LIVER

MUSEUM CATALOGUE

Department of Pathology, University of Adelaide, 2004

LIVER

INTRODUCTION

Pathology is all about understanding disease – how it arises, its patterns, complications and how it causes symptoms and signs. That understanding is aided by having a visual appreciation of the morphological changes in tissues.

Powers of observation and description are not just of use in pathology. They are important when examining patients also. As soon as a patient walks into a room you should be observing them (are they fat, thin, pale, yellow, short of breath etc). Specific site, size, colour, texture, appropriate terminology etc are also important for describing lumps and skin lesions on a patient, and knowledge of pathological features is important in radiological diagnosis, so the observational and descriptive skills which you learn in pathology have a broader application.

The liver is situated in the right upper quadrant of the abdomen and normally weighs around 1450-1750g. It is important that you have an understanding of the histological structure and function of the liver and its vascular supply.

Any comments on this catalogue are welcome. Please contact a member of the department.

HOW TO USE THIS CATALOGUE

This catalogue can be used as a tool to develop your knowledge, as well as provide an opportunity for revision.

It is divided into:

- Introduction and approach to specimens (pages 88-90).
- Index (pages 91-92). Examples of specific diagnoses can be found via the index.
- Core and classic disease processes of the liver (pages 93-104). This gives examples and discussion of core and/or classic diseases of the liver. These are the specimens that students should focus on being able to identify initially. However, it depends to some extent on what you have covered in lectures and practical classes or resource sessions as to what you should know. Some of the specimens and discussion are directed more towards clinical medical students.
- Main catalogue (pages 105-120). This section covers the specimens in numerical order. Questions and/or comments accompany some of the specimens to help you expand your knowledge. In order to fit more specimens in the museum, not all of the pots are in numerical order on the shelves, and large specimens are often found on the bottom shelves.

You might find it useful to work quietly with a few friends and to have a few textbooks handy (e.g. pathology, medical, anatomy). You will also find that you can learn some anatomy and clinicopathological correlation from the specimens and information given.

You do not have to examine every single specimen in the museum. However, just as in clinical practice, you will not become proficient in diagnosing something if you have only seen one case. Exposure to a variety of cases (specific diagnoses can be found via the index) to experience the variability in morphology will help your learning greatly. In general red and blue dots indicate basic and straightforward cases, whereas yellow dots indicate a more complex case. This is not a hard and fast rule, and you will find yellow dot specimens turning up in resource sessions/practical classes and even exams, if they represent classic pathology.

In general

- read the clinical information given

- look at the entire specimen, not just the front
- identify and orientate the organ or tissue (where possible)
- from your knowledge of pathology (which will come with time) look for relevant features to help you make the diagnosis. Of course to appreciate the abnormal you first need to have an appreciation of normal anatomy to be able to recognize and orientate the organ/tissue and the abnormalities
- make a diagnosis or differential diagnosis using any clinical information given to you – it is often relevant – sometimes the diagnosis is only made with knowledge of the clinical features. Even when you know the diagnosis, attempt to identify relevant features in the specimen and understand why this is the diagnosis.
- attempt to correlate the pathological features with the clinical features (clinico-pathological correlation) i.e. explain how the pathological features have caused the patients symptoms and signs (when relevant)
- try to answer any questions presented yourself before reading the answers.

You may prefer to look at the specimen 'blind', without reading the clinical information given first.

Remember that some of these specimens are very old, and some of the investigations and treatments mentioned may be out of date.

Limits to diagnosis on macroscopic examination

In all cases a diagnosis is given in the catalogue, sometimes it was made based on the stated clinical history and histopathological findings. In some cases the macroscopic appearance is classic and even without the clinical information and histopathological findings you should be able to make the diagnosis from the appearance, in others, it might only be possible for you to give a list of differential diagnoses or a more general diagnosis.

In relation to pathology pot specimens in examinations, you may be asked

- for a diagnosis
- for a description
- about the pathogenesis of the disease
- about the predisposing factors and/or causes of the disease
- about the potential complications of the disease and how they arise
- to explain a patient's clinical symptoms and signs or investigation results in light of the pathological abnormalities present
- to describe the expected histological abnormalities in the abnormal areas

or other searching questions that we can concoct.

