"A STUDY FOR ASSESSMENT OF RISK FACTORS AND INCIDENCE OF SURGICAL SITE INFECTION IN CASES OPERATED FOR PERITONITIS IN KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"

BY

REG NO: BH0119008

Dissertation

Submitted to the
KAHER, Belagavi, Karnataka

In partial fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)

in

GENERAL SURGERY

JAWAHARLAL NEHRU MEDICAL COLLEGE
BELAGAVI, KARNATAKA

APRIL -2022

KLE Academy of Higher Education and Research
Belagavi, Karnataka

Endorsement

This is to certify that the dissertation entitled "**A STUDY FOR ASSESSMENT OF RISK FACTORS AND INCIDENCE OF SURGICAL SITE INFECTION IN CASES OPERATED FOR PERITONITIS IN KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"** is a bonafide research work done by **REG NO. BH0119008.**

Dr. A .S Gogate, MS
Professor and Head,
Department of General Surgery,
J. N. Medical College,
Belagavi

Date:
Place: Belagavi

Dr. N. S. Mahantshetti, MD
Principal,
J. N. Medical College,
Belagavi

Date:
Place: Belagavi

PLAGIARISM CERTIFICATE

KLE
JAWAHARLAL NEHRU MEDICAL COLLEGE
(Recognized by Medical Council of India, New Delhi)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831 - 2471350 0831 - 2470750 www.klejnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 16-11-2021

ACCEPTANCE LETTER

The softcopy of thesis entitled **"A STUDY FOR ASSESSMENT OF RISK FACTORS AND INCIDENCE OF SURGICAL SITE INFECTION IN CASES OPERATED FOR PERITONITIS IN KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI."** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **06%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BH0119008.
Postgraduate Student,
2019-20 Batch,
Department of General Surgery,
J. N. Medical College, Belagavi.

LIST OF ABBREVIATIONS

SSI	-	Surgical Site Infection
Hrs	-	Hours
FGF-2	-	Fibroblast Growth Factor-2
ECM	-	Extracellular matrix
PDGF	-	Platelet-derived growth factor
TGF	-	Transforming Growth Factor
EGF	-	Epidermal Growth Factor
IL-1	-	Interleukin-1
TNF	-	Tumour Necrosis Factor
KGF	-	Keratinocyte Growth Factor
IGF-1	-	Insulin like Growth Factor-1
UV	-	Ultraviolet
DOCA	-	Deoxycorticosterone acetate
-ve	-	Negative
+ve	-	Positive
SIRS	-	Systemic Inflammatory response Syndrome.
MODS	-	Multiple Organ Dysfunction Syndrome
FNAC	-	Fine Needle Aspiration Cytology

CT scan	-	Computed Tomography scan
CDC	-	Centers for Disease Control and Prevention
PBP- 2a	-	penicillin-binding protein 2a
MRSA	-	Methicillin Resistant Staphylococcus aureus
CECT	-	Contrast Enhanced Computed Tomography
OPD	-	Out Patient Department
NSAID	-	Non-Steroidal Anti-Inflammatory Drugs

ABSTRACT

Background: Surgical site infections have plagued surgeons since time immemorial. There is significant morbidity and mortality associated with surgical site infections.

Objectives: In this study we tried to identify the incidence of SSI in patients operated for peritonitis, various patient and procedure related factors, the various organism associated with the SSIs.

Methodology: This study was done in the Department of General Surgery, in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre. In this study 75 patients were included who were above the age of 12 years, any gender, and were operated for peritonitis due to various causes between January 2020 to march 2021. Patient data was recorded for all patients who were examined post-operatively for soakage along with culture and antibiotic testing and analysis of data was done.

Results: Incidence of abdominal SSI in cases operated for peritonitis is 26% in our study. Risk factors like hypoalbuminemia, anaemia and diabetes mellitus, obesity, smoking was associated with increased wound infection rate. SSI incidence was most with perforative peritonitis when compared with other causes of peritonitis. E. coli was the most common organism isolated from infected wounds in this study. Most of the organisms were sensitive to higher antibiotics.

Conclusion: Surgical site infection is rapidly identified as a degree of the quality of patient care by surgeons, infection control practitioners, health planners and public. Judicious use of antibiotics and tailoring them according to culture profile whenever possible is needed to reduce SSI rate. A reduction in the infection rate to a minimal level could have significant benefits; both in terms of mortality and morbidity.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF GRAPHS

INTRODUCTION

Surgical site infection can occur following any surgical procedure where there is a breach in the mechanical or anatomical barriers. These are connected with high morbidity, death, and longer hospital stays, putting a financial drain on the family and society. (1)(2)

Surgical site infection occurrence is an interplay between the source of infection, the virulence of organism and host immune status. Efforts are being made to concentrate on these variables in order to limit the occurrence of surgical site infections.(3)

Peritonitis is associated with higher morbidity and mortality rates of 17 to 63 percent.(4) Peritonitis is significantly associated with surgical site infections and has an influence on the public health and clinical practice.

Peritonitis secondary to any perforation already exposes the peritoneum to faecal contaminants having a high bacterial load, hence a high incidence of surgical site infections is already expected. If quantified a surgical site with $> 10^5$ organism has high risk of SSI development.(5) In such cases the factors that play a part in the occurrence of SSI even in clean cases may be additive.

A close monitoring is of utmost significance in such patients as surgical site infection is a potential complication in peritonitis patients that undergo surgical management

A study of the risk factors, micro-organisms and antibiotic sensitivity patterns can help us explore ways to reduce the incidence of surgical site infections in such patients who already are expected to have a high incidence of surgical site infections.(6)

This study can help us gather knowledge, build and make changes in the strategy to reduce if not overcome this menace which results in increased hospital stay, treatment costs and indirect losses such as productivity loss, dissatisfaction of patients and reduced quality of life.

Postoperative care, combined with audit and surveillance of SSI rates and providing feedback, as well as education to health care personnel is required to help formulate proper techniques and intervention to prevent surgical site infection. Hence this study has been undertaken to know the incidence of SSI because data is scarce in this part of the country. Through this study we also want to identify the various risk factors associated with development of SSI in cases operated for peritonitis. An improved knowledge of aetiological variables, as well as timely medical care, are required to improve outcomes in patients with peritonitis.

OBJECTIVE

- To find out the incidence of surgical site infection in cases operated for peritonitis in our setup.
- To identify the risk factors associated with the development of surgical site infection in cases operated for peritonitis.
- To study and compare the common organisms causing SSI including their antibiotic susceptibility.

REVIEW OF LITERATURE

Infection treatment and control has now become a very important part of the surgical practice.

In the past, infection of a surgical wound was common as there was a prominent lack of hygiene and knowledge was scarce.

Father of Indian surgery **Susuruta (6th century BC)** in his ancient Hindu literature "Susuruta Samhita" emphasised the importance of surgical cleanliness and upkeep.

1683 – **Antony Van Leeuwenhock** -saw bacteria under self-built scope.

Mention regarding the 1st laparotomy done for peritonitis dates back to 1687 performed by **Stal pert von der weil,** whereas Elective abdominal surgery began in Danville, Kentucky in 1809 by **Ephraim McDowell**.

Frank Zurfley performed the first known exploratory laparotomy in 1842 for a patient suffering from peritoneal haemorrhage

By the 19th century, physicians' and scientists' observations had become the bedrock of knowledge about the causation, prevention, and treatment of surgical infections.

Ignaz Semmelweis in 1846 proposed the practice of hand washing after noticing the drastic cut down of puerperal fever in obstetric clinic following hand disinfection(7). He was ridiculed back then, but 20 years later **Louis Pasteur** offered theoretical explanation for his observations. Pasteur was able to prove that specific microbes are the cause of contagious disease.(8)

As seen by **Joseph Lister**, more than half of his amputation victims died as a result of a postoperative infection. He advocated the notion of sterile surgery using

carbolic acid (phenol) based on Pasteur's results in 1865.(9) This resulted in a decrease in post-operative infections(8)

Robert Koch (1843-1910) proved the significance of bacteria in disease transmission and proposed the Koch's postulate for identifying organisms with specific diseases. Even today, these postulates are crucial to our knowledge of surgical infections(9)

Charles McBurney, New York college of physicians and surgeons was the one to perform the 1st operation (open appendectomy) to treat infection by "source control" (elimination of the infection source), published in 1889.(10)

Dr. Alexander Fleming discovered the first successful antimicrobial agent (Penicillin) by serendipity in the twentieth century, which led to the creation of numerous more antimicrobial agents for use.

Howard Florey was the first to use penicillin in a therapeutic setting in 1940.

Concurrent advances in clinical microbiology cleared that Aerobes and anaerobes could work together to induce significant soft tissue and intra-abdominal infections. This led to the idea that local bacteria were non-pathogenic until they entered a sterile cavity, and that the majority of surgical infections were polymicrobial in type.(11,12)

Trials revealed significant evidence that efficient source control, as well as the administration of antimicrobial medicines aimed against both aerobes and anaerobes, was essential for optimum therapy.

The discovery of the first cytokines provided insight into the human organism's reaction to infection. With a better knowledge of the numerous pathways involved in the immune response to infectious organism invasion, innovative drugs aimed at lowering the inflammatory response to infection, which appears to be the underlying cause of many organ dysfunction and failure, have been developed.

Classification of surgical wound(13)

1. Clean

An uninfected surgical wound with minimal inflammation and the colonised visceral organs is not accessed.

Rate of infection<2%

2. Clean -contaminated

Wound in which the colonised organs are entered under regulated settings and without unusual contamination.

Rate of infection ~ 30%

3. Contaminated

Wounds with significant disruptions in sterile method or significant leaking from the gastrointestinal system, as well as wounds with acute, non-purulent inflammation. More contamination with micro-organisms is occurred at the operative site without obvious microbial infection.

Rate of infection ~ 60%

4. Dirty

Traumatic wounds having residual devitalized tissue, as well as those involving existing tissue Infection or ruptured viscera leading to active microbial infection.

Rate of infection > 60%.

HEALING OF WOUND

At the molecular level, wound healing is a continuum of complicated interconnected physiological processes.

Healing can be separated into three stages for descriptive purposes: inflammatory stage

proliferative stage and

Phase of maturation(14)

INFLAMMATORY PHASE:

Blood vessel damage after an acute tissue injury exposes the collagen in the vessel wall(subendothelial) to platelets. Initially there is intense vasoconstriction followed by vasodilatation and enhanced vascular permeability.

First of all, haemostasis occurs. Platelets, (1^{st} to arrive) come to the site of injury and their degranulation results in the release of serotonin and various cytokines.(15)

The released Serotonin and histamine (produced by mast cells) cause a response leading to a reversible gap between the endothelium. This allows the neutrophils and monocytes to reach the site of insult. This migration is possible because of the platelet released cytokines and chemotactic cytokines from macrophages. Neutrophils reach first but disappear within 24 hrs. After 24-48 hrs cells forming the majority are macrophages.(15)

PROLIFERATIVE PHASE

This phase is marked by the multiplication of the cells which reach the site of injury. Cells include endothelial cells, fibroblasts. This phase involves the formation of granulation tissue. Cytokine FGF-2 is involved.(16)

Epithelialisation of wound occurs in 48 hrs and there is development of collagen.in addition to this, angiogenesis occurs.

ANGIOGENENSIS:

New blood vessel formation.

The broken basement membrane is re-deposited leading to capillary maturation.(17)

FIBROPLASIA

Fibroblasts which are typically dormant and sparse are chemoattracted to the inflammatory site, where they divide and create ECM components.(15)

EPITHELIALIZATION

Commences within hours ensuing damage. Initially, clot formation seals the wound quickly, followed by epithelial (epidermal) cell migration over the defect. Keratinocytes move to resurface the wound from the basal layer of the remaining epidermis or the depths of epithelium-lined dermal appendages. (15)

EXTRACELLULAR MATRIX

Acts as supportive framework for tissues. It plays an intricate function in controlling the behaviour of cells that come into its contact. Its cells produce macromolecular constituents such as collagen, laminin, elastin and fibronectin.

PHASE OF MATURATION

Collagen is the most prominent characteristic throughout the maturation process. The scar is mostly composed of the thick bundle of fibres that is characteristic of collagen. Wound contraction is more appreciated in wounds healing by secondary intention. Myofibroblasts are the cells responsible for this contraction. The wound is constantly remodelled in an attempt to return to its pre-injury form. At 3-4 months following surgery, the wound retains 70-80% of its initial tensile strength.(16)

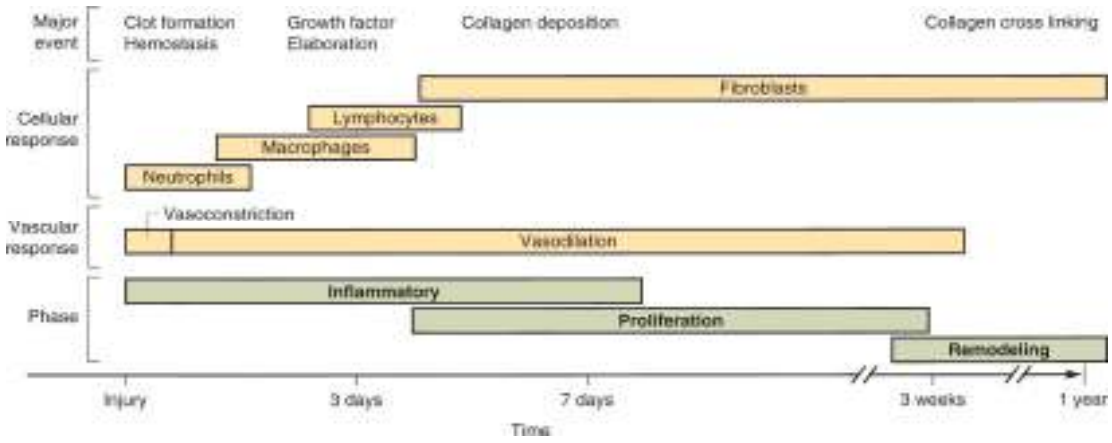


FIGURE 1: STAGES OF WOUND HEALING

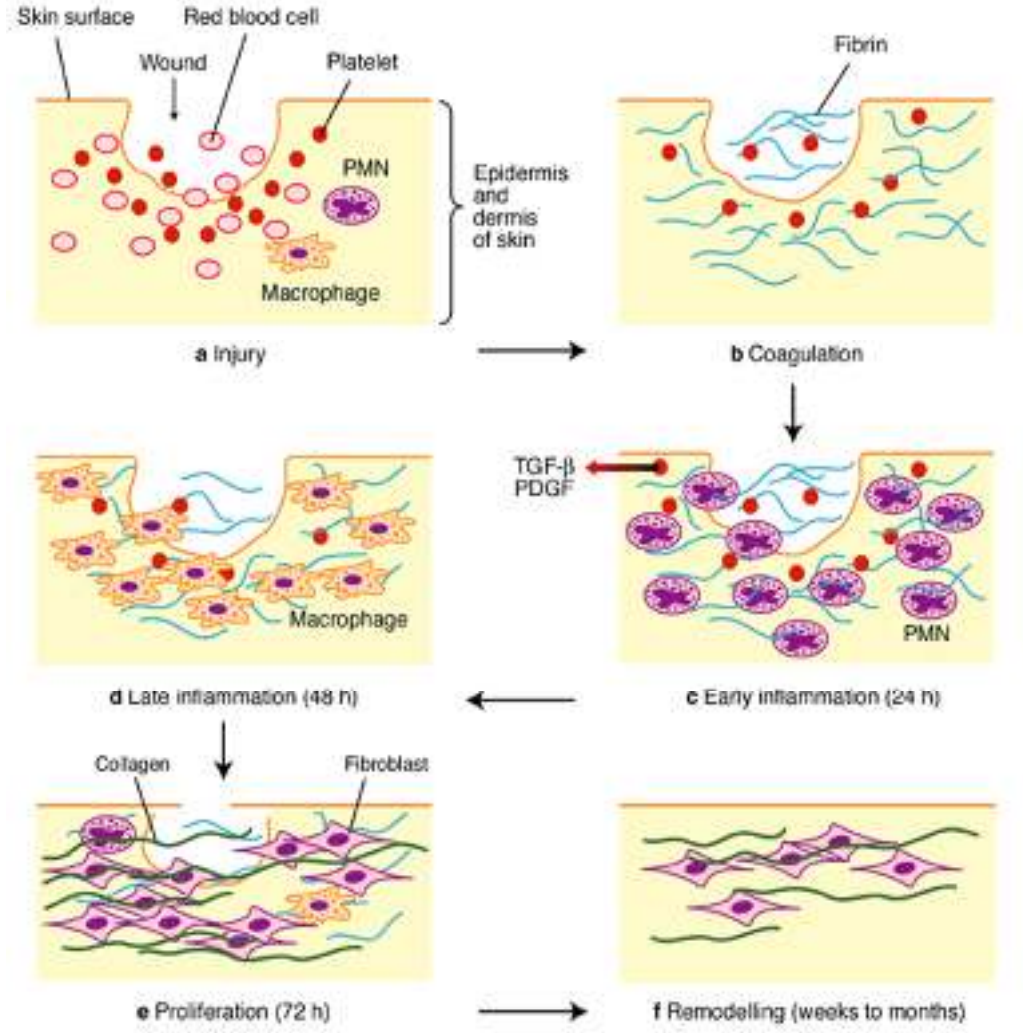


FIGURE 2: PHASES OF WOUND HEALING(18)

SECONDARY UNION OF A WOUND

When the edges of the wound are not approximated and needs a gap to be filled for healing. Difference of secondary union from primary is,

a) Here the epithelialization is formed after 4 to 5 days over the granulation tissue, hence it takes longer duration.

b) Granulation tissue formed in secondary union is usually in excess and has to be trimmed in order to provide optimal healing capacity of the wound.

c) The wound contraction occurs between 3 to 14 days, here the size of the gap decreases. Myofibroblasts play in an important role in this by its contractile action, it reduced the gap almost up to 80% of original size. This helps in reduction of time taken for healing.(19)

FACTORS AFFECTING WOUND HEALING(19)

A) Growth factors (soluble factors)

Growth factors released from damage wound site plays an important role by mitigating the cells and proliferation of cells and help in collagen synthesis.(20)

The following are important growth factors that help in healing:

a) PDGF

b) TGF

c) EGF

d) IL-1 AND TNF alpha

e) KGF

f) IGF -1

FACTORS INFLUENCING WOUND HEALING CAN BE CLASSIFIED UNDER

1) LOCAL FACTORS

a) Oxygenation to the tissue plays a vital role and can be affected by stasis, vasculitis, smoking or by various other causes of ischemia, anaemia and cardiopulmonary related issues.(21)

b) During surgical procedure, it is important to have good haemostasis, dead space should be eliminated, tissue destruction to be avoided due to excessive cautery use or by using ligatures which might cause strangulation.(22)

c) In case of peritonitis or intestinal leak it might cause intraabdominal sepsis.

d) Local wound dehiscence can be secondary to collected hematoma or infection.

e) UV radiations promote wound healing while other irradiations delays wound healing inhibiting its contraction.

f) Adequate blood supply to the wound, with reliable suture material and technique plays important role.(22)

2) GENERAL FACTORS

a) Protein and vitamin deficiency like methionine cysteine in former preventing collagen cross linkage and vitamin C in the latter which is important for helix formation by intracellular hydroxylation causes wound dehiscence.(21)

b) Old age associated with malnutrition, alteration in the metabolism , decrease skin elasticity and loss of muscle tone, have increase chances of wound failure.

c) Affect with change in temperature, higher temperature has quickened wound healing process where as cold temperature inhibits it.

d) Chronic diseases, carcinomas, dehydration leading to hypovolemia inhibits wound healing.(22)

e) Use of steroids (glucocorticoids) inhibits formation of new blood vessels and delays wound contraction long with inhibits collagen synthesis. Wound healing is promoted by anabolic steroids and DOCA.(20)

f) Obesity with increase fat tissue over local wound site prevents good approximation and decreases its blood supply and increases wound gape as it is good culture media for growth of organisms.

RISK FACTORS FOR SSI CAN BE DIVIDED ACCORDING TO THE FOLLOWING(23)

1) Patient related

2) Environmental related

3) Type of abdominal surgery

4) Type of pathogen

PATIENT FACTORS

1) Malnutrition

2) Obesity

3) Old age

4) Diabetes mellitus

5) Hypercholesterolemia

6) Chronic inflammation

7) Postoperative anaemia

8) Recent surgery in the past

9) Immunosuppression

ENVIRONMENTAL FACTORS INCLUDE(23–25)

1) Insufficient disinfectant and sterilization

2) Inadequate use of antisepsis

3) Decreased ventilation leading to increase moisture and humidity

4) Existence of foreign body

5) When a medication is contaminated.

TREATMENT FACTORS INCLUDE(26)

1) Increase preoperative hospitalization

2) Under coverage of antibiotics

3) In case of an emergency procedure

4) Postoperative prolonged hospital stay

5) In presence of prolonged postoperative drains.

TYPE OF PATHOGENS

THESE INCLUDE:

1) BACTERIA

Bacteria is present in the earth since millions of years even before the human race evolved. The important evolving need of the hour is to combat these pathogens in order to prevent sepsis.

To choose the required antibiotic, gram staining is important.(27)

Gram -ve or +ve is based on staining of cell wall of the bacteria.

A) If red → called gram negative

B) When blue, they are called gram positive.

Gram Negative Bacteria include the following, i.e., E. coli, klebsiella pneumonia. Proteus vulgaris, pseudomonas aeruginosa, Enterobacter species, proteus vulgaris. The various Gram-Positive organisms include, the ones lying over the skin termed as skin commensals. When the skin integrity is lost with any cut or incision, these bacteria become pathogenic.

These include, Staphylococcus aureus, staphylococcus epidermidis and streptococcus pyogenes.(28,29)

Some GI commensals include, Enterococci faecalis and faecium, they can cause nosocomial infections.

ANAEROBIC ORGANISMS

These organisms which lack catalase and they mainly colonise in the rectum and oropharynx.

The multiplication of these organisms happens through atmospheric air. These include, C. Perfringes, C. difficle, C. tetani, C. bacteroides.

The other bacteria of importance include(28)

Few Mycobacterium species, Nocardia. These are acid fast bacilli and are slowly growing. Their growth takes several weeks to months.(30)

VIRUSES (31)

Hepatitis B, Herpes simplex, herpes zoster, Epstein Barr virus, hepatitis C and cytomegalovirus.

FUNGI OF IMPORTANCE

They are identified by KOH mount, Giemsa and methamine silver. The various forms include yeast, budding forms with numerous branches with septations (3). Certain fungi can cause SSI in immunocompromised individuals. These include, C. albicans, mucor, Aspergillus fumigatus and Niger, Rhizopus, Cryptococcus neoformans.(30)

SURGICAL SITE INFECTION

DEFINITION(32)

A surgical site infection (SSI) is an infection that develops subsequently after surgery, in the part of the body where surgical procedure was done. (33)

Classified as per the depth of wound infection

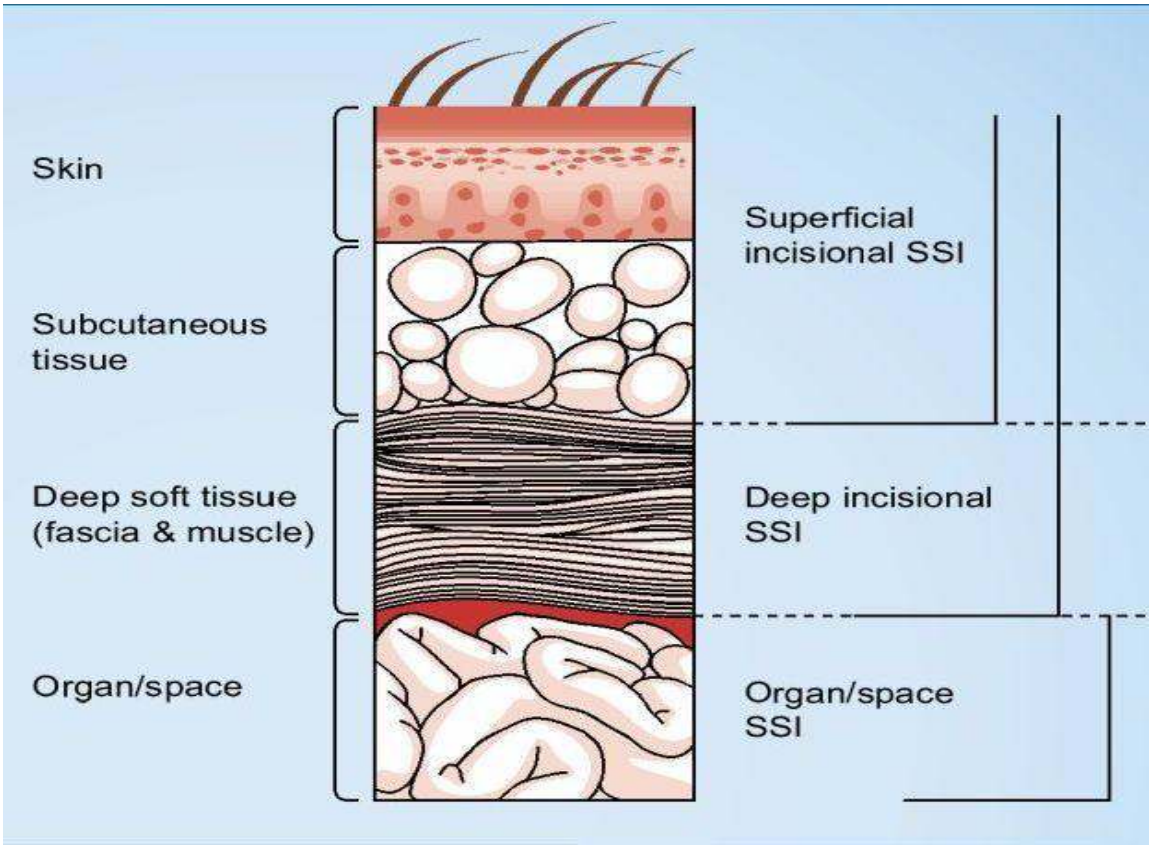


FIGURE 3: CLASSIFICATION OF SURGICAL SITE INFECTION

SUPERFICIAL INCISIONAL SURGICAL SITE INFECTION(32)

CRITERIA:

Occurs within 30 days post-operatively.

AND

Involves only skin or subcutaneous tissue

AND

Patient has at least one: -

a) Purulent discharge

b) Organism isolation has been done by microbiological method.

c) Superficial incision that is deliberately opened but culture or non-culture-based testing has not been performed.

   AND

   At least one of the signs and symptoms of inflammation is present.

d) Surgeon or attending physician declares it to be a superficial surgical site infection.

