

Research Article

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2017 Classification of Peri-Implant Diseases and Conditions

Introduction

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In 2017, a new framework for classifying periodontal and peri-implant illnesses and conditions was introduced by the World Workshop on the Classification of Periodontal and Peri-Implant illnesses and Conditions [1].

On November 9–11, 2017, the European Federation of Periodontology (EFP) and the American Academy of Periodontology (AAP) jointly presented a workshop in Chicago. Academicians from all around the world who study dental implants and periodontal disease were among them. A team from the AAP and EFP reviewed 19 review papers and four consensus reports pertaining to periodontology and implant dentistry. Creating a new classification for peri-implant illnesses and conditions and updating the 1999 classification of periodontal diseases and conditions were the tasks given to the experts. Clinical practice in periodontal and implant specialty will be significantly impacted by the 2017 classification of periodontal and peri-implant disorders [2].

For physicians to accurately diagnose and treat patients, as well as for scientists to study the etiology, pathophysiology, natural history, and treatment of periodontal and peri-implant diseases and disorders, a categorization system is required [3].

The workshop created a new categorization for peri-implant health, peri-implant mucositis, and peri-implantitis. To reach an agreement for this classification that could be accepted globally, an attempt was made to review all facets of peri-implant health, illnesses, and pertinent features of implant site disorders and abnormalities. Case definitions were created so that physicians could utilize them

for both population research and individual case management [3].

The 1989 workshop acknowledged that there were multiple unique clinical manifestations of periodontitis, with varying beginning ages and progression rates. The workshop classified periodontitis as prepubertal, juvenile (localized and generalized), adult, and quickly progressing based on these factors. After deciding that the classification needed to be streamlined, the European Workshop in 1993 suggested dividing periodontitis into two main categories: adult and early onset. Participants in the 1996 workshop concluded that there was not enough fresh data to alter the classification. The 1999 categorization of periodontitis, which has been in use for the past 19 years, underwent significant revisions. Chronic, aggressive (localized and widespread), necrotizing, and a sign of systemic illness are the new classifications for periodontitis. Population studies, basic scientific research, and the findings from prospective studies assessing environmental and systemic risk factors have all produced a significant amount of new material since the 1999 meeting. A new classification paradigm for periodontitis was developed in 2017 as a result of the analysis of this evidence [3].

Classification of Peri-Implant Diseases and Conditions 2017



Peri-Implant Health

It is evident that there are significant structural variations between the two situations, especially with regard to their relationship with surrounding tissues and biological attachment, even though peri-implant health and periodontal health around natural teeth share many clinical characteristics. According to the review by Araujo and Lindhe, the soft and hard tissues surrounding natural teeth and dental implants have different anatomical and histological features. The authors also discussed how these differences could be the cause of the different biological mechanisms governing host response and tissue homeostasis that are seen in the two entities. The trans-mucosal portion of the implant, the abutment, or the restoration is tightly sealed by the peri-implant mucosa in clinical health. The first probing depth is influenced by the height of the soft tissue around the implant after implantation. However, the ideal probing depth for peri-implant health is often ≤ 5.0 mm.⁴ It should be mentioned that different amounts of bone support can result in peri-implant tissue health after peri-implantitis treatment.

In order to diagnose peri-implant health, visual inspection must show that there are no peri-implant symptoms of inflammation, such as pink instead of red, no swelling instead of swollen tissues, and hard instead of soft tissue consistency; absence of extensive bleeding (line or drop) upon probing; The height of the soft tissue at the implant site may affect the depth of the probing pocket. However, peri-implant health is at odds with a gradual rise in probing depth; and absence of additional bone loss after the initial healing period, which shouldn't be more than 2 mm [4].

Either a keratinized (masticatory mucosa) or non-keratinized epithelium (lining mucosa) covers the healthy peri-implant mucosa, which is 3 to 4 mm tall on average. A sulcular epithelium and a thin junctional epithelium line the "coronal" portion of the peri-implant mucosa that faces the implant or abutment, while the more "apical" segment has connective tissue in direct contact with the implant surface. There is a little infiltration of inflammatory cells in the connective tissue lateral to the sulcular epithelium. While the remainder of the implant faces fibrous tissue, vascular structures, or bone marrow, the majority of the intra-bony region is in touch with mineralized bone [5].

Peri-Implant Mucositis

Peri-implant mucositis is an inflammatory lesion of the mucosa around an endo osseous implant that does not result in the loss of the peri-implant bone that supports it. Inflammation of the peri-implant mucosa and the lack of ongoing marginal peri-implant bone loss are crucial factors for the diagnosis of peri-implant mucositis. Bleeding on probing is the clinical indicator of inflammation; other symptoms could be erythema, edema, and suppuration. A non-keratinized barrier epithelium with basal lamina and hemidesmosomes facing the implant or abutment surface is defined by the existence of an oral epithelium that extends into a healthy peri-implant mucosa. Healthy peri-implant mucosa can develop peri-implant mucositis once bacterial biofilms build up around osseointegrated dental implants. It has been shown that the development of an inflammatory response (i.e., experimental peri-implant mucositis) in people is causally related to the experimental accumulation of bacterial biofilms surrounding titanium dental implants [6].

Visual examination proving the presence of peri-implant indicators of inflammation, such as red as opposed to pink, swollen tissues as opposed to no swelling, and soft as opposed to hard tissue consistency, is necessary for the diagnosis of peri-implant mucositis. Existence of suppuration upon probing and/or extensive (line or drop) bleeding; a greater depth of probing than the baseline; and Absence of bone loss beyond the initial remodeling-induced alterations at the crestal bone level [7].

