MED437
KING SAUD UNIVERSITY

SURGERY TEAM 437

Portal Hypertension and Common Surgical Liver Diseases

Objectives:

1. Effects of portal hypertension.
2. Clinical features.
3. Acute variceal bleeding.
4. Ascites.

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Color Index:

- Important
- Doctor's Notes
- Extra
- Davidson's

Editing File / Feedback

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. The middle hepatic vein divides the liver into Right and Left Lobes

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

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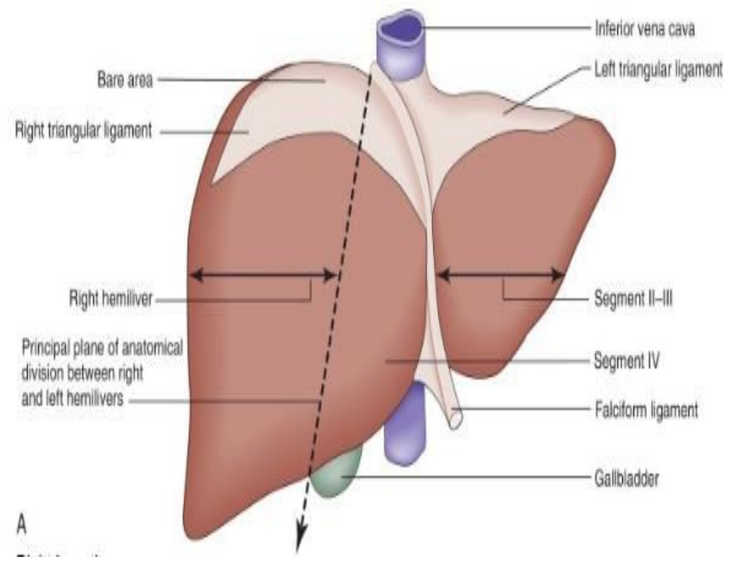
Blood supply:

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).

Liver function:

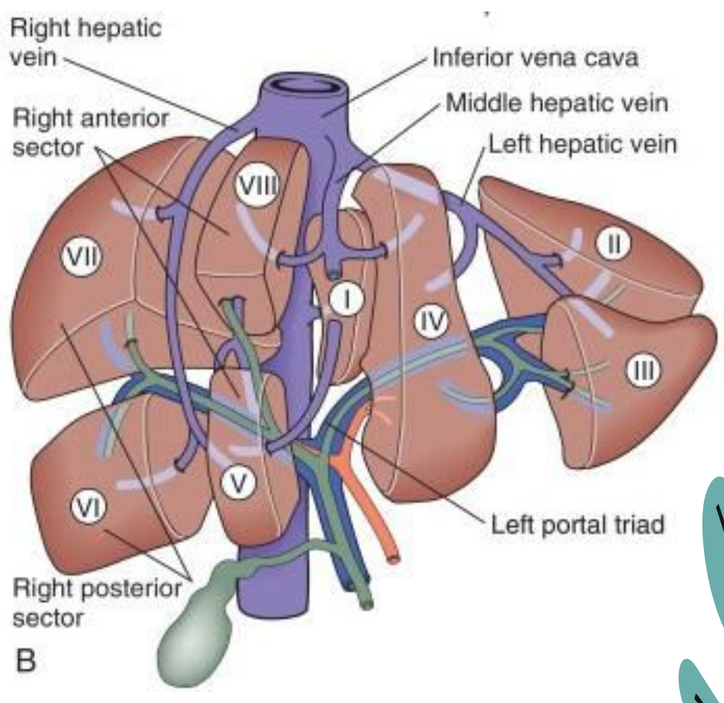
1. Responsible for storing glucose as glycogen, or converting it to lactate for release into the systemic circulation .
2. Amino acids are utilized for hepatic and plasma protein synthesis or catabolized to urea.
3. Metabolism of lipids, bilirubin and bile salts, drugs and alcohol.
4. Production of the coagulation factors, including:

a. Factor I, factor V, and factor XI.

b. Vitamin K dependant factors: factor II, factor VII, factor IX and fac

c. Protein C, protein S and antithrombin.

5. The liver is the largest reticuloendothelial organ, it has kupffer cells which remove damaged RBC , bacteria.

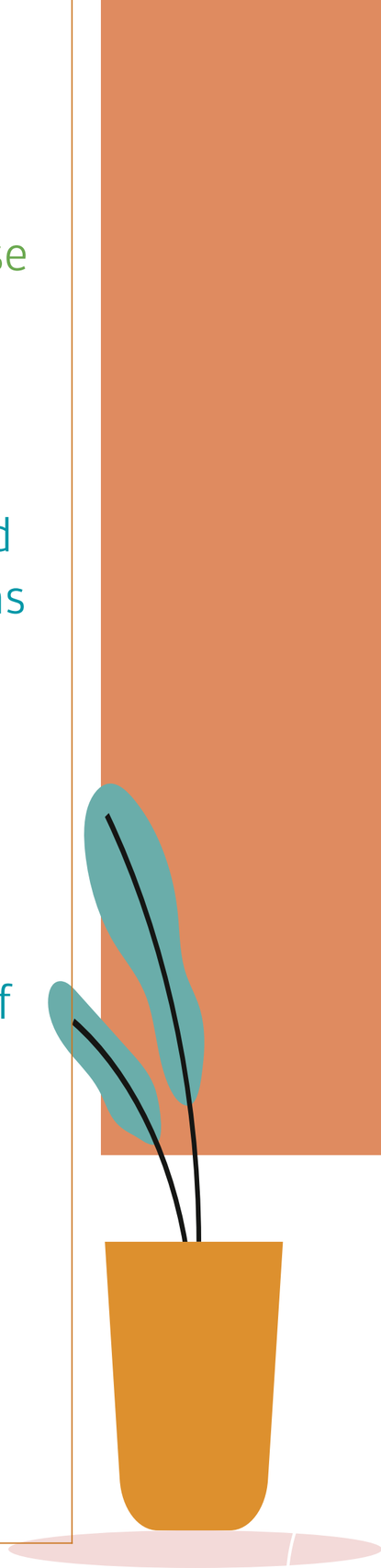


- Coagulation profile is important to assess liver function.

How can we differentiate if the elevated INR is related to vitamin K deficiency or to liver abnormality? Simply by giving them vitamin K supplements; if they showed improvement then this i related to vitamin K, otherwise is a liver problem.

Radiological investigations of the liver:

Ultrasound	It is the first and initial investigation in liver diseases. 1. Non-invasive. So we can use it in the ICU if we have a sick patient post liver resection to check if he has thrombosis or not. 2. Assesses intra and extrahepatic bile duct dilation or gallbladder distension due to obstruction, and confirm the need for more invasive investigations . 3. Detects space occupying lesions in the liver and pancreas (although overlying bowel gas may prevent a clear view of the pancreas). 4. Evaluates vascular system of the liver. 5. Detects gallstones
Computed Tomography (CT)	we usually use it with IV contrast, or we can order the triphasic CT scan,which means the CT will be taken with contrast and in hepatic arterial phase will show us only the arterial system in the body. Then the portal venous phase will show us only the portal system. why? bc some tumors only show up in the arterial phase like HCC. 1. Contrast enhanced CT identifies and stages hepatic, bile duct and pancreatic tumors. 2. Demonstrates **three abnormalities:** a. Dilated biliary tree to the level of the obstruction. b. Vascular abnormality or invasion. c. Lymphadenopathy or distant metastasis. *Positron emission tomography (PET-CT) is used for tumor staging and distant metastasis. It is an advanced technique of CT scan in which we use a nuclear material, we don't use it as a fist step except if the CT was insensitive or did not show us the tumor clearly.
Magnetic resonance imaging (MRI) **MRI had the same features of CT**	Magnetic resonance cholangiopancreatography (MRCP) has largely replaced other forms of invasive radiological imaging of the bile duct and pancreas More sensitive for liver parenchyma. CT scan is better for metastasis than MRI 1. It has the advantage of not introducing an infection to the biliary system. 2. Enables the assessment of the vascular anatomy and the parenchyma of the liver and pancreas. Only indicated in selected cases either to discover the reason of the disease or when US and CT are not clear and that particularly is when the mass is less than 2 cm" Liver biopsy can be done with the help of CT guidance for liver diseases and masses .identifies the cause of liver disease.



