D. Hemorrhagic ascites:

- Traumatic specially rupture spleen.
- Malignancy.
- Hemorrhagic blood diseases.

E. Meig's syndrome: it is an ovarian fibroma associated with ascites (in 20% of cases) & right-sided pleural effusion (in 2% of cases).

Clinical picture:

A. Clinical picture of ascites:

1. **Inspection**:
   - Distended abdomen specially in the flanks.
   - The umbilicus is shifted downward & everted.
   - White abdominal striate.
   - In chronic cases → wide subcostal angle & divarication of recti.
   - Dilated veins in the anterior abdominal wall.
2. **Palpation**:
   - Fluid transmitted thrill.
   - Liver & spleen may be felt by dipping method.
   - Abdominal swelling may be felt in malignancy & TB.
3. **Percussion**:
   - Resonance over the umbilicus & dull flanks (>2L).
   - Shifting dullness from side to side (>1.5 L).
   - Knee elbow position (300 – 500 cc).
4. **Auscultation**:
   - Puddle sign: in the knee elbow position, but the diaphragm on the umbilicus & scratch from outside towards the umbilicus till change tone → + ve = fluid.
   - Venous hum may be heard in cases of portal hypertension (Kenawi sign).

B. Clinical picture of the 2ry effects of ascites:

1. Right-sided pleural effusion:
2. Edema following ascites.
3. Rise of the diaphragm casing:
   - Congested neck veins.
   - Shift of the apex of the heart upward & outward.
   - Dullness over the lung.

C. Clinical picture of the cause: e.g.: cirrhosis – hepatoma – HF – TB ….etc.

Investigation:

1. Aspiration & examination of the peritoneal fluid for:
   - Color, turbidity & specific gravity.
   - Protein content, bacteria & pus.
   - Malignant cells.
2. Us to detect minimal ascites, its cause & exclude ovarian cyst.
3. Investigation for the cause.

Treatment:

1. See portal hypertension.
2. Treatment of the cause as surgical removal of tumors & anti-tuberculous drugs for TB.

HEPATIC BILHARZIASIS

Incidence & aetiology: common in the delta region where S. mansoni prevails, it is more common in males 10 – 40 years old.

Clinical picture:

1. Symptoms:
   - Early it is asymptomatic.
   - Later, symptoms of portal hypertension appear.
   - Pure, hepatic bilharziasis is void of features of liver cell failure.
2. General examination:
   - Infantilism may be present (hypopituitarism) → gynecomastia & loss of hair.
   - Associated deficiency manifestations are usually present.
   - Manifestation of liver cell failure is rare & late.
   - Pulmonary hypertension & bilharzial cor pulmonale are frequently detected.
3. Abdominal examination:
   - Distended with wide subcostal angle, divarication of recti & everted umbilicus.
   - Dilated veins & venous hum.
   - The liver is not nodular in pure bilharzias fibrosis.
   - The spleen may reach huge size.

Staging:

1. Hepatomegaly.
2. Hepatosplenomegaly.
3. .
   a. Enlarged tender liver & splenomegaly.
   b. Shrunken liver & splenomegaly.
4. Ascites.
5. Liver cell failure.

Investigations:

1. Investigations to detect bilharziasis:
   - Urine & stool analysis for ova.
   - Sigmoidoscopy with rectal & colonic biopsy.
   - Skin test, CFT, IF & COPT to detect bilharzial antibodies.
   - Liver biopsy. (The surest method).
2. Investigations for portal hypertension.
3. Liver function test:
   - Decreased albumin & increased globulin.
   - BSP test is impaired.
   - Other test are normal except if there is mixed pathology in the liver.

Treatment:

1. Treatment of portal hypertension.
2. Treatment of ascites.
3. Treatment of hepatic encephalopathy.
4. Treatment of active schistososmiasis.
5. Antifibrotic drugs as colchicines & penicillamine.

