“A CROSS SECTIONAL STUDY OF IMAGE GUIDED PERCUTANEOUS DRAINAGE OF INTRA - ABDOMINAL ABSCESSES, OF PATIENT ADMITTED TO DR. PRABHAKAR KORE HOSPITALS AND MRC BELGAUM”

REG NO. BH0109009

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
GENERAL SURGERY

DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

APRIL - 2013

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ENDORSEMENT

This is to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY OF IMAGE GUIDED PERCUTANEOUS DRAINAGE OF INTRA -ABDOMINAL ABSCESSES, OF PATIENT ADMITTED TO DR. PRABHAKAR KORE HOSPITALS AND MRC BELGAUM"** is a bonafide research work done by **THE CANDIDATE REG NO. BH0109009**.

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LIST OF ABBREVIATIONS USED

ALD	-	Alcoholic Liver Disease
CBD	-	Common Bile Duct
CT	-	Computed Tomography
GIT	-	Gastro Intestinal Tract
HTN	-	Hypertension
I.V	-	Intra Venous
IAA	-	Intra Abdominal Abscess
IL-1	-	Inter leukin-1
PCD	-	Percutaneous Drainage
PID	-	Pelvic Inflammatory Disease
T2DM	-	Type 2 Diabetes Mellitus
TNF	-	Tumour Necrosis factor
USG	-	Ultra Sonography
UTI	-	Urinary Tract Infection

ABSTRACT

Background and objectives

Intra-abdominal abscess continues to be an important and serious problem in surgical practice. Management of intra-abdominal abscess consists of drainage of abscesses which can be percutaneous drainage or surgical drainage. The present study was aimed to assess the feasibility of percutaneous drainage as the primary method to treat intra abdominal abscess through image guided diagnosis.

Methodology

This one year cross sectional study was conducted on a total of 40 patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Data obtained was tabulated on a excel spread sheet analysed.

Results

In the present study the commonest (30%) age group was 36 to 50 years (30%) study. Single abscess was seen in 82.5% of patients whereas 17.5% had multiple abscesses. In the present study 77.5% cases underwent computed tomography and 22.5% underwent ultrasonography. 22.5% cases underwent bedside procedure whereas 77.5% in procedure room. The mean drainage was recorded as 31.15 ± 16.35 mL. On day one was 34.18 ± 32.49 mL, on day two it was 15.68 ± 9.24 mL and on day three 11.68 ± 8.22 mL. The commonest organisms were staph aureus 45.16%, E. coli 38.70%, streptococcus pyogenes 9.67% and klebesialla pneumoniae 6.45%. Overall the mean duration of hospital

stay was 11.08 ± 6.32 days. Of the 40 patients with abscess 39 (97.5%) improved and one patient (2.5%) expired.

Conclusion and interpretation

Percutaneous drainage is safe, effective and feasible treatment option for the management of intra abdominal abscesses. CT being the most sensitive imaging modality for visualizing intra abdominal abscesses should be used to guide percutaneous drainage.

Keywords

Image guided drainage; Intraabdominal abscess; Percutaneous drainage;

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Chapter 1

Introduction

INTRODUCTION

Intra-abdominal abscess continues to be an important and serious problem in surgical practice. Appropriate treatment is often delayed because of the obscure nature of many conditions resulting in abscess formation, which can make diagnosis and localization difficult. Associated patho physiologic effects may become life threatening or lead to extended periods of morbidity with prolonged hospitalization. Delayed diagnosis and treatment can also lead to increased mortality rates; therefore, the economic impact of delaying treatment is significant.

A better understanding of the patho physiology and a high clinical index of suspicion should allow earlier recognition, definitive treatment, and thus reduces morbidity and mortality.[1]

Diagnoses of intra-abdominal abscesses is often difficult as compared to peritonitis. The clinical results of management of patients after operation for the acute peritonitis yields three typical outcomes namely, peritonitis process may resolve, Fulminant peritonitis – septic shock syndrome and finally, a relative biologic "STAND OFF" may merge between host and the invading pathogens – leading to abscess formation.

Whereas diagnosis of acute peritonitis is relatively simple, the diagnosis of abscess is quite difficult especially in the post-operative patient. The infection is contained within a perimeter wall of fibrin and collagen and doesn't provide physical finding of rebound tenderness. Most common clinical features of intra abdominal abscess consist of fever, pain abdomen, lump or mass per abdomen. In

a study of 143 patient by Fry DE et al.[2] (Surgically proven intra abdominal abscess), only one third of patient did have localized tenderness and a palpable mass in less than <10% of cases.

X-ray features of intra-abdominal abscess include raised diaphragms pleural effusion, basal atelectasis, extra luminal gas, localized ileus, soft tissue mass. In a study by Fry DE et al.[2] have shown that abdominal roentgenography was of value in only 15% of patients.

USG and CT scan abdomen in the experienced hand has accuracy in excess of 90%. USG advantages are portability, rapidity and no exposure to radiation. Disadvantages include relatively expensive, obesity, dressing, colostomies, air filled bowel loops and overlying lung can result in unsatisfactory scan. CT scan provides radiological laparotomy, CT scan has sensitivity >90% in detecting IAA.

Radionuclide scanning (Using Gallium 67 and Indium 111). Radionuclide scanning is most likely to be useful for patients with non localized signs of sepsis. Not very useful in post-operative patients as isotope is taken up by all sites of inflammation and high chance of false positivity. The 24-48 hrs delay before the results of radio nuclide scanning is a serious disadvantage.

Management of intra-abdominal abscess consists of drainage of abscesses which can be percutaneous drainage or surgical drainage. Percutaneous drainage consists of either USG guided or CT guided drainage, because abscesses contain high concentrations of bacteria in an environment that is hostile to the biologic

action of most antibiotic therapy, drainage of the intra-abdominal abscess is essential for patient recovery.

The abscesses that have been most amenable to treatment by the percutaneous route are unilocular, although communicating septated or multilocular abscesses may be successfully treated percutaneously. Von-Sonnerberg E et al.[3] showed success rate of 83.6% after PCD. Gerzof SG et al.[4] showed success rate of 96% after PCD. Failure in remaining case was mostly because of enteric communication underlying tumor, multiplicity of abscesses, multi loculated abscesses.

Primary surgical drainage is indicated in cases where abscess is poorly defined or difficult to localize by imaging techniques, abscess material is too viscous and extensive necrotic debris present, underlying malignancy, contraindication for PCD, associated with fistulae/obstruction, failure of PCD. Open surgical drainage is associated with complication rate of 20-30% as by Alan Hemming et al.[5] and mortality of 21% as by Gerzof SG et al.[4]

The antibiotic regimens recommended according to current treatment options if infectious diseases and surgical infectious disease are β lactam/β lactamase inhibitors like amoxicillin-sulbactam, piperacillin tazobactam combination+metronidazole, aminoglycoside+metronidazole and fluoroquinolone +metronidazole.

Hence the present study was undertaken to assess the feasibility of percutaneous drainage as the primary method to treat intra abdominal abscess through image guided diagnosis.

Chapter 2

Objectives

OBJECTIVES

The objective of the present study was to assess the feasibility of percutaneous drainage as the primary method to treat intra abdominal abscess through image guided tube drainage.

Chapter 3

Review of Literature

REVIEW OF LITERATURE

Historical Review

Since intra-abdominal abscess was first described over 200 years ago, its clinical picture and epidemiology have changed. Its relatively more common due to number of surgeries done for various GIT pathologies especially anatomical leak following surgery. Modern medical techniques such as improved hygiene and introduction of antibiotics have been most effective in the field of infectious diseases and consequently, intra-peritoneal abscess as sequelae to the spread of infection into the peritoneal cavity has become steadily less common. On the other hand iatrogenic abscess following elective operations such as cholecystectomy, resection with anastomosis of bowel, various drainage procedures has come into picture. Indeed the history of intra-peritoneal abscess reflects the advance of modern surgical practice. It can be divided into four phases.[6]

In the first phase during eighteenth and early nineteenth century intra peritoneal abscess (Subphrenic abscess) was a curious autopsy finding whose clinical significance was uncertain probably the first subphrenic abscess in the autopsy room was described by Petit in 1873 in a patient with perforated colon. During the next 100 years, postmortem reports of subphrenic abscess became increasingly frequent.[6]

During second phase which started in 1845 when Barlow gave a detailed description of the autopsy finding, subphrenic abscess was recognized in vivo, but nothing could be done and the patients usually perished. Curiously after

Barlow's first description the condition was not at all recognized in a living patient until 1877; in 1879, Leyden described three cases under the title 'subphrenic pyopneumothorax'.[6]

The third phase that surgical intervention began in 1881 when Von Volkoman deliberately treated a subphrenic abscess and saw a gratifying improvement in the patients condition. In 1908 Barnard[1] from the London hospital published the first extensive studies.[6]

Amongst Barnard's 78 cases, 29 (38%), following perforation of peptic ulcer, 21 were gastric ulcer perforation. Of Piqnuard's 890 cases, 251 (30%) were of gastric origin. In both series subphrenic abscess following a ruptured gangrene appendix was not such as a common cause, accounting for 16% of cases.[6]

The fourth phase occurred when the whole picture of IAA abscess was changed by the widespread usage of antibiotics in the year following 1945 ,the largest series in 1964, 128 cases by Carter and Brewer collected cases over 20 years. The extent of the decrease in the number in relation to patient at risk was difficult to analyze. Halliday and Laventhal (1964),[7] quote the incidence of 0.5% of subphrenic abscess during the course of approximately 13000 major abdominal surgeries.[6]

Extensive study done by Lorber B. et al (1974)[8] showed that E. Coli and enterococci were the commonest organisms during peritonitis while bacteriodes and with E coil, enterococci were commonest organisms found in intra-abdominal abscess. Their study also showed remarkable ability of encapsulated anaerobic bacteria to produce abscess is attributed to the capsular polysaccharide

components. Their study also showed the poly microbial infection, specially combination of aerobic and anaerobic which exhibit significant greater lethality than the single species. The subsequent studies in 1996 for Solomin JS et al[9] showed the same features.

In 1990, Upond MN et al.[10] found out that the development of an intra abdominal abscess is due to culmination of sequestration process with the most credible objective of isolating or containment of bacterial contamination and thereby preventing widespread dissemination. That is due to fibrin deposition in excess of fibrin degradation produced by bacterial enzymes, fibrinolysin and phagocytosin.

Rostein OD et al[11] observed that high spiking fever mild localized abdominal pain, anorexia and weight loss can compare the classic presentation of an intra-abdominal abscess. However clinical findings vary with the site of abscess, and presentation is usually non-specific. According to other study, localized tenderness or palpable mass was found in about 50% cases by thorough clinical examination.

Hiyama DT et al[12] (1995) observed that ultrasound scan and CT scan of abdomen have become mainstay in the diagnosis and eventual percutaneous drainage of intra peritoneal abscess ultrasound scan has 90% diagnostic accuracy and CT scan 95% in experienced hands with the added advantage of differentiating intra peritoneal and retroperitoneal structures without much bowel gas shadow, dressing or drain and offers opportunity of better percutaneous drainage.

The advancement of interventional radiology[13] has drastically helped the surgeons to combat the intra peritoneal abscess which initially were associated with operative morbidity and mortality.

Anatomy of peritoneum[14]

Peritoneum (Tunica Serosa) The peritoneum is the largest serous membrane in the body, and consists in the male of a closed sac, a part of which is applied against the abdominal parietes, while the remainder is reflected over the contained viscera. In the female the peritoneum is not a closed sac, since the free ends of the uterine tubes open directly into the peritoneal cavity. The part which lines the parietes is named the parietal portion of the peritoneum, that which is reflected over the contained viscera constitutes the visceral portion of the peritoneum. The free surface of the membrane is smooth, covered by a layer of flattened mesothelium, and lubricated by a small quantity of serous fluid. Hence the viscera can glide freely against the wall of the cavity or upon one another with the least possible amount of friction. The attached surface is rough, being connected to the viscera and inner surface of the parietes by means of areolar tissue, termed the sub serous areolar tissue. The space between the parietal and visceral layers of the peritoneum is named as the peritoneal cavity, but under normal conditions this cavity is merely a potential one, since the parietal and visceral layers are in contact. The peritoneal cavity gives off a large diverticulum, the omental bursa, which is situated behind the stomach and adjoining structures, the neck of communication between the cavity and the bursa is termed the epiploic foramen (foramen of Winslow).

Vertical disposition of the main peritoneal cavity (greater sac)

It is convenient to trace this from the back of the abdominal wall at the level of the umbilicus. On following the peritoneum upward from this level it is seen to be reflected around a fibrous cord, the ligamentum teres (obliterated umbilical vein), which reaches from the umbilicus to the under surface of the liver. This reflection forms a somewhat triangular fold, the falciform ligament of the liver, attaching the upper and anterior surfaces of the liver to the diaphragm and abdominal wall.

Covering the upper and anterior surfaces of the liver, it is continued around its sharp margin on to the under surface, where it presents the following relations: a) It covers the under surface of the right lobe and is reflected from the back part of this on to the right suprarenal gland and upper extremity of the right kidney, forming in this situation the inferior layer of the coronary ligament, a special fold in the hepato renal ligament, is frequently present between the inferior surface of the liver and the front of the kidney. From the kidney it is carried downward to the duodenum and right colic flexure and toward in front of the inferior vena cava, where it is continuous with the posterior wall of the omental bursa. Between the two layers of the coronary ligament there is a large triangular surface of the liver devoid of peritoneal covering, this is named the bare area of the liver and attached to the diaphragm by areolar tissue.

Towards the right margin of the liver the two layers of the coronary ligament gradually approach each other and ultimately fuse to form a small

triangular fold connecting the right lobe of the liver to the diaphragm and named the right triangular ligament of the liver

It covers the lower surface the quadrate lobe, the under and lateral surfaces of the gall-bladder and the under surface and posterior border of the left lobe, it is then reflected from the upper surface of the left lobe to the diaphragm as the inferior layer of the left triangular ligament and from the porta hepatis of the liver and the fossa for the ductus venosus to the lesser curvature of the stomach and the first 2.5cm of the duodenum as the anterior layer of the hepatogastric and hepatoduodenal ligament which together constitute the lesser omentum.

If this layer of the lesser omentum be followed to the tight it will be found to turn around the hepatic artery, bile duct and portal vein and become continuous with the anterior wall of the omental bursa forming a free folded edge of peritoneum. Traced downward, it covers the antero superior surface of the stomach and the commencement of the duodenum and is carried down into a large free fold known as gastrocolic ligament or greater omentum. Reaching the free margin of this fold, it is reflected upward to cover the under and posterior surfaces of the transverse colon, and thence to the posterior abdominal wall as the inferior layer of the transverse mesocolon.

It reaches the abdominal wall and traverses over the head of pancreas and then over the inferior surface of the pancreas on the superior mesenteric vessels and then to the small intestine as the anterior layer of the mesentery. It encircles the intestine and subsequently may be traced as the posterior layer of the

mesentery, upward and backward to the abdominal wall. From this it sweeps down over the aorta into the pelvis where it invests the sigmoid colon, its reduplication forming the sigmoid mesocolon. Leaving first the sides and then the front of the rectum, it is reflected on to the seminal vesicles and fundus of the urinary bladder and after covering the upper surface of that viscus is carried along the medial and lateral umbilical ligaments on to the back of the abdominal wall to the level from which a start was made.

Vertical disposition of the peritoneum.

Between the rectum and the bladder,in the male it forms a pouch the recto vesical excavation, the bottom of which is slightly below the level of the upper ends of the vesiculae seminales- i.e about 7.5cm from the orifice of the anus. When the bladder is distended the peritoneum is carried up with the expanded viscus so that a considerable part of the anterior surface of the latter lies directly against the abdominal wall without the intervention of peritoneal membrane (prevesical space of Retzius). In the female the peritoneum is reflected from the rectum over the posterior vaginal fornix to the cervix and body of the uterus forming the rectouterine excavation (pouch of Douglas).

Vertical disposition of the omental bursa (Lesser peritoneal sac)

A start may be made in this case on the posterior abdomen at the anterior border of the pancreas. From this region, the peritoneum may be followed upwards over the pancreas on to the inferior surface of the diaphragm and then on to the caudate lobe and caudate process of the liver to the fossa from the ductus venosus and the porta of the liver. Traced to the right, it is continuous over the

inferior vena cava with the posterior wall of the main cavity. From the liver it is carried downward to the lesser curvature of the stomach and the commencement of the duodenum as the posterior layer of the lesser omentum and is continuous on the right around the hepatic artery, bile duct and portal vein with the anterior layer of this omentum. The posterior layer of the lesser omentum is carried down as a covering for the postero-inferior surfaces of the stomach and commencement of the duodenum and is continued downward as the deep layer of the gastrocolic ligament or greater omentum. From the free margin of this fold it is reflected upward on itself to the anterior and superior surfaces of the transverse colon, and then as the superior layer of the transverse mesocolon to the anterior border of the pancreas the level from which a start was made .

Horizontal disposition of the peritoneum

Below the transverse colon, the arrangement is simple as it includes the main cavity, above the level of the transverse colon it is more complicated on account of the existence of the omental bursa. Below the transverse colon it may be considered in the two regions, viz in the pelvis and in the abdomen proper.

The peritoneum of the male pelvis

In the pelvis

The peritoneum here follows closely the surface of the pelvic viscera and the inequalities of the pelvic walls and presents important differences in two sexes a) In the male is encircles the sigmoid colon from which it is reflected to the posterior wall of the pelvis as a fold, the sigmoid mesocolon. It then leaves

the sides and finally the front of the rectum and is continued on to the upper sides and finally the front of the rectum and is continued on to the upper ends of the seminal vesicles and the bladder, on either side of the rectum it forms a fossa the pararectal fossa, which varies in size with the distension of the rectum. In front of the rectum the peritoneum form the recto vesicle excavation which is limited laterally by peritoneal folds extending from the sides of the bladder to the rectum and sacrum. These folds are known from their position as the recto vesical or sacrogenital folds.

In female, pararectal and paravesical fossa similar to those in the male are present: the lateral limit of the paravesical fossa is the peritoneum investing the round ligament of the uterus . The recto vesical excavation is, however divided by the uterus and vagina into a small anterior vesico uterine and a large deep posterior recto uterine excavation.

Main cavity

Commencing on the posterior abdominal wall at the inferior vena cava, the peritoneum may be followed to the right over the front of the suprarenal gland and upper part of the right kidney on to the antero-lateral abdominal wall. From the middle line of the anterior wall a backwardly directed fold encircles the obliterated umbilical vein and forms the falciform ligament of the liver.

Omental bursa (bursa omentum , lesser peritoneal sac)

It is bounded in front from above downward by the caudate lobe of the liver, the lesser omentum of the stomach and the anterior two layers of the greater

omentum. Behind it is limited from below upward by the two posterior layers of the greater omentum, the transverse colon and the ascending layer of the transverse mesocolon the upper surface of the pancreas the left suprarenal gland and the upper end of the left kidney. To the right of the esophageal opening of the stomach it is formed by that part of the diaphragm which supports the caudate lobe of the liver. Laterally the bursa extends from the epiploic foramen to the spleen where it is limited by the phrenicolienal and gastrolienal ligaments.

