Portal hypertension & common surgical liver diseases

Objectives:

- Effects of portal hypertension
- Clinical features
- Acute variceal bleeding
- Ascites

PLEASE, focus on the doctor's notes!!

Resources:

- Davidson's.
- Slides
- Surgical recall.
- Raslan's notes.

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Once you stop learning you start dying.

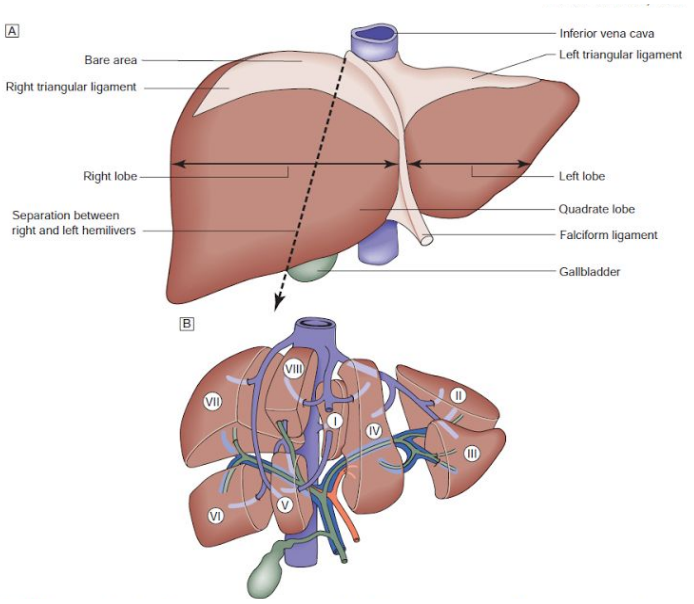
Basic review:

Anatomy of the liver:

-The liver is the largest abdominal organ .It extends from the fifth intercostal space to the right costal margin.
The liver is divided into a large right lobe and a small left lobe by the attachment of the falciform ligament fissures The right lobe is further divided into a quadrate lobe and a caudate lobe. However, the liver segmental anatomy, as defined by **the distribution of its blood supply,** is important to the surgeon.

Segmental anatomy:

The **portal vein and hepatic artery** divide into right and left branches in the porta hepatis. thus separating the two hemilivers.
Each hemiliver is further divided into **four segments** corresponding to the main branches of the hepatic artery and portal vein. In the left hemiliver, segment I corresponds to the caudate lobe, segments II and III to the left lobe (or left lateral section),
and segment IV to the quadrate lobe. The remaining segments (V–VIII) comprise the right hemiliver.

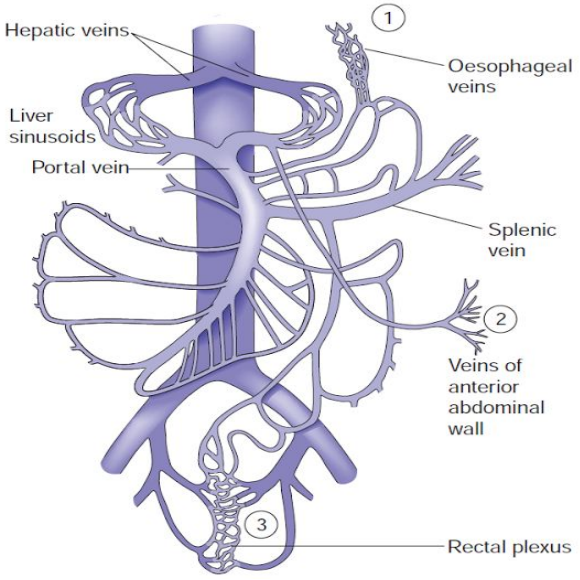


Blood supply:

The liver normally receives 1500 ml of blood per minute and has a **dual blood supply,** 75% coming from the **portal vein** and 25% from the **hepatic artery** ,which supplies 50% of the oxygen requirements.
The principal **venous drainage** of the liver is by the **right, middle** and **left hepatic veins,** which enter the vena cava

Portal venous system :

It is a specific type of anastomosis that occurs between the veins of portal circulation and those of systemic circulation
• In portal hypertension, these anastomoses open and form venous dilatation called varices.
• Sites: A. Esophagus (lower part). B. Upper Anal canal. C. Paraumbilical region. D. Retroperitoneal. E. Intrahepatic (Patent ductus venosus).
-collateral pathways develop between the portal and systemic venous circulations. Portosystemic shunting occurs at three principal sites (as in the picture).



