PROSPECTIVE STUDY OF MANAGEMENT AND OUTCOMES OF LIVER TRAUMA IN BLUNT INJURY ABDOMEN CASES IN GRH, MADURAI

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

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This is to certify that the dissertation entitled "**PROSPECTIVE STUDY OF MANAGEMENT AND OUTCOMES OF LIVER TRAUMA IN BLUNT INJURY ABDOMEN CASES IN GRH, MADURAI"** is a bonafide and genuine research work carried out by **DR. P.PUGAZHENTHI** under the guidance of **DR.K.G.SUBANGI M.S.,D.G.O.**, Professor, Department of General Surgery Madurai Medical College, Madurai.

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DECLARATION BY THE CANDIDATE

I hereby declare that the dissertation entitled **"PROSPECTIVE STUDY OF MANAGEMENT AND OUTCOMES OF LIVER TRAUMA IN BLUNT INJURY ABDOMEN CASES IN GRH, MADURAI"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.K.G.SUBANGI, M.S.,D.G.O.,** Professor, Department of General Surgery, Madurai Medical College, Madurai. The Tamil Nadu Dr. M.G.R. Medical University, Chennai shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic/research purpose.

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INTRODUCTION

Modern day have seen a tremendous improvement in mans life style and comforts. But along with comforts, challenging threats to human health has also been emerging . The technological advancement in the industry of automobiles and the vast increase in the use of automobiles have made motor vehicle accidents are sixth among the leading causes of death . The rapid increase in the number of motor vehicles resulted in very rapid increase in number of victims to blunt abdominal trauma. Motor vehicle accidents accounts for about 75 to 80% of the blunt abdominal trauma. The indian mortality rates for trauma are 20 times more than that of western countries .

Most of the times blunt abdominal trauma is missed during early phase of management. Blunt injury of abdomen is also a result from fall from height, assault with blunt objects, sport injuries, bomb blast. Blunt abdominal trauma is generally not obvious. Hence, often missed, unless examined repeatedly.

Due to the delay in diagnosis and inadequate treatment of liver injuries, most of the cases are becoming fatal.

The knowledge in the management of blunt liver trauma has been increasing progressively. In spite of the best available treatment and advances in diagnostic and supportive care, the morbidity and mortality remains at large. The reasons for this due to the time interval between the trauma and admission of the victim, inadequate and lack of appropriate surgical treatment, delay in the diagnosis, post operative complications. Liver involved in abdominal injuries vast majority of times. In case of patients with hemodynamic stability conservative management is the management of choice.

With GRH,MADURAI being tertiary care centre for south tamilnadu, the availability of blunt injury cases in adequate numbers, this study would serve to evaluate the modes of clinical presentations of blunt liver injury and signifying the precise management of various grades of liver injuries with its post operative morbidity and mortality .

AIM OF THE STUDY

Prospectively study the outcomes of operative and conservative management in isolated liver trauma in blunt injury abdomen cases in GRH,Madurai

OBJECTIVES OF THE STUDY:

- To estimate the burden of isolated liver trauma in blunt injury abdomen cases in GRH,Madurai
- To grade the liver trauma in blunt injury abdomen cases.
- To manage the patients by conservative/operative management.
- To analyze the outcomes and complications associated with the same.

REVIEW OF LITERATURE

Selective history:

Blunt injury as cause of intra abdominal solid organ injuries have been recognized since historical times.

Aristotle was the first to demonstrate visceral injuries from blunt abdominal trauma.

Hippocrates and Galen are said to have given new description of this condition. By 1500 BC distinct triage and protocol for surgery had been developed in Babylonia under the rule of Hummurabi described by Edwin Smith Papyru.

Von Reckiling Huse noticed artery thrombosis occurring as a result of blunt trauma.

Solomen performed peritoneal lavage for the first time in 1906.

Aenhium used puncture of abdominal wall as a diagnostic procedure in abdominal injuries in 1934.

Branch reported 2 cases of liver laceration treated by resection of left lobe of liver in 1938.

The development of emergency medical service is an important milestone in the history of surgical practice of trauma. Greeks required physicians to be there during the battle and Romans constructed the hospitals close to the battlefield.

In 1865 Cincinnati General Hospital first instituted the ambulance system.

In 1965 Root described the flushing of sterile solution through the peritoneal cavity to lavage the peritoneal contents.

Advanced imaging techniques like CT scan and MRI make easier for early detection of blunt liver injuries

ANATOMY :

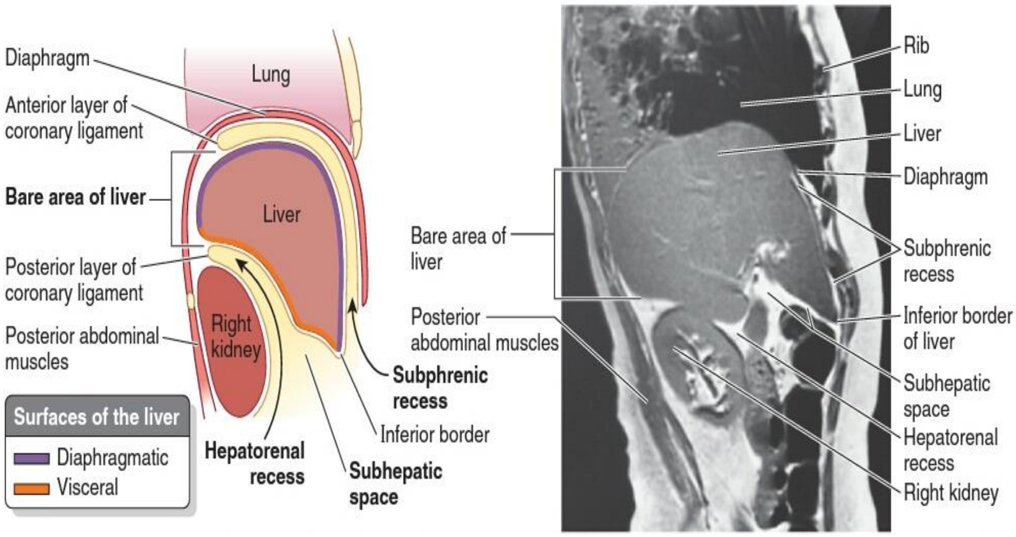
The liver is the largest gland in the body , the largest single organ. It weighs about 1500 g and accounts for 2.5% of adult body weight. In a mature fetus,when it act as a hematopoietic organ. Except for initially it meant to be the liver by the portal venous system. In addition to its metabolic activities, the liver had a function of storing glycogen and secretes bile, yellow-brown or green in colour aids in the emulsification of fat. Bile passes from the liver via the biliary duct right and left hepatic duct,that unite to form the common hepatic duct, which joins with the cystic duct to form the common bile duct.

The liver produces bile continuously; however, between meals it accumulates and gallbladder stored it, which also concentrates the bile by water and salts absorption. During arrival of food in the duodenum, the gallbladder sends concentrated bile through the biliary ducts to the duodenum.

The diaphragmatic surface of the liver is covered with visceral peritoneum, in the posterior aspect , the bare area of the liver , where it lies in direct contact with the diaphragm.

The bare area is demarcated by the reflection of peritoneum from the diaphragm to it as the anterior and posterior layers of the coronary ligament .

These layers joins at the right to form the right triangular ligament and diverge toward the left to enclose the bare area . The anterior layer of the coronary ligament is continuous as falciform ligament on the left side, and the posterior layer is continuous with lesser omentum.

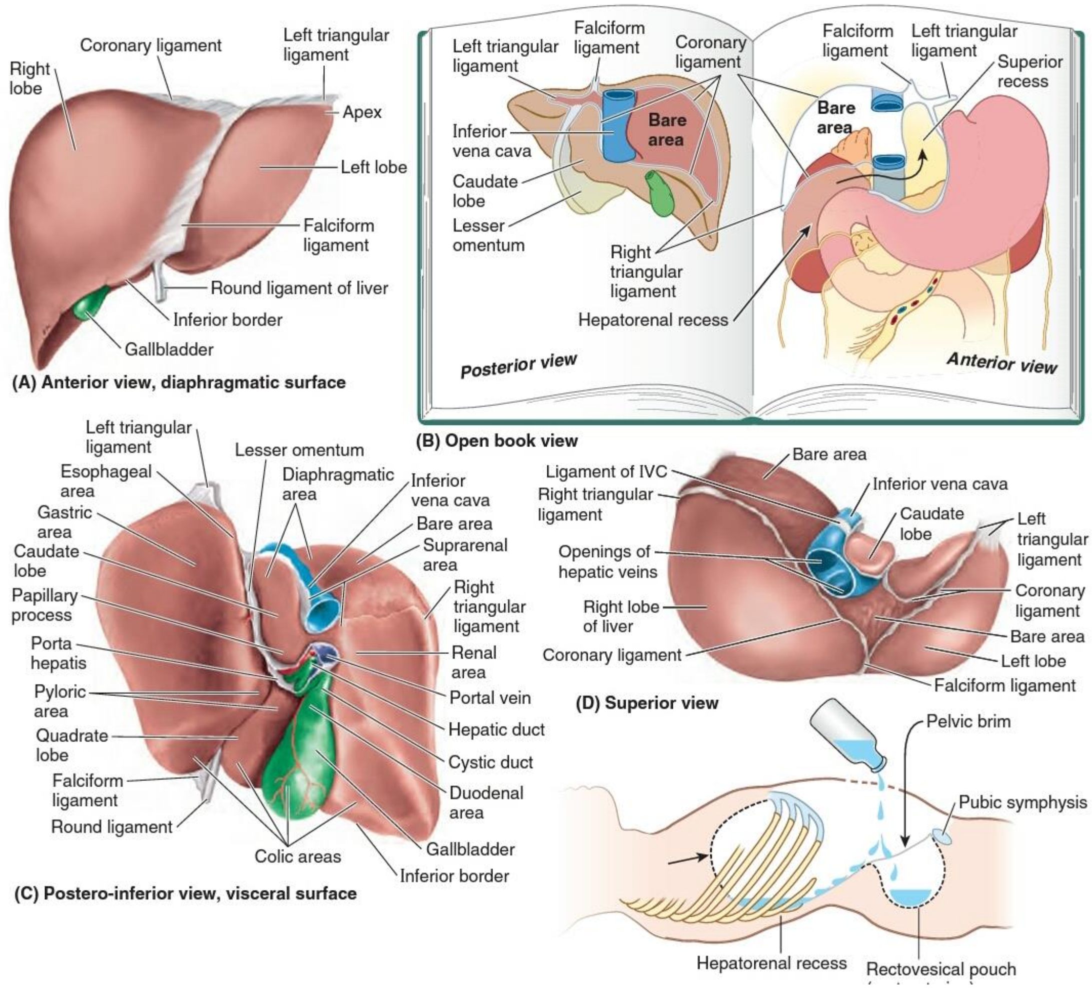


The IVC traverses at groove for the it within the bare area of the liver. The visceral surface of the liver is also covered with visceral peritoneum , except in the porta hepatis and fossa for gallbladder, a transverse fissure through which hepatic artery ,potal vein,bile duct hepatic nerve plexus, and hepatic ducts that supply and drain the liver enter and leave it.

In contrast to the smooth diaphragmatic surface, the visceral surface shows multiple fissures and impressions from contact with other organs.

sagittally oriented fissures,joined centrally by the transverse porta hepatis, form the H shape on the visceral surface.

The right sagittal fissure is the continuous groove formed by fossa for the gallbladder anteriorly and the groove for the vena cava posteriorly. The umbilical fissure is the continuous groove formed by the fissure for the round ligament anteriorly and the fissure for the ligamentum venosum posteriorly. The round ligament of the liver is the remnant of the umbilical vein, carried well-oxygenated blood from the placenta to the fetus. The round ligament and small para-umbilical veins course in the free edge of the falciform ligament. The ligamentum venosum is the fetal ductus venosus remnant, which shunted blood from the umbilical vein to the IVC, bypassing the liver. The lesser omentum, covers the portal triad passes from the liver to the lesser curvature of the stomach and the first 2 cm of the upper part of the duodenum.



(A) Anterior view, diaphragmatic surface
(B) Open book view
(C) Postero-inferior view, visceral surface
(D) Superior view

The thick edge of the lesser omentum that extends between the porta hepatis and the duodenum encloses the structures on the porta hepatis. The sheet-like of the lesser omentum, the hepatogastric ligament, extends between the groove for the ligamentum venosum and the lesser curvature of the stomach.

impressions on areas of the visceral surface reflect the liver's relationship to the:

• anterior aspect of the stomach on the right side.

• Superior part of the duodenum.

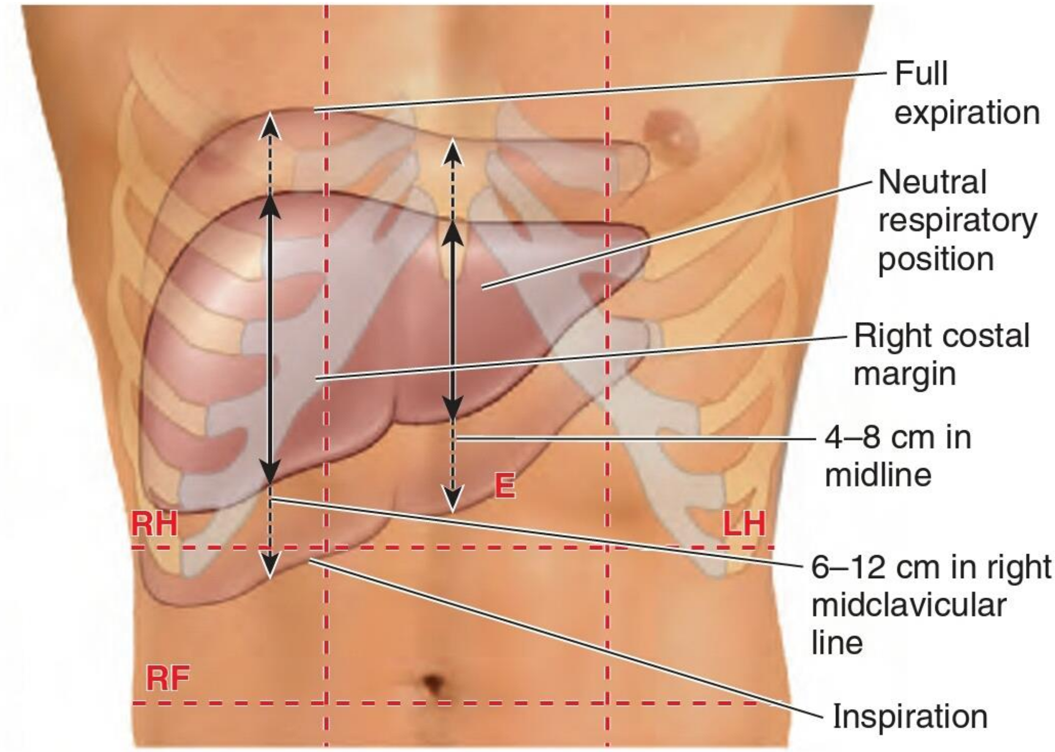
•the Lesser omentum.

