A DISSERTATION ON

"A STUDY ON CLINICAL, LABOROTORY AND MANAGEMENT PROFILE IN PATIENTS WITH LIVER ABSCESS"

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CERTIFICATE

This is certify that the dissertation entitled "A STUDY ON CLINICAL, LABOROTORY AND MANAGEMENT PROFILE IN PATIENTS WITH LIVER ABSCESS" is a bonafide original work of **Dr. A. ARRUN KUMAAR.,** in partial fulfilment of the requirement for M.S. branch – I (General surgery) Examination of the Tamil Nadu Dr.M.G.R. Medical University to be held in APRIL, 2016 under my guidance and supervision in 2014 – 2015.

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DECLARATION

I hereby solemnly declare that the dissertation titled "A STUDY ON CLINICAL, LABOROTORY AND MANAGEMENT PROFILE IN PATIENTS WITH LIVER ABSCESS" is done by me at Madras medical college & Rajiv Gandhi Govt. General Hospital, Chennai during 2014 – 2015 under guidance and supervision of Prof.Dr.P.RAGUMANI, M.S. the dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, Chennai towards the partial fulfilment of requirements for the award of M.S. Degree (Branch – I) in General surgery.

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LIST OF ABBREVIATIONS

TB	- -	Tuberculosis
HIV	- -	Human Immunodeficiency Virus
USG	- -	Ultrasonogram
CECT	- -	Contrast Enhanced Computed Tomography
MRI	- -	Magnetic Resonance Imaging
ALP	- -	Alkaline Phosphatase
CGD	- -	Chronic Granulomatous Disease
AFP	- -	Alfa Feto Protein
CEA	- -	Carcinoembryonic Antigen
GGT	- -	Gamma Glutamyl Transpeptidase
PT	- -	Prothrombin Time
INR	- -	International Normalized Ratio

“A STUDY ON CLINICAL, LABOROTORY AND MANAGEMENT PROFILE IN PATIENTS WITH LIVER ABSCESS”

ABSTRACT

BACKGROUND

Liver abscess is a commom surgical problem in our country. Our study aims to review the literature on the management of liver abscess,focusing on the choice of drainage.A case series of our experience with clinical pathological correlation is presented to highlight the indication and outcome of each modality of drainage.

METHODS

My study is an observational prospective and retrospective study with approximately 50 patients of liver abscess. Method of sampling was non-random, purposive. Baseline investigations, as routinely required, were done followed by imaging studies(USG & CT abd)

RESULTS

Age of 50 patients ranged from 18-75 years. The patients were predominantly of the more than 40 age group. Around forty three patients (86%) were males, the high amount of prevalence of liver abscess among males can be explained by increased risk factors like alcohol consumption.On analysing the risk factors, as expected, chronic alcoholism was the predominant risk factor, seen in 16 patients (32%), with diabetes seen in five patients and systemic hypertension seen in four patients. Both Diabetes Mellitus and systemic hypertension was seen in seven patients (14%). Thirty six percent of the patients had no identifiable risk

factor. On evaluation of the presenting symptoms, thirty four patients (68%) had fever usually associated with chills and rigors. Thirty one patients (62%) had right hypochondrial pain and tenderness. Nearly all the patients had either of these symptoms. None of them presented in exclusion of either of these symptoms.Eighteen patients (36%) had complaints of vomiting and anorexia. Six patients complained of jaundice and another five patients complained of breathlessness. An analysis of the pus culture and sensitivity from the discharge, showed the presence of Entamoeba histolytic in twenty two patients (44%). Bacterial infection was seen in the remaining twenty eight patients with common organisms being E.Coli,Klebsiella, Staphylococcus aureus, with thirteen patients (26%) having polymicrobial infection. Regarding the procedures performed, nineteen patients (38%) underwent laparotomy and drainage, while the majority of the patients, around twenty nine (58%) underwent ultra sonogram guided pig tail drainage. Conservative management was tried in two patients (4%). On comparing the procedure underwent with the size of abscess the patient had it was found that, fifteen of twenty three patients with abscess size greater than 5 cm underwent laparotomy and drainage with pig tail drainage done for eight patients and conservative management attempted in none of these patients. A vast majority of patients, more than eighty percent with size less than five cm, were managed with pig tail drainage, while open drainage was done in eight patients and two patients were attempted conservative management. Six patients had ruptured liver abscess with laparotomy and peritoneal lavage being done for all these six patients. All six of these patients developing complications, with three patients going into sepsis while three patients expired due to multi organ dysfunction syndrome. Residual abscess was seen in three patients who had underwent pigtail drainage. Both the conservatively managed patients recovered Uneventfully.

CONCLUSION

Chronic Alcohol intake is a definitive risk factor for development of liver abscess. Diabetes Mellitus is the prevalent comorbid factor, seen especially in the elderly. Other comorbid factors include hypertension, bronchial asthma etc. None of these seemed to have a significant correlation with the disease process. Pig tail drainage is preferred in patients with single abscess of size less than 5 cm situated in superficial segments. Laparotomy and drainage was done in patients with ruptured or impeding rupture abscess. Conservative management with antibiotics was also useful in very small single cavity abscess. The incidence of complications was predominantly seen in patients with ruptured liver abscess who underwent laparotomy, in form of sepsis followed by MODS and death. Residual abscess cavity was seen in small number of patients following pig tail drainage but it was not Significant.

KEYWORDS:

Liver Abscess, Pigtail catheter, Laparotomy, Alcoholic.

INTRODUCTION

1.1 RATIONALE OF STUDY

Our study aims to review the literature on the management of liver abscess,focusing on the choice of drainage.A case series of our experience with clinicalpathological correlation is presented to highlight the indication and outcome of each modality of drainage.IV antibiotics is the first line of management.Drainage is necessary for large abscess,equal to or larger than 5cm in size to facilitate resolution.While percutaneous drainage is appropriate as first line surgical treatment in most cases,open drainage is prudent in cases of rupture, multiloculation, associated biliary or intra abdominal pathology.Percutaneous drainage may help to optimize clinical condition prior to surgery.Nevertheless, in current practice the choice of therapy needs to be individualized according to patient's clinical status and abscess factors.They are complementary in the management of liver abscess

1.2 AIMS AND OBJECTIVES

The purpose of the study is to

1. To analyse the various modes of clinical presentation of liver abscess
2. To analyse the common causative organism in patients presenting to RGGGH
3. To determine the choice of drainage
4. To analyse the morbidity and mortality and to determine the prognostic factors

CHAPTER 2

REVIEW OF LITERATURE

REVIEW OF LITERATURE

2.1 ANATOMY OF LIVER

Couinaud derived the anatomic concepts of modern liver surgery in 1957 which was later developed by Ton That Tung in 1962 and Bismuth in 1982.

LOCATION AND EXTENT

The liver occupies the right hypochondrium and the epigastric regions and extends inferiorly into the right lumbar region and occupies part of the left hypochondrium, reaching to the left lateral line. The liver is covered by ribs and costal cartilages, except in the epigastric region where it reaches the anterior abdominal wall just below the infrasternal notch.

The right side of the liver is closely applied to the costal muscle fibers and the central tendon of the right leaf of the diaphragm. The left lobe (the apex of the "wedge") reaches for a variable distance into the left upper part of the abdominal cavity, abutting the left leaf of the diaphragm.

The anatomic and nonanatomic factors responsible for the fixation of the liver at the right upper quadrant of the abdomen was described by Flament et al.

Anatomic Inferior vena cava

Suprahepatic veins

Nonanatomic

Positive intraabdominal pressure

MOTION AND ACTIVITY

The position of the liver in the body is not static. The liver moves up and down with the diaphragm and rotates during respiration. It rotates backward when an individual lies down in the supine position. The upper surface of the liver can move upward from 1 cm to 10 cm when full expiration follows deep inspiration. The most cranial level attained by the upper border of the liver during quiet respiration shows great individual variation and reflects the type of diaphragm — high, intermediate, or low.

The healthy adult liver weighs between 1.0 kg and 2.0 kg.

Dimensions of the liver are as follows:

Anteroposteriorly, the distance extends 10.0 cm to 12.5 cm from the area related to the anterior abdominal wall to its rounded posterior surface

– The transverse diameter is 20.0 cm to 25.5 cm from the right paracolic gutter to the midpoint of the left diaphragmatic leaflet

– The anteroinferior edge stretches vertically 15.0 cm to 17.5 cm to the top of the dome of the right hepatic lobe

According to Gelfand, the obscurity of the hepatic borders on x-ray is due to the specific gravity of the liver (1.05). This is almost the same as all other "water density" tissues such as the diaphragm and gastrointestinal wall. The presence of fat helps prevent the blending of the liver margins with other organs. The same author stated that the anatomic relations between hepatic substance and fat are extremely important for radiographic determination of the hepatic borders.

The shadow of the inferior border expressed on x-rays is due to the combined effects of the amount of retroperitoneal fat, habitus of the patient, and "hepatic angle" (junction between lateral and inferior hepatic borders).

MORPHOLOGY ANATOMY

The external appearance of the liver and its ligamentous attachments are described in the classical morphological anatomy. The liver is a thoracoabdominal organ:

It is covered by a thin fibrous capsule—an expansion of the peritoneum(Glisson capsule)and is molded by the adjacent organs.

Ligamental attachments of the liver

The falciform ligament and three other peritoneal folds suspends the liver from the diaphragm and anterior abdominal wall. Ligamentum teres arises from the end of the left portal branch and forms the lower, visceral border of the falciform

ligament.It is a fibrous cord formed by the obliteration of the umbilical vein.The left lobe is smaller than the right lobe. The round ligament and the umbilical fissure corresponds with the division of 2 lobes at the inferior surface. The superior surface of the right lobe is convex under the diaphragm.The inferior concave surface contains a fissure (the hilum or porta hepatis), in which the major vessels and bile ducts enter and leave the liver through a fissure.Mostly retroperitoneal posterior surface lies in contact with the IVC and is enclosed by the leaves of the coronary and triangular ligaments . The posterior part of the inferior surface of the right lobe is divided by the transverse fissure of the hilum. At the inferior surface of the right lobe the quadrate lobe lies to the left of the gallbladder fossa, to the right of the umbilical fissure, anterior to the hilum.The left lobe, the smaller portion of the liver, lies to the left of the falciform and round ligaments and the hilum. A fourth lobe named The Caudate lobe lies posterior to the transverse fissure of the hilum, and it is delimited at the left by the ligament of Arentius (ligament venosum), with the insertion of the lesser omentum. It has no visible structure that delimited it on the right side.The caudate lobe has been subdivided in a paracaval portion that encircled the side of theIVC and a spigelian lobe. At the right side of vena cava, a hepatocaval ligament is always present between the high part of the right lobe and the vena cava.some authors consider this ligament as the ultimate right part of the caudate lobe that surrounds the vena cava, and is not apparent when viewing the liver from the anterior surface. The Arantius ligament

separates the spigelian lobe on the left underneath, and the left lobe on the top. The Arantius ligament is secondary to the obliteration of the ductus venous that,during fetal development, connected the umbilical portion of the portal vein to the inferior vena cava, shunting oxygenated umbilical cord blood away from the liver. Fine anatomic analysis has recently demonstrated that this ligament runs from the left branch of the portal vein to the left hepatic vein or at the junction between the left and middle hepatic veins.

Hence, the liver comprises two main lobes (right and left) and two accessory lobes (quadrate and spigelian) separated by fissures. The term *lobe* is justified because it is defined as a "part of parenchyma limited by fissures or grooves."

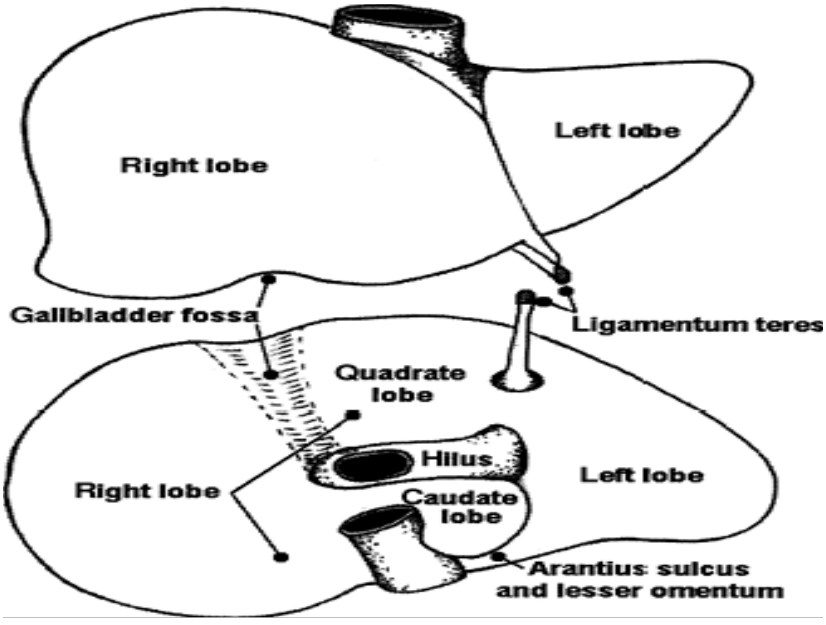


FIG 1. ANATOMY

TOPOGRAPHIC RELATIONS

Diaphragmatic Surface

The diaphragmatic surface may be divided into superior, posterior, anterior, and right portions.

Superior: The superior portion is related to the diaphragm and the following organs from right to left: right pleura and lung, pericardium and heart (cardiac impression), left pleura and lung. The superior surface is covered with peritoneum except where, more dorsally, the superior reflection of the coronary ligament bounds the bare area of the liver.

Posterior: The posterior portion is related to the diaphragm and lower ribs. It contains the greater part of the bare area and the sulcus of the inferior vena cava (IVC).

Anterior: The anterior part is related to the diaphragm and costal margin, xiphoid process, the abdominal wall, and the sixth to tenth ribs on the right.

Right: The right portion is related to the diaphragm and the seventh to eleventh ribs. It is a lateral continuation of the posterior portion.

The diaphragmatic surface separates from the visceral surface at the inferior border. This surface is blunt, rounded, and unmarked posteriorly but sharp anteriorly. The clinician palpates this sharp anterior portion. However, the liver

"edge" seen in a plain x-ray is the rounded posterior border. This is not a true border but represents the interface of the posterior aspect of the right lobe of the liver with retroperitoneal fat. Anteriorly, the inferior border of the liver is marked by two notches to the right of the median plane. These are: Deep notch accommodating the ligamentum teres & Shallow notch allowing space for the gallbladder.

FUNCTIONAL ANATOMY

Functional anatomy describes the hepatic segmentation that is the real anatomic basis for modern hepatic surgery. The description by Couinaud is the most complete and, although it appears complex at first, its exactness has been provenuseful by its extensive application to liver surgery. In this functional anatomy, eight segments are numbered in a clockwise manner starting with the caudate lobe as segment 1 seen in an anterior view point.

The glissonian pedicles (also called *portal pedicles* or *portal canals*) enter the liver at the hilum. The the triad of portal vein, hepatic artery, and bile duct inside a sheath formed by an extension of Glisson capsule constitutes the glissonian pedicles.The glissonian pedicle divides into left and right branches at the right of the hilum. The left branch containing the left branch of the hepatic vein, is longer and curves forward along the umbilical scissura before entering the

hepatic parenchyma at the recessus of Rex. The right branch is short and directly enters the hepatic parenchyma.

The porta scissurae is the three scissurae containing the right, middle, and left hepatic veins.It divides the liver into four sectors called *portal sectors* in Couinaud's nomenclature (in Anglo-Saxon nomenclature they are called *segments*, but in the Couinaud nomenclature, the term *segment* is reserved for a specific subdivision of a sector). Each sector is invested by a glissonian pedicle and its ramifications and is independent of the others. The main portal scissura, containing the middle hepatic vein, begins anteriorly at the middle of the gallbladder bed and posteriorly at the left of the vena cava. It follows an angle of 75 degrees from the left horizontal plane. Thus, the main portal scissura (sometimes called *Cantle line*) separates the liver into two parts—the right and left hemilivers. The right and left livers individualized by the main portal scissura are independent with respect to their portal and arterial vascularization and their biliary drainage. These functional right and left livers should not be called *lobes* because there are no surface markings to distinguish them, which could cause confusion with the morphologic anatomic description. The functional right liver doesnot correspond to the anatomic right lobe.The right liver is divided into two portions by the right portal scissura containing the right hepatic vein; the left liver is divided by the left portal scissura containing the left hepatic vein.The right portal scissura divides the right liver into

anteromedial and posterolateral sectors,clearly seen when the liver undergoes autopsy and is placed on a flat table, where it adopts the bench position. In situ, however, the liver is molded around the spine, and these two sectors are better described as anterior and posterior sectors in all the morphologic examinations of the liver (e.g., ultrasound, CT scan, arteriography). The right portal scissura is inclined approximately 40 degrees to the right. The exact location of the right portal scissura is not well defined because it has no external landmark. According to Couinaud, it extends from the anterior surface of the liver at the anterior border, midway between the right angle of the liver and the right side of the gallbladder bed, to the confluence of the inferior vena cava and the right hepatic vein posteriorly . According to Ton That Tung, this scissura follows a line parallel to the right lateral edge of the liver three fingerbreadths from the edge. The right hepatic vein runs along the right portal scissura, but in CT scans it can be an inaccurate indicator of the position of the scissura, and intraoperative ultrasonography is better for localizing this structure.

