Chapter 9

Biological complications with dental implants: their prevention, diagnosis and treatment


Biofilms form on all hard non-shedding surfaces in a fluid system, i.e., both on teeth and oral implants. As a result of the bacterial challenge, the host responds by mounting a defence mechanism leading to inflammation of the soft tissues. In the dento-gingival unit, this results in the well-described lesion of gingivitis. In the implanto-mucosal unit, this inflammation is termed “mucositis”. If plaque is allowed to accumulate for prolonged periods of time, experimental research has demonstrated that “mucositis” may develop into “periimplantitis” affecting the periimplant supporting bone circumferentially. Although the bony support may be lost coronally, the implant still remains osseointegrated and hence, clinically stable. This is the reason why mobility represents an insensitive, but specific diagnostic feature of “periimplantitis”. More sensitive and more reliable parameters of developing and existing periimplant infections are “bleeding on probing”, “probing depths” and radiographic interpretation of conventional or subtraction radiographs. Depending on the diagnosis made continuously during recall visits, a maintenance system termed Cumulative Interceptive Supportive Therapy (CIST) has been proposed.

Rationale

All implants may be subject to mechanical complications. Since these are dependent on technical rather than biological factors, they will be dealt with in a separate report. Although the possibility of the loss of osseointegration due to traumatic forces from occlusal overload is not disregarded (Isidor 1996), the scientific evidence for such processes has not yet been established (Tonetti & Schmid 1994). An analysis of the clinical trials of the ITI® system revealed a very small proportion of failures which seem to be associated with occlusal overload.

Many retrospective and prospective studies on the survival of ITI® dental implants have noted late (defined as occurring following successful prosthetic reconstruction) failures (Buser et al. 1990; Mericske-Stern 1990; ten Bruggenkate et al. 1990; Buser et al. 1991; Mericske-Stern et al. 1994; Versteegh et al. 1995; Buser et al. 1997; Ellegaard et al. 1997).
From these studies the following general statements may be made:

- The success rate with ITI® dental implants after 5 to 8 years matched or exceeded the success rates reported for other dental implant systems. The major cause of late failures should be attributed to periimplant infections.
- In longer term studies, patients with good oral hygiene tended to keep implants longer. With adequate oral hygiene practices, the presence of keratinized periimplant mucosa appears not to be essential for the maintenance of implant stability.
- In long-term studies of up to 8 years, there was no statistically significant difference between the success rates of shorter implants (8 mm) and those of implants 10 mm and 12 mm in length.
- In the most recent study documenting success between 5 and 8 years, the 4.1 mm diameter solid screw yielded the highest percentage of success.

Although the percentage of biological failures of the ITI® dental implant system is low, the fact that late failures occur establishes the rationale for careful consideration of the etiology, pathogenesis, diagnosis, prevention and management of late complications.

**Etiology and pathogenesis**

**Microbial colonization**

Biofilms will form on all hard, non-shedding surfaces in fluid systems (Gristina 1987). The oral cavity represents a perfect fluid system in which the microbiota present in saliva may colonize on teeth and artificial surfaces following the deposition of a glycoprotein-containing pellicle. In addition, bacteria may accumulate in specific ecological niches which provide optimal ecological conditions for growth and division, such as periodontal pockets, tonsils, and crypts and folds of the tongue. Plaque formation on teeth has been described recently (Mombelli & Lang 1994). On oral implants, plaque formation has been studied using scanning electron microscopy. The results have indicated that formation patterns identified on implants may be identical to those observed on teeth.

**Development of the periimplant microbiota**

**Natural colonization in edentulous patients**

The development of the microbiota in the periimplant sulcus was first studied in edentulous patients using anaerobic culturing techniques (Mombelli et al. 1988). In this study, it was evident that the colonization process of the periimplant sulcus in an edentulous patient originates from the microbiota floating in saliva and is not disturbed by the microbiota residing in already existing gingival sulci or periodontal pockets. Mucosal swab samples were obtained from edentulous ridges prior to the installation of one-stage transmucosal implants. Subsequently, sterile paper point samples were retrieved at weekly intervals for the first 2 months and then at monthly intervals for the next 4 months. Already after 2 weeks, a microbiota in the periimplant sulcus was established which was predominated by gram-positive facultative bacteria closely resembling the microbiota associated with gingival health or gingivitis (Mombelli et al. 1988; Mombelli & Mericske-Stern 1990). In one implant sulcus of a patient with a history of a previous periimplant infection which led to the loss of the implant, however, high proportions of gram-negative anaerobic bacteria and spirochetes were detected after 120 days. Clinically, this microbiota was associated with heavy signs of inflammation and early signs of infection, leading to the antimicrobial treatment of this patient (Mombelli et al. 1988).