Portal Hypertension

Causes : It's very important to know this because the management is different.

Cirrhotic	Non-Cirrhotic
• **Cirrhosis (>90% of cases):** The **most common cause** of portal hypertension is cirrhosis resulting from chronic liver disease and is characterized by **liver cell damage**, **fibrosis** and **nodular regeneration**.The fibrosis obstructs portal venous return and portal hypertension develops. Arteriovenous shunts within the liver also contribute to the hypertension. The chance of death is high because it means advance stage of cirrhosis and advance stage of PHTN.	• **Thrombosis of portal or splenic vein** Most important one especially in pt with hypercoagulable disease (protein C & S, usually in females)→ thrombus in their portal vein, note that their liver is normal. In case of hypercoagulable disease, patients may get clots in their legs and swellings. • Congenital atresia of the portal vein • Schistosomiasis[1] :most common cause of portal hypertension outside North America • Hepatitis. • Hepatocellular carcinoma • Metabolic : hemochromatosis[2], Wilson's disease[3] thrombosis • Budd-Chiari syndrome[4] The chance of death is lower because u only have one risk instead of tow. * PHTN can come even with normal liver pts.

Symptoms: The problem with PHTN is its asymptomatic .

Asymptomatic: symptoms are **usually absent until they develop complications.** (most of the time, they present with bleeding)

Complications:

1. **Ascites** (**most common**)
   - Due to increased formation of hepatic and splanchnic lymph*, **hypoalbuminemia, and retention of salt and water.**
   - the most common complication of cirrhosis
   - Ascites can be controlled by **bed rest, salt and water restriction**, and **spironolactone (aldosterone inhibitor)**. If refractory, ascites can be treated by **inserting a peritoneo-jugular (LeVeen) shunt**[5], which allows one-way flow between the peritoneum and the jugular vein
   - Patients should be instructed to **avoid NSAIDs,** which can cause sodium retention and affect renal function
   - Diuretic therapy, a single morning oral doses of spironolactone and furosemide, beginning with 100 mg and 40 mg
   - Serial therapeutic paracentesis and TIPS are usually reserved for patients with refractory ascites.
   - Complication of ascites: spontaneous bacterial peritonitis (SBP): an infection of preexisting ascitic fluid without evidence of for an intra abdominal secondary source.
     - The diagnosis is established by: **positive ascitic fluid bacterial culture** and elevated ascitic fluid absolute polymorphonuclear leukocyte count (≥250 cells/mm3)

[1] also known as snail fever and bilharzia is a disease caused by parasitic flatworms called schistosomes
[2] indicates accumulation of iron in the body from any cause (risk for cirrhosis)
[3] is a genetic disorder in which copper builds up in the body Symptoms are typically related to the brain and liver Liver related symptoms include vomiting, weakness, fluid build up in the abdomen, swelling of the legs, yellowish skin, and itchiness
[4] very rare condition, affecting 1 in a million adults. The condition is caused by occlusion of the hepatic veins that drain the liver. It presents with the classical triad of abdominal pain, ascites, and liver enlargement
[5] shunt which drains peritoneal fluid from the peritoneum into veins, usually the internal jugular vein or the superior vena cava.

