A STUDY ON POST CAESAREAN WOUND INFECTION

Dissertation submitted to

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OBSTETRICS AND GYNAECOLOGY

Register No.: 221716208

THANJAVUR MEDICAL COLLEGE

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

CHENNAI, TAMILNADU

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CERTIFICATE

This is certify that the dissertation titled **"A STUDY ON POST CAESAREAN WOUND INFECTION"** is a bonafide work done by **Dr.N.SUKANYA** in the Department of Obstetrics and Gynaecology, Thanjavur Medical College, in partial fulfillment of the university rules and regulations for the award of MS degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2017-2020.

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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3997538/
https://www.researchgate.net/publication/313849868_Postcesarean_wound_infection_Prevalence_impact_prevention_and_management_challenges
https://women.texaschildrens.org/sites/pavilion/files/documents/CSectionSSI_072817.pdf
https://www.ijrcog.org/index.php/ijrcog/article/download/32/32
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https://link.springer.com/article/10.1186/s12884-018-2075-8
https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0129434
https://www.ijrcog.org/index.php/ijrcog/article/viewFile/2618/2276
https://www.researchgate.net/publication/262021926_A_Study_of_Post-Caesarean_Section_Wound_Infections_in_a_Regional_Referral_Hospital_Oman
https://www.researchgate.net/publication/266561197_Prevention_and_Management_of_Cesarean_Wound_Infection
https://healthdocbox.com/amp/70950542-Chronic_Pain/Surgical-site-infection-in-obstetrics-practice.html

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DECLARATION

I solemnly declare that this dissertation titled **"A STUDY ON POST CAESAREAN WOUND INFECTION"** was done by me at Dept. of Obstetrics and Gynaecology, Thanjavur Medical College during year 2017-2020 under guidance and supervision of **Prof.Dr.R.RAJARAJESWARI,MD.,DGO.,DNB OG.,** This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards partial fulfillment of requirements for the award of MS degree in Obstetrics and Gynaecology (BRANCH II).

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CONTENTS

1. INTRODUCTION

Surgical site infection is one of the most common complications following caesarean section.Incidence of wound infection ranges from 0.5% to 15%.[1]

Surgical Site Infection is associated with a maternal mortality rate of upto 3% with global increase in caesarean section rate. It is expected that the rate of occurrence of Surgical Site Infection will increase in parallel.

Optimization of maternal co-morbidities, appropriate antibiotic prophylaxis and evidence based surgical techniques are practiced to reduce the incidence of Surgical Site Infection.

SSI accounts for significant extension of hospital stay.Since, Surgical Site Infection continues to be common post operative complication in both the developed and developing world, there is need to implement Surgical Site Infection Surveillance.

If prophylactic Antimicrobials are given, the incidence of wound infection ranges from 2-10% depending upon risk factor.

Many Studies have been conducted regarding the Surgical Site Infection's under the guidelines provided by CDC. Under the guidelines of CDC, a clinical study of wound infection following caesarean section occurring in Raja Mirasudhar hospital, Thanjavur has been conducted to find the incidence of wound infection and to analyze the various risk factors associated with wound infection, common bacterial pathogens and antibiotic sensitivity.

2. AIM AND OBJECTIVES

- To determine the incidence of post caesarean surgical site infection,
- To identify the risk factors, common bacterial pathogens causing infection and
- To analyze antibiotic sensitivity

Study Centre **:** Department of Obstetrics and Gynaecology,
Government Raja Mirasudhar Hospital (RMH),
Thanjavur Medical college,
Thanjavur.

Duration of study **:** January 2018 to December 2018

Time Period **:** 12 months

Study Design **:** **Prospective Cohort Study**

3. REVIEW OF LITERATURE

"Caesarean section is an operative procedure where by fetuses after the end of 28th week are delivered through an incision on the abdominal and uterine walls. Caesarean delivery is the most commonly performed operation in Obstetrics".

The word sepsis was derived from Greek word "Sepo" which means "I rot". Hippocrates viewed sepsis as a dangerous biological decay that could potentially occur in the body. Effective control of wound sepsis developed around 2 key moments

-the adoption of Antiseptic practices from 1860's and the advent of antiseptic practices from the late 1930's. Joseph Lister & Louis pasteur developed the concept of Antiseptic surgery and germ theory respectively.

3.1 SURGICAL SITE INFECTION

Definition:*As per National Healthcare Safety Network division of CDC, "SSI are defined as infections which develops at the surgical site within 30 days of surgery"*

Types:

1. Superficial: involving skin and subcutaneous level
2. Deep: involving Muscle and fascia
3. Organ Space: involving Organ

Criteria for diagnosing SSI:

1. **Clinical:**
    - Purulent discharge from surgical site
    - Presence of signs of infection (Swelling, pain, tenderness, redness
    - 5 Signs of inflammation (Rubor, Calor, Tumor, Dolor &Functiolaesa)[8]
2. **Culture:**
    - Positive bacterial / organism isolated
3. **Others**:
    - Clinical diagnosis for superficial type
    - Abcess/Histopathological/USG evidence of infection

SSI rate[24] = (No. of SSI/No. of surgeries) X 100

SSI Monitoring must be done as a part of HAI Surveillance.

Advantages of Monitoring include:

1. Baseline occurrence of SSI in our institution obtained
2. Helps in analyzing HAI
3. Provide data for Root cause analysis
4. Feedback to adopt best practices possible only if cases are noted.

Superficial Wound disruption:

Defined as “post operative wound disruption of the layers of abdominal incision superficial to the fascia”.

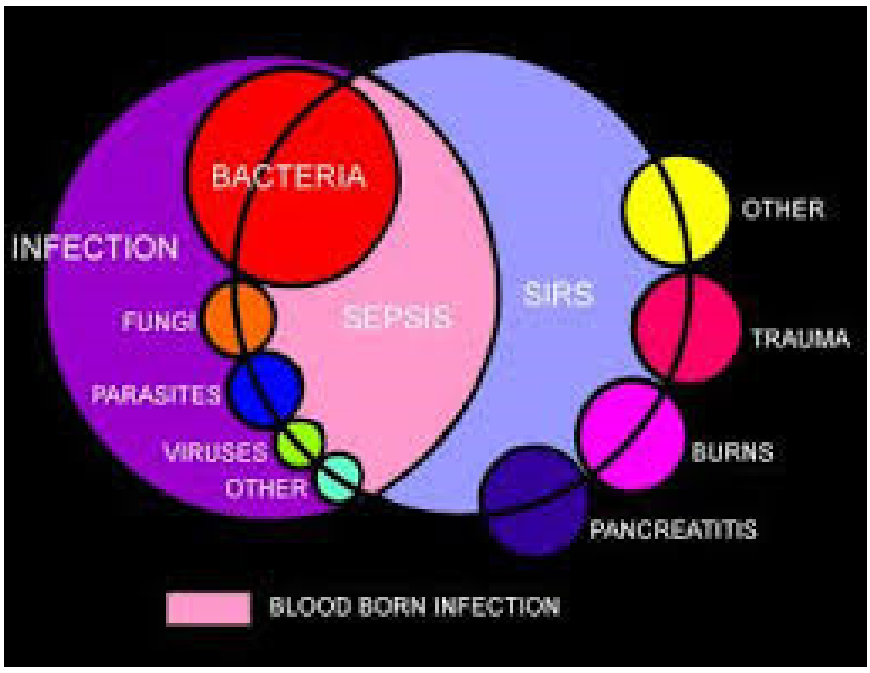
3.2 DEHISCENCE[8]

The definition of fascial dehiscence is postoperative separation of the abdominal musculoaponeurotic layers. It occurs during 3rd to7th postoperative day. The early presentation of the problem emphasizes the importance of proper wound closure simple improvements in delayed absorbable suture materials (although their importance in hernia prevention has been shown). As compared to superficial wound disruption, the incidence of fascial dehiscence is less common but the mortality rate was found to be 24% in a recent study of 198 cases by Madsen et al. The incidence of wound breakdown in 12 studies before 1940 was 0.4%.

3.3 SIRS[52]

Systemic inflammatory response syndrome (SIRS) is A serious condition in which there is inflammation throughout the whole body. It may be caused by a severe bacterial infection (sepsis), trauma, or pancreatitis.

Fig 1: SIRS vs Sepsis



Manifestations of SIRS include, but are not limited to:

- Body temperature less than 36 °C (96.8 °F) or greater than 38 °C (100.4 °F)
- Heart rate greater than 90 beats per minute
- Tachypnea (high respiratory rate), with greater than 20 breaths per minute; or, an arterial partial pressure of carbon dioxide less than 4.3 kPa (32 mmHg)
- White blood cell count less than 4000 cells/mm³ (4 x 109 cells/L) or greater than 12,000 cells/mm³ (12 x 109 cells/L); or the presence of greater than 10% immature neutrophils (band forms). Band forms greater than 3% is called bandemia or a "left-shift.

When two or more of these criteria are met with or without evidence of infection, patients may be diagnosed with "SIRS."

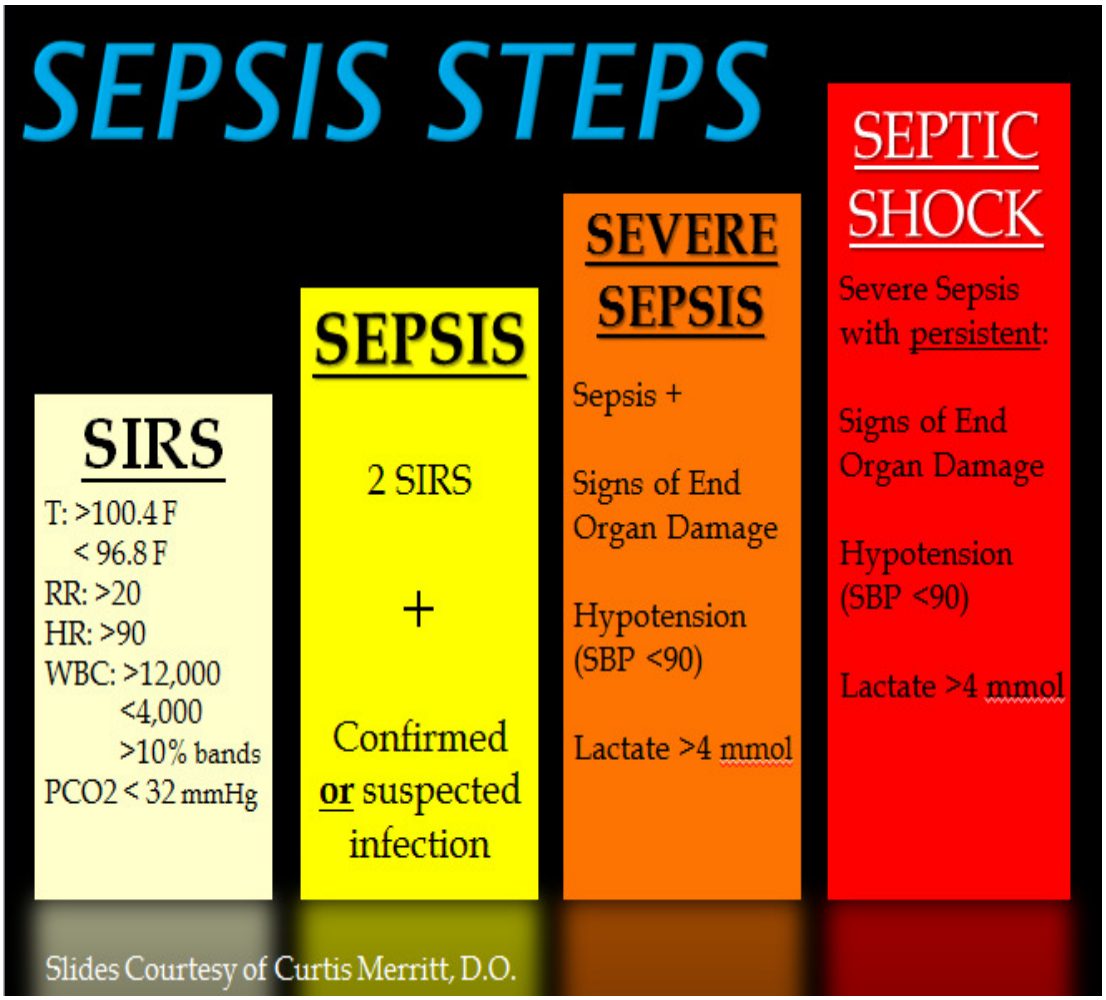


Fig 2: Sepsis Continuum

3.4 CLASSIFICATION OF OPERATIVE WOUNDS [9]

I. CLEAN

- Elective
- primarily closed and undrained
- Nontraumatic, uninfected
- No inflammation encountered
- No break in aseptic technique
- Respiratory, alimentary, genitourinary tracts not entered

II. CLEAN CONTAMINATED

- Alimentary, respiratory, or genitourinary tract entered under controlled conditions and without unusual contamination
- Appendectomy
- Vagina entered
- Genitourinary tract entered in absence of culture-positive urine
- Minor break in technique and Mechanical drainage
- **Caesarean section is a clean contaminated type of surgery where procedure related chance of infection is less.**

III. CONTAMINATED

- Open, fresh traumatic wounds
- Gross spillage from gastrointestinal tract
- Entrance of genitourinary tract in presence of infected urine,
- Majorbreakintechnique
- Incisionsinwhichacutenonpurulentinflammationis present

IV. DIRTY OR INFECTED

- Traumatic wound with retained devitalized tissue, foreign bodies
- Fecal contamination
- Delayed treatment
- Wounds from a dirty source
- Perforated viscus encountered
- Acute bacterial inflammation with pus encountered during operation

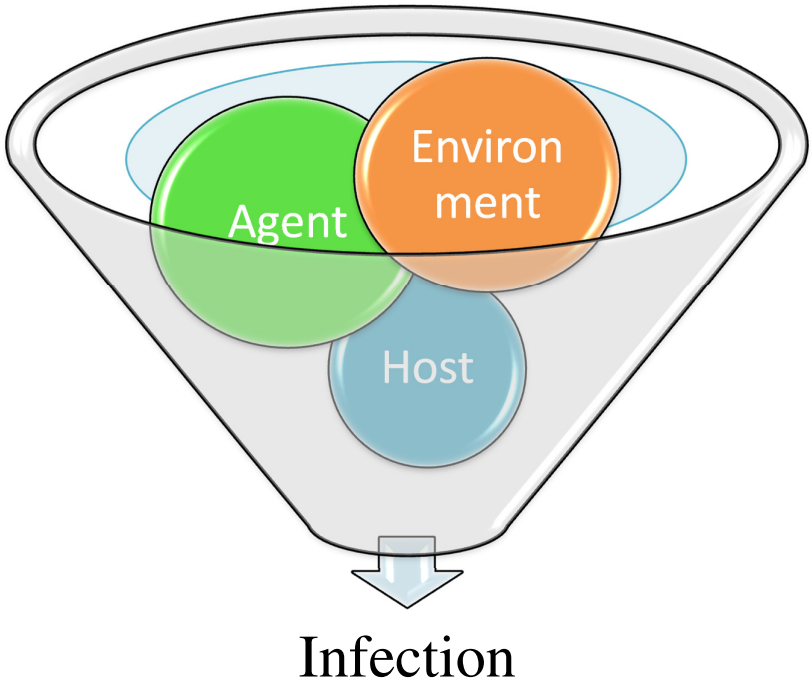
Table 1: Risk of SSI in relation to Contamination[9]

TYPE OF WOUNDS	RISK OF SSI %
Clean	2.1
Clean contaminated	3.3
Contaminated	4.6
Dirty or infected	7.1

3.5 DETERMINANTS OF WOUND INFECTION [12]

Agent, Host and environmental factors interrelate in a variety of complex ways to produce disease.

Fig 3: Determinants of Wound Infection



Agent:

Referred to an infectious microorganism or pathogen. The agent must be present for disease to occur. However, the presence of that agent alone is not always sufficient to cause disease.

Bacterial count:[20]

The important factor that affects wound healing is the innoculum of bacteria. The mode of entry of bacteria may be by droplet, or by direct contact from the surgeons or by instruments or self contamination from the endogenous bacterial flora. Despite proper preparation of the skin, bacteria are always present. The risk increases if the operative site is inoculated with greater than 10^5 organisms per gram of tissue. The risk increases if the operation involves a body structure that is heavily innoculated by organisms, such as bowel. Surgeries of the female genital tract will encounter10^6– 10^7bacterial/ml.

Virulence of the Bacteria:[46]

Virulence of the bacteria depends on the ability to produce certain toxins and other substances that invade the host,produce tissue damage orsurvive within the host tissue. Coagulase-positive staphylococci are more virulent (require smaller innoculum) than the coagulase-negative species.Some strains of clostridium perfringens or group A streptococci are more virulent but may require small inoculums to cause severe necrotizinginfection at the surgical site. The virulence of Escherichia coli is due to endotoxin in its outer cell membrane.

Host:

A variety of factors intrinsic to the host called as risk factors.

It includes age, personal hygiene, nutrition, immunological status and presence of co-morbidities including hyperglycemia, anemia and patient oncortico steroid, Uremia.

Environment:

Extrinsic factors that affect the agent and the opportunity for exposure.

It includes the geology, climate, socioeconomic factors such as crowding, sanitation and availability of health services.

Since, the Agent-Host-Environment model did not work well, Multifactorial causation theory also been proposed.

Pregnancy Related factors:

GDM,GHT, Twin Pregnancy, PROM, greater no. of PV examinations, prolonged trial of labor, use of internal foetal monitoring and chorioaminonitis, Duration of surgery >1 hour increases the rate of wound infection.

Aggregate effect:

When above all 4 determinants are evaluated in the aggregate, wound infection is a complex biological process and that identification of the causes of an infection in a specific situation can be problematic.

Wound healing physiology:[8]

Wound healing process is defined by the Wound Healing Society as "a complex and dynamic process that results in restoration of anatomic continuity and function".

Healing by First Intention: When the injury involves only the epithelial layer, the principal mechanism of repair is epithelial regeneration, also called primary union or healing by first intention.

24 hours	fibrin, neutrophils, increased basal cell mitoses
24-48 hrs	epithelial cell migration, depositing BM
Day 3	macrophages, granulation tissue
Day 5	neovascularization maximal
Week 2	fibroblast proliferation, collagen deposition, blanching
Month 1	scar, regression

Fig 4: Time period - Healing by First Intention

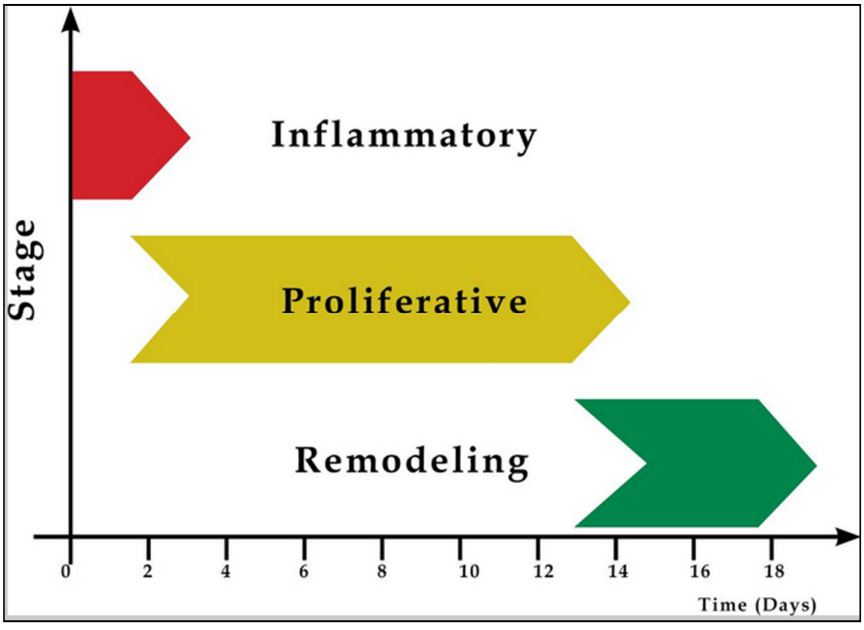
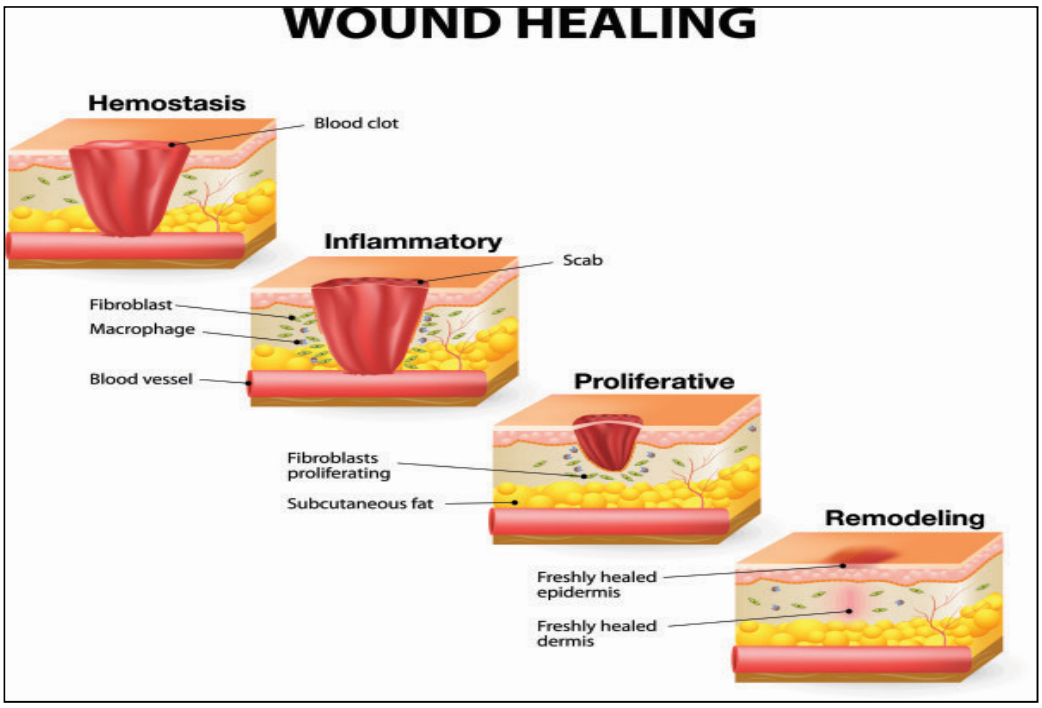


Fig 5: Healing by First Intention



Healing by Second Intention: When cell or tissue loss is more extensive, such as in large wounds, abscesses, ulceration, and ischemic necrosis (infarction) in parenchymal organs, the repair process involves a combination of regeneration and scarring. In healing of skin wounds by second intention, also known as healing by secondary union. Differs from first intention in that:

- Inflammatory reaction more intense
- More granulation tissue
- wound contraction (5-10% of original size) from myofibroblasts

Wound strength: 10% at one week, by third month plateau at 70-80% original tensile strength

Wound Strength: Carefully sutured wounds have approximately 70% of the strength of normal skin, largely because of the placement of sutures. When sutures are removed, usually at 1 week, wound strength is approximately 10% of that of unwounded skin, but this increases rapidly over the next 4 weeks. The recovery of tensile strength results from the excess of collagen synthesis over collagen degradation during the first 2 months of healing,

Repair of tissues involves regeneration (replacement of damaged cells by cells of the same type) or fibrosis (replacement by connective tissue).

