# A STUDY ON CLINICAL PRESENTATION, ETIOLOGYAND MANAGEMENT OF LIVER ABSCESS BASED ON DIAGNOSIS BY RADIOLOGICAL IMAGING

# A DISSERTATION SUBMITTED TO THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the

Degree of M.S., (GENERAL SURGERY)

BRANCH – I



# DEPARTMENT OF GENERAL SURGERY STANLEY MEDICAL COLLEGE AND HOSPITAL THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY CHENNAI

**APRIL 2014** 

#### **CERTIFICATE**

This is to certify that the dissertation entitled "A STUDY ON CLINICAL PRESENTATION, ETIOLOGYAND MANAGEMENT OF LIVER ABSCESS BASED ON DIAGNOSIS BY RADIOLOGICAL IMAGING" is the bonafide work done by Dr. NIRMAL J, Post Graduate student (2011 – 2014) in the Department of General Surgery, Government Stanley MedicalCollege and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2014.

Prof. R.V. SURESH, M.S.,

Prof. KAMARAJ M.S.,

Professor of Surgery,

Professor and Head of surgery,

Dept. of General Surgery,

Dept. of General Surgery,

Stanley Medical College,

Stanley Medical College,

Chennai-600001.

Chennai-600001.

#### PROF. S. GEETHA LAKSHMI, M.D., PhD,

The Dean,
Stanley Medical College,
Chennai-600001.

**DECLARATION** 

I, DR. NIRMAL J solemnly declare that this dissertation titled

STUDY ON CLINICAL PRESENTATION, ETIOLOGYAND

MANAGEMENT OF LIVER ABSCESS BASED ON DIAGNOSIS BY

RADIOLOGICAL IMAGING" is a bonafide work done by me

in the Department of General Surgery, Government Stanley Medical

College and Hospital, Chennai under the supervision of my unit

SURESH, M.S., chief **Prof.** R.V with the guidance of

PROF. P. DARWIN, M.S., and my Head of the Department

PROF. KAMARAJ, M.S.

This dissertation is submitted to The Tamilnadu Dr. M.G.R.

Medical University, Chennai in partial fulfillment of the university

regulations for the award of M.S., Degree (General Surgery) Branch - I,

Examination to be held in April 2014.

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Date: December 2013.

DR. NIRMAL J

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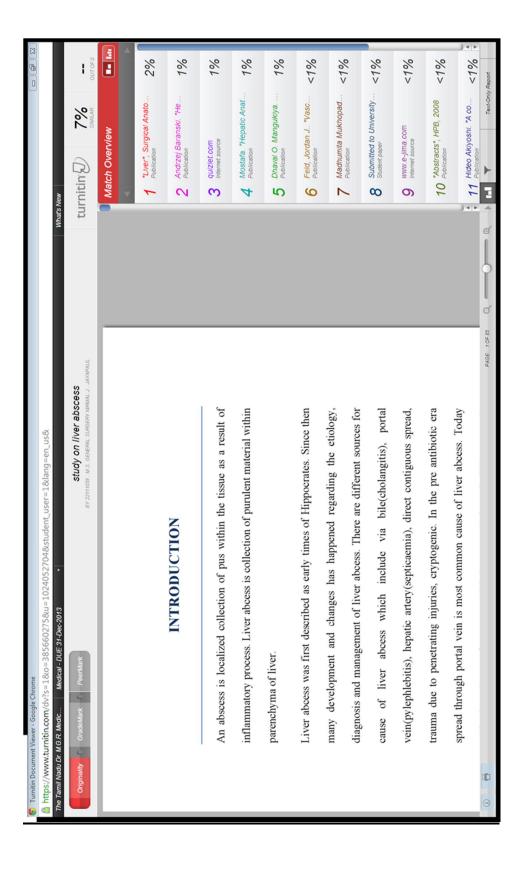
I consider it a privilege to have done this study under the supervision of my beloved Professor and Head of the Department **Prof. Kamaraj**, who has been a source of constant inspiration and encouragement to accomplish this work.

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# **ABSTRACT**

#### TITLE:

A STUDY ON CLINICAL PRESENTATION, ETIOLOGY AND MANAGEMENT OF LIVER ABSCESS BASED ON DIAGNOSIS BY RADIOLOGICAL IMAGING.

#### **AUTHOR:**

Dr. NIRMAL J

#### **KEYWORDS:**

LIVER ABSCESS; HEPATIC ABSCESS; PERCUTANEOUS DRAINAGE; PYOGENIC ABSCESS; AMEBIC ABSCESS

#### **BACKROUND:**

90 cases of liver abscess were included in this study. Patients with classical clinical features were finalized after confirming the diagnosis by ultrasonogram. The treatment modalities were categorized based on number, size and location of liver abscess and also taking mode of presentation into consideration. This study comprises the analysis of age & sex distribution; variations in symptomatology; laboratory investigations and the outcome of various modes of treatment. Interpretations were compared with previous analytical studies.

# **RESULTS & CONCLUSION:**

Final results of the study being tat males have more predominance 6:1 ratio; 5<sup>th</sup> decade is peak incidence. Alcohol as etiology was seen in 80% of cases. Abdomen pain (93%) and tenderness (64%) being commonest clinical feature with classical mode of presentation seen in 78% of cases. Right lobe abscess were seen in 70% of cases. Commonest mode of treatment being aspiration (39%). There were no mortalities observed in tis study.

# **INTRODUCTION**

An abscess is localized collection of pus within the tissue as a result of inflammatory process. Liver abscess is collection of purulent material within parenchyma of liver.

Liver abscess was first described as early times of Hippocrates. Since then many development and changes has happened regarding the etiology, diagnosis and management of liver abscess. There are different sources for cause of liver abscess which include via bile (cholangitis), portal vein (pylephlebitis), hepatic artery (septicemia), direct contiguous spread, trauma due to penetrating injuries, cryptogenic. In the pre antibiotic era spread through portal vein is most common cause of liver abscess. Today with the advent of newer antibiotics spread through biliary tree is commonest cause.

Earlier times laparotomy and drainage was the mainstay of treatment for liver abscess whereas today with advent of better imaging modalities, image guided aspiration or catheter insertion is mainstay of treatment now a days.

This study is conducted to know different presenting symptoms, etiology and outcomes of management of patients.

# HISTORICAL DEVELOPMENTS

HIPPOCRATES	First description of liver abscess in 4000
	BC
OSCHNER	Association of liver abscess with
	pylephlebitis due to appendicitis
MACFADZEAN	Advocated closed aspiration and antibiotics
	for pyogenic liver abscess
WILLIAM OSLER	Discovered amoebae in stools and pus from
	liver
COUNCILMAN AND	pathogenic role of amebae and coined the
LAFLEUR	terms "amebic dysentery" and "amebic liver
	abscess"
JOHN BRIGHT	First description liver abscess in modern
	medicine

# AIMS AND OBJECTIVES

This Analytical study was conducted in Dept. of general surgery, Stanley medical college during the period of year 2011 to 2013.

Following are the objectives of this study:

- To study the incidence of hepatic abscess in our institution
- To study the Age and Sex distribution
- To analyze the clinical features by difference in symptomatology and signs
- To ascertain the microbiology of the disease
- To throw light on the importance of radiological imaging in establishing the diagnosis and plan the management accordingly
- To categorize the treatment modality of the disease

# **ANATOMY OF LIVER**

Liver is the largest solid organ in the body. It weighs around 1.5Kg in an average adult. It has remarkable regenerative properties as compared to other organs in the body.

It lies in the right upper quadrant of the abdomen well protected by the thoracic rib cage. It occupies the right hypochondrium, epigastrium and part of left hypochondrium. It is almost completely surrounded by peritoneal layer called the Glisson's capsule.

# **TOPOGRAPHIC RELATIONS:**

The liver has surface relations and peritoneal attachments.

The liver has two surfaces:

- Diaphragmatic surface
- Visceral surface

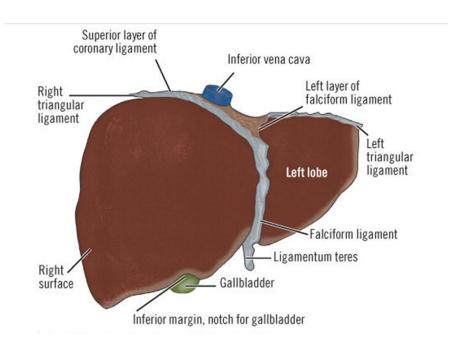
# **DIAPHRAGMATIC SURFACE:**

It has the following portions

**SUPERIOR**: related to diaphragm following organs from right to left: right pleura and lung, pericardium and heart, left pleura and lung. It is mostly covered by glisson's capsule except in bare area of liver.

**POSTERIOR**: related to the diaphragm and lower ribs. It contains the greater part of the bare area and the sulcus of the inferior vena cava

ANTERIOR: related to the diaphragm and costal margin, xiphoid process and the sixth to tenth ribs



**RIGHT**: related to the diaphragm and the seventh to eleventh ribs

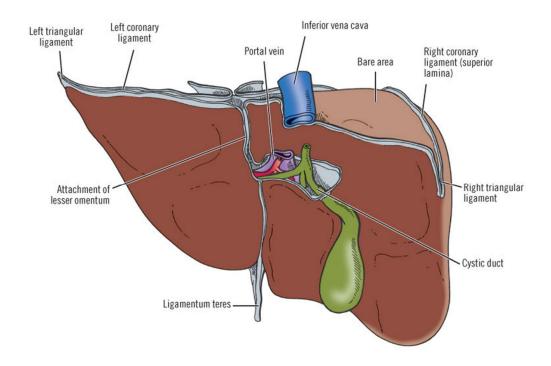
The two surfaces are separated by inferior border of liver which has two notches to accommodate the ligamentum eres and space for gall bladder.

# **VISCERAL SURFACE:**

This surface is distinctively concave as compared to diaphragmatic surface and has impressions by organs.

It is related to following organs from right to left.

- The hepatic flexure of the transverse colon is related to anterior  $1/3^{\rm rd}$  of the right lobe and ends in quadrate lobe.
- Renal impression is behind colic impression made by the right kidney and adrenal gland.
- gallbladder lies in a fossa just beneath the anterior inferior border of the liver
- first and second portions of the duodenum lie in depression to left of the gallbladder



# **PORTA HEPATIS:**

This is seen in the visceral surface of the liver. The portahepatis acts as a gate way to the liver. It admits portal vein, Hepatic artery and the plexus of Nerves and lets out the right and left Hepatic ducts and lypmhatics. These structures are arranged in shape of "H". The right limb of the "H" bordering the porta is formed anteriorly by the fossa for the gallbladder and posteriorly by the fossa for the inferior vena cava. The left limb is formed anteriorly by the fissure for the round ligament and posteriorly by the fissure for the ligamentumvenosum. The portahepatis forms the crossbar of the "H."

# **PERITONEAL ATTACHMENTS:**

- Falciform ligament
- Superior coronary ligament
- Inferior coronary ligament
- Right triangular ligament
- Left triangular ligament
- Lesser omentum

#### **FALCIFORM LIGAMENT:**

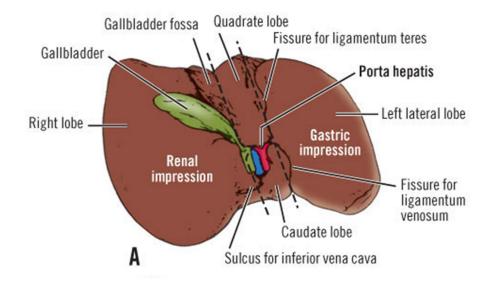
It forms attachment between superior surface of left lobe of liver. It marks the fissure between the medial and lateral segments of the left lobe. The falciform ligament contains the remnant of the proximal part of the left umbilical vein, the round ligament of the liver or ligamentumteres.

#### **CORONARY LIGAMENTS:**

Reflection of peritoneum from diaphragm to bare area of liver anteriorly forms the superior coronary ligament and posteriorly, inferior coronary ligament.

#### **LEFT TRIANGULAR LIGAMENTS:**

The anterior and posterior layer of coronary ligaments meets to form the left triangular ligament which is in close relation to abdominal esophagus

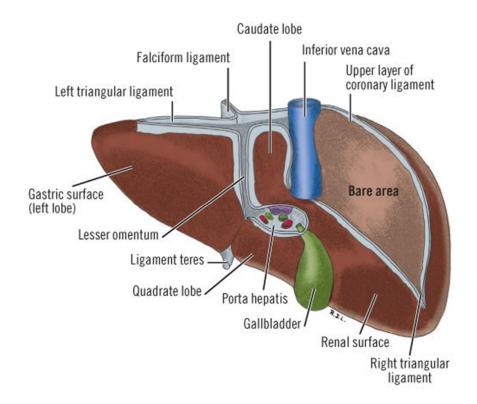


#### **RIGHT TRIANGULAR LIGAMENTS:**

The anterior and posterior layer of coronary ligaments meet on the right to form the right triangular ligament.

