PERI-IMPLANTITIS VERSUS PERIODONTITIS: AN IMPLIED METAPHOR

Review Article

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ABSTRACT

Hard and soft tissues around an osseointegrated dental implant have some similarities with the periodontium of natural teeth. The major difference occurs in collagen fibers, which are parallel to the dental implant surface compared with insertion on the natural teeth, which is perpendicular and functional between bone and cementum. As untreated periodontitis can ultimately lead to loss of natural teeth, peri-implantitis can result in loss of dental implants. Recent studies show that the main causative factor of both tooth loss from Periodontitis and loss of dental implants due to Peri-implantitis is the microbial dental plaque. Another disease that resembles peri-implantitis is mucositis; affecting only the soft tissue component, the occurrence of inflammation at this level is due to plaque accumulation. Peri-implantitis can be considered analogous to periodontitis.

KEYWORDS: Periodontitis, Mucositis, Peri-implantitis
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INTRODUCTION:

Soft and hard tissues surrounding a dental implant have certain similarities with those of natural dentition. The major difference appears in collagen fibers, which are unattached and parallel to the dental implant surface, compared to the insertion of the natural teeth, which is straight and functional, between bone and cementum.[1]

The epithelial junction sealing achieved in the gingival sulcus of natural teeth offers protection against bacterial penetration in the mouth. If this seal is broken or gingival epithelial apical fibers are damaged or destroyed, the epithelium migrates rapidly in apical direction, and resulting in periodontal pockets.[2]

Since there is no cementum or insertion of epithelial fibers in the case of a dental implant, mucosal sealing is extremely important. If the seal is lost, the bacterial infiltration rapidly expands to bone structures.[1]

The periodontal parameters' values (depth appreciation of the space around the implant, clinical gingival insertion levels, gingival bleeding on examination with the probing, mobility) are important indicators for detecting potential implant pathological conditions.[3]

PERIODONTITIS:

Chronic periodontitis is defined as infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss.

The etiology of periodontitis has long been unspecified, however etiological factors are classified in: local, systemic, environmental and genetic factors.[2]

The initial lesion in the development of periodontitis is the inflammation of the gingiva in response to a bacterial challenge. Pocket formation starts as an inflammatory change in the connective tissue wall of the gingival sulcus. The cellular and fluid inflammatory exudate causes degeneration of the surrounding connective tissue, including the gingival fibers. Just apical to the junctional epithelium, collagen fibers are destroyed and the area is occupied by inflammatory cells and edema.

Clinical features in periodontitis[4,2]:

Characteristic clinical findings in patients with untreated chronic periodontitis may include

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- supragingival and subgingival plaque accumulation (frequently associated with calculus formation),
- gingival inflammation,
- pocket formation,
- loss of clinical attachment,
- loss of alveolar bone,
- and occasional suppuration.

In patients with poor oral hygiene, the gingiva typically may be slightly to moderately swollen and exhibit alterations in color ranging from pale red to magenta. Loss of gingival stippling and changes in the surface topography may include blunted or rolled gingival margins and flattened or cratered papillae.

PERI IMPLANTITIS:

Peri-implantitis describes pathological changes that occur in soft and hard tissues surrounding the implant. It implies bone damage, in addition to soft tissue damage[7], and it is caused by bacterial factors, in combination with other predisposing factors.[3] Peri implant mucositis is a disease affecting only the soft tissue component due to plaque accumulation.[6]

Tissue integration of oral implants consists of two main components[7]:

A. Bone integration[6]

B. Peri-implant soft tissue integration[8]

Clinical features of periimplantitis:
- Bleeding and suppuration on probing
- Swelling of the peri implant tissues
- Pain
- Vertical bone destruction associated with the formation a peri implant pocket.
- Radiological evidence for vertical destruction of the crestal bone.

Criteria for successful dental implant treatment[3]:
- Favorable underlying bone without any inflammatory processes
- Primary stability of the implant after placement
- Osseointegration
- Obtaining a superior aesthetic result
- Optimal morphological and functional integration of the implant

Main reasons of peri-implantitis occurrence are[6]:
- The patient did not maintain a good oral hygiene, hence failed osseointegration.
- Abutment was not installed correctly;
- Unadapted prosthesis in relationship to the gingiva in the neck of the implant.

Implant failures due to infection are characterized by a peri-implant bacterial complex resembling that of adult periodontitis. In edentulous subjects, Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis are not as frequently associated with peri-implant infection as in partially edentulous cases.[9]

The bone around a dental implant has a higher thickness and density than that which surrounds natural teeth.