Cell Cycle and Proliferative Potential

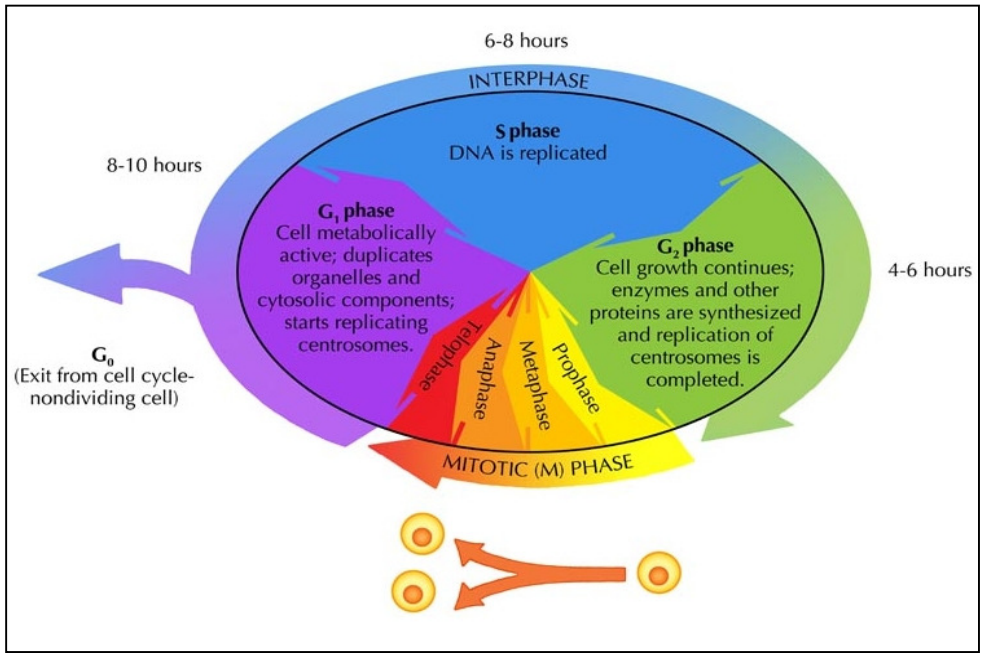
G1 - presynthetic

S - DNA synthesis

G2 - premitotic

M - mitotic

Fig 6: Cell cycle



Wound Healing

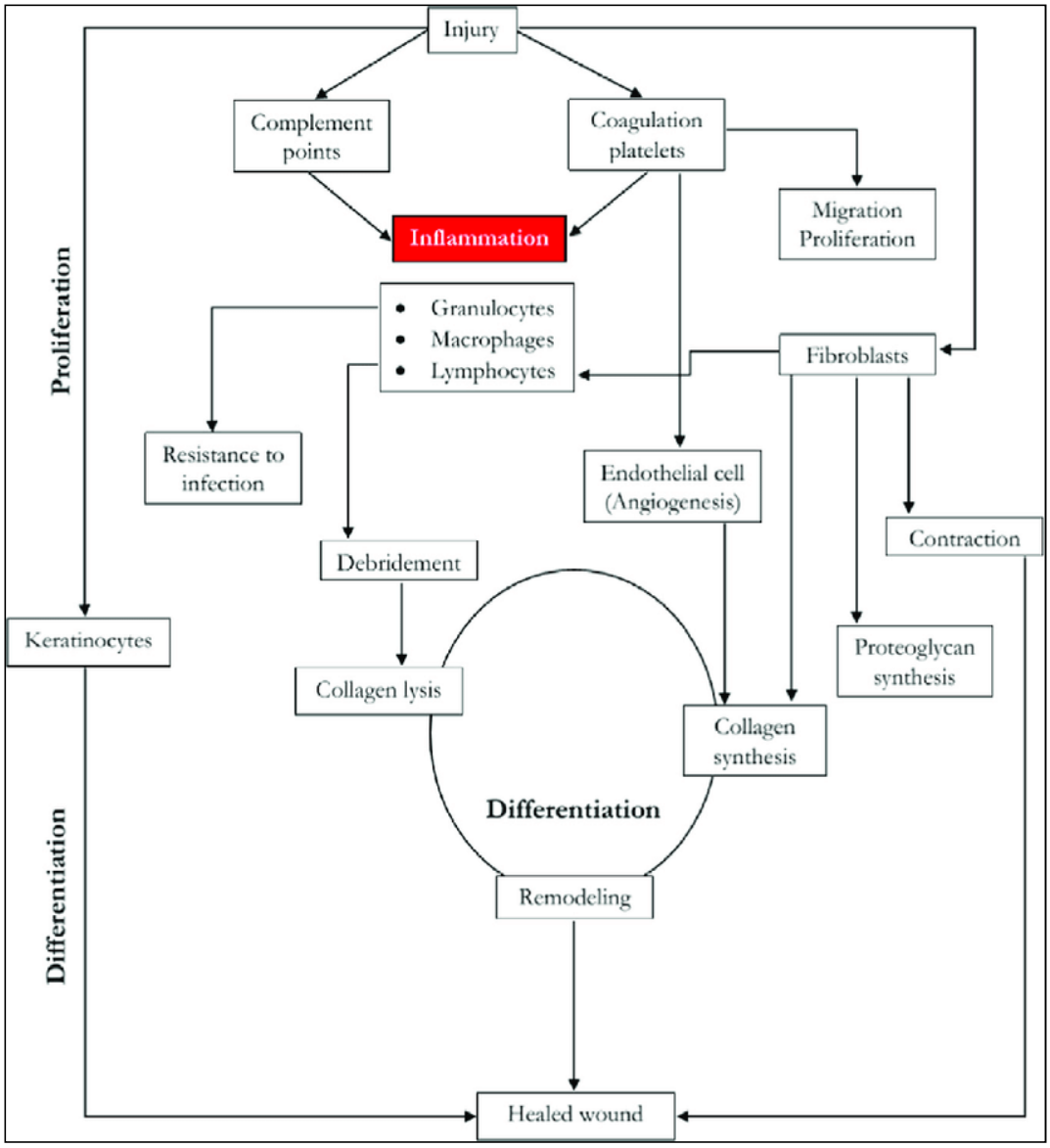


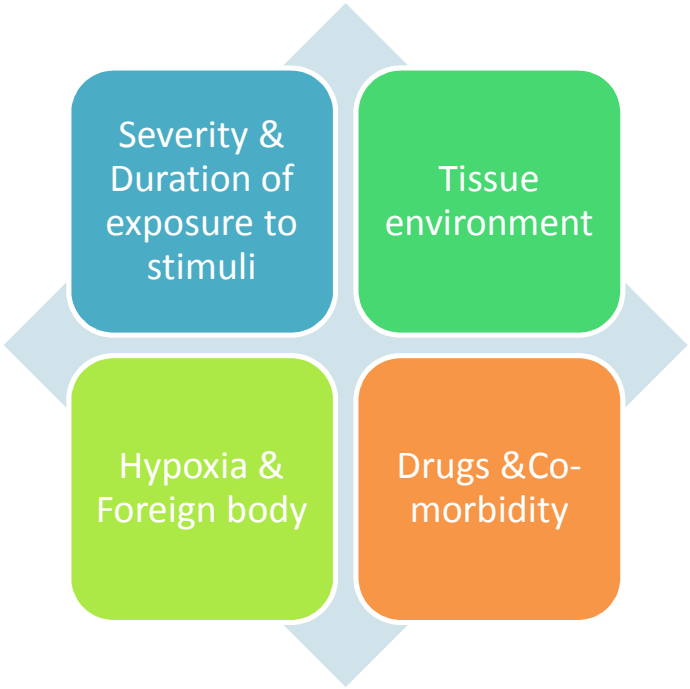
Table 2: Growth factors involved in Wound healing[8]

GROWTH FACTOR	FUNCTIONS
Epidermal growth factor (EGF)	Mitogenic for keratinocytes and fibroblasts; stimulates keratinocyte migration; stimulates formation of granulation tissue
Transforming growth factor-α (TGF-α)	Stimulates proliferation of hepatocytes and many other epithelial cells
Vascular endothelial growth factor (VEGF)	Stimulates proliferation of endothelial cells; increases vascular permeability
Platelet-derived growth factor (PDGF)	Chemotactic for neutrophils, macrophages, fibroblasts and smooth muscle cells; stimulates ECM protein synthesis
Fibroblast growth factors (FGFs), including acidic (FGF-1) and basic (FGF-2)	Chemotactic and mitogenic for fibroblasts; stimulates angiogenesis and ECM protein synthesis
Transforming growth factor-β (TGF-β)	Chemotactic for leukocytes and fibroblasts; stimulates ECM protein synthesis

Impaired Wound healing:

Many factors will lead to impaired wound healing such as anemia, Malnutrition, chronic inflammatory disorders, hyperglycemia, peripheral vascular disease and cardio pulmonary disease.

Fig 7: Factors affecting wound healing



3.6 MICROBIOLOGY OF SSI[20,24]

Staphylococcus aureus is the most common organism causing wound infection. Bacteria of surgical interest, which are encountered in SSI'sfrequently, are discussed here. Infections that occur in the first 24 hours after surgery usually are caused by Gram-positive cocci or occasionally by facultative Gram-negative rods. Infections that occur after the first 48 hours more frequently have an anaerobic component

Vaginal flora:[9]

The most frequent source of bacteria that cause postoperative pelvic infection among women is the vagina. Mean bacterial counts in vaginal secretions are 10^8 to10^9 bacteria/mL, with three to six different species presentThe most frequent aerobic bacteria are Lactobacillisp, Gardnerellavaginalis, Staphylococcus epidermidis, Corynebacteriumsp, Enterococcusfaecalis species of Streptococcus and Enterobacteriacea. Anaerobes outnumber aerobes and include Peptostreptococcussp, Peptococcussp, Prevotellabivia, Prevotelladisiens, and members of the Bacteroidesfragilis.

Agents Causing SSI:

Bacterial (for clean wounds)

1. S.Aureus
2. CONS
3. Enteroceus

Fungi:

1. Candida albicans

If bowel integrity lost

1. E-Coli

Staphylococcus Aureus:

Staphaureus is a gram positive cooci which is a pluripotent pathogen causing various problems. One of the most common cause of skin and soft tissue Nosocomial infection

If untreated, it will lead to septic shock. Normal human commensal, colonization

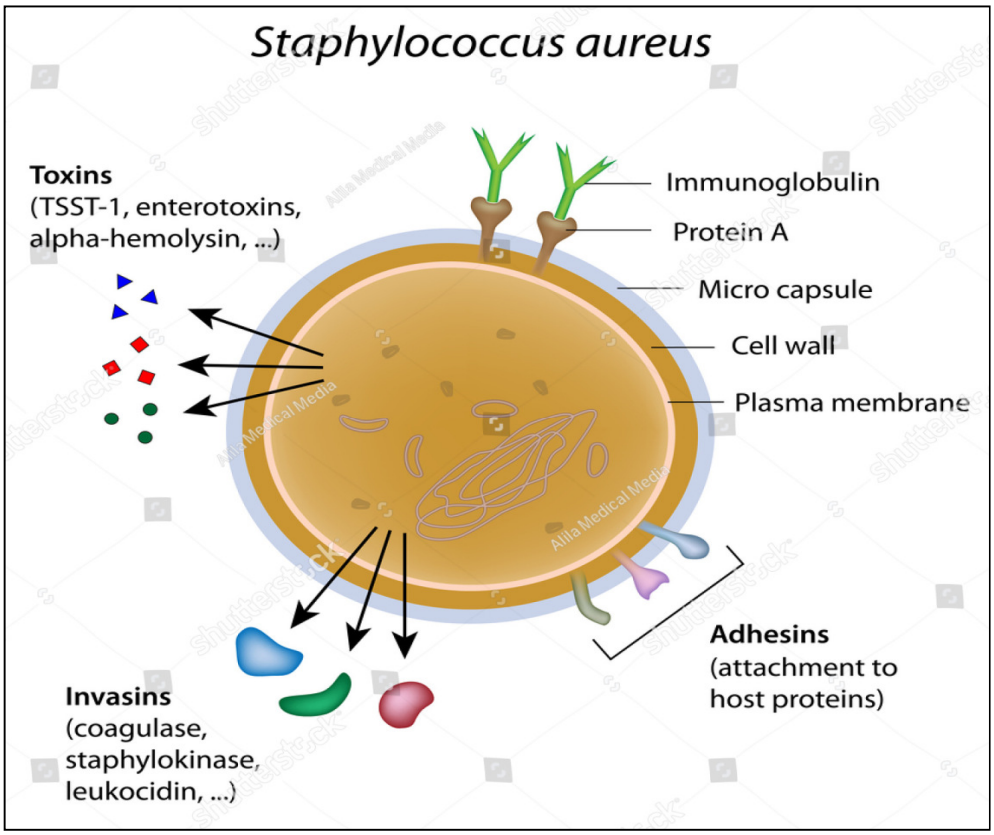
occurs in nose, vagina, axilla, perineum and oropharynx

Colonization is higher in insulin dependent diabetics, HIV, pt on hemodialysis and skin damage. 25% of health professional were carriers of S.aureus and responsible for transmission of infection.

S.aureus adhere to tissue[33]

- By clumping factor
- Invasion by proteases, hyalurinidase& lipase
- Escapes from our immunity
- Antiphagocytic (Protein-A)
- Inhibit chemotanis
- Survive intracellurally

Fig 8: S-Aureus



Hospital acquired MRSA:[24,37]

Express Mec-A gene which is Multidrug resistant, cause perioperative wound infection. S.aureus infection must be treated according to antibiotic sensitivity testing

CONS-Coagulase Negative Staph.aureus:

- CONS is less virulent than S.aureous
- S.epidermidis is the most commonly isolated CONS.
- CONS present in all human as Normal skin flora. It forms Bioflim which protects bacteria from our immunity

E-Coli:

- Aerobic, gram negative bacilli, found in gut of humans.
- It causes UTI, Diarrhea, skin injections and peritorritis.
- E-coli have 4 surface antigen, Fimbrail Antigen is responsible for adherence and colonization to skin & E-coli is the most common agent causing UTI.

Klebsiella:

- Commensal of human intestine
- k.pneumoniae – most pathogenic of all species causes wound infection
- Nosocomial spread

Pseudomonas:

- Gram negative, pigment producing bacilli
- Pseudomonas found in prolonged hospitalized patients and cause skin and soft tissue infection
- Produce inflammatory and suppurative lesions in humans; the purulent
- Discharge produced usually being greenish-blue in color with a characteristic sweetish odour
- Resistances of these organisms to the common groups of antimicrobial agents used now are frequently met with in hospital settings
- As such, owing to its resistance, it has become one of the front-runners amongst drug resistant bacteria to cause fulminant septicemia owing to secondarily infected burn wounds, SSIs, urinary tract infections and respiratory infections in mechanically ventilated patients.

Proteus:

- Pleomorphic bacilli belongs to enterobacteriacea family
- Opportunistic pathogen causing Urinary, wound and soft tissue infections
- Nosocomial outbreaks noted
- Exhibit swarming &Dienes phenomenon

Fungi of surgical interest:

- For ages, fungi have been neglected and treated with impunity by physicians and surgeons alike. However, these groups of microbes now

seem to draw everyone's attention and a detailed knowledge of them is now a must, as fungal infections are now not only notoriously common in surgical scenario but also increasingly fulminant.

- Renewed fungal pathogenicity can be attributed to the increase in number of immunocompromised patients as a whole and increase in numbers of such people being subjected to surgery.
- Fungal isolates are now increasingly common in abscesses of wound infections with sterile culture yields.
- Histoplasma capsulatum, Cryptococcus neoformans, and Coccidioides immitis, Aspergillus, Rhizopus and Mucor cause opportunistic infections. Dreaded amongst the fungi, however is the candida species. Candidiasis is an opportunistic endogenous infection, the commonest being in diabetes mellitus.

ESKAPE pathogens[24]

Nowadays, "ESKAPE" pathogens are responsible for many of Nosocomial infections which includes

- Enterococcius faecium
- Staphylococcus aureus
- Klebsiella pneumonia
- Acinetobacter baumanii
- Psuedomonas aeruginosa
- Enterobacter spp

Immune mechanisms:[8]

Immune mechanisms are essentially represented by the humoral and cellular immunity. Humoral immunity is so termed as its components circulate within blood and body fluids as proteins, and consists of two components namely the antibodies (immunoglobulin) and the complement. These antibodies are produced by B lymphocytes in response to the presence of substances including microbes that the mammalian host recognizes as a foreign antigen and part of itself. They are activated in a sequence generally triggered by binding certain types of antibodies to microbial antigens (especially the IgM variety). This activated complement aids cellular immunity.

Cellular immunity comprised of the T lymphocytes of different clonal varieties receives the initial load of antigens (microbial or otherwise) and phagocytose them for further killing. Further killing of the microbes is usually via lysis as governed by multiple mechanisms including the bacterial degradation by vacuolation and by release of oxygen free radicals. **Cellular immunity generally is triggered within 2 to 4 hours of microbial inoculation (in this instance, after the incision is made) into the tissues and it is this lag period that a surgeon should guard and cover by means of antimicrobial prophylaxis (AMP) whenever justified.**

The cellular immunity is influenced by multiple factors, innate or acquired. Cytokines, like the interferon's, tumor necrosis factors, and interleukins offer innate co-ordination of cellular immunity. They further aid to regulate the host defenses via the suppression and augmentation of specific defense components,

including their own activity (by feedback

Regulatory mechanisms). This may act as a double-edge sword if mediators of these antigen-toxic immune mechanisms "Spill on'' to damage the normal cells, as hazardously demonstrate in the sepsis syndrome where it is these mediators which cause much damage to the tissues than the infectious agent or its products per se. The acquired factors governing cellular immunity include the previous exposure of antigenic material (as in immunization), immune compromised status like as in diabetes mellitus, malnutrition, AIDS and long term steroid therapy. In summary, immune mechanism is the ''cog in the wheel'' in prevention of infections, at surgical site.

Table 3: Risk factors associated with wound infection[46]

Patients characteristics	**Operation characteristics**
Obesity	Scrub
Diabetes Mellitus	Skin antisepsis
Chorioamnionitis[18]	Pre operative shaving
Postoperative endometritis	Preoperativepreparation
Prolonged rupture of membranes[3]	Surgical drape
Severe Anemia	Duration of operation
Stress–physiological or psychological	Excessive blood loss during surgery
Smoking	Antimicrobial prophylaxis
ASA scoring >3	Foreign body in surgical site
Anticoagulant therapy	Poor surgical technique

Diabetes:

High blood glucose inhibits the immune response by affecting neutrophil chemotaxis. Diabetes increases the wound infection by six times[40].

Obesity:

Major risk factor for post caesarean wound complications.[65]

1. Serous fluid collection
2. Haematoma formation
3. Proper vascularity of subcutaneous fat

   The above are the 3 factors interfere with healing.

Wloch et al observed that being overweight with BMI >35 was a major risk factor for infection

Anemia:

Low oxygen levels caused by Anemia halt the wound healing stages. Iron is a vital cofactor for proteins involved in energy metabolism, respiration, DNA synthesis, Cell cycle arrest and apoptosis.Various mechanism of which iron deficiency impairs wound healing. Current evidence shows that HIF1 (Hypoxia Inducible Factor-1) plays a role in cell migration, cell survival under hypoxic conditions, cell division, growth factor release and matrix synthesis.[52]

Recent interest in Lactoferrin, an iron binding glycoprotein secreted from glandular epithelial cells has focused on its role in promoting cutaneous wound healing by enhancing the initial inflammatory phase, cell migration and proliferation.

