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Original Research Article

A STUDY OF SURGICAL SITE INFECTIONS IN OG PRACTICE

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ABSTRACT

Background: Surgical site infections (SSIs) remain a major postoperative concern in obstetrics and gynecology, despite modern surgical techniques and the use of antibiotic prophylaxis. SSI is one of the most common complications encountered in surgery. These infections impose a heavy burden on both the patient and the healthcare system. **Objectives:** This study aimed to determine the incidence of SSI in patients who underwent caesarean sections and hysterectomies between January 2024 and December 2024 at PIMS Hospital(Walayar). Additionally, independent risk factors and management strategies were identified.

Materials and Methods: Patients who had caesarean sections, whether elective or emergency, were assessed for wound infections. Out of 72 deliveries, 42 (58.3%) were caesarean sections. Among them, 30 (71.4%) were elective cases, while 12 (28.5%) were emergency procedures. The age of patients who underwent caesarean section ranged from 19 to 37 years. In the same period, 140 hysterectomies were performed for various gynecological conditions, with patient ages ranging from 40 to 80 years. Among these, 103 (73.57%) were total abdominal hysterectomies (TAH), while 37 (26.4%) were vaginal hysterectomies (VH). All hysterectomies were planned procedures, performed with proper preoperative care, antibiotic coverage, blood transfusion availability and efficient nursing support.

Results: Of the 42 caesarean section cases, 5 patients developed SSI (11.9%). Among 140 hysterectomy cases, 15 patients (10.7%) developed wound infections. Thus the overall incidence of SSI for both procedures during the study period was observed in 20 patients (22.6%) within a 30-day postoperative observation period. The study identified higher SSI rates in caesarean patients with maternal age extremes (≤19 and >35 years), obesity, gestational diabetes mellitus (GDM) and high parity (>2). SSIs were more frequent in emergency caesarean cases and prolonged labor (>24 hours). Among hysterectomy patients, infections were more common in obese women, those with excessive blood loss and those with preoperative anemia or diabetes. The mode of hysterectomy (TAH vs. VH) did not show a significant difference in SSI rates. Laparoscopic hysterectomy was not performed.

Conclusion: Despite adequate perioperative and postoperative care SSIs remain a persistent challenge in surgical practice. Increased awareness among healthcare professionals regarding risk factors and early preventive measures can help reduce SSIs, ultimately improving surgical outcomes.

Keywords: Surgical site infections, Caesarean section, Hysterectomy, Risk factors, Treatment.

INTRODUCTION

Surgical site infections (SSIs) are among the most frequent nosocomial infections encountered in obstetrics and gynecology and frequently cause morbidity & mortality among hospital inpatients. Several factors affect the development of SSI.

Abdominal hysterectomy (TAH) was the preferred surgical approach for most uterine conditions although vaginal hysterectomy (VH) is associated with fewer complications, shorter hospital stays, faster recovery and lower costs.[1-3] VH is not suitable for patients with large uterine size, concurrent pelvic disease, or without uterine prolapse.[4] Globally, SSI rates range from 3% to 15%.[5-7] despite significant advancements in hygiene, prophylactic antibiotics, sterile surgical techniques and infection control measures.[8,9] Post-caesarean SSIs can lead to prolonged hospital stays, increased healthcare costs, maternal morbidity and, in severe cases, mortality.[10,11] Infections occur when bacteria enter the surgical incision site leading to localized or systemic infections. They typically develop within 30 days of surgery and may affect skin, soft tissue, or deeper structures.

Common risk factors for SSI include anemia, malnutrition, obesity, diabetes, prolonged surgery duration, excessive intraoperative blood loss, inadequate preoperative preparation and nosocomial infections. Surgical techniques, instrument contamination and improper wound care further increase the risk. The Centers for Disease Control and Prevention defines SSI as an infection occurring within 30 days from the operative procedure in the part of the body where surgery took place.[12] It categorizes SSIs into incisional SSIs and organ/space SSIs. Incisional SSI is further divided into superficial, involving the skin and subcutaneous tissue, and deep SSI, involving fascial and muscle layers.[12] Organ/space SSI affect any part of the anatomy that is manipulated during surgery, except for wall incisions.

