REVIEW ARTICLE

Peri-Implantitis: A Prosthetic Induced Challenge in Implant Dentistry

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Abstract

Background:

Peri-implantitis refers to a biofilm-associated inflammatory disease affecting the tissues surrounding dental implants, leading to progressive destruction of supporting bone. Despite high implant survival rates, biological complications remain a significant concern, with prosthodontic factors increasingly recognized as key contributors to disease initiation and progression.

Objective:

This review aims to analyze prosthodontic causes associated with peri-implantitis and to summarize preventive and management strategies from a prosthodontic perspective.

Methods:

A narrative review of the literature was conducted using electronic databases including PubMed, Google Scholar, and Scopus. Articles published in English focusing on prosthodontic risk factors, implant prosthesis design, retention methods, occlusal considerations, and management strategies were analyzed.

INTRODUCTION

A dental implant or a fixture is a surgical element that connects with the jaw or cranium bone to anchor a dental prosthesis such as a crown, bridge, denture, or facial prosthesis, or to act as an orthodontic anchor. Implants have evolved into a largely useful surgical technique for resolving single or multiple tooth loss.[1] In recent times, dental implantology has revolutionized the field of restorative dentistry, furnishing cases with a dependable and aesthetically pleasing results for tooth relief. The implant's success is based on the factors such as: type of surgical technique used, bone quality, volume, implant shape and surface topographic qualities all of which can be modified as demanded to achieve strong primary trustability and prolonged implant curatives accomplishments.[2]

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Though, alongside the remarkable success of dental implants, challenges such as peri-implantitis have surfaced as a significant concern in the implant dentistry. Peri-implantitis, characterized by inflammation and bone loss around dental implants, can lead to implant failure if not managed instantly and effectively.[3]

Peri-implantitis is characterised by inflammation in the preimplant connective tissue and progressive bone loss.[4] Preimplant conditions are substantially distributed into two types i.e. peri implant mucositis and peri-implantitis depending upon the severity.[5] Peri-implant mucositis is a reversible inflammatory change of the peri-implant soft tissue without bone loss. Peri-implantitis is characterized by inflammatory changes around osseo-integrated implants in function, affecting the mucosa and resulting in the loss of supporting bone around the implant, indicated by ≥6mm probing depth in confluence with profuse bleeding and suppuration.[6] The present review suggests that prosthetic design, type of retention, occlusal scheme, and ease of conservation are not just restorative factors, but important rudiments that directly affect the health and stability of peri-implant tissues.

A comprehensive understanding of prosthodontic etiological factors is essential for precluding, early diagnosis, and effective control of peri-implantitis. Accordingly, this review focuses specifically on prosthodontic causes and discusses preventive and management strategies from a prosthodontic perspective.

According to Glossary of Prosthodontics Terms(2023) Peri-implantitis can be defined as a pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant connective tissue and progressive loss of supporting bone.[7]

Peri-implantitis presents with various signs, symptoms, and radiographic changes. Clinically, the signs may include swelling, bleeding on probing, erythema (redness), suppuration or pus discharge, formation of peri-implant pockets, soft-tissue hyperplasia, and in advanced stages, implant mobility. Patients may also experience certain symptoms such as bleeding while brushing, foul taste, halitosis (bad breath), and occasionally pain(rare). Radiographically, it presents with bone loss around the implant that exceeds the normal initial bone remodeling as seen after implant placement. Based on the extent of bone loss relative to the implant length, peri-implantitis can be classified into three categories: mild when bone loss is less than 25% of the implant length, moderate when bone loss ranges from 25% to 50%, and severe when bone loss exceeds 50% of the implant length.[8,9]

Potential Etiologies[1] : The implicit etiologic factors of peri-implantitis include bacterial plaque and corrosive products of implant body(fig-1).

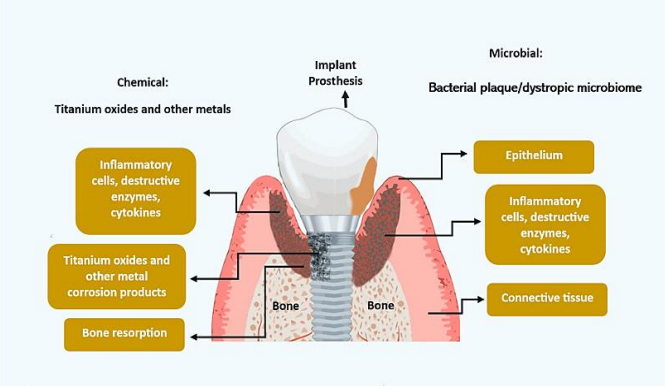

Figure 1: Potential etiologies leading to peri-implantitis[11]

Causes and Risk Factors[3]

Various risk factors related to periimplantitis include:

1. Local Risk Factors

1. **Inadequate Oral Hygiene:** Poor plaque control promotes biofilm accumulation, leading to inflammation and bone loss.
2. **Mechanical Trauma:** Trauma during surgical or prosthetic procedures may initiate inflammation.
3. **Poor Implant Positioning:** Malpositioned implants are delicate to clean and predispose to plaque retention.

