Review Article

EVALUATION PERI-IMPLANTITIS AND ITS PREVENTION MEASURES, REVIEW ARTICLES

Mohammed Mady[1], Abdulhameed Saeed Alghamdi[2], Nagham Talal Maddah[2], Rowaa Faisal Aljehani[2], Dana Nabil Khojah[2], Bayan Sami Jambi[2], Anas Saleh Siragalddin[3], Khadijah Hilal Bamkhayer[4], Ashwaq Mohmmad Alashjai[5], Rasha Mohammed ALaradi[6], Mohammad Fahd Alsobeie[7], Ala'a Atef Siam[8]*

[1]*Faculty of Dentistry Medicine, King Khalid Hospital, Al Kharj, Saudi Arabia.*
[2]*Faculty of Dentistry, Batterjee Medical College, Jeddah, Saudi Arabia. Abdulhameed-17@hotmail.com*
[3]*Faculty of Dentistry, International Medical Center, Jeddah, Saudi Arabia.*
[4]*Faculty of Dentistry, ALBndar Health Center, Yanbu, Saudi Arabia.*
[5]*Faculty of Dentistry, King Abdullah bin Abdulaziz University Hospital, Riyadh, Saudi Arabia.*
[6]*Faculty of Dentistry, Al-Ahli General Medical Care Complex, Alulah, Saudi Arabia.*
[7]*Faculty of Dentistry, Alsalimy Hospital, Al-Sulaimi, Saudi Arabia.*
[8]*Faculty of Dentistry, Tanta University, Tanta, Egypt.*

ABSTRACT

Peri-implant inflammations represent serious diseases after dental implant treatment, which affect both the surrounding hard and soft tissue. Due to prevalence rates up to 56%, peri-implantitis can lead to the loss of the implant without multilateral prevention and therapy concepts. The Medline, Pubmed, Embase, NCBI, and Cochrane databases were searched for studies of patients with non-alcoholic fatty liver disease. Incidence, etiology, and management options were analyzed. Peri-implant inflammations, potentially leading to implant loss at a 56% prevalence rate, require preventive measures like continuous check-ups, addressing risk factors, and choosing appropriate treatments. Conservative methods are effective for early stages, while advanced cases benefit from surgical interventions following the Cumulative Interceptive Supportive Therapy (CIST) protocol, providing essential guidance for practitioners.

Key words: Peri-implantitis, Peri-implant disease, Periodontal disease, Mucositis.

Introduction

The utilization of dental implants for oral rehabilitation has demonstrated positive outcomes in both short-term and long-term studies over the past few decades. Dental implants serve as a dependable therapeutic option for various restoration and replacement scenarios, such as bridge abutments, crown support, and the attachment of removable dentures. Despite their effectiveness, complications, both short-term and long-term, may arise from this practice [1]. Prosthetic replacements like titanium implants, when introduced into the oral cavity, differ significantly from natural teeth in their connection to supporting alveolar bone and connective tissues. Implants have parallel-running supracrestal connective tissue fibers, unlike the perpendicular fibers of the periodontal ligament that surround natural teeth. Whether this distinction makes implants more susceptible to infections compared to natural teeth remains uncertain and requires further investigation. In oral implantology, osseointegrated implants are biocompatible titanium rods surgically implanted directly into alveolar bone without an interposed layer of soft tissue [2]. Prosthetic fixtures such as crowns and abutments are then attached to these implants, forming a strong bond between the bone and the implant, a process known as osseointegration. Infected implants harbor bacterial species similar to those around healthy teeth, necessitating tailored treatments based on specific infections due to varying microbial complexes among patients. Post-operative infections, though rare, can occur within the first month after implant placement, with prevalence rates ranging from 1.6% to 11.5%. Implant failure, marked by quantitative measurements falling below survival criteria, may necessitate implant removal [3]. Primary implant stability is crucial for successful osseointegration, and the absence of pain under forces is a key subjective criterion for implant survival. Local bone density in the patient is also a significant determinant, impacting the stability required for successful implantation [4]. Despite the role of occlusal factors in implant health, peri-implant mucositis and peri-implantitis are primarily bacterial diseases, sharing clinical similarities with periodontal diseases. Patients with a history of periodontitis face an increased risk of peri-implantitis, as shown in various studies and systematic reviews [5]. While dental implants have proven to be a valuable solution, ongoing research is essential to better understand their clinical and histomorphometric definitions, ensuring successful implantation and minimizing complications. The formation of a biofilm on an implant follows a bacterial colonization pattern similar to that observed on natural teeth, as described by Kilian *et al*. in 2016. Initially, aerobic and facultative anaerobic gram-positive cocci and rods are the primary colonizers. Specific species within this group, such as Streptococci, have the ability to adhere to the hard surfaces of teeth, implants, or crown restoration materials. Fusobacterium nucleatum, a significant secondary colonizer,

