Hepatic dysfunction:

- Its result from damage to the liver parenchymal cell, either

- directly from primary liver disease
- or indirectly obstruction of bile flow or Disturbances of hepatic circulation .

- Its may be acute or chronic (which more common, Chronic liver disease such as cirrhosis).

- Diseases that lead to hepatocellular dysfunction can be caused by infection (bacteria, virus) , endocrine , toxins and medication , nutritional deficiencies .

- The parenchyma respond by replacing glycogen with lipid , producing fatty infiltration with or without cell death or necrosis the result is the shrunken , fibrotic cell.

- **The most common symptom of hepatic dysfunction** :

1. Jaundice
2. Portal hypertension
3. Nutritional deficiencies

Jaundice

Its increase concentration of bilirubin in the blood .

- sclera, skin become yellow- tinged or greenish- yellow . jaundice become clinically evidence when the serum level of bilirubin exceeds 2.5 mg/dl .

Types of Jaundice:

1. ***Hemolytic* *Jaundice*** :destruction of the RBC. Fecal and urine urobilinogen level is increased .

Causes : blood transfusion reaction, hemolytic disorder (anemia)

Complications: gallbladder stone .

2. ***Hepatocellular* *Jaundice*** : inability of damaged liver cell to clear normal amount of bilirubin from blood .

Causes: infection (hepatitis), medication and chemical toxicity ,

Symptoms: mildly or severely ill (loss of appetite, nausea , weakness, wt loss).

Jaundice

3. **Obstructive Jaundice** : occlusion of bile duct by a gallstone , an inflammatory process, a tumor, enlarged organ .

Obstruction may be within the liver (intrahepatic obstruction) result from swelling of liver or ducts, and thickening of bile within the canaliculi.

Elevation in serum AST. ALT .

4. **Hereditary Hyperbilirubinemia**: e.g (Gilberts syndrome is a familial disorder characterized by an increased level of unconjugated bilirubin .

Portal Hypertension

- Its increase pressure throughout the portal venous system . result from obstructed blood flow throughout the damaged liver.
- Its commonly associated with liver cirrhosis .
- two major consequences of portal hypertension are ascites and varices.

Ascites pathophysiology: Portal hypertension --- increase capillary pressure and obstruction of venous blood throughout damaged liver cell--- failure liver to metabolize aldesterone --- increase sodium and water retention --- increased intravascular fluid volume and decrease albumin --- fluid shifting from vascular to peritoneal space .

