International Surgery Journal
Kumar S et al. Int Surg J. 2025 Oct;12(10):1889-1896
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20253056

Review Article

Surgical site infections in GI surgery: update on bacteriology, antibiotic resistance, and ERAS integration

Supreet Kumar*, Suryalok Pratap Shah, Sonam Gupta, Vivek Tandon, Deepak Govil

Department of Surgical Gastroenterology and GI oncology, Indraprastha Apollo Hospital, New Delhi, India

Received: 12 August 2025
Accepted: 16 September 2025

***Correspondence:**
Dr. Supreet Kumar,
E-mail: Supreet.mvj@gmail.com

ABSTRACT

Surgical site infections (SSIs) remain one of the most significant complications following gastrointestinal (GI) surgery, contributing to increased morbidity, prolonged hospitalization, and heightened healthcare costs. Despite major strides in perioperative care, SSI rates in GI surgery still range between 10–30%, particularly in high-risk emergency settings. The microbial landscape of SSIs is shifting, driven by rising antimicrobial resistance and regional variability in bacteriology. Multidrug-resistant (MDR) organisms- especially extended-spectrum β-lactamase (ESBL)-producing *Enterobacteriaceae*, methicillin-resistant *Staphylococcus aureus* (MRSA), and carbapenem-resistant *Klebsiella pneumoniae*- have emerged as formidable threats. Parallelly, enhanced recovery after surgery (ERAS) pathways are gaining traction as comprehensive perioperative bundles that reduce SSI incidence through patient optimization, minimally invasive approaches, targeted prophylaxis, and early mobilization. This review consolidates evidence from 2015-2025 to present a comprehensive overview of: (a) the evolving microbiology and antibiotic resistance trends in GI SSIs; (b) contemporary antimicrobial prophylaxis strategies and stewardship programs; (c) the impact of ERAS protocols in mitigating SSI risks; and (d) future directions including precision SSI prevention, AI-assisted risk stratification, and rapid diagnostics. Effective SSI prevention in GI surgery necessitates a multidisciplinary approach rooted in local bacteriology, rational antibiotic use, and evidence-based perioperative care. Integrating ERAS with antimicrobial stewardship and personalized risk models may herald a new era in surgical infection control.

Keywords: Gastrointestinal surgery, Surgical site infection, Antimicrobial resistance, ERAS, SSI prevention, Multidrug-resistant organisms, Bacteriology

INTRODUCTION

Surgical site infections (SSIs) continue to represent one of the most formidable challenges in gastrointestinal (GI) surgery, accounting for a substantial proportion of postoperative complications, reoperations, and mortality worldwide.[1] Defined as infections occurring at or near the surgical incision within 30 days postoperatively- or up to one year in cases involving prosthetic implants- SSIs exert a profound impact on patient recovery, resource utilization, and long-term surgical outcomes.[2]

Despite notable advancements in sterile technique, operating room protocols, and perioperative care, the reported incidence of SSIs in GI surgery remains unacceptably high, ranging from 10-30% in elective cases and climbing even higher in emergency or contaminated procedures.[3,4] These figures are compounded by emerging evidence from low- and middle-income countries (LMICs), where resource constraints, limited infection surveillance, and inconsistent antibiotic practices contribute to even greater risk.[5]

The pathogenesis of SSIs in GI surgery is multifactorial- often originating from a complex interplay between host

factors, intraoperative contamination, and perioperative care gaps. Unlike clean surgical fields, GI procedures inherently involve exposure to endogenous flora of the alimentary tract, especially polymicrobial combinations of Gram-negative bacilli, anaerobes, and gram-positive cocci.[6] Add to this the threat of hospital-acquired pathogens and antimicrobial-resistant strains, and the landscape becomes significantly more complex.[7]

The burden of SSIs extends beyond clinical morbidity. Patients experiencing SSIs face significantly prolonged hospital stays, increased need for advanced wound care, delayed adjuvant treatments (especially in oncologic surgery), and up to five-fold increases in readmission risk.[8] Economically, SSIs are estimated to account for 20-30% of total postoperative expenditures in GI units.[9]

In response to this ongoing crisis, several strategic advancements have emerged in recent years. Most notably, the enhanced recovery after surgery (ERAS) paradigm has provided a robust, evidence-based framework to optimize perioperative care and reduce infectious complications. When combined with antimicrobial stewardship and microbiological surveillance, ERAS offers a promising model to reduce SSI rates while preserving antibiotic efficacy.[10] Given the rising global prevalence of antimicrobial resistance, the emergence of novel biofilm-forming pathogens, and the integration of AI-driven perioperative strategies, this review is both timely and imperative for guiding surgical infection control in the modern era.

