Endocrine system

Thyrotoxicosis

Diagnostic tools

1. Patient complaints- significant weight loss, palpitation, heat intolerance, sleeplessness, excessive sweating, hyperdefecation, loss of appetite (in old age).

2. On examination: pulse tachycardia (may be irregular if atrial fibrillation), tremor- present in out stretched hand, lid retraction, lid lag, exophthalmos and thyroid gland may be palpable.

3. Investigation-S. TSH, T3, T4 (T3 and T4 will be raised and S. TSH will be low). To determine the underlying cause of thyroxicosis- TSH receptor antibodies (TRAb, elevated in Graves' disease) and radioisotope scanning.

Management

Treatment options-may be one of the following three

1. Antithyroid drugs- indicated for patient less than 40 years. Antithyroid drugs are carbimazole (40-60mg daily), methimazole and propylthiouracil (400-600 mg daily).

2. Radio 131I ablation- in patient with age more than 40 years, recurrence following surgery irrespective of age and in patient with other serious co-morbidity.

3. Surgery-subtotal thyroidectomy, usually indicated for patient with multinodular goiter (patient should make euthyroid before surgery by potassium iodide 60 mg 8 hourly for 2 weeks).

Commonly following treatment is given

1. Tablet carbimazole 40 -60 mg daily, when patient become euthyroid then 5-20 mg for 18 months. If relapse occur then surgery (subtotal thyroidectomy).
2. Betablocker-propanolol 20-40 mg daily, gradually taper after patient becomes euthyroid to control symptoms due to sympathetic over activity.

N:B: Beta-blockers should not be used for long-term treatment of thyrotoxicosis but are extremely useful in the short term, while patients are awaiting hospital consultation or following 131I therapy. Verapamil may be used as an alternative to β-blockers, e.g. in patients with asthma.

Generic name	Dose	Disadvantage
Carbimazole	Carbimazole 5 mg (খাবার পর) ৩ টি বড়ি সকালে, ৩ টি বড়ি দুপুরে ও ৩ টি বড়ি রাতে খাবেন চলবে। when patient becomes euthyroid- Carbimazole 5 mg (খাবার পর) ১টি বড়ি সকালে, ১টি বড়ি দুপুরে ও ১টি বড়ি রাতে খাবেন চলবে।	Hypersensitivity rash 2%, Agranulocytosis 0.2%, > 50% relapse rate usually within 2 years of stopping drug
Propanolol	Propanolol 10 mg ২ টি বড়ি সকালে ও ২ টি বড়ি রাতে খাবেন ৩০ দিন, এরপর ১ টি বড়ি সকালে ও ১ টি বড়ি রাতে খাবেন ৫ দিন, এরপর ১ টি বড়ি সকালে খাবেন ৫ দিন, এরপর ১/২ বড়ি সকালে খাবেন ৫ দিন।	

* Subjective improvement occur within 10-14 days.

* Patient biochemically euthyroid at 3-4 weeks. At this time carbimazole should be given at a dose of 5-20 mg daily for 12- 15 months.

Atrial fibrillation in thyrotoxicosis

1. Atrial fibrillation occurs in about 10% of patients with thyrotoxicosis.

2. The incidence increases with age and subclinical thyrotoxicosis.

3. Characteristically, the ventricular rate is little influenced by digoxin but responds to the addition of a beta-blocker.

4. Thromboembolic vascular complications are particularly common in thyrotoxic atrial fibrillation so that anticoagulation is required.

5. Once thyroid hormone and TSH concentrations have been returned to normal, atrial fibrillation will spontaneously revert to sinus rhythm in about 50% of patients but cardioversion may be required in the remainder.

Thyrotoxic storm or crisis (It is a medical emergency)

Diagnostic tools

1. Thyrotoxic crisis is most commonly precipitated by infection

in a patient with previously unrecognised or inadequately treated thyrotoxicosis, shortly after thyroidectomy in an ill-prepared patient or within a few days of 131I therapy, when acute radiation damage may lead to a transient rise in serum thyroid hormone levels.

2. The most prominent signs are fever, agitation, delirium, tachycardia or atrial fibrillation and in the older patient, cardiac failure.

3. Investigation-CBC (neutrophilic leucocytosis if any infection), TSH, T3, T4 (TSH will be low or undetectable, T3, T4 raised).

