

# AN OVERVIEW THE IMPACT OF VARIOUS DENTAL PROSTHETIC MATERIALS ON PERIODONTAL/PERI-IMPLANT APPARATUS

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#### Abstract:

A collection of clinical measurements and the analysis of radiographic images are the primary components that are utilized in the process of diagnosing peri-implantar and periodontal conditions. On the other hand, these clinical situations are not sufficient on their own to determine, much less forecast, periimplant bone loss or potential implant failure. Through the evaluation of biomarkers, it may be feasible to make an early diagnosis of periimplant illnesses and determine the rate at which they progress. In the event that they are discovered, biomarkers of peri-implant and periodontal tissue degradation have the potential to notify doctors prior to the manifestation of clinical symptoms. Because of this, it is essential to take into consideration the possibility of producing chair-side diagnostic tests that are specific for a certain biomarker and indicate the current activity of the illness condition. For metal-free ceramic prostheses, the inflammatory response is reduced regardless of the manufacturing (CAD/CAM) systems is advised for their construction. Additionally, it is hypothesized that metal-ceramic prostheses produce alterations in the composition of the subgingival microbiota, resulting in a biofilm that is more dysbiotic and contains a larger prevalence of periodontopathogenic bacteria. This may further contribute to the deterioration of periodontal health.

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#### Introduction:

The inflammation of peri-implant tissues and the gradual loss of supporting bone around implants are the primary characteristics of peri-implant diseases [1]. These illnesses might potentially result in the failure of implants. The infection of the periimplant mucosa, which corresponds to the criteria of peri-implantitis, and/or the immune response, which corresponds to the description of a foreign body reaction, could be the cause of bone loss [2]. The diagnosis of peri-implantitis is primarily based on a number of clinical parameters that reflect abnormal inflammation and destruction around implants. These parameters include bleeding on probing (BOPi) and/or suppuration, an increase in peri-implant probing depth (PiPD). and radiographic evidence of bone loss that has occurred after the initial healing process [3]. The prevalence of peri-implantitis, on the other hand, was extremely variable and ranged from 1 to 47% [5]. This was due to the fact that the definitions of peri-implantitis differed substantially from one study to the next. This was because the classifications were based on the different combinations of clinical symptoms that were associated with different levels of severity [4]. A definition of peri-implantitis was recently proposed at the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions that took place in 2017 [4]. Or, as an alternative, it was based on specific thresholds, such as a PiPD of at least 6 mm and a bone level of at least 3 mm at least in one site around the implant. This definition is based on the combination of the existence of BOPi or suppuration, the longitudinal measurement of PiPD, and changes in bone level. However, correlations between various periimplant tissue characteristics, such as the mean PiPD, bone level (BLi), and bone ossification pressure (BOPi), as well as between these parameters and peri-implantitis, did not systematically correspond [6]. This was in contrast to the periodontal parameter associations that have traditionally been observed in periodontal diseases and during periodontal maintenance. It is possible that the various peri-implant tissue parameters that used to define disease severity and are activity/progression may not only correspond to the responses of the host's peri-implant tissues to the accumulation of plaque or biofilm or to implant foreign bodies, but they may also reflect the complex and specific influence of the periodontal environment and implant/prosthesis procedures [7].

The periodontium is comprised of two soft tissues, namely the gingiva and the periodontal ligament, as well as two hard tissues, namely the root cementum and the alveolar bone [7]. The periodontium is the tissue that is responsible for supporting the teeth. It is now generally acknowledged that periodontal disease (PD) is a complex pathological entity that is brought on by polymicrobial dysbiosis and inflammation that is mediated by the host [7]. On the other hand, gingival inflammation, destruction of the periodontal ligament, bone loss, bacterial colonization and invasion, increased numbers of polymorphonuclear (PMN) and epithelial cells, increased volume and decreased pH of the gingival crevicular fluid (GCF), and increased periodontal and gingival indices are all important components in the pathophysiology of Parkinson's disease (PD) and its associated clinical features [8]. The frequency of Parkinson's disease is believed to be between 30 and 50 percent across the globe [7,8]. The most recent classification of Parkinson's disease (PD) is based on the severity of the disease (stages I-IV) and the progression of the disease (grades A–C). However, for practical purposes, we can divide it into gingivitis, which refers to inflammation of the gums, and periodontitis, which is characterized by the destruction of periodontal tissues in addition to inflammation [9].

One of the variables that specifically contributes to the development of periodontal disease is the utilization of prosthetic restorations that are poorly constructed, characterized by a marginal and internal fit that is insufficient and exceeds 120 micrometers. As a result of this, and as a result of a greater marginal discrepancy, the cement forms a thicker layer and comes into contact with the environment of the oral cavity. This causes the cement to dissolve, which in turn leads to an increased accumulation and retention of bacteria in the area, which can cause irreversible damage to the periodontal and pulpal tissues if it is not detected in a timely manner [10].