Nutrition:

Nutrition and nutritional supplementation to improve wound healing has been written about extensively, especially in the area of chronic wounds. Many recommendations have been made particularly with regard to vit C, vit A and zinc. Adequate nutrition does seem essential to proper wound healing. Surgical procedures increase protein requirements. Vitamin is necessary for collagen synthesis, capillary wall integrity, fibroblast function & immunologic functions. Vitamin C deficiency can delay wound healing, although there is no strong evidence for supplementation in patients who do not have scurvy. Zinc supplementation for accelerating wound healing has been studied. Low serum Zinc levels have been associated with impaired healing. Zinc aids collagen formation and supports immune function. Vit. A increases the number of monocytes and macrophages andstabilizes the intracellular lysosomes of WBC. Vit. A has also beenshown to accelerate collagen production in animals.

3.7 OPERATIVE CHARACTERISTICS:

Hair clipping:[10]

As per CDC SSI GUIDELINES 1999, routine removal of hair preoperatively is not recommended and if needed remove immediately before surgery by clipping(category IA recommendation). A Cochrane review published in 2012 suggested that hair removal at the time of surgery was not associated with lower postoperative SSI rates and that it should be done only to facilitate surgery or for applying adhesive dressings Shaving the surgical site has been shown to be

associated with significantly higher rates of SSI compared to clipping, as a result of microscopic breaks in the skin caused by the razor.

Skin preparation:

The skin is a main source of pathogens causing SSI. Preoperative skin preparation with antiseptic agents has been proven to reduce the risk of SSI Two large randomized controlled trials (RCTs) assessed this issue. Ngai ET al31 compared chlorhexidine with alcohol, povidone–iodine with alcohol, and the sequential combination of both solutions for preventing SSI postcaesarean section. Their study included 1,404 women undergoing non emergent caesarean section. The three skin preparation groups had similar SSI rates (3.9%–4.6%), leading to the conclusion that no particular method of skin preparation before caesarean section is recommended. However, TuuliET al32 evaluated the use of chlorhexidine with alcohol compared to povidone–iodine with alcohol for skin antisepsis in 1,147 women undergoing caesarean section. The use of chlorhexidine–alcohol resulted in a significantly lower risk of overall SSI (4.0%) after caesarean section compared to iodine–alcohol (7.3%)

Vaginal preparation:

Dahlke et al reported no difference in the incidence of wound infection when adding vaginal preparation to the standard abdominal preparation in caesarean section. In a Cochrane review, vaginal preparation with povidone–iodine solution before caesarean section reduced the risk of postcaesarean endometritis from

7.2% to 3.6% (RR: 0.39, 95% CI: 0.16–0.97), particularly in women with ruptured membranes

Antibiotic prophylaxis:[13,28,51]

A significant component that affects the rate of SSI is the use of antibiotic prophylaxis in caesarean section. Three Cochrane reviews evaluated the role of antibiotic prophylaxis in caesarean section. When comparing antibiotic prophylaxis to no prophylaxis or placebo for preventing infection following caesarean section, the use of prophylactic antibiotics significantly reduced the incidence of wound infection (RR: 0.40, 95% CI: 0.35–0.46), endometritis (RR: 0.38, 95% CI: 0.34–0.42), and maternal serious infectious complications (RR: 0.31, 95% CI: 0.20–0.49) The American College of Obstetricians and Gynecologists, in its committee opinion, recommends antimicrobial prophylaxis for all caesarean deliveries unless the patient is already receiving an antibiotic regimen with appropriate coverage (eg, for chorioamnionitis). The antibiotics should be administered within 60 minutes before the procedure**. A single dose of a targeted antibiotic, such as a first-generation cephalosporin, is the first-line antibiotic of choice, unless significant drug allergies are present.** In obese women (body mass index .30 kg/m2), a higher dose of preoperative antibiotics prophylaxis should be considered. Repeated doses intraoperatively are reserved for particular situations, as in the case of major intraoperative bleeding, surgery lasting for more than 1 hour.[35,41]

For antibiotic prophylaxis to work effectively, several important criteria must be fulfilled: [34,49]

- The operative procedure must have a significant risk of bacterial contamination
- The prophylactic antibiotic administered should be effective against expected pathogens and have a low rate of side effects
- The antibiotic should not be one that would be routinely used therapeutically
- The tissue levels of the antibiotic need to be optimal at the time surgery occurs.

IntraOp Characteristics:

Surgical personnel:

Staff education programs and refresher courses in aseptic and scrub techniques have been shown to reduce the incidence of SSI in elective and non elective caesarean deliveries intraoperative practices.

Hand Hygiene Measures:[24,47]

Fig 9: Follow6 steps of hand washing for 20 – 40s as per WHO guidelines

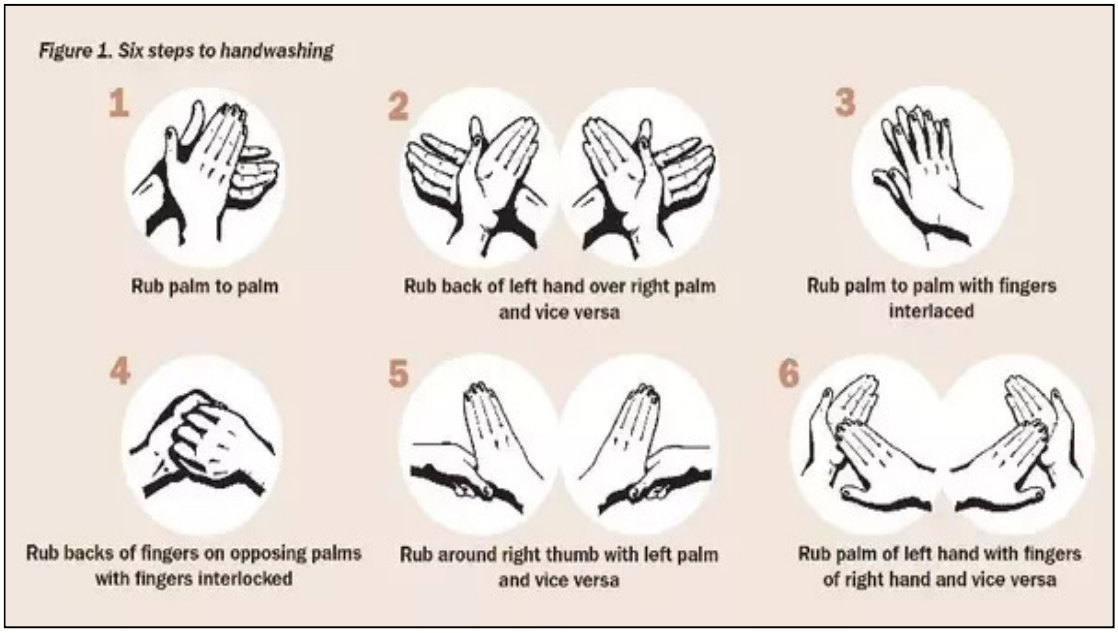
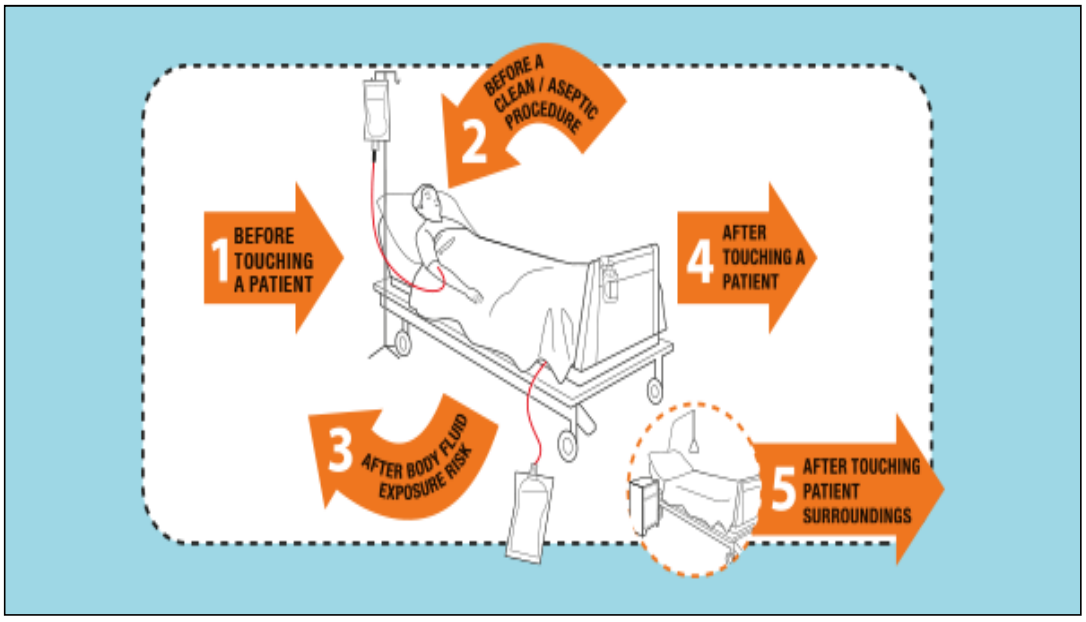


Fig 10: 5 Moments of hand hygiene must be strictly followed



Use Alcohol based hand rub for 20-30 s (70-80% ethyl alcohol or chlorheridine 2-4%)

Donning & Doffing:

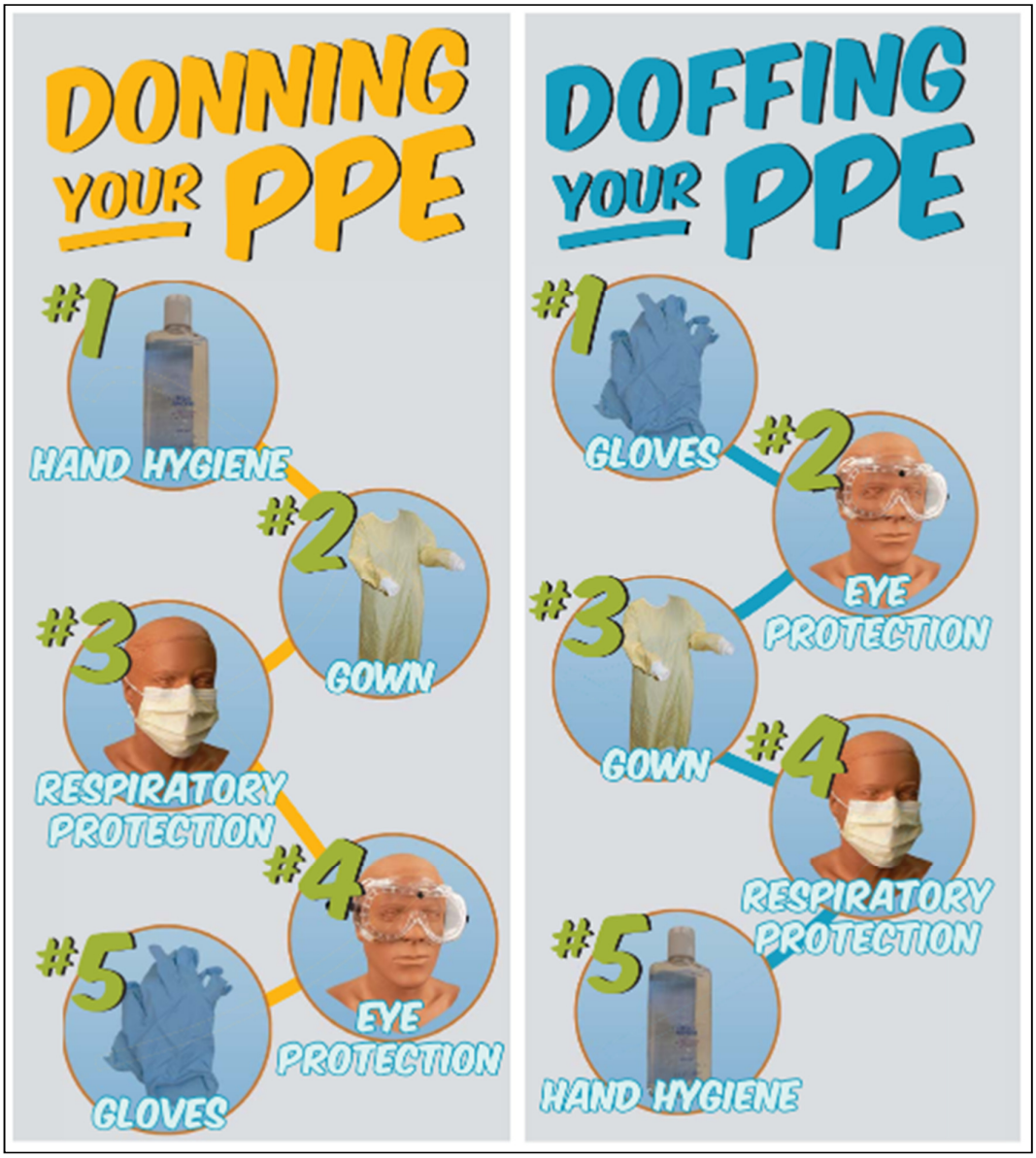
Donning & Doffing of PPE in a proper manner to prevent transmission of infection.

Donning – Gown-Mask-Goggles-Gloves

Doffing – Gloves-Goggles-Gown-Mask

HAI audit to ensure cleanliness in OT

Fig 11: Donning & Doffing



3.8 SURGICAL SITE INFECTION GUIDELINES 2016[11]

- Routine shaving is not allowed,only clipping is advised
- Chlorhexidine reduces the bacterialcolonization of skin and chlorhexidine needs to dry on the skin for maximal effect and it is superior to povidone
- Early showering after surgery as early as 12 hours does not increase the risk of SSI
- Administraton of supplemental oxygen (fio2–80%) during surgery and perioperatively reduces the risk of SSI
- Intraoperative maintenance of normothermia is recommended
- Administration of prophylactic antibiotics within 1 hour of incision is recommended
- Perioperative glycemic control is mandatory to avoid SSI
- Recommendations for preoperative preparation of the patient to prevent surgical site infections(telindes)[9]

1. Identify and treat all infections remote to the surgical site before elective operation, and postpone elective operations on patients with remote site infections until the infection has resolved.
2. Do not remove hair preoperatively unless hair at or around the incision site will interfere with the operation.
3. If hair is removed, remove immediately before the operation, preferably with electric clippers.
4. Adequately control serum blood glucose levels in all diabetic patients, and particularly avoid hyperglycemia perioperatively.

5. Encourage tobacco cessation. At a minimum, instruct patients to abstain for at least 30 days before elective operation from smoking cigarettes, cigars, pipes, or other form of tobacco consumption.
6. Do not withhold necessary blood products from surgical patients as a means to prevent surgical site infections.
7. Require patients to shower or bathe with an antiseptic agent on at least the night before the operative day
8. Thoroughly wash and clean at and around the incision site to remove gross contamination before performing antiseptic skin preparation.
9. Use appropriate antiseptic agent for skin preparation.
10. Apply preoperative antiseptic skin preparation in concentric circles, moving toward the periphery. The prepared area must be large enough to extend the incision or create new incisions or drain sites, if necessary.
11. Keep preoperative hospital stay as short as possible while allowing for adequate preoperative preparation of the patient.
12. No recommendation to taper or discontinue systemic steroid use (when medically permissible) before elective operation. (unresolved issue)
13. No recommendation to enhance nutritional support for surgical patients solely as a means to prevent surgical site infection. (unresolved issue)
14. No recommendation to preoperatively apply mupirocin to nares to prevent surgical site infection. (unresolved issue)
15. No recommendation to provide measures that would enhance space oxygenation to prevent surgical site infections. (unresolved issue)

Surgical techniques:

Skin incision type[26]:

A Cochrane review published in 2013 included two studies comparing the Joel-Cohen incision with the Pfannenstiel incision. Overall, there was a 65% reduction in postoperative febrile morbidity (RR: 0.35; 95% CI: 0.14–0.87; P=0.023) with the Joel-Cohen incision. Only one study noted the incidence of wound infection separately and found no difference between the two techniques

Uterine exteriorization:

Extra-abdominal compared to intra-abdominal repair of the uterine incision was evaluated in the CORONIS study 2013 and in a large meta-analysis.55,61 Both found no significant differences in complication rates, including endometritis and wound infection, between the two techniques and concluded that both options are acceptable.

Cervical dilatation:

Two reviews evaluated the effect of mechanical cervical dilatation during caesarean section on infectious morbidity. Both found that mechanical cervical dilatation did not affect postcaesarean infection and infectious morbidity (including wound endometritis).

Closure of the uterine incision:

Single layer uterine closure versus double layer was examined in two large

RCTs and a Cochrane review. There was no difference in postoperative febrile morbidity, wound infection, and endometritis between the two techniques.

Peritoneal closure:

A Cochrane review and two recent large RCTs found no significant difference in the incidence of postoperative endometritis or wound infection in cases with peritoneal closure compared to nonclosure.

Subcutaneous tissue closure:

According to a Cochrane review, closure of the subcutaneous tissue reduced wound composite morbidity including hematoma, seroma, wound infection, and wound separation (RR: 0.68; 95% CI: 0.52–0.88; P=0.0039). There was no difference in the risk of wound infection alone or other shortterm outcomes. In regard to subcutaneous thickness, if depth is more than 2 cm, there is no difference in wound disruption between closure and nonclosure. In women with subcutaneous thickness more than 2 cm, closure was associated with a significant decrease in wound complications (RR: 0.66; 95% CI: 0.48–0.91) during caesarean section.

Skin closure:

The two most studied methods for skin closure after caesarean section are staples and subcutaneous sutures. A Cochrane review of eight trials concluded that wound complications and cosmetic outcome are similar between the two

techniques. In contrast, a large meta-analysis concluded that staples closure is associated with twofold increase in wound infection

Wound dressing:

There are several types of bandages available for dressing the surgical wound at the end of a surgery. A meta-analysis of 16 trials found no difference in SSI rate between surgical wounds covered with different types of dressings and those left uncovered. Two Cochrane reviews regarding early (0,48 hours) versus delayed dressing removal and postoperative bathing reported limited data, but no significant difference in SSI rate was shown

Perioperative oxygen supplementation:[61]

Several RCTs evaluated the use of high (80%) perioperative oxygen supplementation concentrations versus low (30%) on the incidence of SSI. None of the trials found a significant difference, concluding that increasing the concentration of oxygen in women undergoing cssesarean deliveries does not decrease the rate of SSI.

3.9 WOUND MANAGEMENT[1,55]

Haematomas&seromas are commonly observed after a caesarean delivery. These types of situations required manual opening of the wounds to allow drainage after infection has been treated and all of the haematoma/ seromas evacuated .An open wound can be managed in 3 ways :

1. Secondary closure

2. Secondary intention with dressings.

3. Secondary intention using negative pressure wound therapy

Secondary Closure:

It can be performed once a wound is free of infection or necrotic tissue and has started to granulate. A wound cleanser is first needed to prepare the area and then a polypropylene mattress suture is used to close the skin & subcutaneous tissue enbloc[1,55]. At 4 to 6 days, healthy granulation tissue is typically present, and secondary en bloc closure of the open layers can usually be accomplished (Wechter, 2005). With this closure, a polypropylene or nylon suture of appropriate gauge enters 3 cm from one wound edge. It crosses the wound to incorporate the full wound thickness and emerges 3 cm from the other wound edge[1]. These are placed in series to close the opening. In most cases, sutures may be removed on postprocedural day 10. Wound vacuum device use is gaining popularity. However, its efficacy remains unproven in randomized trials. In study by Dodson et al, patients who were managed with secondary closure required 17 days to heal. But when, patients were allowed to heal by secondary intention took

61 days to complete wound healing. Wound healed on average 7 weeks sooner in the secondary closure group.

Healing by Secondary Intention:

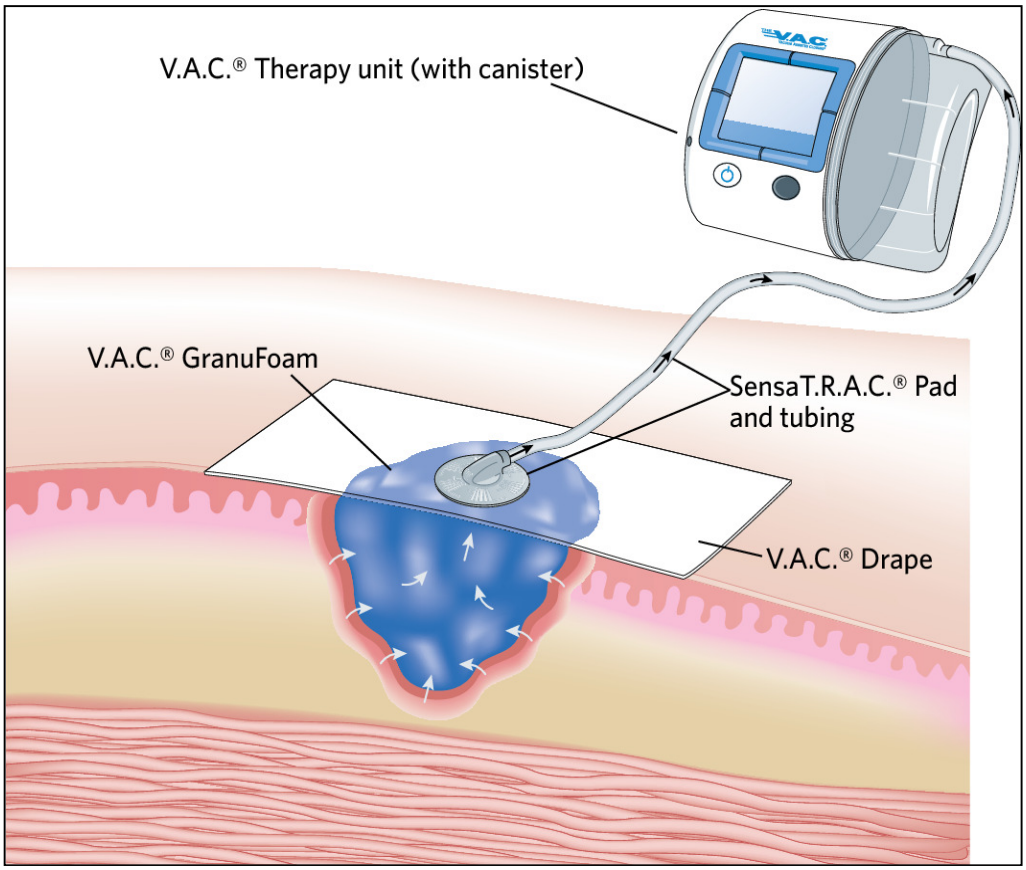
Healing through secondary intention has historically been the most common way to manage wound disruption by doing regular dressing and higher antibiotic.[8]

Secondary intention using negative pressure wound therapy:

Also known as Vacuum assisted wound closure (VAC), Topical Negative pressure (TNP), Negative Pressure Wound Therapy (NPWT)[16,17].

