ASCITES

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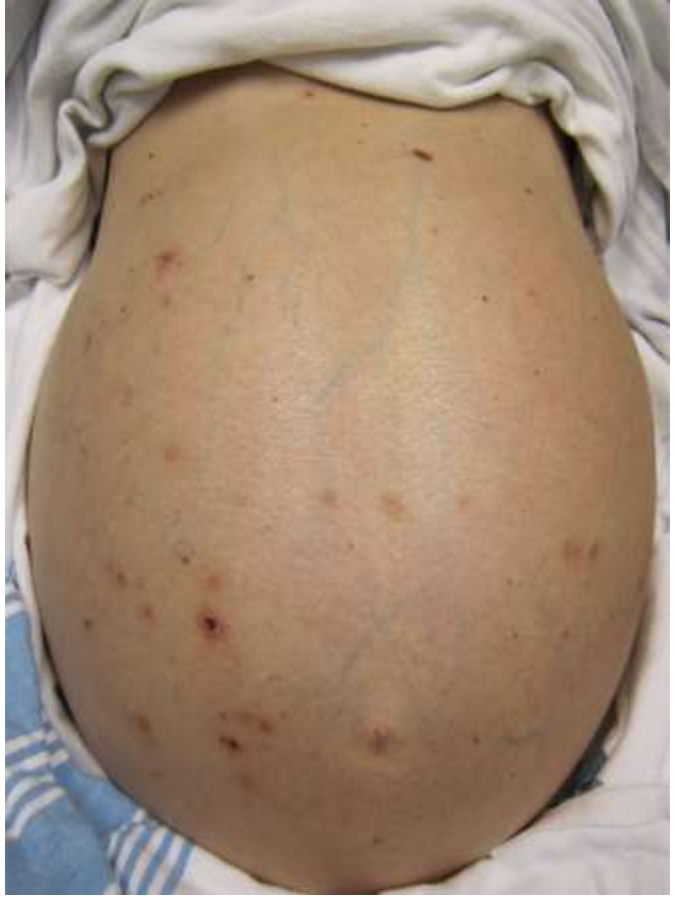
Definition

- **Ascites** (also called hydro-peritoneum) is a pathological accumulation of fluid in the peritoneal cavity.
- Based on the severity of fluid accumulation, ascites can be categorized as mild, moderate, and large.

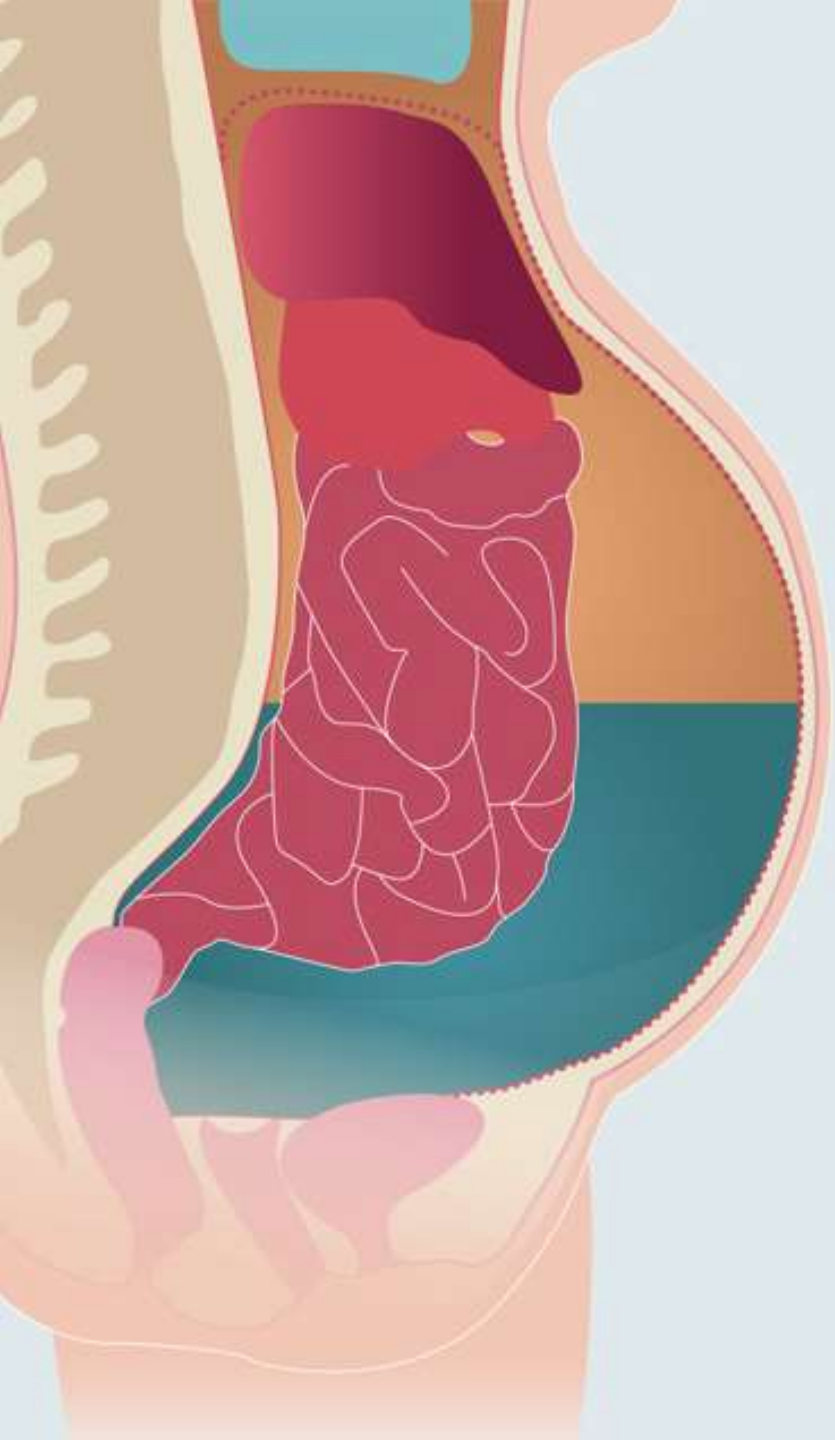
The fluid could be:

- Transudate fluid is due to high pressure in hepatic portal vein greater than 8mmhg like in cirrhosis.
- Exudate fluid is usually due to inflammation and malignancy .



- Healthy males may have little or no peritoneal fluid while females may normally have about 20 ml depending the phase of their menstrual cycle .

Ascites is considered when the fluid exceeds 20-25 ml.



- There are two different types of ascites:
  - ❑ uncomplicated ascites
  - ❑ refractory ascites

Uncomplicated ascites

is the most common type and responds well to treatment.

Refractory ascites

is less common and very difficult to treat,

leading to a high mortality rate. refractory ascites can be associated with kidney failure.

