International Surgery Journal
Vagholkar K et al. Int Surg J. 2026 May;13(5):897-901
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20261066

Review Article

Reducing surgical site infections: mechanisms, risks and prevention

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Received: 18 March 2026
Accepted: 02 April 2026

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ABSTRACT

Surgical site infections (SSIs) remain a leading cause of healthcare-associated infection with significant morbidity, mortality and cost. It is a surgeon's nightmare. SSI risk is multifactorial, involving patient, procedure, perioperative management, microbial and institutional factors. Objectives were to synthesize current evidence on determinants of SSI, mechanisms linking risk factors to infection and evidence-based prevention strategies. Narrative synthesis of guidelines, randomized trials, systematic reviews and key observational studies. Patient comorbidities (diabetes, obesity, immunosuppression), colonization (*S. aureus*), nutritional status, smoking and age increase SSI risk. Procedure-related factors include contamination class, operative time, tissue handling, implants and emergency surgery. Perioperative management which includes timing and weight-based dosing of prophylactic antibiotics, antiseptic skin preparation (alcohol-based chlorhexidine superior in many settings), maintenance of normothermia, perioperative glycaemic control, hair clipping (not shaving) and sterile technique significantly affect SSI rates. Environmental and institutional factors (OR ventilation, sterilization practices, surveillance programs) and pathogen factors (biofilm formation, antimicrobial resistance) also drive SSI risk. Bundled, multimodal prevention programs such as targeted interventions (e.g., *S. aureus* decolonization for carriers) are effective in high-risk populations in reducing SSI rates. Reduction in SSI rates requires identification and optimization of modifiable patient and process risk factors, adherence to evidence-based perioperative practices and systems-level surveillance and stewardship.

Keywords: Surgical site infection, Prevention, Perioperative, Prophylactic antibiotics, Chlorhexidine, Normothermia, Glycaemic control

INTRODUCTION

Surgical site infections (SSIs) occur at or near the operative incision within 30 days of surgery, or within 90 days when prosthetic material is implanted. SSIs range from superficial incisional to deep incisional organ or space infections and are associated with prolonged hospitalization, readmission, reoperation and increased mortality and costs.[1-3]

Epidemiology and impact

SSI incidence varies by procedure type and contamination class. It is low for clean procedures whereas substantially higher for colorectal and contaminated operations.

SSIs increase length of stay, antibiotic use, healthcare costs and contribute to antimicrobial resistance through increased antibiotic exposure.[1-4]

Classification of risk factors

SSI determinants are commonly grouped into the following patient-related, procedure or disease related, perioperative or process-related, microbial or the pathogen-related and environmental or the institutional factors.[1-3,5]

Patient-related factors

Diabetes mellitus and hyperglycaemia

Perioperative hyperglycaemia correlates with higher SSI risk whereas glucose optimization reduces the risk.[6,7]

Obesity is associated with impaired tissue perfusion, larger dead space and dosing challenges for prophylactic antibiotics and therefore linked to higher SSI rates.[8]

Malnutrition and hypoalbuminemia impair wound healing and immunity.[9]

Smoking and alcohol

Smoking reduces tissue oxygenation and impairs healing whereas alcohol abuse compromises immune responses.[10]

Immunosuppressives such as corticosteroids, chemotherapy, biologics and immunodeficiency states increase the risk of SSI.[11]

Colonization with S. aureus (including MRSA)

The carrier state predisposes to SSI necessitating targeted decolonization which reduces risk in selected populations.[12]

Age, frailty and comorbid disease burden (ASA class) are associated with increased SSI risk.[1,5]

Mechanisms include impaired host defence, reduced tissue perfusion and oxygenation, delayed collagen synthesis and decreased wound tensile strength (Table 1).

Table 1: Key risk factors and mechanisms.

