) Type III polyglandular autoimmune syndrome - e) HIV

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: The most likely diagnosis is type 1 polyglandular autoimmune syndrome. This autosomal recessive syndrome is a subtype of autoimmune polyendocrine syndrome, whereby a number of endocrine glands dysfunction. The patient’s oral candidiasis is caused by a mild immune deficiency and hyposplenism. Furthermore, the patient has hypocalcaemia, caused by autoimmune dysfunction of the parathyroid gland and hypoglycaemia with hypotension, caused by autoimmune dysfunction of the adrenal gland.

**>>DESCRIPTION**: A 19-year-old man with collapse episodes, fatigue, and dizziness reports a sore throat and lethargy. Examination reveals oral plaques, sitting BP of 130/80 mmHg, and standing BP of 95/70 mmHg. Labs show Calcium 1.9 mmol/l and Random glucose 3.9 mmol/l.

**>>OPTIONS**: a) HIV b) Thymoma c) Type 1 polyglandular autoimmune syndrome d) Type II polyglandular autoimmune syndrome e) Type III polyglandular autoimmune syndrome

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Type 1 polyglandular autoimmune syndrome

**>>REASONING**: Type 1 polyglandular autoimmune syndrome is the most likely diagnosis, supported by oral candidiasis (immune deficiency), hypocalcemia (parathyroid dysfunction), and hypoglycemia with hypotension (adrenal dysfunction).

## Question #:326

**CLINICAL SCENERIO**: A 19-year-old man presents with recurrent collapse episodes, fatigue, and reports dizziness upon standing. Examination reveals white plaques in the mouth and throat. Blood pressure drops upon standing. Blood tests are provided, including Hb, platelets, WBC, electrolytes, urea, creatinine, calcium, and glucose.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: f) Type II polyglandular autoimmune syndrome g) Thymoma h) Type 1 polyglandular autoimmune syndrome i) Type III polyglandular autoimmune syndrome j) HIV

**CORRECT-CHOICE LINE**: c

**REASONING**: The most likely diagnosis is type 1 polyglandular autoimmune syndrome. This autosomal recessive syndrome is a subtype of autoimmune polyendocrine syndrome, whereby a number of endocrine glands dysfunction. The patient’s oral candidiasis is caused by a mild immune deficiency and hyposplenism. Furthermore, the patient has hypocalcaemia, caused by autoimmune dysfunction of the parathyroid gland and hypoglycaemia with hypotension, caused by autoimmune dysfunction of the adrenal gland.

**>>DESCRIPTION**: A 19-year-old man with collapse episodes, fatigue, dizziness, oral plaques, and orthostatic hypotension. Relevant blood test results are provided.

**>>OPTIONS**: a) HIV b) Thymoma c) Type 1 polyglandular autoimmune syndrome d) Type II polyglandular autoimmune syndrome e) Type III polyglandular autoimmune syndrome

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Type 1 polyglandular autoimmune syndrome

**>>REASONING**: The most likely diagnosis is Type 1 polyglandular autoimmune syndrome, supported by the patient’s oral candidiasis, hypocalcemia, and hypoglycemia. Other options are less likely given the presented clinical picture.

## Question #:156

**CLINICAL SCENERIO**: A 55-year-old woman presents with right-sided loin pain radiating to the groin, right-sided renal angle tenderness, and a history of Sjogren’s syndrome. Blood results reveal hypokalemia, hyperchloremia, low bicarbonate, and elevated glucose.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Addison’s diseas - b) Diabetic ketoacidosis - c) Type 1 renal tubular acidosis - d) Type 2 renal tubular acidosis - e) Type 4 renal tubular acidosis

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Type 1 ( distal ) renal tubular acidosis may be caused by Sjogren’s syndrome

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 2.5) - (120 + 14) = 4.5.

Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include (‘ABCD’):

*  Addison’s
*  Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
*  Chloride excess
*  Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:

*  Lactate
*  Toxins (e.g. methanol, paracetamol, propylene glycol)
*  Ketones
*  Renal failure

Type 1 renal tubular acidosis is correct. The presence of kidney stones, hypokalemia, NAGMA, and a past medical history of Sjogren’s favour the diagnosis of type 1 RTA.

Addison’s disease is incorrect. Although this can also cause a NAGMA, hyperkalemia would be expected as a consequence of hypoaldosteronism. Furthermore, patients with Addison’s will often be hypoglycaemic.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia and likely has undiagnosed diabetes mellitus, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This can also present with hypokalemia and NAGMA, however, there is not an association with Sjogren’s making this a less likely diagnosis.

Type 4 renal tubular acidosis is incorrect. Although diabetes is a cause of type 4 RTA, this condition would be associated with hyperkalemia in contrast to the hypokalemia demonstrated in this case.

**>>DESCRIPTION**: A 55-year-old woman with Sjogren’s syndrome presents with right loin pain, tenderness, hypokalemia, hyperchloremia, low bicarbonate, and elevated glucose.

