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Long-Term Outcomes of Immediate Implants in Endodontic, Periodontal, Endo–Perio Compromised Extraction Sockets and Failed Implant Sites: A Retrospective Single-Center Cohort Study

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Abstract

Objective

To assess long-term outcomes of immediate implant placement in endodontic, periodontal and endo–perio compromised sockets and in sites immediately replaced after failed implant explantation, and to examine the roles of gap width, grafting and indication.

Materials and Methods

In this single-center retrospective cohort, 363 immediate implants were followed for a median of 60 months (36–87). Groups were: I, non-infectious traumatic/prosthetic; II, endodontic; III, periodontal; IV, endo–perio; V, failed implant replacement. Gap width, grafting, osseointegration and peri-implantitis were recorded. Cox models (adjusted for age, sex, gap width, grafting) and ROC analysis evaluated early failure and peri-implantitis.

Results

Group II patients were younger ($p < 0.001$), while sex distribution and follow-up were similar ($p > 0.05$). Early osseointegration failure risk was higher than controls in Group IV (HR = 5.812; $p = 0.019$) and Group V (HR = 6.182; $p = 0.015$). Among osseointegrated implants, peri-implantitis risk increased in Group IV (HR = 3.110; $p = 0.048$) and was borderline in Group V (HR = 3.002; $p = 0.053$). Peri-implantitis severity was greater in Groups III/IV than I/II ($p < 0.001$). Despite the smallest median gap, Group V showed the highest early failure. Gap width did not predict early failure (AUC = 0.527; $p = 0.640$). Grafted 2–4 mm gaps were associated with lower peri-implantitis risk in univariable analysis (HR = 0.245; $p = 0.022$) and showed a non-significant protective trend after adjustment (HR = 0.171; $p = 0.065$).

Conclusions

Endo–perio sockets and immediate replacement of failed implants represent the highest-risk scenarios for immediate implants. Horizontal gap width alone should not guide decisions; site biology related to the indication appears more influential. The potential benefit of grafting 2–4 mm gaps requires prospective validation.

INTRODUCTION

Immediate implant placement (IIP) is widely accepted because it shortens overall treatment time, reduces the number of surgical interventions, and may preserve alveolar architecture [1, 2]. However, a significant limitation in clinical practice is that extraction indications often involve pathologies associated with active or chronic infection, such as failed endodontic treatments, advanced periodontal breakdown, or combined

endo–perio lesions [3]. Evidence indicates that in these infected sites, bacterial biofilms and resistant microorganisms can persist in the bone after tooth extraction [4]. These microbial residues are biological risk factors for retrograde peri-implantitis and early loss of osseointegration, thereby diminishing the predictability of IIP [5].

The biological predictability of IIP is significantly affected by the type of infection, its anatomical extent, and the pattern of hard- and soft-tissue destruction [6]. While favorable outcomes have been reported in carefully debrided sockets with isolated periapical lesions, sites with endo–perio lesions involving both pulpal and periodontal infection, as well as those where a failed implant has been explanted, constitute more vulnerable biological environments [7, 8]. At these compromised sites, chronic inflammation, altered local vascularity, and changes in bone metabolism and healing capacity, induced by infection or prior failure, contribute to a cumulative biological burden, referred to in the literature as a “negative biological legacy” [9, 10]. The precise effect of this negative biological legacy on osseointegration remains uncertain. Therefore, systematic evaluation of both early and long-term biological outcomes of immediate protocols in sockets with complex pathological backgrounds is necessary.

In addition to biological factors, technical parameters such as primary stability, three-dimensional implant positioning, horizontal gap width, and the use of bone grafts are critical for preserving marginal bone and maintaining soft-tissue stability around implants [8, 11]. However, current evidence is inconsistent regarding whether gap width alone is a reliable prognostic indicator and how grafting effects may differ across various biological backgrounds [12]. Most existing studies focus on a single pathological category, and comprehensive cohort data comparing IIP in sockets with distinct infection profiles under standardized surgical and prosthetic protocols are limited [9–12]. Studies evaluating both infected pathological sockets and noninfected extraction sockets within the same cohort, using standardized surgical protocols and time-dependent risk analyses, are rare. A review of the literature did not identify any study that directly and systematically compared different biological socket types in terms of the risks of osseointegration failure and peri-implantitis within a unified analytical framework.

Given this context, infection-related factors and site-specific biology must be clearly distinguished from purely technical parameters when assessing the risk profile of immediate implants. The presence of infection, its anatomical pattern of spread, and tissue alterations from prior treatment failure are key biological determinants that influence both early osseointegration and long-term peri-implant tissue health. In contrast, the prognostic value of gap width and grafting strategies in these high-risk clinical scenarios remains uncertain.

The primary aim of this study was to compare osseointegration success and the long-term prevalence of peri-implantitis between immediate implants placed into infected sockets—following failed endodontic treatment, periodontal disease, combined endo–perio lesions, or implant explantation—and those placed into extraction sockets without clinical or radiographic evidence of infection or chronic inflammation. A secondary aim was to assess the influence of peri-implant gap width and bone grafting on clinical outcomes across these heterogeneous biological backgrounds. The null hypothesis stated that the pathological history of the extraction or explantation site would not have a statistically significant effect

on osseointegration success or peri-implantitis development after adjustment for demographic and technical variables.

MATERIALS AND METHODS

1. Study design and ethical approval

This retrospective, single-center cohort study evaluated clinical and radiographic records of single-tooth IIP procedures performed between 2017 and 2025 at the Department of Periodontology, Faculty of Dentistry, Usak University (Usak, Türkiye). All consecutive cases that met predefined eligibility criteria were included to minimize selection bias. Only one implant per patient was analyzed to ensure that implant-level outcomes accurately reflected patient-level risk patterns.

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Usak University Clinical Research Ethics Committee (Approval No: 670-670-18). Reporting followed the STROBE guidelines for observational cohort studies.

2. Patient selection and eligibility criteria

Inclusion criteria

* Age ≥ 18 years

* Systemically healthy status

* Non-smoking and non-diabetic

* Single missing mandibular molar tooth requiring implant placement

* Immediate implant placement in a fresh extraction or explantation socket

* Minimum continuous clinical and radiographic follow-up of 36 months after implant placement

* No use of systemic medications known to affect periodontal tissues or bone metabolism

Exclusion criteria

* Follow-up period shorter than 36 months

* Incomplete or poor-quality clinical and/or radiographic records

* Uncontrolled systemic disease, immunosuppressive therapy, or a history of bone metabolic disease

A total of 363 implants, each placed in a different patient, met the inclusion criteria and were incorporated into the final analysis.

