DISSERTATION ON

BACTERIOLOGICAL PROFILE, ANTIBIOGRAM AND RISK FACTORS OF SURGICAL SITE INFECTIONS IN A TERTIARY CARE HOSPITAL

Dissertation submitted in partial fulfillment of the

Requirement for the award of the Degree of

M.D. MICROBIOLOGY (BRANCH IV)

TRICHY SRM MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE

IRUNGALUR, TRICHY- 621 105

Affiliated To

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled, **"BACTERIOLOGICAL PROFILE, ANTIBIOGRAM AND RISK FACTORS OF SURGICAL SITE INFECTIONS IN A TERTIARY CARE HOSPITAL"** by **Dr.G.DHANALAKSHMI**, Post graduate in Microbiology (2016-2019), is a bonafide research work carried out under our direct supervision and guidance and is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai, for M.D. Degree Examination in Microbiology, Branch IV, to be held in May 2019.

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DECLARATION

I solemnly declare that the dissertation titled **"Bacteriological Profile, Antibiogram and Risk Factors of Surgical site infections in a Tertiary care hospital"** is bonafide record of work done by me during the period of**May 2017 to April 2018** under the guidance of Professor and HOD **DR.A.UMA, M.D.,** Department of Microbiology, Trichy SRM Medical College Hospital and Research Institute, Trichy.

The dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University in partial fulfillment of the requirement for the award of M.D Degree (Branch IV) in Microbiology.

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ANNEXURE- I

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INSTITUTIONAL ETHICS COMMITTEE CERTIFICATE.

Ref.No: CMCH&RC/IEC –**No 28- :11.04.2017**
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The research proposal submitted by **Dr. G. Dhanalakshmi**- I year post graduate, Department of MD Microbiology, Chennai Medical College Hospital & Research Centre, was discussed by the Institutional Ethics Committee of the CMCH&RC. The committee approved the research project subject to existing rules and regulations.

Title of the Research work/Project: Bacteriological profile antibiogram and risk factors of surgical site infections in a tertiary care hospital.

a. She should abide to the Ethical aspects of the institutional Ethics Committee.
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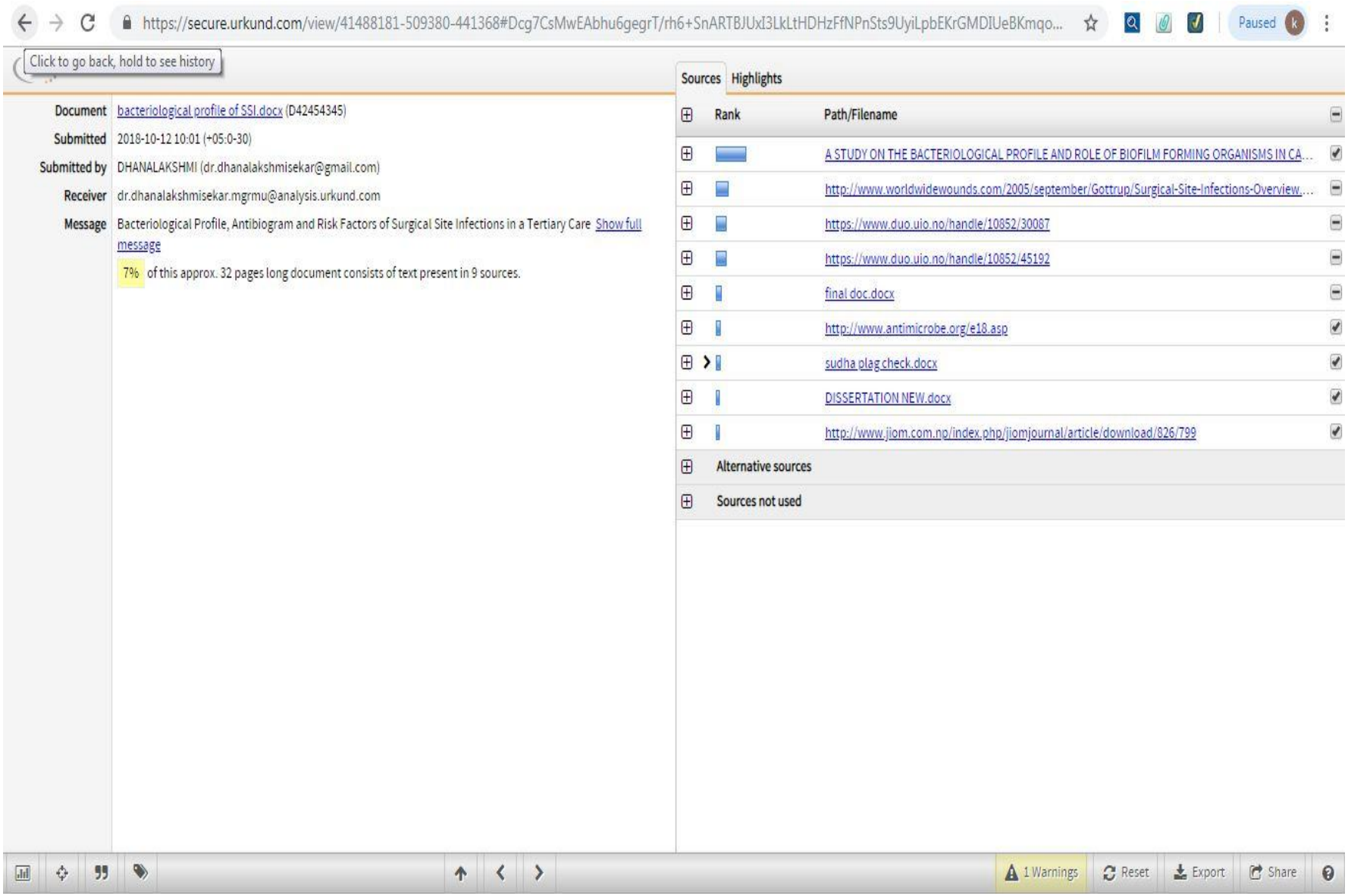
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CERTIFICATE – II

This is to certify that this dissertation work titled **"BACTERIOLOGICAL PROFILE, ANTIBIOGRAM AND RISK FACTORS OF SURGICAL SITE INFECTIONS IN A TERTIARY CARE HOSPITAL"**of the candidate **Dr. G. DHANALAKSHMI** with registration Number **201614602** is for the award of **M.D.MICROBIOLOGY** in the branch of IV. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **7%** percentage of plagiarism in the dissertation.

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I humbly submit this work to **ALMIGHTY**, who has given me the strength, endurance and ability to overcome the difficulties encountered in the process of compilation of my dissertation work.

I wish to express my sincere thanks to our **DEAN Dr. A. Jesudoss M.S., D.L.O.,** Trichy SRM Medical College Hospital and Research Centre, Trichy**,** for permitting me to use the resources of this Institution for my study.

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Special thanks to my senior postgraduates Dr. Shalini, Dr. Jahappriya and Dr. Jane esther and my friend Dr. Anand who helped in my works and for their support. I would also wish to thank my junior postgraduates for their help.

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CONTENTS

APPENDIX – I
ABBREVIATIONS

SSI - Surgical site Infections

NNIS - National Nosocomial infection surveillance program

PBP - Penicillin binding protein

MMM - Mannitol motility medium

TSI – Triple sugar iron

IEC – Institutional ethical committee

CLSI – Clinical Laboratory Standard Institute

CoNS – Coagulase negative *Staphylococcus*

MR – Methyl Red test

VP – VogesProskauer test

ATCC -American Type Culture Collection

MHA - Muller Hinton Agar

MRSA – Methicillin resistant *Staphylococcus aureus*

MSSA – Methicillin sensitive *Staphylococcus aureus*

MBL – Metallobetalactamases

MRM – Modified Radical Mastectomy

LSCS - Lower Segment Caesarean Section

ORIF – Open Reduction and Internal Fixation

IOL – Intraocular lens implantation

URSL - Uretheroscopic lithotripsy

TURP - Transurethral Resection of Prostate

ASA – American society of anesthesiologists

CDC – Center for Disease Control

SENIC – Study on Efficacy of Nosocomial Infection Control

HIV - Human Immunodeficiency Virus

TEM – Temoneira

SHV - sulphhydryl in variable

MIC – Minimum Inhibitory Concentration

CTX - Cefotaxime

CTR - Ceftriaxone

CAZ - Ceftazidime

EDTA – Ethylenediaminetetraacetic acid

MHT - Modified Hodge Test

ESBL - Extended spectrum betalactamase

MDR -Multi Drug Resistance

AST - Antimicrobial Susceptibility Testing

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1.0. **INTRODUCTION**

The infection of a wound can be defined as the invasion of organisms through tissues following a breakdown of local and systemic host defences, leading to cellulitis, lymphangitis, abscess and bacteraemia. Infections of surgical wounds are called as surgical site infections (SSIs).[1]

SSIs are defined as infections occurring within 30 days after a surgery or within one year if an implant is left in place after the procedure and affecting either the incision or deep tissue at the operation site[2].

According to the National Nosocomial Infection Surveillance program (NNIS), it is classified into superficial, deep, organ/space infections[3].

Source of SSIs include the patient's own normal flora, organisms present in the hospital environment that are introduced into the patient by medical procedures, specific underlying disease, trauma or burns which may cause a mucosal or skin surface interruption.[4]

SSIs are serious operative complications that occur in approximately 2% of surgical procedures and account for 20% of health care-associated infections. Many studies reported that SSIs rank third among common nosocomial infection next only tourinary tract and respiratory tract infections.[2,6]

Recent studies reported that SSI rate ranges from 19.4% to 36.5% [7]all over the world, whereas in India it ranges from 3% to 12%.[8,9]

SSI remains a common and widespread problem that contributes to significant morbidity and mortality, prolongs hospital stay and consequently increasing health care cost

Factors which promote SSIs include length of hospital stay, Obesity, Diabetes mellitus, smoking etc..The development of a post operative wound infection depends on the complex interplay of many factors. Most postoperative wounds are endogenous. Exogenous infections are mainly acquired from the nose or skin flora of the operating team and transmitted through the hands of the surgeon or improper operation theatre steriliation[10]which includes pre operative, intra operative and post operative care

Some significant factors that can influence the incidence of subsequent infection are surgical techniques, skin preparation, timing, method of wound closure and antibiotic prophylaxis after certain types of surgery. Also many other factors have been identified as having an effect on the potential for infection and these should be considered by the healthcare professionals before, during and after surgery.[11]

Table no.1. Common causes of SSIs:

Gram positive organisms	Gram negative organisms
Staphylococcus aureus *CONS* *Enterococci*	*Eschericia coli* *Klebsiella spp* *Proteus spp* *Enterobacter spp* *Pseudomonas spp* *Acinetobacter spp*

The resistance offered by a microbe to antimicrobial agent that is used in the prevention or treatment of infections is called antimicrobial resistance.[12]Beta -lactams are the most widely used antibiotics for treatment of postoperative woundsdue to their broad spectrum of activity, safety profile and proven clinical efficacy.[13]There are

different mechanisms which cause resistance to beta lactams namely a reduction in the affinity of the drug targets (penicillin binding proteins) via amino-acid substitution, a phenomenon occurring in both gram positive and gram negative bacteria. Gram negative species, alteration in outer-membrane permeability that prevents passage to the beta lactams and in both Gram-positive and Gram-negative bacteria, the production of beta lactamase that inactivate the drug through hydrolysis of the beta lactam ring. Hence widespread use of these groups of antibiotics has lead to emergence and rapid spread of resistance.[14]

Among the members of the Enterobacteriaceae family, resistance to β lactams has been reported to be associated with ESBL and Amp C β- lactamase.[15] ESBL producing organisms hydrolyze oxyamino β- lactams like Cefotaxime, Ceftriaxone, Ceftazidime and Monobactams but have no effect on Cephamycins, Carbapenems and related compounds.[16]

Production of β- lactamase is frequently plasmid encoded and bears clinical significance. Plasmids responsible for ESBL and Amp C β- lactamase production frequently carry genes encoding resistance to other drugs also and therefore antibiotic options in the treatment of β- lactamase producing organisms are extremely limited.[17]

Data from last few decades show an increasing resistance for drugs that were considered as the first line of treatment for post-operative wound infections.[18]The most frequent co-resistances which are found in ESBL producing organisms are amino glycosides, tetracyclines, chloramphenicol, trimethoprim-sulfamethoxazole and fluoroquinolones. To stress precise empirical therapy, antibiotic

policies should be implemented to reduce hospital length of stay, morbidity and expenditure per day in the hospital.[19]

The carbapenemases are betalactamases that are capable of inactivating or hydrolyzing the carbapenem group of betalactam antibiotics. This is the main cause of carbapenem resistance in gram negative bacilli. Hyperproduction of enzymes called Amp C betalactamases can also result in resistance to carbepenem.[20]

The isolates which showed resistance to at least three or more than three groups of antibiotics were considered as multi drug resistant (MDR).

The prevalence of antimicrobial resistance pattern may vary between geographical areas. However, the publications available on the susceptibility pattern of bacterial isolates causing SSI and ESBL prevalence in South India are minimal. Hence, the present study is under taken at Trichy SRM Medical College and Research Centre situated at Irungalur, Trichy in India, which is a tertiary care hospital serving rural population mostly, prevalent bacteria and their susceptibility pattern, risk factors in order to facilitate effective management of SSI.

2.0. AIMS AND OBJECTIVES

1. To find out the prevalence of SSI in this hospital.
2. To elicit the association between bacterial isolates and anatomical site of infection.
3. To identify the probable risk factors for development of surgical site infections
4. To isolate and identify aerobic pathogenic bacteria from surgical site infections (SSI).
5. To determine the antimicrobial sensitivity pattern of pathogens.

3.0. REVIEW OF LITERATURE

Surgical site infection (SSI) has always been one of the major complications in surgical patients. It has been first mentioned even around BC. They have been described and documented since ancient times (4000-5000 years) and considered as one of the important nosocomial infections worldwide.

In 1846, Ignaz Semmelweis noticed that the mortality from puerperal fever was much higher in teaching ward. He also made interesting observation that women who delivered before arrival in the teaching ward had a negligible mortality rate. The tragic death of a colleague due to overwhelming infection after a knife scratch received during an autopsy of awomen who died of puerperal sepsis led Ignaz to observe that pathologic changes in his friend were identical. Then, he hypothesized that puerperal fever was caused by putrid material transmitted from patients by carriage on examining fingers of medical students and physicians who frequently went from autopsy room to the wards. He posted a notice on the door to the ward requesting all caregivers to rinse their hands thoroughly in chlorine water before entering the area. This simple intervention reduced mortality of puerperal fever to 1.5%.[21]

In 19th century, Louis pauster proposed germ theory. His work in humans followed experiments identifying infectious agent in silk worms. He stated that contagious diseases are caused by specific microbes and that microbes are foreign to the host. Using this principle, he developed the techniques of sterilization.

In 1904, William Osler discovered the first cytokines which began to allow insight into organism's response to infection, and led to the explosion in our understanding of host inflammatory response.[22]

The word 'Hospitalism' was introduced by Sir James Simpson to describe what we now call hospital acquired surgical site infections. The following table describes the Historical background of surgical site infections.

Table no.2: Historical Perspectives of Surgical site infections:[23]

S.No	Contributors	Period	Contributions
1	**Hippocrates**	BC 460 – 375	Used wine & vinegar for simple wound irrigation
2	**Galen**	130-200	Recognized localization of infection (suppuration) in wounds inflicted in the gladiatorial arena often heralded recovery, particularly after drainage.
3	**Theodoric of Cervia** **Ambroise Pare** **Guy de Chaulic**	1210-98?1298-1368 1510-90	Observed clean wounds, closure of wounds favours healing without localization/infection/suppuration
4	**Ignac Semmelweis**	1818-65	Introduced hand washing technique & proved reduction of puerperal sepsis (10% to 2%) by simple hand washing steps in between surgeries

5	**Joseph Lister**	1827-1912	Pioneer of antiseptic surgery. Introduced carbolic acid to clean wounds and for sterilizing surgical instruments.
6	**Alexander Fleming**	1881-1955	Introduced chemotherapeutic agents like sulphonamides and penicillin

3.1. CLASSIFICATION OF SURGICAL WOUNDS:

The risk of infection varies by type of surgical incision site. Invasive procedures that penetrate bacteria-laden body sites, especially the bowel, are more prone to infection. The theoretical degree of contamination, proposed by the National Research Council(USA) over 40 years ago, relates well to infection rates.[23] The traditional wound classification system designed by the CDC stratifies the increased likelihood and extent of bacterial contamination during the surgical procedure into four separate classes of procedures[24]

Based on degree of microbial contamination.[25]

- ✓ **Clean wound**
- ✓ **Clean-contaminated wound**
- ✓ **Contaminated wound**
- ✓ **Dirtywound**

Clean wound:

Elective, not emergency, non-traumatic, primarily closed; no signs of acute inflammation;

No break in technique;

Respiratory, gastrointestinal, biliary and genitourinary tracts not entered

Clean-contaminated: A number of studies carried out in India indicate an overall SSI rate of 4.04 to 30% for clean surgeries and 10.06 to 45% for clean-contaminated surgeries. [26, 27]

Emergency case that is otherwise clean

Elective opening of respiratory, gastrointestinal, biliary or genitourinary tract with minimal spillage (e.g. appendectomy) not encountering infected urine or bile

Minor break in technique.

Contaminated:

Acute, non-purulent inflammation

Gross spillage from gastrointestinal tract and entry into biliary or genitourinary tract in the presence of infected bile or urine.

Major break in technique

Penetrating trauma of less than 4 hours

Chronic open wounds to be grafted or covered

Dirty or Infected:

Purulent inflammation of the wound (e.g. abscess);

Preoperative perforation of respiratory**,** gastrointestinal, biliary or genitourinary tract;

Penetrating trauma of 4hours.[28]

3.2. CLASSIFICATION OF SURGICAL SITE INFECTION:

The CDC Guideline for prevention of surgical site infection, published in 1999 defining an SSI

- Superficial incisional SSI
- Deep incisional SSI
- Organ/ Space SSI

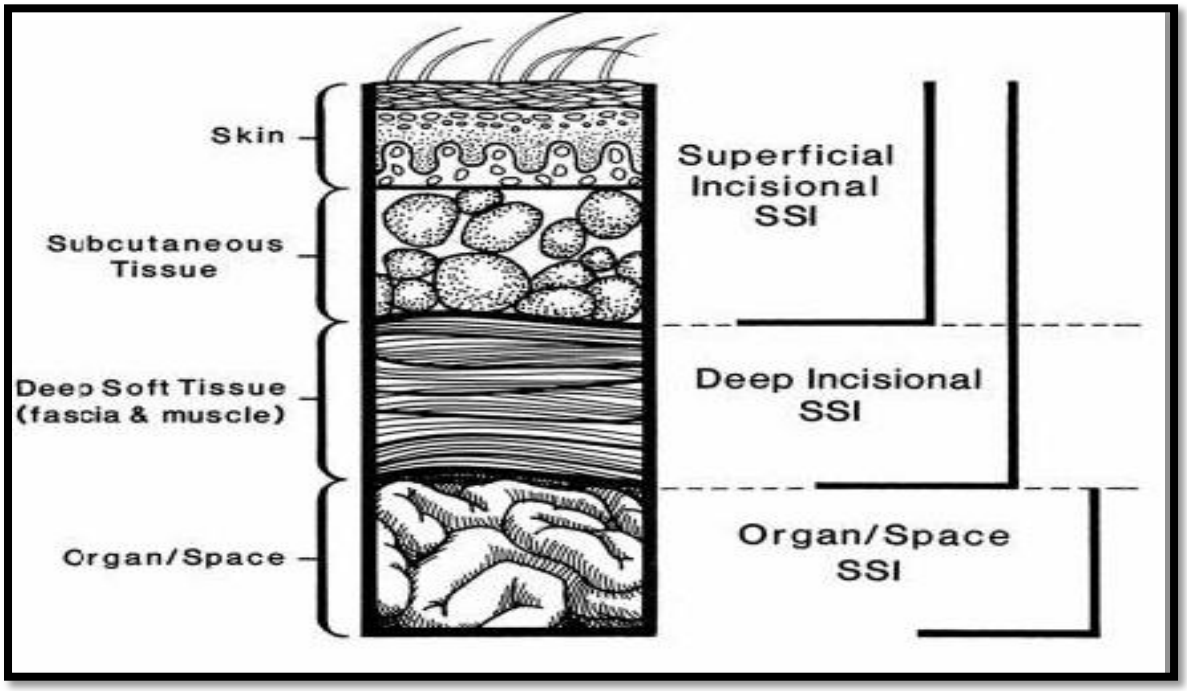


Figure no. 1: Cross section of abdominal wall depicting CDC classification of SSIs[2]

Superficial incisional SSI:

Infection occurs within 30 days of surgery and infection involves only skin or subcutaneous tissue of the incision and patient must present with atleast one of the following criteria:

- Purulent discharge with or without laboratory confirmation.
- Organism isolated from aseptically obtained culture of fluid or tissue from the superficial incision.
- At least one of the following signs of inflammation: pain or tenderness, localized swelling, redness or heat and superficial incision deliberately opened by a surgeon unless incision is culture negative.
- Diagnosis of superficial incisional SSI by the surgeon.
- Excluding stitch abscess, infected burn wounds.

