A COMPREHENSIVE STUDY OF 80 CASES OF LIVER ABSCESS

Submitted to

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

For M.S. DEGREE EXAMINATION

BRANCH – I (GENERAL SURGERY)

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI, INDIA

2007-10

CERTIFICATE

This is to certify that **"A Comprehensive study of 80 cases of Liver Abscess"** is a bonafide work done by **Dr. P.S. GANESH BABU**, Post Graduate in department of General Surgery Kilpauk Medical College, Chennai 600 010. Under my guidance and supervision in fulfillment of regulations of the Tamil Nadu Dr.M.G.R. Medical University for award of M.S. degree Branch I, Part II (General Surgery) during the academic period from March 2007 to March 2010.

Dr. S. Udaya kumar, M.S.,
Prof. & H.O.D. of General Surgery,
Kilpauk Medical College, Chennai-10.

Dr. P.K. Baskaran,M.S.,
Prof. & Chief of General Surgery,
Kilpauk Medical College, Chennai-10.

Dr.Kanagasabai,M.D.,
The Dean
Kilpauk Medical College,
Chennai-10.

ACKNOWLEDGEMENT

I am extremely thankful to **Prof. Dr. S. Udayakumar, M.S.,** Professor and Head of the Department of General surgery Kilpauk Medical College for his guidance and help in conducting this study.

I am immensely grateful to my **Chief Prof. Dr. P.K. Baskaran, M.S.,** Department of General Surgery, Government Kilpauk Medical College, for his valuable guidance and encouragement in conducting this study.

I wish to express my sincere thanks to **Prof. Dr. Kanagasabai, M.D.,** Dean. Kilpauk Medical College, Chennai-10, for permitting to conduct this study in Government Kilpauk Medical College Hospital.

My sincere thanks to **Prof. Dr. A. Periyasamy, M.S., & Prof. Dr. T. Bhavani Shankar**, M.S..

I extend my special thanks to Assistant Professors of our unit **Dr.V.Vijayalakshmi, M.S., Dr. K. Shantha kumar M.S.,** and **Dr. T. Suresh Babu, M.S.,** for their valuable guidance in initiating this study.

I am thankful to all my post graduate colleagues for their help and encouragement.

Lastly, I am grateful to the numerous patients who participated and Co-operated with me during the Course of Dissertation.

CONTENTS

INTRODUCTION

INTORDUCTION

Descriptions of Liver abscesses date back to Hippocrates in approximately 4000 BC. But an understanding of their Etiology, Bacteriology, diagnosis and treatment is a recent event in Twentieth century and is still emerging.

Hepatic abscess often presents a pitfall in diagnosis and challenge to surgical diagnostic acumen. Early diagnosis and prompt initiation of treatment almost certainly leads to complete cure.

Of the two types of hepatic abscess, the Amoebic and Pyogenic, the former seems to be more prevalent in our country. Because studies of Hepatic abscess especially amoebic abscess have often originated from endemic areas, they offer little information on the application of modern diagnostic techniques. Diagnosis in this area mainly depends on the clinical presentation. But since the introduction of Ultra sonogram and CT Scan as a diagnostic device a more Accurate diagnosis can be made in every case.

In this study, about 600 cases of Hepatic abscesses admitted in Government Kilpauk Hospital and Royapettah Government Hospital in the year 2007-2009. Out of 600 cases, 80 cases were selected and discussed in this study.

AIM OF THE STUDY

AIMS AND OBJECTIVES

Clinically impressed with the great importance of establishing early diagnosis in patients with liver abscess and this study has been made to obtain better knowledge of the nature of the disease.

To determine the incidence of Hepatic abscess in our Hospital.

To determine the Age and Sex incidence.

To evaluate the various clinical parameters of both Uncomplicated and complicated Liver abscess and various modalities of treatment available.

To know about the disease Prevalence occurring as Abdominal Emergencies.

To emphasis the immense use of modern Investigation likes USG and CT Scan especially in the confirmation of diagnosis as well as in the treatment and follow up.

HISTORICAL FACTS

HISTORICAL FACTS

1845 -	GROS	-	Discovery of Amoeba
1875 -	LOSCH	-	Discovered Histolytica
1887 -	KOCH	-	Reported cases of amoebic dysentery with liver abscess and showed E.Histolytica trophozoites in hepatic capillaries.
1887 -	KARTULIS	-	Described presence of E. Histolytica in Liver abscess
1891 -	COUNCILMAN AND LAFLEUR	-	Coined the term Amoebic Abscess of Liver
1893 -	QUINCKE AND ROOS	-	Described the cystic form of E.Histolytica and its life cycle, differentiated pathogens from other non-pathogenic intestinal amoebae in man.
1904 -	KARULIS	-	Described amoeba in Brain abscess
1927 -	CRAUC	-	Defined the modern concept of clinical amoebiasis.

Amoebic liver abscess has been known since antiquity in India. In the Sanskrit Manuscript, BHRIGUSAMHITA (around 3000 B.C.) there is a mention of germs, which cause diarrhea with stools containing blood and mucus and that these germs can also give rise to a hard ball like mass in the abdomen, apparently a reference to an abscess.

ANATOMY AND FUNCTIONS OF THE LIVER

ANATOMY OF THE LIVER

The liver is the largest metabolic organ in the body. It is situated in the upper and right parts of an abdominal cavity, occupying almost the whole of the right hypochondrium, greater part of epigastrium, extending into left hypochondrium upto left lateral line. In male it weighs about 1400-1800g and 1200-1400g in female.

It has 5 surfaces. (Anterior, posterior, superior, inferior and the right). One prominent border (Inferior Border). On the posterior surface there lies the "Bare area of liver" which is contained within the reflection of superior and inferior coronary ligaments. Liver abscess is commonly situated in the upper part of the right lobe close to the Bare area. Liver abscess close to the bare area is painless because they are devoid of peritoneum.

Surface:

Diaphragmatic Surface:

a.Anterior

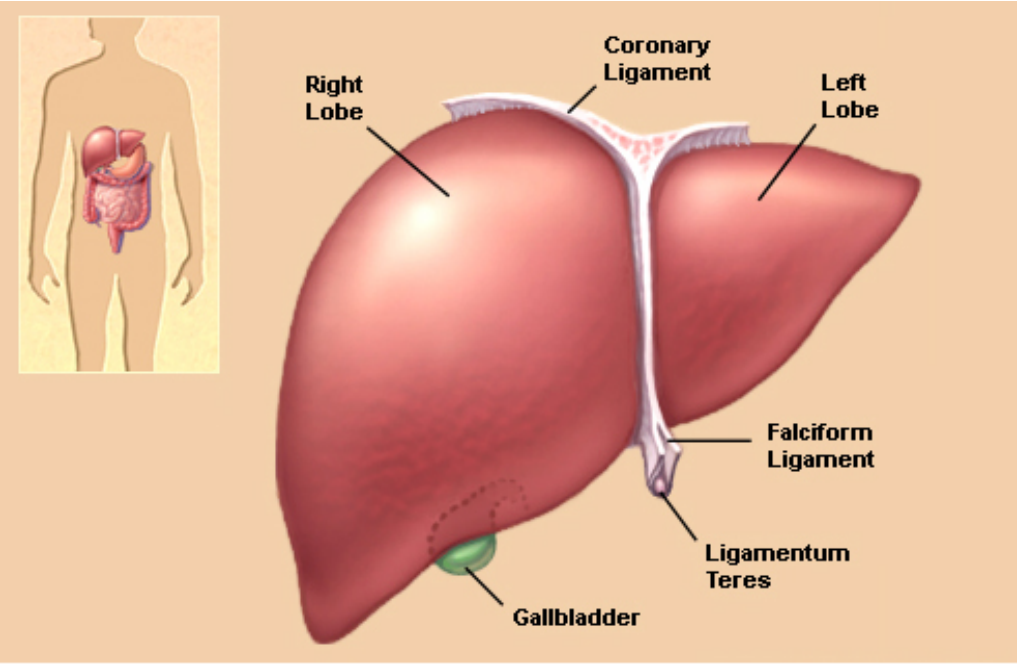
b.Superior

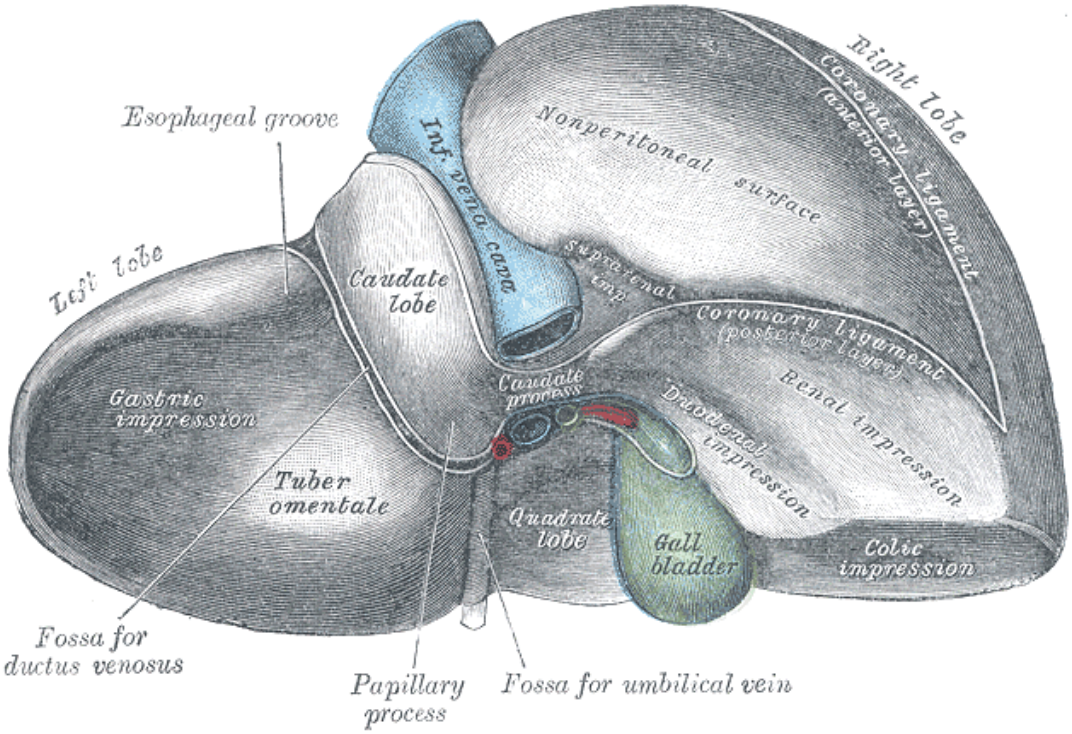
c.Posterior

Visceral Surface:

a. Postero Inferior

ANATOMY OF LIVER





Diaphragmatic Surface:

Smooth and doom shape, related to concavity of inferior surface of diaphragm, separated from diaphragm by sub-phrenic recesses which are separated by falciform ligament into right and left.

The convex or antero superior surface of liver is in relation with right lung and the pleura. This explains the erosion of hepatic abscess through the diaphragm into the pleural cavity and right lung.

PERITONEAL LIGAMENTS OF THE LIVER

a. Falciform ligament
b. Upper (Anterior) coronary ligament
c. Lower (Posterior) coronary ligament
d. Right Triangular ligament
e. Left triangular ligament
f. Lesser omentum

Falciform ligament

It is a sickle-shaped fold which stretches from the liver to the diaphragm and anterior abdominal wall. It consists of a right and left layer which are attached between the right and left lobes of the liver on its anterior and superior surfaces. The two layers are continuous with each other at the lower free border which lodges the round ligament of the liver.

Upper (anterior) Coronary Ligament

It is continuous with the right layer of the facliform ligament. It stretches between the posterosuperior aspect of right lobe of the layer and the diaphragm.

Lower (posterior) Coronary Ligament

It stretches between the postero inferior aspect of right lobe of the liver and the diaphragm.

Right Triangular Ligament

The upper and lower layers of the coronary ligament, when traced to the right are continuous with each other at the right triangular ligament which is a short triangular fold the connects the upper surface of the right lobe of liver with the diaphragm.

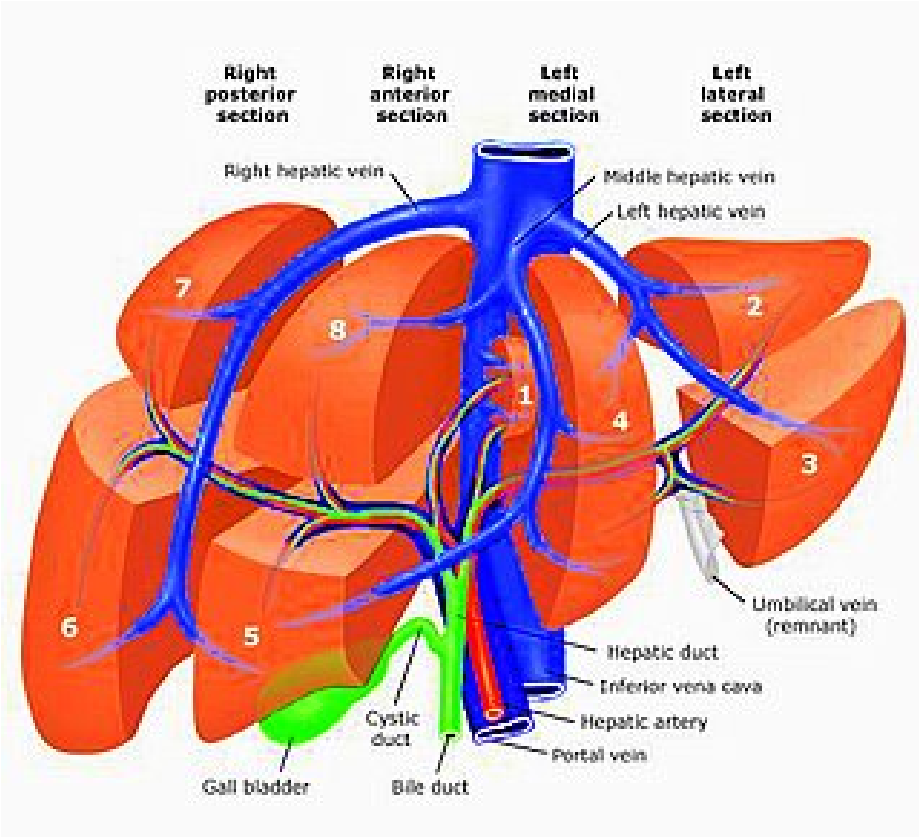
Left triangular ligament:

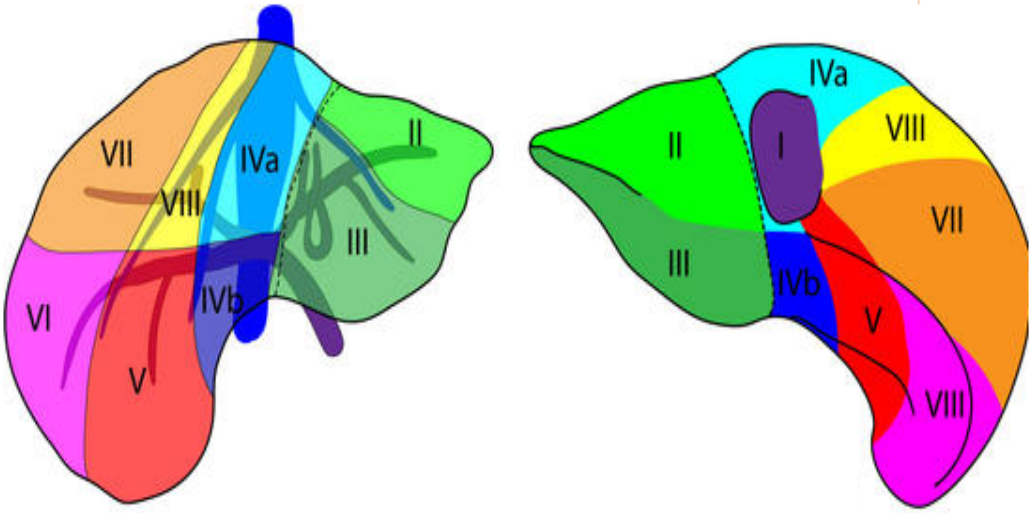
It is short triangular fold connecting the upper surface of the left-lobe of liver with the diaphragm. It consists of an anterior and posterior layer. The anterior layer is continuous with the left layer of the falciform ligament and the posterior layer is continuous with the anterior layer of the lesser omentum.

PORTA HEPATIS

Is a deep transverse fissure about 2 inch long, situated on the inferior surface of the right lobe. It admits portal vein, Hepatic artery and the Hepatic

SEGMENTAL ANATOMY OF LIVER





plexus of Nerves and lets out the right and left Hepatic ducts and few lymphatics. Relations within the Porta Hepatis are from behind far wards – the portal vein, hepatic artery, and Bile ducts.

MORPHOLOGICAL ANATOMY

Liver is divided into the right and left lobes by the falciform ligament anteriorly and superiorly, by the fissure for ligamentum teres inferiorly and by the fissure for ligamentum venosum posteriorly. Right lobe is much larger and forms 5/6 part. It has 2 additional lobes called the caudate and quadrate lobe.

The **caudate lobe**, lying between the inferior vena cava and the fissure for the ligamentum venosum, and the **quadrate lobe**, lying between the gallbladder fossa and the fissure for the ligamentum teres, were consequently considered to be part of the right lobe.

FUNCTIONAL ANATOMY OR SURGICAL ANATOMY OR SEGMENTAL ANATOMY

Functional anatomy was first initiated by cantle in 1898 and was enhanced by Counseller in 1929 and Couinaud in 1957. Couinaud's is the most exact and complete description of liver anatomy.

In essence the liver should be regarded as a paired organ (right and left livers) that are fused along a line extending from the middle of the gallbladder fossa anteriorly to the left edge of the suprahepatic inferior vena cava posteriorly.

Within the liver is this corresponds to a vertical plane (the main portal scissura or Cantlie's line) in which lies the middle hepatic vein. The right lobe liver receives the right portal vein, hepatic artery and bile duct and the left lobe liver the corresponding left portal vein, hepatic artery and bile duct.

THE RIGHT LOBE LIVER

The right liver is further subdivided into two sectors by the vertical right portal scissura in which lies the right hepatic vein.

The medial sector is composed of segments V (antero-inferior) and VIII (postero-superior) and the lateral sector of segment VI (antero-inferior) and VII (Postero-superior). Thus in the supine patient segments V and VI partially overlap segments VIII and VII, respectively. Each segment receives a portal pedicle and is drained by a separate bile duct.

THE LEFT LOBE LIVER

This is divided by the left portal scissura, in which lies the left hepatic vein, into a medial sector that forms segment IV and a lateral sector that is further divided into an anterior segment III and a posterior segment II.

Caudate (dorsal, Spigel) lobe

Although this is customarily labeled as segment I, it is really a separate 'liver' because it has its own hepatic veins and bile ducts although it receives portal and arterial branches from both right and left sides.

Because of this segmental liver anatomy, it is possible to resect a single or several segments even in the liver that has been distorted by chronic disease.

HEPATIC STRUCTURE

The greater part of the liver is invested with peritoneum, which covers a thin capsule of connective tissue (Glisson's capsule). The bulk of the cells within the liver constituting about 80% are the hepatocyctes or parenchymal cells. Hepatocytes carry out a multitude of metabolic activities.

Conventional morphology considers that the liver is composed of pyramidal lobules based on a central vein surrounded on the periphery by portal trunks with terminal radicles of bile duct, portal vein and hepatic artery. The two vascular systems of central vein and portal tract lie on planes at right angles to one another and never interdigitate. Thus the sinusoids are arranged perpendicular to the planes of the central veins and portal blood passes to the central vein along a pressure gradient. The walls of the sinusoids are composed of endothelial and phagocytic cells termed Kupffer cells. Between the hepatocytes and Kupffer cells is the space of Disse. Bile canaliculi are shown to be channels or grooves in the hepatocyte surface, lined by microvilli. The network of canaliculi drains the liver lobules into the terminal bile ducts.