Causes

- Cirrhosis accounts for 84% of cases of ascites.
- Cardiac ascites (CHF), peritoneal carcinomatosis, and “mixed” ascites resulting from cirrhosis and a second disease account for 10–15% of cases.
- Less common causes of ascites include:

massive hepatic metastasis

infection (tuberculosis, Chlamydia infection)

Pancreatitis

Renal disease (nephrotic syndrome).

- Rare causes of ascites include:

hypothyroidism (myxedema) and Familial Mediterranean fever.

Pathogenesis

❖ *Factors involved in Pathogenesis:*

- ***•Portal hypertension***
- ***•Sodium and water retention***
- ***•Low serum albumin***
- ***•Lymphatic obstruction***

Leakage of lymphatic fluid to the peritoneal cavity (chylous ascites).

- ***•peritoneal disease (inflammation and infection):***

Secondary infections such as tuberculosis or fungal infection.

Secondary malignant infiltration (carcinomatosis peritonei).

Pathogenesis

- ❖ PRESENCE OF CIRRHOSIS
  - ■ 1) Portal hypertension

It signifies elevation of the pressure within the portal vein.

How?

-According to Ohm's law, pressure is

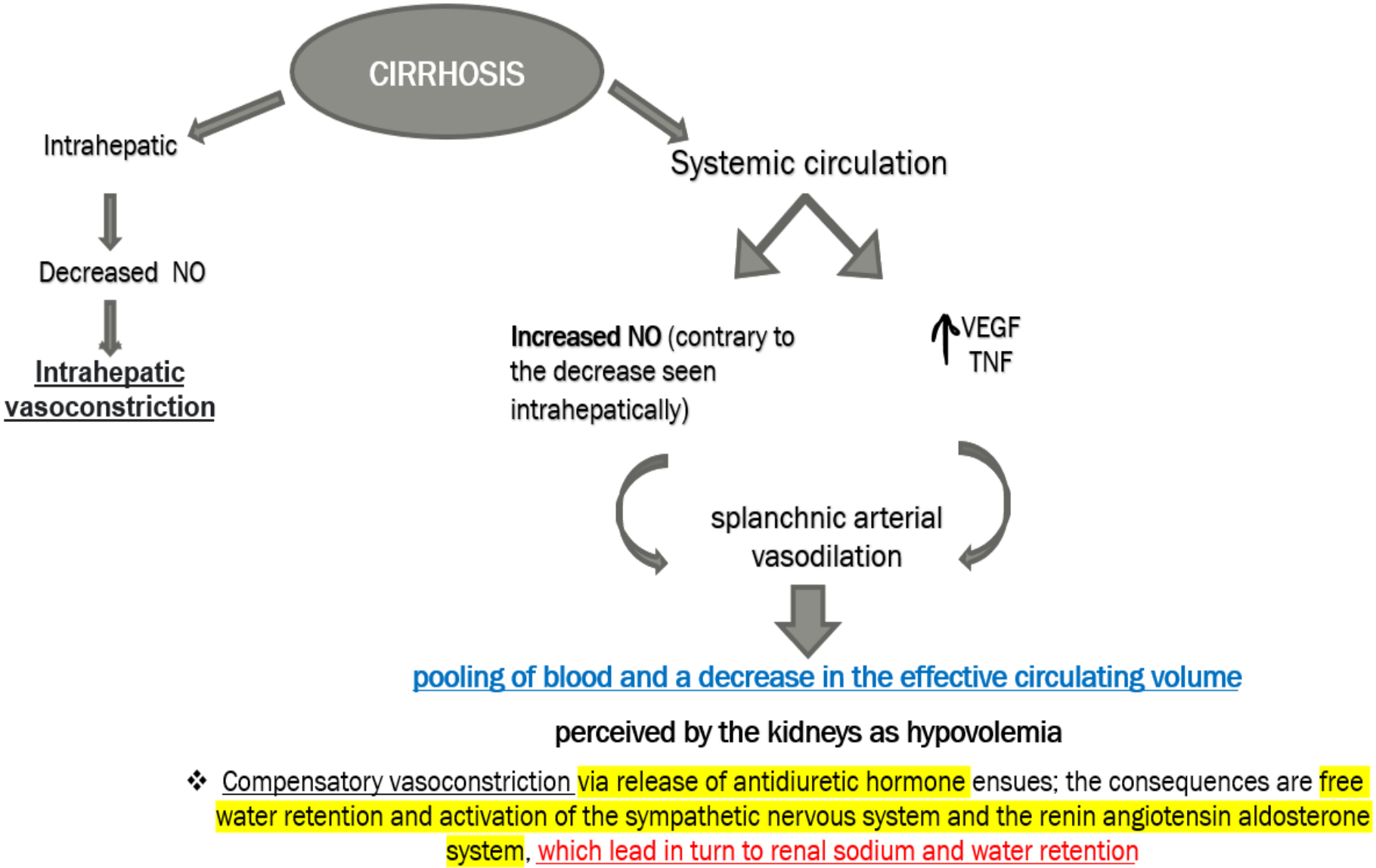
the product of resistance and flow.

Increased hepatic resistance occurs by several mechanisms:

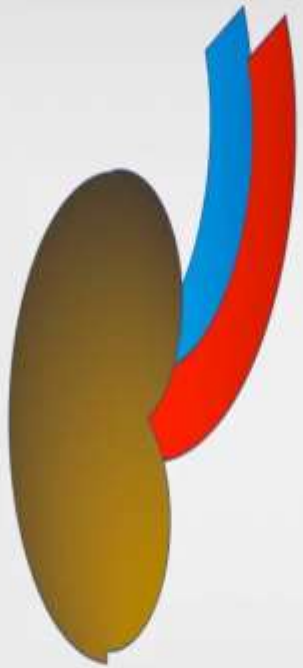
- **Hepatic Fibrosis**
- **Activation of Stellate cells** ⟹ this will mediate fibrogenesis, leads to smooth-muscle contraction and fibrosis.
- ⬇ **eNOS** (endothelial nitric oxide synthetase)

which results in decreased nitric oxide production and increased intrahepatic vasoconstriction.