Congenital abnormalities

❖ **Liver cyst:** is a benign disease

- Polycystic disease is a rare cause of liver enlargement and may be associated with polycystic kidneys as an autosomal dominant trait.
- How is biliary cystadenoma different from cyst?
  - Biliary cystadenoma is a premalignant disease
  - Communicates with the bile system
- **Histology**:
  - The cyst is lined by biliary epithelium and contain serous fluid.
  - Does not communicate with the biliary tree. To look for communication? **MRCP**
- **Incidence**: Sporadic or polycystic disease.
- **Symptoms**:
  - patients rarely develop symptoms, they only have them due to the mass effect on surrounding structures.
- **Diagnosis**:
  - US, CT, MRI.
- **Treatment**:
  - Only for symptomatic patients. So observation first, if symptoms occur we go for surgery
  - **Surgical**: deroofing or resection. Vs biliary cystadenoma is resection
  - Aspiration of serous fluid, recurrence with aspiration is high.
  - It may be necessary to combine a deroofing procedure with hepatic resection or to consider liver transplantation.

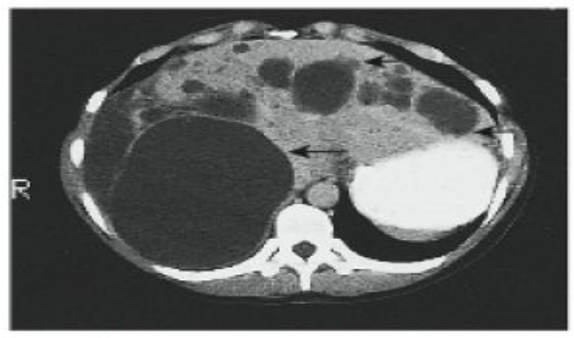


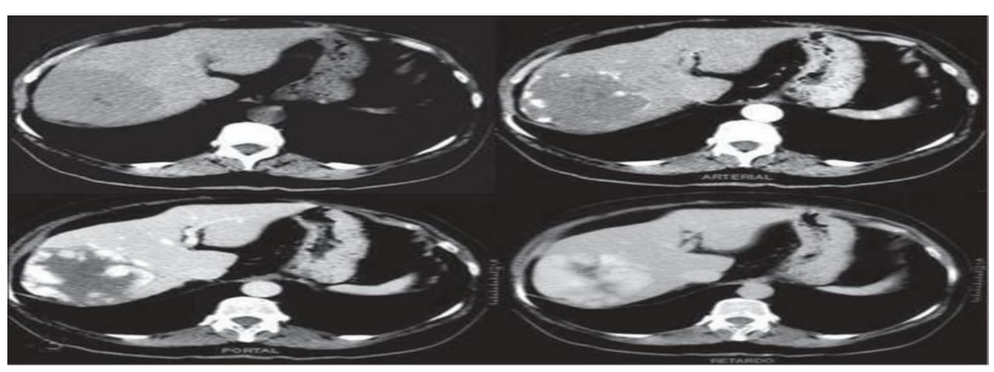
Fig. 14.6 CT demonstrating multiple biliary cysts appearing as hypodense areas within both lobes of the liver (arrowed).

❖ **Cavernous hemangioma:** video

- One of the most common benign tumors of the liver (affects up to 5% of the population) and may be congenital, most hemangiomas are small solitary subscapular growths found incidentally at laparotomy or autopsy.
- Womens are affected six times more frequently than men. (W:M,6:1)
- **Histology:**
  - The nodule is made of cavernous vascular spaces lined by flattened endothelium.
- **Symptoms**:
  - patients rarely develop symptoms, they only have it due to the mass effect on surrounding structures, it may cause angioembolization or bleeding if it ruptures. Trauma-> rupture -> hypotension->shock
- Diagnosis:
  - US: sometimes detected as densely hyperechoic lesions.
  - CT and MRI: centripetal filling in of contrast during dynamic
  - imaging. Very characteristic (starts at periphery then goes into the center)
- Treatment:
  - Observation for asymptomatic patients. Until symptoms appear
  - Rupture: Angioembolization.
  - Symptomatic: surgical resection

It could be hypodense or isodense compared to liver parenchyma in plain CT

We start with plain CT 1st: isointense/ hypointense (Rule out calcification)



2- Arterial phase CT: peripheral enhancement

3- Portovenous phase: Starts to go into the center

4- Delayed phase

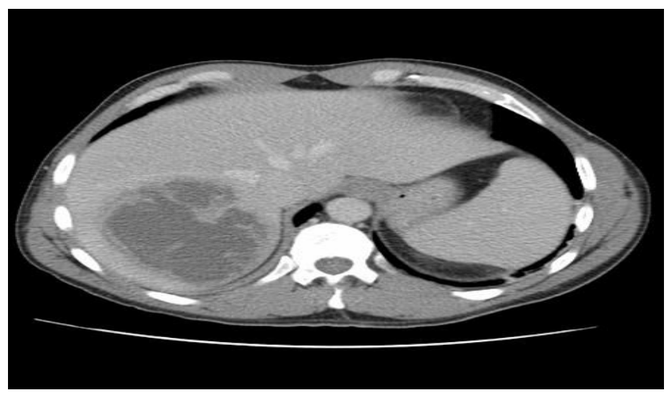
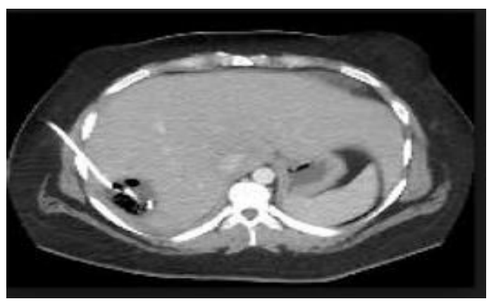
Hepatic infections

liver abscesses can be classified as bacterial, parasitic or fungal. Bacterial abscess is the most common type in western medicine, but parasitic infestation is an important cause world-wide. Fungal abscesses are found in patients receiving long-term broad-spectrum antibiotic treatment or immunosuppressive therapy, and may complicate actinomycosis..

A- Pyogenic liver abscess most common Infection from the biliary system is now more common due to the increasing use of radiological and endoscopic intervention.	
Sources of infection	• Biliary system: cholelithiasis, benign strictures, acute cholangitis, and periampullary tumors (obstruction of the biliary system; bacteria ascends) • Portal vein: abdominal sepsis: -Anorectal abscess, pelvic abscess, postoperative sepsis, intestinal perforation, pancreatic abscess,**appendicitis** or diverticulitis (most common) . GI infection. patient had appendicitis ,treated after 5 days presented with severe RUQ pain ,fever ? **portal empyema** • Hepatic artery: septic focus anywhere in the body: Intravascular infection ○ Endocarditis, vascular sepsis, ENT[1] or dental infection. Bypass , synthetic material • Direct spread from a contiguous organ: ○ Cholecystitis or empyema of the gallbladder. ○ Gastroduodenal perforation. Duodenal ulcer/abscess ○ Colonic perforation. • Following a blunt or penetrating injury. Trauma ->infection • In one-third of the patients, the source of infection is indeterminate (cryptogenic). Root unknown
Organisms	• Gram positive aerobes (hepatic artery spread) : **streptococcus milleri , staphylococcus aureus , and enterococcus species.** From infective endocarditis example • Gram negative aerobes (portal vein spread) bacteria of the GI : Escherichia Coli , klebsiella pneumonia, pseudomonas aeruginosa, proteus species and enterobacter cloacae. • Gram positive anaerobes (portal vein spread) bacteria of the GI : bacteroides and fusobacterium species. • Biliary system; E.coli , klebsiella pneumonia
Symptoms	• Fever. • Right upper abdominal pain (hypochondrium). • Swinging pyrexia of Chills and rigors. • Marked toxicity.

