A CLINICAL STUDY, DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS PRESENT IN OUR INSTITUTION CHENGALPATTU MEDICAL COLLEGE

A PROSPECTIVE STUDY

Dissertation submitted to

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations
for the award of the degree of

M. S. GENERAL SURGERY (BRANCH I)

CHENGALPATTU MEDICAL COLLEGE
THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

APRIL 2016

DECLARATION

I, **Dr.MURUGESAN.V**, solemnly declare that the dissertation **"A CLINICAL STUDY,DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS PRESENT IN OUR INSTITUTION CHENGALPATTU MEDICAL COLLGE"** A bonafide work done by me in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu, Under the guidance of **PROF. Dr.C.SRINIVASAN. M.S**, professor, Department of General Surgery, Chengalpattu Medical College, Chengalpattu.

Place: Chengalpattu.

Date:

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This is to certify that this dissertation titled **"A CLINICAL STUDY, DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS PRESENT IN OUR INSTITUTION CHENGALPATTU MEDICAL COLLGE"** has been prepared by **DR.MURUGESAN.V,** under my supervision in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu, during the academic period 2013 – 2016, and is being submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the University regulation for the award of the Degree "Master Of Surgery" (M. S., General Surgery) and his dissertation is a bonafide work.

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1. Trial protocol, dated _____version no
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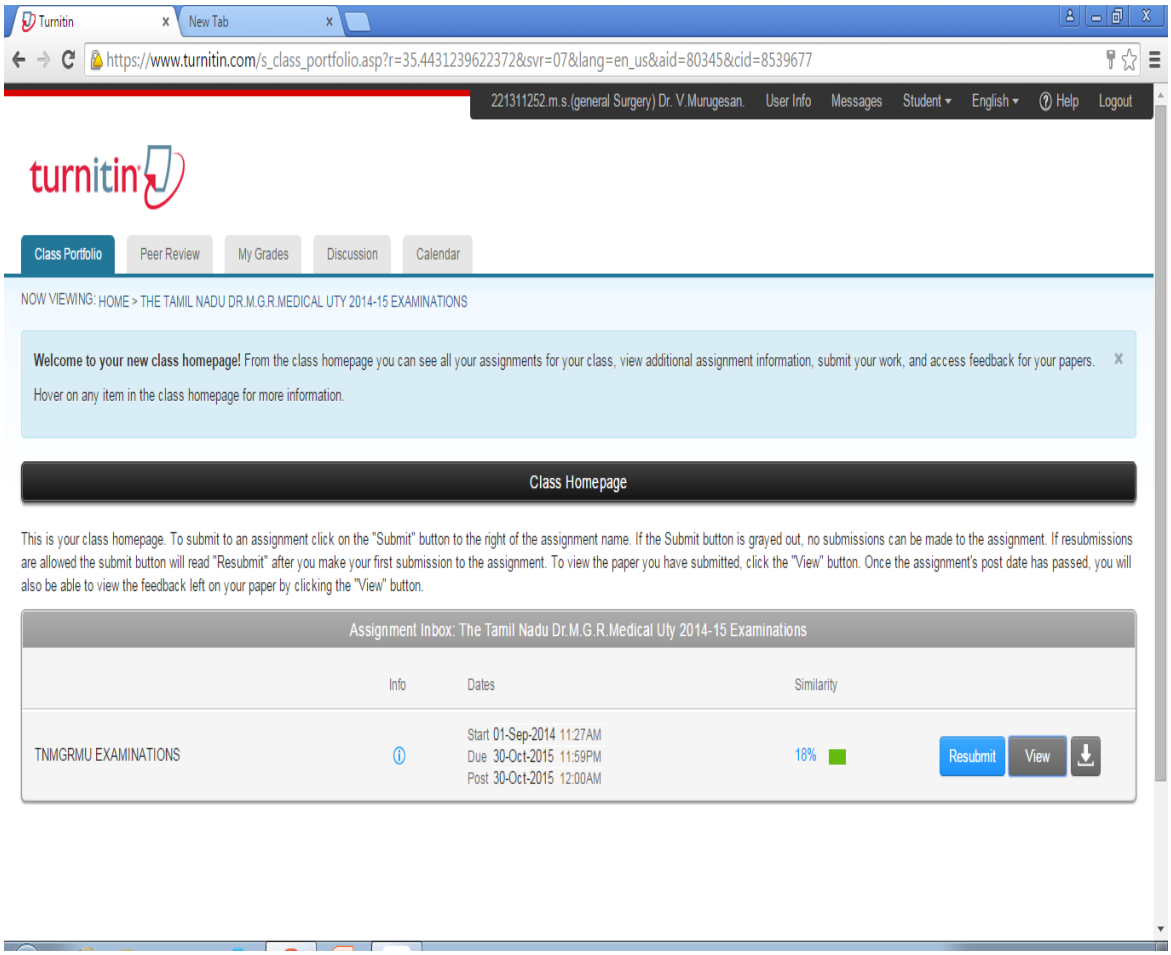
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ABSTRACT

TITLE: A CLINICAL STUDY, DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS PRESENT IN OUR INSTITUTION CHENGALPATTU MEDICAL COLLEGE

AIMS AND OBJECTIVES OF THE STUDY

1. To study clinical presentation of liver abscess ie, Distribution with respect to age and sex, mode of presentations in our area.
2. To study risk factors associated with liver abscesses.
3. To study effectiveness of different modes of management**.**

MATERIAL AND METHODS

SOURCE OF DATA

All cases of Liver abscesses presenting to the Surgery OPD or Casualty of CHMC Hospital, referred from medical wards of CHMC hospital or referred from outside diagnosed as case of liver abscess, in Chengalpattu over a period of 1 and half years with Clinical/ Sonological/CT features of Liver abscess.

METHOD OF COLLECTION OF DATA

Inclusion Criteria:

1. All cases of liver abscess diagnosed clinically and/or ultrasonographically.
2. All cases of bacterial and parasitic liver abscess
3. All cases in evolving, liquefied & ruptured stage with or without peritonitis
4. All cases of Diagnosed Liver Abscess being referred to CHMC.

Exclusion Criteria:

- Traumatic Liver Abscess
- Past history of liver abscess

Sample size: 60cases.

Study design: Prospective study

Duration of study: 1 1/2 years.

This study was done from MARCH 2014 to AUGUST 2015 on 60 patients. Case selection for the study will be done in the initial 1 yr followed by follow up totally for 6 months.

CONCLUSION

Most common liver abscess presenting in our institution was amoebic in nature. Most common age of presentation was 45 years. Most common presentation of liver abscess was abdominal pain. Most common clinical sign was intercostal tenderness.

Alcoholism becomes the most frequently associated risk factor. Ultrasound and CT scan abdomen plays an important role in diagnosing most of the liver abscess patients presented in our institution. Eighteen percent (18%) of the liver abscess patients were managed successfully with medical management alone.

Those who are all not responding to medical management were treated with image guided drainage/aspiration. Forty five percent (45%) of the patients presented in our institution were successfully treated by image guided drainage/aspiration.

Those who developed complications (ruptured liver abscess) and who are all not responding to conservative line of management required emergency open drainage. Thirty seven percent (37%) of patients who developed complications on presentation or later were treated with open drainage in our institution.

KEY WORDS

LIVER ABSCESS

AMOEBIC LIVER ABSCESS

PYOGENIC LIVER ABSCESS

ULTRASONOGRAM

CT ABDOMEN

MEDICAL MANAGEMENT

IMAGE GUIDED ASPIRATION/DRAINAGE

OPEN DRAINAGE

LIST OF ABBREVIATIONS

CBD	-	COMMON BILE DUCT
CHD	-	COMMON HEPATIC DUCT
PT	-	PROTHROMBIN TIME
INR	-	INTERNATIONAL NORMALISED RATIO
A:G	-	ALBUMIN:GLOBULIN
IM	-	INTRAMUSCULAR
IVC	-	INFERIOR VENA CAVA
AST	-	ASPARTATE TRANSAMINASES
ALT	-	ALANINE TRANSAMINASES
CXR	-	CHEST X RAY
USG	-	ULTRASONOGRAM
CT	-	COMPUTED TOMOGRAM
PTC	-	PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAM

INTRODUCTION

Liver abscess was described early in 460-377 B.C. by *Hippocrates*, still it remains a challenging situation (especially in tropical countries due to poor

hygiene & sanitation, alcoholism and reduced literacy rate) due to its highly variable presentation, causing diagnostic difficulties.

As India is a one of the tropical country and home to 400 million people harbouring E.histolytica, the causative organism of amoebic liver abscess, it is important to thoroughly understanding of the liver abscess.

- Due to the rising incidence in alcoholism, diabetics & immunocompromised status, liver abscess becomes a matter of grave concern as complications rate are high especially in this sub-group, leading to increased morbidity and mortality.

- Due to the advancement in imaging modalities, a more concrete picture to treat liver abscesses is slowly evolving. However, much work remains to be done. The story has not ended: it has just begun**.**

- All these factors has inspired me to select Liver Abscess as my thesis topic which assumes more importance in our country where rural population constitutes approximately 70% and therefore it is mandatory to develop appropriate & realistic guidelines for early diagnosis and management strategies for liver abscesses in order to reduce the morbidity and mortality associated with it.

AIMS AND OBJECTIVES OF THE STUDY

1. To study clinical presentation of liver abscess ie, Distribution with respect to age and sex, mode of presentations in our area.

2. To study risk factors associated with liver abscesses.
3. To study effectiveness of different modes of management**.**

REVIEW OF LITERATURE

The liver is one of the most common organ subjected to the development of abscesses. In one study of 540 intraabdominal abscesses over a 12 year period liver abscesses made 48% of all visceral abscesses.

In 400.B.C, Hippocratesestablished drainage of liver abscess as a form of therapy.

Oschner & Debakey described Pyogenic liver abscess in 47 cases in the year 1938 in their classic paper and reviewed the world literature.

In 1953, McFadzean and associates advocated closed aspiration and antibiotics for treatment of solitary pyogenic liver abscess.

In 1846, Waller described Pyogenic abscess as a diseased characterized by suppurative thrombophlebitis of the portal vein and formation of single or multiple abscesses.

In older days, Pyogenic liver abscess was largely a disease of people of 20-30yrs age group but now the spectrum of disease has changed to age group 50-60yrs with biliary tract diseases & cryptogenic as the main etiology.

With the advent & development of improved antibiotics over the passage of time, incidence of pyogenic liver abscess would be expected to decrease, but the incidence is increasing as indicated by studies of Huang &

Associates(1996) in which there were 20-22 cases per 100,000 hospital admissions, appears to be double those of previous 2 decades.

In 1883, Koch initially demonstrated amoebas in the capillaries and tissues adjoining the wall of the liver abscess.

In 1887 Entamoeba Histolytica was first recovered from the wall of a hepatic abscess by Kartulis.

Yeoh Kg et al-National University Hospital, Singapore, Reviewed 41 cases from 1994 to 199827 (67%) pyogenic, 6 (15%) amoebic, 2 (5%) tuberculosis, 6(15%) intermediate. Percutanious needle aspiration was performed for 85% of pyogenic abscess, and surgery was indicated in only two cases because of complications. They found that percutaneeous aspiration of liver abscess not only help to confirm the diagnosis but also to uncover clinically unsuspected conditions like malignancy and tuberculosis which may mimic liver abscess.

Hai aa singh a et al Department of Surgery Patna Medical College Hospital India reviewed 220 cases of ameobic liver abscess between 1981-86.the majorities were young middle aged males belonging to lower socioeconomic group and 85% gave history of drinking toddy fermented palm juice. Over 88%responded well to conservative treatment with aspiration[4]. Laparotomy was required in slightly over 10% of cases and in these the mortality was 12% as compared to 2% with conservative treatment.

Alvarez Perez et al-Depatment of Surgery, San Aagustin Hospital Avilies Spain Reveiwed pyogenic liver abscess, 133patients treated in 5

hospitals during the year 1985 to 1987were studied. 63(47%) were subjected to percutanious drainage, 45 (34%) were treated by open drainage (surgical) and the remaining 25 cases(19%)received antibiotic therapy only. Prognostic variables were the presence of shock, anaemia, elevated prothrombin time and mixed infection. Treatment of pyogenic liver abscess should be tailored to each patient, the most of them can be successfully treated with antibiotics and percutaneous methods. Those with signs of multiorgan failure or septicemia should preferably be managed in ICU.

Amoebiasis are present in 10% of the population, (most commonly affects young adults 20-30yr age group with 19:1 male to female ratio) around 500 million persons in developing countries with 50 million cases of invasive disease, and may account for as many as 100,000 deaths . Amoebic abscess is one of the most common complication of intestinal amoebiasis and can occur many years after exposure in endemic areas.

Indirect haemagglutination test is a reliable serologic test for hepatic amoebiasis, yielding positive results in atleast 85% of patients with extra intestinal disease and titres often exceed 1:256 almost always noted by 2 weeks into disease and may remain high for many years following successful therapy.

Leukocytosis with white cell counts averaging between 18,000 – 20,000 is noted in great majority of the patients with liver abscess (mostly pyogenic), Anaemia associated with long standing infection, the most significant chemical abnormality is an elevation of serum alkaline phosphatase. Grossly raised serum levels of vitamin B12 (2000 to 4000 pg/ml)

have been reported present in pyogenic liver abscess. The “chocolate sauce,” “anchovy paste” aspirate is considered pathognomic of an amoebic abscess.

Most hepatic abscesses involve the right lobe of liver (postero-superior segment), accounting for three-fouth of the cases , in 20% of cases the left lobe is involved and in rare cases caudate lobe is involved.

Most of the pyogenic abscesses are polymicrobial in nature and account for about 40% of the cases. In pyogenic liver abscess Eschericia.Coli and Klebsiella.pneumonia most commonly cultured organisms.

The classic description of the presenting symptoms of liver abscess are fever, jaundice, and right upper quadrant pain & tenderness. A recent study from Taiwan of 133 patients found fever in 96% of the patients, chills in 80%, abdominal pain in 53%, and jaundice in 29%.

Patients with liver abscess who require aspiration irrespective of the etiology includes abscess with size > 5 cm, both lobes of the liver involved and duration of symptoms >1 week and advanced age.

Abscesses smaller than 5cm size were treated with parenteral antibiotic therapy while those larger than 5cm size were treated by image guided percutaneous aspiration/drainage.

Large abscesses>10cm and multi-loculated abscesses with exaggerated necrotic process were managed by open surgery.

Hepatic abscesses, both pyogenic and amoebic, becomes the important cause of morbidity and mortality in tropical countries. Although the primary

mode of treatment of amoebic liver abscess is medical, amoebic abscesses (15%) are not responding to medical therapy. Secondary bacterial infection occurs in 20% of amoebic liver abscesses. Thus drainage may be required in those cases with amoebic liver abscesses. For most intra abdominal abscesses percutaneous drainage is now considered as the standard treatment of choice.

Ultrasound & CT are the mainstay in the diagnostic modalities for hepatic abscess. The sensitivity of ultrasound in diagnosing hepatic abscess is around 80-95%. The sensitivity of CT abdomen in diagnosing hepatic abscess is around 95-100%.

Factors independently associated with poor outcome in Amoebic Liver Abscess are 1)elevated serum bilirubin (>3.5mg/dl),

2) Encephalopathy,

3) Hypoalbuminaemia (<2.0gm/dl),

4) Multiple abscess cavities,

5) Abscess volume greater than 500ml,

6) Anaemia& diabetes.

ANATOMY

General Description and Topography of Liver

The liver is a solid gastrointestinal organ whose mass (1.2kg-1.6kg) largely occupies the right hypochondrium and part of epigastrium.

The lower margin of liver coincides with costal margin and the superior surface is draped over by the diaphragm.

Most of the right liver and most of the left liver is covered by the thoracic cage.

The liver extends superiorly to the height of the 5^{th} rib on the right and the 6^{th} rib on the left. Posterior surface of the liver straddles the inferior vena cava (IVC).

A wedge of liver extends to the left half of the abdomen across the epigastrium to lie just above the anterior aspect of the stomach and under the central and left diaphragm.

The superior surface of the liver is convex and lies underneath the diaphragm, whereas the inferior surface is somewhat concave and extends upto sharp anterior border.

The liver is invested in the peritoneum in all areas except

1. The gallbladder bed,
2. The porta hepatis and
3. Posteriorly on either side of the IVC in two wedge-shaped areas (The bare area of liver to the right of IVC).

Ligaments in relation to Liver

The peritoneal reflections on the liver surface are referred as ligaments.

1. Coronary and triangular ligament

The diaphragmatic peritoneal duplications are referred to as the coronary ligament, whose lateral margins on either side are

1. Right triangular ligament and
2. Left triangular ligament.

2. Falciform ligament

From the center of the coronary ligament emerges the falciform ligament, which extends anteriorly as a thin membrane connecting the liver surface to the diaphragm, abdominal wall, and umbilicus.

3. Ligamentum teres

The ligamentum teres (the obliterated umbilical vein) runs along the inferior aspect of the falciform ligament from the umbilicus to the umbilical fissure.

The umbilical fissure is on the inferior surface of the left liver and contains the left portal triad.

The falciform ligament, the most obvious surface marking of the liver, historically was used to mark the division of the right and left lobes of the liver in early descriptions of hepatic anatomy.

However the description is inaccurate and it has minimal utility to the hepatobiliary surgeon.

4. Ligamentum venosum

On the posterior surface of left liver, running from the left portal vein in the porta hepatis toward the left hepatic vein and the IVC, is the ligamentum venosum (obliterated sinus venosus), which also runs in a fissure.

Hepatic arterial and portal venous blood flow enter the liver at the hilum and branch throughout the liver as a single unit that also includes the bile ducts (portal triad). These portal triads are invested in a peritoneal sheath that invaginates at the hepatic hilum. Venous drainage is through hepatic veins that empty directly into the IVC.

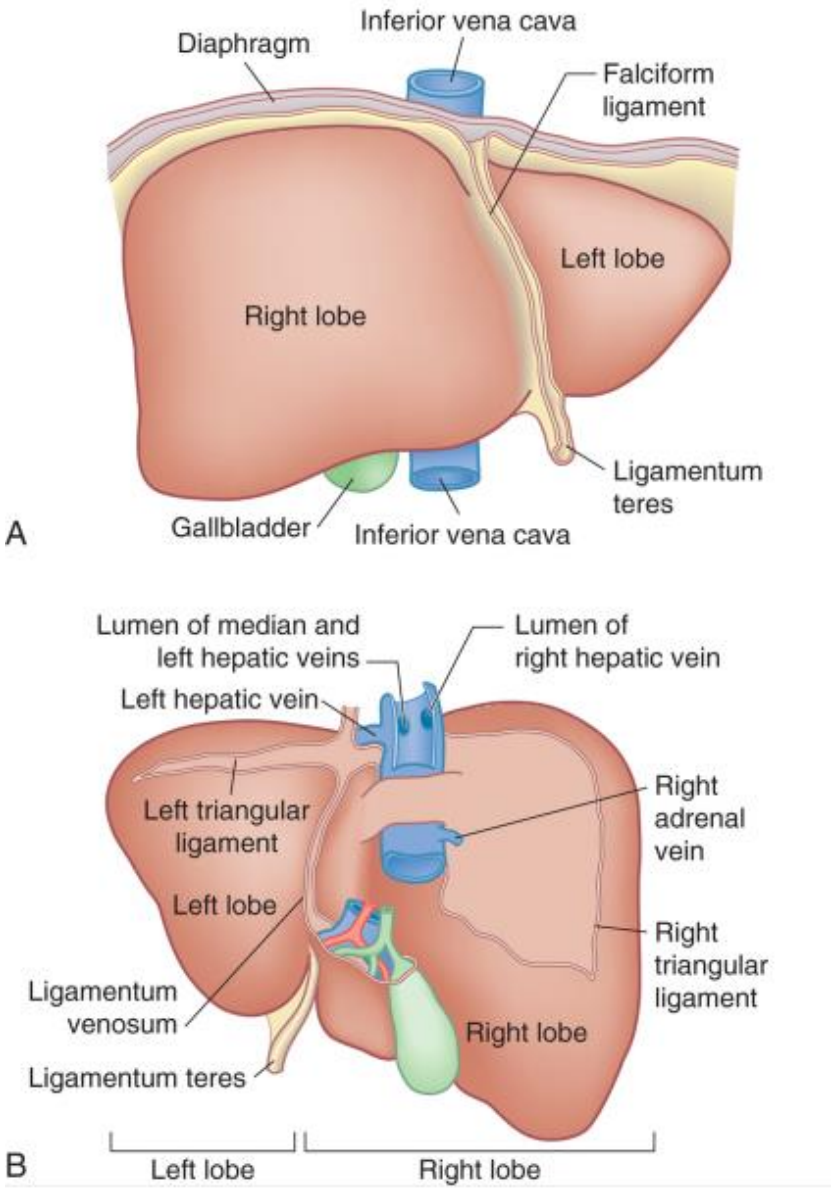


Figure.1

A, Historically, the liver was divided into right and left lobes by the external marking of the falciform ligament. On the inferior surface of the falciform ligament, the ligamentum teres can be seen entering the umbilical fissure. **B,** The posterior and inferior surface of the liver is shown. The liver embraces the inferior vena cava (IVC) posteriorly in a groove. The lumens of the three major hepatic veins and the right adrenal vein can be seen directly

entering the IVC. The bare area, bounded by the right and left triangular ligaments, is illustrated. To the left of the IVC is the caudate lobe, which is bounded on its left side by a fissure containing the ligamentum venosum. The lesser omentum terminates along the edge of the ligamentum venosum; thus, the caudate lobe lies within the lesser sac, and the rest of the liver lies in the supracolic compartment. A layer of fibrous tissue can be seen bridging the right lobe to the caudate lobe posterior to the IVC, encircling the IVC. This ligament of tissue must be divided on the right side when mobilizing the right liver off of the IVC

Normal Development and Embryology

The liver primordium is formed in the 3rd week of gestation as an outgrowth of endodermal epithelium (known as the hepatic diverticulum, or liver bud). It arises from foregut.

