BELMATT

HEALTHCARE TRAINING

Introduction to Blood Results in General Practice

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Session Aims and Objectives

Time	Topic
09:00- 09:10	Introduction
09:10- 09:50	FBC, haemoglobinopathies, inflammatory markers (ESR, CRP)
09:50- 10:00	Q&As/Break
10:00- 10:40	U&Es and TFTs
10:40- 10:50	Q&As/Break
11:00- 11:50	LFTs
11:50- 12:00	Q&As/Break

How would you deal with this?

Report Type : Pathology

! Full blood count - FBC

Status : Not Filed

Specimen : BLOOD Taken : 08-Dec-2023 19:09 Received : 08-Dec-2023 19:09

!	**Full blood count - FBC**		
!	Total white cell count	11.2 10*9/L	(4.2 - 10.6)
!	Red blood cell (RBC) count	5.98 10*12/L	(4.23 - 5.46)
	Haemoglobin estimation	162 g/L	(130 - 168)
	Haematocrit	0.489 L/L	(0.390 - 0.500)
!	Mean corpuscular volume (MCV)	81.8 fL	(83.5 - 99.5)
!	Mean corpusc. haemoglobin(MCH)	27.1 pg	(27.5 - 33.1)
	Mean corpusc. Hb. conc. (MCHC)	332 g/L	(315 - 350)
	Red blood cell distribut width	14.4 %	(10.0 - 16.0)
	Platelet count	317 10*9/L	(130 - 370)
	Mean platelet volume	9.6 fL	(8.0 - 12.0)
	Nucleated red blood cell count	0.0 10*9/L	(0.0 - 0.1)
	Neutrophil count	6.4 10*9/L	(2.0 - 7.1)
	Lymphocyte count	3.4 10*9/L	(1.1 - 3.6)
!	Monocyte count	1.0 10*9/L	(0.3 - 0.9)
	Eosinophil count	0.3 10*9/L	(0.0 - 0.5)
	Basophil count	0.1 10*9/L	(0.0 - 0.2)

SOB

Red Cells

Haemoglobin (Hb): amount of haemoglobin (oxygen-carrying protein) in whole blood

Haematocrit (Hct): percentage of the blood sample that is made up of red cells

Mean corpuscular volume (MCV): the average size of the red cells present in the blood sample

Red cell distribution width (RDW): a range from the largest red cell present to the smallest red cell present

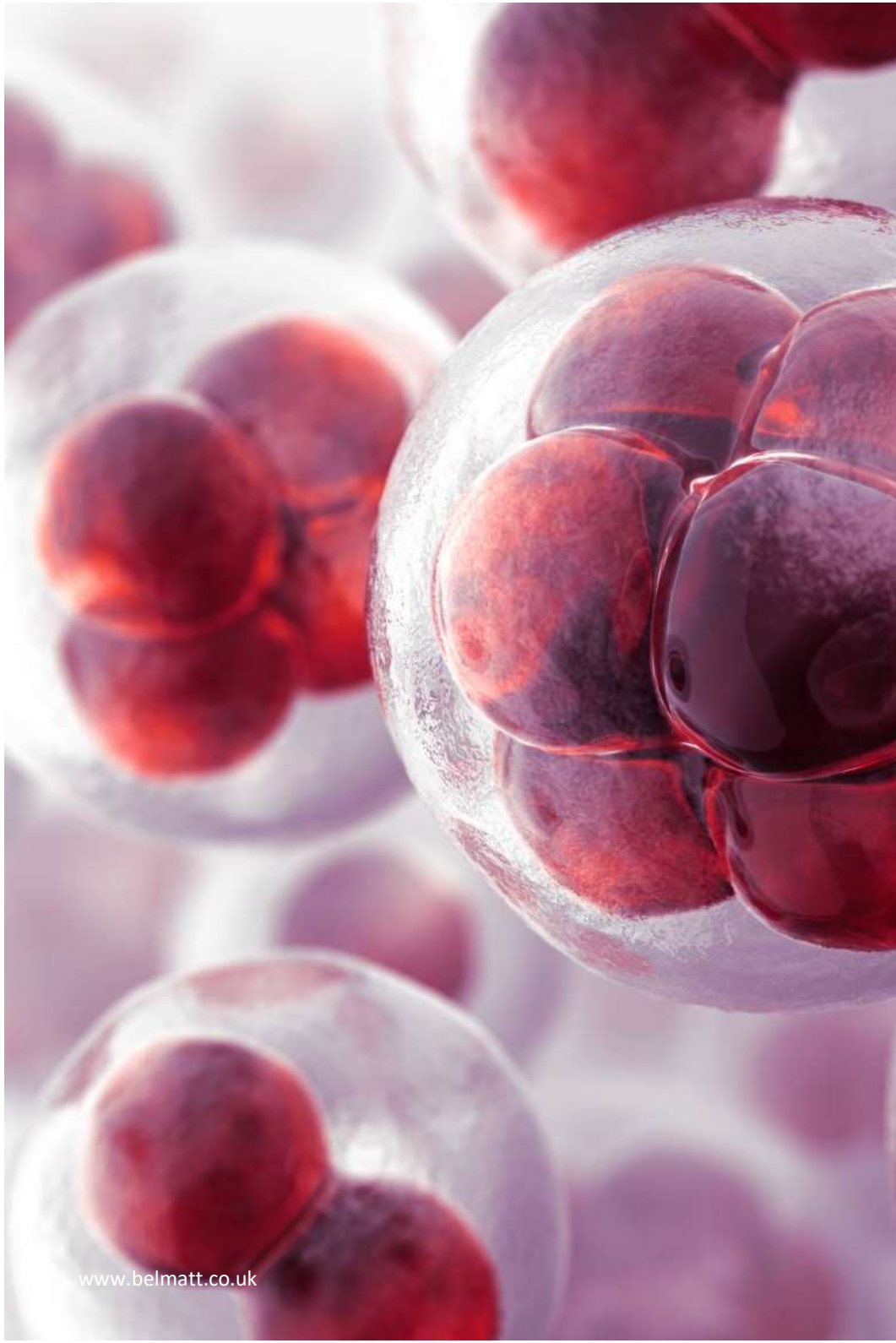
Red cell count (RCC): the number of red cells present per unit volume of blood

Reticulocyte count: the number of reticulocytes (immature red cells)

