MRCP PACES: 180 Clinical Cases

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Chapter 4

Abdominal system (station 1)

Case 80: The syndrome of chronic liver disease

Instruction to the candidate

You are the registrar in the gastroenterology clinic. A 53-year-old man has been referred by his GP after a recent finding of abnormal liver function tests. Please examine the patient's abdominal system and report your findings to the examiner.

Begin with a summary of positive findings

Seek to identify not only signs suggestive of a diagnosis of chronic liver disease, but also signs which would suggest an underlying cause.

Signs of chronic liver disease

- On general inspection – cachexia with muscle wasting, scratch marks
- In the hands – clubbing (**Figures 4.1** and **4.2**), leukonychia (**Figure 4.3**), palmar erythema
- In the face – scleral icterus
- On the praecordium – spider naevi (**Figure 4.4**), paucity of body hair, gynaecomastia
- In the abdomen – jaundice, ascites, collaterals and caput medusae (all of which are suggestive of advanced or decompensated disease)

Additional signs suggestive of an underlying cause

- Alcoholic liver disease:
  - Facial telangiectasia – non-specific but common in alcohol excess
  - Dupuytren's contracture – predominantly idiopathic but with a recognised association with alcoholic liver disease
  - Bilateral swollen parotid glands
  - Neurological signs of alcoholism – including peripheral neuropathy, proximal myopathy and cerebellar syndrome
- Signs suggestive of viral hepatitis:
  - Tatoos
  - Track marks suggestive of intravenous drug use
- Primary biliary cirrhosis:
  - Periorbital xanthelasma, commonly with jaundice
- Haemachromatosis:
  - Slate grey skin pigmentation

The character of the liver edge can also be of use in forming a differential. A cirrhotic liver will either be impalpable, or shrunken, hard and irregular. In fatty liver disease or active hepatitis, the liver may be tender, smooth and enlarged. Non-tender hepatomegaly is suggestive of a range of causes, but if craggy should direct investigations towards hepatocellular carcinoma.

Follow with a summary of relevant negative findings

Advanced liver disease progresses towards cirrhosis with implications both on portal venous pressure and reduced functional reserve with eventual loss of liver function. The clinical manifestations of portal hypertension should not be missed, not least as they serve as a useful indicator of the severity of disease and alert to the likelihood of varices. Eliciting the features of hepatic failure and establishing decompensated disease is fundamental to a basic evaluation where liver disease is suspected and must be commented upon.

Patients with portal hypertension may have some or all of: splenomegaly, a venous hum on auscultation, abdominal collateral vessels (and caput medusa) and/or ascites.

Decompensated hepatic failure can presents with a constellation of the following signs:

- Asterixis
- Jaundice
- Coagulopathy. On clinical examination this may present as bruising and/or active bleeding
- Ascites
- Hepatic encephalopathy. If encephalopathy is present, seek to grade its severity

State the most likely diagnosis on the basis of these findings

'This patient has clinical signs consistent with compensated chronic liver disease with no evidence of portal hypertension.'

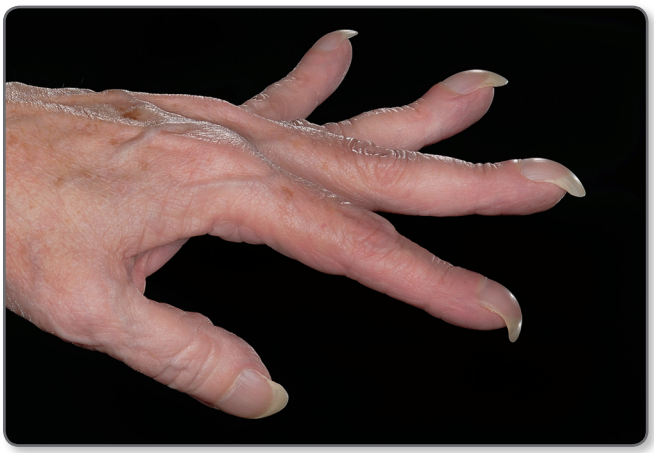

Figure 5.1 Clubbing of the fingers. Loss of the normal angle between the nail bed and cuticle/skin is evident; normally approximately 160 degrees, in clubbing the angle is commonly greater than 180 degrees. There is an exaggerated convexity of the nail fold. Thickening of the distal portions of the fingers is appreciable and sponginess would be expected with softening and fluctuation of the nail bed. The skin is also taught and has a shiny quality.

