Orthopaedic Knowledge Update®

OKU® 2

Musculoskeletal Infection

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Preface

Orthopaedic Knowledge Update®: Musculoskeletal Infection 2 is a comprehensive and updated guide to the diagnosis, prevention, and management of musculoskeletal infection, a complex and challenging problem that affects millions of people around the world. Musculoskeletal infection can cause severe complications for patients, their families, and the healthcare system, and it requires a multidisciplinary approach involving surgeons, infectious disease specialists, and basic scientists.

This book aims to provide a comprehensive and up-to-date overview of the current knowledge and best practices in the diagnosis, prevention, and treatment of musculoskeletal infection. Because the first edition of this book was published by AAOS in 2009, all chapters in this second edition have been newly written to reflect the recent advances in the field.

The first section discusses general aspects of musculoskeletal infection, such as epidemiology, risk factors, and risk reduction strategies. It also explores the basic science of infection, including diagnostic biomarkers and methods, microbiology of pathogens, biofilm biology, and irrigation solutions and techniques.

With recent advances in antibiotic therapy, an entire section is devoted to this topic. An in-depth review of antibiotic therapy is presented, covering general principles, local and systemic delivery, and specific considerations for different types of bone and joint infections. It also discusses the role of long-term antibiotic suppression in some cases.

The second half of the book addresses clinical scenarios of musculoskeletal infection—prosthetic joint infections, fracture-related infections, and other bone and joint and soft-tissue infections, including pediatric infections, hand and foot infections, spine infections, and necrotizing fasciitis. Chapters discuss the latest advances in diagnosis, surgical treatment, and antibiotic therapy for these conditions.

The editors and authors hope that this book will be a useful resource for residents, fellows, and practitioners who aim to provide optimal professional care to patients with musculoskeletal infection.

M. Daniel Wongworawat, MD, FAAOS
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SECTION 1

General Considerations

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CHAPTER 1

The Epidemiology of Musculoskeletal Infections

COLE HOWIE, MD • ELIE S. GHANEM, MD, FAAOS

ABSTRACT

As people live longer, in combination with increased morbidity and medical complexity, the projected trends suggest an increasing demand for elective and nonelective orthopaedic procedures with associated complications, including surgical site infection. The rate of surgical site infection after surgery, organism profile, and the organisms' evolving antibiotic resistance patterns can differ according to patient demographics, anatomic location, procedure performed, and several other confounding variables, creating a difficult scenario for all specialists involved in treating the infection. The patient faces high risk of treatment failure irrespective of treatment type, with burdensome and life-changing economic and social effects that can directly affect quality of life.

Keywords: economics; organism profile; prevalence; quality of life; surgical site infection

INTRODUCTION

Surgical site infections (SSIs) are devastating complications that may occur following elective orthopaedic surgery or a traumatic event. Rates of SSI vary across procedures and anatomic locations, which include primary joint arthroplasty (0.5% to 2%),[1] revision arthroplasty (3% to 9%),[2,3] fracture open reduction and internal fixation (ORIF) (1% to 5%),[4] and spine surgery (3.1%).[5] Treatment options across all specialties generally range from débridement, antibiotics, and implant retention[6] to staged revision surgery,[7] fusions, or even amputations for recalcitrant cases.[8] The natural history of SSI and subsequent treatment with a prolonged antibiotic course and recovery with extensive rehabilitation can be debilitating and costly for the patient, leading to disabilities and restricted activities, direct financial costs for treatment, indirect costs due to missed work or potential unemployment, and mental health burdens.[9] The hospital and health care workers also incur financial burdens consequent to repeated treatment strategies for recurrent infections with consequent readmissions and complications.[10] The patient's quality of life (QoL) can be greatly challenged by a musculoskeletal infection, affecting both the patient's physical and mental health with potential for irreversible disability compared with their initial functional state after the index procedure.[11,12]

PREVALENCE

The number of total hip arthroplasty (THA) and total knee arthroplasty (TKA) procedures performed each year is expected to increase by 2030, with a subsequent rise in incidence of prosthetic joint infections (PJI) thereafter.[13] The annual rate of PJI in the literature ranges anywhere from 0.5% to 2%[1] after primary total joint arthroplasty (TJA) and up to 7.0% after revision surgery.[14] The American Joint Replacement Registry 2020 Annual Report showed a steady increase in the rate of TKA revisions performed because of PJI since 2013 before peaking at 29.9% and subsequently dropping to 27.2% between 2019 and 2020 with similar findings reported for revision THA.[15]

Although total shoulder arthroplasty is performed less frequently than THA and TKA, their infection rates are

Dr. Ghanem or an immediate family member serves as a paid consultant to or is an employee of Symcel and has stock or stock options held in PSI. Neither Dr. Howie nor any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this chapter.

comparable and can reach up to 3%.[16] Similarly, the Mayo Clinic's Total Joint Registry found hemiarthroplasties to have a 1% infection rate[17] with approximately 98% infection-free survival rates at 5-, 10-, and 20-year follow-up. In contrast, reverse total shoulder arthroplasty performed on more complex cases showed a higher incidence of PJI, reportedly 3% to 4% in a 2020 study.[18] Another 2020 study reported that, of arthroplasty surgeries, total elbow arthroplasty (TEA) is the least commonly performed but has one of the highest postoperative infection rates of 3% to 8%; PJI comprised 43.5% of their primary TEA failures.[19]

A 2020 meta-analysis of spine surgeries found the prevalence of SSIs to be 3.1%, with superficial and deep SSI rates estimated at 1.4% and 1.7%, respectively.[5] This analysis concluded that the highest incidence of SSI was present in patients with neuromuscular scoliosis undergoing corrective deformity surgery (13%).[5] SSI rates for spinal deformity correction have been estimated at an overall rate of 1.2%, with kyphosis corrections reaching up to 2.4% compared with scoliosis and spondylolisthesis deformities (both 1.1%, $P < 0.0001$).[20] Similarly, SSI rates differ with the surgical approach used; a posterior-based approach (5.0%) has higher infection risk than an anterior-based approach (2.3%), and infection is less likely to develop after noninstrumented surgeries compared with instrumented surgeries (1.4% versus 4.4%).[5]

