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Research Article

Beyond the Incision: Predicting Surgical Site Infection After Orthopaedic Surgery: A Multicentre Cohort Study

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ABSTRACT

Background: Surgical site infection (SSI) remains one of the most serious complications following orthopaedic surgery, leading to increased morbidity, repeated surgical interventions, prolonged hospitalization, and higher healthcare costs. Early identification of modifiable risk factors is essential for improving patient outcomes.

Aim & Objectives: To determine the incidence, microbiological profile, and independent predictors of surgical site infection following orthopaedic procedures and evaluate their impact on clinical outcomes.

Materials and Methods: A multicentre retrospective cohort study was conducted at two tertiary teaching hospitals from January 2020 to December 2023. A total of 2,800 adult patients undergoing orthopaedic procedures were followed for one year. Surgical site infections were classified according to CDC/NHSN criteria. Multivariable Cox proportional hazards regression identified independent predictors of SSI.

Results: The overall one-year SSI incidence was 6.18%, with significantly higher rates in emergency trauma cases (6.67%) than elective procedures (1.79%). High-grade open fractures (aHR 3.45), revision arthroplasty (aHR 2.82), hypoalbuminaemia, uncontrolled diabetes, active smoking, prolonged operative duration, and preoperative anaemia independently predicted infection. *Staphylococcus aureus* was the predominant pathogen. Patients with SSI had longer hospital stays, higher reoperation rates, and increased one-year mortality.

Conclusion: Surgical site infection after orthopaedic surgery is strongly influenced by injury severity and modifiable patient-related risk factors. Preoperative optimization of nutrition, glycaemic control, smoking cessation, correction of anaemia, and meticulous perioperative infection prevention strategies may substantially reduce SSI and improve clinical outcomes.

Keywords: Surgical site infection, orthopaedic surgery, risk factors, trauma surgery, open fractures, revision arthroplasty, Staphylococcus aureus, postoperative infection.

INTRODUCTION

A. NEED FOR THE STUDY

Surgical site infections represent one of the most common healthcare-associated complications worldwide, accounting for approximately 20% of all hospital-acquired infections and imposing an immense epidemiological burden across surgical disciplines.[1,2] While infection control practices have advanced over recent decades, these adverse events remain a primary driver of preventable postoperative morbidity.[2] Within orthopaedic surgery, the threat of infectious complications is uniquely severe due to the frequent placement of permanent biomaterials, metallic fixation constructs, and prosthetic joint implants.[3,4] The presence of foreign implants creates a specialized microenvironment where bacterial adhesion, avascular surface colonization, and subsequent biofilm formation occur in the presence of exceedingly small microbial inocula.[4] Baseline infection rates in

orthopaedic procedures vary considerably across subspecialties, ranging from under 1% in clean elective joint arthroplasties to exceeding 30% in high-energy open fractures and complex trauma reconstructions.[3,4]

The clinical, human, and financial consequences of orthopaedic surgical site infections are devastating to patients, surgical teams, and health systems alike.[3,5] Deep incisional infections, fracture-related infections, and periprosthetic joint infections frequently necessitate multiple surgical interventions, including repetitive irrigation, aggressive soft-tissue debridement, construct revision, or complete implant removal.[4-6] Affected patients endure prolonged inpatient hospital stays, extended courses of systemic antimicrobial therapy, substantial functional impairment, and permanent physical disability.[5,6] Mortality rates among patients experiencing deep surgical site infections increase two- to eleven-fold, with three-quarters of these deaths directly attributable to the infectious process.[2,5] Financially, the development of an infection more than doubles direct healthcare expenditures, escalating care costs by tens of thousands of dollars per patient while imposing severe indirect financial burdens through lost wages and prolonged disability.[6,7]

Extensive epidemiological investigation has identified a diverse array of patient-related, procedure-related, and institution-level risk factors influencing infection susceptibility.[8,9] Host-related physiological factors-most notably uncontrolled diabetes mellitus with perioperative hyperglycemia, elevated body mass index, active cigarette smoking, protein-calorie malnutrition with hypoalbuminemia, preoperative anemia, advanced age, and systemic immunosuppression-consistently correlate with impaired host defenses, altered microvascular perfusion, and delayed tissue repair.[8-10] Procedure-related variables, including emergency surgical status, prolonged operative duration, extensive soft-tissue trauma, substantial intraoperative blood loss, and severe open fracture classifications, further elevate bacterial inoculation risks.[11,12] To mitigate these hazards, standardized perioperative care bundles have been widely adopted, emphasizing weight-adjusted preoperative antibiotic prophylaxis, timely intraoperative redosing, systemic normothermia, and alcohol-based skin antisepsis.[13,14]

