Marginal Bone Loss A Review Of Selected Literature Marginal Bone Loss In Posterior Region

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ABSTRACT

Marginal bone loss is an unavoidable complication of implant placement which occurs in the first year of loading of implants and continues to occur at the rate of 0-0.2mm per year thereafter. But bone loss exceeding this proposed rate might result in loss of implant. The purpose of this article is to study and review the present concepts in marginal bone loss, so that better understanding can provide successful treatment.

With the introduction and discovery of concept osseointegration by Branemark in the middle of 1960s[1], a new treatment modality came into existence. With this treatment option long-term Successful treatment of complete or partially edentulous patients was now possible. But it has an unavoidable complication of marginal bone loss, which has been observed around the premucosal portion of dental implants for decades[2].

MARGINAL BONE LOSS

Implants get attached to bone through a complex phenomenon of osseointegration. And this process occurs due to continuous modeling and remodeling of bone. during this modeling and remodeling of bone marginal bone loss takes place.

Marginal bone loss was first reported by Adell et al[2]. Progressive and continuous marginal bone loss can lead to failure of implants. Aalbrektsson et al stated that an average bone loss in the first year of loading should not be more that 1.5mm and thereafter it should occur at the rate of 0-0.2mm per year1. Marginal bone loss exceeding this proposed rate can cause implant failure. Other studies reported an average first year bone loss of 0.93mm, with a range of 0.4 to 1.6mm and mean loss of 0.1mm after first year of implant functioning[2]. But there are many authors who argue and doubt that a firm measurement of marginal bone loss cannot be established and so it has been proposed that an implant is considered failed when marginal bone loss has reached upto apical 1/3 of the implant[1,3].

Continuous marginal bone loss does not only cause implant failures but also results in poor esthetics as soft tissue recession proceeds bone loss. The exact cause of marginal bone loss is unknown, There are many hypothesis postulated in this regard, but none of them is widely accepted[2]. But many authors believe that occlusal trauma may cause marginal bone loss. Finite element analysis (FEA) has shown that occlusal forces are distributed primarily to the crestal bone, rather that evenly throughout the surface area of implant[4]. Thus stress at that site may be the cause of marginal bone loss.

1. Periosteal reflection hypothesis

This theory states that, Periosteal reflection affects crestal bone blood supply, causing osteoblast death on the surface of crestal bone due to trauma and lack of nutrition[2]. Even though crestal cells die due to trauma and lack of blood supply, the blood supply is reestablished once periosteum regenerates. New blood vessels also form in that region and trabecular bone beneath the surface is a constant source of blood supply which is maintained even after periosteal reflection[2].

Preiosteal reflection would lead to generalized horizontal bone loss and not only at the implant site. And the bone loss would be visible immediately after 2nd stage surgery[2], but during implant placement such generalized bone loss is not visible. Thus this theory does not seem to be the cause of marginal bone loss.

2. Implant osteotomy hypothesis

This theory states that heat produced due to implant preparation site at bone destroys bone cell, causing early bone loss. A devitalized zone of 1mm is created around the implant and blood supply and cutting cones are necessary for remodeling of the bone. The crestal bone is more susceptible to bone loss as it has diminished blood supply and greater heat production[2].

If heat production due to site preparation would have been the cause, its effect would have been noticeable at the II stage surgery, implant failure after 4-6 months would have been visible, however, bone growth takes place. Therefore, this theory cannot be the primary reason for marginal bone loss[2].

3. Autoimmune response of host hypothesis.

This theory states presence of bacteria around natural teeth and implants to be the primary reason of marginal bone loss. Literature and repeated studied have shown that bacteria is the cause of vertical bone loss around natural teeth. Occlusal trauma may accelerate the procedure but alone is not the cause of marginal bone loss[2,5].