- Mean corpuscular haemoglobin (MCH): the amount of haemoglobin per red blood cell
- Mean corpuscular haemoglobin concentrate (MCHC): average concentration of haemoglobin in a given volume of blood

Red cell count (RCC):

- ♂ 4.5 – 6.5 x 10^9/L
- ♀ 3.8 – 5.8 x 10^9/L



White Cells

- **White blood cell count** (WCC): the number of white blood cells
- **White blood cell differential**: the breakdown of the white blood cell count into different cells

White cell count (WCC):

- Total: 3.6 – 11.0 x 10^9/L
- Neutrophils: 1.8 – 7.5 x 10^9/L
- Lymphocytes: 1.0 – 4.0 x 10^9/L
- Monocytes: 0.2 – 0.8 x 10^9/L
- Eosinophils: 0.1 – 0.4 x 10^9/L
- Basophils: 0.02 – 0.10 x 10^9/L

- **Platelet tests**

Other Ranges

- **Haemoglobin (Hb)**:
- ♂ 130 – 180 g/L
- ♀ 115 – 165 g/L
- **Platelet count**: 140 – 400 $x10^9$/L
- **Haematocrit**:
- ♂ 0.40 – 0.54 L/L
- ♀ 0.37 – 0.47 L/L
- **Mean cell volume (MCV)**: 80 – 100 fL
- **Mean corpuscular haemoglobin (MCH)**: 27 – 32 pg/cell
- **Reticulocyte count**: 0.2 – 2%

Eosinophilia

- Relatively common
- Allergic responses (eczema, asthma)
- **Drug reactions**
- Skin disorders
- Autoimmune conditions
- Parasites
- Hodgkin and bone marrow disorders

Anaemia

- Anaemia refers to a decrease in the total amount of haemoglobin in the blood.
- There are a wide range of causes of anaemia, which can be sub-categorised based on the average red cell size (mean cell volume/MCV):
- Microcytic anaemia: low haemoglobin associated with a reduced MCV
- Macrocytic anaemia: low haemoglobin associated with an increased MCV
- Normocytic anaemia: low haemoglobin associated with a normal MCV
- When you identify a low haemoglobin, you should look to the MCV to see which sub-type of anaemia is present as this information, alongside a good clinical assessment, can help narrow the differential diagnosis.
- In general, when you identify anaemia it is sensible to check haematinics (e.g. ferritin, B12/folate) as deficiencies are common and easy to treat.

Microcytic Anaemia

- Microcytic anaemia is defined as anaemia with an MCV of **less than 80**. As there is a lack of haemoglobin (Hb), an extra division of red blood cells (RBCs) occurs to maintain adequate Hb concentration.[10] This results in **smaller** and **paler** (hypochromic) RBCs.
- The most common cause of microcytic anaemia is iron-deficiency anaemia.

Causes of Microcytic Anaemia

- Iron deficiency anaemia: the commonest cause in the UK (see our article on iron deficiency anaemia)
- Haemoglobinopathies (e.g. thalassemia syndrome/trait)
- Anaemia of chronic disease/inflammation (can be microcytic, but often normocytic)
- Lead poisoning (rare)
- Sideroblastic anaemia (rare)
- Hookworm infection (a common cause of microcytic anaemia in low/middle-income countries)

Iron Deficiency Anaemia

Aetiology

- In iron-deficiency anaemia, there is a lack of iron, which is a vital component of haemoglobin. This condition often presents with mild symptoms (e.g. fatigue, palpitations) and can sometimes lead to strange cravings for ice, dirt, or starch
- Iron deficiency is most often caused by chronic blood loss but can also be due to dietary deficiency, malabsorption or increased requirements during childhood or pregnancy.[11]
- Any patient over 40 with iron deficiency anaemia requires an upper and lower gastrointestinal endoscopy.

Investigation findings

- Investigation findings in **iron deficiency anaemia** include:
- Low MCV <80
- Serum iron: low
- Transferrin saturation: low
- Ferritin: low
- Total iron-binding capacity: high (there is an inverse relationship between ferritin and TIBC

Thalassaemia

- Thalassaemias occur due to inherited mutations affecting the **globin gene**. They can be further classified into the **specific gene** that is affected (alpha or beta), as well as the **severity** of the condition (major, minor or trait).
- People with the thalassaemia trait may be asymptomatic or have very mild symptoms, while **thalassaemia major** can be severe enough to require regular transfusions.
- Most frequent in the Mediterranean, Africa, Western and Southeast Asia, as well as India and Burma.
- This condition seems to be protective against *Plasmodium falciparum* malaria, which is why the population distribution is so similar for the two conditions.
- Thalassaemias result in classic clinical features such as chipmunk facies and a crew cut appearance on X-ray.

Investigation findings

- Low MCV <80 (usually lower relative to iron deficiency anaemia)
- Ferritin: normal (unlike the low ferritin found in iron deficiency anaemia)
- Haemoglobin electrophoresis: abnormal
- Positive genetic testing for HBB, HBA1 or HBA2

Normocytic Anaemias

- This subset of anaemias is identified by a **normal MCV** of between 80-100.
- Since the size of the RBCs isn't altered, the decreased haemoglobin must be due to another cause, either **haemolysis** (intravascular or extravascular) or **underproduction** of normal-sized RBCs.
- The **reticulocyte count** enables differentiation between the two causes.
- A high reticulocyte count would indicate that the bone marrow was functioning normally and therefore the anaemia is not likely to be due to underproduction.
- Unlike microcytic anaemias, normocytic anaemias are usually **normochromic**.

Causes of normocytic anaemia

- Anaemia of chronic disease/inflammation
- Acute blood loss
- Increase plasma volume (e.g. pregnancy, fluid overload)
- Mixed aetiology anaemias
- Haemoglobinopathies (e.g. thalassaemias)
- Aplastic anaemia
- Haemolysis
- Hypersplenism (leads to increased destruction of red blood cells)

Anaemia of Chronic Disease

- In anaemia of chronic disease, **underlying chronic diseases** such as malignancy, chronic infections, or autoimmune conditions, cause the liver to produce acute phase reactants such as hepcidin.
- Hepcidin "hides" the iron in ferritin to reduce availability in the serum. Anaemia of chronic disease starts out as **normocytic anaemia** but it can progress to microcytic anaemia.

