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Association between periodontitis and fracture-related infection in patients with severe open fractures of the upper and/or lower extremities

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Abstract

Background Periodontitis is a chronic inflammatory condition that affects the oral cavity and has systemic implications beyond its localized impact, potentially increasing the risk of various health issues. This study aimed to investigate the role of periodontitis as a risk factor for fracture-related infection (FRI), a severe complication encountered in orthopaedic trauma.

Methods A prospective cohort study was conducted to analyze data from 235 patients with severe open fractures (grades II, IIIA, or IIIB) who were monitored for at least 12 months. During the perioperative period, the periodontal health of these patients was evaluated through various clinical measures, including clinical attachment levels, bleeding on probing (BOP), and probing depth, which were used to identify instances of periodontitis. Additionally, the Simplified Oral Hygiene Index (OHI-S) was employed as part of the assessment. Comprehensive data were collected for subsequent analysis. To explore the associations between periodontal status and the occurrence of FRI, statistical methods, including both univariate and multivariate regression models, were applied FRI, with a significance level set at 5%.

Results Among 235 patients, 169 individuals (71.9%) exhibited normal wound healing, while 66 patients (28.1%) developed FRI. The data analyses indicated a significant association between periodontitis and an increased risk of FRI, with an adjusted risk ratios (RR) of 3.03 (95% CI: 1.02–9.44, $p=0.045$). Additionally, elevated BOP levels were identified as an independent risk factor, with an adjusted RR of 1.02 (95% CI: 1.00–1.04, $p=0.035$). Advanced stages of periodontitis, particularly Stage II (adjusted RR 2.63, 95% CI: 1.11–6.18, $p=0.02$) and Stage III (adjusted RR 3.06, 95% CI: 1.53–6.20, $p=0.001$), were strongly linked to higher rates of FRI. Notably, the presence of periodontitis was significantly associated with early FRI occurrences (proportion difference 0.3658, 95% CI: 0.240–0.491, $p<0.0001$), while no significant association was observed with late or delayed FRI cases. Moreover, additional factors influencing the risk of FRI included the severity of open fracture and delayed wound closure.

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Conclusions Periodontitis was significantly associated with an increased risk of developing FRI in patients with open fractures. Advanced stages of periodontal disease and elevated BOP are recognized as independent risk factors. Additionally, other important contributing factors include the severity of the open fractures and delayed wound closure.

Keywords Periodontitis, Open fracture, Fracture-related infection, Systemic inflammation, Remote infection, Risk factor

Introduction

Periodontitis is a common chronic inflammatory disease that affects the supporting structures of the teeth. If left untreated, it can lead to progressive resorption of the alveolar bone and ultimately result in tooth loss. Globally, periodontitis affects approximately 20–50% of the adult population, with severe cases impacting nearly 11% [1, 2]. This makes it one of the most widespread chronic inflammatory conditions worldwide. The prevalence of periodontitis increases with age and is closely linked to systemic diseases such as diabetes mellitus, cardiovascular disease, rheumatoid arthritis, and osteoporosis, highlighting its significance as a major public health concern [3–6].

The pathophysiology of periodontitis involves a disruption in the homeostasis of oral microbiota, which leads to an imbalance between bacterial colonization and host immune responses. This dysbiosis triggers chronic inflammation and tissue destruction, driven by key periodontal pathogens such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Fusobacterium nucleatum*. These bacteria compromise the integrity of the epithelial barrier, allowing for bacterial translocation and systemic dissemination [4–6]. The resulting inflammatory response is characterized by elevated levels of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β). This contributes to a persistent low-grade inflammatory state and immune dysregulation [6, 7]. Emerging evidence suggests that this systemic inflammation may increase susceptibility to infections beyond the oral cavity.

In the context of surgery, poor oral health or periodontitis has been identified as a significant risk factor for surgical site infection (SSI). This includes infections following periprosthetic joint, elective spinal, and mandibular fractures surgeries. Studies indicate that oral infections contribute to periprosthetic joint infection (PJI), with bacteria from the oral cavity responsible for 6–13% of cases. Furthermore, patients with severe periodontitis who undergo mandibular fracture surgery are over seven times more likely to develop postoperative infections compared to those without periodontal disease [8–10]. These findings suggest a potential link between periodontitis and an increased risk of infection in orthopaedic trauma patients.

Fracture-related infection (FRI) is a major complication in orthopedic trauma, particularly in cases of severe open fractures. Open fractures, accounting for approximately 3-4.5% of all fractures, are highly susceptible to infection due to the direct exposure of bone and soft tissue to the external environment [11]. Despite advances in surgical techniques, infection prevention protocols, and antibiotic prophylaxis, the incidence of FRI in open fractures ranges from 5 to 40% [12, 13]. This variation depends on factors such as the severity of the injury, treatment delays, and individual host factors. *Staphylococcus aureus*, including methicillin-resistant strains (MRSA), is the most commonly isolated pathogen. However, polymicrobial infections, which include Gram-negative bacteria and anaerobes, are also prevalent in high-energy trauma cases [11, 14]. The pathophysiology of FRI involves bacterial biofilm formation, immune evasion, and local tissue necrosis, leading to chronic infections, delayed bone healing, and nonunion of the fracture [15]. Systemic factors, such as diabetes, smoking, malnutrition, and immunosuppression, are well-established contributors to the risk of FRI [16, 17]. However, the association between periodontitis and FRI remains unclear, with limited research directly investigating this relationship.

This study aims to explore the association between periodontitis and the risk of FRI in patients with severe open fractures. By elucidating this potential link, we seek to improve infection prevention strategies and optimize patient outcomes in the field of orthopedic trauma.

Methods

This prospective cohort study was approved by the Research Ethics Committee of Khon Kaen University, in accordance with the 1975 Declaration of Helsinki and its revised 2013 version. Clinical trial number: not applicable. The study included patients with open fractures classified as Gustilo-Anderson types II, IIIA, or IIIB [18], who were treated at a single tertiary regional hospital from August 2022 to September 2023. Informed consent to participate was obtained through a clear written consent form, which provided patients with information about the research objectives, data collection, potential risks and benefits.