- 2)Renal salt and water retention



❖ Compensatory vasoconstriction via release of antidiuretic hormone ensues; the consequences are free water retention and activation of the sympathetic nervous system and the renin angiotensin aldosterone system, which lead in turn to renal sodium and water retention



The kidney interprets this vasodilatation as hypovolemia

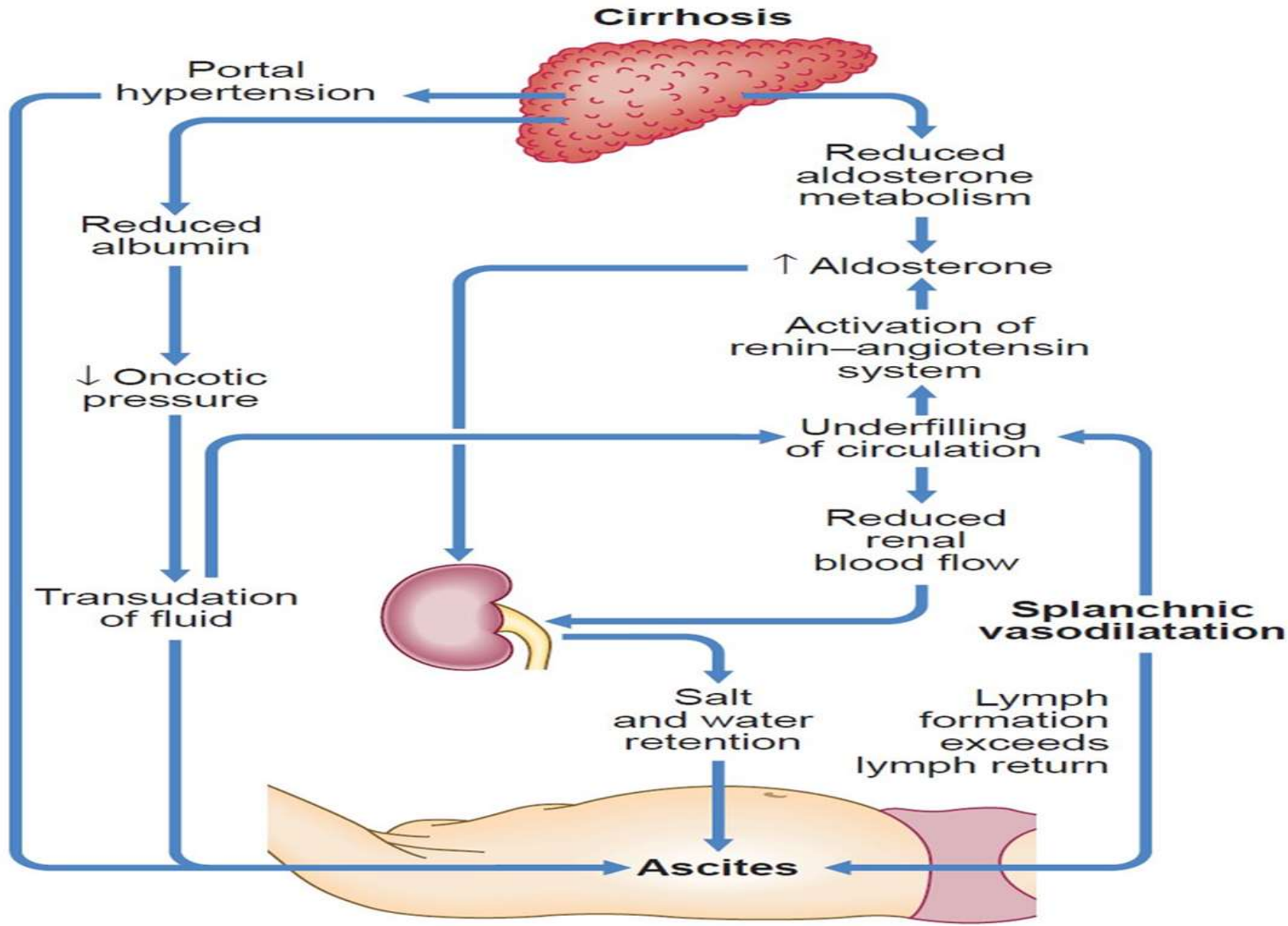
Activation of renin-angiotensin system and increased ADH

Retention of sodium and water

LOW SERUM ALBUMIN

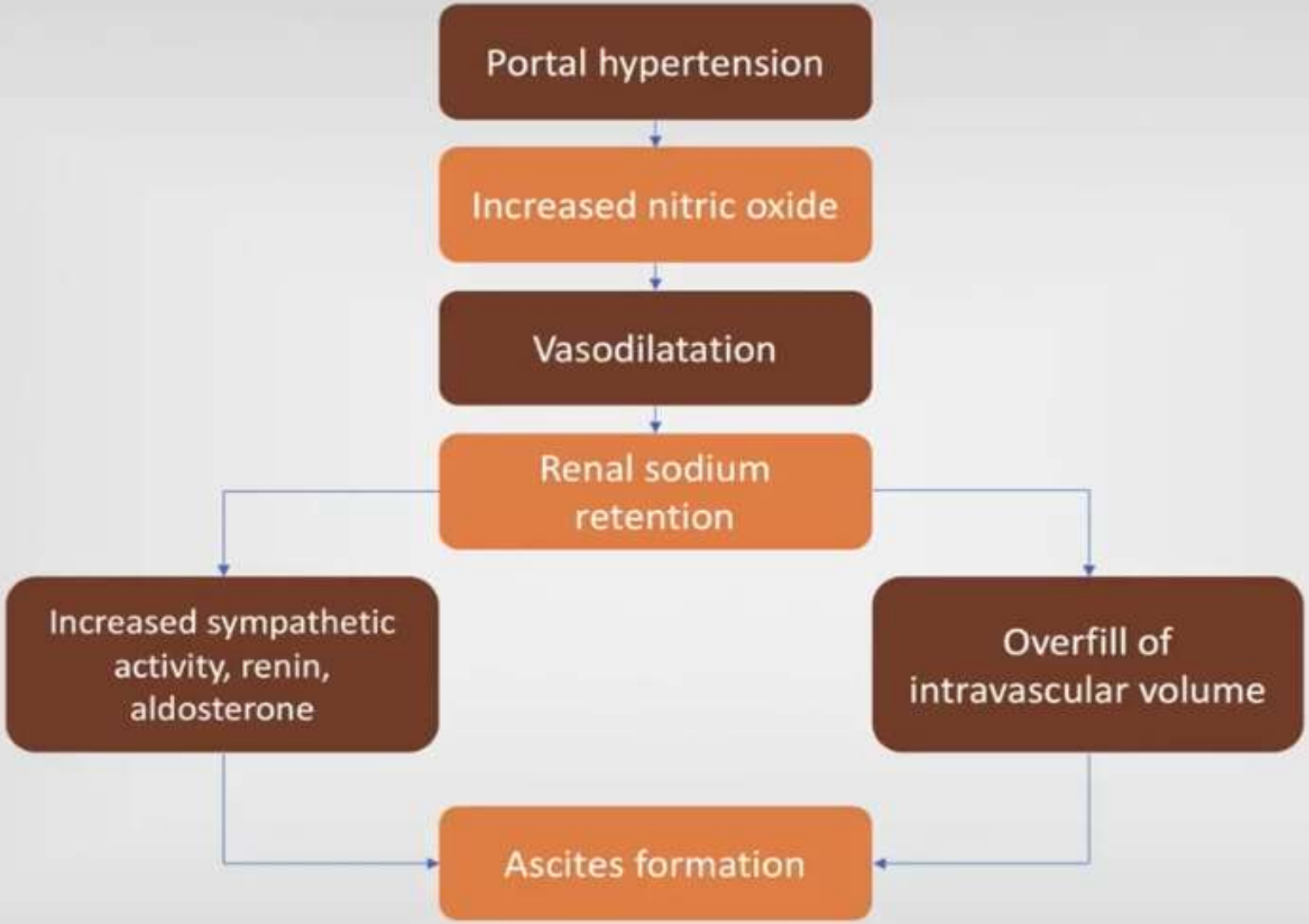
- **Low serum albumin (a consequence of poor synthetic liver function)**
- • It also may happen in malnutrition.
- •Kidney damage (proteinuria).