**>>OPTIONS**: a) Addison’s diseas b) Diabetic ketoacidosis c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosis

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Type 1 renal tubular acidosis

**>>REASONING**: Type 1 renal tubular acidosis (RTA) is most likely due to a history of Sjogren’s and presents with hypokalemia and normal anion gap metabolic acidosis (NAGMA). Addison’s disease, DKA, type 2 and 4 RTA are less likely due to conflicting electrolyte abnormalities or lack of association with Sjogren’s.

## Question #:221

**CLINICAL SCENERIO**: A 28-year-old woman presents with dysphagia, fatigue, and gritty eyes. She struggles to swallow bread and has mild myalgia.

**QUESTION LINE**: What other finding would be consistent with the most likely diagnosis?

**OPTIONS**: a) C-ANCA positive b) P-ANCA positive c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosis

**CORRECT-CHOICE LINE**: c

**REASONING**: Type 1 ( distal ) renal tubular acidosis may be caused by Sjogren’s syndrome. This patient has features consistent with Sjogren’s syndrome. The xerostomia (dry mouth) can lead to difficulty swallowing, particularly foods needing more saliva. The other characteristic feature is xerophthalmia (dry eyes) which can make the eyes feel gritty and uncomfortable. Sjogren’s syndrome is associated with type 1 renal tubular acidosis. Type 1 (distal) renal tubular acidosis occurs due to an inability to excrete H+ in the distal tubule. The resulting acidosis is usually accompanied by hypokalaemia. Complications include nephrocalcinosis and renal stones. Causes include idiopathic, rheumatoid arthritis, SLE, Sjogren’s, amphotericin B toxicity, and analgesic nephropathy. A sometimes helpful reminder is that type 1 renal tubular acidosis is largely associated with structural/rheumatological problems, whereas, type 2 renal tubular acidosis is associated with metabolic disorders such as Fanconi syndrome, Wilson’s disease, and cystinosis. Type 4 renal tubular acidosis is associated with hypoaldosteronism and diabetes, rather than Sjogren’s. Type 4 is associated with hyperkalaemia, whilst types 1 and 2 are associated with hypokalaemia. Sjogren’s is typically associated with elevated levels of anti-Ro and anti-La. Neither cANCA nor pANCA are typically associated. ANCA antibodies are commonly associated with vasculitides. cANCA (cytoplasmic-ANCA) is associated with granulomatosis with polyangiitis. pANCA is found in microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis.

**>>DESCRIPTION**: A 28-year-old woman with dysphagia, fatigue, gritty eyes, and myalgia.

**>>OPTIONS**: a) C-ANCA positive b) P-ANCA positive c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosis

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Type 1 renal tubular acidosis

**>>REASONING**: Type 1 renal tubular acidosis is consistent with Sjogren’s syndrome, which is the most likely diagnosis given the patient’s symptoms. The other options are incorrect because they are not typically associated with Sjogren’s syndrome.

## Question #:218

**CLINICAL SCENERIO**: A 43-year-old man with glaucoma on acetazolamide presents with aches, pains, and proximal myopathy. Examination reveals no synovitis. Blood tests show hypokalemia, low bicarbonate, low calcium, raised PTH, and low vitamin D.

**QUESTION LINE**: What is the unifying diagnosis that explains the presentation?

**OPTIONS**: a) Osteoporosis b) Primary hyperparathyroidism c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosi

**CORRECT-CHOICE LINE**: d

**REASONING**: Type 2 renal tubular acidosis, caused by acetazolamide, explains hypokalemia, osteomalacia, and acidosis. Type 4 RTA causes hyperkalemia. Type 1 RTA is less likely to cause osteomalacia. Primary hyperparathyroidism and osteoporosis don’t fit the presentation.

**>>DESCRIPTION**: A 43-year-old man on acetazolamide for glaucoma presents with aches, proximal myopathy, hypokalemia, low bicarbonate, low calcium, raised PTH, and low vitamin D. What is the diagnosis?

**>>OPTIONS**: a) Osteoporosis b) Primary hyperparathyroidism c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal acidosi

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Type 2 renal tubular acidosis

**>>REASONING**: Type 2 renal tubular acidosis is the correct diagnosis due to acetazolamide use, hypokalemia, and osteomalacia. Type 4 RTA causes hyperkalemia. Type 1 RTA is less likely to cause osteomalacia. Primary hyperparathyroidism and osteoporosis are incorrect.

## Question #:299

**CLINICAL SCENERIO**: A 39-year-old man with a history of migraine with aura, treated with topiramate, presents to the neurology clinic. Relevant investigation results are provided, including electrolyte levels, venous blood gas analysis, and urinalysis findings.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Membranous glomerulonephritis b) Type 1 renal tubular acidosis c) Type 2 renal tubular acidosis d) Type 3 renal tubular acidosis e) Type 4 renal tubular acidosis

