ASCITES

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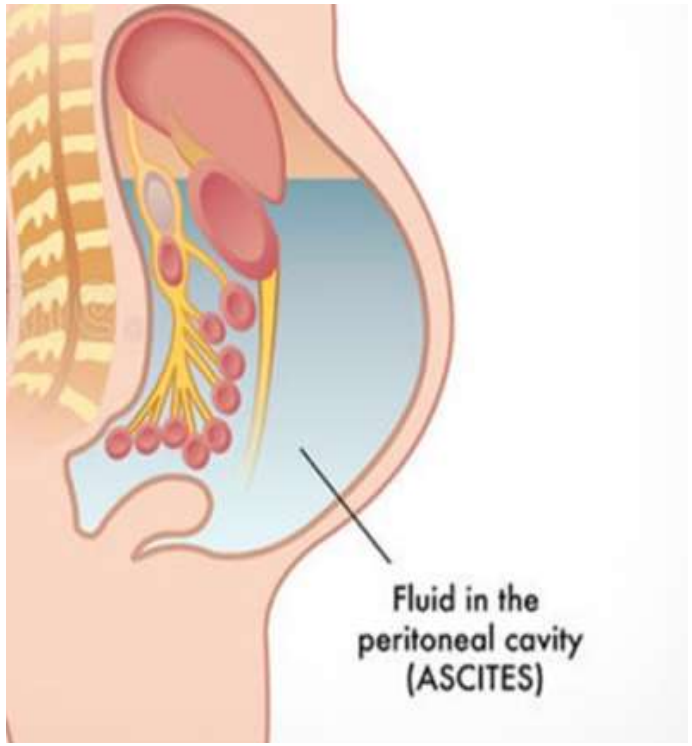
Ascites

Abnormal build up or accumulation of free fluid in the peritoneal cavity. Ascites is the most common complication of cirrhosis

Small amounts of ascites are asymptomatic, but with larger accumulations of fluid more than 1 L there :