Inclusion and exclusion criteria

The inclusion criteria for the study were patients over the age of 18 who had open fractures of the upper and/or lower extremities, specifically grade II, IIIA, or IIIB open fractures. The exclusion criteria were as follows: (i) fractures associated with compartment syndrome or amputation, (ii) previous surgery, FRI, or chronic osteomyelitis, (iii) open fractures of the distal phalanges of the fingers and toes, (iv) a history of any periodontal treatment, including supragingival and subgingival scaling, root planning, mouth rinses, and antibiotic usage within the previous month, (v) having ≤ 10 teeth, (vi) patients on ventilator support, (vii) patients with maxillofacial injuries, and (viii) vulnerable patients. Participants were monitored for at least 12 months [19]. The flow chart of the participant's recruitment in the study is summarized in Fig. 1.

Data source

Data regarding demographic characteristics, lifestyle behaviors, and general and oral health conditions were gathered through structured interviews conducted by trained researchers. Additional information was obtained from the participants' medical records, which included details such as gender, age, BMI, smoking status, medical history, mechanism of injury, open fracture grade, time to antibiotics (in hours), time to surgery (in hours), instances of delayed wound closure, and the use of wound vacuum dressing (Supplementary Material 1).

Oral health status was assessed by a trained dentist who was blinded to the participants' clinical status. The oral examination was conducted perioperatively within 48 h of hospital admission, and relevant clinical data were obtained from patient records. The assessment included the following parameters: probing depth (PD), clinical attachment level (CAL), bleeding on probing (BOP), and the Simplified Oral Hygiene Index (OHI-S) [20]. BOP and PD were recorded for all teeth present, excluding third molars, at six sites per tooth: mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual. CAL was measured at the same six sites, but only when a probing depth of ≥ 4 mm was detected. This methodology, adapted from the diagnostic criteria established by Gomes-Filho et al. [21], aimed to identify clinically significant periodontal deterioration while maintaining diagnostic specificity for severity classification. The dental examiner underwent calibration through replicated periodontal measurements, using an experienced periodontist as the reference. The interrater reliability, measured using Cohen's kappa coefficient (k), was 0.83 for PD, 0.81 for CAL, and 0.79 for BOP. The intraexaminer agreement for these measurements was 0.86, 0.88, and 0.85, respectively.

Study variables

Independent variable-Periodontitis

The primary independent variable in this study was the diagnosis of periodontitis, categorized as either present or absent. This diagnosis was based on the criteria established by Gomes-Filho et al. [21], which include considerations of PD, CAL, and the presence of BOP. This criterion also defines the severity levels of periodontitis.

Individuals were diagnosed with severe periodontitis if they had at least four teeth with at least one site showing a PD of 5 mm or greater, CAL of 5 mm or greater at the same site, and the presence of BOP. Moderate periodontitis was identified when at least four teeth had at least one site with a PD of 4 mm or greater, CAL of 3 mm or greater at the same site, and BOP. Mild periodontitis was defined as having at least four teeth with at least one site with a PD of 4 mm or greater, CAL of 1 mm or greater at the same site, and BOP.

The first secondary independent variable was mean PD, measured in millimeters, defined continuously. The total of all PD measurements taken from the patient's teeth was calculated to determine the average. The second secondary independent variable was BOP, defined dichotomously as either the presence or absence of bleeding when probing. The BOP index was calculated by dividing the number of sites where bleeding was observed by the total number of available sites in the mouth, then multiplying by 100 to express the result as a percentage. The third secondary independent variable was oral hygiene status, represented by the OHI-S [20]. This index comprised two components: the Simplified Calculus Index (CI-S) and the Simplified Debris Index (DI-S), which assess the amount of calculus and debris found on the tooth surface. The OHI-S score was categorized as follows: good (score 0-1.2), moderate (1.3-3.0), and poor (3.1-6.0).

Dependent variable-FRI

The primary dependent variable was FRI, defined dichotomously based on FRI confirmatory criteria outlined in the international consensus definition. FRI was identified by the presence of at least one of the following criteria: (i) a fistula, sinus, or wound breakdown with communication to the bone or implant; (ii) purulent drainage from the wound or the presence of pus during surgery; (iii) pathogens that were phenotypically indistinguishable from culture results obtained from at least two separate deep tissue or implant specimens collected during surgery; (iv) microorganisms identified in deep tissue through histopathological examination using specific staining techniques for bacteria or fungi [22]. Patients who healed normally and did not exhibit any clinical confirmatory or microbiological criteria for infection were classified as having normal healing (NH).