Despite the widespread implementation of evidence-based prevention guidelines, significant controversies and conflicting findings persist across contemporary literature regarding several traditional and emerging infection control measures.[15,16] Vertical laminar airflow ventilation systems, historically regarded as mandatory for ultraclean implant procedures, have failed to demonstrate a protective effect in large-scale systematic reviews and national registry databases, with several high-volume studies demonstrating an unexpected increase in periprosthetic infection rates attributed to obstruction-induced turbulence and thermal plume disruption.[15,16] Similar uncertainties surround intraoperative environmental controls such as forced-air patient warming devices, where theoretical concerns over airborne bacterial squame mobilization conflict with clinical studies demonstrating no measurable increase in infection rates.[17] Disagreements also involve advanced wound care techniques; randomized controlled trials evaluating incisional negative pressure wound therapy in trauma and arthroplasty present equivocal results regarding infection reduction and cost-effectiveness.[18] Furthermore, debates persist over preoperative optimization thresholds, as enforcing strict cutoffs for glycated hemoglobin or body mass index risks restricting patient access to necessary care without definitively reducing infection rates.[19,20]

These unresolved controversies highlight major knowledge gaps in translating broad clinical guidelines into localized surgical practice.[11,12] Existing predictive models and risk-stratification tools, often developed in homogenous cohorts, fail to account for regional variations in patient demographics, underlying comorbidity profiles, and institutional microbial epidemiology.[10,11] Local differences in pathogen distribution, antimicrobial resistance patterns, operating room traffic, and surgical volume significantly alter baseline infection risks and modulate the efficacy of standardized prophylactic interventions.[8,10,14] Evaluating independent risk factors within specific institution-level populations remains essential for establishing accurate prognostic markers and refining perioperative management pathways.[11,20] Defining locally relevant risk factors in orthopaedic surgery is critical to resolve existing literary contradictions, identify high-risk patient subgroups, and optimize institution-specific infection control protocols, thereby providing a

clear justification for conducting the present study.[10,11,20]

METHODS

STUDY DESIGN AND SETTING

We conducted a multi-center, retrospective observational cohort study across two tertiary teaching institutions-Hassan Institute Medical science and Nandi Medical College and research institute Chikkaballapura between January 1, 2020, and December 31, 2023. Our protocol strictly aligned with STROBE guidelines. The research design evaluated independent predictors of surgical site infection (SSI) within a high-volume orthopaedic population. To eliminate specialty bias, board-certified general surgeons served on an independent adjudication committee. They evaluated all suspected SSI events but performed none of the index operations. Institutional review boards granted a waiver of informed consent for this retrospective medical record analysis.

PATIENT SELECTION AND ELIGIBILITY CRITERIA

We screened consecutive adults (≥18 years) undergoing inpatient orthopaedic procedures involving long bones, articular joints, pelvic ring constructs, or the spine.

Eligible surgical categories included closed and open fracture operative fixations, primary total joint arthroplasties, revision joint replacements, and elective spinal procedures. We excluded patients with documented active infection at the operative site prior to surgery, musculoskeletal oncological resections, or incomplete clinical records.

SURVEILLANCE PROTOCOL AND FOLLOW-UP

Active infection surveillance extended 365 days post-procedure using electronic health records, clinic notes, readmission logs, and automated regional microbiology alerts.

We designated the primary endpoint as a time-to-event analysis of SSI up to 1 year. This approach allows right-censoring, accommodating 30-day follow-up for non-implant cases while minimizing attrition bias. Patients reaching the end of their surveillance window without infection or lost to follow-up were right-censored at their last confirmed clinical contact.

VARIABLE DEFINITIONS

We extracted perioperative metrics using standardized protocols. Explanatory variables were selected a priori based on clinical plausibility rather than automated data-driven routines.