Infection rates are relatively higher for skeletal trauma surgeries, ranging from 1% to 4%, which is attributed to the injury mechanism disrupting the soft-tissue envelope, leading to potential contamination.[4] The anatomic location of the fracture plays a significant role in the incidence of fracture-related infection (FRI), with fractures of the elbow (6.6%), tibial plateau (7.6%), and tibial shaft (8.7%) occurring most often.[21] Open fractures are known to have an increased risk of FRI compared with closed fractures, with increasing frequency according to the Gustilo-Anderson classification, where type I, II, and III open fractures have zero to 2%, 2% to 12%, and 10% to 50% risk, respectively.[22]

ORGANISM PROFILE

Successfully treating a postoperative infection is heavily reliant on isolating the offending microbe at the surgical site and determining its antibiotic susceptibility, especially with the emergence of evolving drug-resistant organisms. The organism profile has been extensively described in TJA infection[23,24] (**Figure 1**). The most common culprit of TJA PJI is *Staphylococcus* species, with the incidence of *Staphylococcus epidermidis* (coagulase-negative staphylococci, CoNS) ranging between 20% and 35% and *Staphylococcus aureus* (including methicillin-sensitive *S aureus* and methicillin-resistant *S aureus* [MRSA]) from 8.5% to 21% for early-onset and late-onset infections.[24,25]

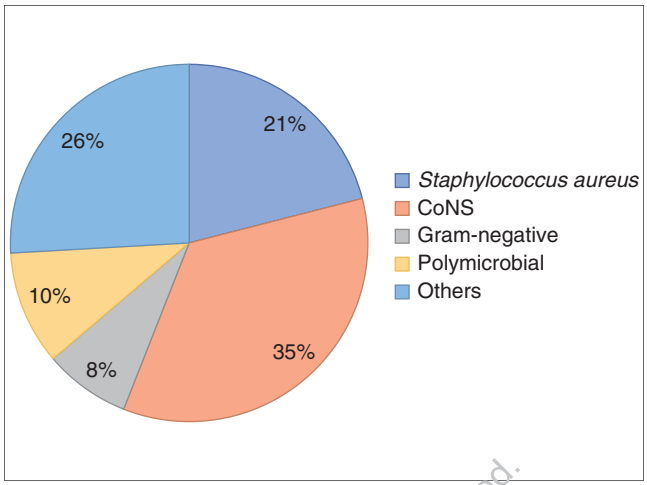

FIGURE 1 Graph showing the organism profile of bacteria commonly isolated from total hip arthroplasty and total knee arthroplasty prosthetic joint infection. CoNS = coagulase-negative staphylococci

CoNS and *Enterococcus faecalis* were found to be paired most frequently as copathogens in polymicrobial PJIs.[24] However, gram-negative and anaerobic pathogens are three times more likely to be in the mix of polymicrobial PJIs compared with gram-positive pathogens.[23] Anaerobic bacteria have been isolated in 3% to 6% of PJIs,[26] including *Cutibacterium* species, that are isolated more commonly in late infections.[27] Fungal organisms, although rare, have been reported in 1% of PJIs, with *Candida* being the most frequently identified pathogen.[28] Culture-negative infections occur in cases of high clinical suspicion of PJI with no culprit organism isolated and constitute 11% of infections but have no correlation with infection chronicity (acute versus chronic), implant type used, or antibiotic administration.[25] Culture-negative cases could be attributed to organisms that are challenging to culture in the laboratory or are rare pathogens not commonly isolated using routine culture methods, including *Coxiella burnetii*, *Brucella*, *Bartonella*, *Mycoplasma*, and mycobacterial and/or some fungal pathogens.[28]

The organisms causing PJI and their distinct profile, however, differ according to the anatomic location (**Figure 2**). A systematic review of shoulder PJI concluded that *Cutibacterium acnes* was the most frequent isolate, appearing in 38.9% of shoulder PJIs, followed by CoNS and *S aureus* in 14.8% and 14.5% of cases, respectively.[29] Other organisms that have been isolated in shoulder PJIs included *Enterobacter* (5.9%), *Finegoldia magna* (5.9%), and *Escherichia coli* (6.3%).[30] Polymicrobial infections occur in 11% of shoulder PJIs, whereas culture-negative cases are also relatively common, occurring in 5% to 15% of cases.[30] The literature on TEA microbiologic profile is scarce but shows a similar pattern of high CoNS prevalence (49%) followed by *S aureus* (12%).[31]

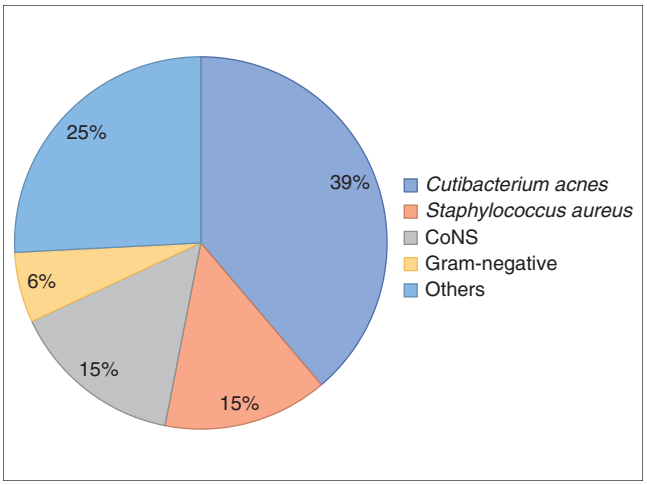

FIGURE 2 Graph showing the organism profile of bacteria commonly isolated from shoulder arthroplasty prosthetic joint infection. CoNS = coagulase-negative staphylococci

