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Review Article

Peri-Implantitis; Comparison with Periodontitis from Etiological Perspective: A Literature Review

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Abstract

Peri-implantitis is the inflammation of mucosa around a dental implant along with loss of supporting bone. This disease has some similarities with periodontitis but there are important differences as well. Microorganisms play an important role in the development of both periodontitis as well as peri-implantitis. The presence of a foreign body in case of peri-implantitis and a lack of periodontal ligament make this condition more complicated. Bad oral hygiene and cigarette smoking are risk factors for both the diseases. Certain systemic conditions are also implicated as risk factors for the periodontitis but regarding peri-implantitis the evidence collected so far is not sufficient. An aberrant host response to microbiological stimuli is also important the causation of both the diseases yet the exact nature of this association is not fully understood. The knowledge of risk factors for developing peri-implantitis is necessary for the clinicians to provide detailed counseling to the high-risk patients and stress the need for good personal and clinical care for the implants.

Keywords: *Peri-Implantitis; Periodontitis; Inflammation; Oral Hygiene*

Background

Periodontitis is the inflammatory process that occurs in tissues surrounding natural teeth in response to dental plaque and bacteria and affects the soft and hard tissues around teeth. In addition to bacteria, plaque and other risk factors; host response is also critical in the development and progression of periodontitis. Periodontitis is one of the most prevalent chronic diseases in humans the world over. Periodontitis usually starts in the form of local gingival inflammation called "gingivitis" and when left untreated, this can progress to different stages of periodontitis. The chronic inflammation in periodontitis results in periodontal soft and hard tissue destruction, loss of epithelial attachment and alveolar bone. This results in the formation and deepening of periodontal pockets. The severity of periodontitis is usually assessed by certain clinical parameters such as periodontal pocket depth (PD), bleeding on probing (BOP), alveolar bone loss and mobility. Among these, periodontal pocket depth is one of the most frequently used indicators for the assessment of the severity of periodontitis. In periodontal health, there is no loss of epithelial attachment and the periodontal pocket is less than 2 mm deep [1]. Patients with periodontal pocket depth of 6mm or more are diagnosed with severe periodontitis [2]. Severe forms of periodontitis may affect 10-16% of the population [3,4]. Physical cleaning methods combined with local or systemic antimicrobial therapy is widely used for treating periodontitis but recurrence of the disease is a frequent problem and may occur in up to 8% of the population [1]. Recurrent periodontitis is also called refractory periodontitis and it is one of the mains causes of tooth loss.

Lost natural teeth can be replaced with a variety of dental prosthesis, dental implants being one of the most successful methods. Several types of dental implants are used presently in dentistry to replace the roots of natural teeth. Broadly they are classified as endosteal (implants within the bone), subperiosteal (placed on bone), or transosteal (implants placed through the bone from the superior to the

inferior aspect). Currently endosteal dental implants are the most commonly used. Surgical procedure involved in the installation of endosteal implants can be either two-stage (a portion of the implant is placed within the bone and allowed to heal uninterrupted for 4-6 months prior to placement of the coronal aspect) or single-stage (the implant is placed within the bone but the coronal aspect is left at the gingival margin and not covered with soft tissue, therefore not needing a second surgical stage of uncovering the implant). Three basic types of synthetic biomaterials have been in use for dental implants: metals (Titanium) and alloys (titanium-aluminum-vanadium alloy), ceramics (aluminum oxide, hydroxylapatite) and carbon, and polymers, mainly polymethylmethacrylate (PMMA) [5]. Dental implants in contemporary dentistry are almost exclusively made from commercially pure titanium or titanium alloys. Furthermore, titanium dental implants can be coated with Hydroxylapatite to produce a bio-active surface which promotes and induces a direct bond between the implant and bone tissue surrounding it [6]. The formation of a direct interface between an implant and bone, without intervening soft tissue is called osseointegration and this process was discovered and described first by P.I Branemark [7].

Despite high success rates mentioned for dental implants [8], failure of dental implants also occur and can lead to the loss of dental implant. Failure of dental implant can occur due to the inability of tissue to establish osseointegration prior to the placement of dental prosthesis and this kind of failure is categorized as early implant failure and is suggested to occur due to interference with the healing process. Possible causes include a lack of adequate bone volume; surgical trauma, lack of primary stability; intra-osseous infection and bacterial contamination of the recipient site [9,10]. Failures of dental implants occurring after occlusal loading are classified as late failures and are suggested to occur due to the breakdown of the already established osseiointegration. Breakdown of the established osseointegration occurs due to peri-implantitis and/or mechanical overload [9,11-13]. Five-year failure rate of 7.7 % has been reported for the Branemark implants in one study [14].