Management

1. Diet - normal

2. Rehydration with normal saline. 3. Propanolol 80 mg 6 hourly orally or 1-5 mg IV 6 hourly.

4. Sodium ipodate 500 mg orally or dexamethasone 2 mg 6 hourly + amiodarone.

5. Carbimazole if cannot take orally then give per rectally.

6. Antibiotic (broad spectrum)-if any features of infection.

Graves opthalmopathy

Management

1. The majority of patients require no treatment other than reassurance.

2. Stop smoking

3. Use side shield attached spectacle.

4. Methylcellulose eye drop & gell.

5. Mild Graves' ophthalmopathy-oral selenium (100 µg twice daily for 6 months)

6. Severe cases-prednisolone (short term 7-10 days, 1 mg/ kg body weight) and sometimes orbital radiotherapy.

7. There is also an increasing trend to use alternative immunosuppressive therapies, such as rituximab and cyclosporin.

8. If loss of visual acquity then urgent surgical decompression.

Hypothyroidism

Diagnostic tools

1. Patients complaints-rapid weight gain, cold intolerance, somnolence, swelling of whole body etc.

2. On examination- bradycardia, skin-dry, rough; non-pitting edema, slow and delayed relaxation of the ankle jerk.

3. Investigation-S. TSH and FT4 (FT4 will be low and TSH usually high).

Management

Thyroxine replacement for life long.

1. Levothyroxine (50µgm) daily (half hour before breakfast) for 3 weeks then 100 µgm daily. After 6 weeks do FT4 and TSH then adjust dose of levothyroxine (usually 100-150 µgm). Levothyroxine usually needs to continue for life long period.

Note:

1) Patient usually feel better within 2-3 weeks.

2) Reduction in weight & periorbital puffiness occurs quickly.

3) Restoration of skin & hair texure and resolution of any effusion take 3-6 months.

4. After become euthyroid FT4 and TSH should be checked 1-2 yearly.

Hypothyroidism in IHD

The basic principle of management of hypothyroidism in IHD is to start levothyroxine at a lower dose than usual (25 µgm or lower), because it can aggravate or precipitate IHD. It will be better to start a beta blocker before starting levothyroxine.

Management

1. Diet- normal, avoid fatty & salty food

2. Levothyroxine (25 µgm) daily (half hour before breakfast) for 3 weeks. (after 3 weeks dose should be increased).

3. Coronary intervention may be required if angina is exacerbated by levothyroxine replacement therapy.

Hypothyroidism in pregnancy

Management

Levothyroxine replacement therapy dose requirements: increase by 30–50% from early in pregnancy. Because placenta metabolise thyroxine & increase thyroxine binding globulin in pregnancy. FT4 & TSH should be assessed in every trimester.

Increased dose of levothyroxine is required in the following condition

1. Pregnancy

2. Anticonvulsant drugs

3. Rifampicin

4. Sertraline

5. Aluminium hydroxide

6. Ferrous sulphate

7. Calcium carbonate

8. Malabsorption

9. Chloroquine

Thyrotoxicosis in pregnancy

1. Gestational thyrotoxicosis: Usually associated with multiple pregnancies and hyperemesis gravidarum. Transient and usually does not require antithyroid drug treatment.

2. Graves' disease: The most common cause of sustained thyrotoxicosis in pregnancy.

3. Antithyroid drugs: Propylthiouracil should be used in the first trimester, with carbimazole substituted in the second and third trimesters. Surgery if poor compliance (usually done in 2nd trimester).

Post-partum thyroiditis

1. Screening: not recommended for every woman.

2. TSH, T3, T4 should be tested 4–6 weeks post-partum in those with history of thyroid disease, goitre or other autoimmune disease including type 1 diabetes, in those known to have positive antithyroid peroxidase antibodies, or when there is clinical suspicion of thyroid dysfunction.

Myxoedema coma

Diagnostic tools

1. Elderly patient.

2. History of hypothyroidism.

3. Altered consciousness, convulsion.

4. Hypothermia-temperature may be as low as 25 °C.

Management (treatment should be started before getting the laboratory reports)

1. Slow rewarming-blanket.

2. NG tube feeding.

3. O2 inhalation SOS

4. Broad spectrum antibiotics.

5. Intravenous injection of 20 μg liothyronine, followed by further injections of 20 μg 3 times daily until there is sustained clinical improvement. If patient recover then after 48–72 hours, it is usually possible to switch patients to oral levothyroxine in a dose of 50 μg daily.

6. Hydrocortisone 100 mg IM 8 hourly.

Diabetes mellitus

Diagnostic tools

1. Patient usually asymptomatic

2. Patient may compliant-weight loss, frequency of micturition, increase thirst, weakness etc.

3. Incidentally found high RBS>7.8 mmol/l

4. Patient with above features should have FBS & 2 ABF or OGTT.

5. Diabetes is confirmed if

(a) Either plasma glucose in random sample or 2 hours after a 75 g glucose load ≥ 11.1 mmol/L (200 mg/dL) or

(b) Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) or

(c) HbA1c ≥ 48 mmol/mol (>6.5%)

In asymptomatic patients, two diagnostic tests are required to confirm diabetes; the second test should be the same as the first test to avoid confusion.