Perihepatic Spaces

We follow the nomenclature of Whalen and of Ochsner and DeBakey in part, realizing that it is arbitrary. The spaces formed by the peritoneum in the supracolic compartment are those above and below the liver.

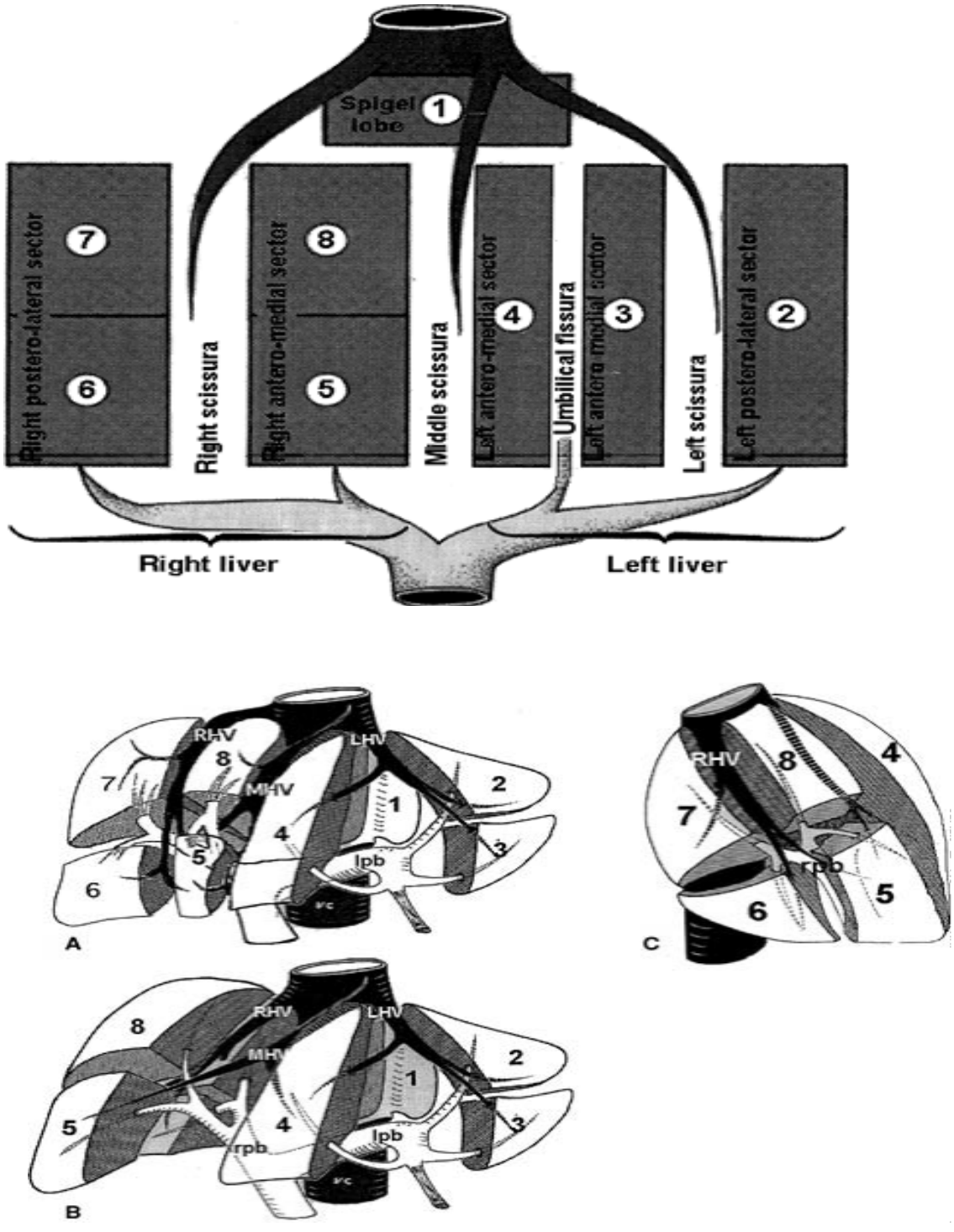


FIG 2. SEGMENTS OF LIVER

Suprahepatic Spaces

The bare area of liver is the portion where the superior surface of the liver and a corresponding portion of the inferior surface of the diaphragm are in direct contact with one another without being covered by peritoneum. Its margins are the falciform, coronary, and left and right triangular ligaments of the liver

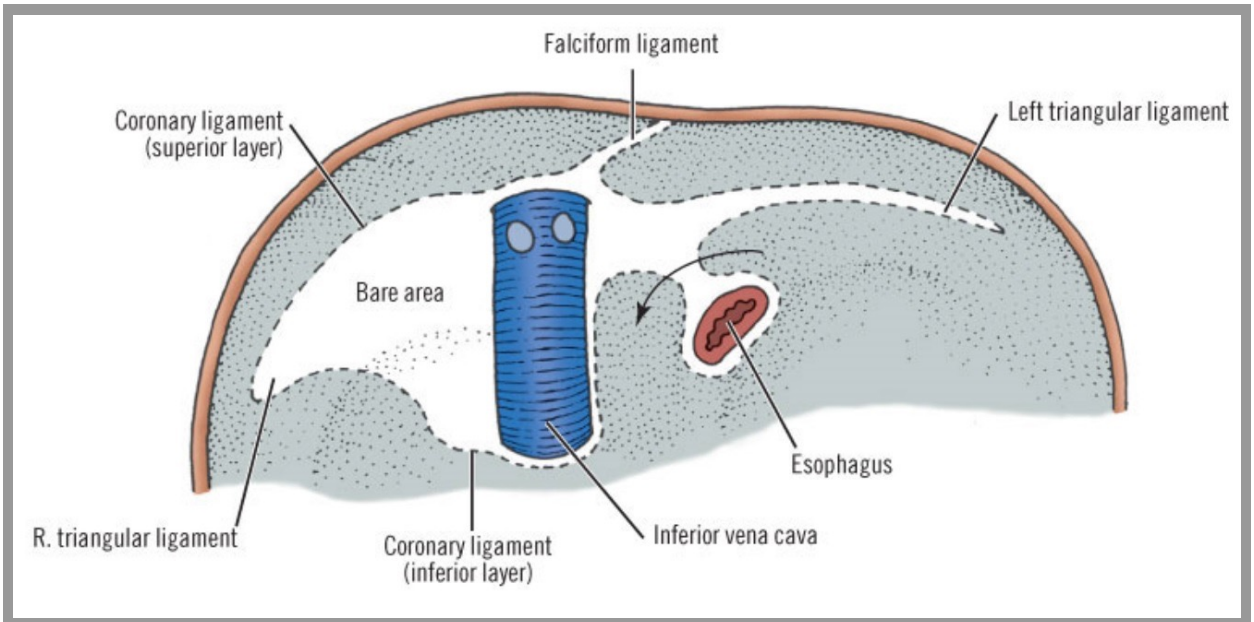


FIG 3

The inferior surface of the diaphragm showing the peritoneal attachments of the liver *(dashed lines)*. Within the boundaries of these attachments is the "bare area" of the liver and the diaphragm. The arrow represents the pathway behind the abdominal esophagus where surgeons may pass a finger through the inferior layer of the coronary ligament.

Except over its bare area, the serous surfaces of the liver and diaphragm are side by side and separated by a potential space. This potential space may become the site of intraperitoneal fluid collection and of suprahepatic (subphrenic) abscess.

The suprahepatic potential space is divided into right and left spaces by the falciform ligament. The right suprahepatic space lies between the diaphragm and the anterosuperior surface of the right lobe and the medial segment of the left lobe of the liver. The boundaries are:

Left — falciform ligament

Posterior — right superior coronary and right triangular ligaments

Inferior — right lobe and medial segment of the left lobe of the liver

The space opens into the general peritoneal cavity anteriorly and inferiorly.

The corresponding suprahepatic space on the left is between the diaphragm and the superior surface of the lateral segment of the left lobe of the liver and the fundus of the stomach. To the right, the left suprahepatic space is bounded by the falciform ligament and, posteriorly, by the left coronary and triangular ligaments. Anteriorly and laterally, the space communicates with the infrahepatic space and the general peritoneal cavity. On the left, the anterior and posterior leaves of the coronary ligament are side by side. The left triangular ligament separates the anterior and superior suprahepatic spaces.

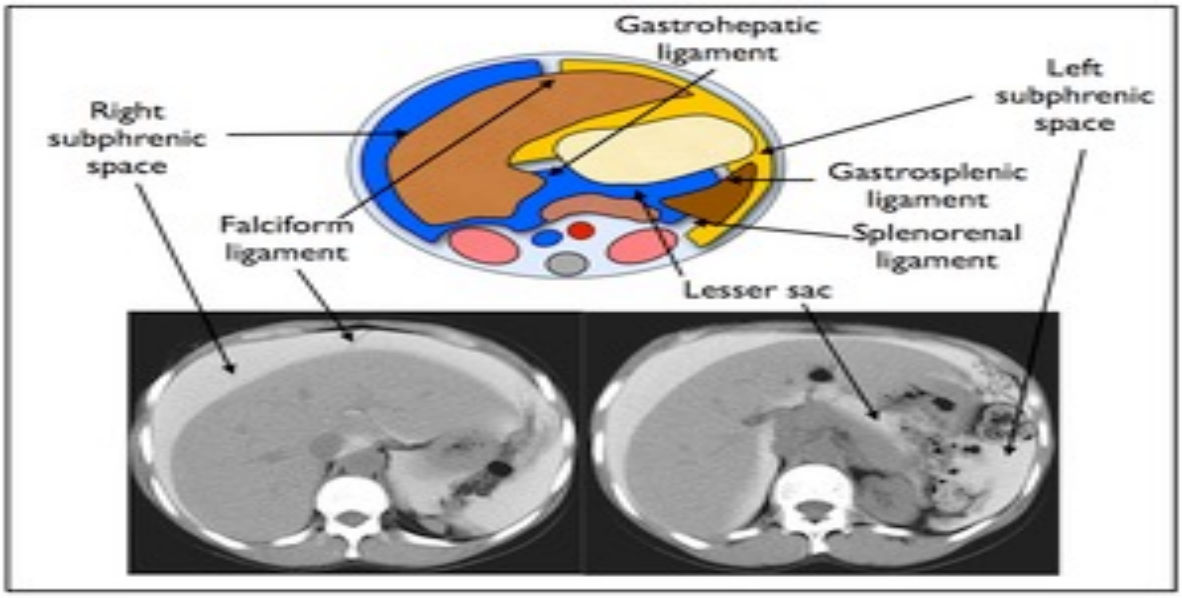


FIG 4. ANATOMIC RELATIONS

Min et al. report that the posterior left suprahepatic space is located anterior and superior to the lesser sac, with inferior continuation to the gastrohepatic space. These authors emphasize that the left posterior suprahepatic space and the lesser sac are separated by the lesser omentum and the stomach. Each suprahepatic space may be divided into anterior and posterior portions. The distinction is unimportant in the absence of disease. On the right, fluid may collect or an abscess may form between the liver and diaphragm anteriorly just beneath the sternum (right anterior suprahepatic abscess) . Or an abscess may form at the reflection of the superior leaf of the coronary ligament (right posterior suprahepatic abscess) .The single space of the anatomist may be divided by pseudomembranes into two spaces.

The left suprahepatic space may be similarly compartmentalized by pseudomembranes between the liver and diaphragm or the abdominal wall. The left suprahepatic and left anterior infrahepatic spaces are not separated anatomically, but they may become separate pathologically by pseudomembranes. Large accumulations of fluid may extend into the subhepatic space where the stomach, spleen, and liver participate in walling off the infection. The diaphragm is usually elevated over the abscess or fluid collection.

Anteriorly, a surgical approach from beneath the costal margin presents no anatomic complications. Posteriorly, the approach must be by an incision at the level of the spinous process of the first lumbar vertebra. This method avoids the pleura. The pleura and the twelfth rib are related at the vertebral spine. Thus the surgeon must avoid traversing the bed of the twelfth rib.

Infrahepatic Spaces

The right infrahepatic space(subhepatic space, hepatorenal space, pouch of Morison) is bounded superiorly and anteriorly by the right lobe and medial segment of the left lobe of the liver and the gallbladder. It is limited superiorly and posteriorly by the inferior layer of the coronary ligament and the posterior layer of the right triangular ligament. Inferiorly, the space opens into the general peritoneal cavity and is partly bounded by the hepatic flexure of the colon and the transverse mesocolon and, medially, by the hepatoduodenal ligament. The right suprahepatic

space communicates with the right infrahepatic space in three places: the margin of the right lobe of the liver; the right triangular ligament; and a small space, the quadrangular space of Mitchell. The quadrangular space is bounded above by the quadrate lobe of the liver, below by the transverse colon, on the left by the falciform ligament, and on the right by the gallbladder. The coronary ligament suspends the liver not from above but from the dorsum. The left triangular ligament suspends the left lobe not from the apex of the diaphragm but from the dorsal aspect of the diaphragm. The left infrahepatic space may be divided into the smaller antegastric space and the larger lesser sac of the peritoneum.

Antegastric Space

The anterior space lies between the left lobe of the liver above and the stomach below and behind . The boundaries are:

Superior and anterior, the left lobe of the liver and the anterior abdominal wall

Posterior, the stomach and lesser omentum

Inferiorly, the middle third of the transverse colon.

This space has been termed "perigastric," but this is misleading; "paragastric" might be better. This space is entirely anterior to the stomach. Hollinshead believes it is merely part of the left suprahepatic (subphrenic) space in general.

Lesser Sac

The lesser sac of the peritoneum becomes, by the terminology used here, the left posterior infrahepatic space. This is a valid concept. For practical purposes, our two preferred terms are the "lesser sac" or the "omental bursa."

Extraperitoneal Spaces

There are two potential extraperitoneal spaces in which abscesses can occur. On the right, abscesses may form over the bare area of the liver that is outlined by the falciform, coronary, and triangular ligaments. On the left, they may occur in a poorly defined space that is bounded by the distal pancreas, the descending (left) colon, the upper pole of the left kidney, the left adrenal gland, Gerota's perirenal fascia, and fat. Altemeier and Alexander discuss these and other sites of retroperitoneal abscesses.

Surgical Considerations

Ultrasonography, CT scan, or MRI will help the surgeon decide whether to use the closed or open method in the treatment of a perihepatic abscess. Percutaneous needle-guided drainage (closed method) may be achieved by guiding the needle into the abscess cavity, advancing a wire into the cavity, dilating the tract around the wire with successively larger dilators, and placing a self-retaining catheter into the cavity or cavities.

Open method

– Drain a right anterior subphrenic (suprahepatic) abscess using a small right subcostal incision to establish an extraperitoneal route.

– Reach a right posterior subphrenic abscess (suprahepatic or infrahepatic) using a posterior route through the bed of the already excised 12th rib. Push the right kidney and the right adrenal gland downward

– Approach a left anterior suprahepatic or infrahepatic abscess anteriorly using a small LUQ incision and proceeding intra- or extraperitoneally.

– A left posterior suprahepatic or infrahepatic abscess can be approached as on the right posterior, pushing the peritoneum down over the spleen and the gastric fundus. This forms a space between these two organs and the diaphragm

PORTAL VEIN

The portal vein provides approximately 75% of the hepatic blood inflow. Despite being postcapillary and largely deoxygenated, its high flow rate provides 50% to 70% of the liver's oxygen. 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. This allows for the measurement of portal venous pressure at any point along the system. The portal vein forms behind the neck of the pancreas at the confluence of the superior mesenteric vein and the splenic vein at the level of

the second lumbar vertebrae. The length of the main portal vein ranges from 5.5 to 8 cm and its diameter is usually approximately 1 cm. Cephalad to its formation behind the neck of the pancreas, the portal vein runs behind the first portion of the duodenum and into the hepatoduodenal ligament, where it runs along the right border of the lesser omentum, usually posterior to the common bile duct and proper hepatic artery.

The portal vein divides into main right and left branches at the hilum of the liver. The left branch of the portal vein runs transversely along the base of segment 4 and into the umbilical fissure, where it gives off branches to segments 2 and 3 and feedback branches to segment 4. 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; it 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 caudate process branch off the main right portal vein or at the right portal vein bifurcation, which comes off posteriorly to supply this portion of liver

Hepatic artery

The hepatic artery, representing high-volume oxygenated systemic arterial flow, provides approximately 25% of the hepatic blood flow and 30% to 50% of its

oxygenation. A number of smaller perihepatic arteries derived from the inferior phrenic and the gastroduodenal arteries also supply the liver.These vessels are important sources of collateral blood flow in case of occlusion of the main hepatic arterial inflow. In the case of ligation of the right or left hepatic artery, intrahepatic collaterals almost immediately provide for nutrient blood flow in most cases.