• Low serum albumin leads to reduction in plasma oncotic pressure causing edema and ascites.



Pathogenesis of ascites.

Source : Davidsons Essentials of Medicine, 2e


Portal hypertension
Increased nitric oxide
Vasodilatation
Renal sodium retention
Increased sympathetic activity, renin, aldosterone
Overfill of intravascular volume
Ascites formation

❖ PATHOGENESIS IN THE ABSENCE OF CIRRHOSIS

- ***Ascites in the absence of cirrhosis generally results from:***

➢ peritoneal carcinomatosis

The tumor cells lining the peritoneum produce a protein-rich fluid that contributes to the development of ascites. Fluid from the extracellular space is drawn into the peritoneum, further contributing to the development of ascites.

➢ peritoneal infection

Tuberculous peritonitis causes ascites via a similar mechanism; tubercles deposited

on the peritoneum exude a proteinaceous fluid.

➢ pancreatic disease

Pancreatic ascites results from leakage of pancreatic enzymes into the peritoneum.

•*Lymphatic obstruction*

- Chylous **ascites** (CA) is a rare form of **ascites** that results from the leakage of lipid-rich **lymph** into the peritoneal cavity. This usually occurs due to trauma and rupture of the **lymphatics** or increased peritoneal **lymphatic** pressure secondary to **obstruction**.

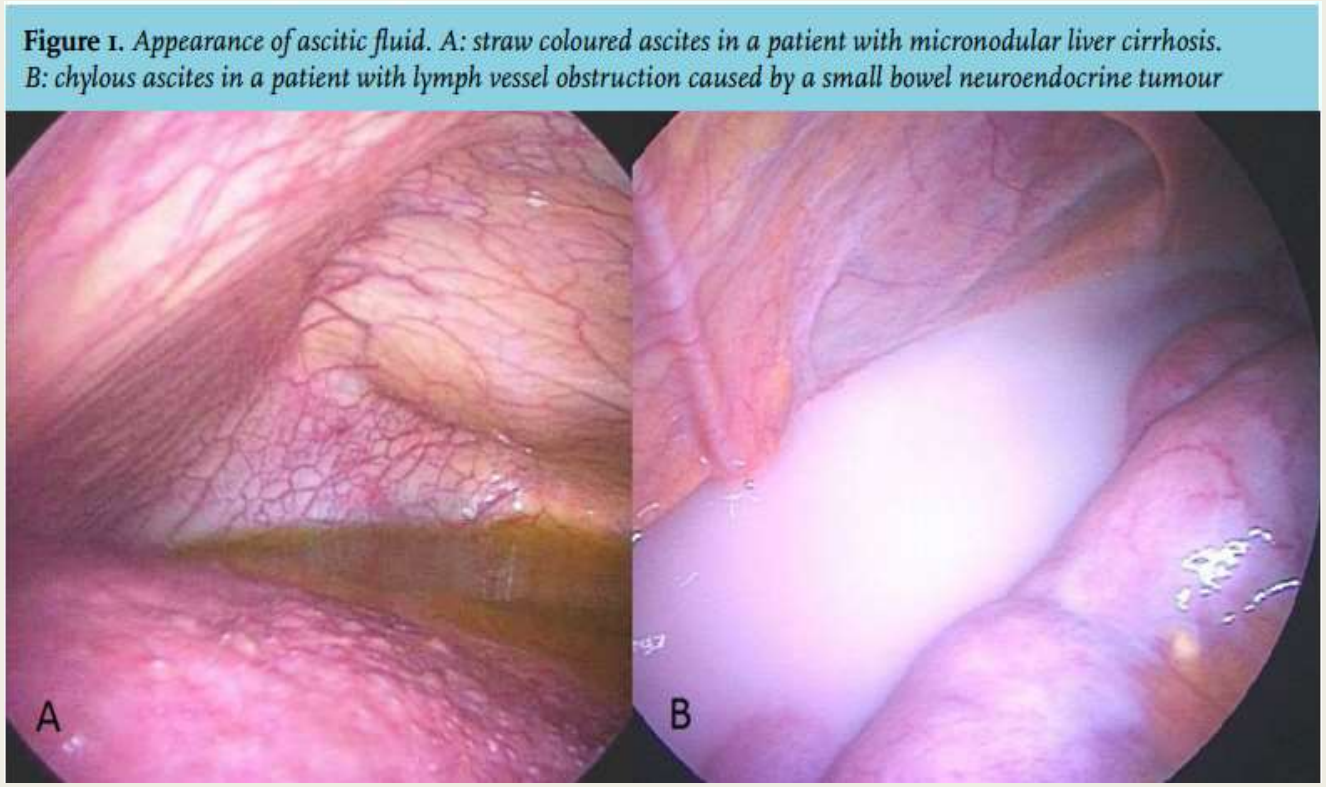


Figure 1. *Appearance of ascitic fluid. A: straw coloured ascites in a patient with micronodular liver cirrhosis. B: chylous ascites in a patient with lymph vessel obstruction caused by a small bowel neuroendocrine tumour*

CLINICAL FEATURES

- According to the volume of fluid.
- There may be no symptoms associated with ascites especially if it is mild (usually less than about 100 – 400 ml in adults).
- As more fluid accumulates, increased abdominal girth and size are commonly seen (> 1 L)

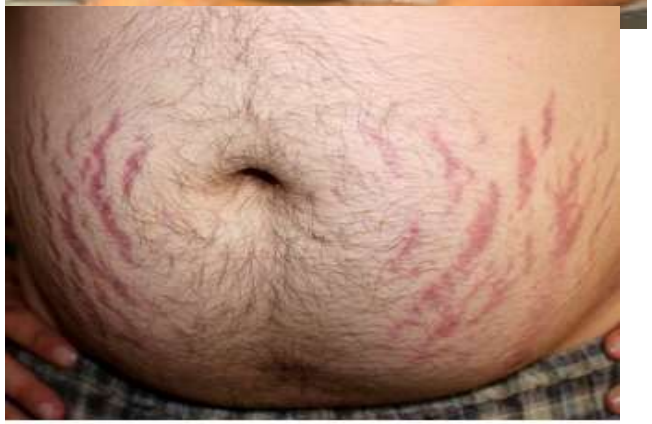
Signs & symptoms :