6. When a patient diagnosed as diabetic then identification of complications is necessary prior to start treatment. S. creatinine, urine R/M/E-to see any nephropathy, ECG- to see any IHD, fasting lipid profiles-to see dyslipidaemia.

Management of DM

Following are the treatment options of DM either singly or in combination

1. Lifestyle and dietary modifications

2. OAD

3. Insulin

Life style and dietary modifications are

1. Maintain normal weight (reduce weight if overweight & obese)

2. Avoid physical inactivity

3. Regular physical exercise (brisk walking for 30 minutes per day, ideally on most of days of the week)

4. Meal plana Breakfast-ruti 2 to 3 (medium size) with vegetables.

b) Midday-chira or muri.

c) Lunch-rice, vegetables, fish or meat or egg.

d) Evening meal- chira or muri or fresh fruit like a banana, 2 slice of apple etc. (fruit should not take whose blood glucose is uncontrolled).

e) Dinner-ruti or rice, vegetables, fish or meat or egg.

f) Bed time snacks-very important who take insulin to prevent nocturnal hypoglycaemia.

Key message regarding lifestyle and dietary modifications: wgwó Lvevi, wgwó dj, wgwó we¯‹zU, †h †Kvb wgwó wRwbm Lv‡eb bv|cÖwZw`b 30 wgwbU হাঁটবেন, Ggbfv‡e হাঁটবেন †hb kixi †_‡K Nvg †ei nq|

Generic name	Mechanism of action	Dose	Side effects/disadvantage
Glimiperide	Increase insulin secretion	1-4 mg tablet, available as 1mg and 2 mg tablet. ১ টি বড়ি সকালে নাস্তার ৩০ মিনিট আগে খাবেন চলবে।	Sulfur allergy, Weight gain, hypoglycaemia
Gliclazide	Increase insulin secretion	30-320 mg, available as 30 mg, 60 mg and 80 mg tablet. ১ টি বড়ি সকালে নাস্তার ৩০ মিনিট আগে খাবেন চলবে।	Sulfur allergy, Weight gain, hypoglycaemia

Metformin	Increase insulin sensitivity	up to 2500 mg/day, available as 500 mg, 850 mg and 1gram tablet	Abdominal bloating, large volume stool, can not use if S. creatinine >1.5 mg/dl
Dipeptidyl peptidase-4 (DPP-4) inhibitors- vildagliptin, sitagliptin, linagliptin	Glucose-dependent insulin secretion, slowed gastric emptying, and reduction of postprandial glucagon	Vildagliptin 50-100 mg/day, linagliptin 5 mg/day	Costly drug not suitable for poor patient

Newer OAD

1. Incretin based therapies- DPP-4 inhibitors. Incretin are glucagon-like peptide 1 (GLP-1) and gastric inhibitory polypeptide (GIP), which act to potentiate insulin secretion. These are rapidly broken down by dipeptidyl peptidase 4 (DPP-4). DPP-4 inhibitors inhibit breakdown of GLP-1 and GIP thus sustained their action. These are vildagliptin, linagliptin- can be used in both lean thin and obese patient. But relatively high cost than other OAD.

2. The GLP-1 receptor agonists have a similar structure to GLP-1 but have been modified to resist breakdown by DPP-4. These agents are not orally active and have to be given by subcutaneous injection.

Currently available GLP-1 receptor agonists include exenatide (twice daily), exenatide modified-release (once weekly), liraglutide (once daily), lixisenatide (once daily) and albiglutide (once weekly). Recently, GLP-1 receptor agonists and long-acting insulin analogue have been combined, enabling co-administration of insulin and GLP-1 receptor agonists with one injection.

3. SGLT2 inhibitors: The sodium and glucose transporter 2 (SGLT2) inhibitors are dapagliflozin, canagliflozin and empagliflozin. Glucose is filtered freely in the renal

glomeruli and reabsorbed in the proximal tubules. SGLT2 is involved in reabsorption of glucose. Inhibition results in approximately 25% of the filtered glucose not being reabsorbed, with consequent glycosuria. Although this helps to lower blood glucose and results in calorie loss and subsequent weight loss, the glycosuria does also lead to genital fungal infections.

