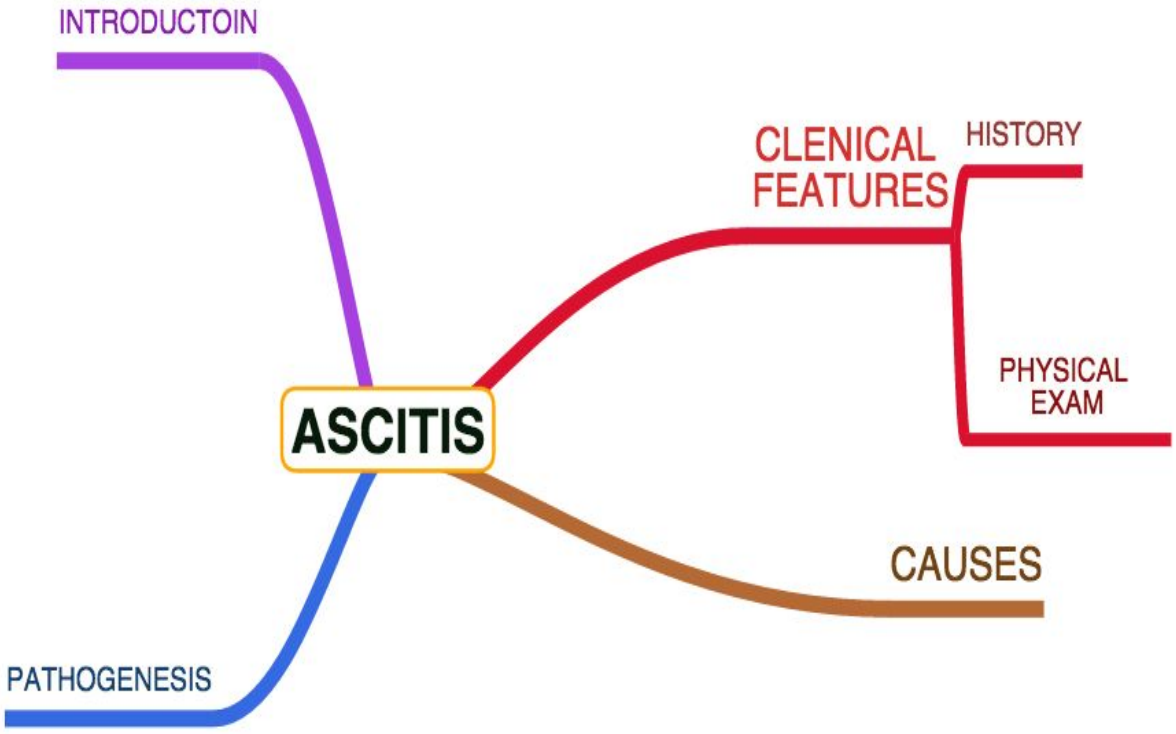
Pathophysiology of Ascites

Red = must know

Grey= extra information

Mind Map


INTRODUCTOIN
CLENICAL FEATURES
HISTORY
PHYSICAL EXAM
ASCITIS
CAUSES
PATHOGENESIS

Introduction:

The peritoneal cavity:

- ➔ Derived from the coelomic cavity of the embryo
- ➔ Normally the peritoneal cavity contains about **50** ml and may rise to **2000** ml in severe ascites
- ➔ The peritoneal cavity functions in facilitating **bowel movements** and it is normally sterile (no bacteria)

The peritoneal fluid:

- ➔ It is a normal, lubricating fluid found in the peritoneal cavity.
- ➔ The fluid is mostly water with **electrolytes, antibodies, white blood cells, albumin, glucose** and other biochemicals. (one of the life threatening complication of ascites is infection. how do we diagnose it? we aspirate and look at the WBCs.)

Ascites: the accumulation of fluid in the peritoneal cavity, causing abdominal swelling.

(Most patients (85%) with ascites have **cirrhosis)**

❑ Causes of ascites:

Cirrhosis, Infection (TB), Malignancy, CHF, Nephrotic syndrome, Pancreatic or biliary ascites.

