Pathology tutorials for Year 5 medical students

Please note the lifts to the 11th floor are slow, so please aim to get in

15 minutes before you are meant to start, as each table lasts 20

minutes, so you are likely to miss a chunk of information if you are late.

SESSION PLAN

- · Each teaching session lasts 1 hour 40 mins
- The session comprises 5 consecutive stations each lasting 20 mins.
- · 8 students at each of the five station

Station 1: Liver disease (20 minutes)
Station 2: Bowel (20 minutes)
Station 3: Heart (20 minutes)
Station 4: Neurology (20 minutes)
Station 5: Lung (20 minutes)

PLEASE NOTE THAT PHOTOGRAPHY OF THE SPECIMENS IS STRICTLY PROHIBITED

Station 1: Liver (20 minutes)

Case 1:

This 70-year-old patient presented with breathlessness with a raised JVP and ankle oedema and died a week later. His heart showed a previous myocardial infarction, but this is his liver which shows the features of heart failure. This is a reminder of what you were shown in October 2022.

Q1: What is the blood supply and draining of the liver?

Specimens:

56.M3816.0

Q2: Explain the distribution of the change in colour of the liver parenchyma in this patient.

Case 2:

This 52 year old arrives in casualty vomiting large amounts of blood. Despite massive blood transfusion and intensive care, his blood loss was too great to keep up with, and he dies shortly after arrival in hospital. He is found to have had chronic hepatitis B.

Q3. What can you see in this specimen?

56.M4855.0 56.M4855.1

Q4. Explain how one gets portal hypertension, and why he bled to death.

Station 2: BOWEL (20 mins)

Case history

A 65 year old man presents to his GP after noticing bright red blood in the pan after passing a stool. On questioning he describes a change in bowel habits over the last 2-3 months, with increasing occurrences of diarrhoea, and feeling tired.

What are the possible diagnoses?

1. A mass can be felt during digital examination. What would you do?

Look at the two bowel specimens and compare them.

Specimens: 67.M8140.0

68.M8143.2

What symptoms would you expect this specimen to have caused?

What can you see in this liver? 56.M8146.0

2. What is the usual staging method for this condition?

3. What are the treatment options?

Case history 2

A 63 year old man presents to his GP with loft lower abdominal quadrant pain and a fever. He'd previously presented twice in the last 12 months, with abdominal cramping and constipation that had been diagnosed as irritable bowel syndrome.

1. What are your thoughts about the presentation? Why? Specimens 67.M4641.1

67.M4641.0

2. What complications might occur, based on this appearance?

3. What is the standard treatment?

Station 3: HEART (20 mins)

Specimens:

Case 1:

This patient with a past history of diabetes had a myocardial infarction, but didn't have much pain and thus, did not seek medical attention. Why might this have happened? How do they thus present to hospital?

32.M5491.0 32.M5470.0

How does diabetes alter the pain sensation?

Case 2:

This patient developed severe central crushing chest pain and collapsed on the way to hospital, and was pronounced dead on arrival in hospital. What is the likely cause of death.

32.M3700.M5472.0 = shows Coronary Disease (with a recent infarct) -

Case 3:

34.M4201.5 = Infective endocarditis following previous rheumatic fever in childhood.

Clinical hist:

The patient was a female aged 32. Eleven years previously (aged 21) she had rheumatic fever, puerperal fever a year later (age 22), and pneumonia with pleurisy (age 25) three years subsequently. She had been dyspnoeic on exertion for ten years and recently this had become worse. Two months before admission, she developed a fever, and was noted to have splinter haemorrhages on her fingernails, and in the left hypochondrium, a mass could be felt. On examination the pulse was irregular in force and rhythm and the heart was dilated to the right side of the sternum. The first sound at the apex was replaced by a loud systolic murmur conducted into the axilla and preceded by a mid-diastolic murmur.

What is rheumatic fever?

What is puerperal fever?

What is the likely valvular problem causing these murmurs?

RhF: Strep sore throat (age 21) results in anti-streptolysin O antibodies that cause autoimmune carditis, arthritis and Sydenham's chorea as well as erythema marginatum in children, and in the longer term, the carditis causes mixed mitral valve disease.

Puerperal sepsis is post partum sepsis. This used to be a leading cause of maternal death.

What happened two months before death?

How do we now treat damaged valves?

Case 4 F008 forensic IV drug abuser with a similar problem with infective endocarditis, but this time caused by intravenous injection of staphylococcus from contaminated needles.