Numerous peritoneal folds extend between the various organs or connect them to the parietes, they serve to hold the viscera in position and at the same time enclose the vessels and nerves proceeding to them. They are grouped under the three headings of ligaments, omentum and mesenteries.

There are two omentum, the lesser and the greater. The lesser omentum (omentum minus, small omentum gastro hepatic omentum) is the duplicator which extends to the liver from the lesser curvature of the stomach and the commencement of the duodenum. It is extremely thin, and is continuous with the two layers of peritoneum which cover respectively the antero-superior and postero-inferior surfaces of the stomach and first part of the duodenum. When these two layers reach the lesser curvature of the stomach and the upper border of the duodenum they join together and ascend as a double fold to the porta of the liver, to the left of the porta the fold is attached to the bottom of the fossa for the ductus venosus, along which its carried to the diaphragm, where the two layers separate to embrace the end of the esophagus. The portion of the lesser omentum extending between the liver and stomach is termed the hepatogastric ligament, while the liver and duodenum is the hepatoduodenal ligament. Between the two

layers of the lesser omentum, close to the right free margin are the hepatic artery the common bile duct the portal vein, lymphatics and the hepatic plexus of nerves all these structures being enclosed in a fibrous capsule (Glisson's capsule).

The greater omentum (omentum majus, great omentum , gastrocolic omentum) is the largest peritoneal fold. It consists of a double sheet of peritoneum, folded upon itself so that it is made up of four layers. The two layers which descend from the stomach and commencement of the duodenum pass in front of the small intestine, sometimes as low down as the pelvis, they then turn upon themselves and ascend again as far as the transverse colon, where they separate and enclosed that part of the intestine. The left border of the greater omentum is continuous with the gastrolienal ligament, its right border extends as far as the commencement of the duodenum.

Physiology of the peritoneal cavity[12]

The peritoneum is a single layer of mesothelial cells with a basement membrane supported by a underlying layer of highly vascularized connective tissue. Though thin the surface area of the peritoneum is extensive averaging to $1.8m^2$ in the adult male and is comparable to the surface area of the skin. It has been estimated that 1mm increase in the thickness of the peritoneum by fluid accumulation can result in the sequestration of 18L of fluid, a fact relevant to the massive fluid shifts associated with diffuse peritonitis.[15]

Under normal conditions, < 50 ml of sterile fluid is present within the peritoneal cavity. The fluid itself closely resembles lymph fluid and has a low specific gravity, protein content and < 3000 cells per cubic mm. secreted from the

visceral peritoneal surfaces the fluid is circulated through the peritoneal cavity. Contrast material introduced into the peritoneal cavity in the paracecal area primarily transmigrates towards the right subphrenic area primarily transmigrates towards the right subphrenic area and into the pelvis.[16]

The cephalic movement proceeds along the paracolic gutter and subhepatic spaces. It is though that the cephalic movement of fluid is produced by the creation of a negative pressure area in the subphrenic space by diaphragmatic motion. Most of the peritoneal fluid is absorbed into the lymphatic circulation via the parietal peritoneal surfaces with the remainder absorbed through diaphragmatic lymphatics.[17]

The clearance of particulate matter cells and microorganisms contained in peritoneal fluid may be largely dependent upon diaphragmatic lymphathics. Localized to the peritoneum overlying the muscular portion of the diaphragmatic surface, intercellular gaps or stomata are situated between peritoneal mesothelial cells. The diameter of these stomata can be varied by diaphragmatic stretching and contraction from 4 to 12µm. Both fluid and substances not amenable to absorption through the peritoneal membrane are channeled via the stomata through fenestrations in the basement membrane and conveyed to specialized diaphragmatic lymphatics called lacunae. During the respiratory cycle, relaxation of the diaphragm in expiration opens the stomata and promotes rapid filling of the lacunae.[17]

Maddaus has suggested that the primary role of this mechanism is fluid removal from the peritoneal cavity and not the elimination of microorganisms. It

is possible that the carriage of bacteria into the systemic circulation may overwhelm systemic defenses, such as the reticu lo endothelial system, leading to the high incidence of bacteremia associated with peritonitis.

The second clearance mechanism is by phagocytosis by resident peritoneal macrophages. Dunn and associate have noted that in animals half of the bacteria in an intra peritoneal inoculum are cleared physically via the diaphragmatic lymphatics and another half undergo phagocytosis by resident macrophages. These two highly efficient mechanisms probably represent the first line of clearance after bacterial contamination.[18]

Anatomy of subphrenic spaces[14]

The undersurface of the diaphragm is marked out into four peritoneal spaces and two cellular ones. The four peritoneal spaces are separated from one another by the cruciform arrangement of the ligaments of the liver namely, the coronary, falciform and the right and left lateral ligaments. The falciform ligament divides the subphrenic space into two parts right and left. Each of these again are subdivided into a larger anterior and smaller posterior part by the corresponding lateral ligament. The four peritoneal subphrenic fossae may therefore be classified and named as follows:

A. Intra peritoneal

I. Right 1. Anterior

2. Posterior

II. Left 1. Anterior

2. Posterior

B. Extra peritoneal

1. Right

2. Left

Paracolic gutters

These potential spaces lay between the body wall and on the right, the descending colon segments. On the left, communication between the gutter and the subphrenic space is limited by the phrenocolic. Inferiorly communication with the pelvis is prevented by the paracolic gutter and the right subphrenic and subhepatic spaces, as well as pelvis.

Lesser sac

This space lies posterior to the stomach and gastrohepatic ligament. Superiorly the space extends behind the caudate lobe of the liver and inferiorly to the transverse mesocolon. The anterior surface of the pancreas forms much of the posterior border of the lesser sac. Despite the free communication of the lesser and greater sacs through the foramen of Winslow.

Pathophysiology of abscess formation

The objective of the local response to infection is the removal or containment of microorganisms from the peritoneal cavity. The inflammatory

response that occurs within the peritoneal cavity is similar to inflammation that occurs elsewhere in the body. This response characterized by hyperemia the influx of fluid the recruitment of phagocytes and fibrin deposition.

Any noxious stimulus that causes mesothelial or vascular endothelial cell injury is capable of initiating peritonitis. Though endotoxin associated with gram-negative bacteria is considered the classical stimulating agent of peritonitis based upon experimental models of peritonitis, a number of other agents are recognized as capable of inducing similar responses. Wiles has noted that local physiological effects similar to those elicited by endotoxin can be produced by organisms such as gram-positive bacteria, Bacteroids species and yeasts that have no endotoxin or only biologically inactive forms of endotoxin.[19]

This implies that other bacterial products such as exo enzymes or capsular polysaccharides also act as stimulators of inflammation. In addition the similarity in systemic response to both anaerobic gram-negative and fungal peritonitis namely fever, hypotension, leukocytosis, platelet aggregation and shock, suggests that the systemic action of these stimulators is also not direct, but mediated by cytokines such as tumor necrosis factor (TNF) and interleukin-1 (IL-).[20-21]

Alterations in blood flow and vascular permeability

The release of histamine is triggered by mesothelial cell injury, though later, antigen-antibody complexes, complement-derived C3a and CA and platelet activating factors also stimulate histamine release. Both histamine and bradykinin cause pain, vasodilatation and increased permeability of the small peritoneal blood vessels. The production of other vasoactive substances such as

prostaglandin E2a (PGE2∞) and leukotriene C4 (LTC4) are effected by bradykinin and contribute to the observed vascular changes. Normally the peritoneum allows bi-directional flow of fluid, but with inflammation there is an unidirectional influx of extracellular fluid into the peritoneal cavity. Depending upon the extent of the peritoneal insult, fluid volumes of 10 L or more may be accumulated in the peritoneal cavity.[22]

Bacterial phagocyotsis

The central purpose of the local accumulation and interaction of the humoral mediators is the initiation and enhancement of bacterial phagocytosis. The recruitment and accumulation of large numbers of leukocytes to the site of inflammation is accomplished by changes in local blood flow as well as increased migration and adherence of leukocytes to surfaces such as endothelial or mesothelial cells. Bradykinin, anaphylatoxins, platelet activating factors (PAF), TNF and IL-1 appear to promote these effects.[23]

Fibrin deposition

Under normal circumstances, intact mesothelial cells maintain fibrinolytic activity within the peritoneal cavity by the secretion of tissue plasminogen activator (t-PA). In the setting of mesothelial cell injury and active inflammation, local fibrinolytic activity is suppressed owning to the loss of plasminogen activator. With high concentrations of fibrinogen present, fibrin deposition readily occurs by activation of the intrinsic pathway

Fibrin deposition appears to play a role in the local inflammatory response. The most credible objective of this process is to isolate or contain contamination and thereby prevent widespread dissemination. At one level, bacteria are entrapped in fibrin matrices. At a larger level, these omentum adhere to one another as well as parietal peritoneal surface, thus creating physical barriers against the spread of bacterial contamination.

Abscess formation

The development of an intra peritoneal abscess is the culmination of the sequestration process described above. The rate of fibrin deposition exceeds the fibrin degradation produced by bacterial enzymes, fibrinolysis and phagocytosis. Within the adhered mass of viscera, fibrin and bacteria, liquefaction develops from the release of proteolytic enzymes from perishing leukocytes and the action of bacterial exoenzymes. The osmolarity of the developing abscess fluid is high, promoting an influx of water and increasing the internal hydrostatic pressure within the cavity.[15]

Systemic response to peritoneal injury or infection

The development of hypovolemia is a phenomenon central to the systemic response and probably results from the influx occurring in the peritoneal cavity. The subsequent intravascular volume change leads to a reduction in venous return and cardiac output. Incomplete compensation is obtained with increase in heart rate. Systemic hypotension also may be the result of the secretion of TNC, IL-1, platelet activating factors, and nitric oxide, which all have vasodilator effects and may reduce systemic vascular resistance.[20-21]

In particular, a significant degree of precapillary shunting may occur in the pulmonary and splanchnic circulation, which leads to a reduction in oxygen delivery and subsequent consumption. Diminished urine flow develops as a result of the effects of increased aldosterone and antidiuretic hormone secretion the reduced cardiac output, and intrarenal shunting of blood.

Pulmonary function also is altered in this setting. Abdominal distention secondary to accumulated fluid within the peritoneal cavity and bowel creates mechanical restriction to diaphragmatic mobility and decreases ventilatory volume, creating eventual atelectasis. Ventilation perfusion mismatching results from both the atelectasis and intrapulmonary shunting due to beta-adrenergic stimulation. Increases in pulmonary vascular permeability also develops as a result of a number of the inflammatory mediators. The accumulation of fluid in the pulmonary interstitium and alveoli decreases pulmonary compliance and increases the work of breathing. Early manifestation of these changes include hyperventilation and the development of a respiratory alkalosis. With the worsening of the pulmonary edema and alveolar collapse, severe hypoxemia will develop creating the adult respiratory distress syndrome (ARDS).

Tissue metabolism is severely altered during the response to peritonitis. The metabolic rate is increased owing to the increased secretion of catecholamines and cortisol. However, hypovolemia reduces cardiac output. Increasing anaerobic glycolysis produces accumulating amounting of lactic acid and acid by-products. Renal and pulmonary clearance of this increased acid load leads to metabolic acidosis, unless perfusion is restored. A significant conversion in substrate metabolism also occurs in peritonitis.

Etiology of abscess formation

Intra abdominal abscess may be primary or secondary and each may be acute or chronic. They may follow from various abdominal catastrophies such as perforation of ulcer from any part of gastrointestinal tract with or without operative treatment. Intra abdominal abscesses may arise as a result of complication of surgeries of GI tract.

Causes of various intra abdominal abscess

Right superior (anterior) intra peritoneal (right subphreic space)

- Perforated duodenal ulcer
- Duodenal cap blow out following gastrectomy and appendicitis.
- Perforating cholecystitis
- Infection through the portal vein, CBD, hepatic artery and umbilicus to the liver.

Right inferior (posterior) intra peritoneal (right subhepatic) space

- Appendicitis
- Cholecystitis
- Perforated duodenal ulcer
- Following upper abdominal surgery
- Right colon pathology

Left anterior intra peritoneal space

- Surgery of stomach and colon and spleen

- Anterior gastric perforation
- Infections of left colon, pancreas and colon

Pelvic abscess

- PID ruptured appendix, Crohn's disease ruptured colonic diverticulum's
- Any surgery of small intestine stomach duodenum or urogenital system

Paracolic abscess

- Ruptured diverticulum
- Ruptured appendix
- Crohn's disease
- PID

Retroperitoneal abscess

- Uncommon secondary to infection of pancreas kidney and colon
- Surgery on retroperitoneal structures

Microbiology

The virulence of contaminating bacteria is influenced by a number of factors. Several organisms are well recognized for their innate ability to produce intra abdominal abscess in humans. As an example in secondary peritonitis, common fecal pathogens include aerobic coliform bacteria, anaerobic Bacteroides species.

Despite the massive contamination and complexity of the microbial spectrum that occurs with fecal perforation, within 24 to 48 hours only a few isolates are recovered in peritoneal fluid culture. This indicates that only a few pathogenic bacteria survive to predominate in the infection. In addition the predominance of a particular microorganisms within the local inflammatory process has been shown to vary. In an animal model of a polymicrobial colonic peritonitis, Weinstein demonstrated that E.coil and entercoccus were the predominant organisms during the peritonitis phase while B, fragilis predominated during the abscess phase.[15]

Adjuvant factors and possible effects upon host defenses

Adjuvant Factor	**Effect**
Hemoglobin, ferrous iron	Enhances bacteria-mediate inhibition of neutrophil function, iron source may support bacterial growth
Intraperitoneal fluid	Dilution of opsonins, inhibition of phagocytosis flooding of diaphragmatic lymphatics
Fibrin	Impairment of phagocytosis, premature neutrophil degranulation
Platelets	Occlusion of diaphragmatic lymphatics
Necrotic tissue	Depletion of complement, neutrophil inactivation
Gastric juice, pancreatic juice, urine, meconium	Induces sterile chemical peritonitis
Bile, bile salts	Facilitates bacterial spreading, toxic to neutrophils and peritoneal mesothelial cells
Barium sulfate	Impairs access of phagocytic cells to bacteria
Talc, drains, suture material, cellulose (gel foam, Ocycel)	Premature degranulation of neutrophils.

Another example of unique pathogenicity is the remarkable ability of encapsulated anaerobic bacteria to produce abscess formation, a characteristic attributed to the capsular polysaccharide components. The size of the bacterial inoculum also influences virulence and as expected, the ability to adhere to the mesothelial surface may also enhance the virulence of some organisms such as the Enterobacterioceae and Bacteroides fragilis.[24]

Organism[25]

- Gram-negative aerobic and facultative anaerobes
- Escherichia coli
- Enterobacter species
- Klebsiella species
- Pseudomonas aeruginosa
- Proteus species
- Serratia marcescens
- Citrobacter morganii
- Citrobacter species
- Others
- Yeast
- Candida species
- Gram-positive aerobic and facultative anerobes
- Nonenterococcal streptococci
- Enterococci
- Staphylococcus aureus or S. epidermidis

- Anaerobic organism
- Bacteriodes fragilis
- Other bacteroides
- Clostridium species
- Peptococci/streptococci
- Fusobacterium species
- Lactobacillus
- Eubacterium species
- Others

Clinical features

Fever

Spiking fever is most consistent finding in patients with abscess. But in patient who are on broad spectrum antibiotics fever may not be a consistent finding.

Pain

- Depending upon the location of abscess
- Right or left hypochondriac pain in subphrenic abscess with radiation to the shoulder. Pain increased after coughing in subphrenic abscess and there may be difficulty in breathing.
- Pain in right and left flanks in paracolic abscess
- Pelvic pain or lower abdominal discomfort in pelvic abscess

Diarrhea

It may be present in case of patients with pelvic abscess or paracolic abscess. Anorexia is a non specific symptom and is related to toxemia. Weight loss is also common due in large hyper catabolic state and associated anorexia.

Brief description and Clinical features associated with specific abscess

Liver abscess

Amoebic liver abscess is commonest in India and other tropical countries caused by parasite entamoeba histolytica and others. Common in alcoholics and cirrhotic patients. Infection commonly occurs from caecum after an attack of amoebic typhlitis through superior mesenteric vein and portal vein to liver.

Right lobe is commonly involved over postero superior surface due to stream line effect and larger size of the right lobe. Trophozoites destroy hepatocytes by releasing histiolysin leading to amebic hepatitis with multiple micro abscesses. It causes liquefaction necrosis leading to formation of anchovy sause pus. Manipulation of biliary tract, portal infection and hematogenous spread (i.v. drug abuse), local spread(diverticulitis/crohn's) and through indwelling catheters are the commonest causes .

Course and sequelae of liver abscess: It can rupture into lungs leading to expectoration of chocolate coloured sputum resulting in natural regression of abscess , it can rupture into peritoneum causing peritonitis ,it can rupture into pleural cavity leading to empyema or into bare area of liver causing retro

peritoneal abscess. Septicaemia and liver failure can occur in a patient with cirrhosis.

Clinical features: It is common in males (20:1), patients present with fever, loss of weight ,chills and rigors and non productive cough . Liver is tender, smooth with increased liver span. Intercostals tenderness is elicited and is a useful clinical sign, right sided pleural effusion may be evident. Mild jaundice may be present in cirrhotics and multiple abscesses.

Splenic abscess: It is an uncommon and potentially fatal illness. Predisposing illnesses include malignancies, polycythemia vera, endocarditis, UTI, I.V. drug abuse and AIDS. About 70% of splenic abscesses result from hematogenous spread of infecting organism from another location. Gram positive cocci such as staphylococcus, streptococcus or enterococcus species. The clinical presentation of splenic abscess is often non specific and insidious including abdominal pain, fever, peritonitis and pleuritic chest pain. The abdominal pain is localized in the left upper quadrant less than half the time and is more often vague abdominal pain. The spleenomegaly is present in minority of patients. The initial approach depends on whether it is unilocular or its multilocular. Unilocular abscess are amenable to CT guided drainage. Failure of a prompt clinical response to PCD leads to spleenectomy without delay.

Psoas abscess: They are commonly due to tubercular spine. Lower thoracic and upper lumbar spine are commonly affected. Clinical features includes pain in the back localized to lesion. Evening pyrexia and protective muscular spasm especially of sacrospinalis. In psoas abscesses pus enters the the psoas sheath

tracks downwards and causes mass in the iliac fossa. From here it traverses beneath inguinal ligament. If untreated it collects in the subcutaneous plane.

