**Question 1**

An elderly patient presents to the emergency department complaining of severe abdominal pain, vomiting, and bloody diarrhoea, absent bowel sounds. What is the most likely diagnosis?

A Ischaemic bowel disease

B Bowel obstruction

C Appendicitis

D Gastroenteritis

Explanation A

Ischaemic bowel disease tends to occur in older patients with coexisting cardiac disease and vascular disease. Acute transmural infarction typically presents with the above clinical picture and may progress rapidly to shock and vascular collapse within hours. Due to these physical signs overlapping with other GIT disorders including perforated gastric ulcer, cholecystitis appendicitis, delay in diagnosis can occur. The pathogenesis occurs in two phases. The initial hypoxic phase occurs at the onset of the vascular compromise. While some damage occurs at this stage, the epithelial cell lining of the intestine is relatively resistant tot transient hypoxia. The second phase, reperfusion injury, is initiated by restoration of blood flow and it is at this time that the GREATEST damage occurs. (Mechanism: free radicals, neutrophil infiltration, and inflammatory mediators)

**Question 2**

Regarding peptic ulcer disease (PUD), which is FALSE?

A The ulcer in PUD has normal gastric mucosa adjacent to it, as opposed to stress ulcer which has abnormal mucosa adjacent to it

B PUD is most often associated with H.pylori-induced hyperchlorhydric chronic gastritis

C H. pylori induced ulcers occur more in the duodenum than in the stomach

D PUD may occur in any portion of the GIT exposed to gastric juices

Explanation A

Peptic ulcer disease (PUD) is most often associated with H.pylori-induced hyperchlorhydric chronic gastritis, which is present in 85% to 100%of individuals with duodenal ulcers and in 65% with gastric ulcers. The presence of chronic gastritis can help to distinguish PUD form acute erosive gastritis or stress ulcers, since the mucosa adjacent to the ulcer is generally normal in the latter two conditions. PUD may occur in any portion of the GIT exposed to gastric juices, but is more common in the gastric antrum and the first portion of the duodenum. PUD may occur in the oesophagus as a result of GORD (gastro-oesophageal reflux disease) or acid secretion by ectopic gastric mucosa. Gastric mucosa within a Meckel diverticulum can result in peptic ulceration of adjacent mucosa

**Question 3**

Which of the following hepatitis infections does not cause chronic liver disease?

A Hepatitis B

B Hepatitis C

C Hepatitis D

D Hepatitis A

Explanation D

Hepatits infections

Frequency of chronic liver disease (hepatitis)

A: NEVER

B: 10%

C: about 80%

D: 5% (co-infection), <70% for superinfection.

E: NEVER

**Question 4**

Conjugated hyperbilirubinaemia results from which of the following options?

A Excess production of bilirubin

B Decreased hepatic uptake

C Cholestasis

D Gilbert's syndrome

Explanation C

Conditions which lead to an obstruction of the biliary ducts, both intra-hepatic and extra-hepatic, and a decrease in excretion of bilirubin glucuoronides will lead to conjugated hyperbilirubinaemia. Hereditary conjugated hyperbilirubinaemia syndromes include Dubin Johnson and Rotor syndrome

**Question 5**

In relation to jaundice, which of the following statements is correct?

A Unconjugated bilirubin does not colour the sclera

B Conjugated bilirubin produces kernicterus in adults

C Unconjugated bilirubin produces bilirubin in the urine

D In unconjugated hyperbilirubinaemia, bilirubin is tightly bound to albumin

Explanation D

Unconjugated bilirubin is insoluble and tightly bound to albumin. It cannot be excreted in the urine but is deposited in tissues giving rise to the yellow discolouration of the skin and sclera. Haemolytic disease of the newborn may lead to the accumulation of unconjugated bilirubin in the brain, which can cause severe neurological damage called kernicterus. Conjugated bilirubin is soluble and loosely bound to albumin

**Question 6**

Which is the most common route of Hepatitis C transmission?