#### **LESSER OMENTUM:**

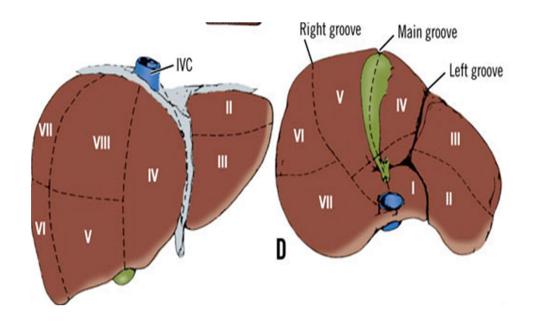
This forms the attachment from the visceral surface of liver to stomach and duodenum. It consists of hepatogastric and the hepatoduodenal ligament. The hepatogastric ligament extends from the portahepatis to the lesser curvature of the stomach and the abdominal esophagus. The hepatoduodenal ligament extends between the liver and the first portion of the duodenum and is continuous with the right border of the hepatogastric ligament. It contains the common bile duct, hepatic artery, and portal vein as well as the hepatic plexus and lymph nodes.



# **SEGMENTAL ANATOMY:**

The liver is morphologically divided into left and right lobes anatomically by an apparent plane of division passing through the falciform ligament, round ligament and ligamentum venosum. This division does not correspond to anatomical division of lobes which corresponds to internal distribution of bile ducts and blood vessels. Morphologically the right lobe is much larger compared to left lobe.

Segmental or surgical anatomy was first described by cantlie in 1898 and revised by Couinad in 1957. In this the liver is divided into equal lobes by a median fissure that passes through fossa of IVC above and fossa of gall bladder below. The liver is divided into 8 segments.

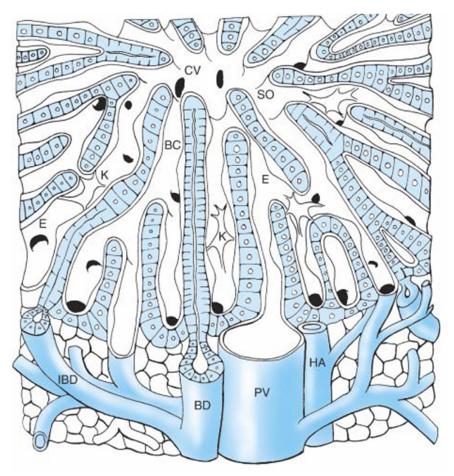


The left lobe consists of segments II to IV and right lobe consists of segments V to VIII. The **Caudate lobe** is divided by the interlobar plane. It has separate bile duct, arteries and portal veins arising from both the lobes and drained by hepatic veins directly into vena cava.

# **HEPATIC MICROARCHITECTURE:**

The liver is divided into lobes and in turn into lobules. The hepatocytes are arranged in plates within the parenchyma. Hepatic lobule is composed of central vein in the middle and surrounded at the periphery are portal triads. Portal triads contain branches of portal vein, hepatic artery and bile duct.

Portal lobule consists of adjoining parts of 3 hepatic lobule and bile from which is drained into biliary canaculi. Between the plates of hepatocytes are spaces forming the sinusoids. The **hepatic macrophages or Kupffer**cells are found lining around the hepatic sinusoids. Between the sinusoids and the basal sides of the plates of hepatic cells is the perisinusoidal space of Disse.



The hepatic acinus. BC = bile cannaliculus; BD = bile duct; CV = central vein; E = endothelial cell; HA = hepatic artery; H = hepatocyte; K = Kupffer cell; PV = portal vein.

# **BLOOD SUPPLY:**

The liver has dual blood supply from hepatic artery (20%) and portal vein (80%). At the portahepatis the hepatic artery and portal vein divide into left and right branches. Further they divide into lobar segmental and interlobular branches which forms the portal triads. The mixed blood supply drains into the hepatic sinusoids which in turn into the central vein.

#### **VENOUS DRAINAGE:**

Blood from central vein join together as tributaries of hepatic veins which finally form the right, middle and left hepatic veins. The three major veins drain into the inferior vena cava.

#### LYMPHATIC DRAINAGE:

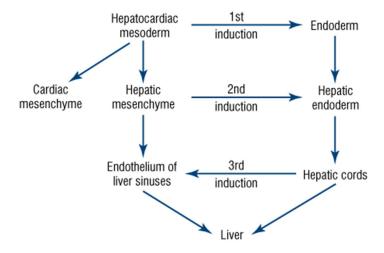
The perivascular space of disse is the source of lymph from the liver. The lymphatics are divided into superficial and deep. Superficial lymphatics lie near the surface just below the glisson's capsule. These drain into the hepatic, caval, paracardial and coeliac lymph node groups. The deep group drain into the phrenic nodes and around the hepatic veins and vena cava and also the nodes of portahepatis.

#### **NERVE SUPPLY:**

The liver receives both sympathetic and parasympathetic nerve supply. Fibers from the anterior hepatic plexus around the hepatic artery and the posterior hepatic plexus around the portal vein reach the liver. The sympathetic fibers arise from thoracic spinal cord segments 7 to 10. The parasympathetic efferent fibers arise from the hepatic division of the anterior and posterior vagal trunks.

# **EMBRYOLOGY:**

The liver develops as hepatic diverticulum from the developing foregut. This hepatic diverticulum extends into the septum transversum and expands the ventral mesentery. This divides to form the large cranial part which gives rise to hepatocytes and epithelial lining of intrahepatic part ofbiliary apparatus.



# **FUNCTIONS OF LIVER**

#### **SYNTHESIS:**

- 1. The liver synthesizes the plasma proteins namely the albumin immunoglobulins.
- 2. The clotting factors namely fibrinogen, prothrombin factors 7, 9 and 10.
- 3. Acute phase proteins, steroid and hormone binding proteins
- 4. Formation and secretion of bile

#### **METABOLISM:**

- 1. Carbohydrate metabolism modulate glucose
- 2. Protein metabolism modulate free amino acids
- 3. Fat metabolism modulate free fatty acids
- 4. Urea metabolism converts ammonia to urea

#### **DETOXIFICATION:**

Toxins, drugs, steroids and other hormones

# **IMMUNITY:**

Kupffer cells forms part of reticuloendothelial systems and acts by neutralizing pathogens

# **STORAGE:**

Storage of vitamins, iron, carbohydrate in form of glycogen.

# **REVIEW OF LITERATURE**

Liver abscess is broadly classified as

- 1. Pyogenic abscess
- 2. Amoebic abscess

# **PYOGENIC LIVER ABCSESS:**

Pyogenic liver abscess is caused by bacterial infection.

# **INCIDENCE:**

The incidence of pyogenic liver abscess was 5 - 13 per 1 lakh. But recently there has been an increase in incidence to 15 or upto 22 cases per 1 lakh patients. This has been attributed to better investigative modalities and aggressive treatment in hepatobiliary and pancreatic cancers.

# **ETIOLOGY:**

Liver abscess occurs when hepatic clearance mechanisms fail or when the mechanisms are overloaded. The kupffer cells act as clearing mechanisms for the microorganism entering liver.

Following are the modes of sources of infection

- 1. Bile ducts ascending cholangitis
- 2. Portal vein pylephlebitis from appendicitis or diverticulitis
- 3. Direct extension from contiguous areas
- 4. Trauma Penetrating or blunt injury abdomen
- 5. Hepatic artery systemic septicemia
- 6. Cryptogenic

Cholangitis accounts for upto 35 - 40 % of all pyogenic liver abscesses and is due to underlying malignancy. Obstruction to biliary tree is present in 50% of patients. It may be due to stone pathology or stricture. Procedures like cholanging raphy, PTC and stenting increase the predisposition.

Spread through portal vein in form of pylephlebitis accounts for 20%.

In the pre antibiotic era spread through portal vein from intestinal pathologies like appendicitis accounted for 34%. Currently appendicitis accounts for only 2%. Colonic malignancy, intraabdominal abscess and diverticulitis are other common causes of pyogenic liver abscess.

Contiguous spread can occur from perforated ulcers, gangrenous cholecystitis and subphrenic abscess.

Trauma in form of penetrating or blunt injury can cause parenchymal necrosis and clot which creates a nidus for infection and abscess formation.

Septicemia via hepatic artery is responsible for 12% of pyogenic liver abscess. IV drug abuse is common cause but other sources are infection in heart, lungs, kidneys etc.

Cryptogenic abscess account for  $10-45\ \%$  in which the etiology is unknown.

# PREDISPOSING FACTORS:

Children	Adults
Chronic granulomatous disease	Diabetes mellitus
Complement deficiencies	Cirrhosis
Leukemia	Chronic pancreatitis
Malignancy	Peptic ulcer disease
Sickle cell anemia	Inflammatory bowel disease
Polycystic liver disease	Jaundice
Congenital hepatic fibrosis	Pyelonephritis
Posttransplant liver failure	Malignancy
Necrotizing enterocolitis	Leukemia and lymphoma
Chemotherapy and steroid therapy	Chemotherapy and steroid therapy
Acquired immunodeficiency syndrome	Acquired immunodeficiency syndrome

# **PATHOLOGY:**

The number, size and location of the hepatic abscesses can localize the source of infection. Generally 60% of pyogenic abscesses occur in the right lobe, 14% involving left lobe and 22% with bilobar disease. Traumatic, portal and cryptogenic abscess are usually solitary and large whereas biliary and arterial are multiple and small. Portal sources are usually in right lobe due phasic flow from superior mesenteric vein.

# **BACTERIOLOGY:**

It is established by aspiration of abscess and positive cultures are obtained in 80 - 90 % of cases. Blood cultures are positive in 50 - 60 % of cases. Other organisms include candida seen in cancer patients and also due to use broad spectrum antibiotics. Fungal cultures are positive in 22% of cases. Most common species are the e.coli, klebsiella, enterococci and pseudomonas. Other anaerobic species include bacteroides and fusobacterium species. Mycobacterium tuberculosis is seen in AIDS syndrome complex.

Category of Organism	% of Patients
Gram-Negative Aerobes	50-70
Escherichia coli	35-45
Klebsiella	18
Proteus	10
Enterobacter	15
Serratia	Rare
Morganella	Rare
Acinetobacter	Rare
Gram-Positive Aerobes	55
Stretococcal species	20
Enteroccocus faecalis	10
β-Streptococci	5
α-Streptococci	5
Staphylococcal species	15
Anaerobes	40-50
Bacteroides species	24
Bacteroides fragilis	15
Fusobacterium	10
Peptostreptococcus	10
Clostridium	5
Actinomyces	Rare
Fungal	26
Sterile	7

# **CLINICAL PRESENTATION:**

Most common symptom is fever (80%) followed by abdominal pain (50%). Hepatomegaly is seen in 50% of patients; jaundice 50% and tenderness in 65% of patients.

% of Pyogenic Abscesses
83
60
55
50
50
37
34
30
17
12
52
40
31
25
25
20

# LABORATORY EVALUATION:

More than 60% of patients have elevated bilirubin, transaminases and alkaline phosphatases. Prolonged prothrombin time, hypoalbuminemia and anemia are seen in 60-75% of patients.

LABORATORY DATA	
Increased alkaline phosphatase	87
WBC count >10,000/mm <sup>3</sup>	71
Albumin <3 g/dL	55
Hematocrit <36%	53
Bilirubin >2 mg/dL	24

# **DIAGNOSIS:**

Diagnosis is usually done by radiological imaging with ultrasound and contrast enhanced CT

# **TREATMENT:**

Treatment of liver abscess includes antibiotics, drainage of abscess and concurrent treatment of the source of infection. Multiple small abscesses and miliary fungal abscesses require only IV antibiotics without a drainage procedure.

# **ANTIBIOTICS:**

Antibiotic regimens include clindamycin, aminoglycoside and vancomycin / ampicillin. Flouroquinolones can be used instead of

aminoglycosides and metronidazole in place of clindamycin especially if an amoebic source is suspected.

Single agent regimen such as imipenem-cilastatin, ticarcillin-clavulanate or piperacillin-tazobactum can be used. Treatment is usually given for 4-6 weeks but current recommendations are only for two weeks. Systemic fungal therapy is preferred for fungal abscess.