According to Rappaport, there are three zones of liver parenchyma. Zone 1 and Zone 2 may form nidus of surviving cells which then regenerate in nodular

form. Zone 1 adjacent to the portal triad is the best vascularized and least susceptible to injury. Zone 3 is adjacent to the central vein and most susceptible to injury.

BLOOD SUPPLY

The dual afferent blood supply consists of hepatic artery and portal vein. Liver receives 20% of its blood supply from hepatic artery and 80% from the portal vein. Before entering the liver the hepatic Artery and portal vein divide into right and left branches. Within the liver they redivide to form the segmental and then to interlobular vessels which run in the portal canals. Further ramification of interlobular branches open into the hepatic sinusoids. Thus hepatic arterial blood mixes with the portal venous blood in the sinusoids.

VENOUS DRAINAGE

The hepatic venous drainage beings as a central vein of a liver lobule. The central vein receives sinusoids from all sides and unite with the central vein of other lobules to form the sub lobular veins which in turn fuse to form collecting veins which forms 3 major hepatic veins.

LYMPHATIC DRAINAGE

The superficial lymphatics drain into caval, hepatic, paracardial and celiac lymph nodes. Deep lymphatic end in the nodes around the end of IVC and party into hepatic nodes.

NERVE SUPPLY

Nerves of liver are derived from Hepatic Nerve plexus which are largest derivative of celiac plexus. Hepatic plexus accompanies branches of Hepatic artery and portal vein to the liver. It consists of sympathetic fibres from celiac plexus and para sympathetic fibres from anterior and posterior vagal trunks.

DEVELOPMENT

The liver arises from Hepatic diverticulum from most caudal part of the foregut. The Hepatic diverticulum extends into septum transversum and expands the ventral mesentry. It divides into large cranial parts which give rise to interlacing cords of liver cells and intra epithelial lining of intra Hepatic portion of biliary apparatus.

FUNCTIONS OF THE LIVER

1. The formation of bile and the metabolism of bilirubin and of bile salts.
2. The synthesis of albumin, fibrinogen and prothrombin.
3. Storage and metabolism of carbohydrates, including the conversion of monosaccharides (e.g. dextrose) into glycogen, and vice versa.
4. Formation of phospholipids and cholesterol, synthesis of fatty acids from carbohydrate.
5. Deamination of amino acids with formation of urea. Removal of ammonia from portal blood.
6. Heat production

7. Reticuloendothelial activities
8. Storage of Vitamin B_{12} and Vitamin A
9. Iron and Copper Storage
10. Destruction of bacteria
11. Detoxication of drugs and hormones.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

INCIDENCE

Entamoeba histolytica Infection affects an estimated 10% of the world's population. The great majority of such Infections occurring in people living in Indian sub-continent, sub Saharan Africa, and parts of Central and South America. In these endemic areas approximately 50% of the population is infected with 90% or more being Asymptomatic cyst Passers.

Amoebic Liver abscess occurs in less than 10% of individuals infected with these organisms. Amoebic Liver abscess are 3-5 times more frequent than Pyogenic Liver abscess. Average age of the patients is between 28-48 years, with striking male predominance (7:1). Particularly severe Invasive disease occurs in patients with compromised cellular immunity in young infants, in the malnourished, in pregnant women and in patients receiving corticosteroid. On the global scale, Amoebiasis is the third most common Parasitic cause of Death after malaria and schistosomiasis.

Incidence of Pyogenic Liver abscess being estimated at 8-16 cases /1,00,000 admissions. Fifty years ago the majority of patients were under the age of 40 and Appendicitis was the leading cause of the disease. Today the average age being 43-60. This change corresponds to the finding that appendicitis has been replaced by Biliary tract disease as the most common underlying aetiology.

LIFE CYCLE OF E-HISTOLYTICA

Amoebiasis is defined as the condition of Harboring of entamoeba Histolytica in Humans with or without clinical Manifestation (WHO Tehran in 1968)

E.Histolytica exists in two forms

1. Trophozoite

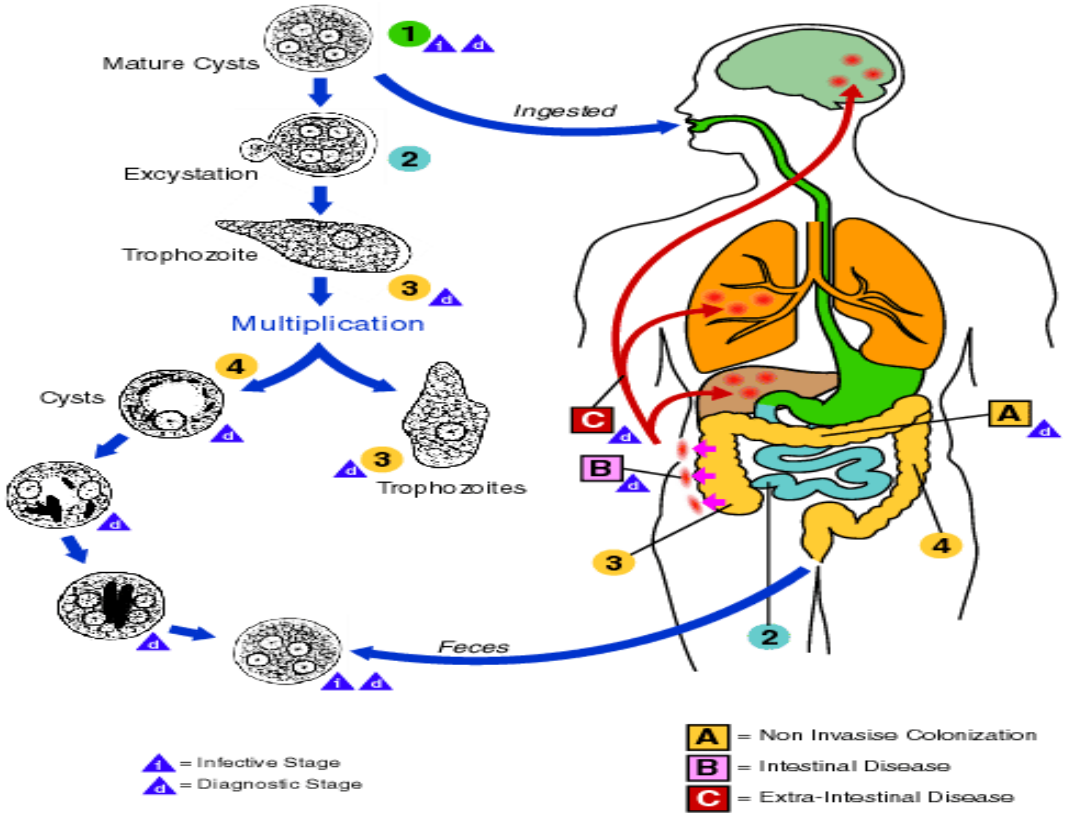
2. Cyst with a Transitory stage of Precystic Form.

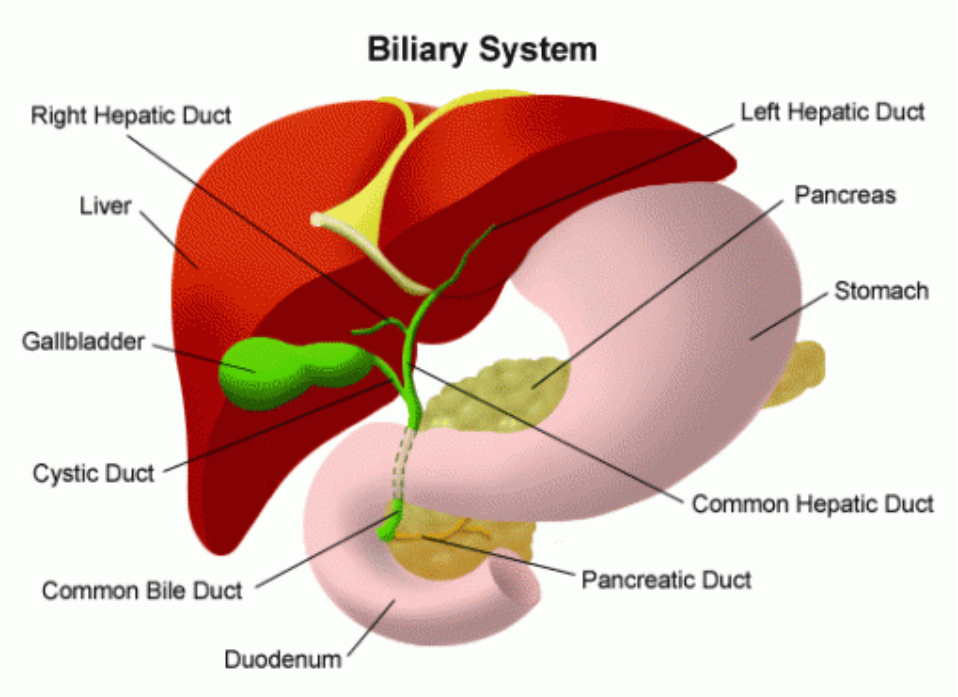
E.Histolytica Passes its life cycle only in one Host the man.

The mature quadrinucleate cysts are the infective forms of the parasite. When these cysts are swallowed along with contaminated food and drinking water by a susceptible person, they are capable of further development inside his gut. The fully developed cysts thus gaining entrance in to the alimentary canal, pass unaltered through the stomach. The “Excystation” occurs when the cyst reaches the caecum or lower part of the ileum. Each cyst Liberates a single Amoeba with four Nuclei, a tetranucleate Amoeba which eventually forms eight Amoebulae (Metacystic trophozoites) by division of nuclei with successive fission of cytoplasm. The young Amoebulae being actively motile, invade the tissues and ultimately lodge in the submucous tissue of the large gut their normal habitat. Here they grow and multiply by Binary fission.

It is to be noted that the Trophozoite phase of the parasite is responsible for producing the characteristic lesion of amoebiasis. The Trophozoite of E.Histolytica enter into the deeper layers and many find their way into the portal

LIFE CYCLE OF ENTAMOEBA HISTOLYTICA



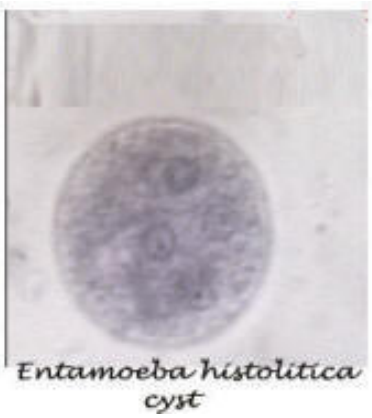


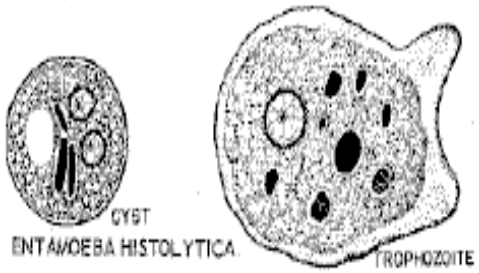
vein, to be carried away to the liver where their further progress may be arrested. In the liver the Trophic forms may for a time grow and multiply but encystations does not occur.

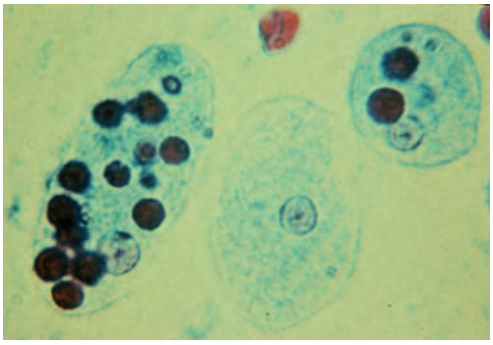
Hence such an invasion is always to be overlooked as an accident on the part of the Parasite because so far as its biological aspect is concerned it has reached a dead end. The parasites that remain in the intestinal wall may cause an attack of acute dysentery (amoebic colitis). A certain number of these Trophozoites are discharged into the lumen of the Bowel and are transformed into small precystic forms from which the cysts develop.

The mature quadrinucleate cysts are the most resistant and infective forms of the parasite. But the cysts produced in an infected individual are unable to develop in the host in which they are produced. Transfer to another susceptible host enables them to grow and continue their life cycle.

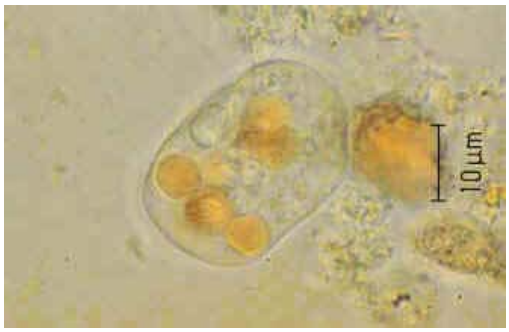
There are 2 types of E.histolytica namely pathogenic and non pathogenic. It is now firmly believed that pathogenic and non-pathogenic entamoeba isolates are distinct species. Pathogenic is called E.Histolytica, Non pathogenic as E.Dispar. Pathogenic behavior of pathogenic and non pathogenic entamoeba depends on the quantitative and qualitative difference between molecules (genes/proteins) identifiable in both forms.







E. histolytica **trophozoite**



TROPHOZOITE OF ENTAMOEBA HISTOLYTICA WITH INGESTED RBCS

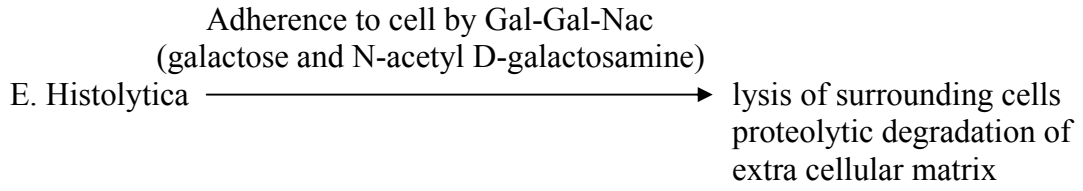
AEITOLOGY AND PATHOGENESIS – AMOEBIC LIVER ABSCESS (Syn TOPICAL OR DYSENTERIC ABSCESS)

The transmission of amoebiasis is clearly related to lack of sanitation and low socio-economic status rather than to climate. Common modes of transmission are by food contaminated with cysts or contaminated water through those engaged in preparation and handling of food. The commonest extra intestinal manifestation of amoebic infection occurs when organisms escape the colon and reach the liver via the portal vein.

There is another view that retrograde lymphatic spread of Amoebiasis can occur from the colon to the liver and from the liver to the lungs. Lymphangiographic study in case of carcinoma stomach have shown that the lymphatics from the stomach, colon and liver join the thoracic duct – an evidence for the retrograde lymphatic spread of amoebiasis. It was favored by the demonstration of multiple amoebae in the small interlobular branches of the portal vein. After reaching the liver in large number, the trophozoites set up a thrombus formation which in turn gives raise to an infarct, the wall of the vessel is destroyed and amoebae feed on the products of cytolysis. This view was supported by the demonstration of thrombosed radicals of the portal vein in the walls of the abscess and amoebae were found entangled in the thrombus.

E-histolytica produces enzymes which cause hydrolytic dissolution of liver tissue. Various enzymes are produced like hyaluronidase, Glutaminase, amylase, maltase, esterase, succinly dehydrogenase and gelatinase. Alcholism, nutrition

particularly poor in protein and poor immune status predispose to the development of amoebic liver abscess.



(Tropical Hepatogastroenterology, Chapter 38 Intestinal and extraintestinal amebiasis-MP Sharma & Vineet Ahuja)

PATHOGENIC CLASSIFICATION OF AMOEBIC LIVER ABSCESS

(Gastro Enterology Today Vol-I No.3)

Acute	Chronic
a. Benign	a. Benign
b. Aggressive	b. Acclerated
1. Illness less than 10 days	1. Low Grade symptom for a month or more
2. Fever & Tender Hepatomegaly present	2. Fever uncommon, mildly Tender hepatomegaly
3. Low Serological titres Leukocytosis, increase transminase	3. High Serological titres, severe anemia ↑ alkaline phosphatase, normal SGOT

MACROSCOPIC PATHOLOGY

About 83% of liver abscess are located in the right posterosuperior surface of liver. The propensity for this site reflects the fact that venous return from the right side of colon (Amoebic infection having a particular impact on the caecum and Right colon) into the portal vein is predominantly delivered to the right lobe of

liver. To the naked eye, the appearance of the abscess area is reddish brown in color with a semifluid or grumous consistency. The wall of the abscess cavity is ragged and shaggy in appearance and is formed by the necrotic liver tissue which gradually merges into the healthy zones with an intervening zone of hyperaemia. In an old abscess the wall is smooth and is formed by dense connective tissues. Multiple small abscesses in which the whole organ becomes riddled with scattered foci of necrosis are probably more common. The liver enlargement is explained on the basis of oedema and congestion **(lamot and pooler 1958).**

MICROSCOPY

If a section is made through the margin of a liver abscess, three zones can be differentiated from the center to the periphery.

1. A central zone of cytolysed granular material with no amoebae.
2. An intermediate zone consisting of degenerated liver cells, a few leucocytes, connective tissue cells, red blood cells and an occasional trophozoite of E.hostolytica.
3. A peripheral zone consisting of congested capillaries with varying degrees of necrosis of liver cells. The amoebae can be seen to be multiplying in this area and invading and adjoining healthy liver tissue.

In long standing cases, the third zone may consist of actively proliferating connectives tissue cells, lymphocytes and monocytes, walling off the abscess cavity.

PUS OF LIVER ABCESS

The 'pus' is not of suppuration but is a mixture of sloughed liver tissue and blood. It is chocolate brown in color and thick in consistency (so called 'Anchovy-sauce' pus). The smell is rarely offensive. The 'pus' is bacteriologically sterile. Microscopy reveals degenerated liver cells, a few red blood cells and occasional leucocytes. The trophozoites of E.histolytic are not generally found in freshly aspirated pus but appear in the escaping 'pus' four or five days after the initial evacuation.

Microscopic examination of the wet mount of pus from the liver or lung amoebic abscesses is frequently carried out for the demonstration and identification of amoebic trophozites.

Sensitivity of the method is however very low. Amoebic trophozoites can be demonstrated only less than 15% cases of amoebic liver abscess. **(Text book medical parasitology 2nd Edition by Subash Chandra Parija)**

INTESTINAL LESIONS IN AMOEBIC LIVER ABSCESS

In all cases of hepatic amoebiasis, as also in other metastatic amoebiasis, the primary lesion is in the large gut. The changes in the intestine observed at autopsy may be any one of the following.

i) Small superficial ulcers, with thickening of the colon.

ii) A single latent ulcer, located most commonly in the caecum.

iii) Pigmented or non-pigmented scars in the large intestine, representing the sites of previous ulcers.

iv) No change in the large gut.

v) Extensive ulcers scattered throughout the large intestine are quite uncommon.

vi) E-Histolytica can produce diffuse inflammation and ulcertation of colon which may be indistinguishable endoscopically from idiopathic ulcerative colitis. Further more reappearance of colonic symptoms in patients with chronic ulcerative colitis has at times found to be due to infection with E.Histolytica.

Clinical syndromes associated with E. histolytica infection.

