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Histopathological and microradiological features of peri-implantitis. A case report

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SUMMARY

Aim. The aim of this study was to describe the histological characteristics of a peri-implantitis case in the anterior maxilla.

Case report. A dental implant inserted in the missing upper right lateral incisor region has been removed with its adjacent tissues. The samples were placed in 4% formalin for 10 days and, were embedded in methacrylate prior to sawing and grinding. The samples were processed with Donath´s sawing and grinding technique, stained with toluidine blue and mounted on high-sensitivity plates for histology and microradiography.

The structure of the connective tissue revealed that there was a lack of collagen fibers running parallel to the implant surface. The connective tissue showed a loose granulation tissue with medium-density lymphocyte infiltration and neutrophilic leukocytes. In addition to the collagen loss in the infiltrated tissue, an excessive bone resorption was present. Peripherally, the light microscopy showed the osteoclasts and their adhesive apparatus which promote bone resorption.

Conclusion. With the increasing number of implants being placed, peri-implantitis has become much more prevalent. Every additional study focusing on the characteristics of peri-implantitis would be beneficial to gain an understanding of bone and soft tissue behavior around the implant and could help to develop appropriate therapeutic approaches for peri-implant disease.

Keywords: peri-implantitis, histology, dental implant.

INTRODUCTION

Dental implant failures could be related to perioperative causes such as overheating, contamination and trauma during surgery, poor bone quantity and/or quality, lack of primary stability and incorrect immediate loading. Besides that, peri-implant diseases, occlusal trauma, and overloading failures can result in loss of dental implants in the long term (1, 2).

Peri-implant diseases are a common cause of complications that may be associated with dental implant failures. They are characterized by an inflammatory reaction in the tissues surrounding an implant and present in two forms – peri-implant mucositis and peri-implantitis. In peri-implant mucositis, the presence of inflammation is confined to the soft tissues surrounding a dental implant, however, no signs of loss of supporting bone following initial bone remodeling are observed. The term peri-implantitis refers to an inflammatory process around an implant, which includes both soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling (3).

The etiology of peri-implantitis remains still challenging and unfortunately the knowledge regarding the histological hard and soft tissue changes in chronic peri-implantitis situations in humans are limited to case reports (4). Therefore, every additional report focusing on the histological aspects of the condition in humans is critical to gain an understanding of peri-implantitis to develop appropriate therapeutic approaches.

The aim of this study was to describe the histological characteristics of a peri-implantitis case in the anterior maxilla.

CASE REPORT

A 56 year old healthy, non-smoker female patient admitted with the complaints of pain and swelling around the dental implant at the missing upper right lateral incisor region. Her history revealed that, 6 months following the extraction of the upper lateral

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incisor due to an unsuccessfull endodontic treatment, a dental implant of 3.5×13 mm (NTI 13-SR, Hi Tec, München, Germany) was placed in the corresponding area seven years ago (Figure 1 A and B).

The intraoral clinical examination revealed pus and swelling at the corresponding area. A panoramic radiograph showed the presence of the excessive bone loss around the dental implant. (Figure 1 C) The probing depths were 8 and 9 mm at the buccal and palatinal sites respectively. The implant was mobile and the bleeding index and the plaque index were both 100%.

Owing to the excessive bone loss around the dental implant, it was decided to remove the implant under local anaesthesia after taking the local infection under control. A 1 g of Amoxicillin (STADA Arzneimittel AG, Bad Vilbel, Germany) and 0.4 mg Metronidazol (×2 per day) were prescribed for 7 days (ALIUD® PHARMA GmbH, Laichingen, Germany).

On the fourth day following the drug administration, a sulcular incison extending from upper right canine to the upper left central and vertical releasing incisons were performed under local anesthesia. A full thickness mucoperiostal flap was elevated and the bone defect was exposed. The implant was removed via a Piezotome (Piezotome®2, Satelec Acteon) with its adjacent tissues. The defect was curetted and closed primarily via 3/0 silk sutures. The healing period was uneventful and the sutures were removed 5 days later.

