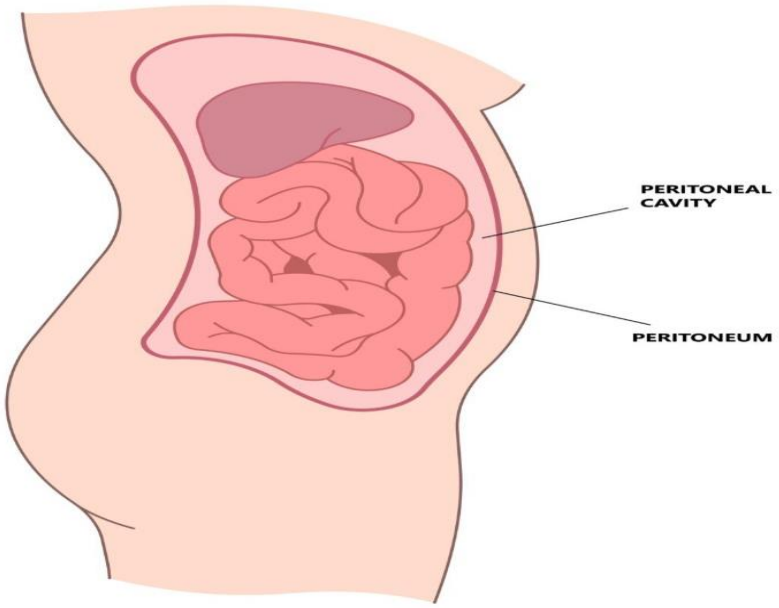
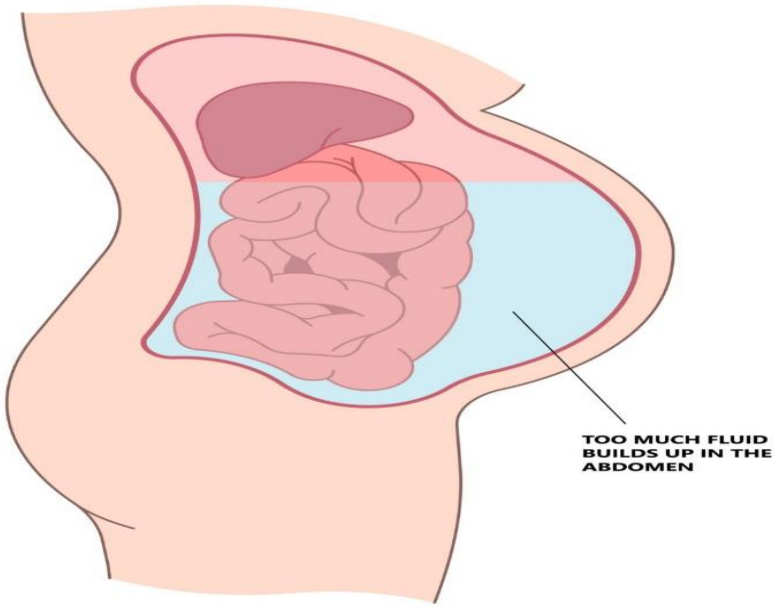
ASCITES



NORMAL ABDOMEN



ABDOMEN WITH ASCITES

Dana Al_Hazaimeh.

Lujain Sa’adeh.

Ahmad Khalid

Bailsan Al_hrout

Ascites

The word ascites is of Greek origin (askos) and means bag or sac. Ascites describes the condition of pathologic fluid collection within the abdominal cavity .

The amount of fluid required to consider intraperitoneal fluid as ascites is generally around 25 milliliters (mL) or more.

Not every intraperitoneal fluid accumulation is ascites; it can also result from physiological conditions like pregnancy.

Healthy men have little or no intraperitoneal fluid, but women may normally have as much as 20 mL, depending on the phase of their menstrual cycle.

