

Periimplantitis. Aetiology, diagnosis, treatment.

A review from the literature

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ABSTRACT Orofacial rehabilitation through the use of implants offers very high success rates. In this paper, we describe some of the complications involved with this technique, such as periimplant disease and, within this category, periimplantitis, an inflammatory reaction in which there is a loss of the bony support of the implant accompanied by inflammation. The aetiology of the disease is conditioned by the status of the tissue surrounding the implant, implant design, degree of roughness, external morphology and excessive mechanical load. The microorganisms most commonly associated with implant failure are spirochetes and mobile forms of Gram-negative anaerobes, unless the origin is the result of simple mechanical overload. Diagnosis is based on changes of colour in the gum, bleeding and probing depth of periimplant pockets, suppuration, x-ray and gradual loss of bone height around the tooth. Treatment will differ depending upon whether it is a case of mucositis or periimplantitis. Therapeutic objectives focus on correcting technical defects by means of surgery and decontamination techniques (abrasion with carbon particles, citric acid solution, topical tetracycline application and laser surgery).

KEY WORDS *periimplantitis, aetiology, diagnosis, therapy*

Introduction

Implant-based dental rehabilitation techniques has come to offer highly predictable results, hence it has become one more element to be included in the wide range of therapeutic alternatives for totally or partially edentulous patients, albeit some complications have been described in relation with this type of treatment; of these complications, the progressive loss of alveolar bone surrounding the implant is perhaps the most salient.

The name periimplant disease refers to the pathological inflammatory changes that take place in the tissue surrounding a loadbearing implant (15); for some authors it is the most common complication in orofacial implantology (8).

Two entities are described within the concept of periimplant disease:

- Mucositis: a clinical manifestation characterised by the appearance of inflammatory changes restricted to the periimplant mucosa. If treated properly, it is a reversible process (11).

- Periimplantitis: a clinical manifestation where clinically and radiologically evident loss of the bony support for the implant occurs, together with an inflammatory reaction of the periimplant mucosa (4).

Osseointegration is defined as the direct connection between live bone and a functioning endosseous implant, the term "functioning" implying that the contact between live bone and the surface of the implant is sustained while active or load-bearing (5). This point must also be emphasised when referring to periimplantitis; the implant must be a "functioning" one, because this implies that all other inflammatory syndromes that

course with loss of osseointegration, but that present in implants that do not support the forces transmitted to them by the prosthesis to which they are attached, have been ruled out.

Examples of non-integration or the loss of osseointegration that cannot be considered periimplantitis include processes that appear during the theoretical period of passive osseointegration. They are usually the consequence of poor surgical technique (overheating of the bone) or insufficient trabecular bone density in the receptor (10).

The so-called apical periimplantitis in which the periimplant infection is located in the apical region of the implant, would also be excluded from the disease category of periimplantitis. It may also be the result of implant contamination by epithelial rests of Malassez that remain within the bone despite proper alveolar scaling following extraction of the tooth to be replaced by the implant (16, 18, 20).

Etiopathogeny of periimplantitis

Periimplant tissue morphology. Healthy periimplant tissue plays an important role as a biological barrier to some of the agents that cause periimplant disease.

The epithelium and the interface between the supralveolar connective tissue and the titanium surface of an implant differ from the interface of the dental-gingival unit. Like the connective tissue attachment, the epithelium presents a hemidesmosomal attachment to the implant surface; the difference lies in the fact that the

epithelial fibres are predominantly longitudinal to the surface of the implant and not perpendicular, as in the case of a natural tooth. In the most coronal region, they are circumferential, in addition to presenting a low degree of vascularisation and a higher collagen fibre to fibroblast ratio in comparison to the tooth (a ratio of 4 in a tooth to 109 in the implant) (2). This attachment is fairly weak, so that if destroyed, bacterial contamination spreads directly to the bone, leading to its rapid destruction.

In animal studies, some authors have demonstrated that following removal of the periimplant ligature that caused the inflammation in that area initially, these inflamed areas become encapsulated over time, leaving a "non-aggressive residual lesion" separate from the bony tissue surrounding the implant that would limit the progress of the destructive inflammatory symptoms (14).

