

Haematology SBA Questions

Questions were made by students on behalf of The Peer Teaching Society. We hope there are no mistakes but are not liable for any false or misleading information.

1. Which of the following is not a cause of microcytic anaemia?
 - a) Iron deficiency
 - b) Beta thalassaemia
 - c) Sideroblastic anaemia
 - d) Folate deficiency
 - e) Anaemia of chronic disease

2. Which of the following is the correct mechanism of absorption of vitamin B12?
 - a) B12 binds to CCK produced by enterochromaffin cells
 - b) B12 binds to intrinsic factor produced by the parietal cells of the stomach
 - c) B12 binds to intrinsic factor produced by the terminal ileum
 - d) B12 binds to pepsinogens produced by the chief cells of the stomach
 - e) B12 diffuses freely into the epithelial cells of the terminal ileum

3. What is the management for hereditary spherocytosis?
 - a) Ferrous sulphate
 - b) Life-long blood transfusions
 - c) Fresh frozen plasma
 - d) Splenectomy
 - e) Bone marrow transplant

4. Which of the following describes the appearance of a blood film of a patient with beta thalassaemia major?
 - a) Large and small irregular hypochromic RBCs
 - b) Sickled erythrocytes
 - c) Oval macrocytes (large RBCs) with hypersegmented neutrophil polymorphs with six or more lobes in the nucleus
 - d) Blast cells present
 - e) Plasmodium falciparum present

5. A 72-year-old man presents to A&E complaining of general malaise, back pain and not passing urine in a few days. Blood tests were performed with the results below:

- Hb 64g/dL (130-180)
- WCC 6.8×10^9 ($4 \times 10^9 - 11 \times 10^9$)
- Platelets 300×10^9 ($150 \times 10^9 - 450 \times 10^9$)
- Creatinine 400umol/L (60 – 110)
- Sodium 138mmol/L (135 -145)
- Calcium 3.5mmol/L (2.1-2.6)

Which of the following conditions is most likely to be the cause of his symptoms?

- a) Chronic Lymphocytic Leukaemia
- b) Acute Myeloid Leukaemia
- c) Multiple Myeloma
- d) Hodgkin's Lymphoma
- e) Non-Hodgkin's Lymphoma

6. Which of these findings would you expect to confirm a diagnosis of multiple myeloma in a patient?

- a) Leukaemic blast cells
- b) Rouleaux formation
- c) Auer rods
- d) Reed-Sternberg cells
- e) Raised myeloid cells

7. The most severe form of the disease malaria with the highest rate of mortality in humans is caused by which species of mosquito?

- a) Plasmodium vivax
- b) Plasmodium ovale
- c) Plasmodium malariae
- d) Plasmodium knowlesi
- e) Plasmodium falciparum

8. A 38-year-old lady presents to her GP complaining of feeling very 'warm and cold' for the past few days. She recently got back from visiting family in Africa and since then she has had some vomiting and diarrhoea, abdominal pain and has just been feeling unwell. Which diagnosis is most likely in this lady?
- a) Uncomplicated malaria
 - b) Tuberculosis
 - c) Complicated malaria
 - d) Food poisoning
 - e) Cholera

9. You see a 52-year-old female who went to her GP as she noticed her lymph nodes were enlarged. She has also had a fever recently and is worried she has an infection. You run some blood tests and see a raised ESR and lactose dehydrogenase. You refer her to oncology who do a lymph node biopsy and imaging.

The images show non-Hodgkin lymphoma in both her upper and lower body. There are no signs of non-lymph node involvement.

What would the staging for this woman's lymphoma be?

- a) Stage II A
 - b) Stage II B
 - c) Stage III A
 - d) Stage III B
 - e) Stage IV B
10. You are a CT2 training in oncology. You see a 3-year-old child who has been unwell recently, is breathless and keeps getting headaches. Her GP has referred to her to your department for further investigations.

Without any further investigations, what is your immediate concern?

- a) Acute Myeloid Leukaemia
- b) Acute Lymphoblastic Leukaemia
- c) Chronic Myeloid Leukaemia
- d) Chronic Lymphoblastic Leukaemia
- e) Multiple Myeloma

11. You have been seeing a 57-year-old male regularly in your clinic. He has lost a significant amount of weight in the last 6 months, has gout, abdominal pain and is anaemic. Your most recent investigations show raised myeloid cells and a high white cell count. You also see increased cellularity on a bone marrow biopsy. You diagnose chronic myeloid leukaemia.

Which of the following would you give this man?