Moue's and colleagues are more circumspect about its use for disturbed abdominal wounds because of scarce data. Other reviewers conclude that vacuum therapy is the most efficient method of temporary abdominal closure for patients with open abdominal wounds.

Fig 12: Vacuum Assisted Wound Closure



Necrotizing Fasciitis:[1,9]

This uncommon, severe wound infection is associated with high mortality rates. In obstetrics, necrotizing fasciitis may involve abdominal incisions, or it may complicate episiotomy or other perineal lacerations. As the name implies, there is significant tissue necrosis. Of the risk factors for fasciitis summarized by Owen and Andrews (1994), three of these—diabetes, obesity, and hypertension—are relatively common in pregnant women. Like pelvic infections, these wound complications usually are polymicrobial and are caused by organisms that make up the normal vaginal flora. In some cases, however, infection is caused by a single virulent bacterial species such as group A β-hemolytic streptococcus

Early diagnosis, surgical debridement, antimicrobials, and intensive care are paramount to successfully treat necrotizing soft-tissue infections (Gallup, 2004; Urschel, 1999). Surgery includes extensive debridement of all infected tissue, leaving wide margins of healthy bleeding tissue. This may include extensive abdominal or vulvar debridement with unroofing and excision of abdominal, thigh, or buttock fascia. Death is virtually universal without surgical treatment, and rates approach 50 percent even if extensive debridement is performed.

Management of Superficial Wound Break Down:

Superficial wound separation can be managed by widely opening thewound followed by local care to promote granulation formation andclosure by secondary intention. [60]Nowadays treatment is mainly by dailydressings to promote granulation tissue followed by secondary suturing.

The principle in treating wound breakdown is thorough debridement topromote the healing process. Then perform moist-to-dry dressing changesusing saline soaked gauze every 8 hours. Also, the presence of significantbacterial contamination or necrotic tissue impedes wound healing. Thewound is inspected and daily debridement is done. No antibiotics aregiven without a specific indication. Patients are allowed to bath and canwash the wound while bathing. Generally, during the ensuing 3 to 5 daysthe wound will be covered with healthy granulation tissue. At this pointmost patients can safely carry out the remainder of their care with familyassistance, teaching, and medical supervision and assessment at regularintervals. In the proper setting this should represent no

increased risk tothe patient and allows subsequent recovery at home. In general it isimpossible to distinguish the resultant scar at 6 months after surgery fromthe wound that remained intact after primary closure.

In most patients now delayed reclosure of the disrupted wound isperformed. Several authors have described and refined this technique inthe gynecologic literature. Based on earlier observations dating totraumatic combat-associated wounds, the concept that clean woundscould be reclosed with a high success rate was studied. Walters et alshowed a success rate of 85% in 35 disrupted abdominal incisions whenthey were surgically reclosed. Compared with the control group patients,who received wound care and closure by secondary intention, closuretimes were reduced from 71.8 days to 15.8 days. Dodson et al.subsequently refined the closure technique to avoid the use of theoperating room or regional anesthesia and intravenous sedation, anddescribed the evolution of a procedure that could be performed at thebedside. Progressing from deep enbloc closure of the subcutaneoustissues and skin to the description of a technique of superficial skinclosure, the authors reported a success rate of 94% when wounds werereclosed after superficial wound separation.

Dodson et al study 2013 of bedside approach using a local anesthetic iswell tolerated by many patients and no patient required more than6 days of wound care before secondary suturing with mean time of4 days.

4. METHODOLOGY

Prospective Cohort Study

This is a prospective cohort study by classification is planned to be done from January 2018 to December 2018 (12 Months) at RMH, Thanjavur Medical College, Thanjavur.

All women undergoing caesarean sections in Raja Mirasudhar Hospital will be evaluated in the study to identify the patients who are developing any form of wound infection within 30 days from the date of caesarean section.

Among the patients identified, they are continuously monitored for development of signs of wound sepsis such as wound induration, wound edema and wound gapping

To identify the Risk factors, common organism causing wound sepsis and antibiotic sensitivity.

The study data will be evaluated using Relative risk and P value <0.05 taken as level of statistical significance.

- **INCLUSION CRITERIA**

All cases undergoing caesarean sections in RMH elective as well as emergency section.

- **EXCLUSION CRITERIA**

Caesarean sections done outside RMH.

5. RESULTS AND ANALYSIS

Our Study conducted at Thanjavur Medical College has included all patients undergoing C-section at RMH & patients developing wound infection were studied during the period of January 2018 to December 2018.

The total number of cases undergone C-section in RMH, Thanjavur during my study period was 6211. We have listed only the cases who had developed wound infection (92 cases) in our Master chart for further analysis and discussion.

The associated risk factors like socioeconomic status, BMI, Anemia, hypertension, diabetes, PROM, Handled outside, No. of PV examinations and induction were studied.

The relation between wound infection & duration of surgery , type of skin incision and closure, duration of hospital stay were analyzed.The most common organism causing wound infection identified and he antibiotic sensitivity pattern was also studied.

The results were tabulated and presented in chart for easy interpretation. Data are expressed as percentage (%). Chi Square test was applied to find statistically significant association between groups based on postoperative complications. P <0.05 was considered to be statistically significant

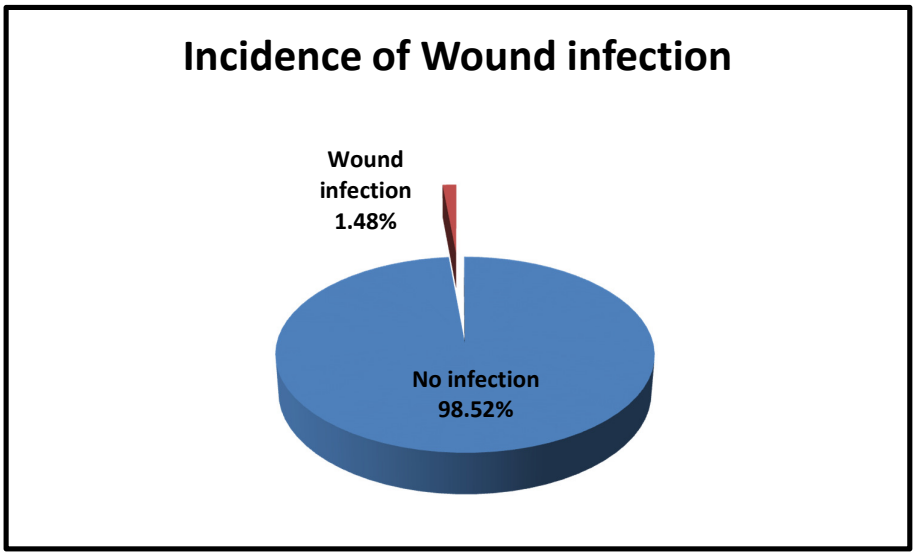
5.1 INCIDENCE OF WOUND INFECTIONS

In our study of 6211 patients, 92 cases developed wound infections which accounted for 1.48%.

Table 4: Incidence of wound infection

S.No.	Study Period	Total C-section	Wound infection
1	Jan-18	470	8
2	Feb-18	416	7
3	Mar-18	527	6
4	Apr-18	475	7
5	May-18	566	14
6	Jun-18	544	8
7	Jul-18	558	5
8	Aug-18	524	6
9	Sep-18	515	7
10	Oct-18	533	6
11	Nov-18	539	11
12	Dec-18	544	7
	Total cases	**6211**	**92**

Incidence of wound infection



5.2 AGE DISTRIBUTION

Table 5: Age Distribution

Age (In years)	Wound infected Cases	
	No.	%
< 20 years	1	1.1
21-35 years	89	96.7
> 35 years	2	2.2
Total	92	100.0
Range	19-39 years	
Mean	25.3 years	

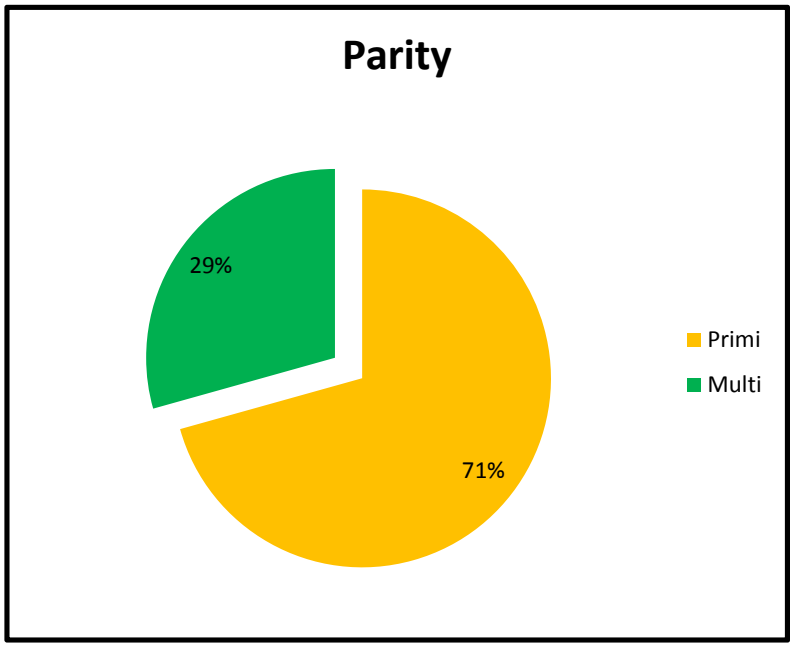
From the above table, it is found that there is no statistically significant correlation between the age of the patient and wound infection development.

5.3 PARITY

Table 6: Parity

Parity	Wound infected Cases	
	No.	%
Primi	65	70.7
Multi	27	29.3
Total	92	100.0

Distribution of type of gravida in my study population



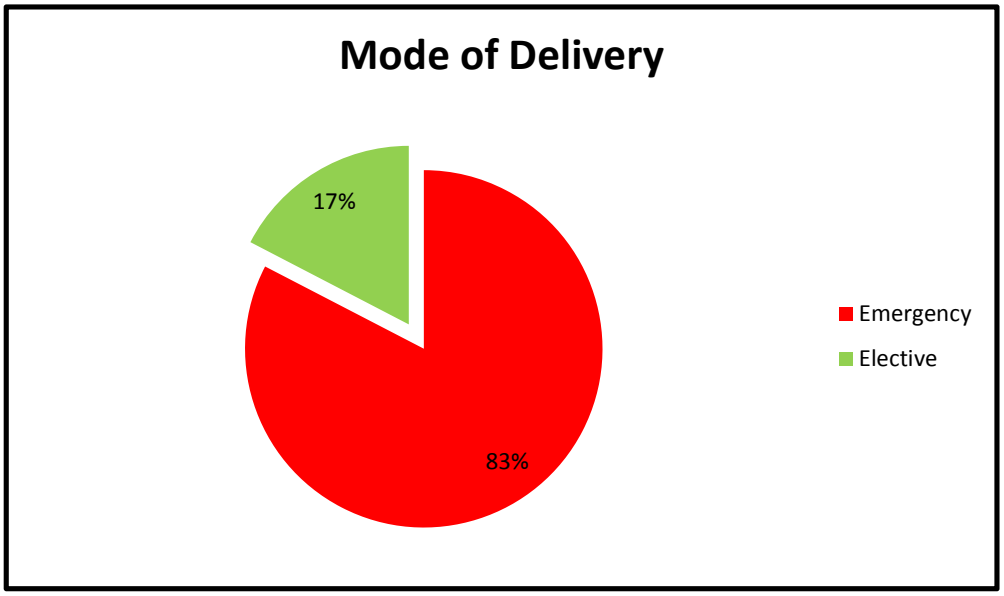
71% of Primi and 29 % of Multi gravida had wound infection.

5.4 MODE OF DELIVERY

Table 7: Mode of Delivery

Parity	Wound infected Cases	
	No.	%
Elective	16	17.4
Emergency	76	82.6
Total	92	100.0

Distribution of mode of delivery in my study population



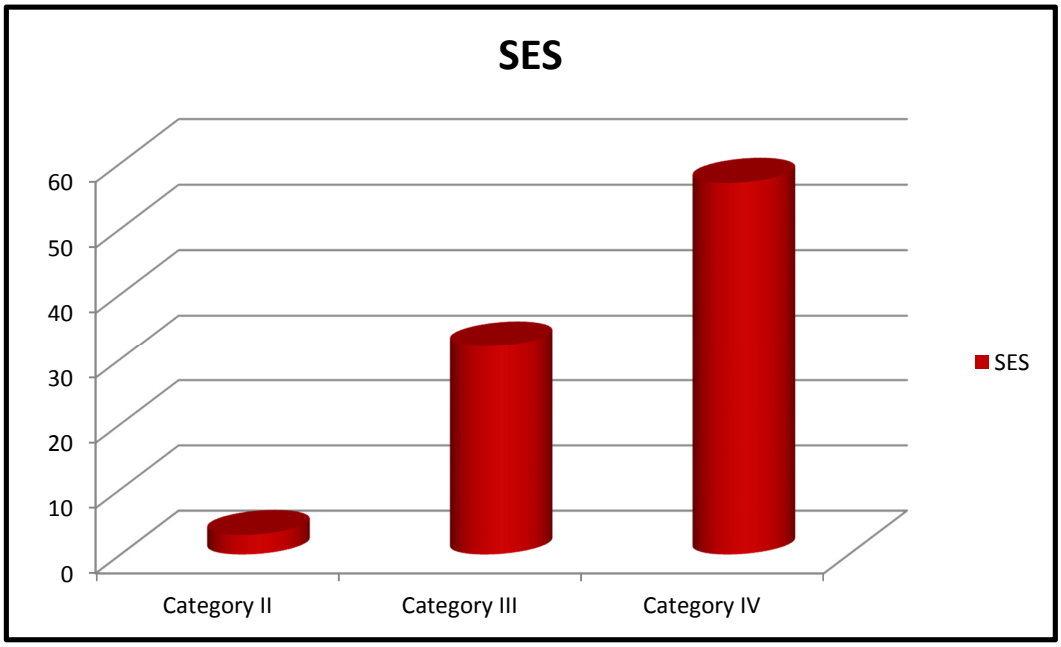
83% of Emergency and 17 % Elective had wound infection. Hence Emergency cases are more prone in Emergency cases

5.5 SOCIO ECONOMIC STATUS

Table 8: Socio Economic Status (SES)

SES	Wound infected Cases	
	No.	%
Class II	3	3.3
Class III	32	33.7
Class IV	57	62.0
Total	92	100

Distribution of socio economic status of my study subjects



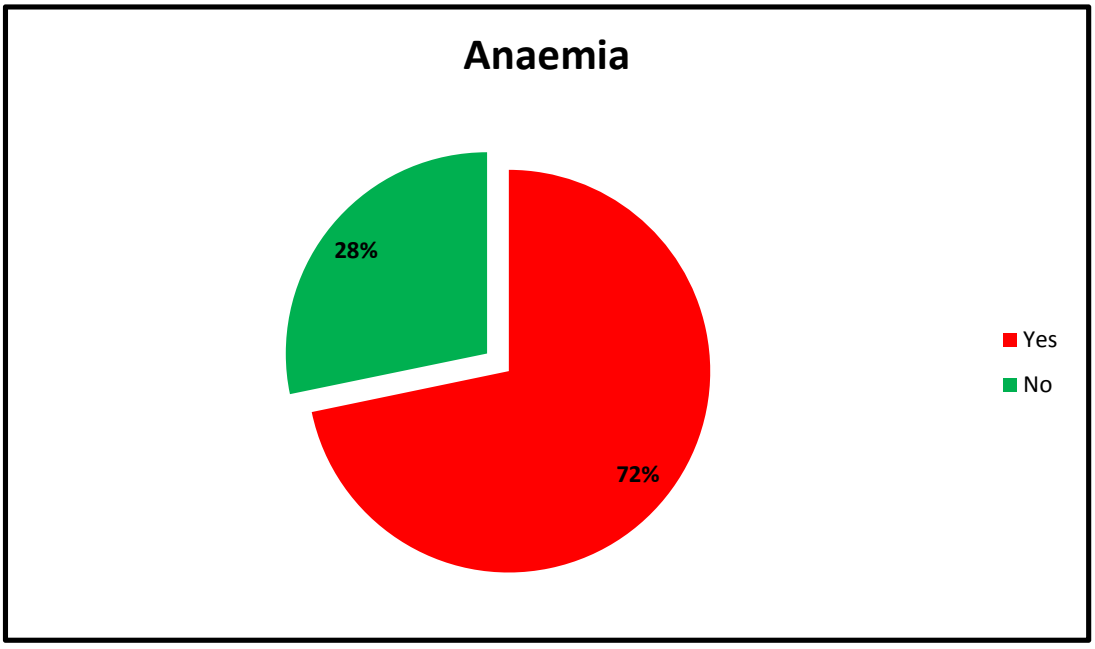
62% of women belonging to SES IV had wound infection. According to pearson chi square test, p value is <0.05, hence lower socio economic status is a significant risk factor.

5.6 ANAEMIA

Table 9:Frequency distribution Hemoglobin status of my study population

Anemia	Wound infected Cases	
	No.	%
Yes	66	71.7
No	26	28.3
Total	92	100.0

Distribution of status of anemia in my study subjects



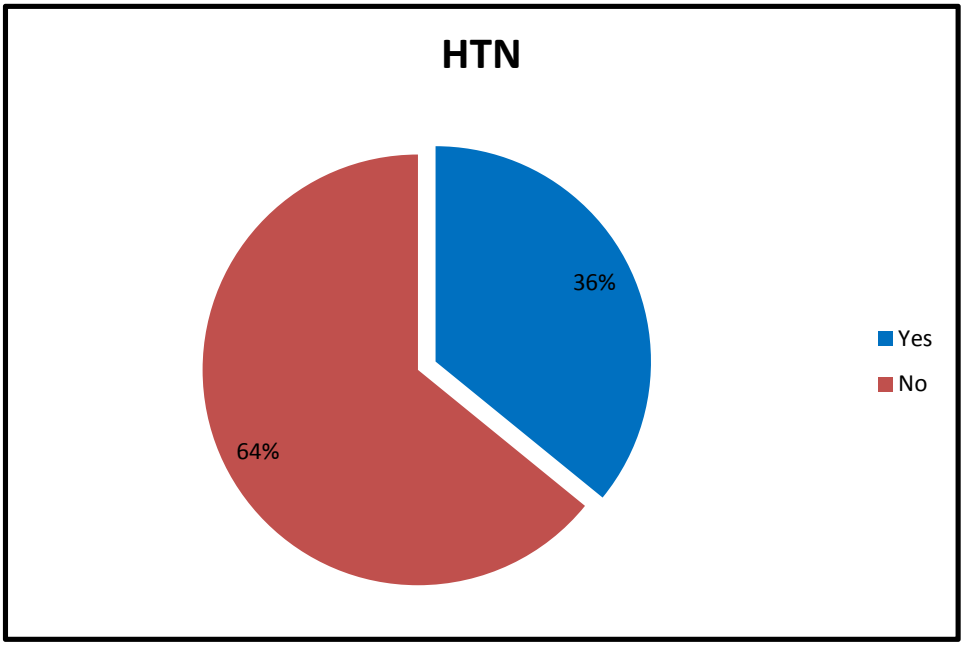
From above figure it was clearly shown that anemia predisposes to wound infection