Implant structure. The design of the implant is an important factor in the onset and development of periimplantitis. Poor alignment of the components that comprise an implant prosthesis system may foster the retention of bacterial plaque, as well as enabling microorganisms to pass inside the transepithelial abutment. As Binon et al described in their study, this is possible because on average, there is a difference of between 20 and 49 micra between the components of the different types of implants currently on the market (3). This space provides a point of entry for microorganisms of the oral flora measuring less than 10 micra.

The external morphology of the titanium implant seems to be less relevant provided that it has been properly placed. However, the influence of the macroscopic design should be taken into account in terms of the pattern of stress transmission to the bone, which can lead to excessive mechanical stress at certain points, particularly at the junction between the bone and the cervical collar of the implant. Bone loss at this biomechanically weakspot increases the likelihood of bone defect formation at this level and subsequently becoming contaminated. Another reported cause of periimplantitis is the corrosion that can occur when a non-noble metal structure is connected to a titanium implant. In these cases, increased amounts of macrophages have been observed in the tissues surrounding the implant; which would favour the initial bony reabsorption due to non-infectious causes (17).

Microbial infection

Another cause of periimplantitis, as previously mentioned, is the bacterial colonization of the

periimplant pocket. The association between different microorganisms and destructive periodontal or periimplant disease is governed by the same biological parameters.

The microorganisms most commonly related to the failure of an implant are the Gram-negative anaerobes, like *Prevotella intermedia*, *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Bacterioides forsythus*, *Treponema denticola*, *Prevotella nigrescens*, *Peptostreptococcus micros* and *Fusobacterium nucleatum* (9, 21). This is very common in the case of partially edentulous patients with active periodontal disease and who also bear implants. In this group of patients, colonisation of the periimplant sulcus by these microorganisms is observed one month following connection of the implant to its prosthetic abutment. Said colonisation does not necessarily imply that periimplantitis will develop with the subsequent rapid loss of bone height, hence, it is suggested that, in addition to the presence of these periodontal disease-causing germs, other local, systemic and genetic factors must coexist in order for prolonged, active infection to actually take place. The most remarkable of these factors include poorly controlled diabetes, long term treatment with corticoids, radiation and chemotherapy, smoking and excessive mechanical stresses on the implant. It should be pointed out that patients with severe periodontal disease, who eventually become totally edentulous and receive an implant, do not present *Actinobacillus actinomycetemcomitans* or *Porphyromonas gingivalis* in the first month following extraction, thereby confirming the observation that the presence of these pathogenic organisms depends on the existence of the periodontal sulcus. Decreased levels of spirochetes and *Streptococcus mutans* and sanguis are also observed, confirming that the tooth acts as a reservoir that facilitates the spread of certain pathogenic germs. It is very important to point out that when the initial mechanism involved in the loss of bone height is attributable to a simple excess of mechanical forces, the microorganisms detected in the culture of a sample collected from the peri-implant pocket do not correspond to those of a patient with teeth who presents with active periodontal disease (19).

Excessive mechanical stress. Another factor that intervenes in periimplantitis aetiopathogeny is excessive mechanical stress. The process begins with the appearance of microfractures of the bone around an osseointegrated implant, as a result of being subjected to axial or lateral stresses that are excessive for its load-bearing capacity. On

occasions, these forces cause a prosthetic component (resin, ceramic or the transepithelial abutment screw) or the implant itself to fracture, without any loss of bone height or osseointegration whatsoever.

An implant does not tolerate lateral stresses as well as a natural tooth does because, amongst other reasons, it has only half the resilience of a tooth (100 micra in the case of a natural tooth, 50 micra in the case of an implant) and implants do not allow for the same degree of proprioception. In any event, this factor takes on particular relevance when bone quality is poor. This might explain why it presents more frequently in the maxillary bone as opposed to the mandible (19).

