Clinical assessment of the adult patient with possible liver disease: history and physical examination

CHAPTER 1

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Key points

- A thorough history and physical examination must always be the starting point in the approach to the patient with possible liver disease.
- Many individuals with chronic liver disease are asymptomatic. Except for pruritus, when symptoms are present they tend to be non-specific.
- A detailed history regarding use of prescription, non-prescription, and alternative medicines, as well as risk factors for liver disease such as obesity, high-risk sexual behavior, alcohol and illicit drug use, transfusions, piercings, tattoos, and travel, deserve special attention. Family history is essential.
- The physical examination in patients with possible liver disease requires a full routine internal medicine physical, inclusive of a search for signs compatible with cirrhosis and portal hypertension such as ascites, jaundice, hepatic encephalopathy (flapping tremor), a firm liver (normal, large, or small size), splenomegaly, spider nevi, and palmar erythema.

Introduction

The liver has an enormous functional reserve, a unique potential to regenerate, and, except for its capsule, no sensory innervation. It is therefore not surprising that symptoms and signs of both acute and chronic liver disease are only apparent when function is severely compromised.

History

When taking a history, it is important to gather information regarding symptoms and signs related to the current illness, both those specific to liver disease

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and including those that may well have occurred in the decades past. Complications of other medical issues may have occurred entirely in the past, not extending to the present. For example, in a patient presenting with new-onset ascites, who had a variceal bleed 3 years ago, and who experimented with illicit drugs 25 years ago, all these facts really belong in the history of "current" illness.

The following reviews the key elements in the history of patients with suspected liver disease.

Overt symptoms/signs of liver disease

Jaundice

Hyperbilirubinemia becomes noticeable as scleral icterus when the serum bilirubin exceeds approximately 50 μmol/L. Bilirubin is deposited in tissues including the skin. The degree of skin pigmentation, that is the ethnicity, makes skin icterus more (e.g. African American) or less (Caucasian) difficult to detect. Jaundice (hyperbilirubinemia) may be a consequence of the liver's inability to efficiently conjugate and/or excrete bilirubin into bile, or due to extrahepatic biliary obstruction, for example choledochlithiasis or pancreatic cancer. As conjugation of bilirubin is possible even when the liver is failing, the conjugated fraction predominates unless the cause is hemolysis, in which case this rarely results in visible jaundice. Hyperbilirubinemia does not allow one to discriminate between hepatocyte dysfunction and biliary obstruction.

Ascites and peripheral edema

Fluid retention with increasing abdominal girth due to ascites and/or swollen lower extremities (feet and ankles) secondary to peripheral edema is the most frequent presenting symptom of patients with cirrhosis, whereas acute liver disorders, such as hepatic vein obstruction (Budd–Chiari syndrome or right-sided heart failure), usually causes just ascites initially. Many patients will not interpret this as such, but state that their pants and shoes no longer fit and/or complain about weight gain. Fluid retention with ascites and peripheral edema is not specific for liver disease; the differential includes congestive cardiac or renal failure. In the context of liver disease, fluid retention with ascites and peripheral edema does not indicate etiology, but rather the presence of portal hypertension. Fluid retention is typically associated with processes impairing portal venous flow at the sinusoidal (cirrhosis) or postsinusoidal level (Budd–Chiari syndrome, veno-occlusive disease), but only very rarely with presinusoidal processes such as portal vein thrombosis or chronic biliary disease.

Acute liver diseases, including fulminant hepatic failure, are, at least in their initial phase, not associated with portal hypertension and are therefore not typically associated with fluid retention. Having said that, rapid onset of massive ascites, right upper quadrant pain, and mild jaundice is the classic triad of acute hepatic outflow obstruction, such as in acute Budd–Chiari syndrome, or sinusoidal obstruction syndrome following a bone marrow transplant.

