OEDEMA increased interstitial fluid

- Pitting' oedema reflects increased interstitial fluid, which ' can result from disruption of the 'Starling forces' which dictate fluid transit across capillary basement membranes It is usually influenced by the effect of gravity on venous hydrostatic pressure and so accumulates in the ankles during the day and improves overnight ('dependent' oedema).
- Non-pitting oedema may reflect protein deposition: for example, in myxoedema associated with hypothyroidism

edema

is an abnormal accumulation of fluid under the skin or in one or more cavities of the body(interstitial space).

Generally, the amount of interstitial fluid is determined by the balance of fluid (homeostasis), and increased secretion of fluid into the interstitium or impaired removal of fluid from it.

Mechanism

Six factors can contribute to the formation of edema:

*increased hydrostatic pressure;

*reduced oncotic pressure within blood vessels;

*increased tissue oncotic pressure;

*increased blood vessel wall permeability e.g. inflammation;

*obstruction of fluid clearance via the lymphatic system;

*changes in the water retaining properties of the tissues themselves.

Raised hydrostatic pressure often reflects retention of water and sodium by the kidney

this fluid may cause edema.

Classification

1-Generalized

A rise in hydrostatic pressure occurs in cardiac failure.

A fall in osmotic pressure occurs in nephrotic syndrome and liver failure. Gut disorder ,idiopathic

2-Locolised venous obstraction ,lymphatic obstraction ,allergy ,inflamation

3-Postural oedema common but un important

Locolised edema(Organ-specific)

Edema will occur in specific organs as part of inflammation, as in pharyngitis, tendonitis or pancreatitis, :

Cerebral edema is extracellular fluid accumulation in the brain. It can occur in toxic or abnormal metabolic states and conditions such as systemic lupus. It causes drowsiness or loss of consciousness.

Pulmonary edema occurs when the pressure in blood vessels in the lung is raised because of obstruction to remove blood via the pulmonary veins. This is usually due to failure of the left ventricle of the heart. It can also occur in altitude sickness or on inhalation of toxic chemicals. Pulmonary edema produces shortness of breath.

Pleural effusions may occur when fluid also accumulates in the pleural cavity.

Edema may also be found in the cornea of the eye with glaucoma, severe conjunctivitis or keratitis or after surgery. It may produce coloured haloes around bright lights.

DIFFERENTIAL DIAGNOSIS OF PERIPHERAL OEDEMA

Cardiac failure

right or combined left and right heart failure, pericardial : constriction, cardiomyopathy

Chronic venous insufficiency

varicose veins **:**

Hypoalbuminaemia

nephrotic syndrome, liver disease, protein-losing **:** enteropathy; often widespread, can affect arms and face

Drugs

Sodium retention: fludrocortisone, non-steroidal anti-inflammatory agents

Increasing capillary permeability: nifedipine, amlodipine

Idiopathic

women > men **:**

Chronic lymphatic obstruction

CAUSES OF OEDEMA •

Low plasma oncotic pressure(Low serum albumin) •

due to •

Increased loss-nephrotic syndrome •

Decreased synthesis-liver failure •

Malnutrition/malabsorption •

Increased capillary permeability Leakage of proteins into the • interstitium, reducing the osmotic pressure gradient which draws fluid into the lymphatics and blood

*Local-infection/inflammation •

*Systemic-severe sepsis •

*Drug-related, e.g. calcium channel blockers •

Increased hydrostatic pressure (High venous pressure/obstruction •

Deep venous thrombosis or venous insufficiency-local oedema •

Pregnancy •

Pelvic tumour •

Congestive heart failure •

(Intravascular volume expansion (iatrogenic, renal failure, Conn's syndrome •

Lymphatic obstruction •

(Infection-filariasis, lymphogranuloma venereum •

Malignancy •

Radiation injury •

Congenital abnormality •

•

Ascites is defined as the accumulation of free fluid in the peritoneal cavity.

It is a common clinical finding with a variety of both extraperitoneal and peritoneal etiologies.

It is most often caused by liver cirrhosis which accounts for over 75% of patients while the remaining 25 % is due to malignancy (10%), heart failure (3%), pancreatitis (1%), TB (2%), or other rare causes.

