A PROSPECTIVE STUDY ON INCIDENCE, ETIOPATHOGENESIS, CLINICAL PRESENTATION, MANAGEMENT AND PROGNOSIS OF LIVER ABSCESS

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GOVERNMENT MOHAN KUMARAMANGALAM

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CERTIFICATE

This is to certify that the dissertation entitled **"A PROSPECTIVE STUDY ON INCIDENCE, ETIOPATHOGENESIS, CLINICAL PRESENTATION, MANAGEMENT AND PROGNOSIS OF LIVER ABSCESS IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE & HOSPITAL, SALEM"** is a bonafide work done by **Dr. R. BANUREKHA** post graduate in **M.S. BRANCH - I GENERAL SURGERY** at Government Mohan Kumaramangalam Medical College and Hospital, Salem, to be submitted to The Tamil Nadu Dr. M.G.R Medical University, in fulfillment of the University Rules and Regulation for the award of M.S. Degree Branch I General Surgery, under my supervision and guidance, during the academic period from June 2010 to December 2012.

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DECLARATION

I solemnly declare that this dissertation **"A PROSPECTIVE STUDY ON INCIDENCE, ETIOPATHOGENESIS, CLINICAL PRESENTATION, MANAGEMENT AND PROGNOSIS OF LIVER ABSCESS IN GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE & HOSPITAL, SALEM"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem under the guidance and supervision of **Prof. Dr. R. KATTABOMMAN, M.S.** Professor and H.O.D. of General Surgery, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfillment of the University regulations for the award of the degree of M.S. Branch I General Surgery.

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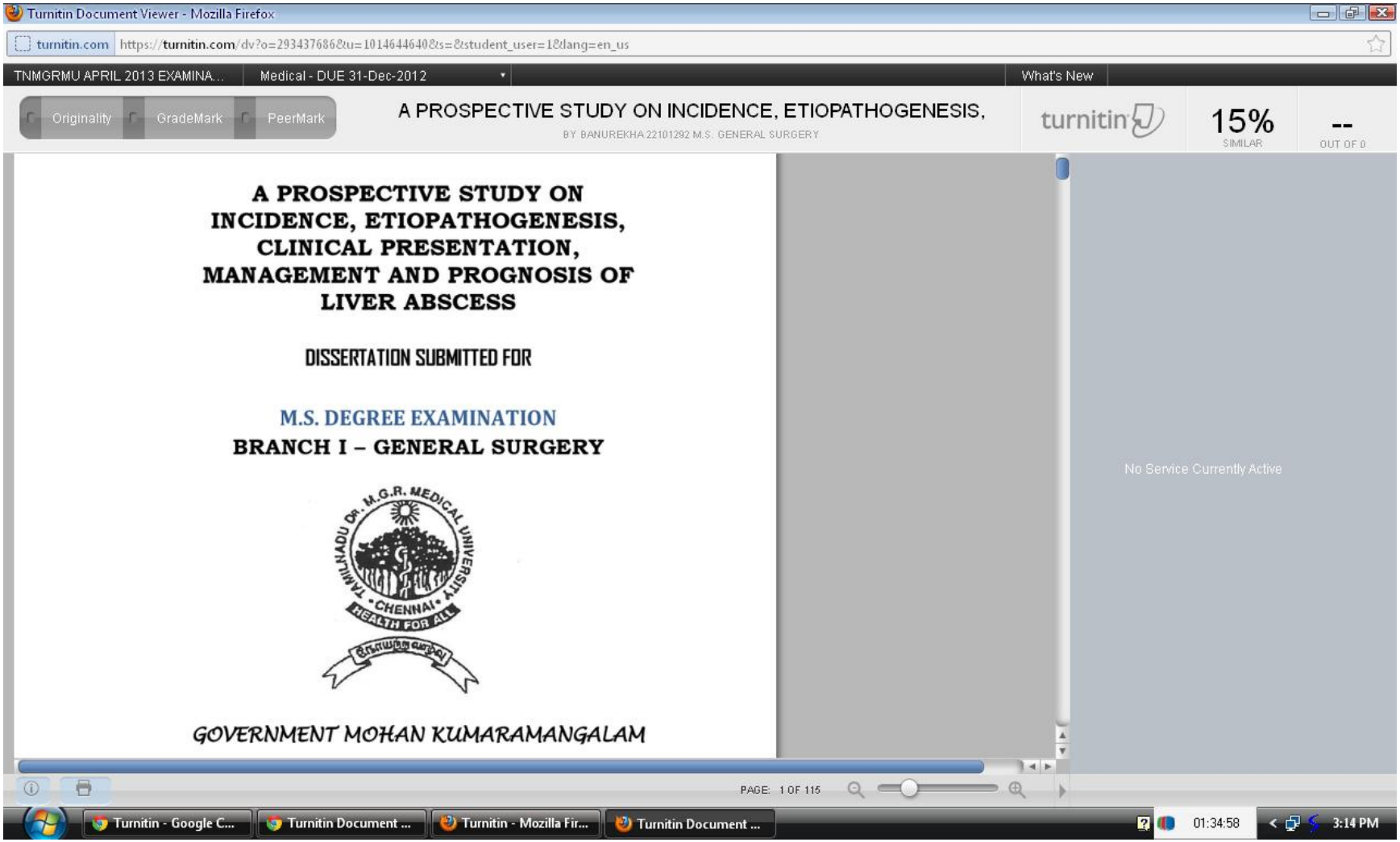

CONTENTS

INTRODUCTION

Liver abscess continues to be a major diagnostic and therapeutic challenge to the medical fraternity. It is a life threatening and a potentially serious condition if left untreated. Therefore, it is very important for prompt diagnosis and appropriate management at the earliest.[1]

The two common types of liver abscess encountered are amoebic liver abscess and pyogenic liver abscess.

Amoebiasis is a common infestation in developing countries due to poor sanitary facilities. It affects about 10 % of the population all over the world. Amoebic liver abscess is the commonest extra intestinal manifestation of amoebiasis. It affects about 3-9 % of victims. India is an endemic zone for amoebic liver abscess. It may present as acute abdomen

requiring emergency laparotomy. Spontaneous intraperitoneal rupture, extra and retroperitoneal rupture and intrathoracic rupture are frequently seen in liver abscess. Delay in diagnosis may lead to rupture of liver abscess which may increase the morbidity as well as mortality.[2]

Pyogenic liver abscess is not an uncommon entity. It is a relatively rare complication of intra-abdominal infection or biliary tract infection. It is usually polymicrobial in nature due to the ascending route of infection from the gastrointestinal tract.[3]

The overlapping of symptoms between amoebic and pyogenic liver abscess makes early clinical differentiation difficult.

This study was conducted to assess the incidence, etiology, clinical presentation, management and prognosis of liver abscess in patients attending a tertiary referral hospital.

AIM OF THE STUDY

The aim of this prospective study is

1. To study the incidence and etiology of liver abscess.
2. To evaluate the clinical features of liver abscess.
3. To evaluate the management and prognosis in liver abscess.

REVIEW OF LITERATURE

Anatomy of the Liver

The liver is a large solid organ situated in the right hypochondrium of the abdominal cavity.

Development

The liver originates from the dorsal end of the foregut as a solid bud of endodermal cells. The hepatic bud which grows anteriorly into septum transversum divides into right and left branch and gets canalized to form the common hepatic duct and then into right and left hepatic ducts.

The paired vittelline vein and umbilical vein that passes through the septum transversum gets broken up by the invading column of hepatocytes and form the liver sinusoids.

Location

It occupies the whole right hypochondrium, greater part of epigastrium and extends partly into the left hypochondrium.[4]

Anatomical lobes of the liver

The liver is divided into two lobes namely the right and left lobe by the attachment of the falciform ligament anteriorly and superiorly, by the fissure for the ligamentum venosum posteriorly and fissure for the ligamentum teres inferiorly.

Surgical lobes of the liver

The liver is divided into two lobes namely the right and left lobes by hepatic and portal vein branches. A plane without any surface marking which runs from the gall bladder to the left side of the inferior venacava known as cantlie's line divides the liver into right and left lobe.

The right lobe is further divided into anterior and posterior segments. The left lobe is further divided into medial segment which is called as quadrate lobe and a lateral segment.

The functional anatomy of the liver is made up of eight segments, each of which in turn is supplied by a portal triad composed of hepatic artery, portal vein and bile duct.

These segments are organized further into four sectors which are separated by scissurae which contain the three main hepatic veins.

This system was first described in 1957 by Woodsmith and Goldburne as well as Couinaud.

The main scissura which contains the middle hepatic vein runs in the gallbladder fossa to the

left side of inferior venacava which divides the liver into right and left hemi liver. This line of the main scissura is also called as the cantlie's line.

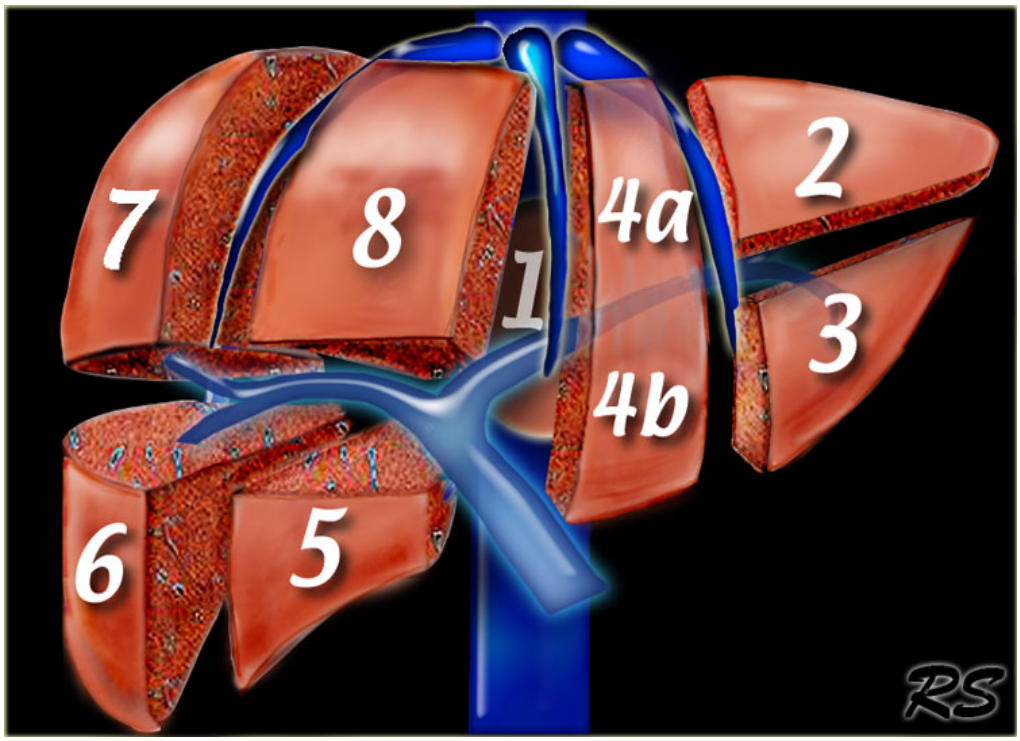

FIG. 1. COUINAUD'S SEGMENTAL ANATOMY OF LIVER

Right liver is divided into an anterior (segment 5 & 8) and posterior (segment 6 & 7) sector by the right scissura containing the right hepatic vein.

Left liver has a visible fissure along its inferior aspect which is called as the umbilical fissure. The ligamentum teres runs into this fissure. The

umbilical fissure has the left portal pedicle which contains the left portal vein, hepatic artery and bile duct.

The left scissura runs posterior to the ligamentum teres and has the left hepatic vein. The left liver is split into median segments 3, 4 and lateral segment 2 by the left scissura.[5]

Caudate lobe

It is situated on the posterior aspect. It is bounded at right by the groove for the inferior venacava and at the left by the fissure for the ligamentum venosum and inferiorly by porta hepatis.[4]

The vascular supply, biliary drainage from the caudate lobe comes from both the right and left systems. Hepatic venous drainage of the caudate lobe is through multiple small veins which drain posteriorly directly into the inferior venacava.[5]

Quadrate lobe

It is situated on the inferior aspect. It is bounded anteriorly by the inferior borderof liver, posteriorly by the porta hepatis at right by the fossa for the gallbladder and at left by fissure for ligamentum teres.

Porta hepatis

It is a deep transverse fissure located on the inferior surface of the right lobe of liver. It is related with the caudate lobe above and quadrate lobe below.

The hepatic artery, portal vein and the hepatic plexus of nerves enter through porta hepatis. The right and left hepatic duct as well as few lymphatics leave it.[5]

Hepatic artery

It accounts for about 25% of the hepatic blood flow and about 30 to 50% of oxygenation of liver. The common hepatic artery arises from the coeliac trunk. After giving the branch of gastro duodenal artery it is called as proper hepatic artery. It in turn gets divided into right and left hepatic arteries at the hilum which supply the respective lobes.

Hepatic vein

The hepatic veins are formed by the union of central vein of the lobules. Three major hepatic veins drain it from the superior and posterior aspects of the liver directly into the inferior venacava.

Portal vein

The portal vein accounts for about 75% of the hepatic blood flow. It is formed behind the neck of the pancreas by the union of the superior mesenteric

vein and the splenic vein at the level of the second lumbar vertebra.

The length of the main portal vein is about 5.5 to 8 cm. Its diameter is about 1 cm. The portal vein divides into the right and left branches at the hilum of the liver.

Microscopic Anatomy

The liver lobule is the functional unit of the liver. It is composed of a central terminal hepatic venule surrounded by 4- 6 terminal portal triads which form a polygonal unit. This unit is lined in its periphery by the terminal portal triad branches.