The connection between the duodenum and hepatic diverticulum narrows to form the bile duct, and the gallbladder and cystic duct are formed by the out pouching from the bile duct.

Hepatic cells develop cords and intermingle with the umbilical and vitellineveins to form hepatic sinusoids. Simultaneously, Kupffercells ,hematopoietic cells, and connective tissue form from mesoderm of the septum transversum.

The septum transversum (mesodermal origin) connects the liver to the anterior abdominal wall and to the foregut. As the liver protrudes into the abdominal cavity, these structures are stretched into thin membranes,

ultimately forming the falciform ligament and the lesser omentum, respectively.

The mesoderm on the surface of the developing liver differentiates into visceral layer of the peritoneum except superiorly, where contact between the liver and mesoderm (future diaphragm) is maintained, forming a bare area devoid of visceral peritoneum.

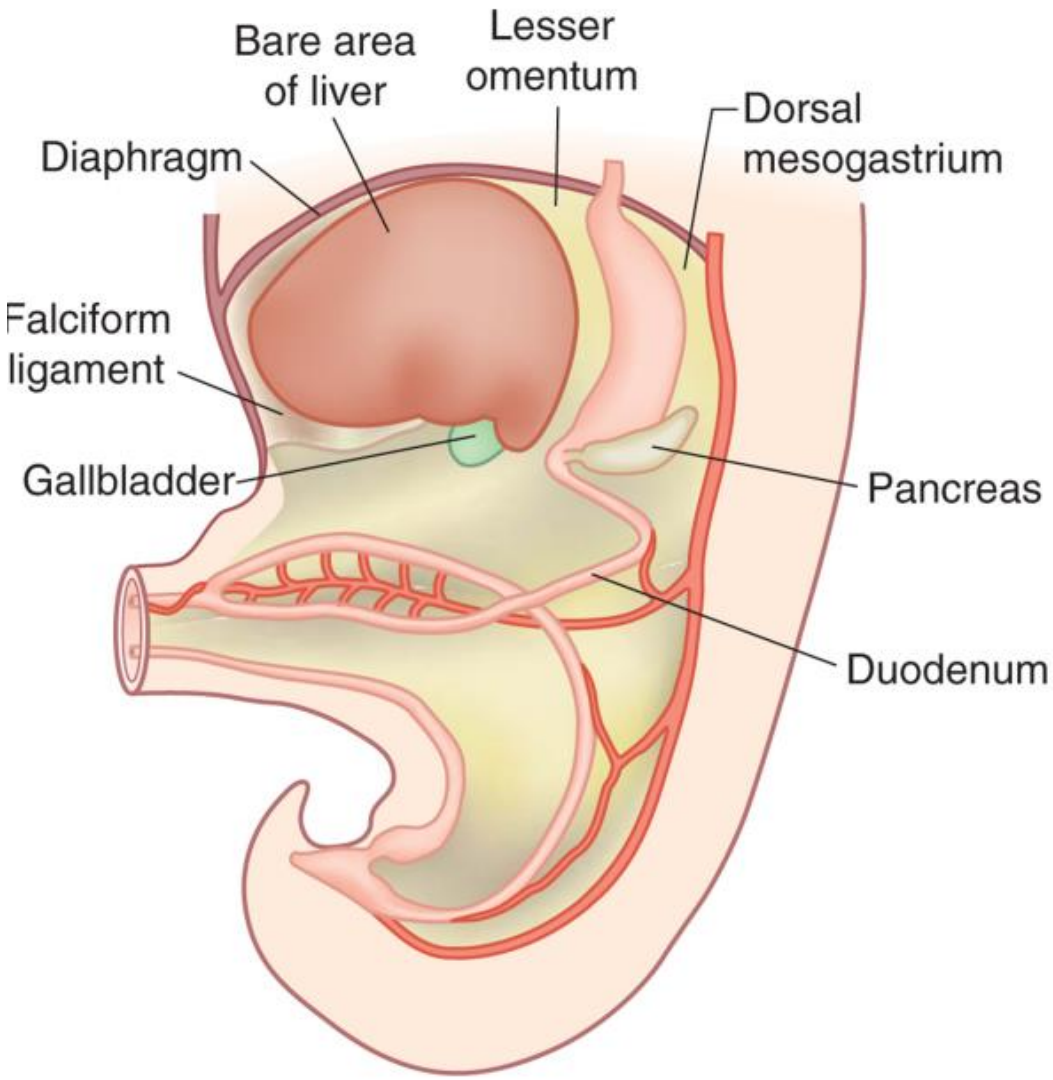


Figure.2

An approximately 36-day-old embryo is shown. The extensions of the septum transversum can be seen developing as the liver protrudes into the abdominal cavity, stretching out and forming the lesser omentum and the falciform ligament. The liver is completely invested in visceral peritoneum except for a portion next to the diaphragm known as the bare area

The primitive liver plays a central role in fetal circulation.

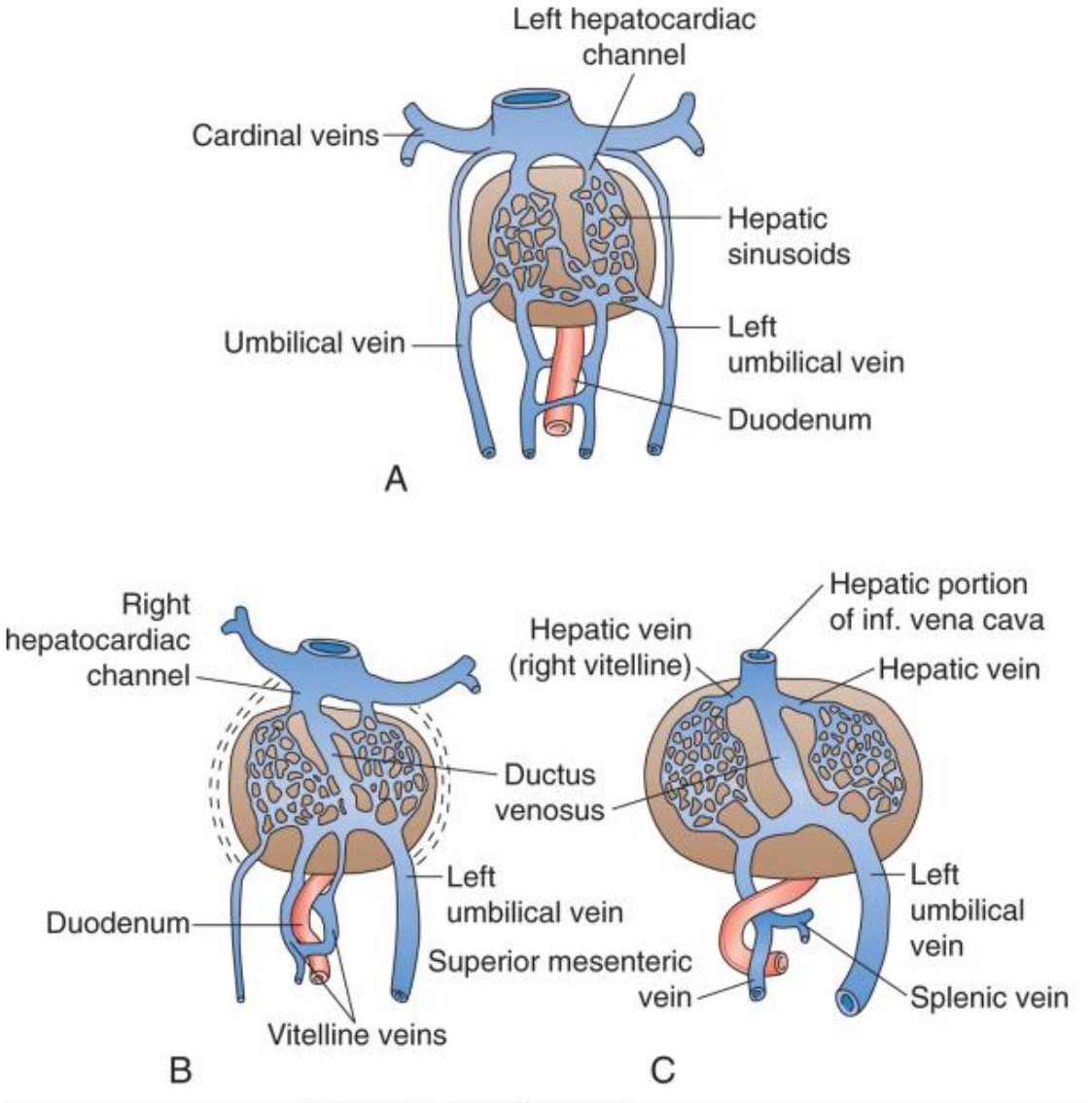


Figure.3 Shows the development of portal venous system

The fetal liver is one of the most important organ of hematopoiesis. In the 10th week of gestation, the liver is 10% of the body weight, which is due to developing hepatic sinusoids and active hematopoiesis.

During the last 8 weeks of intrauterine life, hepatic hematopoiesis decreases, and the liver weight is decreased to 5% of total body weight.

By the 12th week of gestation, bile forms in hepatic cells, along with the simultaneous development of the gall-bladder and bile duct, allowing drainage of bile into the foregut.

Functional Anatomy

Historically, the liver was divided into left and right lobes by the obvious external landmark of the falciform ligament. Not only was this description oversimplified, but it was also anatomically incorrect in relationship to the blood supply to the liver.

Later, more accurate descriptions of the liver lobar anatomy were developed. The liver was separated into right and left lobes determined by portal and hepatic vein branches.

Briefly, a plane without any surface markings running from the gall-bladder to the left side of the IVC (known as the cantlie's line or portal fissure) divided the liver into

1. The right and
2. The left lobes.

The right lobe was further divided into

1) Anterior and

2) Posterior segments.

The left lobe was divided into a

1) A medial segment (also known as the **quadrate lobe**) that lies to the right side of the umbilical fissure and falciform ligament and

2) A lateral segment lying to the left side of the umbilical fissure.

This system, although anatomically more correct, is only sufficient for mobilization of the liver and simple hepatic procedures.

The functional anatomy of the liver is composed of VIII segments, each of which is supplied by a single portal pedicle (triad) composed of

1) Portal vein branch,

2) Hepatic artery branch,

3) Bile duct.

These segments are further organized into 4 sectors that are separated by scissurae containing the three main hepatic veins.

The four sectors are even further organized into the right and left liver (The phrase **right and left liver** is preferable to **right and left lobe** because there is no external mark that allows the identification of the right and left liver).

This system was originally described in 1957 by Woodsmith and Goldburne as well as Couinaud and defines hepatic anatomy as it is most relevant to surgery of the liver. The functional anatomy is more often seen as cross-sectional imaging.

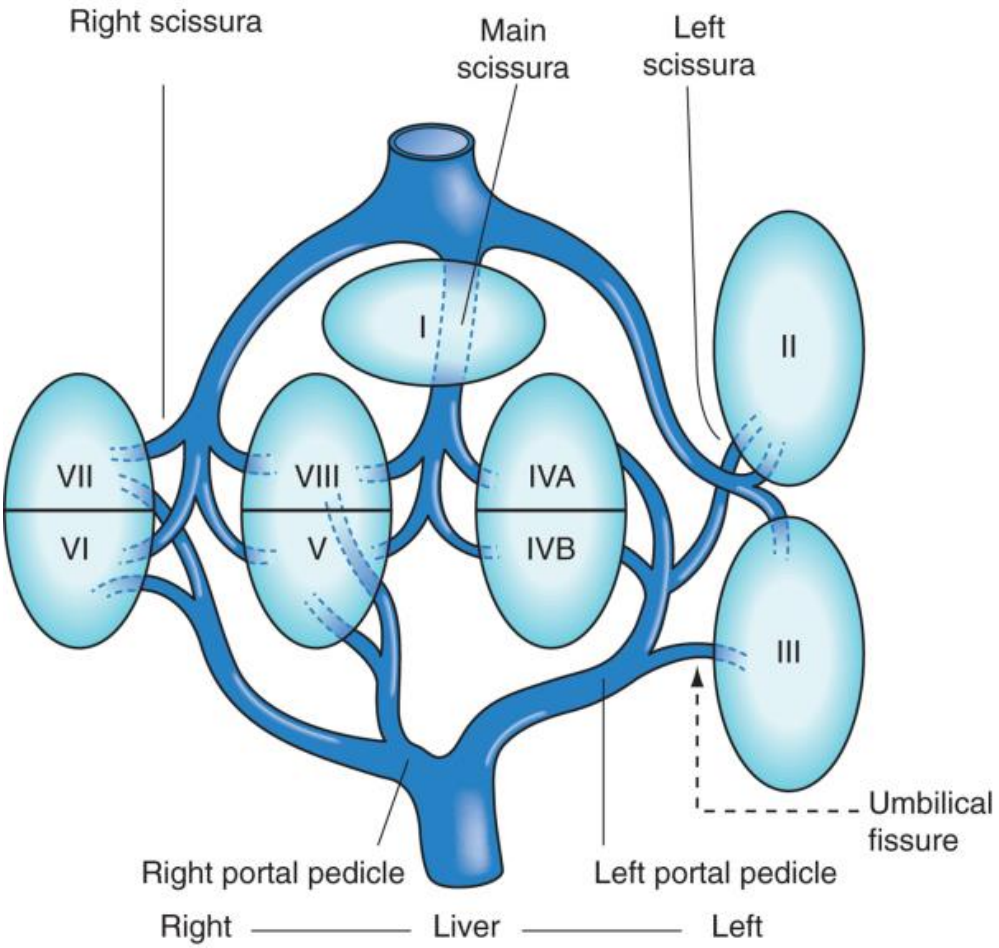


Figure.4

A schematic demonstrating the segmental anatomy of the liver. Each segment receives its own portal pedicle (triad of a portal vein, a hepatic artery, and a bile duct branches). The eight segments are illustrated, and the 4 sectors, divided by the 3 main hepatic veins running in scissurae, are shown. The umbilical fissure (not a scissura) is shown to contain the left portal pedicle

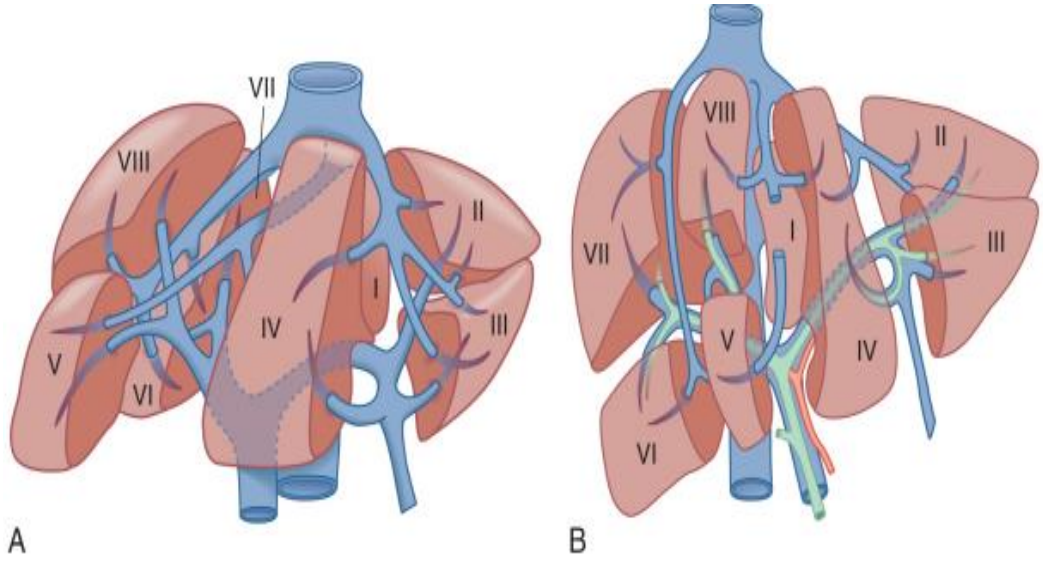


Figure.5

Segmental anatomy of the liver as seen at laparotomy in the anatomic position **(A)** and in the ex vivo position **(B).**

The main scissura which contains the middle hepatic vein, which runs in a direction anteroposteriorly from the gallbladder fossa to the left side of the inferior vena cava and divides the liver into right and left hemi-livers. The line of the main scissura is also known as Cantlie's line.

Right liver

The right scissura divides the liver into an anterior (segments V and VIII) and posterior (segments VI and VII) sector by , which contains right hepatic vein.

The right portal pedicle, composed of the right portal vein, hepatic artery, and bile duct, splits into right anterior and posterior pedicles that supply the segments of the anterior and posterior segments.

Left liver

The left liver has a visible fissure along its inferior surface called the umbilical fissure.

The ligamentum teres (containing the remnant of the umbilical vein) runs into this fissure. The falciform ligament is contiguous with the umbilical fissure and ligamentum teres. The umbilical fissure is *not* a scissura, does not contain a hepatic vein, and in fact, contains the left portal pedicle (triad containing the left portal vein, hepatic artery, and bile duct), which runs in this fissure, branching to feed the left liver.

The left scissura runs in a direction posterior to the ligamentum teres and which contains the left hepatic vein. The left scissura splits the liver into an anterior (segments III and IV) and posterior (segment II—the only sector composed of a single segment) sector.

At the hilum of the liver, the right portal triad has a short extrahepatic course of about 10mm to 15 mm before entering the substance of the liver and branching into anterior and posterior sectoral branches. The left portal triad, however, has a long extrahepatic course of up to 3 or 4 cm and runs transversely along the base of segment IV in a peritoneal sheath that is the upper end of lesser omentum.

The left portal triad, as it runs along the base of segment IV, is separated from the liver substance by connective tissue known as the hilar plate. The continuation of the left portal triad runs anteriorly and caudally in the umbilical fissure and gives branches to the segments II and III and recurrent branches to the segment IV.

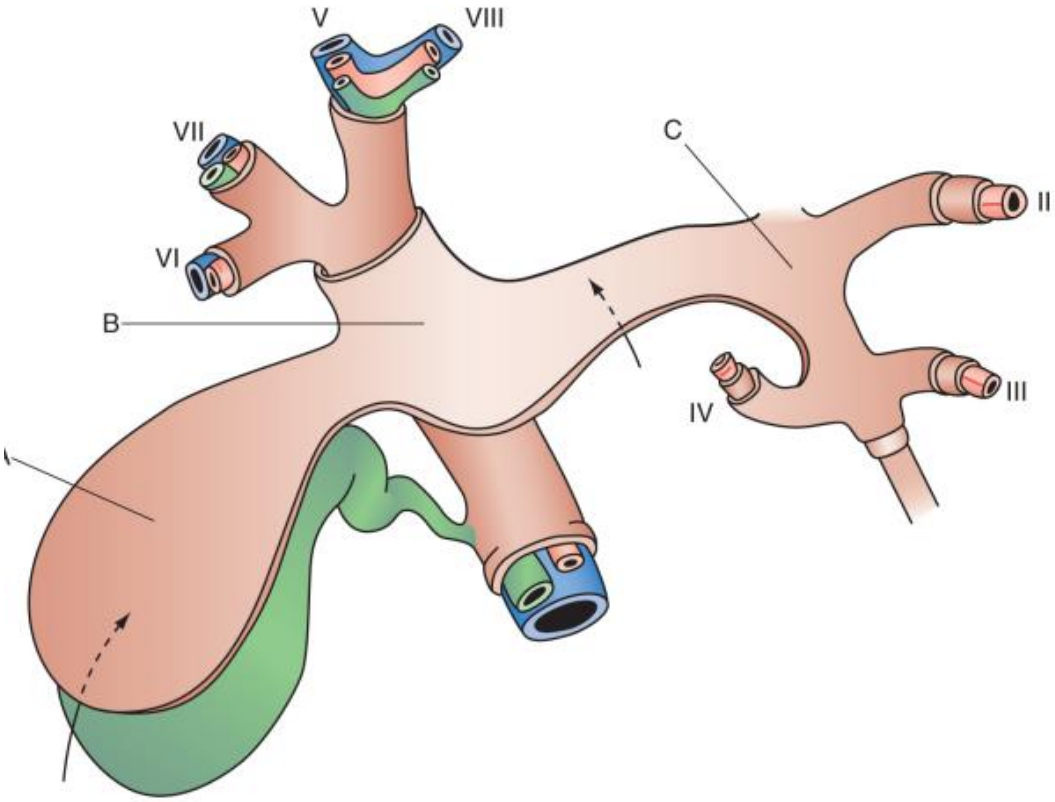


Figure.6

The plate system is illustrated. A, The cystic plate between the gallbladder and the liver. B, The hilar plate at the confluence of bile ducts at the base of segment IV. C, The umbilical plate is present above the umbilical portion of the portal vein. The arrows show plane of the dissection of cystic plate for cholecystectomy and the hilar plate for exposure of the confluence hepatic duct and the main left hepatic duct.