Peri-implantitis is a condition affecting implants and it is the inflammation of mucosa around a dental implant along with loss of supporting bone [15]. Peri-implant mucositis is defined as the inflammation of peri-implant soft tissue without bone loss and can be considered the peri-implant equivalent of gingivitis. Continued inflammation and bone loss around an implant can lead to mobility and eventual loss of the implant. Diagnosis of peri-implantitis is made like periodontitis [16] and involves measuring clinical parameters such as bleeding on probing, plaque index, peri-implant loss of gingival attachment, suppuration and mobility. Radiographic assessment of peri-implant bone loss and microbiological sampling also form part of the current diagnostic tools for peri-implantitis. Peri-implantitis has been reported to occur in 5-8% of cases for different implant systems [17,18]. In terms of late implant failures, results of meta-analyses suggest that peri-implantitis accounts for 10 - 50% of failed implants after one year of loading [14,19].

The aim of current study was to update the knowledge of clinicians and dental students about peri-implantitis, its etiology and risk factors associated with it, as compared to periodontitis.

Structural Aspects

Although there are similarities between the natural periodontium and an osseointegrated dental implant's union with surrounding tissues (bone-fiber-implant interface), basic structural differences exist. Natural tooth's union with the alveolar bone is cushioned by periodontal ligament (PDL) and the fibers of this PDL are attached and run perpendicular to the tooth surface. This arrangement is of importance to natural teeth from a functional point of view. In contrast, the fibers in between a dental implant and bone are not attached to the implant and they run parallel to the implant surface. The presence of a foreign body (dental implant) is also another important factor which distinguishes peri-implantitis from periodontitis and might play a role in the host response to inflammation [20].

Etiology and risk factors

There is enough evidence that bacteria, especially gram negative anaerobes, are implicated in the causation of periodontitis but the inflammatory response of periodontal tissues to bacterial infection is influenced both by environmental as well as genetic factors [21,22]. Importance of the host response in periodontitis is highlighted by the fact that known periodontal pathogens strongly associated with

periodontitis are sometimes found in the oral cavity without causing any obvious periodontal problems [23]. Studies have shown that periodontitis aggregates in families [24] which support the hypothesis of genetic factors influencing periodontitis. Genetic polymorphism has also been suggested to have a role in causing periodontitis although the evidence is inconclusive [25]. Among the environmental factors, there is ample evidence that smoking is a risk factor for chronic periodontitis [26-29]. Oral hygiene is another important factor often implicated in the causation of periodontitis. Studies have shown a modest correlation between plaque scores and measures of early periodontitis in young (20 - 40 years of age) population [30,31] but this association was not found in older population [32,33]. Stress, depression and anxiety are also reported to be potential risk factors for periodontitis [34-37]. Certain systemic diseases such as diabetes mellitus, osteoporosis and obesity [21,38,39] are also known to worsen the clinical manifestations of periodontitis but whether they are causative is still not clear. The immunomodulatory potential of certain systemic diseases like diabetes mellitus can alter the clinical course of periodontitis.

Peri-implantitis has some parallels with periodontitis from etiological point of view however, in most of the literature it is dealt with as a separate disease entity. Microorganisms play an important role in the development and progression of peri-implantitis and this view is supported by several animal and human studies [17,18,40]. A complex subgingival microbiota dominated by Gram negative anaerobes such as *Porphyromonas gingivalis*, *Prevotella intermedia/Prevotella nigrescens* and *Aggregatibacter actinomycetemcomitans*, is established during the tissue breakdown in peri-implantitis which closely resembles that found in chronic adult periodontitis [41,42]. Other organisms not primarily associated with periodontitis such as *Staphylococcus* spp., *Enterics* and *Candida* spp. have also been found in peri-implantitis lesions [42,43]. In contrast, implants surrounded by tissue free from clinical inflammation demonstrate a microbiota which is associated with periodontal health [43].

Population studies for peri-implantitis show a clustering of the disease in sub-groups of individuals and patients who have already lost an implant are at elevated risk for other implant losses [12,13,44]. This may be a direct effect of genetic factors influencing peri-implant disease or may relate to an aberrant immune response from the host. Whether the host response to inflammation in peri-implantitis is different from the inflammatory host response in periodontitis, is not clear. A hypersensitivity of the neutrophils is thought to contribute to the tissue destruction in periodontitis [27,28] and since a significant number of patients receiving dental implants have a history of periodontitis, it's logical to assume that a microbial challenge coupled with an aberrant host response could be the possible initiating factors for developing peri-implantitis. This argument is supported by some studies where they show high prevalence of peri-implantitis in patients with a history of periodontitis [45-47] but the evidence does not seem conclusive.

