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PERI-IMPLANTITIS - TIPS IN SURGICAL APPROACH

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Abstract

Bacterial colonization of the peri-implant sulcus, development of inflammation of the soft peri-implant tissues, followed by a progressive loss of the peri-implant bone, result in development of peri-implantits and subsequent implant failure.

The aim of this case study is to prove that successful treatment of peri-implantitis integrates conservative and surgical approach with an accent on decontamination of the implant surface, complete elimination of microorganisms and their toxins, stabilization of progressive bone loss, and regeneration of bone tissue.

Case report: In this paper initial surgical treatment of a peri-implant case is described of a 43-year-old, non-smoker, in good general health. A nonsurgical periodontal supportive therapy was firstly conducted to reduce the inflammation, followed by the surgical treatment of the defect.

After a mechanical and chemical decontamination with chlorhexidine 0.2%, a regenerative approach was adopted including application of bovine bone mineral (Cerabone) and a CTG membrane. An antibiotic therapy was associated with the treatment, according to molecular analysis findings for periodonthopatogenic bacteria.

The decision for following the newest up-dates of the surgical protocols for peri-implantitis results in predictable clinical improvement in the cases with peri-implantitis.

Key words:peri-implantitis, GBR, CTG

Introduction

Peri-implantitis is a pathological condition occurring in tissues around dental implants, characterized by inflammation in the periimplant mucosa and progressive loss of supporting bone [1-4]. The inflammation of the affected peri-implant tissues is located around osseointegrated implant in function.

The etiological factors of the peri-implantitis are divided in two groups: general and local factors. General factors include genetic factors and systemic conditions (like diabetes).

Gene polymorphisms affect gene expression, protein production and cytokine secretion, with the majority focusing on the role of IL1 [5-9].

Cross-sectional studies report an association between peri-implantitis and pre-existing systemic conditions like diabetes, cardiovascular disease, rheumatoid arthritis, osteoporosis, osteopenia, thyroid disease, hepatitis, as well as radiation and chemotherapy [10-16].

Previous history of periodontitis, smoking, lack of regular maintenance therapy accompanied with poor plaque control, lack of keratinized mucosa, excess cement, iatrogenic factors, and occlusal overload are all listed in local conditions that are responsible for peri-implantitis.

In the etiopathogenesis of peri-implantitis there are several reasons: bacterial colonization of the peri-implant sulcus, inflammation development of the soft peri-implant tissues, all that followed by progressive loss of the peri-implant bone with a subsequent implant failure. In implants where the process of inflammation of the soft and hard peri-implant tissues has started, gram negative microorganisms, spirochetes, and conventional types of periodontopathogens are present.

Peri-implant osteitis (peri-implantitis) in its initial phase has the same symptoms as peri-implant mucositis, signs of bone affection (loss of bone tissue) are added later (presence of plaque and calculus, mucosal hyperplasia, peri-implant pockets, bleeding during probing or formation of a purulent collection after probing and/or palpation, radiological finding of bone destruction, implant mobility, pain is sometimes present).

Presence of implant mobility as well as peri-implant radiolucency are signs of continuous progression of peri-implant infection reaching terminal stages characterized by complete loss of the implant-bone interface.

The aim of the successful treatment of peri-implantitis is decontamination of the implant surface with a complete elimination of microorganisms and their toxins, stabilization of progressive bone loss and regeneration of bone tissue.

In the first stage of the treatment (initial treatment), various conservative techniques can be used: manual-mechanical methods of bacterial plaque control, physical methods (diode laser -1W for 20 sec), chemical methods - local application of 0.12% chlorhexidine/citric acid/local application of tetracyclines and systemic administration of antibiotic therapy.

In the second phase, surgical methods are used - resective (bone resection and apically repositioned flap) and regenerative methods (GTR/GBR). Resective methods are generally recommended for minimal bone loss while regenerative methods are recommended for cases with greater bone loss.

Case report

A female patient, 43 years old, non-smoker, in good general health was admitted at the private dental practice Vega with a main complaint at the implant site 11. Detailed anamnesis, intraoral clinical and radiographic data was taken. The implant at the position of the left central incisor was 10 years in function; the last 3 years patient had mild pain with intermittent suppurative discharge from the implant site.

Clinical signs of inflammation including redness, edema, mucosal enlargement, BOP+ were noted. Clinical findings of CAL (clinical attachment loss) were as follows: BD=8mm, B=10mm, BM=10mm, PM=6mm, P=10mm and PD=7mm. Suppuration was present during probing (figure 1). The defect was a buccal dehiscence-type defect with circular bone resorption in presence of the lingual bone plate -Class Ic [17].

In the same visit subgingival samples and swabs were taken for molecular diagnostics (micro-IDent®plus 11 analysis) for periodontopathogenic bacteria (figure 2).

