



432 Surgery Team

21 portal hypertensive & surgical disease of liver



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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

Objectives

Not given - -

PORTAL HYPERTENSION

1 INTRODUCTION

Portal hypertension (defined as hydrostatic pressure >5 mmHg) results initially from obstruction to portal venous outflow. Obstruction may occur at a presinusoidal (portal vein thrombosis, portal fibrosis, or infiltrative lesions), sinusoidal (cirrhosis), or postsinusoidal (veno-occlusive disease, Budd Chiari syndrome) level. Cirrhosis is the most common cause of portal hypertension; in these patients, elevated portal pressure results from both increased resistance to outflow through distorted hepatic sinusoids, and enhanced portal inflow due to splanchnic arteriolar vasodilation.

1.1 CAUSES:

Causes of Portal hypertension can be classified as:

☐ Cirrhotic (definition)

☐ Non-cirrhotic: most important non-cirrhotic causes are: schistosomiasis and splenic vein thrombosis (mainly caused by hypercoagulable state and pancreatitis)

☐ 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 veins rises above 12 mmHg; patients with lower values do not form varices and do not bleed.

1.2 SYMPTOMS OF PORTAL HYPERTENSION

☐ Asymptomatic: portal hypertension is asymptomatic until complications develop, where patients present according to the ongoing pathological process. These complications are in the form of:

☐ Gastroesophageal varices

☐ Ascites

☐ Splenomegaly: can sometimes cause dull abdominal pain.

☐ Underlying disease

☐ The risk of Esophageal Varices development can be predicted by the Child-Pugh score, calculated by computing different values for certain conditions like: presence of Ascites, encephalopathy, bilirubin and albumin levels, and other factors.

☐ Red wale signs are longitudinal red streaks seen in endoscopies on varices that resemble red corduroy wales. See below>

2 VARECIAL BLEEDING

☐ Approximately one-third of all patients with varices will develop variceal hemorrhage

☐ **A major cause of morbidity and mortality in patients with cirrhosis.** ☐ **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, varices tend to bleed profoundly, when they rupture.



2.1 PREVENTION OF VARECIAL BLEED

☐ AASLD RECOMMENDATIONS — Recommendations for prevention of variceal bleeding have been issued by the American Association for the Study of Liver Diseases

☐ These Recommendations are as follows:

❑ No treatment is given to people who haven't developed Cirrhosis.

In patients who have **compensated cirrhosis** and **small varices**(seen by endoscopy) that have not bled but have criteria for increased risk of hemorrhage (Child B/C or presence of red wale marks on varices), **nonselective beta blockers**

o In patients with **medium/large varices** that have not bled, **nonselective beta blockers** (propranolol or nadolol) is recommended or **undergo EVL**(**esophageal variceal ligation**)

o In patients who receive beta-blockers, a follow-up EGD is not necessary.

o **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.

TREATMENT OF ACTIVE VARECIAL BLEED:

- ❖ Initial therapy: hemodynamic **RESUSCITATION**, prevention and treatment of complications. **Hb=8 g/dL**
- ❖ Prophylactic antibiotics, preferably before endoscopy (although effectiveness has also been demonstrated when given after).**for 7 days in any patient with cirrhosis and hemorrhage**
Suggest intravenous ceftriaxone (1 g IV) or Cipro (400 mg IV BID)
- ❖ UGD should be performed for diagnosis and possible treatment (**within 12h, diagnostic and therapeutic by EVL or Endoscopic sclerotherapy**)
- ❖ Suggest **terlipressin** in countries where it is available and somatostatin or **OCTREOTIDE** (50 mcg bolus followed by 50 mcg/hour by intravenous infusion) where terlipressin is unavailable.
- ❖ If the patient's bleeding is still not controlled, Surgery mostly in the form of TIPS is usually performed.

⊞ The principal complications that cause death are aspiration pneumonia, sepsis (antibiotic administration), acute-on-chronic liver failure, hepatic encephalopathy (lactulose and treatment of other precipitating factors), and renal failure (careful fluid balancing and avoid giving nephrotoxic substances)

⊞ Vasoactive substances agents directly constricts mesenteric arterioles and decreases portal venous inflow, thereby reducing portal pressure. However, Terlipressin is the only pharmacological agent shown to reduce mortality in compared to placebo.

⊞ EVL should be performed as soon as possible. It involves the placement of rubber bands around a portion of oesophageal mucosa that contains the varix. EVL is superior to sclerotherapy in general, but sclerotherapy maybe used in cases when esophageal visualization is limited due the bleeding mainly because sclerotherapy is quicker and provides better visualization of the esophagus.

*Balloon tamponade should be used as a **temporizing** measure (**maximum24 hours**) in patients with uncontrollable bleeding for whom a more definitive therapy (eg, TIPS or endoscopic therapy) is planned.

Balloon tamponade :Nasogastric tube but in the end of it there's a balloon .. They insert it to the esophagus and upper stomach and then inflate the balloon => pressure on the bleeding to stop it .. it used temporally to transport the patient when he's in hospital with no interventional radiologist. They put it while transporting the patient to other hospital or on the ED while waiting for the endoscopic or interventional radiologist to come .. we don't use it more than 24 hours because it might cause perforation.

