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Correlation Between Histopathological Findings and Clinical Causes of Dental Implant Failure

[1]Priya Nagar, [2]Stiti Pragnya, [3]Swati Choudhary, [4]Preetika Yadav,[5]Swapnil Kurhade,[6]Rohit Nandan

[1]Professor, Department of Prosthodontics and Crown & Bridge, Post Graduate Institute of Dental Sciences, Pt B D Sharma University of Health Sciences, Rohtak, Haryana, India nagarpriya78@gmail.com
[2]Associate Professor, Department of Dentistry, Pandit Raghunath Murmu Medical College and Hospital, Baripada, Odisha, India pragnya5764@gmail.com
[3]Professor, Department of Oral & Maxillofacial Pathology and Oral Microbiology, ITS Dental College, Hospital & Research Centre, Greater Noida, Uttar Pradesh, India, dr_swati007@yahoo.co.in
[4]Assistant Professor, Department of Pediatric & Preventive Dentistry, Faculty of Dental Sciences, SGT University, Budhera, Gurugram, Haryana, India preetika_fdsc@sgtuniversity.org
[5]Assistant Professor, Department of Prosthodontics and Crown & Bridge, Dr D Y Patil Dental College & Hospital, Pimpri, Dr D Y Patil Vidyapeeth, Pimpri, Pune, Maharashtra, India swpnl31@gmail.com
[6]Senior Lecturer, Department of Prosthodontics and Crown & Bridge, Teerthanker Mahaveer Dental College & Research Centre, Moradabad, Teerthanker Mahaveer University, Uttar Pradesh, India rohitsingh877@gmail.com
Corresponding author: Priya Nagar, nagarpriya78@gmail.com

ABSTRACT:

Background

Dental implant failure is a multifactorial clinical problem involving biological, mechanical, and prosthetic factors. Histopathological examination of failed implants may provide important information regarding the tissue changes underlying implant failure and may help distinguish biologically mediated from mechanically related complications.

Aim

To evaluate the clinical causes and histopathological characteristics of dental implant failure and to identify clinical and histopathological factors independently associated with biologically mediated implant failure.

Materials and Methods

This study evaluated 100 patients with dental implant failure. Demographic and clinical characteristics, including age, gender, smoking status, diabetes mellitus, history of periodontal disease, and implant location, were recorded. The clinical causes of implant failure were categorized as peri-implantitis, lack of osseointegration, mechanical overload, implant fracture, prosthetic complications, infection/abscess, and other causes. Histopathological examination assessed inflammatory infiltrates, fibrous connective tissue formation, bone resorption, necrotic tissue, foreign-body giant cells, and reduced or absent osseointegration. Associations between clinical causes and histopathological findings were evaluated using the Chi-square test or Fisher's exact test. Spearman's correlation analysis assessed relationships between histopathological characteristics and clinical severity. Multivariable logistic regression was performed to

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identify independent predictors of biologically mediated implant failure. Statistical significance was established at $p < 0.05$.

Results

Peri-implantitis was the most frequent clinical cause of implant failure (32.0%), followed by lack of osseointegration (24.0%) and mechanical overload (16.0%). Chronic inflammatory infiltrate was the most common histopathological finding (62.0%), followed by bone resorption (53.0%) and lymphoplasmacytic infiltration (48.0%). A significant association was observed between clinical failure causes and histopathological findings (probability (p) = 0.044). Reduced osseointegration demonstrated the strongest correlation with clinical severity Spearman's rank correlation coefficient (r) = 0.61, $p < 0.001$), followed by bone resorption ($r = 0.58$, $p < 0.001$) and chronic inflammatory infiltrate ($r = 0.52$, $p < 0.001$). In multivariable analysis, reduced osseointegration (adjusted Odds Ratio (OR) = 4.76, $p < 0.001$), bone resorption (adjusted OR = 4.12, $p < 0.001$), chronic inflammatory infiltrate (adjusted OR = 3.26, $p = 0.003$), history of periodontal disease (adjusted OR = 2.74, $p = 0.005$), and smoking (adjusted OR = 2.18, $p = 0.030$) were significant independent predictors of biologically mediated implant failure.

Conclusion

Dental implant failure was predominantly associated with peri-implantitis and impaired osseointegration. Histopathological evidence of inflammation, bone resorption, and reduced osseointegration was strongly associated with clinical severity. Periodontal disease history and smoking were also significant clinical predictors of biologically mediated failure, emphasizing the importance of periodontal and risk-factor assessment in implant therapy.

