CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.2691377

Available online at: http://www.iajps.com ***Research Article***

MANAGEMENT APPROACH OF PERI-IMPLANTITIS

[1]Dr. Israa Yahya Al-kabsi , [2]Dr. Awad Ali Al-azmi, [3]Dr. Areif Salmah Al-Atwi, [4]Dr. Ahmad Hussain Al-neami

Article Received: March 2019 **Accepted:** April 2019 **Published:** May 2019

Abstract:

The aim of this review, to discuss the definition and characteristics of peri-implant disease and specifically per-implantitis, in order to better understand the background and different management techniques emphasized as well. We conducted a detailed review over the literature using electronic databases as; MEDLINE, and EMBASE for studies involving data on dental per-implantitis management approaches, published in English language up to January, 2019. Indicators of peri-implant diseases resemble signs of gum illness: red or tender gums around the implants, or bleeding when cleaning. And much like your natural teeth, implants need regular tooth cleaning and flossing and normal examinations from a dental expert. Various other risks aspects for developing peri-implant disease include previous gum ailment medical diagnosis, poor plaque control, smoking, and diabetes mellitus. It is essential to routinely check dental implants as part of an extensive periodontal analysis. If undiagnosed, preimplantation disorder might lead to total loss of osseointegration and implant loss.

Corresponding author:
Dr. Israa Yahya Al-kabsi,

QR code

Please cite this article in press Israa Yahya Al-kabsi et al., ***Management Approach Of Peri-Implantitis.,*** *Indo Am. J. P. Sci, 2019; 06(05).*

INTRODUCTION:

Peri-implant illness following effective integration of an end-osseous implant is the outcome of a disproportion between microbial load and host protection. Peri-implant disorders might impact the preimplant mucosa only (peri-implant mucositis) or additionally include the supporting bone (peri-implantitis) [1]. Bleeding on probing (BOP) is constantly existing with peri-implant disorder [1]. Various other clinical indications of disease may include suppuration, raised probing depths relative to baseline, mucosal recession, a draining sinus (fistula) and peri-implant mucosal swelling/ hyperplasia. If undiagnosed, preimplant ailment may lead to complete loss of osseointegration and implant loss.

Food impaction around natural or artificial teeth is a well-recognized concern in dental care. The Glossary of Periodontal Terms specifies food impaction as "the forceful wedging of food right into the interproximal area by masticatory pressure (vertical impaction) or the forcing of food interproximal by tongue or cheek stress (horizontal impaction) " [2]. For implant restorations, this can likewise include the wedging of food into the preimplant sulcus. Usual foods that are empirically associated with food impaction include popcorn, seeds, legumes, and nuts. The hull (husk) found in a lot of seeds, consisting of sunflower seeds, is primarily composed of cellulose, a polysaccharide that cannot be broken down by human enzymes. The difference in the orientation of supracrustal connective tissue between natural teeth and dental implants is well understood [3]. The fibers around the implants run parallel to the abutment surface and only follow the joint surface instead of being attached [3]. Consequently, the preimplant sulcus may be inclined to food and foreign body impactions.

The aim of this review, to discuss the definition and characteristics of peri-implant disease and specifically per-implantitis, in order to better understand the background and different management techniques emphasized as well.

METHODOLOGY:

We conducted a detailed review over the literature using electronic databases as; MEDLINE, and EMBASE for studies involving data on dental per-implantitis management approaches, published in English language up to January, 2019. we then reviewed the references lists of included studies to find more relevant articles to be for additional evidence.

DISCUSSION:

- **Peri-Implantitis Characteristics:**

Histopathologic characteristics of naturally occurring peri-implantitis:

The histopathologic characteristics of naturally taking place peri-implantitis lesions have been thoroughly evaluated in human biopsy materials [4-6]. When compared with peri-implant mucositis, the lesions at peri-implantitis sites (situation interpretation: BOP+, suppuration, radiographic bone loss) harbored more neutrophil granulocytes and larger "percentages of B cells (CD19+)" [10]. Similar to periodontitis, the lesions at peri-implantitis sites were likewise dominated by plasma cells and lymphocytes, but identified by larger proportions of polymorphonuclear leukocytes and macrophages [5]. Lately, it was likewise shown that the dimensions of peri-implantitis sores (case interpretation: interproximal implant sites with BOP+ and probing depth (PD) ≥ 7 mm) was more than two times as big as that noted at periodontitis sites (3.5 mm2 vs. 1.5 mm2) [6]. Furthermore, peri-implantitis lesions were identified by bigger area percentages, numbers and thickness of plasma cells, macrophages and neutrophils, in addition to a greater density of vascular frameworks outside and lateral to the cell infiltrate [5]. One more research study utilizing immunohistochemical analysis of harvested soft tissue biopsies revealed that IL-1 was a leading osteoclast activating cytokine at peri-implantitis sites [4]. It should be emphasized that the above evaluations of human peri-implant tissue biopsies did, for ethical reasons, not consist of the osseous part of the sites.

