430 MEDICINE NOTES

COMPLICATIONS OF LIVER CIRRHOSIS

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Complications of liver cirrhosis

- Continuous inflammation of the liver > liver tries to heal through continuous scarring > fibrosis > Cirrhosis (entire damage of liver = end-stage)
- Cirrhosis is a chronic liver disease characterized by fibrosis, disruption of the liver architecture and widespread nodules in the liver.
- Causes include alcoholic liver disease, fatty liver, hepatitis, drugs (acetaminophen toxicity, methotrexate) and ischemia.

Portal Hypertension (PHT):

- A large amount of blood flows through the portal system to the liver.
- If the liver is damaged then the hepatocytes surrounding the sinusoids will block the flow of blood through the sinusoids resulting in the backflow of blood to the hepatic vein.
- Hepatocellular dysfunction >> Portal Hypertension (PHT)
- If this system is blocked for any reason, the blood needs to find a way back to the heart, and so it reverses flow back down to the portal system into other veins to do so. (system overflowing)

Varices:

- The involved veins include the splenic vein, umbilical vein, hemorrhoidal veins and esophageal/gastric veins.
- These veins are not accustomed to handling such large blood volume and they dilate (varices).
- Since dilated vein walls are more fragile and since these varices are under high pressure they can rupture causing esophageal bleeding.
- Blockage in liver > PHT > Blood goes to areas of low resistance > Back flow > Veins dilate (varices) > Bleeding could occur
- The backup into the following structure results in:
    - In Spleen > enlarges > over does its job (hypersplenism) > removal of blood elements > anemia
    - Umbilical veins > Caput medusa (on Physical Exam)
    - Hemorrhoidal veins around the anus >enlarge > presents as hemorrhoids
    - Esophageal & gastric (varices) can rupture > causing a massive bleed In the esophagus
    - It manifests as: hematemesis + melena > patient could go into shock

Management

- ABC
- Airway, Breathing > make sure the pt is breathing,
- Circulation > Resuscitate pt by giving him/her fluids (2 IV lines of saline). Never wait until the blood comes from the blood bank. (type & cross match)

Rx:

1. **Octreotide:** splanchnic VC > helps stop the bleeding
2. **Variceal Banding:** Scope with band at end > stops bleeding and prevents further bleeding
3. **Shunts:** through JV to find a large hepatic vein and put a metal stent (lowers portal pressure by 20-3 0%) = **TIPS** >> used either because there's active bleeding or when unresponsive to treatment

Prevention:

- Treat underlying cause (always the first step)
- Endoscopic banding protocol
- Beta-blockers (to prevent future attacks, whereas octreotide is for acute bleeding)
- Liver transplant (last resort)

Bleeding varices require immediate intervention using vasoconstrictive drugs to reduce flow to the varices eg. **Vasopression or octreotide**, endoscopic **ligation** (putting rubber bands around the varices). If the patient survives, beta-blockers can be used to reduce portal pressure, reducing the risk of variceal rupture.
Another way to reduce pressure in varices is by **surgical shunting**. Shunts give the blood a path of lesser resistance so as to reduce pressure on varices, in an attempt to prevent their rupture. A shunt can be placed from the hepatic portal vein to the inferior vena cava, or from the splenic vein to the renal vein, or a transjugular intrahepatic portosystemic shunt (TIPS) can be placed linking the hepatic portal vein to the hepatic vein directly.(Jugular part of the name is because the shunt is placed through catheters inserted into the jugular vein) Although shunts reduce flow to varices, much of the blood that normally passes through the liver now gets back to the heart without undergoing the myriad of chemical reactions that normally occur in the liver (the major metabolic and detoxification center). If liver failure decreases detoxification reactions, or if a shunt allows circumvention of the liver, toxic chemicals can affect the brain, resulting in delirium (hepatic encephalopathy).

Ascites

- Accumulation of fluid in the peritoneal cavity
- PHT > Endothelial cells get irritated > Release of NO > VD > afferent arterioles have pressure receptors that sense a decrease in the renal perfusion (interprets it as hypovolemic state of the body) > Renal Na Retention
- (NO= nitric oxide, VD= vasodilatation)

Signs:

- Distended (bulging) flanks > sensitive but not specific
- Shifting Dullness
- Fluid wave

Dx:

- U/S > to confirm ascites
- SAAG (serum-ascites albumin gradient):
    - It can help determine if the etiology is portal hypertension (PTH) or not.
    - How to differentiate between liver-related ascites and that of other causes?
        - SAAG is measured by subtracting the albumin concentration in the ascites fluid from the albumin level in the serum.
        - Fluid in PTH tends to be relatively dilute, and thus has a lower albumin concentration than ascites fluid in infection or malignancy. Thus, the **SAAG tends to be higher in PHT than in other causes of ascites** (since a smaller number is being subtracted from the serum albumin concentration).
        - Generally, in PTH, SAAG > 1.1 g/dl (11 mg/dl), and in other causes of ascites, SAAG < 1.1 g/dl (11 mg/dl)

- Inflammation >> Exudate (pus ..)
- Liver >> Transudate (fluid) = high albumin gradient

In PHT & HF

- Serum albumin – ascetic albumin = >11 mg/dl
- (since it's a transudate ascetic fluid in this case, it won't contain albumin making the albumin in serum higher unlike the exudate in which the opposite occurs)

In kidney / peritoneal disease

- Serum albumin – ascetic albumin = <11 mg/dl

#In Pleural Effusion, the same concept is applied but we measure the ratio of protein not albumin instead

Rx:

- Treat underlying cause
- Salt restriction > to not further stimulate aldosterone
- Aldosterone inhibitor **(Spironolactone) – most important**
- **Lasix** is also important because it's faster. It also balances out the increasing Ca level that results from the use of Spironolactone
- Tapping
- Shunt
- TIPS > will prevent accumulation of ascites

Spontaneous Bacterial Peritonitis (SBP):

- Peritonitis usually occurs after surgery
- Bacteria comes from the bowel due to weakness of bowel peritoneal barrier which allows the bacteria to pass through it and enter the peritoneum where there is high sugar in ascetic fluid (convenient environment) >> abscess – peritoneum filled with pus
- We measure WBCs: If PMN (polymorphonuclear cells) > 250/ml >> then it's SBP
- We give empiric antibiotics (Cephalosporins)

Hepatic Encephalopathy

- The blood that goes to the varices does not pass through the liver so it does not get detoxified. So basically it still contains high nitrogen products (waste) that continues on to the brain > encephalopathy
- Differs from dementia in the fact that it fluctuates (intermittent dementia)
- Sign: Flapping tremor (asterixis)

Causes:

- Azotemia (renal impairment)
- GI bleeding (more ammonia is produced)
- Infection (oxidative stress, free radicals because the liver isn't detoxifying)
- Constipation (stool stays in the colon where bacteria works on it producing ammonia)

Rx:

- Identify cause and treat it
- Most important cause is constipation in which we give laxatives

Hepatocellular Carcinoma

- Very common (does not happen in non-cirrhotic)
- Any pt that has cirrhosis is in high risk of it
- Must routinely screen the pt by U/S

Added information about TIPS (shunting):
#One of the complications of TIPS > Hepatic Encephalopathy

- They measure the portal pressure, if it decreases by 20-30% and the pt wakes up then that's good = Successful TIPS
- But if it decreases by 10% only, that's not enough so we use a smaller stent. If it decreases for example by 5% only that's not enough so its considered = Failed TIPS
- Usually in that stage we go for liver transplant instead of TIPS