APPROACH TO PATIENT WITH ASCITES

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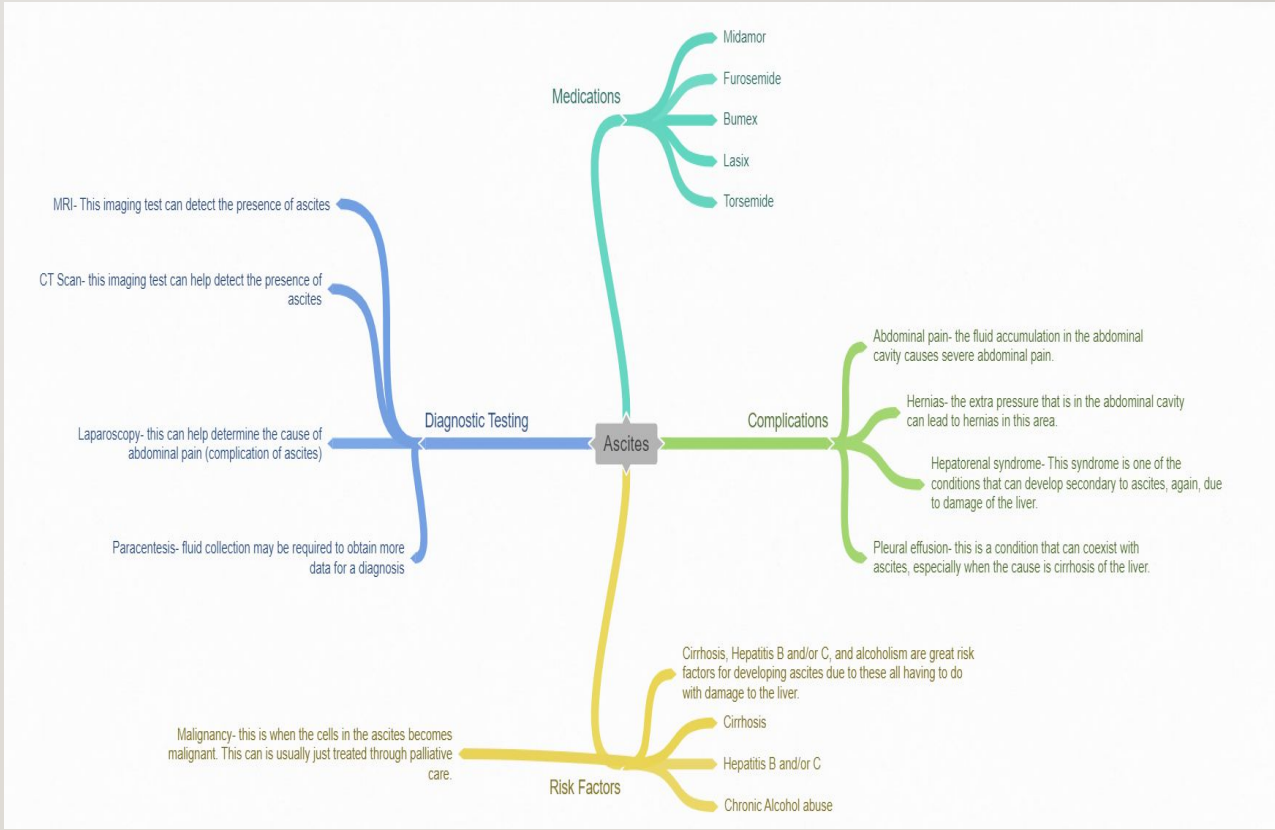
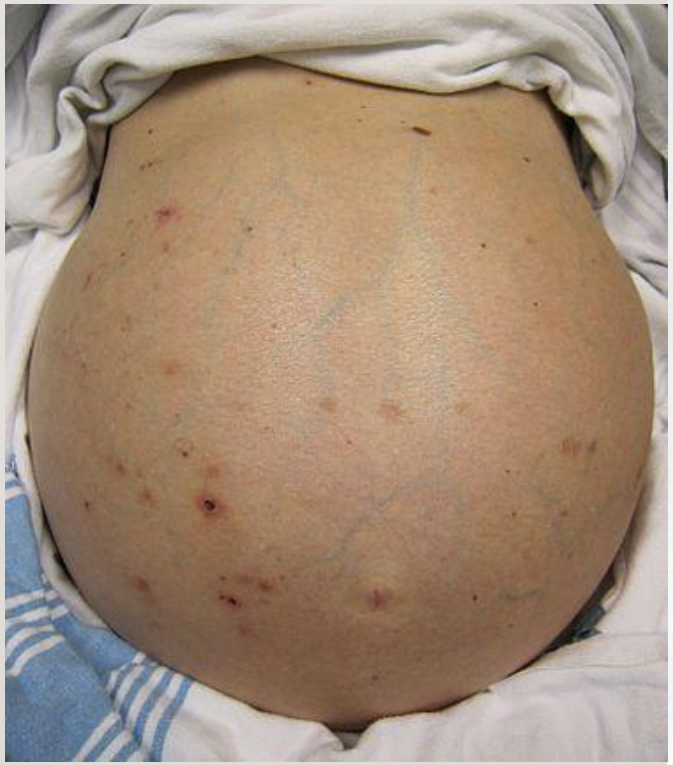
OBJECTIVES

- Definition
- Etiology & Pathophysiology
- History & Physical Examination
- Investigations
- Differential Diagnosis
- Treatment
- Complications



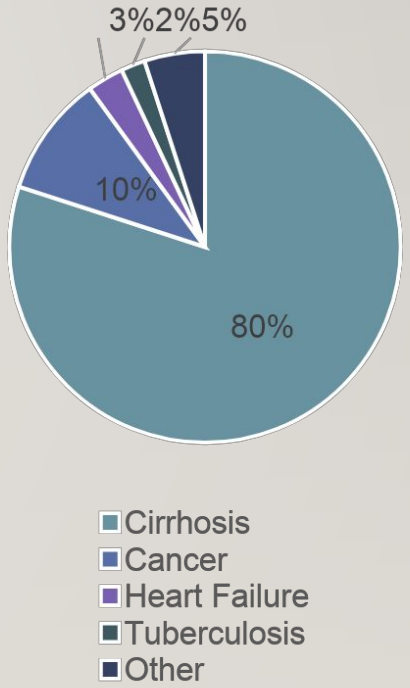
HISTORICAL BACKGROUND & DEFINITION

- The word "Ascites" came from the Greek "*askos*" meaning "wineskin" since it resemble it's shape.
- Medically, it's defined as an abnormal collection of fluid in the peritoneal cavity (more than 25 ml)



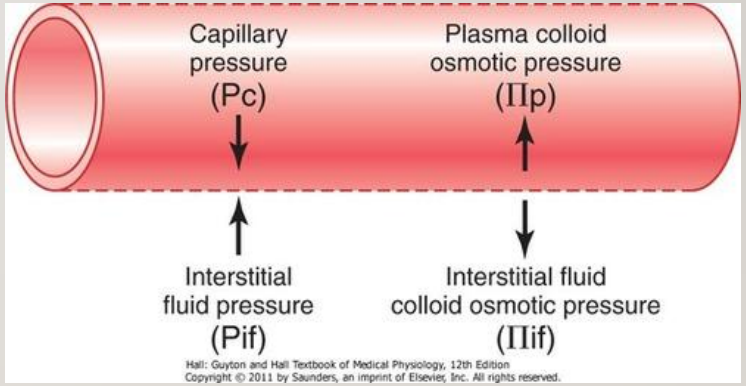
ETIOLOGY

- The most common cause for ascites is *cirrhosis*. in up to 80% of cases.
- Cancer in 10% of cases and the most common type is peritoneal carcinomatosis.
- Cardiac ascites in case of CHF in 3%.
- TB ascites in 2% of cases.
- Other causes include:
  - •Nephrotic Syndrome
  - •Severe malnutrition
  - •Pancreatic Ascites
  - •Budd-Chiari Syndrome
  - •Chronic Alcoholic Abuse
  - Hepatits B
  - Hepatitis C