Bain and Moy have recently described the deleterious effects of smoking on dental implants [48]. They reported that a significantly higher percentage of implants failed in the smokers than in nonsmokers. Smokers had an overall implant failure rate of 11.3% compared to nonsmokers who had only 4. 8% implants failed. Cigarette smoking was also associated with significantly higher levels of marginal bone loss [49,50].

Uncontrolled diabetes is a risk factor for periodontitis but its effects are not very well documented on peri-implantitis. A one year study of implant survival in non-insulin dependent diabetes patients reports a 7.3% failure rate [51] which indicates that osseointegration can be achieved in diabetics. The medium to long term prognosis of implants and peri-implantitis in diabetics is currently unknown but keeping in mind the immunomodulatory nature of the disease it is likely to be a potential risk factor for peri-implantitis. Other systemic factors such as a history of medications usage such as, bisphosphonates, has been reported to positively correlate with implant loss [52,53].

Plaque and calculus deposition acts as mechanical and chemical irritant for oral soft tissues and keeping good oral hygiene is a key modifiable factor in the prevention of certain diseases of the soft and hard oral tissues. Inadequate oral hygiene is also a possible risk factor for implant failure and peri-implantitis [54]. In another study a synergistic effect of cigarette smoking and bad oral hygiene has been reported [55].

Clinical significance

It is important to understand differences between periodontitis and peri-implantitis from a clinical point of view and both conditions should be treated as different disease entities, both theoretically as well as in clinical settings. High risk patients must be counseled in detail before the installation of dental implant and high standards of clinic and home care advised for such patients. Knowledge of the risk factors and indicators associated with peri-implantitis is needed to improve and maintain over time the high success rate of dental implants.

Bibliography

1. Angeli, F., *et al.* "Association between Periodontal Disease and Left Ventricle Mass in Essential Hypertension." *Hypertension* 41.3 (2003): 488-92.

2. Elter J R., *et al.* "Relationship of Periodontal Disease and Tooth Loss to Prevalence of Coronary Heart Disease." *Journal of Periodontology* 75.6 (2004): 782-790.

3. Fuster V., *et al.* "The Pathogenesis of Coronary Artery Disease and the Acute Coronary Syndromes (1)." *New England Journal of Medicine* 326.4 (1992): 310-318.

4. Holmgren C J., *et al.* "Periodontal Conditions among the Middle-Aged and the Elderly in Hong Kong." *Community Dental Oral Epidemiology* 22.5 (1994): 396-402.

5. Lemons J E. "Dental Implant Biomaterials". *Journal of the American Dental Association* 121.6 (1990): 716-719.

6. Denissen H W. "Implants of Hydroxyapatite and Titanium". *Zahnarzt* 29.4-5 (1985): 297-300.

7. Branemark P I., *et al.* "Osseointegration and Its Experimental Background." *Journal of Prosthetic Dentistry* 50.3 (1983): 399-410.

8. Fiorellini J P., *et al.* "Longitudinal Studies of Implant Systems." *Periodontology* 2000 17 (1998): 125-131.

9. Quirynen M., *et al*. "Infectious Risks for Oral Implants: A Review of the Literature". *Clinical Oral Implants Research* 13.1 (2002): 1-19.

10. Shibli J A., *et al*. "Analysis of Failed Commercially Pure Titanium Dental Implants: A Scanning Electron Microscopy and Energy-Dispersive Spectrometer X-Ray Study". *Journal of Periodontology* 76.7 (2005): 1092-1099.

11. Mombelli A. "*In Vitro* Models of Biological Responses to Implant Microbiological Models". *Advances in Dental Research* 13 (1999): 67-72.

12. Roos-Jansaker A M., *et al*. "Nine- to Fourteen-Year Follow-up of Implant Treatment. Part I: Implant Loss and Associations to Various Factors". *Journal of Clinical Periodontology* 33.4 (2006): 283-289.

13. Roos-Jansaker A M., *et al*. "Nine- to Fourteen-Year Follow-up of Implant Treatment. Part Iii: Factors Associated with Peri-Implant Lesions". *Journal of Clinical Periodontology* 33.4 (2006): 296-301.

