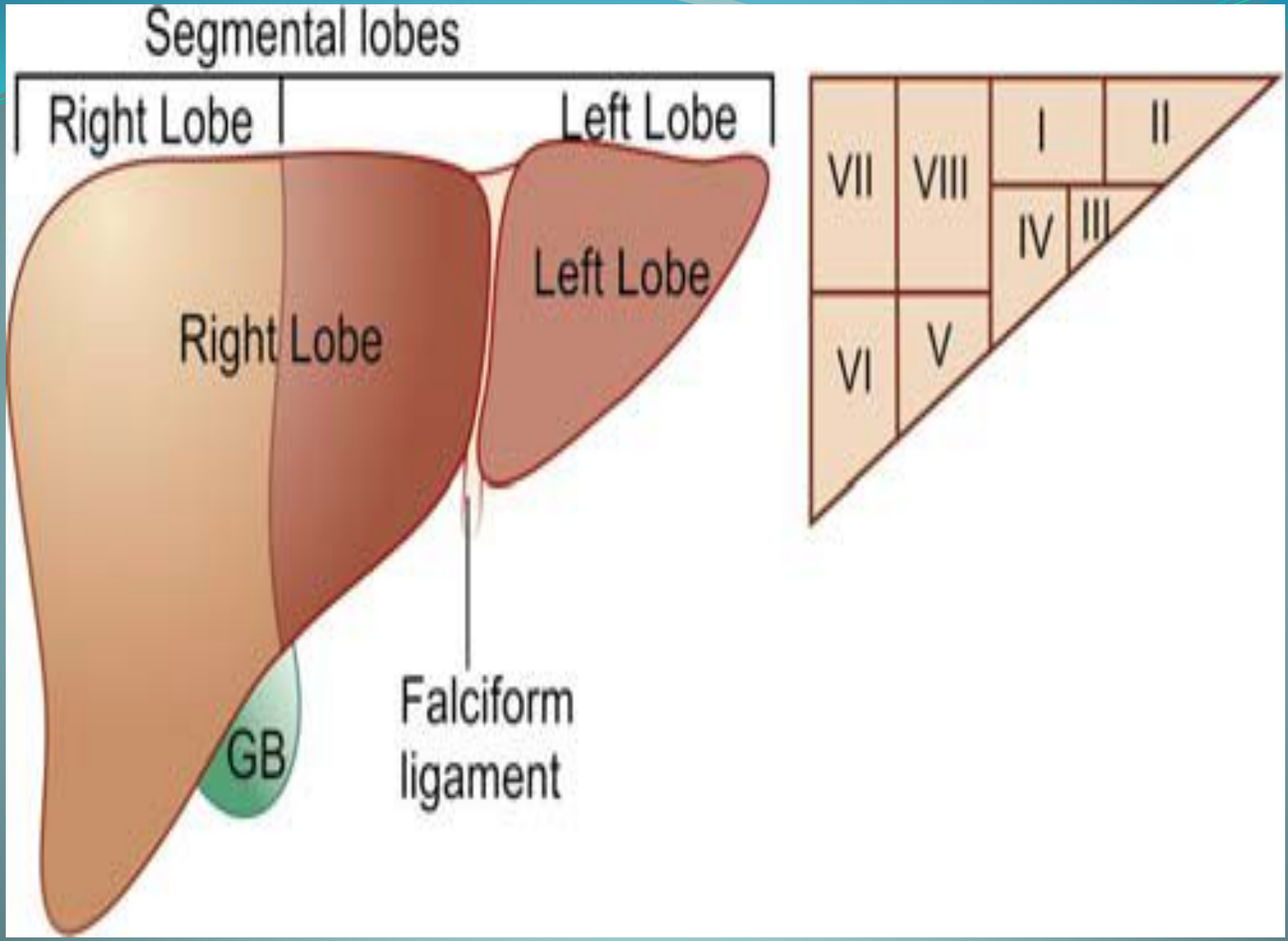
INTRODUCTION

The liver is the largest organ in the body, weighing 1.7 kg in the average 80-kg man. It sits in the right upper quadrant beneath the diaphragm, and is protected by the rib cage. The liver parenchyma is entirely covered by a thin capsule(Glisson's capsule) and by visceral peritoneum on all but the posterior surface of the liver, termed the 'bare area'. The liver is divided into a large right lobe, which constitutes three-quarters of the liver volume, and a smaller left lobe.


Segmental lobes
Right Lobe
Left Lobe
Left Lobe
Right Lobe
GB
Falciform
ligament
VII
VIII
I
II
IV
III
VI
V

Segmental Anatomy of Liver

Liver is divided into functional right and left lobes by a line passing from the left of the gallbladder fossa to the left of IVC—*Cantlie's line creating Couniaud's segments. There are eight segments:*

Segments I, II, III, and IV are of left lobe.

Segments V, VI, VII, VIII are of right lobe.

Segment I is the caudate lobe of the liver and has independent supply of portal and hepatic veins. This hepatic vein directly joins IVC. Right lobe is having right hepatic artery, right branch of portal vein, and right hepatic duct.

Left lobe is having left hepatic artery, left portal vein, left branch of bile duct. *Functional unit is called as hepatic lobule and it contains central hepatic vein and portal triad (hepatic arteriole, portal venule, bile ductule).*

- **Functions of the liver**
- Removal of gut endotoxins and foreign anti gens—liver acts as the first filter
- Drug and hormone metabolism
- Formation of bilirubin and its metabolism
- Formation of urea from protein catabolism
- Glucose metabolism, glycolysis, and gluconeo genesis
- Clotting factors synthesis
- pH balance and correction of lactic acidosis
- Maintaining body temperature
- Storage of vit. B12, vit. A, Cu, Fe

1. Serum bilirubin which includes both direct and indirect. Test is known as *van den Bergh's test.*
2. Serum albumin, globulin and A : G ratio; serum albumin is the indicator of chronic liver disease.
3. Prothrombin time: Normal value is *12-16 seconds. Difference* between control and test more than 4 seconds or test being more than 1½ times the control is significant. When it is altered it is corrected by injecting vitamin K, 10 mg IM for 5 days or by fresh frozen plasma (FFP)—*For cell synthesis.*
4. Alkaline phosphatase—*Secretary function.*
5. Aspartate amino transaminase *5-40 IU/litre (AST,* SGOT)—*signifies inflammation.*
6. Alanine transaminase *5-40 IU/litre (ALT, SGPT), liver* specific.
7. 5 nucleotidase.
8. Gamma glutamyl transpeptidase (GGT) 10-48 IU/L.
9. Immunological tests: Antimitochondrial or anti nuclear antibodies.
9. (*Hay's test), for bile pigments* (*Fouchet's test) and for urobilinogen (Ehrlich's aldehyde test).*