Insulin

Generic name	Dose	Frequency of administration	Side effects/disadvantage
Short acting insulin/regular insulin	Starting dose 0.1-0.5 unit/kg body weight/day	Three times daily	Weight gain, hypoglycemia
Mixed insulin (30/70-30% short acting, 70% intermediate acting insulin; also available as 50/50, 70/30.	Starting dose 0.1-0.5 unit/kg body weight/day	Two times daily	Weight gain, hypoglycemia
Long acting insulin	Starting dose 0.2 unit/kg body weight/ day	One time daily	

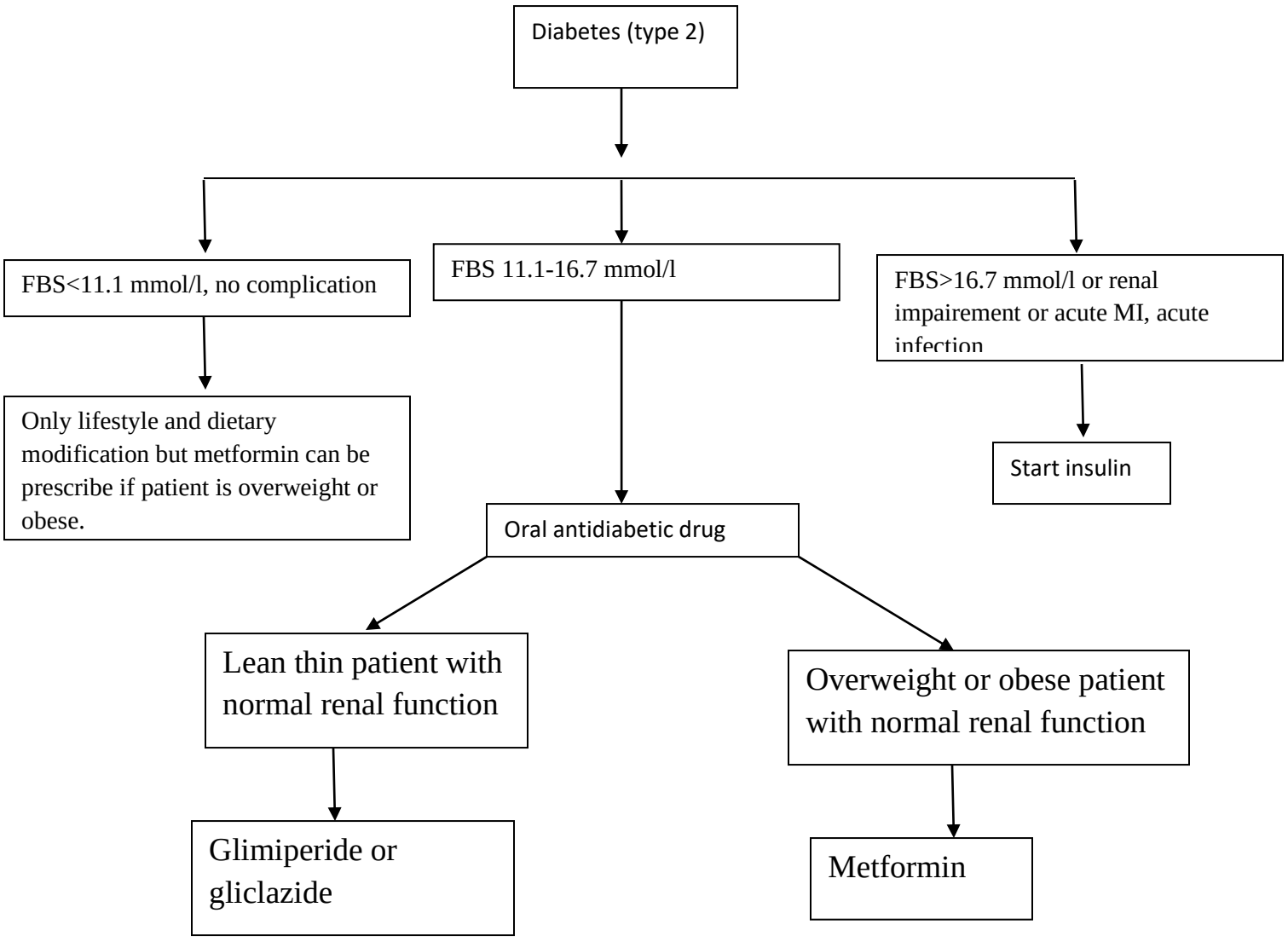
When to choice lifestyle and dietary modification or drugs or insulin?

Treatment option	FBS
Lifestyle and dietary modification	<11.1 mmol/l, no complications
Drugs	11.1-16.7 mmol/l, no complications
Insulin	>16.7 mmol/l or DM with complication

How you will choice antidiabetic drug?

Generic name	When to choice
Glimiperide	Lean thin patient with normal renal function
Gliclazide	Lean thin patient with normal renal function
Metformin	Overweight or obese patient with normal renal function

Following algorithm can be easy to memorize



Target blood glucose in DM

1. Fasting glucose levels of 5–7 mmol/L (90–126 mg/dL)

2. Pre-meal values of 4–7 mmol/L (72–126 mg/dL) and

3. 2 hour post-meal values of 4–8 mmol/L (72–144 mg/dL)

represent optimal control.