Intestinal amoebiasis:

- Asymptomatic cyst passers
- Acute amoebic colitis
- Mucosal disease
- Transmural disease.
- Ulcerative postdysentric colitis,
- Appendicitis
- Amoeboma,
- Amoebic stricture

Extraintestinal amoebiasis:

- Amoebic liver abscess

- Perforation and peritonitis
- Pleuropulmonary amoebiasis
- Amoebic pericarditis
- Cutaneous amoebiasis

PYOGENIC LIVER ABSCESS

ETIOLOGY – BACTERIOLOGY – PATHOGENESIS

In the liver the kupffer cell functions as a primary Barrier and filter for the clearance of micro organisms from arterial, venous, Biliary and local sources. Pyogenic liver abscess are believed to occur when their normal hepatic clearance fails or is overwhelmed. Distal infectious sites may seed the liver with pathogenic Bacteria via portal vein or the Hepatic Artery.

ORGANISMS

Gram Negative Aerobs (50-70%)

E.Coil (35-45%), klebsiella, Proteus, enterobacter.

Gram Positive Aerobs (30%)

Streptococcal Sp, enterococcus Faecalis.

Anaerobes (40-50%)

Bacteroides Sp, Fusobacterium, Pepto Streptococcus.

Fungal

Sterile

ETIOLOGY	%	SOURCE	DISTRIBUTION	1° MICRO ORGANISMS
Biliary System	40	Cholangitis Biliary Obstruction	Both Lobes Multiple	Single Species Gram-aerobes and anaerobes – E-Coli
Portal Circulation	20	Intra abdominal Infection Liver metastasis	Right Lobe>Left Multiple or single Area of Metastasis	E.Faecalis, E.Coli, B. Fragilis B. Fragilis
Arterial Circulation	12	Bacteremia, Systemic Infection	Both lobes-Multiple	S. Aureus, S.Pyogenes
Trauma	4	Direct Exposure, Necrosis	Area of injury	S. Aureus, S. Pyogenes
Direct Extension	6	Cholecystitis Perforated ulcer	Adjacent area	E.Coli
Cryptogenic		Unknown	Right lobe>left	B. Fragilis

CLINICAL PRESENTATION

The clinical feature often reflects the site, size and the number of abscesses as well as degree of involvement of adjacent tissues. The onset of symptoms may be acute or chronic. The clinical features of amoebic abscess are similar to Pyogenic abscess.

However acute presentations are more common with amoebic abscess. In amoebic abscess pain in Right Hypochondrium and Right Lower Chest are the commonest symptom (90%) followed by fever. In Pyogenic abscess, fever is the most common symptom (80%). Hepatomegaly and Right upper Quadrant tenderness are the only consistent Physical findings.

Triad of fever, pain in Right Hypochondrium and Tender Hepatomegaly is strongly suggestive of liver abscess.

Co-morbid diseases associated with Pyogenic liver abscess.

Children	**Adults**
Chronic granulomatous disease	Diabetes Mellitus
Complement deficiencies	Cirrhosis
Leukemia	Chronic Pancreatitis
Malignancy	Peptic ulcer disease
Sickle cell anaemia	Inflammatory Bowel disease
Polycystic liver disease	Jaundice
Congenital hepatic fibrosis	Tuberculosis
Post transplant liver failure	Malignancy
Necrotizing enterocolitis	Leukemia and Lymphoma
Chemotherapy and steroid therapy	Chemotherapy and steroid therapy
Acquired Immuno Deficiency Syndrome	Acquired Immuno Deficiency Syndrome

SYMPTOMS	SIGNS
Pain	Hepatomegaly
Fever	Right Upper Quadrant Tenderness
Nausea and Vomiting	Guarding and rigidity
Weigh loss	Ascites
Malaise	Jaundice
Diarrhea	Pleural effusion
Pruritus	Pleural rub
Cough	

AMOEBIC	PYOGENIC
Age<50 years	>50 years
Male : Female 10:1	Male = Female
Recent Travel to endemic area	Malignancy
Pulmonary dysfunction	High fevers
Abdominal Pain	Pruritus
Diarrhea	Jaundice
Abdominal Tenderness	Septic shock

It has been noted infrequently at the extremes of age and is seven to nine times more common in males. Amoebic Liver Abscess (ALA) may present as an acute process or as a chronic indolent disease. ALA usually occurs in the right lobe of the liver and is solitary (30% - 70%). Unusual presentations include multiple abscesses, left lobe abscesses, abscesses presenting as compressive lesion, and abscesses rupturing into viscera.

Multiple liver abscesses:

Fifteen per cent of patients may have multiple abscesses. They present with fever, toxaemia, deep jaundice, and encephalopathy. Toxaemia is suggestive of an

added bacterial infection leading to a more severe disease. E.coli and Klebseilla are the commonly cultured organisms. These patients present with a clinical picture indistinguishable from hepatic encephalopathy due to acute hepatocellular failure. Hepatic encephalopathy in ALA patients possibly results from combination of right hepatic vein occlusion, pylophlebitis, and occlusion of several portal vein radicles.

Left lobe abscess :

Thirty-five per cent of patients present with a left lobe abscess. Half of these have associated lesions in the right lobe while the remaining have solitary left lobe abscess. These patients have longer duration of symptoms (3-4 weeks) and fever is less commonly observed as compared to right lobe abscesses. It may present as a large epigastric mass with minimal movement with respiration. Often, to the clinician's despair, it has been confused with pseudocyst of pancreas. These patients also have weight loss with poor hepatic localisation of symptoms. Complications like peritonitis and toxaemia are significantly more common in left lobe abscess. Needle aspiration may be more rewarding in combination with anti-amoebic drugs.

Compression lesions :

A posteriorly located ALA in the right lobe may present as inferior vena cava obstruction or hepatic outflow obstruction. This is suggested by bilateral pedal oedema, ascites, visible veins on anterior and posterior abdominal wall,

along with clinical, radiological, and serological features of ALA. These features disappear after aspiration of the abscess. Leakage of the abscess may occur into the pleural cavity, with empyema thoracis. Intraabdominal extension following perforation into the peritoneal cavity is usually associated with shock and generalised peritonitis and may occur in upto 7% of cases. Rupture into the colon and biliary tree has also been reported. **(Journal, Indian Academy of Clinical Medicine _ Vol. 4)**

COMPLICATIONS OF LIVER ABSCESS

About 40% of patients with Pyogenic liver abscess and 10% of amoebic liver abscess patients develop complications. Generalized sepsis is the most common systemic complication in Pyogenic abscess. Rupture into peritoneum and thorax are the most common complications in amoebic abscess. Posteriorly located Amoebic Liver Abscess in Right Lobe can present as IVC obstruction or Hepatic out flow obstruction (Gastro Enterology Today April, June 1997).

Severe Amoebic Liver Abscess (Chau. Sk., Chang, Chiencs, Sheen IS)

Severe Amoebic Liver Abscess was defined as the Rupture of an abscess that was resistant to 72 hours of medical treatment, complicated by secondary bacterial infection or Diabetes Mellitus.

PLEUROPULMONARY COMPLICATION

It is the most frequent site affected when liver abscess ruptures. It is the third most common manifestation of amoebiasis after amoebic colitis and amoebic liver abscess. The reported incidence of pleuropulmoary amoebiasis varied from 14 to 30 percent. Clinical manifestations include chest symptoms with expectoration of anchovy – sauce sputum. Pleural rub, signs of pleural effusion and lung abscess may also be present. Typically such lesions originate from right lobe.

PERITONEAL COMPLICATION

Amoebic peritonitis is the second most common complication of Amoebic liver abscess and it is the commonest form of rupture into a serous cavity. Amoebic peritonitis results more often due to rupture of Right Lobe Abscess. Extension into Peritoneal cavity may form acute generalized peritonitis, chronic generalized peritonitis or a localized abscess. In sudden rupture of amoebic liver abscess the clinical picture mimics perforation of a hollow viscus. Mortality may approach a great as 20%.

PERICARDIAL COMPLICATION

Pericardial complications are relatively rare and are often associated with left lobe abscess. The reported incidence of this complication varies from 0.6 to 5 per cent. Pericardial extension may cause suppurative pericarditis due to sudden rupture of a abscess in to the pericardial cavity. Non-purulent type may present as

pericardial effusion. It can also cause constrictive amoebic pericarditis or amoebic hydro pericardium.

UNCOMMON COMPLICATIONS

Erosion into the intestinal tract, cutaneous fistulation, secondary infection, obstructive jaundice, subphrenic abscess, hemobilia and vascular complications are other rare complications. Siddiqui MN, Rizvi SB, Ahmed M, in their case report, reported a case of Amoebic Liver Abscess complicated by Hepato duodenal fistula (a finding of air in the liver abscess – on Ultra sonogram – confirmed by Gastograffin Swallow).

DIAGONSTIC STUDIES

Laboratory Investigations: Even though most of the laboratory investigations are non specific, they are helpful to some extent in the diagnosis, to assess the involvement of liver, response to treatment and also as prognostic factor.

BLOOD

- Leucocytosis varying from 15,000-25,000 with a left shift present in 50-60%.
- Anemia is also encounted in liver abscess. It is related to duration and size of Abscess.
- Erythrocyte sedimentation rate is also usually raised.

LIVER FUNCTION TESTS

Abnormal liver function test exist more commonly in Pyogenic liver abscess than Amoebic Liver Abscess.

SERUM BILIRUBIN

- Serum bilirubin is elevated in 10-24% of patients.

ENZYMES

- Serum Alkaline phosphatase is raised in about 50-60% of patients.
- Elevations of SGOT or SGPT and Gamma – Glutamyl Transpeptidase occur in approximately 40-50% of patients.

SERIUM PROTEINS

- Hypoalbuminemia is quite common with increase in Gamma – Globulin
- Hypoalbuminemia is considered as a poor prognostic marker especially if it is < 2gm/dl

PROTHROMBIN LEVEL

- There is also prolonged plasma prothrombin level.

PARASITOLOGICAL EXAMINATION

STOOL EXAMINATION

Cyst & Trophozoites of Entamoeba Histolytica are present in the stools in <15% of Amoebic liver abscess patients –verlenden et al. This gives information regarding the persistence of intestinal infection.

E. histolytica cysts may remain viable for some time in unpreserved stools while trophozoites are labile and remain in stool for about 30 minutes.

ASPIRATE

Aspirate is odourless gram stain and culture are negative in amoebic liver abscess which is sterile and unless it is secondarily infected, wall scrapings of the abscess cavity may contain viable tropozoites can increase the sensitivity of Aspirate. But we can culture the organisms in case of Pyogenic abscess.

SEROLOGY

Serum antibodies to amoebae develop only during E. histolytica infection and not during E. dispar infection. The absence of serum antibodies to E. histolytica after 1 week of symptoms is strong evidence against the diagnosis of invasive amoebiasis of the colon or liver.

Serum antibodies to amoebae are detected in 85-95% of all patients who present with invasive amoebiasis or liver abscess. Purified native and recombinant parasitic antigens have been utilised in serological studies with good results.

INDIRECT HAEMAGGLUTINATION TEST (IHA)

* 95% Sensitive – pahuchonma, Patterson et al.

Positive if dilutions exceed 1:128

Highly specific for invasive amoebic disease

Remains positive for 20 years.

GEL DIFFUSION PRECIPITIN TEST (GDP)

* 90-95% Sensitive. Detects non-invasive amoebic colitis as well. So it is sensitive and non specific test. Remains positive for 6 months.

OTHER TESTS USED RARELY

1. Enzyme Linked ImmunoSorbent Assay (ELISA) Amoebic Antibodies in Titres>1:400 is considered as evidence of Amoebic Liver Abscess.
2. Latex agglutination (LA) Test
3. Immuno Electrophoresis (IE)
4. Counter Current Electrophoresis (CEP)
5. Immuno – Fluorescent Antibody (IF) Test
6. Radio Immuno Assay (RIA) Test
7. Compliment Fixation Test (CF)
8. Intradermal Test
9. Immobilization Test
10. Specific DNA probes:

* Specific DNA probes for pathogenic strain is 145 Base pair multi copy gene. For non pathogenic strain is a 133 Base Pair multicopy – DNA fragment.

Distinct epitopes present on the 170kDa heavy subunit of the galactose inhibitable adherence lectin, an important surface adhesin, and a highly conserved antigen are all found in E.histolytica but not in E. dispar.

NON – INVASIVE METHODS

Radiology

* **X RAY –CHEST & ABDOMEN**

Radiological changes are found in 60-75% of cases **(Wilmot 1962, Ramachandran et al 1971).** There may be no changes when abscess are small or situated in the lower part of liver.

Radiological signs

1. Elevation of hemi diaphragm
2. Pleural effusion
3. Pulmonary infiltrates
4. Basal Atelectasis
5. Soft tissue mass may be see in Epigastrium causing Displacement of stomach or colon especially in case of left lobe abscess.

Non Specific Findings

a. Right upper quadrant gas
b. Air – fluid levels the abscess
c. Ileus

ULTROSONOGRAPHY (USG)

The advent of ultrasonography has opened a new horizon in the diagnosis of liver abscess. It is non – invasive, less expensive, free from radiation hazards, easy and rapid diagnostic procedure with high specificity. Ultrasonogram was done in all cases using 3.5 MHZ sector transducer.

* Advantage – usefulness

1. Sensitivity of 90-95%.
2. Detect lesions as small as 2 cm diameter
3. For detecting the site & number of abscess
4. For guiding the aspiration of abscess
5. For follow up of patients
6. To differentiate from malignant conditions
7. To detect associated Intra abdominal pathology in case of Pyogenic liver abscess

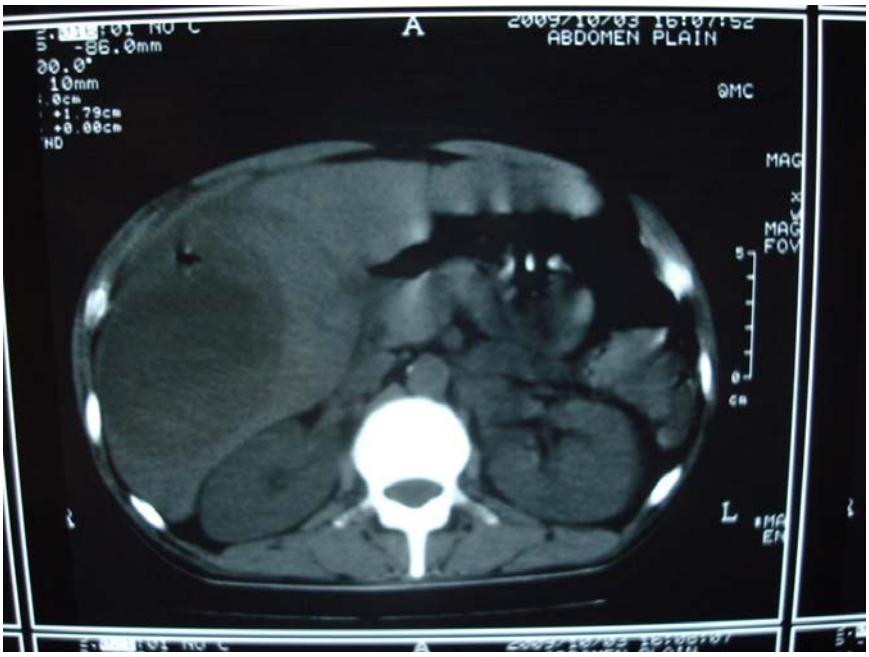
Boultbee and Ralls demonstrated the appearance of liver abscess in sonogram. McCarthy and Doust et al also reported the appearance.

*** Ultrasonographic findings in liver abscess**

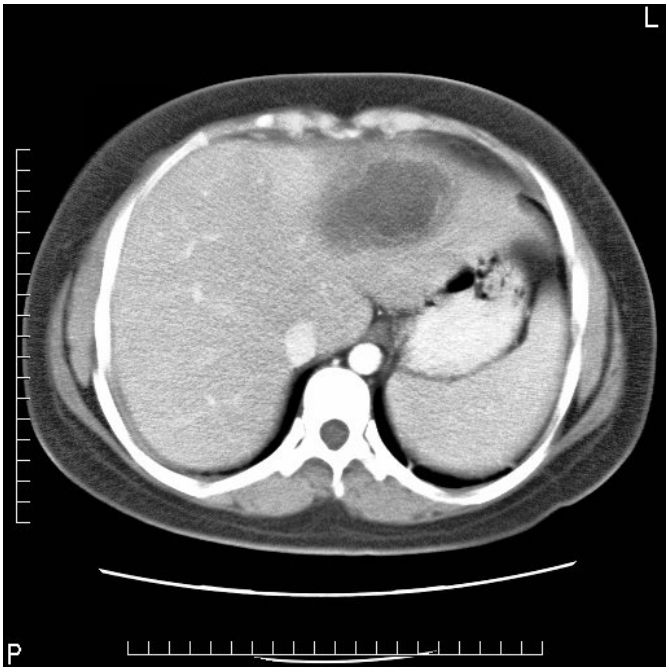
1. Rounded lesion abutting the liver capsule without significant rim echoes interpreted as an abscess wall. The contents of the cavity are usually hypoechoic and nonhomogeneous.
2. Cyst homogenicity : Uniform distribution of weak echoes or band like internal echoing, structures representing the walls of loculi or debris.
3. Smooth poorly defined wall. In contrast cyst have well defined walls
4. Distal sonic enhancement
5. A location contiguous with liver capsule.

* Pyogenic abscess hypoechoic mass with thick walls with fluid debris level posteriorly Abscess with bright echogenic foci may contain air or micro bubbles. Necrotic liver tumors also have fluid – filled center and may be confused with abscess but they generally have thicker walls. Nature of Internal echoes correlate to the thickness of the pus. Coarse internal echoes indicate the presence of thick pus and fine internal echoes indicated the presence of relatively fluid pus. Fluid pus noted to be present in early stage of the disease with a short history, while thick pus was present in a patient who has disease for long duration. Fungal abscess will have hypoechoic with echogenic center that are multiple & irregular.

CT OF LIVER ABSCESS IN THE LEFT LOBE



CT OF LIVER ABSCESS IN THE RIGHT LOBE



DRAWBACKS

a. Inaccurate in detecting multiple, small abscess near the dome of diaphragm.
b. Difficult to differentiate liver abscess and tumors with central necrosis.
c. Difficult to detect liver abscess in fatty livers.

- After successful therapy complete resolution of ultrasound abnormality may be expected in a period of 6 weeks to 23 months (average 7 months)

C.T. SCAN

Abdominal CT scan is probably more sensitive than ultrasound and is helpful in differentiating amebic from pyogenic abscess, with rim enhancement noted in the latter. CT scan can also be helpful in identifying simple cysts and necrotic tumors.

Accuracy 95-100%

Indications

1. Procedure of choice for initial assessment of a suspected pyogenic liver abscess.
2. Positive Amoebic serological Test exist but Hepatic Sonogram negative.
3. Detects abscess as small as 0.5 cm. More precise anatomical definition than ultrasound.
4. Better for small abscess near the dome of diaphragm and abscess in fatty livers.

5. For visualizing intra abdominal pathology in case of pyogenic liver abscess such as appendicitis, pancreatic mass, Diverticulitis, Colonic Cancer and Intra peritoneal abscess.

MRI (MAGNETIC RESONANCE IMAGING)

- Detects lesions as small as 0.3 cm Superior in defining hepatic venous anatomy Useful in patients requiring liver resection to treat the abscess

On M R I, Amoebic abscess often have multiple rims of variable signal intensity. After treatment, the abscess cavity demonstrates concentric rings, Corresponding to an inner area of inflamed tissue with band of collagen and outer margin of inflamed liver tissue.

GALLIUM SCANNING OR TECHNETIUM

Technetium 99m liver scans can be helpful in differentiating pyogenic from amebic abscesses because the latter typically do no contain leukocytes and therefore do not light up on those scans.

HEPATIC SCINTIGRAPHY

Liver scanning is a valuable aid in the diagnosis and location of liver abscess with sensitivity of 80-97%. Commonly used scans are 99 m Technetium I 131 Rose Bengal or BSP, Indium – 113 scans. They show abscess as non specific hepatic filling defect.