Subphrenic abscess: (pus somewhere , pus nowhere , pus under the diaphragm)

Upper quadrant abdominal pain associated with fever and tachycardia. There can be upper abdominal guarding and distention other suggestive findings include hiccoughs, tachyapnea, cough or jaundice. In the pre antibiotic era the large abscess ultimately produced characteristic patterns of abdominal tenderness especially over the twelfth rib or costal margin but with the use of antibiotics, the clinical presentation of subphrenic abscess has changed considerably. Patients currently demonstrate a decrease incidence of abdominal tenderness, an attenuated pattern of fever and less leukocytosis than previously shown, probably because the abscesses are smaller. The earliest findings are generally a persistent ileus or signs suggesting partial intestinal obstruction after a period of improved function. Non specific thoracic manifestation include pleural effusion, elevation of diaphragm and decrease in basilar breath sounds.

Inter loop abscess

There are no reliable symptoms or signs. A huge abscess containing more than 1 liter of pus may occur without any significant physical findings. Occasionally an interloop abscess may produce enlarging mass. The possible presence of an interloop abscess must be suspected if there has been a proceeding peritonitis with incomplete clinical resolution.

Pelvic abscess

Women usually have evidence of genital tract infection for days to weeks before the abscess develops. In those who develop a tubo ovarian abscess, pelvic inflammatory disease is manifested by diffuse lower abdominal pain, high fever, leukocytosis and occasional ileus. Post delivery or post-operative pelvic infection causes fever and purulent vaginal discharge.

Five presentations of pelvic abscess in the female have been noticed

1) Unilateral or bilateral parametrial abscess that bulge into the vagina or inguinal area
2) Cul-de-sac collections palpable in the posterior fornix
3) Tubo ovarian abscess fixed high on the pelvic wall
4) Tubo ovarian or ovarian abscess that present as an abdominal mass
5) Rupture of an abscess with peritonitis

Retroperitoneal abscess – Abdominal pain and fever are present in over 90% of patients with retroperitoneal infections. Other symptoms include nausea, vomiting, anorexia, chills and lumbar or psoas muscle spasm. About half of patients have a flank mass and tenderness with or without overlying edema. An unexplained lump may be the earliest sign of retroperitoneal abscess. The patients frequently lie in bed with the psoas muscle relaxed by flexing and abducting the thigh. Extension of thigh increases pain. Sigmoid carcinoma perforation can present with pelvic, thigh, per rectal or gluteal abscesses.

Lab investigations

Complete blood picture with coagulation profile

- Hb-Reduced hemoglobin/Anemia in long standing disease
- Total leukocyte count- Increase with neutrophila
- Liver function test- May be altered in case of patient in sepsis
- Renal function test- May be altered in view of ongoing sepsis
- Urine routine and microscopy may show increase in pus cells in case of pelvic abscess
- Serum electrolytes- May be deranged in view of hyper metabolic state in patients with gastrointestinal symptoms.

Specific investigation

Plain X-ray chest is not specific, but may show pleural effusion or pulmonary infiltrate in subphrenic abscess. Elevation of diaphragm in approximately 75% of patient with sub diaphragmatic abscess. X Ray abdomen may show[26] extra luminal air, effacement of preperitoneal/psoas outlines. Retroperitoneal abscess may show radiographic evidence of soft tissue mass.

Specific investigations

USG (Ultrasonography)[27]

Gray – Scale USG is sensitive investigation to diagnose intra abdominal abscess. Accuracy in experienced hands is >90% (~96.8%).

Advantages of USG are portability, low cost, rapidity, no X ray radiation, used for interventional purposes. Disadvantages include obesity, wounds, dressings, colostomies, air filled bowel loops and overlying lung can result in unsatisfactory scans.

Fluid collections are not specific and can overlap the features of cysts and sterile collections. Sensitivity of USG is between 75-82%.[28] Echogenicity[29] varies from homogenous hypoechoic fluid to complex echogenic collection. Gas within fluid collections, although present inconsistently is highly suggestive of infection which appears as an area of increased echogenecity with/without acoustic shadowing.

USG is effective for detecting an abscess in the right upper quadrant, retro peritoneum, pelvic but has low sensitivity in detecting interloop abscess.

Computed Tomography (CT) Scan

CT Scan is a superior but more costly mode of investigation. In patients with suspected intra abdominal abscess, sequential CT from just above the diaphragm to symphysis pubis allows a radiological laparotomy.[30] CT Scan has a sensitivity of 78% to 100%. As with USG the sensitivity of CT for detecting abscess in the interloop area is lower (60%).[27] The specificity of CT is also high (98%). When it is used in conjugation with diagnostic aspiration and clinical correlation.[26] CT is superior to USG for all anatomic sites with the possible exception of pelvis.[31] Criteria for identification of an abscess by CT include areas of low attenuation either within parenchymal organs or in an extraluminal location within a peritoneal cavity and additional features are intra cavitory gas,

thick or irregular walls, contrast enhancement of the wall and heterogeneous internal debris. Gas has been estimated to be present in 40% to 50% of intra abdominal abscesses and when present is highly suggestive of diagnosis.

The advantages are, CT scan provides radiological laparotomy, most specific and sensitive, wound, dressings, ostomies, and drain and obesity does not interfere with CT scan and gas resolutions of anatomy and delineation of retroperitoneal structures. The disadvantages are, CT scan is expensive and non availability in all centers, cannot differentiate whether collection is sterile or infected, thick collection with debris can give false positive rates, uses ionizing radiation, requires administration of both oral and IV contrast and hence limiting its use in patients with ileus and renal insufficiency.

Radionuclide scanning

Early infection, before the development of discrete fluid collection, is more difficult to detect, particularly in patients with distortion of normal anatomy due to recent trauma or surgery. In these clinical situations radionuclide scanning may be of some value.

a) Gallium 67 scanning[32]

It is administered intravenously which binds to lactoferrins released by leukocyte at the site of inflammation. It is very non specific test and readily images neoplasms as well as abscesses. The material is excreted in the gastro intestinal tract and vigorous bowel preparation is necessary (Disadvantage in patients with ileus). Because of the short half-life of the isotope there is

considerable cost in generating and rapidly transporting the dose necessary for the test. 24-72 hours are required for uptake before scanning. However biggest disadvantage is the lack of specific labeling of inflammatory foci.

b) Indium-111[33]

In an attempt to increase the accuracy rate over that of gallium-67 indium-111 has been used as an in vitro-label for autologous neutrophils, which are then infused intravenously. The leucocytes subsequently migrates to sites of inflammation. Several advantages have been found. The neutrophils can be separated in vitro and specifically labeled giving amazingly high blood rations (as high as 81:1 in experimental models). A corresponding lower total dose of radiation can be used. Disadvantages include, it still is relatively expensive and involves moderate dose of radiation to patient. 24 hours are required for the neutrophils to localize at the inflammatory site. Fully functional neutrophils are necessary and neutrophil dysfunction is common in the severely septic patient.

More recently alternative WBC labeling agents have been evaluated. The most promising of these agents is technetium 99m-hexamethyl-propylene-amine-oxide (99^{m}TCHM-PAO).[34] The labeling procedure for this agent is easier and more widely available than for other agents. The most significant advantage of 99^{m} TcHM-PAO labeled WBC is that scanning may be done 4 hours after this agent is administered. A comparative study of WBC labeled with 99^{m} TcHM-PAO and ^{111}In-osine that were administered to 41 patients with suspected intra abdominal sepsis revealed that the sensitivity of Tc^{99m} HM-PAO scanning after 4 hours was equivalent to that of In^{111} scanning after 24 hours.[35]

Magnetic resonance imaging (MRI)[36]

Contrast enhanced magnetic resonance imaging may play a role in defining intra abdominal abscesses. However at present MRI cannot replace CT as a technique for general screening of the entire abdomen.

Management of intra abdominal abscess

As with generalized peritonitis, management of abdominal abscess requires resuscitation, supportive measures, abdominal drainage and systemic antibiotics.

Resuscitation

Resuscitation aims to maintain patient's hydration status, to maintain blood pressure and to maintain urinary output. As patient with sepsis they have some amount of hypovolemia, the initial resuscitation aims to correct the hypovolemia and require immediate resuscitation with intravenous crystalloids. Usually several liters are needed to normalize urinary output and blood pressure. If oxygen saturation is low patient may require O_2 supplementation and mechanical ventilation. Naso gastric decompression is required if there is associated ileus.

Antibiotic therapy

The initial selection of antibiotics is empirical but if diagnostic tap is done antibiotics can be started according to culture sensitivity.

Systemic antimicrobial agents[37]

There are numerous observational studies and prospective randomized double blind clinical trials of antimicrobial efficacy in abdominal infections. From these studies certain principles are listed below :

- Antimicrobial therapy is merely an adjunct to abdominal drainage that has the primary role in determining the therapeutic outcome of abdominal infection
- Initial antimicrobial therapy is empirical, since identification of infecting pathogens takes 1 or 2 days .
- Whether used single or in combination, the antibacterial spectrum of therapy must cover E.coil and other gram negative facultative and aerobic rods and anaerobes such as Bacteroides species[38]
- Although certain clinical settings (i.e hospital acquired peritonitis in a patient who has received antibiotics) are associated with the finding of multiple resistant pathogens in infected abdominal fluid and treatment failures often are caused by infections with such organisms where there is no valid evidence that expanding the spectrum of antimicrobial coverage beyond E.coil and Bacteroides species is associated with improved clinical outcome.[39,40]
- Prolonged antibiotic administration is associated with adverse effects and unnecessary cost. Treatment has only to be continued until the patient has recovered clinically, if a patient is still sick after receiving a week of antimicrobial therapy, the drugs should be stopped and the

source of infection including residual recurrent drainable infection in the abdomen should be sought aggressively.

- The intravenous route of administration ensures that therapeutic drug concentrations reach the infected abdominal tissues. Recent studies have shown that once gastrointestinal function returns, oral antibiotics are efficacious as intravenously administered therapy for abdominal infections.

Amino glycoside plus anti-anaerobe[30]

This regimen has a high risk of adverse effects and low cost. The amino glycosides include gentamycin, tobramycin or netilmycin, combined with an anti-anaerobe like clindamycin or metronidazole

Standard dosage

Aminoglycosides should be administered as a single daily dose of 5mg/kg (for amikacin, 15-20 mg/kg). the dose of metronidazole is 500 mg every 12 hours to every 8 hours, the clindamycin dose is 600 to 900 mg every 8 hours.

Side agents and combination regimens

This regimen has a low risk of adverse effects and high cost. Single agents include carbapenems (impenem, meropenem) second (cefotetan, cefoxitin) and β-lactam/ β-lactamase inhibitors (ampicillin sulbactam, piperacillin tazobactam and ticarcillin clavulanic acid) and trovafloxacin. Combination regimens include anti-anaerobes (clindamycin or metronidazole) plus third and

fourth generation cephalosporins (Cefoperazone cefotaxim, ceftazidime, ceftizoxime, ceftriaxone, cefepime, aztreonam or ciprofloxacin.

Standard dosage

Imipenem 500 mg every 6 hours. Meropenem 1g every 8 hours. Cephalosporins: cefoxitin, cefotaxime: 1 to 2 g every 6 hours, cefoperazone ceftazidime, ceftizoxime every 8 hours. Cefotetan, cefepime, every 12 hours or ceftriaxone every 24 hours. Ampicillin tazobactam 4.5 g every 8 hours. Ticarcillin-clavulanic acid 3.1g every 6 hours. Aztreonam 2g every 8 hours, Ciprofloxacin 400mg every 12 hours

Cost effectiveness

Although costs vary among the different regimens named in this group, drug acquisition is more expensive than for amino glycoside based regimens.

Oral ciprofloxacin plus metronidazole amoxicillin clavulanic acid

These regimens are low risk of adverse effects and low cost

Standard dosage:

The doses for oral administration often differ from that for parenteral use: ciprofloxacin 500 to 700 mg every 12 hours, metronidazole 500mg every 12 hours, amoxicillin-clavulanic acid 500mg every 8 hours.

Diagnostic tap- diagnostic fluid aspiration

It is done by USG or CT scan and requires only a small sterile needle and local anesthesia. Area is cleaned with Betadine solution and spirit and local anesthesia agent is infiltrated. Abscess is localized using USG/CT scan and then needle is directed into the collection and sample aspirated for analysis.

If percutaneous drainage is considered care should be taken to aspirate only enough fluid for analysis because repeat puncture can be more difficult if a large portion of the collection has been evacuated sample is then sent for gram staining and culture. Also sterile abscess are not uncommon, some specimen may yield white cell without bacteria, particularly if a patient is taking antibiotics.

Drainage of abscesses

An established intra abdominal abscess can be drained by percutaneous CT or US guided drainage, extra peritoneal surgical drainage or transperitoneal midline abdominal exploration

Percutaneous drainage (PCD)

Percutaneous catheter drainage under CT or US guidance is now an acceptable alternative to surgery. A safe drainage route that avoids puncturing of solid organs or hollow viscera can be identified in 85% to 90% of patients. USG guided PCD can be done bedside which is very advantageous.

Criteria for consideration of percutaneous drainage includes;[15]

1. Well defined abscess cavity.

2. A safe percutaneous route.

3. Immediate operative capability in case of failure.

4. The abscesses that has been most amenable to treatment by the percutaneous route are unilocular although communicating septated or multilocular abscesses may be successfully treated.

Success rate for PCD for well defined unilocular abscesses have generally ranged from 80 to 90%[41,42] Drainage of more complex abscesses (loculated, poorly organized or interloop, intra mesenteric, pelvic, splenic and appendiceal abscesses) has been less successful and higher rate of complication have been associated with draining of these abscesses.

Contradictions to percutaneous drainage:

Absence of a safe route for drainage. The presence of foreign or solid or semisolid material or a source of continuous diffuse peritoneal contamination with gastrointestinal contents is a relative contraindication.

Complications of percutaneous drainage

- Injury to viscus or blood vessel
- Inadequate drainage
- Malpositioning of tube
- Dislodgement of tube
- Erosion of neighboring structures when tube is kept for long time.
- Peri-drain leakage

- Sepsis
- Bowel perforation
- Pleural space complication like empyema, pneumothorax

Failure or percutaneous drainage

- Multiloculated abscesses not suitable for percutaneous drainage
- Presence of necrotic material
- Tumor with central necrosis simulating abscess
- Poorly defined parenchymal abscess
- Viscous pus that could not be drained adequately
- Persistent fistulae from gastrointestinal tract
- Diffuse micro abscesses and phlegmonous collection
- Contamination by candid albicans or other opportunistic agent

Advantages of USG/CT guided aspiration:

- Reduced operation time
- Decreased post operative pain
- Decreased wound infection rates
- Reduced hospital stay
- Faster return to normal activity
- Avoids unnecessary open exploration.
- Economical

Disadvantages of USG/CT guided aspiration

- Previous surgeries
- Ongoing intra abdominal sepsis
- Morbid obesity

- Pregnancy
- Difficult access
- Patients with coagulative disorder

Advantages of open drainage

- Less treatment failures
- Multiloculated abscesses
- Helpful in recurrent abscesses
- Abdominal abscess with peritonitis
- Abscess with thick pus
- Better access to difficult sites
- Better hemostasis in patients with coagulative disorders .

Disadvantages of open drainage:

- Increased hospital stay
- Increased morbidity
- Anesthetic complications

Advantages of laparoscopic drainage

- Reduced hemorrhage
- Smaller incision and less post operative scarring
- Decreased post operative pain and disability
- Faster return to normal activity
- Reduced infection rate

Disadvantages of laparoscopic drainage

- Longer procedure time
- Needs laparoscopic trained personnel

- Complications of G.A.

Drainage methods[43]

The shortest possible route to the abscess cavity that avoids intervening organs or vital structures is selected. Often a combined CT-USG approach is used to plan procedures. If possible transperitoneal routes and approaches through the anterior abdominal wall are avoided unless the precise location of the nearby loops of bowel is unknown.

Trocar catheter technique

After confirmatory aspiration, the sheath of the needle is removed and a 10 to 16 French trocar catheter with multiple side holes is selected, 5ml retention balloon suture cuff and self contained central stainless steel trocar must be inserted at the same site angle and depth as the immediately preceding aspiration needle (21-24). The trocar should be removed and the abscess aspirated and the catheter sutured in place. Because of a tendency of the catheter to slide out through the suture cuff, the latter is removed and the suture tied around catheter itself.

Modified seldinger technique

After confirmatory aspiration with a sheathed needle an 0.089 cm floppy angiographic guide wire is inserted through the sheath which is then removed. A single angiographic dilator is passed over the wire to enlarge the track slightly. After removal of the dilator an 8 French multiple side hole pigtail catheter is passed over the guide wire into the abscess and the wire removed. The abscess

was completely evacuated and the catheter sutured securely in place. The catheter tip immediately assumes its coiled pigtail shape within abscess cavity and protects against perforation of the far wall or accidental dislodgement.

Cope method[8]

With the cope method a 22 gauge needle is advanced into the abscess. After aspiration of fluid an 0.018 inch guide wire is threaded through the needle. The needle is removed and a curved 5fr Teflon dilator is introduced over the guide wire. This dilator has a long tapered tip and follows the guide wire without kinking. It also has a side hole in the inside curve, through which a tight 0.038 inch guide wire will exist. Subsequent dilators and larger catheters can be introduced over this guide wire. This method is fast and relatively atraumatic and replaces a small with a large (0.038 inch) guide wire system with one puncture.

Tandem technique

In the tandem technique an 18G needle inserted in tandem to the same depth and angle alongside a 22 gauge needle. Once fluid is aspirated 0.035 inch or 0.038 inch guide wire is passed through the needle and coiled in the cavity. Dilators are passed with the guide wire upto size 1 or 2 french larger than the drainage catheter that has been positioned and the wire is removed and fluid is aspirated. Contrast medium is injected for documentation of the extent and size and fistulous communication of the cavity and to evaluate the position of the catheter size holes to avoid their placement in the tract.

Single 18 or 20 gauge needle technique. In some patients in whom a safe tract into the abscess is present or in whom the collection is superficial, a direct puncture with an 18 to 22 gauge needle can be performed with imaging guidance. Following insertion of an 18 gauge needle into abscess cavity the procedure is completed as in tandem technique.

Irrigation of the abscess cavity following catheter insertion

The utility of daily irrigation of catheter is debatable. Some authors do not irrigate the catheter as long as the purulent material is drained others prefer a small amount of irrigation with normal saline, depending on the size of cavity with a gradual decrease in the amount of irrigation until it is injected only once every day still others prefer to install proteolytic fluids, antibiotics or both. Intra cavitory urokinase installation which can reduce the viscosity of purulent material and decrease flow transit time has been shown to be a safe adjunct to abscess drainage without significant changes in coagulation parameters or development of bleeding complications.

After successful drainage there should be prompt abatement of clinical signs of infection with defervescence within 24 to 48 hours. Persistent fever or leukocytosis is an indication for repeated scanning to detect incomplete drainage. Placement of additional catheter or open surgical drainage may be required.