BASIC APPROACH TO INTERPRETATION AND DESCRIPTIONOF OF LIVER PATHOLOGY SPECIMENS

Students are expected to be able to give a brief succinct description of relevant macroscopic features of a specimen using appropriate terminology, as well as to arrive at a diagnosis or differential diagnosis. Even if not asked for a description, identification of relevant features is helpful in the diagnostic process. Your descriptive skills will improve with practice.

In any aspect of medicine, one needs to approach things in a systematic manner; otherwise important points may be omitted.

- Read the clinical history, it will often provide relevant information (although sometimes it is helpful to look at the specimen without any information and work out what is going on for yourself)
- Look at the front of the pot first (i.e. the one with the number and the dot), but always make sure to look at the back and sides as well.

- Normally one would decide and state whether the organ is of normal size, too small or too large though this is difficult for most liver specimens as there is no normal entire one for comparison and most specimens comprise only portions of liver.
- Identification of and description of the abnormality. Is the abnormality focal, diffuse (involving the entire organ, region or tissue) or multifocal? The lesion itself should then be described.

  Focal lesion

  The description of a discrete or focal macroscopic lesion can incorporate a number of features.
  Size: Give an approximate measurement
  Shape
  Colour: What colour is it? Is it all one colour or is it many colours (variegated)? Does it look homogenous (all the same the whole way through)?
  Consistency: This is of course difficult when the specimen is in a pot and you are unable to touch it. But even just by looking you can get some idea: Does it look solid or firm? Does it look friable (as if it is falling to pieces) or are there bits missing or greyish areas (altered blood) to suggest necrosis?
  Margins: Are they well defined/demarcated, surrounded by a band of fibrous tissue (encapsulated) or irregular or diffuse?

  Multifocal

  This means that there is more than one distinct lesion in the specimen. All the comments regarding the description of focal lesions apply here as well. In addition, it may be important to note any variation between lesions.

  Diffuse

  Diffuse lesions involve most or all of the organ. The commonest diffuse lesion in the pots in the museum is cirrhosis. Cirrhosis is defined as involvement of the entire liver by regenerative nodules of parenchyma, the nodules being completely surrounded by fibrous bands. Another common diffuse lesion is chronic passive venous congestion, seen in right heart failure.
- Identification of the major pathological process. In some cases it may be helpful to identify the general pathological process that the abnormality represents e.g. inflammatory or neoplastic (benign or malignant, primary or metastatic). This will be especially useful if you don't immediately know what the diagnosis is, at least you will be able to 'ball park' it. To do this it may be helpful to go through the surgical/pathological sieve.
- Identification of related lesions. By now you should have some idea of what you think the diagnosis, or at least the differential diagnosis, is. You should now think about what you know of this condition and look for, and describe, other relevant features that may confirm or refute this diagnosis. It may be useful to include relevant negatives e.g that residual liver parenchyma appears normal in a case of potential hepatocellular carcinoma.
- Other pathologies. Have a look at the rest of the specimen to see if there are any other abnormalities. If they are present, describe them. For example, there may be venous congestion surrounding a mass lesion, or background cirrhosis in a case of hepatocellular carcinoma. It may be useful to include relevant negatives, e.g that residual liver parenchyma appears normal in a case of potential hepatocellular carcinoma.
- Diagnosis. State your diagnosis or differential diagnosis. Be as precise and specific as possible. Use any relevant clinical information given to help you. Sometimes a precise diagnosis is not possible but a presumptive diagnosis based on the macroscopic and/or clinical findings is. If you can't decide on one diagnosis, give a list of reasonable differential diagnoses, in order of decreasing likelihood, give a more general diagnosis (e.g. malignant tumour), or at least attempt to identify the pathological process.

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CORE AND CLASSIC DISEASE PROCESSES

LIVER: CHRONIC PASSIVE VENOUS CONGESTION

CASE 50122/81

Clinical information
The patient was a woman aged 64 who had congestive cardiac failure.

Specimen description
The specimen consists of a slice of liver that shows a classical but severe nutmeg pattern. The congestion around central veins appears dark, with the paler areas surrounding portal tracts.