LIMITATIONS

1. Unable to detect lesions smaller than 2 cm
2. Superficial abscess are frequently missed
3. Unable to differentiate between abscess, cyst or tumor
4. Non specific to the etiology of filling defect

DIAGNOSTIC LAPARASCOPY

Laparoscopy offers much more advantage in that you can see an abscess with your naked eye. Although it is an invasive investigation, it is one of the best examinations for visualizing an abscess. Not only that but the differential diagnosis of an abscess from conditions like malignancy can be made with elegance. One can also take an open biopsy.

It is worth knowing that superior surface liver abscesses can be better visualized with laparoscopy than at laparotomy. The reason is that at laparoscopy the air insufflation exposes the whole superior surface.

INVASIVE

Cavitogram

Catiogram were performed by injecting a contrast material viz CONRAY 280 (50 ml) into the abscess cavity through the drainage tube (Malecot's or foley's catheter). Most of these cases reveal a collapse of the abscess cavity with resorption.

Treatment

Anti amoebic agents

Amoebic abscess

With the introduction of metronidazole in 1960s surgical drainage of most amoebic liver abscess has become unnecessary. Imidazole antibiotics including metronidazole, tinidazole and niridazole will eradicate both intestinal and hepatic amoebic organisms.

Amoebicidal drugs may be classified into 3 groups : luminal, tissue, and mixed amoebicides. Duodohydroxyquin, diloxanide furoate, and paromomycin are luminal amoebicides. The amoebicides effective in tissues are emetine and dehydroemetine, which act in the liver, intestinal wall; along with chloroquine which acts only in the liver. Emetine and dehydroemetine because of their cardiotoxicity are currently not used. Amoebicides effective in both tissue and the intestinal lumen include nitroimidazole derivatives-metronidazole, tinidazole, and ornidazole. They are the drugs of choice in invasive amoebiasis. Oral or intravenous metronidazole or tinidazole also leads to rapid clinical improvement of amoebic liver abscess. This drug should be followed by a luminally active drugs.

Pharmacotherapy for E. histolytica infection in adults.

Intraluminal infection	Diloxanide furoate 500 mg tid X 20 days Paromomycin 30 mg/kg/day X 10 days (in 3 divided doses) Iodoquinol 650 mg tid X 20 days
Invasive colitis	Metronidazole 800 mg tid X 5 days Tinidazole 1 gm bd X 3 days
Amoebic liver abscess	Metronidazole 800 mg tid PO X 10 days (500 mg qid IV)

(110 Journal, Indian Academy of Clinical Medicine _ Vol. 4, No. 2 _ April-June 2003)

Nitroimidazoles (including metronidazole) are effective in over 90% of cases. Therapy should continue for at least 10 days. Relapses have been reported with this duration of therapy and the drug may be administered for upto 3 weeks. The dose of metronidazole is 40 mg/kg/day in divided dosages. Tinidazole has been used in a dosage of 1.2 g per day for 7 days , but this dosage has not been firmly established. Chloroquine, emetine, and dehydroemetine may also be used.

Emetine and deydroemetine are indicated primarily when patients develop pulmonary complications from amoebic liver abscess.

Chloroquine given in a dose of 600 mg/day for first 2 days then 300 mg/day – up to 21 days. According to **Wilmot A.J. & Powell S.J.** addition of chloroquine is valuable because of its specific concentration in the liver.

- Sumeet Bhatia, Dilip R. Karnead, Jyotsna Loak Department of Medicine – King Edmond Memorial Hospital has conducted a Randomized double trail of metronidazole VS Secnidazole in Amoebic Liver Abscess and found secnidazole 500 mg tds for 5 days or as a single dose of 1.5 gm/day – for 5 days is as effective as metronidazole in Amoebic Liver Abscess. It is safe and well tolerated.

FOR PYOGENIC ABSCESS

Although the identification of the specific micro organisms involved in pyogenic liver abscess requires appropriate culture information, initial empiric treatment with antibiotics is prudent in most cases. The aetiology of the abscess if known may suggest appropriate antibiotics regimes; however broad spectrum coverage with antibiotics effective against gram '-' ve rods, streptococcal species and anaerobes should be initiated and continued until definitive organisms are isolated and their sensitivity established.

Conventionally a prolonged course of antibiotics, lasting 4-6 weeks is required for patients with a pyogenic abscess. The justification of long term antibiotic therapy has been, the perceived avascularity of both intact and decompressed abscess cavities and the concern for concurrent, but undiagnosed purulent collections within the liver. Prolonged therapy of pyogenic liver abscess with Antibiotics resulted in lower mortality. (VM DAYAL, AK JAIN, JP BHATT, VK DIXIT AK AGARVAL Institute of Medical Science BHU / Varanasi).

Recent studies suggest, however that in adequately drained abscess only 2 weeks of antibiotic administration are necessary. However prolonged antibiotics therapy does remain necessary in patients with multiple or fungal hepatic abscess. In most patients antibiotic treatment must be accompanied by adequate drainage of the abscess cavity. Patients with multiple, small (<1.5 cm diameter) abscess,

uncomplicated by concurrent surgical disease are best treated with several weeks of antibiotic treatment. Finally most fungal abscess are miliary; therefore they are not amenable to percutaneous or surgical drainage. Prolonged therapy with a systemic antifungal agent is appropriate.

PERCUTANEOUS TRANSHEPATIC ABSCESS DRAINAGE (PTAD)

ASPIRATION

Percutaneous aspiration of abscess with a large bore needle is a safe procedure if done with meticulous aseptic precaution.

INDICATIONS

1. Patients receiving systemic amoebicidal agents yet with symptoms persisting for greater than 72 hours after the drug initiation.
2. To rule out secondary bacterial infection of the amoebic abscess
3. If volume of abscess is greater than 250 ml in volume – to reduce the risk of rupture.
4. Those located in left hepatic lobe
5. Lesions associated with marked tenderness and diaphragmatic elevation
6. When metronidazole is contraindicated during pregnancy
7. In case of pyogenic abscess aspiration provides valuable diagnostic and bacteriologic data and for young patients and if there is not co-existing intra abdominal pathology.
8. Sero Negative abscess

With ultra sonogram and CT scan localization & aspiration is easier and safe. The needle should not be introduced deeper than 10 cm as portal vein may be entered.

Three usual complications are hemorrhage, tear of the liver and secondary infection.

Aspiration is contraindicated in doubtful cases, suspected cases of hydatid cysts and in patients with bleeding tendency and also in hepatic malignancy. Balasegram described the complications of leakage which may follow needle aspiration namely subphrenic abscess (14%) and peritonitis (9%)

PERCUTANEOUS TRANSHEPATIC ABSCESS DRAINAGE BY CATHETER

Recommended for selected cases of abscess catheter drainage.

INDICATIONS

1. Thick viscous contents of an abscess
2. Failure of repeated aspiration
3. Large abscess bulging through the abdominal wall
4. Most useful for the management of pulmonary, peritoneal and pericardial complications.

The technique of percutaneous catheter drainage of abscess involves the following:

1. The two techniques: (a) **Seldinger technique** for placement of an 8 French pigtail catheter or 12 french sump drain and (b) **A Trocar & Cannula technique** for placement of a 8 French pig tail catheter. Once in position, the catheters were managed as a surgically placed drain.
2. The abscess was localized by computed tomography or USG and a safe drainage route planned that avoided the bowel and costophrenic recess by the shortest pathway.
3. A skin incision was made under lock anesthesia at the planned cutaneous entry site.
4. A no. 18 sleeve needle was inserted, choosing a pathway that traversed some parenchymal tissue ahead of the abscess to decrease the risk of peritoneal spillage of infected material.
5. A guidewire was placed within the abscess.
6. A dilator was passed over the guidewire.
7. An 8.5 French pigtail catheter or a 12 French sump drain was introduced over the guidewire by the Seldinger technique. Abscesses were evacuated by manual syringe suction, and the catheters were sutured to the skin and connected to biliary collection bags. **(World Journal of surgery-14 492-497, 1990.)**

* Average length of catheter drainage lasting from 11-19 days. Inadequate drainage and subsequent treatment failure most commonly results from catheter obstruction with purulent (obstruction) material or untreated loculations within the

abscess. Percutaneous drainage is also frequently unsuccessful in immune compromised patients

CONTRAINDICATIONS

1. Associated biliary or Intra abdominal pathology requiring simultaneous operative intervention.
2. Coagulopathy and anatomical inaccessibility preclude percutaneous catheter placement, owing to the increased risk of life threatening hemorrhage or violation of hollow viscus.
3. Multiple abscess and generalized ascites are relative contraindications.

COMPLICATIONS

Sepsis, Hemorrhage, contamination of the pleural & peritoneal cavity. Bowel perforation, Catheter related pain, Catheter displacement.

SURGICAL DRAINAGE

Surgical drainage procedure now are performed less frequently than in the past.

INDICATIONS FOR OPEN DRAINAGE

1. Most common indication is failure of conservative therapy
2. When amoebic liver abscess erodes into neighboring viscus
3. Patients with septicemia from secondarily infected amoebic abscess

4. In case of pyogenic abscess laparotomy is indicated for patients with co-existing biliary pathology, such as biliary stone or strictures
5. Patients with loculated or multiple abscess, abscess inaccessible to percutaneous drainage or involving entire lobe.
6. Shock with multi system organ failure
7. Respiratory and Renal failure
8. Weight loss greater than 10 Kgs.
9. Albumin less than 3 gm/dl

APPROACHES:

a. Transperitoneal approach

Transperitoneal approach allows for abscess and abdominal exploration to identify previously undetected abscess and location of an etiological source.

b. Posterior Transpleural apparoach

For high Posterior lesions, a posterior Transpleural approach is performed. Although this allows easy access to the abscess, the identification of multiple lesions or a concurrent abdominal pathology is not possible.

* Transperitoneal operative drainage is now the standard treatment for most patients.

HEPATIC RESECTION

- **Rarely required**

INDICATIONS

1. Isolated lobar involvement with single or multiple non healing abscess.
2. Patients with infected hepatic malignancies, Hemobilia & chronic granulomatous disease.

MANAGEMENT OF COMPLICATED LIVER ABSCESS

Management of pleuropulmonary complication

Amoebic liver abscess rupturing into pleural cavity is usually managed conservatively with Intercostal drainage. Most of the patients, resolve with this treatment. If this is not drained adequately then we have to do decortication. In case of Broncho Pleural fistula the treatment is also conservative. ICD is enough. If there is residual abscess in the bronchus not getting resolved with ICD then we have to resect the particular segment of lung. If there is broncho Pleuro Biliary fistula then we have to resect the diseased bronchous and suture the Biliary fistula.

Management of peritoneal complications

Acute generalized peritonitis with shock due to sudden rupture of an Amoebic Liver Abscess requires surgical treatment. Conservative management of intraperitoneal requires of Amoebic liver abscess was associated with a mortality of 75% to 100%. Laparotomy with complete toileting of the liver abscess as well as the peritoneal cavity have shown recoveries of 50 percent to 100 percent.

Management of pericardial complications

Pericardial drainage will be enough for abscess rupturing into the pericardial cavity but if is not doing well and the patients is not improving well then we have to do pericardiectomy. The prognosis is not good.

PROGNOSIS

- POOR

1. Age > 70 years
2. Diabetes mellitus
3. Associated malignancy
4. Multiple abscess
5. Septicemia
6. WBC > 20,000
7. Biliary etiology
8. Poly microbial bacterimia
9. Increased Bilirubin
10. Albumin Level < 2 gm/dl
11. Pulmonary Complications
12. Rupture
13. Late presentation
14. Increased SGOT
15. Aerobic abscess

MATERIALS AND METHODS

MATERIALS AND METHODS

The material used in the study consisted of 80 cases of liver abscess which were admitted in the Department of General Surgery Government Kilpauk Hospital Chennai – 10,and Govt. Royapettah Hospital, Chennai during the year 2007-2009.

CRITERIA FOR INCLUSION

1. Enlarged and tender liver.
2. Presence of Macroscopic and Microscopic features of pus in the liver.
3. Culture and sensitivity of aspirated pus.
4. Radiological evidence of raised and fixed right dome of diaphragm.
5. Ultrasonogram evidence of liver abscess.

- The pathophysiology, Clinical behavior and investigative modalities and treatment patterns are thoroughly analyzed and presented here.

CRITERIA FOR EXCLUSION

1. Clinical and Radiologically confirmed cases of Liver cyst, Liver Malignancy

RESULTS OF STUDY

RESULTS OF STUDY

TABLE

AGE DISTRIBUTION IN LIVER ABSCESS

Age in years	No. of Cases	Percentage
10-20	0	0
21-30	7	9
31-40	20	25
41-50	30	38
>50	23	28

SEX DISTRIBUTION IN LIVER ABSCESS

Sex	No. of Cases	Percentage
Male	69	87
Female	11	13

CLINICAL PRESENTATION

Clinical Features	No. of Cases	Percentage
1. Abdominal pain	76	95
2. Intercostal Tenderness	31	39
3. Enlarged Liver	35	44
4. Fever	52	65
5. H/o Dysentery	4	5
6. Nausea and Vomitting	20	25
7. Anorexia	28	35
8. Loss of weight	16	20
9. Jaundice	11	14
10. Hiccup	2	2
11. H/o. Alcohol	65	82

MODES OF PRESENTATION

Presentation	No. of Cases	Percentage
Classical	62	77
Peritonitis	5	7
Atypical	13	16

LABORATORY INVESTIGATIONS

HEMATOLOGICAL INVESTIGATION

Investigation	Value	No. of Cases	Percentage
Hemoglobin	7 -10gms./dl.	54	67
	10 – 14 gms./dl.	26	33
WBC count	5,000 – 10,000 cells / cu.mm.	33	42
	10,000 – 15,000 cells / cu. Mm.	41	51
	>15,000 cells / cu.mm.	6	7
ESR	<15mm	8	10
	>15 mm	72	90

LIVER FUNCTION TESTS

Investigation	No. of Cases (80)	Percentage
Increased Bilirubin	42	53
Increased Serum Alkaline Phosphatase	44	55
Decreased Serum Albumin	17	21

RADIOLOGICAL INVESTIGATION

X-Ray Chest and X-Ray abdomen

Radiological Feature	No. of Cases (80)	Percentage
Raised dome of Diaphragm	32	40
Pleural effusion	11	14
Basal atelectasis	8	10
Enlarged Liver	24	30
Ground Glass appearance	5	6

ULTRA SONO GRAM

In our present study all were analysed with 2 dimension ultra sonography.

USG DATA – REGARDING SIZE

Size in Cms.	No. of Cases	Percentage
<5	15	19
5 – 10	44	55
>10	21	26

DISTRIBUTION OF ABSCESS

Right Lobe		Left Lobe		Both Lobes
56 (70%)		12 (15%)		12 (15%)
Single	**Multiple**	**Single**	**Multiple**	
40 (50%)	16 (20%)	12 (15%)	0	

Right Lobe is involved predominantly

MODES OF TREATMENT

Mode of Treatment	No. of Cases	Percentage
Conservative	30	37.5
Aspiration	40	50
Percutaneous catheter drainage	5	6.25
Open Drainage	5	6.25

BACTERIOLOICAL CULTURE STUDY OF PUS ASPIRATED

No. of persons found to have bacterial growth	Organisms isolated
24 (30%)	E. coli[common] Klebsiella Pseudomonas Polymicrobial abscess Streptococci viridians, Enterococci

TROPHOZOITE AND CYST OF E.HISTOLYTICA RECOVERED

Stools	Pus
12 (15%)	8(9%)

QUANTITY OF PUS ASPIRATED

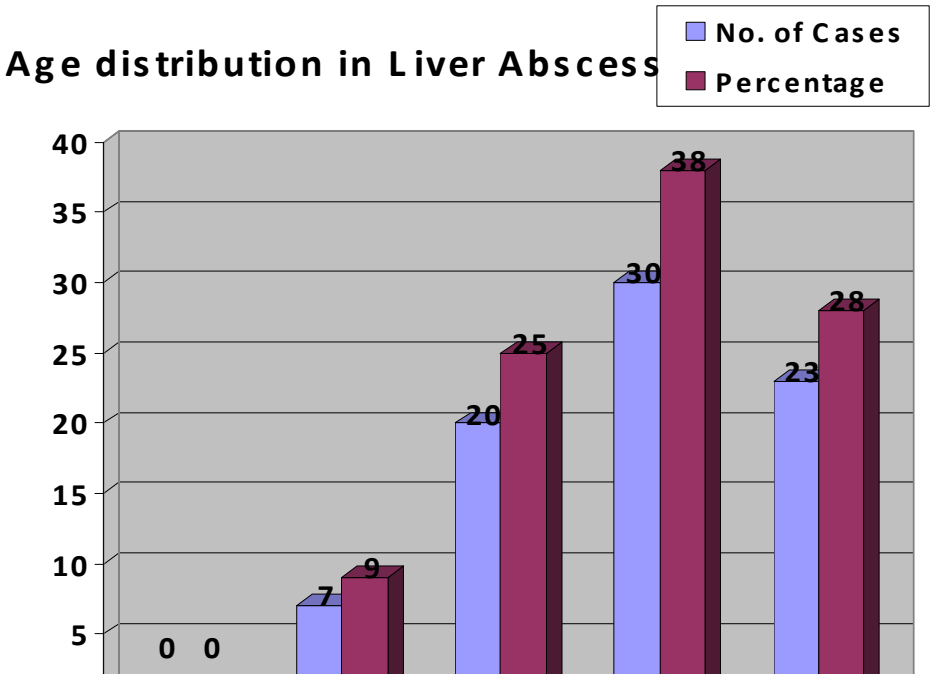
Quantity	No. of Cases	Percentage
< 100 ml	22	27
100 – 500 ml	36	45
500 – 1000 ml	12	15
> 1000 ml	10	13

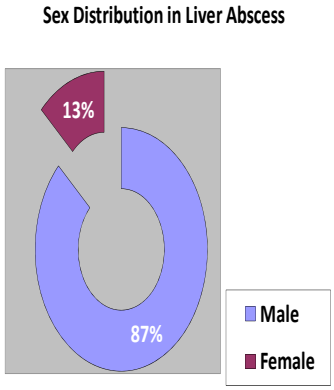
TYPES OF ORGANISM REPORTED IN PUS CULTURE

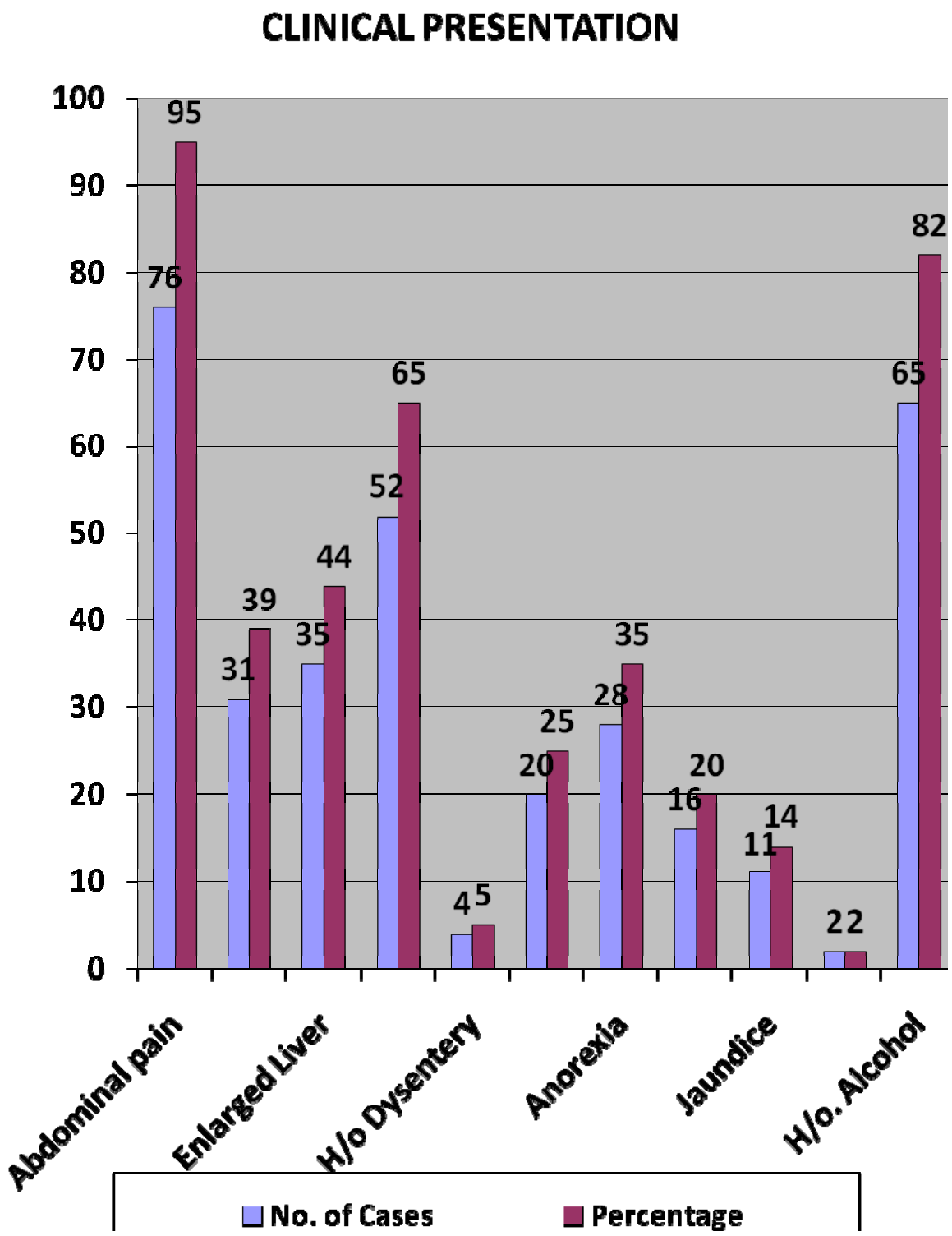
Bacterial Growth is +ve for 24 cases out of those cases.