Deep incisional SSI:

Infection involves incision site that extend into the fascial and muscle layers and patient must present with atleast one of the followingcriteria:

- Purulent discharge
- Deep incision spontaneously dehisces or deliberately opened by a surgeon and is culture positive or not cultured when the patient has any of the signs and symptoms of inflammation.
- Evidence of infection by direct examination, during reoperation, or by histopathological and radiological examination.

- Diagnosis of deep incisional SSI by the surgeon.

Organ/ Space SSI:

Infection involves any part of anatomy (organs / spaces) other than the incision.

- Purulent discharge from drain that is placed through a stab wound into organ/ space.
- Evidence of infection by direct examination, during reoperation, or by laboratory confirmation, histopathological and radiological examination.
- Diagnosis of Organ/ Space SSI by the surgeon or attending physician.[2]

3.3. PATHOPHYSIOLOGY:[29] Normally entry of microorganism is prevented by the intact epithelial surfaces. Apart from this there are also other protective mechanism in the host namely

➢**Cellular**: Phagocytic cells, macrophages, polymorphonuclear cells and killer lymphocytes.

➢**Humoral**: Antibodies against the microorganisms, complement and opsonins

➢**Chemical**: Acidic pH of the stomach

Reduced host response to infection may be due to:

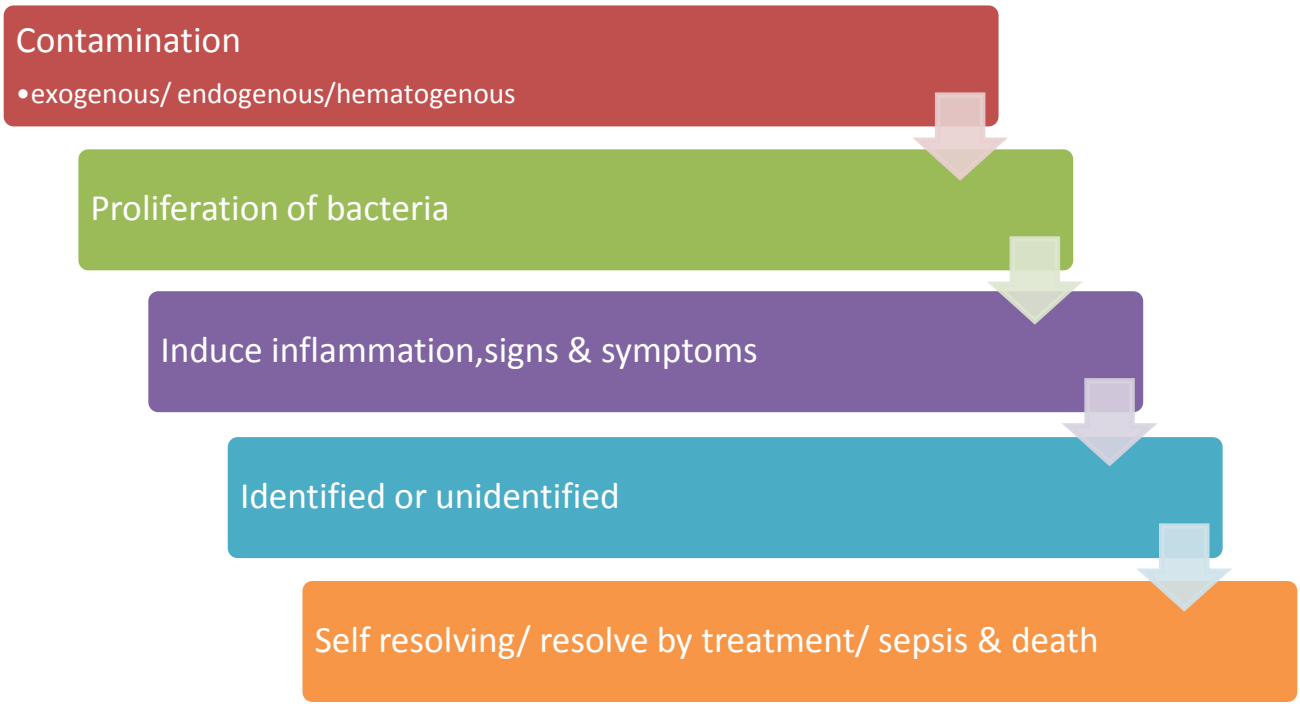
➢ Metabolic: Malnutrition, Diabetes mellitus, Uremia, Jaundice.

➢ Cancer, Acquired Immune Deficiency Syndrome (AIDS)

➢ Iatrogenic: Chemotherapy, radiotherapy and steroids.

Source: Endogenous> exogenous origin

3.4. Pathogenesis of surgical site infections:



3.5. Risk factors of SSI:

Kowli et al. (1985) found an infection rate of 17.4% when preoperative stay was 0-7 days, and an infection rate of 71.4% with a preoperative stay of more than 21 days.[12]**Nichols et al** (1997) in his study on Prolonged postoperative hospitalization, which is a major concern of most of the hospitals, has been evident in patients developing surgical site infection.[30]**Anvikar et al**. (1999) established that preoperative hospital stay predisposed an individual to 1.76% risk of nosocomial infection. With an increase in preoperative stay, the risk increased proportionally. A preoperative stay of one week increased the risk rate to 5% [31].

A mean postoperative stay in patients who developed infection was almost three times as compared to patients who did not develop SSI. The results indicated that 12% of patients undergoing surgery developed SSI.[31]

In 1988 *Lilienfeld* et al published reports have demonstrated that patients with diabetes mellitus and obesity are more susceptible to wound infection because of impaired neutrophil chemotaxis and phagocytosis.

Malnutrition has long been identified as a risk for nosocomial infections, including SSI, among patients undergoing any type of surgery.[32]

Clip the hair immediately before an operation also has been shows a lower risk of SSI than shaving or clipping the night before an operation (SSI rates immediately before = 1.8% vs night before = 4.0%). Dessie et al reported emergency surgeries more prone to SSIs. Dirty and contaminated surgeries are more likely to develop SSIs.[32a,b,c,e]

The risk for developing SSI is a complex interaction between the patient, the procedure and environmental factors which have been listed in the boxes given below.[33,34,35]

Host related factors:

- Age
- Obesity
- Severity of disease
- ASA score(American society of anesthesiologist)
- Nasal carriers of MRSA
- Remote infection
- Duration of preoperative hospitalization
- Malnutrition
- Diabetes mellitus
- Malignancy

Procedure related factors:

- Type of procedure
- Preoperative hair removal
- Antibiotic prophylaxis
- Duration of surgery
- Skin disinfection
- Trauma to tissue
- Foreign materials
- Drains
- Blood transfusion
- Emergency surgery

Environment factors:

- Improper post-operative wound care
- Length of post-operative stay
- Uncontrolled blood glucose
- Inadequate Hand hygiene of HCWs

In 1964, Altemeir and Culbertson conceptualized the pathogenic relationship, key factors of SSIs and also stated that risk of SSIis directly proportional to the microbial

contamination of the operative wound and to virulence of the microorganism and inversely proportional to the integrity and resistance of the host defenses.

$$\text{Risk of SSI} = \frac{\text{Dose of bacterial contamination} \times \text{Virulence of microorganism}}{\text{Resistance of patient defence}}$$

As per American Society of Anesthesiologists (ASA), SSI has been scored based on preoperative physical status of the patient and shown in Table 2

Table no.3: American Society of Anesthesiologists score based on physical status	
ASA Score	Patient's preoperative physical status
1	Normally healthy patient
2	Patient with mild systemic disease
3	Patient with severe systemic disease that is not incapacitation
4	Patient with incapacitation systemic disease that is constant threat to life
5	Moribund patient who is not expected to survive 24hrs with or without surgery

ASA score is an index to assess overall physical status of patient before operation ranging from 1 to 5. It has been shown highly predictive for development of SSI.[36]

CDC has developed National Nosocomial Infections Surveillance System (NNIS) risk index in the year 1991[37] as an improvement over SENIC (Study on

Efficacy of Nosocomial Infection Control) risk index which ranges from 0 to 3 points and is defined by three independent and equally weighted variables.

One point is scored for each of the following if present:

• ASA physical status score >2

• Either contaminated or dirty/infected wound classification

• Length of operation > T hours (where T is approximate 75th percentile of duration of the specific operation being performed.[38]

3.6. Causative Agents:[22]

Table no.4: Causative agents of SSIs:

Gram positive cocci	Other bacteria
Staphylococcus aureus	*Mycobacterium spp*
Staphylococcus epidermidis	*Nocardia asteroids*
Streptococcus pyogenes	*Legionella spp*
Streptococcus pneumoniae	*Listeria monocytogenes*
Enterococcus feacalis, E. faecium	Fungi
Gram negative bacilli	*Candida spp.*
Escherichia coli	*Cryptococcus spp*
Hemophilus influenzae	*Blastomyces dermatitidis*
Klebsiella pneumonia	*Aspergillus spp*
Proteus mirabilis	*Coccidioides immitis*
Enterobacter aerogenes, e. cloacae	*Mucor/rhizopus*
Serratia marcescena	Viruses
Acinetobacter spp	Cytomegalovirus
Citrobacter freundii	

Pseudomonas aeroginosa *Xanthomonas maltophilia* Anaerobes *Bacteroids spp.* *Fusobacterium spp.* *Peptostreptococcus* *Clostridium spp*	Epstein –Barr virus Hepatitis A,B,C Herpes simplex virus HIV Varicella zoster virus

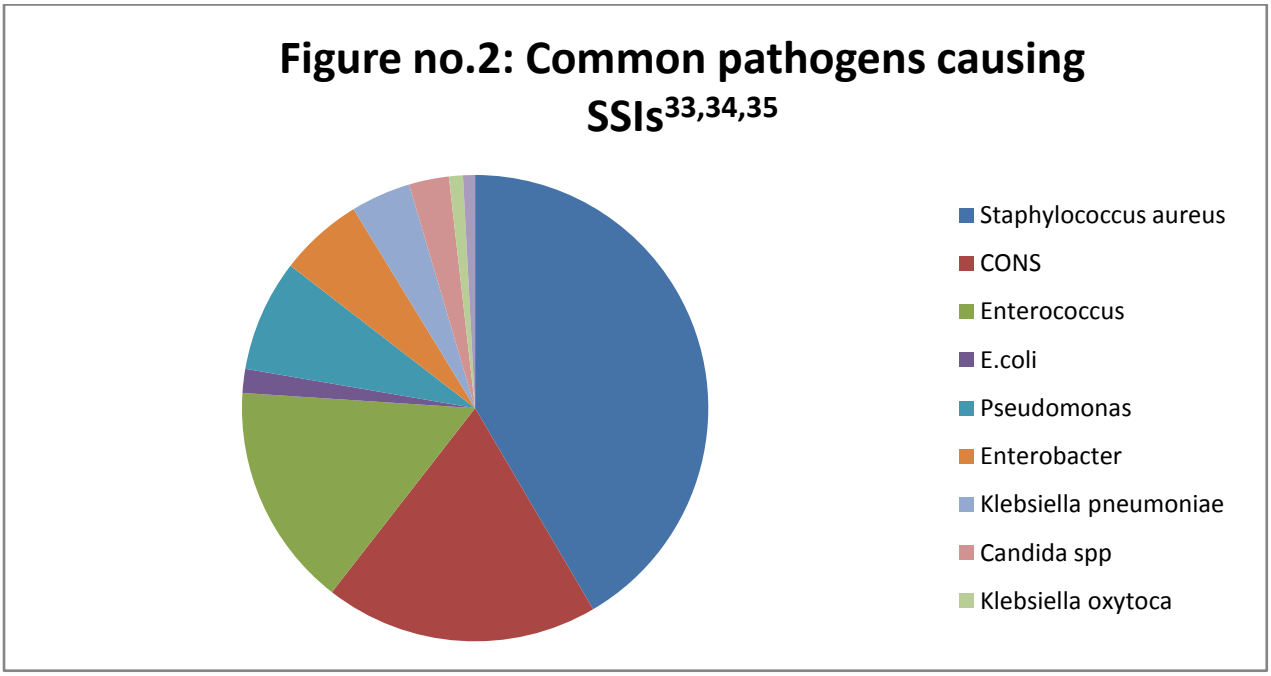


Figure no.2: Common pathogens causing SSIs[33,34,35]

3.7. Historical Aspects of antibiotic prophylaxis:

Experimental studies published during the early 1960s helped clarify many of these problems and resulted in a more scientifically accurate approach to antimicrobial prophylaxis. Most important was the report by Burke [39], which demonstrated the crucial relationship between timing of antibiotic administration and its prophylactic efficacy. His experimental studies showed that to greatly reduce experimental skin infection produced by penicillin-sensitive S. aureus, the penicillin had to be in the skin shortly before or at the time of bacterial exposure. This study and others fostered the attitude that to prevent subsequent infection the antibiotic must be in the tissues before or at the time of bacterial contamination. This important change in strategy helped correct the common error of first administering the prophylactic antibiotic in the recovery room.

As early as 1964, Bernard and Cole[40] reported on the successful use of prophylactic antibiotics in a randomized, prospective, placebo-controlled clinical study of abdominal operations on the gastrointestinal tract. The success of antibiotic prophylaxis noted in this early study was clearly due to the authors' appropriate patient selection and wise choice of available agents, as well as the timing of administration.

Further advances in understanding of antibiotic prophylaxis in abdominal surgery occurred in the 1970s. During this decade, the qualitative and quantitative nature of the endogenous gastrointestinal flora in health and disease was appropriately defined [41]. Many prospective, blinded clinical studies in the 1980s and 1990s prompted definitive recommendations concerning the proper approaches to antibiotic prophylaxis in surgeryand shown in table no.5.

3.8. Table no.5: Antibiotic prophylaxis for surgical procedure[42,33]

Surgical procedures	**Antibiotics**
Cardiac surgery	Cefuroxime 1.5g 8 hourly
Neurosurgery	Cefuroxime 1.5g single dose
Head and Neck	Cefuroxime 1.5g and metronidazole 500mg 8 h(single dose) involving mucous, and upto 3 doses if membrane and deep tissue involved
Biliary tract surgery	Cefuroxime 1.5g single dose
Endoscopic retrograde cholangiopancreatography	Cefuroxime 1.5g single dose
Gastroduodenal	Cefuroxime 1.5g single dose
Appendectomy	Cefuroxime 1.5g/ gentamycin 2-3mg/kg and metronidazole 500mg (single dose)
Colorectal surgery	Cefuroxime 1.5g/ gentamycin 2-3mg/kg and metronidazole 500mg (single dose)
Orthopaedic surgery	Cefuroxime 1.5g single dose
Lower limb amputation	Benzylpenicillin 2mega units IV 6 h; metronidazole /clindamycin for patient allergic to penicillin All antibiotic should be given for 24 h duration
Peripheral vascular surgery	Cefuroxime 1.5g 8 hourly (3 doses)
Urological surgery	IV antibiotic depends upon urine sensitivity report. In emergency condition gentamycin 2-3mg/kg

Hysterectomy	Cefuroxime 1.5g and metronidazole 500mg or amoxiclav 1.2g alone(single dose)
Caesarean section	Cefuroxime 1.5g or amoxiclav 1.2g IV after umbilical cord is clamped (single)

3.9. Prevalence of SSIs:

It is estimated that 234 million major surgical procedures are performed annually worldwide.[43] Among all types of Health care associated infections, SSI varies from 2.5% to 41.9% all over the world[44,45]. They are associated with longer post-operative hospital stays, additional surgical procedures, treatment in intensive care units and higher mortality.[46]Many studies reported that it varies from hospital to hospital based on infection control measures and antibiotic policy. One review study reported that SSI develops around 1 in 20 surgical patients in hospitals[47]

Suchithra et al observed that the prevalence of SSIs was 12%; and the common etiologic agents are gram-positive organisms like S*taphylococcus aureus* and *Enterococcus* spp and gram-negative organisms are *Pseudomonas aeruginosa*, *Escherchia coli* and *Klebsiella* spp their results are consistent with various other literature reports indicating that S*taphylococcus aureus* was the commonest isolate from postoperative wound infection. *E. faecalis* was seen in 33.3% of surgical site infections. Also among the gram-negative bacilli, the predominant isolate was *P. aeruginosa* (24.4%), followed by *E. coli* (7.4%) and *Klebsiella* spp. (1.4%). [48]CDC reported a mortality rate of 3%,Weigelt et al reported a total mortality rate of 0.95%

for SSIs.[49]Mortality rate of appendectomy is 0.7% and 2.4% in patients without and with perforation[50]

The modern surgeon cannot escape the responsibility of dealing with infections and when dealing with them, should have knowledge of the appropriate use of aseptic and antiseptic technique, proper use of prophylactic and therapeutic antibiotics and adequate monitoring and support with novel surgical and pharmacological modalities, as well as nonpharmacological aids[50].

3.10. Antimicrobial Resistance in surgical site infections

Antibiotic era started with discovery of penicillin by Alexander Fleming in 1928 [58]. Use of Penicillin started in 1941. Emergence of penicillin resistance is identified in S*taphylococcus aureus* due to plasmid encoded β-lactamase. First plasmid mediated β-lactamase in gram negative organisms- TEM-1 was described in early 1960's[58]. It was first isolated in *Escherichia coli* from a patient Temoniera in Greece and the gene responsible for it was named after him. It spread to other genera soon. Evolution of drug resistance is shown in table no.6 given below

Table no.6: Evolution of drug resistance

Year	**Event (Antimicrobial resistance)**
1937	Sulfonamides introduced for treatment[52]
1940	Penicillin came into clinical use[53]
1940	First evidence of betalactamases (Penicillinase) demonstrated in *E.coli* by Abraham and Chain[53]

1940	Tetracycline came into clinical use[54]
1953	First tetracycline resistance was reported in *Shigella dysentria*[54]
1970s	Plasmid mediated β-lactamases assumed importance in *Enterobacteriaceae* and other gram negative bacteria[54]
1972	First epidemic of Chloramphenicol resistant *Salmonella* in Kerala reported by Paniker et al.[55]
1989	MDR *S.Typhi* outbreaks resistant to Chloramphenicol, Ampicillin, Trimethoprim, Streptomycin, Tetracycline and Sulfonamides were reported in India and Pakistan[55]
1992	*S.Typhi* resistant to Ciprofloxacin was first reported in UK.[55]
1970-80s	Development of broad spectrum Cephalosporins, Cephamycins, Monobactams and Carbapenems[53]
1990	Inducible chromosomally mediated β-lactamases among gram negative bacteria[53]

Beta lactamases:

Enzymes which inactivate betalactam antibiotics by hydrolysing the nitrogen carbonyl bond in their betalactam ring are collectively known as betalactamases. They are members of a super family of active site serine proteases and act by cleaving an amide bond of beta- lactam ring to form an acyl-enzyme complex. They can be plasmid mediated or chromosomal .These β-lactamases are secreted as exozymes in gram positive bacteria and within the periplasmic space in bacteria that are gram negative. More than 170 enzymes of this kind has been discovered [56].