# ASPIRATION AND PERCUTANEOUS AND

#### **CATHETER DRAINAGE:**

Needle aspiration and percutaneous catheter drainage has become the mainstay of treatment of liver abscess. Both have similar mortality rates but recurrence rates are higher in case of aspiration. Complication rates are higher in case of catheter drainage. Randomized controlled trials show a 60% success rate with needle aspiration and a 100% success rate with catheter drainage.

Following are conditions in which percutaneous drainage is not appropriate:

- 1. Multiple large abscess
- 2. Source of abscess is intra-abdominal that requires surgery

- 3. Abscess of unknown etiology
- 4. Ascites
- 5. Abscess requiring Trans pleural drainage.

#### **SURGICAL DRAINAGE:**

Surgical drainage of liver abscess has reduced currently with incidence of newer antibiotics and image guided aspiration and catheter insertion. Extra peritoneal drainage via the 12<sup>th</sup> rib resection was practiced earlier to avoid contamination to peritoneal cavity. Currently Trans peritoneal approach is considered to be superior. Following are the advantages:

- 1. Gain complete access and exposure of entire liver
- 2. Source can be dealt with
- 3. Evaluation of biliary tree in form CBD exploration

Surgical drainage is currently reserved only for patients with failed non operative treatment, multiple macroscopic abscess, concomitant ascites and those presenting with rupture and peritonitis.

# **COMPLICATIONS:**

Incidence of complications in pyogenic liver abscess can be upto 40%. These include:

- 1. Pleural effusion, pneumonia and empyema
- 2. Intraperitoneal rupture and peritonitis
- 3. Perihepatic collection
- 4. Hemobilia; hepatic vein thrombosis

# FACTORS ASSCOCIATED WITH POOR PROGNOSIS:

Age >70 years	WBC count >20.000/mm <sup>3</sup>
Diabetes mellitus	Increasing bilirubin
Associated malignancy	Increasing SGOT
Biliary etiology	Albumin <2 g/dL
Multiple abscesses	Aerobic abscess
Septicemia	Significant complication
Polymicrobial bacteremia	

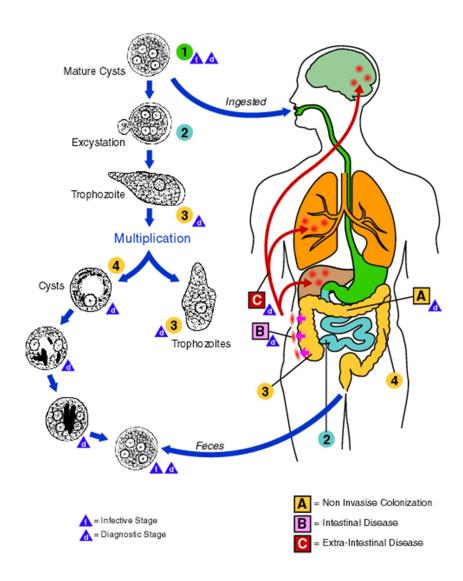
# **AMOEBIC LIVER ABCESS:**

Amoebic liver abscess is caused by a parasitic protozoan namely entamoebahistolytica. Two species of entamoeba are found to pathologic to man E.dispar and E.histolytica. E. dispar is associated with asymptomatic carrier state while E. histolytica is responsible for all forms of invasive disease.

#### **INCIDENCE:**

Worldwide incidence of carriers of both species is 500 million. Incidence of active invasive disease is seen in 50 million people. Annual death census worldwide is around 50 thousand to 1 lakh annually. High risk groups include immigrants, homosexual, immunosuppressed and tourists. Age wise incidence has a bimodal peak at 2-3 yrs. And>40 yrs. with a fatality rates of 20% and 70%. Unsanitary conditions and low socioeconomic status are predisposing factors.

# LIFECYCLE OF E.HISTOLYTICA:



# **ETIOPATHOGENESIS:**

Life cycle involves cysts and invasive trophozoites. Following feco-oral transmission, cyst passes through the stomach and intestine. After digestiontrophozoite is released and multiplies in the intestine. If invasive disease does not ensue patient becomes an asymptomatic

carrier or develops amebic dysentery. Amoebae can invade the intestinal mucosa, traverse through lymphatics, veins to reach the liver parenchyma causing abscess. "Anchovy sauce" appearance is due to liquefied hepatic parenchyma with blood forming an abscess.

90% of entamoeba infected becomes asymptomatic carriers due to organism colonized in the colon. Trophozoites are found in colon and feces of humans which become carriers. Incubation period is 1-4 weeks.

Invasive amoebiasis can vary from amoebic dysentery to metastatic abscess most common form being amebic colitis. Amoebic ulcers with undermined edges are seen in colonic mucosa. Commonest extra intestinal site affected is the liver seen in 50% of invasive disease. Usually start as multiple small abscesses which coalesce to form single large abscess.

#### **CLINICAL PRESENTATION:**

Amoebic liver abscess occurs commonly in young adult males.

Usual presentation of amebic abscess is not with concurrent colitis.

History of dysentery is present within 1 yr. duration. 80% of patients with amoebic abscess develop symptoms within 2-4 weeks.

SYMPTOM	
Pain	90
Fever	87
Nausea and vomiting	85
Anorexia	50
Weight loss	45
Malaise	25
Diarrhea	25
Cough or pleurisy	25
Pruritus	<1
SIGN	
Hepatomegaly	85
Right upper quadrant tenderness	84
Pleural effusion or rub	40
Right upper quadrant mass	12
Ascites	10
Jaundice	5

## LABORATORY EVALUATION:

Derangement of liver functions is seen in > 50% of patients. Jaundice is uncommon but patient may have elevated prothrombin time. Amebic abscesses are associated with positive stool sample in 50% of cases.

Increased alkaline phosphatase	80
WBC count >10,000/mm <sup>3</sup>	70
Hematocrit <36%	49
Albumin <3 g/dL	44
Bilirubin >2 mg/dL	10

#### SEROLOGY AND DIAGNOSTIC ASPIRATION:

Patients with invasive colitis have positive serum antibodies in 85% of times and 99% of times in cases of liver abscess. Biopsy taken from edge of an ulcer or wall of abscess reveal trophozoites with periodic acid Schiff stain. Serology report can be obtained within 24-48 hrs. Diagnostic aspiration is needed when serology is negative. Aspirate is negative for gram stain and cultures. Amoebae are recovered in 30 – 90% of times. Diagnosis is attempted by a combination of stool testing, serology and colonoscopic biopsy of intestinal lesions. Antigen detection in stool sample by PCR is better investigation.

#### TREATMENT:

After the advent of metronidazoles in 1960, antibiotics are the mainstay of treatment in amebic liver abscess.

#### **ANTIBIOTICS:**

Noninvasive amebiasis can be treated with paromomycin.

Nitroimidazoles (metronidazole) are the mainstay of treatment. Longer half-life drugs (tinidazole, secnidazole, ornidazole) are better.

Metronidazole reaches high concentration in intestines and liver.

Response to treatment is seen by third day of treatment. Cure rate is 85% at 5 days and 95% at 10 days. 40-60% of cases parasites persist in intestine hence treatment should be followed with paromomycin or diloxanatefuroate.

#### THERAPEUTIC ASPIRATION:

Randomized controlled trials with treatment with antibiotics alone vs. therapeutic aspiration along with antibiotics demonstrated no significant difference in the outcome. Antibiotics alone is enough in uncomplicated abscess even upto the size of 10cm located in the right lobe. Drainage may be considered in patients with no clinical response with 1 week of drug therapy with a high risk of rupture or coinfectionwith amoebic liver abscess.

#### **SURGICAL DRAINAGE:**

Surgical drainage of abscess is reserved only for patients with complications.

#### **PERCUTANEOUS:**

It is reserved only for treating peritoneal, pulmonary or pericardial complications.

#### **SURGICAL:**

Indication for surgery is managing abscess that has failed conservative management. It is indicated in ruptured abscess with complications and in sepsis due to secondary infection.

### **COMPLICATIONS:**

Rupture from amebic abscess into the pericardium, pleural cavity or peritoneal cavity. Incidence of ruptured abscess is 2-17% of patients with mortality rates 12% - 50%.

Peritonitis can occur due to rupture as seen in (78%) or due to perforated amebic colitis (22%). Usually free rupture is rare as it gets walled off by adhesions and omentum.

Commonest complication is thoracic amebiasis which includes empyema, bronchohepatic fistulas, and pleuropulmonary abscess. Next commonest is pericardial amebiasis; acute pericarditis with tamponade. Rupture of abscess cavity into pleural space is treated by tube thoracostomy. X-ray might show white out lung. Rupture of abscess cavity into bronchi may result in cough with expectoration of copious brown sputum.

Left lobe abscess can open into the pericardium resulting cardiac tamponade, pericardial effusion, pericardial thickening. In which case aspiration of pericardial collection, liver abscess aspiration and systemic antibiotics is treatment of choice.

### **PROGNOSIS:**

Following are factors with poor outcome of disease.

Increased age
Increased bilirubin level
Pulmonary involvement
Rupture or extension
Late presentation

### **DIFFERENCE IN CLINICAL FEATURES:**

Following are some of distinguishing features between pyogenic and amoebic liver abscess

Amebic	Pyogenic
Age <50 years	Age >50 years
Male:female ratio 10:1	Male:female ratio 1:1
Hispanic descent	No ethnic predisposition
Recent travel to endemic area	Malignancy
Pulmonary dysfunction	High fevers
Abdominal pain	Pruritus
Diarrhea	Jaundice
Abdominal tenderness	Septic shock
Hepatomegaly	Palpable mass

## **RADIOLOGICAL IMAGING:**

Radiological imaging is the best modality of investigation and confirming the diagnosis of liver abscess. It includes x-ray, ultra sonogram, and computerized tomography. Treatment modality can be planned based on the imaging.

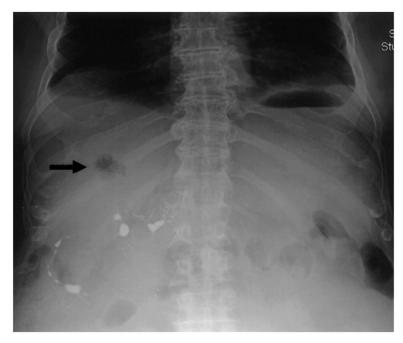
# **RADIOGRAPH:**

In pyogenic liver abscess 50% of patients have abnormal chest radiographs.



Barium enema performed on a patient with a large gas-filled abscess

In amoebic liver abscess chest radiograph may show chest infiltrates, pleural effusion or an elevated hemi diaphragm. It includes right sided pleural effusion, elevated right hemi diaphragm; right lower lobe atelectasis. Abdominal radiographs may show hepatomegaly. Air fluid level may be seen in cavity in of gas forming organisms or portal venous gas if pyelephebitis.

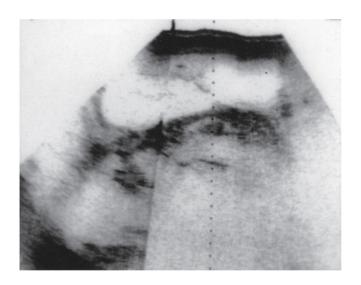


Abnormal collection of air in the right upper quadrant consistent with pyogenicliver abscess

### **ULTRASONOGRAM:**

Ultrasound is the most efficient and cost effective investigation in diagnosing hepatic abscess. It can distinguish solid and cystic lesions.

Ultrasound has sensitivity of 80-95 %. Only limiting factor is that sensitivity goes down in morbidly obese patients and those under ribs or in an inhomogenous liver.



Amoebic liver abscess are usually single in number about 75-80% mostly present in right lobe. About 10% are found in left lobe. Caudate lobe can have upto 6%. Only 40% of cases have typical sonographic appearance. Resolution of abscess radiologically takes about 70% persisting for more than 6 months. Complete radiological resolution may result in small residual cavity resembling a simple cyst of liver.



Ultrasound typical sonographic appearance of amoebic liver abscess

### **COMPUTERISED TOMOGRAPHY**

CAT scan of the abdomen is more sensitive than ultra-sonogram which is about 95-100%. Usually abscess cavity show lower attenuation than the surrounding liver parenchyma.



CT demonstrating multiple pyogenic abscesses



CT demonstrating solitary liver abscess

On administration of intravenous contrast wall of abscess cavity may show typical peripheral rim enhancement. Minimal size of lesion that is detectable by CT scan is 0.5cm.



