

Chapter 18

The liver and biliary tract:

The hepatocyte

- (a) is usually diploid and uninucleate
- (b) closest to the portal tract is said to be centrilobular
- (c) has great variation in size, depending on the level of cellular activity
- (d) is supplied principally by hepatic arterial blood
- (e) stores and metabolises vitamin A

The primary diseases of the liver include all of the following except

- (a) hepatitis C
- (b) alcoholic liver disease
- (c) ascending cholangitis
- (d) hepatocellular carcinoma
- (e) hepatitis B

The most common cause for chronic liver disease in the West is

- (a) hepatitis C
- (b) alcoholic liver disease
- (c) non-alcoholic fatty liver disease
- (d) drug induced hepatitis
- (e) hepatitis B

Concerning patterns of hepatic injury

- (a) centrilobular necrosis is rare
- (b) alcoholic fatty liver affects virtually every hepatocyte
- (c) fibrotic change is generally considered reversible in the liver
- (d) necrosis is usually liquefactive, and hence causes cystic change
- (e) none of the above is true

Regarding hepatic failure

- (a) 60-70% of hepatic capacity must be eroded before hepatic failure ensues
- (b) Mortality of hepatic failure without transplantation is 60%
- (c) Paracetamol overdose is the most common cause of massive hepatic necrosis
- (d) Hepatitis C is a cause of massive hepatic necrosis
- (e) It is not a complication of pregnancy

Regarding the clinical findings in hepatic failure

- (a) patients suffering from hepatic encephalopathy are flaccid and hyporeflexic
- (b) patients are often prothrombotic
- (c) asterixis is the non rhythmic movement of the extremities
- (d) jaundice occurs in 60% of patients
- (e) hyponatraemia is a common finding in hepatorenal syndrome

Regarding hepatic failure (old paper)

- (a) 60% of hepatic capacity must be eroded before hepatic failure ensues
- (b) Encephalopathy is the result of increased ammonia formation
- (c) The liver is the predominant site of albumin synthesis

- (d) Encephalopathy is universally irreversible
- (e) none of the above is true

Regarding hepatorenal syndrome (old paper)

- (a) it is irreversible
- (b) the ability to concentrate urine is lost
- (c) the urine is high in sodium
- (d) the favoured theory is the increase in renal blood flow
- (e) the urine is hyperosmolar

Cirrhosis of the liver (old paper)

- (a) results in changes to the vascular channels
- (b) shows a basically normal liver architecture with chronic hepatocyte necrosis
- (c) rapid development of fibrosis allows for the development of large nodules
- (d) can usually be reversed if the causative agent is treated or removed
- (e) is caused by alcohol abuse in 50% of cases

Clinical features of cirrhosis include all except

- (a) osteoporosis
- (b) atrophy of the spleen
- (c) anorexia
- (d) development of hepatocellular carcinoma
- (e) oesophageal varices

In cirrhosis (old paper)

- (a) fibrosis is confined to delicate bands around the central veins
- (b) nodularity is uncommon
- (c) vascular architecture is preserved
- (d) The Ito cell is a major source of excess collagen
- (e) the left lobe of the liver is most affected

Which of the following is most correct regarding portal hypertension (old paper)

- (a) prehepatic + splenic vein thrombosis
- (b) intrahepatic + Budd Chiari syndrome
- (c) post hepatic + cirrhosis
- (d) schistosomiasis + prehepatic
- (e) none of the above

Oesophageal varices (old paper)

- (a) occur in one third of all cirrhosis patients
- (b) account for more than 50% of haematemesis episodes
- (c) are most often as a result of hepatitis C induced cirrhosis
- (d) have a 40% mortality during the first episode of rupture
- (e) are primarily in the mid portion of the oesophagus

Ascites

- (a) is clinically detectable when 200mL has accumulated
- (b) caused by cirrhosis results in a decrease in hepatic lymphatic flow
- (c) can cause a hydrothorax, usually on the left

- (d) usually has a low proteinous content (30g/dL)
- (e) with the presence of red cells points to possible disseminated intra-abdominal cancer

Bilirubin

- (a) is formed exclusively from the breakdown of senescent erythrocytes
- (b) is produced in quantities of 0.2-0.3g per day
- (c) is formed in the liver
- (d) is soluble in aqueous solution at physiological pH
- (e) is 90% resorbed as urobilinogen from the gut