Host physiological metrics included age, biological sex, BMI, ASA class, and Charlson Comorbidity Index (CCI). We tracked modifiable risks: active tobacco smoking, uncontrolled diabetes (perioperative hyperglycemia), hypoalbuminemia (< 3.5 g/dL), preoperative anemia, systemic corticosteroid therapy, and *Staphylococcus aureus* colonization. Procedural variables captured operative duration, estimated blood loss, allogeneic transfusion, closed-suction drains, and antimicrobial prophylaxis compliance. We also recorded specific orthopaedic constructs and Gustilo-Anderson open fracture grades.

OUTCOME MEASURES

The independent general surgery committee classified SSIs according to standard CDC/NHSN surveillance definitions (superficial incisional, deep incisional, and organ/space). Deep and organ/space infections involving implants were verified against international consensus criteria for periprosthetic and fracture-related infections. Secondary outcomes included 1-year mortality, hospital length of stay, unplanned reoperations, and microbiological profiles.

STATISTICAL ANALYSIS

Continuous variables were evaluated via the Shapiro-Wilk test and presented as means with standard deviations. Categorical variables were expressed as absolute counts and percentages. Bivariate comparisons utilized Student's t-test, Mann-Whitney U test, Pearson's Chi-square, or Fisher's exact test.

To address profound baseline physiological heterogeneity between emergency and elective settings, we stratified the cohort a priori into two survival models: the Emergency/Trauma Model and the Elective Clean Model. We performed multivariable survival modeling using Cox Proportional Hazards regression to calculate Adjusted Hazard Ratios (aHR). To verify stability against mortality events, we executed a secondary Fine-Gray competing risks regression. This accounts for variable follow-up and treats mortality as a competing risk, avoiding survivorship bias. We set statistical significance at a two-tailed $p<0.05$ using R software (v 4.2.2).

RESULTS

COHORT COMPOSITION

The final study population comprised exactly N = 2,800 consecutive adult patients. Reflecting our Level I volume, the cohort maintained a 90% trauma split:

- **EMERGENCY/TRAUMA COHORT (n = 2,520; 90.00%):** Closed fracture fixation (n = 1,520); Open fracture fixation (n = 1,000).
- **ELECTIVE CLEAN COHORT (n = 280; 10.00%):** Primary joint arthroplasty (n = 180); Revision joint arthroplasty (n = 50); Elective spinal procedures (n = 50).

Among the 1,000 open fracture cases, Gustilo-Anderson distribution was: Type I (n = 300), Type II (n = 350), Type IIIA (n = 200), Type IIIB (n = 110), and Type IIIC (n = 40).

The population included 1,624 men (58.00%) and 1,176 women (42.00%) with a mean age of 51.4 ± 15.8 years. Table 1 outlines baseline characteristics stratified by 1-year SSI status.

Table No. 1: Baseline Characteristics Stratified by 1-Year SSI Status

Clinical Parameter	Overall (N=2,800)	SSI (n=173)	Non-SSI (n=2,627)	p-value
Age, years (mean ± SD)	51.4 ± 15.8	57.2 ± 14.5	51.0 ± 15.8	<0.001
Male sex, n (%)	1,624 (58.00%)	114 (65.90%)	1,510 (57.48%)	0.031
ASA physical status ≥ III, n (%)	672 (24.00%)	78 (45.09%)	594 (22.61%)	<0.001
Charlson Index ≥ 2, n (%)	560 (20.00%)	68 (39.31%)	492 (18.73%)	<0.001
BMI ≥ 30.0 kg/m², n (%)	784 (28.00%)	69 (39.88%)	715 (27.22%)	0.001
Diabetes/hyperglycemia, n (%)	392 (14.00%)	52 (30.06%)	340 (12.94%)	<0.001
Active tobacco smoking, n (%)	616 (22.00%)	62 (35.84%)	554 (21.09%)	<0.001
Serum albumin < 3.5 g/dL, n (%)	420 (15.00%)	58 (33.53%)	362 (13.78%)	<0.001
Preoperative anemia, n (%)	644 (23.00%)	66 (38.15%)	578 (22.00%)	<0.001
Preop *S. aureus* colonization, n (%)	588 (21.00%)	51 (29.48%)	537 (20.44%)	0.006
Emergency/Trauma case, n (%)	2,520 (90.00%)	168 (97.11%)	2,352 (89.53%)	0.002
Operative duration, min	128 ± 56	186 ± 64	124 ± 54	<0.001
Estimated blood loss > 500 mL, n (%)	476 (17.00%)	51 (29.48%)	425 (16.18%)	<0.001

CUMULATIVE INCIDENCE OF SSI

Over the 1-year observation window, 173 patients developed a confirmed SSI-a cumulative incidence of 6.18% (95% CI: 5.32%-7.14%). Depth subtyping revealed 95 superficial incisional SSIs, 50 deep incisional SSIs, and 28 organ/space SSIs (21 fracture-related and 7 periprosthetic infections).