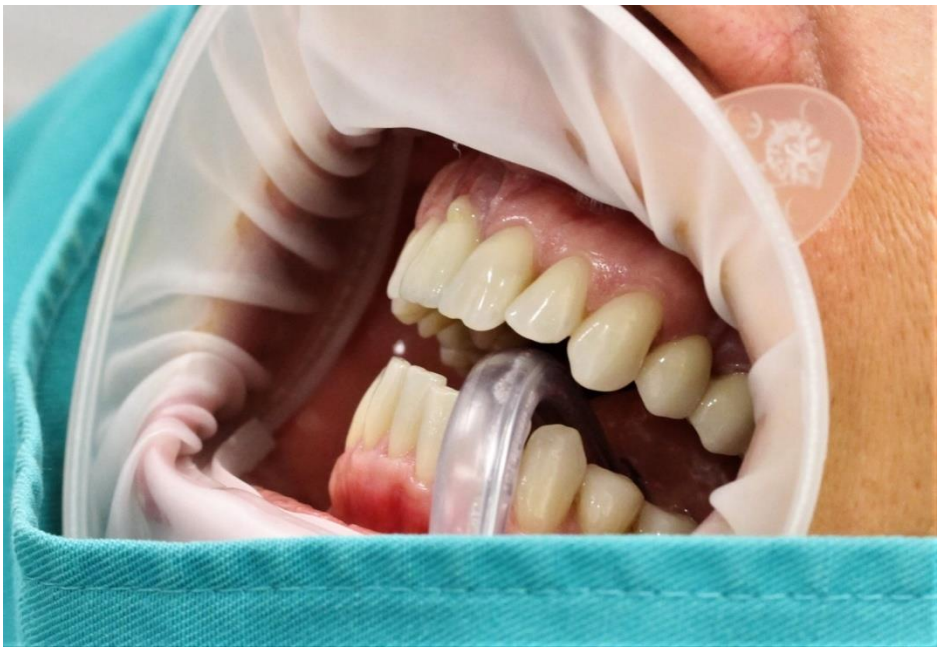
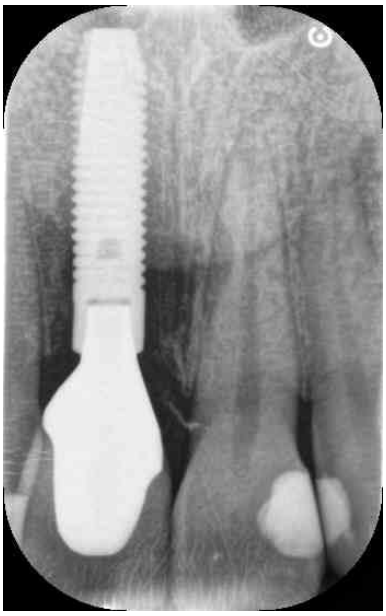


Fig 1. Suppuration during probing

bacteria	positive	negative	quantity
Aa = Aggregatibacter actinomycetemcomitans	X		+
Pg = Porphyromonas gingivalis	X		+ + +
Tf =Tannerella forsythia	X		+ + +
Td = Treponema denticola		X	-
Pi = Prevotella intermedia		X	-
Pm = Peptostreptococcus micros		X	-
Fn = Fusobacterium nucleatum/periodonticum	X		+ + +
Cr = Campylobacter rectus			-
En = Eubacterium nodatum		X	-
Ec = Eikenella corrodens		X	-
Cs = Capnocytophaga spec. (gingivalis, ochracea, sputigena)		X	-

Fig 2. Periodontopathogenic bacteria positive results



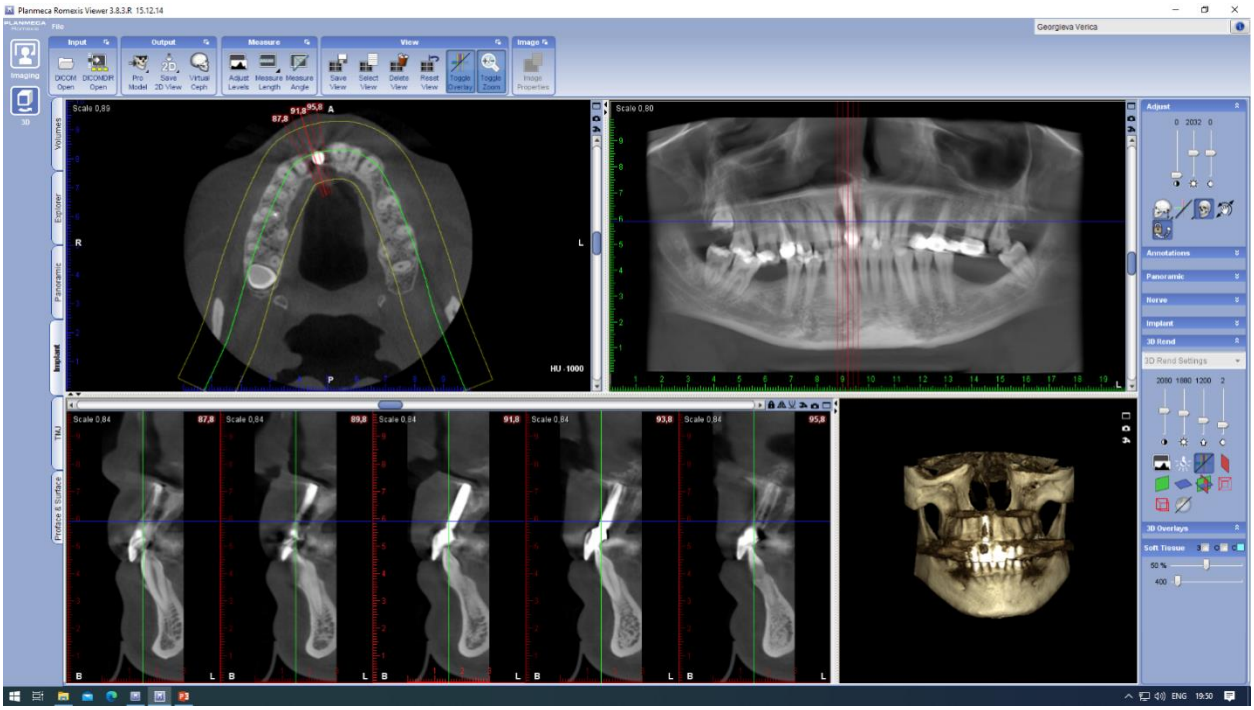


Fig 3, 4. Radiographic 3D evaluation

After the clinical and 3D CBCT radiographic evaluation **(**figure 3,4) a decision for treatment of the peri-implantitis was made.

