# *STUDY OF INCIDENCE OF POSTOPERATIVE SEPSIS AFTER EMERGENCY ABDOMINAL SURGERIES*

**Dissertation submitted in partial fulfillment of the regulation for the award of M.S. Degree in General Surgery**

**(Branch I)**

**THE TAMILNADU**

## Dr. M. G. R. MEDICAL UNIVERSITY

**CHENNAI – 600 032.**

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## COIMBATORE MEDICAL COLLEGE

**COIMBATORE – 641 014.**

# CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. R.NANDHA KUMAR** and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The TamilNadu Dr. M.G.R. Medical University, Chennai

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## ETHICS COMMITTEE

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*Course* : M.S. General Surgery

*Period of Study* : 2007 - 2010

*College* : Coimbatore Medical College

*Dissertation Topic* : Incidence of post-operative sepsis after emergency abdominal surgeries.

*The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.*

*Coimbatore - 14.*

*Date :* 13.02.08

*Secretary*
*Ethics Committee*

# DECLARATION

I solemnly declare that the dissertation titled "**STUDY OF INCIDENCE OF POSTOPERATIVE SEPSIS AFTER EMERGENCY ABDOMINAL SURGERIES**" was done by me from 2007 onwards under the guidance and supervision of **Prof. Dr. G. MOHAN, M.S**.

This dissertation is submitted to the TamilNadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of M.S Degree in General Surgery (Branch I).

Place : **Dr. R.NANDHA KUMAR**

Date :

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# INTRODUCTION

Postoperative sepsis represents one of the most frustrating and difficult occurrences experienced by surgeons in the post operative period and it remain a significant cause of morbidity and mortality following emergency abdominal surgeries. It increases the cost of treatment and is associated with lost work productivity, disruption of normal life and unanticipated stress to patients in general.

Although preoperative predictive factors are well recognized, early recognition of postoperative sepsis remains problematic. The complex deregulated host response to infection includes uncontrolled inflammation and immune suppression. At its most basic level overt clinical infection represents a shift of balance of forces comprising defense and microbial invasion. Over the time, the virulence of infection amount of microbial inoculum, and host defense has occupied the interests of surgeons in their fight against infection.

Numerous studies have evaluated postoperative sepsis, but because of the complexity of the problem, some reports have limitations which prevent meaningful interpretation. Some overlook the necessity of rigorous statistical control to discriminate between the random effects of chance and relevant

clinical factors determining the incidence of postoperative sepsis. Other reports bulk together widely diversified surgical experience so that conclusions in regard to sepsis rates may be confounded by alterations in the case material from time to time. Such changes will affect calculated sepsis rates by the inclusion of various cases in different periods with greater or lesser propensities to develop postoperative sepsis.

Commonly a precise definition of surgical sepsis or the details as to the methods employed with appropriate checks are omitted. The low incidence of postoperative sepsis following clean surgery, in the order of one to five per cent, requires that many cases be collected to permit meaningful interpretation of the statistics. The complex interdependence of the factors contributing to the development of postoperative sepsis makes it extremely difficult to extract any one factor as the critical one among several hundred that could be responsible for a change in the incidence. These basic problems are difficult to resolve in the analysis of results, and although the present study has its own shortcomings, the incidence of postoperative sepsis has been studied in the patients who underwent emergency abdominal surgeries.

## AIM

- To study the incidence of post operative sepsis which includes SSI and major septicemia following emergency abdominal surgeries in all surgical units in Coimbatore medical hospital between August 2007 to September 2009
- To study the various preoperative, intraoperative and postoperative factors influencing post operative sepsis
- To study the microbiology of infection
- To study the mortality of post operative sepsis

# HISTORY

In the past, when medical hygiene was unknown, wound infections were a common and greatly feared complication of surgery. Wound sepsis was blamed to be the cause. The main historical aspects in the field of sepsis which has led to the present day concept of novel antiseptics and antimicrobials have been dealt here.

Though the term **Sepsis** is linked closely to modern intensive care, the medical concept is rather older.

The word "sepsis" was first introduced by Hippocrates (ca. 460-370 BC) and is derived from the Greek word **sipsi** ("make rotten"). **Ibn Sina** (979-1037 BC) observed the coincidence of blood putrefaction (septicaemia) and fever. This concept of sepsis which was introduced in classical antiquity was used until the 19th century. Only few examples of pathophysiological investigations are known.

**Herrmann Boerhave** (**1668-1738**), a doctor in **Leyden,** thought that toxic substances in the air were the cause for sepsis. At the beginning of the 19th century, the chemist **Justus von Liebig** expanded the theory by claiming that the contact between wounds and oxygen was responsible for the development of sepsis.

**Ignaz Semmelweis (1818-1865)** was the first researcher who developed a modern view of sepsis. He was an obstetrician at the Vienna General Hospital at a time when the death of women in childbed from puerperal fever was a common complication. His department had an especially high mortality rate of ca. 18 %. Semmelweis discovered that it was common for medical students to examine pregnant women directly after pathology lessons. Hygienic measures such as hand washing or surgical gloves were not customary practice.

Semmelweis deducted that childbed fever was caused by "decomposed animal matter that entered the blood system". As a matter of fact, he succeeded in lowering the mortality rate to 2.5 % by introducing **hand washing** with a chlorinated lime solution before every gynecological examination. However, in spite of the clinical success, the hygienic measures were not accepted, and colleagues harassed him, forcing him to leave the city. It took him until 1863, more than 15 years after his findings, to publish his work "Aetiology, terminus and prophylaxis of puerperal fever" (Die Aetiologie, der Begriff und die Prophylaxis des Kindbettfiebers). The failure to achieve a professional reputation and the unrelenting opposition of the medical establishment may have facilitated the development of psychiatric symptoms. Semmelweis was eventually committed to a lunatic

asylum where he died from a wound infection probably as a result of the beatings he underwent there. It is an irony of fate that he died from a disease that he dedicated his life to fight.

The French chemist **Louis Pasteur (1822-1895)** discovered that tiny single cell organisms caused putrefaction. He called them bacteria or microbes and correctly deducted that these microbes could be causing disease. He also made the significant discovery that bacteria in fluids could be killed by heating. This meant that a fluid could be sterilized.

**Joseph Lister (1827-1912)** worked as a surgeon at Glasgow Royal Infirmary. At the time when he became chairman of the surgery department about 50 % of patients with amputations died of sepsis. Lister drew a correlation between Semmelweis' observations, Pasteur's findings and the deaths in his hospital. By almost modern scientific studies, first with animals, then with humans, he examined the effects of skin and instrument disinfection with carbolic acid (the so-called antiseptic method). By doing so, Lister was able to drastically reduce post-amputation mortality. Unlike Semmelweis, Lister managed to persuade his colleagues of the reasonableness of his antiseptic method.

In 1887, **Robert Koch (1843-1910)** introduced steam sterilization and thus refined Lister's techniques.

In Germany the physician **H. Lennhartz**, who worked as medical director at Eppendorf Hospital, initiated the change in the understanding of sepsis from the ancient concept of putrefaction to the modern view of a bacterial disease. It was, however, his student Hugo **Schottmüller** (**1867-1936)**, who in 1914 paved the way for a modern definition of sepsis: "Sepsis is present if a focus has developed from which pathogenic bacteria, constantly or periodically, invades the blood stream in such a way that this causes subjective and objective symptoms." Thus, for the first time, the source of infection as a cause of sepsis came into focus. Schottmüller explained: "A therapy should not be directed against bacteria in the blood but against the released bacterial toxins." With this thinking he was well ahead of his time.

Although antiseptic procedures meant a huge medical breakthrough, it soon became apparent that a number of patients still developed sepsis. . In this pre-antibiotic time, the death rate was very high. These patients often showed a very low blood pressure. This condition was called septic shock. Only with the introduction of antibiotics after WW II could the death rate of sepsis be reduced further. With technological progress, intensive care medicine started to develop and sepsis patients soon became the main patient fraction on intensive care units (ICU).

In 1967 **Asbough** and colleagues observed a severe lung disease which developed in intensive care patients with severe shortness of breath, loss of lung compliance, and diffuse alveolar infiltration. This disease was called Adult Respiratory Distress Syndrome (ARDS) and was frequently a fatal complication. It was soon understood that particularly sepsis patients suffered from this complication. Apart from that, it appeared that the development of ARDS was a result of an inflammatory reaction and thus caused by substances produced in the diseased body. In the 1980s it was discovered that this inflammatory reaction was not only apparent in the lungs but in the whole body. Hence it became clear that the onset of sepsis did not derive from an infectious focus alone, but that the host response against infection must in some way play a role.

In 1989, US-American ICU specialist **Roger C. Bone** (1941-1997) offered a sepsis definition that is still valid until today: "Sepsis is defined as an invasion of microorganisms and/or their toxins into the bloodstream, along with the organism's reaction against this invasion."

On December 19, 2005, **Dr. Med. Frank Martin Brunkhorst** was awarded the Federal Cross of Merit for his achievements in the field of sepsis research.

## RELEVANT ANATOMY

The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac (ie, coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extraperitoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (ie, the peritoneal cavity).

The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.

Normally, the amount of peritoneal fluid present is less than 50 mL, and only small volumes are transferred across the considerable surface area

in a steady state each day. The peritoneal fluid represents a plasma ultrafiltrate, with electrolyte and solute concentrations similar to that of neighboring interstitial spaces and a protein content of less than 30 g/L, mainly albumin. In addition, peritoneal fluid contains small numbers of desquamated mesothelial cells and various numbers and morphologies of migrating immune cells (reference range is <300 cells/μL, predominantly of mononuclear morphology).

The peritoneal cavity is divided incompletely into compartments by the mesenteric attachments and secondary retroperitonealization of certain visceral organs. A large peritoneal fold, the greater omentum, extends from the greater curvature of the stomach and the inferior aspect of the proximal duodenum downward over a variable distance to fold upon itself (with fusion of the adjacent layers) and ascends back to the taenia omentalis of the transverse colon. This peritoneal fold demonstrates a slightly different microscopic anatomy, with fenestrated surface epithelium and a large number of adipocytes, lymphocytes, and macrophages, and it functions as a fat storage location and a mobile immune organ.

The compartmentalization of the peritoneal cavity, in conjunction with the greater omentum, influences the localization and spread of peritoneal inflammation and infections

## ETIOLOGY

The determinants of sepsis can be divided into three major factors.

- The micro organism involved in the infection
- The environment (local factors) in which infection is produced
- The host defense mechanisms

There is a continuous dynamic interaction among these factors that represent the state of homeostasis. The first two determinants of infection i.e., the bacteria and the environment have been extensively investigated.

## MICROBIOLOGY OF PATHOGENS IN SURGICAL INFECTIONS

The diverse numbers and the types of microorganisms that cause clinical sepsis in surgical patients continue to grow. Until 30 years ago bacteria were the principal pathogens of concern to surgeons. Now the novel modes of therapeutic options aids in the survival of critically ill patients. It has led to the evolution of newer pathogens in the other end of the spectrum prolonging the longevity of the patient with immunosuppression and malnutrition now becoming a common feature that complicates the management of many surgical patients especially when they are subjected to emergency abdominal surgeries where the preoperative risk factors are different from those in the case of elective surgeries.

It is important to recognize that the vast majority of the infections occurring in surgical patients are caused by endogenous bacteria. Specific bacteria are found in the specific parts of the body, and the exposed anatomic areas during a surgical procedure are usually the source of microorganisms that cause the infection. It is wise to know the normal bacterial flora of the body because such knowledge helps direct prophylactic antibiotics, to start intelligent empirical therapy.

## GRAM POSTIVE COCCI

Gram positive cocci of importance to surgeons include staphylococci and streptococci.

## STAPHYLOCOCCI

- Divided into coagulase positive and negative strains.
- Coagulase positive Staph.aureus is the most common pathogen in surgical infections. Mostly resistant to penicillin and sensitive to penicillinase resistant antibiotic and hence the treatment is difficult.
- MRSA is found to increase in the past two decades. The treatment of choice is Vancomycin, Quinupristin/Dalfopristin, Daptomycin, Linezolid.

## STREPTOCOCCI

- Most common are β hemolytic streptococci, S.pneumoniae and α hemolytic streptococci.
- Sensitive to Penicillin G and β lactam antibiotics.

## ENTEROCOCCI

- Commonly encountered as part of mixed flora in the intra-abdominal infections.
- The most effective combination is gentamycin combined with ampicillin or vancomycin.
- Prognosis is grave.

## AEROBIC AND FACULTATIVE GRAM NEGATIVE RODS

- Mostly Enterobacteriaceae – Escherichia, Proteus and Klebsiella.
- Common in hospital acquired infections and post operative surgical infections.
- Empirical antibiotic therapy includes third generation cephalosporin, expanded spectrum penicillin, quinolones and aminoglycosides.