Post Caesarean SSI can also be classified into three groups: (1) host-related factors, (2) pregnancy and intrapartum related and (3) procedure-related factors.[13] Host-related risk factors include extremes of maternal age, obesity, rural residence and preexisting conditions like diabetes or hypertension, previous caesarean delivery, recurrent pregnancy loss, twin pregnancy, preterm rupture of membranes, prolonged labor associated with frequent pelvic examinations and chorioamnionitis. Emergency caesarean sections and duration of surgeries exceeding 1 hour showed an increased incidence of SSI.[14-16] Preoperative assessment of hemoglobin and blood sugar levels is critical. Uncontrolled diabetes impairs immune function and delays wound healing.[16] Among hysterectomy patients SSIs were most frequently observed in obese, anemic, or malnourished women, especially those with diabetes or prolonged surgery duration. The type of hysterectomy (TAH vs. VH) did not significantly impact infection rates.

Pathogen analysis revealed that SSIs were commonly caused by Staphylococcus aureus responsible for 15%–20% of cases. Other isolated microorganisms included Gram-negative bacilli, coagulase-negative staphylococci, Enterococcus species and Escherichia coli.[17] Most SSIs are polymicrobial, involving both aerobic and anaerobic bacteria.[18-20]

Given the burden of SSIs, proper risk assessment, preventive measures and early management are crucial for improving post-surgical outcomes in caesarean sections and hysterectomies.

Aim & Objectives

To study the incidence, identify risk factors and treatment strategies for SSI over a period of 1 year from January to December 2024. A total of 182 patients were included in the study. Of them 42 were C-sections & 140 were hysterectomies. Among 42 C-sections, 30 were elective & 12 were emergency C-sections. Among 140 hysterectomies, 103 were abdominal hysterectomies & 37 were vaginal hysterectomies. All postoperative patients were screened & a clinical classification was done based on the risk factors for SSI. Surgical sites were examined & culture and sensitivity testing was done on infected wounds.

MATERIALS AND METHODS

Table 1 & 2 show the clinical classification of patients who underwent surgery (both c- sections and hysterectomies) from January 2024 to December 2024 based on the risk factors for SSI.

Table 1: Clinical classification of C-section pts. (total 42)

RISK FACTOR	NO. OF PATIENTS	PERCENTAGE
AGE		
<19 yrs	**2**	**4.76%**
20- 34 yrs	**35**	**83.33%**
>35 yrs	**6**	**14.2%**
RESIDENCE		
URBAN	**12**	**28.5%**
RURAL	**30**	**71.4%**
PARITY		
1- 2	**37**	**88.09%**
> 2	**5**	**11.90%**
GESTATIONAL AGE		
< 37 wks	**7**	**16.66%**
> 37 wks	**35**	**83.33%**
TYPE OF C-SECTION		
ELECTIVE		

EMERGENCY	**30** **12**	**71.4%** **28.5%**
DURATION OF SURGERY **< 1 hr** **> 1 hr**	**37** **5**	**88.09%** **11.90%**
BLEEDING **> 1000 ml**	**3**	**7.14%**
DURATION OF LABOR **< 24 hrs** **> 24 hrs**	**39** **3**	**92.85%** **7.14%**
PROM **NO** **YES**	**30** **12**	**71.4%** **28.5%**
OBESITY **NO** **YES**	**36** **6**	**85.71%** **14.28%**
GESTATIONAL DIABETES **NO** **YES**	**38** **4**	**90.47%** **9.52%**

Table 2: Clinical classification of hysterectomy PTs. (total 140)

RISK FACTOR	NO. OF PATIENTS	PERCENTAGE
AGE **40 -60 yrs** **> 60 yrs**	**120** **20**	**85.71%** **14.28%**
RESIDENCE **URBAN** **RURAL**	**87** **53**	**62.1%** **37.85%**
ANAEMIA **NO** **YES**	**117** **23**	**83.57%** **16.42%**
OBESITY **NO** **YES**	**126** **14**	**90%** **10%**
DIABETES MELLITUS **NO** **YES**	**114** **26**	**81.42%** **18.57%**
SURGERY BLOOD LOSS **> 1000 ml** **< 1000 ml**	**32** **108**	**22.85%** **77.14%**
DURATION OF SURGERY **> 2 hrs** **< 2 hrs**	**52** **88**	**37.14%** **62.85%**
DURATION OF HOSPITAL STAY **> 1 week** **< 1 week**	**81** **59**	**57.85%** **42.14%**
TYPE OF HYSTERECTOMY **TAH** **VH**	**103** **37**	**73.57%** **26.4%**

