Commentary

The Case for Routine Maintenance of Dental Implants

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The large majority of dental implants are successful over the long term. Failure is usually associated with infection, trauma, inflammation, or a combination of these factors. Early identification and appropriate treatment can identify and eliminate these problems in the majority of cases. Thus routine implant maintenance structured along the guidelines for patients with periodontal diseases is recommended. J Periodontol 2014;85:657-660.

KEY WORDS

Dental cement; dental implants; foreign body reaction; inflammation; maintenance; peri-implantitis.

The vast majority of dental implants remain successfully integrated over long periods. However, failure does occur. One recent publication estimated that 5 to 10 years after placement 10% of the implants in 20% of the patients would suffer from peri-implantitis.[1] Although this estimate is somewhat clouded because of the lack of a standardized definition for peri-implantitis, it signals to the profession that methods for prevention and treatment of these disease entities must be addressed.

ETIOLOGY

We are in the early stages of understanding which factors contribute to peri-implant disease; however, numerous studies from multiple sources are beginning to shed light on the etiology of this problem.[2] What follows is an overview of possible causative factors and how the need for routine evaluation of these factors argues for periodic maintenance.

Peri-implant disease can be separated into two distinct phases, peri-implant mucositis and peri-implantitis.[3] The former is inflammation of the peri-implant soft tissues, whereas the latter is defined as progressive bone loss associated with clinical signs of inflammation.

The Role of Biofilms

At present many assume that the primary extrinsic etiology of peri-implant disease is bacteria and that peri-implant mucositis, untreated, can progress to peri-implantitis.[4] Many of the clinical and histologic features of peri-implant disease mimic those associated with periodontal lesions. If one accepts these assumptions concerning the pathophysiology of peri-implant disease, an argument can be made for routine monitoring and appropriate maintenance care for these devices.

Luting Agents

Peri-implant disease has been related to excess dental cement. This is of concern because the vast majority of implant-supported fixed partial dentures are cemented.[5] This excess cement has been shown to be associated with peri-implant disease.[6] A study on excess cement used a dental endoscope to evaluate consecutive implants found to have clinical

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and/or radiographic signs of peri-implant disease.[6] Implants in the same patients with no signs of inflammation or progressive bone loss served as controls. Eighty-one percent of the implants with peri-implant disease were associated with excess cement. There was no excess cement found in any of the control implants. The test implants were then cleaned using the endoscope and reevaluated for clinical signs of inflammation at 30 days. Seventy-six percent of those test implants showed no further signs of peri-implant disease at reevaluation. Three of the non-responding patients required surgical intervention to remove the remaining particles of cement. One of the most disturbing findings of the study was that the minimal time between crown cementation and the onset of clinical or radiographic signs of peri-implant disease was 4 months, while the maximum interval was 9.5 years. Thus routine observation of cemented implants can be justified because of the delay between cementation and the appearance of peri-implant disease associated with excess cement.

Foreign Bodies in Soft Tissue

In an unpublished companion study, dental cement and titanium debris have been identified in biopsies taken around implants in humans with peri-implant disease.[7] Exactly how these materials found their way into the soft tissues is still in question. It can be assumed that the cement particles are a byproduct of cementation of the overlying fixed partial dentures. Their introduction into the soft tissues may occur at the time of cementation or at a later secondary attempt to remove excess subgingival luting material. These fragments may induce foreign body reactions as a result of bacterial infiltration before their fragmentation, or the response may be a hypersensitivity reaction.

The genesis of the apparent titanium particle generation or accumulation is less obvious. Such particles may be dislodged by mechanical forces during implant placement, related to occlusal function, or result from the use of certain types of abutment material such as zirconia. Others have suggested that this results from a foreign body reaction.[8] In vitro studies have shown a greater rate of wear of the internal surface of the titanium screw with zirconia abutments compared with titanium abutments, from which titanium debris could be released into the soft tissues, initiating an inflammatory response.[9,10] Reports of titanium particle generation from the orthopedic literature indicate that these particles can be generated by a combination of an acidic medium and micromotion.[11] This combination may induce breakdown of the titanium oxide barrier that protects against metallic corrosion. The lower pH can be generated by bacteria as well as certain chemicals. The conjoined effect of cyclic loading and implant micromotion along with acidic environment results in permanent damage of the oxide film. This sequence of events can lead to exposure of the bulk metal and to active oxidation (corrosion).[12] This is of particular relevance because to achieve new bone-to-implant contact, it is apparently necessary to have a layer of titanium oxide.[2] The process of reintegration may be impeded by corrosion seen on the surface. In addition, it is understood that in some circumstances excessive occlusal forces can lead to mechanical failure of the implant and/or its prosthesis. These findings strengthen the rationale for periodic evaluation of the patient's personal oral hygiene as well as the occlusal forces on the implant superstructure.

