ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

http://rmcglobaljournal.com/

RMC Global Journal

ORIGINAL ARTICLE CLINICAL MICROBIOLOGY

Microbiological profile and antibiogram of surgical site infections

Sandeep Varma Manthena[1]

[1]Department of Medicine, Rangaraya Medical College, Kakinada, India

***Corresponding author:**
Sandeep Varma Manthena,
Department of Medicine,
Rangaraya Medical College,
Kakinada, India.
msvarmaauthorized@gmail.com

Received: 15 January 2025
Accepted: 28 April 2025
Published: 31 July 2025

DOI
10.25259/RMCGJ_1_2025

Quick Response Code:

ABSTRACT

Objectives: To evaluate the incidence, bacteriological profile, and antibiotic sensitivity patterns of surgical site infections (SSIs) in patients undergoing surgeries in the department of General Surgery at a tertiary care hospital.

Material and Methods: This prospective observational study was conducted over a two-month period from September 2022 to October 2022. Among 119 patients who underwent major surgeries, 15 clinically diagnosed cases of SSIs were included. Pus samples from infected wounds were collected and processed using standard microbiological techniques, including Gram staining, culture on selective media, biochemical characterization, and antibiotic susceptibility testing by the Kirby–Bauer disk diffusion method.

Results: The incidence of SSIs was found to be 12.6%. Culture positivity was observed in 66.7% of cases, with Gram-negative organisms predominating. *Escherichia coli* (*E. coli* – 50%) was the most common isolate, followed by *Klebsiella pneumoniae* (30%), *Pseudomonas aeruginosa* (10%), and coagulase-negative *Staphylococcus aureus* (10%). *E. coli* showed 100% resistance to cotrimoxazole, amoxicillin, clavulanic acid, and levofloxacin. High levels of resistance were also noted for multiple antibiotics among *K. pneumoniae* and *P. aeruginosa*. The majority of SSIs were observed in exploratory laparotomy procedures and were more frequent among diabetic and smoking patients.

Conclusion: SSIs remain a significant postoperative complication, with Gram-negative multidrug-resistant organisms being the predominant pathogens. The study underscores the need for regular surveillance of microbial patterns and resistance trends to inform hospital-specific antibiotic policies and promote rational antibiotic use. Identification of modifiable risk factors can aid in targeted preventive strategies to improve surgical outcomes.

Keywords: Antibacterial agents, *Escherichia coli*, Exploratory laparotomy, Infections, *Staphylococcus aureus*

INTRODUCTION

Infections that occur in the wound created by an invasive surgical procedure within 30 days or after a year in the case of an implant are generally referred to as surgical site infections (SSIs).[1] The development of a surgical site infection depends on contamination of the wound site at the end of a surgical procedure and specifically relates to the pathogenicity and inoculum of microorganisms present, balanced against the host's immune response. The microorganisms that cause SSIs are usually derived from the patient (endogenous infection), being present on their skin, or from an opened viscus. Exogenous infection occurs when microorganisms from instruments or the theater environment contaminate the site at operation, when microorganisms from the environment contaminate a traumatic wound, or when microorganisms gain access to the wound after surgery before the skin has sealed.[2] SSIs are one of the major postoperative complications that are responsible for increasing the length of hospital stay, treatment cost, significant morbidity, and mortality. SSIs are universal, and etiological agents may vary with geographical locations, between surgeons, between various procedures, from hospital to hospital, or even in different wards of the same hospital.[3] Despite the use of prophylactic antibiotics pre- and postoperatively and other preventive measures, SSIs remain a burden to postoperative patients. In addition, the irrational use of broad-spectrum antibiotics and resulting antimicrobial resistance (AMR) has further deteriorated the condition.[1,3] This use of antimicrobials increases selection pressure favoring the emergence of pathogenic drug-resistant bacteria. The emergence of resistance to antimicrobial agents is a global public health problem, particularly in pathogens causing nosocomial infections.[4] The emergence of multidrug-resistant strains in hospital environments, particularly in developing countries, poses a significant challenge to infection control and is associated with a high incidence of healthcare-associated infections and increased rates of antibiotic resistance.[5]

The World Health Organization (WHO) even declared AMR as one of the top 10 global public health threats plaguing humanity. This is because the efficacy of any antimicrobial agent is compromised by the potential emergence and development of tolerance or resistance to that compound from the time it is employed for the first time, leading to significant burdens on healthcare systems in terms of the management of infections. Researchers have estimated that 4.95 million deaths across the world are associated with bacterial AMR alone.[6]