• fossa of Gallbladder

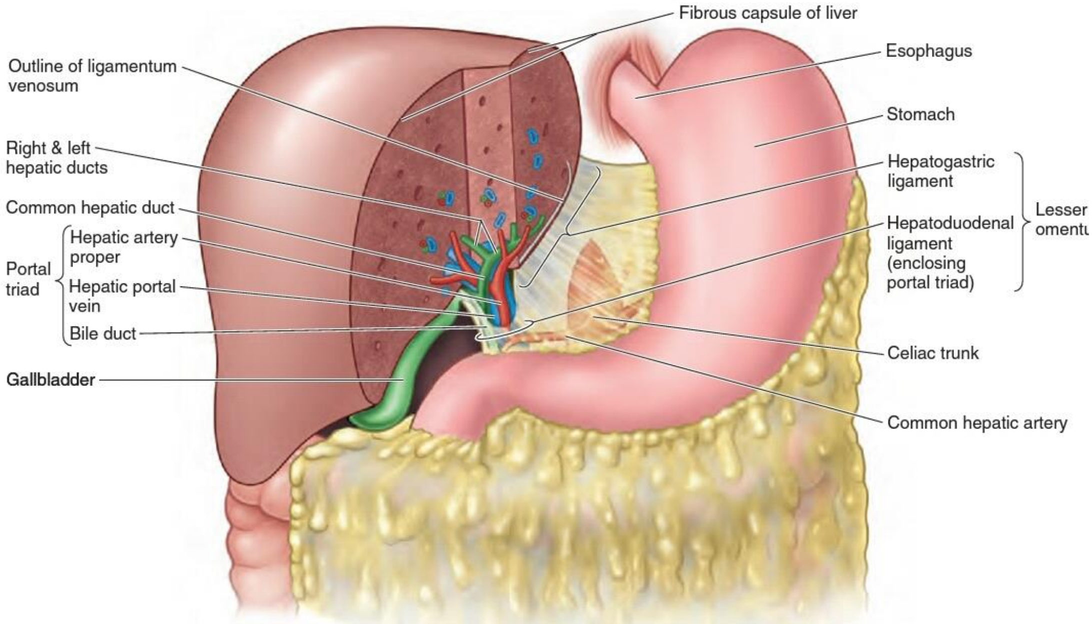
• Right colic flexure and right transverse colon

• Right kidney and suprarenal gland.

The liver lies mainly in the right upper quadrant of the abdomen, where it is protected by the thoracic cage and the diaphragm . The normal liver lies deep to ribs 7–11 on the right side and crosses the midline toward the left nipple. The liver occupies most of the right hypochondrium and upper epigastrium and extends into the left hypochondrium.



The liver moves with the excursions of the diaphragm and is located more inferiorly when one is erect because of gravity. This mobility facilitates palpation .



The liver has a convex diaphragmatic surface (anterior, superior, and some posterior) and a relatively flat or even concave visceral surface (postero-inferior), which are separated anteriorly by its sharp inferior border that follows the right costal margin. inferior to the diaphragm. The diaphragmatic surface of the liver is smooth and dome shaped, where it is related to the concavity of the inferior surface of the diaphragm, which separates it from the pleurae, lungs, pericardium, and heart. Subphrenic recesses—superior extensions of the peritoneal cavity (greater sac)—exist between diaphragm and the anterior and superior aspects of the diaphragmatic surface of the liver. peritoneal cavity immediately inferior to the liver is the subhepatic space. The hepatorenal recess (hepatorenal pouch; Morison pouch) is the posterosuperior extension of the subhepatic space, lying between the right part of the visceral surface of the liver and the right kidney and suprarenal gland. The hepatorenal recess is a gravity-dependent part of the peritoneal cavity in the supine position; fluid draining from the omental bursa flows into this recess. The hepatorenal recess

Communicates anteriorly with the right subphrenic recess.

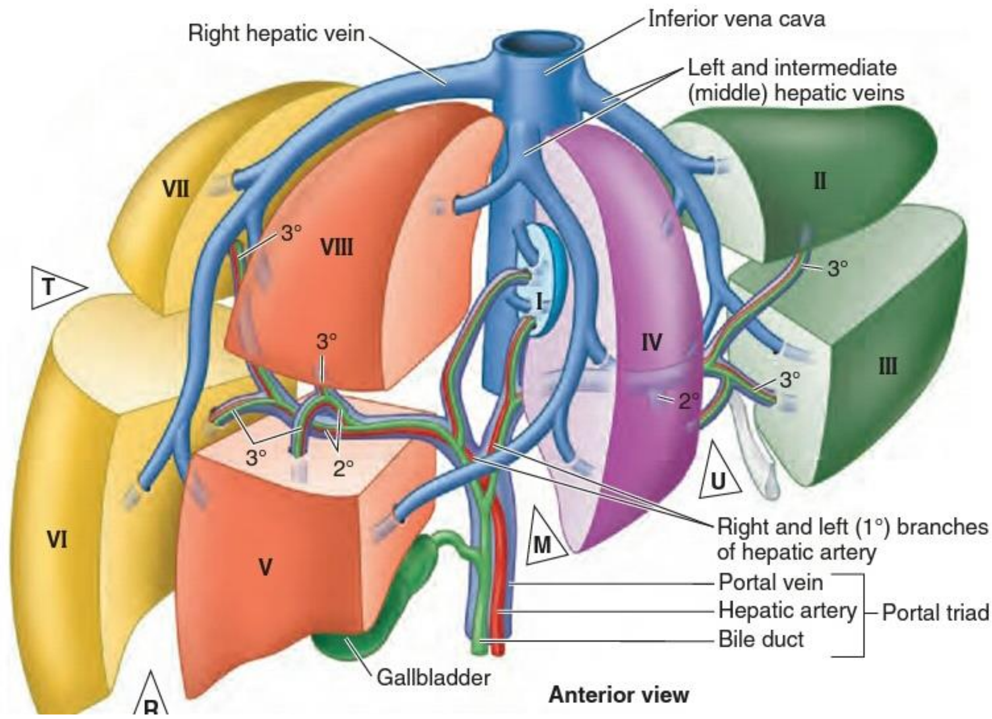
ANATOMICAL LOBES OF LIVER

Externally, the liver is divided into two anatomical lobes and two accessory lobes by the peritoneum from on its surface.the fissures formed in relation to those reflection and the vessels serving the liver and the gallbladder. These superficial lobes are not a true lobes.midline defined by the attachment of the falciform ligament and the left sagittal fissure that seperates right lobe from a much smaller left lobe. On the visceral surface, the right and left sagittal fissures continues on each side and the transverse porta hepatis separate two accessory lobes: anteriorly the quadrate lobe, and the caudate lobe posterioro superiorly. The caudate lobe was so-named because it often gives rise to tail in the form of an elongated papillary process.

A caudate process extends to the right, between the IVC and the porta hepatis, that connecting the caudate and right lobes.

FUNCTIONAL SUBDIVISION OF LIVER:

Although not distinctly demarcated internally, where the parenchyma appears continuous, the liver has functionally independent right and left livers (parts or portal lobes) that are much more equal in size than the anatomical lobes; however, the right liver is still somewhat larger. Each part receives its own primary branch of the hepatic artery and hepatic portal vein and is drained by its own hepatic duct.



The caudate lobe may infact be considered a third liver; its vascularization is independent of the bifurcation of the portal triad (it receives vessels from both bundles and is drained by one or two small hepatic veins, which enter directly into the IVC distal to the main hepatic veins. The liver can be further subdivided into four divisions and then into eight surgically resectable hepatic segments, each served independently by a secondary or tertiary branch of the portal triad, respectively . Hepatic (Surgical) Segments of Liver. Except for the caudate lobe (segment I), the liver is divided into right and left livers based on the primary (1°) division of the portal triad into right and left branches, the plane between the right and the left livers being the main portal fissure in which the middle hepatic vein lies. On the visceral surface, this plane is demarcated by the right sagittal fissure. The plane is demarcated on the diaphragmatic surface by extrapolating an imaginary line—the Cantlie line,from the notch for the fundus of the gallbladder to the IVC. The right and left livers are subdivided vertically into medial and lateral divisions by the right portal and umbilical fissures, in which the right and left hepatic veins lie . The right portal fissure has no external demarcation . Each of the four divisions receives a secondary (2°) branch of the portal triad.

A transverse hepatic plane at the level of the horizontal parts of the right and left branches of the portal triad subdivides three of the four divisions (all but the left medial division), creating six hepatic segments, each receiving tertiary branches of the triad. The left medial division is also counted as a hepatic segment, so that the main part of the liver has seven segments (segments II–VIII, numbered clockwise), which have also been given a descriptive name. The caudate lobe (segment I, bringing the total number of segments to eight) is supplied by branches of both divisions and is drained by its own minor hepatic veins.

<table>
<tr><th>Anatomical Term</th><th colspan="3">Right Lobe</th><th>Left Lobe</th><th colspan="2">Caudate Lobe</th></tr>
<tr><td rowspan="4">Functional/
Surgical Term**</td><td colspan="2">Right (part of) liver [Right portal lobe*]</td><td colspan="2">Left (part of) liver [Left portal lobe+]</td><td colspan="2">Posterior (part of) liver</td></tr>
<tr><td>Right lateral division</td><td>Right medial division</td><td>Left medial division</td><td>Left lateral division</td><td>[Right caudate lobe*]</td><td>[Left caudate lobe+]</td></tr>
<tr><td>Posterior lateral segment
Segment VII
[Posterior superior area]</td><td>Posterior lateral segment
Segment VIII
[Anterior superior area]</td><td rowspan="2">[Medial superior area]
Left medial segment
Segment IV
[Medial inferior area = quadrate lobe]</td><td>Lateral segment
Segment II
[Lateral superior area]</td><td colspan="2" rowspan="2">Posterior segment
Segment I</td></tr>
<tr><td>Right anterior lateral segment
Segment VI
[Posterior inferior area]</td><td>Anterior medial segment
Segment V
[Anterior inferior area]</td><td>Left lateral anterior segment
Segment III
[Lateral inferior area]</td></tr>
</table>

While the pattern of segmentation described here is the most common pattern, the segments vary considerably in size and shape as a result of individual variation in the branching of the hepatic and portal vessels.

BLOOD VESSELS OF LIVER:

The liver, like the lungs, has a dual blood supply (afferent vessels): a dominant venous source and a lesser arterial one. The hepatic portal vein brings 75–80% of the blood to the liver. Portal blood, containing about 40% more oxygen than blood returning to the heart from the systemic circuit, sustains the liver parenchyma . The hepatic portal vein carries virtually all of the nutrients absorbed by the alimentary tract to the sinusoids of the liver. The exception is lipids, which are absorbed into and bypass the liver via the lymphatic system.

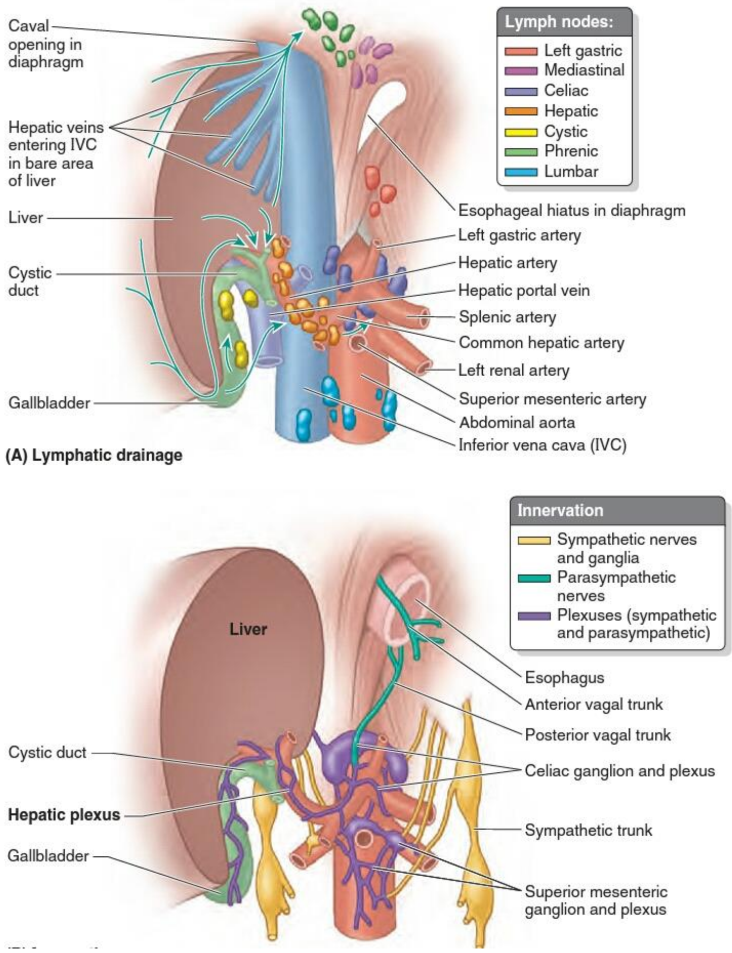
Arterial blood from the hepatic artery, accounting for only 20–25% of blood received by the liver, is distributed initially to nonparenchymal structures, particularly the intrahepatic bile ducts. The hepatic portal vein, a short, wide vein, is formed by the superior mesenteric and splenic veins posterior to the neck of the pancreas. It ascends anterior to the IVC as part of the portal triad in the hepatoduodenal ligament. The hepatic artery, a branch of the celiac trunk, may be divided into the common hepatic artery, from the celiac trunk to the origin of the gastroduodenal artery, and the hepatic artery proper, from the origin of the gastroduodenal artery to the bifurcation of the hepatic artery .

At or close to the porta hepatis, the hepatic artery and hepatic portal vein terminate by dividing into right and left branches; these primary branches supply the right and left livers, respectively . Within the right and left livers, the simultaneous secondary branchings of the hepatic portal vein and hepatic artery supply the medial and lateral divisions of the right and left liver, with three of the four secondary branches undergoing further (tertiary) branchings to supply independently seven of the eight hepatic segments. Between the divisions are the right, intermediate (middle), and left hepatic veins, which are intersegmental in their distribution and function, draining parts of adjacent segments. The hepatic veins, formed by the union of collecting veins that in turn drain the central veins of the hepatic parenchyma , open into the IVC just inferior to the diaphragm. The attachment of these veins to the IVC helps hold the liver in position.

LYMPHATIC DRAINAGE AND INNERVATION OF LIVER:

The liver is a major lymph-producing organ. one half of the lymph entering the thoracic duct comes from the liver.

The lymphatic vessels of the liver forms as superficial lymphatics in the subperitoneal fibrous capsule covers the liver, which forms its outer surface, and as deep lymphatics in the connective tissue, which accompany the tributries of the portal triad and hepatic veins .



lymph is formed in the perisinusoidal spaces of Disse and drains to the inner lymphatics in the surrounding intralobular portal triads. Superficial lymphatics from the anterior aspects of the diaphragmatic and visceral surfaces of the liver, and deep lymphatic vessels accompanying the portal triads, converge toward the porta hepatis. The superficial lymphatics drain to the hepatic lymph nodes scattered along the hepatic vessels and ducts in the lesser omentum.

Efferent lymphatic vessels from the hepatic nodes drain into celiac lymph nodes, which in turn drain into the cisterna chyli , a dilated sac at the inferior end of the thoracic duct. Superficial lymphatics from the posterior aspects of the diaphragmatic and visceral surfaces of the liver drain toward the bare area of the liver. Here they drain into phrenic lymph nodes, or join deep lymphatics that have accompanied the hepatic veins converging on the IVC, and pass with this large vein through the diaphragm to drain into the posterior mediastinal lymph nodes. Efferent lymphatic vessels from these nodes join the right lymphatic and thoracic ducts.

lymphatic vessels follow different routes are:

• From the posterior surface of the left lobe of the liver toward the esophageal hiatus of the diaphragm to end in the left gastric lymph nodes.

• From the anterior central diaphragmatic surface along the falciform ligament to the parasternal lymph nodes.

• Along the round ligament of the liver to the umbilicus and lymphatics of the anterior abdominal wall. The nerves of the liver are derived from the hepatic plexus, the largest derivative of the celiac plexus. The hepatic plexus accompanies the branches of the hepatic artery and hepatic portal vein to the liver.

This plexus consists of sympathetic fibers from the celiac plexus and parasympathetic fi bers from the anterior and posterior vagal trunks. Nerve fibers accompany the vessels and biliary ducts of the portal triad. Other than vasoconstriction, their function is unclear.