ETIOLOGY & PATHOPHYSIOLOGY

- The underlying mechanisms behind ascites are determined by the cause of the disease or the etiology. And those can be divided into four groups:



I. Conditions that increases portal vein pressure (⬆Pc)
II. Conditions that lower the protein content in the blood (⬇ ***Πp***)
III. Conditions that causes chronic peritonitis
IV. Conditions that obstruct the lymph flow

ETIOLOGY & PATHOPHYSIOLOGY

I. Conditions that increases portal vein pressure

Those can be further divided depending on site of pathology into:

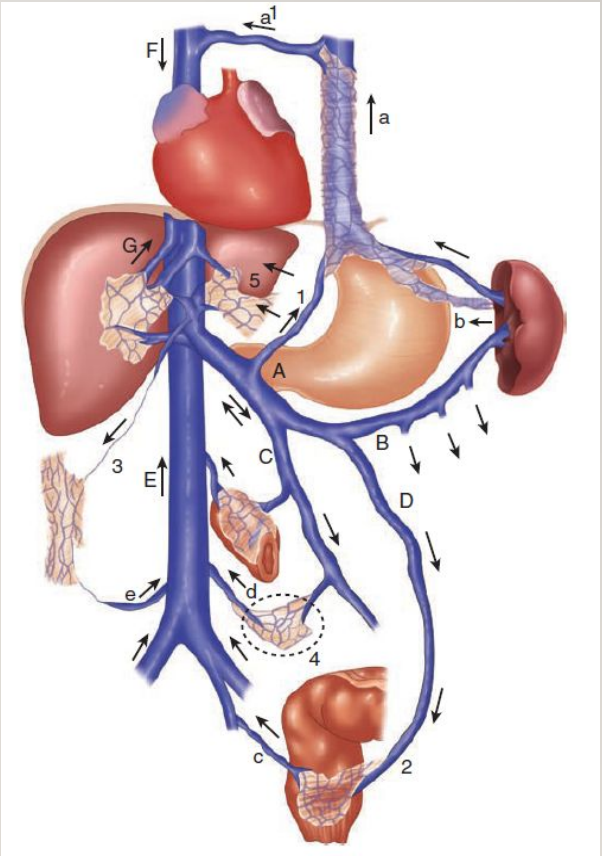
a) Pre-hepatic: *Portal vein thrombosis*

b) Hepatic: *Cirrhosis*

c) Post-hepatic: *Budd-Chiari Syndrome*

d) Cardiac: *Constrictive Pericardritis, Right-sided HF* by either:

mitral stenosis, tricuspid regurgitation, pulmonary hypertension (**cardiac ascites**)



- In those diseases, the increased hydrostatic pressure □ NO release □ splanchnic arterial dilatation & low BP □ activation of RAAS □ Na+ & H2O retention

ETIOLOGY & PATHOPHYSIOLOGY

II. Hypoalbuminemia

a) Reduced protein intake (Malnutrition) : *Kwashiorkor*

b) Reduced protein absorption: in Protein-Losing Enteropathies like *IBD*.

c) Increases protein excretion: *Nephrotic Syndrome*

d) Cachexia in wasting disease: *malignancy* in general

- In those diseases the hypoalbuminemia □ decreased venous osmotic colloid pressure □ more fluid leaks out into the peritoneal cavity

ETIOLOGY & PATHOPHYSIOLOGY

III. Conditions causing Chronic Peritonitis

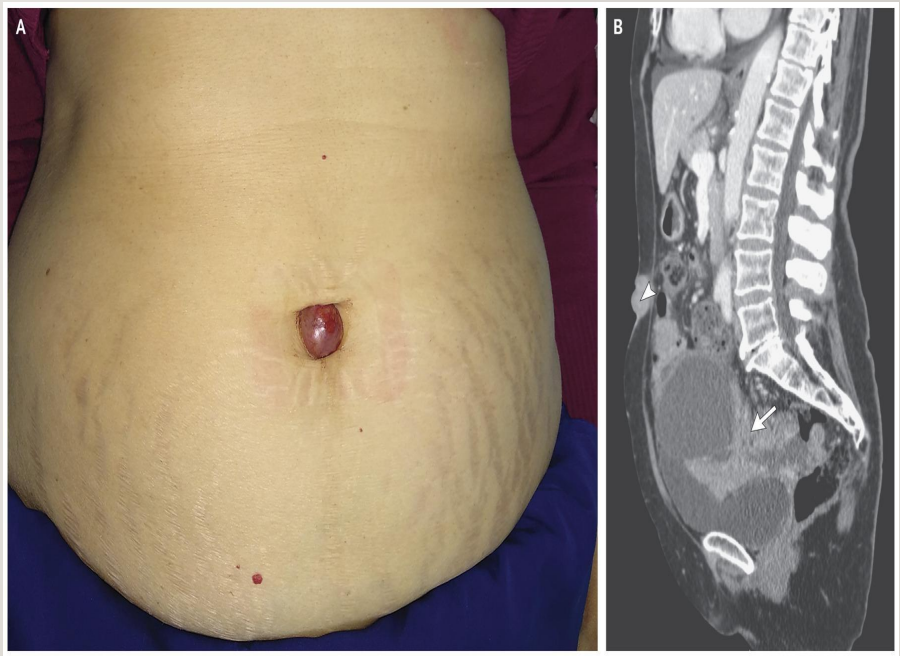
a) Physical cause: *Post-irradiation, Starch granuloma*

b) Infection: *Tuberculosis (**TB ascites**)*

c) Neoplasms: *Peritoneal carcinomatosis, pseudomyxoma peritonei*. **(Malginant ascites)**

- In those disease there is an increased capillary permeability (more proteins leak out) and lymphatic obstruction (proteins don't return back into circulation after leaking out)

IV. Conditions obstructing lymphatic flow (**Chylous Ascites**)



ETIOLOGY & PATHOPHYSIOLOGY

- Traditionally, ascites is divided into 2 types; **transudative or exudative**. This classification is based on the amount of protein found in the fluid. A more useful system has been developed based on the amount of albumin in the ascitic fluid compared to the serum albumin (albumin measured in the blood).

Summary box 61.12

Causes of ascites

Transudates (protein <25 g/L)
- Low plasma protein concentrations
  - Malnutrition
  - Nephrotic syndrome
  - Protein-losing enteropathy
- High central venous pressure
  - Congestive cardiac failure
- Portal hypertension
  - Portal vein thrombosis
  - Cirrhosis

Exudates (protein >25 g/L)
- Tuberculous peritonitis
- Peritoneal malignancy
- Budd–Chiari syndrome (hepatic vein occlusion or thrombosis)
- Pancreatic ascites
- Chylous ascites
- Meigs' syndrome

HISTORY

- When you approach a patient with ascites, try to ask the questions that will make you figure out the cause of the ascites
- Ascites usually comes as a non-painful condition, so following the SOCRATES mnemonic may not be practical.
- For the rest of the parts of the history try to follow the normal routine sequence of questions: Patient Profile, Chief Complain, HPI, System Review, Drug History, Family History and Social History

HISTORY

- **Patient Profile:** (Name , Age , Gender , Marital Status , Occupation , Address m Rout of Admission)
  - White ethnic group has higher incidence of peritoneal carcinomatosis that the Blacks

- **Chief Complaint:** What brought you to the hospital today?
  - Patients with ascites often state that they have recently noticed an increase in their abdominal girth.
- **History of Presenting Illness:**
  - Patients should be asked about change in abdominal girth and the rapidity of onset of weight gain.
  - Abdominal obesity can be masquerade as ascites .
  - Obesity usually develops overtime while ascites accumulates more rapidly

HISTORY

- **Systematic Review :**
  - This could be the most valuable part of the history since it covers the various etiologies of ascites