14. Esposito M., *et al.* "Biological Factors Contributing to Failures of Osseointegrated Oral Implants. (I). Success Criteria and Epidemiology." *European Journal of Oral Sciences* 106.1 (1998): 527-551.

15. Zitzmann N U and T Berglundh. "Definition and Prevalence of Peri-Implant Diseases". *Journal of Clinical Periodontology* 35.8 (2008): 286-291.

16. Bragger U., *et al.* "Correlations between Radiographic, Clinical and Mobility Parameters after Loading of Oral Implants with Fixed Partial Dentures. A 2-Year Longitudinal Study." *Clinical Oral Implants Research* 7.3 (1996): 230-239.

17. Berglundh, T., *et al.* "Soft Tissue Reaction to De Novo Plaque Formation on Implants and Teeth. An Experimental Study in the Dog." *Clinical Oral Implants Research* 3.1 (1992): 1-8.

18. Berglundh T L., *et al.* "A Systematic Review of the Incidence of Biological and Technical Complications in Implant Dentistry Reported in Prospective Longitudinal Studies of at Least 5 Years." *Journal of Clinical Periodontology* 29 (2002): 197-212.

19. Esposito M., *et al.* "Biological Factors Contributing to Failures of Osseointegrated Oral Implants. (Ii). Etiopathogenesis." *European Journal of Oral Sciences* 106.3 (1998): 721-764.

20. Irshad Muhammad., *et al*. "Influence of Titanium on *in Vitro* Fibroblast–Porphyromonas Gingivalis Interaction in Peri-Implantitis." *Journal of Clinical Periodontology* 40.9 (2013): 841-849.

21. Heitz-Mayfield L J. "Disease Progression: Identification of High-Risk Groups and Individuals for Periodontitis." *Journal of Clinical Periodontology* 32 (2005): 196-209.

22. Kinane D F., *et al*. "The Genetic Basis of Periodontitis". *Periodontology* 2000 39 (2005): 91-117.

23. Lamont R J and H F Jenkinson. "Life Below the Gum Line: Pathogenic Mechanisms of Porphyromonas Gingivalis". *Microbiology and Molecular Biology Reviews* 62.4 (1998): 1244-1263.

24. Petit M D., et al. "Prevalence of Periodontitis and Suspected Periodontal Pathogens in Families of Adult Periodontitis Patients". Journal of Clinical Periodontology 21.2 (1994): 76-85.

25. Nikolopoulos G K., *et al*. "Cytokine Gene Polymorphisms in Periodontal Disease: A Meta-Analysis of 53 Studies Including 4178 Cases and 4590 Controls". *Journal of Clinical Periodontology* 35.9 (2008): 754-767.

26. Gelskey S C. "Cigarette Smoking and Periodontitis: Methodology to Assess the Strength of Evidence in Support of a Causal Association." *Community Dentistry and Oral Epidemiology* 27.1 (1999): 16-24.

27. Gustafsson A and B Asman. "Increased Release of Free Oxygen Radicals from Peripheral Neutrophils in Adult Periodontitis after Fc Delta-Receptor Stimulation." *Journal of Clinical Periodontology* 23.1 (1996): 38-44.

28. Gustafsson A B., *et al.* "Cigarette Smoking as an Aggravating Factor in Inflammatory Tissue-Destructive Diseases. Increase in Tumor Necrosis Factor-Alpha Priming of Peripheral Neutrophils Measured as Generation of Oxygen Radicals." *International Journal of Clinical and Laboratory Research* 30.4 (2000): 187-190.

29. Haber J., *et al.* "Evidence for Cigarette Smoking as a Major Risk Factor for Periodontitis." *Journal of Periodontology* 64.1 (1993): 16-23.

30. Tanner A C., *et al*. "Clinical and Other Risk Indicators for Early Periodontitis in Adults". *Journal of Periodontology* 76.4 (2005): 573-581.

31. Craig R G., *et al.* "Progression of Destructive Periodontal Diseases in Three Urban Minority Populations: Role of Clinical and Demographic Factors." *Journal of Clinical Periodontology* 30.12 (2003): 1075-1083.

32. Haffajee A D., *et al.* "Clinical Risk Indicators for Periodontal Attachment Loss." *Journal of Clinical Periodontology* 18.2 (1991): 117-125.

33. Tanner A., *et al*. "Microbiota of Health, Gingivitis, and Initial Periodontitis". *Journal of Clinical Periodontology* 25.2 (1998): 85-98.

34. Breivik T., *et al.* "Emotional Stress Effects on Immunity, Gingivitis and Periodontitis." *European Journal of Oral Sciences* 104.4 (1996): 327-334.