Risk factors	Mechanism
Diabetes/hyperglycaemia	Impaired neutrophil function; poor healing
Obesity	Poor perfusion; larger dead space
Malnutrition/ hypoalbuminemia	Impaired collagen synthesis
Smoking	Reduced tissue oxygenation
Immunosuppression	Reduced host defence
***S. aureus* colonization**	Autoinoculation of wound
Prolonged surgery	Increased exposure and tissue trauma
Contaminated/dirty procedures	High microbial load
Implants/foreign material	Biofilm formation
Hypothermia	Impaired immunity; vasoconstriction

PROCEDURE AND DISEASE RELATED FACTORS

Contamination class (clean to dirty)

This is highly predictive of SSI risk.

Gastrointestinal and emergency operations carry higher baseline risk.[1,13]

Operative duration

Longer operative procedures increase exposure and tissue trauma which serves as surrogate for complexity.[14]

Surgical approach

Minimally invasive techniques often have lower SSI rates as compared to open approaches for comparable procedures.[15]

Implants and foreign bodies promote biofilm formation and the persistent infection thereby increasing the SSI rates.[16]

Tissue handling, hematoma, dead space and intraoperative spillage (e.g., bowel contents) increase infection risk.[13,16]

PERIOPERATIVE MANAGEMENT AND PROCESS-RELATED FACTORS

Antimicrobial prophylaxis

Timely administration (typically within 60 minutes before incision, 120 minutes for agents with prolonged infusion times) and appropriate weight-based dosing reduce SSI.

Prolonged postoperative prophylaxis (>24 hours) offers no additional benefit for most procedures.[2,17]

Skin antisepsis

Alcohol based chlorhexidine solutions have demonstrated superiority over aqueous povidone-iodine in several trials and provide guideline recommendations for many procedures.[18]

Preoperative bathing

Preoperative chlorhexidine bathing reduces skin bioburden. However, evidence for universal reduction in SSI is mixed but benefits are shown in bundled programs and targeted populations.[19]

Hair removal

If necessary, clipping immediately before surgery is preferred over shaving to avoid micro abrasions.[2]

Normothermia

Intraoperative hypothermia impairs immune function and tissue perfusion whereas active warming reduces SSI as shown in various randomized trials.[20]

Glycaemic control

Perioperative hyperglycaemia increases SSI risk even in non-diabetics. Moderate glycaemic control reduces infections while avoiding hypoglycaemia.[6,21]

Supplemental oxygen

Though trials show mixed results yet benefit may be context-dependent and more likely when part of bundles.[22]

Antiseptic wound management and sterile technique, including limiting OR traffic and proper instrument sterilization are critical in reducing SSI rates.[2]

Local adjuncts (wound protectors, antibiotic impregnated materials, triclosan coated sutures)

Meta-analyses show benefit in some settings, though effect sizes and persistence of benefit vary by procedure.[23-25]

Table 2: Core prevention measures.

Measures	Key action
Antimicrobial prophylaxis	Weight-based dose ≤60 min before incision; re-dose if prolonged
Skin prep	Alcohol-based chlorhexidine
***S. aureus* decolonization**	Mupirocin + chlorhexidine for carriers (selected patients)
Normothermia	Active warming to maintain ≥36°C
Glycaemic control	Avoid perioperative hyperglycaemia
Hair removal	Clip only, immediately preop
OR practices	Limit traffic; strict asepsis
Surveillance	Active monitoring and feedback

MICROBIOLOGY AND PATHOGEN FACTORS

Common pathogens include *S. aureus* (including MRSA), coagulase-negative *Staphylococci, Enterobacteria, P. aeruginosa, enterococci*, and anaerobes in intra-abdominal infections.[1,16] Biofilm formation on implants and suture material complicates eradication and requires specific management strategies.[16]

Antimicrobial resistance increases treatment complexity and adverse outcomes.[26]

Table 3: Common pathogens and prophylaxis.