Left lobe hepatic abscess



Multiple small abscesses

### **CAVITOGRAM:**

In this technique contrast material is injected into an abscess cavity and x-rays are taken. Contrast can be injected by malecot's or pigtail catheter used for drainage. This method is mainly used for follow-up and to know reduction in cavity size after resorption. This method is outdated is not use now.





Serial cavitograms showing decrease in size of cavity

### MATERIALS AND METHODS

#### **AIMS AND OBJECTIVES:**

To study the various forms of clinical presentation, etiology and different modalities of treatment of liver abscess, their outcome based on diagnosis by radiological imaging.

#### PLACE OF STUDY:

Department of general surgery, Stanley medical college

#### **DURATION OF STUDY:**

January 2012 to June 2013

### **SAMPLE SIZE:**

90 patients were included in the study during the above mentioned period

#### **PATIENT SELECTION:**

Patients are clinically diagnosed as liver abscess and are proven by ultra-sonogram

#### **INCLUSION CRITERIA:**

- Clinical history suggestive of liver abscess right hypochondrial pain, fever, dysentery etc.
- 2. Examination findings enlarged and tender liver
- 3. Radiograph showing raised right dome of diaphragm
- 4. Ultra sonogram evidence of liver abscess.
- 5. Age criteria -> 10 yrs

#### **EXCLUSION CRITERIA:**

- 1. Ultra sonogram is normal
- 2. Ultra sonogram not conclusive as liver abscess or doubtful
- 3. Radiological differential diagnosis of SOL liver
- 4. Previously treated recurrent liver abscess

- 5. Patients with hematological abnormalities
- 6. With associated decompensated liver disease
- 7. Pregnant female patients with liver abscess

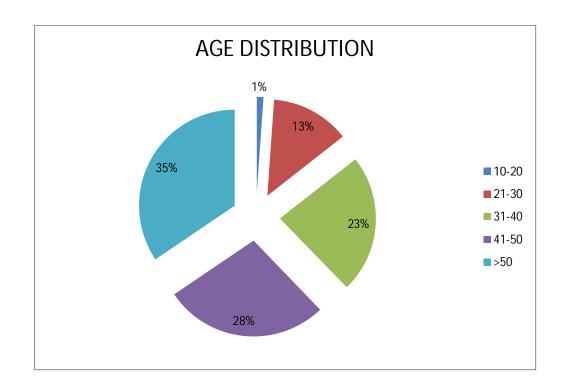
A detailed history is obtained from the patients followed by clinical examinationand basic blood investigations are done. These patients are subjected to ultra-sonogram and those confirmed to be liver abscess are finalized in the study. Cases are filtered by exclusion criteria. Treatment modalities are planned based on size, number and location of liver abscess.

# **RESULTS OF STUDY**

### **TABLES:**

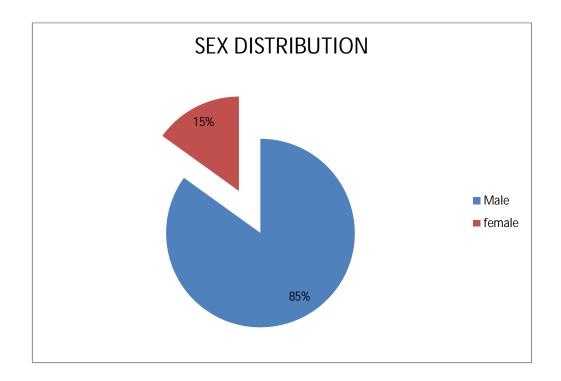
#### **AGE DISTRIBUTION**

Age in years	No of cases	Percentage
10-20	1	1.1
21-30	12	13.3
31-40	21	23.3
41-50	25	27.7
>50	31	34.4



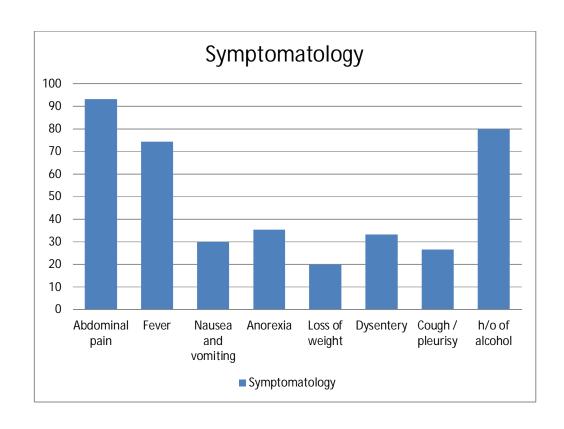
### **SEX DISTRIBUTION**

Sex	No of cases	Percentage
Male	76	84.4
female	14	15



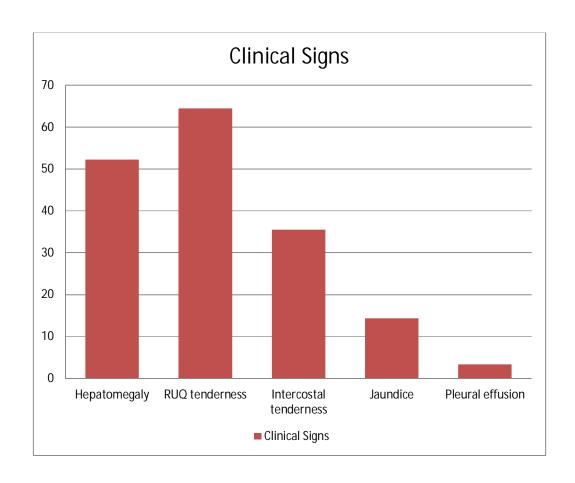
### **CLINICAL PRESENTATION**

Symptoms	No of cases	Percentage
Abdominal pain	84	93.3
Fever	67	74.4
Nausea and vomiting	27	30
Anorexia	32	35.5
Loss of weight	18	20
Dysentery	30	33.3
Cough / pleurisy	24	26.6
h/o of alcohol	72	80



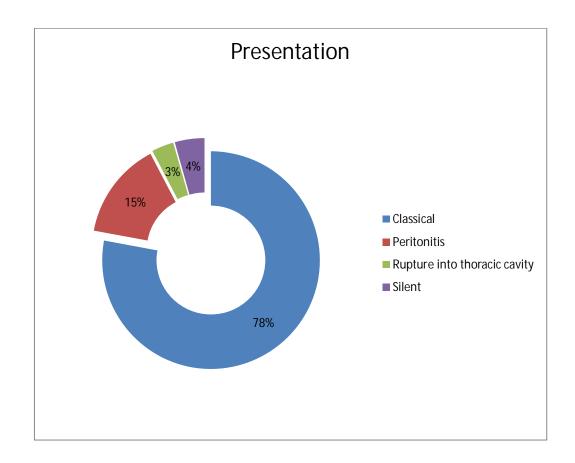
### **EXAMINATION FINDINGS**

Clinical Signs	No of cases	Percentage
Hepatomegaly	47	52.2
RUQ tenderness	58	64.4
Intercostal tenderness	32	35.5
Jaundice	13	14.4
Pleural effusion	3	3.3



### MODES OF PRESENTATION

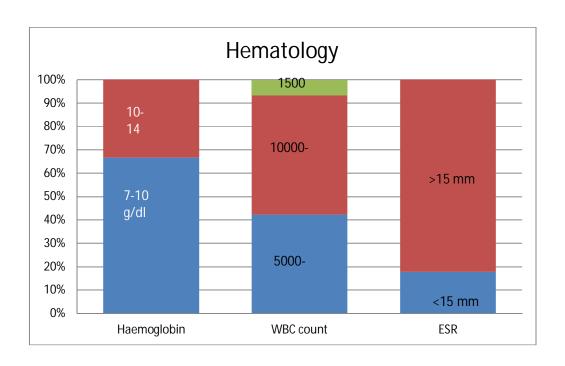
Presentation	No of cases	Percentage
Classical	70	77.7
Peritonitis	13	14.4
Rupture into thoracic cavity	3	3.3
Silent	4	4.4



### LABORATORY INVESTIGATIONS

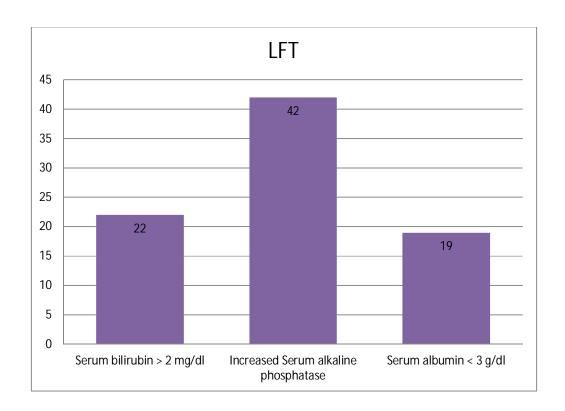
# **COMPLETE BLOOD COUNT:**

Investigation	Value	No of cases	Percentage
Haemoglobin	7-10 gms/dl	60	66.6
	10-14 gms/dl	30	33.3
WBC count	5000-10000 cells/cu.mm	38	42.2
	10000-15000 Cells/cu.mm	46	51.1
	>15,000 cells/cu.mm	6	6.6
ESR	<15 mm	16	17.7
	>15 mm	74	82.2



### **LIVER FUNCTION TEST:**

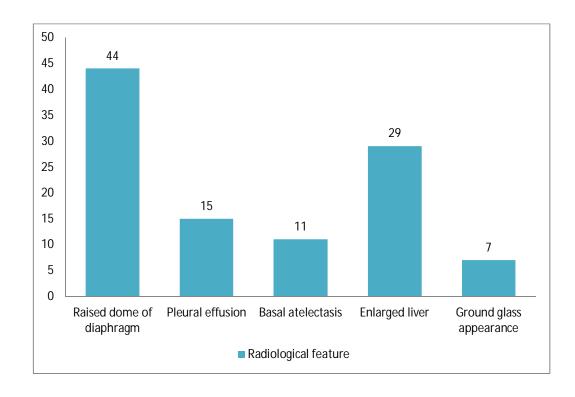
Investigation	No of cases	Percentage
Serum bilirubin > 2 mg/dl	22	24.4
Increased Serum alkaline phosphatase	42	46.6
Serum albumin < 3 g/dl	19	21.1



### RADIOLOGICAL INVESTIGATION

CHEST AND ABDOMEN RADIOGRAPH:

Radiological feature	No of cases	Percentage
Raised dome of diaphragm	44	48.8
Pleural effusion	15	16.6
Basal atelectasis	11	12.2
Enlarged liver	29	32.2
Ground glass appearance	7	7.7

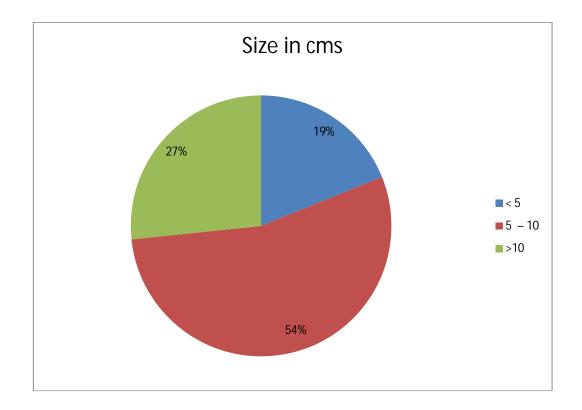


### **ULTRASONOGRAM ABDOMEN:**

In this study all patients were subjected to B mode 2D ultra sonogram.

