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Treatment modalities for peri-implantitis: A review of literature

Alyaa I Naser, Rayan S Hamed

ABSTRACT

Introduction: Implant Dentistry was created to show reliable treatment approaches for restoring the oral cavity's esthetic and function. However, while dental implants have a high long-term success rate, dental implants are exposed to mechanical or biological complications.

Objective: The therapeutic approaches will be addressed in this review, which is the main objective of this study.

Materials and Methods:

Data sources: The PubMed/MEDLINE, SCOPUS, and Web of Science databases were used to conduct the literature search from June to September 2021.

Study selection: Non-surgical and surgical peri-implantitis treatment modalities are the main themes of this study.

Result: Implant dentistry was created to show reliable treatment approaches for restoring the oral cavity's esthetic and function. However, while dental implants have a high long-term success rate, dental implants are exposed to mechanical or biological complications.

Conclusion: Peri-implantitis appears to be a multifactorial disease including the patient's host/microbe response, implant characteristics, soft tissue and the hard conditions surrounding the implant, and the dentist's surgical and prosthetic part experience. Different treatment modalities are present today, all are used to treat peri-implantitis, such as surgery, laser therapy, and innovative treatments such as cold atmospheric pressure air plasma jet (CAPAJ), enamel matrix derivative, and PRF gel.

Keywords: Bone substitutes, Chlorhexidine, Dental implants, Peri-implantitis

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INTRODUCTION

The revolution of implantology developed to display constant treatment modalities to restore the esthetic and function of the oral cavity. While the dental implant has a high success rate in the long period, the possibility of failure does exist. Documented classification based on pocket probing depth (PPD), clinical bleeding on probing (BOP), and radiographical evaluation of marginal bone-loss reduction. Two subtypes of peri-implant disease are prefaced by pathogenic bacteria [1] peri-implantitis and peri-implant mucositis [2, 3]. Peri-implant mucositis is a reversible inflammatory condition that affects the soft tissue surrounding dental implants, causing swelling, redness, and bleeding on probing [4]. Peri-implantitis is a prevalent condition that involves soft and hard tissue which results in loss of bony support and establishment of a peri-implant pocket around the osseointegrated implant [5]. There are three categories which classify peri-implant based on pocket depth and bony loss, early peri-implantitis occurs when pocket depth is more than 4 mm and bone loss is lower than 25%, moderate peri-implantitis

establishes when pocket depth is more than 6 mm and bony loss is in-between 25% and 50%, and in addition to severe peri-implantitis manifests when pocket depth is more than 8 mm with bone loss is higher than 50% [6]. Multiple risk factors play an essential role in the development of peri-implantitis include past periodontal condition, improper plaque removal, smoking, residual cement, and occlusal overloading, genetic factor, improper surgical placement, poor oral hygiene, and diabetic condition [7–10]. *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans* microbiotas are linked with biofilm formation on dental implants [11]. Bacterial infection-induced immune response to recruit neutrophils, macrophages, T cells, and B cells migrates into the implant-bone interface. Finally, loss of alveolar bone leads to instability and loss of the implant [12]. Different therapies present today, such as surgery, laser therapy, and innovative treatments such as cold atmospheric pressure air plasma jet (CAPAJ), enamel matrix derivative, and platelet rich fibrin (PRF) gel, are all used to treat peri-implantitis [13].

MATERIALS AND METHODS

Data sources: The PubMed/MEDLINE, SCOPUS, and Web of Science databases were used to conduct the literature search from June to September 2021.

Study selection: Non-surgical and surgical peri-implantitis treatment modalities are the main themes of this study.