Diagnosis
Chronic passive venous congestion of the liver

Comment
Otherwise known as nutmeg liver (as the appearance is similar to a cut nutmeg) congestion of the liver occurs where there is impedence to the outflow of blood through the central or hepatic veins. It is most commonly caused by right heart failure, either on its own or in congestive cardiac failure. The appearance is mottled as the blood initially backs up around the central veins that become dilated and filled with erythrocytes. There may be mild alterations of liver function tests. With severe chronic venous congestion, hepatocytes around the central veins can undergo pressure atrophy, and in association with chronic ischaemia, also resulting from congestive cardiac failure, there is eventual fibrosis in these areas, resulting in cardiac ‘sclerosis’ The once used term cardiac cirrhosis is inappropriate as it is not a true cirrhosis.

Similar areas of venous congestion may also occur as a localized phenomenon in the liver around for example tumours that impair the flow of blood locally.

Acute hypoperfusion of the liver, that may for example occur in shock, can result in centrilobular necrosis of the liver, where centrilobular hepatocytes, being furthest from the incoming portal venous and hepatic arterial blood, undergo ischaemic necrosis. In such instances there will be more severe alteration in liver function tests and ultimately liver failure when severe.

LIVER: ABSCESS

CASE 20650

Clinical information
This man aged 79 had a cholecystectomy 5 months previously with good recovery. Two weeks before his last admission he developed upper abdominal pain. A choledocho-duodenostomy was performed. At the operation the pancreas was firm and there was fat necrosis in the omentum. He developed a gram-negative septicaemia with a swinging fever. Further laparotomy just before his death did not disclose any subphrenic collection.

Specimen description
The specimen is a slice of liver showing numerous coalescing abscesses containing thick creamy pus measuring up to 2cm in diameter throughout a large portion of the lobe. There is some evidence of fibrous capsule formation around some. The intervening liver substance is congested.

Diagnosis
Liver abscesses

Comment
At postmortem there was fibrinous peritonitis with multiple adhesions and 4 litres of fibrin-flecked straw-coloured fluid in the peritoneal cavity, suggesting leakage of the surgical anastomosis or perforation.

Abscesses in the liver are not common. Organisms may ascend via the bile ducts from the GIT in ascending cholangitis, as is likely here. Alternatively they can ascend from the GIT via the portal venous system, complicating for example acute appendicitis or amoebic colitis. Bacterial seeding may also occur with bacteraemia and direct seeding may occur with trauma. Tuberculous ‘abscesses’ of the liver are being seen more commonly in immunocompromised patients e.g. AIDS.

LIVER: AMYLOID INFILTRATION

CASE 3452

Clinical information
The patient was a woman aged 27 with a history of massive chronic pelvic abscess for 3 months, originating from the left fallopian tube. At postmortem the kidneys were large and the liver greatly enlarged.

Specimen description
The specimen is a slice of pale liver. An infiltrate diffusely replaces much of the liver, with only small pale flecks of residual liver remaining.

Diagnosis
Amyloid infiltration of the liver

What type of amyloid is this likely to be?
As the patient has a history of chronic infection, it is likely to be secondary, reactive systemic or AA amyloid.

Comment
Histology in this case showed typical massive amyloid deposition between the liver cords. Most of the liver cells had atrophied and only scattered islands of liver cells survived.

Amyloid is a pathological proteinaceous material deposited extracellularly (especially in basement membranes, vessel walls and connective tissues) in various organs and tissues of the body in a wide variety of clinical situations. Although it has a characteristic appearance under the microscope, amyloid can be formed from a number of different proteins, the specific type depending on it's underlying cause. The 2 main types of amyloid are AL and AA. AL amyloid is formed from immunoglobulin light chains produced in various plasma cell disorders, most notably multiple myeloma. AA amyloid is derived from serum amyloid associated protein (a normal protein produced by the liver) in various chronic inflammatory diseases (e.g. rheumatoid arthritis, TB) and some neoplastic processes. It is uncertain why these proteins deposit.

Amyloidosis may involve many organs or only a single organ. Tissues that are frequently involved include the kidney, spleen, liver, heart and tongue. A distinct form of amyloid is found at the centre of plaques in the brain in cases of Alzheimer's disease. Depending on the organ involved, amyloid deposition can cause all sorts of signs and symptoms. Renal involvement is characterised by proteinuria and even nephrotic syndrome and as more glomeruli are destroyed the patient will eventually develop chronic renal failure. Cardiac involvement may lead to congestive cardiac failure. Congophilic angiopathy of the brain can cause intracerebral haemorrhage.