* abdominal distension

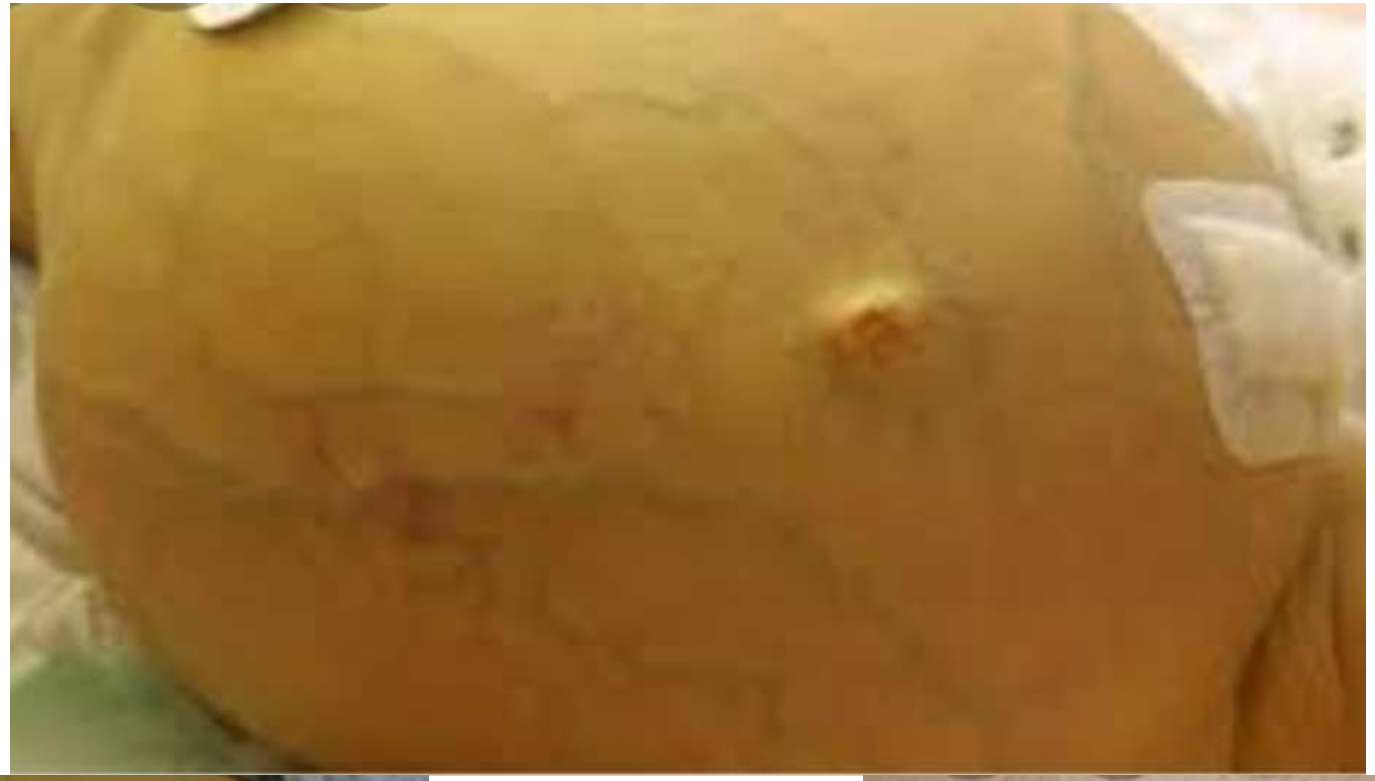
*fullness in the flanks

* shifting dullness on percussion

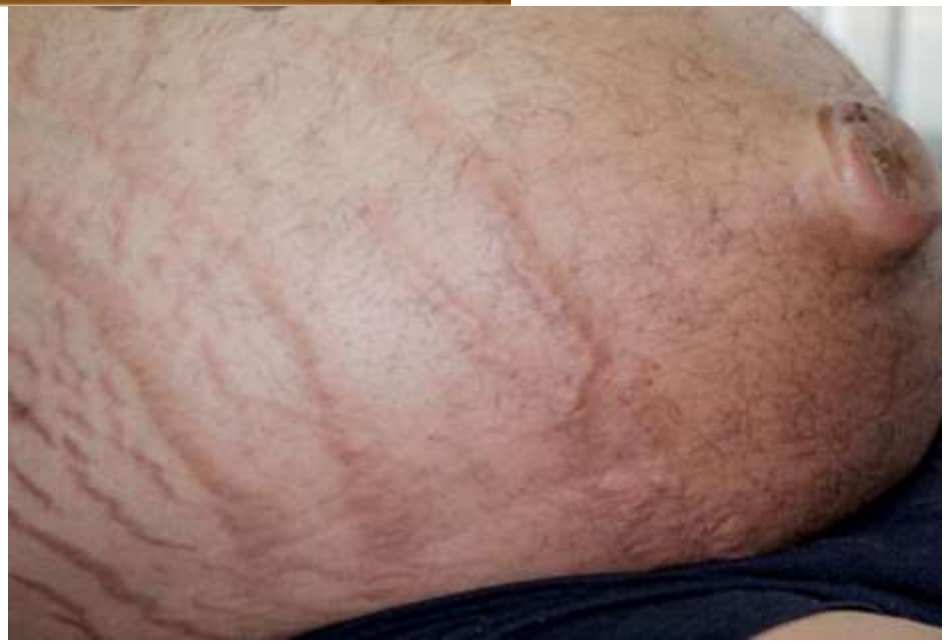
* fluid thrill/ fluid wave when ascites marked