3. Group allocation

Implant sites were stratified into five groups according to the pre-existing biological condition at the time of tooth extraction or implant explantation:

Group I – Control (n = 48)

Fresh, non-infected extraction sockets of teeth removed because of traumatic crown/root fractures or non-restorable structural damage, with no clinical or radiographic evidence of pulpal or periodontal infection.

Group II – Endodontic (n = 132)

Extraction sockets of teeth removed because of failed root canal therapy or persistent periapical pathology.

Group III – Periodontal (n = 61)

Sockets of teeth extracted due to advanced periodontal destruction, chronic periodontal infection, and tooth mobility following unsuccessful periodontal therapy.

Group IV – Endo–perio (n = 61): Sockets with combined endodontic–periodontal lesions meeting all of the following criteria [13]: Endodontic component: periapical radiolucency ≥ 2 mm in diameter, history of inadequate or leaking root canal treatment, and clinical signs of pulpal necrosis or a non-vital pulp. Periodontal component: probing depth ≥ 6 mm, clinical attachment loss ≥ 5 mm, and bleeding and/or suppuration on probing at the affected site. All criteria had to be present for classification as Group IV.

Group V – Failed implant replacement (n = 61)

Sites in which a previously placed implant was explanted due to peri-implantitis that did not respond to non-surgical peri-implant therapy and showed vertical bone loss affecting at least 50% of the implant length on preoperative periapical radiographs, with the buccal and lingual cortical plates remaining intact [14].

In Group V, explantation was standardized using an atraumatic reverse-torque technique. For implants originally placed at the study institution, the manufacturer's specific removal ratchet was used. For externally placed implants where the original system-specific driver was unavailable, a dedicated fixture-removal kit designed to minimize additional bone loss was employed (Surgident Fixture Remover Kit, Daegu, Republic of Korea).

4. Definitions of pre-existing pathologies and outcomes

4.1. Failed endodontic therapy

Failed endodontic therapy was defined as the presence of at least one of the following clinical and radiographic features: persistent pain, tenderness to percussion or palpation, sinus tract formation, and/or radiographic evidence of a poorly condensed, underfilled (> 2 mm short) or overextended root canal filling associated with a persistent or enlarging periapical radiolucency ≥ 2 mm [15, 16].

4.2. Failed periodontal therapy

Failed periodontal therapy was defined as persistence of active periodontal disease despite treatment, characterized by one or more of the following [17, 18]:

Tooth mobility grade II–III

Periodontal probing depth ≥ 6 mm with bleeding and/or suppuration on probing

Vertical bone loss affecting ≥ 50% of the root length

Grade II–III furcation involvement

4.3. Failed endo–perio lesion therapy

A failed endo–perio lesion was diagnosed when periapical pathology of endodontic origin was contiguous with a deep periodontal pocket (probing depth ≥ 6 mm) along the same root surface, with vertical bone loss extending from the apex toward the crestal bone and absence of pulp vitality, indicating a combined endodontic and periodontal infection [7, 19].

4.4. Failed implant therapy

Failed implant therapy was defined as peri-implantitis that did not respond to non-surgical peri-implant treatment and presented with bleeding and/or suppuration on probing, together with vertical bone loss affecting > 50% of the implant length on radiographic examination [20, 21].

4.5. Early osseointegration failure

Early osseointegration failure woading, at least one of the following criteria was met [22, 23]:

1. Clinically detectable mobility of the implant;

2. Persistent pain and/or suppuration that failed to resolve after appropriate mechanical debridement and antibiotic therapy;

3. A radiolucent zone > 1 mm surrounding the implant body;

4. Clinical decision to remove the implant based on these findings.

4.6. Peri-implantitis

Peri-implantitis was defined as a site-specific inflammatory condition surrounding an osseointegrated implant, characterized by [24, 25]:

Probing depth ≥ 6 mm at ≥ 1 aspect of the implant;

Bleeding and/or suppuration on probing; and

Radiographic marginal bone loss ≥ 2 mm compared with the post-loading baseline radiograph.

Peri-implantitis status and marginal bone loss were evaluated at the most recent follow-up visit for each patient.

5. Surgical protocol, decontamination, and gap management

5.1. Surgical approach and implant system

All surgical procedures were performed by a single experienced periodontist (F.K.) with more than 10 years of experience in implant surgery. All implants were from the same commercially available system, with a sand-blasted, acid-etched (SLA) surface and a bone-level design (T6 Standard Bone Level Implant, Nucleoss, Izmir, Türkiye). Teeth were extracted using a flapless, atraumatic technique to preserve the surrounding alveolar bone and soft-tissue architecture.

5.2. Decontamination of infected sites

In endodontic, periodontal, endo–perio, and failed-implant sockets, a standardized decontamination protocol was implemented before implant placement:

Mechanical debridement: All granulation tissue was carefully removed from the socket walls with manual Gracey curettes. At explantation sites, the previous implant bed was additionally debrided with titanium-safe curettes.

Chemical decontamination: Following mechanical debridement, all sites were irrigated with 0.12% chlorhexidine gluconate for 60 seconds and then thoroughly rinsed with sterile saline.

The socket was considered suitable for immediate implant placement only if the following macroscopic criteria were satisfied:

Absence of visible pus;

Presence of fresh bleeding from the bone walls;

No remaining necrotic or granulation tissue.

5.3. Implant placement and primary stability

Implants were placed using a standardized drilling protocol, typically engaging the lingual cortical plate as a reference to achieve optimal three-dimensional positioning. Primary stability was assessed intraoperatively using a calibrated surgical motor and recorded as insertion torque; a minimum insertion

torque of ≥ 35 Ncm was required. Healing abutments were placed in all cases to support peri-implant soft tissues and maintain socket morphology during healing.

Implant dimensions were distributed as follows:

4.1 × 8 mm: 56 implants

4.1 × 10 mm: 128 implants

4.7 × 8 mm: 59 implants

4.7 × 10 mm: 107 implants

5.4. L-PRF protocol

To support soft-tissue healing, all patients received leukocyte- and platelet-rich fibrin (L-PRF) using a standard protocol. Ten milliliters of venous blood were collected and centrifuged at 2,700 rpm for 12 minutes to obtain an L-PRF membrane. The membrane was placed over the socket entrance and around the healing abutment. L-PRF was used solely as a biological adjunct to modulate soft-tissue healing and not as a filler for the bone gap [26]. The same L-PRF protocol was applied uniformly across all study groups.

5.5. Horizontal gap measurement and classification

Immediately after implant placement, the horizontal gap between the implant surface and the inner aspect of the buccal alveolar wall was measured intraoperatively using a sterile Williams periodontal probe. The largest distance was recorded.

Gap width was documented both as a continuous variable (mm) and as a categorical variable using the following predefined classes [11, 27, 28]:

≤ 2 mm

2 to 4 mm

≥ 5 mm

5.6. Graft application

For sites with a horizontal gap ≥ 2 mm, the need for bone grafting was assessed on a case-by-case basis. The surgeon decided whether to graft based on [11, 12, 29]:

Buccal cortical wall thickness and defect morphology;

Level of primary stability;

Patient preference and consent regarding the use of allograft material.