10. AFP.
11. Specifi c tests: (a) For haemochromatosis: Serum iron, total iron binding capacity, serum ferritin. (b) Wilson's disease: Serum copper, urinary copper, serum ceruloplasmin.
12. Fluorodeoxyglucose-positron emission tomo graphy (FDGPET):
It is to find out the uptake of labelled glucose which varies in different diseases of liver, i.e. benign, malignant and infl ammatory.
13. Technetium-99m labelled radioisotope scan shows the uptake and excretion of bile.
14. A sulphur colloid liver scan shows specifically Kupffer cell activity. Sulphur colloid will not show uptake in adenoma and haemangioma as Kupffer cells are absent in these lesions.
15. Urine for bile salts (*Hay's test), for bile pigments* (*Fouchet's test) and for urobilinogen (Ehrlich's aldehyde test).*

Hepatocyte function

AST, ALT

Synthetic function and metabolism

PT-INR, factor V. VII, albumin, bilirubin

Biliary canalicular function ALP, 5' nucleotide, gamma glutamyl transferase, bilirubin

Other investigations for liver diseases

U/S abdomen
Angiography
CT Scan
PTC
ERCP
MRI
Laparoscopy and laparoscopic U/S
Liver biopsy

LIVER BIOPSY

Prerequisites:

Prothrombin time should be normal before doing liver biopsy, otherwise severe bleeding can occur.

Indications

1. Cirrhosis, chronic hepatitis, haemochromatosis, Wilson's disease.
2. Hepatoma, secondaries in liver.

Complications

Haemorrhage.

Bile leak and biliary peritonitis.

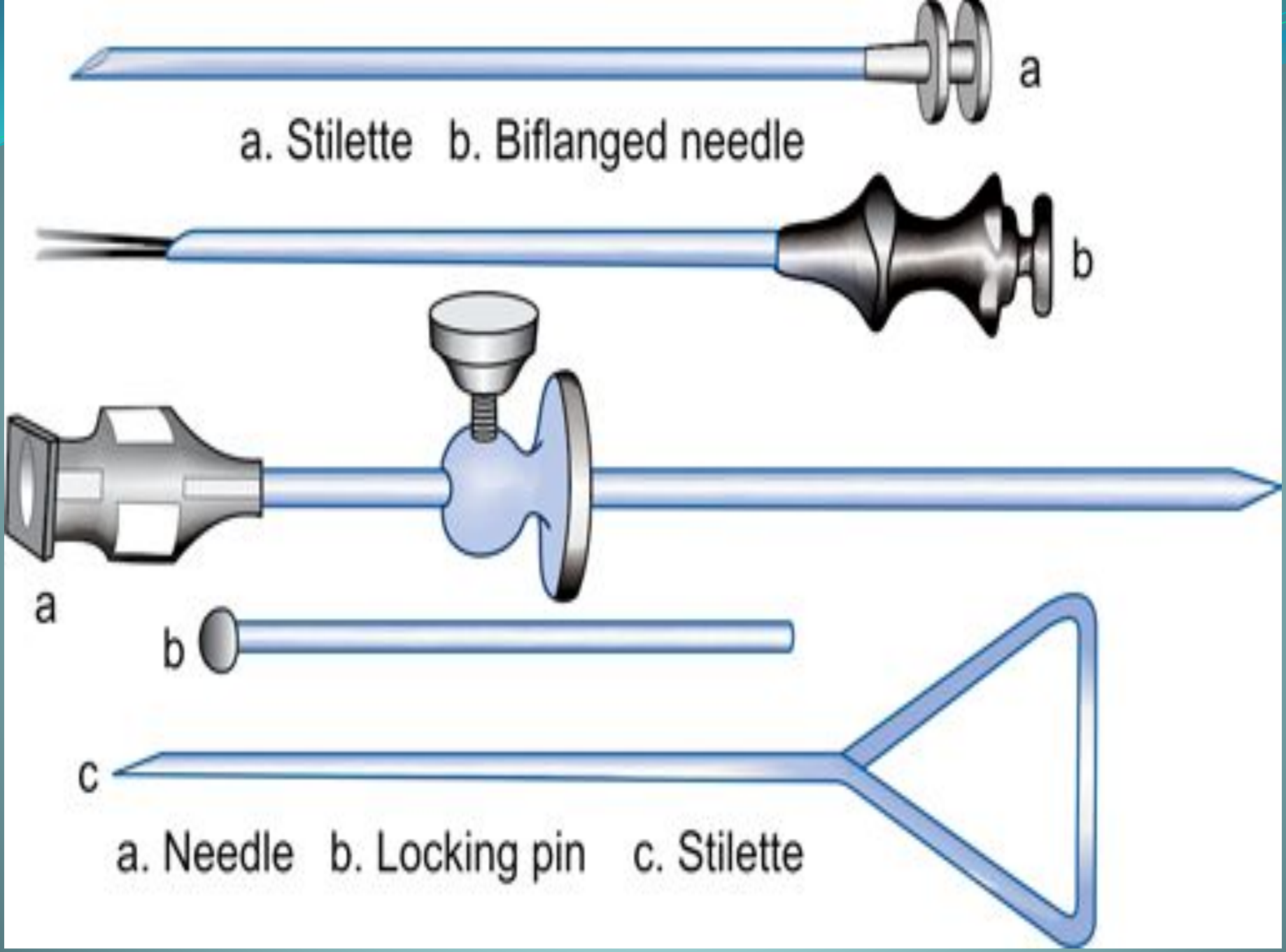
Infection.

Contraindications

Hydatid disease, where it will precipitate anap hylaxis.

Haemangioma, bleeding disorders.

Ascites.


a
a. Stilette b. Biflanged needle
b
a
b
c
a. Needle b. Locking pin c. Stilette

INFECTIONS OF LIVER

AMOEBIC LIVER ABSCESS

It is common in India and other tropical countries and it is caused by a parasite entamoeba histo lytica.

It is more common in *alcoholics and cirrhotic patients*.

It is the commonest extraintestinal presentation of amoebiasis.

It is often called *as tropical abscess*.

Infection commonly occurs from the caecum after an attack of amoebic typhlitis (infl ammation of the caecum) through the superior mesenteric vein and portal vein. Infection from sigmoid (rectosigmoid) colon spreads through the inferior
mesenteric vein and portal vein to liver. Right lobe is commonly involved over posterosuperior surface (because of streamline effect and larger size of the right lobe). Trophozoites destroy the hepatocytes by releasing *histiolysin, a* cytolytic agent. It causes amoebic hepatitis with multiple microabscesses formation. It leads into liquefaction necrosis, thrombosis of blood vessels, release and, breaking of red cells. It causes formation of "*Anchovy sauce' pus which is chocolate* brown coloured and odourless (Anchovy sauce is sauce prepared from a type of fish). Pus may be green coloured if mixed with bile. Secondary infection is common (30%).