A Unknown

B Employment in medical or dental fields

C Multiple sex partners

D Having surgery within the last six months

Explanation C

IVDU drug abuse 54%

Multiple sex partners 36%

Having surgery within the last six months 16%

Needle stick injury 10%

Multiple contacts with a HCV infected person 10%

Employment in medical or dental fields 1.5%

Unknown 32%

**Question 7**

Which of the following statements is correct regarding hepatic failure?

A It is not associated with ascites

B Encephalopathy is a result of decreased ammonia breakdown

C It occurs with loss of functional liver capacity of approximately 60%

D It has a mortality of <10%

Explanation B

Whatever the cause, 80-90% of hepatic function must be eroded before hepatic failure ensues. Overall mortality from hepatic failure is 70-95% (Current textbook says 80% mortality). Clinical features include jaundice, cerebral compromise due to hyperammonaemia as a result of impaired liver function and of shunting blood around the liver (muscle wasting-a common feature in liver disease patients can also contribute to increased levels of ammonia as muscle is an important site for the extrahepatic removal of ammonia), foetor hepaticus, ascites, palmar erythema, spider angiomata, gynaecomastia and hypogonadism

Note: Ammonia formation is not increased in liver failure. Encephalopathy is due to decreased hepatic breakdown of ammonia

**Question 8**

Regarding liver failure, which of the following statements is correct?

A Can be caused by tetracyclines

B Rarely results from cirrhosis

C Is not associated with ascites

D Has a 20-40% mortality

Explanation A

Whatever the cause, 80-90% of hepatic function must be eroded before hepatic failure ensues. Overall mortality from hepatic failure is 70-95%. Clinical features include jaundice, cerebral compromise due to hyperammonaemia as a result of low albumin levels, foetor hepaticus, ascites, palmar erythema, spider angiomata, gynaecomastia and hypogonadism. The ultimate mechanism of most cirrhotic deaths is progressive liver failure. Tetracyclines can produce idiosyncratic reactions including liver failure.

**Question 9**

With regard to jaundice, which of the following options is correct?

A Conjugated bilirubin is tightly bound to albumin

B Unconjugated bilirubin does not colour the sclera

C Unconjugated bilirubin is tightly bound to albumin

D Unconjugated hyperbilirubinaemia will result in bilirubin in the urine

Explanation C

Unconjugated bilirubin is insoluble and tightly bound to albumin. It cannot be excreted in the urine but is deposited in tissues giving rise to the yellow discolouration of the skin and sclera. Haemolytic disease of the newborn may lead to the accumulation of unconjugated bilirubin in the brain, which can cause severe neurological damage called kernicterus. Conjugated bilirubin is soluble and loosely bound to albumin

**Question 10**

Which of the following does not cause fatty liver?

A Obesity

B Alcohol

C Protein malnutrition

D Smoking

Explanation D

Alcohol, obesity, vitamin A deficiency, starvation, diatetes mellitus (DM), corticosteroids, poisons (carbon tetrachloride and yellow phosphorus), Cushing’s syndrome, and hyperlipidaemia are some of the cause of fatty liver. Microvesicular fatty liver may be caused by valproic acid toxicity, high-dose tetracycline or during pregnancy.

**Question 11**

Regarding cirrhosis of the liver, which of the following statements is correct?

A The Ito cell is a major source of excess collagen

B Vascular architecture is preserved

C Fibrosis is confined to the delicate bands around central veins

D The left lobe of the liver is most affected

Explanation A

Cirrhosis is defined by three characteristics: fibrous septa (collagen depostion), parenchymal nodules and disruption of the architecture of the liver. Vascular architecture is reorganised by the parenchymal damage and scaring. The major source of excess collagen in cirrhosis appears to be perisinusoidal hepatic stellate cells (Ito cells). The right lobe is bigger and thus more affected.

**Question 12**

Which of the following statements is correct regarding oesophageal varices?