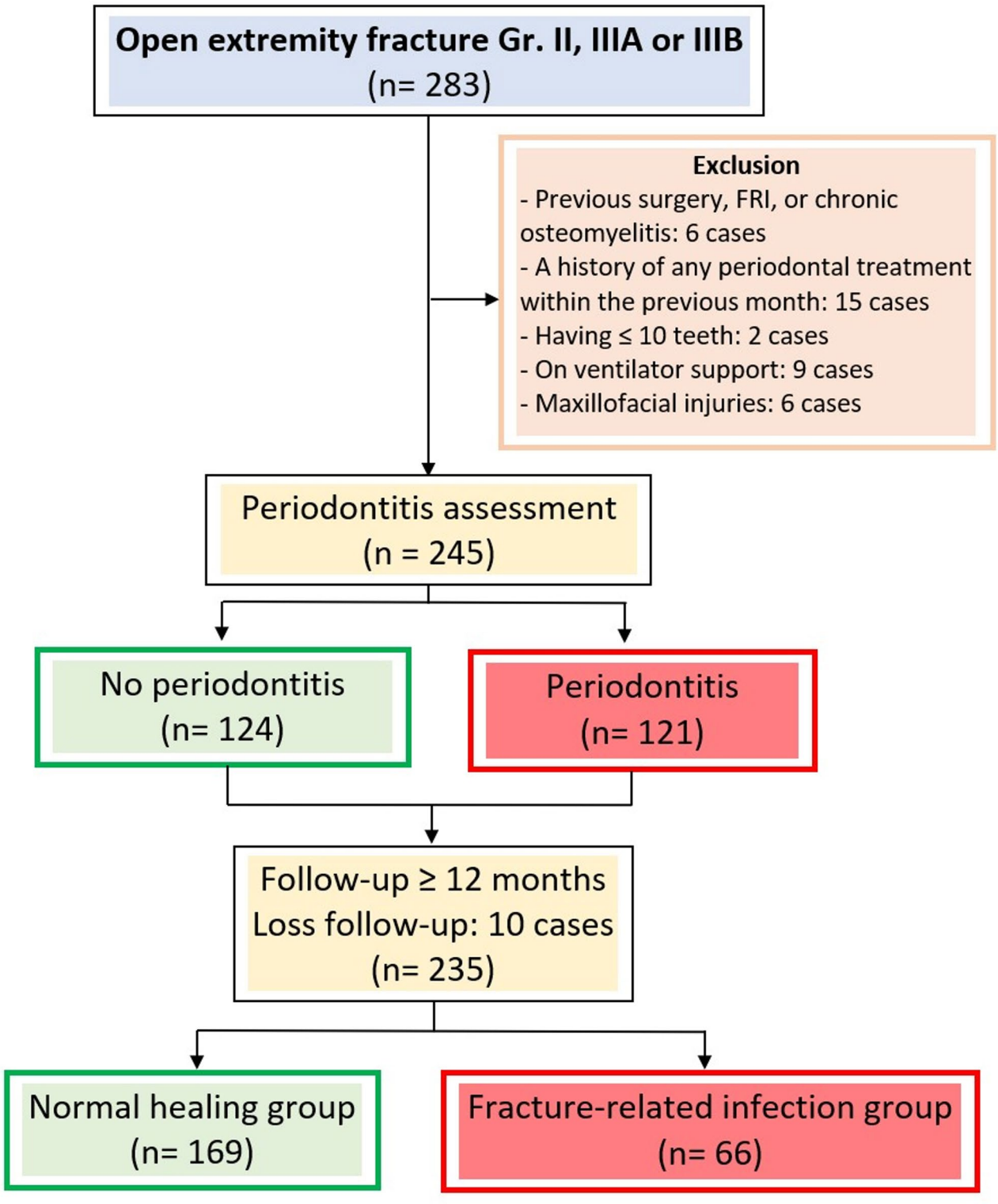

Fig. 1 Flow diagram for study participants

The secondary dependent variable was a type of FRI, categorized based on the timing of onset: early (0–2 weeks), delayed (2–10 weeks), and late (> 10 weeks). This classification was based on by the time interval between the initial injury and the clinical presentation of infection [23].

Covariables

Data were collected from medical records and clinical measurements, which served as covariables in a conceptual theoretical model investigating the association between periodontitis and FRI in patients with open fractures. The demographic characteristics recorded included gender, age, BMI, smoking status, medical history, mechanism of injury, grade of open fracture, time to antibiotic administration (in hours), time to surgery (in hours), instances of delayed wound closure, and the use of wound vacuum dressing.

Table 1 Demographic and clinical characteristics of patients in both groups

The demographic and clinical characteristics of patients	Normal healing ($n=169$)	Fracture-related infection ($n=66$)	*P*-value
Sex			
Male	129 (76.33)	48 (72.73)	0.565[a]
Female	40 (23.67)	18 (27.27)	
Age (Year) Mean (S.D.)	42.86 (15.80)	43.21 (17.65)	0.885[b]
BMI Mean (S.D.)	22.98 (4.14)	23.59 (4.60)	0.322[b]
Smoking			
No	85 (50.30)	36 (54.55)	0.565[a]
Yes	84 (49.70)	30 (45.45)	
Comorbid			
No	131 (77.51)	48 (72.73)	0.852[c]
Hypertension	11 (6.51)	5 (7.58)	
Diabetes mellitus	8 (4.73)	4 (6.06)	
Heart disease	1 (0.59)	0 (0.00)	
Other	18 (10.65)	9 (13.64)	
Mechanism of injury			
Car accident	10 (5.92)	7 (10.61)	**0.024**[c]
Motorcycle accident	94 (55.62)	45 (68.18)	
Pedestrian injury	0 (0.00)	1 (1.52)	
Fall from height	9 (5.33)	1 (1.52)	
Other	56 (33.14)	12 (18.18)	
Open fracture grade			
GradeII	84 (49.70)	4 (6.06)	**< 0.001**[a]
Grade IIIA	75 (44.38)	39 (59.09)	
GradeIIIB	10 (5.92)	23 (34.85)	
Time to antibiotics (Hours) Mean (S.D.)	4.36 (3.86)	3.67 (2.93)	0.194[b]
Time to surgery (Hours) Mean (S.D.)	19.83 (8.91)	23.88 (13.05)	**0.021**[b]
Delay wound closure			
No	165 (97.63)	46 (69.70)	**< 0.001**[a]
Yes	4 (2.37)	20 (30.30)	
Wound Vacuum Dressing			
No	169 (100)	63 (95.45)	**0.021**[c]
Yes	0 (0.00)	3 (4.55)	

Significant values are in bold

[a] Pearson chi-squares test, [b] Independent T-test, [c] Wilcoxon rank sum test with continuity correction

Statistical analysis

The clinical characteristics of patients with and without FRI were compared using the chi-square test for categorical variables and the Student's t-test for continuous variables. To identify independent factors that predict FRI, log-binomial regression analysis was conducted using the logistf package in R (R program Ver. 4.4.2) (https://www.r-project.org). This statistical modeling employed Firth's penalized likelihood technique to address issues related to small sample sizes and sparse data, allowing for the estimation of risk ratios (RR) and their corresponding confidence intervals. This method offered a more intuitive measure of association and enhanced the robustness of the findings. Statistical significance was defined at a p-value of <0.05. For sample size estimation, the method proposed by Hsieh, Bloch, and Larsen for multiple logistic regression was adapted to provide an accurate approximation for calculating sample size specific to log-binomial regression [24]. Based on this formula, the required sample size for log-binomial regression was 219 participants [25]. Accounting for a 10% dropout rate, the total sample size required was 243 participants.