Follow up of DM patient after starting treatment

1. Getting oral treatment-follow up after 2-4 weeks (usually after 4 weeks) with FBS, 2 ABF.

2. Getting insulin-follow up after 7 days with FBS, 2 ABF.

3. After control of DM-follow up 3 monthly with FBS, 2 ABF and HbA1c.

4. Cheeck serum creatinine, ECG, fasting lipid profile, urine R/E at least once in a year.

Dc‡`k (Wvqv‡ewUm †ivMxi Rb¨)

1) Wv³v‡ii civgk© Qvov Wvqv‡ewU‡mi Ilya eÜ Ki‡eb bv|

2) wgwó RvZxq Lvevi Lv‡eb bv|

3) cÖwZw`b 30 wgwbU nvU‡eb|

4) wba©vwiZ mg‡q Ilya I Lvevi Lv‡eb |

5) Lvevi c~‡e© i‡³i গ্লুকোজ 6 mmol/L-Gi Kg I Lvevi ci 8 mmol/L-Gi Kg n‡j Wvqv‡ewUm wbqš¿‡Y Av‡Q|

6) eQ‡i 1 evi †PvL cix¶v Ki‡eb|

7) cv‡qi hZœ wb‡eb| cv‡q †Kvb Nv n‡j `ªæZ wPwKrm‡Ki civgk© wb‡eb |

8) nvB †cÖmvi _vK‡j wbqš¿‡Y ivL‡eb |

9) i‡³i Møy‡KvR K‡g †M‡j wb‡gœi j¶b¸‡jv n‡Z cv‡i- ¶zav jvMv, gv_v e¨_v, eyK aici, †ekx Nvg nIqv, GgbwK AÁvb n‡q †h‡Z cv‡ib| Ggb j¶b †`Lv w`‡j nv‡Zi Kv‡Q hv cv‡eb `ªZ Lv‡eb| Ggb Ae¯’vq wgwó RvZxq LveviI LvIqv hv‡e| j¶b¸‡jv K‡g †M‡j `ªZ wPwKrm‡Ki civgk© wb‡eb |

Gestational Diabetes

Screening for gestational diabetes

All women at high risk should have an oral glucose tolerance test at 24–28 weeks.

Measurement of HbA1c and/or blood glucose at booking visit is usually recommended.

Risk factors for gestational diabetes

1. Body mass index > 30 kg/m2

2. Previous macrosomic baby weighing ≥ 4.5 kg

3. Previous gestational diabetes

4. Family history of diabetes (first degree relative with diabetes)

5. Family origin with a high prevalence of diabetes:

a) South Asian (specifically women whose country of family origin is India, Pakistan or Bangladesh)

b) Black Caribbean

Management of gestational diabetes: Reduce intake of refined carbohydrate, and add metformin, glibenclamide and/or insulin if necessary to optimise glycaemic control.

Targets are

-Pre-prandial level of < 5.3 mmol/L (95 mg/dL) and

-1 hour or 2 hour post prandial level of < 7.8 mmol/L (140 mg/dL) and < 6.4 mmol/L (114 mg/dL), respectively.

Follow up

Fasting blood glucose measured at 6 weeks post-partum and have HbA1c concentrations measured annually to screen for the development of diabetes.

DM and Surgery

Pre-operative assessment

1. Assess glycaemic control: consider delaying surgery and refer to the diabetes team if HbA1c > 75 mmol/mol (9%); this should be weighed against the need for surgery.

2. Assess cardiovascular status

-Optimize blood pressure

-Perform an ECG for evidence of (possibly silent) ischaemic heart disease and to assess QTc

3. Assess foot risk

-Patients with high-risk feet should have suitable pressure relief provided during post operative nursing.

4. For minor/moderate operations where only one meal will be omitted, plan for the patient to be first on the list.

Perioperative management

Major operation/prolonged fasting

Or

Minor operation with HbA1C >64mmol/mmol

↓

Omit any oral antidiabetic drugs and GLP-1 analogues on morning of operation
Omit quick- or medium-acting insulin prior to miss meal.
Continue long-acting insulin (e.g. glargine or detemir)

↓

Start IV insulin infusion and IV fluids early on morning of operation and
Maintain on IV insulin until eating and drinking

↓

Check urea & electrolytes at least daily while on IV insulin and fluids
Ensure adequate potassium replacement and
Avoid hyponatraemia from dextrose infusion

↓

Once patient is eating, prescribe usual oral or injectable treatment with a meal and
Discontinue insulin infusion 1 hr later
Withhold metformin if eGFR < 30 mL/min/1.73 m2.