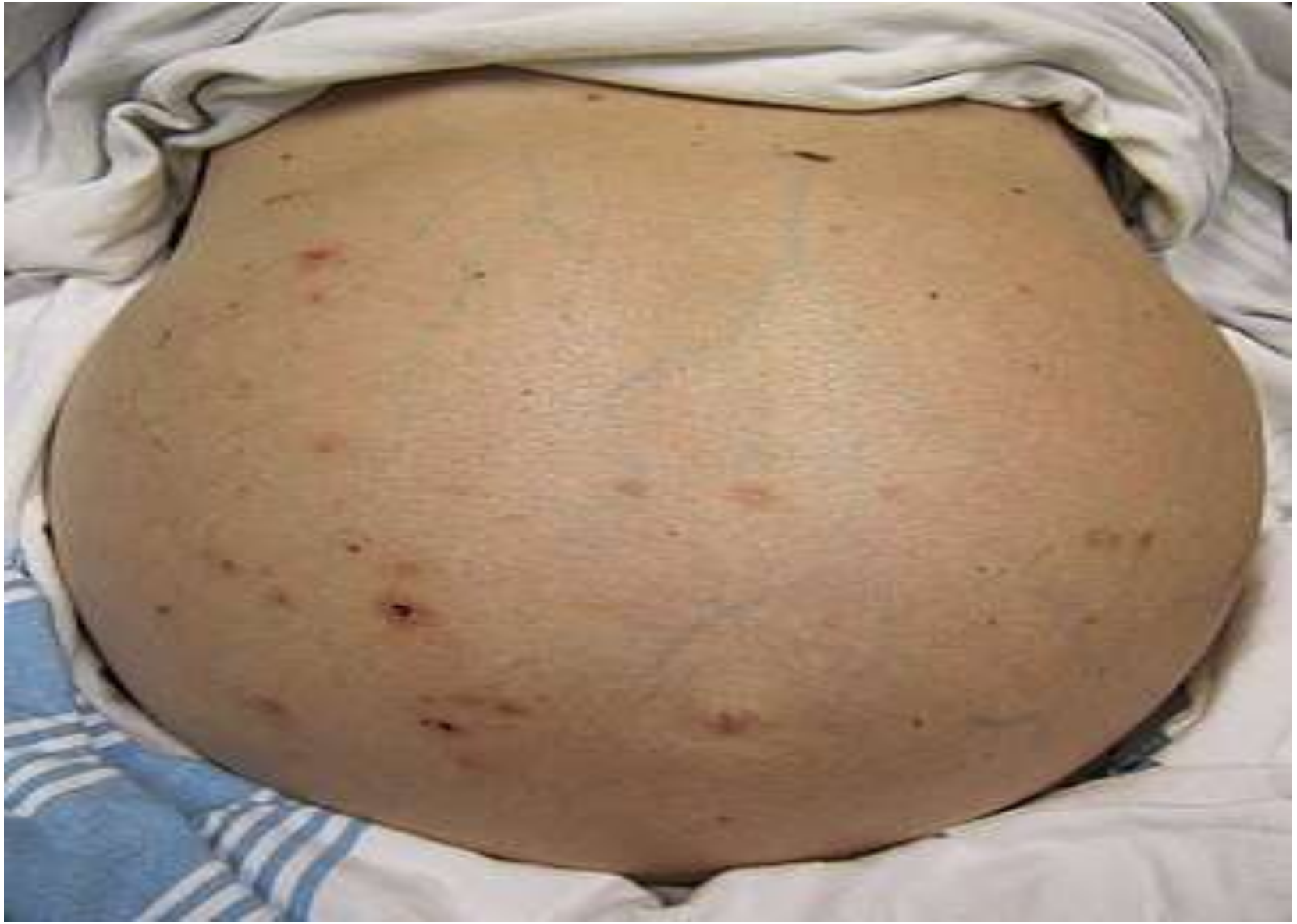
Feature of ascites

- Eversion of umbilicus.
- Hernia.
- Abdominal striae.
- Divarication of recti and scrotal edema.
- Dilated superficial abdominal veins due to portal hypertension.




Ascites





Causes:

- **Causes of high serum ascites albumin gradient(SAAG) are:**

- Heart failure
- Hepatic cirrhosis
- Hepatic venous occlusion as(budd-chiari syndrome)