**Colonization in partially edentulous patients**

Prospective studies on the colonization of periimplant sulci in partially edentulous patients are sparse. Obviously, residual periodontal pockets harboring high proportions of presumptive periodontal pathogens may influence the colonization of the periimplant sulcus. In a study in which such colonization was analyzed after three and six months following installation of one-stage transmucosal implants in one practice and following abutment connection of two-stage submerged implants in another, it was demonstrated that the same bacteria which were found in residual periodontal pockets also colonized the periimplant sulcus (Mombelli et al. 1995). If periodontal pathogens were identified in pockets they were also detected at implant sites three months later.

**Microbiota associated with periimplant infections**

A number of association studies have identified the microbiota in the periimplant sulcus or pocket with either adjacent healthy or inflamed mucosal tissues. Initially, bacterial morphotypes were identified using electron (Rams & Link 1983) and darkfield microscopy (Rams et al. 1984). Later on, anaerobic bacterial culturing techniques were applied to study the association of the microbiota with different periimplant conditions (Krekeler et al. 1986; Mombelli et al. 1987; Apse et al. 1989; Leonhardt et al. 1992). Basically, it has been established that the microbiota associated with healthy periimplant
tissues or mucositis closely resembles the microbiota associated with gingival health or gingivitis, respectively. On the other hand, the microbiota identified in periimplant infections is almost identical to that encountered in pockets with advanced periodontitis.

Periimplant mucositis

Animal models

De novo plaque formation and its eliciting host response have been studied histologically in a beagle dog model (Berglundh et al. 1992). It was found that the inflammatory infiltrate developing as a result of the bacterial challenge was equal in size regardless of whether it was adjacent to control teeth or to oral implants indicating that the host response to bacterial colonization triggered in gingiva is equal to that of periimplant mucosa.

Human studies

Several investigations have dealt with the local defense mechanisms of the periimplant soft tissue seal and compared them to those of the dento-gingival unit. The production of inflammatory mediators and the expression of cytokines appear to be very similar in these two soft tissue compartments (Tonetti & Schmid 1994). Also, the experimental gingivitis model originally described by Löe et al. (1965) and representing the ultimate proof for a cause-and-effect relationship between bacterial plaque accumulation and developing gingivitis was duplicated with regard to the periimplant situation (Pontoriero et al. 1994). Following a period of six months with meticulous plaque control after abutment connection of a two-stage submerged implant system, patients were asked to discontinue all oral hygiene practices for a period of three weeks. At the end of the three-week period there were no significant differences between any of the clinical parameters assessed at gingival control and periimplant mucosal sites. Both soft tissue compartments yielded increased gingival indices and increased probing depths as a result of increased plaque accumulation, and hence, the cause-and-effect relationship between bacterial plaque and the developing mucositis was convincingly established for oral implants as well (Pontoriero et al. 1996).

Periimplantitis

Experimental studies of periimplant infections cannot be conducted in humans for ethical reasons. The information gathered in this field, therefore, must rely on animal studies. Unfortunately, the results in previous periimplantitis studies have been somewhat conflicting as to the rate and extent of progression of periimplantitis lesions. While a pilot study (Klinge 1991) proposed a slower progression rate of disease at the implant site in comparison with the natural tooth, a series of beagle dog studies (Lindhe et al. 1992) cautioned that periimplant lesions may develop directly in the alveolar bone, whereas periodontitis lesions always seem to yield a supracrestal region with intact periodontal fibers.

Other groups of researchers (Lang et al. 1993; Schou et al. 1993) induced periimplantitis and periodontitis in control teeth by applying plaque-accumulating ligatures and compared the disease process with that induced by natural plaque accumulation. The increase in the clinical parameters such as plaque and gingival indices as well as pocket depth and loss of attachment around teeth exactly paralleled that of the ligated periimplantitis sites. In eight months of ligation, approximately 3.5 mm of attachment was lost, while the implants which were only exposed to natural plaque accumulation did not lose more than 0.5 mm over the same time. The microbiota identified around the ligated teeth also corresponded to that surrounding the ligated implants. Also, the lesions analyzed histologically after 8 months were very similar and represented intrabony defects. Digital subtraction radiography yielded loss in bone density and identified the development of intrabony lesions around ligated teeth as well as around ligated implants, while the bone height and density did not significantly change around the implants with natural plaque accumulation during the experimental period. This, in turn, means that under heavy plaque accumulation and in a time period long enough for the development of infections – lesions may progress into the supporting tissues around implants as they do around teeth. Periimplantitis, however, may not develop in all periimplant sites with mucositis, just as periodontitis may not develop in all sites with gingivitis.