Investigation findings

- Investigation findings in **anaemia of chronic disease** include:
- Normal MCV (80-100) or low MCV (<80)
- Serum iron: low
- Transferrin saturation: low
- Ferritin: high
- TIBC: low

Sickle Cell Anaemia

Aetiology

- This condition occurs due to an **autosomal recessive mutation** in the **beta-globin chain** of haemoglobin, causing valine to replace glutamic acid.
- Intravascular haemolysis may occur due to the deformed **sickle shape** of the RBC, but the spleen has a role in the more predominant **extravascular haemolysis** of the misshapen cells.
- For more information, see the Geeky Medics guide to sickle cell anaemia.

Investigation findings

- Investigation findings in **sickle cell anaemia** include:
- Reticulocyte count: increased due to compensation by the bone marrow for intravascular/extravascular haemolysis
- Serum uric acid: increased due to lysed RBCs
- Blood film: sickling of erythrocytes and features of hyposplenism including target cells and Howell-Jolly bodies

G6PD6

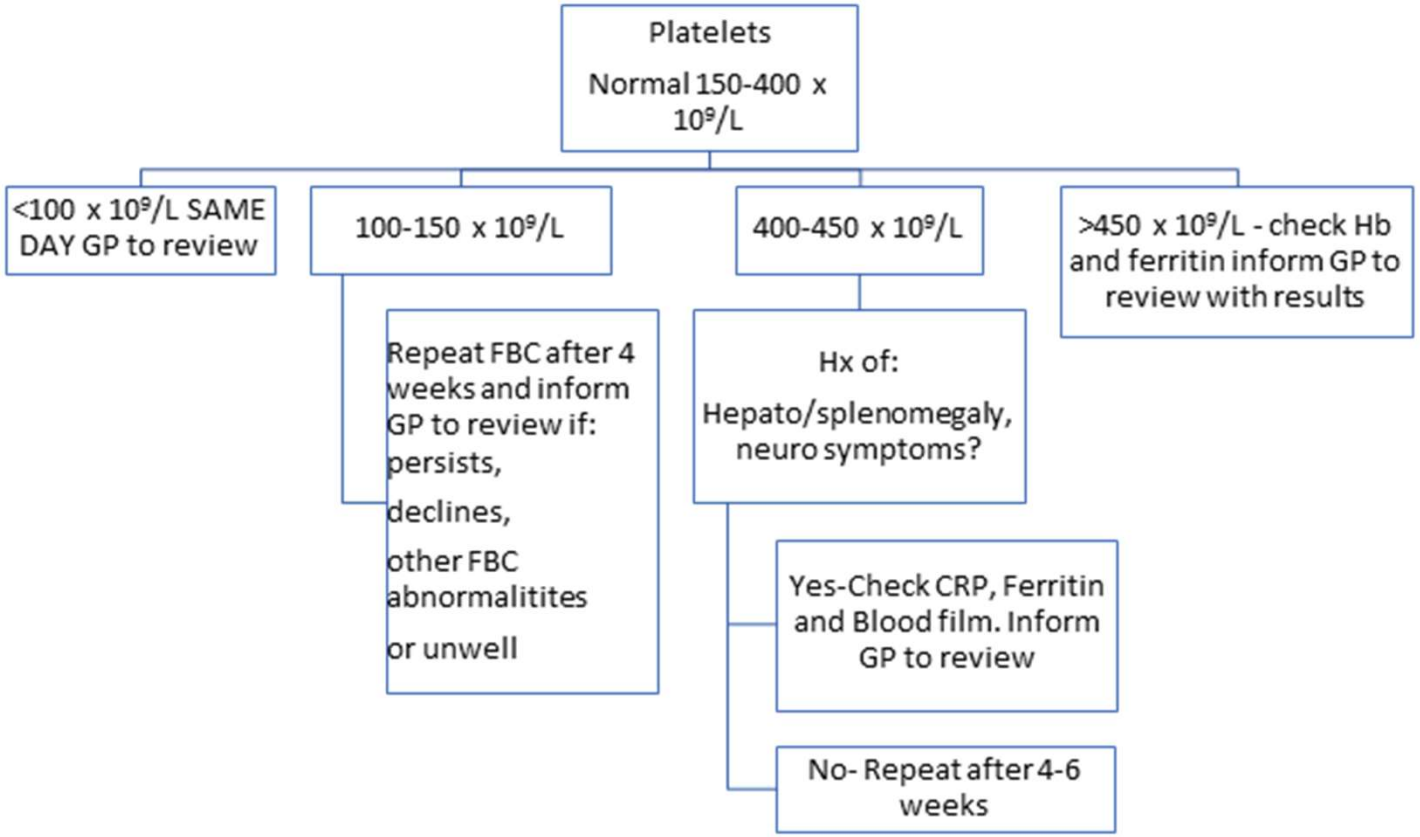
Aetiology

- A deficiency in glucose-6-phosphate dehydrogenase (G6PD) makes RBCs more susceptible to **oxidative stress**.
- Normally, G6PD creates nicotinamide adenine dinucleotide phosphate (NADPH), which in turn creates reduced glutathione to protect the cell from oxidative injury. Lack of G6PD results in **intravascular haemolysis** due to destruction via oxidative stress.

Investigation findings

- Investigation findings in **G6PD deficiency** include:
- Reticulocyte count: increased
- Serum uric acid: increased due to lysed RBCs

