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A Case-Control Study on Risk Factors for Early Dental Implant Failure in Smokers and Non-Smokers

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Abstract

A case-control study evaluated risk factors associated with early dental implant failure in smokers versus non-smokers. Seventy patients with early failure (implant loss or mobility within 12 months) were matched by age and implant site to 140 controls with successful implants at 12 months. Baseline data included smoking status, implant characteristics (length, diameter, surface), bone quality, and perioperative antibiotic use. Inflammatory markers, including peri-implant crevicular fluid interleukin-1β (IL-1β) and C-reactive protein (CRP), were measured at prosthesis placement. Smokers had a significantly higher failure rate (OR 3.2; 95% CI 1.8–5.5; $p < 0.001$). Elevated IL-1β levels (> 150 pg/mL) strongly predicted failure (OR 2.9; CI 1.6–5.1; p=0.001), as did poor bone quality (Lekholm grade III–IV) (OR 2.5; CI 1.4–4.5; p=0.002). No significant association was observed with implant dimensions or antibiotic prophylaxis. Smokers had higher IL-1β and CRP levels compared to non-smokers. Multivariate analysis confirmed smoking status, elevated IL-1β, and low bone quality as independent predictors. This study emphasizes that smoking significantly elevates early implant failure risk, exacerbated by inflammatory burden and compromised bone quality. Preventive strategies should include smoking cessation, assessment of peri-implant inflammation, and careful bone evaluation prior to implantation.

Keywords

Dental implant failure; smoking; peri-implant inflammation; interleukin-1β; bone quality

Introduction

Dental implants have become a standard and successful modality for tooth replacement; however, early implant failure—defined as failure to achieve osseointegration within 12 months—remains a clinical challenge. Risk factors are multifactorial, including patient-related variables such as smoking, systemic diseases, bone quality, and implant-specific factors. Smoking has long been implicated as a major risk determinant due to its negative effects on wound healing, vascularity, and immune response. Existing literature consistently documents higher failure rates in smokers compared to non-smokers; yet, few studies robustly control for confounding variables or analyze inflammatory mediators in the peri-implant environment.[1-4]

Peri-implant crevicular fluid interleukin-1β (IL-1β) and systemic markers such as C-reactive protein (CRP) reflect local inflammation and systemic response to implantation. Elevated IL-1β levels have been associated with early peri-implant infection and delayed osseointegration. Nevertheless, the differential expression of peri-implant inflammatory markers in smokers and their prognostic value for early implant failure remains underexplored.[5-7]

Bone quality at the implant site, graded using the Lekholm and Zarb classification, modulates primary stability and osseointegration. Poor bone quality (grade III-IV) may predispose implants to micro-movement, impaired vascular supply, and higher failure risk. Existing case series and retrospective cohorts have hinted at elevated failure in low-quality bone; however, prospective controlled studies offering matched comparisons are limited.[8-10]

This case-control study was designed to identify risk factors for early dental implant failure, with particular focus on smoking, peri-implant inflammation (IL-1β, CRP), and local bone quality. Matching on age, implant location, and surgical protocol allowed isolation of key variables. Hypothesis: smoking increases early failure risk, mediated through elevated IL-1β and systemic inflammatory burden, and poorly mineralized bone further elevates failure probability. Understanding these interactions may improve preoperative risk assessment, patient counselling, and postoperative monitoring to reduce early implant loss.

Methodology

This case-control study enrolled 70 adult patients aged 25–65 who experienced early implant failure within 12 months of placement at Bakhtawar Amin Medical and Dental College, matched

2:1 with 140 controls who had functional implants at 12 months. Matching criteria included age (±5 years), implant site (maxilla/mandible and region), and implant system and surgeon. Baseline data included smoking status (current smoker ≥10 cigarettes/day for ≥1 year), bone quality at insertion (Lekholm grade I–IV), implant length, diameter, surface characteristics, and perioperative antibiotic regimen. At prosthesis connection (approx. 3 months post-placement), crevicular fluid was collected adjacent to implant and IL-1β quantified by ELISA. Serum CRP was measured concurrently. Failed implants were assessed via radiographs and clinical mobility. All participants received standardized surgical protocols and postoperative care. Exclusion criteria included uncontrolled diabetes, history of bisphosphonate use, irradiated bone, immunosuppression, periodontal disease, and poor oral hygiene. Ethical approval was obtained; verbal informed consent was documented. Statistical analysis employed conditional logistic regression for matched pairs. IL-1β and CRP values were compared via paired t-test or Wilcoxon signed-rank test. Variables significant in univariate analysis ($p < 0.05$) were included in multivariate model. Odds ratios (OR) with 95% CI were reported.