RESULTS

Etiologies of peri-implantitis

Peri-implantitis appears to be multifactorial pathology, including the response of the patient's host/microbe, implant properties, soft tissue and the hard condition around the implant, the experience of the dentist in surgical and prosthetic parts. As in general periodontal diseases, dental plaque formation is considered the main predisposing factor for the development of peri-implantitis [14]. Canullo et al. categorized the etiological factors of peri-implantitis concerning keywords of many systematic reviews into (individual susceptibility to peri-implant disease, plaque as etiological factors of peri-implantitis, biomechanics as trigger factor of peri-implantitis, surgical and prosthetic procedures as etiological factors of peri-implantitis) [15]. Smoking patients with IL-1 gene polymorphism might have a higher risk for peri-implant bone loss since a synergic effect is expected. In these reports, a clear association between heavy smokers with a positive IL-1 genotype and implant complications (loss of implant, or peri-implantitis) was found [16, 17]. As a result of the meta-analysis of Clementini et al., cigarette smoking raised the yearly rate of bone resorption to 0.16 mm/year [18] and minimized the amount of osseointegration, and endangered the oral hygiene surrounding dental implants [19]. Medical diseases like diabetes mellitus, immunosuppression disease play a significant effect in the establishment of peri-implantitis around the dental implant. Iatrogenic causes as cementitis, lack of keratinized gingiva at the area of implantation, history of failures of implants [20, 21]. The remaining teeth with periodontitis should be considered a possible infection source (edentulous vs partially edentulous, which promotes periodontal pathogen colonization of peri-implant tissues) [22].

Pathogenesis of peri-implantitis

Peri-implantitis is a condition that is both progressive and irreversible attacks soft and hard tissue surrounding the dental implant, furthermore, it invades by inflammatory cells, plasma cells, and B-lymphocytes with the absence of a protective tissue layer over the bone [2]. From a histological point of view, peri-implantitis when compared with periodontitis has two-fold large and extra blood vessels with invasion connective tissue than periodontitis [23]. In response to bacterial infection, the immune system recruits the neutrophils, macrophages, T cells, and B cells to the lesion [24]. Corrêa et al. identified the role of fibroblasts in the pathogenesis of peri-implantitis by upregulating both vascularity and matrix degradation [25]. Compared to periodontal tissues, peri-implant tissues are more prone to inflammatory illness due to decreased vascularization and parallel orientation of collagen fibers. This hypothesis is supported by immunohistochemical examination through increased formation of inflammatory infiltrate, vascular endothelial growth factor (VEGF), nitric oxide 1/3, leukocytes, Ki-67, and lymphocytes [26]. Also higher level of matrix metalloproteinase (MMP-8) shows an increase of up to 97% in peri-implantitis [27]. Papi et al. demonstrated the extracellular matrix antibodies are more abundant in peri-implantitis [28]. Rokaya et al. stated that the rapid disease progression rate in peri-implantitis causes a faster and more severe bone loss than the periodontal disease [2]. In peri-implantitis, the difference in microorganism in implant site, host defense mechanism, in addition to the absence of periodontal ligaments may induce a nonlinear bone destruction form that occurs over time in peri-implantitis [29].

Preventive strategies

Whatever the type of treatment used, the successful treatment strategy is highly dependent on the preventive measures taken by the patients, in addition to the early diagnoses and treatment planning. Appropriate and organized treatment planning inhibits the development of peri-implantitis. This is achieved by thorough individual assessment and eliminating of risk factors, establishing

of healthy soft and hard tissue conditions with excellent implant design according to the available surgical area for implantation followed by atraumatic surgical procedure and periodic clinical examinations with periodontal probing status. One of the most important preventive strategies is the toothbrush for plaque removal, followed by stannous fluoride-sodium hexametaphosphate containing dentifrice and chlorhexidine containing mouthwash which has an important role in the elimination of gingivitis and plaque removal [30].

Peri-implantitis treatment

Generally, the treatment should be focused on infection and bacterial control as microbial biofilm plays a crucial role in disease progression. Lang et al. affirmed that the dental implant should only be removed completely when there is mobility and bone loss surpasses 60% [31]. Treatment modalities are including surgical and non-surgical treatment according to the rate of disease progression.

Non-surgical peri-implantitis treatment

Multiple non-surgical peri-implantitis therapies involve mechanical, chemical, antibiotic, antiseptic, laser, and photodynamic therapies.

Mechanical approaches

The main goal of mechanical treatment is eliminating microbial biofilm from the subgingival surface of the dental implant by leaving a smooth surface to avoid further plaque formation. This was reached by using plastic or metal curettes or ultrasonic scalers or piezoelectric scalers and air abrasive devices [30]. Toma et al. consider a combination between mechanical techniques and using of antibiotics or surgical methods in their study to achieve better consequences [32]. Moreover, another study supports surgical treatment with mechanical debridement, to increase the effectiveness of mechanical treatment [33]. Hence, the mechanical techniques alone are unsuccessful to eradicate bacterial contamination and the treatment should be coupled with other treatments as antiseptic with surgical procedure.