DEEP INCISIONAL SURGICAL SITE INFECTION (32)

Occurs within 30- or 90-days post operatively (different for different procedures)

30-day Surveillance			
Category	**Operative Procedure**	**Category**	**Operative Procedure**
AAA	Abdominal aortic aneurysm repair	LAM	Laminectomy
AMP	Limb amputation	LTP	Liver transplant
APPY	Appendix surgery	NECK	Neck surgery
AVSD	Shunt for dialysis	NEPH	Kidney surgery
BILI	Bile duct, liver or pancreatic surgery	OVRY	Ovarian surgery
CEA	Carotid endarterectomy	PRST	Prostate surgery
CHOL	Gallbladder surgery	REC	Rectal surgery
COLO	Colon surgery	SB	Small bowel surgery
CSEC	Cesarean section	SPLE	Spleen surgery
GAST	Gastric surgery	THOR	Thoracic surgery
HTP	Heart transplant	THYR	Thyroid and/or parathyroid surgery
HYST	Abdominal hysterectomy	VHYS	Vaginal hysterectomy
KTP	Kidney transplant	XLAP	Exploratory laparotomy

90-day Surveillance	
Category	**Operative Procedure**
BRST	Breast surgery
CARD	Cardiac surgery
CBGB	Coronary artery bypass graft with both chest and donor site incisions
CBGC	Coronary artery bypass graft with chest incision only
CRAN	Craniotomy
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]
[illegible]	[illegible]

AND

Involves deep soft tissue (muscle, fascial layers)

AND

Patient has at least one: -

a) Pus discharge from deep incision.

b) Spontaneous dehiscence of a deep incision or deliberate separation or aspiration by surgeon

   AND

   Organisms are isolated from the depth of tissue

   AND

   Presence of at least one of the signs and symptoms-fever, local pain or pain on examination.

c) Abscess / infection in deep incision detected by pathological or radiological examination.

ORGAN/SPACE SURGICAL SITE INFECTION(32)

Occurs within 30- or 90-days post-surgery (different for different procedures)

30-day Surveillance			
Category	**Operative Procedure**	**Category**	**Operative Procedure**
AAA	Abdominal aortic aneurysm repair	LAM	Laminectomy
AMP	Limb amputation	LTP	Liver transplant
APPY	Appendix surgery	NECK	Neck surgery
AVSD	Shunt for dialysis	NEPH	Kidney surgery
BILI	Bile duct, liver or pancreatic surgery	OVRY	Ovarian surgery
CEA	Carotid endarterectomy	PRST	Prostate surgery
CHOL	Gallbladder surgery	REC	Rectal surgery
COLO	Colon surgery	SB	Small bowel surgery
CSEC	Cesarean section	SPLE	Spleen surgery
GAST	Gastric surgery	THOR	Thoracic surgery
HTP	Heart transplant	THYR	Thyroid and/or parathyroid surgery
HYST	Abdominal hysterectomy	VHYS	Vaginal hysterectomy
KTP	Kidney transplant	XLAP	Exploratory laparotomy

90-day Surveillance	
Category	**Operative Procedure**
BRST	Breast surgery
CARD	Cardiac surgery
CBGB	Coronary artery bypass graft with both chest and donor site incisions
CBGC	Coronary artery bypass graft with chest incision only
CRAN	Craniotomy
FUSN	Spinal fusion
FX	Open reduction of fracture
HER	Herniorrhaphy
HPRO	Hip prosthesis
KPRO	Knee prosthesis
LAM	Pacemaker surgery
PVBY	Peripheral vascular bypass surgery
VSHN	Ventricular shunt

AND

Involves tissues deeper to fascial/muscle layers manipulated during surgery

AND

At least one of the following: -

a) pus discharge from a drain placed into the space or organ

b) micro-organisms identified by microbiological tests.

c) abscess / infection in organ or deep located space in abdominal cavity detected by pathological tests or radiological investigations.

AND

Has at least a criterion fulfilled for organ space infections.

Classification according to severity(34)

1 major SSI

Discharges pus, significant in quantity.

Patients are systemically ill.

2 minor SSI

Infected serous or purulent discharge, minimal in quantity

Patients -not ill systemically.

Various **scoring systems** for wound infection severity -help in audits and surveillance.

Some important scoring systems are-(35,36)

- **Southampton wound scoring system**
- **The ASEPSIS wound score**

SOUTHAMPTON WOUND GRADING SYSTEM

Grade	Appearance
0	Normal healing
I	Normal healing with mild bruising or erythema
Ia	Some bruising
Ib	Considerable bruising
Ic	Mild erythema
II	Erythema plus other signs of inflammation
IIa	At one point
IIb	Around sutures
IIc	Along wound
IId	Around wound
III	Clear or haemoserous discharge
IIIa	At one point only (≤2 cm)
IIIb	Along wound (>2 cm)
IIIc	Large volume
IIId	Prolonged (>3 days)
Major complication	
IV	Pus
IVa	At one point only (≤2 cm)
IVb	Along wound (>2 cm)
V	Deep or severe wound infection with or without tissue breakdown; haematoma requiring aspiration

THE ASEPSIS WOUND SCORE

Criterion	Points
Additional treatment	0
Antibiotics for wound infection	10
Drainage of pus under local anaesthesia	5
Debridement of wound under general anaesthesia	10
Serous discharge[a]	Daily 0–5
Erythema[a]	Daily 0–5
Purulent exudate[a]	Daily 0–10
Separation of deep tissues[a]	Daily 0–10
Isolation of bacteria from wound	10
Stay as inpatient prolonged over 14 days as result of wound infection	5

[a] Scored for 5 of the first 7 days only, the remainder being scored if present in the first 2 months.

PERITONEUM-a balloon into which organs are pressed from outside.(37)

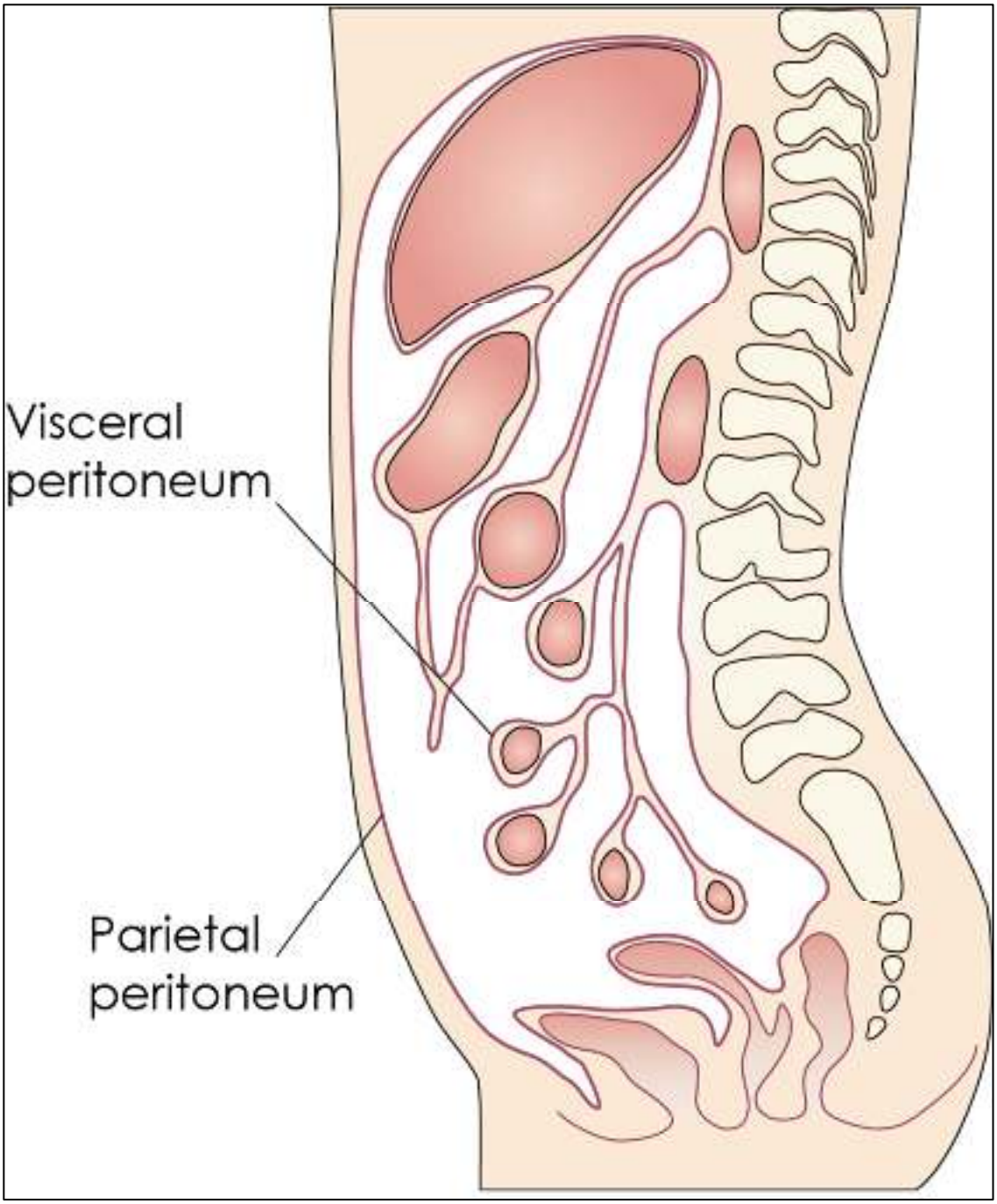
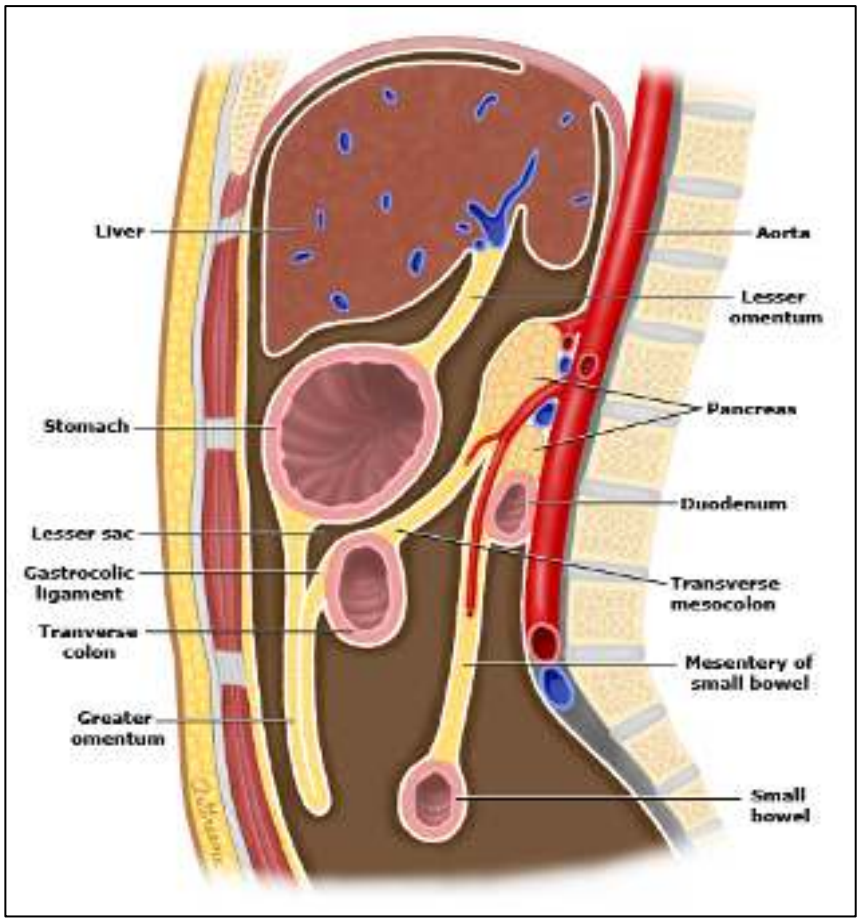


FIGURE 4: THE PERITONEUM

The peritoneum

- Divided into visceral (70% surface area) and parietal peritoneum (30% surface area).
- Heterogenous, semipermeable membrane.
- Surface area 1-2 m^2
- Peritoneal cavity -largest cavity in the body.
- Males – closed sac
- Females -open sac (via fallopian tubes)
- Acts like a cloth for the abdominal and pelvic organs.
- Healthy peritoneum – can be stretched without tearing.
- Peritoneal cavity divided into(38)
    - Greater sac (main compartment)
    - Lesser sac (smaller; behind stomach)
    - Connection between the two – EPIPLOIC FORAMEN (foramen of Winslow)
- Peritoneal space when healthy has neutral pressure oscillating between +ve and -ve pressure with diaphragmatic movements.(39)
- The peritoneal cavity never includes gas under normal conditions, while the volume of fluid may be raised in inflammatory conditions of the viscera(38)
- The peritoneal cavity never includes gas under normal conditions, while the volume of fluid may be raised in inflammatory conditions of the viscera(38)



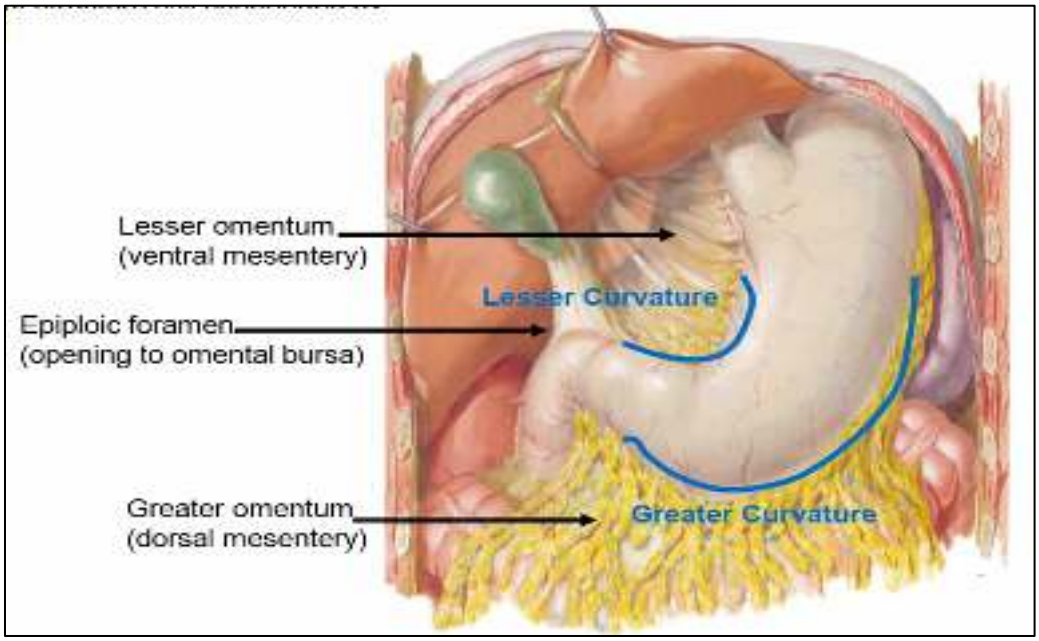
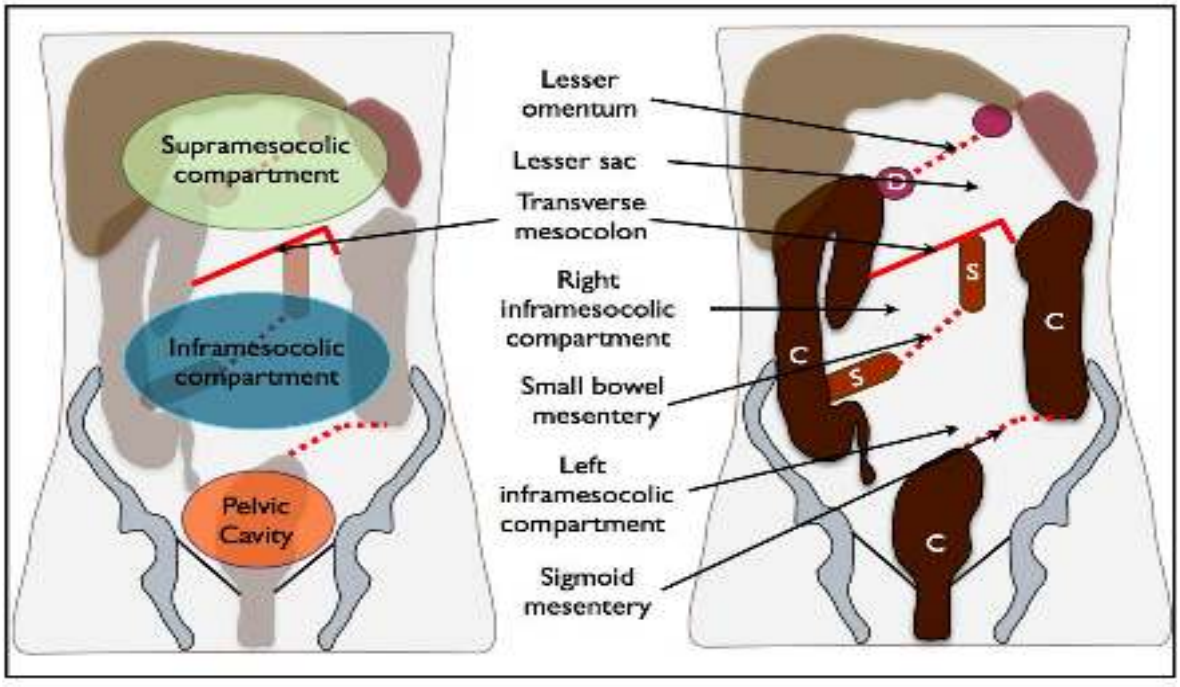


FIGURE 5: ANATOMY OF PERITONEUM AND MESENTERY

- **Division into 3 compartments**



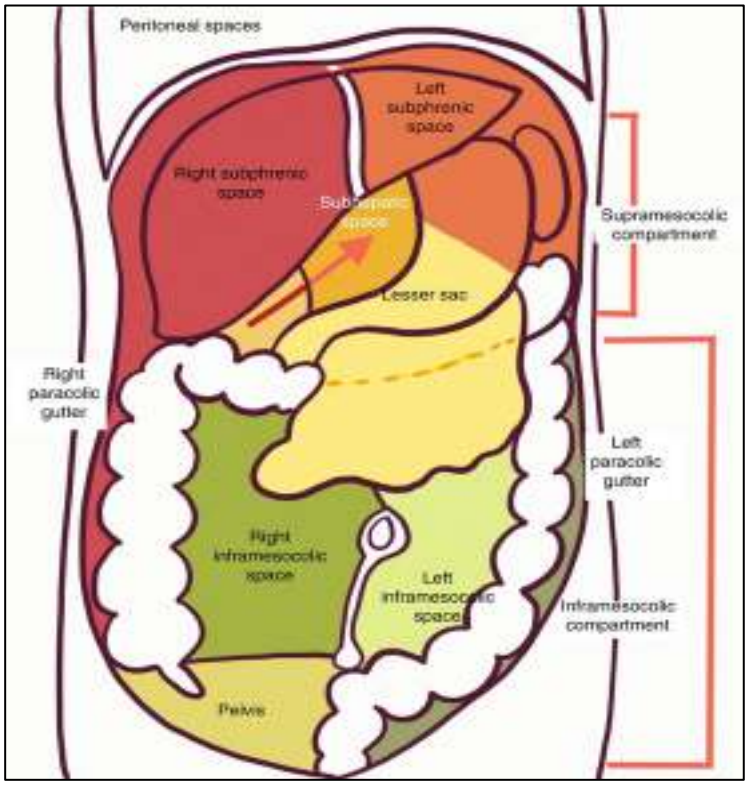


FIGURE 6: MORPHOLOGY OF PERITONEAL CAVITY

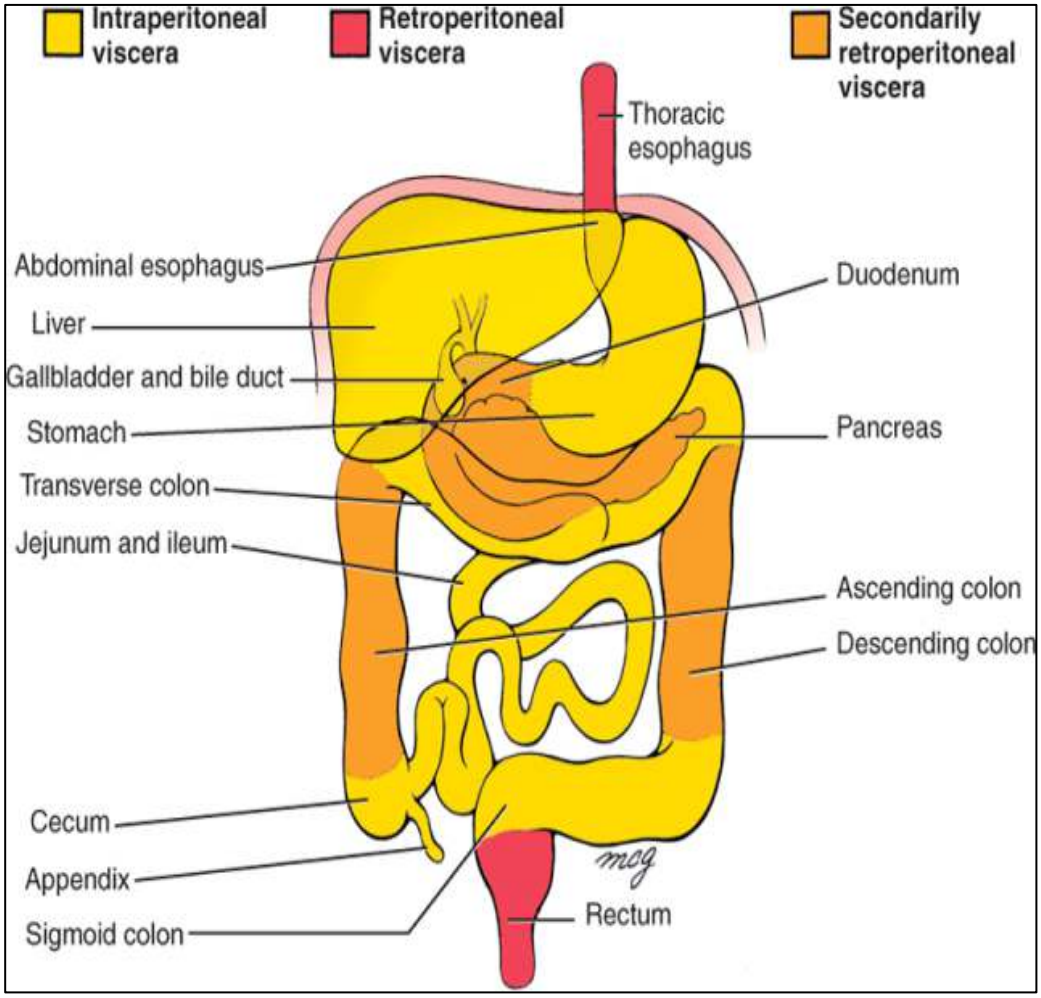


FIGURE 7: IMAGE SHOWING INTRA PERITONEAL VISCERA

- **NERVE SUPPLY** (40)
- parietal peritoneum – somatic afferents – well localised pain.
- Visceral peritoneum – autonomic nerves – poorly localised.

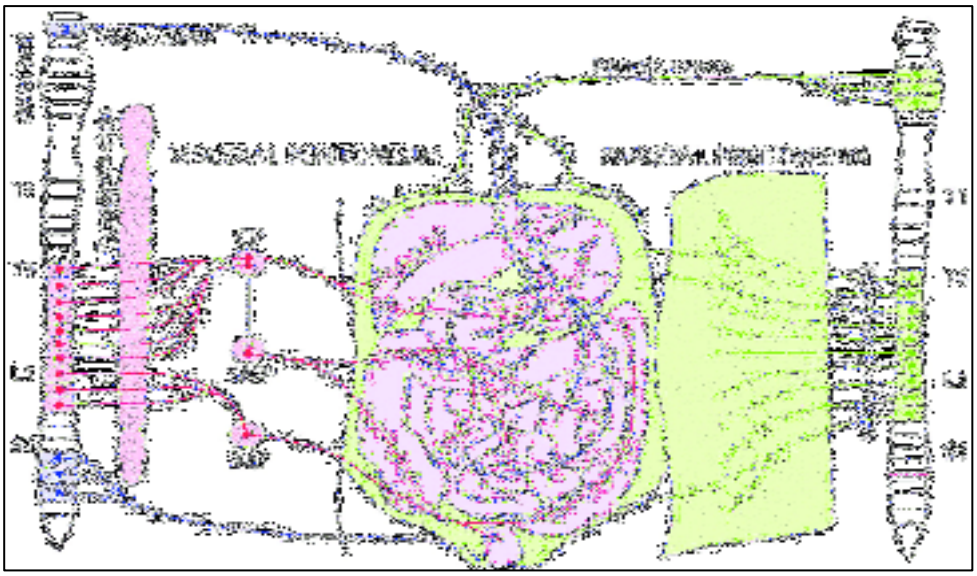


FIGURE 8: NERVE SUPPLY OF THE PERITONEUM

- **In cases of infection** - Diaphragm lymphatics have a significant role in the elimination of particulate materials, cells, and microbes.(39)

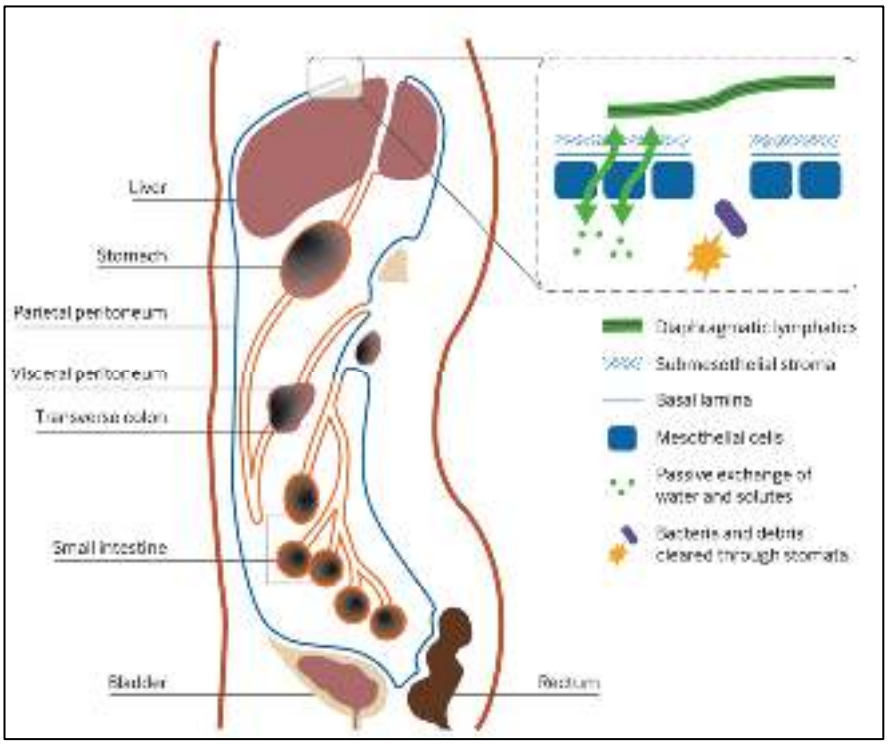


FIGURE 9: PERITONEAL AND DIAPHRAGMATIC LYMPHATIC DRAINAGE (39)

- 2nd mode of clearance – phagocytosis by indwelling macrophages.(37)

PERITONITIS

Peritonitis is an inflammatory condition that affects the serous layer that borders the abdominal wall and cavity. (41)

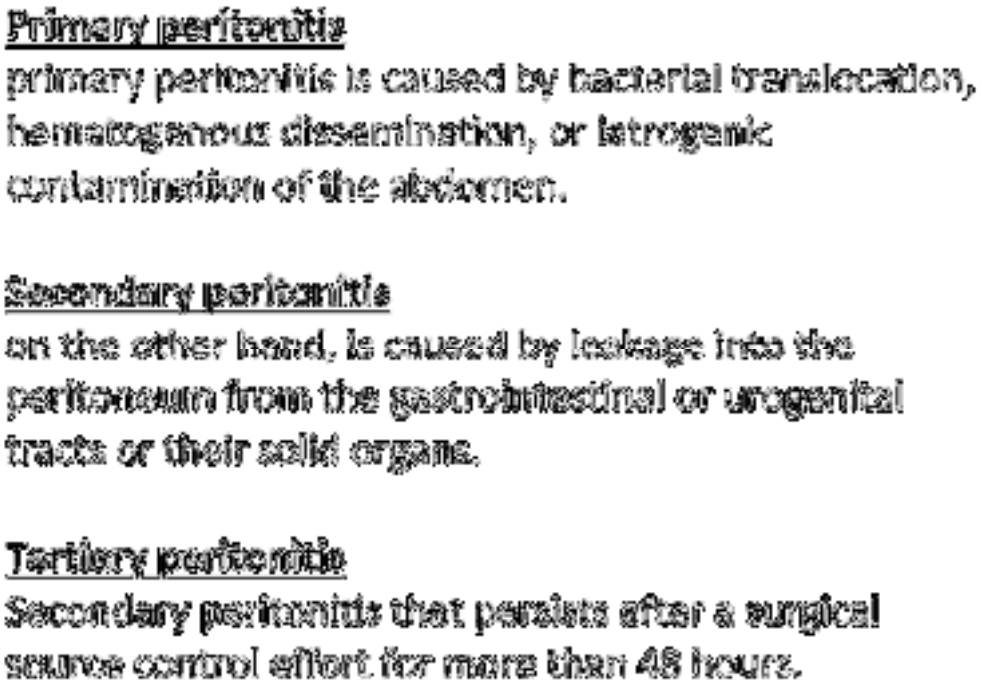
It might be the result of a local or generalized cause.

