National Journal of Clinical Orthopaedics

ISSN (P): 2521-3466
ISSN (E): 2521-3474

www.orthoresearchjournal.com
2024; 8(3): 20-22
Received: 21-06-2024
Accepted: 26-07-2024

Maphanga Koali
Tshwane South College, Faculty of Health Sciences, Pretoria, South Africa

Corresponding Author:
Maphanga Koali
Tshwane South College, Faculty of Health Sciences, Pretoria, South Africa

Host factors and their impact on the development of periprosthetic joint infections

Maphanga Koali

DOI: https://doi.org/10.33545/orthor.2024.v8.i3a.448

Abstract
Periprosthetic joint infections (PJI) represent one of the most severe complications following joint arthroplasty, often necessitating complex revision surgeries and prolonged antibiotic therapy. Host factors significantly influence the risk and prognosis of PJI, affecting both the likelihood of infection and the response to treatment. This review explores the impact of various host-related factors, including immunosuppression, comorbidities, obesity, and nutritional status, on the development of PJI, while also discussing emerging strategies to mitigate these risks.

Keywords: Periprosthetic joint, infections, development

Introduction
Periprosthetic joint infections (PJI) are among the most serious complications following joint replacement surgeries, such as total hip and knee arthroplasty. PJIs can result in significant morbidity for patients, leading to prolonged hospital stays, multiple revision surgeries, extended antibiotic therapy, and even long-term disability. They also pose a substantial economic burden on healthcare systems due to the complexity of their treatment. Understanding the factors that contribute to the development of PJIs is essential for preventing their occurrence and improving patient outcomes. Host factors, specifically, play a critical role in the susceptibility to and development of PJIs. Unlike surgical or procedural factors that are related to the technique or materials used in joint replacements, host factors are inherent to the patient's physiology and health status. These include conditions like diabetes mellitus, obesity, immunosuppression, and smoking, all of which compromise the immune system's ability to respond to infections. Patients with these conditions often have impaired wound healing, reduced immune function, or chronic inflammatory states that increase their vulnerability to bacterial colonization and biofilm formation on prosthetic surfaces. Diabetes mellitus, for example, is well-established as a major risk factor for PJIs. Hyperglycemia, a defining feature of diabetes, impairs the function of neutrophils and macrophages, which are essential for controlling infections. In addition, diabetes is associated with microvascular disease, which limits blood flow to the surgical site, further reducing the body's capacity to fight off infections. Similarly, obesity contributes to a pro-inflammatory state, disrupts immune function, and makes surgical procedures more challenging due to increased adipose tissue and technical difficulties during implantation. Immunosuppression, whether due to medical conditions such as rheumatoid arthritis or the use of medications like corticosteroids, also significantly increases the risk of PJIs. Immunosuppressive therapies weaken the body's defenses against infections, making it more difficult to control bacterial growth and prevent the formation of biofilms. Smoking, malnutrition, and systemic inflammation further exacerbate these risks by impairing tissue oxygenation, reducing immune cell efficacy, and promoting an environment conducive to bacterial survival. The interplay between these host factors and the development of PJIs underscores the need for a comprehensive, multidisciplinary approach to patient management. Preoperative optimization of modifiable risk factors, such as controlling blood glucose levels in diabetic patients, promoting smoking cessation, and improving nutritional status, can significantly reduce the likelihood of infection. Moreover, identifying patients who are immunosuppressed or have systemic inflammatory conditions can help

clinicians tailor their perioperative management strategies to minimize the risk of PJI.

Main Objective

The main objective of this paper is to explore the influence of host-related factors on the development of periprosthetic joint infections (PJI) following joint arthroplasty.

Diabetes Mellitus and Hyperglycemia

Diabetes mellitus and hyperglycemia have been established as significant risk factors for the development of periprosthetic joint infections (PJI), particularly following total hip and knee arthroplasty. Hyperglycemia, which commonly occurs in individuals with diabetes mellitus, impairs several critical aspects of the immune system and wound healing processes, leading to a higher likelihood of infection and complications after surgery. In individuals with diabetes, both innate and adaptive immune responses are compromised. Hyperglycemia, the hallmark of diabetes, reduces the effectiveness of neutrophils, which are the body's first line of defense against infections. Neutrophils in hyperglycemic states exhibit diminished chemotaxis, phagocytosis, and intracellular killing of pathogens. This weakened neutrophil response is a critical factor in the susceptibility of diabetic patients to infections, including PJI. Additionally, hyperglycemia adversely affects macrophage function, delaying the clearance of dead cells and reducing the overall inflammatory response needed to combat infection. Another pathway through which diabetes increases the risk of PJI is through its negative impact on wound healing. High blood glucose levels impair collagen synthesis, fibroblast proliferation, and angiogenesis, which are all essential for the effective healing of surgical wounds. Hyperglycemia is also associated with microvascular disease, which further limits the delivery of oxygen and essential nutrients to the surgical site, contributing to delayed wound healing and the formation of chronic wounds. These poorly healing wounds provide an ideal environment for bacterial colonization and infection, significantly increasing the risk of developing PJI. The metabolic dysregulation seen in diabetic patients also promotes an inflammatory state that exacerbates the risk of infection. Elevated blood glucose levels lead to the increased production of advanced glycation end-products (AGEs), which trigger oxidative stress and chronic inflammation. This pro-inflammatory environment not only weakens the immune system but also disrupts the delicate balance required for optimal tissue repair, creating conditions that favor the establishment and persistence of infections such as PJI.