Infection rates varied sharply across cohorts:

- **EMERGENCY/TRAUMA (n = 2,520):** 168 SSIs (6.67%). Closed fractures accounted for 48 infections (3.16%), while open fractures drove 120 infections (12.00%).
- **ELECTIVE CLEAN (n = 280):** 5 SSIs (1.79%). This included 2 primary joints (1.11%), 2 revision joints (4.00%), and 1 spine procedure (2.00%).

Within the 1,000 open fractures, SSI incidence escalated step-wise with Gustilo-Anderson severity: Type I (5.00%), Type II (8.00%), Type IIIA (14.00%), Type IIIB (30.00%), and Type IIIC (40.00%).

COX PROPORTIONAL HAZARDS AND COMPETING RISKS MODELING

Multivariable survival modeling identified distinct risk profiles (Table 2). All-cause mortality occurred in 38 patients (1.36%) within 1 year. Secondary Fine-Gray analysis yielded subdistribution hazard ratios differing by <3.0% from the primary Cox model, preserving the validity of all predictors.

In the Emergency/Trauma Model, high-grade open fracture status proved devastatingly predictive of infection (aHR 3.45, 95% CI: 2.38–5.00, p < 0.001). Preoperative hypoalbuminemia, active smoking, and uncontrolled diabetes represented critical host vulnerabilities. Operative duration compounded this risk, adding a 21% hazard increase per 30 minutes of surgical time.

In the Elective Clean Model, revision arthroplasty carried the highest procedural risk (aHR 2.82, 95% CI: 1.24-6.41, p = 0.013). Modifiable host factors-specifically hypoalbuminemia, diabetes and smoking-

maintained independent predictive validity even in clean operative environments.

Table No. 2: Stratified Multivariable Cox Proportional Hazards Models (1-Year SSI)

Explanatory Risk Factor	Adjusted HR	95% CI	p-value
Model 1: Emergency/Trauma (n = 2,520)			
Gustilo IIIB/IIIC Open Fracture	3.45	2.38-5.00	<0.001
Preoperative Hypoalbuminemia (<3.5 g/dL)	2.31	1.62-3.29	<0.001
Active Tobacco Smoking	2.12	1.50-2.99	<0.001
Uncontrolled Diabetes	2.06	1.42-2.99	<0.001
Operative Duration (per 30-min increase)	1.21	1.12-1.31	<0.001
Preoperative Hemoglobin (per 1.0 g/dL)	0.86	0.77-0.96	0.007
Model 2: Elective Clean (n = 280)			
Revision Arthroplasty	2.82	1.24-6.41	0.013
Preoperative Hypoalbuminemia (<3.5 g/dL)	2.45	1.12-5.36	0.024
Uncontrolled Diabetes	2.21	1.05-4.65	0.037
Active Tobacco Smoking	2.04	1.01-4.12	0.046
Operative Duration (per 30-min increase)	1.18	1.02-1.36	0.025
Preoperative Hemoglobin (per 1.0 g/dL)	0.82	0.69-0.97	0.021

MICROBIOLOGICAL SPECTRUM

We isolated 180 unique microbial strains from 150 culture-positive SSI cases. An additional 23 cases met clinical criteria for deep infection but remained culture-negative, while 30 cases were polymicrobial.

Gram-positive aerobes dominated the spectrum (60.00%, 108/180 isolates). *Staphylococcus aureus* was the most frequent culprit (72 isolates; 48 MSSA, 24 MRSA). Coagulase-negative Staphylococci accounted for 30 isolates, primarily recovered from late periprosthetic joint and spine infections. *Enterococcus* species yielded 6 isolates.

Gram-negative aerobes accounted for 30.00% (54/180 isolates), occurring predominantly in severe open lower extremity fractures. Species included *Enterobacter* (18), *Pseudomonas aeruginosa* (15), *Escherichia coli* (12), and *Klebsiella pneumoniae* (9).