Types

1 Primary peritonitis

2 Secondary peritonitis

3 Tertiary peritonitis



Localised peritonitis (42)

supracolic and infracolic compartments are two separate compartments, the infection can be discovered in either and can be localised around the organ of insult. When the supracolic compartment overflows, the infection may spread to the infracolic compartment via the colon, or by the right paracolic gutter into the right iliac fossa and then into the pelvis.

Generalised peritonitis (43)

The following variables promote the spread of infection, resulting in widespread peritonitis:

1) The spillage of contents and infectious material such as in perforation.

2) Peristalsis stimulation by food/water intake and administration of enema

3) The causal organism's high virulence

4) Small omental surface in young children

5) Disruption of the locally collected data

6) Immunocompromised persons

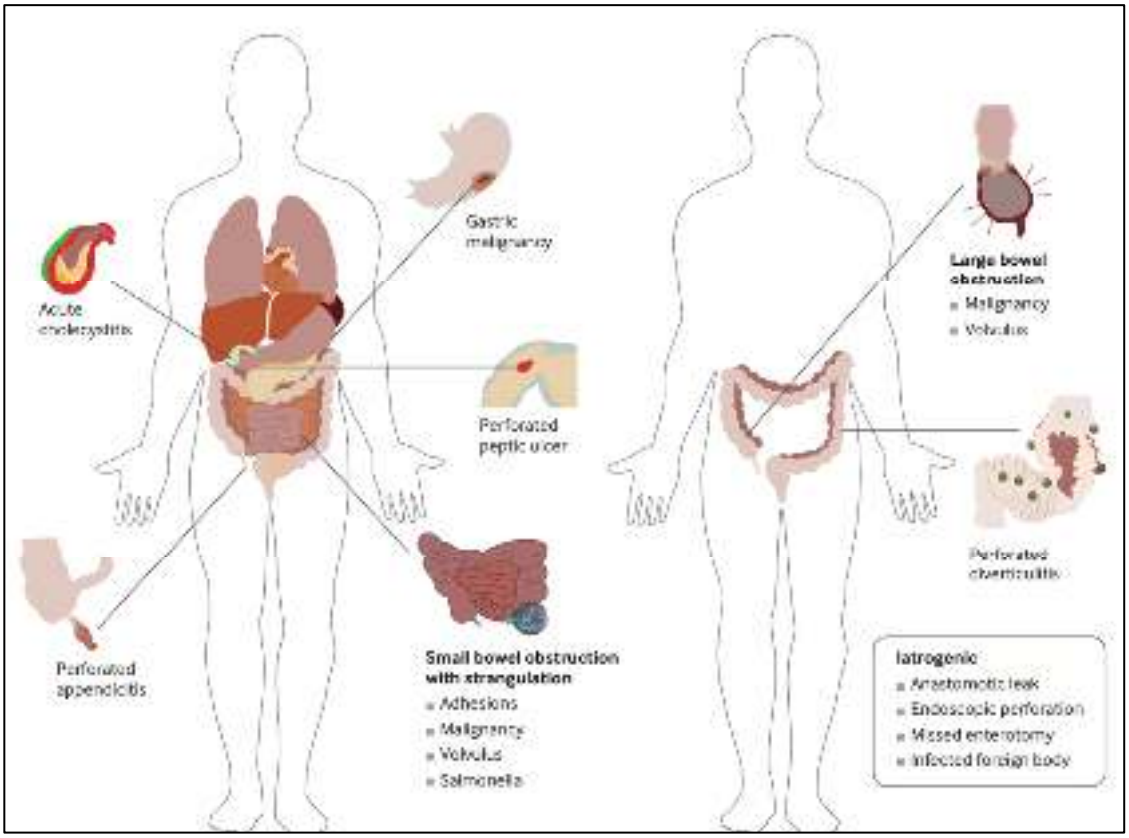


FIGURE 10: COMMON AETIOLOGIES OF SURGICAL PERITONITIS(39)

CLINICAL FEATURES OF PERITONITIS (39,41,44)

Peritonitis presents as acute abdomen usually.

Findings include

- Severe abdominal pain
- Distension of abdomen
- Guarding or rigidity
- Patient lies still
- Constipation/obstipation
- Diminished bowel sounds
- Fever
- Tachycardia
- Tachypnoea

Late signs include (45)

- lack of bowel sound
- stiffness
- the Hippocrates facies
- oliguria
- Confusion and disorientation
- and, eventually, shock.

BACTERIOLOGY OF PERITONITIS

The knowledge learned about the disease's bacterial origin has resulted in substantial advances in antimicrobial treatment. (46,47)

MIXED AEROBIC AND ANAEROBIC CONTAMINATION is what it is.

Organisms that are generally separated at different locations of bowel perforation are shown in the table below(48)

Site of perforation	Organism encountered
Oesophagus and Stomach	Gram-positive bacteria Candida
Small bowel	Enterobacteriaceae
Large bowel	Anaerobes Enterobacteriaceae
Appendix	E. coli Gram-negatives Anaerobes

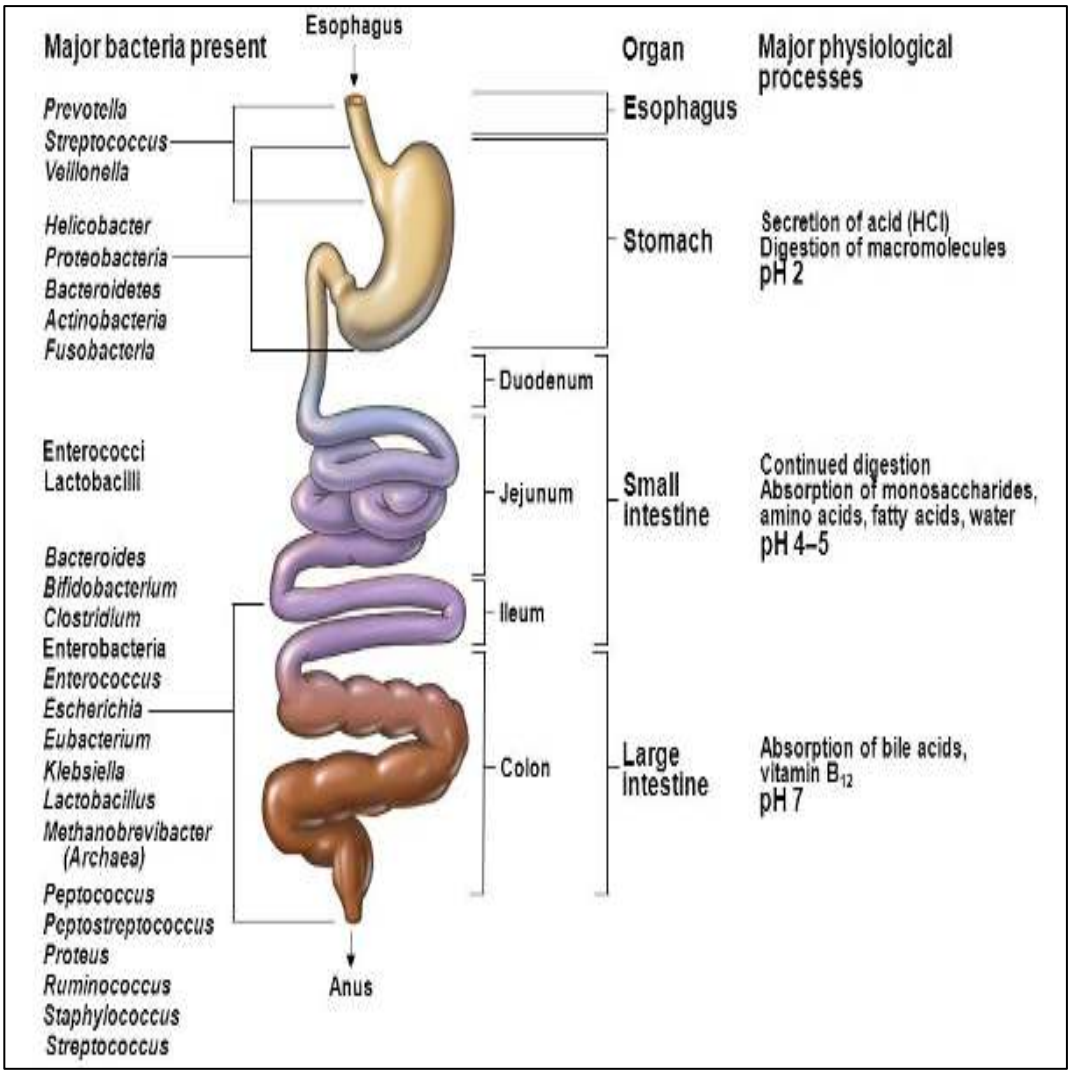


FIGURE 11: INTESTINAL MICROFLORA

Until the distal region of the small bowel, the natural bacterial flora of the gut lumen is often minimal. Proximal colonisations grow due to a variety of factors, including obstruction and acute or chronic motility problems. The biliopancreatic system is normally sterile as well; however, it can get contaminated in instances such as cholelithiasis. A mixed strain of bacterial colonisation frequently causes peritoneal infection.(48)

The organisms seen in peritonitis are usually the flora that is normally seen at the site of perforation, unless the patient has presented late, there has been prior antibiotic therapy, and, superinfection has occurred(49)

In the context of perforative peritonitis, the normal bacterial flora is disrupted as a result of the rupture of the hollow viscus, organism make their way into the peritoneum. The development of extremely aggressive pathogens has resulted from changes in typical bacterial flora in immunocompromised and hospitalised individuals.

Common microbiology in peritonitis(49)	
Gram negative	E. coli (Most common, seen in up to 65% cases) Enterobacteriaceae Klebsiella Pseudomonas Proteus
Gram positive	Streptococcus Enterococcus Staphylococcus
Anaerobes	Bacteroides species (Most commonly seen in up to 80% of cases) Clostridia (Seen in gallbladder, or rarely, colonic perforations)
Fungi	Candida (approx. 15% cases)

RESPONSE TO PERITONITIS

The body first responds to peritonitis with a local reaction that includes vasodilation, increased microcirculatory flow, and local tissue oedema, which leads to the subsequent phase of phagocytic cell entrance into inflamed peritoneal tissues.(50) Another way for microbial removal from the peritoneal cavity is the clearing of germs through the lymphatic system. Thus, phagocytes or the lymphatic system remove pathogens from the peritoneal fluid.(51)

When these two methods fail to cope with the microbial density, it resorts to the last-ditch mechanism, which is to loculate, or seal off, the instigating pathogen. The inflammatory process causes a fibrin build up surrounding the dense collection of bacteria, which culminates in the creation of an abscess. As a result of the abscess development, the microorganisms are confined within the abdominal cavity. However, with time, the abscess cavity becomes a source of bacteria that may enter the systemic circulation.(52)

Even though the microorganisms are confined, the patient may experience systemic consequences from the abscess.such collections are generally located in pelvis , paracolic gutter and sub diaphragmatic space.(50)

Thus, while the body's inflammatory response may be efficient in confining the bacteria within the abscess pocket, SIRS and MODS usually follow.(52)

The systemic reaction is mostly apparent as hypovolemia, which is caused by fluid build-up in the peritoneal cavity. As a result of the shift in intravascular volume, venous return and cardiac output are reduced as a result of factors such as of platelet activating factor, nitric oxide, TNF, IL-1.(53)

There is also a decrease in urine production, which may be explained by lower cardiac output, vasculature shunting, and distributive shock with symptoms like increased heart rate, rise in body temperature, low urine output and a fall in blood pressure.(54)

There is also atelectasis of the lower lung fields and limitation in diaphragmatic mobility due to peritoneal fluid collection and abdominal distension precipitated by it. As a result, hyperventilation comes into play, resulting in respiratory alkalosis(51)

ANTIBIOTIC SELECTION

Peritonitis has a biphasic reaction that impacts the outcome of the illness, according to experimental research. The initial step of this reaction is clearly purulent peritonitis, with E. coli (acting through endotoxins) as the main pathogen, which can also be identified in the bloodstream. If this bacillus is not addressed, this stage has a very high death rate (almost 40%). As a result, the notion of antibiotic treatment to combat this bacterium and other comparable infections has gained traction.(55)

The second phase begins around the fifth day after perforation and is mostly caused by the presence of Bacillus fragilis. This is the stage linked with the production of many intra-abdominal abscesses and chronicity as a result of this organism. As a result, targeting E. coli (to minimise mortality) and the anaerobic B. fragilis (to avoid the formation of intra-abdominal abscesses) is critical.(49)

When a patient arrives at the hospital, antibiotics are usually started empirically with the goal of targeting the most commonly encountered organisms in one's set up, as well as based on other factors such as the timing of presentation, severity of disease, patient condition, cost of antibiotic therapy, and the patient's age.

The patient's financial situation, accessibility, and side effects are all factors to consider.(51)

There are several medication regimens available, all of which are equally effective: (49)

- The traditional triple regimen of ampicillin, metronidazole, and gentamicin has largely been rendered obsolete.
- A two-drug regimen, such as a third or fourth generation cephalosporin and an anti-anaerobe, is typical therapy.
- Because they target both aerobes and anaerobes, single medicines (such as Clindamycin or Imipenem) are equally effective.
- The addition of an aminoglycoside has no effect on the result and may worsen the renal failure that is typical in such individuals.

INVESTIGATIONS IN A SUSPECTED PERITONITIS PATIENT

Combination of the clinical state with the radiological findings should be done before deciding whether to proceed with surgery or conservative therapy. A wide range of disorders can cause an acute abdomen similar to peritonitis, although none of them need surgery. A combination of clinical acumen (and knowledge) and radiological evidence aids in making the accurate diagnosis and therapy decisions for such individuals.(56)

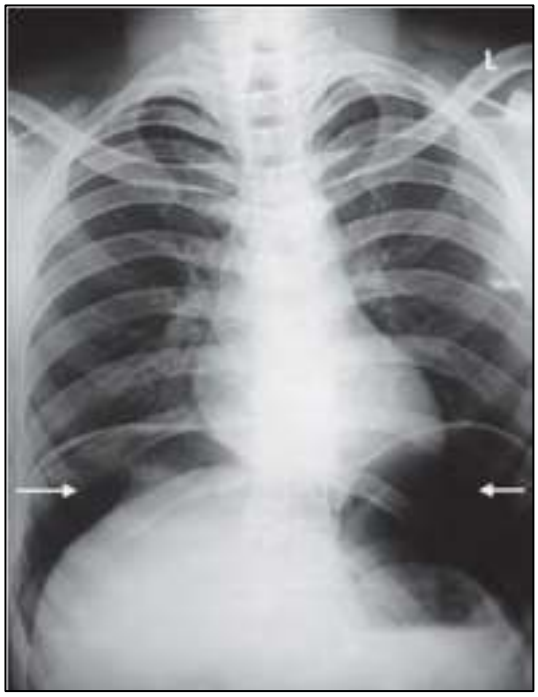


FIGURE 12: CLASSICAL "GAS UNDER DIAPHRAGM" ON XRAY

This characteristic X-ray has various subtle changes, all of which imply perforation of a hollow viscus in the relevant clinical situation.(57)

It is crucial to note, however, that situations other than perforation can also provide an image of gas under the diaphragm, such as: (58)

• Recently undergone abdominal surgery (open or laparoscopic).

• Attempts to aspirate the abdomen or FNAC an intra-abdominal mass in the past.

• Dialysis patients on peritoneal dialysis.

• Manipulation of the female reproductive system, as observed in hysterosalpingography, strong vaginal douching, or, occasionally, following sexual activity.

• Air may trickle down from a pneumothorax to provide free air beneath the diaphragm domes at times.

• Excessive weeping in babies and children can cause dilated bowel loops, which might be misinterpreted for free air behind the diaphragm.(59)

- **Chilaiditi's syndrome** - in this condition, a loop of colon arises between the superior surface of the liver and the right dome of the diaphragm, and on X-ray, it may be confused with air underneath the right diaphragm.(60)

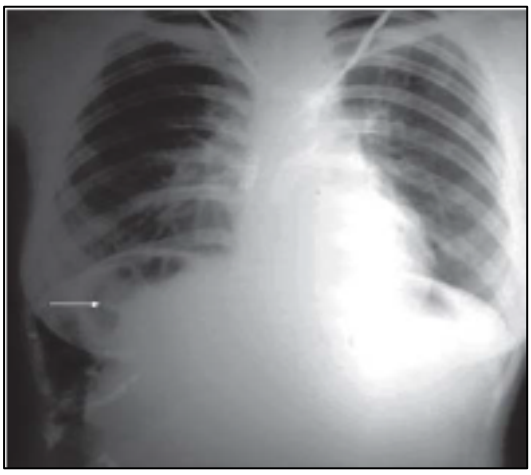


FIGURE 13: XRAY SHOWING CHILAIDITI'S SYNDROME

- Repeating the x-ray in a left lateral decubitus posture or after injecting roughly 200 to 300 ml of air through the Ryle's tube (and clamping it) may occasionally reveal the presence of free air.
- In situations where the question continues, an ultrasound-guided aspiration of the peritoneal fluid or a bedside aspiration of the peritoneal fluid may aid in making

an accurate diagnosis by demonstrating the type of the contents (bile, faecal matter) or by investigation under a microscope for vegetable fibres.(45)

- Despite several publications on the sensitivity and specificity of CT in acute abdominal diseases, a **CT scan is frequently not required to make the diagnosis of peritonitis!** However, radiographic examinations to test for free intraperitoneal air should be performed prior to tapping the abdomen, since this may result in free air within the peritoneal cavity, committing such a patient to a laparotomy.(39)
- The patient may have the conventional clinical signs of peritonitis, yet there is no gas on X-ray. Other options that must be considered in this case are: (39)
    - Perforation of the appendix
    - Acute pancreatitis
    - Vascular disease of the mesentery
    - Ectopic pregnancy rupture
    - Ruptured liver abscess
    - Acute myocardial infarction
    - Pneumonitis of the right lower lobe.
- The 'Double Wall' sign is another name for the Rigler's sign. Normally, gas only outlines the luminal (mucosal) part of the intestine, but the presence of free intraperitoneal air allows for good visibility of both sides of the intestinal wall on a plain X-ray. This is the second most commonly seen radiological sign.(61)

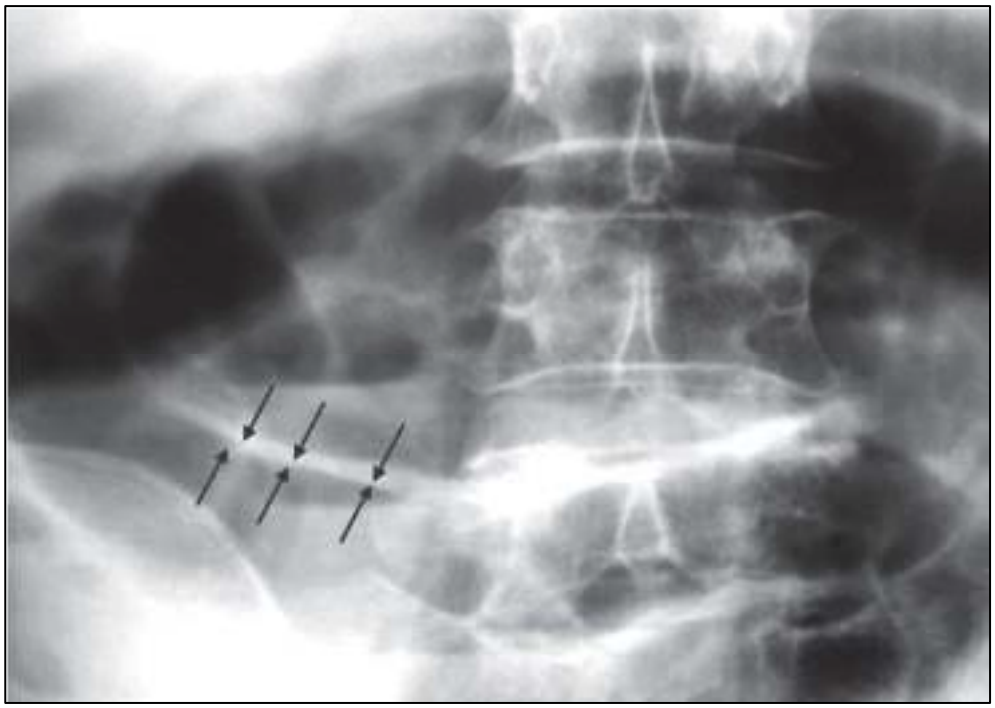


FIGURE 14: XRAY SHOWING RIGLER'S SIGN

- A **CT scan** may be required to confirm the diagnosis at times, although it is not the first study to be explored in such circumstances. It does, however, have the benefit of clearer delineation of fluid and air within the peritoneal cavity, and it may even detect dye leaking from a torn viscus or any other accompanying disease.

OPERATIVE MANAGEMENT OF PERITONITIS PATIENTS

Once a clinical diagnosis of peritonitis is obtained, it is critical to institute both physiologic support and intensive anti-infective treatment as soon as possible.

In the event that When in doubt, early surgical surgery is preferable to waiting and seeing policy.(51)

The primary goals of peritonitis treatment are:

1. Intravenous access and fluid replenishment

2. initiate antimicrobial treatment

3. Source control

4. Organism load reduction.

5. Metabolic support (62)

• Intravenous access and fluid replenishment: In no instance should a venesection be performed in haste; an intravenous line can be started with a wide diameter peripheral cannula. – There isn't much of a difference between the results of colloids and crystalloids.(63)

• Inhalation of oxygen using a mask or nasal cannula, as these patients are frequently acidotic and hypoxic.

• Maintain a urine production of at least 0.5 to 1 ml/kg/hour.

• Begin antimicrobial treatment

• Insert a nasogastric tube

• Routine investigations should be sent to the laboratory

- Prepare and encourage the patient to undergo surgery as soon as possible. Spend as little time as possible resuscitating the patient and 'preparing' for surgery, and aim to get the patient into the operating room as soon as possible. (54)
- Remember that if too much time is spent on ‘resuscitating,' the patient's condition will worsen until the perforation is addressed and the persistent infection is managed;
- In such a case with expected delay, introducing an abdominal drain under local anaesthetic may be a prudent alternative because:

  – It would enable the purulent peritoneal fluid to evacuate, lowering the infective component within. Furthermore, intra-abdominal pressure and diaphragmatic tenting would be alleviated, which would enhance lung function and hypoxia. However, don't feel content after inserting that abdominal drain

—remember that this is only a temporary solution, not a permanent cure. The patient must still be operated on as soon as feasible.

- **Source control** can be accomplished by: (63)

  -Organ removal (e.g., appendectomy, malignancy-related perforations).

  - Perforation repair (e.g., simple closure, omental patching of duodenal ulcer perforations).

  - Bringing the organ out of the abdominal cavity for a brief period of time, as seen in ileostomy or colostomy when the perforation can be brought out as a stoma. (45)

  - The damaged organ is bypassed.

  - Multiple operations, such as perforation repair and proximal bypass or stoma creation.

- Another critical factor is minimising contamination within the peritoneal cavity. This is often done by:

  - Lavage intraoperatively

  - Leaving an abdominal drain in place

  - At the time of operation, the diseased parts of the abdomen are debrided.(64)

- Peritoneal cavity drainage (63)

  - Although very much in favour, it is vital to recognise that draining the whole peritoneal cavity is difficult, as drains tend to get plugged by adhesion development and cease to drain after only a couple of days.

  - Keep in mind that installing a drain does not guarantee success. that the anastomosis is not going to leak

- A 'non-draining' drain, on the other hand, may offer you a false feeling of security and actually mislead you from the reality that the repair has failed or that an abscess is building within the 'tummy.'(64)

- After the source has been managed and the contamination has been addressed, the final surgical technique is to avoid recurring infection within the peritoneal cavity. A laparostomy, in which the abdomen is left open so that it may be viewed and cleaned regularly, is a superior technique for preventing recurring infections. - A planned relaparotomy is another option for detecting and treating any persistent or recurring infections. - Recognizing the risk of abdominal compartment syndrome in specific settings and taking precautions to avoid it is also critical.
- Another critical (and frequently overlooked) issue in our nation is the maintenance of nutrition in these individuals. Patients are frequently malnourished to begin with, and the catabolism of peritonitis (and subsequent surgery) exacerbates this. Add to that the fact that total parenteral nutrition is prohibitively expensive and not available through the hospital, and you have a patient who deteriorates over time due to a lack of proper nutritional support, particularly when there is a prolonged period of fasting, the need for repeated surgery, or when there are fistulae or infections.(63)
- As a result, the threshold for conducting a feeding jejunostomy should be very low, especially in perforations/disruptions above the jejunum, so that nourishment may be quickly supplied to the patient if the need arises.

SSI IN CASE OF PERITONITIS

The incidence of Surgical Site Infection grows as wound contamination rises. According to the CDC, surgical wounds in peritonitis are classed as Contaminated (Category 3) or Dirty (Category 4) wounds.

In sepsis or peritonitis, the oedematous gut induces fluid extravasation into the abdominal cavity, which, if not thoroughly drained during surgery, might track into the subcutaneous area of the surgical incision. This causes bacteria to colonise the wound site, which affects wound healing in the following ways: (65)

1. Microorganisms that cause tissue hypoxia
2. Nutrient deficiency
3. Proteolysis produced by microorganism-released enzymes
4. granulation tissue and cellular multiplication is hampered.