1. **Abdominal pain** , **discomfort** , and **bloating** are also frequently seen as ascites becomes larger

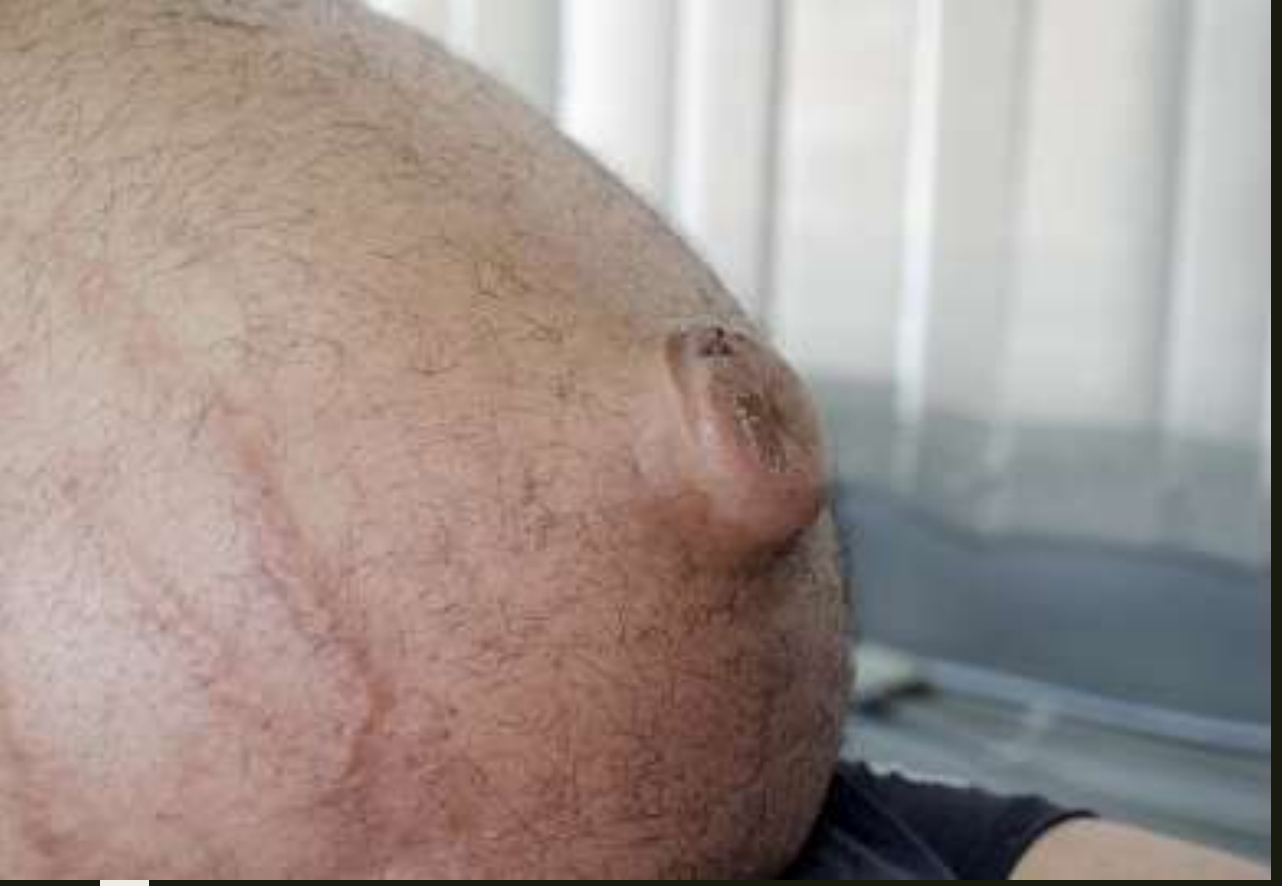
2. **Fullness of the flanks** and **dullness on percussion.**
3. **Leg swelling** : (liver cirrhosis and heart failure)

