THE PERI-IMPLANTITIS CONUNDRUM – A MINI REVIEW

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ABSTRACT

Peri-implantitis (PI) is "a ticking time bomb in dental implants" and can lead to its failure even in cases of successful osseointegration. This is of special concern to us as it can cause immense financial and psychological burdento the patient. **Objective:** To review critical scientific evidence for a through understanding of the various aspects of peri-implantitis (definition, classification, incidence and prevalence and the treatment) and thus develop recommendations to reduce its occurrence. **Methodology:** Extensive literature search was done to get a better understanding of peri-implant diseases. Changes in trend towards treatment of peri-implantitis were also mentioned. **Conclusion:** Peri-implantitis is one of the most common late failure complication of dental implants. Its prevention encompasses a set of various actions aimed at controlling the disease. A through understanding into the etiology, pathogenesis, classification of peri-implantitis is needed to finally ensure proper treatment planning.

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KEYWORDS

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INTRODUCTION

Dental implants have become an indispensable established therapy for the replacement of missing teeth and their high success rate has led to their acceptance as a realistic treatment alternative in modern dentistry. However, in spite of their high survival rate, even an implant with successful osseointegrationcan develop complications eventually leading to its failure/loss. Dental implant complications have been claasified as (i) compromised successful implant (presence of inflammation and fistula near a successfully osseointegrated implant), (ii) failing implant (increasingboneloss inafunctional implant) and (iii) failed implant (infection around a compromised implant). Mellonig et al.(1) placed dental implant failures in two categories; namely, failure due to infection (periimplantitis or retrograde periimplantitis) and failure due to trauma (excessive overloading or implant fracture). Meffert et al.(2) also categorized problematic implants into ailing, failing or failed. Ailing implants demonstrate bone loss with pocket formation which is static at maintenance phases. Failing implants demonstrate bone loss with pocket formation, bleeding upon probing and exudates. Failed implants are clinically mobile. All these failures can also be categorized into two 1.biological 2. Biomechanical. (3,4) Biomechanical failures are associated with functional loads exceeding the implant-bone interface due to overloading condition such as bruxism/ clenching and fracture or mechanical damage to implant or super structure. Biological failures are associated with microbial plaque accumulation and fibrointegration due to surgical trauma. Biological failures again divided into early and late failures. Early failures are associated with dental implant infection as result of contaminated surgical placement or impaired host healing.(5, 6) Late dental implant infections generally occur due to periimplantitis (PI) which is considered as one of the most common causes of implant failure.(7,8) It is defined as an inflammatory condition characterized by loss of supporting bone in the tissues surrounding the implant.(9,10) The term peri-implantitis first appeared in the literature in 1987 in a study by Mombelli et al. It was described as an infectious disease with many features common to periodontitis. (11)

This review gives an insight into the understanding of peri-implantitis and its various treatment modalities.

Peri-implant mucositisvs Peri-implantitis

The term peri-implant mucositisdiffers from peri-implantitis and is defined as "the presence of inflammation of the per-implant mucosa without loss of supporting bone whereas supporting bone loss in addition to the inflammation of the mucosa is present inperi-implantitis". (12) Several classifications describing the various stages of peri-implant diseases are present.(13,14)

Recently an attempt was made to unify the concepts of peri-implant mucositis and peri-implantitis within the same classification. (15)

MATERIALS AND METHOD

Thirty sound human premolars extracted due to periodontal or orthodontic reasons were selected for the study. Teeth with intact buccal/lingual surfaces, which were free from restoration and fluorosis were included and teeth with caries present on the buccal and lingual surfaces, or presence of white spot lesions, hypo plastic or hypo mineralized teeth and teeth with any kind of developmental anomaly were excluded from the study. They were cleaned of calculus, soft tissue and debris and were stored in saline at room temperature. Standardized rectangular cavities (5×3×1.5mm) were prepared on the cervical one-third on the buccal surfaces. All cavity preparations were done by a round bur in a high speed airotor with water coolant followed by straight fissure. All the prepared teeth were bonded by a seventh generation, self etching, single bottle bonding agent, Xeno V (Dentsply) applied according to the manufacturer's instructions and was light-cured for 20 seconds. Thereafter all the samples were randomly divided into three groups of 10 samples each depending upon the different placement techniques used.

In Group I, the bulk placement technique was utilized wherein the entire cavity was filled with composite and then light-cured.

In Group II, the incremental technique was utilized where the first increment was placed to fill half of the cavity, which was 0.75mm in thickness and then light cured. Similarly the second increment filled the rest of the cavity and was light cured.

In Group III, the split horizontal incremental technique was used where in a 0.75 mm thick increment was applied to fill the cavity. Prior to light curing, two diagonal cuts were made in this increment using a blunt filling instrument. This cut extended through the entire thickness of the restoration, splitting it into four triangular shaped flat portions and was then followed by light curing. The two diagonal cuts were then filled completely and were light cured. The second horizontal increment of 0.75mm thickness was then placed to cover the first light cured increment.

In all the groups, cavities were restored with composite, Ceram-X (Dentsply) with different placement techniques and were light cured for 20 seconds.

All the specimens were thermocycled between 5 to 55 + 2°C for 100 cycles and were then placed into acrylic blocks, wherein the lingual surface of the tooth was placed inside the acrylic thus exposing the buccal surface of the tooth. The teeth were later tested for resistance to fracture with a universal Testing Machine (INSTRON). A 3mm diameter steel sphere applied force on the restoration at a cross head speed of 0.5 mm/min until fracture occurred. All the values were then statistically analysed.