When grafting was performed, a mineralized corticocancellous allograft (Maxxeus Allograft, Community Tissue Services, Dayton, OH, USA) was used.

5.7. Loading protocol

All implants were restored using a conventional loading protocol. Prosthetic loading was initiated no earlier than 3 months after placement, provided that the implant exhibited no clinical mobility, pain, or inflammation and no radiographic radiolucency. All definitive restorations were single cement-retained crowns.

6. Post-operative care

A standardized postoperative regimen was implemented for all patients to ensure consistent infection control and promote healing.

6.1. Systemic pharmacological regimen

All patients received systemic antibiotic therapy as follows:

Amoxicillin/clavulanic acid 875/125 mg twice daily for 5 days; or

In penicillin-allergic patients, clindamycin 300 mg three times daily for 5 days.

Analgesic medication (ibuprofen 400 mg every 6–8 hours as needed) was prescribed for pain control.

6.2. Antiseptic oral care

On the first postoperative day, patients were instructed to rinse twice daily for 7 days with 0.12% chlorhexidine gluconate mouthwash to support chemical plaque control.

6.3. Surgical site management

Non-resorbable sutures were placed to stabilize soft tissues, and the L-PRF membrane was removed between 7 and 10 days after surgery, with uneventful healing observed. Suture placement and L-PRF application were standardized across all patients to support early soft-tissue maturation, particularly in previously infected sockets.

6.4. Oral hygiene instruction and transition to mechanical cleaning

Detailed oral hygiene instructions were provided to all patients. During the first 7 days, the surgical area was protected from mechanical trauma, and only chemical plaque control was recommended. After suture removal, gentle brushing with a soft-bristled toothbrush was initiated in the surgical area and gradually reintegrated into the patient’s routine oral hygiene regimen.

7. Clinical and radiographic follow-up protocol

Following prosthetic loading, all patients were enrolled in a structured maintenance program. Follow-up visits were scheduled at 6, 12, 18, 24, 30, and 36 months, and annually thereafter. Clinical records demonstrated that the majority of patients attended at least 75% of the scheduled visits.

Peri-implantitis status and marginal bone loss outcomes were assessed at the final available follow-up visit for each implant.

7.1. Radiographic assessment and calibration

Marginal bone level (MBL) was evaluated using standardized digital periapical radiographs obtained with the parallel technique. All radiographic measurements were performed using ImageJ software (National Institutes of Health, Bethesda, MD, USA). For each radiograph, the image was calibrated by referencing the known diameter of the implant platform, enabling conversion from pixels to millimeters. This calibration was repeated for each radiograph to minimize errors due to magnification or distortion.

7.2. Baseline reference and measurement protocol

The reference point for MBL measurements was the most coronal point of the implant platform (implant–abutment junction). The vertical distance from this reference point to the first bone–implant contact was measured at the mesial and distal aspects. The periapical radiograph taken on the day of prosthetic loading served as the baseline image.

7.3. Calculation of marginal bone loss

For each implant, marginal bone loss was computed as:

MBL change = (MBL at last follow-up) − (MBL at baseline)

The mean of the mesial and distal measurements was used as the implant-level marginal bone loss value.

7.4. Intra-observer reliability

To assess measurement reliability, a random sample of 30 patients (all serial radiographs) was re-measured by the same examiner (F.K.) after a two-week interval. Agreement between the two measurement sets was evaluated using a two-way mixed-effects, absolute-agreement intraclass correlation coefficient (ICC). The ICC was 0.87 (95% confidence interval: 0.79–0.92), indicating excellent intra-observer reliability.

8. Statistical analysis

All statistical analyses were conducted using IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, NY, USA) and R software, version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria).

The distribution of continuous variables was assessed with the Kolmogorov–Smirnov and Shapiro–Wilk tests.

For non-normally distributed continuous or ordinal variables across three or more groups, the Kruskal–Wallis H test was applied, with Dunn's post hoc test for pairwise comparisons where appropriate. For normally distributed data, one-way analysis of variance (ANOVA) with Bonferroni post hoc correction was used.

The predictive value of horizontal gap width (mm) for distinguishing implants with and without early osseointegration failure was assessed using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) and corresponding p-values were reported. Because no clinically meaningful cut-off was identified, no further sensitivity or specificity indices were calculated.

Time-dependent associations between potential predictors and (i) early osseointegration failure and (ii) peri-implantitis development were assessed using univariate and multivariate Cox proportional hazards regression models. In multivariate models, hazard ratios (HRs) were adjusted for age, sex, group allocation, horizontal gap width, grafting status, and gap × graft categories. Variables such as detailed smoking exposure, glycaemic control, history of periodontitis at non-index sites, home oral-hygiene compliance, and maintenance attendance were not systematically recorded in a standardized format and therefore could not be included as covariates in the multivariate analyses; this limitation is further discussed in the Limitations section.

Associations between categorical variables were assessed using Fisher's exact test, with Monte Carlo simulation to account for low expected cell counts in some categories. When overall significance was observed, post hoc pairwise comparisons were conducted using z-tests with Bonferroni adjustment.

Quantitative data are presented as mean ± standard deviation and/or median (minimum–maximum), and categorical data are reported as frequencies and percentages. Statistical significance was set at $p < 0.05$ for all analyses.

RESULTS

1. Demographic characteristics and follow-up duration

There was a statistically significant difference in median age among the groups (Kruskal-Wallis H = 150.921, $p < 0.001$). Sex distribution did not differ significantly between groups (Fisher's exact test with Monte Carlo simulation, $p = 0.196$), and follow-up duration was also similar (Kruskal-Wallis H = 3.577, $p = 0.466$) (Table 1).

2. Osseointegration status and peri-implantitis

A significant association was found between study group and early osseointegration outcome (Fisher's exact test with Monte Carlo simulation, $p < 0.001$). Post hoc multiple comparisons showed that early failure rates in Groups I and II were significantly lower than those in Groups IV and V (Table 2). Early failure rates ranged from approximately 4–5% in Groups I-II to 25–30% in Groups IV-V.

Table 2
Comparison of osseointegration success, peri-implantitis prevalence, and marginal bone loss among study groups.