Pathogens	Typical setting	Prophylaxis
***S. aureus* (MSSA)**	Skin, orthopaedics, cardiac	Cefazolin (weight-based)
MRSA	High-prevalence or colonized patients	Consider vancomycin (start earlier)
Enterobacteria/ anaerobes	Colorectal, biliary	Add anaerobic coverage (metronidazole or cefoxitin)
Pseudomonas	Burns, high-risk wounds	Not routine prophylaxis; treat if indicated
Polymicrobial	Dirty wounds, perforation	Broad-spectrum or culture-directed therapy

ENVIRONMENTAL AND INSTITUTIONAL FACTORS

OR ventilation and laminar flow may not uniformly reduce SSI and must be considered with other practices.[27] Sterilization and instrument processing failures increase SSI risk. Institutional resources, staffing, surveillance programs and adherence to bundles significantly affect SSI rates.[2,28] Active surveillance with feedback reduces SSI incidence and aids in benchmarking.[2,28]

PREVENTION BUNDLES AND IMPLEMENTATION

Evidence supports multimodal prevention bundles combining pre-, intra and postoperative measures. Optimized comorbid conditions, appropriate antibiotic prophylaxis, chlorhexidine-alcohol skin prep, normothermia, glycaemic control, *S. aureus* screening or decolonization for selected patients and strict aseptic technique are pivotal in reducing SSI rates to a bare minimum.

Implementation of scientific principles such as local adaptation, education, audit and feedback are required for sustained reductions.[2,12,18,20,28]

SPECIAL CONSIDERATIONS

Emergency surgery has higher SSI rates due to contamination and limited opportunity for optimization.[13]

Obesity requires weight-based dosing, consideration of subcutaneous drains in selected cases and careful wound closure techniques for reducing SSI.[8]

Immunocompromised patients need individualized prophylaxis and close surveillance.[11]

Implant-associated surgery

Device coatings, perioperative antibiotic strategies and limiting contamination are critical to prevent biofilm-related infections.[16,23]

DIAGNOSIS, SURVEILLANCE AND OUTCOMES MEASUREMENT

Standardized definitions (e.g., CDC/NHSN) are essential for surveillance and benchmarking. Diagnosis relies on clinical signs, imaging for deep or organ-space infections and microbiological cultures. Process measures (timing of antibiotics, achievement of normothermia) are useful quality indicators.[1,2,28]

GAPS IN KNOWLEDGE AND FUTURE DIRECTIONS

Key areas for future research include host genetic and immunologic determinants of SSI susceptibility, microbiome modulation, optimal prophylaxis strategies in the setting of increasing obesity. Antimicrobial resistance, cost-effectiveness of technological interventions (antimicrobial sutures, coatings) are undoubtedly useful. However high-quality RCTs of bundle components, particularly in low- and middle-income settings are essential.[16,23,26,28]

CONCLUSION

SSIs are multifactorial and many are preventable through patient optimization, adherence to evidence-based perioperative practices, multimodal bundles and institutional commitment to surveillance and continuous quality improvement. Tailored strategies based on procedure type, patient risk and resource setting maximize effectiveness.

Recommendations

Preoperative

Optimize comorbidities (glycaemic control, nutrition), counsel and facilitate smoking cessation. Screen and decolonize S. aureus carriers when indicated (e.g., high-risk procedures). Preoperative antiseptic bathing as part of a bundle for selected patients. Administer weight-based prophylactic antibiotics within 60 minutes of incision (120 minutes for specific agents).

Intraoperative

Use alcohol-based chlorhexidine for skin prep unless contraindicated. Maintain normothermia with active warming. Minimize operative time and tissue trauma. Use meticulous haemostasis and gentle tissue handling. Re-dose antibiotics for prolonged procedures or major blood loss.

Postoperative

Evidence-based wound care, timely dressing management, glycaemic control and early removal of drains or catheters when safe.

Systems

Implement surveillance, standardized definitions, audit and feedback, surgical safety checklists and antimicrobial stewardship programs.

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

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Cite this article as: Vagholkar K, Bhambri C, Paradkar I, Naidu P, Dudala B, Karnatki B. Reducing surgical site infections: mechanisms, risks and prevention. Int Surg J 2026;13:897-901.