Results

A total of 245 patients with grade II, IIIA, or IIIB open fractures were admitted for operative intervention. Out of these, 235 patients were included in the analysis after accounting for those lost to follow-up. The patients were divided into two groups: the NH group ($n=169$) and the FRI group ($n=66$). The demographic and clinical characteristics of the patients are summarized in Table 1. There were no significant differences in sex distribution, age, BMI, smoking status, or the presence of comorbidities between the two groups. However, significant differences were noted in the mechanism of injury ($p=0.024$), with motorcycle accidents and falls being more common in the FRI group. Moreover, the open fractures were significantly more severe in the FRI group, with a higher proportion classified as Grade IIIA or IIIB ($p<0.001$). Patients in the FRI group experienced longer times to surgery, with a mean duration of 23.88 ± 13.05 h compared to 19.83 ± 8.91 h in the NH group ($p=0.021$). They also had higher rates of delayed wound closure, with 30.3% affected compared to only 2.37% in the NH group ($p<0.001$). Additionally, vacuum-assisted wound closure dressings were used more frequently in the FRI group, at 4.55% versus 0% in the NH group ($p=0.021$). Within this

Table 2 Multivariate analysis of risk factors associated with fracture-related infection

Factor	Crude RR (95% CI)	Adjusted RR (95% CI)	*P*-value[a]
Periodontitis			
No	Reference	Reference	**0.045**
Yes	1.92 (1.08–3.48)	3.03 (1.02–9.44)	
BOP	1.01 (1.00–1.03)	1.02 (1.00–1.04)	**0.030**
Pocket depth (mm)	1.06 (0.78–1.42)	0.76 (0.41–1.36)	0.361
OHI-S score	1.22 (0.98–1.54)	1.18 (0.82–1.73)	0.351
Age	1.00 (0.98–1.01)	0.97 (0.94–0.99)	0.051
BMI	1.03 (0.96–1.10)	1.06 (0.97–1.17)	0.152
Smoking			
No	Reference	Reference	0.528
Yes	0.84 (0.47–1.48)	0.77 (0.97–1.17)	
Comorbidity			
No	Reference	Reference	0.177
Hypertension	1.29 (0.41–3.64)	2.73 (0.62–11.62)	0.530
Diabetes mellitus	1.43 (0.39–4.55)	1.67 (0.32–8.39)	0.191
Heart disease	0.90 (0.006–17.23)	18.87 (0.10–566.28)	0.207
Other	1.39 (0.57–3.19)	2.05 (0.66–6.09)	
Mechanism of injury			
Car accident	Reference	Reference	0.651
Motorcycle accident	0.66 (0.24–1.88)	0.74 (0.20–2.74)	0.783
Pedestrian injury	4.20 (0.19–648.81)	0.58 (0.01–107.37)	0.344
Fall from height	0.22 (0.02–1.30)	0.34 (0.02–2.99)	0.154
Other	0.31 (0.10–0.98)	0.36 (0.08–1.47)	
Open fracture grade			
Grade II	Reference	Reference	
Grade IIIA	9.82 (3.88–31.46)	7.46 (2.46–27.12)	**0.0002**
Grade IIIB	42.02 (13.80–156.15)	5.12 (0.94–28.44)	**0.049**
Time to antibiotics (Hours)	0.94 (0.86–1.02)	1.01 (0.90–1.11)	0.838
Time to surgery (Hours)	2.12 (1.29–3.48)	1.05 (1.01–1.10)	**0.020**
Delay wound closure			
No	Reference	Reference	**0.003**
Yes	16.21 (6.02–53.79)	9.55 (2.04–51.35)	
Wound Vacuum Dressing			
No	Reference	Reference	0.849
Yes	18.68 (1.77–2525.79)	1.37 (0.07–218.03)	

Significant values are in bold

BOP: Bleeding on probing, OHI-S: Simplified oral hygiene index, [a] Log binomial regression

cohort, the overall incidence rate of FRI was 28.09% (66 out of 235 patients). The incidence rates for each fracture type were as follows: type II at 1.56% (4 out of 88), type IIIA at 34.21% (39 out of 114), and type IIIB at 69.70% (23 out of 33). Most cases of FRI presented with early-onset symptoms, with 62.12% (41 out of 66) occurring within less than 2 weeks. To provide additional context for interpreting periodontitis as the primary independent variable, we have included Supplementary Material 2.

Table 3 Univariate analysis of the effect of dental parameters associated with fracture-related infection

Dental parameters	Normal healing (*n* = 169)	Fracture-related infection (*n* = 66)	Mean difference (95% CI)	*P*-value
BOP	38.00 (21.22)	46.53 (21.26)	8.53 (2.39–14.67)	**0.007**[a]
Pocket depth (mm)	2.34 (0.95)	2.39 (0.86)	0.05 (-0.20–0.30)	0.695[b]
OHI-S score	2.74 (1.28)	3.08 (1.25)	0.33 (-0.02–0.70)	0.068[a]
DI-S score	1.76 (0.79)	1.98 (0.76)	0.21	0.055[a]
CI-S score	0.92 (0.53)	1.06 (0.53)	(-0.004–0.44)	0.077[a]
			0.13 (-0.01–0.29)	
Periodontitis (n (%))	90 (0.79)	24 (0.21)	Proportion	**0.029**[b]
No	79 (0.65)	42 (0.35)	difference	
Yes			0.14 (0.01–0.26)	

Significant values are in bold

BOP: Bleeding on probing, OHI-S: Simplified Oral Hygiene Index, DI-S: Simplified Debris Index, CI-S: Simplified Calculus Index, [a] Independent t test, [b] Proportion test

The multivariate analysis of risk factors associated with FRI is detailed in Table 2. Periodontitis emerged as a significant independent risk factor; patients with periodontitis had an adjusted RR of 3.03 (95% CI: 1.02–9.44, $p = 0.045$). Additionally, elevated BOP showed a significant association, with an adjusted RR of 1.02 (95% CI: 1.00–1.04, $p = 0.035$). The analysis also revealed that higher grades of open fracture, delayed wound closure, and prolonged time to surgery significantly increased the risk of infection in the multivariate model.