Parameter	Groups					Total	Test Statistic	p
	Group I (n = 48)	Group II (n = 132)	Group III (n = 61)	Group IV (n = 61)	Group V (n = 61)			
Osseointegration								
Failed	2 (4.2)[a]	6 (4.6)[a]	8 (13.1)[ab]	15 (24.6)[b]	18 (29.5)[b]	49 (13.5)	31.515	**< 0.001[x]**
Successful	46 (95.8)	126 (95.5)	53 (86.9)	46 (75.4)	43 (70.5)	314 (86.5)		
Periimplantitis								
Absent	42 (91.3)[ab]	114 (90.5)[b]	44 (83)[ab]	35 (72.9)[a]	29 (67.4)[a]	264 (83.5)	17.474	**< 0.001[x]**
Present	4 (8.7)	12 (9.5)	9 (17)	13 (27.1)	14 (32.6)	52 (16.5)		
Peri-implant Bone Loss (mm)	3.2 ± 0.73[bc]	2.98 ± 0.49[c]	4.51 ± 0.3[a]	4.55 ± 0.76[a]	3.94 ± 0.84[ab]	3.93 ± 0.9	11.093	**< 0.001[y]**

[x]Monte Carlo Simulation Fisher's Exact Test; [y]One-Way ANOVA; Frequency (Percent); Mean ± Standard Deviation; [a–] There is no difference between groups sharing the same letter

Analyses of peri-implantitis were limited to implants that achieved osseointegration (n = 314). The prevalence of peri-implantitis differed significantly among the groups (Fisher's exact test with Monte Carlo simulation, $p < 0.001$). Post hoc testing showed that peri-implantitis prevalence in Group II was significantly lower than in Groups IV and V (Table 2). Across the cohort, peri-implantitis prevalence increased from approximately 9–10% in Groups I-II to 27–33% in Groups IV-V.

Among implants diagnosed with peri-implantitis, marginal bone loss differed significantly between groups (one-way ANOVA, $p < 0.001$). Post hoc comparisons showed that mean marginal bone loss in Groups III and IV was significantly higher than in Groups I and II (Table 2).

3. Horizontal gap width and grafting

Analyses of horizontal gap width and graft use were performed exclusively for Groups II-V; Group I was excluded. The median horizontal gap width differed significantly among these groups (Kruskal-Wallis H = 16.122, p = 0.001). Post hoc analyses showed that the median gap width in Group V was significantly lower than in Groups II, III and IV (Table 3).

Table 3
Comparison of peri-implant gap dimensions and grafting status among groups in immediate implant placement.

Parameter	Groups				Total	Test Statistic	p
	Group II (n = 132)	Group III (n = 61)	Group IV (n = 61)	Group V (n = 61)			
Gap size (mm)	3 (1: 6)[a]	3.5 (1: 6)[a]	3 (1: 6)[a]	2 (1: 4)[b]	3 (1: 6)	16.122	**0.001[x]**
Gap size							
≤ 2 mm	16 (25)	9 (28.1)	11 (34.4)	18 (54.6)	54 (33.5)	12.206	0.058[y]
2–4 mm	35 (54.7)	14 (43.8)	13 (40.6)	13 (39.4)	75 (46.6)		
≥ 5 mm	13 (20.3)	9 (28.1)	8 (25)	2 (6.1)	32 (19.9)		
Graft Status							
Grafted	28 (43.8)	15 (46.9)	15 (46.9)	15 (46.9)	73 (45.6)	0.207	0.990[y]
Non-grafted	36 (56.3)	17 (53.1)	17 (53.1)	17 (53.1)	87 (54.4)		

[x]Kruskal Wallis H Test; [y]Monte Carlo Simulation Fisher's Exact Test; Median (Minimum: Maximum); Frequency (Percent); [a–b] There is no difference between groups sharing the same letter

There was no statistically significant association between gap width categories (< = 2 mm, 2–4 mm, >= 5 mm) and group distribution (Fisher's exact test with Monte Carlo simulation, p = 0.058) (Table 3). The frequency of graft use was similar across groups (Fisher's exact test with Monte Carlo simulation, p = 0.990), with comparable grafting rates in Groups II-V (Table 3).

4. ROC analysis for horizontal gap width

Group-based ROC analysis showed that horizontal gap width did not demonstrate statistically significant diagnostic performance for predicting early osseointegration failure in any individual group (all p > 0.05). When all implants were analysed collectively, the area under the curve (AUC) for gap width was 0.527 (p = 0.640), and no clinically meaningful cut-off value was identified to distinguish implants with and without

early failure. This finding indicates poor discriminative ability of horizontal gap width in this cohort (Table 4). Therefore, additional sensitivity and specificity parameters were not reported.

Table 4
ROC analysis for the diagnostic accuracy of gap size (mm) in predicting osseointegration failure

Parameter	Groups	AUC (%95 GA)	p
Gap size (mm)	Group II (n = 132)	0.795 (0.495: 1)	0.086
	Group III (n = 61)	0.667 (0.379: 0.954)	0.209
	Group IV (n = 61)	0.594 (0.332: 0.857)	0.414
	Group IV (n = 61)	0.523 (0.319: 0.727)	0.825
	Overall	0.527 (0.401: 0.653)	0.640
AUC (%95 GA): Area Under the Curve (%95 Confidence Interval)			

5. Cox regression analysis for early osseointegration failure

5.1. Univariate analysis

Compared with Group I, the risk of early osseointegration failure was 5.812-fold higher in sockets with endo-perio lesions (Group IV; HR = 5.812, 95% CI: 1.328–25.436, p = 0.019) and 6.182-fold higher in failed implant replacement sites (Group V; HR = 6.182, 95% CI: 1.434–26.655, p = 0.015).

At endodontic extraction sites (Group II), the risk did not differ significantly from that of Group I (HR = 0.906, p = 0.903). Likewise, at periodontal extraction sites (Group III), the observed increase in risk was not statistically significant (HR = 2.319, p = 0.288).

In univariate models, surgical and clinical variables — including horizontal gap width (HR = 1.159, p = 0.237), graft use (HR = 1.673, p = 0.173), age (p = 0.782), sex (p = 0.224) and all gap x graft categories — showed no significant association with early osseointegration failure (all p > 0.05).

5.2. Multivariate analysis

In the multivariate Cox model adjusted for age, sex, group allocation, horizontal gap width, grafting status and gap x graft categories, none of the variables reached statistical significance. Horizontal gap width (HR = 1.170, p = 0.659), age (HR = 1.013, p = 0.431), sex (HR = 0.825, p = 0.627) and all gap x graft categories were non-significant predictors of early osseointegration failure (Table 5).