Clinical features

***Small amounts of ascites are asymptomatic**

***but with larger accumulations of fluid (> 1 L) there is :**

1.abdominal distension

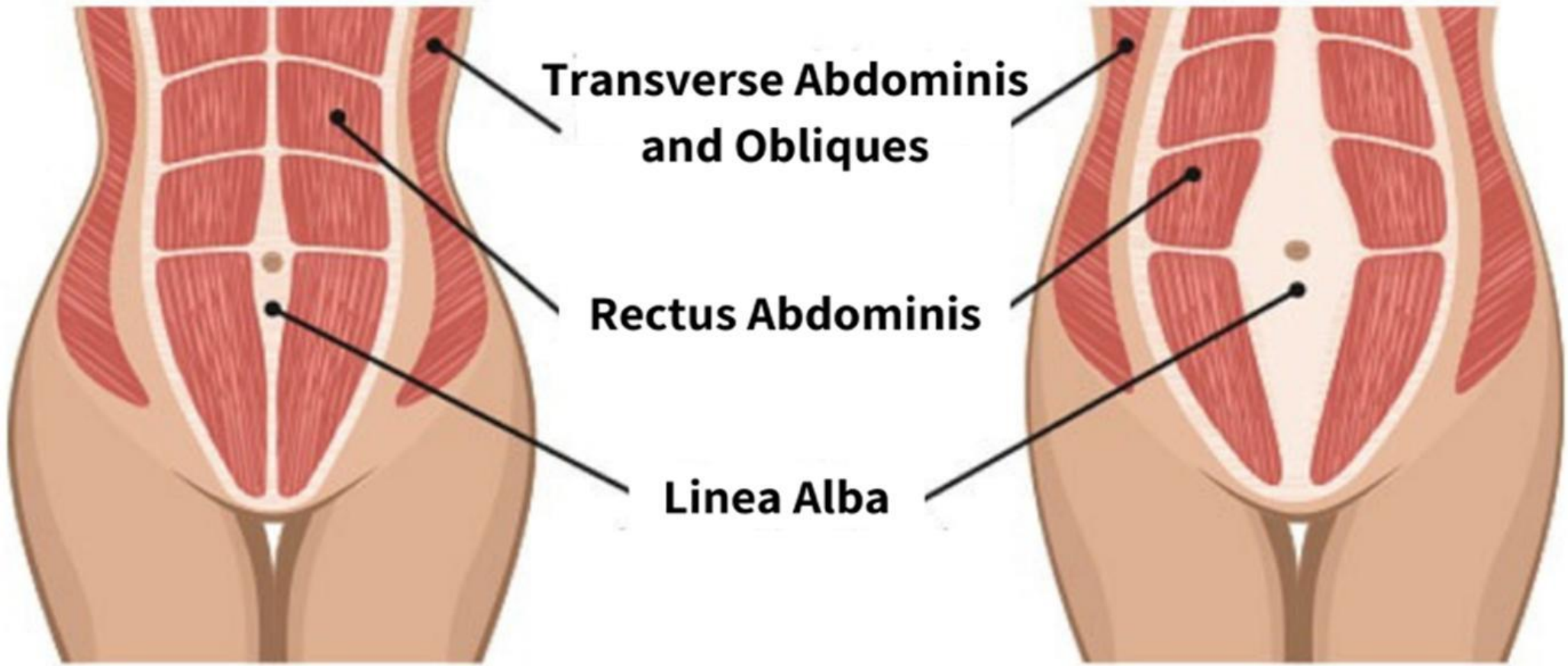
2.fullness in the flanks,

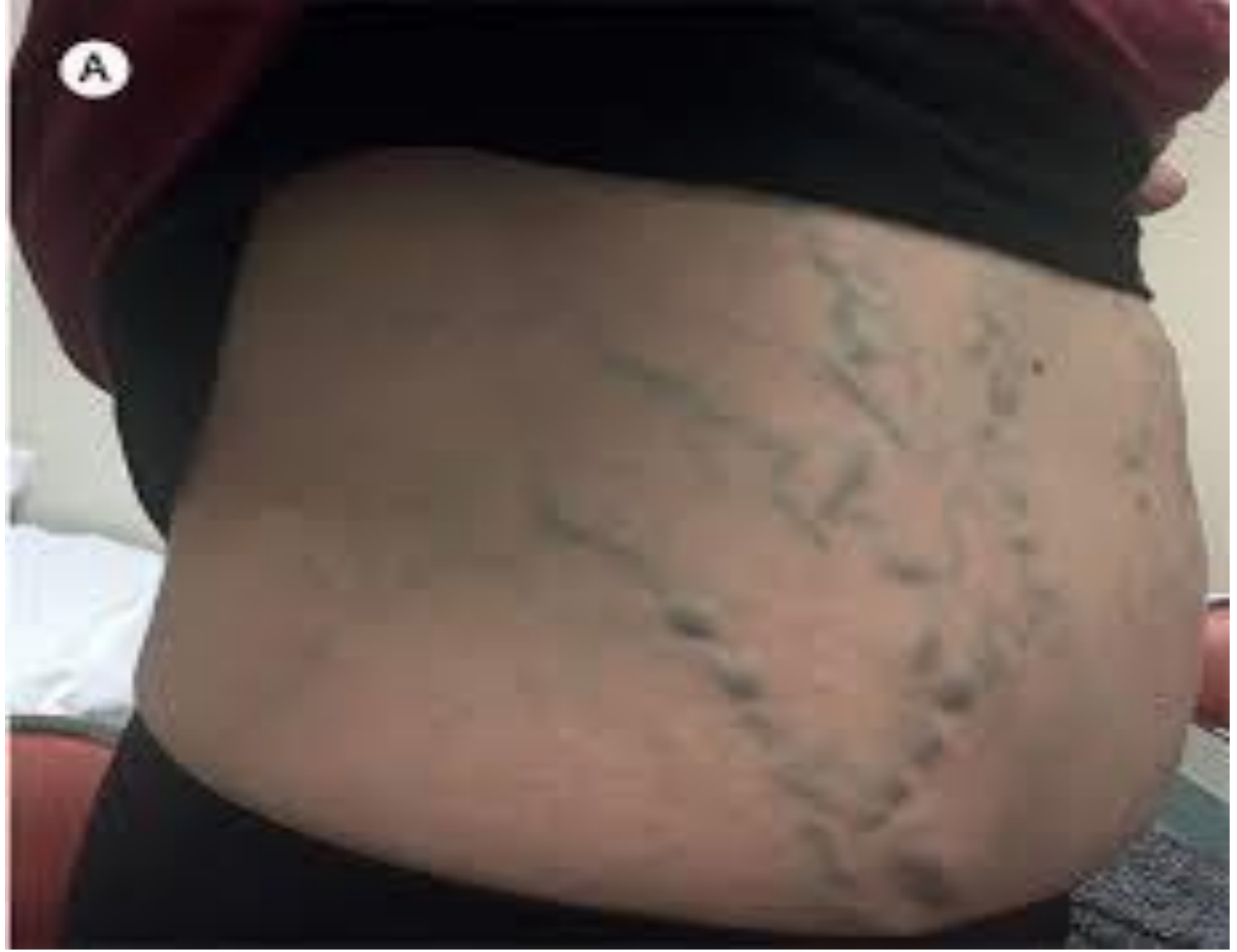
3. shifting dullness on percussion

4. when the ascites is marked, a fluid thrill.

Other features include eversion of the umbilicus, herniae, abdominal striae, divarication of the recti and scrotal oedema. Dilated superficial abdominal veins may be seen if the ascites is due to portal hypertension

DIASTASIS RECTI





dilated Superficial abdominal veins

Causes of Ascites

If ascites is present, the etiology of ascites can be determined by the serum-ascites albumin gradient (SAAG = serum albumin – ascites albumin).

TABLE 2.6-14. Etiologies of Ascites by SAAG

Saag > 1.1	Saag < 1.1
Related to portal hypertension: ■ **Presinusoidal:** Splenic or portal vein thrombosis, schistosomiasis. ■ **Sinusoidal:** Cirrhosis. ■ **Postsinusoidal:** Right heart failure, constrictive pericarditis, Budd-Chiari syndrome.	Not related to portal hypertension: ■ Nephrotic syndrome. ■ TB. ■ Malignancy with peritoneal carcinomatosis (eg, ovarian cancer).

Clinical case

A 55 years old woman,alcohol abuser ,a known case of liver cirrhosis, presented to a private clinic with a cheif complaint of increasing abdominal distension and discomfort over the past several weeks.