A 2020 meta-analysis also studied the prevalence of isolated organisms in spine surgery and found that the organism profile in the spine is similar to that of TJA, with most SSIs attributed to *S aureus* (37.9%) and CoNS (22.7%)[5] (**Figure 3**). Less-frequent organisms identified were *Escherichia* (13%), *Acinetobacter* (10%), *Klebsiella* (8.3%), *Enterococcus* (8.2%), and *Streptococcus* species (6.9%). Interestingly, one study found that approximately 18% of patients undergoing elective anterior cervical diskectomy and fusion had a subclinical infection in the cervical intervertebral disk, with *C acnes* constituting most bacteria.[32] Another study reported that *C acnes* was found to be the most common pathogen in patients who underwent spinal fusion, with late infections manifesting more than 1 year after surgery.[33]

FRIs are complex because of the nature of the trauma mechanism and environment, which creates variability in the microbiologic profile according to the anatomic location or severity of injury (**Figure 4**). *Staphylococcus* organisms are the most common offending organism isolated in FRIs (33.7% to 53.5%),[4,34] with *S aureus* present in 29% to 48%[34,35] and CoNS in 20% to 39%[36] of these patients. Other gram-positive pathogens present in FRI cases include *Streptococcus* and *Enterococcus* species.[35,36] *Enterobacter* species are the most common pathogens isolated from gram-negative monomicrobial FRIs (14% to 27%), whereas anaerobes and culture-negative FRIs make up 16% and 11% of infections, respectively.[36,37] Polymicrobial FRI rates have been reported to range from 14.3% to 57%,[34,38] with higher rates typically found in open fractures; pairings of *Enterobacter/Enterococci*, CoNS/*Enterobacter*, *Enterobacter/Serratia*, and CoNS/*Enterococci* were found to be most prevalent in these cases.[34] A 2018 study revealed that *S aureus* infections were more commonly isolated from FRIs after ORIF of closed fractures compared with open fractures (59% versus 41%, $P = 0.01$), whereas gram-negative organisms were more prevalent in FRIs that developed after treatment of open fractures (54% versus 46%, $P < 0.01$).[35]

RESISTANCE

The widespread use of antibiotics, especially in prophylactic settings, has introduced the emergence of antibiotic-resistant and multidrug-resistant bacterial species, with deaths related to treatment-resistant infection currently estimated to be 700,000 per year and projections estimated to spike to 10 million per year by 2050.[39] The incidence of treatment-resistant bacterial infections including MRSA and vancomycin-resistant *Enterococci*

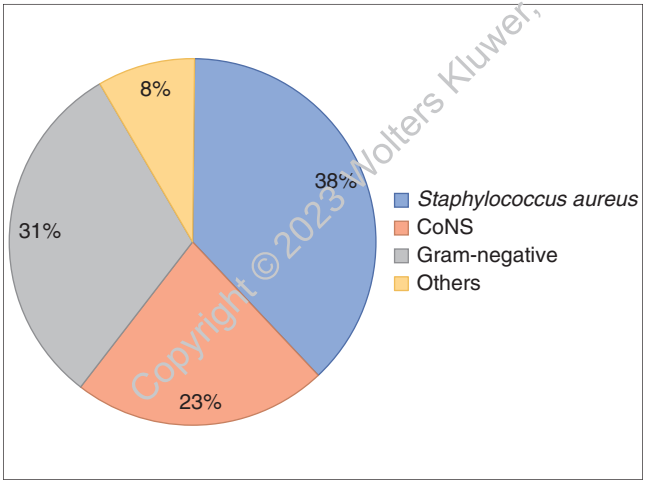

FIGURE 3 Graph showing the organism profile of bacteria commonly isolated from spine surgical site infection. CoNS = coagulase-negative staphylococci

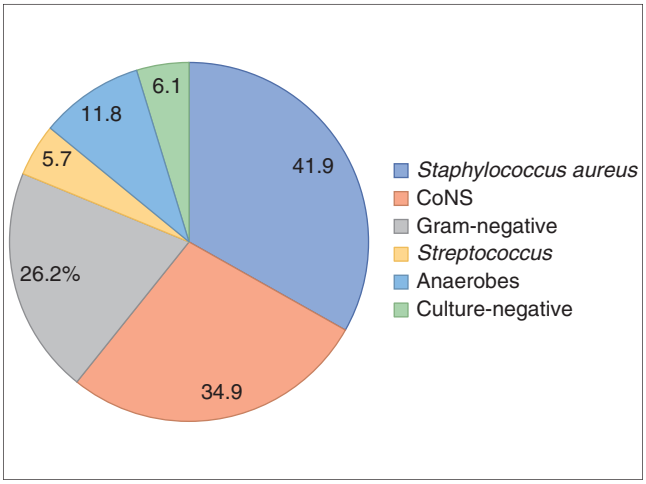

FIGURE 4 Graph showing the organism profile of bacteria commonly isolated from fracture-related infection. CoNS = coagulase-negative staphylococci

in PJI is increasing.[40] A 2018 review of TJA PJI reported that patients initially infected with a multidrug-resistant bacteria may subsequently acquire another treatment-resistant organism with further treatment strategies.[41]