- ***Cause of low Serum ascites albumin gradient (SAAG) are:***

- Malignant : * hepatic
  * peritoneal

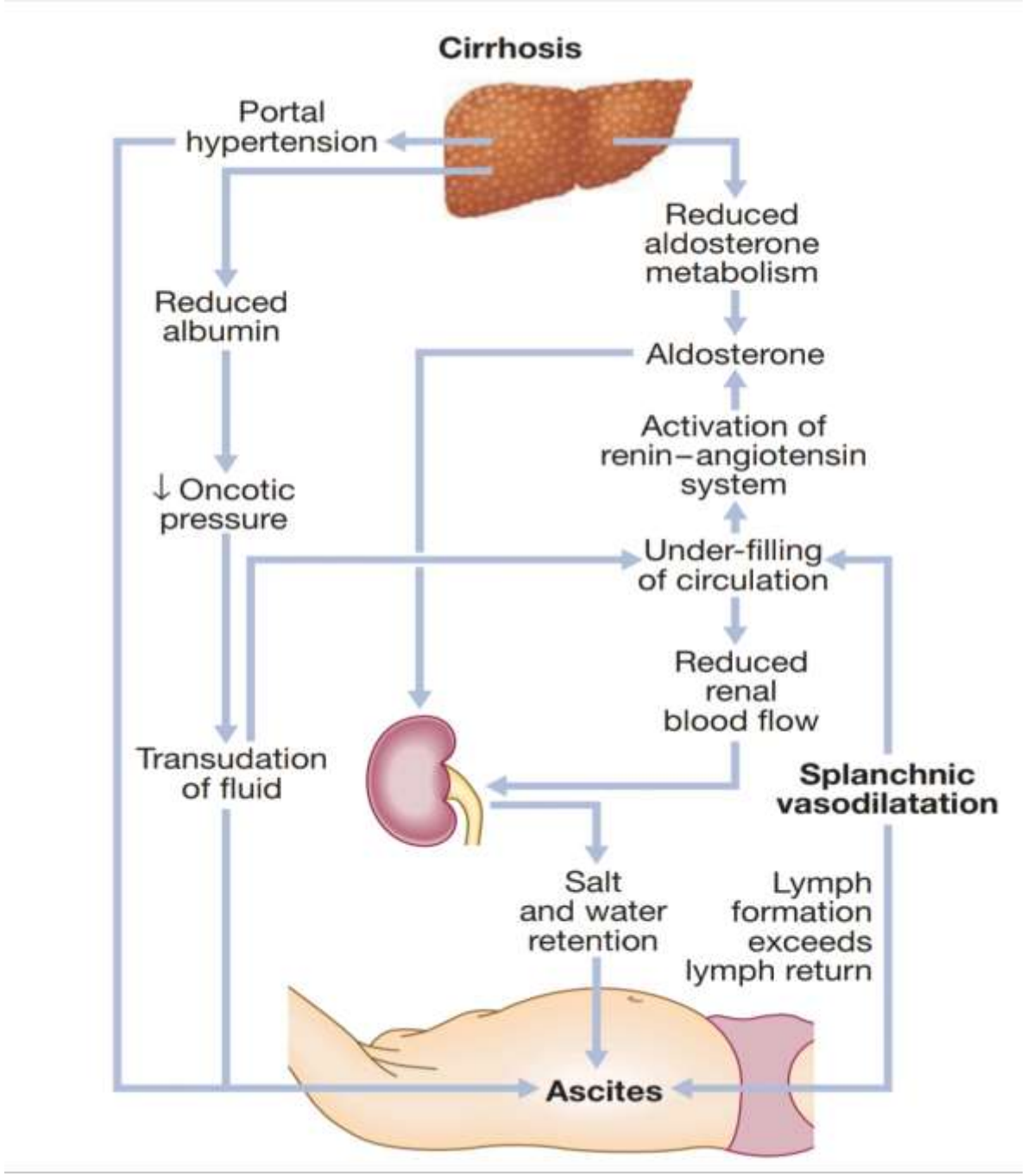
- ***Other causes :***

Acute pancreatitis
Lymphatic obstruction
Infection (TB)
Nephrotic syndrome

Pathophysiology

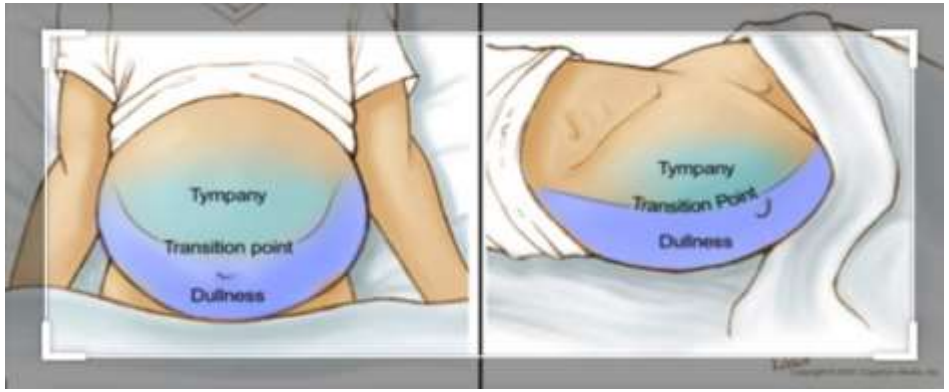
Splanchnic vasodilation is thought to be main factor leading to ascites in cirrhosis. This is mediated by vasodilators (nitric oxide) that are released when portal hypertension causes shunting of blood into the systemic circulation. This lead to fall systemic arterial pressure this lead to activation renin-angiotensin with secondary aldosteronism Increased sympathetic nervous activity and atrial natriuretic hormone secretion.