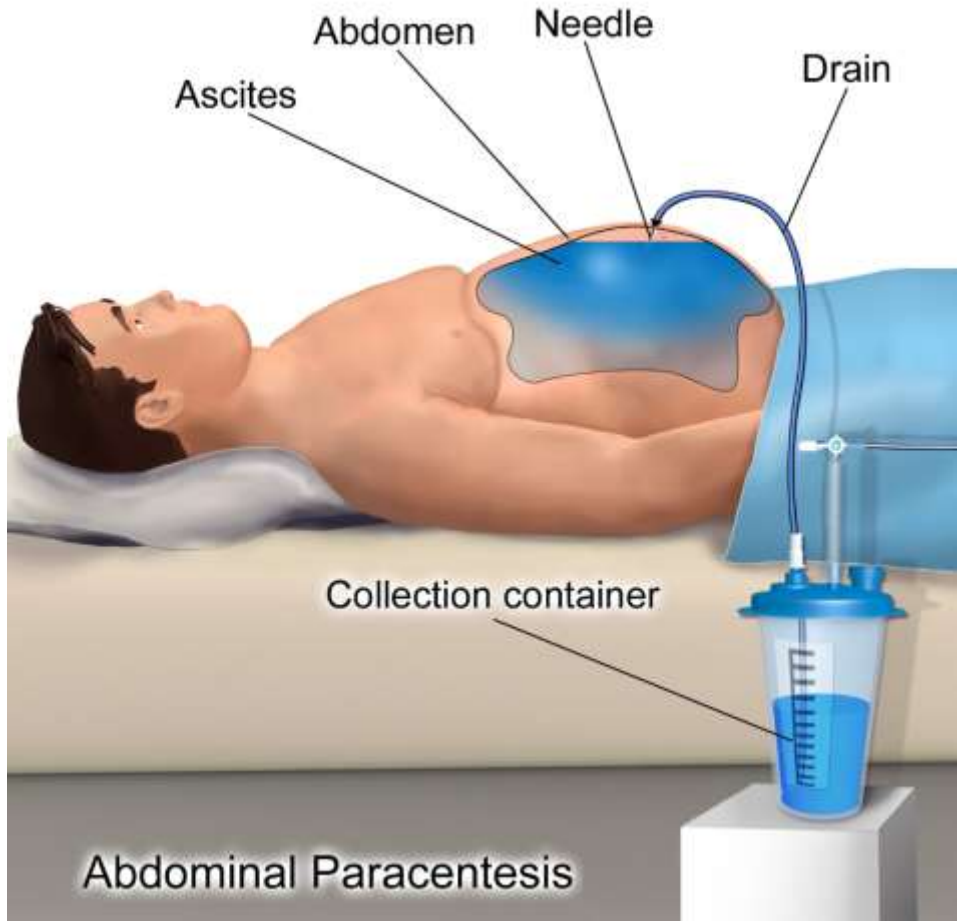
Ascites

C/M: Increased of abdominal girth , and rapid wt gain . short rapid breathing , distended abdominal vein and striae . fluid and electrolyte imbalance .

Assessment and Diagnosis : ***Physical examination*** (shifting dullness , fluid wave)

Management:

1. Dietary Modification : low salt and decrease fluid intake
2. Diuretic :(aldactone(spironolactone (prevent k loss)).
3. Bed rest
4. Paracenthesis : is removal of fluid from peritoneal cavity through a small surgical incision or puncture .
5. Peritonvenous shunt : to redirect ascites fluid from the peritoneal cavity into the systematic circulation .


Abdomen
Needle
Drain
Ascites
Collection container
Abdominal Paracentesis

Other manifestations of liver dysfunction

- vitamin deficiency : vitamin K (altered in clotting factors production) . and vitamins (D.E.A.K).
- metabolic abnormalities : glucose disturbance

(hyperglycemia after meal and hypoglycemia during fasting period) .

- edema and bleeding
- skin changes and Puritus.

Hepatitis is a viral infection of the liver . Five types of hepatitis virus have been identified.

Type	Mode of transmission	Incubation period
Hepatitis A and E	Primarily fecal–oral	Incubation 3-5weeks,
Hepatitis B	Blood. Saliva, sexual activities,	Incubation 2-5 months,
Hepatitis C	blood or blood product transfusion, sexual, dialysis,	Incubation 1 week to several months,
Hepatitis D	Blood. Saliva, sexual activities,	Incubation 2-5 months,

Clinical manifestations

Type	Clinical manifestations
A	**no symptoms. fatigue, anorexia, malaise, headache, low-grade fever, nausea, and vomiting.**
B	**asymptomatic, myalgias, photophobia, arthritis, skin rashes, vasculitis. Jaundice,**
C	**Similar to those associated with HBV but often less severe**
D	**Similar to HBV but more severe**

Diagnostic Evaluation

- Liver function test
- liver biopsy
- Ultrasound, CT-scan, MRI,

Management

- Rest according to patient's level of fatigue.
- Therapeutic measures to control dyspeptic symptoms and malaise.
- Hospitalization for protracted nausea and vomiting or life-threatening complications.
- Small, frequent feedings of a high-caloric, low-fat diet; proteins are restricted when the liver cannot metabolize protein by-products.
- Vitamin K injected SC if PT is prolonged(INR).
- Intravenous fluid and electrolyte replacement as indicated.
- Administration of antiemetic for nausea.
- After jaundice has cleared, gradual increase in physical activity. This may require many months.

Gallbladder disorders

- **Cholelithiasis:** Is the presence of stones in the gallbladder.
- **Cholecystitis** is inflammation of the gallbladder (may be acute or chronic).
- **Choledocholithiasis** is the presence of stones in the common bile duct.