INTRODUCTION

Dental implants have become a widely accepted option for replacing missing teeth, offering functional rehabilitation and reliable prosthetic support [1]. Advances in implant design, surgical techniques, surface characteristics, and maintenance protocols have improved treatment predictability; however, implant failure remains a clinically relevant complication [2]. Implant failure may arise from several biological and mechanical factors, including impaired osseointegration, peri-implant infection, mechanical overload, implant fracture, and prosthetic complications [3].

Biological complications are particularly important because persistent inflammatory reactions may compromise the tissues surrounding dental implants and contribute to progressive peri-implant tissue destruction [4]. Peri-implantitis is an important inflammatory complication characterized by pathological changes in the peri-implant tissues and progressive supporting bone loss [5]. In contrast, failure of osseointegration may involve inadequate bone-to-implant contact and the development of fibrous connective tissue at the implant interface [6].

Histopathological evaluation can provide valuable information regarding the biological processes underlying implant failure. Features such as inflammatory cell infiltration, fibrous connective tissue formation, foreign-body giant cells, necrotic tissue, and bone resorption may provide evidence of the tissue response surrounding failed implants [7]. Histological assessment of the implant–bone interface can additionally provide insight into the extent of osseointegration and the biological changes associated with its failure [8].

Patient-related characteristics may also influence implant prognosis. Smoking can adversely affect tissue healing and may interfere with the biological processes required for successful implant integration. Periodontal disease and diabetes mellitus may also influence implant outcomes through inflammatory, microbial, and wound-healing mechanisms [9].

Understanding the relationship between the clinical causes of implant failure and their histopathological characteristics may improve the interpretation of failed implants and provide greater insight into the mechanisms responsible for failure. Identification of clinical and histopathological factors associated with biologically mediated implant failure may also help

clinicians recognize patients who require closer monitoring and appropriate preventive strategies. Therefore, the present study was undertaken to evaluate the clinical causes and histopathological characteristics of dental implant failure, examine the relationship between clinical failure patterns and tissue-level findings, and identify clinical and histopathological predictors of biologically mediated implant failure.

MATERIALS AND METHODS

Study Design and Study Population

The present study was conducted at a tertiary care centre and included 100 patients presenting with dental implant failure fom July to December 2025. Demographic and relevant clinical characteristics were recorded for each participant. The study population comprised 61 males (61.0%) and 39 females (39.0%). The largest proportion of participants belonged to the 46–60-year age group (43.0%), followed by those aged >60 years (35.0%) and 30–45 years (22.0%).

Clinical Assessment

Relevant clinical characteristics were documented, including smoking status, diabetes mellitus, history of periodontal disease, and implant location. Smoking was reported in 38.0% of patients, diabetes mellitus in 24.0%, and a previous history of periodontal disease in 47.0%. Of the failed implants, 56.0% were located in the mandible and 44.0% in the maxilla.

Classification of Clinical Causes of Implant Failure

The clinical causes of implant failure were categorized into seven groups:

- Peri-implantitis
- Lack of osseointegration
- Mechanical overload
- Implant fracture
- Prosthetic complications
- Infection/abscess
- Other causes

Peri-implantitis represented the most common clinical category (32.0%), followed by lack of osseointegration (24.0%) and mechanical overload (16.0%).

Histopathological Assessment

Histopathological examination was performed to characterize the tissue changes associated with failed implants. The evaluated findings included chronic inflammatory infiltrate, lymphoplasmacytic infiltration, neutrophilic infiltration, fibrous connective tissue surrounding the implant, foreign-body giant cells, necrotic tissue, bone resorption, and reduced or absent osseointegration.

Chronic inflammatory infiltrate was identified in 62.0% of specimens, while bone resorption was observed in 53.0%. Lymphoplasmacytic infiltration occurred in 48.0%, fibrous connective tissue in 41.0%, neutrophilic infiltration in 35.0%, necrotic tissue in 27.0%, and foreign-body giant cells in 19.0%. Reduced or absent osseointegration was identified in 46.0% of failed implants.

Assessment of Clinical–Histopathological Association

The relationship between the clinical cause of implant failure and selected histopathological findings was evaluated. Particular attention was given to chronic inflammatory infiltrate, bone resorption, fibrous connective tissue formation, and reduced or absent osseointegration.

Peri-implantitis was predominantly characterized by inflammatory changes and bone resorption, whereas cases classified clinically as lack of osseointegration demonstrated greater frequencies of fibrous connective tissue formation and reduced/absent osseointegration. The overall association between clinical cause and histopathological findings was statistically significant ($p = 0.044$).