1:Ear, nose, throat.

Hepatic infections

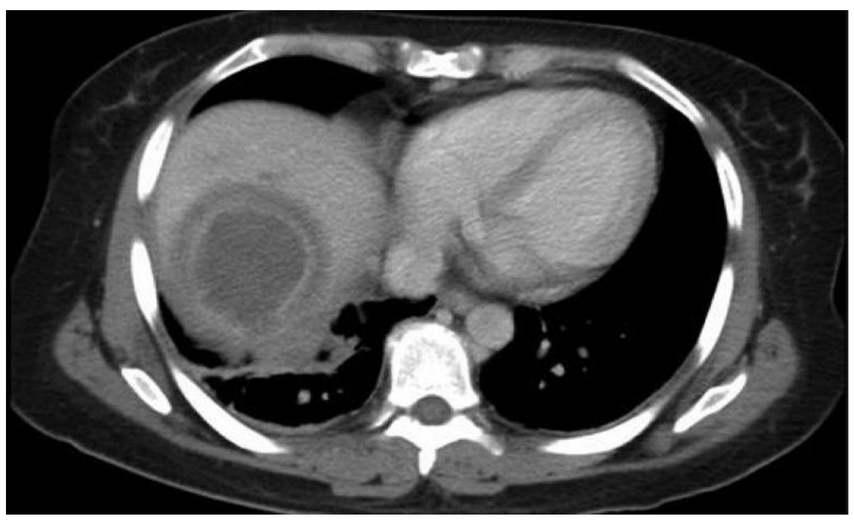
Signs	• Patient looks ill. • General malaise and anorexia. • Jaundice. Could be due to obstruction of the biliary system or due to sepsis. • Vital signs: tachycardia, high temperature, +/- hypotension. • Abdominal exam: enlarged and tender liver. An ICU patient due to pneumonia developed jaundice Cause? SEPSIS Does Not have to mean obstruction of the biliary system could be due to sepsis
Investigation	1. Labs: a. CBC: elevated WBCs b. LFTs: elevated enzymes. Stage of the abscess c. Coagulation profile: normal or elevated. d. Blood and pus culture. 2. Radiology: a. AXR : air in the liver (gas forming infection). b. CXR: right sided pleural effusion. c. US : hypoechoic lesion with thick wall along with biliary dilation. d. CT : central hypodense region and peripheral contrast enhancement during the portal phase. Hypodense + rim of enhancement (edema ; blood supply more than normal) 
Treatment	• Percutaneous drainage of abscesses under ultrasound or CT guidance.(see picture) • Antibiotic therapy. 

B- Amoebic liver abscess:

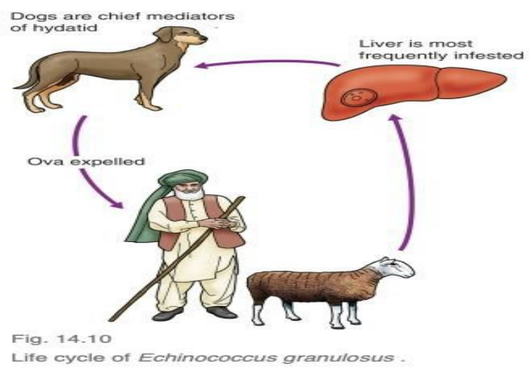
it's common in the endemic area

Pathogenesis	• Protozoal parasite infects the large intestines. • Ingested cyst in the large intestines > releases trophozoites > penetrates the mucosa >portal venous system > liver. • The abscess is large and thin-walled, usually solitary and in the right lobe, and contains brown sterile pus resembling anchovy sauce.
Organism	**Entamoeba histolytica.**
Clinical features	• Symptoms: right upper quadrant pain, anorexia, nausea, weight loss, night sweats and diarrhea. Present with 2 months pain (chronic) -> not pyogenic. They appear less sick than pyogenic liver disease patients (Inf. In GI + abscess) • Physical examination: tender enlargement of the liver with or without jaundice. • Other signs include basal pulmonary collapse, pleural effusion and leukocytosis.

Hepatic infections

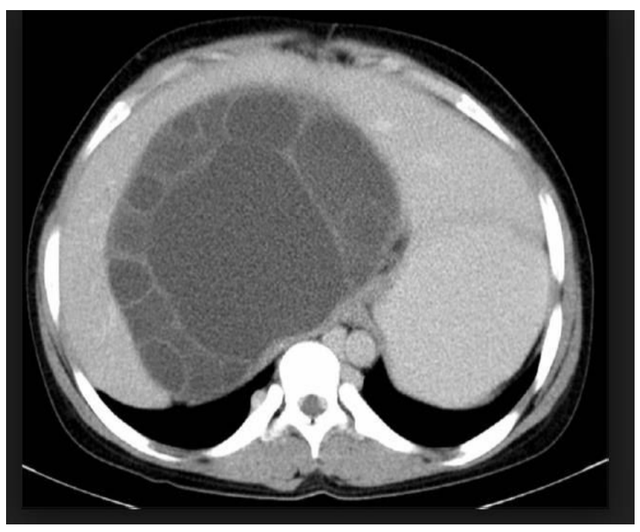
Investigations	● Labs: ○ CBC: elevated WBCs. ○ Direct and indirect serological tests: (amoebic protein) -Indirect haemagglutination (IHA). -Enzyme linked immunosorbent assay (ELISA). ○ Stool analysis: amoebae or cysts. ● **Radiology**: ○ US: hypoechoic lesions with well-defined margins. ○ CT: well-defined lesions with complex fluid, enhancing wall with a peripheral zone of edema around the abscess. Hypodense + rim+ hypodense around it
Treatment	● **Antibiotics**: ○ Metronidazole. More than enough vs the pyogenic; abx and drainage ○ Diloxanide furoate (for carriers). ● **Percutaneous aspiration:** ○ If no improvement after 3 days of antibiotics. ○ Pyogenic abscess severe symptoms due to supra infections When aspiration: white puss 

C- Hydatid disease

Pathogenesis	● The adult tapeworm lives in the intestine of the dog, from which ova are passed in the stool; sheep or goats serve as intermediate host by ingesting the ova whereas humans are accidental hosts. ● Infestation by one of the two forms of tapeworms in the gastrointestinal system. ● Ingested ova hatch in the duodenum > portal system > liver. ● **The layers of a hydatid cyst:** ○ Pericyst: host tissue formed by the body as a reaction to the parasite. ○ Ectocyst: external layer of the cyst. ○ Endocyst: germinative layer.  Fig. 14.10 Life cycle of *Echinococcus granulosus* .
Organism	echinococcus granulosus and E.multilocularis.