RESULTS

Table 3 shows that SSI was documented in 20 patients from a total of 182 (42 c-sections +180 hysterectomy) patients who were included in the study. ie. 5 c-section patients (11.9%) & 15 post hysterectomy patients (10.7%) had postoperative wound infections, thus a total SSI incidence of 22.6%. [Table 3]

Table 4 classifies SSI into 3 types namely superficial incisional SSI i; deep incisional SSI; & organ/space SSI. Majority of patients had superficial incisional SSI(14 patients)70%; 5 patients had deep incisional SSI (25%) & only 1 patient had organ/space SSI(5%). [Table 4]

Patterns of wound infection and common causative organisms are given in Table 5. Pus culture results showed that the common bacterial isolates were Staphylococcus aureus, Escherichia coli, Pseudomonas and Streptococcus. Staphylococcus aureus was the predominant organism in 15 patients with wound abscess; Escherichia coli was isolated in 2 patients who developed localised cellulitis; Streptococcus pyogenes was seen in 2 patients with spreading cellulitis & only one patient with multiple flora (E.coli+Klebsiella+Pseudomonas) developed wound dehiscence. [Table 5]

CLINICAL PRESENTATION OF SSI

Common local features of SSI include severe pain & tenderness over the wound, swelling, induration & warmth, shiny erythematous skin and purulent discharge from wound. Systemic features include pyrexia(>37.8 degree C), leukocytosis, tachycardia, tachypnoea, vomiting and anorexia. Most SSIs present from 3 to 14 days postoperatively. Gram-positive SSIs tend to arise early (3 to 6 days) characterised by prominent signs and symptoms. Wound becomes indurated with discharge being purulent and generous; systemic signs include low-grade fever and irritability.[21] Group A streptococcus SSI typically present dramatically 24 to 48 hours postoperatively with spreading cellulitis, distinct margins and lymphangitis. Discharge is scant and serous in nature. Systemic signs are prominent with high-grade fever and toxemia.[21] Gram-negative SSI arises 7 to 14 days postoperatively, usually after a patient is discharged from hospital. Local signs are less pronounced. Systemic signs are more prominent with high-grade fever and tachycardia. Wound drainage is sero-purulent and maybe foul smelling.

Table 6 shows the treatment of SSI. 15 patients with SSI were managed conservatively with daily dressing & antibiotics based on pus culture reports. 5 patients required surgical treatment. Broad spectrum antibiotics with anaerobic coverage was started immediately after pus culture reports. Around 90% of patients become afebrile 48 -72 hours after starting antibiotics. Once a patient becomes afebrile for 24 hours, intravenous antibiotics can be stopped & oral antibiotics started. Surgical treatment include exploration of wound under anaesthesia, complete drainage of wound, excising infected portions, saline irrigation of wound, secondary & deep tension suturing. [Table 6]

The first dressing was changed on the third postoperative day or earlier if a patient complained of severe pain over the operated site, fever or soakage of dressing. The time of appearance of wound infection was within three weeks following surgery. Wound dehiscence appeared in the second postoperative week. No patient developed septicemia or life threatening condition.

Table 3:

TYPE OF SURGERY	NO. OF PATIENTS	PERCENTAGE
CAESAREAN SECTION	5	11.90%
HYSTERECTOMY	15	10.70%

Table 4: Classification of SSI (total 20 pts.)

TYPE OF SSI	NO. OF PATIENTS	PERCENTAGE
SUPERFICIAL INCISIONAL	14	70 %
DEEP INCISIONAL	5	25 %
ORGAN/SPACE	1	5 %

Table 5: Type of SSI & pathogenic organisms

TYPE OF SSI	ORGANISMS	NO.OF PATIENTS.	PERCENTAGE
WOUND ABSCESS	STAPHYLOCOCCUS AUREUS	15	75%
LOCALISED CELLULITIS	ESCHERICHIA COLI	2	20%
SPREADING CELLULITIS	STREPTOCOCCUS PYOGENES	2	20%
WOUND DEHISCENCE	E.COLI + KLEBSIELLA + PSEUDOMONAS	1	5%

Table 6: Treatment of SSI

TREATMENT	NO. OF PTs.	PERCENTAGE
MEDICAL	15	75%
SURGICAL	5	25%

DISCUSSIONS

Despite advances in operative techniques and better understanding of pathogenesis of wound infection, SSI continues to be a major source of morbidity and mortality for postoperative patients. Effective management of surgical site infections (SSIs) involves antibiotics, wound exploration and debridement as soon as indicated.[22] Preventing SSIs requires multiple approaches beginning with recognizing risk factors and adhering to infection control measures such as proper hand hygiene, preoperative skin preparation, sterile surgical techniques and timely antibiotic prophylaxis.[23] The management of SSIs can be categorized into three phases: preoperative, intraoperative and postoperative care.