HEPATITIS

Aetiology

A. Infective:

1. viral :
   - Hepatitis viruses: A –B – C – D – E – G.
   - Opportunistic viruses as EBV – CMV – HSV.
   - Exotic viruses as yellow fever, Ebola & Lassa.
2. Non-viral:
   - Bacterial: TB, typjoidal & brucellosis.
   - Mycoplasma – rickettesiae.
   - Spirochetes – fungi.
   - Parasites: malaria – leishmania – hydrated cyst – amoeba & toxoplasma.

B. Non-infective:

1. Toxins :
   - Alcohol – CCl_4.
   - Drugs as: INH – Rifampicin – halothane – α-methyl dopa.
2. Hypoxia: all causes of congestion as heart failure.

Items	HAV	HEV	HBV	HDV	HCV
Genome	RNA	RNA	DNA	RNA	RNA
Diameter	27-32 nμ	27-34 nμ	42-45 nμ	40-43 nμ	50-60 nμ
Transmission	oral		Blood sexual congenital	blood	
Epidemics	May be		No (only sporadics)		
I.P.	2-6 weeks		2-6 months		2 weeks – 6 months
Onset	Acute		Insidious		Insidious
Acute attack	Mild		Mild or sever		Usually
Chronicity	No		May be	No	May be
Prophylaxis					
A. vaccine	Yes	.....	yes		...
B. IG	yes	yes	yes		...

Notes:

- HAV & HEV:
  - Occurs mainly in children & young adults.
  - People at risk are those living in areas with bad sanitation.
- HBV: people at risk are:
  - Hemophilic & hemolytic anemia patients (repeated blood transfusion).
  - Drugs abusers (contaminated syringes).
  - Homosexuals & prostitutes.
  - Doctors & nurses.
  - Babies born to HBs Ag +ve mothers.

- HDV:
  - ➢ It is incomplete virus depending on HBs Ag for its survival, so it occurs only in HBs Ag + ve persons.
  - ➢ There are forms of infection: 1- co-infection, 2- super-infection.

ACUTE VIRAL HEPATITIS

Pathology

A. HAV & HBV:

1. **Hepatic**:
   - Portal tract: infiltration by inflammation cells & proliferation of bile ducts.
   - Hepatic lobules: centrizonal necrosis & central cholestasis.
   - Healthy framework.
   - Regeneration stars from starts from the periphery to the center.
2. **Extra-hepatic:**
   - RES hyperplasia & splenomegaly.
   - Bone marrow suppression.
   - Nephritis.
   - Vasculitis, PAN.
   - Arthritis.

B. HCV:

- The lesion affects the portal tracts mainly.
- There is ballooning & rupture of bile ducts.
- Infiltration of portal tracts by lymphocytes with follicle formation.
- Perisinusoidal infiltration.

Clinical picture

A. Non-icteric phase:

- Flu-like symptoms.
- No jaundice.
- Complication: cirrhosis which may be diagnosed as idiopathic cirrhosis.

B. Icteric phase:

1. **Pre-icteric stage**:
   - 3 – 9 days.
   - Fever, headache, malaise, bone aches & mylagia.
   - Anorexia (specially to cigarettes), nausea & vomiting.
2. **Icteric stage**:
   - 2 – 6 days.
   - Jaundice appears with relief of the patient.
   - RES hyperplasia →hepatosplenomegaly & enlarged right supracalaviculare LNs.
   - Urine → dark frothy.
   - Stool → pale.
3. **Post-icteric stage**: gradual return to normal.

N.B: sclera has high affinity for bilirubin → it may remain yellow after jaundice disappear, if persist > 6 months → chronic hepatitis.

Variants (sequelae) of viral hepatitis:

1. Recovery: occurs in 90 % of cases.
2. Relapsing: either clinical or laboratory.
3. Acute fulminant hepatitis: leading to death within 10 days, no treatment.
4. Chronisty:

Carrier	chronic active hepatitis
Carcinogenicity	chronic persistent hepatitis
Cirrhosis	chronic lobular hepatitis

5. Prolonged cholestasis (Watt's syndrome):
   Sever hepatitis → massive edema → closes bile canaliculi.
   Clinical picture: pruritus & jaundice.
   Treatment: steroids to decrease edema of the hepatocytes.
6. Post-hepatitis syndrome:
   Occur in psychic patient.
   Clinical picture: palpable liver due to spasm of the diaphragm which pushes the liver downwards.
   Treatment: assurance.