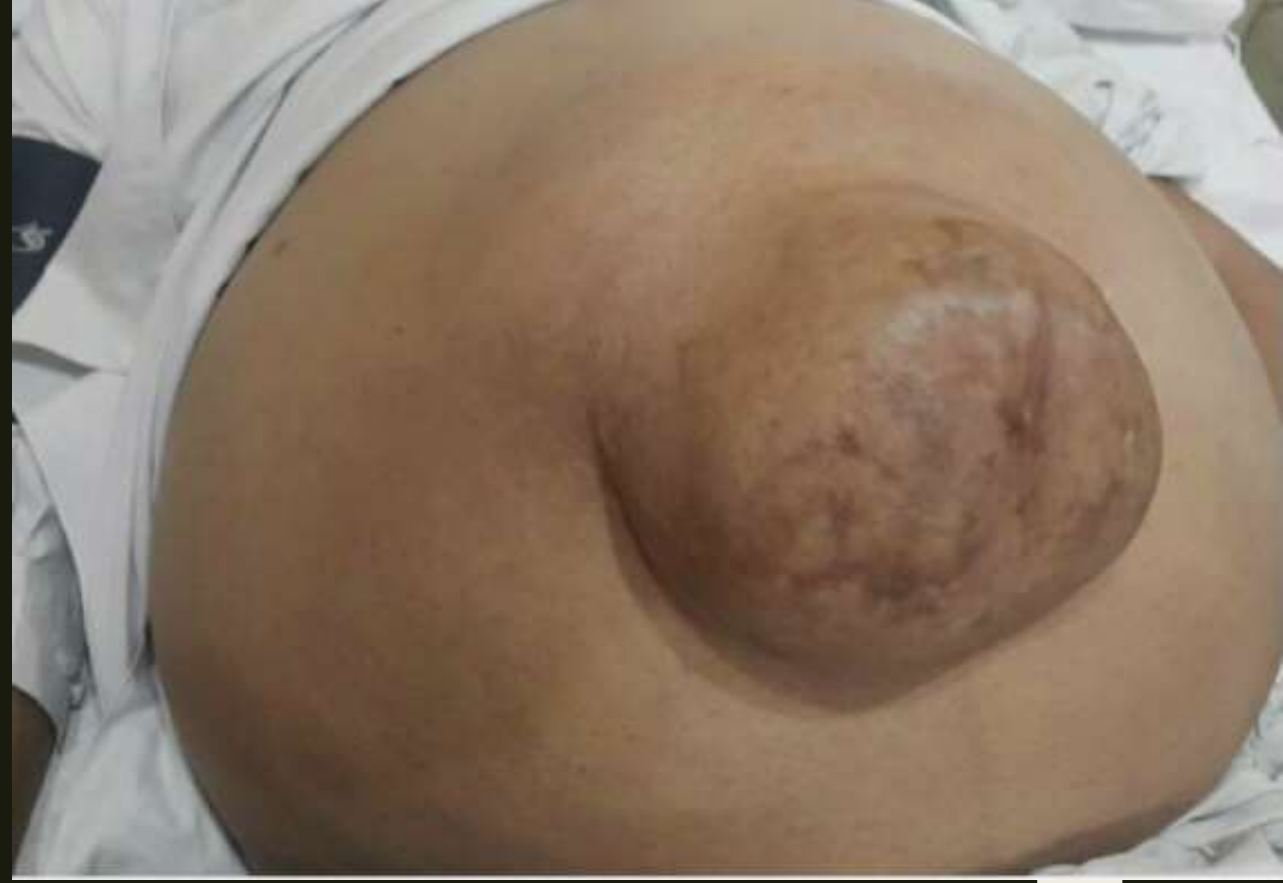
4. **Abdominal striae**

5. **Pleural effusions** are found in about 10% of patients , usually on the right side (hepatic hydrothorax) that can cause shortness of breath.



- In severe ascites the patient may present with :
- Fluid thrill test positive
- Eversion of the umbilicus
- Hernia





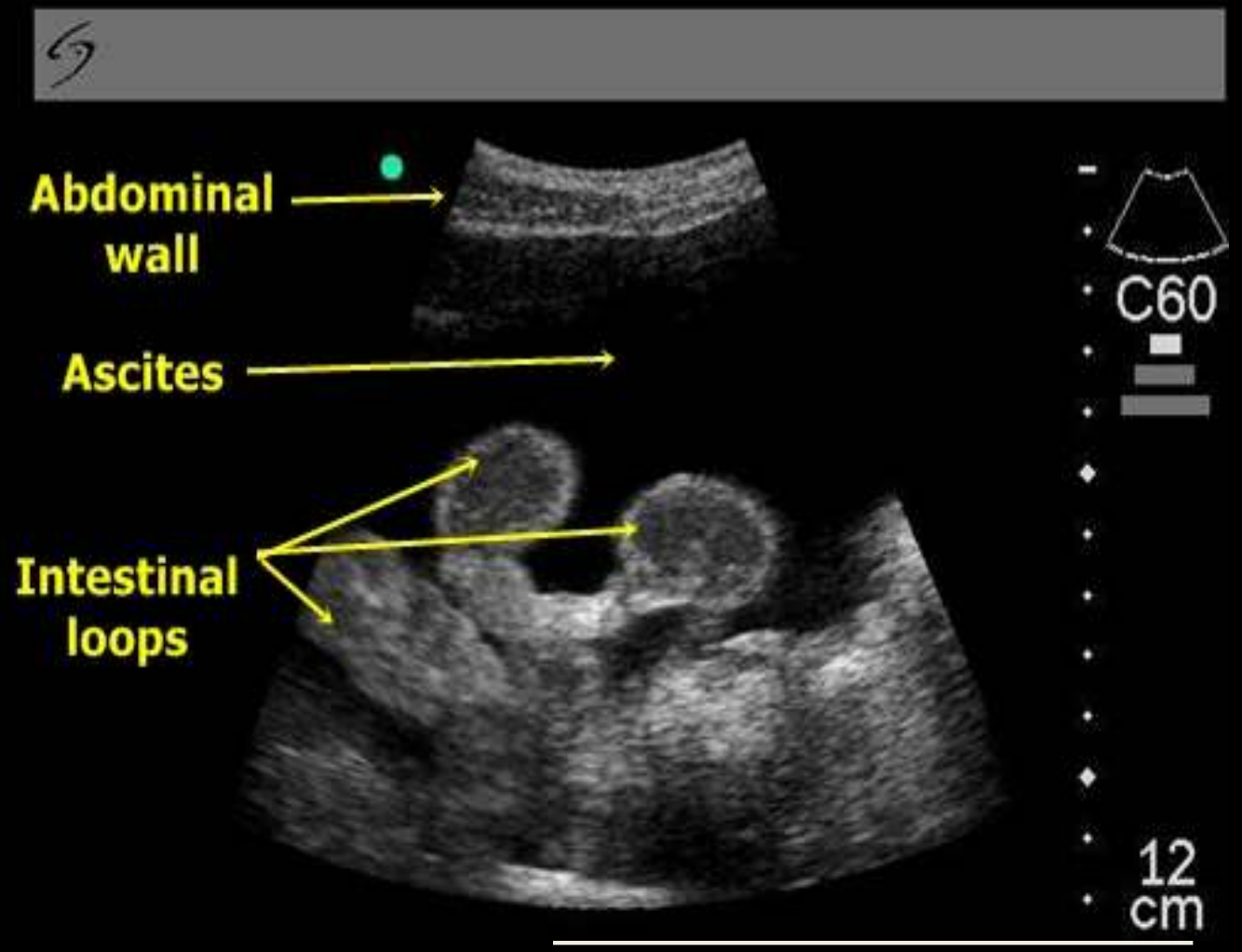
MARKED ABDOMINAL ASCITES WITH SUPRAUMBILICAL HERNIA

Investigations

- Ultrasonography
- Paracentesis
- Laparoscopy
- Biochemical tests
- Cytological studies

INVESTIGATIONS

- Ultrasonography
  - The best way to detect ascites.
  - Can estimate the amount of fluid especially in the obese and those with fluid of low volume .



PARACENTESIS

- Diagnostic or Therapeutic
- Used to obtain ascitic fluid for analysis under ultrasonic guidance

➢ Fluid appearance :

✓ Clear – Uncomplicated ascites in the setting of cirrhosis is usually translucent yellow; it can be completely clear if the bilirubin is normal and the protein concentration is very low.

✓ Turbid or cloudy – Spontaneously infected fluid is frequently turbid or cloudy.

✓ Milky – referred to as "chylous ascites“ eg: lymphatic obstruction, Malignancy.

✓ Pink or bloody – Mostly due to a "traumatic injury“, or malignancy.

✓ Brown – jaundice.