**CORRECT-CHOICE LINE**: c

**REASONING**: The correct answer is type 2 renal tubular acidosis . This patient has metabolic acidosis with hypokalaemia, proteinuria and glycosuria. The presence of proteinuria and glycosuria suggest a problem at the proximal convoluted tubule, as protein and glucose should be fully reabsorbed at this part of the nephron. Type 2 renal tubular acidosis is a consequence of a defect of the proximal tubular cells, leading to incomplete protein and glucose reabsorption, as well as defective bicarbonate reabsorption leading to acidosis. This is the most likely diagnosis in this patient and has likely been precipitated by the drug topiramate. Membranous glomerulonephritis is incorrect. To diagnose membranous glomerulonephritis, one would expect to see nephrotic range proteinuria (>3g per 24h), and ideally, histological confirmation on renal biopsy. Membranous glomerulonephritis is more associated with NSAIDs and penicillamine than topiramate and is unlikely to cause metabolic acidosis in the context of the normal renal function seen here. Type 1 renal tubular acidosis is incorrect. Although this can cause hypokalaemic metabolic acidosis, the distal tubular cells are affected rather than the cells of the proximal tubule. This means there is less of an association with proteinuria and glycosuria. Finally, topiramate is not known to precipitate type 1 renal tubular acidosis. Type 3 renal tubular acidosis is incorrect. This tends to be seen in children or as a result of congenital carbonic anhydrase deficiency. It is epidemiologically far rarer than type 2 renal tubular acidosis and is not specifically associated with topiramate use. Type 4 renal tubular acidosis is incorrect. This is associated with hyperkalemia rather than hypokalaemia and tends to be associated with mineralocorticoid receptor antagonists and NSAIDs rather than topiramate.

**>>DESCRIPTION**: A 39-year-old man with migraine on topiramate presents with electrolyte imbalances and urinalysis findings.

**>>OPTIONS**: a) Membranous glomerulonephritis b) Type 1 renal tubular acidosis c) Type 2 renal tubular acidosis d) Type 3 renal tubular acidosis e) Type 4 renal tubular acidosis

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Type 2 renal tubular acidosis

**>>REASONING**: Type 2 renal tubular acidosis is the most likely diagnosis due to metabolic acidosis, hypokalemia, proteinuria, and glycosuria, indicating a proximal tubular defect often linked to topiramate. Other options are less likely due to differing electrolyte imbalances or lack of association with the presenting symptoms or medication.

## Question #:48

**CLINICAL SCENERIO**: A 55-year-old male surgical patient is reviewed for persistent hyperkalaemia. He was admitted for 5 weeks following AP resection of sigmoid carcinoma complicated by a superficial wound infection. He has a history of type 2 diabetes mellitus, non-alcoholic steatohepatitis, and neuromyelitis optica. Regular medications include gliclazide, Lantus, prednisolone, and baclofen. Blood tests show K+ 6.9 mmol/l, raised renin, and decreased aldosterone. Blood gases show pH 7.24, PaO2 15.8 kPa, PaCO2 2.2 kPa, and bicarbonate 24 mmol/l. Urinary pH = 6.2. CT abdomen and pelvis demonstrate appropriate wound healing.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: - a) Type 1 renal tubular acidosis - b) Type 2 renal tubular acidosis - c) Type 4 renal tubular acidosis - d) Waterhouse-Friderichsen syndrome - e) Addisonian crisis

**CORRECT-CHOICE LINE**: Correct answer is c.

**REASONING**: Refractory hyperkalaemia in a patient with a prolonged illness should raise suspicions for adrenal insufficiency. Note that mineralocorticoid deficiency can occur with hyperkalaemia alone without hyponatraemia. In this case, the serum demonstrates a metabolic acidosis with normal bicarbonate and urinary pH greater than 5.5, ruling out type 1 and 2 renal tubular acidosis (RTA). WaterhouseFriedrichsen syndrome is caused by adrenal haemorrhage, classically secondary to tuberculosis or meningococcal infection, which if present, should be visualised on CT imaging. He does not demonstrate circulatory collapse, abdominal pain or nausea suggestive of Addisonian crisis. Type 4 RTA, causing a failure of the sodium-potassium antiporter is thus the most appropriate diagnosis, in the context of a patient with chronic steroid use and hence predisposition for adrenal insufficiency during acute severe illness.

**>>DESCRIPTION**: 55-year-old male post-sigmoid resection with persistent hyperkalaemia (K+ 6.9 mmol/l), raised renin, decreased aldosterone, and metabolic acidosis (pH 7.24). History includes T2DM, NASH, neuromyelitis optica, and current medications of gliclazide, Lantus, prednisolone, and baclofen. Urinary pH = 6.2. CT abdomen/pelvis shows normal wound healing.

**>>OPTIONS**: a) Addisonian crisis b) Type 1 renal tubular acidosis c) Type 2 renal tubular acidosis d) Type 4 renal tubular acidosis e) Waterhouse-Friderichsen syndrome

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Type 4 renal tubular acidosis

**>>REASONING**: Type 4 RTA is the most likely diagnosis due to chronic steroid use predisposing to adrenal insufficiency during illness, causing failure of the sodium-potassium antiporter. Type 1 and 2 RTA are less likely given the urinary pH > 5.5. Waterhouse-Friderichsen syndrome would be visualized on CT. Addisonian crisis is unlikely without circulatory collapse or abdominal pain.

## Question #:160

**CLINICAL SCENERIO**: A 64-year-old woman with a history of type 2 diabetes presents with heart palpitations. Her blood work reveals electrolyte imbalances.