Criteria for catheter removal

Clinical criteria

- Defervescence
- Decreased white blood cell count
- Improved appetite and overall condition

Radiologic criteria

- Smaller size on image of contrast filled abscess
- Smaller size by USG or CT
- No evidence of localization or multiple collections

Catheter criteria

- Decreased drainage

Surgical drainage[8]

Primary surgical drainage is indicated in any situation in which

a) The abscess is poorly defined or difficult to localize by imaging techniques (eg interloop abscesses).
b) The abscess material is vicious or extensive necrotic debris is present.
c) The approach for percutaneous drainage is likely to cause bowel injury.
d) Associated with fistulae/obstruction.

Secondary surgical drainage should be performed if either clinical sign of sepsis persist after percutaneous drainage or complete evacuation of the abscess

cavity cannot be achieved. During operation the abscess wall should be identified and the cavity aspirated with a needle to confirm the nature of the abscess with the presence of pus. The abscess should then be widely opened and contents evacuated. Necrotic tissue is debrided and copious irrigation of the cavity is performed. Drains are placed in dependent positions and externalized via separate incisions if necessary.

Operative treatment

The extra serous approach became the accepted procedure prior to the antibiotic era for several reasons

1) The abscess were large and relatively easy to locate.

2) Spillage of large volumes of purulent material into the peritoneal cavity would cause septicemia and hypovolemic shock.

3) With large abscesses loculations were usually not a problem and the cavities were evacuated readily. The alternative to transthoracic or extra serous drainage is laparotomy.[44]

The transperitoneal approach allows visualization of all potential spaces, aspiration of all loculated purulent material, debridement of some if not all fibrin deposits, precise placement of drains and lavage of the freshly contaminated peritoneal cavity with antibiotics. Whenever doubt exists about the precise cause of intra-abdominal infection the transperitoneal approach should be taken.

Post-operative care

Parenteral antibiotics depending upon culture and sensitivity should be given till fever and leukocytosis subside.

Catheter/drain = Monitor quantity and quality of drained fluid. Irrigation with 25cc of sterile normal saline can be performed one to three times a day depending upon the viscosity of the contents. If the drain is nonpurulent and less than 30ml/day then USG obtained by injecting a dilute contrast to verify the collapse of the cavity before drain are removed. It may take 2-3 weeks for a large cavity to become small enough to permit drains to be slowly advanced, allowing drainage tract to seal as they are withdrawn.

Specific abscesses

Abscesses are most common in right and left lower quadrants as a consequence of appendicitis, diverticulitis and pelvic sepsis. Abscesses around the liver comprise the next most common group of intra-abdominal septic collections. Six spaces around the liver are described. Superiorly the falciform ligament divides the left and right subphrenic spaces, later being divided into anterior and posterior spaces by triangular ligament. In the infra hepatic region there are also 3 spaces- two on the left side including the lesser sac of the peritoneal cavity and the space immediately below the lateral lobe of the left hepatic lobe.

Interloop abscess

These are actually multiple abscesses created by adherent loops of intestine, mesentery, omentum and the abdominal wall. Though superior extension of these abscesses is prevented by transverse mesocolon, synchronous pelvic abscesses may be found.

CT scan is the diagnostic modality of choice. CT scanning is also best to plan the intended catheter use. Because of the ill defined nature of these abscesses surgical drainage is the preferred approach.

Retroperitoneal abscess

The retro peritoneum is a potential space with clearly defined boundaries between the peritoneum and transversalis fascia lining the posterior abdominal cavity. Extending laterally to the edges of the quadrates lumborum muscles the diaphragm superiorly and pelvis inferiorly.

US guided transvaginal approaches can also be performed for drainage of deep pelvic abscesses. Van Sonnen Vannkuberg et al. demonstrated 100% success rate without major complication using this approach in 13 patients. One drawback of this technique has been difficulty in the passage of a drainage catheter through the tough vaginal wall tissues.

Transrectal pelvic abscesses drainage has become an acceptable alternative for gaining access to deep pelvic collections. The transrectal approach can be guided by fluoroscopy endosonography or CT. Regardless of image

guidance employed the transrectal approach appears to be safe, well tolerated and effective procedure.

A transgluteal approach through the greater sciatic foreman has been described by Butch et al. in which a needle is directed to the sacrospinious ligament in order to avoid traversing major sacral vascular and neural structures. The disadvantage of this route is the need to traverse substantial amount of soft tissue in order to reach the abscess cavity. Catheter kinking and patient discomfort are other common problems with this catheter approach.

A CT guided paracoccygeal infragluteal approach has been described by Longo et al. as an alternative to conventional transgluteal approach for presacral abscesses. In this technique a needle site is selected 1-2cm caudal and lateral to the coccyx and is directed in a paracoccygeal position towards the presacral collection. This approach appeared to minimize patient discomfort and pelvic abscesses.

Most pelvic abscesses are surgically drained through the rectum or vagina. However as in the abdomen the advent of CT and USG has provided additional non surgical percutaneous approached for pelvic abscess drainage.

Primarily with the aid of CT or USG a trans abdominal approach has been the mainstay of pelvic abscess drainage. While effective in the majority of cases, several limitations can exist due to presence of interposing bowel, vessels, bladder, uterus or large amount of abdominal or pelvic soft tissue. These are the limitations of trans techniques such as transgluteal, paracoccygeal, transperineal and transrectal approaches.

CT scan is the diagnostic modality of choice. CT is particularly useful for evaluation of the retrofascial musculature and the perineal compartments. Drainage can be PCD or surgical. If an abscess is unilocular relatively free of particulate matter and safety approachable percutaneous drainage is attempted as initial maneuver.

Surgical drainage is done preferably by retro peritoneal approach (flank incision) rather than by transabdominal approach to avoid contamination of abdominal cavity.

Outcomes and prognosis

Abscesses within abdomen carry a high mortality and a high morbidity rate. A renew of the surgical literature discloses an 80-100% mortality of un drained abscesses and even with surgical drainage mortality was about 10-30%.

Studies comparing PCD with historical surgical controls have shown favorable results for PCD in terms of both success rates and complication rates.

The improved success rate of PCD may be partially due to earlier diagnosis and earlier treatment associated with the use of CT or USG as well as improvement in supportive and antimicrobial therapy.

Chapter 4

Methodology

METHODOLOGY

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010.

Study design

A one year cross sectional study.

Place

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum attached to Jawaharlal Nehru Medical College, Belgaum.

Study period

One year from January 2010 to December 2010.

Source of data

Patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period.

Sample size

A total of 40 patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were studied.

Sampling method

Based on the hospital statistics, average 80% of previous three years admissions with intra abdominal abscesses the sample size was calculated as 40 patients.

Selection criteria

Inclusion

- Diagnosed cases of intra abdominal abscess due to primary disease of any viscera in the abdomen.

Exclusion

- Multiloculated abscesses.(more than 2 abscesses)
- Immuno compromised patients.
- Abscess with minimal accessibility.
- Perinephric abscesses.
- Deep pelvic abscess where transrectal drainage in necessary.
- CT showing poor wall definitions.
- Patient not willing to enroll in the study.

Procedure

Ethical clearance for the study was obtained from Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum. Based on the selection criteria patients admitted with intra abdominal abscess in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre,

Belgaum were screened for eligibility. The eligible patients were briefed about the nature of the study and a written informed consent (Annexure I) was obtained from the selected patients.

Thorough history was taken and clinical examination was done for all the patients and findings were recorded on predesigned and pretested proforma (Annexure II).

Based on the clinical signs patients underwent the relevant clinical investigations such as prothrombin time (PT), International normalized ratio (INR), clotting time (CT), bleeding time (BT) and complete blood count (CBC). Further these patients were subjected to special investigations such as ultrasonography (USG) or computed tomography (CT) for detection and localisation of intra-abdominal abscesses.

On the basis of these images, routes for diagnostic aspiration and percutaneous drainage were planned. After taking the patient to the procedure room (CT scan room) / bedside, part is painted and draped and local anesthesia is given. Over the routes, indwelling catheters (10G to 22 G trocars) were inserted keeping in mind the depth of the abscess, immediate decompression, evacuation and continuous drainage of the abscess cavity was done until the abscess was resolved.

Drainability of abscess cavity was considered as first outcome. Mezol wash was given two times a day until the drainage stopped. Result of the Respondent imaging studies included, resolution (no residual collection) or residual collection (any collection on next follow up even if small). The pus was

sent for culture and antibiotic sensitivity examination. Need for repeated drainage was decided accordingly. Duration of hospital stay, duration of drainage and outcome were recorded.

Statistical Analysis

Data obtained was tabulated and expressed as rates, ratios and percentages. A probability value (‘p’ value) of less 0.05 was considered as statistically significant.

Chapter 5

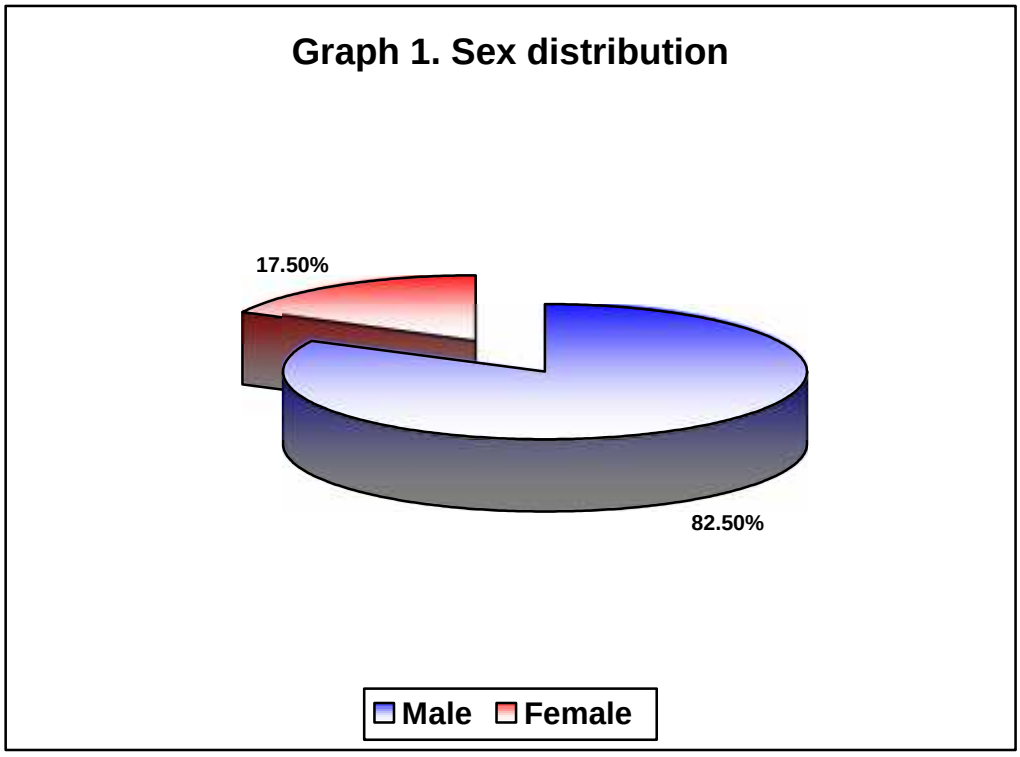
Results

RESULTS

This one year cross sectional study was conducted on a total of 40 patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Data obtained was tabulated on a excel spread sheet analysed.

Table 1. Sex distribution

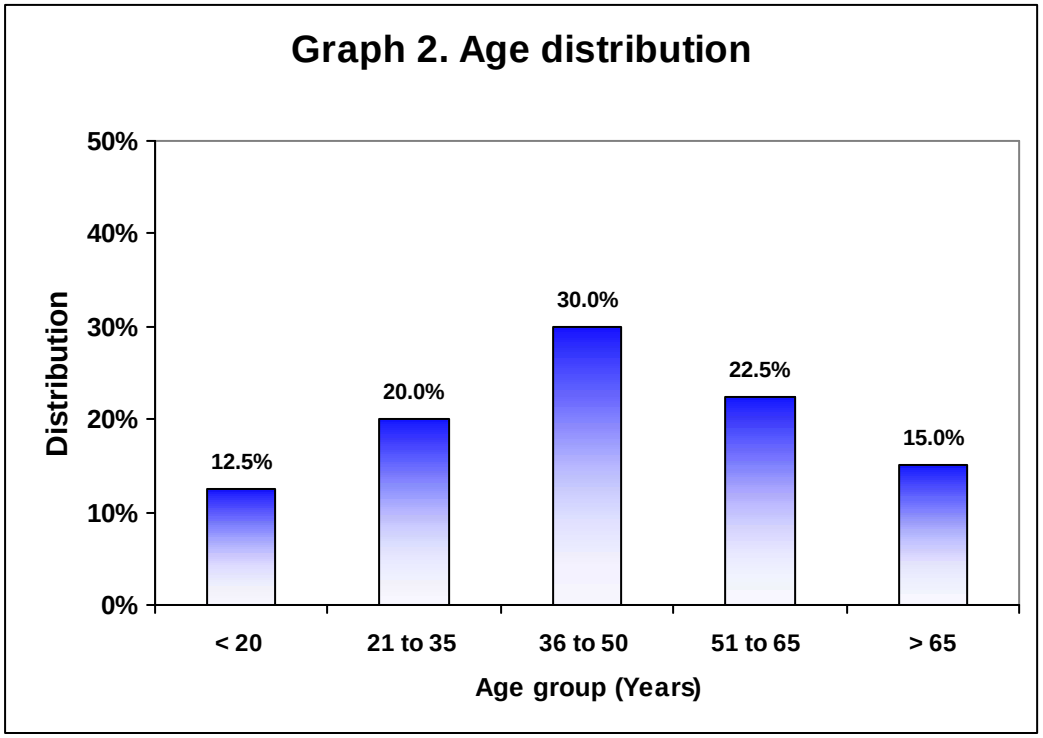
Sex	Distribution (n=40)	
	Number	**Percent**
Male	33	82.50
Female	7	17.50
Total	**40**	**100.00**



In the present study 82.50% were males and 17.50% were females with male to female ratio of 4.71:1.

Table 2. Age distribution

Age group (Years)	Distribution (n=40)	
	Number	**Percent**
< 20	5	12.50
21 to 35	8	20.00
36 to 50	12	30.00
51 to 65	9	22.50
> 65	6	15.00
Total	**40**	**100.00**

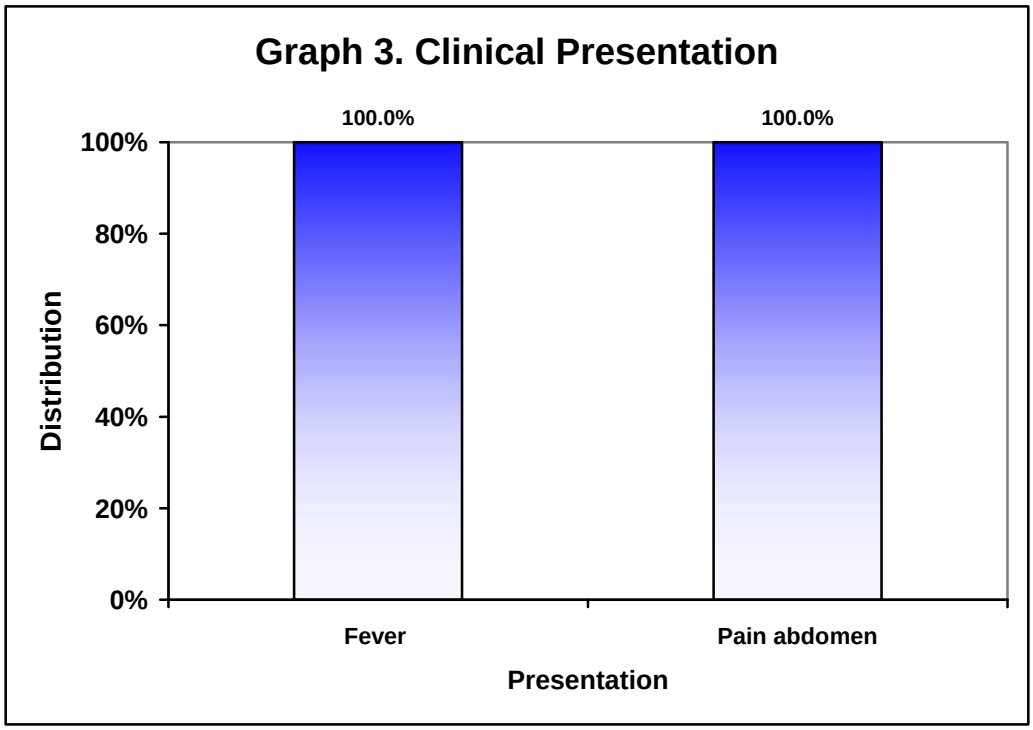


In this study the commonest (30%) age group was 36 to 50 years followed by 51 to 5 years (22.50%) and 21 to 35 years (20%). The mean age was 45.85 ± 18.31 years with range being 17 to 84 years.

Table 3. Clinical Presentation

Presentation	Distribution (n=40)*	
	Number	**Percent**
Fever	40	100.00
Pain abdomen	40	100.00

* Multiple Presentations



In the present study all the patients presented with fever and pain abdomen (100%).

In this study, the commonest associated condition was previous surgery in 25.00% of cases followed by diabetes mellitus in 17.50%. However other associated conditions recorded were abscess (7.50%), trauma (5.00%), alcoholic liver disease, hypertension and tuberculosis in 2.50% cases each.