- ➔ The most common causes of cirrhosis are **chronic alcoholic liver disease & viral hepatitis.**(fatty liver disease will become the #1 cause of liver cirrhosis)

❑ Pathogenesis of ascites:

Ascites can be caused by: Increased hydrostatic pressure, Decreased colloid osmotic pressure, Increase in the permeability of peritoneal capillaries, Leakage of fluid into the peritoneal cavity, Miscellaneous.

- ❖ **There are two forces that dictate the movement of solutes across the capillary endothelium:**
  1)Increased Hydrostatic Pressure: increased in cases of **congestive heart failure.**
  2)Decreased colloid osmotic pressure: mainly controlled by albumin.
    - **Decreased** albumin decreases colloid osmotic pressure and leads to edema.
    - **Increased** albumin excretion in nephrotic syndrome.

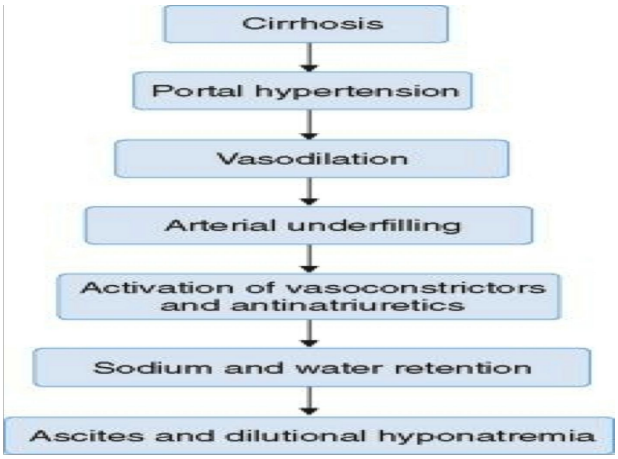
Cirrhotic Ascites (Pathophysiology and Causes):

The most recent theory of ascites formation, the "**peripheral arterial vasodilation hypothesis**,". This happens as a consequence of portal hypertension.

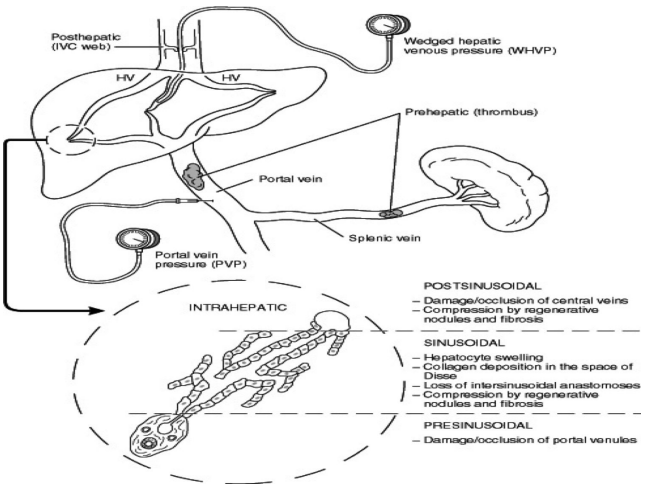
- ➔ Hepatic blood flow is normally about 1500 mL/minute.(70% portal vein, 30%hepatic artery)
- ➔ Normal, uncorrected pressure in the portal vein ranges from **5 to 10 mm Hg**. Gradient of 2-6.
- ➔ Portal HPN present when **gradient > 12 mmHg.**
- ➔ The high-pressure, well-oxygenated hepatic arterial blood mixes completely with the low-pressure, low-oxygen-containing, nutrient-rich portal venous blood within the **hepatic sinusoids.**
- ➔ **The sinusoids are normally protected from upstream portal perfusion pressure and fluctuations:**

- they are lined by an **endothelium** contains a multitude of large (50 to 200 nm), highly permeable fenestrae.

- *hepatic arterial buffer response* "**an adenosine-mediated vascular reflex**."



- ➔ After perfusing the sinusoids, blood flows into the hepatic venules, hepatic veins, and inferior vena cava.
- ➔ Normal hepatic sinusoidal microcirculation has low perfusion pressure which is attributed to the unusually high precapillary to postcapillary resistance in the liver.
- ➔ 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 portal collateral resistance) that form during the evolution of portal hypertension.



****Extra Information:**

For example:

• In both pre hepatic and intrahepatic presinusoidal portal hypertension
(PVP) is elevated with N (WHVP) and (HVPG).