- a) Ibrutinib
- b) Rituximab
- c) Imatinib
- d) Morphine
- e) Dexamethasone

12. Which of the following statement is false regarding thalassaemia?

- a) Alpha thalassaemia is incompatible with life
- b) Not all beta thalassaemia patients are dependent on blood transfusions
- c) Alpha thalassaemia is diagnosed with haemoglobin electrophoresis
- d) Beta thalassaemia is diagnosed with a blood film
- e) Alpha thalassaemia results in alpha chain gene depletion

13. Which of the conditions below will lead to elevated bilirubin levels?

- a) Immune thrombocytopenic purpura
- b) Thrombotic thrombocytopenic purpura
- c) Haemophilia A
- d) Haemophilia B
- e) Von Willebrand disease

14. Which of the following is a cause for secondary polycythaemia vera?

- a) Dehydration
- b) Acute blood loss
- c) High altitude
- d) JAK2 kinase mutation
- e) Leukaemia

15. Which of the following conditions are not a myeloproliferative disorder?

- a) Myelofibrosis
- b) Polycythaemia vera
- c) Essential thrombocytopenia
- d) Lymphoma
- e) Hypereosinophilic syndrome

16. Which of the following is not part of Virchow's Triad?

- a) Stasis of blood flow
- b) Hypercoagulability
- c) Endothelial injury
- d) Diameter of the blood vessel

Question	Answers
<p>1. D</p>	<p>Folate deficiency is a cause of macrocytic megaloblastic anaemia. Folate is essential for DNA synthesis and so in deficiency there is impaired DNA synthesis, resulting in delayed nuclear maturation and large RBCs, as well as decreased RBC production in the bone marrow. Peripheral film shows oval macrocytes (large RBCs) with hypersegmented neutrophil polymorphs with six or more lobes in the nucleus. The other options are all causes of microcytic anaemia. Anaemia of chronic disease may be microcytic or normocytic. Microcytic anaemia - MCV < 80 fL Normocytic anaemia – MCV = 80-100 fL Macrocytic anaemia - MCV > 100 fL</p> <div style="text-align: center;"> <p>Bone marrow erythroid series</p> <pre> graph TD A[Bone marrow erythroid series] --> B[Normoblastic] A --> C[Megaloblastic] A --> D[Sideroblastic] B --> B1[Iron-deficiency anemia] B --> B2[Infection] B --> B3[Renal disease] B --> B4[Malignancy] B --> B5[Connective tissue disorders] B --> B6[Hemolytic anemia] C --> C1[Vitamin B12 deficiency] C --> C2[Folic acid deficiency] C --> C3[Miscellaneous] C --> C4[Congenital disorders in DNA synthesis] C --> C5[Acquired disorders in DNA synthesis] C --> C6[Drug-induced] D --> D1[Hereditary/congenital] D --> D2[Acquired clonal] D --> D3[Acquired reversible] </pre> </div>
<p>2. B</p>	<p>B12 binds to intrinsic factor which is produced by the parietal cells of the stomach, it is then absorbed in the terminal ileum. The parietal cells are also involved in acid secretion. Chief cells do produce pepsinogen, but they are not involved in B12 absorption. In pernicious anaemia (most common cause of vitamin B12 deficiency) the parietal cells of the stomach are attacked by the immune system, resulting in atrophic gastritis and the loss of intrinsic factor production.</p>
<p>3. D</p>	<p>Pathophysiology of hereditary spherocytosis:</p> <ul style="list-style-type: none"> • Caused by defects in the red cell membrane resulting in large RBCs losing part of their cell membrane as they pass through the spleen, possibly because the lipid bilayer is inadequately supported by the membrane skeleton due to deficiency in the structural protein spectrin • There is an increased permeability to Na⁺ requiring an increased rate of active transport out of cells • Surface to volume ratio decreases and the cells become spherocytic (more rigid and less deformable than normal RBCs - unable to pass through splenic microcirculation, so become trapped and destroyed via extravascular haemolysis) <p>Management:</p> <ul style="list-style-type: none"> • Relieves symptoms due to anaemia or splenomegaly, reverses growth failure and prevents recurrent gallstones • Best to postpone until after childhood due to infection risk post-op • After operation give appropriate immunisation and life-long penicillin prophylaxis