Diagnostic aspects

Mobility

Since periimplant infections represent lesions originating from the marginal periimplant sulcus (Lindhe et al. 1992; Lang et al. 1993; Schou et al. 1993), the bone loss encountered in association with the development of such infections is also observed to be marginal and results in the formation of intrabony defects. This, in turn, means that the implant still remains fully osseointegrated in the apical portion, and hence, an increase in implant mobility cannot be expected. On the other hand,
loss of clinical stability as a result of complete loss of osseointegration would be reflected in a sudden increase in implant mobility. Therefore, increase in clinical mobility represents a highly specific, but not at all sensitive parameter for monitoring clinical stability. Assessment of implant mobility in routine evaluations and clinical monitoring of implants is, therefore, not essential, but when used must always be performed in conjunction with the evaluation of other parameters.

Bleeding on probing

“Bleeding on probing” (BOP) represents a clinical parameter which is defined as the presence of bleeding noticed after the penetration of a periodontal probe into the periimplant sulcus or pocket using gentle force. Obviously, the size (point diameter) of the probe applied as well as the application force should be standardized. For teeth, the probing pressure for this parameter has been determined. In the healthy and normal periodontium, the probing force used is 0.25 N (Lang et al. 1991). The same force is used in a healthy but periodontally reduced dentition (Karayiannis et al. 1991). It is reasonable to use the same probing force for the determination of BOP around oral implants. Hence, standardized probes which produce standardized probing forces may be recommended.

BOP has been studied for its value in predicting future attachment loss around teeth (Lang et al. 1986). While the positive predictive value remained rather low for repeated BOP prevalence in one retrospective (Lang et al. 1986) and two prospective (Lang et al. 1990; Joss et al. 1994) studies (30% or less), the negative predictive value in the same studies reached almost 100%. This showed that absence of BOP is a very reliable indicator for periodontal stability (Lang et al. 1990). Although similar data for oral implants are not yet available, it appears logical to apply these associations to the soft tissue seal around osseointegrated implants. Hence, from a clinical point of view, absence of BOP around implants would indicate healthy periimplant tissues.

Modified Gingival Index (mod GI)

The Gingival Index System (Löe & Silness 1963) has been modified and adapted by Mombelli et al. (1987) for application around oral implants. While the mod GI may very well be used with success to assess the status of health or inflammation in periimplant mucosal tissues, and hence, to indicate mucositis in clinical research, it may be preferable to use BOP for routine clinical documentation.

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Calibration exercises to determine accuracy and repeatability of examiners using BOP should be performed prior to initiating studies in the same manner as for the GI.

Probing depth and “loss of attachment”

Periodontal probing to determine probing depth and the level of periodontal attachment in relation to the cemento-enamel junction (CEJ) is the most widely used clinical parameter in periodontal practice. Again, it appears logical to apply these parameters to the periimplant soft tissue seal. Instead of relating probing depth to the CEJ, examiners may use the implant shoulder, which provides a landmark easy to localize in clinical practice.

Although opinions have been expressed that periimplant probing may sever the soft tissue seal and hence, jeopardize the integrity of an implant, there is no scientific evidence for such concern. On the contrary, it may be assumed that following probing the periimplant epithelial attachment to the titanium surface may be re-established within the course of 2–5 days, as demonstrated for teeth (Taylor & Campbell 1972). Studies to evaluate this are under way.

Christensen et al. (1997) found that clinical probing depth determined by three automatic probing devices yielded slightly higher values around oral implants (approximately 0.5 mm higher) than around healthy contralateral control teeth. Also, the buccal and lingual aspects of oral implants generally scored 0.5–1.0 mm less than the interproximal aspects. Probing depth around oral implants may be system-specific and dependent on access of the probe to the periimplant sulcular region. Hence, different probing depth values may be considered as “normal” in different implant systems. For the ITI® dental implant system, normality associated with healthy periimplant mucosal tissues averaged 3–3.5 mm (Christensen et al. 1997).

