

# PTS 2a Mock SBA Series 2020

## *Paper 3- [Answers]- Version 3*



### **Marking Instructions:**

- Award **1 mark for each question** on the paper
- Multiple 'correct' answers may exist, a mark is awarded for the **single best answer**
- There are **100 marks** in total.
- There is **no identified 'pass mark'**.

### **Disclaimer:**

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Please **do not share** this document on **google drives** or **directly to future 2a students**, this takes away from their opportunity to complete the mock SBA in the run up to their exams when it has maximal impact as a revision resource. **This mock paper will be updated and repeated for future years.** Thank you.

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## Paper 1- Topics Assessed

<b>Cardiovascular (i)</b> 1. ACS 2. Heart Failure 3. Hypertension management 4. Aortic stenosis 5. Heart failure on CXR 6. Cardiovascular risk score 7. ACE-I side effects 8. Valvular disease investigation 9. Atrial fibrillation 10. Hypertrophic cardiomyopathy	<b>Haematology</b> 41. Anaemia- causes 42. Anaemia- treatment 43. Acute leukaemia- hx / ix 44. Chronic myeloid leukaemia 45. MGUS/Myeloma 46. Hodgkin's lymphoma 47. Myeloma hx / ix 48. Polycythaemia vera 49. DVT- risk factors 50. Anaemia	<b>Respiratory (ii)</b> 81. Asthma 82. Asthma 83. TB 84. Pneumonia 85. Pulmonary Embolism 86. TB 87. Sarcoidosis 88. Cystic Fibrosis 89. Hypersensitivity reactions 90. Goodpasture's syndrome
<b>Cardiovascular (ii)</b> 11. Hypertension- management 12. ECG- hyperkalaemia 13. Cardiomyopathy 14. Postural Hypotension 15. Cardiovascular risk factors 16. Septic shock 17. Hypovolaemic shock 18. Aortic Dissection 19. Pericarditis 20. Aortic Aneurysm	<b>Liver and Friends</b> 51. Chronic liver failure- signs 52. Acute liver failure 53. Acute pancreatitis 54. Pancreatic cancer 55. Liver cancer- risk factors 56. Wilson's disease 57. Alpha-1-antitripsin deficiency 58. Cholera 59. Rotavirus 60. Haemochromatosis	<b>Neurology</b> 91. Subarachnoid haemorrhage 92. Parkinson's disease 93. Cluster headaches 94. Seizure localisation 95. Meningitis histopathology 96. Stroke – vessel affected 97. TIA management 98. Cauda equina 99. Motor neurone disease 100. Guillain-Barre syndrome
<b>Endocrine</b> 21. Hyperthyroid Disease 22. Graves' Disease 23. Hypothyroid Disease 24. Acromegaly 25. Secondary Hypoadrenalism 26. Addison's Disease 27. SIADH 28. Carcinoid Syndrome 29. Hypocalcaemia 30. Hyperparathyroidism	<b>Microbiology</b> 61. Legionnaire's diagnosis 62. Meningitis in community 63. Identifying bacteria 64. UTI in pregnancy 65. Encephalitis management 66. Antibiotic mechanisms 67. Agars 68. Bacteria identification 69. Infective endocarditis 70. Identifying bacterial types	
<b>Gastrointestinal</b> 31. UC vs. CD. 32. Crohn's disease- diagnosis 33. Barrett's oesophagus 34. Duodenal ulcers- complications 35. Appendicitis- symptoms 36. Coeliac disease- symptoms 37. GORD- treatment 38. Small bowel obstruction 39. Oesophageal carcinoma 40. Ulcerative colitis- treatment	<b>Respiratory (i)</b> 71. Dyspnoea scale [COPD] 72. Lung cancer 73. Pleural effusion 74. Vasculitis [GPA] 75. ABG [Pneumonia] 76. A1AT deficiency 77. Occupational lung disorders 78. Spirometry [COPD] 79. Bronchiectasis 80. COPD management	

## Cardiovascular (1)

### **Question 1- Answer B- Fibrinolysis with IV Tenecteplase**

Patient has symptoms in keeping with acute coronary syndrome (acute onset central chest pain, sweating, vomiting). ECG shows STEMI (ST elevation myocardial infarction) with ST segment elevation in V1-V6 (anterolateral STEMI). Left anterior descending artery is the most commonly affected vessel causing ST elevation in anterior leads (V1-V4) and lateral leads (V5-V6, Lead I, AVL). Management of patients with STEMI is classically MONA (morphine for analgesia, Oxygen- but not given as standard only if hypoxaemic, Nitrates- GTN spray, Aspirin- antiplatelet treatment 300mg dose). Definitive management to resolve a STEMI is with PCI or fibrinolysis. PCI is first line for STEMI but must be done within 2 hours of onset, if this is not possible then fibrinolysis with IV Tenecteplase is done- this should be within 12 hours.

- (A)- CTPA is diagnostic investigation for suspected PE, performed in patients with Wells Score >4.
- (B)- Fibrinolysis with IV Tenecteplase performed within 12 hours of STEMI onset if patients cannot get PCI done (PCI must be done within 2 hours of onset and our patient presents to A&E after 4 hours)
- (C)- High flow oxygen is only indicated in patients with STEMI that are hypoxic, our patient has sats of 97% OA.
- (D)- PCI is first line treatment for STEMI if it can be performed within 2 hours of onset, otherwise it is fibrinolysis with IV Tenecteplase
- (E)- The patients observations show they are afebrile, infective cause is unlikely in this scenario and hence broad-spectrum antibiotics are not indicated.

### **Question 2- Answer C- NT-proBNP (BNP) levels.**

- (A) CXR shows signs of heart failure (ABCDE) but is not diagnostic and not first line
- (B) CT scan wouldn't be first line investigation for heart failure.
- (C) NICE guidelines recommend NT-proBNP for diagnosing heart failure.
- (D) ECG can show changes but not diagnostic.
- (E) Echocardiogram is gold standard but not a first line investigation.

### **Question 3- Answer C- Ramipril**

This patient has stage 1 hypertension. (stage 1.  $\geq 140/90$  mm Hg, stage 2.  $\geq 160/100$ , stage 3.  $\geq 180$  systolic or 120 diastolic.)

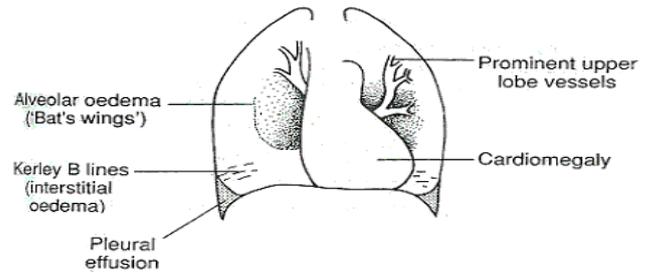
- (A) Bisoprolol is a beta-blocker, a rate limiting drug not used as first line treatment for HTN.
- (B) Furosemide is a loop diuretic not used for the treatment of HTN, thiazide like diuretics such as Bendroflumethiazide are second line options.
- (C) ACEi is first line treatment based on this patients' demographics.
- (D) Amlodipine is a dihydropyridine calcium-channel blocker first line for >55yrs or of Afro-Caribbean ethnicity.
- (E) Nifedipine is a dihydropyridine calcium-channel blocker first line for >55yrs or of Afro-Caribbean ethnicity.

### **Question 4- Answer E- Aortic Stenosis**

- (A) Patients with HF will only present with a murmur if there is also valvular disease, however there can be a gallop rhythm due to the presence of S3.
- (B) An early diastolic murmur is characteristic for aortic regurgitation
- (C) A pansystolic murmur is characteristic for mitral regurgitation.
- (D) A rumbling mid-diastolic murmur is characteristic for mitral stenosis.
- (E) An ejection-systolic murmur is characteristic for aortic stenosis.

**Question 5- Answer C- Dilated Bronchioles**

Chest Xray signs for heart failure are ABCDE (Alveolar oedema, Kerley B lines (interstitial oedema), Cardiomegaly, Dilated upper lobe vessels, pleural Effusion). Therefore C- dilated bronchioles is incorrect.



**Question 6- Answer B- QRisk3**

- (A) Cha2Ds2-VaSc= risk of stroke in AF.
- (B) QRisk3= 10-year probability of CV event.
- (C) ABCD2= risk of stroke after TIA.
- (D) Wells score= risk of DVT.
- (E) Modified Duke Criteria= Infective endocarditis criteria.

**Question 7- Answer D- Ramipril**

Most common side effect of ACEi (ramipril) is a dry cough due to increased levels of bradykinin. It is unlikely to be infective exacerbation of asthma / pneumonia given all observations and examinations are normal- these cases would likely be abnormal; you would expect at least patient to be pyrexical. Unlikely to be lung cancer due to lack of other symptoms (weight loss, SOB etc). Metformin does not cause dry cough.