Methicillin resistant *Staphylococcus aureus* (MRSA):

Methicillin was the first penicillinase resistant penicillin and has been widely used in testing susceptibility of *S. aureus* to penicillinase resistant β-lactam agents. Hence, despite the fact that methicillin is no longer available and oxacillin and cefoxitin have replaced it for susceptibility testing, resistant strains are commonly known as MRSA.

MRSA strains are a continuing and increasing problem in healthcare settings, with outbreaks now occurring in the community. Screening for MRSA provides a means of identifying patients and staff who may be at risk of infection and/or involved in transmission of the organism.

MRSA were first described in the 1960s [67]. During the late 1970s and early 1980s, strains of *S. aureus* resistant to multiple antibiotics including methicillin and gentamicin were increasingly responsible for outbreaks of hospital infection worldwide and several clonal types have shown extensive international spread [68,69,70] In England and Wales, the spread of MRSA was well controlled until the 1990s. Between 1989 and 1991 only 1.6% of *S. aureus* bacteraemia isolates were methicillin resistant [71]. However, methicillin resistance rates increased steadily throughout the 1990s, there were also significant increases in the percentages of isolates resistant to erythromycin, clindamycin, ciprofloxacin, gentamicin, trimethoprim and rifampicin[72]. MRSA reached in excess of 40% in several regions in 2001 which triggered the introduction of mandatory surveillance of MRSA bacteraemia[73]. In 2005, trusts were tasked with reducing the number of cases of MRSA and since that time cases have fallen[74,75] Studies have shown that the majority of patients from whom MRSA strains

are isolated are colonised rather than infected with the organism [76]. Factors predisposing to superficial colonisation include procedures involving “hands on” care especially in acute surgical, renal dialysis and critical care units [77]. The risk of colonisation resulting in infection is increased in the presence of any breach in the skin, such as surgical wounds and devices penetrating the skin, for example prostheses and catheters, which provide a portal of entry for bacteria [77]. MRSA and MSSA are similar in virulence and this is often connected to mobile genetic elements the presence or absence of which determines the clinical outcome [78]

Extended spectrum of β-lactamase: (ESBL)

The ESBL enzymes are plasmid - mediated enzymes capable of hydrolyzing and inactivating a wide variety of β-lactams (oxyimino side chain). These cephalosporins include cefotaxime, ceftriaxone, and ceftazidime, as well as the oxyimino-monobactamaztreonam. [57]

Another common plasmid mediated β-lactamase gene found in *Klebsiella pneumonia* and *Escherichia coli* are SHV-1 (SulphHydryl in Variable). Over the last 20 years many new β - lactam antibiotics have been developed which were resistant to hydrolytic action of β - lactamases but, because of indiscriminate use, these antibiotics alsobecame resistant. To overcome it, around **1980**, 3rd generation cephalosporins also called broad spectrum Cephalosporins were introduced. Because of their extensive use, they also became resistant. Widespread use of third generation cephalosporins and aztreonam is believed to be the major cause of the mutations in these enzymes that has led to the emergence of the ESBLs[59].

Various classification schemes have been proposed by many researchers since 1968.[60]However, a more modern scheme based on molecular structure classification was proposed by Ambler especially of only those enzymes that have been characterized.

All ESBLs have serine at their active sites except for a small (but rapidly growing) group of metallobetalactamases belonging to class B. They share several highly conserved amino acid β sequences with penicillin binding proteins (PBPs)[61] β--lactamases attack the amide bond in the betalactam ring of penicillins and cephalosporins, with subsequent production of pencillinoic acid and cephalosporic acid, respectively, ultimately rendering the compounds antibacterially inactive [62] . Plasmids responsible for ESBL production tend to be large (80 Kb or more in size) and carry resistance to several agents, an important limitation in the design of treatment alternatives [63]. The most frequent coresistances found in ESBL producing organisms are aminoglycosides, fluoroquinolones, tetracyclines, chloramphenicol and sulfamethoxazole-trimethoprim [59.]

1. Impermeability of the Membrane mediated by both chromosome and plasmid.

2. Alteration of target protein e.g., Penicillin binding protein.

3. Increased efflux of the drug from the periplasmic space.

Characteristics of ESBLs: [56]

They are mostly class- A Cephalosporinases carried on plasmids.

They are more common in *Klebsiella species* followed by *Escherichia coli* described first in Germany and France.

1) All enzymes active against Cephalothin.

2) Imipenem and Cefoxitin not hydrolysed.

3) Comparative activity against Cefotaxime and Ceftazidine varies with enzymes.

4) Some enzymes active against Aztreonam.

5) Inhibition of activity by β-lactamase inhibitors can be demonstrated.

Major risk factors for ESBL production:

Risk factors are prolonged stay in ICU, long term use of antibiotics, nursing home residency, severe illness, high rate of use of Ceftazidime and other Third Generation Cephalosporins and use of life lines

Medical significance of detection of ESBL:

Patients having infections caused by ESBL – producing organisms are at increased risk of treatment failure with expanded spectrum β-lactam antibiotics. So, it is recommended that if an organism is confirmed to produce ESBL it is considered as resistant to all 3rd Generation Cephalosporins.

Many ESBL isolates will not be phenotypically resistant; even through their MIC is so high. ESBL producing strains have been established in many hospitals producing epidemic diseases especially in Intensive Care Units.[64] Failure to control outbreaks has resulted in new mutant types in some institution.

Staphylococcus aureus was the most frequently isolated pathogenic bacteria from post-operative wounds. A majority of the isolates were methicillin resistant *Staphylococcus aureus* (MRSA). Most of the gram-negative bacteria which were isolated, ie; *Escherichia coli, Proteus mirabilis, Klebsiella* species and *Pseudomonas aeruginosa* were sensitive to quinolones and aminoglycosides, but were resistant to

cephalosporins. Rest had *Enterobacteriaceae*, either extended-spectrum β-lactamase (ESBL) producers or Amp-C hyperproducers. Indiscriminate use of antibiotics is a major problem predisposing patients to harm by multi-resistant pathogens. Carbapenems were in use nowadays, but the selection pressure exerted by cephalosporins, suggesting a role of single plasmid carrying resistance genes to multiple classes.[66]

Carbapenemases:

Carbapenemases are beta lactamases that cause resistance to carbapenem, the β-lactam group with the broadest spectrum of antibacterial action. Carbapenems were less susceptible to the inactivating activity of many betalactamases till the recent past. But now, even these efficient antibiotics are becoming susceptible to the enzymatic inactivation by betalactamases.

The enzymes hydrolysing carbapenems can be grouped into classes A or B by molecular analysis. The former has serine as the active site member and the latter has zinc at the active site. Since these enzymes are dependent on zinc, a metal, they are called Metallobetalactamases. Some class C cephalosporinases can hydrolyse/inactivate carbapenems and result in carbapenem resistance, but they are not called carbapenemases because they are not carbapenem specific.

Antibiotic resistance is rising to dangerously high levels in all parts of the world. New resistance mechanisms are emerging and spreading globally, threatening our ability to treatand sometimes impossible. **Defezz et al.,** noted that multi drug resistance (MDR) in *P. aeruginosa* is usually defined as resistance to three or more of the antimicrobial agents.[51]

4.0. MATERIALS AND METHODS:

This was a Hospital based Prospective Cross sectional study and carried out at the Department of Microbiology, Trichy SRM Medical College Hospital and Research Centre, Irungalur, Trichy, Tamilnadu. The study was carried out over a period of one year (May 2017 to April 2018).

4.1. Materials:

Consecutive cases of both sexes and all adults belonging to various surgical wards and underwent surgical procedure during the study period comprising of elective as well as emergency were considered for the present study.

Patients belonging to anyone of the following were excluded.

1. Paediatric cases.
2. Cases taken for second surgery at the same site for any reason.
3. Patients on immunosuppressant or with immunodeficiency status.
4. Patients on antibiotics already for any other infections.
5. Presence of infection elsewhere in the body or focal sepsis.

The work was carried out after getting approval from Institutional research board and Institutional ethics committee (copy enclosed – Annexure –I). Informed consent (in vernacular) was obtained from every case (model copy of informed consent enclosed – Annexure-III).

4.2. Patient history

Age, sex demographic details, clinical details including name of the procedure, date and duration of surgery, experience of surgeons, preoperative hospital stay, nature of surgery, antibiotic prescribed (prophylactic/post operative), post operative hospital stay, risk factors, onset of illness and other relevant history were collected and recorded in a proforma (copy enclosed - Annexure- II).

4.3 Specimen collection and transport

After 48 hours of surgery, dressings on the surgical wounds were removed. Evidence of wound infection was considered if the patient had local inflammatory changes such as edema, redness, warmth or discharge from wound site. These were looked into each case and the changes were documented. If there was any discharge, samples were collected before dressing of the wounds. If only inflammatory changes were present without any discharge, the wounds were monitored till discharge of the patient and for development of discharge from wound. If no inflammatory signs were noticed within 48 hrs, cases were followed up with the help of respective surgeons. The surgeons incharge of the case was requested to inform/call the postgraduate scholar doing this work whenever he/she suspected signs of SSIs in the form of fever and local signs of inflammation. In addition, these patients were educated and followed up through mobile phone for the development of SSIs over the period of 30 days.

4.3.1 Pus swab and aspirate:

Preparation of wound site– The suspected as well as overt infected areas were cleaned with sterile normal saline followed by 70% alcohol and then the specimen

was collected using sterile swab. Two swabs were taken from the depth of the wound or lesion and aspirates were collected in a sterile disposable syringe and transported to the laboratory within two hours.[79]The color, consistency and odor of the samples were observed and recorded.

4.4. Laboratory works:

Gram stain:

Direct thin smear was made from each wound swab and/or aspirates on a clean grease free glass slide and was air dried. It was then heat fixed and Gram staining was done with positive and negative control (ATCC *Staphylococcus aureus* 25923 and *E.coli* 25922). The presence of pus cells and microorganisms was observed under the oil immersion (100 X) objective.

The samples were cultured onto Nutrient agar, 5% Sheep blood agar and Mac Conkey agar plates by adopting standard microbiological techniques. After 24 hrs of incubation aerobically at 37°c, plates were read and the isolates were identified based on colony morphology, Gram stain, motility and biochemical tests. Antibiotic sensitivity test (AST) was performed by Kirby-Bauer disc diffusion method for all isolates according to the CLSI 2017 guidelines. Repeat subculture was carried out on next day for samples showing no growth on plates on first day and were processed further[80]. All the isolates were identified by colony morphology, microscopic appearance, biochemical tests and phenotypic tests for drug resistance.

A) Identification of Gram positive cocci:

Staphylococcus aureus, Enterococci and Micrococci were identified by colony morphology, Gram staining and biochemical test as per standard microbiological procedures.

i) *Staphylococcus aureus*, was identified based on the following characteristics i.e; gram positive cocci in clusters on Grams staining, golden yellow pigment on Nutrient agar plate, positive for catalase and tube coagulase test and showing fermentative pattern in Oxidative Fermentative (OF) test of Hugh and Leifson.

ii) All coagulase negative gram positive clusters were considered as CoNS.

iii) Micrococci were identified based on grams staining and oxidative pattern in OF test and excluded as commensal.

iv) *Enterococci* were identified based on microscopic morphology i.e; gram positive cocci in diplos, negative for catalase, positive for bile esculin hydrolysis, heat tolerence property and mannitol fermentation[80] .

Biochemical tests:[81]

Catalase test:

It was performed by Tube test with controls.

A small portion of colony was transferred from the Nutrient agar plate by a clean platinum wire or glass rod into a tube containing 3% hydrogen peroxide.

Positive control: *Staphylococcus aureus*

Negative control: *Streptococcus sp*

Interpretation:

Positive - Evolution of effervescence within 10 seconds

Negative – no or delayed effervescence

Coagulase test:

This was performed by slide test (for detecting bound coagulase) and tube test (for detecting free coagulase**).**

Slide Coagulase Test:

The suspected Staphylococcal colony was emulsified in a drop of water on a microscope slide. A flamed and cooled straight inoculating wire was dipped into the undiluted plasma at room temperature, the adhering traces of plasma was stirred into the Staphylococcal suspension on the slide with control.

Positive – Coarse visible clumping within 10 seconds

Negative - Absence of clumping in less than 10 seconds.

Tube coagulase test:

A 1/6 dilution of the plasma was prepared in normal saline (0.85%Nacl) and 1ml volume of the diluted plasma was taken in a small tubes. A colony of *Staphylococcus* was emulsified in a test tube with diluted plasma. It was incubated at 37ºC for up to 4 hours. The tubes were examined at 1, 2 and 4 hours for clot formation by tilting the tube through 90º. The negative tubes were left at room temperature overnight and re-examined.

Positive control: *Staphylococcus aureus* ATCC 25923

Negative control: *Staphylococcus epidermidis*

Interpretation:

Positive - Any degree of clot formation

Negative - If the plasma remained liquid or showed only a flocculent or ropy precipitate.

Bile Esculin hydrolysis:

One to two colonies from an 18 to 24 hours growth on nutrient agar plate was inoculated on to the surface of the bile esculin agar slant. It was incubated at 35ºC in ambient air for 48 hours.

Positive control: *Enterococcus spp*

Negative control: *Viridans streptococcus*

Interpretation:

Positive - Blackening of the agar slant

Negative - no colour change.

B) IDENTIFICATION OF GRAM NEGATIVE BACILLI (GNB)

The gram negative bacilli were identified based on the colony morphology, motility, catalase test, oxidase test, indole test, Methyl red, Voges Proskauer, triple sugar iron agar, citrate utilisation and urease production.

Oxidase test:

It was performed by picking a colony using platinum loop or glass rod. The colony was tested on freshly prepared solution of 1% oxidase reagent (tetra methyl paraphenylene diaminedihydro chloride) with control.

Positive control: *Pseudomonas aeruginosa* ATCC 27853

Negative control: *Escherichia coli* ATCC 25922

Interpretation:

Positive – deep purple colour change within 10 seconds.

Negative – colour change after 10 seconds.

Indole test:

The organism was inoculated into peptone water and incubated for 24 hrs. Later, Kovacs reagent was added. If the color changed to red on the top of the test tube it was considered as positive.

Positive control: *Escherichia coli* ATCC 25922

Negative control: *Klebsiella pneumoniae*

Interpretation:

Positive – Red coloured ring

Negative – Yellow coloured ring

Methyl red test (MR):

The gram negative bacteria from a 24 hrs growth culture was inoculated in glucose phosphate broth and incubated at 35ºC to 37ºC for 48 to 72 hrs aerobically. Then 5 to 6 drops of 0.04% solution of Methyl red was added. The results were read immediately after mixing well.

Positive control: *Escherichia coli* ATCC 25922

Negative control: *Enterobacter aerogenes*

Interpretation:

Positive – stable bright red color in the surface of medium.

Negative – no colour or intermediate orange colour change.

Voges Proskauer test (VP):

The test organism was inoculated in glucose phosphate broth and incubated at 35°C to 37ºC for 48 to 72 hours. 6 drops of solution A (alpha naphthol) and 2 drops of solution B (KOH) were added to 1 ml of the broth and was observed after mixing well for 5 minutes.

Positive control: *Enterobacter aerogenes*

Negative control: *Escherichia coli* ATCC 25922

Interpretation:

Positive - Red color within 15 minutes or more after addition of reagent.

Negative – no colour change or copper colour after 1 hour.

Citrate utilization test:

Bacterial colony was picked by touching the tip of the needle on the colony that was 18 to 24 hrs old and inoculated into solid (Simmon's) media with indicator bromothymol blue, lightly on the slant and incubated at 37ºC. Then it was observed for development of blue color and growth.

Positive control: *Enterobacter aerogenes*

Negative control: *Escherichia coli* ATCC 25922

Interpretation:

Positive - Intense blue color and/ or growth on the slant.

Negative - No change in color and growth

Christensen's urease test:

The test was done by using Christensen's medium. The organism was inoculated on the entire slope of the medium and overnight incubated at 37°C for up to 7 days.

Positive control: *Proteus spp*

Negative control: *Escherichia coli* ATCC 25922

Interpretation:

Positive – Pink Colour

Negative – Pale yellow colour

Triple sugar iron (TSI) test:

The medium was inoculated with bacterial culture using a straight wire (Stab culture) and then streaked on the slant. It was incubated at 37°C 24 to 48 hours.

Interpretation:

Acid / Acid with gas – Glucose and Lactose/ Sucrose fermenter

Alkaline / Acid– Glucose fermentor

Alkaline / Acid with abundant black colour – Glucose fermentor with Hydrogen sulphide production

Alkaline / Alkaline – Non fermenting GNB

Nitrate reduction test:

The test organism was inoculated with one drop from a 24 hrs nitrate broth culture which was incubated at 35ºC for 48 – 72 hrs. It was then examined for nitrogen gas in the inverted Durham tubes and 5 drops of nitrate reagent A and B (sulphanilic acid and α–naphthylamine) were added. It was observed for 3 min for red color to develop.

Positive control: *Escherichia coli* ATCC 25922

Negative control: *Acinetobacter baumannii*

Interpretation:

Positive - Red color change within 30 seconds

Negative – no colour change

Table no.7: Biochemical reactions and isolation of microbes[81]:

Organisms	Grams	Catalase	Oxidase	I	NR	MR	VP	C	TSI	U	MMM
E.coli	GNB	+	-	+	+	+	-	-	A/A	-	+/+
K.pneumoniae	GNB	+	-	-	+	-	+	+	A/A	+	+/-
K.oxytoca	GNB	+	-	+	+	-	+	+	A/A	+	+/-
Proteus spp	GNB	+	-	-	+	+	-	+	K/A^{+}	+	-/+
Enterobacterspp	GNB	+	-	+	+	-	+	+	A/A	-	+/+
Citrobacterkoseri	GNB	+	-	+	+	+	-	+	A/A	-	+/+
Pseudomonas aeruginosa	GNB	+	+	-	+	ND	ND	+	K/K	-	-/+
Acinetobactersp	GNB	+	-	-	-	ND	ND	+/-	K/K	-	-/-

GNB-Gram negative bacilli, I – Indole, MR – Methyl Red, VP- VogesProskauer, C- Citrate, U- Urease, MMM- mannitol motility medium, NR – Nitrate Reduction, TSI –Triple Sugar Iron, A- Acid, K- alkaline, $^{+}$ Hydrogen sulphide production, ND- not done.

4.5. ANTIMICROBIAL SENSITIVITY TESTING[80]

The antimicrobial sensitivity testing for all the isolates was done on Muller Hinton Agar by Kirby – Bauer disc diffusion method as per CLSI 2017 guidelines using antibiotic discs (Himedia, Mumbai)

I. Kirby Bauer Disk Diffusion Test:

Preparation of turbidity standard:

McFarland 0.5 standard was prepared by adding 99.55 ml of 1% Suphuric acid and 0.5 ml of 1.175 % barium chloride. This solution was dispersed into tubes comparable to those used for inoculum preparation. It was sealed tightly and stored in the dark at room temperature. The McFarland 0.5 standard provides turbidity comparable to that of a bacterial suspension containing approximately 1.5 X 10^8 CFU/ml.

Preparation of Inoculum:

In order to prepare the inoculum**,** about 3-5 representative colonies were picked up and inoculated in 4 - 5 ml of peptone water and incubated at 37ºC for 2 – 6 hrs to attain 0.5 McFarland's standard and if it was found more turbid, then some more quantity of peptone water was added and adjusted to 0.5 McFarland's standard by comparing against a card with white background and contrasting black lines.

Inoculation of Muller Hinton Agarplates:

Within 15 minutes of adjusting the turbidity of the inoculum suspension, a sterile cotton swab was dipped into broth and rotated several times. During this process, the swab was pressed firmly on the inside wall of the tube above the fluid level to remove excess of broth from the swab. Then, the dried surface of Muller

Hinton agar plate was inoculated by streaking the swab over the entire sterile agar surface. This procedure was repeated by streaking two more times by rotating the plates at an angle of approximately 60ºc to ensure an even distribution of inoculum and finally, the rim of the agar was swabbed. The plate was closed and left for 3-5 minutes to allow any excess surface moisture to be absorbed before applying antibiotic impregnated discs.