First initial conservation treatment was focused on plaque control performing professional peri-implant hygiene, mechanical plaque elimination with carbon fiber or titanium curettes, rubber bands and polishing pastes, 0.12% chlorhexidine peri-implant sulcus irrigation, disinfection of the implant supported

prosthesis and systemic antibiotics application according to the results from the molecular analysis of periodonthopatogenic bacteria (beta lactam antibiotics Amoxicillin 3x500mg in combination with Metronidazole 3x400 mg during 7 days). For home care a protocol for personal hygiene was prescribed (4 weeks intensive irrigation with chlorhexidine and topical chlorhexidine gel application).

Surgical protocol

After 4 weeks patient demonstrated adequate plaque control and regenerative surgery treatment was performed. For local anesthesia administration Amp. Ubistesin 4% was used (figure 5). Full thickness mucoperiosteal flap was raised respecting aesthetic demands (papilla preservation technique). Granulation tissue was eliminated from bone defect with titanium curettes. Irrigation with physiological solution stopped dehydration.

The debridement and decontamination of the implant surface were performed by titanium curettes, salt-abrasian spray and ultrasonic activation of chlorhexidine solution 0.2% for 20s. Bone defect was debrided, minimal osteoplasty was done in sites with no observable bleeding and was extensively irrigated with saline and antibiotic metronidazole solution (figure 6).

Xenograft material of bovine origin (Cerabone 0.5 ml, particle size 0.5-1mm) was prepared in surgical mixing cup by adding few blood and saline drops until the mixture got consistency for easy handling and modeling.

The bone defect was fulfilled with graft material well-adapted to surrounding bone contours (figure 7). For flap mobilization periosteal incisions were performed and CTG harvested from the palate was stabilized with resorbable sutures over the graft granules (GTR/GBR regenerative surgical technique, (figure 8). The flap margins were adequately repositioned, tension-free and non-absorbable polypropylene sutures (5-0).

Patient received systemic antibiotics (Amoxicillin -clavulanic acid 1g twice daily for 14 days) and rinsed with 0.12% chlorhexidine gluconate twice daily until suture removal. Patient was instructed for oral hygiene (brushing) and continued use of 0.12% chlorhexidine rinse twice daily for another 2 weeks (figure 9).