The localization of the periodontal probe tip around implants has been studied in different mucosal tissue conditions such as health, mucositis and periimplantitis (Lang et al. 1994). While the probe tip reached and identified the true level of attachment – i.e. the most apical cell of the junctional epithelium within 0.2 mm in health and mucositis – the histological level of attachment was generally determined to be up to 1.2 mm more coronal than measured by clinical probing in periimplantitis sites (Lang et al. 1994). These results confirmed the excellent sealing effect of the soft tissue collar in health and mucositis and the relatively uninhibited penetration to the alveolar crest of the probe in periimplantitis lesions. In another
animal study (Ericsson et al. 1994) in which higher probing forces were used, the probe tip penetration usually went through the epithelial attachment until resistance was met as a result of hitting the alveolar crest.

Since the soft tissue seal inhibited probe tip penetration in healthy and only slightly inflamed peri-implant soft tissues, but did not do so in peri-implantitis, probing around oral implants must be considered as a sensitive and reliable clinical parameter for long-term clinical monitoring of peri-implant mucosal tissues.

Repeated systematic comparisons of probing depth and loss of implant support (“loss of attachment”) in comparison with baseline measurements obtained at the time of prosthetic reconstruction are, therefore, highly recommended.

Radiographic interpretation

Conventional radiography

When using conventional radiographs for the evaluation of implant position in relation to anatomical structures and neighboring teeth, appropriate correction factors have to be considered for different radiographic techniques and positions within the oral cavity. Orthopantomograms generally demand a correction factor of 1:1.3, while periapical dental exposures are to be evaluated with a factor of between 1:1.0 and 1:1.1 depending on exposure geometry and differences in radiographic set-ups and sites. The long-cone parallel technique and positioning devices should be applied.

In evaluating the bony structures adjacent to the implants over long periods of time, conventional radiography is a widely applied technique in clinical practice. However, it should be noted that minor changes in bone morphology in the crestal area may not be revealed until they reach a significant size and shape (Lang & Hill 1977). In this respect, conventional radiography yields a high proportion of false negative findings, and hence has a rather low sensitivity for detecting early pathological and/or remodeling changes (Brägger et al. 1988).

Nevertheless, DIB (the Distance from the Implant Shoulder to the Alveolar Bone crest) represents a reliable radiographic parameter for long-term monitoring in clinical practice (Buser et al. 1990, 1991; Weber et al. 1992) provided that optimal exposure geometry has been achieved. Since the implant shoulder is usually placed 3 mm coronal to the alveolar crest for one-stage transmucosal implants, the difference between the various DIB values has to be considered over time. In two-stage submerged implant systems, however, the landmark to be used as a reference on the implant has to be defined clearly. Usually, the apical termination of the cylindrical part of implant fixtures is used, despite the fact that subcrestal placement utilizing a countersink procedure is recommended for most submerged implant systems.

Conventional radiographs have a low proportion of false positive findings and hence, yield high specificity for the detection of peri-implant bone loss. However, this characteristic limits radiographs to be confirmatory rather than exploratory.

It must be recognized that radiographic evidence of bone-to-implant contact does not imply osseo-integration on a histologic level (Sewerin et al. 1997).

Digital Subtraction Radiography (DSR)

In digitizing radiographs of identical exposure geometry, minute changes in the level and density of the alveolar bone may be revealed by subtracting subsequent images from a baseline radiograph. In doing this, the sensitivity of radiographs may be increased significantly (Brägger et al. 1988). Hence, for clinical research, DSR is highly recommended and has been successfully applied in longitudinal studies (Brägger et al. 1996).

Implications for the ITI® Dental Implant System

ITI implants are readily accessible for clinical probing. However, access to the peri-implant sulcus may be hindered by the prosthetic reconstruction. Since probing depth is a critical parameter for the evaluation of peri-implant mucosal health or disease, clinical monitoring using this parameter is highly recommended. While absolute values for probing depth have to be interpreted in the context of the surgical placement of the implant, increases in probing depth represent an alarming change. It is, therefore, recommended to establish baseline probing values, preferably at the time of the prosthetic reconstruction, against which subsequent probing depths can be compared. Probing depths for conventionally placed ITI implants generally range between 2 and 4 mm under healthy peri-implant soft tissue conditions (Christensen et al. 1997). In sites of esthetic priority, where implants have been placed according to the Esthetic Plus® concept and where mucosal tissues have been conditioned, deeper baseline probing depths may be acceptable. However, increases above these values should also be viewed as a sign of potential pathology.