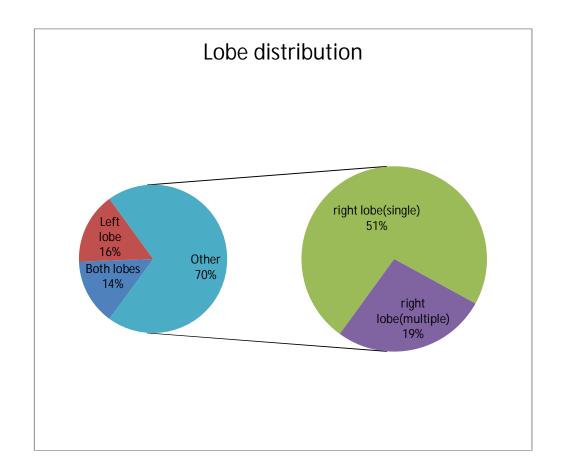
**SIZE OF ABSCESS IN USG:** 

Size in cms	No of cases	Percentage
< 5	17	19
5 - 10	49	54.4
>10	24	26.6



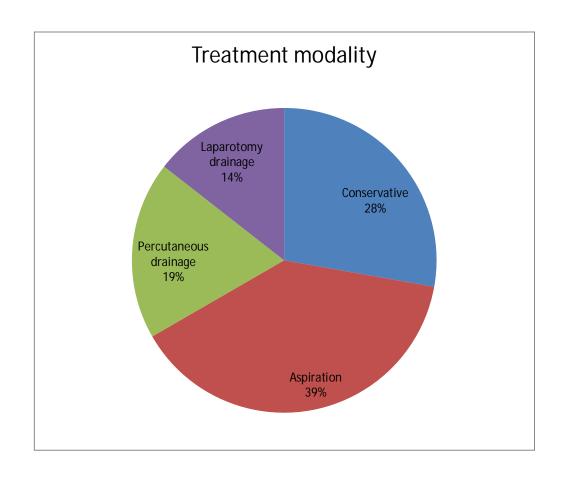
### **DISTRIBUTION OF ABSCESS:**

Right lobe		Left	lobe	Both lobes		
63(7	(0%)	14(16%)		13(14%)		
Single	Multiple	Single	Multiple			
46(51%)	17(19%)	14(15%)	0			



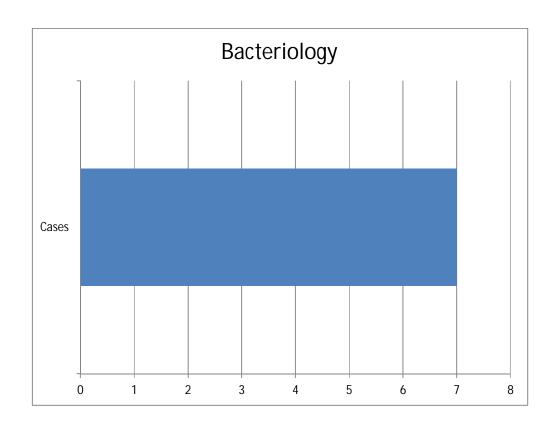
TREATMENT MODALITY

Mode of treatment	No of cases	Percentage
Conservative	25	28
Aspiration	35	39
Percutaneous	17	19
drainage		
Laparotomy drainage	13	14



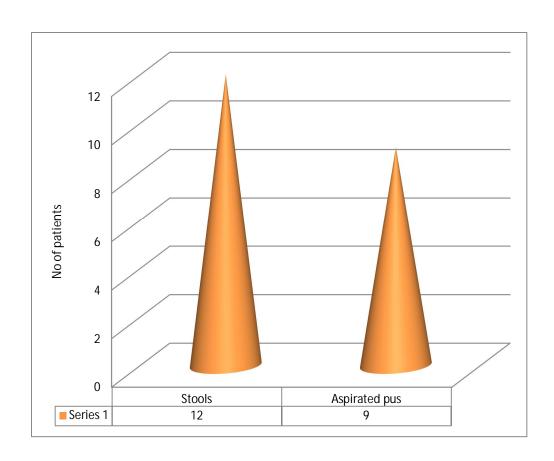
## **BACTERIOLOGICAL STUDY**

No of persons found to have bacterial growth	Organisms isolated
7(8%)	E.coli
	Klebsiella
	Pseudomonas



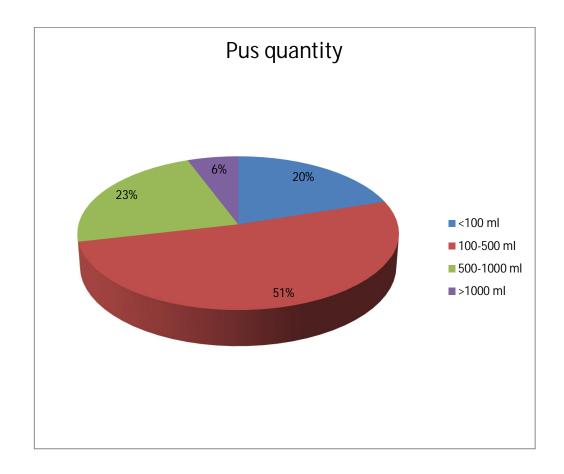
### TROPHOZOITE AND CYST OF E.HISTOLYTICA ISOLATED

Stools	Aspirated pus		
12(13%)	9(10%)		



QUANTITY OF PUS ASPIRATED

Quantity	No of cases	Percentage
<100 ml	7	8
100-500 ml	18	20
500-1000 ml	8	9
>1000 ml	2	2



#### **DISCUSSION**

The condition of hepatic abscess and its grave prognosis were known in ancient times to **Hippocrates**(460 BC – 370 BC) and **Celsus** (53 BC – 7 AD) Hippocrates was able to distinguish 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 Murray1938 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. Chuttaniet al1963 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 mortality rates approaching 100%.

Reports of successful medical management with or without aspiration describe case fatality rates as low as 6%.

### **CURRENT STUDY:**

### **INCIDENCE:**

In our study out of 11250cases admitted in our hospital from January 2012 to June 2013 we reported 90 cases of liver abscess.

Incidence of hepatic abscess is 1.2 %

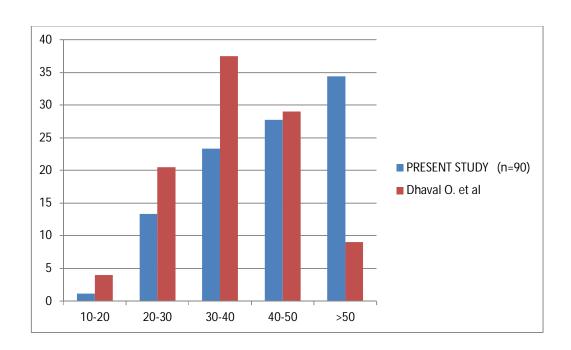
### **AGE:**

Mean age of incidence being 44 while the peak age incidence being 5<sup>th</sup> decade.**Dhaval O. et al** shows the mean age was 35 yrs.**Chang AC et al** has mean age 70.2 years

In our study peak age incidence was noticed in 5<sup>th</sup> decade followed by Fourth 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.

### **COMPARITIVE STUDY:**

AGE in yrs	PRESENT STUDY (n=90)	Dhaval O. et al (n=400)
10-20	1	16
20-30	12	82
30-40	21	150
40-50	25	116
>50	31	36

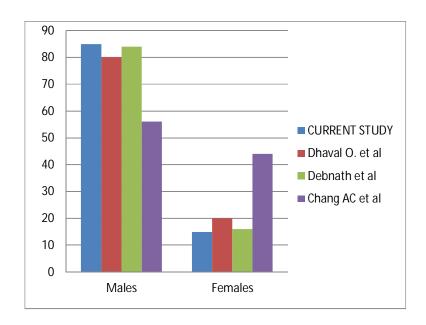


### **SEX:**

In our study incidence of liver abscess in males is 85% which also correlates with history of alcoholism present in 80% of cases. This in comparison to **Dhaval O. et al** which has male incidence of 80% andfemales 20%. **Debnath et al** shows distribution of males 84% and females 16%. **Chang AC et al** shows males 56% males and 44% females.

#### **COMPARISON:**

SEX	CURRENT	Dhaval	О.	Debnath	et	Chang	AC
	STUDY	et al		al		et al	
Males	85%	80%		84%		56%	
Females	15%	20%		16%		44%	



Highest incidence of liver abscess in males 96% in our study has been attributed to alcoholism, Present study H/o alcoholism was present in 80% Cases. This correlates with the study of Oschner&Debakeywhich predispose to hepatitis. Alcohol produces hepatocellular damage and may make it prone to develop hepatic abscess – **Sheila – Sherlock.** 

#### **CLINICAL FEATURES:**

In our study cases have varied presentation and clinical features. As our observation the most common symptom being abdominal pain 93% followed by fever 74%. Amongst clinical signs liver enlargement 52% and RUQ tenderness 65%; jaundice 14%.

**Debnath et al** shows fever 74%, nausea and vomiting in 30%, anorexia 42%, diarrhea in 4% of cases. Clinical signs of hepatomegaly in 80% and RUQ tenderness in 95% of cases.

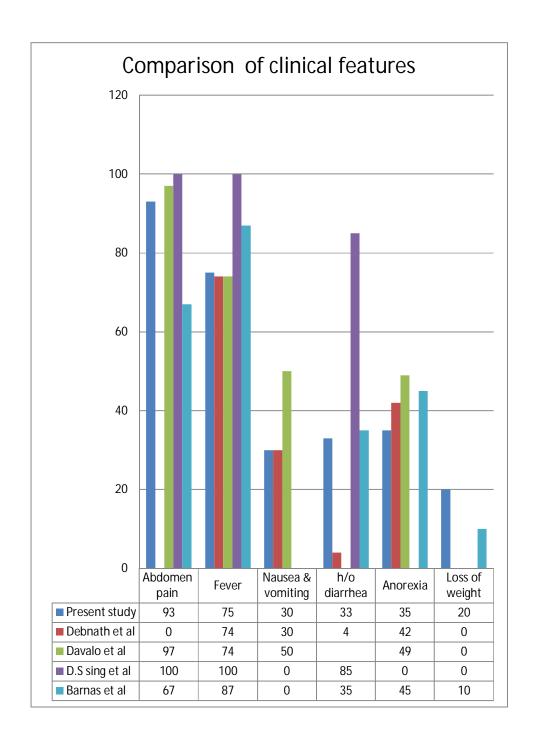
**Daval o et al** shows abdominal pain in 97%, fever 74%, nausea and vomiting 50%, anorexia 49% of cases. Clinical signs tenderness in right hypochondrium (RHC) was the predominant sign (95%), followed by localized guarding (47%), whereas 26% patients were presented with hepatomegaly

**D.S sing et al** studyshows abdomen pain and fever in 100% of cases; diarrhea 85% and jaundice in 24% of cases.

**Barnas et al** study shows abdomen pain in 67% of cases; fever in 87%; anorexia in 45%; diarrhea in 35%.

### **COMPARISON:**

CLINICAL	Present	Debnath	Davalo et	D.S sing	Barnas
FEATURE	study	et al	al	et al	et al
Abdomen	93	-	97	100	67
pain					
Fever	75	74	74	100	87
Nausea &	30	30	50	-	-
vomiting					
h/o	33	4		85	35
diarrhea					
Anorexia	35	42	49	-	45
Loss of	20	-	-	-	10
weight					



### **CLINICAL SIGNS:**

In present study RUQ tenderness is commonest clinical sign accounting to 65%; hepatomegaly 52% and jaundice 15%.

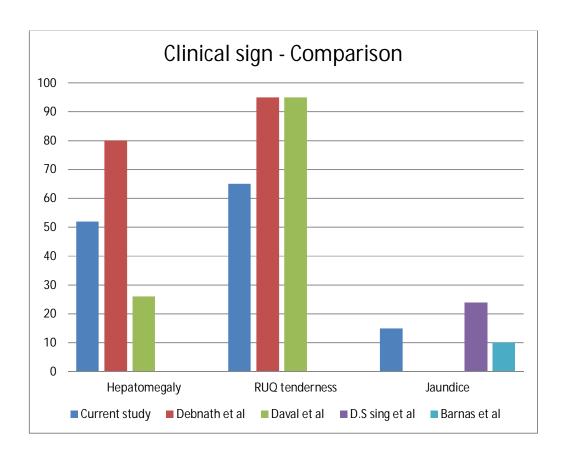
**Debnath et al study** shows tenderness in right hypochondrium as the predominant signs (95%) associated with hepatomegaly in 80% cases.

**Daval et al** study shows tenderness in right hypochondrium (RHC) as the predominant sign (95%), followed by localized guarding (47%), whereas 26% patients were presented with hepatomegaly

**D.S Sing et al** study has incidence jaundice in 24% of cases and **Barnas et al** showing jaundice in 10% of cases.

#### **COMPARISON:**

Clinical sign	Current	Debnath et	Daval et	D.S sing	Barnas
	study	al	al	et al	et al
Hepatomegaly	52	80	26	-	-
RUQ	65	95	95	-	-
tenderness					
Jaundice	15	-	-	24	10



## LABORATORY INVESTIGATIONS:

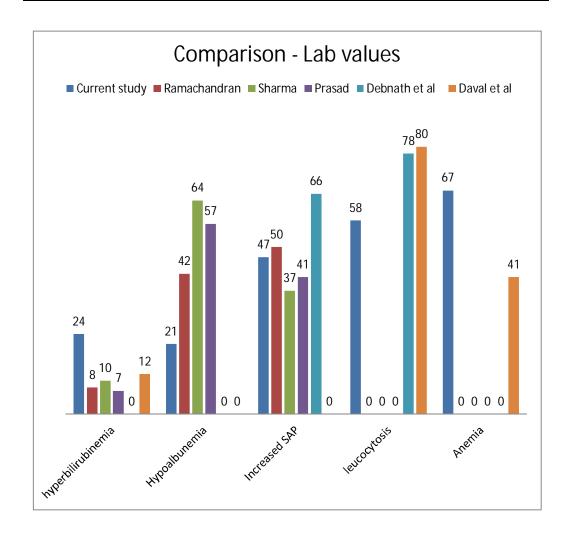
In our current study among laboratory values anemia was seen in 67%; leukocytosis in 58% of cases; increased bilirubin in 24% and increased SAP 47%.