**QUESTION LINE**: What is the most likely diagnosis?

**OPTIONS**: a) Addison’s disease b) Diabetic ketoacidosis (DKA) c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosis

**CORRECT-CHOICE LINE**: e

**REASONING**: Renal tubular acidosis (type 4) causes hyperkalaemia

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 6.5) - (125 + 12) = 5.5.

Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include (‘ABCD’):

*  Addison’s
*  Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
*  Chloride excess
*  Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:

*  Lactate
*  Toxins (e.g. methanol, paracetamol, propylene glycol)
*  Ketones
*  Renal failure

Type 4 renal tubular acidosis is correct. The presence of NAGMA, hyperkalemia, and a history of diabetes favours a diagnosis of type 4 renal tubular acidosis.

Type 1 renal tubular acidosis is incorrect. Although this condition also presents with NAGMA, it is associated with hypokalemia.

Addison’s disease is incorrect. Addison’s can result in NAGMA, with hyperkalemia as a consequence of hypoaldosteronism. However, the presence of hyperglycaemia and hypertension makes this a less likely diagnosis. One would expect hypoglycaemia and hypotension in Addison’s.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This condition is associated with NAGMA and an hypokalemia.

**>>DESCRIPTION**: A 64-year-old woman with type 2 diabetes presents with heart palpitations and electrolyte imbalances.

**>>OPTIONS**: a) Addison’s disease b) Diabetic ketoacidosis (DKA) c) Type 1 renal tubular acidosis d) Type 2 renal tubular acidosis e) Type 4 renal tubular acidosis

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Type 4 renal tubular acidosis

**>>REASONING**: Type 4 renal tubular acidosis is most likely due to the presence of normal anion gap metabolic acidosis (NAGMA), hyperkalemia, and a history of diabetes. Other options are less likely due to the absence of ketones (ruling out DKA), and the presence of hyperkalemia (ruling out type 1 and 2 RTA). Addison’s disease is less likely given the patient’s hyperglycemia and hypertension.

## Question #:260

**CLINICAL SCENERIO**: A 43-year-old man presents with a 4-week history of a 7mm lump on the right side of his neck, in the anterior triangle, which moves on swallowing. Blood tests are within normal limits.

**QUESTION LINE**: What is the most appropriate first-line investigation?

**OPTIONS**: a) Radioisotope scan of thyroid b) Magnetic resonance scan of head and neck c) Excision biopsy d) Fine needle aspiration biopsy e) Ultrasound scan of thyroid

**CORRECT-CHOICE LINE**: E

**REASONING**: High-resolution ultrasound scanning is an ideal first-line initial imaging investigation for most neck lumps. Because most lesions in the neck are sitespecific, once a lesion has been located, specific ultrasound features can be used to establish the diagnosis

**>>DESCRIPTION**: A 43-year-old man presents with a 7mm lump on the right side of his neck that moves on swallowing. What’s the best initial investigation?

**>>OPTIONS**: a) Excision biopsy b) Fine needle aspiration biopsy c) Magnetic resonance scan of head and neck d) Radioisotope scan of thyroid e) Ultrasound scan of thyroid

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Ultrasound scan of thyroid

**>>REASONING**: Ultrasound is the best first step for neck lumps. It helps locate the lesion and find features to diagnose it.

## Question #:26

**CLINICAL SCENERIO**: A 75-year-old woman presents to her general practitioner (GP) due to increased tiredness and weight gain. Her past medical history includes hypothyroidism and hypertension, for which she takes levothyroxine and amlodipine.

She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

**QUESTION LINE**: What is the most likely cause of her symptoms?

**OPTIONS**: a) Bowel malignancy b) Addison’s disease c) Amlodipine toxicity d) Undercorrected hypothyroidism e) Worsening anaemia

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: The most likely explanation here is undercorrected hypothyroidism. Ferrous sulphate is well known to reduce the absorption of other medications, particularly levothyroxine. Patients should be advised to take these tablets separately from their regular medications.

Although anaemia in a post-menopausal woman should always be investigated, there are no symptoms to suggest malignancy here. Addison’s disease would be unlikely to cause weight gain.

**>>DESCRIPTION**: 75-year-old woman presents with increased tiredness and weight gain. History of hypothyroidism (on levothyroxine) and hypertension (on amlodipine). Started on ferrous sulphate for mild anaemia two weeks prior.

**>>OPTIONS**: a) Addison’s disease b) Amlodipine toxicity c) Bowel malignancy d) Undercorrected hypothyroidism e) Worsening anaemia

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Undercorrected hypothyroidism

**>>REASONING**: Undercorrected hypothyroidism is most likely due to ferrous sulphate reducing levothyroxine absorption. Addison’s disease typically does not cause weight gain, and there are no symptoms suggestive of malignancy.

## Question #:31

**CLINICAL SCENERIO**: A 75-year-old woman presents to her general practitioner (GP) due to increased tiredness and weight gain. Her past medical history includes hypothyroidism and hypertension, for which she takes levothyroxine and amlodipine. She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

**QUESTION LINE**: What is the most likely cause of her symptoms?

**OPTIONS**: a) Bowel malignancy b) Addison’s disease c) Amlodipine toxicit d) Undercorrected hypothyroidism e) Worsening anaemia

**CORRECT-CHOICE LINE**: Correct anssewr is d.

**REASONING**: The most likely explanation here is undercorrected hypothyroidism. Ferrous sulphate is well known to reduce the absorption of other medications, particularly levothyroxine. Patients should be advised to take these tablets separately from their regular medications.

