PATHOPHYSIOLOGY OF ASCITES

لا يمتطي المجد من لم يركب الخطر ... ولا ينال العلى من قدم الحذرا

و من اراد العلا عفوا بلا تعب ... قضى و لم يقض من إدراكها وطرا

Revised by

هشام الغفيلي & خولة العماري

Important Doctor's notes Extra explanation

Introduction:

★ Ascites is of Greek derivation ("askos") and refers to a bag or sack and describes pathologic fluid accumulation within the peritoneal cavity.

★ Most patients (85%) with ascites have cirrhosis.

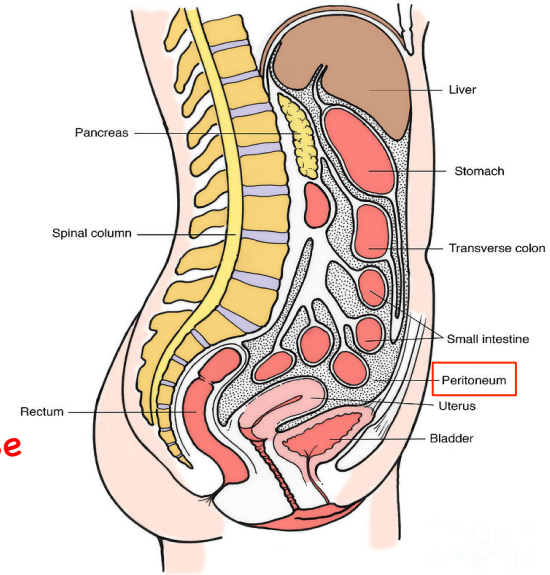
★ The most common causes of cirrhosis at the present time are chronic viral hepatitis and alcoholic liver disease.

PERITONEAL CAVITY

★ It is a potential space between the parietal peritoneum and visceral peritoneum; the two membranes separate the organs in the abdominal cavity from the abdominal wall.

★ Derived from the coelomic cavity of the embryo.

★ Largest serosal sac in the body and secretes approximately 50 ml of fluid per day.



PERITONEAL FLUID

★ Normal, lubricating fluid found in the peritoneal cavity.

★ The fluid is mostly water with electrolytes, antibodies, white blood cells, albumin, glucose and other biochemicals.

:

★ Reduce the friction between the abdominal organs as they move around during digestion.

Usually we have a small amount of fluid in the peritoneal cavity, which prevents ulceration and friction. But if the fluid is more then normal it's ascites.

CAUSES OF ASCITES:

- **Cirrhosis** — MOST COMMON
- **CHF**
- **Infection (TB)**
- **Malignancy**
- **Nephrotic syndrome**
- **Pancreatic or biliary ascites**

PATHOGENESIS OF ASCITES:

★ Essential hypertension will not lead to ascites , in long term it will lead to heart failure which cause ascites !

PATHOGENESIS

1-Increased hydrostatic pressure.

→

2-Decreased colloid osmotic pressure.

eg. Nephrotic syndrome

→

3-Increase in the permeability of peritoneal capillaries.

Like in some infections.

eg. TB

↓

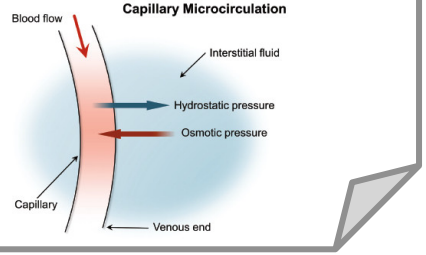
4-Leakage of fluid into the peritoneal cavity.

Like in chronic pancreatitis.

←

5-Miscellinious

Remember always if there's an imbalance between the hydrostatic pressure and the oncotic pressure (colloid osmotic pressure), the patient will have fluid accumulation in surrounding space.



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PATHOGENESIS OF ASCITES:

Cirrhotic Ascites:

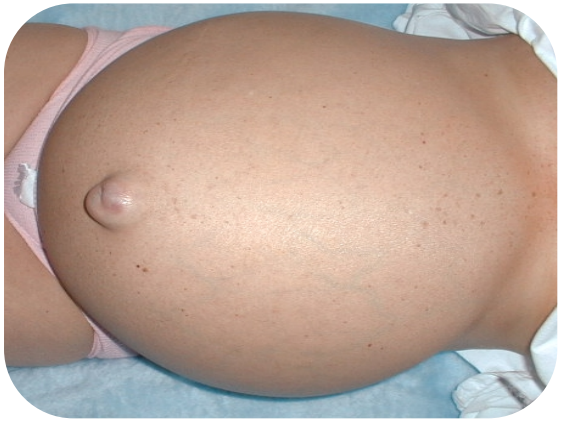
The most recent theory of ascites formation, the **"peripheral arterial vasodilation hypothesis"**

This happens as a consequence of portal hypertension.