## OBLIGATE AEROBIC GRAM NEGATIVE RODS

- Pseudomonas and Acinetobacter
- Mostly seen in hospital acquired pneumonia
- May also be recovered from a peritoneal cavity
- Often antibiotic resistant
- Requires specific antipseudomonal antibiotics
  - Ceftazidime and Cefipime
  - Aztreonam, Imipenam and Meropenam
  - Ciprofloxacin
  - Aminoglycosides

## ANAEROBES

- Inhabitants of the normal gastrointestinal tract
- Most common isolate is Bacteroides fragilis.
- Most effective antibiotics are Metronidazole, Clindamycin and combination of Penicillin and β lactamase inhibitor (Ampicillin/Sulbactum and Piperacillin/Tazobactam)

## ANTIMICROBIALS

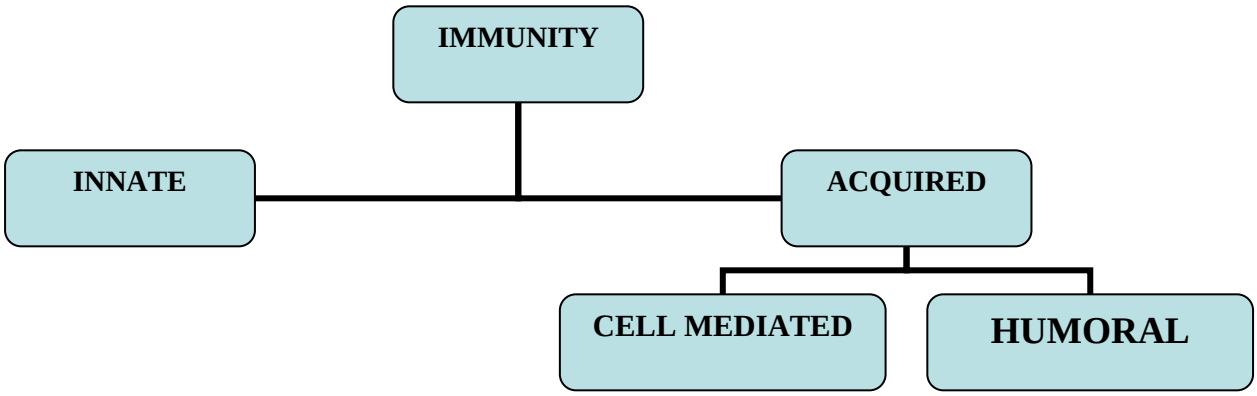
- The goal of therapy is to achieve antibiotic levels at the site of infection that exceed the minimum inhibitory concentration for the pathogen present.
- Guidelines for empirical treatment
  - Ensure coverage for the presumed pathogens involved – Broad Spectrum antibiotic then tailored to the type of organism isolated, avoid anti-anaerobic antibiotics.
  - Antibiotic that can reach the site of infection.
  - Toxicity to be considered.
  - Time bound antibiotic regimen

## THE MICRO ENVIRONMENT (LOCAL FACTORS)

- Oxygen Tension And Perfusion
- Tissue pH levels
- Non viable debris
- Hematoma
- Seroma
- Suture material contamination with exogenous bacteria
- Foreign body(e.g. prostheses)
- Drains

## HOST DEFENCES TO INFECTION

The human body has the ability to resist almost all types of organisms or toxins that tend to damage the tissues and organs. This capability is called **Immunity**.




### Innate Immunity

- Non specific Immunity
- It includes
  - Phagocytosis by white blood cells and cells of the tissue macrophage system
  - Acid secretion of the stomach and digestive enzymes
  - Skin

- Certain chemical compounds in the blood such as lyzozyme, basic polypeptides, complement complex, natural killer lymphocytes.

**Acquired Immunity**

- Specific Immunity
- Two types
  - Cell mediated Immunity and Humoral Immunity

**Cell Mediated Immunity**

- Formation of Activated T Lymphocytes by the Lymph nodes
- There are four types of T cells
  - Helper T cells
  - Cytotoxic T cells
  - Suppressor T cells
  - Memory T cells

**Humoral Immunity**

- B cells
- Formation of Antibodies
- Five Classes
  - IgG, IgM, IgA, IgD, IgE

**Complement System**

'Complement is a collective term that describes a system of about 20 proteins, many of which are enzyme precursors. The principal actors in this system are 11 proteins designated C1 through C9, B and D. All these are present normally among the plasma proteins in the blood as well as among the proteins that leak out of the capillaries in to the tissue spaces. There are two pathways namely, Classic and Alternate Pathways .

**HOST FACTORS**

- Age
- Malnutrition
- Obesity
- Smoking
- Diabetes
- Steroids and Immunosuppressants
- Transfusion
- Multiple comorbid conditions.

## PATHOGENESIS

The initial act of surgical incision by breaching the skin disrupts the primary barrier to the infection. Microorganisms then gain access to the blood stream and deep tissues through the incision. Dead space may result, carrying an increased risk of infection. Areas of tissue ischemia, necrosis and inadequate blood flow are created, predisposing to the formation of exudates and hematomas. Exudates and hematomas increase the risk of SSI because they provide a suitable environment and nourishment in which microorganisms may thrive. Poor hemostasis may lead to the formation of hematomas and thus increase the risk of sepsis. If the patient is immunocompromised, the risk of sepsis is increased.

Foreign bodies predispose to the infection by reducing the number of organisms required to produce an infection. The critical size of the inoculum varies with the foreign body. For example, the number of organisms needed to cause an infection with tape closure of a wound is more than with staple closure. Staple closure, in turn, requires a larger inoculum than suture closure. The kind of suture material also has an effect on the risk of sepsis. Generally, monofilament sutures require a larger inoculum than non synthetic sutures. Foreign body implants, such as prostheses and bone wax, variably predispose to infection. Obviously, the choice of suture and foreign

body materials is not based solely on the risk of sepsis, but involves consideration of the intended function and structure of these devices.

Technical factors, such as the skill and experience of the surgeon affect the risk of sepsis. Increased tissue trauma and prolonged duration of surgery are contributing factors.

## CLASSIFICATION OF WOUNDS

This classification of operative wounds is based on the degree of microbial contamination.

### CLASS I / CLEAN

Elective, not emergency, non-traumatic, primarily closed; no acute inflammation; no break in technique; respiratory, gastrointestinal, biliary and genitourinary tracts not entered.

### CLASS II /CLEAN CONTAMINATED

Urgent or emergency case that is otherwise clean; elective opening of respiratory, gastrointestinal, biliary or genitourinary tract with minimal spillage (e.g. appendectomy) not encountering infected urine or bile; minor technique break

### CLASS III/CONTAMINATED

Non-purulent inflammation; gross spillage from gastrointestinal tract; entry into biliary or genitourinary tract in the presence of infected bile or

urine; major break in technique; penetrating trauma <4 hours old; chronic open wounds to be grafted or covered.

**CLASS IV/DIRTY**

Purulent inflammation (e.g. abscess); preoperative perforation of respiratory, gastrointestinal, biliary or genitourinary tract; penetrating trauma >4 hours old.

## MANIFESTATIONS

The signs of postoperative infections could be increase in body temperature, tachycardia, tachypnoea, increase in local warmth, tenderness, edema, drainage from the surgical site or drain site. The manifestations of postoperative sepsis has been dealt below.

Purulent drainage from a wound is definitive evidence of sepsis regardless of whether cultures yield growth. Although purulent drainage contains many more leukocytes than serous or serosanguinous drainage, it is usually classified on the basis of gross observation. However, the absence of pus does not exclude an infection.

## SURGICAL SITE INFECTIONS

### SUPERFICIAL INCISIONAL SURGICAL SITE INFECTIONS

Superficial incisional surgical site infections must meet the following two criteria:

- Occur within 30 days of procedure
- Involve only the skin or subcutaneous tissue around the incision.

Plus

At least one of the following criteria:

- purulent drainage from the incision

- organisms isolated from an aseptically obtained culture of fluid or tissue from the incision

- at least one of the following signs or symptoms of infection - pain or tenderness, localized swelling, redness or heat - and the incision is deliberately opened by a surgeon, unless the culture is negative

- diagnosis of superficial incisional SSI by a surgeon or attending physician

The following are not considered superficial SSIs:

- stitch abscesses (minimal inflammation and discharge confined to the points of suture penetration)
- infection of an episiotomy or neonatal circumcision site
- infected burn wounds
- Incisional SSIs that extend into the fascial and muscle layers.

## DEEP INCISIONAL SURGICAL SITE INFECTIONS

Deep incisional surgical site infections must meet the following three criteria:

- Occur within 30 days of procedure (or one year in the case of implants)

- Are related to the procedure
- Involve deep soft tissues, such as the fascia and muscles.

  Plus

At least one of the following criteria:

- Purulent drainage from the incision but not from the organ/space of the surgical site
- A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms - fever (>38Â°C), localized pain or tenderness - unless the culture is negative
- An abscess or other evidence of infection involving the incision is found on direct examination or by histopathologic or radiological examination
- Diagnosis of a deep incisional SSI by a surgeon or attending physician.

## ORGAN SPACE

- Infection less than 30 days after surgery with no implant
- Infection less than 1 year after surgery with an implant and infection; involves any part of the operation opened or manipulated, plus one of the following:
    - Purulent drainage from a drain placed in the organ space
    - Cultured organisms from material aspirated from the organ space
    - Abscess found on direct or radiologic examination or during reoperation
    - Diagnosis of organ space infection by a surgeon

## RISK FACTORS FOR SURGICAL SITE INFECTIONS BASED ON THE DETERMINANTS OF INFECTION

## MICROORGANISMS

- Duration of the procedure
- Wound class
- Recent hospitalization
- Previous antibiotic therapy
- Preoperative shaving
- Bacterial number, virulence and antimicrobial resistance

## LOCAL WOUND

- Surgical technique
- Hematoma
- Seroma
- Necrosis
- Sutures
- Drains
- Foreign bodies

## PATIENT FACTOR

- Age
- Malnutrition
- Obesity
- Smoking
- Diabetic status /Glucose control
- Multiple comorbid conditions
- Immunosuppresant
- Malignancy
- Transfusions

## RISK SCORES FOR SURGICAL SITE INFECTIONS

SSI risk has traditionally been correlated to wound class. The accepted range of infection rates has been 1% to 5% for clean, 3% to 11% for clean-contaminated, 10% to 17% for contaminated and greater than 27% for dirty wounds.

## POSTOPERATIVE FEVER

One of the most concerning clinical findings in a patient postoperatively is the development of fever. Fever refers to a rise in core temperature, modulation of which is managed by the anterior hypothalamus. Numerous disease states can cause fever in the postoperative period. The most common infections, however are healthcare associated infections; surgical site infection, urinary tract infection, intravascular catheter –related infection and pneumonia.

## THE ABDOMINAL COMPARTMENT SYNDROME

The abdominal compartment syndrome represents the pathophysiologic consequence of a raised intra-abdominal pressure. Various clinical conditions are associated with this syndrome and include massive intra-abdominal or retroperitoneal hemorrhage, severe gut edema or intestinal obstruction, and ascites under pressure.

Various systems are involved in this syndrome. First, the increased intra-abdominal pressure is transmitted to the pleural space so that lung compliance decreases. Hypoventilation and alteration of ventilation/perfusion distribution lead to hypoxemia and hypercapnia. When mechanical ventilation is applied, very high inspiratory pressures are often required to deliver tidal volume. Second, the combined increase in

abdominal pressure and pleural pressure leads to a decrease in venous return, direct compression of the heart, and increased afterload (especially in the right ventricle). Third, perfusion to the intra-abdominal organs can be critically reduced by the combined effects of the decreased cardiac output, increased interstitial pressure, and increased outflow pressure. This can lead to oliguria and renal failure. Splanchnic ischemia can also occur as reflected by a decreased mucosal pH, decreased liver metabolism, and bacterial translocation. In addition, perfusion of the abdominal wall may be decreased, so that wound healing may be impaired. Finally, intracranial pressure may also be increased due to the decrease in cerebral venous return and increased venous pressure.

The magnitude of this syndrome and the involvement of the various organs depend on the level of the intra-abdominal pressure. The normal intra-abdominal pressure ranges between 0 and 5 mmHg. When it is mildly increased to between 10 and 15 mmHg, cardiac index is usually maintained or even increased because abdominal viscera are mildly squeezed and venous return increases. Respiratory and renal symptoms are unlikely to occur. Hepatosplanchnic blood flow may decrease. At this point, intravascular volume optimization will probably correct these alterations. When intra-abdominal pressure is moderately increased to between 15 and

25 mmHg the full syndrome may be observed, but usually responds to aggressive fluid resuscitation, and surgical decompression should be considered. At high pressures (< 25 mmHg) surgical decompression associated with fluid resuscitation and transient use of vasoconstrictive agents is mandatory. When surgical decompression is not feasible, application of a negative abdominal pressure should be considered.

The diagnosis of this syndrome is difficult because it usually occurs in critically ill patients with other causes of circulatory or respiratory failure. One should always consider the abdominal compartment syndrome when confronted with acute circulatory failure with wide systolic-diastolic pressure variation and elevated filling pressures. After exclusion of cardiac tamponade and increased pleural pressure (tension pneumothorax, status asthmaticus, etc), the intra-abdominal pressure should be measured.