Platelets



Inflammatory markers

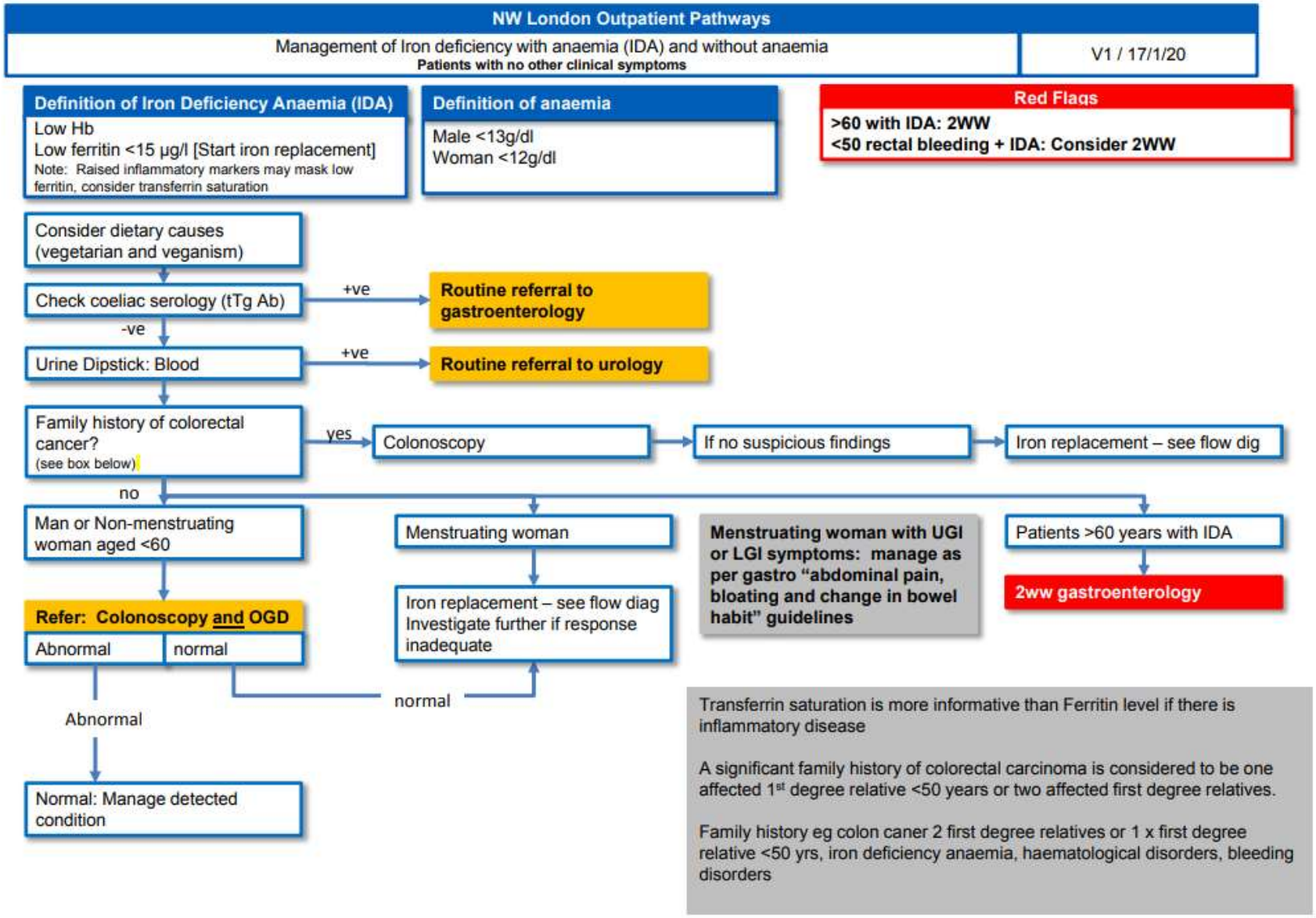
- CRP[1]
  - CRP is recommended over ESR to detect acute phase inflammation in patients with undiagnosed conditions because it is more sensitive and specific than ESR
  - CRP is also a useful measure because concentrations change rapidly within the first 6-8 hours after injury, peak after 48 hours, and return to normal levels once the issue has resolved
- ESR
  - Relates to the ability of red cells to form rouleaux. Normal ESR values are specific to age and sex; the rate increases steadily with age and is higher in women than in men
  - Calculation of predicted value[2]:
    - Men - ESR given by age in years divided by 2
    - Women - ESR given by age in (years+10) divided by 2
- Other: Complement factors, Ferritin, alpha 1-antitrypsin, vWF, prothrombin

1. Polepalle T, Moogala S, Boggarapu S, Pesala DS, Palagi FB.Acute Phase Proteins and Their Role in Periodontitis: A Review.J Clin of Diagn Res.2015; 9(11):ZE01-ZE05

2. *Br Med J (Clin Res Ed)* 1983;286:266

Overview

Ref:
https://www.nwlondonccg.nhs.uk/application/files/4715/9376/8471/iron_deficiency_anaemia_v1.pdf



Case Study 1

Emily is a 28-year-old female with a vegetarian diet. She complains of fatigue and is short of breath on exertion. Her Hb is 90 g/L {normal: 111-147} and Ferritin is 9 {normal: 10-120}. What would you do next?

Case Study 2

Linda is a 54-year-old female who has been having unexplained weight loss. She complains of fatigue and is short of breath on exertion. Her Hb is 80 g/L {normal: 111-147} and Ferritin is 9 {normal: 10-120}. What would you do next?

Case Study 3

Robert is a 63-year-old male who has rheumatoid arthritis. He complains of mild joint pain with general weakness. His Hb is 105 g/L {normal: 130-168} and ESR is 40. What would you do next?