Caudate lobe

The segment I (caudate lobe) is the dorsal portion of the liver and embraces the Inferior Vena Cava on its posterior surface and lies posterior to left portal triad inferiorly and left and middle hepatic veins superiorly.

The most part of the caudate lobe is on the left side of the IVC, but inferiorly, it traverses between the left portal triad and IVC, where it fuses to the right liver (segments VI and VII). This part of the caudate lobe is known as the caudate process (right portion). The left portion of caudate lobe lies in the lesser omental bursa and is covered anteriorly by the gastrohepatic ligament (lesser omentum) that separates it from segments II and III anteriorly. The gastrohepatic ligament attaches to the ligamentumvenosum (sinus venosus remnant) along the left side of the left portal triad.

The vascular inflow and biliary drainage to the caudate lobe comes from both right and left systems. The caudate process largely derives its portal venous supply from the bifurcation of the main portal vein or the right portal vein, whereas the left part of the caudate derives its portal venous supply from the left main portal vein.

The biliary drainage and the arterial supply of the right portion are generally through the right posterior sectoral system and the left portion through the left main vessels.

The hepatic venous drainage of the caudate is somewhat unique in that multiple small veins drain posteriorly into the IVC directly.

The posterior edge of left side of caudate terminates into a fibrous component that attaches to the crura of the diaphragm and also runs posteriorly, wrapping behind the IVC and attaching to segment VII of the right liver.

IVC ligament

Up to 50% of the time, this fibrous component is composed either partially or completely of liver parenchyma, and thus liver tissue may completely encircle the IVC. This important structure is known as the IVC ligament and is important when mobilizing the right liver or the caudate lobe off of the IVC.

Riedel's lobe

A tongue of tissue extending inferiorly off of the right liver.

Portal Vein

The 75% of hepatic blood flow is through portal venous system, and although it is postcapillary and largely deoxygenated, its large-volume flow rate provides 50% to 70% of the liver's oxygenation.

The lack of valves in the portal venous system provides a system that can accommodate high flow at low pressure because of the low resistance and allows measurement of portal venous pressure anywhere along the system.

The portal vein forms behind the pancreas neck at the confluence of the splenic vein and superior mesenteric vein at the height of the L2 vertebra. The

length of the main portal vein ranges from 5.5 to 8 cm, and its diameter is usually about 1 cm.

Cephalad to its formation behind the pancreas neck, the portal vein runs behind the 1st portion of the duodenum and into the hepatoduodenal ligament, where it runs along the right border of the lesser omentum, usually posterior to the bile duct and hepatic artery. The portal vein divides into main right and left branches at the hilum of the liver.

The portal vein's left branch runs transversely along the base of segment IV and into the umbilical fissure, where it gives off branches to segments II and III and feedback branches to segment IV.

The left portal vein also gives off posterior branches to the left side of the caudate lobe.

The right portal vein has a short extrahepatic course and usually enters the substance of the liver, where it splits into anterior and posterior sectoral branches. These sectoral branches can occasionally be seen extrahepatically and can come off the main portal vein before its bifurcation.

There is usually a small branch off the right portal vein or at the bifurcation that comes off posteriorly to supply the caudate process.

9).

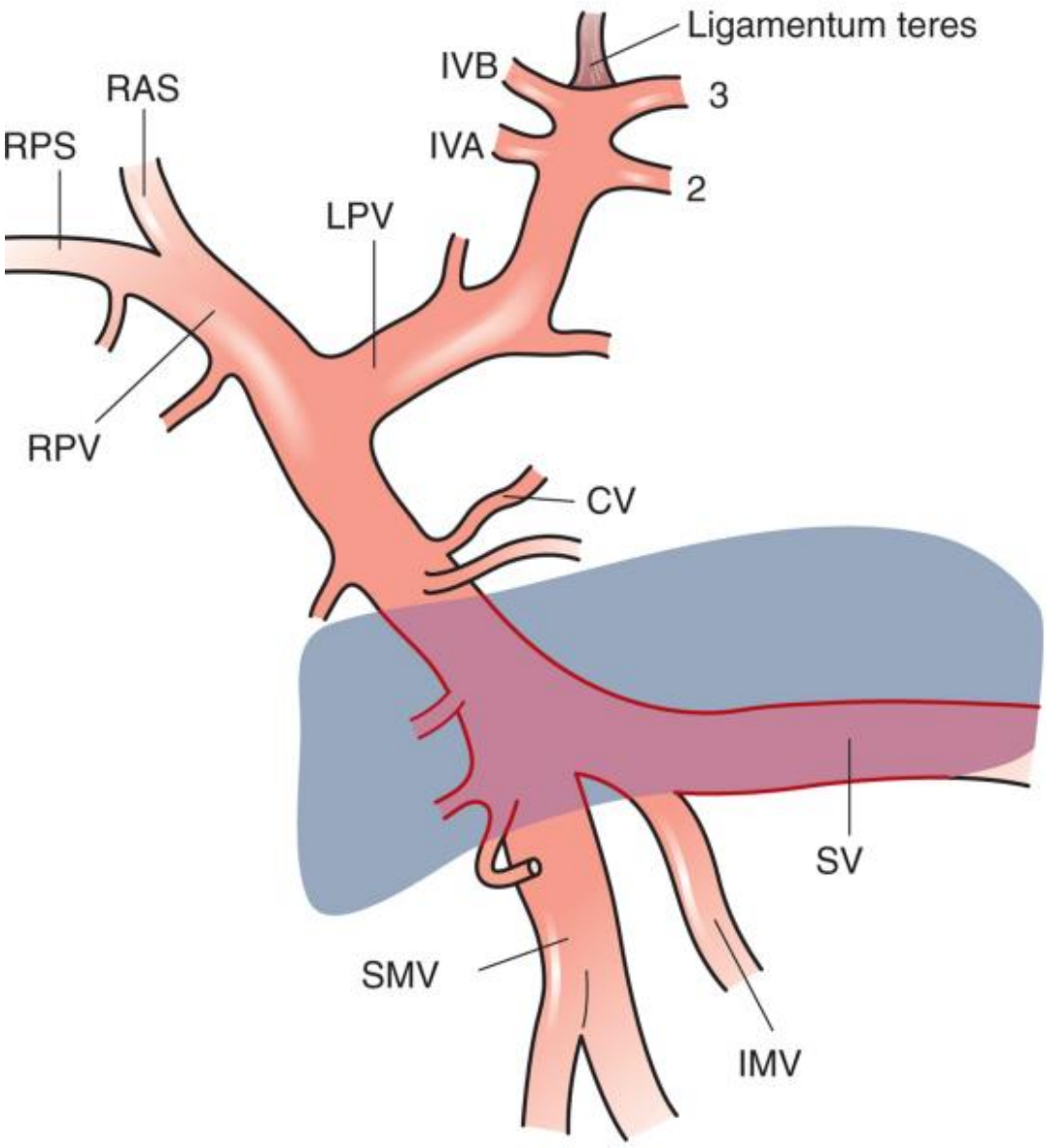


Figure.7

The anatomy of the portal vein is demonstrated. The superior mesenteric vein (SMV) joins the splenic vein (SV) posterior to the pancreas neck (shaded) to form the portal vein. Note the entrance of the inferior mesenteric vein (IMV) into the splenic vein—the most common anatomic

arrangement. In its course superiorly in the edge of the lesser omentum posterior to the hepatic artery and the common bile duct, the portal vein receives venous effluent from the coronary vein (CV). At the hepatic hilum, the portal vein bifurcates into a larger right portal vein and a smaller left portal vein. The left portal vein runs transversely at the base of segment IV and enters the umbilical fissure to supply the segments of the left liver. Just before the umbilical fissure, the left portal vein (LPV) usually gives off a sizable branch to the caudate lobe. The right portal vein (RPV) enters the substance of the liver and splits into a right anterior sectoral (RAS) and right posterior sectoral (RPS) branch. It also gives off a posterior branch to the right side of the caudate lobe/caudate process.

Porto-systemic shunts

There are a number of connections between the portal venous system and the systemic venous system. Under conditions of high portal venous pressure, these portosystemic connections may enlarge secondary to collateral flow. The most significant portosystemic collateral locations are listed:

1. Submucosal veins of the distal esophagus and proximal stomach, which receive portal flow from the short gastric veins and the left gastric vein and can result in varices with the potential for intestinal hemorrhage
2. Umbilical and abdominal wall veins, which recanalize from flow through the umbilical vein in the ligamentum teres, resulting in caput medusa.
3. Superior hemorrhoidal plexus, which receives portal flow from inferior mesenteric vein tributaries and yields large haemorrhoids.

4. Other retroperitoneal communications yielding collaterals that can make abdominal surgery hazardous

The anatomy of the portal vein and its branches is relatively constant and has much less variation than the ductal and hepatic arterial system.

The portal vein is rarely found anterior to the neck of the pancreas and the duodenum. Entrance of the portal vein directly into the vena cava has also been described.

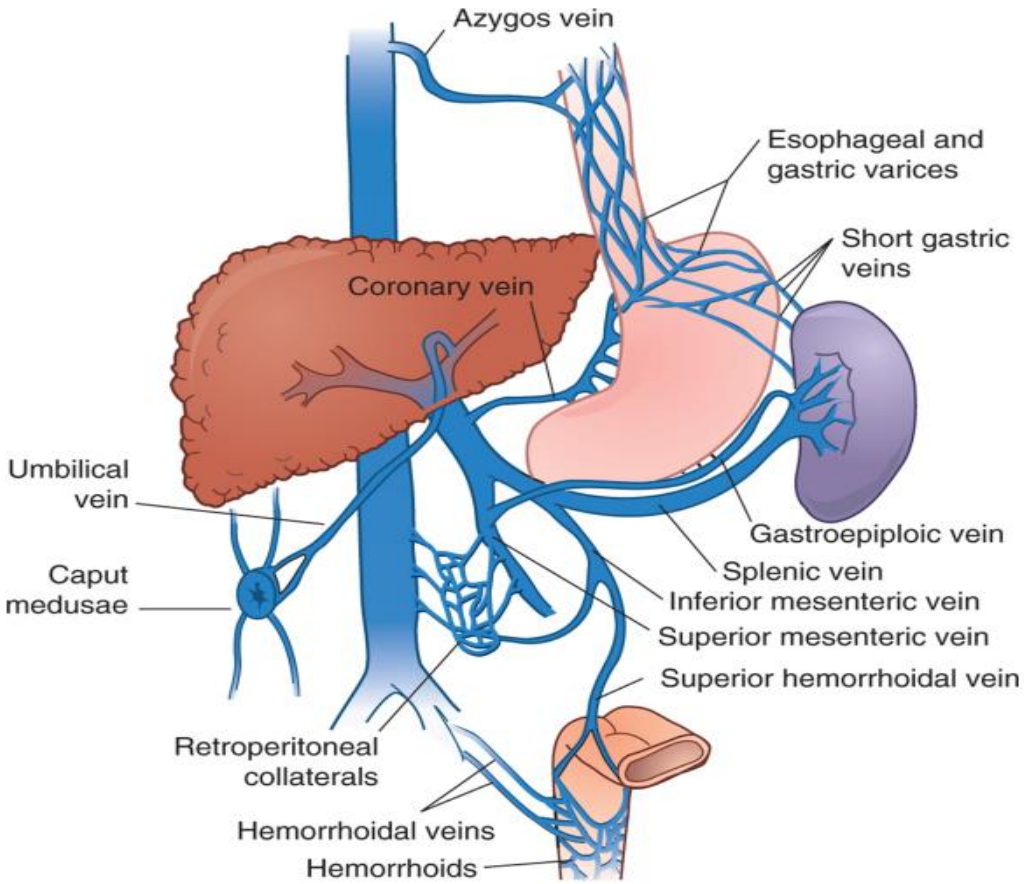


Figure.8

Portosystemic collateral (shunting) pathways develop where the portal venous and systemic venous systems are in close apposition.

Hepatic Artery

The hepatic artery, representing high-flow oxygenated systemic arterial flow, provides about 25% of the blood flow to the liver and 30% to 50% of its oxygenation.

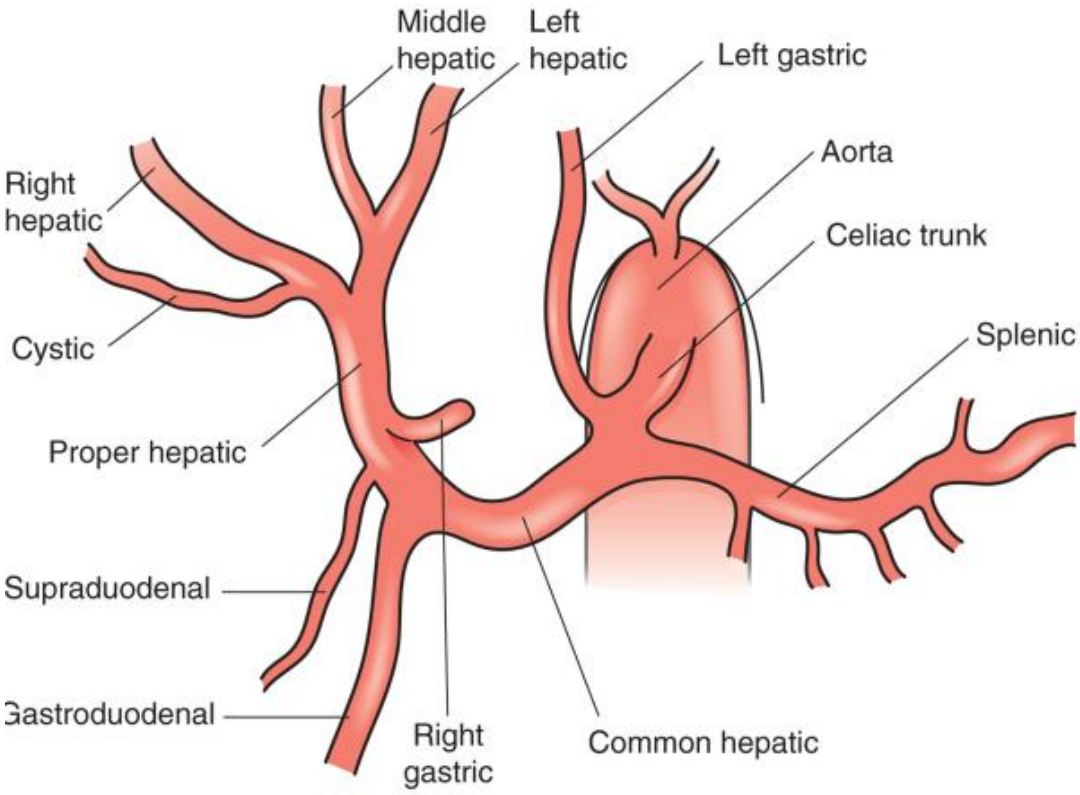


Figure.9

The anatomy of the celiac axis and hepatic arterial system is illustrated. The celiac axis, lies just below the diaphragmatic hiatus, divides into1)the splenic,2) left gastric, and 3)common hepatic arteries. The common hepatic artery runs in the right direction and turns superiorly toward the hilum. At the point of this turn, the proper hepatic artery is formed after giving the gastroduodenal artery. The hepatic artery proper gives off right and left hepatic arteries in the hilum. Note the middle hepatic artery off of the

proximal part of the left hepatic artery, which goes on to supply segment IV. The cystic artery most commonly comes off the right hepatic artery within the triangle of Calot.

Hepatic Veins

The three major hepatic veins drain from the superior and posterior surface of the liver directly into the IVC.

The right hepatic vein runs in the right scissura and drains most of the right liver after a short (1-cm) extrahepatic course into the right side of the IVC.

The middle and left hepatic veins usually join intrahepatically and enter the left side of the IVC as a single vessel, although they may drain separately. The left hepatic vein runs in the left scissura and drains segments II and III, and the middle hepatic vein runs in the portal scissura (between segment IV and the anterior sector of the right liver) draining segment IV and some of the anterior sector of the right liver.

The umbilical vein is an additional vein that runs under the falciform ligament, between the left and middle veins, and usually empties into the left hepatic vein.

Multiple small venous branches from the right posterior sector and caudate lobe drain posteriorly directly into the IVC. There is often a venous tributary from caudate that drains superiorly into the left hepatic vein.

Biliary System

The intrahepatic bile ducts are terminal branches of the main right and left hepatic ductal branches that invaginate Glisson's capsule at the hilum along with corresponding portal vein and hepatic artery branches, forming the peritoneal covered portal triads. Along these intrahepatic portal pedicles, the bile duct branches are usually superior to the portal vein, whereas the hepatic artery branches run inferiorly.

Left Hepatic Duct

The left hepatic bile duct drains segments 2,3and 4, which constitute the left liver. The intrahepatic ductal branches of the left liver join to form the main left duct at the base of the umbilical fissure, where the left hepatic duct courses transversely across the base of segment 4 to join the right hepatic duct at the hilum. In its transverse portion, the left hepatic duct drains 1 to 3 small branches from segment 4.

Right Hepatic Duct

The right hepatic duct drains the right liver and is formed by the joining of the anterior sectoral duct (draining segments 5 and 8) and the posterior sectoral duct (draining segments 6 and 7). The posterior sectoral duct runs in a horizontal and posterior direction, whereas the anterior sectoral duct runs vertically. The main right hepatic duct bifurcates just above the right portal vein. The short right hepatic duct meets the longer left hepatic duct, forming the confluence anterior to the right portal vein, constituting the common hepatic duct.

The segment I (caudate lobe) has its own biliary drainage, which is usually through both right and left systems, although in up to 15% of cases, drainage is through the left system only, and in 5%, it is through the right system only.

Common bile duct

The common hepatic duct drains inferiorly, and below the takeoff of the cystic duct is referred to as the common bile duct.

The common bile or hepatic duct runs along the right side of the hepatoduodenal ligament to the right side of the hepatic artery and anterior to the portal vein. The common bile duct continues inferiorly (usually ~10-15 cm in length and 6 mm in diameter) behind the 1st part of the duodenum and into the head of the pancreas in an inferior and slightly rightward direction.

The intrapancreatic distal common bile duct then joins with the main pancreatic duct (of Wirsung), with or without a common channel, and enters the second portion of the duodenum through the major duodenal papilla of Vater.

At the choledochoduodenal junction, a complex muscular complex known as the sphincter of the oddi regulates bile flow and prevents reflux of contents of the duodenum into the biliary tree. There are three major parts to this sphincter: the sphincter choledochus, which is a circular muscle that serves to regulate bile flow and the filling of the gallbladder; the pancreatic sphincter, present to variable degrees, which surrounds the intraduodenal

pancreatic duct; and the sphincter ampullae, made up of longitudinal muscle, which serves to prevent duodenal reflux.[4]

Gallbladder

The gallbladder is a biliary reservoir that lies against the inferior surface of segments IV and V of the liver, usually making an impression against it. A peritoneal layer covers most of the gallbladder except for the portion adherent to the liver. Where the gallbladder is adherent to the liver, there is a layer of fibroconnective tissue known as the **cystic plate**, which is an extension of the hilar plate. Variable in size, but usually about 10 cm long and 3 to 5 cm wide, the gallbladder is composed of a1) fundus, 2) body, 3) infundibulum, and4)neck that ultimately empties into the cystic duct. The fundus usually projects just slightly beyond the liver edge anteriorly and when folded on itself is described as a **Phrygian cap**. Continuing toward the bile duct, the body of the gallbladder is usually in close proximity to the 2nd portion of the duodenum and the transverse colon. The infundibulum (or Hartmann's pouch) hangs forward along the free edge of hepatoduodenal ligament and can fold in front of the cystic duct. The portion of gallbladder between the infundibulum and the cystic duct is the neck of the gallbladder. The cystic duct is variable in its length, its course, and its insertion into the main biliary tree. The first portion of the cystic duct is usually tortuous and contains mucosal duplications, referred to as the **fold of Heister,** that regulate the filling and emptying of the gallbladder. Most commonly, the cystic duct joins the common hepatic duct to form the common bile duct.