**Debnath et al** study shows leukocytosis in 78% of cases and raised alkaline phosphatase in 66% cases.

**Daval et al** study showed anemia in 41%; leukocytosis 80% and hyperbilirubenemia in 12% of cases.

#### **COMPARISON:**

Test	Current study	Ramachandran	Sharma	Prasad	Debnath et al	Daval et al
hyperbilirubinemia	24	8	10	7	-	12
Hypoalbunemia	21	42	64	57	-	-
Increased SAP	47	50	37	41	66	-
leucocytosis	58	-	-	-	78	80
Anemia	67	-	-	-	-	41



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 mortalityin liver abscess with Jaundice we have not encountered such thing in this study.

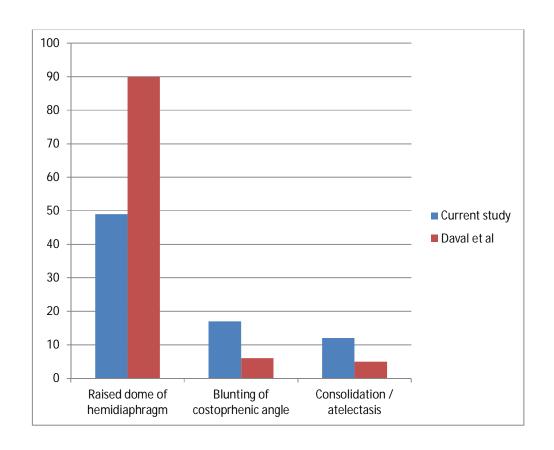
#### **RADIOGRAPHS:**

Most common feature in chest and abdomen radiograph raised dome of diaphragm seen in 49%. Pleural effusion seen in 17% and basal atelectasis in 12% of cases.

**Daval et al** study showed elevated dome of diaphragm in 90% of cases, pleural effusion in 6% and consolidation in 5% of cases.

#### **COMPARISON:**

Radiological feature	Current study	Daval et al
Raised dome of hemi diaphragm	49	90
Blunting of costophrenic angle	17	6
Consolidation / atelectasis	12	5



#### **ULTRASONOGRAM:**

Cases can be categorized by the size of abscess, location of abscess and also wether they are single or multiple. The mean size of abscess cavity in our study is 9.4cm. Majority of cases ranging in size of abscess cavity from 5-10 cm. About 70% of cases have right lobe involvement as compared to 16% in left lobe. 14% of cases have both lobes involved.

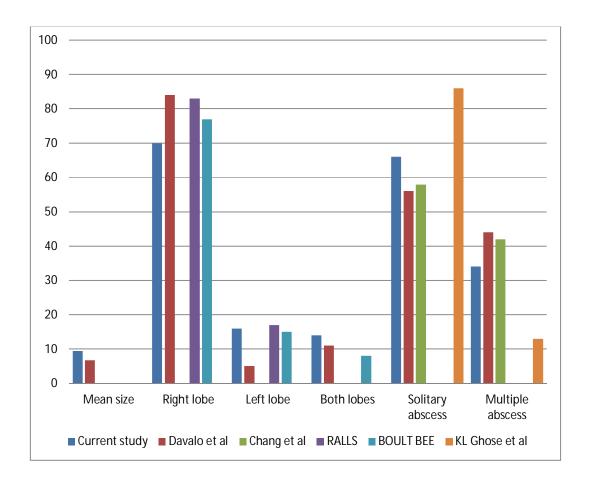
Solitary abscess is seen in 66% of cases as compared to multiple abscesses in 34%.

**Davalo et al** study showed mean abscess size to be 6.8cm. Solitary abscess was seen in 56% compared to multiple in 44%. Right lobe involvement in 84%, left lobe in 4.5% and bilobe in 12.5%

**Chang AC et alstudy**showed single abscess cavity in 58% to multiple abscess in 42%.

#### **COMPARISON:**

Feature	Current study	Davalo et al	Chang et al	RALLS	BOULT BEE	KL Ghose et al
Mean size	9.4	6.8	-	-	-	-
Right	70	84	-	83	77	-
lobe						
Left lobe	16	5	-	17	15	-
Both	14	11	-	-	8	-
lobes						
Solitary	66	56	58	-	-	86
abscess						
Multiple	34	44	42	-	-	13
abscess						



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.

All right Lobe Abscess were diagnosed by clinical enlargement ofliver whereas such definite Lobe enlargement was not seen in Left Lobe Abscess. The highest incidence of right lobe abscess in this study (70%) 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.

#### **CONSERVATIVE MANAGEMENT:**

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 Ciprofloxacin 200 mg twice daily, metronidazole 750 mg 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 –

Ceftriaxone 1g twice daily, Amikacin 750mg once daily and Metronidazle 500 mg thrice daily and then changed according to culture and sensitivity we haven't faced any major complications of chloroquine except vomiting. 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 90 cases studied, for about 35 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 abscessof **more than 5 cm size less than 10cm 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 a drug has been given, thick pus begins to liquefy and it can be aspirated 3 days later under ultra-sonogram control.

No biliary peritonitis was encounter in this study, whereasBalasegaram 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 ultra-sonogram 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 ultra-sonogram.

#### **PERCUTANEOUS DRAINAGE:**

We did percutaneous drainage for 17 cases out of 90 cases which accounts for 19%. Percutaneous catheter insertion was done under ultrasound guidance. Usually 14 or 16 Frenchmalecot's catheter or pig tail catheter is used for drainage. Among 17 cases of percutaneous drainage, we did ICD for 3 cases as presented as pyothorax. No mortality was seen in our study.

#### **LAPROTOMY AND DRAINAGE:**

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 13 cases which presented as

- a. Acute abdomen where abscess burst mimicking as perforated peptic ulcer, pancreatitis, ileal perforation or as perforated appendicitis.
- b. Patients not responding to aspiration.
- c. For very large abscess more than 10 cm.
- d. For large multiple abscess.

Post-operative period was uneventful and we did not have any mortality in our study.

#### **COMPARISON:**

The most common treatment modality in our study was aspiration 39% followed by conservative management in 28%.

**Daval et al** study showed aspiration in 78%; percutaneous catheter 10%; ICD 5% and laparotomy in 8%

TREATMENT MODALITY	CURRENT STUDY	DAVAL ET AL
Conservative	28	32
Aspiration	39	53
Percutaneous catheter	16	8
Intercostal drainage	3	3
Laparotomy	14	4
60 — 50 — 40 — 7 — 7 — 7 — 7 — 7 — 7 — 7 — 7 — 7 —		
Conservative Aspir		ercostal Laparotomy ainage L

## **CONCLUSION AND SUMMARY**

Out of 90 cases included in our study majority presented with classical features

- ➤ Incidence of liver abscess is 0.8% of total admissions in our hospital. Incidence of Amoebic liver abscesses very common in our study.
- ➤ h/o alcoholism was seen in 80% of patients
- ➤ Male predominates both in amoebic and pyogenic liver abscess in the ratio of 6:1
- ➤ Anemias, Leucocytosis were common Accompaniments
- Commonest symptom is abdominal pain and fever, sign being hepatomegaly and tenderness
- ➤ Only 14% of patients presented with Jaundice
- ➤ Right Lobe was predominantly involved in the ratio of 5:1
- Clinical diagnosis of liver abscess is straight forward except for those presenting with complications

- ➤ E.histolytica was isolated only 13% in stools and 10% of patients in aspirated pus.
- ➤ Out of 90 cases treated, 25 cases were treated conservatively, 35 cases required aspiration, 17 cases required percutaneous catheter drainage and 13 cases required open drainage.
- ➤ Ultrasound is the commonest and most useful investigation for diagnosis, treatment as well as follow up
- CT Scan mainly reserved for doubtful cases and those presenting with complications.
- ➤ In our series, there is more number of abscess seen in low socioeconomic status patients about 70%.
- ➤ Mortality in our series

Uncomplicated liver abscess: No mortality

Ruptured into abdominal cavity: No mortality

Ruptured into thoracic cavity : No mortality

#### **BIBILOGRAPHY**

- 1. Ochesner A, Debakay, M. Murray, A. Pyogenic abscess of the liver. A.M.J.Surg 1938: 40:292.
- 2. Barnes PF, Decock KM et al. A Comparison of Amoebic and Pyogenic abscess of the liver. Medicine 1987 66: 472.
- 3. Dela Rey N.J, Simjee AE, Patel A. Indications for Aspiration of Amoebic liver abscess.SAfr med.J.1989: 75:373.
- Balasegaram M. Management of Hepatic abscess. CurrSurg 1981
   18:283.
- 5. Bailey and Love's short Practice of Surgery. 24<sup>th</sup> Edition.
- Lamont N.M, and Pooler NR (1958). Hepatic Amoebiasis. Quart,
   J.Med 27:389.
- 7. Patterson M, Healey, GR. Shabot JM (1980). Serological Testing for Ameobiasis.Gastroenterology 78, 136.
- 8. Essential Surgical Practice A.Cuschieri M.D., G.R. Giles MS, A.R.Moosa M.D. 4<sup>th</sup> Edition.

- Wilmott AJ. (1962) Clinical Ameobiasis, Blackwell Scientific Publication, Oxford.
- Lippincott's Color Atlas and Text Book of Diagnostic Micro Biology 5<sup>th</sup> Edition.
- Singh DS, Sankaian A, Bhata V.N. Srivatsava K.K.
   Chandrasekaran S. Extra intestinal Amoebiasis A Clinical and Serological study.
- 12. Reynolds T.B. Amoebic abscess of liver Gastroenterology 60:952-953 1971.
- 13. Diseases of the liver 5<sup>th</sup> Edition. Leon. Schiff M.D., Ph. D., Eugene R. Schiff M.D.
- 14. Sheila Sherlock & James Dooley 10<sup>th</sup> Edition.Diseases of the liver and Biliary System.
- 15. WHO (1969) Amoebiasis Tech. Report Series. 421.
- 16. Meng XY, WU, JX Perforated Amoebic liver abscess clinical analysis of 110 cases. Southern Medical Journal.

- Ibrarullah. M. Agrawal D.K. Baijal SS, Mittal B.R. Kappor U.K.
   Amoebic Liver abscess with Intra Biliary Rupture HPB
   Surgery.
- 18. Oxford Text Book of Surgery Peter. J. Morris and Ronal A. Malt First Edition.
- 19. MadanaGopalan N, (1976) Amoebiasis in progress in clinical medicine in India. Ed., M.M.S.Ahuja, New Delhi, Arnold Heinman, 307.
- 20. Chatterjee K.D., Parasitology 12<sup>th</sup> Edition.
- 21. Maingot's Abdominal operations 10<sup>th</sup> Edition.
- 22. Chuah S.K. Chang Chien C.S., Sheen IS, Lin HH, Chiou SS, Chiu CT The Prognostic factors of severe Amoebic liver abscess retrospective study of 125 cases.
- 23. Adams EB, Macleod IN Invasive Amoebiasis Amoebic liver abscess and its complications medicine 1977 56:325.
- 24. 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.

- 25. Filice C, Dipierri G, Strosselli, M. Brunett. E. outcome of Hepatic amoebic abscess managed with 3 different therapeutic strategies.
- 26. Agarwal DK, Baijal SS, Mittal BR, Choudhuri G. Percutaneous Catheter drainage of Amoebic liver abscess.
- Lee KC, Yamazaki O.Hamba H. Analysis of 69 Patients with Amoebic liver disease. Journal of Gastroenterology 31 (1) 40.5
   1996 Feb.
- 28. Eggleston Fc et al. The results of Surgery in Amoebic liver disease. Experience in 83 Patients surgery 83 536-539, 1978.
- 29. Schwartz SI Extraperitoneal drainage of liver abscess. Surgical diseases of liver McGraw Hill 1964.
- Vakil BJ. 1983 Amoebic disease. A treatise, Basic and Business
   Publications, Bombay.
- Clinical Sonography 2<sup>nd</sup> Edition. Roger C. Saders MA B.M.Bch
   MRCP, FRCR Nancy, Smith, Miner.
- 32. Johnson RD. Mueller Pr. Ferrucc JT. Jr. et al, Percutaneous drainage of pyogenic liver abscess AJR; 1985 144: 463.