LIVER INJURY:

The liver is the most commonly injured intraabdominal organ with an incidence of 30% to 40%. The overwhelming majority of liver injuries, however, are minor, with spontaneous cessation of hemorrhage almost always the rule, and operative intervention is rarely required.On the other hand,complex hepatic injuries continue to challenge even the most experienced trauma surgeons. Hepatic injuries have been a fascinating topic since the publication of “Notes on the Arrest of Hepatic Hemorrhage Due to Trauma” in 1908 by J. Hogarth Pringle of the Glasgow Royal Infirmaries who provided the first published scientific foray into the management of severe hepatic trauma and describes one of the operative maneuvers that remains a mainstay in hepatic hemorrhage control to this day. Perhaps the single greatest advance in the management of hepatic trauma over the past two decades has been advancement and remarkable success of nonoperative management of blunt hepatic injuries. Other advances include the combination of portal triad occlusion, finger-fracture technique (hepatotomy) and omental packing for complexhepatic injuries, and perihepatic packing with planned reexploration in trauma patients demonstrating signs of the “triad of death” (acidosis, coagulopathy, and hypothermia) as well as evolving transfusion strategies stressing 1:1:1 ratio of packed red blood cells (PRBCs), fresh frozen plasma (FFP), and platelets with the goal of prevention of intraoperative coagulopathy. In the new millennium, a “multidisciplinary approach” concept has evolved as the standard of care in the treatment of complex hepatic trauma. In addition to prompt surgical intervention, when indicated, adjunctive interventional techniques such as hepatic angiography, endoscopic retrograde cholangiopancreatography (ERCP), biliary stenting, and percutaneous computed tomography (CT)– guided drainage have become a part of the trauma surgeon’s armamentarium.

INCIDENCE:

Hepatic injury occurs in approximately 5% of all trauma admissions. Nationwide, there has been a steady decline in the incidence of penetrating liver injuries. However, blunt injuries seem to be on the rise predominantly because their presence has been morereadily detected by the almost routine use of CT scanning in patients sustaining blunt trauma. The incidence of complex hepatic injuries, however, has remained relatively stable over the past 25 years, ranging from 12% to 15%. Motor vehicle crashes (MVCs) continue to account for most blunt hepatic injuries (approximately 80%), followed by pedestrian and car collisions, falls, assaults, and motorcycle crashes. Most patients with blunt hepatic trauma have associated injuries, both intra-abdominal and extraabdominal. Concomitant chest trauma is the most common associated injury encountered with blunt hepatic trauma,occurring in over 50% of patients.Patients with right sided lower rib fractures, particularly ribs 9 to 11, have at least a 20% chance of sustaining an underlying hepatic injury.In spite of the high aforementioned incidence of associated chest trauma, injury to the brain remains the single most significant determinant in overall survival outcome.

In the era of nonoperative management of blunt trauma, the risk of a missed injury, especially to the diaphragm or small bowel, is of major concern. Adherence to meticulous interpretation on imaging studies by experienced personnel should limit this pitfall to 1% to 2%.Associated intra-abdominal injuries(e.g.,stomach,duodenum,colon, and pancreas) are detected preoperatively

MECHANISM OF INJURY:

Blunt Hepatic Injury In MVCs, those most susceptible to hepatic injury are unrestrained front-seat passengers. These passengers are particularly vulnerable to a compression injury especially during periods of rapid deceleration. Although the anterior abdominal wall stops, the posterior abdominal wall continues to move forward, and the intra-abdominal organs are “trapped” and compressed, resulting in stretching/tearing of the liver at its vascular and structural attachments. As the liver is only partially protected by the rib cage, liver injury from steering wheel contact is one of the most important contributing factors to driver injury. In lateral impact (broadside or “T-bone”) collisions, the target vehicle is hit on its side and accelerated rapidly at 90 degrees to its previous direction of travel. The unrestrained passenger is subject to both compression and shear injuries that cause stretching and tearing and at times result in avulsion of the liver. Furthermore, in lateral impact injuries, because the spine and posterior abdominal wall are not in the line of impact, in contrast to frontal impact injuries, more relative motion of the intraabdominal organs ensues, resulting in a greater likelihood of injury.

DIAGNOSIS

Hemodynamically Unstable Patients:

Patients who arrive with hemodynamic instability (systolic blood pressure <90 mm Hg) and who do not immediately respond to appropriate fluid resuscitation are expeditiously taken to the operating room without delay, irrespective of mechanism of injury. Further diagnostic evaluation at this point is contraindicated, as unnecessary delays inevitably follow and are often responsible for the ensuing fatalities.

In the hemodynamically unstable patient with pelvic fractures from blunt trauma, diagnostic peritoneal lavage (DPL) which has evolved to a quick screening diagnostic peritoneal aspirate (DPA)—or DPA consisting of the initial aspiration portion of the DPL only and focused assessment with sonography in trauma (FAST) are currently the diagnostic modalities used to detect the presence of intraperitoneal blood.

A grossly positive aspiration on DPA(>10 mL of gross blood) mandates immediate operative intervention. In most trauma centers, FAST has replaced DPL/DPA as the preferred diagnostic modality for the determination of hemoperitoneum in the unstable bluntly injured patient.Although FAST has a 97% sensitivity for hemoperitoneum greater than 1 L, the location of the parenchymal injury cannot be reliably identified.The sensitivity of FAST drops precipitously when the quantity of intraperitoneal fluid is less than 400 mL.Kuncir and Velmahos found that the sensitivity and specificity of DPA was 89% and 100%, respectively, whereas for FAST it was significantly less at 50% and 95% in their prospective series of hemodynamically unstable patients with blunt abdominal trauma. If a FAST examination is equivocal in a hemodynamically unstable trauma patient, a rapid DPA should be performed

Hemodynamically Stable:

Patients The hemodynamically stable blunt trauma patient,on the otherhand, may undergo further diagnostic studies. Hemodynamic stability, however,shouldnot full the trauma surgeon into a false sense of security, as significant intra-abdominal injuries may be present despite normal vital signs and a normal abdominal examination.

The ability to accurately assess the presence or absence of significant intraabdominal Injuries by physical examination alone in the blunt trauma patient is notoriously poor, as up to 20% to 30% of patients with a benign abdomen on physical examination have been shown to subsequently have significant intra-abdominal injuries on imaging or at laparotomy. CT scanning is the preferred initial diagnostic modality in the hemodynamically stable patient with blunt abdominal or lower thoracic cage injuries.High-speed resolution scanning with a spiralscanner is employed after the administration of intravenous (IV) contrast agent. In most trauma centers, oral contrast material is no longer routinely given for screening abdominal pelvic CT scan for blunt abdominal trauma. Administration of oral contrast agent is usually reserved for the focused assessment of specific hollow viscus injuries such as identification of a duodenal laceration or delineation of a duodenal hematoma. Five-millimeter cuts are obtained after 120 mL of noniodinated contrast agent (Omnipaque) is injected at a rate of 2mL/second.Scanning commences 50seconds after injection,a delay that corresponds to the portal venous phase of liver imaging.

Scans should immediately be interpreted and classified according to the American Association for the Surgery of Trauma Liver Injury Scale by the CT fellow or attending radiologist,

TABLE I: American Association for the Surgery of Trauma Liver Injury Scale

	Grade*	Injury Description	ICD-9	AIS-90
I	Hematoma	Subcapsular, <10% surface area	864.01–864.11	2
	Laceration	Capsular tear, <1 cm parenchymal depth	864.02–864.12	2
II	Hematoma	Subcapsular, 10%–50% surface area; intraparenchymal, <10 cm in diameter	864.01–864.11	2
	Laceration	1–3 cm parenchymal depth, <10 cm in length	864.03–864.13	2
III	Hematoma	Subcapsular, >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma Intraparenchymal hematoma >10 cm or expanding		3
	Laceration	>3 cm parenchymal depth	864.04–864.14	3
IV	Laceration	Parenchymal disruption involving 25%–75% of hepatic lobe or 1–3 Couinaud segments within a single lobe	864.04–864.14	4
V	Laceration	Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud segments within a single lobe		5
	Vascular	Juxtahepatic venous injuries, i.e., retrohepatic vena cava/central major hepatic veins		5
VI	Vascular	Hepatic avulsion		6

always in the presence of the chief trauma resident and trauma attending. As a senior trauma attending usually has more experience than the designated in-house radiology resident, the surgical attending physician's initial impartial review of the CT scan is vital.

The senior trauma attending in presence makes the final decision as to the appropriateness of nonoperative therapy. It should be noted that the grade of injury or degree of hemoperitoneum on CT does not determine the need for operative intervention, as this decision is based primarily on the patient's hemodynamic stability and the absence of peritoneal sign and the absence of need for laparotomy if a concomitant hollow viscus injury is identified. Instead, the CT scan merely provides the surgeon with a general anatomic overview of the injury, identifies associated abdominal injuries requiring operative intervention, and can be used as a base for comparing future healing of the hepatic injury and resorption of intraperitoneal blood. CT can also identify injuries involving the bare area of the liver, which commonly present with minimal intra-abdominal bleeding,a paucity of abdominal signs, and often a negative DPL/DPA. The role of FAST as a screening examination in hemodynamically stable patients is evolving.

Currently, many trauma centers forgo CT scanning in stable patients with negative initial FAST examinations and merely repeat the FAST in 6 hours. However, scanning for only free fluid has its diagnostic limitations because not all blunt hepatic injuries result in hemoperitoneum.

In a recent study looking specifically at sonographic detection of blunt hepatic trauma,Richards etal determined the overall sensitivity of FAST for blunt hepatic injuries (all grades) to be 67%, based on the detection of free fluid alone. On the other hand,It is clear that most solid organ injuries without intraperitoneal fluid on FAST are, in general, of minimal clinical significance. At present, most trauma surgeons agree that those patients who are hemodynamically stable and who have either intraperitoneal blood on their initial FAST examination or positive findings on physical examination over the lower chest and upper abdomen should have a CT scan to specifically identify a hepatic or splenic injury that can be managed nonoperatively. Once identified, the hepatic injury may be followed with ultrasound if necessary. The role of DL in patients with blunt hepatic injury is less clear. DL should allow for an accurate assessment of most hepatic injuries and, as advances in laparoscopic instrumentation progress , perhaps allow for repair of some liver injuries. However, reports of missed enteric and other intra-abdominal injuries with DL are sufficiently numerous to significantly limit the usefulness of DL.

MANAGEMENT:

Resuscitation:

Approximately 80% of all the patients who die of hepatic injuries in the perioperative period is mainly from bleeding and hypovolemic shock. Profound hypothermia is frequently present when there is severe hepatic injury, particularly after repeated transfusions of non warmed blood.

The most important resuscitative technique in the patient with a major hepatic trauma includes insertion of large bore IV cannula, rapid transfusion of warm crystalloid solution and colloids (blood) and early laprotomy for controlling of ongoing bleeding.

Nonoperative Management of Blunt Hepatic Trauma:

Currently, non operative management of adult blunt hepatic injuries is the standard of care. Approximately 85% to 90% of all liver injuries may be successfully managed nonoperatively in both adults and the pediatric population. A recent publication examined the data on 14,919 liver injuries submitted to the National Trauma Data Bank and revealed that only 13.6% of all liver injuries underwent operation. As the grade of liver injury increased so did the likelihood of operative intervention:

grades I and II 8.5%, grade III 21%, grade IV 27.2% , grade V 37.4% , grade VI 42.6% . Initial hemodynamic stability or hemodynamic stability achieved and maintained with moderate fluid resuscitation is the single most crucial prerequisite qualifying patients for nonoperative management. Once hemodynamic stability has been ascertained, the following criteria must be met: Absence of peritoneal signs, Precise CT scan delineation and AAST grading) , Absence of associated intra-abdominal or retroperitoneal injuries on CT scan that require operative intervention n Avoidance of excessive hepatic-related blood transfusions Previously cited inclusion criteria such as neurologic integrity are no longer valid, as neurologically impaired patients can be safely managed nonoperatively in a monitored setting. Furthermore, mandatory repeat CT scans to document improvement or stabilization of injury are unnecessary and contribute little to patient outcome. Rather, the patient's clinical course should dictate the need for additional evaluation. Interestingly, in his landmark 1908 article describing clamping of the portal triad to arrest hepatic hemorrhage, Pringle also alluded to the physiologic tamponade provided by the abdominal wall and the potential advantage of nonoperative management in patients with less severe injuries: "The mere act of opening the abdomen, in some, at any rate, of these cases is, I feel certain, associated with an increase of the amount of blood that is lost to the patient. The blood pressure in the portal vein is not great and as the result of the local injury and the extravasation of blood there is produced reflexly a state of firm contraction of the abdominal muscles. The abdominal wall in these cases becomes absolutely rigid and board-like, the tension in the abdominal cavity thereby brought about must prevent atleast a rapid escape of blood and may lead to its arrest altogether.” Today, the majority of blunt hepatic trauma patients can be successfully managed nonoperatively. Although nonoperative management was initially limited to AAST grades I to III injuries, it is now clear that the hemodynamic status of the patient, rather than AAST grade of injury, is the most significant factor in determining the need for operative intervention. Select patients with grades IV and V injuries canbe managed nonoperatively. although hemodynamic stability determines which patients can be managed nonoperatively, the subgroup of patients with complex hepatic injuries (grades IV and V) are at substantially higher risk for treatment failure and should therefore be closely monitored in a critical care unit.

Conversely, the same basic standards apply to patients with lower AAST-grade injuries (i.e., I throughIII). Inthese instances, the initial injury maybe deemed as“notsignificant,”and thus it becomes tempting to avoid surgical intervention despite hemodynamic instability or a decreasing hematocrit, relying instead on further fluid and blood transfusions. This course of action is fraught with pitfalls and should be avoided to minimize the morbidity and mortality risks of nonoperative management.

To summarize, of all the variables monitored, hemodynamic stability appears to be the most crucial and is considered the watershed for nonoperative or operative interventioneratively.

Contrast "Blush" on Computed Tomography:

Specific cause for concern is the presence on the initial CT scan, after the administration of IV contrast agent, of a contrast "extravasation,""blush," or "pooling" of contrast material within the hepatic parenchyma. This finding indicates active bleeding. Even in the context of hemodynamic stability and irrespective of AAST grade of injury, preparation for possible surgical intervention should promptly be made, as patients can suddenly and unpredictably decompensate clinically.

If the patient remains hemodynamically stable, angiography with the intent of embolizing the lacerated vessel should be attempted (with an operating room on standby secured). An experienced interventional radiologist will usually have little difficulty in selectively catheterizing and embolizing the injured vessel, most often with stainless steel coils rather than Gelfoam to achieve the most dependable and permanent embolization.

Successful embolization can then potentially permit further nonoperative management. As the natural history of intrahepatic vessels with evidence of extravasation is unknown, they are best dealt with immediately so that sudden bleeding, false aneurysm formation, and late hemobilia may be avoided.Persistent and prolonged attempts at controlling the bleeding vessel through angiographic means should be discouraged.In the rare event in which angioembolization(AE) fails to control ongoing bleeding,surgical intervention using the angiogram as ananatomic marker to more rapidly achieve intrahepatic hemostasis should promptly be undertaken.

Operative Management:

General Principles: The four basic principles in the management of liver trauma requiring surgery are hemostasis, adequate exposure, prevention of coagulopathy, and consideration of damage control Débridement and the need for drainage are also important considerations. With hepatic injuries, these objectives can be reached by the use of the finger-fracture technique (hepatotomy) to incise hepatic parenchyma, often combined with temporary occlusion of the portal triad for hemostasis using the Pringle maneuver. Extensive débridement of injured hepatic tissue can then be done, followed by application of a viable pedicled omental pack and closed-suction drainage. Before the incision is made, the patient should receive a dose of antibiotics to cover aerobic and anaerobic microbes and is placed on a warming blanket.The surgeon must keep in mind that hypothermia is a frequent complication of resuscitation and operation in patients with major hepatic injuries. Appropriate maneuvers to decrease hypothermia. Adherence to these maneuvers will usually prevent the development of intraoperative coagulopathies, excessive hemorrhage, and fatal arrhythmias secondary to hypothermia. The skin is prepped from the chin to the knees and a standard midline incision is made.The midline incision not only affords excellent exposure of the entire liver but also provides wide access to all peritoneal and retroperitoneal structures. The combination of a long midline incision and the use of large “upper-hand” retractors have, for the most part, eliminated the need for thoracic extension of the abdominal exposure. It should be kept in mind that extending the midline incision to the sternal notch (i.e., completing a median sternotomy) exposes the patient to two open cavities with the attendant increased risks of hypothermia and coagulopathy. Exsanguinating hemorrhage continues to remain the most immediate cause of death in patients sustaining hepatic trauma. The initial incision into the peritoneal cavity can be accompanied by profuse hemorrhage once the tamponading effect has been lost. At this time, all efforts should be directed toward intraoperative resuscitation and consideration for utilizing the technique of damage control, temporary packing of the liver, and attendant correction of coagulopathy and hypothermia in the intensive care unit (ICU) with delayed laparotomy as an adjunct.