In between these terminal portal triads and the central hepatic venule, the hepatocytes are arranged in plates and are surrounded on each side by endothelial lined blood filled sinusoids. The blood flows from the terminal portal triad through these sinusoids into the hepatic venule.

Bile is formed in the hepatocytes which empty into the terminal canaliculi that form on the lateral wall of the intracellular hepatocytes which finally coalesce into the bile duct, following towards the portal triad.[5]

Surface Anatomy

The upper border of the liver is indicated by a line connecting the following points.

1. First point is in the left 5th intercostal space 9 cms from the median plane.
2. Second point is at the xiphisternal joint.
3. Third point is at the upper border of the right 5th costal cartilage in the right lateral vertical plane.
4. Fourth point is at the sixth rib in the mid axillary line.
5. Fifth point is at the inferior angle of right scapula.
6. Sixth point is at the 8th thoracic spine.

The lower border of liver is marked by a curved line joining the following points.

1. First point is at the left 5th intercostal space 9 cms from the median plane.
2. Second point is at the tip of the 8th costal cartilage on the left costal margin.
3. Third point is at the transpyloric plane in the midline.
4. Fourth point is at the tip of the ninth costal cartilage on the right costal margin.
5. Fifth point is about 1cm below the right costal margin at the tip of the 10th costal cartilage.
6. Sixth point is at the 11th thoracic spine.

The right border of liver is marked on the front by a curved line which is convex laterally, drawn from a point little below the right nipple to a point one centimeter below the right costal margin at the tip of the 10th costal cartilage.[4]

Amoebic Liver Abscess

History

Entamoeba histolytica was first identified by Losch of Russia in 1875 and was recovered from the wall of a hepatic abscess by Kartulis of Egypt in 1887. It was named later by Prof. Shauddin in 1903.[6] In 1891 Councilman and Loefleur, stated that so called "tropical abscess" of the liver secondary to amoebic dysentry.[7]

It is the commonest extra intestinal manifestation of amebiasis. It is often called tropical abscess. Entameba histolytica is the second leading cause of parasite related death in the world next to malaria. Amoebic liver abscess was found to be associated with blood and mucus diarrheal stool in the 5th century BC by hippocrates.

Incidence

Worldwide, about 500 million people are found to be carriers of Entamoeba histolytica. 50 million people are found to have active disease of which about 50,000 to 1,00,000 die annually. Death from amoebiasis is mostly due to extra intestinal amoebiasis of which amoebic liver abscess is the most common. Amoebiasis is endemic in India. The incidence of amoebiasis is increasing in areas with higher poverty, poor sanitation facilities, public health and hygiene.

Amoebic liver abscess is found to have a male: female ratio of 10:1. The average age group is about 20 to 40 years. The cause for the greater male preponderance is unclear. Alcohol consumption, hormonal effect in pre menopausal women, and positive protective effect of iron deficiency anemia in menstruating women are few causes. Alcohol consumption is an immunosupressive factor which

indirectly impairs the kupffer cell function in the liver thereby depressing the cellular and humoral immunological response to E.histolytica.[8]

Etiology

Two species of ameobae commonly infect human beings. Entamoeba histolytica is responsible for invasive disease. Entamoeba dispar is associated with asymptomatic carrier state.

Risk factors

- Alcoholism.
- Recent travel to endemic area.
- Disorders of cell mediated immunity.
- Malnutrition
- Corticosteroids use.
- HIV infection
- Homosexual activity.
- Malignancy.

Mode of Spread

The infestation is transmitted through contaminated water and vegetables. It begins with ingestion of the quadrinucleate cyst of Entamoeba histolytica. The liver abscess most commonly occurs in the postero-superior right lobe of the liver. Preferential laminar blood flow to the right side has been postulated as a cause and amebiasis of the colon has its highest incidence in the cecum and ascending colon. It is most common in six to eight segments of liver.

Pathogenesis of Amoebic liver abscess

The pathogenesis of amoebic liver abscess was first described by Sir. Leonard Rojer. The cyst of E.histolytica is resistant to the acidic pH of the stomach and excystation occurs in the small intestine which releases the trophozoites. It then passes to the large intestine using lectin carbohydrate interaction

adhering to the colonic mucosa. The infectious cyst can reform in the colon and can be excreted.

Amoebae migrate from the bowel in to the portal vein which embolise in the small interlobular vein of the liver, causing thrombosis and subsequent congestion of liver causing presuppurative amoebic hepatitis. If sufficient amoebae reach the liver, it causes thrombosis in several contiguous vessels and infarction.

Invasion of amoebae into the portal system causes an acute cellular infiltrate containing polymorphs. Subsequently polymorphs are lysed by contact ameobae which release neutrophil toxins resulting in the necrosis of hepatocytes.

The amoebic liver abscess is a clearly circumscribed area. The liver parenchyma is replaced by necrotic tissue. The abscess contains an acellular

fluid which is called as anchovy sauce pus. It is chocolate brown in colour and contains necrotic liver tissue, bile and fat products. Trophozoites are absent in the abscess fluid.[7, 8]

PYOGENIC LIVER ABSCESS

History

Hepatic abscess was described by Hippocrates as early by 400 BC. In 1938 Ochsner described this disease to have occurred in young males with an underlying intra abdominal infection. In recent years it has been found to be a disease of elderly, associated with intra abdominal infection, bacteremia and malignancy.[9]

Definition

It is defined as a focal suppurative lesion within the liver tissue. It results from microbial infection of liver parenchyma with infiltration by inflammatory cells and formation of pus collection.[1]

It is defined as one or more discrete lesion in the liver and associated with positive bacterial culture of material obtained from blood or percutaneous aspirate of the lesion in the presence of an intrahepatic cavity observed in imaging studies.

Pyogenic liver abscess secondary to appendicitis now rarely occurs in younger people. It is more common in elderly and debilitated patients. The most common predisposing condition of pyogenic liver abscess is biliary tract obstruction.[10]

Etiology

It is mostly polymicrobial in nature.

The common organisms are:

Gram negative aerobes

Eschericia coli

Klebsiella

Proteus

Enterobacter

Serratia,

Morganella

Acinetobacter

Gram positive aerobes

Streptococcal species

Enterococci fecalis

Beta hemolytic streptococci

Staphylococci

Anaerobes

Bacteroides species

Fusobacterium

Peptostreptococci

Clostridium

Pathogenesis of pyogenic liver abscess

The possible sources of infection are from

1. Bile ducts causing ascending cholangitis.
2. Portal vein causing pyelophlebitis from diverticulitis or appendicitis.

3. Hepatic artery due to septicemia
4. Trauma due to blunt or penetrating injury
5. Direct extension from a contiguous disease.
6. Cryptogenic

About 35% - 45 % of the pyogenic liver abscess is caused by biliary tract obstruction. About 40% of pyogenic liver abscess is due to an underlying liver malignancy. Diverticulitis, pelvic abscess, sub phrenic abscess, perforated colon cancer being the less common causes of pyogenic liver abscess.

Appendicitis accounts only for about 2% of pyogenic liver abscess. Trauma to the liver leads to parenchymal necrosis and proliferation of micro organisms resulting in subsequent abscess formation.

About 12% of the pyogenic liver abscess is a result of arterial embolisation of the bacteria through the hepatic artery. About 10 – 40% of the pyogenic

liver abscess is of unknown etiology (cryptogenic). Pyogenic abscess is often associated with co-morbidities like diabetes mellitus, immunosupression and malignancy.

Pyogenic liver abscess manifests commonly with bilateral involvement and multiple abscesses are more common secondary to biliary etiology.

CLINICAL PRESENTATION OF LIVER ABSCESS

Symptoms

- Pain in right hypochondrium
- Fever
- Sweating and rigors
- Nausea and vomiting
- Diarrhoea
- Cough
- Anorexia, malaise and weight loss

Signs

- Right hypochondrial tenderness
- Intercostal tenderness
- Hepatomegaly
- Jaundice
- Pleural rub/Pleurisy
- Pleural effusion
- Ascites[7, 8]

DIAGNOSIS OF LIVER ABSCESS

The diagnosis of amoebic liver abscess is based on the following criteria:

1. Clinical symptoms such as fever and right hypochondrial pain.
2. Enlarged and/or tender liver, with or without jaundice, right lower intercostal tenderness.
3. Raised right dome of diaphragm on chest x-ray

4. Space occupying lesion in the liver identified by ultrasound / CT scan or other imaging modality suggestive of a hepatic abscess.
5. Liver aspirate like anchovy sauce and bacteriologically sterile.
6. Positive Indirect Haemagglutination of serum antibodies showing a titre of > 1:128 against Entamoeba histolytica.
7. Improvement after treatment with anti amoebic drugs.

Other investigations like determination of white blood cell count ranging from 10,000–20,000 cells per cubic millimeter.

Serum bilirubin showing rise in conjugated fraction with elevated alkaline phosphatase level.[11]

X-Ray Chest PA View

May show elevation of the right hemidiaphragm with or without pleural effusion.

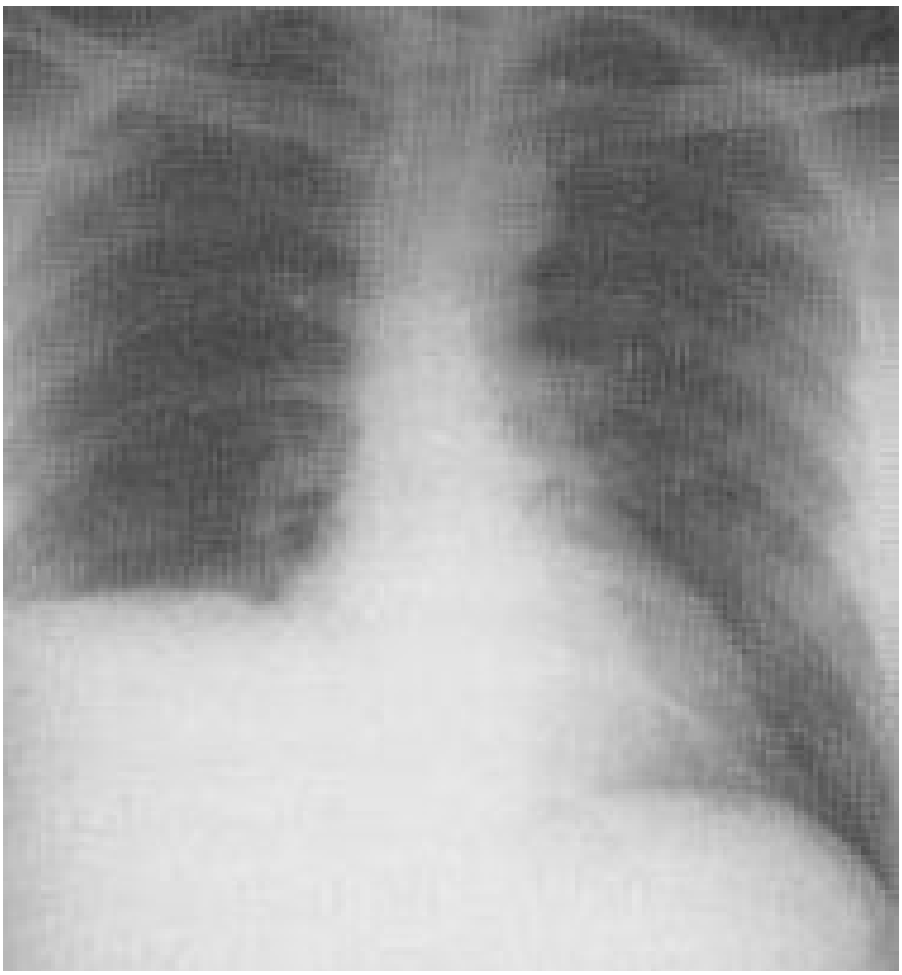

FIG. 2. X-RAY CHEST SHOWING ELEVATED RIGHT HEMIDIAPHRAGM

Stool Examination

To determine the presence of parasite.

Ultrasound

It is the initial choice of diagnostic study to confirm the presence of abscess, its site, its size, whether single or multiple and the need for aspiration.

Abscesses in ultrasound show well defined margins with lack of prominent peripheral echoes with hypoechoic lesion.

CT scan:

The advantage of CT scan is its ability to identify even a small lesion. Liver abscess in CT scan appear isodense with normal liver parenchyma on unenhanced scan and will show low attenuation in contrast enhanced equilibrium phase images. They may be either unilocular or complex and multi locular

with or without air fluid level. Abscesses usually have an enhancing rim or peripheral capsule.

MRI

Abscesses in MRI usually exhibit a low signal on T1 weighted image and hyperintense image on T2. Most of the abscesses have peripheral enhancement after intravenous administration of Gadolinium. Biliary fistulas can be demonstrated in MRI by fluoroscopic contrast injection study.