Investigations:

1. CBC:
   - Leucopenia with relative lymphocytosis.
   - Pancytopenia due to bone marrow suppression.
   - Elevated ESR.
2. Urine analysis: increased urobilinogen then decreased after obstruction.
3. Stool analysis: increased stercobilinogen.
4. Liver function tests: elevated ALT & AST – normal albumin – biphasic bilirubin.
5. Electron microscopy → diameter of each type of the viruses.
6. Viral hepatitis markers.

Viral hepatitis markers

HAV markers

HAV – Ag: it is found in the stool 2 weeks before & 1 week after the appearance of jaundice & disappear with the peak of transaminases.

HAV-Ab:

A. IgM:
- Indicates recent infection.
- Appears in the blood after appearance of the virus with the peak of transaminases.
- Good prognosis.

B. IgG: indicates past infection & immunity for recent.

HBV markers:

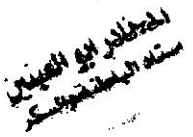
A. *HBs-Ag:* it appears after 6 weeks of infection & disappears after 3 months.
- Value:

1. HBs-Ag +ve person is infected & infective.
2. Disappearance of this antigen & appearance of Ab indicates good prognosis.
3. If persists > 6 months with no Ab → chronicity or carrier state.

4. Quantitative estimation helps in prediction of prognosis (if the initial concentration of the Ag doesn't decrease to 1/4 its value within 6 weeks → bad prognosis.

B. *HBs-Ab:*

1. Starts after HBs-Ag & lasts for 18 months or longer.
2. Window phase: it is the period between disappearance of HBs-Ag appearance of HBs-Ab.
3. value:
   - Its appearance indicates good prognosis.
   - If appeared after vaccination → immunity.



C. *HBc-Ag:* detected in the liver only by biopsy.

D. *HBc-Ab:*

A. IgM:
- Appears 4-6 weeks after HBs-Ag & lasts for 3 months or more.
- The only diagnostic during the window phase.
- Indicates recent infection.

B. IgG: appears later & indicates past infection.

E. *HBe-Ag:*
- Appears 1 week after HBs-Ag & disappears before it.
- It indicates viral replication.

F. *Hbe-Ab:*
- Appears 3 – 6 months after the disease.
- It indicates starting immunity but not guaranteed aginst infection.

G. *HBV DNA:* the virus itself → indicates infectivity.

H. *HBV DNA polymerase:*
- Appears within few days & lasts for few weeks.
- If persist, it indicates viral replication → bad prognosis.

I. *HBs-Ag-Ig M complex:*
- Value: indicates recent infection & infectivity.0
  Causes immune-complex nephritis.

HCV markers

A. *HCV-Ab:* detected by:
- ELISA I & II (enzyme linked immunosorbant assay).
- RIBA I & II (recombinant immune blot assay) → may give false + ve results.

B. *HCV RNA:* detected by PCR (polymerase chain reaction): which is highly diagnostic but very expensive.

HDV markers

A. *HDV-Ag.*

B. *HDV-Ab:* IgM & Ig G.

C. *HDV-RNA* (by PCR) → indicates infectivity.

D. *HBs-Ag.*

Treatment of viral hepatitis

A. Prophylaxis:

- HAV:
  - Good hygienic measures.
  - Non-specific Ig 0.02 – 0.12 mg/kg IM.
- HBV:
  - Passive immunization: by giving HB Ig 0.05 .0.057 mg / kg IM within 6 hours & not after 48 hours.
  - Active immunization: by giving HBV vaccine, 20 mg IM at birth, 1 & 6 months.