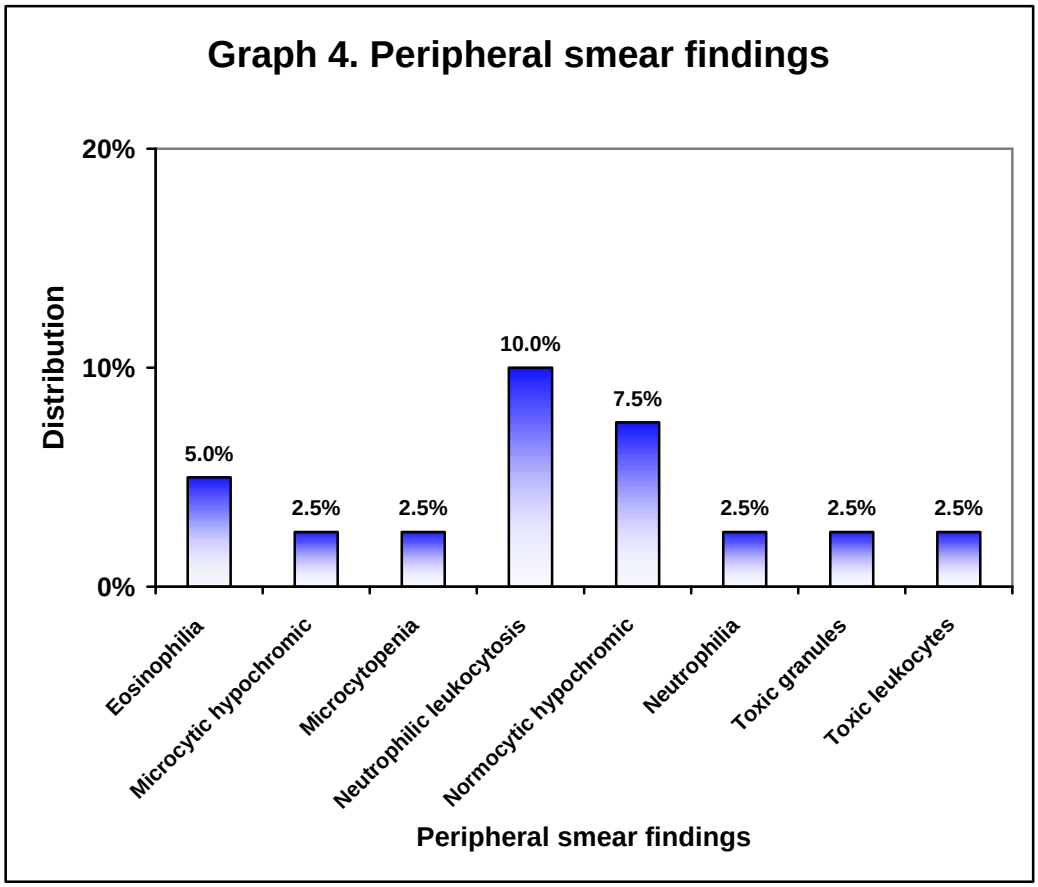
Table 4. Investigations

Investigations	Mean values	
	Mean	**SD**
Haemoglobin (gm%)	10.82	9815.20
Total count (/mm3)	15297.5	12806.73
Platelet count (/mm3)	2028.06	30.02
Serum urea (mg/dL)	27.15	1.21
Serum creatinine (mg/dL)	1.04	0.36
Bleeding time (Sec)	2.64	0.78
Clotting time (Sec)	4.03	1.49
Total Bilirubin (mg/dL)	1.75	3.02
Direct Bilirubin (mg/dL)	1.38	4.82
Prothrombin Time (Sec)	19.19	2.41
International Normalised ratio	1.91	0.62
Total protein (gms)	6.17	0.67
Serum albumin (gms)	2.28	0.28
A:G Ratio	0.64	83.59
SGOT	62.37	25.52
SGPT	47.1	38.54
Alkaline phosphatase	136.98	38.54

Table 4 shows the mean values of complete blood picture, liver function tests and renal profile.

Table 5. Peripheral smear findings

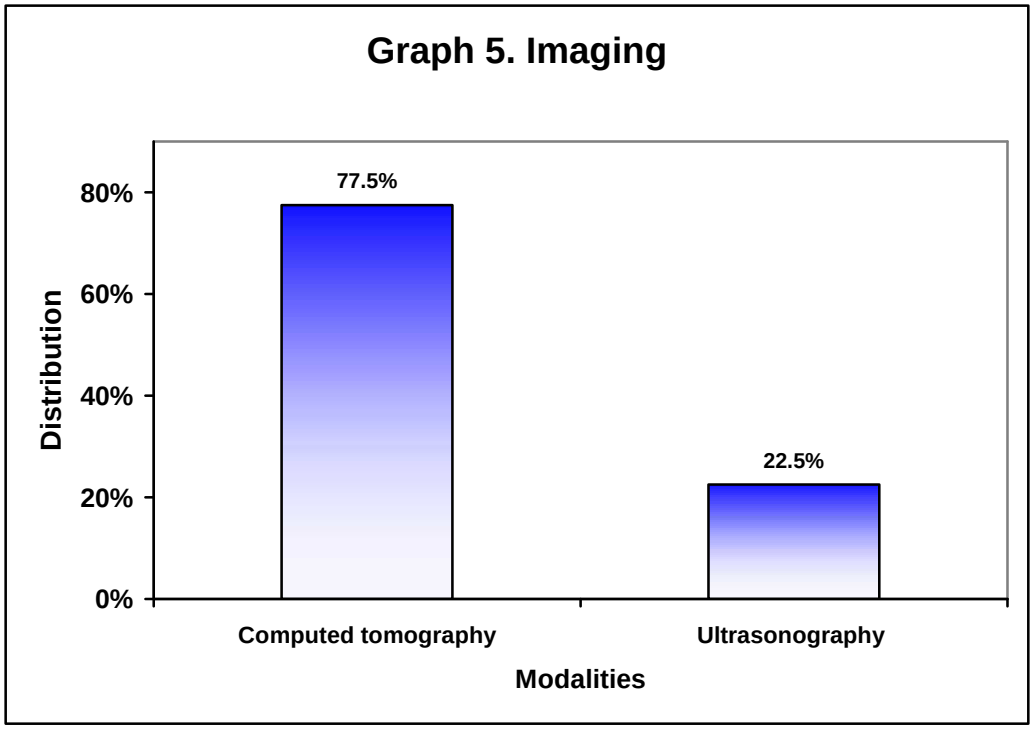
Findings	Distribution (n=40)	
	Number	**Percent**
Eosinophilia	2	5.00
Microcytic hypochromic	1	2.50
Microcytopenia	1	2.50
Neutrophilic leukocytosis	4	10.00
Normocytic hypochromic	3	7.50
Neutrophilia	1	2.50
Toxic granules	1	2.50
Toxic leukocytes	1	2.50
Total	**14**	**35.00**



In this study peripheral smear in four cases (10%) revealed neutrophilic leukocytosis, three (7.5%) normocytic hypochromic and two cases (5%) with eosinophia. The other findings are as shown in table 6 and graph 5.

Table 6. Imaging

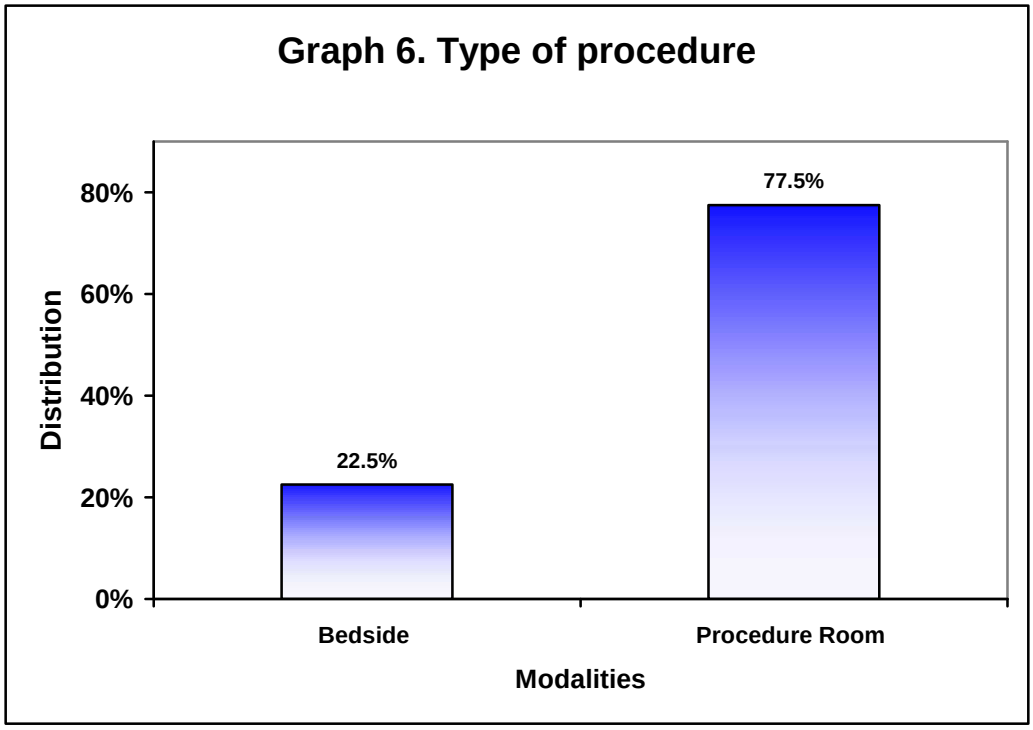
Imaging	Distribution (n=40)	
	Number	**Percent**
Computed tomography	31	77.50
Ultrasonography	9	22.50
Total	**40**	**100**



In the present study 77.5% cases underwent computed tomography and 22.5% underwent ultrasonography.

Table 7. Type of procedure

Procedure	Distribution (n=40)	
	Number	**Percent**
Bedside	9	22.50
Procedure Room	31	77.50
Total	**40**	**100**

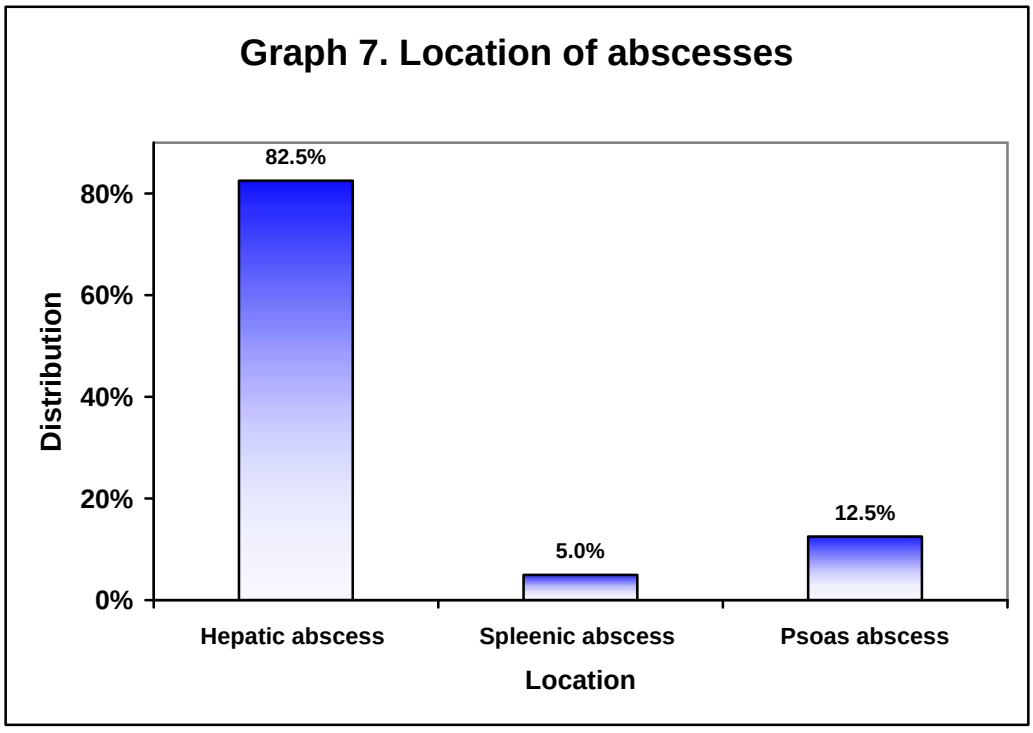


In this study 77.50% cases underwent bedside procedure whereas 22.5% in procedure room.

Table 8. Location of abscesses

Location	Distribution (n=40)	
	Number	Percent
Hepatic abscess	33	82.50
Splenic abscess	2	5.00
Psoas abscess	5	12.50

* Multiple conditions

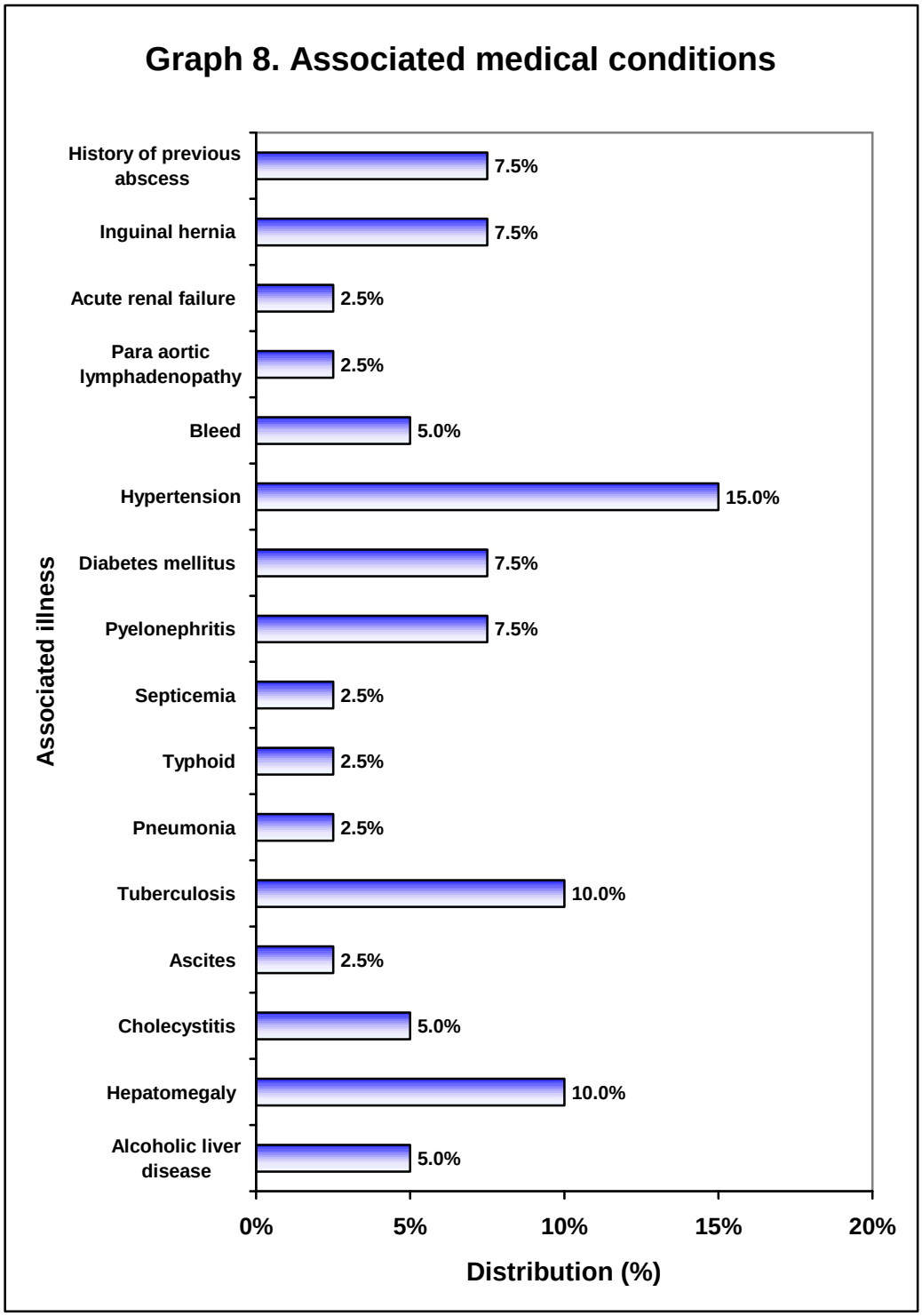


In the present study majority of the patients had hepatic abscess (82.50%).

Table 9. Associated medical condition

Associated illness	Distribution (n=40)	
	Number	**Percent**
Alcoholic liver disease	2	5.00
Hepatomegaly	4	10.00
Cholecystitis	2	5.00
Ascites	1	2.50
Tuberculosis	4	10.00
Pneumonia	1	2.50
Typhoid	1	2.50
Septicemia	1	2.50
Pyelonephritis	3	7.50
Diabetes mellitus	3	7.50
Hypertension	6	15.00
Bleed	2	5.00
Para aortic lymphadenopathy	1	2.50
Acute Renal failure	1	2.50
Inguinal hernia	3	7.50
History of previous abscess	3	7.50

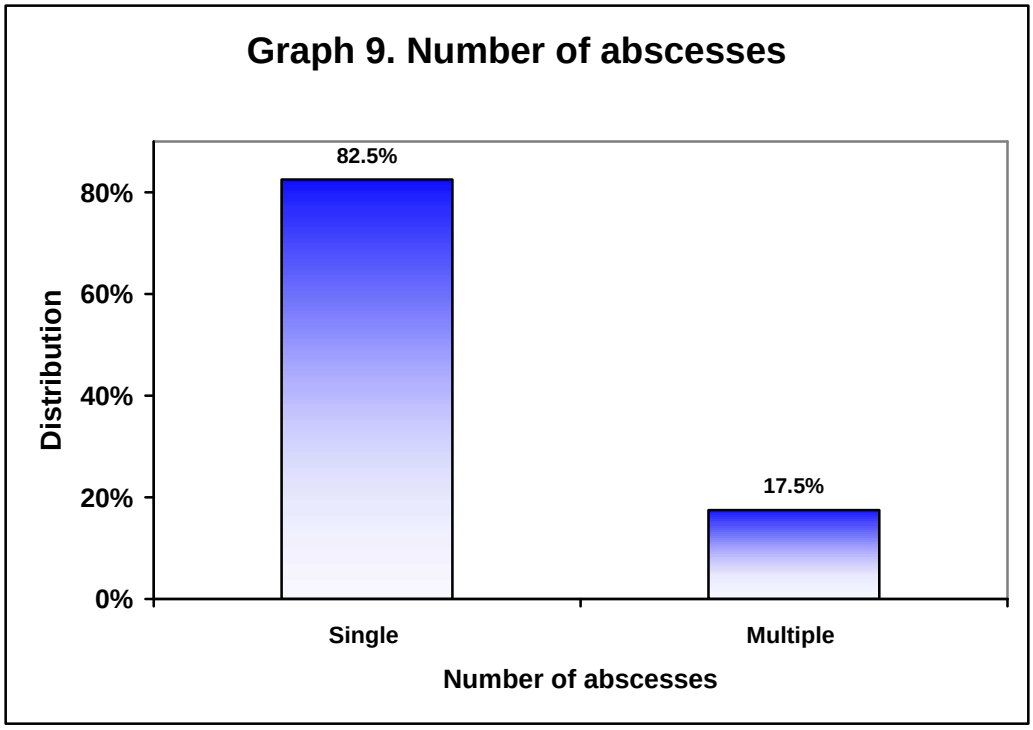
* Multiple conditions



In the present study hypertension, tuberculosis and hepatomegaly was the commonest associated medical conditions (10%) The other associated illnesses are as shown in table 7 and graph 8.

Table 10. Number of abscesses

Number of abscesses	Distribution (n=40)	
	Number	**Percent**
Single	33	82.50
Multiple	7	17.50
Total	**40**	**100**



In this study single abscess was seen in 82.5% of patients whereas 17.5% had multiple abscesses.

The drainage was done using 12 and 14 single tube except in one case where multiple tubes were used. There was no evidence of peritube leakage and tube dislodgement. The mean duration of tube in situ was 5.97 ± 2.67days.

Table 11. Mean amount drained

	Mean drainage (mL)	
	Mean	**SD**
Drainage	31.15	16.35

In this study the mean drainage was recorded as 31.15 ± 16.35 (mL).