Blood supply of biliay system

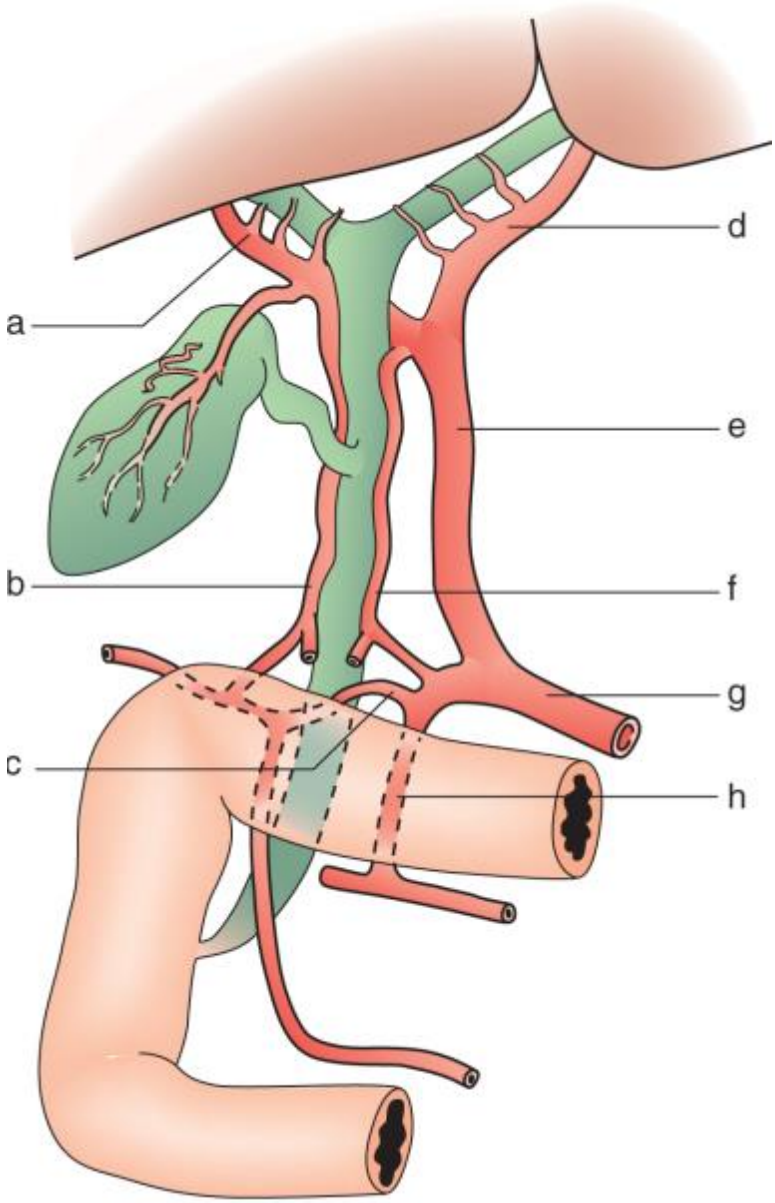


Figure.10

The blood supply to the CBD and CHD is illustrated. a) The Right hepatic artery; b) artery at 9'o clock; c) the retroduodenal artery; d) the left hepatic artery; e) the proper hepatic artery; f) artery at 3'o clock; g) the common hepatic artery; h, the gastroduodenal artery.

Nerves supply

The innervation of the liver and biliary tract is by

1) sympathetic fibers originating from T7 through T10

2) parasympathetic fibers from both vagal nerves.

The sympathetic fibers pass through celiac ganglia before giving off postganglionic fibers to the liver and bile ducts. The right-sided celiac ganglia and right vagal nerve form an anterior hepatic plexus of nerves that runs along the hepatic artery. The left-sided celiac ganglia and left vagal nerve form a posterior hepatic plexus that runs posterior to common bile duct and portal vein. The hepatic arteries are supplied by sympathetic fibers, and the gallbladder and extrahepatic bile ducts receive innervation from both sympathetic and parasympathetic fibers. The clinical significance of these nerves is still not well understood. Pain elicited from acute distention of the liver (and thus the liver capsule) is referred to the right shoulder because of innervation of the capsule from the phrenic nerve.

Lymphatics drainage

Most lymph node drainage from the liver is to the lymph nodes of hepatoduodenal ligament. Lymphatic drainage usually continues along the hepatic artery to the celiac lymph nodes and from there to the cisterna chyli. Lymphatic drainage can also follow the hepatic veins to lymph nodes in the area of the suprahepatic IVC and through the diaphragmatic hiatus. The lymphatic drainage of the gallbladder and most of the extrahepatic biliary tract

is generally into the lymph nodes of the hepatoduodenal ligament. This drainage can also follow along the hepatic artery to the celiac lymph nodes, but can also run to lymph nodes behind the head of the pancreas or in the inter-aortocaval groove.

MICROSCOPIC ANATOMY OF THE LIVER

The Functional Unit of Liver

The organization of liver parenchyma into microscopic functional units has been described in a number of ways and referred to as an **acinus** or a **lobule**. This was originally described by Rappaport and was modified by Matsumoto and Kawakami.[6]

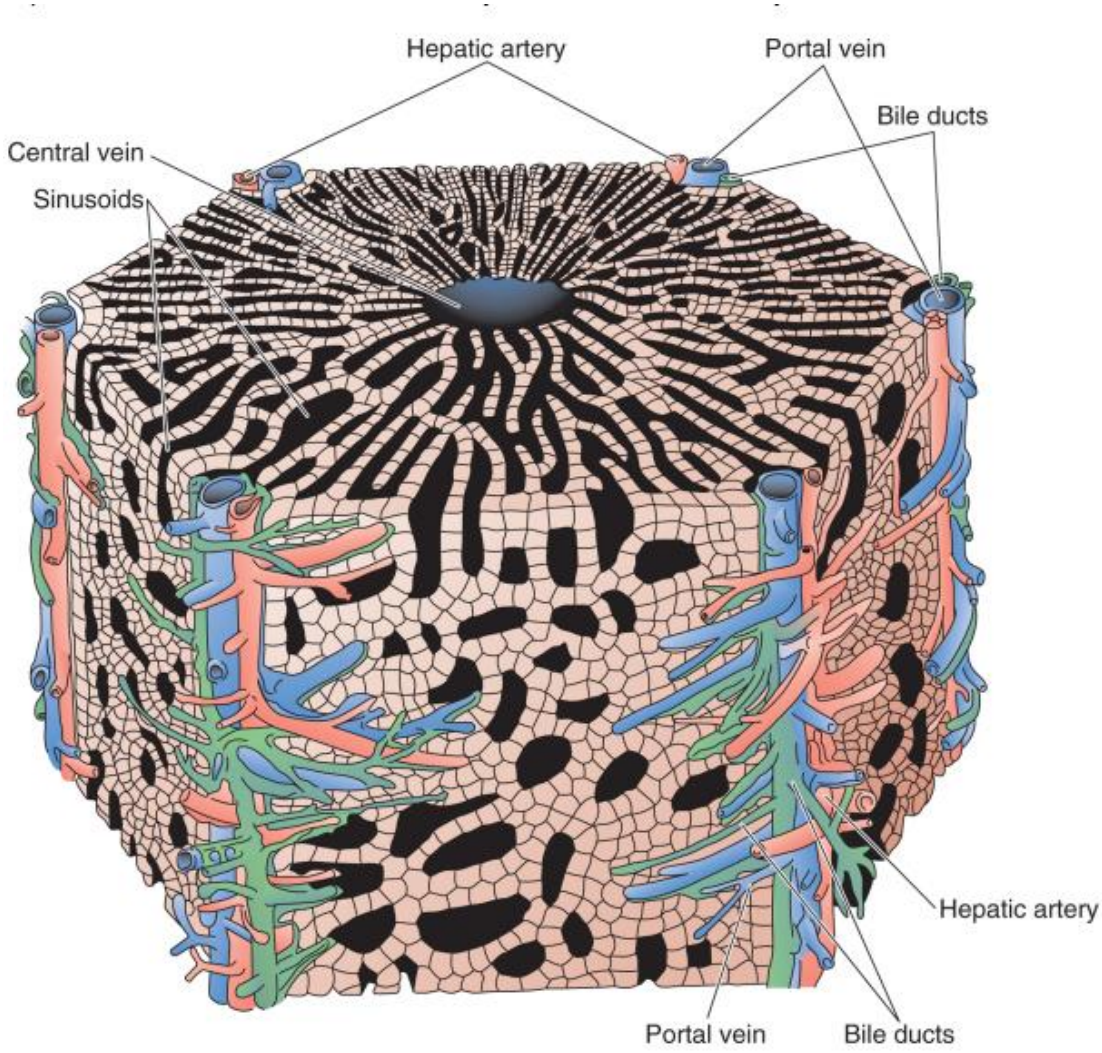


Figure.11

Schematic illustration of a hepatic lobule seen as a three-dimensional polyhedral unit. The terminal portal triads (the portal vein, hepatic artery, and bile duct) are at each corner and give off branches along the sides of the lobule. Hepatocytes are in single-cell sheets with sinusoids presenton either end aligned radially toward a central hepatic venule.

Between the portal triad and the central hepatic venule in the centre of the lobule, there are three zones that differ in their enzymatic makeup and exposure to nutrients and oxygenated blood. Although there is debate as to the shape of these zones and their relationship to the basic lobular unit, in general, zones 1 through 3 fan out from the terminal portal triad toward the central hepatic venule.

Zone 1, the periportal zone, is exposed to an environment rich in nutrients and oxygen. Zones 2 (intermediate zone) and Zone 3 (perivenular zone) are exposed to environments less rich in oxygen and nutrients.

The cells of the different zones differ enzymatically and respond differently to toxin exposure as well as hypoxia. This anatomic arrangement also explains the phenomenon of centrilobular necrosis from hypotension because zone 3 is the most susceptible to decreases in oxygen delivery.

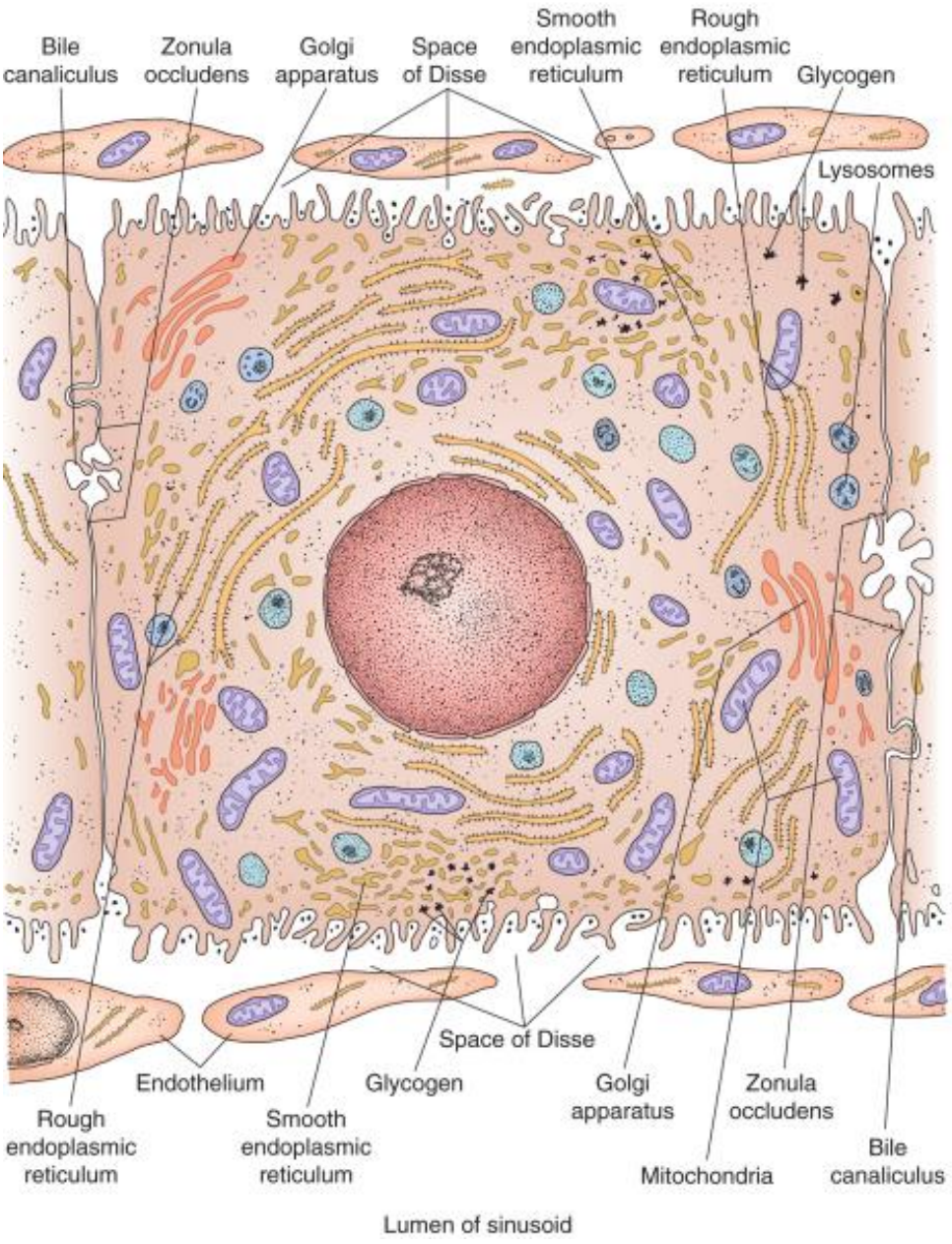


Figure.12

Hepatocytes –microscopic anatomy

FUNCTIONS OF THE LIVER

1. Removal of gut endotoxins and foreign antigens-liver acts as the first filter.
2. Drug and hormone metabolism.
3. Formation of bilirubin and its metabolism.
4. Formation of urea from protein catabolism.
5. Glucose metabolism, glycolysis, and gluconeogenesis.
6. Clotting factors synthesis.
7. pH balance and correction of lactic acidosis.
8. Maintaining body temperature.
9. Storage of vit. B12, vit. A, Cu, Fe

LIVER FUNCTION TESTS (LFT)

1. Serum bilirubin which includes both direct and indirect. Test is known as **van den Bergh's test.**
2. Serum albumin, globulin and A : G ratio; serum albumin is the indicator of chronic liver disease.
3. Prothrombin time: Normal value is **12-16 seconds.**

Difference between control and test more than 4 seconds or test being more than 1½ times the control is significant. When it is altered it is corrected by injecting vitamin K, 10 mg IM for 5 days or by fresh frozen plasma (FFP)—**For cell synthesis.**

4. Alkaline phosphatase - ***Secretary function.***
5. Aspartate amino transaminase *5*-**40 IU/litre** (AST, SGOT)-**signifies inflammation**.
6. Alanine transaminase *5*-**40 IU/litre** (ALT, SGPT).
7. 5 nucleotidase.
8. Gamma glutamyl transpeptidase (GGT) 10-48 IU/L.
9. Immunological tests: Antimitochondrial or antinuclear antibodies.
10. AFP.
11. Specific tests: (a) For haemochromatosis-serum iron, total iron binding capacity, serum ferritin. (b) Wilson's disease: Serum copper, urinary copper, serum ceruloplasmin.

12. Flurodeoxy glucose—positron emission tomography (FDG-PET): It is to find out the uptake of labeled glucose which varies in different diseases of liver, i.e., benign, malignant and inflammatory.
13. Technetium-99 m labelled radioisotope scan shows the uptake and excretion of bile.
14. A sulphur colloid liver scan shows specifically Kupffer cell activity. Sulphur colloid will not show uptake in adenoma and haemangioma as Kupffer cells are absent in these lesions.
15. Urine for bile salts **(Hay's test**), for bile pigments **(Fouchet's test)** and for urobilinogen **(Ehrlich's aldehyde test).**

Other investigations for liver diseases

1. U/S abdomen
2. Angiography
3. CT Scan
4. PTC
5. ERCP
6. MRI
7. Laparoscopy and laparoscopic U/S
8. Liver biopsy

INFECTIOUS DISEASES

PYOGENIC LIVER ABSCESS

Pathogenesis

The liver is probably exposed to portal venous bacterial loads on a regular basis and clears this bacterial load without problems in the usual circumstance.

The development of a hepatic abscess occurs when the inoculum of bacteria, irrespective of the route of exposure, exceeds the liver's ability to clear it. This results in tissue invasion, and the formation of an organized abscess after neutrophil infiltration.

The potential routes of hepatic exposure to bacteria follow:

1. Biliary tree route.
2. Portal vein route.
3. Hepatic artery route.
4. Direct extension of a nearby focus of infection.
5. Trauma.

1. Bilairy route

Biliary tree infections are presently the most common identifiable cause of hepatic abscess. Biliary obstruction results in bile stasis with the potential for subsequent bacterial infection, colonization, and ascension into the liver.

This process is called as ascending suppurative cholangitis. The nature of biliary obstruction is mostly related to malignancy or stone disease.

In Asia, stones in the hepatic ductal system and cholangitis are a common cause, whereas in the Western world, malignancy causing obstruction is becoming a more predominant factor.The common link between all causes of hepatic abscess from the biliary tree obstruction and bacteria in the biliary tree.

Previous biliary-enteric anastomosis has also been associated with hepatic abscess formation, likely due to unimpeded exposure of the biliary tree to enteric organisms.

2. Portal venous route

The portal venous system drains the most of the gastrointestinal tract, and any infections of the gastrointestinal tract can result in pyelophlebitis (ascending portal vein infection) with exposure of the liver to large amounts of bacteria. The most common causes of pyelophlebitis are appendicitis, diverticulitis, pancreatitis, inflammatory bowel disease, pelvic inflammatory disease, hollow viscus perforation, or omphalitis in the newborn. Hepatic abscess has also been associated with colorectal malignancy.

3. Hepatic artery route

Any systemic infection (pneumonia, endocarditis, osteomyelitis) can result in bacteremia and infection of the liver through the hepatic artery. Hepatic abscess from systemic infections may also reflect an altered immune

response, such as in patients with malignancy, acquired immunodeficiency syndrome, or disorders of granulocyte function. Children with chronic granulomatous disease are particularly susceptible.

4. Direct extension

Hepatic abscess can occurs as the result of direct extension of an infective process. Common examples of this include subdiaphragmatic abscess, suppurative cholecystitis, perinephric abscess, and perforation of the hollow viscera directly into the liver.

5. Trauma

Penetrating injury and blunt trauma can result in anan area of necrotic liver or intrahepatic hematoma, which can subsequently develop into an abscess. Bacteria may have been introduced from the trauma, or the affected area may be seeded from systemic bacteremia. Hepatic abscesses associated with trauma have a delayed presentation, up to weeks after the injury. Iatrogenic hepatic necrosis causes include hepatic artery embolization or thermal ablative procedures, can be complicated by abscess.

6. Cryptogenic

Cryptogenic abscesses are the most common cause in recent case series. Possible theories for cryptogenic hepatic abscess are resolved infective process at the time of presentation ,undiagnosed abdominal pathology, and host factors such as malignancy or diabetes rendering the liver more susceptible to transient hepatic artery or portal vein bacteremias.

In patients with cryptogenic hepatic abscess who have had computed tomography (CT) and ultrasonography, it has been argued that a diligent search for a cause should ensue.

Predisposing Factors

Pyogenic liver abscesses occur more commonly in adults with comorbid conditions including diabetes , cirrhosis, pancreatitis, inflammatory bowel disease, pyelonephritis, and peptic ulcer disease. Solid organ cancers as well as lymphoma and leukemia are present in 17–36% of patients with liver abscesses. The combination of chemotherapy and steroid use is thought to be responsible in these cases.

In children, pyogenic liver abscesses tend to occur in patients with host-defense abnormalities or immune disorders. Complement deficiencies, chronic granulomatous disease, and leukemia and other malignancies place these children at increased risk for liver abscess. Hepatic abscesses are also seen in sickle cell anemia, congenital hepatic fibrosis, polycystic liver disease, and after liver transplantation

Pathology and Microbiology

Most commonest site of hepatic abscess is the right lobe of the liver75%.It is due to preferential laminar flow of blood to the right side. The left lobe is involved in 20% of the time, and the caudate lobe is uncommonly involved (5%). About 50% of hepatic abscesses are solitary. The size of hepatic abscesses can vary from <1 mm to several centimeters in diameter and

can be multiloculated or a single cavity.Surrounding inflammation can cause adhesion to local structures.

In early series, sterile abscesses were commonly reported but probably reflected inadequate culture techniques, whereas in modern series, few abscesses are sampled before the administration of antibiotics. Abscesses from pyelophlebitis or cholangitis tend to be polymicrobial, with a high preponderance of gram-negative rods. Systemic infections are usually the cause infection with a single organism.

Most of the hepatic abscesses are polymicrobial in nature (40%). Solitary abscesses are more likely to be polymicrobial. Anaerobes are involved about 40% to 60% of the time. The most common micro organisms cultured are E.coli and Kleb.pneumoniae. Other common organisms encountered are Staph.aureus, Enterococcus species, Strep.viridans, and Bacteroides species. Klebsiella is frequently associated with gas-forming abscesses. Uncommonly encountered organisms (<10% of cultures) include Pseudomonas, Enterobacter, Proteus, Citrobacter, Serratia, β-hemolytic streptococci, microaerophilic streptococci, Fusobacterium, Clostridium, and other rare anaerobes. Blood cultures are positive in about 50% to 60% of cases. Fungal and mycobacterial hepatic abscesses are rare and are almost always associated with immunosuppression, usually from chemotherapy.

Clinical Features

Fever, jaundice, Weight loss, Pain, Nausea and vomiting, Malaise, chills, anorexia, Cough and pleurisy, pruritus, diarrohea.

Physical Examination –

Right upper quadrant tenderness, hepatomegaly, Jaundice, Right upper quadrant mass, Ascites, Pleural effusion or Rub.

Laboratory investigation

Increased Alkaline phosphatase, TC count>10000/mm3, Albumin <3g/dl, Hematocrit <36%, Bilirubin >2mg/dl.

Imaging studies

The most essential element to making the diagnosis of hepatic abscess is radiographic imaging.

Chest x-ray

Chest x-rays are abnormal in about 50% of the time, and findings generally reflect subdiaphragmatic pathology such as right pleural effusion, an elevated right hemidiaphragm, or atelectasis. Occasionally, these can be left-sided findings in the case of an abscess involving the left liver.

Xray abdomen erect

Plain abdominal x-rays, in rare cases, can be helpful. They can show portal venous gas or air-fluid levels.