Macroscopically affected organs are often enlarged and firm. Histologically on H&E sections, the material is eosinophilic. The congo red stain is classically used to highlight amyloid in tissue sections where it shows an 'apple green' birefringence on polarisation (using special lenses). On electron microscopy, amyloid has a characteristic fibrillary structure.

LIVER: HYDATID CYST

CASE 13489

Clinical information
The patient was a boy aged 12, the son of a city newsagent who had two dogs. Two years previously a cardiac murmur was apparently detected for which cardiac catheterization and ECG were done, but the records reveal little of what was found. He suddenly collapsed and died at school.

Specimen description
The specimen is part of the liver sectioned to show an encapsulated 10cm diameter cyst containing many thin walled free-floating 1cm diameter daughter cysts and folded opaque fibrous membranes. A smaller 3.5cm diameter encapsulated cyst containing folded membranes and putty-like material is present adjacent to the larger cyst. The liver substance shows evidence of chronic passive venous congestion.

Diagnosis
Hydatid cysts of the liver

Comment
At postmortem a hydatid cyst 8cm in diameter was also found in the posterior wall of the left ventricle bulging forward into the chambers of both ventricles.

What is a hydatid cyst and how are they acquired?
Hydatid disease is endemic in herd animals and their attendant dogs. It is caused by the ingestion of eggs of the tapeworm Echinoccocus granulosus from dog faeces. Eggs hatch in the intestine and the larvae invade the liver especially, but can also spread to other organs. Cysts form that contain brood capsules and scolices. The cysts have a dense fibrous capsule that often becomes calcified. When material from cysts in the offal of the herbivore (e.g. sheep) is ingested by the carnivore (e.g. dog), the scolices develop into sexually mature worms in the latter, thus completing the life cycle. Humans are infected accidentally, from eating material contaminated with eggs. The disease is especially common in areas where herd animals are farmed. The slowly growing cysts are often asymptomatic, often being found incidentally, or from causing pressure symptoms. Treatment is via surgical removal. Rupture of a hydatid cyst into a body cavity may cause fatal allergic reactions.

LIVER: HAEMOCHROMATOSIS WITH CIRRHOSIS AND HEPATOCELLULAR CARCINOMA

CASE 17564

Clinical information
The patient was a man aged 66. Ten years previously he had been a heavy drinker and apparently had a haematemesis. His last admission was with abdominal pain, anorexia, ascites and wasting. The liver was enlarged. Abdominal paracentesis was performed and thereafter the patient became febrile, gradually deteriorated and died. At postmortem the liver was enlarged (weight 2190gm) and it showed a fine cirrhosis with deep brown pigmentation. Tumour masses were present throughout its substance and there were also moderate numbers of metastases up to 1cm in diameter in both lungs. The pancreas was normal in size and was also brown.

Specimen description
The specimen consists of a slice of liver that is golden-brown in colour. The cut surface shows diffuse involvement by small nodules less than 3mm in diameter that are completely surrounded by fibrous bands. A pale 6cm tumour mass with ill-defined margins is present at one edge. Several smaller pale tumour nodules are scattered throughout the liver substance elsewhere.

Diagnosis
Haemochromatosis with cirrhosis and hepatocellular carcinoma

Comment
Histology of this case showed cirrhosis with massive deposition of haemosiderin in liver cells and macrophages. The tumour was a large-celled pleomorphic hepatocellular carcinoma conspicuously invading veins.

Hepatocytes normally contain a small amount of iron in the form of ferritin. However, iron can accumulate in the liver in a number of diseases in the form of haemosiderin, which when in large enough quantities can cause the liver to become brown. On light microscopy on an H&E stain, haemosiderin is seen as intracytoplasmic brown granular pigment. A Perl's stain is often used to confirm the nature of the intracytoplasmic pigment as iron, with which it stains blue. Mild quantities of excess iron can be seen microscopically in the liver in alcoholics and in chronic hepatitis C, however, large amounts as present here, are normally associated with total body iron overload, as occurs in hereditary (primary) haemochromatosis, a not uncommon autosomal recessive disorder where there is increased absorption of iron from the gut, or in patients who have iron overload for other reasons e.g. numerous blood transfusions or ineffective erythropoiesis (secondary haemochromatosis). The excessive deposition of iron in the liver causes chronic liver damage, cirrhosis and often ultimately hepatocellular carcinoma.