The univariate analysis of dental parameters in relation to FRI is presented in Table 3. Among these parameters, BOP and periodontitis showed a significant association with FRI, with p-values of 0.007 and 0.029, respectively. Notably, patients within the FRI group exhibited significantly higher mean BOP scores (46.53 ± 21.26) compared to those in the NH group (38.00 ± 21.22, $p = 0.007$). In contrast, no significant differences were observed in measures such as PD, OHI-S, DI-S, or CI-S between the two groups. In this study, the overall prevalence of periodontitis was 121 out of 235 patients (54.49%). However, periodontitis was more prevalent in the FRI group, with 42 out of 66 patients (63.63%) affected, compared to 79 out of 169 patients (46.75%) in the NH group. The difference in proportion was 0.14 (95% CI: 0.01–0.26), $p = 0.029$, indicating a significant association between periodontitis and FRI.

The univariate analysis investigating the association between the severity of periodontitis and the risk of FRI is summarized in Table 4. Patients with periodontitis had a significantly increased risk of FRI, with a crude RR of 1.97 (95% CI: 1.11–3.56, $p = 0.02$) compared to those without periodontitis. Furthermore, when the data was stratified by severity level, both moderate and severe

Table 4 Univariate analysis of periodontitis severity level associated with fracture-related infection

Factors	Fracture-related infection Crude RR (95% CI)	*P*-value[a]
Periodontitis		
No	Reference	**0.02**
Yes	1.97 (1.11–3.56)	
Periodontitis severity level		
No periodontitis	Reference	
Mild	0.71 (0.25–1.77)	0.48
Moderate	2.63 (1.11–6.18)	**0.02**
Severe	3.06 (1.53–6.20)	**0.001**

Significant values are in bold

[a] Log binomial regression

Table 5 Univariate analysis of the periodontitis associated with type of fracture-related infection

Type of fracture-related infection	Periodontitis (*N*, %)	Proportion difference (95% CI)	*P*-value[a]
Early	No 13, (31.71) Yes 28, (68.29)	0.3658 (0.240–0.491)	**< 0.0001**
Delayed	No 9, (47.37) Yes 10, (52.63)	0.1112 (-0.031–0.253)	0.1277
Late	No 3, (50.00) Yes 3, (50.00)	0 (−0.148–0.148)	> 0.999

Significant value are in bold

[a] Proportion test

periodontitis showed markedly increased risks of FRI, with RR of 2.63 (95% CI: 1.11–6.18, $p = 0.02$) and 3.06 (95% CI: 1.53–6.20, $p = 0.001$), respectively.

The univariate analysis examining the relationship between periodontitis and the type of FRI according to the Willenegger and Roth classification [23] is presented in Table 5. For early FRI, the difference in proportions was 0.3658 (95% CI: 0.240–0.491), indicating a significant association between periodontitis and early FRI. However, there were no significant differences in the proportion of patients with periodontitis when comparing delayed or late FRI to the reference group (NH group).

Discussion

The key findings of this study indicate a positive and statistically significant association between the presence of periodontitis and the diagnosis of FRI in patients with open fractures undergoing in-hospital surgical procedures. This is consistent with a systematic review and meta-analysis, which concluded that periodontitis is a risk factor for overall postoperative complications after surgical procedures, with an odds ratio of 4.76. This suggests that patients with periodontitis are at a higher risk of developing complications following surgery [26]. However, a retrospective study conducted by Nakamura et al. was the only one that did not find any connection between periodontal disease and postsurgical complications [27].

In this study, the prevalence of periodontitis was 54.49%, with 121 out of 235 patients diagnosed. This figure is lower than the pooled prevalence reported in Trindade et al.'s systematic review, which estimated that between 2011 and 2020, approximately 61.6% of dentate adults were affected by periodontitis, with severe periodontitis affecting 23.6% of individuals [28]. The Global Burden of Disease 2021 study reported that over 1 billion people worldwide suffer from severe periodontitis. Since 1990, the burden of periodontitis has nearly doubled, showing significant increases in both prevalence and total cases [1]. This trend reflects the growing global importance of periodontitis as a major public health issue over the past three decades.

Our analysis revealed a significant association between periodontitis and the risk of developing a FRI. Patients with periodontitis had an increased risk of FRI, with an adjusted RR of 3.03 (95% CI: 1.02–9.44, $p = 0.045$) based on multivariate analysis. BOP was identified as a significant independent risk factor for FRI, showing an adjusted RR of 1.02 (95% CI: 1.00–1.04, $p = 0.030$). BOP indicates periodontal tissue inflammation caused by bacterial accumulation beneath the gumline. A high BOP score suggests chronic periodontitis, which can lead to bacteremia. On the other hand, the lack of a significant association between OHI-S and FRI suggests that the severity of periodontitis may be a stronger determinant of infection risk than overall oral hygiene. Although there were no significant differences in mean OHI-S and DI-S scores between the two groups, patients who had FRI were categorized as having poor OHI-S scores (3.1-6.0) and DI-S scores (1.9-3.0), whereas those in the NH group had fair OHI-S scores (1.3-3.0) and DI-S scores (0.7–1.8). This suggests that plaque accumulation may indirectly contribute to the infection risk in patients with FRI.

There are several established criteria for defining periodontitis, including those set forth by the Centers for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) [29], as well as the collaborative criteria developed by the AAP and the European Federation of Periodontology (EFP) [30]. This study specifically adopted the diagnostic criteria proposed by Gomes-Filho et al. [21], which are widely used in periodontal medicine research. These criteria are particularly valued for their high specificity in association studies, thereby minimizing the potential for false-positive diagnoses [31].