Symptoms suggesting Cirrhosis

- Because most cases of ascites are due to liver disease, patients with ascites should be asked about risk factors for liver disease. These include the following:

a) Long-term heavy alcohol use

b) Chronic Viral Hepatitis or jaundice

c) IV drug use

HISTORY

d) Multiple sexual partners

e) Homosexual activity with a male partner, or heterosexual activity with a bisexual male

f) Transfusion with blood not tested for hepatitis virus:

g) Tattoos

h) Living or birth in an area endemic for hepatitis

i) Alcoholic hepatitis can cause ascites without cirrhosis

j) Obesity , hypercholesterolemia and type 2 diabetes mellitus are causes of **nonalcoholic steatohepatitis** which can lead to cirrhosis which could lead to Ascites

HISTORY

Symptoms suggesting Cardiac Ascites

- Dyspnea
- Orthopnea
- Peripheral Edema
- Coughing, also in (Tuberculosis)

Symptoms suggesting Nephrotic Syndrome

- Waking up with buffy eyes (peri-orbital edema)

Symptoms Suggesting Malignancy

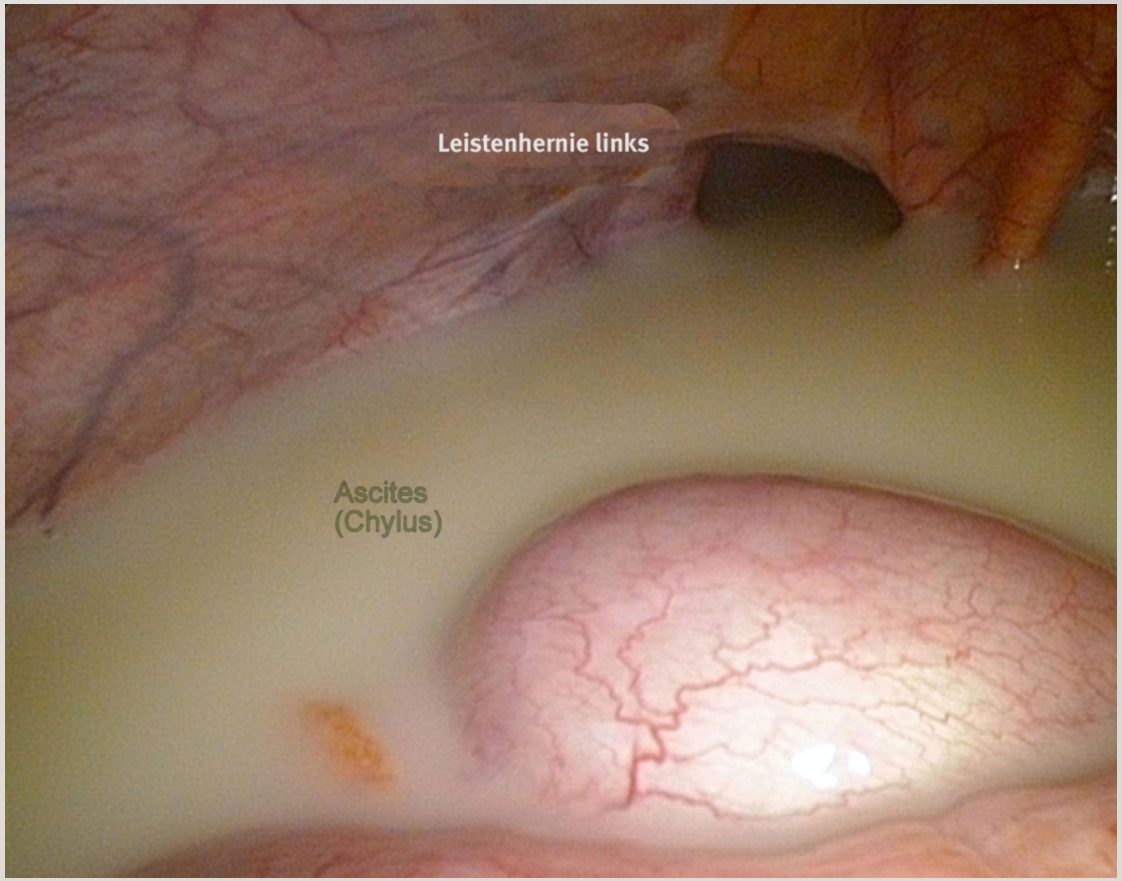
- Weight loss
- Loss of Appetite
- Prior History of Cancer
- Associated abdominal pain
- History of long-standing Cirrhosis followed by abrupt development of ascites , Consider Hepatocellular Carcinoma

- A history of acute pancreatitis may be found in pancreatic ascites and biliary ascites occurs following biliary surgery

HISTORY

Symptoms suggesting Chylous Ascites

- Diarrhea
- Steatorrhea
- Edema
- Enlarged lymph nodes
- Early satiety
- Fevers, and night sweats



HISTORY

History of Tuberculosis

- More likely in Patients of Alcoholisim , Poor nutritional status

- **Drug History** : Certain Medications may cause liver damage which could lead to Ascites

methotrexate, methyldopa, amiodarone

- **Family history** of Liver diseases Should be obtained (Liver cirrhosis , Wilson Disease)

Social History :

- **Alcohol Consumption** (Quantity and Duration)
- Alcoholic hepatitis can cause ascites without cirrhosis
- Ask About **Recent pregnancy and oral Contraceptive Use** (Budd-Chiari Syndrome)

RISK FACTORS FOR VIRAL HEPATITIS

- IV Drug use
- Needle sharing
- Blood Transfusions
- Tattoos
- Acupuncture
- Sexual History
- Travel History

PHYSICAL EXAMINATION

- Ascites can usually be recognized clinically only when the amount of fluid present exceeds 1.5 L depending on body habitus: like in obese individuals a greater quantity than this is necessary before there is clear evidence
- In women, ascites must be differentiated from an enormous ovarian cyst.
- The physical examination starts by the minute you walk into the room and general examination is of significant importance

PHYSICAL EXAMINATION

- Before you start the examination, you need to introduce yourself ,maintain privacy ,ask for a permission , maintain hygiene and ask for a chaperon .
- Check for vital signs :

1. Blood pressure
2. Pulse rate
3. Respiratory rate
4. Temperature

PHYSICAL EXAMINATION

- General Examination:
  - With each person you have to be aware of signs of liver disease (palmar erythema, spider naevi, splenomegaly)
  - signs of heart failure (peripheral oedema ,jugular venous distention ,third heart sound)
  - malignancy (lymphadenopathy in Virchow node).