India, being the world's leading consumer of antibiotics, is at increased risk of having one of the highest rates of resistance to antimicrobial agents. As per the Indian Council of Medical Research (ICMR) reports, 65,000 AMR cases were reported in 2020 from just 30 tertiary care centers in various metropolitan cities across India.[7]

Even though mechanisms of drug resistance development involve several genetic and molecular factors unique to different microbial cells, several factors create havens for the increased incidence, prevalence, and dissemination of resistant strains in the community.[8] These factors include overuse and misuse (especially with over-the-counter availability and irrational usage) of antimicrobial drugs, inadequate compliance with treatment regimens, and improper infection control measures in hospitals.[9]

Given the grave inadequacy of existing data on AMR patterns on SSIs, this study intends to discuss the prevalence of AMR and the profile of resistant strains to various antibiotics in Kakinada, India. This topic has been chosen to shed light on the burden of AMR and to draw conclusions to progress further in the management of diseases. The information obtained from this study allows a better understanding of the microbial etiology of SSIs in our hospital. Based on the results of this study, an initiative for establishing improved hospital antimicrobial policy and antimicrobial prescribing guidelines can be undertaken.

Aim of the study

To evaluate the incidence, bacteriological profile, and antibiotic sensitivity patterns of SSIs in patients undergoing surgery in the Department of General Surgery.

Objectives of the study

1. To identify the predominant microorganisms responsible for SSIs by analyzing the bacteriological profile of wound cultures.
2. To determine the antibiotic sensitivity and resistance patterns of the bacterial isolates.
3. To assess the risk factors contributing to the development of SSIs.
4. To calculate the incidence rate of SSIs among patients undergoing surgical procedures.

MATERIAL AND METHODS

The present study on the bacteriological profile of SSIs and antibiograms was carried out in the Department of Microbiology in a tertiary care hospital from September 2022 to October 2022. Out of the 119 major surgeries performed in 2 months in the General Surgery department, a total of 15 clinically diagnosed cases of SSIs were included in the study.

Inclusion criteria

1. All clinically diagnosed cases of SSIs, classified according to the Centers for Disease Control and Prevention guidelines (2017).
2. Patients of all ages (age groups ranging from 11–20 to 71–80 years).

Exclusion criteria

1. Patients with terminal illnesses, such as septic shock, advanced malignancies, multi-organ failure, or end-stage renal disease.
2. The patients who are visiting the hospital more than once during the study period.

Sample collection

The wounds were examined for signs and symptoms suggestive of SSIs during the postoperative period, and a sample was collected if the surgical site was found to be infected according to the criteria recommended by the Surgical Wound Infection Task Force. Before collecting the sample, careful cleaning of the infected surgical site was done using 70% ethyl alcohol, followed by 10% povidone-iodine, and allowed to remain for 2 minutes. Wearing sterile gloves, the wound margins were separated with the thumb and forefinger of one hand, and gentle pressure was applied with the other hand. The pus exudate was collected from the depth of the wound using two sterile cotton swabs, which were then placed into a sterile, dry, screw-capped test tube and immediately transported to the microbiology laboratory for further processing.

Processing of specimen

Direct microscopic examination of a Gram-stained smear

↓

Inoculation of samples into nutrient agar, blood agar, MacConkey agar, and selective media such as mannitol salt agar

↓

Preliminary identification of growth by colony morphology

↓

Biochemical tests for the characterization of species

↓

Antibiotic sensitivity test

The samples collected were subjected to direct microscopic examination and culture-based identification. For direct Gram staining, a smear was prepared using the first swab on a clean glass slide, stained by the Gram method, and examined microscopically for the presence of pus cells, Gram reaction, size, shape, arrangement, and types of organisms.

For aerobic culture, the second swab was inoculated using a sterile bacteriological loop onto nutrient agar, 5% sheep blood agar, and MacConkey agar. Plates were incubated at 37°C for 24–48 hours, with blood agar incubated under 5–10% CO_2 to support fastidious organisms.