Microbiologic and immunologic characteristics of naturally occurring peri-implantitis:

Practicing traditional DNA probe and cultural analyses, usual periodontopathogenic bacteria have been separated at both healthy and balanced and infected implant sites, 40 and the circulation of the found varieties did not substantially differ by medical implant status (i.e. healthy, peri-implant mucositis, periimplantitis) [7]. Nevertheless, when compared with healthy implant sites alone, peri-implantitis was related to higher counts of 19 bacterial varieties, consisting of Porphyromonas gingivalis and Tannerella forsythia [8]. Additionally, observational studies have suggested that peri-implantitis was much more often linked with opportunistic pathogens such as Pseudomonas aeruginosa and Staphylococcus aureus (S. aureus), fungal organisms (e.g. Candida albicans, Candida boidinii, Penicillum spp., Rhadotorula laryngis, Paelicomyces spp.), and viruses (i.e. human cytomegalovirus, Epstein-Barr virus), thus pointing to a rather complex and heterogenous infection [9], [10]. It ought to be highlighted that the

submucosal microbiota of peri-implantitis lesions have not been extensively examined using culture-independent methods. Thus, the microbial picture related to peri-implantitis ought to be regarded as incomplete.

Clinical characteristics of naturally occurring peri-implantitis:

Medical indications of inflammation consisting of redness, edema, mucosal enlargement, BOP+ with or without suppuration in addition to rises in PD and radiographic bone loss are typically utilized in case meanings for peri-implantitis [3-6]. Implant sites diagnosed with peri-implantitis generally reveal raised PD. In a research study examining 588 patients with 2,277 implants after a function time of 9 years, PD ≥ 6 mm was documented at 59% of all implants presenting with moderate/severe peri-implantitis (case interpretation: BOP+ and bone loss > 2 mm) [11]. Out of the implants categorized as healthy and balanced (case definition: BOP-) or detected with mucositis (case meaning: BOP+ however no bone loss > 0.5 mm), 3% and 16% revealed PD ≥ 6 mm, respectively. It was also taken note that the frequency of implants showing PD ≥ 6 mm raised with raising severity of peri-implantitis.

In a cross-sectional analysis, Schwarz et al. examined a total of 238 patients (n = 512 implants) after a median function time of 23 months (1 to 80 months) [12]. At peri-implant mucositis sites (case interpretation: BOP+ on at the very least one aspect of the implant), the regularity of BOP scores mainly varied in between 33% and 50%, while the peak was 67% at periimplantitis sites (case definition: BOP+ and/or suppuration and modifications in the radiographic bone level compared to baseline). Diseased implant sites were related to greater frequencies of 4 to 6 mm PD than implants with a healthy and balanced preimplant mucosa, with an equivalent distribution in between mucositis and peri-implantitis sites. PD values of ≥ 7 mm were only observed at one implant diagnosed with peri-implantitis [12]. In this context, it needs to be realized that the determination of what constitutes a physiological PD at implant sites is difficult. A recent evaluation explained a high degree of variation in the vertical mucosal density estimated at healthy implant sites, ranging from 1.6 to 7.0 mm (i.e. mucosal margin to the crestal bone degree) [13]. One cross-sectional analysis additionally reviewed and compared the horizontal mucosal thickness (hMT) at healthy and diseased implant sites. Median hMT were substantially enhanced at diseased, when compared to healthy implant sites (1.1 mm), however were comparable at mucositis and peri-implantitis sites (i.e. 1.7 vs. 1.6 mm), respectively. In all groups investigated, these values did not markedly differ by implant area (i.e., upper/lower jaws) or position (i.e., anterior/posterior sites) [14].