Table 5
Cox Regression analysis of independent factors affecting osseointegration failure

Parameter	Univariate		Multiple	
	HR (%95 GA)	p	HR (%95 GA)	p
Groups				
Group I (n = 48)	Reference			
Group II (n = 132)	0.906 (0.183: 4.491)	0.903	---	---
Group III (n = 61)	2.319 (0.491: 10.965)	0.288	---	---
Group IV (n = 61)	5.812 (1.328: 25.436)	**0.019**	---	---
Group IV (n = 61)	6.182 (1.434: 26.655)	**0.015**	---	---
Gap size (mm)	1.159 (0.908: 1.479)	0.237	1.17 (0.583: 2.345)	0.659
Graft status				
Grafted	Reference			
Non-grafted	1.673 (0.799: 3.503)	0.173	---	---
Age	1.003 (0.98: 1.027)	0.782	1.013 (0.981: 1.047)	0.431
Gender				
Male	Reference			
Female	0.697 (0.389: 1.247)	0.224	0.825 (0.379: 1.793)	0.627
Greft x gap				
Grafted ≤ 2 mm	Reference			
Grafted 2–4 mm	0.515 (0.104: 2.553)	0.416	0.431 (0.061: 3.065)	0.401
Grafted ≥ 5 mm	1.904 (0.453: 8)	0.379	1.258 (0.072: 21.907)	0.875
Non-grafted ≤ 2 mm	1.653 (0.437: 6.251)	0.459	1.817 (0.475: 6.954)	0.383
Non-grafted 2–4 mm	1.192 (0.306: 4.644)	0.800	1.129 (0.196: 6.492)	0.892
Non-grafted ≥ 5 mm	2.804 (0.668: 11.776)	0.159	1.662 (0.084: 32.911)	0.739
HR (%95 CI): Hazard Ratio (%95 Confidence Interval)				

6. Cox regression analysis for peri-implantitis

6.1. Univariate analysis

Among implants that remained osseointegrated (n = 314), compared with Group I, the risk of peri-implantitis was 3.11-fold higher in sockets with endo-perio lesions (Group IV; HR = 3.110, 95% CI: 1.012–9.557, p = 0.048). In Group V, a similar increase in risk was observed but did not reach conventional significance (HR = 3.002, 95% CI: 0.987–9.132, p = 0.053).

Among gap x graft subcategories, the grafted 2–4 mm gap category had a significantly lower risk of peri-implantitis than the reference category of grafted < = 2 mm gaps (HR = 0.245, 95% CI: 0.074–0.816, p = 0.022). No significant associations were observed for the other gap x graft categories (all p > 0.05).

Per univariate analyses, horizontal gap width as a continuous variable (HR = 1.061, p = 0.706), graft use (HR = 0.754, p = 0.479), age (p = 0.928) and sex (p = 0.105) were not significantly associated with peri-implantitis.

6.2. Multivariate analysis

In the multivariate Cox model, adjusted for age, sex, group allocation, horizontal gap width, grafting status and gap x graft categories, none of the variables significantly influenced peri-implantitis risk (all p > 0.05). The risk-reducing trend observed for grafted 2–4 mm gaps in the univariate model persisted but did not reach statistical significance (HR = 0.171, 95% CI: 0.026–1.117, p = 0.065) (Table 6).

Table 6
Cox Regression analysis of independent factors affecting the development of peri-implantitis.

Parameter	Univariate		Multiple	
	HR (%95 GA)	p	HR (%95 GA)	p
Groups				
Group I (n = 48)	Reference			
Group II (n = 132)	0.9 (0.29: 2.792)	0.855	---	---
Group III (n = 61)	1.287 (0.39: 4.249)	0.679	---	---
Group IV (n = 61)	3.11 (1.012: 9.557)	**0.048**	---	---
Group IV (n = 61)	3.002 (0.987: 9.132)	0.053	---	---
Gap size (mm)	1.061 (0.778: 1.447)	0.706	1.236 (0.526: 2.908)	0.627
Graft status				
Grafted	Reference			
Non-grafted	0.754 (0.345: 1.649)	0.479	---	---
Age	1.001 (0.979: 1.024)	0.928	0.999 (0.964: 1.035)	0.956
Gender				
Male	Reference			
Female	0.623 (0.351: 1.104)	0.105	0.98 (0.418: 2.302)	0.964
Graft x gap				
Grafted ≤ 2 mm	Reference			
Grafted 2–4 mm	0.245 (0.074: 0.816)	**0.022**	0.171 (0.026: 1.117)	0.065
Grafted ≥ 5 mm	0.632 (0.166: 2.411)	0.502	0.312 (0.014: 6.784)	0.458
Non-grafted ≤ 2 mm	0.286 (0.076: 1.082)	0.065	0.299 (0.077: 1.165)	0.082
Non-grafted 2–4 mm	0.328 (0.105: 1.018)	0.054	0.233 (0.038: 1.426)	0.115
Non-grafted ≥ 5 mm	1.086 (0.284: 4.153)	0.904	0.533 (0.024: 12.066)	0.693
HR (%95 CI): Hazard Ratio (%95 Confidence Interval)				

DISCUSSION

The age distribution observed in the study groups aligned with established age-dependent patterns of tooth loss. Endodontically compromised teeth were more prevalent among younger patients, consistent

with previous findings that tooth loss in young adults is primarily linked to pulpal pathologies [30, 31]. Conversely, extractions due to periodontal breakdown or implant failure were more common in older individuals, reflecting the progressive nature of periodontitis and the accumulation of late biological complications around teeth and implants over time [13, 32]. To better isolate site-specific biological effects, the cohort was limited to individuals with low systemic risk, thereby reducing potential confounding by systemic factors.

The balanced sex distribution and comparable follow-up durations across groups minimized potential confounding by these variables and established a consistent baseline for analyzing time-dependent biological complications. Age was included as a covariate in the Cox regression models, enabling risk estimates to be interpreted primarily in relation to the local pathobiology of the socket rather than demographic or temporal influences. Although age was statistically controlled, the greater biological vitality of younger bone in Group II may have contributed to more favorable outcomes compared with older sites with a negative biological legacy.

The findings indicate that the implant site's pathological history is a critical biological determinant of outcome, exerting a greater influence on osseointegration success than standardized surgical technique alone. Despite a largely uniform protocol, Groups IV and V exhibited substantially higher early failure rates, suggesting that a compromised site history can significantly reduce the biological capacity for osseointegration [30, 31].

Significantly elevated failure rates in Groups IV and V provide clinical evidence for a “negative biological legacy” at compromised sites [14, 33–35]. This legacy may manifest as cumulative tissue alterations, including fibrotic healing, reduced vascularity, and persistent or difficult-to-eliminate microbial niches, which collectively undermine the regenerative capacity required for stable bone–implant integration [8, 30–33]. Cox regression analyses confirmed that both Group IV and Group V had similarly high and significantly increased risks of early osseointegration failure compared with Group I. This finding indicates that, despite differing etiological origins, both scenarios result in a negative biological legacy of comparable clinical severity. In Group V, this legacy is clinically evident as a tissue bed altered by previous implant failure, with reduced healing and remodeling capacity at the time of the new osteotomy [14, 36]. In Group IV, the increased risk appears to result from the combined detrimental effects of concurrent pulpal and periodontal pathologies. Unlike isolated lesions, combined endodontic–periodontal defects create a dual pathway for infection, with communication between the root canal system and the marginal periodontium establishing a more complex and persistent microbial environment [13, 34, 37]. Even with thorough debridement, the anatomical complexity of these lesions may prevent complete biofilm removal, often leading to more extensive bone destruction that compromises both crestal stability and apical healing potential. The ongoing inflammatory burden in such sites likely impedes the transition from the inflammatory to the proliferative phase of osseointegration. This observation reinforces the concept that the complexity and extent of pre-existing infection, rather than the mere presence of bacteria, ultimately determine the biological outcome [34, 38].

