Invited Review

Peri-implantitis: Periodontal and restorative implications

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ABSTRACT

Dental implants are being increasingly used in dentistry. Millions of implants are being placed on a yearly basis around the world by dental surgeons. These implants support restorations ranging from a single crown to a denture. There are significant costs associated with implant supported restorations. The advent of peri-implant diseases threatens the survival and maintenance of implant supported restorations leading to significant impact on the sustainability of such restorations while affecting the patient's functionality. This review article discusses the current concepts, as well as the etiopathological and treatment considerations related to peri-implant diseases.

CLINICAL RELEVANCE TO INTERDISCIPLINARY DENTISTRY

Peri-implant disease affects implants, the peri-implant tissues and implant supported restorations. This article is relevant to this journal as peri-implant disease and its management is detrimental to the practice of the various disciplines of dentistry including periodontics, prosthodontics, implant dentistry and restorative dentistry

Key words: Implants, peri-implant mucositis, peri-implantitis

INTRODUCTION

Dental implants have become a critical necessity for replacement of lost natural teeth in modern times. Made of inert biocompatible metals, dental implants integrate with surrounding bone and provide support for a restoration such as a crown, a bridge or a denture. Today, approximately 500,000 implants are being placed on an annual basis with a 95% success rate.[1] However, implant success is only defined as a short-term effect that is, whether the implant has osseointegrated or not and whether it is able to support a restoration following its placement. There are no proper guidelines that define implant success or maintenance over a long-term, except for implant survival. This together with the transient changes in host factors has given rise to the advent of peri-implant disease.

The term "peri-implantitis" was first introduced to describe an inflammatory process caused by microorganisms affecting the supporting tissues around an osseointegrated implant in function, resulting in a loss of alveolar bone.[2] Peri-implant diseases include peri-implant mucositis and peri-implantitis. Peri-implant mucositis is similar to gingivitis and is defined by the presence of an erythematous peri-implant mucosa, bleeding on probing and a pocket with a probing depth of < 4 mm. On the other hand, peri-implantitis is synonymous to periodontitis and is defined by the presence of bleeding on suppuration, a pocket with a probing depth of > 4 mm, and a saucer-shaped bone loss which can be observed on a radiograph or up on opening a flap [Figure 1].[3-6] The prevalence of peri-implant mucositis is 80%, and that of peri-implantitis is 28–56% in patients with dental implants.[7] The progressive peri-implant disease may lead to the mobility of the affected implants and failure of the restorations they support. This is a significant concern in patients with implant supported dentures, considering the amount of effort, and costs associated with such restorations [Figure 2].

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ETIOPATHOGENESIS

Anatomic and histological factors

There are several anatomic and histological differences between the supporting structures of a tooth as compared to an implant. The gingiva around the tooth has fibers that run perpendicular and attach to the tooth surface and the alveolar bone. In the peri-implant mucosa, however, the fibers are parallel and have no attachment to the implant surface. The tooth root is immediately surrounded by a periodontal ligament that connects it to the alveolar housing. The presence of the periodontal ligament provides support, proprioception, blood supply, and an unending supply of stem cells for regeneration upon injury or insult. However, the periodontal ligament is absent in a dental implant. Such differences in the supporting structures may have critical implications in the etiopathogenesis, as well as management of peri-implant diseases.

Risk factors

The etiological factors including poor oral hygiene, smoking, history of periodontitis, postmenopausal changes, diabetes mellitus, genetic traits, excessive alcohol consumption, mechanical overload, and the type of implant surface may contribute to the development of peri-implantitis.[8-12] Of all, the most important factors leading to peri-implantitis with bone loss appears to be an inflammatory process due to the duration of biofilm accumulation and smoking dose.[13]

Microbiological and immunological factors

The peri-implant infections present a microbiota very similar to that found in the periodontal disease. Subgingival biofilms in peri-implantitis have been found to exhibit greater bacterial diversity with the presence of *Porphyromonas gingivalis*, *Prevotella intermedia*, *Prevotella nigrescens*, and *Aggregatibacter actinomycetemcomitans*.[12] Apart from the above bacteria other species such as *Staphylococcus aureus*, enteric bacilli, and *Candida albicans* have also been detected. These microorganisms secrete higher levels of both lactoferrin and elastase which result in site-specific inflammatory changes in the soft tissues surrounding oral implants, and it may lead to their progressive destruction (peri-implantitis) and ultimately to implant failure. The apical extension of the inflammatory cell infiltrate in peri-implantitis lesions is more prominent and in most cases located further apical of the pocket epithelium and comprised a greater proportion of neutrophils and macrophages when compared with periodontitis lesions.[10,12,14,15] Lymphocytes and plasma cells are pronounced in both types of lesions.[14] Furthermore, fibroblasts in peri-implantitis lesions exhibited a discrete cytokine profile that may contribute to matrix breakdown.[16] These features of peri-implantitis imply a more acute inflammatory response when compared with periodontitis.