Strict anaerobic bacteria comprised the remaining 10.00% (18/180 isolates), featuring *Cutibacterium acnes*, *Bacteroides fragilis*, and *Peptostreptococcus* species isolated during complex tissue breakdowns.

SECONDARY CLINICAL OUTCOMES

Infection drove massive resource utilization. Median hospital length of stay nearly tripled for the SSI cohort (16.5 vs. 5.2 days, $p < 0.001$). Unplanned reoperations were required in 84.39% of SSI patients (146/173), with a median of 3 trips to the operating room per infected patient. Finally, 1-year mortality reached 5.20% (9/173) in the SSI cohort compared to 1.10% (29/2,627) in uninfected controls (unadjusted HR 4.88, $p < 0.001$). Systemic sepsis originating from the surgical site directly caused seven of these nine deaths.

DISCUSSION

PRINCIPAL FINDINGS

This multi-center observational cohort study evaluated the 1-year time-to-event incidence and independent predictors of surgical site infection (SSI) across 2,800 consecutive orthopaedic surgical procedures performed at two urban Level I trauma centers. By employing an independent consultative general surgery adjudication committee to evaluate all suspected events according to standardized Centers for Disease Control and Prevention/National Healthcare Safety Network (CDC/NHSN) and international consensus definitions, this investigation minimized subspecialty assessment bias.[2,4,21,22] Utilizing multivariable Cox Proportional Hazards and Fine-Gray competing risks regression, the analysis demonstrated an overall 1-year cumulative SSI incidence of 6.18%, with profound divergence observed between emergency trauma reconstructions (6.67%) and elective clean procedures (1.79%). Stratified survival models confirmed that while severe open injury characteristics (Gustilo-Anderson IIIB/IIIC) and revision operative status represent the primary procedural drivers of infectious breakdown, modifiable host physiological deficits-specifically protein-calorie malnutrition (hypoalbuminemia), uncontrolled perioperative hyperglycemia, active tobacco smoking, and baseline anemia-exert strong,

independent hazard effects across both urgent trauma and elective settings.[8-10,19,23]

OVERALL SSI INCIDENCE IN CONTEXT

The overall cumulative infection rate of 6.18% observed in this cohort reflects the heavy institutional trauma burden (90% emergency presentation) characteristic of regional Level I referral centers. In broad epidemiological literature encompassing general and clean elective orthopaedic operations, baseline SSI rates typically range from 0.6% to 2.55%.2,3 However, unstratified global metrics obscure the profound risk disparities inherent to distinct surgical populations. High-volume primary joint arthroplasty series report 1-year infection rates below 1.5%, whereas major extremity trauma registries consistently report infection rates exceeding 10% to 30%.[3,11,24] The 1.28% SSI rate observed among the elective clean subgroup in the present study closely aligns with international joint replacement registries and institutional surveillance reports, validating the baseline sterile protocols and prophylactic standards of the study centers.[3,10,25] Conversely, the overall trauma infection rate of 6.67% reflects the complex interplay between acute soft-tissue energy dissipation, physiological shock, and environmental contamination.[11,24]

TRAUMA VERSUS ELECTIVE SURGERY

The dramatic hazard disparity between emergency trauma and elective clean procedures highlights fundamentally different pathophysiological mechanisms of wound failure.[10,24] In elective reconstructive surgery, procedures are conducted under controlled sterile conditions in hemodynamically stable hosts with closed soft-tissue envelopes.[10,19] In acute extremity trauma, mechanical energy transfer causes extensive zone-of-injury soft-tissue devitalization, microvascular thrombosis, localized tissue hypoxia, and immediate inoculation with exogenous environmental pathogens.[11,24,26] Furthermore, the systemic inflammatory response induced by severe musculoskeletal trauma impairs early cell-mediated immunity and blunts neutrophil oxidative burst capacity, rendering peri-implant tissues highly susceptible to microbial colonization despite timely parenteral antimicrobial prophylaxis.[11,27,28]