Case 5:

34.M3211.0 and 34.M3211.1 = Prosthesis

Station 4: Neurology: 20 mins

Case 1: A 25 year old male was admitted unconscious having collapsed some hours previously. He was semi-conscious with head deviated to the left, left cranial nerve palsy, brisk LEFT sided reflexes with increased tone on the left side. BP 170/120 and he died the same day.

X1.M3850.9 = what can you see in this specimen?

Questions:

What does the fact that the patient has brisk left sided reflexes suggest?

What can cause this, and what is the differential diagnosis?

Case 2:

Clinical history:

The patient, a 50 year old female, had rheumatic fever in childhood, followed by rheumatic carditis. Eight months before her death she developed subacute bacterial endocarditis which responded to penicillin. Later she developed palpitations and was found to be fibrillating. She was treated for a month at home with digitalis, without effect, but on admission to hospital she responded to quinidine. Two days later; however, she suddenly developed dysarthria and left hemiplegia. Despite heparin therapy she became comatose, began to fibrillate again and died.

What has happened?

Case 3

Clinical History:

The patient, a 52 year old female, had a history of mitral stenosis following rheumatism. Two and half years before her death she had a 'stroke' followed by left sided hemiplegia from which she made a considerable degree of recovery. A fortnight before death she as admitted with severe pains in the back and legs and was found to be fibrillating. The left leg became gangrenous and required amputation, but she died a few days later.

What can you see in the specimens for cases 2 and 3? X2.M3710.0 X2.M3710.3

What do we do now to prevent this from happening. Make sure you know the CHADS VASC scoring system. Case 4:

X2.M9443.2 and X2.M9443.3

Clinical history:

The patient, a 63 year old female, presented with left sided homonymous hemianopia of one month's duration. She also had frontal headache, and mild mental deterioration had been noted by others. These symptoms increased over the next 5 weeks until she had developed a left hemiparesis.

Examination revealed brisk reflexes on the left hand side of his arms and legs. She was admitted with signs of greatly raised intracranial pressure, too ill to stand operation.

Question: Are these upper or lower motor neurone signs?

What clue is there in the history that this is not a stroke?.

Station 5: LUNG (20 mins)

This is a section of a lung of a young child who is known to have cystic fibrosis. What lung pathology does this show? (see <u>specimen</u> 28.M3412.1, 28.M3412.2).

Explain the pathogenesis of this condition.

Bronchiectasis: Dilated pus-filled bronchi are visible, especially near the apex. There is also patchy consolidation, indicating a bronchopneumonia (that was terminal in this instance).

What organism is likely to cause problems for this patient?

Cases 2 onwards:

These patients all developed lobar pneumonia, and died after different periods of illness. What is the commonest organism that causes lobar pneumonia?

28.M4102.1 = Lobar pneumonia throughout lower lobe (with red "hepatisation")

28.4102.0 (previouslyM50/76): lobar pneumonias (with grey "hepatisation")

This other patient has a different appearance in his lungs with pneumonia. What organism causes this distribution of pneumonia?

28.M4000.E1600.0 (K0321)= Staphylococcal pneumonia or klebsiella for bronchpneumonia

What is the main approach to treatment? What happens without treatment?

- **<u>1.</u>** Lobar pneumonia (e.g.**E1600** <u>323</u>): the contiguous air spaces of part or all of a lobe are homogeneously filled with an exudate that can be visualised on radiographs as a lobar or segmental consolidation.
- <u>2.</u> Bronchopneumonia (e.g. <u>K0321</u>): implies a patchy distribution of inflammation that often involves more than one lobe. This pattern occurs from an initial infection of the bronchi/bronchioles, which then extends down to the adjacent alveoli. However, despite the anatomical differences, the differences between lobar and bronchopneumonia are blurry, as many organisms can cause either pattern, plus confluent bronchopneumonia can be difficult to distinguish form lobar pneumonia. As a result, pneumonias tend to be classified based on their causative organism.
- **2.** The pathological stages of lobar pneumonia:red "hepatisation", grey "hepatisation", and resolution (or death).
 - a) Congestion: the affected lobe is heavy and red, with vascular congestion (hyperaemia) and many bacteria in alveoli
 - **b)** Red "hepatisation": the lobe has a liver-like consistency, the alveolar spaces are packed with neutrophils, red cells and fibrin
 - c) Grey "hepatisation": the lung is dry, grey and firm, as the red cells are lysed. Fibrinosuppurative exudate persists within the alveoli. (28.4102 M50/76)
 - d) Resolution: in uncomplicated cases, the exudate is enzymatically digested to produce debris, which is resorbed by macrophages, coughed up, or organised by fibroblasts.