History and physical

History

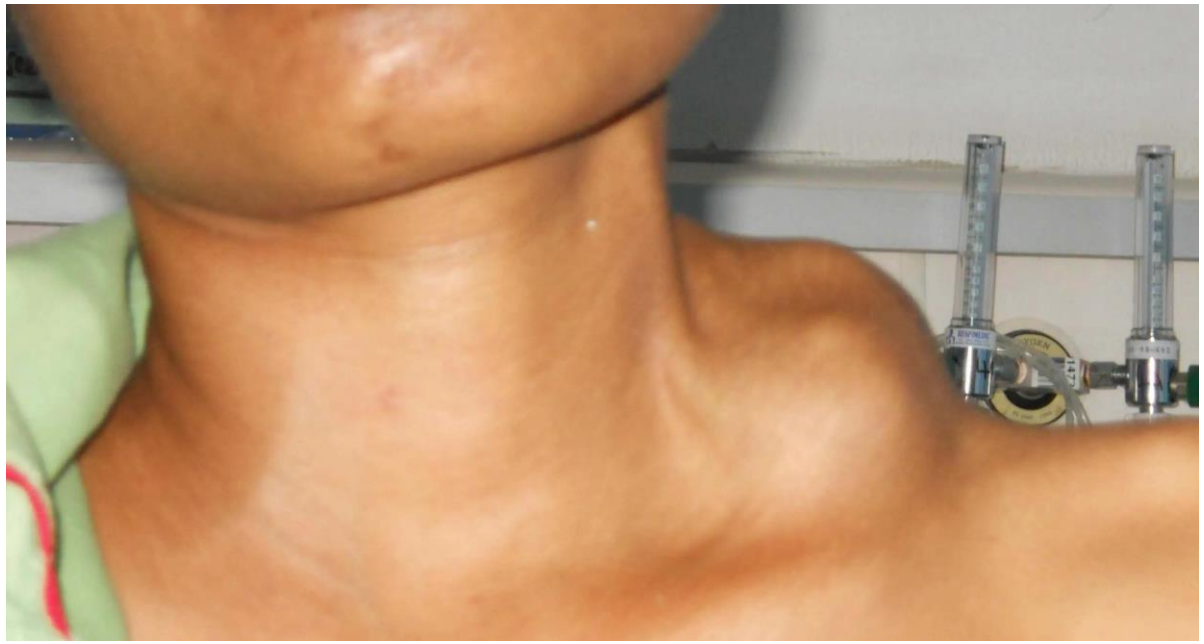
- Patient profile helps in narrowing the DDx through age (ped think of septic cause more common, compared to middle age where cirrhosis is more common), sex (females more susceptible to UTIs and ovarian lesions).

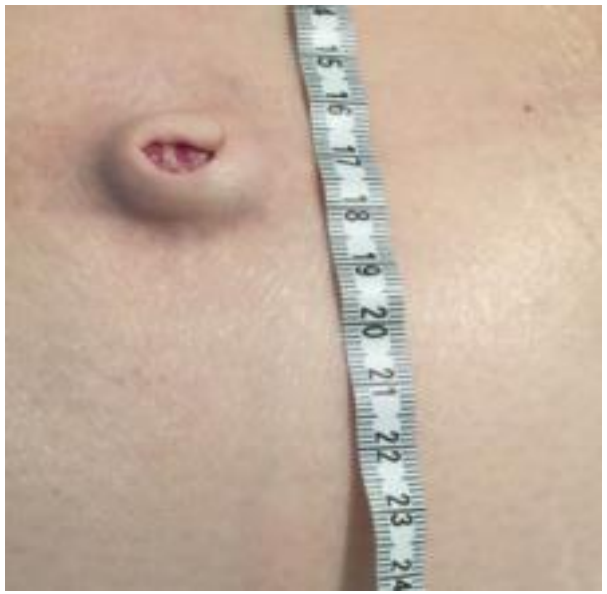
- Risk factors for the possible causes (most commonly liver disease) so we ask about:
  - Long-term heavy alcohol use
  - Chronic viral hepatitis or jaundice
  - IV drug abuse
  - Blood transfusion history
  - Sexual history
  - Tattoos
  - Manifestation of chronic liver disease including jaundice, pedal edema, gastrointestinal hemorrhage, or hepatic encephalopathy
  - Active tumor S&S (especially HCC in a patient with a very long history of stable cirrhosis)
  - History of malignancy (especially GI cancer, they are at risk for malignant ascites)
  - Metabolic syndrome (Obesity, hypercholesterolemia and DM2 as they can cause nonalcoholic steatohepatitis, which progresses to cirrhosis)
  - nephrotic syndrome (nephrotic ascites)

Physical examination

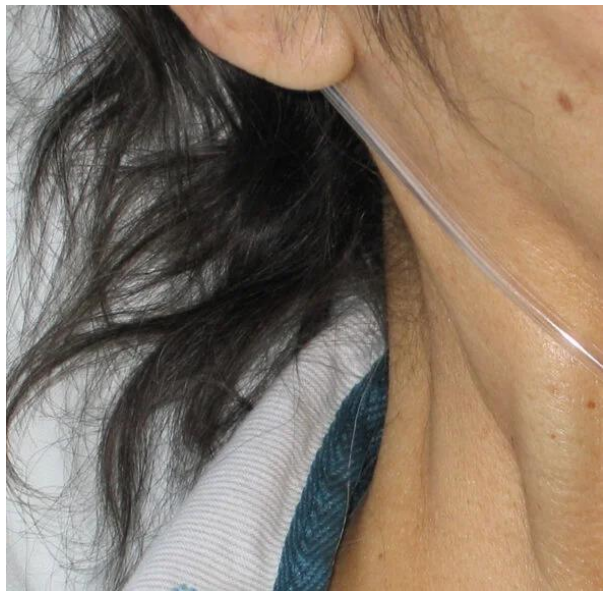
- Vitals
- General look (confusion sign for encephalopathy)
- Inspection (mainly head, neck, chest, abdomen and hands)
  1. Jaundice (sclera)
  2. spider naevus (unusual below the umbilicus)
  3. Gynecomastia
  4. Muscle wasting
  5. palmar erythema and leukonychia (white nails).
  6. Elevated jugular venous pressure.
  7. left-sided supraclavicular node (Virchow node)
  8. Sister Mary Joseph nodule