Our findings aligned with previous research demonstrated a significant association between periodontal disease and SSI. Various studies have explored insights into the underlying mechanisms and quantified risks. For instance, Mirzashahi et al. found a significant association

between periodontal disease and SSI in elective spinal surgeries, reporting an odds ratio (OR) of 2.9 ($p = 0.049$). The study emphasized that bacteremia and chronic inflammation caused by periodontal pathogens, such as *P. gingivalis* and *F. nucleatum*, contribute to systemic inflammation and impaired immune responses, increasing the risk of SSI [8]. Similarly, Nobuhara et al. demonstrated that perioperative oral management significantly reduced the risk of SSI in colorectal cancer surgeries, achieving an OR of 0.484. This reduction in SSI was attained through various interventions, including professional mechanical teeth cleaning, dental plaque and calculus removal, tongue coating removal, and patient self-care instructions. The study also observed that patients who received two or more sessions of perioperative oral care experienced significantly fewer SSI (7.16%) compared to those who received only one session (9.21%) or no oral care at all (17.0%). Furthermore, the average postoperative hospital stay was shorter for the group that received oral care, demonstrating the broader benefits of these interventions beyond just SSI prevention [32].

Oral infections are known to be a potential cause of PJI. Rakow et al. found that 12% of hematogenous PJI were attributed to oral infections, with the oral cavity recognized as a significant source of bacteremia. The study highlighted those oral pathogens, such as *Streptococcus mitis* and *Streptococcus sanguinis*, can enter the bloodstream and adhere to prosthetic surfaces, promoting biofilm formation. These biofilms lead to persistent infections that are difficult to treat due to their antibiotic resistance and the host's immune responses [9]. Moreover, Young et al. supported this association by noting that 6–13% of PJI were linked to bacteria originating from the oral cavity. Also, dental caries that progress to pulpitis and periapical periodontitis (forming apical abscesses) involve chronic bacterial infection. These infections are thought to arise from transient bacteremia caused by common activities like chewing or brushing, particularly in patients with poor oral hygiene [33]. Fenske et al. further emphasized the importance of preoperative dental screening, observing a significant reduction in early PJI when standardized referral protocols were used to manage oral infections prior to surgery. The study reported an OR of 0.43, demonstrating that patients who underwent preoperative dental screening had a significantly lower risk of developing early PJI compared to those who did not [34]. Additionally, Schmalz and Ziebolz proposed two hypotheses to explain how oral infections contribute to PJI. First, during the early healing phase after prosthetic joint surgery, which occurs within the first three months, an acute exacerbation of an oral infection, such as dental abscess, can lead to bacteremia and systemic inflammation. This can disrupt the healing process and result in an infection. Second, for late-stage PJI, systemic immunological imbalances create conditions that allow oral pathogens, such as *P. gingivalis* and *F. nucleatum*, to colonize periprosthetic tissues after an initial phase of periprosthetic inflammation. These hypotheses underline the critical role of local and systemic immune barriers, with oral pathogens acting as opportunistic colonizers rather than primary initiators of infection [35]. While recent findings by Simon et al. suggest that antibiotic prophylaxis prior to dental procedures does not significantly reduce the risk of periprosthetic joint infection in arthroplasty patients, their study also emphasizes the importance of considering the baseline oral health status of patients. In contrast to general prophylactic measures, our findings point to chronic periodontitis—characterized by sustained inflammation and microbial dysbiosis—as a possible contributor to early postoperative infections, highlighting a potential role for targeted preoperative periodontal assessment and management rather than routine prophylaxis alone [36].

No prior studies have investigated the association between periodontitis and FRI in the context of open fractures. Severe open fractures are significantly different from elective surgeries due to the extensive tissue damage involved, and most cases require implant fixation. In our study, early FRI was the most frequently observed type of infection, and periodontitis was significantly associated with early FRI, which occurred within the first two weeks postoperatively. This finding suggests that the inflammatory burden and transient bacteremia induced by periodontitis play a crucial role in the early postoperative phase when the immune system is recovering from trauma and surgery. However, no significant association was found between periodontitis and delayed or late FRI, indicating that other factors, such as implant biofilm formation, impaired soft tissue healing, and immune system adaptations, become the dominant contributors in the later stages of infection. Our hypothesis is that the acute inflammatory response following severe trauma, combined with the chronic inflammatory state associated with periodontitis, impairs local defenses, delays wound healing, and creates a favorable environment for bacterial colonization and biofilm formation on implants or damaged bone surfaces.

Moreover, our analysis revealed that the severity of periodontitis is a critical factor in relation to postoperative infection risk. Moderate and severe periodontitis were significantly associated with higher FRI risk (RR 2.63, $p = 0.02$, and RR 3.06, $p = 0.001$, respectively). This suggests a threshold effect, where only advanced stages of periodontitis generate sufficient systemic inflammation to influence the risk of postoperative infection. In contrast, mild periodontitis did not show a significant association, likely because it does not induce the same level of systemic immune response or bacteremia.

This finding is consistent with the research conducted by Janaphan et al., which revealed a strong association between periodontal staging and postoperative SSI. They also found that patients with severe periodontitis (stages III and IV) were over seven times more likely to develop SSI after mandibular fracture surgeries (OR: 7.17, 95% CI: 2.95–17.41, $p < 0.001$). The study highlighted that severe periodontitis significantly exacerbates the risk of postoperative infections due to increased bone loss and the systemic inflammatory burden it imposes [10]. The connection between periodontitis and FRI is likely complex and involves multiple factors. Chronic periodontal inflammation leads to elevated systemic levels of pro-inflammatory cytokines, such as IL-6, TNF-α, and IL-1β. These cytokines are known to hinder both wound healing and immune function [5]. Furthermore, periodontal bacteria have been implicated in distant-site infections due to their ability to enter the bloodstream and adhere to compromised tissues [37], including orthopaedic implants and fracture sites. It is also important to note that periodontal disease is part of the oral health index, which includes other conditions like dental caries and apical periodontitis. Therefore, other dental-related factors may complicate the relationship between periodontal disease and its connection to FRI.

This study demonstrates a significant association between periodontitis and FRI in patients with severe open fracture, particularly during the early postoperative period. Importantly, other critical risk factors identified include high-grade open fracture and delayed wound closure. Patients with Grade IIIA or IIIB fractures had significantly higher infection rates, supporting previous findings that emphasize the importance of early debridement and soft tissue coverage. Delayed wound closure increased the risk of FRI nearly tenfold, underscoring the importance of timely wound management in preventing infection. Lu et al. reported infection rates as high as 27% for grade III fractures [38]. Moreover, delays in wound closure significantly increased the risk of FRI. Delays longer than seven days impair local immunity and promote bacterial growth [16, 39].