B. Curative treatment:

- Rest.
- Diet: only limitation of diet is the patient's appetite.
- Drugs:
  - Cholestyramine → binds bile.
  - Steroids → acute fulminant & Cholestatic hepatitis.
  - Antiviral drugs as interferon.

CHRONIC HEPATITIS

Def.: Hepatitis lasting more than 6 months.

Types:

1. Chronic persistent hepatitis (CPH).
2. Chronic lobular hepatitis (CLH).
3. Chronic active hepatitis (CAH).

1. Chronic persistent hepatitis (CPH):

- Aetiology:
  1. HBV & HCV.
  2. Amoebiasis & bilharziasis.
  3. Alcoholism.
  4. Inflammatory bowel diseases (Crohn's disease & ulcerative colitis).
- Pathology: infiltration of the portal tracts by chronic inflammatory cells.
- Clinical picture:
  1. May be asymptomatic.
  2. Vague manifestations: fatigue - right hypochondrial discomfort – fat & alcoholic intolerance.
- Diagnosis: liver enzymes may be elevated – liver biopsy is diagnostic.
- Treatment: assurance of the patient as it never causes cirrhosis.

2. Chronic lobular hepatitis(CLH):

- Aetiology: unresolved attack of viral hepatitis.

3. Chronic active hepatitis (CAH):

- **Aetiology:**
  1. HBV & HCV - Lupiod (autoimmune hepatitis).
  2. Drugs as INH & α-methyl dopa.
  3. α 1-antitrypsin deficiency.
  4. Wilson's disease (hepatolenticular degeneration).

- **Clinical picture:**

1. Chronic viral hepatitis: (HBV, HCV).

- Occurs in males more than females.
- History of viral hepatitis.
- Asymptomatic.
- Vague manifestation (as before). See (CPH).
- C/O:
  - Decomposition → liver cell failure & portal hypertension.
  - Post-hepatitis type. (In ♂).

2. Chronic non-viral hepatitis:

- Occur in females more than males.
- Acne vulgaries – arthralagia – amenorrhea – alcohol intolerance.
- Diabetes mellitus – glomerulonephritis.
- Hashimoto's thyroiditis – interstitial lung fibrosis.
- Auto-immune hemolytic anemia.
- C/O:
  - Liver cell failure.
  - Portal hypertension → oesophageal varices.

- **Investigations:**

1. Investigations for HBV & HCV.
2. Liver enzymes are elevated > 2.5 times the normal levels.
3. Lupoid hepatitis:
   - IgG is elevated > 3.5 gm.
   - Liver enzymes are elevated > 5 times the normal levels.
   - Antinuclear Ab (ANA) > 1/4 titre.
   - Antismooth muscle (ASMA) > 1/40.
   - Antimitochondreal Ab (AMA) > 1/40.

- **Treatment:**

1. HBV:

- HBeAg + ve cases: interferon 5 million units SC3 times / week for 3 – 6 months.
- HBeAg + ve cases: (no replication of the virus): steroids to suppress the immune system, then give interferon to kill the activated virus.

2. HCV: interferon :

- Dose: 3 million units SC 3 times / week for 3 – 6 months.
- It produces response in 40 – 50 % of cases only.
- Side effects:

Depression	flu-like symptoms
Thrombocytopenia	thyroid abnormality

Relapse occurs in 50 % of the cured patients & Expensive

3. Lupoid hepatitis:

- Steroids: prednisone 60 mg / day for 1 week then decrease to 1 – 15 mg / day for 6 months to years, if improved → gradual withdrawal.
- Azathioprine: 50 mg /day added to low dose steroids for 6 – 24 months.

  Side effects: ovarian damage.