THE RATIONALE FOR MAINTENANCE

Given the nature, prevalence, and often delayed onset of peri-implant disease, it seems prudent to recommend routine maintenance therapy for patients with dental implants. Current approaches to implant maintenance are somewhat haphazard and not standardized. It is therefore suggested that information gathered at maintenance visits for patients with implants be modeled on the data gathered for individuals with periodontal diseases. This would include accurate baseline and follow-up radiographs, probing, monitoring the occlusion, and evaluating and modifying personal oral hygiene where applicable, along with early intervention for those individuals with peri-implant disease. Because the prevalence of peri-implant disease is greater in individuals with periodontitis, these patients should be seen more frequently than individuals without periodontal disease.[13]

Treating Peri-Implant Mucositis

Signs of peri-implant mucositis are not unusual around implants. The most common signs include color changes and bleeding on probing. When these signs are present, curettage and exploration of the peri-implant tissues is prudent, along with scaling of the subgingival portions of the implant and its superstructure using a metallic instrument. Screw-retained prostheses should be checked for possible loosening. Evaluation of personal oral hygiene and appropriate behavioral modification should also be an important part of these visits. For these patients, a reevaluation should be performed ≈30 days after treatment. If no clinical signs of inflammation (peri-implant or periodontal) are found, the patient is normally scheduled for a reevaluation in 1 year. For individuals with periodontal disease, the maintenance interval is determined by the relative health or disease of the periodontium. If clinical signs of inflammation still exist at the reevaluation appointment and the crown has been cemented, then whatever steps are necessary to rule out excess cement should

be performed. For non-cemented crowns with clinical signs of peri-implant mucositis but no signs of periodontitis, the area is debrided again, and the patient is reevaluated at 3 months.

Treating Peri-Implantitis

The treatment of peri-implantitis often involves surgical therapy. If suppuration, increased probing depths, or increased bone loss seen on radiographs are present, it is suggested that intervention be accomplished as soon as feasible. For non-cemented restorations with secure crowns, initial intervention can be facilitated by use of the dental endoscope or videoscope. If these devices are not available, closed procedures (e.g., scaling, soft tissue curettage) are suggested. A reevaluation of these cases is normally accomplished at 3 months.

For cemented restorations presenting with symptoms of peri-implantitis, the use of either an endoscope or videoscope is suggested. If these devices are not available, surgical intervention is usually warranted. The goal is to remove any remaining cement particles adhering to the implant or superstructure or in the soft tissues.

New bony attachment on implant surfaces previously coated with biofilm has proven to be unpredictable.[14] A number of approaches to detoxifying implant surfaces during surgery have been suggested. Mechanical methods such as titanium brushes, air powder abrasives, and periodontal scalers have been used. Chemical means including chlorhexidine, sterile saline, EDTA, hydrogen peroxide, citric acid, and tetracycline have been discussed in the literature. Other approaches including the use of lasers of various types along with photodynamic therapy have been used in an attempt to halt the disease process. To date, to our knowledge, predictable re-osseointegration has not been shown with any of these methods. Indeed, chemicals that are in the acidic pH range such as citric acid and tetracycline may in fact result in increased corrosion of the implant surfaces. The most predictable way to stop the progress of peri-implantitis is to minimize surface roughness of the implant (implantoplasty). This approach has obvious drawbacks, including the probability of gingival recession and minimal if any re-osseointegration.

We are left with a number of unanswered questions, including: 1) Which treatment or combination of treatments is optimal? 2) How will chemicals affect the surfaces in the long term? 3) What graft materials, if any, are optimal for regeneration?

CONCLUSIONS

Major causes for implant failure are infection associated with biofilm, inflammation and occlusal loading. Other factors, including allergic reactions to dental materials, also play a role in some cases. In a number of cases, excess cement has been shown to play a role in the initiation of peri-implant inflammation, whereas its removal eliminates the progression of bone loss in most cases. Macromotion from occlusal forces can result in mechanical failure of the implant or its superstructure, while micromotion from these same forces may contribute to the disease process by causing corrosion and release of particles of titanium into the surrounding soft tissues. Many cases with these problems progress slowly, at least initially. Evidence gathered to date indicates that when these problems are identified and treated in their early stages, the progression of bone loss can be arrested or ameliorated. Thus the recommendation for routine maintenance of implants appears justified.

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