PARACENTESIS

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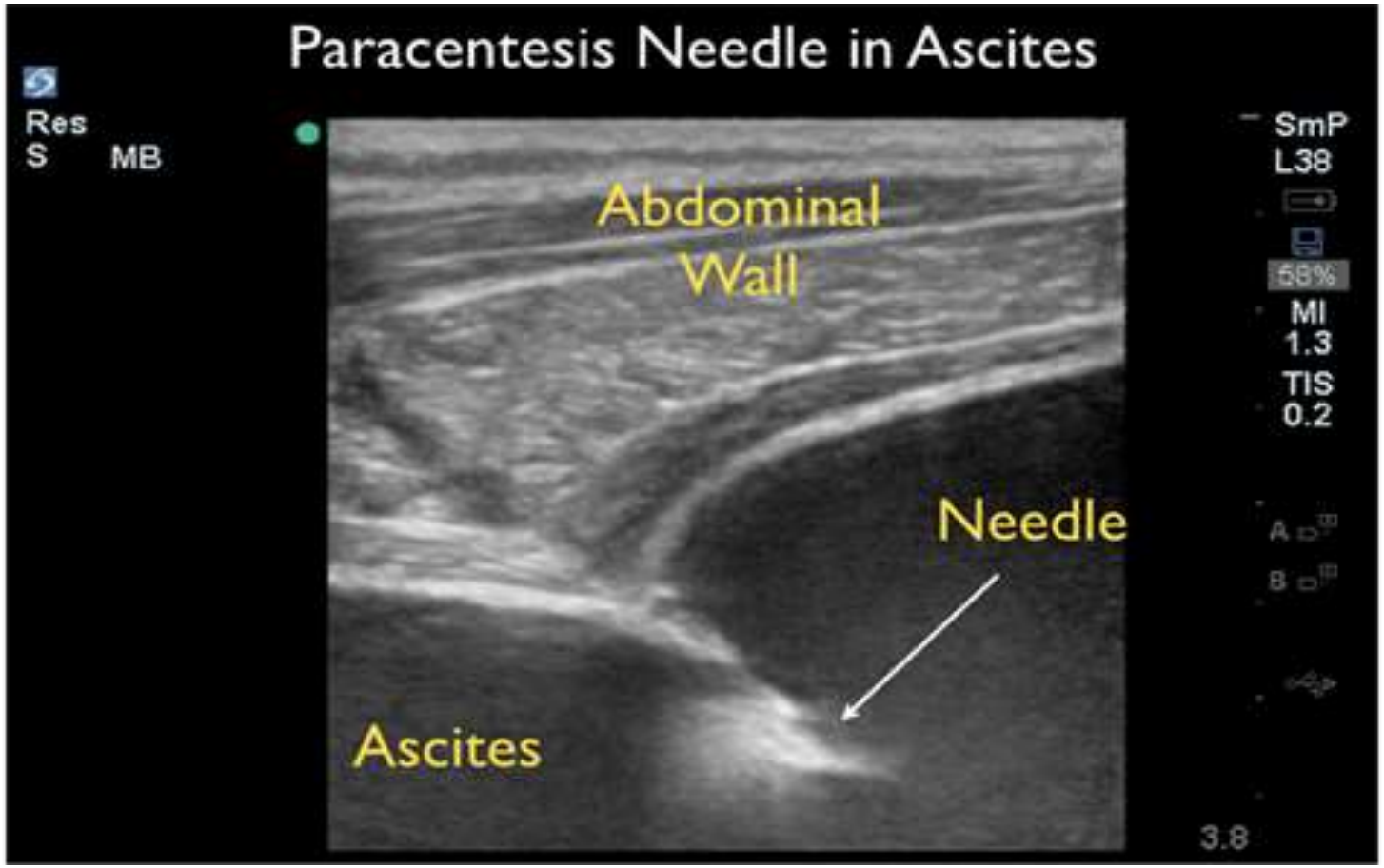
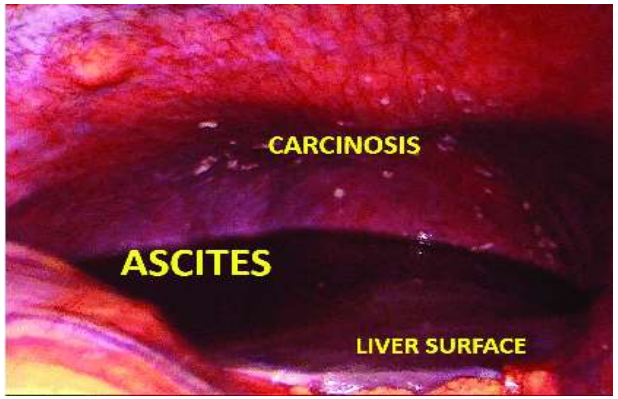
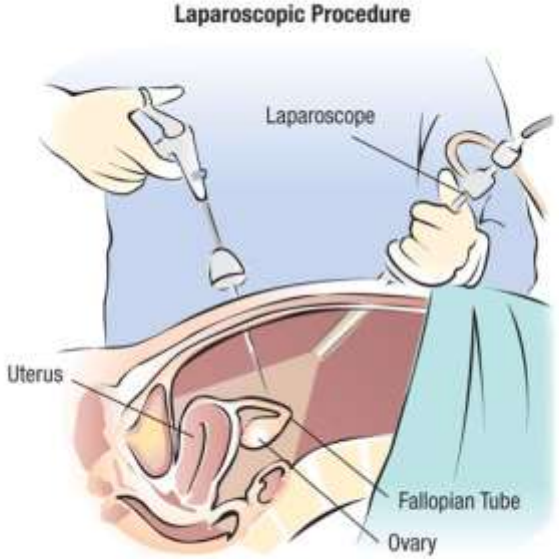


Laproscopy

- **Laparoscopy** in combination with biopsy can clarify the causes of unexplained **ascites** in the majority of cases: it failed to reveal any gross abnormality in only 15% of cases. Therefore, **laparoscopy** is a valuable tool for the detection of the cause of unexplained **ascites**.





Fluid analysis

- Biochemical tests:

1)- Protein concentration & serum-ascites albumin gradient (SAAG).

- Used to distinguish transudate from Exudate ascites.

2)- Ascites amylase activity .

- Above 100 U/L identifies pancreatic ascites .

3)- Lactate dehydrogenase(LDH).

4)- Adenosine deaminase.

(SAAG – ascites total protein) test

Causes	SAAG = Albumin ascites – Albumin serum g/L	Ascites T. protein g/L	
Cirrhosis, liver failure ,myxedema and SBP	> 11	<25	
Right heart failure, Budd-Chiari syndrome,	> 11	>25	
TB peritonitis, bacterial/fungal peritonitis, nephrotic syndrome, pancreatitis, and peritoneal carcinomatosis	< 11	> 25	

MANAGEMENT AND TREATMENT

- Sodium and water restriction
- Diuretics
- Therapeutic Paracentesis
- Transjugular intrahepatic portosystemic stent shunt (TIPSS)
- Peritoneo-Venous shunt

Sodium & water restriction

- Decrease Sodium intake to 100 mmol/day is usually adequate.
- Some **drugs** contain significant amounts of sodium (e.g. phenytoin and antacids)
- others cause sodium retention (NSAIDs and corticosteroids)
- **Water intake restriction**

Diuretics

- The first line diuretic is **Spironolactone.**
- If it causes painful gynaecomastia and hyperkalemia (complications?) replace with ameloride (diuretic / complications) (5-10mg/day).
- If not responsive : loop diuretic (furosemide) are used.
- can cause hyponatremia, hypokalemia and fluid depletion.