Hematemesis and melena

Massive vomiting of blood is the most dramatic presenting symptom of cirrhosis. This may be a consequence of bleeding lesions in the upper GI tract secondary to portal hypertension, including esophageal or gastric varices, or less frequently portal hypertensive gastropathy or duodenal varices. In children, portal vein thrombosis is a more frequent cause of portal hypertensive bleeding than in adults. A massive portal hypertensive upper GI bleed can also lead to loss of red blood per rectum. Such massive bleeds are typically associated with hemodynamic instability and represent a serious emergency as they carry a high mortality (around 30%, depending on the severity of the underlying liver disease). Slower GI transit times and/or slower bleeds allow for (partial) degradation of hemoglobin to hematin and may present as vomiting of "coffee grounds" and/or melena. Varices are portosystemic collaterals. Less frequently, they are found in the rectum. Dilatation of small intramucosal vessels, the correlate of portal hypertensive gastropathy, can also occur in the distal colon (portal hypertensive colopathy). These lesions can cause a lower GI bleed, that is red blood per rectum, often in the absence of hemodynamic instability. Other locations where portosystemic collaterals may form include the umbilicus (caput medusae; low spontaneous bleeding risk) and the retroperitoneum (e.g. spontaneous splenorenal shunt; low spontaneous bleeding risk but high rate of portosystemic encephalopathy). In patients with an intestinal stoma, the area around the stoma (stomal varices) has a high spontaneous bleeding risk and is very difficult to manage, therefore do not advise this surgery in a cirrhotic.

It goes without saying that hematemesis and melena are by no means pathognomonic for liver disease nor for portal hypertension as they may occur secondary to a variety of lesions, including severe gastroesophageal reflux disease and Mallory–Weiss tears, gastric and duodenal ulcers, and colonic cancer, to name just a few. Of note, gastric and duodenal ulcers are more common in cirrhotic patients than in the normal population. Thus, even in the presence of known portal hypertension, any hematemesis or melena requires expedited endoscopic diagnosis of the bleeding source (and, if necessary, appropriate endoscopic therapy).

As is the case with fluid retention, portal hypertensive bleeding does not typically occur in acute liver disease. In chronic liver disease, a portal hypertensive GI bleed (hematemesis or melena) does not allow one to draw conclusions as to the etiology of the underlying cause of portal hypertension. Having said that, the presence of gastric, in the absence of esophageal, varices must raise the differential of isolated splenic vein thrombosis with left-sided portal hypertension (formation of collaterals via the short gastric veins).

Cognitive dysfunction (hepatic encephalopathy)

Patients with liver disease (or their family members) may complain about subtle cognitive dysfunction, such as difficulties in concentrating and impaired short-term memory. These and a reversal of the day–night cycle, that is

Table 1.1 Hepatic encephalopathy

Grade	Symptoms/signs
Minimal*	Subtle cognitive impairment (difficulties concentrating, impaired short-term memory); reversal of the day/night cycle
I	Lethargy (slowly thinking/speaking), asterixis† possible
II	Confusion (disorientation to time, location and/or person); asterixis† typical
III	Stupor, asterixis† often absent
IV	Deep coma, no asterixis†

*Sometimes also termed "subclinical" or "stage 0" hepatic encephalopathy.
†Asterixis or flapping tremor is the characteristic five to six times per minute tremor observed in patients with hepatic encephalopathy who are asked to hold their hands in a fixed position (with wrists pulled back, fingers spread out, and eyes closed).

day-time fatigue and night-time insomnia, are subtle symptoms of what is called minimal (stage 0) hepatic encephalopathy. Hepatic encephalopathy is a neuropsychiatric disorder of varying severity associated with liver disease and typically classified into stages 0 to IV (Table 1.1). Hepatic encephalopathy is thought to result from an imbalance of excitatory and inhibitory neurotransmitter activities in the brain. While the exact mechanism(s) remain debated, it is generally thought that gut-derived central nervous system inhibitory substances become systemically available secondary to portosystemic shunting and/or an impaired metabolic capacity of the liver (hepatic encephalopathy does not occur in those whose only cause for portal hypertension is a portal vein thrombosis). While arterial ammonia levels are typically elevated in hepatic encephalopathy, the extent of this elevation correlates poorly with the clinical picture and abnormalities in many neurotransmitters systems have been described. Hepatic encephalopathy is a diagnostic hallmark of fulminant hepatic failure. Early in the course of the disease it is often mild (stage 0–II) but often progresses rapidly to coma. The presence/absence of hepatic encephalopathy does not allow any conclusion as to the etiology of the underlying liver disease.

Other, less frequent symptoms and signs

The following symptoms and signs (described in random order) may be associated with liver disease but are less commonly reported by patients with liver disease.