Virchow node

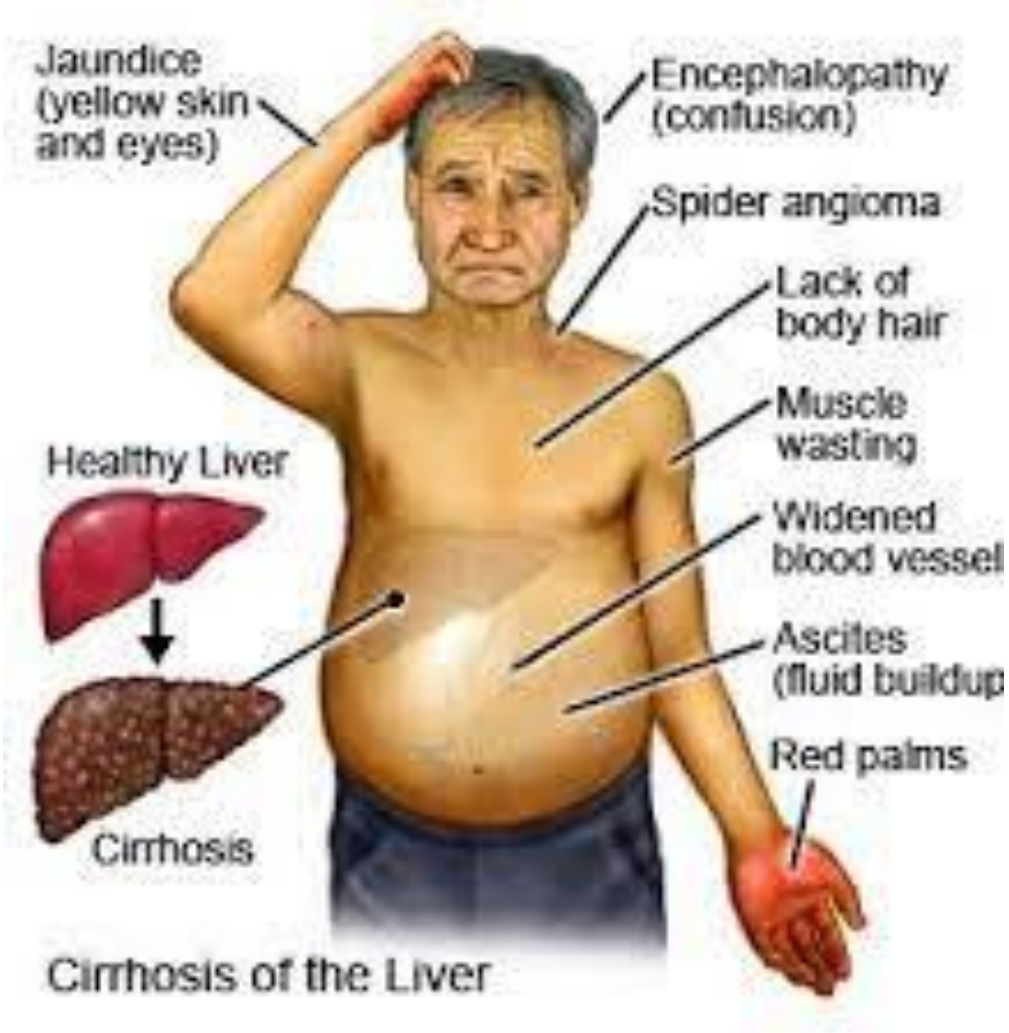




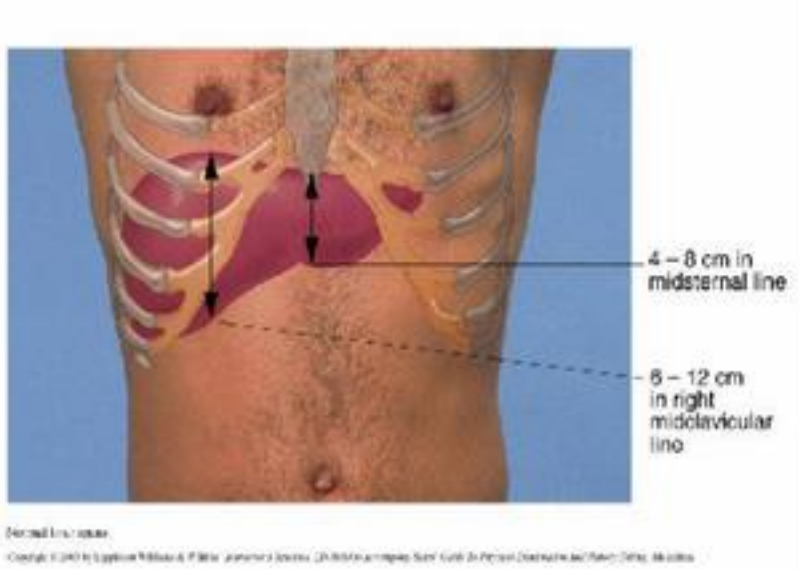
Sister Mary Joseph nodule

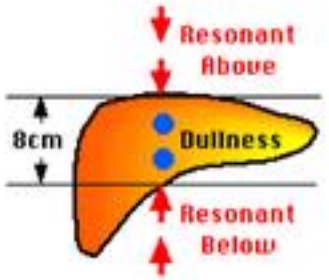


Elevated JVP



- Palpation of abdomen:
  1. Tenderness
  2. Masses
  3. Organomegaly





Midclavicular line

- Percussion
  1. Shifting dullness
  2. Fluid wave test
  3. Puddle sign

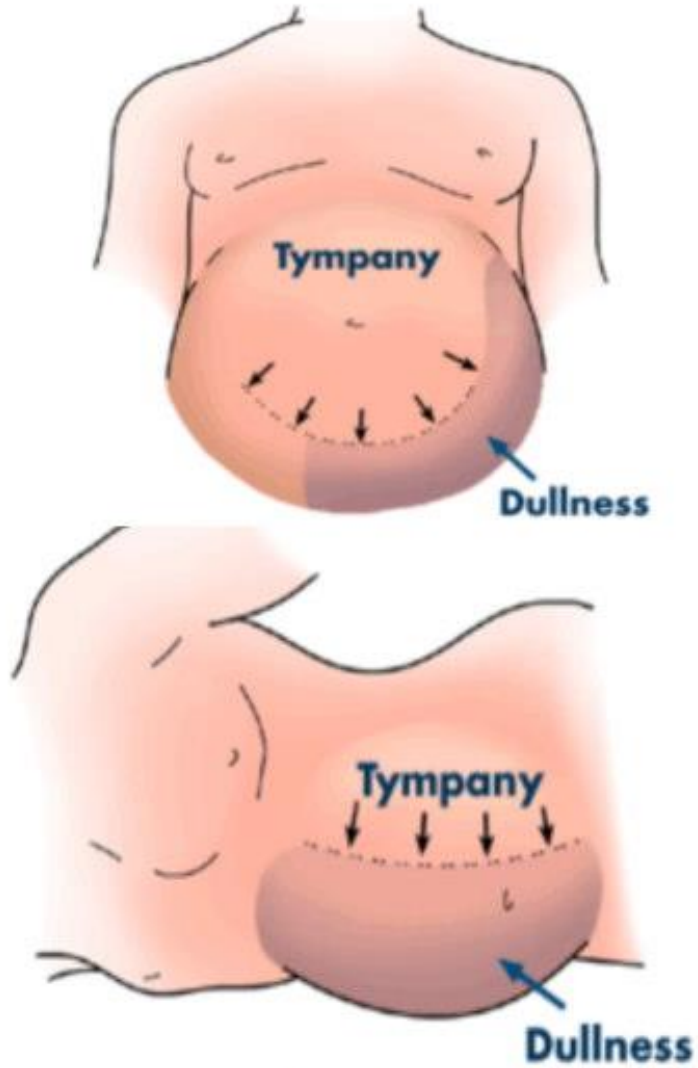
	LR
Flank dullness	NS
Puddle sign	1.2
Shifting dullness	2.3
Fluid wave	5.0