Similarly, resistance to antibiotics used in preoperative prophylaxis is frequently encountered among patients undergoing spine surgery (up to 50% of cases).[42] The inciting resistant organism differs according to the location of surgery in which cefazolin-resistant enteric organisms (58.4% of SSIs) mostly affect the lower thoracic and lumbosacral spine, whereas methicillin-resistant gram-positive organisms (38.9% of SSIs) affect the cervical and upper thoracic spine.[42] Trauma patients are more likely to be in a catabolic state and therefore require longer stays in the intensive care unit and have higher exposure to treatment-resistant organisms. An FRI study found that speciation of at least one treatment-resistant organism occurred in 36% of infected patients, with 32% of the infections caused by MRSA and a smaller number caused by vancomycin-resistant *Enterococci* and multidrug-resistant organisms.[43] MRSA infections are isolated from 25% of open fractures, with a notable upward trend in incidence over time.[44]

ECONOMICS OF SSI

The cost of treating an increasing number of SSIs across all orthopaedic subspecialties places a major financial burden on both the patients and the healthcare industry. In the current healthcare setting, orthopaedic surgeons must recognize the financial burden SSIs impart and focus on delivering high-value surgical outcomes without compromising patient care. Alternative payment models such as the Bundled Payments for Care Improvement from the Centers for Medicare & Medicaid Services have been increasingly used to limit postoperative complications, including SSI, and generate greater value to the system, where the risk of patient complications and/or readmissions are shared with the hospital and clinician, during the entire care episode extending from the patient's admission through a 90-day postdischarge period for a certain diagnosis episode.[10]

For both THA and TKA PJI, costs can increase threefold to fourfold and take approximately twice as long to treat compared with matched patients without postoperative hip or knee PJI. The higher treatment costs of PJI are rooted in longer hospital stays, readmissions, longer course of antibiotic treatment, and extended rehabilitations postoperatively.[45] Treatment costs for TJA infections also vary according to the inciting organism: MRSA PJIs cost substantially more ($100,000) than methicillin-sensitive *S aureus* PJIs ($70,000) ($P < 0.001$).[46] Recent regression models project a national total cost of treatment for THA PJIs of $753.4 million and TKA PJIs to cost approximately $1.1 billion annually by 2030.[47]

Based on the limited data available, the average hospitalization cost to related to postoperative FRI is approximately $20,000, with potential to reach up to $100,000.[9,48] In addition, patients sustain an average income loss of $3,160 during the first year of treatment and accrue a loss of $6,080 per year starting 6 years posttreatment.[9] Taking inflation and FRI rates into account, lost earnings for all patients with FRI would exceed $1 billion per year. However, the window of opportunity to medically optimize trauma patients at higher risks for postoperative complications is nonexistent as in elective procedures. For example, patients undergoing nonelective joint arthroplasty because of trauma had a mean bundle payment loss of $23,122 with 91% of cases exceeding the target price, compared with bundled elective THA cases that generated an average $1,648 net profit per bundle ($P < 0.001$) and only 20% of cases going over target pricing ($P < 0.001$).[49]

Spine surgery also is negatively affected by the exuberant costs for treating SSI that can vary widely, dependent mostly on the procedure, with costs ranging from $16,000 to more than $300,000.[50,51] Treatment expenditures can reach up to 2.36 and 3.78 times higher for cervical and lumbar SSIs, respectively, compared with performing spine surgery for noninfectious etiologies.[51] The costs of treating shoulder arthroplasty PJI are staggering, with higher expenditures attributed to longer length of hospital stay, implant costs, medications, and various clinical tests required.[52] The average cost of treating a TEA PJI with two-stage exchange revision surgery has been reported to be on average twice as much as a primary TEA and 87% higher compared with the cost of revising a TEA for aseptic etiologies.[53]

QUALITY OF LIFE

Little is known about the effect of SSIs on a patient's long-term QoL. One study found that PJI after THA has a negative effect on QoL, including lower EuroQol-5 Dimension-5 Level index score and increased requirements for assisted living and walking aids compared with matched control patients with minimum 10-year follow-up.[54] A systematic review found similar findings in patients who underwent two-stage revision for hip PJI who had substantially lower physical QoL scores but mental health scores comparable with those of the general population after treatment.[55] Recurrence of PJI after treatment predisposes reinfected patients to lower health-related QoL scores compared with patients with successful treatment and no reinfections.[56] However, PJI successfully managed with débridement, antibiotics, and implant retention was not a significant risk factor for poor QoL, but patients sustained similar improvements in 12-Item Short Form scores from prearthroplasty to 12 months postarthroplasty compared with patients in whom PJI did not develop.[11]

Trauma patients who undergo ORIF of a tibial plateau fracture and in whom SSI develops are at higher risk for significantly poorer overall Knee Injury and Osteoarthritis Outcome Score and subscores for pain, activities of daily living, and QoL compared with patients without SSI.[57] Although vertebral osteomyelitis has a high mortality rate and leads to functional disability, surgical treatment leads to significantly improved QoL that remains well below the QoL levels of the general population.[58]

SUMMARY

SSIs are devastating complications that may occur following elective and nonelective orthopaedic surgery. Revision TJA and ORIF for open fractures have one of the highest rates of infection. Most orthopaedic infections are caused by *S aureus* and CoNS organisms except for shoulder arthroplasty infections, which are mostly attributed to *C acnes*. Polymicrobial infection with gram-negative organisms such as *Enterobacter* is isolated frequently from FRI cases after index ORIF of open fractures. Antibiotic-resistant SSIs, including MRSA and vancomycin-resistant enterococci, are becoming increasingly prevalent across different orthopaedic procedures. The cost to treat orthopaedic SSIs is at least double the cost of the index surgery across all subspecialties, with the major cost drivers including readmissions and extended length of hospital stay. Overall, orthopaedic infections even after treatment have a lifelong negative effect on patients' QoL and can diminish functionality with associated long-term disability.