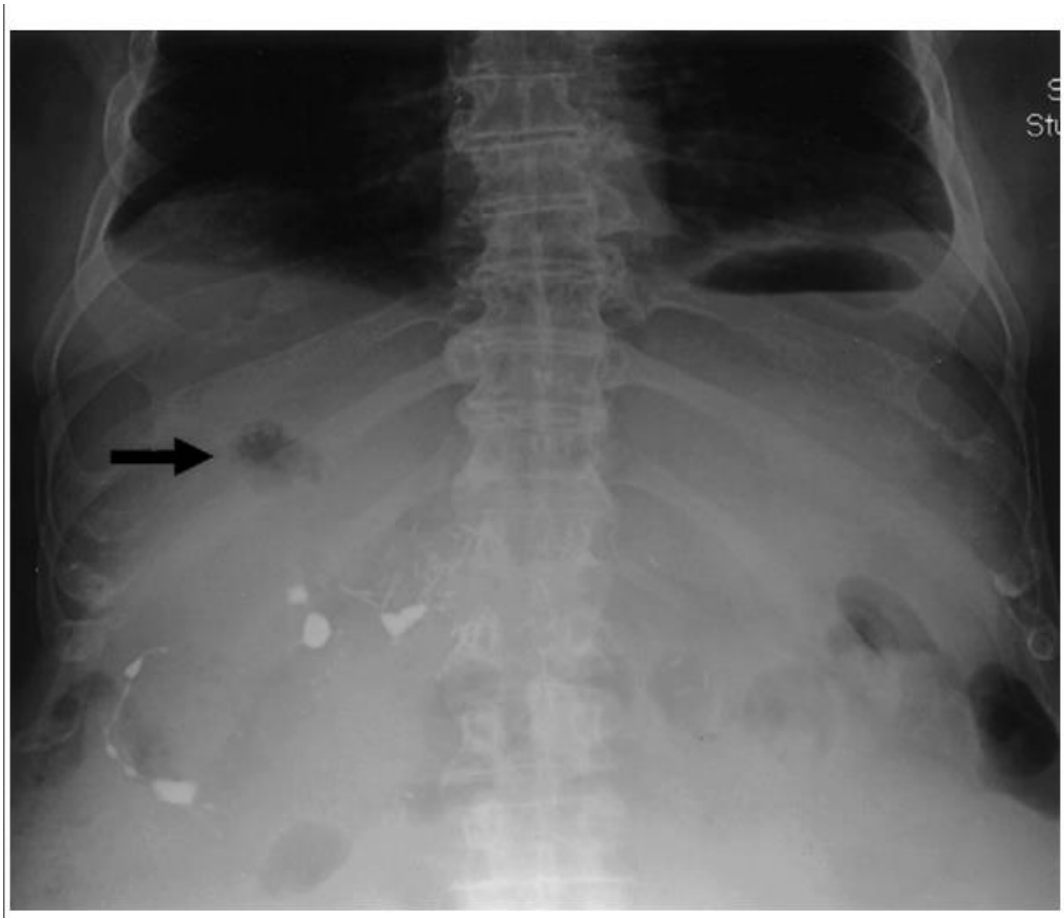


Figure. 13

Plain abdominal x-ray demonstrating an abnormal collection of air in the rt upper quadrant consistent with a pyogenic hepatic abscess (black arrow). Ultrasound and CT are the mainstays in diagnostic modalities for hepatic abscess.

Ultrasonogram abdomen

Ultrasound usually demonstrates

1) A oval or round area that is less echogenic than the liver and
2) Distinguish solid from cystic lesions.

The limitations of ultrasound are in its relative inability to visualize lesions high up in the dome of the liver and the fact that it is a user-dependent modality. The sensitivity of ultrasound in diagnosing liver abscess is 80% to 95%.

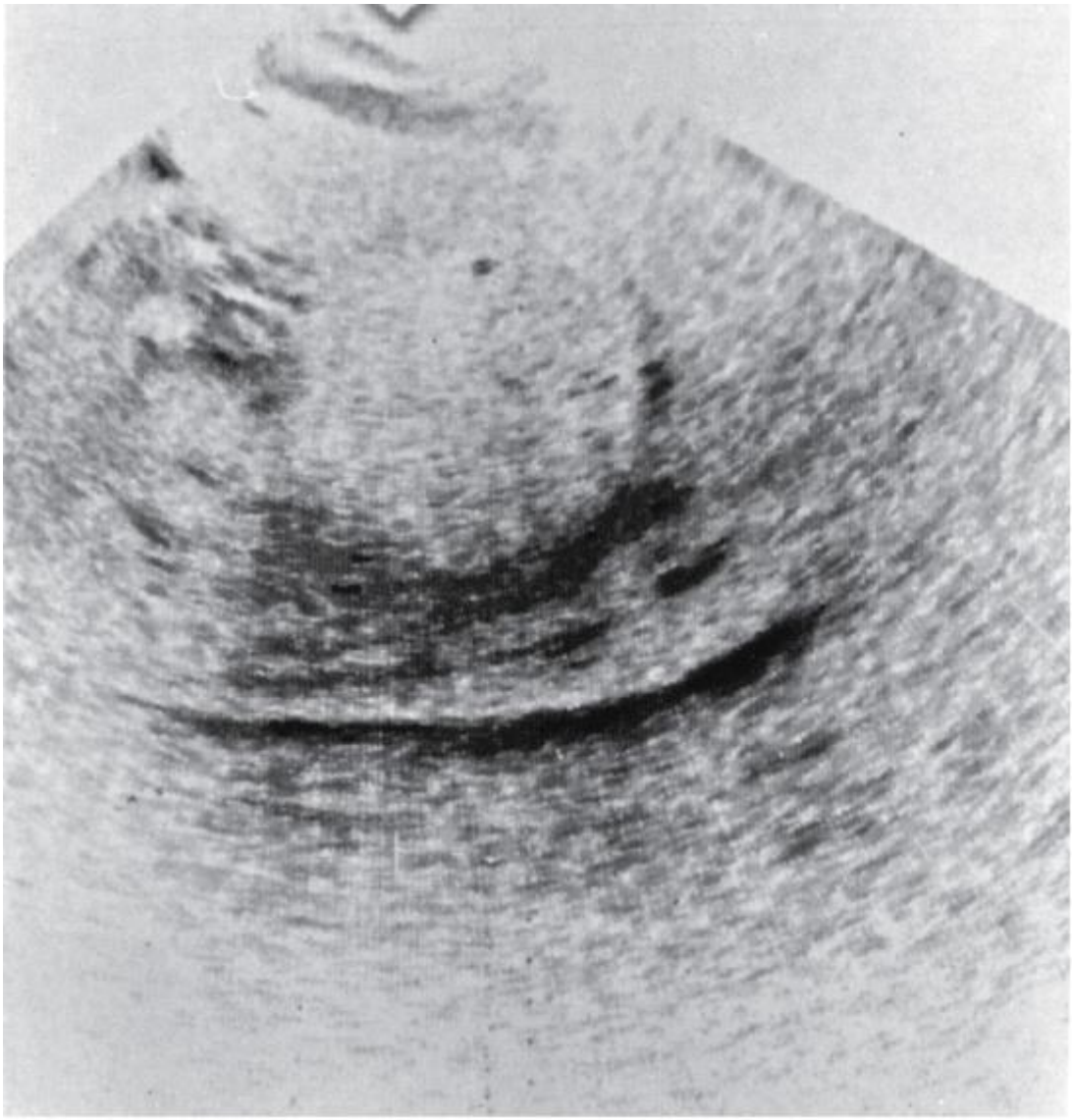


Figure.14

CT abdomen Plain and Contrast

CT demonstrates findings similar to ultrasound, and lesions are of lower attenuation than surrounding hepatic parenchyma. High-quality CT can demonstrate very small abscesses 0.5cm and can more easily pick up multiple small abscesses. It cannot be limited by air or ribs. The abscess wall usually shows an intense enhancement on contrast-enhanced CT. The sensitivity of CT in diagnosing liver abscess is 95% to 100%.

Both CT and ultrasound are useful in diagnosing other intra-abdominal pathology, such as biliary disease (ultrasound) or inflammatory disorders like appendicitis or diverticulitis (CT).

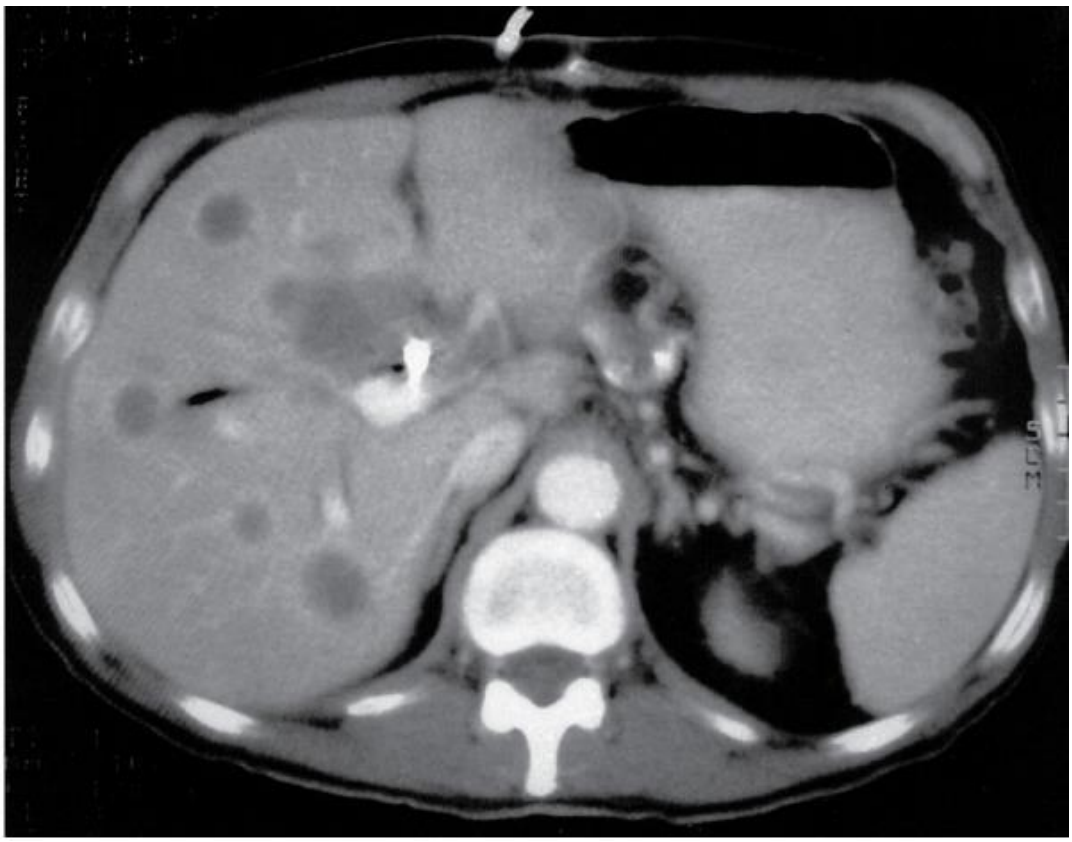


Figure.15

Differential Diagnosis

1) Amoebic abscess
2) Echinococcal cyst.

Features of Amoebic Versus Pyogenic Liver Abscess

Table.1

CLINICAL FEATURES	AMOEBIC ABSCESS	PYOGENIC ABSCESS
Age (yr)	20-40	>50
Male-to-female ratio	≥10:1	1.5:1
Solitary vs. Multiple	Solitary 80%[*]	Solitary 50%
Location	Usually right liver	Usually right liver
Travel in endemic area	Yes	No
Diabetes	Uncommon (~2%)	More common (~27%)
Alcohol use	Common	Common
Jaundice	Uncommon	Common
Elevated bilirubin	Uncommon	Common
Elevated alkaline phosphatise	Common	Common
Positive blood culture	No	Common
Positive amoebic serology	Yes	No

In acute amoebic abscess, 50% are solitary.

Treatment

Before the availability of antibiotics and the routine use of drainage procedures, untreated hepatic pyogenic abscess was almost uniformly fatal. It was not until the classic review by Ochsner and DeBakey in 1938 that routine surgical drainage was employed and dramatic reductions in mortality were noted. Open surgical drainage of pyogenic abscesses was the sole treatment (with the addition of antibiotics eventually) for hepatic abscess until the 1980s. Since the 1980s, less invasive percutaneous drainage techniques, along with the use of intravenous (IV) antibiotics, have been employed. Laparotomy is generally reserved for failures of percutaneous drainage.

When the diagnosis of pyogenic hepatic abscess is suspected, broad-spectrum IV antibiotics are started immediately to control ongoing bacteremia and its associated complications.

Blood cultures and cultures of the abscess from aspiration are sent for aerobic and anaerobic cultures. In immunosuppressed patients, mycobacterial and fungal cultures of the aspirate need to be considered.

Patients who are at risk for amoebic infections have amoebic serologies drawn. Until cultures have specifically identified the offending organism, broad-spectrum antibiotics covering gram-negatives, gram-positives, and anaerobes are used.

Medical management

Combinations such as ampicillin, an aminoglycoside, and metronidazole or a third-generation cephalosporin with metronidazole are appropriate. The optimal duration of antibiotic treatment is not well defined and must be individualized depending on the success of the drainage procedure. Antibiotics are certainly continued while there is evidence of ongoing infection, such as fever, chills, or leukocytosis. Beyond this, it is unclear how long to continue antibiotics, but recommendations usually are for 2 or more weeks.

Percutaneous Drainage Procedures

During the past 20 years, percutaneous catheter drainage has become the treatment of choice for most patients. Success rates range from 69% to 90%.

Advantages

1. The simplicity of treatment (usually employed at the time of radiologic diagnosis)
2. Avoidance of general anesthesia and
3. Avoidance of laparotomy.

Contraindications

1. Ascites,
2. Coagulopathy, or
3. Proximity to vital structures..

A recent retrospective study comparing surgical with percutaneous drainage for large (>5 cm) abscesses showed a better success rate with surgical drainage. Despite this, two thirds of the percutaneous treatments were successful, and the overall morbidity and mortality rates were similar. There has never been a randomized prospective comparison between percutaneous and surgical therapy for hepatic abscess, but case series suggest that for most cases, there are similar success rates and mortality rates.

Modern series attempting to compare these two techniques retrospectively must be read with caution because most patients treated surgically have failed other, less invasive techniques. In general, surgery is reserved for patients who require surgical treatment of the primary pathology (e.g., appendicitis) or for those who have failed percutaneous techniques.

Percutaneous aspiration

Percutaneous aspiration without the placement of a drain is another treatment modality. Success rates are generally 60% to 90% and are somewhat similar to those for percutaneous catheter drainage. Usually, however, more than one aspiration is required, and one fourth of patients require three or more aspirations. One randomized trial has evaluated percutaneous aspiration versus percutaneous catheter drainage. Success rates were 60% in the aspiration group and 100% in the catheter group, but all but one patient in the aspiration group had a single aspiration. Another recent randomized trial compared aspiration alone to catheter drainage. Sixty-four randomized patients were analyzed, and there were similar outcomes in terms of treatment success rate, hospital stay, antibiotic duration, and mortality. In the aspiration-only group,

40% required two aspirations, and 20% required three aspirations. In general, catheter drainage remains the treatment of choice, although a trial of a single aspiration is reasonable to consider.

Some investigators have reported success with antibiotics alone. Most of these patients, however, have had a diagnostic aspiration and thus at least a partial drainage. Additionally, other series have reported that antibiotic treatment without drainage carries a prohibitively high mortality rate (59%-100%). In patients who are not surgical candidates or who absolutely refuse any invasive procedure, an attempt at antibiotic treatment is reasonable; however, this is not recommended in all other situations.

Open drainage

Ruptured liver abscesses

Liver resection

Liver resection is occasionally required for hepatic abscess. This may be required for an infected hepatic malignancy, hepatolithiasis, or intrahepatic biliary stricture. If hepatic destruction from infection is severe, some patients may benefit from resection.

Outcome

Mortality from pyogenic hepatic abscess has dramatically improved during the past 6 decades. Before the routine use of surgical drainage, pyogenic abscess was uniformly fatal. With the routine use of surgical drainage and the use of IV antibiotics, mortality was reduced to about 50%, a

figure that stayed relatively constant from 1945 until the early 1980s. Since the 1980s, mortality has been reported from 10% to 20%, and series from the 1990s now routinely demonstrate a mortality rate of less than 10%.

Factors predictive of a poor outcome

- The presence of malignancy,
- Factors associated with malignancy (jaundice, markedly elevated LFTs), or
- Signs of sepsis
- Hypoalbuminemia
- Signs of severe infection, such as marked leukocytosis
- Acute Physiology and Chronic Health Evaluation (APACHE) II scores,
- Abscess rupture
- Bacteremia, and
- Shock.

Amoebic Liver Abscess

Epidemiology

Amoebic liver abscesses tend to be more common in Hispanic males, aged 20 to 40 years, with a history of travel to (or origination from) an endemic area.

Poverty and cramped living conditions are associated with higher rates of infection. A male preponderance of greater than 10:1 is seen. Heavy alcohol

consumption is commonly reported and may render the liver more susceptible to amoebic infection.

Patients with impaired host immunity also appear to be at higher risk for infection and have higher mortality rates. Patients with amoebic liver abscess without a travel history to an endemic area often have an associated immunosuppression, such as human immunodeficiency virus (HIV) infection, malnutrition, chronic infection, or chronic steroid use.

Pathogenesis

Entamoeba. histolytica is a protozoa that exists as a trophozoite or as a cyst.

All other species in the genus Entamoeba are found to be nonpathogenic, and not all strains of *E.* histolytica are considered virulent. Ingestion of cysts of Entamoeba histolytica occurs i.e through a fecal-oral route. Humans are the principal host, and the main source of infection is human contact with a cyst-passing carrier.

Contaminated water and vegetables are also a route of human infection. Once ingested, the cysts are not degraded in the stomach and pass to the intestines where the trophozoite is released and passed on to the colon. In the colon, the trophozoite can invade mucosa, resulting in disease.

Route of spread

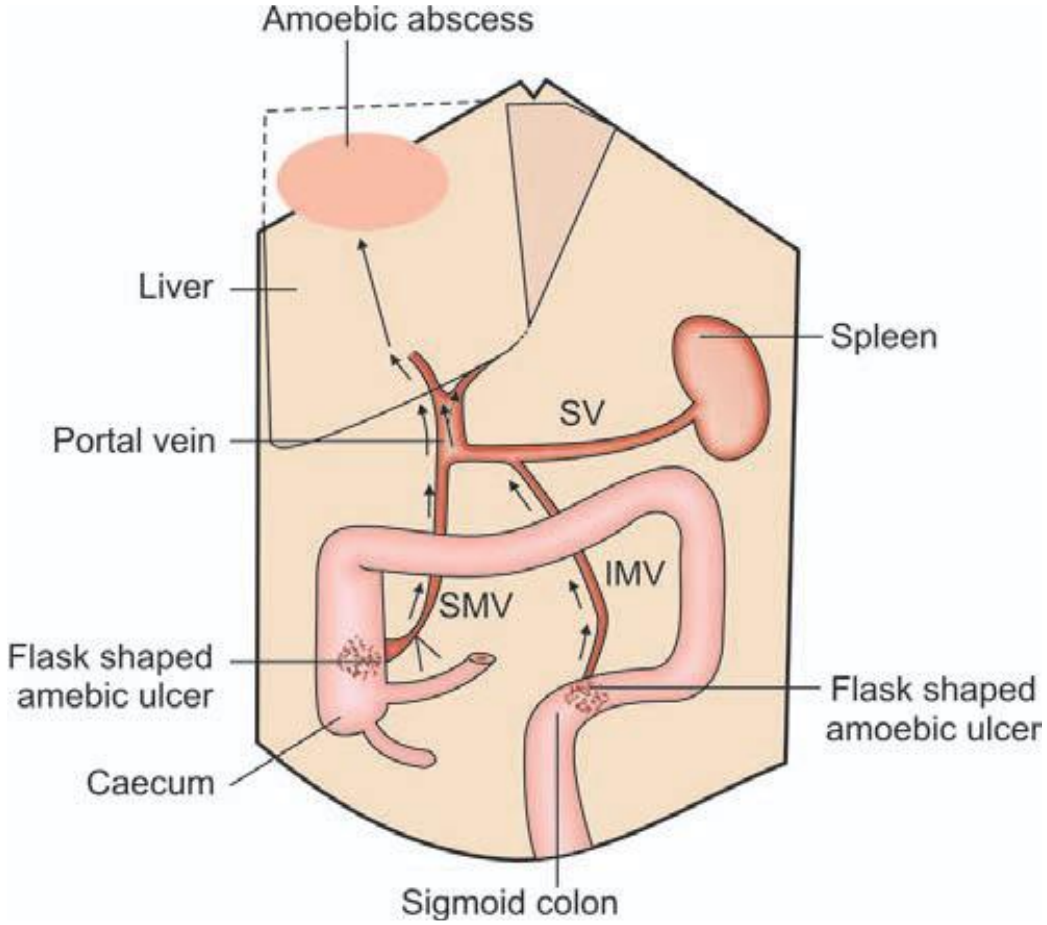


Figure.16

It is believed that the trophozoites reach the liver through the portal venous system. There is no evidence for trophozoites passing through lymphatics. As implied by its name, E. Histolytica trophozoites have the capacity to lyse tissues through a complex set of events, including cell adherence, cell activation, and subsequent release of multiple enzymes resulting in necrosis. The major mechanism is probably enzymatic cellular hydrolysis. Amoebic liver abscesses are thus formed by progressing, localized hepatic necrosis resulting in a cavity containing acellular proteinaceous debris

surrounded by a rim of invasive amoebic trophozoites. Early development of an amoebic liver abscess is associated with an accumulation of polymorphonuclear leukocytes, which are then lysed by the trophozoites.