This review consolidates key developments in the epidemiology, bacteriology, resistance trends, and preventive strategies related to SSIs in GI surgery, with an emphasis on the transformative potential of ERAS integration.

EPIDEMIOLOGY AND CLINICAL IMPACT OF SSIs IN GI SURGERY

SSIs following GI surgery continue to represent a formidable clinical and public health concern across the globe. Despite the evolution of sterile techniques and infection control measures, multicentre surveillance data from both high- and low-income nations report an overall SSI incidence of 7% to 24% in elective GI procedures, with significantly higher rates in emergency settings and among high-risk patient populations.[11] Recent WHO global reports confirm that SSI incidence remains disproportionately high in LMICs, often exceeding 30% in high-risk abdominal procedures due to infrastructural constraints and delayed antimicrobial initiation. Abdominal surgeries, particularly colorectal resections and exploratory laparotomies, remain disproportionately affected due to the inherent contamination risks of the GI tract.[12]

A comparative snapshot of SSI incidence across geographic regions is summarized in Table 1, highlighting the disparity between high-income countries and LMICs. The implications of SSIs are far-reaching. Studies have demonstrated a two- to five-fold increase in postoperative mortality among patients developing SSIs, particularly when infections progress to organ-space involvement.[13] Hospital stays are commonly prolonged by an additional 7-11 days, and there is a marked rise in unplanned readmissions and the need for reoperations.[14] These infections frequently delay initiation of adjuvant chemotherapy in GI malignancies and increase the likelihood of stoma creation, wound dehiscence, or incisional hernias, thereby negatively impacting long-term functional and oncologic outcomes.[15]

Table 1: Summary of global SSI incidence (2015-2025).

Region/country	SSI incidence in GI surgery (%)
India (Cureus, ISJ)[3,4,12]	18-32
USA (CDC, ACS)[1,2,14]	5-15
Europe (ECDC, BJS)[13,15]	10-20
Africa (WHO Report)[40,46]	25-35
Southeast Asia (Bangladesh, Nepal)[5,11]	20-28

From a resource utilization perspective, the burden is equally grave. Surgical site infections are responsible for approximately 20-30% of total postoperative care costs in GI surgery, driven by prolonged hospital stays, re-interventions, advanced wound care needs, and antimicrobial therapy.[16] This burden is even more pronounced in resource-constrained environments where wound management technologies and targeted antibiotics are either unavailable or unaffordable. Host-related risk factors for SSI development include older age, malnutrition, obesity, diabetes mellitus, immunosuppression, and uncontrolled remote infections.[17]

Procedural determinants such as longer operative times, high blood loss, poor skin antisepsis, use of contaminated wound class (class III or IV), and inadequate antibiotic prophylaxis further amplify the risk. Institutional practices- particularly non-adherence to SSI prevention bundles and lack of postoperative surveillance- can significantly alter infection profiles between centres.

Despite these challenges, encouraging trends have emerged from healthcare systems that have institutionalized infection prevention strategies. Standardized perioperative care bundles, strict wound classification adherence, and multidisciplinary audits have resulted in measurable declines in SSI rates over the past decade.[18] However, the rising tide of multidrug-resistant organisms in postoperative infections has simultaneously complicated management, underscoring the critical need for sustained epidemiological monitoring and data-driven local policy formulation.

