MED438
KING SAUD UNIVERSITY

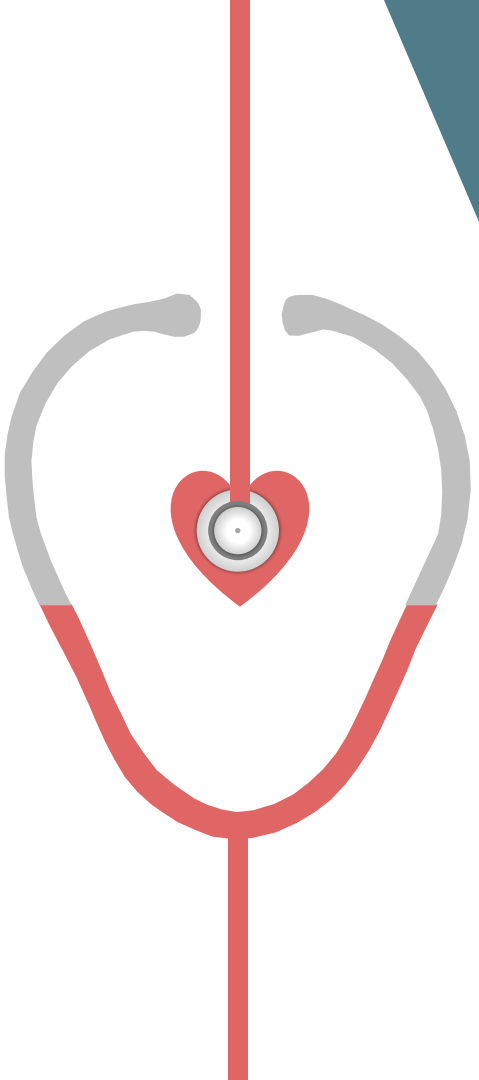
Family & Community Medicine

Reviewed by Razan AlRabah & Ibrahim AlDakhil

Pathophysiology Of Ascites

Editing file

- Important
- Original content
- Only in girls slides
- Only in boys slides
- Doctor's notes



OBJECTIVES:

01 To understand basic pathophysiologic steps in the development of ascites secondary to cirrhosis.

02 To correlate the anatomic and pathophysiologic changes with clinical manifestations.

03 To understand the basic steps in evaluation of patients with ascites.

Ascites The pathologic accumulation of fluid in the peritoneal cavity & it's the most common complication of cirrhosis and indicates advanced stage of liver disease and poorer prognosis.

Causes if Ascites: cirrhosis 85%, others 15% (peritoneal malignancy, heart failure, & Peritoneal tuberculosis).
Any infection

Ascites & The development of Portal hypertension

1. The development of portal hypertension (PHT) is the **first step** toward fluid retention in the setting of cirrhosis.
2. Patients with cirrhosis but without PHT do not develop ascites or edema.
3. A portal pressure >12 mmHg appears to be required for fluid retention.
4. 2 main mechanisms involved in portal hypertension:
   A. **Mechanical** (due to structural changes in the liver with fibrosis and regenerative nodules).
   B. **Hemodynamic** (circulatory, vascular, functional, and biochemical abnormalities).

Cirrhosis Late stage of chronic liver inflammation and fibrosis, in which liver parenchyma is distorted and replaced by fibrous tissue and regenerating nodules.

IMPORTANT!

Portal hypertension & Vasodilation (VD)

1. Portal hypertension leads to VD.
2. VD initially develops in the arterial splanchnic circulation (i.e., the mesenteric arteries).
3. Subsequently, vasodilation develops in the arterial systemic circulation.

Mechanism of Vasodilation (VD)

1. Increase production of nitric oxide (NO), which is the **primary** mediator of VD in cirrhosis (for splanchnic and peripheral vasodilation).

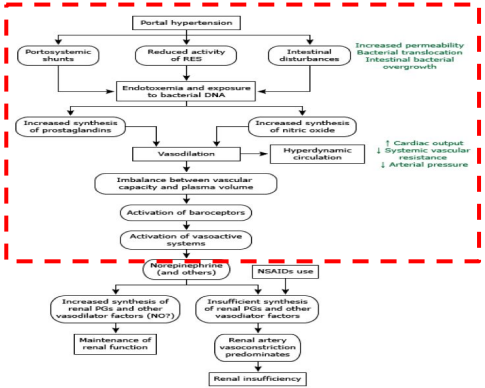
2. Increased levels of other circulating VDs. (Glucagon , vasoactive intestinal peptide (VIP) & prostacyclin.

production of these VDs may be stimulated by endotoxins or other bacterial products.