**Question 8- Answer A- Echocardiogram**

- (A) Echocardiogram is the gold standard for diagnosing valvular disease.
- (B) ECG may show arrhythmias but not diagnostic.
- (C) Troponin- T I used to aid diagnosis of Acute Coronary Syndromes (ACS).
- (D) NT-proBNP levels used to diagnose heart failure.
- (E) CXR doesn't diagnose valvular disease but may show LA enlargement.

**Question 9- Answer B- Absent P waves**

- (A) Sawtooth flutter waves= seen in atrial flutter.
- (B) Absent P waves, narrow QRS + irregularly irregular ventricular rhythm are all ECG changes associated with AF.
- (C) Delta waves= Wolff-Parkinson-White syndrome.
- (D) Saddle-shaped ST segment elevation= acute pericarditis.
- (E) ST segment depression= sometimes seen in myocardial ischaemia.

**Question 10- Answer E- Hypertrophic cardiomyopathy**

Hypertrophic cardiomyopathy is most likely given that he's young and has a +ve family history of young cardiac death. Other diagnoses are unlikely due to his young age.

## Cardiovascular (iii)

### **Question 11- Answer E- Prescribe Ramipril in addition to Amlodipine**

*Hypertension management: patients under 55 should be started on ACEi/ARB, patients over 55 (like in this case- age 58) or afrocaribbean should be started on CCB. If hypertension persists then the 2<sup>nd</sup> line treatment is to combine the ACEi/ARB and the CCB. If hypertension persists add Thiazide-like diuretic. Therefore answer is (E) prescribe ramipril in addition to amlodipine.*

*Percutaneous Coronary Intervention (A) is used for the management of MI not HTN. Atenolol (B) is a beta blocker, rate-control drug with limited use in HTN management. Adding Candesartan and removing amlodipine is not appropriate as we need to combine ACEi/ARB and CCB for 2<sup>nd</sup> line not switch to the other. Prescribing Bendroflumethiazide (D) is the third line management not 2<sup>nd</sup>. ACEi example= Ramipril. ARB example= Candesartan. CCB example= Amlodipine. Thiazide-like diuretic example= Bendroflumethiazide.*

### **Question 12- Answer E- U waves**

*The presentation described Diabetic Ketoacidosis- patient with known T1DM with missed doses of insulin. Insulin normally transports glucose and potassium into cells therefore a lack of insulin causes hyperglycaemia and hyperkalaemia. As a result of the lack of glucose in cells the body starts to break down fat causing an increase in ketones. Ketones are acidic and hence cause metabolic acidosis, they can be smelt on patients breath as a sweet 'pear-drop' smell. Patients in DKA often take deep, fast breaths called Kussmaul breaths- this is an attempt to 'blow off' CO<sub>2</sub> to try and reverse the acidosis. It is important to recognise the ECG signs of hyperkalaemia as it can cause arrhythmias and subsequent death. Hyperkalaemia= Absent P, Long PR interval, Wide QRS, Tall Tented T waves (Go, Go long, Go wide, Go tall= Gonner). Hypokalaemia= U waves*

### **Question 13- Answer D- Dilated**

*Cardiomyopathy types: dilated, restrictive, hypertrophic. Cardiomyopathy is generally congenital and can also be idiopathic. Answers A,B,C,E are not types of cardiomyopathy, they are ways of describing potential ways in which cardiomyopathy may be acquired.*

### **Question 14- Answer C- Lying 137/103, standing 109/88**

*Lying and standing blood pressure is carried out by getting the patient to lie down for 5 minutes then measure BP. Ask them to stand up and at 1 minute and 3 minutes measure their BP. If there is a systolic drop >20mmHg or if the systolic BP drops to less than 90 (from any original starting point) the patient is diagnosed with postural hypotension. Management includes lifestyle advice such as maintaining good hydration, regular exercise, salty meals. Pharmacological management includes medications such as fludrocortisone 1<sup>st</sup>, midrodine 2<sup>nd</sup>. Note that normally HR and BP should rise when going from lying to standing, in this scenario only patient A elicited a normal response.*

*(A) Normal lying and standing BP response- 118/82 then increased to 138/90*

*(B) Narrow pulse pressure- 120/110 seen in aortic stenosis*

*(C) Postural hypotension- 137/103 to 109/88 because 137-109= drop of 28mmHg.*

*(D) Stage 1 hypertension- 147/99 (BP>140/90= stage 1)*

*(E) Wide pulse pressure- 150/102 seen in aortic regurgitation*

**Question 15- Answer B- Smoke 20 cigarettes a day and has done for the last 10 years**

*(A) Cardiac family history is only applicable if it occurred before the age of 55. (B) Smoking is relevant as it is a significant risk factor for MI, his 10 year pack history is likely to play a role in his current presentation. (C) Is irrelevant to his current presentation of chest pain given it was decades ago. (D) Thyroid disorders are also important when taking a history but out of the answers smoking is most relevant to the type of symptoms. (E) An allergy to penicillin is also very important, but the question asks specifically about making a diagnosis.*

**Question 16- Answer C- Bounding Pulse**

*(A)- septic shock is caused by wide-spread infection in the blood therefore the patient will be pyrexia. (B)- bradycardia -a classical sign of cardiogenic shock- septic shock most likely to be tachycardic. (D)- reduced airway entry- anaphylactic shock because of swelling of the airways. (E) Paraesthesia isn't a common feature of any type of shock. Therefore, the answer is C- bounding pulse.*

**Question 17- Answer E- Pulmonary Embolism.**

*PE does not cause any form of fluid/blood loss.*

*Hypovolemic shock is caused by blood/ fluid loss. (A) GI bleeding causes loss of blood. (B) severe diarrhoea/vomiting would cause loss of fluid. (C) burns cause loss of fluid. (D) pancreatitis is a known cause of hypovolaemic shock.*

**Question 18- Answer A- Aortic Dissection**

*Aortic dissection typically is described as having a 'tearing/shearing' pain which goes to the back. Note that this can also be a symptom described in aortic aneurysm rupture- a medical emergency. (B) MI typically presents with central crushing chest pain. (C) Cardiac tamponade is caused by build up of fluid in the pericardial cavity but does not typically cause pain. (D) pulmonary embolism presents with pleuritic chest pain, SOB and haemoptysis. (E) infective exacerbation of COPD would likely present with respiratory symptoms such as SOB and productive cough.*

**Question 19- Answer C- pleuritic chest pain**

*Pericarditis presents with pleuritic, sharp chest pain which classically is worse on lying down and relieved by leaning forwards. (A) is typical of MI. (B) is typical of acute pancreatitis. (E) haemoptysis is generally respiratory e.g TB, lung CA, PE.*

**Question 20- Answer E- Ultrasound Scan**

*From the history of sudden onset epigastric pain and her vital signs indicating shock one diagnosis to work to exclude is a ruptured abdominal aortic aneurysm (AAA). This is done by performing a rapid USS of the aorta, if confirmed it requires immediate surgical repair.*

**Endocrine**

**Question 21- Answer B- polyuria**

*Answers A, C, D and E are symptoms of hyperthyroid disease. A good way to remember the symptoms of this is that everything in hyper speeds up, whilst in hypo everything slows down. B- polyuria is not a symptom of hyperthyroid instead it's a symptom of Diabetes*

**Question 22- Answer B- Grave's disease**

*Graves' Disease is the most common cause of hyperthyroidism. This is an autoimmune condition in which the body produces TSH receptor stimulating antibodies. It causes the typical presentation of Graves' ophthalmopathy, diplopia, eye pain and other hyperthyroid symptoms. De Quervain's thyroiditis is a transient condition due to a viral infection. You get no eye involvement. In peri-orbital cellulitis the orbit would be red, painful and sore and would be systemically unwell. Thyroid cancer would not typically present with eye symptoms. Thyrotoxicosis is when you get an overproduction in thyroid hormones and get non eye related symptoms: palpitations, delirium, hyperpyrexia etc.*

**Question 23- Answer A- High TSH, low T3/T4**

*This patient's presentation is indicative hypothyroid disease by their fatigue, weight gain and low mood. indicated . Primary hypothyroidism results show a low T3/T4 and a high TSH (pituitary is trying to respond to negative feedback of low t3/t4 by increasing release of TSH. B is a completely made up blood result. C- low TSH, High T3/T4 indicates primary hyperthyroidism. D demonstrates a patient who has pituitary failure (Low TSH released from pituitary hence low T3/T4, this would also be a sensible answer however the patient is more likely to present with a broad spectrum of signs and symptoms caused by a lack of other pituitary hormone release not just hypothyroid symptoms).E: demonstrates a Euthyroid 'normal'.*

**Question 24- Answer B- Oral Glucose Tolerance Test**

*Oral glucose tolerance test (B) is the gold standard test for acromegaly. IGF-1 (C) is a good indicator of GH levels but not gold standard. MRI of pituitary (D) may identify a pituitary tumour as the cause but independently would not diagnose Acromegaly- the tumour could be causing Cushing's not acromegaly. GH levels (E) are not a reliable test as they fluctuate during the day.*