Current methodology for intra-abdominal pressure assessment relies on the measurement of bladder pressure. Alternative methods include indirect estimations of inferior vena cava pressure, rectal and gastric pressure measurements, and direct measurement of the intra-abdominal pressure by direct puncture. In experimental conditions, bladder pressure is closely related to abdominal pressure .

## ACUTE WOUND FAILURE (DEHISCENCE)

Acute wound failure (wound dehiscence or a burst abdomen) refers to postoperative separation of the abdominal musculoaponeurotic layers. Acute wound failure occurs in approximately 1% to 3% of patients who undergo an abdominal operation. The majority of burst abdomen occurred between 7th and 10th post-operative day, with the highest incidence on the 7th post-operative day.

Diagnosis is mainly clinical. Patient presented withserosanguinous discharge from the wound on the 6th or 7th post-operative day.

**Factors associated with wound dehiscence**

- Technical error in fascial closure
- Emergency surgery
- Intra-abdominal infection
- Advanced age
- Wound infection, hematoma , seroma
- Elevated Intra-abdominal pressure
- Obesity Chronic corticosteroid use
- Previous wound dehiscence
- Malnutrition
- Radiation therapy and chemotherapy

- Systemic disease (uremia, diabetes mellitus)

The incidence of burst abdomen was much higher in patients operated as emergency surgery as compare to planned surgery.

Anatomical factors which might make a vertical upper abdominal wound more likely to burst are as follows:

- Interference with blood supply as it runs transversely. The rectus abdominal muscle has a segmental blood and nerve supply.
- If incision is little more laterally, the medial part of the rectus abdominal muscle gets denervated and ultimately atrophied. This creates a weak spot in the wall and burst abdomen. This is the reason why one should not go beyond the midline.
- The rectus sheath is disturbed in vertical direction. The fibers of the sheath run transversely, so by vertical incision all of them are cut. Similarly, the anterior sheath is detached from the tendinous insertion.
- With upper abdominal incision, pain prevents chest movements thus favoring more respiratory complications and cough. Cough will increase intraabdominal pressure more in the upper part leading to tension strain in the fresh wound.
- Elastic fibers of the skin also run transversely, so they are cut by vertical incision. The strength of the wound is decreased. But as the

linea alba is a weaker structure below the umbilicus, burst abdomen is more common with lower incision.

The following are the important factors enhancing the chances of burst abdomen:

- Inadequate muscle relaxation during abdominal wound closure.
- Undue tension over the stitches and increased intra-abdominal pressure due to peritoneal fluid; drainage relieves the tension.
- Forgetting to suture the peritoneal layer with the transversalis fascia as it has the tendency to get retracted.

## INTRA-ABDOMINAL AND RETROPERITONEAL INFECTIONS

- ♦ Despite modern antibiotics and intensive care mortality from serious intra-abdominal infections remains high (5%-50%).
- ♦ Severe hypermetabloic and catabolic response is universal.
- ♦ If corrective surgery and effective antibiotics are not delivered promptly will lead to multiple organ dysfunction syndrome.
- ♦ The risk increases with increased age, malnutrition and underlying comorbid conditions.
- ♦ Goal of surgical intervention is source control.

## DIAGNOSIS

The diagnosis of post operative sepsis may be difficult. Although the presence of purulent drainage is diagnostic of SSI, cultures of the drainage or of the wound may not reflect the actual cause of the infection. False positive rates may exceed 80% and the predictive value of interpretive cultures may be as low as 32%. Moreover absence of growth in cultures of purulent drainage does not rule out SSI. Positive cultures must be combined with other data including clinical findings and laboratory or radiographic data.

Blood culture may eventually be helpful in the septic patient, although treatment is usually started empirically before results are available.

Although peripheral blood leukocytosis is a non specific finding, the presence of increased numbers of polymorphonuclear neutrophils, especially bands, in conjunction with other data may help in establishing the diagnosis of infection.

Ultrasound and especially CT scans have revolutionized the detection and management of deep infection, especially intra abdominal abscesses.

The ultra sound is often the referred modality because of its general utility. Ultra sound examination has the virtue of bedside applicability, but the information may be more limited.

In addition to the routine bedside ultrasound for the detection of postoperative sepsis, CT is useful to find out the infection in the abdominal wall and abscess in the psoas muscle. By CT, abscesses are characteristically well defined, low attenuated masses that may displace the adjacent organs and obliterate nearby fat planes. Differentiation of an abscess from a collection of inflammatory liquid may not be possible by CT alone, but CT guided aspiration with gram-stain and culture is often definitive. Special stains and cultures, as for acid-fast bacteria or fungi, may occasionally be helpful, although postoperative wound infections are usually caused by bacteria. CT-guided percutaneous drainage may be the preferred therapy because the risks of anaesthesia and surgery are avoided. Generally, CT scans are not helpful in identifying collections of liquid in the first week postoperatively, but they may be very helpful thereafter.

The differential diagnosis of SSI includes a normally healing wound, a stitch abscess, dehiscence of the wound from other causes, other sources of postoperative fever and deep infections such as necrotizing fascitis.

Healing wounds may manifest one or more signs of infection such as erythema swelling and tenderness. In the absence of infection, these findings are localized and have only minimal severity.

A stitch abscess causes localized inflammation and drainage. Purulence may be present but is limited to the sites of sutures and clears with in 72 hrs after sutures are removed. Removal of infected sutures is curative, and the incision itself not considered infected.

A surgical wound may dehisce as a result of infection; however dehiscence may result from sub optimal closure of the wound, or failure of the tissue surrounding the wound. In both cases, there will probably be few or no signs of infection. Failure of the incision or of the surrounding tissues should be evident on inspection. Mechanical factors that may lead to dehiscence include increased abdominal pressure from a distended bowel, ascites, cough and vomiting.

The differential diagnosis of postoperative fever includes not only SSI but also infections at other sites like infections of the respiratory or urinary tract and non infectious cause of fever.

## MANAGEMENT

Proper treatment of SSI requires a combination of surgical and medical therapies tailored to the needs of the patient.

A suture or stitch abscess is a localized infection that requires only removal of the suture for complete cure.

An incisional infection is more extensive, and in most cases, sutures or staples must be removed, opening the wound to allow drainage.

While the internist's initial reflex is to treat all infections with antimicrobial agents, many SSIs are appropriately managed and cured solely by opening the incision to allow drainage.

Therapy may involve the parenteral administration of antimicrobial agents especially if systemic signs and symptoms of infection are present. However, antimicrobial agents are adjunctive to debridement of necrotic tissue and drainage of abscesses.

Drainage may be either surgically or more commonly, percutaneously under the guidance of CT or Ultrasound. The method used depends on the location, accessibility and complexity of the abscess, as well as the availability of skilled radiologist.

Bacteremia with sepsis requires parenteral antimicrobial agents often with aggressive administration and management of fluids and electrolytes.

The choice of antimicrobial agents is based on the site of infection, the most likely pathogens and data from culture and sensitivity reports. When chemoprophylaxis was given perioperatively, an empiric parenteral regimen consisting of different drugs should be chosen and the antimicrobial agents to be changed in accordance with the culture and sensitivity reports.

If there are signs of deterioration of the general condition of the patient such as abdominal distention, anastamotic leak may need a relaparotomy.

## PROGNOSIS

The prognosis of sepsis depends on the following factors

- Site of infection
- Extent of infection
- Pathogen involved
- Patient's underlying condition.

Complete recovery is usual in localized infections such as abscesses associated with sutures and incisional infections and at the other end of the spectrum mortality mainly due to multiple organ dysfunction syndrome associated with comorbid pre-existing disease of the patient.

## PREVENTIVE MEASURES

### PREOPERATIVE PERIOD

- Appropriate preoperative hair removal or no hair removal
- Antimicrobial prophylaxis
- Strict glucose control
- Preoperative warming

### INTRAOPERATIVE PERIOD

- Asepsis and antisepsis
- Avoid spillage in gastrointestinal cases
- Surgical technique
    - Avoid seroma/hematoma
    - Good perfusion
    - Obliterate dead space
    - Justified drain use (closed)
    - Limit use of sutures/foreign bodies
    - Use monofilament sutures
- Supplement oxygen
- Adequate fluid resuscitation
- Strict glucose control

## POSTOPERATIVE PERIOD

- ❖ Protect incision for 48 – 72 hours
- ❖ Remove drains as soon as possible
- ❖ Avoid postoperative bacteremia
- ❖ Early enteral nutrition
- ❖ Early ambulation
- ❖ Chest Physiotherapy

# METHODOLOGY

This study was conducted in the Department of Surgery in the Coimbatore Medical College Hospital after getting the due approval from the Ethics Committee of the Coimbatore medical college.

180 patients (144 male and 36 female patients) who underwent emergency abdominal surgeries during the period from August 2008 to September 2009 in the six surgical units of the Department of Surgery were chosen for the study.

After getting prior informed written consent for the surgery and study, subjects were enrolled for the study.

The details regarding the patient, investigations, diagnosis, surgical procedure, intra-operative findings, prophylactic antibiotics, postoperative period and follow-up were recorded.

The following basic investigations were done for the patients before being taken up for surgery.

- Hemoglobin
- Blood Sugar
- Blood Urea

- ❖ Serum Creatinine
- ❖ Chest X-Ray
- ❖ ECG

**Preoperatively**

- ♦ After initial resuscitation with intravenous fluids or blood (depending upon the clinical status), the patients were taken up for surgery.
- ♦ Preoperatively all patients received prophylactic antibiotics which could be either of these given below

  - Third generation Cephalosporin with Aminoglycoside and Metronidazole

  - Quinolone with Aminoglycoside and Metronidazole

- ♦ Preoperatively hair shaving was done just prior to surgery.

**Intraoperatively**

- ♦ To disinfect the surgical site povidone iodine solution is used.

- Spinal Anesthesia was given to most of the patients with acute appendicitis and general anesthesia was given to most of the cases for laparotomy.
- Duration of surgery varied depending on the peroperative findings.
- Intra-operatively peritoneal fluid or abscess if present, fiuid/ material were sent for culture and sensitivity.
- Empirical antibiotics were started prior to the reporting of the culture and sensitivity tests.

**Postoperatively,**

  - General condition of the patient was monitored with pulse rate, temperature and respiratory rate chart.
  - Wound was inspected after 48 hours.
  - Looked for edema, tenderness, hyperemia,discharge and wound dehiscence.
  - Discharge from the wound site or drain site were sent for culture and sensitivity and the antimicrobial agents were changed accordingly.

- If the patient presented with postoperative diarrhea, Ultrasound abdomen and pelvis was done to rule out intra-abdominal collections. If present, initially patient was treated conservatively, if the general condition deteriorates, patient was subjected to relaparotomy.
- Patients were followed up for 30 days.

# OBSERVATION

The study group of 180 patients was chosen from those who underwent emergency abdominal surgeries in the six surgical units of Coimbatore Medical College Hospital during the period of August 2007 to September 2009.

144 were male patients and 36 were female patients in the study group. The male to female ratio was 4:1.

The most commonly performed emergency abdominal surgery was Appendicectomy ( 75 cases; 41.6%) followed by laparotomy for hollow viscus perforation (54 cases; 30%). Duodenal perforation was the most common cause of hollow viscus perforation during the study.

Other emergency abdominal surgeries which were studied were done for Liver Abscess, Small bowel gangrene, Intestinal obstruction and abdominal trauma.

In the 180 cases observed during the study, 41 patients had postoperative infection and there were 35 male patients and 6 female patients and the ratio of males: females is 5.8 : 1.

The following were the conditions for which emergency abdominal surgeries have been taken up and it has been tabulated below

| **Conditions for which emergency abdominal surgeries have been done** | **No. of male patients** | **No. of female patients** |
|---|---|---|
| **Appendicitis & appendicular abscess** | **56** | **19** |
| **Hollow viscus perforation** | **49** | **5** |
| **Intestinal obstruction** | **10** | **5** |
| **Liver abscess** | **5** | **1** |
| **Obstructed hernia** | **4** | **2** |
| **Small bowel gangrene** | **6** | **1** |
| **Blunt injury** | **6** | **-** |
| **Stab injury** | **2** | **-** |
| **Sigmoid volvulus** | **2** | **-** |
| **Pancreatitis** | **2** | **-** |
| **others** | **2** | **3** |
| **Total** | **144** | **36** |

The above said 180 patients were classified based on wound class which has been tabulated below

| WOUND CLASS | TOTAL NO. OF CASES | NO. OF CASES WITH INFECTION |
|---|---|---|
| CLASS I | 6 | 0 |
| CLASS II | 70 | 11 |
| CLASS III | 18 | 3 |
| CLASS IV | 86 | 27 |
| TOTAL | 180 | 41 |

The maximum number of cases with postoperative infections was seen in Class IV (27 cases; 15%) and which was followed by Class II (11cases;6.1%).

Appendicectomy performed for acute appendicitis is considered under Class II which is the most common emergency abdominal surgery being performed. Gangrenous appendix and appendicular abscess were considered under Class IV.

