# "CASE CONTROL STUDY TO ASSESS INDEPENDENT RISK FACTORS INFLUENCING SURGICAL SITE INFECTION AFTER CESAREAN SECTION"

**By**

(REG.NO. BJ0112005)

# Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY

in

OBSTETRICS AND GYNAECOLOGY

**DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,**
**JAWAHARLAL NEHRU MEDICAL COLLEGE,**
**BELGAUM, KARNATAKA**

**MAY - 2015**

**KLE UNIVERSITY, BELGAUM, KARNATAKA**

## ENDORSEMENT BY THE HOD/PRINCIPAL/ HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"CASE CONTROL STUDY TO ASSESS INDEPENDENT RISK FACTORS INFLUENCING SURGICAL SITE INFECTION AFTER CESAREAN SECTION"** is a bonafide research work done by **REG.NO. BJ0112005.**

**Dr. M. K. Swamy** **MD**
Professor and Head,
Department of Obstetrics
and Gynaecology,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

**Dr.Mahanshetti** **MD Paed**
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

## *LIST OF ABBREVIATIONS USED*

| | | |
|---|---|---|
| SSI | - | Surgical site infection |
| NNIS | - | National Nosocomial Infection Surveillance |
| CDC | - | Centres for Disease Control and Prevention |
| LSCS | - | Lower Segment Cesarean section |
| C-Section | - | Caesarean section |
| SIRS | - | Systemic inflammatory response syndrome |
| NINSS | - | National Nosocomial Infection Surveillance System |
| TNF | - | Tumor necrosis factor |
| IL | - | Interleulins |
| PDGF | - | Platelet derived growth factor |
| IGF-1 | - | Insulin like growth factor |
| FGF | - | Fibroblast like growth factor |
| TGF-alpha | - | Transforming growth factor alpha |
| TGF-beta | - | Transforming growth factor beta |
| NFHS | - | National Family Health Survey |
| MRSA | - | Methicillin resistant Staphylococcus aureus |

| | | |
|---|---|---|
| VISA | - | Vancomycin intermediate staohylococcus aureus |
| HIV | - | Human Immunodeficiency Virus |
| GDM | - | Gestational Diabetes Mellitus |
| HELLP | - | Hemolysis Elevated Liver enzy |
| APH | - | Antepartum haemorrhage |
| DIC | - | Disseminated intravascular Coagulation |
| ANC | - | Antenatal care |
| df | - | Degree of freedom |

# ABSTRACT

## "CASE CONTROL STUDY TO ASSESS INDEPENDENT RISK FACTORS INFLUENCING SURGICAL SITE INFECTION AFTER CESAREAN SECTION"

**Objective:** The objectives of the present study were to determine independent risk factors (Patient, Obstetrics, Operation related variables) for surgical site infection after caesarean section.

**Study design:** A case control study.

**Place:** A teaching hospital: J N Medical College, Belgaum.

**Sample size:** 1050 (350 cases, 700 controls).

**Results:** A total of 1050 womens were devided into cases (350) and controls (700). The outcome for analysis was based on the independent risk factor for surgical site infection. In this study the independent risk factors found to be significant were GDM(oddsratio5.4, p value of 0.0001), HIV (p value 0.001,anemia (odds ratioof 10.66, p value of0.0001), unregistered antenatal cases (odds ratio of 4.7, p value of <0.0001), primary caesarean section (odds ratio of 5, p value 0.0001), PROM( odds ratio 7.4, p value 0.0001), chorioamnionitis ( p value 0.04), preeclampsia/ eclampsia (odds ratioof 7.90, p value of 0.0001), induction of labour (odds ratio8.46, p value 0.0001), meconium stained liquor (odds ratio of 5.1, p value 0.0001), emergency caesarean section ( odds ratio of 9.6, p value of <0.0001), conservative surgery for PPH( odds ratio of 14.2, p value of 0.01), peripartum hysterectomy ( odds ratio of 30.54, p value of 0.01).

The factors which were not significantly associated with SSI are age, gestational age, gravida, APH, prophylactic antibiotics.

**Conclusion and interpretation:**

The present study showed that following were independent risk factors for SSI, gestational diabetes, HIV, haemoglobin between 9-11gm%, Unregistered antenatal cases, Primary cesarean section, pereclampsia/ eclampsia, induction of labour, PROM, chorioamnionitis, meconium stained liquor, emergency cesarean section, conservative surgery for PPH, were found to be significant.

**Keywords:**

Cesarean section, Surgical site infection, gestational diabetes mellitus, preeclampsia/ eclampsia.

# *CONTENTS*


| SL. NO. | TOPIC | PAGE NO. |
|---|---|---|
| 1 | INTRODUCTION | 1 |
| 2 | OBJECTIVES | 4 |
| 3 | REVIEW OF LITERATURE | 5 |
| 4 | METHODOLOGY | 30 |
| 5 | RESULTS | 35 |
| 6 | DISCUSSION | 56 |
| 7 | CONCLUSION | 64 |
| 8 | SUMMARY | 65 |
| 9 | BIBLIOGRAPHY | 68 |
| 10 | ANNEXURES | |
| | ANNEXURE I – ETHICAL CLEARANCE | 74 |
| | ANNEXURE II – CONSENT FORM | 75 |
| | ANNEXURE III – DATA COLLECTION INSTRUMENT | 88 |
| | ANNEXURE IV – MASTER CHART | 92 |

# LIST OF TABLES


| TABLE. NO. | DESCRIPTION | PAGE NO. |
|---|---|---|
| 1 | Demographic characteristics age | 36 |
| 2 | Gestational age | 37 |
| 3 | Obstetric history, current pregnancy | 38 |
| 4 | Gestational Diabetes Mellitus and SSI | 39 |
| 5 | Human immunodeficiency Virus and SSI | 40 |
| 6 | Anemia and SSI | 41 |
| 7 | Patient related variables: Antenatal Rare and SSI | 42 |
| 8 | Previous Cesarean Section | 43 |
| 9 | Premature rupture of membranes | 44 |
| 10 | Chorioamnionitis | 45 |
| 11 | Antepartum haemorrhage | 46 |
| 12 | Preeclampsia/ Eclampsia | 47 |
| 13 | Hemolysis, elevated liver enzymes, Low platelet count | 48 |
| 14 | Disseminated intravascular coagulation | 49 |
| 15 | Induction of Labour | 50 |
| 16 | Meconium stained liquor | 51 |
| 17 | Operation related variables: Nature of LSCS | 52 |
| 18 | Additional surgical procedures | 53 |
| 19 | Prophylactic antibiotic therapy | 54 |
| 20 | Suture material used for skin closure | 55 |

# LIST OF GRAPHS


| GRAPH NO. | DESCRIPTION | PAGE NO. |
|---|---|---|
| 1 | Demographic characteristics age | 36 |
| 2 | Gestational age | 37 |
| 3 | Obstetric history, current pregnancy | 38 |
| 4 | Patient related variables:Gestational Diabete Mellitus and SSI | 39 |
| 5 | Human immunodeficiency Virus and SSI | 40 |
| 6 | Anemia and SSI | 41 |
| 7 | Obstetric related variables: Antenatal Rare and SSI | 42 |
| 8 | Previous Cesarean Section | 43 |
| 9 | Premature rupture of membranes | 44 |
| 10 | Chorioamnionitis | 45 |
| 11 | Antepartum haemorrhage | 46 |
| 12 | Preeclampsia/ Eclampsia | 47 |
| 13 | Hemolysis, elevated liver enzymes, Low platelet count | 48 |
| 14 | Disseminated intravascular coagulation | 49 |
| 15 | Induction of Labour | 50 |
| 16 | Meconium stained liquor | 51 |
| 17 | Operation related variables: Nature of LSCS | 52 |
| 18 | Additional surgical procedures | 53 |
| 19 | Prophylactic antibiotic therapy | 54 |
| 20 | Suture material used for skin closure | 55 |

# LIST OF FIGURES


| PHOTOGRAPH NO. | DESCRIPTION | PAGE NO. |
|---|---|---|
| 1 | Schematic representation of the anatomical classification of surgical site infection | 9 |
| 2 | The consequences of inflammation | 16 |

# Chapter 1

# Introduction

# INTRODUCTION

Cesarean section is a common surgical procedure performed worldwide.[1] The proportion of births delivered by cesarean section has risen substantially over the past 30 years from 9% in 1980 to 25% in 2009/10[1]. Cesarean sections are performed as emergency or elective procedures depending upon the indications. Elective operation is defined as a cesarean section that was planned at least 24 hours before the intervention.[2]

Surgical site infection (SSI) is infection occurring in surgical wound within 30 days of operation. It can be superficial, deep incisional or organ/ space related. Host susceptibility, degree of microbial contamination of a surgical site and duration of operation are reliable predictors of surgical site infection risk.[3]

Surgical site infection accounts for 20% of all health care-associated infections. The rates of SSI after cesarean section reported in the literature range from 3% to 15%, depending on the surveillance methods used to indentify infection, the patient population, and the use of antibiotic prophylaxis.[4] Given the short length of postoperative stay in hospital, typically 2–4 days, the overall rate of infection following cesarean section is likely to be significantly higher than these estimates.[5,6]

Although most cesarean section wound infections are superficial, this represents a substantial burden to the health system, given the high number of women undergoing this type of surgery. However, a proportion of infections are more severe infections of the deeper tissue or reproductive organs necessitating

extended hospital stays or readmission to hospital,[7,8] further adding to the economic burden of these infections. On very rare occasions the development of an infection after cesarean section can lead to devastating outcomes.

Recovery after cesarean section can be more demanding for women who develop a postoperative SSI. Surgical site infection (SSI) after cesarean section increases maternal morbidity and costs.[1] Indeed, some of these infections can be very serious, and lead to severe health problems or even death.[9] SSI results in failure of wound healing with subsequent increased treatment costs, a greater likelihood of admission to the intensive care unit, prolonged hospital stay and higher postoperative mortality.[9]

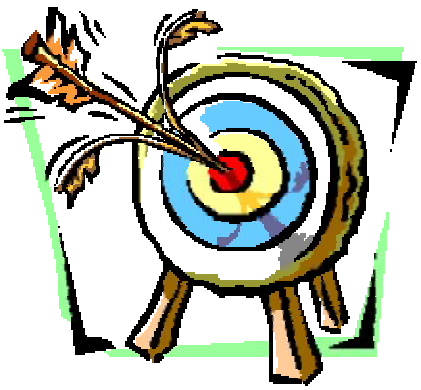
Despite numerous investigations, there is disagreement about risk factors of surgical site infection after cesarean delivery. Many factors affect infection rates in different settings. Confounding variables were not sufficiently controlled in many of the earlier studies.[10] A better understanding of predictors might improve infection control by reducing clinical effects of post cesarean section infection.

Expanding our knowledge of risk factors associated with post cesarean section surgical site infection is essential for developing targeted prevention strategies to reduce the risk of surgical site infection. Independent risk factors for surgical site infection after cesarean section have not been well documented, despite the fact that it is the most common performed surgical procedure[11] and the reported rate of surgical site infection is relatively high. So therefore there is a need to determine clinically relevant independent risk factors for surgical site

infection after lower segment caesarean section. The goal of this study was to find out the frequency of surgical site infection in elective and emergency cesarean sections. By assessing independent risk factors(Patient, Operation, Obstetric related variables)

# Chapter 2

# Objectives



# OBJECTIVES

The objectives of the present study is to determine independent risk factors (Patient, Obstetrics, Operation related variables) for surgical site infection after caesarean section.

# Chapter 3

# Review of Literature

# REVIEW OF LITERATURE

## CESAREAN DELIVERY

The word cesarean is derived from the Latin word Caesar, which means "to cut". Cesarean section is the delivery of an neonate, alive or dead, through an abdominal incision and from an intact uterus, after the period of viability.

Cesarean section symbolizes the most important operative intervention in all of obstetrics. It has undergone tremendous evolution from the time of its conception to make it both, safer and easier for the mother and fetus, and the obstetrician respectively.

Over the last century, the cesarean section from a post mortem procedure became largely a procedure that results in a living mother and a neonate most of the times. From classical cesarean to classical lower segment cesarean section to the present day concept of lower segment transverse incision cesarean section, the procedure is now safe and is a transformation as significant to women and families involved as to the medical profession.

Surgical Site Infections continues to be a major source of morbidity following cesarean delivery. The NNIS report for 1986-1996 described an SSI rate of 2.6% for all operations at the reporting hospitals. It seems likely that overall SSI rates are likely to be greater than reported. All surgical wounds are contaminated by bacteria, but only a minority actually demonstrate clinical infection. The SSI are the biological summation of several factors: the inoculum of bacteria introduced into the wound during the procedure, the unique virulence

of contaminants, the microenvironment of each wound, and the integrity of the patients host defense mechanisms. Although an SSI rate of zero may not be achievable, continued progress in understanding the biology of infection at the surgical site and consistent applications of proven methods of prevention will allow to further reduce the frequency, cost, and morbidity associated with SSI.[12]

**A. Historical note**

The ancient Egyptians were the first civilization to have trained clinicians to treat physical aliments. Medical papyri, such as the Edwin Smith papyrus (circa 1600 BC) and the Ebers papyrus (circa 1534 BC), provided detailed information of management of disease, including wound management with the application of various potions and grease to assist healing.[13]

Galen (Roman gladiatorial surgeon, 130-200 AD) was first to recognize that pus from wounds inflicted by the gladiators heralded healing. The link between pus formation and healing was emphasized so strongly that foreign material was introduced into wounds to promote pus formation-suppuration. The concept of wound healing remained a mystery, as highlighted by the famous saying by Ambroise Paré (French military surgeon, 1510-1590), "I dressed the wound. God healed it."[14]

Koch (Professor of Hygiene and Microbiology, Berlin, 1843-1910) first recognized the cause of infective foci as secondary to microbial growth in his 19$^{th}$ century postulates. Semmelweis (Austrian obstetrician, 1818-1865) demonstrated a 5-fold reduction in puerperal sepsis by hand washing between performing postmortem examinations and entering the delivery room. Joseph Lister

(Professor of Surgery, London, 1827-1912) and Louis Pasteur (French bacteriologist, 1822-1895) revolutionized the entire concept of wound infection.[15]

Antisepsis derived from the Greek ‘‘against putrefaction’’ and its use in modern medicine is most frequently linked to the work of Lister. It refers to the use of solutions for disinfection.[16]

In the late eighteenth and early nineteenth centuries operative outcomes were poor. Wounds were allowed to heal by secondary intention and morbidity and mortality were associated largely with surgical-site infections (SSIs). The first use of an antiseptic skin agent in surgery is credited to the English surgeon Joseph Lister (1827- 1912).[17]

Before the mid-19th century, surgical patients commonly developed postoperative “irritative fever,” followed by purulent drainage from their incisions, overwhelming sepsis, and often death.[18]

Prior to the mid-19th century, limb amputation was associated with an alarming 50% postoperative mortality from sepsis. Following Louis Pasteur’s discovery that tissue decay was caused by microscopic organisms, Lister theorized that the spread of these microbes through surgical wounds was responsible for death in the postoperative period.[19]

Lister placed carbolic acid into open fractures to sterilize the wound and to prevent sepsis and hence the need for amputation.[20] Listers work radically changed surgery from an activity associated with infection and death to a discipline that could eliminate suffering and prolong life.

As early as 1882, Labarraque, a French pharmacist, demonstrated that solutions containing chlorides of lime or soda could eradicate foul odour associated with human corpses and that such solutions could be used as disinfectants and antiseptics.[21] As late as the 19th century, aseptic surgery was not routine practice. Sterilization of instruments began in the 1880s as did the wearing of gowns, masks, and gloves. Halsted (Professor of Surgery, Johns Hopkins University, United States, 1852-1922) introduced rubber gloves to his scrub nurse (and future wife) because she was developing skin irritation from the chemicals used to disinfect instruments. The routine use of gloves was introduced by Halsted's student J. Bloodgood.[21]

Alexander Fleming (microbiologist, London, 1881-1955) performed many of his bacteriological studies during World War I and is credited with the discovery of penicillin.[15]

Penicillin first was used clinically in 1940 by Howard Florey. With the use of antibiotics, a new era in the management of wound infections commenced. Unfortunately, eradication of the infective plague affecting surgical wounds has not ended because of the insurgence of antibiotic-resistant bacterial strains and the nature of more adventurous surgical intervention in immunocompromised patients and in implant surgery.[15]

Despite of all these, in the present era SSI remains one of the major risk factor for maternal morbidity. Factors like anemia, diabetes, hypertension, obesity.

**B. Definition of Surgical Site Infections[3]**

Surgical Site Infection is a difficult term to define accurately because it has a wide spectrum of possible clinical features. Surgical-site infection (SSI) is defined by the Centres for Disease Control and Prevention (CDC) as a proliferation of pathogenic micro-organisms which develops in an incision site either within the skin and subcutaneous fat (superficial), musculo-fascial layers (deep), or in an organ or cavity, if opened during surgery.

Since the skin is normally colonized by bacterial flora, an SSI cannot be diagnosed by the microbiological evidence alone but in conjunction with clinical signs which include: redness, heat, pain and swelling, separation of the suture line (dehiscence), or the presence of an abscess in the deeper tissues. Patients may mount a systemic inflammatory response syndrome (SIRS) with an elevated white cell count, body temperature $<35^0$C or $>38^0$C, pulse rate >100/min, respiratory rate >20/min, or in severe cases develop signs of sepsis, with an attendant increase in morbidity and mortality.

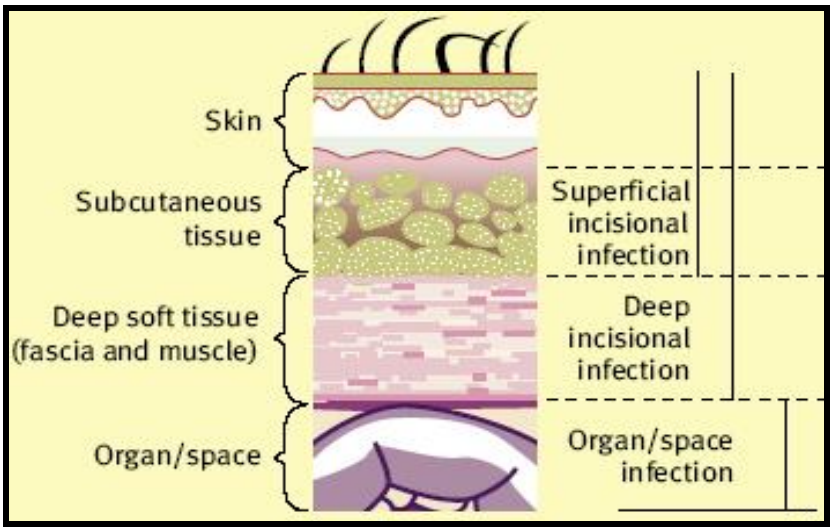



**Figure 1. Schematic representation of the anatomical classification of surgical site infections**

**C. Classification of surgical site infection**[22]

I. Superficial Incisional SSI

Infection involves only skin and subcutaneous tissues of incision.

- Occurs within 30 days after the operation;
- Involves only the skin or subcutaneous tissue; and
- At least 1 of the following:
    - Purulent drainage (culture documentation not required)
    - Organisms isolated from fluid/tissue of superficial incision
    - At least 1 sign of inflammation (eg, pain or tenderness, induration, erythema, local warmth of the wound)
    - Wound is deliberately opened by the surgeon
    - Surgeon or attending physician declares the wound infected.

A wound is not considered a superficial site infection if a stitch abscess is present, the infection is at an episiotomy or circumcision site or a burn wound, or the SSI extends into the fascia or muscle.

II, Deep Incisional SSI

Infection involves deep tissues such as fascial and muscle layers. This also includes infection involving both superficial and deep incision sites.