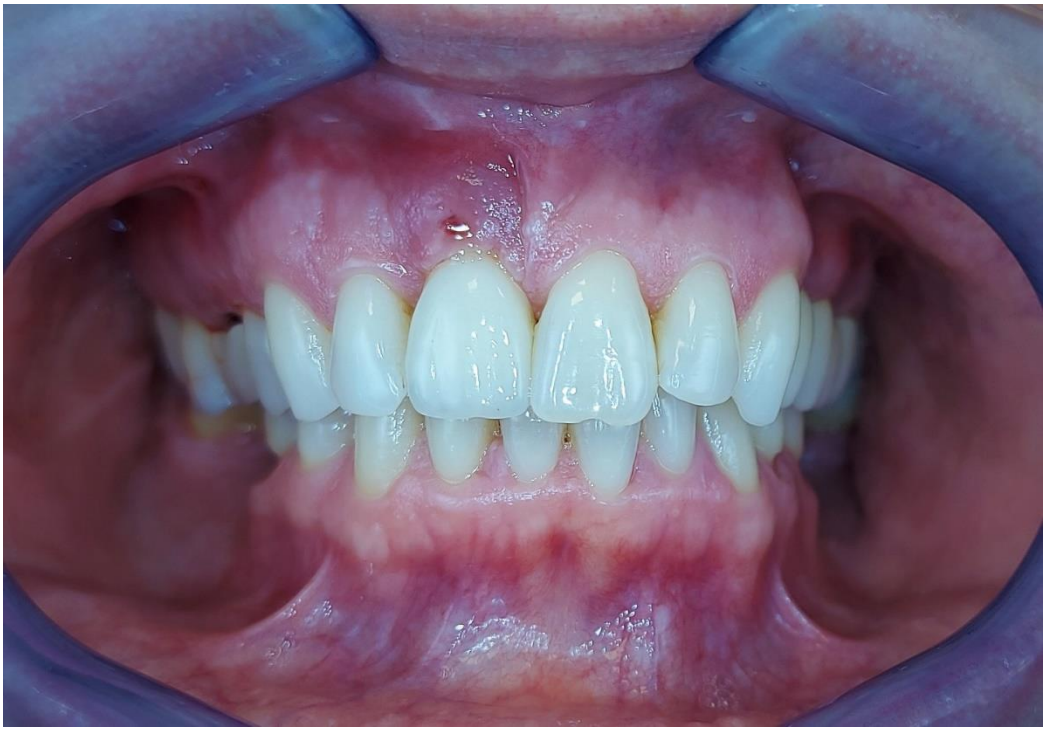


Fig 5. Initial clinical situation

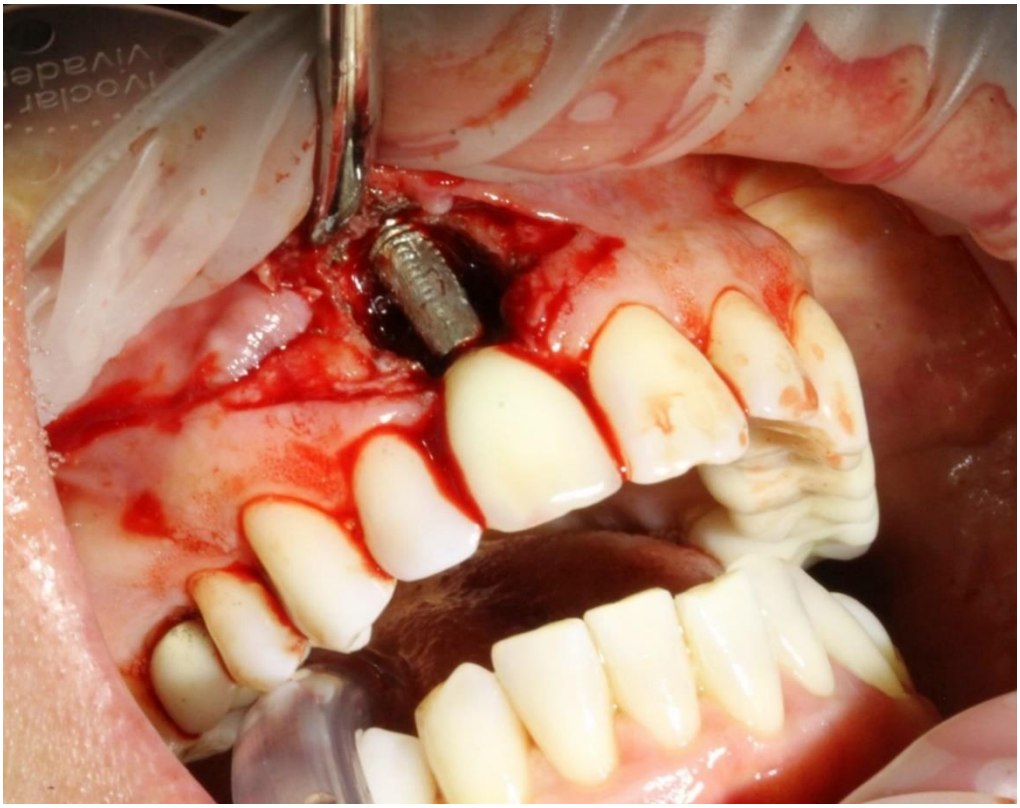


Fig 6. Debridement and decontamination of the implant surface and bone defect

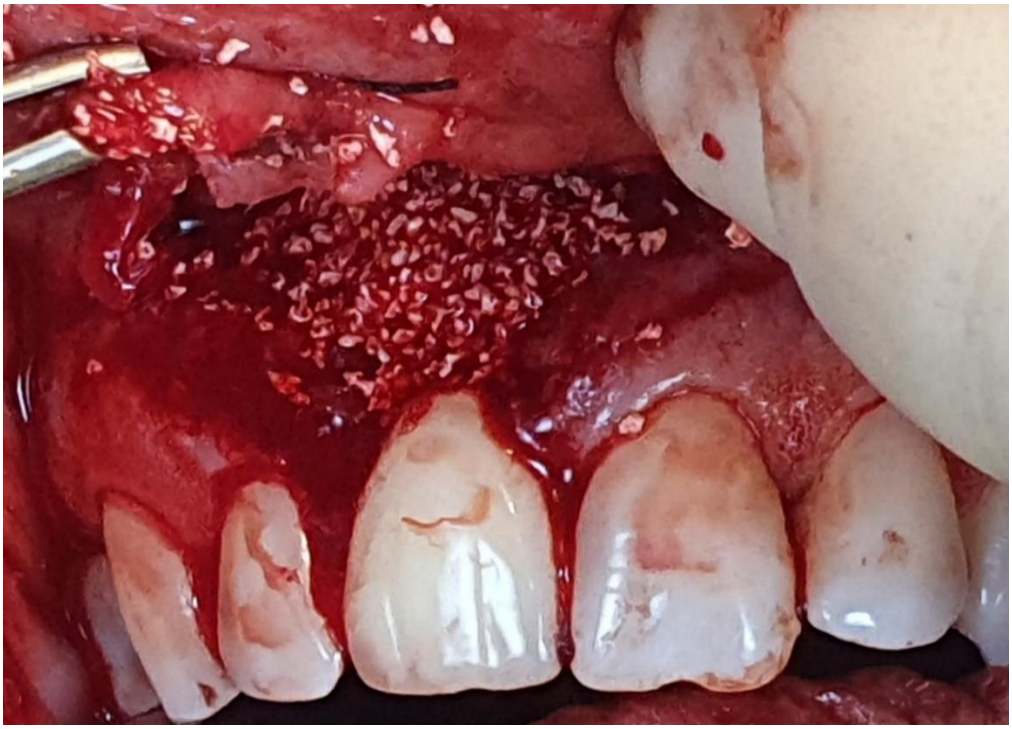


Fig 7. Bone defect was fulfilled with graft material

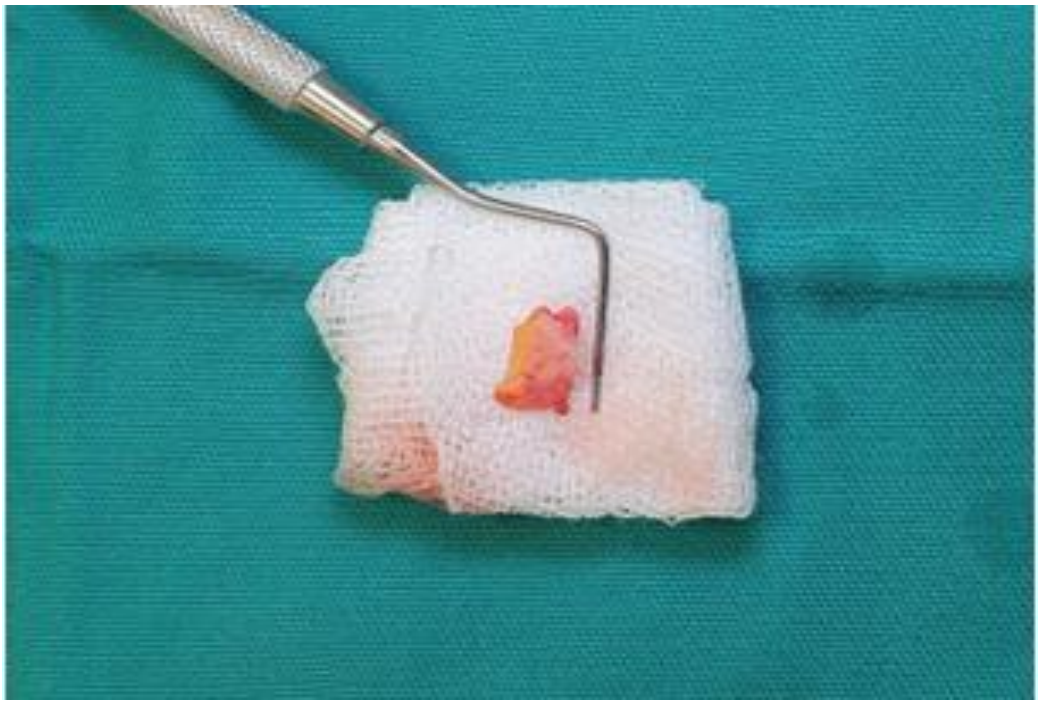


Fig 8. CTG harvested from the palate

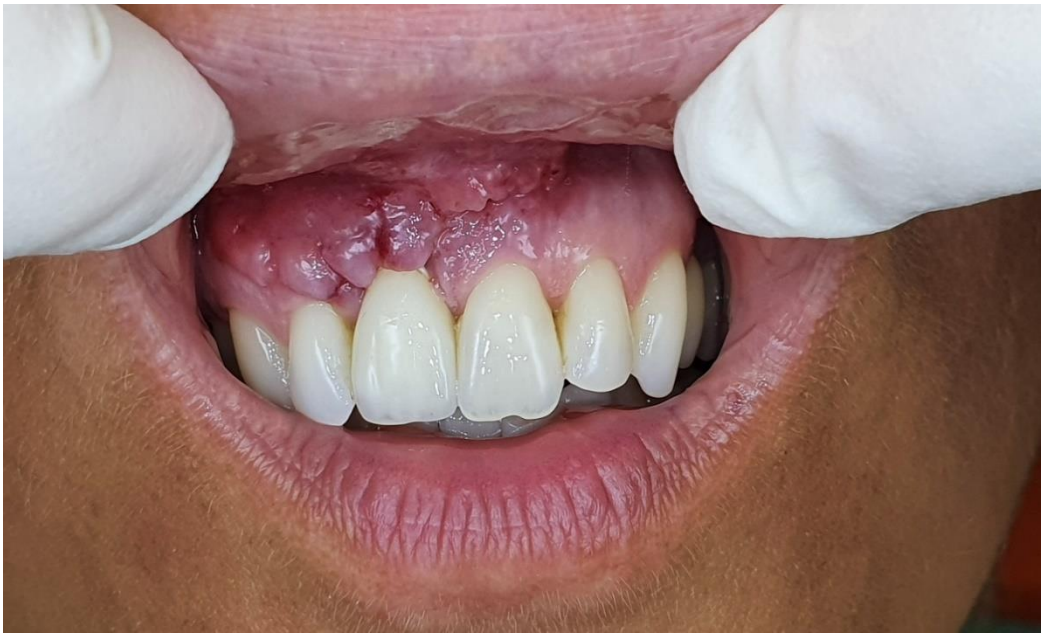


Fig 9. Suture removal 14th day post op

According to the patient agreement to visit for regular check- ups we were able to follow the clinical outcome of our proposed conservative and surgical protocol for the treatment of peri-implantitis. The patient showed evident improvement in clinical and radiological follow-up (figure 10, 11).

The six-month-evaluation follow-up showed: absence of pain, no present edema, nor suppuration.

CAL measurements: BD=3mm, B=2mm, BM=3mm, PM=3mm, P=4mm and PD=3mm.

Follow-up at 12 months: absence of pain, edema nor suppuration.

CAL: BD=3mm, B=3mm, BM=3mm, PM=3mm, P=4mm and PD=3mm.