Although anaemia in a post-menopausal woman should always be investigated, there are no symptoms to suggest malignancy here. Addison’s disease would be unlikely to cause weight gain.

**>>DESCRIPTION**: A 75-year-old woman with hypothyroidism, hypertension, and mild anemia presents with increased tiredness and weight gain. She takes levothyroxine, amlodipine, and was recently started on ferrous sulphate.

**>>OPTIONS**: a) Addison’s disease b) Amlodipine toxicit c) Bowel malignancy d) Undercorrected hypothyroidism e) Worsening anaemia

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Undercorrected hypothyroidism

**>>REASONING**: Undercorrected hypothyroidism is the most likely cause because ferrous sulphate reduces levothyroxine absorption. Bowel malignancy is less likely due to the absence of specific symptoms. Addison’s disease would not typically cause weight gain.

## Question #:345

**CLINICAL SCENERIO**: A 75-year-old woman presents to her general practitioner (GP) due to increased tiredness and weight gain. Her past medical history includes hypothyroidism and hypertension, for which she takes levothyroxine and amlodipine. She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

**QUESTION LINE**: What is the most likely cause of her symptoms?

**OPTIONS**: - f) Bowel malignancy - g) Addison’s disease - h) Amlodipine toxicity - i) Undercorrected hypothyroidism - j) Worsening anaemia

**CORRECT-CHOICE LINE**: Correct answer is d.

**REASONING**: The most likely explanation here is undercorrected hypothyroidism. Ferrous sulphate is well known to reduce the absorption of other medications, particularly levothyroxine. Patients should be advised to take these tablets separately from their regular medications. Although anaemia in a post-menopausal woman should always be investigated, there are no symptoms to suggest malignancy here. Addison’s disease would be unlikely to cause weight gain.

**>>DESCRIPTION**: A 75-year-old woman with hypothyroidism (on levothyroxine) and hypertension (on amlodipine) presents with increased tiredness and weight gain. Two weeks prior, she started ferrous sulphate for mild anaemia.

**>>OPTIONS**: a) Addison’s disease b) Amlodipine toxicity c) Bowel malignancy d) Undercorrected hypothyroidism e) Worsening anaemia

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Undercorrected hypothyroidism

**>>REASONING**: Undercorrected hypothyroidism is the most likely cause. Ferrous sulphate, recently started, can reduce levothyroxine absorption, leading to symptoms. Other options are less likely: no malignancy symptoms, and Addison’s disease typically causes weight loss, not gain.

## Question #:250

**CLINICAL SCENERIO**: A 45-year-old man presents to the endocrine clinic for review with weight gain, hypertension, and abdominal striae. Initial 24hr urinary free cortisol is elevated. Which finding suggests an adrenal adenoma producing cortisol?

**QUESTION LINE**: Which of the following would be most suggestive of an adrenal adenoma producing cortisol?

**OPTIONS**: a. Normal 9am serum cortisol b. Raised urinary free cortisol on repeat testing c. Serum cortisol of 220 mmol/l at 9am after an overnight dexamethasone suppression test d. Serum potassium of 2.4 mmol/l e. Undetectable levels of ACTH6

**CORRECT-CHOICE LINE**: E

**REASONING**: The key fact is that an adrenal adenoma producing cortisol would suppress the release of ACTH. For this reason, if ACTH is undetectable then an adrenal tumour is the most likely cause. In this case, a CT abdomen would be the obvious next step.

Normal 9 am serum cortisol would make a diagnosis of Cushing’s less likely, and a raised urinary free cortisol on repeat testing is not specific for the underlying cause. Failure to suppress after overnight dexamethasone suppression test merely confirms a diagnosis of Cushing’s. Serum potassium levels as low as 2.4 mmol/l would be very unusual in a case of Cushing’s.

**>>DESCRIPTION**: A 45-year-old man with weight gain, hypertension, abdominal striae, and elevated urinary free cortisol presents to the endocrine clinic. Which finding suggests an adrenal adenoma?

**>>OPTIONS**: a. Normal 9am serum cortisol b. Raised urinary free cortisol on repeat testing c. Serum potassium of 2.4 mmol/l d. Serum cortisol of 220 mmol/l at 9am after an overnight dexamethasone suppression test e. Undetectable levels of ACTH6

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Undetectable levels of ACTH6

**>>REASONING**: Undetectable ACTH suggests an adrenal adenoma because the tumor suppresses ACTH production. Other options are less specific or less indicative of the cause.

## Question #:43

**CLINICAL SCENERIO**: An 18-year-old woman is brought into the hospital at midnight with generalised weakness and mild difficulty breathing.

On examination, there is weakness in the upper and lower limbs with proximal predominance. Reflexes in the upper limbs are reduced. Muscle tone is normal. Chest sounds clear but expansion is reduced bilaterally. GCS is 15/15.

The patient describes similar episodes in the past that have lasted for only minutes, the episodes decreased in frequency after commencing a ketogenic diet. They had been attributed to hemiplegic migraines, though she had had bilateral symptoms.