- Occurs within 30 days of operation or within 1 year if an implant is present;
- Involves deep soft tissues (fascia and/or muscle) of the incision; and
- At least 1 of the following:
    - Purulent drainage from the deep incision but without organ/space involvement

- Fascial dehiscence or fascia is deliberately separated by the surgeon due to signs of inflammation
- Deep abscess is identified by direct examination or during reoperation, by histopathology, or by radiologic examination
- Surgeon or attending physician declares that deep incisional infection is present.

III. Organ/Space SSI

Infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during operation.

- Occurs within 30 days of operation or within 1 year if an implant is present;
- Involves anatomic structures not opened or manipulated during the operation; and
- At least 1 of the following:
  - Purulent drainage from a drain placed by a stab wound into the organ/space
  - Organisms isolated from organ/space by aseptic culturing technique
  - Identification of abscess in the organ/space by direct examination, during reoperation, or by histopathologic or radiologic examination
  - Diagnosis of organ/space SSI by surgeon or attending physician.

**D. Epidemiology**

Frequency

*I.Worldwide*

Over the last few decades, the Cesarean rates have increased remarkably in the developed and the developing world.

The National Center for Health Statistics reported that the percentage of cesarean births in the United States increased from 20.7% in 1996 to 32% in 2007.[23]

In the United States, 1.2 million or 29.1% of births were by c-section delivery in 2004. According to the report of the National Institute of Health (2006), in Brazil about 37% of the births were through c-section.[24]

Another study shows that the c-section birth rate in China has gone up from 8.9% in 1993-94 to 24.8% in 2001-02.[25]

*II. International*

Internationally, the frequency of SSI is difficult to monitor because criteria for diagnosis might not be standardized. A survey sponsored by the World Health Organization demonstrated a prevalence of nosocomial infections varying from 3-21%, with wound infections accounting for 5-34% of the total.[12] The 2002 survey report by the Nosocomial Infection National Surveillance Service (NINSS), which covers the period between October 1997 and September 2001, indicates that the incidence of hospital acquired infection related to surgical

wounds in the United Kingdom is as high as 10% and costs the National Health Service in the United Kingdom approximately 1 billion pounds (1.8 billion dollars) annually.

*III. United States*

Surgical site infections (SSIs) are not an extinct entity; they account for 14-16% of the estimated two million nosocomial infections affecting hospitalized patients in the United States.[12]

IV. *Indian scenario*

Over the past 2 decades, India is also experiencing a sharp increase in cesarean section delivery. Growing access to obstetric care and the high rural urban differences in the cesarean delivery rates raise a thought on the possible reason for such an increase. National Family Health Survey (NFHS) analyzed the trend of cesarean deliveries from 1992-93 to 2005-2006, and concluded that it is an upward trend. In India, the rate has increased from 2.9% of the childbirth in 1992-93 to 10.6% in 2005-06.[26]

According to NFHS-3, cesarean section rate in rural India was 6.2% compared to urban India which is 17.8% with the remarkable difference of 11.6%.[26]

Data collected from 30 medical colleges/ teaching hospital revealed that cesarean section rates have increased from 21.8% in 1988-89 to 25.4% in 1993-94 and 34.4% in 2006.[27] The rates were comparable to that of another study that reported cesarean section rates of 32.6%.[28]

The surgical site infection rate reports by different workers have differed considerably. A study[12]done in India reported overall infection rate as 8.95% and number of studies carried out in India indicate an overall infection rate of 4.04 to 30% for clean surgeries and 10.06 to 45% for clean-contaminated surgeries.[8]

Collated data on the incidence of wound infections probably underestimate true incidence because most wound infections occur when the patient is discharged, and these infections may be treated in the community without hospital notification.[8]

VI. Mortality/Morbidity

Surgical site infections are associated not only with increased morbidity but also with mortality. Seventy-seven percent of the deaths of surgical patients were related to surgical wound infection.[9] Kirkland et al calculated a relative risk of death of 2.2 attributable to SSIs, compared to matched surgical patients without infection.[29]

## E. Pathogenesis of SSIs[30,31,32]

- *The Human Inflammatory Response*

With the creation of the surgical incision through the skin and in to subcutaneous tissues, 5 critical initiators of the human inflammatory response are activated (Figure 2).

Coagulation proteins and platelets are initially activated as part of the human hemostatic mechanism, but they also herald the onset of inflammation. Mast cells and complement proteins are activated, and bradykinin is produced from its ubiquitous protein precursors. The net effect of these 5 factors is vasodilation and increased local blood flow at the site of the surgical incision. While bulk flow is increased, flow velocity is reduced in preparation for margination of phagocytes.

The simultaneous occurrence of increased vascular permeability and local vasodilation facilitates the formation of edema fluid, resulting in increased space between endothelial cells. The increased vascular permeability provides phagocytic access to the injured soft tissue, while edema provides aqueous conduits for the navigation of these phagocytes through the normally condensed extracellular tissues. Activation products from the 5 initiator events described above result in the production of nonspecific chemoattractant signals, while mast cells produce specific chemokine signals that "draw" specific neutrophil, monocyte, and other leukocyte populations into the area of the surgical site.

The important point of this discussion about inflammation is that tissue injury from the incision initiates the mobilization of phagocytes into the wound before bacterial contamination actually occurs from the procedure itself. This mobilization of the innate host defenses before significant intraoperative contamination occurs undoubtedly gives the patient an advantage against infection as an outcome.

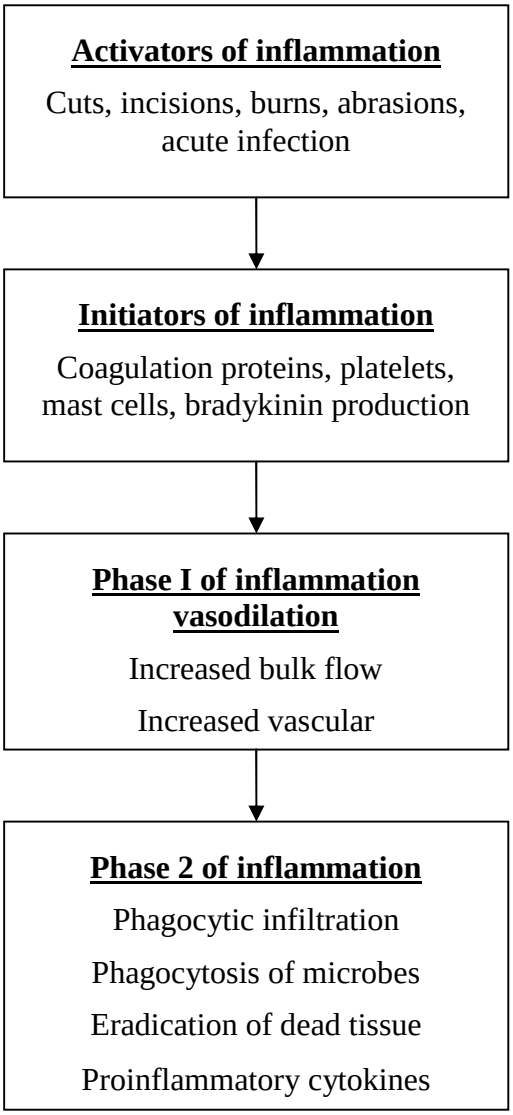



**Figure 2. The consequences of inflammation**

The abundant release of chemoattractant signals, products of tissue injury, orchestrates the movement of phagocytes into the wound. Chemoattractant signaling proteins bind to local vascular endothelial cells and upregulate selectin proteins on the endothelial surface of these cells, which results in neutrophil "rolling" on the endothelial surface within the post-capillary venule. Further interaction between neutrophil and endothelial cell adhesion proteins anchor the neutrophil to the surface of the endothelial cell, and the chemoattractant gradient then acts as a biological "beacon" to direct neutrophil movement toward the site of injury. Neutrophil presence at the surgical site allows systematic ingestion and digestion of any microbial contaminants from the operation.

By about 24 hours after creation of the surgical wound, monocytes enter the surgical site and initiate 1 of 2 different scenarios. When microbial contamination has been minimal and the early arriving neutrophils have been able to adequately control the bacteria that are present, then monocytes produce local chemical signals to regulate the wound-healing process. Myofibrocytes migrate into the fibrin matrix of the wound, and collagen deposition displaces its fibrin latticework. However, if microbial contamination and proliferation overwhelm the initial neutrophil infiltration, the monocyte assumes the role of a proinflammatory cell with the release of potent cytokines.

Tumor necrosis factor (TNF)-alpha is produced and released by the monocytes and serves numerous functions; notably, it becomes a potent paracrine signal to upregulate vigorous neutrophil activity within the wound. TNF-alpha-stimulated neutrophils consume microbes, and lysosomal vacuoles may release reactive oxygen intermediates and acid hydrolases into the extracellular space

from its lysosomal vacuoles. The extracellular release of reactive oxygen intermediates and the acid hydrolases results in lipid peroxidation of the local environment, with further tissue injury and further activation of the initiator signals. In this way, the entire inflammatory response is further intensified. Interleukin (IL)-1, IL-6, and other proinflammatory signals are released by the activated monocyte and serve as endocrine signals responsible for fever, stimulation of acute phase reactants, and other responses.

The net effect of vigorous neutrophilic stimulation, tissue autolysis, and sustained stimulation of inflammatory initiation is the creation of a wound space that is a host-pathogen battlefield. Ultimately, the wound space is filled with necrotic tissue, neutrophils, bacteria, and proteinaceous fluid that together constitute pus. The viable tissues around the infected wound typically exhibit the classic signs of inflammation. Wound *rubor* reflects local vasodilation. *Calor* is the warmth of the vasodilated tissues resulting in increased heat conduction. *Tumor* reflects the presence of edema fluid about the wound. *Dolor* occurs from stimulation of nerve nociceptors by the numerous products of the inflammatory cascade and tissue injury. The discharge of pus from the wound interface via the incision completes the natural history of SSI.

## F. Pathophysiology of wound healing[33]

Wound healing is a continuum of complex interrelated biological processes at the molecular level. Healing is divided into the following phases for descriptive purposes: inflammatory phase, proliferative phase, and maturation phase.

The inflammatory phase commences as soon as tissue integrity is disrupted by injury; this begins the coagulation cascade to limit bleeding. Platelets are the first of the cellular components that aggregate to the wound, and, as a result of their degranulation (platelet reaction), they release several cytokines (or paracrine growth factors). These cytokines include platelet derived growth factor (PDGF), insulinlike growth factor-1 (IGF-1), epidermal growth factor (EGF), and fibroblast growth factor (FGF). Serotonin is also released, which, together with histamine (released by mast cells), induces a reversible opening of the junctions between the endothelial cells, allowing the passage of neutrophils and monocytes (which become macrophages) to the site of injury.

This large cellular movement to the injury site is induced by cytokines secreted by the platelets (chemotaxis) and by further chemotactic cytokines secreted by the macrophages themselves once at the site of injury. These include transforming growth factor alpha (TGF-alpha) and transforming growth factor beta (TGF-beta). Consequently, an inflammatory exudate that contains red blood cells, neutrophils, macrophages, and plasma proteins, including coagulation cascade proteins and fibrin strands, fills the wound in a matter of hours. Macrophages not only scavenge but they also are central to the wound healing process because of their cytokine secretion.

The proliferative phase begins as the cells that migrate to the site of injury, such as fibroblasts, epithelial cells, and vascular endothelial cells, start to proliferate and the cellularity of the wound increases. The cytokines involved in this phase include FGFs, particularly FGF-2, which stimulates angiogenesis and epithelial cell and fibroblast proliferation. The marginal basal cells at the edge of

the wound migrate across the wound, and, within 48 hours, the entire wound is epithelialized. In the depth of the wound, the number of inflammatory cells decreases with the increase in stromal cells, such as fibroblasts and endothelial cells, which, in turn, continue to secrete cytokines. Cellular proliferation continues with the formation of extracellular matrix proteins, including collagen and new capillaries (angiogenesis). This process is variable in length and may last several weeks.

In the maturation phase, the dominant feature is collagen. The dense bundle of fibers, characteristic of collagen, is the predominant constituent of the scar. Wound contraction occurs to some degree in primary closed wounds but is a pronounced feature in wounds left to close by secondary intention. The cells responsible for wound contraction are called myofibroblasts, which resemble fibroblasts but have cytoplasmic actin filaments responsible for contraction.

The wound continuously undergoes remodeling and try to achieve a state similar to that prior to injury. The wound has 70-80% of its original tensile strength at 3-4 months postoperative.

## G. Causes

All surgical wounds are contaminated by microbes, but in most cases, infection does not develop because innate host defenses are quite efficient in the elimination of contaminants. A complex interplay between host, microbial, and surgical factors ultimately determines the prevention or establishment of a wound infection.[34]

- Factors that affect surgical wound healing

**I. Microbial factors:**

Microbial factors that influence the establishment of a wound infection are the bacterial inoculum, virulence, and the effect of the microenvironment. When these microbial factors are conducive, impaired host defenses set the stage for enacting the chain of events that produce wound infection.[35,36,37]

Most SSIs are contaminated by the patient's own endogenous flora, which are present on the skin, mucous membranes, or hollow viscera. The traditional microbial concentration quoted as being highly associated with SSIs is that of bacterial counts higher than 10,000 organisms per gram of tissue (or in the case of burned sites, organisms per $cm^2$ of wound).[36]

Organisms associated with SSIs vary with type of procedure and anatomic location of the operation. The usual pathogens on skin and mucosal surfaces are gram-positive cocci (notably staphylococci, streptococci) account for most exogenous flora involved in SSIs. The most common group of bacteria responsible for SSIs are *Staphylococcus aureus*. The emergence of resistant strains has considerably increased the burden of morbidity and mortality associated with wound infections. However, gram-negative aerobes and anaerobic bacteria contaminate skin in the groin/perineal areas. The contaminating pathogens in gastrointestinal surgery are the multitude of intrinsic bowel flora, which include gram-negative bacilli (eg, *Escherichia coli*) and gram-positive microbes, including enterococci and anaerobic organisms. Sources of such pathogens include surgical/hospital personnel and intraoperative

circumstances, including surgical instruments, articles brought into the operative field, and the operating room air.[36]

Methicillin resistant *Staphylococcus aureus* (MRSA) is proving to be the scourge of modern day surgery. Like other strains of *S aureus,* MRSA can colonize the skin and body of an individual without causing sickness, and, in this way, it can be passed on to other individuals unknowingly. Problems arise in the treatment of overt infections with MRSA because antibiotic choice is very limited. MRSA infections appear to be increasing in frequency and are displaying resistance to a wider range of antibiotics.[37]

Of particular concern are the vancomycin intermediate *Staphylococcus aureus* (VISA) strains of MRSA. These strains are beginning to develop resistance to vancomycin, which is currently the most effective antibiotic against MRSA.[36]

**Table 1. Pathogens Commonly Associated with Wound Infections and Frequency of Occurrence**

| Pathogen | Frequency (%) |
|---|---|
| *Staphylococcus aureus* | 20 |
| Coagulase-negative staphylococci | 14 |
| Enterococci | 12 |
| *Escherichia coli* | 8 |
| *Pseudomonas aeruginosa* | 8 |
| *Enterobacter* species | 7 |
| *Proteus mirabilis* | 3 |
| *Klebsiella pneumoniae* | 3 |
| Other streptococci | 3 |
| *Candida albicans* | 3 |
| Group D streptococci | 2 |
| Other gram-positive aerobes | 2 |
| *Bacteroides fragilis* | 2 |

In clean surgical procedures, in which the gastrointestinal, gynaecologic, and respiratory tracts have not been entered, *Staphylococcus aureus* from the exogenous environment or the patient's skin flora is the usual cause of infection. In other categories of surgical procedures, including clean-contaminated, contaminated, and dirty, the polymicrobial aerobic and anaerobic flora closely resembling the normal endogenous microflora of the surgically resected organ are the most frequently isolated pathogens.[36]

- *Independent risk factors*:[38]

**II. Patient Related variables:**

Diabetes: The mechanism for increased post operative infection may relate to high serum glucose inhibiting the immune response needed for fighting bacterial and fungal infection.[39]

It has 5 times the risk of infection of non diabetic even with clean incision.[40] The explanation for difference in wound healing is complex. The disparity starts with alteration in inflammatory response generated by injury/ incision. These differences in enzyme secretion and growth factor affect all the aspects of normal wound healing such as collagen synthesis and deposition, leukocyte tissue and tissue perfusion.[41]

Obesity increases risk substantially when the subcutaneous abdominal fat layer exceeds 3 cm (1.5 inches) (Nyström et al 1987). The risk is increased by the need for a larger incision, decreased circulation to the fat tissue or the technical difficulty of operating through a large fat layer. Obesity Obesity, usually defined as having a body-mass index greater than or equal to 30 kg/m,2 is another patient risk factor for SSI that has proven difficult to pin down.23-25 A lot of the confusion around the extent of risk caused by obesity is that its role is often difficult to extricate from the effect of severe DM, a common comorbid partner. Often there is insufficient time prior to the surgery to significantly reduce the patient's degree of obesity. However, evaluation for the existence of diabetes and implementation of serum glucose control, if necessary, will go a long way to minimize the risk obesity may present for subsequent SSI. Additionally, major

surgery is often viewed as a life-altering event and may serve to provide patients with the motivation needed to adopt healthier eating habits and other positive lifestyle changes. Personalized education and diets from nutritionists, and support from weight-loss groups have also shown positive long term effects.[42]

Infection at another site may increase the risk of spreading infection through the bloodstream.

Immunocompromised patients (those with HIV/AIDS, those with chronic corticosteroid use such as occurs with asthma and heavy smokers or users of other tobacco products) are at significantly greater risk of SSIs.

Age, socioeconomic status and chronic diseases, such as diabetes and hypertension, are difficult to assess because they are frequently associated with other factors that independently contribute to risk.

Wound characteristics include nonviable tissue in wound; hematoma; foreign material, including drains and sutures; dead space; poor skin preparation, including shaving; and pre-existent sepsis (local or distant).[35]

**III. Obstetrics related variables:**

Characteristics include type of service, previous ceaserean section, pre mature rupture of membranes, maternal fever, chorioamnionitis, ante partum haemorrhage, gestational hypertension, HELLP, DIC, induction of labour, total duration of labour, presence of meconium.

Chorioamnionitis: Tissue infection and clinical circumstances that predispose to infection comprise the major reason for suboptimal wound healing. Specifically long labour, prolonged rupture of membranes and frequent "vaginal examination" are all know risk factors for increasing the rate of infection. The intra-uterine environment during labour can tremendously impact post partum healing.[43]

**IV. Operation related variables:**

Characteristics include type of ceaserean section (elective/ emergency), Type of skin and uterine incision, presence of adhesions, exteriorization of the uterus, additional surgical procedures done, presence of drains, prophylactic antibiotic therapy, closure of subcutaneous fat layer, type of skin closure, suture material used, duration of surgery(>2hrs).[44,]

Have been shown to reduce the incidence of maternal post-operative infection morbidity. Many different antibiotic regimens have reported to be effective. All the clear evidence exists to support this practice, it appears that in clinical practice antibiotic prophylaxis for CESAREAN section is utelized in an inconsistent manner. Both the rate of utilization and the choice of agent for prophylaxis are known to vary. Antibiotic prophylaxis reduces risk of febrile morbidity, endometriosis, wound infection, urinary tract infection (Gibbs 1980; Boggers 1996; Leigh 1999) other serious post operative complication. It has been demonstrated that there is reduction in the relative risk of endometriosis and wound infection for women undergoing elective CESAREAN section as well, as those having emergency procedure (Padiallla 1983; Mohamed 1988). It in not

clear whether any one particular agent is the drug of choice. Many other differen regimengs have been reported to be effictive in decreasing post operative infectious morbidity.[45]

The type of procedure is a risk factor. Certain procedures are associated with a higher risk of wound contamination than others. Surgical wounds have been classified as clean, clean-contaminated, contaminated, and dirty-infected as shown in table number 3.