Clinical implication, proactive oral health management is crucial for identifying and treating active periodontal disease and other oral infections, as it can help prevent a range of systemic diseases and SSI. However, this study has several limitations. First, the single-center design may limit the generalizability of the findings to other populations and healthcare settings. Second, although we accounted for known confounders, unmeasured variables, such as nutritional status, serum albumin, and blood glucose levels, were not collected in our study. These factors are known to influence immune function and wound healing and they may have contributed to the risk of FRI. For example, diabetes mellitus is a well-documented predictor of postoperative infection (RR 1.72) due to impaired immune function and delayed wound healing [25]. Similarly, although nutritional status was not directly assessed in our study, it may influence infection risk through its effects on immune competency [40]. Third, the diagnosis of periodontitis relied on clinical criteria rather than microbiological or biomarker-based methods, which could have influenced the accuracy of severity classification. Finally, the observational nature of the study makes it challenging to establish causality. This emphasizes the need for further research, including prospective and interventional studies, to validate these findings and explore targeted prevention strategies for FRI.

Conclusion

This study demonstrates a significant association between periodontitis and FRI in patients with severe open fractures, particularly during the early postoperative period. This finding suggests that periodontitis may play a contributory role, which warrants further investigation in future prospective and interventional studies. Importantly, other critical risk factors identified include high-grade open fractures (Grade IIIA and IIIB) and delayed wound closure. These factors significantly increase the risk of infection and should be carefully considered in clinical management.

Abbreviations

AAP	American Academy of Periodontology
BOP	Bleeding On Probing
CAL	Clinical Attachment Level
CDC	Centers for Disease Control and Prevention
CI-S	Simplified Calculus Index
DI-S	Simplified Debris Index
FRI	Fracture-Related Infection
IL-1β	Interleukin-1 beta
IL-6	Interleukin-6
MRSA	Methicillin-Resistant Strains
NH	Normal Healing
OHI-S	Oral Hygiene Index-Simplified
OR	Odds Ratio
PD	Probing Depth
PJI	Periprosthetic Joint Infection
RR	Risk Ratios
SOHI	Simplified Oral Hygiene Index
SSI	Surgical Site Infection
TNF-α	Tumor Necrosis Factor-Alpha

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

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Author contributions
SSu and SL established the study's conception, design, and contributed to collection of data, analysis, interpretation of the results, and drafting of the manuscript. SSi contributed to data analysis. SSJ, DS and TC contributed to manuscript formatting and critically revising the manuscript. All authors gave final approval and agreed to be accountable for the work done, ensuring its integrity and accuracy.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration

Ethics approval and consent to participate
This study was conducted in accordance with the principles of the Declaration of Helsinki and International Conference on Harmonization Good Clinical Practice (ICH-GCP). Approval was granted by the Ethics Committee of Khon Kaen University (Jul 12, 2022; No. HE651205) and Khon Kaen Hospital (Aug 8, 2022; No. KEMOU65024). Participants gave informed consent to be involved in the study.

Consent for publication
All participants gave informed consent for publication of anonymized data.

Competing interests
The authors declare no competing interests.