Pathophysiology/Etiology

- **A. Cholelithiasis**
- Stones occur when cholesterol supersaturates the bile in the gallbladder and precipitates out of the bile, predisposes to the formation of gallstones .
- Four times more women than men develop cholesterol stones.
- older than 40 years of age, multiparous, and obese.
- Stone formation increases in users of contraceptives, and cholesterol-lowering drugs, which are known to increase biliary cholesterol saturation.
- Bile acid malabsorption, genetic predisposition, and rapid weight loss
- Pigment stones occur when free bilirubin combines with calcium.
- Found in patients with cirrhosis, haemolysis, and infections in the biliary tree.
- GI disease, bile fistula, gallstone ileus, carcinoma of the gallbladder, or in those who have had ileal resection or bypass

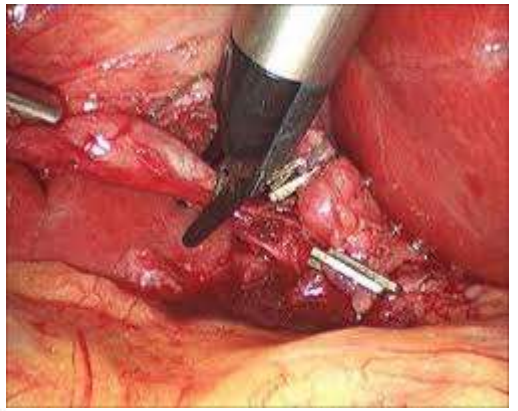
B. Cholecystitis

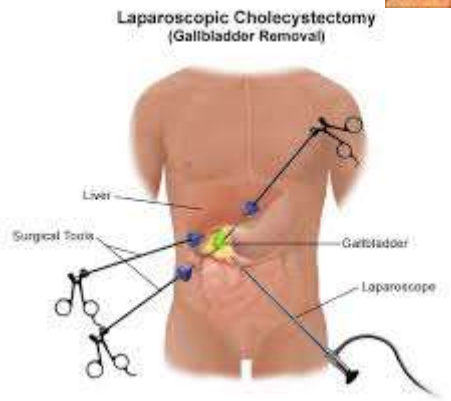
- Acute cholecystitis is an acute infection of the gallbladder.
- If the gallbladder is filled with pus, there is empyema of the gallbladder.
- caused by gallstone obstruction of the cystic duct, causing edema, inflammation, and bacterial invasion. This is called calculous cholecystitis.
- Occurs after major surgical procedures, severe trauma, or burns.
- Chronic cholecystitis occurs when the gallbladder becomes thickened, rigid, and fibrotic and functions poorly.
- Results from repeated attacks of cholecystitis, presence of **calculi**, or chronic irritation.

Clinical Manifestations

- usually asymptomatic.
- Biliary colic.
- Steady, severe aching pain or sensation of pressure in the epigastrium or RUQ, radiate to the right scapular or shoulder.
- Begins suddenly and persists for 1 to 3 hours
- **Acute cholecystitis** causes biliary colic pain that persists more than 4 hours and increases with movement, including respirations.
- Nausea and vomiting, low-grade fever, and jaundice (with stones or inflammation in the common bile duct).
- RUQ guarding and **Murphy's sign** (inability to take a deep inspiration when examiner's fingers are pressed below the hepatic margin)
- **Chronic cholecystitis** causes heartburn, flatulence, and indigestion.
- Repeated attacks of symptoms may occur resembling acute cholecystitis

Management





- Supportive management includes:
- rest,
- IV fluids,
- nasogastric suction,
- pain management,
- and antibiotics (in the presence of a positive culture).
- Surgical management.
- a. Cholecystectomy, open or laparoscopic(lap chole)

Nursing interventions

- Relieving Pain
- Restoring Normal Fluid Volume
- Patient Education/ Health Maintenance

1.Instruct patient in care of any tubes .

a. Observe for bleeding or drainage around insertion site.

b. Replace gauze dressing when it becomes wet or soiled.

c. Report any change in drainage.

2. Review postoperative discharge instructions for activity, diet, medications, and postoperative follow-up.