With systemic iron overload, iron deposition can also occur in the
- pancreas -> fibrosis, atrophy and diabetes mellitus
- heart -> cardiomyopathy, arrhythmias and heart failure
- skin -> pigmentation
- joints -> inflammation, deposition of calcium pyrophosphate -> arthritis
- testes -> impotence, loss of libido
- adrenals, thyroid

Affected organs are also often pigmented.

LIVER: CIRRHOSIS

CASE 18999

Clinical information
The patient was a man aged 57 who had been a heavy drinker for at least 20 years. Two years before his last admission he had a transfusion in private and x-ray studies a year later showed oesophageal varices. His final admission followed a large haematemesis with melaena. On examination the BP was 200/170, there was bilateral parotid swelling, multiple cutaneous spider naevi, liver palms, slight hand tremor with early Dupuytren's contractures, ascites and palbable liver and spleen. He was treated with transfusion, a Sengstaken tube and oxytocin drip. He later developed epileptiform convulsions from hepatic encephalopathy. This was treated by low protein diet, oral neomycin, blood transfusions and sedation. Surgery was considered but rejected and the patient deteriorated and died 2 months after admission. At postmortem there was massive ascites, a small cirrhotic liver (weight 830gm) and splenomegaly (weight 550gm).

Specimen description
The specimen is a slice of liver. The architecture is diffusely abnormal. The surface is nodular and the cut surface shows nodules of liver parenchyma, mostly measuring less than 3mm in diameter that are completely surrounded by fibrous bands. Scattered dark patches may represent bile staining.

Diagnosis
Cirrhosis

Comment
This is an example of micronodular cirrhosis.

Medical students, especially clinical medical students, should be able to explain the pathogenesis of the clinical features in light of the pathology.

CASE 3616

Clinical information
The only information available for this old specimen is that the patient was an inmate of a mental hospital, apparently with the mental deficit of Wilson's disease.

Specimen description
The specimen is a small cirrhotic liver. The islands of regenerating liver cells vary in size up to almost 1cm in diameter and there is dense intervening fibrosis.

Diagnosis
Cirrhosis

Comment
This is an example of macronodular cirrhosis.

Comments

Cirrhosis results from ongoing liver cell death, chronic inflammation and fibrosis, and can arise in a wide range of diseases. It is characterised by:

- involvement of the entire liver
- regenerating nodules of hepatocytes that are completely surrounded by fibrous bands
- disruption of the vascular architecture and thus normal blood flow through the liver

Cirrhosis used to be divided into micronodular and macronodular forms, as little was known about the causes apart from alcohol. However, in many cases the cause can now be identified and this is more important than the size of the nodules.

There are many causes of cirrhosis including:

- chronic alcoholism
- chronic infection by hepatitis viruses B (+/- D) and C
- inherited metabolic conditions
    - hereditary haemochromatosis
    - alpha-1-antitrypsin deficiency
    - Wilson's disease
- autoimmune hepatitis
- chronic intrahepatic biliary obstruction
    - primary biliary cirrhosis (autoimmune)
- chronic extrahepatic biliary obstruction
    - gallstones
    - primary sclerosing cholangitis (autoimmune)
- chronic intake of certain drugs

In some cases a cause cannot be found.

Cirrhosis may be asymptomatic, but other symptoms and signs relating to acute or chronic liver impairment may arise.

Disturbance of the architecture with disruption of blood flow through the liver in cirrhosis is one of the causes of portal hypertension. High pressures in the portal venous system are transmitted back to anastomoses between the portal and systemic venous systems leading to their dilatation. The most significant of these is around the oesophago-gastric junction. The dilated 'oesophageal varices' can rupture and bleed profusely.

LIVER: HAEMANGIOMA

CASE 11192/83

Clinical information
The patient was a woman aged 38. The liver lesion was an incidental finding at postmortem.

Specimen description
The specimen consists of a slice of liver. Protruding from the surface is a dark red oval lesion 40x15mm whose cut surface has a spongy consistency.

Diagnosis
Haemangioma of the liver

Comments
Haemangioma is the commonest benign tumour of the liver. They are usually embedded within the liver parenchyma, unlike this one. Many remain asymptomatic. Large ones may bleed and rupture or sequester platelets leading to thrombocytopaenia. Microscopically they consist of dilated vascular spaces lined by bland endothelium supported by fibrous tissue. Primary angiosarcomas may also arise in the liver.