Portal hypertension:

Portal hypertension > 5 mmHg.

Approximately 2/3 of the hepatic blood supply is provided by portal venous blood.
Hepatic blood flow is normally about 1500 mL/minute.
(70% portal vein, 30%hepatic artery) unlike other organs.



Pulging + hernia
Can patient with ascites develop hernia ? **yes** !
Because u have one room and u fill it with fluids !
the pressure will increase causing hernia !

Pathophysiology and causes:

The pathogenesis of portal hypertension involves the relationship between portal venous blood flow and the resistance to this blood flow within the liver (the portohepatic resistance) and within portosystemic collateral blood vessels (the portocollateral resistance) that form during the evolution of portal hypertension.

VERY IMPORTANT !! "IF U UNDERSTAND THIS SLIDE U WILL UNDERSTAND THE WHOLE LECTURE"

DR.BANDAR

how do we measure the portal pressure??!

the interventional radiologist well insert a catheter that has a ballon into the internal jugular vein >>> inferior vena cave >>> common hepatic vein >>> inside the liver >>> into small venules >>> first they will measure the pressure when the balloon is deflated and this is the free pressure outside "before" the liver.

Then inflate the balloon to measure the wedge pressure which indicate the pressure inside the liver !

Then gonna subtract BOTH and this is called **Hepatic Venous Pressure Gradient** (HVPG)

Normal Hepatic Venous Pressure Gradient should be **less than** 5 mmHg !

In order for patient to develop ascites they need to have HVPG of 8 mmHg or above

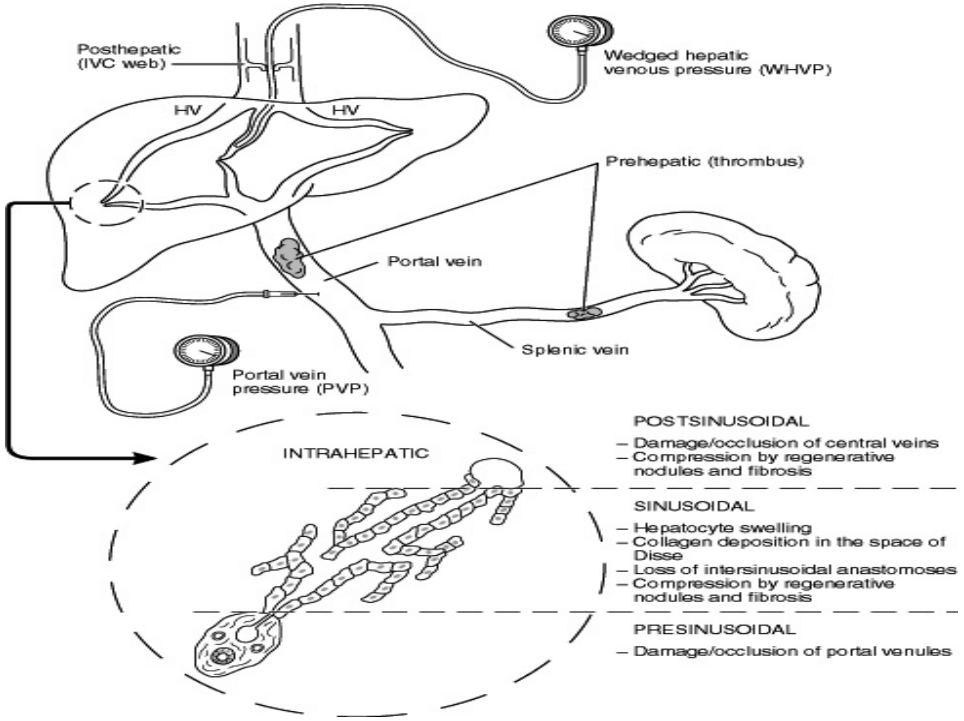
In order for patient to have esophageal varices they need to have HVPG of 11 mmHg or above

NOTE :

Varices can happen ANYWHERE!!

Patient could have both ascites and esophageal varices !