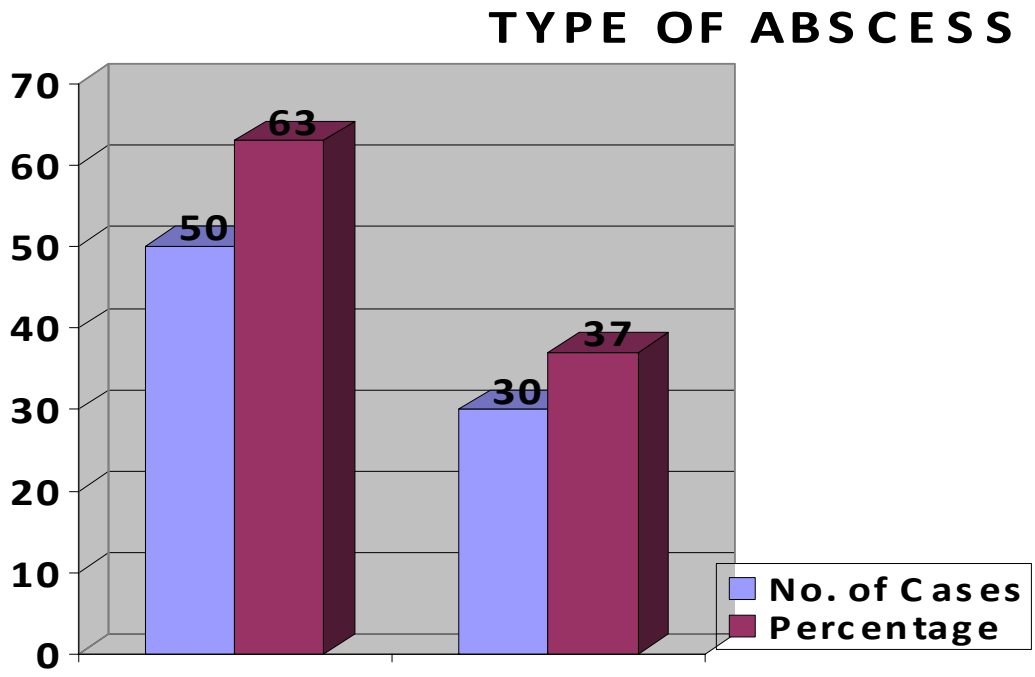
Monomicrobial	**Polymicrobial (mixed)**
19 cases (80%)	**5 cases (20%)**

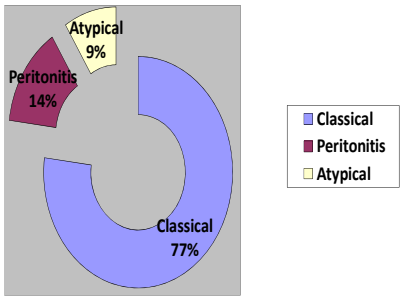
ANALYTICAL ILLUSTRATIONS


Age distribution in Liver Abscess
No. of Cases
Percentage
40
35
30
25
20
15
10
5
0
0
7
9
20
25
30
38
23
28


Sex Distribution in Liver Abscess
13%
87%
Male
Female

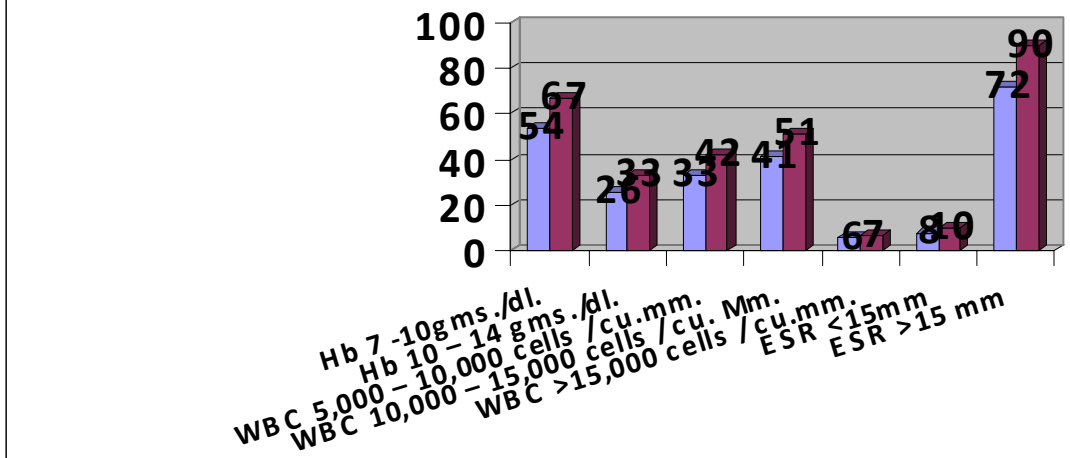

CLINICAL PRESENTATION
100
90
80
70
60
50
40
30
20
10
0
95
76
39
31
44
35
65
52
5
4
25
20
35
28
20
16
14
11
22
65
82
Abdominal pain
Enlarged Liver
H/o Dysentery
Anorexia
Jaundice
H/o. Alcohol
No. of Cases
Percentage