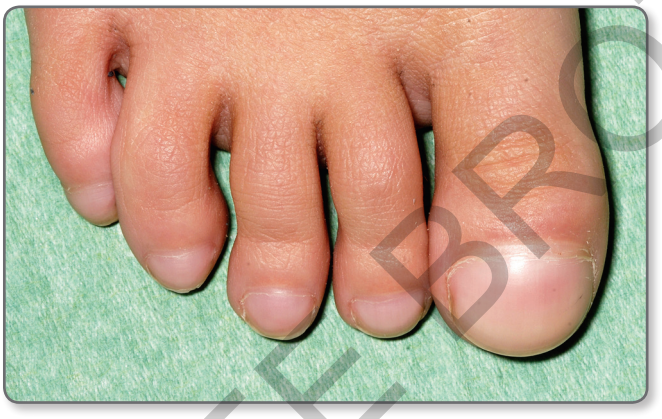

Figure 5.2 Clubbing of the toes. The clinical findings are identical to that described in Figure 5.1. The process is indiscriminate, affecting toes and fingers alike.

Figure 5.3 Leukonychia. Whitening of the entire nail (leukonychia totalis) reflects hypoalbuminaemia. This should not be confused with partial leukonychia secondary to illness (Mee's lines) or nail bed injury, where white lines or spots can be observed in an otherwise normal nail.

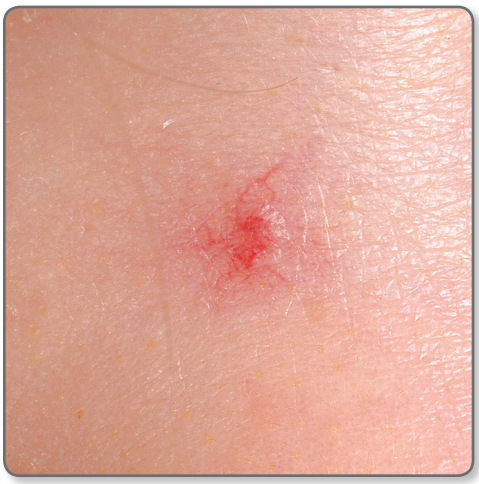

Figure 5.4 Spider naevus. Telangiectasia which blanch and subsequently refill centrally when pressure is applied; they are found within the distribution of the superior vena cava.

Offer relevant differential diagnoses

From the examination findings it may be possible to offer and justify a likely underlying aetiology for the evident liver disease, as outlined above, followed by a differential. Where this is not possible, proceed directly to a sensible differential:

- Common causes:
  - Alcoholic liver disease
  - Non-alcoholic fatty liver disease
  - Chronic hepatitis C infection
- Less common:
  - Chronic hepatitis B
  - Autoimmune hepatitis
  - Primary biliary cirrhosis
- Rare:
  - Primary sclerosing cholangitis
  - Wilson's disease
  - Haemachromatosis

Demonstrate the importance of clinical context – suggest relevant questions that would be taken in a patient history

Screening for risk factors associated with commonly recognised underlying aetiologies. Where alcoholic liver disease is suspected screening for dependency and establishing current level of consumption is paramount. Risk factors for hepatitis B and C include blood transfusions; intravenous drug use; tattoos; and high-risk sexual intercourse. It is important to enquire about medication use, such as methyldopa, amiodarone and methotrexate.

Establishing a history of previous episodes of decompensation requiring admission and/or treatment, particularly in the high dependency or intensive care setting, gives important context for the potential for deterioration. Ask specifically about previous episodes of ascites, and where there is a history of varcies, haemorrhage.