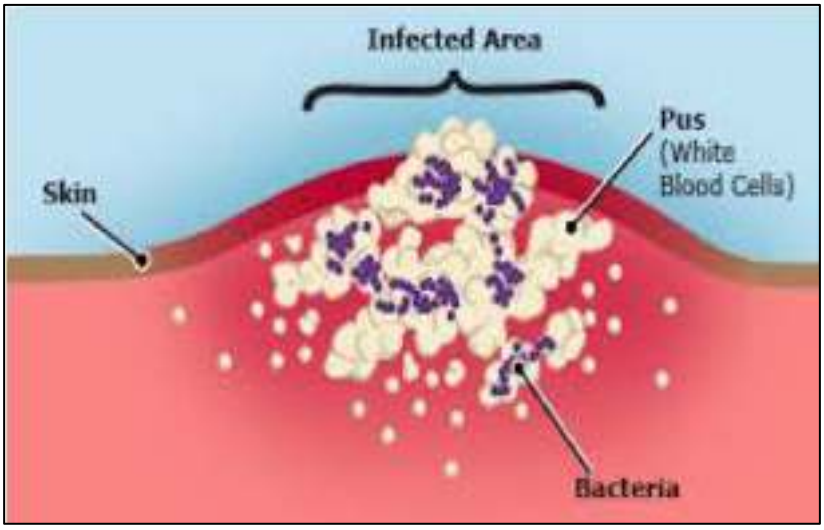


FIGURE 15: BACTERIAL COLONISATION IN SSI

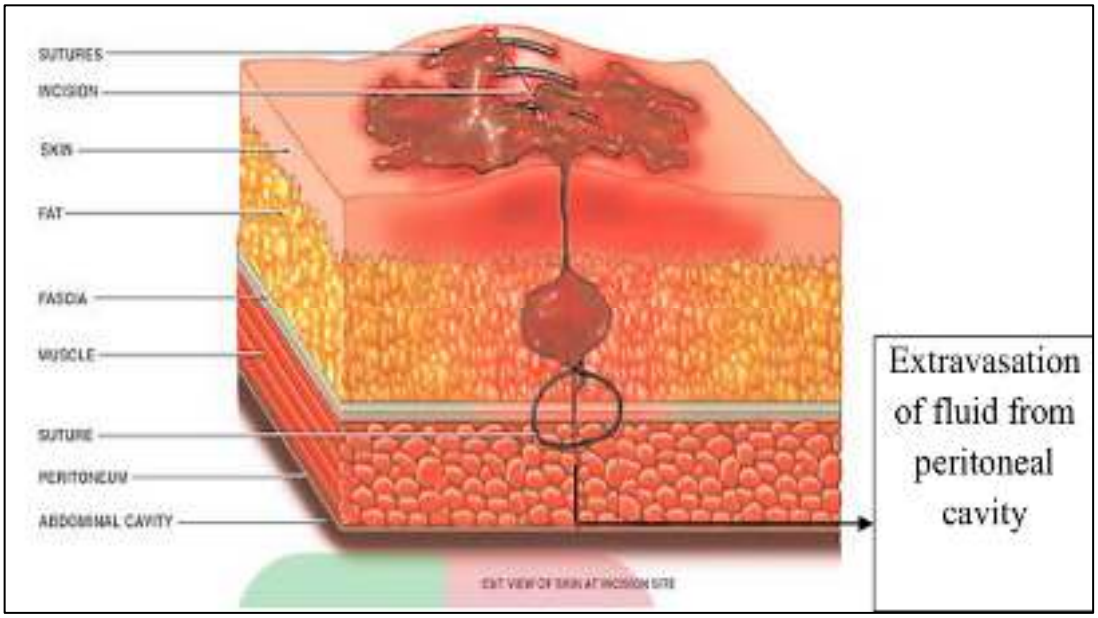


FIGURE 16: MECHANISM OF SURGICAL SITE INFECTION IN CASE OF PERITONITIS

Antibiotic sensitivity testing (66)

It is a laboratory test used to assess the effectiveness of antibiotic therapy against bacterial infections.

Antibiotic sensitivity testing will help to control antibiotic use in clinical settings.

The testing will help doctors choose which drugs to use to treat infections.

Uses

1 Pathogen identification in exudates and body fluids.

2. Sensitivity tests are performed to evaluate the degree of sensitivity or resistance of microorganisms isolated from the patient to a suitable spectrum of antimicrobial medicines.

3. Measurement of the concentration of a medicine provided in a blood or bodily fluids in order to manage the dosing schedule.

Kirby-Bauer method with disc diffusion

Antibiotic-impregnated chips are used.

After swabbing the bacteria on agar, antibiotic discs are placed on top. As one goes farther from the disc, the antibiotic diffuses into the agar in decreasing concentrations.

If the antibiotic concentration kills or inhibits the organism, there will be no growth in the immediate area around the disc: this is called inhibition zone.

Antibiotics resistance mechanism (67)

Access is being denied:

ntibiotics try to penetrate through the bacterial cell membrane, but the barrier becomes impermeable: Imipenem, for example.

Antibiotic alterations:

The antibiotic is then changed in the second phase with the aid of a bacterial enzyme. Penicillin, becomes inactive by beta lactamase.

Changed target site:

The antibiotic is unable to attach to its intended target because the target has been altered. The antibiotic is being pumped out quicker than it is being absorbed: Tetracyclines, for example.

Alternative target (usually an enzyme): (PBP2a) in MRSA

MATERIALS AND METHODS

This study was done in the Department of General Surgery, in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2020 to December 2020 on 75 patients who underwent surgery in cases of peritonitis.

1 Data source: patients getting admitted and getting operated for peritonitis under surgery department in KLES Dr. Prabhakar Kore hospital and MRC, Belgaum.

2 method of data collection:

INCLUSION CRITERIA:

Only those who have undergone laparotomy surgeries for peritonitis and admitted under surgery department in KLES Dr. Prabhakar Kore hospital and MRC, Belagavi.

EXCLUSION CRITERIA:

1. Paediatric age group <12 years
2. Patients having any previous infections at the surgical site.

ETHICAL CLEARANCE:

Clearance from the Ethical Committee of Jawaharlal Medical College was obtained before commencement of the study.

TYPE OF STUDY: It is a prospective observational study.

STUDY PERIOD: One-year hospital-based study i.e., 1st January 2020 to 1st December 2020.

STUDY DURATION: 1 Year.

SAMPLE SIZE:

The minimum sample size formula based on two proportions is

$n = 4pq/d2$

where p=Prevalence (from previous studies)

q=100-p

d=allowable error

Taking allowable error as 10% and from previous studies p=22.1%(68)

So, applying in formulae n=68.86, rounding off we get 70

So, sample size for this study is 70.

SAMPLING PROCEDURE:

All consecutive patients fulfilling the criteria during the period of study were selected as the sample of this study.

STATISTICAL ANALYSIS:

All data was obtained in terms of, mean ± standard deviation and analysis was obtained. Frequency of all variables was derived to check the completeness of data. Magnitude was expressed in percentages. The incidence rate of SSI was estimated, also with respect to various risk factors.

CONSENT: A waiver of consent was obtained for this study as it involves the review of medical records of patients who underwent laparotomy, and data was collected from the data already recorded.

METHODOLOGY:

The patients who were getting admitted under surgery department with complaints of peritonitis –

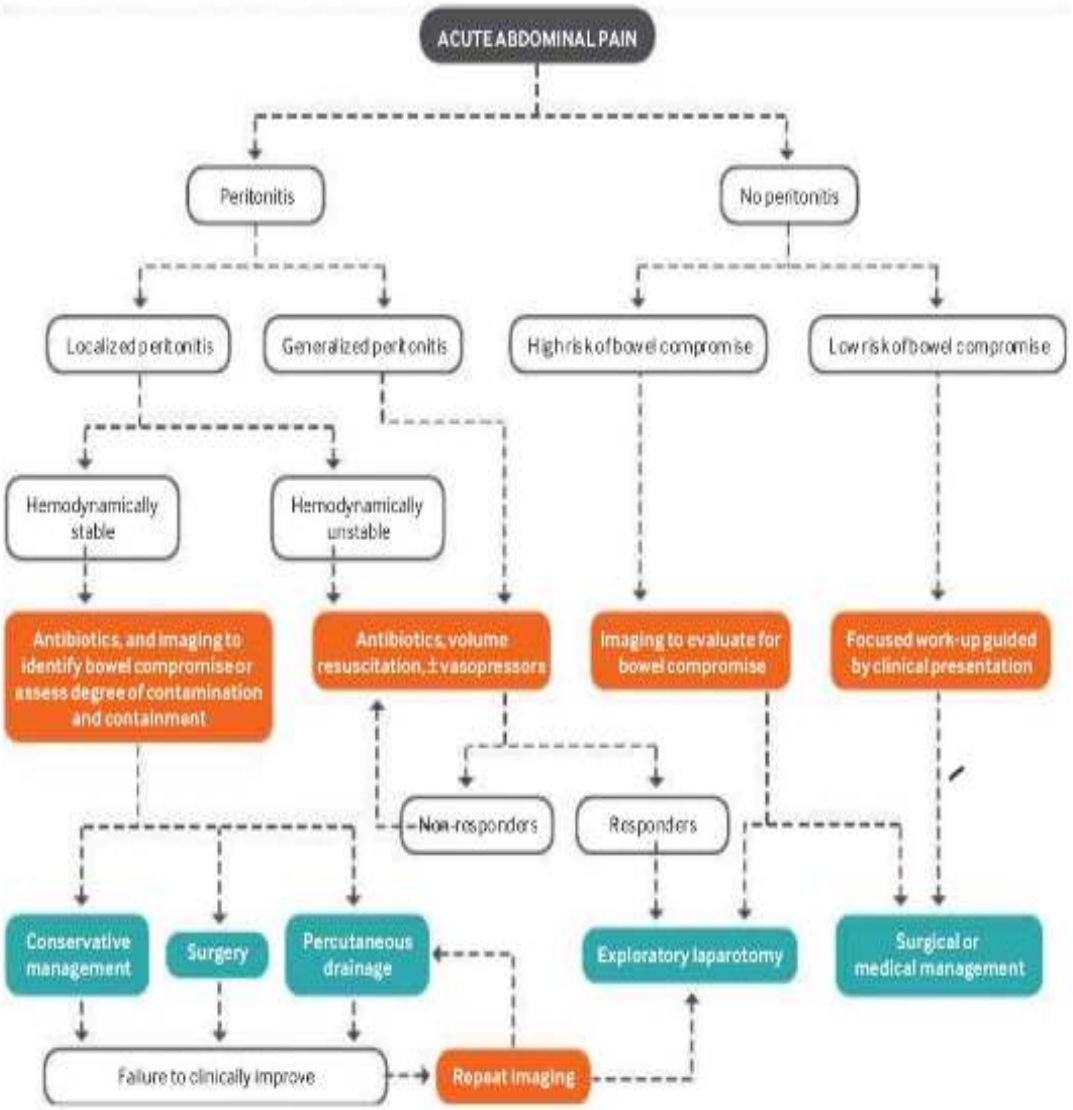
clinical features (history)

physical examination

were confirmed by x-ray erect abdomen or CT scan as appropriate.

Routine investigations which include haemoglobin, renal function test, liver function tests, electrolytes were sent for assessment along with viral markers (HIV 1 AND 2 AND HBsAg), ECG

Following algorithm was followed to access what to do with the patient.(39)



PER ABDOMINAL EXAMINATION: careful examination of abdomen to look for any tenderness, guarding, rigidity, diminished bowel sound etc.

IMAGING –

erect x-ray abdomen

lateral decubitus x-ray in unstable patient

Ultrasonography and CECT abdomen might also be performed for selective cases as per requirement.

After a decision was made to operate the patient following was done:

Pre-operatively

- resuscitation-iv fluids, Ryle's tube insertion, SRC insertion.
- informed and written consent was obtained for surgery
- parts preparation
- patients were kept NBM.
- investigations

Peri-operatively

- higher antibiotics were started.
- laparotomy was performed
- hand scrubbing was done by the operating surgeons and assisting surgeons
- cleaning and painting were done with 10% povidone iodine in all cases.
- Incision was decided as per the suspected organ of pathology.
- careful examination of the abdominal organs to find any pathology was done.
- Procedure to be performed was decided on table by the operating surgeon as per the pathology and viscus involved.
- adequate peritoneal lavage – was given in all cases using warm saline.

post-op

- Wound was covered with sterile dressing after cleaning wound with 10%povidone iodine.
- post-operative monitoring.
- recovery of the patients was observed

ASSESSMENT OF SURGICAL SITE INFECTION

Following their surgery, patients were monitored for signs of SSI. A positive SSI is considered if any one of the following is positive at the surgical site;

- Signs of inflammation (pain or tenderness, erythema, swelling, warmth)
- Pus discharge
- Positive culture of swab or fluid

Wound was checked for infection (SSI) on 3rd, 5th, 7th, 9^{th}, 12^{th} day postoperatively. Wounds were graded as per SOUTHAMPTON'S WOUND GRADING. If pus discharge was noted, pus was sent for culture and sensitivity assessment. All findings were duly recorded in case sheets.

FOLLOW-UP: Upon discharge, patients were requested to return for a follow-up visit at 30th post-operative day for re-examination of surgical site infection or in between whenever they have any complaints. Data was recorded in the OPD follow up cards of the patients.

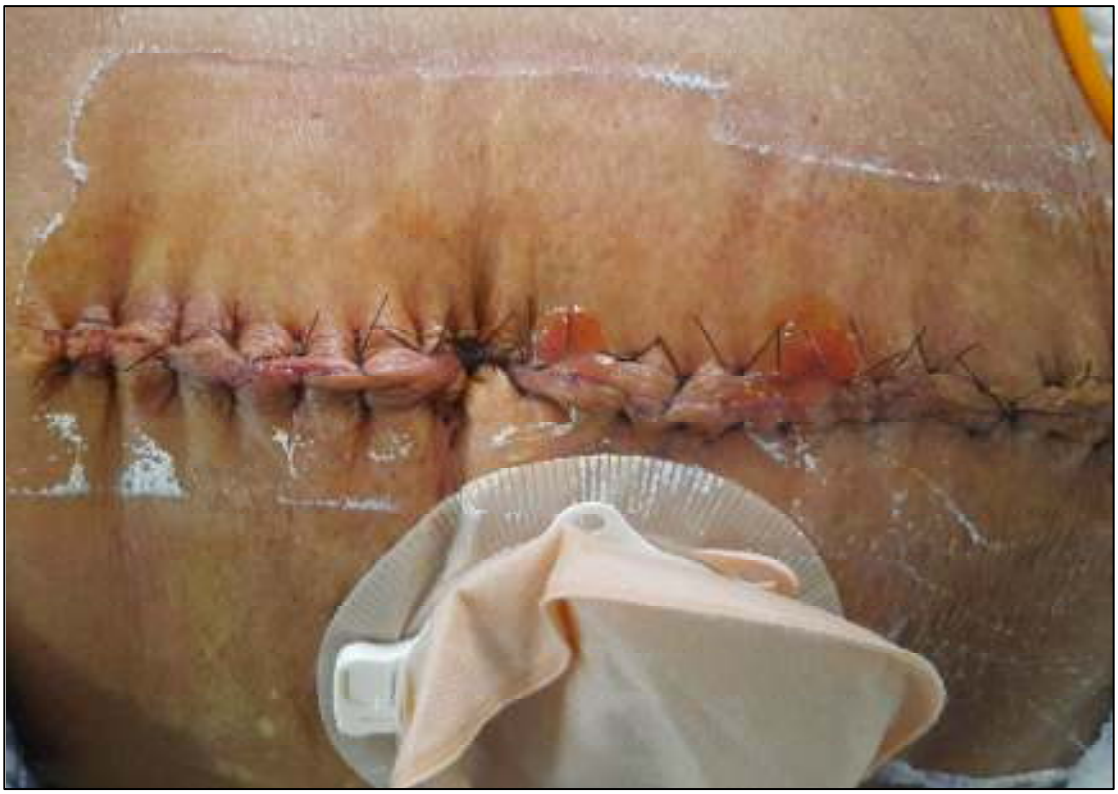


PHOTO 1: SHOWING INFECTED POST OPERATIVE WOUND WITH PUS DISCHARGE

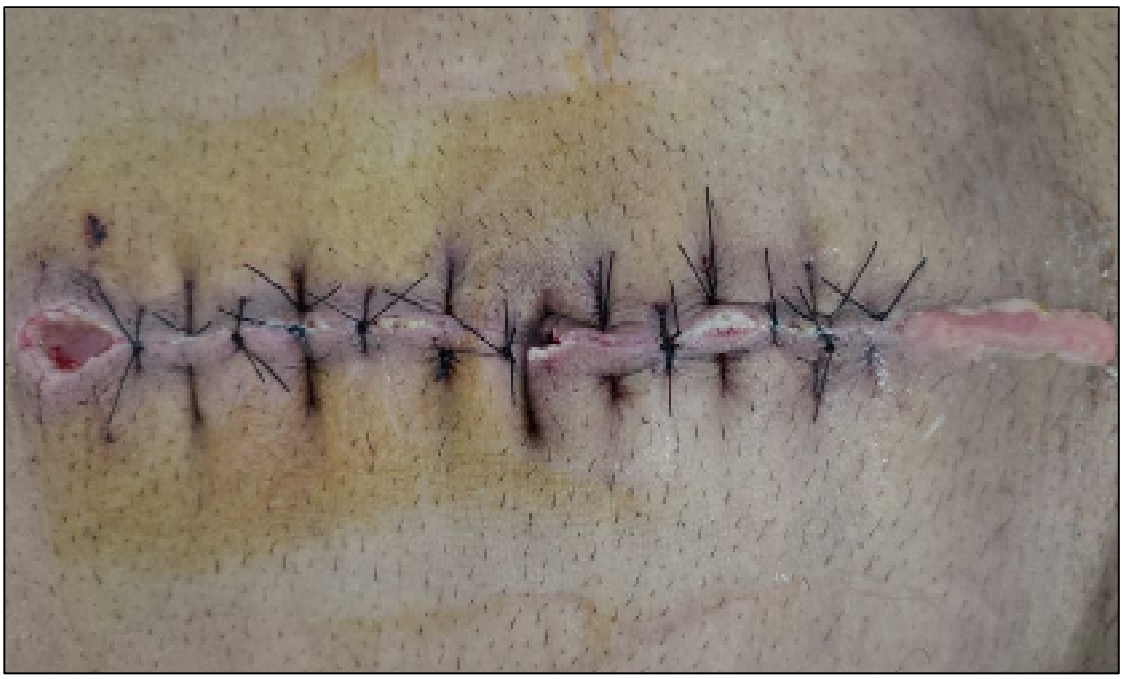


PHOTO 2: SHOWING ANOTHER POST OPERATIVE WOUND WITH PUS DISCHARGE

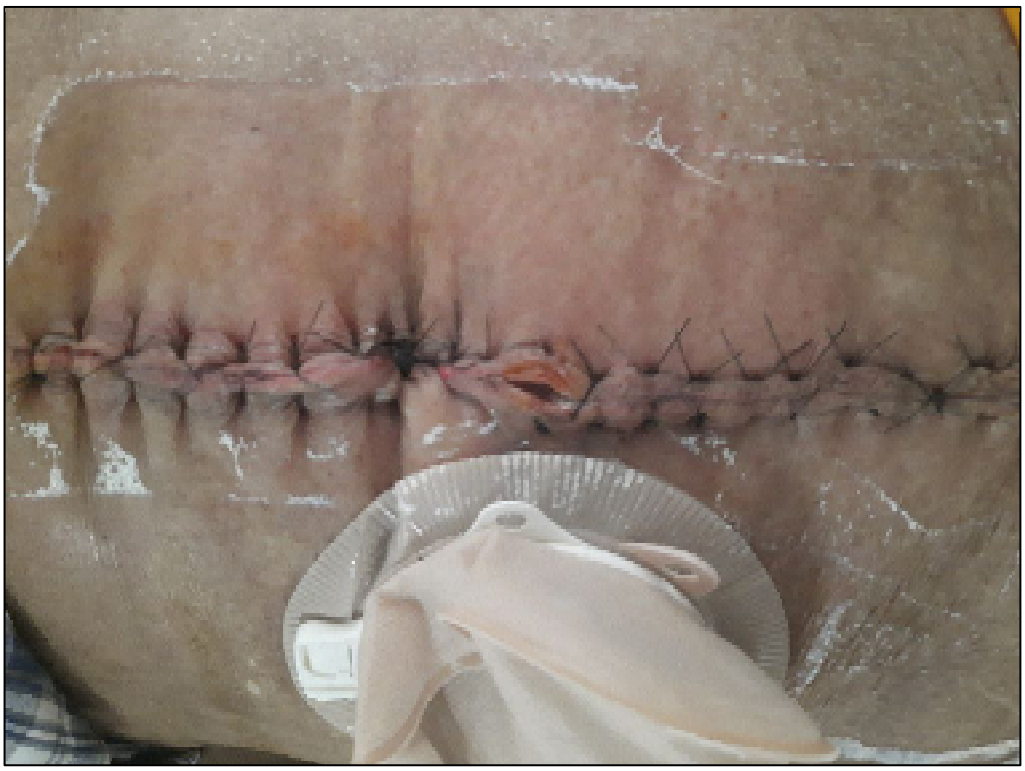


PHOTO 3: SHOWING WOUND AFTER SUTURE REMOVAL

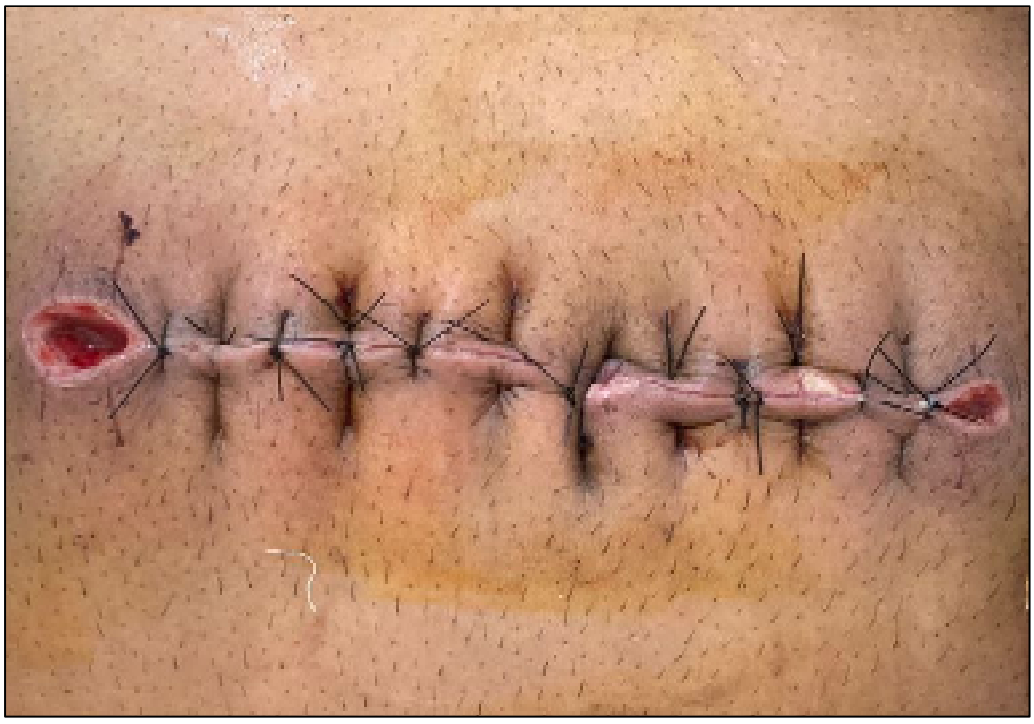


PHOTO 4: SHOWING WOUND IN HEALING PHASE

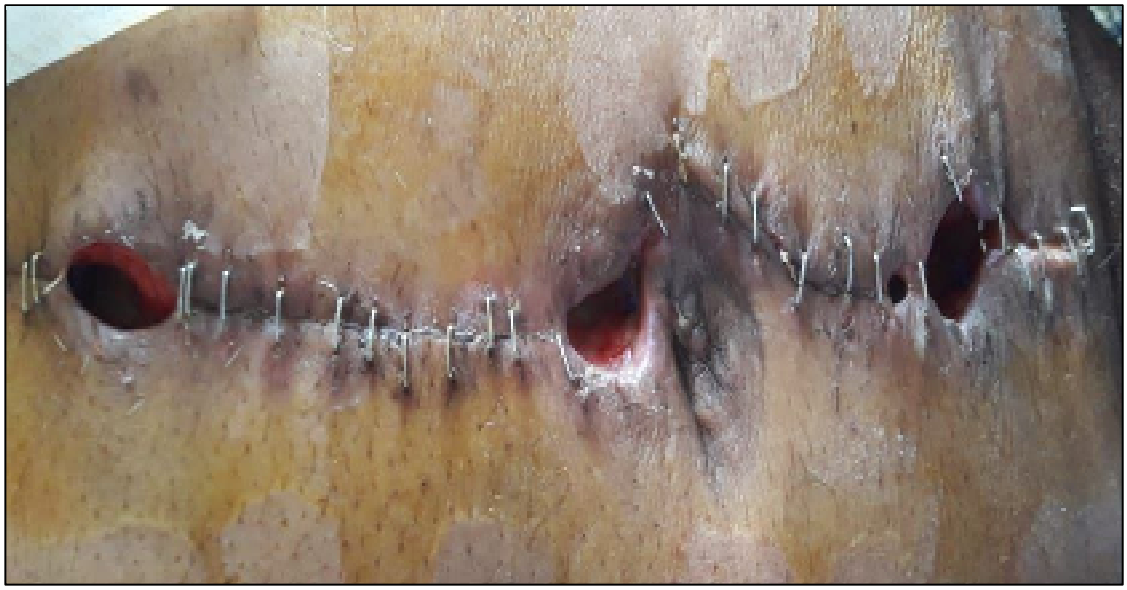


PHOTO 5: ANOTHER WOUND IN HEALING PHASE

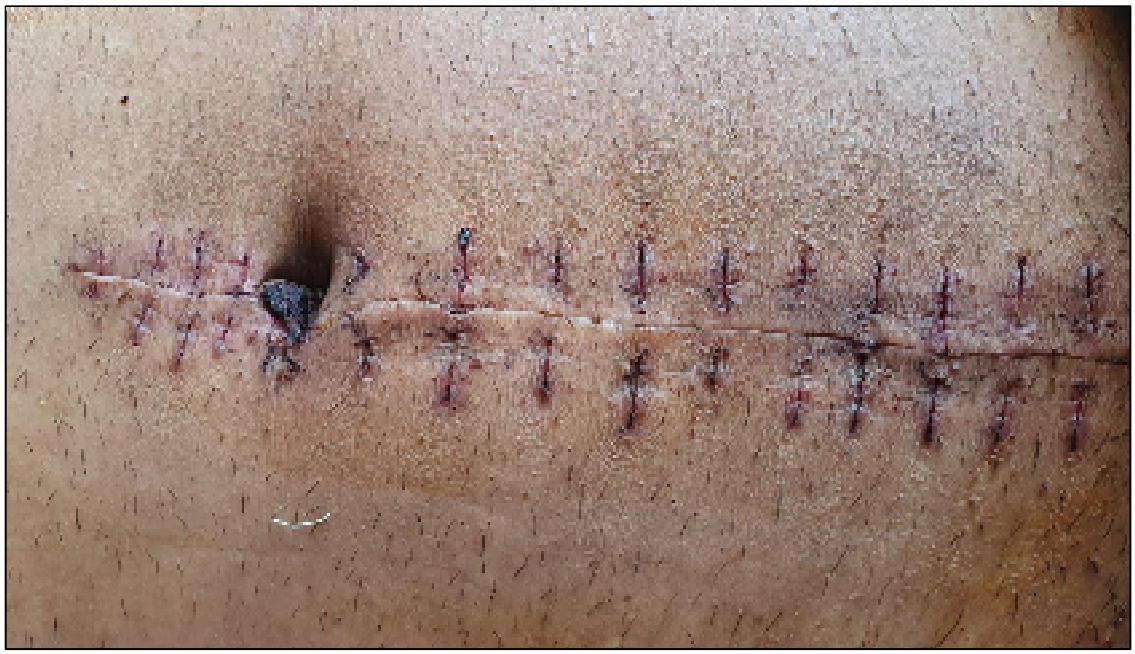


PHOTO 6: HEALED POST OPERATIVE WOUND

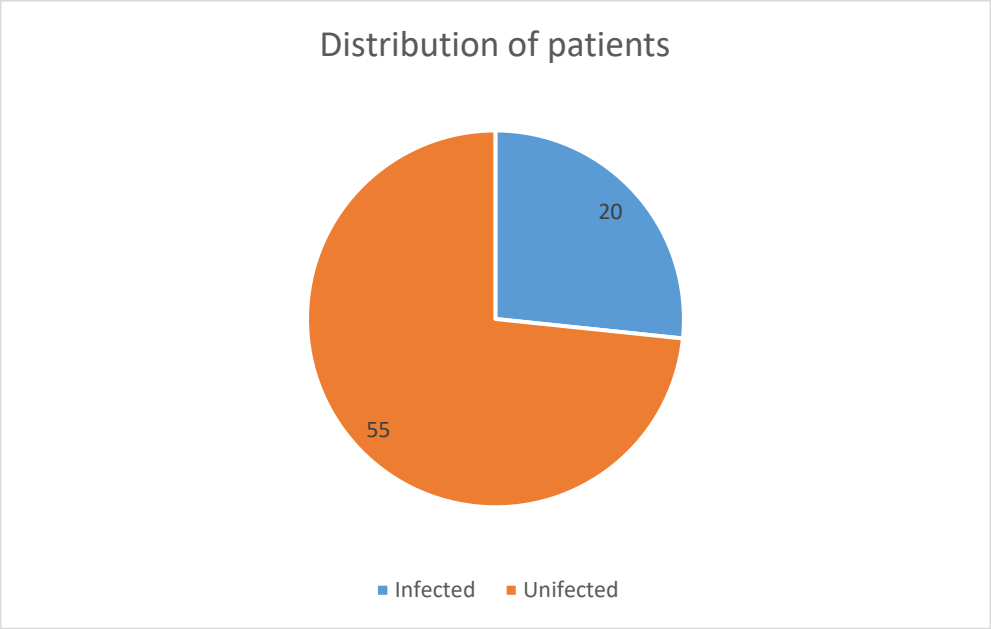
RESULTS

This study made an inclusion of 75 cases which were observed prospectively from January 2020 to march 2021.