Reducing the inflammatory response may be an effective way to prevent the tissue destruction caused by the peri-implant disease. It has been recognized that during the inflammatory process, inflammatory mediators such as prostaglandin E2 and other arachidonic acid metabolites are responsible for bone resorption and periodontal disease.[17] New research has been focused on the development of daily topical administration forms such as gels, toothpastes, and rinses which contains nonsteroidal anti-inflammatory drugs (NSAIDs) and also opening the chance to manage failing implants with topical NSAID delivery systems.[17]

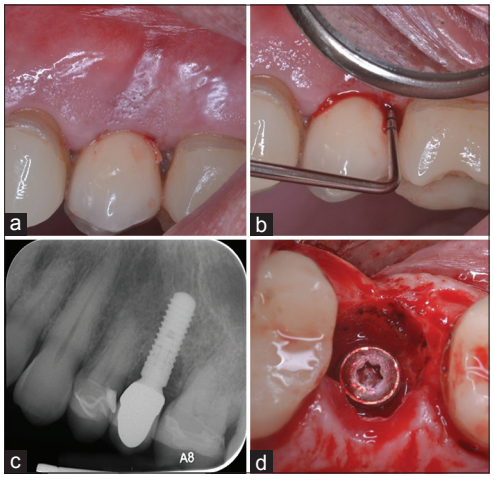

Figure 1: Diagnosis of peri-implantitis. (a) Bleeding on probing the peri-implant sulcus. (b) Probing depth of >4 mm. (c) Saucer shaped radiographic bone loss. (d) Osseous defect surrounding the implant upon flap opening

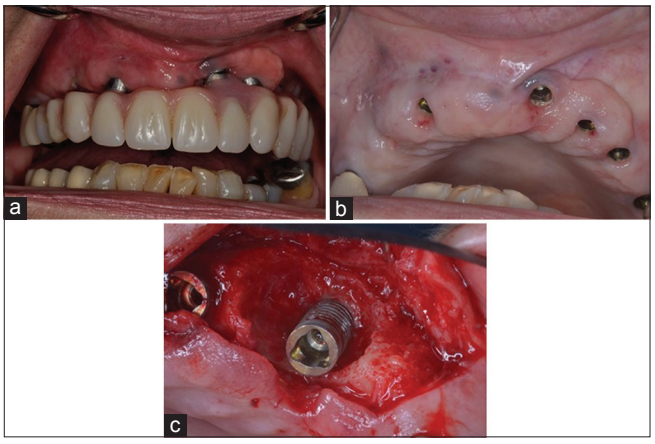

Figure 2: Peri-implant disease affecting implants supporting an upper complete denture. (a) Upper denture supported by implants. (b) Multiple implants with erythematous peri-implant mucosae. (c) Severe bone loss around one of the implants

TREATMENT

Nonsurgical therapy

Nonsurgical treatment for peri-implantitis includes motivation, oral hygiene instructions and scaling with a hand plastic instrument. This alone has very limited effect on the clinical signs of peri-implantitis. Hence, other treatment options including submucosal glycine powder air polishing and locally delivered antibiotics minocycline microspheres or doxycycline hyclate as an adjunct to submucosal debridement were developed. These methods may result in greater reduction in bleeding on probing and probing depths compared with submucosal debridement with adjunctive submucosal irrigation using chlorhexidine digluconate alone.[12] Surgical therapy is ideal for lesions that do not resolve following nonsurgical therapy.

Surgical therapy

The existing guidelines for surgical therapy around implants follow the principles of periodontal therapy. A flap is raised for access and visibility. Mechanical debridement is done to eliminate the biofilm. The implant threads can be eliminated or alternatively the implant surface is treated by acidic reagents like citric acid or by local application of antibiotics like tetracycline or doxycycline. Osseous respective surgery can be done to eliminate bony ledges. In case of deep osseous craters and defects, the principles of guided bone regeneration can be applied by using bone substitutes with membrane barriers.[18] Past studies have shown that following the application of nanocrystalline hydroxyapatite or a bovine-derived xenograft in combination with a collagen membrane, at intrabony peri-implantitis defects, resulted in significant probing depth reductions, and clinical attachment gains.[19]

Lasers

Lasers are being increasingly used in periodontal therapy. The combinations of toluidine blue O (100 μg/ml) and irradiation using a diode soft laser with a wavelength of 905 nm resulted in the reduction of bacterial population of *P. gingivalis*, *P. intermedia*, and *A. actinomycetemcomitans* on different implant surfaces (machined, plasma-flame-sprayed, etched, hydroxyapatite-coated).[20] The treatment of peri-implantitis by regenerative osseous surgery in conjunction with erbium, chromium-doped: yttrium, scandium, gallium, and garnet laser around an implant was found to be successful without any complications.[21] Furthermore, diode laser light decontamination (1-W output, a maximum of 20 s) is one of the approved treatment options for peri-implantitis.[21]

CONCLUSIONS

The peri-implant disease is biofilm based and relies on the host inflammatory response for its manifestation. Thus, it is similar in its etiopathogenesis to periodontal disease. However, the inflammatory response in peri-implant disease is acute leading to a wide area of tissue destruction within a short period of time. The guidelines for treating peri-implant disease follow periodontal therapy currently. However, there is a need for more studies in the near future to develop specific protocols for successfully treating peri-implant diseases. Implant maintenance with regular periodic recalls is critical for preventing peri-implant disease and for ensuring the long-term success of implant-supported restorations.

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