Conjugated hyperbilirubinaemia results from (old paper)

- (a) Gilbert's syndrome
- (b) Physiologic jaundice
- (c) Excess production of bilirubin
- (d) Decreased hepatic uptake of bilirubin
- (e) Cholestasis

Regarding jaundice (old paper)

- (a) Unconjugated bilirubin is excreted in the urine
- (b) Excess conjugated bilirubin causes kernicterus in adults
- (c) Unconjugated bilirubin does not colour the sclera
- (d) Unconjugated bilirubin is tightly bound to albumin
- (e) Conjugated bilirubin is tightly bound to albumin

Which of the following conditions is associated with an unconjugated hyperbilirubinaemia (old paper)

- (a) Haemolysis
- (b) Rotor syndrome
- (c) Dubin Johnson syndrome
- (d) Pancreatic cancer
- (e) Ascending cholangitis

Hepatitis A

- (a) has a chronic carrier state
- (b) has an associated mortality of up to 10%
- (c) infection is not affected by alcohol consumption
- (d) has an incubation period of 2-6 weeks
- (e) is related to the hepatitis C virus

Regarding hepatitis B (old paper)

- (a) HBe antigen amounts to active replication by the virus
- (b) IgG anti Hepatitis A virus amounts to recent infection
- (c) Anti He antibody amounts to increased infectivity
- (d) it has an incubation period of 2 weeks
- (e) it is only found in the stool and blood

In hepatitis B

- (a) anti-HBs appears soon after HBsAg
- (b) infection is not associated with hepatocellular carcinoma

- (c) HBsAg appears soon after overt disease
- (d) the majority of cases of persistent infection result in cirrhosis
- (e) acute infection causes sub-clinical disease in 65% of cases

Hepatitis C (old paper)

- (a) has a high association with sexual transmission
- (b) transmission increases in pregnancy
- (c) causing fulminant hepatitis is a common presentation
- (d) infections become chronic in greater than 50% of infected patients
- (e) patients with elevated titres of anti-HCV IgG confers effective immunity

Hepatitis D

- (a) is a double stranded DNA virus
- (b) induces anti-HD surface immunoglobulin
- (c) is unable to replicate independently
- (d) does not cause fulminant hepatitis
- (e) requires hepatitis C co-infection

Hepatitis E infection (old question)

- (a) is transmitted primarily parentally
- (b) accounts for a greater than 20% mortality in pregnant mothers
- (c) is common in Russia
- (d) is associated with chronic disease and cirrhosis
- (e) is associated with an increased incidence of hepatocellular carcinoma

Regarding acute viral hepatitis infection

- (a) Acute viral hepatitis is common with Hepatitis C infection
- (b) Chronic disease results in chronic icterus
- (c) Icterus is common in adults with hepatitis A infection, but is rare in children
- (d) Most patients with acute disease have a serum sickness-like syndrome
- (e) The icteric phase of infection is associated with the worst associated systemic symptoms

Concerning biliary lithiasis p928

- (a) stones are produced exclusively in the gallbladder
- (b) 50% of stones are symptomatic
- (c) 50% are cholesterol stones
- (d) Caucasian women are twice as likely to be affected than men
- (e) Family history is not a risk factor

Concerning the pathogenesis of cholesterol stones, all the following defects are required except

- (a) Infection of the biliary tract by *E coli*
- (b) Bile must be supersaturated with cholesterol
- (c) Gallbladder hypomotility which promotes nucleation
- (d) Cholesterol nucleation acceleration
- (e) Mucus hypersecretion in the gallbladder which traps the crystals, permitting aggregation into stones

All of the following are risk factors for the formation of cholesterol stones in the gallbladder except

- (a) Obesity
- (b) Rapid loss of weight
- (c) Oral contraceptive pill
- (d) Female gender
- (e) Crohn's disease

- (a)
- (b)
- (c)
- (d)
- (e)

- (a)
- (b)
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- (a)
- (b)
- (c)
- (d)
- (e)

Answers

The hepatocyte (p878-9)

- (a) **is usually diploid and uninucleate, but older livers can have cytes that are octaploid and binucleate**
- (b) closest to the portal tract is said to be *periportal*. *Those closest to the terminal hepatic vein are said to be centrilobular*
- (c) *has minimal variation in size*, irrespective of the level of cellular activity
- (d) is supplied by *hepatic arterial blood, and mixed portal venous blood*, making them some of the most well perfused cells in the body
- (e) *perisinusoidal stellate cells* stores and metabolises vitamin A