A Are most often associated with cirrhosis due to hepatitis C

B Occur in approximately one third of all patients with cirrhosis

C Lie primarily in the middle portion of the oesophagus

D Additional instances of haemorrhage occur in over 50% within one year

Explanation D

Current TB

Oesophageal varices are present in nearly half of patients with cirrhosis and 25-40% of these develop variceal bleeding. Approximately 12% of previously asymptomatic varices bleed each year. Despite any intervention, 30% or more patients with variceal haemorrhage die as a direct consequence of haemorrhage. Furthermore, 50% of patients who survive the first variceal bleed will have another one within a year and this carries a mortality rate similar to the first bleed

Older edition

Oesophageal varices occur in 90% of cirrhotic patients (GIT chapter), especially when the condition is due to alcohol. Varices account for less than half of the cases of haematemesis. Hepatitis C bears a small association as some cases develop cirrhosis. The varices lie primarily in the lower portion of the oesophagus. Among those patients who survive (first rupture), additional instances of haemorrhage occur in over 50% within one year

Note: in the liver and biliary chapter, there is mention of oesophageal varices that appear in 40% of patients with advanced cirrhosis of the liver and cause massive haematemesis and death in about half of them. (Go figure). Also note the information in the new TB above. I have however left the question as is

**Question 13**

Which of the following may occur in acute pancreatitis?

A Glycosuria in 10% of cases

B Hypercalcaemia

C Metabolic alkalosis

D Serum levels of amylase increase before levels of lipase

Explanation A

Acute pancreatitis is a relatively common condition with an incidence rate of 20 per 100000 cases in the western world. 80% of cases are associated with 2 conditions; biliary tract disease and alcoholism. Gallstones are present in 35-60% of cases of pancreatitis, and about 5% of patients with gallstones develop pancreatitis.

Male to female ratio 1:3.

Alcohol as a cause differs depending on country; USA up to 65% of cases and in Sweden 20%. Other causes include: infection, drugs, hypercalcaemic states, hyperparathyroidism and acute ischaemia. 10-20% of cases are idiopathic.

Laboratory findings include a metabolic acidosis. Serum lipase elevations occur earlier and last longer than elevations in amylase and are therefore useful in patients who present after 24hrs. Serum lipase is also more sensitive as compared with amylase in pancreatitis secondary to alcohol. Hypocalcaemia may arise from precipitation of calcium soaps in the fat necrosis. Glycosuria occurs in 10% of cases. Pancreatitis results from autodigestion of the pancreas by activated pancreatic enzymes. The mechanisms by which activation of pancreatic enzymes is initiated are: pancreatic duct obstruction, primary acinar cell injury and defective intracellular transport of proenzymes within acinar cells.

One of the major exocrine enzymes produced by the pancreas is trypsin. It can activate other proenzymes and converts prekallikrein to its activated form, initiating the kinen system.

NOTE: S-lipase rises within 4 to 8 hours of the onset of symptoms, peak at 24hrs, and return to normal within 8-14days. S-amylase rises within 6-12hours of the onset of acute pancreatitis. Amylase has a short half-life of 10hrs and in uncomplicated pancreatitis, returns to normal levels within 3-5days.

The current text states that there is a marked elevation of serum amylase levels during the first 24hrs, followed within 72-96hrs by a rising serum lipase. This is a very broad statement. The note about serum markers above is correct, just form a different source

**Question 14**

Regarding acute pancreatitis, which of the following statements is true?

A 35% of patients with gall stones develop pancreatitis

B Less than 5% are idiopathic

C Trypsin activates prekallikrein to its active form

D Gall stones are present in 80% of cases

Explanation C

See Q13

**Question 15**

Which of the following is a result of chronic pancreatitis?