Results

Table 1. Demographic and Implant Characteristics by Failure Status

Variable	Failures (n=70)	Controls (n=140)	p-value
Current smokers (%)	38 (54%)	38 (27%)	<0.001*
Poor bone quality III–IV (%)	40 (57%)	45 (32%)	0.002*
Implant length ≤10 mm (%)	24 (34%)	44 (31%)	0.65
Implant diameter ≤3.5 mm (%)	30 (43%)	55 (39%)	0.57
Antibiotic prophylaxis (%)	68 (97%)	136 (97%)	0.88

Higher failure is associated with smoking and poor bone quality.

Table 2. Inflammatory Markers: IL-1β and CRP Levels

Marker	Failures (mean ± SD)	Controls (mean ± SD)	p-value
IL-1β (pg/mL)	180 ± 45	130 ± 30	<0.001*
CRP (mg/L)	4.2 ± 1.3	3.1 ± 1.0	<0.001*

Elevated peri-implant IL-1β and serum CRP preceded implant failure.

Table 3. Multivariate Conditional Logistic Regression Predicting Early Implant Failure

Predictor	Odds Ratio (OR)	95% CI	p-value
Smoking (current)	3.2	1.8–5.5	<0.001*
IL-1β >150 pg/mL	2.9	1.6–5.1	0.001*
Bone quality grade III–IV	2.5	1.4–4.5	0.002*

Smoking, elevated IL-1β, and poor bone quality were independent risk factors.

Discussion

Consistent with prior observational evidence, current smokers exhibited a more than threefold increased risk of early dental implant failure. Smoking's deleterious effects on neo-vascularization, osteoblast activity, and immune response likely impair osseointegration. The independent association with failure after controlling for bone quality and inflammation confirms smoking as a pivotal risk factor in implant survival.[11-13]

Elevated peri-implant IL-1β levels in failed cases underscore the role of local inflammation in implant prognosis. IL-1β is a key cytokine in peri-implantitis and inflammatory bone resorption. Findings indicate that smokers have significantly higher IL-1β even at prosthesis placement, suggesting subclinical inflammatory processes are already underway and predictive of future failure.[14-15]

Systemic inflammation, as measured by CRP, was also elevated in the failure group, though IL-1β had stronger predictive value. Elevated CRP may reflect systemic response to inflammation or smoking-induced immune activation. While CRP offers a broader systemic picture, IL-1β provides focused insight into site-specific biocompatibility and early bone-implant interface health.[16-18]

Bone quality remained an independent predictor. Poor mineralization (Lekholm grade III–IV) compromises primary stability and vascular supply, rendering implants more susceptible to micromotion and bacterial colonization. Even among non-smokers, low bone quality increased failure risk, reinforcing the need for careful preoperative bone assessment.[19-20]

Interaction between smoking and bone quality likely compounds risk. Smokers with poor bone quality exhibited highest failure probability. Preoperative risk profiling should consider both variables. In clinical practice, smoking cessation should be encouraged prior to implantation, and bone augmentation or alternative treatment planning considered for low-quality bone sites.

No significant associations were observed with implant dimensions or perioperative antibiotic use. This suggests that host factors outweigh mechanical specifications in early failure. Standard prophylactic antibiotic protocols were uniformly applied and thus did not influence outcomes in this cohort.

These findings advocate for routine peri-implant IL-1β assessment as a prognostic biomarker. Elevated levels at prosthesis placement may identify patients who require closer monitoring, anti-inflammatory interventions, or delayed loading protocols. Smokers and patients with poor bone quality could benefit from adjunctive peri-implantitis prevention strategies, such as biomaterial selection or guided bone regeneration.

Limitations include case-control design, which does not permit causal inference, and reliance on IL-1β and CRP at a single timepoint. Future longitudinal studies measuring serial inflammatory markers and including microbiome analysis would deepen understanding. Confirmation in larger multi-center cohorts would support generalizability.

Conclusion

Smoking status, elevated peri-implant IL-1β, and poor bone quality independently predict early dental implant failure. Assessment of these risk factors enables tailored surgical planning and proactive interventions to enhance implant survival. Smoking cessation and inflammation containment should be integral to pre-implant evaluation and postoperative management.

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