Demonstrate an understanding of the value of further investigation

A sensible approach to investigation of a patient with chronic liver disease would include simple blood testing and imaging tests, with further investigations as appropriate. The two key aims of investigation are to assess liver function (importantly including synthetic function) and to identify the underlying cause if possible.

Blood tests

Appropriate blood testing to assess liver function would include:

- Liver function tests (LFTs), including bilirubin (conjugated and unconjugated), transaminases and alkaline phosphatase. An AST:ALT ratio of greater than two suggests alcoholic liver disease. This is due to chronic alcohol consumption causing a lack of vitamin B6, which is required for ALT function. A ratio of less than one suggests non-alcoholic liver disease. An elevated alkaline phosphatase is suggestive of cholestasis
- Markers of synthetic function include the prothrombin time, serum albumin and platelets. Impaired synthetic function is suggested by a prolonged prothrombin time and/or a reduced serum albumin and/or a reduced platelet count

Blood tests to identify the aetiology of the chronic liver disease would include:

- Serological testing to identify viral infections such as:
  - Hepatitis A, B, or C
  - CMV
  - EBV
- Auto-antibody testing:
  - Primary biliary cirrhosis – positive anti-mitiochondrial antibodies in 95%
  - Autoimmune hepatitis – positive ANA, anti-smooth muscle antibody
  - Primary sclerosing cholangitis – positive ANCA in 80%
- Other tests for miscellaneous causes such as:
  - Ferritin as a screen for haemachromatosis
  - Caeruloplasmin for Wilson's disease
  - Alpha-1-antitrypsin levels
  - Tumour markers if appropriate

Imaging

In the acute setting it is prudent to consider arranging simple imaging with ultrasound to rule out an obstructive cause before embarking upon expensive laboratory investigations.

Abdominal ultrasound is the first line imaging modality of the liver. It provides information about the liver echotexture (cirrhosis, fatty infiltration) and can identify masses. Additionally, ultrasound allows a Doppler assessment of portal blood flow and will provide information on the presence or absence of splenomegaly. If ascites is present, this can be identified, quantified and marked for drainage.

Biopsy may be considered for diagnostic purposes where the aetiology is unclear with equivocal or unexplained laboratory results, or where there is suspicion of multiple causes of liver disease such as alcohol and viral hepatitis. Importantly, where biopsy is proposed, ensure clotting is not significantly deranged.

Always offer a management plan

A discussion on the management of chronic liver disease should involve discussion not only of general aspects of management, but also of the management of the underlying cause, assuming one has been identified through clinical examination.

General management aspects include:

- Dietary and lifestyle advice with B vitamin supplementation in patients with chronic alcohol consumption, and a low salt and high protein diet in those with ascites
- Endoscopic surveillance for varices
- Screening ultrasound and α-fetoprotein levels 6 monthly for hepatocellular carcinoma
- Need for early referral to high-dependency/intensive care (if appropriate) in the deteriorating liver patient.
- Medication prescribing in the liver patient

Management dependent upon the underlying cause:

- Alcohol – abstinence
- Viral hepatitis (B/C) – interferon, ribavirin, protease inhibitors such as telapravir and beceprivir
- Autoimmune hepatitis – prednisolone, azathioprine
- Haemochromatosis – therapeutic phlebotomy (usually to maintain a ferritin of 20–50 ng/mL) and chelation
- Primary biliary cirrhosis – ursodeoxycholic acid
- Wilson's disease – trientine and zinc

Case 81: Organomegaly – isolated hepatomegaly

Instruction to the candidate

This 45-year-old man has been referred to the gastroenterology clinic following a routine medical examination. Please examine his abdominal system.

Begin with a summary of positive findings

Hepatomegaly is suggested by a palpable mass below the right costophrenic margin which moves towards the right iliac fossa on inspiration, and is dull to percussion. Further characteristics of the hepatomegaly to identify include:

- Is it smooth or craggy?
- Is it firm/hard?
- Is it tender or non-tender?

Associated findings to look for include any scars to indicate previous biopsy or paracentesis.

