



Beyond Bone: A Narrative Review of Multifactorial Causes of Dental Implant Failure

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Abstract

Dental implant failure is a multifactorial phenomenon that extends beyond failed osseointegration. This narrative review discusses the major biological, mechanical, prosthetic, systemic, and patient-related factors influencing implant survival. Biological complications such as peri-implant mucositis, peri-implantitis, bacterial accumulation, microleakage, and inadequate keratinized mucosa, along with mechanical complications including screw loosening, implant and abutment fractures, and prosthetic failure, are reviewed. These factors may interact and contribute to progressive tissue destruction or structural failure. A comprehensive approach involving proper treatment planning, appropriate prosthetic design, plaque control, management of risk factors, and regular maintenance is essential for preventing complications and improving the long-term success of dental implants.

Keywords: Dental implants; Implant failure; Osseointegration; Peri-implantitis; Mechanical complications; Prosthetic complications; Implant maintenance.

Introduction

The rehabilitation of edentulous patients has historically represented one of the biggest challenges in dentistry. For a long time, conventional removable and fixed prostheses remained the principal treatment choices for tooth replacement, despite their inherent functional, biomechanical, and psychosocial limitations. The introduction of dental implants in prosthodontic rehabilitation provided a predictable treatment outcome with enhanced biomechanical stability, improved esthetics, and long-term functional success. The pioneering work of Per-Ingvar Brånemark (1929–2014), particularly his discovery and clinical application of osseointegration, was a major breakthrough in modern dentistry and profoundly transformed the management of partial and complete edentulism worldwide.[1]

Clinically, the implant therapy has the favorable outcome in most of the cases. Through clinical

trials and longitudinal studies, Brånemark showed that the success rate for titanium implants was 90% or higher even after 10 years of use. Clinical data continue to prove that: dental implants have an overall success rate of up to 98% in healthy patients, with a 10-year survival rate of approximately 90–95%.[2]

Initially, primary implant failure was mainly attributed to inadequate osseointegration, defined as the host bone's failure to maintain and form a stable biological interface with the implant surface during the healing. This view considered osseointegration as the principal determinant factor of implant success. However, increasing clinical and scientific evidence has led to a re-evaluation of this concept. Today, implant failure is recognized as a multifactorial phenomenon resulting from the complex interaction of biological,

mechanical, biomechanical, prosthetic, and patient-related factors.

In light of this evolving clinical scenario, the present narrative review aims to transcend the traditional osseointegration-focused model of implant failure and comprehensively examine the various factors influencing the long-term outcomes of osseointegrated dental implants. By systematically integrating evidence on biological, biomechanical, prosthetic, systemic, and behavioural determinants, this review focuses on the comprehensive understanding of the multifaceted nature of implant failure and its impact on successful implant therapy.

Biologic Causes of Implant Failure

Biological failures involve processes linked to bacterial biofilm and pathogens that display destructive virulence factors. These failures are arguably more insidious, developing over months to years, and by the time they become clinically apparent, significant and irreversible damage may already have occurred.[3]

Biological causes of dental implant failure are multifactorial and involving local and systemic factors that adversely impact the maintenance of healthy peri-implant hard and soft tissues. Periimplant disease is a significant biological factor in implant failure. A biofilm-driven inflammatory condition that affects the peri-implant tissues is known as peri-implantitis. It starts with inflammation of the peri-implant mucosa and progresses to the progressive loss of supporting alveolar bone.

The peri-implant biofilm is a structured and organized microbial community, predominantly composed of Gram-negative anaerobic pathogens, including *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*, which imitate a host-mediated inflammatory response leading to peri-implant tissue destruction and subsequent implant compromise.[4] The implant surface, unlike natural tooth structure, lacks a periodontal ligament and the rich vascular supply, reducing the capability of mounting an effective local defense.