Preoperative assessment of haemoglobin & blood sugar levels is the cornerstone for prevention of SSIs. Perioperative glycemic control reduces the risk of SSIs.[23] In this study, four patients undergoing caesarean sections had gestational diabetes, accounting for 9.52 percent of cases. Among 140 hysterectomy patients, 26 had diabetes mellitus, contributing to 18.57 percent. Maintaining hemoglobin levels above 10 grams per deciliter before surgery is essential for minimizing SSIs.

The skin serves as a primary source of pathogens responsible for SSIs. Preoperative showering and skin preparation with antiseptic solutions has been proven to lower infection risk.[24] Shaving the surgical site has been associated with higher SSI rates due to microscopic skin abrasions caused by razors, making clipping a safer alternative.[25]

Administering prophylactic antibiotics reduces SSIs significantly. Cephalosporins and penicillins have demonstrated similar efficacy in lowering wound infections.[26] The timing of antibiotic administration plays an important role. Studies indicate that patients who received antibiotics before surgery had lower infectious morbidity compared to those who received them after cord clamping during caesarean sections.[27] No adverse neonatal effects were reported. In this study, all women undergoing caesarean sections and hysterectomies received a single dose of one gram intravenous cefazolin one hour before surgery. Antibiotics were continued for five to seven days postoperatively. Improper & prolonged use of antibiotics can lead to the development of resistant strains of microorganisms.

Intraoperative measures contribute to infection prevention. Proper surgical technique, timely blood transfusion, repeat antibiotic dosing for prolonged surgeries and optimal wound closure methods are essential. Prophylactic intraoperative wound irrigation (IOWI) of subcutaneous and deep soft tissue before skin closure using saline or antiseptic solutions is an easy and economical option to reduce SSI rates.[28,29] When subcutaneous wounds exceed two centimeters in depth, closing them carefully prevents complications. Suturing with non-absorbable material is preferred over staples due to the lower risk of infection. Application of topical antiseptics before wound closure can reduce SSI rates.

Postoperative wound care plays a key role in preventing SSIs. Routine wound inspection, cleansing with povidone-iodine or chlorhexidine-alcohol solutions and keeping the incision site open after dressing removal unless discharge is present help prevent secondary infections. When there is suppuration, stitches were removed, wound laid open, pus was taken for culture and sensitivity and antibiotics continued. Dressings were done twice daily with povidone soaked gauze packs. Antibiotics were changed based on sensitivity reports.

Timely detection and intervention prevent complications such as wound dehiscence, abscess formation and systemic infections. Signs like redness, swelling, warmth, pain and abnormal discharge require immediate medical attention. Once diagnosed, appropriate treatment including wound care, antibiotics, or drainage leads to better recovery outcomes.

CONCLUSION

SSIs account for 14 to 16 percent of all hospital-acquired infections. Identifying risk factors, implementing preventive strategies and ensuring early detection and treatment help reduce complications. A structured approach to infection prevention and management improves surgical outcomes and enhances patient recovery.

Conflict of Interest: I declare that there was no conflict of interest in the study.