Histological studies have shown that fiber orientation around the implant has the appearance of slings.[2] Although bone-implant interface can be seen, most tissue consists of collagen fibers.[1] Peri implant tissue has the same functions as the periodontal ligament, but structurally it is different. In the Peri-implant fibrous tissue, collagen fibers have a distinctive orientation and a specific interaction with the bone surrounding the implant in direct interrelation with the implant design and loading.[7] These fibers are oriented in the three dimensional space between implant and bone, following the distribution of biomechanical forces.[2]

The bundles of collagen fibres in the periimplant tissue are longer than those of the periodontal ligament, passing from the bone trabeculae to the implant surface. The length of these collagen bundles is the key point for the stability and longevity of the implant.[3]

Peri-implant tissue functions:
1. Piezoelectric effect: It is assumed that occlusal forces are transmitted to the peri-implant ligament,

stimulating the bone where the fibers are inserted. Studies have shown that deformation of the surface of the implant socket, immediately around the implant causes compression, generating a negative charge, while the distal part of the trabeculae is in tension, causing a release that generates a positive charge. The cells help post-traumatic scarring, removes debris and form a protein network, which will calcify later.[1]

2. The hydraulic effect: Peri-implant ligament is bathed in fluids in the implant socket. Axial occlusal forces are transmitted to collagen fibers, which in turn act on the fluid that is incompressible and are pushed in the bone, to the bone marrow. Blood vessels create a hydraulic effect appearing also in the natural tooth. When the action ceases, the fluid returns, then the process restarts.[1]

3. The buffer effect: Because collagen fibers can be deformed, a buffer effect appears between implant and bone.[1]

TREATMENT OF PERIODONTITIS

The goal of periodontal treatment is to maintain the health of the teeth and dental implants, to ensure comfort, functionality and aesthetics. With the increasing number of patients receiving dental implants the prevalence of inflammatory problems of the implant receiving tissues is also increasing.[10]

The treatment stages of Periodontitis[2]:

1. Preliminary phase:
- Treatment of emergencies: Dental or periapical
  Periodontal
  Other

Extraction of hopeless teeth and provisional replacement if needed (may be postponed to a more convenient time)

2. Phase I periodontal therapy or Nonsurgical periodontal therapy:
- Complete removal of calculus.
- Correction or replacement of poorly fitting restorations and prosthetic devices
- Restoration or temporization of carious lesions
- Orthodontic tooth movement
- Treatment of food impaction areas
- Treatment of occlusal trauma
- Extraction of hopeless teeth
- Possible use of antimicrobial agents including necessary plaque sampling and sensitivity testing.

3. Phase II Surgical periodontal therapy
- Periodontal therapy, including placement of implants
- Endodontic therapy

4. Phase III Restorative phase
- Final restorations
- Fixed and removable prosthodontic appliances
- Evaluation of response to restorative procedures
- Periodontal examination

4. Phase IV Maintenance phase
- Periodic rechecking: Plaque and calculus

Gingival condition (pockets, inflammation) Occlusion, tooth mobility.

TREATMENT OF PERI-IMPLANTITIS

The treatment goals for Peri implantitis[11]:

The treatments proposed for Peri-implant disease are based on the evidence gained from the treatment of periodontitis. The surface of the implants facilitates adherence of the bacterial biofilm and complicates its elimination.[10]

- Regeneration of bone structures
- Complete elimination of inflammatory processes in the peri-implant tissues
- Reduction in the duration of the treatment
- Creation of aseptic conditions around the implant
- Securing the reliability of the implanted artificial supports

Treatment methods of Peri implantitis [2,6,7]:

1. Acute bacterial infection control to reduce tissue inflammation:

- Mechanical debridement in local areas.
- Irrigation in subgingival sulcus to reduce environmental pathogens
- Administration of topical and systemic antibiotics.[11]

2. Regeneration of bone defects.[10]

3. The treatment is surgical correction of the pockets and regeneration of bone defects around the implant.

Re-evaluation intervals post-therapy for Peri-implantitis: Once a patient has developed Peri-implantitis, there is an increased risk of recurrence and is considered a high-risk patient especially if he has a history of periodontal disease [2] and should be assessed at every 3-4 months.

CONCLUSION:

Peri implant mucositis can be considered analogous to gingivitis and Peri-implantitis can be considered analogous to periodontitis. The borderline between gingivitis to periodontitis and mucositis to peri-implantitis is defined by the degradation of connective tissue and it is followed by epithelial migration and bone resorption.

Microbial colonization and inflammatory reactions in the peri-implant tissues might be analogous to key events in the pathogenesis of periodontitis. Peri-implantitis can result in the loss of dental implants just as untreated Periodontitis can lead to the loss of natural teeth. Periodontitis and Peri implantitis both demand an aggressive treatment approach otherwise the exacerbation of the disease can lead to perpetual damage to the surrounding tissue.

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