Follow with a summary of relevant negative findings

In patients with isolated hepatomegaly it is important to report the absence of signs to suggest chronic liver disease or hepatic decompensation. Additionally, comment on the absence of:

- Concurrent splenomegaly
- Lymphadenopathy: if present, lymphadenopathy may suggest an infective cause, but the presence of hepatosplenomegaly and lymphadenopathy would raise suspicion of a lymphoproliferative disorder
- Features of congestive cardiac failure or tricuspid regurgitation. Tricuspid regurgitation classically presents with a pulsatile liver edge

State the most likely diagnosis on the basis of these findings

'This patient has signs consistent with isolated hepatomegaly.'

Offer relevant differential diagnoses

There is a wide range of causes of hepatomegaly and these can be classified in various ways.

Congential causes of hepatomegaly include Riedel's lobe and polycystic disease.

Acquired causes include:

- Infections, such as the viral hepatitides, CMV, EBV, amoebiasis, toxoplasmosis, malaria
- Drugs, such as alcohol, amiodarone, methotrexate
- Metabolic and Infiltrative causes, such as NASH, haemachromatosis, Wilson's disease, amyloidosis, Gaucher's
- Autoimmune conditions such as autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis
- Neoplastic disease, in which you would expect to find irregular firm hepatomegaly on clinical examination. Secondary liver metastasis are more common than primary hepatic neoplasms
- Passive venous congestion due to right heart failure with resistance to right ventricular filling:
  - Tricuspid regurgitation: classically pulsatile hepatomegaly. Examination of the JVP can be useful in this regard, as giant V waves would be virtually pathognomonic
  - Constrictive pericarditis
  - Restrictive cardiomyopathy
- Vascular causes such as Budd–Chiari or sickle cell disease

In view of the extensive differential, to avoid the potential of listing and appearing prescriptive it is useful to consider the differential diagnosis of isolated hepatomegaly in relation to the additional features sought on examination. It is paramount that the presentation of positive and negative findings be comprehensive and clear enabling the candidate to proceed with a narrowed differential.

Demonstrate the importance of clinical context – suggest relevant questions that would be taken in a patient history

A suitable clinical history would include questions to screen for known risk factors of liver disease, including:

- Alcohol: ask about average usage, including episodes of bingeing
- Recent travel: has the patient been to any areas known to increase risk for certain underlying causes?
- Risk factors for viral hepatitis such as blood transfusions, intravenous drug use, tattoos, or unprotected or high risk sexual intercourse

Demonstrate an understanding of the value of further investigation

Blood testing would include inflammatory markers, liver function tests and liver disease screen.

Ultrasound of the liver would be the first line imaging modality to confirm clinical findings of hepatic enlargement and assess presence of fatty infiltrate, masses, cysts or abscesses. It can also rule out biliary duct dilatation to help distinguish parenchymal liver disease from extrahepatic bililary obstruction.

Further imaging such as CT or magnetic resonance cholangiopancreatography should be considered where ultrasound scanning fails to provide adequate assessment, is limited by obesity or poor views or where a malignant process is suspected.

Needle aspiration or biopsy, as appropriate, for evaluation of lesions suggestive of an infectious process, a cystic mass or neoplastic disease.

Always offer a management plan

Directed to the cause. The focus will vary greatly from case to case and may inevitably be guided by the examiner.

Case 82: The distended abdomen – ascites

Instruction to the candidate

This 57-year-old woman has presented complaining of abdominal swelling. Please examine her abdominal system and present your findings to the examiner.

Begin with a summary of positive findings

On clinical examination, the abdomen is distended – and may be tense – but soft and non-tender to palpation, with demonstrable shifting dullness. Abdominal herniae may be prominent or be suggested by a flattened or everted umbilicus.