Pathology

Initially from infected rectosigmoid or ileocaecal region, amoebic trophozoites reach the liver through portal veins causing amoebic hepatitis, may be in the form of microabscesses all over the liver. This might resolve on its own or with anti amoebic drugs, but often leads to a localized *amoebic liver abscess.*

In 70% of cases it is *single large abscess, in 30% it is* multiple, may involve both lobes. Problems and diffi culties in treating, in addition to poor prog nosis are more common in *multiple abscesses.*

Amoebic liver abscess is more common in *right posteriorsuperior* region (80%) because of streamline effect, i.e. the portal vein is in direct continuation with the right branch. It can be *multiloculated also.*

•Course and Sequelae of Amoebic

•Liver Abscess

- It can *rupture into lungs leading to expectoration of chocolate-* coloured sputum resulting in natural regression of abscess—commonest site of rupture.
- It can rupture into the peritoneum causing *perito nitis which* requires emergency laparotomy.
- It can rupture into pleural cavity leading to *empyema.*
- Rupture into bronchus can cause bronchopleural fi stula leading into coughing out of Anchovy sauce pus.
- Rupture into bare-area of liver causing *retroperitoneal abscess.*
- Rupture into the intestines, or to the skin (*Amoebias is cutis).*
- Most dangerous complication is rupture into pericardial cavity (*cardiac tamponade) which has very high mortality* (30%) requiring emergency thoraco tomy and pericardial decompression.
- *Septicaemia and liver failure can occur in a patient with* amoebic liver abscess with *cirrhosis*

Clinical Features

It is common in males (20:1), may be after an attack ofnamoebic dysentery or many months after the attack orn history of dysentery may not be there at all.

They present with fever, loss of weight, chills and rigors, non-productive cough, shoulder pain.

Pain in the right hypochondrium—90%.

Soft, tender, smooth, liver with increased liver span—70%.

Right sided pleural effusion may be evident.

Mild jaundice is not uncommon especially in cirrhotics and multiple abscesses which may signify poor prognosis—20%.

Tenderness, rigidity and skin oedema in right hypochondrium may be present in acute cases.

In chronic amoebic liver abscess, smooth, firm/hard, nontender liver may be palpable.

Investigations

Total count may be increased.

Liver function tests may show altered bilirubin and albumin level.

Prothrombin time may be widened and if it is so Inj. vit K 10 mg IM for 5 days should be given. Even with this if P.T. remains widened then fresh frozen plasma (FFP) is needed to rectify the P.T.

Serum alkaline phosphatase, SGPT, SGOT levels are altered.

U/S abdomen shows altered echogenicity (anechogenic, hypoechogenic), size, location, number of abscess, nature of the liver—90% sensitivity.

CT scan-contrast study. CT scan shows raised diaphragm; abscess cavity (low density area)—its size, location, number; presence of effusion; changes in the lung—95% sensitivity.

Sigmoidoscopy/colonoscopy are used to identify the active ulcers. Scrapings of the ulcer show trophozoites.

Treatment

Drugs

Tab. metronidazole 800 mg tid or Inj. metroni dazole 500 mg IV tid for 10 days.

Tinidazole 600 mg BD dose for 5 days.

IV or oral antibiotics are essential to control secondary infection (cefotaxime, ciprofl oxacin, amoxycillin) (Small abscesses < 3 cm respond to drugs).

Percutaneous Drainage

Under U/S guidance pigtail catheter is placed into the abscess cavity percutaneously to drain the pus. Catheter tube and abscess cavity has to be washed and irrigated at regular intervals with normal saline. It may fail if there is thick pus, multiloculated abscess, and multiple abscesses. Procedure may cause bleeding and infection.

Indications for surgery

Even after repeated aspirations if abscess cavity fi lls again

Thick pus

Multiloculated abscess

Left lobe abscess, because of danger of rupture into pericardial cavity

Ruptured abscess

Caudate lobe abscess

Multiple abscesses

PYOGENIC LIVER ABSCESS

Aetiology

1. *Biliary sepsis 35%; commonest route.*

a. Empyema gallbladder.

b. Cholangitis.

c. After biliary tract surgery.

d. Instrumentation.

e. Stone disease, Caroli's disease, biliary ascariasis, biliary enteric anastomosis.

2. *Portal vein sepsis:*

a. Appendicitis.

b. Diverticulitis.

c. Infl ammatory bowel disease, pancreatitis, perforation, PID, colorectal carcinoma.

d. Omphalitis in newborn.

3. *Distant infections (through hepatic artery):*

a. Pneumonia.

b. Upper UTI.

c. Endocarditis, osteomyelitis, bacteraemia

4. *Super added infections*:

a. Amoebic liver abscess.

b. Hydatid cyst.

5. *Cryptogenic liver abscess—No identifi ed primary infection.*

6. *Trauma becoming common cause.*

7. *Direct extension:* From suppurative cholecystitis, subphrenic abscess, perforation, perinephric abscess.

Causative bacteria

E. coli—commonest

Klebsiella

Proteus

Pseudomonas

Clostridia

Enterococci, streptococci viridians in polymicrobial infection

Pathology

Due to laminar blood fl ow right lobe (75%) is commonly involved; left lobe (20%), caudate lobe (5%) are also often involved.

Usually solitary—60%; occasionally it can be bilobar and multiple.

Cavity contains pus with virulent organisms. Usually abscess is acute. In cryptogenic type chronic presentation is known to occur.

Ascites and splenomegaly is not common.

It is more common in diabetics.

Male to female ratio is *2:1. It is more common in old people* after 55 years of age.

Blood culture commonly shows positive for bacteria.

Clinical Features

Pain in the right hypochondrium—60%.

High fever, with rigors—90%.

Weight loss.

Jaundice—occasionally—20%.

Intercostal tenderness.

Tender, soft liver-60%.

Features of toxicity.

Constitutional symptoms like malaise, lethargy, vomiting.

- **Diagnosis**
- Ultrasound abdomen, CT scan. Sensitivity is 90% for USG; 97% for contrast CT scan.
- LFT, total count.
- Ultrasound guided aspiration of pus after controlling PT.
- Chest X-ray shows elevated diaphragm often with right sided pleural effusion.
- Blood culture is very relevant.

Differential Diagnosis

Amoebic liver abscess, hydatid cyst, subphrenic abscess.

Treatment

Systemic antibiotics—combination of third gener ation cephalosporins and metronidazole.