Diuretic-resistant ascites management

- **Refractory ascites** (or diuretic-resistant ascites), i.e. ascites that cannot be mobilized by medical treatment (low sodium diet and high doses of furosemide and spironolactone) is an infrequent phenomenon in cirrhosis.

- **Therapeutic options for patients who fail noninvasive treatments include:**

❖ **Liver transplantation.**

❖ **Serial therapeutic paracenteses.**

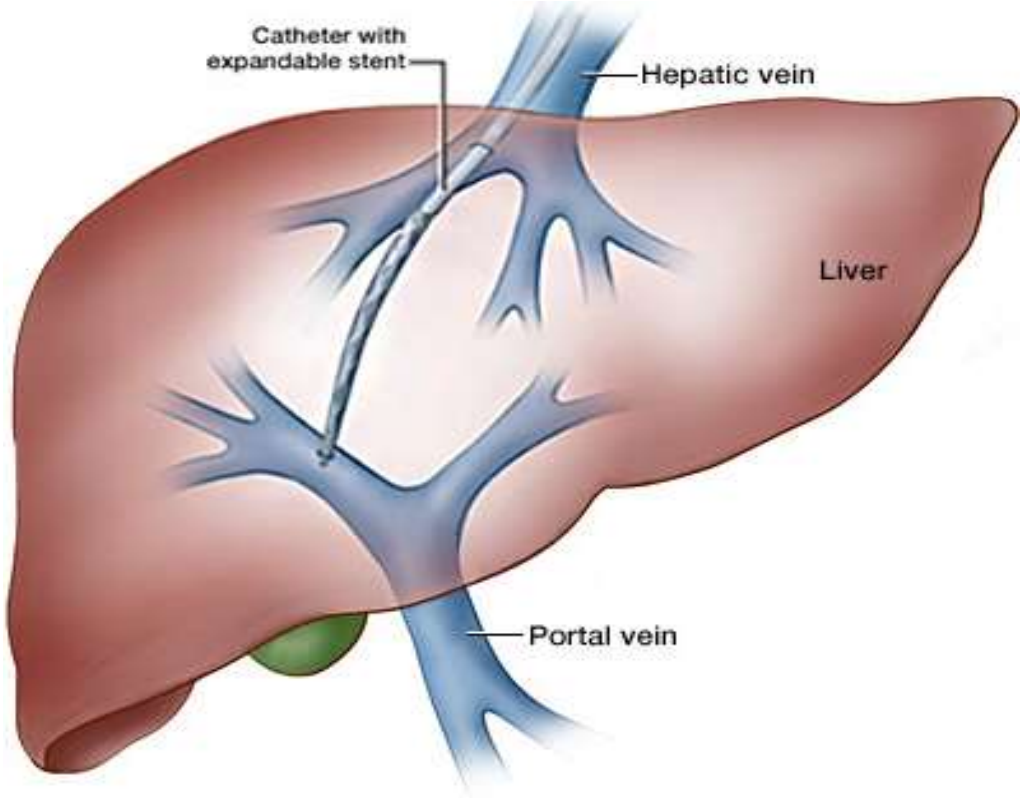
❖ **Trans-jugular intrahepatic portosystemic stent shunt (TIPS) placement.**

Paracentesis

- FIRST LINE treatment in Refractory (resistance) ascites
- Used as an initial therapy, or when other methods failed.
- IV colloid (E.g. human albumin) of (6-8 g / liter of ascites removed) should be administered to support the circulation and prevent hypovolemia and renal dysfunction

Transjugular intrahepatic portosystemic stent shunt (TIPSS)

- A stent placed between the portal vein and the hepatic vein within the liver to provide a portosystemic shunt and reduce portal pressure.



TIPSS

- Can relieve resistant ascites
- It can be used in patients waiting for liver transplantation or in those with reasonable liver function.
- Can aggravate hepatic encephalopathy in those with poor function. caused by increased porto-systemic passage of nitrogen from the gut.

COMPLICATIONS

1. **Renal failure:**

   due to vasodilatation from sepsis ,diuretic therapy or hepatorenal syndrome.

2. **Hepatorenal syndrome (HRS)**

3. **Spontaneous bacterial peritonitis (SBP)** (a life-threatening infection of the ascites fluid).

HEPATORENAL SYNDROME "OLIGURIC HEPATIC FAILURE"

- Life-threatening medical condition that consists of rapid deterioration in kidney function in individuals with cirrhosis
- Deteriorating liver function is believed to cause changes in the circulation that supplies the intestines, altering blood flow and blood vessel tone in the kidneys.
- The kidney failure of HRS is a consequence of these changes in blood flow, rather than direct damage to the kidney

THERE ARE TWO TYPES OF HRS:

- Type 1 : characterized by rapidly progressive kidney failure, with a doubling of serum creatinine to a level greater than 2.5 mg/dL - have a median survival time of two weeks - Almost everyone with this type of the disease will die within 8 to 10 weeks.

- Type 2 : is associated with ascites that does not improve with standard diuretic medications. serum creatinine level to >1.5 mg/dL -The median survival time for type 2 is six months.

SPONTANEOUS BACTERIAL PERITONITIS "SBP"

Spontaneous bacterial peritonitis (SBP) is indicated by a peritoneal fluid with > 250 PMN/mL in a patient with ascites. Usual causes are £. coli, then S. pneumoniae, then Klebsiella.

Because SBP patients may not have abdominal pain or tenderness, you must consider SBP if there is deterioration in the status of any patient with ascites; e.g., new onset confusion, fever, signs of hepatic encephalopathy, or renal failure.

- You must rule out 2 other possible causes of high WBC in ascitic fluid before you can assume it is SBP:

  I) Neutrocytic ascites: Basically, this is PMNs > 250/ ml with no evidence of SBP and negative cultures.

  2) Primary bacterial peritonitis (PBP) is due to perforated viscus. In cirrhotic, it can be confused with SBP.

SBP DIAGNOSIS

- The diagnosis of SBP requires paracentesis from the abdominal cavity .
- Cloudy fluid , neutrophil > 250 x 10^6 \L and antibiotics required to avoid complications
- Tx: Start immediately with broad spectrum antibiotics (e.g. cefotaxime) .
- Recurrence of SBP is common but may be reduced with prophylactic quinolone such as norfloxacin or ciprofloxacin
  - *IV albumin is also given in the treatment of SBP (1.5 g/kg on day I and I g/kg on day 3).*
    Albumin maintains blood volume and thereby decreases the incidence of irreversible renal impairment and mortality.

OTHER COMPLICATIONS

4. Pre-renal due to vasodilation from sepsis and\or diuretics.
5. Pleural effusion.
6. Weight loss and protein malnutrition.
7. Mental confusion, change in the level of alertness, or coma (hepatic encephalopathy).
8. Bleeding from the upper or lower intestine.

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Thank you