Application of discs to inoculated agar plates: Disc container was taken out from refrigerator one or two hours before use and brought to room temperature. Once a cartridge of discs has been removed from its sealed package, it was replaced in a tightly sealed dry container after use in refrigerator. The entire discs were placed on agar plates and pressed down to ensure complete contact with the agar surface. Discs were distributed evenly so that they were not closer than 25 mm from centre to centre of the disc and incubated at 37º C for 16 – 18 hrs.

Reading and interpretation of results:

After 16-18 hrs of incubation, each plate was examined for satisfactory streaking with confluent lawn of growth uniformly and circular zones of inhibition. The diameter of the zones of complete inhibition including the diameter of the discs was measured. The zones were measured to the nearest millimeter using a ruler that was held on the back by inverting Petri plate. The Petri plate was held a few inches above a black, non reflecting background and illuminated with reflected light. The zone margin showing no obvious visible growth that could be detected with unaided eyes was considered as a zone of inhibition. The sizes of the zones of inhibition were interpreted as per CLSI

standards and reported as '**susceptible', 'intermediate' or 'resistant'** to the drugs that were tested.

A bacterium can be

Susceptible – when it is inhibited by the concentration of the drug usually used

Intermediate – when it is susceptible to drug at higher than normal dosages

Resistant – when it is not inhibited by the drug[82]

Control strains used with each batch:

i. *Escherichia coli* ATCC 25922

ii. *Pseudomonas aeruginosa* ATCC 27853

iii. *Staphylococcus aureus* ATCC 25923

iv. *Enterococcus faecalis* ATCC 29212

Table no.8: List of antibiotics tested:

As per CLSI 2017 guideliness[83]

Gram positive cocci	**Gram negative bacilli**
Penicillin(10U) Ampicillin (10 µg), Erythromycin (15 µg), Clindamycin (2 µg), Gentamicin (10 µg), Co-trimoxazole (1.25/ 23.75 µg), Tetracycline (30 µg), Ciprofloxacin (5 µg) High level gentamycin(120 µg) Linezolid (30µg))	Ampicillin (10 µg) Amoxclav(20/10µg) Amikacin (30 µg) Gentamycin(10µg) Ciprofloxacin (5 µg) Trimethoprim/sulfoethoxazole (1.25/23.75µg) Ceftriaxzone (30 µg) , Cefotaxime (30 µg) Ceftazidime (30µg) Cefepime (30µg) Piperacillin/ tazobactum (180/ 18 µg) Imipenem(10 µg)

4.6. Detection of MRSA:

MRSA isolates were detected by standard disc diffusion method using Cefoxitin (30µg). Cefoxitin is considered as a better inducer of mec-A gene than oxacillin or methicillin, and can be used to screen heterogeneous MRSA populations. As per CLSI 2017 guidelines, zone of inhibition ≤ 21 mm was considered as Methicilin resistant isolates.[84]

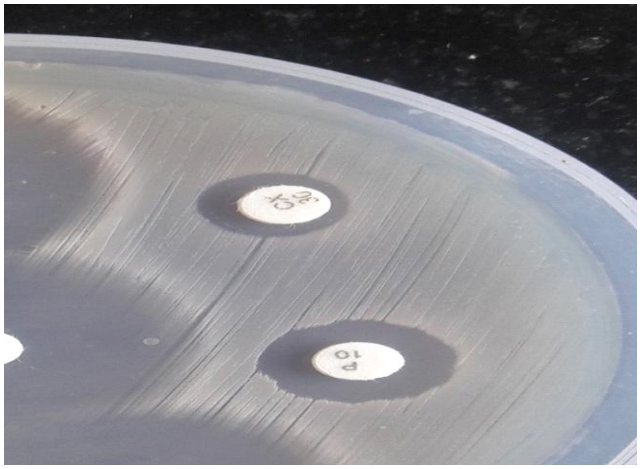


Fig 3 - Cefoxitin disc diffusion method for detection of MRSA ZOI ≤ 21 mm.

4.7. Detection of Extended Spectrum Betalactamases:

As per CLSI 2017 guidelines, the test isolates which showed an inhibition zone of ≤27mm for cefotaxime (CTX), ≤25mm for Ceftriaxone(CTR) and ≤ 22mm for Ceftazidime (CAZ) were considered as presumptive ESBL producer. All these isolates were further tested for phenotypic confirmation test for ESBL.

Phenotypic Confirmation Test:

Antibiotic susceptibility testing was done on Muller Hinton Agar with 0.5 McFarland's standard of the organism[85].

Lawn culture of the organism was made and 3rd generation cephalosporin, Ceftazidime and Cefotaxime (30µg) disc was tested alone and along with their combination for 10µg of Clavulanic acid. Organisms with 5mm increase in zone of inhibition for Ceftazidime and Cefotaxim / Clavulanic acid (30µg/10µg) are confirmed as ESBLs. [86,87].

Indicators of ESBLs: 5 mm increase in diameter of inhibition zone when using disc diffusion method with 3rd generation Cephalosporin and Clavulanic acid combined disc.

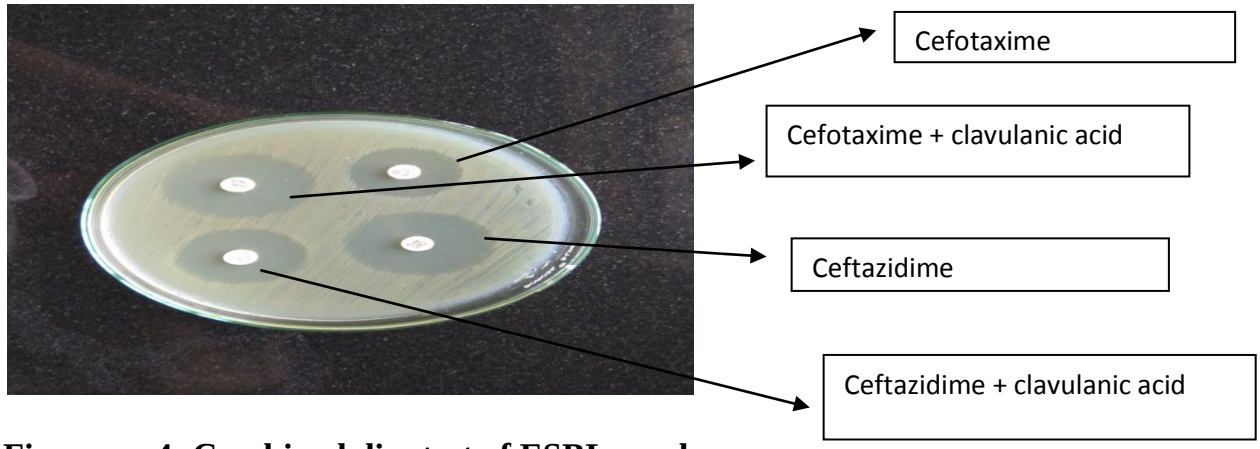


Figure no.4: Combined disc test of ESBL producers

4.8. DETECTION OF AMP C PRODUCERS

As per CLSI 2017 guidelines, the test isolates which showed an inhibition zone of ≤ 18 mm for Cefoxitin disc (30µg) were considered as presumptive Amp C producer. All these isolates were further tested by Amp C disk test.

AMP C DISK TEST:

All the Cefoxitin resistant strains were subjected to Amp C disk test to detect the production of Ambler class C β-lactamase.[88]

- An overnight culture suspension of ATCC *E.coli*25922 was prepared in peptone water, matched to 0.5 McFarland turbidity standards and inoculated as lawn culture over a 90mm MHA plate as for routine disk diffusion procedure.[89]
- A Cefoxitin disk with a potency of 30 microgram was placed over the lawn.
- An empty disk moistened with sterile saline and inoculated with the test organism was placed at the vicinity of the Cefoxitin disk almost touching it. The culture plate was kept in the incubator for overnight incubation at 37° C.[88]
- Blunting of the zone of inhibition of cefoxitin near the test strain inoculated disc was taken as indicative of the strain being a producer of Ambler class C betalactamase, as shown in Fig no.5.
- The results were recorded and tabulated.

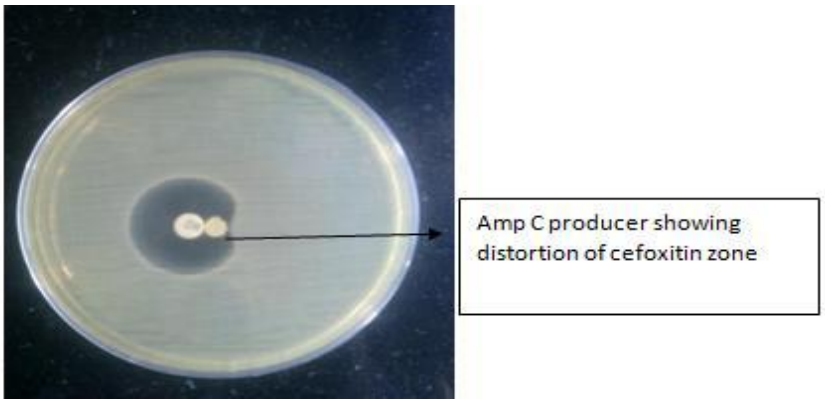


Figure no.5: Amp C disc test

4.9. Detection of Carbapenemase producing organisms:

As per CLSI 2017 guidelines, the test isolates which showed an inhibition zone of imepenem were subjected to combined disc test.

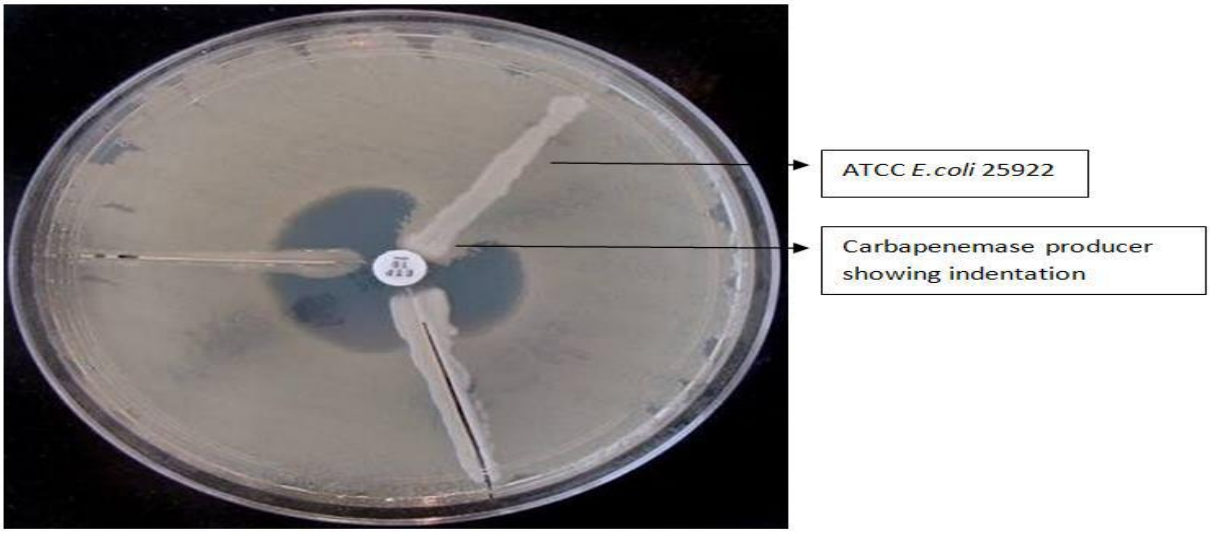
Table no.9: Disc diffusion - CLSI guidelines for Carbapenems:

Antibiotic	S (mm)	I (mm)	R (mm)
Enterobacteriaceae			
Meropenem	≥23	20-22	≤19
Imipenem	≥23	20-22	≤19
Pseudomonas aeruginosa			
Meropenem	≥19	16-18	≤15
Imipenem	≥19	16-18	≤15
Acinetobacter spp			
Meropenem	≥18	15-17	≤14
Imipenem	≥22	19-21	≤18

4.10. MODIFIED HODGE TEST:

- An overnight culture suspension of ATCC *E.coli*25922 was prepared in peptone water, matched to 0.5 McFarland turbidity standards, diluted to one in ten and inoculated as lawn culture over a 90mm MHA plate as for disk diffusion.[90]
- After waiting for 3-5 mins for drying, a Meropenem disc was placed at the centre of the plate.
- Using a loop which can deliver 10 microlitre, the test organism was taken and streak inoculated from the disk edge towards all four directions. 4 isolates were tested in a plate with a single Meropenem disc. The plate was incubated at 37°C for 16-20 hrs.
- The plates were examined the next day for enhanced growth around the test organism and the zone of inhibition giving a clover leaf appearance, which was indicative of Carbapenemase production[90] as shown in Figure no.6. The results were recorded and tabulated.

Figure no.6. Modified Hodge test



4.11. Ten disc method:[91]

This procedure helps in screening of a bacterial isolate for all β-lactamases (ESBLs, AmpC and Carbapenemases). Aztreonam (30µg), Cefotaxime (30µg), ceftazidime (30µg), Cefotaxime + clavulanic acid(30/10), ceftazidime + clavulanic acid(30/10µg), Ceftriaxone (30µg), Cefoxitin (30µg), Cefepime, Imipenem(10µg), Imipenem + EDTA are the drugs for which the sensitivity of the organisms is detected , by using Kirby Bauer disc diffusion assay.

Detection of ESBLs:

Ceftazidime or cefotaxime discs with and without clavulanic acid are used to detect ESBLs. If the zone increases by 5mm or above with clavulanic acid combination, the isolate is an ESBL producer.

Detection of AmpC β-lactamases:

Amp C β-lactamases are resistant to Cefoxitin and Cefotetan. High level AmpC producers are even resistant to Carbapenems and Aztreonam.[91]

Detection of Metallobetalactamases:

Imipenem or Meropenem discs with and without EDTA are used to screen for carbapenemases. If the zone increases by 7mm or above with EDTA combination, the isolate is an MBL producer.

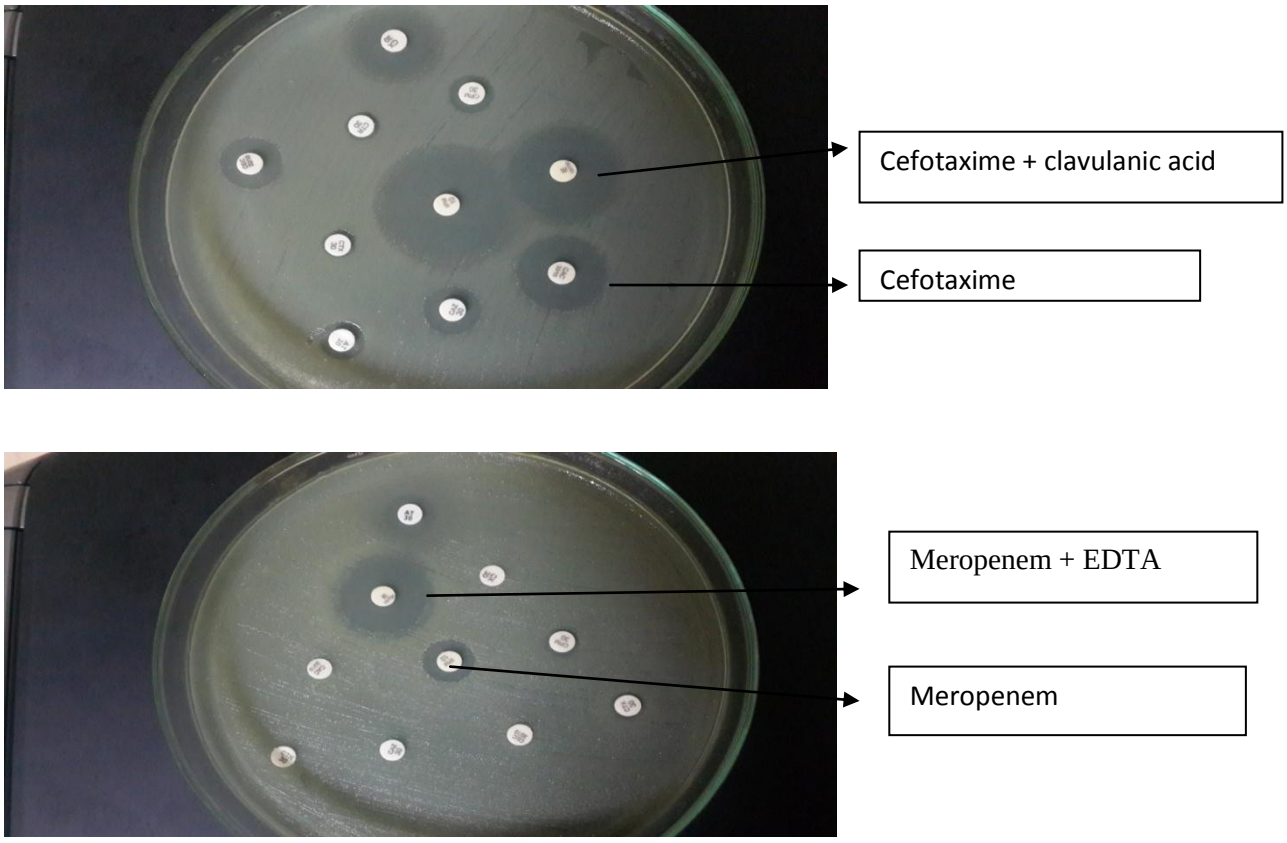


Figure no.7. Ten disc procedure

The data were entered in the Microsoft Excel sheet and analyzed using SPSS.

5.0. RESULTS

The study, **"Bacteriological Profile, Antibiogram and Risk Factors of Surgical Site Infections in a Tertiary Care Hospital"** was carried out in the Department of Microbiology, Trichy SRM Medical college Hospital and research centre, Trichy and the results were analyzed for the Surgical site infections (SSIs) rate as per class of wound, type of surgery, antibiotic prophylaxis, risk factors, drug resistance and American society of anesthesiologist index..

5.1. Prevalence of SSIs:

A total of 2076 patientsunderwent different types of surgeries comprising of elective as well as emergency during a 12-month period (May 2017 – April 2018). The types of surgeries done in this hospital during the study period are listed in the table no.10. During the 12 consecutive months of study period, 116 surgical site infections were documented and hence,the overall prevalence of surgical site infection rate during the study period was 5.6%(n=116). Among the 2076 surgeries, abdominal surgeries constituted (n =739; 35.6%) the highest rate of SSI occurred in the category of exploratory laparotomy. 78 underwent exploratory laparotomy, 20 developed SSIs (25.6%). The number of cases who developed SSIs in relation to type of surgery are shown in table no.10.

Table no.10: Types and number of surgeries carried out (May 2017- April 2018)

Site of surgery	Types of surgeries	No. of surgeries N=2076	SSI(5.6%) N=116
Abdomen (N=739)	Appendectomy	82(3.94%)	13(15.6%)
	Hernia repair	86(4.14%)	16(18.6%)
	Exploratory laparotomy	78(3.7%)	20(18.6%)
	Cholecystectomy	67(3.22%)	12(17.9%)
	LSCS	266(12.8%)	6(2.2%)
	Hysterectomy	160(7.7%)	4(2.5%)
Pelvis (N=154)	Sphincterotomy	43(2.0%)	2(4.6%)
	Hemorrhoidectomy	41(1.97%)	4(9.7%)
	Fistulectomy	39(1.87%)	2(5.1%)
	Hip replacement	31(1.58%)	6(19.3%)
Urogenital (N=91)	Transuretheral Resection of Prostate	25(1.2%)	2(8%)
	Uretheroscopic lithotripsy	66(3.17%)	Nil
Breast & axilla (N=85)	Modified Radical Mastectomy	24(1.1%)	3(12.5%)
	Fibroadenoma excision	61(3.02%)	Nil
Skin, Bone & Joints(N=302)	Knee replacement	47(2.26%)	4(8.5%)
	Varicose vein	41(1.97%)	Nil
	Open Reduction and Internal Fixation	214(10.3%)	13(6.0%)
Eye	Intraocular lens implantation	454(21.8%)	Nil
ENT (N=219)	Tonsillectomy	123(5.92%)	2(1.6%)
	Mastoidectomy	96(4.62%)	Nil
Neurosurgery		32(1.54%)	Nil
Total		2076	116

Table no.11: Comparison between site of surgery and organism isolated

Site	***S.aureus (32)***	***Entero cocci spp(3)***	***E. coli (27)***	***Kleb spp (19)***	***Proteus Spp (7)***	***Citrobacter spp (1)***	***Entero bacter spp (7)***	***P.aeruginosa (19)***	***A. baumanii (9)***
Abd	43%	100%	70%	52.6%	57.1%	100%	100%	52.6%	44.4%
Ortho	15%	-	30%	26.3%	28.5%	-	-	15.7%	22.2%
Pelvis	19%	-	-	21%	-	-	-	10.5%	22.2%
Breast	9%	-	-	-	-	-	-	-	-
ENT	12%	-	-	-	14.2%	-	-	10.5%	11.1%
Uro	-	-	-	-	-	-	-	10.5%	-

Abd- Abdomen, Uro- Urology, ENT- Ear, Nose and Throat.