TABLE 1: INCIDENCE-SSI IN PERITONITIS PATIENTS

TOTAL NO. OF CASES	NO. OF CASES INFECTED	PERCENTAGE
75	20	26%

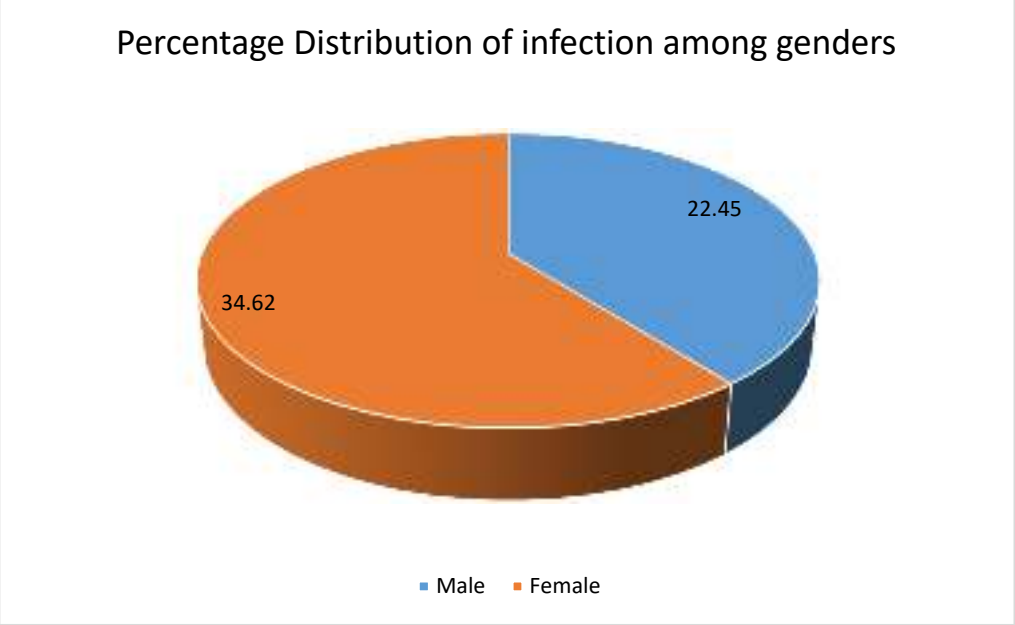
Out of the 75 patients, surgical site infection was noted in 20 patients. So, the incidence is 26%.



GRAPH 1: Pie chart showing distribution of patients having SSI vs no SSI.

TABLE 2 : INCIDENCE IN RELATION TO SEX

SEX	NO. OF CASES	INFECTED	%
MALE	49	11	22.45
FEMALE	26	9	34.62



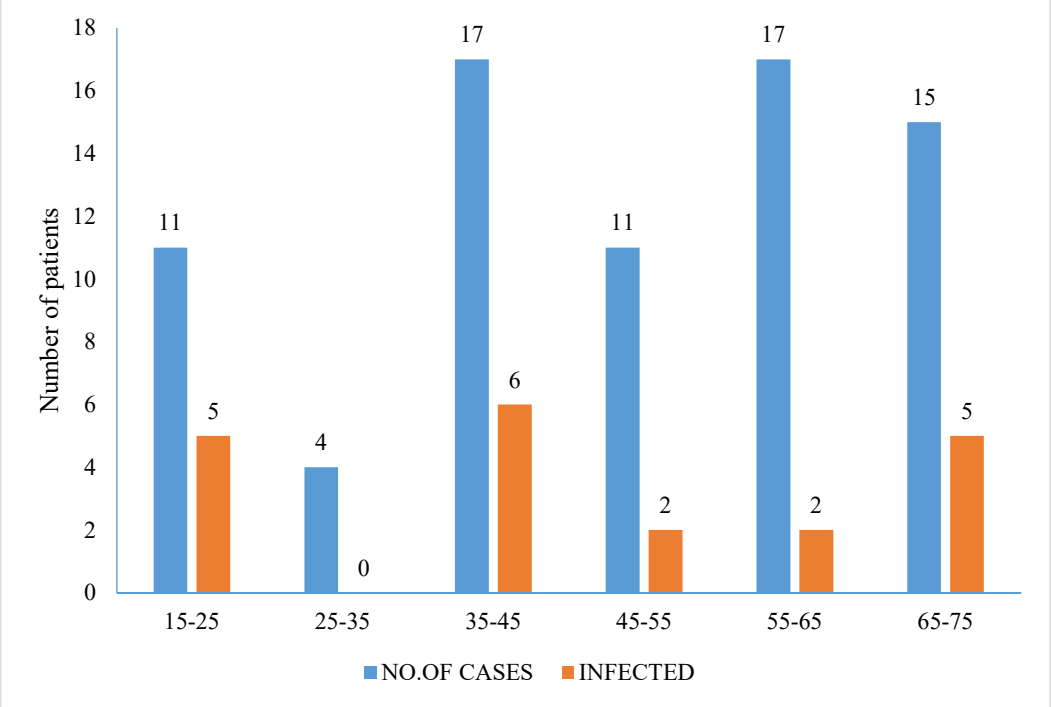
GRAPH 2: Pie chart showing gender distribution of SSI in study subjects.

Incidence of SSI in males is 22.45% whereas in female is 34.62% in our study.

TABLE 3: INCIDENCE IN RELATION TO AGE GROUP

AGE	NO.OF CASES	INFECTED	PERCENTAGE
15-25	11	5	45.45
25-35	4	0	0.00
35-45	17	6	35.29
45-55	11	2	18.18
55-65	17	2	11.76
65-75	15	5	33.33
TOTAL	75	20	

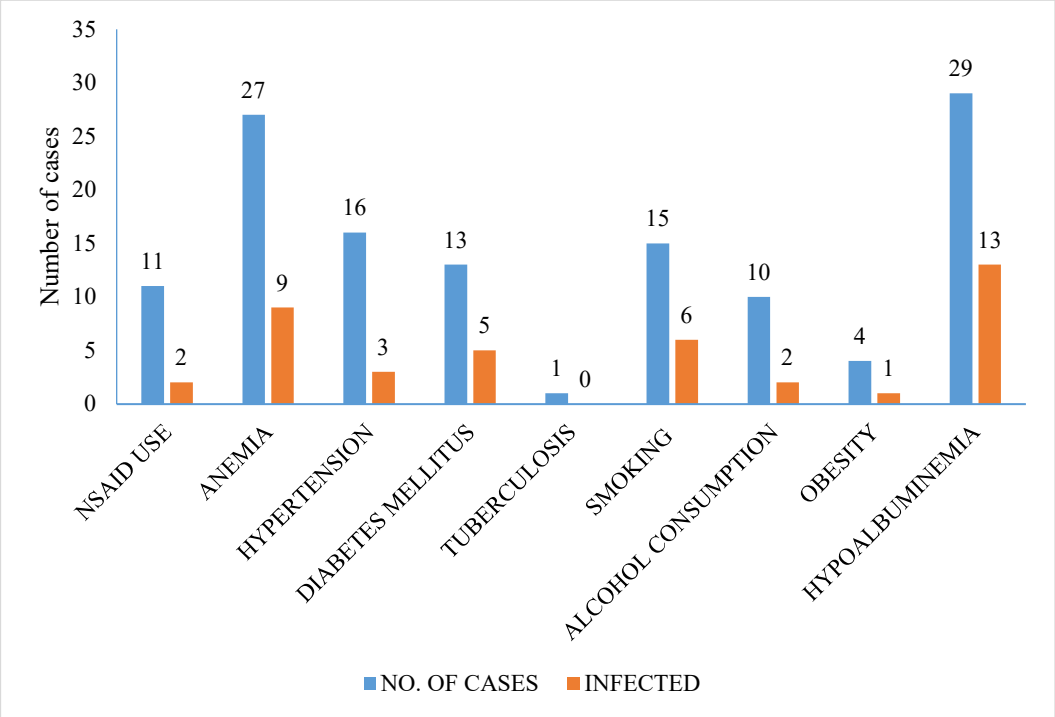
SSI incidence in the age group of 15-25 was 45.45%, followed by 35.29% in the 35-45-year age group and 33.33% in the older age group.



GRAPH 3: Bar diagram showing age distribution of SSI among study subjects.

TABLE 4 : INCIDENCE IN RELATION TO

	NO. OF CASES	INFECTED	%
NSAID USE	11	2	18.2%
ANEMIA	27	9	33.3%
HYPERTENSION	16	3	18.7%
DIABETES MELLITUS	13	5	38.5%
TUBERCULOSIS	1	0	0%
SMOKING	15	6	40%
ALCOHOL CONSUMPTION	10	2	20%
OBESITY	4	1	25%
HYPOALBUMINEMIA	29	13	44.8%



GRAPH 4: Bar diagram showing SSI incidence distribution in relation to NSAID use, anaemia, hypertension, diabetes, tuberculosis, smoking, alcohol intake, obesity and hypoalbuminemia.

Most of the patients had hypoalbuminemia (38.7%) with an SSI rate of 44.8%

Diabetic patients had an SSI incidence rate of 38.5%.

SSI in smokers turned out to be 40%.

Anaemia culminated to an SSI rate of 33.3%

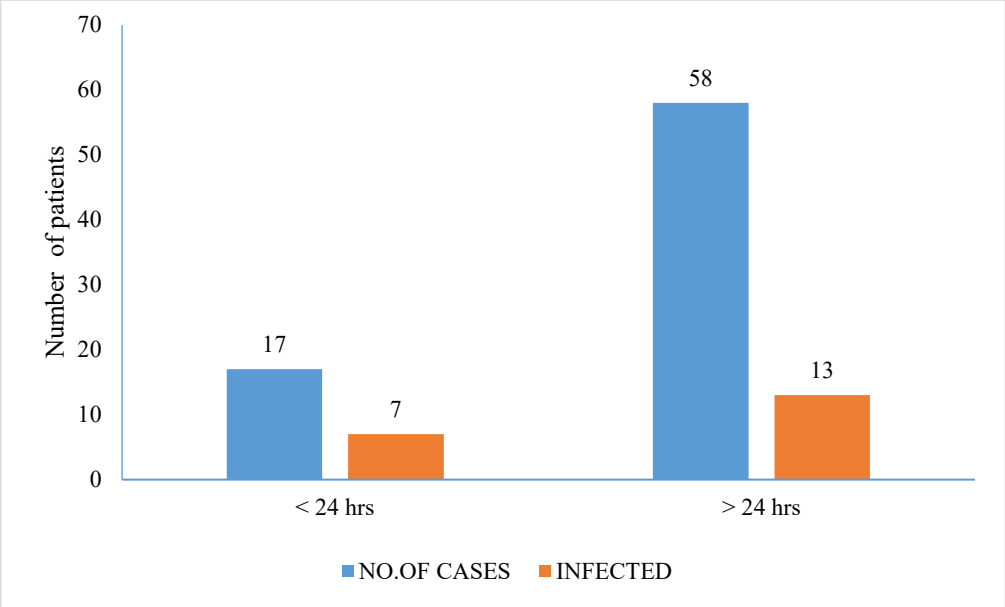
SSI Rates in patients with obesity was 25%

In patients who were alcohol consumers, SSI rate was 20%.

Incidence in hypertensives was seen in 18.7% patients

TABLE 5: INCIDENCE IN RELATION TO TIME TAKEN TO REACH HOSPITAL

TIME TAKEN TO REACH HOSPITAL	NO.OF CASES	INFECTED	PERCENTAGE
< 24 hrs	17	7	41.2%
> 24 hrs	58	13	22.4%
TOTAL	75	20	

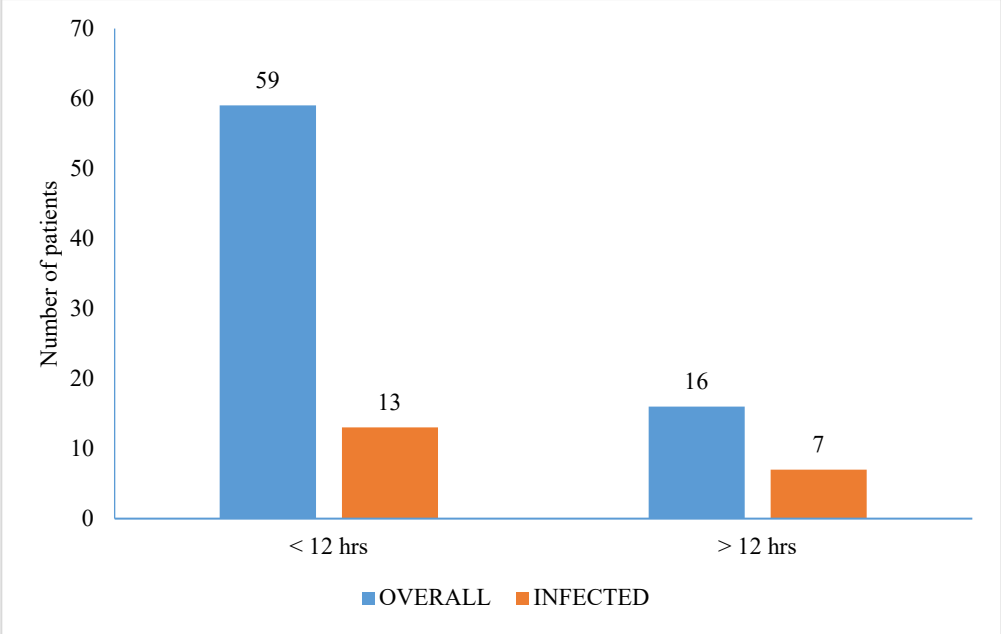


GRAPH 5: Bar diagram showing distribution of SSI in relation to time taken to reach hospital.

Most of the patient presented to the hospital after 24 hrs of their symptom onset. 41.2% of patients who reached the hospital within 24 hrs of first symptom onset developed SSI.SSI rate in patients reaching the hospital after 24 hrs of symptom onset was 22.4%.

TABLE 6: INCIDENCE IN RELATION TO ADMISSION TO OPERATION TIME

ADMISSION TO OPERATION TIME	OVERALL	INFECTED	PERCENTAGE
< 12 hrs	59	13	22%
> 12 hrs	16	7	43.7%
TOTAL	75	20	

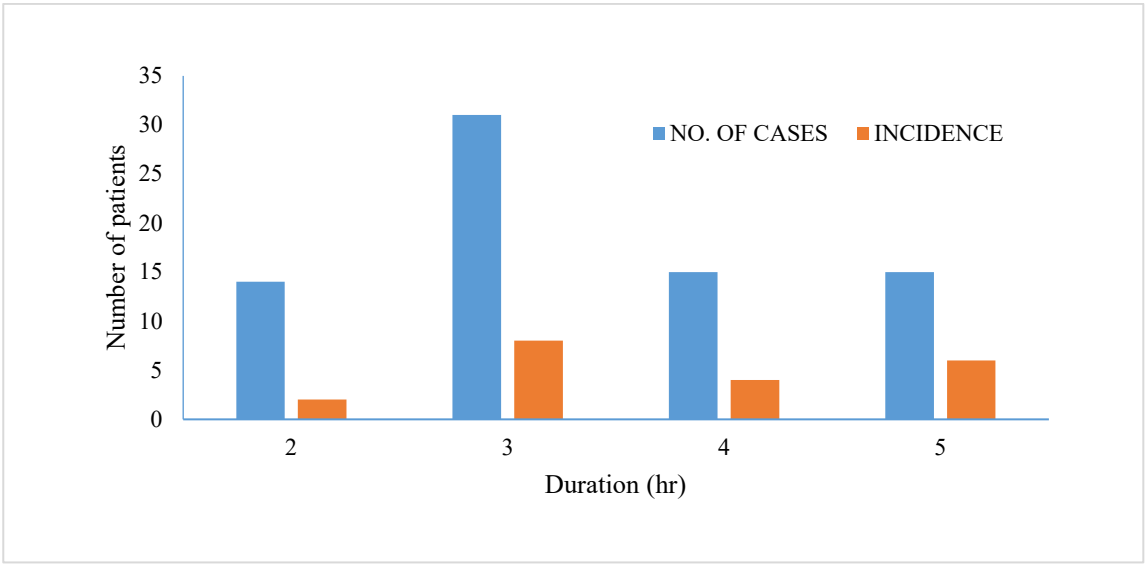


GRAPH 6: Bar diagram showing distribution of SSI in relation to admission to operation time.

Most of the patients were operated within 12 hr of their admission to the hospital. Patients who were operated after 12 hrs of admission to hospital had a higher SSI rate of 43.7% against 22% in patients who were operated within 12 hrs of admission.

TABLE 7 : INCIDENCE IN RELATION TO DURATION OF SURGERY

DURATION IN HOURS	NO. OF CASES	INCIDENCE	PERCENTAGE
2-3	14	2	14.3%
3-4	31	8	25.8%
4-5	15	4	26.7%
>5	15	6	40%
TOTAL	75	20	



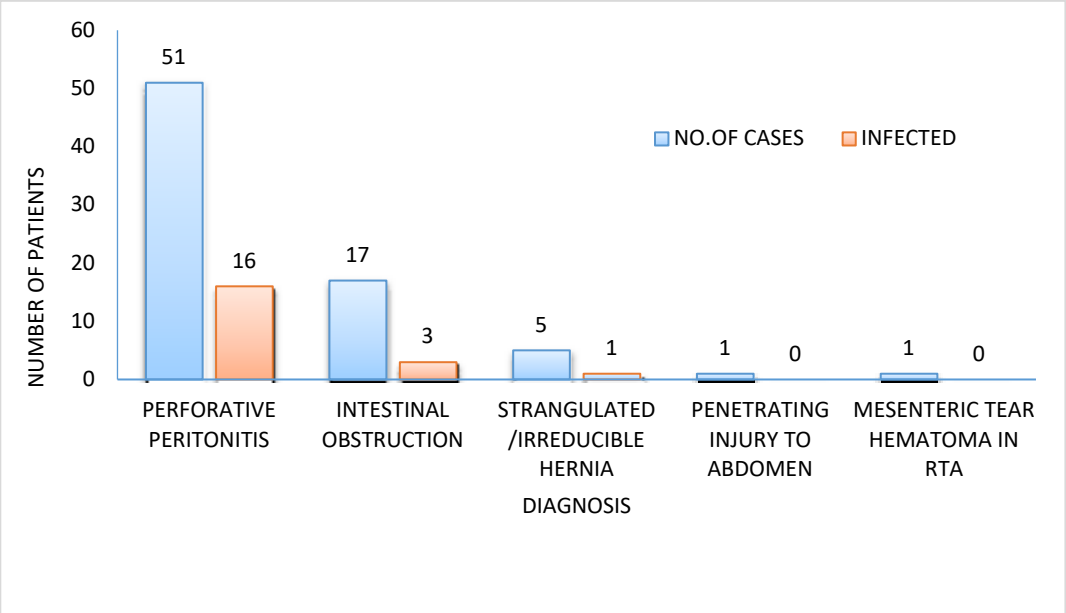
GRAPH 7: Bar diagram showing SSI distribution in relation to duration of surgery.

Incidence of surgical infection was more i.e. 40% in cases where the operative procedure lasted >5 hrs.

SSI rates where operative procedure was done between 4-5 hrs was 26.7%, between 3-4 hrs was 25.8 % and between 2-3 hrs was 14.3%.

TABLE 8: INCIDENCE IN RELATION TO DIAGNOSIS

DIAGNOSIS	NO.OF CASES	INFECTED	PERCENTAGE
PERFORATIVE PERITONITIS	51	16	31.4%
INTESTINAL OBSTRUCTION	17	3	17.6%
STRANGULATED /IRREDUCIBLE HERNIA	5	1	20%
PENETRATING INJURY TO ABDOMEN	1	0	0%
MESENTERIC TEAR HEMATOMA IN RTA	1	0	0%
TOTAL	75	20	



GRAPH 8: Bar diagram showing SSI incidence in relation to diagnosis.

Most of the patient presenting with peritonitis presented with peritonitis secondary to bowel perforation.

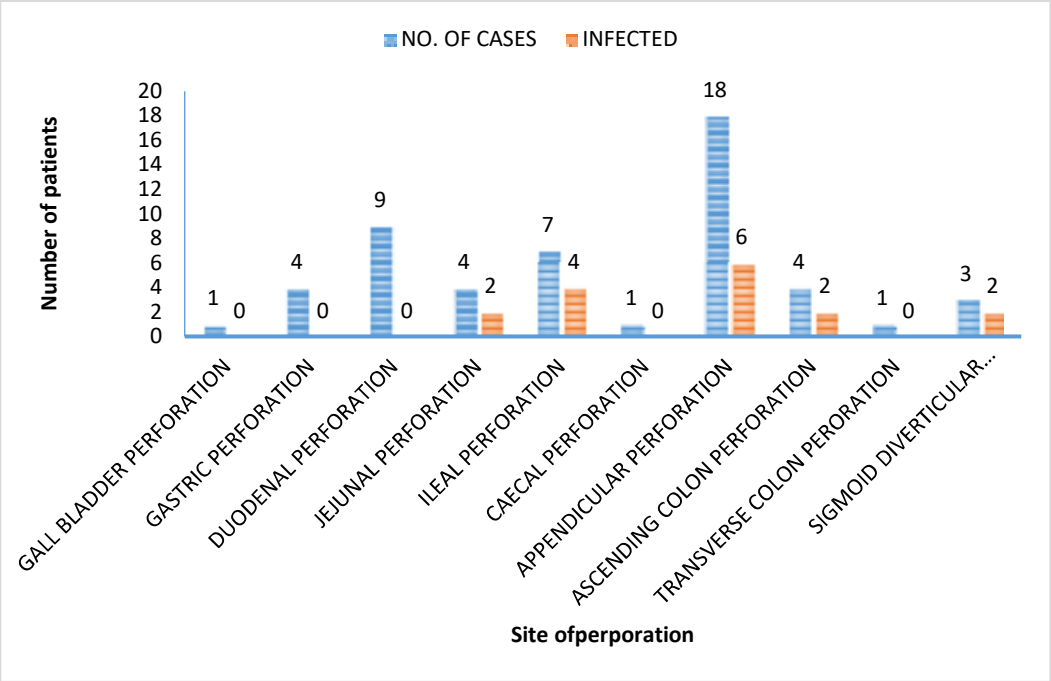
followed by this was peritonitis secondary to irreducible /strangulated hernia.

SSI rate was higher, 31.4 % in patients with perforative peritonitis.

It was 20% in patients with strangulated or irreducible hernia and 17.6% in patients with peritonitis secondary to intestinal obstruction.

TABLE 9: INCIDENCE IN RELATION TO SITE OF PERFORATION

SITE OF PERFORATION	NO. OF CASES	INFECTED	PERCENTAGE
GALL BLADDER PERFORATION	1	0	0
GASTRIC PERFORATION	4	0	0
DUODENAL PERFORATION	9	0	0
JEJUNAL PERFORATION	4	2	50%
ILEAL PERFORATION	7	4	57.1%
CAECAL PERFORATION	1	0	0
APPENDICULAR PERFORATION	18	6	33.3%
ASCENDING COLON PERFORATION	4	2	50%
TRANSVERSE COLON PERORATION	1	0	0
SIGMOID DIVERTICULAR PERFORATION	3	2	66.6%



GRAPH 9: Bar diagram showing distribution of SSI in relation to site of perforation.