After incubation, primary culture plates were examined for visible growth. Colony morphology, including size, shape, surface, elevation, margin, and pigmentation, was documented using a magnifying lens. Smears from isolated colonies were gram-stained and observed under oil immersion for cellular details. Nutrient agar colonies were assessed for their general appearance, while MacConkey agar helped identify lactose fermenters based on color change and typical appearances of gram-negative bacilli, coccobacilli, or pleomorphic forms. On blood agar, the type of hemolysis and colonial morphology were noted. Gram-positive cocci were identified based on their arrangement in pairs, chains, or clusters. Enterobacteriaceae typically produce large, grey, dry, or mucoid colonies. Organisms were grouped as gram-positive or gram-negative, cocci or bacilli, and identified using standard biochemical tests. For gram-negative bacilli, tests such as catalase, oxidase (dry filter paper method), indole, methyl red, Voges-Proskauer, citrate utilization, urease, triple sugar iron agar, and motility by hanging drop method were performed. For gram-positive organisms, catalase, coagulase, and bile esculin hydrolysis tests were utilized where appropriate.

For the catalase test, 1 ml of 3% hydrogen peroxide was placed in a sterile test tube, and colonies from nutrient agar were introduced using a sterile glass rod. Immediate and sustained effervescence indicated a positive reaction. The oxidase test was performed using filter paper impregnated with 1% tetramethyl para-phenylenediamine dihydrochloride; a deep blue color within 5–10 seconds signified a positive result. Motility was assessed using the hanging drop method.

Antibiotic susceptibility was determined using the modified Kirby-Bauer disk diffusion method. Two to three colonies from the primary culture were suspended in sterile saline, and turbidity was adjusted to 0.5 McFarland standard (prepared by adding 0.05 ml of 1% anhydrous $BaCl_2$ to 9.95 ml of 1% H_2SO_4). A sterile swab dipped in the standardized inoculum was streaked over Mueller-Hinton agar plates. After drying, antibiotic disks were placed using sterile forceps and incubated at 37°C for 16–18 hours. The diameter of inhibition zones was measured in millimeters and interpreted as sensitive or resistant.

RESULTS

In the present study, a total of 119 cases were operated on in the Department of General Surgery; among them, a total of 15 cases developed SSIs, accounting for a rate of 12.6% (1 in 8 patients who underwent surgery developed SSIs). Out of the 15 clinically diagnosed cases, nine (60%) were males and six (40%) were females, resulting in a male-to-female ratio of 1.5:1, indicating a higher prevalence of SSIs among males. The highest incidence of SSIs was observed in the 41–50 and 61–70 age groups, as depicted in Table 1 and Figure 1. SSIs were predominantly noted in patients with prolonged postoperative hospital stays, with a marked increase in incidence after the fourth postoperative day [Figure 2].

Pain, redness, and purulent discharge were the most frequently reported symptoms, occurring in 93.3%, 86.7%, and 66.7% of patients, respectively, as illustrated in Figure 3. A significant association was found between smoking and the development of SSIs, with 60% of cases occurring in smokers and 40% in non-smokers. Similarly, diabetic patients were more likely to develop SSIs compared to non-diabetics, as evidenced by data in Tables 2 and 3.

Regarding the type of surgery, exploratory laparotomy accounted for the highest proportion of SSIs (40%),

Table 1: Age distribution of patients with SSIs

Sr. no.	Age group in years	Male		Female		Total	
		No.	%	No.	%	No.	%
1	11–20	1	6.7	1	6.7	2	13.3
2	21–30	2	13.3	0	0	2	13.3
3	31–40	1	6.7	1	6.7	2	13.3
4	41–50	2	13.3	1	6.7	3	20
5	51–60	1	6.7	1	6.7	2	13.3
6	61–70	1	6.7	2	13.3	3	20
7	71 and above	1	6.7	0	0	1	6.7
Total		9	60	6	40	15	100

SSIs: Surgical site infections.

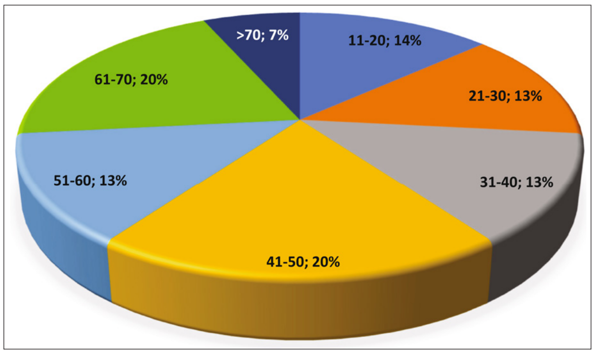

Figure 1: Age distribution of patients with surgical site infections (SSIs).