The primary diseases of the liver include all of the following except p880

- (a) hepatitis C
- (b) alcoholic liver disease
- (c) **ascending cholangitis**
- (d) hepatocellular carcinoma
- (e) hepatitis B

The most common cause for chronic liver disease in the West is (p880)

- (a) **hepatitis C (57%)**
- (b) *alcoholic liver disease (24%) 2nd*
- (c) non-alcoholic fatty liver disease **9%**
- (d) drug induced hepatitis (*generally not a cause*)
- (e) hepatitis B (**4%**)

Concerning patterns of hepatic injury p880

- (a) centrilobular necrosis is the most common, *midzone, or periportal necrosis is rare (eclampsia)*
- (b) **alcoholic fatty liver affects virtually every hepatocyte**
- (c) fibrotic change is generally *considered irreversible in the liver*. Fibrosis with proliferating hepatocytes divided into nodules= cirrhosis
- (d) necrosis is usually *coagulative*, with some lytic necrosis
- (e) none of the above is true (*wrong*)

Regarding hepatic failure

- (a) **80-90% of hepatic capacity** must be eroded before hepatic failure ensues
- (b) Mortality of hepatic failure *without transplantation is 70-95%*
- (c) **Paracetamol overdose is the most common cause of massive hepatic necrosis (38% of all in the USA)**
- (d) Hepatitis C is **NOT** a cause of massive hepatic necrosis
- (e) a complication of *pregnancy is acute fatty liver, which can cause failure*

Regarding the clinical findings in hepatic failure p882

- (a) patients suffering from hepatic encephalopathy *are rigid and hyPERreflexic*
- (b) patients have bleeding tendencies secondary to *decreased production of factors II, VII, IX, X*
- (c) **asterixis is the non-rhythmic movement of the extremities**
- (d) jaundice occurs *ubiquitously in patients*
- (e) **Sodium retention with impaired free water excretion** is a common finding in hepatorenal syndrome

Regarding hepatic failure (old paper)

- (a) **80-90% of hepatic capacity** must be eroded before hepatic failure ensues
- (b) Encephalopathy is *associated* with increased blood ammonia levels
- (c) The liver is the predominant site of albumin synthesis, and so hypoalbuminaemia is a common finding**
- (d) Encephalopathy is *reversible if the underlying cause is treated*
- (e) none of the above is true (*wrong*)

Regarding hepatorenal syndrome (old paper) p882

- (a) it is irreversible (not stated, but the outlook is poor)
- (b) the ability to concentrate urine is *retained*
- (c) the urine is *surprisingly low* in sodium
- (d) the favoured theory is the *decrease in renal blood flow*, secondary to systemic vasodilation
- (e) the urine is hyperosmolar, devoid of proteins and sediment and low in sodium**

Cirrhosis of the liver (old paper)

- (a) results in changes to the vascular channels**
- (b) shows an *abnormal liver architecture* with areas of fibrosis and regeneration
- * (c) rapid development of fibrosis *does not* allow for the development of nodules
- * (d) is thought to be *basically irreversible*
- * (e) is caused by alcohol *in 60-70% of cases, viral hepatitis (10%), idiopathic (10-15%), biliary diseases (5%), Wilson's disease and α_1 -antitrypsin deficiency (rare)*

Clinical features of cirrhosis include all except p883-4

- (a) osteoporosis
- (b) splenomegaly, not atrophy is usual**
- (c) anorexia
- (d) development of hepatocellular carcinoma
- (e) oesophageal varices

In cirrhosis p882-3 (old paper)

- (a) fibrosis is confined to delicate bands around the central veins
- (b) nodularity *is common, unless rapid in onset*
- (c) vascular *architecture is altered*
- (d) The sinusoidal stellate cell [the Ito cell] (Davidson's 17th edition p487) is a major source of excess collagen**
- (e) the liver is *diffusely affected*

Which of the following is most correct regarding portal hypertension p884 (old paper)