Add edema and LR increases

Ascites

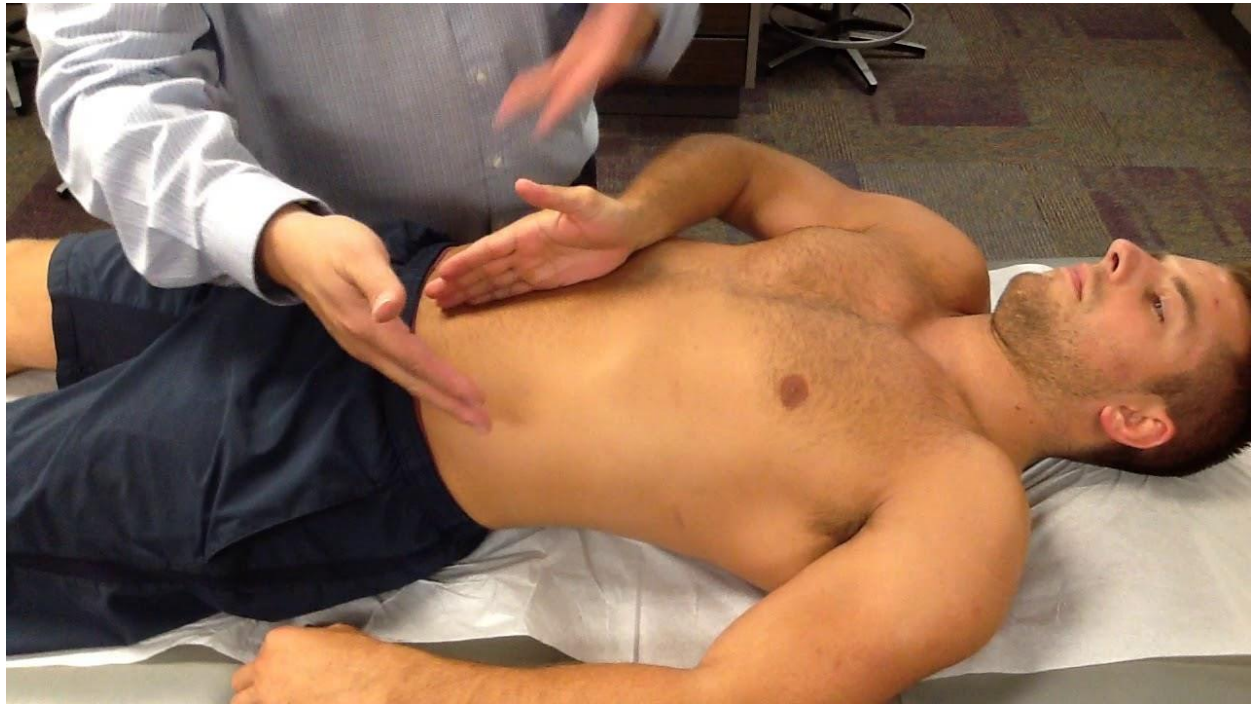
Shifting Dullness

1. This maneuver is performed with the patient supine.
2. Percuss across the abdomen as for flank dullness, with the point of transition from tympany to dullness noted.
3. The patient then is rolled on his/her side away from the examiner, and percussion from the umbilicus to flank area is repeated.
4. Positive test: When ascites is present, the area of dullness will shift to the dependent site. The area of tympany will shift toward the top.

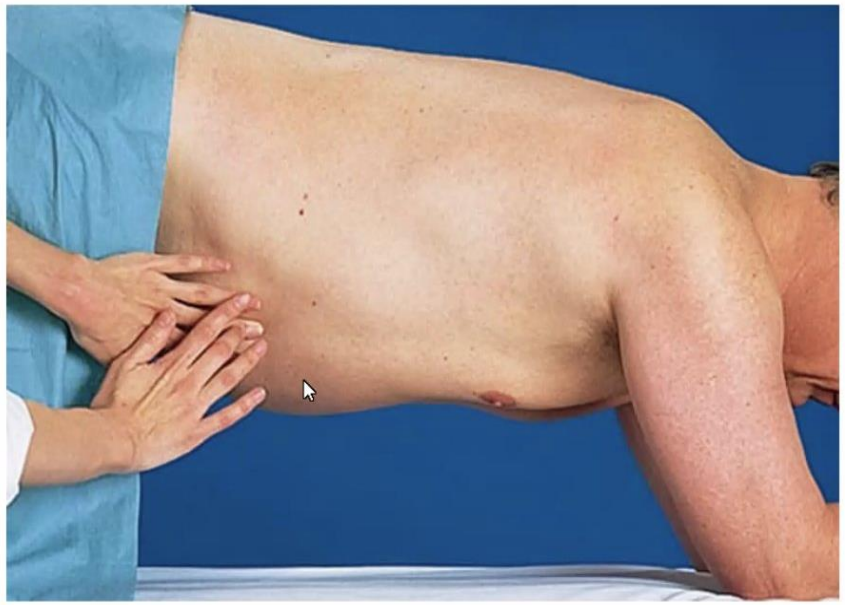


Note: The shift in zone of tympany with position change will usually be at least 3 cm when ascites is present.