5.7 HYPERTENSION

Table 10: HYPERTENSION status of my study population

HTN	Wound infected Cases	
	No.	%
Yes	33	35.9
No	59	64.1
Total	92	100.0

Distribution of status of Hypertension in my study subjects

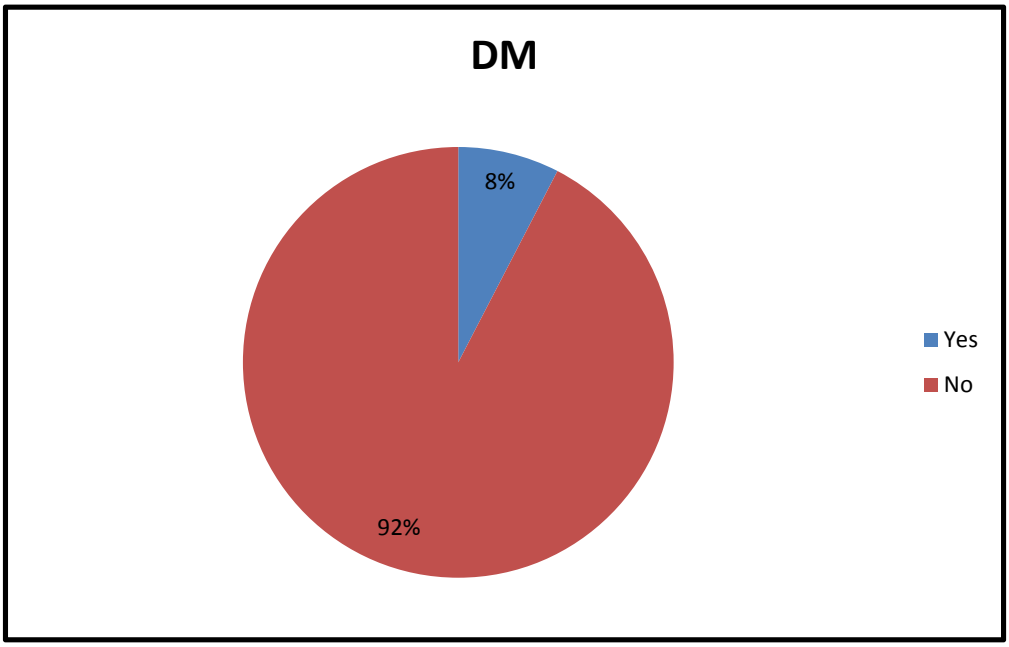


5.8 DIABETES MELLITUS

Table 11: Diabetes Mellitus status

DM	Wound infected Cases	
	No.	%
Yes	7	7.6
No	85	92.4
Total	92	100.0

Distribution of status of Diabetes Mellitus in my study subjects

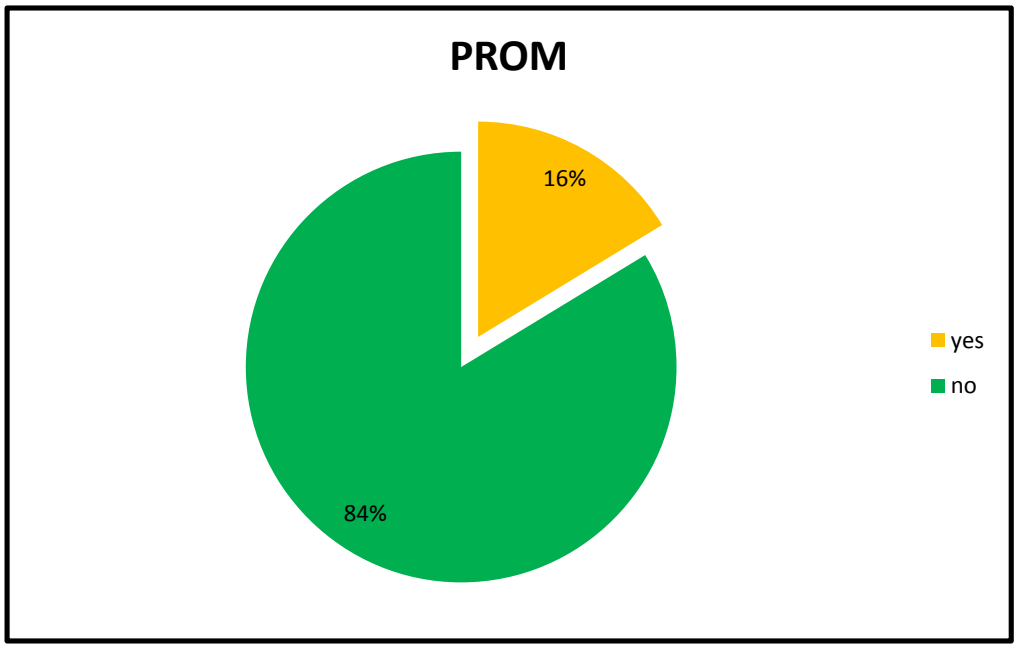


5.9 PROM

Table 12: PROM status

PROM	Wound infected Cases	
	No.	%
Yes	15	16.3
No	77	83.7
Total	92	100.0

Distribution of status of PROM in my study subjects

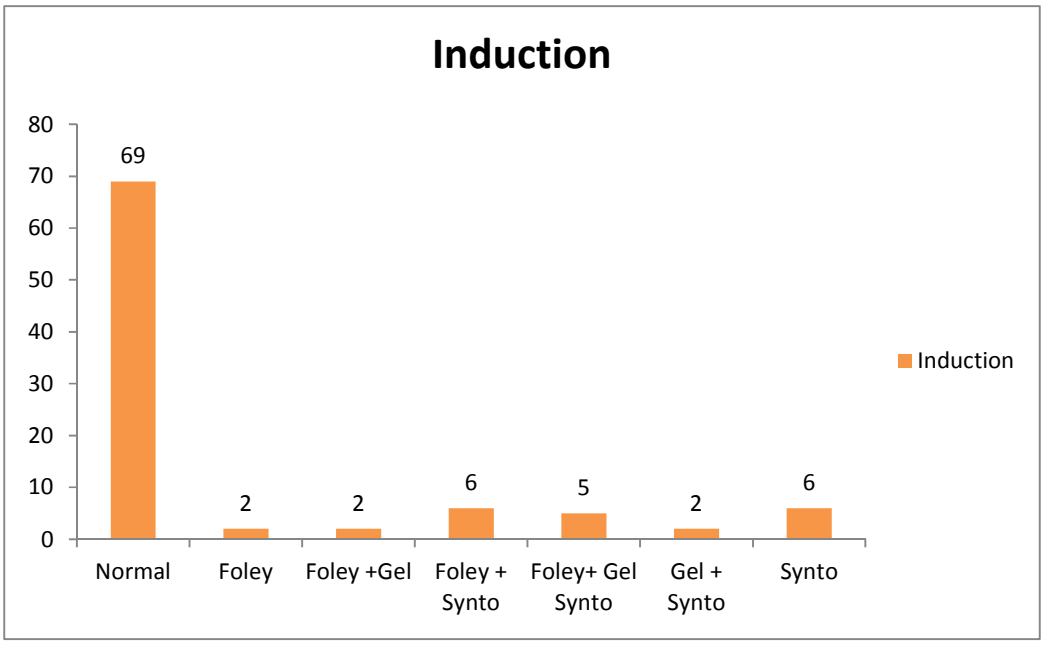


5.10 MODE OF INDUCTION

Table 13 : Mode of induction

Mode of induction	Wound infected Cases	
	No.	%
Normal	69	75.0
Foley	2	2.2
Foley + Gel	2	2.2
Foley + Synto	6	6.5
Foley + Gel+Synto	5	5.4
Gel+Synto	2	2.2
Synto	6	6.5
Total	92	100.0

Distribution of Mode of Induction in my study subjects

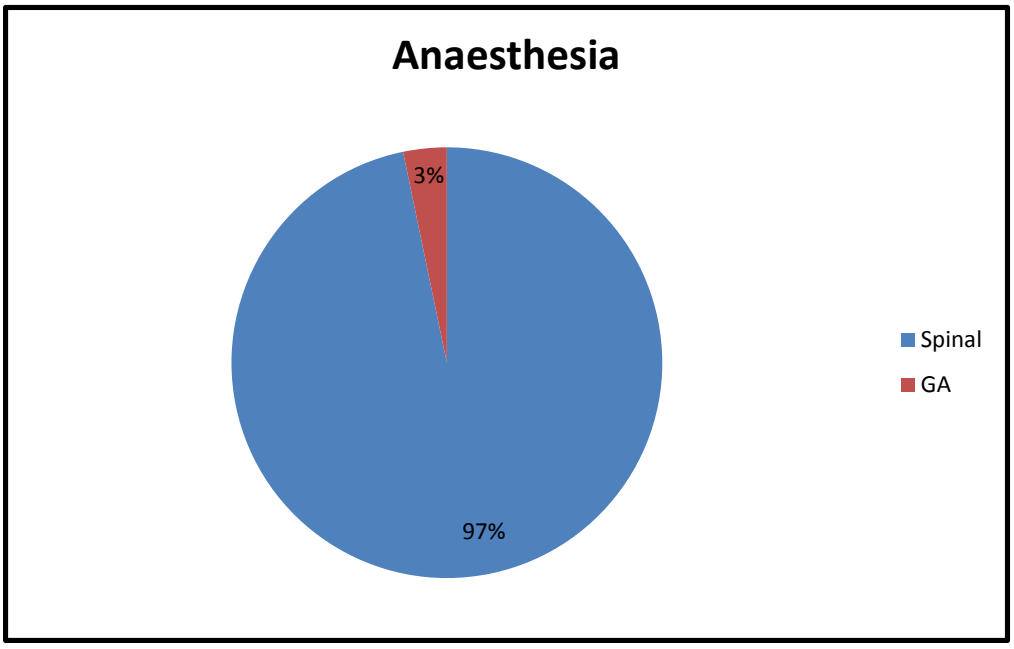


5.11 ANAESTHESIA

Table 14: Anaesthesia

Anaesthesia	Wound infected Cases	
	No.	%
Spinal	89	96.7
General	3	3.3
Total	92	100.0

Distribution of Type of Anesthesia in my study subjects

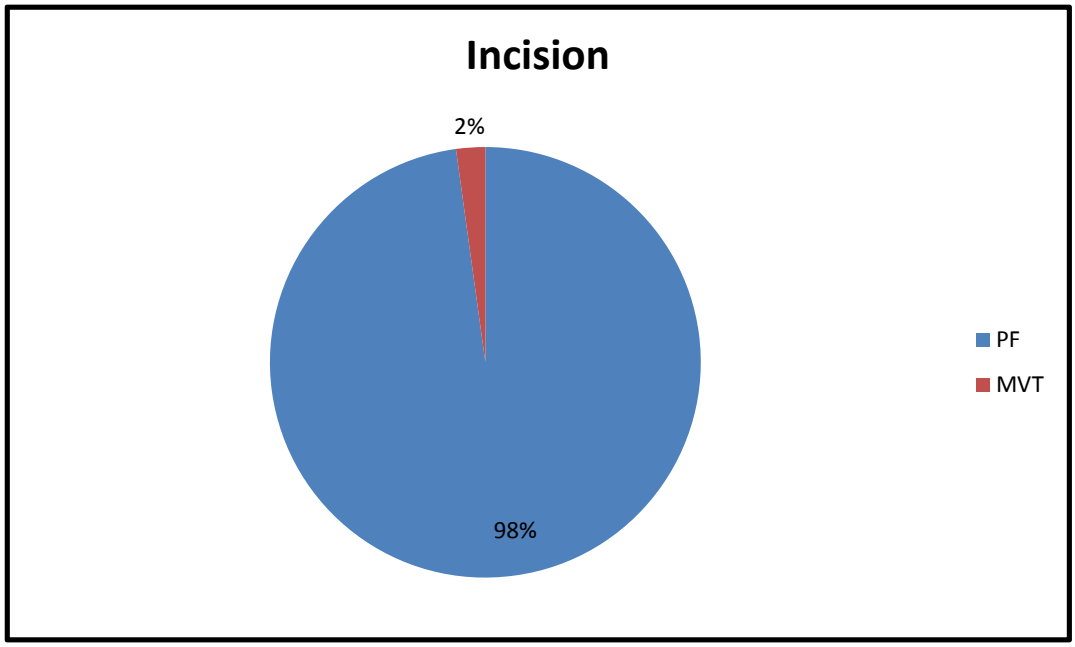


5.12 INCISION

Table 15: Incision

INCISION	Wound infected Cases	
	No.	%
PF= PFANNENSTEIN	90	97.8
MVT=MIDLINE VERTICAL	2	2.2
Total	92	100.0

Distribution of type of incision in my study subjects

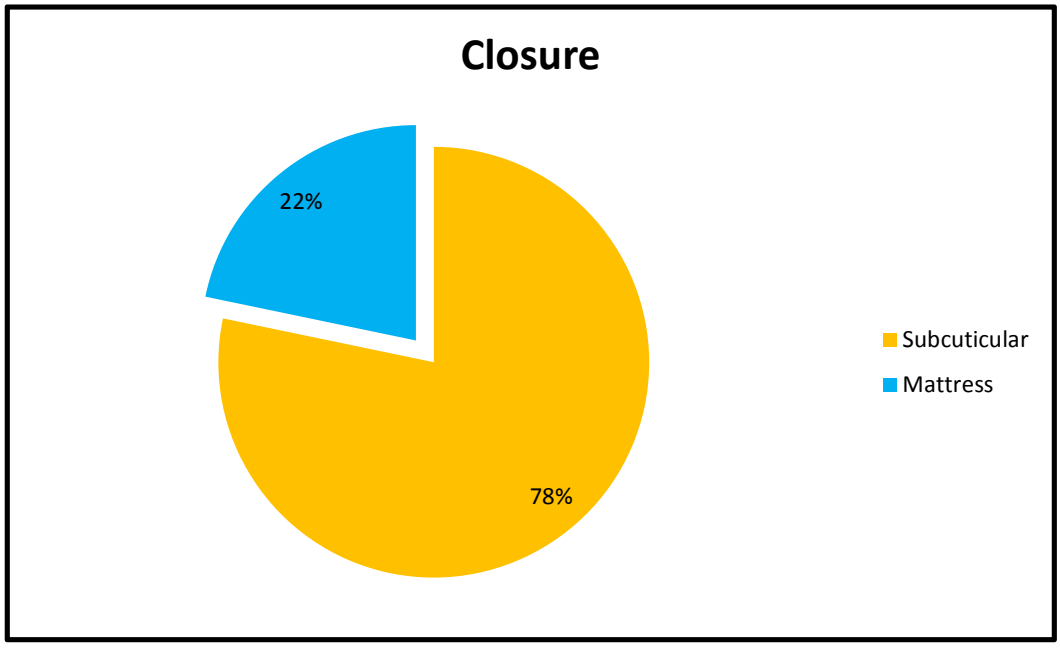


5.13 SKIN CLOSURE

Table 16: Skin Closure

Closure	Wound infected Cases	
	No.	%
Subcuticular	72	78.3
Mattress	20	21.7
Total	92	100.0

Distribution of type of skin closure in my study subjects



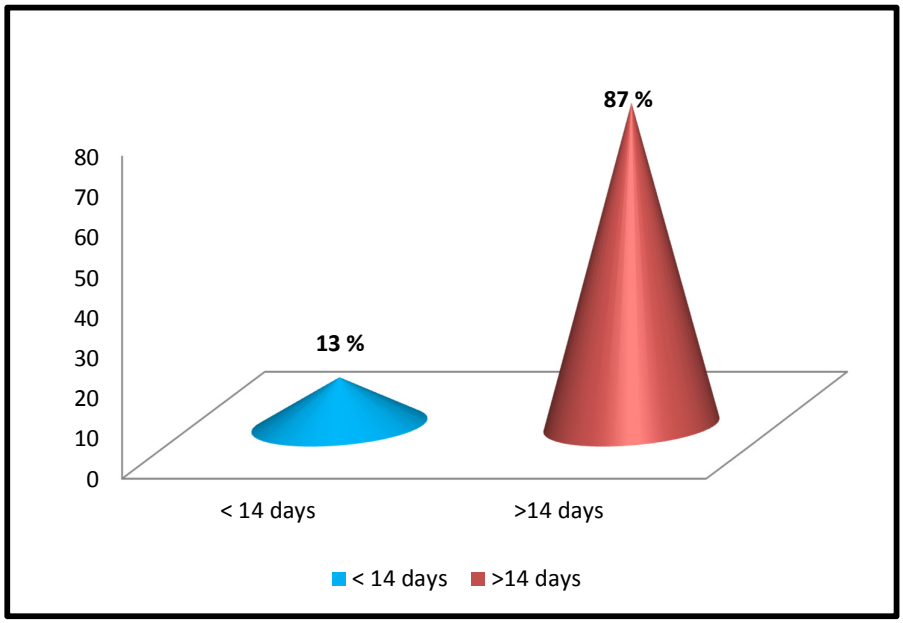
The percentage of wound infection was 78.3%with patient having subcuticular sutures, 21.7% of mattress sutures.

5.14 DURATION OF HOSPITAL STAY

Table 17: Duration of Hospital Stay

Duration of Stay	Wound infected Cases	
	No.	%
<14 days	12	13
>14 days	80	87
Total	92	100.0

Duration of Hospital Stay



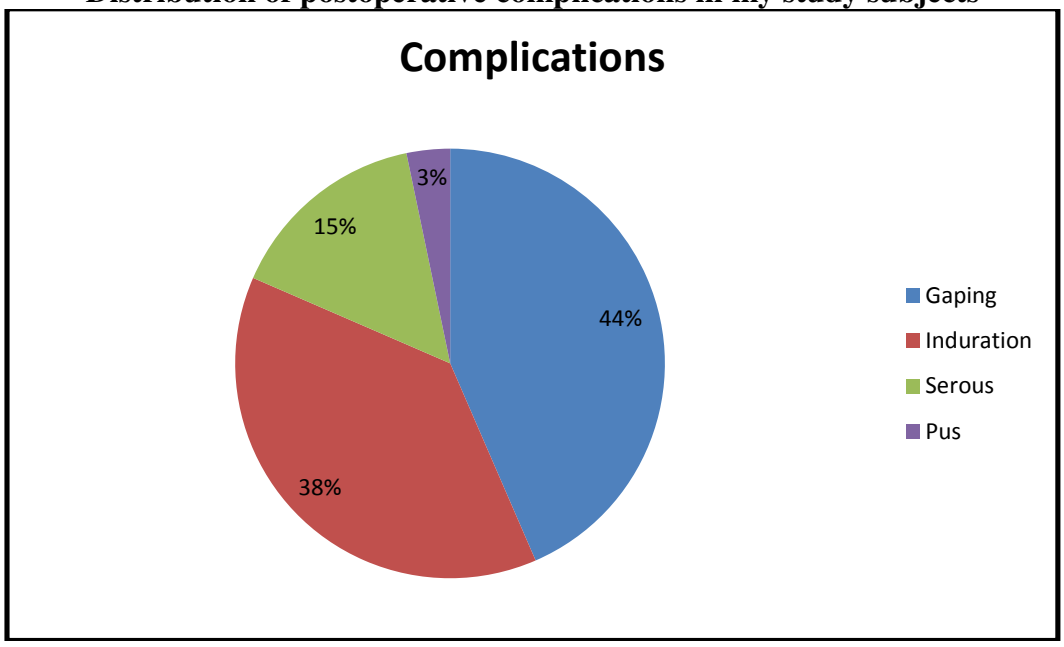
Wound infection leads to significant extension of hospital stay in 87% of the patients, which is statistically significant.

5.15 POST-OPERATIVE COMPLICATIONS

Table 18: Post-operative Complications

Closure	Wound infected Cases	
	No.	%
Gaping	40	43.5
Serous	14	15.2
Induration	35	38.0
Pus	3	3.3
Total	92	100.0

Distribution of postoperative complications in my study subjects



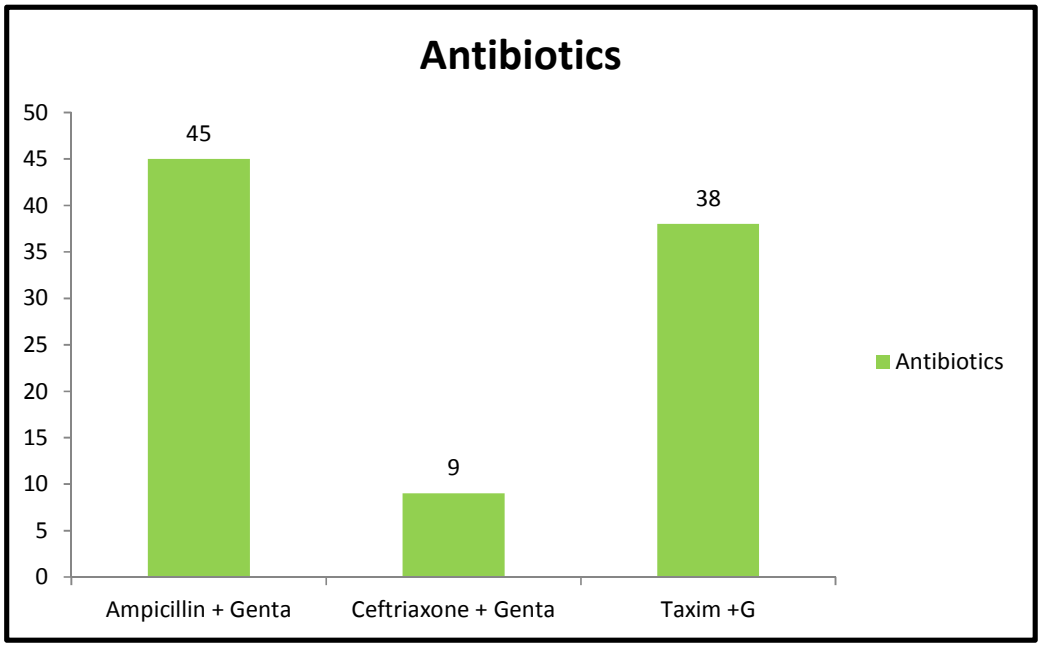
The various types of wound infection in my study are wound induration, wound gaping, wound discharge. All of those are superficial wound infection. None of the patients developed fascial dehiscence.