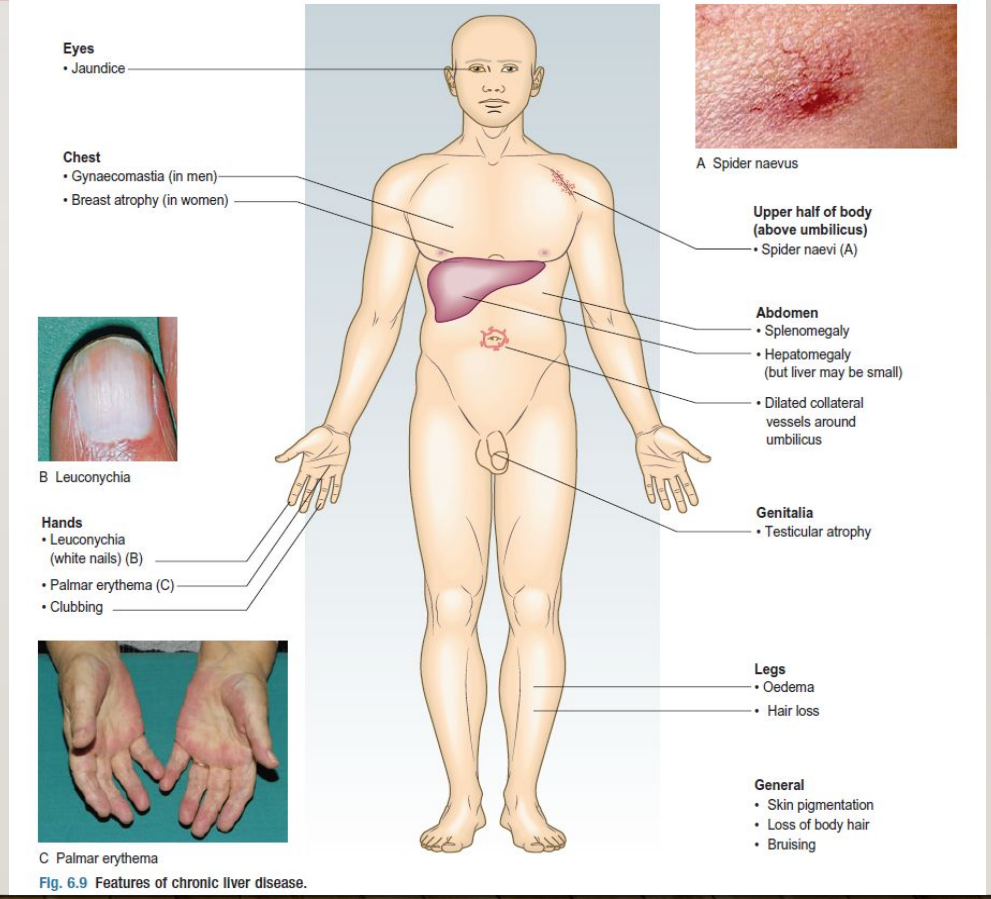


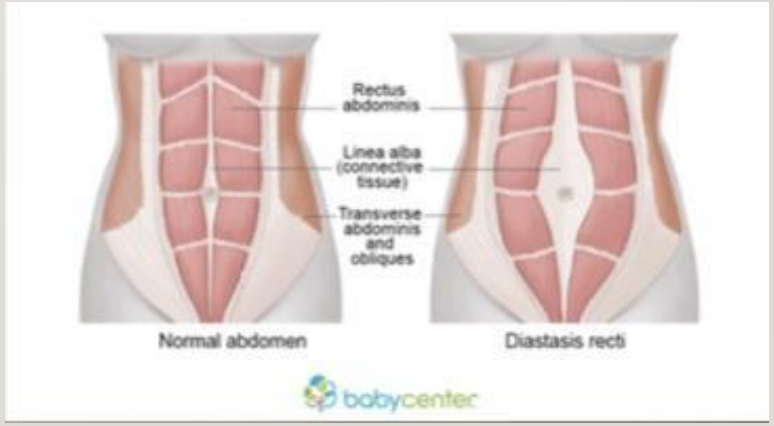
Fig. 6.9 Features of chronic liver disease.

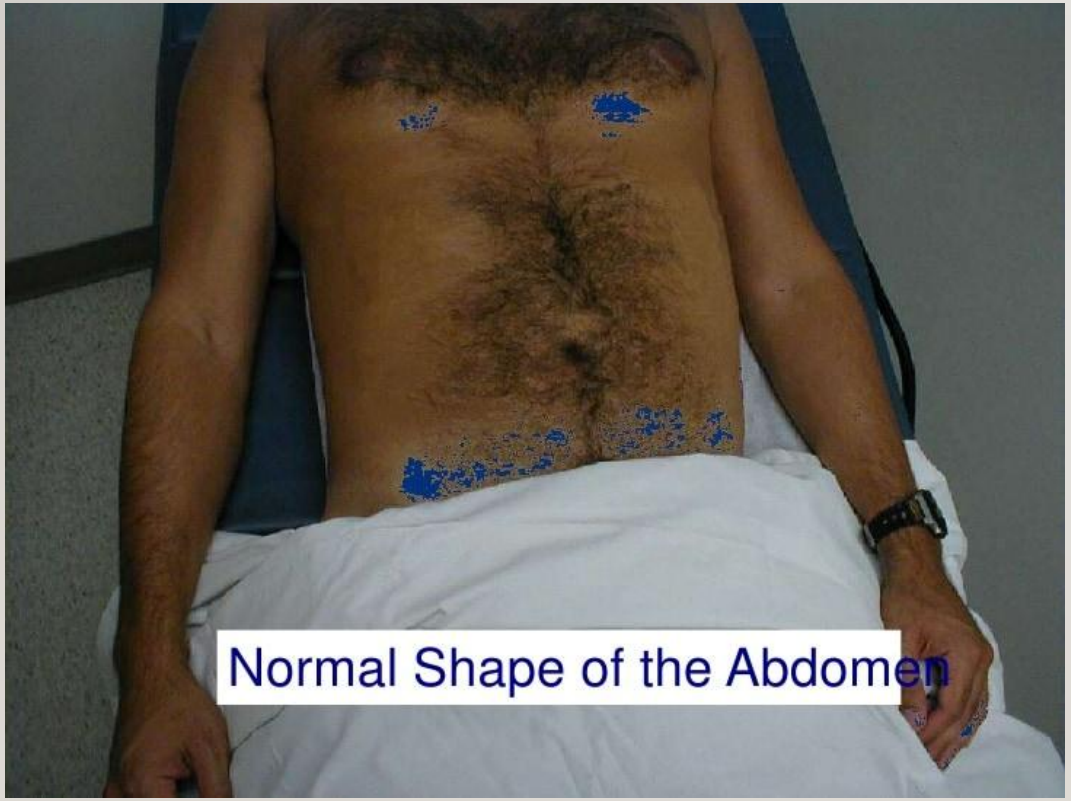
PHYSICAL EXAMINATION

- For the abdomen exam remember:
  - Inspection
  - Palpation
  - Percussion
  - Auscultation

INSPECTION:

- You should start from the foot of the bed by describing how the patient looks like ; ill or well , oriented or not and if there is any drainage or cannula presented.
- You should expose the abdomen from the nipples to the middle of the thigh .
- Normally the abdomen will be flat or slightly scaphoid and symmetrical , it moves with respiration and the umbilicus is usually inverted . But in ascites we see:
- The abdomen is distended evenly with fullness of the flanks.
- Umbilicus eversion in ascites in general and Sister Mary Joseph nodule in case of peritoneal carcinomatosis
- Distended abdominal veins (*caput medusae*)
- Skin is stretched and shiny
- Divarication of recti muscle




Normal Shape of the Abdomen

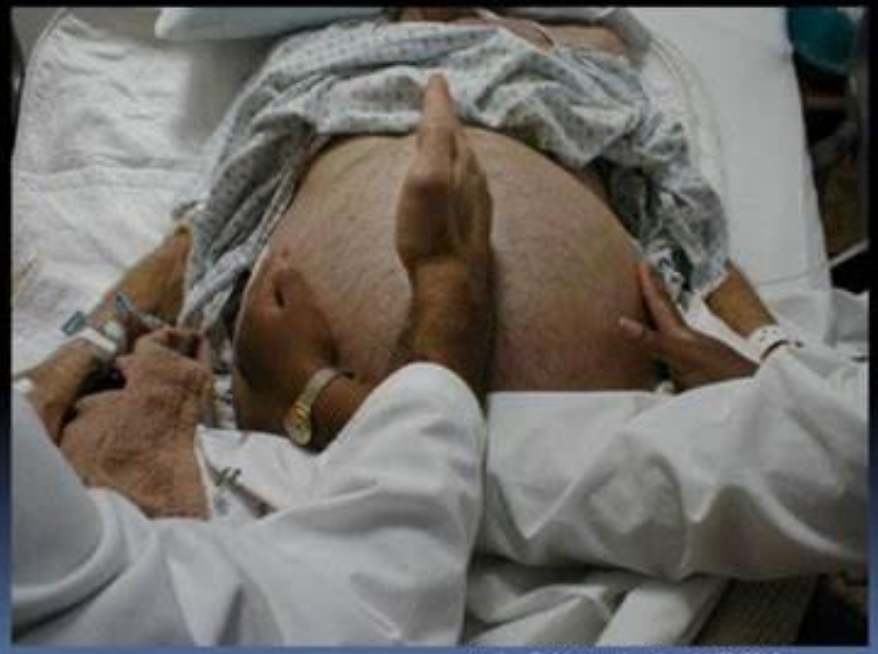
PALPATION :

- Before you do the palpation you need to make sure that your hands are warm and clean .
- You start the palpation by asking the patient if there is any pain to start away from it and then with your right hand start superficial palpation each region of the abdomen to check for any superficial masses or tenderness , and then repeat the same regions in deep palpation and check for any organ enlargement .
- In ascites we need to do the fluid thrill sign .