U&Es

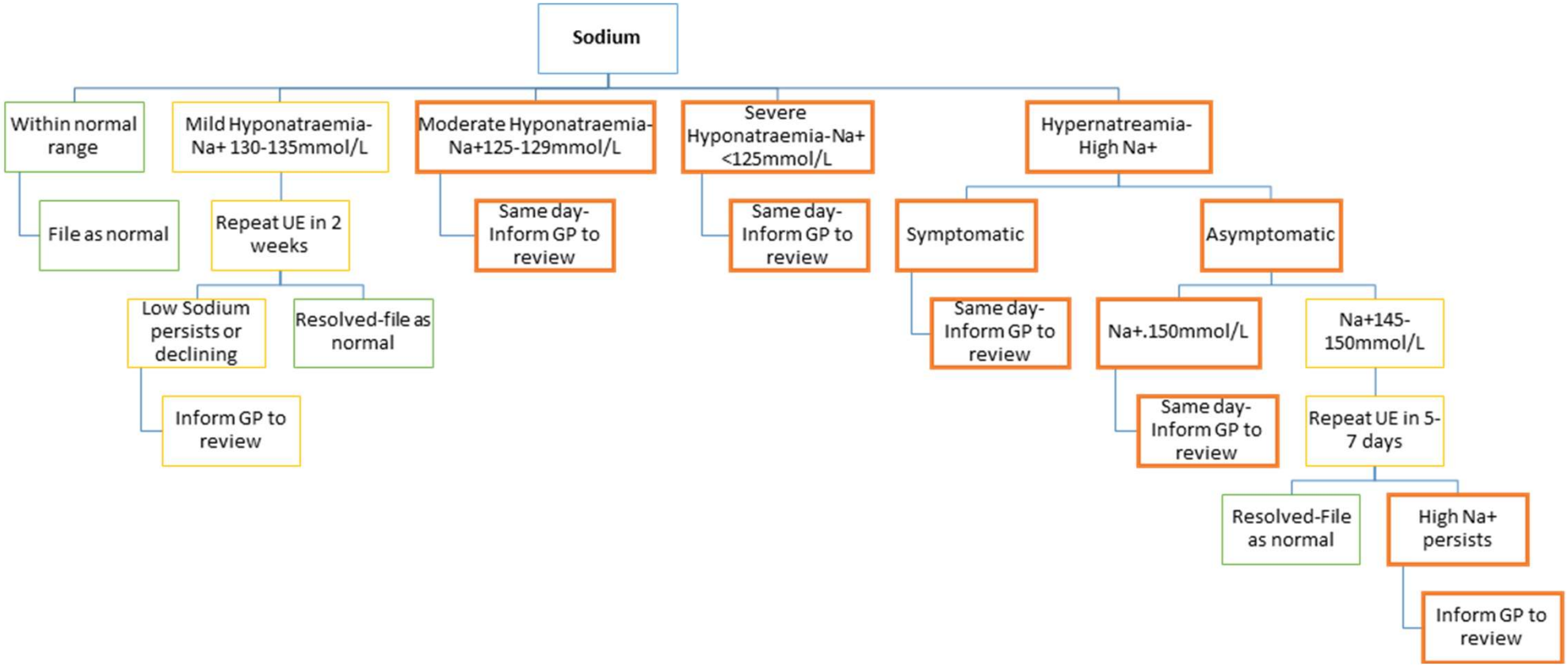
Test	Reference range
Serum Sodium	135-146 mmol/L
Serum Potassium	3.5-5.3 mmol/L
Urea	2.5-7.8 mmol/L
Serum Creatinine	55-110 µmol/L
eGFR	>90 ml/min

Overview

- U&Es are commonly ordered to assess **renal (kidney) function**. The kidney excretes **urea** and **creatinine**, and these can be used as **surrogate markers** of renal function. Sodium and potassium are included in the panel, as renal dysfunction can lead to **electrolyte derangements**.

- Other reasons to order U&Es include suspected **electrolyte disturbances** due to non-renal causes, **medication monitoring** (e.g. after starting ACE inhibitors), or assessing **urea levels** in the case of suspected upper gastrointestinal haemorrhage.

Overview for non-GPs filing results



Drug monitoring

- Common drugs which require **monitoring of U&Es** include:
- Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs)
- Diuretics: spironolactone, thiazide diuretics (e.g. indapamide), loop diuretics (e.g. furosemide)
- Direct-acting oral anticoagulants (DOACs)
- Carbamazepine
- Lithium
- Digoxin

Urea

- Urea is a waste product of protein breakdown produced in the liver. The kidneys **predominantly excrete urea**, and it can be used as a surrogate marker of renal function. However, this is fairly non-specific.
- Causes of a **raised serum urea** (uraemia) include:
  - **Renal dysfunction**: decreased excretion of urea into the urine.
  - **Dehydration**: urea rises quickly in dehydration, even in the presence of normally functioning kidneys. This is physiologically mediated by anti-diuretic hormone (ADH), released from the posterior pituitary gland in response to intravascular volume depletion. ADH increases urea and water reabsorption in the collecting ducts.
  - **Upper gastrointestinal bleeding**: blood in the upper GI tract is broken down into proteins, which are transported to the liver via the portal vein and metabolised into urea.
- Low serum urea levels are **non-pathological**, associated with pregnancy and those on a low-protein diet.

eGFR

- The eGFR is a **mathematically derived number** based on a patient's serum **creatinine** in conjunction with **age**, **sex** and **race**.[2]
- eGFR aims to estimate the **glomerular filtration rate** (GFR), which cannot be directly measured in humans. As the **serum creatinine rises**, the **eGFR will decrease**, indicating worsening kidney dysfunction.
- A 'normal' glomerular filtration rate is around 100ml/min/1.73m^3. When discussing results with patients, the eGFR can be roughly equated to a **percentage** of kidney function.
- This is particularly relevant in **chronic kidney disease (CKD)**, where the eGFR is used to 'stage' the disease:
- **Stage 1**: eGFR >90 (normal), with other tests showing signs of kidney damage (e.g. proteinuria)
- **Stage 2**: eGFR of 60 to 89 ml/min, with other tests showing signs of kidney damage (e.g. proteinuria)
- **Stage 3a**: eGFR of 45 to 59 ml/min
- **Stage 3b**: eGFR of 30 to 44 ml/min
- **Stage 4**: eGFR of 15 to 29 ml/min
- **Stage 5**: eGFR <15 ml/min

Limitations of eGFR

Both equations are well-validated. However, in some circumstances, the eGFR can **vary significantly** from the true glomerular filtration rate (e.g. patients with extremes of body type – malnourished, bodybuilders, severe obesity or limb amputees). The equations are also not valid for those under 18 years of age.

Assess eGFR with urine Alb/Cr ratio

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

AKI assessment

AKI Warning Stage Test Result Confirm or refute automated AKI Test Result by comparing patient's current creatinine against patient's baseline creatinine	**Clinical Context Within Which Blood Test Taken**	
	LOW Pre-test Probability of AKI Stable Clinical Context	**HIGH Pre-test Probability of AKI** Context of Acute Illness
AKI Warning Stage 1 Current creatinine> 1.5 x baseline level (*or* creatinine rise >26 µmol/L ≤ 48 hrs)	**Consider clinical review ≤ 72 hours of e-alert** **If AKI confirmed**→ manage as per table 2	**Consider clinical review ≤ 24 hours of e-alert** **Likely Stage 1 AKI**→ manage as per table 2
AKI Warning Stage 2 Current creatinine> 2 x baseline level	**Consider clinical review ≤ 24 hours of e-alert** **If AKI confirmed**→ manage as per table 2	**Consider clinical review ≤ 6 hours of e-alert** **Likely Stage 2 AKI**→ manage as per table 2
AKI Warning Stage 3 Current creatinine> 3 x baseline level (*or* creatinine> 1.5 x baseline and > 354 µmol/L)	**Consider clinical review ≤ 6 hours of e-alert** **If AKI confirmed**→ consider admission	**Consider Immediate Admission** Likely Stage 3 AKI

AKI management

"Think" Cause	"Think" Medication	"Think" Fluids	"Think" Review
History of acute illness? • Think Sepsis • Think Hypotension Intrinsic renal disease (e.g. vasculitis)? • Think Urinalysis Urinary tract obstruction?	Any drugs which could **exacerbate** AKI? Consider withholding: • NSAIDs • Diuretics • Antihypertensive medication Any drugs which may **accumulate** and cause harm during AKI? Any **new** drugs that may be **causing** AKI (e.g. drug-induced interstitial nephritis)?	What is the patient's volume status? If hypovolaemia present: • When did patient last pass urine? • Can patient increase fluid intake? • Does patient have and / or need carer support? • Is admission for IV fluids and monitoring required?	Does patient need admission? If not, when will you review? Have you ensured handover?