Additional associated signs include:

- Hepatomegaly, which is often difficult to elicit in the distended abdomen but may indicate non-alcoholic fatty liver, early cirrhosis with fatty infiltration or chronic cirrhosis with hepatocellular carcinoma
- Peripheral oedema: ascites caused by liver disease is usually isolated or disproportionate to peripheral oedema whereas the reverse is true where the cause is generalised fluid retention attributable to congestive heart failure
- Signs of an underlying cause of liver disease

Follow with a summary of relevant negative findings

Important relevant negative findings to consider include:

- Signs of portal hypertension: splenomegaly, venous hum, caput medusa
- Other signs of hepatic decompensation, importantly encephalopathy, asterixis, jaundice

- Signs of sepsis, which if present would raise the possibility of spontaneous bacterial peritonitis (SBP)

State the most likely diagnosis on the basis of these findings

'This patient has ascites, of which the most common cause is cirrhosis with portal hypertension.'

Offer relevant differential diagnoses

The differential diagnosis of ascites can be divided into hepatic and non-hepatic causes.

Hepatic causes include portal hypertension due to cirrhosis. This is the underlying cause inapproximately 90% of all cases. Such patients demonstrate the stigmata of chronic liver disease. An additional hepatic cause would include severe alcoholic hepatitis (without cirrhosis).

Non-hepatic causes of ascites would include:

- Peritoneal malignancy
- Intra-abdominal tuberculosis
- Fluid retention due to congestive cardiac failure, which on clinical examination would reveal peripheral oedema and a raised JVP (note comparison with portal hypertension where, as a result of venous dilatation, the cardiac filling pressure is low with a JVP typically difficult to elicit)
- The nephrotic syndrome and generalised hypoalbuminaemia will also give the picture of generalised oedema and fluid overload
- An important vascular cause is that of hepatic vein thrombosis, known as Budd–Chiari syndrome
- Less common causes include, pancreatitis, SLE, hypothyroidism, Meig's syndrome and chylous ascites

Demonstrate the importance of clinical context – suggest relevant questions that would be taken in a patient history

Questions identifying risk factors for, and symptoms of, the conditions listed below:

- Cirrhosis and portal hypertension
- Cardiac disease
- Malignancy
- TB

It would also be important to ask about fevers and abdominal pain, which would lead to consideration of SBP.

Demonstrate an understanding of the value of further investigation

Investigations should include an assessment of the ascitic fluid. This will aid both the identification of the underlying cause of the ascites and allow exclusion (or confirmation) of spontaneous bacterial peritonitis.

A diagnostic paracentesis should be performed with appropriate analysis of the fluid according to the following parameters:

- Colour and appearance: is it blood stained, chylous, turbid, or straw coloured?
- White cell count: if the polymorph count is >250 this is indicative of spontaneous bacterial peritonitis: Further microscopy, culture and sensitivity will allow identification of the causative organism
- Fluid biochemistry: the protein level differentiates transudate versus exudate. Additionally, the serum albumin to ascites albumin gradient (SAAG) differentiates portal hypertensive ascites (SAAG > 11g/L) from non-portal hypertensive ascites
- Cytological assessment allows for identification of malignant cells in the ascitic fluid

Serum-to-ascites albumin gradient

Assessment of the ascitic fluid will include comparison of the albumin content in the ascites with the serum albumin content to give the serum to ascites albumin gradient (SAAG). Measuring SAAG enables the classification of portal hypertensive (SAAG >11 g/L) versus non-portal hypertensive (SAAG < 11 g/L) causes of ascites. This is calculated by subtracting the ascitic fluid albumin value from the serum albumin value, from samples obtained at the same time. The value correlates with portal pressure. While typically absolute protein levels, transudates and exudates are rarely applied to ascitic fluid, the protein level in conjunction with SAAG can be useful. An elevated SAAG and a high protein level are observed in most cases of ascites due to hepatic congestion. However, the combination of a low SAAG and a high protein level is characteristic of malignant ascites.