Chemical methods

It includes topical application of antibacterial agents. The most common chemical agents used to eliminate bacterial growth are citric acid, ethylenediaminetetraacetic acid (EDTA), hydrogen peroxide (HP), and saline. Citric acid is a chemotherapeutic agent with a high capacity of biofilm removing and decreasing contamination from the implant titanium surface, has a low toxic effect on osteoblasts cells in low concentration (4–10%) in contrast to high concentration 40% with (pH = 1) may be toxic to peri-implant tissue [34]. The most common agent chelating to eradicate the smear layer and decontaminate implant surface is EDTA. Its effect apparently on periodontal regeneration; however, it should be used with caution due to some cytotoxic effects [35]. The biofilm that formed on the implant surface can be diminished by 10% of hydrogen peroxide and also may decrease bacterial colonization up to 99.9%. Furthermore, swabbing the implant surface for 1 minute with HP can induce re-osseointegration in peri-implantitis lesions, on the other hand, due to its high reactivity, it can harm oral mucosa, so it should be used with caution [34]. Surgical decontamination of implant surfaces followed by the toilet with sterile saline with postsurgical antibiotic prescription prevents the advancement of peri-implantitis [36].

Antiseptic

Adequate implant adhesion with subsequent decreasing inflammatory reaction is achieved by topical application of chlorhexidine gluconate (CHX) as irrigant solution in concentration 0.12% [37]. Chlorhexidine inhibits bacterial colonization, hence, there is a reduction in clinical parameters [bleeding on probing (BOP), probing pocket depth (PPD)] with radiographical confirmation. Moreover, laboratory investigation showed a decrease in inflammatory signs at a high level [IL-1 beta, prostaglandin E2 (PGE-2), and VEGF] [36]. These findings came in the same line with the study of Rokaya et al., who found that the local application of CHX with controlled-release induces periodontal re-osseointegration. Chlorhexidine gluconate has certain drawbacks if used in concentration 2% which permanently jeopardizes cell migration and the survival of fibroblast, myoblast, and osteoblast [2].

Antibiotics drugs

Bacteriostatic (doxycycline, minocycline) or bactericidal (gentamicin, cefazolin) drugs can be used along with mechanical debridement for control of the infection [38, 39]. Different topical and systemic antibiotic applications have been studied in the treatment of peri-implantitis. Doxycycline and minocycline, when applied topically after local debridement of the lesion with continuous irrigation, appeared more effective in treating a moderate deep lesion. Host defense mechanisms and immunity can be enhanced by administering systemic antibiotics to eliminate infection through combating residual subgingival bacteria that stay after mechanical curettage [40]. Different sustained release devices like a chip, gel, polymeric fiber, and microcapsules have been innovative to act as a reservoir of a constant level of specific antibiotics at specific infection areas [30].

Photodynamic therapy

Photodynamic therapy is considered a supplementary therapy for peri-implantitis treatment. As it is a relatively new approach, it decreases bacterial colonization and

promotes crestal bone remodeling after mechanical debridement. Regarding decontamination of implant surfaces and surrounding tissue, 200 μg/mL methylene blue under red laser has been used against aerobic and anaerobic bacteria such as *A. actinomycetemcomitans*, *Enterococcus faecalis*, *P. gingivalis*, *Streptococcus mutans*, *P. intermedia* [41]. Bacterial infection also can be subsided by utilizing various antimicrobial agents like bioactive glass (BAG), which possess high efficacy against infection and because of these characteristics, BAG may be the optimal bone substitute for curing peri-implant infections [42].