**Question 25- Answer C- Long term corticosteroid usage**

*Long term corticosteroid use is the number one cause of secondary hypoadrenalism. This condition is more common than Addison's. Other causes include cessation of corticosteroid treatment and disorders of the pituitary (such as surgery to it). A and E are causes of Addison's disease*

**Question 26- Answer D- Synacthen test**

*This patient has Addison's disease (primary hypoadrenalism) an autoimmune condition when the entire adrenal cortex is destroyed. Reduced cortisol, aldosterone and sex hormones cause a range of symptoms such as postural hypotension in this case. Hyper pigmentation is due to increased ACTH. The correct test is D this gives an infusion of ACTH.*

**Question 27- Answer B- Dehydration**

*Dehydration is not a cause of SIADH. Alcohol withdrawal, head injury, pneumonia and small cell lung cancer are potential causes of SIADH. SIADH is a condition of inappropriate ADH release. This causes the plasma to become too dilute. It leads to water retention, excess blood volume and hyponatraemia. The patient will have a low serum Na<sup>+</sup>, high urine Na<sup>+</sup>. Management is through restricting fluid intake and use of ADH inhibitors (demeclocycline)*

**Question 28- Answer E- Somatostatin analogue**

*Carcinoid Syndrome occurs as a result of a tumour of enterochromaffin cells which secrete 5-HT. They are most commonly found in the terminal ileum or appendix. Once there is hepatic involvement it's called carcinoid syndrome. Excess secretion of substance P, insulin, serotonin, ACTH and bradykinin causes there to be a range of symptoms from hyperglycaemia to bronchoconstriction. The classical triad of carcinoid syndrome is palpitations, diarrhoea and flushing. If someone is in a crisis due to carcinoid syndrome give them a somatostatin analogue and do surgery.*

**Question 29- Answer B- Hypocalcaemia**

*B is the correct answer. Common symptoms of hypocalcaemia are numbness and tingling in the extremities. The case describes Trousseau's Sign: wrist flexion following the inflation of a BP cuff. You can also get Chvostek's Sign: tapping the facial nerve in the parotid gland causes ipsilateral facial muscle twitching. In Acromegaly obstructive sleep apnoea would be common.*

**Question 30- Answer A- High PTH, High Calcium, Low Phosphate**

*This patient has primary Hypercalcaemia (bones, stones, groans and psychiatric moans).*

	PTH	Calcium	Phosphate	Alk Phos
Primary	High	High	Low	High
Secondary	High	Low	High	High
Tertiary	High	High	High	High

**Gastrointestinal****Question 31- Answer D- Non-continuous 'skip lesions'**

*(A)(B) UC presents as continuous inflammation extending from rectum to ileo-caecal valve. This is circumferential meaning it covers the circumference of the bowel*

*(C) UC inflammation stops at the ileo-caecal valve, which is the point between the small intestine becomes the large intestine*

*(E) The mucosa becomes inflamed, red and is susceptible to bleeding in UC*

**Question 32- Answer B- Colonoscopy + Mucosal biopsy**

*Colonoscopy and biopsy are essential for diagnosis of Crohn's or Ulcerative colitis. Histology can be assessed + specific features can be identified*

*(A) Abdo XR- allows exclusion of colonic dilatation, will not show specific signs of Crohn's disease. Histological analysis of tissue is essential*

*(C) Faecal calprotectin is a non-specific marker of inflammation in bowel, will not differentiate between ulcerative colitis + Crohn's disease.*

*(D) PR exam- allow clinician to examine for anal strictures, haemorrhoids or perianal abscesses. Will not allow for diagnosis of Crohn's disease, but useful if patient has blood in stools*

*(E) Stool MC&S- useful investigation to exclude infectious gastroenteritis, will not show whether PT has Crohn's disease*

**Question 33- Answer E- Stratified squamous → simple columnar epithelium**

*Pre-malignant change seen in lower part of oesophagus, primarily caused by chronic acid exposure to simple columnar epithelium of oesophagus*

**Question 34- Answer A- Gastroduodenal artery**

*Gastroduodenal artery: terminal branch of common hepatic artery and supplies the proximal duodenum. Runs posterior to proximal duodenum, so ulceration into posterior wall + into this artery can cause major bleeds*

*(B) Left gastric artery: branch of coeliac trunk than runs to the left and away from the duodenum*

*(C) Short gastric artery: supplies superior part of stomach, not in close proximity to duodenum*

*(D) Superior mesenteric artery: arises directly from abdominal aorta and supplies organs of midgut, passes anteriorly to distal duodenum*

*(E) Superior pancreaticoduodenal artery: arises from gastroduodenal artery after it passes posterior to the duodenum*

**Question 35- Answer C- Peri-umbilical region**

*Visceral pain is poorly localised and so inflammation of appendix (right iliac region) is referred to umbilical region. Only when the inflammation becomes more serious and touches the parietal peritoneum does the pain localise to McBurney's point/right iliac region*

*(B) McBurney's point- specific point of pain seen in PTs with appendicitis after inflammation has reached parietal peritoneum. Pain shifts here from umbilical region. Point is located 2/3 from umbilicus to ASIS*

**Question 36- Answer C- Loss of appetite**

*Loss of appetite is not a typical symptom of coeliac disease, there isn't a direct loss of appetite. Patients have weight loss but this isn't due to decreased appetite, it's due to malabsorption*

*(A) Angular stomatitis: soreness at corners of lips, seen in severe cases of coeliac disease*

*(B) Aphthous ulcers: mouth ulcers, commonly seen in more severe cases*

*(D) Steatorrhea: classic symptom of coeliac disease, stinking/fatty/loose stools*

*(E) Unintentional weight loss: malabsorption due to villous atrophy*

**Question 37- Answer C- Antibiotics**

*Antibiotics are not used to alleviate GORD. They are used for infections or commonly as part of triple therapy management of H.pylori peptic ulcers (PPI + Metronidazole + Clarithromycin)*

*(A) Alginates: form a physical barrier and work by forming neutral floating gel raft on top of the stomach to prevent acids from backing up into the oesophagus*

*(B) Antacids: work by neutralising the HCL in the stomach*

*(C) Histamine receptor antagonists: histamine binds to H2 receptors of parietal cells to trigger acid production, if these receptors have antagonists bound, histamine cannot bind meaning less acid is produced*

*(D) Proton-pump inhibitors: inhibit gastric acid secretion by blocking H<sup>+</sup>/K<sup>+</sup> ATPase enzyme*

**Question 38- Answer D- Surgical Adhesions**

*Surgical adhesions: scar-like tissue that form between organs, these can compress the small intestine, these are the most common cause of small bowel obstruction*

- (A) Malignant tumours- most common cause of large bowel obstruction, can also cause small bowel obstruction but not the most common cause*
- (B) Meckel's diverticulum: congenital disorder resulting in outpouching of small intestine, can cause obstruction, not most common cause as only present in 2% of population*
- (C) Strictures from Crohn's disease: can also cause small bowel obstruction*
- (E) Volvulus: loop of bowel twists round itself, one of the main causes of large bowel obstruction*

**Question 39 – Answer A- Enlarged Virchow's node**

*Enlarged Virchow's node (Trosier's sign)- enlargement of left supraclavicular node commonly associated with gastric cancer, NOT oesophageal cancer*

- (B) Lymphadenopathy- enlargement of lymph nodes is a sign of many malignancies, could indicate metastasis to lymph tissues*
- (C) Progressive dysphagia- classic symptom of oesophageal carcinoma. Initially PT has difficulty swallowing solids, but dysphagia for liquids follows as tumour grows*
- (D) Retrosternal chest pain- may be experienced by PTs with oesophageal carcinoma, epigastric pain more associated with gastric cancer*
- (E) Weight loss- common symptom observed in most malignancies*

**Question 40- Answer C- Mesalazine**

*Mesalazine- member of 5-ASA drug group that is used for mild UC*

- (A) Colectomy- definitive surgical removal of colon, only used when severe UC + other treatments exhausted*
- (B) IV Hydrocortisone- corticosteroids used for severe UC*
- (D) NSAIDs e.g. Ibuprofen- NSAIDs tends to aggravate the GI tract, do not use in treatment of UC*
- (E) Oral prednisolone- corticosteroid used for moderate UC*

**Haematology**

**Question 41- Answer D- Chronic Disease**

*Chronic disease is classically associated with Normocytic or Microcytic anaemia. The rest impair meiosis and cell division, hence the cells that are produced are larger than they would ordinarily be i.e. macrocytic cells.*

**Question 42- Answer D- Oral iron supplements**

*The most common form of anaemia is Iron deficiency anaemia, a microcytic anaemia. (D) Iron supplements are the initial treatment for iron deficiency anaemia. (A) Blood transfusion is a treatment for Iron-deficiency anaemia, but it is not the 1<sup>st</sup> line treatment. (B) Folic acid is the treatment for macrocytic anaemia to stimulate cell division. (C)Hydroxycarbamide is generally used as prophylaxis for sickle cell crises. (E) Pyridoxine is the treatment for Sideroblastic anaemia*