Where there is clinical suspicion of non-cirrhotic aetiology, consider the following:

- Cardiac investigations should include ECG and echocardiogram
- For malignancy consider appropriate imaging and serum tumour markers
- For pancreatic pathology assess ascitic amylase
- Thyroid function tests

Always offer a management plan

In portal hypertensive ascites, initial management should include bed rest and salt restriction. Salt restriction represents the best conservative approach, aiming for <2 g/day. Fluid restriction should be applied where hyponatraemia exists. Diuresis with spironolactone should be considered if salt and fluid restriction unsuccessful. Ideally, weight loss of 0.5 kg per day should be achieved. Where more weight is lost, it is likely to represent diuresis from the intravascular compartment, not peritoneal, and can result in complications including renal dysfunction and worsening electrolyte derangement.

Management options for refractory ascites include:

- Therapeutic paracentesis: ascitic drains should remain in situ for no longer than 6–8 hours and should not be clamped. 100 mL of 20% albumin should be administered for every 2 litres drained. Transjugular intrahepatic porto-systemic shunting (TIPS) is utilised in ascites resistant to other treatment or where rapid reaccumulation occurs with haemodynamic compromise. An invasive technique not without its complications one must ensure against a coagulopathy, and thereafter be alert to the potential for porto-systemic encephalopathy and worsening of hepatic disease.
- Transplantation

Non-portal hypertensive ascites is largely unresponsive to diuretics and requires recurrent paracentesis.

Where SPB is suspected, it should be treated as a medical emergency with broad spectrum antibiotics, in line with local guidance. Where possible, obtain ascitic, blood, and urine cultures prior to initiation of antibiotics. Diagnostic paracentesis should not delay the start of treatment where there is strong clinical suspicion. Culture yield is traditionally poor from ascitic fluid but diagnosis can be made in the first instance on the presence of greater than 250 neutrophils per micro-litre. Antibiotic prophylaxis may be considered for those patients at high risk of developing SBP with advanced cirrhosis and significant ascites, subsequent to successful treatment, and in those admitted with acute variceal bleeding.

Case 83: Portal hypertension

Instruction to the candidate

This 69-year-old man has a history of excess alcohol consumption. Please examine his abdomen and present your findings to the examiner.

Begin with a summary of positive findings

Portal hypertension, a process heralding cirrhosis in the context of chronic liver disease, is manifest as:

- Splenomegaly
- Porto-systemic anastomoses such as caput medusae and oesophageal varices
- Ascites
- A venous hum on auscultation

Additional findings to identify include those of chronic liver disease

Follow with a summary of relevant negative findings

Important relevant negative findings would include:

- Evidence of confusion caused by porto-systemic encephalopathy

- Evidence of decompensated liver disease, particularly coagulopathy given the propensity for gastrointestinal bleeding from varices

State the most likely diagnosis on the basis of these findings

'This patient has clinical signs suggestive of portal hypertension.'

Offer relevant differential diagnoses

The most common cause of portal hypertension in developed countries is liver cirrhosis.

Causes of non-cirrhotic portal hypertension include:

- Schistosomiasis (in travellers from endemic areas) and HIV are important infective causes
- Increased resistance to right ventricular filling – constrictive pericarditis, restrictive cardiomyopathy and tricuspid regurgitation
- Hepatic vascular aetiologies such as Budd–Chiari causing a post hepatic obstruction
- Increased portal venous flow – although rare, this may occur in arteriovenous malformation or as the result of massive splenomegaly caused by a primary haematological disorder (see *Splenomegaly*, p. 163).

Demonstrate the importance of clinical context – suggest relevant questions that would be taken in a patient history

In a clinical history, the patient should be asked regarding whether varices have been identified and also whether there has been any previous gastrointestinal bleeding and/or intervention. The risk factor profile for liver disease should be explored, specifically asking about alcohol and – where suspected – other precipitants including infection and heart disease.

Demonstrate an understanding of the value of further investigation

A diagnosis of portal hypertension is inferred in patients with evidence of cirrhosis by clinical examination. Direct measurement of the portal pressure with transjugular catheter is rarely performed due to the incumbent risks involved. Thus, the diagnosis is confirmed with ultrasound or CT demonstrating engorged intra-abdominal collaterals and with Doppler to assess portal vein patency and flow.