DRUGS & LIVER

Common	Less frequent
1. Drugs causing intrahepatic cholestasis: ▪ Contraceptive pills. ▪ Anti-thyroid drugs. ▪ Anabolic steroids. ▪ Erythromycin. ▪ Chlorpromazine. ▪ Anticoagulants. ▪ Anti-hyperglycemic drugs. ▪ PAS.	1. Drugs causing granuloma: ▪ Phenylbutazone. ▪ Sulfonamides. ▪ Phenobarbitone. ▪ Allopurinol.
2. Drugs causing hepatitis: ▪ Halothane. ▪ Rifampicin. ▪ Aldomet (α-methyl dopa). ▪ INH.	2. Drugs causing portal hypertension: ▪ Vitamin A. ▪ Vinyl chloride. ▪ Arsenic.
3. Drugs causing cirrhosis: ▪ Aldomet. ▪ Methotrexate. ▪ INH.	3. Drugs causing adenoma: ▪ Oral contraceptives pills. ▪ Anabolic steroids.
4. Drugs casing fatty liver: ▪ Tetracycline. ▪ Valproic acid. ▪ Oral contraceptives pills. ▪ Methotrexate. ▪ Steroids.	4. Drugs causing angiosarcoma: ▪ Arsenic. ▪ Vinyl chloride.
5. Drugs causing fibrosis: ▪ Methotrexate.	5. Drugs causing veno-occlusive disease: ▪ Cyclophosphamide.
6. Drugs casing necrosis: ▪ Heavy metals. ▪ Paracetamol (acetaminophen). ▪ Halothane. ▪ Cocaine.	6. Drugs causing peliosis hepatitis: ▪ Oral contraceptive pills. ▪ Anabolic steroids

LIVER FUNCTION TESTS

1. Protein related tests:

A. Chemical separation of protein:

Protein	Normal value
Total plasma proteins	7 – 9 gm %
Albumin	4 – 5 gm %
Globulin	2 – 3 gm %
Fibrinogen	0.24 gm %

B. Electrophoresis:

- Albumin half life is 26 days, so it isn't affected in acute liver disease but decrease in chronic liver disease.
- α 1-globulin decrease in acute & chronic liver diseases.
- α 2 –globulin & β- globulin are excreted by the liver & increase in obstructive jaundice.
- γ- globulin (immunoglobulin) is formed in the RES & increase in acute & chronic liver disease. There are 3 types of γ- globulin:
  i. IgA → increases in alcoholic liver cirrhosis.
  ii. IgM → increases in primary biliary cirrhosis.
  iii. IgG → increases in chronic active cirrhosis & idiopathic liver cirrhosis.

2. Carbohydrate related tests: glucose tolerance & galactose tolerance are non specific tests for the liver.
3. Fat related tests:
   - Quantitative → in obstructive liver disease.
   - Qualitative changes → esterase enzymes decrease in the liver failure → no esterification of cholesterol in the liver → increase cholesterol level in the blood in case of obstruction (normal cholesterol level is 150 – 300 mg/dl).
4. Bile salts & pigments:
   - Bile salts: they appear in urine in obstructive jaundice → frothy urine with + ve hay sulfur test.
   - Bile pigment:
     - Bilirubin in the blood is normally < 1 gm / dl.
     - Urobilinogen in urine is normally 0.5 – 0.2 mg /day while stercobilinogen in stool is normally 20 – 200 mg / day.
5. Homeostasis related tests:
   - Clotting factors: all decrease in chronic liver disease except factor VIII.
   - Prothrombin time (PT):
     - Normally 12 – 16 seconds.
     - Prolonged in: Liver cell failure → not improved by IV vitamin K.
       Obstructive jaundice → improved by IV vitamin K.
6. Ammonia level in the blood: normally 0.8 – 1 μg/ml.
7. α-fetoprotein: Normal level is < 10 ng /dl.
   - It increases in cases of: cirrhosis – pancreatitis – GIT malignancies.
   - Levels > 2000 ng /dl indicate malignancy (hepatoma).
8. Liver enzymes:

Enzyme	Normal value
SGOT (AST)	5 – 8 IU /ml
SGPT (ALT)	5 – 42 IU /ml
Alkaline phosphatase	3 – 13 king Armstrong units It increase in liver malignancy, obstructive jaundice (marked increase) & bone disease
γ- glutamyle transpeptidase – ornithine cabamyl transferase	

JUNDICE

Def.: yellowish discoloration of the skin & mucous membranes due to increased serum bilirubin above 2 mg /dl.