The gender differences in the postoperative infection has been tabulated below

| Wound Class | Males | | Females | |
|---|---|---|---|---|
| | Total No. Of cases | No.of cases with postoperative infection | Total No. Of cases | No. Of cases with postoperative infection |
| CLASS I | 6 | 0 | 0 | 0 |
| CLASS II | 49 | 8 | 21 | 3 |
| CLASS III | 13 | 2 | 4 | 1 |
| CLASS IV | 76 | 25 | 11 | 2 |

**POSTOPERATIVE INFECTIONS CLASSIFIED BASED ON SSI**

The 41 cases which had varied postoperative infections were classified as Superficial Incissional, Deep Incissional and Organ Space SSI

The most commonly encountered one is Superficial incisional SSI (22 cases;12.2%) which was followed by Deep Incisional (12 cases; 6.6%)

Although there were only 7 cases (3.8%) involved in organ space SSI, it was associated with high mortality and morbidity.

SURGICAL SITE INFECTIONS

| **SSI** | **NO.OF CASES** |
|---|---|
| Superficial incisional | 22 |
| Deep incisional | 12 |
| Organ Space | 7 |
| Total | 41 |

## POSTOPERATIVE INFECTIONS BASED ON DURATION OF SURGERY

The patients were classified based on the duration of surgery which varied depending on the diagnosis and intraoperative findings. The cases with postoperative infections were classified based on the duration of surgery and tabulated.

| DURATION OF SURGERY | NO. 0F CASES INFECTED |
|---|---|
| < 1 HOUR | **2** |
| 1HR - 2HRS | 11 |
| >2HOURS | **28** |
| TOTAL | 41 |

Peroperative peritoneal fluid was sent for culture and sensitivity. Mostly the culture report was negative for duodenal ulcer perforation. It showed mixed bacterial flora in appendicular and lower intestinal perforation. Empirical antibiotics were given in anticipation for the reports and they were changed once the sensitivity reports obtained.

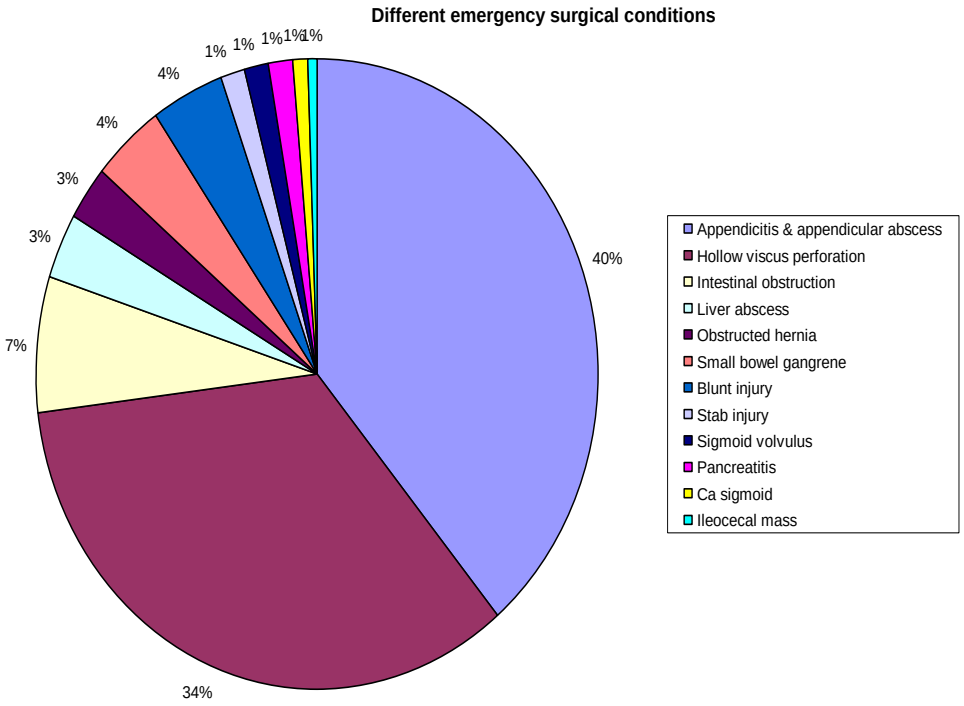
In the 41 cases which had the manifestations of postoperative infections the most common organisms encountered were

- E coli
- Klebsiella species
- Proteus species

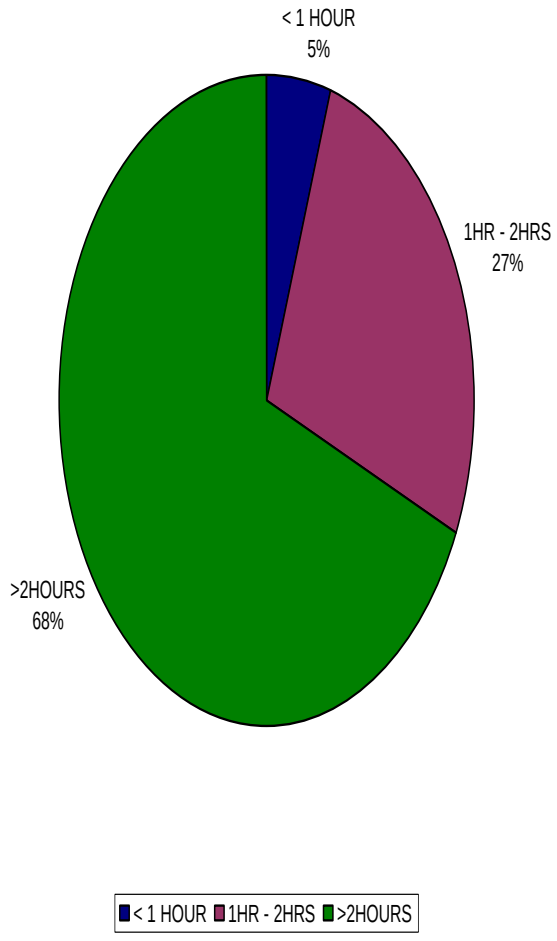
They were sensitive to third generation cephalosporins and amikacin.

Out of the seven cases which had the evidence of intra-abdominal collection were due to anastamotic leak. Four cases were taken up for relaparotomy. The rest three were treated with perrectal drainage of pelvic collection.

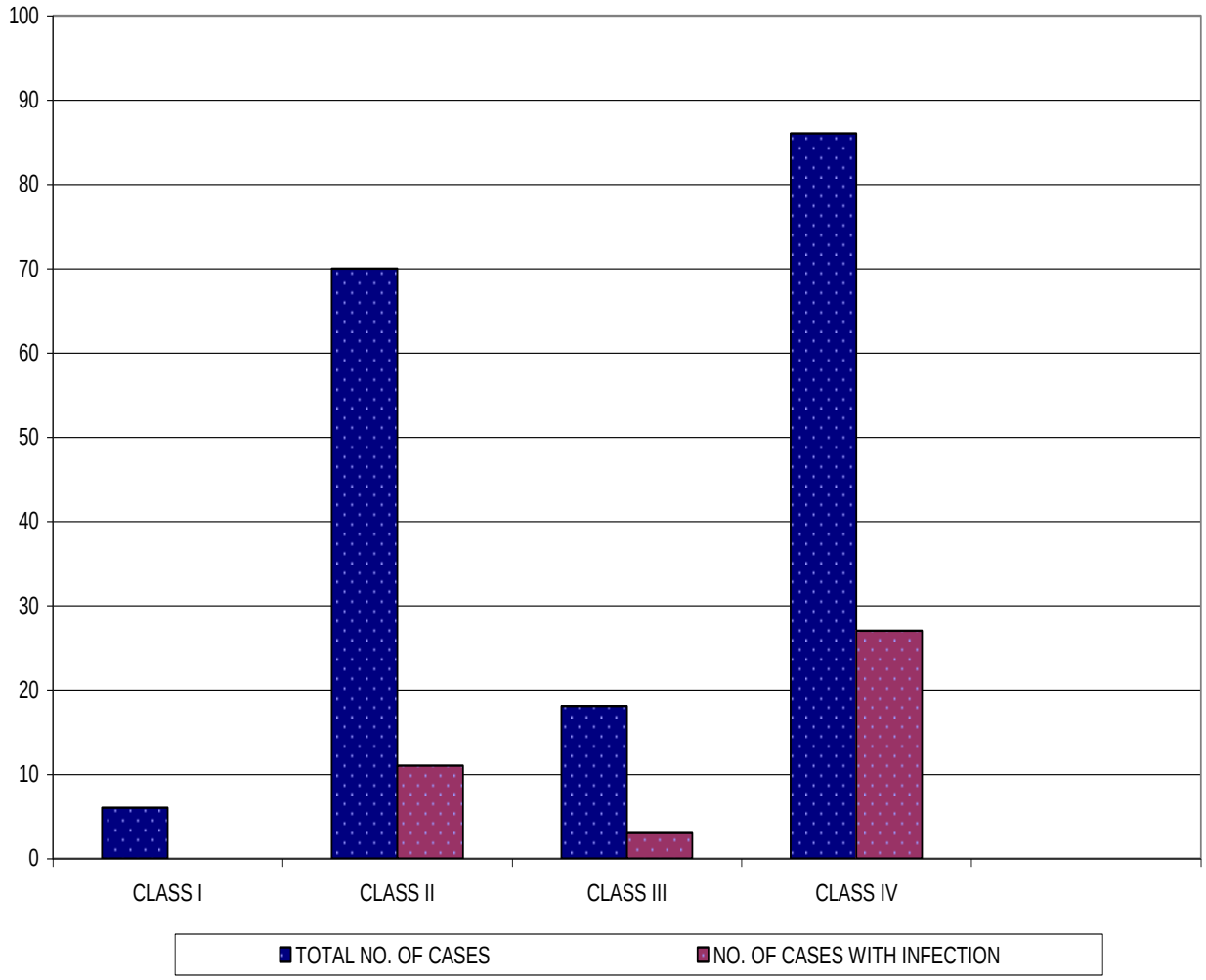
Amongst the four cases taken up for relaparatomy two cases died of septicemia and multi organ dysfunction syndrome.


Different emergency surgical conditions
40%
34%
7%
3%
3%
4%
4%
1%
1%
1%
1%
1%
Appendicitis & appendicular abscess
Hollow viscus perforation
Intestinal obstruction
Liver abscess
Obstructed hernia
Small bowel gangrene
Blunt injury
Stab injury
Sigmoid volvulus
Pancreatitis
Ca sigmoid
Ileocecal mass