Hepatic infections

Clinical features	• Could be asymptomatic. Incidental findings • Chronic right upper quadrant abdominal pain (the most common presentation) . • If it ruptures: anaphylactic shock due to absorption of foreign hydatid protein . • Communication with biliary system: obstructive jaundice. Very thick material goes into the biliary system and obstructs it

Investigations	• Labs: after imaging
	  ○ CBC: eosinophilia. Parasite
	  ○ Serology tests:
	  ○ Immunoelectrophoresis (IEP): not for follow-up.
	  ○ Enzyme linked immunosorbent assay (ELISA): IgE or IgG4 (4 years), IgM (6 months).
	  ○ Immunoblotting: first-line test for diagnosis and follow-up.
	• **Radiology**:
	  ○ AXR: calcification.
	  ○ US, CT and MRI: well-defined, circumscribed cystic lesions with a clear membrane (thick wall)
	  and multiple daughter cysts.
	  V. Characteristic
	 

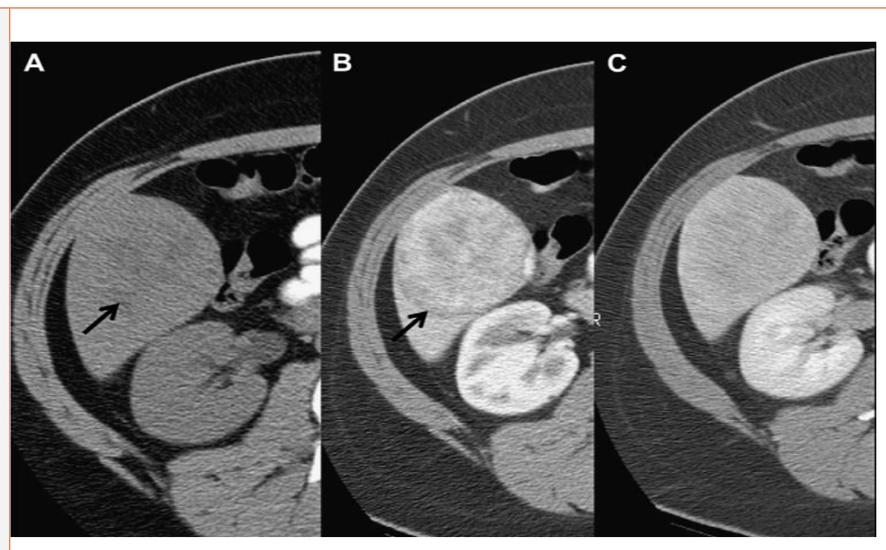
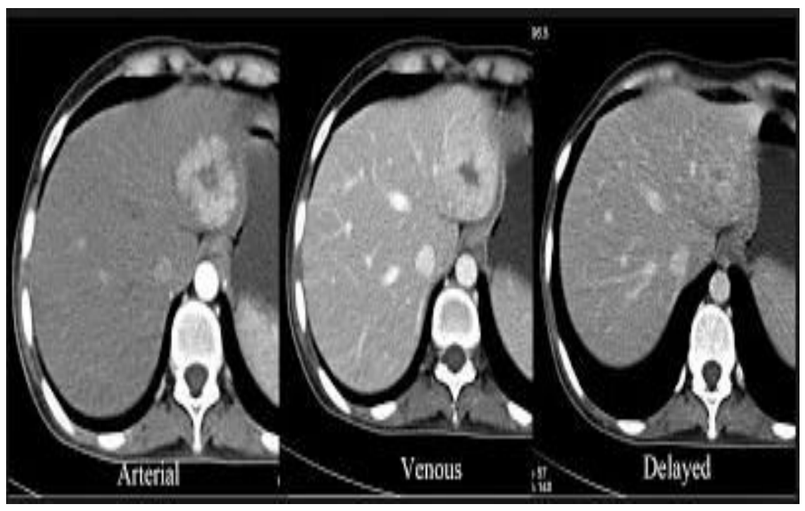
Treatment	• In asymptomatic patients, small calcified cysts may require no treatment.
	• **Medical**: albendazole or mebendazole for a month before surgery.
	• **Surgical**: • Deroofing: resection of the upper part of liver (the best)
	  ○ Pericystectomy (large symptomatic cysts are best managed by complete excision , together with the parasites contained within) .
	  ○ Liver resection: Resection of normal tissue with the cyst
	• Puncture-aspiration-injection-re-aspiration (PAIR) : If the patient can't go for surgery we do PAIR although **it's too risky** could rupture and get into an anaphylactic shock.

Tumors of the liver

A- Benign hepatic tumors :

	Liver cell adenoma	Focal nodular hyperplasia (FNH)
Prevalence	Women to men ratio is 9:1. This tumor is relatively uncommon and is found almost exclusively in women.	More common in females.
Causese	Estrogen and anabolic steroid play a causative role.	Not related to estrogen Incidental
Clinical features	A. Right Upper Quadrant pain as a result of hemorrhage within the Tumor. B. Superficial tumors may bleed spontaneously and present with symptoms of hemoperitoneum	○ The lesion may be asymptomatic and may regress with time or on withdrawal of oral contraceptives. ○ Right Upper Quadrant pain
Complications	That's why we do surgery ○Rupture> angioembolization. ○ Malignant transformation **Pregnant lady** with adenoma developed RUQ pain -> shock (Ruptured cyst)	-
Investigations	US , CT : solitary, well-encapsulated lesions. ● MRI. MRI and CT are the diagnostic tests.	○ US , CT: it can be differentiated from adenoma by the central fibrous scar. ○ MRI
Treatment	○ Female: < 5 cm : stop oral contraceptives > 5 cm : surgery (resection) ○ Male: surgery directly because the risk of malignant transformation is higher ○ Rupture: Angioembolization	Observation, such lesions do not undergo malignant transformation and do not require excision unless symptomatic.

Tumors of the liver

Radiological images	 A- Plain CT: Isodense B- arterial phase: hyperintense (brightness but not that much comparing to liver) Disappears in the venous phase	 Arterial: hyper intense with hypointense in the middle Central sky shines up

B- Malignant hepatic tumors:

- **Primary**:
  - Hepatocellular carcinoma (hepatoma)
  - Cholangiocarcinoma
  - Angiosarcoma
  - Hepatic mucinous cystic neoplasm

Hepatocellular carcinoma (hepatoma)	
Prevalence	More common in males than females Malignancy of the hepatocytes
Risk factors	• In the west, about two-thirds of patients have pre-existing liver cirrhosis (alcoholic or non-alcoholic), and many others have evidence of hepatitis B or C infection. Cirrhosis is a premalignant condition of HCC. Anything that causes cirrhosis caused HCC. • In africa, Aflatoxin (derived from the fungus, aspergillus flavus , which contaminates maize and nuts) is an important hepatocarcinogen.
Clinical features	• **In liver cirrhotic patients:** Picture (see it!!!!) ○ Sudden deterioration in liver disease symptoms ○ Abdominal pain, weight loss, abdominal distension, fever and spontaneous intraperitoneal Haemorrhage. • **Non cirrhosis:** ○ Abdominal pain or swelling.

Tumors of the liver

Investigation

- LFT may be normal or elevated(acute diseases) depending on the stage of the disease , CBC, coagulation profile
- **Screening** : every cirrhotic pts. Every 6 months
  - US abdomen (shows splenomegaly) .
  - Alpha-fetoprotein (AFP).
- CT , MRI the best:liver lesion with arterial enhancement hyperintense and early washout on porto-venous phase
- Vs. Adenoma; HCC is brighter (more hyperintense) than adenoma in arterial phase
- HCC is the only one that can be diagnosed on Imaging

Diagnosis:

- > 1 cm: one image with characteristic feature diagnostic
- Cytology: if the nodule is > 1 cm and features are not typical
- < 1 cm: 3-6 month follow-up cause slow growing

Treatment

The disease is usually advanced at presentation and the 5-year survival rate is less than 10%

1. **Transplantation:** the best. Gets rid of cirrhosis and the disease
   a. Milan criteria: single tumour of 5 cm or less in diameter, or with no more than three tumour nodules each one 3 cm or less in size (for cirrhotic patients with no contraindication to transplantation). Don't expose patient to immunosuppression for the transplant when he has a poor prognosis