Correlation Analysis

Spearman's rank correlation coefficient (r) was used to evaluate the relationship between histopathological characteristics and clinical severity of implant failure. Significant positive correlations were observed for all evaluated histopathological parameters. Reduced osseointegration demonstrated the strongest correlation ($r = 0.61$, $p < 0.001$), followed by bone resorption ($r = 0.58$, $p < 0.001$) and chronic inflammatory infiltrate ($r = 0.52$, $p < 0.001$).

Multivariable Logistic Regression

Multivariable binary logistic regression was performed to determine independent predictors of biologically mediated implant failure. Clinical variables included smoking, history of periodontal disease, and diabetes mellitus, while relevant histopathological variables included chronic inflammatory infiltrate, bone resorption, and reduced osseointegration.

Adjusted odds ratios (OR) with corresponding 95% confidence intervals (CI) were calculated. Reduced osseointegration, bone resorption, chronic inflammatory infiltrate, periodontal disease history, and smoking demonstrated statistically significant associations with biologically mediated implant failure. Diabetes mellitus did not demonstrate a statistically significant independent association.

Statistical Analysis

The data were analyzed using appropriate descriptive and inferential statistical methods. Categorical variables, including age group, gender, smoking status, diabetes mellitus, history of periodontal disease, implant location, clinical cause of implant failure, and histopathological findings, were expressed as frequencies and percentages. Histopathological findings were considered as categorical variables and could coexist within the same failed implant specimen.

The association between clinical causes of dental implant failure and histopathological findings was assessed using the Chi-square test of independence. Where expected cell frequencies were small, Fisher's exact test was considered appropriate. The results were expressed as proportions within each clinical failure category.

The relationship between histopathological features and clinical severity of implant failure was evaluated using Spearman's rank correlation coefficient (r), as the histopathological parameters and clinical severity were treated as ordinal/non-normally distributed variables. The correlation coefficient was used to determine both the direction and strength of the association.

A multivariable binary logistic regression analysis was performed to identify independent predictors of biologically mediated implant failure. Clinical variables including smoking, history of periodontal disease and diabetes mellitus, along with relevant histopathological characteristics such as chronic inflammatory infiltrate, bone resorption and reduced osseointegration, were entered into the regression model. The results were presented as adjusted OR with 95% CI.

For all inferential analyses, a p-value <0.05 was considered statistically significant. A p-value ≥ 0.05 was considered statistically non-significant. The statistical analysis was designed to determine the relationship between the clinical causes of implant failure and corresponding histopathological changes, and to identify histopathological and clinical factors independently associated with biologically mediated implant failure.

RESULTS

Table 1 & Figure 1 present the demographic and clinical characteristics of the 100 patients with dental implant failure. The largest proportion of patients belonged to the 46–60-year age group (43.0%), followed by those aged >60 years (35.0%) and 30–45 years (22.0%). Males constituted the majority of the study population (61.0%), while females accounted for 39.0%.

Regarding clinical risk factors, 38.0% of patients had a history of smoking, and 24.0% had diabetes mellitus. A previous history of periodontal disease was reported in 47.0% of patients. In terms of implant location, failed implants were more frequently observed in the mandible (56.0%) than in the maxilla (44.0%). Overall, the study population was predominantly middle-aged and male, with a substantial proportion of patients having a history of periodontal disease and smoking.

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Table 1: Demographic and Clinical Characteristics of Patients with Dental Implant Failure

	Characteristic	n (%)
Age	Age 30–45 years	22 (22.0)
	Age 46–60 years	43 (43.0)
	Age >60 years	35 (35.0)
Gender	Male	61 (61.0)
	Female	39 (39.0)
Clinical Characteristics	Smoking present	38 (38.0)
	Diabetes mellitus	24 (24.0)
	History of periodontal disease	47 (47.0)
	Mandibular implant	56 (56.0)
	Maxillary implant	44 (44.0)