*Note: Dr.Mazen mentioned that **Proton pump inhibitors should** be added even in cases with known Liver disease. The rationale behind this is A) that peptic ulcer bleeding hasn't been ruled out and B) decreased acid secretion may help in the healing of these gastrointestinal bleeds. I did not know where to add this point, so I did here

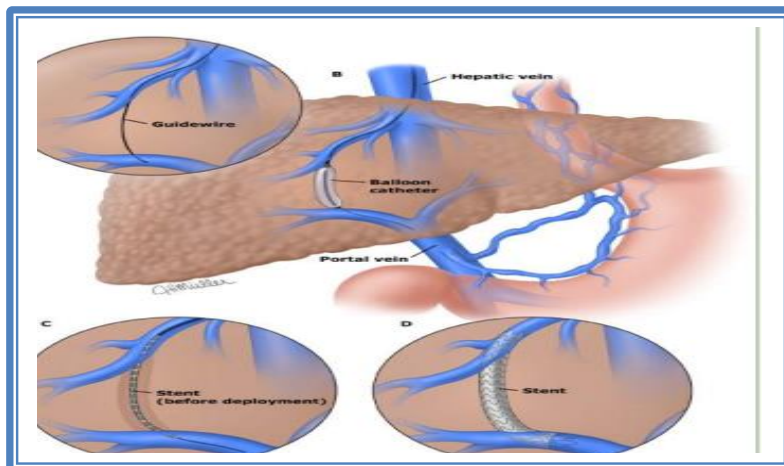
Salvage Treatment

- ❖ TIPS (**transjugular intrahepatic portosystemic shunt**)
- ❖ Surgery is one 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. Thus, a portacaval shunt would be preferable in patients with marked ascites.

TIPS is done by interventional radiologist and under the visualization by ultrasound

Shunt Surgery:

Definition: Transjugular intrahepatic portosystemic shunts (TIPS) involve creation of a low-resistance channel between the hepatic vein and the intrahepatic portion of the portal vein (usually the right branch) using angiographic techniques. The tract is kept patent by deployment of an expandable metal stent across it, thereby allowing blood to return to the systemic circulation. Portosystemic shunts are classified as nonselective, selective, and partial, depending on how much hepatic portal flow is preserved.



Types Of Shunts:

Nonselective — those that decompress the entire portal tree, such as porta-caval shunts (portal connected to cava without passing to liver)

Selective — those that compartmentalize the portal tree into a decompressed variceal system while maintaining sinusoidal perfusion via a hypertensive superior mesenteric-portal compartment, such as a distal spleno-renal shunt

Partial — those that incompletely decompress the entire portal tree and thereby also maintain some hepatic perfusion

Non-shunt operations generally include either esophageal transection (in which the distal esophagus is transected and then stapled back together after varices have been ligated) or devascularization of the gastroesophageal junction (Sugiura procedure).

Ascites : (our doctor skip it)

- ❖ Cirrhosis is the most common cause of ascites in the United States, accounting for approximately 8%
- ❖ Ascites is the most common complication of cirrhosis
- ❖ Fluid leaks from the surface of the liver and intestine.
- ❖ Factors responsible: portal hypertension, decreased ability of the blood vessels to retain fluid, fluid retention by the kidneys, and alterations in various hormones and chemicals that regulate bodily fluids

TREATMENT OF ASCITES :

- ❖ Dietary sodium restriction is a central component, 2000 mg / day
- ❖ Patients should be instructed to avoid NSAIDs, which can cause sodium retention and affect renal function
- ❖ Fluid restriction is equivocal and not strongly recommended
- ❖ 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.
- ❖ Peritoneovenous shunts (LeVeen or Denver) or surgical portosystemic shunts have very limited indications

COMPLICATIONS OF ASCITES:

- ❖ **Spontaneous bacterial peritonitis**: (SBP) is an infection of preexisting ascitic fluid without evidence for an intra-abdominal secondary source such as a perforated viscus
- ❖ The diagnosis is established by:
 - positive ascitic fluid bacterial culture, and/or
 - elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count (≥ 250 cells/mm³)

PORTAL VEIN THROMBOSIS:

Causes:

- ❖ Can be picked up **Ultrasound with Doppler** flow studies, **CT scanning**, and **magnetic resonance angiography**
- ❖ UGD should be performed to establish whether varices are present
- ❖ In cases of detected acute thrombosis (e.g. pancreatitis) Anticoagulation therapy for at least three months starting with low molecular weight heparin and shifting to oral anticoagulation as soon as the patient's condition has stabilized.
- ❖ Anticoagulation should be continued long-term in patients with acute portal vein thrombosis who have a permanent thrombotic risk factor that is not correctable.
- ❖ Mainly caused by abdominal sepsis, cirrhosis, pancreatitis ..etc.