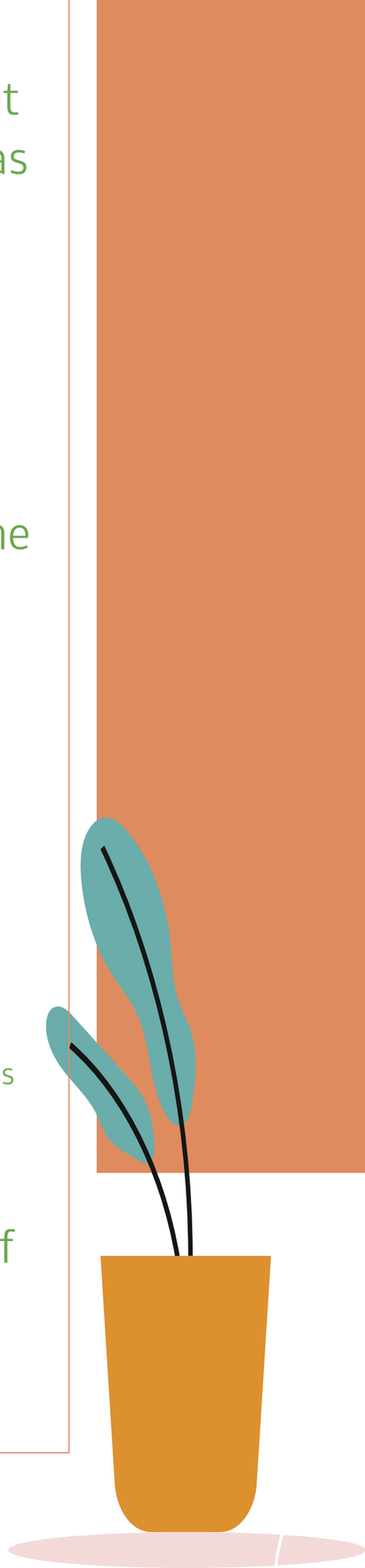
2. **Liver resection:**

***They do resection when transplant is not indicated or available.**
***Those with Child C criteria, they won't tolerate transplant and may develop post-hepatic resection liver failure.**

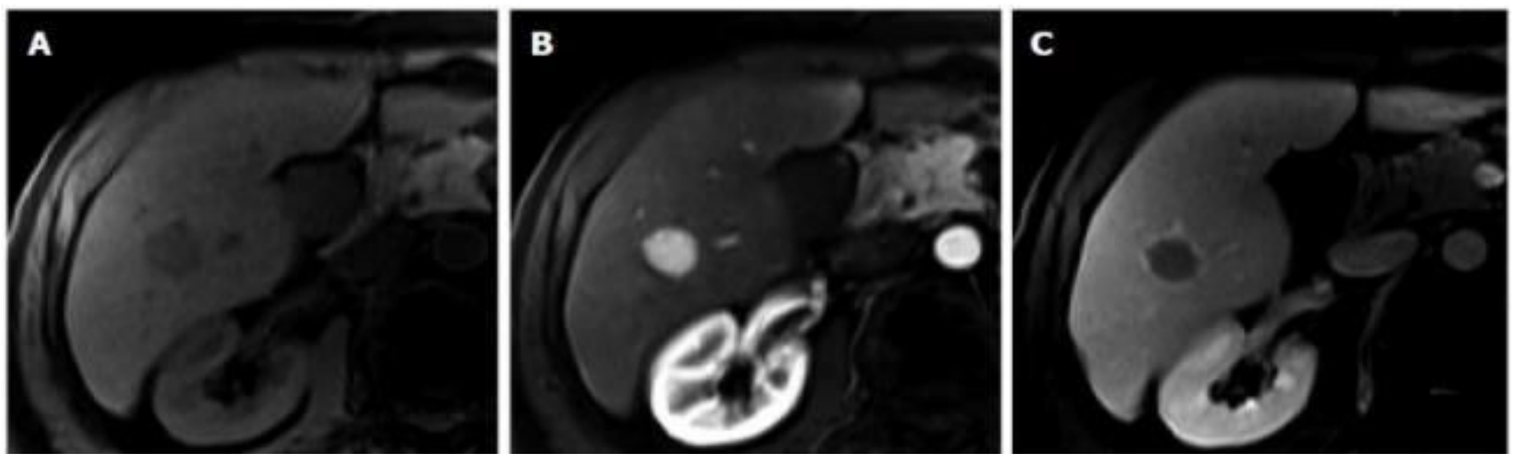
   a. Non cirrhotic patient because cirrhotic patients will not tolerate the procedure.
   b. Child A liver cirrhosis (explained later). They may tolerate the procedure but they shouldn't have portal hypertension.

3. **Locoregional therapy:** its an alternative if the patient can't do transplantation or resection, the aim is to kill the tumor inside the liver without resection or surgery.
   a. TRACE
   b. Local ablation : RFA (Radio frequency Ablation is equal to resection when tumor size is less than 2 cm) , microwave energy burn the tumor

4. **Chemotherapy**: if the patient is not fit to go for surgery or any kind of treatment.
   a. Sorafenib (multitargeted oral kinase inhibitor).



Tumors of the liver

Radiological images	 A. plain CT: Isointense / hypointense B. arterial phase: very bright (early enhancement) hyper intense C. venous phase: early washout

Cholangiocarcinoma	
Prevalence	Adenocarcinoma may arise anywhere in the biliary tree, Could be intrahepatic or extrahepatic including intra-hepatic radicles. It accounts for less than 10% of malignant primary neoplasms of the liver in Western medicine, although its incidence is rising
Risk factors	1) chronic parasitic infestation of the biliary tree 2) Choledochal cysts we resect it when we see it to kill the chances of development to cholangiocarcinoma
Clinical features	● Jaundice ● Abdominal pain, weight loss , anorexia ● Enlarged liver. ● There may be co-existing biliary infection causing the tumor to falsely present as a hepatic abscess.
Investigation	● Labs: ○ LFT : obstructive Jaundice (increase total bilirubin) ○ CBC ,Coagulation Factor CA 19-9 ● Radiology: ○ CT , MRI , MRCP, ERCP, PTC: usually we start with CT and MRI if we reach the diagnosis no further investigations are needed if not the we will proceed to ERCP or PTC. CA 19-9: tumor marker. Diff between: ○ Pancreatic cancer ○ Hepatobilliary ○ Cholangiocarcinoma ○ Ampullary cancer

Tumors of the liver

Treatment	• **Curative**:
	  ○ Resection
	• **Metastatic**:
	  ○ Palliative chemotherapy
	
	Vs HCC;
	HCC 4 treatment
	Here; resection or chemotherapy
	HCC: enhancement and early washout
	Here: NO washout only enhancement

B- Malignant hepatic tumors:

- **Secondary (metastatic) tumors.** These tumors are more common than primary tumors
  - **Sites**
    - Gastrointestinal tract: the most common sites are: colorectal then stomach, small intestine, pancreas.
    - Breast
    - Ovaries
    - Bronchus
    - Kidney

    Patient had colorectal cancer treated with surgery 8 months later presented with liver mass? Colorectal liver Metastasis