Approach to U&E interpretation Step 1: Assess urea, creatinine & eGFR

- Assessing the urea and creatinine in conjunction can allow you to determine whether there is potential kidney injury/damage. If **both are raised**, this suggests **renal dysfunction** and the eGFR will be low.
- Remember, an **isolated raised urea** can indicate dehydration or upper gastrointestinal bleeding.
- **Step 1: Compare to previous creatinine / eGFR**
- Without a factor leading to an inaccurate eGFR calculation (e.g. obesity), a **high creatinine** and **low eGFR** suggest renal dysfunction.
- It is then important to assess whether this is **acute** (i.e. acute kidney injury) or **longstanding** (i.e. chronic kidney disease).
- **Approach to U&E interpretation**

- **Step 2: Assess urea, creatinine & eGFR**
- Assessing the urea and creatinine in conjunction can allow you to determine whether there is potential kidney injury/damage. If **both are raised**, this suggests **renal dysfunction** and the eGFR will be low.

Sodium

- Sodium is the main determinant of **plasma osmolality**, and serum levels are closely related to hydration (volume) status. Serum sodium levels are normally regulated by **antidiuretic hormone** (ADH), amongst other homeostatic mechanisms.
- Symptoms of both hypernatremia and hyponatremia are primarily **neurological**. Mild symptoms include fatigue, weakness and confusion, but can progress to severe symptoms such as seizures and coma.
- The severity of symptoms is related to the **severity of sodium derangement** and the **pace of the change** in serum sodium concentration (rapid changes lead to severe symptoms).

Hypernatraemia

- Rarer causes of hypernatraemia include:
  - Diabetes insupidus
  - Drugs (e.g. loop diuretics)
  - Osmotic diuresis (e.g. in hyperglycaemic states)
  - Extreme levels of salt ingestion
  - In most cases, where there is a clear history implicating dehydration (e.g. vomiting/diarrhoea), treatment involves **gentle rehydration** with intravenous hypotonic fluids (e.g. 5% dextrose).
- It is essential to **monitor sodium levels** during treatment, as correcting the hypernatraemia too rapidly can lead to **intracerebral fluid shifts** and devastating consequences such as **central pontine myelinolysis** (also known as osmotic demyelination syndrome).

Hyponatraemia

- Even mild levels of hyponatraemia may predispose to falls and cognitive deficits. Chronic hyponatraemia is also a risk factor for osteoporosis and fragility fractures.
- Symptoms range in severity and include:
  - Lethargy (excessive)
  - Headache
  - Nausea
  - Vomiting
  - Irritability
  - Confusion
  - Seizures
  - Reduced conscious level, loss of consciousness, coma
  - Cardiorespiratory distress
  - Worsening symptoms suggest cerebral oedema, which is a life-threatening medical emergency.

Hyponatraemia causes

Diuretics - Thiazides	Indapamide, Bendroflumethiazide, Chlorthalidone.	Renal losses
Diuretics – Loop Particularly in combination with ACE-I or spironolactone	Furosemide, Bumetanide	Renal losses
Anticonvulsants	Carbamazepine, Oxcarbazepine, Phenytoin, Sodium valproate, Lamotrigine and other antiepileptics	ADH release or action stimulus (SIADH)
Opiates	Morphine, Tramadol	ADH release or action stimulus (SIADH)
Chemotherapuetic agents	Vincristine, Vinblastine, Carboplatin, Cisplatin, Cyclophosphamide	ADH release or action stimulus (SIADH)
Anti-psychotics	Aripiprazole, Clozapine, Fluphenazine, Haloperidol, Risperidone , Thioridazine	ADH release or action stimulus (SIADH)
Anti-depressants (tricyclics, SSRIs)	Sertraline, Fluoxetine, Paroxetine, Citalopram, Venlafaxine, Amitriptyline	ADH release or action stimulus (SIADH)
NSAIDs and COX-2 inhibitors Particularly in combination with thiazides or heart failure	Ibuprofen, Diclofenac, Naproxen, Celecoxib	Loss of ADH inhibition (SIADH)

Potassium

- Derangements in potassium levels are common in hospital inpatients.

- Under physiological conditions, potassium is stored almost entirely **intracellularly** and is **excreted by the kidneys**.

- Derangements in potassium levels cause **myocardial instability**, leading to risks of potentially fatal arrhythmias.