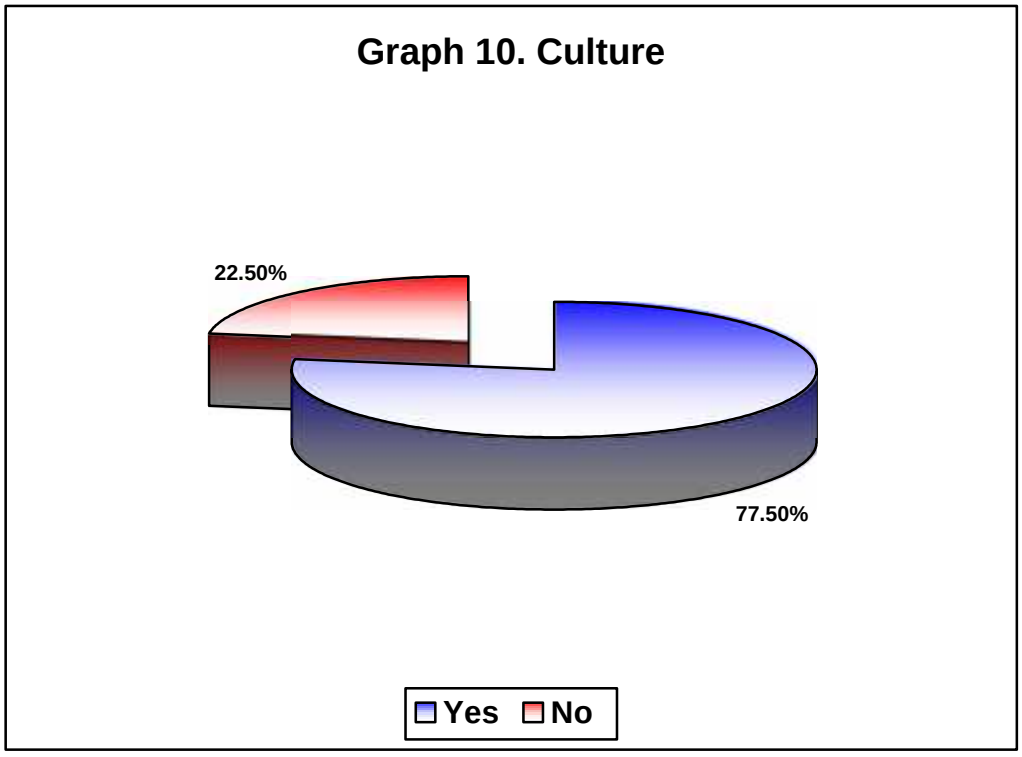
Table 12. Mean drainage on Day 1, 2 and 3

Days	**Mean drainage (mL)**	
	Mean	**SD**
Day 1	34.18	32.49
Day 2	15.68	9.24
Day 3	11.68	8.22
Total	80.13	54.89

In the present study mean drainage on day one was 34.18 ± 32.49 mL, on day two it was 15.68 ± 9.24 mL and on day three 11.68 ± 8.22 mL. Overall the mean drainage was recorded as 80.13 ± 54.89 mL.

Table 13. Culture

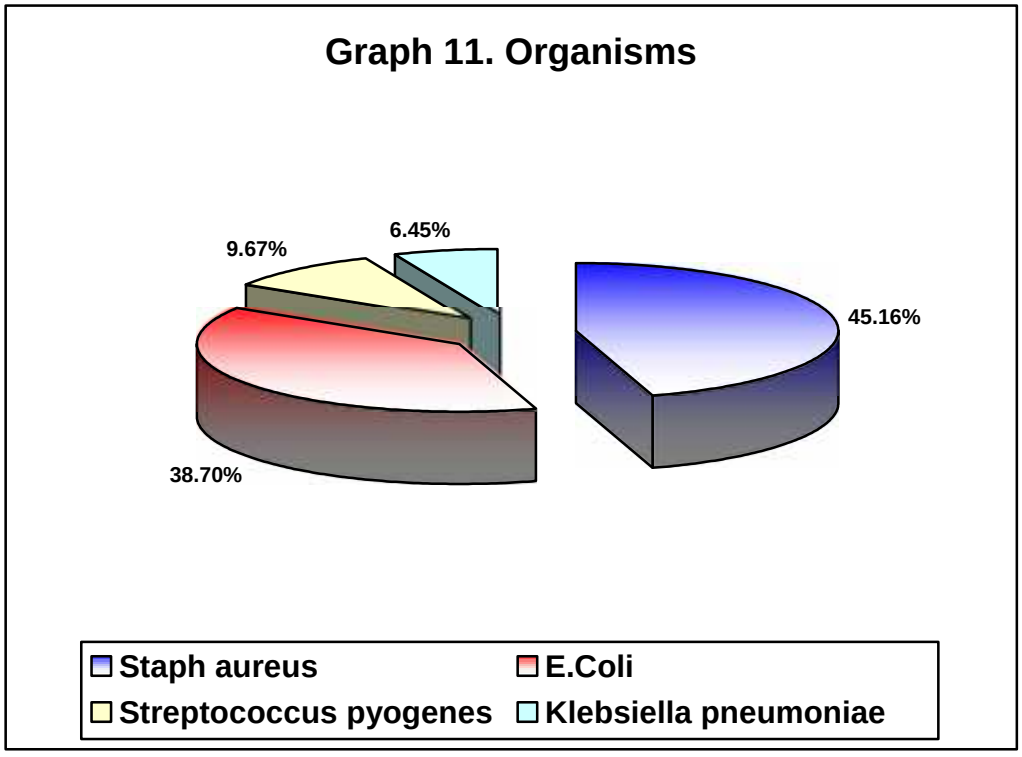
Culture	Distribution (n=40)	
	Number	**Percent**
Positive	31	77.50
Negative	9	22.50
Total	**40**	**100.00**



In this study, among 31 (77.50%) patients organisms were seen.

Table 14. Organisms

Organisms	Distribution (n=31)	
	Number	**Percent**
Staph aureus	14	45.16
E.Coli	12	38.70
Streptococcus pyogenes	3	9.67
Klebsiella pneumoniae	2	6.45
Total	**31**	**100.00**



In this study, among nine patients with organisms, the commonest organisms isolated were staph aureus (45.16%) followed by E. coli (38.70%), streptococcus pyogenes (9.67%), and klebsiella pneumonia (6.45%).

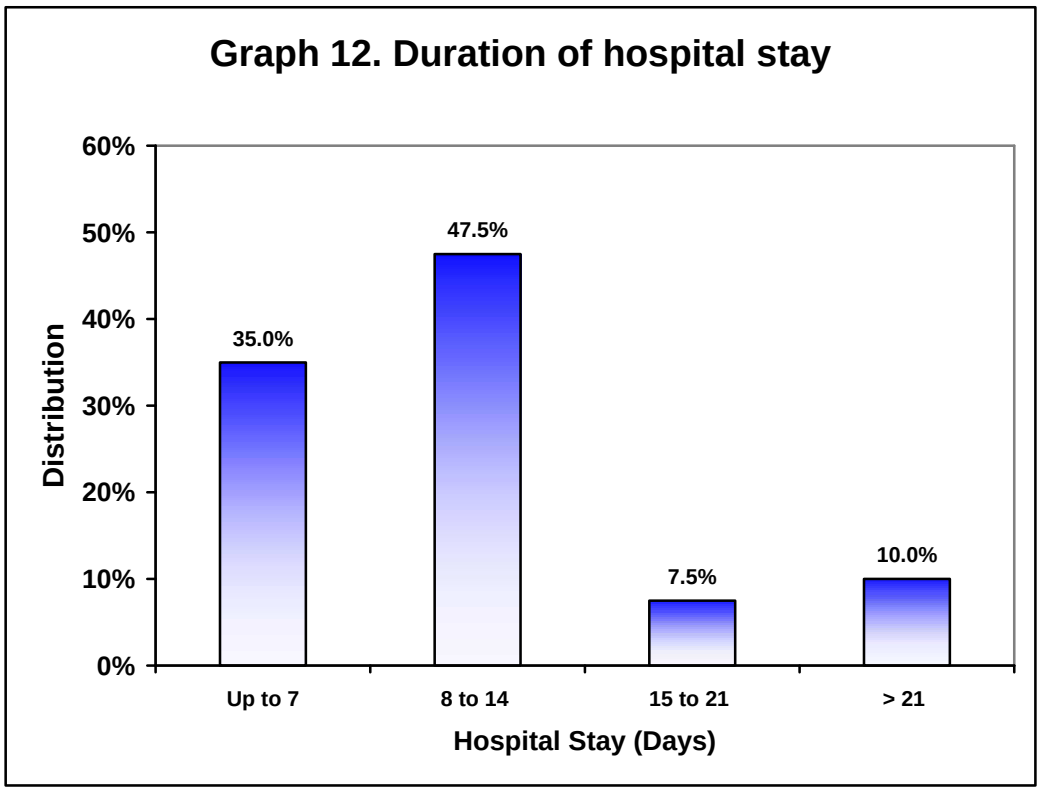
Table 15. Sensitivity

Drug	Distribution	
	Number	**Percent**
Imipenam	8	25.80
Amikacin	7	22.58
Ertapenam	4	12.90
Erythromycin	2	6.45
Cefodroxil	2	6.45
Cefelexin	2	6.45
Ciprofloxacin	2	6.45
Azithromycin	1	3.22
Ampicillin	1	3.22
Ofloxacin	1	3.22
Penicillin	1	3.22
Total	**31**	**100**

In this study, among 31 patients with organisms, 25.8% were sensitive to imipenam, 22.58% to amikacin and 12.90% to ertapenam The other drugs showing sensitivity to organisms are as shown in table.

Table 16. Duration of hospital stay

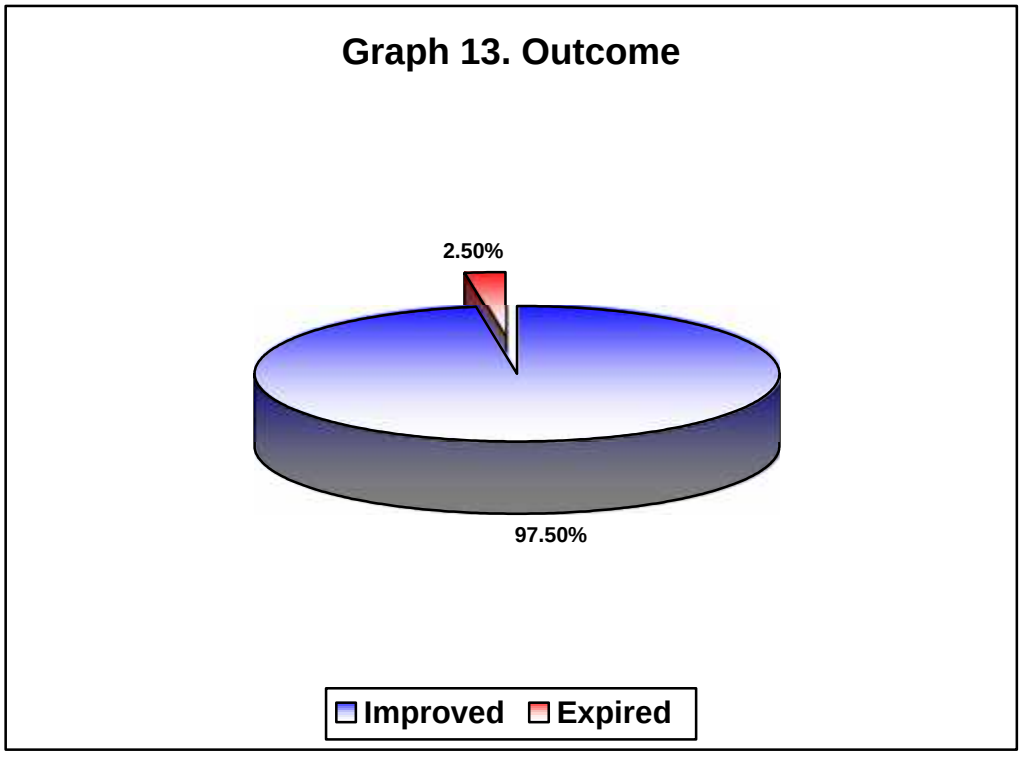
Duration (Days)	Distribution (n=40)	
	Number	**Percent**
Up to 7	14	35.00
8 to 14	19	47.50
15 to 21	3	7.50
> 21	4	10.00
Total	**40**	**100.00**



In the present study 47.5% had hospital stay between 8 to 14 days and 35.0% stayed less than seven days. However, 7.5% and 10% of patients required hospital stay of 15 to 21 days and more than 21 days respectively. Overall the mean duration of hospital stay was 11.08 ± 6.32 days.

Table 17. Outcome

Outcome	Distribution (n=40)	
	Number	**Percent**
Improved	39	97.50
Expired	1	2.50
Total	**40**	**100.00**



In this study out of 40 patients with abscess 39 (97.5%) improved and one patient (2.5%) expired.

Chapter 6

Discussion

DISCUSSION

Intra-abdominal infections is one of the common causes of hospitalization.[1] It continues to be an important and serious problem in surgical practice. Appropriate treatment is often delayed because of the obscure nature of many conditions resulting in abscess formation, which can make diagnosis and localization difficult. Delayed diagnosis and treatment leads to increased mortality rates

Intra-abdominal abscesses are highly variable in presentation and the diagnosis in the postoperative period is difficult. Computed tomography (CT) scanning has greater than 95% accuracy and is the best diagnostic imaging method for abdominal abscess. The presence of ileus, dressings, drains, or stomas does not interfere with reliability. In patients who are critically ill, initial percutaneous drainage can control sepsis and improve hemodynamics before definitive surgical treatment (if this becomes necessary). A visualized collection may be sterile (bile, hematoma) or infected, and CT-guided aspiration is most helpful in distinguishing between these states.[45]

CT-guided drainage delineates the abscess cavity and may provide safe access for percutaneous drainage. Criteria for removal of percutaneous catheters include resolution of sepsis signs, minimal drainage from the catheter, and resolution of the abscess cavity as demonstrated by an ultrasonogram or a CT scan. Risk factors for morbidity and mortality include multiple surgical procedures, age older than 50 years, multiple organ failure, and complex, recurrent, or persistent abscesses.[1,46]

The present study was undertaken to assess the bacteriology, culture and sensitivity of the bacteria causing intra-abdominal abscess and feasibility of image guided diagnosis and therapy as the primary modality of treatment.

This one year cross sectional study was conducted on a total of 40 patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

In the present study the commonest (30%) age group was 36 to 50 years (30%) study. Single abscess was seen in 82.5% of patients whereas 17.5% had multiple abscesses. In the present study 77.5% cases underwent computed tomography and 22.5% underwent ultrasonography. 22.5% cases underwent bedside procedure whereas 77.5% in procedure room. Majority of the patients had hepatic abscess (82.50%). Single abscess was seen in 82.5% of patients whereas 17.5% had multiple abscesses. Drainage was done using 12 and 14 single tube except in one case where multiple tubes were used. There was no evidence of peritube leakage, tube dislodgement and haemorrhage. The mean duration of tube in situ was 5.97 ± 2.67days. In this study of peripheral smear, four cases (10%) revealed neutrophilic leukocytosis, three (7.5%) normocytic hypochromic and two cases (5%) with eosinophia. The mean drainage was recorded as 31.15 ± 16.35 mL. On day one was 34.18 ± 32.49 mL, on day two it was 15.68 ± 9.24 mL and on day three 11.68 ± 8.22 mL. Overall the mean drainage was recorded as 80.13 ± 54.89 mL. In our study, the commonest organisms were staph aureus 45.16% , E.coli 38.70% , streptococcus pyogenes 9.67% and klebesialla pnumoniae 6.45%. 8 patients were sensitive to imipenem, 7 patients were sensitive to amikacin, 4 patients to ertapenam, 2 patients were

sensitive to erythromycin ,cephalexin and cefodroxil each. And one patient was sensitive to azithromycin, ampicillin, ofloxacin and penicillin each. While 47.5% had hospital stay between 8 to 14 days and 35.0% stayed less than seven days. However, 7.5% and 10% of patients required hospital stay of 15 to 21 days and more than 21 days respectively. overall the mean duration of hospital stay was 11.08 ± 6.32 days. And one patient did not respond to PCD and underwent open drainage. Another patient expired due to multi organ dysfunction syndrome, septicemia and ARF secondary to intra abdominal abscesses with hypertension and type 2 diabetes mellitus.

In a study conducted by Lo RH[47] and others, sixty-four consecutive cases of hepatic abscess diagnosed over a 6-year period and all treated by CT/ USG guided percutaneous needle aspiration were analysed. All the patients received systemic antibiotics. 64 patients had in total 101 liver abscesses which averaged 4.7 cm in diameter. Two patients (3.1%) required surgery for associated biliary tract disease. Five patients (7.8%) died from septicaemia and/or serious condition or malignancy. The remaining 57 patients (89.1%) were successfully treated .45 cases (70.3%) showed fully-resolved abscess/es after a mean period of 69.9 days after initial aspiration; 12 patients (18.8%) showed markedly-shrunken abscess cavity size over an average of 30.7 days and all were asymptomatic on discharge from hospital. No correlation was demonstrated between number of abscesses and successful patient recovery--42 of 46 patients (91.3%) with solitary hepatic abscess recovered, 6 of 8 patients (75.0%) with 2 abscesses and 9 of 10 patients (90.0%) with more than 2 abscesses were successfully treated. Results demonstrated, the effectiveness of percutaneous needle aspiration and it should

be considered a first-line treatment in the management of liver abscess, irrespective of their number and sizes.

In a study conducted by Baek SY et al,[48] seventeen of the hepatic abscesses were caused by pyogenic organisms, Six by amoeba, and two by unknown organisms. Persistent fever, pain and tenderness in the right upper quadrant, and leukocytosis were the indications for multiple aspirations. Follow-up sonography was performed to evaluate the outcome of treatment.

In 16 cases (64%), the abscesses disappeared within a mean of 84 days. In eight cases (32%) with only partial follow-up, the patients were asymptomatic at the time of discharge and the abscesses were markedly smaller on the last follow-up sonograms (mean, 43 days). One patient (4%) did not respond to aspiration and had surgical drainage. The length of hospitalization varied from 5 to 42 days (mean, 22 days). In patients who became afebrile during the treatment, the fever had lasted from 0 to 10 days (mean, 3 days). Only one patient had a complication of the procedure, a pleural effusion that was treated conservatively.

Bennion et al.[49] and Baron et al.[50] reported the bacteriology of 12 patients with gangrenous, and 18 patients with perforated, appendicitis with associated peritonitis and recovered 21 genera and more than 40 species. They recovered 2.7 aerobes and 7.4 anaerobes per specimen, which is much higher than usually reported in the literature (one survey noted an overall mean of 1.2 aerobes and 0.9 anaerobes per patient specimen). Bennion et al.[49] recovered the following percentage of various bacteria in patients with gangrenous and perforated appendicitis, respectively: *E. coli*, 70%, 77%; viridans streptococci, 19%, 43%;

enterococci, 18%, 9%; group D streptococci, 7%, 27%; *Staphylococcus* spp., 15%, 11%; *Klebsiella*/*Enterobacter* spp., 11%, 7%; and *P. aeruginosa*, 11%, 18%. Subsequently, Baron et al.[50] compared the bacteriology of acute and complicated (perforated and gangrenous) appendicitis and noted that the bacteria isolated were similar to those noted above but that 'some bacteria traverse the intact appendiceal wall prior to perforation' and that subsequent perforation allows more bacteria into the peritoneal cavity.