**Question 43- Answer B- Acute myeloid leukaemia**

*The history suggests a more acute course, making the chronic leukaemia's less likely but not impossible (C)(D). They will, however, tend to present more insidiously. Hodgkin's Lymphoma (E) will typically present with a painless lump and a white cell count that is not normally raised. The difficulty is distinguishing between ALL (A) and AML (B). ALL is typically a disease of children whereas AML is the most common leukaemia of adults which would make you lean towards AML in this case as the lady is the perfect age for AML. The 2 diseases are differentiated by the presence of Auer rods in blast cells on bone marrow biopsy.*

**Question 44- Answer B- Philadelphia chromosome**

*Philadelphia chromosomes are characteristic in cytogenetics of CML (B). Bite cells (A) are a characteristic finding in G6PD deficiency, a haemolytic disorder. Roth spots (C) are signs found in the eyes in Infective endocarditis. Schistocytes (D) are cell fragments found post haemolysis which is not a feature of Leukaemia's. Xanthoma (E) are yellowy fatty spots which develop under the skin, commonly in liver disease.*

**Question 45- Answer C- Myeloma/multiple myeloma**

*MGUS is a precursor condition which has a 1% chance per year of developing into myeloma. It causes no end organ damage and has no symptoms. It is characterised by overproduction of a monoclonal immunoglobulin, generally found incidentally on blood tests. It has no relation to any of the other conditions.*

**Question 46- Answer D- Raised white cell count**

*In Hodgkin's Lymphoma white cells are not typically raised. Pruritis can occasionally be the only presenting symptom in Hodgkin's lymphoma. Reed-Sternberg cells are diagnostic if found on a blood film. It is counter intuitive that the WCC is not raised and it can be easy to be caught out.*

**Question 47- Answer E- Multiple myeloma**

*Myeloma symptoms can be remembered by the acronym CRAB – Calcium (raised due to bone resorption), Renal impairment (creatinine high), Anaemia and Bone (osteoporosis). The above history and investigations are indicative in derangement of all of these. In addition, Bence Jones proteins in the urine and rouleaux formation in the blood are characteristic of Myeloma. While advanced Bronchial Ca could have metastasised into various organs to produce the spread of symptoms and investigative results we have here, it would be very advanced and the patient would be significantly more unwell with a very severe cough likely with haemoptysis. Acute lymphoblastic leukaemia presents typically with an anaemia/infection/bleeding picture and tends to develop more rapidly and do not typically cause renal impairment or calcium derangement until later in their disease course. CML tends to be much slower in bone erosion, and more typically causes fatigue, sweats and potentially gout over a chronic course. Follicular lymphoma is also a very slow developing (indolent) cancer which does not fit the presented symptoms or investigations.*

**Question 48- Answer D- Weight loss**

*Weight loss (D) is not a known complication or feature of PCV. PCV is a condition in which the bone marrow over produces blood cells caused in 95% of cases by a JAK2 mutation. Dizziness (A) occurs because the blood is overly viscous causing various CNS abnormalities. Itching (B) occurs because the abnormal numbers of RBC's stimulate histamine. Haemorrhage (C) can occur due to defective platelet function.*

**Question 49- Answer E- Tay-sachs disease**

*You almost certainly will not know what Tay-Sachs disease is and you're not meant to. It is a very rare neuro-degenerative disease of children. The point is that you should know that all of the other options are major risk factors for DVT – immobility, trauma and surgery because they all impair the body's ability to move the blood from the legs back to the heart and malignancy because of cell wall damage in the blood vessels.*

**Question 50- Answer B- Heiz Bodies**

*Heinz bodies are seen in G6PD deficiency not in IDA. The RBC's will be pale due to insufficient haemoglobin and small for the same reason. They will also form in abnormal shapes as the normal structure requires a set amount of haemoglobin. The cells will vary wildly in size depending on the store of iron in the bone marrow which created them.*

**Liver and Friends**

**Question 51- Answer D- Kussmaul Breathing**

*Kussmaul breathing is a deep, laboured breathing you may observe in patients with diabetic ketoacidosis but not chronic liver failure. It is a form of hyperventilation meant to get rid of carbon dioxide in the blood.*

*(A) Asterix is a jerking movement of the hand that occurs when the arms are outstretched and wrists extended (cocked back towards the face), it is more commonly known as liver flap and can be seen in liver or type 2 respiratory failure. (B) Spider Naevi is a type of telangiectasia caused by dilated arterioles and capillaries as a result of increased oestrogen levels in the blood. This increased oestrogen level also causes gynaecomastia (C), usually seen in liver failure as the liver cannot break down oestrogen. Liver failure causes jaundice as the liver cannot break down bilirubin and there is mild biliary congestion as well.*

**Question 52- Answer C- Low albumin levels**

*According to the European Association for the Study of the Liver any of the definitions are applicable with the exception of low albumin levels.*

**Question 53- Answer A- Acute pancreatitis**

*(A) This presentation is typical of acute pancreatitis- key clues are abdominal pain which radiates to the back, severity of pain, periumbilical bruising (Cullen's sign) and flank bruising (Grey Turner's sign). (C) Pancreatic cancer usually presents with no pain except in later stages where it impinges on other structures. There is no history of smoking, previous stroke, hypertension or AF which make ischaemic colitis/ruptured AAA (B)(E) less likely (ruptured AAA would also be very sudden onset). (D) A perforated peptic ulcer presents very similarly to acute pancreatitis although it wouldn't explain the periumbilical and flank bruising which is indicative of retroperitoneal haemorrhage.*

**Question 54- Answer B- Cancer of the pancreatic head**

*Painless jaundice is a red flag, especially in a 72-year old man. Jaundice there is something wrong with the hepatobiliary system. The fact that his stool and urine colour has been affected suggests that this is an obstructive jaundice which rules out Chronic Liver Failure and Hepatocellular carcinoma although you can get colour changes although less likely. Pancreatitis is not correct as he is not in any pain. In theory, a cholangiocarcinoma is possible although it is rare in the Western world. A cancer in the head of pancreas can obstruct the Common Bile Duct, causing obstructive jaundice and is therefore the correct answer.*

**Question 55- Answer D- Benzene exposure**

*Benzene exposure is a risk factor for Renal cell carcinoma and therefore, it is the correct answer. Hepatitis B and chronic alcohol use are both big risk factors for liver cancer because of the associated cirrhosis risk. Non-alcoholic fatty liver disease can also cause cirrhosis, although it is less common and also causes liver cancer. Aflatoxin is a type of toxin produced by fungi such as Aspergillus and can cause liver cancer.*

**Question 56 answer D- Liver biopsy**

*Ferritin levels just indicate inflammation and is not specific for Wilson's. The other blood tests are indeed tests for Wilson's and would be used first-line but they are not always fool-proof. Serum caeruloplasmin can be elevated in other forms of hepatitis such as autoimmune hepatitis and 15 – 50% of Wilson's Disease patients will have normal serum caeruloplasmin levels. Similarly, patients with severe liver failure can have raised 24hr urinary copper levels. A diagnosis of Wilson's can get obtained from a liver biopsy.*

**Question 57- Answer C- Autosomal recessive**

*The mode of inheritance of  $\alpha$ 1-antitrypsin deficiency is autosomal recessive.*

**Question 58- Answer B- Rehydration with IV fluids**

*This patient most likely has cholera characterised by "rice-water stools". The most appropriate management for patients with profuse diarrhoea due to cholera is rehydration with intravenous fluids (B). Rehydration with intravenous fluids is favoured here as the patient is tachycardic and therefore signs of hypovolaemia. Oral rehydration solutions (A) are favoured in developing countries and recommended by the WHO. Metronidazole (C) is not used in the treatment of cholera. However, ciprofloxacin is occasionally used. Codeine phosphate (D) and oral azithromycin (E) are not used in the treatment of profuse diarrhoea due to cholera.*

**Question 59- Answer C- Rotavirus**

*It is reassuring that the boy does not have bloody diarrhoea so that rules out (E) Campylobacter jejuni which causes bloody diarrhoea. (A) Adenovirus cause respiratory infections and is not correct. (D) Noroviruses are commonly found in nursing homes among the elderly. E.coli is a close answer but rotaviruses are more likely to be transmitted among children and is therefore the correct answer*

**Question 60- Answer B- Haemochromatosis**

*Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism which results in iron accumulation. Features include fatigue, erectile dysfunction, arthralgia, bronze skin, diabetes mellitus, chronic liver disease signs, cardiac failure. Investigations include having a raised transferrin saturation, high ferritin and low TIBC. Mx is with venesection 1st and desferrioxamine 2<sup>nd</sup>. Addison's (C) also causes hyperpigmentation and fatigue however the other symptoms such as ED and signs of liver failure are not in keeping with this diagnosis. Renal cell carcinoma (D) does not present with skin changes. Wilson's disease (A) is caused by increased copper can cause jaundice but does not cause bronzing. T2DM only explains the polyuria but none of the other symptoms.*

**Microbiology**

**Question 61- Answer C- Legionella pneumophila**

This is a typical presentation of Legionnaire’s disease, an often severe form of pneumonia caused of Legionella pneumophila. It is found in natural / artificial water sources and unclean air conditioning units. (Med school love this question and its always Spain)

Chlamydomphila psittacia is another atypical pneumonia not indicated by the history. Contact with birds is characteristic.