Normal bilirubin level is up to 1 mg /dl.

Sub clinical jaundice: is a laboratory finding in which bilirubin level is 1 – 2 mg/dl.

Types & causes of hyperbilirubinemia

A. Unconjugated hyperbilirubinemia:

1. Increased production: hemolytic anemia – infective erythropoiesis.
2. Displacement form albumin by drugs as salycilates & oral anticoagulant.
3. Decreased uptake by the liver: liver disease – Gilbert syndrome.
4. Decreased conjugation :
   - Liver disease.
   - Deceased enzymes as in Crigler – Najjar syndrome.
   - Inhibited enzymes as in breast milk jaundice & lucey-Driscoll syndrome.

B. Conjugated hyperbilirubinemia: due to cholestasis (obstructive jaundice) either intrahepatic or extrahepatic.

Types of jaundice

1. Hemolytic jaundice. 2. Obstructive jaundice. 3. Hepatocellular jaundice.

1. Hemolytic jaundice:
   - **Causes**: same causes of hemolytic anemia.
   - **Pathophysiology**:
     - Increased RBCs hemolysis →increased hemobilirubin → jaundice.
     - The liver can't pick hemobilirubin completely so part of it is retained in the blood & large part is converted in the liver into cholebilirubin.
     - The excess cholebilirubin is excreted by the liver into the intestine → increased stercobilinogen → dark stool.
     - Increased stercobilinogen absorbed from intestine → increased urobilinogen → urine normal color bur darken on standing.
     - No bilirubin in urine → acholuric jaundice.
   - **Clinical picture**:
     - Jaundice → lemon yellow.
     - Stool → dark.
     - Urine → normal color bur darken on standing.
     - Clinical picture of hemolytic anemia as leg ulcers & pigmentation in sickle cell anemia.
2. Obstructive jaundice:
   - **Causes**:
     - **extrahepatic causes**:
       - Lumen: stones in the common hepatic duct or common bile duct.
         Mass of ascaris worms.
       - Wall: stricture either congenital or traumatic.
         Tumors, choledochal.
       - Outside: cancer head of pancreas.
         Enlarged LNs at porta hepatic.

- **Intrahepatic causes:**
  - Chronic hemolysis → inspissated bile.
  - Watt's syndrome → cholestatic type of viral hepatitis.
  - Primary biliary cirrhosis.
  - Pregnancy (3rd trimester).
  - Dubin-Jonson syndrome → jaundice without pruritus & green-black liver.
  - Rotor syndrome → the same but with normal-colored liver.
  - Drugs: oral hypoglycemics, Oral contraceptives, Oral anticoagulants, PAS, anabolic steroids, chlorpromazine.

- **Clinical picture:**
  - Jaundice → olive green.
  - Urine → dark & frothy.
  - Stool → steatorrhea.
  - Pruritus & bradycardia.
  - Xanthoma & xanthelasma.

3. Hepatocellular jaundice:

- **Causes:**
  - Same causes of liver cell failure.
  - Gilbert's disease → defect in the transport of bilirubin from blood to the site of conjugation in the liver.
  - Crigler-Najjar syndrome.
  - Breast milk jaundice → inhibition of the conjugating enzymes by a factor present in the maternal immunity.
- **Pathophysiology:**
  - Liver can't extract & excrete all bile pigments.
  - Part of hemobilirubin is retained in the blood → increased indirect bilirubin.
  - Part of cholebilirubin regurgitates into the blood → increased direct bilirubin.
  - Cholebilirubin & some bile salts appear in urine → dark frothy urine.
  - Stercobilinogen absorbed from the intestine can't be fully re-extracted by the liver → increased urobilinogen.
  - Decreased stercobilinogen → pale stool.
- **Clinical picture:**
  - Jaundice → orange yellow.
  - Urine → dark & frothy.
  - Stool → pale.
  - Clinical picture of liver cell failure.