All the above organisms were isolated in abdominal surgeries ranging from 43% to 100%. In pelvic surgeries, *Acinetobacter baumanii* and *Klebsiella spp* were commonly encountered whereas it was *E.coli* and *Proteus mirabilis* in orthopedic surgeries.

5.2. GENDERWISE DISTRIBUTION OF SSI:

Among the 1297males who underwent surgery, SSIs were seen in 84 (6.4%) of them and among the females (779) it was noticed in 32 (4.1%). The odd's ratio was 1.61. Distribution of cases in relation to gender is given in table no.12.

Table no.12: Distribution of cases in relation to gender

No	**Infected**	**Not infected**	**Total**
Males	**84**	**1213**	**1297**
Females	**32**	**747**	**779**
Total	**116**	**1960**	**2076**

5.3. AGE WISE DISTRIBUTION OF SSI*:*

The age of the study subjects ranged from 16 years to 72 years. 33 (28.4%) of them belonged to >55years of age followed by 29 (25%) and 25 (21.5%) in 35-44 years and 45-54 years respectively. The least belonged to below 35 years. The distribution of the SSI in relation to age group is depicted in figure no.8 and in relation to age group is given in table no.13. The odd's ratio for the development of SSIs among those below the age of 25 was 2.45.

Figure no.8: Age wise distribution of SSIs

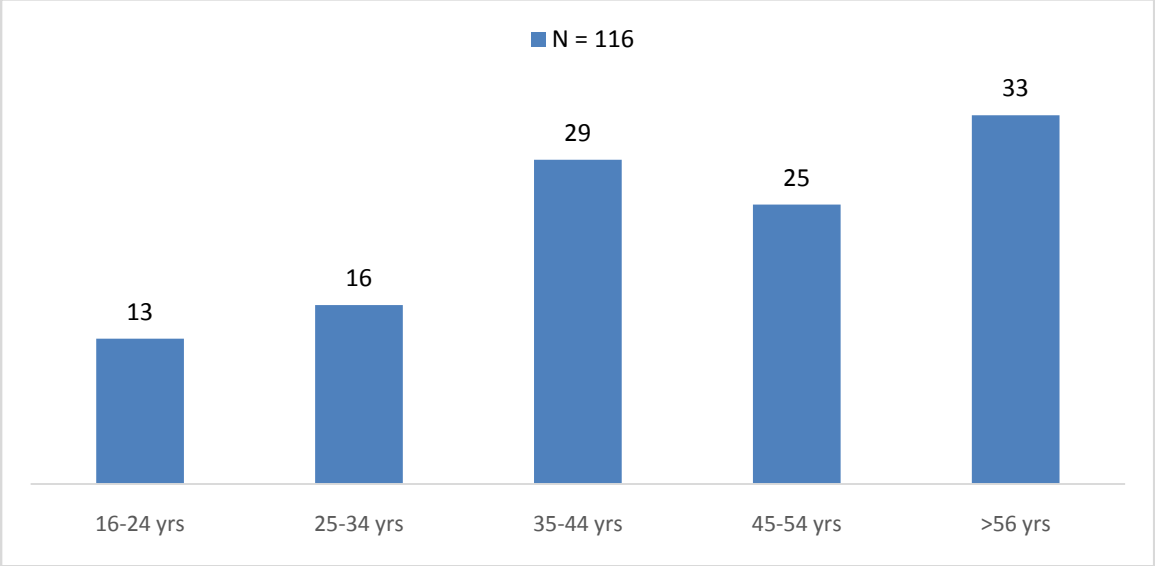


Table no.13: Distribution of SSIs and age group

Age group	No of cases	SSIs	%
16 – 24	**109**	**13**	**11.9**
25 - 34	**237**	**16**	**6.7**
35 – 44	**556**	**29**	**5.2**
45 -54	**501**	**25**	**4.9**
> 55	**673**	**33**	**4.9**
Total	**2076**	**116**	

5.4. COMPARISION OF SSI IN ELECTIVE VS EMERGENCY SURGERIES:

The present study which included 1820 elective surgeries and 256 emergency surgeries, in which SSI rate was 5.16% and 8.59% respectively. Emergency surgeries showed higher rate of SSI as compared to elective surgeries and shown in figure no.9. The odd's ratio was 0.57. The distribution of the cases and occurrence of SSIs are furnished in table no.14.

Figure no.9: Comparison of Elective vs Emergency surgeries

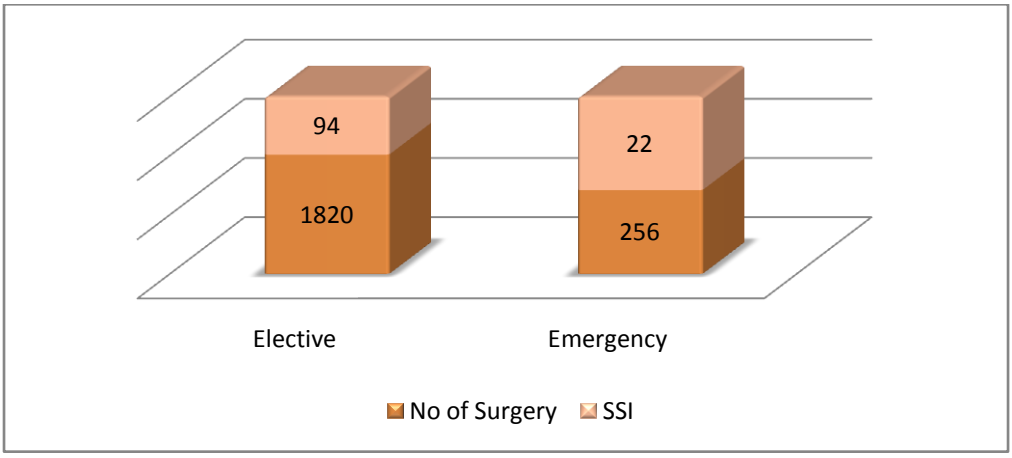


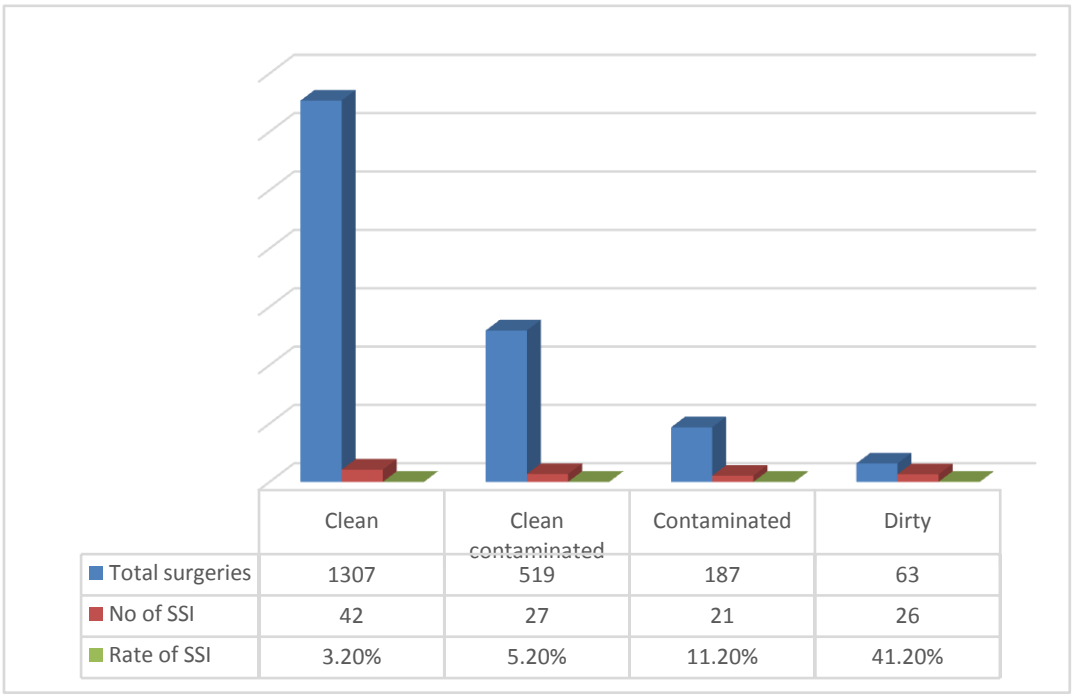
Table no.14: Distribution of SSIs and category of surgery

Category	Infected	Not infected	Total
Elective	**94**	**1726**	**1820**
Emergency	**22**	**234**	**256**
Total	**116**	**1960**	**2076**

5.5. DISTRIBUTION OF SSI BASED ON NATURE OF WOUND:

Among 2076 patients, 1307 underwent clean surgeries, of these 42 developed SSI (3.2%). The occurrence of SSIs among clean contaminated (n=519), contaminated (n=187) and dirty wounds (n=63) were 5.2%,11.2% and 41.2% respectively. The distribution of SSIs in relation to nature of wound is provided in figure no.10.

Figure no.10. Distribution of SSIs and nature of wound

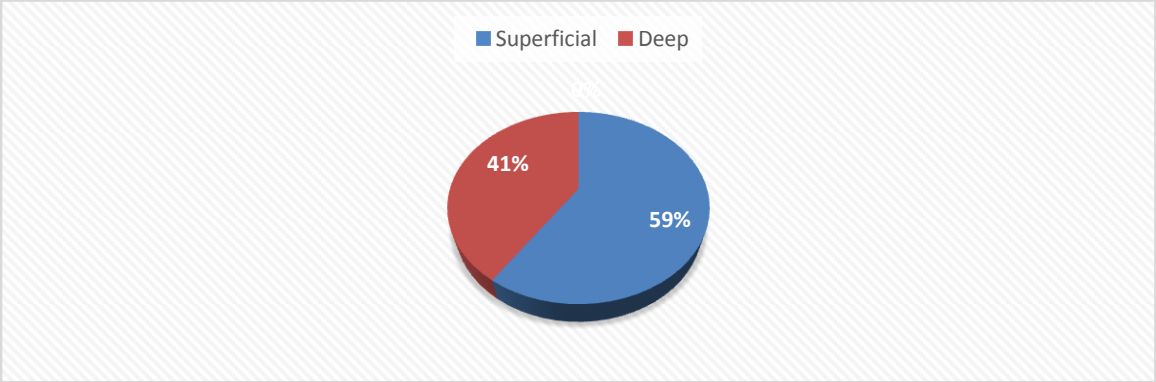


	Clean	Clean contaminated	Contaminated	Dirty
Total surgeries	1307	519	187	63
No of SSI	42	27	21	26
Rate of SSI	3.20%	5.20%	11.20%	41.20%

5.6. TYPE OF SSI BASED ON EXTENT OF WOUND:

As per CDC, SSI has been categorized into superficial, deep and organ/ space SSIs. In the present study, it was observed that 69 (59%) had superficial SSI and the rest (n=47) deep ones. There were no organ /space SSIs observedduring the study period and their distribution is depicted in figure no.11.

Figure no.11: Distribution of SSIs and extent of wound



5.7. RISK FACTORS OF SSIs:

All 116 SSI occurred in patients who had one or more risk factors like diabetes mellitus, smoking, alcohol, blood transfusion etc. Among them 7 (6.03%) had only single risk factor (diabetes mellitus), 23(19.8%), 40 (34.4%), 34(29.3%) and 12(10.3%) had combination of 2, 3, 4 and 5 risk factors respectively.

Table no.15: Distribution of risk factors among SSIs

Risk Factors	**SSI N = 116**	**Percentage**
Diabetes mellitus	**64**	**55.1%**
Smoking	**49**	**42.2%**
Alcoholism	**41**	**35.3%**
Anaemia	**31**	**26.7%**
Hospital stay **1-7 days** **>7 days**	 **27** **89**	 **23.2%** **76.7%**
Drain	**18**	**15.5%**

The distribution of cases with SSIs in relation to ASA score are provided in table no.16.

Table no.16: Distribution of ASA score along with SSIs

ASA	SSIs	%
I	16	13.7%
II	35	30.1
III	59	50.8
IV	6	5.17
V	Nil	Nil

Though all the cases received prophylactic antibiotics before and after surgery, 116 developed SSIs. The category of antibiotics used either alone or in combination and the development of SSIs are shown in table no.17.

Table no.17: Distribution of SSIs in relation to prophylactic antibiotic usage

Antibiotics	No	%
Single drug < 5 days	39	32.4%
Single drug > 5 days	22	11.2%
Multiple drug < 5 days	42	36.8%
Multiple drug > 5 days	13	19.2%

5.8. Laboratory works:

The occurrence of inflammatory signs were noticed on 4th day of surgery in 12 cases, 5th day in 76 cases and 6th day in 46 cases. Hence, the samples were collected from the respective cases and subjected to microbiological studies. Culture was positive among 116 of 134 samples. Among the 134 samples, 93 (69.4%) belonged to wound swabs and the rest (n=41; 30.6%) were wound aspirates. The details of the day of sample collection and its association with culture positivity are depicted in table no.18.

Table no.18: Day of sampling and surgical infections.

SL.NO	Day of sampling	Inflammatory signs	Culture report	%
1.	48 hrs (Day 2)	-	-	-
2.	96 hrs (Day 3)	-	-	-
3.	Day 4	12	05	41.2%
4.	Day 5	76	69	90.7%
5.	Day 6	46	42	91.3%

Gram staining:

These cases were classified into those who showed< 20 pus cells per oil immersion field or more than that. An attempt was made to find out the association between presence of pus cells and culture positive status. Microscopic studies of the gram stained smear showed pus cells in 122/134 (91%) and microorganisms in 37/134 (27.6%). The distribution of pus cell in relation to culture positive status is given in table no.19.

Table no.19: Distribution of pus cells and culture positivity

Culture status	Pus cells/oil immersion field		Total
	< 20	≥20	
Positive	32	84	116
Negative	13	05	18
Total	45	89	134

After distributing the data in2/2 table an attempt was made to find out positive predictable value. Positive predictable value for culture (0.27) was high among those who had ≥ 20 pus cells/ oil immersion field, thereby indicating that greater the number of pus cells more the chance of getting positive culture and irrespective of the presence of bacteria.

Table no.20: Association betweengram stain and culture positivity

Microorganisms in smear	Culture		Total
	+	-	
Present	32	5	37
Absent	84	13	97
	116	18	134

Since the smear studies were made at the bedside to look for pus cell and bacteria, an attempt made to distribute the results as shown in below table no.21. The odd's ratio was 0.99.

Table no.21: Association between pus cells and microorganisms in smear

Microorganisms in smear	Pus cells/ oil immersion field		Total
	<20	≥20	
Present	17	20	37
Absent	28	69	97
	45	89	134

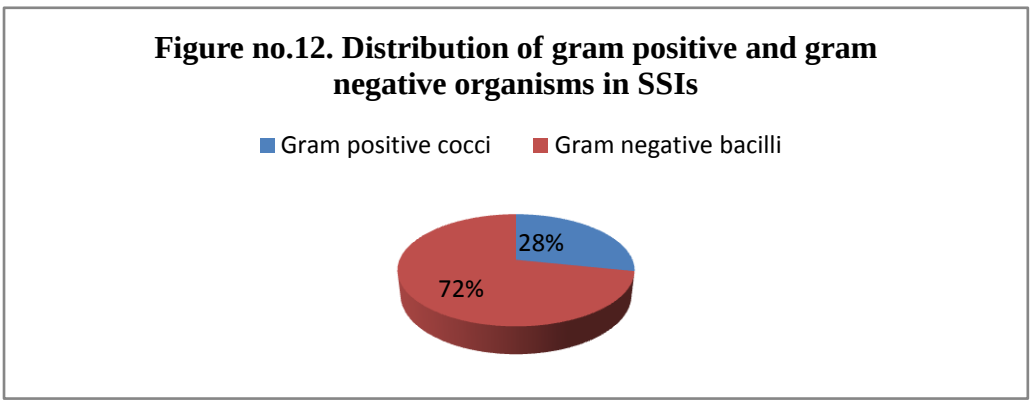
Subsequent analysis of the number of pus cells with smear studies revealed that the presence of pus cells were more important than seeing bacteria alone in gram staining. The odd's ratio was 2.09.

Higher culture positivity (72.4%) was seen in those patients whose smear had more no of pus cells and it was significant statistically (p=<0.01) in contrast to those who showed presence of bacteria but no pus cells. These observations indicate much weightage for the presence of pus cells. In otherwords, simple examination of discharge for pus cells may be a clue for SSIs for the practitioner.

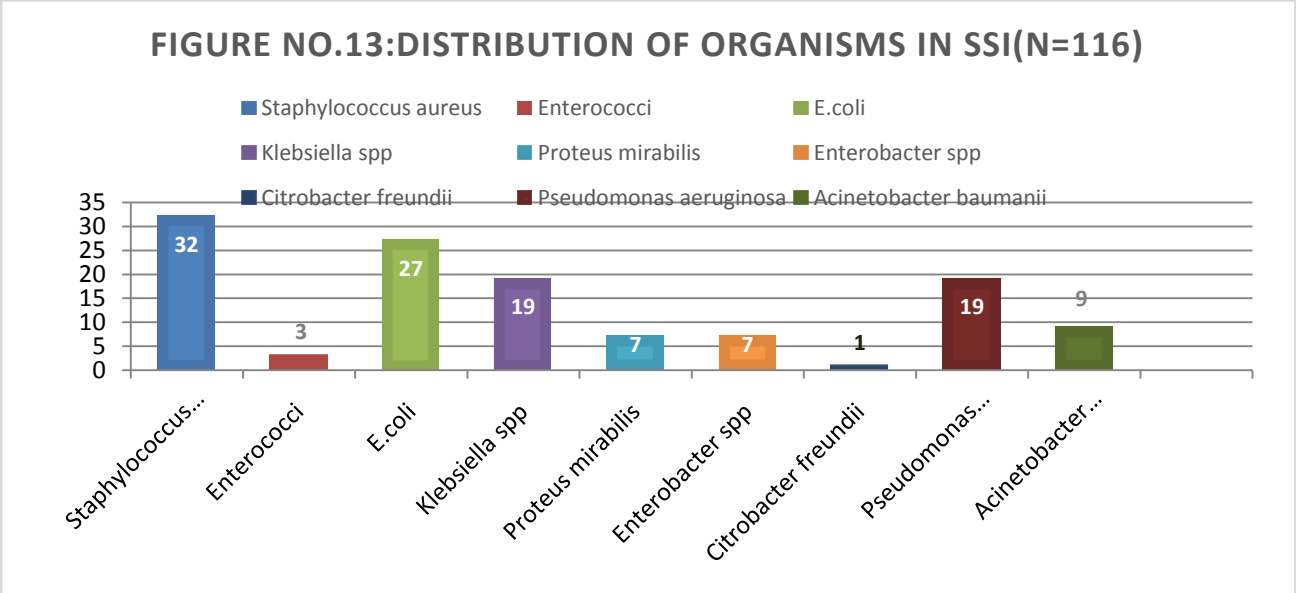
5.9. DISTRIBUTION OF VARIOUS BACTERIA IN SSI:

In our study, bacteria were isolated from 116/134 samples subjected to culture. 108 samples showed monomicrobial growth and 8 showed polymicrobial growth *(E.coli + Staphylococcus aureus =2, P.aeruginosa + E.coli =3, Acinetobacter baumanii + Staphylococcus aureus = 2, E.coli + Acinetobacter baumanii = 2)*. So, atotal of 124 isolates were obtained. Among them, 35(28.2%) were gram positive cocci. of the 89 gram negative bacilli, there were 61(68.5%) Enterobacteriaceae and

28 (31.4%) non fermentors. The details have been furnished in Figure no.12.below.