In a study carried out at two community medical centres, Goldstein & Citron14 noted that the *B. fragilis* group accounted for 34.6% of all anaerobes isolated (relative frequency), of which *B. fragilis* itself was the most common isolate, accounting for 18.9%. *B. fragilis* was more likely to be associated with bacteraemia, accounting for 46% of intra-abdominal isolates.

Brook[51] found similar results for children with perforated appendicitis. In their studies, Bennion et al.[49] and Baron et al.[50] isolated a previously undescribed anaerobic bacterium, now named *Bilophila wadsworthia*, in approximately half of the patients. Further, they reported the following percentage of various anaerobic bacteria in patients with gangrenous and perforated appendicitis, respectively: *B. fragilis*, 58%, 83%; *B. thetaiotaomicron*, 50%, 83%; *Peptostreptococcus micros*, 33%, 72%; and *Bacteroides splanchnicus*, 42%, 39%. This study had excellent microbiology but suffered from the small number of patients studied; hence, broad conclusions should not be drawn from this unique patient population from a single medical facility.

In the most recent published findings of the survey of *Bacteroides* susceptibility to antimicrobial agents, metronidazole, imipenem, meropenem, ertapenem, ampicillin–sulbactam, piperacillin–tazobactam and ticarcillin–clavulanate have maintained excellent activity. Increased resistance to the quinolones, including trovafloxacin and clinafloxacin, has been noted. The newest quinolone, moxifloxacin, has shown resistance rates strikingly similar to those of trovafloxacin for *Bacteroides* species. Although imipenem metallo-β-lactamase, which can confer resistance to all current carbapenems, has been reported in Japan, its presence in the USA and Europe has been quite limited. In addition, although metronidazole resistance genes have been reported in Europe, they have not been common in the USA, and metronidazole resistance has been very rare in *Bacteroides*.

Our study has demonstrated PCD as safe and effective means of drainage of intra abdominal abscesses. In this study we have used polymed catheter keeping in mind the cost and availability of the catheter, however other catheters are also available. As our study is on drainage of intra abdominal solid organs, usage of this tube in other abscesses like interloop abscess and pelvic abscess needs further study.

Chapter 7

Conclusion

CONCLUSION

Percutaneous drainage is safe, effective and feasible treatment option for the management of intra abdominal abscesses. CT being the most sensitive imaging modality for visualizing intra abdominal abscesses should be used to guide percutaneous drainage. The most common organism associated with intra abdominal abscesses is staphylococcus aureus.

Chapter 8

Summary

SUMMARY

Intra-abdominal abscess continues to be an important and serious problem in surgical practice. Diagnoses of intra-abdominal abscesses is often difficult as compared to peritonitis. Management of intra-abdominal abscess consists of drainage of abscesses which can be percutaneous drainage or surgical drainage. Percutaneous drainage consists of either USG guided or CT guided drainage, because abscesses contain high concentrations of bacteria in an environment that is hostile to the biologic action of most antibiotic therapy, drainage of the intra-abdominal abscess is essential for patient recovery. The present study was undertaken to assess the feasibility of percutaneous drainage as the primary method to treat intra abdominal abscess through image guided diagnosis.

This one year cross sectional study was conducted on a total of 40 patients admitted with intra abdominal abscesses in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Data obtained was tabulated on a excel spread sheet analysed.

In the present study the commonest (30%) age group was 36 to 50 years (30%) study. Single abscess was seen in 82.5% of patients whereas 17.5% had multiple abscesses. In the present study 77.5% cases underwent computed tomography and 22.5% underwent ultrasonography. 22.5% cases underwent bedside procedure whereas 77.5% in procedure room. The mean drainage was recorded as 31.15 ± 16.35 mL. On day one was 34.18 ± 32.49 mL, on day two it was 15.68 ± 9.24 mL and on day three 11.68 ± 8.22 mL. The commonest

organisms were staph aureus 45.16%, E.coli 38.70%, streptococcus pyogenes 9.67% and klebesialla pneumoniae 6.45%. Overall the mean duration of hospital stay was 11.08 ± 6.32 days.

Percutaneous drainage is safe, effective and feasible treatment option for the management of intra abdominal abscesses. CT being the most sensitive imaging modality for visualizing intra abdominal abscesses should be used to guide percutaneous drainage.

Chapter 9

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Annexures

Annexure I

ANNEXURE I – CONSENT FORM

RESEARCH PARTICIPANT INFORMATION AND CONSENT FORM

Introduction

Mr. Miss/Mrs._____

your are invited to participate in our research study that is **"A Cross-sectional study of image guided percutaneous drainage of intra-abdominal abscesses, of patients admitted to Dr. Prabhakar Kore Hospitals and MRC"**.

Since you have been diagnosed to be suffering from intra abdominal abscess, you will be requiring treatment for the same, you are eligible to be part of the study and hence asked to participate. This research is to study the efficacy of the tube drainage of intra abdominal abscess and the result of this research will help in a better treatment of similar participants in the future.

If you agree to be part of this research, we would ask you some relevant clinical history. You are free to avoid the questions which you think are not relevant. A thorough history will be collected and clinical examination will be done. You will be asked to undergo CT/USG in order to determine the exact location, size and no of abscesses and also to find out the safest and nearest accessible window for the drainage procedure.

You will undergo a drainage procedure with the help of CT/USG, where under local anasthesia a trocar will be inserted percutaneously through the nearest accessible window directly into the abscess capacity and the pus will be sent to culture and sensitivity, and indwelling tube will be kept in place.

Your decision of whether or not to participate in this study will not effect the quality of treatment you receive. Further you may withdraw from the study at any time.

All the new information collected about you during this course of study will be kept confidential to the extent permitted by law. Any information which identifies you personally, will not be released without your written consent.

This study does not have any damaging aspect and there are no chances of injury during the course of the study, but if injured the investigator is not responsible. There will be no extra cost incurred by you. However you will have to pay for the routine investigations, which are a part of the existing management compensation for the participant. The participation in this study is entirely voluntary and you may withdraw from the study at any time. At any time during or after the study, for any information you may contact the researcher.

Dr. ***** ******
J.N. Medical College,
Nehru Nagar, Belgaum.
Karnataka.
Phone No. ***** ******.

OR

Chairman,
Institutional Ethics Committee,
Phone No. ****** *****.

Signature of the participant or legally authorized representative:

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name : _____ Signature : _____

Investigators Name : _____ Signature : _____

Date : _____ Place : _____

Annexures

Annexure II

ANNEXURE II – PROFORMA

1) PATIENT IDENTIFICATION DATE:

NAME IP/OPD NO.

AGE DOA:

SEX DOD:

OCCUPATION

ADDRESS

2) presenting complaints with duration

3) Past history:

past history of similar complaints

past history of surgery/trauma of abdomen

history of any treatment

other contributory factors.

4) Personal history

DIET

APPETITE

SLEEP

BOWEL AND BLADDER

HABITS

5) In case of females

Obstetric history

Gynecological history

6) Family history

7) Drug history/history of allergy

8) GENERAL PHYSICAL EXAMINATION

PULSE

BP

PALLOR

ICTERUS

CYANOSIS

TEMPERATURE

RESPIRATORY RATE

9) ABDOMINAL EXAMINATION

INSPECTION

PALPATION

PERCUSSION

AUSCULTATION

MOVEMENTS AND MEASUREMENTS

10) PER RECTAL EXAMINATION -

11) SYSTEMIC EXAMINATION -

12) INVESTIGATIONS

BLOOD INVESTIGATIONS

- CBC
- BT

- CT
- PT
- INR
- LFT { OPTIONAL}

RADIOLOGICAL INVESTIGATIONS

- CT
- USG
- CHEST XRAY (SOS)
- ABDOMEN XRAY (SOS)

CULTURE AND SENSITIVITY EXAMINATION OF PUS.

CT/USG FINDINGS :

Location of the abscess and extension :

Size of the abscess :

Single / multiple :

Contents :

FOLLOWUP CT/USG (POST PROCEDURE)

Size of the abscess :

Diagnosis :

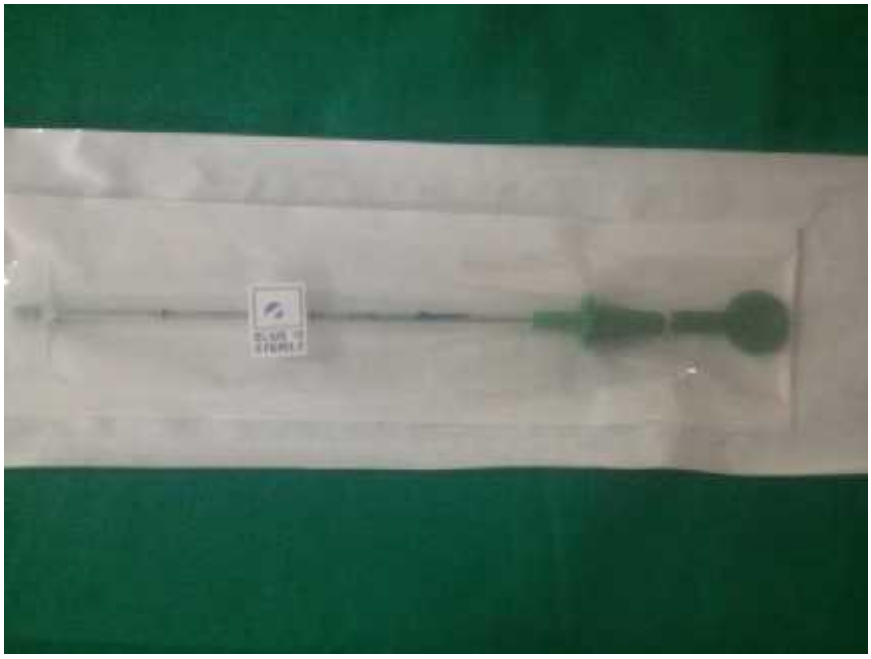
Outcome :

Details of the procedure : (Including size of tube used ,no of days tube kept ,amount of pus drained , mezol wash)

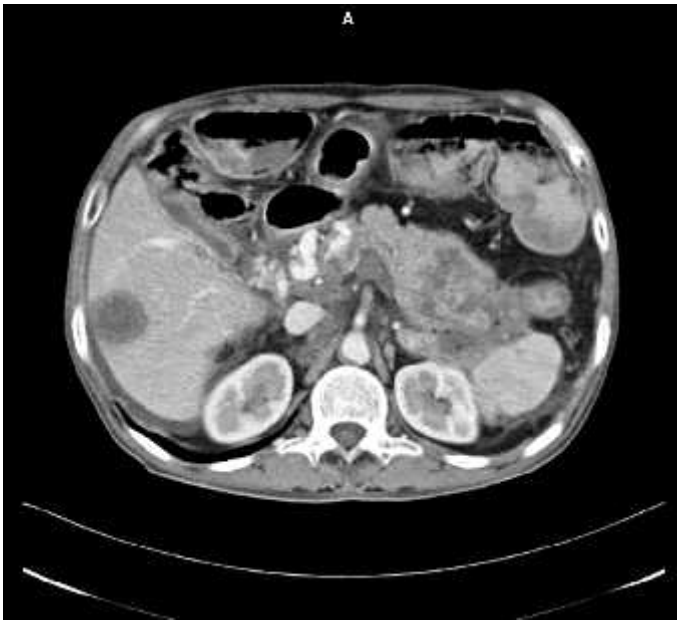
Annexures

Annexure III

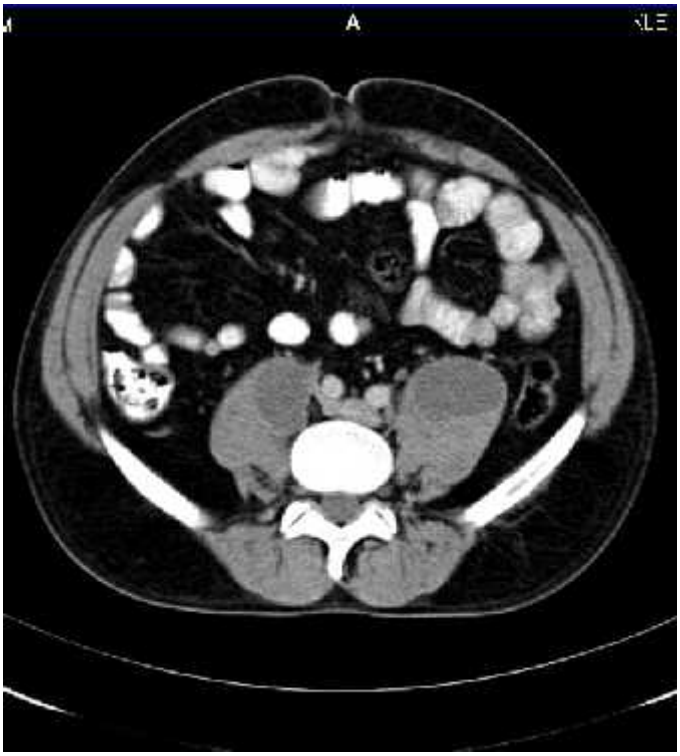
ANNEXURE III – PHOTOGRAPHS



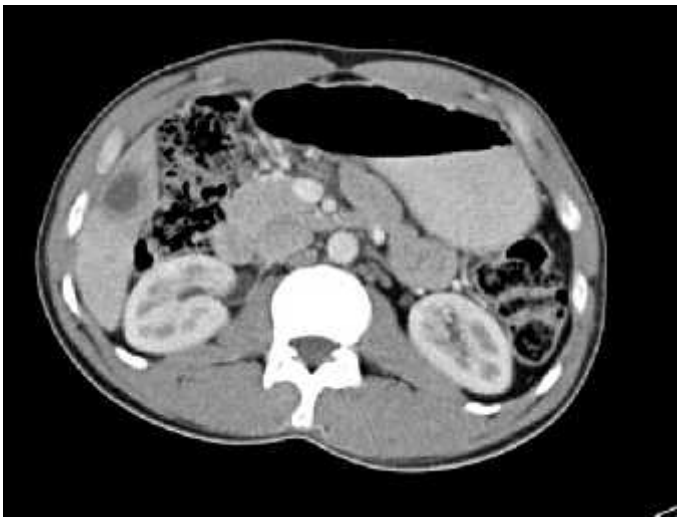
Photograph 1. Percutaneous drainage trocar with catheter



Photograph 2. Hepatic Abscess



Photograph 3. Psoas abscess



Photograph 4. Hepatic abscess

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Date of admission	Date of Discharge	Complaint		History			Investigations													
																		Liver function tests					
						Pain Abdomen (Days)	Fever (Days)	Trauma	Previous Surgery	Contributing factors	Haemoglobin (gm%)	Total count (/mm3)	Platelet count (/mm3)	Serum Urea (mg/dL)	Serum Creatinine (mg/dL)	Bleeding time (Sec)	Clotting time (Sec)	Total bilirubin (mg/dL)	Direct Bilirubin (mg/dL)	Prothrombin time	International noramlized ratio	Peripheral smear	Total protein (gms)
1	363434	M	30	24.04.10	29.04.10	30	30	-	-	-	11.3	8000	3.35	20	0.8	2:30	3:30	0.7	0.2	18.3	1.28	-	6.5
2	331854	M	35	29.01.10	05.02.10	9	5	-	-	-	11.6	9000	3.62	16	0.8	2:30	4:00	0.7	0.3	22.9	1.74	Eosinophilia	6.9
3	359541	M	58	27.12.09	08.01.10	3	4	-	-	-	10.4	11600	3.25	38	0.9	3:00	5:00	3.3	2.9	22.9	1.6	Toxic leukocytes	6.8
4	363534	M	56	28.04.10	06.05.10	8	7	-	-	-	7.4	8200	2.1	11	0.8	3:00	5:00	0.7	0.3	16.5	1.13	-	6.2
5	367844	M	35	24.02.10	06.03.10	6	5	-	-	-	13	13100	2.8	41	1.2	2:30	4:00	0.7	0.6	18.5	1.3	-	5.9
6	391462	M	65	08.11.10	01.12.10	15	7	-	+	HTN	8.7	9200	2.4	10	0.5	2:30	3:30	0.4	0.17	21.9	1.56	-	5.6
7	395104	M	41	02.12.10	05.12.10	7	3	-	-	-	11.7	11000	1.9	16	0.8	3:00	4:00	0.9	0.4	22.9	1.65	-	5.9
8	350924	M	72	21.01.10	01.02.10	7	5	-	-	T2DM	9.5	22800	8.84	30	0.9	2:30	3:30	0.7	0.4	18.9	1.3	-	7.1
9	356433	F	35	06.03.10	11.03.10	60	30	-	Stenting	-	10.4	11600	2.2	22	0.8	2:30	5:00	0.8	0.5	21	1.4	-	6
10	380102	M	22	17.08.10	14.09.10	90	21	-	-	-	11.5	7600	2.12	16	0.9	2:30	3:00	0.7	0.4	20.1	1.33	-	7
11	355040	M	58	23.02.10	24.02.10	7	7	-	Colonoscopy	-	10.2	16700	2.01	20	1	2:30	4:00	0.9	0.3	18.3	1.28	-	4.4
12	345871	M	62	11.02.09	17.12.09	7	5	-	-	ALD,HTN	11.5	35000	81000	24	0.9	3:00	5:00	2.2	1.8	25.9	1.81	-	5.1
13	347434	M	40	09.01.10	28.01.10	4	7	-	-	-	13	19100	5.98	8.6	1.1	2:00	3:00	5	4	18.5	1.3	Neutrophilic leukocytosis	5.2
14	363533	M	34	28.04.10	06.05.10	6	3	-	-	-	11.4	19000	4.89	22	1	2:00	3:00	0.7	0.3	16.5	1.13	-	6.8
15	363510	F	84	26.04.10	03.05.10	10	5	-	Hysterectomy	T2DM,HTN	12.12	60000	2.3	168	2	3:00	5:00	1.2	0.9	18.4	1.3	-	5.8
16	366851	M	40	21.03.10	31.03.10	15	15	-	-	-	11.5	12800	2.8	4.47	0.9	2:30	3:00	0.7	0.5	21	1.4	-	5.8
17	356512	F	50	10.03.10	17.03.10	6	4	-	Tubectomy	-	12	10800	2.6	18	0.6	2:30	3:30	0.8	0.5	17.4	1.2	Neutrophilic leukocytosis	6.2
18	373743	F	60	26.07.10	16.08.10	60	30	-	-	-	7	13500	2.97	27	1.1	2:30	3:30	0.9	0.4	24	1.7	-	6
19	363683	F	46	24.04.10	03.05.10	365	15	-	-	-	10.4	22700	2.3	17	0.6	3:00	4:00	1.7	1	29	1.25	-	5.8
20	390530	M	70	30.10.10	15.11.10	10	7	-	-	HTN	9.6	5100	3.4	14	0.8	2:30	3:30	1.8	1.2	29.8	2.22	-	6
21	399872	M	49	07.01.11	02.02.11	8	3	-	-	-	8	16200	2.12	22	0.8	3:00	6:00	0.9	6.2	21.6	1.39	Microcytopenia, Neutrophic leukocytosis	6.7
22	381552	F	17	28.08.10	01.09.10	2	2	-	-	-	8.3	25800	1.2	20	0.8	2:30	3:30	0.7	0.3	18.1	1.23	-	6.2
23	373152	M	55	03.07.10	17.07.10	8	20	-	-	-	9	12500	2.02	26	0.8	2:30	3:30	0.8	0.3	18.4	1.3	-	6.3
24	361201	M	36	12.04.10	24.04.10	45	45	-	-	-	11.7	11000	4.61	18	0.7	3:00	4:00	0.9	0.3	17.4	1.2	-	6.3
25	350050	M	24	14.01.11	19.01.11	5	4	-	-	Tuberculosis	13.1	21400	2.12	21	0.8	2:30	3:30	1	0.3	20.2	1.52	Toxic granules	6.9
26	334789	M	75	17.09.09	29.09.09	4	4	-	-	HTN	9.8	12500	3.82	11	0.9	3:00	5:00	1.7	1.5	15.6	1.06	-	5.7
27	351004	M	36	22.01.10	04.02.10	30	30	-	-	-	12	9900	3.49	24	1	3:00	5:00	0.6	0.4	18.1	1.2	-	6.3
28	373880	M	19	08.07.10	22.07.10	60	15	-	Liver Abscess	-	13	18600	1.84	20	0.7	3;00	4:30	0.8	0.5	16.5	1.13	Eosinophilia	6