Nuclear Medicine Studies

Nuclear medicine studies like Gallium scanning and Technitium 99 liver scan are useful in differentiating pyogenic and amoebic liver abscess. Amoebic liver abscess does not contain leucocytes and hence do not light up on those scans.[12, 13]

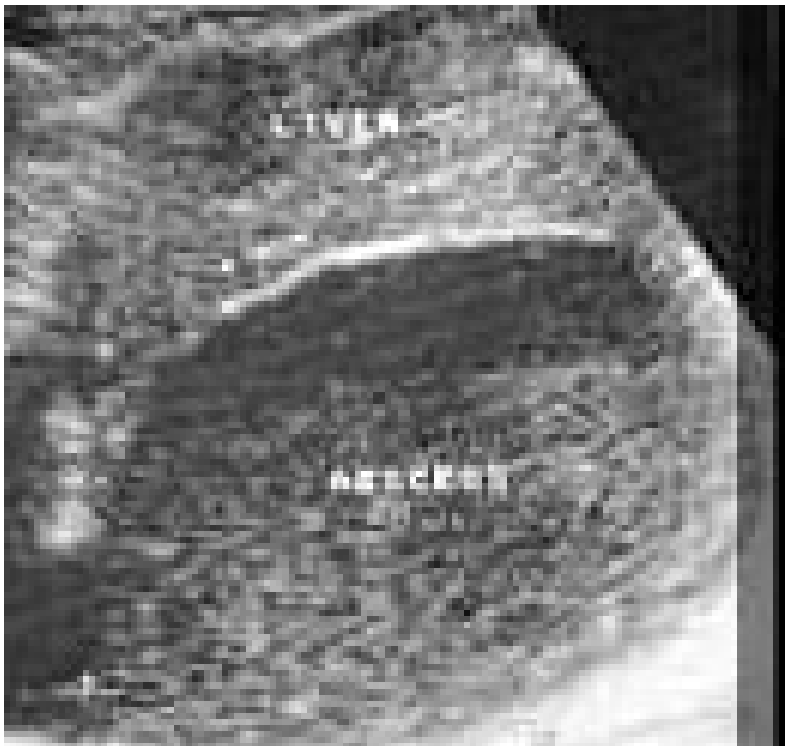

FIG. 3, ULTRASOUND SHOWING LIVER ABSCESS

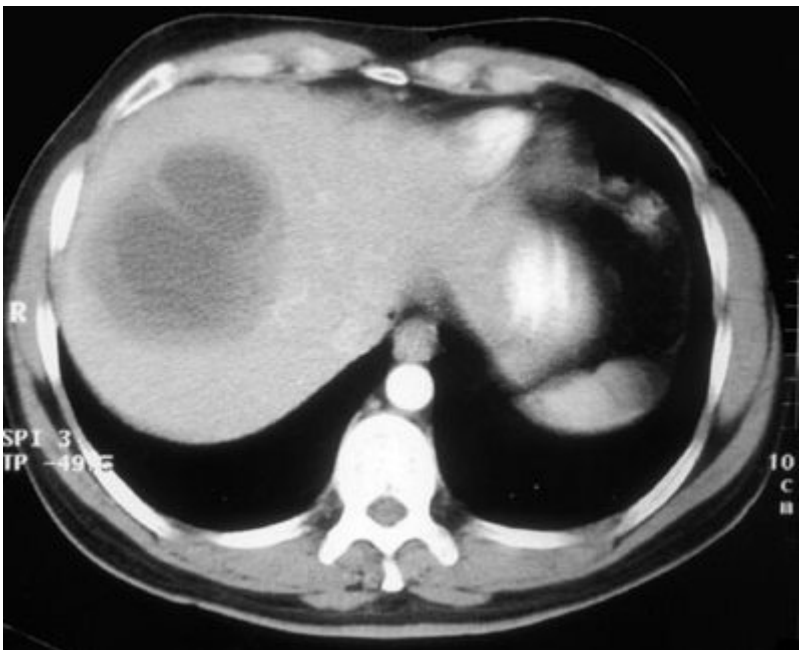

FIG. 4. CT SCAN SHOWING LIVER ABSCESS

Serology

Serological tests to confirm the diagnosis of amoebiasis include

Immunoflourescence

Indirect Hemagglutination test

Enzyme Linked Immuno Sorbent Assay

Anti amoebic serum antibodies are seen in more than 90% of patients. During first week of onset serology may be negative. Titres usually reach a peak by the second or the third month and then decrease later.

The techlab E.histolytica II test differentiates E.histolytica from E.dispar. It identifies circulating E. histolytica Gal / Gal Nac Lectin antigen which is positive in almost all patients with amoebic liver abscess and who have not received metronidazole.

Most of patients with amoebic liver abscess do not have co-existent intestinal infection with Entamoeba histolytica. Hence absence of E. histolytica antigen or its DNA in the stool samples is not useful for the diagnosis of amoebic liver abscess.

The present modality for diagnosing amoebic liver abscess is the identification of the anti amoebic antibody by serological test along with aspiration of the abscess. The presence of serum antibody against E. histolytica and absence of bacteria in the abscess aspirate are suggestive of amebic liver abscess.

The lacunae in the serological tests are that serum antibody may remain positive even for several years after infection, especially in people living in endemic areas of amebiasis.[14, 15]

The trophozoites of Entamoeba histolytica can be detected only in about 15% of the liver pus

aspiration.

E.histolytica DNA can be detected in saliva, urine, amoebic liver abscess pus by real time PCR. Therefore saliva and urine are more ideal specimens than blood for detection of E. histolytica DNA in patients with amoebic liver abscess.[15]

Molecular studies

Molecular studies using Polymerase chain reaction amplifies the Entamoeba histolytica genes from extracted fecal DNA. The advantage of molecular studies is it is extremely sensitive. It may detect even a single parasite and may also differentiate pathogenic and non pathogenic species.[16]

COMPLICATIONS OF LIVER ABSCESS

The common complications of amoebic liver abscess are

Rupture of the abscess into the peritoneal cavity, pleural cavity or in the pericardial cavity.

Abscesses situated near the diaphragmatic surface of the liver may produce inflammatory reaction of the diaphragm, pleura and the pericardium.

The abscess may extend into the chest and may produce atelectasis of right lung and pleural effusion. It may erode into the bronchus and may present with productive cough and expectoration of the abscess material.

The abscess if it ruptures through the diaphragm may result in formation of either amoebic empyema or pulmonary abscess. This usually presents with persistent cough, pleuritic pain and breathlessness.

Rupture of the abscess into the peritoneum produces abdominal distension, abdominal pain, peritonitis and paralytic ileus.

The abscess rupturing into the pericardium is a rare complication and is more common with left side or centrally located abscesses.[8, 17]

Other complications

Hemobilia

Secondary bacterial infection

Metastatic brain abscess.

Differential diagnosis of Amoebic liver abscess

Pyogenic liver abscess

Echinococcal cyst

Malignant hepatic tumor[18]

Echinococcal cyst

It is mostly asymptomatic. Imaging studies may show calcification of daughter cysts. Eosinophilia is present in about 40% of patients.

Malignant hepatic tumor

It is commonly seen in cirrhotic patients and may rarely present as cystic lesion. Serum alfa foeto protein levels may be rised. In three phase CT scan hematoma in tumor may enhance during arterial phase of contrast admistration.

DISTINCTION BETWEEN AMOEBIC AND PYOGENIC LIVER ABSCESS[5]

CRITERIA	***AMOEBIC***	***PYOGENIC***
Age	<50 years	>50 years
M:F ratio	10:1	1:1
Recent travel to endemic area	present	-
Alcohol use	More common	common
Associated symptoms	Diarrhea Pulmonary symptoms	Jaundice High fever
Increased bilirubin	uncommon	common
Increased ALP	More common	common
Positive blood culture	No	Yes
Amoebic serology	Positive	Negative

Others

The other differential diagnoses for liver abscess are acute cholecystitis, acute hepatitis, perforated peptic ulcer, pseudocyst of pancreas, pneumonia, pleural effusion, empyema, chronic pulmonary disease, tuberculosis, pyrexia of unknown origin and malignancy.[18]

TREATMENT OF AMOEBIC LIVER ABSCESS

Four groups of treatment modalities are found to be effective:

1. Drug therapy alone
2. Ultrasound guided aspiration and drug therapy
3. Percutaneous catheter drainage and drug therapy
4. Laparatomy drainage and drug therapy.

Chemotherapy

The indications for chemotherapy are

1. Non-complicated abscess
2. No features of rupture or impending rupture
3. No compression effect

Amoebicidal agents

Metronidazole is the drug of choice in the treatment of uncomplicated liver abscess. The recommended oral dose is 1 gm twice a day for 10 – 15 days in adults and 30 -50 mg per kg per day for 10 days three times a day in children. The intravenous dose is 500 mg 6^{th} hourly for adults and 7.5 mg/ kg 6^{th} hourly in children for 10 days.

Other nitroimidazoles like tinidazole, secnidazole and ornidazole are useful as tissue amoebicides. The common side effect of these nitroimidazoles is metallic taste.

Other alternative drugs for eradicating liver trophozoites aree chloroquine at a dose of 600 mg per day for 2 days and then 200 mg per day for 2 – 3 weeks. The side effects include diarrhea, abdominal cramps and hypotension.

Dihydroemetine and emetine are not routinely used due to their significant side effects.

Luminal agents

The luminal agents are used for associated intestinal infestation even if stool is negative for the trophozoite. These include

Paromomycin in a dose of 30 mg/kg/ day orally thrice a day in divided doses for ten days.

Di-iodo-hydroxyquin in a dose of 650 mg orally thrice a day for 20 days.

Diloxanide furoate in a dose of 500 mg orally thrice a day for 10 days.[19]

Modern management of amoebic liver abscess is intravenous antibiotics along with imaging guided percutaneous aspiration or percutaneous catheter drainage.

Percutaneous needle aspiration

The evacuation of pus from the abscess is done with an 18 G disposable trochar needle. Ultrasound of abdomen is done every three days and size of the abscess cavity noted. If there is no significant reduction in the size of abscess cavity on subsequent examination, aspiration will be repeated. It's advantage is, it is relatively safe and effective as percutaneous catheter drainage. It is easy to perform, less complicated, less risky and the incidence of post procedure septicemia is low.[20]

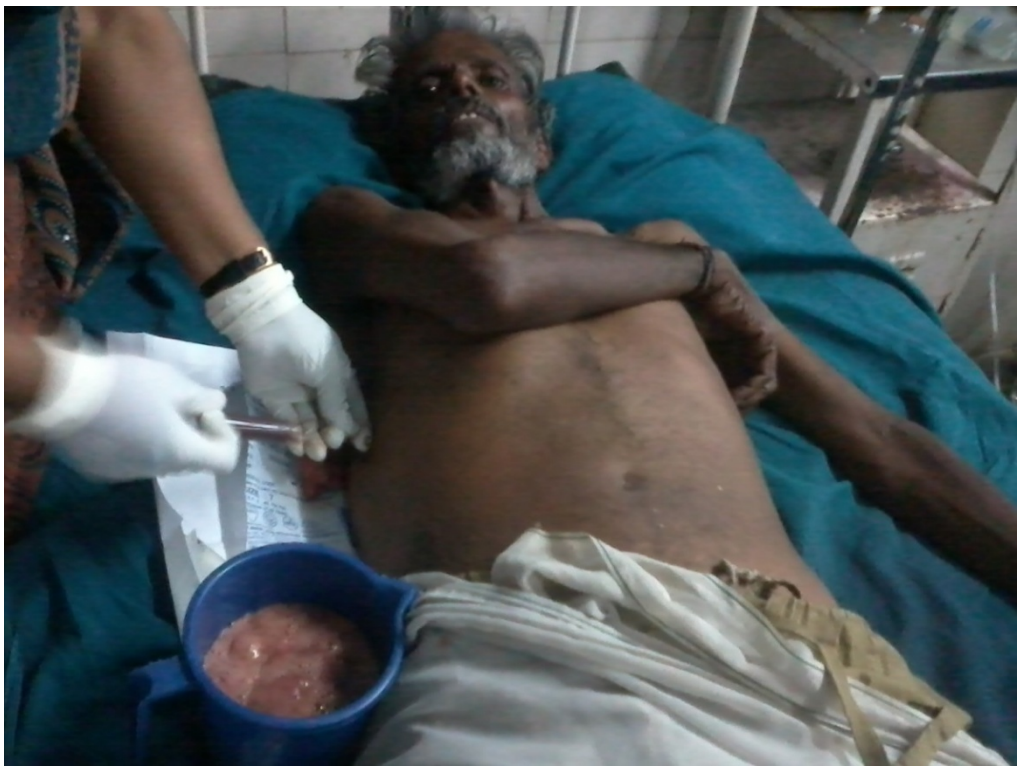

FIG. 5. PERCUTANEOUS NEEDLE ASPIRATION OF AMOEBIC LIVER ABSCESS SHOWING ANCHOVY-SAUCE PUS

Percutaneous catheter drainage

Percutaneous catheter drainage or surgery is required when amoebic liver abscess fails to respond to routine medical treatment. The indications for percutaneous catheter drainage are

1. Abscess volume exceeding 250 ml.
2. Thin rim of tissue less than one cm thick around the abscess.
3. Features of systemic toxicity
4. Failure to respond to medical treatment.
5. Patients with abscess drainage output more than 25 ml per day for 2 weeks.
6. Presence of bile in the abscess drainage fluid.
7. Patients who underwent endoscopic biliary drainage.