SSI rate was found to be most (66.6%) when there was a sigmoid diverticular perforation, followed by 57.1 % in cases of ileal perforation

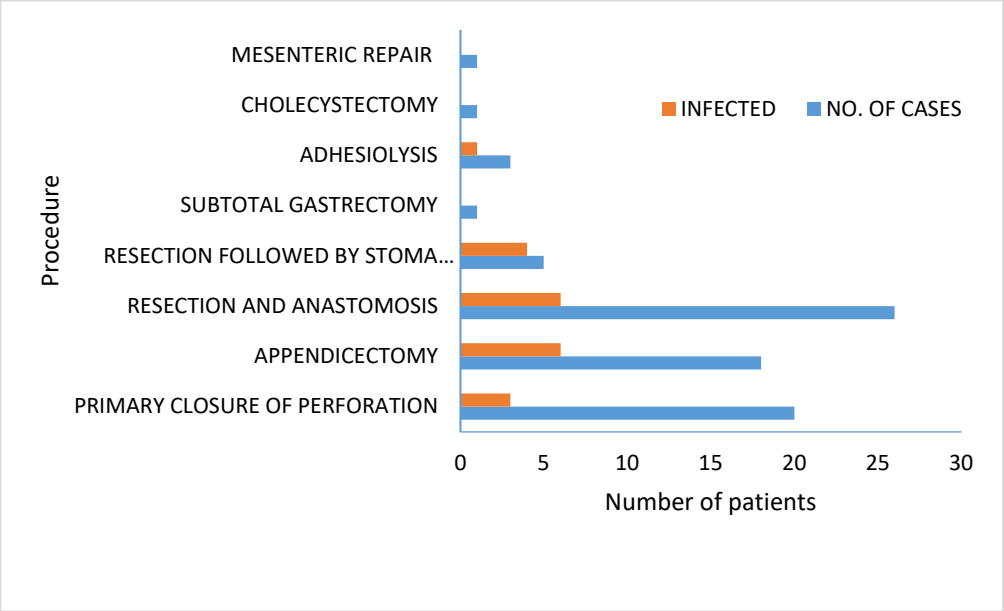
Incidence of SSI was 50% each in cases with jejunal and ascending colon perforation.

Patients with appendicular perforation had a SSI rate of 33.3%

No infection was noted in cases of gall bladder perforation, gastric perforation, duodenal perforation, caecal perforation and ascending colon perforation.

TABLE 10: INCIDENCE IN RELATION TO THE PROCEDURE PERFORMED

PROCEDURE	NO. OF CASES	INFECTED	PERCENTAGE
PRIMARY CLOSURE OF PERFORATION	20	3	15%
APPENDICECTOMY	18	6	33.3%
RESECTION AND ANASTOMOSIS	26	6	23.1%
RESECTION FOLLOWED BY STOMA FORMATION	5	4	80%
SUBTOTAL GASTRECTOMY	1	0	0
ADHESIOLYSIS	3	1	33.3%
CHOLECYSTECTOMY	1	0	0
MESENTERIC REPAIR	1	0	0
TOTAL	75	20	



GRAPH 10: Bar diagram showing distribution of SSI in relation to the procedure performed.

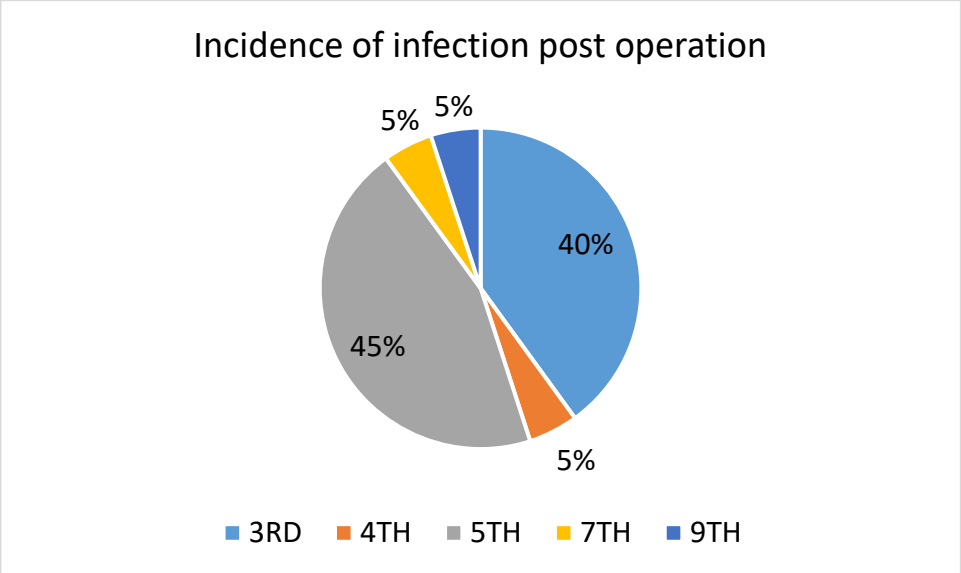
Resection and anastomosis was the procedure done for the maximum number of cases, however SSI infection rate in these cases was 23.1% in our study.

Infection was found to be maximum in case operated for resection followed by stoma formation in the form of end colostomy or jejunostomy (80%), whereas no SSI was

noted in cases operated for subtotal gastrectomy, cholecystectomy for gall bladder perforation and mesenteric repair patient.

TABLE 11: INCIDENCE OF INFECTION NOTED ON POST OPERATIVE DAY

DAY	NO. OF INFECTED CASES	PERCENTAGE
3RD	8	40%
4TH	1	5%
5TH	9	45%
7TH	1	5%
9TH	1	5%
TOTAL	20	

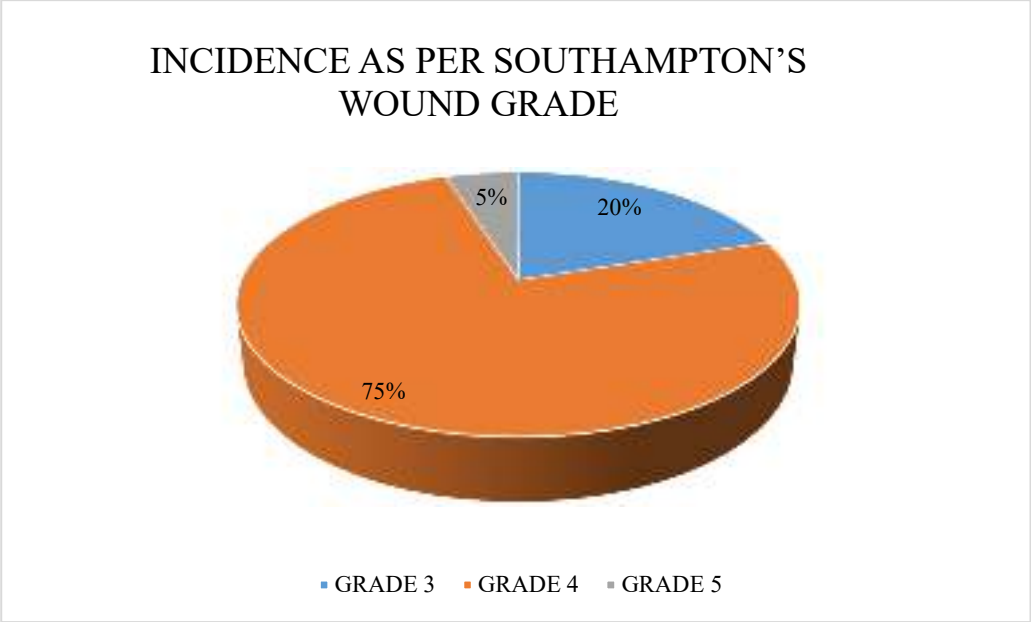


GRAPH 11: Pie chart showing distribution of infection noticed in relation to post operative day.

In 45 % of the patients with SSI, infection was noted on 5th post-operative day whereas on the 3rd post-operative day in 40% of the infected cases.

TABLE 12: INCIDENCE AS PER SOUTHAMPTON'S WOUND GRADE

GRADE	NO. OF CASES	PERCENTAGE
GRADE 3	**4**	**20%**
GRADE 4	**15**	**75%**
GRADE 5	**1**	**5%**

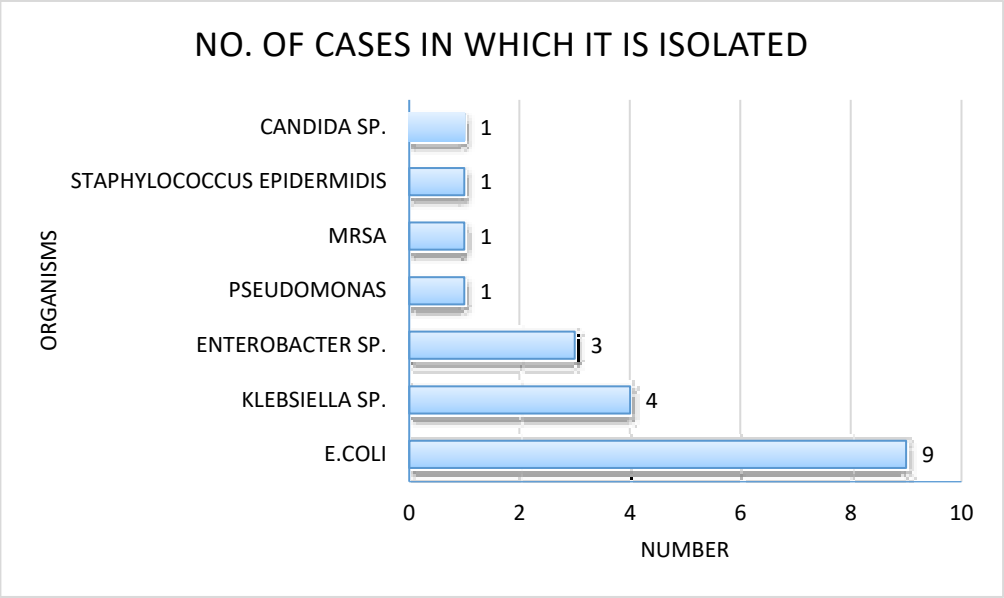


GRAPH 12: Pie chart showing percentage distribution of Southampton's wound grade among those with SSI.

75% of the infected cases were graded as grade 4 in our study with peritonitis patients. 20% of the patients with SSI showed features corresponding with grade 3 as per Southampton's wound grade. Only 1 patient who suffered an anastomotic site leak was graded as grade 5 corresponding to 5 % of the infected cases.

TABLE 13: INCIDENCE OF ORGANISM ISOLATED

ORGANISM	NO. OF CASES IN WHICH IT IS ISOLATED	PERCENTAGE
E.COLI	9	45%
KLEBSIELLA SP.	4	20%
ENTEROBACTER SP.	3	15%
PSEUDOMONAS	1	5%
MRSA	1	5%
STAPHYLOCOCCUS EPIDERMIDIS	1	5%
CANDIDA SP.	1	5%
TOTAL	20	

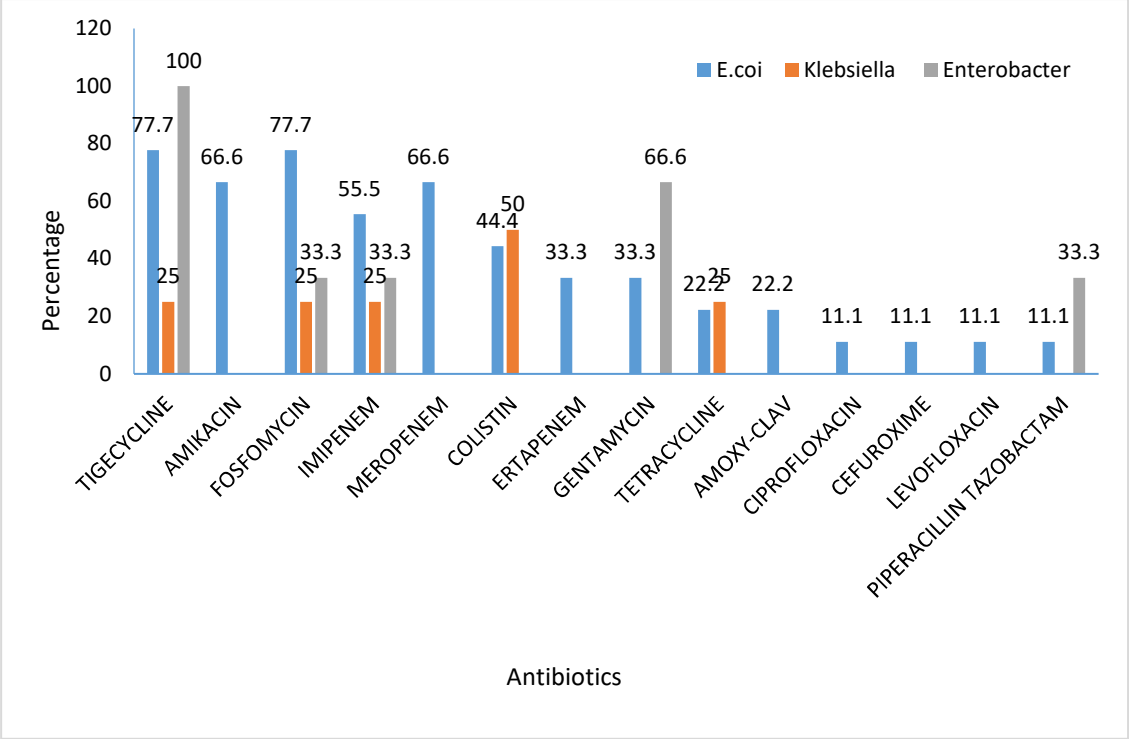


GRAPH 13: Bar diagram showing distribution of the organism isolated among those with SSI.

E. coli was noted as the most common organism isolated from 40% of the infected cases, followed by klebsiella species which was isolated from 20% of the cases with surgical site infection.

TABLE 14: ANTIBIOTIC SENSITIVITY SPECTRUM

ANTIBIOTIC	ECOLI	%	KLEBSI-ELLA	%	ENTERO-BACTER	%
TIGECYCLINE	7/9	77.7	1/4	25	3/3	100
AMIKACIN	6/9	66.6				
FOSFOMYCIN	7/9	77.7	1/4	25	1/3	33.3
IMIPENEM	5/9	55.5	1/4	25	1/3	33.3
MEROPENEM	6/9	66.6				
COLISTIN	4/9	44.4	2/4	50		
ERTAPENEM	3/9	33.3				
GENTAMYCIN	3/9	33.3			2/3	66.6
TETRACYCLINE	2/9	22.2	1/4	25		
AMOXY-CLAV	2/9	22.2				
CIPROFLOXACIN	1/9	11.1				
CEFUROXIME	1/9	11.1				
LEVOFLOXACIN	1/9	11.1				
PIPERACILLIN TAZOBACTAM	1/9	11.1			1/3	33.3



GRAPH 14: Bar diagram showing antibiotic sensitivity pattern of the isolated organism

E. coli was found to be most sensitive to tigecycline and Fosfomycin, followed by meropenem and amikacin followed by imipenem, colistin as per the culture sensitivity reports. Klebsiella species that was isolated showed sensitivity maximum to colistin followed by Fosfomycin, imipenem, tigecycline and tetracycline

DISCUSSION

This study was conducted at KLE's Dr. Prabhakar Kore Hospital and MRC, Belgaum and included a total of 75 patients who underwent surgery in view of peritonitis.

Surgical site infection is one of the leading causes of morbidity in patient who undergo surgery and its incidence varies greatly from one geographical place to another and one hospital to another. A varied difference has been observed in developing nations when comparison is drawn with developed nations.

The SSI incidence in the present study with peritonitis patients was 26%. This is consistent with studies dealing with similar patients in India,

26.4% in study done by Singh and Yadalwar(69),25.2 % in a study by Satyanarayana et al(70),26.3% in study by Deshmukh and Bhise(71), Rajesh K. abbey 25.43%(72), and lower than study by Mekhla and Borle -39%.(73)

The study population in this study belonged to age group between 16 to 72 years with a mean age of 47.07. Age has been associated with increased risk of SSI as per previous studies(74,75), however, Incidence was noted to be 45.5% in younger age group and 33.3% in the older population, being inconsistent with those studies.

A study done by Lawson et al(76) for surgical site infection noted a significantly lower risk adjusted odds of SSI in older age categories compared with patients younger than 55 years which stands true for our study showing 30.23% in age <55 years and 21.9% in age >55 years.

Some investigators have come to the conclusion that increasing age was not an independent risk factor for SSI.(77,78)

This aspect needs to be studies in further details with a larger sample size.

Regarding sex distribution, the rate of SSI in patients operated for peritonitis in our study in male was 22.45% and in female patients was 34.62%. Relation of sex distribution of surgical site infection is not very well understood. In few studies previously male preponderance was noted (70,72,79,80)and justified to be in association with smoking, alcohol consumption and poor wound healing.

Incidence among the risk factors like anaemia 33.3%, diabetes mellitus 38.5% and hypoalbuminemia 44.8%. Similar results and association have been noted in studies with similar category of patients.(81,82).This can be justified as these factors play a major role in wound healing, growth of micro-organisms (hyperglycaemia), immunocompromised state and poor collagen deposition.(81)

25% of patients who were obese were seen to develop surgical site infection which is consistent with the literature showing increase in SSI with obesity quoting the decreased supply of blood in fatty tissues. In contrast, in a study done by Mekhla and Borle no such association could be drawn.(73)

Association with smoking was noted in in 40% of the patients. Previous studies(69,73,81,82) have shown a significant linkage between smoking and poor wound healing, poor collagen deposition, reduced oxygen delivery to tissues which prepares an environment where surgical site infection can happen.

This study noted that the number of cases took more time to reach the hospital after the onset of their first symptom, similar to that noted in study done by Kaur et al(83), which mentions an increased morbidity due to late hospital presentation.

However, in this study the patients who reached the hospital within 24 hrs of symptom onset have 41.2% infection rate in comparison to those reaching after 24 hrs of symptom onset which was 22.4%. This can be attributable to the fact that patients with acute abdomen and perforative peritonitis may present within 24 hrs because of

the severity of their symptoms and have increased SSI risk due to well established peritonitis.

This study depicts that the time taken from the admission to operation was <12 hrs in majority of the cases and the SSI rates were higher in those who were operated >12 hrs after admission 43.7% against 22 %(operated within 12hrs). This is in accordance with a previous study where SSI rates were the following- 12.9% if operated <24 hrs,18.9% if operated 24-48 hrs later,31.7 % if operated 48-72 hrs later and 55.2% if operated after > 72hrs (Singh and Yadalwar)(69)

This study depicted a rise in SSI rates with increase in duration of surgery which is a finding in almost all previous studies performed. Studies have suggested that surgical time more than 2 hrs causes the risk of SSI to be doubled up. This finding can be attributed to the fact that severe adhesions requiring adhesiolysis, complex surgeries, more handling to tissues cause more risk of micro-organism growth and decreases the prophylactic antibiotics to reach the tissues due to reduced circulation. With every hour of operation, a doubling up of SSI rates was noted by Doherty et al in their study.(84)

Study shows that incidence of SSI was 31.4% for cases with perforative peritonitis, 20% in cases with strangulated /irreducible hernia and 17.6% with cases of intestinal obstruction. Increased rate in perforative peritonitis patients is attributable to direct exposure of gut organisms to the peritoneal cavity leading to more risk of SSI.(81)

Incidence of SSI was noted to be maximum with sigmoid diverticular perforation (66.6%) in comparison to duodenal and jejunal perforations, attributable to the feculent content with high bacterial load in sigmoid.

The incidence of SSI was the highest for resection followed by stoma formation noted to be 80% which is in accordance with previous studies done for similar emergency procedures.(85)

In present study the most common organism isolated is E. coli- 45%, followed by klebsiella species in 20% cases with SSI. This finding is consistent with study done for abdominal operations by A alkaaki (86)and Dessie et al.(87), Singh and yadalwar(69)

In the present study the organisms were found sensitive to higher antibiotics .This can be attributed to the use of higher antibiotics as and when peritonitis is suspected in patients presenting to our hospital.

The present study points out to the need for detailed study as SSI in peritonitis has been less studied and is a major cause of morbidity still. Stringent measures are the need of the hour along with a sound antibiotic policy.

LIMITATIONS

There are several factors which affect the development of surgical site infection but all of the factors could not be studied in our study.

Results of this study may not be completely generalizable due to the small sample size.

Long term follow up of these patients was not assessed in order to check for

long term postoperative complications.

CONCLUSION

- Incidence of abdominal surgical site infection in cases operated for peritonitis in KLES Dr. Prabhakar Kore hospital and MRC is 26%.
- Incidence of surgical site infection among the risk factors like anaemia 33.3%, diabetes mellitus 38.5% and hypoalbuminemia 44.8% is noted to be higher.
- 25% of patients who were obese were seen to develop surgical site infection.
- 20% (15) of the study population had the habit of smoking. 40% incidence of SSI was noted among smokers in our study.
- Maximum patients were operated within 12 hours of presentation to the hospital, and had lesser incidence of SSI when compared with those operated after 12 hours of admission.
- Incidence of surgical site infection was noted to be 14.3% when surgery lasted between 2-3 hrs duration, and was 40% if the time taken for the procedure exceeded 5 hours.
- SSI incidence was 31.4% for cases with perforative peritonitis ,20% in cases with strangulated /irreducible hernia and 17.6% with cases of intestinal obstruction
- Maximum incidence of SSI was noted in cases with sigmoid diverticular perforation (66%) followed by ileal perforation (57.1%)
- Resection and anastomosis were the most common procedure performed in patients presenting with peritonitis however incidence of SSI was 23.1%
- Patients in whom resection was followed by stoma creation have an SSI incidence of 80%.
- Most common organism isolated from wound culture is E. coli- 45%, followed by klebsiella species in 20% cases with SSI.
- Overall tigecycline and Fosfomycin were the most sensitive antibiotics.

SUMMARY

- ✓ Incidence of abdominal SSI in cases operated for peritonitis is 26% in our setup.
- ✓ Risk factors like hypoalbuminemia, anaemia and diabetes mellitus, obesity, smoking are associated with increased wound infection rate.
- ✓ Lesser time gap between admission to operation associates with a lesser incidence of SSI.
- ✓ Longer the duration of surgery more is the SSI rate.
- ✓ SSI incidence is most with perforative peritonitis when compared with other causes of peritonitis.
- ✓ Procedure wise resection followed by stoma formation has the higher SSI rate.
- ✓ E. coli is the most common organism isolated from infected wounds in this study.
- ✓ Most of the organisms are sensitive to higher antibiotics.

Following are recommended for reducing SSI in patients operated for peritonitis.

- Strict surveillance and feedback results to surgeons
- Reducing the door to surgery time to as low as possible.
- Minimizing the length of operation.
- Taking stringent care in patients who have risk factors,comorbidities.

BIBLIOGRAPHY

1. de Lissovoy G, Fraeman K, Hutchins V, Murphy D, Song D, Vaughn BB. Surgical site infection: Incidence and impact on hospital utilization and treatment costs. American Journal of Infection Control [Internet]. 2009;37(5):387–97. Availablefrom: https://www.sciencedirect.com/science/article/pii/S019665530900073X
2. McLaws M-L, Irwig LM, Mock P, Berry G, Gold J. Predictors of surgical wound infection in Australia: a national study. Medical Journal of Australia [Internet]. 1988 Dec 1;149(11–12):591–5. Available from: https://doi.org/10.5694/j.1326-5377.1988.tb120796.x
3. Barie PS. Surgical Site Infections: Epidemiology and Prevention. Surgical Infections [Internet]. 2002 Dec 1;3(s1):s9–21. Available from: https://doi.org/10.1089/sur.2002.3.s1-9
4. Ballus J, Lopez-Delgado JC, Sabater-Riera J, Perez-Fernandez XL, Betbese AJ, Roncal JA. Surgical site infection in critically ill patients with secondary and tertiary peritonitis: epidemiology, microbiology and influence in outcomes. BMC Infectious Diseases [Internet]. 2015;15(1):304. Available from: https://doi.org/10.1186/s12879-015-1050-5
5. Krizek TJ, Robson MC. Evolution of quantitative bacteriology in wound management. The American Journal of Surgery [Internet]. 1975;130(5):579–84. Available from: https://www.sciencedirect.com/science/article/pii/0002961075905164

6. Owens CD, Stoessel K. Surgical site infections: epidemiology, microbiology and prevention. Journal of Hospital Infection [Internet]. 2008;70:3–10. Available from: https://www.sciencedirect.com/science/article/pii/S0195670108600171
7. Sawyer Robert .G., and Timothy L. pruett, "Wound Infections". Surgical Clinics of North America, 1994, 74; 519 –36.
8. Howard.J.R., „Surgical infections" Principles of surgery vol.I edtd by Schwartz published by Mc Grawhill, inc 143-75.
9. Wangensteen OH, Wangensteen SD, Freshwater MF. The Rise of Surgery: From Empiric Craft to Scientific Discipline. Plastic and Reconstructive Surgery [Internet]. 1980;65(4). Available from: https://journals.lww.com/plasreconsurg/Fulltext/1980/04000/The_Rise_of_Surgery__From_Empiric_Craft_to.29.aspx
10. Rutkow IM. Appendicitis: The Quintessential American Surgical Disease. Archives of Surgery [Internet]. 1998 Sep 1;133(9):1024. Available from: https://doi.org/10-1001/pubs.Arch Surg.-ISSN-0004-0010-133-9-ssh0998
11. Meleney FL. Bacterial synergism in disease processes: with a confirmation of the synergistic bacterial aetiology of a certain type of progressive gangrene of the abdominal wall. Annals of surgery [internet]. 1931 dec;94(6):961–81. Available from: https://pubmed.ncbi.nlm.nih.gov/17866704
12. Altemeier WA. Manual of Control of Infection in Surgical Patients. Chicago: American College of Surgeons Press: 1976:1. 6. Bartlett JG. Intra-abdominal sepsis. Med Clin North Am. 1995;79:599-617. 7. Dunn DL, Simmons RL. The role of anaerobic bacteria in intra- abdominal infections. Rev Infect Dis. 1984;6:S139-S146.

13. B'erard F. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. Ann Surg. 1964;160(1):1-92.

14.Chen WJ, Rogers AA, Lydon MJ. Characterization of biologic properties of wound fluid collected during early stages of wound healing. Journal of investigative dermatology. 1992 Nov 1;99(5):559-64.

15. Beldon P. Basic science of wound healing. Surgery (Oxford). 2010 Sep 1;28(9):409-12.

16. Phillips SJ. Physiology of wound healing and surgical wound care. ASAIO journal. 2000 Nov 1;46(6):S2-5.

17. Pakyari M, Farrokhi A, Maharlooei MK, Ghahary A. Critical role of transforming growth factor beta in different phases of wound healing. Advances in wound care. 2013 Jun 1;2(5):215-24.

18. Shahrim Z, Zaliha O, Zainal Z, Aldrin Z. Palm Tocotrienol: A Good Antioxidant for Skin Wound Healing. 2016.

19. Tissue Renewal and Repair: Regeneration, Healing and Fibrosis. 9th ed. In: Robbins and Cotron Pathologic Basis of Disease, Kumar V, Abbas KA, Fausto N (ed). Pennsylvania: Saunders; 2015. pp. 111-14.

20. Moucha CS, Clyburn T, Evans RP, Prokuski L. Modifiable risk factors for surgical site infection. JBJS. 2011 Feb 16;93(4):398-404.

21. Lobley SN. Factors affecting the risk of surgical site infection and methods of reducing it. Journal of perioperative practice. 2013 Apr;23(4):77-81.

22. Evans RP. Surgical site infection prevention and control: an emerging paradigm. JBJS. 2009 Nov 1;91(Supplement_6):2-9.

23. Alfonso-Sanchez JL, Martinez IM, Martín-Moreno JM, González RS, Botía F. Analyzing the risk factors influencing surgical site infections: the site of environmental factors. Canadian Journal of Surgery. 2017 Jun;60(3):155.

24. Gould D. Causes, prevention and management of surgical site infection. Nursing Standard (through 2013). 2012 Jul 25;26(47):47.

25. Owens CD, Stoessel K. Surgical site infections: epidemiology, microbiology and prevention. Journal of hospital infection. 2008 Nov 1;70:3-10.

