Ascites

Definition of ascites:

- Accumulation of fluid within the peritoneal cavity.

Causes of ascites:

A) As part of generalized anasarca:

1) Hepatic e.g. liver cirrhosis.
2) Cardiac e.g. congestive heart failure.
3) Renal e.g. nephrotic syndrome.
4) Nutritional e.g. severe malnutrition.

Causes of ascites:

B) Local causes:

1) Abdominal malignancy.
2) TB peritonitis.
3) Bacterial or fungal peritonitis.

Causes of ascites:

Ascites is better classified as:

1) **Portal hypertensive ascites:** serum ascites albumin gradient (SAAG) more than 1.1 g% e.g. hepatic and cardiac.
2) **Non-portal hypertensive ascites:** SAAG <1.1 g% in renal, malignant and inflammatory (TB) ascites.

Causes of ascites:

Differential criteria between transudate and exudate ascites:

Item	Transudate	Exudate
Specific gravity	< 1017	> 1017
Protein contents	< 2.5 g/dl	> 2.5 g/dl
Protein ascites/serum ratio	< 0.5	> 0.5
Lactic dehydrogenase	< 200 IU/L	> 200 IU/L
Causes	Cases of generalized anasarca due to hepatic, cardiac, renal and nutritional causes	In association with infections, malignancy, pancreatic or connective tissue diseases

Differential diagnosis of ascites:

1) Obesity = fat.
2) Gaseous distension = flatus.
3) Intestinal obstruction = feces.
4) Pregnancy = fetus.
5) Ovarian cyst.

Clinical Features of portal hypertension ascites:

A) Symptoms:

- Insidious onset. It may develop suddenly.
- Increased abdominal girth.
- Recent weight gain.
- Shortness of breath.
- Early satiety.
- Generalized abdominal pain.

Clinical Features of portal hypertension ascites:

B) Signs:

- Physical signs suggestive of cirrhosis and portal hypertension (e.g., spider navei, palmar erythema, splenomegaly, or flapping).
- The patient is sallow, muscle wasting is profound, and the thin limbs with protuberant belly lead to the description of the patient as a spider man.

Clinical Features of portal hypertension ascites:

B) Signs:

- The abdomen is distended (mainly in the flanks), the umbilicus is everted, protrusion of hernias, distended abdominal wall veins (portal & IVC collaterals), the latter disappeared when ascites is controlled. Abdominal striae may develop.
- Ascites is mainly detected by percussion.

Clinical Features of portal hypertension ascites:

C) Associations:

- Pleural effusion: due to diaphragmatic defect allowing ascites to pass into the pleural cavity, mainly right sided.
- The lung bases may be dull on percussion, with crepitations due to elevated diaphragm.
- Oedema usually follows ascites.

Clinical Features of portal hypertension ascites:

C) Associations:

- Cardiac apex: beat is displaced up and out.
- Neck veins are distended by the increased right atrial pressure and intrapleural pressure.

Investigations of portal hypertension ascites:

A) Ultrasonography:

- It detects minimal ascites, encysted ascites.
- It shows the size of the liver, spleen, portal vein diameter, thus it can diagnose portal hypertension.
- It shows the echopattern and surface of the liver and thus can diagnose liver cirrhosis and tumours.
- It may detect lymphadenopathy.

Investigations of portal hypertension ascites:

B) Diagnostic paracentesis:

- The fluid is mainly transudate with proteins less than 2.5 gm/dL, straw coloured. Blood points to malignancy.
- SAAG is > 1.1 g/dL.
- In sterile ascites, ascitic fluid WBC count is usually less than 100/mm^3 with a predominance of mononuclear cells and a low number of PMNLs.

Investigations of portal hypertension ascites:

C) Plain X-ray abdomen:

- shows diffuse ground glass appearance.

D) Laparoscopy:

- It is highly diagnostic for TB lesions allowing biopsy, histopathology and culture which settles the diagnosis beyond doubt. Laparoscopy also show clearly the spleen, liver and allow for aimed biopsy if needed.

Evaluation of patients with cirrhosis and ascites:

A) Clinical evaluation:

1. Ask for the rate of re-accumulation and the manifestations of TB toxaemia.
2. Look at shape of abdomen, percuss for shifting dullness, elicit fluid thrill or do padell sign.
3. Look for signs of portal hypertension, liver cell failure and heart failure.

Evaluation of patients with cirrhosis and ascites:

A) Clinical evaluation:

4. Look for signs of hypoproteinaemia.
5. Look for abdominal masses or lumps.
6. Look for signs of chronic increase of intra-abdominal pressure.
7. Look for associations such as hernias, pleural effusion, oedema, displaced apex, etc.

Evaluation of patients with cirrhosis and ascites:

B) Evaluation of liver disease:

1. Liver function and coagulation tests.
2. Standard haematologic tests.
3. Abdominal ultrasonography, plain X ray or CT.
4. Endoscopy of the upper gastrointestinal tract.
5. Liver biopsy in selected patients.

Evaluation of patients with cirrhosis and ascites:

C) Evaluation of renal and circulatory function:

1. Measurement of serum creatinine and electrolytes.
2. Measurement of urinary sodium.
3. Measurement of urinary protein.
4. Arterial blood pressure.

Evaluation of patients with cirrhosis and ascites:

D) Evaluation of ascitic fluid:

1. Cell type and count.
2. Blood fluid points to malignancy, but is extremely rare in TB, and is initial in traumatic tap.
3. Bacterial culture and sensitivity.
4. Measurement of total protein, SAAG.

Evaluation of patients with cirrhosis and ascites:

D) Evaluation of ascitic fluid:

5. Other tests (albumin, glucose, LDH, amylase, triglycerides, acid-fast smear, PCR and cytologic examination).

Complications of ascites:

1. Infection: spontaneous bacterial peritonitis, secondary bacterial peritonitis.
2. Hepatic hydrothorax.
3. Abdominal hernias.
4. Refractory ascites.
5. Hepatorenal syndrome.

Treatment of cirrhotic ascites:

1. Bed rest, high protein diet, body weight and urine volume daily.
2. Restrict fluid to 1 litre, sodium to 22 mEq, KCL 100 mEq.
3. After two days, if weight loss is less than 1 Kg in 4 days, start spironolactone 150 mg or amiloride 10 mg if after two days response is not satisfactory, add frusemide 80 mg or bumetanide 1mg.

Treatment of cirrhotic ascites:

4. After 10 days if weight loss below 2 Kg, give amiloride 20 mg and spironolactone 300, with response, stop frusemide or bumetanide, then spironolactone, amiloride then the low sodium diet is relaxed.
5. Paracentesis is generally a 2nd line treatment except for patients with tense and refractory ascites.

Refractory ascites:

Definition: ascites that cannot be mobilized or prevented from recurring by medical therapy. **It is divided into:**

- **Diuretic-resistant:** ascites is not mobilized despite maximal diuretic dosage.
- **Diuretic-intractable:** development of diuretic-induced complications that preclude the use of an effective diuretic dosage.

Refractory ascites:

- Dietary history, use of NSAIDs, ACEIs or ARBs, and patient compliance with the treatment regimen must be reviewed before confirming the diagnosis.

Treatment of refractory ascites:

1. Repeated large volume paracentesis (LVP) with or without IV salt free albumin.
2. TIPS should be considered 2nd line treatment, and reserved for patients who require frequent LVP.
3. Peritoneovenous (Le-Veen) shunt, it is mostly indicated in patients who require LVP frequently and who are not candidates for TIPS.

Treatment of refractory ascites:

4. Liver transplantation.

Thank you