Please note – this learning resource has been produced by the GUMS Academic Team. It is possible that there are some minor errors in the questions/answers, and other possible answers that are not included below. Make sure to check with other resources.

What are the likely morphological classification of anaemia seen in the following cases (eg. Macrocytic, microcytic and normocytic) and discuss why

**1) A 20 year old woman complaining of fatigue, lethargy and occasional shortness of breath, has just found out she is pregnant, eats a vegetarian diet and has suffered from heavy menstrual bleeding.**

**Microcytic anaemia**

This is a case of Iron deficiency anaemia which is commonly a mixed picture with diet alone being seldom sufficient for an iron deficiency.

It is more common therefore, for iron deficiency anaemia to present with low iron diet and one of the following

blood loss (chronic) --- heavy menstrual, peptic ulcers etc

 Malabsorption of iron (duodenum);

↑ iron requirement (rapid growth in infancy, childhood, pregnancy, lactation, cancer)

Essentially this will result in a **microcytic** anaemia (small MCV)

Microcytic anaemia occurs as there is a defective haemoglobin synthesis due to the lack of iron

**2) A 6 year old boy who really likes Mediterranean food (because that’s where his family is from but otherwise eats a stable diet for a 6 year old) and has been for haemoglobin electrophoresis which shows a Beta- Thalassaemia**

**Microcytic** **anaemia** -> defective haemoglobin due to mutation in beta chain gene -> Structural issue with the beta chain of haemoglobin resulting in aggregation of alpha chains and changes in haemoglobins structure.

3 **) A 30 year old women diagnosed with Crohn’s disease (*an inflammatory bowel disease that affects transmural gastrointestinal tract and impairs absorption*) that is particularly affecting her terminal ileum *(hint (this is covered in systems): Iron (fe = duodenum) fist ( folate = jejunum) buddy buddy (bile and B 12 = terminal ileum)***

B 12 deficiency leads to **MACROCYTIC** anaemia

B12 combines with intrinsic factor to be absorbed in the terminal ileum, thus if Crohn’s disease was present at the terminal ileum, it could result in B12 malabsorption.

Due to lack of B12 this causes defective DNA synthesis – thus the RBCs are unable to fully mature and become large (macrocytic) and premature

Note: Iron deficiency anaemia or mixed is the most common type of anaemia that arises in crohns disease(IBD). This is due to intestinal bleeding, the malabsorption of B12 in this scenario is to highlight B12s absorption only.

1. **A 50 year old man self-presented to the ED who was just stabbed 8 times in the abdomen because he forgot to pay his friend for that sick bike they sold him… and now is bleeding profusely**

**Normocytic:** No issues in haemoglobin synthesis or degradation etc (no pathophysiology) at the moment because most of the blood is on the floor.

NB: If you took a blood sample right at the time they were bleeding profusely, they might not even be anaemic.

1. **A 70 year old woman who is currently receiving dialysis for failing kidneys**

**Normocytic:**

**CKD -** ↓erythropoietin = ↓ no. of RBC made however synthesis process is still normal (eg. Nil haem/ globin issues)

**What does the P wave represent?**

Atrial depolarisation

**What does the T wave represent?**

Ventricular Repolarisation

Lub dub:

**S1 is heard due to**

Beginning of Systole: so the ventricles are beginning to contract and rising pressure causes the mitral and tricuspid valves to close, the sound is turbulent blood flow caused by blood hitting the closed mitral and tricuspid valves.

**S2 is heard due to**

Beginning of diastole: the relaxation of the ventricles aortic and pulmonary valves closing, the sound is turbulent blood flow caused by blood hitting the closed aortic and pulmonary valves

 **Explain the difference between essential and secondary hypertension with examples.**

Essential HTN (essential HTN) : no exact identifiable cause of HTN, however thought to be associated with lifestyle factors (age, diabetes, smoking, obesity, environment, family history)

Secondary HTN: identifiable cause e.g. renal artery stenosis (↓blood flow to kidney = ↑ renin) Pheochromocytoma (adrenaline producing tumour), Aortic coarctation (↓blood flow to kidneys ↑ renin) etc.

**Explain the difference between primary and secondary hyperlipidaemia with examples**

Primary hyperlipidaemia: due to family history/ genetic disposition (therefore is non-modifiable and requires medications needed to address lipid profile.

Secondary hyperlipaemia: Due to secondary causes (eg. Diabetes, lifestyle, see secondary HTN)

**PART 2 ANSWERS**

**Question 1:**

Nitrates cause relaxation of smooth muscle cells within the tunica media of blood vessels causing vasodilation, specific to the heart, they increase coronary blood flow.

**Question 2:**

During diastole, the heart's muscles are relaxed and blood is entering its chambers. The AV (mitral and tricuspid) valves are open to allow blood flow from the atria into the ventricles. The semilunar (aortic and pulmonary) valves are closed to prevent backflow from the aorta and pulmonary trunk.

**Question 3:**

**Name 6 medical conditions that could have cause Mr Sander’s chest pain (you may not have covered all 6):**

Vascular (2): Aortic dissection, Pulmonary Embolism

Cardiac (2): Acute coronary syndrome (STEMI) = angina, Cardiac Tamponade

Respiratory (1): Pneumothrax, pleural effusion

GI (1): Heart burn (Gastro-reflux) Boerhaave's syndrome (oesophageal rupture)

**Question 4:**

**What is the main clinical difference between Stable and Unstable Angina?**

Stable Angina: Pain on exertion. Resolves with rest

Unstable Angina: Pain at rest

**Question 5:**

**Name 2 Modifiable and 2 Non-Modifiable Risk Factors Mr Sanders has for Angina**

**Modifiable:** Diet and Smoking

**Non Modifiable:** Male and Age

**Question 6**

**Place the following in the correct order of the formation and development of an atherosclerotic plaque (1-10)**

|  |  |
| --- | --- |
| LDLs (low density lipids) become oxidised and release chemotactic agents.  | **3** |
| Endothelial cells become activated and macrophages produce chemicals that activate smooth muscle cells and cause their proliferation and migration into the tunica intima blood vessels. | **7** |
| A fibrous cap forms, containing ECM (extracellular matrix) proteins such as collagen and fibrin  | **8** |
| There is a loss of endothelial integrity and initiation of the inflammatory process  | **1** |
| LDLs move from the lumen of the blood vessels into its tunica media | **2** |
| Macrophages engulf the oxidised LDLsm transforming into foam cells.  | **6** |
| Monocytes are attracted to the area | **4** |
| The lipid core can become necrotic.  | **10** |
| The fibrous cap hardens and has the potential to rupture, this triggers the formation of a thrombus.  | **9** |
| Monocytes migrate between the tight junctions of the endothelial cells to reach the sub-endothelial space and differentiate into macrophages.  | **5** |

Appendix to help





**Please provide feedback for this case at:** [**https://gums2020.typeform.com/to/e0h7US**](https://gums2020.typeform.com/to/e0h7US)