The common description of the arterial supply to the liver and biliary tree is only present approximately 60% of the time. The celiac trunk originates directly off the aorta,just below the aortic diaphragmatic hiatus, and gives off three branches—the splenic artery, left gastric artery, and common hepatic artery. The common hepatic artery beyond the takeoff of the gastroduodenal is called the *proper hepatic artery*; it divides into right and left hepatic arteries at the hilum. The left hepatic artery heads vertically towards the umbilical fissure to supply segments 2, 3, and 4.

The left hepatic artery usually also gives off a middle hepatic artery branch that heads toward the right side of the umbilical fissure and supplies segment 4. The right hepatic artery usually runs posterior to the common hepatic bile duct and enters Calot's triangle, bordered by the cystic duct, common hepatic duct, and liver edge, where it gives off the cystic artery to supply the gallbladder and then continues into the substance of the right liver.

Hepatic veins

The three major hepatic veins drain from the superior-posterior surface of the liver directly into the IVC The right hepatic vein runs in the right scissura between the anterior and posterior sectors of the right liver and drains most of the right liver after a short (1-cm) extrahepatic course into the right side of the IVC. The left and middle 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 between segments 2 and 3 and drains segments 2 and 3; the middle hepatic vein runs in the portal scissura between segment 4 and the anterior sector of the right liver, comprised of segments 5 and 8, and drains segment 4 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. A number of small posterior venous branches from the right posterior sector andcaudate lobe drain directly into the IVC. A substantial inferiorly located accessory right hepatic vein is commonly encountered.

There is also often a venous tributary from the caudate lobe,which drains superiorly into the left hepatic vein.

2.2 PHYSIOLOGY

Functional Unit of the Liver is referred to as an acinus or a lobule was originally described by Rappaport and then modified by Matsumoto and Kawakami. A polygonal unit is formed by a lobule is made up of a central terminal hepatic venule surrounded by four to six terminal portal triads. This unit is lined on its periphery between each terminal portal triad by terminal portal triad branches. Hepatocytes are arranged in one cell–thick plates, surrounded on each side by endothelial lined and blood-filled sinusoids in between the terminal portal triads and the central hepatic venule. Blood flows from the terminal portal triad through the sinusoids into the terminal hepatic venule.

Bile is formed within the hepatocytes

Empties into terminal canaliculi

Combines to form bile duct and flow towards portal triad

Between the terminal portal triad and central hepatic venule are three zones that differ in their enzymatic makeup, as well as exposure to nutrients and oxygenated

blood. zones 1 through 3 splay out from the terminal portal triad toward the central hepatic venule.

Zone 1 (periportal zone) is an environment rich in nutrients and oxygen

Zone 2 (intermediate zone) and Zone 3 (periventricular zone) are exposed to environment poorer in nutrients and oxygen. Terminal portal venous and hepatic arterial branches directly supply the hepatic sinusoids with blood. The portal branches provide a constant-, but minimal flow into this low-volume system; the arterial branches provide the sinusoids with pulsatile-, but low-volume flow that enhances flow in the sinusoids. Hepatic arterial branches terminate in a plexus around the terminal bile ductules and provide nutrients. Arterial and portal vein flow vary inversely in the sinusoids and can be compensatory. Local control of blood flow in the sinusoids likely depends on arteriolar sphincters and contraction of the sinusoidal lining by endothelial cells and hepatic stellate cells or portal myofibroblasts. Blood within the sinusoids empties directly intoterminal hepatic venules at the center of a functional lobule. This process results in the unidirectional flow of blood in the liver from zones 1 to 3.

Hepatic microcirculation : The endothelium-lined sinusoids of the hepatic lobule represent the functional unit of the liver, where afferent blood flow is exposed to functional hepatic parenchyma prior to being drained into hepatic venules. The hepatic sinusoids are 7 to 15 μm wide but can increase in size by up to 10-fold.This

yields a low-resistance and low-pressure (generally, 2 to 3 mm Hg) system. The sinusoidal endothelial cells account for 15% to 20% of the total hepatic cell mass.

Sinusoidal endothelial cells are separated from hepatocytes by the space of Disse (perisinusoidal space). This is an extravascular fluid compartment into which hepatocytes project microvilli,which allows proteins and other plasma components from the sinusoids to be taken up by the hepatocytes. Within this space, the endothelial cells are specialized in that they lack intercellular junctions and a basement membrane but contain multiple large fenestrations. This arrangement provides for the maximal contact of hepatocyte membranes with this extravascular fluid compartment and blood in the sinusoidal space. Thus, this system permits bidirectional movement of solutes (high and low-molecular-weight substances) into and out of hepatocytes,providing tremendous filtration potential. On the other hand, the fenestrations of the endothelial cells restrict movement of molecules between the sinusoids and hepatocytes and vary in response to exogenous and endogenous mediators. Kupffer cells, derived from the macrophage-monocyte system,are irregularly-shaped cells that also line the sinusoids insinuating between endothelial cells. Kupffer cells are phagocytic, can migrate along sinusoids to areas of injury, and play a major role in the trapping of foreign substances and initiating inflammatory responses. Major histocompatibility complex II antigens are

expressed on Kupffer cells, but do not confer efficient antigen presentation compared with macrophages elsewhere in the body.

Other lymphoid cells also exist in hepatic parenchyma, such as natural killer (NK), natural killer T (NKT), CD4 T, and CD8 T cells. These provide the liver with an innate immune system.Hepatic stellate cells, previously known as Ito cells, are cells high in retinoid content (accounting for their phenotypic identification) found in the space of Disse.

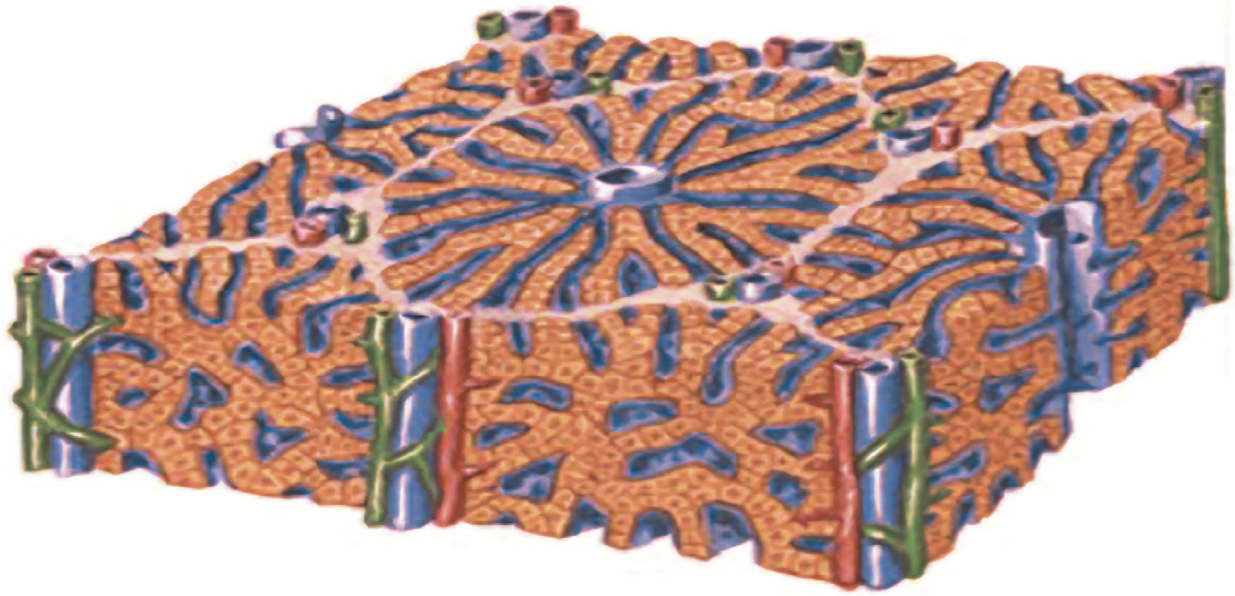


FIG 5. SINUSOIDAL ARCHITECTURE

FUNCTION OF LIVER

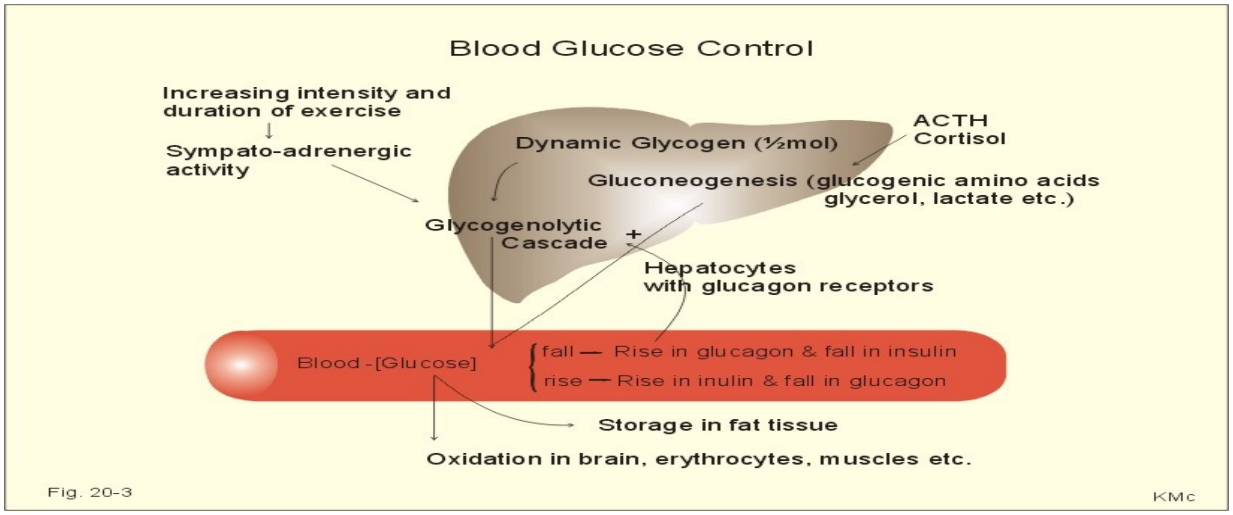
BILIRUBIN METABOLISM

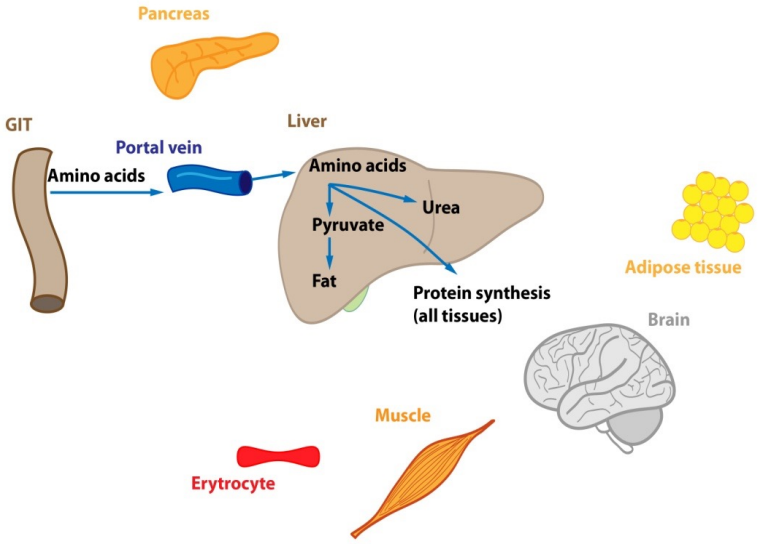
Bilirubin is the breakdown product of normal heme catabolism.Bilirubin is bound to albumin in the circulation and sent to the liver. In the liver, it is conjugated to glucuronic acid to form bilirubin diglucuronide in a reaction catalyzed by the enzyme glucuronyl transferase, making it water soluble. This glucuronide is then excreted into the bile canaliculi. A small amount dissolves in the blood and is then excreted in the urine. The majority of conjugated bilirubin is excreted in the intestine as waste, because the intestinal mucosa is relatively impermeable to conjugated bilirubin. However, it is permeable to unconjugated bilirubin and urobilinogens, a series of bilirubin derivatives formed by the action of bacteria. Thus, some of the bilirubin and urobilinogens are reabsorbed in the portal circulation;they are again excreted by the liver or enter the circulation and are excreted in the urine.

Energy

The liver is the critical intermediary between dietary sources of energy and the extrahepatic tissues that require this energy. The critical and central nature of the liver in regulating the body's energy metabolism is evidenced by the fact that despite accounting for only 4% of the total body weight, the liver consumes about

28% of the total body blood flow and 20% of the total oxygen consumed. The liver also uses about 20% of the total body caloric intake.The liver receives dietary byproducts through the portal circulation and sorts, metabolizes, and distributes them into the systemic circulation. The liver also plays a major role in regulating endogenous sources of energy such as fatty acids and glycerol from adipose tissues and lactate, pyruvate, and certain amino acids from skeletal muscle. The two major sources of energy that the liver releases into the extrahepatic circulation are glucose and acetoacetate. Glucose is derived from the glycogenolysis of stored glycogen and from gluconeogenesis from lactate, pyruvate, glycerol, propionate, and alanine. Acetoacetate is derived from the β-oxidation of fatty acids. Also, storage lipids such as triacylglycerols and phospholipids are synthesized and stored as lipoproteins by the liver. These can be circulated systemically for uptake by peripheral tissues. These complex and essential functions are regulated by hormones, overall nutritional state of the organism, and requirements of obligate glucose-requiring tissues.





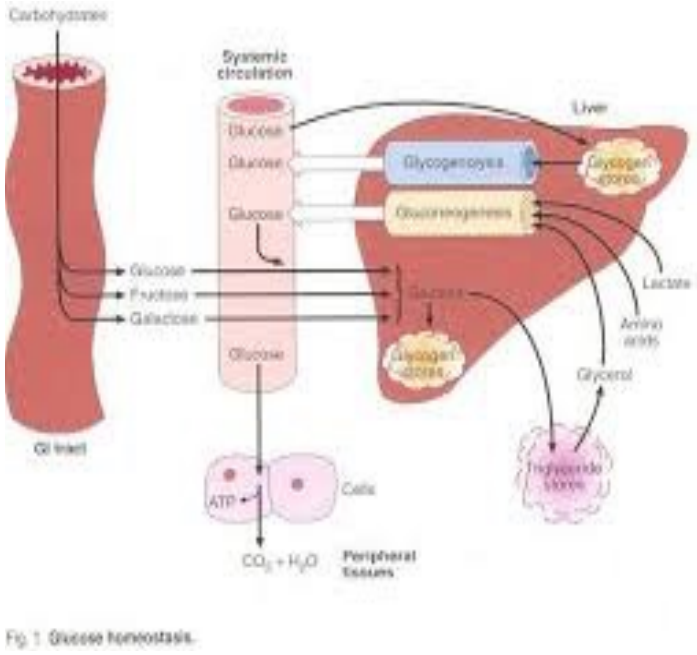


Fig. 1 Glucose homeostasis.

FIG 6. FUNCTIONS OF LIVER

DRUG METABOLISM

The liver plays an important role in providing mechanisms for ridding the body of foreign molecules (xenobiotics) that are absorbed from the environment. In most cases, a drug is relatively lipophilic to ensure good absorption. The liver participates in the elimination of these lipid-soluble drugs by transforming them into more readily excreted hydrophilic products. There are two main reactions important for drug metabolism. Phase I reactions include oxidation, reduction, and hydrolysis of molecules.These result in metabolites that are more hydrophilic than the original chemicals. The cytochrome P450 system is a family of hemoproteins important for oxidative reactions involving drugs and toxic substances. Phase II reactions, also known as *conjugation reactions,* are synthetic reactions that involve addition of subgroups to the drug molecule. These subgroups include glucuronate, acetate, glutathione, glycine, sulfate, and methyl groups. These drug reactions occur mainly in the smooth endoplasmic reticulum of hepatocytes.

Many factors can affect drug metabolism in the liver. When the rate of metabolism of a drug is increased (i.e., enzyme induction),the duration of the drug action will decrease. However, when the metabolism of a drug is decreased (i.e., enzyme inhibition),then the drug will circulate for a longer period of time. It is important to note that some drugs may be converted to active products by metabolism in the liver. An example is acetaminophen when taken in larger doses.

Normally, acetaminophen is conjugated by the liver to harmless glucuronide and sulfate metabolites that are water soluble and eliminated in the urine. During an overdose, the normal metabolic pathways are overwhelmed, and some of the drug is converted to a reactive and toxic intermediate by the cytochrome P450 system. Glutathione normally reacts with this intermediate, leading to the production and subsequent excretion of a harmless product. However, as glutathione stores are diminished, the reactive intermediate cannot be detoxified and it combines with lipid membranes of hepatocytes, which results in cellular necrosis. Thus, treatment of acetaminophen overdoses consists of replenishing glutathione stores by supplementing with sulfhydryl compounds such as acetylcysteine.