Minor operation/only one meal omitted

↓

HbA1C ≤64mmol/mmol

↓

Aim for first in OT list

↓

Omit any oral antidiabetic drugs and GLP-1 analogues on morning of operation
Omit quick- or medium-acting insulin prior to miss meal.
Continue long-acting insulin (e.g. glargine or detemir)
But consider reducing dose if surgical list is in afternoon

↓

No need for IV insulin unless unable to eat post-operatively, blood glucose > 14 mmol/L (250 mg/dL), or ketones present in urine or blood

↓

Resume usual medication with first meal; if it is lunch for patient using mixed insulin, give half of usual morning dose.

Post-operative management

Patients who need to continue fasting after surgery should be maintained on intravenous insulin and fluids until they are able to eat and drink. During this time, care must be taken with fluid balance and electrolyte levels. Intravenous fluids during prolonged insulin infusion should therefore include saline and potassium supplementation.

Hypoglycemia

Diagnostic tools

1. History of diabetes mellitus.

2. History of treatment with insulin or secretogogues.

3. History of missed or delayed meal, more exercise than usual.

4. Patient complaint-headache, dizziness, profuse sweating, palpitation, slurring of speech, confusion, drowsiness, loss of consciousness.

5. On examination- bilateral planter extensor may be present.

6. Urgent blood glucose (by glucometer or laboratory)- < 3.9 mmol/L (70 mg/dL)

(Due to chronic hyperglycaemia DM patient may experience hypoglycaemic symptoms in higher blood glucose level than non-diabetic).

Management

Mild to moderate hypoglycaemia (patient is conscious and able to take oral food)

Most cases are diagnosed and treated by the patient himself or by a family member.

Management

Mild to moderate hypoglycaemia (patient is conscious and able to take oral food)

Most cases are diagnosed and treated by the patient himself or by a family member.

1. Stop oral antidiabetic drug and insulin.

2. 15 gm glucose drink (or equivalent food) by mouth e.g. a glass of soft drink or fruit juice or meal (if it is due).

3. Repeat capillary glucose measurement 1–15 mins later. If still < 4.0 mmol/L, repeat above treatment.

4. If blood glucose remains < 4.0 mmol/L after three cycles (30–45 mins), contact a doctor. Consider glucagon 1 mg IM or 150–200 mL 10% glucose over 15 mins IV.

5. If cause of hypoglycaemia not found, the patient should reduce the next dose of insulin by 10–20%.

Severe hypoglycaemia

Diagnostic tool

1. History of treatment with insulin or secretogogues.

2. Only neuroglycopenic symptoms like drowsiness, convulsion or coma.

Management

1. For type 1 DM inj. glucagon 1mg IM stat, if recovery is satisfactory then consider another shot.

2. IV 150–200 mL 10% dextrose over 15 minutes. Repeat blood glucose measurement after 10–15 minutes and manage as per biochemical hypoglycaemia.

3. Identification & correction of the underlying cause of hypoglycaemia (delayed or missed meal, taking high amount insulin etc).

4. If cause of hypoglycaemia not found, the patient should reduce the next dose of insulin by 10–20%.

(When patient develop hypoglycemia without significant causes then search whether there is any nephropathy or hepatic impairment, because in hepatic or renal impairment metabolism of antidiabetic drug or insulin decrease. So the drug or insulin acts longer than usual, so hypoglycemia occurs).

Diabetic ketoacidosis

Diagnostic tools

1. History of DM (usually occurs in type 1 DM but can also occur in type 2 DM patient).

2. History of irregular treatment or stopped drug or insulin or missed dose of insulin or drugs.

3. Stressful condition e.g. infection, trauma, pregnancy.

4. On examination - severe dehydration, pulse-tachycardia, BP- hypotension, acidotic breathing, there may be acetone smell in breath, patient may be unconsciousness.

5. Biochemical features-hyperketonaemia (≥ 3.0 mmol/L) or ketonuria (more than 2+ on standard urine sticks), blood glucose≥11mmol/L, metabolic acidosis (venous bicarbonate < 15 mmol/L and/or venous pH < 7.3 (H+ > 50 nmol/L).

6. Investigation to screen infection-full blood count, blood and urine culture, C-reactive protein, chest X-ray.

Management

1. Establish IV access, assess patient and perform initial investigations.

2. Fluid management

a) Commence 0.9% sodium chloride:

-If systolic BP > 90 mmHg, give 1 L over 60 minutes

-If systolic BP < 90 mmHg, give 500 mL over 10–15 minutes, then re-assess; if BP remains < 90 mmHg, repeat and seek senior review.

b) IV infusion of 0.9% sodium chloride with potassium chloride added as indicated below:

-1 L over 2 hrs

-1 L over 2 hrs

-1 L over 4 hrs

-1 L over 4 hrs

-1 L over 6 hrs

c) Add 10% glucose 125 mL/hr IV when glucose < 14 mmol/L (252 mg/dL)

3. Insulin treatment: commence insulin treatment: 50 U human soluble insulin in 50 mL 0.9% sodium chloride infused intravenously at 0.1 U/kg body weight/hr. Continue with SC basal insulin analogue if usually taken by patient.