2. **Splenomegaly**
    - Progressive enlargement of the spleen occurs as a result of vascular engorgement and associated hypertrophy resulting in Hypersplenism.
    - Haematological consequences are anemia, thrombocytopenia[6] and leukopenia[7] (with the resulting syndrome of hypersplenism).
3. **Esophageal varices**[8]
    - **Definition** : Engorgement of the esophageal venous plexuses **secondary to increased collateral blood flow from the portal system** as a result of portal hypertension.
    - Approximately one-third of all patients with varices will develop variceal hemorrhage.
    - The most important complication of portal hypertension is Bleeding from esophageal varices[9]
    - the bleeding presents as **Hematemesis, melena, and hematochezia**[10]
4. **Hepatic encephalopathy**:
    - Portosystemic encephalopathy is due to an increased level of toxins such as **ammonia** in the systemic circulation

Bleeding prevention:

- Approximately one-third of all patients with varices **will develop variceal hemorrhage**
- A major cause of morbidity and mortality in patients with cirrhosis
- **AASLD**[11] **Recommendations** for prevention of variceal bleeding have been issued by the (AASLD's details are not included in the exam)
    - In patients who have compensated cirrhosis and **small varices** that have **not bled** but have criteria for increased risk of hemorrhage, **nonselective beta blockers**
    - In patients with **medium/large** varices that have not bled, nonselective beta blockers (propranolol or nadolol) is recommended or undergo **EVL (Endoscopic variceal ligation)**
    - In patients who receive beta-blockers, a follow-up EGD is not Necessary.
    - If a patient is treated with EVL, it should be repeated until the varices are obliterated. EGD should performed one to three months after obliteration and then every 6 to 12 months to check for variceal recurrence

Most important thing to do is when u dx PHTN in cirrhotic or non-cirrhotic pt is **bleeding prevention**!! because once bleeding occurs, things go down.
In PHTN u will have dilated congested systemic veins due to dilated congested portal vein, the most we worry about are the ones around the **esophagus and upper stomach**. It could be also the lower rectum. the rest of veins are less likely to bleed by themselves.
The way u prevent is by **endoscopy** u go find which veins are bleeding and stop them, different ways to do this: put clips, put a band زي الربطه or sclerose. it differs according to the pt, site and how big the congested veins.
Medically, u can prevent it by prescribing B blockers (propranolol, atenolol) can also be used as it reduce the splanchnic vein pressure → reduce portal vein pressure → reduce the risk of bleeding. (they work very well!)
THE BEST TREATMENT FOR PHTN is to PREVENT BLEEDING .
In case of bleeding the first thing to do is RESUSCITATION احفظوها زي اسمكم !!!

[6] low blood platelet count
[7] decrease in the number of white blood cells
[8] Varices develop in order to decompress the hypertensive portal vein and return blood to the systemic circulation , they are seen when the pressure gradient between the portal and hepatic vein rises above 12 mmHg , patient with lower value do not form varices and do not bleed
[9] Veins don't have much smooth muscles and as a result do not go into spasm once they bleed. With this lack of smooth muscle and engorgement of the esophageal veins with, varecies tend to bleed profoundly, when they rupture.
[10] With rapid bleeding
[11] American Association for the Study of Liver Diseases

Treatment of bleeding

- **Initial therapy: hemodynamic resuscitation[12], prevention and treatment of complications.**

❖ First line of treatment :

1- General approach of any upper GI bleeding is ABCs!!

- **AIRWAYS** : **intubation** to protect from aspiration if needed. Common because there's too much bleeding they can't maintain their airway .
- **BREATHING** : check the ventilation and oxygenation.
- **CIRCULATION** :check BP and end organ perfusion.

- start resuscitation with 2 large peripheral IV and foley catheter and start 2 L normal saline or ringer lactate. In severe bleeding, blood is often given (give the patient O- until you get crossmatch blood). correct coagulopathy (vitamin K, fresh frozen plasma).

Then give prophylactic antibiotic, simply because venous bleeding is different than arterial bleeding. In veins, the pressure is low even in case of PHTN which allows bacteria to grow, so it's recommended to give 3rd generation cephalosporins new fluoroquinolones as part of ur resuscitation.