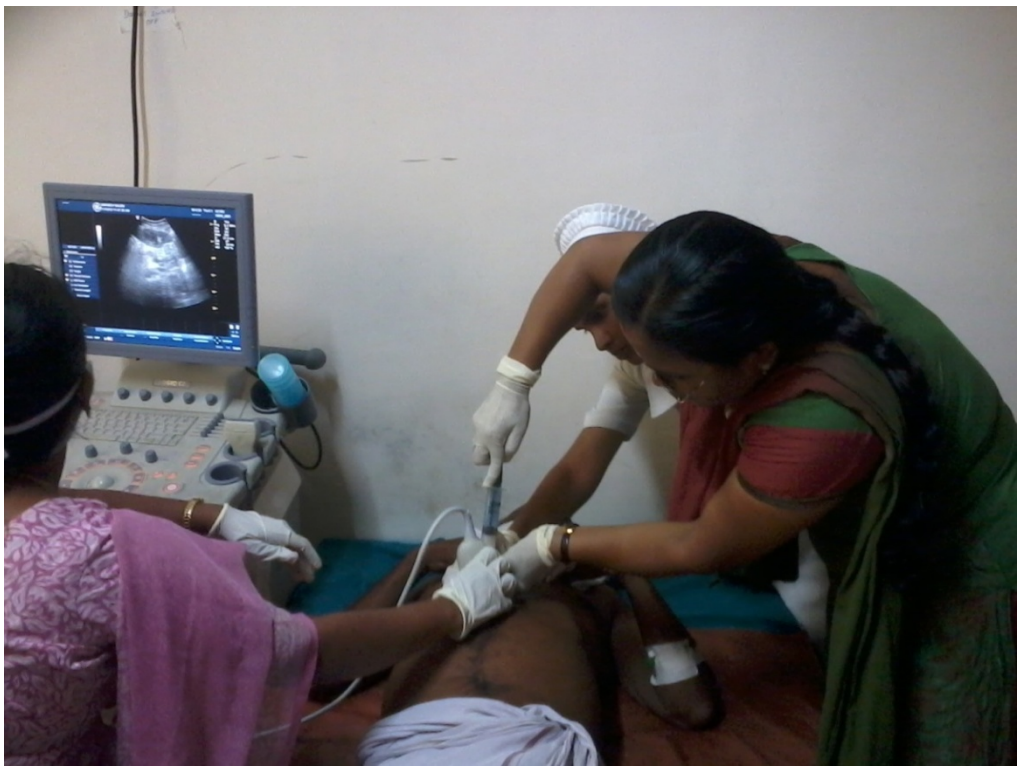

FIG. 6. ULTRASOUND GUIDED PERCUTANEOUS NEEDLE ASPIRATION

Procedure

The drainage technique consists of a trochar method using an 8 Fr multiple side hole pig tail catheter which is introduced into the abscess cavity. The procedure is done under local anesthesia with the patient lying supine. Careful localization of the abscess and accurate selection of the entry site are essential. Aspiration is performed using a catheter until no more pus is draining. The catheter is then fixed to the skin for continuous external drainage and is left in place until stoppage of contents.[19, 20]

Laparascopic drainage

Presently, laparoscopy is used for drainage of liver abscess and also for management of its intra peritoneal complications.

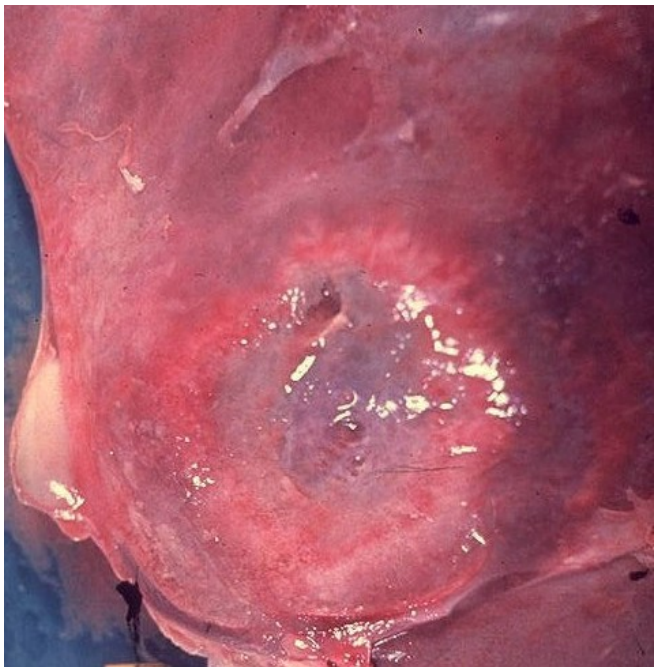

FIG. 7. LAPAROTOMY SHOWING LIVER ABSCESS

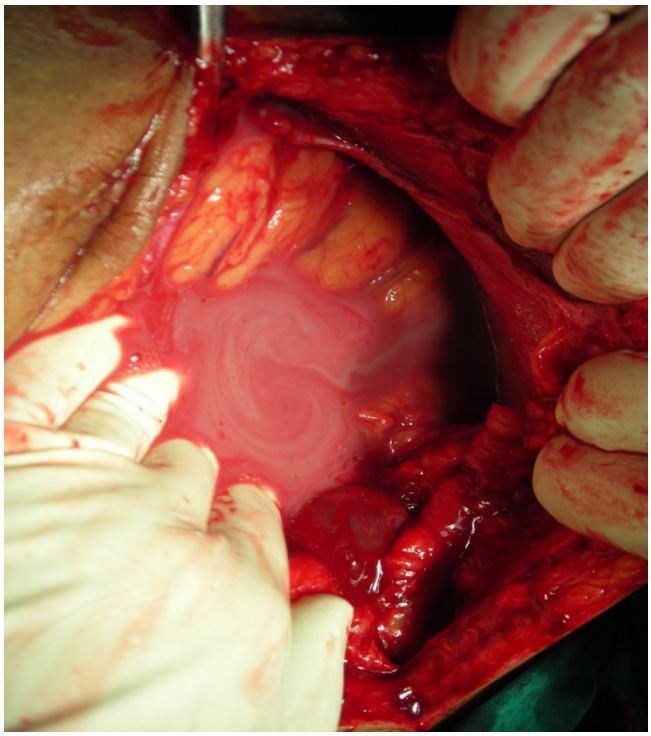

FIG. 8. LAPAROTOMY SHOWING RUPTURE LIVER ABSCESS

Laparotomy and surgical drainage

The indications for laparotomy and surgical drainage are

1. Patients presenting with complications of amoebic liver abscess.
2. Patients presenting with large left sided abscess.
3. Patients not amenable to catheter based drainage.
4. Patients with risk of rupture of abscess into the pericardium.[19]

Recent advances in amoebic liver abscess

Presently research is underway on development of a vaccine to prevent amoebiasis.

The Entamoeba histolytica Gal / Gal Nac Lectin antigen is an ideal vaccine candidate for many reasons.

The antigenically conserved surface molecule is a distinct isolate of Entamoeba histolytica. It is the major antigen recognized by the humoral system which is vital in adherence of the trophozoite to host cells which stimulates the production of amoebicidal immune peripheral lymphocytes and in the production of protective cytokines.[8, 21]

Prognostic markers in amoebic liver abscess

Based on clinical, biochemical and sonographic findings, the prognostic markers for mortality in amoebic liver abscess are

1. Serum bilirubin level > 3.5 mg/dL.
2. Serum albumin level < 2g/dL.
3. Quantity of abscess collection > 500 ml
4. Multiple abscesses.
5. Features of encephalopathy.[19]

FOLLOW UP AND OUTCOME

Follow up ultrasonogram of abdomen is done 24 hours after intervention and is repeated every three days and the size of the abscess cavity is recorded. The criteria for successful treatment are clinical subsidence of infection and sonographic evidence of resolution of abscess such as disappearance and marked decrease in the size of abscess cavity. The mean time for disappearance of ultrasonographic abnormalities in resolution of liver abscess is about 6-9 months. Serial imaging studies are strictly not necessary for follow up if the patient responds satisfactorily to therapy.[5]

Prevention

Amoebiasis being a social and public health problem, its definitive eradication depends on the improvement of public health measures with adequate sanitation, protected drinking water supply and better health education.[22]

TREATMENT OF PYOGENIC LIVER ABSCESS

The treatment of pyogenic liver abscess consists of an empirical antimicrobial regimen. The most commonly used regimen is a combination of third generation cephalosporin, an aminoglcoside which may be changed later based on the culture and sensitivity of the aspirate from the abscess. Quinolones may be used as an alternative for cephalosporin.[23]

KEY POINTS OF AMEOBIC LIVER ABSCESS

- It is the commonest extra intestinal manifestion of amebiasis.
- Diagnosis is confirmed by imaging and serology.
- Curative treatment is Metronidazole.
- Treatment should always consist of a luminal agent to eradicate colonization and reduce the risk of invasive disease.

- Prevention is adequate hygiene, sanitation and protected drinking water supply.[12]

KEY POINTS OF PYOGENIC LIVER ABSCESS

- Pyogenic liver abscess is most commonly the result of ascending biliary infection. It also spreads by intra abdominal infection, hematogenous spread and spread through the portal vein.
- The microbiology of abscess varies and more frequently it is polymicrobial.
- Clinical presentation may be non-specific.
- Definitive treatment is drainage and antibiotics.[24]

Khan R A A, et al in a case series of 188 amoebic liver abscesses in a tertiary hospital in Pakistan over a period of two years from 2007 to 2008 observed a male: female ratio of 2:1 and more than 75% patients had solitary abscess. About 83 % had right lobe abscess and 15 % had left lobe abscess. Right upper quadrant pain and fever were found to be the common clinical symptoms.[25]

Abdullah A A in a clinical analysis of 33 Kurdish patients of amoebic liver abscess in Sulaimany University observed a male: female ratio of 13:20. Abdominal pain and fever were present in 82 % and 72 % of cases followed by abdominal tenderness and hepatomegaly in 67% and 62 % of cases.[26]

Kebede A, et al in a retrospective study on amoebic liver abscess over a 20 year study period from 1982 to 2002 found an incidence of 2.35 for 5854 hospitalizations (0.04%) per year. They have observed

right upper quadrant pain (88%) and fever (75%) as most common symptoms followed by hepatomegaly (69%). Right lobe involvement was seen in 94% and left lobe involvement in 13 % of patients in their study.[27]

Sharma N, et al in a retrospective study on 86 cases of amoebic liver abscess over a 5 year period from 2000 to 2004 in a tertiary referral centre in North India found a male: female ratio of 7:1. History of alcohol consumption was seen in 46.5 % of patients. Fever and abdominal pain were common symptoms in 94% and 90% of patients respectively. Diarrhea was seen in only 10.5% of patients. Right lobe involvement was seen in 65 % of patients. Left lobe and multiple abscess involvement were 13% and 22% respectively. They have concluded that in patients presenting to emergency department with prolonged fever and abdominal pain amebic liver abscess has to be suspected.[28]

K Seeto R, et al reviewed 56 patients with amoebic liver abscess in a tertiary hospital in San Francisco over a 15 year period from 1979 to 1994 observed alcohol consumption in about 84% of patients and have opined alcohol being an immunosuppressant, impairs kuppfer cell function and suppresses cellular and humoral immunity against Entamoeba histolytica.[29]

Ahsan I, et al on a study on clinical features and complications of liver abscess in 52 patients observed a male: female ratio of 8:1. 88 % of patients were amoebic and 12 % were pyogenic abscess. 20 % of patients with amoebic abscess had diarrhea while it was absent in pyogenic group. They also observed multiple abscesses in 22 % of amoebic abscess and 100% in pyogenic abscess group.[30]

Alvarez J A, et al on a retrospective study on pyogenic liver abscess over a period of 13 years from 1985 to 1997 in a tertiary hospital in Spain found a total of

133 patients of whom 97 were solitary and 36 were multiple abscesses with a male: female ratio of 1.6: 1. They found that ascending cholangitis of biliary origin more common in multiple abscesses and multiple abscesses were more frequent in right lobe.[31]

Bukhari A J, et al in a prospective study on clinical presentation of amoebic liver abscess over a two year study period in a tertiary hospital in Lahore from 2000 to 2002 observed in a total of 53 patients, the male: female ratio was 2:1. Upper abdominal pain and fever was noted in 90% and 85% of patients. Diarrhoea was noted only in 7.5% of patients. Hepatomegaly was noted in 79% and Jaundice in 20 % of patients. Of these 83% were solitary abscess and the remaining 17 % were multiple.[18]

Goh K L, et al in a review of 204 cases of liver abscess over a 16 year period in a tertiary hospital in Kuala lampur observed a male: female ratio of 6:1. Fever and

right hypochondrial pain were found in 80% and 70 % of patients with amoebic abscess and diarrhea in 20% of patients. 75 % of multiple abscess were pyogenic and only 25 % were amoebic. Eschericia coli was cultured in 33% of cases of pyogenic abscess aspirate followed by Klebsiella in 25 % of cases.[32]

Wang J H, et al in a retrospective study over a seven years period from 1990 to 1996 in Taiwan on pyogenic liver abscess found 182 cases. They found that 160(87.9%) cases were due to Klebsiella pneumoniae and 22 cases were polymicrobial. Of the 160 cases due to K.pneumaniae 108 (67.5%) had frank diabetes and only 1 (4.5%) case was diabetic in the polymicrobial group.[3]

Pang T C, et al in a study of 63 patients with pyogenic liver abscess over an eleven year study period from 1998 to 2008 in an Australian tertiary hospital observed a male: female ratio of 2:1. Streptoccoccus

milleri was found in 25% of cases followed by Klebsiella pneumonia in 21% and Eschericia coli in 16% of patients of pyogenic liver abscess in their study.[33]

Rahimian J, et al on a review of 79 patients with pyogenic liver abscess in a tertiary hospital in NewYork over a study period of ten years from 1993 to 2003 found 43% of patients had underlying biliary disease and Klebsiella pneumoniae was isolated from 41% of patients.[9]

Tan J A, et al in a study on 15 patients of amoebic liver abscess over a period of one year in1986 in a referral hospital in Phillipines observed a male: female ratio of 4:1. Indirect hemagglutination test for antibody of Entamoeba histolytica was found to be positive in a titre of 1:128 or more in all the patients with amoebic liver abscess in their study.[34]