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plays a crucial role by interacting and communicating with other bacteria, facilitating the continuous growth of the biofilm with late colonizers. The mature biofilm consists of a complex matrix of bacteria, polysaccharides, and proteins, forming a biological system regulated by bacterial interactions, including quorum sensing [6]. Within the biofilm, bacterial products such as toxins, protein, or lipopolysaccharide antigens can interact with epithelial cells and neutrophil granulocytes that migrate through the gingival/peri-implant mucosal sulcus. If the biofilm is not removed from the implant or crown restoration, microorganisms and their products within the biofilm will trigger a host response in the mucosal connective tissue alongside the sulcular and junctional epithelium around the implant [6].

Peri-implantitis

In peri-implantitis, typical signs of inflammation include bleeding or suppuration during probing, increased probing depth, and evidence of bone loss on X-rays [7]. Comparative studies have meticulously analyzed the microbial compositions of healthy and diseased peri-implant sites, revealing significant changes in the submucosal microbiome and dysbiosis with increased pocket depth. Although peri-implantitis and periodontitis share clinical similarities, their onset and progression patterns differ significantly [8]. Peri-implantitis can manifest early and progress nonlinearly, often accelerating faster than periodontitis around natural teeth. Histopathological analysis highlights crucial differences between peri-implantitis and periodontitis. In periodontitis around teeth, the inflammatory infiltrate resides in connective tissue lateral to the pocket epithelium, separated from the crestal bone by periodontal ligament fibers [9]. Peri-implantitis, however, presents distinct features: the pocket compartment contains substantial plaque, but the pocket epithelium doesn't fully cover the mucosal dimension. Consequently, the apical third of the pocket exhibits exposed, inflamed tissue facing a significant microbial presence on the implant surface. Unlike natural teeth, implants lack a periodontal ligament, allowing the inflammatory infiltrate to extend directly to the crestal bone. Experimental studies show numerous osteoclasts in peri-implantitis sites, indicating active bone resorption [10]. The absence of root cementum, periodontal ligament, and supra-crystal attachment fibers in implants contributes to these differences. Disease progression in peri-implantitis reflects complex inflammatory events driven by continuous exposure to microorganisms and their products. This exposure triggers ongoing inflammatory mechanisms, including the production of pro-inflammatory elements due to cell membrane degradation. Additionally, osteoclast activation leads to bone resorption and the separation of crestal bone from infiltrated connective tissue, acting as a protective response. Understanding these distinct characteristics is crucial for devising effective treatment and prevention strategies for peri-implantitis [11].

Preventions

Long-term results from surgical treatments of peri-implantitis are encouraging, but challenges still persist. Studies indicate that implant surface characteristics significantly impact outcomes, with non-modified surfaces showing better results than modified ones [12]. Despite extensive clinical research, no chemical agent has proven superior to saline in decontaminating implant surfaces. Given the technical complexities and substantial resources required for peri-implantitis treatment, there is a growing emphasis on preventive strategies. Preventing peri-implantitis is crucial, involving the prevention of peri-implant mucositis and the treatment of existing cases to prevent their progression. The prevention of peri-implant diseases encompasses thorough patient education in self-performed oral hygiene practices around implants [13]. A personalized follow-up plan, tailored to individual patient needs and risk factors, is essential. Patients with a history of severe periodontitis, poor plaque control, and those lacking regular maintenance care after implant therapy face an increased risk of developing peri-implantitis. While smoking and diabetes are suspected risk factors, current evidence on their association with peri-implantitis remains inconclusive [14]. Regular recall visits are essential, involving clinical examinations, radiological assessments, and probing of peri-implant tissues to detect signs like bleeding on probing and monitor changes in probing depth and mucosal margin position. Clinicians should establish baseline radiographic and probing measurements after completing implant-supported therapy. In the context of a film project outlining the storyline, the overview emphasized the key aspects of peri-implant health, biofilm formation, host responses leading to peri-implant mucositis, and, subsequently, peri-implantitis. While treatment approaches for peri-implantitis were briefly discussed, the narrative underscored the importance of prioritizing prevention in implant dentistry. The discussion also highlighted the 2017 World Workshop on Classification of Periodontal and Peri-implant Diseases and Conditions, which introduced new disease and case definitions for peri-implant health, peri-implant mucositis, and peri-implantitis [15].