Attempts at definitive surgical hemostasis without proper intraoperative resuscitation usually results in systemic hypothermia and profound coagulation defects with their dire consequences. This fundamental pitfall should be avoided at all costs. Irrespective of the severity of hepatic injury, almost all liver injuries canbe initially managed by manually compressing the injury with lap pads while hemodynamic and metabolic stability are restored by the anesthesia team. Failure to correct hypovolemia and acidosis before attempts at surgical control will likely lead to cardiac arrest and subsequent death. Once intraoperative resuscitation has been achieved, manual compression of the liver is slowly released so that a more accurate assessment of the injury can be made. Division of the falciform ligament allows for placement of an "upper-hand,self-retaining retractor in the incision.In order to better visualize injuries on the superior or lateral aspects of an injured hepatic lobe, it is often necessary to mobilize the liver into the midline wound.Once this is done,careful traction on its hepatic end can aid in exposing the dome of the liver and the suprahepatic inferior vena cava.

Additional exposure is obtained by placing laparotomy pads behind the posterior surface of the liver. Mobilization of the right and left lobes proceeds with division of the triangular ligaments .

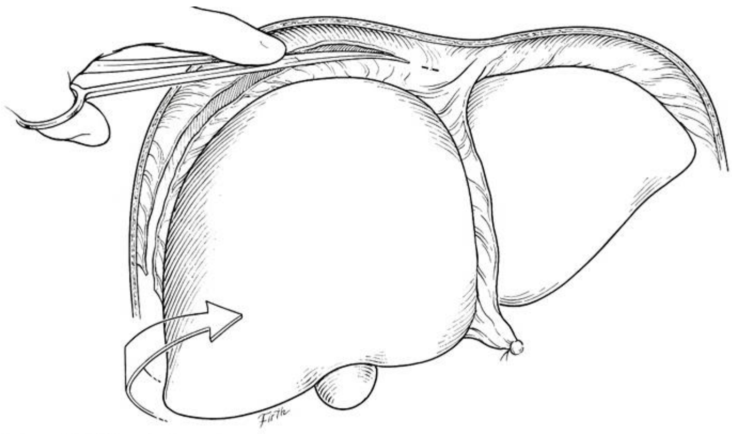
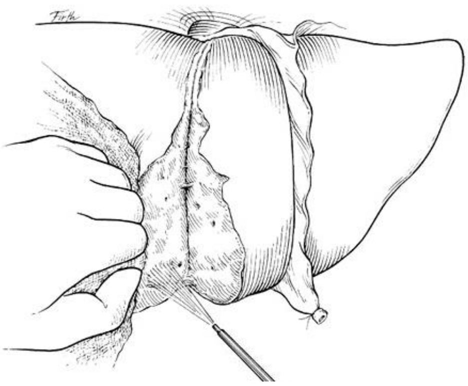


FIGURE 4 Mobilization of right triangular ligament.



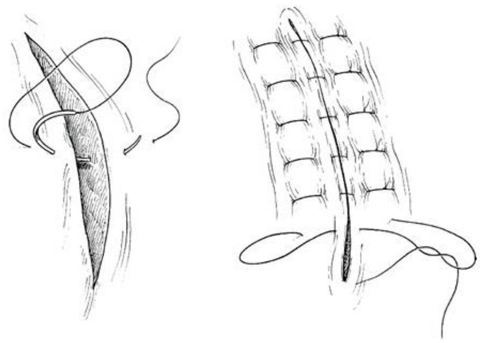


FIGURE 6 Suture hepatorrhaphy.

If there is a hematoma within the leaves of the triangular ligament, a hepatic vein or venal caval injury is most likely. If the hematoma is not expanding and there is no immediate active hemorrhage requiring control, entering a stable retrohepatic hematoma is not advised. Extreme caution must be taken even during traction as this may disrupt a stable hematoma and can create massive bleeding.

Minor Injuries (Grades I and II) Simple techniques of controlling hemorrhage include a 5- to 10 minute period of compression,application of topical agents including fibrin glue, electrocautery/argon beam electrocoagulation, and suture hepatorrhaphy. In many patients with superficial lacerations of the capsule, a 5- to 10-minute period of compression will frequently control any hemorrhage. If there is no visible leakage of bile, no further therapy is indicated. Topical agents, such as fibrin glue, Surgicel, and Avitene, are useful when avulsion of Glisson's capsule is present. Five minutes of compression with lap pads is performed after the application of a topical agent to the raw surface.After releasing compression, the electrocautery can be used for any remaining bleeders. Fibrin glue or the other hemostatic agents may be overlaid with a large Gelfoam pad creating an on adherent surface to compress a gauze laparotomy pad against. Drainage is not necessary in the absence of obvious bile leakage. Suture hepatorrhaphy has historically been the mainstay of hepatic hemostasis in grade II and some grade III injuries.It is important to first enter the hepatic wound and selectively ligate any open or avulsed bile ducts or blood vessels. Figure-of-eight 2-0 or 3-0 Prolene sutures are usually employed. Alternatively, 2-0 or 3-0 chronic sutures or hemoclips can also be used. Small defects in the hepatic parenchyma can be closed with simple interrupted 0-chromic or 2-0 chromic liver sutures either with regular or blunt-nosed needles. For deeper lacerations, attempts at primary closure of the hepatic defect should not be undertaken. Instead, a flap of omentum on a pedicle is placed within the hepatic parenchymal defect and is then held in place with interrupted liver sutures. It is important to loosely approximate the edges because portions of the liver beneath can Become necrotic in the postoperative period if the sutures are tied too tightly.Complex Injuries (Grades III and V) If significant hemorrhage continues after the release of manual compression of the liver, the portal triad should be occluded with an atraumatic vascular clamp (the Pringle maneuver.

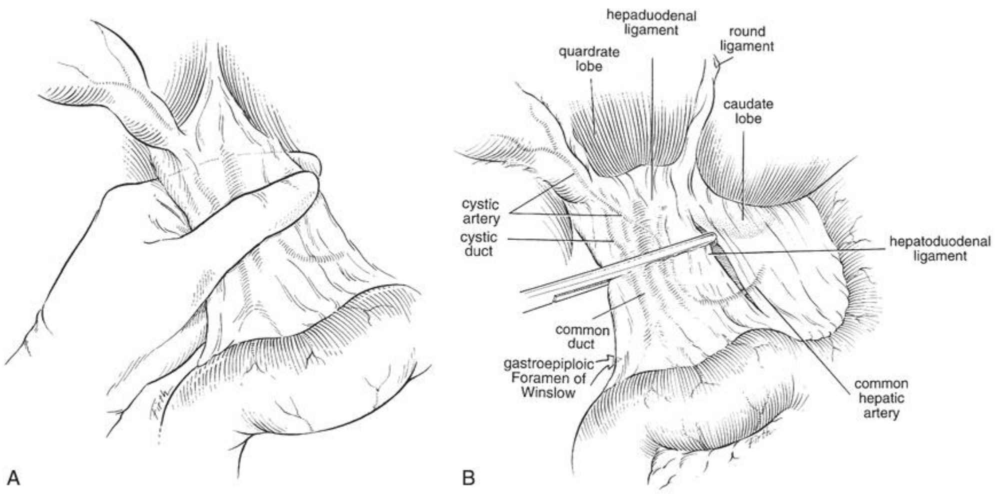


FIGURE 8 **A** and **B,** Pringle maneuver.

In over 85% of patients with complex hepatic injuries, occlusion of the portal triad will temporarily stop the bleeding.

This maneuver,coupled with the finger-fracture technique to expose lacerated blood vessels for direct repair, is responsible for the dramatic decrease in deaths from exsanguination.

Complex hepatic injuries (gradesIII to V) canbe managed by adhering to several sequential crucial steps:

1. Portal triad occlusion (Pringle maneuver)
2. Finger fracture of the hepatic parenchyma (hepatotomy), exposing lacerated vessels and bile ducts for direct ligation/ repair
3. Consideration of temporary packing with laparotomy pads to allow appropriate intraoperative resuscitation
4. Consideration of temporary intrahepatic packing with hemostatic agents such as surgical Nu-Knit
5. Débridement of nonviable hepatic tissue
6. Placement of an omental pedicle, with its blood supply intact, into the injury site
7. Closed-suction drainage

Much controversy has surrounded the normothermic ischemic time produced by the Pringle maneuver. The data are clear that complex hepatic injuries can be managed with continuous cross-clamping of the porta hepatis for up to 75 minutes without adverse sequelae.

With portal triad occlusion achieved by an atraumatic vascular clamp, the surgeon then opens the liver parenchyma (hepatotomy) in the direction of the injury. Although it initially seems crude, the finger-fracture technique constitutes the benchmark of obtaining rapid, adequate exposure. Specifically, using the electrocautery, Glisson's capsule is incised in the direction of the injury. Normal hepatic parenchyma is then crushed between the surgeon's thumb and index finger, thereby rapidly exposing injured blood vessels and bile ducts, which are repaired or ligated under direct vision. Narrow Deaver or malleable retractors can be inserted into the hepatotomy tract for better intrahepatic exposure .Large lacerated intralobar branches of the portal vein or hepatic veins can be repaired in a lateral fashion using 5-0 Prolene sutures . After intrahepatic hemostasis has been achieved, thorough débridement of devascularized hepatic tissue is essential to avoid postoperative septic complications. The use of omentum is extremely beneficial in the management of complex hepatic injuries, as it provides viable tissue to fill dead space, tamponades minor venous oozing,and provides a rich source of macrophages that may help combat infection. The choice to drain a liver injury is controversial and debatable.

If bile is noted intraoperatively, drainage is not controversial and is mandatory. The preferred method of drainage is with closed-suction Jackson-Pratt (JP) drains anterior and posterior to the injury. The data rendering drains unnecessary in elective hepatic resection cannot be applied to complex hepatic trauma, in which blood loss,hypotension,and the frequent need to terminate surgery are the usual order of the day. In addition, the "zone" of injury may extend centimeters beyond what appears to be normal hepatic parenchyma, leading to eventual necrosis and abscess formation. Although routine drainage after elective hepatic resection may be superfluous, enough variables exist in the trauma setting to merit consideration of the use of closed-suction drains for complex hepatic injuries.

Damage Control:

Perihepatic Packing and Planned Reexploration Perihepatic packing has emerged as an essential lifesaving maneuver in patients with complex injuries refractory to conventional methods of treatment and usually complicated by brisk bleeding, hypothermia (less than 34° C), acidosis (pH <7.2), and coagulation defects from massive transfusion (over 10 units PRBCs).

The effectiveness of perihepatic packing is directly related to the tamponading effect of the packs on the hepatic injury. Specifically, the packs raise intraabdominal pressure (IAP), causing tamponade of low-pressure venous and non mechanical capillary bleeding. The key to the success of perihepatic packing is to insert the packs early in the course of the operation before the onset of repeated episodes of hypotension.

Primary indications for perihepatic packing follow:

Onset of intraoperative coagulopathy Extensive bilobar injuries in which bleeding cannot be controlled, Large expanding subcapsular hematomas or ruptured hematomas, The necessity to terminate surgery as a result of profound hypothermia,which usually results in hemodynamic instability n Failure of other maneuvers to control hemorrhage n Patients who require transfer to Level I trauma centers Juxtahepatic venous injuries As a general rule, the liver should be mobilized before packing to help establish a tamponading effect. If, however, a significant hematoma is encountered in the triangular ligament (indicative of a vena caval or hepatic vein injury), further mobilization is contraindicated as massive and uncontrollable bleeding may follow.

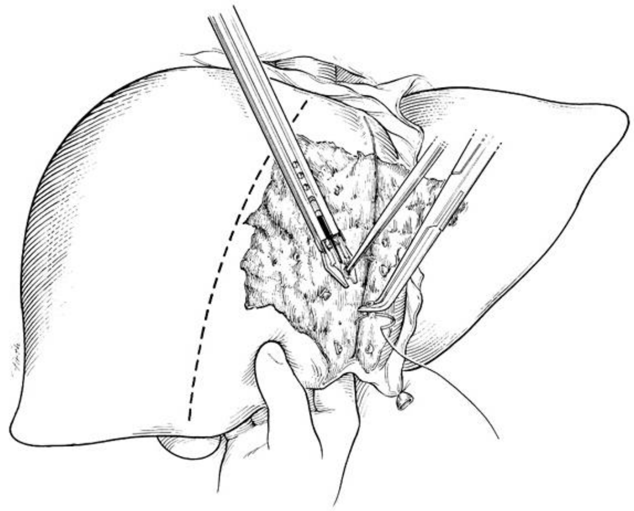


FIGURE 11 Ligating large branches with Prolene sutures or clips.

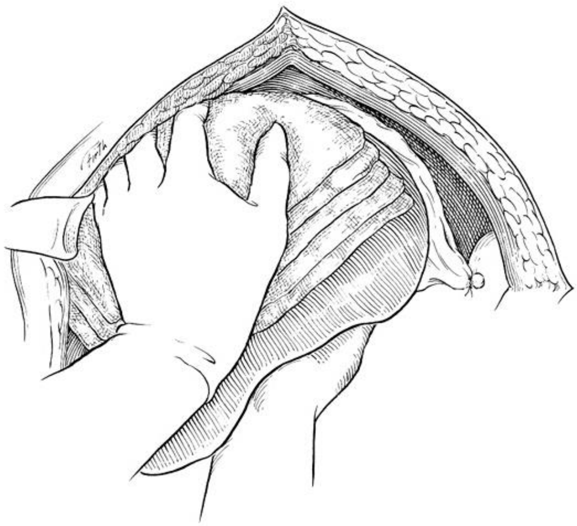


FIGURE 12 Perihepatic packing.

Most often, dry multiple-lap pads are placed on top of the injured liver until the ipsilateral hemidiaphragm is reached. In order to lessen the degree of bleeding when lap pads are peeled off the raw liver surface, an option is to routinely place a Steri-Drape directly upon the liver surface to serve as an interface between the injured liver and the lap pads.

Another alternative is to place large noncompressed sheets of Gelfoam over the raw liver surface as a hemostatic adjunct which additionally provides a layer of protection preventing bleeding when the laparotomy packs Resorting to packing is usually synonymous with a dire situation. Under these circumstances, rapid closure of the abdomen with towel clips can be undertaken. Several large Steri-Drapes impregnated with Betadine cover the entire incision, encompassing all towel clips. Towel-clip closure takes minutes to perform and facilitates rapid patient transfer to a critical care setting where the patient's metabolic status can be optimized.Alternatively, prosthetic abdominal wall closure with a sterilized IV bag, commonly known as a Bogata bag, can also be employed. Commercially designed wound VAC (vacuum assisted closure) systems are also available and extremely useful with both the wound VAC and the latest version of this system.which is now being used most of the time. Because perihepatic packing raises IAP, monitoring IAP in the perioperative period is critical to avoid the development of an abdominal compartment syndrome (ACS). Pack removal should be dictated by the reversal of the patient's hypothermia, acidosis, and coagulopathy. These goals can usually be achieved within 36 to 48 hours.