Like when his HVPG is 11 FOR SURE he has ascites and have esophageal varices, but if the HVPG is 9 the patient will have ascites and less likely to have esophageal varices !



So patient is said to have portal hypertension when his Hepatic Venous Pressure Gradient above 5 mmHg !!

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PVP = Portal Venous Pressure

WHPV = wedged Hepatic Venous Pressure

HVPG = Hepatic Venous Pressure Gradient

Causes of portal hypertention

Pre-hepatic

any issue with the portal vein

Intra-hepatic

post-hepatic

any issue with the hepatic vein also including the IVC

In both **pre-hepatic** and intrahepatic **pre-sinusoidal** portal hypertension (PVP) is elevated with Normal (WHVP) and (HVPG).

In **post-hepatic** portal hypertension, the WHVP equals the increased PVP.

presinusoidal

sinusoidal

postsinusoidal

What I want u to remember about it is *Schistosoma*

Patient with schistosomiasis they may not be cirrhotic but they will have portal hypertension!

Anything leads to liver cirrhosis Like hepatitis B or C or alcoholic liver disease.. etc.

In **sinusoidal** and **intrahepatic post-sinusoidal** portal hypertension, the (WHVP) tends to approximate or equal the directly measured (PVP) and the HVPG is increased.

extension of thrombus from hepatic vein to the venules

 # Budd-Chiari syndrome: there is thrombus or micro thrombi!

 # Veno-occlusive disease: in patient who has BM transplant & receiving chemotherapy

★ Most of the relevant information has been provided by **direct measurement of pressure in the portal system** and **indirect estimation of the intra-sinusoidal pressure from the WHVP** in conjunction with **details of the morbid anatomic features.**

Portal Blood Flow :

★ Primary High Portal Flow States Although uncommon, conditions leading to high-flow states in the portal system (arterioportal fistulas, splenomegaly resulting from myelofibrosis or myeloid metaplasia) are well-recognized causes of portal hypertension.

★ Portal hypertension is maintained during collateral formation by increased portal inflow, and, as a consequence, portal hypertension persists even when all portal flow escapes through collaterals.

Patient with liver cirrhosis will have increase in the pressure inside the liver >> collaterals surrounding the liver will dilate through the action of nitric oxide, trying to decrease the portal pressure ! but unfortunately it wont be decreased to the point that's its gonna be normal, so will remain high ! where we can see these collaterals ? in the esophagus + in the stomach + as caput medusae in the abdomen + spider nevi !

★ Hyperdynamic Circulation of Portal Hypertension its **hallmarks** are increased cardiac output and reduced arterial blood pressure.

Patient with chronic liver disease and portal hypertension will have normal or LOW systemic blood pressure because of the dilation of the collaterals! Then why the patient will have hyperdynamic circulation ? once the vasodilation happen there will be increase in the plasma volume in these vasodilated vessels > increase in cardiac contractility! > This is why its hyperdynamic circulation despite that the blood pressure is low !

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★ Collective data from hemodynamic studies in patients with portal hypertension who was treated with selective and nonselective B-blockers point to a role for both increased cardiac output (β1 receptor-mediated) and splanchnic arteriolar vasodilation (β2 receptor-mediated) in generating the increase in portal venous inflow .

beta blockers doesn't only used to decrease the systemic circulation ! it also has a direct effect on portal pressure ! it will decrease the portal pressure by decreasing the cardiac contractility which will decrease the systemic then the portal pressure.. but remember ! some patient will **not** tolerate beta blockers because they have bad blood pressure to start with from advanced liver disease secondary to portal hypertension. On the other hand there are patients with liver cirrhosis and portal hypertension who may tolerate beta blockers!

Why patients with liver cirrhosis and portal hypertension will develop ASCITES ?

Decreasing the oncotic pressure is a cooperate may lead to ascites but its not the main reason !

After the vasodilation of the collaterals, the kidneys will be HYPOperfused >> there will be signals to the kidneys telling them we are intravasculary depleted >> that will stimulate the Renin-Angiotensin >> retention of sodium and water >> the water gonna accumulate somewhere >> with the continuation of retention and reabsorption >> the sodium and water will go either to the peritoneal cavity or to the leg >>in advanced stages patient may have Hepato-Renal syndrome .