In contrast, Group II exhibited low early failure rates. In these cases, endodontic infection is typically confined to the periapical region, preserving the crestal bone architecture and periodontal ligament support. Furthermore, meticulous debridement at the time of extraction can substantially reduce the microbial load in anatomically accessible defects [3, 20, 40]. Collectively, these factors create a more favorable biological environment for immediate implant placement, even in the presence of prior infection.

These results challenge the simplistic binary classification of extraction sockets as “infected” or “non-infected.” Instead, they support a biologically graded risk assessment in which clinical decision-making for immediate implant placement considers the depth and pattern of infection spread, the presence or absence of a periodontal component, and the extent and morphology of residual bone support. Within this framework, indications can be stratified into biologically meaningful risk categories, such as lower risk in isolated endodontic sockets and higher risk in combined endodontic–periodontal lesions or failed implant sites. This approach may more effectively guide patient selection, the timing and sequencing of therapy, and prognostic counseling [41, 42].

Horizontal gap width has traditionally been considered a pivotal technical parameter in immediate implant placement, influencing primary stability, early bone healing, and maintenance of the buccal contour [11, 27]. However, the present findings indicate that its prognostic value is not absolute and is fundamentally modulated by the site’s biological context. In Group V, the median gap width was intentionally maintained at approximately 2 mm through an undersized osteotomy to maximize primary stability. Despite this favorable mechanical configuration, Group V exhibited the poorest osseointegration outcomes. This result illustrates a clinically significant “gap paradox,” in which a mechanically optimal, narrow gap does not prevent early failure [11, 12, 14, 34]. This paradox highlights that adverse biological factors, including chronic micro-inflammation and the negative biological legacy of prior disease, may outweigh even favorable mechanical conditions [14, 34, 43]. Biologically, while a narrow gap facilitates close implant–bone contact, it may also create a confined, potentially hypoxic environment in compromised sites. This spatial limitation reduces the volume available for stable fibrin clot formation, restricts infiltration by osteoprogenitor and immune-regulatory cells, and may impede the diffusion of angiogenic mediators necessary for early healing. These limitations are particularly critical in previously infected or metabolically exhausted sockets, where regenerative potential is already diminished.

In contrast, gaps of 2–4 mm in this study consistently showed a protective association with peri-implantitis in univariate analysis. Although this association did not reach statistical significance after multivariable adjustment, the consistent direction of the effect across analyses suggests a biologically plausible trend that warrants further investigation. In specific defect morphologies, this intermediate gap may provide sufficient biological space for clot stability, graft particle integration, and neovascularization, thereby partially mitigating unfavorable baseline biology [29, 35]. Nevertheless, this finding should not be interpreted as a definitive clinical threshold; rather, it may represent a functional “Goldilocks zone” within this cohort and requires validation in controlled prospective studies.

Receiver operating characteristic analysis supports the secondary role of gap width, as the horizontal gap did not show meaningful discriminatory ability in predicting early failure. The absence of a significant cutoff suggests that decision models based solely on millimetric thresholds are unlikely to accurately reflect the biological risk landscape of immediate implant placement, particularly in previously infected sites. Furthermore, the lack of a consistent gradient between gap width and failure rates across other groups reinforces the interpretation that horizontal gap width should be considered a secondary modifier of risk rather than a primary determinant.

Peri-implantitis prevalence remained relatively low in Group II but was substantially higher in Groups IV and V. Cox regression analyses corroborated this gradient: the hazard ratio for peri-implantitis was significantly increased in Group IV, with a similar, though borderline, trend in Group V. These estimates were derived exclusively from implants that had achieved osseointegration. Consequently, in Group V, overall biological vulnerability is likely underestimated, as the most susceptible implants fail during early healing and are excluded from long-term evaluation.

The severity of MBL after peri-implantitis was strongly influenced by periodontal history. The mean MBL in Groups III and IV approached 4.5 mm and was significantly higher than in Group II, indicating that a history of periodontitis is associated with both increased disease incidence and more extensive hard-tissue breakdown once peri-implant pathology occurs. This pattern is unlikely to be attributable solely to local anatomical factors or plaque accumulation. Experimental and clinical evidence indicate that periodontitis can induce persistent phenotypic changes in immune and bone cells through epigenetic reprogramming, thereby shifting the host response toward a more pro-inflammatory, bone-destructive profile [36, 37]. In this context, the present findings provide clinical support for the hypothesis that an “epigenetic memory” of prior inflammation may predispose peri-implant tissues to more pronounced breakdown in response to biofilm challenge [37–40].

Collectively, these findings delineate a two-stage risk profile. Groups IV and V are disadvantaged at both stages, showing high early failure rates and, among surviving implants, a higher risk of peri-implantitis and greater bone loss. Group III occupies an intermediate position; thorough debridement at extraction may sufficiently reduce the local microbial burden to permit acceptable early osseointegration, yet the underlying host susceptibility to excessive inflammatory bone loss persists and becomes apparent once peri-implant disease develops. In contrast, Group II represents the most favorable scenario, characterized by a more localized infection pattern and, when combined with meticulous decontamination and structured supportive care, a comparatively stable course for both osseointegration and long-term peri-implant tissue health [4, 41].

Several limitations must be acknowledged when interpreting these findings. This retrospective, single-center study relied on existing clinical and radiographic records. The sample was intentionally limited to a highly selected, low-systemic-risk cohort of systemically healthy, non-smoking, non-diabetic patients treated at mandibular molar sites. This restriction aimed to minimize systemic confounding and isolate the influence of local site biology on outcomes; however, it also limits the external generalizability of the results to broader implant populations, which often include older, medically compromised, or smoking

patients. Furthermore, all surgeries were performed by a single experienced periodontist using one implant system. This high level of standardization reduces operator- and device-related variability and strengthens internal validity, but it may further restrict the applicability of the findings to other clinicians, implant designs, and clinical settings.