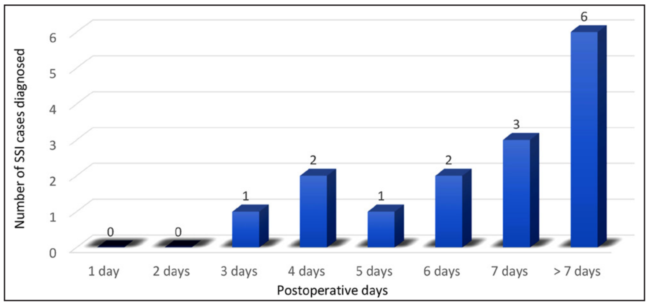

Figure 2: SSIs in relation to postoperative day of diagnosis. SSIs: Surgical site infections.

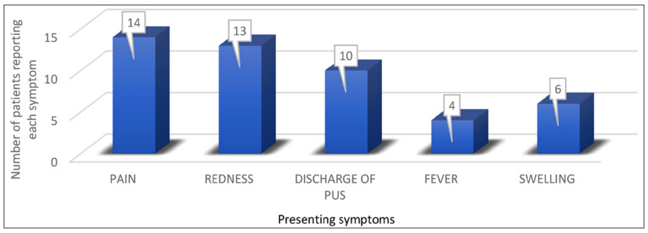

Figure 3: SSIs in relation to presenting symptoms. SSIs: Surgical site infections.

Table 2: SSIs in relation to smoking

Sr. no.	Type	SSIs developed	Percentage (%)
1	Smoker	9	60
2	Non-smoker	6	40

SSIs: Surgical site infections.

Table 3: SSIs in relation to diabetic patients

Sr. no.	Type	SSIs developed	Percentage (%)
1	Diabetic	10	66.7
2	Non-diabetic	5	33.3
Total		15	100

SSIs: Surgical site infections.

Table 4: Total culture positives among bacterial isolates

Sr. no.	Culture growth	No. of cases	Percentage (%)
1	Positive	10	66.7
2	Negative	5	33.3
Total cases		15	100

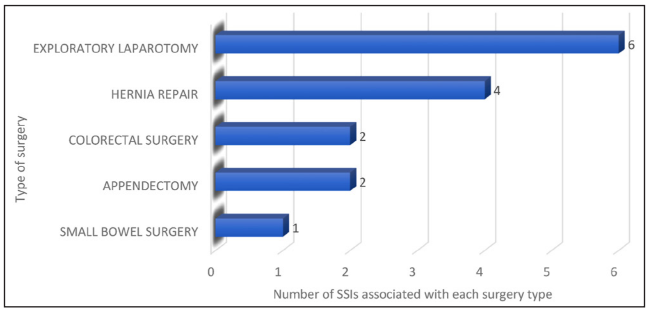

Figure 4: SSIs in relation to type of surgery. SSIs: Surgical site infections.

followed by hernia repair (26.7%), appendectomy (13.3%), and colorectal surgery (13.3%), as shown in Figure 4. Out of the 15 SSI cases, 10 (66.7%) yielded positive cultures, while 5 (33.3%) were culture negative [Table 4]. Among the culture-positive cases, Gram-negative organisms were predominant (90%), with only one isolate (10%) being Gram-positive. Escherichia coli was the most frequently isolated organism (50%), followed by Klebsiella pneumoniae (30%), Pseudomonas aeruginosa (10%), and coagulase-negative Staphylococcus aureus (10%), as illustrated in Figure 5.

All five *E. coli* isolates showed resistance to cotrimoxazole, amoxicillin, clavulanic acid, and levofloxacin, whereas

Table 5: Resistance pattern of bacterial isolates

Sr. no	Antibiotics	Organisms			
		Escherichia coli	*Klebsiella pneumoniae*	*Pseudomonas aeruginosa*	Coagulase negative *Staphylococcus aureus*
1	Amikacin	3	1	1	0
2	Cefepime	3	0	1	0
3	Cotrimoxazole	5	1	0	0
4	Cefoperazone	3	0	1	0
5	Sulbactam	3	0	1	0
6	Piperacillin	3	1	1	0
7	Tazobactam	3	1	1	0
8	Amoxycillin	5	1	0	0
9	Clavulanic acid	5	1	0	0
10	Gentamicin	3	1	1	0
11	Levofloxacin	5	1	1	0
12	Polymyxin B	3	0	0	0
13	Cefotaxime	2	0	0	0
14	Ampicillin	0	2	0	0
15	Ceftriaxone	0	1	0	0
16	Ceftazidime	0	0	1	0