4. Potassium replacement

-If plasma potassium > 5.5 mmol/L do not give potassium.

-If plasma potassium 3.5-5.5 mmol/L, give 40 mmol/L of fluid infusion.

-If plasma potassium < 3.5mmol/L, senior review, additional potassium require.

5. Establish monitoring schedule:

a) Hourly capillary blood glucose and ketone testing

b) Venous bicarbonate and potassium after 1 and 2 hours, then every 2 hours for first 6 hours

c) Plasma electrolytes every 4 hours

d) O2 saturation, pulse, BP, respiratory rate and urine output every hour

6. Treat any precipitating cause like treatment of infection.

7. Re-initiate SC insulin: if ketoacidosis has resolved and patient is able to eat and drink, do not discontinue IV insulin until 30 minutes after SC short-acting insulin injection.

HONK (hyperosmolar non-ketotic diabetic coma)

Diagnostic tools

1. History of DM (usually occurs in type 2 DM patient).

2. History of irregular treatment or stopped drug or insulin or missed dose of insulin or drugs.

3. Stressful condition e.g. infection, trauma, pregnancy.

4. On examination - severe dehydration, pulse-tachycardia, BP- hypotension, unconsciousness.

5. Investigation- Blood sugar- > 30 mmol/L

a) No keton body in urine

b) Hyperosmolality (> 320 mOsm/ kg).

Hyperosmolality can be calculated by

= 2[Na+] + 2[K+] + [glucose] + urea (all in mmol)

{ Normal osmolality is 280-300 mOsm/kg}

Management:

Same as DK but less insulin is required.

N:B: when a DM patient become unconsciousness primary task of the physician is to confirm the diagnosis whether this is hypoglycaemia or DK or HONK. This can be confirmed only after having blood glucose test. But some of the clinical features can differentiate them.

Points	Hypoglycemia	DK	HONK
History of excessive	Present	Absent	Absent

sweating, palpitation			
History of missed or delayed meal or taking extra insulin or drugs	Present	Absent	Absent
History of irregular treatment or stopped drug or insulin or missed dose of insulin or drugs	Absent	Present	Present
Dehydration	Absent	Present	Present
Acetone smell in breath	Absent	Present	Absent

Cushing syndrome

Cushing syndrome occurs due to excessive secretion of glucocorticosteroids. This can be ACTH dependent (pituitary adenoma-cushing disease, ectopic ACTH secretion, ACTH therapy) and ACTH non-dependent (itrogenic- chronic glucocorticoid therapy, adrenal adenoma, adrenal carcinoma). In practice common cause of Cushing syndrome is itrogenic (excess intake of prednisolone, dexamethasone etc).

Diagnostic tools

1. History of taking prednisolone, dexamethasone etc for long duration.

2. Patient usually obese (moon face, abdominal obesity with striae, buffalo neck).

3. Skin-thin, echymoses, bruise may be present.

4. Signs of infection may be present (fever, cough, dysuria) due to immunocompression.

5. Patient may have features of hypertension and DM.

6. For confirmation-dexamethasone suppression test. (Usually not done to diagnose itrogenic cushing syndrome).

Management

Cushing disease

1. Medical treatment before surgery-metyrapone and ketokonazole- the dose should be titrated against 24 hour free urine cortisol level.

2. Surgery- transphenoidal surgery, if unsuccessful then bilateral adrenalectomy. Nelson syndrome can be prevented by pituitary irradiation.

Adrenal tumour-laparoscopic surgery.

Itrogenic cushing syndrome

1. Gradual withdrawal of steroid (sudden withdrawl may precipitate adrenal insufficiency).

2. Treatment of con-current infections.

3. Control of hypertension and DM (if any).

4. Calcium supplementation (to prevent osteoporosis).

A simple, practical steriod withdrawal protocol

- Tab. Prednisolone 20 mg ২ টি বড়ি সকালে খাবেন ভরা পেটে ৪৫ দিন।এরপর
- Tab. Prednisolone 20 mg ১ &১/২ বড়ি সকালে খাবেন ভরা পেটে ৭ দিন।এরপর
- Tab. Prednisolone 20 mg ১ বড়ি সকালে খাবেন ভরা পেটে ৭ দিন।এরপর
- Tab. Prednisolone 10 mg ১ &১/২ বড়ি সকালে খাবেন ভরা পেটে ৭ দিন।এরপর
- Tab. Prednisolone 10 mg ১ বড়ি সকালে খাবেন ভরা পেটে ৭ দিন এরপর
- Tab. Prednisolone 5 mg ১ & ১/২ বড়ি সকালে খাবেন ভরা পেটে ১৪ দিন।
- Tab. Prednisolone 5 mg ১ বড়ি সকালে খাবেন ভরা পেটে ১৪ দিন।
- Tab. Prednisolone 5 mg ১/২ বড়ি সকালে খাবেন ভরা পেটে ১৪ দিন।