KEY STUDY POINTS

- Prosthetic joint infections range from 0.5% to 3% of cases, with rates reaching up to 8% in elbow arthroplasty.
- The rate of FRIs is 1% to 4%, with much higher rates in open fractures.
- Most orthopaedic infections are caused by *Staphylococcus* species (*S aureus* and CoNS).
- Open fractures have higher rates of gram-negative and polymicrobial infections with *Enterobacter* species most prevalent in these cases.
- Antibiotic-resistant organisms are becoming increasingly prevalent in postoperative SSI.
- The cost of treating postoperative orthopaedic infections is more than double the cost of the primary index surgery, with the main drivers of cost including readmission, extended length of hospital stay, and prolonged antibiotic courses.
- Patients with postoperative infections can sustain loss of income and functionality, along with diminished QoL outcome scores.

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CHAPTER 2

Local Patient Risk Factors

MATTHEW J. DIETZ, MD, FAAOS • KEENAN D. ATWOOD, MD

ABSTRACT

There are numerous modifiable and nonmodifiable risk factors that must be considered before surgical intervention, which can affect the outcome of surgery, specifically the development of surgical site infections. Understanding the potential local bioburden and local risk factors present at the time of surgery can help inform surgeons how to best manage these complex patients to mitigate, if possible, the risk of surgical site infections. These risk factors include local skin/wound breakdown and ulceration, bacterial colonization, and prior trauma or surgery at or near the surgical site.

Keywords: colonization; gunshot wounds; local bacterial burden; prior surgery; skin breakdown

INTRODUCTION

Steps taken to prevent surgical site infection (SSI) and deep infection are of paramount importance, especially when considering the devastating effects these infections can have on patients' overall health and socioeconomic activity. The local surgical environment can be affected by the bacterial burden, skin colonization, skin breakdown, and prior surgeries/trauma at or near the surgical site. To reduce the risk of SSI, modification of the modifiable local risk factors is imperative, whereas the nonmodifiable risk factors pose a conundrum that warrants possible modification of the surgical procedure.

COLONIZATION WITH *STAPHYLOCOCCUS* SPECIES

A normal flora known as the human microbiome exists within the human body, but the microorganisms vary among patients and anatomic regions in each individual patient while often following geographic trends.[1] *Staphylococcus aureus* is the single most common bacterial pathogen responsible for skin and soft-tissue infections in North America, Latin America, and Europe.[1] *Pseudomonas aeruginosa*, *Enterococcus*, *Escherichia coli*, and *Klebsiella* are the next most common pathogens, but their incidence can vary depending on the geographic location of the hospital that the patient is receiving care at.[1] Staphylococci colonization in the nares has been reported in multiple studies to increase the risk of prosthetic joint infection (PJI), however it was not found to be an independent risk factor for infection.[2] Conversely, patients with nasal swabs positive for methicillin-resistant *S aureus* (MRSA) have a significantly higher risk of SSI than noncarriers.[3] A 2018 retrospective single-center review found a nasal colonization rate of 17.5% for methicillin-sensitive *S aureus* and 1.8% for MRSA, with risks for colonization attributed to diabetes, renal insufficiency, and immunosuppression.[4] Similarly, a 2020 review of the spine literature demonstrates increased relative risk (RR) of SSI (RR = 2.52) and MRSA-associated SSI (RR = 6.21) with positive

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MRSA nasal colonization.[5] However, nasal colonization with methicillin-sensitive *S aureus* was not associated with an increased risk of SSI after spine surgery.[5] Other studies revealed similar results with increased rates of SSI following spine surgery when patients were colonized with MRSA compared with those who were colonized with methicillin-sensitive *S aureus* and no colonization.[6,7] Limited evidence exists in the setting of orthopaedic trauma procedures and the role of nasal colonization on postoperative SSI rate. However, some studies indicate an increased odds ratio (weighted OR, 9.9; 95% confidence interval [CI], 4.51-21.79) of SSI with a positive nasal swab.[8,9] Similarly, some studies in sports medicine have addressed this topic. Although one study reported high nasal (90%) and skin (46%) colonization rates, with coagulase-negative *Staphylococcus* as the most commonly identified organism, this has not translated into higher postoperative SSI rates.[10]

The potential for increased SSI risk in the setting of positive nasal colonization has led to the development of decolonization protocols before surgery. Some studies showed that institutional implementation of nasal decolonization programs has led to a decrease in staphylococcal SSI.[3,11,12] A meta-analysis showed that bundling both nasal decolonization and glycopeptide prophylaxis for MRSA carriers decreased SSI rates because of *S aureus* and gram-positive bacteria.[12] Although decolonization procedures have demonstrated a decreased risk for SSI in some studies, others have questioned the durability of decolonization because patients decolonized preoperatively are often recolonized after surgery.[13] Despite these rigorous decolonization protocols, it was reported that some patients remained colonized with MRSA, and in those recalcitrant cases, there was no difference in SSI rates postoperatively.[14]

BACTERIAL/FUNGAL SKIN BURDEN

The bacterial burden present on a patient varies considerably based on multiple factors including the patient's preexisting medical comorbidities.[4] Different areas of the body have different levels of bacterial burden where, for example, the ductal tissue around the periareolar region of the breast has greater bacterial load than the axilla, with the predominant bacteria being *Staphylococcus epidermidis* and *Cutibacterium acnes* (formerly *Propionibacterium acnes*). In a 2021 study, *C acnes* was often implicated in postoperative surgical shoulder infections.[15] A 2018 study and others have reported that regions of the body with a large number of sebaceous glands that can develop acne have been associated with shoulder arthroplasty SSI.[16,17] In the setting of trauma and fracture care, the presence of local bacterial load may influence wound and bone cultures obtained at the time of injury or definitive surgery, but there is little evidence associating this bioburden with postoperative complications including SSI.[18] Advances in next-generation sequencing can potentially shed light on this association and have generated new studies further exploring the effect of trauma, open fractures, and the interplay with the local microbial community on SSI rates.[19]