Several aspects of measurement and diagnosis warrant consideration. Marginal bone levels were assessed using standardized digital periapical radiographs obtained with the parallel technique and calibrated in dedicated software using known implant platform dimensions. This two-dimensional approach reflects routine clinical practice and is widely accepted for monitoring marginal bone changes; however, it does not provide three-dimensional information on buccal or lingual plate thickness or defect morphology, which would require cone-beam computed tomography. Peri-implantitis was diagnosed at the last available follow-up visit based on clinical parameters and radiographic evidence of bone loss. Variability in probing force, local inflammation at the time of examination, and differences in patient attendance may have introduced measurement and detection bias. Horizontal gap width was measured intraoperatively with a periodontal probe and recorded as both a continuous and a categorical variable. This semi-quantitative, manual method lacks the precision of three-dimensional imaging and carries a risk of measurement error, particularly in inflamed and bleeding surgical fields. Nevertheless, it closely mirrors real-world surgical decision-making, thereby conferring ecological validity regarding how gap-related choices are made in daily practice.

Graft allocation was not randomized but was determined intraoperatively by the surgeon based on multiple operator-dependent factors, including defect morphology, buccal wall integrity, implant primary stability, and patient preference regarding allograft use. Confounding by indication, in which sockets perceived to have better regenerative potential were more likely to receive grafting within a given range, cannot be excluded and may partly account for the observed association. Future prospective, ideally randomized, studies are required to clarify the independent contributions of grafting and gap width to long-term outcomes.

Although the total number of implants was relatively high, the number of events was modest. This likely reduced the statistical power of multivariable Cox models and increased the risk of type II error, particularly for trends that appeared clinically meaningful yet did not reach conventional significance. Within the limits of this sample, sex did not emerge as a significant predictor in the Cox models, and no clear sex-specific interaction pattern was observed in the older, periodontally or implant-compromised groups. However, the study was not powered for detailed sex-stratified analyses, and subtle sex-related differences in bone metabolism or implant response cannot be excluded.

Although all patients were enrolled in a structured maintenance program, adherence to recall intervals and home oral hygiene recommendations could not be fully standardized or controlled. In high-risk groups, such as those with endodontic–periodontal lesions and failed implant replacement sites, variable compliance and potential loss to follow-up may have influenced survival and peri-implantitis estimates. Collectively, these limitations indicate that, while the adjusted models support a predominant role of extraction or explantation indication and local site biology in determining risk patterns, residual

confounding related to measurement imprecision, operator-dependent decisions, and behavioral factors remains possible and should be considered when extrapolating these findings to broader clinical contexts.

Conclusion

Within the limitations of this retrospective cohort, the findings indicate that the biological history of the extraction or explantation site is a primary determinant of long-term outcomes in IIP. When surgical and technical variables are standardized, site biology, as reflected by the pathological indication, emerges as a critical factor that often supersedes conventional mechanical parameters. Sockets with combined endodontic–periodontal lesions and sites receiving immediate replacement of failed implants consistently represent the highest-risk scenarios, exhibiting a two-stage vulnerability that compromises both initial osseointegration and long-term peri-implant stability.

The present data highlight the prognostic limitations of using horizontal gap width as a stand-alone decision-making metric. The observed gap paradox at failed implant replacement sites, where the narrowest median gap coincided with the highest failure rate, demonstrates that even optimal mechanical configurations cannot compensate for the adverse biological legacy of prior disease. In this context, anatomical measurements serve as secondary modifiers constrained by the underlying site biology. Furthermore, the trend toward a protective association for grafted 2–4 mm gaps suggests a potential biological buffer strategy; however, this remains a hypothesis-generating observation that requires prospective validation before definitive clinical thresholds can be established.

In contrast, isolated endodontic and periodontal extraction sites present more predictable, lower-risk biological outcomes when managed with meticulous decontamination and structured supportive care. Clinicians should remain vigilant about the epigenetic memory of periodontally susceptible individuals, as a history of periodontitis remains a significant risk factor for more aggressive marginal bone loss after peri-implant inflammation. This propensity underscores the need for rigorous, long-term maintenance in this population, regardless of successful early integration.

Ultimately, these results support a paradigm shift in immediate implant placement, advocating a transition from rigid millimeter-based rules to a risk-adaptive framework that prioritizes site biology in clinical decision-making. In this model, technical choices, such as gap management and grafting strategies, are not applied as isolated criteria but are instead tailored to the unique pathological history and regenerative capacity of each individual site.

Abbreviations

• **ANOVA**
Analysis of Variance
• **AUC**
Area Under the Curve

• **CI**
Confidence Interval
• **HR**
Hazard Ratio
• **ICC**
Intraclass Correlation Coefficient
• **IIP**
Immediate Implant Placement
• **L**
PRF–Leukocyte–and Platelet–Rich Fibrin
• **MBL**
Marginal Bone Level
• **ROC**
Receiver Operating Characteristic
• **SLA**
Sand–Blasted, Acid–Etched

Declarations

Ethical approval and consent to participate

All patients provided written informed consent at the time of radiographic examination, stating that, as the institution is a teaching hospital, their radiographic images may be used for scientific, educational, or research purposes while keeping personal identifiers confidential. The study was approved by the Uşak University Non-Interventional Clinical Research Ethics Committee (Approval No: 670-670-18).

Consent for publication

Not applicable

Availability of data and materials

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

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

FK conceived and designed the study, conducted the statistical analysis, and drafted the manuscript. DDB, ECÇ and MK coordinated data collection and contributed to the literature review and final editing of the manuscript. All authors have read and approved the final version of the manuscript.

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References

1. Mello C, Lemos C, Verri F, dos Santos D, Goiato M, Pellizzer E (2017) Immediate implant placement into fresh extraction sockets versus delayed implants into healed sockets: A systematic review and meta-analysis. Int J Oral Maxillofac Surg 46(9):1162–1177
2. Akin R, Chapple AG (2022) Clinical advantages of immediate posterior implants with custom healing abutments: Up to 8-year follow-up of 115 cases. J Oral Maxillofac Surg 80(12):1952–1965
3. Sebring D, Kvist T, Derks J (2019) Indications for extraction before implant therapy: Focus on endodontic status. J Endod 45(5):532–537
4. Flanagan D (2016) Implant placement in failed endodontic sites: A review. J Oral Implantol 42(2):224–230
5. Sisli SN, Pektas ZO (2020) What is the role of endodontic predisposing factors in early implant failure? J Oral Implantol 46(5):491–495
6. Gamborena I, Sasaki Y, Blatz MB (2020) Updated clinical and technical protocols for predictable immediate implant placement. J Cosmet Dent ;35(4)
7. Chen B, Zhu Y, Lin M, Zhang Y, Li Y, Ouyang X et al (2024) Expert consensus on the diagnosis and therapy of endo-periodontal lesions. Int J Oral Sci 16(1):55
8. Amid R, Kadkhodazadeh M, Moscowchi A (2023) Immediate implant placement in compromised sockets: A systematic review and meta-analysis. J Prosthet Dent 130(3):307–317
9. Staedt H, Rossa M, Lehmann KM, Al-Nawas B, Kämmerer PW, Heimes D (2020) Potential risk factors for early and late dental implant failure: A retrospective clinical study on 9080 implants. Int J Implant Dent 6(1):81
10. Kang D-W, Kim S-H, Choi Y-H, Kim Y-K (2019) Repeated failure of implants at the same site: A retrospective clinical study. Maxillofac Plast Reconstr Surg 41(1):27
11. Naji B, Abdelsameaa S, Alqutaibi A, Ahmed WS (2021) Immediate dental implant placement with a horizontal gap more than two millimetres: A randomized clinical trial. Int J Oral Maxillofac Surg 50(5):683–690
12. Seyssens L, Eeckhout C, Cosyn J (2022) Immediate implant placement with or without socket grafting: A systematic review and meta-analysis. Clin Implant Dent Relat Res 24(3):339–351
13. Evans M (2023) The endodontic-periodontal juncture: Where two worlds meet. An overview of endo-perio lesions. Aust Dent J 68(Suppl):S56–S65