**QUESTION LINE**: From the following options, what investigation will be most helpful in confirming

the likely diagnosis?

**OPTIONS**: - a) CT head - b) Electromyography - c) Lumbar puncture - d) MRI brain and spinal cord - e) Urea & electrolytes

**CORRECT-CHOICE LINE**: Episodes of weakness, hypokalaemia, symptoms triggered by carbohydrate meals → hypokalaemic periodic paralysis

Urea and electrolytes is the correct answer.

**REASONING**: The most likely diagnosis is a periodic paralysis of which hypokalaemic periodic paralysis is the most common. Attacks can be precipitated by high-carbohydrate meals which explains why the number of episodes reduced for this patient when she commenced on a low-carbohydrate ketogenic diet.

Differentials might include myasthenia gravis (though this is commonly associated with fatigable weakness rather than ‘attacks’), metabolic myopathies (which involve weakness typically worsened by exercise), or in a first attack Guillain-Barre, transverse myelitis, of infective paralysis (tick paralysis, botulism) may be considered.

Urea and electrolytes is the correct answer. A low potassium level would make the diagnosis here clearer. Average potassium levels in hypokalaemic periodic paralysis are around 2.4mmol/L. If hypokalaemia is significantly low, less than 2.0mmol/L for example, you should think about secondary causes of hypokalaemia such as losses from the gut, and renal losses.

MRI brain and spinal cord is incorrect. This would be a reasonable investigation to investigate the possibility of multiple sclerosis (MS). Here, the pattern of weakness does not fit a particular cerebral or spinal territory which you would expect with MS.

Electromyography is incorrect. Although this would be able to demonstrate myopathy during an acute attack, it would not assist us in determining the specific underlying diagnosis as there are many causes of myopathy.

Lumbar puncture is incorrect. A lumbar puncture would be helpful in looking for inflammatory or infective causes of neurological symptoms, but would not be able to confirm a diagnosis of hypokalaemic periodic paralysis. Given the episodic nature of the attacks, transverse myelitis or infection, for example, would not explain the symptoms here.

CT head is incorrect. This patient’s symptoms are recurrent and do not correspond to a particular cerebral territory. A CT head would be unhelpful in confirming the likely diagnosis.

**>>DESCRIPTION**: 18-year-old woman presents with generalized weakness and mild breathing difficulty. Examination reveals proximal limb weakness, reduced reflexes, and decreased chest expansion. She has a history of similar, brief episodes, which decreased on a ketogenic diet. Initially attributed to hemiplegic migraines with bilateral symptoms.

**>>OPTIONS**: a) CT head b) Electromyography c) Lumbar puncture d) MRI brain and spinal cord e) Urea & electrolytes

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Urea & electrolytes

**>>REASONING**: Urea & electrolytes are most helpful in confirming hypokalemic periodic paralysis. Low potassium would support the diagnosis. MRI, EMG, LP, and CT head are less likely to confirm this episodic condition.

## Question #:17

**CLINICAL SCENERIO**: A 68-year-old gentleman with small cell carcinoma of the lung, COPD, ischaemic heart disease, hypertension, hypercholesterolaemia, and depression was admitted with increasing drowsiness and new onset confusion. He had new onset generalised aches and pains treated with Oramorph, and developed abdominal pain diagnosed as opiate-induced constipation. His medications included oramorph, paracetamol, dihydrocodeine, lactulose, aspirin, atorvastatin, bisoprolol, Ramipril, and furosemide. Examination revealed drowsiness (GCS 12), BP 102/68, HR 58bpm, RR 10/min, SpO2 95% on air, and temperature 36.6ºC. Neurological examination was limited but no focal signs were found.

**QUESTION LINE**: Which investigation is most likely to be diagnostic of the underlying cause?

**OPTIONS**: - a) Urgent CT head scan - b) Urgent serum liver function and calcium profile - c) Urgent isotope bone scan - d) Urgent septic screen - e) Urgent PET scan

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: This gentleman has developed signs of life-threatening hypercalcaemia, having manifested previous potential symptoms including new onset abdominal pain and constipation. This question is asking what the single next most important investigation should be, and whilst most of the above options are relevant, candidates are asked to discriminate from what is deemed to be the next essential management should be. It is likely that he has developed metastasis to his spine thus whilst an isotope bone scan may be an important part of his overall management, it is not as relevant in the acute setting. Likewise, it is likely that a septic screen and CT head would be performed, but clinically speaking there is little definite evidence of acute raised intracranial pressure or sepsis. There is no indication at present that he is suffering from opiate toxicity and therefore IV naloxone should not be administered at this stage.

**>>DESCRIPTION**: A 68-year-old man with small cell lung cancer presents with increasing drowsiness and confusion. He has a history of COPD, IHD, hypertension, hypercholesterolemia, and depression. He was recently started on Oramorph for pain and developed constipation. Examination shows drowsiness (GCS 12), BP 102/68, HR 58, RR 10, and no focal neurological signs.