26. McHugh SM, Hill AD, Humphreys H. Intraoperative technique as a factor in the prevention of surgical site infection. Journal of Hospital Infection. 2011 May 1;78(1):1-4.

27. Lilani SP, Jangale N, Chowdhary A, Daver GB. Surgical site infection in clean and clean-contaminated cases. Indian journal of medical microbiology. 2005 Oct 1;23(4):249-52.

28. Anderson DJ, Sexton DJ, Kanafani ZA, Auten G, Kaye KS. Severe surgical site infection in community hospitals: epidemiology, key procedures, and the changing prevalence of methicillin-resistant Staphylococcus aureus. Infection Control & Hospital Epidemiology. 2007 Sep;28(9):1047-53.

29. Engemann JJ, Carmeli Y, Cosgrove SE, Fowler VG, Bronstein MZ, Trivette SL, Briggs JP, Sexton DJ, Kaye KS. Adverse clinical and economic outcomes attributable to methicillin resistance among patients with Staphylococcus aureus surgical site infection. Clinical infectious diseases. 2003 Mar 1;36(5):592-8.

30. Weigelt JA, Lipsky BA, Tabak YP, Derby KG, Kim M, Gupta V. Surgical site infections: causative pathogens and associated outcomes. American journal of infection control. 2010 Mar 1;38(2):112-20.

31. Ballus J, Lopez-Delgado JC, Sabater-Riera J, Perez-Fernandez XL, Betbese AJ, Roncal JA. Surgical site infection in critically ill patients with secondary and tertiary peritonitis: epidemiology, microbiology and influence in outcomes. BMC Infectious Diseases [Internet]. 2015;15(1):304. Available from: https://doi.org/10.1186/s12879-015-1050-5
32. National Healthcare Safety Network, Centers for disease control and prevention. Surgical site infection event (SSI) [Internet]. 2021. [cited 20.03.2021]. Available from: https://www.cdc.gov/nhsn/pdfs/pscmanual/9pscssicurrent.pdf.
33. Kirby JP, Mazuski JE. Prevention of surgical site infection. Surgical Clinics of North America. 2009 Apr 1;89(2):365-89.
34. Pauli EM, Krpata DM, Novitsky YW, Rosen MJ. Negative pressure therapy for high-risk abdominal wall reconstruction incisions. Surgical infections. 2013 Jun 1;14(3):270-4.
35. Campwala I, Unsell K, Gupta S. A comparative analysis of surgical wound infection methods: predictive values of the CDC, ASEPSIS, and Southampton scoring systems in evaluating breast reconstruction surgical site infections. Plastic Surgery. 2019 May;27(2):93-9.
36. Gottrup F, Melling A, Hollander DA. An overview of surgical site infections: aetiology, incidence and risk factors. EWMA journal. 2005 Sep;5(2):11-5.
37. Kastelein AW, Vos LM, de Jong KH, van Baal JO, Nieuwland R, van Noorden CJ, Roovers JP, Lok CA. Embryology, anatomy, physiology and pathophysiology of the peritoneum and the peritoneal vasculature. InSeminars in cell & developmental biology 2019 Aug 1 (Vol. 92, pp. 27-36). Academic Press.

38.Blackburn SC, Stanton MP. Anatomy and physiology of the peritoneum. InSeminars in pediatric surgery 2014 Dec 1 (Vol. 23, No. 6, pp. 326-330). WB Saunders.

39. Ross JT, Matthay MA, Harris HW. Secondary peritonitis: principles of diagnosis and intervention. Bmj. 2018 Jun 18;361.

40. Sulaiman H, Gabella G, Davis C, Mutsaers SE, Boulos P, Laurent GJ, Herrick SE. Presence and distribution of sensory nerve fibers in human peritoneal adhesions. Annals of surgery. 2001 Aug;234(2):256.

41. Borgonovo G, Amato A, Varaldo E, Mattioli FP. Definition and classification of peritonitis. Médecine et maladies infectieuses. 1995 Dec 1;25:7-12.

42. Thereaux J, Veyrie N, Corigliano N, Servajean S, Czernichow S, Bouillot JL. Is laparoscopy a safe approach for diffuse appendicular peritonitis? Feasibility and determination of risk factors for post-operative intra-abdominal abscess. Surgical endoscopy. 2014 Jun;28(6):1908-13.

43. Nascimbeni R, Amato A, Cirocchi R, Serventi A, Laghi A, Bellini M, Tellan G, Zago M, Scarpignato C, Binda GA. Management of perforated diverticulitis with generalized peritonitis. A multidisciplinary review and position paper. Techniques in Coloproctology. 2021 Feb;25(2):153-65.

44. Prasad NB, Reddy KB. A study of acute peritonitis: evaluation of its mortality and morbidity. International Surgery Journal. 2016 Dec 8;3(2):663-8.

45. Rohit DK, Verma RS, Pandey G. Clinical study and management of peritonitis secondary to perforated peptic ulcer. International Surgery Journal. 2017 Jul 24;4(8):2721-6.

46. Bennion RS, Thompson JE, Baron EJ, Finegold SM. Gangrenous and perforated appendicitis with peritonitis: treatment and bacteriology. Clinical therapeutics. 1990 Jan 1;12:31-44.

47. Dudgeon LS, Sargent PW. The bacteriology of peritonitis. Constable; 1905.

48. Dubreuil L, Veloo AC, Sóki J. Correlation between antibiotic resistance and clinical outcome of anaerobic infections; mini-review. Anaerobe. 2021 Sep 28:102463.

49. Robin Kaushik. Antibiotic therapy in peritonitis:Emergency Surgery for Peritonitis Made Easy®. 2009. 31–40.

50. Clements TW, Tolonen M, Ball CG, Kirkpatrick AW. Secondary peritonitis and intra-abdominal sepsis: An increasingly global disease in search of better systemic therapies. Scandinavian Journal of Surgery. 2021 Jan 7:1457496920984078.

51. Wittmann DH, Schein M, Condon RE. Management of secondary peritonitis. Annals of surgery. 1996 Jul;224(1):10.

52. van Till JO, Van Veen SQ, van Ruler O, Lamme B, Gouma DJ, Boermeester MA. The innate immune response to secondary peritonitis. Shock. 2007 Nov 1;28(5):504-17.

53. Hall JC, Heel KA, Papadimitriou JM, Platell C. The pathobiology of peritonitis. Gastroenterology. 1998 Jan 1;114(1):185-96.

54. Johnson CC, Baldessarre J, Levison ME. Peritonitis: update on pathophysiology, clinical manifestations, and management. Clinical infectious diseases. 1997 Jun 1;24(6):1035-45.

55. Wong PF, Gilliam AD, Kumar S, Shenfine J, O'Dair GN, Leaper DJ. Antibiotic regimens for secondary peritonitis of gastrointestinal origin in adults. Cochrane database of systematic reviews. 2005(2).

56. Doklestić SK, Bajec DD, Djukić RV, Bumbaširević V, Detanac AD, Detanac SD, Bracanović M, Karamarković RA. Secondary peritonitis-evaluation of 204 cases and literature review. Journal of medicine and life. 2014 Jun 15;7(2):132.

57. Spalding DR, Williamson RC. Peritonitis. British Journal of Hospital Medicine (2005). 2008 Jan;69(Sup1):M12-5.

58. Kloth C, Vogele D, Brunner H, Beer M, Schmidt SA. Pathognomonic imaging signs in abdominal radiology. Abdominal radiology. 2020 Feb;45(2):576-86.

59. Mularski RA, Ciccolo ML, Rappaport W. Nonsurgical causes of pneumoperitoneum. Western Journal of Medicine. 1999 Jan;170(1):41.

60. Saber AA, Boros MJ. Chilaiditi's syndrome: what should every surgeon know?. The American surgeon. 2005 Mar 1;71(3):261-3.

61. Pinto A, Miele V, Schillirò ML, Nasuto M, Chiaese V, Romano L, Guglielmi G. Spectrum of signs of pneumoperitoneum. InSeminars in Ultrasound, CT and MRI 2016 Feb 1 (Vol. 37, No. 1, pp. 3-9). WB Saunders.

62. Nathens AB, Rotstein OD. Therapeutic options in peritonitis. Surgical Clinics of North America. 1994 Jun 1;74(3):677-92.

63. Robin Kaushik. General Principles in Operative Management of Secondary Peritonitis:Emergency Surgery for Peritonitis Made Easy. 2009. 23–30.

64. Strobel O, Werner J, Büchler MW. Surgical therapy of peritonitis. Der Chirurg; Zeitschrift fur Alle Gebiete der Operativen Medizen. 2011 Mar 1;82(3):242-8.

65. Fry DE. The prevention of surgical site infection in elective colon surgery. Scientifica. 2013 Oct;2013.

66. Devriese LA, Dutta GN. Antibiotic sensitivity testing: correlations between in vitro tests and in vivo situations. InAnnales de Recherches Vétérinaires 1981 (Vol. 12, No. 1, pp. 41-46).

67. Giedraitienė A, Vitkauskienė A, Naginienė R, Pavilonis A. Antibiotic resistance mechanisms of clinically important bacteria. Medicina. 2011 Mar;47(3):19.

68. Mukherjee R, Samanta S. A comparative study of primary skin and subcutaneous tissue closure and open skin technique in emergency laparotomy for perforative peritonitis: an observational and prospective study. International Surgery Journal. 2018 Dec 27;6(1):166-72.

69. 12. Singh LM, Yadalwar V. Study of Post-operative Surgical Site Infection in Patients of Emergency Laparotomy in Teaching Hospital of Central India. IJSS Journal of Surgery 2018;4(1):59-64.

70. Satyanarayana V, Prashanth HV, Basavaraj B, Kavyashree AN. Study of surgical site infections in abdominal surgeries. J Clin Diagn Res. 2011 Oct;5(5):935-.

71. Mukta M. Deshmukh, P.R. Bhise (2020); A Study of Surgical Site Infections (SSI) In Emergency Surgeries in A Tertiary Care Hospital; International Journal of Scientific and Research Publications (IJSRP) 10(07) (ISSN: 2250-3153).

72. Abbey RK, Mohan M, Malik N, Tiwari R, Nahar S. Surgical site infection in a rural teaching hospita of North India. Int J Adv Integ Med Sci 2017;2;11-6.

73. Mekhla FR. Determinants of superficial surgical site infections in abdominal surgeries at a Rural Teaching Hospital in Central India: A prospective study. Journal of family medicine and primary care. 2019 Jul;8(7):2258.

74. Mulu W, Kibru G, Beyene G, Datie M. Postoperative nosocomial infections and antimicrobial resistance patterns of bacterial isolates among patients admitted at Felege Hiwot Referral Hospital, Bahirdar, Ethiopia. Ethiop J Health Sci. 2012;22(1):7–18.

75. Khan AKA, Rashed MR, Banu G. A Study on the Usage Pattern of antimicrobial agents for the prevention of surgical site infections (ssis) in a tertiary care teaching hospital. J Clin Diagn Res. 2013;7(4):671–74.

76. Lawson EH, Hall BL, Ko CY. Risk factors for superficial vs deep/organ-space surgical site infections: implications for quality improvement initiatives. JAMA surgery. 2013 Sep 1;148(9):849-58.

77. Borgar MA, Rao V, Weisel RD, et al. Deep sternal wound infection : risk factors and outcomes.

78. Barie PS, Surgical site infections : epidemiology and prevention, Surg Infect (larcht),2002, Vol 3, Suppl. 1 (pg S9-21).

79. Maheshwari MK. A prospective study of surgical site infection in elective and emergency abdominal surgery in Cssh, Meerut. J Adv Res Biol Sci 2013;5:413-8.

80. Chauhan S, Chauhan B, Sharma H. Assessment of postoperative complications in emergency abdominal surgery in a tertiary care centre. Sch J App Med Sci 2017;5:1581-7.

81. Jain S, Shivhare R, Pardhan S, Chaurasiya D. A prospective study of post-operative surgical site infections after abdominal surgeries. International Surgery Journal. 2021 Sep 28;8(10):3088-92.

82. Janugade HB, Nagur BK, Sajjan KR, Biradar SB, Savsaviya JK, Reddy M. Abdominal surgical site infection occurrence and risk factors in krishna institute of medical science, Karad. Int J Sci Stud. 2016;3(11):53-6.

83. Kaur A, Sharma M, Singh K, Bansal D. Triclosan coated vicryl versus uncoated vicryl in preventing surgical site infection: a randomized controlled trial. International Surgery Journal. 2020 Dec 28;8(1):103-10.

84. Doherty G.M: Surgical Site Infections; in Complications in Surgery, eds Mulholand MW, Doherty GM (2006) p 114 – 125, Lippincott Williams Wilkins, Walnut Street, Philadelphia.

85. Xu Z, Qu H, Gong Z, Kanani G, Zhang F, Ren Y, Shao S, Chen X, Chen X. Risk factors for surgical site infection in patients undergoing colorectal surgery: A meta-analysis of observational studies. PloS one. 2021 Oct 28; 16(10):e0259107.

86. Alkaaki A, Al-Radi OO, Khoja A, Alnawawi A, Alnawawi A, Maghrabi A, Altaf A, Aljiffry M. Surgical site infection following abdominal surgery: a prospective cohort study. Canadian Journal of Surgery. 2019 Apr;62(2):111.

87. Dessie W, Mulugeta G, Fentaw S, Mihret A, Hassen M, Abebe E. Pattern of bacterial pathogens and their susceptibility isolated from surgical site infections at selected referral hospitals, Addis Ababa, Ethiopia. International journal of microbiology. 2016 Jun 30;2016.

ANNEXURE I. ETHICAL CLEARANCE.

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (India)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http://www.jnmc.edu
E-Mail : jnmc@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 264 Date: 24/12/2019

To

REG NO: BH0119008

PG student in Surgery
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A STUDY FOR ASSESSMENT OF RISK FACTORS AND INCIDENCE OF SURGICAL SITE INFECTION IN CASES OPERATED FOR PERITONITIS IN KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, BELGAUM"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Rohpa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

05

ANNEXURE II

WAIVER OF CONSENT

STUDY TITLE: **A study for assessment of risk factors and incidence of surgical site infection in cases operated for Peritonitis in KLE`S Dr. Prabhakar Kore Hospital and MRC, Belgaum.**

PRINCIPAL INVESTIGATOR

REG NO: BH0119008

Post Graduate Student

Department of General Surgery

Jawaharlal Nehru Medical College

Belagavi

The study will involve the review of medical records of patients who will undergo laparotomy from January 2020 to January 2021, and data will be collected from the data already recorded.

Waiver of informed consent is applicable to this research, because the study protocol meets all of the following criteria:

1. **The research presents no more than "minimal risk "of harm to subjects:** The study is observational; no intervention is being done. Hence the study results will not pose any risk to the patient.
2. **The waiver will not adversely affect the rights and welfare of the subjects:** The study will not affect the course of treatment. All investigations that will be collected is clinically and routinely indicated, therefore would be done regardless of the research. No study results would affect clinical decisions about the individual's care.

3. **The research could not be practicably carried out without the waiver:** taking a consent for a study in an emergency situation is not appropriate. Besides, a review of the medical records would not change the care that the individual will receive.
4. **Whenever appropriate, subject will be provided with additional pertinent information after participation:** since the results of the research would have no effect on the subjects. There is no anticipated benefit to the subject that that would change what has already occurred.

ANNEXURE III

PROFORMA

PROFORMA

- CASE NO.
- NAME
- AGE
- SEX
- ADDRESS
- IP NO.
- UNIT/WARD
- DATE OF ADMISSION
- DATE OF SURGERY
- DATE OF DISCHARGE
- CHIEF COMPLAINTS

  SYMPTOMS

  1) PAIN/TENDERNESS
  2) DISTENSION
  3) VOMITING
  4) CONSTIPATION
  5) FEVER
  6) SHOCK

- TIME TAKEN TO REACH HOSPITAL FROM SYMPTOM ONSET
  1) <24 HOURS
  2) >24 HOURS
- SITE OF PERFORATION IF ANY
- PAST HISTORY

  COMORBID CONDITIONS

  1) NSAID INTAKE
  2) HYPERTENSION

3) TB
4) DIABETES
5) MALIGNANCY
6) SMOKING
7) ALCOHOL INTAKE
8) OBESITY

- PERSONAL HISTORY

- TREATMENT HISTORY

- CLINICAL DIAGNOSIS

- GENERAL PHYSICAL EXAMINATION
    1) PALLOR
    2) EDEMA
    3) PULSE
    4) BP
    5) TEMPERATURE
- INVESTIGATIONS
    1) CBC
    2) RBS
    3) UREA
    4) CREATINE
    5) LFT
    6) XRAY ERECT ABDOMEN
    7) OTHERS

- PRE-OP ANTIBIOTICS
- PRE-OP OPRATION PREPARATION
- ADMISSION TO OPERATION TIME
    1) <12 HOURS
    2) >12 HOURS

- OPERATION DETAILS
    1) PROCEDURE
    2) TYPE OF SURGERY-ELECTIVE OR EMERGENCY
    3) DURATION OF SURGERY IN HOURS
    4) OPERATIVE FINDINGS
    5) OPERATIVE DIAGNOSIS
    6) DRAIN USED OR NOT
    7) IF ADDITIONAL ANTIBIOTIC GIVEN DURING OP
- POST OPERATIVE
    1) ANTIBIOTICS
    2) SIGNS OF WOUND INFECTION
    3) DAY OF WOUND INFECTION
    4) MANAGEMENT OF WOUND INFECTION
- WOUND CULTURE SWAB GROWTH
- ANTIBIOTIC SENSITIVITY/RESISTANCE

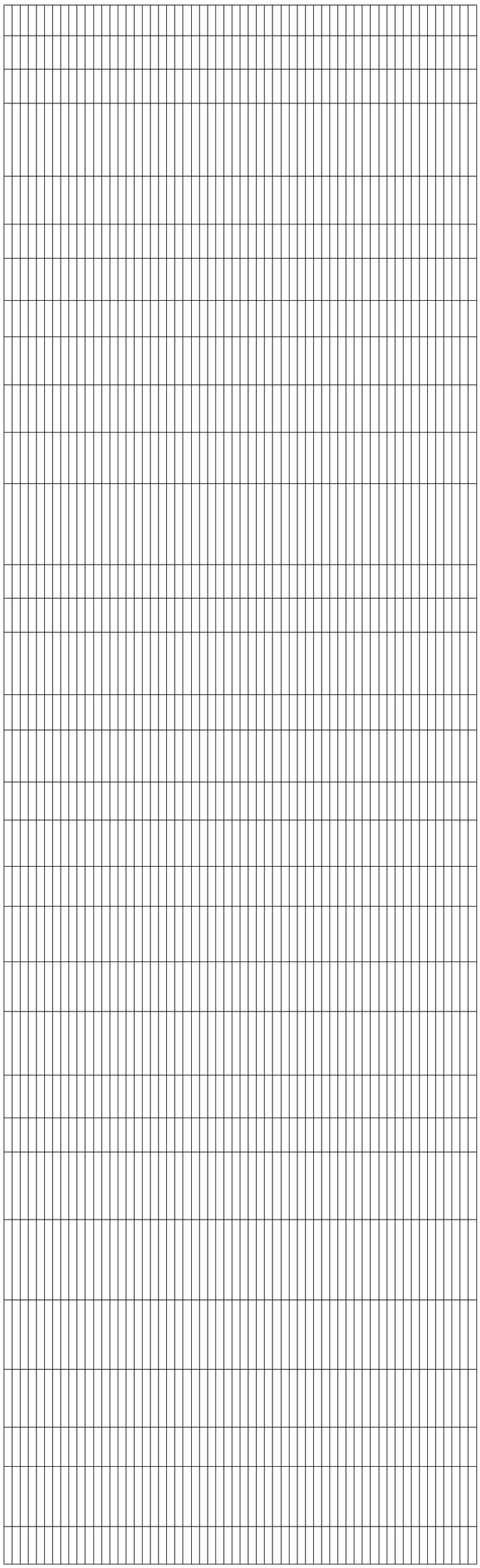
- FOLLOW UP

ANNEXURE IV- KEY TO MASTERCHART

Ip no.	In patient number
DOA	Date of admission
DOS	Date of surgery
DOD	Date of discharge
hrs	hours
DM	Diabetes mellitus
TB	Tuberculosis
BP	Blood pressure
HB	Haemoglobin
TC	Total white blood cell count
RBS	Random blood sugar
CEF-SUL	Cefoperazone-sulbactum
PIPZO	Piperacillin-tazobactam
MEZOL	Metronidazole
ORNIDA	Ornidazole
CLINDA	Clindamycin
TIGE	Tigecycline
AMI	Amikacin
FOS	Fosfomycin
IMIP	Imipenem
ERTAP	Ertapenem
Genta	Gentamycin

Sr no	Ip no	Age	Gender	Doa	Dos	Dod	Symptoms							Time taken to reach hospital		Comorbidities								General examination							
							pain	distension	GUARDING/RIGIDITY	vomiting	constipation	fever	shock	<24hrs	>24hrs	NSAID use	Hypertension	Dm	TB	Malignancy	Smoking	Alcohol	Obesity	Pallor	Edema	Pulse	Bp	Temp	HB	TC	RBS
1	1009472	35	MALE	13-04-2020	14-04-2020	26-04-2020	YES	YES	NO	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	YES	NO	NO	YES	NO	78	110/70	AFEBRILE	12.2	6800	
2	1008707	45	MALE	28-03-2020	30-03-2020	06-04-2020	YES	NO	NO	YES	NO	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	98	140/80	AFEBRILE	15.2	13000	81
3	1008687	70	FEMALE	27-03-2020	27-03-2020	02-04-2020	YES	NO	NO	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	80	120/80	AFEBRILE	11.7	11300	169
4	1001467	42	MALE	14-02-2020	14-02-2020	26-02-2020	YES	YES	YES	NO	NO	NO	YES	YES		NO	NO	NO	NO	YES (CA STOMACH)	YES	YES	NO	YES	YES	110	90/60	AFEBRILE	10.8	3200	301
5	1009514	19	MALE	14-04-2020	14-04-2020	27-04-2020	YES	NO	YES	YES	NO	YES	YES		YES	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	120	80/50	FEBRILE	14.3	5000	160
6	996117	44	FEMALE	17-01-2020	18-01-2020	27-01-2020	YES	YES	YES	NO	YES	NO	NO	YES		NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	102	150/100	AFEBRILE	12.5	9100	
7	999124	72	MALE	02-02-2020	05-02-2020	11-02-2020	YES	YES	YES	NO	NO	NO	NO		YES	NO	YES	YES	NO	NO	NO	NO	NO	YES	NO	98	140/80	AFEBRILE	11.1	8600	
8	992877	16	MALE	01-01-2020	01-01-2020	23-01-2020	YES	YES	YES	NO	YES	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	88	110/80	AFEBRILE	13.3	29600	69
9	999631	45	MALE	05-02-2020	05-02-2020	18-02-2020	YES	YES	YES	NO	NO	NO	YES		YES	NO	NO	NO	NO	NO	NO	YES	NO	YES	NO	130	70/60	AFEBRILE	11	6900	111
10	1043666	43	FEMALE	12-03-2021	12-03-2021	22-03-2021	YES	YES	YES	YES	NO	NO	NO		YES	NO	NO	NO	NO	HIV +STATUS	NO	NO	NO	YES	YES	74	110/70	AFEBRILE	8.5	16800	74
11	1040198	18	MALE	18-02-2021	18-02-2021	24-02-2021	YES	NO	YES	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	78	130/80	AFEBRILE	12.9	6500	118
12	1035044	50	MALE	12-01-2021	12-01-2021	24-01-2020	YES	YES	YES	NO	NO	NO	NO	YES		NO	NO	YES	NO	NO	YES	YES	NO	NO	YES	110	150/80	AFEBRILE	8.9	19900	
13	1035733	70	MALE	19-01-2021	20-01-2021	25-01-2021	YES	YES	YES	NO	NO	YES	YES		YES	NO	NO	NO	NO	HCV +STATUS	NO	NO	NO	NO	NO	128	90/70	FEBRILE	15.5	30700	95
14	1010118	55	FEMALE	26-04-2020	26-04-2020	04-05-2020	YES	YES	YES	YES	NO	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	80	140/60	AFEBRILE	10.4	13000	91
15	1013380	42	FEMALE	03-06-2020	05-06-2020	12-06-2020	YES	NO	YES	YES	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	80	110/70	AFEBRILE	12.4	12000	93
16	1008803	58	MALE	30-03-2020	31-03-2020	06-04-2020	YES	NO	NO	YES	YES	NO	NO		YES	NO	NO	NO	NO	YES(CA COLON)	NO	NO	NO	NO	NO	102	140/90	AFEBRILE	16.5	10000	111
17	1009258	40	FEMALE	08-04-2020	08-04-2020	12-04-2020	YES	YES	YES	YES	YES	YES	YES		YES	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	120	90/70	FEBRILE	9	8400	349
18	1017522	67	FEMALE	03-07-2020	03-07-2020	11-07-2020	YES	YES	YES	NO	YES	NO	NO	YES		NO	YES	YES	NO	NO	NO	NO	NO	YES	YES	120	170/110	FEBRILE	10.7	16000	269
19	998515	70	FEMALE	29-01-2020	30-01-2020	24-01-2020	YES	NO	YES	NO	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	120	90/60	AFEBRILE	10.2	9300	94
20	1033044	38	MALE	29-12-2021	07-01-2021	15-01-2021	YES	YES	YES	NO	NO	NO	NO		YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	110	110/60	AFEBRILE	12.8	9070	98
21	1033283	70	MALE	31-12-2020	31-12-2020	04-01-2021	YES	YES	YES	NO	YES	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	84	130/90	FEBRILE	12.6	14,100	91
22	1021794	49	MALE	27-08-2020	27-08-2020	06-09-2020	YES	NO	YES	YES	NO	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	96	110/70	AFEBRILE	9.9	13,600	114
23	1020872	35	MALE	13-08-2020	13-08-2020	21-08-2020	YES	YES	YES	YES	YES	NO	YES		YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	120	100/60	FEBRILE	12.6	15,600	120
24	1019145	40	FEMALE	19-07-2020	19-07-2020	27-07-2020	YES	NO	YES	YES	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	132	100/70	AFEBRILE	5.1	47,900	110
25	1013192	65	MALE	02-06-2020	02-06-2020	10-06-2020	YES	YES	YES	YES	NO	NO	YES		YES	NO	YES	NO	NO	NO	YES	YES	YES	NO	NO	122	110/70	AFEBRILE	17.5	16,100	98
26	1006816	32	MALE	13-03-2020	13-03-2020	26-03-2020	YES	NO	YES	NO	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	110	130/70	AFEBRILE	14	18,300	252
27	1013275	39	MALE	02-06-2020	03-06-2020	14-06-2020	YES	YES	YES	NO	YES	NO	NO		YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	80	120/80	FEBRILE	16	12,600	181
28	1014462	67	FEMALE	11-06-2020	12-06-2020	26-06-2020	YES	NO	YES	NO	YES	NO	NO		YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	100	170/90	AFEBRILE	13.6	11,600	145
29	1012450	20	MALE	26-05-2020	26-05-2020	15-06-2020	YES	NO	YES	YES	YES	YES	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	120	120/70	FEBRILE	13.2	9,800	148
30	1011192	60	MALE	12-05-2020	12-05-2020	19-05-2020	YES	NO	YES	NO	NO	NO	NO	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	88	110/70	AFEBRILE	13.2	8,200	84
31	1011017	47	MALE	09-05-2020	10-05-2020	19-05-2020	YES	NO	YES	YES	YES	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	100	110/80	AFEBRILE	13	13,700	142
32	1010832	62	FEMALE	07-05-2021	07-05-2021	14-05-2021	YES	NO	YES	YES	NO	YES	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	120	120/80	FEBRILE	10.9	9,600	109
33	1010794	21	MALE	06-05-2020	07-05-2020	26-05-2020	YES	NO	YES	NO	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	98	110/70	AFEBRILE	16.3	18,600	127
34	1010292	65	MALE	29-04-2020	29-04-2020	20-05-2020	YES	YES	YES	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	110	100/60	AFEBRILE	14.5	31,700	135
35	1010270	55	MALE	29-04-2020	29-04-2020	15-05-2020	YES	YES	YES	NO	NO	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	98	110/70	AFEBRILE	16.3	2,800	139
36	1009541	65	FEMALE	14-04-2020	15-04-2020	21-04-2020	YES	YES	NO	NO	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	90	110/70	AFEBRILE	13.8	10,800	160
37	997799	58	MALE	26-01-2020	26-01-2020	28-01-2020	YES	YES	YES	NO	NO	NO	YES		YES	NO	NO	NO	YES	NO	NO	NO	NO	YES	YES	122	90/60	AFEBRILE	10.4	38,200	154
38	1010117	24	MALE	26-04-2020	26-04-2020	15-05-2020	YES	YES	YES	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	90	130/80	AFEBRILE	12.4	19,200	118
39	1003017	53	FEMALE	23-02-2020	07-03-2020	14-03-2020	YES	YES	YES	YES	NO	YES	NO		YES	NO	NO	YES	NO	NO	NO	NO	YES	YES	YES	100	130/90	FEBRILE	11.5	12,800	124
40	999517	66	MALE	04-02-2020	07-02-2020	12-03-2020	YES	YES	YES	NO	YES	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	90	140/100	AFEBRILE	7.1	14,500	142
41	1016762	62	FEMALE	28-06-2020	30-06-2020	09-07-2020	YES	YES	YES	YES	NO	YES	NO		YES	NO	NO	YES	NO	NO	NO	NO	NO	YES	YES	112	110/70	AFEBRILE	8.9	2,400	56
42	1009279	27	MALE	09-04-2020	09-04-2020	11-04-2020	YES	YES	YES	YES	YES	NO	YES		YES	YES	NO	NO	NO	NO	YES	YES	NO	YES	YES	120	90/60	AFEBRILE	11.1	10,800	110
43	1009383	41	MALE	10-04-2020	11-04-2020	24-04-2020	YES	NO	YES	YES	NO	NO	NO		YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	YES	120	110/70	AFEBRILE	12.5	11,900	112
44	1013285	57	MALE	02-06-2020	02-06-2020	10-06-2020	YES	NO	YES	NO	YES	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	90	140/90	AFEBRILE	10.4	6,000	130
45	1009624	37	FEMALE	15-04-2020	16-04-2020	30-04-2020	YES	YES	YES	YES	NO	YES	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	120	110/70	FEBRILE	11.9	26,900	256