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References

1. Nascimento GG, Alves-Costa S, Romandini M. Burden of severe periodontitis and edentulism in 2021, with projections up to 2050: the global burden of disease 2021 study. J Periodontal Res. 2024;59(5):823–67.
2. Genco RJ, Sanz M. Clinical and public health implications of periodontal and systemic diseases: an overview. Periodontol 2000. 2020;83(1):7–13.
3. Dioguardi M, Bizzoca ME, Cantore S, Caloro GA, Musella G, Mastrangelo F, et al. Impact of cerebrovascular stroke on inflammatory periodontal indices: a systematic review with meta-analysis and trial sequential analysis of case-control studies. Front Oral Health. 2024;5:1473744.
4. Hajishengallis G, Chavakis T. Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. Nat Rev Immunol. 2021;21(7):426–40.
5. Martínez-García M, Hernández-Lemus E. Periodontal inflammation and systemic diseases: an overview. Front Physiol. 2021;12:709438.
6. Wu Q, Zhang W, Lu Y, Li H, Yang Y, Geng F, et al. Association between periodontitis and inflammatory comorbidities: the common role of innate immune cells, underlying mechanisms and therapeutic targets. Int Immunopharmacol. 2024;128:111558.
7. Łasica A, Golec P, Laskus A, Zalewska M, Gędaj M, Popowska M. Periodontitis: etiology, conventional treatments, and emerging bacteriophage and predatory bacteria therapies. Front Microbiol. 2024;15:1469414.
8. Mirzashahi B, Tonkaboni A, Chehrassan M, Doosti R, Kharazifard MJ. The role of poor oral health in surgical site infection following elective spinal surgery. Musculoskelet Surg. 2019;103(2):167–71.
9. Rakow A, Perka C, Trampuz A, Renz N. Origin and characteristics of haematogenous periprosthetic joint infection. Clin Microbiol Infect. 2019;25(7):845–50.
10. Janaphan K, Hashem I, Smith C, Holmes S, Chatzopoulou D. Periodontal disease as a primary cause of surgical site infection in fractures of the mandible: is smoking a confounding variable? Br J Oral Maxillofac Surg. 2022;60(10):1424–9.
11. Whiting PS, Obremskey W, Johal H, Shearer D, Volgas D, Balogh ZJ. Open fractures: evidence-based best practices. OTA Int. 2024;7(3 Suppl):e313.
12. Rupp M, Walter N, Bärtl S, Heyd R, Hitzenbichler F, Alt V. Fracture-Related Infection-Epidemiology, etiology, diagnosis, prevention, and treatment. Dtsch Arztebl Int. 2024;121(1):17–24.
13. Metsemakers WJ, Moriarty TF, Morgenstern M, Marais L, Onsea J, O'Toole RV, et al. The global burden of fracture-related infection: can we do better? Lancet Infect Dis. 2024;24(6):e386–93.
14. Craxford S, Vris A, Ahluwalia R, Saini A, Harrison WD, Graham S, et al. Fracture related infection in open tibial fractures. J Orthop. 2024;51:98–102.
15. Sukpanichyingyong S, Sae-Jung S, Stubbs DA, Luengpailin S. Microbiota shifts in fracture-related infections and pathogenic transitions identified by 16S rDNA sequencing. Sci Rep. 2025;15(1):7732.
16. Iliadis AD, Shivji F, Debuka E, Trompeter A, Narayan B, Heidari N. Current concepts in the prevention, diagnosis and treatment of fracture-related infection (FRI). Eur J Orthop Surg Traumatol. 2021;31(5):957–66.
17. Masters EA, Ricciardi BF, Bentley KL, de Moriarty M, Schwarz TF, Muthukrishnan EM. Skeletal infections: microbial pathogenesis, immunity and clinical management. Nat Rev Microbiol. 2022;20(7):385–400.
18. Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 1976;58(4):453–8.
19. Zalavras CG, Aerden L, Declercq P, Belmans A, Metsemakers WJ. Ninety-Day Follow-up is inadequate for diagnosis of Fracture-related infections in patients with open fractures. Clin Orthop Relat Res. 2022;480(1):139–46.
20. Greene JC, Vermillion JR. THE SIMPLIFIED ORAL HYGIENE INDEX. J Am Dent Assoc. 1964;68:7–13.
21. Adeyemi AT. Clinical diagnosis criteria for periodontal disease: an update. JDHODT [Internet]. 2018 Sep 17 [cited 2025 Jan 22];9(5). Available from: https://medcraveonline.com/JDHODT/critical-appraisal-of-systematic-and-narrative-reviews-of-literature-in-the-field-of-orthodontics.html
22. Metsemakers WJ, Morgenstern M, McNally MA, Moriarty TF, McFadyen I, Scarborough M, et al. Fracture-related infection: A consensus on definition from an international expert group. Injury. 2018;49(3):505–10.
23. Willenegger H, Roth B. [Treatment tactics and late results in early infection following osteosynthesis]. Unfallchirurgie. 1986;12(5):241–6.
24. Hsieh FY, Bloch DA, Larsen MD. A simple method of sample size calculation for linear and logistic regression. Stat Med. 1998;17(14):1623–34.
25. Kortram K, Bezstarosti H, Metsemakers WJ, Raschke MJ, Van Lieshout EMM, Verhofstad MHJ. Risk factors for infectious complications after open fractures; a systematic review and meta-analysis. Int Orthop. 2017;41(10):1965–82.
26. Cueto Urbina A, Guzmán Opazo J, Sagredo Ramírez K, Parra Parra M, De López S. Association between periodontitis and postoperative complications in hospital medical surgical procedures: a systematic review. Rev Cient Odontol (Lima). 2023;11(4):e177.
27. Nakamura Y, Tagusari O, Seike Y, Ito Y, Saito KI, Miyamoto R, et al. Prevalence of periodontitis and optimal timing of dental treatment in patients undergoing heart valve surgery. Interact Cardiovasc Thorac Surg. 2011;12(5):696–700.
28. Trindade D, Carvalho R, Machado V, Chambrone L, Mendes JJ, Botelho J. Prevalence of periodontitis in dentate people between 2011 and 2020: A systematic review and meta-analysis of epidemiological studies. J Clin Periodontol. 2023;50(5):604–26.
29. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. J Periodontol. 2007;78(7 Suppl):1387–99.

30. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal of a new classification and case definition. J Periodontol. 2018;89(Suppl 1):S159–72.
31. Batista JET, Figueiredo ACMG, Souza ES, Lyrio AO, Passos-Soares JS, Trindade SC, et al. Excessive weight in pregnant women increases the chance of diagnosing periodontitis combined with tooth loss. J Periodontol. 2024;95(11):1097–108.
32. Nobuhara H, Yanamoto S, Funahara M, Matsugu Y, Hayashida S, Soutome S, et al. Effect of perioperative oral management on the prevention of surgical site infection after colorectal cancer surgery: A multicenter retrospective analysis of 698 patients via analysis of covariance using propensity score. Med (Baltim). 2018;97(40):e12545.
33. Young H, Hirsh J, Hammerberg EM, Price CS. Dental disease and periprosthetic joint infection. J Bone Joint Surg Am. 2014;96(2):162–8.
34. Fenske F, Kujat B, Krause L, Meyer S, Sander AK, Repmann J, et al. Preoperative dental screening can reduce periprosthetic infections of hip and knee endoprostheses in the first month after surgery: results of a cohort study. Infection. 2024;52(2):535–43.
35. Schmalz G, Ziebolz D. The hypotheses of oral disease-associated perioprosthetic joint infections to understand the role of oral health for endoprostheses - a narrative review. Spec Care Dentist. 2024;44(2):328–38.
36. Simon SJ, Aziz AA, Coden GS, Smith EL, Hollenbeck BL. Antibiotic prophylaxis prior to dental procedures after total hip and knee arthroplasty does not decrease the risk of periprosthetic joint infection. J Arthroplasty. 2024;39(9S2):S420–4.
37. de Andrade KQ, Almeida-da-Silva CLC, Coutinho-Silva R. Immunological pathways triggered by Porphyromonas gingivalis and Fusobacterium nucleatum. Therapeutic Possibilities? Mediators Inflamm. 2019;2019:7241312.
38. Lu V, Zhang J, Patel R, Zhou AK, Thahir A, Krkovic M. Fracture related infections and their risk factors for treatment Failure-A major trauma centre perspective. Diagnostics (Basel). 2022;12(5):1289.
39. Marais LC, Zalavras CG, Moriarty FT, Kühl R, Metsemakers WJ, Morgenstern M. The surgical management of fracture-related infection. Surgical strategy selection and the need for early surgical intervention. J Orthop. 2024;50:36–41.
40. Rong A, Franco-Garcia E, Zhou C, Heng M, Akeju O, Azocar RJ, et al. Association of nutrition status and hospital-acquired infections in older adult orthopedic trauma patients. JPEN J Parenter Enter Nutr. 2022;46(1):69–74.

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