Skin conditions can also lead to an increased risk of infections because of increased local bacterial loads at or near the surgical site. Psoriasis and the associated psoriatic plaques have increased bacterial density compared with unaffected skin[20] and in some studies have been shown to increase the risk of PJI in total hip arthroplasty (THA).[20] Psoriasis is also thought to increase the risk of postoperative infection in elective foot and ankle surgery.[21] Patients with atopic dermatitis, defined as dryness, erythema, and pruritus, have increased rates of local colonization with *S aureus*, in which the more severe dermatitis cases and acute lesions have higher rates of colonization.[22]

Dermatophytosis, also known as tinea or ringworm, can act as a portal of entry for bacteria in the areas it is present, especially on the foot or inguinal crease. A 2018 study reported that fungal infections are rare but devastating orthopaedic complications can be exceptionally difficult to manage.[23] Reports of implant-related infections consequent to fungus-associated skin conditions are limited, although a 2022 case report highlights the concerns of dermatophytosis associated with relapsing osteomyelitis.[24]

SKIN LESIONS, BOILS, SKIN BREAKDOWNS, AND ULCERATIONS

Streptococci and staphylococci species are common causes of cutaneous infections.[25] Skin breakdown and ulceration after a skin lesion biopsy or excision have been shown to increase the risk of surgical wound infections especially in the setting of total joint arthroplasty (TJA)[26] (**Figure 1**). In addition, in 2018, it was reported that venous insufficiency ulcers and diabetic foot ulcers larger than 10 cm^2, with active exudate and sloughing, are all risks for postoperative infection.[27] Although there is a paucity of evidence associating skin ulceration and breakdown with increased SSI rates in other orthopaedic subspecialties, any skin openings or abnormalities should be fully evaluated and managed before surgical intervention, especially in elective cases.

PRIOR SURGERY IN JOINT/AREA

The anatomic location and extent of prior surgery at or near the surgical site that can vary from open reduction

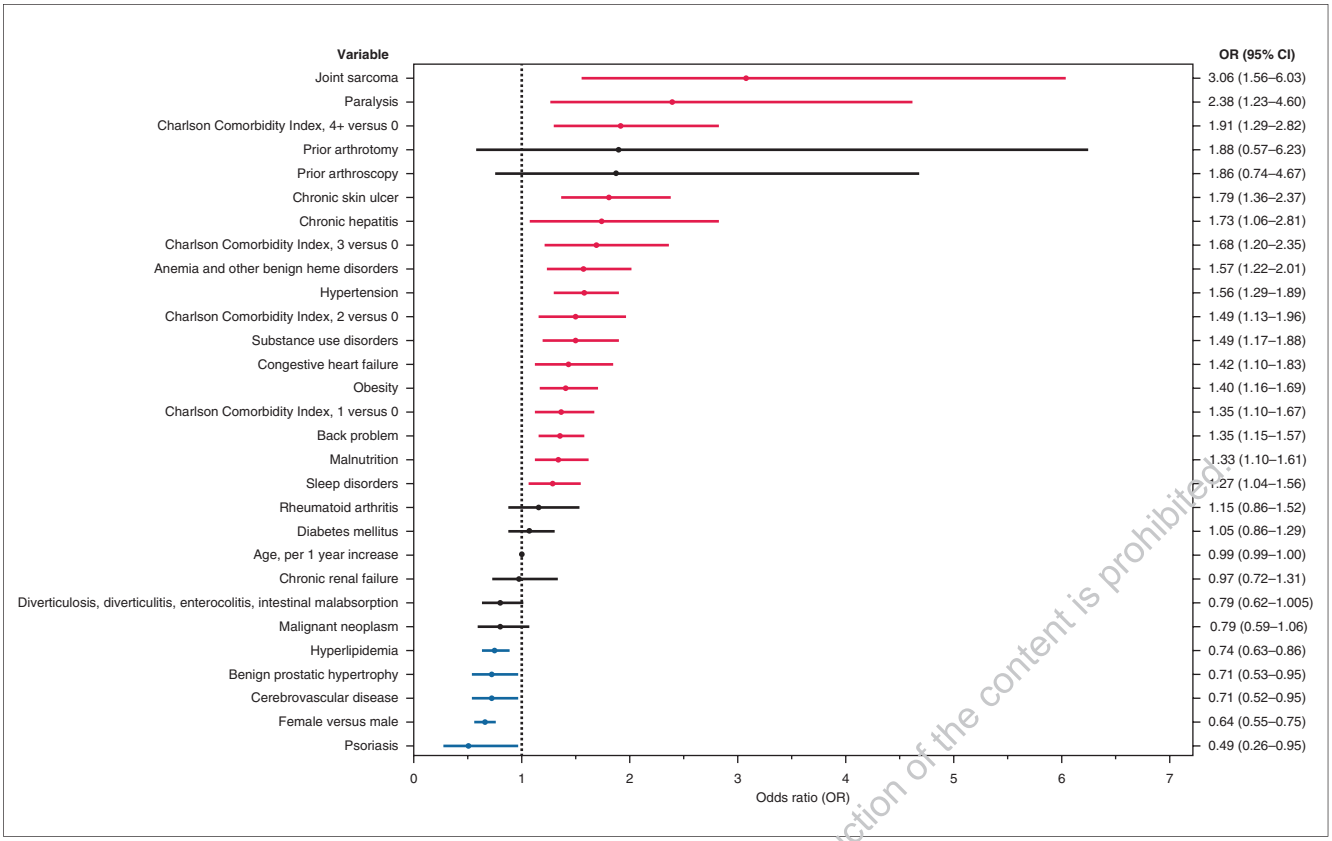

FIGURE 1 Graph showing the multivariate analysis of 147,053 patients undergoing primary total hip and knee arthroplasty, in which prosthetic joint infection (PJI) occurred in 0.5% of patients. The adjusted odds ratio of PJI increased with various skin conditions and prior surgery (CI = confidence interval). (Reprinted from Tande A, Asante D, Sangaralingham L, et al: Risk factors for early hip or knee prosthetic joint infection (PJI): Analysis of a nationwide American insurance claims dataset. *Open Forum Infect Dis* 2017;4(suppl 1):S5, by permission of Oxford University Press.)