35. Genco R J., *et al.* "Relationship of Stress, Distress and Inadequate Coping Behaviors to Periodontal Disease." *Journal of Periodontology* 70.7 (1999): 711-723.

36. Genco, R. J., *et al.* "Models to Evaluate the Role of Stress in Periodontal Disease." *Annals Periodontology* 3.1 (1998): 288-302.

37. Freeman R and S. Goss. "Stress Measures as Predictors of Periodontal Disease--a Preliminary Communication." *Community Dentistry and Oral Epidemiology* 21.3 (1993): 176-177.

38. Kinane D F. "Periodontitis Modified by Systemic Factors". *Annals of Periodontology* 4.1 (1999): 54-64.

39. Kuo L C., *et al*. "Associations between Periodontal Diseases and Systemic Diseases: A Review of the Inter-Relationships and Interactions with Diabetes, Respiratory Diseases, Cardiovascular Diseases and Osteoporosis". *Public Health* 122.4 (2008): 417-433.

40. Pontoriero R., *et al*. "Experimentally Induced Peri-Implant Mucositis. A Clinical Study in Humans". *Clinical Oral Implants Research* 5.4 (1994): 254-259.

41. Becker W., *et al.* "Clinical and Microbiologic Findings That May Contribute to Dental Implant Failure." *International Journal of Oral & Maxillofacial Implants* 5.1 (1990): 31-38.

42. Rams T E., *et al*. "Clinical and Microbiological Findings on Newly Inserted Hydroxyapatite-Coated and Pure Titanium Human Dental Implants". *Clinical Oral Implants Research* 2.3 (1991): 121-127.

43. Leonhardt A., *et al*. "Microbial Findings at Failing Implants". *Clinical Oral Implants Research* 10.5 (1999): 339-345.

44. Hutton, J. E., *et al*. "Factors Related to Success and Failure Rates at 3-Year Follow-up in a Multicenter Study of Overdentures Supported by Branemark Implants." I*nternational Journal of Oral and Maxillofacial Implants* 10.1 (1995): 33-42.

45. Klinge B., *et al*. "Peri-Implantitis". *Dental Clinics of North America* 49.3 (2005): 661-676.

46. Hardt C R., *et al.* "Outcome of Implant Therapy in Relation to Experienced Loss of Periodontal Bone Support: A Retrospective 5- Year Study." *Clinical Oral Implants Research* 13.5 (2002): 488-494.

47. Wennstrom J L., *et al*. "Oral Rehabilitation with Implant-Supported Fixed Partial Dentures in Periodontitis-Susceptible Subjects. A 5-Year Prospective Study". *Journal of Clinical Periodontology* 31.9 (2004): 713-724.

48. Bain C A and P K Moy. "The Association between the Failure of Dental Implants and Cigarette Smoking." *International Journal of Oral and Maxillofacial Implants* 8.6 (1993): 609-615.

49. Lindquist LW., *et al*. "A Prospective 15-Year Follow-up Study of Mandibular Fixed Prostheses Supported by Osseointegrated Implants. Clinical Results and Marginal Bone Loss". *Clinical Oral Implants Research* 7.4 (1996): 329-336.

50. Haas R., *et al.* "The Relationship of Smoking on Peri-Implant Tissue: A Retrospective Study." *Journal of Prosthetic Dentistry* 76.6 (1996): 592-606.

51. Shernoff A F., *et al*. "Implants for Type Ii Diabetic Patients: Interim Report. Va Implants in Diabetes Study Group". *Implant Dentistry* 3.3 (1994): 183-185.

52. Zahid T M., *et al*. "Influence of Bisphosphonates on Alveolar Bone Loss around Osseointegrated Implants". *Journal of Oral Implantology* 37.3 (2011): 335-346.

53. Starck W J and B N Epker. "Failure of Osseointegrated Dental Implants after Diphosphonate Therapy for Osteoporosis: A Case Report". *International Journal of Oral and Maxillofacial Implants* 10.1 (1995): 74-78.

54. Weyant R J. "Characteristics Associated with the Loss and Peri-Implant Tissue Health of Endosseous Dental Implants". *International Journal of Oral and Maxillofacial Implants* 9.1 (1994): 95-102.

55. Lindquist L W., *et al*. "Association between Marginal Bone Loss around Osseointegrated Mandibular Implants and Smoking Habits: A 10-Year Follow-up Study". *Journal of Dental Research* 76.10 (1997): 1667-1674.

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