Laser therapy

Concerning of superior properties of laser in pain relief, patient comfort, and better result in terms of bleeding with bactericidal effect, various laser types utilized for treating the peri-implantitis are CO_2, Er, Cr:YSGG-(erbium, chromium-doped: yttrium scandium-gallium-garnet) lasers and Diode-Er:YAG-(erbium-doped: yttrium-aluminum-garnet) [43]. When the laser is applied to the infected area around the dental implant, it improves the cellular photoreceptors activation (cytochrome C oxidase) to absorb the laser radiation and convey it to the cell's mitochondria. The adenosine triphosphate of cells will increase, which is the main product of the Krebs cycle and cytochrome C oxidase, also laser causes an increase in cellular activity. Increased adenosine triphosphate activates macrophages, endothelial cells, fibroblasts, growth factors, nerve cells, mast cells, and bradykinin. As a result of this sequence, there is a reduction of plaque index [44] with an increase of collagen fiber synthesis, which is resulting in stimulating tissue regeneration around the dental implant [45].

Surgical peri-implantitis treatment

Regarding the presence of deep pockets ≥5 mm, with inferior changes in clinical parameters (BOP, PPD), the principal purpose of surgical approaches is to get direct access to peri-implant site subgingivally to get rid of granulation tissue and decontamination of the implant surface to enhance new attachment and a re-osseointegration is established. During the last five years, many literature reviews dealt with surgical treatment in form of resective therapy, regenerative therapy, implantoplasty, and air-abrasive powder [2, 30, 38]. Recently, a narrative review by Roccuzzo et al. categorized the surgical treatment in a different pattern [46]. Roccuzzo et al. reported in their systemic review that high survival rates with moderate composite success rates when estimating the result of open flap debridement without resection of the bony pocket of peri-implantitis with PPD ≤ 5 mm. They found that there is no bleeding or suppuration on probing and that no additional bone loss has occurred [47]. This systemic review is supported by the study of Heitz-Mayfield et al.; in which they evaluate the success rate of surgical treatment with mechanical debridement in conjunction with the delivery of systemic antibiotic (amoxicillin and metronidazole) without bone resection, they found there is a complete resolution of sign and symptoms of disease [48]. These results highlight the challenge faced by clinicians in keeping up with mid- to long-term, positive short-term results. Interestingly, recurrent peri-implantitis with an increased level of marginal bone loss in presence of PPD ≥ 6 mm and subsequent loss of the implant, this scenario could be treated with open flap debridement with bone recontouring as approved by Carcuac et al. in their five years' study. Moreover, they reported the effect of modification of implants surfaces on the progression of the disease [49]. Concerning implantoplasty, the removal of supracrestal implant threads in presence of a bony defect is indicated to have a smooth implant surface and achieve a good surgical approach that prevents chronic peri-implantitis compared to bone resection alone or using glycine air abrasive [50, 51]. The drawback of implantoplasty and its inferior consequence such as fracture implant or precipitate titanium particles in the peri-implant soft tissue limited its use and need more clinical evidence to approve its usage [52]. Different reconstructive procedures utilizing barrier membranes, autogenous bone, and/or numerous bone substitutes are effective in peri-implantitis to affect re-osseointegration around dental implants [53, 54]. The result of a combination between resective surgery and reconstructive surgery using bone graft shows clinical improvements in periodontal pocket depth and clinical attachment level [55]. On contrary, the study of Roccuzzo et al. found that there is an increase in mucosal recession after one year of treatment about 2.5 mm that restricted their efficacy and limited usage in the posterior implant as an esthetic priority not significant [56]. The importance of keratinized mucosa after treatment of peri-implantitis is unclear [57]. Recently, four protocols have been reported to treat supracrestal or dehiscence bone defect with deficient of keratinized mucosa: (A) partial-thickness flap apically positioned, (B) bone recontouring to have a flat design, (C) implantoplasty to manage exposed surface implant, and finally (D) Stabilized free epithelial graft on vascular recipient bed [58]. Interestingly, the result of this protocol in the case series study demonstrated complete resolution of the diseased implant.

CONCLUSION

Implants have become a well-known therapy in dentistry for replacing lost teeth. This treatment option appears to be a "safe" treatment option when followed appropriately and anatomical and intra-individual limiting factors are taken into account. However, there is growing evidence that peri-implant inflammations

exist, and they are the most common problems affecting both the soft and hard tissues around a dental implant, potentially leading to its loss. As microbial biofilm plays such an important role in disease progression, treatment should generally focus on infection and bacterial management.

Ethical approval

The authors disclosed that this work received institutional ethical approval (protocol number **3558** on 7/6/2021).

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Author Contributions

Alyaa I Naser – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.

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