Hyperkalaemia

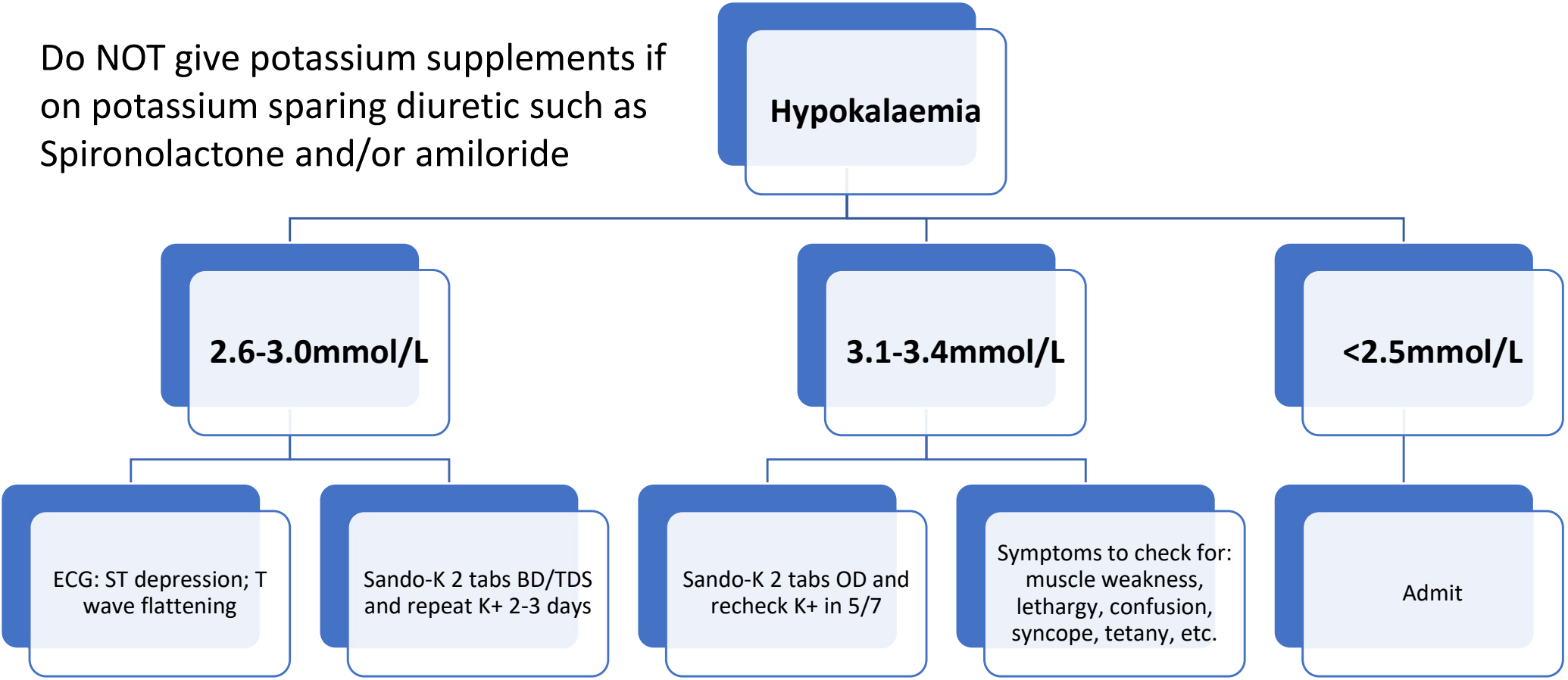
- Hyperkalaemia is defined as serum potassium >5.5mmol/L. It can be further classified in terms of severity as per European Resuscitation Council guidelines:

- Causes of hyperkalaemia can be varied:
  - Renal: decreased renal excretion (e.g. AKI or CKD)
  - Medications: ACE inhibitors, potassium-sparing diuretics, potassium supplements
  - Tissue damage: burns, rhabdomyolysis
  - Metabolic: diabetic ketoacidosis
  - Endocrine: Addison's disease

Severe hyperkalaemia is a medical emergency requiring immediate management. If ECG changes occur, the most critical initial step is administering intravenous calcium gluconate, stabilising the myocardium. Once this is administered, treatment focuses on lowering the serum potassium concentration, often via insulin/dextrose infusion.

Hypokalaemia

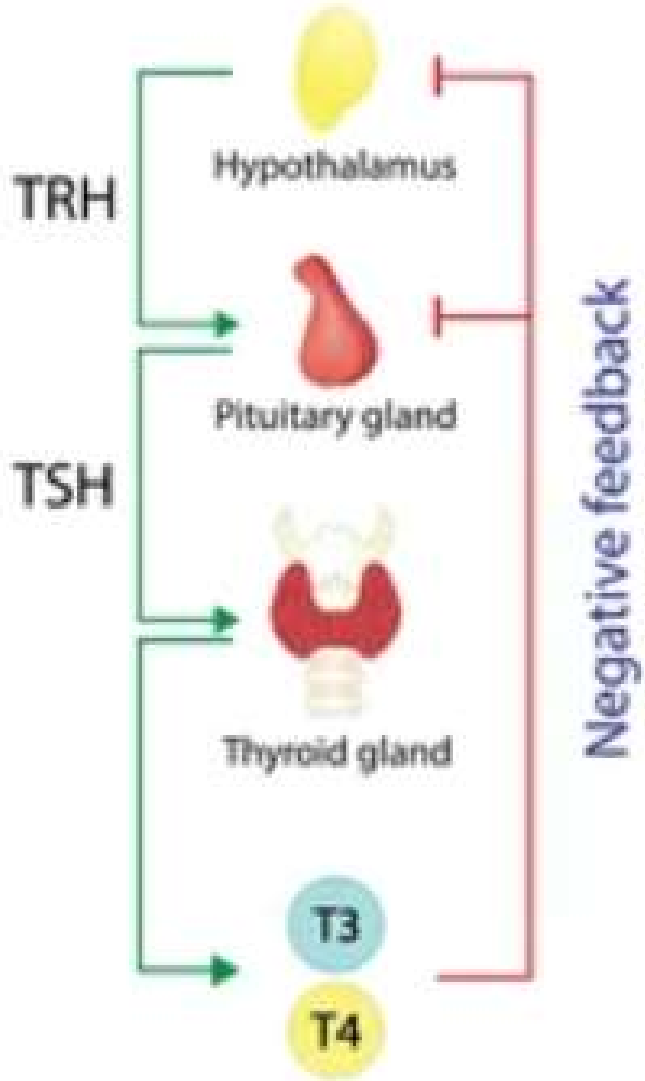
Do NOT give potassium supplements if on potassium sparing diuretic such as Spironolactone and/or amiloride



Thyroid Function Test

Test	Reference range
Serum TSH	0.30- 4.2 mU/L
Serum free T4 level	9.0- 23.0 pmol/L
Serum free T3 level	2.4- 6.0 pmol/L

Thyroid Hormonal Axis



Thyroid Hormonal Axis

Triiodothyronine (T3)

Thyroxine (T4)

Thyroid stimulating hormone (TSH)

Thyroglobulin antibodies (Tg Ab)

Thyroid peroxidase antibodies (TPO Ab)

Thyroid-stimulating hormone receptor antibodies (TSHR Ab)

Key clinical features

Hypothyroidism	Hyperthyroidism
Lethargy	Hyperactivity
Weight gain	Weight loss with increased appetite
Intolerance to cold	Intolerance to heat
Constipation	Diarrhoea
Dry Skin	Sweating
Bradycardia	Tachycardia/AF
Menorrhagia	Oligomenorrhoea/amenorrhoea
Hair loss	Fine tremor
Depression	Muscle weakness and wasting

Hypothyroidism

- Primary hypothyroidism involves reduced secretion of thyroid hormone from the thyroid gland itself.
- Pathology which decreases the thyroid's ability to release T4 and T3 or respond to TSH can, therefore, cause primary hypothyroidism.
- Primary hypothyroidism is the most common cause of hypothyroidism, accounting for 99% of all cases.