Packing has been historically associated with a 20% to 30% incidence of perihepatic sepsis. Early pack removal, evacuation of intraperitoneal clots, and the thorough débridement of necrotic hepatic tissue have lessened the incidence of this complication.

MORBIDITY AND COMPLICATIONS MANAGEMENT:

Failure of Nonoperative Management Nonoperative management of adult blunt hepatic injuries has resulted in a survival rate approaching 100%. Overall complication rates in most series range from 0% to 13%.As the mortality rate associated with nonoperative management essentially is 0%, focus has shifted to the delineating and managing the associated morbidity.Primary hepatic morbid conditions include bleeding, biliary complications, ACS, and infection complications. Most common hepatic complications included bleeding , biliary complications , ACS , and infectious complications . Bleeding and ACS tended to develop early—within 3 days after injury; but biliary and infectious complications tended to develop late—greater than 3 days after injury. Hemorrhage and Abdominal Compartment Syndrome,Hemorrhage complicating the nonoperative management of hemodynamically stable patients with blunt injuries occurs at a frequency of 5% to 10%.

Rates of bleeding are particularly low when a helical contrast-enhanced CTscan fails to show an active blush.Active blush on CT scan mandates AE even when a patient's physiology does not show any current sign so decompensated shock in apremptive measure to ensure that the patient maintains an acceptable state of hemodynamics. For the most part, failed nonoperative management follows a predictable pattern of ongoing hemorrhage,rather than sudden decompensation.Most failures of nonoperative managemen tsecondary to bleeding occur within the first 24 hours. The reported incidence of delayed hemorrhage requiring laparotomy is well under 2% to 3%. Bleeding after operative management of hepatic injuries is usually not subtle as evidenced by hemodynamic instability that can also be accompanied by brisk bleeding from operatively placed intraperitoneal drains. However, a more subtle presentation is the hemodynamically stable patient with a partially distended abdomen accompanied by a decreasing hematocrit. In the past, reoperation to control bleeding from within the injured liver was promptly undertaken after correction of acidosis, hypothermia, and coagulation defects. Currently, in the absence of hemodynamic instability, AE is the preferred treatment.

In the hemodynamically unstable postoperative patient, reexploration is warranted. The same techniques apply, namely manual compression of the liver with concomitant intraoperative resuscitation, the Pringle maneuver, and finger fracture, if necessary, through the repaired area.If there is a concern that extensive hepatotomy may sever major vascular structure And hepatic bileducts,persistent bleeding may be controlled by extralobar hepatic arterial ligation or balloon tamponade with Penrose and red rubber catheters.If a diligent search has failed to reveal a mechanical source of bleeding, a transfusion related coagulopathy is the most likely culprit. Under these circumstances, packing of the injured liver should be undertaken rapidly, following the guidelines for packing removal as described earlier. Findings from recent military data have revolutionized transfusion practices and contribute to a higher likelihood of prevention of coagulopathy and thus cessation and prevention of hemorrhage. Current transfusion practices of a ratio of 1:1 of FFP to PRBCs have shown to significantly decrease coagulopathy and have had a remarkable impact on overall mortality rate. The goal target ratio of PRBCs to FFP is now agreed upon to be 1:1 with an ideal range of 1:1 to 1:1.8.

More frequent platelet administration is also warranted, although the ideal ratio has not yet been identified. These changes in transfusion practices have had a marked impact upon controlling coagulation defects and managing hemorrhage from hepatic injuries, although the specific effects of this new approach in transfusion practices on outcomes in higher grade hepatic injuries has yet to be delineated.

Late Complications: Biliary and Infectious Looking at all the studies to date;biliary and infectious complications run neck in neck, with rates averaging 3% to 6% for both.Biliary complications developed at a mean of 12days after injury with a range of 2 to 38 days. Infectious complications on average developed on post injury day 15 with a range of 1 to 90 days.Hemobilia is an uncommon complication of hepatic trauma, occurring at most in 1%. Hemobilia may result from blunt or penetrating trauma or iatrogenically induced by deep suture hepatorrhaphy.Signs and symptoms of gastrointestinal hemorrhage, right upper quadrant pain, and jaundice (Sandblom triad) may occur 4 days to 1 month after injury. Repeat endoscopy is usually unrevealing. In this setting, a history of trauma mandates celiac angiography.

If hemobilia is the cause of the bleeding, angiography will demonstrate a hepatic artery pseudoaneurysm that can be embolized with steel coils, Gelfoam, or acrylate glue. Coils form the most permanent embolization when other methods may recannulize and present a risk of rebleeding. Surgical intervention is rarely necessary and not necessarily advisable secondary to the difficulty associated with anatomically accessing the bleeding vessel, which is often deep within the hepatic parenchyma, unless hemobilia is either associated with a large intrahepatic cavity or angiography is not available. If surgery is required, the optimal treatment is hepatic resection encompassing the large cavity and the pseudoaneurysm. Vascular control (by intraoperative Pringle maneuver or direct ligation of the hepatic artery) is essential before attempting to débride or resect large intrahepatic cavities associated with hepatic artery pseudoaneurysms.

Injury to the Intrahepatic Bile Ducts and Late Stricture Injuries to the intra hepatic bileducts are rare.The long-term sequelae of spontaneous healing of the injured hepatic parenchyma surrounding both normal and disrupted intrahepatic bile ducts are presently unknown.

Although disruption of secondary and tertiary biliary radicals within the liver occurs often, late intrahepatic bile duct stricture formation is an exceedingly rare occurrence.

Perihepatic Sepsis/Abscess:

Perihepatic sepsis/abscess associated with complex hepatic injuries is a late complication and develops at a rate of 3% to 6%. Perihepatic sepsis, especially in the multiply injured patient, can lead to septic shock formation, systemic inflammatory response syndrome, and multiple organ failure, placing the patient at risk for a late death. Noninfectious factors can also initiate severe inflammatory responses that may culminate in multiple organ failure and death. A variety of risk factors for postoperative abscesses after hepatic trauma have been identified, including associated enteric injuries, extent of parenchymal damage, transfusion requirements, and inadequate débridement/drainage at the initial operation. For the most part, the rate of hepatic abscess formation in operatively managed hepatic trauma can be significantly reduced with meticulous hemostasis, adequate débridement of nonviable hepatic parenchyma, and avoiding open-suction drainage. Most abscesses can be drained percutaneously. Failure of the septic patient to improve within 24 to 36 hours after percutaneous drainage is a compelling reason to repeat the CT scan to determine if the catheter needs to be readjusted or whether operative intervention is needed. If surgery is required, the abscess cavity should be unroofed, devitalized tissue débrided, and closed-suction drainage established. Rarely, resectional débridement or frank lobectomy may be required to eradicate either an infected biloma or abscess cavity. Moreover, healing by secondary intention resulted in wound bursting strength equal or greater than hepatorrhaphy or hepatorrhaphy and omental packing at 3 and 6 weeks. The healing mechanism responsible for the increased wound bursting strengths appears to be the proliferative fibrosis throughout the injured hepatic parenchyma and the overlying Glisson's capsule. Hepatic parenchymal healing appears to be virtually complete at 6 to 8 weeks after injury. A reasonable and safe approach to pursue would be to allow patients with grade III or greater injury to resume normal activities after CTscan documentation, at 3months of major injury resolution.

MORTALITY:

The overall liver-related mortality rate in most large series of nonoperatively managed blunt hepatic injuries is 0.5%. Most liver-related fatalities result from complex hepatic trauma (grades IV and V), especially juxtahepatic venous injuries and portal triad injuries, which often result in prohibitively high mortality rates. Over the past 2 decades, the mortality rate of complex hepatic injuries has decreased, predominantly because of a reduction in deaths from liver hemorrhage.Responsible contributing factors include prolonged inflow occlusion times, hepatotomy with selective vascular ligation, early packing and reexploration,and adjunctive interventional procedures, especially hepatic artery AE.

MATERIALS AND METHODS

DESIGN OF STUDY : Prospective Study

PERIOD OF STUDY : 1 year (June 2018 to June 2019)

SELECTION OF SUBJECTS: Age more than 15 years presenting with blunt injury abdomen with suspected liver injuries in trauma casualty.

Inclusion criteria:

Patients more than 15 years of age groups in both sexes presenting with blunt injury abdomen with liver injuries in GRH Madurai.

Patients consented for inclusion in the study according to designated proforma.

Exclusion criteria:

Patients less than 15 years of age.

Patient not consented for inclusion in the study.

Patient presented with penetrating abdominal injuries.

Patient with blunt injury abdomen associated with other solid organ injuries excluding liver injuries

Associated hollow viscous injury or other systemic injuries.

Methods of collection of data:

After admission of the patient , data was collected for my study by: Direct interview with the patient attenders or the patient accompanying the patient and obtaining a detailed clinical history. Thorough clinical examination. Clinical findings and needed diagnostic investigations performed over the patient.

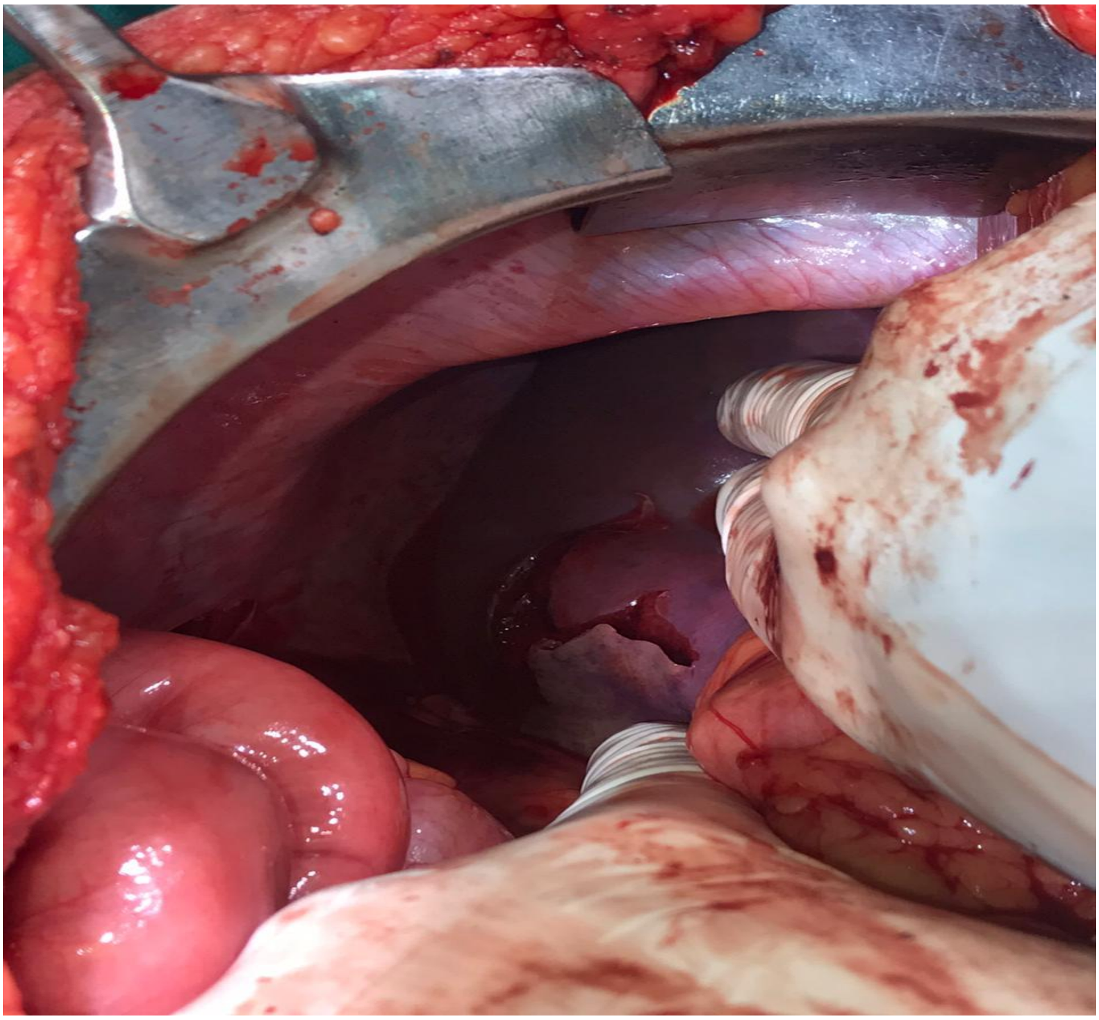
thorough assessments for liver injury were carried out After initial resuscitation of the patients.Documentation of patients, which included, gender,age, history includes nature and mode of injury, clinical findings, diagnostic test, operative findings, operative procedures, complications during the hospital stay and during subsequent follow-up period, were all recorded on a Proforma specially prepared.decision was taken for further investigations such as Complete blood count,four – quadrant aspiration , x ray abdomen erect, chest , FAST , CT abdomen & pelvis.The decision for operative or non operative management depended on the outcome of the examination findings, hemodynamic status and CECT abdomen. Patients selected for non operative or conservative management were placed on strict bed rest, were subjected to serial clinical examination which included hourly pulse rate, blood pressure,serial hematocrit measurement and repeated examination of abdomen and other systems.

Appropriate diagnostic tests especially ultrasound of abdomen was repeated as and when required. FAST was done in all patients. In general following cases are taken up for immediate surgery.

1) patient with evidence of significant hemoperitoneum

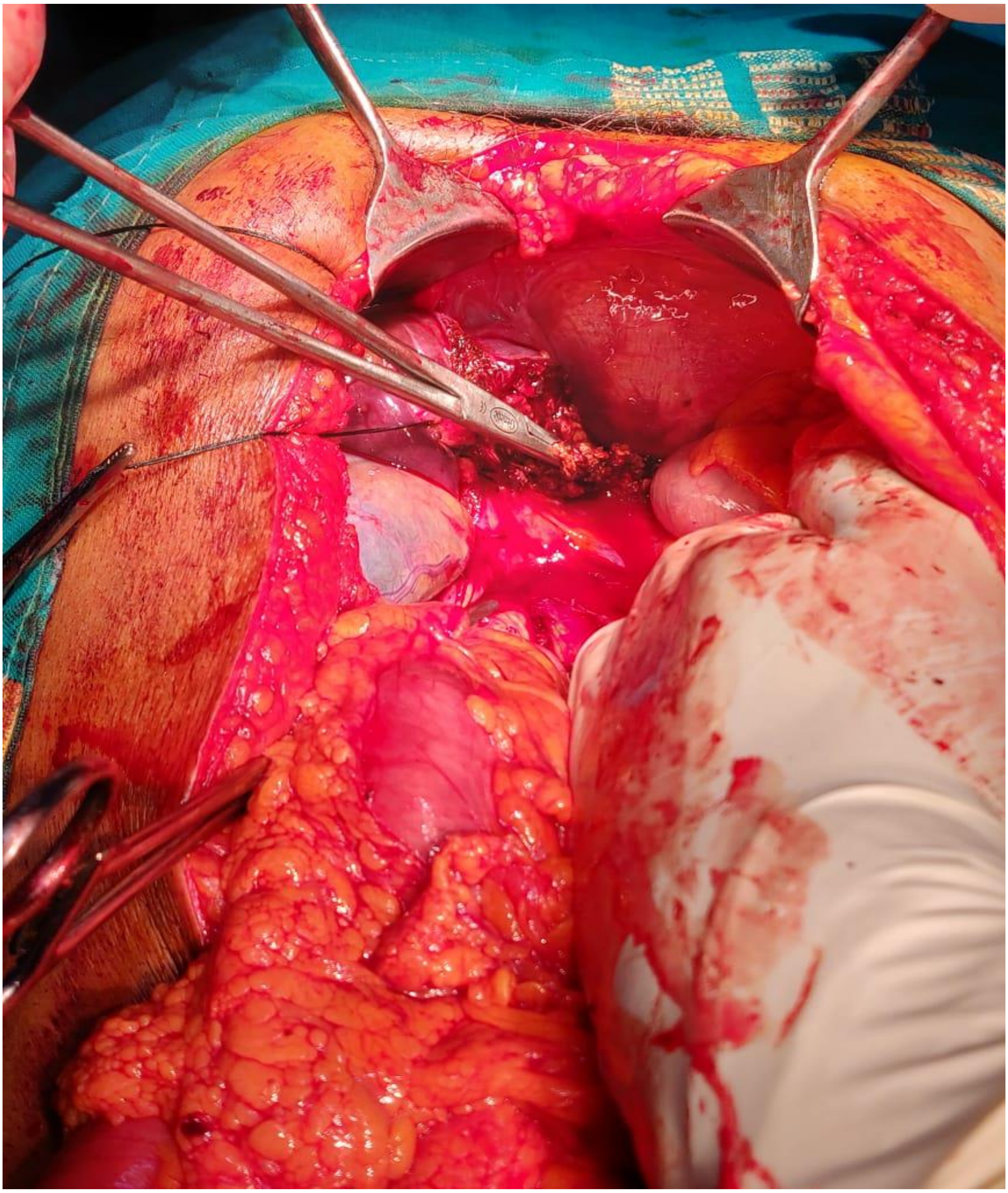
2) Those patients presented with signs of peritonitis

3) Patients not responding to non operative/conservative management and those deteriorating despite adequate Resuscitation .