5.16 ANTIBIOTICS

Table 19: Antibiotics

AB	Wound infected Cases	
	No.	%
AMPICILLIN+G	45	48.9
CETRIAXONE+ G	9	9.8
CEFOTAXIM+ G	38	41.3
	92	100.0

Distribution of type of antibiotics used in my study subjects



5.17 COMPARISON OF VARIABLES

Table 20: Comparison of Baseline Characteristics based on Postoperative Wound Gaping

S.no	Variable	Wound Gaping			P value
		Yes	No		
1.	**Age group** <20	0	1	1	>0.05
	21-35	38	51	89	
	>35	2	0	2	
	Total	40	52	92	
2.	**Type of Parity**				>0.05
	Primi	30 (46.1)	35 (53.9)	65	
	Multi	10 (37)	17 (63)	27	
	Total	40	52	92	
3.	**LSCS**				>0.05
	Elective	5 (31)	11(69)	16	
	Emergency	35 (46)	41(54)	76	
	Total	40	52	92	
4.	**SES**				<0.05*
	Class II	2 (66.7)	1(33.3)	3	
	III	13 (42)	19 948)	31	
	IV	25 (43.9)	32(56.1)	57	
		40	52	92	

*significant P<0.05

While comparing elective vs. Emergency C section, 31% of elective cases and 46% of emergency cases developed gaping, but p value >0.05 which is statistically insignificant. But the relative risk is 1.5 which shows that emergency sections predisposes to wound infection

Low socioeconomic status predisposes to wound infection

Table 21: Comparison of Risk Factors with Postoperative Wound Gaping

S.no	Variable	Wound Gaping			P value
		Yes	No	Total	
1.	**BMI**	N (%)	N (%)		
	<19	0	0	0	< 0.05*
	20-25	11	19	30	
	26-30	22	29	51	
	>30	7	4	11	
	Total	40	52	92	
2.	**Anemia**				<0.05*
	Yes	35 (53.1)	31 (46.9)	66	
	No	5 (19.2)	19 (81.8)	26	
	Total	40	52	92	
3.	**Hypertension**				>0.05
	Yes	14 (42.4)	19 (57.6)	33	
	No	26 (44)	33 (56)	59	
	Total	40	52	92	
4.	**Diabetes mellitus**				>0.05
	Yes	5(71.4)	2 (28.6)	7	
	No	35 (41.1)	50 (58.9)	85	
		40	52	92	
5.	**PROM**				
	Yes	10 (66.7)	5 (33.3)	15	<0.05*
	No	30 (38.9	47 (61.1)	77	
		40	52	92	

*significant P<0.05

From above table , we conclude that BMI>26,Anemia and PROM are significant risk factors which are statistically significant

Table 22: Comparison of Risk Factors with Postoperative Wound gaping in my study subjects

S.no	Variable	Wound Gaping			P value
		Yes	No	Total	
1.	**No of Vaginal examinations**	N (%)	N (%)		
	0-4	25(35.7)	45(64.3)	70	<0.05*
	>4	15	7	22	
	Total	40	52	2	
2.	**Handled out**				
	Yes	7 (100)	0	7	<0.05*
	No	33 (40)	52(60)	85	
	Total	40	52	2	
3.	**Incision**				
	Pfannesteil	38 (42.2)	52(57.8)	90	>0.05
	Midline Vertical	2(100)	0	2	
	Total	40	52	92	
4.	**Closure**				
	SubCuticular	26 (36.1)	46(63.9)	72	<0.05*
	Mattress	14 (70)	6 (30)	20	
		40	52	92	
5.	**Anesthesia**				
	Spinal	38 (42.7)	51(57.3)	89	<0.05*
	GA	2(66.7)	1 (33.3)	3	
		40	52	92	

*significant P<0.05

From the above table, it is noted that No. of P/V more than 4 was associated with wound gaping, p value <0.05, which shows statistical significance.

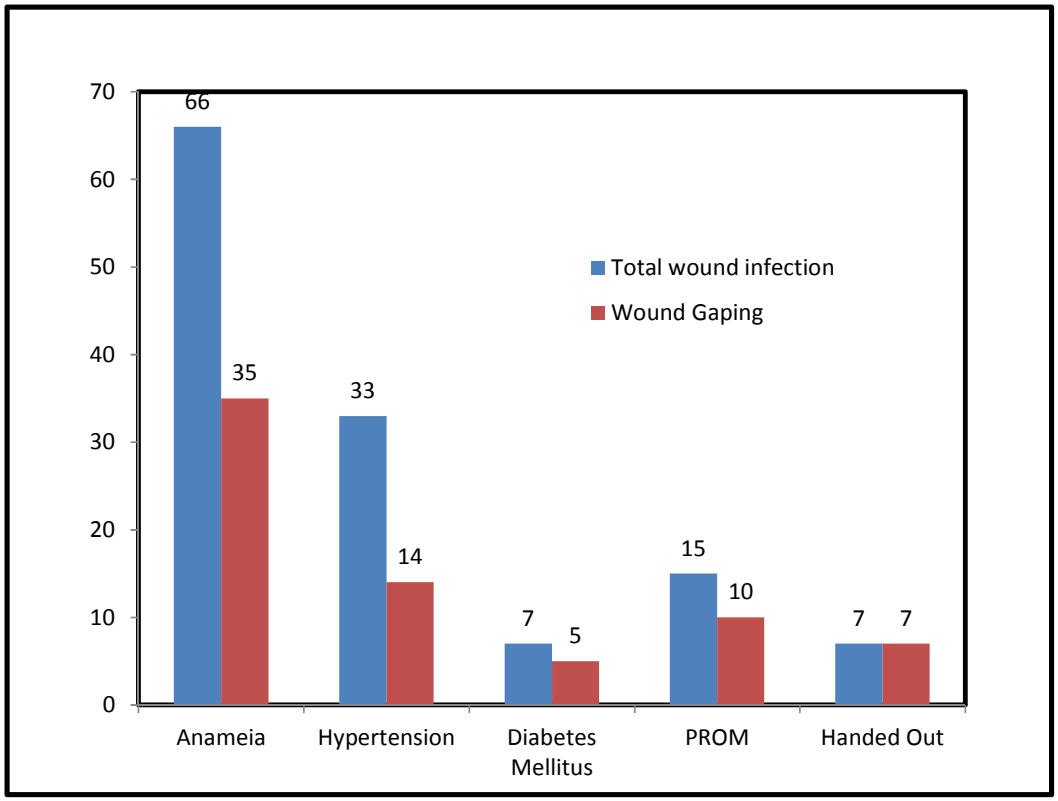
All of the 7 cases handled outside developed wound gaping.

About 90 patients who had pfannenstail incision, 38 of them developed gaping, but p value >0.05. So, type of incision does not influence gaping.

But, the type of skin closure does impact gaping. **Mattress suture accounts for 70% of gaping which is statistically significant.**

PROM & handled outside are the most important risk factors associated with gaping, both of them are statistically significant.

Distribution of cases with risk factors with postoperative wound gaping



The various risk factors associated with wound gaping in this study are Anemia (53.1%), Hypertension (42.4%), DM (71.4%), PROM (66.7%) and handled outside (100%)

Table 23: Comparison of indications for delivery with postoperative Wound Gaping

S.no	Variable	Wound Gaping		
	INDICATION	**Yes**	**No**	**Total**
1	Pre LSCS	8	17	25
2	Foetal distress	2	15	17
3	Failed induction	9	8	17
4	Abnormal presentation	1	4	5
5	Cord prolapsed	0	3	3
6	Non reactive NST	6	1	7
7	Obstructed Labor	6	0	6
8	Oligohydraminos	1	1	2
9	Placentabrevia	3	0	3
10	Abnormal Doppler	1	0	1
11	Abruption	0	2	2
12	Eclampsisa/unfavorable Cervix	3	1	4
	Total	40	52	92

Table 24: Comparison of Mode of Induction of Labor with postoperative Wound Gaping

S.no	Variable	Wound Gaping			P value
		Yes	No	Total	
1.	**Induction of labor**	N (%)	N (%)		
	Foley	10 (66.7)	5(33.3)	15	>0.05
	Gel	0	2 (100)	2	
	Synto	1 (16.7)	5 (83.3)	6	
	Total	11	12	23	

Among those patients who are induced, patients who had undergone Foley induction had higher rate of wound gaping, but p value >0.05, which is statistically insignificant.

Table 25: Comparison of intraoperative duration of surgery of with postoperative wound gaping

S.no	Variable	Wound Gaping			P value
		Yes	No	Total	
1.	**Intraoperative Duration**	N (%)	N (%)		
	< 40 mins	22(43)	29 (47)	51	>0.05
	>40 mins	18 (44)	23(46)	41	
		40	52	92	

5.18 ANTIBIOTIC SENSITIVITY PATTERN

Table 26: Different Types of organisms isolated in my Study Population

ORGANISM	No.	%
No Growth	35	38
Citrobacter	5	5.4
Cons	3	3.3
E.Coli	12	13.0
Klebsiella	9	9.8
No Growth	11	12.0
Proteus	1	1.1
Pseudomonas	1	1.1
S.Aureus	15	16.3
Total	92	100.0

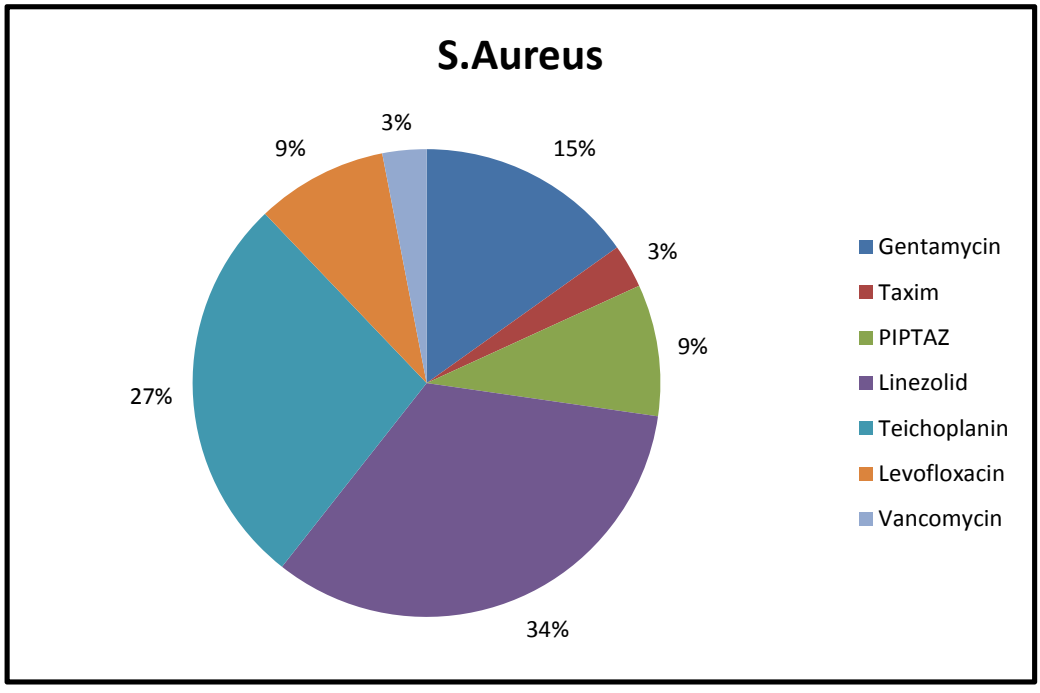
S-Aureus and E-Coli are the most common organisms found in pus, which leads to wound gaping

5.19 S-AUREUS SENSITIVITY

Table 27:S.Aureus Sensitivity

Antibiotic sensitivity	No.
Gentamycin	5
Cefotaxim	1
Piperacillintazobactum	3
Linezolid	11
Teichoplanin	9
Levofloxacin	3
Vancomycin	1

S.Aureus Sensitivity



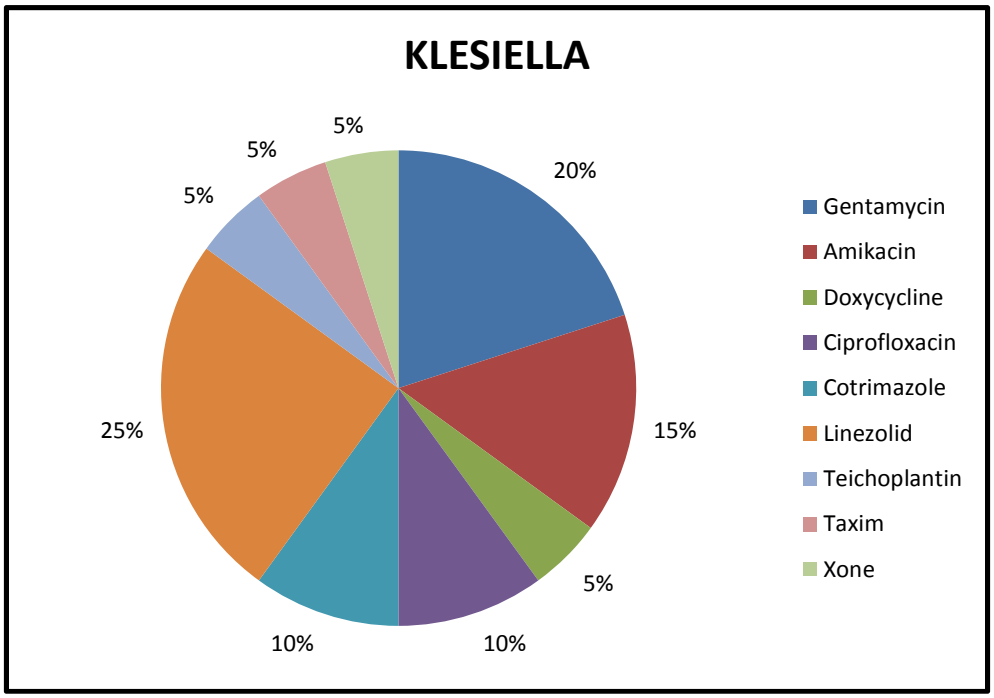
S-Aureus is most sensitive to Linezolid and Teichoplanin.

5.20 KLESIELLA SENSITIVITY

Table 28: Klesiella Sensitivity

Antibiotic sensitivity	No.
Gentamycin	4
Amikacin	3
Doxycycline	1
Ciprofloxacin	2
Cotrimazole	2
Linezolid	5
Teichoplantin	1
Cefotaxim	1
Ceftriaxone	1

Klesiella Sensitivity



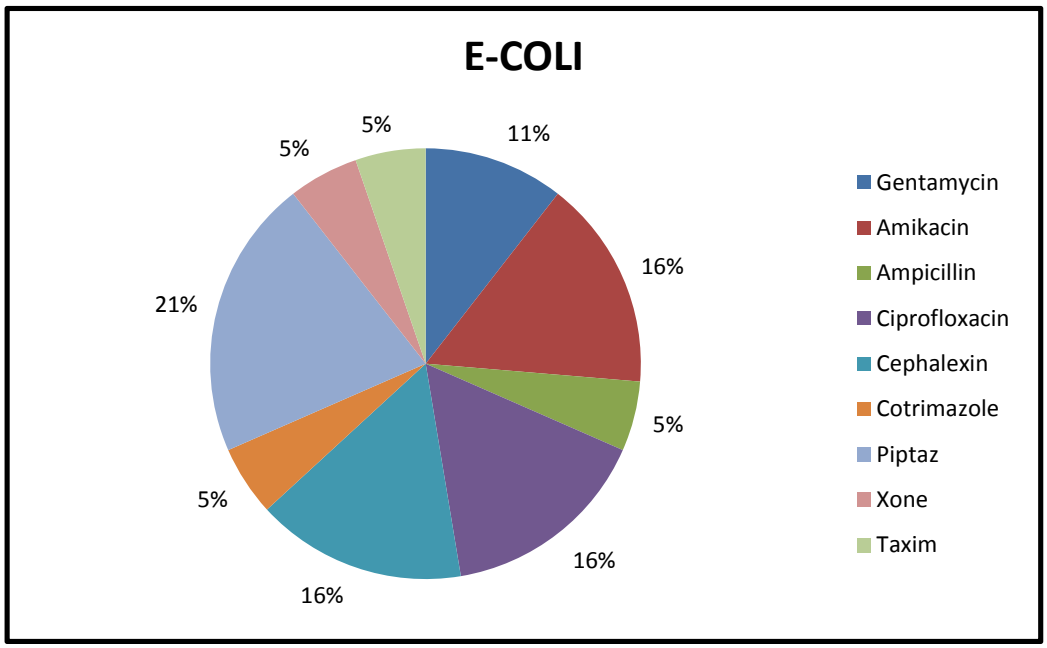
Klesiella is most sensitive to Linezolid and Gentamycin.

5.21 E-COLI SENSITIVITY

Table 29: E-Coli Sensitivity

Antibiotic sensitivity	No.
Gentamycin	2
Amikacin	3
Ampicillin	1
Ciprofloxacin	3
Cephalexin	3
Cotrimazole	1
Piperacillintazobactum	4
Ceftriaxone	1
Cefotaxim	1

E-Coli Sensitivity



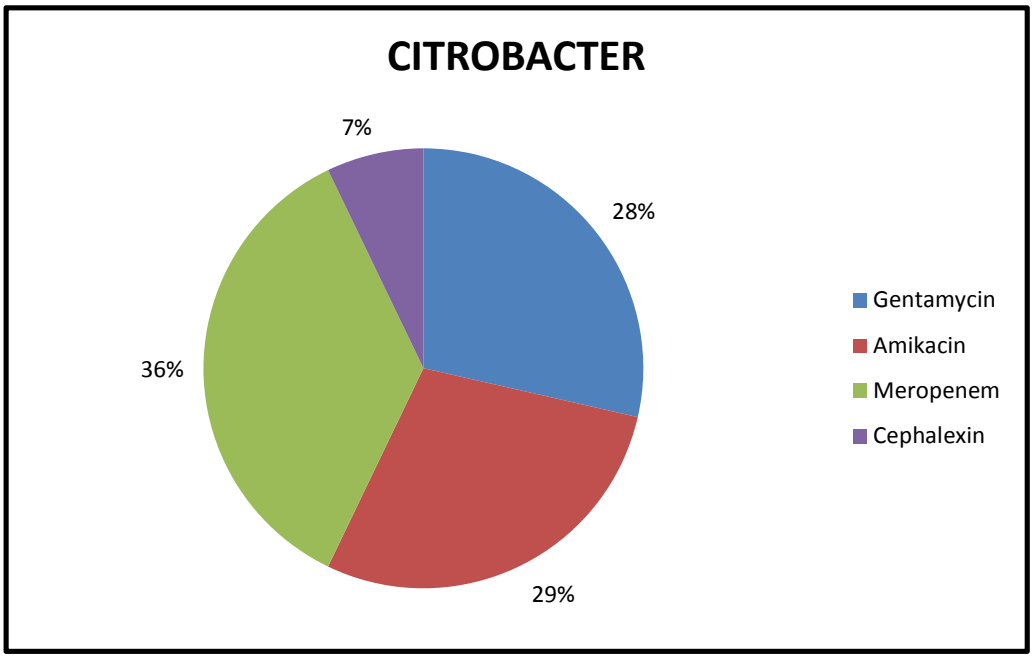
E-Coli is most sensitive to Piperacillintazobactum and Cephalexin.

5.22 CITROBACTER SENSITIVITY

Table 30: Citrobacter Sensitivity

Antibiotic sensitivity	No.
Gentamycin	4
Amikacin	4
Meropenem	5
Cephalexin	1

Citrobacter Sensitivity



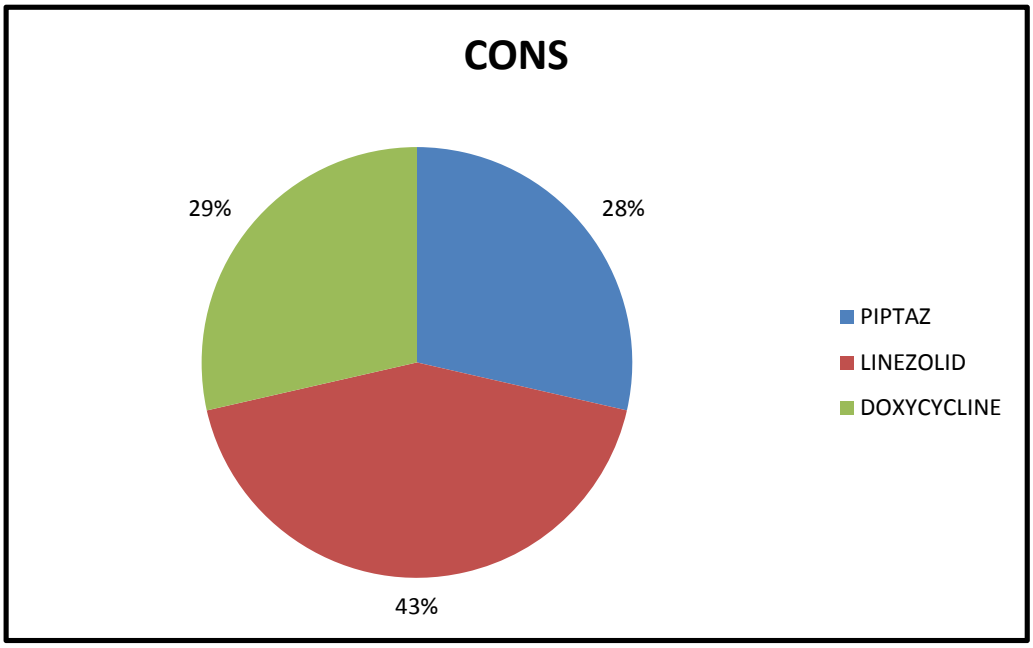
Citrobacter is most sensitive to Meropenem and Amikacin.