46	1026862	25	FEMALE	02-11-2020	14-11-2020	09-12-2020	YES	YES	YES	YES	YES	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	YES	YES	110	130/80	AFEBRILE	10.1	16,600	80
47	1027422	54	MALE	08-11-2020	09-11-2020	20-11-2020	YES	YES	YES	NO	YES	NO	NO		YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	112	120/80	AFEBRILE	14	8,400	320
48	1026315	66	MALE	26-10-2020	27-10-2020	01-11-2020	YES	YES	YES	YES	YES	YES	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	120	80/50	FEBRILE	8.9	17,900	65
49	1025007	22	FEMALE	09-10-2020	09-10-2020	29-10-2020	YES	YES	YES	YES	YES	YES	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	110	110/70	AFEBRILE	9	19,500	109
50	1022912	65	FEMALE	10-09-2020	24-09-2020	04-10-2020	YES	YES	YES	YES	YES	YES	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	YES	YES	100	110/70	AFEBRILE	10.5	16,300	135
51	1022664	35	FEMALE	07-09-2020	07-09-2020	15-09-2020	YES	YES	YES	NO	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	130	110/70	AFEBRILE	12.5	15,700	76
52	1019776	25	MALE	28-07-2020	28-07-2020	07-08-2020	YES	YES	YES	NO	NO	YES	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	124	100/60	FEBRILE	14.1	6,000	133
53	1019353	24	MALE	22-07-2020	22-07-2020	31-07-2020	YES	YES	YES	YES	NO	NO	NO	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	100	130/80	AFEBRILE	11.9	14,500	77
54	1013807	63	MALE	07-06-2020	07-06-2020	15-06-2020	YES	YES	YES	NO	NO	NO	NO	YES		YES	YES	YES	NO	NO	NO	NO	YES	NO	NO	80	120/80	AFEBRILE	10.2	10,800	121
55	1013630	55	FEMALE	05-06-2020	06-06-2020	10-06-2020	YES	YES	YES	YES	NO	NO	NO		YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	88	130/70	AFEBRILE	13.5	8,500	150
56	1011080	45	FEMALE	11-05-2020	14-05-2020	19-05-2020	YES	YES	YES	YES	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	110	110/70	AFEBRILE	7.1	8,100	100
57	1019316	65	FEMALE	21-07-2020	21-07-2020	01-08-2020	YES	YES	YES	YES	YES	YES	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	110	110/70	AFEBRILE	14	18,000	142
58	1009703	60	MALE	17-04-2020	17-04-2020	25-04-2020	YES	YES	YES	YES	YES	NO	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	130	90/60	AFEBRILE	8.6	24500	122
59	1020422	60	MALE	06-08-2020	07-08-2020	20-08-2020	YES	YES	YES	NO	YES	NO	NO		YES	YES	NO	NO	NO	NO	NO	YES	NO	NO	YES	110	150/90	AFEBRILE	13.3	21000	71
60	1025009	22	MALE	09-10-2020	09-10-2020	19-10-2020	YES	NO	YES	NO	NO	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	100	130/90	AFEBRILE	13	11000	111
61	1026099	62	MALE	23-10-2020	23-10-2020	31-10-2020	YES	YES	YES	NO	NO	YES	NO		YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	YES	130	150/80	FEBRILE	12.9	22000	122
62	1001106	45	MALE	12-02-2020	13-02-2020	21-02-2020	YES	YES	YES	NO	YES	NO	NO		YES	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	110	110/70	AFEBRILE	8.4	16300	146
63	1020059	55	MALE	31-07-2020	31-07-2020	11-08-2020	YES	YES	YES	NO	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	135	110/70	AFEBRILE	9.4	2100	129
64	1011261	38	MALE	11-05-2020	11-05-2020	18-05-2020	YES	YES	YES	YES	YES	YES	NO		YES	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	110	120/70	AFEBRILE	19	9400	280
65	1042081	20	MALE	02-03-2021	02-03-2021	10-03-2021	YES	YES	YES	YES	NO	NO	NO	YES		NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	106	120/70	AFEBRILE	15	10800	105
66	998602	45	MALE	30-01-2020	31-01-2020	08-02-2020	YES	YES	YES	NO	NO	NO	NO		YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	84	160/90	AFEBRILE	15	8400	144
67	1033087	36	MALE	29-12-2020	29-12-2020	11-01-2020	YES	NO	YES	NO	NO	NO	NO	YES		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	90	130/80	AFEBRILE	13.8	11,400	87
68	1031032	55	FEMALE	11-12-2020	11-12-2020	19-12-2020	YES	YES	YES	YES	NO	NO	NO		YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	90	110/70	AFEBRILE	8.2	20,500	114
69	1026980	19	MALE	03-11-2020	04-11-2020	11-11-2020	YES	YES	YES	YES	YES	NO	NO		YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	88	130/80	AFEBRILE	16.2	18200	250
70	1008694	61	FEMALE	28-03-2020	28-03-2020	17-04-2020	YES	YES	YES	YES	YES	NO	NO		YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	120	110/60	AFEBRILE	14.5	14500	100
71	1006130	42	MALE	09-03-2020	10-03-2020	24-03-2020	YES	YES	YES	YES	YES	NO	NO		YES	YES	NO	NO	NO	NO	YES	YES	NO	NO	NO	120	140/80	AFEBRILE	14.7	5100	140
72	1029072	46	MALE	24-11-2020	24-11-2020	30-11-2020	YES	NO	YES	NO	NO	YES	NO		YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	110	130/80	AFEBRILE	15.6	14800	102
73	1026715	65	FEMALE	31-10-2020	31-10-2020	20-11-2020	YES	YES	YES	NO	NO	YES	YES		YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	104	90/60	FEBRILE	14.5	9500	155
74	1026710	64	MALE	30-10-2020	31-10-2020	08-11-2020	YES	YES	YES	YES	YES	YES	YES		YES	YES	YES	YES	NO	NO	YES	YES	NO	NO	NO	110	100/70	AFEBRILE	15.6	9800	225
75	1006897	35	FEMALE	14-03-2020	14-03-2020	24-03-2020	YES	YES	YES	YES	NO	NO	YES	YES		NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	115	80/50	AFEBRILE	10.6	7900	130



Investigations			Pre-op antibiotics	Admission to operation time		Operation details							Postoperative findings					Wound culture	Antibiotic sensitivity	Follow up
S. Urea,S. creat	Sr. Albumin	Site of perforation	Yes/No	<12 hrs	> 12 hrs	Procedure	Type of surgery (emergency/ elective)	Duration surgery	Operative finding	Diagnosis	Drain(Yes/No)	Repeat antibiotics intraop (Yes/No)	Antibiotics	Signs of wound infection	SOUTHAMPTONS WOUND GRADING	Day of wound infection	Management of wound infection			
28/0.8	3.4	NIL	YES	YES		ADHESIOLYSIS	EMERGENCY	3HOURS	ADHESIONS ,MULTIPLE STRICTURES	SMALL BOWEL OBSTRUCTION	YES	NO	CEF-SUL , MEROPENEM	PUS DISCHARGE	GRADE 4	DAY 5	REGULAR DRESSING	E.COLI	TIGE/ami/fos/imip/mero/colistin/ertap/genta	30 DAYS
15/1.03	4	NIL	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	4 HOURS	MECKEL'S DIVERTICULUM	SMALL BOWEL OBSTRUCTION	YES	NO	PIPZO,MEZOL	NO						30 DAYS
33/0.3	4.4	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	GANGRENOUS BOWEL	SMALL BOWEL OBSTRUCTION	YES	NO	PIPZO,MEZOL	NO						30 DAYS
29/0.75	3.1	GASTRIC PERFORATION	YES	YES		SUBTOTAL GASTRECTOMY	EMERGENCY	5 HOURS	GASTRIC PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
98/3.87	2.5	ILEAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	ILEAL PERFORATION WITH PERITONITIS	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,MEZOL	PUS DISCHARGE	GRADE 4	DAY 5	SUTURE REMOVAL,REGULAR DRESSING	E.COLI	TIGE/FOS	30 DAYS
10/0.62	4.6	NIL	YES	YES		ADHESIOLYSIS	EMERGENCY	4HOURS	ADHESIONS	IRREDUCIBLE INCISIONAL HERNIA	NO	NO	CEFTRIAXONE,ORNIDA,PIPZO	NO						30 DAYS
39/1.5	3.8	SIGMOID DIVERTICULAR PERFORATION	YES		YES	RESECTION AND ANASTOMOSIS ILEOSTOMY	ELECTIVE	4 HOURS	DIVERTICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEF-SUL , ORNIDA	HEMOSEROUS DISCHARGE	GRADE 3	DAY 3	REGULAR DRESSING	ENTEROBACTER CLOACAE	TIGE/FOS/IMIP/GENTA	30 DAYS
55/0.6	2.2	JEJUNAL PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	GANGRENOUS APPENDIX,SLOUGHED OUT JEJUNUM	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,MEZOL	PUS DISCHARGE	GRADE 4	DAY 5	PUS DISCHARGE	E.COLI	TIGE/AMI/FOS/IMIP/MEROPENEM/COLISTIN/TETRACYCLINE/ERTAP	30 DAYS
75/2.04	2.9	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	MECKELS DIVERTICULUM	SMALL BOWEL OBSTRUCION	YES	YES	MEROPENEM,CLINDA	NO						30 DAYS
108/3.16	2.5	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	3 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,CLINDA	NO						30 DAYS
16/0.62	3.2	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	3 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,ORNIDA	NO						30 DAYS
24/0.91	2.3	ILEAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	ILEAL PERFORATION WITH PERITONITIS	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	WOUND GAPING ,PUS DISCHARGE	GRADE 4	DAY 3	DAILY DRESSING	E.COLI	FOS/COLISTIN/TIGE	30 DAYS
40/1.43	3.5	GALL BLADDER PERFORATION	YES	YES		CHOLECYSTECTOMY	EMERGENCY	2 HOURS	GALL BLADDER PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,CLINDA	NO						30 DAYS
23/0.80	3.1	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	GANGRENOUS ILEAL SEGMENT	SMALL BOWEL OBSTRUCION	YES	NO	PIPZO,MEZOL	NO						30 DAYS
18/0.58	3.4	NIL	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	4 HOURS	GANGRENOUS JEJUNUM	SMALL BOWEL OBSTRUCION	YES	NO	CEF-SUL,MEZOL	NO						30 DAYS
21/0.92	3.3	NIL	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	4 HOURS	GROWTH IN ASCENDING COLON	INTESTINAL OBSTRUCION	YES	NO	MEROPENEM,ORNIDA	NO						30 DAYS
28/0.47	3.5	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	3 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
21/0.64	2.4	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	3 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO , MEZOL	HEMOSEROUS DISCHARGE	GRADE 3	DAY 5	DAILY DRESSING	MRSA	CHLORAMPHENICOL/LEVOFLOXACIN/TETRACYCLINE/CIPLOX/GENTAMICIN	30 DAYS
29/0.64	2.3	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,ORNIDA	NO						
118/2.3	2.3	PELVIC ABSCESS + APPENDICULAR PERFORATION	YES	NO	YES	APPENDICECTOMY	ELECTIVE	3 HOURS	PELVIC ABSCESS + ADHESIONS	PERFORATIVE PERITONITIS	YES	NO	CEFTRIAXONE , MEZOL, AMIKACIN	PUS DISCHARGE	GRADE 4	DAY 9	SUTURE REMOVAL,DAILY DRESSING	KLEBSIELLA OXYTOCA	TETRACYCLINE	30 DAYS
53/0.86	2.5	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	PRE PYLORIC PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO, ORNIDA	NO						30 DAYS
23/0.93	3	NIL	YES	YES		MESENTERIC REPAIR	EMERGENCY	3 HOURS	MESENTERIC TEAR	RTA- MESENTERIC TEAR HEMATOMA	YES	YES	CEFTRIAXONE , MEZOL,	NO						30 DAYS
88/2.41	2.4	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	3 HOURS	DECOMPRESSIVE ENTEROTOMY	SMALL BOWEL OBSTRUCTION	YES	NO	MEROPENEM , ORNIDA	PUS DISCHARGE	GRADE 4	DAY 3	REGULAR DRESSING	E COLI	TIGECYCLINE, FOSFOMYCIN	30 DAYS
49/2.45	2.1	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	GANGRENOUS BOWEL	SMALL BOWEL OBSTRUCTION	YES	YES	PIPZO, ORNIDA	NO						30 DAYS
69/1.44	3.1	GASTRIC PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	GASTRIC PERFORATION	PERFORATIVE PERITONITIS	NO	NO	PIPZO, ORNIDA	NO						30 DAYS
34/0.32	2.8	ILEAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	ILEAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEFPARAZONE, ORNIDA	NO						30 DAYS
17/1.7	2.6	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO, MEZOL, AMIKACIN	NO						30 DAYS
33/0.78	3.4	NIL	YES		YES	RESECTION AND ANASTOMOSIS	EMERGENCY	3 HOURS	LADDS BAND RELEASE WITH RESECTION SIDE TO SIDE ANASTOMOSIS	INTESTINAL OBSTRUCION	YES	YES	CEFTRIAXONE , ORNIDA	NO						30 DAYS
26/0.83	3.8	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEF-SUL,MEZOL	PUS DISCHARGE	GRADE 4	DAY 3	SUTURE REMOVAL,REGULAR DRESSING	PSEUDOMONAS	IMIP/MEROPENEM/AMI/GENTA	30 DAYS
21/0.84	3.1	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFOARTION	PERFORATIVE PERITONITIS	YES	NO	CEF-SUL,ORNIDA	NO						30 DAYS
24/0.87	4.7	NIL	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	5 HOURS	ISCHEMIC JEJUNAL SEGMENT	STRANGULATED HERNIA	YES	NO	CEF-SUL,MEZOL	NO						30 DAYS
54/1.06	2.9	APPEDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	4 HOURS	SUBHEPATIC COLLECTION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL,GENTA	NO						30 DAYS
25/1.02	4.2	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	4 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
363/3.46	3.4	NIL	YES		YES	RESECTION AND ANASTOMOSIS	EMERGENCY	3 HOURS	GANGRENOUS JEJUNAL SEGMENT	STRANGULATED HERNIA	YES	NO	PIPZO,MEZOL	NO						30 DAYS
25/0.91	4.1	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
96/1.95	3.8	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	GANGRENOUS BOWEL	STRANGULATED HERNIA	YES	NO	CEF-SUL,MEZOL	PUS DISCHARGE	GRADE 4	DAY 5	SUTURE REMOVAL,DAILY DRESSING	E.COLI	GENTA/MEROPENEM/IMIP/AMI	30 DAYS
94/1.46	3.1	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	GANGRENOUS BOWEL	STRANGULATED HERNIA	YES	YES	IMIPENEM,CLINDA	NO						30 DAYS
21/0.80	4.2	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	OBSTRUCTION SECONDARY TO BANDS	SMALL BOWEL OBSTRUCTION	YES	YES	CEF-SUL,MEZOL	NO						30 DAYS
18/0.55	2.8	ILEAL PERFORATION	YES		YES	RESECTION AND ILEOSTOMY	ELECTIVE	5 HOURS	GANGRENOUS BOWEL,ILEAL PERFORATION AND ISCHEMIA SMA THROMBOSIS	PERFORATIVE PERITONITIS	YES	YES	MEROPENEM,MEZOL	PUS DISCHARGE,WOUND GAPING	GRADE 4	DAY 5	SUTURE REMOVAL	KLEBSIELLA	COLISTIN	30 DAYS
22/0.74	3.7	ASCENDING COLON PERFORATION	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	5 HOURS	HEPATIC FLEXURE PERFORATION	PERFORATIVE PERITONITIS	YES	YES	CEFTRIAXONE,MEZOL	NO						30 DAYS
47/0.60	2.9	NIL	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	5 HOURS	GANGRENOUS BOWEL	SMALL BOWEL OBSTRUCTION	YES	YES	MEROPENEM,MEZOL	PUS DISCHARGE	GRADE 4	DAY 7	SUTURE REMOVAL,DAILY DRESSING	KLEBSIELLA PNEUMONIAE	TIGECYCLINE/IMIP/COLISTIN	30 DAYS
72/1.81	2.5	JEJUNAL PERFORATION	YES	YES		PRIMARY REPAIR OF PERFORATION	EMERGENCY	2 HOURS	JEJUNAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,CLINDA	NO						30 DAYS
40/0.83	2.9	ILEAL PERFORATION	YES	YES		PRIMARY REPAIR OF PERFORATION	EMERGENCY	3 HOURS	ILEAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEFTRIAXONE,MEZOL	PUS DISCHARGE	GRADE 4	DAY 3	SUTURE REMOVAL,DAILY DRESSING	KLEBSIELLA OXYTOCA	TIGECYCLINE, FOSFOMYCIN	30 DAYS
55/1.92	3.7	APPENDICULAR PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	GANGRENOUS APPENDIX,SLOUGHED OFF DITAL ILEUM	PERFORATIVE PERITONITIS	YES	YES	CEF-SUL,MEZOL	NO		DAY 3	SUTURE REMOVAL,DAILY DRESSING	E.COLI	TIGECYCLINE/AMI/FOS/IMIP/MEROPENEM/COLISTIN/ERTAP	30 DAYS
26/0.47	3.3	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	3 HOURS	APPENDICULAR ABSCESS	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM ,MEZOL	INTRA ABDOMINAL LEAK	GRADE 5	DAY 4	SUTURE REMOVAL,DAILY DRESSING,DRAIN OUTPUT MONITORING	E.COLI	TIGECYCLINE, FOS,AMI,AMOXYCLAV, CIPLOX,CEFUROXIME, MERPENEM,LEVOFLOX	30 DAYS

166/4.13	2.7	ILEAL PERFORATION	YES		YES	RESECTION AND END ILEOSTOMY	ELECTIVE	5 HOURS	MULTIPLE ILEAL PERFORATION,SLOUGHED OUT ILEUM	PERFORATIVE PERITONITIS	YES	YES	PIPZO,MEZOL	NO						30 DAYS
88/1.49	2.5	ASCENDING COLON PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	INTRA ABDOMINAL ABSCESS	PERFORATIVE PERITONITIS	YES	NO	TIGECYCLINE	NO						30 DAYS
51/2.08	2.8	CAECAL PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	POLYPOIDAL GROWTH I N ASCENDING COLON,CAECAL PERFORATION	PERFORATIVE PERITONITIS	YES	YES	TIGECYCLINE	NO						30 DAYS
36/0.35	1.8	SIGMOID DIVERTICULAR PERFORATION	YES		YES	RESECTION AND END COLOSTOMY	ELECTIVE	5 HOURS	RECTOVESICAL FISTULA ,SIGMOID COLON PERFORATION	PERFORATIVE PERITONITIS	YES	YES	CEF-SUL,CLINDA	PUS DISCHARGE	GRADE 4	DAY 3	SUTURE REMOVAL, DAILY DRESSING	ENTEROBACTER SPECIES	TIGECYCLINE/GENTA/COTRIMOXAZOLE	30 DAYS
42/0.86	1.2	ASCENDING COLON PERFORATION	YES		YES	RESECTION AND ANASTOMOSIS	ELECTIVE	5 HOURS	DIVERTICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	YES	CEF-SUL,CLINDA	PUS DISCHARGE	GRADE 4	DAY 3	SUTURE REMOVAL, DAILY DRESSING	ENTEROBACTER CLOACAE	TIGECYCLINE/PIPZO/COTRIMOXAZOLE	30 DAYS
27/0.87	3.9	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
23/0.85	3.2	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEF-SUL,MEZOL	NO						30 DAYS
23/0.88	4	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	2 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEFTRIAXONE,MEZOL	NO						30 DAYS
37/2.21	3.3	GASTRIC PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	GASTRIC PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEFTRIAXONE,MEZOL	NO						30 DAYS
17/0.57	3	NIL	YES		YES	ADHESIOLYSIS	ELECTIVE	3 HOURS	MULTIPLE ADHESIONS	SMALL BOWEL OBSTRUCTION	YES	NO	CIPLOX,MEZOL	NO						30 DAYS
13/0.55	3.2	NIL	YES		YES	RESESCTION AND ANASTOMOSIS	ELECTIVE	3 HOURS	MULTIPLE ADHESIONS ,BOWEL GANGRENE	SMALL BOWEL OBSTRUCTION	YES	NO	PIPZO,MEZOL	NO						30 DAYS
26/0.72	2.8	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	3 HOURS	GANGRENOUS BOWEL	SMALL BOWEL OBSTRUCTION	YES	NO	PIPZO,MEZOL	NO						30 DAYS
28/0.75	3.4	NIL	YES	YES		RESECTION AND JEJUNOSTOMY	EMERGENCY	3 HOURS	GANGRENOUS ILEUM	SMALL BOWEL OBSTRUCTION	YES	NO	PIPZO,CLINDA	NO						30 DAYS
34/0.64	2.4	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,CLINDA	NO						30 DAYS
17/0.83	4.7	ASCENDING COLON PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	ASCENDING COLON PERFORATION	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM ,MEZOL	NO						30 DAYS
96/1.15	2.7	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	GANGRENOUS APPENDIX	PERFORATIVE PERITONITIS	YES	NO	MEROPENEM,MEZOL	NO						30 DAYS
17/0.98	3.9	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	PERFORATED APPENDIX	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
125/2.24	3.2	ILEAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	4 HOURS	ILEAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
13/0.8	4.5	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
24/0.95	4	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	PUS DISCHARGE	GRADE 4	DAY 5	SUTURE REMOVAL, DAILY DRESSING	E.COLI	GENTAMYCIN/AMOXYCLAV/LEVOFLOXACIN/MEROPENEM/PIPZO/IMIP/AMI/TETRACYCLINE	30 DAYS
15/1.0	4.1	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
18/0.84	4.4	TRANSVERSE COLON PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	TRANSVERSE COLON PERFORATION,HEMOPERITONEUM	PENETRATING INJURY TO ABDOMEN	YES	NO	MEROPENEM,CLINDA							30 DAYS
28/0.81	3.6	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORAtIVE PERITONITIS	YES	NO	CIPLOX,MEZOL	NO						30 DAYS
19/1.0	4	NIL	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	2 HOURS	GANGRENOUS BOWEL	SMALL BOWEL OBSTRUCTION	YES	NO	CEF-SUL,MEZOL	NO						30 DAYS
111/2.98	2.6	JEJUNAL PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	4 HOURS	JEJUNAL PERFORATION WITH INFLAMMED BOWEL	PERFORATIVE PERITONITIS	YES	YES	PIPZO,MEZOL	HEMOSEROUS DISCHARGE	GRADE 3	DAY 5	DAILY DRESSING	STAPHYLOCOCCUS EPIDERMIDIS	RIFAMPIN	30 DAYS
47/0.98	3.2	GASTRIC PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	GASTRIC PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,CLINDA	NO						30 DAYS
37/1.53	4.2	APPENDICULAR PERFORATION	YES	YES		APPENDICECTOMY	EMERGENCY	2 HOURS	APPENDICULAR PERFORATION	PERFORATIVE PERITONITIS	YES	NO	CEF-SUL,MEZOL	NO						30 DAYS
47/2.0	2.6	SIGMOID DIVERTICULAR PERFORATION	YES	YES		RESECTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	SIGMOID DIVERTICULAR PERFORATION	PERFORAIVE PERITONITIS	YES	YES	PIPZO,MEZOL	NO						30 DAYS
26/0.93	3.8	DUODENAL PERFORATION	YES	YES		PRIMARY CLOSURE OF PERFORATION	EMERGENCY	3 HOURS	DUODENAL PERFORATION	PERFORATIVE PERITONITIS	YES	NO	PIPZO,MEZOL	NO						30 DAYS
32/0.76	2.5	JEJUNAL PERFORATIONS	YES	YES		RESESCTION AND ANASTOMOSIS	EMERGENCY	5 HOURS	MUTIPLE JEJUNAL PERFORATIONS	PERFORATIVE PERITONITIS	YES	YES	PI[PZO,MEZOL	PUS DISCHARGE	GRADE 4	DAY 5	DAILY DRESSING	CANDIDA SPECIES ISOLATED		30 DAYS