MICROBIOLOGY OF SSIs IN GI SURGERY

The microbiological landscape of SSIs in GI surgery is uniquely complex, shaped by the diversity of enteric flora and influenced by institutional infection control practices and antibiotic use patterns. Unlike clean surgical fields, GI procedures inherently expose patients to a rich array of commensal and pathogenic microorganisms, predisposing to polymicrobial infections and facilitating the emergence of multidrug-resistant strains.[19]

Traditional pathogens

Historically, SSIs in GI surgery have been dominated by Gram-negative bacilli, especially *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*, which originate from intraoperative spillage of bowel contents or translocation from the gut lumen.[20] Anaerobes such as *Bacteroides fragilis* and *Clostridium* species are also frequently implicated, particularly in colorectal procedures or in cases of deep organ-space infections.[21] Gram-positive organisms like *Staphylococcus aureus* (including methicillin-resistant *S. aureus* or MRSA) and *Enterococcus spp* are typically isolated from superficial incisional infections or upper GI surgeries.[22]

Emerging and multidrug-resistant organisms

A rapidly evolving concern is the emergence of multidrug-resistant (MDR) pathogens in postoperative SSIs. Extended-spectrum β-lactamase (ESBL)-producing *E. coli* and *K. pneumoniae* are increasingly prevalent, often associated with prior hospital exposure or antibiotic prophylaxis.[23] Carbapenem-resistant Enterobacterales (CRE) and *Acinetobacter baumannii* pose critical threats, particularly in ICUs and oncology units.[24] The detection of vancomycin-resistant *Enterococci* (VRE) and linezolid-resistant staphylococci, while still sporadic, underscores the expanding antimicrobial resistance frontier in surgical infections.[25]

Polymicrobial infections and biofilms

GI SSIs frequently harbour polymicrobial flora, with combinations of aerobic and anaerobic organisms complicating both diagnosis and empirical therapy. Additionally, biofilm-forming bacteria such as *S. aureus* and *Enterococcus faecalis* can colonize surgical implants or devitalized tissue, rendering standard antimicrobial regimens ineffective and promoting chronic wound infections.[19,22] Biofilm formation not only facilitates immune evasion but also confers intrinsic resistance to multiple antibiotic classes, often resulting in delayed healing, persistent wound infection, and failure of empirical regimens.[22]

Geographic and institutional variation

The microbial profile of GI SSIs varies markedly by geography, institutional practices, and patient population. Data from low- and middle-income countries suggest a higher burden of resistant Gram-negative organisms and limited access to targeted antimicrobials, thereby influencing local empiric protocols.[23] Hence, continuous local microbiological surveillance is indispensable to guide prophylaxis and early therapy.

Table 2 categorizes the predominant pathogens encountered in GI SSIs according to surgical region, guiding anatomical site- based prophylaxis and empiric therapy.

Table 2: Summary of global SSI incidence (2015-2025).

Surgical sites	Common organisms
Esophagus and stomach	*Staphylococcus aureus, Streptococci, Candida spp.*
Small bowel	*E. coli, Klebsiella spp., Anaerobes*
Colon and rectum	*E. coli, Bacteroides fragilis, Enterococcus spp.*
Upper abdomen (e.g. hepatobiliary)	*Pseudomonas, Klebsiella, MRSA*
Emergency GI surgery	Highly polymicrobial + MDROs (*CRE, VRE, Candida*)

In summary, the bacteriology of SSIs in GI surgery is no longer limited to classical flora. Rising antimicrobial resistance, the presence of biofilms, and the diversity of pathogens involved necessitate vigilant microbiological profiling, infection control, and data-driven antimicrobial stewardship.

ANTIBIOTIC RESISTANCE IN GI SSIs- TRENDS AND CHALLENGES

Antibiotic resistance has emerged as a defining challenge in the management of SSIs following GI procedures. The increasing prevalence of MDROs has drastically altered the empiric and definitive treatment landscape, rendering traditional antibiotic regimens ineffective and contributing to greater morbidity, prolonged hospitalizations, and higher mortality rates.[26]

The rise of ESBL and CRE organisms

ESBL-producing *Escherichia coli* and *Klebsiella pneumoniae* have become the predominant resistant pathogens in GI SSIs. These organisms exhibit high-level resistance to penicillins, cephalosporins, and monobactams, leaving carbapenems as the mainstay of treatment. However, over-reliance on carbapenems has accelerated the emergence CRE, particularly in high-risk surgical and intensive care populations.[27] CRE infections are associated with increased treatment failure, sepsis-related mortality, and transmission risk within surgical wards.