2- Upper GI endoscopy very imp for diagnosis, it might be peptic ulcer so u can't assume that it is PHTN without confirming it. Endoscopy is also imp in treatment u can clip, cauterise, band and sclerose it.
is the treatment of choice, where it can be diagnostic and also therapeutic. Endoscopic therapy can either be Endoscopic variceal ligation (EVL) or Endoscopic scleropathy.

- **Endoscopic sclerotherapy**[13][14]: a sclerosing substance is injected into the esophageal varices under direct endoscopic vision (repeated weekly until the variances are completely sclerosed but excessive intervention may cause ulceration and necrosis)
- **EVL**[15] :elastic band ligation of varices. **The method of choice for control of variceal haemorrhage.**

- If haemorrhage is torrential and prevents direct injection, balloon tamponade[16], TIPS or other surgical treatment may be used to stop the bleeding.
- Balloon tamponade arrests bleeding from varices in over 90% of patients, but the tube **is not left in place** for more than 24–36 hours for fear of causing **esophageal necrosis.**

3- Pharmacologic

- **Octreotide** (where terlipressin is unavailable) as bolus IV infusion **(**lowers portal venous pressure and arrests bleeding**)**: to achieve **vasoconstriction** of the splanchnic vessels. like beta blockers, but octreotide is used in **acute** settings while B blockers as prevention.
- **Nitroglycerin:** to avoid MI
- **Beta-blocker.**
- **PPI (omeprazole):** if you suspect peptic ulcer.
- **Prophylactic antibiotics** Suggest intravenous **ceftriaxone** (1 g IV) or **Cipro** (400 mg IV BID)
    - About 20% of patients with **variceal bleeding will have an infection**. Most commonly a UTI, but other more serious conditions like a respiratory infection or peritonitis may develop.

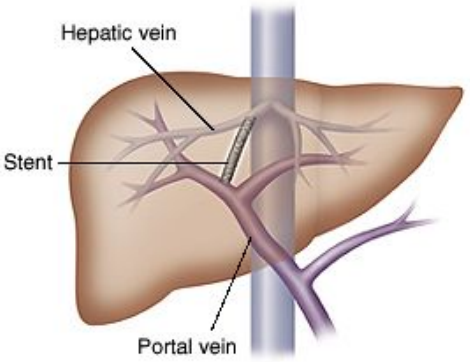
[12] By ABC an IV fluid
[13] Studies have shown that as many as 50%-80% of injected veins may be eliminated with each session of sclerotherapy
[14] Sclerotherapy is not recommended in pregnancy
[15] Endoscopic Variceal Ligation
[16] refers to the use of balloons inserted into the esophagus, stomach or uterus, and inflated to alleviate or stop refractory bleeding.

If sclerotherapy and conservative methods **fail to stop the variceal bleeding** or **bleeding recurs** then consider the following:

1- Repeat sclerotherapy/banding and treat conservatively

2- TIPS (Transjugular Intrahepatic Portosystemic Shunt)[17] It's radiological intervention not surgical

- Angiographic radiologist places a small tube stent intrahepatically between the hepatic vein and a branch of the portal vein via a percutaneous jugular vein route .
- Used occasionally to **decompress the portal system** and reduce the risk of further variceal haemorrhage in patients with well preserved liver function who fails emergent endoscopic treatment and has no complications from the bleeding or endoscopy.
- The choice of surgery usually depends upon the availability, training, and expertise of the surgeon. Although a selective shunt has some physiologic advantages, it may significantly exacerbate marked ascites.
- **portosystemic encephalopathy** is a major complication so in severe liver disease, **transplantation** is more likely to be considered if there is no contraindication