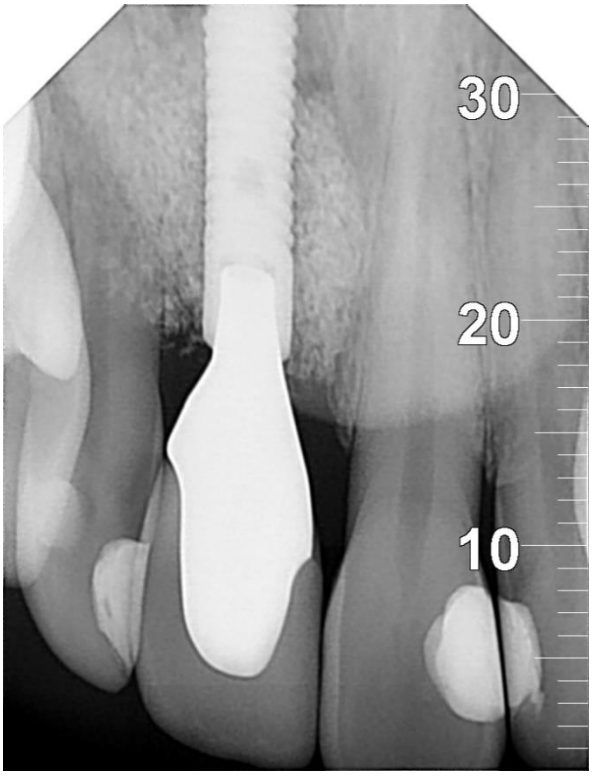


Fig 10. Radiographic evaluation after 6 months

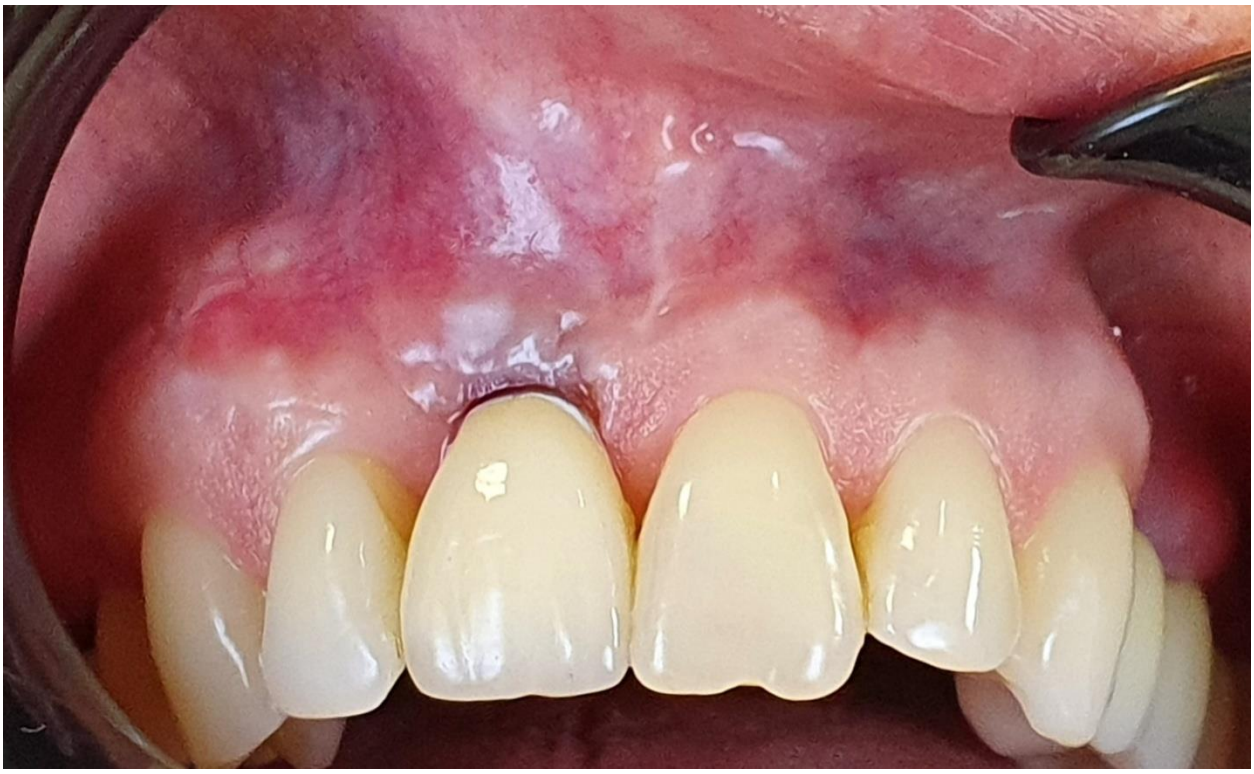


Fig 11. Clinical appearance after 6 months

Discussion

Microbiology of peri-implant infection

The microorganisms present in the oral cavity also determine the peri-implant microbiology: the colonization of implants is similar to that of the periodontal sulcus after dental eruption. A healthy osseointegrated implant in function is colonized by the bacterial flora present in the oral cavity, including aerobic bacteria, coccoid bacteria, gram positive species of microorganisms and an extremely small number of periodontopathogenic bacteria.

When the implant is in condition of pathological overloads, local loss of osseointegration occurs as a result of bone microfractures and an apical migration of the peri-implant junctional epithelium starts favoring bacterial infection.

Peri-implant disease is multifactorial disease that mainly have a bacterial etiology. Microorganisms that have been detected as causative agents of periodontal disease have also been found in the soft tissues around implants that are affected by peri-implant disease.

In implants, where the process of inflammation of the soft and hard peri-implant tissues has started, gram negative microorganisms, spirochetes and conventional types of periodontopathogens prevail: Porphyromonas gingivalis, Bacteroides forsytus (Tannarella forsytus), Fusobacterium nucleatum, Streptococcus intermedius, Peptostreptococcus micro, Aggregatibacter actinomycetemcomitans, Micromonas micros, Prevotella intermedia, and Dialister pneumosyntes.

Using both conventional DNA probe and cultural analysis, common periodontopathogenic bacteria have been isolated at both healthy and diseased implant sites, and the distribution of the detected species did not markedly differ from the clinical implant status (i.e. healthy, peri-implant mucositis, peri-implantitis).

However, when compared with healthy implant sites alone, peri-implantitis was associated with higher counts of nineteen bacterial species, including Porphyromonas gingivalis and Tannerella forsythia [18-20]. Other studies indicated that peri-implantitis was more frequently linked with opportunistic pathogens, such as Pseudomonas aeruginosa and Staphylococcus aureus fungal organisms (Candida albicans, Candida boidinii, Penicillium spp.,) and viruses (human cytomegalovirus, Epstein-Barr virus), thus pointing to a complex and heterogenous infection [21].

A lot of systematic reviews have focused on the correlations between various cytokines (proinflammatory/osteoclastogenesis related) measured in the peri-implant crevicular fluid (PICF) and the clinical condition at implant sites.

Accordingly, the systematic reviews indicated that the assessment of proinflammatory cytokines (IL1β and TNFα levels) in the PICF might be of beneficial value to differentiate between peri-implant health and disease [22].

Other studies correlated current periodontitis with peri-implantitis, also reporting strong associations, especially in patients with a history of periodontitis.

The results also showed that treatment of peri-implantitis was more time-consuming in patients with a previous history of periodontitis [23].