#### **Review:**

The clinical manifestations of periodontitis include a pathological deepening of the gingival sulcus, loss of attachment, and the creation of periodontal pockets with supporting alveolar bone resorption [10]. Periodontitis is an inflammatory dental illness. The commencement and progression of this disease are both the result of an interaction between the pathogenic bacteria that are present in the subgingival dental biofilme and the reaction of the host. Generally speaking, periodontal tissue degradation is a progressive process that is defined by periods of active and remission disease without clearly alarming symptoms. Damage to the periodontal tissues that is irreversible can occur when they are neglected. The presence of specific bacteria has been shown to be associated with periodontal and periimplantar inflammation [11], despite the fact that this association has been well known. In particular, smoking, a history of periodontal disease, inadequate oral hygiene, and leftover excess cement have all been reported to be related with peri-implant disorders. Additional variables and clinical confounding factors have also been identified. Recent research has also begun to concentrate on prosthetic characteristics, such as the emergence profile and angle of the restoration. These studies have demonstrated that restorations that are over-contoured have a greater likelihood of developing periimplantitis [12]. An excellent method for lowering the likelihood of developing periodontitis or peri-implantitis, respectively, is to make a prompt diagnosis of gingivitis or mucositis. An assortment of clinical measurements and pocket probing depths, bleeding on probing, and the evaluation of radiographic images are the primary factors that are utilized in the process of diagnosing peri-implantar and periodontal illnesses. When taken by themselves, these clinical characteristics are not sufficient to indicate whether or not there is ongoing periimplant disease, future crestal bone loss, or future implant failure. Additionally, it is vital to have additional information based on medical records; however, this information does not provide any information regarding the current state of disease activity, nor does it identify the individuals who are susceptible to the advancement of the disease in the future [7–9]. Conventional diagnostic techniques necessitate the use of many manual recordings, the presence of professional examiners who possess specialized knowledge, and the fact that clinical data only pertain to pre-existing illness states, making it impossible to anticipate the onset of clinical symptoms [10].

Recently, a consensus was reached between the European Federation of Periodontology (EFP) and the American Academy of Periodontology (AAP) about a new classification of periodontal disorders. This classification takes into account the severity, extent, and advancement of the disease through the utilization of a staging and grading system [11]. The development of tools for reliable diagnosis and the prediction of the prognosis of peri-implant illness is one of the objectives of this new categorization [12]. As a result, this new classification scheme was developed to enable the incorporation of changes that are in accordance with future advancements, such as diagnosis based on biomarkers.

It is feasible that the evaluation of biomarkers may allow for the early detection of peri-implant illnesses as well as the prediction of their rate of progression. Clinicians may receive a warning from biomarkers of peri-implant and periodontal tissue degradation after they have been identified. This may occur before clinical indications manifest themselves. It is possible for experts to improve the accuracy of early identification of peri-implant and periodontal disorders, as well as the prognosis of disease progression and the monitoring of treatment outcomes, by combining these tactics with standard procedures [13].

The degree to which periodontal and peri-implant disorders are associated with one another is highly variable, depending on the description of the disease and the frequency with which it is evaluated. When it comes to cross-sectional research that investigate the connection between current periodontal status and the periimplantitis/bone loss, there is a significant amount of heterogeneity [13]. According to the findings of their investigation, the selection of disease parameters had an effect on the connection between periodontal and peri-implant illnesses. For example, the cut-off values of 5% periodontal pocket probing depth (PPD) that is greater than or equal to 4 millimeters and bleeding on probing around teeth (BOP) that is greater than or equal to thirty percent per patient were shown to be connected with the mean and percentage of periodontal pocket probing depth (PPD) that is greater than or equal to four millimeters and bleeding on probing around teeth (BOPi), but they were not associated with the mean bone level around the implant (BLi) [12]. Based on the presence of BOPi, BLi  $\geq 2$  mm, and PiPD  $\geq 5$  or 6 mm around implants, the mean PPD and residual pockets with % PPD  $\geq$ 5 mm were connected with two alternative definitions of peri-implantitis in a study [13]. These definitions were utilized to determine the existence of peri-implantitis. The maximum periodontal pressure (BOP) was only linked with the first one (PiPD  $\geq 5$  mm), and the mean periodontal clinical attachment level (CAL) was only associated with the second one (PiPD  $\geq 6$ mm) [13]. These different associations between the parameters suggested clinical that the characteristics and profiles of patient responses to periodontal treatment may selectively impact the peri-implant status [13]. This is because each periodontal parameter and combination represented different aspects of the pathogenesis and morbidity of periodontal disease, as well as severity, complexity, treatment response, and progression.

It is generally agreed upon within the scientific community that the success of an implant cannot be only evaluated based on the implant's ability to survive; rather, it should also take into account the conditions around the implant and the stability of the crestal bone level. It is generally acknowledged that the initial remodeling of peri-implant bones takes place as a result of the biological adaptation of peri-implant tissues, and it is anticipated that subsequent tissue stabilization will have place because of this [12]. Although the physicians consider it to be a normal process of bone remodeling, the fact that an unstable bone might create a variety of difficulties leaves the clinician confused as to whether or not the implant will be stable for a longer period of time. Therefore, it is the responsibility of the doctor to try to achieve the least amount of bone loss feasible [14].