and internal fixation (ORIF) to arthroscopic procedures have varying effects on SSI rates after definitive surgery, and in some scenarios, the data are inconclusive. Regarding the knee joint, prior trauma to the joint that leads to subsequent posttraumatic osteoarthritis can increase the risk of PJI after total knee arthroplasty (TKA) compared with TKA performed for primary knee osteoarthritis.[28] Similarly, previous ORIF around the knee with retention of hardware is associated with a significant risk factor for PJI after TKA.[29] In contrast, a 2018 study concluded that although the presence of retained hardware before a TKA in 55 patients increased the risk of postoperative mechanical complications, it did not significantly increase the risk of PJI.[30] A similar study found PJI rates of 0.9% when hardware was removed at the time of TKA after prior ORIF, which is similar to primary TKA PJI rates, therefore advocating performing these cases in a single-stage manner.[31] As described in a 2018 study, the extent of prior surgical intervention can play a key role in postoperative infections where wound complications including SSI were found to be higher in TKAs performed in patients who had undergone a previous ORIF for fracture versus patients who underwent previous knee arthroscopy for soft-tissue injury.[32] Other studies have analyzed the timing of a TKA after arthroscopy and the associated risk of postoperative infection. Studies have concluded that TKA performed within 6 months of arthroscopy can increase the risk of PJI[26,33] (**Figure 2**), whereas another study showed no difference in outcomes including PJI for TKA performed within 1 year of arthroscopy versus more than 1 year afterward.[34] Patients who underwent prior anterior cruciate ligament reconstruction have increased risks of revision surgeries after TKA for infection and other complications compared with patients without prior anterior cruciate ligament reconstruction.[32,35] A review of 35 patients who had undergone osteochondral allograft surgery concluded that this patient population is at increased risk of PJI after TKA, but it should be noted that infection developed only in two patients in the cohort and both had previously undergone multiple knee surgeries.[36]

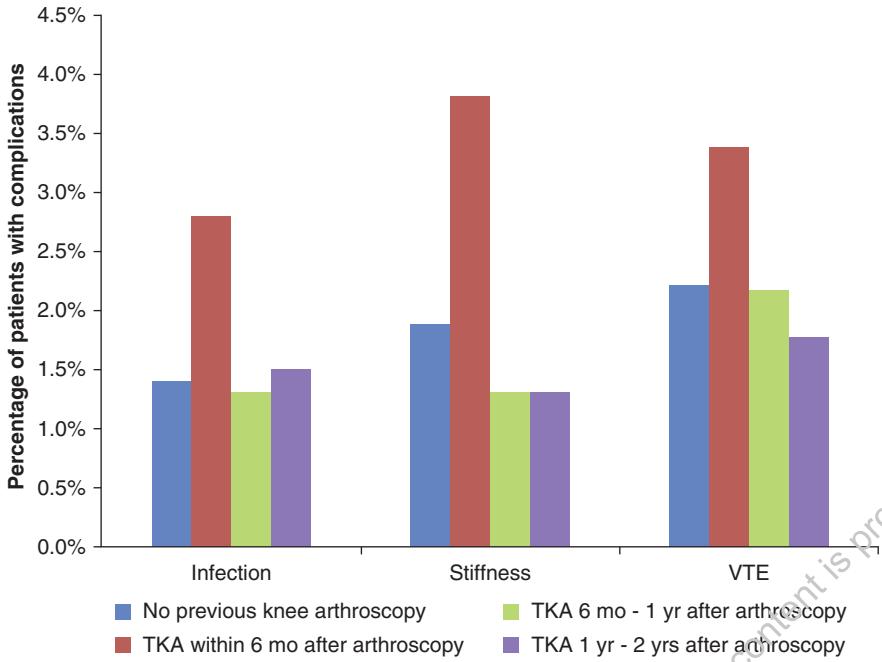

FIGURE 2 Graph showing the incidence of postoperative infection, stiffness, and venous thromboembolism (VTE) after total knee arthroplasty (TKA) with prior knee arthroscopy over specific intervals of time when compared with age-matched control patients. Patients undergoing TKA within 6 months of knee arthroscopy were at significant risk for all complications. (Reprinted from Werner BC, Burrus MT, Novicoff WM, Browne JA: Total knee arthroplasty within six months after knee arthroscopy is associated with increased postoperative complications. *J Arthroplasty* 2015;30[8]:1313-1316. Copyright 2015, with permission of Elsevier.)

Although the knee joint data show a predisposition to higher risk of infections after nonarthroscopic procedures, the hip joint literature is more conflicting and variable. Multiple studies have shown that hip arthroscopy does not increase the risk of infection after THA.[37] There has also been no increase in bacterial contamination for THA in patients who retained hardware from prior hip fracture surgery treated with intramedullary nail, screws, dynamic hip screws, or plates. In 2018, lack of bacterial contamination was evaluated with a preoperative hip aspirate and confirmed with intraoperative cultures.[38] The study also showed no increase in PJI, with only one deep infection in 109 patients.[39] Similar risks for infection were found in patients undergoing THA after rotational acetabular osteotomy compared with the respective control group.[40] Another study found that younger patients who had undergone prior hip salvage/preservation surgery such as pelvic and/or femoral osteotomies or core decompression were at increased risk of superficial infections after THA but no increase in deep infections compared with the control group.[38] In recent studies, patients undergoing a conversion THA after prior acetabular ORIF are at increased risk of PJI.[41,42] A 2022 study showed a PJI rate of 10.3% to 13.3% when hardware was retained in conversion THA after acetabular ORIF.[41] A 2020 retrospective study compared 72 conversion THAs after acetabular ORIF with 215 age-matched control patients and showed an increase in PJI rate of 6.9% compared with 0.5% in the control group.[42]