- (a) prehepatic + *portal vein thrombosis, and narrowing of the portal vein before it ramifies within the liver. Splenomegaly can shunt excessive blood into the portal vein also*
- (b) intrahepatic + Budd Chiari syndrome caused by obstruction of two or more major intrahepatic veins**
- (c) *intrahepatic + cirrhosis, schistosomiasis, millitary TB*
- (d) Post hepatic + severe right sided heart failure, constrictive pericarditis and hepatic vein outflow obstruction
- (e) none (wrong)

Oesophageal varices p803, 885 (old paper)

- (a) occur in **90% of cirrhosis patients (chapter 17), 65% (chapter 18)**
- (b) account for **less than 50%** of haematemesis episodes; gastritis, Mallory Weiss, peptic ulcers collectively are more common.
- (c) are most often as a result of **alcohol induced cirrhosis**
- (d) have a 40-50% mortality during the first episode of rupture**
- (e) are primarily in the **distal portion** of the oesophagus

Ascites p884

- (a) is clinically detectable **when 500mL+** has accumulated
- (b) caused by cirrhosis results **in an increase** in hepatic lymphatic flow
- (c) can cause a hydrothorax, **usually on the Right, with longstanding ascites**
- (d) usually has a high **proteinous content (3g/dL)**
- (e) with the presence of red cells points to possible disseminated intra-abdominal cancer**

Bilirubin

- (a) is formed from the breakdown of **senescent erythrocytes, cytochrome P-450, haemolysis of abnormal erythrocytes**
- (b) is produced in quantities of 0.2-0.3g per day**
- (c) is formed **outside** the liver
- (d) is **insoluble in aqueous solution** at physiological pH
- (e) is **20%** resorbed as urobilinogen from the gut

Conjugated hyperbilirubinaemia results from (old paper)

- (a) Gilbert's syndrome (**unconjugated, impaired conjugation mechanisms**)
- (b) Physiologic jaundice (**unconjugated, impaired conjugation mechanisms**)
- (c) Excess production of bilirubin (**unconjugated**)
- (d) Decreased hepatic uptake of bilirubin (**unconjugated**)
- (e) Cholestasis (impaired bile flow), Dubin-Johnson, Rotor syndrome** (decreased hepatocellular excretion), are the only causes of conjugated hyperbilirubinaemia. Mixed pictures occur in hepatitis

Regarding jaundice p885-6 (old paper)

- (a) Unconjugated bilirubin **cannot be excreted in the urine, even when plasma levels are high**
- (b) **Excess unconjugated bilirubin** causes kernicterus (damage to the basal ganglia and cerebral cortex in premature babies) **in neonates, prevented by rhesus incompatibility testing and treatment**
- (c) **Both unconjugated and conjugated** bilirubin colours the sclera (**icterus**)
- (d) Unconjugated bilirubin is tightly bound to albumin**
- (e) Conjugated bilirubin is **non-toxic, water soluble and loosely bound to albumin**

Which of the following conditions is associated with an unconjugated hyperbilirubinaemia p887 table 18-3(old paper)

- (a) Haemolysis, haemolytic anaemia, resorption of GI bleeding, pernicious anaemia, thalassaemia, Crigler-Najjar syndrome, Gilbert syndrome (some cases), breast milk jaundice, physiologic jaundice of the newborn, drug & viral induced hepatitis, cirrhosis**

- (b) Rotor syndrome (*conjugated*)
- (c) Dubin Johnson syndrome (*conjugated*)
- (d) Pancreatic cancer (*impaired bile flow:- conjugated*)
- (e) Ascending cholangitis (*impaired bile flow:- conjugated*)

Hepatitis A

- (a) **does not have a** chronic carrier state, and blood-borne transmission is rare, hence blood is not routinely screened.
- (b) has an associated mortality of **0.1%**, **and is generally a self limiting benign disease**
- (c) infection is **more severe in the setting of concurrent alcohol consumption**
- (d) has an incubation period of 2-6 weeks**
- (e) **has its own genus (hepatovirus)**

It is shed in the stool for 2-3 weeks before and one week after the onset of jaundice. Sporadic outbreaks occur due to the consumption of shellfish contaminated with human faeces. Faecal shedding ends as the IgM titre against the virus rises.