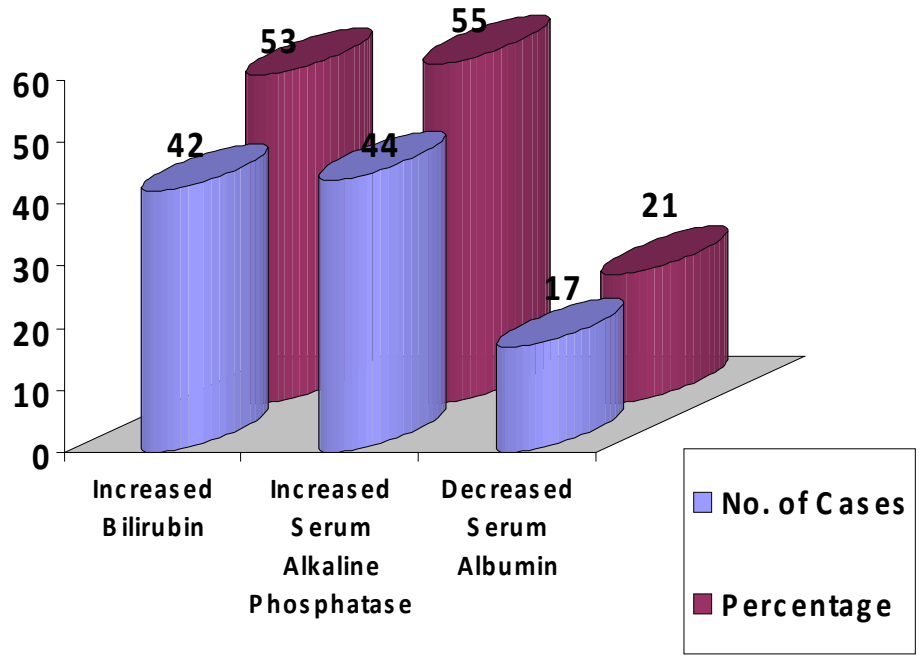


MODES OF PRESENTATION

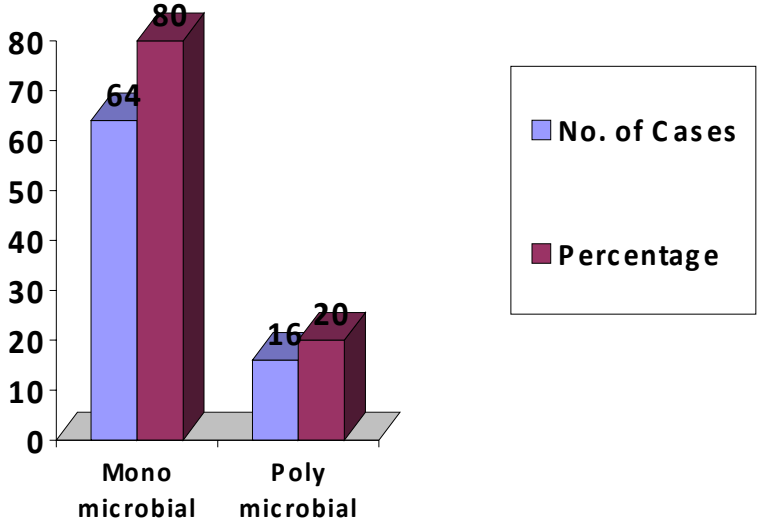
HEMATOLOGICAL INVESTIGATION



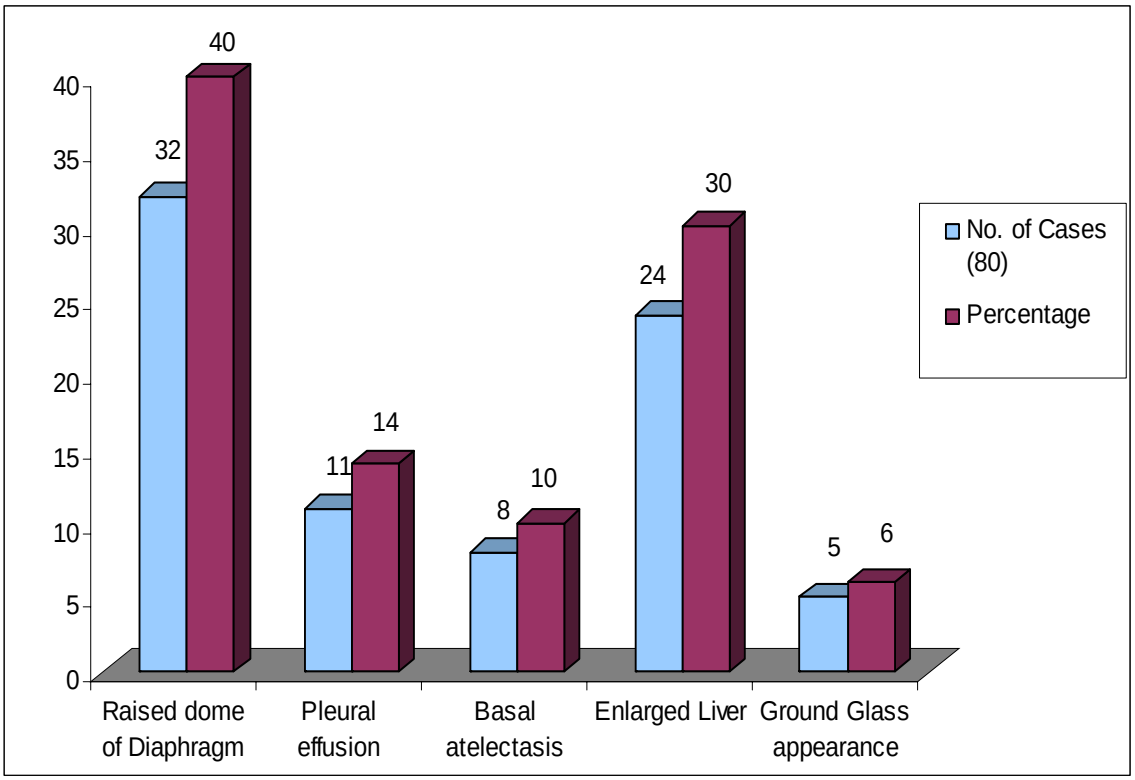
LIVER FUNCTION TESTS



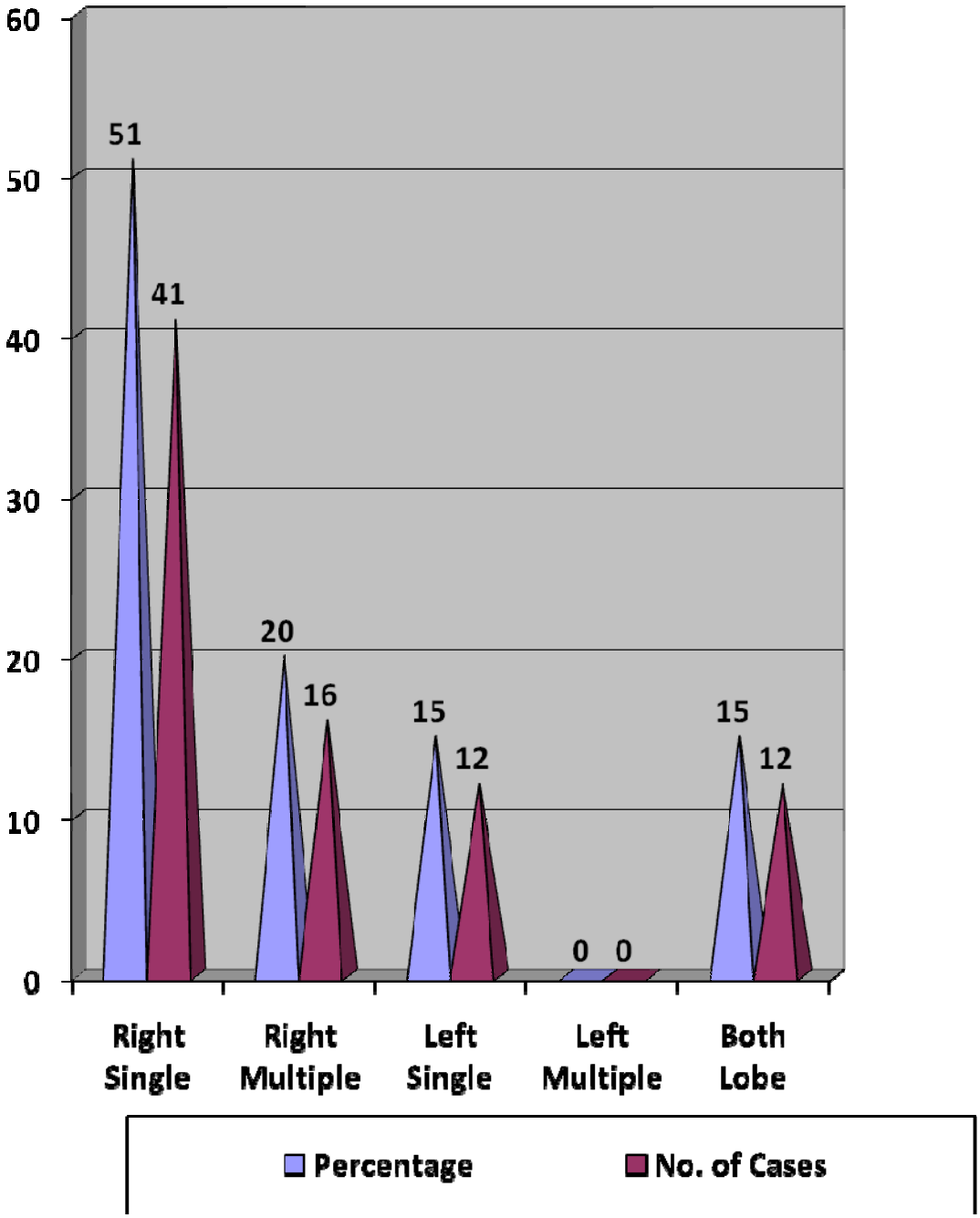
TYPES OF ORGANISM REPORTED IN PUS CULTURE



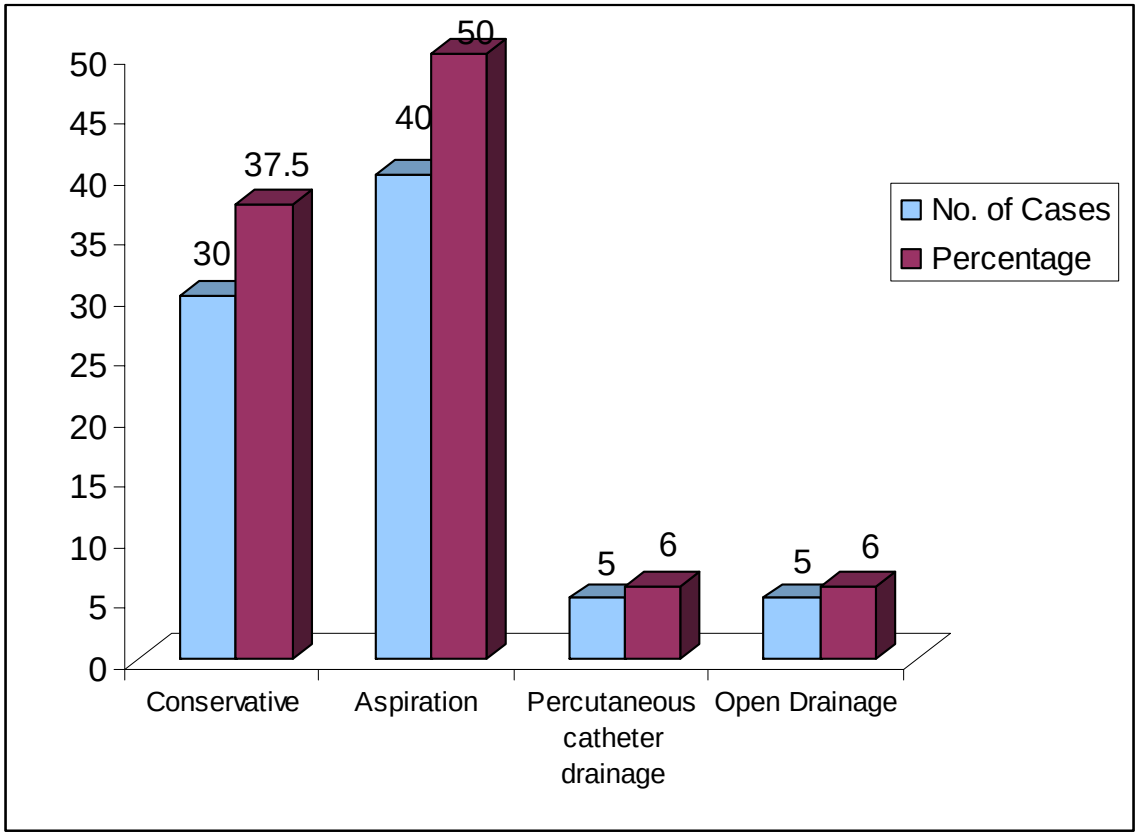
X-Ray Chest and X-Ray abdomen



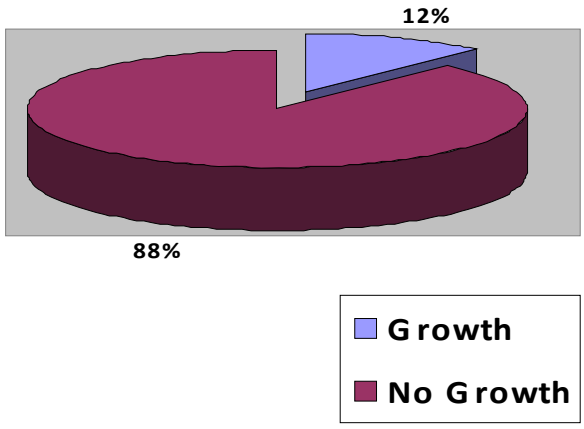
DISTRIBUTION OF ABSCESS



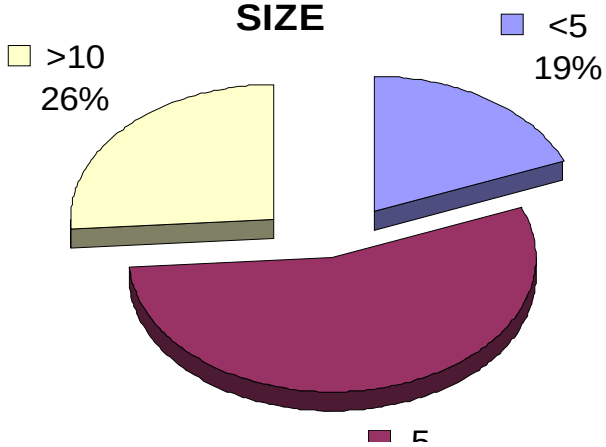
MODES OF TREATMENT



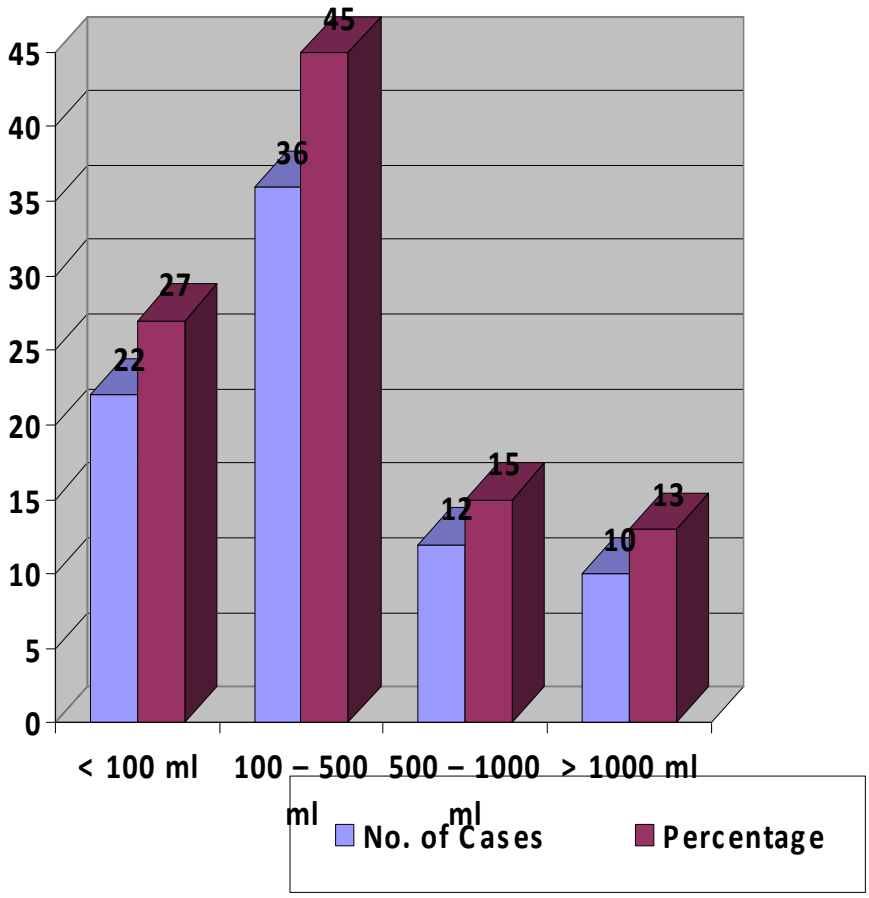
BACTERIOLOICAL CULTURE STUDY OF PUS ASPIRATED



USG DATA – REGARDING SIZE



QUANTITY OF PUS ASPIRATED



DISCUSSION

DISCUSSION

The condition of hepatic abscess and its grave prognosis were known in ancient time to **Hippocrates** (160 BC -370BC) and **Celsus** (53 BC - &AD) Hippocrates was able to distinguish liver abscess from cystic liver disease. Celsus appreciated the poor prognosis of hepatic abscess associated with Jaundice. Not until 1936 did Bright in his own observation on Jaundice clearly described hepatic suppuration with true abscess formation.

Oschner, Debakey and a Murray 1938 in their classic article reported amoebic liver abscess, 75% as very common in the warmer southern climate. Liver abscess though a well defined clinical entity yet many difficulties were faced in determining the site, size and number of abscess. **Chuttani et al** 1963 have commented that the difficulties in clinical diagnosis of hepatic amoebiasis can be diverse and real. Those who do not meet the condition frequently are not likely to appreciate them fully. Untreated liver abscess has higher mortality rates approaching 100%. Reports of successful medical management with or without aspiration describe case fatality rates as low as 6%.

In our study out of 22,000 cases admitted in our hospital in 2007 to 2009. We are reported about 80 cases of liver abscess. Incidence being 2.7%. In our study peak age incidence was noticed in 4^{th} decade followed by Fifth and Sixth decade. In our study there is more number of cases in low socio economic status. According to Garewal the highest incidence was noted in people who consume

alcohol and also in people who live with poor hygienic conditions, contaminated drinking water, malnutrition, hepatic dysfunction and low host resistance.

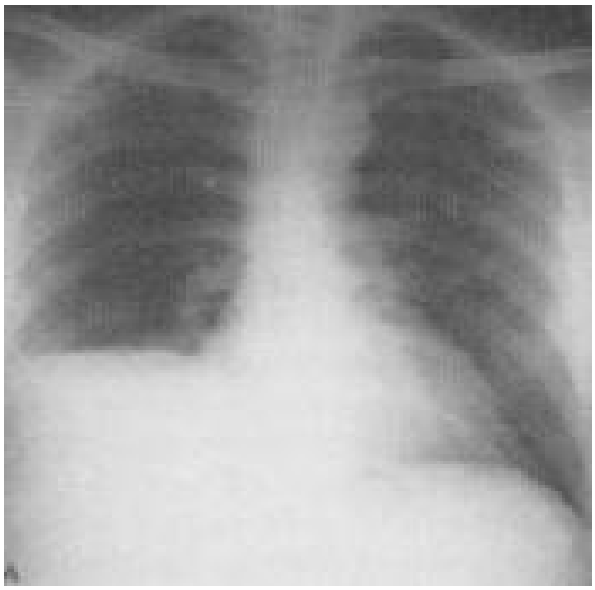
Highest incidence of liver abscess in males 87% in our study has been attributed to acoholism, (Present study H/o alcoholism was present in 82% cases.) This correlates with the study of **Oschner & Debakay** which predispose to hepatitis. Alcohol produces heptaocellular damage and may make it prone to develop hepatic abscess – **Sheila – Sherlock.**

Presentation	**Present Study (n=80) %**	**D.S Sing et al (1980)(n=42) %**	**Barnas et al (1987(n=96) %**
Pain right Hypochondrium	95	100	67
Fever	65	100	87
H/o. Diarrhoea/Dysentry	5	85	35
Jaundice	14	24	10
Weight Loss	20	-	10
Appetite	35	-	45
Breathelessness	5	-	24

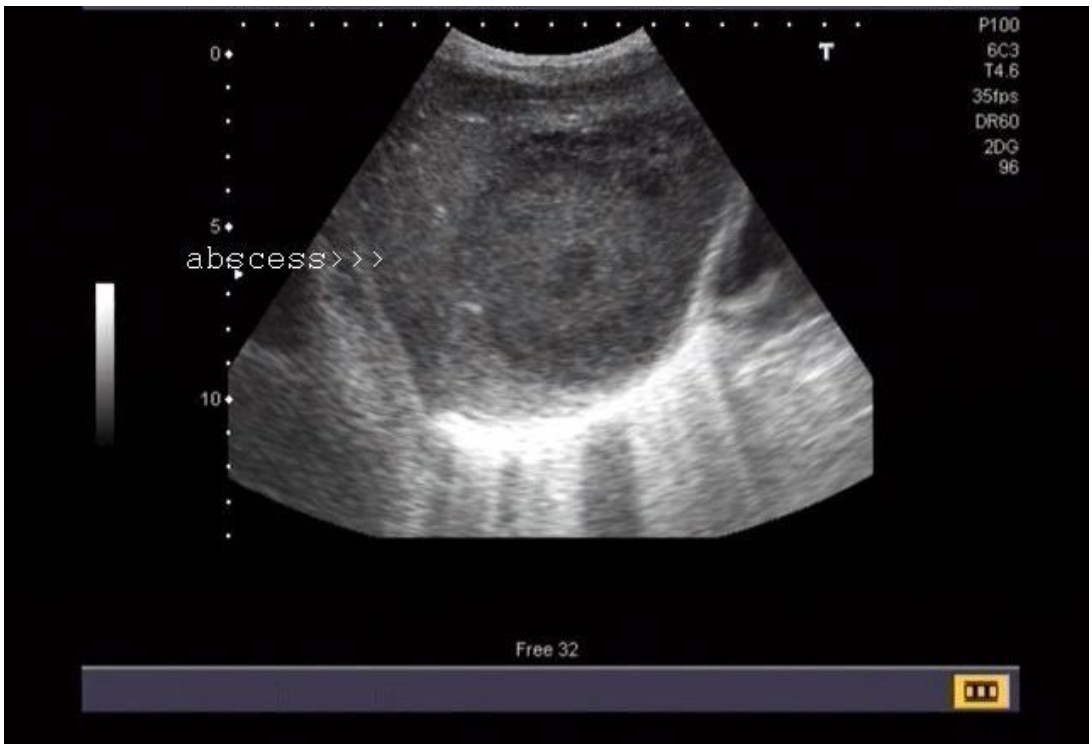
In our study commonest symptom being abdominal pain and fever. Commonest sign being Intercostal Tenderness and Tender hepatomegaly.

In present study, anemia was noted in 67% cases and Jaundice in 14% patients especially in Pyogenic liver abscess patients.

X-RAY SHOWING THE LIVER ABSCESS WITH ELEVATION OF THE RIGHT DOME OF DIAPHRAM



USG SHOWING THE LIVER ABCESS



COMPARATIVE STUDY

Test	Ramachandran % (1976)	Sharma % (1980)	Prasad % (1987)	Present % (2007)
Hyper Bilirubinemia	8	10	7	53
Hypoalbuminemia	42	64	57	55
Increased Serum Alkaline Phosphatase	50	37	41	21

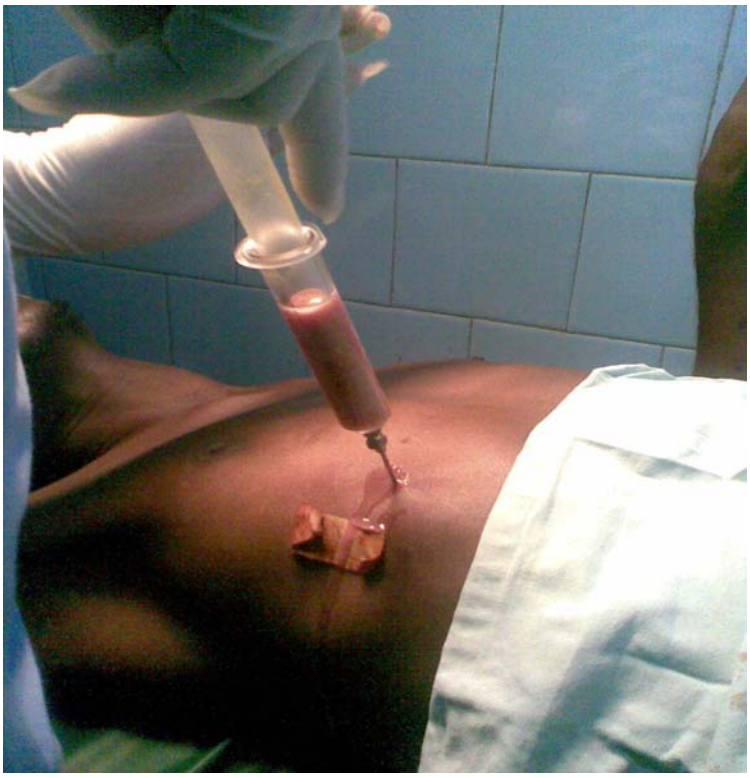
Data concluded in his study that Jaundice in liver abscess is primarily of cholestatic origin. Intrahepatic cholestasis which is due to compression of Both hepatic ducts. Though Lamot and Pooler, Vakil et al, Hazra et al have noted an increase mortality in liver abscess with Jaundice we have not encounted such thing in this study.

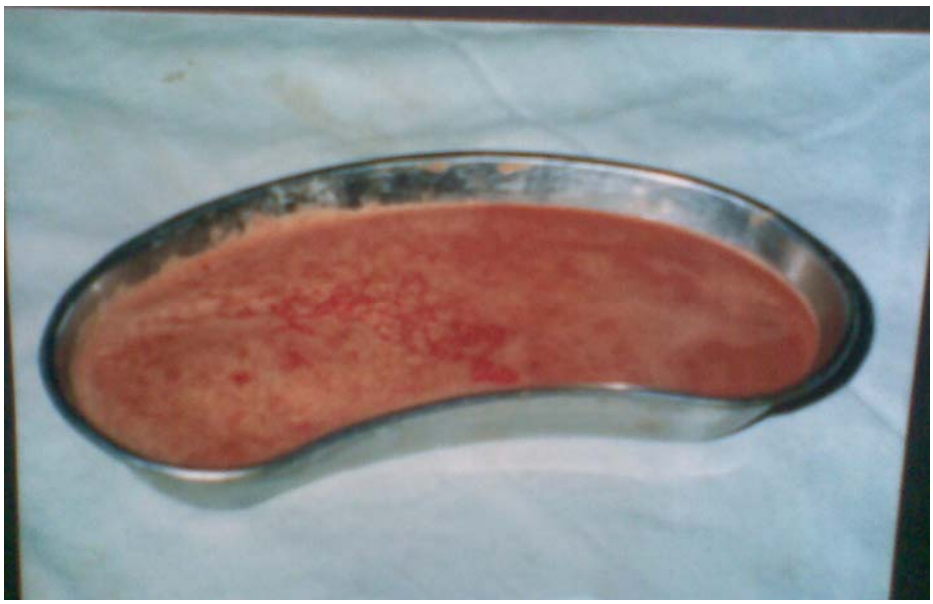
NON INVASIVE METHODS

PLAIN X RAY CHEST & ADDOMEN

In the present study raised right hemidiaphragm was noted in 47% cases and enlarged liver in 36% of cases.

ANCHOVY-SAUCE PUS ASPIRATED





ULTRA SONOGRAPHY

The advent of Ultrasonography has opened a new horizon of the diagnosis of hepato biliary disease. Sonography is of immense help in finding the abscess cavity, its location, number and valuable in aspiration and follow up. It is also useful in finding out the nature of PUS, So that helpful in determining the size of needle for aspiration.

In our study the sensitivity of ultrasonogram is around 97%. However false positive results (3%) was encounted in this study turned out to be degenerating Hepatomas.

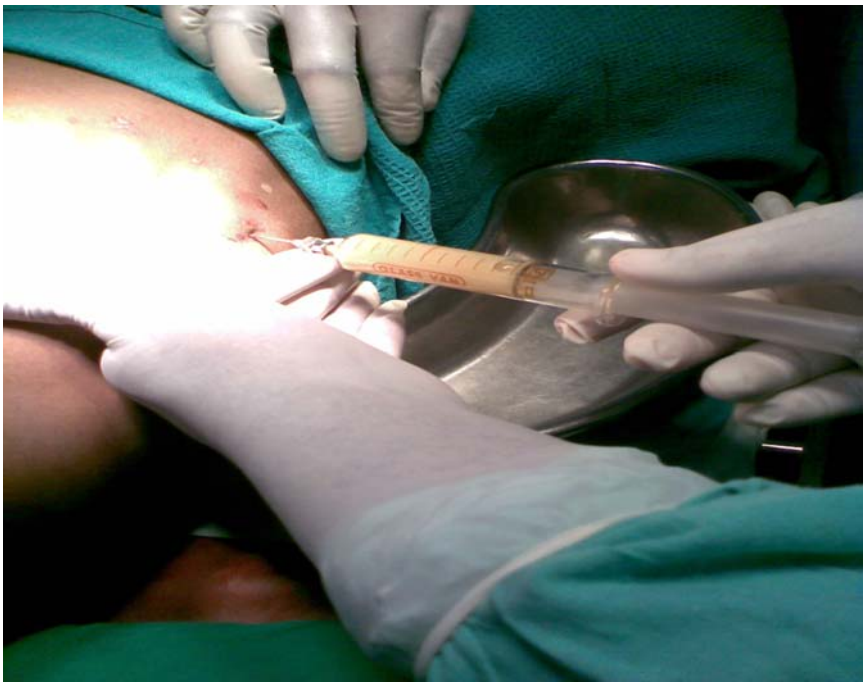
Similarly **Cimmino CV. Scott DW** reviewed as case report of a benign liver tumor with central necrosis which was misdiagnosed clinically as liver abscess.