Periapical peri-implantitis:

Besides peri-implant infections at sites with deepened probing depths, a variety of instance series also reported on the occurrence of periapical peri-implantitis lesions. The affected implants were generally identified by a periapical radiographic radiolucency with or without concomitant clinical indications of inflammation, such as redness, edema, fistula and/ or abscess development [15]. These clinical and radiographic signs of inflammation were noted between 2 to 8 weeks and approximately 4 years after implant placement [15], [16], [17]. Most of the research studies reported a straight connection between retrograde periimplantitis and the existence of periapical endodontic sores at nearby teeth [15], [16], [17].

Oral-mucosal lesions mimicking peri-implantitis:

Case reports have explained a range of oral-mucosal lesions at dental implants that might mimic peri-implant ailments. Such lesions include primary malignant tumors (i.e. oral squamous cell carcinoma) or metastases in addition to gigantic cell and pyogenic granuloma [18-20]. While these pathologic conditions share numerous medical attributes with peri-implant illness, they reveal distinct distinctions to a nonspecific inflammation at the histopathologic level [18-20].

- **Management modalities of peri-implantitis:**

Local debridement:

The implant must be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss, interdental brushes, or using plastic scaling instruments. These have been revealed not to roughen the implant surface area unlike metal and ultrasonic scalers [21]. Although implant surface area damages can almost be prevented by utilizing either ultrasonic scalers with a nonmetallic tip or resin/carbon fiber curettes, the presence of implant threads and/or implant surface area roughness might compromise the accessibility for cleansing [21].

The study by Karring et al. showed that sub-mucosal debridement alone, completed by using either an ultrasonic tool or carbon fiber curettes, is not sufficient for the decontamination of the surface areas of

implants with peri-implant pockets ≥ 5 mm and exposed implant threads [22]. So it seems affordable to recommend that mechanical or ultrasonic debridement alone may not be a sufficient modality for the resolution of peri-implantitis.

Implant surface decontamination:
Four implant surface purification approaches were compared in a monkey model: (1) air-powder abrasive strategy adhered to by citric acid application, (2) air-powder abrasive technique, (3) gauze soaked in saline complied with by citric acid application, and (4) gauze soaked at the same time in 0.1% chlorhexidine and saline [23]. Medical criteria, radiography (consisting of quantitative electronic subtraction radiography), histology, and stereology did not reveal substantial differences in between any one of the techniques utilized. Findings from an in vitro study integrating photosensitization by toludine blue solution and soft laser irradiation have indicated that removal of bacteria from different titanium surfaces without modification of the implant surface was feasible [23].

Photodynamic therapy is a non-invasive approach that could be made use of to minimize microorganisms in peri-implantitis [26]. 2% chlorhexidine or 3% hydrogen peroxide can be utilized as topical antiseptics. Purification of impacted implants with titanium plasma-sprayed or sandblasted/acid-etched surface areas may most conveniently and efficiently be attained by applying gauze soaked alternately in chlorhexidine and saline [21].

The non-surgical therapy of peri-implantitis lesions making use of an erbium-doped: yttrium, aluminum, and garnet (Er: YAG) laser showed lower counts of F. nucleatum 1 month after treatment [26]. According to Schwarz et al., the Er: YAG laser and the combination of mechanical debridement/chlorhexidine are similarly effective at 6 months after treatment in dramatically strengthening peri-implant probing pocket depth and clinical attachment level, however using the Er: YAG laser offers a substantially greater decrease of bleeding on probing compared with the adjunctive application of chlorhexidine [24]. Nonetheless, in a subsequent research study by Schwarz et al., the efficacy of the Er: YAG laser seemed limited to a 6-month period, particularly for innovative peri-implantitis sores [25]. It was further recommended that a single course of treatment with the Er: YAG laser might not suffice for attaining a secure treatment of peri-implantitis and that added restorative procedures, such as additional use of the Er: YAG laser and/or succeeding osseous regenerative treatments, might be called for.

Anti-infective therapy:
Specific microbial data concerning the presence of putative pathogens is indispensible to make a meaningful choice pertaining to systemic or local antibiotic treatment. Although the composition of the subgingival microbial component is important for the choice of the medicine, oral distribution patterns of prospective microorganisms are additionally vital in deciding whether an antimicrobial agent must be provided locally or systemically. To achieve this task, medical professional needs to consider the periodontal condition of the residual teeth.