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2. Patient may complaint nausea, vomiting, vertigo and weight loss.

3. On examination-blood pressure usually low, postural hypotension present, pigmentation present in the oral cavity, palmar creases, knuckles etc.

4. S. electrolytes-hyponatraemia, blood glucose-usually low.

5. Confirm by ACTH stimulation test.

Management

1. Diet- normal

2. Hydrocortisone 15 mg at morning & 5 mg at 3 pm (lifelong).

3. Fludrocortisone 0.05-0.1 mg daily.

4. Treatment of the underlying cause e.g. antitubecular drugs, treatment of haemocromatosis etc.

Follow up

1. Pulse

2. BP

3. S. electrolytes

Advice:

1. If fever- double the dose of the steriod.

2. In vomiting- injection hydrocortisone should be given (if patient cannot swallow tablet).

3. Minor surgery- Inj. hydrocortisone 100 mg IM with premedication.

4. Major surgery- Inj. hydrocortisone 100 mg IM 6 hourly until patient able to take oral tablet.

5. Patient should carry steroid card.

6. Bracelet-Patients should be encouraged to buy a bracelet and have it engraved with the diagnosis, current treatment and a reference number for a central database.

Adrenal crisis

Diagnostic tools

1. Diagnosed case of Addison's disease.

2. Sudden loss of consciousness.

3. Severe hypotension, hyponatraemia, hyperkalaemia and in some instances, hypoglycaemia and hypercalcaemia.

4. The crisis is often precipitated by intercurrent disease, surgery or infection.

Management

1. NPO TFO

2. O2 inhalation SOS

3. Infusion normal saline to normalize blood pressure.

4. For hypoglycaemia-infusion 10 % DA 1000cc IV @ 20 drops/minute.

5. Inj. hydrocortisone 100 mg IV stat, and 100 mg 4 times daily for first 12–24 hours. Continue parenteral hydrocortisone (50–100 mg IM 4 times daily) until patient is well enough for reliable oral therapy

6. Hyperkalaemia: should respond to volume replacement but occasionally requires specific therapy.

7. Treatment of the underlying cause e.g. antibiotic for infection.

Phaeochromocytoma

Management

1. Definitive treatment is surgery.

2. Preparation of patient before surgery minimum for 6 week.

3. Control of hypertension by alpha blocker phenoxybenzamine 10-20 mg orally 6-8 hourly. If tachycardia occurs add betablocker -propanolol or labetalol.

4. During surgery- sodium nitroprusside & short acting alpha antagonist phentolamine are used to control hypertension.

5. Post operative period hypotension should be managed by -fluid & noradrenaline infusion.

Hypopituitarism

Management

1. Glucocorticoid replacement (prednisolone).

2. Thyroxine 100-150 µgm daily (before this steroid must be given).

3. Sex hormone in men of any age & in female <50 years.

4. GH in children & adolescent S/C injection.

5. Treatment of the underlying cause.

Prolactinoma

Management

1. Dopamine agonist (bromocryptine 1st line drug)

2. Surgery-2nd line treatment.

Acromegaly

Management

1. Surgery-1st line treatment.

2. Radiotherapy-second-line treatment if acromegaly persists after surgery.

3. Medical treatment-if acromegaly persists after surgery

    a) Somatostatin analogue (cctreotide, lanreotide) slow releasing inj. every few weoks interval.

    b) Bromocryptine-if associated with prolactin excess.

    c) GH receptor antagonist Pegvisomant indicated in whom GH & IGF1 fail to suppress sufficiently by Somatostatin analogue.

Diabetes insipidus

Management

Cranial DI

1. Desmopressin nasal spray 5 µgm in morning & 10 µgm at night. In sick patient IM injection.

Nephrogenic DI

1. Bendroflumethazide 5-10 mg/day

2. Amiloride 5-10 mg/day

3. Indomethacin 15 mg 8 hourly.

Amenorrhoea (for details see obstetric chapter)

Management

2. Exclude physiological cause e.g. pregnancy, menopause etc.

3. Decrease exercise, work load.

4. Weight gaining if under weight.