Several studies have provided strong evidence supporting the association between diabetes, hyperglycemia, and PJI. For example, a study by Jämsen *et al*. found that patients with diabetes were at a significantly higher risk of PJI following total knee arthroplasty compared to non-diabetic patients, particularly if their diabetes was poorly controlled. Another study by Bozic *et al*. reported that elevated preoperative hemoglobin A1c levels, indicative of poor long-term blood glucose control, were associated with higher rates of PJI in patients undergoing total hip arthroplasty. These studies underscore the importance of optimizing glycemic control in diabetic patients both before and after surgery to minimize the risk of infection. In addition to long-term glycemic control, perioperative hyperglycemia has been shown to be a critical factor in infection risk. Patients who experience significant elevations in blood glucose levels immediately before or after surgery are at an increased risk of PJI, even if their diabetes is generally well-managed. This phenomenon is thought to be due to the acute stress of surgery, which triggers the release of stress hormones like cortisol, leading to transient hyperglycemia. Given the profound impact that perioperative hyperglycemia can have on infection risk, many clinical guidelines now recommend strict blood glucose monitoring and management during the perioperative period. Studies suggest that maintaining blood glucose levels below 180 mg/dL during the perioperative period significantly reduces the incidence of PJI. In conclusion, diabetes mellitus and hyperglycemia are well-established risk factors for periprosthetic joint infections. Hyperglycemia impairs immune function, delays wound healing, and promotes chronic inflammation, all of which contribute to the increased susceptibility to PJI seen in diabetic patients. Optimizing glycemic control, both long-term and in the perioperative period, is critical for reducing the risk of infection and improving surgical outcomes in this vulnerable population. With the growing prevalence of diabetes worldwide, it is essential that clinicians remain vigilant in managing blood glucose levels in patients undergoing joint arthroplasty to mitigate the risk of PJI.

Systemic Inflammation and Immune Dysregulation

Systemic inflammation and immune dysregulation play pivotal roles in the development and progression of periprosthetic joint infections (PJI). The body's immune response is crucial for preventing infections following joint replacement surgeries; however, when systemic inflammation becomes dysregulated, it can impair this defense mechanism, increasing susceptibility to infections like PJI. Systemic inflammation is typically characterized by an excessive or prolonged inflammatory response that affects the entire body, rather than being localized to a specific site. This condition is often driven by underlying diseases such as rheumatoid arthritis, autoimmune disorders, obesity, or chronic infections, which can result in an overactive or misdirected immune response. In the context of joint replacement surgeries, systemic inflammation can interfere with proper wound healing and immune function, creating an environment that fosters bacterial growth and persistence, leading to PJI. One of the key ways systemic inflammation contributes to PJI is through its impact on immune cell function. Inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) are often elevated in individuals with systemic inflammation. These cytokines, while critical for initiating the body's immune response, can have deleterious effects when present in excess. Chronic elevation of these pro-inflammatory markers can lead to immune exhaustion, where immune cells such as neutrophils, macrophages, and lymphocytes become less effective at responding to pathogens. This impaired immune function allows bacteria to colonize and form biofilms on prosthetic surfaces, a common mechanism through which PJIs occur. Immune dysregulation, often a consequence of systemic inflammation, further complicates the body's defense against infections. In diseases such as rheumatoid arthritis or systemic lupus erythematosus (SLE), the immune system attacks the body's own tissues, causing chronic inflammation. Patients with these conditions frequently require immunosuppressive medications, such as corticosteroids or biologics, to control their symptoms. While these drugs are effective at reducing inflammation, they also weaken the immune system's ability to fight off infections. For example, corticosteroids inhibit the function of phagocytes, cells responsible for engulfing and destroying pathogens. The reduced activity of phagocytes in individuals on immunosuppressive therapies is a major contributor to the higher incidence of PJIs observed in this patient population. Moreover, systemic inflammation can exacerbate the process of

biofilm formation on prosthetic surfaces. Biofilms are structured communities of bacteria that adhere to surfaces and produce a protective matrix, making them highly resistant to antibiotics and the immune system. Inflammatory cytokines can promote the recruitment of immune cells to the site of infection, but these cells often become trapped within the biofilm matrix, rendering them ineffective. Additionally, the inflammation-induced oxidative stress damages surrounding tissues and further impairs the body's ability to clear the infection. Chronic systemic inflammation has also been associated with metabolic syndromes such as obesity and diabetes, both of which are recognized risk factors for PJI. These conditions not only promote a pro-inflammatory state but also impair microcirculation, limiting the delivery of immune cells and antibiotics to the infected site. This poor circulation hinders both the immune response and the efficacy of treatments, making the management of PJIs more challenging in patients with systemic inflammation. In conclusion, systemic inflammation and immune dysregulation are critical factors that contribute to the development of periprosthetic joint infections. By impairing immune cell function, promoting biofilm formation, and interfering with wound healing, systemic inflammation creates an environment that allows infections to thrive. Understanding the relationship between systemic inflammation, immune dysregulation, and PJI is essential for developing strategies to prevent and manage these infections in high-risk patient populations, particularly those with autoimmune disorders or metabolic diseases.