4. A	<p>Beta thalassaemia is an example of a haemolytic anaemia. RBCs can either be normocytic or if there are many young RBCs (reticulocytes – which are larger) due to excessive destruction of old RBCs then macrocytic.</p> <p>Sickled erythrocytes are seen in sickle cell anaemia, another type of haemolytic anaemia.</p> <p>Oval macrocytes (answer C) would be seen on the blood film of a patient who has megaloblastic anaemia i.e. B12 or folate deficiency.</p> <p>Blast cells are seen in acute lymphoblastic leukaemia.</p> <p>Plasmodium falciparum is one of the causative organisms of malaria.</p>
5. C	<p>CLL is a leukaemia affecting the B cells, it is the most common leukaemia normally affecting adults > 70-years-old. Often, those presenting with CLL are asymptomatic or have enlarged, rubbery non-tender nodes – not the symptoms described in the question. As for the blood results, CLL causes a high WCC with high lymphocytes and a blood film would show small, mature lymphocytes. The blood results do not confirm this diagnosis as the WCC is not raised.</p> <p>AML tends to affect those aged over 40-years. It presents with anaemia, bleeding and infection. The blood results in a patient in AML would show anaemia, thrombocytopenia and neutropenia. On a bone marrow biopsy, you would expect to see Auer Rods to confirm a diagnosis. The blood results do not confirm this diagnosis as the patient has normal platelet levels not thrombocytopenia.</p> <p>Multiple myeloma presents in adults aged over 70. The most prominent features on presentation of multiple myeloma can be remembered using CRAB. There are also non-specific presentations of tiredness/malaise or infection.</p> <p style="padding-left: 40px;"> C – calcium over 2.75mmol/L R – Renal impairment A – Anaemia B – Bone lesions -> pepper pot skull, cord compression, back pain </p> <p>The blood results confirm this diagnosis, the low Hb shows anaemia, WCC and platelets remain normal, high creatinine suggests renal impairment and high calcium.</p> <p>Hodgkin’s Lymphoma has a bimodal incidence affecting young adults and the elderly. The most common presentation of HL is fevers, sweating, enlarged rubbery non-tender nodes and systemic B symptoms (loss of appetite/night sweats). You would expect to see anaemia on the blood results however the remaining blood results do not fit with this diagnosis.</p> <p>Non-Hodgkin’s Lymphoma affects adults aged over 40 years. The presentation of NHL is similar to HL except B symptoms are less common and there may be some GI and skin involvement.</p>
6. B	<p>You would expect to find Leukaemic blast cells on the blood film of a patient with acute lymphoblastic leukaemia (ALL)</p> <p>Rouleaux formation is seen on the blood film of patients with multiple myeloma</p> <p>Auer Rods are seen on a bone marrow biopsy of someone with Acute Myeloid Leukaemia (AML)</p> <p>The Reed-Sternberg cell is a characteristic cell of Hodgkin’s Lymphoma</p> <p>Raised myeloid cells would be seen in chronic myeloid leukaemia (CML)</p>
7. E	<p>P. vivax can also cause severe disease and kill people; however, it contributes much less to the global burden of malaria than P. falciparum. Mainly found in Asia and South America. This species can stay in the liver for up to 3 years therefore there is the potential for relapses.</p>

	<p>P. ovale – this is very uncommon and usually is found in West Africa. This species can remain in your liver for several years without producing symptoms</p> <p>P malariae – this species is quite rare and usually only found in Africa</p> <p>P. knowlesi – this species is very rare and found in parts of southeast Asia.</p> <p>P. falciparum – causes the most severe form of malaria and has the highest rate of mortality. This species is mainly found in Africa, it is the most common type of malarial parasite.</p>
8. A	<p>Uncomplicated malaria – the key feature in this question was the feeling ‘warm and cold’ which represents the fever causing sweats and chills in an individual with malaria. Questions with fever and recent travel – THINK could it be malaria!</p> <p>TB – These symptoms this lady presented with are not typical of TB. A persistent cough, weight loss, night sweats and a fever are the symptoms expected in a TB infection</p> <p>Complicated malaria is characterised by vascular occlusion which can affect different organs therefore causing specific symptoms e.g., cerebral malaria leads to micro-infarcts and therefore symptoms include drowsiness, increased intracerebral pressure causing seizures and coma.</p> <p>Food poisoning – this would cause similar symptoms to uncomplicated malaria however the key in this question is the recent travel!</p> <p>Cholera is caused by the bacteria <i>Vibrio cholerae</i> and this releases a toxin which increases water release from intestinal cells resulting in severe diarrhoea. Infection occurs through ingestion of contaminated food or water.</p>
9. D	<p>This patient’s lymphoma has spread to nodal areas on both sides of the diaphragm so it is Stage III. She also presents with a fever so has systemic symptoms, therefore her stage would include the suffix B. Stage II describes a cancer that is one a single side of the diaphragm, and stage IV has spread beyond the lymph nodes.</p>
10. B	<p>ALL is your paediatric cancer. It is most common in 2-4 year olds and can present with recurrent infection and general unwellness. You might also see hepatosplenomegaly, SVC obstruction and back pain (bone marrow failure) however these are not always present. AML and CML are seen in adults over 40, and CLL is seen in adults over 70.</p>
11. C	<p>Imatinib is a tyrosine kinase inhibitor, which is given in CML alongside chemotherapy. Ibrutinib is a bruton kinase inhibitor, and is given in CLL. Rituximab is a monoclonal antibody given in CLL, and dexamethasone is a steroid, which is part of the treatment therapy in multiple myeloma.</p>
12. A	<p>Alpha thalassaemia can be incompatible with life, however it is only the 4 gene deletion sub-group that this applies to . The 3 gene deletion and 2 gene deletion groups will survive. Beat thalassaemia minor patients do not require life-long transfusions, and it is diagnosed with a blood film. Alpha thalassaemia is diagnosed with haemoglobin electrophoresis, and results in gene deletion of one or both alpha chains.</p>
13. B	<p>ITP affects platelet aggregation. Antibodies attach the GpIIb/IIIa receptors preventing platelet aggregation</p> <p>Haemophilia A and B are deficiencies in factors 8 and 9, respectively.</p>