Excessive mechanical stress can be the consequence of:

- Occlusal load factors: when the implant is subjected to excessive stress due to the presence of natural teeth in the opposing arch or because the patient has some form of parafunction, particularly bruxism.

- Treatment regime: due to poor implant distribution (they are too close to each other) or too few implants being placed in the patient.

- Prosthesis-related factors: as a result of cantilevers in the prosthesis (fixed or bar-type structure), either mesial or distal, or in an anterior position, which often occurs in teeth in the incisal area of the maxillary bone. It might be also included here the improper relationship between crown length and implant length and the lack of passive adjustment of the prosthetic structure, which generates stresses when screwed onto the implants (19). It is important to bear in mind that periimplant bone loss must be considered to be due to a host of factors and that both bacterial infection and excessive mechanical stresses contribute to the problem, albeit there is currently no evidence available to indicate which of the two is the trigger mechanism. In any case, when there is poor bone quality in addition to one or more of the afore mentioned risk factors, the effect in terms of implant loss is exponential.

Diagnosis of periimplantitis

Periimplantitis can be diagnosed early or once clear clinical evidence has developed. The most common signs and symptoms are:

- Colour changes in keratinised gum tissue or in the oral mucosa.
- Bleeding on probing.
- Increased probing depth of periimplant pockets.
- Suppuration.
- Periimplant radiotransparency.

- Progressive loss of bone height around the implant.

The absence of bleeding on probing is indicative of good health. Probing depth depends on the force applied, so that when equal amounts of force are exerted, the depth reached by the probe is greater in periimplantitis than in the case of a natural tooth. It is recommended the use of probes calibrated to a force of 0.25 n (25 g) to avoid test errors. At any rate, a pocket larger than 5 mm is deemed to have a greater likelihood of being contaminated.

On x-ray, the problem can be detected once 30% of the bone mass has been lost, hence this is not an optimal method for early diagnosis of periimplantitis. Improperly performed x-rays can also lead to errors in determining the size and morphology of the bony defect, in addition to the fact that they do not record defects at the level of the vestibular cortex (1).

When bone loss is due to infection, Gram-negative bacteria, suppuration, increased depth and bleeding on probing, higher gingival and plaque indices, pain on chewing and the presence of granulation tissue surrounding the implant are all detected. However, when bone loss is due to excessive biomechanical forces, initially Gram-negative, nonmobile microorganisms are absent and on x-ray, the periimplant space appears widened and a loss of bone height is observed without signs of suppuration or remarkable signs of inflammation and the implant is encapsulated within fibrous tissue, with little granulation tissue.

Analysis of the fluid in the periimplant sulcus reveals certain early changes that demonstrate the existence of bone resorption, for instance, increased levels of chondroitin sulfate, as seen

in non-treated chronic gum diseases or in patients undergoing orthodontic treatment. Elastase, β -glucuronidase, aminotransferase and prostaglandin E2 levels are also high.

Traditional culture methods are capable of identifying the colonising germs. Another useful method is BANA (benzoyl-arginine-naphthylamide) hydrolysis, which shows the presence of the enzyme trypsin that is produced by pathogens such as *Treponema denticola*, *Bacterioides*

Forsythus and *Porphyromonas gingivalis* (1).

Recording of gingival temperature and peri-implant fluid volume are other testing procedures that have been acknowledged to be valid for the early detection of periimplantitis ; both parameters are elevated in the presence of periimplantitis.

Peri-implantitis treatment

Superficial implant irregularities impede suitable mechanical control of the bacterial deposits located on the exposed implant surface. Optimal treatment for these failed implants should also include the regeneration of the tissue that has been lost around the implant.

The treatment protocol will differ depending on whether it is mucositis or periimplantitis. If there is no bone loss, i.e. in the case of mucositis, bacterial plaque and calculi should be removed and chemical plaque control is achieved with 0.12% chlorhexidine applied topically, every 8-12 hours for 15 days; the patient must also be instructed as to how he/ she can improve oral hygiene. Prosthetic design should also be checked and modified if necessary, in order to correct design defects that impeded proper hygiene, as well as to correct the previously mentioned biomechanical stress factors involved. Once this initial phase is completed, periodic check-ups must be scheduled, gradually reducing the interval between maintenance visits (7).