Antiamoebic antibodies develop rapidly in patients with invasive disease or amoebic hepatic abscess. Secretory immunoglobulin A (IgA) antibodies have been shown in vitro to inhibit adherence to colonic epithelium; however, the development of these antibodies does not halt the progression of disease. Interestingly, children who lack antiamoebic IgG have innate resistance to invasive infection, suggesting an alternative immune-mediated re-sponse. There is now evidence that a cell-mediated T-helper response is probably the major mechanism of resistance.

Pathology

Hepatic amoebic abscess is essentially the result of liquefaction necrosis of the liver, producing a cavity full of blood and liquefied liver tissue.

The appearance of this fluid is typically described as **anchovy sauce,** and the fluid is odorless unless secondary bacterial infection has taken place.

The progressive hepatic necrosis continues until Glisson's capsule is reached because the capsule is resistant to hydrolysis by the amoebae and thus amoebic abscesses tend to abut the liver capsule. Because of the resistance of Glisson's capsule, the cavity is typically crisscrossed by portal triads protected by this peritoneal sheath. Early on, the formed cavity is ill defined, with no real fibrous response around the edges, but a chronic abscess can ultimately

develop a fibrous capsule and may even calcify. Like pyogenic abscesses, amoebic abscesses tend to occur mainly in the right liver.

ANCHOVY SAUCE PUS

Figure.17

Most common location

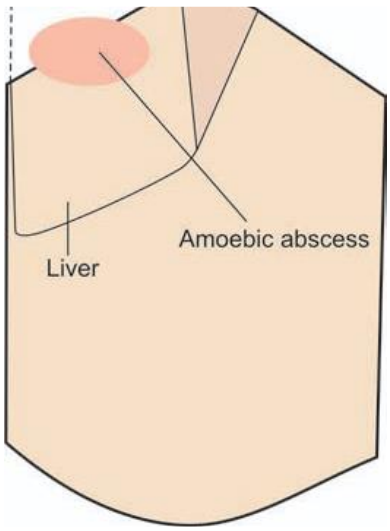


Figure.18

Most common site of amoebic liver abscess was right posterosuperior segment.

Clinical Features

- It is common in males (20:1), may be after an attack of amoebic dysentery or many months after the attack or history of dysentery may not be there at all.
- They present with fever, loss of weight, chills and rigors, non productive cough, shoulder pain.
- Pain in the right hypochondrium.
- Soft, tender, smooth, liver with increased liver span.
- **Intercostal tenderness** is elicited which is a useful clinical sign.
- Right sided pleural effusion may be evident.
- Mild jaundice is not uncommon especially in cirrhotics and multiple abscesses which may signify poor prognosis.
- Tenderness, rigidity and skin oedema in right hypochondrium may be present in acute cases.
- In chronic amoebic liver abscess, smooth, firm/hard, nontender liver may be palpable.

Amoebic liver abscess may be –

- **Acute** – present with high fever, chills, rigors, tender, soft palpable liver, with intercostal tenderness.
- **Chronic** – present with firm/hard, smooth, non-tender palpable liver without acute features.

Features may be of –

- **Systemic** – present with fever, chills and rigors, loss of appetite, reduced weight, and jaundice.
- **Abdominal** – present with pain and tenderness, localized guarding and rigidity, mass in right upper abdomen (tender, soft liver), ascites, splenomegaly, abdominal wall oedema.
- **Thoracic** – present with dry cough, chest pain in right lower part, right shoulder pain, pleural effusion, and intercostal tenderness.
- **Features of complications –** rupture/infection/ septicaemia/liver failure.

Differential diagnosis

For Acute type

1. Acute cholecystitis
2. Acute presentation of hepato-cellular carcinoma
3. (HCC) due to haemorrhage or necrosis.
4. Subphrenic abscess.

Chronic amoebic liver abscess mimics hepatoma in every respect

Investigations

- Total count may be increased. Patients typically have a mild to moderate leukocytosis without eosinophilia Anemia is common
- Liver function tests may show altered bilirubin and albumin level.

- Prothrombin time may be widened and if it is so Inj. vit K 10 mg IM for 5 days should be given. Even with this if P.T. remains widened then fresh frozen plasma (FFP) is needed to rectify the P.T.
- Serum alkaline phosphatase, ALT, AST levels are altered.
- Indirect haemagglutination test (95% positive rate),

ELISA and gel diffusion precipitative test are reliable serological tests. Serological tests are reliable in nonendemic areas than endemic areas. Counter immunoelectrophoresis is more useful in active disease. This test has largely been replaced by enzyme immunoassays (EIAs), which are simple, rapidly performed, and inexpensive. The EIA has 99% sensitivity and 90% specificity in patients with hepatic abscess. Unfortunately, the presence of antibodies may reflect old infection, and interpretation can be difficult in endemic areas. Ongoing studies are focusing on identifying specific E. histolytic antigens in an attempt to identify acute infection.

- Chest X-ray findings:

- Raised fixed diaphragm (tenting)

- Pleural effusion

- Soft tissue shadow

- Atelectasis

- U/S abdomen shows

-altered echogenicity, (anechogenic, hypoechogenic)

-size,

-location,

- number of abscess

-nature of the liver.

Abdominal ultrasound has a reported accuracy of about 90% when combined with a typical history and clinical presentation.

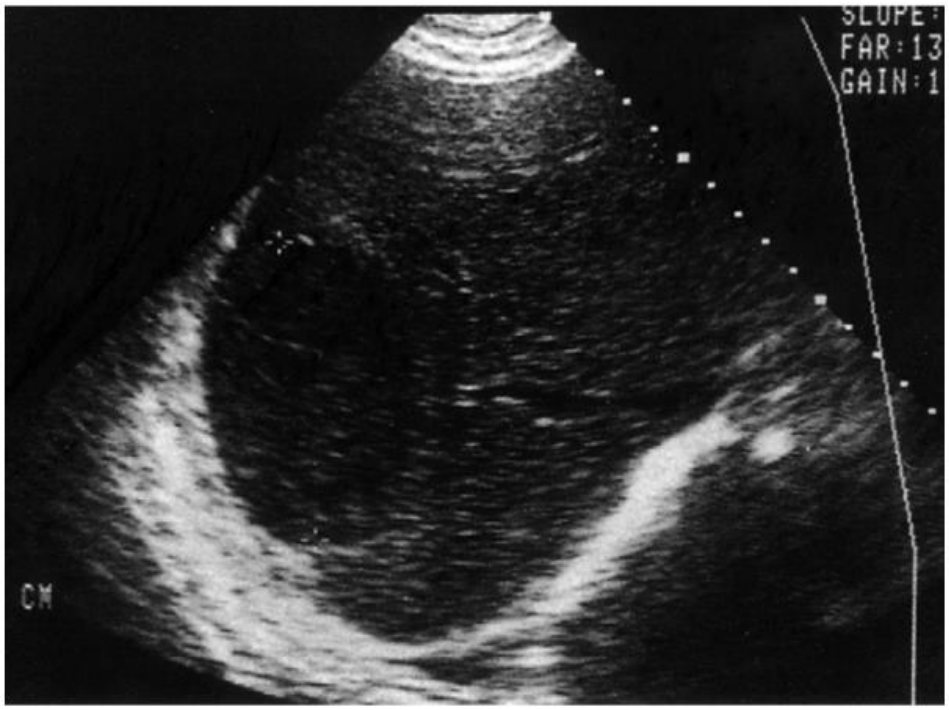


Figure.19

Typical ultrasound of an amoebic hepatic abscess. Note the peripheral location, rounded shape with poor rim, and internal echoes.

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

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

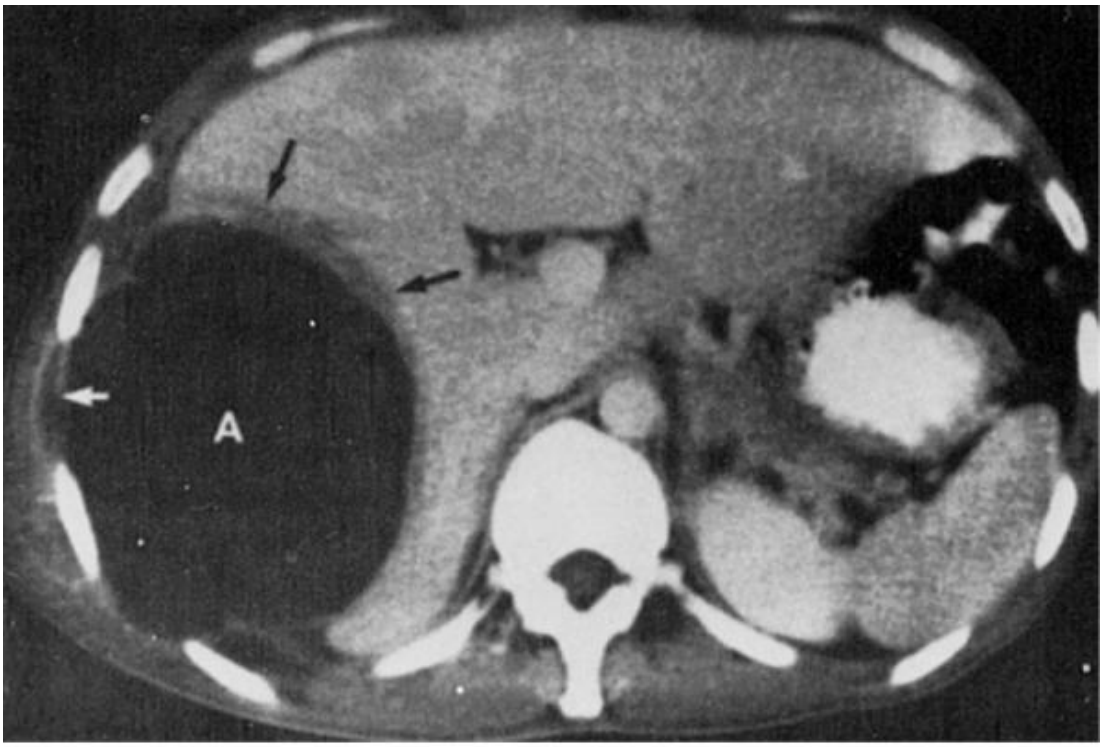


Figure.20

CT scan of amoebic abscess. The lesion is peripherally located and round. Rim is nonenhancing but shows peripheral edema (black arrows). Note the extension into the intercostal space (white arrow).

- Sigmoidoscopy/colonoscopy

-used to identify the active ulcers. Scrapings of the ulcer show trophozoites.

When the previously outlined workup is still not definitive and diagnostic uncertainty persists, two options are considered. A therapeutic trial of antiamoebic drugs in which rapid improvement occurs in most cases of amoebic abscess can be helpful. In situations in which amoebic serology is inconclusive and therapeutic trial of antibiotics is either deemed inappropriate or has failed to improve symptoms, consideration is given to diagnostic aspiration. A pyogenic abscess would have bacteria and leukocytes, whereas an amoebic abscess would contain the typical anchovy sauce appearance. Cultures of amoebic abscess are usually negative and do not contain leukocytes.

In cases in which neoplasm or hydatid disease is given serious consideration, aspiration should not be performed.

Management

The mainstay of treatment for amoebic abscesses is metronidazole (750 mg orally tds per day for 10 days), which is curative in more than 90% of patients. Clinical improvement is usually seen within 3 days. If response to metronidazole is poor or the drug is not tolerated, other agents can be used.

Emetine hydrochloride is effective against invasive amebiasis (particularly in the liver) but requires intramuscular injections and has serious cardiac side effects. A more attractive option is chloroquine, but this is a less effective agent. After treatment of the liver abscess, it is recommended that

luminal agents such as iodoquinol, paromomycin, and diloxanide furoate are administered to treat the carrier state.

Aspiration

- In case of large abscess and infected abscess aspiration with a wide bore needle is done under U/S guidance after correcting the P.T.
- Previously without U/S, aspiration used to be done by passing needle in right 6th intercostal space in midaxillary line.

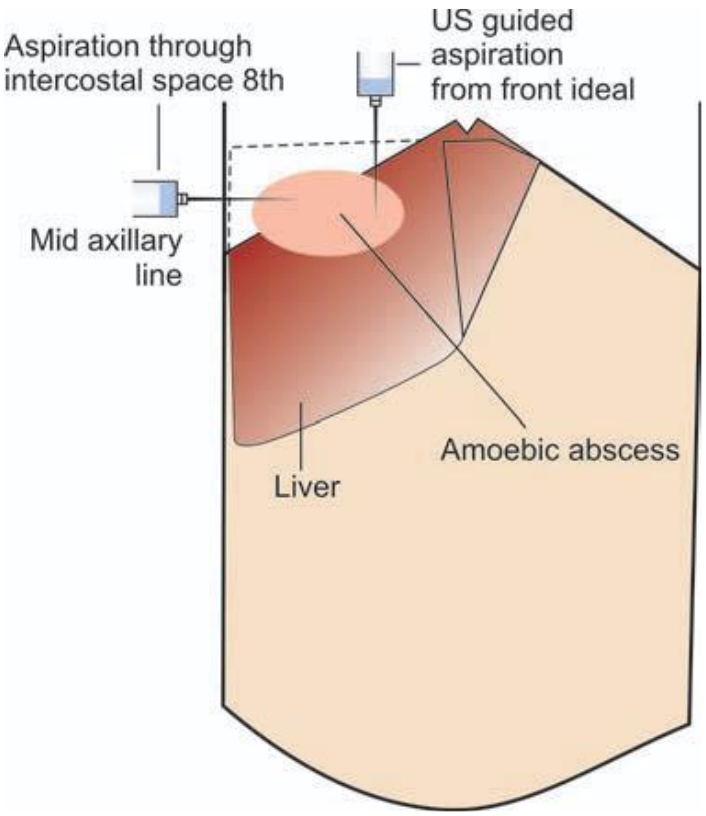


Figure.21

Aspiration of the amoebic liver abscess under US guidance or through right 8th intercostal space in midclavicular line.

Therapeutic needle aspiration of amoebic abscesses has been proposed. Small randomized trials comparing metronidazole with or without aspiration have shown minor benefits with aspiration, but no major improvement to justify routine aspiration. In general, aspiration is recommended for diagnostic uncertainty, failure to respond to metronidazole therapy in 3 to 5 days, or in abscesses felt to be at high risk for rupture. Abscesses greater than 5 cm in diameter and in the left liver are thought to be a higher risk for rupture, and aspiration needs to be considered.

Percutaneous Drainage

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

Note: Aspirated fluid is sent for C/S, cytology and for study of trophozoites (last part of the aspirated fluid should be sent for this).

Open Drainage

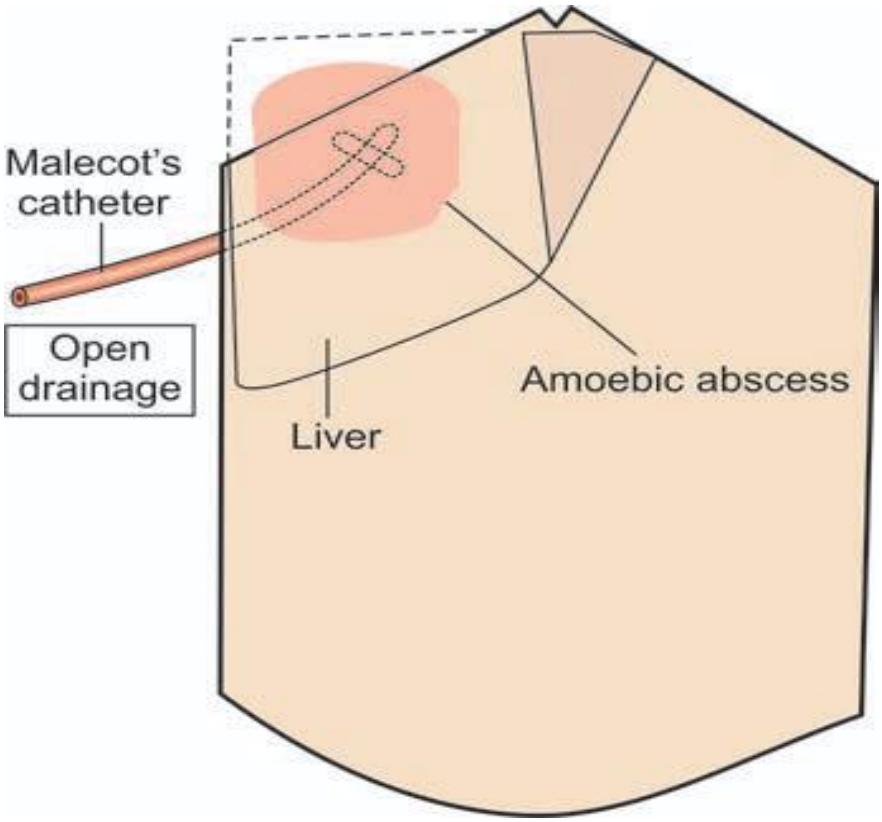


Figure.22

Through transperitoneal approach, abscess area is opened, pus is evacuated. Malecot's catheter is placed and brought out through a separate stab incision. The catheter is kept in situ until drainage stops completely. Complete drainage of pus will be confirmed by repeat U/S. During discharge, advice is given to avoid alcohol, Chloroquine 250 mg BD for 10 days and Diloxanide furaote 500 mg tid is given for 10-14 days.

Complications of surgery – anesthetic problems; bleeding; liver failure (in cirrhotic patients); intraperitoneal abscess formation; bile leak – bile peritonitis and fistula.

Surgery

Indications for surgery

1) Even after repeated aspirations if abscess cavity
2) fills again
3) Thick pus
4) Multiloculated abscess
5) Left lobe abscess, because of danger of rupture into
6) pericardial cavity
7) Ruptured abscess
8) Caudate lobe abscess
9) Multiple abscesses

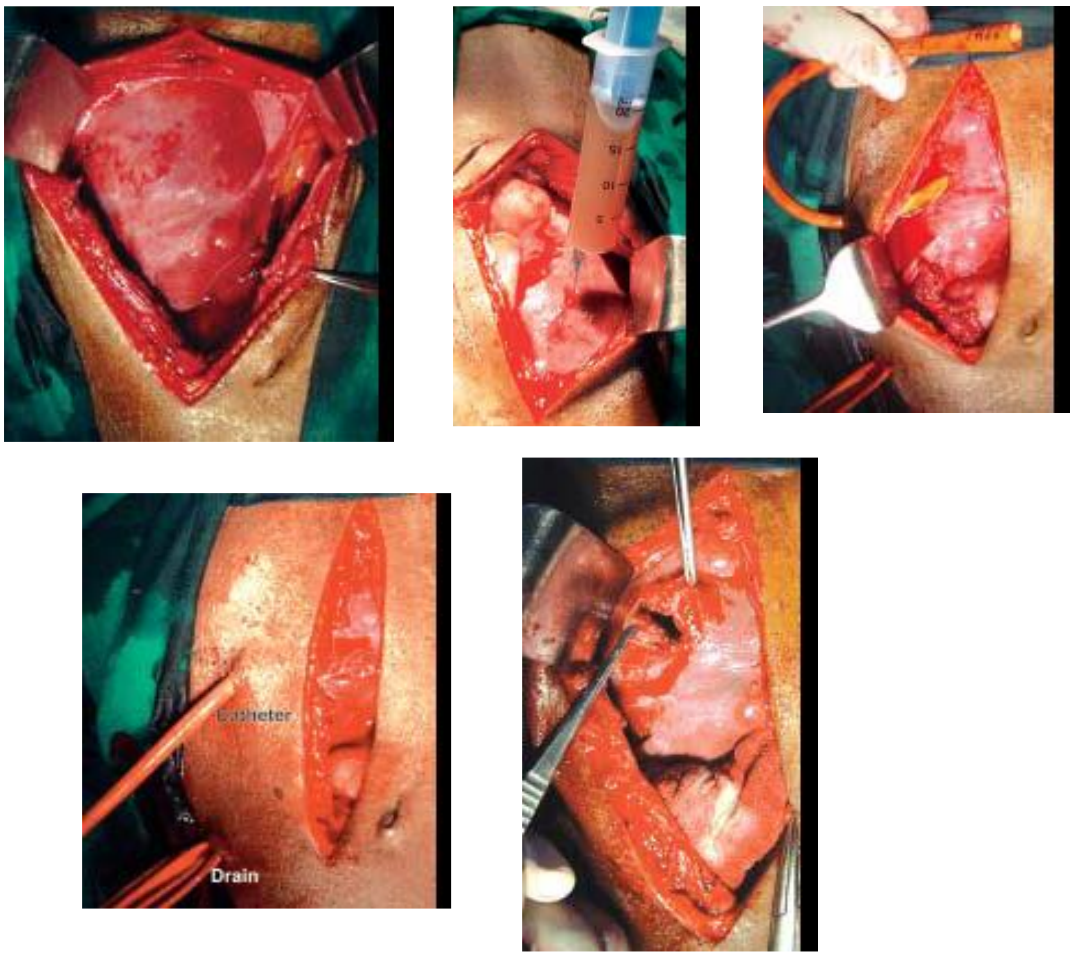


Figure. 23

Operative findings of amoebic liver abscess. Note the exposed abscess in the liver, its aspiration and drainage. Placement of Malecot's catheter in the abscess cavity and drain placed into the peritoneal cavity is also seen.