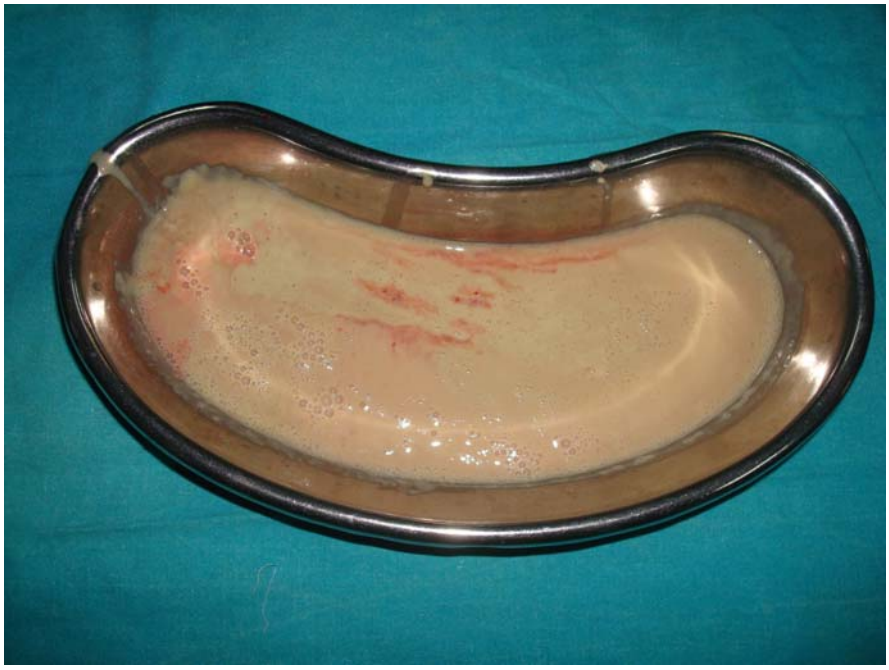
COMPARATIVE STUDY

LOBE INVOLVEMENT – USG

Author	Right %	Left %	Both%
RALLS (1979)	83.3	16.7	-
BOULT BEE (1979)	77	15	8
PRESENT (2009)	70	15	15

PYOGENIC PUS ASPIRATED FROM LIVER ABSCESS





NUMBER OF ABSCESS CAVITY

Number	Present Study %	K.L. Ghose et al %
Single	85	86
Multiple	15	14

LEFT LOBE ABSCESS

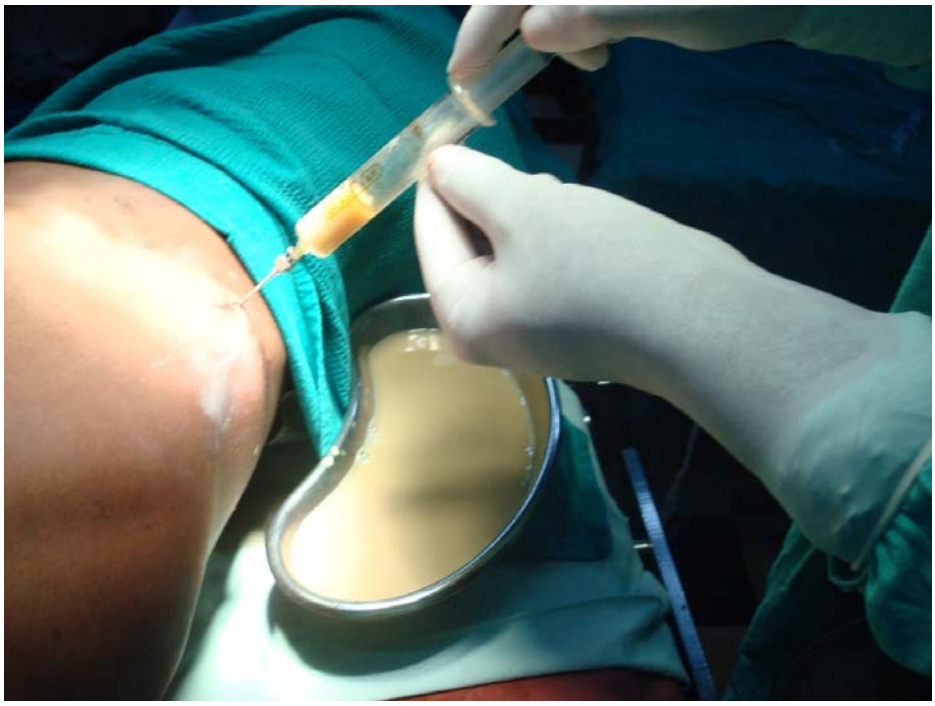
Study Groups	Left Lobe Abscess %
De Bakey and Ochsner	10
Abdul Khair et al	36
Harinasuta et al	8
Wilmot	13
Present Study	15

All right Lobe Abscess were diagnosed by clinical enlargement of liver whereas such definite Lobe enlargement was not seen in Left Lobe correlated with previous study.

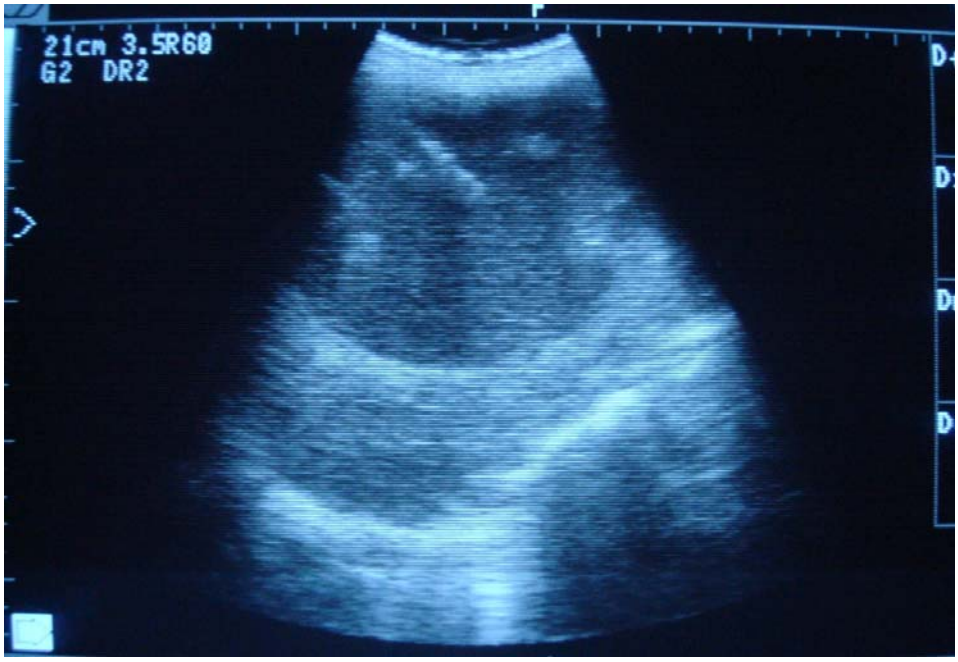
MANAGEMENT

In the present study the diagnosis of liver abscess mainly depends upon the clinical feature and ultrasonography and to lesser extent with stool and pus examination.

USG GUIDED NEEDLE ASPIRATION



USG SHOWING THE NEEDLE INSIDE THE ABSCESS CAVITY



CONSERVATIVE TREATMENT

Out of 80 cases studied, for about 30 conservative treatments was given. In the present study we had a protocol of managing the liver abscess of size less than 5 cm on ultra sonogram with conservative management (drugs).

We used to treat the amoebic liver abscess patients with Ciprofloxaxin 200 mg twice daily, metronidazole 500mg thrice daily both parenterally for five days (and then changed to oral preparation) along with chloroquine 300 mg twice daily orally.

Pyogenic liver abscess were treated first with empirical antibiotics – Ampicillin Gentamycin and Metronidazle, or 3rd generation cephalosporin and Metronidazole then changed according to culture and sensitivity. Most of the patients (90%) resolve and do better with conservative management. About 10% patients whose size doesn't decrease with antibiotics even after 4-5 days were aspirated under ultrasound guidance.

ASPIRATION

Out of 80 cases studied, for about 40 cases closed needle ultrasound guided aspiration was done. We did aspiration using 14 gauge needle for the patients in whom conservative line of management fails and for abscess of more than 5 cm size. Prior to Aspiration we routinely did bleeding time and clotting time. Injection vitamin K One ampule was given daily for 3 days prior to aspiration. When anti

amoebicidal drug has been given, thick pus begins to liquefy and it can be aspirated 3 days later under ultrasonogram control. For 15 cases we did a second aspiration after a week as the size of abscess was more than 5 cm, in spite of giving parental antibiotics.

No biliary peritonitis was encounted in this study, where as Balasegaram has described the complications of leakage which may follow needle aspiration eg. Subphrenic abscess (14%) and peritonitis (9%). No such complication arise in our study.

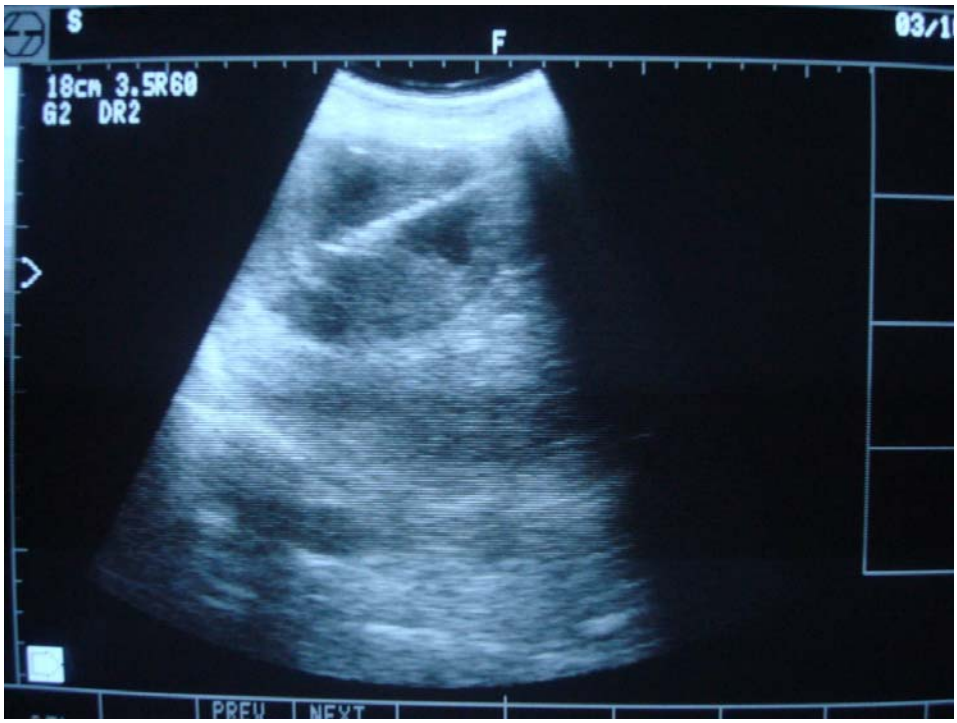
Generally we treated the patients with parenteral antibiotics for 5 days and then changed to oral antibiotics. After discharging the patients we advised them to continue tablet metronidazole for 3 weeks and T.Chloroquine for 2 weeks. We reviewed the size of the abscess with ultrasonogram before discharge and then every 2 months, until the abscess resolves. In our present study it took 3-4 months for complete resolution of abscess by ultrasonogram.

PERCUTANEOUS DRAINAGE (PIG TAIL CATHETER)

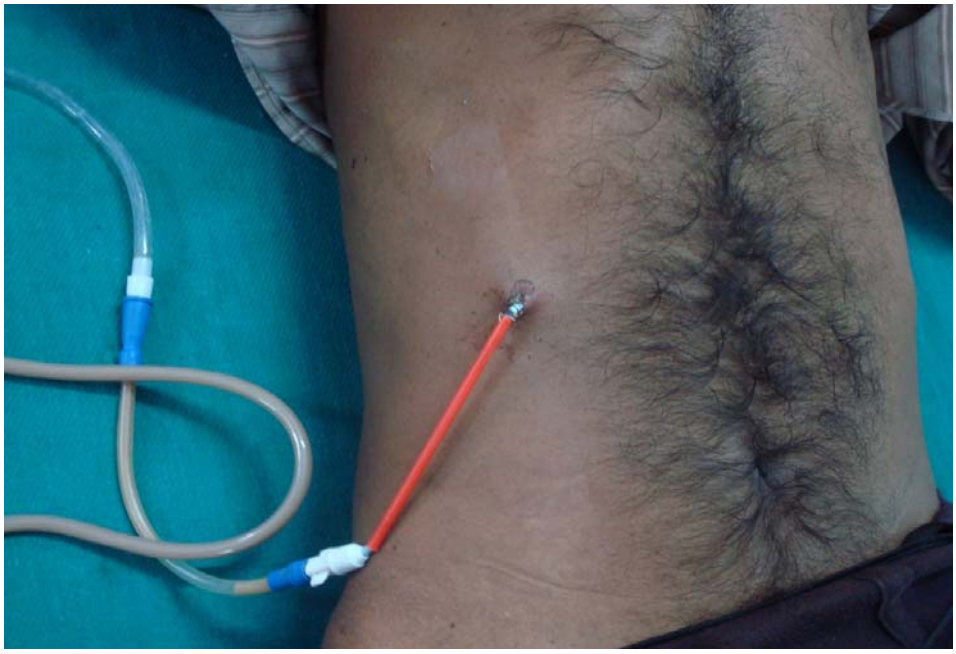
A trocar cannula technique used for placement of a 8 french pigtail catheter. Once in position the catheter were managed as a surgically placed train.

Intravenous antibiotic is the first line, and mainstay, of treatment. Drainage is necessary for large abscesses, equal to or larger than 5 cm in size, to facilitate

USG SHOWING THE PIG TAIL CATHETER INSIDE THE ABSCESS CAVITY



PICTURE SHOWING A PATIENT WITH PIG TAIL CATHETER IN-SITU



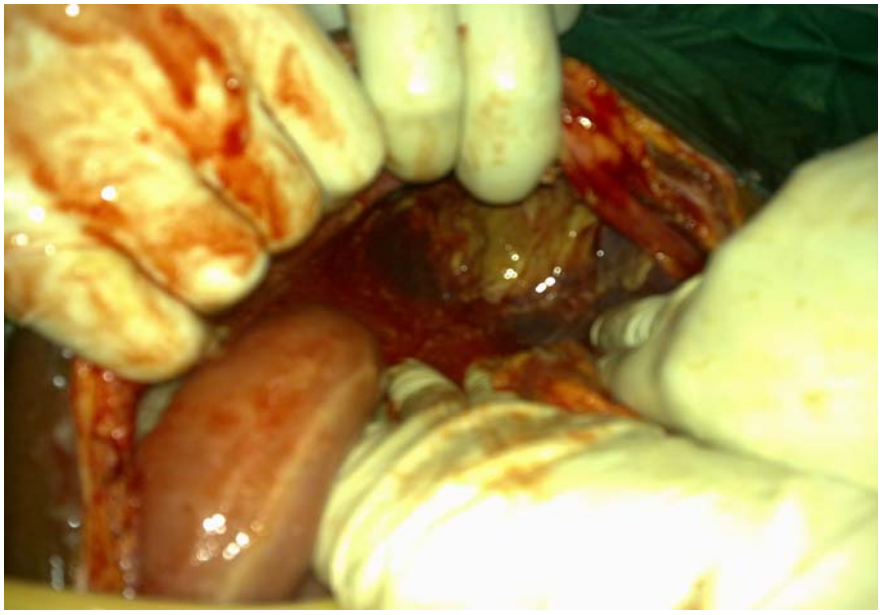
resolution. While percutaneous drainage is appropriate as first-line surgical treatment in most cases, open surgical drainage is prudent in cases of rupture, multi loculation, associated biliary or intra-abdominal pathology. Percutaneous drainage may help to optimise clinical condition prior to surgery. Laparoscopic drainage is a feasible surgical option with promising results in the future.

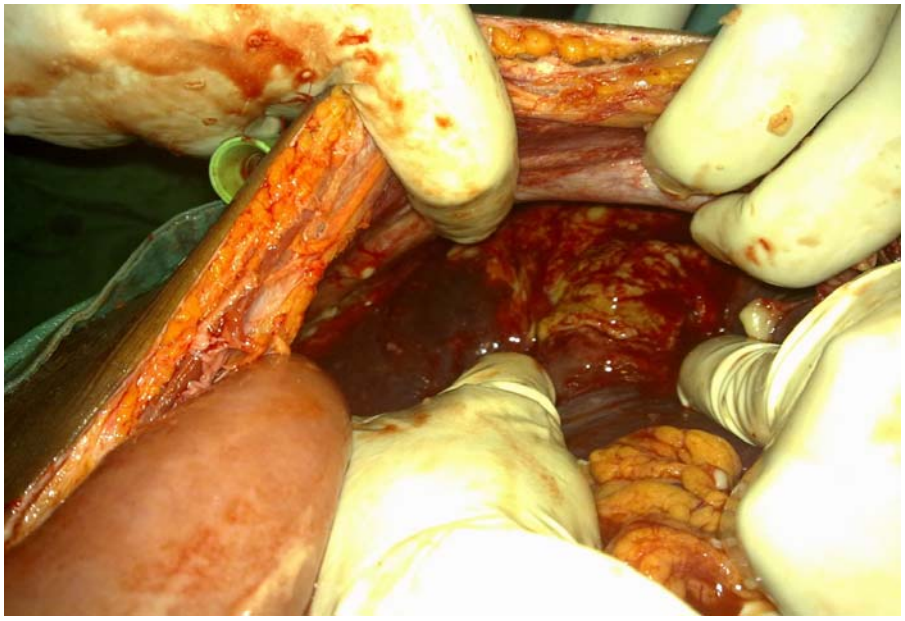
We did percutaneous drainage for 5 cases for those patients USG showed solitary liver abscess size more than 8-10 cm in right or left lobe under Local Anesthesia skin incision made. Percutaneous drainage of the abscess done by Trocar and Cannula method Pigtail catheter was introduced into the abscess cavity under USG guidance and catheter was fixed to the skin. Daily aspiration was done. Saline or metrogyl wash given through the catheter to liquefy the pus and drained.

Repeat computed tomographic scans were obtained to assess resolution. Irrigation was not routinely performed except when the abscess contents were too thick to drain through the 8.5 French catheter. In those cases, saline solution was used for gentle lavage until the contents flowed easily. Initially, broad spectrum antibiotics (usually an aminoglycoside, a cephalosporin, and metronidazole) were administered to all patients, but when sensitivities were obtained, the antibiotic program was changed to be organism specific.

After confirming with the USG for complete resolution of the abscess cavity ,catheter was removed on 7^{th} or10th day.

LAPARATOMY & OPEN DRAINAGE OF RUPTURED LIVER ABSCESS





Before percutaneous catheterization BT, CT, PT, INR, Serum Bilubirin was done and injection vitamin K given. Aspirated Pus sent for culture and sensitivity. According to the sensitivity antibiotic regime is changed.

LAPAROTOMY

With the advent of modern imaging techniques the role of open drainage of hepatic abscess is almost negligible.

