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PERIIMPLANTITIS

Periodontology

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ABSTRACT

The fundamental work of Branemark and associates in 1960s demonstrated that commercially, pure titanium implants can be anchored to the jaw bone and used successfully for tooth replacement. Osseointegration is the direct structural and functional connection between ordered, living bone and surface of a load carrying implant. Periimplantitis is a clinical manifestation where clinically and radiographically evident loss of bone support for the implant occurs, together with an inflammatory reaction of the periimplant mucosa. This manifests clinically as reddening of periimplant mucosa and bleeding on probing; accompanied by increased periimplant pocket depth and progressive mobility of the implant in severe cases. The major etiological factors attributed for such a failure include bacterial infection and excessive mechanical stress. Thus, this review mainly addresses the aetiopathogenesis of periimplantitis , its clinical features and various methods to manage the same.

KEYWORDS

Periimplantitis, Osseointegration, Mucositis.

INTRODUCTION

Developing artificial replacements for missing teeth has been an elusive goal for more than 1500 years. The fundamental work of Branemark and associates in 1960s demonstrated that, commercially pure titanium implants can be anchored to the jaw bone and used successfully for tooth replacement[1]. They described a relationship between titanium and bone as **osseointegration,** which is the direct structural and functional connection between ordered, living bone and the surface of a load carrying implant[2]. The name **periimplant disease** refers to the pathological inflammatory changes that take place in the tissue surrounding a load bearing implant. **Mucositis** is a clinical manifestation characterised by the appearance of inflammatory changes restricted to the periimplant mucosa.

Periimplantitis is a clinical manifestation where clinically and radiographically evident loss of the bony support occurs, together with an inflammatory reaction of the periimplant mucosa[3]. Its clinical features include reddening of periimplant mucosa and bleeding on probing; in advanced cases, accompanied by an increased periimplant pocket depth and progressive mobility of the implant. This inflammatory disease differs from periodontitis in that, there is the absence of periodontal ligament. Instead, breakdown of the perimplant soft tissue cuff occurs, progressing to the localised osteitis and a destructive process that is similar to periodontitis, both clinically and radiographically. The progression of periimplantitis starts from the coronal portion of the implant surface and extends towards the apical portion leading to the failure of the osseointegration and periimplant bone loss, resulting in mobility of the implant and a complete implant failure[4].

Etiology :

Two major etiological factors associated with periimplantitis are:

1. Bacterial infections:

The main cause of periimplantitis is dental plaque[5]. The microbiota associated with failing dental implants is similar to those associated with periodontitis. Healthy perimplant tissue and the biologic seal play an important role as a biologic barrier to some of the agents that cause periimplant disease. The microorganisms most commonly related to the failure of an implant are rods and motile forms of gram negative anaerobes, spirochetes. These include: *Prevotella intermedia, Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Bacteroides forsythus, Treponema denticola, Prevotella nigrescens, Peptostreptococcus micros* and *Fusobacterium nucleatum*[6]. These gram negative bacteria produce endotoxin, a heat stable lipopolysaccharide which initiate an acute inflammatory response in addition to bone destruction.

The macro design of the implant also plays an important role in the onset of periimplantitis. The surface roughness favours bacterial plaque adhesion when exposed to oral environment[7]. Poor alignment and ill-fitting prosthetic components foster plaque retention, enabling the microorganisms to pass inside the transepithelial abutment. Implants in the partially edentulous patients are at a greater risk for periimplantitis than those in completely edentulous patients, because, the natural teeth may act as a reservoir for periodontal pathogens from which they may colonize the implants in the same mouth.

2. Excessive mechanical stress:

It is the mechanism by which integration of the implant is fully or partially lost. The process begins with appearance of microfractures of the bone around an osseointegrated implant, as a result of being subjected to the axial or lateral stresses due to excessive load leading to loss of osseointegration around the neck of an implant.

In addition to the presence of periodontal disease producing microbiota, other local, systemic and genetic factors must co-exist for a prolonged and active infection to take place. These include excessive mechanical stress on implant, poor oral hygiene, patients with increased risk of periodontitis, smoking, poorly controlled diabetes, long term treatment with corticosteroids, radiation or chemotherapy[8].

Clinical features and diagnosis:

The clinical features can be broadly divided into two phases, early mucositis and progressing periimplantitis. When the pathologic process restricts itself to the soft tissue surrounding the implant, it is termed as mucositis. When this early mucositis is left untreated, it increases in severity extending the pathology into the underlying bone causing peri-implantitis. The most common signs and symptoms are, increased redness around the implant, bleeding on probing, increased probing depth of the periimplant pockets, suppuration, radiographic implant radiolucency, progressive loss of bone height around the implant, pain on mastication, presence of granulation tissue around the implant and mobility in late peri-implantitis[9].