Some patients with liver disease complain of non-specific, dull **abdominal discomfort**. Except for its capsule, the liver has no innervation and sharp

pain is not a typical symptom/sign of liver disease. Dull right upper quadrant discomfort/low-intensity pain can be observed in acute hepatitis and is thought to be caused by an acute enlargement of the liver, distending its capsule. Right upper quadrant discomfort is also a common complaint of patients with chronic biliary disease, for example primary biliary cirrhosis and primary sclerosing cholangitis even in the absence of gall stones. Many patients with ascites, especially when new in onset or increasing in volume, complain of bilateral discomfort/low-intensity pain in the lower abdomen; this is probably caused by distension of the abdominal wall. Patients with a massively enlarged spleen secondary to portal hypertension my rarely develop splenic infarcts, which can cause sharp left-sided abdominal pain.

Other gastrointestinal symptoms, such as **a poor appetite** (anorexia) with or without **vomiting**, are not infrequently reported by patients with liver disease (generally with acute liver disease), while **dysgeusia** (altered taste) is a rarer complaint. These symptoms are non-specific, occur in acute and chronic liver disease, and do not offer etiologic clues. Early morning nausea/vomiting (vomitus matutinus) relieved by alcohol (eye opener) is a sign of alcohol dependence.

Pruritus with or without visible scratch marks, but without a visible causative skin lesion, can be a leading presenting symptom of liver diseases, particularly in chronic cholestatic liver disorders such as primary biliary cirrhosis (PBC). Pruritus is, however, not pathognomonic for liver diseases and can be observed in many other conditions, including uremia and many skin disorders. Hepatic pruritus is not specific for PBC and is also observed with other intra- and extrahepatic causes of cholestasis. Rarely, it may even accompany viral hepatitis (particularly in a young woman taking the oral contraceptive pill). Hepatic pruritus is said to be aggravated by warmth, and many report it worsens when going to bed. The mechanism(s) for hepatic pruritus remain(s) debated. It is generally thought that endogenous substances (possibly endorphin-like) normally excreted into bile are retained in cholestasis and act on the central nervous system to cause pruritus.

Primary biliary cirrhosis may be associated with the **sicca syndrome** and/or **Raynaud phenomenon**. These patients may complain of a dry mouth and/or dry eyes, with or without frequent conjunctivitis. Additionally, they may notice that in the cold their fingers turn purple or white ("dead fingers").

Since advanced stages of acute and chronic liver diseases are often associated with decreased synthesis of coagulation factors (increased INR) and a low platelet count, complaints about easy skin **bruising**, gum **bleeding** when brushing their teeth, and/or spontaneous nose bleeds are common. As is the case for most symptoms/signs associated with liver disease, they are non-specific for the presence/etiology of liver disease.

Muscle cramps are a frequent complaint of patients with liver diseases, especially when on diuretic therapy, and are thought to be caused by electrolyte imbalance.

Most patients with liver disease, especially those with severe chronic disease, experience **impaired sexual function** with decreased libido, irregular menstrual cycles up to complete amenorrhea, and erectile dysfunction up to frank impotence. This is thought to be a result of an androgen/estrogen imbalance.

Dyspnea and/or dry **cough** are non-specific symptoms, but may be caused by hepatic hydrothorax, that is a (typically right-sided) pleural effusion associated with portal hypertension. Hepatic hydrothorax is thought to result from ascites being sucked up through preformed anatomical connections in the diaphragm. Fluid is driven into the pleural cavity by the negative pressure generated during inspiration. Hepatic hydrothorax is typically, but not necessarily, accompanied by clinically detectable ascites. In the context of liver disease, dyspnea may also be a symptom of the hepatopulmonary syndrome, that is a functional right–left shunt potentially forming in the lung circulation of patients with portal hypertension, and characterized by orthodeoxia, that is a drop in peripheral O_2 saturation in the upright position that is improved by laying down. Portopulmonary hypertension, that is pulmonary artery hypertension associated with portal hypertension, is another potential cause of, typically, exertional dyspnea in the patient with liver disease.

Apart from bacterial cholangitis associated with primary sclerosing cholangitis (PSC) and other rare conditions that cause biliary strictures (secondary sclerosing cholangitis), **fever and rigors** are not commonly observed with acute and chronic liver diseases.

Many patients with chronic liver disease complain about **feeling cold** all the time. This is thought to be related to heat loss through the skin secondary to liver-disease-related chronic vasodilatation.