• In sinusoidal and intrahepatic postsinusoidal portal hypertension, the (WHVP) tends to approximate or equal the directly measured (PVP) and the HVPG is increased.
•In posthepatic portal hypertension, the WHVP equals the increased PVP.

❑ The causes of portal hypertension (The Role of Increased Resistance) are classified as:

1)Prehepatic: thrombosis of the portal vein or splenic vein.

2)Intrahepatic:

A)Presinusoidal: occlusion of hepatic venules.

B) Intrasinusoidal: collagen deposition in the space of Disse or compression by nodules or fibrosis.

C) Postsinusoidal: compression or occlusion by fibrosis

B&C Explain why cirrhosis causes **portal hypertension.**

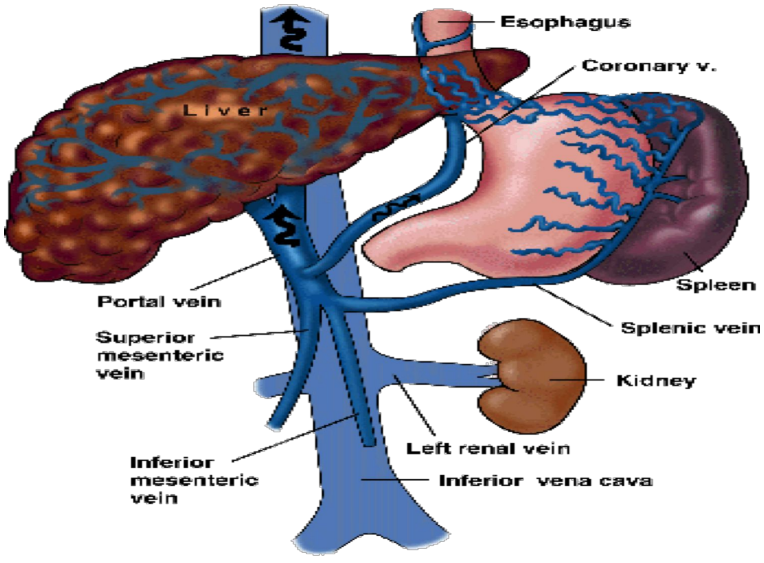
3)Posthepatic: thrombosis of the hepatic vein.

Called Budd-Chiari syndrome

❑ Portal Blood Flow :

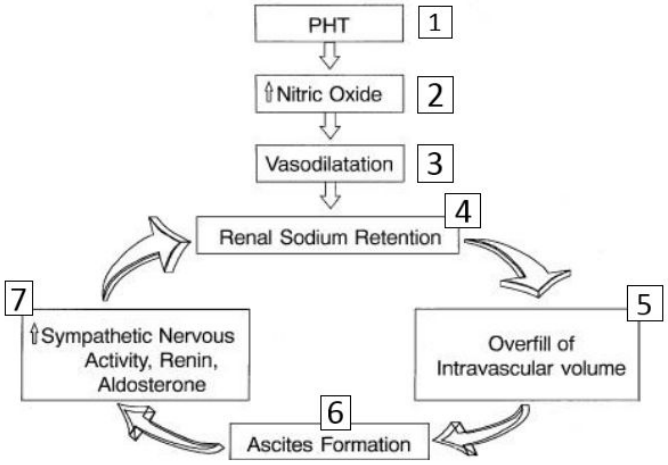
➔ Primary High Portal Flow States Although uncommon, conditions leading to **high-flow states in the portal system** (arterioportal fistulas, splenomegaly resulting from myelofibrosis or myeloid metaplasia) are well-recognized causes of portal hypertension.

➔ 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.



➔ **Hyperdynamic Circulation of Portal Hypertension i**ts hallmarks are **increased cardiac output and reduced arterial blood pressure.**

❑ The peripheral arterial vasodilation hypothesis of ascites formation/ pathogenesis of ascites formation: How does portal hypertension cause ascites?



Note: this picture is important.

The following text aims to describe the steps. The numbers correspond with the text

1) Portal hypertension

2)Nitric oxide (NO) release:

Nitric oxide is released secondarily to endothelial injury

The high pressure in the portal circulation injures endothelial cells ⟶ nitric oxide release.

Nitric oxide (NO) is a vasodilator.