YOU CANT BLAME THE KIDNEYS FOR THAT !! YOU SHOULD BLAME THE LIVER :(

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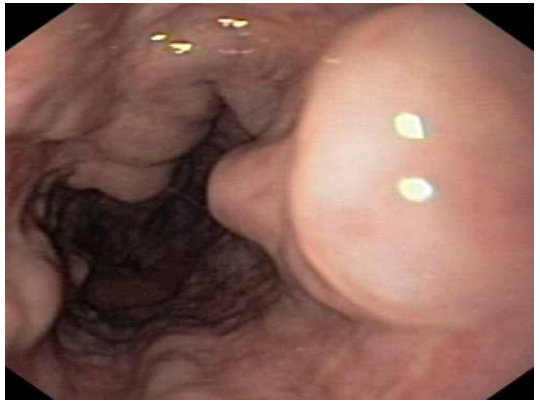
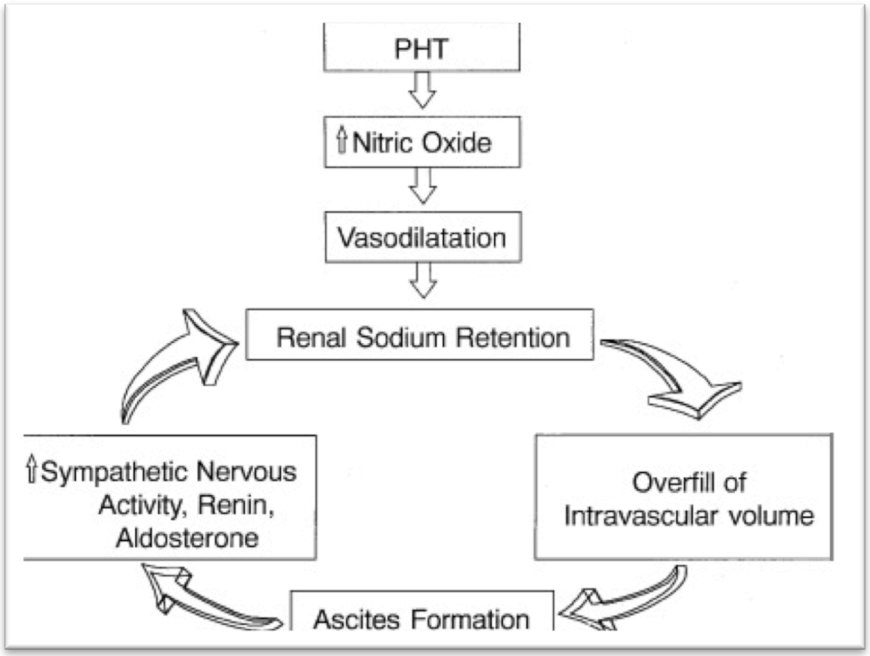
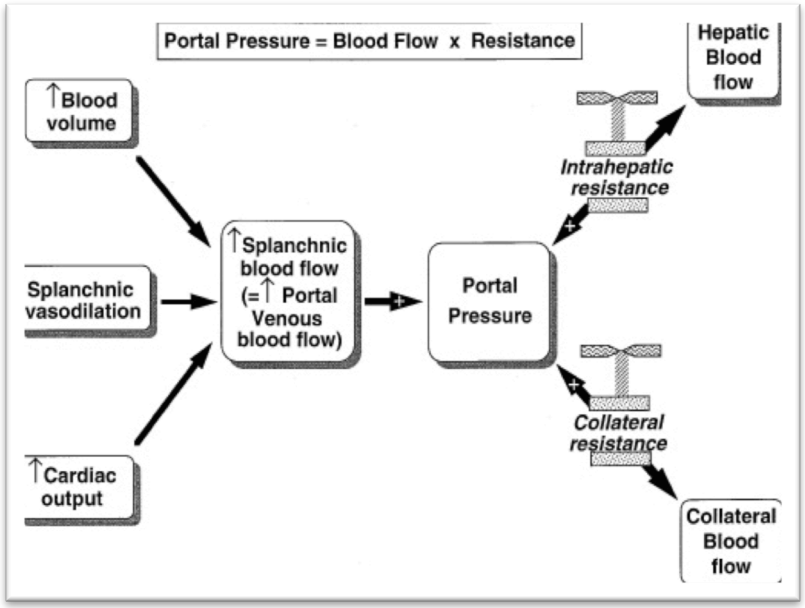
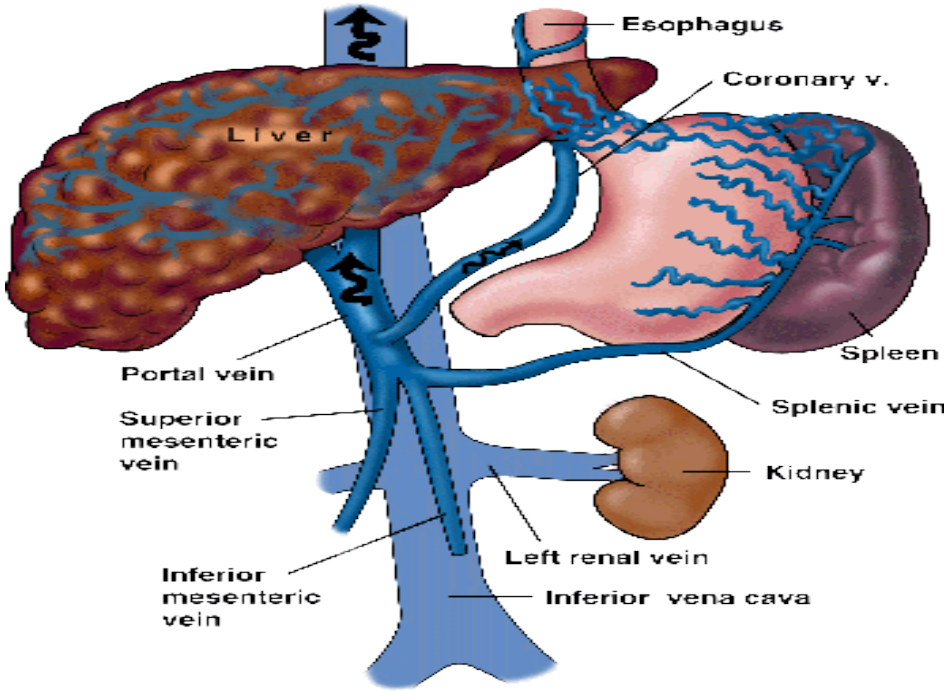
Patient with esophageal varices usually present with hematemesis