These systems tend to normalise arterial pressure but produce salt and water retention. In this setting the combination of splanchnic arterial vasodilation and portal hypertension alters intestinal capillary permeability promoting accumulation fluid within perineum.



Physical examination

Shifting dullness



In ascites the dullness shifting in More dependant side , while tympanic Shift to top

Transmitted thrill/fluid wave

Investigation

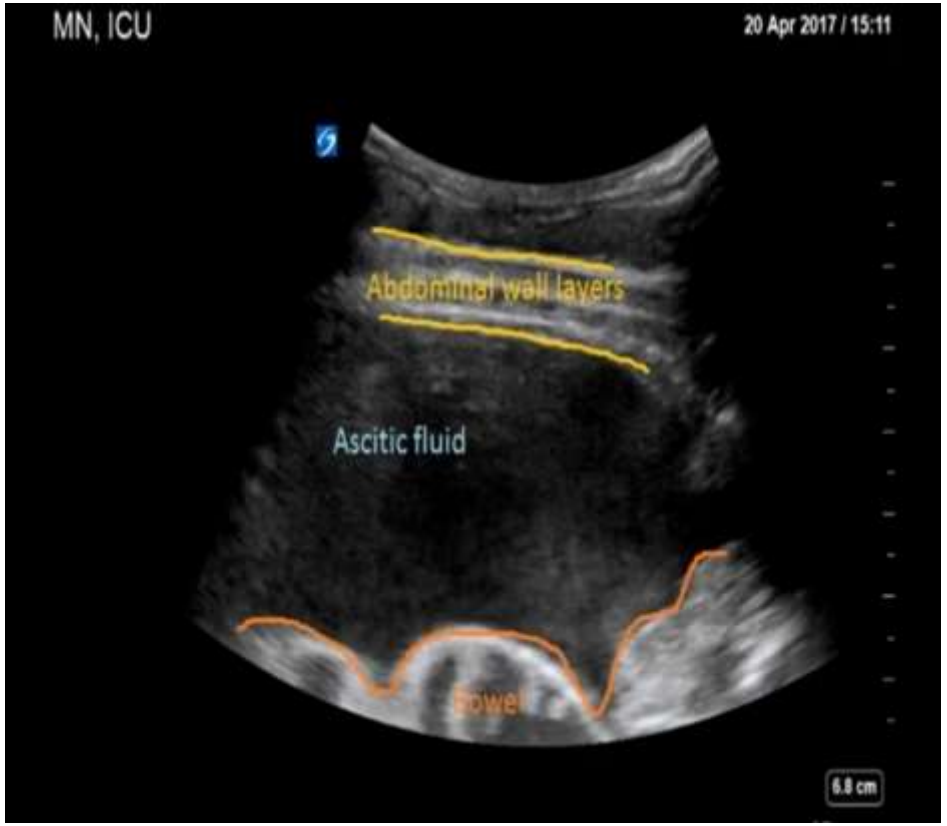
Ultrasonography is the best mean of detecting ascites particularly in the obese patient and those with small volumes of fluid .