**>>OPTIONS**: a) Urgent CT head scan b) Urgent isotope bone scan c) Urgent PET scan d) Urgent septic screen e) Urgent serum liver function and calcium profile

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Urgent serum liver function and calcium profile

**>>REASONING**: The patient likely has life-threatening hypercalcemia, suggested by drowsiness, abdominal pain, and constipation. Serum liver function and calcium profile is the most important next investigation. While other investigations may be relevant, they are not as crucial in the acute setting. There’s no strong evidence for acute raised intracranial pressure, sepsis, or opiate toxicity.

## Question #:220

**CLINICAL SCENERIO**: A 21-year-old female presents with a two-month history of lethargy, muscle pain, and weight loss. Examination is unremarkable. Observations: heart rate 72/min, respiratory rate 14/min, oxygen saturations 99% on air, blood pressure 110/80mmHg, apyrexial. Routine bloods show: Hb 150 g/l, Platelets 200 \* 10 9 /l, WBC 12.0 \* 10 9 /l, Neutrophils 8.0 \* 10 9 /l, Lymphocytes 4.0 \* 10 9 /l, Na + 128 mmol/l, K + 2.9 mmol/l, Urea 7.0 mmol/l, Creatinine 85 µmol/l, CRP 11 mg/l.

**QUESTION LINE**: What is the next most appropriate investigation?

**OPTIONS**: a) Computed tomography (CT) of the thorax b) ltrasound of the renal tract c) Serum aldosterone d) Urinary electrolytes e) Short synacthen test

**CORRECT-CHOICE LINE**: d

**REASONING**: The combination of lethargy, weight loss, low sodium and low potassium in a young patient suggest an inherited renal pathology such as Bartter’s or Gitelman’s syndrome. Liddle’s syndrome is usually associated with high blood pressure.

Urinary electrolytes are a quick and simple test and would be likely to aid the diagnosis here.

Most other causes alluded to here would not cause both sodium and potassium to be low.

**>>DESCRIPTION**: A 21-year-old female presents with lethargy, muscle pain, and weight loss. Blood tests reveal hyponatremia and hypokalemia. What is the next most appropriate investigation?

**>>OPTIONS**: a) Computed tomography (CT) of the thorax b) Serum aldosterone c) Short synacthen test d) Urinary electrolytes e) Ultrasound of the renal tract

**>>CORRECT-CHOICE LINE**: d

**>>CORRECT-CHOICE\_TEXT**: Urinary electrolytes

**>>REASONING**: The patient’s hyponatremia and hypokalemia, along with other symptoms, suggest a renal pathology like Bartter’s or Gitelman’s syndrome. Urinary electrolytes are the next best step to aid in diagnosis. Other options are less helpful in this scenario.

## Question #:290

**CLINICAL SCENERIO**: A 73-year-old woman with hyponatremia, found incidentally after a resolved chest infection and diagnosed with small cell lung cancer. She is euvolemic and suspected of having SIADH.

**QUESTION LINE**: What further investigation would be necessary to diagnose SIADH?

**OPTIONS**: a. Random serum cortisol b. Echocardiogram c. Lung biopsy d. Urinary electrolytes and osmolality e. Water deprivation test

**CORRECT-CHOICE LINE**: D

**REASONING**: SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient. This patient has established hyponatraemia with low serum osmolality. She needs to have elevated urinary osmolality to confirm the diagnosis of SIADH in the context of hyponatraemia and no other cause such as hypervolaemia. A morning cortisol would be helpful in diagnosing Addison’s disease, and if suspected would be a useful investigation, but a random serum cortisol would not be very helpful. An echocardiogram could exclude heart failure in the context of fluid overload as a cause of hyponatraemia, but in the absence of peripheral oedema, a raised JVP and pulmonary oedema this is unlikely to be necessary. Water deprivation test is done to confirm diabetes insipidus

**>>DESCRIPTION**: A 73-year-old euvolemic woman with hyponatremia, small cell lung cancer, and suspected SIADH.

**>>OPTIONS**: a. Echocardiogram b. Random serum cortisol c. Urinary electrolytes and osmolality d. Lung biopsy e. Water deprivation test

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: Urinary electrolytes and osmolality

**>>REASONING**: The diagnosis of SIADH is confirmed by demonstrating elevated urinary osmolality in the context of hyponatremia and low serum osmolality. Other investigations are less relevant: cortisol is for Addison’s, echocardiogram for fluid overload, and water deprivation is for diabetes insipidus.

## Question #:206

**CLINICAL SCENERIO**: A 19-year-old girl presents with lethargy, weakness, muscle cramps, urinary frequency, dehydration, and amenorrhea. Examination reveals a BMI of 17 kg/m², heart rate of 88 bpm, and blood pressure of 108/86 mmHg. Lab results show hypokalemia, metabolic alkalosis.

**QUESTION LINE**: What would be the next most useful investigation?

**OPTIONS**: - a) Transvaginal ultrasound (TVUS) of the ovaries - b) Urine diuretic assay - c) Early morning cortisol - d) Serum renin and aldosterone levels - e) Fasting blood glucose levels

**CORRECT-CHOICE LINE**: b

**REASONING**: The patient’s hypokalemia, metabolic alkalosis, and normal-low blood pressure suggest diuretic abuse, Bartter’s syndrome, or Gitelman’s syndrome. Diuretic abuse is the most common cause, especially in young women, and can be investigated with a urine diuretic assay. Bartter’s and Gitelman’s syndromes are less likely initially.