BILE METABOLISM

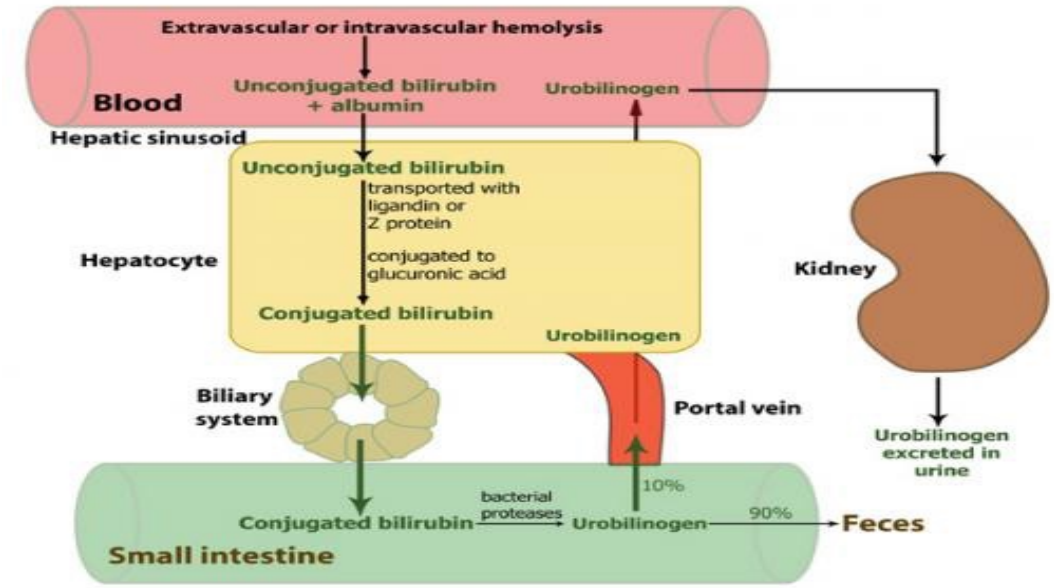


FIG 7. BILE METABOLISM

COAGULATION

The liver is responsible for synthesizing almost all the identified coagulation factors, as well as many of the fibrinolytic system components and several plasma regulatory proteins of coagulation and fibrinolysis. It is also critical for the absorption of vitamin K, synthesizes the vitamin K–dependent coagulation factors, and contains the enzyme that activates these factors. Also, the reticuloendothelial system of the liver clears activated clotting factors, activated complexes of the coagulation and fibrinolytic systems, and the end products of fibrin degradation.

Diseases of the liver are often associated with thrombocytopenia,qualitative platelet abnormalities, vitamin K deficiency with impaired modulation of vitamin K–dependent coagulation factors, and disseminated intravascular coagulation (DIC).

Warfarin, one of the most commonly dispensed anticoagulants,acts in the liver by blocking vitamin K–dependent activation of factors II, VII, IX, and X. Factor VII has the shortest half-life of the coagulation factors and its deficiency is manifested clinically as abnormalities of the measured prothrombin time (PT) or international normalized ratio (INR). Patients with hepatic synthetic dysfunction similarly have an abnormal PT.

LIVER FUNCTION TESTS

Standard liver functions tests (LFTs) are generally not tests of function and are not always specific to hepatic pathology but are valuable as a general screening tool that can yield clues about the cause of the disease. Total bilirubin(direct bilirubin and indirect bilirubin) levels can be affected by a number of processes related to bilirubin metabolism.

Unconjugated hyperbilirubinemia can be a reflection of increased bilirubin production (e.g., hemolysis), drug effects,inherited enzymatic disorders, or physiologic jaundice of the newborn.

Conjugated hyperbilirubinemia is generally a result of cholestasis or mechanical biliary obstruction, but can also be seen in some inherited disorders or hepatocellular disease.

The transaminases, alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are the most common serum markers of hepatocellular necrosis. AST is found in other organs such as the heart, muscle, and kidney, but ALT is liver specific.

Alkaline phosphatase (ALP) is expressed in liver, bile ducts, bone, intestine,placenta, kidney, and leukocytes. Elevations of ALP levels in hepatobiliary diseases are secondary to cholestasis or biliary obstruction. Such

elevations are caused by increased production of this enzyme.The ALP level can also be increased in malignant disease of the liver.

Gamma-glutamyl transpeptidase (GGT) is an enzyme in many organs in addition to the liver, such as the kidneys, seminal vesicles, spleen, pancreas, heart, and brain. Its level can be elevated in diseases affecting any of these tissues. It is also induced by alcohol intake and is elevated in biliary obstruction.

5′-Nucleotidase is also found in a wide variety of organs in addition to the liver, but increased levels are fairly specific to hepatic pathology. Like GGT, it can be helpful in determining whether an elevated ALP level is secondary to hepatic pathology.

Albumin is synthesized exclusively in the liver and can be used as a general measure of hepatic synthetic function. Because of the remarkable protein synthetic capacity of the liver, hypoalbuminemia is a marker of severe liver disease.

Clotting factors are largely synthesized in the liver; abnormalities of coagulation can be a marker of hepatic synthetic dysfunction. Measurement of specific clotting factors, such as factors V and VII, have been used to evaluate hepatic function in the transplantation population. PT or INR is the best test to measure the effects of hepatic disease on clotting and is usually a marker of

advanced chronic liver disease. Hepatic pathology can also affect clotting through intravascular coagulation and vitamin K malabsorption.

SPECIFIC SCORING TESTS

Hepatitis serologies - viral hepatitis.

Autoimmune antibodies - primary biliary cirrhosis (e.g.,antimitochondrial), primary sclerosing cholangitis (e.g., antineutrophil),and autoimmune hepatitis. α1-Antitrypsin and ceruloplasmin - α1-antitrypsin deficiency and Wilson's disease, respectively.

Tumor markers such as AFP and carcinoembryonic antigen (CEA) can be helpful in the diagnosis and management of primary and metastatic tumors of the liver.

Aminopyrine breath test is based on CYP clearance of radiolabeled aminopyrine. A breath test measuring radiolabeled CO2 as a breakdown product of aminopyrine is performed after administration at a specified time. The results largely depend on the functional hepatic mass, which is generally not depleted until end-stage liver disease has developed.

Scoring systems based on clinical observation and standard blood tests have been proposed. The most commonly used system is Pugh's modification of the Child score.

Child-Turcotte-Pugh Classification for Severity of Cirrhosis			
Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time Seconds prolonged International normalized ratio	<4 <1.7	4-6 1.7-2.3	>6 >2.3
*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points) Class A = 5 to 6 points (least severe liver disease) Class B = 7 to 9 points (moderately severe liver disease) Class C = 10 to 15 points (most severe liver disease)			

2.3 PATHOLOGY OF LIVER ABSCESS

There are three major forms of liver abscess, classified by etiology:

- Pyogenic liver abscess, which is most often polymicrobial,
- Amoebic liver abscess due to *Entamoeba histolytica*
- Fungal abscess, most often due to *Candida* species

Demographics

Age:

- A slight peak in incidence is seen in neonates, when liver abscess may be associated with umbilical vein catheterization and sepsis
- A gradual increase is seen beyond age 45 years, due to the average age of patients with biliary disease
- Liver abscess in children and adolescents suggests underlying immunocompromise or trauma

Gender:

- Pyogenic liver abscess shows no gender difference
- Amebic abscess is 10 times more common in men than in women

Race:

- No racial differences other than those related to the geographic distribution of populations with endemic amebiasis

Geography:

- Incidence of amebic abscess is higher in areas of endemic amebiasis, such as Mexico, India, East and South Africa, and parts of Central and South America
- Pyogenic liver abscess shows no geographic influence

Socioeconomic status:

- Malnutrition, immunocompromise, and excess alcohol intake (which is believed to impair immunologic response) predispose to amebic liver abscess in those exposed to *Entamoeba histolytica*

The liver usually clears off the bacterial load well to which it is exposed. When it exceeds its capacity, it results in the formation of liver abscesses. This results in invasion of the tissues followed by neutrophil infiltration, resulting in the formation of an organized abscess. Infections from the biliary tree and the cryptogenic infections cause hepatic abscess most commonly. This process is known as *ascending suppurative cholangitis*.

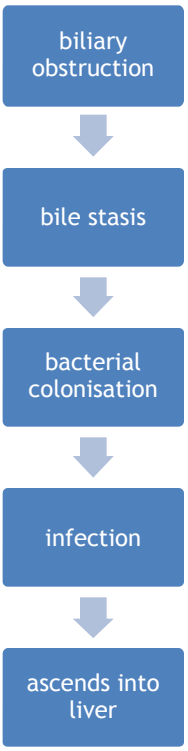


FIG 8. ROUTES OF INFECTION

Causes of biliary obstruction include:

- Stones most common
- Malignancy
- Caroli's disease
- Biliary ascariasis
- Biliary tract surgery
- Biliary enteric anastomosis

The common link between all causes is biliary obstruction and bacteria in the biliary tract. Any infection of the gastrointestinal tract ascends and cause portal vein infection (pyelophlebitis). The most common causes are

- ➢ Diverticulitis
- ➢ appendicitis
- ➢ Pancreatitis
- ➢ Inflammatory bowel disease
- ➢ pelvic inflammatory disease
- ➢ Perforated viscus
- ➢ and omphalitis in the newborn.

Systemic infections like endocarditis, pneumonia,osteomyelitis result in bacteremia and through the hepatic artery cause infection of the liver.

Hepatic abscess from systemic infections may also reflect an altered immune response, such as in patients with malignancy,AIDS, or disorders of granulocyte function. Children with chronic granulomatous disease (CGD) are particularly

susceptible. Some of the causes which result in abscess formation by direct extension include subphrenic abscess, suppurative cholecystitis perinephric abscess, perforation of the bowel.

Penetrating and blunt trauma can cause abscess formation by resulting in intrahepatic hematoma or necrosis. Bacteria may have been introduced from the trauma or the affected area may be seeded from systemic bacteremia. Hepatic abscesses associated with trauma can present in a delayed fashion up to several weeks after injury.

Other mechanisms of iatrogenic hepatic necrosis such as hepatic artery embolization or, more recently, thermal ablative procedures can be complicated by abscess. This is an uncommon complication of these procedures but is seen more often when there has been a previous biliary-enteric anastomosis.

Cryptogenic abscess,where no cause for a hepatic abscess is found predominate in many series and are more common in some case reports. Possible explanations for cryptogenic hepatic abscess are undiagnosed abdominal pathology, resolved infectious process at the time of presentation, or host factors such as diabetes or malignancy rendering the liver more susceptible to transient hepatic artery or portal vein bacteremia.

Contributory or predisposing factors

- Inflammatory bowel disease, particularly Crohns disease, due to loss of integrity of the mucosal barrier
- Liver cirrhosis
- Liver transplant
- Hepatic artery embolization
- Immunocompromise
- Older age (particularly associated with biliary sepsis)
- Malnutrition, malignancy, pregnancy, steroid use, and excessive alcohol intake predispose to liver abscess formation

MICROBIOLOGY

Liver abscess most commonly involve the right hemiliver(75%).

The left liver is involved in approximately 20% of the cases

The caudate lobe is rarely involved (5%).

Bilobar involvement with multiple abscesses is uncommon.

Approximately 50% of hepatic abscesses are solitary.

Gram positive S.aureus/epidermis Enterococcus Streptococci
Anaerobic Bacteroides Microaerophillic streptococci Fusobacterium
Miscellaneous Actinomyces C.albicans

2.4 CLINICAL FEATURES :

The classic features of hepatic abscess is fever, jaundice, and right upper quadrant pain, with tenderness to palpation but it is not present in most of the cases. Fever, chills, and abdominal pain are the most common presenting symptoms but a broad spectrum of nonspecific symptoms can be present.Symptoms such as malaise or vomiting, were constitutional in nature. cough or dyspnea may be the result of involvement of the diaphragm. Rarely, patients can present with peritonitis secondary to rupture. Cases of rupture into the pleural space or pericardium are distinctly uncommon. The duration of presenting

symptoms is variable, ranging from an acute illness to a chronic presentation lasting months. Endopthalmitis is a rare complication of liver abscess caused by klebsiella.

2.5 INVESTIGATIONS

BLOOD INVESTIGATIONS are non specific for diagnosing liver abscess.Some of the abnormalities found are:

Leukocytosis in 70% to 90% of patients

Anemia

Abnormalities of LFT

The ALP level is mildly elevated in 80% of patients.

Total bilirubin is elevated 20% to 50% of the time.

Transaminases are mildly elevated in 60% of patients.

Hypoalbuminemia or elevations of the PT/INR reflects chronicity.

IMAGING

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

X-RAYS

Subdiaphragmatic pathology, such as an elevated right hemidiaphragm, right pleural effusion, or atelectasis. Occasionally,these can be left-sided findings in the case of an abscess involving the left liver. They can show air-fluid levels or portal venous gas

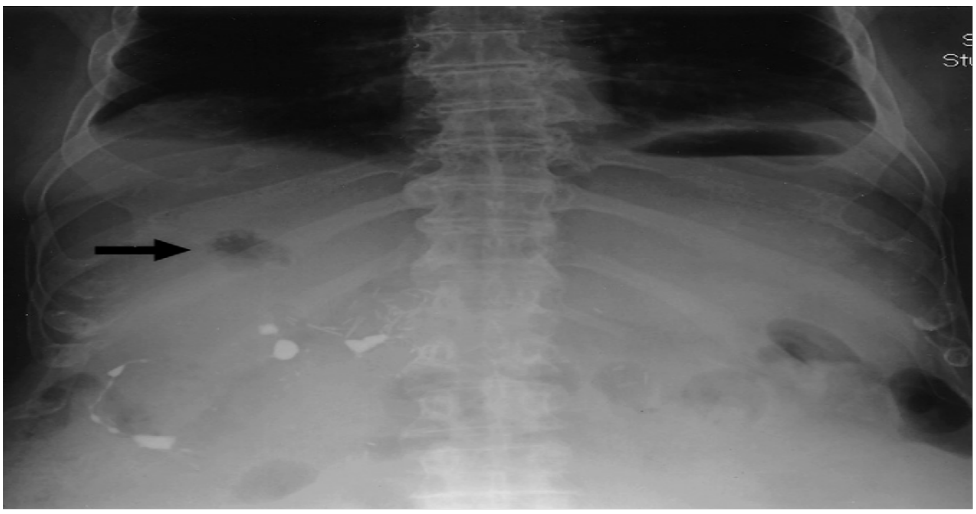


FIG 9. X RAY IN LIVER ABSCESS

Ultrasound

Demonstrates a round or oval area that is less echogenic than the surrounding liver. Ultrasound can distinguish solid from cystic lesions.

The sensitivity of ultrasound in diagnosing hepatic abscess is 80% to 95%.

LIMITATIONS

The limitations of ultrasound are its inability to visualize lesions high up in the dome of the liver

User dependent modality.

CT

CT is the investigation of choice for diagnosing liver abscess

CT scans can demonstrate very small abscesses and can more easily identify multiple small abscesses.

The abscess wall usually has an intense enhancement on contrast-enhanced CT. The sensitivity of CT in diagnosing hepatic abscess is 95% to 100%.

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

MRI

Magnetic resonance imaging (MRI) can be helpful in distinguishing the cause of many hepatic masses and evaluating the biliary tree for pathology

Pyogenic abscesses have variable signal intensity on T1 andT2 weighted images, depending on protein content, but characteristically there is high signal intensity on T2 weighted images, with rim enhancement after injection of gadolinium,plus perilesional edema.

It does not appear to have any distinct advantage over CT in diagnosing hepatic abscess.

2.6 TREATMENT

Once the diagnosis of a single or multiple liver abscess is made, broad-spectrum parenteral antibiotics should be started.

Routine hematologic blood work, liver function tests, indirect hemagglutination tests to rule out an amebic abscess, and blood cultures before onset of antibiotic therapy should be performed.

Various treatment modalities available for pyogenic liver abscesses

- Parenteral broad-spectrum antibiotic therapy alone
- Percutaneous needle aspiration and antibiotic therapy: Single or repeated
- Percutaneous catheter drainage and antibiotic therapy
- Laparoscopic drainage with antibiotic therapy

- Laparotomy with intraoperative drainage and antibiotic therapy

Antibiotic therapy :

INDICATIONS

- Multiple small abscesses
- low risk often abscess rupture
- lack of toxemia (i.e., no hemodynamic instability, patient does not feel acutely ill)

clinical response is gauged by defervescence, fall in leukocytosis, and resolution of symptoms, and should be reassessed frequently by imaging with ultrasonography or CT for resolution of abscess(es).

Lack of improvement after a reasonable course (10 to 14days) indicates failure of treatment. Oral antibiotics should be continued for at least 4 weeks after discontinuance of parenteral antibiotics. Worsening of fever, leukocytosis, and symptoms at any time also indicates failure of treatment and immediately qualifies the patient for a more aggressive treatment regimen involving a drainage procedure.