The study by Schwarz et al. demonstrated that the therapy of peri-implant infection by mechanical debridement with plastic curettes incorporated with antiseptic (0.2% chlorhexidine) treatment might bring about statistically considerable improvements in hemorrhaging on probing, peri-implant probing pocket depth, and clinical attachment level at 6 months compared with baseline [24]. A research by Renvert et al. showed that the addition of disinfectant therapy to mechanical debridement does not provide adjunctive advantages in shallow peri-implant sores where the mean probing pocket depth was <4 mm [27]. Therefore, it appears that the addition of antibacterial treatment to mechanical debridement does not offer adjunctive benefits in shallow peri-implant sores with mean pocket probing depth <4 mm but seems to provide additional clinical improvements in deep peri-implant lesions with mean pocket probing depth >< 4 mm however seems to offer extra clinical enhancements in deep peri-implant lesions with mean pocket probing depth > 5 mm. Patients suffering from localized peri-implant issues in the lack of various other infections might be candidates for treatment by local drug-delivery devices. Local application of antibiotics by the insertion of tetracycline fibers for 10 dayscan provide a sustained high dosage of the antimicrobial agent specifically right into the damaged site for numerous days [28]. The use of minoccline microspheres as an adjunct to mechanical therapy is beneficial in the treatment of peri-implant lesions, but the therapy might have to be duplicated [28]. The research by Renvert et al. showed that the adjunctive benefits originated from the addition of an antibiotic minocycline to mechanical debridement tend to be better, although to a limited extent, than those accomplished by the integrated use a disinfectant (chlorhexidine) and mechanical debridement [27]. The enhancements in peri-implant probing depths acquired

by the adjunctive use minocycline can be preserved throughout a temporary duration of 12 months. In the research by Renvert et al., the displayed bone loss was not more than three implant threads [27].

If the problem is generalized, details microbiological data is accumulated, and antibiotics are carried out systemically. Lang et al. recommend the adhering to antibiotic routines: systemic ornidazole 500 mg bd for 10 days or metronidazole 250 mg td for 10 days or a once daily combination of metronidazole 500 mg and amoxicillin 375 mg for 10 days [28]. If peri-implantitis is connected with persisting periodontal ailment, then both conditions need to be dealt with. In this case, the adjunctive use of systemic antibiotics may be taken into consideration. There are no medical trials offered nowadays on the systemic administration of antibiotics for the therapy of peri-implantitis.

Given that mechanical and antibacterial procedures are complied with prior to carrying out antibiotic therapy, it shows up that superficial peri-implant infection might be effectively regulated utilizing antibioics [1]. However it is still open to question whether much deeper peri-implant lesions can be sufficiently dealt with non-surgically by a mix of a local antibiotic and mechanical debridement.

Surgical technique:
Surgical resection is usually confined to implants positioned in non-aesthetic sites. Surgical flap aids in extensive debridement and purification of the influenced implant. Surgical therapy was carried out, making use of: (1) autogenous bone grafts covered by membrane layers, (2) autogenous bone grafts alone, (3) membranes alone, and (4) a control access flap treatment revealed that defects treated with membrane-covered autogenous bone demonstrated considerably larger amounts of bone regrowth and reosseointegration than those treated with the various other three treatments [21]. Nonetheless, membrane exposure is a regular difficulty after such procedures. Exposure of porous e-PTFE membrane layers might lead to bacterial penetration and bring about infection [21].

Today, no randomized controlled medical tests are offered on the use of access flap surgical procedure (open-flap debridement) alone for the therapy of periimplantitis. A randomized comparative clinical trial by Romeo et al. ended that resective surgical procedures combined with implantoplasty can have a favorable influence on the survival rates of rough-surfaced implants influenced by peri-implantitis along with on peri-implant clinical parameters, such as pocket-probing depth, suppuration, and sulcus bleeding [29]. The research study by Schwarz et al. showed that both nanocrystalline hydroxyapatite and guided bone regeneration supplied clinically considerable improvements in clinical specifications complying with 6 months of non-submerged healing [30]. The 2-year outcomes by Schwarz et al. of the very same clinical research study once again demonstrated that both treatment methods were efficacious in providing clinically substantial reductions of pocket-probing depth and gains in clinical attachment degree, however the application of the combination of natural bone mineral and collagen membrane layer appeared to correlate with higher improvements in those medical specifications and, therefore, was related to a more foreseeable and enhanced healing result [31]. Unfortunately, the fairly small example size of the research study (22 patients) did not allow a reputable analytical contrast of the efficacy of both restorative treatments. In general, extra information on different regenerative strategies for dealing with peri-implantitis must be collected.