three out of five isolates were resistant to amikacin, cefepime, cefoperazone, sulbactam, piperacillin, tazobactam, gentamicin, and polymyxin B. Two isolates are resistant to Cefotaxime. Out of three *K. pneumoniae isolates*, two isolates showed resistant to Ampicillin, and the remaining isolate is resistant to amikacin, cotrimoxazole, piperacillin, tazobactam, amoxicillin, clavulanic acid, gentamicin, levofloxacin, and ceftriaxone. Isolated *P. aeruginosa* is resistant to amikacin, cefepime, cefoperazone, sulbactam, piperacillin, tazobactam, gentamicin, levofloxacin, and ceftazidime. Surprisingly, isolated coagulase-negative *S. aureus* doesn't show any resistance pattern as summarized in Table 5 and Figure 6.

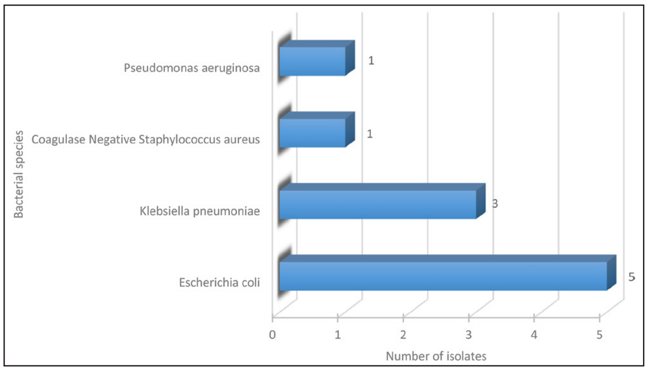

Figure 5: Spectrum of bacterial isolates in SSIs. SSIs: Surgical site infections.

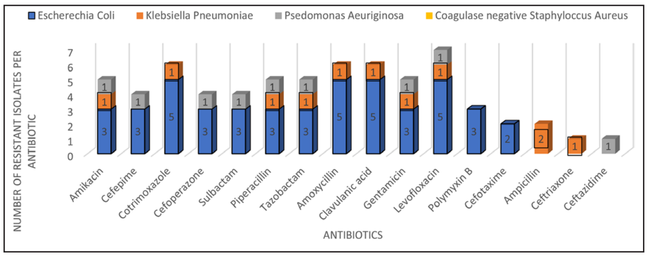

Figure 6: Resistance patterns of the bacterial isolates. SSIs: Surgical site infections.

DISCUSSION

In this study, patients were divided into seven age groups, with SSIs occurring more frequently in the 41–50 and 61–70 age groups. This aligns with Keith S. Kaye *et al.*, who reported higher SSI rates in patients over 65, likely due to factors like malnutrition, low immunity, and malabsorption.[10] Of the 15 SSI cases, 9 (60%) were males and 6 (40%) were females. The higher male infection rate aligns with Anand Saxena *et al.*,[11], possibly due to greater mobility and risk exposure. Bateman's principle may explain sex differences in immunity, suggesting females evolved stronger immunity for reproduction, while males face immune suppression due to reproductive trade-offs.[12]

A positive correlation was observed between SSI and postoperative hospital stay, with longer stays (>5 days) showing higher infection rates, consistent with William J. O'Brien *et al.*[13] SSIs affect both physical and psychological well-being, contributing to prolonged disability and depression post-discharge. These complications also lead to indirect financial burdens due to extended treatments and loss of productivity. Prolonged hospital stays, readmissions, and increased antibiotic use highlight the economic impact of SSIs. Current preventive strategies may inadequately protect high-risk vascular patients, stressing the need for future interventions backed by clinical trials.[14]

Smoking emerged as a major SSI risk factor, with 60% of infected patients being smokers. Lars Tue Sørensen *et al.* found higher postoperative complications in smokers and former smokers compared to never-smokers.[15] Smoking impairs wound healing via vasoconstriction, poor oxygenation, and collagen synthesis, and disrupts immune responses through neutrophil dysfunction. Other mechanisms include endothelial dysfunction, inflammation, and altered immune markers like reduced immunoglobulins and CD4/T-cell imbalance, leading to infections like pneumonia and bacteremia.[16]

