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Crestal Bone Loss: Causes and Medical Correction

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Authors' contributions

This work was carried out in collaboration among all authors. Author AMS designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors M. A. Bakhova and ZKA managed the analyses of the study. Authors M. A. Bereketova and MTM managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

To maintain the long-term stability of the implant, it is important to minimize the loss of bone tissue around the implant. Several clinical studies have shown that the average loss of the crestal bone around the implants is 1.5-2%. mm during the first year after prosthetics has been done. In this article, the author suggests a number of hypotheses related to the loss of crestal bone. He comes to the conclusion that stress and overstrain are the main factors that lead to the loss of crestal bone during the first year of implant functioning.
The author notes that in the hypotheses presented in the article there are causes that can influence the loss of bone around the implant in one way or another. At the same time, it is necessary to carry out the appropriate medical correction of the oral cavity through antibiotic therapy and anti-inflammatory drugs, which will also prevent the process of losing the crestal bone.

Keywords: Crestal bone; implant, osteotomy canal; stress factors; autoimmune reaction of the body; periosteal detachment.

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1. INTRODUCTION

They say, dental implants are one of the most exciting treatment in the modern dentistry. Unlike crowns, bridges, or veneers that attach to existing teeth, dental implants completely replace lost or damaged teeth by connecting the titanium "root" directly to the jawbone and attaching a fully functional, cosmetically perfect ceramic tooth.

Since Branemark discovered that the osseointegration between titanium and bone occurred in the mid-1960s, dental implants have been introduced to replace missing teeth, and the treatment options for partially or completely missing teeth have expanded exponentially [1].

The improvements in implant technology have pushed clinicians to use dental implants as a gold standard treatment to rehabilitate monoedentulism.

The post-implant healing of various types of implant has been documented in various clinical and in vitro studies. The quality and stability of the soft tissue interface with implants and abutments, together with the preservation of the crest bone, are most likely of paramount importance for the short-and long-term prognosis of oral implants [2].

These days, patients undergoing implantation not only expect their chewing function to be restored, but they also expect the prostheses to be aesthetically pleasing, easy to care for, and durable. To maintain the long-term stability of the implant, it is important to minimize the loss of bone tissue around the implant, as well as the accompanying soft tissue atrophy. Saucerization after abutment connection has been reported mainly for two-stage implants with butt joints (Brånemark implants are a typical example).

Albrektsson et al. [3] found that the installation of two-component implants, healing by immersion, led to the loss of bone tissue of the crest by 1.5-2.0 mm after 1 year of loading. Moreover, in experimental studies on dogs, the remodeling of the crest bone with a resorption of 2 mm was confirmed.

Therefore, clinicians, researchers, and implant manufacturers have devoted time to finding ways to control the loss of ridge bone tissue that occurs after the abutment and prothesis are installed.

At the Toronto conference, the consensus for bone loss around the implant was that bone loss of approximately 2 mm during the first year of implant operation is acceptable, and at this level the implant is considered successful. Numerous research reports have been published on the causes of bone loss around implants and on clinical methods to prevent it such as the implant neck in relation with the bone (transmucosal or bone-level implants).

These days, patients undergoing implantation not only expect their chewing function to be restored, but they also expect the prostheses to be aesthetically pleasing, easy to care for, and durable.

The aim of the research is to study the various conditions under which the process of crestal bone loss can be initiated and implemented.

2. MATERIALS AND METHODS

The results of the author's own practical experience and literature on the topic were used in the present paper, and the corresponding array of publications within the framework of the topic under study was analyzed. Comparative-comparative and analytical methods were adopted as the main research methods.

3. RESULTS

There is no doubt that excessive stress acting on the area of contact of the implant with the bone can cause overloading and failure. This condition can develop soon after surgery and lead to implant mobility instead of rigid fixation. In addition, excessive stress can affect the final restoration after successful integration of the implant and lead to its failure. Thus, stressful conditions increase the risk of insolvency.

The clinical success and durability of endosteal dental implants largely depend on the health of the surrounding anal area and soft tissues. Early loss of the crestal bone around the implant is rarely associated with concomitant shrinkage of the surrounding soft tissues, on the contrary, the formation of peri-implantation periosteal and subcostal pockets is essential. The bacteria that live in the implant furrow, according to available data, are similar to those of a natural tooth. Anaerobic bacteria appear when the oxygen tension decreases and often dominate the gingival pockets larger than 5 mm. Anaerobic bacteria are associated with bone loss and

deterioration of periodontal and preimplantation conditions. In addition, daily care of the crestal bone is worsened if the pocket of soft tissue is larger than 4 mm. Thus, it is reasonable to reduce the early loss of the crestal bone in order to preserve the local environment favorable for preimplantation health [4].