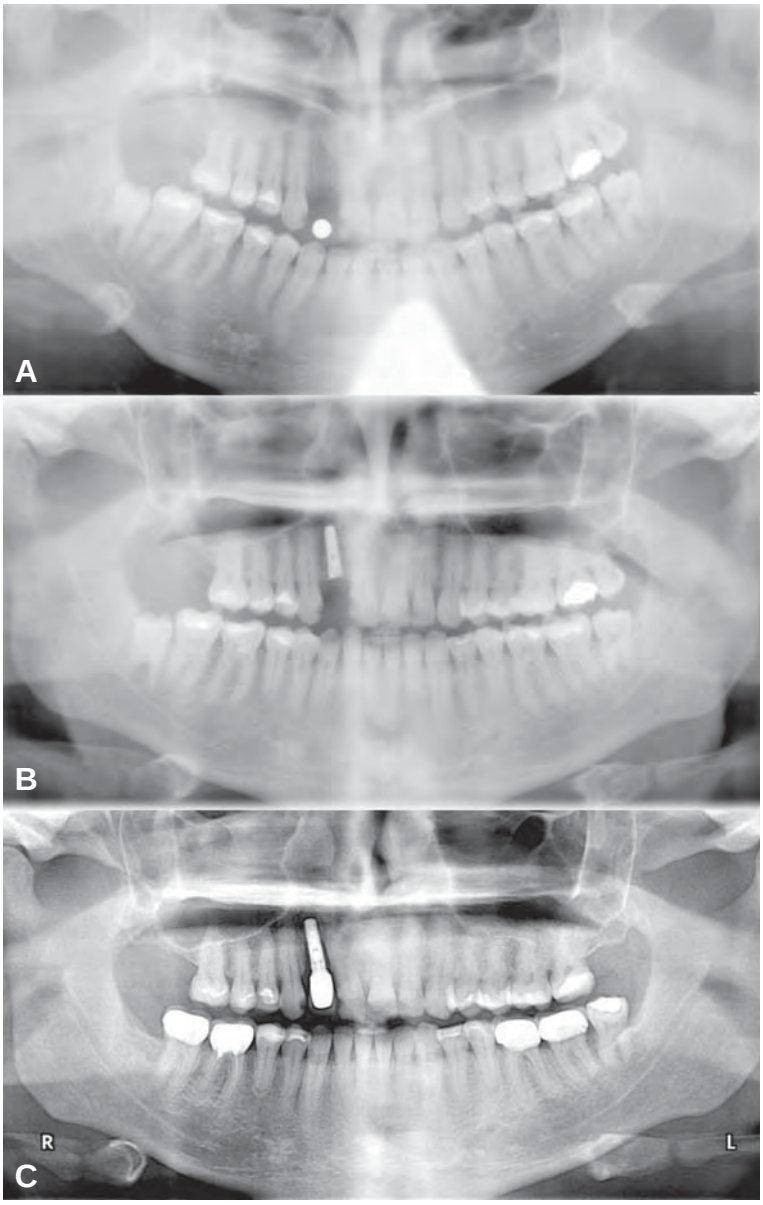

Fig. 1. Preoperative OPTG (A). Immediate postoperative OPTG (B). A 7-year postoperative OPTG revealed excessive bone loss around the dental implant (C).

Sample processing, sawing and grinding

The Implant and the adjacent tissues were placed in 4% formalin for 10 days and embedded in methacrylate prior to sawing and grinding. Sawing and grinding was performed (5) and the samples were placed in glass vessels filled with a monomeric resin solution and incubated at 37°C to 40°C for 2 to 4 days for resin impregnation. The sample was precut with a band saw (Exakt, Norderstedt-Germany) and disks of about 100 µm were obtained via an oscillating diamond saw (Exakt, Norderstedt-Germany), grounded with the Saphir 360 E grinder (ATM, Altenkirchen-Germany) and highly polished with silicon carbide paper (grades 500, 1200, 2400 and 4000).

Staining

Stainings were made by using toluidine blue. The ground surface was decalcified with 0.1% formic acid and 20% methanol was applied for better cell and soft tissue staining. Then the samples were rinsed in distilled water and stained in a toluidine blue solution for 2 minutes.