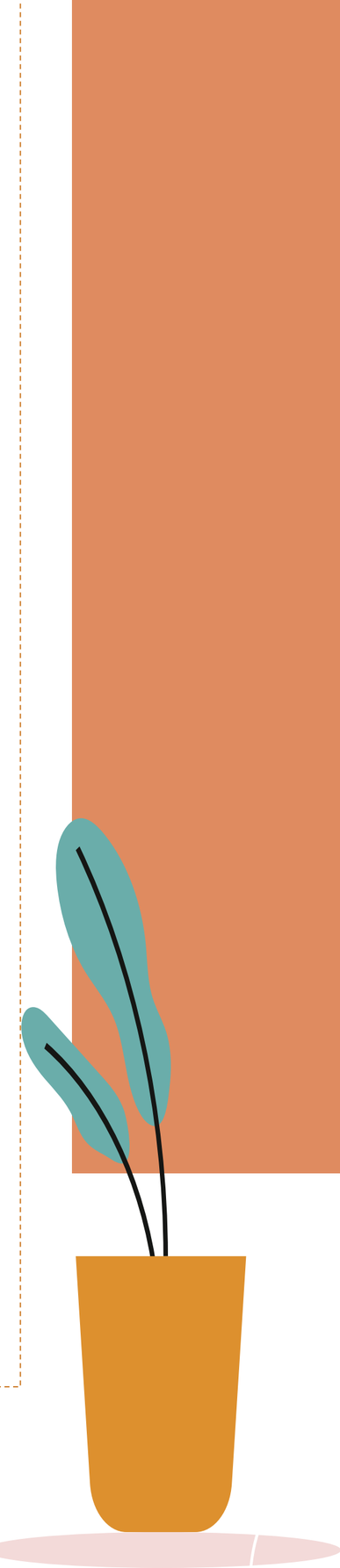
  - **Diagnosis** the diagnosis depends on the history of previous surgery to any kind of cancer. If there is no history then order the tumor markers.
  - **Tumor marker**
    - CEA for colorectal carcinoma.
    - CA 19-9: for pancreatic cancer.
    - CA 125: for ovarian cancer.
  - **Radiology:** CT , MRI, PET CT.

We usually start with CT scan, if the CT scan didn't show the lesion, then we do MRI bc its more sensitive and Specific. We use PET CT to check if there is other Metastasis other than the abdomen from head to toe.

  - **Treatment**
    - Resection very well response in colon,neuroendocrine,and sarcoma
    - Palliative chemotherapy

If its colorectal liver Metastasis then the Resection is the cure. The other Metastasis if its more than 6 months we may consider resection, but if its less than 6 months we treat them with Palliative chemotherapy.

- Encouraging results have been reported following local embolization with chemotherapy by selective arteriography (transarterial chemoembolization - TACE) and percutaneous ablation using radiofrequency and microwave energy have been used to useful effect for small lesions not amenable to surgery.



Portal Hypertension (vedio!!)

❖ Definition:

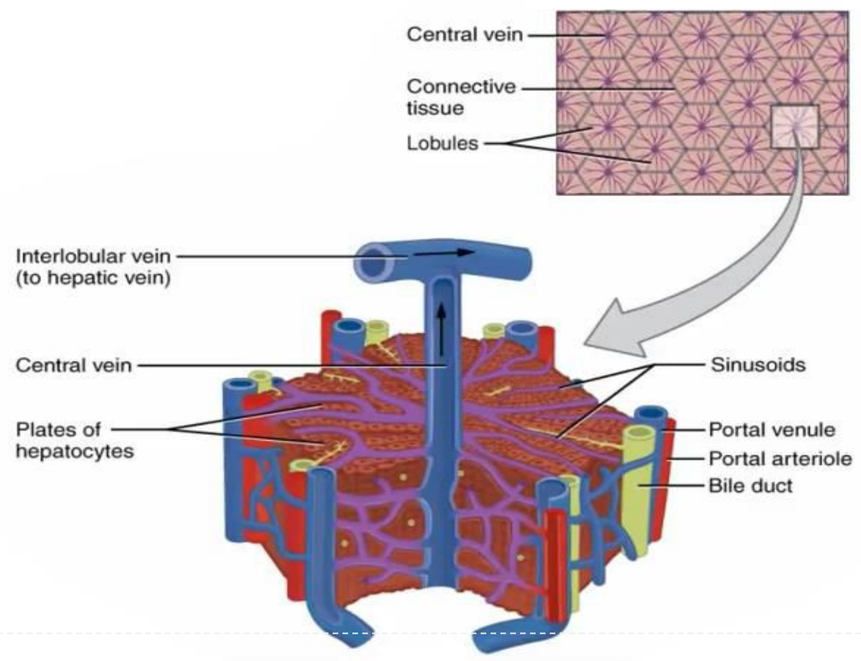
- Increased resistance to portal venous blood flow, the obstruction being pre-hepatic, hepatic or post-hepatic.
- Pressure (P) = Flow (F) X Resistance (R) , changes in either F or R affects the pressure. Problem in resistance or increased flow
- Portal pressure : 3 – 6 mm Hg portal hypertension: >8
  - If PP > 10 : shunting.
  - If PP >12: bleeding.
- Increased flow:
  - 1- splenomegaly: pushes a lot of blood into the portal vein
  - 2-AV fistula: pressure of the arterial systems transmits into the Venous system -> hypertension
- Left side portal HTN is due to the splenic vein (splenomegaly) thus portal HTN and varices -> **splenectomy**. Cant? Cut the artery
- Normal elevation:
  - Eating.
  - Exercise.
  - Valsalva.

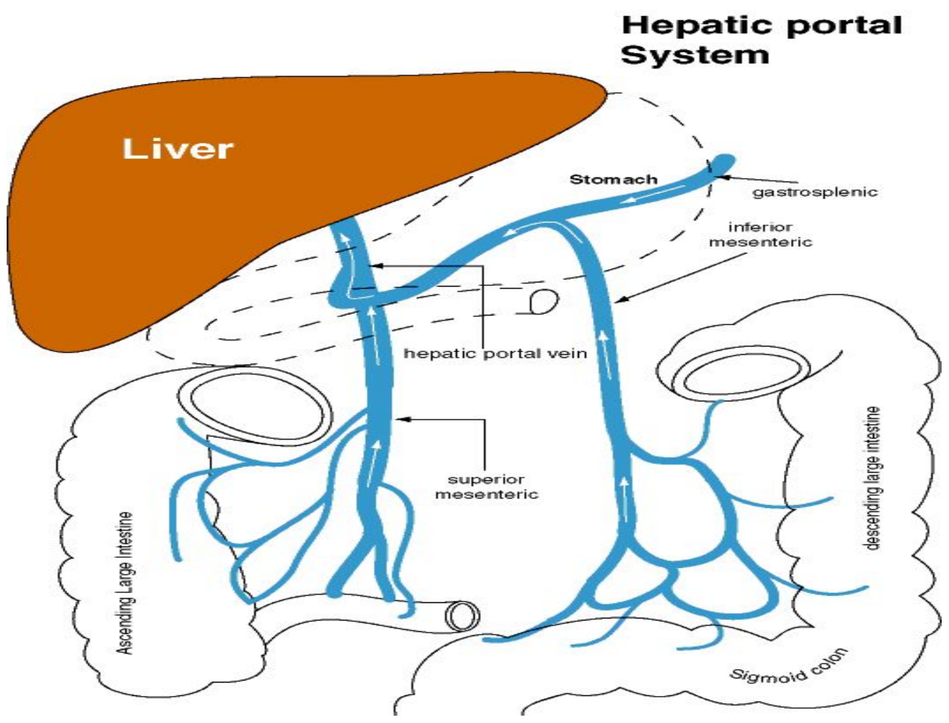
The most common cause of portal hypertension is liver cirrhosis so portal hypertension has the same causes of liver cirrhosis

❖ Pathophysiology:

- **Liver disease :**
  - ↓ portal vascular radius.
    - Recurrent inflammation -> fibrosis and regeneration causes stiffness and compresses the low pressure hepatic vein -> hypertension.
    - Hepatic artery pressure is high therefore it does not occur.

  - Splanchnic arteriolar vasodilation:
    - Decreased sensitivity to catecholamines.
    - Increased endogenous vasodilator (NO, prostacyclin).





Why do we have portal HTN?