- 33. Ralls PW, Colletti. PM, Quinn MFet al. Sonographic findings in hepatic amoebiasis Radiology 1982; 145: 123.
- 34. Van Sonnenberg, E Mmeller PR Schiffman HR et al. Intra hepatic amoebic abscess; Indication and results of Percutaneous Catheter drainage.
- 35. Ralls PW Quinn MF, Bows Well WD Jr et al., Patterns of Resolution of Successfully treated hepaticAmoebic abscess. Sonographic evaluation.
- 36. Mendez RJ, Schiebler ML et al Hepaticabscess; MR imaging findings Radiology 1994; 190 431.
- 37. 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.
- 38. The Indian Journal of Gastroenterology volume 16: Nov: 1997 controversies in Gastroenterology Von.DAYAL, AK JAIN, JP BHATT, VK DIXIT, AKAGARWAL.
- 39. Gastroenterology today: ISSN: 0971-8222. Vol II.No.1 Jan.March: 1998. P/49-51.

- 40. Gastroenterology today Vol. I.No.3 ISSN. 0971-8222 April –

  June 1997 Invasive Amoebiasis MP Sharma, S. Dasavathy,

  AIIMS, New Delhi.
- 41. Indian Journal of Gastroenterology April 1998 Volume 17, No.2
- 42. Tropical disease Bulletin July 1996. Vol. 93 No.7.
- 43. Debnath CR, Debnath MR, Khalid MS, Mahmuduzzaman M. Clinical profile of 250 cases of amoebic liver abscess.

  Mymensingh Med J. 2013 Oct;22(4):712-5.
- 44. A Prospective Series Case Study of Pyogenic Liver Abscess:

  Recent Trands in Etiology and ManagementDhaval O.

  Mangukiya,corresponding author1,2 Jitendra R. Darshan,1,2

  Vijay K. Kanani,1,2 and Saurabh T. Gupta2 Indian J Surg. 2012

  October; 74(5): 385–390.
- 45. An Increasing Prominent Disease of Klebsiellapneumoniae Liver Abscess:Etiology, Diagnosis, and Treatment.Liu Y, Wang JY, Jiang W. Gastroenterol Res Pract. 2013;2013:258514.
- 46. Single and multiple liver abscesses in adults in Delhi are amoebic in origin: a clinical and microbiological study. Tayal A, Lal P,
   Uppal B. TropDoct.2013 Apr;43(2):77-9. doi: 10.1177/0049475513485237.

- 47. Mortality in Emergency Department Sepsis score as a prognostic indicator in patients with pyogenic liver abscess. Kuo SH, Lee YT, Li CR, Tseng CJ, Chao WN, Wang PH, Wong RH, Chen CC, Chen SC, Lee MC. Am J Emerg Med. 2013 Jun;31(6):916-21. doi: 10.1016/j.ajem.2013.02.045
- 48. Pyogenic liver abscess trends in South Australia. Chang AC, Ha
  NB, Satyadas T, Maddern G. ANZ J Surg. 2013 Oct 14. doi:
  10.1111/ans.12411. [Epub ahead of print]
- Liver abscess as a therapeutic and diagnostic challenge. Kolovrat M, Busić Z, Servis D, Situm A, Cavka V. ActaClin Croat. 2013 Jun;52(2):257-60.
- Single and multiple liver abscesses in adults in Delhi are amoebic in origin: a clinical and microbiological study. Tayal A, Lal P, UppalB.TropDoct. 2013 Apr;43(2):77-9. doi: 10.1177/0049475513485237. Epub 2013 Jun 11.
- 51. An Increasing Prominent Disease of Klebsiellapneumoniae Liver Abscess:Etiology, Diagnosis, and Treatment. Liu Y, Wang JY, Jiang W. Gastroenterol Res Pract. 2013;2013:258514. Epub 2013 Sep 30.

## **PROFORMA**

<u>•</u>	NAME:	SL.NO:
•	AGE /SEX:	
•	ADDRESS WITH CONTACT NUMBER:	
•	IP NO:	
•	DATE OF ADMISSION:	
•	DATE OF SURGERY / INTERVENTION:	
HISTO	DRY OF PRESENTING ILLNESS:	
	Pain: location:	
	Duration:	
	Fever: duration:	
	Assoc with chills:	
	Jaundice:	
	Vomiting:	
	Diarrhea:	
	Loss of weight:	
	Loss of appetite:	
PAST	HISTORY:	
	K/C/O DM / HTN / TB / BA / epilepsy / CAD	
	H/o drug intake (steroid / chemotherapy)	

PERSONAL HISTORY: H/o alcohol intake

#### **FAMILY HISTORY:**

#### **CLINICAL EXAMINATION**

GENERAL EXAMINATION: temp: p.r: bp: Icterus:

SYSTEMIC EXAMINATION:

CVS:

RS: B/I air entry

Pleural rub

PER ABDOMEN: Abd soft / distented

Tenderness: epigastrium / Rthypochondrium

Guarding: present / absent

Rigidity: present / absent

Hepatomegaly / Rt upper quadrant mass

Free fluid: + / -

CNS:

**INVESTIGATIONS:** 

CBC / RBS / RFT / LFT / CXR / AXR / ECG / HIV ELISA / HBSAG / HCV / PTINR

USG ABDOMEN: SIZE: NUMBER: LOCATION:

TREATMENT:

CONSERVATIVE / USG GUIDED ASPIRATION / PIG TAIL CATHETER / LAPROTOMY AND DRAINAGE

# MASTER CHART

SL NO NAME	AGE SEX	1.4	IP NO	ABD PAIN	FEVER	ALCOHOL	HEPATOMEGALY	ABD PAIN FEVER ALCOHOL HEPATOMEGALYRUQ TENDERNESSABSCESS SIZE TREATMENT	ABSCESS SIZE	TREATMENT
1 PERUMAL	55 M	Σ	34309 +	9 +	+	+	+	+	7	7 Aspiration
2 KALIMUTHU	21 M	2	36378	3 +	+	+	+	+	11	11 Percutaneous drainage
3 KANNIYAPPAN	46 M	2	47605 +	5 +	-	+		+	9	6 Aspiration
4 MUNNIYAMA	55 F	u.	31224 +	1+	+	-		+	3	3 Conservative
5 SHANMUGAM	53 M	2	36421 +	1 +	-	+	+	+	9	6 Conservative
6 SAROJA	67 F	ш	46539 +	9 +	-	-	+	+	6	9 Aspiration
7 THANGARAJ	23 M	2	37889	9-	+	+		+	4	4 Conservative
8 RAJAMMAL	30 F	ш	43214 +	1 +	+	-	+	+	6	9 Aspiration
9 VELUMANI	49 M	2	47532 +	2 +	-	-	+		7	7 Aspiration
10 VASUDEVAN	26 M	2	38765 +	2 +	+	+			10	10 Percutaneous drainage
11 KUPPUSAMY	57 M	Σ	38451 +	1 +	+	+	+	1	11	11 Percutaneous drainage
12 DURAI	31 M	Σ	36542 +	2 +	+	+		-	6	9 Aspiration
13 NAGARAJ	35 M	2	40967	7 +	-	+	+	+	5	5 Aspiration
14 MARIYAPPAN	50 M	Σ	41342 +	2 +	+	+		+	10	10 Aspiration
15 RAJENDRAN	51 M	Σ	42671 +	1 +	+	+	+	+	14	14 Percutaneous drainage
16 RAMASAMY	37 M	Σ	38888 +	3+	+	+			10	10 Percutaneous drainage
17 KUPPAIYE	38 F	u.	31098 +	3+	+			+	4	4 Conservative
18 DEVARAJ	24 M	Σ	30967	7 -	+	+			4	4 Conservative
19 SUBRAMANI	52 M	Σ	32961 +	+ 1	+	+		+	15	15 Laparotomy drainage
20 KANNAN	32 M	Σ	33376 +	2 +	+	+		+	11	11 Percutaneous drainage
21 VENGAIYAN	36 M	Σ	42356 +	2 +	+	+	+	+	16	16 Percutaneous drainage
22 GOVINDAMMAL	44 F	u.	41567 +	1 +	+			+	8	8 Aspiration
23 KRISHNAMMA	30 F	u.	45278 +	3 +			+	+	7	7 Aspiration
24 PANDIYAN	37 M	Σ	47536 +	2 +	,	+	+		7	7 Aspiration
25 MUNNIYAPPAN	52 M	Σ	32456 +	2 +	+	+			19	19 Laparotomy drainage
26 MURUGAMMAL	50 F	u.	40245 +	+	+		+	+	7	7 Aspiration
27 MURUGAN	46 M	Σ	43165 +	+	,	+	+	+	8	8 Aspiration
28 DEVAGI	34 F	u.	40128 +	3+	+			+	7	7 Conservative
29 THANGAVEL	29 M	Σ	39567 +	+ /	+	+		+	21	21 Laparotomy drainage
30 SHANTHI	33 F	u_	30110 +	1+	+			+	3	3 Conservative

# MASTER CHART

31	31 MANI	37 M	Σ	31024 +	_	+	1	+	4	4 Conservative
32	32 KANNAN	39 M	Σ	36254 +	+	+	+	-	6	9 Aspiration
33	33 SUNDAR RAJAN	65 M	Σ	30475 +	+	+	-	-	13	13 Percutaneous drainage
34	34 SOUNDARARAJAN	42 M	Σ	49881 +	+	+	-	-	18	18 Laparotomy drainage
35	35 PERUMAL	46 M	Σ	46012 +		+	+	+	5	5 Conservative
36	36 MOIDEEN	25 M	Σ	40352 +	+	+	+	+	8	8 Aspiration
37	37 KANNIYAPPAN	61 M	Σ	49887 +	+	+	-	-	16	16 Percutaneous drainage
38	38 VADIVEL	32 M	Σ	46712 +		+	-	-	6	9 Percutaneous drainage
39	39 CHANDRAN	31 M	Σ	44410 +	+	+	+	+	5	5 Conservative
40	40 SHANTHAMMAL	34 F	ч	36668 +	+		+	-	8	8 Aspiration
41	41 MURUGAN	41 M	Σ	35547 +	+	+	-	+	12	12 Percutaneous drainage
42	42 MUNNIYANDI	52 M	Σ	30021 +		+	+	+	7	7 Aspiration
43	43 RAMAIAH	58 M	Σ	32001 +	+	+	+	-	14	14 Laparotomy drainage
44	44 ANDAL	60 F	ш	47784 +	+		-	-	4	4 Conservative
45	45 KUMAR	18 M	Σ	43358 +	+		+	+	9	6 Aspiration
46	46 PERUMALSAMY	44 M	Σ	41025 +	+	+	+	+	13	13 Laparotomy drainage
47	47 PICHAIYYA	48 M	Σ	49901 -	+	+	-	-	4	4 Conservative
48	48 RAMAIAH	39 M	Σ	39991 +	+	+	+	+	8	8 Aspiration
49	49 VELUCHAMY	47 M	Σ	48857 +	+	+	+	+	14	14 Percutaneous drainage
50	50 ESWARAN	27 M	Σ	37777 +	+	+	+	_	13	13 Percutaneous drainage
51	51 RANGARAJ	49 M	Σ	35569 +	+	+	-	+	9	6 Conservative
52	52 SAROJAMMA	42 F	ш	39994 +			+	ï	6	9 Aspiration
53	53 SARGUNAM	57 M	Σ	40041 +	i	+	1	+	4	4 Conservative
54	54 DESIGAM	45 M	Σ	40005 +	+	+	+	+	8	8 Aspiration
55	55 KARUNANITHI	29 M	Σ	47775 -	+	+	-	+	3	3 Conservative
56	56 RAMASAMY	48 M	Σ	31114 +		+	1	-	4	4 Conservative
57	57 ELUMALAI	41 M	Σ	35877 -	+	+		+	8	8 Aspiration
58	58 PONNUSAMY	54 M	Σ	32599 +	+	+	1	+	6	9 Aspiration
59	59 GUNASEKAR	22 M	Σ	34288 +		+	+	1	7	7 Aspiration
9	60 DURAISAMY	49 M	Σ	45687 +	+	+	-	1	13	13 Laparotomy drainage