Peri-Implant Mucositis

Peri-implant mucositis is the earliest clinical sign of peri-implant disease and indicates tissue compromise around the implant. According to the The American Academy of Periodontology, in the absence of increasing bone loss beyond the physiological bone remodelling associated with post-surgical healing, peri-implant mucositis is an inflammatory lesion limited to the soft tissues around a dental implant. The main cause is bacterial biofilm buildup on the implant surface.

Importantly, peri-implant mucositis is a reversible condition, as effective professional biofilm removal combined with adequate patient-performed oral hygiene measures can restore periimplant tissue health. However, peri-implant mucositis is widely recognized as a known predecessor to peri-implantitis, which, may worsen if untreated, and result in significant periimplant bone loss, ultimately threatening implant survival.[5]

Peri-Implantitis

Peri-implantitis causes irreversible alveolar bone destruction and, if untreated, progresses in a “non-linear and accelerating” manner, faster than periodontitis. [5] Dental implants do not have a periodontal ligament like normal teeth do, which serves as a specialized connective tissue structure containing collagen fibers, blood vessels, and neural elements that provide attachment, sensory function, and an effective biological defense against microbial invasion. As a result, peri-implant tissues differ anatomically and physiologically from the periodontium around natural teeth. [Fig 1]

The vascular supply of the peri-implant mucosa mainly comes from the terminal branches of the supra-periosteal vessels, as the periodontal ligament vascular plexus present around natural teeth is absent. Thus, the richly vascularized connective tissue near root cementum in natural dentition is largely missing around implants.

The increased susceptibility of peri-implant tissues to bone loss may be due to the absence of collagen fiber insertion into the implant surface, unlike the perpendicular collagen fiber attachment to natural tooth cementum. Studies show that even after removing ligatures, peri-implantitis lesions can continue progressing with ongoing bone destruction, unlike periodontal lesions around natural teeth. This

suggests peri-implantitis may become a self-sustaining disease once triggered, continuing despite elimination of the original cause.

Microgap at the Implant–Abutment Junction

A biologically critical yet often overlooked infection source is the microscopic gap at the implant–abutment junction. The microgap at the abutment-to-implant junction ranges from 1 and 49 μm ; even when the implant and abutment are properly linked, regardless of the style of connection. **[Fig 2]** This microgap can cause microleakage, permitting the entry of germs and metabolic products, which could lead to soft tissue irritation and jeopardize the implants' durability and clinical success.[6]

As the microgap widens, the risk of bacterial ingress and peri-implant inflammation increases, especially due to the close proximity to the alveolar bone crest and nearby tissues. Different connection designs attempt to mitigate this, and conical and mixed connection appear to perform better in terms of minimizing bacterial leakage compared to simple external or internal hexagonal connections.[7]

Role of Keratinized Mucosa

The width and quality of keratinized mucosa are critical factors for implant success. Proper integration and a biological seal at the mucosa–implant interface are encouraged by mucosal morphologies with a keratinized mucosa width of at least 2 mm and a mucosal thickness of at least 2 mm. This seal lowers the risk of inflammatory peri-implant illnesses, shields the underlying peri-implant bone, and stops pathogen penetration..[8]

Greater plaque accumulation, mucosal recession, deeper interproximal bone levels, and periimplantitis were linked to the lack of keratinized mucosa..[9] A 20-year cohort study found periimplantitis in only 4.2% of implants with keratinized mucosa versus 25% without it—an odds ratio of 6.67. Implants lacking keratinized tissue also showed more bone loss, bleeding during probing, and recession of soft tissues. [10]

The biological rationale is clear: keratinized tissue is denser, more resistant to mechanical trauma from oral hygiene procedures, and better equipped to maintain a stable seal around the implant

neck. Without this protective barrier, the implant is surrounded by softer, more mobile, and more permeable tissue, compromising the protective barrier and increasing susceptibility to bacterial penetration and persistent inflammation.