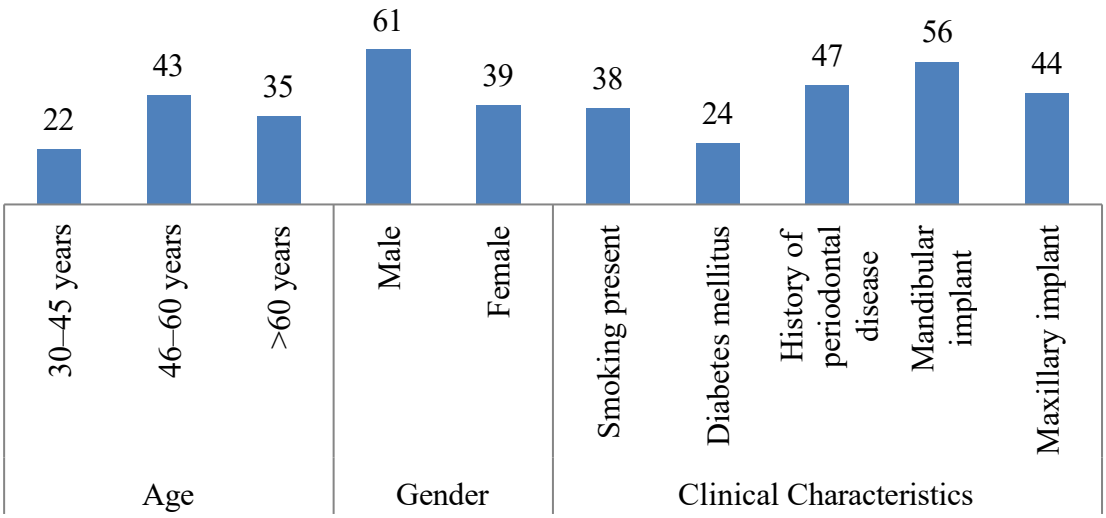


Figure 1: Demographic characteristics and distribution of clinical risk factors and implant location among patients with dental implant failure

Table 2 & Figure 2 illustrate the distribution of clinical causes of dental implant failure among the 100 patients included in the study. Peri-implantitis was the most frequently identified cause, accounting for 32.0% of implant failures, followed by inadequate osseointegration (24.0%) and mechanical overload (16.0%). Implant fracture was responsible for 9.0% of failures, whereas prosthetic complications accounted for 8.0%. Infection or abscess was observed in 6.0% of cases, while other less common causes comprised the remaining 5.0%. Overall, biological factors, particularly peri-implantitis and inadequate osseointegration, constituted the predominant causes of dental implant failure in the study population.

Table 2: Distribution of Clinical Causes of Dental Implant Failure

Clinical cause	n (%)
Peri-implantitis	32 (32.0)
Lack of osseointegration	24 (24.0)
Mechanical overload	16 (16.0)
Implant fracture	9 (9.0)

Prosthetic complications	8 (8.0)
Infection/abscess	6 (6.0)
Other causes	5 (5.0)
Total	100 (100.0)

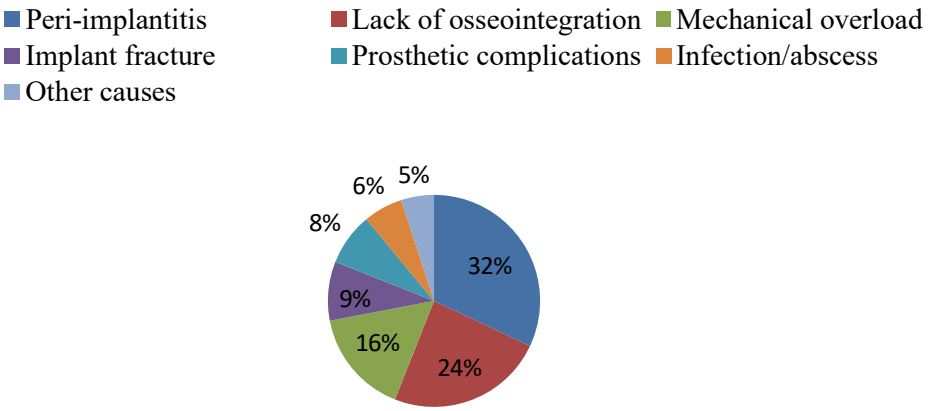


Figure 2: Distribution of the clinical causes of dental implant failure among the study population

Table 3 & Figure 3 summarize the histopathological characteristics observed in specimens obtained from failed dental implants. Chronic inflammatory cell infiltration was the most common finding, occurring in 62.0% of cases, followed by bone resorption (53.0%). Lymphoplasmacytic infiltration was identified in 48.0% of specimens, while fibrous connective tissue formation around the implant was observed in 41.0%. Neutrophilic infiltration was present in 35.0% of cases, whereas necrotic tissue was detected in 27.0%. Foreign-body giant cells were observed in 19.0% of specimens. Evidence of reduced or absent osseointegration was noted in 46.0% of failed implants. Overall, the histopathological profile was predominantly characterized by chronic inflammatory changes and peri-implant bone resorption, with lymphoplasmacytic inflammation and compromised osseointegration also being frequently observed.