In less dense bone, the loss of the crestal part and the failure of the implant in the early period is more common than in dense bone. The failure of the implant in the early period may also be the result of too much stress acting on the implant. Consequently, stress factors can affect both early and late implant failure, and the loss of the crestal bone. Stress is a very important factor, and it should be evaluated before treatment in order to minimize its impact.

Early loss in the crestal region is described by many surgeons, regardless of surgical approaches and protocols , and can range from marginal bone loss to complete implant failure. The loss of the crestal bone varies in volume, and its rate decreases radically after the l-st year of prosthetics. This phenomenon is called saucerization (groove formation).

The initial loss was first reported by Adell et al. [5]. In this work, a fairly significant amount of bone loss was noted in the period when the year from the moment of implant placement has not expired. The average load value was 1.2 mm, and the extreme values were from 0 to 3 mm. The measurement of the volume of bone loss is carried out from the first turn of the thread, which acts as a reference point of 0 mm, the initial ridge of the ridge bone is not taken into account, since the implant will rise above the specified point by 1.8 mm. Accordingly, the total volume of the crestal bone loss is 3.3 mm. The annual subsequent losses then ranged from 0.05 to 0.13 mm per year.

Bone loss is observed with different bone densities and with different implant designs. It is important to determine the causes of the early loss of the crestal bone around the implants and their early failure in order to successfully combat these phenomena, to ensure the long-term health of the peri-implantation area, and a good prognosis of the implantation itself. Implantologists from different countries have long been discussing and searching for the reasons for the early loss of the crestal bone. One other factor that may affect the bone loss and the long-term implant success is the experience of clinicians who have performed the surgery [6].

4. DISCUSSION

There are several main hypotheses for the loss of the crestal bone.

1. The hypothesis of periosteal detachment. It is known that when the periosteum is detached during dental (surgical) manipulations, the nutrition of the bone is disrupted. Up to 87-90% of arterial blood supply and 100% of venous return is associated with the periosteum of the long bones. Detachment disrupts the nutrition of the crestal bone, as a result of the death of bone cells from trauma and lack of nutrition. These phenomena support the theory that periosteal detachment is the main cause of early bone loss around the implant.

First, this theory is opposed by the fact that during implantation, we have a loss of the crestal bone in the form of" grooves", i.e., a localized loss. If the cause of bone loss was the detachment of the periosteum, then we would observe a generalized loss of the entire detached residual ridge.

Secondly, we have the loss of the crestal bone at stage 3 (prosthetics). According to the hypothesis of periosteal detachment, bone loss would already be noticeable at stage 2 (opening) after 3-7 months. After the first stage(implant placement). However, at the opening stage(stage 2), bone loss is rare.

This hypothesis cannot explain the reason for the early loss of the crestal bone, and we cannot accept it as a factor that causes the early loss of the implant after opening.

2. The osteotomy canal hypothesis. The bone is a labile organ, it is very sensitive to heat. During the formation of the implant bed, the bone that is in direct contact with the implant is injured. The injury leads to the formation of a dead zone around the implant with a width of approximately 1 mm.

The crestal region is less well supplied with blood and is subject to greater heat during the formation of the implant bed. This is especially pronounced when using less efficient milling cutters (countersinks).

These facts support this hypothesis. But if the trauma and heat during the formation of the

implant bed were the cause of the loss of the crestal bone, the effect would be noticeable at stage 2 (opening) after 3 to 7 months. When the implant is opened in the second stage, we do not observe the loss of the crestal bone.

3. The hypothesis of an autoimmune reaction of the body. According to this hypothesis, the main cause is bacteria. Bacteria are indeed the cause of vertical defects around the tooth.

The bacterial flora in the implant furrow is similar to the bacterial flora of a natural tooth. It can be assumed that the early loss of bone around the implant is caused by bacteria. Occlusive factors, in turn, can accelerate the process or participate in it.

Adell et al. 125 patients were examined after rehabilitation with implants [7].

Examination of the gingival furrows near the implants revealed that in 80% of cases there was no inflammation. Lekholm et al. It was found that the amount of crestal bone loss does not depend on the depth of the gingival pockets near the implants [8]. During the first year of operation of the implant, approximately 1-1.5 mm of bone is lost, and during each subsequent year, 0.1-0.2 mm is lost. The question arises, why does a large bone loss occur in the first year of exercise? After all, the depth of the pocket only increases over the years, and therefore the number of microorganisms in them increases, maintaining hygiene becomes more difficult. According to this theory, the loss of bone should increase every year.