Host Immune Response and Bone Dysregulation

Once bacteria colonize the peri-implant area, the host immune system triggers a protective inflammatory response; but this defense can also cause tissue damage. Microbial biofilms, worsened by inadequate plaque control and unfavorable prosthetic design, disrupt the physiologic balance by releasing of inflammatory cells that promote osteolytic activity and subsequent bone destruction.[11]

Macrophages, dendritic cells, neutrophils, and mast cells—secrete pro-inflammatory cytokines to fight bacteria. However, these cytokines also stimulate osteoclasts, the cells responsible for bone resorption. Normally, bone resorption and formation are balanced, but in peri-implantitis, this balance shifts toward bone loss. The progression from healthy state to disease is gradual and multifactorial, involving a sequence of biological events that ultimately culminate in a clinical tipping point where peri-implantitis becomes apparent.[12]

Mechanical Causes of Implant Failure

Screw Loosening

The most frequent technical issue in implant dentistry, especially with single-crown restorations, is screw loosening. The implant–abutment joint's mechanical integrity depends on preload—the internal tension created when the screw is tightened. This tension produces a clamping force that keeps the components together. Loosening happens when the joint is subjected to external occlusal forces that exceed this clamping force, decreasing the resistance between the screw threads and the implant's interior hole. **[Fig 3]**

The stability of the screw joint is governed by the relationship between torque, preload, and clamping force. When torque is applied, about 90% of the energy overcomes friction between the screw threads and implant as well as between the screw head and abutment, while only 10%

converts into preload.[12] No machined surface is perfectly smooth; they consist of “peaks and valleys” known as asperities. Upon initial loading, these asperities flatten, causing the screw to “settle” deeper into the implant. This settling effect serves as the primary mechanical catalyst for screw loosening. This microscopic displacement (estimated at 2 to 10 μm) reduces the screw’s elongation and causes a sharp drop in preload.[13] Once preload falls below the threshold of external functional forces, the friction is lost, and the screw begins to back out.[14]

Material and Design Dynamics

The geometry of the implant–abutment interface significantly plays the mechanical durability of the assembly:

- **Internal vs. External Connections:** Internal hex or conical connections offer a deeper “engagement”, protecting the screw from lateral shear forces. However, external hex designs often concentrate the entire lateral load on the screw itself, increasing the risk to loosen and fatigue fracture.[14]
- **Surface Treatments:** Modern screws often feature gold-plating or carbon coatings that act as dry lubricants. These reduce friction, allowing more applied torque to generate preload rather than being lost.[12]
- **Cyclic Fatigue:** Under the repetitive masticatory forces, (up to 300,000 cycles per year), the screw experiences elastic and eventually plastic deformation. If the occlusal load is non-axial — such as in cases of malocclusion or long cantilevers — generating bending moments, which create a “pumping effect” at the joint, accelerating the erosion of the clamping force. [15,16]

Implant Fracture

The fracture of the implant body is a catastrophic mechanical failure that is usually irreversible, often requires the surgical removal of the integrated fixture. It is rarely caused by a single traumatic event; rather, it is typically the culmination of metal fatigue caused by chronic cyclic loading. Repeated stress cycles — particularly in the area of the molar where masticatory forces are the greatest — cause micro-cracks begin to develop through the titanium structure.

Risk is significantly elevated in cases of “biomechanical overload,” such as when a patient suffers from bruxism or when there is a significant crown-to-implant ratio imbalance. In the latter scenario, progressive marginal bone loss leaves a larger portion of the implant unsupported, causing long lever arm that increases the stress at the level of the first few threads of the implant body. **[Fig 4]**

Specific Causes of Implant Fracture

The primary mechanical cause of failure is fatigue, resulting from the cumulative effect of cyclic loading over time:

1. **Biomechanical Overload:** Excessive occlusal forces, specifically in patients with parafunctional habits like bruxism, generate tensions that surpass the fatigue strength of the titanium. It is most frequently observed in the posterior teeth i.e. in molar and premolar regions where masticatory forces are maximum. [17,18]
2. **Progressive Bone Resorption:** When bone loss occurs around the neck of the implant at the marginal area, the fulcrum point shifts apically, which in turn increases the bending moment at the level of the first threads, subjecting the hollow coronal portion of the implant to stresses beyond its design limits.[14,17]

Technical and Design Factors: Small-diameter implants (< 3.75 mm) used in high-load areas are at significantly higher risk. Furthermore, manufacturing defects or a lack of “passive fit” in the prosthesis can cause constant internal strain, decreasing the threshold limit for fatigue-induced fracture. [18,19]

Clinical Management of Implant Fracture

Management strategies focus on either salvaging the remaining fixture or preparing the site for a new implant:

1. **Total Explantation:** The most common approach involves removing the fractured portion with trephine burs or specialized reverse-torque kits. This removes osseointegrated apex but often results in a large bony defect that requires guided bone regeneration (GBR) for placing a new implant. [16,18]

2. **Partial Retention (Sleep-It Strategy):** If the fractured segment is present deep within the bone and is asymptomatic, some clinicians choose to leave the portion in situ and allow the soft tissue to heal over it, provided there is no infection or peri-implantitis.[17]
3. **Prosthetic Modification:** In very few cases where the fracture is very coronal and the internal connection is intact, the remaining fixture may be re-contoured and fitted with a custom abutment, though this is often regarded as a temporary measure due to the compromised structural integrity.[20]

Preventive Strategies

Preventing fracture is more effective than treating it. Key recommendations include:

1. **Selecting Appropriate Diameters:** Utilizing wider-diameter implants in the posterior region for better distribution of occlusal loads.[14]
2. **Occlusal Protection:** Providing patients with occlusal guards (night guards) to mitigate the detrimental effect of bruxism on the implant–prosthesis assembly.[20]
3. **Regular Radiographic Monitoring:** Early detection of marginal bone loss enables timely intervention (such as occlusal adjustment or peri-implant therapy) before the stress becomes critical.[21]

Abutment Fracture

Abutment fracture, involving the failure of the intermediary component or its connecting screw, often stems from stress concentration at the junction where the abutment enters the implant platform. **[Fig 5]** Mechanically, the neck of the abutment screw is the “weakest link”, designed to fail before the implant body to protect the underlying bone and fixture. These fractures often occur due to undetected screw loosening; when the screw is loose, it is no longer protected by the clamping force of the joint, subjecting the metal to shear stresses, it cannot endure. Additionally, imprecise internal connections can cause, “toggling” or lateral micromovement, leading to fatigue at the interface, eventually causing the metal to snap under the functional loads. [16,20]

Prosthesis Fracture

Prosthesis fracture refers to the structural failure of the final restorative crown or bridge, affecting either within the supporting framework or the aesthetic veneering material. Framework fractures often result from insufficient material thickness or poor design, such as extremely long cantilevers that create excessive bending moments during chewing. **[Fig 6]** The use of zirconia and high-strength ceramics has reduced framework fractures in modern dentistry but these materials are susceptible to “chipping” of the overlying porcelain. This veneering failure can occur due to differences in the thermal expansion coefficients between the core and the veneer, or due to “sliding contact” stress during parafunctional movements. If the prosthesis is not “passively fit” — meaning it does not sit on the implants without internal tension — constant strain lowers its fracture resistance under normal biting forces.[15]

Conclusion

With predictable functional and cosmetic results, dental implant therapy has revolutionized the rehabilitation of patients who are either fully or partially edentulous. However, implant failure should no longer be viewed solely as a consequence of failed osseointegration. Rather, it is the result of a complex interaction between biological, mechanical, prosthetic, systemic, and patient related elements that affect the implant's long-term stability.