Hayat A S, et al in a case control study on management of amoebic liver abscess in 50 patients in a tertiary hospital in Pakistan have observed that ultrasound guided needle aspiration along with metronidazole has better therapeutic results in abscesses more than 10 cm in diameter, left lobe abscess and in abscess imminent to rupture rather than treatment with metronidazole alone.[35]

Memon S, et al in a retrospective study on 1083 patients of amoebic liver abscess over a 22 year period from 1986 to 2007 in a tertiary referral hospital in Pakistan found 36 (3.36%) patients with intraperitoneal rupture of abscess. Of these 16 (44.44%) of patients underwent laparotmy and 20 (55.55%) patients were treated with ultrasound guided pus aspiration and drain placement. They found that 6 (37.5%) patients who underwent laparotomy died whereas 1(5%) patient died in ultrasound guided aspiration group.[36]

Zerem E, et al studied the effectiveness of percutaneous catheter drainage with percutaneous needle aspiration in 60 patients with pyogenic liver abscess over a five year period from 2002 to 2006 in Bosnia. They found percutaneous needle aspiration was successful in 67% of patients after single or multiple aspirations whereas percutaneous catheter drainage was curative in 100% of patients. They have concluded percutaneous needle aspiration is useful in simple abscesses less than 5 centimetres in diameter.[20]

Razaque A, et al on a prospective study on Liver abscess over a period of 5 years from 2003 to 2008 in a tertiary hospital in Pakistan observed that in a total of 55 cases the male: female ratio was 4:1. About 80% of abscesses were solitary and right lobe involvement was seen in 70% of cases. Fever and abdominal pain was seen in 81% and 72% of cases. 51 % of patients

had positive bacteriological culture, of which half of them were due to Escherichia coli and one fourth was due to Klebsiella pneumoniae. 25% of patients had serology positive for Entamoeba histolytica. Percutaneous needle aspiration with antibiotics was effective in 60% of patients, 22% of patients needed surgical drainage and 18% of patients responded to antibiotic therapy alone.[37]

Garr P M, et al in their prospective study on amoebic liver abscess in 178 patients over a study period of one year during 1997-1998 in a tertiary hospital have observed conservative management of amoebic liver abscess in 150 patients and ultrasound guided aspiration of abscess in 23 patients. They have concluded conservative management of amebic liver abscess is safe and percutaneous ultrasound guided aspiration is needed only jf patients fail to respond after 48-72 hours.[38]

Ramani A, et al in a prospective study over a period of two and a half years from 1989 to 1991 in a tertiary hospital in Manipal, India on 200 patients of amoebic liver abscess compared the role of ultrasound guided needle aspiration of amoebic liver abscess and drug therapy with drug treatment alone. They found that 87% of the abscesses were in right lobe, 10% were in left lobe and 3% were multiple. Solitary abscess was found in 82% of patients and multiple abscesses were seen in 18% of patients. 60 % of patients received anti amoebic drugs alone and 35% of patients underwent percutaneous ultrasound guided needle aspiration and drugs. 5% of patients needed surgical drainage. They found that the percutaneous needle aspiration group had a rapid clinical resolution of symptoms and resolution of abscess cavity and early discharge which was statistically significant compared to the group receiving drugs alone.[39]

Perez C I, et al in a study on thoracic complications of amebic liver abscess on 501 patients over an 18 year study period from 1961 to 1979 found that about 60% of thoracic complications of amoebic liver abscess is due to rupture through diaphragm, 35% of patients presented with pleural effusion and pneumonitis and about 2% with pericarditis. They have concluded that drainage of pleural or pericardial contents in ruptured amebic liver abscess and promotion of bronchial drainage improves outcome.[40]

SUBJECTS AND METHODS

This study was conducted in Government Mohan Kumaramangalam Medical College Hospital, Salem from June' 2010 to November' 2012.

CASE SELECTION

The study population consists of patients admitted in General Surgery wards of Government Mohan Kumaramangalam Medical College Hospital, Salem with features suggestive of Liver Abscess.

INCLUSION CRITERIA

Patients admitted in General Surgery wards of Government Mohan Kumaramangalam Medical College Hospital, Salem confirmed to be of liver abscess.

EXCLUSION CRITERIA

1. Hydatid cyst of Liver
2. Solid masses of the Liver

3. Primary and Secondary malignancy of Liver

A total of 99 cases satisfying the inclusion criteria were recruited in the study.

MODE OF EVALUATION

An extensive and thorough History taking

Clinical examination

Routine blood investigations

Chest X-ray PA view

X-ray abdomen AP view

Ultrasound abdomen

CT scan abdomen (In selected cases)

Culture and sensitivity of the aspirate

and the findings were entered in the proforma as shown in Appendix ii.

STATISTICAL ANALYSIS

Proportions (%) of various outcome measures of interest of liver abscess were arrived and tabulated.

RESULTS

A master chart shown in Appendix-iii is designed for the data collected during the study period and various epidemiological and statistical details are analyzed and the results calculated with graphs and charts at appropriate places for better understanding.

AGE INCIDENCE

In our study out of 99 cases, there were patients ranging from lowest age of 30 years and highest age of 70 years. In the age group from 30-40 years, 16 (16.16%) cases were seen. In the age group 41-50 years there were 48 (48.48%) cases. In the age group 51-60 years there were 20 (20.20%) cases. In the age group more than 60years 15 (15.15%) cases. (Table-1 & Fig.9)

TABLE - 1

AGE INCIDENCE OF LIVER ABSCESS

AGE GROUP	NO.OF PATIENTS	PERCENTAGE
30-40 YEARS	16	16.16
41-50 YEARS	48	48.48
51-60 YEARS	20	20.20
>60 YEARS	15	15.15

Nearly half (48%) of patients were in the age group of 41-50 years in our study.

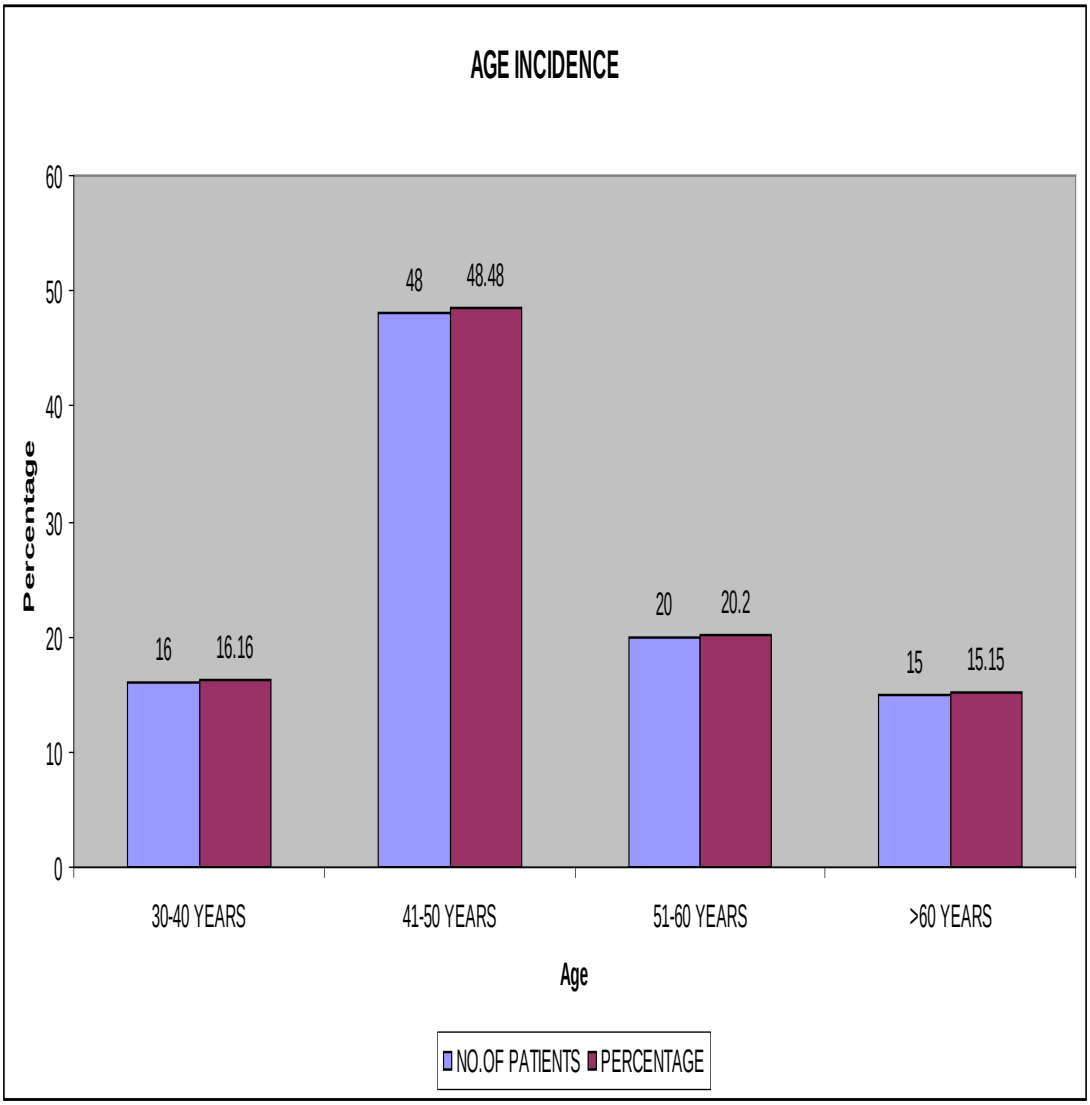

FIG.9. AGE INCIDENCE OF LIVER ABSCESS

SEX DISTRIBUTION

In our study out of 99 cases majority of cases 97 (97.97%) were males and only 2 cases (2.02%) were females. (Table-2 & Fig.10)

TABLE - 2

SEX DISTRIBUTION OF LIVER ABSCESS CASES

SEX	NUMBER OF PATIENTS	PERCENTAGE
MALE	97	97.97
FEMALE	2	2.02

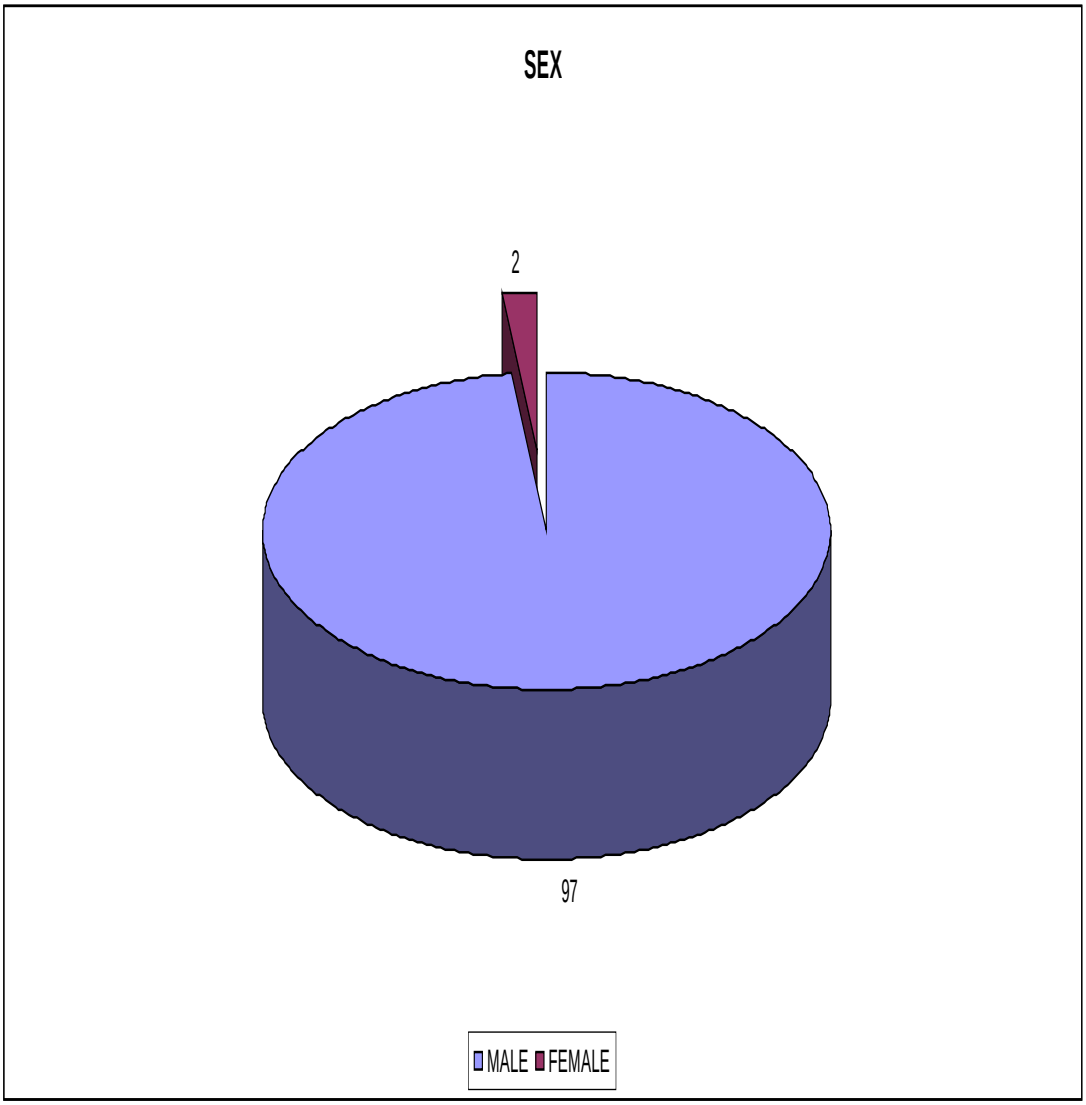

FIG. 10. SEX DISTRIBUTION OF LIVER ABSCESS CASES

HISTORY OF ALCOHOL INTAKE

In our study of 99 cases history of alcohol intake was found in 61 cases. Of these cases, amoebic abscess were 58(95%) and pyogenic abscess were 3(5%). (Table-3 & Fig.11)