MRSA, VRE, and gram-positive resistance

Methicillin-resistant *Staphylococcus aureus* (MRSA) remains a significant cause of superficial and incisional SSIs, especially in upper GI surgeries and among patients with prior hospital exposure. These strains often harbour co-resistance to macrolides and fluoroquinolones, limiting treatment options to glycopeptides or newer agents like linezolid and daptomycin.[28]

The sporadic but concerning emergence of vancomycin-resistant *Enterococci* (VRE) further complicates polymicrobial intra-abdominal infections and is particularly relevant in immunocompromised surgical populations.[29]

Mechanisms and patterns of resistance

Resistance mechanisms in GI SSIs are increasingly complex, involving not only enzymatic degradation (e.g., ESBLs, carbapenemases like NDM and KPC) but also efflux pumps, altered porin channels, and biofilm formation that shields bacterial colonies from host defences and antibiotic penetration.[30] These mechanisms contribute to persistent infections, recurrence, and poor wound healing, especially in patients with comorbidities or those undergoing re-operations.

Table 3 outlines the principal resistance mechanisms, the associated organisms, and their clinical consequences in GI SSI management.

Table 3: Resistance mechanisms in GI SSI pathogens.

Resistance mechanism	Organisms involved	Clinical implications
ESBL production	*E. coli, K. pneumoniae*	Ineffective cephalosporins; carbapenem reliance
Carbapene-mase production	CRE, *Acinetobacter baumannii*	Resistance to last-resort agents; high mortality
Methicillin resistance (mecA)	*Staphylococcus aureus (MRSA)*	Requires vancomycin/li-nezolid; fluoroquinolone failure
Van gene acquisition	*Enterococcus spp. (VRE)*	Resistant to vancomycin; often polymicrobial
Biofilm formation	*S. aureus, Enterococcus faecalis*	Chronic infections; impaired antibiotic penetration

The role of antibiotic stewardship

A cornerstone strategy to combat resistance is the implementation of robust antimicrobial stewardship programs (ASP). These focus on tailoring prophylactic and therapeutic antibiotics based on procedure type, institutional antibiograms, and patient-specific risk factors. Key stewardship principles include avoiding unnecessary broad-spectrum antibiotics, ensuring timely de-escalation, and restricting carbapenem use unless justified.[31] Interdisciplinary ASP teams- comprising surgeons, microbiologists, pharmacists, and infectious disease specialists- have demonstrated reductions in both infection rates and resistance emergence.

Surveillance and rapid diagnostics

Surveillance systems that monitor resistance trends in real-time are essential for updating local empiric guidelines and minimizing treatment delays. The integration of molecular diagnostic tools, such as multiplex PCR and resistance gene panels, allows early pathogen identification and facilitates targeted therapy.[32] These tools are particularly valuable in polymicrobial SSIs, where culture results may be delayed or incomplete.

In summary, rising antibiotic resistance has transformed the landscape of GI SSI management. Combating this threat requires a synergistic approach integrating surveillance, stewardship, rapid diagnostics, and surgical prudence.

ERAS IN SSI PREVENTION

ERAS protocols have transformed the landscape of perioperative care in GI surgery. Designed to reduce physiological stress, hasten recovery, and standardize best practices, ERAS programs have demonstrated significant reductions in SSI rates through multimodal interventions that optimize the patient journey from preoperative preparation to postoperative recovery.[33]

Core ERAS elements that mitigate SSI risk

Several pillars of ERAS directly influence the incidence of SSIs. Pre-operative optimization-including correction of anemia, nutritional supplementation, glycaemic control, and smoking cessation- bolsters host immunity and tissue perfusion, thereby reducing infection risk.[34] Patient education and expectation setting further promote adherence to postoperative mobilization and hygiene protocols.

Intra-operatively, ERAS promotes minimally invasive approaches (laparoscopy/robotics), which are consistently associated with lower SSI rates due to reduced incision size, limited tissue trauma, and shorter exposure times.[35] Strict adherence to evidence-based antimicrobial prophylaxis appropriately timed, weight-adjusted, and procedure-specific- along with intraoperative

normothermia and high-concentration oxygen delivery, enhances local wound defence mechanisms.[36]

Postoperatively, ERAS encourages early enteral nutrition and mobilization, both of which are linked to enhanced immune function and lower infection rates. Standardized wound care protocols, use of advanced dressings, and minimization of indwelling devices also contribute significantly to SSI prevention.[37] Key ERAS components contributing to infection reduction are listed in Table 4, illustrating their mechanistic impact across the surgical timeline.