A cohort study was done in 1997.Which included 969 women who delivered by c-section.

Febrile morbidity and infection complications were noted in 16.2% and 12.4% respectively 9.8% women had Surgical site infection was observed.The risk factors independently associated with infection included, preoperative remote infection(OR-16.5); chorioamnionitis(OR-10.6); maternal-preoperative condition(OR-5.3); pre eclampsia(OR-2.3) higher body mass index(OR-2); nulliparity(0R-1.8); increased surgical blood loss(OR-1.3).45

Another study was done in texas tech health center at Amarillo USA mainly to evaluate obesity (BMI more than 30 )as an independent risk factor for SSI.

Out of the total 574 cases, 360 cases were elective and 214 were emergency in nature. In emergency group significant risk factors identified were labour length(18.4hrs), number of vaginal examination and BMI. In Elective group increased BMI was found to be associated significantly with SSI.[11]

A population based study was done in Soraka university medical center and Ben Gugrion university of Negev mainly to identify risk factors for early wound infection prior to discharge following LSCS.

Of 19416 cesarean deliveries performed during the study period 726 were followed by wound infection. The risk factors identified were obesity (OR-2.2) premature rupture of membranes (OR-1.5) diabetes mellitus (OR-1.4) emergency cesarean delivery (OR 1.3) twin delivery (OR 1.6).Combined obesity and diabetes increased the risk for wound infection 9.3 fold.38

Another study done to assess the effect of subcutaneous tissue thickness at the surgical site on abdominal wound infection after cesarean section reported 7.8% risk of SSI.

Risk factors identified as significantly associated with wound infection by univariate analysis were thickness of subcutaneous tissue, maternal weight and body mass index. Multiple logistic regression analysis confirmed that subcutaneous tissue thickness as the only significant risk factor for wound infection with a relative risk of 2.8.43

A study was done mainly done to assess the effects of prophylactic antibiotics treatment on infectious complications in women undergoing cesarean delivery.

Use of prophylactic antibiotics in women undergoing cesarean section substantially reduced the incidence of episodes of fever, wound infection, urinary tract infection and other serious infections.The relative risk for elective cesarean

section was 0.38 and for non elective cesarean section 0.39.The over all risk in all patients was 0.39.44

**RISK FACTORS:**

| | | |
|---|---|---|
| Patient related variables:<br>1)Age<br>2)Diabetes<br>3)Anemia<br>4)HIV | Obstetric related variables:<br>1)ANC<br>2)Parity<br>3)GA<br>4)Previous cesarean section<br>5)PROM<br>6)Chorioamnionitis<br>7)Antepartum haemorrhage<br>8)Pre eclampsia<br>9)HELLP<br>10)DIC<br>11)Induction of labour | Operation reated variables:<br>1)Elective / emergengcy section<br>2)Type of skin incision<br>3)Uterine incision<br>4)Additional surgical procedure<br>5)Prophylactic antibiotic<br>6)Suture material used<br>7)Duration of surgery |

# Chapter 4

# Methodology

# METHODOLOGY

The present study was conducted in the Department of Obstetrics and Gynecology, at KLES's Dr Prabhakar kore Charitable Hospital attached to JN Medical College, Belgaum, Karnataka.

**Study design**

A Case control study.

**Time line for the study**

| PHASE | TIME PERIOD | OUTLINE |
|---|---|---|
| I | June 2012 to August 2012 | 1.Identification of problem<br>2.Review of Literature<br>3.Development of data collection instrument<br>4.Submission of Synopsis |
| II | February 2013 to June 2014 | 1.Enrolment<br>2.Data Collection |
| III | August 2014 | 1.Analysis of collection data<br>2.Discussion |
| IV | September 2014 | 1.Submission of dissertation |

**Source of data**

All pregnant women who have undergone elective/ emergency cesarean section admitted under the Department of Obstetrics and Gynecology, at the KLES's Dr Prabhakar Kore's Charitable hospital attached to JN Medical college, Belgaum, Karnataka.

**Sample size**

A total of 1050 pregnant women divided into two groups Case (350) and Control (700).

**Sampling procedure**

Sample size was calculated using the formula as below

$n=4pq/z^2$

Where p is prevelence (15%)

Q(100-P)

D-ERROR that is 10%

Assuming a total sample size of 1050, out of which 350 are cases and 700 are controls.

**Method of Collection of Data**

Ethical clearance

The ethical clearance was obtained from JNMC institutional ethics committee on Human Subject Research, Belgaum (Letter No. MDC/DOME/895 dated 11/10/2012) (Annexure I)

Screening and Enrolment

*Eligibility criteria*

Inclusion criteria

Patients who have undergone cesarean section in KLE'S Dr.Prabhakar Kore Charitable Hospital attached to JN Medical College Belgaum, Karnataka.

Exclusion criteria

Patients who have undergone Lower segment Cesarean section outside and admitted in KLE'S Dr.Prabhakar Kore Charitable Hospital attached to JN Medical College Belgaum. For further management.

All potentially eligible women for elective or emergency cesarean section were approached (only if the woman is capable of providing informed consent, which was demonstrated by her ability to respond to questions). These women were informed about the nature of study. Adequate time was provided for describing the study and fielding questions from the patient. No undue pressure was used on the patients to enroll in the study. It was further explained that lack of participation will not affect the usual and anticipated standard of care.

The informed consent form (Annexure II) was explained and provided by the co-investigator in the language understood by the woman to be enrolled (in the local language, Kannada/ English/ Marathi). This was only a small component of the consenting process. Signature of the consenting woman or left hand thumb impression was obtained. This process was done at the time of

enrollment into the study. Recruitment, eligibility, provision of consent and enrollment was conducted by the co-investigator.

Data collection instrument

Data collection instrument (as mentioned in Annexure III) containing information about present and past pregnancies was completed. The co-investigator, previously trained in the administration of the questionnaire, completed the Antenatal Record of the data collection instrument at the time of enrolment into the study. The pre-operative, intra-operative, post-operative information were recorded.

All these variables will be documented in the proforma following cesarean section and surgical site infection will be assesed on 4th post operative

All cases will be advised at the time of discharge to report immediately if they have symptoms of surgical site infection like pain, discharge, wound gaping.

All patients are followed upto one month of cesarean section to reassess the surgical site for evidence of surgical site infection.

All eligible women who has undergone cesarean section (elective/emergency) at KLE'S Dr. Prabhakar Kore Charitable Hospital attached to JN Medical College, Belgaum, were recruited in the study.

And they were divided into 2 categories:

I) Cases

II) Controls

Both cases and controls were assessed for patient, obstetric and surgical related variables for surgical site infection.

CASES:

Patients who develop post cesarean section surgical site infection identified during primary hospitalization prior to discharge or during post operative follow up on out patient basis after discharge or rehospitalization with symptoms and signs of surgical site infection within 30 days after cesarean section.

CONTROLS:

Patient who do not develop surgical site infection within 30 days after cesarean section will serve as control.

Both cases and control will be assessed for patient, obstetric and surgical related variables for surgical site infection.

# Chapter 5

# Results

# RESULTS

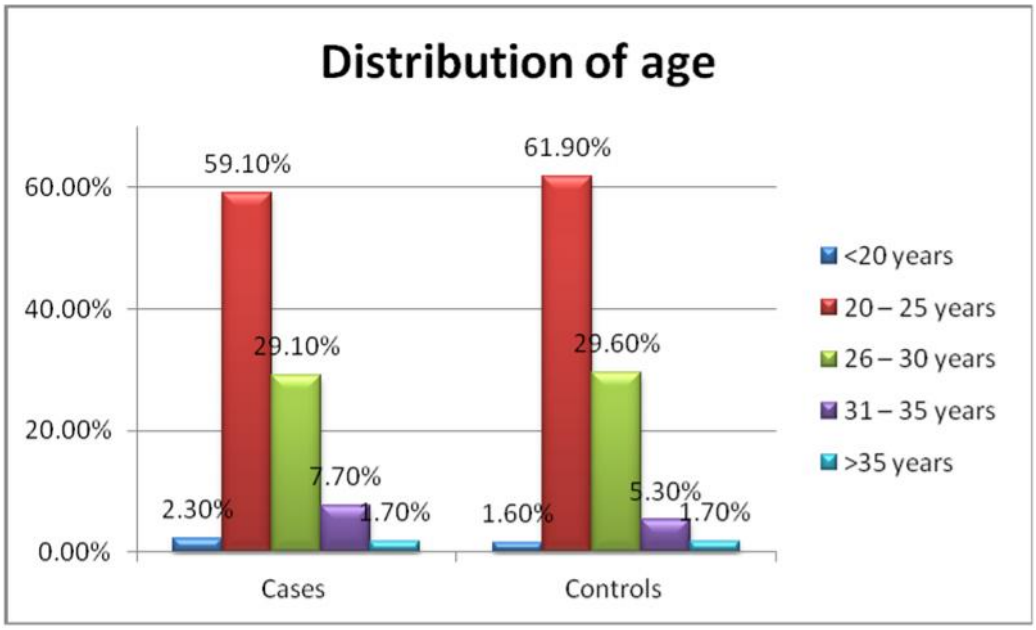
This case control study was conducted in the Department of Obstetrics and Gynecology, KLE'S Dr. Prabhakar Kore Charitable Hospital attached to JN Medical, Belgaum. During the study period from January 2014 to December 2014 a total of 1050 women fulfilled the selection criteria and were divided into case(350), control(700) were enrolled.

Data was obtained and analysed. The final results and observations were tabulated as below.

**Table.no1: Distribution based on age**

| Age group | GROUP | | Odds ratio | P value |
|---|---|---|---|---|
| | **Cases** | **Controls** | | |
| 18-20 years | 8(2.3%) | 11(1.6%) | 1.46 | 0.41 |
| 20 – 25 years | 207(59.1%) | 433(61.9%) | 0.89 | 0.39 |
| 26 – 30 years | 102(29.1%) | 207(29.6%) | 0.97 | 0.88 |
| 31 – 35 years | 27(7.7%) | 37(5.3%) | 0.19 | 0.0001 |
| 35-40years | 6(1.7%) | 12(1.7%) | 1 | 1 |

Pearson Chi-Square - 3.212 df- 4

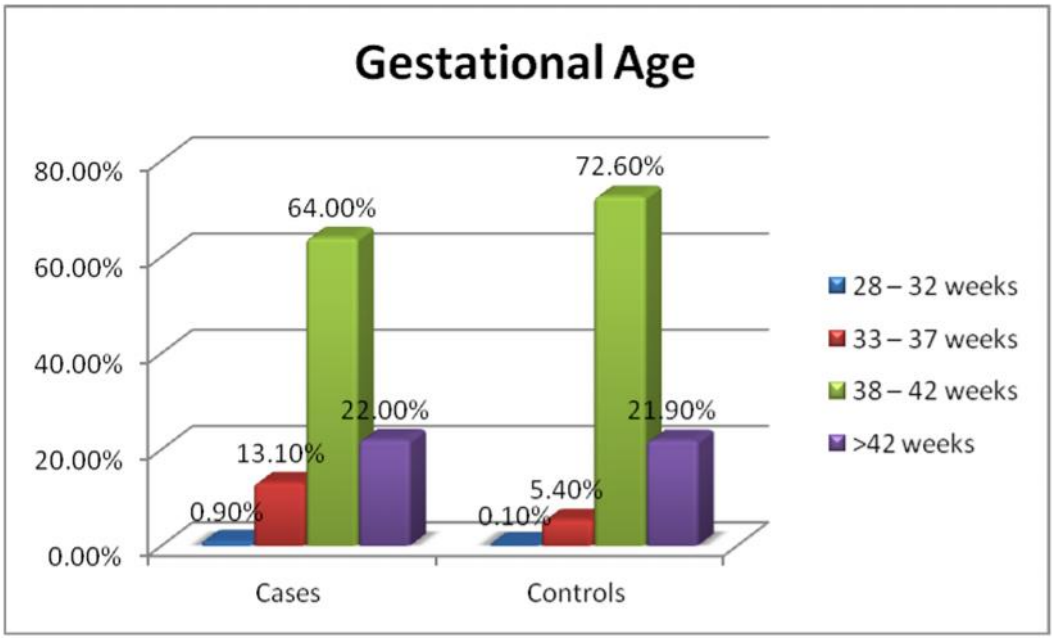



The mean age in cases group was 22.1 ± 3.1 years and the range being 19 to 38 years. In the control group the mean age was 25.84 ± 3.8 years and with range being 19 to 36 years.

**Table.no2: Distribution based on gestational age**

| Gestation | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| 28 – 32 weeks | 3(.9%) | 1(.1%) |
| 33 – 37 weeks | 46(13.1%) | 38(5.4%) |
| 38 – 42 weeks | 224(64.0%) | 508(72.6%) |
| >42 weeks | 77(22.0%) | 153(21.9%) |

Pearson Chi-Square -22.943 [a] df-3 p value=0 .0001

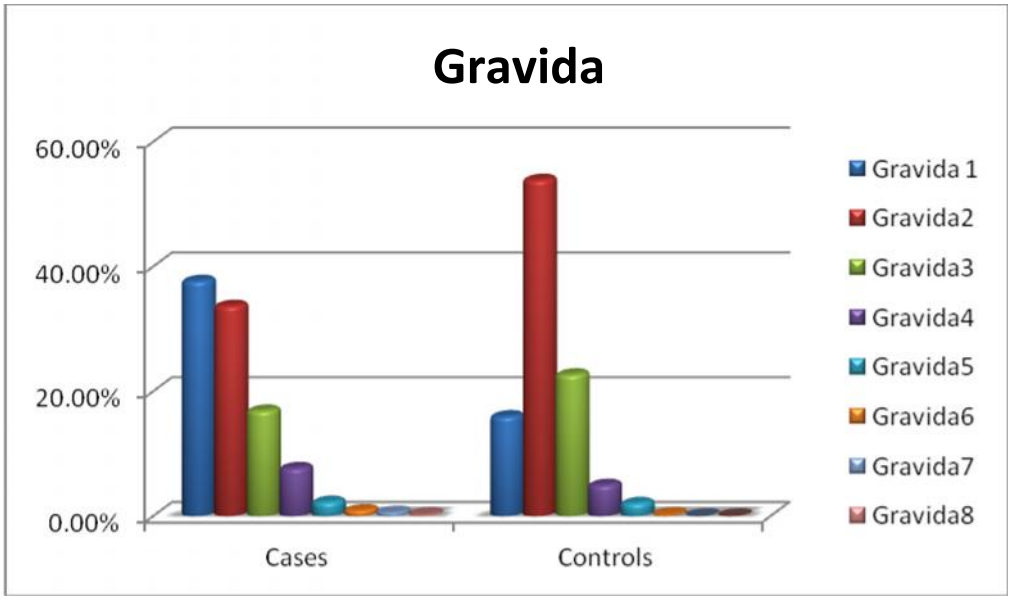



The mean gestational age in case group was (37+2 ± 2wks) with the range being 36-42 weeks. In control group the mean gestational age was (38+2 ± 2wks) with range being 37-41 weeks.

**Table.no3: Distribution based on Gravida**

| Gravida | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| 1 | 132(37.7%) | 112(16.0%) |
| 2 | 118(33.7%) | 377(53.9%) |
| 3 | 59(16.9%) | 159(22.7%) |
| 4 | 27(7.7%) | 35(5.0%) |
| 5 | 8(2.3%) | 15(2.1%) |
| 6 | 3(.9%) | 2(.3%) |
| 7 | 2(.6%) | 00 |
| 8 | 1(.3%) | 00 |

Pearson Chi-Square - 81.81 df- 7 p value=0 .0001




In cases group 132(37.7%) women were primigravida. In control group 373(53.9%) women were gravida 2. With p value of 0.0001. There might be other contributory factors like anemia, hypertensive disorders, previous cesarean section.

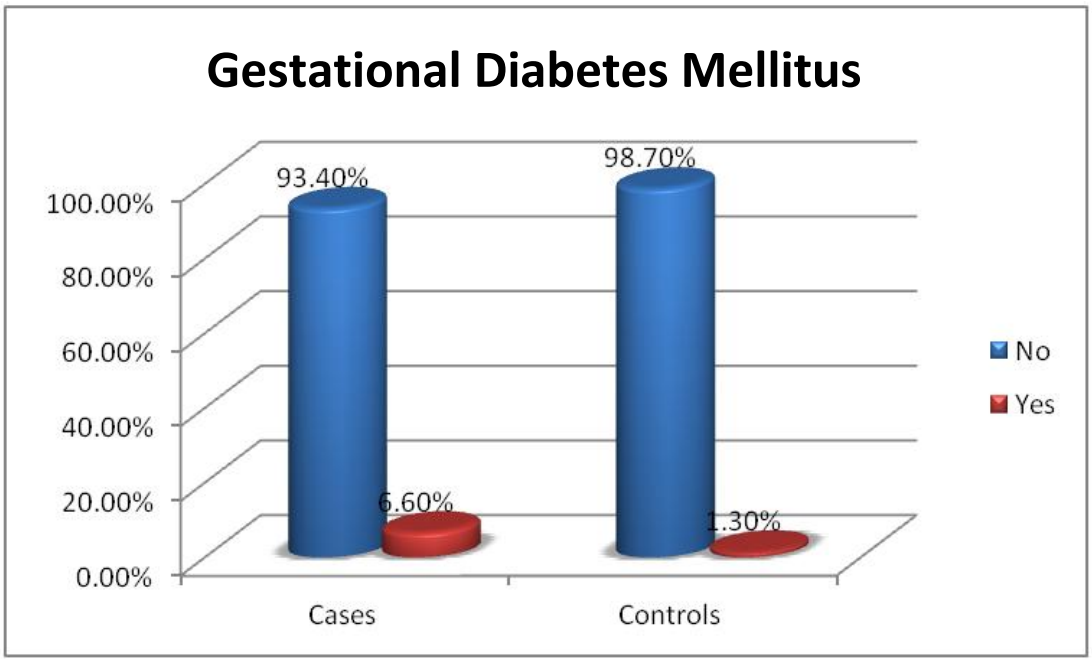
**A. Patient related variables:**

**Table. no 4: Relation between Gestational Diabetes Mellitus and SSI**

| GDM | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 327(93.4%) | 691(98.7%) |
| Yes | 23(6.6%) | 9(1.3%) |

Pearson Chi-Square - 22.063 df- 1 p value-0.0001

Odds ratio = 5.4 95% CI = 2.3 – 12.7

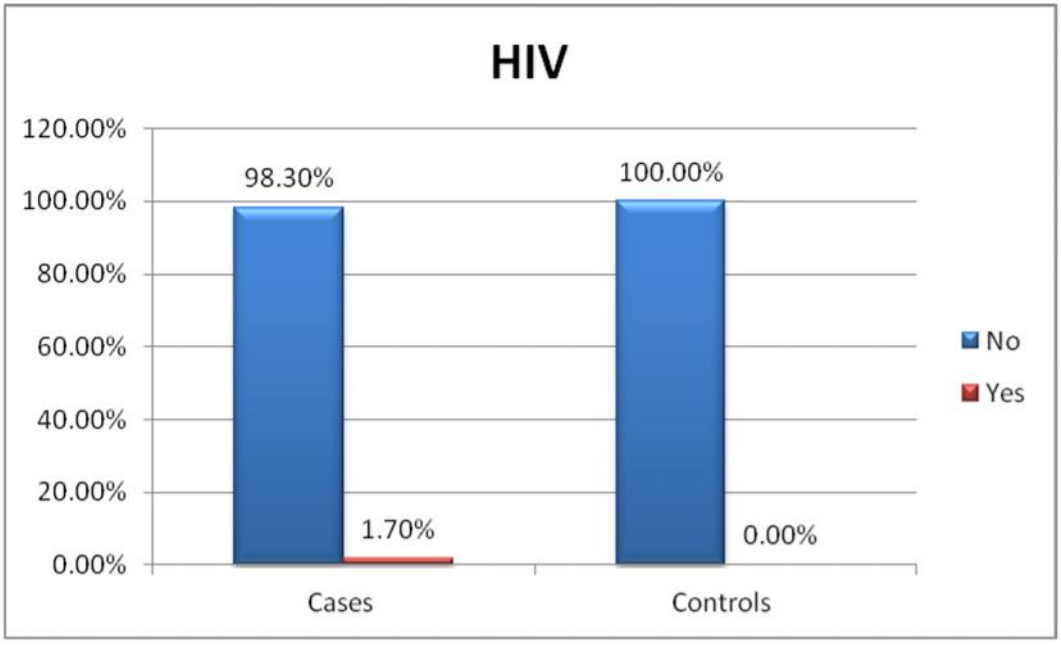



In cases group out of 350 women, 23(6.6%) of them had GDM, who developed wound complication. In control group out of 700 women, 9(1.3%) of them had gestational diabetes who did not develop wound complication. With odds ratio of 5.4 and p value of 0.0001 which was statistically significant.