Peri-implantitis is a condition that causes inflammation in the tissues around dental implants. It is defined by the gradual loss of bone that supports the implant. In accordance with the consensus report of workgroup 4 at the "2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions," it has been determined that periimplant health is characterized by the absence of erythema, bleeding on probing, edema, or suppuration. A range of probing depths that are consistent with health cannot be defined since it is not possible. Despite the presence of diminished bone support, peri-implant health can still be present surrounding dental implants [14].

All of the scientific data, as well as the clinical assessment that is employed by clinicians in the modern day, is only based on clinical, analytical, and radiographic parameters. These parameters, in fact, offer only a limited amount of information that can be used to address the multi-factoral complexity of implant-supported rehabilitation operations. Furthermore, when it comes to identifying and staging peri-implant disorders, those approaches can only register the pre-existing status, and not the current situation itself. Furthermore, they do not take into consideration the clinical condition of the patient. On top of that, it does not take into account systemic diseases, lifestyle, hormonal changes, or ageing, among other characteristics that are associated with individual inflammatory processes that may therefore alter the local immunological response. The most difficult task for any doctor, on the other hand, is to accurately anticipate whether or not a patient will be successful in rehabilitation or to identify patients who are at a high risk of developing a disease [15].

In this manner, it is necessary to develop diagnoses that are backed by methods that are precise and systematic, such as the sciences of omics. The technologies of omics have emerged as a potent instrument for investigating the various molecular pathways that are involved in the transition between healthy and sick states. It is common practice in the field of medicine to make use of molecules such as biomarkers in order to properly evaluate the status of a disease or the reactions to a treatment, as well as to contribute to the discovery of the targets of new medicines [15]. The literature is slowly but surely beginning to take into consideration this technique as a potential future protocol that could be adopted in the monitoring of peri-implant disease.

Surgical and prosthetic methods, as well as the kind of implant, have been proven to have an effect on the peri-implant conditions that occur surrounding implants, such as peri-implant pressure ulcers (PIPD) and blepharitis (BLi), as well as the changes that occur in their follow-up. For example, the location of the implant in the anterior maxilla and the design of the implant at the bone level were both related with a higher bone loss after the implant had healed [16]. There was a correlation between reduced keratinized mucosa conditions and increased plaque accumulation and inflammation, particularly in the vicinity of the implants. When it came to cemented retention prosthesis, the PiPD levels were found to be higher. There was a lack of consistency in the association between periimplantitis and the contour and type of prosthesis. It is possible that the risk of peri-implantitis could be increased by the combination of prosthesis variables, such as an emergence angle more than thirty degrees and implants that are placed at bone level. In spite of this, it was not possible to definitively demonstrate the respective influence of implant/prosthesis variables and the periodontal state on the peri-implant status [16].

It is possible that the diagnostic significance of linked clinical indications, and subsequently the evaluation of risk factor influences, could be influenced by the differences in histology, physiology, and pathophysiology that exist between periodontal and peri-implant tissue. In order to define pre-established probing depth, attachment, and bone level with normal and pathologic values, it was not possible to do so due to the variety of peri-implant tissue conditions that occurred following implant and prosthesis healing. In order to diagnose peri-implantitis and the influence of risk factors, it is now possible to make modifications to the parameters of the tissue around the implant [16].

## **Conclusion:**

As a potential consequence of the initial implant and prosthesis process healing, the literature indicated that the peri-implant and periodontal soft tissue statuses were differentiated from one another. Peri-implant parameters, on the other hand, were primarily connected with their corresponding periodontal parameters during the implant follow-up. This was the case regardless of the fact that these parameters were often associated with the characteristics of the implant and the prosthesis. An additional clinical association between periodontal and peri-implant soft tissue conditions might be noticed during follow-up treatments, as evidenced by these findings, in addition to the pathologic connection that exists between periodontal illnesses and peri-implant diseases. The use of CAD/CAM systems is advised for the manufacturing of metal-free ceramic prostheses; nonetheless, regardless of the method of fabrication, these prostheses generate a milder inflammatory response. A more dysbiotic biofilm with a larger frequency of periodontopathogenic bacteria is produced as a result of the use of metalceramic prostheses, which also cause alterations in the makeup of the subgingival microbiota. Azurocidin is released by neutrophils as a response, and it has a powerful chemoattractant effect, causes vascular leakage, and contributes to the removal of germs by opsonizing microorganisms. This makes it easier for phagocytes to recognize and take up the bacteria. Other types of cells, such as gingival fibroblasts, are responsible for the release of chemokines, such as fractalkine (CX3CL1), which serves a dual purpose as a chemotactic factor.

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