Among the 35 gram positive cocci 32(91.4%) were *Staphylococcus aureus* and 3 (8.5%) were *Enterococci* spp. Out of 61 *Enterobacteriaceae* 27(44.4%) were *E.coli*, 19(31.1%) *Klebsiella spp.*, which included 17 *Klebsiella pneumonia* and 2 *Klebsiella oxytoca*, 7(11.4%) *Proteus mirabilis*, 7 (11.4%) *Enterobacter spp* and 1(1.63%) *Citrobacter freundii*. The remaining 28 were Non fermenters: 19 (67.8%) *Pseudomonas aeruginosa* and 9 (32.1%) *Acinetobacter baumanii*. The isolates are depicted in figure no.13



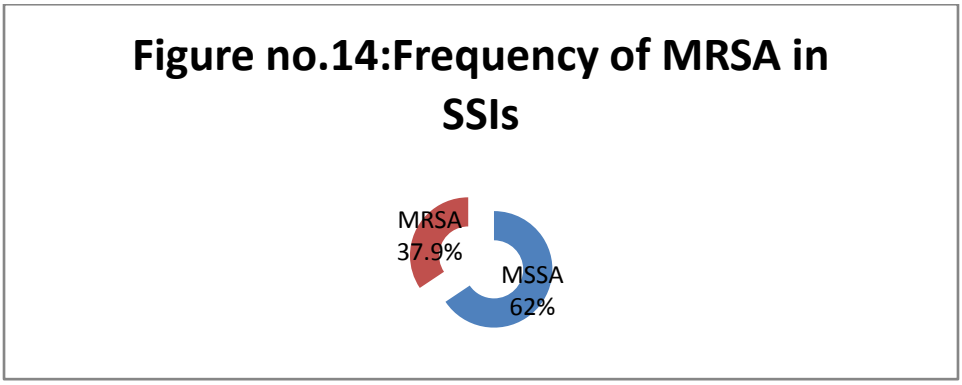
5.10. Antimicrobial susceptibility pattern:

There were 32 *Staphylococcus aureus* and 3 *Enterococcus spp* isolated during the study period and the sensitivity pattern is given in the table no.22.

Table no.22. Antimicrobial susceptibility pattern in gram positive cocci

Antibiotics	*Staphylococcus aureus* N=32	*Enterococcus spp* N=3
Penicillin (10U)	1.2%	0
Doxycycline (30µg)	43.7%	66.6%
Erythromycin (15µg)	46..8%	100%
Clindamycin (2µg)	40.6%	-
Gentamycin (10µg)	68.7%	-
Amikacin(30µg)	81..2%	-
Ciprofloxacin (5µg)	65.6%	33.3%
Cotrimoxazole(1.25/23.75µg)	37.5%	-
Tetracycline (30µg)	40.6%	33.3%
Linezolid (30µg)	100%	100%
High level gentamycin(120µg)	-	100%

Among 32 *Staphylococcus aureus* isolates from SSI, 11 were MRSA strains (37.9%) and the remaining 21 (62%) were MSSA as shown in fig no.14



MRSA – Methicillin resistant *Staphylococcus aureus*, MSSA- Methicillin sensitive *Staphylococcus aureus*

The antibiotic susceptibility of gram negative bacilli are furnished in table no.23 given below

Table no.23: Antibiotic susceptibility pattern in gram negative bacilli:

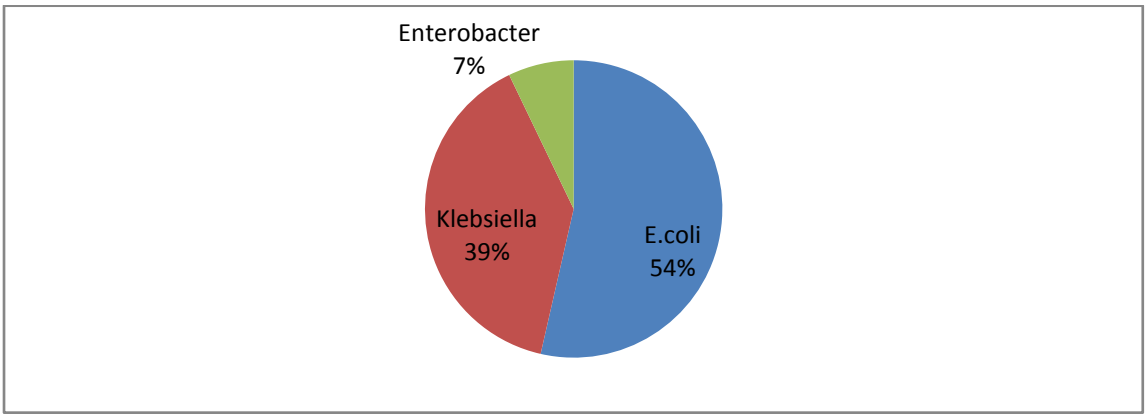
Antibiotics	***E.coli(27)***	***Kleb spp(19)***	***Proteus spp(7)***	***Enterobacter spp(7)***	***P.aeruginosa (19)***	***Acinetobacter spp (9)***
AMP	3.7%	0	0	14.2%	0	0
AMC	14.8%	0	0	14.2%	0	0
CIP	44.4%	31.5%	71.4%	71.4%	36.8%	22.2%
COT	55.5%	31.5%	71.4%	57.1%	31.5%	22.2%
GEN	66.6%	52.6%	57.1%	85.7%	47.3%	11.1
AK	77.7%	63.1%	71.4%	85.7%	52.6%	22.2%
CTR	40.7%	36.8%	85.7%	57.1%	ND	11.1%
CTX	40.7%	36.8%	85.7%	71.4%	ND	11.1%
CAZ	44.4%	42.1%	71.4%	57.1%	63.1%	22.2%
CPM	62.9%	57.8%	85.7%	85.7%	63.1%	22.2%
AT	85.1%	73.6%	71.4%	71.4%	68.4%	22.2%
CX	77.7%	73.6%	85.7%	71.4%	68.4%	33.3%
IPM	88.8%	84.2%	85.7%	85.7%	78.9%	33.3%
PIT	96.2%	89.4%	100%	85.7%	84.2%	55.5%

The gram negative organisms were further tested for production of various enzymes like ESBL, Amp C and MBL. The details are described in the ensuring paragraph.

5.11. DISTRIBUTION OF ESBL PRODUCING GRAM NEGATIVE BAILLI IN SSIs:

Out of 61 *Enterobacteriaceae*, 28 were ESBL producers (46%) on combined disc test. Among them 15 (53.5%) were *E.coli*, 11(39.2%) were *Klebsiella spp*, 2 (7.0%) were *Enterobacter spp* as shown in figure no.15

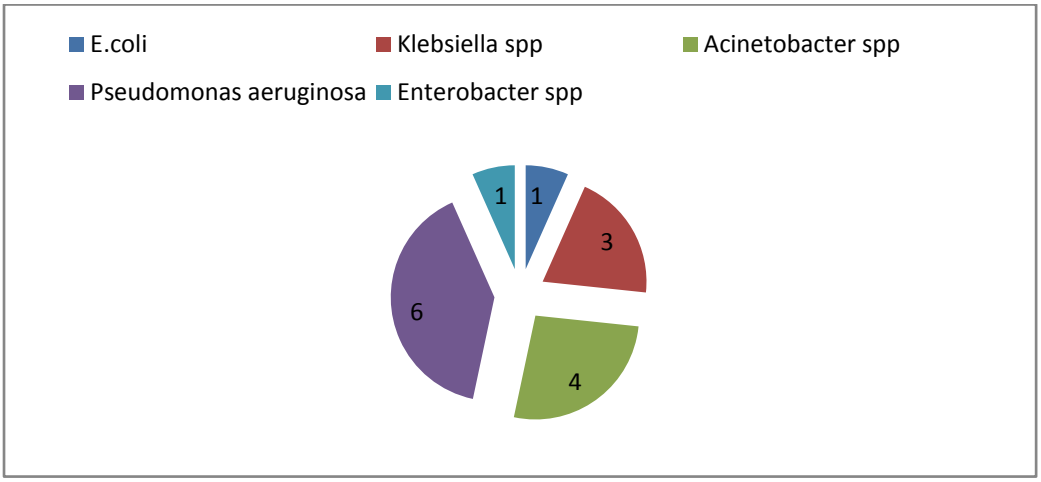
Figure no.15: Distribution of ESBL producers in SSIs



5.12. DISTRIBUTION OF AMP C PRODUCERS IN SSIs:

In the present study, out of 89 gram negative bacilli, 15(16.8%) were Amp C producers, out of which 6 were *Pseudomonas aeruginosa*, 4 *Acinetobacter baumanii*, 1*E.coli*, 3 *Klebsiella spp* and 1 *Enterobacter spp* which are depicted in figure no.16.

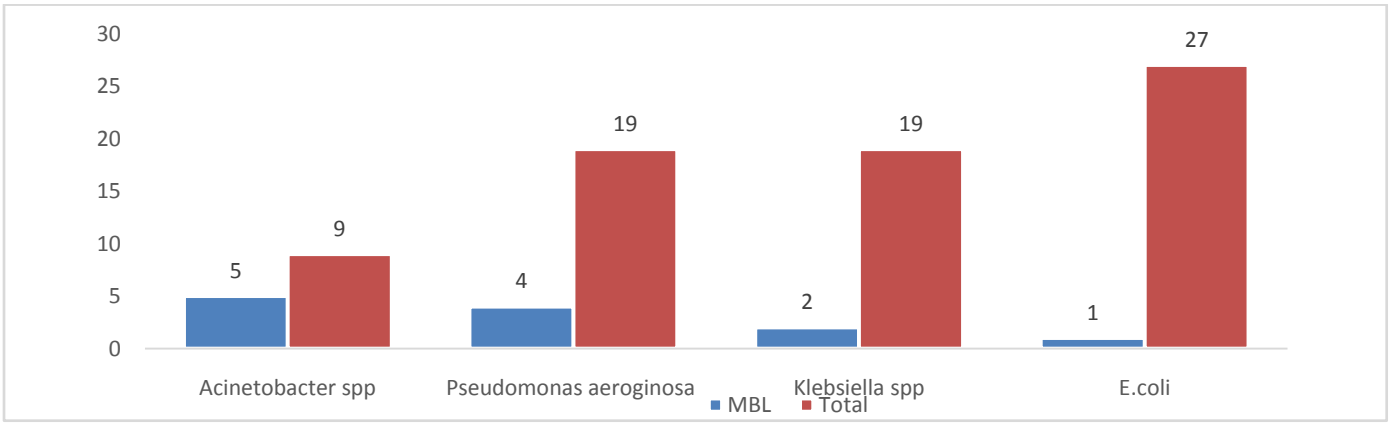
Figure no.16: Distribution of Amp C in SSIs



5.13. DISTRIBUTION OF MBL IN SSIs:

Out of 89 gram negative bacilli, 12 (13.2%) were Metallobetalactamase producers on Modified Hodge test as shown in the figure no.17. (5 out of 9 *Acinetobacter* spp (41.6%), 4 out of 17 *Pseudomonas aeruginosa* (33.3%), 1 out of 27 *E.coli* (8.3%) and 2 out of 19 *Klebsiella spp* (16.6%).

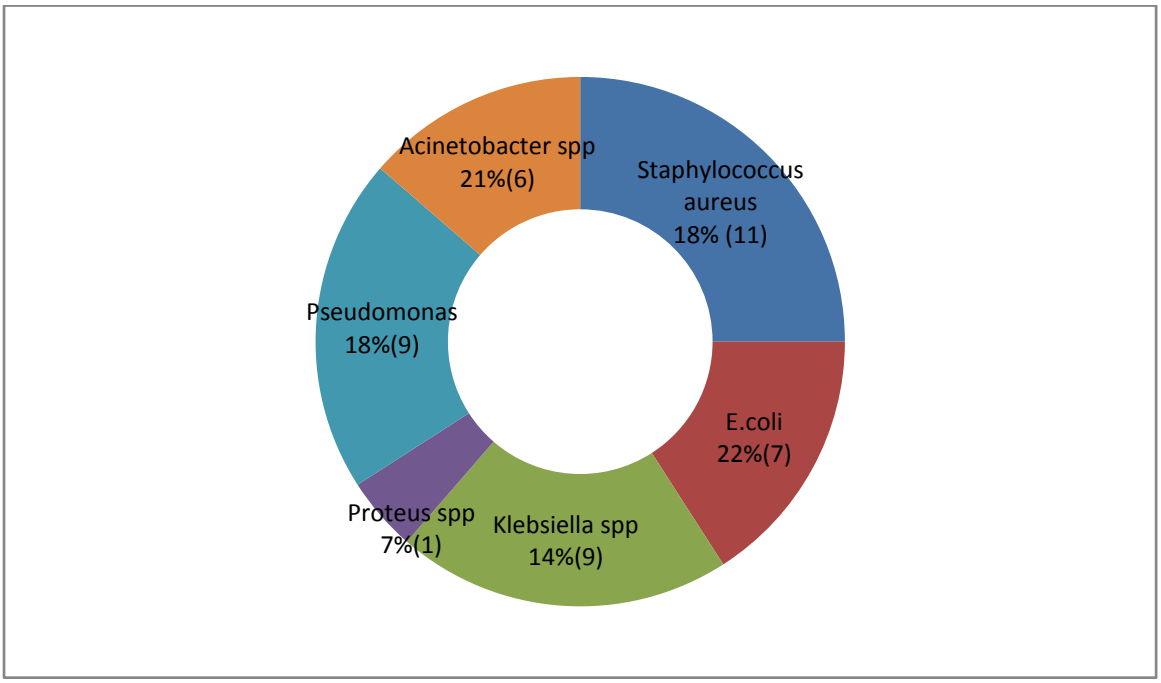
Figure no.17: Distribution of MBL producers in SSIs



5.14. DISTRIBUTION OF MULTIDRUG RESISTANCE IN SSIs:

Out of 124 isolates, 44 were resistant to more than 3 groups of antimicrobial drugs (35.4%) which included *Staphylococcus aureus* 11, *Pseudomonas aeruginosa* 9, *Acinetobacter spp* 6, *Klebsiella spp* 9, *E.coli 7*, *Proteus mirabilis* 2 and are shown in figure no.18.

Figure no.18: Distribution of MDR in SSIs



No MRSA carrier was identified in the present study. During the study period, none of them had hypothermia, hypoxia or shock status. Chlorhexidine bath preoperatively was not adopted for the cases. Razor was used for removal of hair for all patients undergoing surgery.

No significant difference was observed with regard to duration of surgery, experience of surgeon or excess trauma to the tissues as the surgeries were carried out

by senior surgeons. Standard aseptic procedureswere adopted by all surgeons and sterility of the operation theatre was monitored and maintained.

The patients were followed up from 24 hours after surgery till discharge with the help of respective surgeons for signs of local and systemic infection. Only 4 cases developed complications and underwent secondary surgery.

6.0. DISCUSSION

Surgical site infections (SSIs) are a worldwide problem that has far reaching implications on patient morbidity and mortality, and also has impact in thecost of treatment. It is the third most common nosocomial infection, and the frequency of SSIs varies from hospital to hospital. *Watanabe et al* reported SSIs in 15%[92]of their series whereas Leigh Neumayer et al reported 38%[93].

In our study, 2076 patients underwent various surgeries. Among them 739 patients underwent various abdominal surgeries like exploratory laparotomy, hernia repair, appendectomy, hysterectomy, etc. 302 patients had undergone orthopedic procedures like ORIF, hip & knee replacement etc. and 154 and 85 underwent pelvic and breast surgeries respectively.When compared with other studies, Allegranzi B et al, Azoury SC et al and Emil Aga et al also reported abdominal surgeries are commonly done and have high rates of surgical site infections.[95,96,97]Maksimović, J et al reported that orthopedic surgeries were more commonly associated with SSI.[98]

6.1. Prevalence of SSIs:

Among 2076, 134 patients showed local signs and symptoms and suspected to have postoperative wound infections.These caseswere evaluated and followed up. Among them culture was positive in 116(5.5%) cases and hence considered as cases of SSI in our hospital thus overall prevalence rate of SSIs was5.5%. Kumar et al and Fahad et al reported SSIs as 2.5% , which is only half of our present study rate[99,100]. The current status of SSIs identified in their hospital concurs with the studies of SarojGoliaet al, Faizan Iqbal et al and Degnim et al who reported it as 4.3%, 5.4% and

7.3% respectively.[101,102,103]On the contrary, Setty NH et al and Emil Aga et al reported it as 21.66% and 22.2%[101,96]. The comparative studies of SSIs is given in table no.24.

Table no.24: Prevalence of SSIs in different regions

Studies done	Year of Publication	Prevalence
Present study	2018	5.5%
Kumar A et al	2017	2.5%
Fahad A. et al	2014	2.55%
SarojGolia	2017	4.3%
Faizan Iqbal et al	2017	5.4%
Degnim AC	2012	7.3%
Setty NH et al	2014	21.66%
Emil Aga	2006	22.2%

6.2. Gender wise distribution of SSIs:

The occurrence of SSIs were more in males (6.4%) as compared with females (4.1%) in the present study. A study by Hernandez et al (2005) conducted in a Peruvian Hospital reported more among males 65.6%[104]. Moses also reported male preponderance (64.3%) and this is in contrast to the study by Shanmugam et al who reported almost equal among females (52%) and males (48%).[105,106] Increasing occurrence among males was attributable to nature of the infected wounds with which they come to surgical departments and also to more number of emergency among males.

6.3. Age wise distribution in SSIs:

In the present study, distribution of SSIs among the age groups 25 and above was almost nearer to each other and varied from 4.9% to 6.7%. On the contrary, it was more among those below 25 and may be attributable to the nature of wound. In general, occurrence of SSIs was more as age advances since these cases were suffering from Diabetes mellitus and/or other co morbid conditions which contributes to decreased physiological defense mechanisms and poor immune function. It is supported by many studiesfor example Owens et al and Bharatnur et al who reported that more number of SSIs occurred among 36 to 50 years (1.3 times higher risk of acquiring SSIs than the ones who were in the age group of 10 to 35 years)[107,108]. Similarly, high rate of infection was noted in the later age groups by Mundhada AS et al.[109,].

6.4. Comparison of SSI in Elective vs Emergency surgeries:

The present study includes 1820 elective surgeries and 256 emergency surgeries, and among them 94 (5.6%) and 22 (8.59%) developed SSI respectively. When the data was analyzed using 2/2 table it was noticed that the chances of development of SSIs were among emergency surgeries and odd's ratio was 0.57. The increased rate of SSI in emergency surgeries may be due to very narrow time span without proper patient preparation and surgical preparedness as well as contaminated wounds as in cases of road traffic accidents. The same has been citated in most of the studies done earlier on SSIs. Tabiri S et al also reported that emergency cases had higher number of SSIs (23.8%) as compared to elective cases (7.4%)[110,111]. In the

series of Dessie et al SSIs were reported in 61.7% emergency and 38.3% elective cases[112].

6.5. Distribution of SSI based on nature of wound:

Among 2076 patients, the number of clean, clean contaminated, contaminated and dirty surgeries were 1307, 519, 187 and 63 respectively. Dirty wounds (41.2%) had a higher rate of SSI followed by contaminated (11.2%), clean contaminated (5.2%) and clean (3.2%). These variations may be attributable to increased microbial load in the operative field which are of higher risk to SSIs. Similar to this study, Shrestha et al reported SSIs in2.9%, 15.3% and 18.7% of clean-contaminated, contaminated and dirty wounds respectively and none in clean wounds[113]. Dinda et al reported SSI rate as 5.5% for clean wounds, 8.8%, 20.1% and 29.9 % for clean-contaminated, contaminated and dirty wounds respectively[114].

