Phase Ia PoM: TALES FROM THE HAEMATOLOGY CLINIC: RED BLOOD CELLS, October 2025

Case 1

Q1. Reduced Hb, Hct, RBC, MCV, MCH and MCHC
Blood count: low Hb, other clues – low MCV, MCH, MCHC, Hct, RBC
Clinical history: fatigue, dizziness, heavy menstrual bleeding
Blood film:
1. Small (microcytic) red cells – correlates with reduced MCV
2. Pale (Hypochromia)
3. Poikilocytes: pencil cell (elliptocyte) in the top right corner of the film
Q.2 a) **Mechanism**: Failure of production – hypochromic microcytic anaemia consistent with defect in Haemoglobin synthesis
b) **Cause**: iron deficiency secondary to heavy menstrual bleeding (menorrhagia)
c) Further blood test – iron studies will show reduced serum ferritin and increased transferrin
Q 3. **Iron replacement therapy** such as ferrous sulphate tablets can be given. Although the anaemia will improve with iron replacement therapy, it is very important to ascertain the cause of the anaemia. The Haemoglobin will correct fairly quickly, however it takes longer for the iron stores (ferritin) to replenish, which is why it is important to continue iron replacement tablets for around 3 months after correction of Hb.

Case 2

Q1. Blood count:
Reduced MCV, MCH (like case 1)
Raised RBC (unlike case 1)
Preserved Hb and MCHC (unlike case 1)
Q2. –microcytic red cells consistent with defect in Haemoglobin synthesis
But unlike Case 1 he is **not anaemic** (normal Hb) and has a **raised RBC count** and cells are not hypochromic (MCHC normal)
The **normal Hb** with **microcytic indices** and **raised red blood cell count** is consistent with **thalassaemia trait**.
This is also supported by the gentleman's **ethnicity**
Q3. Difference in normal range reflects **gender** difference (male patient)
Q4. 1. Haemoglobin electrophoresis/HPLC
2. Iron studies/Ferritin
Q5. a) Hb electrophoresis on agarose gel at alkaline pH (pH 8.6): (Lane 1) Hb A and increased Hb A2 (beta/β-thalassemia); (Lane 2) Hb A and small trace Hb A2 (normal);
b) **raised Haemoglobin A2 is** found where there is a reduction in beta globin chains, caused by **β thalassemia (or trait),** an example of a **haemoglobinopathy**
HbA2 is made up of 2 alpha globin chains and 2 delta globin chains. HbA2 results from alpha globin chains combining with delta chains as a result of a reduction in beta globin chains. Demonstrating an increase in HbA2 by gel electrophoresis or HPLC (high performance liquid chromatography) is a useful diagnostic tool in β thalassemia or β thalassemia trait. In Case 2 the normal Hb is consistent with the trait (carrier) state.
Note that **Hb electrophoresis** is **normal** in patients with **alpha/α thalassaemia trait,** and definitive diagnosis can only be made by molecular analysis.
Q6. 1. To replace iron where this is deficient and not incorrectly prescribe iron supplements that have no effect in thalassaemia trait
2. To advise individuals with thalassaemia trait on potential risks to future offspring (genetic counseling)
Further reading material on alpha and beta thalassaemia is available as Consolidation material on Insendi

Case 3

Q1 a) **Reduced RBC production** –low MCV suggests defect in haemoglobin synthesis
b) Distinguishing between **iron deficiency anaemia** and **anaemia of chronic disease** is often not straightforward. The patient's clinical history is obviously very important.
Also consider why this is not thalassaemia? Clue is in the RBC count and MCHC!
c) Inflammatory markers e.g. CRP, ESR. Iron studies /Ferritin
Q2. As patients with anaemia of chronic disease have plenty of storage iron, treating with iron replacement therapy (as you would for patients with iron deficiency anaemia) will not help and should be avoided. Controlling the underlying disease to reduce inflammation (e.g. by treating the infection) will treat the anaemia. However, this is not always possible and some of these patients benefit from erythropoietin treatment.

Case 4

Q1. Reduced Hb, raised MCV – Macrocytic anaemia
Blood film shows macrocytes – round and some oval macrocytes,
Occasional tear drop cells – see 9pm (mid-left of film)
Q2. a) **Mechanism**: Failure of production (no evidence haemolysis – no polychromasia)
b) Cause could be B12 or folate deficiency. Liver disease/ethanol can also cause macrocytosis
Q3. As well as being required for DNA synthesis, Vitamin B12 is required for the integrity of the nervous system. The patient's 'pins and needles' symptoms (known medically as 'paraesthesia') is due to Vitamin B12 deficienct

Case 5

Blood count: Low Hb, raised MCV, raised platelet count
Clinical history: suggests longstanding haematology condition, why might he be taking folic acid and penicillin?
Blood film:

- **Sickle cells**
- **Polychromatic cells** (bluish and larger). Polychromasia means that cells have a blue tinge, caused by the ribosomal RNA in young red cells, in addition to the pink colour of the haemoglobin – hence 'polychromasia' meaning 'many colours'
- Increased platelets
- **Target cells** are red cells with an accumulation of haemoglobin in the centre of the area of central pallor
- Target cells may occur in a number of different conditions :
  - obstructive jaundice
  - liver disease
  - haemoglobinopathies
  - hyposplenism

Q.1 Increased destruction/reduced red cell survival (haemolysis)
Q.2 Sickle cell disease
Q.3 HbS and HbF –Note no HbA
Q.4 Homozygous sickle cell disease (HbSS). HbSS is also known as Sickle cell anaemia
Q.5 **Pencillin V:** Patients with HbSS are functionally hyosplenic due to infarction of the spleen, usually by the age of 5 years. This puts them at increased risk of infections by encapsulated bacteria. To reduce this risk they require vaccination against and to take daily prophylactic (preventative) antibiotics
Folic acid: in haemolytic anaemias such as HbSS the increased red blood cell turnover and production increases the folic acid requirement.

Case 6

Q.1 There is cyanosis as shown by the blue colouring of the lips. This indicates hypoxia (reduced oxygen levels)
Q.2 The full blood count shows an increase in Haemoglobin concentration (Hb), Haematocrit (Hct) and Red Blood Cell count (RBC), consistent with **Polycthaemia.**

In this patient the Polycythaemia is due to increased production of Erythropoietin as a response to hypoxia i.e.the erythropoietin is appropriately increased as shown on the next slide. See Red cell lecture slides for further information.