Effect of plaque accumulation and occlusal overload on peri-implant bone loss

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Abstract

This paper presents a case report where the influence of plaque accumulation and overload on a dental implant was observed. The patient presented crown loss on one implant affected by peri-implant bone resorption and gingival inflammation associated with poor oral hygiene. After 12 months a single crown was delivered, but as soon as the occlusal load was applied bone loss and implant failure occurred. In the present case report the dental implant remained stable as long as the implant was not loaded, although a 100% plaque index and bleeding on probing were present during the entire follow-up period. In contrast, as soon as an occlusal load was applied peri-implant bone loss and implant failure occurred. These observations suggest that plaque accumulation alone is not a triggering factor for peri-implant bone loss and implant failure. On the contrary, occlusal load, when not properly controlled, might cause bone resorption.

Introduction

In recent years several studies investigated the etiology of peri-implant disease, the so-called *peri-implantitis*.[1] Triggering factors

for peri-implant bone loss and implant failure are generally gathered under two main categories: biological factors (*e.g.*, presence of an aggressive bacterial strain) and biomechanical factors (*e.g.*, excessive mechanical stress).[2] Plenty of papers focused on these two claimed etiological factors of peri-implant disease, and most of them are animal studies.[1] Dealing with animal studies, it is evident that the literature is not unanimous about peri-implantitis etiology. In fact, studies on a dog model[3] suggested a plaque related etiology. On the contrary, the studies on the monkey model[4] show an overloading etiology or a synergism between plaque accumulation and overloading. The different study design and the different animal model could account for this inconsistency. Moreover, data on animal studies should be used with caution when discussing clinical evidence. In particular, the use of ligatures, in order to induce plaque accumulation on dental implants in animal models, results in a foreign body reaction and induce a destructive process around implants that do not represent clinical reality.[5]

Bone has been demonstrated to be sensitive to loading conditions[6] and several authors consider occlusal load a critical factor influencing the dental implant healing phase and the long-term survival and success of dental implants.[7,8] Nagasawa *et al.*[9] revealed the possibility of bone loss around overloaded implants in the absence of infection using a rat model. These results agree with previous studies realized on monkeys.[4] On the other hand, some animal studies on dogs[3] described overloading as a cofactor in the etiology of peri-implant disease, underlining how in the presence of peri-implant mucosal health, excessive occlusal load on titanium implants did not result in loss of osseointegration or marginal bone loss. In contrast, overloading aggravated the plaque-induced bone resorption when peri-implant inflammation was present. This is confirmed in systematic reviews of animal studies.[10]

As described above, animal studies are not unanimous about peri-implantitis etiology.

Moreover these data should be subjected to careful interpretation when reliable clinical evidence is absent. Overloading research designs used in the animal model do not adequately simulate the human situation. The majority of patients are extremely sensitive to even minute occlusal imperfections in occlusion on implant-supported restorations and would demand an immediate corrective action by the dentist. Moreover, the above-mentioned papers are difficult to compare because of differences in study design.[1]

A recent systematic review[11] of clinical human studies suggested that occlusal overload is positively associated with peri-implant marginal bone loss.

In clinical studies, bruxism or parafunctions were often used as situations where overloading can be manifested and was reported to cause failures.[12] However, a more precise definition of occlusal overload would be required to investigate its role in peri-implant disease.

Some case reports supported the role of overloading in implant failure.[13] In absence of systematic evidence from human

studies, data from case reports might contribute to understand the role of the possible factors influencing the loss of osseointegration of successfully integrated dental implants.

Some clinical studies found that poor oral hygiene is a risk factor for peri-implant disease.[14] These results are in conflict with a split-mouth study[15] which showed that, even though acid-etched titanium surfaces retain more plaque than machined ones, this does not negatively affect peri-implant bone loss and peri-implant soft tissue health.

Up to now, animal studies and clinical trials are not able to identify the etiology of peri-implant bone loss and implant failure. The etiological factors and the definition itself of *peri-implantitis* are discussion topics.[1]

The present paper describes a clinical case where the effect of plaque accumulation and occlusal overload on peri-implant bone loss and implant failure were observed throughout a 2-year period.

Case Report

A 43-year-old male patient came for consultation at the Department of Fixed and Implant Prosthodontics of the University of Genoa on the 21st December 2011 because of pain caused by pulpitis of the teeth 35 and 37. The patient reported a good general health except that he suffered from hiatus hernia and he was in therapy with pantoprazole and calcium carbonate (Gaviscon®; Reckitt Benckiser Group, Slough, UK). He also reported that the last dental visit dated back to more than 1 year before. The clinical examination showed generalized gingival inflammation associated with poor oral hygiene.