Past medical history, review of systems, and family history

Many patients with liver disease will not spontaneously report the symptoms/signs described above, they may only be revealed during a thorough review of systems. A thorough past medical history and family history may also yield valuable clues in the patient with liver disease. Thus, asking for a history of **autoimmune disorders** frequently associated with autoimmune liver diseases, such as hypothyroidism secondary to autoimmune thyroiditis (Hashimoto disease) may suggest an autoimmune etiology to their liver disease. A history/presence of rheumatoid-type **joint pain** may be associated with PBC; degenerative joint pain and/or diabetes may be a hint towards hemochromatosis. A history/presence of the **metabolic syndrome, obesity**, and/or overt **diabetes** predisposes to the development of non-alcoholic fatty liver disease, which may progress to cirrhosis. Hereditary liver diseases, such as genetic hemochromatosis, α_1-antitrypsin deficiency and Wilson disease may be accompanied by a history of **liver disease/liver-related mortality in first-degree relatives**. In all individuals who test positive for hepatitis B, a detailed family history may reveal family members who have died of liver cancer.

Risk factors for liver disease

An *essential* part of the history in all patients with suspected liver disease is a detailed medication history. Many prescribed and over-the-counter medications are able to cause acute or chronic liver disease, either in a dose-dependent or a dose-independent (idiosyncratic) manner. Medications include prescription and non-prescription drugs, as well as herbal and other over-the-counter, alternative medicines.

Taking a history of elements predisposing to liver disease (risk factors for liver disease) is mandatory in all patients with suspected liver issues; these factors are summarized in Table 1.2.

Physical examination

The conduct of the physical examination in a patient with possible liver disease should not be different from a routine physical in anyone. The following highlights some specific aspects to be looked for in a patient with suspected liver disease.

General condition and vital signs. Many, but by no means all, patients with advanced chronic liver disease are **malnourished** and exhibit profound **muscle wasting** and poor dentition. Patients with cirrhosis typically have a hyperdynamic circulation, characterized by a low peripheral vascular resistance (vasodilatation) and a compensatory high cardiac output. Consequently, cirrhotic patients often have blood pressures that are below and heart rates that are above those of an age-matched, normal population.

Nervous system. Look for signs of **hepatic encephalopathy** (Table 1.2) and test for **asterixis**. Patients with alcoholic liver disease may present with signs of organic brain disease such as **Korsakow** or **alcoholic dementia**, as well as **alcohol-induced peripheral polyneuropathy**. **Wilson disease** may affect the central nervous system, its presentation ranging from **slurred speech** (dysarthria) to a **Parkinson-like syndrome** and even to frank **psychosis**.

Head and neck. Special attention is directed to the eyes for detection of a potential **scleral icterus** (see above). A **sicca syndrome** is often only detectable by quantitating tear production using a commercially available filter paper (Schirmer test). A **Kayser–Fleischer ring**, visualized as a circular copper deposit in the rim of the iris, in the context of liver disease is pathognomonic for Wilson disease. Confirmation of its presence/absence usually requires a slit lamp examination by an ophthalmologist. A **hypertrophy of the parotid**, characterized by a bilateral painless palpable enlargement of the gland, may be observed in patients with liver disease thought to be attributable to heavy alcohol consumption.

Table 1.2 Risk factors for liver disease

Risk factor	Comments
Ethnicity or geographic area of origin	Coming from high prevalence areas such as Asia/Pacific for HBV and Egypt or Pakistan for HCV predisposes to the respective chronic viral hepatitis
Sexual preference and behavior	HBV is sexually transmitted; men who have sex with men and individuals with promiscuous life styles are at increased risk of acquiring HBV infection in addition to HIV
History of incarceration or other institutionalization	Incarceration and other institutionalization is associated with a increased risk of acquiring HBV and HCV infection
Illicit drug use	A history or current use of illicit drugs by the i.v. route (or by snorting with a straw) carries an increased risk of acquiring not only HCV, but also HBV and HAV infection
Piercings and tattoos	Non-professionally performed piercings and tattoos carry the risk of HBV and HCV transmission
Transfusions (blood and blood products)	A history of transfusion of blood and blood products prior to introduction of routine HCV testing in the early 1990s is a risk factor for acquiring HCV
Alcohol consumption	Regular consumption of alcohol increases the risk for developing alcoholic liver disease; while individual susceptibility varies and there is no clear dose threshold for harmful drinking, consumption of ≥40–60 g of alcohol a day for women and ≥60–80 g of alcohol a day for men is generally thought to convey an increased risk for alcoholic liver disease About 10–20% of individuals drinking these or larger amounts of alcohol over a 10-year period develop alcoholic liver cirrhosis Small amounts may be responsible for elevated liver biochemistries
Travel history	Travel into regions of the world where HAV and HBV are prevalent combined with respective sexual or dietary risk behavior predisposes to HBV, HAV, and HEV infection, respectively