- Lumed radius decreases
- Thrombus outside liver (PV)
- Pancreatic cancer compresses the PV

Patient presents with melena and hematemesis
How to know if intrahepatic or extrahepatic? **US**
US normal liver -> extrahepatic

Portal Hypertension

❖ Causes of portal hypertension:

Classification	Cause
Prehepatic	Portal or splenic vein thrombosis
	Increased portal flow: arteriovenous fistula, massive splenomegaly from primary hematologic disease
Hepatic	Presinusoidal: schistosomiasis, other periportal disorders (eg, primary biliary cirrhosis, sarcoidosis, congenital hepatic fibrosis), idiopathic portal hypertension
	Sinusoidal: cirrhosis (all etiologies)
	Postsinusoidal: veno-occlusive disease
Posthepatic	Hepatic vein thrombosis (Budd-Chiari syndrome)
	Membranous obstruction of inferior vena cava
	Cardiac causes (eg, constrictive pericarditis, restrictive cardiomyopathy)

Prehepatic normal Liver US

What causes liver cirrhosis will cause portal hypertension

❖ **Mortality and morbidity**:

- Variceal hemorrhage is the most common complication of PH:
  - 90% with cirrhosis develop varices.
  - 30% of these bleed.
  - The first episode is estimated to carry a mortality of 30-50%.

❖ **Clinical features:**

- **Symptom**:
  - Patients with cirrhosis frequently develop anorexia, generalized malaise and weight loss.
  - Hematemesis (due to upper GI bleeding) +/- melena.
  - Chronic liver disease symptom
- **Examination**:
  - Cirrhosis: hepatosplenomegaly, ascites , jaundice and spider naevi. Slurring of speech, flapping tremor or dysarthria may point to encephalopathy.
  - Serum bilirubin may be elevated and the serum albumin depressed, anemia may be present and the leukocyte count raised (or depressed if there's hypersplenism). Prothrombin time and other indices of clotting may be abnormal.
  - Hypotension, tachycardia.
  - Stigmata of liver disease.

In Portal Hypertension due to increase pressure develops Portosystemic anastomosis (collaterals)

- Esophageal varices (collateral between hemizygous and azygous(systemic) and short gastric and left gastric (portal system)
  as a complication = hematemesis
- Anorectal Varices (hemorrhoids) between superior rectal vein (portal system) and middle rectal vein (systemic circulation) but is least common
  Caput madusa = ligamemtum of teres reopen in portal Hypertension

Portal Hypertension

❖ Assessment:

- **Acute setting:**
  - ABC
  - History
  - If a patient came to the emergency with an upper GI bleeding in a form of hematemesis, after resuscitation and investigations, our next step after we put NGT and foley catheter is the ENDOSCOPY
- **Elective setting:**
  - History of chronic liver disease
  - Other differential diagnosis
  - Stigmata of liver disease

❖ Investigations:

- **Labs**
  - Acute setting AST/ ALT not helpful in acute
    - CBC , LFTs, Albumin, PT/PTT, U&E, CXR , INR. Check the liver synthetic function
    - Cross match 10
  - Chronic setting:
    - Hepatitis serology, ANA, Antimitochondrial antibodies, Alpha 1-antitrypsin deficiency.
- Radiology:
  - CXR
  - US, CT if the patient is a known case of cirrhosis the US and CT won't help that much, but if he wasn't we do US, CT maybe helpful if ultrasound isn't clear
- Endoscopy: the key investigation during an episode of active bleeding, this allows the detection of varices and defines whether they are or have been the site of bleeding
- Hepatic venous pressure gradient. The only test that can give us the portal pressure reading, we use it only before the surgery to see if the patient have portal hypertension or not.

❖ Child's grading system : Patients with grade A have a good prognosis, whereas those in grade C have the worst prognosis. (need to know how to calculate score + grade) MUST WATCH VIDEO!

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

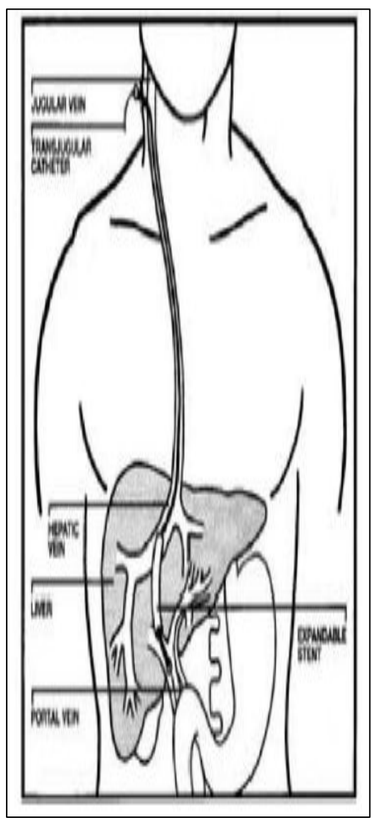
Points scored			
Criterion	**1**	**2**	**3**
Encephalopathy	None	Minimal	Marked
Ascites	None	Slight	Moderate
Bilirubin (μmol/l)	< 35	35–50	> 50
Albumin (g/l)	> 35	28–35	< 28
Prothrombin ratio	< 1.4	1.4–2.0	> 2.0

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

Portal Hypertension

❖ Treatment:

- **Endoscopy:** diagnostic and therapeutic
  - Endoscopic variceal ligation (EVL) by application of a band . The most common procedure bc it has less complications and controls 80 -90% of bleeding.
  - Sclerotherapy (ethanolamine injection) .It has the same rate control of bleeding but with more complication
- **Pharmacology:** if he doesn't improve with endoscopy
  - Octreotide.
  - Vasopressin
- **Balloon tamponade:** if hemorrhage is torrential and prevents direct injection.
  - Four-lumen minnesota, which allows:
  - Aspiration of gastric contents.
  - Compression of the esophagogastric varices by the inflated gastric balloon.
  - Aspiration of the esophagus and pharynx to reduce pneumonic aspiration.
  - Balloon tamponade should not be left for more than 24-38 hours for the fear of causing esophageal necrosis.
  - Sengstaken-Blakemore tube.
- **Transjugular intrahepatic portosystemic stent shunting (TIPSS):**
- A metal stent is inserted via the transjugular route using a guidewire passed through the hepatic vein to the intrahepatic branches of the portal vein. The technique is a relatively safe means of decompressing the portal system as general anesthesia and laparotomy are avoided.
- Contraindication:
  - Heart Failure
  - Prehepatic cause (normal liver)
- Indications:
  - Uncontrolled variceal hemorrhage from esophageal, gastric, and intestinal varices that do not respond to endoscopic and medical management.
  - Refractory ascites.
  - Hepatic pleural effusion (hydrothorax).
- **Surgical:**If the patient doesn't improve or we don't have TIPSS
  - Shunt:
    - Selective.
    - Non-selective.
  - Devascularization



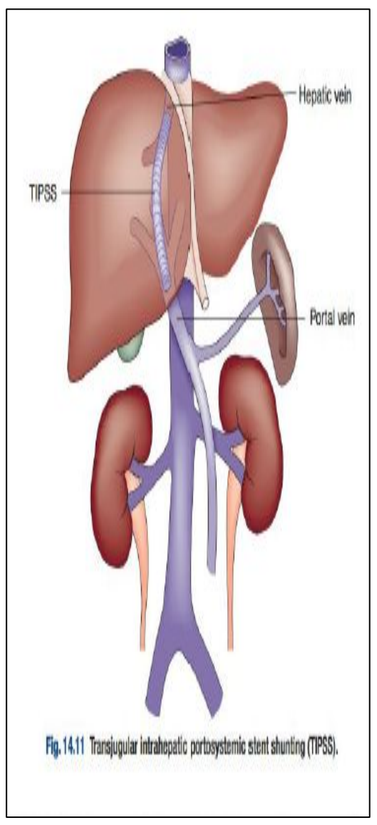


Fig. 14.11 Transjugular intrahepatic portosystemic stent shunting (TIPSS).