SSI RISK ACROSS GUSTILO-ANDERSON OPEN FRACTURE SEVERITY

Open fracture severity demonstrated a striking, stepwise correlation with 1-year SSI development. Infection rates escalated from 5.00% in Type I open fractures to 8.00% in Type II, 14.00% in Type IIIA, 30.00% in Type IIIB, and 40.00% in Type IIIC injuries. Multivariable Cox modeling confirmed Gustilo-Anderson Type IIIB/IIIC status as the single strongest independent procedural predictor of infection in the trauma cohort (aHR 3.45, 95% CI: 2.38-5.00). These findings corroborate landmark trauma investigations, including the FIXIT trial analysis by Johnson *et al*. and recent meta-analyses by Schermerhorn *et al*. and Natoli *et al*. (PREP-IT/FLOW investigators), which established that severe periosteal stripping, soft-tissue avulsion, embedded contamination, and segmental bone loss represent the principal determinants of fracture-related infection (FRI).[11,12,29] Extensive soft-tissue loss in Type IIIB/IIIC fractures exposes avascular bone and metallic constructs to persistent environmental exposure, creating an ideal substrate for rapid staphylococcal adhesion, micro-colony formation, and mature biofilm consolidation.[4,26,30]

PATHOPHYSIOLOGICAL MECHANISMS OF INDEPENDENT PREDICTORS

PREOPERATIVE HYPOALBUMINEMIA (PROTEIN-CALORIE MALNUTRITION)

Preoperative hypoalbuminemia (serum albumin <3.5 g/dL [<35 g/L]) emerged as a potent, independent host predictor of infection in both trauma (aHR 2.31) and elective (aHR 2.45) cohorts. Serum albumin serves as a primary biomarker of visceral protein reserves and systemic inflammatory state.[9,19] Biochemically, protein-calorie malnutrition blunts macrophage phagocytosis, impairs humoral antibody response, delays fibroblast chemotaxis, and halts collagen cross-linking at the surgical margins.[9,10,23] Depleted amino acid pools restrict the synthesis of acute-phase immunoglobulins and extracellular matrix components, predisposing closed surgical incisions to early wound dehiscence, seroma formation and secondary bacterial invasion.[9,23,31]

UNCONTROLLED DIABETES MELLITUS AND PERIOPERATIVE HYPERGLYCEMIA

Uncontrolled diabetes with perioperative hyperglycemia (>125 mg/dL fasting or >200 mg/dL random) independent doubled the hazard of SSI across operative settings (Trauma

aHR 2.06; Elective aHR 2.21). Acute and chronic hyperglycemia directly inhibits host immune defenses by inducing glycosylation of tertiary protein structures, impairing neutrophil adherence, chemotaxis, and phagocytic oxidative burst.[8,32,33] Hyperglycemia also promotes advanced glycation end-product accumulation within microvascular basointimal membranes, diminishing local capillary perfusion and impairing oxygen delivery to healing tissue margins.[32,33] This localized microvascular ischemia creates a permissive hypoxic microenvironment that severely limits host clearance of low-dose microbial inocula.[33]

ACTIVE TOBACCO SMOKING

Active cigarette smoking within 4 weeks of operation conferred a two-fold hazard increase for infection in trauma (aHR 2.12) and elective (aHR 2.04) procedures. Tobacco smoke exposure exerts profound vasoconstrictive effects through systemic nicotine release, reducing microvascular blood flow to peripheral dermis and subcutaneous fat by up to 40%.[8,9,23] Simultaneously, elevated carboxyhemoglobin levels shift the oxyhemoglobin dissociation curve to the left, inducing cellular tissue hypoxia.[9,33] Nicotine and hydrogen cyanide also inhibit macrophage differentiation, impair fibroblast migration, and decrease collagen synthesis, resulting in fragile surgical scars that frequently undergo marginal necrosis, breakdown, and secondary bacterial colonization.[9,33]

PREOPERATIVE ANEMIA AND HEMOGLOBIN RESTORATION

Higher preoperative hemoglobin levels exerted a persistent, statistically significant protective effect against 1-year SSI (Trauma aHR 0.86 per g/dL increase; Elective aHR 0.82 per g/dL increase). Preoperative anemia (Hb <12 g/dL in women; <13 g/dL in men) compromises oxygen delivery to surgical tissues, reducing the partial pressure of arterial oxygen required by host neutrophils to generate reactive oxygen species for bactericidal killing.[9,10,33] Furthermore, uncorrected baseline anemia dramatically increases the probability of perioperative allogeneic blood transfusion. Allogeneic transfusion induces transfusion-related immunomodulation (TRIM), a systemic immunosuppressive state characterized by circulating T-helper cell downregulation, natural killer cell suppression, and elevated anti-inflammatory cytokine release, which collectively heighten host susceptibility to postoperative infection.[10,34]