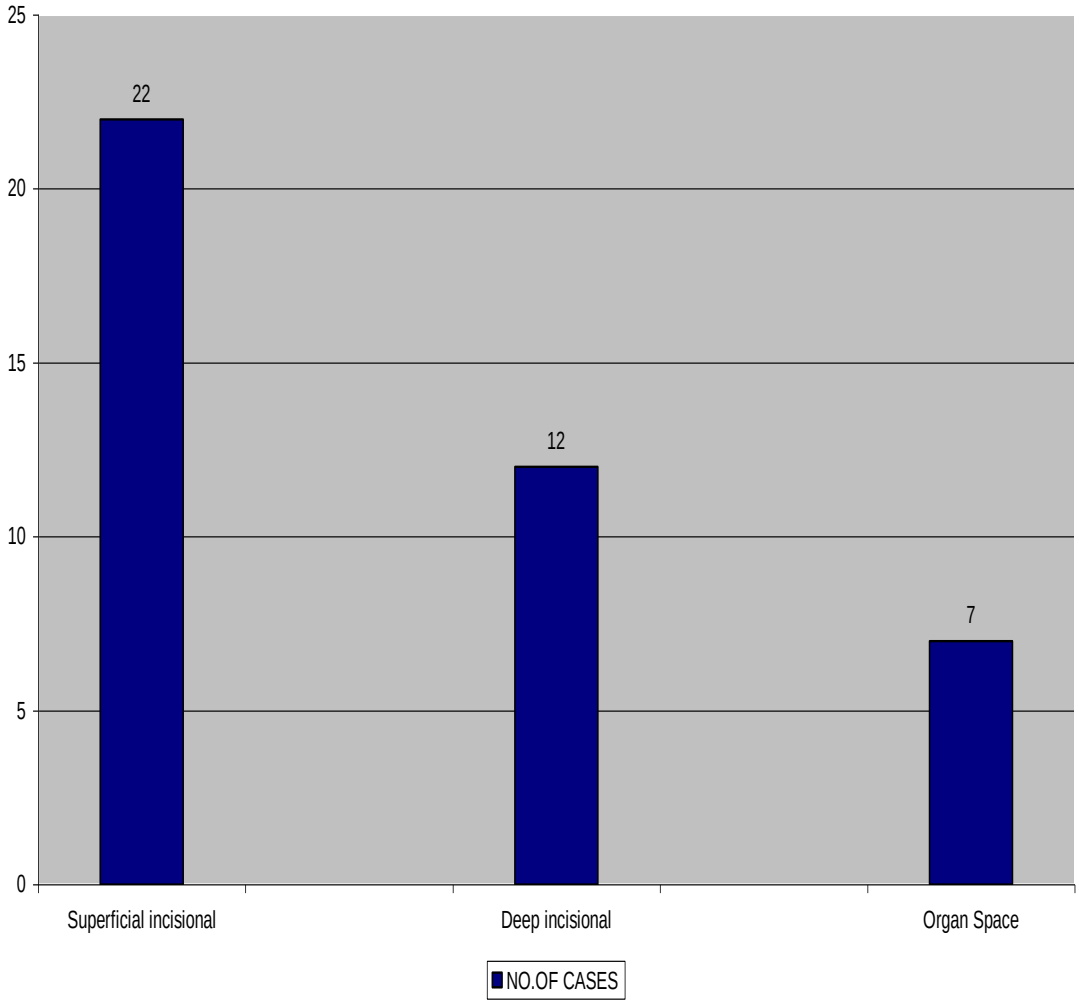
**POSTOPERATIVE INFECTION BASED ON DURATION OF SURGERY**



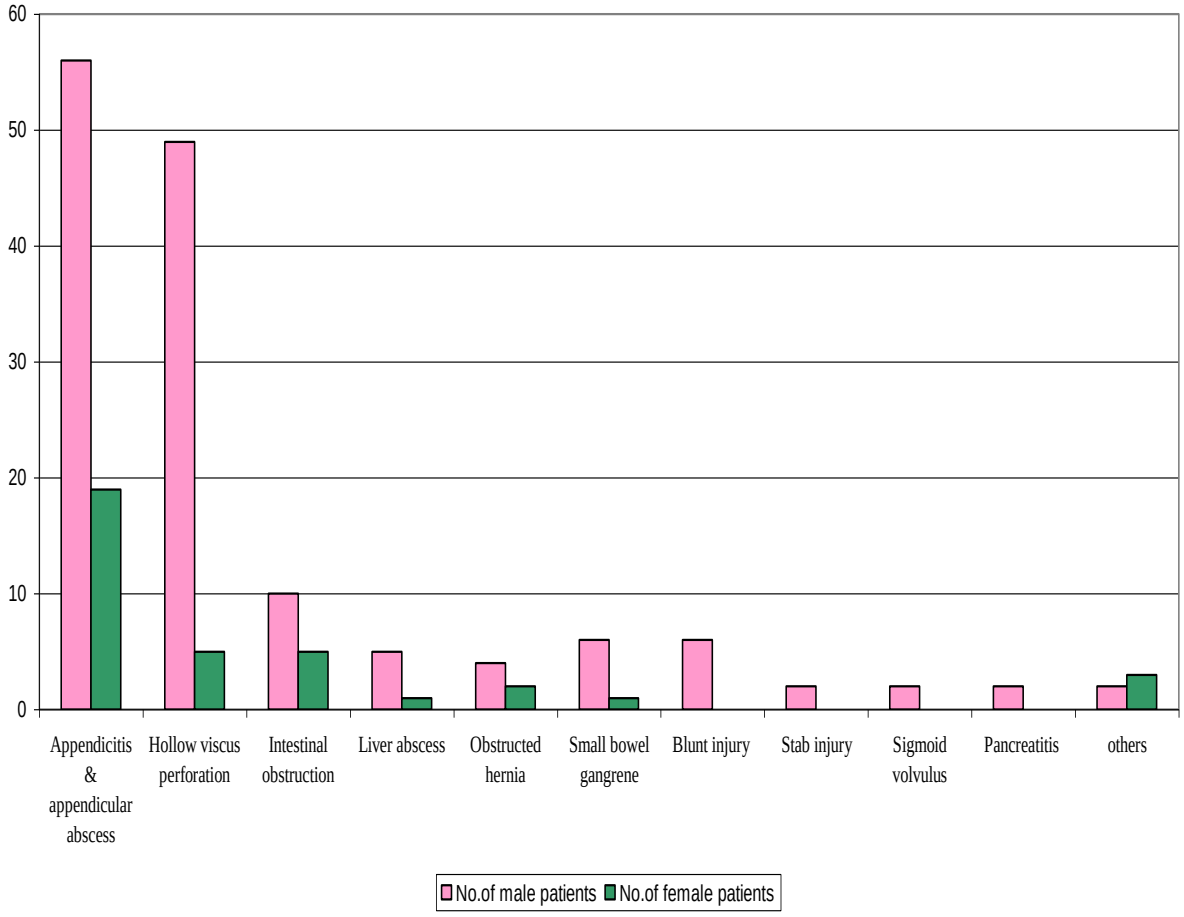
**POSTOPERATIVE INFECTION BASED ON WOUND CLASS**



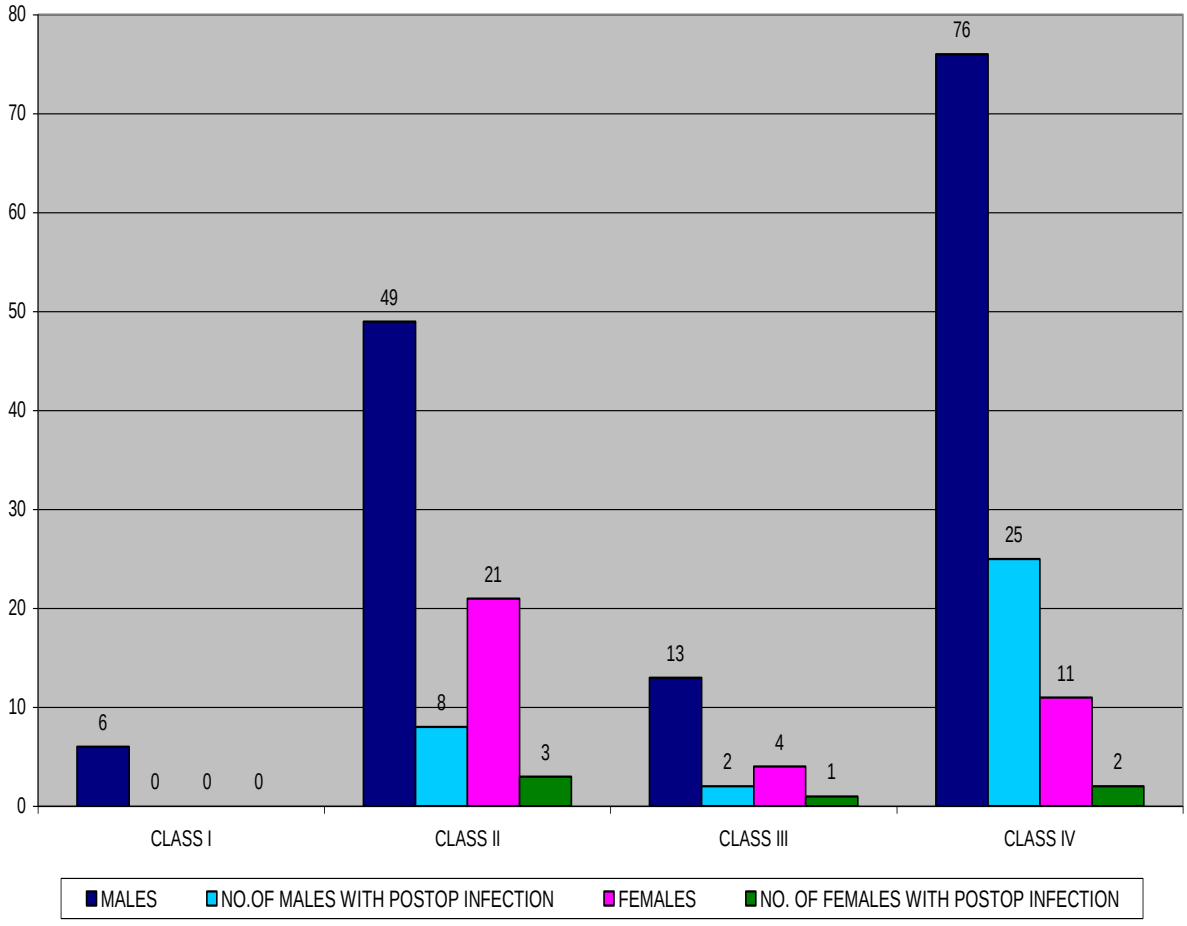
**SURGICAL SITE INFEECTIONS**



## Gender differences



**POSTOPERATIVE INFECTION IN MALES AND FEMALES**



## DISCUSSION

In this study of 180 cases of emergency abdominal surgeries, 41 cases had postoperative infection, amongst which 35 were males and 6 were females. **The incidence of postoperative sepsis as per this study is 22.8%.** The male to female ratio is 5.8:1.

In previous years the incidence of postoperative sepsis was 37.8% in this institution.

The most common emergency abdominal surgery is Appendicectomy (40%) followed by Laparotomy and Perforation closure for Hollow viscus perforation (34%).

The rate of infection was found directly proportional to the amount of contamination, thus highest rate was seen in the **Class IV Surgeries(31.4%) followed by Class III (16.6%) and Class II (15.7%) in order.**

In this study, out of the 41 cases with post operative sepsis, 22 had Superficial incisional SSI, 12 had Deep Incisional SSI and 7 had Organ space SSI.

Ruben Peralta et al in their study found that the incidence of surgical site infection increases with the degree of contamination; therefore, surgical site infection occurs at much higher rates after operations for peritonitis and peritoneal abscess (ie, 5-15% compared to <5% for elective abdominal

operations for noninfectious etiologies). Surgical site infection may be expected if the wound is closed in the setting of gross abdominal contamination.

The duration of surgery was one of the predictors of postoperative sepsis with the duration of surgery being directly proportional to the development of postoperative infection.

Inadequate preparation of the acute emergency cases due to speedy action required to meet that life threatening situation bypassing the routine precautionary measures like bowel wash, inadequate correction of dehydration could be attributed to the development of postoperative infection following emergency abdominal surgical procedures.

In the emergency operation theatre, the possibility of contamination even with fecal matter or bowel contents of a previous case resulting in cross infection, attributing to high rates of postoperative infection.

Unduly early shaving of the operative area, resulted abrasions harboring the nosocomial organism, adequate interval before surgery allowing its establishment as infection.

Though factors related to the skin preparation (antiseptic solution and contact period), length of operation, abuse of diathermy, improper hemostasis, irrigation, the type of suture material used, theatre

contamination due to improper outdated ventilation system, have definite role in the SSI, are variable and difficult to measure.

The factors related to surgeon, such as hand wash, nasal and oral microbial commensals, technical factors, attitude towards protecting aseptic field are highly variable, only self audit can identify but cannot quantify role of these factors.

Apart from the above said factors the patient related factors are inadequate personnel hygiene, delayed recognition of the symptoms, delayed admission to the hospital, smoking and alcohol abuse in males, anemia and malnutrition in females contribute for the development of sepsis.

The hospital being the tertiary care centre and teaching hospital, most of the cases are referred from other periphery hospitals and delayed referral could be the one of the causes of postoperative infection.

In the 180 cases taken up for the study, 7 had Organ Space SSI, amongst which 4 cases were subjected to relaparotomy. 2 cases had mutiorgan dysfunction syndrome as a result of septicemia and died.

However, to check these postoperative infections, the following measures are necessary.

- ❖ Avoidance of hair shaving or usage of clipping of hairs prior to surgery.

- Proper education/instruction to the theatre personnel and medical students to protect the aseptic atmosphere.
- Improvement of the theatre, having check in the direct ventilation with hospital atmosphere and the usage of positive pressure, laminar air flow ventilation and to avoid usage of fans
- In all clean contaminated, the preoperative antibiotic with peak plasma concentration during surgery and its activity should cover the flora of the viscus that is exposed.
- Wherever possible, use of the diathermy and suture material should be minimized.
- Grossly contaminated and dirty group of procedures needs thorough irrigation with NaCl 0.9% solution, even upto 6 litres are required to achieve the clearance of the contamination.
- Peritoneal cavity should be thoroughly cleared of the debris without leaving any collection.
- Perfect hemostasis should be observed.
- Corrugated rubber drain should be used judiciously in the Class IV Cases
- Use of monofilament sutures should be advocated.

- In the postoperative period, adequate hydration, strict glycemic control, proper antibiotic coverage should be given
- Early enteral feeding and early ambulation during the postoperative period.
- In the postoperative period, the drains should be removed as early as possible unless otherwise its use highly warranted.

# CONCLUSION

With the present study, it is concluded that the cause of postoperative sepsis is multifactorial. . The incidence of postoperative sepsis as per this study is 22.8% as compared to 37.8% in previous studies. This decrease of 15% in the incidence of postoperative sepsis can be attributed to the following preventive measures.

- Pre operative period:
  - Good bowel preparation that may not be possible pre operatively in emergency condition, in that circumstances one may go for intraoperative mechanical lavage and avoidance of soilage.
  - Systemic antibiotic pre operatively and throughout procedure.
  - Prompt resuscitation with appropriate correction of fluid imbalance.