The above picture shows that grade 3 liver injury ,patient presented with liver injury with hemodynamic instability,we done a laparotomy with gelfoam packing of liver laceration after achieving hemostasis.

HEPATECTOMY:



The above pictures shows hemostasis ,was done after left hepatectomy due to high grade liver injury includes vascular injury.

Resuscitation and Treatment :

Following definitive management patients are monitored closely for signs of recovery & complications, if any, are managed accordingly. Any mortality is recorded and cause of the death analysed.

Follow up: Patients are discharged when they are fit and advised to come for follow at regular interval.

DATA COLLECTION:

All the above details of every patient is recorded in a specific proforma designed for the study and finally inferences are drawn in tables &charts regarding the causes , age and sex distribution , pattern of liver injury , management , complications , morbidity and mortality.

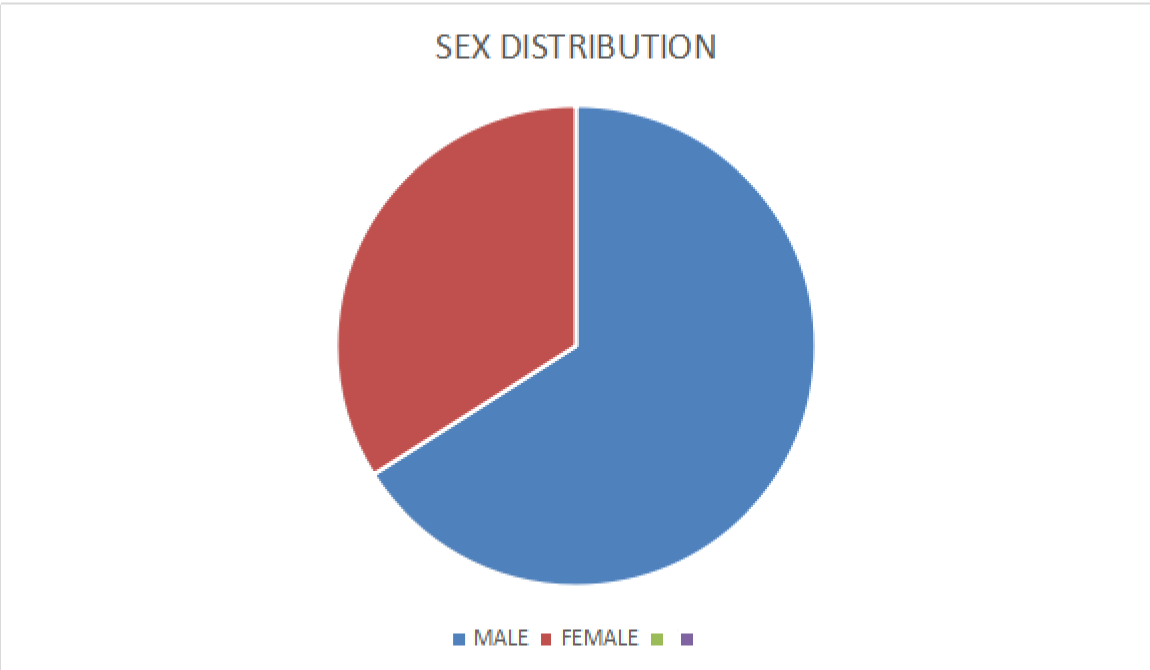
RESULTS AND ANALYSIS

FROM JUNE 2018-2019 ,50 number of cases were studied which belongs to surgical units in GRH, Madurai

SEX INCIDENCE

SEX	NO OF PATIENTS	PERCENTAGE %
MALE	33	66
FEMALE	17	34
TOTAL	50	100

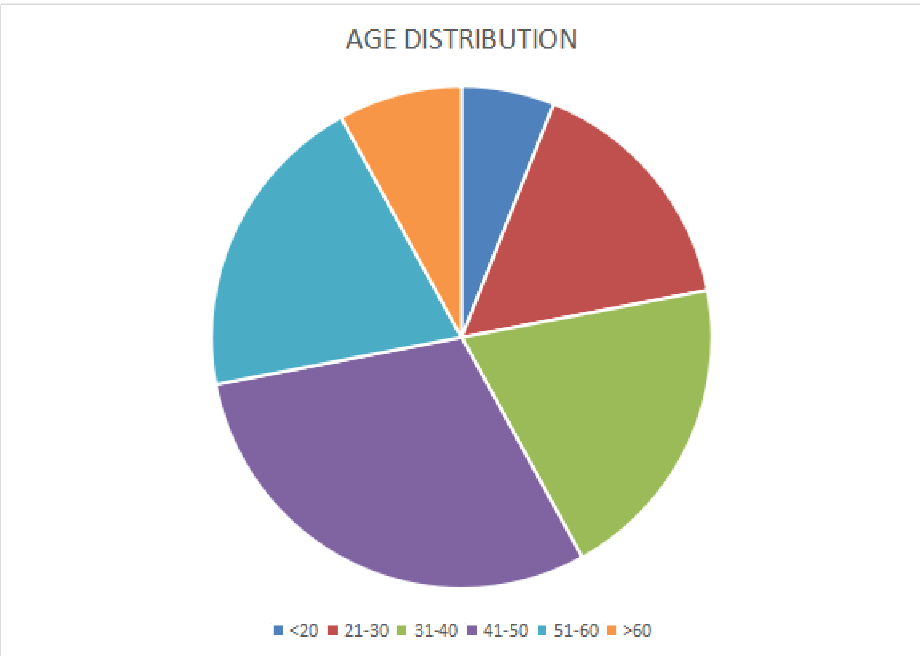
in our study, in 50 cases 33 were males accounting 66% of study population and 17 were females.



AGE INCIDENCE

AGE GROUPS	NO OF PATIENTS	PRECENTAGE %
< 20	3	6
21-30	8	16
31-40	10	20
41-50	15	30
51-60	10	20
>60	4	8

In this study majority belongs to age 41-50 years of age accounting for 30% followed by 31-40 years of age & 51-60 years of age accounting for 20 %

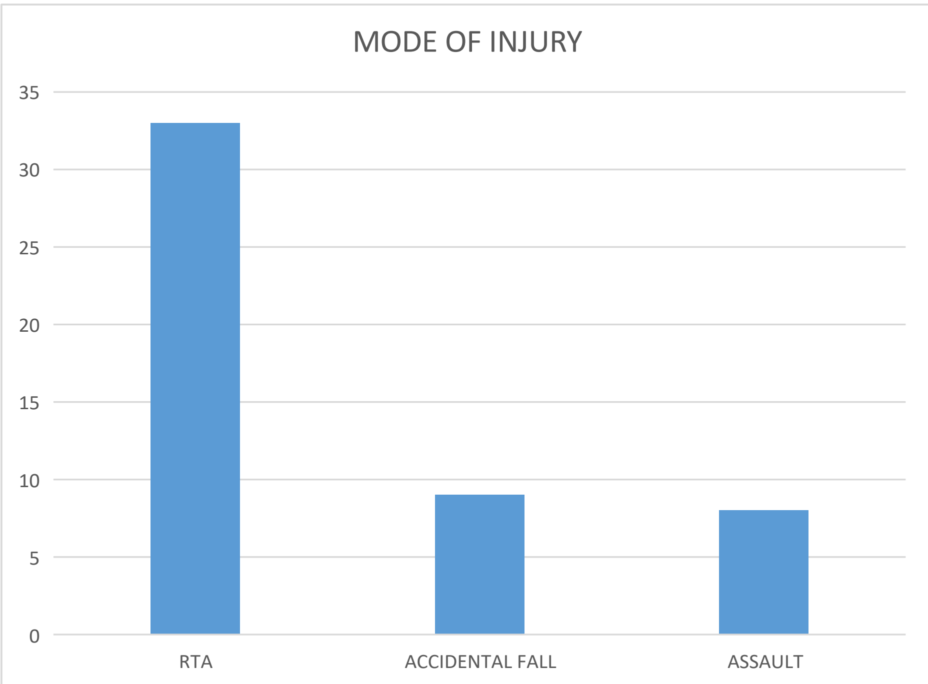


MODE OF INJURY

MODE OF INJURY	NO OF PATIENTS	PERCENTAGE %
RTA	33	66
ACCIDENTAL FALL	9	18
ASSAULT	8	16

In our study,66% of mode of injury belongs to road traffic accident, followed by accidental fall in 18 %.least was assault in 16%

MODE OF INJURY

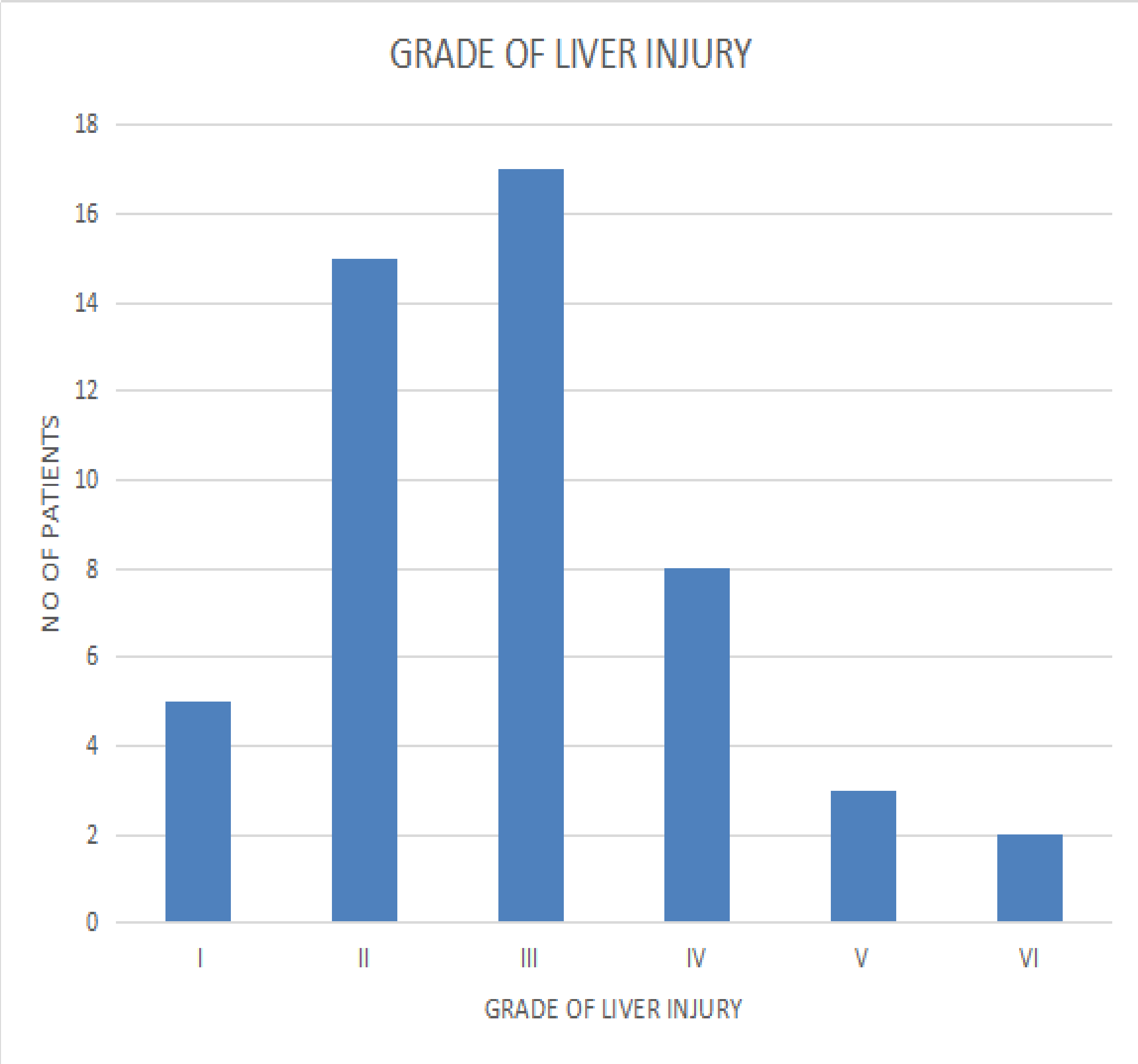


GRADE OF LIVER INJURY

GRADE OF LIVER INJURY	NO OF PATIENTS	PERCENTAGE %
I	5	10
II	15	30
III	17	34
IV	8	16
V	3	6
VI	2	4

In our study ,34 % of the liver injury were grade III followed by grade II injury in 30 % of cases ,least was grade VI in 4%.