E. coli flora bacteria, most common cause of UTI. Not a cause of pneumonia.

TB has a longer natural history (months) and you expect to elicit a contact / travel to high risk country. Contact tracing needed in this case and long-term treatment with RIPE antibiotic regime.

Strep. pneumonia is a typical pneumonia, not suggestive from this history.

**Question 62- Answer B- Benzylpenicillin**

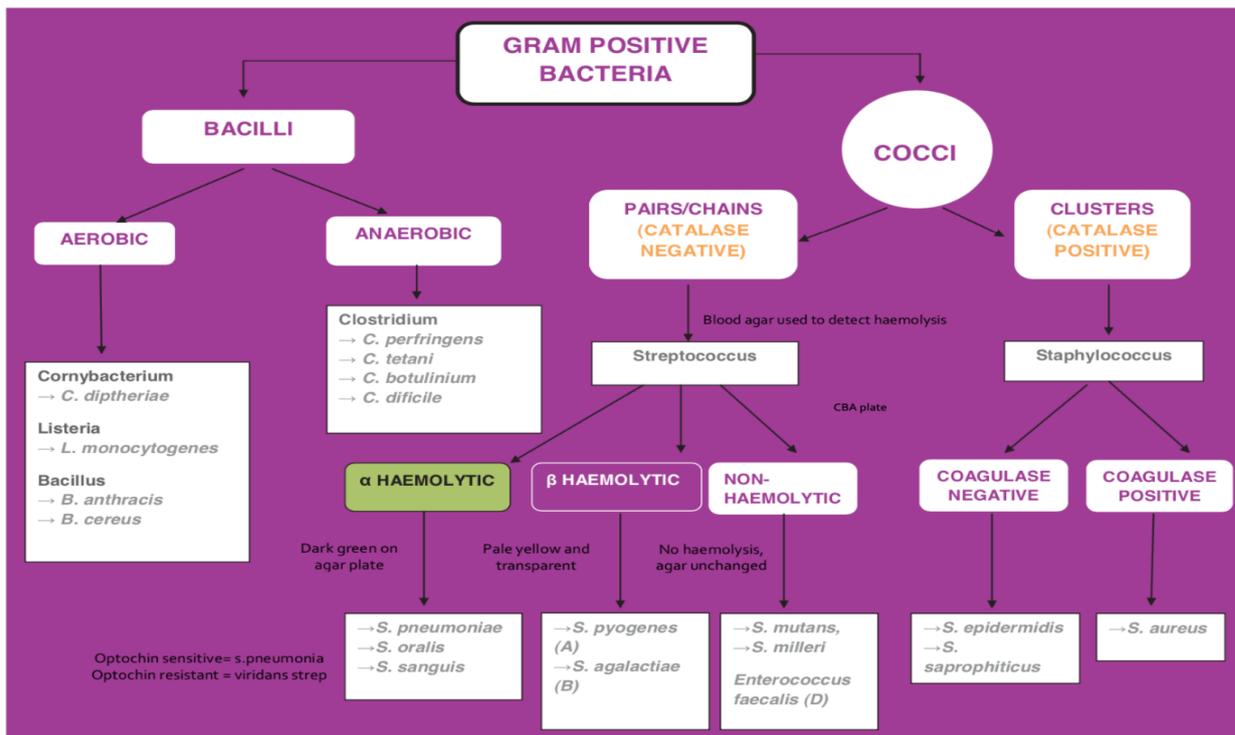
Benpen IM is given immediately to patients with suspected meningococcal septicaemia. This is indicated by the non-blanching petechiae and septic appearance of the child.

Cefotaxime IV (3<sup>rd</sup> gen. cephalosporin) is used in hospital settings to treat meningeal infections but is not available in the community.

Amoxicillin and erythromycin are commonly used in children to treat milder infections such as respiratory tract infections.

Doxycycline is a tetracycline used for a variety of infections as well as severe acne but not in this case.

**Question 63- Answer D- Strep Pyogenes**



**Question 64- Answer D- Nitrofurantoin**

This woman has symptoms of a UTI, the most common causative organism is E. coli. This is normally treated by trimethoprim which inhibits folate synthesis; however, it is teratogenic therefore contraindicated in this case. Nitrofurantoin is the most appropriate treatment for pregnant women with UTI.

**Question 65- Answer A- Acyclovir**

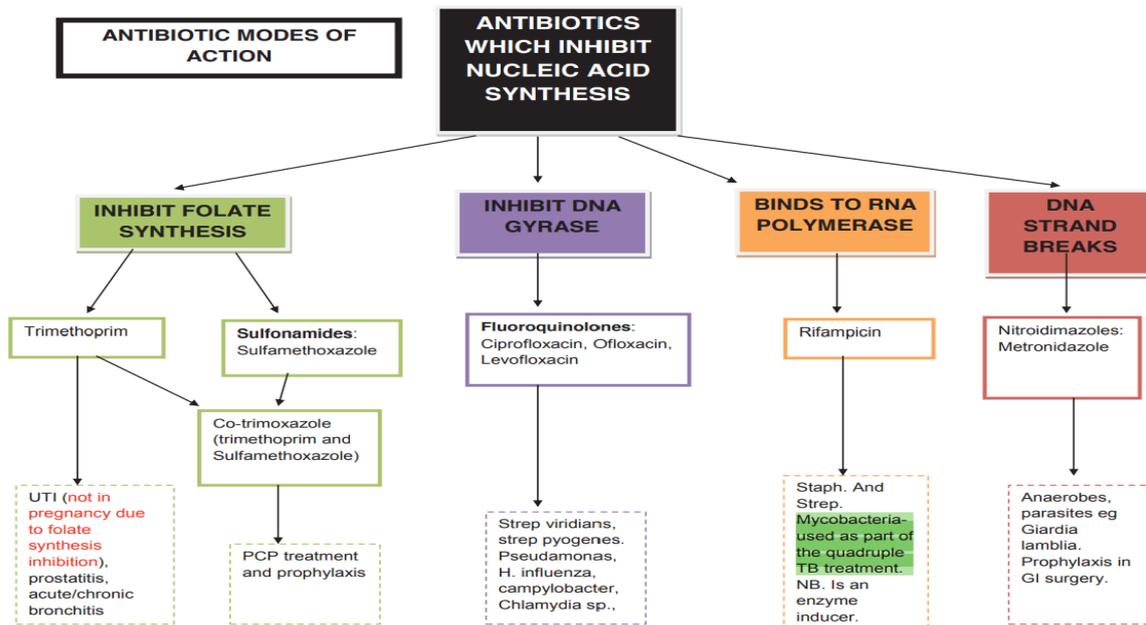
*This patient has encephalitis caused by herpes simplex virus. The classical triad of this infection is fever, altered mental state and headache. It can often present with flu like symptoms and meningisms. IM Benpen should be given in the community as for suspected meningitis or meningococcal septicaemia. In hospital give acyclovir.*

*The role of steroids in the treatment of encephalitis is not established.*

*3<sup>rd</sup> generation cephalosporins are used for bacterial infections such as bacterial meningitis.*

*Broad spec. antibiotics such as metronidazole and vancomycin can be used to treat concurrent bacterial infections but do not treat HSV encephalitis.*

**Question 66- Answer B- Chloramphenicol**



**Question 67- Answer D- Lowenstein-Jensen**

*Lowenstein-Jensen is used to culture mycobacterium tuberculosis*

*Blood agar is used to culture anaerobes such as fusobacteria.*

*Charcoal agar is used to grow campylobacter jejuni.*

*Chocolate agar is used to grow aerobes such as strep. pneumonia.*

*MacConkey is used to grow gram negative bacilli*

**Question 68- Answer C- Pseudomonas**

*Pseudomonas is non-lactose fermenting aerobic bacilli with positive oxidase test*

*C diff. (A) is an anaerobic bacillus. Proteus (B), salmonella (D) and shigella (E) are all non-lactose fermenting aerobic bacilli with negative oxidase test.*

**Question 69- Answer D- Optochin resistant alpha-haemolytic**

*The most common cause of infective endocarditis in adults is strep. viridans. This is optochin resistant alpha-haemolytic strep. Optochin sensitive alpha-haemolytic streptococcus is strep. pneumonia*

*Lancefield A = strep. pyogenes (strep. throat / scarlet fever)*

*Lancefield B = strep. agalactiae (group B strep – colonised vaginal canal, causes really bad neonatal infections such as neonatal meningitis)*