- Intra operative measures:
  - sound surgical procedure
  - anastamosis to be done in healthy bowel with adequate blood supply

    - meticulous haemostasis and correction of anemia by blood transfusion.
- If there is severe peritonitis on first operation then avoidance of anastamosis and exteriorization of bowel.
- Post operative measures:
    - prevent hypotension
    - maintain good nutrition status
    - adequate antibiotic coverage
    - early ambulation.

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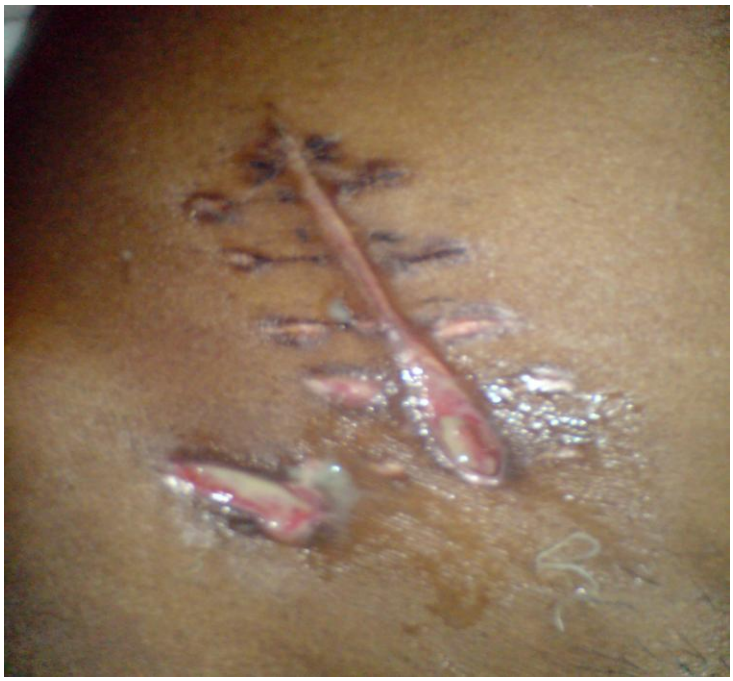
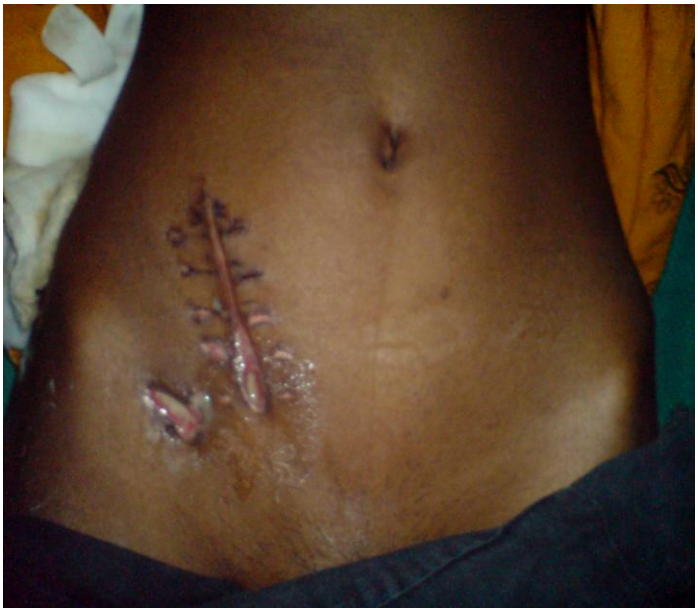
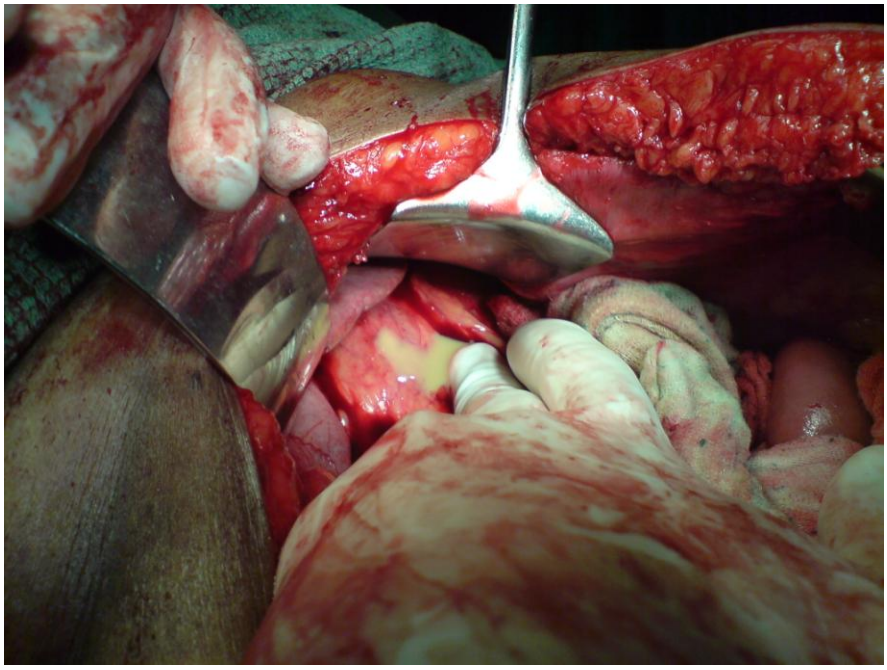
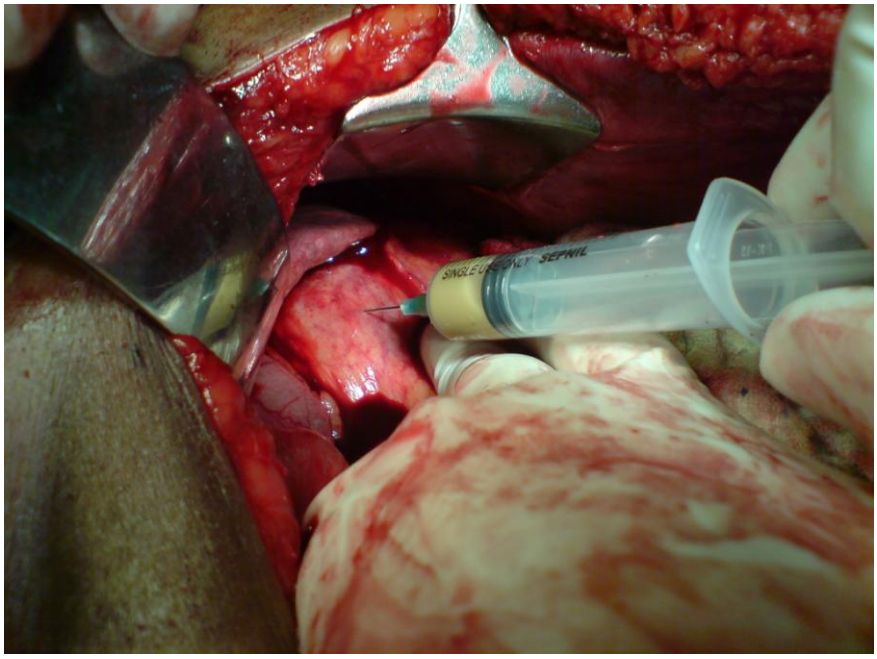