PER CUTANEOUS ASPIRATION

The first-line treatment for most patients with a pyogenic liver abscess should be percutaneous aspiration and antibiotic therapy. Aspiration involves complete drainage of the abscess cavity with no catheter left within the cavity. The patient's symptoms normally improve immediately after aspiration. Aspirated fluid is sent for aerobic and anaerobic cultures. Clinical response is again measured by a fall in fever and leukocytosis, and symptomatic improvement.

➢ Aspiration may have to be repeated when follow-up imaging is performed.

➢ After aspiration, abscess cavities are lavaged with saline, and an intracavitary antibiotic injection (gentamicin or metronidazole)

The clinical importance of placing antibiotics within the abscess cavity requires further prospective evaluation.

PERCUTANEOUS CATHETER DRAINAGE

Percutaneous catheter drainage with ultrasonography or CT guidance is indicated for patients for whom aspiration fails and for whom percutaneous drainage is not contraindicated.

CONTRAINDICATIONS

- Coagulopathy
- the lack of a safe or appropriate access route
- multiple macroscopic abscesses.

The visualization of septae within the abscess cavity on CT or ultrasonography is not a contraindication to catheter drainage, as these rarely represent separate localized abscesses.

METHOD:Modified Seldinger technique

The catheter is placed into the abscess cavity and left to straight drain in a position as dependent as possible to facilitate drainage. Depending on the viscosity of the aspirate the catheter is flushed one to three times daily with 25 mL of sterile saline solution. The patient is again monitored for clinical improvement and cessation of drainage from the abscess (the catheter is slowly removed as the cavity shrinks). In 10% to 15% of cases, percutaneous drainage fails and intraoperative drainage is required.

OPERATIVE DRAINAGE

Operative drainage of hepatic abscesses is indicated for the following patients:

(a) patients who require laparotomy for the underlying problem

(b) those in whom percutaneous catheter drainage fails

(c) patients with contraindications to percutaneous drainage

(d) Patients whose liver abscesses rupture into the peritoneum

A midline or subcostal incision is performed. Intraoperative ultrasonography can be useful to help determine the ideal site for abscess drainage as well as identify portal structures and hepatic veins. Needle aspiration is used to localize the abscess precisely and can be used to identify the portal structures and hepatic veins. The hepatotomy is then performed with electrocautery to open the abscess cavity. Drains (preferably closed suction) should be placed into the abscess cavity or cavities and exited via a separate abdominal stab wound.

Laparoscopic approaches are reported to be successful. If the drainage does not contain bile, the drains can be removed reasonably quickly.

Chapter 3

MATERIALS AND METHODS

MATERIALS AND METHODS

3.1 Type of study : Prospective and Retrospective Observational Study

3.2 Study approval : Prior to commencement of this study - Thesis & Ethical Committee of Madras Medical College and Rajiv Gandhi Government General Hospital, chennai had approved the thesis protocol.

3.3 Place of study : Rajiv Gandhi Government General Hospital

3.4 Period of study : Duration starting from August 2014 to July 2015

3.5 Sample size : 50 cases

3.6 Selection of patients:

a) **Sampling method-** Purposive**.**

b) **Inclusion criteria-** Patients who are suspected to have liver abscess based on the clinical,radiological features

c) **Exclusion criteria - -** Patients having other liver disease such as malignancies,CLD.

3.7 Study procedure:

Method of sampling was non-random, purposive. After admission short history was taken and physical examination was conducted on each patient admitted in surgery department with features of liver abscess. Baseline investigations, as routinely required, were done followed by imaging studies. Patients were then explained about their diseases process and the possible line of management. All the necessary information regarding the study was explained to the patients or their valid guardian. Informed written consent was taken from the patients or their guardian willing to participate in the study. Detailed history was taken from the study group to establish proper diagnosis. Thorough physical examination was done in each case. Data collection sheets were filled in by the investigator himself. All of the preoperative factors related to the patient were noted down in the data sheet. After proper evaluation and preparation, patients who required surgical management were taken up for surgery. Strict aseptic precautions were followed during the operation. Meticulous technique were practiced asa far as possible. The operation procedure and related peroperative factors were observed directly and recorded in the data collection sheet instantly. After completing the collection of data it was complied in a systematic way.

3.8 Variables studied:

i. Age

ii. Sex

iii. Co-morbidities: COPD, jaundice, diabetes, obesity and malnutrition

iv. Chest Xray.

v. USG Abdomen & Pelvis

vi. CECT Abdomen & Pelvis

vii. Blood parameters

viii. Type of management

ix. Post operative complications

x. Pus C/S

3.9 Ethical consideration

All the patients/ legal guardians were given an explanation of the study and about the investigative and operative procedures with their merits and demerits, expected results, and possible complications. If he/she agreed then the case had been selected for this study. The study did not involve any additional investigation or any significant risk. It did not cause economic burden to the patients. The study was approved by the institutional review board prior to commencement of data

collection. Informed consent was taken from each patient/guardian. Data were collected by approved data collection form.

3.10 Data collection

Data were collected by pre-tested structured questionnaire. Data were collected from all the respondents by direct interview after getting informed written consent from them or from their legal guardian.

3.11 Data analysis

Data analysis was done both manually and by using computer. Calculated data were arranged in systemic manner, presented in various table and figures and statistical analysis was made to evaluate the objectives of this study with the help of Statistical Package for Social Science (SPSS).

CHAPTER 4

RESULTS

RESULTS

This descriptive and observational study was carried out to determine the clinicopathology of liver abscess and the various modalities of treatment and their efficacy. Fifty patients fulfilling the inclusion criteria from Surgery department of Madras Medical College and Rajiv Gandhi Government General Hospital during the period of August 2014 to July 2015 were selected. All cases were evaluated clinically. Only essential investigations necessary for diagnosis and preoperative assessment were carried out before operations. All patients underwent surgery or pigtail drainage as warranted in their case. The patients of both sexes and different ages were included in the study. The results obtained are as follows.

Table 1 : Age and Sex Distribution of patients

Age / Sex	Male	Female	Total
18 - 29	0	2	2 (4)
30 - 39	9	1	10 (20)
40 - 49	13	0	13 (26)
50 - 59	12	2	14 (28)
> 60	9	2	11 (22)
Total	43 (86)	7 (14)	50 (100)

* Figures in parentheses indicates percentages

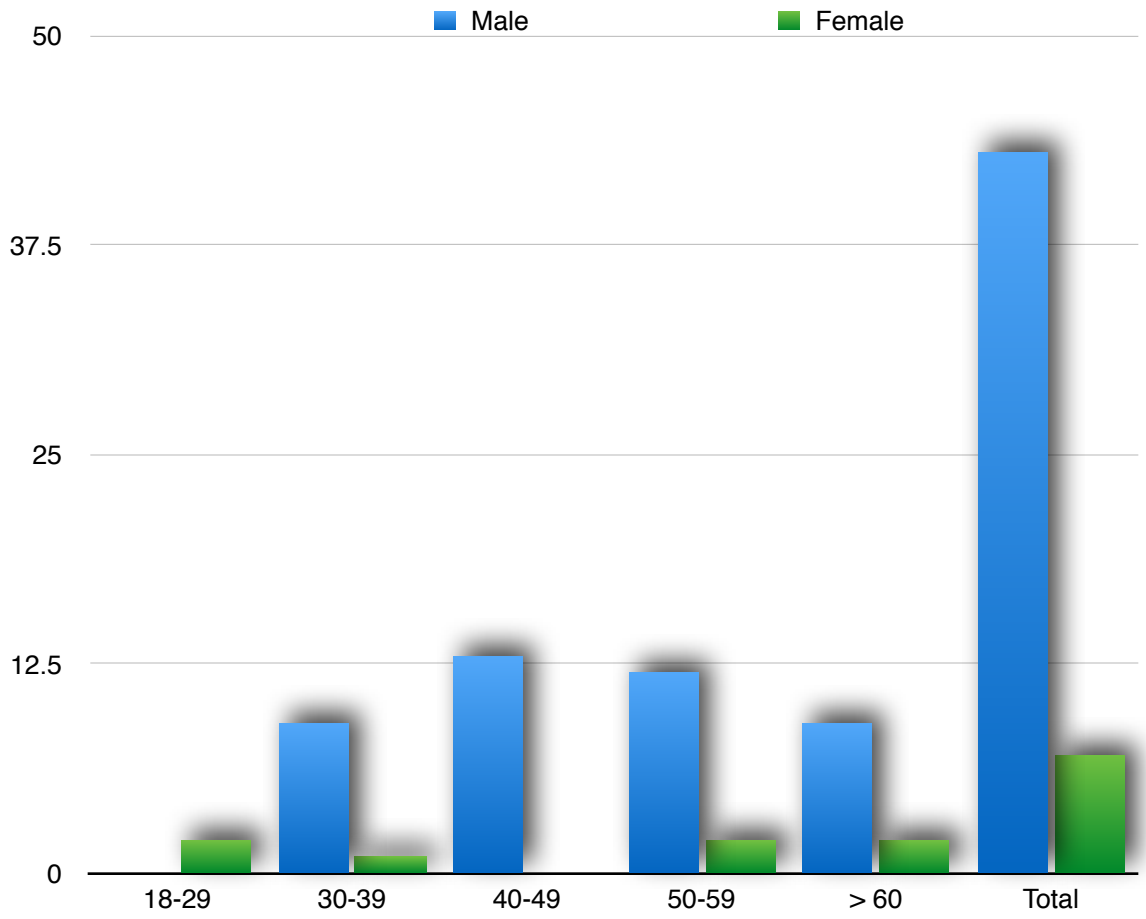


Table 2 : Prevalence of Risk Factors in patient group

Risk Factor	Number	Percentage
Diabetes Mellitus	5	10
Hypertension	4	8
DM & HTN	7	14
Alcoholic	16	32
No Comorbidity	18	36
Total	50	100

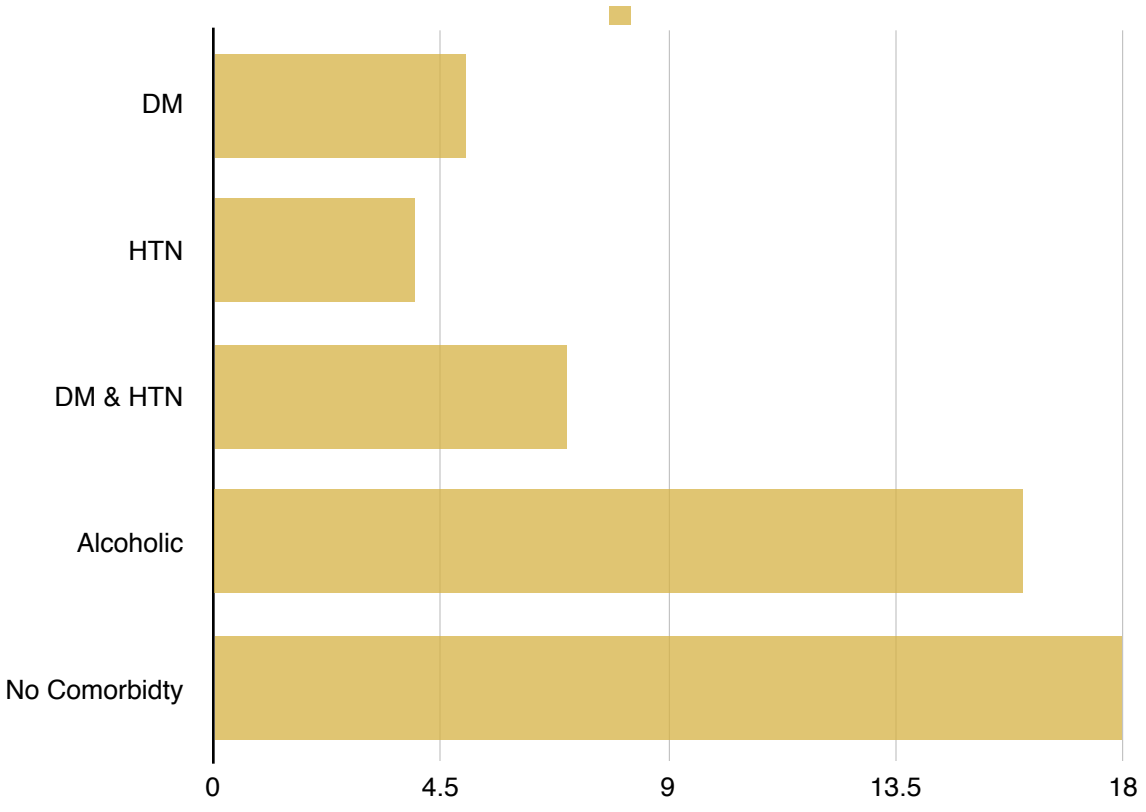


Table 3 : Distribution of Symptoms among the patient group

	Numbers	Percentage
Fever	34	68
Rt. Hypochondrial Pain	31	62
Jaundice	6	12
Vomiting	18	36
Dyspnea	5	10

* - - Patients will have overlapping symptoms so percentages don't add up to 100%

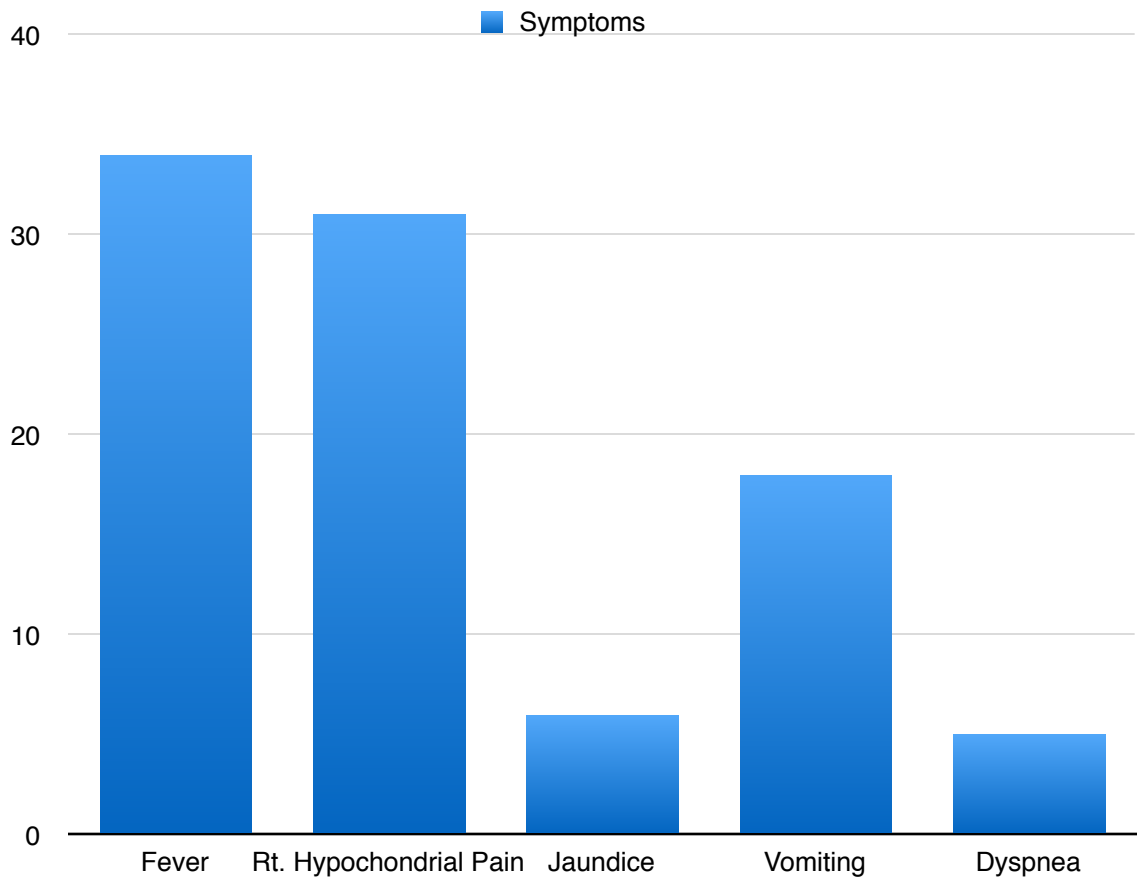


Table 4 : Analysis of Vital Parameters in patients with liver abscess

Parameters	Within Range		Outside Range	
	Number	Percentage	Number	Percentage
SBP	42	84	8	16
PR	28	56	22	44
Hb	42	84	8	16
TC	33	66	17	34
Urea/Creatinine	39	78	11	22
LFT	39	78	11	22
Albumin	44	88	6	12