The pulpotomy of element 35 and 37 was performed to resolve the pain. The root canal treatment were completed at subsequent appointments. The dental visit also revealed a crown loss on one dental implant (Winsix 3.8 x 11 mm) at the level of the left first molar in the mandible. The implant was placed in 2005 by a private dentist. The patient reported that he had lost the crown on the implant 1 year and a half after implant insertion and that he probably ate the crown itself. A radiographic examination was performed and revealed severe peri-implant bone loss (distal side: 6 mm; mesial side: 6 mm at baseline) (Figure 1A).

The implant was considered failed because of severe bone loss, however it was stable, so the decision was taken not to remove it, in agreement with the patient desire not to perform the extraction. The patient also refused any other therapy for economical reasons. Since then, professional oral hygiene sessions were planned and performed every 6 months by a dental hygienist using carbon fiber curettes at the implant site. The patient also received detailed instructions for home dental care, but his compliance was very low.

The patient was followed-up for 2 years and bone level was evaluated over time. The health of soft and hard peri-implant tissues was controlled by periapical x-rays and periodontal indices: plaque index (PI), bleeding on probing (BOP) and probing depth (PD) were recorded at baseline (T0), and after 6 (T6) and 12 months (T12). The radiographs were taken using a long-cone parallel technique with a film holder (Rinn bite film holder for periapical radiographs; Dentsply, York, PA, USA). Measurements of interproximal bone level were performed using as the reference point the implant platform. Bone level was assessed from this reference point to the most coronal bone-implant contact

BOP and PD were recorded using a plastic probe at 6 points around the implant: mesio-vestibular (MV), central-vestibular (CV), distal-vestibular (DV), mesio-lingual (ML), central-lingual

(CL), distal-lingual (DL). PI was recorded using a plaque detector based on erythrosine at 4 points.

After 12 months (Figure 1B), the extractions of the teeth 45, 46, 47 were performed due to destructive caries. Four months after the extractions (T16) a provisional screw-retained composite crown was realized on the implant (site 36) according to the patient desire to improve mastication on the left side of the mouth. Alternative fixed and removable prosthodontic rehabilitations were proposed but the patient refused them for economical reasons. The provisional crown was 6 mm high (from the centre of the occlusal surface to the crown-implant connection), 12 mm wide from the mesial to the distal surface, and 9 mm wide from the vestibular to the lingual surface (Figures 1C).

Clinical and radiographic examinations were repeated two months after crown delivery (T18).

Eight months after loading, the patient came at the Department because of implant mobility, pain and suppuration at the implant level and the implant was removed (Figure 2). A careful curettage of the implant site was performed and the post-extractive site was filled up with a haemostatic reabsorbable gelatin sponge (Spongostan Dental; Johnson&Johnson, New Brunswick, NJ, USA) without the need for suturing.

The mean values of peri-implant bone loss and periodontal indices over time are reported in Figure 3 and Table 1.

Discussion

This paper reports the effect of plaque accumulation and occlusal overload as observed on one single implant. During the entire follow-up period, the patient presented a PI and BOP of 100% at the level of the implant site. Both at T0 and T12, mean PD and mean peri-implant bone level next to the implant were 7 mm and 6 mm respectively. At T18 they were 8.6 mm and 7 mm respectively (Figure 3, Table 1). At T24 suppuration, pain and implant mobility appeared.

A localized marginal bone loss around this single implant took place. Since bone loss occurred only at this site, and not elsewhere

Figure 1. A) Orthopantomography performed during the first examination (T0); B) radiographic view after 6 months (T6); C) radiographic control immediately after crown delivery (T16).

in the patient's mouth, some specific factor must have acted here. Possible factors include a variety of suppositions: microbial attack at this unique site because of the implant's particular qualities, or compromised healing of the local host site (*e.g.* inadequate healing following extraction; immediate placement in a damaged site; *etc.*), or possible contribution of a relative overload.

The implant was placed in 2005 by a private dentist and its dimensions were probably inappropriate from a biomechanical point of view given the wide vertical and mesio-distal space available. However there is no certainty about the crown-to-implant ratio (C/I) of the first rehabilitation, because the patient did not have post-surgical radiographies nor the original crown.