ANNEXURE IV - MASTER CHART

Serial Number	Sr. Albumin (gms)	Albumin:Globulin Ratio	SGOT	SGPT	Alkaline phosphatase	Location			Associated medical conditions																				Imaging		Proc		Abscess			
						Hepatic abscess	Spleenic abscess	Psoas abscess	Alcoholic liver disease	Spleenomegaly	Hepatomegaly	Cholecystitis	Ascites	Tuberculosis	Pneumonia	Typhoid	Septicemia	Perinephric abscess	Pyelonephritis	Diabetes mellitus	Hypertension	GI Bleed	Pleural effusion	Para aortic lymphadenopathy	Acute renal failure	Inguinal hernia	Computed tomography	Ultrasound	Bedside	Procedure Room	Location	Size	Single/multiple	Contents
1	3.3	0.6	29	24	97	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	5X4	SL	+
2	2.4	o.4	36	34	168	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	II,IVA	7.1X6.9X8	SL	+
3	2.6	0.6	27	32	160	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VI,VII	10X8X8	SL	+
4	2.1	0.6	26	30	102	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	V,VI	6.5X5.5	SL	+
5	2.5	0.7	18	50	98	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	RT LOBE	4X4	SL	+
6	1.2	0.3	26	26	140	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	-	+	VI,VII	7.5X5.7X4.8	SL	+
7	2.5	0.8	545	72	102	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VI,VII	10X7.6	SL	+
8	1.5	0.3	22	31	118	-	-	+	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	+	-	-	+	LT PSOAS	8.1X12.8;3X1.4	ML	+
9	1.1	0.3	43	26	93.1	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+	IV	4.3X3.3	SL	+
10	1.2	0.4	48	30	90	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	BL PSOAS	4X3.1X2;2X1.5X2	ML	+
11	2.3	1.1	17	26	128	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	VII,VI	6.3X4.8	SL	+
12	1.7	1.2	102	55	189	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	VI	5X4.3X4	SL	+
13	1.5	0.4	35	30	151	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	II,III,IV	9X8.7X7.6;0X4.2	ML	+
14	2.6	0.6	27	32	160	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	II,IVA	11X5.3X6.7	SL	+
15	1.4	0.6	173	134	220	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	+	-	-	-	+	+	-	+	+	-	CORDATE	8X6X7;6X5X3	ML	+
16	2.2	0.6	50	39	96	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	RT LOBE	7X6	SL	+
17	2.3	0.6	16	15	110	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	rt lobe	5X4	SL	+
18	0.5	0.5	58	19	90	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	psoas iliacus	5X4.3	SL	+
19	1.8	0.4	73	69	173	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	RT LOBE	3X5	SL	+
20	1.7	0.3	41	30	109	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VII,VIII	12.2X11X8.6	SL	+
21	1.7	0.8	92	86	166	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VI,VII	7X6.5X6.4;9X7.5X6.5	ML	+
22	2.7	0.8	29	23	209	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	4X3.5X3.6	SL	+
23	3.3	1.3	68	40	122	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VII,VI	5.4X4.1X4;4.1X3.2X3	ML	+
24	2.7	0.8	57	63	227	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	V,VIII	10X5	SL	+
25	2.6	0.7	25	33	100	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VII	8X6.5X4	SL	+
26	2.2	0.6	54	64	123	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	+	VII VIII	4X3.5X3.6	SL	+
27	2.6	0.7	41	44	120	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	6X4.2X4	SL	+
28	3	0.1	81	61	135	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	10X7.5X6	SL	+

ANNEXURE IV - MASTER CHART

Serial Number	Follow up CT/USG (after 48 hrs)	Pus			Drainage					Tube details						Outcome								
	Size of abscess cavity	Amount drained	Isolated organisms	Sensitivity	Therapeutic/diagnostic	1st Day	2nd Day	3rd Day	Total drainage	Size	Number	Peritube leakage	Tube dislodgement	Duration of Tube in situ	Complications	Hospital stay (Days)	Drainability	Symptomatic relief in 24 hrs	Need for repeated drainage	Residual abscess	Resolution of the abscess	Laparotomy	Expired	Improved
1	2X2	15	staph aureus	IMIPENAM	T	5	3	3	26	12	1	-	-	3	-	3	+	+	-	-	+	-	-	+
2	4.5X4.1	25	staph aureus	AMIKACIN	T	25	8	3	61	12	1	-	-	6	-	8	+	+	-	-	+	-	-	+
3	8X6.1X4	45	E.COLI	AMIKACIN	T	30	30	20	125	14	1	-	-	11	-	12	+	+	-	-	+	-	-	+
4	6X5	24	NO ORG	-	D	-	-	-	-	12	1	-	-	-	-	18	-	-	+	-	-	+	-	+
5	2X1	20	staph aureus	IMIPENAM	T	30	3	2	55	12	1	-	-	3	-	12	+	+	-	-	+	-	-	+
6	3.5X2.1X3	40	E.COLI	IMIPENAM	T	20	10	10	80	12	1	-	-	14	-	22	+	+	-	-	+	-	-	+
7	3X2.2	20	NO ORG	-	T	30	15	5	70	12	1	-	-	7	-	4	+	+	-	-	+	-	-	+
8	3.1X2.8	40	Staph aureus	IMIPENEM	T	15	5	3	63	14	2	-	-	5	-	7	+	+	-	-	+	-	-	+
9	4.1X3	14	klebsiella pneumonia	ERTAPENAM	T	14	10	6	42	12	1	-	-	1	-	5	+	+	-	-	+	-	-	+
10	2X1.5X0.5;1X05X1.0	30	E.COLI	AMIKACIN	T	20	15	5	70	14	2	-	-	10	-	27	+	+	-	-	+	-	-	+
11	3.8X2.8	40	staph aureus	ERYTHROMYCIN	T	60	15	15	130	14	1	-	-	8	Air pockets	6	+	+	-	-	+	-	-	+
12	3X2X2.5	5	NO ORG	-	T	10	3	3	21	12	1	-	-	4	-	6	+	+	-	-	+	-	-	+
13	8X8.1X7.1;5X3.1	40	staph aureus	CEFELEXIN	D	90	30	25	185	12	2	-	-	1	-	19	+	+	-	-	-	+	-	+
14	7.4X4.5	70	E.COLI	AMPICILLIN	T	15	15	10	110	12	1	-	-	11	-	11	+	+	-	-	+	-	-	+
15	3.2X4.1	40	staph aureus	OFLOXACIN	T	60	20	16	136	12	2	-	-	1	-	1	+	+	-	-	+	-	+	-
16	3X2	25	Staph aureus	AMIKACIN	T	35	10	10	80	12	1	-	-	5	-	10	+	+	-	-	+	-	-	+
17	3X3	8	streptococcus pyogenes	IMIPENAM	T	15	5	5	33	12	1	-	-	5	-	7	+	+	-	-	+	-	-	+
18	3X2	8	streptococcus pyogenes	ERYTHROMYCIN	T	7	5	3	23	12	1	-	-	4	-	10	+	+	-	-	+	-	-	+
19	3X2	30	E.COLI	CIPROFLOXACIN	T	48	15	5	98	12	1	-	-	4	-	5	+	+	-	-	+	-	-	+
20	4X3.5X3	14	staph aureus	AMIKACIN	T	40	10	8	72	12	1	-	-	7	-	15	+	+	-	-	+	-	-	+
21	3X2.5X2;4X2.6X2.4	25	NO ORG	-	T	50	10	10	95	12	2	-	-	7	-	25	+	+	-	-	+	-	-	+
22	2X2.5X3	10	E.COLI	ERTAPENAM	T	15	8	5	38	12	1	-	-	5	-	11	+	+	+	+	-	-	-	+
23	2X3X2.1;2.2X1.0X1.6	8	klebsiella pneumonia	AMIKACIN	T	20	15	5	48	12	2	-	-	7	-	14	+	+	-	-	+	-	-	+
24	4X2	10	E.COLI	CEFELEXIN	T	200	35	30	265	12	1	-	-	7	-	12	+	+	-	-	+	-	-	+
25	5X3.2X2	14	NO ORG	-	T	35	20	18	87	12	1	-	-	6	-	6	+	+	-	-	+	-	-	+
26	3X2.1X2	8	staph aureus	PENICILLIN	T	15	12	10	45	12	1	-	-	5	-	12	+	+	-	-	+	-	-	+
27	4X2X2	12	E.COLI	IMIPENAM	T	35	20	12	79	12	1	-	-	7	-	12	+	+	-	-	+	-	-	+
28	7X4.5X4.1	30	Staph aureus	CEFODROXIL	T	22	13	10	75	12	1	-	-	7	-	14	+	+	-	-	+	-	-	+

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Date of admission	Date of Discharge	Complaint		History			Investigations													
																		Liver function tests					
						Pain Abdomen (Days)	Fever (Days)	Trauma	Previous Surgery	Contributing factors	Haemoglobin (gm%)	Total count (/mm3)	Platelet count (/mm3)	Serum Urea (mg/dL)	Serum Creatinine (mg/dL)	Bleeding time (Sec)	Clotting time (Sec)	Total bilirubin (mg/dL)	Direct Bilirubin (mg/dL)	Prothrombin time	International noramlized ratio	Peripheral smear	Total protein (gms)
29	334924	M	17	20.09.10	27.09.10	15	15	-	-	-	11.5	7800	2.14	17	1	2:00	5:00	0.7	0.5	23.3	1.62	-	5.2
30	384374	M	32	16.09.10	22.09.10	4	4	-	-	-	8.9	8400	5.46	20	0.9	2:30	3:00	0.7	0.4	20.1	1.5	Normocytic hypochromic anaemia	6.3
31	362116	M	19	18.04.10	01.06.10	30	30	-	Lt .Thigh Abscess	-	10	10200	1.52	20	0.7	2:00	4:00	0.8	0.4	20	1.4	-	6
32	377958	M	18	03.08.10	08.08.10	60	30	RTA	-	-	13.1	9600	1.89	20	0.8	2:30	3:30	0.7	0.5	20.1	1.42	-	6.2
33	381930	M	76	29.08.10	08.09.10	5	5	-	ERCP+CBD STENT	-	16.8	28700	4.2	34	1.2	2:30	3:30	8.8	7.6	1.89	1.2	Neutrophilic leukocytosis	5.2
34	400216	M	73	10.01.11	18.01.11	8	7	Fall	-	T2DM,HTN	12.7	19100	2.29	26	0.9	3:30	4:30	0.8	0.3	13.8	1.2	-	6
35	395455	M	53	04.12.10	14.12.10	2	2	-	-	-	8.6	11600	3.38	128	8.3	2:30	3:30	0.7	0.3	18.5	1.4	-	7
36	404475	M	50	04.02.11	18.02.11	60	60	-	-	-	11.7	7000	2.8	12	0.6	2:30	3:30	0.3	0.1	16.4	14.3	Normocytic hypochromic, Neutrophilia	6.5
37	420689	M	45	02.06.11	11.06.11	7	4	-	LIVER ABSCESS	-	9.3	9400	2.78	10	0.4	3:30	4:30	0.73	0.28	11.2	97	Normocytic hypochromic anaemia	7.2
38	412931	M	40	11.06.11	09.07.11	10	2	-	-	-	10.3	12400	2.55	23	0.29	2:30	4:00	0.63	0.25	10.5	1	-	6.9
39	421931	F	45	11.06.11	17.07.11	5	6	-	-	-	11.7	9500	8.7	13	0.8	2;30	3:30	0.7	0.4	24	1.25	Microcytic hypochromic anaemia	6.5
40	364741	M	62	06.05.10	20.05.10	30	5	-	ERCP	HTN	9	23500	1.52	58	0.9	2:30	5:30	21.4	17.3	19.16	1.34	-	6.2

ANNEXURE IV - MASTER CHART

Serial Number	Sr. Albumin (gms)	Albumin:Globulin Ratio	SGOT	SGPT	Alkaline phosphatase	Location			Associated medical conditions																					Imaging		Proc		Abscess			
						Hepatic abscess	Spleenic abscess	Psoas abscess	Alcoholic liver disease	Spleenomegaly	Hepatomegaly	Cholecystitis	Ascites	Tuberculosis	Pneumonia	Typhoid	Septicemia	Perinephric abscess	Pyelonephritis	Diabetes mellitus	Hypertension	GI Bleed	Pleural effusion	Para aortic lymphadenopathy	Acute renal failure	Inguinal hernia	Computed tomography	Ultrasound	Bedside	Procedure Room	Location	Size	Single/multiple	Contents
29	1.7	1.1	60	49	120	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	VIII	4X4.1X5	SL	+
30	3.3	1.3	60	40	130	+	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	5X6.4X6	SL	+
31	2.7	0.8	71.9	35	115	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	10X6X5.1	SL	+
32	2.3	0.8	62	38	123	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	SPLEEN	4X5.2X4.2	SL	+
33	2.6	0.3	33	72	223	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT PSOAS	7.6X7X5.8	SL	+
34	2.5	0.5	60	62	175	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	+	-	-	+	SPLEEN	10.5X6.5X5	SL	+
35	2.8	0.6	50	62	134	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	LT PSOAS	20X5.6	SL	+
36	2.34	0.5	16	24	125	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	7X5.5X6	SL	+
37	2.7	0.6	34	44	163	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	5X6.4X5.5	SL	+
38	3	0.7	68	108	142	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	VII,VIII	8.3X7.8X7	SL	+
39	3.4	0.7	45	39	89	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	RT LOBE	7X5.3X4.5	SL	+
40	2.5	0.7	76	93	144	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	+	RT AND LT LOBE	5X4;3X2	ML	+

ANNEXURE IV - MASTER CHART

Serial Number	Follow up CT/USG (after 48 hrs)	Pus			Drainage					Tube details						Outcome								
	Size of abscess cavity	Amount drained	Isolated organisms	Sensitivity	Therapeutic/diagnostic	1st Day	2nd Day	3rd Day	Total drainage	Size	Number	Peritube leakage	Tube dislodgement	Duration of Tube in situ	Complications	Hospital stay (Days)	Drainability	Symptomatic relief in 24 hrs	Need for repeated drainage	Residual abscess	Resolution of the abscess	Laparotomy	Expired	Improved
29	3X2.4X2	12	NO ORG	-	T	15	10	4	41	12	1	-	-	4	-	7	+	+	-	-	+	-	-	+
30	3X.1X3	8	E.COLI	IMIPENAM	T	35	30	25	98	12	1	-	-	6	-	6	+	+	-	-	+	-	-	+
31	6X5X3.4	15	NO ORG	-	T	45	30	30	120	12	1	-	-	8	-	13	+	+	-	-	+	-	-	+
32	3X2.2X1.2	10	E.COLI	IMIPENAM	T	25	15	12	62	12	1	-	-	6	-	5	+	+	-	-	+	-	-	+
33	5X4.3X4	15	staph aureus	CIPROFLOXACIN	T	15	12	8	50	12	1	-	-	5	-	9	+	+	-	-	+	-	-	+
34	7X4X4.6	25	staph aureus	AZITHROMYCIN	T	30	20	16	81	12	1	-	-	7	-	8	+	+	-	-	+	-	-	+
35	17.5X4	40	NO ORG	-	T	45	30	20	135	12	1	-	-	7	-	10	+	+	-	-	+	-	-	+
36	4X3.3X3	15	E.COLI	ERTAPENAM	T	20	15	8	58	12	1	-	-	5	-	14	+	+	-	-	+	-	-	+
37	3X4X3.5	12	NO ORG	-	T	15	10	5	42	12	1	-	-	4	-	9	+	+	-	-	+	-	-	+
38	5X4.8X4.5	30	staph aureus	AMIKACIN	T	42	35	30	137	12	1	-	-	8	-	28	+	+	-	-	+	-	-	+
39	4X3X3.3	25	E.COLI	CEFODROXIL	T	35	27	16	103	12	1	-	-	6	-	6	+	+	-	-	+	-	-	+
40	2X2 ;	5	streptococcus pyogenes	ERTAPENAM	T	30	12	10	57	12	2	-	-	6	-	14	+	+	-	-	+	-	-	+

Annexures

Annexure IV

ANNEXURE IV – MASTER CHART

-	- Negative
+	- Positive
ALD	- Alcoholic liver disease
CBD	- Common bile duct
CT	- Computed tomography
D	- Diagnostic
dL	- Deci litre
E.COLI	- Escherichia coli
ERCP	- Endoscopic retrograde cholangio pancreatography
F	- Female
gm	- Grams
HTN	- Hypertension
M	- Male
mg	- Milli grams
ML	- Multiple
mm	- Millimeter
NO ORG	- No organisms
Proc	- Procedure
RTA	- Road traffic accident
sec	- Seconds
SGOT	- Serum glutamic oxaloacetic transaminase
SGPT	- Serum glutamic pyruvic transaminase
SL	- Single

Staph aur	- Staphylococcus auerus
T	- Therapeutic
T2DM	- Type 2 diabetes mellitus
USG	- Ultrasonography