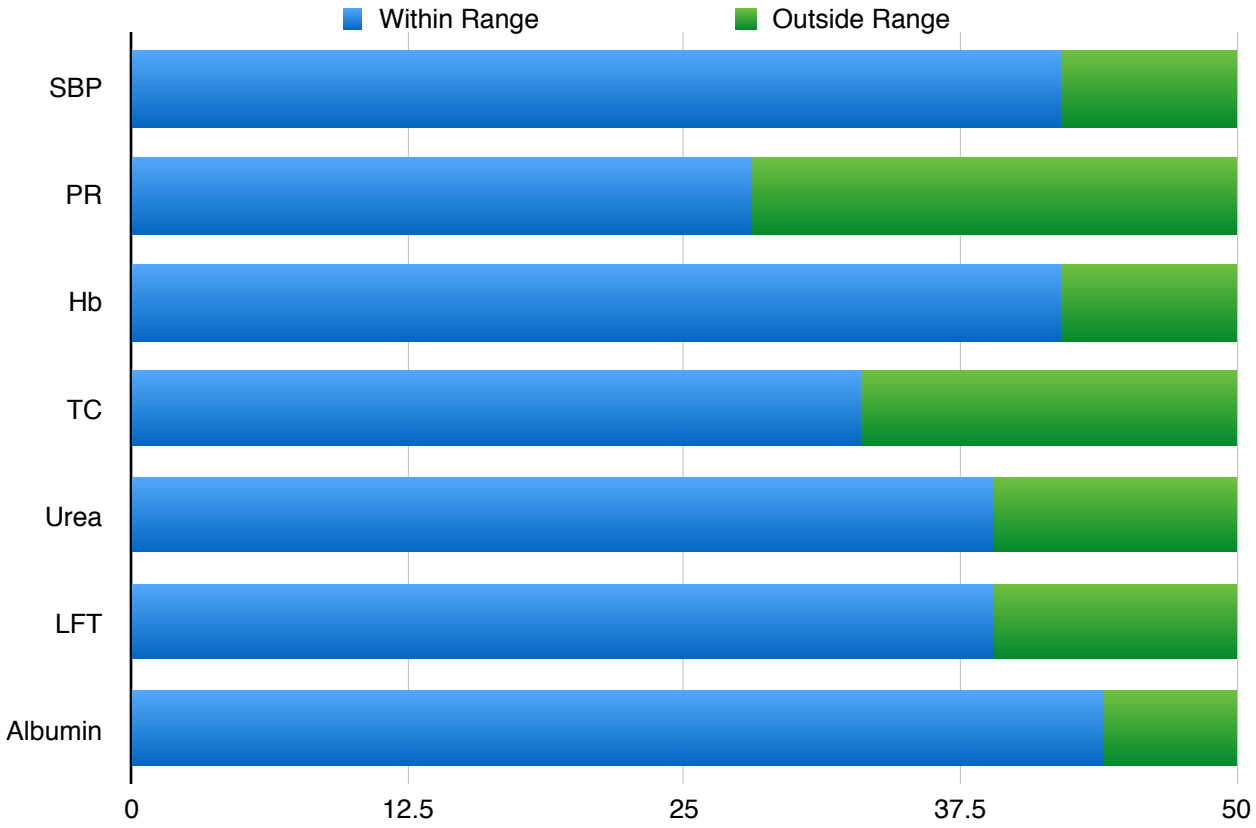


Table 5 : Analysis of Pus C/S among the patients group

Organism	Numbers	Percentage
Entamoeba Histolytica	22	44
Escherichia Coli	4	8
Klebsiella	3	6
Staph. aureus	5	10
Polymicrobial	13	26
No growth	3	6
Total	**50**	**100**

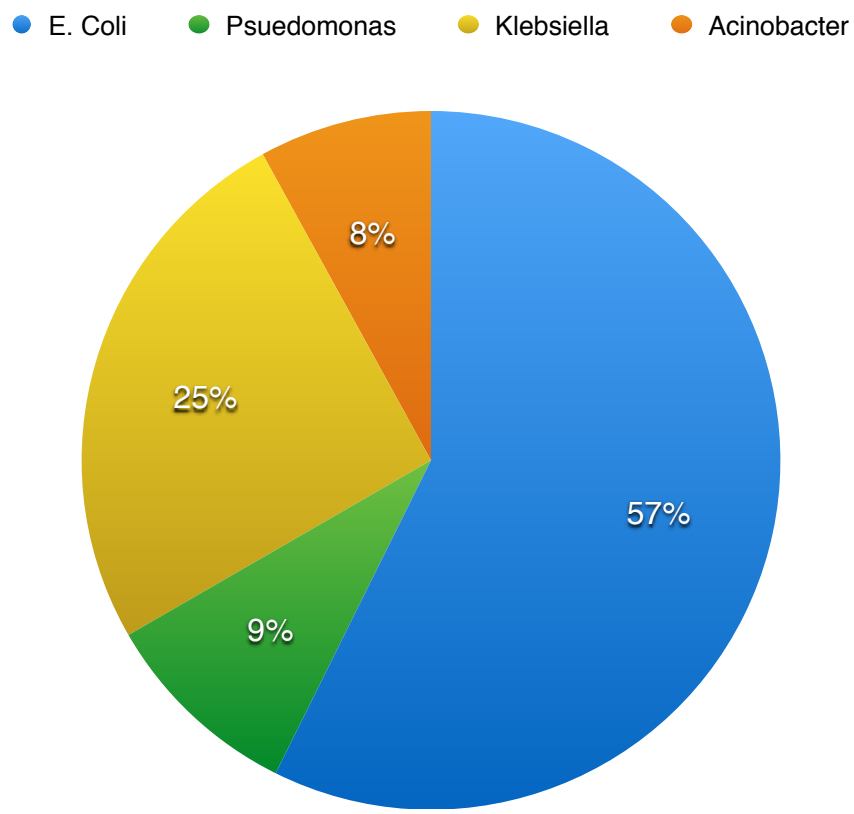


Table 6 : Analysis of various investigations underwent by patients in the study

Investigations		Numbers	Percentage
Size	< 5 cm	27	54
	> 5cm	23	46
Number	Single	44	88
	Multiple	6	12
Location	Right	37	74
	Left	7	14
	Caudate/Quadrate	6	12

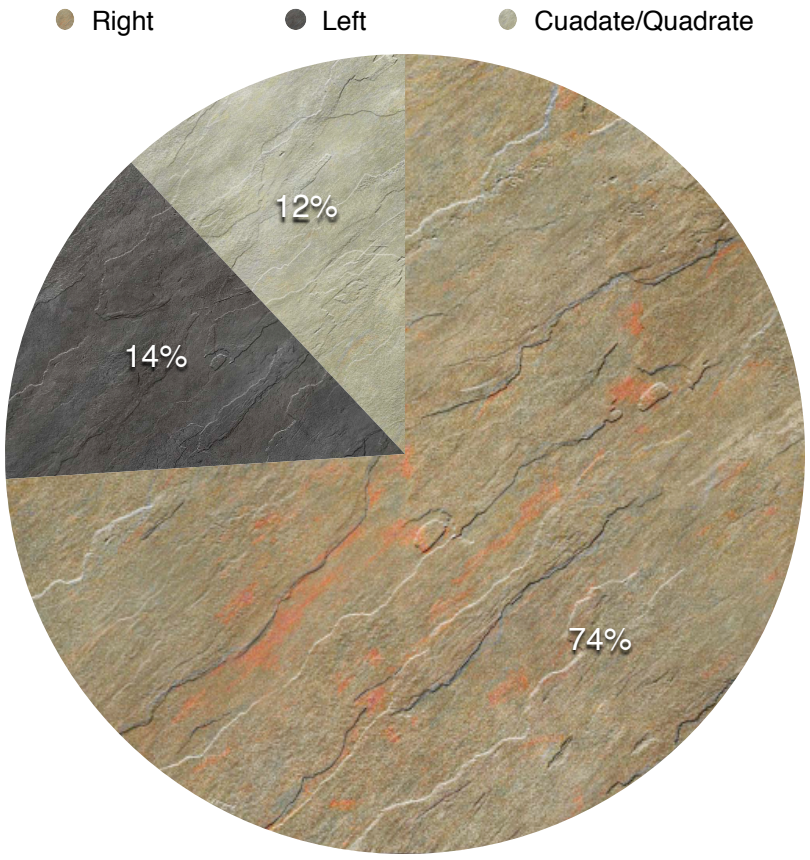


Table 7 : Distribution of procedure underwent among patient groups

Operative Procedure	Numbers	Percentage
Laparotomy & Drainage	19	38
Pigtail Drainage	29	58
Conservative Management	2	4
Total	**50**	**100**

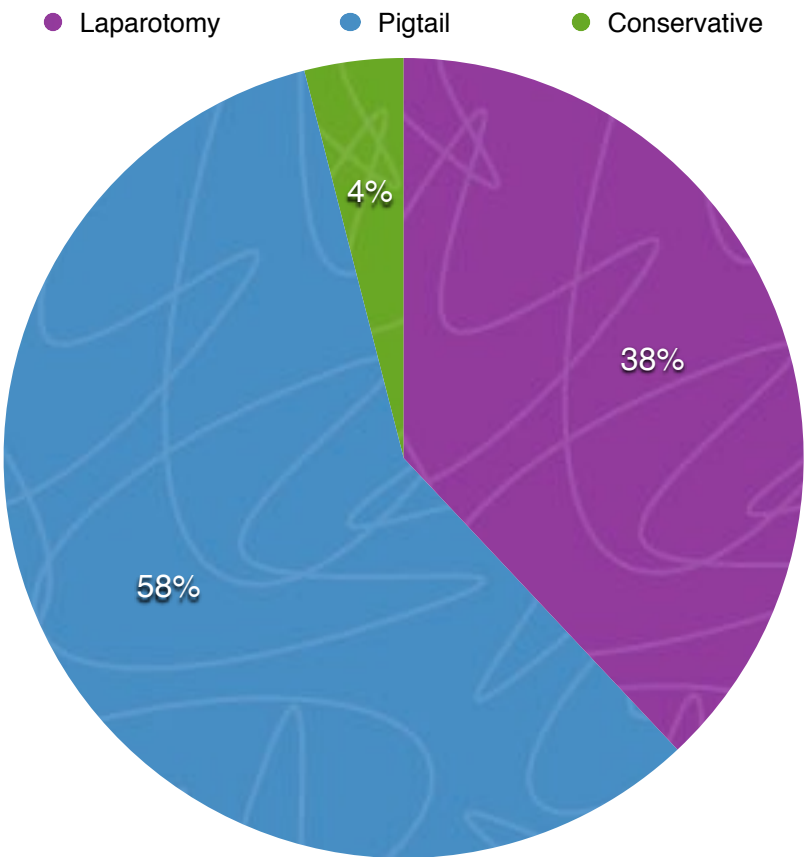


Table 8 : Distribution of procedure underwent with respect to size of abscess

Procedure		Numbers	Percentage
Laparotomy	> 5 cm	15	30
	< 5 cm	8	16
Pigtail	> 5 cm	8	16
	< 5 cm	21	42
Conservative	< 5 cm	2	4
	> 5 cm	0	0
Total		**50**	**100**

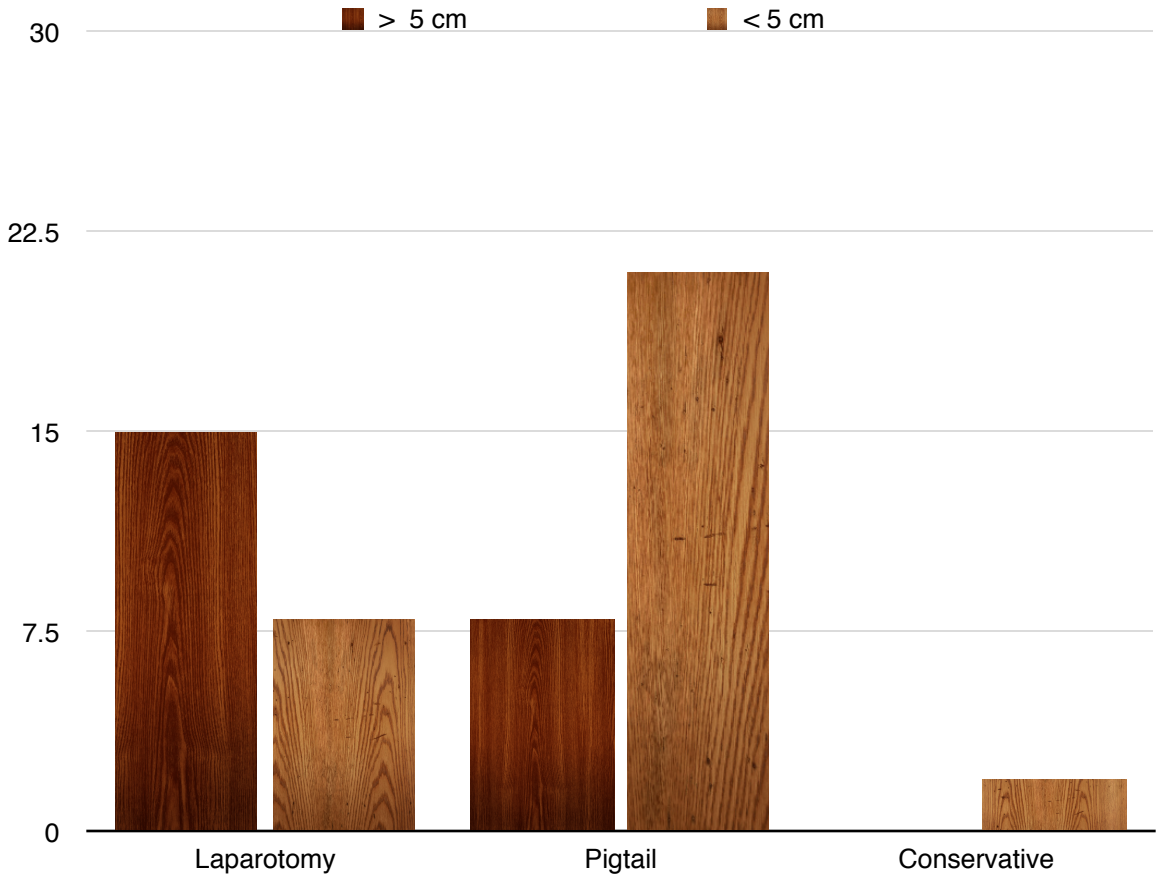
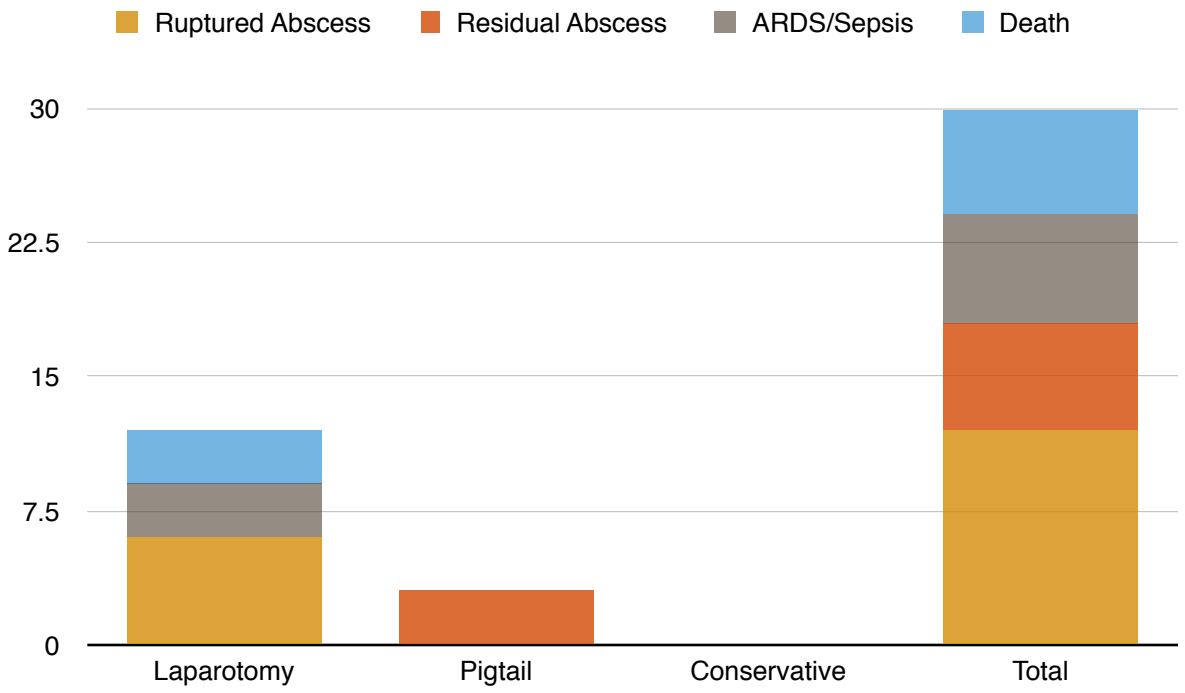


Table 9 : Prevalence of Complications Among Patient Group

Complications	Numbers	Percentage
Ruptured Abscess	6	12
Residual Abscess	3	6
ARDS/Sepsis	3	6
Death	3	6

Table 10 : Analysis of complications with respect to procedure

Complications	Laparotomy	Pigtail	Conservative
Ruptured Abscess	6	0	0
Residual Abscess	0	3	0
ARDS/Sepsis	3	0	0
Death	3	0	0



CHAPTER 5

DISCUSSION

DISCUSSION OF RESULTS

This descriptive and observational study was carried out to determine the clinicopathology of liver abscess, including the various modalities of treatment and its efficacy. Fifty patients fulfilling the inclusion criteria from Surgery department of Madras Medical College and Rajiv Gandhi Government General Hospital during the period of August 2014 to July 2015 were selected.