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★ Noncirrhotic Ascites ★

Related to	About it	Pathophysiology
Malignancy-Related Ascites	depends on the location of the tumor e.g: Peritoneal carcinomatosis produce proteinaceous fluid by tumor cells lining the peritoneum cause extracellular fluid to enters the peritoneal cavity to reestablish oncotic balance.	
Heart Failure	In high-output or low-output heart failure	increased hydrostatic preasure
Chylous Ascites	in patients with malignant lymphoma appears to be caused by lymph node obstruction by tumor and rupture of chyle-containing lymphatics.	
Nephrotic Syndrome	where effective arterial blood volume decreased, and the vasopressin, renin-aldosterone, and sympathetic nervous systems are activated	decreased colloid osmotic pressure
Tuberculosis, Chlamydia infection, and coccidioidomycosis	cause ascites through the production of proteinaceous fluid	increased permeability of peritoneal capillaries
Pancreatic or Biliary Ascites	fluid forms by leakage of pancreatic juice or bile into the peritoneal cavity or by a "chemical burn" of the peritoneum	leakage of fluid into the peritoneal cavity

CLINICAL FEATURES OF ASCITES

✚ History

★ Ascites frequently develops as part of the patient's first decompensation of liver disease.

★ It can be associated with other features of liver decompensation such as jaundice or encephalopath.

★ viral hepatitis, Risk factors:

ivdu, blood tx, sex, acupuncture, tattoos, ear piercing, and country of origin.

★ Non-alcoholic-steatohepatitis NASH from long-standing obesity, many patients who have been obese will spontaneously lose 50 or even 100 pounds after their liver disease decompensate.

★ Alcohol intake

★ history of heart failure may raise the possibility of cardiac ascites.

★ Tuberculous peritonitis is usually manifested by fever and abdominal pain, > 50% have underlying alcoholic cirrhosis.

★ Acute hemorrhagic pancreatitis or hemodialysis.

★ Fitz-Hugh-Curtis syndrome caused by Chlamydia may cause inflammatory ascites in a sexually active woman.

★ Pts with ascites and anasarca in the setting of DM suggest nephrotic ascites.

✚ PHYSICAL EXAMINATION

Patient with Ascites & jaundice >> you will think of chronic liver disease !

- ✔ Signs of chronic liver disease
- ✔ Signs of ascites (bulging abdomen,fank dullness, shiffting dullnes and fluid wave).
- ✔ Large veins on the suggests IVC blockage, an immobile mass in the umbilicus (the Sister Mary Joseph nodule) is suggestive of peritoneal carcinomatosis.
- ✔ Nephrotic syndrome or cardiac failure may have total body edema (anasarca).