Other benign tumours of the liver include liver cell adenoma and biliary cystadenoma.

LIVER: HEPATOCELLULAR CARCINOMA

CASE 19881

Clinical information

This patient was a man aged 51 who was quite well until 3 weeks before admission when he developed retrosternal pain and cough. Two weeks later there was abdominal swelling with anorexia and nausea but no vomiting. Four days later ankle oedema developed and he was admitted to hospital. The liver was enlarged, firm and tender with an irregular edge. There was ascites and there were engorged veins on the anterior abdominal wall. The JVP was elevated 10cm and there were bilateral basal crepitations. Spider naevi and liver palms were present. The spleen was palpable. Many investigations indicated liver failure. Blood platelets were persistently low. He died after a massive haematemesis.

Specimen description

The specimen consists of part of a shrunken liver. The surface is nodular and the cut surface exhibits regeneration nodules mostly less than 3mm in diameter that are completely surrounded by bands of fibrous tissue.

There is extensive involvement of one side by an ill-defined extensively necrotic and haemorrhagic tumour measuring up to 14cm in diameter. In places, areas of tumour are surrounded by fibrosis, resulting in the formation of well-circumscribed tumour nodules. The tumour has extended into a hepatic vein and the inferior vena cava where it is covered by laminated antemortem thrombus.

Diagnosis

Cirrhosis with hepatocellular carcinoma

Comment

Hepatocellular carcinoma is closely associated with infection by hepatitis viruses B and C and/or cirrhosis. A number of other carcinogenic agents have also been identified. Globally it is common, being most common in parts of Africa and Asia where there is a high incidence of hepatitis B. In these populations, co-existing cirrhosis is not necessarily present. Its incidence is increasing in Western countries, mostly attributable to hepatitis C. In Western countries, 85-90% of cases have underlying cirrhosis. Hepatocellular carcinomas may be unifocal, multifocal (with multiple nodules of tumour throughout the liver) or diffusely infiltrative. Cells demonstrate features of hepatocytes and range from well-differentiated lesions to highly anaplastic tumours. They frequently invade veins as macroscopically obvious tumour masses. Spread is usually to local lymph nodes and lung. Elevated levels of Ipha-fetoprotein are found in the serum in 60-75% of patients. Though not specific, this can be a useful feature. Death usually occurs within a year of diagnosis.

The fibrolamellar variant of hepatocellular carcinoma has a more favourable outlook. It tends to affect young adults without cirrhosis.

Microscopically, dysplasia of hepatocytes can be recognized and conveys an increased risk for the development of hepatocellular carcinoma.

Cholangiocarcinoma, an adenocarcinoma that shows features of bile ducts, angiosarcoma, a malignancy demonstrating vascular differentiation and hepatoblastoma, a malignant tumour predominantly arising in infants that demonstrates cells resembling those of fetal or embryonal liver are other malignancies that arise in the liver. Lymphoma may also arise in the liver or involve it as part of systemic disease.

LIVER: METASTATIC MALIGNANCY

CASE 50472/82B

Clinical information
The patient was a man aged 66 with a history of small cell carcinoma of the bronchus.

Specimen description
The specimen is a slice of liver that shows numerous round, well-demarcated pale tumour masses measuring from 1-60mm in diameter, several showing paler central necrosis, with the largest starting to cavitate. The remaining liver appears essentially normal apart form mild congestion around the larger mass.

Diagnosis
Metastatic tumour

Comment
The liver is a very common site of metastatic disease, especially, but not only, from gastrointestinal malignancy.

LIVER: LEUKAEMIC INFILTRATION

CASE 25301

Clinical information
The patient was a man aged 74 with a history of duodenal ulcer in 1957, myocardial infarction in 1967, and chronic lymphatic leukaemia with deep vein thrombosis in 1969. He died in 1974 as a result of acute pulmonary oedema following a further myocardial infarct.

Specimen description
The specimen consists of slices of liver and spleen. The spleen is considerably enlarged and shows many tiny scattered pale nodules in the white pulp. A 20mm diameter old infarct is present at one end. The liver is pale with a smooth surface. Its cut surface shows diffuse infiltration by pale material.

Diagnosis
Leukaemic infiltration

Comment
The liver and spleen are commonly enlarged in patients with leukaemia due to infiltration by leukaemic cells.