RESULTS

Data was subjected to statistical analysis using SPSS version 19. The p-value less than 0.05 is considered as significant and ANOVA with Bonferroni multiple comparison test at 95% confidence was used for fracture resistance of composite restoration

Table 1 shows the mean force values in all the groups. Group III showed the highest mean force value 638.40 N, followed by Group II 552.40N and Group I with the least mean force value of 483.00 N.

Staging	Defintiion
Stage 0A	PPD ≤ 4 mm and BOP and /or SUP , with no signs of loss of supporting bone remodeling during healing
Stage 0B	PPD > 4mm and BOP and /or SUP , with no signs of loss of supporting bone remodeling during healing

Proposed classification of peri-implant mucositis[15]

On applying ANOVA within the various groups p-value of 0.000 was calculated which was statistically significant. (Table 2; Figure 1)

Etiopathogenesis

The etiology and clinical features of human periodontitis and peri-implantitisare similar but research has shown that they differ from a histopathologic point of view and tissue destruction is faster and more extensive around implants as compared to natural teeth.(16) Successful osseointegration is due to the equilibrium between the biomechanics and host – parasite interaction. Disturbance in any of these will result in implant failure. The osseointegrated implants and the surrounding bone constitute a single functional unitwhich bear the various masticatory loads. Implants like natural teeth are subjected to load cycles during mastication and swallowing and these have been calculated as 2500-8000 i.e. 1-3 million cycles per year. Repeated loading leads to the development of fatigue which eventually will lead to a fibrous integration with the implant and hence failure.(17) Dental plaque is considered to be the main etiological agent for the causation of peri-implant disease. Various studies have concluded that the micro-organisms found in peri-implant disease includeAggregatibacter (formally Actinobacillus) actinomycetemcomitans, Porphyromonasgingivalis, Tannerella forsythia, Trepone madenticolaand Campylobacter rectus.(18)

Proposed classification of peri-implantitis[15]

Staging	Defintiion
Stage I	BoP and/or SUP and bone loss ≤ 3mm beyond biologic bone remodeling.
Stage II	BoP and/or SUP and bone loss >3mm and < 5mm beyond biologic bone remodeling.
Stage III	BoP and/or SUP and bone loss ≥ 5mm beyond biologic bone remodeling
Stage IV	BoP and/or SUP and bone loss ≥ 50% of the mplant length beyond biologic bone remodeling

PPD = probing pocket depth ;BoP = bleeding on probing ; SUP = suppuration

Incidence and Prevalence

It has been suggested that although the hazard rates for implant loss decrease with time, the incidence of peri-implant disease seems to increase with time, possibly leading to a later increase in implant loss. Various studies have shown a time-dependent increase in the incidence of probable pockets around implants which suggest that at least in periodontal patients, marginal bone loss and periodontal pockets seem to increase with time. Ellegaard et al.(19) carried out a retrospective study to report the incidence of marginal bone loss during the initial 24 months after insertion of the suprastructure in periodontal patients requiring additional tooth support. The incidence of bone loss increased during the following 3 years, with 45% of all implants displaying marginal bone loss of 1.5 mm or more. Koldsland OC (2010) [20] evaluated the prevalence of peri-implant disease amongst 49 subjects treated at the University of Oslo with 104 implants and concluded that 47.1% of subjects and 36.6% of implants were either diagnosed with peri-implant mucositis or peri-implantitis.

Treatment Modalities

According to the saying "Prevention is better than cure", multipartite prevention techniques(21) have been tried which include controlling the various aspects of the type and structure of the implant surface , continuous checkups , evaluation and elimination of risk factors (e.g., smoking, systemic diseases and periodontitis, etc.). Various conservative and surgical approaches are available for the treatment of peri-implant diseases (mucositis and PI). Along with maintaining adequate plaque control, conservative methods include administration of systemic and local antibiotics alone or in combination with other treatment modalities like non surgical therapy (mechanical debridement of the affected areas, irrigation with antiseptics

such as chlorhexidine, saline, and 10% hydrogen peroxide with or without surface decontamination, laser-supported therapy, photodynamic therapy (PDT) as well as light-activated disinfection (LAD) also known as photodynamic antimicrobial chemotherapy (PACT) can be used for treating mucositis and moderate forms of PI.(22) Rather than conservative approaches surgical therapies are found to be more effective and these include : open flap debridement, regenerative therapies such as guided tissue regenerative, bone graft materials for defect filling depending on the configuration of the defect and resective surgery. The above-mentioned strategies are encompassed in cumulative interceptive supportive therapy which serves as a set of guidelines for the treatment of PI. In cases with advanced PI, surgical therapies are more effective than conservative approaches. Open flap debridement can be done, and depending on the con guration of the defect, regenerative therapies such as guided tissue regenerative and the use of bone graft materials may be applicable for defect flling whereas resective surgery can be carried out for the elimination of peri-implant lesions. The above-mentioned strategies are encompassed in cumulative interceptive supportive therapy which serves as a set of guidelines for the treatment of PI.(23)

Summary and Conclusion

Dental implants have revolutionlised treatment concepts in modern dentistry. Implant failure (early or late) can occur even in most successful cases of osseointegration. Peri-implantitis is the most common late failure cause of dental implants and is considered to be a "ticking time bomb". Even though the etiology and pathogenesis of human periodontitis and peri-implantitis is similar tissue distruction is faster around implants than around natural teeth. It is best to prevent peri-implantits before it occurs and for this various treatment modalities are available. These include various conservative as well as surgical techniques. However long term studies and research are needed in order to prevent and treat peri-implant disease at its inception.

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