Table 4: ERAS elements that impact SSI reduction.

ERAS components	Anti-SSI mechanism
Pre-operative optimization	Glycaemic control, smoking cessation, nutrition
Minimally invasive surgery	Less wound trauma, shorter exposure
Timely antibiotic prophylaxis	Reduces intra-operative bacterial load
Normothermia/oxygenation	Enhances tissue perfusion and immune response
Early enteral nutrition	Promotes healing, reduces bacterial translocation
Early mobilization	Lowers risk of nosocomial infections

Impact on SSI outcomes

A growing body of literature, including systematic reviews and randomized controlled trials, supports the SSI-reducing impact of ERAS. Meta-analyses have demonstrated up to a 40-50% reduction in SSI rates among ERAS-compliant GI surgical cohorts compared to conventional care.[38] Moreover, institutions with well-structured ERAS programs report lower reoperation rates, shorter length of stay, and improved patient satisfaction.

Implementation challenges and adaptive strategies

Despite compelling evidence, ERAS adoption is not uniform across centres. Barriers include surgeon scepticism, logistical challenges in multidisciplinary coordination, and lack of institutional infrastructure. Tailoring ERAS protocols to local epidemiology, surgical volume, and microbiological profiles is essential. The integration of SSI-specific checklists, electronic compliance monitoring, and audit-feedback loops can enhance adherence and outcomes.[39]

To bridge these gaps, several institutions have successfully introduced ERAS champions designated coordinators who monitor compliance, conduct protocol-based training workshops, and spearhead data-driven audits. Integration of mobile compliance checklists and perioperative dashboards have also been effective in improving adherence. ERAS should not be viewed in isolation but rather as a dynamic platform capable of synergizing with antimicrobial stewardship, infection surveillance, and personalized SSI risk prediction. Its success hinges on commitment, adaptability, and continuous education at every node of the perioperative pathway.

FUTURE DIRECTIONS AND RESEARCH GAPS IN SSI PREVENTION

Despite significant strides in reducing SSIs in GI surgery, the path to sustained, universal prevention remains incomplete. Emerging resistance patterns, technological disparities, and patient heterogeneity necessitate a future-forward, precision-oriented approach that integrates innovation with contextual relevance.[40]

Precision medicine and personalized SSI risk stratification

Current SSI prevention strategies rely heavily on standardized protocols, which, while effective at scale, often fail to account for inter-individual differences in immunity, microbiota, wound biology, and healing trajectories. The future lies in personalized risk prediction models- powered by genomics, proteomics, and metabolomics- that can stratify patients preoperatively and direct tailored prophylactic, nutritional, and immunomodulatory interventions.[41] Machine learning algorithms trained on real-world surgical datasets may enable point-of-care decision-making, particularly in high-risk subgroups.

Novel antimicrobial modalities

With the antibiotic pipeline dwindling and resistance escalating, alternative antimicrobial strategies are gaining attention. Bacteriophage therapy offers targeted lysis of resistant organisms without affecting native flora- a major advantage in maintaining gut barrier integrity.[42] Additionally, antimicrobial peptides, quorum-sensing inhibitors, and biofilm-disrupting adjuvants hold promise in enhancing conventional antibiotic efficacy and wound sterilization.[43]

Table 5 highlights novel and emerging strategies that aim to reshape future SSI prevention, from phage therapy to AI-driven risk prediction.

Rapid diagnostics and surveillance

Time-to-pathogen identification remains a critical bottleneck in SSI management. The integration of next-generation sequencing, real-time polymerase chain reaction (PCR), and resistance-gene detection platforms may dramatically improve targeted antimicrobial initiation and de-escalation strategies.[44] Portable molecular diagnostics with high sensitivity and specificity will be key, particularly in emergency or resource-limited surgical settings.

Table 5: Emerging strategies for future SSI management.