TABLE – 3

HISTORY OF ALCOHOL INTAKE IN LIVER ABSCESS

HISTORY OF ALCOHOL INTAKE	NUMBER OF PATIENTS	PERCENTAGE
Amoebic	58	95
Pyogenic	3	5

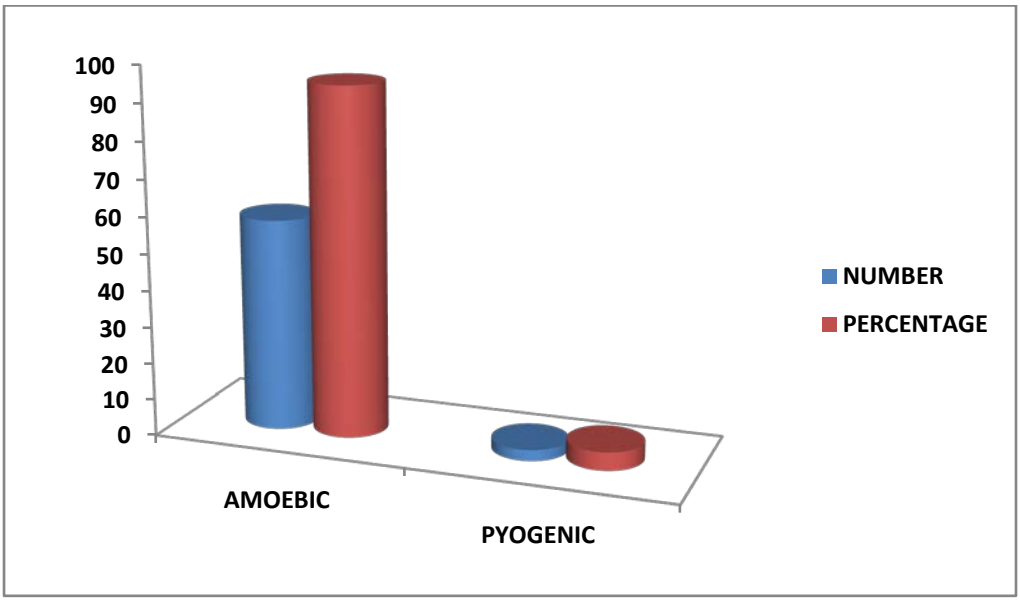

FIG.11. HISTORY OF ALCOHOL INTAKE IN LIVER ABSCESS

CLINICAL SYMPTOMS

All the cases of liver abscess presented with abdominal pain. 51 (51.51%) cases had abdominal distension. 57 (57.57%) cases presented with fever and 16 (16.16%) cases had dysentery. (Table-4 & Fig.12)

TABLE – 4

SYMPTOMS AMONG CASES OF LIVER ABSCESS

CLINICAL SYMPTOMS	NUMBER OF PATIENTS	PERCENTAGE
Abdominal pain	99	100
Fever	57	57.57
Abdominal distension	51	51.51
Dysentery	16	16.16

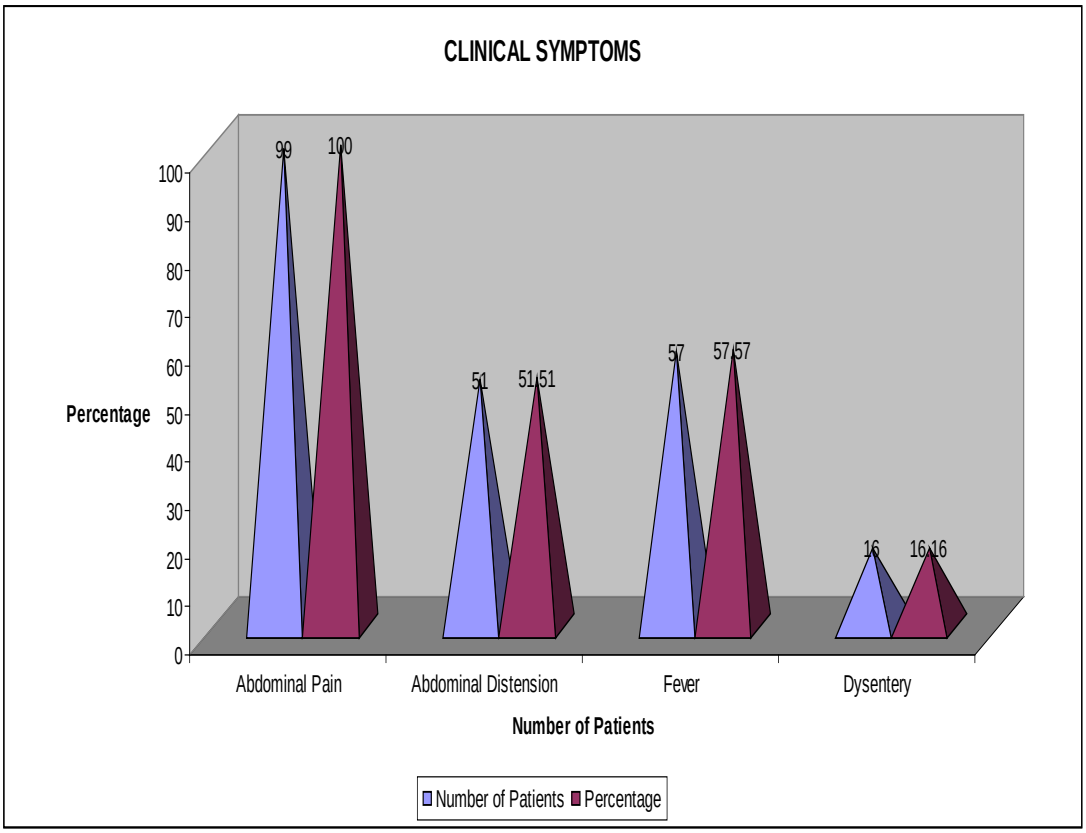

FIG. 12. CLINICAL SYMPTOMS OF LIVER ABSCESS

CLINICAL SIGNS

In our study all liver abscess cases 99 (100%) presented with right hypochondrial pain and intercostal tenderness. 46 (49.49%) cases presented with hepatomegaly, 25 (25.25%) cases were observed to have jaundice and 12 (12.12%) cases had epigastric mass. (Table-5 & Fig.13)

TABLE – 5

CLINICAL SIGNS OF LIVER ABSCESS CASES

CLINICAL SIGNS	NUMBER OF PATIENTS	PERCENTAGE
Right hypo chondrial tenderness	99	100
Intercostal tenderness	99	100
Hepatomegaly	49	49.49
Jaundice	25	25.25
Epigastric mass	12	12.12

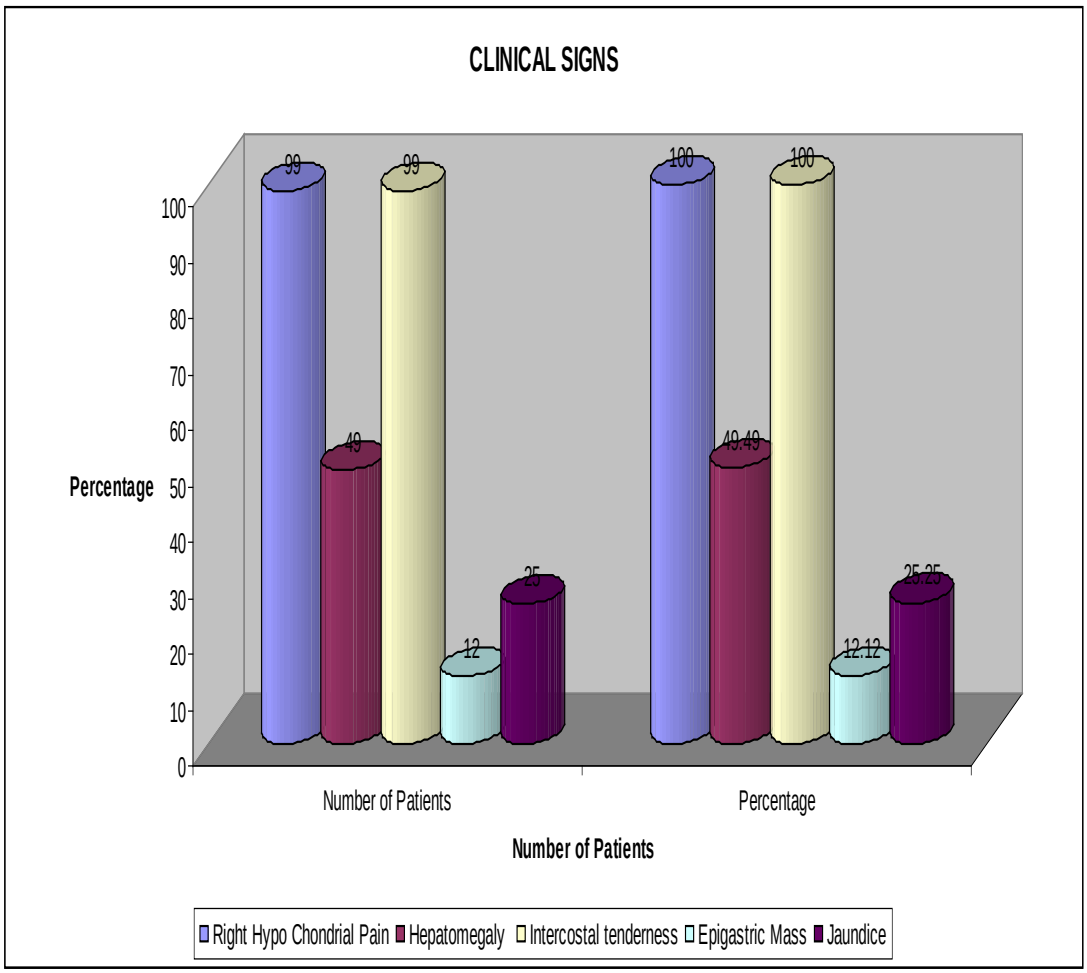

FIG.13. CLINICAL SIGNS OBSERVED IN LIVER ABSCESS

LOBE INVOLVEMENT AND NUMBER OF ABSCESS

In our study out of 99 cases 70 (70.7%) cases were presented with right lobe abscess, 8 (8.08%) cases with left lobe abscess, 5 (5.05%) cases presented with multiple abscess and 21 (21.21%) cases were found to be ruptured abscess at initial presentation. (Table-6 & Fig.14)

TABLE – 6

LOBE INVOLVEMENT AND NUMBER OF ABSCESS

PRESENTATION	NUMBER OF PATIENTS	PERCENTAGE
Right lobe	70	70.7
Ruptured abscess	21	21.21
Left lobe	8	8.08
Multiple abscess	5	5.05

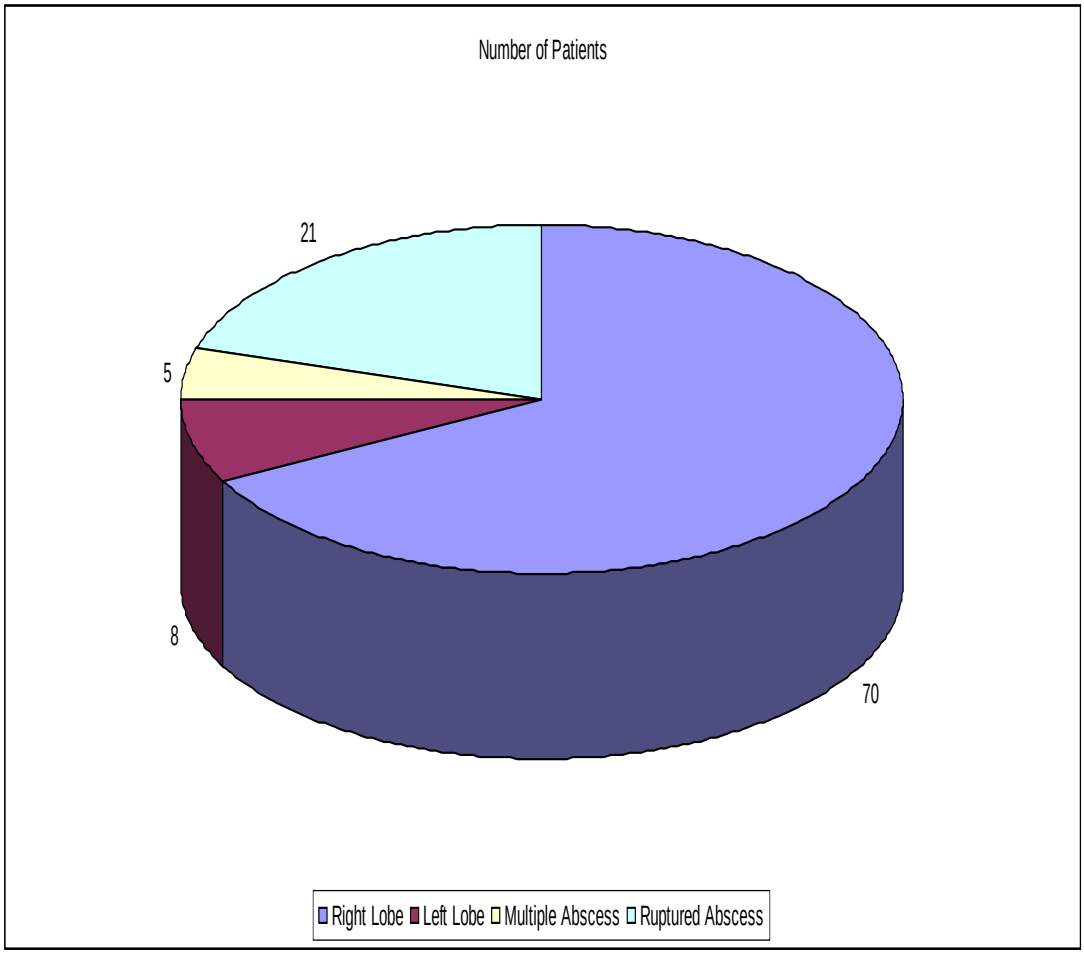

FIG.14. LOBE INVOLVED AND NUMBER OF LIVER ABSCESS

ETIOLOGY OF LIVER ABSCESS

In our study 90 (90.09%) cases were amoebic Abscess and 9 (9.09%) cases were pyogenic abscess in etiology. (Table-7 & Fig.15)