Table 3: Histopathological Findings Associated with Implant Failure

Histopathological finding	n (%)
Chronic inflammatory infiltrate	62 (62.0)
Fibrous connective tissue surrounding implant	41 (41.0)
Neutrophilic infiltration	35 (35.0)
Lymphoplasmacytic infiltration	48 (48.0)
Foreign-body giant cells	19 (19.0)
Necrotic tissue	27 (27.0)
Bone resorption	53 (53.0)
Reduced/absent osseointegration	46 (46.0)

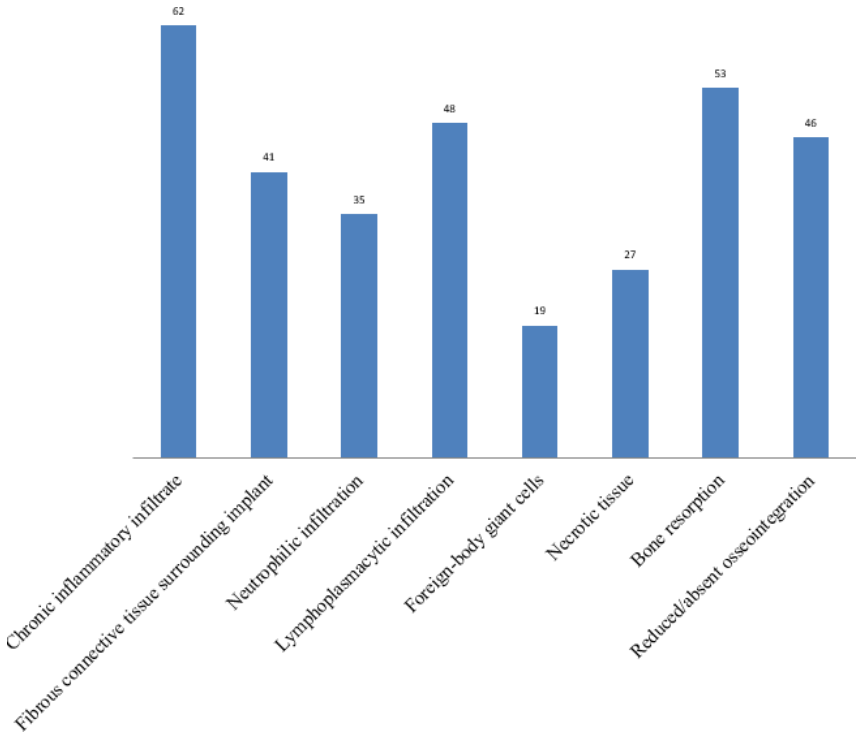


Figure 3: Histopathological distribution of inflammatory, tissue, and osseointegration-related findings in failed dental implants

Table 4 & Figure 4 demonstrate a statistically significant association between the clinical etiology of dental implant failure and the corresponding histopathological features ($p = 0.044$). Chronic inflammatory infiltrates were most prevalent among implants diagnosed clinically with peri-implantitis (87.5%), followed by cases attributed to mechanical overload (62.5%) and failure of osseointegration (54.2%). Similarly, bone resorption was particularly prominent in peri-implantitis cases (84.4%); compared with 56.3% among mechanically overloaded implants and 44.4% in cases of implant fracture.

The presence of fibrous connective tissue surrounding the implant was highest in cases of failed osseointegration (70.8%), followed by peri-implantitis (40.6%) and mechanical overload (31.3%). Reduced or absent osseointegration showed a similar pattern, occurring most frequently in implants clinically classified as having failed osseointegration (75.0%), followed by mechanical overload (56.3%) and peri-implantitis (37.5%).

Overall, the histopathological pattern varied according to the clinical cause of implant failure. Peri-implantitis was predominantly associated with inflammatory infiltration and bone resorption, whereas failure of osseointegration was characterized mainly by fibrous tissue formation and diminished or absent bone-to-implant integration.

Table 4: Association Between Clinical Cause of Implant Failure and Histopathological Findings

Clinical cause of implant failure	Chronic inflammatory infiltrate, n (%)	Bone resorption, n (%)	Fibrous connective tissue, n (%)	Reduced/absent osseointegration, n (%)
Peri-implantitis (n=32)	28 (87.5)	27 (84.4)	13 (40.6)	12 (37.5)
Lack of osseointegration (n=24)	13 (54.2)	9 (37.5)	17 (70.8)	18 (75.0)
Mechanical overload (n=16)	10 (62.5)	9 (56.3)	5 (31.3)	9 (56.3)

Implant fracture (n=9)	4 (44.4)	4 (44.4)	2 (22.2)	3 (33.3)
Prosthetic complications (n=8)	4 (50.0)	2 (25.0)	2 (25.0)	2 (25.0)
Infection/abscess (n=6)	2 (33.3)	0 (0.0)	1 (16.7)	1 (16.7)
Other causes (n=5)	1 (20.0)	2 (40.0)	1 (20.0)	1 (20.0)
Total	62 (62.0)	53 (53.0)	41 (41.0)	46 (46.0)
p-value	0.044 (Significant)			