We know in PHTN the venous pressure is high but the pressure in the systemic circulation is usually low around 0-5 mmHg so we connect them by shunt (like a bridge) to vent out from portal system to systemic circulation.
It's done by interventional radiologist using guide wire and they go through the neck into the jugular vein → SVC → RA → IVC then he goes up → to hepatic veins (which is a systemic circulation its pressure is 0-5 mmhg) once they enter they puncture out the vein inside the liver (مايخاف من البليدنق لانه داخل الكبد) then enter to the portal system and puncture it (how will they know its portal system? using US it will appear enlarged and congested. Then they put bridge after that they put the shunt التوصيلة and inflate it and put stent inside the liver → pressure goes down → bleeding stops ..
What if u did TIPS and bleeding didn't stop? go back and inflate it more to decrease the pressure.
Complications of TIPS : liver detoxifies blood and you are bypassing hepatocyte → encephalopathy what do u do in this case? put another balloon inside it to make it smaller → more blood detoxification. Because of its complications it's not the 1st line tx ..
Transesophageal balloon is done temporarily in case of the health care centers that don't have all tools or expertise are not available so it helps transporting pt .its not therapeutic option .

3- Surgical shunt extremely rare that i don't want you to know it

- The choice of surgery usually depends upon the availability, training, and expertise of the surgeon. Although a selective shunt has some physiologic advantages, it may significantly exacerbate marked ascites.

4- Liver transplantation

So what I want you to know about PHTN: symptoms, the best way to treat it is to prevent it!! how to prevent it? → prevention guidelines, and what to do if the patient is bleeding.
In summary, doctor's notes:
During this time, after resuscitation, if they have no clue of what is happening, they start treating everything: e.i. PPI (for peptic ulcer) + start **octreotide acutely** to reduce splanchnic pressure (especially if there is suspicion of PHTN), and Endoscopy: locating the site + treatment at the same time. Rarely, patients need more than this!
If these work! Perfect! If partially worked → they break! → scope again & try to stop bleeding, if didn't stop! → next step! → TIPS! (by radiologist) What is the problem with it? Hepatic encephalopathy!!!!! U are connecting systemic with portal!! --> complication; if the bleeding doesn't stop, then what to do? Inflate it bigger! If u get encephalopathy as a result, what to do? (it means you are shunting too much!) → put one inside one. If the hospital doesn't have endoscopy or not ready, what to do? **Temporary measure** inflating a balloon (Balloon Tamponade) The problem that u can't keep it more than 24h → it will cause mucosal ischemia!!! After 24hs u need to deflate it! sometimes it works!! (so it might be the therapy for some patients!)
In summ: Resuscitation → endoscopy → Fail? → TIPS → fail? → shunt surgery (Rarely they do it).

[17] TIPS is more effective than endoscopic treatment in reducing variceal rebleeding but does not improve survival and is associated with more encephalopathy

Liver resection

Liver resection is the surgical removal of part of the liver. This operation is for some types of liver cancer and for certain cases of metastatic colorectal cancer. Up to half of your liver can be removed as long as the rest is healthy. Liver can grow easily liver is an intelligent organ it has the capacity to grow, if you cut a part of it, within 6 weeks it will grow. the liver feels your body surface area not your kiloes and accommodates ur body surface area, for example if you put a large liver into small person it will shrink and vice versa, and when it grows it doesn't grow to the same original size, it will grow to the limit that your body needs.

During a liver resection, the part of your liver that contains cancer is removed, along with some healthy liver tissue on either side.

- ## Liver Resection Indications:

We divide everything that needs resection into 2 parts: benign & malignant tumors:

Liver Resection Indications:	E.g :	Outcomes:
Benign tumors	**Adenoma** : the most common benign lesion in liver that can be resected. **It should be resected because of:** 1. It can turn to **HCC** (malignancy potential!!!) imp! 2. High risk of rupture and **bleeding** "liver is rich in blood supply". Other benign tumors like hemangioma FNH (hamartoma) we don't touch it, as it is a normal tissue. Adenoma is more common in females, it +ve for estrogen receptors, so when a female gets pregnant, it increases in size (but don't worry, it is rare :)) In the past, they used to resect hydatid cyst, but nowadays, it became rare.	Mortality rate <2% Complication rate 20-30% We can do the operation safely 80% won't have complications. The outcome depends on the indication.
Malignant tumors	**Primary:** Liver has 2 types of cells: hepatocyte (epithelium) + bile duct cells, cholangiocyte (also epithelium) → so, hepatocyte malignancy is: hepatocellular carcinoma, cholangiocyte malignancy is: cholangiocarcinoma. (carcinoma = arises from epithelium) **1-** HCC (hepatocellular carcinoma) specifically seen in pts with liver disease, most likely liver cirrhosis (alcohol, HCV, HBV, hemochromatosis). HBV (can cause HCC without cirrhosis). **2-** CC (cholangiocarcinoma) usually in normal liver!! Rarely seen in diseased liver (opposite to HCC) → if so, bile duct disease e.g. PBC, PSC **Secondary (Metastasis):** most common liver cancer and more commonly **colon**. One of the indication for surgery in liver malignancies is colorectal cancer metastasis (CRCLM) **common cause of death in cancer pts → liver failure.** **This surgery, will increase 5 years survival in 30-40% of patients.** **In metastasis we focus on colon cancer for 3 reasons:** **1-** Colon cancer is one of the **commonest cancers** especially in males (number 1 cancer) in female (number 2). **2-** Colon is **connected to the liver by portal system.** (special circulation connection) **3-** Today we have clear evidence that we can improve survival!! We actually can cure some patients!!! (usually they are stage 4 (bc it is met to the liver)) → **improve SURVIVAL IN COLON CANCER**!! What about other cancers? They are still studying it. **لا تقولون ما قلت لكم الـ ٣ أسباب!!**	The only potential for cure in case of malignant tumor even in colon metastases.

What's resectable?

How much can we resect from liver?	In **normal** healthy individual **we can resect up to 70%** at one time. (keeping 30% = 2 segment out of 8)[18] remember in a normal liver !! 70% this number is imp .
Can we do anything else?	Make the normal liver bigger and the tumor smaller (from 70% to 80%)

- If the liver is normal we can take up to 70% of the liver; we can add 10% by other techniques (by increasing the size of liver 10%) → by trigger development of liver even before operation (without surgery) → by this way, we can take up to 90%!!.
- The percentage goes down massively if the liver is abnormal like child score B or C.
- Liver regeneration will spend 6 weeks → we have to make sure that the pts will tolerate this duration & won't die :).

Table 14.2 Assessment of patients with portal hypertension using a modification of Child's grading system

Points scored			
Criterion	**1**	**2**	**3**
Encephalopathy	None	Minimal	Marked
Ascites	None	Slight	Moderate
Bilirubin (μmol/l)	< 35	35–50	> 50
Albumin (g/l)	> 35	28–35	< 28
Prothrombin ratio	< 1.4	1.4–2.0	> 2.0

Grade A = 5–6 points; grade B = 7–8 points; grade C = 10–15 points.

HCC vs. CRCLM the 2 most common indication for liver resection

Cancer :	HCC	CRCLM[19]
Background liver	Diseased liver; cirrhosis or fatty or inflamed So there the problem that generate the tumor Usually abnormal liver, Sick pt with comorbidities like cirrhosis ,PHTN,DM	Healthy and normal liver Young healthy person
How much resection can be tolerated	Limited only **30%** (bc it is not a normal healthy liver!)	**70%** as in normal liver
Transplant options	Only in patient with severe cirrhosis and very small primary adenocarcinoma **Transplant is only an option for HCC, not CRCLM!!!**	**Never** U dont have transplant as backup Because it's metastasis there's circulating cells outside the system.

U should know the risk factors of HCC.(see next page)

Liver Resection Complications: IMPORTANT!! the 4 most common complications:

- **Liver Failure: high chance to die** (not common, but it is risky). If you take more than 70% or if the background liver is unhealthy and u remove large portion. because CT scan only shows the size but does not show how bad the liver is. So we can't know the exact state of the liver until we do a surgery. Some patients have normal CT and normal LFTs, but it doesn't mean their liver is normal. for example in child score A you have normal CT normal LFT but they have cirrhosis!