Conclusion

In conclusion, the development of periprosthetic joint infections (PJI) is influenced by a variety of host-related factors, including diabetes mellitus, obesity, immunosuppression, smoking, malnutrition, and systemic inflammation. These factors compromise the immune system's ability to prevent and manage infections, either by impairing wound healing, promoting biofilm formation, or weakening the immune response. While surgical techniques and prosthetic materials play a role in PJI prevention, managing these host factors is crucial in minimizing infection risk. Patients with poorly controlled diabetes or systemic inflammatory diseases are at a particularly high risk due to immune dysfunction and chronic inflammatory states, which foster an environment conducive to infections. In addition, factors such as perioperative hyperglycemia, smoking, and malnutrition can exacerbate this risk, requiring comprehensive preoperative screening and optimization. Emerging strategies, such as personalized treatment plans based on individual risk profiles, are showing promise in reducing PJI incidence. Future research should focus on identifying more targeted interventions that address the specific immune and metabolic pathways involved in PJI development, particularly in high-risk populations. By improving our understanding of how host factors contribute to PJI, clinicians can develop more effective preventative measures and treatment protocols, leading to better long-term outcomes for patients undergoing joint arthroplasty.

Conflict of Interest

Not available

Financial Support

Not available

References

1. Segreti J, Parvizi J, Berbari E, *et al*. Clinical practice guidelines for the diagnosis and management of periprosthetic joint infections. Clin Infect Dis., 2013, 56(1).
2. Jämsen E, Huhtala H, Puolakka T, *et al*. Risk factors for infection after knee arthroplasty: a register-based analysis of 43,149 cases. J Bone Joint Surg Am. 2009;91(1):38-47.
3. Patel VP, Walsh M, Sehgal B, *et al*. Factors associated with prolonged wound drainage after primary total hip and knee arthroplasty. J Bone Joint Surg Am. 2007;89(1):33-38.
4. Stefánsdóttir A, Johansson D, Knutson K, *et al*. Microbiology of the infected knee arthroplasty. Acta Orthop. 2009;80(2):251-257.
5. Bozic KJ, Lau E, Kurtz SM, *et al*. Patient-related risk factors for periprosthetic joint infection and postoperative mortality following total hip arthroplasty in Medicare patients. J Bone Joint Surg. Am. 2012;94(9):794-800.
6. Sculco PK, Abdel MP, Hanssen AD, *et al*. The management of acetabular bone loss in revision total hip arthroplasty: detailed techniques and success with porous metals. J Bone Joint Surg Am. 2013;95(3):230-237.
7. Aggarwal VK, Rasouli MR, Clark CR, *et al*. Surgical site infection and the use of intraoperative vancomycin powder in primary total hip arthroplasty. J Bone Joint Surg Am. 2013;95(24):2104-2111.
8. Rondon AJ, Tan TL, Goswami K, *et al*. Antibiotic prophylaxis in total joint arthroplasty: point of view. J Bone Joint Surg Am. 2017;99(11):854-855.
9. Courville XF, McAnany S, Cheriyan T, *et al*. Smoking cessation and risk of complications after spine surgery: A systematic review. Spine J. 2015;15(5):1019-1028.
10. Malek IA, Royce G, Bhatti SU, *et al*. Factors associated with length of stay in hospital following total knee replacement in patients aged over 75 years. Acta Orthop Belg. 2008;74(4):504-513.
11. Watanabe M, Matsushita T, Nakagawa K, *et al*. Impact of nutritional status on the development of surgical site infections in patients undergoing elective surgery for colorectal cancer. Surgery. 2018;163(4):779-84.
12. Fisman DN, Reilly DT, Karchmer AW, *et al*. Clinical effectiveness and cost-effectiveness of 2-stage exchange for infected total hip arthroplasty. Clin Infect Dis. 2001;32(1):17-27.
13. Aghdassi SJ, Schwab F, Hansen S. Impact of preoperative nutrition on orthopedic surgical site infections. Orthop Res Rev. 2015;7:55-63.
14. Cancienne JM, Werner BC, Browne JA. Obesity and total joint arthroplasty: A literature review. J Arthroplasty. 2017;32(7):2261-6.
15. Goswami K, Parvizi J, Haasper C. Is there a role for antibiotic-loaded cement spacers in the prevention of periprosthetic joint infections? A systematic review. J Bone Joint Surg Am. 2019;101(21):2017-25.
16. Leung F, Richards CJ, Garvin KL. Management of periprosthetic joint infection. J Am Acad Orthop Surg. 2018;26(10).

How to Cite This Article

Koali M. Host factors and their impact on the development of periprosthetic joint infections. National Journal of Clinical Orthopaedics 2024; 8(3): 20-22.