A FLUID THRILL.

- First you must place the edge of the patient's (or an assistant's) hand on the abdomen at the umbilicus to prevent the vibration waves from being transmitted through the fat in the abdominal wall .
- And then flick one side of the abdomen with the index or middle finger, and feeling the vibrations when they reach the other side of the abdomen with your other hand.
- If you felt the vibration this indicates ascites .
- But when there is small accumulation of fluid this sign is absent .
- Fluid trapped in a cyst , or in the renal pelvis or between adhesions ,will have a fluid thrill

Fluid Thrill



HEPATOMEGALY EXAMINATION

■Place your hand flat on the skin of the right iliac fossa. Point your fingers upwards and your index and middle fingers lateral to the rectus muscle, so that your fingertips lie parallel to the rectus sheath. Keep your hand stationary. Ask the patient to breathe in deeply through the mouth. Feel for the liver edge as it descends on inspiration.

■ Move your hand progressively up the abdomen, 1 cm at a time, between each breath the patient takes, until you reach the costal margin or detect the liver edge. The liver may be enlarged or displaced downwards by hyperinflated lungs

■ If you feel a liver edge, describe:

size

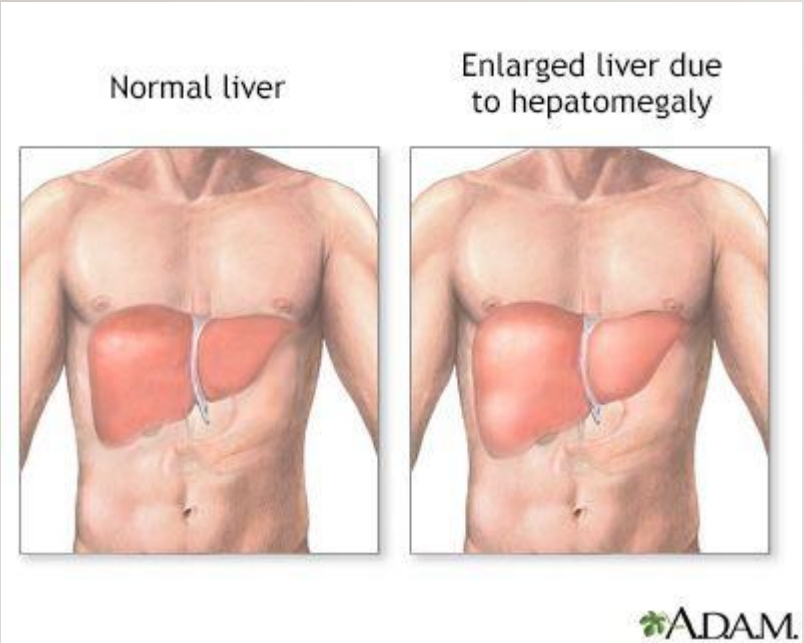
surface: smooth or irregular

edge: smooth or irregular

consistency: soft or hard

tenderness

whether it is pulsatile


Normal liver
Enlarged liver due to hepatomegaly
A.D.A.M.


LIVER

SPLENOMEGALY EXAMINATION

- Place your hand over the patient's umbilicus .with your hand stationary , ask the patient to inhale deeply through the mouth .
- Feel for the splenic edge as It descends on inspiration .
- Move your hand diagonally upwards towards the left hypochondrium (1 cm each breath the patient takes).
- Feel the costal margin along its length .as the position of the spleen tip is variable .
- If you can not feel the splenic edge , palpate with your right hand ,placing your left hand behind the patient's left lower ribs and pulling the ribcage forward .

ABDOMEN

PALPATING THE SPLEEN

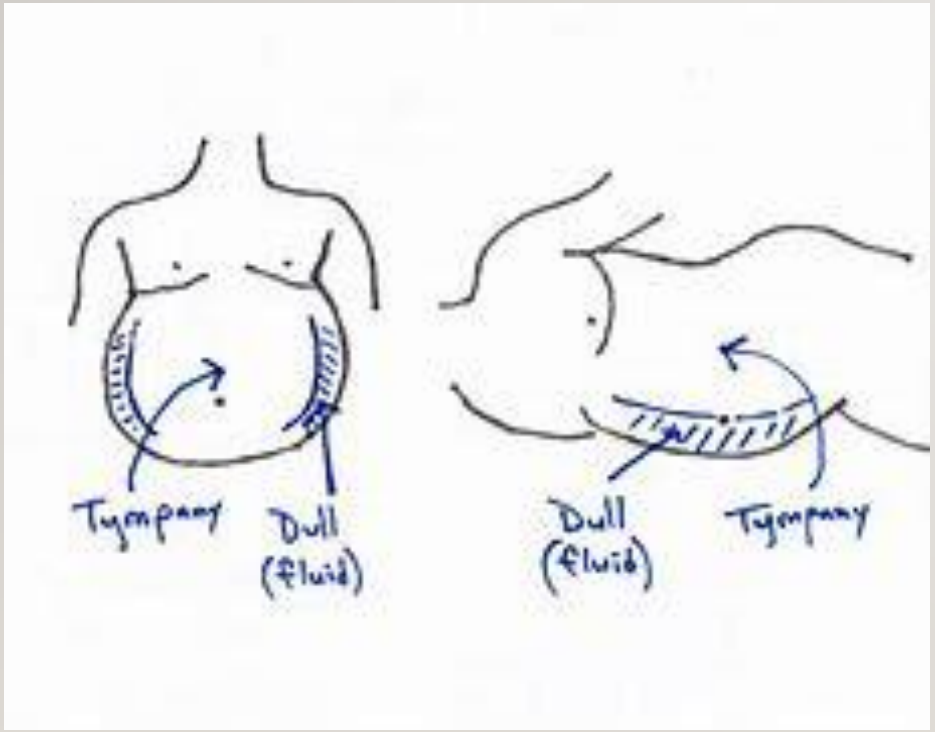
PERCUSSION :

- We do percussion from the midline of the abdomen and then we move to the flanks to note for any changes in the sounds (from tympanic to dull) , incase of ascites we have to do :

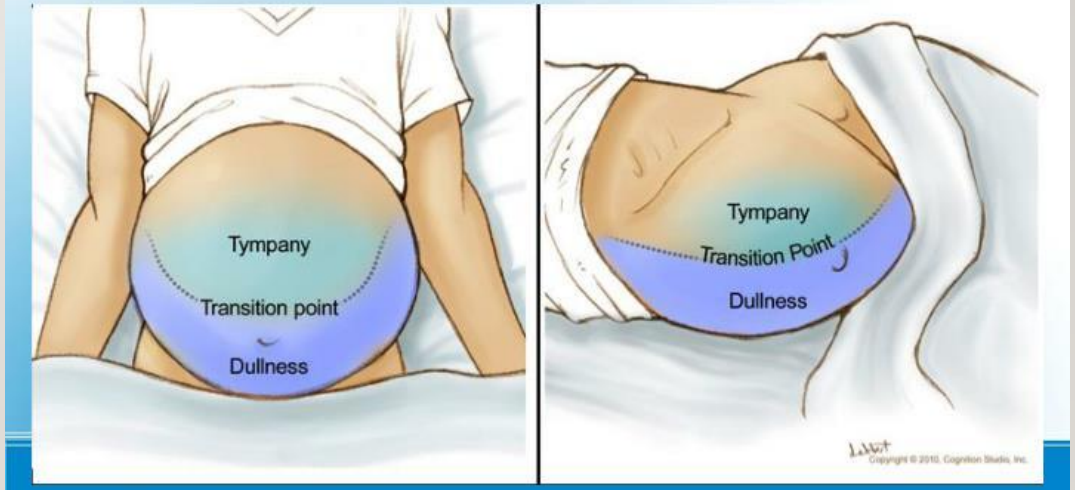
1. Shifting dullness

SHIFTING DULLNESS

- Is a dull area that moves or changes shape when the patient changes position. The dullness of ascites is found in the flanks and across the lower abdomen.
- With the patient supine , percuss from the midline out to flanks , when you notes there is a change from tympanic to dull .
- Keep your finger on the site of dullness in the flank and ask the patient to turn on their opposite side .
- Pause for 10 seconds to allow any ascites to gravitate , then percuss again . If the are of dullness is now tympanic this indicates ascites .
- when there is a very large accumulation of fluid, this sign is absent.