6.6. Type of SSI based on the extent of wound:

In the present study, superficial and deep SSI were 69(59.4%) and 47(40.5%) respectively. Superficial SSI was found to be higher. Anusal kumar et al reported that superficial incision SSI was more prevalent (215 cases) 55.9% followed by deep incisional SSI (169 cases) 44% [99] and van Walraven et al reported the same with majority of these [n=8188, 57.5% of all SSIs] had a superficial component[115]. This is discordant to the study by Dessie W et al who reported superficial SSI as 42.1% and deep SSI as 57.9%[112].

6.7. Risk factors of SSI:

1. Diabetes mellitus – In our study 64(55.1%) diabetic patients had SSI. Many published reports have demonstrated that patients with diabetes are more susceptible to wound infection because of impaired neutrophil chemotaxis and phagocytosis. The occurrence of SSIs among diabetes in the present study concurs with study of Lilienfeld e tal, Talbot et al[116,117]and Akter Z et al who reported SSI among diabetes was 50%. On the contrary, the occurrence of SSIs was very high (91.7%) among diabetic patients in the series of Korol et al[118,119].
2. Smoking – In our study 49 (42.2%) had Smoking habit. It has been shown to be an independent risk factor for SSIs[120,121]. Smoking delays the healing of SSIs by causing local and systemic vasoconstriction and impair tissue oxygenation. This results in tissue hypoxia, an environment conducive to SSI and an adverse effect on wound healing. Korol et al and Prakash et al reported SSI rate of 63.2% and 66.7% respectively among smokers[119,122].
3. Alcoholism – In the present study 41(35.3%) of 116 SSIs were alcoholics. The present observations and the statement of Rantala et al were contradicted by Shabanzadeh et al who stated that alcohol did not affect SSIs and anastomotic leakage[123,124].
4. Prolonged postoperative hospitalization – In the present study 89 (76.7%) stayed for more than 7 days after procedure has been done. Anvikar et al. demonstrated that preoperative hospital stay predisposed an individual to 1.76% risk of acquiring an infection[125]Nichols RL et al says that prolonged postoperative hospitalization, which is a major concern of most of the

hospitals, has been evident in patients developing surgical site infection[126].This is related to altered cellular immune function as a result of hyperglycaemia and advanced glycation end products which result in impaired healing.

5. Anaemia contributed to 31(26.7%) cases of SSIs. Among these 16 received blood transfusion. It has been reported that perioperative transfusion of leukocyte-containing allogeneic blood components is an apparent risk factor for the development of postoperative bacterial infections, including SSI.[127]In three of five randomized trials conducted in patients undergoing elective colon resection for cancer, the risk of SSI was at least doubled in patients receiving blood transfusions.[128-130].Watanabe reported that 58.8 % blood transfused patients develop SSIs. The occurrence of SSIs among those who receive blood transfusion was attributable to immune dysregulation.
6. Drain – In our study with 116 SSIs, drain was kept only in 18(15.5%) cases.The use of surgical drains has been reported to be associated with the occurrence of SSIs[131,132]which was similar to Fujii et al who reported 14.3%. On the contrary, Cardosi et al reported SSIs in 22.4% who had drain[134].
7. **ASA index**- In our study, SSI incidence is higher in ASA III (n=59;50.8%) followed by ASA II(n=35 ;30.1%) and least in ASA I (n=16;13.7%).[48,49] The occurrence of SSIs were significantly more in patients with ASA II to V than in those with ASA I, which is in agreement with many studies,[135,136] suggesting that the ASA score before surgery has a strong influence on the occurrence of SSI rates in clean and clean contaminated cases.Watanabe reported SSIs in ASA II (24.1%) and ASA III (55.0%)[137].

6.8. Antibiotic prophylaxis – There is no standard guidelines for antibiotic prophylaxis for the surgeries. 3rd generation cephalosporins and gentamycin were given to all 71(%) abdominal surgeries and 23(%) received 3rd generation cephalosporins and metronidazole for pelvic surgeries. In thepresent study, prophylactic antibiotic was given to all 116 cases who had SSIs. Even though the patients received prophylactic antibiotics, they developed SSIs which may be due to differential pharmacokinetics of antibiotics, patient's own microbial load and other associated risk factors. Administration of a preoperative antibiotic did not decrease theoccurrence of SSI rate. Crawford CB et al noticed higher chances of occurrence of SSIs among those received prophylactic antibiotics. (12% SSI with antibiotics versus 4% without, $p < 0.0001$)[138].

6.9. Distribution of various bacteria in SSIs:

In our study, out of 124 isolates from 116 patients, 35(28.2%) were gram positive cocci, 89(76.7%) were gram negative bacilli. Among gram negative bacilli, *Enterobacteriaceae* contributed 61(49%), (27(34.4%) *E.coli*, 19 (31.1%) *Klebsiella spp*, 7 (11.4%) *Proteus mirabilis*, 7(11.4%) *Enterobacter spp* and 1(1.6%) *Citrobacter freundii)*. Non fermentor contributed 28(22.5%)(19 (15.3%) *Pseudomonas aeruginosa* and 9 (7.25%) *Acinetobacter baumanii)*. In our study, *Staphylococcus aureus* 32(25.8%) being the most common isolate followed by *E.coli* 27(21.7%), 19(15.3%) *Klebsiella spp*,19 (15.3%) *Pseudomonas aeruginosa* and 9 (7.25%) *Acinetobacter baumanii* and others. Our observations on higher isolation of *Staphylococcus aureus* (25.8%) tallied with Cantlon et al (2006) who also reported 26% of *Staphylococcus*

aureus. Rate of isolation of *Staphylococcus aureus* from SSIs in different series is shown in table no.25.

Tableno.25: Comparative analysis of *Staphylococcus aureus* infection in SSIs[141 -143]

Study	**Year of Publication**	**Isolation of *S.aureus***
1. Cooke et al	1979	30.3%
2. Oni et al.	1997	38.0%
3. Giacometti et al	2000	28.2%
4. Onche and Adedeji	2004	44.0%
5. Lilani et al	2005	56.3%
6. Oni et al	2006	29.0%
7. Cantlon et al	2006	25.8%
8.Suchitra et al	2009	33.0%
9. Shriyan et al	2010	63.0%
10. Mistelia et al	2011	29.5%
11. Present study	2017-18	25.8%

Though the *Enterobacteriaceae* was the second most frequently (49%) isolated organisms in the present study Cantlon et al noticed it to be low (12.4%).[139] Similar to our study, rate of isolation of *E.coli* 28(44%), *Klebsiella spp* 21(31.2%) and *Pseudomonas aeruginosa* 19 (67%) by Arias et al was nearer to the present study[99] whereas Rao and Harsha (1975) observed *P. aeruginosa*, *E. coli* and *Klebsiella* spp. as the common gram-negative organisms. Also, Giacometti et al(2000) noticed

Pseudomonas aeruginosa (25.2%) to be the predominant organism in their study followed by *Escherichia coli* (7.8%) and others.[140]

Surgical site infections caused by bacteria that are resistant to multiple classes of antimicrobials are an important and increasing problem. Organisms such as methicillin-resistant staphylococci, extended spectrum beta-lactamase producing Enterobacteriaceae and multi-drug resistant Acinetobacter and Pseudomonas spp. are among the current concerns; however, the emergence and dissemination of other multi-drug resistant organisms is likely to follow.

Among 32 staphylococcus aureus, 11(37.9%) were MRSA identified using cefoxitin disc diffusion method similar to the studies done by Ranjan (27.96%) [144], Krishna S (28.6%)[103] and Farrin 29%.[145] It is discordant with the study by Golia S et al who reported 88.8% of S. aureus as methicillin resistant strains[103].

Sanjay et al (2010) in their study on isolation and detection of drug resistance gram negative bacilli with special reference to postoperative wound infection noticed that *E. coli* was the predominant agent isolated from wound infections (37.3%), followed by *Pseudomonas aeruginosa* (20.9%), *Klebsiella spp* (17.2%), *Acinetobacter baumanii* (14.2%) and other agents were less common[146].

In the present study, none of the isolates *Klebsiella spp, Proteus spp, Acinetobacter baumanii and Pseudomonas aeruginosa* were sensitive to Ampicillin and Amoxyclav. *E.coli and Enterobacter spp* showed only 14.2% sensitivity to Amoxyclav. The sensitivity of *Acinetobacter baumanii* for different antimicrobial agents commonly ranged from 11% to 55%. The sensitivity was high to Piperacillin-tazobactam followed by Imepenem. In general, *Acinetobacter baumanii* was resistant

to fluoroquinolones, aminoglycosides, and all β-lactams, with the exception of the carbapenems and hence considered as the drug of choice.[147] with regard to *Acinetobacter spp.*

Brown et al noticed high resistance rate to many antimicrobial including carbapenem and it is emerging in many parts of the world,[149] mainly due to carbapenemases and possibly other mechanisms, such as alterations of outer membrane proteins[148] and these multiresistant *Acinetobacter* spp. may still retain susceptibility to the polymyxins (i.e., colistin and polymyxin B), sulbactam, and possibly tigecycline. Pan resistant isolates that are resistant to all available drugs are now being reported[150]. The prevalence of resistance is more in the Europe, America than in Asia/Pacific.

6.11. Distribution of ESBL producing gram negative bacilli in SSIs:

In the present study, 28/61(46%) were ESBL producers on combined disc test. Organisms were 14(53.5%) were *E.coli*, 11(39.2%) were *Klebsiella spp*, 2(7.0%) were *Enterobacter spp*. This is not in concurrence to the study by Rambabu et al who showed a prevalence rate of 35.71% ESBL producers *(E.coli – 56%, Klebsiella spp – 52%, Proteus spp – 40% and Enterobacter spp – 16%).* Asfia Sultan et al reported that 30% were ESBL. Prevalence of ESBL producers is high in a study by Golia et al who noticed 80% of E. coli and 100% of Klebsiella species [151-153]

6.12. Distribution of Amp C producers in SSIs:

In the present study 15(16.8%) were Amp C producers by disc test. 6 were *Pseudomonas aeruginosa*, 4 *Acinetobacter baumanii*, 3 *Klebsiella spp*, 1 *E.coli* and 1 *Enterobacter spp.* On the contrary, Hemalatha reported 9.2% Amp C producers which

was much lower than present study. Compared to ours Asfia Sultan et al and Tapan et al, reported very high prevalence (64.7%) and (48.5%) Amp C producers respectively.[154,152,153].

6.13. Distribution of MBL in SSIs:

In the present study 12(13.2%) were MBL producers. Among them were 5/9*Acinetobacter baumanii* (41.6%), 4/19*Pseudomonas aeruginosa* (21%), 1/27 *E.coli* (8.3%) and 2/19 *Klebsiella spp*(16.6%). Similar to our study Gupta reported 40 % of *A. baumannni* and 20% of *P.aeruginosa* isolates showed resistance to imipenem[155,156].

6.14. Distribution of multidrug resistance in SSIs:

In the present study 44(35.4%) isolates were resistant to three or more group of drugs and these MDR organisms were *Staphylococcus aureus* 11 (25%), *Klebsiella spp* 9(20%), *Pseudomonas aeruginosa* 9(20%), *E.coli 7* (15%), *Acinetobacter baumanii* 6(13.6%) *and Proteus mirabilis* 2(4.5%). In the series by Manyahiet al 63% (93/147) were multidrug resistant (MDR) whereas Zahran et al reported 37.2% of MDR isolates.[157,158].

The present study has revealed the prevalence of SSIs in our centre. The SSIs were noticed more among the patients who underwent abdominal surgeries the highest rate in laparotomy. SSIs were frequent among those who had one or other risk factors. Bacteriological studies revealed SSIs were more due to gram negative bacilli. The present study indicates that every institution has to maintain a surveillance of SSIs and to find out changing trends so as to curtail SSIs and infections due to MDR strains.

7.0. SUMMARY

This study entitled, **"Bacteriological Profile, Antibiogram and Risk Factors of Surgical site Infections in a Tertiary care hospital"**, was carried out in the department of Microbiology, Trichy SRM medical college hospital and research centre, Trichy from May 2017 to April 2018.

- Over a period of 12 months (May 2017 – April 2018), a total of 2076 patient underwent various surgeries. Among them, 134 patients were suspected to have SSI from various departments. 124 pathogens were recovered from 116 samples (8 were polymicrobial infections), the remaining 18 patients yielded no growth.
- Prevalence of SSI in our hospital was 5.6%
- Abdominal surgeries commonly lead to SSI especially laparotomy procedure (20/78; 25.6%) who had one or more risk factors.
- Emergency surgeries (8.5%) pose higher infection rate than elective surgeries (5.1%).
- SSI rate was high in dirty (41.2%) and contaminated wounds (11.2%) when compared to clean surgeries.
- Male predominance was seen in present study.
- 71.7% gram negative bacilli and 28.2% gram positive cocci were isolated. In that, *Staphylococcus aureus* accounted for 25.8%of SSI followed by *E.coli*21.7%, *Pseudomonas aeruginosa* 15.3%, *Klebsiella spp* 15.3% and others.

- Out of 32 *Staphylococcus aureus* 11(38%) were Methicillin resistant *Staphylococcus aureus*(MRSA).
- Gram negative bacilli which showed resistance to 3rd generation cephalosporins, cefoxitin and imepenem in routine antiobiotic susceptibility tests were subjected to phenotypic confirmatory test for ESBL, Amp C and MBL production.
- Phenotypic tests were performed on the 81 gram negative bacilli namely combined disc test, Amp C disk test and Modified Hodge test which showed 46%, 16.8% and 13.2% were ESBL, Amp C and MBL producers.
- 35.3% were MDR strains.

Suggestions:

As the study has brought out the occurrence of surgical site infections, it is time to decide and initiate regular surveillance of SSI on monthly basis and the same should be discussed in the Hospital Acquired Infection Control Committee meetings on departmental basis.

Based on the reports, measures to prevent and reduce the rate of SSIs which also serve on quality indicators and surveillance markers of hospital acquired infections.

The documents related to SSI shall be kept as a valuable document to defend the hospital and the surgeons when they are questioned by administrative, social, accrediting and legal authorities.

The present study reveals the usage of prophylactic antibiotics alone will not prevent the development of SSIs, as occurrence of SSI is a complex interplay of host factors, factors related Healthcare workers and environmental factors.

The study also stresses the importance of formulation of antibiotic policy based on the prevalent bacteria and their antimicrobial sensitivity pattern.

8.0. CONCLUSION

A total of 2076 patients underwent various surgeries including elective as well as emergency surgeries during consecutive 12 months commencing from May 2017 – April 2018. Standard methods were adopted to collect sociodemographic, clinical and microbiological data. SSIs were suspected in 134 patients. The clinical signs and symptoms started appearing from 4^{th} day onwards and more no of cases manifested features of infection either on 5^{th} or 6^{th} postoperative day. 18 samples showed no growth and the remaining 116 samples yielded 124 isolates (8 were polymicrobial infections).

The prevalence rate of SSI in our hospital during the study period was 5.6%.The SSI were more common in abdominal surgeries highest being in laparotomy surgeries (20/78; 25.6%). The odd's ratio for the development of SSIs in emergency cases was 0.57 and among males was 1.61. All these cases had one or other risk factors also.

The occurrence of SSIs was high in dirty (41.2%) and contaminated surgical wounds (11.2%) when compared to clean surgeries. Interestingly, SSIs were more among those belonging to age group 16-24 yrs (11.9%) and odd's ratio was 2.45. SSI was independent of prophylactic antibiotic administration. During the study period, SSIs developed in all patients who received prophylactic antibiotics thereby indicating that prophylactic antibiotics did not protect the individual from developing SSIs.

Smear studies of 134 samples revealed pus cells in all but smear had bacterial agents in only 37. For practical purposes, SSIs have to be considered

essentially if patients had clinical signs and symptoms locally and systemically, provided sample reveal pus cells more than 20/oil immersion field. From 116 SSIs, 124 isolates were obtained (monomicrobial – 108 and polymicrobial – 8). The isolates were gram positive which included *Staphylococcus aureus* (n=32) and *Enterococci* (n=3); and gram negative (n=89) which included Enterobacteriaceae (n=61) and non fermentors (n=28).

Among 32 *Staphylococcus aureus,* 11(38%) were Methicillin resistant *Staphylococcus aureus*. Gram negative bacilli which showed resistance to 3rd generation cephalosporins, cefoxitin and imepenem in routine antiobiotic susceptibility testing were subjected to phenotypic confirmatory test for ESBL, Amp C and MBL producers. Phenotypic test were performed on the 81 gram negative bacilli such as combined disc test, Amp C disk test and Modified Hodge test showed 46%, 16.8% and 13.2% of them were ESBL, Amp C and MBL producers. The prevalence of MDR strains during the study period was 35.3%.

Strengths of the study:

- The isolation and confirmation was monitored by two faculty members and guide.
- Phenotypic confirmatory test for ESBL, Amp C and MBL were done and it was more among general surgery cases.
- The works were monitored by all senior independently.
- Standard media and chemicals were purchased for lab works.
- Clinical correlation when analyzed with regard to SSI, it was noticed more among Diabetes mellitus, elders and those received blood transfusion.

Limitations of the study:

- It is a single center study confined to aerobic bacterial pathogens.
- Resistance genes of MDR strains were not considered during the study period.

Future study:

- Molecular epidemiology using genotypes of the isolates and its antimicrobial resistance is expected to reveal, geographic distribution of the resistant strains.
- It is suggested to work on anaerobic organisms among those cases admitted for treatment of road traffic accidents and penetrating injuries requiring surgical intervention.

ANNEXURE -III

ஒப்புதல் படிவம்

திரு-திருமதி-செல்வி.....

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என்ற முகவரியில் வசிக்கும் நான் சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையத்தின் நூண்ணுயிர்த்துறை சார்பில் நடத்தப்படும் அறுவை சிகிச்சைக்குப் பின் வரும் நோய்த்தொற்று சம்பந்தமான ஆராய்ச்சிக்கு நான் சம்மதித்து என்னிடம் சீழ் மாதிரி சேகரித்துக் கொள்ள சம்மதிக்கிறேன்.

இவ்வாராய்ச்சியைப்பற்றி எனக்கு தெளிவாகவும் விளக்கமாகவும் எடுத்துரைக்கப்பட்டது

இவ்வாராய்ச்சிக்கு இம்மருத்துவமனையிலிருந்து பணமோ, பொருளாலோ ஏதும் பெறவில்லை.

இவ்வாராய்ச்சியின் முடிவுகள் மருத்துவ ஆராய்ச்சிக்கும் மற்றும் மருத்துவ கல்விக்கும் பயன்படுத்தப்படும் என்பதை அறிந்துக்கொண்டேன்.

சாட்சி கையெழுத்து

கையெழுத்து

பெயர்

பெயர்

ANNEXURE- II

PROFORMA

Date:

1. Sl.no:
2. Name:
3. Age/Sex:
4. IP no/Ward/Unit:
5. Address:
6. Occupation:
7. Personal history smoker/ non smoker/ alcoholic/ non alcoholic
8. Diagnosis:
9. Risk factors: Blood glucose control in DM /existing infection/MRSA carrier/old age/obesity/ischaemia/ trauma/shock/hypothermia/hypoxia.
10. Preoperative risk factors: chlorhexidine bath taken/not taken/hair removal by electric clipper/razor/cream/ no of preoperative hospital days
11. Intraoperative risk factors: Duration of surgery/multiple assistance/experience of surgeon/tissue injury/blood transfusion.
12. Type of surgery:
13. Site of surgery:
14. Duration of surgery:
15. Cleaning & disinfection of OT: very good/good/fair
16. Adherence to aseptic procedure: yes/no
17. Prophylactic antibiotic: used/ not used
18. If used, antibiotic prescribed/dose/duration/route of administration
19. Educate the patient regarding incision care & SSI: yes/no

20. Local examination:redness/warmth/swelling/discharge

21. Microbiological examination:

22. Grams stain:

23. Culture & sensitivity:

24. Follow up: improved/ not improved

KEY WORDS TO MASTER CHART:

RISK FACTORS- 1- DIABETES, 2- SMOKING, 3- ALCOHOLISM, 4- ANAEMIA, 5- BLOOD TRANSFUSION, 6-DRAIN, 7-HOSPITAL DAYS.