AscitesAscites is anaccumulation of fluid in the peritoneal cavity(. space between the tissues lining the abdomen and abdominal organs)

Causes

- Cirrhosis and any illness that leads to it
- Clots in the veins of the liver (portal vein thrombosis)
- Congestive heart failure
- Constrictive pericarditis
- Infections such as tuberculosis , Spontaneous bacterial peritonitis
- Nephrotic syndrome
- cancer,peritonial ,endometrial cancer ,ovarian ,liver ,pancriatic ca ,colon ca……
- Pancreatitis
- Protein-losing enteropathy
- Rare
  - Meigs' syndrome
  - Vasculitis
  - Hypothyroidism
  - Renal dialysis

1-history •

2-examination •

3-investigation •

- ultrasound •

-liver function test •

CBP and ESR •

TSP and s.albumin •

CXR ,echo. •

Asperation of ascitic fluid •

- Ascites causes abdominal distension with fullness in the flanks, shifting dullness on percussion and, when the ascites is marked, a fluid thrill .These signs do not appear until the ascites volume exceeds 1 litre, even in thin patients, and much larger volumes can be hard to detect in the obese. Associated features of ascites include distortion or eversion of the umbilicus, herniae, abdominal striae, divarication of the recti and scrotal oedema.
- Pleural effusions are found in about 10% of patients, usually on the right side (hepatic hydrothorax); most are small and only identified on chest X-ray, but occasionally a massive hydrothorax occurs. Pleural effusions, particularly those on the left side, should not be assumed to be due to the ascites.
- Ultrasonography is the best means of confirming ascites, particularly in the obese and those with small volumes of fluid. Paracentesis (if necessary under ultrasonic guidance) can also be used to confirm the presence of ascites but is most useful for obtaining ascitic fluid for analysis. The appearance of the ascites may point to the underlying cause (The ascites protein concentration and the serum-ascites albumin gradient are used to distinguish ascites due to transudation from ascites due to exudation.

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) •

Amylase •

White cell count (no. and diffrantial count of WBC) •

Cytology •

Microscopy and culture •

- **Cirrhotic patients typically develop a transudate with a total protein concentration below 25 g/l and relatively few cells. serum-ascites albumin gradient more than 11 g/l is strongly suggestive of portal hypertension and cirrhosis.**
- **Exudative ascites (ascites protein concentration above 25 g/l or a serum-ascites albumin gradient of less than 11 g/l) raises the possibility of infection (especially tuberculosis), malignancy, hepatic venous obstruction, pancreatic ascites or, rarely, hypothyroidism. Ascites amylase activity above 1000 U/l identifies pancreatic ascites, and low ascites glucose concentrations suggest malignant disease or tuberculosis.**
- **Cytological examination may reveal malignant cells (one-third of cirrhotic patients with a bloody tap have a hepatoma).**
- **Polymorphonuclear leucocyte counts above 250 × 10^6/l strongly suggest infection (spontaneous bacterial peritonitis,**
- **Laparoscopy can be valuable in detecting peritoneal disease.**