There are two infections caused by poor oral hygine around an implant, peri-mucositis, which is a reversible inflammatory reaction around functioning implants and peri-implantitis is inflammatory reactions with loss of bone support[1,6]. Prospective longitudinal data has shown that bone loss vary among patients. Marginal bone loss due to peri-implantitis falls in a range of 1% to 19%[7]. An association between periodontal and peri-implant conditions has been reported8. Longitudinal bone loss around implants was correlated to previous experience of reduced periodontal bone support. Thus the greater the full-mouth attachment loss around natural teeth, the higher the attachment loss is to be expected around implants, Such patients may show increased implant failure rate and marginal bone loss[8,9].

In a study of periodontically treated implant receiving patients, Ellegaard et al[8,10] reported that incidence of bone loss during five years of implant functioning increased by 45% of all implants displaying marginal bone loss of 1.5mm or more. In another study Karoussis et al[8,11] reported that patients with a history of chronic periodontitis had a higher incidence of peri-implantitis (28.6%) as compared with patients of no history of periodontitis (5.8%).

In a prospective study of 125 implants Adell et al[12] reported no inflammation in 80% of sulcular regions of implants. Yet marginal bone loss occurs and its occurrence decreases with time, if bacterial manifestation would have been the cause, bone loss should have increased[2]. Inspite of variation in literature available it is accepted that poor oral hygiene causes a more rapid bone loss and implant failure, bur it cannot be substantiated[2].

4. Biological Width Hypothesis

Biological width is a natural phenomenon that exists with the natural tooth. Biological width refers to the height of the dento-gingival attachment apparatus around a normal tooth and is defined as the distance necessary for a healthy existence of bone and soft tissue from the most apical extent of a dental restoration. It has greater dimension in the posterior teeth as compared with the anterior. Many authors have stated that implants may also exhibit this phenomenon, which may explain marginal bone loss, more in the first year of loading and less thereafter.

Berglundh et al[13] observed 0.5 mm of bone loss below the implant abutment connection within 2 weeks after stage 2 surgery in dogs.

Lindhe et al[14] reported inflammatory connective tissue 0.5 mm below the implant abutment connection. Wallace and Tarnow[15,16] stated that biologic width also occurs with implants. Makigusa[17] on animal study showed that the biological width gets reestablished after implant placement, As this occurs, circumferential bone loss typically occurs around the implant's coronal aspect up to the first implant thread. Abrahamsson et al[18] suggested that a certain width of the peri-implant mucosa is required to enable a proper epithelial-connective tissue attachment, and if this soft tissue dimension is not satisfied, bone resorption will occur to ensure proper establishment of attachment with appropriate biological width. It is suggested that once the implant is in the oral environment and is functioning, a mucosal attachment of a certain minimum dimension is required to protect osseointegration[18].

But the anatomy between tooth-bone-attachments varies between implant-bone attachment. Implants are not designed to interact in the same manner as tooth does with its periodintium, the surrounding tissues are expected to adapt to the inserted post[19]. In implant-gingival attachment only two gingival attachment fiber groups exist and no periodontal fibers are present. Also, there is no cementun, due to absence of progenitor cells at implant insertion prepared site[19]. On the contrary, in experimental studies on dogs, Berglundh and Lindhe[18] found that non-mobile gingiva and periimplant mucosa had many features in common. Both soft tissues included a junctional epithelium which was separated from the bone crest by a zone of connective tissue attachment >1mm high.

However this concept does not completely explain marginal bone loss. Bone loss due to biological width adjustments occurs with in 1 month of implant placement and is related to implant design and abutment-implant connection in relation to bone. Also, this concept does not explain why greater bone loss occurs in soft bones compared to dense bones, nor it explains higher implant failure rates in less quality bones after loading[2].

OCCLUSAL TRAUMA AND LOAD

Present literature available on this concept stating that excessive bone strain may not result in physical fracture but may also cause bone cellular resorption. The amount and distribution of stress in the bone may lead to marginal bone loss, thus affecting osseointegration[20]. Available literature states that bone loss due to stress and strain was observer around the neck of the implant, leading to marginal bone loss[20-24]. According to Frost[25], bone fractures at 10,000 to 20,000 microstrain units (1%-2% deformation), but at 20% to 40% of this value bone cells may trigger cytokines to begain cellular resorption.