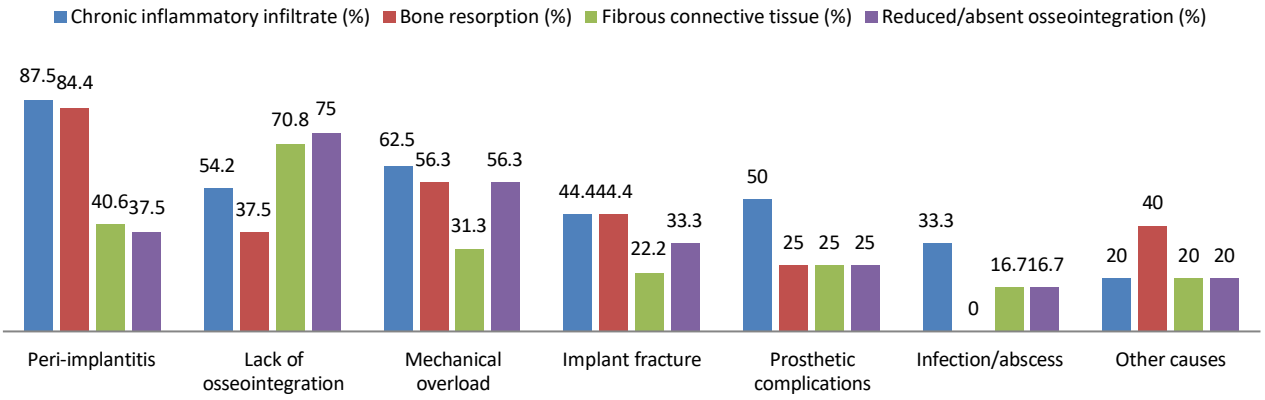


Figure 4: Association between the clinical causes of dental implant failure and their corresponding histopathological findings

Table 5 presents the Spearman's correlation analysis, which revealed statistically significant positive associations between all assessed histopathological features and the clinical severity of dental implant failure. The strongest correlation was observed for reduced osseointegration ($r = 0.61$, $p < 0.001$), followed by bone resorption ($r = 0.58$, $p < 0.001$) and chronic inflammatory infiltrate ($r = 0.52$, $p < 0.001$). Neutrophilic infiltration demonstrated a moderate positive correlation with clinical severity ($r = 0.46$, $p < 0.001$), while fibrous tissue formation showed a significant positive correlation of moderate strength ($r = 0.41$, $p < 0.001$). Foreign-body giant cell formation exhibited the weakest association, although the correlation remained statistically significant ($r = 0.29$, $p = 0.003$). Overall, the findings indicate that increasing clinical severity of implant failure is associated with greater histopathological deterioration, particularly loss of osseointegration, bone resorption, and chronic inflammatory changes.

Table 5: Correlation Between Histopathological Features and Clinical Severity of Implant Failure

Histopathological parameter	Correlation coefficient (r)	p-value
Chronic inflammatory infiltrate	0.52	<0.001
Bone resorption	0.58	<0.001
Fibrous tissue formation	0.41	<0.001
Neutrophilic infiltration	0.46	<0.001
Foreign-body giant cells	0.29	0.003
Reduced osseointegration	0.61	<0.001

Table 6 presents the results of the multivariable logistic regression analysis performed to identify independent predictors of biologically mediated dental implant failure. Among the histopathological parameters, reduced osseointegration demonstrated the strongest independent association with implant failure. Patients exhibiting reduced osseointegration had 4.76 times greater odds of biologically mediated implant failure compared with those without this finding (adjusted OR = 4.76, 95% CI: 2.14–10.58; $p < 0.001$). Bone resorption was another significant predictor, being associated with a 4.12-fold increase in the odds of implant failure (95% CI: 1.92–8.84; $p < 0.001$). The presence of a chronic inflammatory infiltrate was also independently associated with implant failure, with affected cases showing 3.26 times higher odds (95% CI: 1.51–7.03; $p = 0.003$).

With respect to clinical risk factors, a history of periodontal disease was significantly associated with biologically mediated implant failure. Individuals with previous periodontal disease had 2.74-fold greater odds of implant failure (adjusted OR = 2.74, 95% CI: 1.36–5.52; $p = 0.005$). Smoking was similarly identified as an independent predictor, with smokers demonstrating 2.18 times higher odds of implant failure than nonsmokers (95% CI: 1.08–4.39; $p = 0.030$). In contrast, diabetes mellitus, although associated with an elevated estimated risk (adjusted OR = 1.91), did not show a statistically significant independent association with implant failure (95% CI: 0.84–4.34; $p = 0.121$).