Diabetes was another significant risk factor, with 66.7% of diabetic patients developing SSIs, consistent with Chen, Sam *et al.*[17] While hyperglycemia plays a role, diabetes may also reflect underlying vascular issues and impaired white blood cell function. Multiple factors beyond diabetes influence perioperative hyperglycemia and immune suppression. Though BMI had no impact in this study, confounder assessment was limited. Each study's adjusted estimates were used in the analysis, emphasizing the need for further research into diabetes severity and microbial contributors to infection.[18]

Our SSI rate is comparable to other developing nations but remains higher than rates in developed countries like the UK (3.1%) and the Netherlands (4.3%). Within Asia, our rates exceed some reports, though an SSI incidence of 20%–76.9% has been documented. The highest SSI rates were seen in exploratory laparotomies, especially colon surgeries. These rates surpass those in European and National Nosocomial Infections Surveillance studies, likely due to disparities in healthcare standards, infection control, and data collection practices.[19,20]

Common SSI symptoms included pain, redness, swelling, discharge, fever, and occasionally respiratory symptoms. These signs are consistent with classical inflammatory responses—erythema, purulent discharge, wound dehiscence, and delayed healing. Notably, wound photography has been shown to improve diagnostic accuracy in settings where in-person assessment is limited.[21,22]

Bacterial isolates included *E. coli*, *K. pneumoniae*, *P. aeruginosa*, and coagulase-negative *S. aureus*. This aligns with K. Vishnu Vardhana Rao *et al.*, who identified *E. coli* and *Klebsiella* as predominant pathogens in SSIs.[23] Most procedures were laparotomies with clean-contaminated wounds, often involving GI (gastrointestinal) tract spillage, explaining *E. coli* predominance. Fewer orthopedic surgeries likely account for the low number of gram-positive isolates.

S. aureus, though isolated less frequently, remains a major global cause of SSIs, with reported prevalence from 4.6% to 54.4%.[24] It is often endogenous but can be introduced via instruments or personnel. MRSA (Methicillin-resistant *Staphylococcus aureus*) remains a concern, with Vikrant Negi *et al.* reporting 15.7% resistance,[25] Aggarwal *et al.* 10%,[26] and Kownhar *et al.* 21.7%.[27] Eagye *et al.* and Kaye *et al.* reported even higher rates—45% and 58.2%, respectively.[28,29]

Gram-negative multidrug-resistant organisms were prevalent in SSIs. Rational antibiotic use and local antimicrobial surveillance are vital to control resistance. In this study, *E. coli* showed 100% resistance to cotrimoxazole, amoxicillin-clavulanic acid, and levofloxacin; 60% to amikacin, cefepime, cefoperazone-sulbactam, piperacillin-tazobactam, and others; and 40% to cefotaxime, echoing Ethiopian findings by Gemedo Misha *et al.*[30] *K. pneumoniae* showed 66.7% resistance to ampicillin and 33.3% to multiple other agents.[31] *P. aeruginosa* isolates were resistant to several antibiotics. Coagulase-negative *S. aureus* showed no resistance in this study.

Preventive strategies can significantly reduce SSIs and related costs. These include proper antimicrobial prophylaxis, normothermia, and euglycemia, along with adherence to Surgical Care Improvement Project protocols. Practices like razor shaving and antibiotic overuse may increase infection risks. Antiseptics, antimicrobial-coated sutures, and tissue adhesives show promise, though evidence remains limited. Teamwork among surgeons, OR staff, and pharmacists ensures effective implementation.[32]

Outcomes depend on surgical precision and hospital-level factors. Proper antibiotic use shortens hospital stays and curbs resistance. A London RCT (Royal Collection Trust) showed that prophylactic antibiotics administered within 2 hours pre-incision significantly reduced SSI risk.[33] Rising costs of antibiotic resistance and drug development are notable. Yet only half the reviewed studies disclosed funding or conflict of interest, despite the importance of transparency. Most did not report post-expiry price reductions of patented antibiotics, a crucial economic detail.[34]

While the findings of this study provide valuable insights into the risk factors and microbiological profile of SSIs, the relatively small sample size limits the generalizability of the results. Larger, multicentric studies are warranted to confirm these observations and provide more statistically robust conclusions. Additionally, certain confounding factors such as nutritional status, intraoperative variables, and adherence to infection control protocols could not be extensively evaluated due to data limitations.