If periimplantitis is diagnosed, treatment will depend on the amount of bone lost and the aesthetic impact of the implant in question. If bone loss is at an incipient stage, treatment will be identical to that prescribed for mucositis, with the addition of decontamination of the prosthetic abutments and antibiotics. If bone loss is advanced or persists despite initial treatment, it will be necessary to surgically debride the soft, periimplant tissues affected by the chronic infection, decontaminate the microimplant surface and, finally, apply bone regeneration techniques aimed at recovering the lost bone.

Recommended surgical techniques will be performed on the basis of the morphology and size of the periimplant lesion. The apical replacement flap and resection techniques are the first-line treatment of horizontal bone loss and moderate bone defects (smaller than 3 mm), in order to decrease pocket depth and ensure better oral hygiene. Reduction of bacterial plaques ability to adhere to the implant surface is also indicated; this can be achieved by smoothing and polishing rough surfaces or eliminating threads on implants. This technique is known as implantoplasty (7).

Guided bone regeneration techniques are recommended when bone loss is severe or when the implant is strategically important to conserving the prosthesis or if it occupies an aesthetically compromised site. The implant surface must be previously decontaminated in order to enable bone regeneration to take place, but also to permit the implant to osseointegrate

again. The type of implant surface will determine the method of decontamination to be applied (7).

Dennison et al carried out an in vitro study of the relationship between implant surface and decontamination technique, in which the decontaminating efficacy of air-power abrasives, citric acid solution, hydrogen peroxide and chlorhexidine on different implant surfaces (hydroxyapatite, titanium plasma and machined titanium) was assessed (6). They coincided with Zablotsky et al. in their conclusions that air abrasion, using bicarbonate particles with saline solution is the best way to eliminate endotoxins and remains from all surfaces, and that 40% citric acid with a pH of 1 for 30-60 seconds is an effective means of decontamination for hydroxyapatite coated implants; chlorhexidine is not effective in these cases (22). They also determined that machined titanium surfaces are the easiest to decontaminate and that topical tetracyclines (the content of one 250-mg capsule mixed with saline serum until a creamy consistency is obtained) are the antibiotic of choice in these cases. Furthermore, it appears that tetracycline stimulates fibroblast growth in the affected area (22)

Prolonged application times of citric acid solution are not recommended for use on HA surfaces, since this would alter the quality and impair its ability to bond to the titanium body of the implant. Once the application time has transpired, the treated surface must be abundantly irrigated. If the HA is already damaged due to the virulence of the infection surrounding the implant, the recommended approach is to eliminate it completely by drilling and then proceed to apply air abrasion or ultrasound and subsequently decontaminate the area with tetracycline in the same fashion as if it were a machined titanium surface.

In the bibliography of efficacy studies of surgical laser as a method of decontamination on different implant surfaces depending on power intensities, bacteria kill rates of up to 99.4% have been attained(13). The semiconductor 809-nm, the CO₂ and Er:YAG lasers are recommended, since it appears that they do not exert a negative impact on the implant surface(14).

The recommended oral antibiotic treatments consists of: amoxicillin, amoxicillin plus clavulanic acid, amoxicillin associated with metronidazole or, in the case of penicillin-allergic patients, erythromycin and tetracyclines. The standard treatment time is between 7 and 10 days (22).

Conclusions

Most of the factors that lead to implant failure can be controlled by the dentist by means of proper treatment planning prior to implant surgery. The number, diameter and location of the implants depending upon patient bone type and the type of prosthesis to be inserted, are all factors that are clearly within our control.

Patients undergoing chronic corticoid therapy, poorly controlled diabetics, smokers, those who present active periodontal disease and individuals with serious systemic pathology or predisposing genetic factors should be considered high-risk cases.

Prognosis of the affected implant will be contingent upon early detection and treatment of mucositis and periimplantitis.

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