Outcome

Although amoebic liver abscess usually responds rapidly to treatment, there are uncommon complications that the practitioner must be aware of. The most frequent complication of amoebic abscess is intra peritoneal rupture, rupture into pleural cavity, or pericardium. Size of the abscess appears to be the most important risk factor for rupture, and the overall incidence of rupture

ranges from 3% to 17%. Most peritoneal ruptures tend to be contained by the diaphragm, abdominal wall or omentum, but rupture can fistulize into a hollow viscus. A peritoneal rupture usually presents as abdominal pain, peritonitis, and either a mass or generalized distention. Laparotomy was advocated in the past for this complication, but now most cases are managed successfully with percutaneous drainage. Laparotomy is indicated in cases of doubtful diagnosis, failure of conservative therapy, hollow viscus perforation, fistulization resulting in hemorrhage or sepsis. Rupture into the pleural space usually results in a large and rapidly accumulated effusion that collapses the involved lung. Treatment consists of thoracentesis, but if secondary bacterial infection ensues, more aggressive surgical approaches may be necessary. Rupture can occur into the bronchi and is usually self-limited with postural drainage, bronchodilators. Rarely, a left-sided abscess may rupture into the pericardium and can present as an asymptomatic pericardial effusion or even cardiac tamponade. This must be treated with aspiration. Other complications include compression of the biliary tree or inferior vena cava from a very large abscess and development of a brain abscess.

The mortality rate for all patients with amoebic liver abscess is about 5% and does not appear to be affected by the addition of aspiration to metronidazole therapy or chronicity of symptoms. When an abscess ruptures, the mortality rate is reported to be from 6% to as high as 50%. Factors independently associated with poor outcome are elevated serum bilirubin (>3.5 mg/dL), encephalopathy, hypoalbuminemia (<2.0 g/dL), multiple abscess cavities, abscess volume greater than 500 mL, anemia, and diabetes.

Although clinical improvement after adequate treatment with antiamoebic agents is the rule, radiologic resolution of the abscess cavity is usually delayed. The average time to radiologic resolution is 3 to 9 months and can take as long as years in some patients. Studies have shown that more than 90% of the visible lesions disappear radiologically, but a small percentage of patients are left with a clinically irrelevant residual lesion.

MATERIAL AND METHODS

SOURCE OF DATA

All cases of Liver abscesses presenting to the Surgery OPD or Casualty of CHMC Hospital, referred from medical wards of CHMC hospital or referred from outside diagnosed as case of liver abscess, in Chengalpattu over a period of 1 and half years with Clinical/ Sonological/CT features of Liver abscess.

METHOD OF COLLECTION OF DATA

Patient data collection and evaluation.

- Data will be collected from all patients attending CHMC Hospital, Chengalpattu in General Surgery OPD, Casualty and Inpatient department, irrespective of their gender/ background /socio economic status. Age above 18years included in the study. The patients will be evaluated and followed up according to protocol.
- Detailed history of patient will be entered in proforma.
- Complete haemogram, LFT, Prothrombin time, Serology for amoebic antigen will be sent immediately on presentation.
- Ultrasound of Abdomen and Pelvis will be done routinely on the same day of presentation.

- Patient will be treated by medical/image guided drainage/surgical(open drainage) based on its site, size and other clinical parameters.
- Patient will be followed up daily clinically and LFT.
- USG Abdomen will be repeated on the 3rd day if patient symptomatically not relieved.
- If the patient develops any of the complications like ruptured liver abscess into any of the serosal cavity, patient will be immediately take up for surgery.
- Patient will be informed about any surgical procedure and proper informed consent will be taken.

Patient data collected regarding:

Age :

Sex :

Occupation :

Chief presenting complaints :

Past-surgical history :

Past history of similar illness :

History of alcoholism :

History of diabetes :

History of immunodefiency states :

History of biliary tract disorder :

History of amoebic dysentery :

History of jaundice :

General examination of systems :

Respiratory system :

Per abdomen :

Inspection :

Palpation :

Percussion :

Per rectal :

If the patient is referred from elsewhere the details of the same will be considered at the time of admission.

Blood investigations

Total count :

Bilirubin :

PT/INR :

Albumin :

Serology for amoebic antigen :

Imaging studies

CXR

X ray abdomen erect

USG abdomen

CT abdomen

Complications if developed will be assessed in detail.

Management strategies for liver abscesses followed

Medical management

Image guided drainage

Open drainage

Complications –followed up.

Follow-up of patients:

Patients will be followed up over a period of 6 months

1) Once in 2 weeks for first 2 months
2) Then on a monthly basis after discharge , To look for recurrent attacks or To look for the development of complications and To monitor the efficacy of the treatment given.

Inclusion Criteria:

1. All cases of liver abscess diagnosed clinically and/or ultrasonographically.
2. All cases of bacterial and parasitic liver abscess
3. All cases in evolving, liquefied & ruptured stage with or without peritonitis
4. All cases of Diagnosed Liver Abscess being referred to CHMC.

Exclusion Criteria:

- Traumatic Liver Abscess
- Past history of liver abscess

Sample size: 60cases.

Study design: Prospective study

Duration of study: 1 1/2 years.

This study was done from MARCH 2014 to AUGUST 2015 on 60 patients. Case selection for the study will be done in the initial 1 yr followed by follow up totally for 6 months.

Study place: CHMC, Chengalpattu

Does the study required any investigations or interventions to be conducted on patients? If so, please describe briefly.

Routine blood investigations, X rays Chest and Abdomen, USG Abdomen & LFT and Prothrombin time, Serology for amoebic antigen [CT Abdomen if required]. Surgical intervention if the complication necessitates the same.

Has ethical clearance been obtained from your institution?

Yes

OBSERVATION AND RESULTS

Totally 60 patients who met the inclusion criteria were studied.

AGE DISTRIBUTION

Table. 2

	AGE
N	60
Mean	45.95
Median	44
Std. Deviation	12.423
Minimum	24
Maximum	73

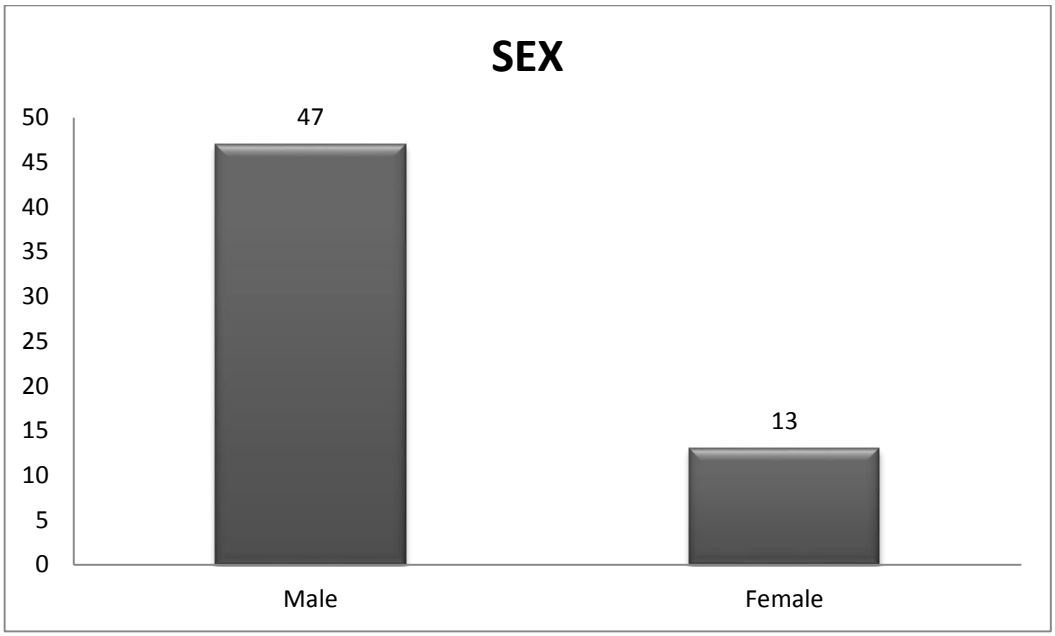
The mean age of distribution is 45.95.

SEX DISTRIBUTION

Table. 3

SEX	**Frequency**	**Percent**
Male	47	78.3
Female	13	21.7
Total	60	100

Chart.1



In this study, out of 60 patients, liver abscess are present in 47 males (78.3%) and 13 are females (21.7%).

CLINICAL FEATURES

Table.4

ABDOMINAL PAIN	Frequency	Percent
Present	44	73.3
Absent	16	26.7
Total	60	100

Table.5

FEVER	Frequency	Percent
Present	31	51.7
Absent	29	48.3
Total	**60**	**100**

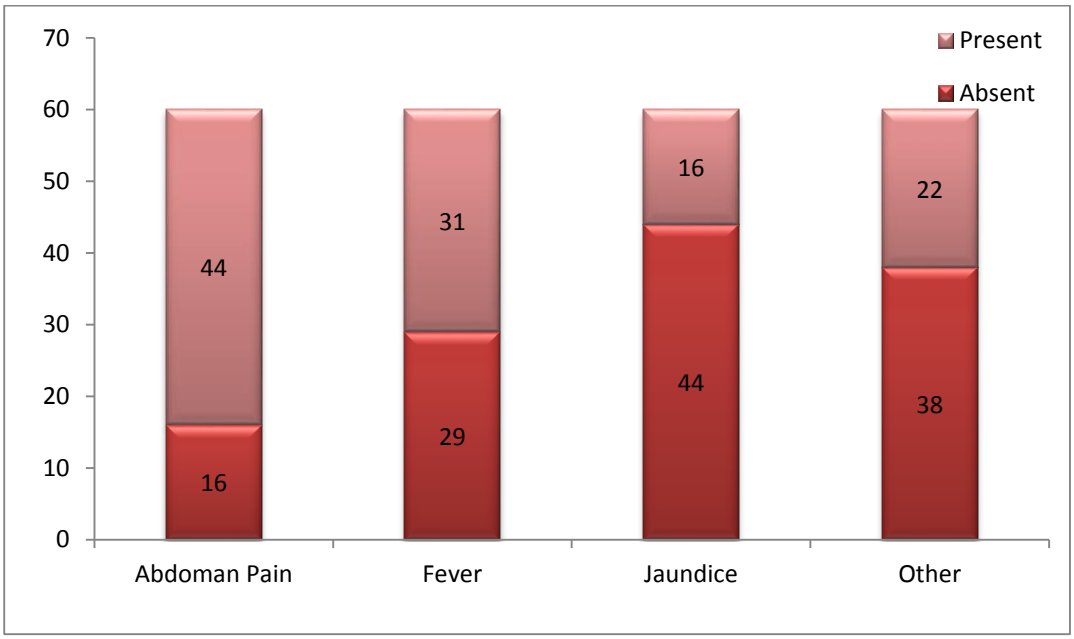
Table. 6

JAUNDICE	Frequency	Percent
Present	16	26.7
Absent	44	73.3
Total	**60**	**100**

Table. 7

Other	Frequency	Percent
Present	22	36.7
Absent	38	63.3
Total	**60**	**100**

Chart.2



Overall, most common presentation of liver abscess in our institution was abdominal pain (44%), then fever (31%).

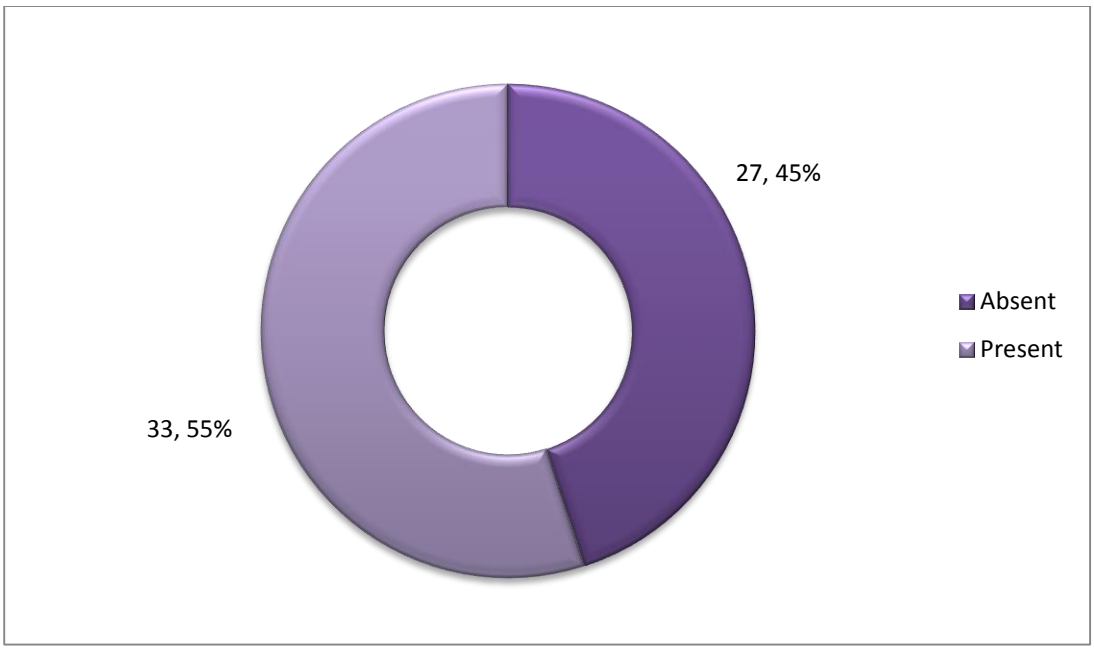
CLINICAL SIGNS

Table.8

INTERCOSTAL TENDERNESS	**Frequency**
Present	33
Absent	27
Total	**60**

Out of 60 patients, 33 patients had intercostal tenderness (55%).

Chart. 3



COMPARISION OF CLINICAL FEATURES OF PYOGENIC AND AMOEBIC LIVER ABSCESS

Table.9

ABD.PAIN	AMOEBIC	PYOGENIC	Total	Chi sq	P
Absent	6	10	16	16.36	**0.0001**
Present	39	5	44		
Total	**45**	**15**	**60**		

Table.10

FEVER	AMOEBIC	PYOGENIC	Total	Chi sq	P
Absent	28	1	29	13.9	**0.001**
Present	17	14	31		
Total	**45**	**15**	**60**		

Table.11

JAUNDICE	AMOEBIC	PYOGENIC	Total	Chi sq	P
Absent	39	5	44	16.36	**0.0001**
Present	6	10	16		
Total	**45**	**15**	**60**		

Table.12

Other	AMOEBIC	PYOGENIC	Total	Chi sq	P
Absent	29	9	38	0.1	0.7
Present	16	6	22		
Total	**45**	**15**	**60**		

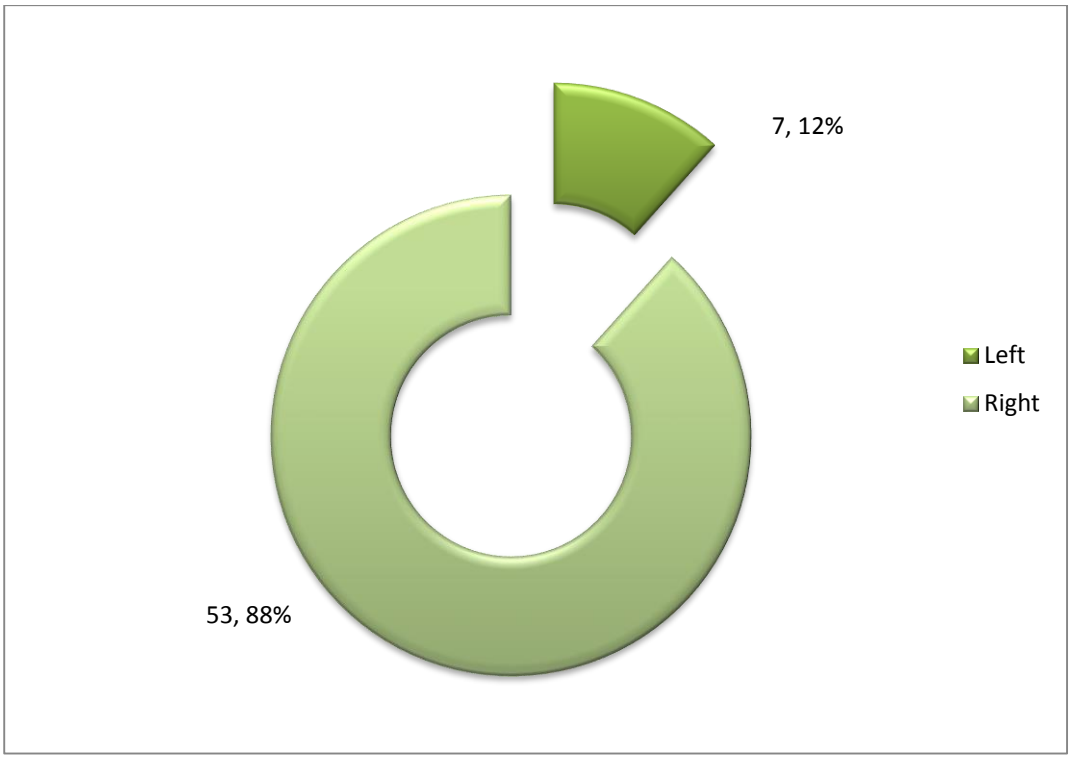
In this study, in case of pyogenic liver abscess most common presentation was fever, whereas in case of amoebic liver abscess most common presentation was abdominal pain.

LOCATION OF ABSCESS

Table.13

SITE	Frequency
Left	7
Right	53
Total	**60**

Chart.4



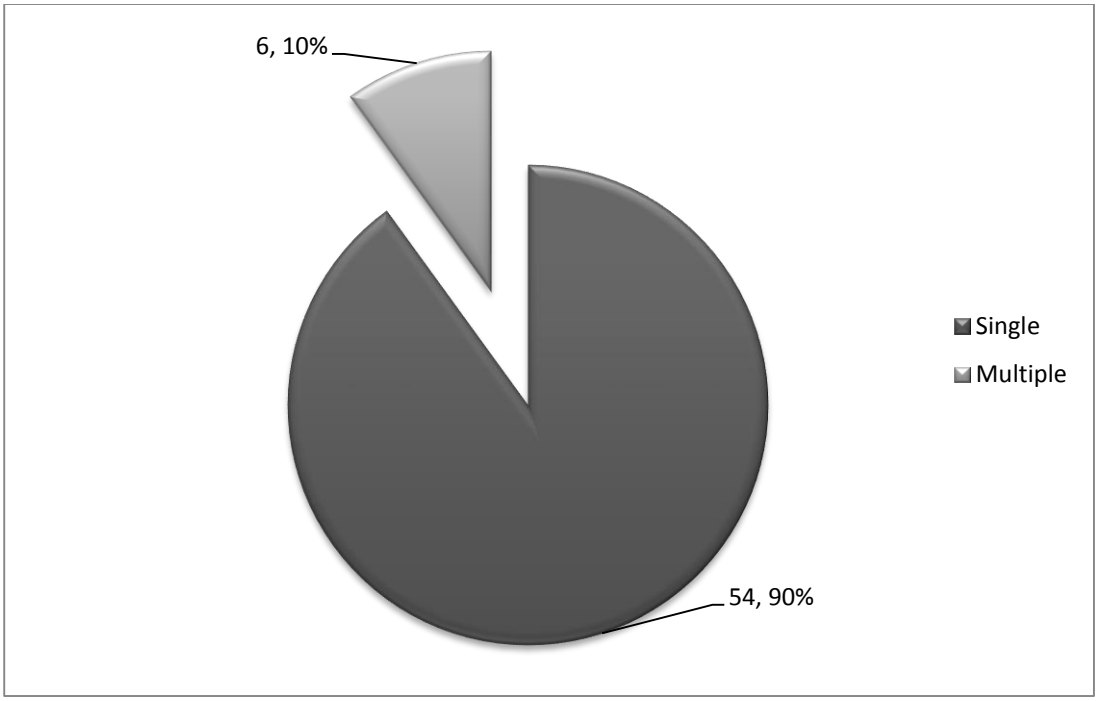
Out of 60 cases, in this study abscess located in right lobe was 53 cases (88.3%)

SOLITARY OR MULTIPLE

Table.14

	Frequency
Single	54
Multiple	6
Total	**60**

Chart. 5



In this study, most of the abscesses were solitary (90%).