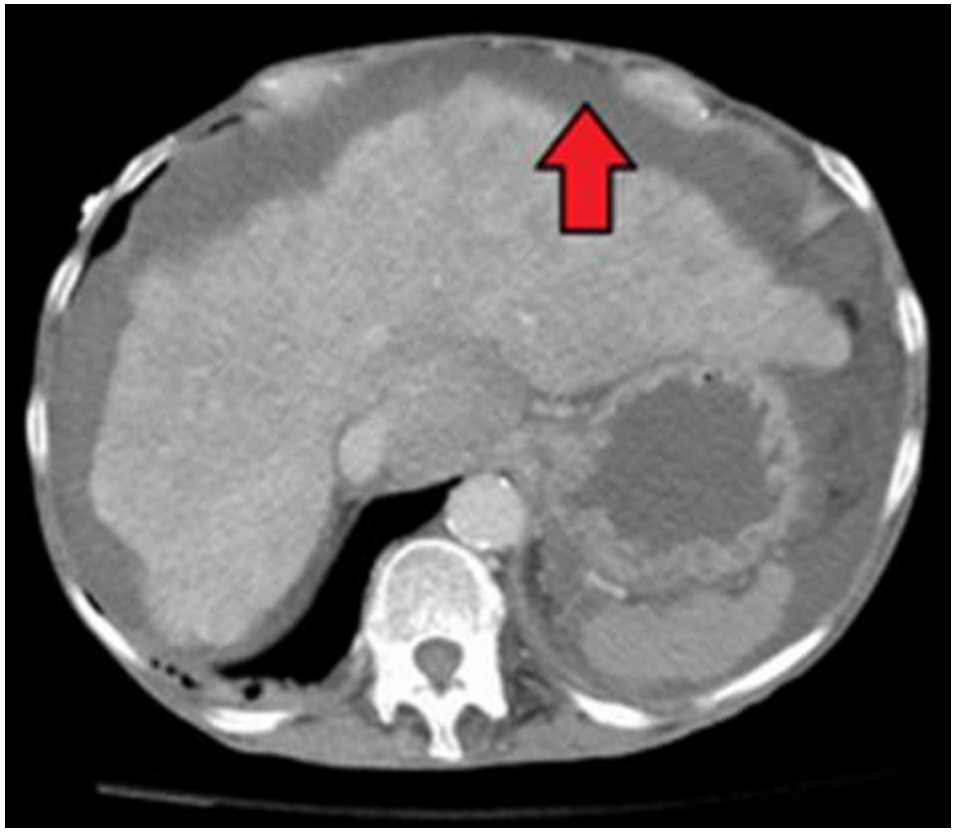
Paracentesis can be used to obtain ascites fluid for analysis (Peritoneal cavity is punctured by needle to sample peritoneal fluid) .

Pleural effusion are found in 10% of patient usually in right side (hepatic hydrothorax) most small and identified only on chest x ray but occasionally a massive hydro thorax occured.

Pleural effusion in left side should not be assumed to be due to the ascites.

CT scan: The most sensitive technique.


MN, ICU
20 Apr 2017 / 15:11
Abdominal wall layers
Ascitic fluid
Bowel
6.8 cm



Fluid analysis

- (SAAG) can be a useful tool to distinguish ascites of different etiologies

- Transudative: protein concentration < 25 g/L (2.5 g/dL)) and relatively few cells so SAAG > 11 g/L (1.1 g/dL) which caused by Venous outflow obstruction due to cardiac failure or hepatic venous outflow obstruction

- Exudative :protein concentration > 25 g/L (2.5 g/dL) or a SAAG of < 11 g/L (1.1 g/dL) raises the possibility of infection (especially tuberculosis), malignancy, pancreatic ascites or, rarely, hypothyroidism

Triglyceride at a level > 1.1 g/L (110 mg/dL suggests anatomical or functional abnormality of lymphatic drainage from the abdomen. The ascites in this context has a characteristic milky-white appearance.