Prophylactic procedures

Instruction in oral hygiene and patient motivation

It is evident that implant installation represents a series of therapeutic steps within the context of a
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Gingival fit is a requisite. This may best be achieved through the use of screw-retained prefabricated copings, even though clinically acceptable marginal gaps may also be achieved with burn-out caps and well-performed castings which are usually cemented.

Maintenance care

After successful periodontal and implant therapy the patient should be offered a maintenance care program adequately designed to fit his or her individual needs. It is important to ensure recall at regular intervals. This will provide optimal preventive services and facilitate treatment of ongoing or emerging disease processes by providing appropriate supportive therapy.

A recall visit may be divided into four different phases:

- Examination, re-evaluation, diagnosis;
- Motivation, reinsertion, instrumentation;
- Treatment of infected sites;
- Polishing, fluoridation, determining recall interval.

Therapeutic strategies

Cumulative Interceptive Supportive Therapy (CIST)

Depending on the clinical and eventually the radiographic diagnosis, a protocol of therapeutic measures has been designed to head off the development of periimplant lesions. This system is cumulative in nature and includes four steps which should not be used as single procedures, but rather as a sequence of therapeutic procedures with increasing antibacterial potential depending on the severity and extent of the lesion. Diagnosis, therefore, represents a key characteristic of this maintenance care program.

The major clinical parameters to be used have been discussed above and include the assessment of the following (see Table 1):

1. presence or absence of dental plaque;
2. presence or absence of bleeding on gentle probing (BOP);
3. presence or absence of suppuration;
4. periimplant probing depth; and
5. evidence of radiographic bone loss.

Oral implants without evident plaque or calculus adjacent to healthy periimplant tissues – as revealed by absence of BOP, absence of suppuration and probing depth usually not exceeding 3 mm – can be considered clinically stable and do not seem to be currently at risk for periimplant pathology. These implants should be re-evaluated at least annually. Of course, the frequency of and interval be-
between supportive therapy visits should be determined by the patient’s oral health status.

**Mechanical debridement (Supportive therapy protocol A)**

Oral implants with evident plaque or calculus deposits adjacent to only slightly inflamed periimplant tissues (BOP positive), but lacking suppuration and having a probing depth not exceeding 3 mm, are to be subjected to mechanical debridement. While calculus may be chipped off using carbon fiber curettes (Hawe Neos, Bioggio, Switzerland), plaque is removed by means of polishing using rubber cups and polishing paste (e.g. Implantin®, Hawe Neos, Bioggio, Switzerland).

Carbon fiber curettes do not sever the implant surface, but are sharp and strong enough to remove light to moderate calcified deposits on implants. Conventional steel curettes or ultrasonic instruments with metal tips leave severe damage on the implant surface and render it conducive to future plaque accumulation. They should not be used (Matarasso et al. 1996).

Removal of gross amounts of calculus, however, without touching the implant surface, is acceptable.

**Antiseptic treatment (Supportive therapy protocol B)**

In addition to performing supportive therapy protocol A (i.e. mechanical debridement), antiseptic treatment is performed in situations where – in addition to the presence of plaque and BOP – probing depth is increased to 4-5 mm. Suppuration may or may not be present. The antiseptic treatment (protocol B) is performed in conjunction with the mechanical treatment (protocol A). Antiseptic treatment comprises the application of the most potent antiseptic available (Lang & Breex 1986), i.e. chlorhexidine digluconate, either in the form of a daily rinse of 0.1%, 0.12% or 0.2%, or as a gel applied to the site of desired action. Generally, 3-4 weeks of regular administration are necessary to achieve positive treatment results. Antiseptic rinses with chlorhexidine or applications of chlorhexidine gels may also be recommended for chemical plaque control on a preventive basis.

**Antibiotic treatment (Supportive therapy protocol C)**

When probing depth values of the periimplant sulcus or pocket increase to 6 mm or more, plaque deposits and BOP are usually encountered. Suppuration may or may not be present. Such a periimplant lesion is usually radiographically evident, and the pocket represents an ecological niche which is conducive to colonization with gram-negative anaerobic, periodontopathic microorganisms (Mombelli et al. 1987). The antibacterial treatment approach must then include antibiotics to eliminate or at least significantly reduce the pathogens in this submucosal ecosystem. This, in turn, will allow soft tissue healing, as demonstrated in a study by Mombelli & Lang (1992). Prior to administering antibiotics, the mechanical (A) and the antiseptic (B) treatment protocols have to be applied. During the last ten days of the antiseptic treatment, an
antibiotic directed at the elimination of gram-negative anaerobic bacteria – e.g. metronidazole (Flagyl®, Rhône-Poulenc, 3×350 mg daily) or ornidazole (Tiberal®, Roche, 2×500 mg daily) – is administered. These therapeutic steps have been validated in a clinical experimental study (Mombelli & Lang 1992) in which periimplant infections were treated successfully and remained stable for a documented period of one year. Subsequently, prophylactic procedures were instituted to prevent reinfection.