Investigations:

A. Laboratory:

1. Blood picture for hemolytic jaundice.
2. Liver function test (bilirubin > 2 mg /dl).
3. Urine & stool analysis.

B. Radiological:

1. Plain x-ray: for GB stones.
2. US: for GB stones, liver cirrhosis & epigastric mass.
3. Barium study:
   - Stones.
   - Cancer head of pancreas → wide C-shaped duodenum.
   - Cancer ampulla of vater → inverted 3 shaped duodenum.
4. CT & MRI.

C. Endoscopy:

1. PTC: done after US shows dilated intrahepatic radicals.
2. ERCP: shows pancreatic duct, CBD, stones, dilatation, stricture & filling defects.
3. Laparoscopy & laparotomy.

D. Steroid test:

1. In intrahepatic biliary obstruction → obstruction may be relieved.
2. In extrahepatic biliary obstruction → obstruction will not be relieved.

Treatment

A. Hemolytic jaundice: treatment of hemolytic anemia.

B. Hepatocellular jaundice: treatment of the cause.

C. Obstructive jaundice:

1. Medical treatment:
   - Cholestyramine for pruritus.
   - Calcium for osteoporosis.
   - Fat soluble vitamins A – D – E – K.
   - Medium chain triglycerides.
2. Surgical treatment.

OBSTRUCTIVE JAUNDICE

Items	Intrahepatic obstruction	Extrahepatic obstruction
Site	In the liver	In the bile duct
Liver	Enlarged	Markedly enlarged
Gall bladder	Not palpable	May be palpable
fever	Present	Absent
Steroid test	+ ve (relieved)	- ve (not relieved)
Investigations	Liver function tests Complete blood picture PTC & ERCP Alkaline phosphates < 30KAU/ml	x-ray , US , CT & MRI barium study urine & stool analysis Alkaline phosphates > 30KAU/ml
Plain x-ray	- ve	+ ve (stones)

Items	Calcular obstruction	Malignant obstruction
Age	Middle age	Old age
sex	More in females	More in males
Onset	Acute	Gradual
Course	Intermittent	Progressive

Duration	Long	Shirt < 2 years
Pain	Colicky in the right hypochondrium & radiates to the right shoulder	Epigastric & radiates to the back
Cachexia	Absent	Marked
Ascites	Absent	May be present
GB	Not palpable	Palpable
Serum amylase	Normal	Very high in cancer head of pancreas

LIVER TRANSPLANTATION

1. History:

- The 1st operation was done in 1963.
- Now, about 5000 operation are performed every year with one year survival rate about 85%.

2. Indications: irreversible & progressive liver disease as:

- Liver cirrhosis.
- Fulminant liver hepatitis.
- Primary biliary cirrhosis.
- Hepatocellular carcinoma.
- Budd-chiari syndrome.
- Sclerosing cholanagitis.

3. Contraindications:

- Absolute contraindications:
  1. Active sepsis.
  2. AIDS.
  3. Metastatic malignancy.
  4. Advanced cardiopulmonary disease.
  5. Cholangiosarcoima.
- Relative contraindications:
  1. Old age.
  2. Impairment of renal function.
  3. Portal vein thrombosis.
  4. Previous Portocaval shunt.

4. Complication:

- Primary graft non-functioning.
- Graft rejection.
- Infection.
- Recurrence of the primary disease.
- Complications of immunosuppressives as recurrence & metastasis.

5. Types of operation:

- Orthoptic liver transplantation: the patient's liver is removed & the donor's liver is put in place.
- Auxiliary liver transplantation :
  - Transplantation of the donor's liver without removal of the whole liver of the recipient.
  - Advantage: original liver may regenerate →stop immunosuppression.
  - Disadvantages: possibly of malignant transfusion.
- Split liver graft transplantation:
  - In young children.
  - The donor's liver is split into parts which can be used for 2 patients instead of one.
- Living related donation: part of relative's liver in transplanted to the patient.
- Xenotransplantation: using liver from animals.

6. Immunosuppressives: cyclosporine, Azathioprine & prednisone for 13 years.