22.22 Ascitic fluid: appearance and analysis

Cause/appearance

- Cirrhosis: clear, straw-coloured or light green
- Malignant disease: bloody
- Infection: cloudy
- Biliary communication: heavy bile staining
- Lymphatic obstruction: milky-white (chylous)

Useful investigations

- Total albumin (plus serum albumin) and protein*
- Amylase
- Neutrophil count
- Cytology
- Microscopy and culture

Management

Treatment of transudative ascites :

- On restricting sodium and water intake
- Promoting urine output with diuretics
- If necessary, removing ascites directly by paracentesis
- During management of ascites, the patient should be weighed regularly. Diuretics should be titrated to remove no more than 1 L of fluid daily, so body weight should not fall by more than 1 kg daily to avoid excessive fluid depletion.

Treatment of exudative ascites :

- Is treated with paracentesis

Sodium and water restriction

- Restriction of dietary sodium intake is essential to achieve negative sodium balance and a few patients can be managed satisfactorily by this alone
- Restriction of sodium intake to 100 mmol/24 hrs (‘no added salt diet’) is usually adequate.
- Drugs containing relatively large amounts of sodium, and those promoting sodium retention, such as non-steroidal anti-inflammatory drugs (NSAIDs), must be avoided
- Restriction of water intake to 1.0–1.5 L/24 hrs is necessary only if the plasma sodium falls below 125 mmol/L

22.23 Some drugs containing relatively large amounts of sodium or causing sodium retention

High sodium content

- Alginates
- Antacids
- Antibiotics
- Phenytoin
- Effervescent preparations (e.g. aspirin, calcium, paracetamol)
- Sodium valproate

Sodium retention

- Carbenoxolone
- Glucocorticoids
- Metoclopramide
- Non-steroidal anti-inflammatory drugs
- Oestrogens

Diuretics

- Diuretics Most patients require diuretics in addition to sodium restriction
- Diuresis may be improved if patients are rested in bed, perhaps because renal blood flow increases in the horizontal position
- **Spironolactone (100–400 mg/day):** is the first-line drug because it is a powerful aldosterone antagonist

  SIDE EFFECT :painful gynaecomastia and hyperkalaemia
- ***amiloride (5–10 mg/day)*** can be substitution for spironolactone
- ***loop diuretics, such as furosemide:*** but these can lead to fluid and electrolyte imbalance and renal dysfunction
- Patients who do not respond to doses of 400 mg spironolactone and 160 mg furosemide, or who are unable to tolerate these doses due to hyponatraemia or renal impairment, are considered to have refractory or diuretic-resistant ascites and should be treated by other measures

Paracentesis

- First-line treatment of refractory ascites is large-volume paracentesis.
- Paracentesis to dryness is safe, provided the circulation is supported with an intravenous colloid such as human albumin (6–8 g albumin for each one liter of aspirated fluid if 5 L removed at once) .
- Paracentesis can be used as an initial therapy or when other treatments fail

Complications

- ***Renal failure***
- It can be pre-renal and due to vasodilatation from sepsis and/or diuretic therapy, or due to hepatorenal syndrome which occurs in 10% of patients with advanced cirrhosis complicated by ascites,
- ***Hepatorenal syndrome***