Instead of administration of systemic antibiotics, the application of local antibiotics through the use of controlled delivery devices has emerged recently as a suitable treatment concept. However, only release devices with adequate release kinetics may be used to assure successful clinical outcomes. The antibiotic must remain at the site of action for at least 7–10 days in a concentration high enough to penetrate the submucosal biofilm. As of today, only a limited number of products have been shown to demonstrate the appropriate characteristics.

Tetracycline periodontal fibers (Actisite®, Alza, Palo Alto, CA, USA) have successfully been applied in some case studies. The therapeutic effect appears to be identical to the effect documented for the systemic administration of antibiotics (Mombelli & Lang 1998), provided that the treatment protocols A and B are utilized as well. Hence, it appears that periimplant infections may be controlled successfully by cumulatively providing mechanical, antiseptic and antibiotic supportive therapy.

Regenerative or resective therapy (Supportive therapy protocol D)

Only if infection is controlled successfully, as evidenced by an absence of suppuration and reduced edema, is it reasonable to discuss treatment approaches to either restore the bony support of the implant by means of regenerative techniques or to reshape the periimplant soft tissues and/or bony architecture by means of resective surgical techniques, depending on esthetic considerations and morphological characteristics of the lesion.

So far, single case presentations have provided evidence that bone fill of periimplant defects resulting from previous periimplantitis may be achieved following anti-infective therapy and using the biological principle of guided tissue regeneration (GTR) (Lehmann et al. 1991; Hämmerle et al. 1995; Persson et al. 1996). However, the re-osseointegration of a previously contaminated implant surface into regenerated bone has not yet been demonstrated histologically (Wetzel et al. 1999). Nevertheless, the fact that new bone does fill osseous defects, as documented by an increase in radiographic bone density, represents a healing process most likely resulting in better implant stability over time.

Regarding attempts at local decontamination of the implant surface during surgical exposure, no conclusive evidence identifies one particular approach as most effective. Occasionally, the clinician may find it appropriate to smooth and polish the supra-alveolar portion of the implant.

Explanation

If a previously osseointegrated oral implant is clinically mobile, explanation is mandatory. The periimplant lesion involves the entire length and circumference of the implant. Radiographically, this may be visible in a radiolucency surrounding the entire outline.

Explanation may also be necessary if the periimplant infection has advanced to a degree where it cannot be controlled by the therapeutic protocols proposed above. Such a situation is clinically characterized by the presence of a suppurative exudate, overt BOP, severely increased periimplant probing depth (usually ≥8 mm), eventually reaching perforations or vents of hollow body implants, and may be associated with pain. Radiographically, a periimplant radiolucency may be recognized extending far along the outline of the implant.

Conclusions and clinical implications

Oral implants are anchored in the jawbone and yet penetrate the mucosa, reaching the highly contaminated environment of the oral cavity. There, biofilms forming on all hard, non-shedding surfaces will also form on titanium implants. As on teeth, bacterial plaque will develop and trigger a host response, resulting in the development of mucositis. If plaque is allowed to accumulate over prolonged periods of time, periimplant mucositis may develop into lesions extending further apically, with associated loss of alveolar bone. Angular bony defects usually extending around the entire circumference of the implant may result, and are termed “periimplantitis”.

The periimplant mucositis lesion is characterized by BOP and periimplant sulcus depth of usually 2–4 mm. Periimplantitis, however, yields increasing probing depth, with occasional suppuration and radiographic loss of crestal bone. However, clinical stability is not yet jeopardized, since the implant affected is not mobile as yet. Osseointegration in the apical portion of the implant usually persists.

Owing to the infectious nature of periimplant
mucositis and periimplantitis, preventive procedures have to be rendered in a well-organized recall program to assure adequate supportive therapy for a lifetime. Depending on continuing diagnosis during maintenance, developing periimplant lesions should be treated according to the Cumulative Intercractive Supportive Therapy (CIST) protocols.

CIST includes as a first sequence mechanical, antiseptic and antibiotic treatment to control ongoing infection. Following this, periimplant bony lesions may be corrected by regenerative or resective surgical techniques. It is evident that preventive measures have to be re instituted following such therapy.

References


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