Biological complications such as peri-implant diseases, inadequate keratinized mucosa, host immune dysregulation, and bacterial accumulation of the implant–abutment junction compromise peri-implant tissue health, while mechanical complications like loosening of prosthetic screw, implant fracture, abutment failure, and prosthetic fractures can jeopardize the structural integrity of implant-supported restorations. These complications are often interconnected, with one factor predisposing or accelerating another, emphasizing that implant failure is rarely attributable to a single cause.

Successful implant therapy therefore requires a comprehensive, patient-centered approach that extends beyond surgical placement. Careful case selection, meticulous treatment planning, appropriate prosthetic design, effective plaque control, management of systemic and behavioural risk factors, and regular maintenance are essential to minimizing complications and improving

long-term outcomes. Future research should focus on identifying predictive biomarkers, improving implant designs and biomaterials, and developing personalized preventive and maintenance protocols that further enhance implant longevity. In the end, understanding the multifaceted nature of implant failure allows medical professionals to transition from a reactive treatment model to a proactive approach meant to maintain peri-implant health and guaranty long-term clinical success.

References

- Brånemark PI, Hansson BO, Adell R, Breine U, Lindström J, Hallén O, et al Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10- year period. *Scand J Plast Reconstr Surg Suppl.* 1977;16:1–132.
- Albrektsson T, Zarb GA, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *Int J Oral Maxillofac Implants.* 1986;1(1):11–25.
- Figuero E, Graziani F, Sanz I, Herrera D, Sanz M. Management of peri-implant mucositis and peri-implantitis. *Periodontol 2000.* 2014;66(1):255–273.
- Alves CH, Russi KL, Rocha NC, Bastos F, Darrieux M, Parisotto TM, Girardello R. Host-microbiome interactions regarding peri-implantitis and dental implant loss. *J Transl Med.* 2022;20(1):425.
- Heitz-Mayfield LJ. Peri-implant mucositis and peri-implantitis: key features and differences. *Br Dent J.* 2024;236(10):791–794.
- D’Ercole S, Dotta TC, Farani MR, Etemadi N, Iezzi G, Comuzzi L, Piattelli A, Petrini M. Bacterial microleakage at the implant-abutment interface: an in vitro study. *Bioengineering.* 2022;9(7):277.
- Sharifi R, Ghaffari T, Ghasemi S, Kafil HS. Evaluation of microgap size and microbial microleakage at the implant fixture–abutment interface in original and compatible abutments. *Biomed Res Int.* 2025;2025:2530986.
- Ríos-Orsorio N, Ladino LG, Guerrero-Torres M. Structure, biology, and function of periimplant soft tissues in health and disease: a comprehensive review of the literature. *J Periodontal Implant Sci.* 2025;55(5):323.
- Kungsadalpipob K, Supanimitkul K, Manopattanasoontorn S, Sophon N, Tangsathian T, Arunyanak SP. The lack of keratinized mucosa is associated with poor peri-implant tissue health: a cross-sectional study. *Int J Implant Dent.* 2020;6(1):28.
- Roccuzzo A, Imber JC, Stähli A, Romandini M, Sculean A, Salvi GE, Roccuzzo M. Role of keratinized mucosa on the risk of peri-implant diseases and soft tissue dehiscence in the posterior mandible — a 20-year prospective cohort study. *J Periodontal Res.* 2025 Jul 21. [Epub ahead of print]
- Ng E, Tay JR, Mattheos N, Bostanci N, Belibasakis GN, Seneviratne CJ. A mapping review of the pathogenesis of peri-implantitis: the biofilm-mediated inflammation and bone dysregulation (BIND) hypothesis. *Cells.* 2024;13(4):315.
- Winkler S, Ring K, Ring JD, Boberick KG. Implant screw mechanics and the settling effect: an overview. *J Oral Implantol.* 2003;29(5):242–245.
- Gali S, Iyer P, Agarwal S. Does torque magnitude influence dental implant abutment screw? A SEM analysis. *J Indian Prosthodont Soc.* 2016;16(4):374–379.
- Gupta S, Gupta H, Tandan A. Technical complications of implant — causes and management: a comprehensive review. *Natl J Maxillofac Surg.* 2015;6(1):3–8.
- Sailer I, Karasan D, Todorovic A, Ligoutsikou M, Pjetursson BE. Prosthetic failures in dental implant therapy. *Periodontol 2000.* 2022;88(1):130–144.
- Jeng MD, Huang TY, Jeng AY. Retrievability of fractured abutment screws in dental implants using three removal techniques: an in vitro pilot study. *J Funct Biomater.* 2026;17(2):85.
- Goiato MC, Haddad MF, Gennari Filho H, Villa LM, Dos Santos DM, Pesqueira AA. Dental implant fractures — aetiology, treatment and case report. *J Clin Diagn Res.* 2014;8(3):300–304.
- Mishra SK, Chowdhary R, Chrcanovic BR. Implant fractures: a systematic review. *J Oral Biol Craniofac Res.* 2019;9(4):364–370.
- Al-Almaie S. Management of broken dental implant abutment in a patient with bruxism: a rare case report and review of literature. *Contemp Clin Dent.* 2017;8(3):485–489.