Ultrasound guided aspiration/pigtail catheter—Percutaneous drainage is the treatment of choice at present.

Complications

Septicaemia, liver failure.

Rarely rupture and peritonitis can occur.

Klebsiella hepatic abscess can cause dangerous endogenous endophthalmitis commonly in diabetic patients impairing vision.

PORTAL PYAEMIA (PYLEPHLEBITIS)

It usually follows after severe infection of areas drained by portal vein.

Causes

Appendicitis.

Diverticulitis.

Any severe abdominal sepsis.

Presently it is becoming *rare because of availability of good* effective antibiotics.

Infective thrombus in the vein (draining the infective area)

↓

It dislodges as infective emboli

↓

Reaches the liver

↓

Causes multiple abscesses in liver parenchyma

↓

Pylephlebitis.

E. coli is the most common organism. Often staphylococci, anaerobes, or combined infections may be involved.

Clinical features

High fever with rigors

Toxicity

Drowsiness, jaundice

Tender soft liver

Ascites

Blood culture is a useful investigation

Treatment

Antibiotics: Combination of third generation cepha losporin + aminoglycoside, ceftriaxone sodium, cefoperazone, ceftazidime, amikacin, tobramy cin, metronidazole.

IV fl uids, blood transfusion, FFP. Ventilator support.

Mortality is very high and usually patients die of hepatic failure, septicaemia, MODS.

It is better to prevent portal pyaemia by prior good antibiotics in suspected cases.

Treatment of primary cause is very important.

HYDATID CYST OF LIVER

Word meaning is '*dew drop' (Latin). In Greek it means 'watery* vesicle'.

Caused by **Echinococcus granulosus (EG), *dog tape worm,*** a parasite.

Life-cycle: Infected offal of sheep

↓

Eaten by the dog (defi nitive host)

↓

EG released, develops in the dog's intestine into a parasite of 1 cm long with a head and three segments, last of which contains about 500 ova.

↓

Ova expelled from the dog's intestine to grass and vegetables

↓

Eggs are ingested by sheep, cattle or human beings *(intermediate host)*

↓

Through portal vein—liver—larva form

↓

Hydatid cysts (70% in liver).

Hydatid cysts (70% in liver).

It takes few years to evolve into a complete hydatid cyst.

Most commonly involved segment is segment VII—27%.

Commonly right lobe—66%; both lobes in 16% and only left lobe is involved in 17%

Pathology

It has got 3 layers

1. *Adventitia (pseudocyst) is an inseparable fi brous tissue due* to reaction of the liver to the parasite.

2. *Laminated membrane (ectocyst), formed of the parasite itself* is whitish, elastic, containing hydatid fl uid, which can be peeled off readily from the adventitia.

3. *Germinal epithelium is the only living part, lining the cyst* (*endocyst).m* This layer secretes *hydatid fl uid, brood capsules with scolices* (heads of future worms).

Course of the Disease

The parasite may die and cyst eventually may get *calcifi ed.*

Commonly cyst *enlarges and is palpable per abdomen.*

It may cause complications like *jaundice due to pressure* over biliary tree.

Rupture into the peritoneal cavity causes anaphylactic reaction which may cause life-threatening shock, requiring proper management with steroids.

Rupture into biliary channels is commonest (60%). Rupture into bowel, pleural cavity can occur.

Secondary infection causing suppuration and septicaemia.

Secondary cysts in the lung, spleen, mesentery, retroperitoneum and other organs can occur**.**

Hepatic dysfunction.

Disseminated abdominal hydatidosis can occur after silent rupture.

Clinical Features

Asymptomatic palpable liver with *classical thrill (hydatidn thrill) elicited by three-fi nger test.*

Jaundice and pain.

Features of anaphylaxis.

Discomfort in right upper quadrant area; dyspepsia; hydatid cachexia in children; weight loss; fatigue; vomiting.

Occasionally splenomegaly, pleural effusion, cholangitis,n allergic asthma, fever.

Camellotte sign: Following intrabiliary rupture, gas enters into cyst causing partial collapse of the cyst wall.

Differential Diagnosis

Hepatoma.

Amoebic liver abscess.

Cystic disease of the liver.

Investigations

U/S is diagnostic. It reveals rosettes of daughter cysts,n double contoured membrane of the cyst due to detachment of the cyst membranes, and calcifi cation of cyst wall. X-ray often shows calcifi cation.

CT scan abdomen is more accurate in identifying cyst characteristics—

.

Primary serological tests—ELISA; indirect haemagglutination test; latex agglutination test; immunofl uorescence antibody test;nimmunoelectrophoresis. 80-95% sensitivity for liver hydatid.

Secondary laboratory tests—Detection of precipitation line—arc 5; immunoblotting; polymerase chain reaction (PCR). More specifi c, very useful in extrahepatic hydatid disease and calcifi ed non-fertile liver hydatid.

Liver function tests.

MRI when there is jaundice to visualise biliary tree and its relation to hydatid cyst; to find out cystobiliary communication; biliary hydatids in bile duct and hepatic ducts.

ERCP can also be done to fi nd out the communications.

Treatment

Drugs

Indications for drug therapy

4 days prior to intervention and to continue it for 1 month (albendazole) to 3 months (mebendazole) after the intervention.

Inoperable cysts.

Multiple or multiorgan cysts.

Recurrent hydatids.

Surgically unfi t patients.

Cysts in lungs, bone, brain, eyes.

Contraindications

Large cysts.

Honeycomb cysts (with septae).

Infected cysts

Calcifi ed cysts.

Pregnancy

Drugs used are:

Albendazole 4-week cycles with 2 weeks drug free interval. It is ovicidal/larvicidal/*vermicidal*.

Commonly used.

Praziquantel—60 mg/kg along with albendazole for 2
weeks.

Mebendazole—600 mg daily for 4 weeks.

Surgery

Surgery is still the choice and *gold standard therapy for* hydatid disease. The abdomen is opened, and the peritoneal cavity is packed with mops [black or coloured mops are used to identify white scolices clearly so as to pick up all and prevent any spillage]. Fluid from the cyst is aspirated and scolicidal agents [cetrimide, chlorohexidine, alcohol, hypertonic saline (15%-20%), 10% povidone iodine or H2O2] are injected into the cyst cavity (formalin should not be used)**. Hypertonic saline should be left within the cavity** for 15-20 minutes to have effective scolicidal effect.