Age of 50 patients ranged from 18-75 years. The patients were predominantly of the more than 40 age group. There was not much involvement in extremes of age group. Around forty three patients (86%) were males, the high amount of prevalence of liver abscess among males can be explained by increased risk factors like alcohol consumption.

On analysing the risk factors, as expected, chronic alcoholism was the predominant risk factor, seen in 16 patients (32%), with diabetes seen in five patients and systemic hypertension seen in four patients. Both Diabetes Mellitus and systemic hypertension was seen in seven patients (14%). Thirty six percent of the patients had no identifiable risk factor.

On evaluation of the presenting symptoms, thirty four patients (68%) had fever usually associated with chills and rigors. Thirty one patients (62%) had right hypochondrial pain and tenderness. Nearly all the patients had either of these symptoms. None of them presented in exclusion of either of these symptoms. Eighteen patients (36%) had complaints of vomiting and anorexia. Six patients complained of jaundice and another five patients complained of breathlessness.

Analysis of vital parameters showed that twenty two patients (44%), had tachycardia at presentation while eight patients came in state of hypotensive shock. Eight patients (16%) had anaemia while seventeen patients (34%) had elevated total count. Pre renal failure indicated by elevated serum urea and creatinine was seen in eleven patients (22%). Low albumin levels was seen in six patients.

An analysis of the pus culture and sensitivity from the discharge, showed the presence of Entamoeba histolytic in twenty two patients (44%). Bacterial infection was seen in the remaining forty six patients with common organisms being E.Coli, Klebsiella, Staphylococcus aureus, with thirteen patients (26%) having polymicrobial infection.

Proceeding to investigations, imaging was done by means of either ultra sonogram or contrast enhanced computed tomography of the abdomen. Forty four

patients (88%) had only a single abscess cavity while the remaining six patients (12%) had multiple abscesses. Twenty seven patients (54%) had abscess cavity of size less than 5 cm while twenty three patients (46%) had maximum abscess cavity size more than five cm. Right lobe comprising segments 5, 6, 7 & 8 was the predominantly involved lobe seen in thirty seven patients (74%) while left lobe comprising segments 1, 2 & 3 was seen in seven patients while caudate lobe was involved in six patients (12%).

Regarding the procedures performed, nineteen patients (38%) underwent laparotomy and drainage, while the majority of the patients, around twenty nine (58%) underwent ultra sonogram guided pig tail drainage. Conservative management was tried in two patients (4%). On comparing the procedure underwent with the size of abscess the patient had it was found that, fifteen of twenty three patients with abscess size greater than 5 cm underwent laparotomy and drainage with pig tail drainage done for eight patients and conservative management attempted in none of these patients. A vast majority of patients, more than eighty percent with size less than five cm, were managed with pig tail drainage, while open drainage was done in eight patients and two patients were attempted conservative management.

Six patients had ruptured liver abscess with laparotomy and peritoneal lavage being done for all these six patients. All six of these patients developing complications, with three patients going into sepsis while three patients expired due to multi organ dysfunction syndrome. Residual abscess was seen in three patients who had underwent pigtail drainage. Both the conservatively managed patients recovered uneventfully.

LIMITATIONS OF THE STUDY

As this study has been carried out over a limited period of time with a limited number of patients and there was lack of financial and infrastructural support, it could not have been large enough to be of reasonable precision. The follow up period was not long enough to comment about long term morbidity and mortality. More number of patients with liver abscess need to be analysed to determine the pathophysiology of the disease. The newer modalities of treatment should also be included in future studies. All the facts and figures mentioned here may considerably vary from those of large series covering wide range of time, but still then, as the cases of this study were collected from a tertiary level hospital in our country, this study has some credentials in reflecting the facts regarding prevalence of liver abscess and the efficacy of various treatment modalities.

SUMMARY

Liver abscess is common surgical problem encountered in our clinical practice. It is a source of great discomfort patient and causes prolonged morbidity in those patients who are not properly managed or who developed complications. It poses a great dilemma as the management protocols are not very well defined. There is an increasing number if newer treatment modalities available. But their efficacy and clinical indications are not very well established. Moreover there is no clear cut data on the prognostic factors and the clinicoepediomology of the disease. This study tries to throw a light on few of those factors

Age and Sex Distribution :

Males of the of more than forty age group was the most affected population. The prevalence of alcoholism among males can be mainly attributed to the predominant involvement of them among liver abscess patients. Since they constitute the primary work force in any society, any morbidity of the disease constitutes a great strain on the economy of that family.

Risk Factors :

Chronic Alcohol intake is a definitive risk factor for development of liver abscess. Diabetes Mellitus is the prevalent comorbid factor, seen especially in the elderly. Other comorbid factors include hypertension, bronchial asthma etc. None of these seemed to have a significant correlation with the disease process.

Diagnostic Studies :

All patients underwent routine blood investigations. Ultra sonogram of the abdomen is sufficient in patients with small, single abscess lying in the superficial segments. CECT abdomen has better sensitivity and specificity in identifying multiple abscesses or deep lying abscess and also for making out any complications like rupture or biliary tree obstruction

Causative Organism :

In this study there was near equal prevalence of amoebic and bacterial liver abscess with not much difference in the prognosis, line of management or the incidence of complications.

Management :

Pig tail drainage is preferred in patients with single abscess of size less than 5 cm situated in superficial segments. Laparotomy and drainage was done in patients with ruptured or impeding rupture abscess. Conservative management with antibiotics was also useful in very small single cavity abscess

Morbidity & Mortality

The incidence of complications was predominantly seen in patients with ruptured liver abscess who underwent laparotomy, in form of sepsis followed by MODS and death. Residual abscess cavity was seen in small number of patients following pig tail drainage but it was not significant.

RECOMMENDATIONS

On the basis of the findings of the study, the following recommendations can be made:

1. Prompt administration of empiric broad spectrum parenteral antibiotics.

2. Ultrasound and/or CT scan to confirm diagnosis, with simultaneous radiologically guided aspiration of all abscesses >3 cm, +/- drain placement.

3. Microbiological analysis of abscess aspirates and blood cultures: antibiotic regimen should be adjusted according to culture results and sensitivities.

4. Early recognition of septicaemia or organ failure and appropriate transfer to critical care unit. Con- sider repeat imaging to confirm correct drain placement and to determine response to treatment and final resolution of the abscess.

5. Surgical intervention should be considered for patients with large, complex, septated or multiple abscesses, underlying disease or in whom percutaneous drainage has failed.

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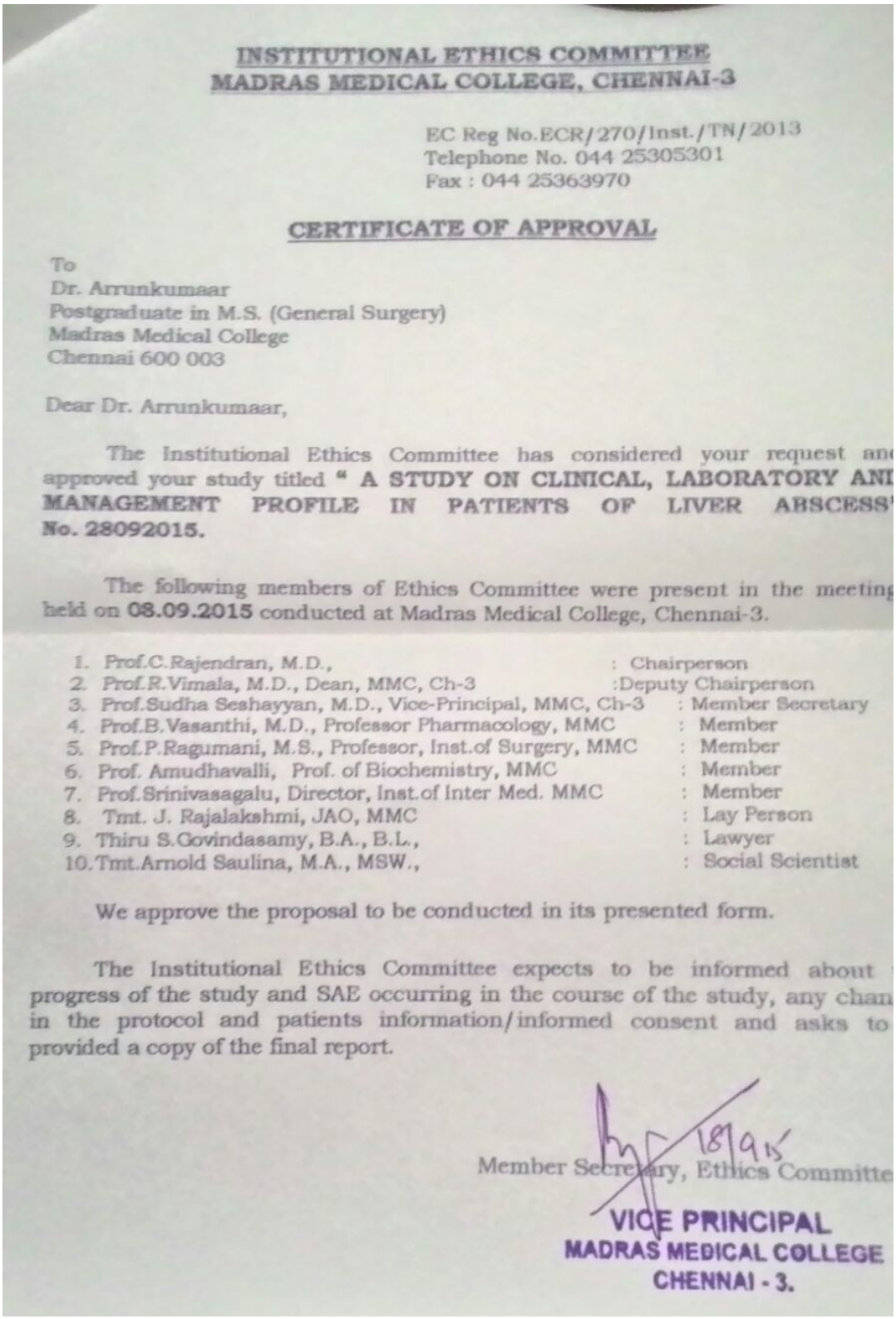
APPENDIX - I : ETHICAL COMMITTEE CLEAREANCE



INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Arrunkumaar
Postgraduate in M.S. (General Surgery)
Madras Medical College
Chennai 600 003

Dear Dr. Arrunkumaar,

The Institutional Ethics Committee has considered your request and approved your study titled **" A STUDY ON CLINICAL, LABORATORY AND MANAGEMENT PROFILE IN PATIENTS OF LIVER ABSCESS"** **No. 28092015.**

The following members of Ethics Committee were present in the meeting held on **08.09.2015** conducted at Madras Medical College, Chennai-3.

1.	Prof.C.Rajendran, M.D.,	: Chairperson
2.	Prof.R.Vimala, M.D., Dean, MMC, Ch-3	:Deputy Chairperson
3.	Prof.Sudha Seshayyan, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.B.Vasanthi, M.D., Professor Pharmacology, MMC	: Member
5.	Prof.P.Ragumani, M.S., Professor, Inst.of Surgery, MMC	: Member
6.	Prof. Amudhavalli, Prof. of Biochemistry, MMC	: Member
7.	Prof.Srinivasagalu, Director, Inst.of Inter Med. MMC	: Member
8.	Tmt. J. Rajalakshmi, JAO, MMC	: Lay Person
9.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
10.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about progress of the study and SAE occurring in the course of the study, any chan in the protocol and patients information/informed consent and asks to provided a copy of the final report.

18/9/15

Member Secretary, Ethics Committe

VICE PRINCIPAL
MADRAS MEDICAL COLLEGE
CHENNAI - 3.

Appendix-II

PROFORMA

Name: **Age:** **Sex:**

IP No. :

Chief Complaints :

Presenting Complaints :

Co Morbid Illness :

Past Surgical / Medical History :

CLINICAL EXAMINATION:

General Condition :

VITAL SIGNS

Pulse: **BP:** **Temp:**

SYSTEMIC EXAMINATION

CVS :

RS :

ABDOMEN :

PR:

CLINICAL DIAGNOSIS

INVESTIGATIONS :

Hemogram: **RFT/ LFT:**

Ultrasound:

CXR: **Xray Abdomen :**

CECT abdomen;

Blood C/s :

INTERVENTION DETAILS

CONDITION ON DISCHARGE

FOLLOW UP:

INFORMATION SHEET

TITLE ::"A STUDY ON CLINICAL,LABARATORY AND MANAGEMENT PROFILE IN PATIENTS OF LIVER ABSCESS"

Name of Investigator : Dr.A.ARRUN KUMAAR Name of Participant :

Purpose of Research : The purpose of the study is to identify clinical,labaratory and management profile in patients of liver abscess patients

Study Design : Observational study (Prospective and Retrospective)

Study Procedures : Patient will be subjected to routine investigations, Xray, Usg, CECT Abdomen, Operative Procedure as indicated and the data analysed

Possible Risks : No risks to the patient

Possible benefits

To patient : A better understanding of their problem so as to devise a plan of management which suits their needs.

To doctor & to other people : The study can throw a light on the interventional procedures, how to handle the post operative complications and the best time for surgerical intervention. This will help in providing better and complete treatment to other patients in future.

Confidentiality of the information obtained from you : The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or

presentation resulting from the research, no personally identifiable information will be shared

Can you decide to stop participating in the study : Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you : Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail	:	**"A STUDY ON CLINICAL,LABARATORY AND MANAGEMENT PROFILE IN PATIENTS OF LIVER ABSCESS"**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
In Patient Number	:	

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment ❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

Dr.A.ARRUN KUMAAR

Appendix – III

Statistical formula

A. Sample size:

To determine the sample size, this formula was used; $n = \frac{z^2 pq}{d^2}$

Where,

n = the desired sample size,

z = the standard normal deviate, usually set at 1.96 at 5% level,

which corresponds to 95% confidence level,

p = proportion of population, q

= 1- p

d = the degree of accuracy level considered as 5.0 %,

which assumes 0.05

If population size, N < 10,000 than the required sample size is very much smaller which was calculated by the following formula –

$$n_f = \frac{n}{1 + \frac{n}{N}}$$

Where,

n_f = the desired sample size, when population size, N < 10,000

n = the desired sample size, when population size, N > 10,000 N

= the roughly estimated population size.

B. Arrithmatic mean, $X = \frac{\sum fx}{N}$ (for grouped data)

C. Standard deviation , $SD = \sqrt{\frac{\sum (X - \bar{X})^2}{}}$

('O' indicates observed value and 'E' indicates expected value)

D. $$Z = \frac{P_1 - P_2}{\sqrt{\left[\frac{P_1 Q_1}{N_1} + \frac{P_2 Q_2}{N_2}\right]}}$$

P_1 indicates proportion in first group

P_2 indicates proportion in second group

$Q_1 = 100 - P_1$

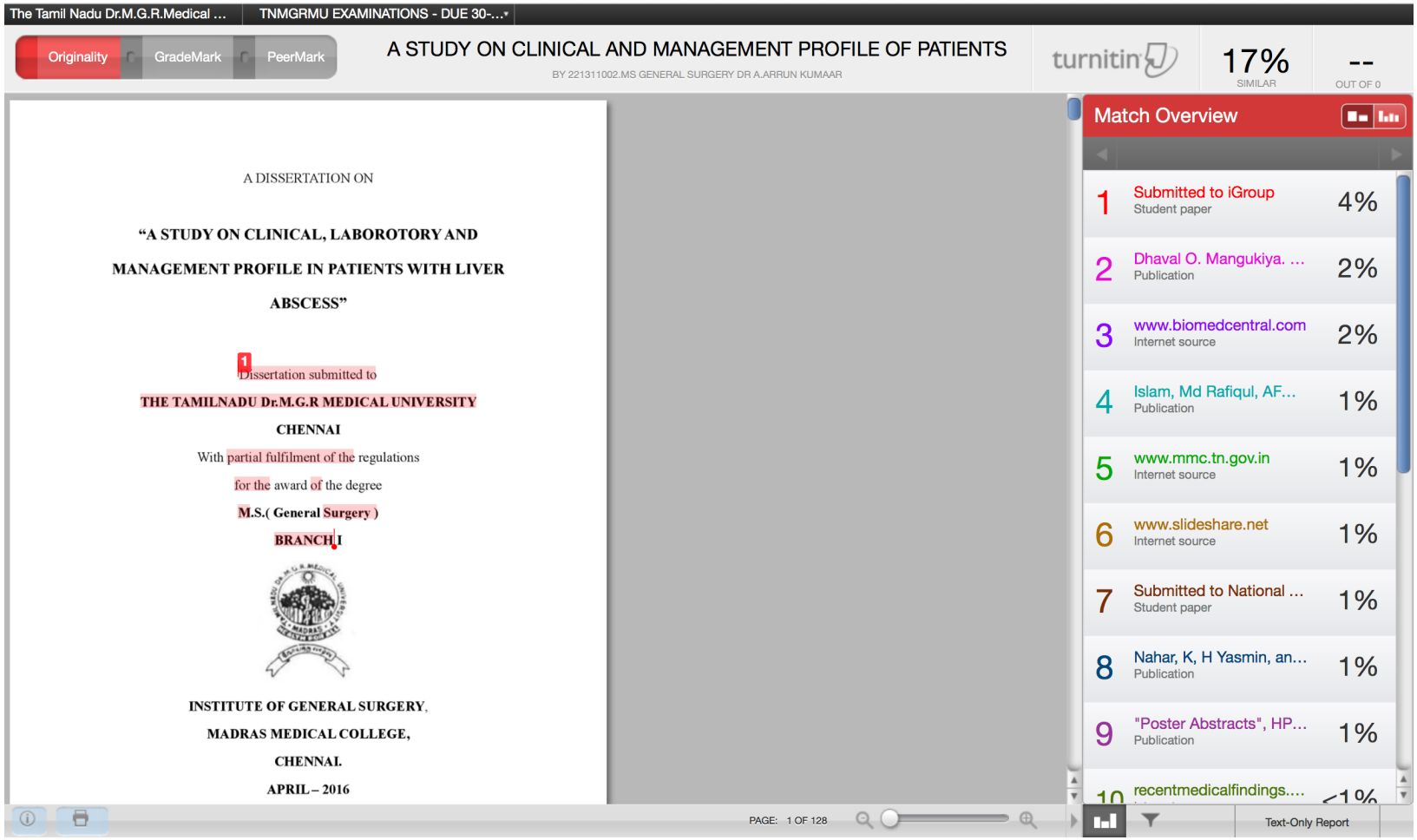
$Q_2 = 100 - P_2$

N_1 indicates sample size of first group

N_2 indicates sample size of second group.