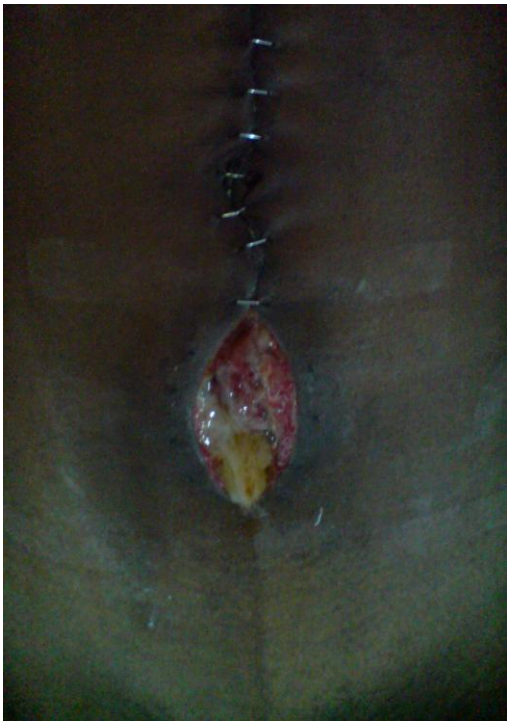
**INFECTED APPENDICECTOMY WOUND**



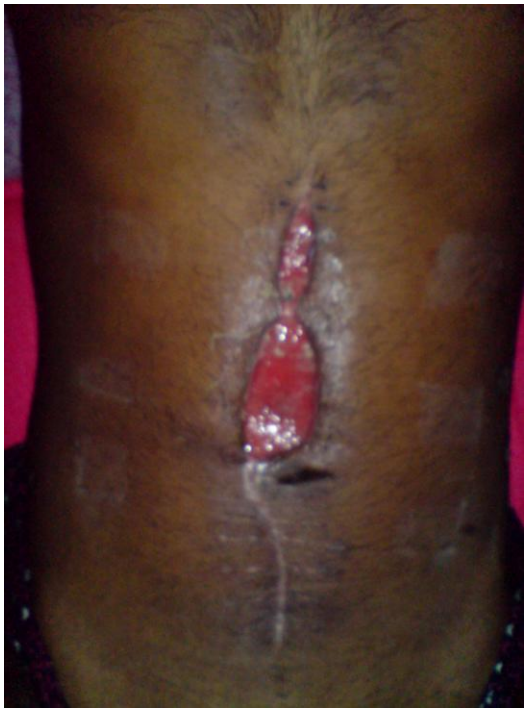


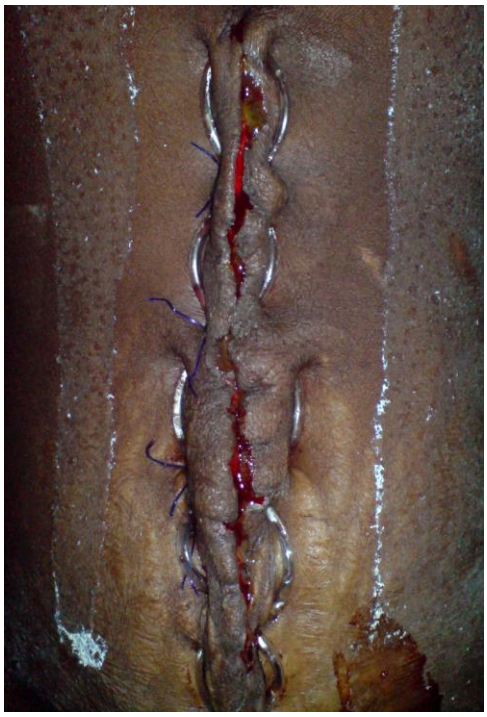
**ASPIRATION OF PUS INTRAOPERATIVELY**



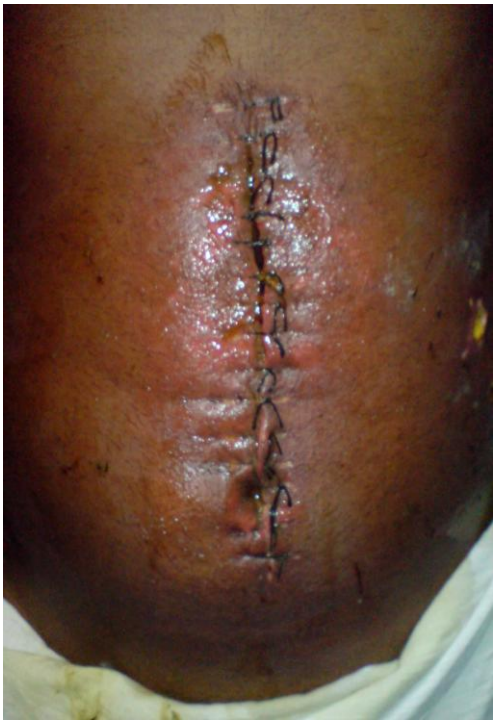


**WOUND DEHISCENCE**

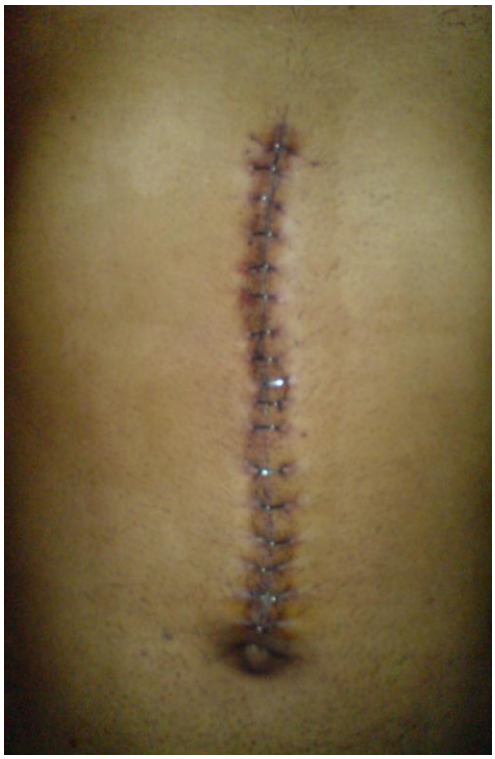




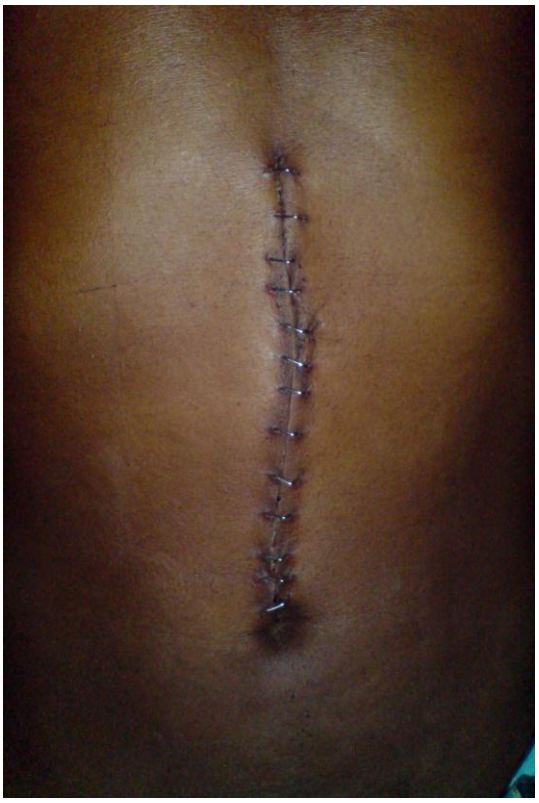
**TENSION SUTURING FOR BURST ABDOMEN**

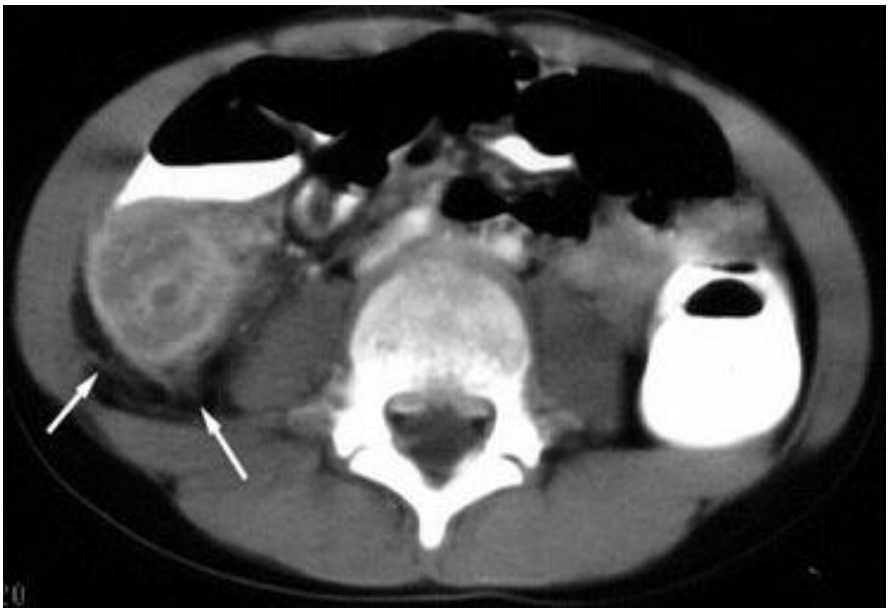


**POSTOPERATIVE BILE LEAK FROM THE SURGICAL SITE**

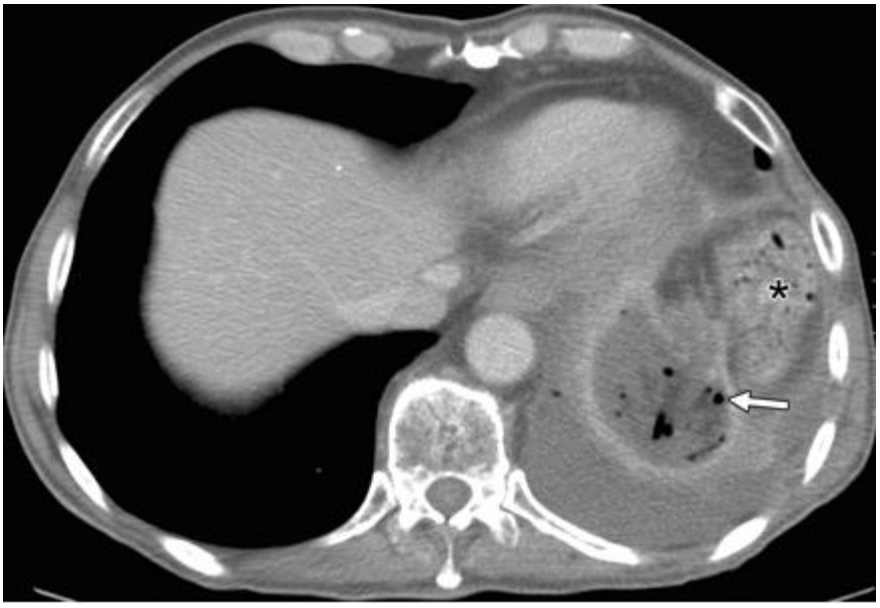


**HEALTHY HEALING SURGICAL WOUND**

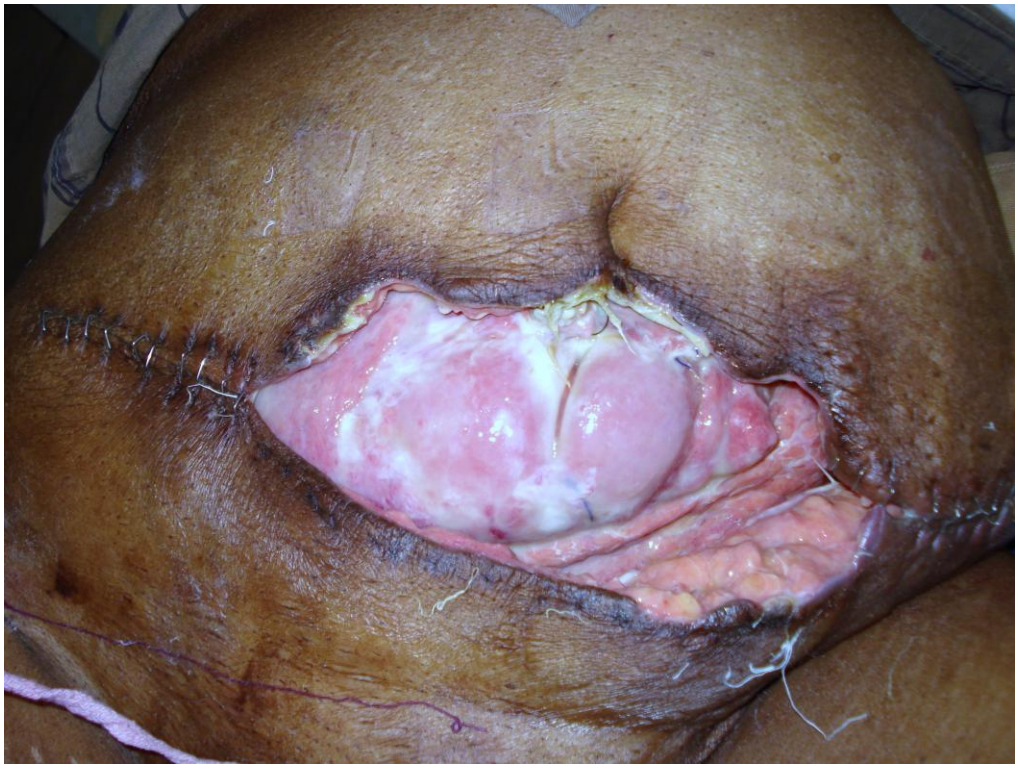




**INTRA ABDOMINAL ABSCESS**



**SUB PHRENIC ABSCESS**



**BURST ABDOMEN**

# INCIDENCE OF POSTPERATIVE SEPSIS AFTER EMERGENCY ABDOMINAL SURGERIES

**NAME** **AGE** **SEX** **IP NO**

**ASA GRADE**

**MAJOR ILLNESS** **JAUNDICE** **ANEMIA** **DM** **TB** **HT**

**NUTRITION** **SMOKER** **ALCOHOLIC**

**DURATION BETWEEN ONSET OF SYMPTOMS AND ADMISSION**

**SURGERY PLANNED**

**DURATION OF SURGERY AND PER OPERATIVE FINDINGS**

**INVESTIGATIONS**

**CULTURE SENSITIVITY**

**POSTOPERATIVE PERIOD**

**ANTIBIOTICS**

**SIGNS OF SEPSIS**

**IF PRESENT INTERVENTION DONE**

**OUTCOME**

# MASTER CHART

| S.NO. | NAME | AGE | SEX | IP NO. | DIAGNOSIS | PROCEDURE | ASA GRADE | WOUND CLASS | UNIT |
|---|---|---|---|---|---|---|---|---|---|
| 1. | Archana | 16 | F | 10466 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 2. | Rihana | 17 | F | 41263 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 3. | Rizwan | 18 | F | 34043 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 4. | Latha | 19 | F | 32556 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 5. | Divya | 20 | F | 30950 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 6. | Ramya | 21 | F | 32331 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 7. | Kamala | 22 | F | 41516 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 8. | Sabiya | 24 | F | 24328 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 9. | Thulasiammal | 24 | F | 26496 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 10. | Josephrani | 29 | F | 27926 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 11. | Palaniyammal | 30 | F | 33282 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 12. | Govindhammal | 31 | F | 35739 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 13. | Rukmani | 35 | F | 14100 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 14. | Maheshwari | 35 | F | 21858 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 15. | Saraswathy | 35 | F | 27452 | Intestinal obstruction | Adhesion Release | I | CLASS II | S6 |
| 16. | Nagammal | 35 | F | 32287 | Intestinal obstruction | Adhesion Release | I | CLASS II | S1 |
| 17. | Palaniammal | 36 | F | 36696 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 18. | Kalamani | 39 | F | 50323 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 19. | Govindammal | 30 | F | 20365 | Obstructed Femoral Hernia | Hernirrhaphy | I | CLASS III | S3 |
| 20. | Chinnamani | 48 | F | 25498 | Obstructed Hernia | Resection Anastamosis | I | CLASS III | S4 |
| 21. | Jansi | 19 | F | 23645 | Ileal Perforation | Resection Anastamosis | I | CLASS IV | S6 |
| 22. | Rithika | 25 | F | 40316 | Appendicular Perforation | Appendicectomy | I | CLASS IV | S1 |
| 23. | Basavamma | 32 | F | 42146 | Gastric Perforation | Perforation Closure | I | CLASS IV | S3 |
| 24. | Radha | 35 | F | 13441 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 25. | Poovathal | 35 | F | 30722 | Ruptured Liver Abscess | Laparotomy | I | CLASS IV | S1 |
| 26. | Paulraj | 17 | M | 28906 | Blunt injury | Laparotomy | I | CLASS I | S4 |
| 27. | Selvalen | 18 | M | 30201 | Blunt injury | Laparotomy | I | CLASS I | S3 |
| 28. | Sasikumar | 19 | M | 26976 | Blunt injury | Splenectomy | I | CLASS I | S4 |
| 29. | Kathirvel | 30 | M | | Blunt injury | Splenectomy | I | CLASS I | S4 |
| 30. | Sabari | 13 | M | 49979 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 31. | Dharunkumar | 14 | M | 32595 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |

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|---|---|---|---|---|---|---|---|---|---|
| 32. | Albert moses | 15 | M | 40500 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 33. | Sivamani | 15 | M | 49929 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 34. | kirubakaran | 15 | M | 43246 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 35. | Sivakumar | 16 | M | 20021 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 36. | Sivaram | 17 | M | 26296 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 37. | Manojkumar | 17 | M | 27893 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 38. | Ravichandran | 18 | M | 41141 | Acute Appendicitis | Appendicectomy | I | CLASS II | S5 |
| 39. | Deepak | 18 | M | 41720 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 40. | Kanagaraj | 19 | M | 16308 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 41. | Vikram | 20 | M | 29641 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 42. | Boopesh kanna | 20 | M | 37401 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 43. | Muthukumar | 20 | M | 41613 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 44. | Anand | 21 | M | 29789 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 45. | Umasankar | 21 | M | 32333 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 46. | David | 22 | M | 41579 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 47. | Ramesh | 22 | M | 18571 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 48. | Karthik | 22 | M | 34004 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 49. | Mahendrakumar | 22 | M | 42640 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 50. | Alagendiran | 23 | M | 21968 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 51. | Boopathy | 23 | M | 25261 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 52. | Silambarasan | 23 | M | 39442 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 53. | Kabeer | 25 | M | 29952 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 54. | Jegan | 26 | M | 8207 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 55. | Rajan | 26 | M | 23945 | Acute Appendicitis | Appendicectomy | I | CLASS II | S5 |
| 56. | Senthil | 26 | M | 33274 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |
| 57. | Murugesh | 26 | M | 37572 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 58. | Arumugam | 27 | M | 42312 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 59. | Balaguru | 27 | M | 21258 | Intestinal obstruction | Adhesion Release | I | CLASS II | S1 |
| 60. | Soundarrajan | 29 | M | 611 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 61. | Senthilkumar | 29 | M | 13224 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 62. | Nagaraj | 29 | M | 22644 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 63. | Selvam | 30 | M | 41639 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 64. | Anand | 33 | M | 23298 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 65. | Erusam | 33 | M | 42141 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 66. | Chinnamna | 35 | M | 1863 | Acute Appendicitis | Appendicectomy | I | CLASS II | S6 |