**>>DESCRIPTION**: A 19-year-old female presents with lethargy, weakness, muscle cramps, urinary frequency, dehydration, amenorrhea, hypokalemia, and metabolic alkalosis. What is the most useful next investigation?

**>>OPTIONS**: a) Early morning cortisol b) Fasting blood glucose levels c) Serum renin and aldosterone levels d) Transvaginal ultrasound (TVUS) of the ovaries e) Urine diuretic assay

**>>CORRECT-CHOICE LINE**: e

**>>CORRECT-CHOICE\_TEXT**: Urine diuretic assay

**>>REASONING**: A urine diuretic assay is the most useful next investigation to rule out diuretic abuse, the most likely cause given the clinical and biochemical findings. Other options address different possible underlying causes (ovarian, adrenal, glucose imbalances) but are less directly relevant to the primary concern of hypokalemia and alkalosis.

## Question #:53

**CLINICAL SCENERIO**: A 37-year-old man is being investigated for a chronic metabolic acidosis. He has an anion gap of 9 mEq/L and your consultant suspects he may have a renal source of his acidaemia.

**QUESTION LINE**: Which of the following can lead to renal tubular acidosis (type 2)?

**OPTIONS**: a) Sjogren’s syndrome b) Wilson’s disease c) Haemochromatosis d) NSAIDs e) Nephrocalcinosis

**CORRECT-CHOICE LINE**: Correct answer is b.

**REASONING**: Type 2 renal tubular acidosis may be caused by Fanconi syndrome

The answer is Wilson’s disease.

Renal tubular acidosis (RTA) is a renal cause of a metabolic acidosis. It has 3 main categories:

*  Type 1: Distal (failure of proton secretion)
*  Type 2: Proximal (failure of bicarbonate reabsorption)
*  Type 4: Aldosterone deficiency/insensitivity

Type 3 is a combination of 1 and 2.

Types 1 & 2 are characterised by hypokalaemia and type 4 is characterised by hyperkalaemia. Sjogren’s syndrome is one of the classic causes of Type 1 RTA, as well as nephrocalcinosis being both a common cause and consequence of long standing RTA.

NSAIDs lead to aldosterone resistance, causing a Type 4 RTA.

Wilson’s disease causes a form of Type 2 RTA called Fanconi syndrome. It can also be caused by other toxic heavy metals (lead & mercury) as well as expired tetracyclines. It leads to proximal loss of protons, phosphate, amino acids and glucose.

Haemochromatosis is a distractor.

**>>DESCRIPTION**: A 37-year-old man with chronic metabolic acidosis (anion gap 9 mEq/L) is suspected to have renal tubular acidosis.

**>>OPTIONS**: a) Haemochromatosis b) Nephrocalcinosis c) NSAIDs d) Sjogren’s syndrome e) Wilson’s disease

**>>CORRECT-CHOICE LINE**: Correct answer is e.

**>>CORRECT-CHOICE\_TEXT**: Wilson’s disease

**>>REASONING**: Wilson’s disease can cause Fanconi syndrome, which leads to type 2 RTA due to failure of bicarbonate reabsorption in the proximal tubule. Sjogren’s syndrome causes type 1 RTA. NSAIDs cause type 4 RTA.

## Question #:113

**CLINICAL SCENERIO**: A 30-year-old male with background of type one diabetes mellitus presents with abdominal pain and shortness of breath. Investigations confirm he has diabetic ketoacidosis.

**QUESTION LINE**: Which one of the following investigations would suggest a discussion

for possible intensive care admission?

**OPTIONS**: - a) Lactate 3 mmol/L - b) Bicarbonate level 19 mmol/L - c) pH 7.27 - d) White cell count 30 x 10^9/L - e) Potassium 3.4 mmol/L

**CORRECT-CHOICE LINE**: Parameters indicate severe diabetic ketoacidosis:

*  pH < 7

**REASONING**: Parameters indicate severe diabetic ketoacidosis:

*  pH < 7
*  Blood ketone > 6 mmol/L
*  Bicarbonate < 5 mmol/L
*  Anion gap >16 mmol/l
*  Potassium < 3.5 mmol/L on admission
*  Tachycardia or bradycardia
*  Systolic blood pressure <90 mmHg
*  Oxygen saturation <92% on air
*  GCS < 12

**>>DESCRIPTION**: A 30-year-old male with type one diabetes presents with abdominal pain, shortness of breath, and confirmed diabetic ketoacidosis.

**>>OPTIONS**: a) Bicarbonate level 19 mmol/L b) Lactate 3 mmol/L c) pH 7.27 d) Potassium 3.4 mmol/L e) White cell count 30 x 10^9/L

**>>CORRECT-CHOICE LINE**: c

**>>CORRECT-CHOICE\_TEXT**: pH 7.27

**>>REASONING**: A pH of 7.27 does not meet the criteria for severe DKA, which requires a pH < 7. The question asks which value would suggest ICU admission, and the severe DKA criteria are indicators for this.