ACCESSORY ORGANS OF DIGESTION

(CHAPTER 43-44)

QUESTION 1

List the functions of the liver.

The liver does a **lot**!

- produces **bile** to aid with fat absorption
- manufactures proteins, such as **albumin** and **clotting factors** II, V, VII, IX, X, XI, and XII
- stores and **recycles red blood cells**

- stores **iron** and **fat-soluble vitamins** (A, D, E, K)
- stores **triglycerides** and **cholesterol**, and exports them to the blood via lipoproteins
- breaks down **tons** of chemicals in the blood that are too big to be immediately filtered by the kidneys

QUESTION 2

Describe portal hypertension.

Quick review of A&P:

The GI tract **absorbs nutrients** from the intestinal lumen and dumps them into the venous circulation

The veins coming from the **GI tract**, **pancreas**, and **spleen** all converge to the **hepatic portal vein**.

The HPV carries nutrients to the liver, where they are initially processed before passing on to the heart.

If bloodflow **through** the liver is impeded due to scar tissue, blood will get backed up into the HPV and its internal pressure will increase.

Important: Portal hypertension is **closely linked** with **cirrhosis**. It is caused by the liver becoming **less permeable** to bloodflow due to the **scarring** associated with advanced liver disease.

QUESTION 3

What are the consequences of long-term portal hypertension?

Gastroesophageal varices are varicose veins **inside** the upper GI tract, caused by portal hypertension.

These varices can cause bleeding into the upper GI tract, presenting as **hematemesis**.

caput medusae – distended, "snake-like" veins visible on surface of abdomen

ascites – remember the unit on fluid balance? Venous obstruction → increased CHP → edema.

splenomegaly – increased venous pressure causes swelling of the spleen

QUESTION 4

Describe the types of viral hepatitis.

There are technically a total of **six** types of viral hepatitis: **A**, **B**, **C**, **D**, **E**, and **G**

The main three to know are **A**, **B**, and **C**, as these are by far the most common.

HAV (hepatitis A virus) is unique in that it spreads through the **fecal-oral** route, rather than bloodborne transmission.

HBV and **HCV** spread through contact with bodily fluids, such as blood, and often become chronic if exposed.

HDV is relatively rare because it **only** occurs in those who are also infected with **HBV**.

HEV spreads through contaminated water and is very rare in developed countries.

HGV is not believed to cause severe disease in humans at all.

QUESTION 5

Which type of viral hepatitis is most likely to become chronic?

Hepatitis **C** (HCV) is the most likely to progress to chronic disease, although chronic hepatitis **B** (HBV) is also fairly common.

Hepatitis A is typically **self-limiting** which means that it causes acute infection, but never gets to the point that the immune system can't deal with it.

A good mnemonic to remember this is that "A is acute" while "B and C can BE chronic."

QUESTION 6

Describe the phases of acute hepatitis.

The **prodromal phase** of acute hepatitis infection is characterized by:

- Anorexia (loss of appetite) and weight loss
- Nausea, vomiting, and diarrhea
- Diffuse arthralgias (joint pain)

Rembember what **prodromal** means from the "Foundations" unit—**vague**, **non-specific** symptoms!

The **icteric phase** of acute hepatitis infection is characterized by:

- Jaundice ("icterus" means jaundice)
- More unconjugated bilirubin → darker urine
- Less conjugated bilirubin → lighter stool

QUESTION 7

Describe cirrhosis.

Cirrhosis is the **gradual**, **chronic scarring** of the liver due to long-term hepatic injury.

Damage to hepatocytes results in the parenchyma (functional tissue) inside the liver being gradually replaced by scar tissue in a process known as **fibrosis**.

This scar tissue buildup is the reason for the decrease in bloodflow that causes **portal hypertension**.

QUESTION 8

Is all cirrhosis caused by alcoholism?

No! Chronic liver inflammation of **any** kind can eventually lead to cirrhosis given enough time, especially if untreated.

This includes viral hepatitis, non-acoholic steatohepatitis (NASH,) autoimmune hepatitis (AIH,) and all sorts of liver diseases not caused by alcohol abuse.

QUESTION 9

Where does biliary cirrhosis (biliary cholangitis) begin?

Biliary cirrhosis (or **biliary cholangitis**, meaning bile duct inflammation) first develops in the distal ends of the bile duct tree, called the **canaliculi**.

From there, the inflammation typically spreads proximally, towards the common hepatic duct.

QUESTION 10

What is the hallmark of primary biliary cholangitis?

Primary biliary cholangitis (PBC) is a rare **autoimmune condition** which is classically associated with the presence of **antimitochondrial antibodies**, an autoantibody that targets the mitochondria of cells.

It results in the gradual **destruction of the biliary tree**, with severe cases eventually progressing to cirrhosis and end-stage liver failure requiring transplantation.

QUESTION 11

Describe the two types of biliary cirrhosis (biliary cholangitis.)