3)NO results in vasodilation of systemic vasculature

4) RAAS increases Na+ reabsorption in the kidney through aldosterone

Na+ reabsorption leads to increased intravascular volume

Increased intravascular volume⟶ increases hydrostatic pressure

- This favours the movement of fluids from the intravascular compartment to the extravascular compartment

5) Fluid starts moving from the intravascular compartment to the extravascular compartment

Due to increased hydrostatic pressure

Because fluid is moving out of the blood vessels, there is a drop in blood pressure

This drop in blood pressure results in a decreased renal perfusion

- The RAAS is activated as mentioned in (7)

6)Ascites formation.

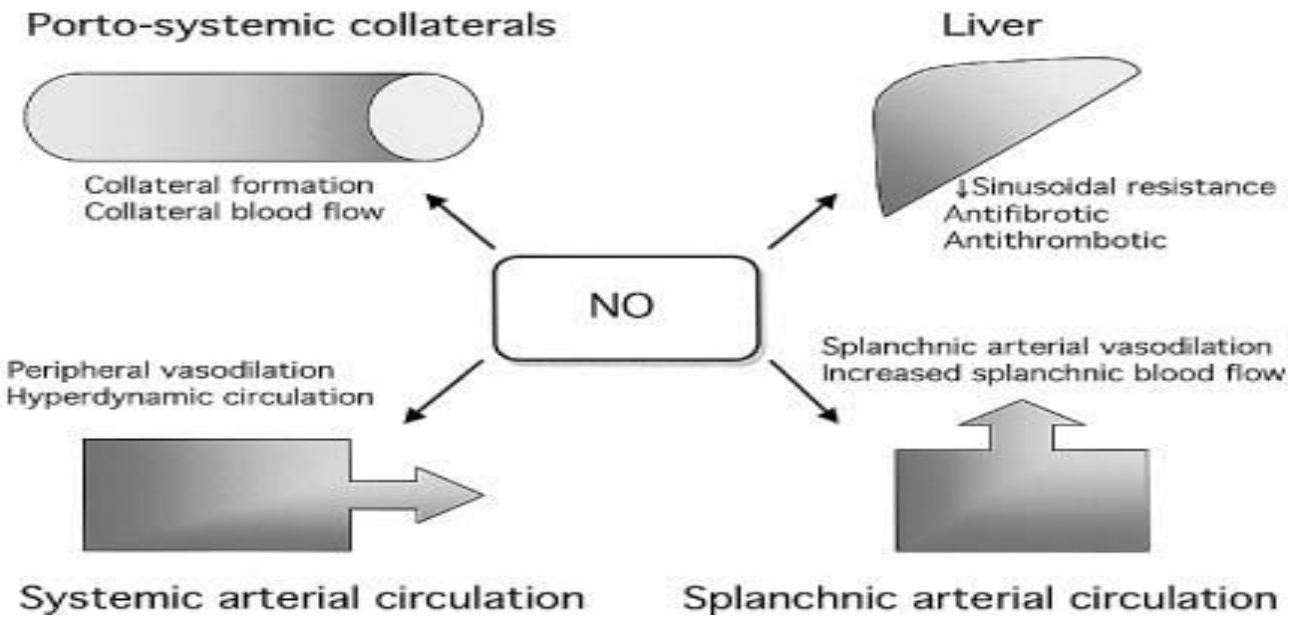
7) Decreased intravascular volume decreases renal perfusion and leads to the activation of RAAS

This leads to Na+ reabsorption in the kidneys

This takes us back to number (4)

The cycle continues

❑ The effects of NO:



A) This vasodilation allows for collateral blood flow (portosystemic anastomosis- see later).

B) Decreases resistance in hepatic sinusoids.

C) Vasodilates splanchnic arterial circulation increasing splanchnic blood flow.

D) **Systemic arterial circulation**.

- Causes **hyperdynamic** flow:
  - This means that blood moves quickly through the capillaries and goes back to the heart quickly.
- **Vasodilation** of systemic arterial circulation **decreases** the **BP**:
  - The RAAS acts to restore BP.
  - The baroreceptors are activated and there is a reflex tachycardia in an attempt to restore BP.
- The kidneys and brain have special mechanisms to control their blood flow. They are not directly affected by NO.