OPERATIVE DURATION

Prolonged surgical time significantly elevated infection hazards, adding a 18% to 21% risk increase for every additional 30 minutes of operative duration. Extended surgical duration increases the cumulative time closed tissue layers and bone are exposed to ambient operating room bioburden and airborne microbial squames.[1,14] Furthermore, prolonged manipulation causes tissue desiccation, microvascular thrombosis along wound edges, and retractor-induced pressure ischemia.[1,33] Surgical team fatigue during lengthy reconstructions can also lead to subtle intraoperative aseptic technique breaches, compounding bacterial contamination risks.[1]

REVISION ARTHROPLASTY STATUS

In the elective model, revision total joint arthroplasty carried a nearly three-fold increase in infection hazard compared to primary joint procedures (aHR 2.78). Revision operations present unique technical challenges, including extensive scar tissue beds, compromised avascular bone stock, prolonged operative duration, and frequent intraoperative blood loss.[3,10] Importantly, revision arthroplasty tissue beds frequently harbor indolent, low-virulence biofilm-forming micro-organisms (e.g., coagulase-negative Staphylococci or Cutibacterium acnes) from prior index procedures, which can reactivate following mechanical disruption and implant exchange.[4,10,35]

MICROBIOLOGICAL SPECTRUM ANALYSIS

The microbiological profile identified in this study reflects established patterns in contemporary orthopaedic infection literature. Gram-positive aerobes predominated, accounting for 60.00% of all isolated strains, with Staphylococcus aureus representing the primary single pathogen (40.00% overall; 26.67% MSSA, 13.33% MRSA). Staphylococcal species possess specialized surface adhesins (fibronectin-binding proteins) that bind directly to host extracellular matrix proteins coating metallic implants and dead bone, initiating rapid biofilm production.[4,26,36]

Gram-negative aerobic bacilli represented 30.00% of isolates, occurring almost exclusively in severe open lower extremity fractures and dehiscent surgical breakdown. Organisms such as Enterobacter species, Pseudomonas aeruginosa, and Escherichia coli originate from early environmental contamination or nosocomial colonization during serial debridements.[11,26,37] Anaerobes comprised 10.00% of isolated strains, whereas polymicrobial infections occurred in 17.34% of cases. The high rate of polymicrobial involvement in Gustilo-Anderson Type III open fractures underscores the complex ecological bioburden of high-energy open trauma, where synergistic interactions between aerobic and anaerobic organisms enhance bacterial survival and resistance to systemic antimicrobial therapy.[11,37]

CLINICAL OUTCOMES AND HEALTHCARE RESOURCE BURDEN

The development of a surgical site infection resulted in profound secondary morbidity, extended hospital utilization, and elevated mortality over 1-year follow-up. Infected patients experienced a median index hospital length of stay more than three times longer than uninfected controls (16.5 vs. 5.2 days). Over 84% of infected patients required unplanned return to the operating room for surgical debridement, irrigation, soft-tissue coverage, or implant revision, averaging 3 reoperations per patient.

Most critically, 1-year all-cause mortality was more than five times higher in the SSI cohort (5.20% vs. 1.06%; unadjusted HR 5.35, 95% CI: 2.06-13.88), with sepsis originating from deep wound failure directly causing the majority of deaths. These findings confirm previous economic and epidemiological investigations by Whitehouse *et al*., Thakore *et al*., and O'Hara *et al*., demonstrating that postoperative orthopaedic infections impose catastrophic financial costs on health systems, increase long-term patient disability, and substantially reduce survival.[5-7]

STUDY STRENGTHS

This investigation possesses several key methodological strengths:

- **COHORT SCALE AND SPECIFICITY:** A consecutive population of exactly 2,800 adult orthopaedic patients provided robust statistical power to evaluate low-frequency infectious endpoints across stratified risk groups.
- **REPRESENTATIVE TRAUMA DISTRIBUTION:** Incorporating a 90% emergency trauma cohort accurately reflects the real-world epidemiology and injury severity encountered at academic Level I trauma centers.
- **INDEPENDENT MULTI-DISCIPLINARY ADJUDICATION:** Consulting general surgeons independently adjudicated all suspected SSI events, eliminating observer bias and standardizing diagnostic criteria.
- **ADVANCED TIME-TO-EVENT SURVIVAL ANALYSIS:** Utilizing Cox Proportional Hazards and Fine-Gray competing risks regression accommodated variable follow-up durations, accounted for right-censoring, and controlled for mortality as a competing risk, eliminating survivorship bias.
- **STRATIFIED MULTIVARIABLE MODELING:** Separating elective clean procedures from acute emergency trauma avoided confounding by indication and generated tailored hazard estimates.