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| 67. | Ayyanar | 35 | M | 26801 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 68. | Balan | 35 | M | 28276 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 69. | Radhakrishnan | 35 | M | 29515 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 70. | Munusamy | 35 | M | 40827 | Acute Appendicitis | Appendicectomy | I | CLASS II | S4 |
| 71. | Mani | 37 | M | 7826 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 72. | Govindhan | 38 | M | 29582 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 73. | Jayaraj | 39 | M | 2021 | Acute Appendicitis | Appendicectomy | I | CLASS II | S3 |
| 74. | Murugan | 40 | M | 32332 | Acute Appendicitis | Appendicectomy | I | CLASS II | S2 |
| 75. | Kaja Moideen | 44 | M | 35245 | Acute Appendicitis | Appendicectomy | I | CLASS II | S5 |
| 76. | Edward | 52 | M | 5620 | Intestinal obstruction | Adhesion Release | I | CLASS II | S6 |
| 77. | Suppan | 57 | M | 8865 | Acute Appendicitis | Appendicectomy | I | CLASS II | S1 |
| 78. | Kamaraj | 18 | M | 27271 | Obstructed Hernia | Hernioplasty | I | CLASS III | S1 |
| 79. | Satheesh | 21 | M | 37470 | Intestinal obstruction | Laparotomy | I | CLASS III | S6 |
| 80. | Blaguru | 25 | M | 7637 | Sigmoid Volvulus | Resection Anastamosis | I | CLASS III | S1 |
| 81. | Kaliappan | 30 | M | 32493 | Sigmoid volvulus | Hartman's procedure | I | CLASS III | S2 |
| 82. | Sikander Basha | 32 | M | 29314 | Obstructed Hernia | Hernioplasty | I | CLASS III | S1 |
| 83. | Ramakrishnan | 37 | M | 40362 | Intestinal obstruction | Adhesion Release | I | CLASS III | S1 |
| 84. | Krishnamoorthy | 45 | M | 1718 | Intestinal obstruction | Resection Anastamosis | I | CLASS III | S1 |
| 85. | Rangasamy | 52 | M | 42893 | Intestinal obstruction | Adhesion Release | I | CLASS III | S1 |
| 86. | Palani | 75 | M | 24751 | Obstructed Hernia | Hernioplasty | I | CLASS III | S1 |
| 87. | Prasanna | 16 | M | 43195 | Small bowel gangrene | Resection Anastamosis | I | CLASS IV | S6 |
| 88. | Raman | 19 | M | 4325 | Appendicular Perforation | Appendicectomy | I | CLASS IV | S6 |
| 89. | Sillu | 20 | M | 12392 | Appendicular Perforation | Appendicectomy | I | CLASS IV | S1 |
| 90. | Kattupitchai | 21 | M | 52069 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S3 |
| 91. | Ismail | 22 | M | 30102 | Appendicular Abscess | Drainage | I | CLASS IV | S2 |
| 92. | Mariappan | 23 | M | 1415 | Jejenal Perforation Mesentery Tear | Resection Anastamosis | I | CLASS IV | S4 |
| 93. | Abudhahir | 24 | M | 5340 | Appendicular Perforation | Appendicectomy | I | CLASS IV | S4 |
| 94. | Ramesh | 24 | M | 29972 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 95. | Vijayakumar | 24 | M | 49450 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S3 |
| 96. | Ayyasamy | 25 | M | 771 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 97. | Kalaiarasan | 26 | M | 49163 | Appendicular Abscess | Appendicectomy | I | CLASS IV | S2 |
| 98. | Abuthaheer | 27 | M | 32943 | Appendicular Perforation | Appendicectomy | I | CLASS IV | S4 |
| 99. | Nandhakumar | 27 | M | 23658 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 100. | Ayyappan | 29 | M | 1099 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 101. | Anand | 29 | M | 7529 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 102. | Thangavel | 30 | M | 29423 | Appendicular Abscess | Drainage | I | CLASS IV | S2 |
| 103. | Muthusamy | 30 | M | 14819 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 104. | Muthu | 30 | M | 30736 | Ileal Perforation | Perforation Closure | I | CLASS IV | S1 |

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|---|---|---|---|---|---|---|---|---|---|
| 105. | Kumar | 31 | M | 42760 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S6 |
| 106. | Saktthivel | 31 | M | 30770 | jejunal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 107. | Mahendran | 31 | M | 34514 | Stab Injury | Laparotomy | I | CLASS IV | S3 |
| 108. | Shanmugam | 32 | M | 1025 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 109. | Vellingiri | 32 | M | 5413 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 110. | Paulraj | 32 | M | 16952 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 111. | Anand | 32 | M | 33620 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 112. | Narayanasamy | 34 | M | 29682 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S3 |
| 113. | Farooq | 34 | M | 49550 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 114. | Rangasamy | 35 | M | 14032 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 115. | Sundaram | 35 | M | 23464 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 116. | Sivanandham | 35 | M | 25292 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S3 |
| 117. | Murugan | 35 | M | 48835 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 118. | Krishnasamy | 35 | M | 38084 | Liver Abscess | Laparotomy&drainage | I | CLASS IV | S3 |
| 119. | Anbarasan | 38 | M | 27465 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 120. | Abdul rahman | 38 | M | 49097 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S3 |
| 121. | Subramani | 39 | M | 38204 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S4 |
| 122. | Othiyappan | 41 | M | 31006 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 123. | Balasubramani | 42 | M | 26483 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 124. | Arumugam | 45 | M | 39939 | Duodenal Perforation | Perforation Closure | I | CLASS IV | S6 |
| 125. | Murugan | 46 | M | 27795 | Ileal Perforation | Perforation Closure | I | CLASS IV | S1 |
| 126. | Murugesan | 47 | M | 35828 | Ruptured Liver Abscess | Laparotomy&drainage | I | CLASS IV | S4 |
| 127. | Veeran | 48 | M | 30927 | Ileal Perforation | Perforation Closure | I | CLASS IV | S2 |
| 128. | Rani | 40 | F | 40922 | Acute cholecystitis | Cholecystectomy | II | CLASS II | S4 |
| 129. | Nanjammal | 50 | F | 30665 | Acute Appendicitis | Appendicectomy | II | CLASS II | S1 |
| 130. | Thilagavathy | 40 | F | 33873 | Appendicular Perforation | Appendicectomy | II | CLASS IV | S2 |
| 131. | Karuppan | 53 | M | 10208 | Blunt Injury | Laparotomy | II | CLASS I | S6 |
| 132. | Babu | 32 | M | 50369 | Pancreatitis | Laparotomy | II | CLASS IV | S1 |
| 133. | Arumugam | 38 | M | 4903 | Pancreatitis | Laparotomy | II | CLASS IV | S1 |
| 134. | Bijai | 40 | M | 49137 | Duodenal Perforation | Perforation Closure | II | CLASS IV | S2 |
| 135. | Dhandapani | 40 | M | 2255 | Ileal Perforation | Perforation Closure | II | CLASS IV | S2 |
| 136. | Pattu | 52 | M | 15455 | Appendicular Perforation | Appendicectomy | II | CLASS IV | S4 |
| 137. | Shajuddin | 65 | M | 40117 | Appendicular Perforation | Appendicectomy | II | CLASS IV | S5 |
| 138. | Subramani | 65 | M | 1393 | Duodenal Perforation | Perforation Closure | II | CLASS IV | S4 |
| 139. | Louis | 65 | M | 4391 | Duodenal Perforation | Perforation Closure | II | CLASS IV | S6 |
| 140. | Thamburaj | 65 | M | 23786 | Duodenal Perforation | Perforation Closure | II | CLASS IV | S3 |

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|---|---|---|---|---|---|---|---|---|---|
| 141. | Periyapalani | 65 | M | 24749 | Duodenal Perforation | Perforation Closure | II | CLASS IV | S1 |
| 142. | Venkatachalam | 69 | M | 49125 | Appendicular perforation | Appendicectomy | II | CLASS IV | S2 |
| 143. | Karuppathal | 60 | F | 41446 | Intestinal obstruction | Adhesion Release | III | CLASS III | S6 |
| 144. | Rajagiriammal | 67 | F | 39940 | Intestinal obstruction | Adhesion Release | III | CLASS III | S6 |
| 145. | Govindammal | 59 | F | 18940 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S1 |
| 146. | Bhagyam | 60 | F | 32736 | Small bowel gangrene | Resection Anastamosis | III | CLASS IV | S3 |
| 147. | Angathal | 70 | F | 12272 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S2 |
| 148. | Swamiappan | 64 | M | 29528 | Blunt injury | Laparotomy | III | CLASS I | S2 |
| 149. | Muthusamy | 75 | M | 8675 | Intestinal obstruction | Adhesion release | III | CLASS II | S6 |
| 150. | Mani | 65 | M | 582 | Intestinal obstruction | Iliostomy | III | CLASS III | S1 |
| 151. | Rangasamy | 65 | M | 19054 | Intestinal obstruction | Laparotomy | III | CLASS III | S1 |
| 152. | Velusamy | 70 | M | 25968 | Obstructed Hernia | Resection Anastamosis | III | CLASS III | S1 |
| 153. | Narayanan | 20 | M | 32692 | Small bowel gangrene | Resection Anastamosis | III | CLASS IV | S3 |
| 154. | Dhandapani | 40 | M | 2255 | Small bowel gangrene | Resection Anastamosis | III | CLASS IV | S2 |
| 155. | Gopal | 40 | M | 14859 | SmallIntestine Gangrene | Resection Anastamosis | III | CLASS IV | S1 |
| 156. | Devaraj | 42 | M | 18987 | Fecal Fistula | Transverse Colostomy | III | CLASS IV | S1 |
| 157. | Subramani | 45 | M | 71011 | Ileocecal mass | Iliotransverse Anastamosis | III | CLASS IV | S2 |
| 158. | Srinivasan | 47 | M | 32740 | Small bowel gangrene | Resection Anastamosis | III | CLASS IV | S3 |
| 159. | Muthusamy | 50 | M | 10129 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S6 |
| 160. | Kudulingam | 50 | M | 40417 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S1 |
| 161. | Manivannan | 50 | M | 67139 | Ileal Perforation | Perforation Closure | III | CLASS IV | S3 |
| 162. | Kalimuthu | 50 | M | 29717 | Ruptured Liver Abscess | Laparotomy | III | CLASS IV | S3 |
| 163. | Sivasubramani | 55 | M | 16947 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S4 |
| 164. | Balan | 55 | M | 25426 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S4 |
| 165. | Subramani | 56 | M | 28196 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S3 |
| 166. | Muthusamy | 58 | M | 25273 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S3 |
| 167. | Soman | 60 | M | 19664 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S4 |
| 168. | Gopal | 60 | M | 35878 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S4 |
| 169. | Samshudin | 65 | M | 2002 | Jejenal Growth | Resection Anastamosis | III | CLASS IV | S1 |
| 170. | Rangasamy | 70 | M | 29669 | Duodenal Perforation | Perforation Closure | III | CLASS IV | S3 |
| 171. | Chinnammal | 70 | F | 25801 | Intestinal obstruction | Resection Anastamosis | IV | CLASS II | S4 |
| 172. | Kamalam | 50 | F | 21849 | Ca Stomach | Sub total gastrectomy | IV | CLASS IV | S6 |
| 173. | Kaveri | 64 | F | 23623 | Fecal fistula | Loop colostomy | IV | CLASS IV | S2 |
| 174. | Krishnan | 75 | M | 16376 | Intestinal obstruction | Laparotomy | IV | CLASS III | |
| 175. | Padmavathy | 55 | M | 20824 | CarcinomaSigmoid | Colostomy | IV | CLASS IV | S3 |

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|---|---|---|---|---|---|---|---|---|---|
| 176. | Murugan | 56 | M | 29195 | Ileal Perforation | Resection Anastamosis | IV | CLASS IV | S1 |
| 177. | Raja | 57 | M | 17448 | Gastric Perforation | Perforation Closure | IV | CLASS IV | S1 |
| 178. | Palani | 57 | M | 2464 | Liver abscess | Laparotomy & wash | IV | CLASS IV | S4 |
| 179. | Subramani | 57 | M | 37053 | Liver Abscess | Laparotomy&drainage | IV | CLASS IV | S3 |
| 180. | Muthusamy | 60 | M | 42901 | Stab injury Abdomen Jejenal perforation | Perforation Closure | IV | CLASS IV | S1 |