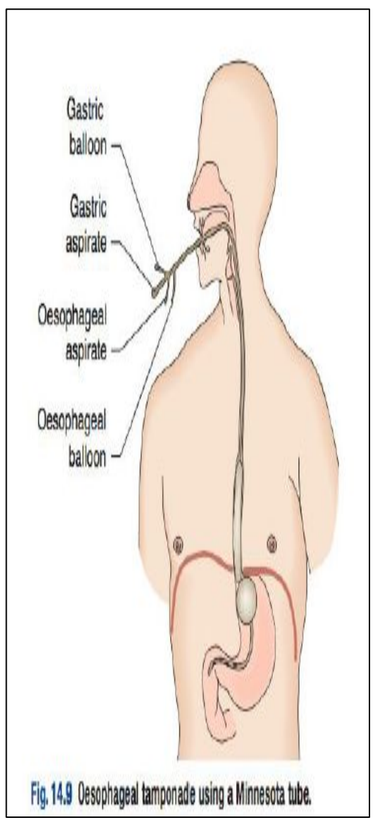
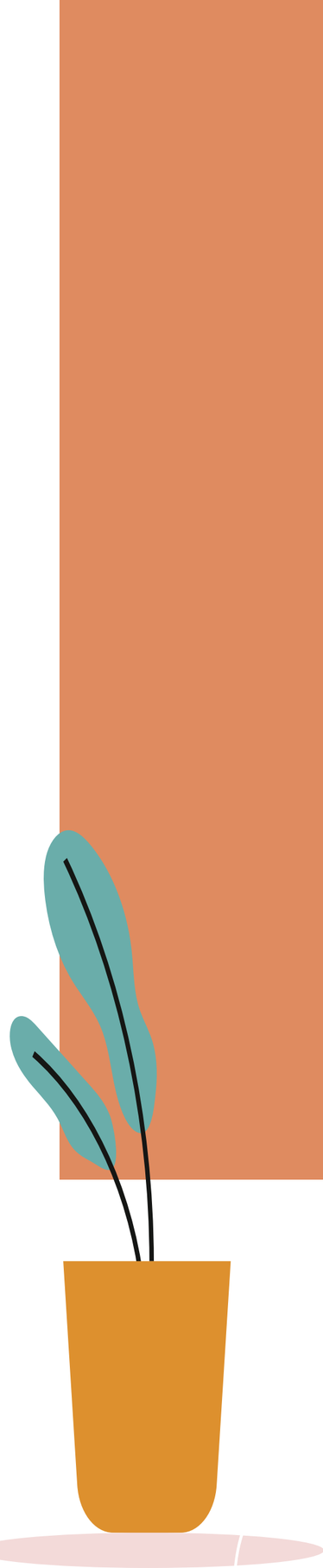


Fig. 14.9 Oesophageal tamponade using a Minnesota tube.

Extra: Surgical 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)



Extra: Surgical Recall

- 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

Summary

congenital			
	Diagnosis	**Treatment**	**Notes**
Liver cyst	- asymptomatic - MRI to assess communication biliary system	- only if symptomatic - deroofing - aspiration (high reoccurrence)	- it does not communicate with biliary system (if it does =biliary cyst adenoma , and its malignant u have to remove it)
Cavernous Hemangioma (Most common benign Tumor)	centripetal "filling in" Of contrast **CT or MRI**	only if symptomatic remove it If it ruptured;angioembolization	

Infection				
	Symptoms	**Diagnosis**	**Organism**	**Treatment**
Pyogenic Liver Abscess	acute pain, fever, jaundice	xray: air in the liver (if gas forming bacteria) CT: central hypodense with rim enhancement	from hepatic artery:gram+ aerobes (staph) from portal vein: gram - aerobes (e coli, klebsiella)	drainage + antibiotic
Amoebic Liver Abscess	chronic pain, weight loss, diarrhea	CT: hypodense with rim then another hypodense serology:amoebic protein	Entamoeba histolytica	metronidazole (if he didn't get better within 3 days he probably have 'pyogenic' so aspirate)
Hydatid Disease	-incidental -obstructive jaundice	CT: well defined +daughter cells xray: wall calcification serology: immunoblotting (first line test+ used for follow up)	Echinococcus granuliosa	1st medical for 1 month: albentazole then surgical: -deerofing -pericystectomy -resection -PAIR(only if surgically not fit bc may cause anaphylaxis)

Note for hydatid disease:

- **hydatid cyst layers: -pericyst: host tissue -ectocyst: external layer of cyst -fundocyst: germinative layer**

Impprtant Q

1-patient had pancreatic abscess or appendicitis after that developed fever what is the diagnosis and most common organism? portal empyema (pyogenic) , gram - aerobes

2- patient had pancreatic abscess or appendicitis after that developed fever what is the diagnosis and most common organism? portal empyema (pyogenic) , gram - aerobes

Summary

TUMORS				
	Risk factors	**investigation**	**treatment**	**notes**
liver cell adenoma	main cause: estrogen and anabolic steroids	CT,US	female: less than 5 cm then stop ocp male or more than 5cm: surgery	complication: rupture causes anaphylaxis
focal nodular hyperplasia	Female	ct arterial phase: hyperintense and hypointense center venous: hypo intense late: central shining star	observation	incidental
hepatocellular carcinoma (hepatoma)	same as cirrhosis: hepB hepC, alcoholic	CBC, CT, MRI (early hyper enhancement and early wash out) diagnosis: - >1cm: one image with characteristics - >1cm but feature is not typical then do cytology - <1cm 3-6 month follow up	• transplant: milan's criteria: -single tumor 5cm or less -no more than 3 tumor nodules each one 3cm or less • resection: non cirrhotic , child A cirrhosis • locoregional therapy: TACE, RFA • chamo: sorafenib	child classification: A: good liver reserve B: moderate liver reserve C: low liver reserve
cholangiocarcinoma	chronic parasitic infestation of biliary tree	labs: CA19-9 CT: (early hyper enhancement and NO early wash out)	-resection -chemo	
metastatic malignant tumor		-tumor markers: CEA (colon), CA19-9 (biliary ,pancreas), CA125 (ovarian) -PET CT	-resection -chemo	most common metastatic tumor to liver is colon cancer

Q:most common metastatic tumor to liver is colon cancer?
rupture of liver cell adenoma

QUIZ

1) The most common benign tumors in the liver are?
A- Cavernous haemangiomas.
B- Adenomas.
C- Focal nodular hyperplasia.
D- Cholangiocarcinoma.

2) Portal hypertension is high risk of bleeding when pressure is?
A- >6 mmHg
B- >12 mmHg
C- >11 mmHg
D- >10 mmHg

3) What is the most common complication of portal hypertension?
A- Ascites.
B- PHT.
C- Esophageal Varices.
D- Splenomegaly.

4)Which of the following is the most effective therapy for refractory hematemesis due to Esophageal Varices?
A- Sclerotherapy.
B- Vasopressin. 14
C- Sengstaken-blakemore tube.
D- Minnesota.

5) Which of the following test is first line test to diagnose Hydatid disease?
A- Immunoelectrophoresis.
B- CBC.
C- Enzyme linked immunosorbent assay.
D- Immunoblotting.

6)Which one of the following statements is accurate regarding a patient with variceal bleeding?
A. Low-dose propranolol alone is the regimen of choice for secondary prophylaxis; it has been shown to reduce the risk of rebleeding
B. If the patient has a history of hypertension, it may be appropriate to use metoprolol succinate to control portal hypertension
C. Most patients should be started on propranolol and isosorbide mononitrate to decrease blood pressure and reduce the risk of repeated bleeding
D. If endoscopic band ligation stops the bleeding, further pharmacologic therapy is not warranted

1-A 2-B 3-A 4-A 5-D 6-A

MCQ for portal hypertension