Overall, the analysis demonstrated that reduced osseointegration, bone resorption, chronic inflammatory infiltrate, a history of periodontal disease, and smoking were significant independent predictors of biologically mediated dental implant failure. Among these variables, reduced osseointegration showed the greatest effect size. Diabetes mellitus did not emerge as a statistically significant independent predictor after adjustment for the other variables included in the regression model.

Table 6: Multivariable Logistic Regression for Predictors of Biologically Mediated Implant Failure

Predictor	Adjusted OR	95% CI	p-value
Smoking	2.18	1.08–4.39	0.030
History of periodontal disease	2.74	1.36–5.52	0.005
Diabetes mellitus	1.91	0.84–4.34	0.121
Chronic inflammatory infiltrate	3.26	1.51–7.03	0.003
Bone resorption	4.12	1.92–8.84	<0.001
Reduced osseointegration	4.76	2.14–10.58	<0.001

DISCUSSION

The present study evaluated the clinical causes and histopathological characteristics of dental implant failure and examined the influence of patient-related and tissue-level factors on biologically mediated failure. The findings demonstrated that implant failure was multifactorial, with peri-implantitis (32.0%), lack of osseointegration (24.0%), and mechanical overload (16.0%) representing the most frequent clinical causes. Histopathological examination further demonstrated a high prevalence of chronic inflammatory infiltrate, bone resorption, and reduced or absent osseointegration among failed implants. These findings emphasize the importance of both biological and mechanical factors in determining implant prognosis.

The clinical pattern observed in the present study is consistent with the multifactorial nature of implant failure described by Wang et al. (1996) [10], who emphasized that implant failure may result from a combination of local and systemic risk factors rather than a single causative factor. In the present study, peri-implantitis was the most frequent identified cause of failure, highlighting the importance of inflammatory peri-implant disease in compromising implant stability. The occurrence of inadequate osseointegration as the second most common cause further indicates that successful biological integration remains a fundamental determinant of implant survival.

The histopathological findings provide additional insight into the biological mechanisms underlying implant failure. Chronic inflammatory infiltrate was identified in 62.0% of specimens, while bone resorption and lymphoplasmacytic infiltration were present in 53.0% and 48.0%, respectively. These findings indicate that persistent inflammatory activity and destruction of supporting bone are prominent tissue-level features in failed implants. The significant association observed between clinical

failure categories and histopathological characteristics ($p = 0.044$) further supports the relevance of tissue-level assessment in understanding the clinical presentation of implant failure.

An important finding of the present study was the strong relationship between reduced osseointegration and clinical severity. Reduced or absent osseointegration demonstrated the strongest positive correlation with clinical severity ($r = 0.61$, $p < 0.001$), followed by bone resorption ($r = 0.58$, $p < 0.001$) and chronic inflammatory infiltrate ($r = 0.52$, $p < 0.001$). These observations suggest that progressive biological compromise of the implant–bone interface is closely associated with more severe clinical failure. The findings are consistent with the concept described by Chrcanovic et al. (2014) [11], in which impaired biological conditions can adversely influence implant integration and ultimately contribute to implant failure.

Patient-related risk factors also appeared to influence implant outcomes. In the present study, 38.0% of patients were smokers, 24.0% had diabetes mellitus, and 47.0% had a history of periodontal disease. Multivariable analysis demonstrated that smoking was independently associated with biologically mediated implant failure (adjusted OR = 2.18, $p = 0.030$). This observation is consistent with the findings of Anner et al. (2010) [12], who evaluated smoking, diabetes, periodontitis, and supportive periodontal treatment as factors associated with long-term implant survival. Their findings support the importance of considering patient-related and periodontal characteristics when evaluating long-term implant prognosis.

The relationship between peri-implant disease and implant failure has also been demonstrated in clinical research. Daubert et al. (2015) [13] investigated the prevalence and predictive factors for peri-implant disease and implant failure and demonstrated that peri-implant complications may be influenced by several patient- and implant-related characteristics. The relatively high frequency of peri-implantitis observed in the present study similarly highlights the importance of inflammatory peri-implant disease as a major clinical pathway leading to implant failure. The present histopathological findings, particularly the high prevalence of inflammatory infiltrates and bone resorption, provide further tissue-level support for this association.