Prior shoulder surgery predisposes patients to higher risk of infection after total shoulder arthroplasty.[43] One retrospective study showed an increased risk for PJI in shoulder arthroplasty if prior shoulder surgeries such as rotator cuff repair, ORIF, and acromioplasty had been performed.[43] Another study showed that prior failed shoulder arthroplasty increased the risk of PJI for repeat shoulder arthroplasty.[44] Previous spine surgery, whether instrumented or not, has shown heterogenous results regarding infection risk after revision surgery. One study revealed that prior instrumentation has no effect on wound infection or complication rates after three-column osteotomy for thoracolumbar deformities.[45] In contrast, a 2022 report of patients undergoing spinal fusion with a history of retained hardware had increased infection rates and implant loosening compared with the control group.[46]

PRIOR LOCAL INFECTION

A history of prior superficial wound or deep infection at a surgical site can increase the 30-day risk of SSI after primary TJA (OR, 5.0 [95% CI, 2.3-10.9]).[47] Similarly, the risk of PJI after primary TJA increases to 10% if the patient had prior native septic joint.[48] A 2021 multicenter study evaluated risk factors for PJI in patients undergoing TJA who had a prior native septic joint and found that within this group, the risk of PJI after TJA increased in patients who had antibiotic-resistant organisms, who were male, or who had diabetes.[49] Both the timing of TJA from resolution of the initial septic joint infection and the number of prior surgeries to manage the initial infection can play a significant role in PJI development.[48,49] One study evaluated patients with a history of childhood septic hip who then subsequently underwent primary THA and found that all the patients in whom PJI developed had their THA performed within 10 years of them concluding treatment for the septic joint.[50] In contrast, another study concluded that the timing of TKA from resolution of the initial native knee infection was not a risk factor for PJI, but that the number of surgeries required to treat the septic knee was a predisposing factor (3.6 versus 1.6 prior surgeries, $P = 0.006$).[48] A 2021 retrospective study evaluating PJI after TJA in patients with prior septic arthritis revealed that serum markers and timing from septic arthritis to TJA did not affect rates of PJI.[51] A native septic joint can occur with or without concomitant osteomyelitis of surrounding bone and hence creates another level of complexity and poses challenges in preventing PJI after TJA. The presence of osteomyelitis in the setting of native septic joint has been shown to significantly increase the risk of PJI after TJA to approximately 15% compared with cases with isolated native septic joint infection.[52] Infections in other regions of the body distant to the surgical site including PJI of separate joints can increase the risk of PJI after a primary TJA.[53] The spine literature follows similar trends where two systematic reviews found that prior infections in the spine especially from prior surgery pose a significant risk for developing future SSIs along with modifiable risk factors including diabetes, smoking, and obesity.[54,55]

SKIN PREPARATION AND HAIR MANAGEMENT

It is a common practice to remove hair from surgical sites during skin preparation, which is often performed to aid in visualization and improve closure of the wound. However, randomized controlled studies, some of which have been underpowered, have produced conflicting data regarding the relationship between SSI and hair removal before surgery.[56,57] A 2021 study evaluated the different techniques for hair removal and concluded that using a razor increased the risk of SSI when compared with no hair removal, using clippers, or using depilatory cream, but there was no difference in SSI rates between clippers and depilatory cream compared with no hair removal.[56] Although definitive evidence is still lacking robustness for hair removal in mitigating SSI risk, hair removal from the surgical site can be performed outside of the operating room, with clippers or depilatory creams within a time frame that is reasonable and convenient before surgery.[56,57]

PREVIOUS GUNSHOT TRAUMA

Gunshot wounds (GSWs), especially intra-articular, often cause cartilage damage and may lead to posttraumatic osteoarthritis requiring TJA. Bacteria can be displaced from outside the body along the bullet track and into the joint, disproving previous concepts, including autosterilization of a bullet wound.[58] A similar study found that intra-articular low-velocity GSWs to the knee can track debris and bacteria into the joint, potentially serving as a nidus for infection.[59] The data regarding risk of SSI developing after GSW are conflicting. One study noted that posttraumatic osteoarthritis of the hip from a GSW did not increase the risk of PJI after THA,[60] whereas another study concluded that a severe GSW to the knee increased the risk of PJI.[61] GSW to the spine is associated with increased sepsis and SSI rates when colonic injury is involved, but retention of the bullet fragments does not appear to increase the likelihood of sepsis.[62] Recent studies that evaluated GSWs resulting in long bone fractures compared the complication rates of femoral fractures and tibial fractures after GSW with those of blunt trauma with open and closed femoral and tibial fractures and found that although the overall complication rates were higher for GSW, particularly compartment syndrome, the fracture-related infection risk was not significantly different.[63,64]

SUMMARY

The risk of SSI or PJI can be influenced by several local patient risk factors, some of which are modifiable including the colonization of the patient's skin and nares, changes in the overall local bacterial flora that can be influenced by prior surgeries or penetrating injuries, and prior infections at or near the surgical site. Steps to manage and mitigate these risks should be considered to reduce the risk of subsequent infection.

KEY STUDY POINTS

- Colonization with MRSA has been demonstrated to increase the risk of SSI.
- Bacterial colonization can vary depending on geography and anatomic location and is influenced by various skin conditions.
- Careful attention should be paid to local skin conditions such as ulcerations and lesions and dermatologic conditions that can increase the risk of SSI.
- Prior surgeries, GSWs, and history of infection at or near the surgical site can increase the risk of SSI.

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