*Non-haemolytic = s. mutans, s. milleri*

**Question 70- Answer A- Catalase**

*Catalase +ve = staph. catalase –ve = strep.*

*Coagulase +ve = staph. aureus, coagulase –ve staph. epidermis*

*Optochin sensitive = strep. pneumonia, optochin resistant = strep. viridans*

*Oxidase +ve = pseudomonas, oxidase –ve = coliforms (shigella, salmonella, proteus)*

*Haemolysis = alpha / beta haemolytic streps.*

**Respiratory (1)**

**Question 71- Answer D- Grade 4**

*This MRC scale is commonly used in General Practice and respiratory medicine. I would develop a good idea of most of the commonly used scales/scores ( MRC dyspnoea, NYHA scale, GCS, CURB-65).*

*It is easily examined and described below: Grade 1: Breathless with strenuous exercise. Grade 2: Short of breath when hurrying or when walking up hill. Grade 3: Walks slower than people of the same age or stops for breath when walking at own pace on flat. Grade 4: Stops for breath after walking 100m on flat. Grade 5: Too breathless to leave the house/ Breathlessness on changing clothes.*

**Question 72- Answer A- CXR**

*The stem of the question features a red flag (unexplained weight loss). It is useful to through each system and know a list of red flags e.g. in resp they are [ weight loss, haemoptysis, night sweats etc].*

*The red flag points towards a cancer of the lung, especially in the absence of a temperature which helps eliminate infective causes. The best FIRST LINE test would be a chest x-ray. A CT can come later for staging. Answers c,d,e provide little diagnostic benefit when suspecting a lung cancer.*

**Question 73- Answer E- Parapneumonic pleural effusion**

*Pleural effusions commonly occur after a pneumonia 'Parapneumonic pleural effusion'. Pleural effusions will present with stony dull percussion {buzz word for pleural effusions}. They will also result in the CXR findings mentioned in the question stem. Bronchiectasis and COPD (A and B) are more chronic, this question describes an acute picture. C- a lung abscess you would expect the individual to be more systemically unwell (fever, night sweats etc) and hence is less likely. D- A lung cancer is possible and can also cause a pleural effusion however given the recent history of a pneumonia and absence of red flags the parapneumonic effusion is most likely. NOTE: underline/ highlight key aspects of the question and if you are stuck eliminate by process of elimination.*

**Question 74- Answer D- C-ANCA**

*D is the correct answer, C-ANCA is associated with granulomatosis with polyangitis. A- ANA is common in many conditions and isn't very specific. B, Anti- DsDNA is most commonly seen in SLE. C, Anti-CCP is the most specific antibody for rheumatoid (more so than rheumatoid factor). E, P-ANCA is associated with Churg-Strauss.*

**Question 75- Answer E- T2RF**

*Type 2 respiratory failure is the correct answer as oxygen is low and carbon dioxide is high. Remember type 2= both affected. Type 1 is a normal CO<sub>2</sub> with a low O<sub>2</sub>. Answers a, b, c can derange an ABG and an ABG may be used to assist with their diagnosis and management however and ABG alone isn't diagnostic can't show them or diagnose them alone. It is worth re-reading the question which is asking which best describes the ABG rather than the most likely diagnosis. ABG's come up and it may be useful to have a basic understanding of metabolic/ respiratory acidosis and alkalosis.*

**Question 76- Answer A- Autosomal Dominant**

Von-Willebrand factor is used in clotting, Von-Willebrand disease is an autosomal dominant condition whereby you lack VWF and therefore have an increased tendency to bleed. Having 1 dominant gene for VWD makes you a sufferer, having both gives you a very severe form of VWD. This can present in a number of ways, one of which is an increased likelihood of haemoptysis as in this case. Autosomal recessive (B) example= cystic fibrosis. Translocation of Ch9 and Ch22 (C) manifests in Philadelphia chromosome- Chronic myeloid leukaemia. X-linked dominant (D) example= fragile X-syndrome. X-linked recessive (E) example= haemophilia A.

**Question 77- Answer E- Silicosis**

*The correct answer is silicosis. Pneumoconioses are a group of chronic lung conditions due to exposure of mineral dust or metal such as silicosis, asbestosis, coal workers lung. These all fall under occupational lung disorders. We can also get Extrinsic allergic alveolitis which is a hypersensitivity reaction to organic dust such as: bird fanciers lung, farmers lung; these also fall under occupational lung disorders. It is useful to know the definitions of pneumoconiosis and EAA and what they encompass to understand the conditions. The rest of the answers are not occupational. A- Atelectasis, is lung collapse. B, Bronchiectasis is permanent dilatation of the airways due to chronic lung disease or infection. C is idiopathic, meaning cause is unknown and therefore isn't occupational. D, Sarcoidosis- not an occupational lung disorder, key finding on investigation is bilateral hilar lymphadenopathy.*

**Question 78- Answer B- COPD**

*The patient hx and spirometry indicates an obstructive defect (FEV<sub>1</sub>/FVC<0.7). Hence, everything on the list apart from asthma and COPD can be eliminated. The lack of reversibility (as well as patient's age) points towards COPD as the correct answer. Restrictive conditions such as fibrosis (D) and sarcoidosis (E) will have a normal FEV<sub>1</sub>/FVC ratio above 0.7 but have a lower independent FVC.*

**Question 79- Answer D- Left Ventricular Failure**

*Left ventricular failure (D) is not a cause of bronchiectasis. Bronchiectasis= chronic infection leading to permanent dilatation of the airways. Bronchogenic carcinoma, cystic fibrosis and immotile ciliary syndrome (aka kartagner's syndrome) all predispose the airways to infection and hence can cause bronchiectasis. Pneumonia is an infection of the airway and pneumonias can cause bronchiectasis too. The main infective organisms involved in bronchiectasis include: Pseudomonas aeruginosa, Haemophilus influenzae, Staph aureus, Strep Pneumoniae .*

**Question 80- Answer A- Add LABA**

Follow link to BNF for full outline of COPD management, beneath is a brief summary <https://bnf.nice.org.uk/treatment-summary/chronic-obstructive-pulmonary-disease.html>  
In this patient case as we have not yet confirmed whether the patient is steroid responsive or not we should add in a LABA as this treatment is indicated in both. When steroid responsiveness has been confirmed you can then add the subsequent medication.

First thing to do is always stop smoking and vaccinate (influenza and pneumococcal)

- 1) SABA / SAMA
- 2) \*If steroid responsive/asthmatic = Add LABA + ICS
- 2) \*If not steroid responsive / non-asthmatic= Add LABA + LAMA
- 3) Oral theophylline
- 4) Long term oxygen therapy

SABA= Short-acting beta agonist, LABA= Long-acting beta agonist,

SAMA= short-acting muscarinic antagonist, LAMA= long-acting muscarinic antagonist (Note: do not prescribe together- if started on LAMA, remove SAMA)

ICS= Inhaled corticosteroid

**Respiratory (iii)**

**Question 81- Answer E- Fruit and Vegetable rich diet**

A fruit and vegetable rich diet is said to be protective in asthma. D- there is a high risk of asthma attacks in those who are anxious. C. aspirin in particular can trigger asthma attacks, Beta blockers have parasympathetic effect which result in airway narrowing, airflow limitation and can precipitate a potential asthma attack.

**Question 82- Answer C- Beclometasone**

NICE guidance says prescribe an inhaled corticosteroid (ICS) as preventer therapy for all people who:

- Use an inhaled SABA three times a week or more, and/or
- Have asthma symptoms three times a week or more, and/or
- Are woken at night by asthma symptoms once weekly or more.

Ipratropium- antimuscarinic, Methotrexate- DMARD, Beclometasone- inhaled corticosteroid, Salmeterol- Long acting beta agonist, Tiotropium- antimuscarinic

**Question 83- Answer E- Tuberculosis**

The answer is TB because of the history: most coughs which persist over 3 weeks are TB or cancer. Her recent travel history makes it more likely that this is TB. Although cancer is a good differential diagnosis- she isn't very old and the travel history makes it less likely. D is incorrect as there is no mention in the history of any risk factors for PE or mention of chest pain.

**Question 84- Answer E- Streptococcus Pneumoniae**

Streptococcus pneumoniae is the most common organism in community acquired pneumonia (CAP). D is an organism involved in hospital acquired pneumoniae (HAP). The other answers are all causative agents of CAP but not the most common. E.coli is found in CAP and HAP. A is an atypical agent.

**Question 85- Answer A- High testosterone**

High testosterone is not said to be a risk factor for PE, although oestrogen and HRT are a risk factor. Obesity is a risk factor as it changes blood flow. Smoking induces changes in the vessel to increase risk. Polycythaemia changes the blood constituents and makes it thicker. A recent leg fracture is an indication of immobility which allows for stasis of the blood and thrombotic formation.