**Table.no 5: Relation between HIV and SSI**

| HIV | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 344(98.3%) | 700(100.0%) |
| Yes | 6(1.7%) | 00 |

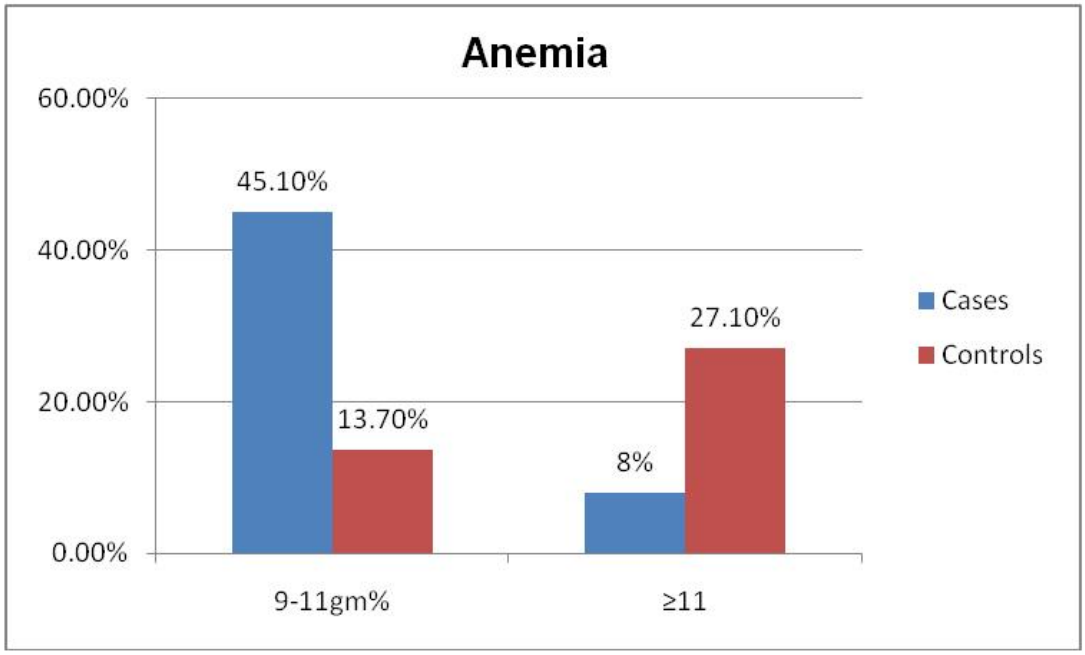
Pearson Chi-Square- 12.069 df- 1 p value =0.001




Among the total of 350 women in cases group only 6(1.7%) women had HIV infection and all 6 of them developed SSI. In control group there were no patients who had HIV infection. The association between HIV infection and SSI with p value of 0.001 was statistically significant. Though the number of HIV positive cases were less in the cases group and none in the control group.

**Table. no 6: Relation between Anemia and SSI**

| | Groups | | Odds ratio | P value |
|---|---|---|---|---|
| | **Cases** | **Controls** | | |
| 9-11gm% | 158(45.1%) | 96(13.7%) | 5.17 | <0.0001 |
| ≥11 | 28(8%) | 152(27.1%) | 0.3 | 0.0001 |
| Total | 350(100.0%) | 700(100.0%) | | |

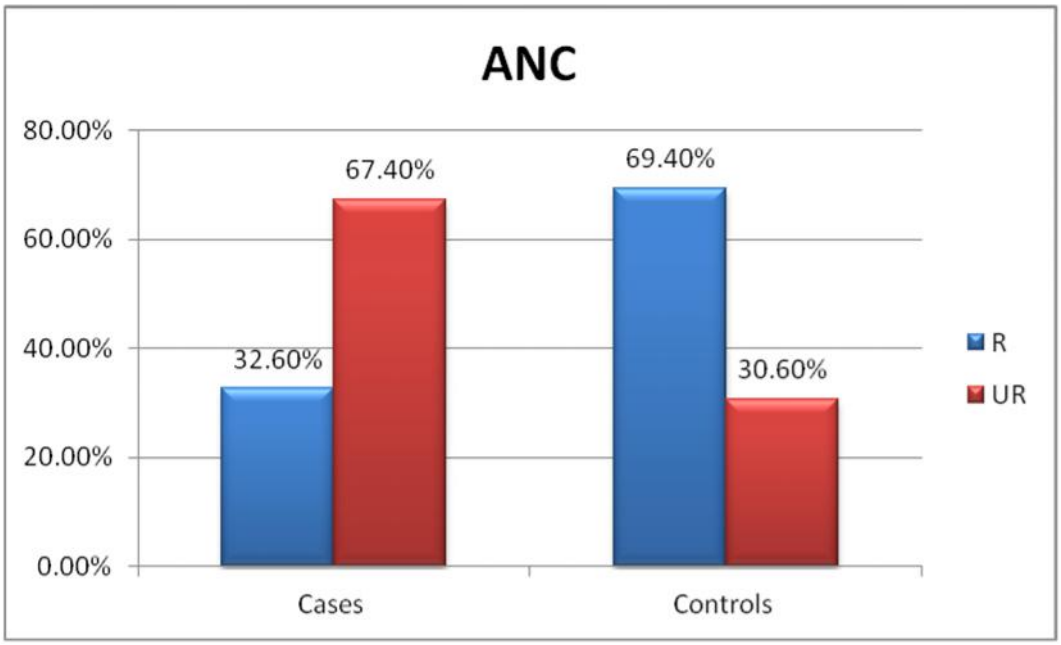



It was observed that in cases group with anemia hemoglobin between 9-11gm% were 158(45.1%) women, >11gm% were 28(8%) women. In control group between 9-11gm% were 96(13.7%) women and >11gm% were 152(27.1%) women. With odds ratio of 5.17 and p value of <0.0001 in haemoglobin between 9-11gm% which is statistically significant. And in women with > 11gm% with odds ratio of 0.3 and p value of <0.0001 which is protective against SSI.

**B. Obstetric related variables**

**Table.no 7: Relation between ANC and SSI**

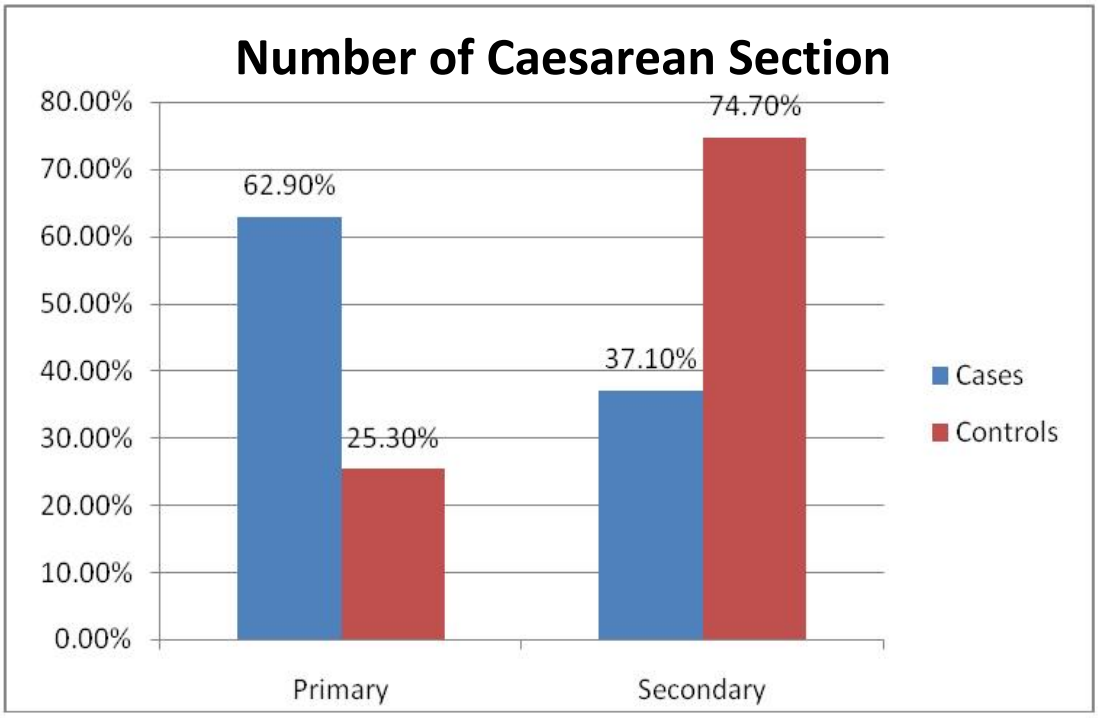
| ANC | GROUP | | Odds ratio | P value |
|---|---|---|---|---|
| | **Cases** | **Controls** | | |
| R | 144(32.6%) | 486(69.4%) | 0.3 | <0.0001 |
| UR | 236(67.4%) | 214(30.6%) | 4.7 | <0.0001 |




In cases group 144(32.6%) women were registered and 236(67.4%) were unregistered. In control group 486(69.4%) women were registered and 214(30.6%) were unregistered. It was found that in patients who were registered had less wound complication with odds ratio of 0.03 and p value of 0.0001, which indicates that registered women were protected from SSI.When compared to unregistered group who had SSI with odds ratio 4.7 and p value of <0.0001 with was statistically significant.

**Table.no 8: Distribution based on number of caesarean section and SSI.**

| | **Groups** | | 0dds ratio | P value |
|---|---|---|---|---|
| | **Cases** | **Controls** | | |
| Primary | 220(62.9%) | 177(25.3%) | 5 | 0.0001 |
| Secondary | 130(37.1%) | 523(74.7%) | 0.2 | 0.0001 |



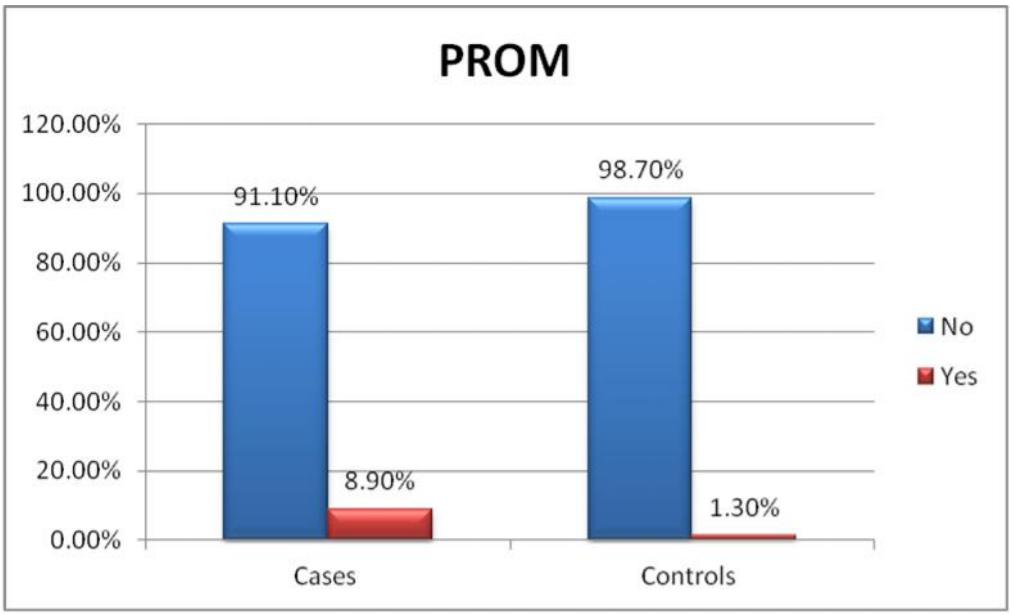

In cases group primary cesarean section were 220 (62.9%) and secondary cesarean section were 130 (37.1%) with odds ratio of 5, p value 0.0001 which was statistically significant. In control group primary cesarean section 177(25.2%) and secondary section 523(74.7%) with odds ratio 0.2 which was statistically not significant. Which is suggestive that women who has undergone primary cesarean section were more prone for wound complication.

**Table.no 9: Relation between Premature rupture of membrane (PROM) and SSI**

| PROM | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 319(91.1%) | 691(98.7%) |
| Yes | 31(8.9%) | 9(1.3%) |

Pearson Chi-Square -6.503 df- 1 p value=0.0001

Odds ratio = 7.4 95% CI = 3.3 – 17.0



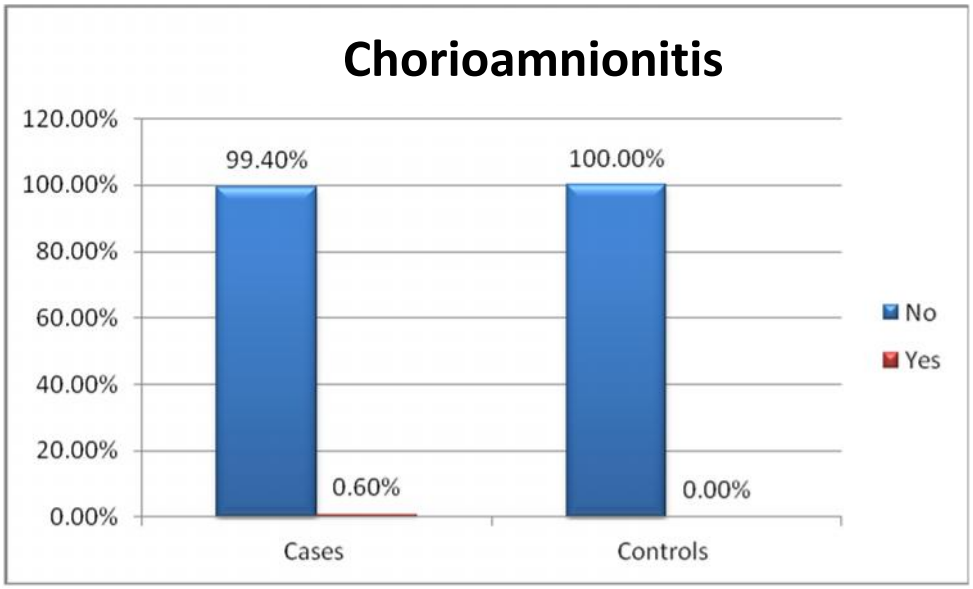

In cases group out of 350 women 31(8.9%) women with PROM had wound complication. In control group out of 700 women 9(1.3%) women had PROM who did not develop wound complication. With odds ratio of 7.4 and p value 0.0001 which was statistically significant. This indicates that there is strong association between PROM and SSI.

**Table.no10: Relation between Chorioamnionitis and SSI**

| CHORIOAMNIONITIS | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 348(99.4%) | 700(100.0%) |
| Yes | 2(.6%) | 00 |
| Total | 350(100.0%) | 700(100.0%) |

Pearson Chi-Square - 4.008 df- 1 p value = 0.045

Odds ratio- 10.6



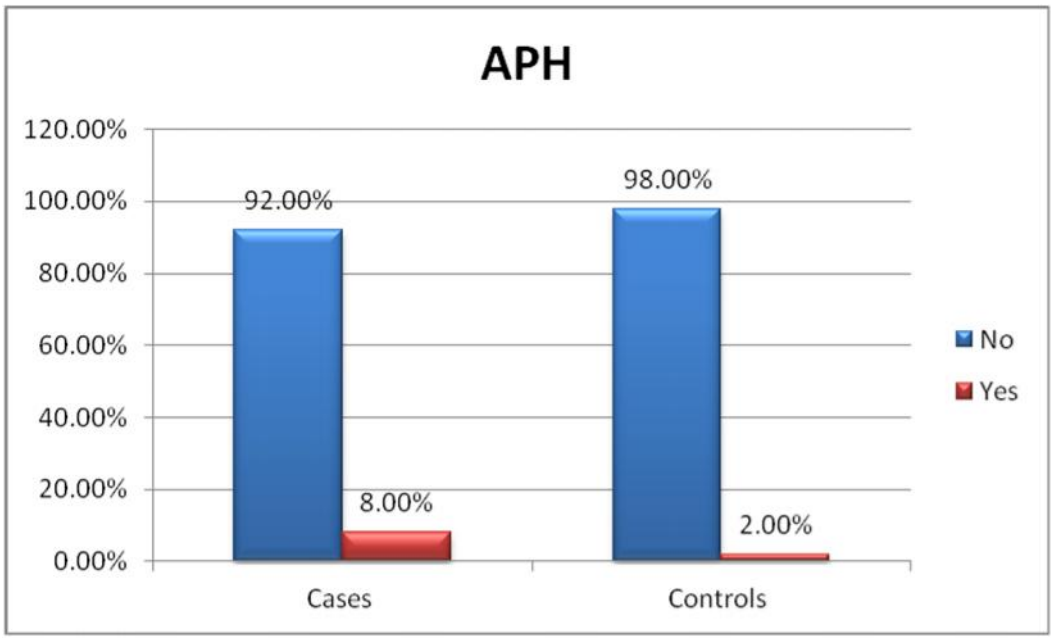

In cases group out of 350 women only 2(0.6%) women had chorioamnionitis and developed wound infection. In control there were no women with chorioamnionitis. With p value of 0.045 which was statistically significant. This shows that there is strong association between chorioamnionitis and SSI, though the number of cases with chorioamnionitis were less in the study.