20. Halstenbach T, Semper-Hogg W, Cieplik F, Würflein E, Kernen F, Metzger M, Schmelzeisen R. Timing and extent of peri-implant bone loss for dental implant removal: a retrospective cross-sectional analysis. Clin Oral Investig. 2026;30(3):111.
21. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Clin Periodontol. 2018;45(Suppl 20):S246–S266.
22. Ramanauskaite A, Obreja K, Schwarz F. Surgical management of peri-implantitis. Current oral health reports. 2020 Sep;7(3):283-303.
23. Kirov D, Stoichkov B. FACTORS AFFECTING THE ABUTMENT SCREW LOOSENING. Journal of IMAB–Annual Proceeding Scientific Papers. 2017 Mar 28;23(1):1505-9.
24. Zeng K, Dong Y, Li L. Retrieval of a fractured implant abutment screw using a microtube extraction device: A dental technique. The Journal of Prosthetic Dentistry. 2024 Nov 1;132(5):867-70.
25. Carlsson GE. Dental occlusion: modern concepts and their application in implant prosthodontics. Odontology. 2009 Jan;97(1):8-17.



Fig 1: Peri Implantitis [21]

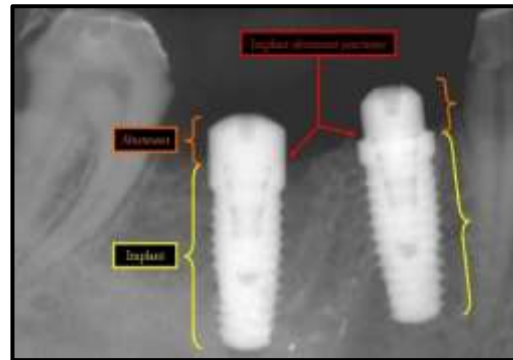


Fig 2: Microgap at Implant Abutment Junction [21]

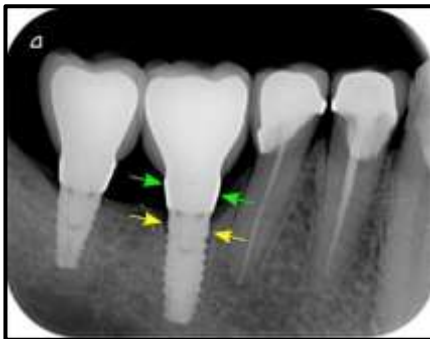


Fig 3: Screw Loosening [23]



Fig 4: Implant Fracture [24]



Fig 5: Abutment Screw Fracture [24]



Fig 6: Prosthesis Fracture [25]