The provisional screw-retained crown was realized to improve mastication on the left side of the mandible in 2013. It led to a high crown-to-implant (C/I) ratio (equal to 2.4) due to peri-implant bone loss. Misch[16] reported that a C/I ratio of 0.5-1.0 reduces stress on the peri-implant bone, thereby preventing bone loss and implant failure. However, C/I ratio guidelines have not yet been established. The high C/I ratio in the present clinical case could have influenced load transmission at peri-implant bone and induced the subsequent bone loss and implant failure. This finding is in contrast with that of other studies.[17,18] The animal study by Okada *et al.*[17] found that increased C/I may not be a risk factor for implant failure if the peri-implant mucosa is kept healthy. The clinical study by Tawil *et al.*[18] focused on 262 short (<10 mm) machined-surface implants consecutively placed in 109 patients and increased C/I values did not seem to be a major risk factor for peri-implant bone loss and implant failure at the medium-term follow-up.

In the present case report the crown was realized in composite resin, a material that appeared to be able to absorb shock from occlusal forces when compared with crowns made of zirconia, ceramic material, or gold alloy.[19] This choice was made in the attempt to reduce the load on the implant. Even though the intensity, frequency and duration of the occlusal overload could not be measured, the implant was considered overloaded because the patient chew only with the left side of his mouth, as the elements 45, 46, 47 had been extracted.

Severe peri-implant bone loss occurred before the patient came for a dental examination at the Prosthodontics Department. Probably bone resorption took place when the original crown was present and the implant was in function under occlusal load. Then peri-implant bone level remained stable as long as the implant was not loaded, although a 100% PI was present. In contrast, as soon as an occlusal load was applied peri-implant bone loss and implant failure occurred.

In successfully osteointegrated implants, the microflora identified is dominated by *Streptococcus* species such as *S. intermedius*,
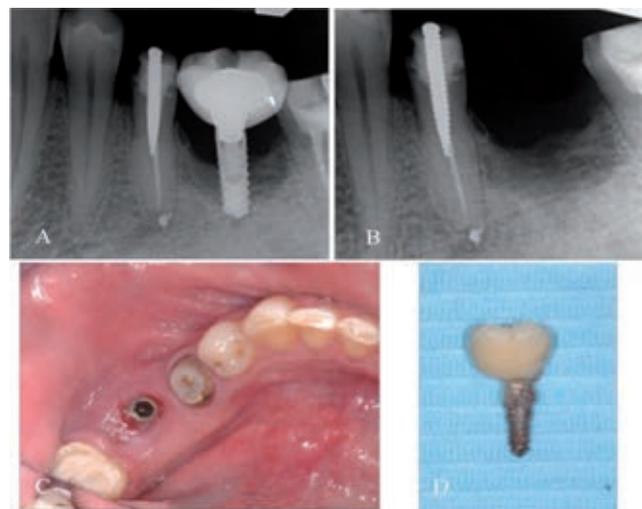

Figure 2 A) Radiographic control after 8 months of loading (T24); B) radiographic control of the post-extractive site; C) occlusal view immediately before implant extraction; D) dental implant extracted.
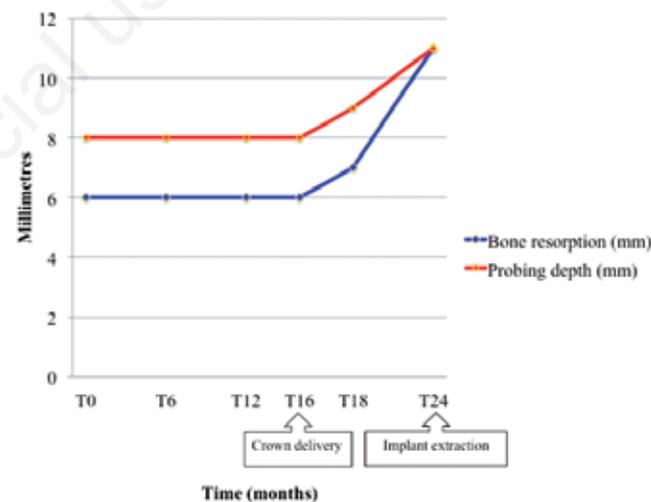

Figure 3. Time trend of probing depth and bone resorption during the 2-year follow-up (mean values).

Table 1. Values of peri-implant bone loss, probing depth, bleeding on probing and plaque index during the entire follow-up.

	Peri-implant bone loss (mm)		PD (mm)		BOP (%)	PI (%)
	Mesial site	Distal site	Mesial site	Distal site		
T0	6	6	7.5	8.5	100	100
T6	6	6	7.5	8.5	100	100
T12	6	6	7.5	8.5	100	100
T16	6	6	7.5	8.5	100	100
T18	7	7	9	9	100	100
T24	11	11	11	11	100	100

PD, probing depth; BOP, bleeding on probing; PI, plaque index; T0, baseline; T6, after 6 months; T12, after 12 months; T16, after 16 months; T18, after 18 months; T24, after 24 months.