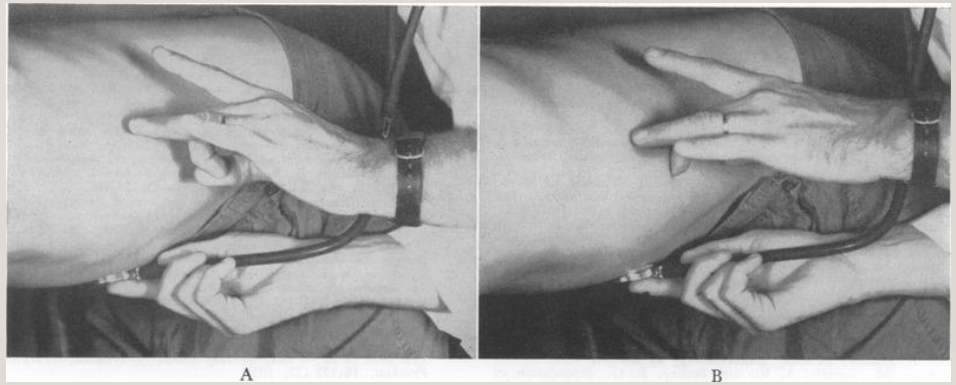
Shifting Dullness



Does the presence of ascites prove that this patient has liver disease?

PUDDLE SIGN

- It is useful for detecting small amounts of ascites (as small as 120 ml , shifting dullness typically requires 500ml) .
- Patient lies prone for 5 minutes then rises onto elbows and knees .Apply stethoscope diaphragm to most dependent part of the abdomen . Examiner repeatedly flicks near flank with finger. Continue to flick at same spot on abdomen then move stethoscope across abdomen away from examiner .A sharp increase in the intensity of the sound picked up by the stethoscope indicates the level of fluid.



Minimum amount of fluid required

Test	Minimum fluid in ml.
Diagnostic tap	10-20
Puddle sign	120
Shifting dullness	500
Fluid thrill	1000-1500
Ultrasound scan	100
CT scan	100

AUSCULTATION :

- First check that your stethoscope is warm .
- Then put your stethoscope in the McBurney point to hear the bowel sounds and wait for 2 minutes before concluding that bowel sounds are absent .
- Listen above the umbilicus over the aorta for arterial bruits
- Listen 2-3 cm above and lateral to the umbilicus for bruits from renal artery stenosis
- Listen above the liver for bruits
- Also in caput medusae you will notice a Venous hum over the distended veins around umbilicus in cirrhosis with portal hypertension

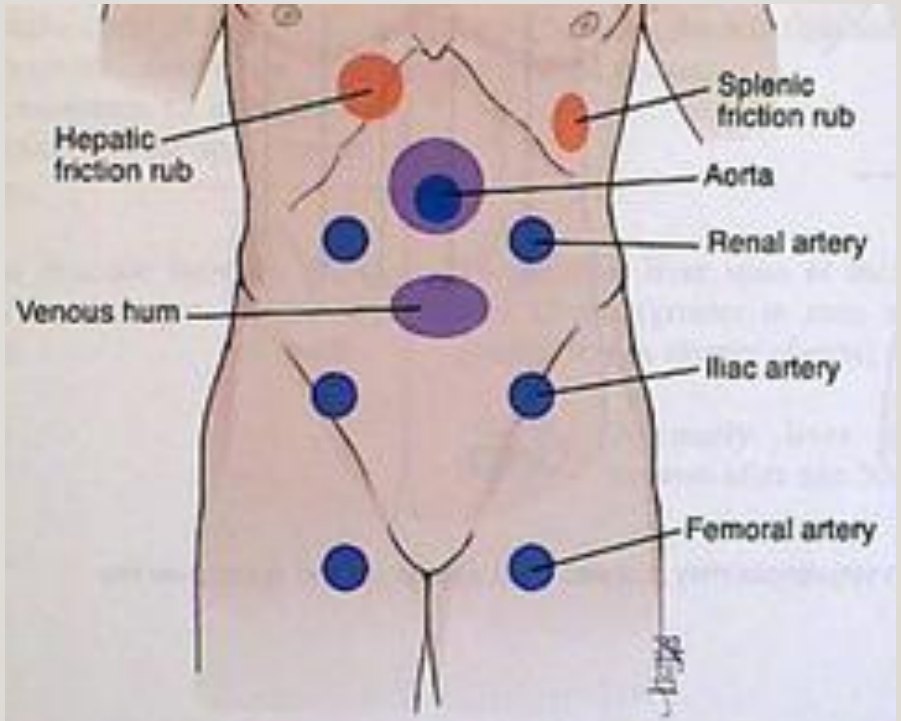
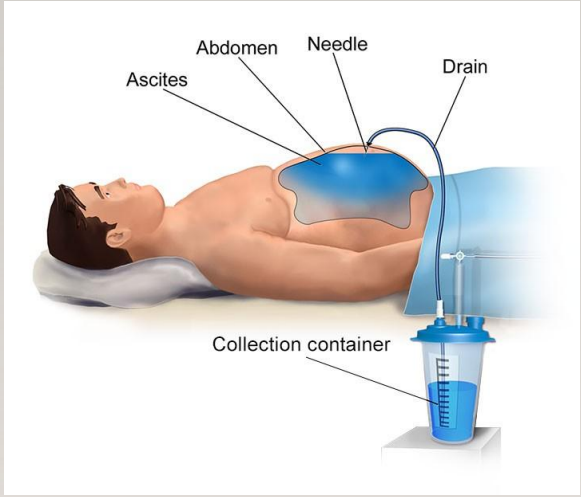


Figure 22-10 Vascular sounds and friction rubs can best be heard over these areas.

DIFFERENTIAL DIAGNOSIS OF ASCITES

- The differential diagnosis of ascites can lead to confusion and inability to exclude its multitude of causes in many patients. Many laboratory chemistry tests may be performed on ascitic fluid, among the most useful is the serum-ascites albumin gradient (SAAG), calculated as: **Serum albumin - Ascites albumin**



DDX OF ASCITES

A. Portal hypertension (the most common) characterized by HIGH ALBUMIN GRADIENT >1.1 g/dl:

1 Cirrhosis

2Alcoholic hepatitis

3-Hepatic congestion

a-Congestive heart failure (transudation from hepatic and peritoneal veins)

b-Constrictive pericarditis (abnormal cardiac filling due to thickening of the pericardium)

c-Hepatic venous outflow obstruction (Budd Chiari Syndrome, PW hepatosplenomegally)

4-Portal vein thrombosis

DDX OF ASCITES

- **<u>Other DDx are characterized by LOW ALBUMIN GRADIENT <1.1 g/dl:</u>**

<u>B. Malignancy</u>

1-Peritoneal carcinomatosis (positive cytology is highly indicative)

2-Hepatocellular carcinoma

3 Mesothelioma

4 Metastatic liver disease

DDX OF ASCITES

C. Infectious

1 Tuberculous peritonitis (DNA of M.tuberculosis in ascitic fluid can be detected by PCR)

2Spontaneous bacterial peritonitis (PMN count ≥ 250 cells/mm3 confirms SBP, also ascitic fluid should be cultured if SBP is clinically suspected)

3 Chlamydia trachomatis (ascites from PID secondary to chlamydia is uncommon)

D. Miscellaneous

1Pancreatitis (amylase ascitic fluid/blood serum concentration ratio of 6 or more is indicative although high levels of amylase have also been detected in patients with malignancy)

2 Nephrotic syndrome (proteinuria + hypoalbuminemia)

DDX OF ASCITES

- If the cause of ascites remains unclear after performing the tests stated above, diagnostic laparoscopy should be considered.