Baggi et al[26] stated that stress concentration areas were located at the cortical bone around the implant neck and the highest values were of von mises and compressive streses were observed for maxillary implants. Hoshaw and coworkers[27] reported that overloading of implants resulted in an increased bone resorption around the implant collar and a decreased percentage of mineralized bone tissue in the cortex within 350µm of the implant.

IMPLANT DESIGN

Different amounts of marginal bone loss have been reported for different implant designs and shapes. It has been stated that implant body may affect the amount of strain at the implant-bone interface[2]. the design of an implant refers to a three-dimensional structure of an implant system characterized by shape, type of implant-abutment mating, presence or absence of threads, thread design, surface topography and chemical composition[22]. the implant-abutment junction should have such a design that it reduces the peak bone-implant interface shear stress and strain[22].

Nordin et al[28] showed that a conical implant had a wider diameter and it resulted in bone resorption along the conical surface down to the first thread. Sakoh et al[29] showed that conical implants had a higher primary stability than cylindrical implants. Quaresma et al[30] in FEA study reported that conical implants connected to conical abutment produced lower stresses on the alveolar bone and prosthesis and greater stress on abutment compared to a cylindrical implant connected to screw-retained, internal hexagonal abutment. Using FEA Rieger and associates[22] showed that tapered implants are better than cylindrical implants and truncated fins or serrations are better than untruncated fins, serrations or threads.

According to hansson[22] the implant-abutment junction should be located at the cervico-occlusal level not close to the region where implant starts to contact bone, because according to Saint Venant's principle, if the implant-abutment junction is close to the crestal bone level, it will increase the magnitude of stress and strain at the site, resulting in bone loss. It was also found that bone resorption could be reduced when the abutments are smaller than the diameter of the implant body- platform switching[24].

Surface of the implant also plays a crucial role. Experimental studies have shown that increased surface roughness requires more time for bone to create mechanical interlocking and risk of bone loss, once exposed to oral cavity, thus initial response is poor but once formed, is more effective[22]. From a biomechanical prespective wider implants allow maximum amount of bone engagement and improved distribution of stresses in the surrounding bone and this may reduce stress around the crestal bone and potential bone loss[4].

BONE PHYSIOLOGY

The bone is less dense and weaker at stage 2 implant surgery than it is after 1 year of prosthetic loading. Bone takes 52 weeks for its complete mineralization[2]. Thus the stress applied to the peri-implant bone may be greater to cause bone resorption during the 1st year of prosthetic loading, because bone strain are greatest at the crest.

DISCUSSION

There are many hypothesis and experimental evidences to evaluate the cause of marginal bone loss, but none of them have explained a satisfactory reason for marginal bone loss.

A plausible reason why bone loss does not occur around natural teeth in function could be that bone cells around teeth accommodate to increasing functional needs during strain, thus strain gradient falls within physiological levels. But the bone-implant interface does not provide physioelasticity and is incapable of detecting forces. Also, bone formation is time dependent, thus bone cells around implant experience high mechanical strain during overloading and functioning. This situation may explain marginal bone loss more during initial year and less thereafter.

Another possible reason of marginal bone loss could be microfractures caused due to stresses at the crest of the ridge during the first year of overloading or functioning of the implant. And when the change in the bone strength after mineralization is complete stress/strain relation reduces the risk of microfracture during the following years[2].

Apart from the factors discussed there are many other factor which should be considered, such as available bone, implant site, bone density, prosthesis design, patient force factors like clenching, tongue thrusting, bruxism and patient's habits like smoking. Bain and Moy[8] reported greater implant failure rate in smokers that in nonsmokers. Smokers had a failure rate of 11.3% as compared to 4.8% in nonsmokers. Smoking has also been associated with greater marginal bone loss[8].

Treatment modality should result in as low marginal bone resoeption as possible. Though this phenomenon is unavoidable, implant placement should be done in such a manner that marginal bone loss does not exceed more than the proposed and documented range of less than 1.5mm in the first year of loading and 0-0.2mm per year thereafter.

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