TABLE – 7

ETIOLOGY OF LIVER ABSCESS

PRESENTATION	NUMBER OF PATIENTS	PERCENTAGE
Amoebic	90	90.9
Pyogenic	9	9.09

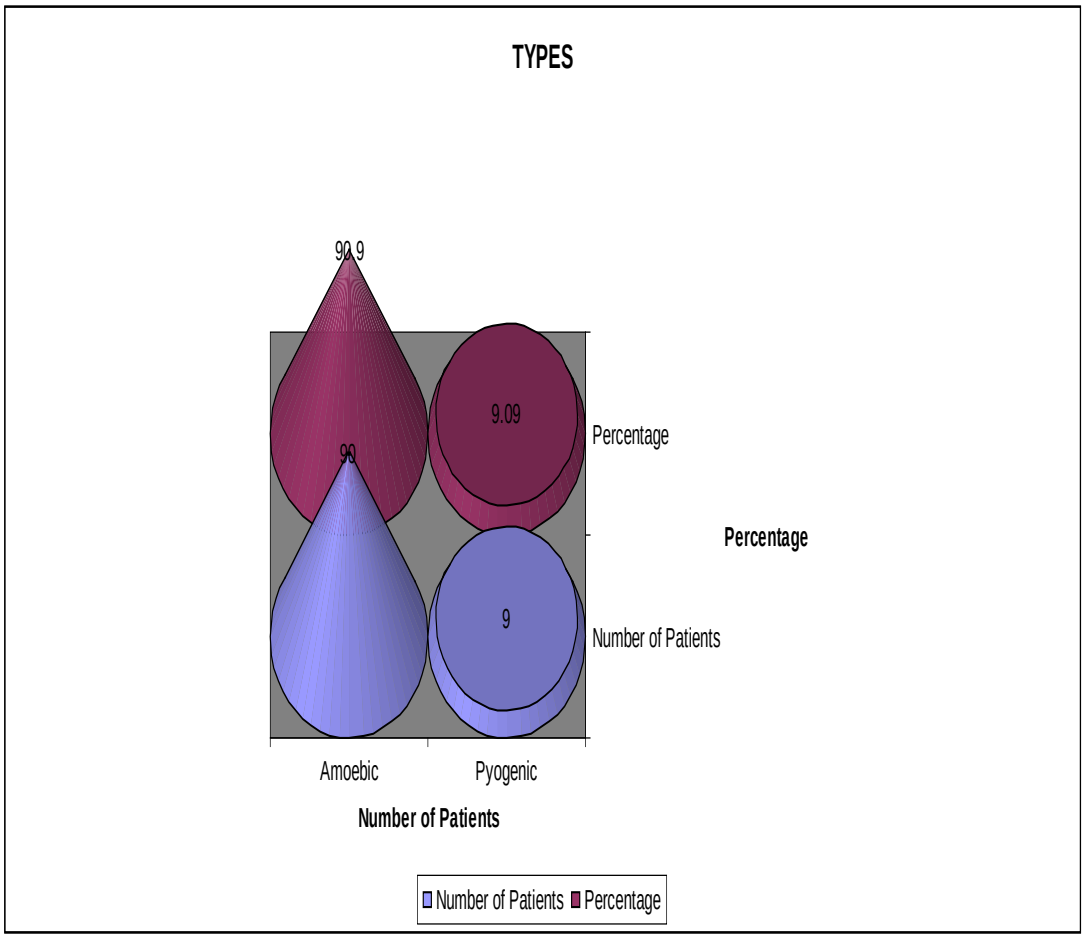

FIG.15. ETIOLOGY OF LIVER ABSCESS

TREATMENT OF LIVER ABSCESS

In our study out of 99 cases, 30 (30.3%) cases were treated with single aspiration, 25 (25.25%) cases by percutaneous catheter drainage. 22 (22.22%) cases underwent laparotomy and drainage. 19 (19.09%) cases were treated by multiple aspiration and 3 (3.03%) cases by conservative line of management. (Table-8 & Fig.16)

TABLE – 8

MODE OF TREATMENT OF LIVER ABSCESS

TREATMENT	NUMBER OF PATIENTS	PERCENTAGE
Single aspiration	30	30.3
Percutaneous catheter drainage	25	25.25
Laparotomy and drainage	22	22.22
Multiple aspiration	19	19.19
Conservative management	3	3.03

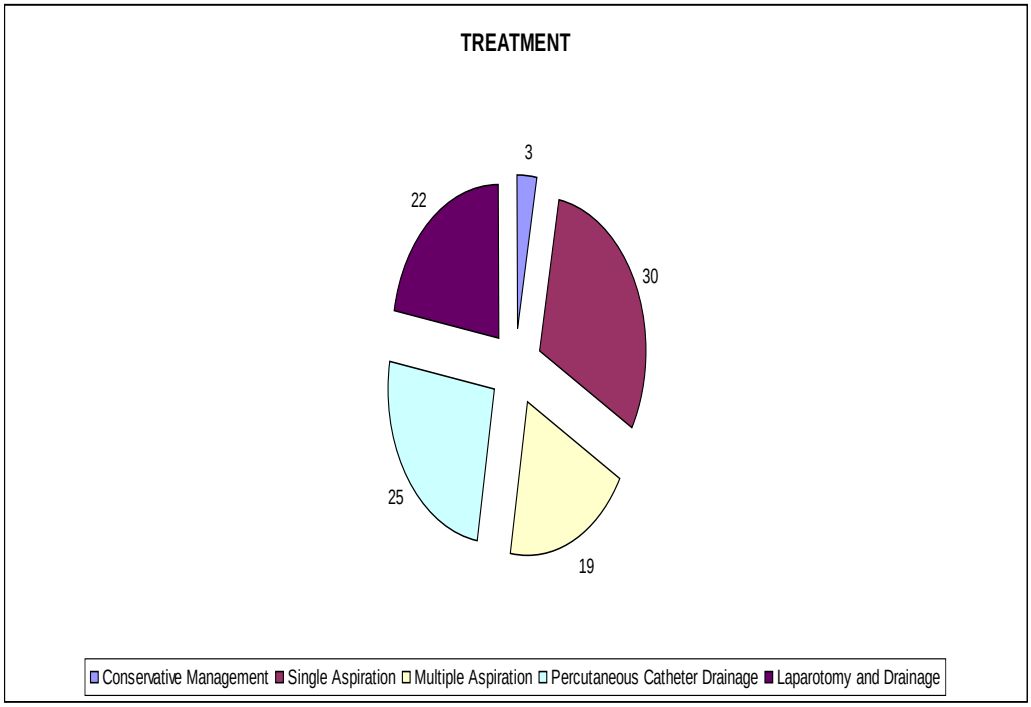

FIG.16. MODE OF TREATMENT OF LIVER ABSCESS

DISCUSSION

Liver abscess continues to be one of the common liver disorders even in the era of improved sanitation and personal hygiene as well as availability of wide range of antimicrobials and antibiotics. This study explores the etiology, clinical presentation and modalities of management of liver abscess cases as seen in a tertiary care referral institution.

INCIDENCE

The number of hospitalizations from June' 2010 to November' 2012 in our surgical ward were 33,769. The number of liver abscess cases admitted in during that period was 99. This accounts for about (0.29%) per year. This is concurrent with the study of **Kebede A, et al.**[27]

AGE

Most of the liver abscess cases (48.8%) in our study were in the age group of 41-50 years. **Seeto R K, et al; Tan J A, et al** have also found this age group to be susceptible for liver abscess.[34] **Ahsan I Et al** in their study have found this age group similar for pyogenic abscess in their study.[30] **Abdullah A A, et al** in their study on amoebic liver abscess have found that amoebic liver abscess is more common in the age group of 20-45 years and differ from our study.[26]

SEX

We have observed male preponderance (97.97%) in our study which concurs with the observation by **Sharma N, et al; Ahsan I, et al; Goh K L, et al and Tan J A et al.**[28, 30, 32, 34] Male predominance is due to different life styles of men and women of our country with males going out for work consume contaminated water and unhygienic food

from street vendors and road side hotels whereas women are mostly house bound.[30]

HISTORY OF ALCOHOL INTAKE

In our study about 61 (62%) of patients with liver abscess had history of alcohol intake, of which 58 (95%) cases were amoebic abscess and 3 (5%) cases were pyogenic abscess. **Sharma N, et al** noted history of alcohol consumption in (46.5%) of patients and **Seeto R K, et al** noted it in (84%) of patients in their study respectively. Alcohol being an immunosuppressant, impairs kuppfer cell function and suppresses cell mediated and humoral immunity against Entamoeba histolytica.[28, 29]

ETIOLOGY

In our study, about 91% were amoebic abscesses and 9% were pyogenic abscesses. Eschericia coli was the cause for 9% of patients with pyogenic abscess in our study. Eschericia coli and Klebsiella

pneumonia were the leading causes of pyogenic liver abscess observed in various studies.[3, 9, 32, 33, 37]Streptococcus milleri too was reported by Pang T CY, et al.[33] Polymicrobial etiology was noted by **Wang JH, et al.**[3]

CLINICAL SYMPTOMS

The commonest clinical presentation observed in our study was abdominal pain which was found in 100% of cases. This was followed by abdominal distension and fever in about half of the cases. The incidence of dysentery in our study was 16.16%.This is similar to the observations made by **Bukhari A J, et al; Abdullah A A; Kebede A, Et al and Sharma N, et al.; Seeto K R, Et al.**[18, 26, 27, 28, 29] The low incidence of dysentery compared to other symptoms is due to invasive form of amoebiasis causing amoebic liver abscess and pyogenic liver abscess occurs mainly due to biliary etiology.

CLINICAL SIGNS

On examination all liver abscess cases in our study had right hypochondrial tenderness and intercostal tenderness. Hepatomegaly was noted in about half of the cases and jaundice was present in about one fourth of cases. The earlier reports observed by **Abdullah A A; Kebde, et al** [26, 27] were concurrent with the findings of our study. In addition mass in the epigastric region was noted in 12.12 % of patients in our study.

PRESENTATION

The most commonly involved region of the liver in our study was the right lobe in about 71% of cases which is in accordance with the findings observed by **Khan R A et al, Kebede A, et al Sharma N et al, Qazi A R et al** [25, 27,28 37] in their studies. The reason why right lobe of the liver is more prone to develop abscess than the left lobe is due to greater volume of blood going to right side than the left

lobe.[37]About 5% of patients presented with multiple abscesses and all were amoebic in our study contrary to the observations made by **Sharma N, et al; AhsanI, et al; Alvarez JA, et al; Goh K L, et al.** [28,30,31,32] where multiple abscesses were predominantly pyogenic. **Bukhari A J, et al** reported predominantly (83%) solitary abscess in their study which was similar to about 80% in our study.[18]Twenty one percentage of patients presented with ruptured abscess all of which were peritoneal rupture which was in accordance with the observations by **Hayat A S, et al; Memon A S et al; Perez C I, et al.**[35, 36, 40]Elevation of right hemidiaphragm was noted in 31% of patients and right sided pleural effusion was noted in 21% of patients in our study.

TREATMENT

Most of the liver abscess cases were managed surgically in our study. About 30% of cases were treated by single aspiration and 20% of cases by

multiple aspirations. Percutaneous catheter drainage was the mode of treatment in 25% of cases. 22% of cases needed laparotomy and drainage all of which were ruptured abscesses. 3% of patients in whom the abscess size was less than 5 centimetres were managed conservatively. **Zerem E, et al** reported needle aspiration either single or multiple was successful in 67% of patients and percutaneous catheter drainage was successful in 100% of patients.[20] A similar observation was made by **Qazi A R, et al; McGarr P L, et al; Ramani a, et al.**[37, 38, 39]

CO-EXISTENT HIV INFECTION

About 3 (3.03%) of patients had co-existent HIV infection in our study and all of them were males with amoebic liver abscess.

MORTALITY

There was 1 (1.01%) death in our study; a male patient aged 70 years who underwent laparotomy and drainage for ruptured amoebic liver abscess.

CONCLUSIONS

Liver abscess constitutes 0.29% of total surgical ward hospitalizations per year.

Liver abscess was more commonly seen in the age group of 41-50 years.

Male predominance 97.9% was seen in liver abscess.

Nearly two thirds of cases of liver abscess had a history of alcohol intake.

Majority of liver abscess were amoebic and nearly 10% were pyogenic.

Eschericia coli was the commonest organism causing pyogenic liver abscess.

Abdominal pain was the commonest symptom of liver abscess followed by abdominal distension and fever.

Right hypochondrial tenderness and intercostal tenderness were the common clinical signs in liver abscess.