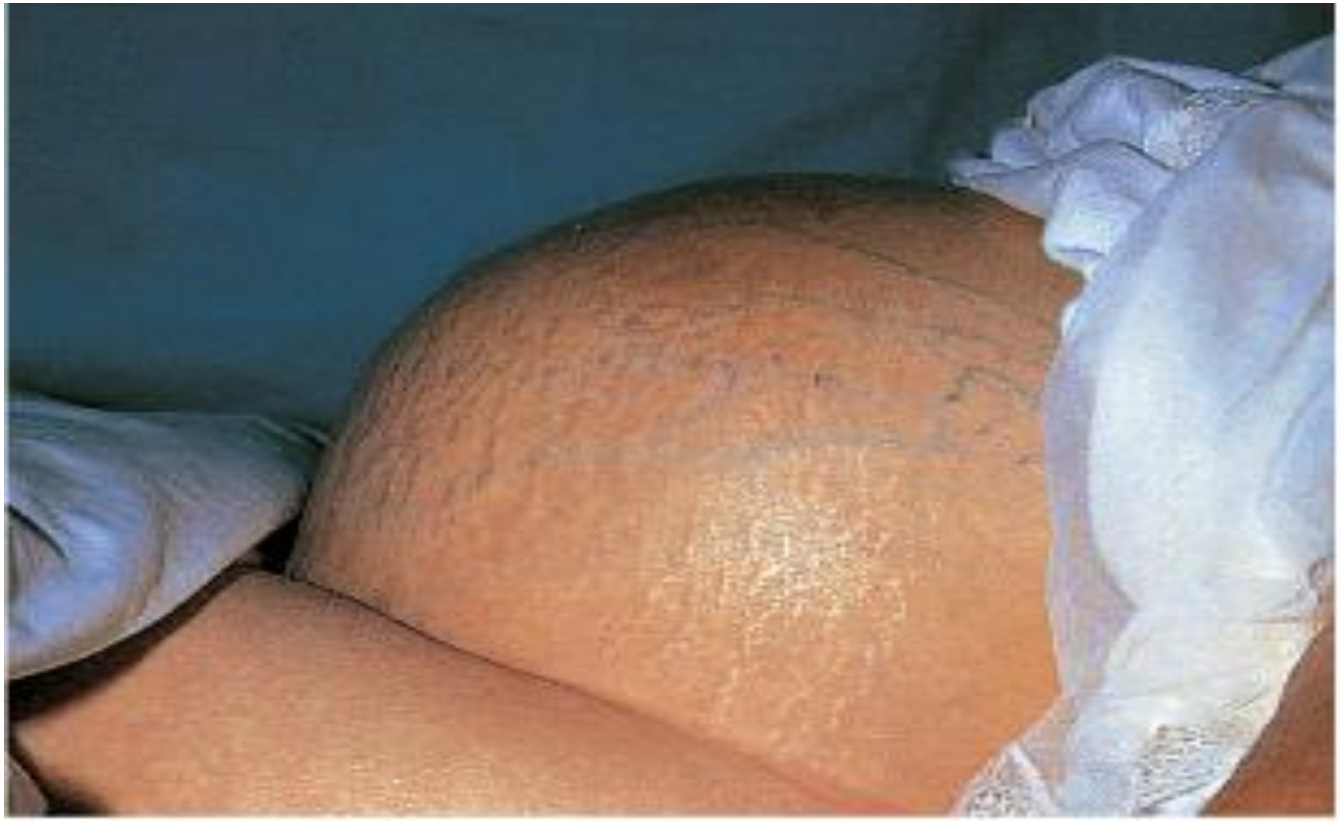
Asperation of ascitic fluid •

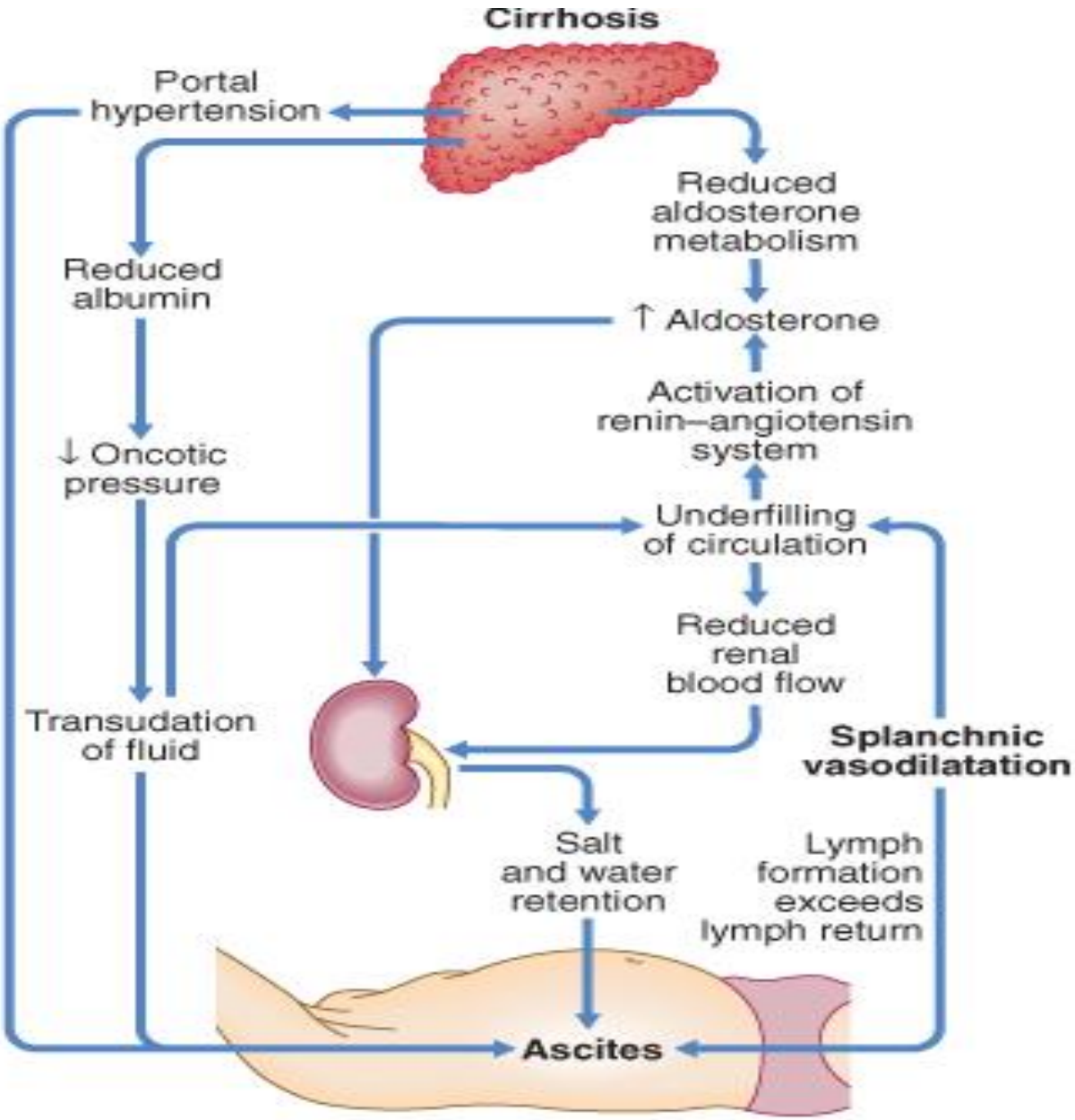
50 to 100 mL •

- Gross appearance •
- Protein content, sugar content •
- Cell count, and differential cell count •
- Gram's and acid-fast stains and culture •
- Cytological and cell-block examination •

Serum-Ascites Albumin gradient (SAAG)

- The gradient correlates directly with portal pressure.
- A gradient >1.1 g/dL → uncomplicated cirrhotic ascites
- A gradient <1.1 g/dL (low gradient) suggests that the ascites is not due to portal hypertension with >95% accuracy and mandates a search for other causes




Cirrhosis
Portal hypertension
Reduced albumin
↓ Oncotic pressure
Transudation of fluid
Reduced aldosterone metabolism
↑ Aldosterone
Activation of renin–angiotensin system
Underfilling of circulation
Reduced renal blood flow
Salt and water retention
Lymph formation exceeds lymph return
Splanchnic vasodilatation
Ascites

Treatment

1-The condition that causes ascites will be treated

,

retics, help remove the fluid; usually, spironolactone (Aldactone) is used at first, and
hen furosemide (Lasix) will be added ,

iotics, if an infection develops ,

miting salt in the diet (no more than 1,500 mg/day of sodium) .

ding drinking alcohol .

Procedures used for ascites that do not respond to medical treatment include: .

acing a tube into the area to remove large volumes of fluid (called a large volume paracentesis)less than 1Ldayly

gular intrahepatic portosystemic shunt (TIPSTIPS), which helps reroute blood around the liver

ents develop end-stage liver disease, and the ascites no longer respond to treatment, liver transplantation becomes necessary.

Possible Complications

Spontaneous bacterial peritonitis (a life-threatening infection of the ascites fluid)

atorenal syndrome (kidney failure) .

nt loss and protein malnutrition .

ntal confusion, change in the level of alertness, or coma (hepatic encephalopathy)

complications of liver cirrhosis .

Legs swlling (oedema)

- 1-bilateral
- 2-unilateral