TYPES OF SURGERY- C- CLEAN, CC- CLEAN CONTAMINATED, CO- CONTAMINATED, D- DIRTY.

ORIF- OPEN REDUCTION AND INTERNAL FIXATION, TURP- TRANSURETHERAL RESECTION OF PROSTATE, LSCS- LOWER SEGMENT CAESAREAN SECTION, MRM- MASTOIDECTOMY,TONSIL- TONSILLECTOMTY, CSOM- MASTOIDECTOMY

SUR- SURGERY, PA- PROPHYLACTIC ANTIBIOTICS, A- CEFOTAXIME, B- CEFTRIAXZONE, C- GENTAMYCIN, D- METRONIDAZOLE.

ND- NOT DONE

SL.NO	AGE	SEX	IP NO	WARD	DIA/PRO	RISK FACTOR	TYPE	PA	ORGANISM	P	DO	E	CD	G	AK	CIP	COT	TET	LZ	HLG	FOLLOW UP	
1	47	F	247927	SUR	APPENDICITIS	1,4,5,6,7	CO	A	MRSA	R	S	R	R	R	S	S	R	R	S	ND	IMP	
2	35	M	248224	SUR	HERNIA	1,2,3,7,6	CO	BC	MRSA	R	S	S	S	R	S	S	S	S	S	ND	IMP	
3	21	M	248195	SUR	APPENDICITIS	2,3	CO	A	MRSA	R	R	R	R	S	S	R	S	S	S	ND	IMP	
4	36	M	195329	SUR	HERNIA	2,3,6	CC	BC	ENTERO	R	S	S	ND	ND	ND	R	ND	R	S	S	IMP	
5	25	F	243843	SUR	CHOLECYST	6,7	CO	A	MRSA	R	S	S	S	S	R	S	S	S	S	ND	IMP	
6	67	F	138745	ORTHO	ORIF	1,4,6,7	CC	AC	MSSA	R	S	S	S	R	S	S	S	S	S	ND	IMP	
7	37	M	246414	SUR	CHOLECYST	1,2,3,7	CO	B	MRSA	R	S	S	S	S	R	S	R	R	S	ND	IMP	
8	29	F	246471	SUR	APPENDIcits	4,5,7	CC	A	ENTERO	R	S	S	ND	ND	ND	S	ND	S	S	S	IMP	
9	40	M	246337	SUR	HERNIA	1,2,3,7,6	CC	BC	MSSA	S	S	S	S	S	R	S	S	S	S	ND	IMP	
10	52	M	247046	SUR	LAP	1,2,3,7,6	D	CA	MSSA	S	S	S	S	R	R	S	S	S	S	ND	AMA	
11	47	F	139402	SUR	MRM	1,4,7	C	A	MRSA	R	S	S	S	S	S	S	S	S	S	ND	IMP	
12	41	M	249411	SUR	HERNIA	1,2,3,6,7	CC	AC	MRSA	R	S	S	S	R	R	S	R	R	S	ND	ABSCOND	
13	47	M	227683	SUR	HERNIA	1,2,3,7,6	CC	BC	MRSA	R	S	S	S	R	S	S	S	S	S	ND	IMP	
14	34	F	180432	SUR	MRM	4,5,7	C	A	MSSA	S	S	S	S	S	S	S	S	S	S	ND	IMP	
15	39	M	227722	ORTHO	ORIF	2,6	CC	BC	MSSA	R	S	S	S	R	S	S	R	S	S	ND	DIED	
16	27	M	227678	SUR	FISTULA	2,6	CC	D	MSSA	R	S	S	S	S	S	S	R	S	S	ND	IMP	
17	48	F	246484	SUR	MRM	1,4,5	C	B	MRSA	R	S	S	R	S	S	R	S	R	S	ND	IMP	
18	44	M	2867	SUR	LAP	1,2,3,6,	D	A	ENTERO	R	R	S	N	N	N	R	ND	R	S	S	IMP	

			98			7		C					D	D	D							
19	22	M	247985	ENT	CSOM	2,3	CC	A	MSSA	R	S	R	S	R	S	S	S	S	S	ND	IMP	
20	42	M	248211	SUR	APPENDIX	1,3,7,6	CC	B	MSSA	R	S	S	S	S	S	S	S	S	S	ND	IMP	
21	64	M	277451	SUR	HEAMORRHOID	1,2,3,6,7	CC	D	MSSA	R	R	S	S	S	S	S	S	S	S	ND	IMP	
22	41	M	247782	ORTHO	ORIF	1,2,3,7,6	CC	BC	MSSA	R	S	S	S	S	S	S	S	S	S	ND	IMP	
23	20	M	245285	SUR	APPENDIX	6,2	CC	A	MSSA	R	S	S	S	S	S	S	S	S	S	ND	ABSCOND	
24	61	M	244910	ORTHO	ORIF	1,2,3,7	C	BC	MSSA	R	S	S	S	S	S	S	S	S	S	ND	AMA	
25	39	M	231551	SUR	CHOLECYST	2,3,6,7	CC	B	MSSA	R	R	S	S	S	S	S	S	S	S	ND	IMP	
26	57	M	230552	ORTHO	KNEE	1,2,3,6	CC	BC	MSSA	R	S	S	S	S	S	S	S	S	S	ND	IMP	
27	21	M	138205	ENT	CSOM	2	C	B	MRSA	R	S	R	R	R	S	R	S	R	S	ND	IMP	
28	43	M	245521	SUR	HERNIA	1,2,3,7	CC	BC	MSSA	S	S	S	S	S	S	S	S	S	S	ND	IMP	
29	29	M	265677	ENT	CSOM	7	C	A	MSSA	R	S	S	S	S	S	S	S	S	S	ND	IMP	
30	21	M	246485	ENT	CSOM	6,7	C	A	MRSA	R	R	R	R	S	R	R	S	R	S	ND	IMP	

AGE	SEX	IP NO	WARD	PRO/DIA	TYPE	RISK FACTOR	PA	ORGANISM	AMP	AMC	G	AK	COT	CIP	CAZ	CTR	CTX	AT	IPM	PIT	CPM	CX	FOLLOW UP
17	M	143359	ENT	MASTOIDECTOMY	C	7,6	B	PSEUDO	R	R	S	S	S	S	S	N	N	S	S	S	S	S	IMP
59	M	245927	SUR	FISTULECTOMY	CC	1,7,6	DA	E.COLI	S	S	S	S	S	S	R	S	S	S	S	S	S	S	IMP
38	M	247239	SUR	SPHINCTER	CC	2,3,	DA	E.COLI	R	S	S	S	S	S	S	S	S	S	S	S	S	S	IMP
65	M	247226	SUR	HERNIOPLASTY	CC	1,2,7,6	A	PSEUDO	R	R	S	S	S	S	S	N	N	S	S	S	S	S	IMP
63	F	247707	SUR	LAPAROTOMY	D	1,7,6	AC	PSEUDO	R	R	S	R	S	S	S	N	N	S	S	S	S	S	IMP
37	F	237740	SUR	CHOLECYSTEC	D	4,5,7,6	B	ACINETO	R	R	S	R	S	S	S	R	S	S	S	S	R	S	IMP
61	M	251474	SUR	LAPAROTOMY	D	1,7,6	AC	E.COLI	R	R	S	R	R	R	R	S	R	R	R	R	R	R	IMP
56	M	252174	SUR	LAPAROTOMY	D	1,2,3	AC	KLEB	R	R	S	R	S	S	S	S	R	S	S	S	S	S	IMP
54	M	252174	SUR	APPEN	CC	1,7,6	A	E.COLI	R	S	R	S	S	S	S	R	R	S	S	S	S	S	IMP
41	M	252088	SUR	LAP	D	7,6	A	KLEB	R	R	S	R	S	R	S	S	S	S	S	S	S	S	IMP
57	F	246929	SUR	HERNIA	CC	1,4,5,7,6	AC	ACINETO	R	R	R	S	S	S	R	S	S	S	S	S	S	S	IMP
51	M	272088	SUR	APPENDIX	C	1,2,3	B	E.COLI	R	S	S	S	S	S	S	R	S	S	S	S	R	R	DIED
59	F	246929	SUR	LAP	D	1,4,5,7,6	BC	KLEB	R	R	S	R	S	R	S	S	S	S	S	S	S	S	AMA
43	M	296946	SUR	LAP	D	1,2,3	B	E.COLI	R	R	S	S	S	S	S	R	R	S	S	S	S	S	IMP
31	F	252586	SUR	APPENDIX	CC	4,5,7,6	B	KLEB	R	R	S	R	S	S	R	R	S	S	S	S	S	S	IMP
55	M	251335	SUR	HAEMOR	CC	1,2,3,7,6	D	KLEB	R	R	S	R	R	R	S	S	S	S	S	S	S	S	IMP
29	M	251367	SUR	CHOLECYST	CO	7,6	A	CITRO	R	R	R	S	S	S	S	S	S	S	S	S	S	S	IMP
61	F	252605	SUR	CHOLECYST	CO	1,7,6	A	E.COLI	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
51	M	252437	SUR	HERNIA	CC	1,2,3,7	B	PROTEUS	R	R	S	S	S	R	R	S	R	S	R	S	R	S	IMP
21	F	282437	OG	LSCS	CC	4,5,7,6	BC	E.COLI	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
39	M	292883	SUR	HAEMORRHOID	C	6,7	D	KLEB	R	R	S	S	S	R	S	R	S	S	S	S	S	S	IMP
48	F	252066	OG	HYSTER	CC	1,4,5,7,6	A	KLEB	R	R	S	S	R	S	S	S	R	S	R	S	S	R	AMA
68	M	252142	SUR	LAPROTOMY	D	1,7,6	AC	E.COLI	R	R	S	S	S	S	R	S	S	S	S	S	S	S	IMP
71	F	259913	ORTHO	HIP	C	6,7	B	KLEB	R	R	S	S	S	S	S	S	R	S	S	S	S	S	DIED
32	F	233483	OG	LSCS	CC	6,7	AC	PROTEUS	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
55	M	252066	SUR	HERNIA	CC	1,2,3	B	E.COLI	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
63	F	253483	SUR	HERNIA	C	1,4,7,6	B	KLEB	R	R	S	S	R	S	S	S	S	S	S	S	S	S	IMP
51	F	252064	OG	LSCS	C	1,4,7,6	BC	E.COLI	R	R	R	R	R	R	R	R	R	R	R	R	R	R	IMP

29	M	249913	SUR	FISTULA	C	2,3	D	PSEUDO	R	R	S	R	R	S	S	N	N	S	S	S	R	S	IMP
46	M	249712	SUR	APPENDIX	CO	6,7	A	ACINETO	R	R	R	S	R	R	S	R	R	R	S	S	R	S	IMP
64	F	253403	ORTHO	KNEE	CO	1,4,5,7,6	B	E.COLI	R	R	S	S	S	S	S	S	R	S	S	S	S	S	IMP
56	M	242742	SUR	FISSURE	C	1,2,3,7,6	D	ACINETO	R	R	R	R	R	R	R	R	R	R	R	R	R	R	IMP
30	F	150832	OG	LSCS	C	6,7	BC	KLEB	R	R	S	S	R	S	R	R	R	S	S	S	S	S	IMP
54	M	253198	SUR	LAPROTOMY	D	1,2,3,7,6	CA	KLEB	R	R	R	S	R	R	R	R	R	S	S	S	S	S	IMP
46	F	231837	SUR	CHOLECYST	C	4,5,7,6	B	E.COLI	R	R	S	S	S	S	S	R	S	S	S	S	S	S	IMP
21	M	251830	SUR	APPENDIX	CO	2,	B	ACINETO	R	R	R	R	R	R	R	R	R	R	R	S	R	R	IMP
33	F	253057	OG	LSCS	C	4,7,6	BC	PROTEUS	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
58	M	253415	SUR	LAPROTOMY	D	1,7,6	AC	E.COLI	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
31	F	257045	SUR	HAEMORRHOID	C	2,3,7,6	D	PSEUDO	R	R	R	S	R	S	S	N	N	S	S	S	S	S	AMA
69	M	256719	SUR	LAPRO	D	1,7,6	B	E.COLI	R	R	S	S	R	R	R	S	R	R	S	S	S	S	IMP
28	F	255736	OG	LSCS	C	6,7	AC	PSEUDO	R	R	S	R	S	S	S	N	N	S	S	S	S	R	IMP
19	M	256436	ENT	TONSIL	C	6,7	A	PROTEUS	R	R	S	S	R	S	S	S	S	S	S	S	S	S	IMP
53	M	255585	SUR	CHOLECYST	CO	1,2,3,7,6	A	PSEUDO	R	R	R	R	R	R	S	N	N	S	S	S	S	R	IMP
66	M	254818	SUR	HERNIA	C	1,7,6	A	E.COLI	R	R	S	S	S	R	S	R	S	S	S	S	S	S	IMP
36	M	256028	SUR	APPENDIX		2,3,7	B	PROTEUS	R	R	R	S	S	S	S	S	S	S	S	S	S	S	AMA
27	M	256221	SUR	LAPRO	CO	2,	AC	KLEB	R	R	R	S	R	R	R	R	R	S	S	S	R	R	IMP
65	M	254303	SUR	LAPRO	CO	1,2,3	AC	PSEUDO	R	R	R	R	R	S	S	N	N	R	S	S	S	S	IMP
37	M	255605	ORTHO	ORIF	C	2,	AC	KLEB	R	R	R	S	R	R	R	R		S	S	S	R	S	ABSCOND
39	M	256453	SUR	APPENDIX	CO	2,3,7	A	E.COLI	R	R	S	S	S	S	R	S	R	S	S	S	S	S	AMA
68	F	186192	SUR	LAPROTOMY	D	1,7	AC	PSEUDO	R	R	S	R	R	R	R	N	N	S	S	R	S	R	IMP
51	F	186240	OG	LSCS	C	1,7	A	PSEUDO	R	R	R	S	R	R	S	N	N	R	S	R	S	R	IMP
28	F	258083	ORTHO	0RIF	CO	4,5	BC	PSEUDO	R	R	S	R	R	R	R	N	N	S	S	R	S	R	IMP
71	M	258858	SUR	HERNIA	CO	1,7	BC	E.COLI	R	R	S	S	R	R	S	R	S	S	S	S	S	S	IMP
34	M	257126	ORTHO	ORIF	C	2,	BC	PSEUDO	R	R	R	S	R	R	S	N	N	R	R	S	R	S	IMP
51	M	257341	SUR	LAPROTOMY	D	1,2,3	B	ENTEROBACTER	S	S	S	S	R	S	S	R	S	S	S	S	S	S	IMP
65	M	257221	SUR	LAPROTOMY	D	1,7	BC	ENTEROBACTER	R	R	S	S	S	S	S	S	S	S	S	S	S	S	IMP
37	M	257984	ORTHO	ORIF	D	2,	BC	PROTEUS	R	R	R	R	S	S	S	S	S	R	S	S	S	S	IMP
48	F	258100	ORTHO	HIP	C	1,7	A	PSEUDO	R	R	R	R	S	R	S	N	N	S	S	S	S	S	IMP

24	F	258099	OG	LSCS	C	4,5,7	BC	ENTEROBACTER	R	R	S	R	S	S	R	S	R	R	S	S	S	R	IMP
67	M	254862	UROLOGY	TURP	C	1,7	D	PSEUDO	R	R	R	S	R	R	S	N	N	R	S	S	S	S	IMP
44	M	251066	SUR	HERNIA	CO	2,	AC	E.COLI	R	R	S	S	R	R	R	R	R	S	S	S	R	S	IMP
38	F	257860	ORTHO	HIP	C	7,	BC	ACINETO	R	R	R	R	R	R	R	R	R	R	R	S	R	R	IMP
58	M	257673	SUR	CHOLECYST	CO	1,7	A	E.COLI	R	R	S	S	R	R	R	R	R	S	S	S	R	S	IMP
38	M	257636	SUR	HERNIA	CO	2,3	BC	KLEB	R	R	R	S	R	R	R	R	R	S	S	S	R	S	IMP
33	M	258089	ORTHO	ORIF	D	2,3	B	PROTEUS	R	R	R	R	R	R	S	R	R	R	S	R	S	R	IMP
56	M	257677	SUR	HERNIA	C	1,7	B	ENTEROBACTER	R	R	R	S	S	S	S	S	S	S	S	S	S	S	AMA
49	M	257462	UROLOGY	TURP	C	1,2,3	A	PSEUDO	R	R	R	S	R	R	R	N	N	R	S	S	R	R	IMP
39	F	255696	ORTHO	ORIF	D	5,7	BC	E.COLI	R	R	R	S	R	R	R	R	R	S	S	S	S	S	DIED
16	M	256781	ENT	TONSILECTOMY	C	7	A	ACINETO	R	R	R	R	R	R	R	R	R	R	R	R	R	R	IMP
60	M	256397	SUR	HERNIA	C	1,7	B	E.COLI	R	R	R	S	R	R	R	R	R	S	S	S	R	S	ABSCOND
41	M	256931	SUR	CHOLECYST	CO	1,2,3	A	PSEUDO	R	R	R	R	R	R	R	N	N	R	S	S	R	S	IMP
61	M	256138	ORTHO	HIP REPLACE	D	1,7	BC	KLEB	R	R	R	S	R	R	R	R	R	R	S	S	R	S	IMP
40	M	247014	SUR	LAPROTOMY	D	1,2,3,7	BC	PSEUDO	R	R	S	S	R	R	R	N	N	S	R	S	R	S	IMP
53	M	248919	ORTHO	HIP REPL	C	1,	A	E.COLI	R	R	R	R	R	R	R	R	R	R	R	S	R	R	IMP
45	M	244826	SUR	LAPROTOMY	C	1,2,3,7	BC	ENTEROBACTER	R	R	S	S	S	S	R	R	S	S	S	R	S	S	IMP
57	M	819666	ORTHO	KNEE FRAC	D	1,7	AC	KLEB	R	R	R	S	R	R	R	R	R	R	S	S	R	S	IMP
53	M	231522	SUR	CHOLECYST	C	1,2,7	B	E.COLI	R	R	R	R	R	R	R	R	R	S	S	S	R	S	IMP
43	M	231987	ORTHO	ORIF	D	7	AC	ACINETO	R	R	R	R	R	R	R	R	R	R	R	R	R	R	IMP
22	M	249187	ENT	CSOM	C	6,7	A	PSEUDO	R	R	R	S	R	R	R	N	N	S	R	S	R	S	IMP
51	M	232193	SUR	CHOLECYST	C	1,7	B	PSEUDO	R	R	S	R	S	R	R	N	N	S	R	S	R	S	IMP
43	M	249235	ORTHO	ORIF	C	6,7	BC	E.COLI	R	R	R	R	R	R	R	R	R	S	S	S	R	S	IMP
47	M	249504	SUR	APPENDIX	C	6,7	B	ENTEROBACTER	R	R	S	S	R	R	S	S	R	S	S	S	S	S	ABSCOND
39	M	232690	SUR	LAPRO	D	6,7	BC	ENTEROBACTER	R	R	S	S	R	R	S	R	R	R	S	R	R	R	AMA
58	M	297739	ORTHO	KNEE FRAC	D	1,3,7	A	E.COLI	R	R	R	S	R	S	R	R	R	S	S	S	R	R	IMP
51	M	249756	ORTHO	0RIF	C	1,3,7	AC	KLEB	R	R	R	S	R	R	R	R	R	R	R	S	R	R	IMP
59	F	248417	ORTHO	HIP REPLACE	D	5,7	BC	E.COLI	R	R	R	R	R	R	R	R	R	S	S	S	R	R	ABSCOND

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