IMPORTANT!

Consequences of Vasodilation (VD)

1. Activation of endogenous vasoconstrictors.
2. Sodium and water retention.
3. Increase renal vasoconstriction.



Activation of endogenous vasoconstrictor agents

Vasodilation → The reduction in pressure (or stretch) at the carotid and renal baroreceptors → activation of the sodium-retaining neurohumoral mechanisms (in an attempt to restore perfusion pressure to normal) by:

- Renin-angiotensin-aldosterone system (RAS).
- Sympathetic nervous system (SNS).
- Antidiuretic hormone - vasopressin (ADH).

(The net effect is <u>avid</u> sodium and water retention and increase plasma volume).

All these with hypoalbuminemia and increase vascular permeability lead to fluid extravasation.

IMPORTANT!

The systemic impairment in patients with ascites

1. The normal regulation of sodium balance is lost (Impaired sodium excretion).
2. Water retention: Initially water excretion is normal in patients with cirrhosis before the development of ascites and then becomes increasingly impaired as the liver disease progresses (Increase ADH).

Thus, patients with cirrhosis and ascites usually demonstrate **urinary sodium retention, increased total body sodium, and dilutional hyponatremia.**

Renal Vasoconstriction

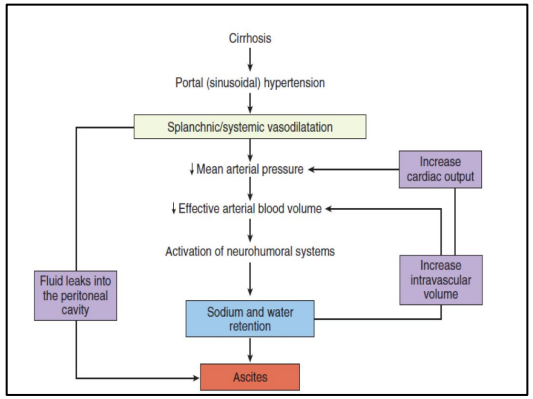
Renal vasoconstriction: VC → Renal hypoperfusion → decrease GFR.

Renal perfusion may initially be maintained due to **vasodilators** such as prostaglandins and perhaps nitric oxide (local).

However, progression renal hypoperfusion can lead to gradual decline in the glomerular filtration rate and, in some patients, the hepatorenal syndrome.

This excess retained blood volume is thought to leak-out (filtered) (extravasate) directly from both the **liver surface**, and the **mesenteric vessels**.

Due to:
– Increased hydrostatics pressure.
– Increase vascular wall permeability.
– Concurrently decreased oncotic (osmotic) pressure (hypoalbuminemia).



Evaluation of Patient with Ascites

1. **History:**

– symptoms of chronic liver disease, abdominal distention.

2. **Examination:**

– Flank fullness, shifting dullness or fluid thrill.

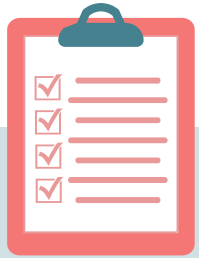
3. **Imaging:**

– Ultrasound.

4. **Ascitic Fluid Analysis.**

Any new ascites should be tapped and analyzed

Quiz!



1 What's the MAIN cause for ascites?

A) Liver cirrhosis.
B) Hepatitis.
C) Heart failure.
D) Viral hepatitis.

2 The FIRST step toward fluid retention in the setting of cirrhosis is:

A) Vasodilation.
B) Activation of endogenous vasoconstrictor agents.
C) Portal hypertension.
D) Hyperdynamic circulation.

3 The PRIMARY mediator of vasodilation in cirrhosis is:

A) Vasoactive intestinal peptide (VIP).
B) Nitric oxide (NO).
C) Prostaglandin.
D) ADH.

4 Which one of the following is a SYSTEMIC consequences to vasodilatation?

A) Hyperdynamic circulation.
B) Vomiting.
C) Headache.
D) Sodium & Water retention.

5 Which of the following is a consequence of vasodilatation?

A) Activation of endogenous vasoconstrictor agents.
B) Sodium & Water retention.
C) Increase renal vasoconstriction.
D) All of the above.

6 Which one of the following is a FUNCTIONAL change in ascites?

A) Increase systemic nitric oxide (NO).
B) Increase systemic prostaglandin.
C) Reduced glomerular filtration rate.
D) Sodium & Water retention.

Answers: 1) A, 2) C, 3) B, 4) A, 5)D, 6) C

THIS WORK IS DONE BY:

Abdulrahman Bedaiwi & Jude Al-Otaibi

Give us your feedback!