14. Oh S-L, Shiau HJ, Reynolds MA (2020) Survival of dental implants at sites after implant failure: A systematic review. J Prosthet Dent 123(1):54–60
15. Jang Y-E, Kim Y, Kim S-Y, Kim BS (2024) Predicting early endodontic treatment failure following primary root canal treatment. BMC Oral Health 24(1):327
16. Pontoriero DIK, Grandini S, Spagnuolo G, Discepoli N, Benedicenti S, Maccagnola V et al (2021) Clinical outcomes of endodontic treatments and restorations with and without posts up to 18 years. J Clin Med 10(5):908
17. Loos BG, Needleman I (2020) Endpoints of active periodontal therapy. J Clin Periodontol 47:61–71
18. Petsos H, Schacher B, Ramich T, Nickles K, Dannewitz B, Arendt S et al (2020) Retrospectively analysed tooth loss in periodontally compromised patients: Long-term results 10 years after active periodontal therapy—Patient-related outcomes. J Periodontal Res 55(6):946–958
19. Wong I, Ton A, Cassidy AJ, Fozzard N, Sharma LA, Love RM, Sharma A (2024) A retrospective study on the prognostic factors and success, survival, and failure outcomes of treated endodontic-periodontal lesions. Clin Exp Dent Res ;10(1)
20. Chatzopoulos GS, Wolff LF (2023) Dental implant failure and factors associated with treatment outcome: A retrospective study. J Stomatol Oral Maxillofac Surg 124(2):101314
21. Kochar SP, Reche A, Paul P (2022) The etiology and management of dental implant failure: A review. Cureus 14(10):e30455
22. Clauser T, Lin GH, Lee E, Del Fabbro M, Wang HL, Testori T (2022) Risk of early implant failure in grafted and non-grafted sites: A systematic review and meta-analysis. Int J Oral Implantol (Berl) 15(1):31–41
23. Grigoras RI, Cosarca A, Ormenișan A (2024) Early implant failure: A meta-analysis of 7 years of experience. J Clin Med ;13(7)
24. Diaz P, Gonzalo E, Villagra LJG, Miegimolle B, Suarez MJ (2022) What is the prevalence of peri-implantitis? A systematic review and meta-analysis. BMC Oral Health 22(1):449
25. Romandini M, Berglundh J, Derks J, Sanz M, Berglundh T (2021) Diagnosis of peri-implantitis in the absence of baseline data: A diagnostic accuracy study. Clin Oral Implants Res 32(3):297–313
26. Lyris V, Millen C, Besi E, Pace-Balzan A (2021) Effect of leukocyte and platelet rich fibrin (L-PRF) on stability of dental implants: A systematic review and meta-analysis. Br J Oral Maxillofac Surg 59(10):1130–1139
27. Deporter D, Khoshkhounejad AA, Khoshkhounejad N, Ketabi M (2021) A new classification of peri implant gaps based on gap location (A case series of 210 immediate implants). Dent Res J (Isfahan) 18(1):29
28. Levine RA, Dias DR, Wang P, Araújo MG (2022) Effect of the buccal gap width following immediate implant placement on the buccal bone wall: A retrospective cone-beam computed tomography analysis. Clin Implant Dent Relat Res 24(4):403–413
29. Liñares A, Dopico J, Magrin G, Blanco J (2023) Critical review on bone grafting during immediate implant placement. Periodontol 2000 93(1):309–326

30. Cooper LF, Shirazi S (2022) Osseointegration—the biological reality of successful dental implant therapy: A narrative review. Front Oral Maxillofac Med ;4
31. Dutta SR, Passi D, Singh P, Atri M, Mohan S, Sharma A (2020) Risks and complications associated with dental implant failure: Critical update. Natl J Maxillofac Surg 11(1):14–19
32. Guglielmotti MB, Olmedo DG, Cabrini RL (2019) Research on implants and osseointegration. Periodontol 2000 79(1):178–189
33. Pandey C, Rokaya D, Bhattarai BP (2022) Contemporary concepts in osseointegration of dental implants: A review. BioMed Res Int 2022:6170452
34. Dimaira M (2019) Immediate placement of trabecular implants in sites of failed implants. Int J Oral Maxillofac Implants ;34(5)
35. Sanz M, Lindhe J, Alcaraz J, Sanz-Sanchez I, Cecchinato D (2017) The effect of placing a bone replacement graft in the gap at immediately placed implants: A randomized clinical trial. Clin Oral Implants Res 28(8):902–910
36. Ferreira SD, Martins CC, Amaral SA, Vieira TR, Albuquerque BN, Cota LOM et al (2018) Periodontitis as a risk factor for peri-implantitis: Systematic review and meta-analysis of observational studies. J Dent 79:1–10
37. Darby I (2022) Risk factors for periodontitis & peri-implantitis. Periodontol 2000 90(1):9–12
38. Romandini M, Lima C, Banaco D, Azevedo R, Sanz M (2025) Incidence and risk factors of peri-implantitis over time—A prospective cohort study. J Periodontal Res
39. Larsson L, Kavanagh NM, Nguyen TV, Castilho RM, Berglundh T, Giannobile WV (2022) Influence of epigenetics on periodontitis and peri-implantitis pathogenesis. Periodontol 2000 90(1):125–137
40. Li Q, Lu H, Zhang M, Ye Y, Chen Q, Sun P (2025) Epigenetic factors associated with peri-implantitis: A review. J Zhejiang Univ Sci B 26(7):657–674
41. Saijeva A, Juodzbalys G (2020) Immediate implant placement in non-infected sockets versus infected sockets: A systematic review and meta-analysis. J Oral Maxillofac Res 11(2):e1

Table 1

Table 1 is available in the Supplementary Files section.

Supplementary Files

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- Table1.docx