S. oralis, *S. Sanguinis*, *S. gordonii*, *V. parvula*, *F. Nucleatum* and *Capnocytophaga* gingivalis.[20] However, conflicting data regarding the microbial species involved in the formation of the biofilms on the surface of implants and tooth during the initial phases of biofilm formation have been published, as either similar[21] or distinct patterns[22] have been observed. With regard to this aspect, it has been highlighted that the dental status significantly impacts microbial colonization of implant surfaces, as in partially edentuolous patients, microorganisms from the subgingival areas of teeth may invade implant sites and lead to colonization of implant surfaces, suggesting that in edentulous patients without any subgingival reservoir no transition of periodontal pathogens occurs.[23]

Gram-negative bacteria can be found in smaller proportions in healthy peri-implant sites and include *Prevotella intermedia*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Prevotella nigrescens*, and *Campylobacter rectus*.[20] As the peri-implant tissues undergo the process from a state of health to that of disease, a shift from a Gram-positive facultative dominated flora to a Gram-negative anaerobic biofilm occurs.[24] The failing implant is characterized by a greater proportion of red (*P. gingivalis*, *Treponema denticola*, and *T. forsythia*) and orange (*P. intermedia* and *Fusobacterium nucleatum*) complex, as well as *Aggregatibacter actinomycetemcomitans* and *Eikenella corrodens* with a lower proportion of the flora associated with health.[25] Hence, the main differences between health and disease are in the proportions of *Actinomyces*, orange and red complex species. Furthermore, increasing peri-implant probing depth has been significantly associated with higher total anaerobic cultivable microbiota and the

Table 2. Microbiota associated with dental implants.[20,25,26]

	Gram-positive bacteria	Gram-negative bacteria
Subgingival plaque associated with implants	S. sanguis *Haemophilus* spp. S. mitis S. morbillorum S. milleri *Streptococcus* spp. P. micros A. viscosus A. naeslundii A. istraelii A. odontolyticus *Lactobacillus* spp.	S. sanguis H. actinomycetemcomitans Copnocytophago spp. E. corrodens F. nucleatum Bacteroides spp. C. sputorum V. parvula L. buccalis - - -
Supragingival plaque associated with implants	S. sanguis *Haemophilus* spp. S. mitis S.salivarius S. morbillorum S. cremoris S. milleri *Streptococcus* spp. G. haemolysans P. micros A. odontolyticus *Lactobacillus* spp.	HS HS H. actinomycetemcomitans Copnocytophago spp. E. corrodens F. nucleatum Bacteroides spp. L. buccalis V. parvula - - -
Microbiota of failing implants	Prevotella intermedia P. nigrescens A. actinomycetemcomitans Staphylococci, coliforms, candida spp. Bacteroides forsythus Spirochetes Fusobacterium spp. Porphyromonas gingivalis Bacteroides spp. Fusiform bacilli, motile and curve rods Staphylococcus spp. P. nicrescens, P. micros Fusobacterium nucleatum Capnocytophaga spp Eikenella corrodens Campylobacter rectus Treponema denticola Tannerella forsythia Streptococcus anginosus (milleri) group Enterococcus spp. Yeast spp.	

frequency of detection of *P. gingivalis*.[26] Table 2 shows microbiota around dental implants.

The observations of this case report suggest that plaque accumulation alone is not a triggering factor for peri-implant bone loss and implant failure. On the contrary, occlusal load, when not properly controlled, might cause bone resorption. This might be exacerbated by microbial insult.

After osseointegration is obtained, a direct and relatively rigid connection between bone and the implant surface is achieved.[27] In these conditions, the peri-implant bone architecture develops an adaptation capacity to change in response to load conditions. According to Frost[28] within the range of a physiological loading, bone undergoes its physiological turn-over. In mild overloading, modelling drifts can begin adding to and/or reshaping bone, but in the case of a pathologic overload bone fractures and bone resorption may occur[28]. However, the amount of load to be defined as *overload* has not been quantified, also because the range of host physiological adaptability varies.[19]

Up to now, clinical evidence on the impact of overloading on peri-implant bone is not available. The inability to quantitatively define *overloading*, as well as several methodological and ethical limitations, has seriously compromised the potential of human studies to draw valid conclusions.

On the other hand, data on animal studies should be used with caution when discussing clinical evidence: dental implants are exposed to a complex series of different phenomena (both chemical and physical) in human and animal oral cavities including diet, oral hygiene, occlusal function, *etc*.

Conclusions

In conclusion, the present case report suggests that occlusal loading plays an important role in bone resorption and implant failure. However, data from case reports must be interpreted with caution without any generalization. Well designed clinical trials based on a greater number of patients are needed to validate the present outcomes.

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