Innovation	Function	Development status
AI risk prediction algorithms	Personalized SSI prevention planning	Early clinical trials
Bacteriophage therapy	Targets MDR pathogens without dysbiosis	Experimental, phase I-II
Rapid resistance diagnostics	Early gene-level detection of resistance	Available in select centres
Microbiome-based prophylaxis	Restores gut flora to prevent sepsis/SSI	Animal studies
SSI apps/surveillance tools	Real-time wound tracking in LMICs	Pilots in India, Africa

ERAS 2.0- adaptive, contextualized protocols

As ERAS protocols mature, the need for adaptability grows. Future ERAS iterations must accommodate patients with frailty, malignancy, immunosuppression, and sepsis. Adjunctive components- such as immunonutrition, microbiome modulation, opioid-sparing multimodal analgesia, and glycaemic precision tools- can bolster surgical resilience and infection prevention.[33]

Implementation science and equity in LMICs

Many innovations remain confined to tertiary centres in high-income countries. The future of SSI prevention must include scalable, cost-effective strategies that are implementable in low- and middle-income countries (LMICs), where infection control challenges are amplified by infrastructural and human resource limitations. Bundled interventions, mobile wound surveillance apps, and context-sensitive antibiotic protocols could bridge this gap.[45] In summary, the next decade must focus not only on refining protocols but also on integrating personalized care, novel technologies, and global equity. Interdisciplinary collaboration, robust research, and pragmatic innovation will define the future of SSI prevention in GI surgery.

CONCLUSION

SSIs in GI surgery remain a formidable postoperative complication despite decades of clinical advancement. Their impact transcends simple wound morbidity- delaying recovery, escalating costs, jeopardizing oncologic timelines, and, increasingly, threatening lives in the era of antimicrobial resistance. From clean-contaminated fields to the emergent belly, the battle against SSIs has shifted from the suture table to the microscopic battlefield of resistant pathogens and biofilms. This comprehensive review underscores that while traditional pathogens still dominate the microbiological landscape, the rise of multidrug-resistant organisms- ESBL producers, CRE, MRSA, and VRE- has significantly eroded the effectiveness of empiric protocols. These organisms thrive in the shadow of antibiotic misuse and procedural vulnerabilities, requiring us to recalibrate how we prevent, diagnose, and treat SSIs. The emergence and widespread adoption of ERAS protocols have redefined infection prevention- not as an isolated target but as an embedded outcome of holistic, evidence-based perioperative care. When integrated with antimicrobial stewardship, robust surveillance, and precision diagnostics, ERAS forms the backbone of modern SSI mitigation. Looking ahead, the future of SSI prevention lies in personalization, innovation, and equitable implementation. From AI-driven risk stratification to phage therapy and LMIC-adapted care bundles, the tools are emerging- what remains is their deliberate and context-sensitive deployment. In essence, the war on SSIs is not yet won. But with science as our sword and strategy as our shield, we are poised to enter the next decade not just treating SSIs-but outsmarting them.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. U. S. Centers for Disease Control and Prevention. Surgical Site Infection Event (SSI). CDC. 2025:1-44.
2. Gillespie BM, Harbeck E, Rattray M, Liang R, Walker R, Latimer S, Thalib L, et al. Worldwide incidence of surgical site infections in general surgical patients: A systematic review and meta-analysis of 488,594 patients. Int J Surg. 2021;95:106136.
3. Islam MS, Faizi SAM, Rahman M, Alam RJ, Hossain MJ, Miraj AK. The surgical site infection following gastrointestinal surgery: a study in Sir Salimullah medical college and Mitford hospital, Dhaka, Bangladesh. Int Surg J. 2005;12(4):487-93.
4. Chadhary SM, Desai S, Pandya P. Assessment of surgical site infections and their risk factors in abdominal surgeries. Healthcare Bulletin. 2025;19(8):3113.
5. Mirnoto J, Irwan AM. Patient knowledge and participation in preventing surgical site infections: an integrative review. Gulhane Med J. 2025;67(2):58-66.
6. Bansal D, Singh RR., Ded KS, Aggarwal A, Puar GS, Shah AS. Bacteriological profile and antimicrobial susceptibility in surgical site infection in elective abdominal surgeries. Int Surg J. 2016;3(4):1879-82.
7. Iskandar K, Sartelli M, Tabbal M. Highlighting the gaps in quantifying the economic burden of surgical site infections associated with antimicrobial-resistant bacteria. World J Emerg Surg. 2019; 14:50.