2. Systemic Risk Factors

1. **Diabetes Mellitus:** Impaired healing and immune response increases vulnerability
2. **Smoking:** Reduces vascularity and immune function, increasing risk.
3. **Compromised Immunity:** Greater susceptibility to infection.
4. **Soft Tissue Defects:** Lack of adequate keratinized tissue may compromise plaque control or hygiene maintenance.

PROSTHODONTIC CAUSES OF PERI-IMPLANTITIS

1. **Residual Cement:** This is considered as one of the most significant prosthodontic risk factors associated with peri-implantitis. Cement-retained restorations, particularly those with subgingival margins, may leave redundant cement within the peri-implant sulcus(figure-2). Unlike natural teeth, peri-implant tissues lack a periodontal ligament and have reduced vascular supply, making them more susceptible to inflammatory breakdown. Retained cement acts as a nidus for plaque accumulation, triggering a chronic inflammatory response that can lead to progressive bone loss. Deeply placed margins and limited visibility during cementation further increase this risk.

Figure 2: (A) retained cement at the crown margin (B)Excess cement in the peri-implant inflamed tissue, and (C) excess cement around the abutment.[9]

2. **Improper Emergence Profile and Overcontouring:** The contour and emergence profile of implant-supported restorations significantly influence plaque control. Overcontoured crowns, convex subgingival biographies, and bulky prostheses create plaque-retentive niches and restrict access for oral hygiene measures. An improper emergence angle exceeding ideal anatomical limits can increase soft tissue inflammation and compromise peri-implant.

3. **Ill-Fitting Prosthesis and Microgap Formation:** Poor marginal fit between the implant and prosthetic components may create microgaps at the implant–abutment interface. These microgaps can harbor bacteria, leading to colonization and persistent inflammation of surrounding tissues. Micromovements under functional loading may exacerbate microbial leakage and bone resorption.

4. **Occlusal Overload and Unfavourable Force Distribution:** Occlusal overload may act as a contributing factor in the presence of inflammation. Implants lack the shock-absorbing capacity of periodontal ligament fibers, making them less tolerant to excessive or non-axial forces. Parafunctional habits, improper occlusal schemes, cantilevers, and poorly distributed contacts can generate biomechanical stress at the crestal bone, potentially accelerating bone loss when combined with plaque-induced inflammation.

5. **Inadequate Prosthetic Access for Oral Hygiene:** Prostheses that are difficult to clean significantly increase the risk of peri-implant disease. Deep subgingival margins, fixed full-arch prostheses with inadequate embrasure space, and splinted restorations

may hinder effective plaque removal. Patients often struggle to maintain hygiene around bulky frameworks or poorly designed pontic areas.

6. **Implant Positioning Influencing Prosthetic Design:** Improper three-dimensional implant positioning can compromise prosthetic outcomes and peri-implant tissue health. Buccally placed implants may result in thin soft tissue coverage and recession, while deeply placed implants necessitate subgingival margins, increasing the risk of cement retention. Malpositioned implants often require prosthetic compensation, which may lead to overcontouring and compromised hygiene access.
7. **Retention Type – Cement vs Screw:** The choice of retention mechanism has significant implications for peri-implant health. Cement-retained restorations offer improved esthetics and passive fit but carry a higher risk of residual cement. Screw-retained restorations, although technique-sensitive and sometimes associated with esthetic limitations, eliminate the risk of cement-induced inflammation and allow easier retrievability for maintenance and management.

Pathogenesis of Peri-implantitis[8, 10] :

It involves a dynamic interplay between the bacterial colonization of implant surface and dys-regulated host inflammatory responses which further leads to the bone loss and failure of the implant(fig-3).