CONCLUSION

In conclusion, this study highlights the significance of SSIs as a persistent postoperative complication, emphasizing the need for comprehensive surveillance and targeted prevention strategies. The identification of predominant bacterial isolates and their resistance profiles underscores the urgency of localized antibiograms to guide effective empirical therapy. The findings further reinforce the role of modifiable and non-modifiable risk factors in the development of SSIs, necessitating individualized patient risk assessment. By evaluating the incidence, microbial etiology, and antibiotic susceptibility patterns, this study provides critical insights to enhance infection control practices, optimize antimicrobial stewardship, and ultimately improve surgical outcomes in tertiary care settings.

Acknowledgments: The author gratefully acknowledges the support of the Department of Microbiology and the Department of General Surgery at Rangaraya Medical College for their guidance and assistance throughout the study. Sincere thanks to the institutional authorities for permitting the conduct of this research. The contributions of postgraduate residents and technical staff are also appreciated for their help during data collection.

Ethical approval: The research/study approved by the Institutional Review Board at Rangaraya Medical College, number IEC/RMC/2022/012, dated 26th November 2022.

Declaration of patients consent: Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol 1999;20:247–80.
2. Dancer SJ, Stewart M, Coulombe C, Gregori A, Virdi M. Surgical site infections linked to contaminated surgical instruments. J Hosp Infect 2012;81:231–8
3. World Health Organization. Global guidelines for the prevention of surgical site infection. Geneva: WHO; 2016. Available from: https://www.who.int/publications/i/item/9789241549882. [Last accessed 2024 May 07].
4. Anderson DJ, Sexton DJ, Kanafani ZA, Auten G, Kaye KS. Severe surgical site infection in community hospitals: Epidemiology, key procedures, and the changing prevalence of MRSA. Infect Control Hosp Epidemiol 2007;28:1047–53.
5. Seni J, Najjuka CF, Kateete DP, Makobore P, Jalali S, Kajumbula H, *et al*. Antimicrobial resistance in hospitalized surgical patients: a silently emerging public health concern in Uganda. BMC Res Notes 2013;6:298.
6. Murray CJ, Ikuta KS, Sharara F, Swetschinski L, Robles Aguilar G, Gray A, *et al*. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. Lancet 2022;399:629–55.
7. Walia K, Madhumathi J, Veeraraghavan B, Chakrabarti A, Kapil A, Ray P, *et al*. Establishing antimicrobial resistance surveillance & research network in India: Journey so far. Indian J Med Res 2019;149:164–79.
8. Valan Arasu M. Editorial on antimicrobial resistance and its prevalence. J Infect Public Health 2021;14:1739.
9. Hu F, Zhu D, Wang F, Wang M. Current status and trends of antibacterial resistance in China. Clin Infect Dis 2018;67:S128–34.
10. Kaye KS, Schmit K, Pieper C, Sloane R, Caughlan KF, Sexton DJ, *et al*. The effect of increasing age on the risk of surgical site infection. J Infect Dis 2005;191:1056–62.
11. Saxena A, Singh DMP, Brahmchari DS, Banerjee DM. Surgical site infection among postoperative patients of tertiary care centre in Central India – A prospective study. Asian J Biomed Pharm Sci 2013;3:41–4.
12. McKean KA, Nunney L. Bateman's principle and immunity: Phenotypically plastic reproductive strategies predict changes in immunological sex differences. Evolution 2005;59:1510–7.
13. O'Brien WJ, Gupta K, Itani KMF. Association of postoperative infection with risk of long-term infection and mortality. JAMA Surg 2020;155:61–68.
14. Totty JP, Moss JWE, Barker E, Mealing SJ, Posnett JW, Chetter IC, *et al*. The impact of surgical site infection on hospitalization, treatment costs, and health-related quality of life after vascular surgery. Int Wound J 2020;18:261–8.
15. Sørensen LT. Wound healing and infection in surgery: The clinical impact of smoking and smoking cessation: A systematic review and meta-analysis. Arch Surg 2012;147:373–83.
16. Durand F, Berthelot P, Cazorla C, Farizon F, Lucht F. Smoking is a risk factor of organ/space surgical site infection in orthopedic surgery with implant materials. Int Orthop 2013;37:723–7.
17. Chen S, Anderson MV, Cheng WK, Wongworawat MD. Diabetes associated with increased surgical site infections in spinal arthrodesis. Clin Orthop Relat Res 2009;467:1670–3.
18. Martin ET, Kaye KS, Knott C, Nguyen H, Santarossa M, Evans R, *et al.* Diabetes and risk of surgical site infection: A systematic review and meta-analysis. Infect Control Hosp Epidemiol 2015;37:88–99.
19. Brandt C, Sohr D, Behnke M, Daschner F, Ruden H, Gastmeier P. Reduction of surgical site infection rates associated with active surveillance. Infect Control Hosp Epidemiol 2006;27:1347–51.