HAV, hepatitis A virus, HBV, hepatitis B virus; HCV, hepatitis C virus; HEV, hepatitis E virus.

Abdomen. Bulging flanks with shifting dullness indicate the presence of **ascites**. Ascites predisposes to the development of umbilical and inguinal **hernias**. Patients with portal hypertension often have **prominent abdominal wall veins**, which connect to the umbilical vein as part of portosystemic shunts and typically fill rapidly from caudal, less so from cranial, when effaced. This is similar to the fully developed **caput medusae**, periumbilical shunts between umbilical and abdominal wall veins (rarely observed).

Percussion of the liver in the midclavicular line allows a rough estimate of its size, which is confirmed by palpation of the liver's lower edge. The latter also gives an impression of its consistency and tenderness. In acute liver disease, the liver is often enlarged and tender. In chronic liver disease the liver is typically firm, sometimes with a palpable nodular edge. The liver may be normal in size or even enlarged, depending on the etiology of the chronic liver disease. The liver may become small and shrunken when disease is in an advanced stage. Chronic cholestatic liver diseases (e.g. primary biliary cirrhosis and primary sclerosing cholangitis) may be associated with an enlarged liver even very late into the disease evolution. An extremely firm ("hard as a rock"), indolent, and massively enlarged liver must raise the suspicion of **hepatic amyloidosis**. A hard, enlarged, grossly nodular, sometimes tender liver is suspicious for widespread **metastatic disease** or **extensive primary liver cancer**.

With large hepatocellular cancers an arterial **bruit** may be heard on auscultation over the liver. This is attributable to a high flow into the feeding artery, hepatocellular carcinomas being primarily perfused via branches of the hepatic artery. A bruit may also be heard in other situations where large arteriovenous shunts may form in the liver, such as after a liver trauma or in the rare hereditary hemorrhagic telangiectasia (Osler–Weber–Rendue disease).

In the context of liver disease, an **enlarged palpable spleen** is a sign of portal hypertensions. These sometimes extremely large spleens may reach down to the right side of the pelvis and may be missed if palpation is not started in the left lower abdomen. Portal hypertensive splenomegaly is firm and non-tender.

Chest. Examination of the chest may reveal gynecomastia, that is a mammary gland in a male patient that palpably extends beyond the area of the nipple. Gynecomastia may or may not be tender and is thought to reflect a estrogen/androgen imbalance secondary to advanced chronic liver disease. Enlarged pectoral fat pads (without enlargement of the mammary gland) is not infrequently observed in male patients with cirrhosis, especially when caused by alcoholic or non-alcoholic fatty liver disease.

In patients with massive ascites, bilaterally decreased air entry into and dullness on percussion over the basal lung fields likely reflects an **elevated diaphragm secondary to ascites**. It may, however, also indicate the presence of pleural effusions, that is **hepatic hydrothorax**. The latter may be present in the absence of clinically detectable ascites and typically, but not necessarily, is right sided or right side dominant, rather than left sided.

Apart from the signs of a **hyperdynamic circulation** (see above), which may also be associated with a functional systolic ejection murmur, examination of the heart is usually unremarkable in patients with liver disease. However, acute and chronic right and left heart failure may cause acute and chronic liver dysfunction, respectively, presenting as "shock liver" (caused by a low flow state due to acute right or left heart failure) or cardiac cirrhosis. It is therefore important to *exclude* by appropriate cardiac examination that the liver disease is not a consequence of heart disease.