- Secondary hypothyroidism involves a reduction in the hormones that stimulate the thyroid to produce thyroxine.
- Pathology which affects the pituitary and hypothalamic glands can result in decreased production of TRH and TSH, causing secondary hypothyroidism.
- Secondary hypothyroidism is a rare cause of hypothyroidism, accounting for 1% of all cases.

Hyperthyroidism

Primary hyperthyroidism involves an excessive production of T3 and T4 by the thyroid gland because of pathology within the thyroid gland itself.

Secondary hyperthyroidism involves stimulation of the thyroid gland by excessive thyroid-stimulating hormone (TSH).

Case Study 1

Mary is found to have a High TSH of 9.1 with a normal T4. She has been feeling tired but has no other symptoms

A) Do nothing

B) treat with thyroxine

C) repeat in 3 months

D) Something else

Case Study 2

- Alexa is found to have a High TSH at 12, with a low T4
- Diagnosis? Specific Action?

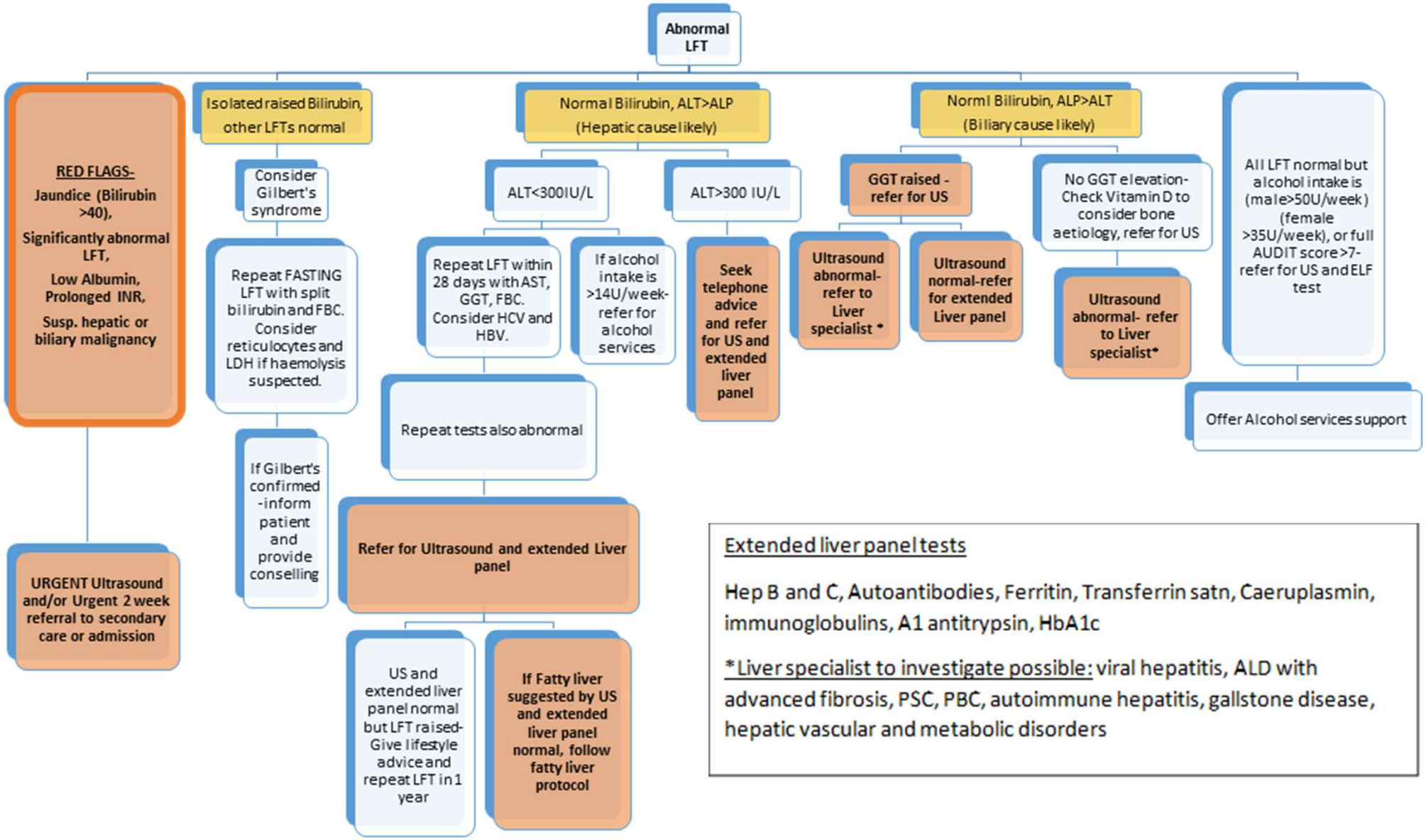
A) Do nothing

B) Treat with thyroxine

C) Repeat in 3 months

D) Something else

Overview



Extended liver panel tests

Hep B and C, Autoantibodies, Ferritin, Transferrin satn, Caeruplasmin, immunoglobulins, A1 antitrypsin, HbA1c

*Liver specialist to investigate possible: viral hepatitis, ALD with advanced fibrosis, PSC, PBC, autoimmune hepatitis, gallstone disease, hepatic vascular and metabolic disorders

Case Study 1

Alex is a 32-year-old male who used to use intravenous drug use. He complains of mild tiredness. He has elevated ALT and AST levels but normal bilirubin and Alk phosphatase. What would you do next?

Case Study 2

Sarah is a 38-year-old female who is obese, has dyslipidaemia and Type 2 diabetes mellitus. She is asymptomatic. He has elevated ALT and AST levels but normal bilirubin and Alk phosphatase. What would you do next?

Case Study 3

John is a 45-year-old male who is a long term heavy alcohol user. He is jaundiced and has abdominal swelling. He complains of fatigue and weakness. He has elevated ALT and AST levels as well as raised gamma-GT. Bilirubin is raised as well. What would you do next?

Thank you

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