Histopathology Training Schools

Bristol Block Week

September 2019

Post Mortem Histology Seminar

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***Case Histories and Microscopic Objectives***

You have some illustrations of each slide as a guide as to some of the things you should look for. Towards the end of the session there will be a presentation of the microscopic features and some background clinicopathological information. You are welcome to copy the slide shows onto a key but please be discrete about their use.

***Case 1 Female 82*** Collapsed suddenly whilst drinking tea with a friend. She was healthy for her age but a little forgetful. You have a slide of her myocardium.She died from a haemopericardium following a ruptured myocardial infarct. There was a fresh coronary thrombosis.

In the section of myocardium identify the site of the rupture and look at the surrounding myocardium. There are heavy infiltrates of polymorphs and areas of myocardial necrosis. These features indicate established myocardial infarction. There was no clinical history of chest pain but many myocardial infarcts are “silent”, especially in the elderly.

***Case 2 Male 46***This patient had longstanding cirrhosis of the liver and type 1 diabetes. He had a TIPSS procedure a week before his sudden death. A Coroner’s autopsy was required in order to exclude complications related the shunt. There were no complications but there appeared to be a coronary thrombosis. There was advanced cirrhosis with ascites. The photographs illustrate some of the features of micronodular cirrhosis. Note the dense bands of connective tissue surrounding regenerating nodules of hepatocytes. Identify the marked fatty change and the bile duct proliferation.

The second slide is a transverse section of the coronary artery which appeared to be thrombosed. Identify the large haemorrhage into the plaque below the fibrous cap of the lesion.. Note that the lumen is patent but narrowed by the haemorrhage. Atheromatous lesions contain many small blood vessels and these frequently bleed, leading to deposition of a brown pigment, haemosiderin, a form of iron. Identify this pigment.

Whenever you can do take a section of the critical coronary artery stenosis at post mortem. Not only does this provide objective evidence of the narrowing it allows, as in this case, an accurate diagnosis.

***Case 3 Male 57*** The patient owned a garage and had no significant past medical history. He was woken at 12.30 am with chest pain. This lasted 30 minutes but then subsided. Severe pain returned an hour later, an ambulance was called. He arrived in A & E 2 hours 30 minutes after the onset of chest pain. ECG showed an inferior STEMI and he was thrombolysed. Two days later he had coronary angiography and two stents were inserted. He recovered well and was discharged on day 6. Four days later he collapsed suddenly at home. A post mortem was ordered largely to exclude complications related to treatment.

The section of myocardium was taken through the area of infarction. A macroscopic photograph of this will be shown in the concluding talk. Identify the residual cardiac myocytes. There are many spindle shaped cells. These are myofibroblasts, cells that contain contractile filaments and which can also secrete collagen. They play a key role in wound healing. There are also many small capillaries. In a healing wound the combination of capillaries and myofibroblasts is so called “granulation tissue”

The patient was thought to have died of a ventricular arrhythmia. These are most common in the first month after a myocardial infarction. Little can be done to prevent them. The patient had developed severe pulmonary oedema, a common feature in acute cardiac failure. Note that the alveolar spaces in the section of lung contain copious eosinophilic fluid. Eosin binds to the small amounts of protein in the transudated fluid. There is some atmospheric (anthracotic) pigment in the lungs.

***Case 4 Male 58*** The patient had longstanding type 2 diabetes and severe renal impairment. He was receiving optimal medical therapy for congestive cardiac failure but was NYHA grade 3. He died suddenly. A post mortem was performed largely because he had not seen his doctor for a month. There was severe hepatic congestion and pleural effusions. These are the two most specific signs of cardiac failure at post mortem. However making a diagnosis of heart failure at post mortem can be very difficult.

The section of liver shows the typical changes of hepatic congestion (nutmeg liver). Identify sinusoids close to the terminal hepatic venule which are dilated with red cells. The changes are less pronounced in the areas around the portal tracts. Identify small bile ducts within the portal tracts. There is some lymphocytic inflammation in portal tracts. Also look for the fine droplets of fat within some hepatocytes. The fatty change is not as florid as in the cirrhotic liver from case 2. It is sometimes called microvesicular change.

***Case 5 Male 79*** This patient died suddenly while walking home from the post office. He had a large pulmonary embolus but also evidence of established pulmonary infarction. There were no known risk factors. The patient was tall but not obese. He was moderately active. No tumours were identified at post mortem. The spleen weighed 500g, raising the possibility of a myeloproliferative disorder. However there were no recent haematology results and coagulation studies and platelet counts cannot be performed on post mortem blood.

In the section of lung distinguish the infarcted and non infarcted segments of lung. They are sharply circumscribed. The infarcted tissue has lost much of its structure and is full of blood. A medium sized pulmonary artery contains a thrombus.