Smoking may adversely affect the biological environment surrounding dental implants through effects on vascularity, tissue healing, immune response, and bone metabolism. Chen et al. (2013) [14], in a meta-analysis examining smoking, radiotherapy, diabetes, and osteoporosis as potential risk factors, reported that these systemic and behavioral characteristics may influence the risk of implant failure. In the present study, smoking remained a significant independent predictor after adjustment for selected clinical and histopathological factors. This finding reinforces the importance of smoking assessment and cessation counseling as part of comprehensive implant therapy.

Diabetes mellitus represents another potential systemic factor because altered glucose metabolism may interfere with wound healing and bone remodeling. Chrcanovic et al. (2014) [15] systematically evaluated the relationship between diabetes and oral implant failure and highlighted the potential adverse influence of diabetes on implant outcomes. In the present study, however, diabetes mellitus did not demonstrate a statistically significant independent association with biologically mediated implant failure after multivariable adjustment. This difference may reflect variations in sample characteristics, diabetes severity, metabolic control, treatment protocols, follow-up duration, or the influence of other confounding factors. Therefore, the absence of statistical significance in the present analysis should not be interpreted as evidence that diabetes has no effect on implant prognosis.

A history of periodontal disease was another clinically important finding in the present study. Patients with previous periodontal disease had significantly greater odds of biologically mediated implant failure (adjusted OR = 2.74, $p = 0.005$). This finding is clinically plausible because periodontal disease may reflect a persistent susceptibility to inflammatory and microbial challenges within the oral environment. Furthermore, inadequate periodontal control may contribute to an unfavorable peri-implant environment. The findings of Anner et al. (2010) [12] and Daubert et al. (2015) [13] support the importance of periodontal and peri-implant conditions when evaluating implant survival.

The findings also demonstrate the value of combining clinical and histopathological assessment. Clinical diagnosis can identify the apparent cause of implant failure, but histopathological examination may reveal the underlying tissue response and provide additional evidence regarding the biological pathway involved. In particular, the strong correlations observed for reduced osseointegration, bone resorption, and chronic inflammation indicates that these findings may be useful markers of biologically mediated implant failure.

From a clinical perspective, the results emphasize the importance of comprehensive risk assessment before implant placement and continued maintenance after treatment. Smoking status and periodontal history should be carefully evaluated, and patients with relevant risk factors may benefit from enhanced periodontal management and closer peri-implant monitoring. In cases of suspected implant failure, histopathological assessment may provide useful complementary information regarding inflammatory activity, bone destruction, and the status of osseointegration.

Overall, the present findings support the concept that dental implant failure results from an interaction of local biological changes, impaired osseointegration, patient-related risk factors, and inflammatory conditions. Peri-implantitis was the leading clinical cause, while reduced osseointegration, bone resorption, and chronic inflammatory infiltrate showed the strongest relationships with clinical severity. Smoking and a history of periodontal disease were independently associated with biologically mediated failure, whereas diabetes did not demonstrate an independent association in the present analysis. These findings reinforce the importance of individualized risk assessment, periodontal control, smoking cessation, and appropriate biological evaluation in improving the long-term prognosis of dental implants.

LIMITATIONS

The findings should be interpreted in light of several limitations. First, the study included a relatively modest sample of 100 patients, which may restrict the generalizability of the results to larger and more diverse implant populations. Second, the observational nature of the analysis limits the ability to establish causal relationships between the identified risk factors and implant failure. Although multivariable analysis was performed, residual confounding from other patient-related, surgical, implant-related, and maintenance factors cannot be excluded.

The study also focused on selected clinical and histopathological variables; therefore, other potentially relevant factors, such as implant surface characteristics, implant dimensions, loading protocol, oral hygiene practices, maintenance frequency, bone quality, and duration of implant function, were not comprehensively represented in the available dataset. Furthermore, the histopathological findings were assessed in failed implants and therefore cannot establish whether the observed tissue changes preceded failure or developed as a consequence of the failure process. Larger prospective studies incorporating detailed clinical, radiographic, microbiological, and histomorphometric assessments are required to validate these findings.

CONCLUSION

Dental implant failure demonstrated a multifactorial pattern, with peri-implantitis and lack of osseointegration being the leading clinical causes. Histopathologically, chronic inflammation, bone resorption, and reduced osseointegration were strongly associated with greater clinical severity. Reduced osseointegration was the strongest independent predictor of biologically mediated failure, while a history of periodontal disease and smoking were also significant predictors. These findings emphasize the importance of adequate osseointegration, periodontal health, inflammation control, and risk-factor management in improving the long-term prognosis of dental implants.

Conflicts of interest: Nil

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