A YOU SHOULD DIFFERENTIATE BETWEEN PORTAL HYPERTENSION & NON PORTAL HYPERTENSION BY MEASURING THE SAAG

Serum-Ascites Albumin Gradient (SAAG)	
High Gradient (≥ 1.1 g/dl)	**Low Gradient (< 1.1 g/dl)**
Cirrhosis	Peritoneal carcinomatosis
Alcoholic hepatitis	Peritoneal tuberculosis
Cardiac failure	Pancreatic ascites
Massive liver metastases	Biliary ascites
Fulminant hepatic failure	Nephrotic syndrome
Budd-Chiari syndrome	Serositis
Portal-vein thrombosis	Bowel obstruction or infarction
Veno-occlusive disease	
Fatty liver of pregnancy	
Myxedema	
"Mixed" ascites	

Indicates portal hypertension and suggests a **nonperitoneal** cause

Indicates nonportal hypertension and suggests a **peritoneal** cause

The serum ascites albumin gradient (SAAG) is a formula used to assist in determining the etiology of ascites.

The formula is below.
SAAG = serum albumin − ascites albumin

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✚ MANAGEMENT

WHAT SHOULD BE YOUR INITIAL STEP IN MANAGING ASCITES ? Salt Restriction Diet.

Has been shown in studies that 10% of the patient will achieve adequate diuresis just by the slat restricted diet.

But even if they didn't achieve diuresis by slat restricted diet and u add diuretics , diuretics will not be very affective unless they restrict their salt intake !

Management of Ascites Due to Cirrhosis
1. Treatment of underlying disorder (e.g. alcoholic liver disease, hepatitis B, autoimmune hepatitis)
2. Dietary sodium restriction (less than 2000mg per day)
3. Diuretic therapy (maintain ratio spironolactone 100mg: furosemide 40mg) Lasix
4. Therapeutic paracentesis
5. Fluid restriction only if serum sodium <120 mEq/L or symptomatic hyponatremia

Method used to decompress the portal pressure

The create a channel between the hepatic vein and the portal vein

Then the pressure inside the liver will drop

THE ULTMATE TREATMENT IS LIVER TRANSPLANTATION IF YOU WEREN'T ABLE TO TREAT THE UNDERLYING LIVER DISEASE !

MORE PAGE !! U CAN DO IT (ง>ᴗ<)ง

Conclusion

The most common cause of ascites is liver cirrhosis and the pathophysiological mechanism is portal HTN leading to systemic vascular changes.

Other pathogenesis include:

1-Increased hydrostatic pressure

2-Decreased colloid osmotic pressure

3-Increase in the permeability of peritoneal capillaries

4-Leakage of fluid into the peritoneal cavity

5-miscellaneous

- THE END -

✚ MCQs

Q1/which of the following is not a cause of ascites?

A. Increase in hydrostatic pressure.

B. Increase in oncotic pressure.

C. Infection like TB.

D. Chorionic pancreatitis.

Q2/Budd-Chiari syndrome is an example of ?

A. Prehepatic

B. Intrahepatic

C. Posthepatic

D. All the above

Q3/ the most common cause of ascites is:

A. Cirrhosis

B. TB infection

C. CHF

D. Cancer

Q4/ Low serum ascites albumin gradient (SAAG) related to which of the following ?

A. Cardiac Failure

B. Nephrotic syndrome

C. Alcoholic Hepatitis

D. Veno-Occlusive disease

✚ SAQs

Why patient with low systemic blood pressure and portal hypertension will have hyperdynamic circulation?

Because the increase in the plasma volume in vasodilated vessels which will increase the cardiac contractility!

What do you think patients with HVPG of 11mmHg will present with ?

Ascites and Esophageal varices

What is the pathophysiology of ascites formation in patient with TB ?

Increased permeability of peritoneal capillaries

MCQs answers

1:B 2:B 3:A 4:B

ALL THE BEST

THANKS FOR CHECKING OUR WORK

Special thanks for our team members:

Lamya Alsaghan Monerah Alsalouli

Dalal Alhezaimy

Kowthr Almosa Moruoj Alharbi

Team leaders:

Abdulrahman A Alshehri Wadha Alotaibi

For any suggestion or correction pleas contact us : 435medicin@gmail.com