# MASTER CHART

61	61 MUNNIYAPAN	48 M	39755 +	+	+	+	+	20	20 Percutaneous drainage
62	62 NAGARAJ	40 M	41294 +	+	+		+	7	7 Conservative
63	63 SUBRAMANIAN	58 M	40032 +	+	+	+	1	16	16 Laparotomy drainage
64	64 RAMAMOORTHY	M 09	36688 +		+	+	1	6	9 Aspiration
65 (	65 CHOCKALINGAM	M 65	46587 +	+	+	+	+	12	12 Percutaneous drainage
99	66 GANESAN	33 M	43958 +	-	+	-	+	4	4 Conservative
67	67 KRISHNAVENI	46 F	39920 +	+	-	-	+	4	4 Conservative
1 89	68 LAKSHMI	51 F	42203 +	+	-	1	+	4	4 Conservative
69	69 SHANMUGAM	53 M	41122 +	+	+	+	+	5	5 Conservative
70	70 RENGANATHAN	47 M	30007 +		+	-	+	3	3 Conservative
71	71 DURAISAMI PILLAI	42 M	36611 -	+	+	-	-	6	9 Percutaneous drainage
72	72 MARUTHAPANDIYAN	M 65	30102 +	+	+	+	-	19	19 Laparotomy drainage
73 8	73 ELUMALAI	63 M	44205 +		+	+	+	6	9 Laparotomy drainage
74	74 PERUMAL	41 M	44215 +	+	+	+	+	8	8 Aspiration
75	75 SEKAR	28 M	33670 +	+	+	+	+	15	15 Laparotomy drainage
76	76 ESWARAMOORTHY	54 M	33570 +	+	+	+	1	8	8 Aspiration
77	77 MUTHAIYAN	57 M	38852 +		+	1	1	7	7 Aspiration
78	78 SAMY	39 M	38811 +	+	-	+	+	9	6 Aspiration
79	79 JAYARAJ	40 M	46601 +	+	+	-	+	17	17 Percutaneous drainage
80	80 SHEIK MOHAMMED	26 M	45351 +	+	+	+	+	5	5 Aspiration
81	81 MUKKAIYAN	64 M	42145 +	+	-	+	+	4	4 Conservative
82	82 ARUMUGAM	43 M	32014 +	+	+	+	+	9	6 Aspiration
83	83 ALBERT	45 M	33019 +		+	1	-	3	3 Conservative
84	84 MOORTHY	27 M	30190 +	+	+	1	+	5	5 Conservative
85	85 KAMARAJ	S9 M	40909 +	+	+	1	-	6	9 Aspiration
86	86 MUTHU	33 M	47012 +	+	+	+	+	6	9 Laparotomy drainage
87	87 VETRI	38 M	44065 +	+	+	+	-	8	8 Aspiration
88	88 ELUMALAI	51 M	45023 +	,	+	+	+	8	8 Aspiration
89	89 KUPPAN	63 M	48702 +	+	+	+	+	9	6 Aspiration
906	90 PONNRAJ	S7 M	49583 +	+	+		-	11	11 Laparotomy drainage

## நுண்கதிரியல்பரிசோதனையின்அடிபடையில்கல்லீரல்சீழ்கட்டியி ன்மருத்துவவழங்கல்,

நோய்காரணிமற்றும்சிகிச்சைமுறைகள் பற்றியஆய்வு

ஆய்வாளர் : டாக்டர் . நிர்மல் .ஜெ முதுநிலைமேற்படிப்புமாணவர் அறுவைசிகிச்சைபட்டபடிப்பு

வழிகாட்டி : டாக்டர் .பேராசிரியர் .சுரேஷ் அறுவைசிகிச்சை பேராசிரியர் அரசுஸ்டான்லிமருத்துவமனை

#### <u>பங்கேற்பாளரின்தகவல்படிவம்</u>

நீங்கள்இந்தஆய்வில்பங்கேற்கஅழைக்கபடுகிறிர்கள் .

இந்தஆய்வில்பங்கேற்கும்முன்னர்இதன்நோக்கத்தையும்முறைகளையும்இத னால்ஏற்படகூடியபின்விளைவுகளையும்ஏதேனையும்நீங்கள்அறிந்துகொள்ளஆய்வா ளர் அளிக்கும்தகவல்பின்வருமாறு

கலீரல்சீழ்கட்டியினால்பாதிகப்பட்டநோயாளிகள்மட்டுமேஇந்தஆய்வில் எடுத் துக்கொள்ளபடுவீர்கள் . உங்கள்நோயின்முழுவரலாறும், உங்களின்முழுஉடல்பரிசோதனையும்தெளிவாகவும்விரிவாகவும்பதிவுசெய்யப்படும். இரத்தபரிசோதனைமற்றும்நுண்கதிரியல்பரிசோதனைகளின்முடிவுகள் ஏற்றவாறுபதி யப்படும்.

பரிசோதனைக்கு முன்னும்பின்னும் மற்றும்பரிசோதனையின் பொழுதும் உங்களிடம் ஏற்படும் உடல் நிலை மாற்றங்கள் பதிவு செய்யப்படும் .

இந்தஆய்வின்முடிவுகள்மருத்துவகாரணங்களுக்காகவும்மருத்துவகல்விக்கா கவும்பயன்படும்.

இந்தஆய்வுபற்றியசந்தேகங்களுக்கு உரியமுறையில்விளக்கம்அளிக்கப்படும். தங்களைபற்றியதகவல்கள்இரகசியமாகபாதுகாக்கப்படும்.

இந்தஆய்வில் இருந்துஎப்போதுவேண்டுமானாலும்தாங்கள்எவ்விதமுன்னறி விப்பின்றியும், விலகிகொள்ளலாம். இவ்வாய்வில் பங்கேற்குமாறுகேட்டுக்கொள்கிறேன்.

இப்படிக்கு

ஆய்வாளர் கையொப்பம் நோயாளியின்கையொப்பம்

டாக்டர்நிர்மல்ஜெ பெயர்

### <u>நுண்கதிரியல்பரிசோதனையின்அடிபடையில்கல்லீரல்சீழ்கட்டியி</u> <u>ன்மருத்துவவழங்கல்</u>

#### நோய்காரணிமற்றும்சிகிச்சைமுறைகள் பற்றியஆய்வு

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வழிகாட்டி : டாக்டர் .பேராசிரியர் .சுரேஷ் அறுவைசிகிச்சை பேராசிரியர் அரசுஸ்டான்லிமருத்துவமனை

#### <u>சுயஒப்புதல்படிவம்</u>

இந்தமருத்துவஆய்வின்விவரங்கள்எனக்குவிளக்கப்பட்டதுஎன்னுடையசந்தே கங்களைகேட்கவும்அதற்கானதகுந்தவிளக்கங்களைபெறவும்வாய்ப்பளிக்கப்பட்டது

நான்இவ்வாய்வில்தன்னிச்சையாகத்தான்பங்கேற்கிறேன்எந்தகாரணத்தினாலு ம்எந்த சட்ட சிக்கலுக்கும் உட்படாமல் விலகிக்கொள்ளலாம் என்றுஅறிந்துக்கொண்டேன்

நான்ஆய்விலிருந்துவிலகிக்கொண்டாலும்ஆய்வாளர்என்னுடையமருத்துவஅ றிக்கைகளைபார்ப்பதற்கோஅல்லதுஉபயோகிக்கவோஎன்அனுமதிதேவையில்லைஎ னஅறிந்துக்கொள்கிறேன்.

என்னைபற்றியதகவல்கள்இரகசியமாகபாதுகாக்கப்படும்என்பதைஅறிவேன்

இந்தஆய்வின்மூலம்கிடைக்கும்தகவல்களையும்பரிசோதனைமுடிவுகளையு ம்ஆய்வாளர்அவர்விருப்பத்திற்கேற்பஎவ்விதமாகபயன்படுத்திக்கொள்ளவும்அதனை பிரகரிக்கவும்என்முழுமனதுடன்சம்மதிக்கிறேன்

இந்தஆய்வில் பங்குக்கொள்ளஒப்புக்கொள்கிறேன்எனக்குகொடுக்கப்பட்ட அறி வுரையின்படிநடந்துக்கொள்வதுடன்ஆய்வாளருக்கு உண்மையுடன் இருப்பேன்என்றும் உறுதியளிக்கிறேன்

என்உடல்நலம்பாதிக்கப்பட்டாலோஅல்லதுஎதிர்பாராதவழக்கத்திற்குமாறான நோய்க்குறிதென்பட்டாலோஉடனேஅதைதெரிவிப்பேன்எனஉறுதிகூறுகிறேன்

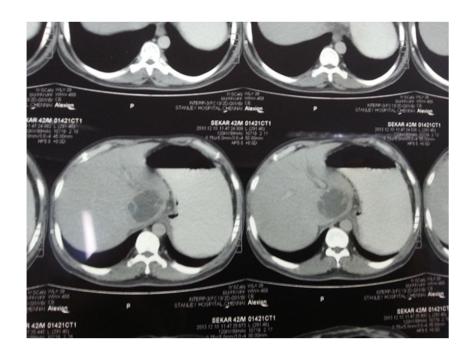
இந்த ஆய்வில்எனக்கு எவ்விதமற்றும்அனைத்து பரிசோதனைகளையும்சிகிச் சைகளையும்மேற்கொள்ளநான்முழுமனதுடன்சம்மதிகிரேன்

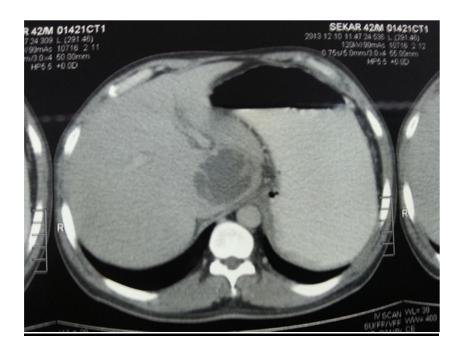
#### இப்படிக்கு

ஆய்வாளர் கையொப்பம் டாக்டர்நிர்மல்ஜெ நோயாளியின்கையொப்பம் பெயர்

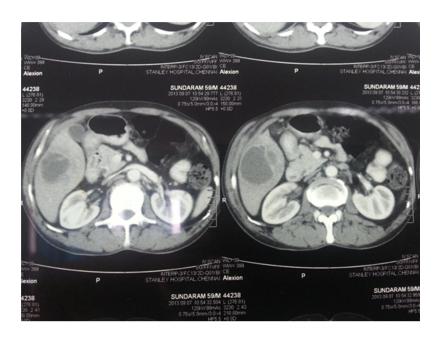
# **CT IMAGES**

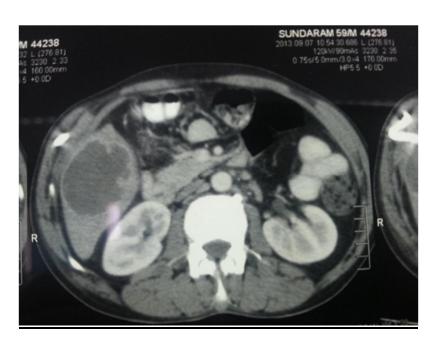
## Caudate lobe liver abscess



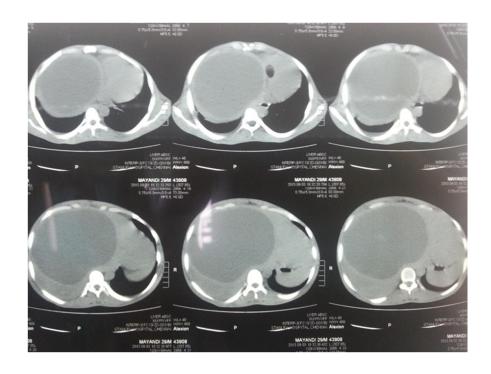


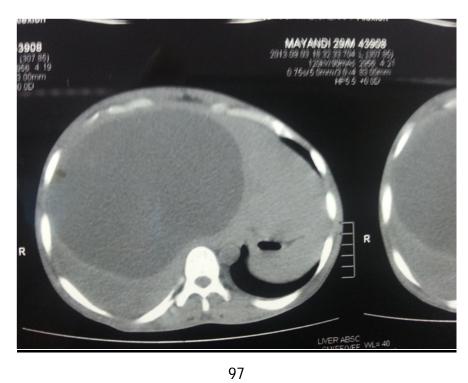
# Right lobe liver abscess





# **Huge solitary right lobe amebic abscess**





# **USG GUIDED CATHETER INSERTION**