❑ Portal HTN results in porto systemic anastomoses:

1) **Esophagus**: esophageal varices
   May bleed⟶ hematemesis

2)**Anus** ⟶ hemorrhoids

3) **Umbilical veins** ⟶ caput medusa

Non portal hypertensive ascites:

- **Malignancy related**(depends on the location of the tumor)
  - Peritoneal carcinomas produce proteinaceous fluids
  - Proteins suck water into the peritoneum by osmosis
- **Heart failure**
  Increased hydrostatic pressure
- **Nephrotic syndrome**
  Increased albumin excretion
- I**nfections (TB, Chlamydia)**
  Produce inflammation⟶ increased vascular permeability
- **Pancreatic or biliary ascites**
  Leakage of fluid into the peritoneum

Clinical features:

History:

Pt. may present with jaundice or encephalopathy
Ask about risk factors of viral hepatitis or chronic alcohol abuse
History of heart failure

Physical examination:

Signs of chronic liver disease: gynecomastia, spider angioma, etc.

- Non-alcoholic-steatohepatitis (NASH) is caused by long standing obesity.
- Malignancy related ascites is painful, whereas cirrhotic causes are not.
- Patients with a long history of stable cirrhosis & suddenly develop ascites ⟶ hepatocellular carcinoma.

Remember that:

- **Blood pressure (BP)= cardiac output (CO) x total peripheral resistance (TPR)**
  Vasodilation⟶ decreases TPR ⟶ decreases BP
- **There are two main compartments in our bodies**
  Intravascular compartment
  Extravascular compartment⟶ divided into interstitial and vascular compartments
  - Interstitial compartment lies between cells
  - Vascular compartment is fluids inside the blood vessels
    - Fluids inside blood vessels are contribute to blood pressure
- **The Renin-Angiotensin-Aldosterone system is activated whenever there is decreased renal blood flow**
  - Renal blood flow is decreased when there is a drop in blood pressure
  - RAAS increases aldosterone which reabsorbs sodium (Na+)
    - Water follows Na+
    - RAAS⟶increased aldosterone ⟶ increased sodium retention⟶ increased fluids inside the intravascular compartment

MCQs

1-Which one of the following is the major cause of ascites ?

A. Liver cirrhosis
B. TB infection
C. Malignancy

2-Which one of the following is a symptom of ascites ?

A. Swelling of the abdomen
B. Umbilical hernia
C. A and B

3- What happen in ascites ?

A. Decreased hydrostatic pressure
B. Increased hydrostatic pressure
C. Increased colloid osmotic pressure

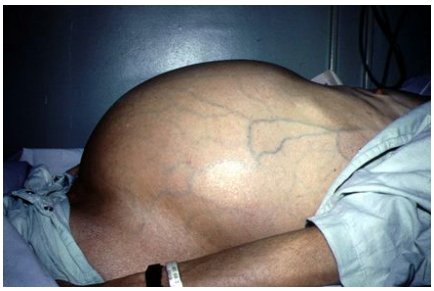
4- Malignancy related ascites depends on the location of the tumor ?

A. True
B. False

1)A 2)C 3)B 4)A

SAQs

Sami is a 40 year old American man, he came to Saudi Arabia for a job opportunity. Meanwhile in his stay he developed nausea and sometimes vomiting. He went to the doctor and Sami admitted that he is a heavy drinker and on clinical examination the following is shown; abdominal swelling and changes to the belly button.



What is the clinical diagnosis?

Ascites

What is the most likely underlying disease?

Liver cirrhosis due to alcoholism

What's the best management plan?

Liver transplant

Mention the two forces that dictate the movement of solutes across the capillary endothelium?

Hydrostatic pressure and osmotic colloidal pressure

How does portal hypertension causes ascites?

- NO results in vasodilation of systemic vasculature
- RAAS increases Na reabsorption in the kidney through aldosterone
- Fluid starts moving from the intravascular compartment to the extravascular

Done By:

➔AbdulRahman AlArfaj
➔Mohammad Kharraz
➔Ghazi AlDlbhi
➔Saleh AlBanyan
➔Rawa AlOhali
➔Nora Helali
➔Lamyaa Althawadi

Never Forget to:

Contact us at:

434medicine@gmail.com