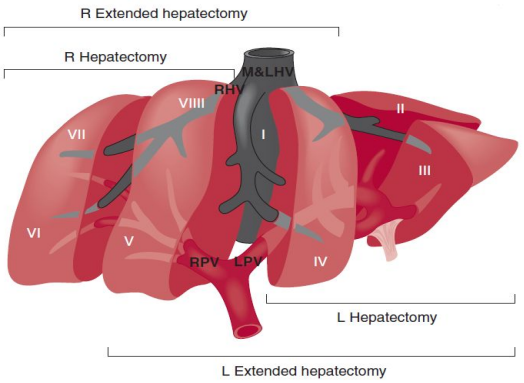
Sometimes in case of fatty liver we resect 50% liver and the patient develops liver failure due to the abnormal background liver.

Liver failure can be temporary until the liver grows back or permanent → patients usually die.

- **Bleeding number 1 complication!** It is **common and serious complication**. We can hear it as harsh sound!!! U can lose 5 L of blood in 2-3 min!!!!!!!!
- **Bile leaks** U have to clip the biliary ducts & prevent them to leak!
- **Infection (wound, deep abscess)** Common in most surgeries, but more common in liver, because the liver is a big immune organ! So when u affect the liver, susceptibility of infection increases.The truth about liver cirrhosis & liver dysfunction is that the usual cause of death is infections!! That's we they get Spontaneous bacterial peritonitis.
- **General complications**

[18] See the segments in the next page
[19] colorectal cancer liver metastases

Types of resections: you don't have to memorize the numbers



1. **Right side:** 4 segments (5,6,7,8) 50% of the liver

In the right we have 3 and one central which is at the back of the liver

2. **Left side:** 3 segments (2,3,4) 30% of liver
3. **Posterior:** segment 1

Liver tumors: the only one you should know

Hepatocellular carcinoma (hepatoma):

- Account for more than **80% of primary** liver cancer
- more common in **male**
- relatively uncommon in the developed world but is common in Africa
- In the West, about two-thirds of patients have pre-existing cirrhosis and many others have evidence of hepatitis B or C
- In non-cirrhotic patients, the tumour may have grown to a considerable size before giving rise to abdominal pain or swelling.
- In cirrhotic patients, hepatoma may become manifest as sudden deterioration in liver function, often associated with extension of the tumour into the portal venous system
- Common presenting features would involve progression of existing liver disease symptoms, and may include abdominal pain, weight loss, abdominal distension, fever and spontaneous intraperitoneal haemorrhage
- The lesion may be detected and characterized by abnormal ultrasound scanning. Percutaneous needle aspiration cytology and needle biopsy for histological confirmation
- Abdominal CT or MRI is valuable in planning resection and excluding nodal involvement

Recall:

What is the pathophysiology of portal hypertension?
Elevated portal pressure resulting from resistance to portal flow.
What is the etiology?
Cirrhosis (90%), schistosomiasis,hepatitis, Budd-Chiari syndrome,hemochromatosis,Wilson's disease,portal vein thrombosis, tumors, splenic vein thrombosis .
What are the associated CLINICAL findings in portal hypertension (4)?

1. Esophageal varices
2. Splenomegaly
3. Caput medusae (engorgement of paraumbilical veins)
4. Hemorrhoids.

What other physical findings are associated with cirrhosis and portal hypertension?
Spider angioma, palmar erythema, ascites, truncal obesity and peripheral wasting, encephalopathy, asterixis (liver ap), gynecomastia, jaundice
What are esophageal varices?
Engorgement of the esophageal venous plexuses secondary to increased collateral blood ow from the portal system as a result of portal hypertension
What are the signs/symptoms of esophageal varices?
Hematemesis, melena, hematochezia
What is the initial treatment of variceal bleeding?
As with all upper GI bleeding: large bore IVs 2, IV fluid, Foley catheter, type and cross blood, send labs, correct coagulopathy (vitamin K, fresh frozen plasma), intubation to protect from aspiration
What is the diagnostic test of choice?
EGD (upper GI endoscopy) Remember, bleeding is the result of varices only half the time; must rule out ulcers, gastritis, etc.
If esophageal varices cause bleeding, what are the EGD treatment options?