20. Prospero E, Cavicchi A, Bacelli S, Barbadoro P, Tantucci L, D'Errico. Surveillance for surgical site infection after hospital discharge: A surgical procedure-specific perspective. Infect Control Hosp Epidemiol 2006;27:1313–7.
21. Skube SJ, Hu Z, Arsoniadis EG, Simon GJ, Wick EC, Ko CY, *et al*. Characterizing surgical site infection signals in clinical notes. Stud Health Technol Inform 2017;245:955–9.
22. Sanger PC, Simianu VV, Gaskill CE, Armstrong CA, Hartzler AL, Lordon RJ, *et al*. Diagnosing surgical site infection using wound photography: A scenario-based study. J Am Coll Surg 2017;224:8–15.e1.
23. Rao VV, Pradeep MSS. A study on surgical site infections, their bacteriological profile and antimicrobial susceptibility pattern. IP Int J Med Microbiol Trop Dis 2019;5:9–13.
24. Chakarborty SP, Mahapatra SK, Bal M, Roy S. Isolation and identification of vancomycin-resistant *Staphylococcus aureus* from postoperative pus sample. Al Ameen J Med Sci 2011;4:152–68.
25. Negi V. Bacteriological profile of surgical site infections and their antibiogram: A study from resource-constrained rural setting of Uttarakhand, India. J Clin Diagn Res 2015;9:DC17–20.
26. Aggarwal A, Khanna S, Arora U, Devi P. Correlation of beta-lactamase production/methicillin resistance and phage pattern of *Staphylococcus aureus*. Indian J Med Sci 2001;55:253–6.
27. Kownhar H, Shankar EM, Vignesh R, Sekar R, Velu V, Rao UA. High isolation rate of *Staphylococcus aureus* from surgical site infections in an Indian hospital. J Antimicrob Chemother 2008;61:758–60.
28. Eagye KJ, Kim A, Laohavaleeson S, Kuti JL, Nicolau DP. Surgical site infections: Does inadequate antibiotic therapy affect patient outcomes? Surg Infect (Larchmt) 2009;10:323–31.
29. Kaye KS, Anderson DJ, Sloane R, Chen LF, Choi Y, Link K, *et al*. The effect of surgical site infection on older operative patients. J Am Geriatr Soc 2009;57:46–54.
30. Misha G, Chelkeba L, Melaku T. Bacterial profile and antimicrobial susceptibility patterns among patients diagnosed with surgical site infection at a tertiary hospital in Ethiopia: a prospective cohort study. Ann Clin Microbiol Antimicrob 2021;20:33.
31. Herman AM, Massenga G, Chilonga KS, Philemon RN, Katundu D, Lugakingira A, *et al*. Surgical site infection: The rate and antimicrobial sensitivity pattern in electively operated surgical and gynecological patients at Kilimanjaro Christian Medical Centre, Tanzania. J Surg Surg Res 2017;3:1–5.
32. Reichman DE, Greenberg JA. Reducing surgical site infections: A review. Rev Obstet Gynecol 2009;2:212–21.
33. Penel N, Lefebvre J-L, Cazin JL, Clisant S, Neu J-C, Dervaux B, *et al*. Additional direct medical costs associated with nosocomial infections after head and neck cancer surgery: a hospital-perspective analysis. Int J Oral Maxillofac Surg 2008;37:135–9.
34. Purba AKR, Setiawan D, Bathoorn E, Postma MJ, Dik J-WH, Friedrich AW. Prevention of surgical site infections: A systematic review of cost analyses in the use of prophylactic antibiotics. Front Public Health 2018;9:776.

How to cite this article: Manthena SV. Microbiological profile and antibiogram of surgical site infections. RMC Glob J. 2025;1:62–69. doi: 10.25259/RMCGJ_1_2025