In this process, hard tissue has not stained or at best assumed a light blue color whereas cells and their nuclei, osteoid, cement lines and collagen fibers has stained blue, while mast cell granules, cartilage matrix and early wound healing sites metachromatically has stained red violet.

Microradiography

The samples were glued onto film-coated light-sensitive glass plates (High-Resolution Plates Type 1A, Imtek) - depending on their thickness - and exposed to 18 kV and 5 mA for 8 to 10 minutes in the microradi-

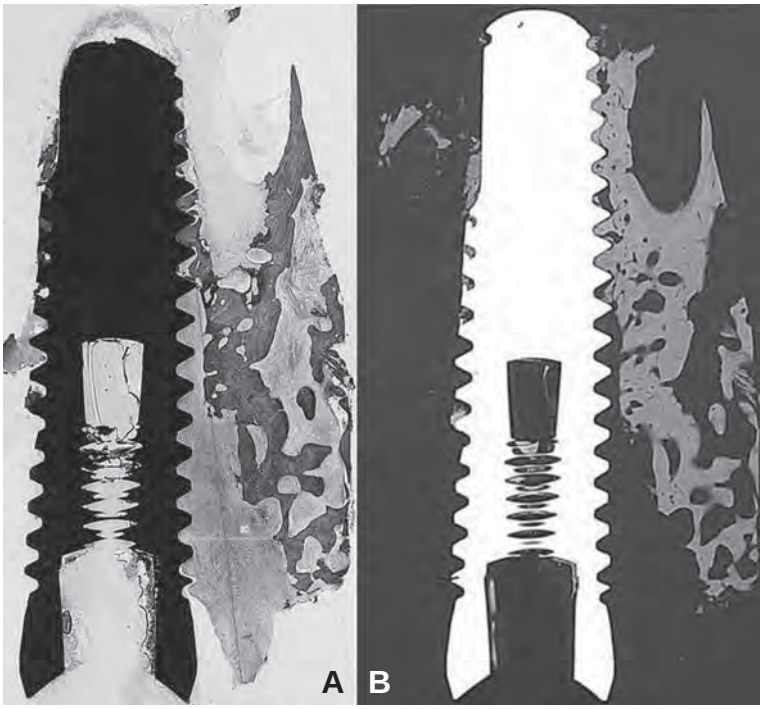

Fig. 2. The histological examination revealed the loss of osseointegration on buccal and palatal sites (A). The microradiography revealed the lack of bone-implant contact (B).

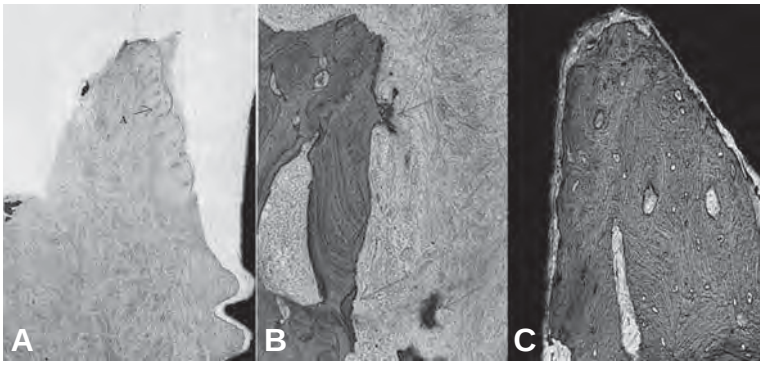

Fig. 3. Peri-implant mucosa showing interlocking between the connective tissue and the epithelium (Toluidin blue ×30) (A). Light microscope showing active bone destruction (Toluidin blue ×30) (B). Resorption lacunas by the osteoclasts. (Toluidin blue ×16) (C).

ography chamber (Faxitron X-ray Systems 43855A, Hewlett Packard GmbH, Böblingen-Germany). The films were developed with an HRP developer and fixed in an A3000 fixation bath (both by Kodak AG, Stuttgart-Germany). After drying, the sensitive film layer was covered with a cover plate and mounted with Eukitt®. The microradiograms were morphometrically evaluated by using a digital image analyzer (Q500MC, Leica, Cambridge-UK). A measuring frame was placed on invariably identifiable screw holes and the surface area of newly formed bone was measured.