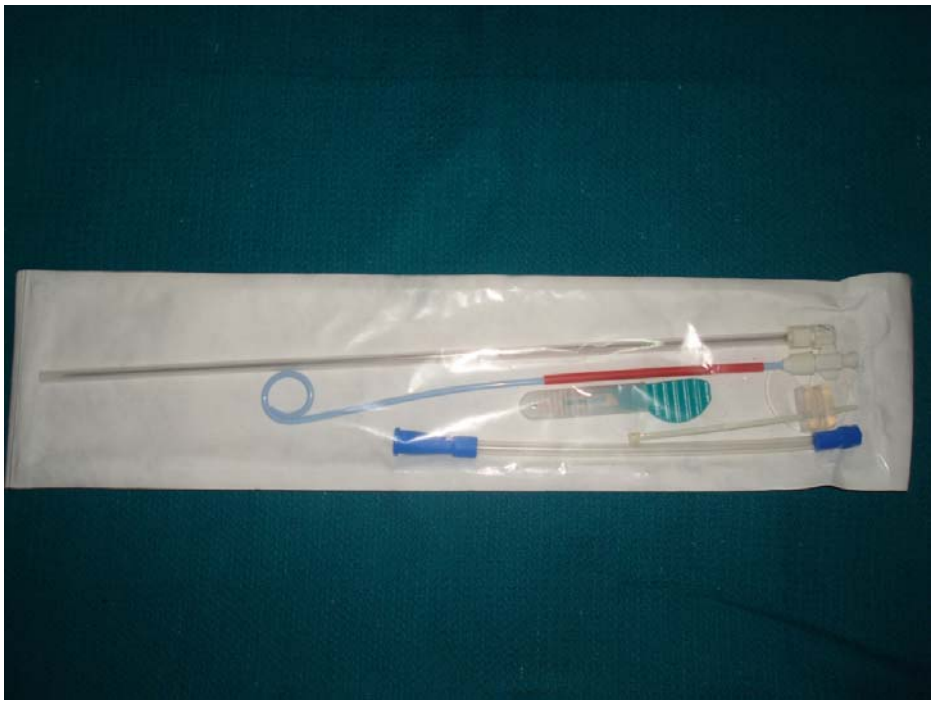
We did laparotomy and open drainage for 5 cases which were presented as

a. Acute abdomen where abscess burst presenting as perforated peptic ulcer, pancreatitis, ileal perforation or as perforated appendicitis.
b. Patients not responding to aspiration/antibiotics.
c. For large multiple abscess

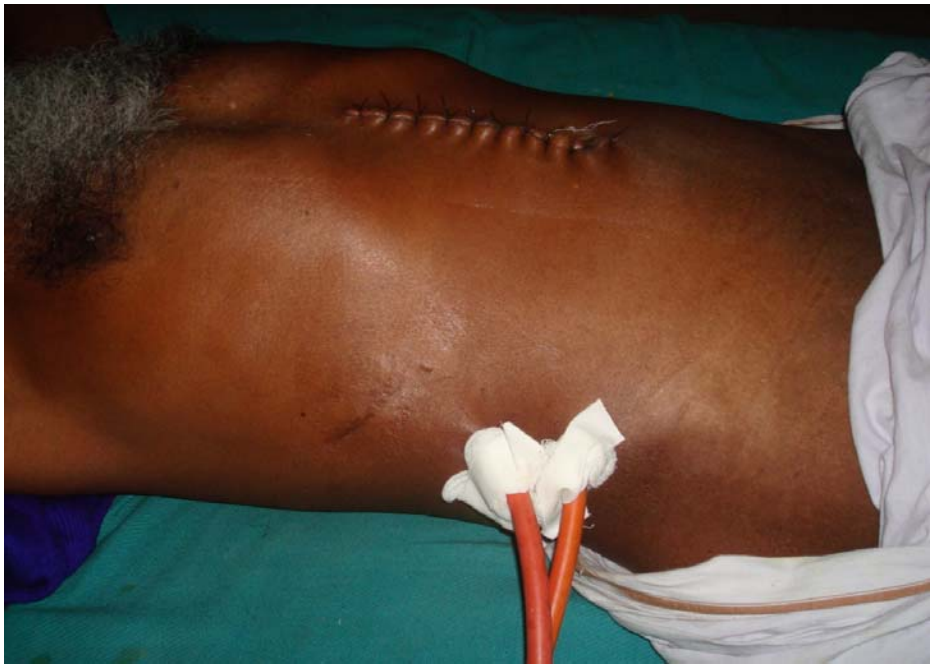
Open drainage done with malecot's or foley's catheter in situ.

One patient who presented as Acute abdomen (USG showed ruptured liver abscess) open laparotomy done abscess cavity present in for posto superior part of right lobe of liver which was removed, cavity washed with saline and external drainage done with malecot's catheter in situ in the abscess cavity. Patient expired on 9^{th} POD due to peritonitis and sepicemia leading to acute renal failure with acute respiratory distress syndrome.

PIG TAIL CATHETER



EXTERNAL DRAINAGE OF LIVER ABSCESS WITH MALECOT'S CATHETER



SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

Out of 80 cases taken out for study majority presented with classical features.

1. Incidence of liver abscess is 2.7% of total admissions in our hospital. Incidence of Amoebic liver abscess very common in our study. The ratio of amoebic liver abscess: pyogenic liver abscess being 5:3
2. About 82% of patient were alcoholic
3. Male predominates both in amoebic and pyogenic liver abscess in the ratio of 7:1
4. Anemias, Leucocytosis were common Accompaniments.
5. Commonest symptom is abdominal pain and fever, sign being Tender hepatomegaly and Intercostal Tenderness.
6. Only 14% of patients presented with Jaundice.
7. Right Lobe was predominantly involved in the ratio of 7:3
8. Clinical diagnosis of liver abscess is straight forward except for those presenting with complications.
9. Only 15% of patients, we are able to isolate E. Histolytica from stool and 9% from PUS.
10. Out of 80 cases treated, 30 cases were treated conservatively, 40 cases required aspiration, 5 cases required percutaneous catheter drainage and 5 cases required open drainage.
11. Ultrasound is the commonest and most useful investigation for diagnosis, treatment as well as follow up.

12. Age incidence: Higher incidence of liver abscess in age group of between 40-50 years.
13. CT Scan mainly reserved for doubtful cases and those presenting with complications.
14. In our series, there is more number of abscess seen in low socioeconomic status patients. 30% of patients are in High Socio Economic status, 70% of patients are in low Socio Economic Status.
15. Mortality in our series

Uncomplicated liver abscess	:	No mortality
Ruptured into abdominal cavity	:	1 death

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Sir Alfred Cuschieri, Rober J.C. Steele, Abdool Rahim Moosaa - Essential Surgical Practice 4th Edition
2. Sabiston textbook of surgery Vol-2, 18th Edition.
3. Chummy S. Sinnatamby – Last's Anatomy Regional and applied 10th Edition.
4. Ochesner A. Debakay, M. Murray, A. Pyogenic abscess of the liver, A.M.J.Surg 1938:40:292.
5. Balasegaram M. Management of Hepatic abscess. Curr Surg 1981 18:283
6. Bailey and Love's short Practive of Surgery.25th Edition.
7. Essential Surgical Practice A. Cuschieri M.D., G.R. Giles MS, A.R.Moosa M.D. 4th Edition.
8. Lippincott's Color Atlas and Text Book of Diagnostic Micro Biology 5th Edition.
9. Oxford Text Book of Surgery Peter. J. Morris and Ronal A. Malt First Edition.
10. Chatterjee K.D., Parasitology 12th Edition.
11. Maingot's Abdominal Operations 10th Edition.
12. Eggleston Fc et al. The results of Surgery in Amoebic Liver disease. Experience in 83 Patients surgery 536-539, 1978.

13. Schwartz SI Extraperitoneal drainage of liver abscess. Surgical diseases of liver McGraw Hill 1964.
14. European Journal of Radiology 20(1):65-7 1995 May – Baijal SS, Agarwal DK, Roy S., Choudhuri. G. Complex Ruptured Amoebic Liver abscess; the role of percutaneous catheter drainage.
15. Indian Journal of Gastroenterology April 1998 Volume 17, No.2
16. Tropical disease Bulletin July 1996, Vol.93 No.7.
17. Gastroenterology today Vol.I. No.3 ISSN. 0971-8222 April June 1997 Invasive Amoebiasis – MP Sharma, S. Dasavathy, AIIMS, New Delhi.
18. Gastroenterology today ISSN: 0971-8222 Vol II No.1 Jan.March:1998 P/49-51
19. Johnson RD. Mueller Pr. Ferrucc JT. Jr. et al, Percutaneous drainage of pyogenic liver abscess AJR:1985 144:463
20. The Indian Journal of Gastroenterology vol 16: Nov 1997 controversies in Gastroenterology Von.DAYAL, AK JAIN, JP BHATT, VK DIXIT, AKAGARWAL.
21. Mendez RJ, Schiebler ML et al Hepaticabscess; MR imaging findings Radiology 1994; 190-431
22. Ralls PW, Colletti PM, Quinn MF et al. Sonographic findings in hepatic amoebiasis Radiology 1982; 145:123

23. Ralls PW Quinn MF, Bows well WD Jr et al., Patterns of Resolution of Successfully treated hepatic Amoebic abscess Sonographic evaluation.
24. Van Sonnenberg, E Mmeller PR Schiffman HR et al. Intra hepatic amoebic abscess; Indication and results of Percutaneous Catheter drainage.
25. Chuah S.K. Chung – Chien C.S., Sheen IS, Lin HH, Chiou SS, Chiu CT The Prognostic factors of severe Amoebic liver abscess a retrospective study of 125 cases.
26. Adams EB, Macleod IN Invasive Amoebiasis – Amoebic liver abscess and its complications medicine 1977 56:325
27. VAkil BJ. 1983 Amoebic disease. A treatise, Basic and Business Publications, Bombay.
28. Lee KC, Yamazaki O. Hamba H. Analysis of 69 patients with Amoebic liver disease. Journal of Gastroenterology 31 (1) 40.5 1996 Feb.
29. Basile JA: Klein, IR. Worthen NJ. Wilson, S.E., Hiatti JR Amoebic liver abscess. The surgeon's role in the Management Am. J. Surg; 146:67:71 1983.
30. Filice C, Dipierri G, Strosselli, M.Brunett. E. outcome of Hepatic amoebic abscess managed with 3 different therapeutic strategies.

31. Agarwal DK, Baijal SS, Mittal BR, Choundhuri G. Percutaneous Catheter drainage of Amoebic liver abscess.
32. Madana Gopalan N, (1976) Amoebiasis in progress in clinical medicine in India. Ed., M.M.S. Ahuja, New Delhi, Arnold Heinman, 307.
33. Singh DS, Sankaian A, Bhata V.N. Srivatsava K.K. Chandrasekaran S. Extra Intestinal Amoebiasis – A clinical and Serological Study.
34. Reynolds T.B. Amoebic abscess of liver Gastroenterology 60:952-953 1971.
35. Sheila Sherlock & James Dooley 10th Edition. Diseases of the liver and Billiary System.
36. Disease of the liver 5th Edition. Leon. Schiff M.D., Ph.D., Eugene R. Schiff M.D.
37. WHO (1969) Amoebiasis Tech. Report Series 421.
38. Ibrarullah M. Agrawal D.K. Baijal SS, Mittal B.R. Kappor U.K. Amoebic Liver abscess with Intra Biliary Rupture – HPB Surgery.
39. Meng XY, WU,JX Perforated Amoebic liver abscess clinical analysis of 110 cases. Southern Medical Journal.
40. Wilmott AJ. (1962) Clinical Ameobiasis, Blackwell Scientific Publication, Oxford.
41. Patterson M, Healey, GR. Shabot JM (1980). Serological Testing for Ameobiasis. Gastroenterology 78, 136.

42. Lamont N.M, Pooler NR (1958). Hepatic Amoebiasis. Quart, J. Med 27:389.

43. Dela Rey N.J. Simjee AE, Patel A. Indications for Aspiration of Amoebic liver abscess S Afr med.J. 1989 75:373

44. Barnes PF, Decock KM et al. A comparison of Amoebic and Pyogenic abscess of the liver. Medicine 1987 66:472

45. American Journal of Roentgenology, Vol 133, Issue 1, 1-8 Copyright © 1979 by American Roentgen Ray Society

46. Singapore Med Journal 2007; 48(12) : 1158

47. 108 Journal, Indian Academy of Clinical Medicine _ Vol. 4, No. 2 _ April-June 2003

48. 109 Journal, Indian Academy of Clinical Medicine _ Vol. 4, No. 2 _ April-June 2003:

49. 110 Journal, Indian Academy of Clinical Medicine _ Vol. 4, No. 2 _ April-June 2003

50. Indian journal of gastroenterology – April 1998 Volume : 17 p.58.

51. Tropical Hepatogastroenterology, Chapter 38 Intestinal and extraintestinal amebiasis-MP Sharma & Vineet Ahuja

52. Text book medical parasitology 2nd Edition by Subash Chandra Parija

53. World Journal of surgery-14 492-497, 1990.

PROFORMA

PROFORMA
DEPARTMENT OF GENERAL SURGERY
GOVERNMENT KILPAUK MEDICAL COLLEGE

Case No : IP No :

Name: Occupation : DOA:

Age : Religion : DOD:

Sex :

Address:

1. Complaints –
2. History of present illness –

   a. Pain abdomen – Present / Absent
      - Onset
      - Duration
      - Type: Dull aching / Sharp / discomfort
      - Site: (Rt) Hypochondrium/ Epigastrium / others
      - Radiation: Present / Absent
      - Relation to cough : increased / decreased / no relation
      - Relation of food - ↑ | ↓
      - Bowel movements/ Present/ Absent.

   b. Fever - high grade / low grade
      - Sustained / Spiking

   c. Nausea / Vomiting

   d. Appetite : ↑ , ↓, no change

   e. Loss of Weight → +/-

f. Jaundice → +/-

g. Diarrhoea → Duration & Frequency

nature – mucous/ Bloody Quantity

h. Cough → Duration

Dry (or) Productive

Sputum → amount and colours.

i. Pleuritis → +/-

j. Lump → +/-, Site, Duration, Progress

3. H/o previous Illness - Similar complaints

- H/o Diarrohea +/-
- H/o Previous Treatment if any
- H/o Previous surgery
- How many weeks before

4. Personal History
- Diet – Veg./mixed
- H/o Smoking
- H/o Alcoholism

5. Menstrual History

6. Obstetric History

7. Family History - +/-

8. Socio economic history - Low/Middle/High

9. Physical Examination :

General Examination

Built and Nourishment	Temperature
Anemia	Respiration
Jaundice	Pulse
Skin lesions	BP
Lymphadenopathy	Pedal edema
	Any others

Local examination:

Abdomen:

Inspection:

- Movement with respiration normal / restricted / absent.
- Mass +/-
- Distention +/-

Palpation:

Tenderness +/-

Hepatomegaly +/-

Tender / Non tender / Soft / firm / Hard/smooth / Nodular / Irregular

Mass +/-

Free fluid +/-

Percussion:

Liver dullness	:	Normal / ↑ | ↓
Over the mass	:	Dull / Resonant / Impaired
Free fluid	:	Fluid thrill / shifting dullness/pudel's Sign

AUSCULTATION:

PR:

SYSTEMIC EXAMINATION:

Cardiovascular system.

Respiratory system

- Movements – Normal / Restricted
- Breath sound → ↑ | ↓
- Percussion – Resonant / Impaired /Dull
- Inter costal dullness → +/-

PROVISIONAL DIAGNOSIS:

DIFFERENTIAL DIAGONOSIS:

BENIGN:

Acute Cholecystitis.

Hepatitis – Viral

Hydatid cyst

Simple cyst

MALIGNANT:

Hepatoma

Secondaries of liver.

INVESTIGATIONS:

Hb%

TC,

DC – P, L, E, M

ESR

Urine – Albumin / Sugar / Deposits

Stool – Ova / cyst / Trophozoite

Pus culture and sensitivity

Blood culture

Serology → [IHA, ELISA] for ALA

LFT

- Serum Alkaline Phosphatase
- Serum Albumin
- Serum Bilirubin – Total / Direct / Indirect
- SGOT / SGPT

9. X-Ray chest – PA view
   X-Ray abdomen – erect AP View
10. USG – Abdomen
11. CT Scan - Abdomen

MASTER CHART

S.No.	Name	Age	Sex	I.P. No.	Treatment
1	Ponnu Samy	48	Male	14756	USG guided PNA under LA
2	Ravindran	45	Male	15637	USG guided PNA under LA
3	Thangaraj	60	Male	21374	USG guided PNA under LA
4	Shanmugam	48	Male	22747	USG guided PNA under LA
5	Meganathan	45	Male	23837	USG guided PNA under LA
6	Ettiyan	44	Male	25050	USG guided PNA under LA
7	Jayavel	40	Male	28654	USG guided PNA under LA
8	Jagadesh	24	Male	3531	USG guided PNA under LA
9	Joseph	42	Male	8953	USG guided PNA under LA
10	Kannan	42	Male	15794	USG guided PNA under LA
11	Vijaya Kumar	30	Male	17827	USG guided PNA under LA
12	Rani	43	Female	12595	USG guided PNA under LA
13	Ramalingam	60	Male	13638	USG guided PNA under LA
14	Ayyanar	60	Male	17717	USG guided PNA under LA
15	Sundaram	58	Male	189711	Lapratomy with External tube drainage Under SA
16	Isac	58	Male	1590	USG guided PNA under LA
17	Jayaraman	63	Male	1629	Conservative
18	Arokiasamy	48	Male	2794	USG guided PNA under LA
19	Jayavel	50	Male	25642	USG guided PNA under LA
20	Surenderan	33	Male	26173	USG guided PNA under LA
21	Chandrasekar	43	Male	27399	Conservative
22	Manokar	32	Male	3297	USG guided PNA under LA
23	Subramni	49	Male	17988	USG guided PNA under LA
24	Murali	56	Male	21292	USG guided PNA under LA
25	Ezhumalai	45	Male	25523	USG guided PNA under LA
26	Sekar	40	Male	3453	USG guided PNA under LA
27	Ramalingam	49	Male	13638	USG guided PNA under LA
28	Venkaiah	45	Male	22210	USG guided PNA under LA

S.No.	Name	Age	Sex	I.P. No.	Treatment
29	Punai	40	Male	25996	USG guided PNA under LA
30	Dhamodaran	26	Male	822	USG guided PNA under LA
31	Kannappan	41	Male	1501	USG guided PNA under LA
32	Babu	45	Male	3603	USG guided PNA under LA
33	Chinna Thambi	37	Male	4514	USG guided PNA under LA
34	Solai	50	Male	10267	Lapratomy with External tube drainage Under SA
35	Perumal	55	Male	10827	USG guided PNA under LA
36	Subramani	44	Male	17988	Conservative
37	Murali	45	Male	21292	Conservative
38	Gunasekar	56	Male	5052	USG guided PNA under LA
39	Selva kumari	30	Female	18342	USG guided PNA under LA
40	Mahalingam	40	Male	33921	Conservative
41	Gurunappan	32	Male	37040	USG guided PNA under LA
42	Kumar	38	Male	37582	Conservative
43	Rajendran	42	Male	883321	USG guided PNA under LA
44	Siva Subramani	49	Male	885184	Conservative
45	Devaki	50	Female	884427	USG guided PNA under LA
46	Thiruvelan	41	Male	883918	USG guided PNA under LA
47	Chinna Thambi	55	Male	885854	USG guided PNA under LA
48	Mani	42	Male	886510	Conservative
49	Sekar	40	Male	886481	Percutaneous Pig Tail catheter drainage Under LA
50	Sivaranjani	35	Female	887293	Conservative
51	Rani	32	Female	883723	Conservative
52	Revathy	29	Female	83432	Conservative
53	Ravindran	40	Male	894146	Conservative
54	Ansari	65	Male	895254	Conservative
55	Kumar	39	Male	96239	Percutaneous Pig Tail catheter drainage Under LA
56	Srinivasan	50	Male	896722	Conservative

S.No.	Name	Age	Sex	I.P. No.	Treatment
57	Sekar	40	Male	3453	Conservative
58	Sridhar	53	Male	897038	Conservative
59	Sekar	40	Male	546920	Conservative
60	Srinivasan	30	Male	896722	Conservative
61	Kumari	36	Female	898390	Conservative
62	Punniyakodi	60	Male	899151	Conservative
63	Mohan	43	Male	899014	Conservative
64	Kumar	51	Male	893412	Conservative
65	Siva	42	Male	400151	Percutaneous Pig Tail catheter drainage Under LA
66	Munuswamy	55	Male	899868	Conservative
67	Valarmathy	65	Female	910956	Percutaneous Pig Tail catheter drainage Under LA
68	Kannayee	55	Female	912726	Conservative
69	Govindhasamy	36	Male	911068	Conservative
70	Sundaram	35	Male	907032	Percutaneous Pig Tail catheter drainage Under LA
71	Mohan	52	Male	904245	USG guided PNA under LA
72	Shankar	32	Male	21363	Lapratomy with External tube drainage Under SA
73	Kumari	55	Female	898390	USG guided PNA under LA
74	Sekar	42	Male	8969201	Conservative
75	Vijayan	72	Male	913326	Conservative
76	Thoplan	60	Male	913363	Lapratomy with External tube drainage Under SA
77	Ramalingam	60	Male	13638	Conservative
78	Ettiyappan	54	Male	25051	Lapratomy with External tube drainage Under SA
79	Selva kumari	30	Female	18342	Conservative
80	Kumar	45	Male	37582	Conservative

USG - Ultrasonogram, PNA-Percutaneous Needle Aspiration, LA-Local Anesthesia, SA-Spinal Anesthesia