Regarding hepatitis B (old paper) p892-3

- (a) HBe antigen, HBV-DNA, and DNA polymerase appear in the serum soon after HBsAg, and all signify active viral replication**
- (b) HBsAg appears **before overt disease**
- (c) Anti H_e antibody appears soon after the disappearance of **HBeAg, and indicates that the infection is on the wane**
- * (d) it has a long incubation period (4-26 weeks)
- * (e) it is found in all **physiological and pathological body fluids, and is present in the stool**

In hepatitis B pp892-4, figure 18-9 (old paper)

- (a) anti-HBs appearance **signals the end of the disease**, and is not detectable for weeks to months after the disappearance of HBsAg. Persistent HBsAg for 6/12 after detection signals a carrier state
- (b) infection **is associated** with hepatocellular carcinoma (0.02% of **all** infections)
- (c) HBsAg **appears in the blood just before overt disease**
- (d) the majority of cases of persistent infection **result in resolution (67-90%), 10-33% go on to cirrhosis and hepatocellular carcinoma**
- (e) acute infection causes sub-clinical disease in 65% of cases**

Hepatitis C pp894-5(old paper)

- (a) has a **low** association with sexual transmission (12 events/ 1000 person years in the sexual partners of HCV infected patients)
 - (b) transmission is **low in pregnancy (6%)** compared with Hep B (20-60%)
 - * (c) causing fulminant hepatitis is a common presentation
 - (d) infections become chronic in 85% of infected patients**
 - * (e) patients with high levels of anti-HCV IgG **does not** confer effective immunity
- In the acute infective phase anti-HCV antibodies are detected in 70% of patients. The disease is milder than Hep B. Episodic elevations in serum aminotransferases is characteristic of chronic infection.

Hepatitis D p896

- (a) *is a single stranded RNA virus*
- (b) induces *anti-HDV immunoglobulin*, as it does not have a surface antigen, using HBsAg for that
- (c) is unable to replicate independently, and requires co-infection with Hep B**
- (d) causes fulminant hepatitis in *3% of previously healthy individuals*, and those HBV carriers, *it induces fulminant hepatitis in 10%*
- (e) requires *hepatitis B* co-infection, either superimposed, or concomitant

Hepatitis E infection p896-7(old paper)

- (a) is transmitted primarily *enterically, and is water-borne*
 - (b) accounts for up to 20% mortality in infected pregnant mothers**
 - * (c) is common in *India, sub-Saharan Africa and Mexico*
 - * (d) *is not associated* with chronic disease and cirrhosis, and is self limiting
 - * (e) *is not associated* with an increased incidence of hepatocellular carcinoma
- It is detectable in stool and liver clinically before the onset of disease.

Regarding acute viral hepatitis infection p898

- (a) Acute viral hepatitis is *not* common with Hepatitis C infection
- (b) Chronic disease *can result in transient jaundice*
- (c) Icterus is common in adults with hepatitis A infection, but is rare in children**
- (d) *10% of patients with acute disease (mostly hepatitis B)* have a serum sickness-like syndrome
- (e) The icteric phase of infection is associated *with improvement in associated systemic symptoms*

Four states: incubation, symptomatic pre-icteric, symptomatic icteric, convalescence. Table 18.3 states that viral hepatitis causes primarily unconjugated bilirubinaemia, and p898 states that it causes a conjugated bilirubinaemia.

Concerning biliary lithiasis p928

- (a) stones are produced in the gallbladder, *and anywhere in the biliary tree*. Asians tend to produce biliary tree stones
- (b) *20% of stones are symptomatic*
- (c) *80%* are cholesterol stones, the rest are bilirubin calcium salts (pigment stones)
- (d) Caucasian women are twice as likely to be affected than men**
- (e) Family history *is a risk factor*

Concerning the pathogenesis of cholesterol stones, all the following defects are required except

- (a) Infection of the biliary tract by *E coli*, causes pigment stones (bilirubin)**
- (b) Bile must be supersaturated with cholesterol
- (c) Gallbladder hypomotility which promotes nucleation.
- (d) Cholesterol nucleation acceleration
- (e) Mucus hypersecretion in the gallbladder which traps the crystals, permitting aggregation into stones

All of the following are risk factors for the formation of cholesterol stones in the gallbladder except p929

- (a) Obesity
- (b) Rapid loss of weight
- (c) Oral contraceptive pill, pregnancy
- (d) Female gender, advancing age, hyperlipidemia syndromes. 20% radio-opaque
- (e) Crohn's disease, haemolysis, ileal disease, cystic fibrosis with pancreatic insufficiency cause pigment stones. 50-75% are radioopaque**