**Table.no11: Relation between Antepartum haemorrhage (APH) and SSI**

| APH | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 322(92.0%) | 686(98.0%) |
| Yes | 28(8.0%) | 14(2.0%) |

Pearson Chi-Square -21.875 df- 1 p value=0 .0001

Odds ratio = 4.2 95%CI = 2.1 – 8.6



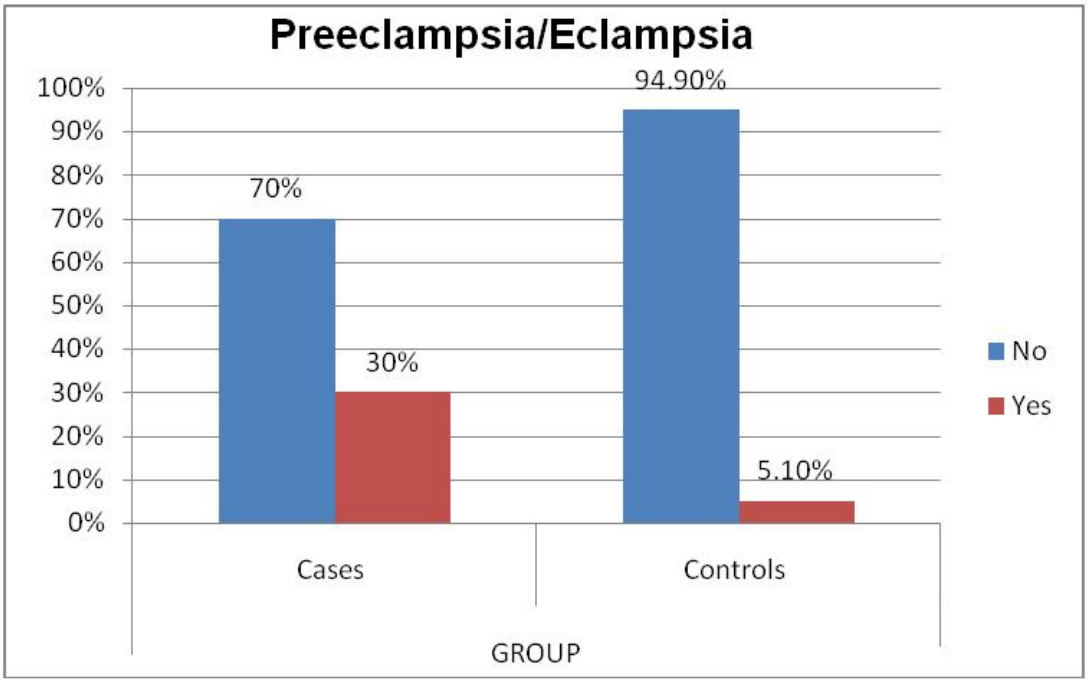

In cases group out of 350 women, 28(8%) women with APH had SSI. In control group 14(2%) women had APH out of 700 who did not develop SSI. With odds ratio of 4.2 and p value of 0.0001 which was statistically significant. This suggests that there is strong association between APH and SSI.

**Table.no12: Relation between Preeclampsia/Eclampsia and SSI**

| Pre eclampsia/Eclampsia | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 245(70%) | 664(94.9%) |
| Yes | 105(30%) | 36(5.1%) |

Pearson Chi-Square-124.015 p value =0.0001 Odds ratio = 7.90

95%CI = 5.2-11.8



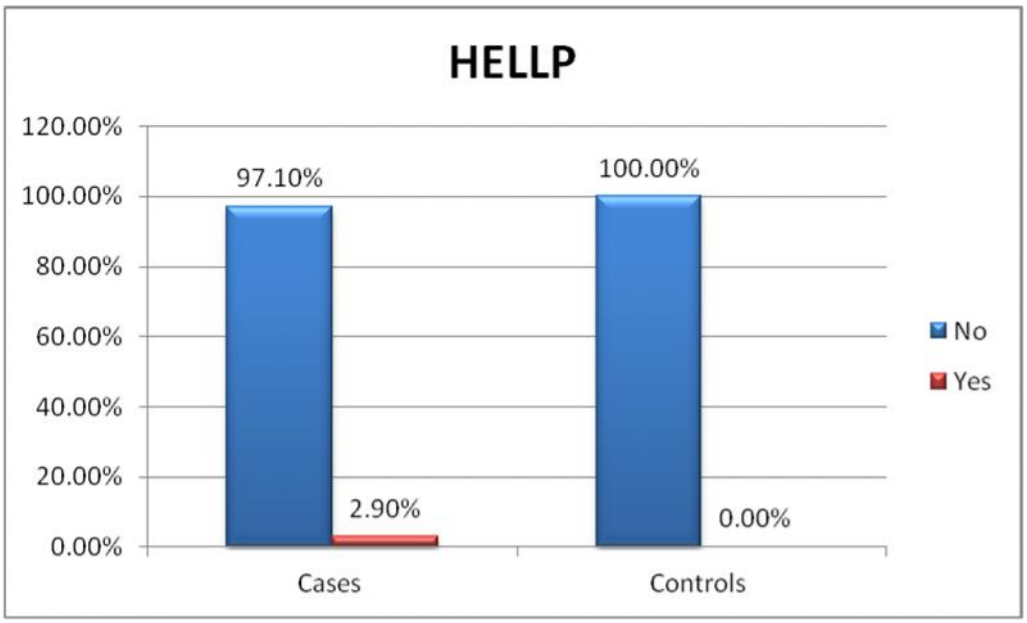

In cases group of 350 women, 105(30%) had preeclampsia/ eclampsia and developed wound complication. In control group of 700 women 36(5.1%) had preeclampsia/ eclampsia who did not develop wound complication. With odds ratio of 7.90 and p value of 0.0001 which was statistically significant.

**Table.no13: Relation between HELLP Syndrome and SSI**

| HELLP Syndrome | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 340(97.1%) | 700(100.0%) |
| Yes | 10(2.9%) | 00 |

Pearson Chi-Square - 32.831 df- 1 p value=0.009

Odds ratio of 43.20

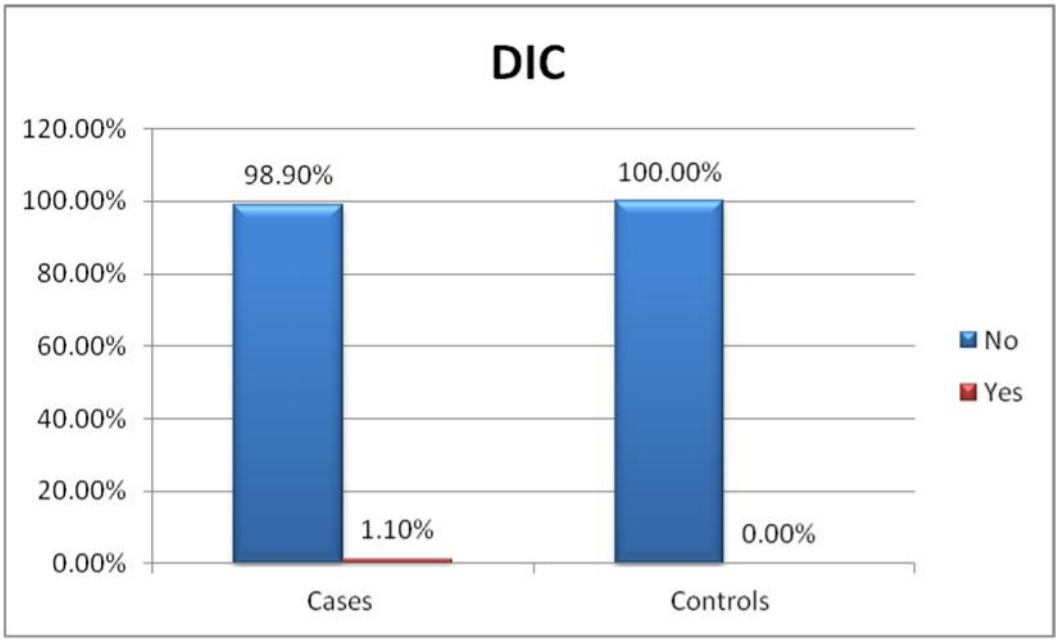



In cases group of 350 women, 10(2.9%) women were with HELLP syndrome. In control group there no women with HELLP syndrome. With P value of 0.0001 which was statistically significant.

**Table.no14: Relation between Disseminated intravascular coagulation (DIC) and SSI**

| DIC | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 346(98.9%) | 700(100.0%) |
| Yes | 4(1.1%) | 00 |

Pearson Chi-Square- 8.031 df 1 p value=0.005



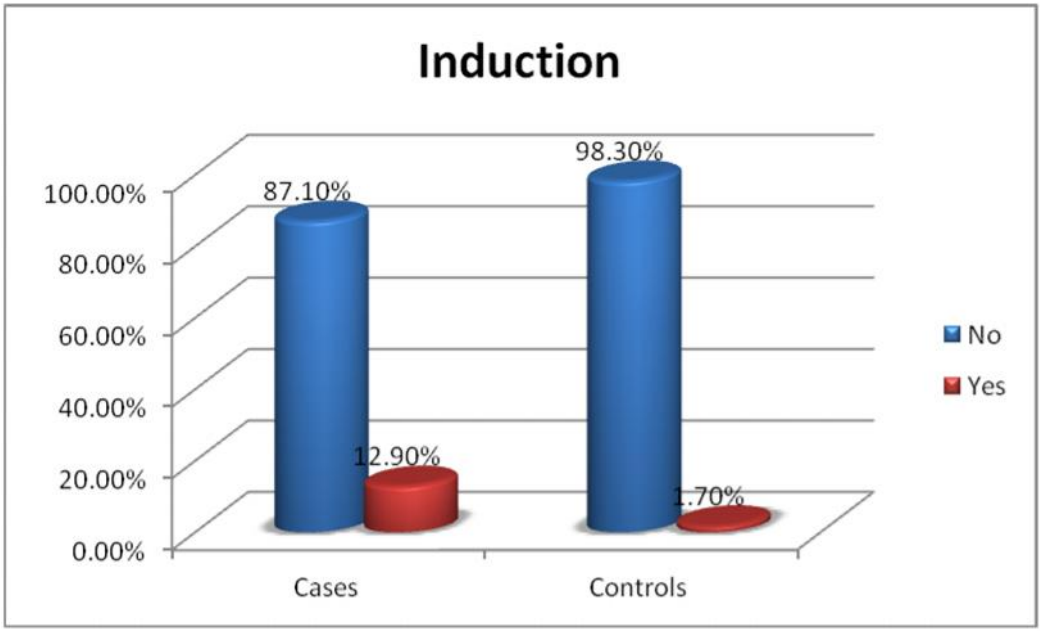

In cases group 4(1.1%) women had DIC out of 350 women. In control group there were no women with DIC. With p value of 0.0005 which was statistically significant.

**Table.no15: Relation between Induction of labour and SSI**

| Induction | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 305(87.1%) | 688(98.3%) |
| Yes | 45(12.9%) | 12(1.7%) |

Pearson Chi-Square -56.432 df- 1 p value =0 .0001

Odds ratio = 8.46 95% CI = 4.26 – 17.1



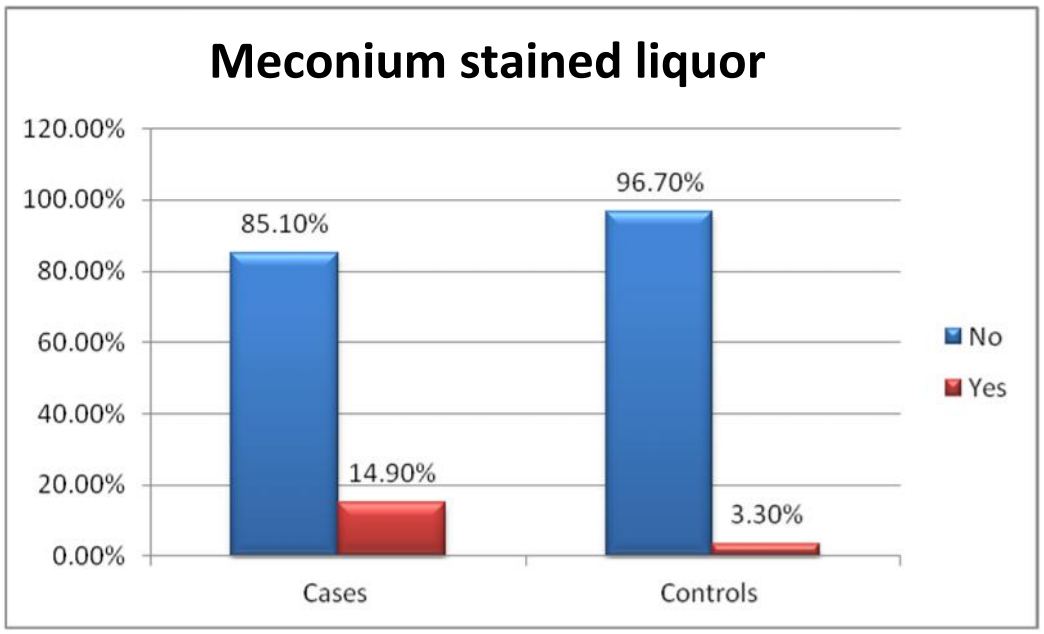

In cases group out of 350 women there were 45(12.9%) women who were induced and had wound wound complication. In control group out of 700 women there were 12(1.7%) women who were induced and did not develop wound complication. With odds ratio of 8.46 and p value 0.0001, which was statistically significant.

**Table.no 16: Relation between meconium stained liquor and SSI**

| MECONIUM | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 298(85.1%) | 677(96.7%) |
| Yes | 52(14.9%) | 23(3.3%) |

Pearson Chi-Square -47.105 df-1 p value =0.0001

Odds ratio = 5.1 95%CI = 3.0 – 8.1

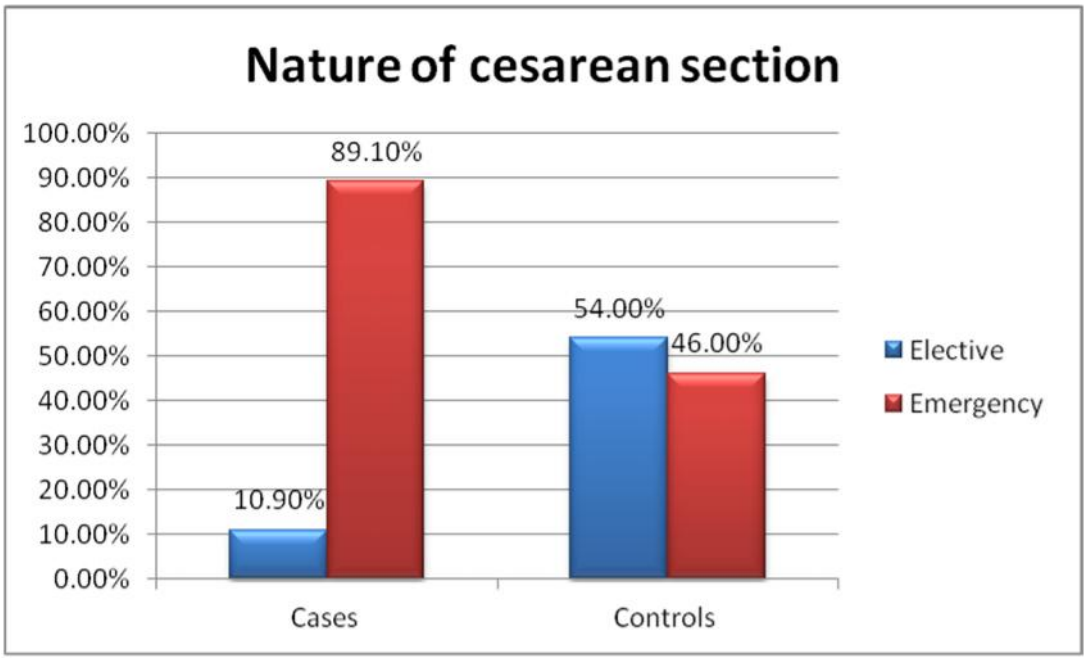



In cases group 52(14.9%) women with meconium stained liquor had wound complication. In control group 23(3.3%) women had meconium stained liquor. With odds ratio of 5.1 and p value of 0.0001, which was statistically significant.

**C. Operation related variables**

**Table.no17: Relation between Nature of LSCS and SSI**

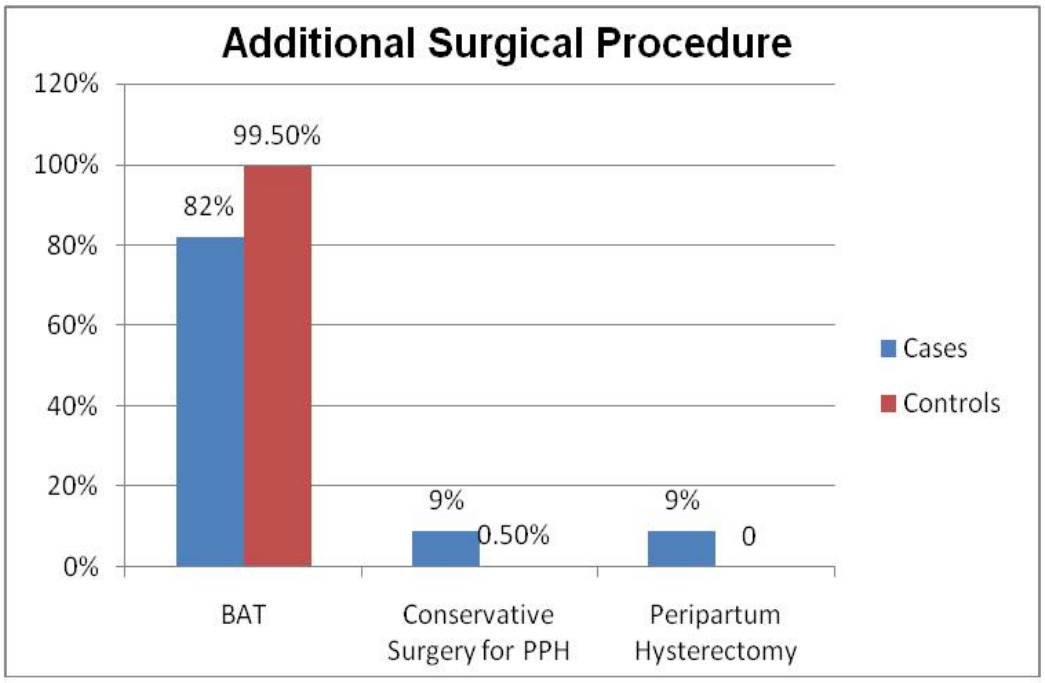
| Nature of LSCS | GROUP | | Odds ration | P value |
|---|---|---|---|---|
| | Cases | Controls | | |
| Elective | 38(10.9%) | 378(54.0%) | 0.1038 | <0.001 |
| Emergency | 312(89.1%) | 322(46.0%) | 9.6 | <0.0001 |




In cases group a total of 38(10.9%) women had elective cesarean section and 312 emergency cesarean section. In control group 378(54.0%) were elective cesarean section and 322(46.0%) were emergency cesarean section.The women who had undergone emergency cesarean section had more wound complication when compared to elective cesarean section. With odd ratio of 9.63 and p value of 0.00011 for emergency cesarean section and odds ratio of 0.10 and p value of 0.001 for elective cesarean section.

**Table. no18: Relation between Additional Surgical Procedure and SSI**

| Additional Surgical Procedure | Groups | | 0dds ratio | P value |
|---|---|---|---|---|
| | Cases | Controls | | |
| BAT | 64 (82%) | 248(99.5%) | 0.4 | |
| Conservative Surgery for PPH | 7 (9%) | 1(0.5%) | 14.4 | 0.01 |
| Peripartum Hysterectomy | 7 (9%) | 0 | 30.54 | 0.01 |

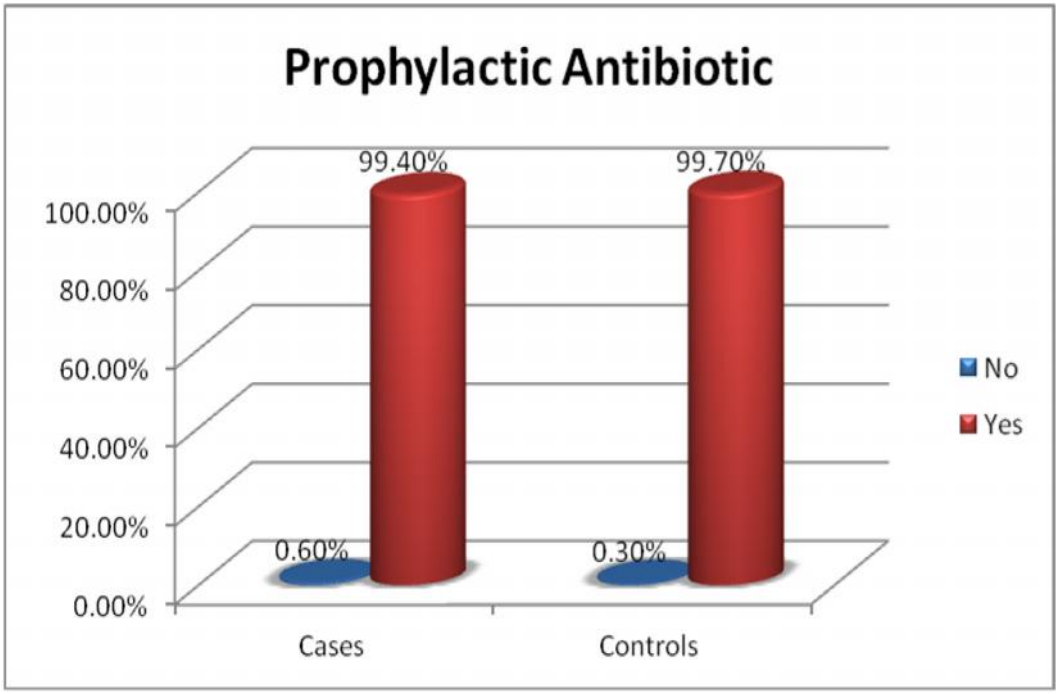



In cases group women who had undergone bilateral abdominal tubectomy (BAT) were 64(82%) (Odds ratio of 0.4), conservative surgery for PPH were 7(9%) (Odds ratio of 14.2 and p value of 0.01) and women who had undergone peripartum hysterectomy were 7(9%) (Odds ratio of 30.54 and p value of 0.01). In control group 248(99.5%) women had undergone BAT, conservative surgery for PPH was 1(0.4) and 0 in peripartum hysterectomy. The risk of SSI was statistically significant in women who had undergone conservative surgery for PPH and peripartum hysterectomy along with C-section.