E. $$SD = \sqrt{\frac{\sum (X - \bar{X})}{(N-1)}}$$

Here, $\bar{X}$ indicates mean value
X indicates individual value
N indicates sample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A DISSERTATION ON
"A STUDY ON CLINICAL, LABOROTORY AND MANAGEMENT PROFILE IN PATIENTS WITH LIVER ABSCESS"
Dissertation submitted to
THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY
CHENNAI
With partial fulfilment of the regulations
for the award of the degree
M.S.(General Surgery)
BRANCH I
INSTITUTE OF GENERAL SURGERY,
MADRAS MEDICAL COLLEGE,
CHENNAI.
APRIL – 2016
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Text-Only Report

APPENDIX V — MASTER CHART

S.No	Name	Age	Sex	Ip. No.	Presenting Complaints	Risk Factors	PR	BP	Investigations					Procedure Done	Complications	Pus C/S report
									Imagine	Hb	RFT	LFT	Album			
1	kesavan	60	M	95822	fever, Rt upper abd pain	Alcoholic	98	110/70	USG abd-4*5cm hypoechoeic lesion in segment 5 of liver	10.5	WNL	WNL	3.3	pigtail catheterisation and drainage		Entamoeba histolytica
2	kamala	57	F	95946	Rt upper abd pain	Alcoholic	86	120/80	USG abd-5*5cm hypoechoeic lesion in segment 8 of liver	9	WNL	WNL	3.6	pigtail catheterisation and drainage		Entamoeba histolytica
3	etiyappan	55	M	96520	fever, Rt upper abd pain		87	100/60	USG abd-2*2cm hypoechoeic lesion in segment 6 of liver	11	WNL	WNL	3.2	conservative management with IV antibiotics		
4	duraisamy	46	M	102482	fever for 2 weeks		90	130/70	CECT abd-5*6cm hypoechoeic lesion in segment 5 of liver	11.3	WNL	WNL	4	pigtail catheterisation and drainage		Escherica coli
5	gomathi	28	F	105639	fever with jaundice	Alcoholic	97	120/60	USG abd-4*4cm hypoechoeic lesion in segment 3 of liver	10	WNL	TB-2.3	2.7	pigtail catheterisation and drainage		Klebsiella, proteus
6	veeramuthu	64	M	106365	fever with chills and vomiting	DM,SHT	76	150/90	USG abd-3*3cm hypoechoeic lesion in segment 6 of liver	10.2	WNL	WNL	3.4	pigtail catheterisation and drainage		Staphylococcus aureus
7	manikam	70	M	106046	fever with chills and diarrhoea		89	130/70	CECT abd-4*5cm hypoechoeic lesion in segment 8 of liver	11.6	WNL	WNL	3.5	pigtail catheterisation and drainage		Entamoeba histolytica
8	muthukaruppan	33	M	105554	Rt upper abd pain	DM	80	110/80	CECT abd-6*7cm hypoechoeic lesion in segment 7 of liver	13	WNL	WNL	3.4	pigtail catheterisation and drainage	repeated pigtail catheterisatoin	Escherica coli,Enterococci
9	jayaraman	50	M	107627	fever with vomiting, malaise	Alcoholic	80	120/70	USG abd-multiple hypoechoeic lesions in segment 5,6 of liver largest of size7*4cm in seg 7	12.5	WNL	WNL	3.2	pigtail catheterisation and drainage		Entamoeba histolytica
10	antony raj	32	M	107498	fever, Rt upper abd pain and jaundice	Alcoholic	89	110/70	USG abd-6*6cm hypoechoeic lesion in segment 5 of liver	13.1	WNL	TB-1.7	3	pigtail catheterisation and drainage		Klebsiella,Enterococci
11	velu	45	M	107916	fever for 10 days		94	100/60	USG abd-5*5cm hypoechoeic lesion in segment 5 of liver	12.2	WNL	WNL	3.3	pigtail catheterisation and drainage		Streptococcus viridans
12	murugesan	40	M	114240	Rt upper abd pain, breathlessness	Alcoholic	102	110/70	CECT abd-8*5cm hypoechoeic lesion in segment 8 of liver	12.4	U-58, C-1.9	WNL	3.1	laparotomy and drainage		Entamoeba histolytica
13	shankaran	37	M	111199	fever, jaundice, vomiting	Alcoholic	107	116/84	USG abd-4*5cm,6*3cm hypoechoeic lesion in segment 4,5 of liver	9.4	U-32,C-2	TB-1.9	2.8	laparotomy and drainage	Ruptured abscess	Entamoeba histolytica
14	ganapathy	58	M	112172	fever, jaundice,difuse abd pain	DM, SHT	96	140/90	CECT abd-7*8cm hypoechoeic lesion in segment 7 of liver	9	U-40,C-1.9	TB-1.7	2.6	laparotomy and drainage	Ruptured abscess	Escherica coli,proteus
15	ravi	45	M	108316	fever with Rt upper abd pain	Alcoholic	84	100/70	USG abd-6*3cm hypoechoeic lesion in segment 6 of liver	11.2	WNL	WNL	3.2	pigtail catheterisation and drainage		Klebsiella
16	sudha	30	F	118263	fever, vomiting		87	140/80	CECT abd-4*4cm hypoechoeic lesion in segment 8 of liver	9.9	WNL	WNL	3.5	pigtail catheterisation and drainage		Entamoeba histolytica
17	kannIyappan	75	M	120805	fever for 1 month		90	110/70	CECT abd-7*5cm hypoechoeic lesion in segment 7 of liver	10	WNL	WNL	3.4	pigtail catheterisation and drainage		Escherica coli,Bacteroids
18	ramesh	38	M	121510	Rt upper abd pain		73	120/80	CECT abd-3*4m hypoechoeic lesion in segment 2 of liver	13.1	WNL	WNL	3.3	pigtail catheterisation and drainage		Staphylococcus aureus
19	thangaraj	51	M	123615	fever with Rt upper abd pain	Alcoholic,DM,	83	104/60	CECT abd-4*4cm hypoechoeic lesion in segment 8 of liver	12.5	WNL	WNL	3.1	pigtail catheterisation and drainage		Entamoeba histolytica
20	selvam	45	M	124218	fever with vomiting with chills	Alcoholic	78	110/80	CECT abd-3*3cm hypoechoeic lesion in segment 7 of liver	13	WNL	WNL	3	conservative management with IV antibiotics		
21	sekar	47	M	124812	fever with Rt upper abd pain	DM,SHT	85	150/100	CECT abd-5*5cm hypoechoeic lesion in segment 3 of liver	12.9	WNL	WNL	3.2	pigtail catheterisation and drainage		Staphylococcus aureus
22	chandra	30	F	3405	fever > 2weeks		88	110/80	CECT abd-4*6cm hypoechoeic lesion in segment 8 of liver	10.2	WNL	WNL	3	pigtail catheterisation and drainage		Entamoeba histolytica
23	asokan	48	M	4276	fever with Rt upper abd pain		79	111/60	CECT abd-7*9cm hypoechoeic lesion in segment 4 of liver	12	WNL	WNL	3	laparotomy and drainage		klebsiella, pseudomonas
24	kuppusamy	72	M	20176	fever with vomiting	Alcoholic	76	100/70	CECT abd-5*5cm hypoechoeic lesion in segment 7 of liver	10	WNL	WNL	3.3	pigtail catheterisation and drainage		Entamoeba histolytica
25	balaraman	69	M	29997	vomiting, Rt upper abd pain	DM	80	114/80	CECT abd-4*4cm hypoechoeic lesion in segment 6 of liver	11	WNL	WNL	3.2	pigtail catheterisation and drainage		Escherica coli

26	gajendran	40	M	22492	Rt upper abd pain		84	130/70	CECT abd-6*5cm hypoechoeic lesion in segment 5 of liver	13.2	WNL	WNL	3.2	pigtail catheterisation and drainage	repeated pigtail catheterisatoin	klebsiella,streptococcus viridans
27	raguraman	32	M	29815	fever, Rt upper abd pain,jaundice, vomiting		108	100/60	CECT abd-7*8cm hypoechoeic lesion in segment 3 of liver	13.5	WNL	TB-1.2	2.9	laparotomy and drainage		Entamoeba histolytica
28	rajendran	53	M	27811	fever, Rt upper abd pain		87	110/70	USG abd-3*5cm,3*4 hypoechoeic lesion in segment 6,7 of liver	11.9	WNL	WNL	3.4	pigtail catheterisation and drainage		Eschricia coli, Bacteroides
29	sampath	68	M	28312	fever with chills, malaise	DM	82	120/80	CECT abd-6*4cm hypoechoeic lesion in segment 4 of liver	11.7	WNL	WNL	3.2	pigtail catheterisation and drainage		klebsiella, proteus
30	gajendran	40	M	26817	fever, Rt upper abd pain		105	100/60	CECT abd-7*10cm hypoechoeic lesion in segment 5 of liver	12.8	WNL	WNL	3.1	laparotomy and drainage		Entamoeba histolytica
31	mani	58	M	33346	Rt upper abd pain	Alcoholic, DM	109	110/80	CECT abd-8*7cm hypoechoeic lesion in segment 7 of liver	11.8	U-35, C-1.2	WNL	2.9	laparotomy and drainage		Pseudomonas
32	vijay kumar	34	M	29117	fever with Rt upper abd pain		73	130/70	CECT abd-4*4cm hypoechoeic lesion in segment 2 of liver	13	WNL	WNL	3.3	pigtail catheterisation and drainage		Entamoeba histolytica
33	annamalai	58	M	38711	vomiting, Rt upper abd pain	SHT	100	140/90	USG abd-7*9cm hypoechoeic lesion in segment 6 of liver	11.9	WNL	WNL	3.2	laparotomy and drainage		klebsiella
34	shankar	55	M	40678	Rt upper abd pain	Alcoholic	90	112/76	CECT abd-4*3cm hypoechoeic lesion in segment 4 of liver	12	WNL	WNL	3.1	pigtail catheterisation and drainage		Entamoeba histolytica
35	arumugam	65	M	40879	Rt upper abd pain	DM, SHT	75	136/80	CECT abd-3*3cm hypoechoeic lesion in segment 8 of liver	11.8	WNL	WNL	3	pigtail catheterisation and drainage		Staphylococcus aureus
36	selvam	55	M	43362	fever,abd pain, vomiting	Alcoholic	106	96/60	CECT abd-multiple hypoechoeic lesion in segment 6,7 of liver	12	WNL	WNL	3.2	laparotomy and drainage	ARDS	Entamoeba histolytica
37	selvaraj	55	M	48816	fever, Rt upper abd pain	DM	101	100/60	CECT abd-7*7cm hypoechoeic lesion in segment 5 of liver	11.8	U-39, C-1.3	Tb-1	2.9	laparotomy and drainage		Eschericia coli
38	naresh babu	35	M	54240	fever with vomiting, diarrhoea, malaise		81	110/60	CECT abd-4*5cm hypoechoeic lesion in segment 7 of liver	13	WNL	WNL	3.1	pigtail catheterisation and drainage		Entamoeba histolytica
39	shabina	58	F	59582	fever, Rt upper abd pain,jaundice, vomiting	SHT,CKD	110	90/60	CT abd-8*10cm hypoechoeic lesion in segment 6 of liver	8.1	U-72, C-2.1	TB-1.6	2	laparotomy and drainage	Ruptured abscess-AKI/ARDS- EXPIRED	Entamoeba histolytica
40	saravanan	41	M	59316	fever, Rt upper abd pain	Alcoholic	77	120/80	CECT abd-multiple hypoechoeic lesion in segment 7,8 of liver largest of size 4*5cm	13.1	WNL	WNL	3.2	pigtail catheterisation and drainage	repeated pigtail catheterisatoin	Klebsiella, enterococcus
41	jegan	42	M	83761	fever, vomiting	SHT	109	140/80	CECT abd-8*6cm hypoechoeic lesion in segment 4 of liver	12.3	U-30, C-1.3	TB-1.2	2.8	laparotomy and drainage		Entamoeba histolytica
42	tharabuddhin	32	M	79689	Rt upper abd pain, breathlessness, vomiting		111	108/70	CECT abd-8*8cm hypoechoeic lesion in segment 8 of liver	13	WNL	WNL	3	laparotomy and drainage		Eschericia coli
43	sekar	50	M	80927	Rt upper abd pain, vomiting	DM, SHT	99	120/80	CECT abd-5*5cm hypoechoeic lesion in segment 7 of liver	9.8	U-34, C-1.2	TB-1	2.1	laparotomy and drainage	Ruptured abscess	Entamoeba histolytica
44	ranjith	31	M	87645	fever, Rt upper abd pain		75	100/60	CECT abd-3*5cm hypoechoeic lesion in segment 2 of liver	13	WNL	WNL	3	pigtail catheterisation and drainage		klebsiella, proteus
45	venkataraman	40	M	91559	Rt upper abd pain, breathlessness	Alcoholic	109	110/70	CECT abd-multiple hypoechoeic lesion in segment 3,4 of liver	12.9	WNL	WNL	3.1	laparotomy and drainage		Eschericia coli, pseudomonas
46	sathya	65	F	93203	fever with Rt upper abd pain, reduced urine output	DM, SHT	106	100/60	USG abd-7*6cm hypoechoeic lesion in segment 5 of liver	10.6	U-57, C-1.7	TB-1.9	2.2	laparotomy and drainage	Ruptured abscess-MODS- EXPIRED	Entamoeba histolytica
47	subramaniyan	45	M	90367	fever > 2weeks	SHT	96	150/90	CECT abd-4*4cm hypoechoeic lesion in segment 6 of liver	12.9	WNL	WNL	3.1	pigtail catheterisation and drainage		Klebsiella
48	govindarajan	65	M	101467	fever, vomiting, breathlessness	DM	110	110/80	CECT abd-7*7cm hypoechoeic lesion in segment 4 of liver	11	WNL	WNL	3	laparotomy and drainage		Staphylococcus aureus
49	indhirani	55	F	101507	Rt upper abd pain, vomiting		101	100/60	USG abd-7*6cm hypoechoeic lesion in segment 5 of liver	9.2	U-40, C-1.6	WNL	3	laparotomy and drainage		Entamoeba histolytica
50	Pushparani	65	F	121536	difuse abd pain, breathlessness, vomiting	DM, SHT	120	90/70	USG abd-8*9cm hypoechoeic lesion in segment 8 of liver	8	U-41, c-1.5	Tb-1	2.4	laparotomy and drainage	Ruptured abscess-SEPTICEMIA-EXPIRED	Entamoeba histolytica

KEY :

DM - - Diabetes Mellitus

SHT / HTN - - Systemic Hypertension

CAD - - Coronary Artery Disease

BA - - Bronchial Asthma

WNL - - Within Normal Limits

Kleb - -Klebsiella

Pseudo - - Psuedomonas

E.Coli - Escherichia Coli