	TTP is the answer – the deficiency in ADAMTS13 enzyme causes the vWF to form thromboses. To compensate, the body will break down these clots down to prevent ischaemic damage to organs hence haemolytic anaemia. Raised bilirubin levels.
14. C	Option A and B are causes of relative polycythaemia Option D is a cause of primary absolute polycythaemia There are 2 types of secondary polycythaemia – compensatory and abnormal In hypoxic conditions, kidneys release high levels of EPO and hence more RBC are produced In abnormal conditions, it could be a tumour secreting EPO, GGWP
15. D	Myeloproliferative disorders are conditions affecting the myeloid pathway. Lymphomas affect the lymphoid pathway

16. D

Suspected Deep Vein Thrombosis (DVT): Pathogenesis and Complications

Notes:

- Venous thrombus causes pulmonary embolism, arterial thrombus causes stroke
- Previous DVT is risk factor for current DVT

Authors: Dean Percy, Yan Yu
Reviewers: Tristan Jones, Ryan Brenneis, Man-Chiu Poon*, Maitreyi Raman*
*MD at time of publication

Virchow's Triad:

- Hypercoagulable State:** ↑ ability for the blood to coagulate upon stimulation. Factors include:
 - Malignancy: Abnormal release of coagulation-promoting cytokines
 - Platelet Activation: Increased clot formation
 - Inherited Disorders: Congenital defect in coagulation (ie. Factor V Leiden, Factor II mutation, Protein S/C deficiency) → ↑ blood clotting ability
 - Trauma/Surgery: Systemic injury → activation of coagulation cascade
 - Estrogen promotes hypercoagulability, especially in presence of other risk factors
 - Pregnancy, Oral Contraceptives (OCP)
- Vessel Injury:** Exposes tissue factor on damaged cells and subendothelium for vWF binding. Factors include:
 - Hypertension: Physically damages blood vessel walls
 - Bacteria: Adhere/invade vessel wall
 - Artificial Valve: Abnormal surface
- Venous Stasis:** Low blood flow rate over site of vessel injury, concentrating blood clotting factors at that site. Factors include:
 - Obesity: Fat contains more aromatase, converts more androgens to estrogen; Sedentary lifestyle, poor venous return
 - Fracture, immobilization, bedrest, long vehicle/airplane ride: ↓ muscle motion = ↓ venous blood flow

Clot formation typically occurs in leg veins:

1. Deep, large veins allow for blood pooling (stasis, hypercoagulability)
2. Venous return from legs often against gravity (stasis)
3. Valves in leg veins prone to backflow (stasis)

Complications:

- Venous Insufficiency:** Destruction of vein valve by clot. Clot prevents blood from returning to heart. Blood accumulating in the leg results in unilateral leg edema and venous inflammation (redness, warmth, tenderness)
- Thromboembolus:** Clot embolizes to the lungs.
 - *Pulmonary embolism (acute life threatening complication)
 - Chronic thromboembolic pulmonary hypertension

Legend: Pathophysiology (blue), Mechanism (purple), Sign/Symptom/Lab Finding (green), Complications (red)

Re-Published September 1, 2019 on thecalgaryguide.com

Authors – Judith Scott, Olivia Stevens, Rebecca Nutt, Suhail Raihan

Editor – Chris Salmon csalmon3@sheffield.ac.uk