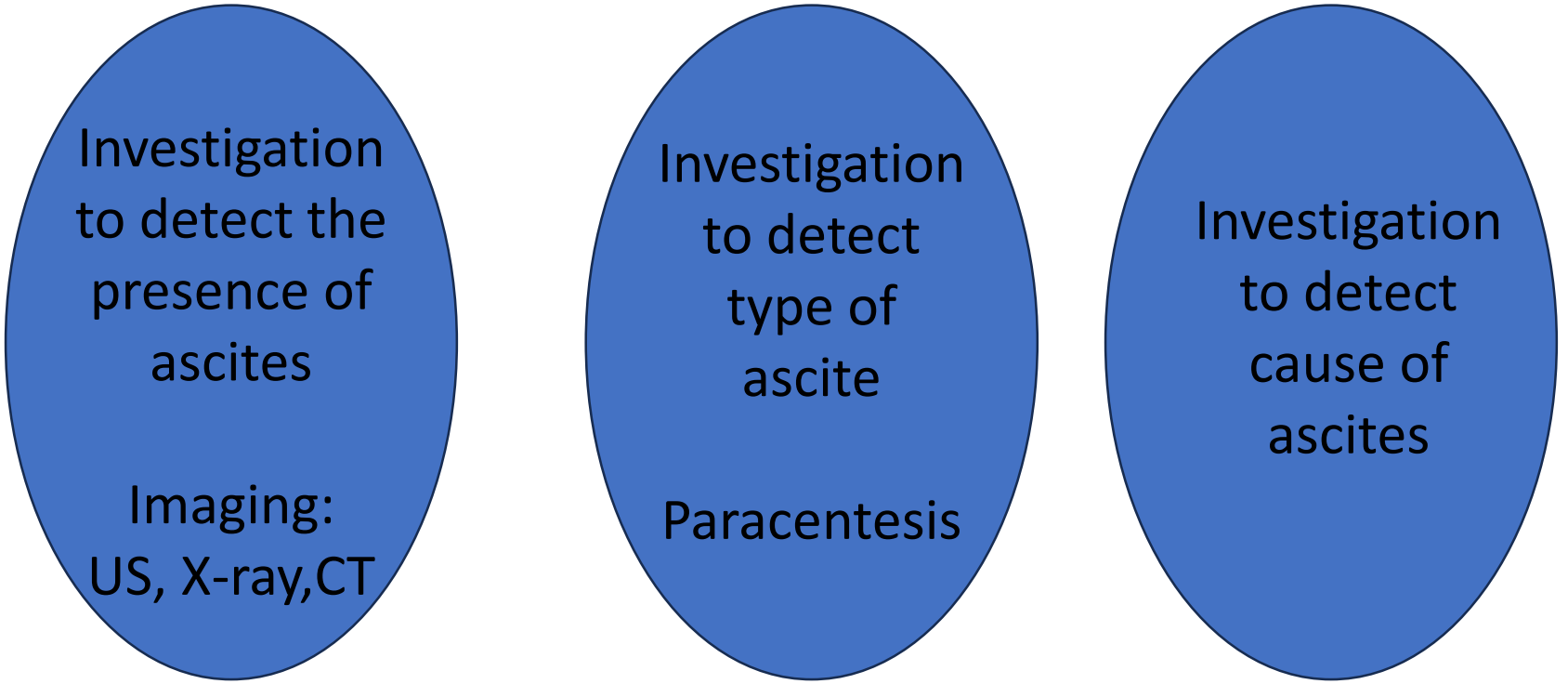
- Fluid wave test
    1. Ask patient to place their hand over the midline
    2. Wave produced by tapping one side of the abdomen in a patient in supine position
    3. This wave will be transmitted to the other side via ascitic fluid.



- Puddle sign
  1. Patient in prone for 3-5 minutes
  2. Put the stethoscope on the most dependent area
  3. Tap abdominal by your finger until sound detected, sound increases as you go toward the opposite flank
  4. 120 ml of fluid is required



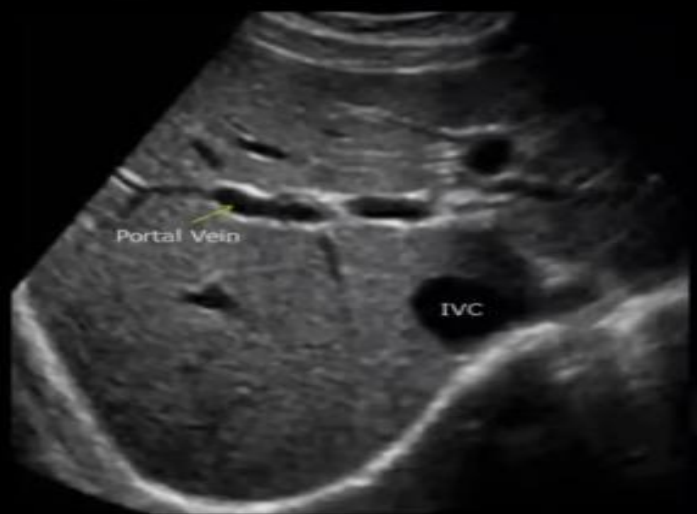
Investigations



Imaging

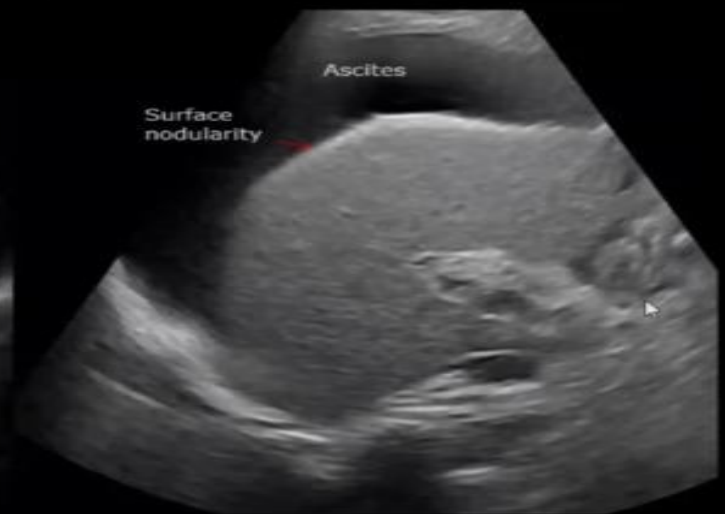
1. **Ultrasonography** is the most sensitive means of detecting ascites, particularly in the obese and those with small volumes of fluid 5-10 mL.
2. **Chest x-ray** :Pleural effusions are found in about 10% of patients, usually on the right side (hepatic hydrothorax)
3. **Computed tomography (CT) scanning**:

Transverse View



Normal Right Lobe

Transverse View



Cirrhosis

- Surface nodularity
- Shrunken liver
- Coarse echotexture

Paracentesis

Defined as aspiration of ascites fluid

- Gross appearance
- SAAG
- Cell count
- Gram stain and culture
- Cytology and amylase

It is indicated in all patients with clinically apparent new-onset ascites

Gross Appearance of Ascitic Fluid

Color	Appearance
Translucent or yellow	Normal/sterile
Brown	Hyperbilirubinemia GB or biliary perforation
Cloudy or turbid	Infection
Pink or blood tinged	Mild Trauma
Grossly bloody	traumatic tap; Malignancy Abdominal trauma
Milky ("chylous")	Cirrhosis Thoracic duct injury Lymphoma

1. Serum ascite albumin gradient(SAAG)

2. Cell count:

-An ascitic fluid with **high RBCs**:

Malignancy

TB

Pancreatitis

-An ascitic fluid with **high WBCs** :