5.23 CONS SENSITIVITY

Table 31: CONS Sensitivity

Antibiotic sensitivity	No.
Piperacillintazobactum	2
Linezolid	3
Doxycycline	1

CONS Sensitivity



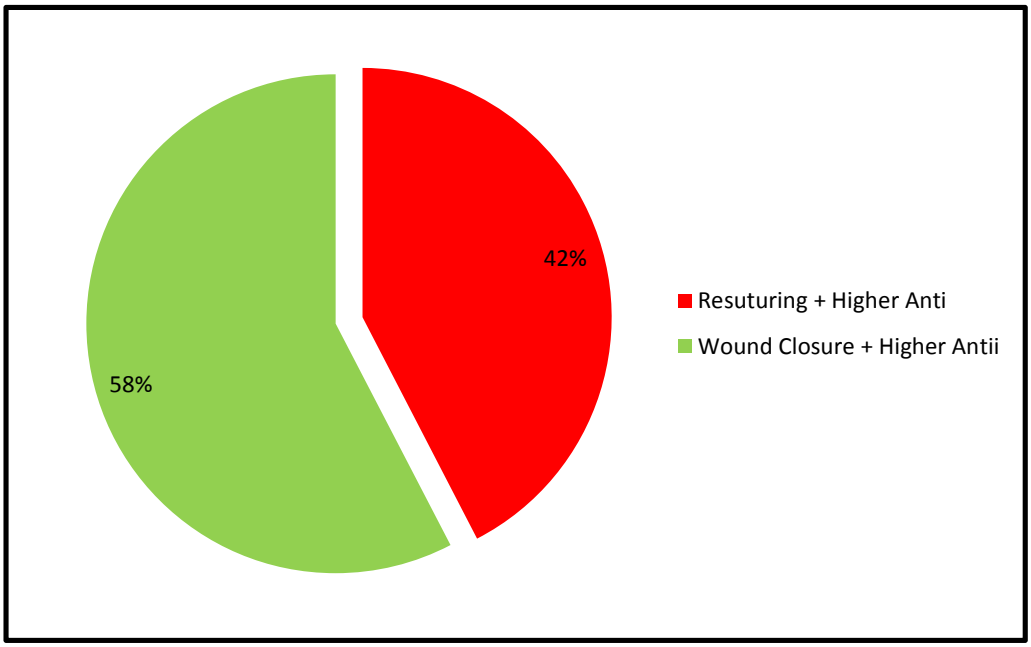
CONS is most sensitive to Linezolid and Doxycycline.

5.24 TREATMENT

Table 32: Treatment

Treatment	Wound infected Cases	
	No.	%
Resuturing + Higher Anti	39	42.4
Wound care + Higher Anti	53	57.6
Total	92	100.0

Distribution of type of treatment in my study subjects



Among those patients developed wound infection, 39 cases has undergone wound resuturing, 53 patients responded to Local wound care and higher antibiotics.

6. DISCUSSION

The Current Study was done with screening 6211 patients who underwentelective and emergency caesarean section during the study period of Jan 2018 to Dec 2018 at Thanjavur Medical College for wound infection. Out of those patients, 92 had developed wound infection.

The wound infection rates vary from 0.5 – 15% in review of literature, the incidence of wound infection in the present study was 1.48%. The study conducted by HansaDhar et al (2014)[5] showed incidence of 2.66%.In a study conducted by Simon M.scheck et al (2017)[2] showed incidence of 5.2% which included 2231 subjects. In a study of SSI following caesarean operation at a Jordanian teaching hospital by Mariam et al (2017)[7], showed an incidence of 15%.

Table 33: SSI Incidence in various studies

S.No.	Study of SSI	SSI Incidence
1	My present Study (2018)	1.48 %
2	HansaDhar et al (2014)[5]	2.66 %
3	Simon M.Schek et al (2017)[2]	5.2 %
4	Mariam et al (2017)[7]	15 %

In a study conducted by HansaDhar et al, the rate of wound infection was more in emergency LSCS (1.5%) than in elective 1.1%. In a study conducted by Simon M Scheck et al the percentage of SSI in emergency LSCS found to be 63.5%. In my present study the rate of wound infection in emergency LSCS was 1.2%, whereas elective was only 0.2%. Hence, the incidence of wound infection was

higher in emergency LSCS and patients undergoing emergency LSCS has 1.5 times more prone to wound infection.

Table 34: SSI Incidence in Emergency Vs Elective LSCS

S.NO	Study of SSI	Emergency LSCS	Elective LSCS
1	HansaDhar et al(2014)	**1.5%**	**1.1%**
2	My Present Study	**1.2%**	**0.2%**

The various risk factors associated with the wound infection in my study like Anemia (71.7%), HTN (35.9%), Diabetes (7.6%), and PROM (16.3%) Handled outside (7.6%) were comparable with results obtained by AR Mahale study (2008) which showed PROM (20.8.%), Anemia (22 %).In my study, Diabetes contributes to 7.6% of wound infection and it increases the risk by 2 times, whereas study conducted by HansaDhar et al & Simon M Scheck et al shown that diabetes increases the rate of SSI by 3 times and 7.7 times respectively. Anemia,PROM and handled outside was significant risk factors for wound infection. Anemia increases the risk of wound infection by 2.5 times.

In a study conducted by HansaDharet al[5] showed that hypertension increases the risk of wound infection by 3 times and in my study hypertension contributes to 35.9% of wound infection.

BMI > 36 and subcutaneous tissue thickness > 2cm wasconsidered to be significant risk factor in a study conducted by Mariam et al[7],whereasin my present study, BMI > 26 is considerd to be the significant risk factor.

In my study, Number of PV examinations more than 4 was found to be significant risk factor for wound infection which is statistically significant.Patients who undergone induction of labor with foley found to have higher risk of wound gaping.

The most common organism isolated inmy study was S.Aureus followed by E-Coli.

Table 35: Common Organisms in various studies

S.No.	Study of SSI	Most Common Organism
1	My present Study (2018)	S-Aureus
2	HansaDhar et al (2014)[5]	S-Aureus (31.27 %)
3	Simon M.Schek et al (2017)[2]	S-Aureus
4	Mariam et al (2017)[7]	S-Aureus (37.3 %), E-Coli (13.4%)

In a study conducted by HansaDhar et al[5] showed that S-Aureus was most sensitive to Aminoglycoside, whereas in my study S-Aureus found to be sensitive to Linezolid. Hence, it is found that antibiotic resistance is increasing among the organisms causing SSI.

Only 42% of patients went for secondary wound resuturing and other patients were treated using local wound care and higher antibiotics.

Wound infection leads to significant extension of hospital stay in 87% of patients.

7 CONCLUSION

Caesarean delivery is one of the most frequent surgical interventions performed around the world and accounts for 60% of all deliveries.Post caesarean wound infection is a major cause of prolonged hospital stay , increases maternal morbidity and increased medical costs which poses a significant burden to health care system.

Caesarean section is a clean contaminated type of surgery where procedure related chance of infection is less. Hence proper assessment of risk factors that predisposes to SSI is critical for developing preventive strategies

The incidence of wound infection in my study was 1.48%. This present study shows that Anemia, PROM , Handled outside , multiple pervaginal examinations , prolonged induction are predominant risk factors leading to wound infection.

Anemia and Diabetes increases the risk of wound infection by 2.5 times and 2 times respectively.The commonest organism isolated is Staphylococcus Aureus which is most sensitive to Linezolid. Superficial wound infection is the most frequent infection which is treated by local wound care& higher antibiotics in 58% of patients and only 42% percent of them requires wound Re-Suturing & Higher Antibiotics.

Strategies for prevention of this morbidity must aim to correct anemia , to avoid prolonged hospital stay prior to delivery ,to correct maternal comorbidities prior to surgery and Strict adoption of asepsis.SSI surveillance must be done as a part of HAI audit which aims at improving Quality control measures and infection control practices.

BIBLIOGRAPHY

1. Williams Obstetrics 25th edition -cunningham, Leveno, Bloom et al; 2019 (chapter 26 , 30 , 37 , 48, 57 , 64)
2. Simon M. Scheck: Caesarea n section wound infection surveillance: Information for action; November 2017.(518 – 523p)
3. DC Dutta's Textbook of Obstetrics 2018;chapter 22 , 34 , 36 , 39 (327 - 340 ,591 – 596,522 – 529)
4. Sivan Zuarez-easton; Postcaesarean wound infection: prevalence, impact, prevention, and ; management challenges; Feb 2017 (81 – 86p)
5. Hansa Dhar; A Study of Post-Caesarean Section Wound Infections in a Regional Referral Hospital, Oman; Apr 2014 (212- 217p)
6. Gideon K. Kurigamb; POST CESEREAN WOUND SEPSIS: Recognizable Risks and Causes at a Rural Ugandan Hospital; Jul 2018 (1 – 4p)
7. Mariam Hantash Abdel Jalil; Surgical site nfections following caesarean operations at jordanina teaching hospital; Sep 2017 (3-8p)
8. Robbins & Cotran Pathological basics of disease; chapter 1 - 4
9. Te Lindes operative gynaecology; 10th edition ;2008; Chapter 8: Preop care & Chapter 9: Postop care, Chapter 11: Post operative infection, prevention & Management; Chapter 13:Wound healing (113-240p)
10. Cliping not shaving guidelines ; March 2014 (1 – 16p)
11. American college of surgeons and surgical infection society; SSI guidelines ; 2016 update

12. Prnciples of epidemiology in public health practice; 3rd edition
13. Alison M Pyper, Manuel W.Impact of antibiotic prophylaxis on wound infection after caesarean section in a situation of expected higher risk. American journal of obstetrics and gynaecology 2001;29:85-8
14. AR Mahale et al.Caesarean section morbiditiy- a study of 1775 cases of abdominal wounds.Obs anb Gyanec.Today; 2008; 8:329331
15. Fabian TS, Kaufman HJ, Lett ED, et al. The evaluation of sub atmospheric pressure and hyperbaric oxygen in ischemic fullthickness wound healing. Am Surg 2000; (1136 – 1143p)
16. John Hopkins Medicine ; Vacuum assisted closure of wounds;
17. SS Scherer; The mechanism of action of the vacuum assisted closure device; NCBI article 2008
18. Arias: Practical guide to High-Risk Pregnancy & Delivery. A South Asian Perspective; 4th edition-2016 Chapter 15: Diabetes in pregnancy (254-268p); Chapter 4: Fetal infection (55p)
19. Nwankwo EO, Ibeh I, Enabulele OI. Incidence and risk factors of surgical site infection in a tertiary health institution in Kano, northwestern Nigeria. Int J Infect Control 2012; (1–6p)
20. Aboorva Sankar Shastri; Essential of Medical microbiology; Jan 2018 ; Chap- Bacterology
21. Wall PD, Deucy EE, Glantz JC, Pressman EK. Vertical skin incisions and wound complications in the obese parturient. Obstet Gynecol 2003; 102:952–6.

22. Nisa M, Naz T, Afzal I, Hassan L. Scope of surgical site infections (SSI) in obstetrics and gynaecology. J Postgrad Med Inst 2005; (438-441p).
23. Macones GA, Cleary KL, Parry S, Stamilio DM, Cahill AG, Odibo AO, et al. The timing of antibiotics at caesarean: A randomized controlled trial. Am J Perinatol 2012: (273-300p)
24. Anathanarayan & Panicker's: Textbook of Microbilogy; 9th edition
25. Arulkumaran: The management of labour: 3rd edition : Chapter 23: Induction of labour (368-353p)
26. Olsen MA, Butler AM, Willers DM, Devkota P, Gross GA, Fraser VJ. Risk factors for surgical site infection after low transverse caesarean section. Infect Control Hosp Epidemiol 2008; (477–478p)
27. Ngonzi J. T (2016) Puerperal sepsis, the leading cause of maternal deaths at a Tertiary University Teaching Hospital in Uganda. . BMC pregnancy and childbirth: 207p
28. Francis (2011) ACOG Practice Bulletin No. 120: use of prophylactic antibiotics in labor and delivery. Obstet Gynecol: (1472-1483p)
29. Charoenboon C, Srisupundit K, Tongsong T. Rise in caesarean section rate over a 20-year period in a public sector hospital in northern Thailand. Arch Gynecol Obstet. 2013 (47–52p)
30. Schneid-Kofman N, Sheiner E, Levy A, Holcberg G. Risk factors for wound infection following caesarean deliveries. Int J Gynecol Obstet. 2005;(10–15p)

31. Salim R, Braverman M, Teitler N, Berkovic I, Suliman A, Shalev E. Risk factors for infection following caesarean delivery: an interventional study. J Matern Neonatal Med. 2012;(2708–2712p)
32. Gur R, Duggal SD, Rongpharpi SR, et al. Post caesarean surgical site infections. Arch Clin Microbiol. 2015; (1–6p)
33. Gilstrap LC, Cunningham FG. The bacterial pathogenesis of infection following caesarean section. Obstet Gynecol. 1979; (545–549p)
34. Brown J, Thompson M, Sinnya S, et al. Pre- incision antibiotic prophylaxis reduces the incidence of post- caesarean surgical site infection. J Hosp Infect 2013; (68–70p)
35. Costantine MM, Rahman M, Ghulmiyah L, et al. Timing of perioperative antibiotics for caesarean delivery: a metaanalysis. Am J Obstet Gynecol 2008;
36. Ahmadzia HK, Patel EM, Joshi D, et al. Obstetric surgical site infections. Obstet Gynecol 2015; (708–715p)
37. Opøien HK, Valbø A, Grinde-Andersen A, Walberg M. Post- caesarean surgical site infections according to CDC standards: rates and risk factors. A prospective cohort study. Acta Obstet Gynecol Scand 2007; (1097–1102p)
38. Ward VP, Charlett A, Fagan J, Crawshaw SC. Enhanced surgical site infection surveillance following caesarean section: experience of a multicentre collaborative post- discharge system. J Hosp Infect 2008; (166–173p).

39. Ghuman M, Rohlandt D, Joshy G, Lawrenson R. Post- caesarean section surgical site infection: rate and risk factors. N Z Med J 2011; (32–36p)
40. Wloch C, Wilson J, Lamagni T, et al. Risk factors for surgical site infection following caesarean section in England: results from a multicentre cohort study. BJOG 2012; (1324–1333p)
41. Costantine MM, Rahman M, Ghulmiyah L, et al. Timing of perioperative antibiotics for caesarean delivery: a metaanalysis. Am J Obstet Gynecol 2008; 301
42. Hickson E, Harris J, Brett D. A journey to zero: reduction of post- operative caesarean surgical site infections over a five- year period. Surg Infect (Larchmt) 2015; (174–177p)
43. Gyte GMI, Dou L, Vazquez JC. Different classes of antibiotics given to women routinely for preventing infection at caesarean section. Cochrane Database Syst Rev 2014. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25402227
44. Khalid B M saeed, R. A (2017) incidence of surgical site infection following caesarean section: a systematic review and meta- analysis protocol. BMJ open
45. Mahesh CB, S. S (2010) A prospective study of surgical site infections in a teaching hospital. J Clin Diagn Res 4: (3114-3119p)
46. Wloch C, W. J (2012) Risk factors for surgical site infection following caesarean section in England: Results from a multicentre cohort study.(1324-1333p)

47. H G (2011) Reducing surgical site infection following caesarean section. Nurs Stand (35-40p)
48. Dyrkorn OA, K. M (2012) Reducing post-caesarean surgical wound infection rate: an improvement project in a Norwegian maternity clinic. BMJ Qual Saf (206-210p)
49. Smaill FM, G. R (2014) Antibiotic prophylaxis versus no prophylaxis for preventing infection after caesarean section. Cochrane Database Syst Rev 10.
50. Chadli M, Rtabi N, Alkandry S, Koek JL, Achour A, Buisson Y, et al. Incidence of surgical wound infections: A prospective study in the Rabat Mohamed-V Military Hospital, Morocco. Med Mal Infect 2005;(218–222p)
51. Scottish Intercollegiate Guidelines Network. National Clinical Guideline 104: Antibiotic prophylaxis in surgery. From: www. sign.ac.uk/pdf/sign104.pdf Accessed: Mar 2014.
52. Gong SP, Guo HX, Zhou HZ, Chen L, Yu YH. Morbidity and risk factors for surgical site infection following caesarean section in Guangdong Province, China. J Obstet Gynaecol Res 2012; (509–515p)
53. Carlson MA. Acute wound failure. Surg Clin North Am. 1997; 77: (607-636p)
54. Robert S, Maccato M,Faro S,et al. The microbiology of post caesarean wound morbidity. Obstet Gynecol 1993 ;(383 – 386p)

55. Dodson MK, Meeks GR. A randomized comparison of secondary closure and secondary intention in patients with superficial wound dehiscence. Obstet Gynecol. 1992; (321 – 324p)
56. Tokars JI, Culver DH, Mendelson MH, Sloan EP, Farber BF, Fligner DJ, et al. Skin and mucous membrane contacts with blood during surgical procedures: risk and prevention. Infect Control Hosp Epidemiol 1995; (703 – 711p)
57. Mastro TD, Farley TA, Elliot JA, Facklam RR, Perlu JR, Hadler JL, et al. An outbreak of surgical wound infections due to group A Streptococcus carried on the scalp. N Engl J Med 1990; (968 – 972p)
58. Tamara C.Takoudes, MD, Sherry et al. Risk of caesarean wound complications in diabetic gestations. American journal of obstetrics and gynaecology 2004; (958-963p)
59. Schoen F J, Robbins LS, Kumar V, Cotran RS. Tissue renewal and repair: Regeneration, healing and fibroses. Robbins' Pathologic Basis of Disease. 7th ed. Philadelphia, PA: WB Saunders Co; 2006
60. Sue Ellen Sarasam, CNM, John P, et al. Management of wound complications from caesarean delivery. Obstetrical and Gynaecological Survey2005; (462-463p)
61. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection. Infect Control Hosp Epidemiology; 1999. (247-278p)

62. L.J.Moultan, surgical site infection after caesarean section;2018 (2)
63. A.Nibamuruke, Reducing postcaesarean wound infection –Emerald insight 2016 (1)
64. HH Jasmin, Incidence and risk factors of surgical site infection – sage journals 2017
65. L Maggio,The association of BMI and wound infection after caesarean delivery ,AJOG article 2016
66. T Kawakita , surgical site infections after caesarean delivery; epidemiology, prevention, risk factors 2017 (3,4)
67. KA Gelaw, Surgical site infections and its associated factors following caesarean section ; June 2017

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **Dr .N.SUKANYA.** Postgraduate in department of obstetrics and gynaecology , Thanjavur medical college & hospital, Thanjavur – 613001 and to use my personal clinical data and result of investigation for the purpose of experimental study and to study the efficacy of the treatment. I also give consent for further investigations

Place :
Date : Signature of participant

KEY TO MASTER CHART

1.	LSCS	-	LOWER SEGMENT CAESAREAN SECTION
2.	BMI	-	BODY MASS INDEX
3.	GHT	-	GESTATIONAL HYPERTENSION
4.	DM	-	DIABETES MELLITUS
5.	PROM	-	PREMATURE RUPTURE OF MEMBRANES
6.	Y	-	YES
7.	N	-	NO
8.	F	-	FOLEY
9.	SYNTO	-	OXYTOCIN INDUCTION
10.	SA	-	SPINAL ANAESTHESIA
11.	GA	-	GENERAL ANAESTHESIA
12.	MV	-	MIDLINE VERTICAL INCISION
13.	PF	-	PFANNENSTEIL INCISION
14.	SC	-	SUBCUTICULAR
15.	MT	-	MATTRESS
16.	AG	-	AMPICILLIN & GENTAMYCIN
17.	XG	-	CEFTRIAXONE & GENTAMYCIN
18.	TG	-	CEFOTAXIM & GENTAMYCIN
19.	S.AUREUS	-	STAPHYLOCOCCUS AUREUS
20.	E.COLI	-	ESCHERICHIA COLI
21.	CONS	-	COAGULASE NEGATIVE STAPHYLOCOCCUS AUREUS
22.	AMPI	-	AMPICILLIN
23.	AMI	-	AMIKACIN
24.	LINE	-	LINEZOLID
25.	PZ	-	PIPERACILLIN TAZOBACTUM
26.	TEICO	-	TEICOPLANIN
27.	G	-	GENTAMYCIN
28.	COTRI	-	COTRIMOXAZOLE
29.	MERO	-	MEROPENAM
30.	DOXY	-	DOXYCYCLINE
31.	LEVO	-	LEVOFLOXACIN
32.	CIPRO	-	CIPROFLOXACIN
33.	R/H	-	RESUTURING + HIGHER ANTIBIOTICS
34.	W/H	-	WOUND CARE+HIGHER ANTIBIOTICS
35.	SSI	-	SURGICAL SITE INFECTION
36.	HAI	-	HOSPITAL ACQUIRED INFECTION