**Question 86- Answer E- Rifampicin**

Note that B, C, D and E (RIPE) are all the antibiotics in the treatment of TB, side effects of each:  
 Rifampicin- red urine, hepatitis, drug interaction ('R'-red urine)  
 Isoniazid – hepatitis, neuropathy  
 Pyrazinamide- hepatitis, arthralgia/gout, rash  
 Ethambutol- optic neuritis

**Question 87- Answer E- Tissue Biopsy**

A tissue biopsy is diagnostic for Sarcoidosis- showing a non-caseating granuloma.  
 A bronchoalveolar lavage will show a)increased lymphocytes in active disease b)increased neutrophils if pulmonary fibrosis present but is not diagnostic.  
 Blood tests are good for assessing the extrapulmonary manifestations of the condition.  
 Chest X-ray is used for staging.  
 Lung function tests give an indication of the effects of the condition and how it is affecting the patient.

**Question 88- Answer B- Cystic Fibrosis**

The answer is CF. Although it is unlikely someone would present with CF this late- this question is to get you to consider the extrapulmonary manifestations of CF.  
 Due to pancreatic insufficiency (the mucus blocking the ducts), people with CF often suffer with steatorrhea due to being unable to digest fats. Males also suffer from infertility due to atrophy of the vas deferens and epididymis.

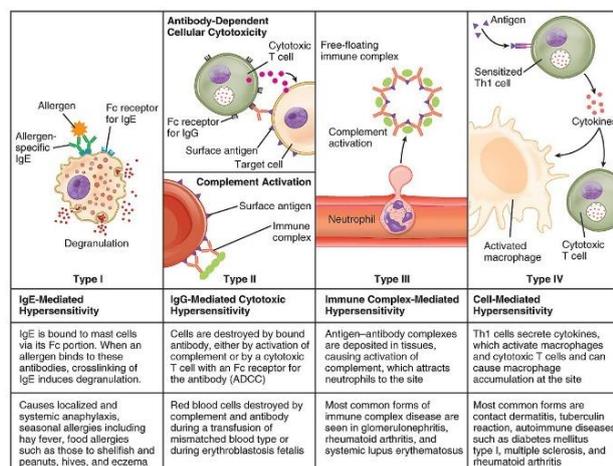
**Question 89- Answer C- Type 3**

C is the correct answer, it is a type 3 hypersensitivity reaction. The allergic response to the inhaled antigen involves both cellular immunity and the deposition of immune complexes (TYPE 3 HYPERSENSITIVITY). Some of the inhaled antigen may directly activate the alternate complement pathway. Both these mechanisms attract alveolar and interstitial macrophages. Prolonged exposure to the antigen eventually leads to pulmonary fibrosis.

**Hypersensitivity Reactions - Types** 

Mnemonic: "ACID"

Hypersensitivity Reaction	Description
<b>Type I</b> IgE-mediated; quick onset after exposure Allergic	Bee stings Latex Certain medications (e.g. Penicillin)
<b>Type II</b> Cytotoxic/antibody-mediated Cytotoxic	Hemolytic reactions Goodpasture syndrome Hyperacute graft rejection
<b>Type III</b> Immune complex/IgG/IgM mediated Immune complex deposition	Hypersensitivity pneumonitis Systemic lupus erythematosus Polyarteritis nodosa Serum sickness
<b>Type IV</b> Delayed or cell-mediated Delayed	Chronic graft rejections PPD test Latex Nickel Poison ivy



**Question 90- Answer C- Goodpasture's syndrome**

*GPS is common in that age group and is more common in men. There is often a prequel of symptoms like an upper respiratory tract infection. This is then superseded by symptoms of diffuse pulmonary haemorrhage (the haemoptysis) and glomerulitis (bloody, frothy urine). D, B and E are differential diagnoses of GPS.*

*B is more common in young children and has less renal involvement.*

**Neurology**

**Question 91- Answer D- Subarachnoid Haemorrhage**

- (A) Extradural haemorrhage. Typically caused by trauma to temple → tear in middle meningeal artery. History: brief loss of consciousness, followed by lucid period, before rapid deterioration with headache / vomiting / confusion / focal neurology. CT head: lens-shaped / lentiform / bi-convex haematoma.*
- (B) Ischaemic stroke. Not typical history – hemiplegic weakness. Hypertension is, however, a risk factor for both ischaemic and haemorrhagic stroke. CT scan: signs of ischaemia (loss of grey-white matter differentiation)*
- (C) Migraine. Classic Migraine History= POUND = Pulsating, Onset over 4-72 hrs, Unilateral, Neurological signs (e.g. photophobia). Disabling (patients often want to be in a dark room)*
- (D) Subarachnoid haemorrhage. Headache in SAH is classically: Sudden onset – ‘thunderclap’, Severe, Occipital, accompanied by signs of meningism (caused by blood irritating the meninges)e.g. Vomiting, Photophobia, Neck stiffness. Note that berry aneurysm rupture is commonest cause of SAH- this can be caused by PKD. Investigating SAH, note that if CT was normal and the suspicion of SAH was high, a lumbar puncture could be performed after 12 hrs, looking for xanthochromia.*
- (E) Subdural haemorrhage. More common in elderly / alcoholics / those on anti-coagulation or anti-platelets. History typically more chronic – headache / fluctuating consciousness / cognitive decline. CT head: sickle-shaped / crescent-shaped haematoma.*

**Question 92- Answer C- Bradykinesia, Rigidity, Tremor**

*Bradykinesia: slow to initiate movements / slow, low-amplitude movements. Tremor: resting, coarse/pill-rolling  
Parkinsonian gait: shuffling, reduced arm swing, festinant. Other signs of Parkinson's: slow blink rate, micrographia, expressionless face*

**Question 93- Answer B- Cluster Headache**

- (A) *Brain tumour- Less likely to be acute onset. Increased ICP in brain tumours - headache worse by bending / straining + vomiting. No accompanying focal neurology (signs specific to brain areas – e.g. temporal lobe epilepsy)*
- (B) *Cluster headache- Epidemiology: male, smoking, 30-50 yo. Circadian periodicity (tend to occur at same time each day – classically early hrs of morning). Acute management= high-flow 100% oxygen + subcutaneous sumatriptan. Preventative management= verapamil*
- (C) *Migraine- Classic Migraine History= POUND = Pulsating, Onset over 4-72 hrs, Unilateral, Neurological signs (e.g. photophobia). Disabling (patients often want to be in a dark room)*
- (D) *Tension headache- ‘Band-like’, +/- scalp tenderness / neck muscle stiffness or tightness*
- (E) *Trigeminal neuralgia – unilateral, severe paroxysms of electric shock-like / shooting pains. Limited to one/more divisions of trigeminal nerve (ophthalmic, maxillary, mandibular). No focal neurology.*

	<i>Tension-type</i>		<i>Migraine</i>		<i>Cluster</i>	
<i>Pain location</i>	Bilateral		Unilateral (or bilateral)		Unilateral Around the eye	
<i>Pain quality</i>	Pressing / tightening (non-pulsating)		Pulsating		Variable (sharp / boring / burning / throbbing / tightening)	
<i>Pain intensity</i>	Mild - moderate		Moderate - severe		Severe	
<i>Effect on activities</i>	Not aggravated by ADLs		Aggravated by, or causes avoidance of, ADLs		Restless / agitated	
<i>Other symptoms</i>	-		Unusual sensitivity to light and / or sound Nausea and / or vomiting <b>Aura</b> Symptoms can occur with / without headache and: <ul style="list-style-type: none"> <li>• are fully reversible</li> <li>• develop over at least 5 minutes</li> <li>• last 5-60 minutes</li> </ul>		Ipsilateral to headache: <ul style="list-style-type: none"> <li>• Red and / or watery eye</li> <li>• Nasal congestion and / or runny nose</li> <li>• Swollen eyelid</li> <li>• Forehead and facial sweating</li> <li>• Constricted pupil and / or drooping eyelid</li> </ul>	
<i>Duration of headache</i>	30 minutes – continuous		4-72 hours		15-180 minutes	
<i>Frequency of headache</i>	<15 days per month	15+ days per months for >3 months	<15 days per month	15+ days per months for >3 months	1 every other day – 8 per day, with remission >1 month	1 every other day – 8 per day, with a continuous remission for <1 month in a 12-month period
<i>Diagnosis</i>	Episodic tension- type headache	Chronic tension- type headache	Episodic migraine	Chronic migraine	Episodic cluster headache	Chronic cluster headache

**Question 94- Answer E- Temporal Lobe Seizure**

- (A) Frontal lobe seizure- Seizures in the frontal lobe may cause ‘Jacksonian motor seizures’ (Proximal spread of clonic jerking- begins in finger / toe / corner of mouth, spreads proximally as abnormal epileptic discharge moves along motor cortex). Post-ictal Todd’s paralysis- Affected limb(s) may remain temporarily weak
- (B) Occipital lobe seizure=typically cause floaters / flashes in the eyes.
- (C) Parietal lobe seizure- typically cause non-specific sensory symptoms (tingling / pain / numbness / prickling)
- (D) Psychogenic non-epileptic attack- Complex (but probably more common than people think). No abnormal electrical brain activity. Factors favouring: female, pelvic thrusting, hx of childhood physical/sexual abuse, PTSD, depression.
- (E) Temporal lobe seizure:
  - > Pre-seizure aura- rising epigastric sensation, fear / anger, déjà vu, hallucinations - olfactory / gustatory
  - > Seizure- Automatism: lip smacking, chewing, grimacing, fidgeting, picking at clothes
  - > Post-ictal confusion= common