4. The biological width hypothesis. Anatomically and histologically, the areas of the gingival sulcus around the natural tooth and the implant are very similar.

The fundamental difference is at the base of the gingival sulcus .The average biological width for a natural tooth is 2.03 mm. This is the distance between the bottom of the dentoalveolar sulcus and the crestal edge of the alveolar bone. The biological width functions as a barrier, it prevents the penetration of bacteria, thereby protecting the underlying periodontal tissues. With improper orthopedic treatment, when the edge of the crown intrudes into the biological width, there is a decrease in the crestal bone, it retreats to restore the biological width, to create favorable conditions for the gingival fibers.

Surgical protocols of many implantation systems recommend the placement of implants below the edge of the ridge or at its level.

The connection of the abutment to the implant body can be compared to the edge of the crown.

Bergludh et al. in their studies in dogs, they showed bone loss of 0.5 mm below the connection of the implant with the abutment for 2 weeks after stage 2 (opening) [9].

Lindhe et al. connective tissue inflammation was reported to have spread 0.5 mm above and below the implant-abutment junction [10].

Wallace and Tamow argue that biological width is one of the factors behind the early loss of the crestal bone. In other words, the bone level cannot be higher than the connection of the implant with the abutment [11].

The biological width hypothesis explains the small loss of the crestal bone in stage 1 of implantation, and it also explains the early loss during the first year after stage 2.

The natural tooth is surrounded by about 11 types of gum fibers, more than half of which penetrate the root cement. There are crestal fibers that are woven into the cement above the alveolar bone. There are only 2 types of fibers in the implant area. They do not penetrate the implant body below the edge of the abutment. A living tooth has gingival and periosteal fibers that connect the tooth to the surrounding tissues, unlike the transossal part of the implant.

Therefore, the attachment of connective tissue as a component of the biological width for teeth is different from the area of attachment to the abutment of implants.

There is no doubt that the early loss of the crestal bone may be the result of tissue remodeling to create a biological width below the level of the abutment. There are no areas of connective tissue attachment to the implant. The furrow around the obturation screw of the implant body during primary healing is similar to the furrow at the junction of the implant and abutment. Nevertheless, the bone can outgrow the limits of the obturation screw. It should be concluded that the furrow between the gum and the abutment itself is not the cause of bone loss. However, if the furrow is filled with bacteria, after it comes into contact with the local environment of the oral cavity, bone loss is noted. This theory

cannot explain the early loss of the crestal bone, which is also observed when using solid (non-removable) implants that do not have an implant-abutment junction area. There is indeed a biological width around the "implant-abutment" joint, which is formed 2-4 weeks after the implant is exposed. Therefore, the volume of bone loss for this reason is formed before the prosthesis is made. With a single-step installation, the effect of bone loss is determined even before the soft tissue becomes mature.. Many authors discuss the impact of different implant designs on the biological width and early loss of the crestal bone. An implant with a smooth-polished neck of 2 mm is associated with less bone loss than a neck of 4 mm. Bone loss before the third turn of the thread implies that the amount of bone loss is similar for different implant designs. However, on different implant designs, the first turn of the thread is located at different distances. Biological width is certainly important in the loss of bone in both the early and late period, but it is not the cause of the loss of the cross bone in the first year of the implant operation. This concept also does not explain the greater loss in the lower density bone than in the higher density bone.

5. The hypothesis of stress factors. The bone is capable of remodeling, i.e. it can adapt to the biomechanical situation. Hoshaw and co-authors placed dental implants in the tubular bones of dogs. A load was applied to the implants, after which the bone cells were reorganized along the thread in order to resist the load falling on them [12]. A change in the pattern of bone tissue was observed in the layer with a thickness of 3-4 mm around the implants. This means that the bone around the implant is able to remodel in relation to the current load.

Frost conducted studies in which he revealed a bone response to micro-stresses [13]. At 10-20 thousand units of microstrain, the bone breaks, which is about 1-2% of the deformation. Already at a third of these values, the bone tissue in the zone of pathological overload begins to disappear and form fibrous tissue. What does it mean? This means that a strong tension in the area of contact of the implant with the bone causes the loss of the latter. In addition, tension in the bone can be caused by the stress applied to the prosthesis. Most current dental implants are made of titanium or its alloy, the modulus of elasticity or elasticity of which is 5-7 times higher than that of the cortical bone. When two materials come into contact with different elastic modules without using an intermediate material and when one of them is subjected to a load, an increase in the stress contour will be observed at the point of contact of these two materials - this is one of the principles of mechanics. Contours are formed that have a V - or U-shaped pattern with the largest value at the place of primary contact.