Endoscopy with direct visualisation of oesophageal and gastric fundus varices confirms the diagnosis and allows intervention as appropriate.

Where no clear precipitant for liver disease has been identified, the patient should be investigated with a full liver screen. Suspected cirrhosis, most commonly due to alcohol with a clear history, should be investigated to assess severity with blood tests and imaging ensuring no signs of decompensation.

Always offer a management plan

Prevention of bleeding is key. Acute variceal bleeds carry mortality rates upwards of 50%, and are associated with high rates of re-bleeding in survivors.

All patients diagnosed with cirhosis should undergo an endoscopy to screen for varices. Primary prophylaxis is with beta-blockade (typically propranolol or carvedilol) to reduce portal blood flow. The dose is titrated up to maintain a resting heart rate of <55 beats/min. In addition to reducing the likelihood of a variceal bleed, if successful in reducing portal pressure, beta-blockers may also reduce chronic bleeding from gastric mucosal vascular congestion (portal hypertensive gastropathy). Patients who respond poorly to medical therapy may be considered for TIPS or porto-caval shunting. Liver transplantation may be considered depending on the clinical picture.

Splenomegaly rarely causes complications in the context of portal hypertension and thus splenectomy is avoided as a general rule.

The patient should be educated on avoidance of causative/contributory factors.

Prognosis

In the presence of cirrhosis, clinical examination and the results of laboratory information can be combined to provide a prognostic score, as in the Child–Pugh score (**Table 4.1**). A CPT score greater than 10 carries a 50% 1 year mortality in those with advanced cirrhosis. The Model for End-stage Liver Disease (MELD) scoring system has become increasingly favoured over the CPT classification in predicting mortality although the modified-CPT which takes into consideration creatinine levels has been shown to be as useful as MELD in predicting short and medium term mortality and is significantly simpler in execution. Reference: Papatheodoridis et al. MELD vs Child–Pugh and creatinine-modified Child–Pugh score for predicting survival in patients with decompensated cirrhosis.

Table 4.1 Child–Pugh score for prognosis in liver cirrhosis

Clinical variable	1 point	2 points	3 points
Encephalopathy	None	Grade 1–2	Grade 3–4
Ascites	Absent	Slight	Moderate/large
Bilirubin mg/dL	<34	34–50	>50
Albumin g/L	>35	35–28	<28
INR	<1.7	1.7–2.3	>2.3

INR, international normalised ratio
Child–Pugh classification serves as a measure of the severity of liver disease. The grades correlate with one and two year patient survival as follows. Grade A (5–6 points): 100% and 85%; Grade B (7–9 points): 80% and 60%; Grade C (10–15 points): 45% and 35%

Further reading

Papatheodoridis GV, Cholongitas E, Dimitriadou E, et al. MELD vs Child-Pugh and creatinine-modified Child-Pugh score for predicting survival in patients with decompensated cirrhosis. World J Gastroenterol 2005; 11:3099–3104.

Case 84: Abnormal skin pigmentation – jaundice

Instruction to the candidate

This 47-year-old man has presented with abnormal skin discolouration. Please examine and present your findings to the examiner.

Begin with a summary of positive findings

Jaundice is demonstrated by yellow pigmentation of the sclera, skin and mucosa (**Figures 4.5, 4.6,** and **4.7**).

Associated findings which can help form a suitable differential diagnosis include:

- Signs of chronic liver disease and portal hypertension (see *Portal hypertension* p. 149)) If these signs are present, signs suggesting an underlying cause of the liver disease should be sought
- Signs suggestive of acute onset liver disease – tender hepatomegaly, abdominal discomfort
- Tattoos or needle track marks which would suggest an increased risk of viral hepatitis

Follow with a summary of relevant negative findings

Important relevant negatives include:

- Signs of liver failure, such as confusion and encephalopathy. Jaundice as the result of acute on chronic liver disease, in a cirrhotic liver, may be associated with other signs of decompensation, importantly hepatic encephalopathy. Indeed, jaundice and encephalopathy serve as sensitive indicators of both severity and decompensation in chronic disease. In the context of ascites or portal hypertension one should consider jaundice as decompensation and be prepared to discuss the CPT score with the examiner (see *The syndrome of chronic liver disease,* p. 142)
- The absence of a palpable gallbladder. This mainly concerns 'surgical' jaundice but one should bear in mind Courvoisier's law which states that in the presence of a palpable gallbladder, painless jaundice is unlikely to

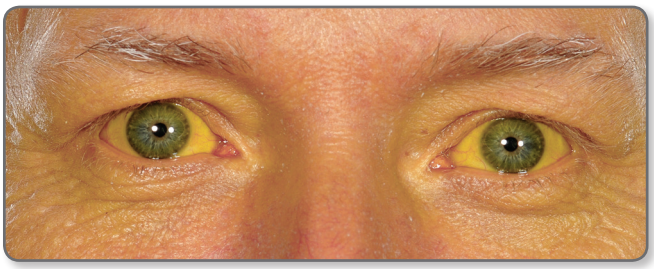

Figure 4.5 Jaundice. The normally white sclerae are noticeably discoloured with a yellow hue, scleral icterus, consistent with jaundice. There is also visible yellowing of the peri-orbital skin.

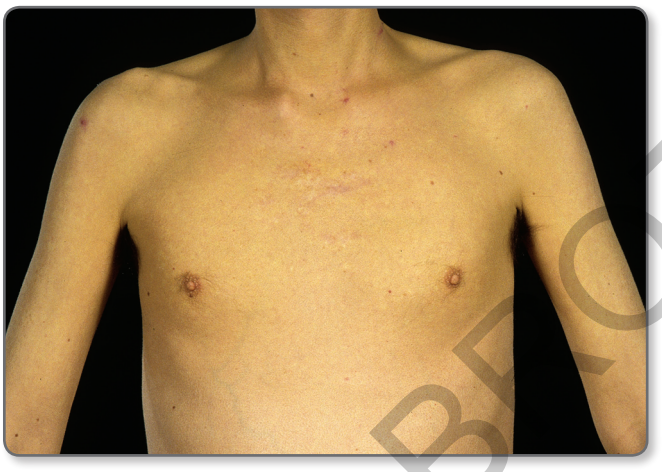

Figure 4.6 Jaundice. Gross yellowing of the skin is evident. There is also praecordial spider naevi and the paucity of body hair consistent with underlying liver disease.

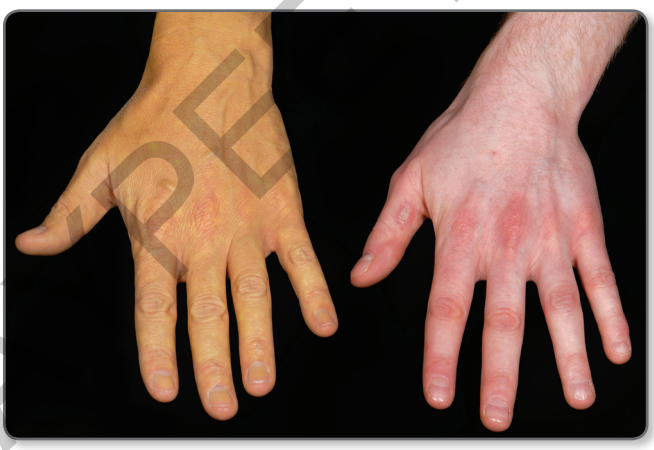

Figure 4.7 Jaundice. Jaundice may be appreciable on general inspection of the hands prior to examination of the torso and abdomen.

be due to cholelithiasis. The implication is that the clinical picture is more likely to be due to pancreatic malignancy

- Lymphadenopathy: where jaundice exists in the context of diffuse lymphadenopathy, it is important to consider viral causes and haematological malignancy

State the most likely diagnosis on the basis of these findings

A possible presentation to the examiner in the case of jaundice could be: 'This patient appears grossly jaundiced. There are no clinical signs suggestive of a clearly identifiable cause and thus I would like to take a full history and

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