✓ It is a acute reversible form of renal failure in patient with cirrhosis

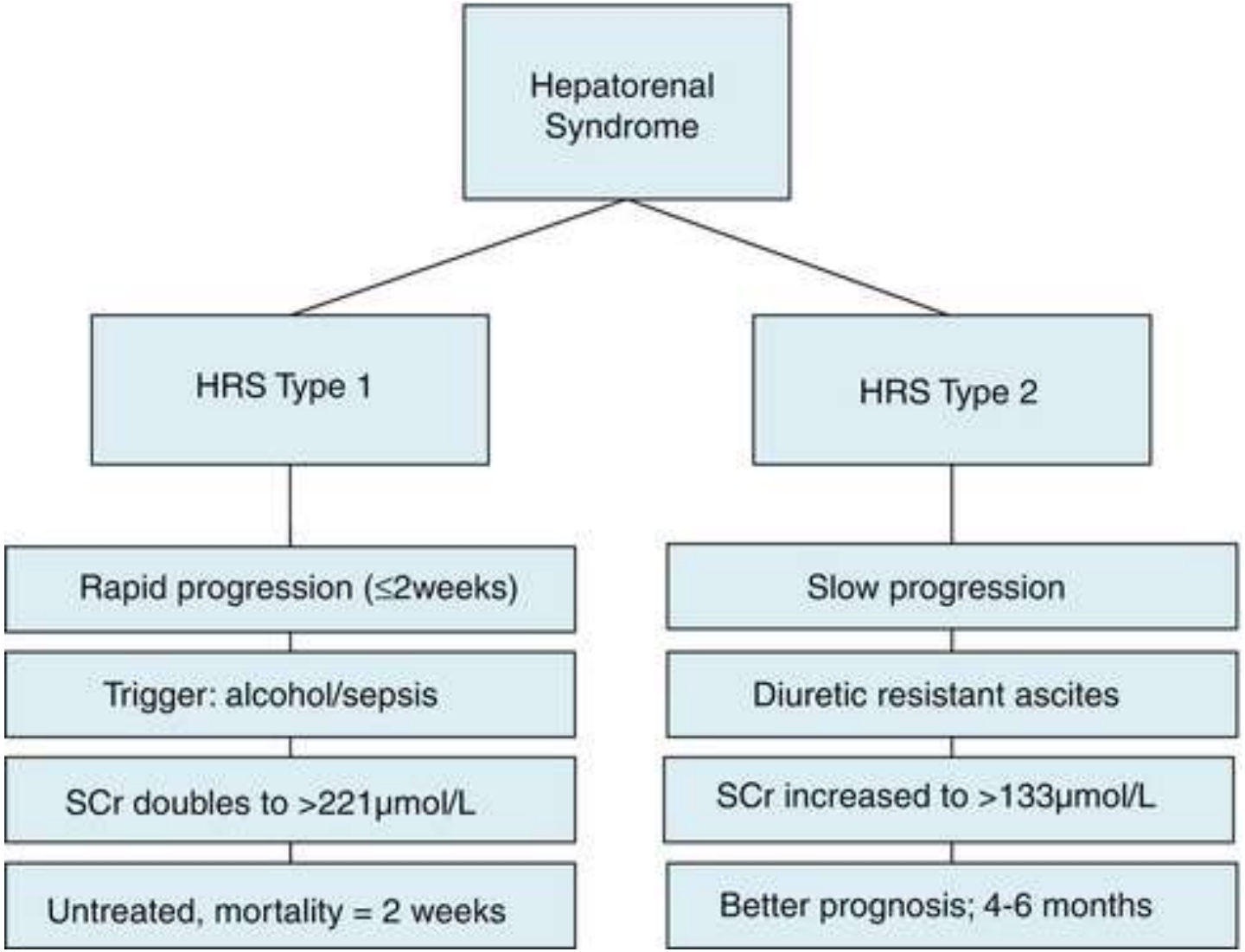
✓ Cause: unkown

✓ Triggers: refractory ascites….large volume paracentesis without albumin replacement ….SBP

✓ Caused by combination of VD in splanchnic and VC in renal circulation

✓ The first line of treatment : vasopressin (terlipressin) and albumin

✓ Liver transplantation is the best option esp. for type 1


Hepatorenal Syndrome
HRS Type 1
HRS Type 2
Rapid progression (≤2weeks)
Trigger: alcohol/sepsis
SCr doubles to >221μmol/L
Untreated, mortality = 2 weeks
Slow progression
Diuretic resistant ascites
SCr increased to >133μmol/L
Better prognosis; 4-6 months

- ***Spontaneous bacterial peritonitis***

✓ Infection of ascites fluid without detectable intra abdominal infection and caused usually by E.coli

✓ Mechanism :

1.Translocation of gut bacteria through intestinal wall to colonize mesenteric LN ,thus entering the blood circulation and ascitic fluid

2.Hematogenous

✓ S&S: fever –abdominal pain and tenderness—rebound tenderness-no bowel sounds—altered mental status

✓ Diagnosis is based on :

✓ 1.Positive ascitic bacterial culture (monomicrobial)

✓ 2.Elevated absolute PMN count in ascitic fluid (more than 250cells /mm3)

✓ Treatment: broad-spectrum antibiotics, such as cefotaxime (5-10 days)

Thank you