In order to treat peri-implant mucositis and peri-implantitis without surgery, the implant surface is typically mechanically debrided using curettes, ultrasonic devices, air-abrasive devices, or lasers. Local antibiotics or antiseptics may be used as an adjuvant. When nonsurgical treatment for peri-implantitis is unable to control the inflammatory changes, surgery is recommended. The features of the peri-implant lesion should guide the surgical procedure selection. Surgical procedures should strive to provide complete debridement, implant-surface cleaning, and defect repair when deep circumferential and intra bony flaws are present. Although this goal may jeopardize the aesthetic outcome of the implant-supported restoration, the goal of the surgical intervention should be the complete debridement and repositioning of the marginal mucosa in the presence of defects with a predominant supra bony component or without clear bony walls. This will allow the patient to practice good oral hygiene [8].

Peri Implantitis

Inflammation of the peri-implant mucosa and the ensuing progressive loss of supporting bone are the hallmarks of peri-implantitis, a plaque-associated pathological illness that affects the tissues surrounding dental implants. Observational studies have shown that individuals who do not receive routine maintenance therapy and have poor plaque management are more likely to develop peri-implantitis. Anti-infective treatment approaches are effective in reducing soft tissue inflammation and slowing the progression of the disease, according to studies on the management of peri-implantitis.

In addition to radiographic bone loss relative to prior examinations, peri-implantitis locations show clinical symptoms of inflammation, probing depth increases, suppuration, and/or recession of the mucosal margin. Probing depth is associated with bone loss at peri-implantitis sites, making it a measure of the disease's severity. It is crucial to understand that each patient may have bone loss at a different rate. Large numbers and densities of neutrophils, macrophages, and plasma cells are present in peri-implantitis lesions, which extend apically of the junctional/pocket epithelium. In addition, peri-implantitis lesions are larger than those at peri-implant mucositis sites.

Strong evidence suggests that individuals with a history of severe periodontitis, poor plaque management, and no routine maintenance care following implant therapy are more likely to develop peri-implantitis. There is conflicting evidence linking diabetes and smoking to an increased risk of developing peri-implantitis. In daily practice, we frequently come across implants that were placed under less than optimum conditions. These circumstances may therefore be linked to a higher incidence of peri-implantitis. There is no evidence that peri-implantitis is caused by things like implant placement that makes oral hygiene

and maintenance difficult and the presence of submucosal cement after restoration. Occlusal overload, titanium particles, bone compression necrosis, overheating, micromotion, biocorrosion, and peri-implant keratinized mucosa as risk factors [9].

Peri-Implant Soft and Hard Tissue Deficiencies

Decreased alveolar process/ridge dimensions brought on by normal healing after tooth loss cause inadequacies in both soft and hard tissues. Sites linked to significant loss of periodontal support, extraction stress, endodontic infections, root fractures, weak buccal bone plates, poor tooth position, damage, and maxillary sinus pneumatization may have larger ridge deficiencies. Agenesis of teeth, pressure from prostheses, and drugs and systemic disorders that decrease the amount of naturally generated bone can all have an impact on the ridge [3].

Reduced alveolar process/ridge dimensions, which indicate inadequacies in both hard and soft tissues, are a result of the healing process after tooth loss. Larger deficiencies may arise at sites exposed to the following: agenesis of teeth, pressure from soft-tissue supported removable prosthesis, loss of periodontal support, endodontic infections, longitudinal root fractures, thin buccal bone plates, buccal/lingual tooth position in relation to the arch, extraction with additional tissue trauma, injury, maxillary sinus pneumatization, medications, and systemic diseases that reduce the amount of naturally formed bone. Implant malposition, buccal bone loss, thin soft tissue, absence of keratinized tissue, the state of attachment of the neighboring teeth, and surgical trauma are the main causes of recession of the peri-implant mucosa. The level of the periodontal tissues on the teeth next to the implants affects the papilla height between the implants and the teeth. The bone crest between implants determines the height of the papilla between implants. The findings are unclear regarding the necessity of the buccal bone plate for long-term buccal soft tissue support of the implant [9].

Deficits in both soft and hard tissues at dental implants are frequent clinical observations. They may necessitate therapeutic measures since they can cause difficulties and jeopardize implant survival. Therefore, it's critical to comprehend the causes of inadequacies in both soft and hard tissues. In order to improve the clinical results of implant therapy, solutions to address deficits in both soft and hard tissues should be devised based on this understanding [10].

Conclusion

The establishment of optimal and acceptable therapeutic outcomes for periodontal therapies, the systematic evaluation of the biological burden of periodontal inflammation, the classification of gingival and periodontal disease prevalence in populations, and the assessment of individual risk for future disease development all depend on a definition of periodontal health and wellness. To accomplish these objectives, periodontal health needs to be evaluated and characterized at the patient and site levels. Furthermore, epidemiological studies may employ different definitions of periodontal health than those used to guide treatment decisions for specific individuals [11].

Since the 1999 International Classification of Periodontal Diseases, new knowledge has emerged from both biological and clinical

research, which has led to the development of the 2017 World Workshop Classification system for periodontal and peri-implant diseases and conditions. Crucially, it makes the first distinction between an intact and a decreased periodontium and defines clinical health [12]. In terms of treatment and epidemiology, peri-implant disease is a significant issue that besets modern dentistry. The prevalence of peri-implant disease has significantly increased due to the growth of the implantology profession and the rise in the number of implants placed each year. In the lack of a universally recognized classification, its clinical manifestations are peri-implant mucositis and peri-implantitis, which are the equivalents of gingivitis and periodontitis, respectively [13].

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