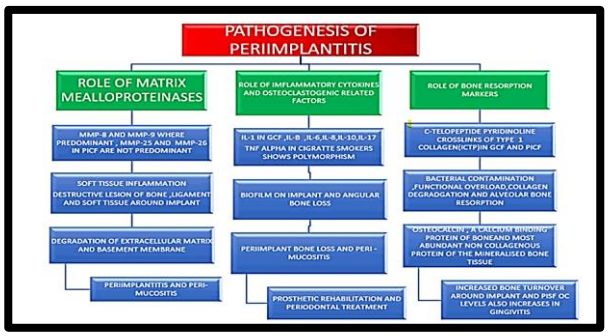

Figure 3: Pathogenesis of Peri-implantitis[8]

Role of Metalloproteinases[8]:

Matrix metalloproteinases contribute to connective tissue degradation and breakdown of peri-implant structures.

Role of Inflammatory Cytokines and Osteoclastogenic Factors[10]:

Elevated cytokines (IL-1β, TNF-α) and RANKL-mediated pathways promote osteoclast activation and bone resorption.

Role of Bone Resorption Markers[8] :

Increased biochemical markers reflect active bone remodeling and progressive destruction.

Management[9,11]

Management of the Peri-implantitis depends on the severity of the disease and patient factors.

1. Early Intervention: Management of Peri-Implant Mucositis: It includes: Non-surgical debridement, drug therapy such as antiseptics and antibiotics, as well as elimination of local prosthetic irritants.

2. Non-Surgical Management of Established Peri-Implantitis: It include prosthesis removal(for accessing) followed by mechanical debridement with adjunctive antimicrobials, surface decontamination using chemicals(citric acid, EDTA, hydrogen peroxide, saline) or lasers.

3. Surgical Management: It include two pathways:

a. Resective Surgery: Pocket elimination, apically positioned flaps, and implantoplasty.

b. Regenerative Surgery: Bone grafts and membranes in contained defects to promote re-osseointegration.

4. Prosthetic Modification and Redesign: Management can also be attained by correcting the overcontoured prosthesis, elimination of excess cement, conversion to screw-retained designs, and occlusal adjustments. These steps may improve the prognosis.

5. Implant Removal: If the condition worsens so implant removal is indicated as there will be advanced bone loss with mobility of implant and persistent infection in the surrounding tissues.

6. Long-Term Supportive Care: Regular monitoring is mandatory for the assessment of probing depths, bleeding, and radiographic bone levels to prevent the recurrence.

Prevention[3]

(a) Preoperative Preventive Measures: Before any surgical interventions, the measures to be followed include:
1. Careful patient selection and risk assessment.
2. Comprehensive treatment planning with prosthetically driven placement.
3. Oral hygiene education.
4. Management of existing periodontal disease.

(b) Postoperative Preventive Measures: After any surgical procedure, the measures that should be followed include:
1. Regular follow-ups (3–6 months based on risk).
2. Reinforcement of hygiene instructions.
3. Professional implant-safe debridement.
4. Periodic clinical and radiographic evaluation.
5. Smoking cessation counselling.
6. Systemic health optimization.

(c) Advancing Preventive Strategies: Other advanced measures that can help in prevention of this condition include:
1. Adherence to updated guidelines and evidence.
2. Use of digital planning and surgical guides.

3. Risk assessment tools.
4. Interdisciplinary collaboration.

PREVENTION OF PERI-IMPLANTITIS: A PROSTHODONTIC PERSPECTIVE

1. Comprehensive Treatment Planning and Implant Positioning

Prosthetically driven implant placement ensures ideal three-dimensional positioning, allowing for appropriate emergence profile, optimal soft tissue support, and favorable load distribution. Adequate buccolingual and apico-coronal positioning reduces the need for prosthetic compensation that may lead to overcontouring or subgingival margins.

2. Prosthesis Design and Emergence Profile Control

The contour of implant-supported restorations should mimic natural tooth anatomy while permitting effective plaque control. Emergence profiles must be designed to allow gentle, cleansable transitions from the implant platform to the crown. Overcontoured restorations and convex subgingival profiles should be avoided, as they create plaque-retentive areas and restrict hygiene access. Open embrasures and cleansable pontic designs are recommended in fixed prostheses to facilitate interdental cleaning.

3. Selection of Retention Method

The choice between screw-retained and cement-retained restorations plays a critical role in prevention. Screw-retained prostheses are generally preferred in situations where maintenance access is crucial, as they eliminate the risk of residual cement and allow retrievability. If cement-retained restorations are indicated for esthetic or angulation reasons, margins should be placed supragingivally whenever possible. Controlled cementation techniques, including extraoral cementation, use of minimal cement, venting channels, and radiopaque cements, significantly reduce the risk of excess cement retention.

4. Occlusal Considerations

Occlusal overload may exacerbate peri-implant bone loss when combined with inflammatory conditions. Prosthetic occlusion should be carefully adjusted to ensure axial loading, light centric contacts, and minimal lateral interferences. Cantilevers should be minimized, and occlusal schemes must be tailored to distribute forces evenly across multiple implants where possible. In patients with parafunctional habits, the use of occlusal splints may be indicated to protect implant restorations.