Probing pocket depth:

The increase in sulcus depth around an implant is related to disease and bone loss[10]. The pocket depth of 3 to 4 mm is commonly seen even in successful implants and should not be considered as pathologic bone loss due to infection.

Care should be taken not to contaminate the implant sulcus with bacteria from other diseased sites in the oral cavity. Although the risk of potential damage to fragile surface makes probing contraindicated in implants, careful monitoring of probing depth and clinical attachment level over a period of time is useful in detecting changes in peri-implant tissues. Plastic pressure controlled probes are preferred to manual probes as longitudinal measurements are essential.

Stability measures:

Stability assessments of implants indicate maintenance of osseointegration. Periotest and resonance frequency analysis have been used as non-invasive ways of evaluating implant stability.

Bacterial identification:

Traditional culture methods can be used to identify colonising microorganisms. DNA probes capable of identifying specific nucleotides of certain bacterial spores can be used to identify

microorganisms that colonize the pocket. Another useful method is BANA(benzoyl-arginenapthylamide) hydrolysis, which shows the presence of enzyme trypsin that is produced by pathogens such as *Bacteroids forsythus, Treponema denticola and Porphyromonas gingivalis*. Increased gingival temperature and peri-implant sulcular fluid volume are helpful in early detection of peri-implantitis.

Radiographic features:

Most commonly used to assess bone loss on the implant crestal and the facial aspect of the implant. Peri-implant radiolucency indicates the presence of surrounding soft tissues and is a sign of implant failure. In case of bone loss due to excessive mechanical force, a fibrous tissue replaces the bone around the implant. Radiographs at baseline and at regular intervals are used to assess bone quality and compare the changes in bone height over a period of time[11]. Paralleling cone technique is more suitable as it has high degree of reproducibility.

Histological features:

Bacterial contamination and an inflammatory infiltrate with epithelial down growth is seen similar to periodontitis lesion[12]. Analysis of the fluid in the peri-implant sulcus reveals increased level of chondroitin sulphate, elastase, beta- glucuronidase, amino transferase and prostaglandin E2.

Treatment:

The treatment modality for an implant is basically governed by the severity of the lesion. While a case of mucositis is treated using non-surgical procedures, a case of peri-implantitis may require surgical intervention not only to arrest the disease process but also to regenerate the lost structure.

Non-surgical treatment:

In case of mucositis where there is no evidence of radiographic bone loss, simple non surgical procedures like, removal of plaque and calculus with the help of scaling and chemical plaque control in form of topical application of 0.12% chlorhexidine are advised[13] .Modifications of prosthesis is necessary, in order to correct design defect that impedes proper oral hygiene and to correct bio mechanical stress factors involved.

If periimplantitis is diagnosed, treatment will depend on the amount of bone loss. If the bone loss is incipient, along with non surgical procedures, chemical decontamination of the prosthetic abutments is done and antibiotics are prescribed[14].

Surgical treatment:

Severe or persistent bone loss necessitates surgical intervention in the form of surgical debridement of soft peri-implant tissue, decontamination of microimplant surface and bone regeneration techniques which are aimed at regenerating lost bone[15]. Presence of moderate horizontal bone loss indicates apically repositioned flap and resection techniques, in order to reduce the pocket depth and to ensure better oral hygiene maintenance. Also smoothening and polishing rough surfaces and even eliminating threads on the implant are necessary to prevent plaque adhesion. Severe bone loss and prosthesis in aesthetically compromised sites indicate guided tissue regeneration techniques which aim to regenerate the lost bone. However it is important to decontaminate the implant surface to enable bone regeneration and permit osseointegration prior to this.

CONCLUSION

Periimplantitis surrounding oral implants is an inflammatory process affecting the hard and the soft tissues resulting in rapid loss of supporting bone. A microbial colonization of the dental implants and the infection of the peri-implant tissues results in bone destruction and implant failure. The microbiota found at the sites of periimplantitis is very similar to the microbiota of advanced periodontitis. Various therapies have been advocated for the resolution of peri-implant infection and restoration of periimplant tissues. Regardless of the prevalence of peri-implantitis represents a threat to the longevity of the associated prosthetic replacement. Therefore detection and treatment of early pathogenic changes during regular recall maintenance visits prevents periimplant soft tissue inflammation and progressive bone loss.

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