**Table.no 19: Relation between Prophylactic antibiotic and infection**

| Antibiotic | GROUP | |
|---|---|---|
| | **Cases** | **Controls** |
| No | 2(.6%) | 2(.3%) |
| Yes | 348(99.4%) | 698(99.7%) |

df -1 p value =0.48 Odds ratio = 0.49 95%CI = 0.06 – 3.5

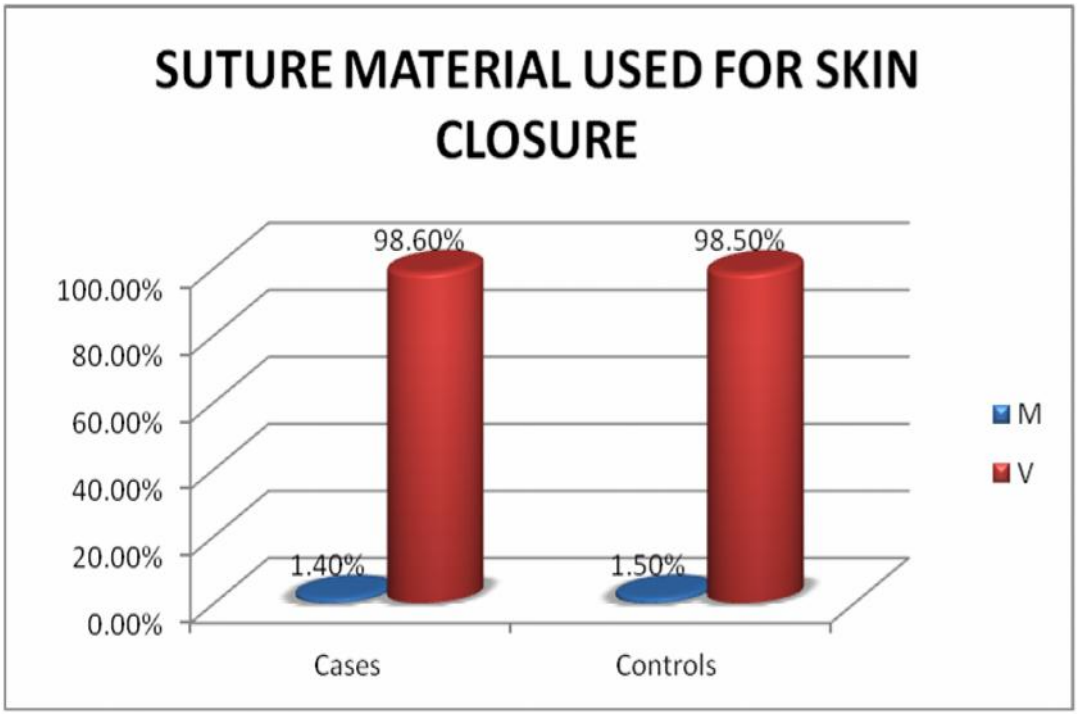



All most all women (99.4%) in both the cases and control group had received prophylactic antibiotic (odds ratio 0.49, p value of 0.48). Therefore there was no statistically significant difference in the risk of SSI among the cases and control in respect to prophylactic antibiotic.

**Table.no20: Relation between suture material for skin closure and SSI**

| SUTURE | GROUP | | Odds ratio | P value |
|---|---|---|---|---|
| | **Cases** | **Controls** | | |
| M | 5(1.4%) | 10(1.5%) | 1 | 1 |
| V | 345(98.6%) | 690(98.5%) | 1 | 1 |

df-1 p value = 1.0 Odds ratio = 1.0 95% CI = 0.3 – 3.0




In this study in both groups use of monocryl or vicryl for skin closure did not show much difference in SSI. With odds ratio of 1 and p value of 1 for monocryl and odds ratio of 1 and p value pf 1 for vicryl respectively. How ever the number of patients in whom skin closed with monocryl were less in both the groups.

# Chapter 6

# Discussion

# DISCUSSION

This case control study was done in Department of Obstetrics & Gynaecology, KLE's Dr Prabhakar Kore Charitable Hospital attached to JN Medical College, Belgaum Karnataka. A total of 1050 women were included, out of which 350 were cases and 700 were controls. All patients who had undergone Lower Segment Cesarean Section at KLE's Dr Prabhakar Kore Charitable Hospital attached to JN Medical College belgaum who consented to be included in the study were recruited. This study was mainly done to assess the independent risk factors influencing surgical site infection (SSI) after cesarean section[1]. Patient related, obstetric related, operation related variables were assessed.

The rate of SSI after cesarean section reported in the literature ranged from 3% to 15%.[38] depending on the surveillance methods used to identify infections, the patient population and the use of prophylactic antibiotics.[38]

In the present study the mean age in cases group was 22.1 ± 3.1 years and the range being 19 to 38 years. In the control group the mean age was 25.84 ± 3.8 years and with range being 19 to 36 years. There was no much difference in relation to age and development of SSI.

The present study showed that GMD is one of the independent risk factor for SSI. With odds ration of 5.4 and p value of 0.0001 which is statistically significant.

Study conducted by Zerr KJ et al.[39,40,41] The mechanism for increased SSI following cesarean section may relate to high serum glucose inhibiting the

immune response needed for fighting bacterial and fungal infection.[39] It has 5 times the risk of infection of non diabetic even with clean incision.[40] The explanation for difference in wound healing is complex. The disparity starts with alteration in inflammatory response generated by injury/ incision. These differences in enzyme secretion and growth factor affect all the aspects of normal wound healing such as collagen synthesis and deposition, leukocyte tissue and tissue perfusion.[41]

Study conducted by Sheined-Kofman N et all [42] showed that GDM is identified as a risk factor for SSI (odds ratio of 1.4; 95% CI, 1.1-1.7) which was statistically significant. Similar results were found in our study also.

With regards to association of SSI in risk factors like HIV (p value 0.001), in the present study there were 6 patients with HIV infections, out of which all 6 had developed surgical site infection, this is mainly due to immunocompromised status which has poor wound healing status. There are no studies available where HIV status has been assessed as an independent risk factor for SSI. In the present study it was found statistically significant but the number of patients were less.

In the present study it was observed that anemia (9-11gm %) is one of the risk factor for SSI (odds ratio of 5.17 and p value of <0.0001) which is statistically significant. Similar study was conducted Margaret A et al [43] showed that anemia is one of the risk factor for SSI ( odds ratio 2.18, 95%CI, 1.30-3.68). This in mainly due hypoprotenemia, low osmotic pressure so this leads to delayed wound healing. Similar results were found in our results.

In the present study the risk of SSI between the antenatal womens who were registered ( odds ratio of 0.3 and p value 0.0001) and unregistered ( odds ratio 4.7, p value <0.0001) was statistically significant. SSI was more in antenatal unregistered women when compared to antenatal registered women. This was mainly due to other variables like anemia, hypertensive disorders, induction of labour which increased the risk for wound complication in unregistered patients. Similar study conducted by Oslen, M.A.Butler.A.M et al[38] was based upon the number of antenatal visits less than 3 visits had increased risk for SSI and more than 5 did not develop SSI.

Study done in Nigeria by Jido TA et al[44] showed that women who didn't have ANC follow up developed more severe type of SSIs than those with ANC follow up (p value <0.05) which is statistically significant. Similar results were observed in our study also.

In the present study it was found that SSI was significantly increased in women undergoing primary cesarean section (62.9%), when compared to repeat cesarean section (37.1%). With odds ratio of 5 and p value of 0.0001 which was statistically significant. Similar study done by Ezechi OC et all [45] showed that most of the women had primary cesarean section (93.1%) which are at a risk of infectious morbidity compared to repeat cesarean section. As most of the repeat cesarean section will be carried out electively. Most of the primary cesarean section were emergency.

In present study it was observed that PROM in cases group 31(8.9%) women and 9(1.3%) womens in control group. With odds ratio of 7.4 and p value

0.0001 which is statistically significant. Which indicates that there is strong association between PROM and SSI. This study was conducted by AR Mahale et al (2008)[46] results were PROM (9.6%) .This is due to prolonged duration of labour, induction of labour, repeated vaginal examinations.

Similar study was conducted Schneid-Kofman N et al[42] showed that premature rupture of membrane is one of the independent risk factor for SSI (odds ratio of 1.5; 95% CI, 1.2-1.9) which is statistically significant.

In this study 2(0.6%) womens in cases group had chorioamnionitis who had SSI. In control group there were no patients who had chorioamnionitis.(odds ratio 10.6%, p value of 0.04) which is statistically significant. Hence there is strong association between chorioamnionitis and SSI. Similar study was conducted Schneid-Kofman N et al [42] showed that chorioamnionitis has increased risk for SSI (odds ratio of 10.6, 95%CI 2.1) which was statistically significant. This it due to prolonged labour, repeated number of vaginal examination.

Another study was done Demisew et al [47] where women with chorioamnionitis at the time of surgery had increased severe form of SSI (p value of 0.001) which is statistically significant.

The present study showed that 28(8%) women had APH and all 28 had developed SSI. In control group 14(2%) women had APH who did not develop SSI. With odds ratio of 4.2 and p value of 0.0001 which is statistically significant. Hence there is association between APH and SSI. This is due to increased blood loss, anemia, prolonged duration of surgery. In the reported literature they have not assessed APH as a risk factor for SSI.

In present study in cases groups 105(30%) women had preeclampsia/ eclampsia. In control group 36(5.1%) women had preeclampsia/ eclampsia. With odds ratio of 7.90 and p value of 0.0001 which is statistically significant. Similar study was conducted Tran TS et al [48] were it was found that preeclampsia is one of the important predictors of SSI (odds ratio of 2.3, 95% CI 1.1, 4.9) which was statistically significant.

Similar study was done Schneid-Kofman N et al [42] which showed that hypertension is one of the independent risk factor for SSI (odds ratio of 1.7; 95% CI 1.4-2.1) which is statistically significant. The increasing in risk of SSI in preeclampsia/ eclampsia is due to extravesation of fluids, edematous abdominal wall, ascites, decreased osmotic pressure, hypoproteinemia.

In this present study, amongst the cases 10(2.9%) womens had HELLP syndrome and all 10 women had wound complication. In the control group none of the patients had HELLP syndrome. With odds ratio of 43.20 and p value of 0.009 which is statistically significant. This is mainly due to the reason that in tissue there will be subcutaneous edema, haemotoma, thick subcutaneou tissue, improper haemostasis or they might be preeclampsia/ eclampsia. Similar study was done Vermillion ST [49] to estimate the thickness of subcutaneous tissue as risk factor for SSI (relative risk of 2.8) (95% CI 1.3)

In present study in cases group 4(1.1%) women were in DIC out of 350 women and all 4 women had developed SSI. In control group there were no women who were in DIC. With p value of 0.005 which is statistically significant. The wound complication in DIC is probably due to defect in clotting factor

mechanism, reduced tissue perfusion. No studies have been done to assess DIC as a independent risk factor for SSI.

The present study showed that induction of labour is one of the risk factor for surgical site infection (Odd's Ratio:8.46, 95%, CI: 4.26-17.1, p value 0.0001) which is statistically significant. This is mainly due to prolonged labour and increased number of vaginal examinations. Similar study was conducted by Margaret A et al [43] which showed that induction of labour is one of the risk factor for SSI (odd s ratio 1.9, p value 0.01) which was statistically significant. Similar study was conducted by Myles TD, Gooch J, SantolayaJ et al[6] that labour length (18.4hours, p<0.003) was found to be significant and risk factor for surgical site infection.

The present study showed that meconium stained liquor is a risk factor for SSI (odds ratio of 5.1 and p value of 0.0001) which is statistically significant. Hence there is strong association between meconium stained liquor and SSI. Similar study was conducted by Demisew A et all [44] it was found that presence of meconium was strongly associated with increased severe form of SSI (p value <0.009). The findings revealed that women might be in prolonged labour, PROM.

In the present study, it was found that patient who has undergone emergency LSCS (odds ratio of 9.6, p value of <0.0001) which is statistically significant, when compared to women who have undergone the elective LSCS (Odd's Ratio of 0.10 and p value of <0.001) which signifies that women who had undergone elective LSCS are protective against SSI. Hence women who had undergone emergency cesarean section are at risk for developing SSI.

In a study conducted by AR Mahale et al (2008)[46] elective and emergency cesarean sections did not show difference in overall wound complication rate, but in their study wound gapes were relatively more in emergency LSCS, whereas in our present study the incidence of wound complication was more in patients who has undergone emergency LSCS (60.4%), when compared to elective LSCS (39.6%).

Similar study was conducted Schneid-Kofman N et al [42] showed that women who had undergone emergency cesarean section are more prone for SSI when compared to elective cesarean section (odds ratio 1.3; 95% CI, 1.1-1.5) which is statistically significant. Similar results were found in our study.

In present study, in cases group women who had undergone bilateral abdominal tubectomy (BAT) were 64 (82%)womens (odds ratio of 0.4), conservative surgery for PPH were 7 ( 9% )womens (odds ratio of 14.2 and p value of 0.01) and womens who had undergone peripartum hysterectomy were 7 (%) women ( odds ratio of 30.54 and p value of 0.01). In control group 248(99.5%) women had undergone BAT, conservative surgery for PPH was 1( 0.5%) and 0 in peripartum hysterectomy. It was statistically significant in women who had conservative surgery for PPH and peripartum hysterectomy. No similar studies are done to assess additional surgical procedure as a independent risk factor for SSI. In our study it was significant in patients who had conservative surgery for PPH and peripartum hysterectomy this is due to prolonged duration of surgery, increased blood loss.

In the present study, almost all women received prophylactic antibiotic therapy, (intravenous 3rd generation Cephalosporins) during both elective and emergency LSCS. Therefore it was found that there was no much significant difference in the risk of SSI in both cases and control (Odd's Ratio:0.49 with p value 0.48). In other study it was found that prophylactic antibiotic therapy given 1hour before incision was protective for surgical site infection.[50,51] But the patients who received antibiotic after 1 hour of incision had 2.1 fold increased Odd's ratio of surgical site infection when compared with receipt of standard surgical prophylaxis.

# Chapter 7

# Conclusion

# CONCLUSION

The present study showed that the following independent risk factors were significantly associated with SSI following c-section, gestational diabetes (0dds ratio 5.4, p value 0.0001), HIV (p value 0.001), haemoglobin between 9-11gm% (odds ratio 5.17, p value of <0.0001), Unregistered antenatal cases (odds ration 4.7, p value of <0.0001), Primary cesarean section (odds ratio 5, p value 0.0001), pereclampsia/ eclampsia (odds ratio 7.9, p value 0.0001), induction of labour (odds ratio 8.46, 95% CI 4.2-11.7, p value of 0.0001), PROM (odds ratio 7.4, p value 0.0001), chorioamnionitis (odds ratio of 10.6%, 95% CI 2.1, p value 0.04), meconium stained liquor (odds ratio of 5.1, p value of 0.0001), emergency cesarean section (odds ratio 9.6, p value 0.0001), conservative surgery for PPH ( odds ratio of 14.2, p value of 0.01), was found to be significant.

# Chapter 8

# Summary

# SUMMARY

A case control study was performed in 1050 consenting women fulfilling eligibility criteria (who ha undergone lower segment cesarean section) at KLE's Dr Prabhakar Kore Charitable Hospital attached to JN Medical College, Belgaum.

In this study, a total of 1050 women, 350 were cases and 700 were controls. And they were assessed for patient, obstetric, operation related variables.

Gestational diabetes was found as one of the independent risk factor for SSI. In cases group 6.6% women (23 out of 350), and in control group 1.3% women(9 out pf 700) had GDM. (0dds ratio 5.4, p value 0.0001)

HIV in cases group were 1.7% ( 6 out of 350). In control there were no women with HIV. (p value 0.001)

Anemia was a significant independent risk factor for SSI (odds ratio of 5.17, p value of <0.0001).

SSI was more common in women who were unregistered women ( odd s ratio of 4.7, p value of <0.0001), when compared to registered women ( odds ratio of 0.3)

Women who had undergone primary cesarean section were more prone for SSI. In cases group 220 (62.9%) women had primary cesarean section. In

control group 177 (25.3%) women had primary cesarean section. (odds ratio of 5, p value of 0.0001)

PROM in cases group were 31(8.9%) women, 9(1.3%) women in control group. It was found that PROM was independent risk factor for SSI ( odds ratio of 7.4, p value of 0.0001).

Chorioamnionitis was found to be independent risk factor for SSI. In cases group 0.6% ( 2 out of 350), in control group there were no patients with chorioamnionitis. With odds ratio of 10.6, p value of 0.04.

Womens with preeclampsia/ eclampsia with odds ratio of 7.90, odds ratio of 0.0001. In cases group 30% ( 105 out of 350 ) and in control group 5.1% ( 36 out of 700) had preeclampsia/ eclampsia.

There was strong association between HELLP syndrome and SSI with p value of 0.0001.

Induction of labour is one the risk factor for surgical site infection ( odds ratio of 8.46, p value of 0.0001 ) Which is statistically significant. In cases group 12.9% ( 45 out of 350), in control group 1.7% ( 12 out 700 ).

In cases group 52( 14.9% ) women , in control group 23 ( 3.3%) women had meconium stained liquor. ( odd s ratio of 5.1 and p value of 0.0001) which was independent risk factor for SSI.

Women who had undergone emergency cesarean section had increasing risk for SSI, with odd s ratio of 9.6, p value of <0.0001.