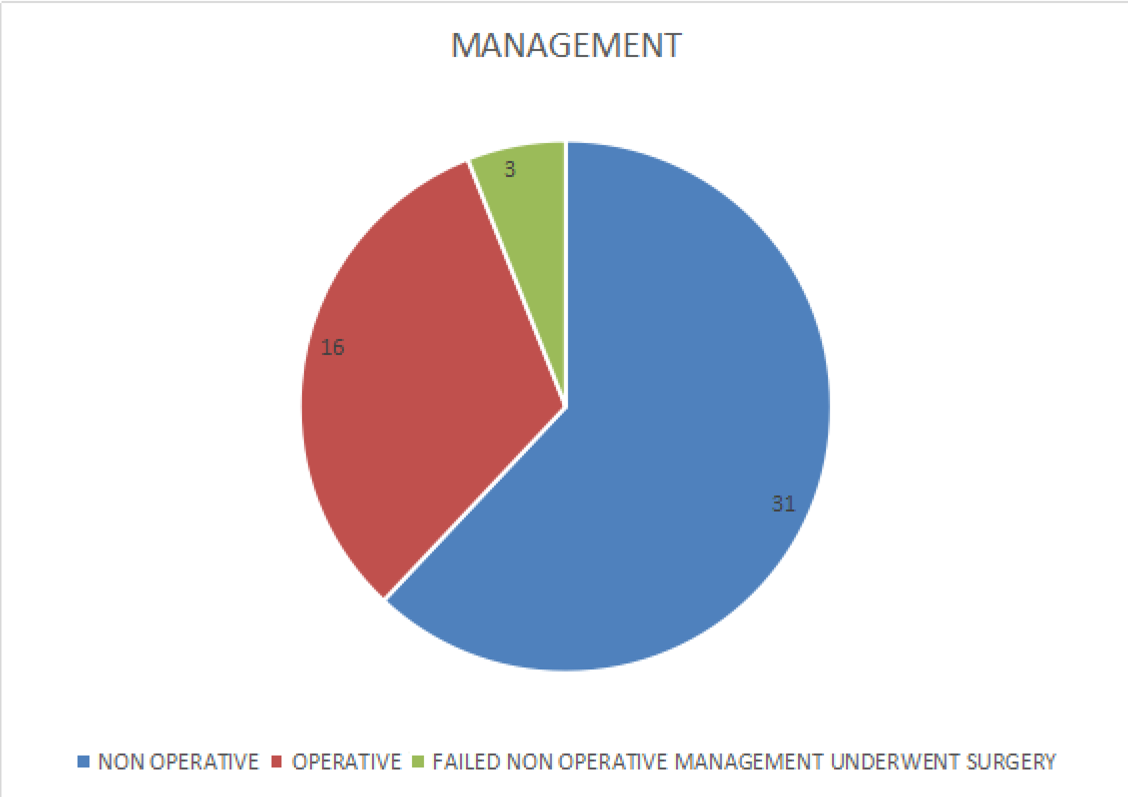
GRADE OF LIVER INJURY



TYPE OF MANAGEMENT

MANAGEMENT	NUMBER OF PATIENTS	PERCENTAGE %
NON OPERATIVE	31	62
OPERATIVE	16	32
FAILED NON OPERATIVE PATIENTS UNDERWENT OPERATIVE PROCEDURES	3	6

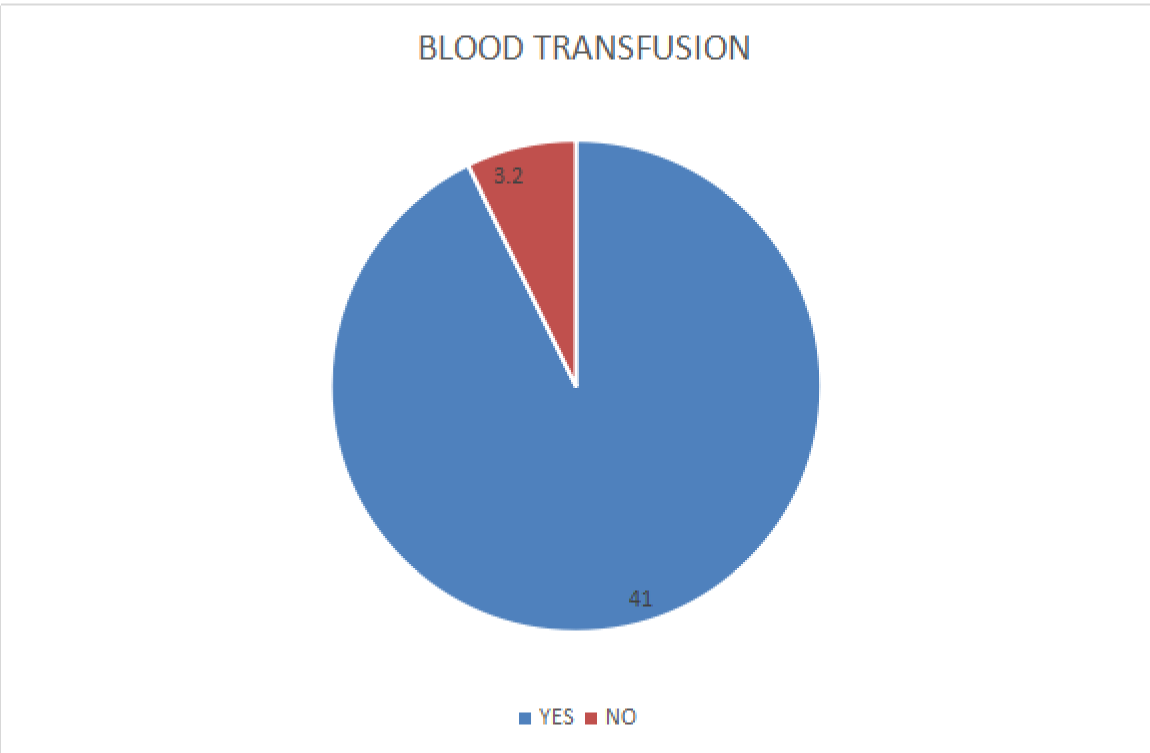
In our study, 62% of patients managed non operatevely because they had no signs of peritonitis and hemodynamically stable,32 % patients directly underwent emergency laparotomy because of hemodynamic instability at initial presentation,6 % of the patients were initially in non operative management underwent emergency laparotomy because of failed non operative management.


MANAGEMENT
3
16
31
NON OPERATIVE
OPERATIVE
FAILED NON OPERATIVE MANAGEMENT UNDERWENT SURGERY

BLOOD TRANSFUSION

BLOOD TRANSFUSION	NO OF PATIENTS	PERCENTAGE %
YES	41	82
NO	9	18

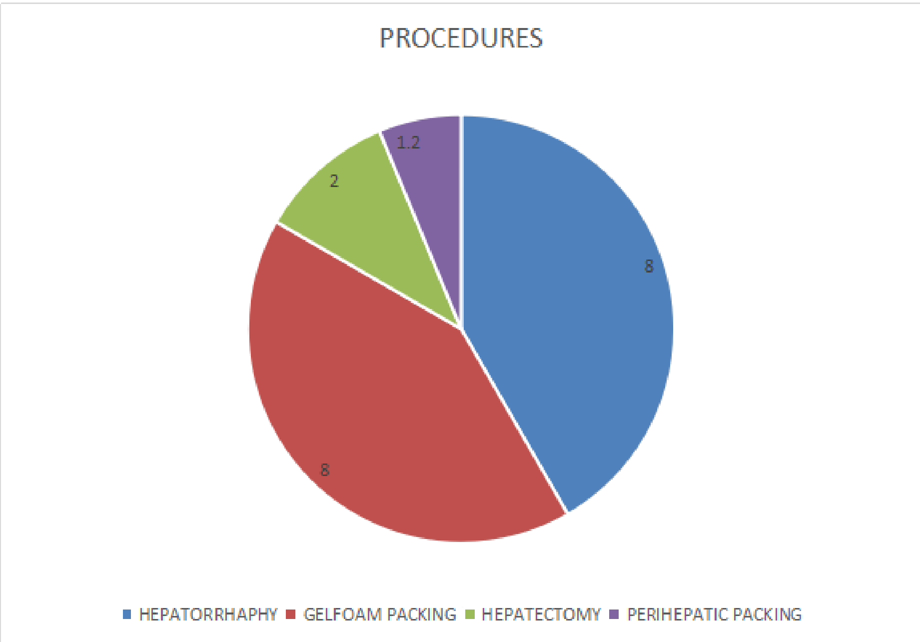
In our study,82 % of the patients received blood products transfusion ,18 % were not received blood transfusion.



PROCEDURES

PROCEDURE	NO OF PATIENTS	PERCENTAGE %
HEPATORRHAPHY	8	42
GELFOAM PACKING	8	42
HEPATECTOMY	2	11
PERIHEPATIC PACKING	1	5

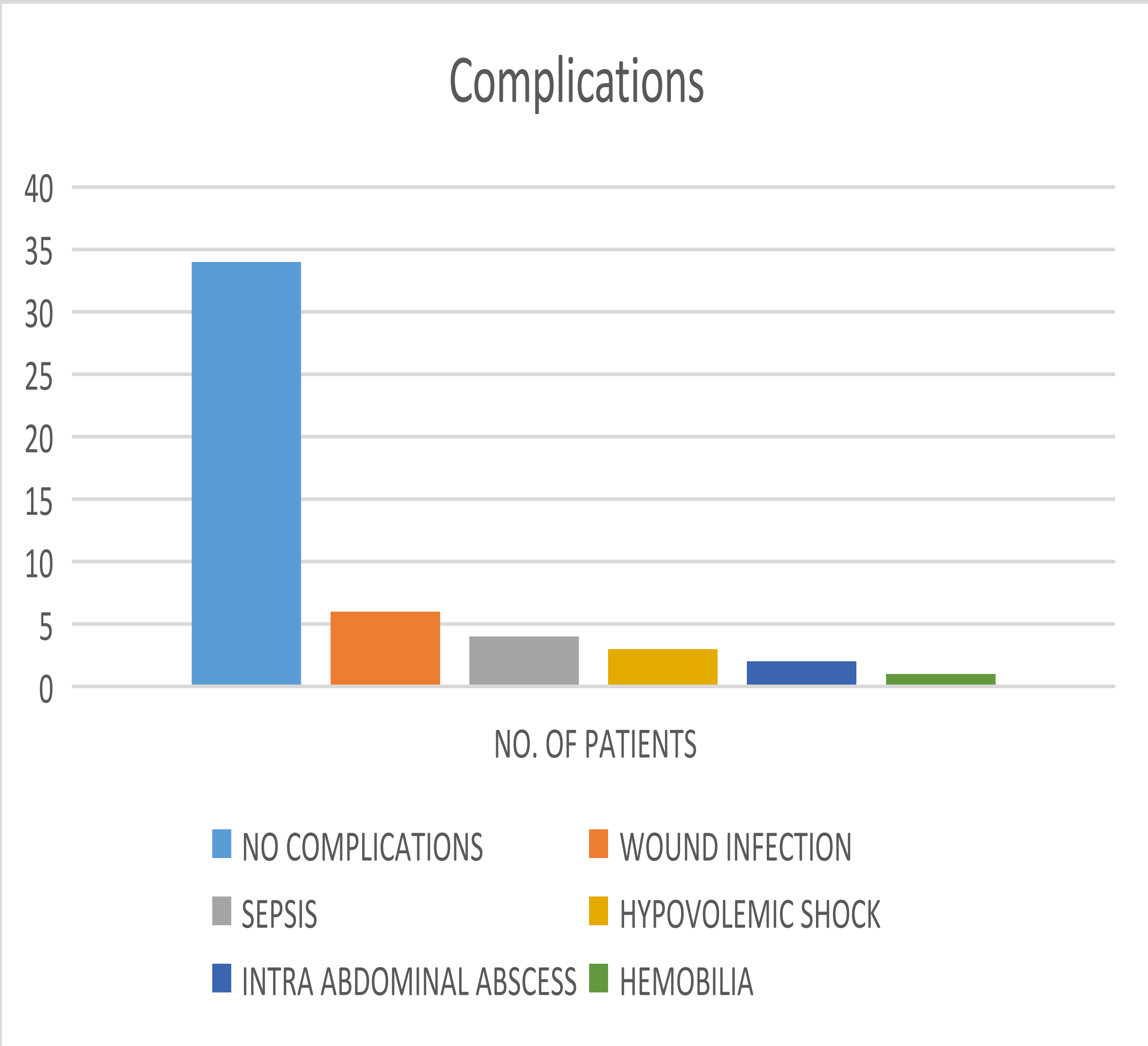
In our study ,19 patients were underwent operative procedures ,42 % were hepatorrhaphy ,42 % were gelfoam packing ,least done procedure was perihepatic packing in 5 % of patients.



COMPLICATIONS

COMPLICATIONS	TOTAL NO OF PATIENTS	PERCENTAGE %
NO COMPLICATIONS	34	68
WOUND INFECTION	6	12
SEPSIS	4	8
HYPOVOLEMIC SHOCK	3	6
INTRAABDOMINAL ABSCESS	2	4
HEMOBILIA	1	2

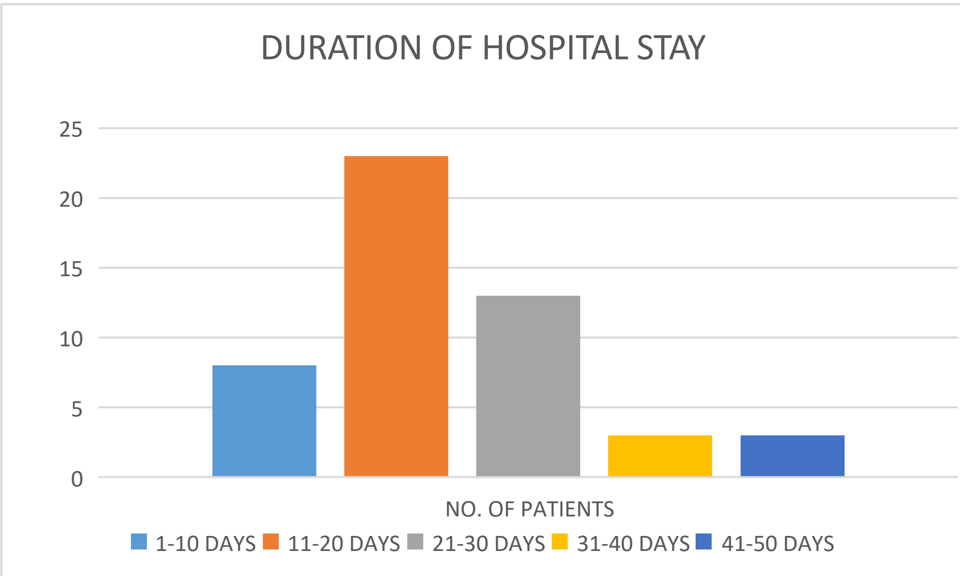
In our study ,majority of the patients (68 %) had no complications, wound infections were in 12 % of the patients, hemobilia was a least complication noted in 2 % of the patients.


Complications
40
35
30
25
20
15
10
5
0
NO. OF PATIENTS
NO COMPLICATIONS
WOUND INFECTION
SEPSIS
HYPOVOLEMIC SHOCK
INTRA ABDOMINAL ABSCESS
HEMOBILIA

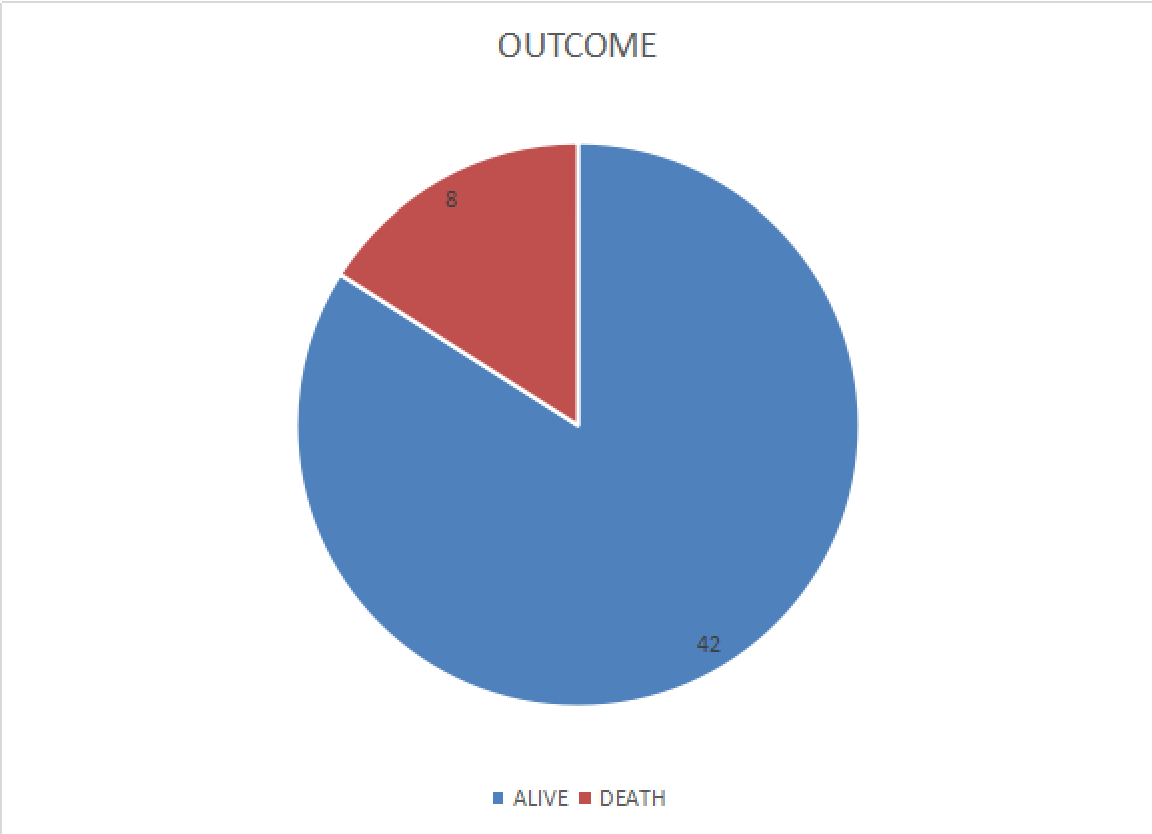
DURATION OF HOSPITAL STAY

DURATION OF HOSPITAL STAY	NO OF PATIENTS	PERCENTAGE
1-10 DAYS	8	16
11-20 DAYS	23	46
21-30 DAYS	13	26
31-40 DAYS	3	6
41-50 DAYS	3	6

In our study, 46 % of the study population were stayed in the hospital accounts 11-20 days, least of the study population 6 % were stayed in the hospital accounts 41-50 days



OUTCOME



In our study, total of 50 study population ,42 patiients were alive, 8 patients were died with the mortality rate of 16 %.