PMN > 250/mm3 suggests SBP

Lymphocytes > 70% suggests TB peritonitis

3. Culture & Sensitivity test:

Ziehl-Neelsen stain (ZN) for TB (detects acid-fast organisms)

Lowenstein-Jensen medium or BACTEC : selective medium for Mycobacteria

4. Amylase to exclude pancreatic ascites.

5. Cytology for malignant ascites.

6.Total protein

Table 1-14: Causes of Ascites and Associated Findings

Protein and Albumin in Ascites

Causes	SAAG	Ascites T. protein
Cirrhosis, liver failure, Budd-Chiari syndrome, myxedema, and SBP	> 1.1	< 2.5
Right heart failure	> 1.1	> 2.5
TB peritonitis, bacterial/fungal peritonitis, nephrotic syndrome, pancreatitis, and peritoneal carcinomatosis	< 1.1	> 2.5

Features of transudate and exudative ascites

	Transudate	Exudate
Proteins	< 2.5g/dl	> 2.5g/dl
Specific Gravity	< 1016	> 1016
LDH	< 200 IU/L	> 200 IU/L
Cells (WBCs)	< 1000/cmm	>1000/cmm
SAAG	>= 1.1	< 1.1

A-Features of Spontaneous Bacterial Peritonitis:

The ascitic polymorph count exceeds 250 cells / cmm.

The ascitic fluid culture is positive for organisms.

The infecting organisms are usually: gram negative organisms.

B- Features of Hemorrhagic ascites:

The fluid is bloody & contains many RBCs.

C-Features of Chylous ascites:

The fluid is milky white & contains many fat, clears on addition of ether & stains orange with Sudan III

D-Features of malignant ascites:

Features of exudate: massive, hemorrhagic, may contain malignant cells.Rapidly reaccumulating after tapping

E-An ascites fluid with low glucose

suggest malignant disease or tuberculosis.

Investigation to detect cause of ascites

For liver cirrhosis e.g. liver function tests

For heart failure e.g. echocardiography.

For TB & malignancy e.g. laparoscopy & biopsy.

COMPLICATIONS

1. Renal failure: It can be pre-renal and due to vasodilatation from sepsis and diuretic therapy,or due to hepatorenal syndrome.
2. Hepatorenal syndrome (HRS)
3. Spontaneous bacterial peritonitis (SBP)
4. Pleural effusion.
5. Weight loss and protein malnutrition.
6. Mental confusion, change in the level of alertness, or coma (hepatic encephalopathy).
7. Bleeding from the upper or lower intestine.

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
- The kidney failure of HRS is a consequence of these changes in blood flow, rather than direct damage to the kidney.

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 without treatment. 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 E.coli, then S. pneumoniae, then Klebsiella.

Clinical features: abdominal pain, rebound tenderness, absent bowel sounds and fever.

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 cirrhotics, it can be confused with SBP.

Management

Bailsan Alhroot

Dietary salt restriction

- Dietary salt restriction alone can create a negative sodium balance in 10% of patients. Sodium restriction has been associated with lower diuretic requirement, faster resolution of ascites, and shorter hospitalization.
- Dietary salt should be restricted to ~90 mmol/day (5.2 g) salt by adopting a no-added salt diet and avoidance of pre-prepared foodstuffs.
- Drugs : - Antiacids (containing high amounts of sodium) ,- NSAIDs (promoting sodium retention) , -and all prostaglandin medications (decrease urinary Na+ excretion) ,,, MUST BE AVOIDED .

water restriction

- Water intake restriction (1.0-1.5 L/day) is necessary **only** if plasma sodium falls below (125 mmol/L)

Diuretics : Spironolactone

- Spironolactone is an aldosterone antagonist, acting mainly on the distal tubules to increase natriuresis and conserve potassium.
- Spironolactone is the drug of choice in the initial treatment of ascites due to cirrhosis.The initial daily dose of 100 mg may have to be progressively increased up to 400 mg to achieve adequate natriuresis. There is a lag of 3–5 days between the beginning of spironolactone treatment and the onset of the natriuretic effect.
- Most frequent side effects of spironolactone in cirrhotics are those related to its antiandrogenic activity, such as decreased libido, impotence, and gynaecomastia in men and menstrual irregularity in women
- Hyperkalaemia is a significant complication that frequently limits the use of spironolactone in the treatment of ascites.

Frusemide

- Frusemide is a loop diuretic which causes marked natriuresis and diuresis in normal subjects. It is generally used as an adjunct to spironolactone treatment because of its low efficacy when used alone in cirrhosis.
- The initial dose of frusemide is 40 mg/day and it is generally increased every 2–3 days up to a dose not exceeding 160 mg/day.
- High doses of frusemide are associated with severe electrolyte disturbance and metabolic alkalosis.
- Simultaneous administration of frusemide and spironolactone increases the natriuretic effect.