Explantation:
If there is advanced bone loss and the implant cannot be saved, it needs to be eliminated. If a choice has been made to get rid of the implant, explanation trephines are readily available to match the dental implant system concerned. It needs to be kept in mind that these trephines have an exterior size of approximately 1.5 mm greater than the size of the dental implant to be removed [24]. Hence, explanation might be related to considerable bone elimination consisting of buccal or linguistic bone cortices, and damage to adjacent natural teeth where the inter-radicular area is restricted. An alternate method is to enable modern bone loss from peri-implantitis to happen, causing enough bone loss to allow for the elimination of the implant with extraction forceps [21]. Implants might be gotten rid of by forceps when there is less than 3 to 4 mm of residual bone support.

CONCLUSION:

Peri-implant illness is identified right into two categories. 1) **In peri-implant mucositis**, gum inflammation is discovered just around the soft tissues of the dental implant, without indications of bone loss. Usually, peri-implant mucositis is a precursor to peri-implantitis. Evidence recommends that peri-implant mucositis may be effectively dealt with and is reversible if caught early. 2) In peri-implantitis, gum inflammation is discovered around the soft tissue and there is deterioration in the bone supporting the dental

implant. Peri-implantitis normally needs surgical therapy.

Indicators of peri-implant diseases resemble signs of gum illness: red or tender gums around the implants, or bleeding when cleaning. And much like your natural teeth, implants need regular tooth cleaning and flossing and normal examinations from a dental expert. Various other risks aspects for developing peri-implant disease include previous gum ailment medical diagnosis, poor plaque control, smoking, and diabetes mellitus. It is essential to routinely check dental implants as part of an extensive periodontal analysis. If undiagnosed, preimplant disorder might lead to total loss of osseointegration and implant loss.

REFERENCE:

1. Zitzmann, N. U. & Berglundh, T. (2008) Definition and prevalence of peri-implant disease. Journal of Clinical Periodontology 35 (Suppl. 8), 286–291.
2. Glossary of periodontal terms 2001. 4th ed. Chicago: American Academy of Periodontology; 2001. p. 20, 38. Available at: http:// www.joponline.org/page/glossary. Accessed October 28, 2013.
3. Listgarten MA, Lang NP, Schroeder HE, Schroeder A. Periodontal tissues and their counterparts around endosseous implants. Clin Oral Implants Res 1991;2:1-19.
4. Konttinen YT, Lappalainen R, Laine P, Kitti U, Santavirta S, Teronen O. Immunohistochemical evaluation of inflammatory mediators in failing implants. Int J Periodontics Restorative Dent. 2006;26:135–141.
5. Berglundh T, Gislason O, Lekholm U, Sennerby L, Lindhe J. Histopathological observations of human periimplantitis lesions. J Clin Periodontol. 2004;31:341–347.
6. Carcuac O, Berglundh T. Composition of human peri-implantitis and periodontitis lesions. J Dent Res. 2014;93:1083–1088.
7. 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.
8. Persson GR, Renvert S. Cluster of bacteria associated with periimplantitis. J Periodontal Res. 2016;51(6):689–698.
9. Leonhardt A, Renvert S, Dahlen G. Microbial findings at failing implants. Clin Oral Implants Res. 1999;10:339–345.
10. Rakic M, Grusovin MG, Canullo L. The microbiologic profile associated with peri-implantitis in humans: a systematic review. Int J Oral Maxillofac Implants 2016;31:359–368.
11. 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.
12. Schwarz F, Becker K, Sahm N, Horstkemper T, Rousi K, Becker J. The prevalence of peri-implant diseases for two-piece implants with an internal tube-in-tube connection: a cross-sectional analysis of 512 implants. Clin Oral Implants Res. 2017;28:24–28.
13. Fuchigami K, Munakata M, Kitazume T, Tachikawa N, Kasugai S, Kuroda S. A diversity of peri-implant mucosal thickness by site. Clin Oral Implants Res. 2017;28:171–176.
14. Schwarz F, Claus C, Becker K. Correlation between horizontal mucosal thickness and probing depths at healthy and diseased implant sites. Clin Oral Implants Res. 2017;28:1158–1163.
15. Penarrocha-Diago M, Maestre-Ferrin L, Penarrocha-Oltra D, Canullo L, Piattelli A, Penarrocha-Diago M. Inflammatory implant periapical lesion prior to osseointegration: a case series study. Int J Oral Maxillofac Implants. 2013;28:158–162.
16. Kutlu HB, Genc T, Tozum TF. Treatment of refractory apical periimplantitis: a case report. J Oral Implantol. 2016;42:104–109.
17. Tozum TF, Sencimen M, Ortakoglu K, Ozdemir A, Aydin OC, Keles M. Diagnosis and treatment of a large periapical implant lesion associated with adjacent natural tooth: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;101:132– 138.
18. Kaplan I, Hirshberg A, Shlomi B, et al. The importance of histopathological diagnosis in the management of lesions presenting as peri-implantitis. Clin Implant Dent Relat Res. 2015;17 Suppl 1:e126–133.
19. Bischof M, Nedir R, Lombardi T. Peripheral giant cell granuloma associated with a dental implant. Int J Oral Maxillofac Implants. 2004;19:295–299.
20. Ozden FO, Ozden B, Kurt M, Gunduz K, Gunhan O. Peripheral giant cell granuloma associated with dental implants: a rare case report. Int J Oral Maxillofac Implants. 2009;24:1153– 1156.
21. Schou S, Berglundh T, Lang NP. Surgical treatment of peri-implantitis. Int J Oral Maxillofac Implants. 2004;19(Suppl):140–9.
22. Karring ES, Stavropoulos A, Ellegaard B, Karring T. Treatment of periimplantitis by the Vectors