Taking into consideration all these data from the literature review, we initially decided to determine the presence of the periodothopatogenic bacteria by molecular diagnostics. The test confirmed the presence of periodonthopatogenic bacteria from the red complex (with the concentration +, one plus signifies microbial count of 10^4) and pink complex (with the concentration +++).

The selection of the individual personal therapy is based primarily on the clinical findings and pathogenic bacteria detection. Individual therapy is also a combination of the mechanical and chemical debridement, and systemic and local antibiotic administration.

If molecular analysis shows presence of bacterial species from one bacterial complex, then an adequate therapy is prescribed according to the resistance/sensitiveness of that bacterial species.

When different types of pathogens are present from different bacterial complexes, then a combined therapy of antibiotics is used. In case of antibiotic allergy, an alternative antibiotic therapy should be recommended.

Ultrasonic activation of antiseptics

Irrigating solutions during surgical treatment of the peri-implantitis cases function as disinfectant and cleaning agent during biomechanical debridement, improving the elimination of the contaminated soft and peri-implant hard tissues. Irrigation is conducted by slow dispensing of the irrigant of choice onto the implant surface and surgical field. Passive irrigation has some limitations in circulation, cleansing potential of the irrigation solution and its penetration [24].

So, different ultrasonic techniques have been proposed to improve irrigant distribution, such as irrigation combined with simultaneous ultrasonic instrumentation [25].

Weber et al. [26] evidenced prolonged antimicrobial action of chlorhexidine when applied with ultrasonic activation. Ultrasound activation of chlorhexidine in three cycles of 20 second applications, was just as effective in eliminating the debris as the laser activated techniques [27].

In our clinical case we included in the protocol ultrasonic activation of the antiseptic solution chlorhexidine 0.2% in two working cycles of 20s, in order to improve its cleansing potential, antimicrobial efficiency, penetration and elimination of the surgical debris.

Connective tissue graft benefits

The concept of guided tissue and bone regeneration (GTR/GBR) rests on the following principles: maintenance and protection of the blood clot source of the bone regeneration; maintenance of the space required for bone regeneration (this space will promote angiogenesis and bone formation), placement of a barrier to cellular invasion of the gingival epithelium (prevention of cellular competition), and adequate placement of surgical sutures to maintain successful management of underlying postoperative forces [28].

Regeneration occurs due to using bone grafts, bone substitutes, guided tissue regeneration and resorbable or a non-resorbable barrier membrane to prevent the migration of epithelial cells and gingival tissues to the wound area.

Bone grafts are used in the regenerative surgical treatment of peri-implantitis because of their osteoconductive or osteoinductive properties and to maintain the space under the membrane preventing it from collapsing into the bone defect, facilitate the wound stability and provide space to enable the regeneration process [29, 30].

Bone graft materials from bovine origin (xenografts) have a high osteoconductive property, are tolerable by the receiver site tissues and do not cause allergic reactions.

Gingival connective tissue cells contain mesenchymal cells and have osteogenic, chondrogenic and osteoblastic capacity. These cells are also able to modulate the immune system [30]. Gingival tissue is a richer source of mesenchymal stem cells in comparison with bone marrow [31].

Palatal autogenous connective tissue graft (CTG) has some advantages like lower cost, availability and adaptability. Periosteal and non-periosteal connective tissue grafts have been used in regenerative treatments and both show successful results [3].

CTG can function as a biological barrier membrane, it prevents the epithelial cells from proliferation into the lesion site and no proliferation of a tissue graft of this type into the lesion was observed [30, 32].

The periosteum, as a structure rich in osteoprogenitor cells, has the ability to stimulate osteogenesis in the area. Osteogenic progenitor cells available in the periosteum work with osteoblasts in initiating the cell-differentiation process of bone repair [32].

All these studies indicate that the connective tissue itself or with periosteum, has the ability to promote the regeneration process. Xenografts and palatal connective tissue grafts with and without periosteum can be equally effective in peri-implant defect regeneration.

Conclusion

Precise diagnostics, following the newest up-dates of the surgical protocols for peri-implantitis should result in predictable clinical improvement, re-osseointegration of the implant, extending the longevity of the implant in function and promotion of the patient's quality of life.

References:

1. Lang NP, Berglundh T, Working Group 4 of Seventh European Workshop on P. Peri-implant diseases: where are we now?–Consensus of the Seventh European Workshop on Periodontology. J Clin Periodontol. 2011;38 Suppl. 11:178–181.
2. Sanz M, Chapple IL, Working Group 4 of the VEWoP. Clinical research on peri-implant diseases: consensus report of Working Group 4. J Clin Periodontol. 2012;39 Suppl 12:202–206
3. Jepsen S, Berglundh T, Genco R, et al. Primary prevention of peri-im- plantitis: managing peri-implant mucositis. J Clin Periodontol. 2015;42 Suppl. 16:S152–157.
4. Lindhe J, Meyle J, Group DoEWoP. Peri-implant diseases: consensus report of the Sixth European Workshop on Periodontology. J Clin Periodontol. 2008;35 Suppl. 8:282–285.
5. Laine ML, Leonhardt A, Roos-Jansaker AM, et al. IL-1RN gene poly- morphism is associated with peri-implantitis. Clin Oral Implants Res. 2006;17:380–385.
6. Gruica B, Wang HY, Lang NP, Buser D. Impact of IL-1 genotype and smoking status on the prognosis of osseointegrated implants. Clin Oral Implants Res. 2004;15:393–400.
7. Garcia-Delaney C, Sanchez-Garces MA, Figueiredo R, Sanchez-Torres A, Gay-Escoda C. Clinical significance of interleukin-1 genotype in smoking patients as a predictor of peri-implantitis: A case-control study. Med Oral Patol Oral Cir Bucal 2015;20:e737–743.