1. Emergent endoscopic sclerotherapy: a sclerosing substance is injected into the esophageal varices under direct endoscopic vision
2. Endoscopic band ligation: elastic band ligation of varices.

What are the pharmacologic options?
Somatostatin (Octreotide) or IV vasopressin (and nitroglycerin, to avoid MI) to achieve vasoconstriction of the mesenteric vessels; if bleeding continues, consider balloon (Sengstaken Blakemore tube) tamponade of the varices, beta-blocker
What are the options if sclerotherapy and conservative methods fail to stop the variceal bleeding or bleeding recurs?

- Repeat sclerotherapy/banding and treat conservatively
- TIPS
- Surgical shunt (selective or partial)
- Liver transplantation.

What does the acronym TIPS stand for?
Transjugular Intrahepatic Portosystemic Shunt
What is a TIPS procedure?
Angiographic radiologist places a small tube stent intrahepatically between the hepatic vein and a branch of the portal vein via a percutaneous jugular vein route
What is the most common perioperative cause of death following shunt procedure?
Hepatic failure, secondary to decreased blood ow (accounts for two thirds of deaths)
What is the major postoperative morbidity a er a shunt procedure?
Increased incidence of hepatic encephalopathy because of decreased portal blood ow to the liver and decreased clearance of toxins/metabolites from the blood
What medications are used to treat hepatic encephalopathy?
Lactulose PO, with or without neomycin PO
What is a right hepatic lobectomy?
Removal of the right lobe of the liver (i.e., all tissue to the right of Cantlie's line is removed)
What is left hepatic lobectomy?
Removal of the le lobe of the liver (i.e., removal of all the liver tissue to the le of Cantlie's line)
What is a right trisegmentectomy?
Removal of all the liver tissue to the right of the falciform ligament
What is the most common liver cancer?
Metastatic disease outnumbers primary tumors 20:1; primary site is usually the GI tract
What lab tests comprise the workup for liver metastasis?
LFTs (AST and alkaline phosphatase are most useful), CEA for suspected primary colon cancer
What are the associated imaging studies?
CT scan, ultrasound, A-gram
What are the three common types of primary benign liver tumors?
1. Hemangioma (**the most common**) 2. Hepatocellular adenoma 3. Focal nodular hyperplasia
What are the four common types of primary malignant liver tumors?

1. Hepatocellular carcinoma (hepatoma) **the most common**
2. Cholangiocarcinoma (when intrahepatic)
3. Angiosarcoma (associated with chemical exposure)
4. Hepatoblastoma (most common in infants and children

What are the other benign liver masses?
Benign liver cyst, bile duct hamartomas, bile duct adenoma
What is Hepatocellular Carcinoma?
Most common primary malignancy of the liver **also known as?** Hepatoma
What is its incidence?
Accounts for 80% of all primary malignant liver tumors
What are the signs/ symptoms?
Dull RUQ pain, hepatomegaly (classic presentation: painful hepatomegaly), abdominal mass, weight loss, paraneoplastic syndromes, signs of portal hypertension, ascites, jaundice, fever, anemia, splenomegaly
What tests should be ordered?
Ultrasound, CT scan, angiography, tumor marker elevation
What is the tumor marker?
Elevated -fetoprotein
What is the most common site of metastasis?
Lungs
What is the treatment of hepatocellular carcinoma?
Surgical resection, if possible (e.g., lobectomy); liver transplant
What are the indications for liver transplantation?
Cirrhosis and NO resection candidacy as well as no distant or lymph node metastases and no vascular invasion; the tumor must be single, 5-cm tumor or have three nodules, with none 3 cm