Location	Typical seizure type
Temporal lobe (HEAD)	Hallucinations (auditory/gustatory/olfactory) Epigastric rising / Emotional Automatism (lip smacking/grabbing/plucking) Déjà vu / Dysphasia post-ictal
Frontal lobe (motor)	Head/leg movements Posturing Post-ictal weakness Jacksonian march
Parietal lobe (sensory)	Paraesthesia
Occipital lobe (visual)	Floaters/flashes

**Question 95- Answer E- Streptococcus Pneumoniae**

- (A) Coxsackie B virus- causes an aseptic meningitis – negative culture / staining. Viral meningitis tends to be less serious – i.e. not likely to have reduced GCS (drowsiness)
- (B) Haemophilus influenzae type B- gram-negative coccobacillus – catalase and oxidase positive. Note it is less common cause due to HiB inclusion in UK vaccination schedule
- (C) Herpes simplex virus- more commonly a cause of encephalitis, detected on PCR not gram stain.
- (D) Neisseria meningitidis-gram negative diplococcus, ‘reddish-pink’
- (E) Streptococcus pneumoniae- gram positive diplococcus, ‘blue’, causes severe meningitis

**Question 96- Answer C- Left Middle Cerebral Artery**

- (A) Basilar artery- produces ‘locked-in’ syndrome
- (B) Left anterior cerebral artery- right-sided hemiparesis / sensory loss, lower extremity affected > upper
- (C) Left middle cerebral artery- right-sided hemiparesis / sensory loss, upper extremity affected > upper. UMN facial weakness – forehead sparing. Right-sided homonymous hemianopia. Dysphasia (in this case, receptive) – caused by the fact that the dominant lobe (left) is affected
- (D) Right anterior cerebral artery- same as b) but left-sided symptoms.
- (E) Right middle cerebral artery- same as c) but left-sided symptoms (no dysphasia as right=non-dominant lobe)

**Question 97- Answer A- Aspirin 300mg daily**

- (A) Aspirin 300mg daily: NICE guidelines state that if a patient presents within 7 days of suspected TIA: aspirin 300mg daily – immediately, specialist review within 24 hrs, advise Ptx not to drive. Note that if >7 days, as above but with specialist review within 7 days
- (B) Aspirin 300mg + clopidogrel daily- 1<sup>st</sup> line long-term anti-thrombotic therapy in TIA (as in stroke) is clopidogrel monotherapy. This is given after specialist review has been conducted + TIA confirmed (rather than suspected, in this case)
- (C) Clopidogrel daily- see b)
- (D) Dipyridamole daily- aspirin + dipyridamole dual therapy is 2<sup>nd</sup> line long-term anti-thrombotic therapy if clopidogrel is not tolerated.
- (E) Warfarin daily- warfarin increases the risk of stroke within 30 days of ischaemic symptoms

**Question 98- Answer B- Cauda Equina Syndrome**

- (A) Brown-Sequard syndrome (lateral hemisection of the spinal cord). Features: ipsilateral weakness below lesion, ipsilateral loss of proprioception + vibration, contralateral loss of pain + temperature.
- (B) Cauda equina syndrome- note that cauda equina begins at L1/2 – level at which the spinal cord terminates. LMM signs only. Lower back pain, LMN weakness (decreased power and reflexes), sphincter disturbance (urinary hesitancy / impaired sensation of flow / retention, foecal incontinence). Impotence, saddle paraesthesia, decreased anal tone.
- (C) Conus medullaris syndrome- similar to cauda equina but UMN features.
- (D) Spinal cord compression at T10- mixture of UMN + LMN features. LMN at level of lesion (T10) → sensory level (umbilicus). UMN below level of lesion → UMN (spastic) weakness + ↑ reflexes. Note you get UMN + LMN here because the spinal cord has not yet terminated – in cauda equina, it has
- (E) Syringomyelia = central cord syndrome. Weakness upper limb affected > lower limb. ‘Cape-like’ ↓ pain / temperature caused by spinothalamic tract damage. Normal proprioception / vibration

	CONUS MEDULLARIS SYNDROME	CAUDA EQUINA SYNDROME
Presentation	Sudden and bilateral	Gradual and unilateral
Reflexes	Diminished- at the level Brisk- below the level	Diminished
Radicular pain	-	+
Low back pain	More	Less
Impotence	Frequent	Absent
Numbness	Symmetrical	Asymmetrical
Motor strength	Symmetric Hyperreflexic Distal paresis of lower limbs	Asymmetric Areflexia Paraplegia
Sphincter dysfunction	Present early Both urinary and fecal incontinence	Present later Only urinary retention

**Question 99- Answer A- Motor Neurone Disease**

(A) Motor neurone disease

- i. 3 broad categories – based on UMN / LMN features:
  - i. Amyotrophic lateral sclerosis UMN **and** LMN – most common
  - ii. Primary lateral sclerosis UMN only
  - iii. Progressive muscular atrophy LMN only
- ii. In this case, we have a combination of UMN + LMN signs – note that we have UMN signs **above** LMN signs (in terms of spinal level)
  - i. For example:
    - 1. Brisk jaw jerk (UMN – innervated by trigeminal nerve)  
'above'
    - 2. 1<sup>st</sup> dorsal interosseus wasting (LMN – innervated by ulnar nerve – C8-T1)
  - ii. If the pathology was from the spinal cord, remember that we would have LMN features at the level of the lesion, with UMN features **below** the level only.
- iii. Progressive symptoms over months, Bulbar symptoms – difficulties with speech / swallowing. Normal sensory examination.

(B) Multiple sclerosis- more likely to begin in younger women (20-40 yo)

- i. Defined by discrete episodes disseminated in:
  - i. Time multiple events – with recovery in between episodes
  - ii. Place neuroanatomically – optic neuritis / arm numbness / ED
- ii. 'Characteristic' clinical features absent in this case e.g. Lhermitte's phenomenon – neck flexion → electric shock-like sensation in arms / legs. Uhthoff's phenomenon – worsening of symptoms with ↑ temperature
- iii. UMN predominance in MS e.g. spastic weakness in the legs (spinal cord involvement)

(C) Myasthenia gravis more likely to be in younger women (~90% are <40 yo at diagnosis)

- i. ↑ muscle fatigue on sustained / repeated activity
  - i. Extra-ocular **ptosis / diplopia**
  - ii. Bulbar dysarthria / dysphonia / dysphagia / **difficulty chewing**
  - iii. Face myasthenic snarl when smiling
- ii. **NB:** reflexes are normal as not 'enough time' for muscles to fatigue

(D) Parkinson's disease- cardinal signs bradykinesia, rigidity, tremor

(E) Spinal cord compression - LMN signs at level of lesion, UMN signs below level of lesion

**Question 100 -Answer D- Guillain-Barre Syndrome**

- (A) *Bacterial meningitis- clinical features would be more indicative of infection (headache / meningism / etc.). LP= ↓ glucose + ↑ neutrophils*
- (B) *Charcot-Marie Tooth- genetic disorder → LMN signs + deformity – more chronic process than this. LP= no CSF change.*
- (C) *Chronic inflammatory demyelinating polyneuropathy- Acquired immune-mediated inflammatory disorder of the peripheral nervous system. CIDP is more chronic (vs. GBS)*
- (D) *Guillain-Barre syndrome- immune-mediated demyelination of the PNS classically 1-3 post infection with campylobacter jejuni (food poisoning). classic presentation = symptoms that begin peripherally-rapid, progressive onset muscle weakness, sensory symptoms – paraesthesia / pain, absence/reduced reflexes. LP= isolated increased protein.*
- (E) *Myasthenia gravis- Younger women (~90% are <40 yo at diagnosis). (see answer 9c for more). Note that reflexes are normal as not ‘enough time’ for muscles to fatigue. LP= no change in CSF. No sensory symptoms.*

**Google Form Scores/Feedback**

- **Record your scores-** this enables us to calculate an average mark for the mock and gauge the difficulty of the paper as a whole. Note that this data is anonymous. No identifying information will be released.
- **Inform us of mistakes-** from spelling mistakes, to incorrect explanations let us know where we’ve gone wrong so we can change it.
- **Ask for more clarification-** maybe you want a clearer explanation of the difference between 2 answers or more justification for the single best answer, ask us and we’ll get on it.

PAPER 3 Google Form- <https://forms.gle/mna7eDjWDMJfshzq6>

**I hope you found the Mock useful! Thanks for taking part!**

**Andrew Maud  
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