MASTER CHART - A study on post caesarean wound infection

S.No	IP No.	NAME	AGE	AGE GROUP	PARITY	LSCS	DAYS OF STAY	SOCIO ECONOMIC STATUS	BMI	ANAEMIA	GHT	DM	PROM	HANDLED OUTSIDE	NO. OF PV	INDUCTION, IF ANY	INDICATION FOR LSCS	INTRAOPERATIVE				ANTIBIOTICS	POST OP COMPLICATION	ORGANISM	SENSITVITY	RESISTANCE	TREATMENT	REMARKS, IF ANY
				(<20 - A, 20-35 - B, >35 - C)	(PRIMI-1 / MULTI-2)	(ELECTIVE-1/ EMERGENCY-2)												ANAESTHETIA	DURATION (MINS)	INCISION	CLOSURE							
1	503980	AARTHI	22	B	1	2	16	III	25	Y	N	N	N	Y	8	N	OBSTRUCTED LABOR	SA	45	PF	SC	TG	GAPING	E.COLI	AMPI	G,AMI,COTRI	R/H	
2	504868	VASUKI	25	B	1	2	22	III	24	Y	N	N	N	N	6	F,GEL	FAILED INDUCTION	SA	40	PF	SC	AG	GAPING	S.AUREUS	G,LINE,TEICO	AMPI,COTRI	R/H	
3	504955	SANGEETHA	20	B	1	2	13	IV	26	Y	Y	N	N	N	4	N	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	SEROUS	KLEBSIELLA	G,AMI,CIP,COTR	AMPI	W/H	
4	505747	MURUGESHWARI	20	B	1	2	15	IV	26	Y	N	N	Y	N	4	SYNTO	FOETAL DISTRESS	SA	40	PF	SC	AG	SEROUS	NO GROWTH			W/H	
5	505981	RAGINI	29	B	1	2	30	III	27	Y	N	N	N	N	0	N	PRE LSCS/PLACENTA PRAEVIA	GA	80	MV	MT	TG	GAPING	KLEBSIELLA	G,AMI,CIP,	AMPI	R/H	
6	506798	MUVITHA	21	B	1	2	14	IV	27	Y	N	N	N	N	4	N	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	SEROUS	KLEBSIELLA	XONE,G,AMI	AMOX,CORTI,DOXY	W/H	
7	507544	NADHIYA	29	B	2	1	14	IV	24	Y	Y	Y	N	N	2	N	PRE LSCS/CPD	SA	60	PF	SC	TG	INDURATION				W/H	
8	508643	EZHILARASI	22	B	2	2	25	IV	24	Y	N	N	N	N	2	N	PRE LSCS/CPD	SA	75	PF	MT	AG	GAPING	CONS	PZ, LINE	AMI,G,DOXY,TAXIM	R/H	
9	512898	KAUVERY	32	B	2	1	18	IV	28	Y	N	N	N	N	2	N	PRE 2 LSCS	SA	90	PF	MT	TG	SEROUS	NO GROWTH			W/H	HYPOTHYROID
10	513765	NAMATHA	24	B	2	1	16	IV	21	Y	N	N	N	N	2	N	PRE LSCS/PLACENTA ACCRETA	GA	120	MV	MT	XG	GAPING	KLEBSIELLA	LINE, TEICO,G	AMPI,DOXY,COTRI	R/H	
11	514567	SANGEETHA	28	B	1	2	28	IV	22	Y	N	N	Y	N	4	SYNTO	FAILED INDUCTION	SA	30	PF	SC	TG	GAPING	S.AUREUS	PZ,LINE	AMI,G,DOXY,TAXIM	R/H	
12	514908	GOWRI	26	B	1	2	20	III	30	Y	N	Y	N	N	4	N	FOETAL ALARM SIGNAL	SA	45	PF	SC	TG	INDURATION				W/H	
13	515786	NANDHINI	26	B	1	2	14	IV	24	Y	N	N	Y	Y	8	N	OBSTRUCTED LABOR	SA	60	PF	SC	XG	GAPING	S.AUREUS	LINE,G,TEICO	AMI,DOXY,TAXIM	R/H	
14	516798	SRIPRIYA	26	B	1	2	15	IV	24	N	N	N	Y	N	4	SYNTO	FAILED INDUCTION	SA	40	PF	SC	TG	INDURATION				W/H	
15	517908	MUNIYAMMAL	20	B	2	1	12	III	26	Y	Y	N	N	N	1	N	PRE LSCS/OBLIQUE LIE	SA	45	PF	SC	TG	SEROUS	NO GROWTH			W/H	HYPOTHYROID
16	518868	GIRIJA	26	B	1	2	14	IV	36	Y	Y	N	N	N	8	F,GEL,SYNTO	FAILED INDUCTION	SA	45	PF	MT	TG	GAPING	KLEBSIELLA	LINE	AMPI,G,COTRI	R/H	HYPOTHYROID
17	519543	KAMATCHI	24	B	2	1	23	III	23	Y	Y	N	N	N	2	N	AP ECLAMPSIA/UNFAVOURABLE	SA	40	PF	SC	TG	GAPING	E.COLI	CEPHALEXIN,C	AMPI,G,AMI	R/H	
18	521376	KASTHURI	39	C	1	2	16	II	25	Y	N	Y	N	N	2	N	BREECH	SA	40	PF	SC	AG	GAPING	CONS	PZ,LINE	AMPI,G,DOXY,AMI	R/H	
19	522543	SANKARI	24	B	1	2	12	IV	26	Y	N	N	N	N	6	F, GEL	FOETAL DISTRESS	SA	45	PF	SC	TG	INDURATION				W/H	
20	522895	SARASWATHY	25	B	1	2	25	III	35	N	Y	N	N	N	3	N	NON REACTIVE NST	SA	40	PF	SC	TG	INDURATION				W/H	
21	523643	SANGEETHA	26	B	2	2	23	IV	28	Y	N	N	N	N	8	F, SYNTO	MSAF/FOETAL DISTRESS	SA	40	PF	SC	XG	GAPING	S.AUREUS	TAXIM, G, V,TEI	AMPI,G,COTRI	R/H	
22	524321	VIMALADEVI	26	B	1	2	16	IV	27	Y	Y	N	N	N	2	N	ABNORMAL DOPPLER STUDY	SA	30	PF	SC	TG	GAPING	E.COLI	TAXIM, XONE, A	AMPI, G , COTRI	R/H	
23	526798	SENTHAMILSELVI	26	B	1	2	12	IV	23	Y	N	N	N	N	3	GEL	MSAF/FOETAL DISTRESS	SA	45	PF	SC	AG	SEROUS	NO GROWTH			W/H	
24	528571	PASAMALAR	23	B	1	2	16	IV	28	N	N	N	N	N	6	GEL,SYNTO	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
25	529768	THENMOZHI	25	B	2	2	10	III	25	N	N	N	N	N	1	N	PRE LSCS/CPD	SA	60	PF	SC	AG	INDURATION				W/H	

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				(<20 - A, 20-35 - B, >35 - C)	(PRIMI-1 / MULTI-2)	(ELECTIVE-1/ EMERGENCY-2)												ANAESTHETIA	DURATION (MINS)	INCISION	CLOSURE							
26	530049	SUGUNA	30	B	2	2	13	II	27	Y	N	N	N	N	2	N	PRE LSCS/CPD	SA	60	PF	MT	TG	INDURATION				W/H	
27	530344	MOVILA	27	B	1	2	17	II	28	Y	Y	N	Y	Y	6	N	PROM>16HRS/FAILURE TO PROGI	SA	40	PF	SC	XG	GAPING	CITROBACTER	CEPHALEXIN, N	AMI,AMPI,PZ,LINE	R/H	
28	530832	DURGADEVI	21	B	1	2	14	III	24	Y	N	N	Y	N	1	N	CORD PROLAPSE	SA	40	PF	SC	AG	SEROUS	NO GROWTH			W/H	
29	532365	AMALA	22	B	1	2	20	IV	27	N	Y	N	N	N	2	N	OLIGO/VARIABLE FHR	SA	30	PF	SC	AG	INDURATION				W/H	
30	534566	TAMILARASI	21	B	2	1	18	IV	28	N	N	N	N	N	3	N	PRE LSCS/CPD	SA	80	PF	MT	TG	INDURATION				W/H	
31	536789	MAHALAKSHMI	27	B	2	2	15	IV	30	Y	Y	N	N	N	2	N	PRE 2 LSCS	SA	90	PF	MT	TG	INDURATION				W/H	
32	539506	KALAIMAGAL	26	B	1	1	10	III	25	Y	N	N	N	N	1	N	MAJOR CPD	SA	40	PF	SC	AG	SEROUS	NO GROWTH			W/H	
33	539987	KARTHIGA	24	B	2	2	14	IV	28	Y	N	Y	N	N	1	N	FOETAL DISTRESS	SA	50	PF	SC	TG	GAPING	S.AUREUS	LINE,TEICO	AMPI,G,DOXY,AMI	R/H	
34	541287	PRAVEENA	21	B	1	2	12	III	26	N	N	N	N	N	6	F,SYNTO	FAILED INDUCTION	SA	40	PF	SC	AG	INDURATION				W/H	
35	541908	JAYAPREETHI	27	B	1	2	10	IV	25	Y	N	N	Y	N	4	SYNTO	FOETAL DISTRESS	SA	40	PF	SC	TG	SEROUS	E.COLI	AMI,G,CEPHALI	AMPI,DOXY,COTRI	W/H	
36	542209	SHARMILABANU	22	B	1	2	16	IV	26	Y	N	N	N	N	3	N	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
37	543927	KANIMOZHI	20	B	1	2	36	III	26	Y	Y	N	N	N	0	N	PLACENTA PRAEVIA	SA	60	PF	SC	TG	GAPING	PSEUDOMONA	CEPHALEXIN,N	AMI,G,DOXY,PZ	R/H	
38	545866	TAMILARASI	21	B	2	1	18	IV	32	Y	N	N	N	N	2	N	PRE LSCS /PLACENTA PRAEVIA	SA	80	PF	MT	TG	SEROUS	PROTEUS	AMI ,PZ,MERO	G,CEPHALEXIN,CIP	W/H	
39	545887	KABILADEVI	20	B	1	2	15	III	24	N	Y	N	N	N	6	F,GEL,SYNTO	FAILED INDUCTION	SA	40	PF	SC	TG	INDURATION				W/H	
40	545897	AMALA	22	B	1	2	16	IV	27	Y	Y	N	N	N	1	N	ABRUPTION GRADE 2	SA	40	PF	SC	TG	INDURATION				W/H	
41	547890	MAHALAKSHMI	27	B	2	2	15	III	30	N	Y	N	N	N	1	N	PRE LSCS/TERM GHT	SA	45	PF	SC	AG	INDURATION				W/H	
42	548516	SUDARMANI	30	B	1	2	22	IV	33	Y	Y	N	N	N	3	N	NON REACTIVE NST	SA	40	PF	MT	AG	GAPING	E.COLI	PZ	AMPI, G,COTRI, DO?	R/H	HYPOTHYROID
43	549768	ANJUGAM	26	B	1	1	14	III	28	N	N	N	N	N	1	N	BREECH	SA	40	PF	SC	AG	INDURATION				W/H	HYPOTHYROID
44	549976	JEYARANI	19	A	1	2	23	IV	24	Y	Y	N	N	N	1	N	AP ECLAMPSIA/UNFAVOURABLE	GA	60	PF	SC	TG	INDURATION				W/H	
45	550987	KALPANA	24	B	2	2	13	IV	27	Y	N	N	N	N	1	N	PRE 2 LSCS	SA	60	PF	MT	TG	INDURATION				W/H	
46	551276	ABILA	22	B	1	2	16	III	28	Y	Y	N	N	N	5	SYNTO	FAILURE TO PROGRESS	SA	40	PF	SC	AG	SEROUS	CONS	LINE,DOXY	G,COTRI,CEPHALE?	W/H	
47	552198	CHELLAMAL	27	B	1	2	18	IV	25	Y	N	N	N	N	3	N	BROW PRESENTATION	SA	40	PF	SC	AG	INDURATION				W/H	
48	553254	ARUNADEVI	21	B	1	2	14	III	24	Y	Y	N	N	N	6	F,GEL,SYNTO	FAILED INDUCTION	SA	45	PF	SC	AG	SEROUS	K.LEBSIELLA	COTRI,DOXY,LI	AMPI,G,TAXIM	W/H	
49	553748	RAMESHWARI	20	B	1	2	30	III	29	Y	N	Y	N	N	5	F,SYNTO	FAILED INDUCTION	SA	45	PF	SC	TG	GAPING	S.AUREUS	LINE	AMPI,TAXIM,G	R/H	FEVER
50	554736	SUMITHRA	29	B	2	1	30	III	28	Y	N	N	N	N	1	N	PRE 2 LSCS	SA	60	PF	MT	XG	GAPING	K.LEBSIELLA	LINE	AMOX,CORTI,DOXY	R/H	

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				(<20 - A, 20-35 - B, >35 - C)	(PRIMI-1 / MULTI-2)	(ELECTIVE-1/ EMERGENCY-2)												ANAESTHETIA	DURATION (MINS)	INCISION	CLOSURE							
51	554879	KAYALSRI	26	B	1	2	12	IV	23	Y	N	N	N	N	2	N	CORD PROLAPSE	SA	40	PF	SC	AG	INDURATION				W/H	
52	556533	SURYAKALA	28	B	2	2	18	IV	25	Y	N	N	N	N	1	N	PRE LSCS/CPD	SA	60	PF	SC	AG	SEROUS	E.COLI	AMI,G,CEPHALI	AMPI	W/H	
53	558793	GUNASUNDARI	25	B	1	2	35	IV	29	Y	N	N	N	N	2	F	OLIGO/NON REACTIVE NST	SA	40	PF	SC	AG	GAPING	S.AUREUS	LINE, TEICO,G	AMI,AMPI,TAXIM	R/H	
54	558825	UMA	24	B	1	2	17	III	50	Y	Y	N	N	N	2	N	AP ECLAMPSIA/UNFAVOURABLE	SA	80	PF	MT	XG	GAPING	S.AUREUS	PZ,LINE	AMI,TAXIM,G	R/H	
55	558967	PERIYANAYAKI	26	B	2	2	15	III	31	Y	Y	N	N	N	1	N	PRE LSCS/TERM GHT	SA	45	PF	SC	AG	INDURATION				W/H	
56	560078	THEIVASELVI	24	B	1	2	16	IV	28	Y	N	N	N	N	3	N	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
57	560978	KALAESWARI	32	B	1	2	15	IV	24	Y	N	N	Y	N	2	N	VARIABLE FHR/OLIGO	SA	35	PF	SC	TG	GAPING	NO GROWTH			R/H	
58	561276	SHAKILA	33	B	1	2	22	IV	35	Y	Y	N	N	N	3	N	NON REACTIVE NST	SA	40	PF	MT	AG	GAPING	E.COLI	PZ	AMPI, G,COTRI, DOX	R/H	HYPOTHYROID
59	561345	DEVIGA	22	B	1	2	12	III	28	Y	N	N	N	N	6	F,SYNTO	FAILED INDUCTION	SA	40	PF	SC	AG	INDURATION				W/H	
60	562765	UMA	28	B	2	1	30	III	26	Y	N	N	N	N	1	N	PRE 2 LSCS	SA	60	PF	MT	XG	GAPING	KLEBSIELLA	LINE	AMOX,CORTI,DOXY	R/H	
61	564576	SANGEETHA	21	B	1	2	14	IV	22	Y	N	N	N	N	3	N	FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
62	566890	SUGANTHI	23	B	1	2	17	III	45	N	Y	N	N	N	2	N	AP ECLAMPSIA/UNFAVOURABLE	SA	80	PF	MT	XG	GAPING	S.AUREUS	PZ,LINE	AMI,TAXIM,G	R/H	
63	568991	MUVITHRA	21	B	2	1	18	IV	23	N	N	N	N	N	2	N	PRE LSCS/CPD	SA	45	PF	SC	TG	PUS	S.AUREUS	LEVO,TEICO	AMI,G,DOXY,TAXIM	W/H	
64	569876	ANANDHI	25	B	1	2	18	IV	27	Y	N	N	N	N	3	N	BROW PRESENTATION	SA	40	PF	SC	AG	INDURATION				W/H	
65	570431	RATHIKARANI	29	B	1	2	14	IV	29	Y	N	N	Y	Y	6	N	OBSTRUCTED LABOR	SA	40	PF	SC	TG	GAPING	CITROBACTER	AMI,G,MERO	AMPI,COTRI,CIPRO	R/H	
66	571432	BHUVANESWARI	23	B	1	1	14	III	27	N	N	N	N	N	1	N	BREECH	SA	40	PF	SC	AG	INDURATION				W/H	HYPOTHYROID
67	572343	THENMOZHI	37	C	1	2	23	IV	28	Y	Y	N	N	N	6	F,GEL,SYNTO	FAILED INDUCTION	SA	40	PF	SC	AG	GAPING	E.COLI	CIPRO	AMPI,G,COTRI, DOX	R/H	
68	573123	CHITHARA	30	B	1	2	15	IV	23	N	N	N	Y	N	2	N	VARIABLE FHR/OLIGO	SA	35	PF	SC	TG	GAPING	NO GROWTH			R/H	
69	574532	MAHALAKSHMI	25	B	2	2	15	III	32	N	Y	N	N	N	1	N	PRE LSCS/TERM GHT	SA	45	PF	SC	AG	INDURATION				W/H	
70	575876	KAVITHA	30	B	1	2	22	IV	31	Y	Y	N	N	N	3	N	NON REACTIVE NST	SA	40	PF	MT	AG	GAPING	E.COLI	PZ	AMPI, G,COTRI, DOX	R/H	HYPOTHYROID
71	577865	KARTHIKA	27	B	1	2	12	IV	21	N	N	N	N	N	2	N	CORD PROLAPSE	SA	40	PF	SC	AG	INDURATION				W/H	
72	577995	VENNILA	35	B	1	2	23	IV	27	N	Y	N	N	N	6	F,GEL,SYNTO	FAILED INDUCTION	SA	40	PF	SC	AG	GAPING	E.COLI	CIPRO	AMPI,G,COTRI, DOX	R/H	
73	579887	GUNAVATHI	25	B	1	2	35	IV	32	Y	N	N	N	N	2	F	OLIGO/NON REACTIVE NST	SA	40	PF	SC	AG	GAPING	S.AUREUS	LINE, TYCO,G	AMI,AMPI,TAXIM	R/H	
74	581243	KOWSALYA	20	B	1	2	14	IV	26	Y	N	N	N	N	3	N	FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
75	582345	SENBAGANAYAKI	22	B	1	2	16	IV	28	N	Y	N	N	N	1	N	ABRUPTION GRADE 2	SA	40	PF	SC	TG	INDURATION				W/H	

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				(<20 - A, 20-35 - B, >35 - C)	(PRIMI-1 / MULTI-2)	(ELECTIVE-1/ EMERGENCY-2)												ANAESTHETIA	DURATION (MINS)	INCISION	CLOSURE							
76	583465	NITHYAVANI	26	B	2	1	30	III	27	Y	N	N	N	N	1	N	PRE 2 LSCS	SA	60	PF	MT	XG	GAPING	K.LEBSIELLA	LINE	AMOX,CORTI,DOXY	R/H	
77	584465	SYEDHA	28	B	1	2	14	IV	26	Y	N	N	Y	Y	6	N	OBSTRUCTED LABOR	SA	40	PF	SC	TG	GAPING	CITROBACTER	AMI,G,MERO	AMPI,COTRI,CIPRO	R/H	
78	585645	SUDHA	34	B	1	2	22	IV	30	N	Y	N	N	N	3	N	NON REACTIVE NST	SA	40	PF	MT	AG	GAPING	E.COLI	PZ	AMPI, G,COTRI, DOX	R/H	HYPOTHYROID
79	586543	SATHYABAMA	33	B	1	2	14	IV	29	Y	N	N	Y	Y	6	N	OBSTRUCTED LABOR	SA	40	PF	SC	TG	GAPING	CITROBACTER	AMI,G,MERO	AMPI,COTRI,CIPRO	R/H	
80	587865	LAVANYA	24	B	2	1	18	IV	26	N	N	N	N	N	2	N	PRE LSCS/CPD	SA	45	PF	SC	TG	PUS	S.AUREUS	LEVO,TEICO	AMI,G,DOXY,TAXIM	W/H	
81	588098	HARSHITHBANU	23	B	1	2	30	III	28	Y	N	Y	N	N	5	F,SYNTO	FAILED INDUCTION	SA	45	PF	SC	TG	GAPING	S.AUREUS	LINE	AMPI,TAXIM,G	R/H	FEVER
82	588262	PRIYA	22	B	2	2	16	III	27	Y	Y	Y	N	N	1	N	PRE LSCS/ABRUPTION GRADE 2	SA	60	PF	MT	AG	GAPING	S.AUREUS	LINE	AMOX,AMPI,COTRI,	W/H	
83	589765	SHANMATHIPRIYA	26	B	1	2	14	IV	24	N	Y	N	N	N	2	N	FAILURE TO DESCENT	SA	45	PF	SC	AG	INDURATION				W/H	
84	590876	TAMIZHALAGI	24	B	1	2	14	IV	29	N	Y	N	Y	N	4	SYNTO	PROM>16HRS/FAILURE TO PROG	SA	40	PF	SC	AG	SEROUS	NO GROWTH			W/H	
85	591254	KAVITHA	27	B	1	2	16	IV	26	Y	N	N	N	N	6	F,SYNTO	FAILED INDUCTION	SA	45	PF	SC	AG	GAPING	E.COLI	CIPRO	AMPI, G,CORTI	R/H	
86	591984	MAHESWARI	30	B	1	2	15	IV	23	N	N	N	Y	N	2	N	VARIABLE FHR/OLIGO	SA	35	PF	SC	TG	GAPING	NO GROWTH			R/H	
87	592678	PRAVEENA	21	B	1	2	14	IV	24	Y	N	N	N	N	3	N	FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
88	593421	JAYAPRABA	24	B	2	2	12	III	28	N	Y	N	N	N	1	N	PRE 2 LSCS	SA	60	PF	SC	TG	INDURATION				W/H	HYPOTHYROID
89	594876	SHANTHIPRIYA	27	B	2	2	23	IV	28	Y	N	N	N	N	1	N	PRE LSCS/CPD	SA	60	PF	SC	AG	GAPING	NO GROWTH			R/H	
90	595087	SAROJADEVI	31	B	1	2	14	IV	26	Y	N	N	Y	Y	6	N	OBSTRUCTED LABOR	SA	40	PF	SC	TG	GAPING	CITROBACTER	AMI,G,MERO	AMPI,COTRI,CIPRO	R/H	
91	596791	AKILA	23	B	1	2	16	III	27	N	Y	N	N	N	2	N	MSAF/FOETAL DISTRESS	SA	40	PF	SC	AG	INDURATION				W/H	
92	598741	SHANMUGAPRIYA	22	B	2	1	18	IV	23	N	N	N	N	N	2	N	PRE LSCS/CPD	SA	45	PF	SC	TG	PUS	S.AUREUS	LEVO,TEICO	AMI,G,DOXY,TAXIM	W/H	