Laparoscopic pericystectomy

Liver resection

Cetrimide—can cause acidosis

Alcohol 80%—can cause cholangitis

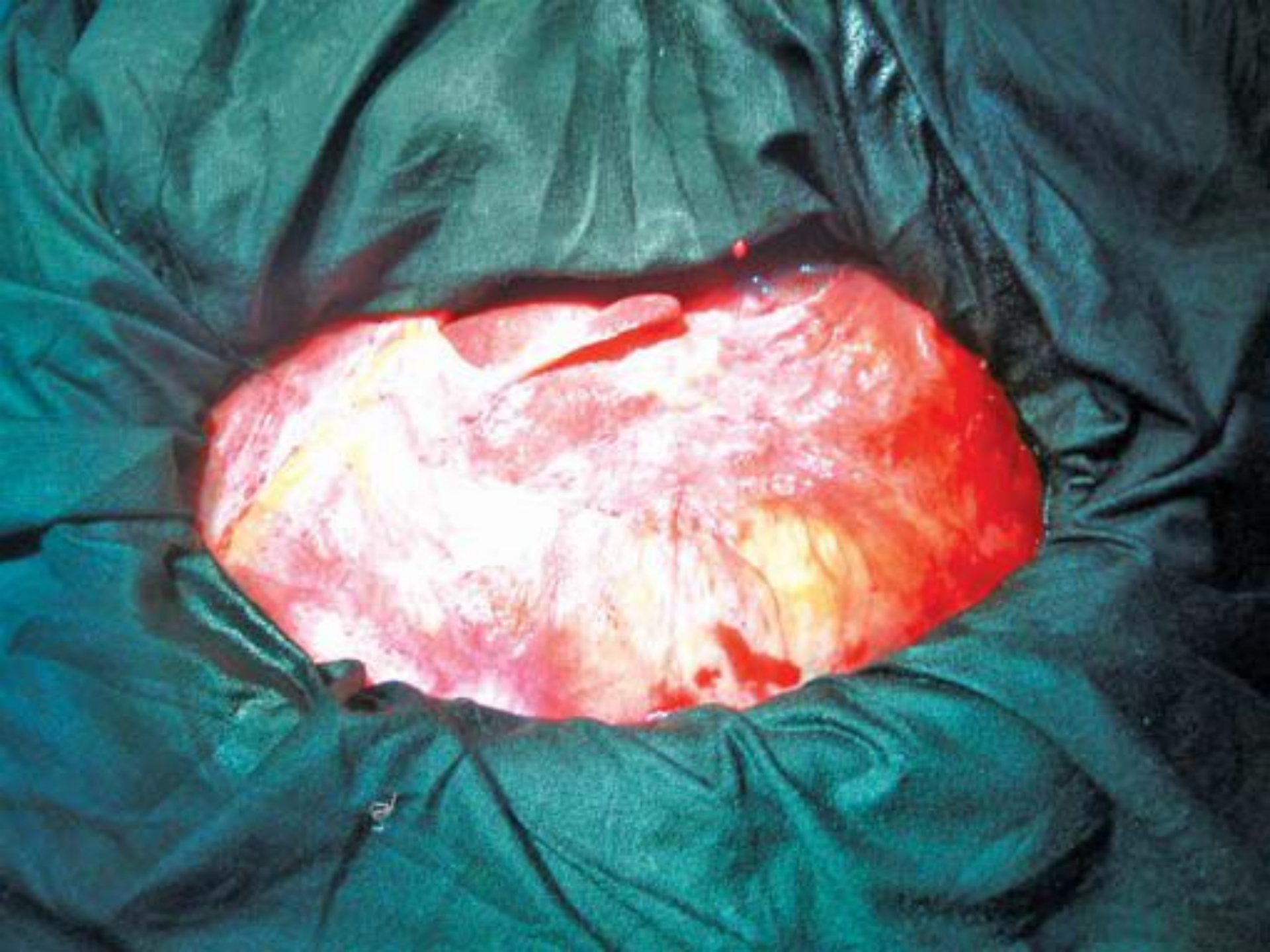
Hypertonic saline—hypernatraemia

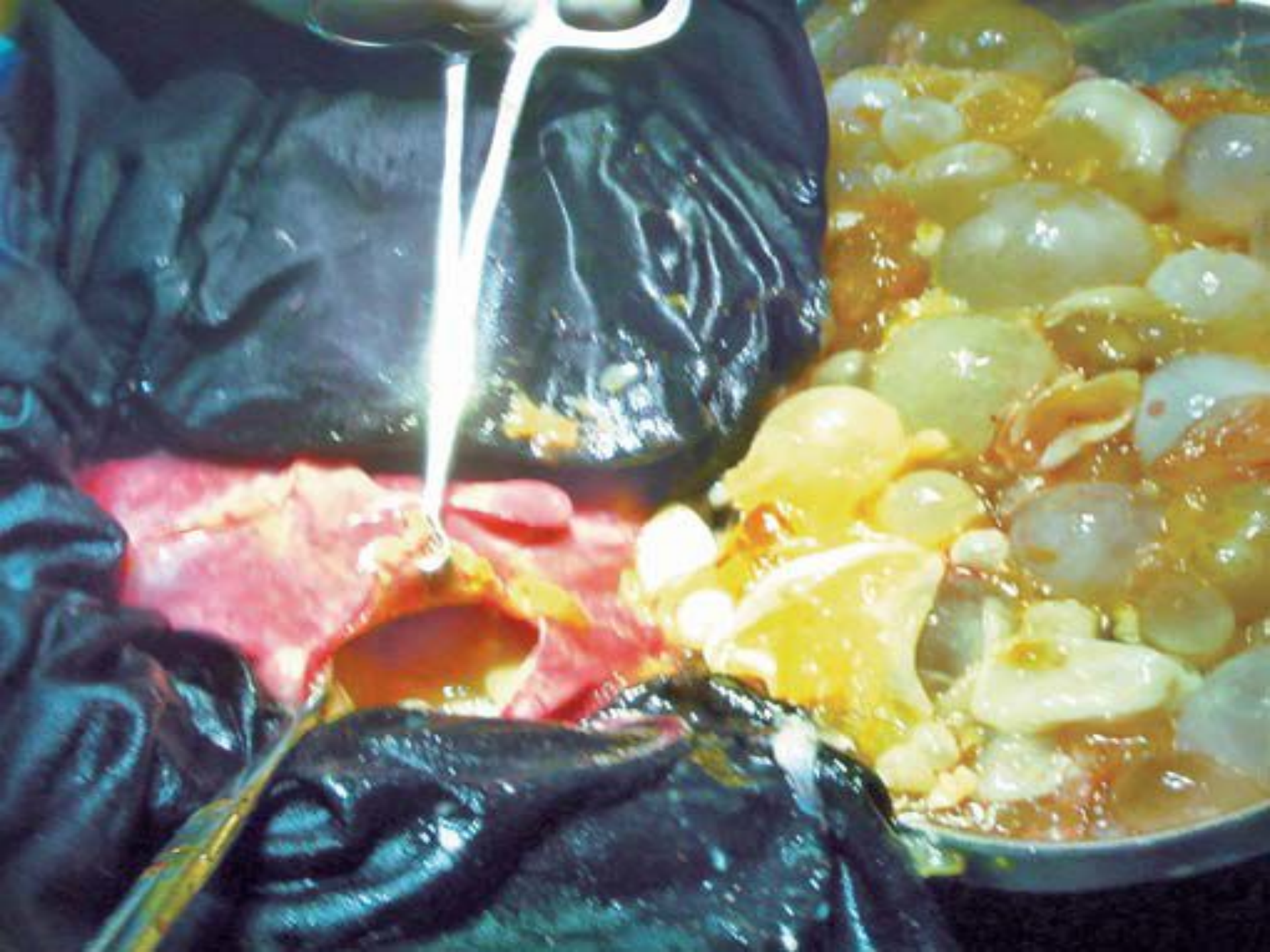
Sodium hypochlorite—hypernataraemia

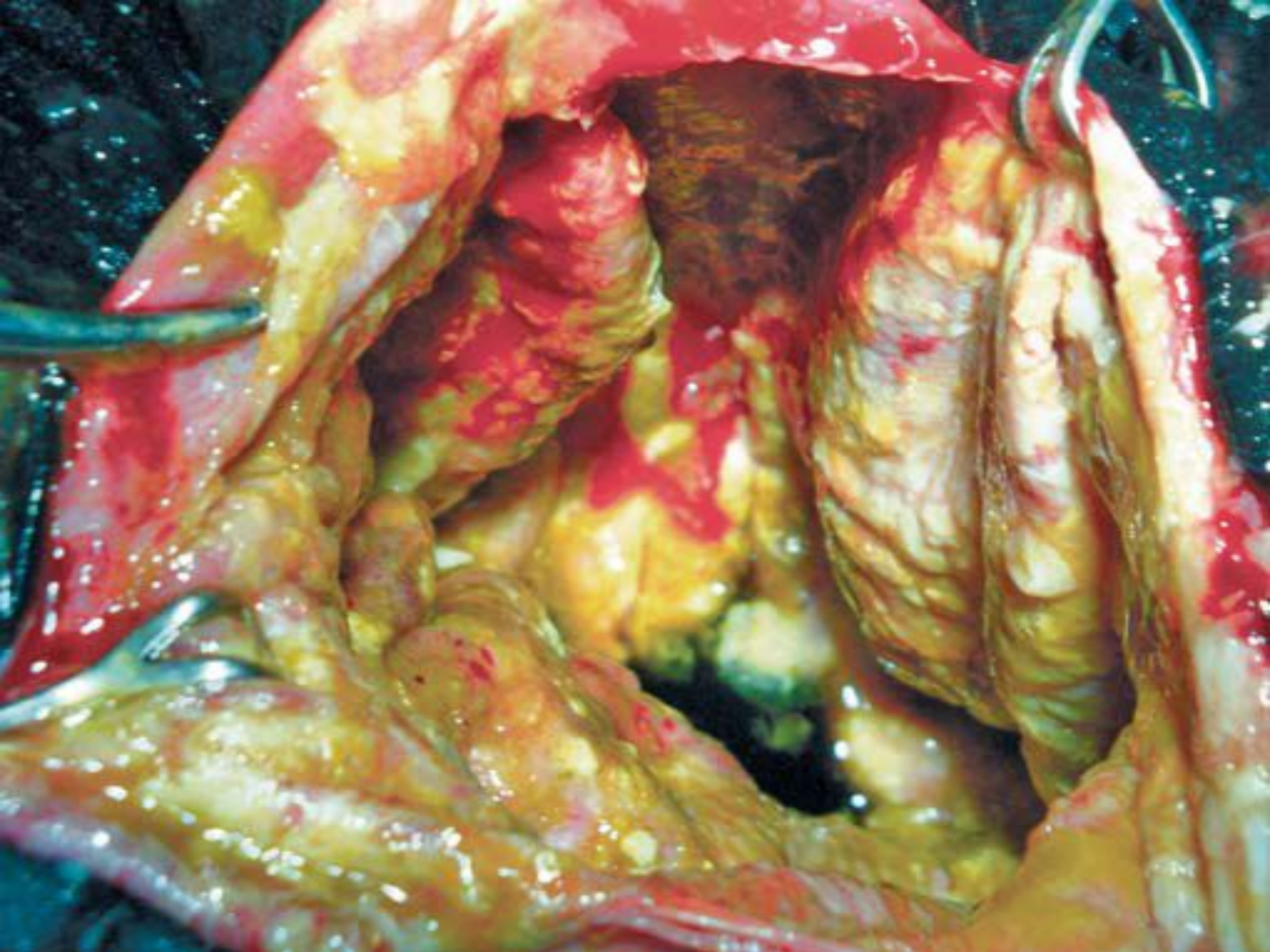
Hydrogen peroxide

In cases with biliary communication only hypertonic saline

(15-20%) is used (not other agents)







Malignant hydatid disease

It is a misnomer as it is a benign condition

It is caused by *Echinococcus multilocularis (Alveolaris). It* presents with multiple small cysts in both lobes of the liver, all over

It is diffi cult to treat, and mimics clinically and prognosis-wise to malignancy, hence the name

They die of liver failure

LIVER TUMOURS

BENIGN TUMOURS OF THE LIVER

Benign tumours of the liver

It is two times common than malignant liver tumor It is seen in 20% of population

Haemangioma is the most common benign tumor

CT and MRI are the essential investigations

MRI shows 95% accuracy

Diagnostic accuracy of liver biopsy is low—40%

Hepatic adenoma is potentially malignant

FNH and haemangiomas are not premalignant

Primary (HCC) tumour or secondaries are differential diagnosis;

AFP and CEA are helpful to differentiate

1. Haemangiomas

They are the *commonest benign tumour of the liver.*

It is usually solitary but can be multicentric.

Compressibility of tumour is diagnostic.

It is common in females (3:1) in 5th decade.

Commonly they are asymptomatic.

Complications

Bleeding rupture

Thrombosis

DIC

Infection

Complication usually occurs if haemangioma is *more than 8 cm in size.*

Needle aspiration and biopsy are contraindi cated.

2. Hepatic adenomas

They are common in females (10:1). They present as asolitary nodular lesions in the liver.

It is said to be due to use of oral contraceptive pills (OCPs).

It is uncommon in males. It is relatively rare compared to FNH and haemangioma

They might turn into malignancy, hence resection is advised.

U/S, CT scan are diagnostic but angiography is needed prior to resection, as it is vascular.