Treatment

Implant failure can occur due to untreated infections, which are the most common cause of complications during the initial healing period [16]. Signs such as suppuration, fistulas, swelling, and early or late mucosal dehiscence indicate potential implant failure. If these symptoms manifest early in the healing process, they indicate a more serious issue, disrupting the primary bone healing process and jeopardizing successful implant integration. Redness and swelling in the surrounding tissue may or may not be present, and pain is not a consistent symptom [17]. The term "Peri-implant mucositis" is used to describe the infection as long as it has not resulted in significant bone loss beyond what can be attributed to natural remodeling. This condition is comparable to gingivitis in natural teeth. Peri-implantitis, on the other hand, involves well-demarcated alveolar bone loss around the implant [17]. It is possible for bone

destruction to occur without the implant showing signs of mobility until complete osseointegration failure, as the bottom of the implant may remain healthy. In case of infection onset, it is recommended to prescribe antibiotics for seven days, such as amoxicillin with potassium clavulanate or clindamycin. Local and systemic antibiotics help reduce anaerobic bacteria counts, including certain periodontal pathogens, leading to improvement [18]. Chlorhexidine digluconate mouthrinses are also advised. If these treatments fail to control the infection, a different antimicrobial agent should be prescribed, followed by removal of the failed implant if there is mobility and/or advanced bone loss. Notably, peri-implantitis induced by functional overloading and peri-implantitis due to infection have distinct bacterial profiles, as identified through direct phase-contrast microscopy and culturing [17].

Results and Discussion

The initial two years following implant placement are crucial in determining its success. The overall condition of the site where the implant is located greatly influences the nature and consequences of any infection that may occur. Furthermore, the impact of implant failure, as well as its effect on the supported crown, bridge, or denture, is more significant than natural tooth loss, as it leads to rapid peri-implant bone loss. In severe cases of implant failure, hospitalization may be necessary. Various factors are suspected to increase the risk of implant failure, including mechanical and anatomical aspects like improper implant positioning or insufficient alveolar bone height and density. Insufficient bone quality and quantity are particularly likely to contribute to early implant failure because successful bone healing demands substantial biological effort from skeletal tissues. Adequate bone quality is essential for preserving the alveolar bone around oral implants, ensuring a high success rate. Additionally, smoking adversely affects proper wound healing and is likely to compromise the success of bone grafts and dental implants. Smokers, regardless of whether they undergo bone grafts, have shown higher rates of implant failure and increased post-operative complications.

Conclusion

Understanding peri-implantitis is crucial for both dental practitioners and patients. This inflammatory condition around dental implants, characterized by symptoms like bleeding during probing, increased pocket depth, and bone loss, demands careful monitoring. While it shares similarities with periodontitis, peri-implantitis exhibits distinct features, including a non-linear, accelerating progression and unique histopathological differences influenced by the absence of periodontal ligament and other natural tooth components. Microbial dysbiosis and inadequate oral hygiene contribute to its onset, emphasizing the importance of preventative strategies. Recognizing risk factors, such as smoking and poor bone quality, is essential, as they significantly impact treatment outcomes. The initial two years of post-implant placement are pivotal, requiring diligent evaluation to ensure success. To address peri-implantitis effectively, tailored preventive measures, regular follow-ups, and awareness of individual risk factors are essential in safeguarding the long-term health of dental implants and overall oral well-being.

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