In cases group 7 ( 9%) women and in control group only 1( 0.5%) had conservative surgery for PPH. With odds ratio of 14.2, p value of 0.01. In cases group 7(9%) women and in control group none had undergone peripartum hysterectomy. ( odds ratio of 30.54, p value of 0.01)

# Chapter 9

# Bibliography

# BIBLIOGRAPHY

1. Boyle A, Reddy UM. Epidemiology of cesarean delivery: the scope of the problem. Seminars in Perinatalogy 2012;36(5):308-14.
2. Torloni MR,Betran AP, Souza JP et al. classification of cesarean section: a systematic review. Plos one.2001;6(1):e14566.
3. Horan TC, Gaynes RP. Surveillance of nosocomial infections. In: Mayhall CG, ed. Hospital Epidemiology and Infection Control. Philadelphia: Lippincott Williams & Wilkins; 2004:1659 –1702.
4. Smaill F, Hofmeyr GJ. Antibiotic prophylaxis for cesarean section. Cochrane Database Syst Rev 2002;(3):CD000933.
5. Mitt P, Lang K, Peri A, Maimets M. Surgical-site infections following cesarean section in an Estonian university hospital: postdischarge surveillance and analysis of risk factors. Infect Control Hosp Epidemiol 2005;26:449–454.
6. Myles TD, Gooch J, Santolaya J. Obesity as an independent risk factor for infectious morbidity in patients who undergo cesarean delivery. Obstet Gynecol 2002;100(5 Pt 1):959 –964.
7. Beckman C, Ling F, Smith R, Barzansky B, Herbert W, Laube D. Postpartum infection. In: Beckman C, edr. Obstetrics and Gynecology. Philadelphia: Lippincott Williams & Wilkins; 2006:146 –52.
8. William O. Primary Caesarean Sections: Factors related to postpartum infection. J Obstet Gynaecol 1987;57:171-6.

9. Ezechi OC, Nwokoro CA, Kalu BK, Njokanma FO, Okeke GC. Caesarean section: Morbidity and mortality in a private hospital in Lagos Nigeria. Trop J Obstet Gynaecol 2002;19:97-100.
10. Killian CA, Graffunder EM, Vinciguerra TJ, Venezia RA. Risk factors for surgical-site infections following cesarean section. Infect Control Hosp Epidemiol 2001;22:613– 617.
11. DeFrances CJ, Hall MJ. 2005 National Hospital Discharge Survey. Adv Data 2007;385:1–19.
12. National Nosocomial Infections Surveillance System. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. Am J Infect Control 2004;32:470–485.
13. Fraenkel G. Some historical notes on infection in surgery. In: Watts MJ, Infection in surgery – basic and clinical aspects. New York; 1981. p. 27-37.
14. Egah DZ, Bello CS, Banwat EB, Allanana JA. Antimicrobial Sensitivity pattern for Staphyllococcus Aureus in Jos Nigeria. Niger J Med 1999;8:58-61.
15. Germ theory of disease. World of micrology and immunology.Ed.Brenda Wilmoth Lerner and K.Lee.Lerner.Detroit:Gale ,2007.Biography in Contxt.Web.14 April 2013 .
16. Larson E. Guideline for use of topical antimicrobial agents. Am J Infect Control 1988; 16: 253 – 66.
17. Wade JJ, Casewell MW. The evaluation of residual antimicrobial activity on hands and its clinical relevance. J Hosp Infect 1991; 18 (Suppl B): 23

18. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection. Infect Control Hosp Epidemiol; 1999. p.247-78.
19. Smith M, Enquist IF. A quantitative study of the impaired healing resulting from infection. Surg Gynecol Obstet 1967; 125: 965 – 73.
20. Rotter ML. Hygenic hand disinfection. Infect Control 1984; 5: 18 – 22, 197.
21. Larson E. Guideline for use of topical antimicrobial agents. Am J Infect Control 1988; 16: 253 – 66.
22. CDC/ NHSN Survillance Definitions for specific types of Infections. 2012.
23. CDC: NCHS data brief. Centres for disease control and prevention. Available from: URL: http:/www.cdc.gov/nchs/data/databriefs/db35.htm.
24. NIH Consensus and state of the silence statements. 2006;23(1):27-9.
25. Sufang G, Padmadas SS, Fengmin Z, Brown JJ, Stones RW. Delivery settings and Cesarean section rates in China. Bulletin of WHO 2007;85(10)
26. International Institute for Population Sciences (IIPS) and Macro International. 2007. National Family Health Survey (NFHS-3), 2005–06: India: Volume II Mumbai: IIPS.
27. Bhasin SK, Rajgoura OP, Sharma AK, Mehta M, Gupta N, Kumar S, et al. A high prevalence of cesarean section rate in East Delhi. Indian J. Community Med 2007;32:222-4
28. Sreevidya S, Sathiyasekarah BW. High cesarean rates in Madras (India): A population based cross sectional study. BJOG 2003;110:106-11

29. L.Raka, D.Zounman. Prevalence of nosocomial infections in high risk units in the university clinical center of Kosovo. Infect control and hospital epidemiol, 2006; 27(4):421-3.
30. Sue Ellen Sarasam, CNM, John P, et al. Management of wound complications from caesarean delivery. Obstetrical and Gynaecological Survey2005; 60: 462-63.
31. C Henry G, Garner WL: Inflammatory mediators in wound healing .Surg Clin North Am 83 : 483 – 507, 2003.
32. Cobb JP, Schmieg R, Hunt TK, Mundy LM. Inflammation, infection and antibodies. In: Way LW, Doherty, Current surgical diagnosis and treatment. Tata McGraw-Hill Companies; 2003. p. 112-41.
33. 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.
34. Edlich RF, Rodeheaver GT, Thacker JG. Technical factors in the prevention of wound infections. In: Howard RJ, Simmons RL, eds. Surgical Infectious Diseases. Norwalk, CT: Appleton & Lange; 1995:423– 463.
35. Smith M, Enquist IF. A quantitative study of the impaired healing resulting from infection. Surg Gynecol Obstet 1967; 125: 965 – 73.
36. Bucknall TE. Factors influencing wound complications: a clinical and experimental study. Ann R Coll Surg Engl 1982; 21: 71-8.
37. Brown RB, Bradley S, Opitz E, Cipriani D, Pieczarka R, Sands M. Surgical wound infections documented after hospital discharge. Am J Infect Control 1987; 15: 54 - 8.

38. Oslen .M.A.Butler.A.M et,al. Risk Factors for Surgical Site Infection After Low Transverse Cesarean Section. infection control and hospital epidemiology june 2008, vol. 29, no. 6
39. Rayfield EJ, Ault MJ, Keusch GT, Brothers MJ, Nechemias C, Smith H.Infection and diabetes: the case of glucose control. Am J Med 1982; 72: 439-50.
40. Latharam R, Lancaster AD, Covington JF, et al. The association of diabetes and glucose control with surgical site infections among cardiothoracic surgery patients. Infect Control Hosp Epidemiol 2001; 22: 607 – 612.
41. Sue Ellen Sarasam, CNM, John P, et al. Management of wound complications from caesarean delivery. Obstetrical and Gynaecological Survey2005; 60: 462-63.
42. Schneid-Kofman N, Sheiner E, Levy A, Holcberg G. Risk factors for wound infection following cesarean deliveries. *Int J Gynaecol Obstet* 2005;90:10 – 15.
43. Margaret A, Olsen, Phd, MPH; Butler, MS; Denise M. Risk factor Endometriosis after Low Transverse Cesarean Delivery. Infection Control of Hospital Epidemiology January 201., vol 31, No. 1.
44. Demisew Amenu, Tefera Belachew et al. Surgical Site infection rate and risk factor among obstetric cases of Jimma University specialized, Southwest Ethiopia. Original article.
45. Jido TA, Garba ID. Surgical-site infection following cesarean section in Kano, Nigeria. Ann Med Health Sci Res 2012;2:33-6.

46. AR Mahale et al.Caesarean section morbiditiy- a study of 1775 cases of abdominal wounds.Obs anb Gyanec.Today; 2008; 8:329-331.
47. *Demisew A. et al* surgical site infection rate and risk factors among obstetric cases of jimma university Specialized hospital, southwest ethiopia. Ethiop J Health Sci July 2011. Vol. 21, No. 2.
48. Tran TS, Jamulitrat S, Chongsuvivatwong V, et al. Risk factors for post cesarean surgical site infection. Obstet Gynecol 2000; 95: 367 – 371.
49. Vermillion ST, Lamoutte C, Soper DE, Verdeja A. Wound infection after cesarean: effect of subcutaneous tissue thickness. *Obstet Gynecol* 2000;95(6Pt 1):923–926.
50. Hopkins L Smaill F.Antibiotic prophylaxis regimens and drugs for caesarean.Section.Cochrane Database of Systematic Reviews1999, Issue2 Art.No:CD001136. DOI: 10.1002 /14651858.
51. Cooper NJ, Sutton AJ, Abrams KR. Decision analytical economic modeling within a Bayesian framework: application to prophylactic antibiotics use for caesarean section. Stat Methods Med Res 2002;11:491–512.

# *Annexures*

# Annexure I

# ANNEXURE I – ETHICAL CLEARANCE

K.L.E.SOCIETY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELGAUM-590010 (KARNATAKA-INDIA)
(Affiliated to KLE University, Belgaum)

Website: http/www.jnmc.edu
E-Mail : domejnmc@sancharnet.in
: jnmc@sancharnet.in

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

**Ref: MDC/DOME/** 895 **Date: 11/10/2012**

To,

J.N.Medical College,
BELGAUM.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled "**CASE CONTROL STUDY TO ASSESS INDEPENDENT RISK FACTORS INFLUENCING SURGICAL SITE INFECTION AFTER CESAREAN SECTION**" is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

**(Dr.Hema Dhumale)**
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belgaum.

**(Dr.Ganga Pilli)**
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belgaum.

# *Annexures*

# Annexure II

# ANNEXURE II

## INFORMED CONSENT DOCUMENT

**I.D.NO.** | | | | | |

**"Case control study to assess independent risk factors influencing surgical site infection after caesarean section"**

The study is conducted by Dr_____ Post graduate student in M.S Obstetrics and gynecology under guidance of Dr._____, J N Medical College, Belgaum.

Respected Sir/Madam, we invite you to participate in our study as, you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

**Purpose of the study:**

As there is increase in incidence of surgical site infection after cesarean section, though it is most commonly performed surgical procedure.This mainly increases the maternal morbidity and the cost. Hence it is necessary to assess independent risk factors influencing surgical site infection after cesarean section.The patient will be assessed on the $4^{th}$ post op day for any surgical site infection. All the patients who are included in this study will be requested to participate in this study during the period of one year.

**Procedure and treatment:**

Should you choose to participate, you will be asked to give a detailed history, undergo a physical examination, and consent to a few investigations like Hb%, wound discharge for culture sensitivity . The patients will be followed up till 1month after discharge from hospital for surgical site infection like wound gapping, discharge, induration. As such no much procedure is required and there s no harm to the patients.And line of treatment remains the same for all the patients who participate or who don't participate in the study.

**Risks and benefits:**

However all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding of risk factors influencing surgical site infection after cesarean section, and thus we will be able to provide improved patient care.

**Alternatives:**

If you decide not to participate in this study, you will still be receiving the usual standard care.

**Privacy and confidentiality:**

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

**Financial incentives:**

You shall not be receiving any payment or any financial incentives for participating in this study.

**Authorization to publish results:**

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

**Voluntary participation:**

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital.For any queries or any details you may contact Dr._____ Telephone No _____ or _____ Telephone No _____In case you need further information regarding your rights as a study participant, you may please contact Dr.Ganga.S.Pilli, chairman of the ethical committee, J N Medical College, Belgaum on telephone No.9448863866

**STATEMENT OF CONSENT:**

**I.D.NO:** | | | |

I Mr/Ms/Mrs

_____

volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

## INFORMED CONSENT DOCUMENT - KANNADA

ಅನುಬಂಧ 5
ಸಮ್ಮತಿ ಪತ್ರ

ಗುರುತಿನ ಸಂಖ್ಯೆ ☐☐☐

**"ಸಿಜೇರಿಯನ್ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರ ಸೋಂಕಿನ ಪ್ರಭಾವವನ್ನು ನಿರ್ಣಯಿಸಲು ಕೇಸ್ ನಿಯಂತ್ರಣ ಅಧ್ಯಯನ"**

ಈ ಅಧ್ಯಯನವನ್ನು [ ] ಪ್ರಾಧ್ಯಾಪಕರು, ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ವಿಭಾಗ, ಜವಾಹರಲಾಲ ನೆಹರೂ ವೈದ್ಯಕೀಯ ಮಹಾವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ ಇವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ [ ], ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ, ಪ್ರಸೂತಿ ಮತ್ತು ಸ್ತ್ರೀರೋಗ ವಿಭಾಗ ಇವರು ನಡೆಸುತ್ತಿದ್ದಾರೆ.

ಗೌರವಾನ್ವಿತ ಸರ್/ಮೇಡಂ, ನೀವು ನಮ್ಮ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಅರ್ಹರಿರುತ್ತೀರೆಂದು. ನಿಮ್ಮನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಆಹ್ವಾನಿಸುತ್ತಿದ್ದೇವೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಾವು ನಿಮಗೆ ನಿಮ್ಮ ವರ್ತಮಾನದ ತೊಂದರೆಗಳನ್ನು ಕುರಿತು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೆಳುತ್ತೆವೆ.

**ಅಧ್ಯಯನದ ಉದ್ದೇಶ:**
ಸಿಜೇರಿಯನ್ ಇದು ತುಂಬಾ ಸಾಧಾರಣ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯಾದರೂ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ಮಾಡಿದ ಜಾಗದಲ್ಲಿ ಉಂಟಾಗುವ ಸೋಂಕಿನ ಪ್ರಮಾಣದಲ್ಲಿ ಏರಿಕೆಯಾಗುತ್ತಿರುವ ಬಗ್ಗೆ ತಿಳಿದುಬಂದಿದೆ. ಇದರಿಂದ ತಾಯಿಗೆ ಹೆರಿಗೆ ಸಮಯದಲ್ಲಿ ಉಂಟಾಗುವ ತೊಂದರೆಗಳ ಪ್ರಮಾಣದಲ್ಲಿ ಏರಿಕೆಯಾಗಿದೆ. ಆದ್ದರಿಂದ ಆಗುವ ತೊಂದರೆಗಳನ್ನು ಒಂದೊಂದಾಗಿ ಮೌಲ್ಯಮಾಪನ ಮಾಡುವ ಅವಶ್ಯಕತೆಯಿದೆ. ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ಮಾಡಿದ ಜಾಗದಲ್ಲಿ ಉಂಟಾಗುವ ಸೋಂಕಿನಿಂದ ಆಗುವ ಯಾವುದೇ ತರಹದ ತೊಂದರೆಗಳನ್ನು ಕಂಡುಹಿಡಿಯಲು ರೋಗಿಯನ್ನು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯಾದ 4ನೇ ದಿನದಂದು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದು. ಈ ಅದ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಇಚ್ಚಿಸುವಲ್ಲಿ ರೋಗಿಗಳನ್ನು ಅಧ್ಯಯನದ ಒಂದು ವರ್ಷದ ಅವಧಿವರೆಗೆ ಭಾಗವಹಿಸಲು ಕೇಳಿಕೊಳ್ಳಲಾಗುವುದು.

**ವಿಧಾನ ಮತ್ತು ಚಿಕಿತ್ಸೆ:**
ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಇಚ್ಚಿಸಿರುವಿರಾದರೆ, ನಿಮಗೆ ನೀಮ್ಮ ಆರೋಗ್ಯದ ವಿಸ್ತಾರವಾದ ಮಾಹಿತಿಯನ್ನು ಕೇಳಲಾಗುವುದು.ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಪಡಿಸಲಾಗುವುದು, ಮತ್ತು ರಕ್ತ ಪರೀಕ್ಷೆ, ವುಂಡ್ ಡಿಸ್ಚಾರ್ಜ ಫಾರ್ ಕಲ್ಚರ್ ಸೆನ್ಸಿಟಿವಿಟಿ ಎಂತಹ ಪರೀಕ್ಷೆ ಮಾಡಿಸಲು ಸಮ್ಮತಿ ಕೇಳಲಾಗುವುದು. ರೋಗಿಯನ್ನು ಆಸ್ಪತ್ರೆಯಿಂದ ಡಿಸ್ಚಾರ್ಜ ಮಾಡಿದ ಒಂದು ತಿಂಗಳ ವರೆಗೆ ಫಾಲೋ ಅಪ್ ಮಾಡಲಾಗುವುದು. ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ಮಾಡಿದ ಜಾಗದಲ್ಲಿ ಉಂಟಾಗುವ ಸೋಂಕಿನಿಂದ ಉಂಟಾಗುವ ವುಂಡ್ ಗ್ಯಾಪಿಂಗ್, ಡಿಸ್ಚಾರ್ಜ ಮತ್ತು ಗಟ್ಟಿಯಾಗುವ ಕುರಿತು. ಇದನ್ನು ತಡೆಗಟ್ಟಲು ಕೆಲವು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ವಿಧಾನಗಳನ್ನು ಮಾಡಬೇಕಾಗಬಹುದು ಆದರೆ ಇದರಿಂದ ರೋಗಿಗೆ ಯಾವುದೇ ತರಹದ ತೊಂದರೆಗಳಾಗುವುದಿಲ್ಲ. ಉಳಿದ ಎಲ್ಲ ಚಿಕಿತ್ಸಾ ವಿಧಾನವು ಎಲ್ಲ ರೊಗಿಗಳಿಗೆ ಸಮವಾಗಿರುತ್ತದೆ. ಅಂದರೆ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವರು ಮತ್ತು ಭಾಗವಹಿಸಿದೆ ಇರುವವರು.

**ತೊಂದರೆಗಳು ಮತ್ತು ಲಾಭಗಳು:**
ನಿಮ್ಮ ಸುರಕ್ಷತೆಗಾಗಿ ಎಲ್ಲಾ ತರಹದ ಕ್ರಮಗಳನ್ನು ಮತ್ತು ಮುನ್ನೆಚ್ಚರಿಕೆಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಆದರೆ ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಆರೋಗ್ಯ ಸೇವೆಯನ್ನು ಒದುಗಿಸುವವರಿಗೆ ಸಿಜೇರಿಯನ್ ನಂತರ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಜಾಗದಲ್ಲಿ ಉಂಟಾಗುವ ಸೋಂಕಿನಿಂದ ಉಂಟಾಗುವ ತೊಂದರೆಗಳ ಬಗ್ಗೆ ತಿಳಿದುಕೊಳ್ಳಲು ಸಹಾಯವಾಗುತ್ತದೆ ಇದರಿಂದ ನಾವು ಉಳಿದ ರೋಗಿಗಳಿಗೆ ಗುಣಮಟ್ಟದ ಆರೋಗ್ಯ ಸೇವೆಯನ್ನು ಒದಗಿಸಲು ಸಹಾಯವಾಗುತ್ತದೆ.

**ಪರ್ಯಾಯ:**
ಒಂದು ವೇಳೆ ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಿದರೆ ನಿಮಗೆ ಸಿಗುವ ಗುಣಮಟ್ಟದ ಆರೈಕೆಯಲ್ಲಿ ಯಾವುದೇ ಬದಲಾವಣೆಯಾಗುವುದಿಲ್ಲ.

**ಗೌಪ್ಯತೆ:**
ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಒದಗಿಸುವ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು. ಅಧ್ಯಯನದ ಯಾವುದೇ ದಾಖಲೆಗಳಲ್ಲಿ ನಿಮ್ಮ ಪೂರ್ಣ ಹೆಸರು ಕಂಡು ಬರುವುದಿಲ್ಲ.

**ಹಣಕಾಸಿನ ಪ್ರೋತ್ಸಾಹ:**
ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ನಿಮಗೆ ಯಾವುದೇ ತರಹದ ವೇತನ ಹಾಗೂ ಹಣಕಾಸಿನ ನೆರವು ದೊರೆಯುವುದಿಲ್ಲ.

**ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸುವ ಅಧಿಕಾರ:**
ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ವೈದ್ಯಕೀಯ ಸಭೆಗಳಲ್ಲಿ ಹಂಚಿಕೊಳ್ಳಬಹುದು. ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಮುದ್ರಿಸಬಹುದು. ಮಾಹಿತಿ ಪ್ರಸ್ತುತ ಪಡಿಸುವಾಗ ನಿಮ್ಮ ಹೆಸರನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ.