Indications for surgical resection

Rupture of adenomas—50%

If there is possibility of turning into malignancy (large adenomas)

3. Focal nodular hyperplasia (FNH)

It is a benign condition of unknown aetiology, seen in females showing focal overgrowth of func tioning liver tissue with fi brous stroma support.

It contains hepatic cells as well as Kupffer cells which is characteristic.

It is 2nd most common benign tumour.

Usually it presents as solitary nodule.

A sulphur colloid liver scan is diagnostic, shows ‘hot spot’ with a spoke wheel pattern—85%.

It is a harmless condition.

CT scan shows central scar with stellate distri bution of the blood vessels.

PRIMARY MALIGNANT TUMOURS OF THE LIVER

1. Hepatocellular carcinoma/Hepatoma/HCC (80%).
2. Cholangiocarcinoma (20%).
3. Hepatoblastoma in infants and children.

Hepatoblastoma occurs within 2 years of life. It is most common primary malignant tumour of liver in children. It is common in male child. It is derived from fetal or embryonic hepatocytes. Serum AFP is *elevated in 90% of cases. CT scan shows* vascular mass with speckled calcifi cation. It is highly sensitive to chemotherapy

HEPATOCELLULAR CARCINOMA (HCC/HEPATOMA)

Its incidence is rising.

It is common in cirrhotics and hepatitis B and hepatitis C virus infection.

It is common in Mozambique, South East Asia, tropical Africa, Taiwan. In Mozambique it is often seen in younger age group - below 30 years.

Male to female ratio is 4:1.

It is usually unicentric but occasionally can be multicentric.

Right lobe is commonly involved.

Aetiology

1. Afl atoxin B1, a product of fungus aspergillus. It is powerful carcinogen.
2. Hepatitis B and hepatitis C virus infection. It is morecommon in individuals who have chronic positive status for HBs Ag and chronic carriers. Vertical transmission of virus at birth raises HCC rate. In Europe, Japan, USA, HCV infection is more common cause. It is not necessaryn to develop cirrhosis to cause HCC.
3. Alcoholic cirrhosis. It is co-carcinogen.
4. Clonarchis sinensis infestation.
5. Smoking.
6. Haemochromatosis, α1 antitrypsin defi ciency.
7. Hepatic adenoma—potentially malignant.
8. Environment related chemicals like DDT, nitrite and nitrate related food products

Pathology

Gross

It is highly vascular with indistinct margin. Often it is well demarcated by fi brous tissue. Haemorrhage and necrosis are common.

Hanging type of tumour—tumour attached to normal liver by a small vascular stalk—always very well resectable.

Pushing type of tumour—large, well demarcated, displaces normal vasculature—resectable.

Infi ltrative type of tumour—indistinct, infiltrative—often diffi cult to resect.

Histology

Well/moderate/poorly differentiated tumour.

Clinical Features

Painless mass in right hypochondriac region with loss of appetite and weight. Liver is hard, smooth/irregular and often massively enlarged. Cirrhotic liver may be nodular.

Acute presentation is not uncommon, when the tumour undergoes necrosis and haemorrhage. Both types of presentations mimic amoebic liver abscess (Haemoperitoneum is also known).

Jaundice when present is commonly due to hepatic dysfunction, but occasionally due to compression of bile ducts.

Ascites (40%) often it is massive, splenomegaly and features of portal hypertension may be present.

Hepatic thrill and bruit—25%.

Fever (10-20%) may be present due to tumour necrosis.

Dull aching pain in right upper quadrant is common.

Features of chronic liver disease—jaundice, dilated veins, palmar erythema, gynaecomastia, testicular atrophy, etc.

Liver failure sets in once tumour replaces the functioning liver parenchyma or portal vein gets occluded by tumour thrombus.

.Occasionally may present with paraneoplastic syndromes. 1%; hypercalcaemia, hypoglycaemia, hyperlipidaemia, hyperthyroidism, erythrocytosis.

Spread of Tumour

Lymphatic spread: It can spread to other part of liver through lymphatics within the liver, to the lymph nodes in the porta hepatis and other abdominal lymph nodes later. Often spread occurs directly to cisterna chyli.

Blood spread: To lungs, bones and adrenals often can occur.

Direct infiltration: To diaphragm and neighbouring structures.