INVESTIGATIONS AND DIAGNOSIS OF ASCITES

- In addition to relevant investigations that may determine the underlying cause, e.g. liver and cardiac function tests, ultrasound and/or CT imaging will determine much smaller quantities of ascites than possible clinically. These will often also diagnose etiology, e.g. carcinomatosis, liver disease.
- Presentation ranges from ascites detected only by imaging methods to a distended, bulging, and sometimes tender abdomen. Percussion of the abdomen may reveal shifting dullness.

INVESTIGATIONS AND DIAGNOSIS OF ASCITES

- Routine diagnostic testing should include

1. SAAG calculation
2. red and white blood cell counts and differential
3. total protein
4. culture
5. Amylase and triglyceride measurement, cytology, and mycobacterial smear/culture can be performed to confirm specific diagnoses.

INVESTIGATIONS AND DIAGNOSIS OF ASCITES

- **SAAG(serum ascites albumin gradient)** is calculated as serum albumin minus the ascites albumin; a gradient > 1.1 indicates portal hypertension-related ascites (97% specificity). A SAAG of <1.1 is found in nephrotic syndrome, peritoneal carcinomatosis, serositis, TB, and biliary or pancreatic ascites.
- Ultrasonography, CT, and MRI are sensitive methods to detect ascites.

INVESTIGATIONS AND DIAGNOSIS OF ASCITES

- **Diagnostic paracentesis** (60 mL) should be performed in the setting of new-onset ascites, suspicion of malignant ascites, or to rule out SBP.
- Ascitic aspiration or tap (below) is now most commonly performed under imaging guidance to minimize the risk of visceral injury.
- The bladder having been emptied, puncture of the peritoneum is carried out under local anesthetic using a moderately sized trocar and cannula. Alternatively, a peritoneal drain may be inserted.
- In cases where the effusion is caused by cardiac failure, the fluid must be evacuated slowly.
- Fluid is sent for microscopy/cytology, culture, including mycobacteria, and analysis of protein content and amylase.

TREATMENT

- GOALS achieve ascites-free status AND maintain it thereafter.
- Treatment of ascites must be gradual to avoid sudden changes in systemic volume status that can precipitate HE, renal failure, or death.
- Treatment of the specific cause is undertaken whenever possible; for example, if portal venous pressure is raised, it may be possible to lower it by treatment of the primary condition.

OPTIONS USED IN TREATMENT

- Bed rest
- Diet :

- Sodium restriction

- Fluid Restriction

- Diuretics
- Paracentesis
- TIPSS (Transjugular intrahepatic portosystemic shunt)
- Shunts
- Transplant

TREATMENT

- **Bed rest :**

Ascites patients are positioned supine since the upright posture activates sodium retaining mechanisms , impairs renal perfusion and sodium excretion

- **Dietary sodium restriction:**

Restrict the intake to 200 mg/day

This method may be helpful ,but diuretics are usually required

TREATMENT

- **Fluid restriction:**

Central hypovolemia stimulates ADH receptors decreases free water clearance dilutional hyponatremia .

no trials to assess effect of water restriction in patients with cirrhosis and dilutional hyponatremia. Restriction may worsen central hypovolemia.

Water restriction isn't the first option, sodium restriction is an appropriate first line

TREATMENT

- **Diuretic therapy:**
- **initiated along with salt restriction**
- **Diuretics should be used with caution and gradually**
- **their use should be discontinued in patients with an increasing serum creatinine level.**
- Weight loss shouldn't be more than 500 g/day to avoid significant side effects.
- Both frusemide and spironolactone are combined and given in the state of liver ascites

Table 1. Diuretic Dosage

Diuretic	Range	Dosage (mg/day)		
		Edema	No edema	p[a]
Furosemide	80–400	229.3 ± 61.1	262.8 ± 61.2	NS
Amiloride	25–100	42.4 ± 21.0	41.8 ± 8.4	NS
Spironolactone	100–400	273.0 ± 73.3	228.0 ± 55.6	NS

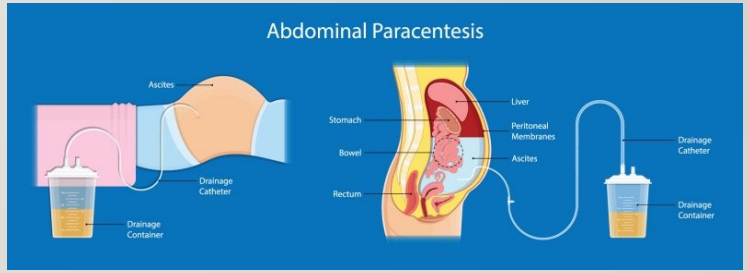
TREATMENT

- potassium-sparing diuretics :**Spironolactone is the initial diuretic of choice** 100 mg PO daily is indicated.
- Furosemide may be added if spironolactone fails to initiate diuresis.
- Volume status must be monitored closely by daily weight check and frequent examinations during initial furosemide treatment.
- Loop diuretics, such as **furosemide** (20-40 mg, increasing to a maximum dose of 160 mg PO daily) or bumetanide can be added to spironolactone.
- Patients should be observed closely for signs of dehydration, electrolyte disturbances, encephalopathy, muscle cramps, and renal insufficiency.
- **Nonsteroidal anti-inflammatory agents** may blunt the effect of diuretics and increase the risk of renal dysfunction.

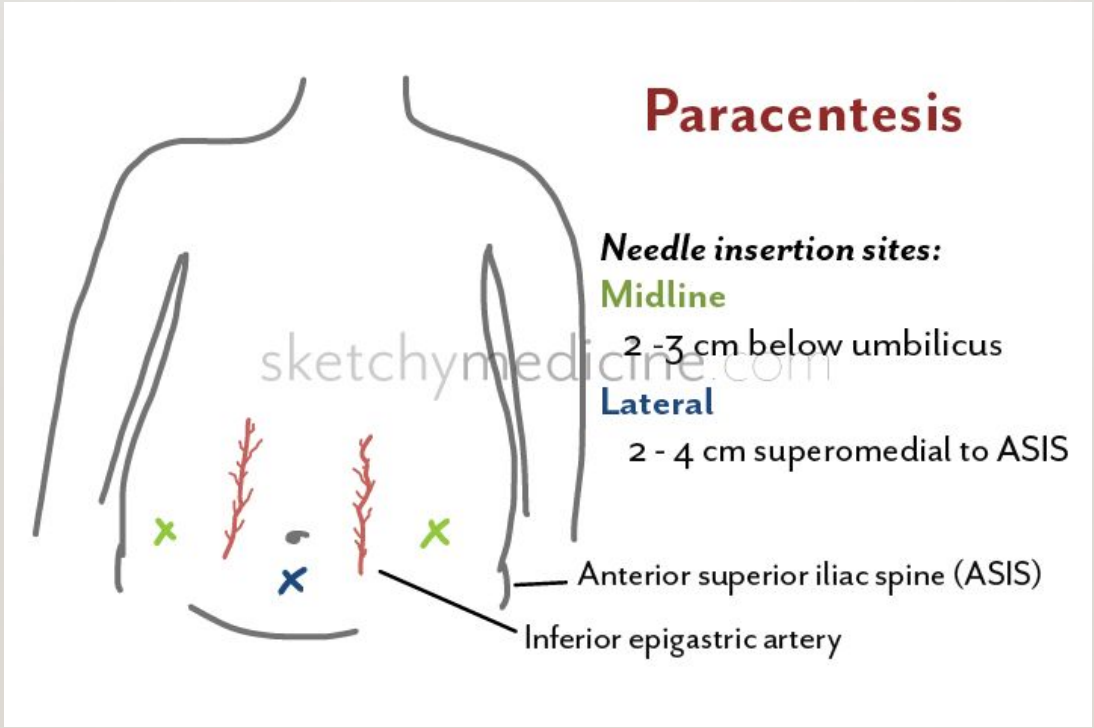
TREATMENT

- **Therapeutic abdominal paracentesis**

It is to place a needle into the abdominal area, under sterile conditions, Up to 10 L of ascites can be removed safely if the patient has peripheral edema, the fluid is removed over 30 to 90 minutes, and oral fluid restriction is instituted to avoid hyponatremia.