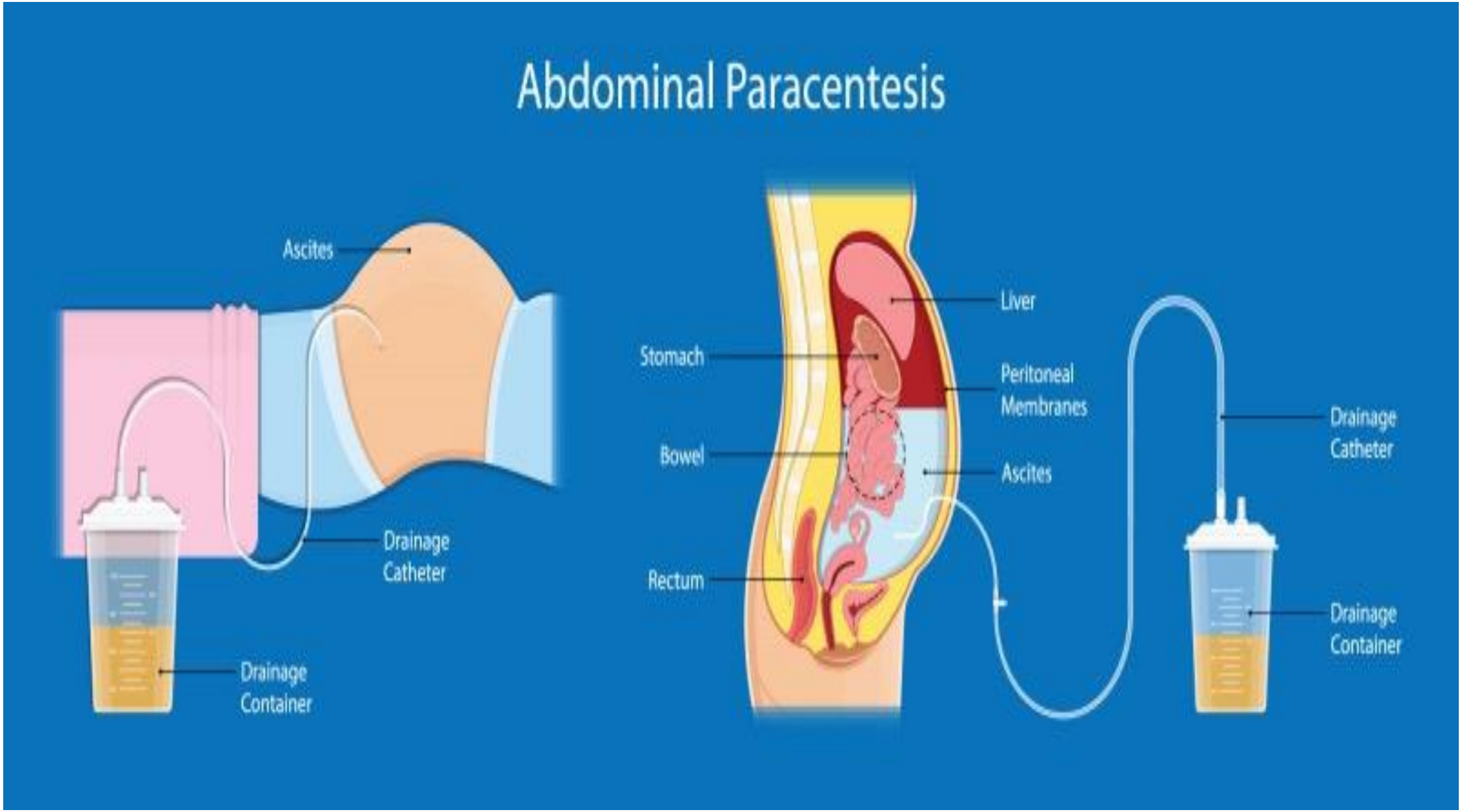
- Diuresis is improved by bed rest because horizontal position increases renal blood flow
- Body weight during the use of diuretics should be measured in a regular basis to make sure it not fall more than (1 kg / day) to avoid excessive fluid depletion

- **In patients**

- 1.Not responding to [100- 400 mg spironolactone / Up to 160 mg furosemide] ... OR
- 2.Not able to tolerate these doses due to hyponatremia or renal impairment
- They are considered to have (refractory ascites) or (diuretic-resistant ascites) ,**other methods of treatment should be used**

Therapeutic paracentesis

- removal of 1-5 liters of fluid to reduce intra-abdominal pressure and relieve the associated dyspnea, abdominal pain, and early satiety .
- With large volume paracentesis , IV colloid (humanalbumin) of (6-8 g / liter of ascites removed) should be administered to support the circulation and prevent hypovolemia and renal dysfunction .
- It is okay to do daily paracenteses during the initial treatment of recent-onset ascites or with severe refractory ascites if the patient's renal function is normal and there is: No GI bleeding , No sepsis , No portosystemic encephalopathy (PSE)

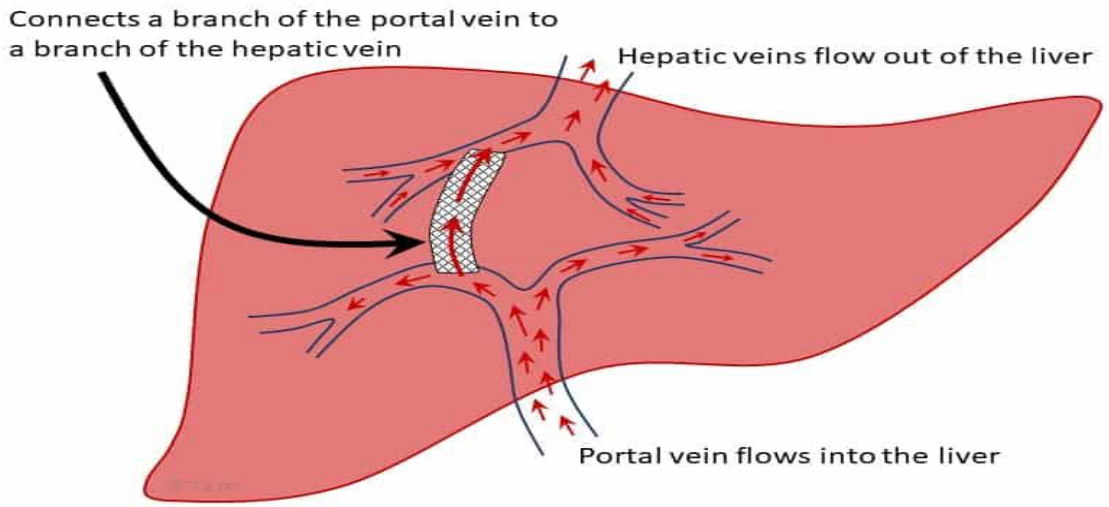

Abdominal Paracentesis
Ascites
Drainage Catheter
Drainage Container
Liver
Stomach
Peritoneal Membranes
Bowel
Ascites
Rectum
Drainage Catheter
Drainage Container

Transjugular intrahepatic portosystemic shunt (TIPSS)

- As elevated portal pressure is one of the main factors contributing to the pathogenesis of ascites, it is not surprising that TIPS is a highly effective treatment for refractory ascites.

- Transjugualr: stent inserted via the internal jugular vein
- Intrahepatic: stent placed within the liver
- Portosystemic: stent connects the portal and systemiccirculations
- Stent Shunt

- It reduces portal pressure by shunting blood to systemic circulation
- A shunt that runs percutaneously from the peritoneum to the internal jugular vein in the neck to allow the ascetic fluid to pass back to systemic circulation
- Complications include (infection, disseminated intravascular coagulopathy , superior vena caval thrombosis , pulmonary edema, bleeding from esophagealvarices)
-



Spontaneous bacterial peritonitis

- In patients with ascitic fluid neutrophil count of >250 cells/mm3, empiric antibiotic therapy should be started
- Third generation cephalosporins such as cefotaxime have been most extensively studied in the treatment of SBP and have been shown to be effective.
- Patients with SBP and signs of developing renal impairment should be given albumin at 1.5 g albumin/kg in the first six hours followed by 1 g/kg on day 3.

- Patients recovering from one episode of SBP should receive prophylaxis with continuous oral norfloxacin 400 mg/day (or ciprofloxacin at 500 mg once daily).
- Liver transplantation should be considered in patients with cirrhotic ascites.
- All patients with SBP should be considered for referral for liver transplantation

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