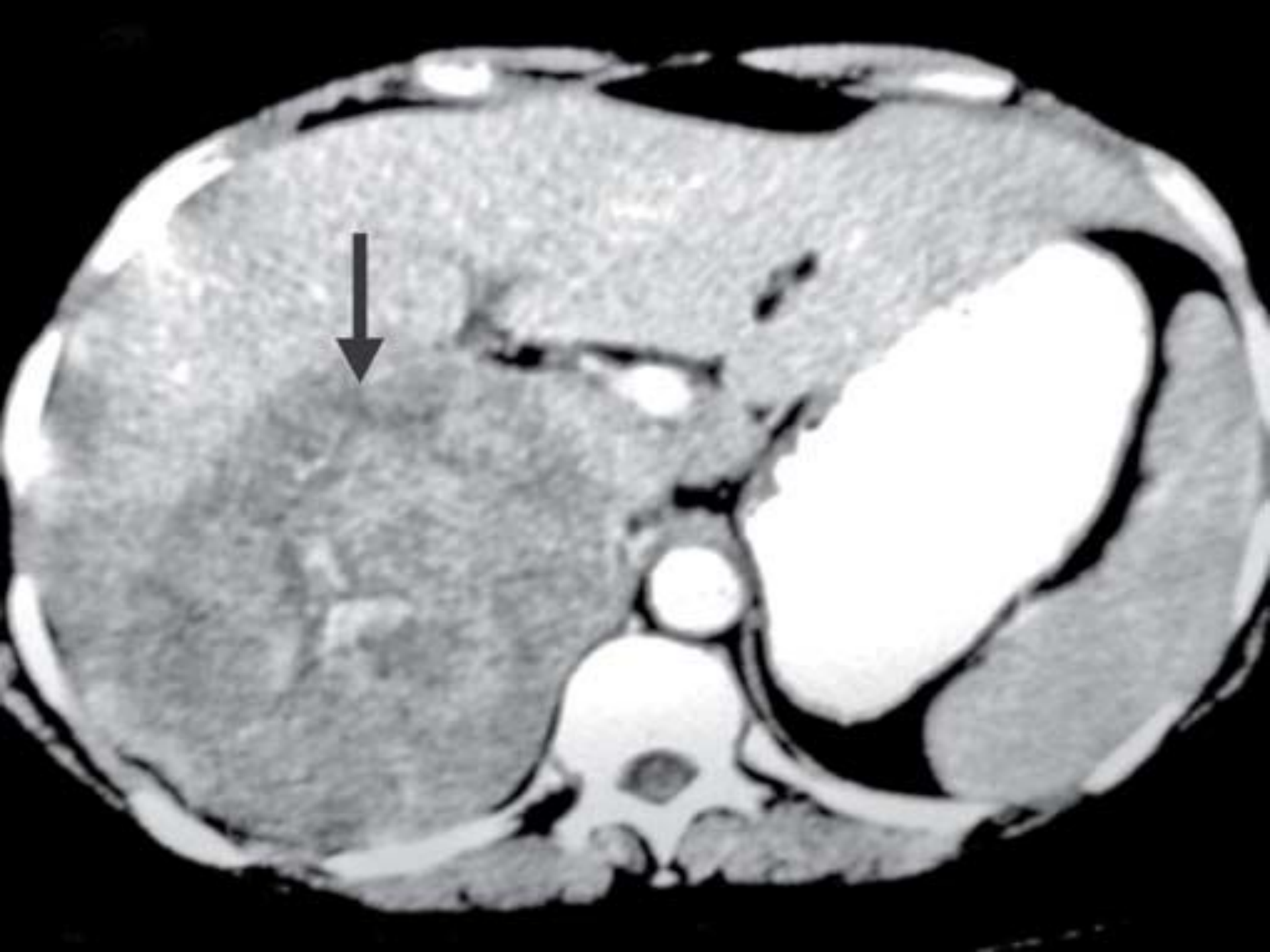
Differential diagnosis

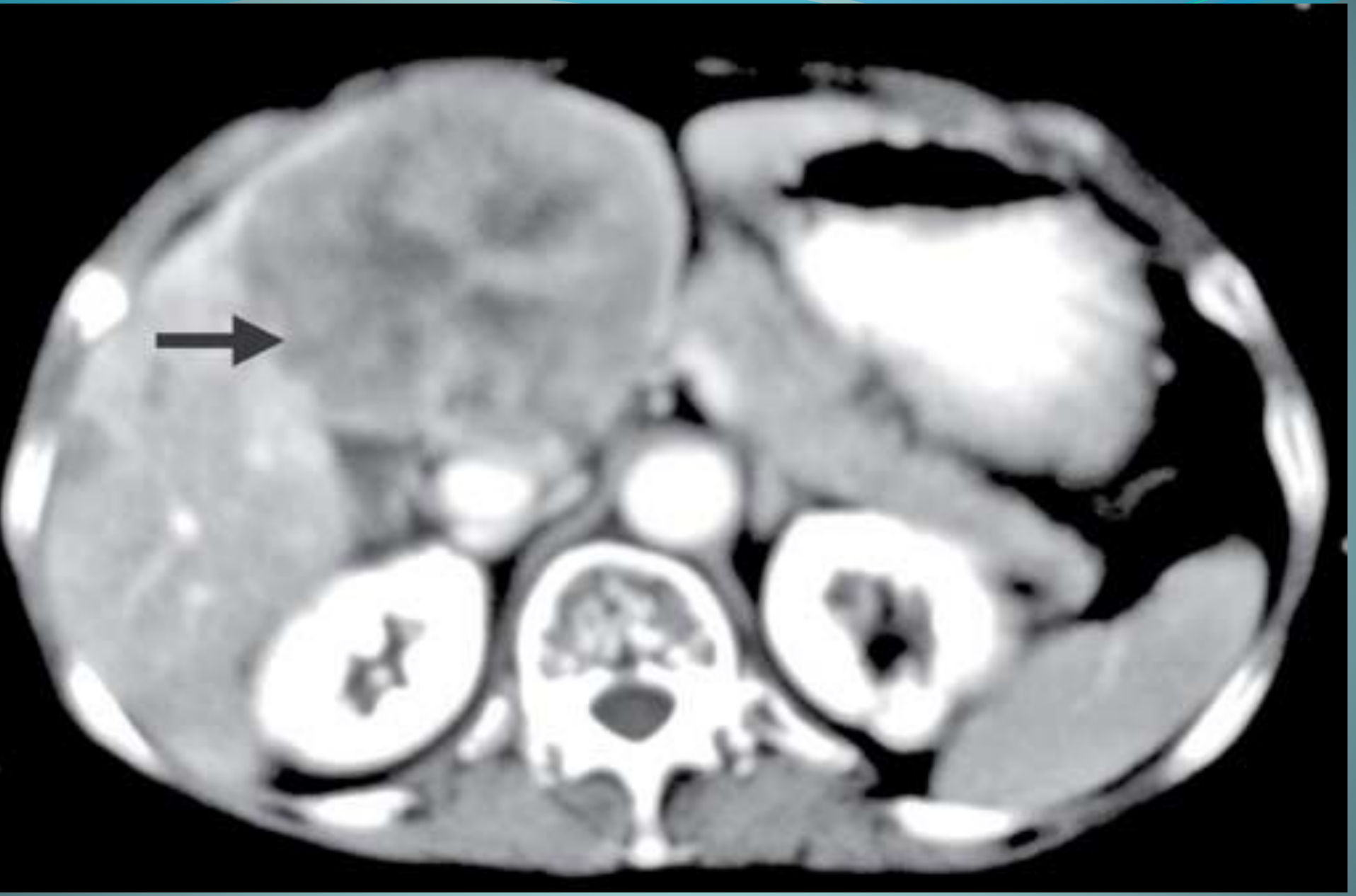
Secondaries in liver

Polycystic disease of liver

Amoebic liver abscess

Hydatid cyst of the liver





Investigations

1. U/S abdomen—very useful method. It shows hyperechoic mass; mosaic pattern with thin halo and lateral shadows. Extent, tumour thrombi extension can be made out.
2. CT scan abdomen (CECT) more reliable and ideal (hypodense, mosaic, vascular lesion with irregular margin). reveals the size, location and extent, vascularity, portal vein invasion, nodal status, portal vein thrombosis. helps to assess operability.
3. *Tumour markers—α feto protein (AFP). AFP will be raised* more than 100 IU; as high as 1000 IU is possible in HCC.

4. Celiac angiography/CT angiography—
5. Liver function tests like serum bilirubin, albumin, enzymes (alkaline phosphatase, transaminase, 5' nucleotidase) including prothrombin time.
6. Liver biopsy is done after controlling prothrombin time
7. Contrast MRI/CT scan
8. MRI—T2 weighted studies are useful for small HCC.
MR angiography is also done to see tumour thrombus in portal vein, hepatic vein and IVC.
9. Ascitic tap when ascites is present for cytology.
10. Laparoscopic evaluation and laparoscopic US is useful for proper assessment of the tumour.
11. Investigations in relation to hepatitis B and hepatitis C virus infections.
12. Metastatic work up