8. StatPearls. Postoperative Wound Infections. NCBI Bookshelf. 2025.
9. Mekhla, Borle FR. Determinants of superficial surgical site infections in abdominal surgeries at a Rural Teaching Hospital in Central India: A prospective study. J Family Med Prim Care. 2019;8(7):2258-63.
10. Wondmeneh TG, Mohammed JA. The incidence of surgical site infection and its predictors among women delivered via cesarean sections in Ethiopia: a systematic review and meta-analysis. Front Med (Lausanne). 2024;11:1395158.
11. Kouzu K, Tsujimoto H, Shinji S. Effectiveness of advanced dressings in preventing surgical site infections compared to that of standard dressings in gastrointestinal surgery: A systematic review and meta-analysis for guideline revision by the Japanese Society for Surgical Infection. Ann Gastroenterol Surg. 2025;9(3):408-17.
12. Mohan N, Gnanasekar D, Ignatious A. Prevalence and Risk Factors of Surgical Site Infections in a Teaching Medical College in the Trichy District of India. Cureus. 2023;15(5):e39465.
13. Birgand G, Dhar P, Holmes A. The threat of antimicrobial resistance in surgical care: the surgeon's role and ownership of antimicrobial stewardship. Br J Surg. 2023;110(12):1567-69.
14. McLean KA, Goel T, Lawday S, Riad A, Simoes J, Knight SR, Ghosh D, et al. Prognostic models for surgical-site infection in gastrointestinal surgery: systematic review. Br J Surg. 2023;110(11):1441-50.
15. Calderwood MS, Anderson DJ, Bratzler DW, Dellinger EP, Garcia-Houchins S, Maragakis LL, et al. Strategies to prevent surgical site infections in acute-care hospitals: 2022 Update. Infect Control Hosp Epidemiol. 2023;44(5):695-720.
16. Costabella F, Patel KB, Adepoju AV, Singh P, Attia Hussein Mahmoud H, Zafar A, et al. Healthcare Cost and Outcomes Associated With Surgical Site Infection and Patient Outcomes in Low- and Middle-Income Countries. Cureus. 2023;15(7):e42493.
17. Cheadle WG. Risk factors for surgical site infection. Surg Infect (Larchmt). 2006;7:S7-S11.
18. Wolfhagen N, Boldingh QJJ, Boermeester MA, de Jonge SW. Perioperative care bundles for the prevention of surgical-site infections: meta-analysis. Br J Surg. 2022;109(10):933-42.
19. Rezaei AR, Zienkiewicz D, Rezaei AR. Surgical site infections: a comprehensive review. J Trauma Inj. 2025;38(2):71-81.
20. Kaur K, Oberoi L, Devi P. Bacteriological profile of surgical site infections. IAIM. 2017;4(12):7-83.
21. Allegranzi B, Bischoff P, de Jonge S, Kubilay NZ, Zayed B, Gomes SM, et al. New WHO recommendations on preoperative measures for surgical site infection prevention: an evidence-based global perspective. Lancet Infect Dis. 2016;16(12):e276-7.
22. Zimmerli W, Moser C. Pathogenesis and treatment concepts of orthopaedic biofilm infections, FEMS Imm Med Microb. 2012;65(2):158-68.
23. Bargavi T, Secunda R, Saravanan J, Satyanesan J. Surgical Site Infections in Gastrointestinal Surgeries: Estimation of Prevalence, Risk Factors and Bacteriological Profile. Cureus. 2024;16(6):e62589.
24. O'Driscoll T, Crank CW. Vancomycin-resistant enterococcal infections: epidemiology, clinical manifestations, and optimal management. Infect Drug Resist. 2015;8:217-30.
25. Bassetti M, Vena A, Giacobbe DR, Truchi C, Ansaldi F, Antonelli M, et al. Risk Factors for Intra-Abdominal Candidiasis in Intensive Care Units: Results from EUCANDICU Study. Infect Dis Ther. 2022;11(2):827-40.
26. Amrutham R, Reddy MMB, Pyadala N. A prospective study of surgical c in a teaching hospital. Int Surg J. 2016;4(1):237-41.
27. Kalakouti E, Simillis C, Pellino G, Pughal N, Warren O, Mills S, et al. Characteristics of Surgical Site Infection Following Colorectal Surgery in a Tertiary Center: Extended-spectrum β-Lactamase-producing Bacteria Culprits in Disease. Wounds. 2017;30(4):108-13.
28. Menz BD, Charani E, Gordon DL, Leather AJM, Moonesinghe SR, Phillips CJ. Surgical Antibiotic Prophylaxis in an Era of Antibiotic Resistance: Common Resistant Bacteria and Wider Considerations for Practice. Infect Drug Resist. 2021;14:5235-52.
29. O'Toole RF, Leong KWC, Cumming V, Van Hal SJ. Vancomycin-resistant Enterococcus faecium and the emergence of new sequence types associated with hospital infection. Res Microbiol. 2023;174(4):104046.
30. Yoon YK, Yang KS, Kim J, Moon C, Lee MS, Hur J, et al. Clinical implications of multidrug-resistant microorganisms and fungi isolated from patients with intra-abdominal infections in the Republic of Korea: a multicenter study. Diagn Microbiol Infect Dis. 2021;100(3):114960.
31. Sarang B, Tiwary A, Gadgil A, Roy N. Implementing antimicrobial stewardship to reduce surgical site infections: Experience and challenges from two tertiary-care hospitals in Mumbai, India. J Glob Antimicrob Resist. 2020;20:105-9.
32. Hrynyshyn A, Simões M, Borges A. Biofilms in Surgical Site Infections: Recent Advances and Novel Prevention and Eradication Strategies. Antibiotics (Basel). 2022;11(1):69.
33. Ljungqvist O, Scott M, Fearon KC. Enhanced Recovery After Surgery: A Review. JAMA Surg. 2017;152(3):292-8.
34. Gustafsson UO, Scott MJ, Hubner M, Nygren J, Demartines N, Francis N, et al. Guidelines for Perioperative Care in Elective Colorectal Surgery: Enhanced Recovery After Surgery (ERAS®) Society Recommendations: 2018. World J Surg. 2019;43(3):659-95.