In 1972 Kummer put forward the theory of cross bone remodeling and revealed a direct relationship between stress and the amount of bone loss. According to this theory, an increase in stress at the edge of the ridge beyond the physiological limits causes such tension in the bone that it causes its resorption (due to pathological overload). Stress and overload also reduce the capacity of blood vessels, which in turn can contribute to bone loss and make the local environment more susceptible to anaerobic bacteria.

Excessive loads on the implant can cause a fracture of its body or a loosening of the fixing screw, followed by a fracture of the latter.

These same types of stress also may lead to the loss of the crestal bone. When the implant is opened after 3-4 months, the bone is weaker than a year after that, i.e. less mineralized. After 3.5 – 4 months, bone mineralization reaches only about 58-60%, and complete mineralization takes about 52 weeks.

Bone density depends on the degree of its mineralization. After we create a bed for the implant and install the last one, we have a necrosis zone about 1 mm wide, which is then replaced by a fibrous bone. It is not organized and is less strong than mature bone, which is less susceptible to stress. It takes several months of time to replace the fibrous bone in the lamellar mature. If we injure the crestal bone at the opening stage, then it also takes additional time for it to heal, so that a mature bone is formed in the contact zone. The strength of the bone in the crestal region is different and depends on its density. The bone can react to stress and change its strength and density as the implant is exposed to functional loads. In other words, it is able to change its microscopic organization within the first year.

Piatelli et al. implants were installed and loaded in monkeys. After the load, the thin trabecular pattern was replaced by a rough one, more pronounced in the crestal region [14].

Hermann et al. we conducted similar experiments on dogs. They installed the implants, and then applied a tilting force to them once a month for 3 months. There was a change in the bone structure from thin to coarse trabecular [15].

Hoshaw, Hoshaw et al. the implants in dogs were loaded with a stretching load, and the result was similar [16].

Accordingly, it is necessary to conclude that the strength of the bone can increase depending on the functional load. As a result, occlusal overload initially leads to bone loss, but it is not large enough to cause prolonged bone loss, as the bone becomes denser. The bone has become stronger, so the loss can be stopped. In addition, more time passes before a higher level of stress reaches this zone, which, in turn, allows for a greater degree of implementation of the processes of mineralization and organization of the bone.

Appleton et al. clinical studies were conducted where they placed implants on the human lower jaw from different sides [17].

One side was loaded progressively, the other non-progressively. Progressively loaded implants showed less loss of the crestal bone, with an increase in the density of the latter. A lower loss of the crestal bone at a higher density indicates that the stress-strain system is the main cause of the loss of the crestal bone after the implant is loaded.

Thus, stresses at the edge of the ridge can cause micro-fracture or overload during the first year, but changes in bone strength due to the current load and its mineralization completely change the "stress-stress" relationship and reduce the risk of micro-fracture during subsequent years.

During the first year of implant operation, the amount of bone loss varies, depending on the amount of stress and the initial bone density.
The upper jaw is less dense in structure than the lower jaw, so with the same amount of stress, the volume of bone loss there will be greater. The denser bone on the lower jaw stops stress closer to the sacrum, while the softer bone in the upper jaw allows stress to spread further along the graft. The softer the bone, the further the stress will spread in the apical direction. The absence of bone loss is noted when the stress factors are reduced.

Jung et al. It was found that the loss of the crestal bone during the first 12 months of exercise corresponded to the length of the polished neck of various designs [18].

Miyata et al. crowns with a mismatch of occlusal surfaces of 100, 180, and 250 microns were installed in monkeys [19]. Four weeks later, the implants were evaluated. The levels of the crestal bone in the group with a discrepancy of 100 microns and in the control group without load were similar. A V – shaped bone loss was observed with a discrepancy of 180 mmcm. At 250mkm, the bone loss was 2-3 times greater. The higher the occlusal mismatch, the greater the bone loss and the higher the risk of implant failure.

Lindquist et al. found that the consoles on the implants also negatively affect the crestal bone causing its loss. The longer the console was, the earlier the loss of the crestal bone was [20]. The amount of stress can also be influenced by the shape and design of the implant.

5. CONCLUSIONS

All of the above studies indicate that stress and overload are the main factors that affect the loss of the crestal bone in the first year of implant operation.

The crestal bone may become overloaded during the first year of implant operation. In this area, the stress is highest. Along with this, the underlying bone can be subjected to physiological stress, allowing the bone to remodel, it becomes more durable and dense.

There is no doubt that the causes that are considered in the hypotheses can affect the crestal bone around the implant during its service life and lead to its loss.

CONSENT

It’s not applicable.

ETHICAL APPROVAL

It’s not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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