REFERENCES

1. Byrnes JN, Occhino JA. Hysterectomy for benign conditions of the uterus: total vaginal hysterectomy. Obstet Gynecol Clin North Am. 2016; 43:441-462.
2. Byrnes JN, Trabuco EC. Evidence basis for hysterectomy. Obstet Gynecol Clin North Am. 2016; 43:495-515.
3. Moen M. Hysterectomy for benign conditions of the uterus: total abdominal hysterectomy. Obstet Gynecol Clin North Am. 2016; 43:431-440.
4. Chen I, Lisonkova S, Allaire S, Williams C, Yong P, Joseph KS. Routes of hysterectomy in women with benign uterine disease in the Vancouver Coastal Health and Providence Health Care regions: a retrospective cohort analysis. CMAJ Open. 2014;2:E273-E280.
5. Olsen MA, Butler AM, Willers DM, Devkota P, Gross GA, Fraser VJ. Risk factors for surgical site infection after low transverse caesarean section. Infect Control Hosp Epidemiol. 2008;29(6):477-484. Discussion 485-486.
6. Schneid-Kofman N, Sheiner E, Levy A, Holcberg G. Risk factors for wound infection following caesarean deliveries. Int J Gynecol Obstet. 2005;90(1):10-15.
7. Opoien HK, Valbo A, Grinde-Andersen A, Walberg M. Post-cesarean surgical site infections according to CDC standards: rates and risk factors. A prospective cohort study. Acta Obstet Gynecol.
8. Gibbs RS. Clinical risk factors for puerperal infection. Obstet Gynecol. 1980;55(Suppl 5):S178-S184.
9. Krieger Walfisch A, Sheiner E. Surgical site infection following caesarean deliveries: trends and risk factors. J Matern Fetal Neonatal Med. 2016;29(5):15.
10. Salim R, Braverman M, Teitler N, Berkovic I, Suliman A, Shalev E. Risk factors for infection following caesarean delivery: an interventional study. J Matern Neonatal Med. 2012;25(12):2708-2712.
11. Award S. Adherence to surgical care improvement project measures and postoperative surgical site infections. Surg Infect (Larchmt). 2012;13(4):234-237.
12. Horan TC, Gaynes RP, Martone WJ, Jarvis WR. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol. 1992;13(10):606-608.
13. Rubin RH. Surgical wound infection: epidemiology, pathogenesis, diagnosis and management. BMC Infect Dis. 2006; 6:171.
14. Killian CA, Graffunder EM, Vinciguerra TJ, Venezia RA. Risk factors for surgical-site infections following caesarean section. Infect Control Hosp Epidemiol. 2001;22(10):613-617.
15. Tran TS, Jamulitrat S, Chongsuvivatwong V, Geater A. Risk factors for post-cesarean surgical site infection. Obstet Gynecol. 2000;95(3):367-371.
16. Suonio S, Saarikoski S, Vohlonen L, Kauhanen O. Risk factors for fever, endometritis and wound infection after abdominal delivery. Int J Gynecol Obstet. 1989;29(2):135-142.
17. Korol E, Johnston K, Waser N. A systematic review of risk factors associated with surgical site infections among surgical patients. PLoS One. 2013;8(12):e83743.

18. Gur R, Duggal SD, Rongpharpi S. Post caesarean surgical site infections. Arch Clin Microbiol. 2015;6(1):16.
19. Gilstrap LC, Cunningham FG. The bacterial pathogenesis of infection following caesarean section. Obstet Gynecol. 1980;53(5):545-549.
20. Watts DH, Eschenbach DA, Kenny GE. Early postpartum endometritis: the role of bacteria, genital mycoplasmas and Chlamydia trachomatis. Obstet Gynecol. 1989;73(1):52-60.
21. Mollit D. Surgical Infections. In: Ziegler MA, et al. Operative Pediatric Surgery. McGraw-Hill, 2003, Pp 161-178.
22. Newton ER, Wallace PA. Effects of prophylactic antibiotics on endometrial flora in women with post-cesarean endometritis. Obstet Gynecol. 1998;92(2):226-226.
23. McKibben RA, Pitts SI, Suarez-Cuervo C, Peri TM, Bass EB. Practices to reduce surgical site infections among women undergoing caesarean sections: a review. Infect Control Hosp Epidemiol. 2015;36(8):915-921.
24. Fitzwater J, Tita AT. Prevention and management of caesarean wound infection. Obstet Gynecol Clin North Am. 2014; 41:671-689.
25. Tanner J, Norrie P, Melen K. Preoperative hair removal to reduce surgical site infection. Cochrane Database Syst Rev. 2011;11:CD004122.
26. Gyte GM, Dou L, Vazquez JC. Different classes of antibiotics given to women routinely for preventing infection at caesarean section. Cochrane Database Syst Rev. 2014;11:CD008726.
27. Mackeen AD, Packard RE, Ota E, Berghella V, Baxter JK. Timing of intravenous prophylactic antibiotics for preventing postpartum infectious morbidity in women undergoing caesarean delivery. Cochrane Database Syst Rev. 2014;12:CD009516.
28. Barnes S, Spencer M, Graham D, Johnson HB. Surgical wound irrigation: a call for evidence-based standardization of practice. Am j Infect Control.2014;42(5):525-9
29. Edmiston Jr CE, Leaper DJ. Intraoperative surgical irrigation of the surgical incision: what does the future hold-saline, antibiotic agents, or antiseptic agents? Surg Infect. 2016;17(6):656-64.