All components of the infra-bony pocket at the buccal site could be successfully observed (Bony structure, epithelium, inflammated connective tissue and dental implant). The histological examination revealed the absence of the osseointegration on both buccal and palatal sites. In addition to the collagen loss in the infiltrated tissue, an excessive bone resorption was seen. Adjacent to the apical part of the peri-implant pocket, an active bone resorption was observed (Figure 2 A). The microradiography revealed the lack of bone-implant contact (Figure 2 B).

The peri-implant tissue was loosened from the implant surface during the preparation of the histological specimen; however, was congruent to the implant surface. The ortho-keratinised epithelium of the gingiva could not be fixed. The non-keratinized junctional epithelium showed an attachment of 2 mm. Oral sulcular epithelium showed interlocking between the epithelium and the underlying connective tissue. An increase of inflammatory cell infiltration and the proliferation of ulcerated granulation tissues were present apico-laterally. The structure of the connective tissue revealed that there was a lack of collagen fibers running parallel to the implant surface, which are present around successfully osseointegrated implants The connective tissue showed a loose granulation tissue with medium-density lymphocyte infiltration and neutrophilic leukocytes and a loose, cicatricial tissue which is rich of fibroblasts and with a few angioblasts (Figure 3 A). Peripherally, the light microscopy showed the osteoclasts and their adhesive apparatus which promote bone resorption. In addition, bone fragments dissolved from the bone surface were detected (Figure 3 B). Adjacent to the threads of the implants an increased concentration of osteoclasts was seen. In the time of the implant removal, the osteoclasts showed various sizes of lacunas, which refers to a progressing bone destruction (Figure 3 C).

DISCUSSION

Potential risk factors for peri-implant disease were reported as poor oral hygiene, history of periodontitis, smoking habit, diabetes, genetic triats, implant related factors (surface characteristics and implant placement-related factors), insufficient width of keratinized peri-implant mucosa and occlusal overload (6-10). According to general health- and oral hygiene status and past medical history of the patient, none of the above mentioned risk factors was present in the current case.

Similar to periodontitis, peri-implantitis occurs primarily as a result of an overwhelming bacterial

insult and subsequent host immune response (11). Esposito et al have suggested that bacterial plaque associated inflammation of peri-implant supporting structures and consequent progressive peri-implant bone loss are the main reasons for biologically induced dental implant failures and account for up to half of all late implant failures (12, 13).

In the literature, there are numerous articles focusing on the pathogenesis of peri-implantitis cases (14). However, the knowledge regarding the histology of the condition is limited to experimentally created peri-implantitis studies (15, 16) and the histological aspects of the condition are rarely evaluated in humans (4).

According to the results of the experimental studies on peri-implantitis histology, the increased susceptibility for bone loss around implants may be related to the absence of inserting collagen fibers into the implant (16). The same finding was also observed in the current report, whereas the structure of the connective tissue revealed that there was a lack of collagen fibers running parallel to the implant surface.

Berglundh et al (17) have compared the histology of periodontitis and peri-implantitis and described a "self-limiting" process existing in the tissues around natural teeth that resulted in a protective connective tissue capsule of the supra-crestal gingival fibers of the tooth that separated the lesion from the alveolar bone. According to their findings, such a "self-limiting" did not occur in peri-implant tissues and that the lesion extended to the bony crest, which was different than the periodontitis lesions (11).Similarly, the results of the current study revealed that, adjacent to the threads of the implant, where the bone resorption macroscopically not appears, an increased concentration of osteoclasts was present. In addition, in the time of the implant removal after 7 years of insertion, the osteoclasts showed various sizes of lacunas, which refers to progressive bone destruction. Therefore, it might be concluded that, the bone loss secondary to peri-implantitis is a progressive and continual process.

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