There was a predominant involvement of the right lobe of the liver.

Percutaneous aspiration of abscess with anti amoebic and antibiotics forms the mainstay of treatment.

Percutaneous catheter drainage is method of choice in failed aspiration and laprascopic drainage or laparotomy and drainage is indicated in ruptured liver abscess

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PROFORMA

Name :

Age / Sex :

I.P. No. : Occupation:

Address :

HISTORY

Abdominal Pain : Mode of onset Site Character

Radiation Referred Pain

Fever :

Abdominal Distension :

Vomiting : Frequency Quantity

Relationship

Dysentry :

Appetite : Alcohol Intake:

PHYSICAL EXAMINATION

Appearance : Obese Well Built Thin Built

Pulse: /min B.P: mmof Hg R.R: /min

Temperature : Normal Elevated

Anemia

Jaundice

Clubbing

Lymphadenopathy

INSPECTION

Contour of Abdomen	:	Distended	Normal
Respiratory Movement	:		
Peristaltic Movement	:		
Pulsatile Swelling	:		
Skin over the abdomen:			
Umbilicus	:	Normal	Abnormal
Abdominal Distension	:		
Hernial Orifices	:		

PALPATION:

Temperature	:	Normal	Abnormal
Rt. Hypochondrial tenderness	:	Present	Absent
Intercostal Odema	:	Present	Absent
Intercostal tenderness	:	Present	Absent

Hepatomegaly	:	Present	Absent
Muscular Rigidity	:	Present	Absent
Rebound Tenderness	:	Present	Absent
Distension	:	Present	Absent
Lump	:	Present	Absent
Palpation of Hernial Orifices	:	Normal	Abnormal

PERCUSSION

Upward enlargement :

Fluid Thrill :

Shifting Dullness :

Obliteration of Liver dullness :

AUSCULTATION

ABDOMINAL GIRTH MEASUREMENT

PER RECTAL EXAMINATION	Normal	Abnormal
DOA:	DOS:	DOD:

TREATMENT DETAILS:

MASTER CHART

S.NO	PATIENT NAME	AGE	SEX	IN PATIENT NUMBER	HISTORY OF ALCOHOL	CLINICAL SYMPTOMS	CLINCAL SIGNS	PRESENTATION R.LOBE/L.LOBE/MULTIPLE	AMEBIC ABSCESS	PYOGENIC ABSCESS	TREATMENT
1	POOMALAI	70	M	90429	YES	Abd. P, F	I.T.	L.LOBE	A	-	S.A
2	PERIYASAMY	44	M	90487	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	R.LOBE	A	P	M.A
3	SAKTHIVEL	30	M	30975	NO	Abd. P, F, Dy	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	S.A
4	RANGANATHAN	46	M	27302	YES	Abd. P, Dy	I.T., HEPATO	L.LOBE	A	-	S.A
5	PALANISAMY	45	M	37913	NO	Abd. P, Abd. D, F, Dy	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	M.A
6	RAJANGAM	9	M	24077	YES	Abd. P, F	I.T., HEPATO, JAUNDICE	MULTIPLE/R.LOBE	A	-	M.A
7	PERUMAL	65	M	27545	NO	Abd. P, F	I.T., HEPATO	MULTIPLE/L.LOBE	A	-	S.A
8	MUNIYAPPAN	60	M	3862	YES	Abd. P, F	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	PERCUT. CATH. DRAIN
9	MANI	55	M	11908	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	LAPARATOMY & DRAIN
10	NATESAN	55	M	19547	NO	Abd. P, F	I.T., HEPATO-	R.LOBE	A	-	S.A
11	MALLAPPAN	70	M	17098	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	PERCUT. CATH. DRAIN
12	SIVALINGAM	45	M	24803	YES	Abd. P,F	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
13	RAMASAMY	47	M	15007	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	LAPARATOMY & DRAIN

14	SUNDARAM	49	M	17114	NO	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	LAPARATOMY & DRAIN
15	MANI	35	M	15383	NO	Abd. P, F	I.T.	R.LOBE	A	P	S.A
16	RAJENDRAN	44	M	743	YES	Abd. P, Dy, F	I.T.	R.LOBE	A	-	S.A
17	DURAISAMY	65	M	7966	NO	Abd. P, F	I.T.	R.LOBE	A	P	S.A
18	SUBRAMANI	50	M	29998	NO	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	LAPARATOMY & DRAIN
19	ARUMUGAM	55	M	25227	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	LAPARATOMY & DRAIN
20	KANDASAMY	41	M	13683	NO	Abd. P, Abd. D	I.T., HEPATO	L.LOBE	A	-	S.A
21	DHANAKODI	44	M	19044	YES	Abd. P, Abd. D, F	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	S.A
22	KARUPAIYAN	49	M	20126	YES	Abd. P, F	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	EMERG. LAPAR & DRAIN
23	MURUGESAN	40	M	31217	YES	Abd. P, F	I.T.	R.LOBE	A	-	S.A
24	SARAVANAN	43	M	31207	YES	Abd. P, Abd. D, F	I.T., JAUNDICE	R.LOBE	A	-	S.A
25	ARUMUGAM	70	M	31368	NO	Abd. P, F	I.T.	R.LOBE	A	P	S.A
26	CHINNAPPAN	55	M	31309	NO	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	-	P	S.A
27	RAJENDRAN	35	M	32427	NO	Abd. P, Abd. D, F	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
28	PALANISAMY	43	M	33780	NO	Abd. P, Abd. D, F	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
29	PALANI	50	M	34835	NO	Abd. P, Dy.	I.T.	R.LOBE	A	-	S.A
30	SENGODAN	40	M	36038	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	M.A
31	NATARAJ	35	M	37282	YES	Abd. P, Abd. D	I.T.	R.LOBE	A	-	M.A
32	JOSEPH	50	M	42147	YES	Abd. P, F	I.T.	R.LOBE	A	-	PERCUT.

											CATH. DRAIN
33	VELU	70	M	53072	NO	Abd. P, Abd. D	I.T., HEPATO, JAUNDICE	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
34	MARIAPPAN	45	M	34434	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
35	MANICKAM	43	M	42141	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
36	PALANIVEL	40	M	47666	YES	Abd. P, F	I.T., JAUNDICE	L.LOBE	A	-	PERCUT. CATH. DRAIN
37	MANICKAM	43	M	46497	YES	Abd. P, Abd. D	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
38	SIVAKUMAR	40	M	39412	NO	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
39	KANNAN	48	M	1423	NO	Abd. P, F, Dy.	I.T., JAUNDICE	R.LOBE	A	-	PERCUT. CATH. DRAIN
40	CHINNARAJ	44	M	50806	NO	Abd. P, Abd. D, F, Dy.	I.T., JAUNDICE	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
41	KARUPPAIAH	70	M	6483	YES	Abd. P, Abd. D, Dy.	I.T., HEPATO-	R.LOBE/MULTIPLE	A	-	S.A
42	KUMAR	38	M	7528	YES	Abd. P, Abd. D, F, Dy.	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
43	KRISHNAN	50	M	7531	YES	Abd. P, Abd. D, F	I.T., JAUNDICE	R.LOBE	A	-	PERCUT. CATH. DRAIN

44	MOORTHY	56	M	7541	YES	Abd. P, Abd. D, Dy.	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
45	RANGAN	49	M	8633	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	M.A
46	SETTU	47	M	35209	YES	Abd. P, Abd. D, F	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
47	SURESH	42	M	8639	YES	Abd. P, Abd. D	I.T.	R.LOBE	A	-	S.A
48	MURUGESAN	70	M	8666	YES	Abd. P, F	I.T.	R.LOBE	-	P	S.A
49	VENKATCHALM	43	M	981	NO	Abd. P, Abd. D, F	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
50	GOVINDAN	50	M	9879	YES	Abd. P, F	I.T.	R.LOBE	A	-	M.A
51	ARUMUGAM	57	M	14811	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
52	KALIAPPAN	49	M	15811	YES	Abd. P, F	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
53	AYANPERUMAL	62	M	21887	NO	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
54	ARUMUGAM	41	M	16926	YES	Abd. P, F	I.T.	R.LOBE	A	-	M.A
55	SRINIVASAN	47	M	36908	NO	Abd. P, Abd. D	I.T.	R.LOBE	A	-	S.A
56	DURAI	60	M	37007	NO	Abd. P, F	I.T.	L.LOBE	-	P	S.A
57	SELVAM	42	M	40200	NO	Abd. P, Dy.	I.T., JAUNDICE	R.LOBE	A	-	PERCUT. CATH. DRAIN
58	RAJI	70	M	40585	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
59	MANI	35	M	51711	YES	Abd. P, F	I.T. JAUNDICE	R.LOBE	A	-	PERCUT.

											CATH. DRAIN
60	AYUP	47	M	40865	NO	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
61	ANGAMUTHU	60	M	25516	YES	Abd. P, Abd. D	I.T.	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
62	THERTHAMALAI	40	M	62837	YES	Abd. P, F	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
63	KANDASAMY	44	M	60447	YES	Abd. P, Abd. D, F	I.T.	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
64	GOVINDARAJ	55	M	14182	YES	Abd. P, Abd. D.	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
65	GNANPRAKASH	43	M	15016	YES	Abd. P, F	I.T.	R.LOBE	A	-	S.A
66	MANI	45	M	15386	NO	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
67	RAJA	39	M	15307	NO	Abd. P, Dy.	I.T.	R.LOBE	A	-	M.A
68	VEERAVEL	62	M	23405	NO	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	S.A
69	RAJAN	47	M	53067	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE/MULTIPLE	A	-	M.A
70	DHANAKODI	70	M	19044	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
71	MADHU	44	M	2090	NO	Abd. P, F, Dy.	I.T.	R.LOBE	A	-	S.A
72	MOORTHY	49	M	14526	NO	Abd. P, F	I.T.	L.LOBE	A	-	S.A
73	CHINCHANRAN	54	M	15774	NO	Abd. P, F, J	I.T., HEPATO, JAUNDICE	R.LOBE	A	-	M.A

74	PERIYASAMY	59	M	17634	YES	Abd. P, F,	I.T., HEPATO	R.LOBE/MULTIPLE	A	-	PERCUT. CATH. DRAIN
75	DURAISAMY	41	M	22501	YES	Abd. P, F, Dy.	I.T.	RL.LOBE	A	-	S.A
76	THOPPAIYAN	47	M	22981	NO	Abd. P, F	I.T., JAUNDICE	R.LOBE	A	-	M.A
77	MURUGESAN	60	M	31217	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
78	RAJEE	54	M	48598	YES	Abd. P, Abd. D	I.T., HEPATO	RUPTURED ABSCESS	A	-	EMERG. LAPAR & DRAIN
79	MADHU	42	M	25847	YES	Abd. P, F	I.T.	R.LOBE	A	-	S.A
80	RANGARAYAN	40	M	56951	YES	Abd. P, Abd. D, F	I.T., JAUNDICE	R.LOBE	-	P	S.A
81	MADESWARAN	70	M	58141	NO	Abd. P, F	I.T.	R.LOBE	A	-	M.A
82	ANGAPPAN	43	M	9118	YES	Abd. P, Dy.	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
83	PALANIMUTHU	60	M	78949	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN
84	ASHOK	41	M	34099	YES	Abd. P, F	I.T.	L.LOBE	A	-	PERCUT. CATH. DRAIN
85	PALANI	65	M	56655	NO	Abd. P, F	I.T., HEPATO	R.LOBE	-	P	PERCUT. CATH. DRAIN
86	MUTHUSAMY	47	M	34982	NO	Abd. P, Abd. D	I.T.	R.LOBE	A	-	PERCUT. CATH. DRAIN
87	ARUMUGAM	60	M	31669	NO	Abd. P, F	I.T., JAUNDICE	R.LOBE	A	-	M.A
88	PERUMAL	49	M	31684	YES	Abd. P, Abd. D	I.T.	R.LOBE	A	-	M.A
89	MUTHU	44	M	33193	YES	Abd. P, F, J	I.T., HEPATO	R.LOBE	A	-	M.A
90	SETTU	62	M	35209	NO	Abd. P, Dy.	I.T.	R.LOBE	A	-	S.A

91	VENKATESAN	35	M	35236	YES	Abd. P, Abd. D	I.T., HEPATO	R.LOBE	A	-	M.A
92	PERUMAL	50	M	41295	YES	Abd. P, Abd. D, F	I.T.	R.LOBE	A	-	S.A
93	MOORTHY	40	M	42476	YES	Abd. P, Abd. D	I.T.	R.LOBE	A	-	M.A
94	KANDASAMY	55	M	46775	YES	Abd. P, F	I.T.	R.LOBE	A	-	M.A
95	THEERTHAMAL	60	F	90425	NO	Abd. P	I.T.	R.LOBE	A	-	CONS.MGMT
96	BALU	35	M	43846	YES	Abd. P	I.T.	R.LOBE	A	-	CONS.MGMT
97	PAPPATHI	50	F	7019	NO	Abd. P	I.T.	R.LOBE	A	-	CONS.MGMT
98	DHANDAPANI	55	M	109671	YES	Abd. P	I.T.	R.LOBE	A	-	S.A
99	GANESAN	51	M	61432	YES	Abd. P	I.T., HEPATO	R.LOBE	A	-	PERCUT. CATH. DRAIN

ABBREVIATIONS

M	:	Male
F	:	Female
Abd.p	:	Abdomimal pain
Abd. D	:	Abdominal distension
F	:	Fever
Dy	:	Dysentery
I.T	:	Intercostal tenderness
Hepato	:	Hepatomegaly
S.A	:	Single aspiration
M.A	:	Multiple aspiration