A Hypermagnesaemia

B Hypercalcaemia

C Hypoglycaemia

D Steatorrhoea

Explanation D

Chronic pancreatitis is defined as inflammation of the pancreas with irreversible destruction of exocrine parenchyma, fibrosis and at a later stage destruction of endocrine parenchyma. Patients with chronic pancreatitis usually present with persistent abdominal pain or steatorrhoea. Steatorrhoea is due to the malabsorption of fats. The development of steatorrhoea occurs late in the disease process and is a manifestation of severe exocrine deficiency. Diabetes is a common complication of chronic pancreatitis. Pseudocysts are also common. Note that hypercalcaemia and hyperlipidaemia can both cause chronic pancreatitis.

**Question 16**

Regarding acute pancreatitis, which of the following statements is correct?

A Backflow of bile is a significant risk factor

B Alcohol and gallstones cause 60%

C It affects intraperitoneal fat only

D Intraductal activation of enzymes is an important aetiology

Explanation D

Pancreatitis results from autodigestion of the pancreas by activated pancreatic enzymes. The mechanisms by which activation of pancreatic enzymes is initiated are: pancreatic duct obstruction, primary acinar cell injury and defective intracellular transport of proenzymes within acinar cells.

The TB refers to the activation of pancreatic enzymes via three potential unclear mechanisms. Pancreatic duct obstruction is one way, but the explanation refers to enzyme rich interstitial fluid and lipase which is already active. I do not feel that this mechanism is well explained

One of the major exocrine enzymes produced by the pancreas is trypsin. It can activate other proenzymes and converts prekallikrein to its activated form, initiating the kinen system.

**Question 17**

Regarding acute pancreatitis, which of the following options is correct?

A Gallstones are present in 80% of cases

B 35% of patients with gallstones develop pancreatitis

C Less than 5% of cases are idiopathic

D Trypsin plays a central role in the activation of the kinin system

Explanation D

See Q13

**Question 18**

Complications of alcoholic chronic pancreatitis include the following except?

A Pancreatic cancer is common

B Severe chronic pain can be the dominant problem

C About 10% of patients develop pseudocysts

D There is a 20-25 year mortality rate of 50%

Explanation A

While patients with hereditary pancreatitis have a 40% lifetime risk of developing pancreatic cancer, the degree to which other forms of pancreatitis predispose to the development of pancreatic cancer is unclear. There is only a modest increased risk of pancreatic carcinoma in patients with pancreatitis, due to the risks of smoking and alcohol consumption rather than a casual role of chronic pancreatitis per se

**Question 19**

All of the following are acute effects relating to acute pancreatitis, with the exception of?

A Renal failure

B Diabetes mellitus

C DIC

D Acute respiratory distress syndrome (ARDS)

Explanation B

Diabetes mellitus is not an acute effect of pancreatitis but rather the result of chronic pancreatic inflammation. Glycosuria occurs early in acute pancreatitis

Note: pseudocysts can develop as a result of an acute pancreatitis episode. It takes about 4 weeks to develop. They are not solely a result of chronic pancreatitis

**Question 20**

Which of the following statements is correct in relation to pancreatitis?

A The chronic form is usually due to gallstones

B Trypsin is implicated as an activator of the kinin system

C The second most common cause is infectious agents

D Duct obstruction is not the causal mechanism in alcoholic pancreatitis

Explanation B

The mechanisms by which activation of pancreatic enzymes is initiated are: pancreatic duct obstruction, primary acinar cell injury and defective intracellular transport of proenzymes within acinar cells.

One of the major exocrine enzymes produced by the pancreas is trypsin. It can activate other proenzymes and converts prekallikrein to its activated form, initiating the kinen system.

Extra: according to UpToDate: "Alcohol may act by increasing the synthesis of enzymes by pancreatic acinar cells to synthesize the digestive and lysosomal enzymes that are thought to be responsible for acute pancreatitis or over-sensitization of acini to cholecystokinin. However, the exact mechanism of pancreatic injury, the genetic and environmental factors that influence the development of pancreatitis in alcoholics, and the reason why only a small proportion of alcoholics develop pancreatitis, are unclear."