35. Lohsiriwat V. Enhanced recovery after surgery vs conventional care in emergency colorectal surgery. World J Gastroenterol. 2014;20(38):13950-5.
36. Arrick L, Mayson K, Hong T, Warnock G. Enhanced recovery after surgery in colorectal surgery: Impact of protocol adherence on patient outcomes. J Clin Anesth. 2019;55:7-12.
37. Lord AS, Nicholson J, Lewis A. Infection Prevention in the Neurointensive Care Unit: A Systematic Review. Neurocrit Care. 2019;31(1):196-210.
38. Dong F, Li Y, Jin W, Qiu Z. Effect of ERAS pathway nursing on postoperative rehabilitation of patients undergoing gastrointestinal surgery: a meta-analysis. BMC Surg. 2025;25(1):239.
39. Seidelman JL, Mantyh CR, Anderson DJ. Surgical Site Infection Prevention: A Review. JAMA. 2023;329(3):244-52.
40. Boggi U. Precision surgery. Updates Surg. 2023;75(1):3-5.
41. Edwards R, Charani E, Sevdalis N, Alexandrou B, Sibley E, Mullett D, et al. Optimisation of infection prevention and control in acute health care by use of behaviour change: a systematic review. Lancet Infect Dis. 2012;12(4):318-29.
42. Abedon ST, García P, Mullany P, Aminov R. Editorial: Phage Therapy: Past, Present and Future. Front Microbiol. 2017;8:981.
43. Edmiston CE, McBain AJ, Roberts C, Leaper D. Clinical and microbiological aspects of biofilm-associated surgical site infections. Adv Exp Med Biol. 2015;830:47-67.
44. Mo Y. Rapid Diagnostics for Antibiotic Resistance: Urgent Need for Strong Clinical Evidence. Clin Infect Dis. 2022;75(12):2076-8.
45. Monahan M, Jowett S, Pinkney T, Brocklehurst P, Morton DG, Abdali Z, et al. Surgical site infection and costs in low- and middle-income countries: A systematic review of the economic burden. PLoS One. 2020;15(6):e0232960.

Cite this article as: Kumar S, Shah SP, Gupta S, Tandon V, Govil D. Surgical site infections in GI surgery: update on bacteriology, antibiotic resistance, and ERAS integration. Int Surg J 2025;12:1889-96.