5. Maintenance Protocols and Supportive Therapy

Long-term success of implant therapy depends heavily on structured maintenance programs. Patients should be enrolled in individualized recall schedules based on risk assessment, typically every 3–6 months. Professional debridement using implant-safe instruments, reinforcement of oral hygiene instructions, and periodic radiographic evaluation are essential components of preventive care. Early detection of mucositis allows intervention before progression to peri-implantitis.

6. Patient Education and Risk Factor Modification

Patient compliance is fundamental to prevention. Detailed instruction on oral hygiene techniques, including the use of interdental brushes, water irrigators, and floss specifically designed for implants, should be provided. Smoking cessation counseling and glycemic control in diabetic patients further contribute to risk reduction. A collaborative approach between clinician and patient enhances long-term peri-implant health.

Discussion[9-14]

Peri-implantitis remains one of the most challenging biological complication associated with implant therapy. Although microbial biofilm is recognized as the primary etiological factor, this review clearly demonstrates that prosthodontic variables substantially influence both the initiation and progression of peri-implant disease. Peri-implant tissue stability is closely linked to decisions made during the surgical–prosthetic transition and definitive restorative phase. Repeated abutment disconnection and reconnection disrupts the peri-implant soft tissue seal, leading to crestal bone remodeling. Therefore, a one-time definitive abutment placement—either at implant insertion or second-stage surgery—appears biologically advantageous, particularly in deeply placed implants. Minimizing manipulation at the implant–abutment interface reduces hemidesmosomal disruption, soft tissue trauma, and contamination risk. Reusing healing abutments may also harbor the residual debris, reinforcing the need for strict decontamination protocols. Early wound stability, prevention of saliva ingress, and controlled plaque accumulation are critical during the immediate post-surgical period to prevent early inflammatory changes.

The restorative and laboratory phases introduce additional risks. Contamination from impression materials, provisional resins, and bonding agents may compromise osseointegration and promote bacterial colonization, particularly on rough implant surfaces. Titanium and zirconia abutments exhibit superior biocompatibility compared to cast or non-implant-grade alloys, while precision of fit is critical to prevent screw loosening and microbial leakage. The use of generic or inaccurately milled components may negatively affect long-term stability.

Retention strategy further impacts peri-implant health. Screw-retained restorations eliminate the risk of subgingival cement extrusion but demand passive fit to prevent mechanical stress. Cement-retained restorations, especially with subgingival margins, are strongly associated with excess cement retention and inflammation. Prosthesis design must prioritize cleansability, as over-contouring and inaccessible embrasures favor plaque accumulation. Occlusal overload, although debated, may contribute to mechanical instability and tissue breakdown.

Management requires a multifactorial approach combining debridement(fig-4), antimicrobial therapy, surgical intervention(fig-5) when necessary, and correction of prosthetic deficiencies. However, heterogeneity in diagnostic criteria and treatment protocols limits current evidence, and long-term trials evaluating prosthodontic modifications are scarce. Overall, the review underscores that peri-implantitis is a multifactorial condition in which meticulous prosthetic planning, material selection, and maintenance-oriented design are fundamental for long-term implant success.

Figure 4: Mechanical treatments of peri-implantitis: (A) plastic curettes, (B) air abrasive, and (C) metallic brush[9]

Figure 5: (a) Soft-tissue dehiscence at the implant site with exposure (b) Surgical procedure: coronally advanced flap with connective tissue graft (c) The flap was advanced and sutured coronally[14]

CONCLUSION

Peri-implantitis represents a significant threat to the long-term success of implant therapy, with multifactorial etiology in which prosthodontic variables play a decisive role. While microbial biofilm remains the primary initiating factor, improper prosthetic design, residual cement, unfavorable emergence profiles, occlusal overload, and compromised maintenance access substantially increase the risk of peri-implant tissue breakdown.

A prosthetically driven approach—emphasizing ideal implant positioning, cleansable restoration contours, appropriate retention selection, and controlled cementation protocols—is fundamental to prevention. Early diagnosis and timely intervention, coupled with correction of prosthetic contributing factors, are critical for successful management. Long-term stability further depends on structured maintenance programs and patient compliance.

Ultimately, peri-implant health is not determined solely at the time of surgical placement but is profoundly influenced by prosthodontic planning and execution. An interdisciplinary, prevention-oriented strategy should be implicated to enhance the implant longevity and minimize biological complications.

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