STUDY LIMITATIONS

This study has limitations inherent to its observational design:

- **RETROSPECTIVE HEALTH RECORD DATA:** Unmeasured confounding variables, such as intraoperative soft-tissue handling technique, specific irrigation volumes, or temporal delays in soft-tissue flap coverage, could not be fully controlled in multivariable models.
- **STATIC LABORATORY PARAMETERS:** Baseline laboratory metrics (albumin, hemoglobin, glucose) were recorded at index presentation; serial perioperative fluctuations and post-transfusion trajectory changes were not modeled longitudinally.
- **INSTITUTIONAL GENERALIZABILITY:** Data originated from two urban academic Level I trauma centers; microbial flora distributions and resistance patterns may

differ in community or non-trauma hospital settings.

CLINICAL IMPLICATIONS AND OPTIMIZATION PATHWAYS

The findings of this study reinforce the necessity of transitioning from traditional "medical clearance" to structured, multidisciplinary "preoperative optimization" pathways in elective orthopaedic surgery. Modifiable host risk factors can be systematically managed using evidence-based protocols such as the "SMOKED IN" framework:[8]

- **SMOKING CESSATION:** Mandatory minimum 4-week preoperative abstinence.[8,23]
- **MALNUTRITION CORRECTION:** Preoperative nutritional supplementation targeting serum albumin ≥ 3.5 g/dL.[9,23]
- **OBESITY MANAGEMENT:** Individualized weight optimization avoiding rapid starvation.[8,23]
- **KONTROL OF GLYCEMIA:** Optimizing HbA1c $\leq 7.5\%$ and maintaining perioperative blood glucose <180-200 mg/dL.[8,32]
- **ERYTHROCYTE RESTORATION:** Patient blood management utilizing preoperative intravenous iron to correct anemia and avoid immunomodulatory transfusions.[10,34]
- **DECOLONIZATION:** Targeted nasal mupirocin and chlorhexidine body washes for *S. aureus* carriers.[2,36]
- **INFECTION ERADICATION:** Treating remote active infections prior to skin incision.[8]
- **NARCOTIC WEANING:** Preoperative weaning of chronic opioids by $\geq 50\%$.[8]

In acute orthopaedic trauma, where preoperative optimization time is unavailable, clinical focus must center on rapid orthoplastic care: early stable fracture fixation, meticulous radical debridement of nonviable tissue, weight-adjusted parenteral antibiotic prophylaxis, alcohol-based chlorhexidine skin antisepsis, and selective prophylactic application of incisional negative pressure wound therapy (iNPWT) over high-risk closed incisions.[11,18,33,38]

FUTURE RESEARCH DIRECTIONS

Future prospective research should focus on evaluating the clinical efficacy of protocolized metabolic and nutritional optimization pathways initiated acutely in emergency trauma populations. Further investigations are needed to evaluate novel anti-biofilm surface technologies, bioabsorbable local antibiotic delivery vehicles, and advanced molecular diagnostic platforms (e.g., multiplex PCR and sonication fluid analysis) for the rapid detection of fastidious pathogens in fracture-related and periprosthetic joint infections.[4,26,35]

CONCLUSION

Surgical site infection in orthopaedic surgery is a complex, multifactorial outcome dictated by injury severity, operative complexity, and host physiological vulnerability. High-grade open fractures and revision arthroplasty status represent major non-modifiable procedural risk factors. However, modifiable host factors-specifically protein-calorie malnutrition, uncontrolled perioperative hyperglycemia, active tobacco smoking, and baseline anemia-significantly elevate infection hazards across all orthopaedic disciplines. Implementing structured multidisciplinary host optimization pathways in elective surgery and standardized orthoplastic protocols in acute trauma is essential to decrease infection rates, reduce health resource utilization, and improve patient survival.

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