BLEEDING FROM PORTAL VEIN THROMBOSIS:

- ❖ Gastric fundal varices: endoscopic variceal obturation using tissue adhesives such as cyanoacrylate is preferred, where available. Otherwise, endoscopic variceal ligation is an option.
- ❖ **Splenectomy** is curative for cases of splenic vein thrombosis and gastric varices formation.
- ❖ TIPS should be considered in patients in whom hemorrhage from fundal varices cannot be controlled or in cirrhosis whom bleeding recurs despite combined pharmacological and endoscopic therapy.

Liver resection: "liver is composed of 8 segments and if we resect, we need to leave at least two adjacent segments to maintain the normal function of the liver".

Benign causes

- * **Hydatid cyst** (parasitic infection treated it first with albendazole, then resect it)
- * Abscess
- * **Large adenoma** "more common in females" (5 cm) **INDICATIONS FOR SURGERY**
Liver adenoma caused mainly by contraceptive pills and it is usually solitary and – encapsulated. It is usually detected by US,CT and serum alpha-fetoprotein is normal.
"principle and practice of surgery book"
- * Intrahepatic biliary stones
 - Oriental cholangiohepatitis
- * Hemangioma Multiple cysts FNH (focal nodular hyperplasia) => hemangiomas are abnormal distribution of normal tissues.
- * Choledochal cyst (congenital cyst in the bile duct)
- * Cystadenoma.

Malignant

- * Primary liver cancers:
- HCC
- Cholangiocarcinoma
 - * **Secondary liver cancers** : (outside the liver) **more likely metastasis from Colorectal cancer** and it is not the Most common metastasis to liver **But it's the most common liver metastasis which indicates a liver resection.** It will increase 5 years survival in 60% of the patients
 - * Neuroendocrine tumors. Rare tumors.
 - * Others (isolated, stable, chemo responsive)

solated = metastasize to the liver only

stable = disease is not progressed as we treat it.

Chemo-responsive= we have to test it's chemo-responsiveness before the operation!

e.g pt. present with breast cancer and metastasize to the liver, shall we operate
we have to know is it isolated? Stable? chemo-responsive?
if ALL these criteria are fulfilled => we can operate!

Echinococcosis disease:

- ❖ Parasitic infection we start them on albendazole for enough time to kill the parasite .. > then we take away the cyst by surgical resection of the cyst

HCC: (usually we don't resect here becuz it is a diffused neoplasm)

- ❖ Cirrhosis most common cause
- ❖ Follow Roles of resectability
- ❖ Survival benefit those who did surgery have good major survival

Cholangiocarcinoma :

- ❖ Resectability
- ❖ The role of bile duct

CRC metastasis (colorectal cancer)

- ❖ Metastasis liver tumors is more common than the primary tumors
- ❖ CRC metastasis due to Portal connection
- ❖ Survival benefit is massive
- ❖ Limits of resection up to 70%-80% of liver can be taken out and the liver function is still maintained
- ❖ Increase resectability
 - Decrease disease
 - Improve function

The liver has the ability to regenerate , when we

remove 70 - 80% of the liver

in 8 weeks (2 months) it comes back to almost its normal size ! -when less than 20% of the liver is normal we cannot operate less

because it not enough to maintain the liver function

so what we do to increase the percentage of normal liver? , we make the liver grow before the operation by making the liver think we resect part of it so it will grow

how ?

by asking the interventional radiologist to block part of the portal vein of that part which we want it to grow => the liver think this part is resected => it will regenerate to restore that part !

or

we divide the surgery into small multiple surgeries, we take small portion of the tumor then the liver grow => take another portion etc.. until we remove the whole tumor.

We'd like to thank all the doctors, you really did
a great job and we highly appreciate it

Dr.Arwa Almashaan

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Questions

1) Portal hypertension is diagnosed when pressure is:

- A. >5 mmHg
- B. >12 mm Hg
- C. >30mm Hg
- D. >40 mmHg

2) What is the most common complication of cirrhosis?

- A. Ascites
- B. PHT
- C. Esophageal Varices
- D. Splenomegaly

3) Echinococcosis liver disease caused by Echinococcus granulosus:

- A. Is a neoplasm.
- B. Is endemic to parts of United States.
- C. Is usually curable by resection.
- D. Is more deadly than in its Echinococcus multilocularis form.

4) A portal venous pressure of 30 mm. Hg (elevated) and a hepatic venous wedge pressure of 5 mm. Hg (normal) may be associated with which of the following causes of portal hypertension?

- A. Portal vein thrombosis.
- B. Alcoholic cirrhosis.
- C. Schistosomiasis.
- D. Alcoholic hepatitis.

5) Which of the following is the most effective definitive therapy for both prevention of recurrent variceal hemorrhage and control of ascites?

- A. Endoscopic sclerotherapy.
- B. Distal splenorenal shunt.
- C. Esophagogastric devascularization (Sugiura procedure).
- D. Side-to-side portacaval shunt.
- E. End-to-side portacaval shunt.

6) Which of the following complications of portal hypertension often require surgical intervention (for more than 25% of patients)?

- A. Hypersplenism.
- B. Variceal hemorrhage.
- C. Ascites.
- D. Encephalopathy.

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Answers:

1st Questions:B

2nd Questions:A

3rd Questions:C

4th Questions:A-C

5th Questions:D

6th questions:**B**