OUTCOME

outcome	No of patients	percentage
Alive	42	84
Death	8	16

DISCUSSION

SEX INCIDENCE

GENDER	OUR SERIES	STAVROS GOURGIOTIS ET AL
MALE	66 %	74.4 %
FEMALE	34%	25.6%

From the above table it is clear that males are more common victims of Liver trauma in blunt injury abdomen as in stavros gourgiotis et al .The incidence is slightly more in males as males are involved in RTA and Assaults.

MODE OF INJURY:

Road traffic accident was the most common mode of injury in our study accounting 66%,as the usage of motor vehicles are gradually increased.

X-RAY ABDOMEN ERECT VIEW:

X-ray abdomen erect was done in all the cases of our study,100 shows no evidence of associated hollow viscous Injuries.

ULTRASOUND EXAMINATION (FAST):

USG abdomen (Focussed Abdominal Sonography for Trauma) was done in all cases all the cases had liver injury. Therefore USG abdomen is more reliable in detecting liver injury and free fluid in the abdomen. Imaging is essential in early decision making. Focussed Assessment with Sonography in Trauma (FAST) examination of spaces like pericardial, perihepatic, perisplenic and pelvic region help in early detection of significant liver injury.

CT ABDOMEN & PELVIS:

CECT was done in 40 cases. Rest of patients were not affordable and some were hemodynamically unstable and were taken for exploratory laparotomy immediately.

Computed tomography (CT) can provide good information about haemoperitoneum, grade of liver injuries.

GRADE OF LIVER INJURY:

GRADE OF LIVER INJURY	OUR SERIES	STAVROS GOURGIOTIS ET AL
I	10 %	30..2%
II	30%	26.7%
III	34%	24.5%
IV	16%	15.1%
V	6%	3.5%
VI	4%	-

From the above table it is clear that low grade liver injuries accounts more than a high grade injuries as in stavros gourgiotis et al.low grade injuries favours the management towards non operative management most of the times ,also favours better outcome.

MANAGEMENT

TYPE OF MANAGEMENT	OUR SERIES	STAVROS GOURGIOTIS ET AL
NON OPERATIVE	62%	36%
OPERATIVE	16%	50%
FAILED NON OPERATIVE MANAGEMENT UNDERWENT OPERATIVE PROCEDURE	6%	14%

Compare to stavros gourgiotis et al study , our study has high percentages of non operative management about 62% excluding the failed non operative management. failed non operative management also in least percentage compare to the stavros gourgiotis et al study.

OPERATIVE PROCEDURES

PROCEDURE	OUR SERIES	STAVROS GOURGIOTIS ET AL
HEPATORRHAPHY	42%	32.6 %
PERIHEPATIC PACKING	5%	7 %
HEPATECTOMY	11%	2.3%

Above table showing that various methods of surgical procedures and its percentages in compare with stavros gourgiotis et al study. Hepatorraphy accounts 42% and perihepatic packing accounts for 15%.

BLOOD TRANSFUSION:

In our study 82 % of the cases receives blood transfusion in both in operative and non operative patients.

COMPLICATIONS:

In our study 68 % of the patients had no complications.

12 % of the patients developed wound infections.

Sepsis noted in 8 % of cases,hypovolemic shock noted in 6 % of the cases

OUTCOME:

Mortality rate in our study was 16 % ,compare to stavros gourgiotis et al study, that shows 9.3 % mortality rate.

CONCLUSION

Following conclusions are drawn from our study:

• Blunt injury abdomen with isolated liver injury forms considerable load of patients in our surgical ward and trauma casualty.

•Most common age group involved is 41-50 years. Predominantly males are affected in large numbers.

• Road traffic accident forms the most common mode of injury. So efforts should be made to bring road traffic regulations into strict action and traffic norms regulated.

• Well established trauma care centres should be established at every district hospitals and taluk hospitals. Measures should taken for early transport of the patients from the accident site to the trauma centres should be undertaken.

- Blunt injury abdomen is usually less obvious. Hence, repeated examination by multi specialty personnel in a specialized trauma care centre was required.

- X ray abdomen erect is a useful investigation to exclude associated hollow viscus injuries.

- Falling titres in serial hemoglobin and hematocrit value indicates ongoing bleeding.

- With the advent of high resolution ultrasonography (FAST), DPL and four quadrant aspiration blood investigations are not done routinely nowadays.

- CECT forms the core investigation of choice in dealing with Liver trauma in blunt injury abdomen patients ,becomes more important investigation modality for grading of liver injury and to deciding operative and conservative management.

- Early diagnosis and repeated clinical examination and use of appropriate investigations are key for managing liver trauma in Blunt abdominal injuries.

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BLUNT INJURY ABDOMEN

PROFORMA

Patient details: Case no: I P no: Unit

Name: Age: Religion:

Occupation: Address:

Injury on: Admission on:

Operated on: Discharged on:

Expired on:

Presenting complaints:

Injury: Time: Place:

Mode:- Road Traffic Accident/ Assault/ Fall

Events that followed:

History of present illness:

PAIN ABDOMEN:

Onset: Time ,mode ,Duration, Site

VOMITING :

Duration: Relation to pain: Freqency and quantity ,

Character of Vomitus

DISTENSION OF ABDOMEN :Duration/Uniform or localized/

Associated bowel disturbances/ Micturition disturbance/
Retention of
urine/ Hematuria

Any other complaints:

Associated injuries if any:

Previous history:

Personal history:

EXAMINATION:

General physical examination:

Consciousness/Hydration/Pulse/Blood pressure/Respiratory rate/ Pallor/ Decubitus/ Temperature/ Shock signs/ Icterus/ Surgical emphysema/ ENT bleeding

PER ABDOMEN EXAMINATION:

Inspection : Shape/ Skin over abdomen/ Visible injuries/ Prominent swellings/ Hernial orifice/ Movement with respiration.

Palpation: Temperature/ Tenderness/ Girth of abdomen/ Guarding/ Rigidity

Percussion: Liver dullness/ Splenic dullness/ Shifting dullness/ Fluid thrill

Auscultation :Bowel sounds/ Bruit

External genitalia:

Per rectal findings:

Associated injuries: Head and neck

Thorax: ribs, hemopneumothorax

Spine:

Pelvis:

Extremities:

Systemic examination:

Respiratory system:

Cardiovascular system:

Central nervous system:

Provisional Diagnosis:

Investigations:

U S G ABDOMEN:

Blood Grouping: serial Hb%: serial Hematocrit:

RBC: T C: DC

Urine :color: clarity: albumin:

Sugar: Microscopy:

SERUM: Amylase: Bilirubin:

Electrolytes:

X ray: Erect Abdomen/Chest/Pelvis/Spine:

Four quadrant aspiration:

Quantity/ Character/ Microscopy/ Amylase level

FAST:

CECT SCAN abdomen:

Management:

Conservative: Fluids given:

Blood transfusion:

Operative:

If operative: Date/ Started and ended/ Anesthesia/ Operative findings

Final diagnosis:

Follow up:

CONSENT FORM

I_______ Hosp No _______ in my full senses hereby give my complete for _______________or any other procedure deemed fit which is a diagnostic/therapeutic/procedure/biopsy/transfusion/operation to be performed on me/my/son/daughter/ward _________ age _________ under any anaesthesia deemed fit. The nature and risks involved in the procedure have been explained to me in my own language to my satisfaction. For academic and scientific purpose, the operation/procedure be television or photographed, or used for statistical measurements.

Date:

Signature/Thumb Impression/ of Patient/Guardian

Name:

Designation:

Guardian

Relationship

Full Address

S.NO	NAME	AGE/SEX	TYPE OF INJURY	MODE OF INJURY	HEMODYNAMIC STATUS AFTER RESUSCITATION	USG -FAST	INITIAL CECT SCAN	OPERATIVE MANAGE	LED CONSERVATIVE MANAGEME	OPERATIVE MANAGEMENT	RADE OF LIVER INJUR	BLOOD TRANFUSION	ATION OF HOSPITAL	COMPLICATIONS	ECONDARY SUTURIN	DEATH
1	MUTHUPANDI	38/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	NO	14 DAYS	NO		NO
2	AMMASI	52/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	YES	19D	NO		NO
3	TAMILARASI	41/F	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	HEPATORRHAPHY	3	YES	24D	NO	NO	NO
4	KARTHICK	22/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	YES	15D	NO		NO
5	SURULIRAJ	56/M	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	NO		4	YES	23D	SEPSIS		NO
6	PANDIYAMMAL	62/F	BLUNT INJURY	ASSAULT	UNSTABLE	DONE	NOT DONE		NO	HEPATECTOMY	6	YES	2D	HYPOVOLOMIC SHOCK	NO	YES
7	KAVIN	19/M	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		1	NO	12D	NO		NO
8	PAUNRAJ	47/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	10D	NO		NO
9	PANDIKUMAR	27/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	GELFOAM PACKING		5	YES	43D	NO	NO	YES
10	NIZAM	41/M	BLUNT INJURY	ASSAULT	UNSTABLE	DONE	NOT DONE	–	NO	GELFOAM PACKING	3	YES	21D	NO	NO	NO
11	PALPANDI	48/M	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE	–	NO	HEPATORRHAPHY	3	YES	35D	INTRAABDOMINAL ABSCESS	YES	YES
12	VELUSAMY	56/M	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		2	YES	18D	NO		NO
13	RAJA	71/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		1	NO	9D	NO		NO
14	MANIMARAN	43/M	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	HEPATORRHAPHY		5	YES	47D	SEPSIS	NO	NO
15	PANDISELVI	27/F	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	GELFOAM PACKING	3	YES	35D	WOUND INFECTION	YES	NO
16	ANTHONYSAMY	37/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	YES	17D	NO		NO
17	PUSHPA	30/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	GELFOAM PACKING		3	YES	27D	NO		NO
18	MURUGAN	51/M	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	HEPATORRHAPHY	4	YES	19D	SEPSIS	NO	YES
19	RATHINAM	43/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	NO	12D	NO		NO
20	ARUMUGAM	28/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	16D	NO		NO
21	SRIDHAR	42/M	BLUNT INJURY	RTA	UNSTABLE	DONE	DONE		NO	PERIHEPATIC PACKING	5	YES	3D	HYPOVOLOMIC SHOCK	NO	YES
22	MEENA	48/F	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		2	YES	9D	NO		NO
23	KARUPPUSAMY	17/M	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	NO		2	YES	12D	NO		NO
24	PAPATHY	56/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	22D	NO		NO
25	VELMURUGAN	26/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	NO	18D	NO		NO
26	SANGEETHA	31/F	BLUNT INJURY	ACCIDENTAL FALL	UNSTABLE	DONE	DONE		NO	HEPATORRHAPHY	4	YES	29D	WOUND INFECTION	YES	NO
27	GOKULAKRISHNAN	19/M	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	HEPATORRHAPHY	4	YES	17D	NO	NO	NO
28	SELVARAJ	31/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	13D	NO		NO
29	NARMATHA	43/F	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	NO		1	NO	16D	NO		NO
30	KANNAMAL	53/F	BLUNT INJURY	ACCIDENTAL FALL	UNSTABLE	DONE	NOT DONE		NO	HEPATECTOMY	6	YES	1D	HYPOVOLOMIC SHOCK	NO	YES
31	RAMUTHAI	60/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	NO	14D	NO		NO
32	MUTHUSUDHA	25/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		4	YES	26D	NO		NO
33	RAJESWARAN	38/M	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		1	YES	8D	NO		NO
34	PANDI	59/M	BLUNT INJURY	RTA	UNSTABLE	DONE	DONE		NO	GELFOAM PACKING	3	YES	23D	WOUND INFECTION	NO	NO
35	VEERAPANDI	50/M	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	NO		3	YES	26D	HEMOBILIA		NO
36	PADMAVATHI	33/F	BLUNT INJURY	ASSAULT	STABLE	DONE	DONE	YES	NO		2	YES	19D	NO		NO
37	VELLAIPANDI	50/M	BLUNT INJURY	RTA	UNSTABLE	DONE	DONE		NO	GELFOAM PACKING	3	YES	31D	WOUND INFECTION	YES	YES
38	VEERAMMAL	44/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	YES	11D	NO		NO
39	MARIMUTHU	63/M	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	GELFOAM PACKING	4	YES	21D	WOUND INFECTION	YES	NO
40	PALPANDI	56/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	12D	NO		NO
41	KAMATCHI	40/F	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		2	NO	21D	NO		NO
42	NISHA	33/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	14D	NO		NO
43	SADIQ BASHA	47/M	BLUNT INJURY	RTA	STABLE	DONE	DONE		NO	GELFOAM PACKING	4	YES	22D	WOUND INFECTION	NO	NO
44	NEEDHI	45/M	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		1	YES	7D	NO		NO
45	CHELLAMAL	56/F	BLUNT INJURY	RTA	UNSTABLE	DONE	DONE		NO	HEPATORRHAPHY	4	YES	25D	SEPSIS	NO	YES
46	MOHAMED KANI	69/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		3	YES	12D	NO		NO
47	DURAIPANDI	37/M	BLUNT INJURY	ACCIDENTAL FALL	STABLE	DONE	DONE	YES	NO		3	YES	17D	NO		NO
48	KAMALA	38/F	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	YES	17D	NO		NO
49	SEETHA	45/F	BLUNT INJURY	RTA	UNSTABLE	DONE	NOT DONE		NO	HEPATORRHAPHY	3	YES	41D	INTRAABDOMINAL ABSCESS	NO	NO
50	MANIKANDAN	29/M	BLUNT INJURY	RTA	STABLE	DONE	DONE	YES	NO		2	NO	11D	NO		NO

MADURAI MEDICAL COLLEGE
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(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.P.Pugazhenthi
Designation	:	PG in M.S., General Surgery
Course of Study	:	2017- 2020
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Prospective study of Management and outcomes of liver trauma in blunt injury abdomen cases in GRH, Madurai
Ethical Committee as on	:	25.04.2019

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean // Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

DEAN
Madurai Medical College
Madurai-20

03 MAY 2019

URKUND

Urkund Analysis Result

Analysed Document: pugazh theory NEW.doc (D57192128)
Submitted: 17/10/2019 19:00:00
Submitted By: pugazhsanth92@gmail.com
Significance: 1 %

Sources included in the report:

liver abscess.docx (D42439520)
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Instances where selected sources appear:

3

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation work titled "**PROSPECTIVE STUDY OF MANAGEMENT AND OUTCOMES OF LIVER TRAUMA IN BLUNT INJURY ABDOMEN CASES IN GRH, MADURAI**" of the candidate **DR. P. PUGAZHENTHI** with registration number **221711120** for the award of **MASTER DEGREE** in branch of **GENERAL SURGERY.**I have personally verified the urkund.com website for plagiarism check.I found that the uploaded thesis file contains all from introduction to conclusion pages and the result shows **ONE percentage** of plagiarism in the dissertation.

Guide and supervisor sign with seal