Table.15

Medical	Correlation Coefficient	-0.019	0.099	0.144	-0.072	-0.041	-0.032	0.008	-0.177	0.096	-0.035	**-.314***	0.012	-0.013
	Sig. (2-tailed)	0.883	0.451	0.274	0.584	0.754	0.808	0.951	0.177	0.465	0.792	**0.015**	0.925	0.92
	N	60	60	60	60	60	60	60	60	60	60	60	60	60

Table.16

IMAGE GUIDED DRAINAGE	Correlation Coefficient	-0.065	0.109	-0.039	0.056	-0.048	-0.074	-0.207	0.039	-0.066	0.092	0.247	-0.065	0.056	-0.147
	Sig. (2-tailed)	0.621	0.405	0.768	0.671	0.716	0.574	0.113	0.765	0.618	0.484	0.057	0.55	0.67	0.262
	N	60	60	60	60	60	60	60	60	60	60	60	60	60	60

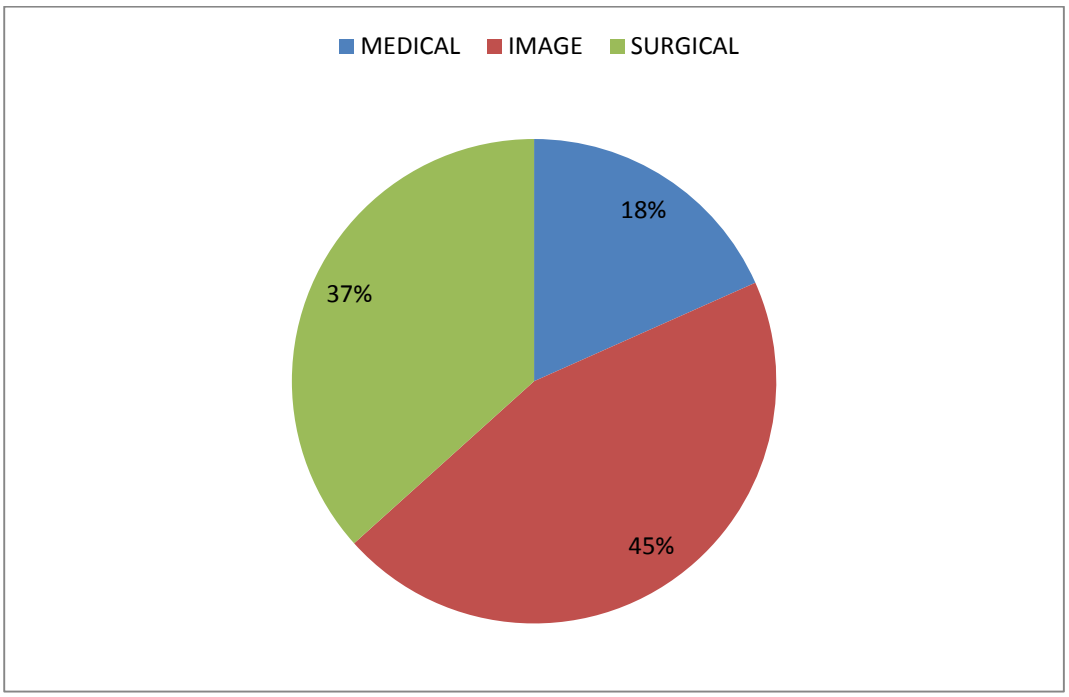
Table.17

OPEN DRAI NAGE	Correlation Coefficient	-0.029	**-.293***	0.118	**.293***	-0.024	0	**.264***	**.270***	0.131	-0.085	**.728****	0.037	0.118	**-.338****	0.19
	Sig. (2-tailed)	0.828	**0.023**	0.37	**0.023**	0.853	1	**0.042**	**0.037**	0.317	0.517	**0.0001**	0.781	0.37	**0.008**	0.147
	N	60	60	60	60	60	60	60	60	60	60	60	60	60	60	60

Table.18

MEDICAL	IMAGE	SURGICAL
11	27	22

Chart. 6.



Out of 60 patients in our study, 11 patients (18%) responded to medical management alone. In this study 27 patients (45%) were treated by image guided aspiration or drainage. Those who required surgical drainage were 22 patient (37%), who are all not responding to the above modalities of treatment and those with complication.

MICROBIOLOGY

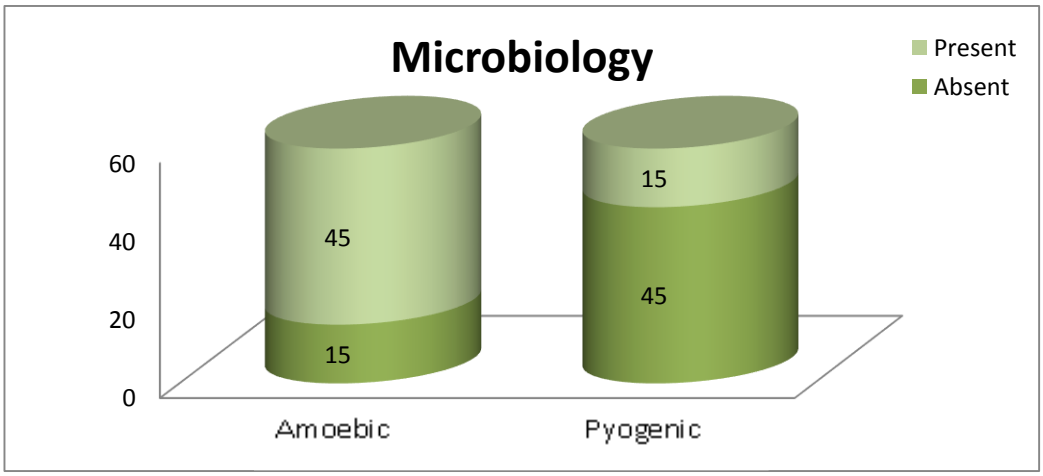
Table.19

AMOEBIC	Frequency
Absent	15
Present	45
Total	**60**

Table.20

PYOGENIC	Frequency
Absent	45
Present	15
Total	**60**

Chart. 7.



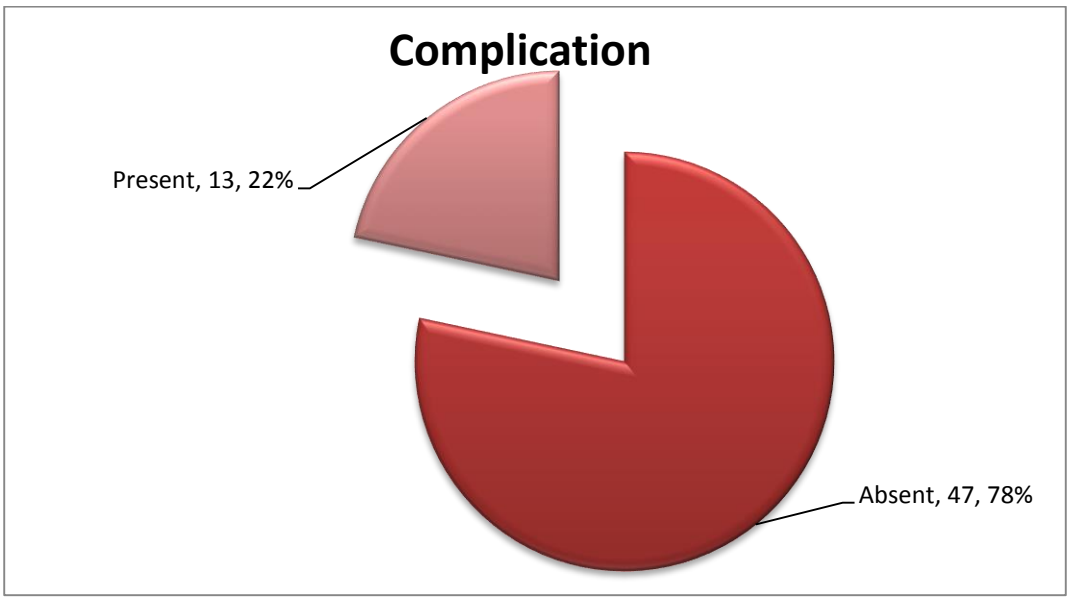
Out of the 60 cases in this study, 45 patients (75%) were diagnosed to have amoebic liver abscesses, remaining 15 patients(25%) were diagnosed to have pyogenic liver abscesses.

COMPLICATIONS

Table. 21

COMP	Frequency
Absent	47
Present	13
Total	**60**

Chart. 8.



In this study complications occurs in 22% of people who are all not responding to conservative line of management.

DISCUSSION

In this study 60 patients who met the inclusion criteria were included. In our study, the mean age of distribution of liver abscess is 45.95.

In the study, liver abscess are present in 47 males (78.3%) and 13 are females (21.7%).

In our study, most common presentation was abdominal pain (44%), then fever (31%). Out of 60 patients, 33 patients had intercostal tenderness (55%).

In this study, in case of pyogenic liver abscess most common presentation was fever, whereas in case of amoebic liver abscess most common presentation was abdominal pain.

Out of 60 cases, abscess located in right lobe is 53 cases (88.3%). In this study, most of the abscesses were solitary (90%). Out of 60 patients in our study, 11 patients (18%) responded to medical management alone .Then 27 patients (45%) were treated by image guided aspiration or drainage. Those who required and treated by surgical drainage was 22 patient (37%), who are all not responding to the above modalities of treatment and those with complication.

Out of the 60 cases in this study, 45 patients (75%) were diagnosed to have Amoebic liver abscesses, remaining 15 patients (25%) were diagnosed to have pyogenic liver abscesses. In this study complications occurs in 22% of people who are all not responding to conservative line of management.

CONCLUSION

Most common liver abscess presenting in our institution was amoebic in nature. Most common age of presentation was 45 years. Most common presentation of liver abscess was abdominal pain. Most common clinical sign was intercostal tenderness.

Alcoholism becomes the most frequently associated risk factor. Ultrasound and CT scan abdomen plays an important role in diagnosing most of the liver abscess patients presented in our institution. Eighteen percent (18%) of the liver abscess patients were managed successfully with medical management alone.

Those who are all not responding to medical management were treated with image guided drainage/aspiration. Forty five percent (45%) of the patients presented in our institution were successfully treated by image guided drainage/aspiration.

Those who developed complications (ruptured liver abscess) and who are all not responding to conservative line of management required emergency open drainage. Thirty seven percent (37%) of patients who developed complications on presentation or later were treated with open drainage in our institution.

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PROFORMA

PARTICULARS OF PATIENT:

Name	:	Case no	:
Age	:		
Sex	:	Date of Admission	:
Religion	:	Date of Operation	:
IP.No	:	Date of Discharge	:
Address	:		

Chief Complaints:

- Abdominal pain
- Fever
- Jaundice
- Other manifestions

History of presenting complaints:

Abdomen pain:

- Duration
- Time of onset
- Mode of onset

- Site of pain
- Radiation of pain
- Character of pain
- Aggravating factors
- Relieving factors

Fever:

- Duration
- Type
- Severity
- Jaundice
- Loss of appetite
- Loss of weight

Past history:

- Similar complaints
- Abdominal surgery

Personal history:

- Diet
- Appetite
- Sleep
- Alcohol intake
- Smoking
- Bowel/Bladder

Family history:

Menstrual history:

- Menarche
- Lmp
- Menstrual cycles

PHYSICAL EXAMINATION

General physical examination:

Built, Pallor

Nourishment, Icterus

Lymhpadenopathy, Cyanosis

Clubbing , Pedal edema

VITALS:

Pulse

Blood pressure

Respiratory rate

Temperature

ABDOMINAL EXAMINATION

Inspection:

- Contour of abdomen
- Movements of all quadrants with abdomen
- Visible pulsation and peristalsis
- Skin
- Hernial orifices
- Umbilicus
- Scrotum

Palpation:

- Local rise of temperature
- Local Tenderness
- Right hypochondrial tenderness
- Intercostal tenderness
- Any organomegaly

Percussion:

Auscultation:

Rectal examination:

SYSTEMIC EXAMINATION

Cardiovascular system :

Respiratory system :

Central Nervous system :

Provisional diagnosis :

Blood investigations :

Total count

Bilirubin

PT/INR

Albumin

Serology for amoebic antigen.

Imaging studies

CXR

X ray abdomen erect

USG abdomen

CT abdomen

Complications if developed will be assessed in detail.

Management strategies for liver abscesses followed

Medical management

Image guided drainage

Open drainage

Complications –followed up.

Follow up of patients:

Patients will be followed up over a period of 6 months

1. once in 2 weeks for first 2 months
2. then on a monthly basis after discharge, To look for recurrent attacks or To look for the development of complications and To monitor the efficacy of the treatment given.

CONSENT FORM

For Operation / Anaesthesia/ Study

I _____Hosp. No._____ in my full Senses hereby give my complete consent for _____ or any other Procedure deemed at which is a / and diagnostic procedure / biopsy / Transfusion/ operation to be performed on me / my ward _____ age _____under any anaesthesia deemed fit. The nature and risks. Involved in the procedure have been explained to me to my satisfaction. For academic and scientific purpose, the operation / procedure may be. Televised or photographed.

Date:

Signature / Thumb Impression

Name :

of patient / Guardian

Designation :

Guardian

Relationship :

Full address

KEYWORDS TO MASTER CHART

S.No- SERIAL NUMBER

SEX

1. MALE
2. FEMALE

ABDOMINAL PAIN

1-PRESENT

0-ABSENT

FEVER

1-PRESENT

0-ABSENT

JAUNDICE

1-PRESENT

0-ABSENT

OTHER SYMPTOMS

1-PESENT

0-ABSENT’

IT-INTERCOSTAL TENDERNESS

1-PRESENT

0-ABSENT

TC-TOTAL COUNT

INR-INTERNATIONAL NORMALIZED RATIO

ALB-ALBUMIN

SITE

2-RIGHT

1-LEFT

SINGLE/ MULTIPLE

1-SINGLE

2-MULTIPLE

MEDICAL MANAGEMENT

1-RESPONDING

0/2-FAILURE

IMAGE GUIDED DRAINAGE

1-RESPONDING

0-FAILURE

OPEN DRAINAGE

0-NOT REQUIRED

1-REQUIRED PTS

ETIOLOGY

1-AMOEBIC

0-NON AMOEBIC

1-PYOGENIC

0-NOT PYOGENIC

COMPLICATIONS

1-PRESENT

0-ABSENT

S.NO	AGE	SEX	ABD. PAIN	FEVER	JAUNDICE	OTHER	IT	TC	BILIRUBIN	INR	ALB	SIZE	SITE	SIN/ MULTI	MEDICAL	IMAGE	OPEN DRAIN	AMOEBIC	PYOGE	COMP
1	40	1	1	1	0	0	1	12000	0.9	14	3.8	4	2	1	1	0	0	1	0	0
2	38	1	1	1	1	1	1	9000	3.8	13	3.9	6	2	1	0	1	0	1	0	0
3	60	1	1	1	1	1	1	13000	4.2	14	4	5	2	1	2	1	0	0	1	0
4	30	1	1	0	0	0	1	10020	0.6	15	4.1	3	2	1	1	0	0	1	0	0
5	42	1	1	1	0	0	1	6800	0.8	14	4.2	4	2	1	2	1	0	1	0	1
6	38	2	1	0	0	0	0	7820	0.9	12	4	3	2	1	1	0	0	1	0	0
7	41	2	0	1	0	1	1	8810	0.8	13	3.9	7	2	1	2	1	0	0	1	1
8	26	1	1	1	0	1	0	9820	0.9	14	4	5	2	2	2	2	1	1	0	1
9	40	1	1	0	0	0	1	7000	0.6	16	3.8	4	1	1	2	1	0	1	0	0
10	55	1	0	1	1	0	0	11000	3	16	3.9	7	2	1	2	2	1	0	1	1
11	40	1	1	0	1	1	0	8400	4.1	15	4.1	3	2	1	0	1	0	1	0	0
12	32	2	0	0	0	1	1	9200	0.9	14	4.2	4	2	2	0	1	0	1	0	0
13	36	1	1	0	0	0	0	11808	1	13	3.9	6	2	1	0	0	1	0	1	0
14	65	2	0	1	0	1	1	12000	0.8	17	4	7	1	1	0	0	1	0	1	1
15	44	1	1	1	0	0	1	12000	0.9	14	3.8	4	1	1	0	1	0	1	0	0
16	40	1	1	1	0	1	0	7000	0.8	14	4.1	3	2	1	1	0	0	1	0	0
17	55	1	1	0	0	1	0	8800	1.1	14	4.1	4	2	1	2	1	0	1	0	0
18	50	2	1	1	1	0	1	9900	2.9	18	4.2	6	2	1	2	1	0	1	0	0
19	38	1	1	0	0	0	0	10100	1	13	4	8	2	1	0	2	1	1	0	0
20	52	2	0	1	1	0	0	11200	4	16	4	4	2	1	2	1	0	0	1	0

S.NO	AGE	SEX	ABD. PAIN	FEVER	JAUNDICE	OTHER	IT	TC	BILIRUBIN	INR	ALB	SIZE	SITE	SIN/ MULTI	MEDICAL	IMAGE	OPEN DRAIN	AMOEBIC	PYOGE	COMP
21	30	1	1	0	0	1	1	10000	1	14	4.1	3	2	1	1	0	0	1	0	0
22	24	1	1	0	0	1	0	9620	0.8	13	3.9	4	2	1	0	1	0	1	0	0
23	50	1	1	0	1	0	1	8400	3	16	4.2	5	1	1	0	2	1	1	0	0
24	48	1	1	1	0	0	1	9900	0.9	13	4.2	4	2	1	0	1	0	1	0	0
25	46	1	0	1	0	0	1	10110	1	12	4.1	5	2	2	0	2	1	1	0	1
26	44	2	1	1	0	1	0	7800	1.1	12	4.5	6	2	1	0	2	1	1	0	0
27	38	1	1	0	0	0	0	6400	0.8	12	4.4	4	2	1	0	1	0	1	0	1
28	70	1	0	1	1	1	0	7100	4.2	15	3.2	6	2	1	0	2	1	0	1	0
29	68	1	1	0	0	0	1	8900	0.7	13	3.1	5	2	1	0	2	1	1	0	0
30	49	1	1	1	0	0	1	12800	0.8	13	4	5	2	1	2	1	0	0	1	0
31	41	1	1	0	0	0	1	8900	1	14	4.2	3	2	1	1	0	0	1	0	0
32	72	1	0	1	1	0	1	11000	2.4	16	4	5	2	1	0	1	0	0	1	0
33	32	1	1	0	0	1	0	8900	1.1	13	4.5	4	2	1	0	1	0	1	0	0
34	38	1	1	1	0	0	0	12000	0.9	12	4.6	3	1	1	2	1	0	1	0	0
35	44	1	1	0	0	0	0	9800	0.8	14	4.7	5	2	1	0	2	1	1	0	0
36	51	1	0	1	0	0	0	6000	1	13	3.8	2	2	2	1	0	0	1	0	0
37	45	2	1	0	0	1	1	10200	0.6	16	3.4	3.5	2	1	0	1	0	1	0	0
38	62	1	0	1	1	0	0	13000	4	17	3	7	2	1	0	0	1	0	1	1
39	29	2	1	0	0	0	1	7620	0.9	12	3.9	5	1	1	0	1	0	1	0	0
40	40	1	1	0	0	0	1	8880	1.1	14	4.1	4	2	1	0	1	0	1	0	0

S.NO	AGE	SEX	ABD. PAIN	FEVER	JAUNDICE	OTHER	IT	TC	BILIRUBIN	INR	ALB	SIZE	SITE	SIN/ MULTI	MEDICAL	IMAGE	OPEN DRAIN	AMOEBIC	PYOGE	COMP
41	45	1	1	0	0	0	1	8800	1	13	4.2	3	2	1	1	0	0	1	0	0
42	40	1	1	1	0	0	0	11000	1.2	14	4.5	5	2	1	0	1	0	1	0	0
43	38	1	1	0	0	1	0	8900	0.6	14	4.1	2.5	2	1	1	0	0	1	0	0
44	44	1	0	1	0	1	0	4800	0.9	12	3.8	4.5	2	1	0	1	0	1	0	0
45	26	1	1	0	0	0	1	8800	0.8	13	4.7	5.8	2	1	0	2	1	1	0	1
46	31	2	1	1	0	0	0	9600	1.1	12	4.3	4	2	2	0	1	0	1	0	0
47	42	1	1	0	1	0	1	11000	3.9	17	3.1	7	2	1	0	2	1	1	0	1
48	56	1	1	0	0	0	0	8600	0.8	18.5	3.8	3.5	1	1	0	1	0	1	0	0
49	73	1	0	1	1	1	1	12000	3.4	16	4.6	7.5	2	1	0	0	1	0	1	1
50	66	2	1	1	0	1	1	11900	0.9	13	4.8	8	2	1	0	0	1	0	1	0
51	42	1	1	0	0	0	0	8900	0.9	12	4.6	3.5	2	1	0	1	0	1	0	0
52	44	1	1	0	1	0	0	9600	1.1	13	4.1	4.9	2	1	0	1	0	1	0	0
53	38	1	0	0	0	1	1	7800	1.2	12	3.8	7	2	1	0	0	1	1	0	0
54	46	1	1	0	0	0	0	11000	0.8	14	3.1	2	2	1	1	0	0	1	0	0
55	52	1	0	1	0	1	1	7400	1	15	3	3.9	2	1	0	1	0	1	0	0
56	65	2	1	1	1	0	1	12000	4	16	4.1	7	2	1	0	0	1	0	1	1
57	46	1	1	1	0	0	1	9900	1.1	15	4.6	5	2	1	0	1	0	1	0	0
58	68	1	0	1	1	0	1	13000	3.6	14	3.9	8	2	1	0	0	1	0	1	0
59	42	2	1	0	0	1	1	9000	0.9	13	4.6	4	2	1	0	1	0	1	0	0
60	70	1	0	1	1	0	0	11800	3.8	15	3.1	5	2	2	0	0	1	0	1	1