**ಸ್ವಯಂಪ್ರೇರಿತವಾದ ಭಾಗವಹಿಸುವಿಕೆ:**
ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳದಿದ್ದರೂ, ನಿಮಗೆ ಸಿಗುವ ಸಾಮಾನ್ಯ ವೈದ್ಯಕೀಯ ಆರೈಕೆಯಲ್ಲಿ ಯಾವುದೇ ತೊಂದರೆಯಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮಲ್ಲಿ ಏನಾದರೂ ಪ್ರಶ್ನೆಗಳಿದ್ದಲ್ಲಿ ದೂರವಾಣಿ
ಅಥವಾ ಡಾ.ಹೇಮಾ ಧುಮಾಳೆ ದೂರವಾಣಿ
ಅವರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗಿಯಾಗುವಿಕೆಯ ಹಕ್ಕುಗಳ ಬಗೆಗೆ ನಿಮ್ಮಲ್ಲಿ ಪ್ರಶ್ನೆಗಳಿದ್ದರೆ ದಯವಿಟ್ಟು ಡಾ.ಗಂಗಾ ಎಸ. ಪಿಳ್ಳಿ, ಚೇರಮನ್ ಜೆ.ಎನ್.ಎಮ್.ಸಿ ಮಾನವ ಹಕ್ಕು ಮತ್ತು ನೀತಿ ಸಂಹಿತೆ ಸಮಿತಿ, ದೂರವಾಣಿ ಸಂಖ್ಯೆ: 9448863866 ಇವರನ್ನು ಸಂಪರ್ಕಿಸಿರಿ.

**ಸಮ್ಮತಿ ಪತ್ರ**

**ಗುರುತಿನ ಸಂಖ್ಯೆ** ☐☐☐

ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ

_____

ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಭಂದಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಅಥವಾ ನನಗಾಗಿ ಓದಲಾಗಿದೆ ಮತ್ತು ಅಧ್ಯಯನದಲ್ಲಿ ನಾನು ಭಾಗಿಯಾದರೆ ಏನು ಬೇಕೆಂಬುದರ ಬಗೆಗೆ ನಾನು ತಿಳಿದು ಕೊಂಡಿದ್ದೇನೆ. ನನ್ನ ಎಲ್ಲ ಪ್ರಶ್ನೆಗಳಿಗೆ ಮತ್ತು ಸಂಶಯಗಳಿಗೆ ನೀವು ಉತ್ತರಿಸಿದ್ದೀರಿ. ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಾನು ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ತೆಗೆದುಕೊಳ್ಳುವುದನ್ನು ಇಚ್ಚಿಸಿದಲ್ಲಿ ಯಾವುದೇ ಕಾರಣ ಕೊಡದೇ ನಾನು ಹಾಗೆ ಮಾಡಬಹುದೆಂಬುದನ್ನು ಮತ್ತು ಇದು ನನಗೆ ಲಭ್ಯವಾಗುವ ಆರೋಗ್ಯ ಕಾಳಜಿಗೆ ಯಾವುದೇ ಪರಿಣಾಮ ಬೀರುವದಿಲ್ಲ ಎಂಬುದರ ಬಗೆಗೆ ತಿಳಿದು ಕೊಂಡಿದ್ದೇನೆ. ನಾನು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪಿದ್ದೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಭಾಗವಹಿಸುವರ ಸಹಿ ಅಥವಾ ಹೆಬ್ಬೆಟ್ಟು ಗುರುತು:

ಸಾಕ್ಷಿ ಹೆಸರು:

ಸಾಕ್ಷಿ ಸಹಿ:

ಸಂಶೋಧಕರ ಸಹಿ:

ದಿನಾಂಕ:

ಮಹಿಳೆಯು ಅಪ್ರಾಪ್ತ ವಯಸ್ಸಿನವಳಾಗಿದ್ದರೆ (18 ವರ್ಷಕ್ಕಿಂತ ಕಡಿಮೆ ಇದ್ದರೆ) ಭಾಗವಹಿಸುವವರ ಬದಲು ಅವರ ಪೋಷಕರ ಸಹಿ ತೆಗೆದುಕೊಳ್ಳಬೇಕು.

**अनुबंध ५**

सूचित सहमति पत्रक

पहचान नं ☐☐☐

**"संक्रमण का शल्यक्रियात्मक प्रसव के बाद कारकों को आकलन करने के लिए स्वतंत्र जोखिम को प्रभावित स्थान की शल्य चिकित्सा मामला नियंत्रण अध्ययन कर रहे है"**

ये अध्ययन डॉ.हेमा धुमाळे प्राध्यापक प्रसूति और स्त्री रोग जवाहरलाल नेहरु वैद्यकीय महाविद्यालय बेळगाव के मार्गदर्शन मे डॉ.स्वरुपा बिरादार पदव्युत्तर छात्र एम एस प्रसूति और स्त्री रोग के द्वारा आयोजित किया जाता है.

आदरणीय महोदय/महोदया हम अपने अध्ययन में भाग लेने के लिए आपको आमंत्रित करते है आप उसी के लिए पात्र हैं. अध्ययन के दौरान आपको विस्तार से वर्तमान के शिकायतों के बारे में कुछ सवाल पूछे जायेंगे.

**अध्ययन का उद्देश्य:**

शल्यक्रियात्मक प्रसव के बाद घटना में चिकित्सा के संक्रमण के रुप मे वृद्धि हुई है. हालांकि शल्य प्रक्रिया यह सबसे अधिक किया जाता है. मातृ रुग्णता और लागत में यह मुख्य रूप से वृद्धि होति है. इसलिए यह जरूरी है की संक्रमण का शल्यक्रियात्मक प्रसव के बाद कारकों को आकलन करने के लिए स्वतंत्र जोखिम को प्रभावित स्थान की शल्य चिकित्सा कर रहे है. किसी भी शल्य संक्रमण के लिए मरीज को 4 दिन के बाद मूल्यांकन किया जाएगा. एक वर्ष की अवधि के दौरान इस अध्ययन में शामिल किए गए सभी रोगियों के लिए अनुरोध किया जाएगा.

**प्रक्रिया और उपचार:**

आप भाग लेने के लिए चुनना च्याहेंगे तो आप को एक विस्तृत इतिहास देने के लिए कहा जाएगा, एक शारीरिक परीक्षा से गुजरना और जांच एक करने के लिए सहमति जैसे कुछ हीमोग्लोबिन (HB%) संवर्धन संवेदनशीलता के लिए घाव मुक्ति.शल्य चिकित्सा के संक्रमण के लिए रोगियों को अस्पताल से छुट्टी के बाद 1 महीने तक पालन किया जाएगा जैसे भारी घाव, मुक्ति, कठोरता. ऐसी कोई ज्यादा प्रक्रिया आवश्यक है और रोगियों के लिए कोई बुराई नहीं है. और उपचार की लाइन सभी रोगियों के लिए ही रहता है अध्ययन में जो भाग लेने वाले है या जो न भाग लेने वाले है.

**जोखिम और लाभ:**

हालांकि सभी आवश्यक कदम और सावधानियों को आपकी सुरक्षा सुनिश्चित करने के लिए लीया जाएगा. आप इस शोध में भाग लेने का परिणाम संक्रमण का शल्यक्रियात्मक प्रसव के बाद कारकों को आकलन करने के लिए स्वतंत्र जोखिम को प्रभावित स्थान मे एक बेहतर समझ की दिशा स्वास्थ्य सेवा प्रदाताओं में मदद मिलेगी, और इस तरह हम रोगी की देखभाल प्रदान करने के सुधार में सक्षम हो जाएगा.

**विकल्प:**

इस अध्ययन में भाग न लेने का आप निर्णय लेते हैं तो आप को कभी भी सामान्य रोगी जैसी देखभाल प्राप्त हो जायेगी.

**एकांत और गोपनियता:**

आपकी गोपनीयता का सम्मान किया जाएगा और इस अध्ययन के दौरान आप के बारे में एकत्र सभी जानकारी को गोपनीय रखा जाएगा. आपकी पहचान गुप्त रहेगी.

**वित्तीय प्रोत्साहन:**

इस अध्ययन में भाग लेने के लिए आपको किसी भी भुगतान या वित्तीय प्रोत्साहन की प्राप्त नहीं की जाएगी.

**परिणामों को प्रकाशित करने के लिए प्राधिकरण:**

इस अध्ययन के परिणामों से वैज्ञानिक उद्देश्य के लिए या एक वैज्ञानिक समूह के समक्ष प्रस्तुत प्रकाशित किया जा सकता है. हालांकि हर समय आपकी पहचान गोपनीय रखी जायेगी.

**स्वैच्छिक भागीदारी:**

इस अध्ययन में आपकी भागीदारी स्वैच्छिक है. अपने भविष्य के संबंधों डॉक्टर या अस्पताल के साथ आपकी मौजूदा बीमारी की देखभाल को प्रभावित करेगा न तो आपका निर्णय भाग लेने या नहीं लेने के लिए किया जाएगा.

**सहमति के बयान**

पहचान नं: ☐☐☐

मै श्री/सुश्री/श्रीमती

_____

इस अध्ययन में भाग लेने के लिए मेरी स्वयंइच्छा और सहमति है. मैंने सहमति दस्तावेज़ पढ़ लिया है और ये मुझे मेरी देशी भाषा मे बताया गया है. मैं इस अध्ययन में भाग लेने के लिए स्वीकार करती हुं. इस अध्ययन के बारे में मुझे सभी जानकारी प्रदान की गई है और मैंने ये समझ लिया है. मुझे सवाल पूछने का मौका दिया गया है और उचित जवाब भी प्राप्त होंगे.

प्रतिभागियों का नांम:

हस्ताक्षर या प्रतिभागी के बाएं अंगूठे का निशान:

गवाह का नांम:

गवाह के हस्ताक्षर:

अन्वेषक के हस्ताक्षर:

तिथि:

अगर प्रतिभागिया १८ साल से कम उम्र का है तो माता पिता के हस्ताक्षर लेना जरुरी है.

परिशिष्ट ५

माहिती दिलेले संमती पत्रक

**ओळख** क्रमांक | | | |

**"गर्भीणी स्त्रियांमध्ये पोटाचा छेद करुन काढलेल्या बाळाच्या प्रसुती नंतर शल्यचिकित्सेचा झालेल्या जखमेचे स्वतंत्रपणे झालेले स्वरुप पहाणे"**

हा अभ्यासक्रम डॉ.स्वरुपा बिरादार पदव्युतर विद्यार्थी प्रसुतिशास्त्र आणि स्त्रीरोग तज्ञ या शास्त्राचा अभ्यासक्रम एम एस करत असताना मार्गदर्शक म्हणून डॉ.हेमा धुमाळे प्राध्यापक प्रसुतिशास्त्र आणि स्त्रीरोग तज्ञ जवाहरलाल नेहरु वैद्यकीय महाविद्यालय बेळगाव.

माननिय सर व श्रीमती, आपण एकच पात्र आहोत म्हणून आम्ही आमच्या सर्वेक्षणात तुम्हाला सहभागी होण्यासाठी आमंत्रित करीत आहोत.प्रस्तुत तपशील संबंधित तक्रारीबद्दल तुम्हाला या अभ्यासा दरम्यान आपण काही प्रश्न विचारले जातील.

**अभ्यासाचा उद्देश:**
पोटाचा छेद कींवा पोट फ़ाडुन मुल बाहेर आल्यानंतर शल्यचिकित्सेबद्दल खूप वेळा त्रास कींवा तक्ररी निर्माण होतात. कारण शल्यचिकित्सेच्या पद्धतीमुळे होऊ शकतात. मुख्यत्वे करुन मातेमध्ये रोगदोष वाढतो व पैशानी कींमत पण वाढते. त्या करता छेदचिकित्सा झाल्यानंतर आपणच जबाबदारी व धोका पत्करुन त्याचे निवारण करणे गरजेचे आहे. कोणत्याही शल्यचिकित्सेनंतर चौथ्या दिवशी नंतर रुग्णाला जखमेचा त्रास सुरु होतो. अशा प्रकारे सगळे रुग्ण ज्यांचे ऑपरेशन झालेले आहे त्याना या अभ्यास क्रमामध्ये
एक वर्षापर्यंत घेतले जाईल.

**प्रक्रिया आणि उपचार:**
तुंम्हाला अभ्यासक्रमामध्ये प्रवेश केल्यानंतर कांही प्रश्न व पूर्ण इतिहास विचारला जाईल (थोडक्यात पूर्ण माहिती)शारिरिक तपासणी व रक्त तपासणी कांही वेळा रक्ताचा लाल भाग कितपत आहे वगैरे, जखमे मधील स्त्राव व त्यात कोणते जंतु आहेत व त्याचा प्रतिकार कितपत आहे हे पाहीले जाईल. दवाखान्यामधुन रुग्ण घरी गेल्यानंतर एक महीन्यापर्यंत त्याचा पाठपुरावा केला जाईल. त्यामध्ये जखम कितापत भरुन आली आहे, त्यातील रक्तस्त्राव कमी कींवा जास्त आहे का? तसेच किती वेळ लागेल वगैरे सर्व पाहीले जाईल. यामध्ये फार काही करावे लागणार नाही तसेच त्रास पण कांही होणार नाही. तसेच औषध व गोळ्या यांची चिकित्सा सर्व रुग्णांना सारख्याच पद्धतीची असणार आहे. त्यामध्ये कांहीही बदल होणार नाही.

**जोखीम आणि फायदे:**

तुम्ही सुरक्षित रहाल याची पुर्ण काळजी व जबाबदारी घेतली जाईल. तुंम्ही या अभ्यासक्रमामध्ये भाग घेतल्यानंतर त्यामुळे जो परिणाम दिसुन येईल त्यामुळे संशोधकाला धोक्याच्या सुचनांचा आत्मविश्वास वाढेल व त्यामुळे पोट फाडुन मुल काढले तरी पुढे कांही पण त्रास होणार नाही याची खात्री होईल.

**पर्याय:**

जरी तुंम्ही या अभ्यासक्रमात भाग घेतला नाहीत तरीही तुमच्या चिकित्सेत कांहीही फरक होणार नाही. नेहमीप्रमाणे सर्व सुविधा तुम्हाला मिळतीलच.

**एकांत आणि गोपनियता:**

तुमच्याकडुन घेतलेल्या माहितीबद्दल, नांव इतिहास वगैरे याची पुर्णपणे गुप्तता पाळण्यात येईल. फक्त तुमचा ओळख क्रमांकच वापरला जाईल.

**आर्थिक लाभांश:**

यामध्ये भाग घेतल्यानंतर कोण्त्याही प्रकारचा पैशाचा मोबदला मिळणार नाही.

**परिणाम प्रकाशित करण्यासाठी अधिकृतता:**

संशोधनासाठी कोणत्याही पत्रिकेत तुमच्या अभ्यासक्रमाचा परिणाम दिला तरी फक्त ओळख क्रमांकामार्फतच प्रसिद्ध होईल.

**ऐच्छिक सहभाग:**

या अभ्यासक्रमामध्ये तुमचा सहभाग पूर्णपणे स्वईच्छेनीच असेल, जरी भाग घेतला अगर नाही तरीही तुमच्या चिकित्सेत व डॉक्टरच्या वागणूकीत कांहीही फरक पडणार नाही. या बद्दल तुंम्हाला कांहीही शंका किंवा प्रश्न असले तर तुम्ही खालील डॉक्टरांशी संपर्क साधु शकता. डॉ.स्वरुपा बिरादार फोन नं ८७४७८०६६४५ कींवा डॉ.हेमा धुमाळे फोन नं ९४८०५३७४९९. याच्या व्यतिरिक्त काही माहिती तुंम्हाला हवी असेल तर कृपया डॉ.गंगा एस पिल्ली, नैतिक समितीचे अध्यक्ष, जवाहरलाल नेहरु वैद्यकीय महाविद्यालय बेळगाव फोन नं ९४४८८६३८६६.

**सहमती विधान**

**ओळख क्रमांक** | | |

मी श्री/श्रीमती/सौ

_____

स्वइच्छेनी या अभ्यासक्रमात भाग घेत आहे. या अभ्यासक्रमाबद्दल ची पूर्ण माहीती माझ्या स्वत:च्या भाषेत मला सांगण्यात आली आहे व ती पूर्णपणे मला मान्य आहे. ही माहीती मला पूर्णपणे समजली आहे. यामध्ये मला प्रश्न विच्यारण्याची संधी दिली आहे व त्या प्रश्नांची उत्तरे पण समाधानकारक मिळाली आहेत.

सहभागीचे नांव:

सहभागीची स्वाक्षरी किंवा डाव्या हाताचा अंगठा

साक्षीदाराचे नांव:

साक्षीदाराची सही:

संशोधकाची सही:

तारीख:

जर का सहभागी १८ वर्षापेक्षा लहान असेल तर त्यांच्या पालकांची सही घेणे आवश्यक आहे.

# *Annexures*

# Annexure III

**PROFORMA**

**"CASE CONTROL STUDY TO ASSESS INDEPENDENT RISK FACTORS INFLUENCING SURGICAL SITE INFECTION AFTER CESAREAN SECTION"**

Sl. No. Date:

IP / OP No. Unit:

Patients Name

Age

Address

Contact No.: Socioeconomic status:

Obstetric history

G: P: L: A: D:

Menstrual history

LMP

EDD

Period of Gestation:

Examination findings:

GPE: Height(cms): Weight( Kgs): BMI:

Pulse: BP: RR: Temp:

Pallor ☐ Pedal edema ☐ Icterus ☐

**PATIENT RELATED VARIABLES:-**

Diabetes/GDM ☐ Yes ☐ No

HIV ☐ Positive ☐ Negative

Anemia ☐ Yes ☐ No

If yes pre op Hb% ☐

OBSTETRIC RELATEDVARIABLES

ANC ☐ Registered ☐ Unregistered

Previous Cesarean Section ☐ Yes ☐ No

If yes numbers

PROM ☐ Yes ☐ No

If yes duration since onset to c-section hrs

Chorioamnionitis ☐ Yes ☐ No

Ante partum haemorrhage ☐ Yes ☐ No

Preeclampsia /Eclampsia ☐ Yes ☐ No

HELLP Syndrome ☐ Yes ☐ No

DIC If present Post op

Induction of labour Yes No

If yes duration from induction to c-section hrs

Presence of meconium Yes No

OPERATION RELATED VARIABLES:

Nature of c-section Elective Emergengy

Type of skin incision Vertical Pfannential

Type Uterine incision Lower segment Inverted T-shaped

Exteriozation of the uterus done Not done

Any additional surgical procedure done Not done

If done specify

Prophylactic antibiotic therapy Yes No

Timing

Closure of subcutaneous fat layer Yes No

Nature of suture material used for

Skin closure Vicryl Monocryl Silk Linen

Cleaning the subcutaneous tissue

With betadine before closure of skin — Yes / No

Oxum used on subcutaneous tissue — Yes / No

Total duration of surgery — ____ hrs ____ min

Post op Hb% ____

C-section wound assessement on 4th post op day: Healthy

Purulent discharge

Induration(Swelling/tenderness/redness)

Gaping

Culture of the discharge

If positive organism — Positive / Negative

C/S report

At the time of discharge — Case / Control

4wks after c-section — Case / Control

# *Annexures*

## Annexure IV

# ANNEXURE IV – KEY TO MASTER CHART

| | | |
|---|---|---|
| GDM | - | gestational diabetes mellitus |
| HIV | - | Human immunuodeficiency viru |
| HELLP | - | Hemolysis, elevated liver enzymes, low platelet count |
| DIC | - | Disseminated intravascular coagulation |
| PROM | - | Premature rupture of membrane |
| C-section | - | Cesarean section |
| gms | - | Grams |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | Risk factors | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | Risk factors | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |

| Serial Number | In Patient Number | Age (Years) | Obstetric history | | | | | Period of gestation | Clinical examination | | | | | | | | Risk factors | | | | | | | |