Skin and nails. Spider nevi are arteriovenous shunts in the skin formed by a small central artery from which venous collaterals radiate. Upon emptying the spider nevus by applying pressure with a fingertip, the venous collaterals fill from the central small artery once pressure is removed. "Spiders" are suggestive, but not pathognomonic, for liver disease; they may occur in other states associated with a hyperdynamic circulation such as pregnancy. However, numerous spiders (more than three in women, more than two in men) are not typically seen in the absence of liver disease. They are most frequent on chest, neck, and hands, but may occur anywhere. The highest numbers are said to be found in florid alcoholic liver disease and autoimmune hepatitis. **Palmar erythema** is a blotchy, purplish-red discoloration of the thenar and hypothenar eminences and is frequently, but not exclusively, observed in chronic liver disease (it also occurs in rheumatoid arthritis). **Petechial hemorrhage** and **bruising** are consequences of thrombocytopenia, with or without a coagulopathy, commonly associated with liver disease and portal hypertension. **Scratch marks** are a reflection of severe pruritus and may be found in cholestatic liver diseases. **Vitiligo** may be associated with other autoimmune diseases, including autoimmune hepatitis. A **feminine pattern of body hair distribution** is common in men with cirrhosis and is attributed to a estrogen/androgen dysbalance. **Clubbing**, an abnormal shape of the finger nail characterized by an emergent angle of the nail of more than 180° and extreme nail convexity, sometimes forming so-called "drum stick" fingers, may, in the context of cirrhosis, be associated with the hepatopulmonary syndrome or portopulmonary hypertension. "**Terry's nails**" are a whitish discoloration of the nails to within 1–2 mm of the end. They may be observed in cirrhosis, but also in other conditions including congestive heart failure, rheumatoid arthritis, and the nephrotic syndrome.

Miscellaneous. Dupytren's contracture of a palmar tendon is not infrequently observed in individuals with alcoholic liver disease, but also occurs in the absence of alcohol misuse and liver disease, for example traumatic injury. **Testicular atrophy** occurs in men with cirrhosis and is thought to be a result of cirrhosis-related estrogen/androgen dysbalance.

Fever with or without shaking chills may be a result from biliary sepsis, for example PSC or infected ascites (spontaneous bacterial peritonitis) or a liver abscess.

Case study 1.1

A 42-year-old male construction worker, who was laid off 6 months ago, presents with anorexia, increasing abdominal girth, swollen ankles, and jaundice. His symptoms started about 4 weeks ago and have increased. Since the death of a friend in a motor vehicle accident 3 months ago, the patient reported adding three to four whiskeys to his daily alcohol consumption (six beers daily since age 16). Upon direct questioning, he admits to regular vomiting in the morning when brushing his teeth. A month ago he started to drink a shot of whiskey immediately after getting out of bed as this prevented the early morning vomiting.

Physical examination revealed profound jaundice and moderate muscle wasting. The patient was oriented three times and had no asterixis. Examination of the abdomen showed moderate ascites and hepatomegaly (measuring 18 cm in the midclavicular line). The liver was firm, and the spleen palpable to about 4 cm below the costal margin. Moderate "pitting edema" on both lower extremities was present to the knees. He had multiple, large spider nevi on the chest, marked palmar erythema, and bilateral Dupuytren's contractures.

A presumed diagnosis of acute alcoholic hepatitis superimposed on alcoholic cirrhosis was made, later confirmed with appropriate lab tests, an ultrasound, and a transjugular liver biopsy (see Chapter 8).

Multiple choice questions

1. Which of the following symptoms/signs is NOT causally related to liver disease?

- **A.** Fluid retention
- **B.** Reversal of the day/night cycle
- **C.** Circular lipid disposition in the rim of the cornea (arcus senilis)
- **D.** Dyspnea
- **E.** Fatigue
- **F.** Muscle wasting
- **G.** None of the above
- **H.** All of the above

2. Which of the following statements is correct?

- **A.** Risk factors for chronic liver disease include ALL of the following: history of recreational i.v. drug use, blood transfusions, cannabis smoking, piercings and/or tattoos, time spent in countries of the developing world.
- **B.** Tender hepatomegaly can be observed in early stages of fulminant hepatic failure.
- **C.** Splenomegaly is typically observed in acute liver disease, because enlargement of the spleen is associated with portal hypertension.

D. Spider nevi never occur in the absence of liver disease.
E. A family history of autoimmune disorders is typically observed in patients with hereditary liver diseases.
F. Many symptoms and signs of liver diseases allow precise conclusions as to the underlying etiology.
G. None of the above
H. All of the above

Answers

1. C
2. B

Further reading

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