Primary biliary cholangitis (PBC,) mentioned in the last question, is **autoimmune** and has no cure, although some degree of treatment is possible.

Secondary biliary cirrhosis (SBC) presents similarly, but the inflammation is caused by chronic **bile duct blockage** for **other reasons**, such as a tumor or gallstones.

QUESTION 12

Describe post-necrotic cirrhosis.

Post-necrotic cirrhosis is a potential **complication** or **sequela** of viral hepatitis infection, in which large areas of **necrotic cells** killed off during the infection are replaced with **scar tissue**.

QUESTION 13

What is cholelithiasis?

Cholelithiasis simply refers to the presence of **gallstones** somewhere in the biliary tract (gallbadder and bile ducts.)

Gallstones are solid masses made primarily of **cholesterol** that usually form within stagnant bile in the gallbladder.

Cholelithiasis is **often asymptomatic** as gallstones are able to sit undisturbed in the gallbladder for long periods of time.

When they do become problematic, one of the main symptoms is **biliary colic** (RUQ abdominal pain and tenderness.)

The diagnosis of cholelithiasis can be made with an abdominal ultrasound.

Related terms that you may also see include:

choledocholithiasis – occurs when gallstones leave the gallbladder and become **stuck** or lodged in the common bile duct

cholecystitis – **gallbladder inflammation**, commonly caused by the presence of gallstones and sometimes requiring gallblader removal (cholecystectomy or "chole" for short)

QUESTION 14

Describe the two types of gallstones.

Gallstones are typically made mostly of **cholesterol**, hence why high cholesterol is a risk factor.

Process begins with excretion of **cholesterol-supersaturated** bile.

This enables the **nucleation** of cholesterol **crystals** to occur over time.

Combined with **hypomotility** (impaired bile flow,) crystals can coalesce to form gallstones.

Pigment stones are different from normal cholesterol stones, and represent only about 20% of gallstones.

Their pigment comes from a high **bilirubin** content.

QUESTION 15

Compare acute and chronic pancreatitis.

Acute and chronic **pancreatitis** are similar in presentation. Both can cause:

Epigastric/LUQ pain, often radiating to the back

Severe **tenderness** on palpation

Nausea and **vomiting**

Abdominal distension and **hypoactive** bowel sounds

Both are also most commonly caused by **chronic alcohol abuse**. Sometimes acute pancreatitis resolves quickly, but other times it may **progress** to chronic disease.

Ironically, one of the main differences is that chronic pancreatitis typically produces pain that is **less severe** than acute pancreatitis.

QUESTION 16

Describe esophageal cancer. List some risk factors.

Esophageal cancer is strongly linked with poorly controlled **gastroesophageal reflux disease** (GERD) due to the inflammation it causes to the esophagus.

Other risk factors include **tobacco use**, **alcohol abuse**, and **obesity**.

Symptoms of esophageal cancer, if any, may include **abdominal pain**, **dysphagia** or **odynophagia**, **loss of appetite**, as well as upper GI bleeding resulting in **hematemesis** or **melena**.

QUESTION 17

Describe stomach cancer. List some risk factors.

The major risk factor for stomach cancer is **chronic gastritis**, as well as its major risk factor, **H. pylori infection**.

Symptoms are similar: **pain**, **dysphagia/odynophagia, loss of appetite, and upper GI bleeding.**

QUESTION 18

How common is colorectal cancer? List some risk factors.

Colorectal cancer is the **4th** most common type of cancer in the U.S., but the **2nd** by mortality.

Obesity, **sedentary lifestyle**, **diet** (red meats, etc.,) **tobacco use**, and **alcohol abuse** are all risk factors, as are a history of **polyps** or **inflammatory bowel disease** (IBD.)

QUESTION 19

What is the usual cause of liver cancer? List some risk factors.

Hepatocellular carcinoma (true "liver cancer") is not actually that common; more often, cancer in the liver will be a metastasis from advanced cancer elsewhere in the body.

Keep in mind that this isn't actually "liver cancer," though, because it doesn't **start** in the liver. We'll talk about this more in unit 23.

Risk factors include:

Male sex, **African-American** race

Chronic hepatitis B/C infection

Cirrhosis, including PBC or NASH

Liver fluke (flatworm) infection

QUESTION 20

Which sex is more likely to develop gallbladder cancer?

Two-thirds of gallbladder cancer cases occur in **women**.

This coincides with the fact that **gallstones** are also more common in women by a roughly 2:1 ratio.

QUESTION 21

How common is primary gallbladder cancer?

Gallbladder cancer is **very rare**, and usually only occurs in those with a past history of gallstones, meaning it is likewise more common in women.

QUESTION 22

How does pancreatic cancer rank as a cause of mortality?

Pancreatic cancer has an **extremely poor** survival rate, with only 20% of patients even surviving one year past diagnosis.

This is attributable to the fact that pancreatic cancer is **hard to detect**, leading to most cases being caught after the cancer has already metastasized.