system.A pilot study. Clin Oral Implants Res. 2005;16:288–93.
23. Schou S, Holmstrup P, Jorgensen T, Skovgaard LT, Stoltze K, Hjorting-Hansen E, et al. Implant surface preparation in the surgical treatment of experimental peri-implantitis with autogenous bone graft and ePTFE membrane in cynomolgus monkeys. Clin Oral Implants Res. 2003;14:412–22.
24. Schwarz F, Sculean A, Bieling K, Ferrari D, Rothamel D, Becker J. Two year clinical results following treatment of peri-implantitis lesions using a nanocristalline hydroxyapatite or a natural bone mineral in combination with a collagen membrane. J Clin Periodontol. 2008;35:80–7.
25. Schwarz F, Bieling K, Bonsmann M, Latz T, Becker J. Nonsurgical treatment of moderate and advanced peri-implantitis lesions: A controlled clinical study. Clin Oral Invest. 2006;10:279–88.
26. Rutger GP, Ann-Marie RJ, Christel L, Stefan R. Microbiologic Results After Non-Surgical Erbium-Doped:Yttrium, Aluminum, and Garnet Laser or Air-AbrasiveTreatment of Peri-Implantitis: A Randomized Clinical Trial. J Periodontol. 2011;82:1267–78.
27. Renvert S, Lessem J, Dahlen G, Renvert H, Lindahl C. Mechanical and repeated antimicrobial therapy using a local drug delivery system in the treatment of peri implantitis: A randomized clinical trial. J Periodontol. 2008;79:836–44.
28. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis and treatment. Clin Oral Implants Res. 2000;11(Suppl 1):146–55.
29. Romeo E, Lops D, Chiapasco M, Ghisolfi M, Vogel G. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part II: Radiographic outcome. Clin Oral Implants Res. 2007;18:179–87.
30. Schwarz F, Bieling K, Latz T, Nuesry E, Becker J. Healing of intrabony peri-implantitis defects following application of a nanocrystalline hydroxyapatite (Ostimt) or a bovine-derived xenograft (Bio-Osst) in combination with a collagen membrane (Bio-Gidet). A case series. J Clin Periodontol. 2006;33:491–9.
31. Schwarz F, Sculean A, Bieling K, Ferrari D, Rothamel D, Becker J. Two year clinical results following treatment of peri-implantitis lesions using a nanocristalline hydroxyapatite or a natural bone mineral in combination with a collagen membrane. J Clin Periodontol. 2008;35:80–7.