8. Hamdy AA, Ebrahem MA. The effect of interleukin-1 allele 2 genotype (IL-1a(-889) and IL-1b(+3954)) on the individual's susceptibility to peri- implantitis: case-control study. J Oral Implantol. 2011;37:325–334.
9. Lachmann S, Kimmerle-Muller E, Axmann D, Scheideler L, Weber H, Haas R. Associations between peri-implant crevicular fluid volume, concentrations of crevicular inflammatory mediators, and composite IL-1A -889 and IL-1B +3954 genotype. A cross-sectional study on implant recall patients with and without clinical signs of peri-implantitis. Clin Oral Implants Res. 2007;18:212–223
10. de Araujo Nobre M, Mano Azul A, Rocha E, Malo P. Risk factors of peri-implant pathology. Eur J Oral Sci. 2015;123:131–139.
11. Renvert S, Aghazadeh A, Hallstrom H, Persson GR. Factors related to peri-implantitis—a retrospective study. Clin Oral Implants Res. 2014;25:522–529.
12. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. Clin Oral Implants Res. 2017;28:144–150.
13. Máximo MB, de Mendonca AC, Alves JF, Cortelli SC, Peruzzo DC, Duarte PM. Peri-implant diseases may be associated with increased time loading and generalized periodontal bone loss: preliminary re- sults. J Oral Implantol. 2008;34:268–273.
14. Daubert DM, Weinstein BF, Bordin S, Leroux BG, Flemming TF. Prevalence and predictive factors for peri-implant disease and implant failure: a cross-sectional analysis. J Periodontol. 2015;86:337–347.
15. Canullo L, Penarrocha-Oltra D, Covani U, Botticelli D, Serino G, Penarrocha M. Clinical and microbiological findings in patients with peri-implantitis: a cross-sectional study. Clin Oral Implants Res. 2016;27:376–382.
16. Dvorak G, Arnhart C, Heuberer S, Huber CD, Watzek G, Gruber R. Peri-implantitis and late implant failures in postmenopausal women: a cross-sectional study. J Clin Periodontol. 2011;38:950–955.
17. Schwarz F, Herten M, Sager M, Bieling K, Sculean A, Becker J. Comparison of naturally occurring and ligature-induced peri-implantitis bone defects in humans and dogs. Clin Oral Implants Res. 2007;18:161–170
18. Casado PL, Otazu IB, Balduino A, de Mello W, Barboza EP, Duarte ME. Identification of periodontal pathogens in healthy peri-implant sites. Implant Dent. 2011;20:226–235.
19. Renvert S, Roos-Jansaker AM, Lindahl C, Renvert H, Rutger Persson G. Infection at titanium implants with or without a clinical diagnosis of inflammation. Clin Oral Implants Res. 2007;18:509–516.
20. Persson GR, Renvert S. Cluster of bacteria associated with peri-im- plantitis. J Res. 2016;51(6):689–698
21. Jankovic S, Aleksic Z, Dimitrijevic B, Lekovic V, Camargo P, Kenney B. Prevalence of human cytomegalovirus and Epstein-Barr virus in sub- gingival plaque at peri-implantitis, mucositis and healthy sites. A pilot study. Int J Oral Maxillofac Surg. 2011;40:271–276
22. Faot F, Nascimento GG, Bielemann AM, Campao TD, Leite FR, Quirynen M. Can peri-implant crevicular fluid assist in the diagnosis of peri-implantitis? A systematic review and meta-analysis. J Periodontol. 2015;86:631–645.
23. Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Effectiveness of implant therapy analyzed in a Swedish population: prevalence of peri-implantitis. J Dent Res. 2016;95:43–49.
24. Gulabivala K, Ng YL, Gilbertson M, Eames I. The fluid mechanics of root canal irrigation. Physiol Meas. 2010;31(12):R49-84.
25. Lee SJ, Wu MK, Wesselink PR. The effectiveness of ultrasonic irrigation to remove artificially placed dentine debris from different-sized simulated plastic root canals. Int Endod J 2004;37:607-12.

26. Weber C, Scott B, McClanahan SB, Miller GA, Diener-West M, Johnson JD. The effect of passive ultrasonic activation of 2% chlorhexidine or 5,25% sodium hypochlorite on residual antimicrobial activity in root canals. J Endod. 2003;29:562-4
27. de Moor RJ, Blanken J, Meire M, Verdaasdonk R. Laser induced explosive vapor and cavitation resulting in effective irrigation of the root canal. Part 2: evaluation of the efficacy. Lasers Surg Med. 2009;41:520-3.
28. Ibrahim Elgali,Omar Omar,Christer Dahlin,Peter Thomsen (2017) Guided bone regeneration: materials and biological mechanisms revisited, Volume125, Issue5, 315-337
29. Moghaddas H, Soltani L, Moghaddas O. Efficacy of palatal connective tissue graft as a membrane in the treatment of intrabony defects. J Periodontol Implant Dent 2010;2:70-6.
30. Esfahanian V, Moghaddas H, Moghaddas O. Efficacy of connective tissue as a membrane with an organic bone using platelet-rich plasma in the treatment of intrabony vertical defects. Journal of Isfahan Dental School 2012;8:1-17.
31. Tomar GB, Srivastava RK, Gupta N, Barhanpurker AP, Pote ST, Jhaveri HM, et al. Human gingiva-derived mesenchymal stem cells are superior to bone marrow derived mesenchymal stem cells for cell therapy in regenerative medicine. Biochem Biophys Res Commun 2010;393:377-83
32. Finley JM, Acland RD, Wood MB.Revascularized periosteal grafts--a new method to produce functional new bone without bone grafting. Plast Reconstr Surg 1978;61:1-6