TREATMENT


Paracentesis
Needle insertion sites:
Midline
2 -3 cm below umbilicus
Lateral
2 - 4 cm superomedial to ASIS
sketchymedicine.com
Anterior superior iliac spine (ASIS)
Inferior epigastric artery

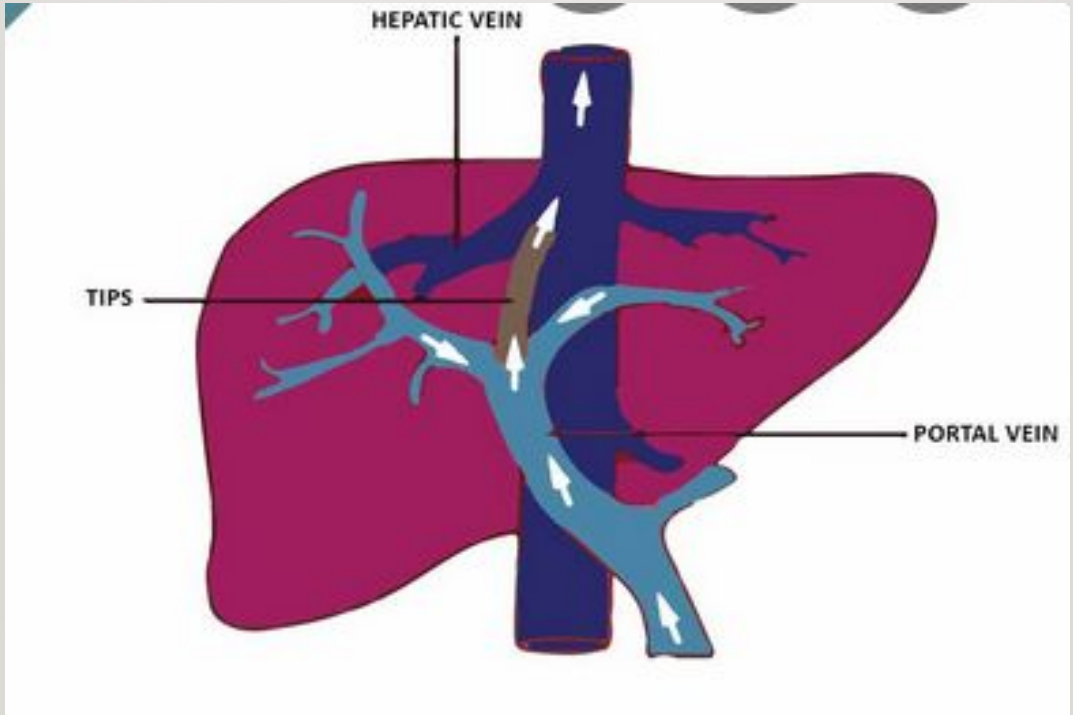
TREATMENT

- Paracentesis is **useful in the initial evaluation of ascites**, when SBP is included in the differential diagnosis, to provide acute decompression of tense ascites also used in malignant ascites
- **symptoms of tense ascites**, including respiratory compromise, impending peritoneal rupture through an ulcerated umbilical hernia, or severe abdominal discomfort.
- The disadvantage of this technique is that if used alone it will lead to post-paracentesis circulatory dysfunction (PPCD) in 20% cases.

TREATMENT

- **Transjugular intrahepatic Portosystemic shunts(TIPS)**
- It is a procedure done through the internal jugular vein under local anesthesia. Where a shunt is placed between the portal venous system and the systemic venous system , thereby reducing the portal pressure.
- TIPS can be used for refractory hard to treat ascites, also for patients who have minimal response to aggressive medical treatment.
- Complications of TIPS include shunt occlusion, bleeding, infection, cardiopulmonary compromise, hepatic encephalopathy, hepatic failure, and death

TREATMENT

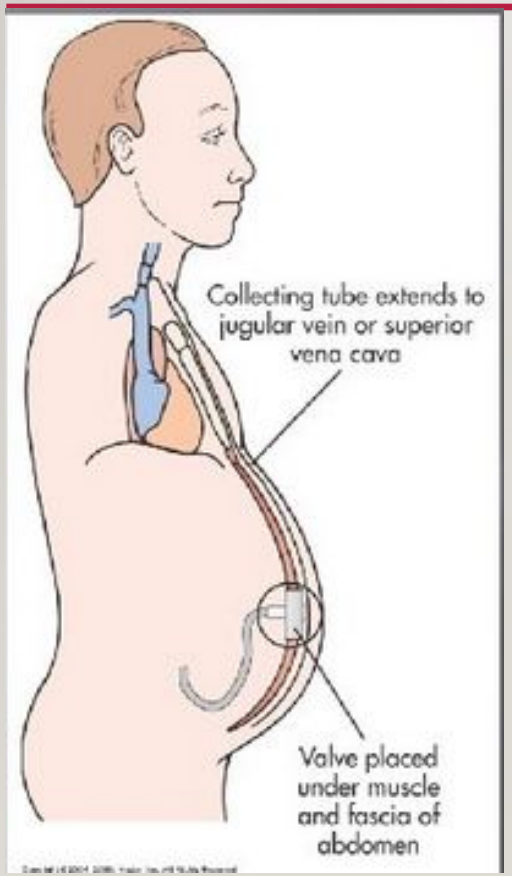


TREATMENT

Peritoneovenous shunt

- This method reinfuses ascites into the vascular space, is now rarely used for ascites refractory to medical therapy.
- In rare cases in which ascites accumulates rapidly after paracentesis and the patient is otherwise fit, permanent drainage of the ascitic fluid via a peritoneovenous shunt (e.g. LeVeen, Denver) may render the patient more comfortable.
- The main complication of peritoneal venous shunting is disseminated intravascular coagulation, which can be fulminant after shunt placement and requires shunt occlusion.

TREATMENT



- A chamber placed subcutaneously over the chest or abdominal wall which may be included for manual compression.
- Complications include overloading the venous system, cardiac failure and disseminated intravascular coagulopathy. The frequency of these complications may be reduced by evacuating ascitic fluid and partially replacing it with normal saline at the time of shunt insertion.
- **The procedure may also be used for patients with terminal malignant ascites, giving improved quality of life despite the risk of further dissemination of malignant cells.**

TREATMENT

Liver transplant

Liver transplantation is considered as a treatment for advanced cirrhosis and nonalcoholic liver diseases

ASCITES COMPLICATIONS

1. Spontaneous bacterial peritonitis (SBP)

is an acute bacterial infection of ascitic fluid.

Due to spontaneous transmigration of gut bacteria (single pathogen) into peritoneum.

Clinical features usually include :

1- local symptoms and/or signs of peritonitis

2- GI upset (secondary to ileus, e.g. nausea and vomiting)

3- Signs of systemic inflammation (hyper- or hypothermia, chills, tachycardia and tachypnoea ± signs of septic shock)

4- worsening liver and renal function

5- Hepatic encephalopathy and GI bleeding.

ASCITES COMPLICATIONS

2. Hepatorenal Syndrome

Cirrhosis and portal hypertension → can trigger the production and release of vasodilators and cytokines like nitric oxide and prostaglandins → which cause splanchnic and systemic vasodilation. The systemic drop in circulating pressure triggers the carotid and aortic arch baroreceptors to activate three separate compensatory mechanisms. These include the renin-angiotensin-aldosterone system, vasopressin release, and activation of the sympathetic nervous system (SNS).

The progression of the cirrhosis causes the fall in cardiac output and the fall in systemic vascular resistance in a cycle → that induces further renal vasoconstriction. → This leads to further renal hypoperfusion, worsened by renal vasoconstriction with the end point of renal failure.

ASCITES COMPLICATIONS

3. Pleural effusion (hepatic hydrothorax)

Ascites associated with liver disease elevates the diaphragm and causes basilar lung atelectasis with resulting dyspnea and hypoxemia. Furthermore, a hepatic hydrothorax may develop from flow of ascites fluid into the pleural space via diaphragmatic defects

4. Abdominal wall hernias
5. Cellulitis
6. Tense ascites

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