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EFFICACY OF IMPLANTOPLASTY ON TREATING PERI-IMPLANTITIS

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Abstract:
This study is to examine the definition and characteristics of peri-implant disease, with a specific focus on per-implantitis. The objective is to enhance comprehension of the subject matter and explore several highlighted management strategies. We conducted a comprehensive literature review utilizing electronic databases such as MEDLINE and EMBASE. We specifically searched for research on the use of Implantoplasty for treating Peri-Implantitis, with a focus on publications in the English language. Peri-implant diseases exhibit similar characteristics to gum disease, such as inflamed or sensitive gums surrounding the implants, as well as bleeding during oral hygiene practices. Similar to natural teeth, implants require regular dental hygiene practices such as cleaning and flossing, as well as routine check-ups by a dental professional. Additional risk factors for developing peri-implant disease include a history of gum disease, inadequate plaque control, smoking, and diabetes mellitus. Regularly examining dental implants is crucial as a component of a comprehensive periodontal assessment. Undetected preimplantation abnormality can potentially result in complete failure of osseointegration and subsequent loss of the implant.

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INTRODUCTION:

Peri-implant sickness occurs when there is an imbalance between the number of microbes present and the body's ability to defend against them, after a successful integration of a dental implant into the bone. Peri-implant diseases can affect either the preimplant mucosa alone, known as peri-implant mucositis, or involve both the preimplant mucosa and the supporting bone, known as peri-implantitis [1]. Bleeding on probing (BOP) is consistently present in cases of peri-implant dysfunction [1]. Additional clinical signs of disease may encompass the presence of pus, increased probing depths compared to the initial measurements, recession of the mucosal tissue, the presence of a draining sinus (fistula), and swelling or excessive growth of the mucosal tissue around the implant. Failure to diagnose preimplant illness may result in total osseointegration failure and subsequent implant loss.

Food impaction, which refers to the trapping of food particles around natural or artificial teeth, is a widely acknowledged issue in dental care. Food impaction, as defined by the Glossary of Periodontal Terms, refers to the act of forcefully wedging food into the interproximal area through vertical pressure from chewing (vertical impaction) or with the application of tongue or cheek pressure (horizontal impaction) [2]. Implant restorations may also result in the entrapment of food within the preimplant sulcus. Commonly implicated foods in cases of food impaction include popcorn, seeds, legumes, and almonds, based on empirical evidence. The husk present in many seeds, including sunflower seeds, is mainly made of cellulose, a polymer that is indigestible by human enzymes. The disparity in the alignment of supracrustal connective tissue between natural teeth and dental implants is widely comprehended [3]. The fibers surrounding the implants align in a parallel manner with the abutment surface and just adhere to the joint surface rather than being connected [3]. As a result, the preimplant sulcus may be prone to food and foreign body blockages.

The objective of this study is to examine the definition and features of peri-implant disease, with a particular focus on per-implantitis. This will enhance our comprehension of the subject matter and the various emphasized approaches to its care.

METHODOLOGY:

We conducted a comprehensive literature analysis utilizing electronic databases like as MEDLINE and EMBASE. We specifically focused on research that provided data on techniques for managing dental per-implantitis, and only included papers published in the English language. Subsequently, we examined the reference lists of the papers that were included in order to identify any further pertinent articles that could provide supplementary evidence.

DISCUSSION:

The histopathological features of naturally occurring peri-implantitis lesions have been extensively examined in human biopsy specimens [4-6]. In comparison to peri-implant mucositis, the lesions at peri-implantitis sites (as indicated by bleeding on probing, suppuration, and radiographic bone loss) had a higher number of neutrophil granulocytes and a greater proportion of B cells (CD19+). Like periodontitis, the lesions in peri-implantitis sites were also characterized by a high presence of plasma cells and lymphocytes. However, they were distinguished by a greater abundance of polymorphonuclear leukocytes and macrophages [5]. Recent research has also demonstrated that the size of peri-implantitis lesions (defined as implant sites between teeth with bleeding on probing and probing depth of 7 mm or more) was more than twice as large as the lesions observed at periodontitis sites (3.5 mm2 vs. 1.5 mm2) [6]. Moreover, the presence of peri-implantitis lesions was determined based on larger proportions of area, quantities, and thickness of plasma cells, macrophages, and neutrophils, as well as a higher concentration of vascular frameworks located outside and adjacent to the infiltrated cells [5]. Another research investigation, using immunohistochemistry analysis of collected soft tissue samples, found that IL-1 was a prominent cytokine that activates osteoclasts at peri-implantitis areas [4]. It is important to note that the studies mentioned above of human peri-implant tissue samples did not include the bone component of the sites, due to ethical considerations.

By employing conventional DNA probe and cultural analyses, researchers have successfully isolated typical periodontopathogenic bacteria from both healthy and infected implant sites. The distribution of these identified species did not significantly vary based on the medical implant status, whether it was healthy, peri-implant mucositis, or periimplantitis [7]. However, in comparison to healthy implant sites, peri-implantitis was associated with increased levels of 19 different bacterial species, including Porphyromonas gingivalis and Tannerella forsythia [8]. Furthermore, observational studies have indicated that peri-implantitis is frequently associated with opportunistic pathogens such as Pseudomonas aeruginosa and Staphylococcus aureus (S. aureus), fungal organisms like Candida albicans, Candida boidinii, Penicillum

spp., Rhadotorula laryngis, Paelicomyces spp., and viruses such as human cytomegalovirus and Epstein-Barr virus. This suggests that the infection is complex and varied. It is important to note that the submucosal microbiota of peri-implantitis lesions have not been thoroughly investigated utilizing approaches that do not rely on culturing. Therefore, the microbiological profile associated with peri-implantitis should be considered as inadequate.

Peri-implantitis is commonly defined based on medical signs of inflammation, including redness, swelling, expansion of the mucosa, bleeding on probing (BOP+) with or without pus, as well as increases in pocket depth (PD) and loss of bone shown on radiographs [3-6]. Peri-implantitis identified implant sites typically have elevated probing depth (PD). A study investigated 588 individuals who had a total of 2,277 implants for a period of 9 years. The study found that 59% of the implants that showed signs of moderate/severe peri-implantitis (as indicated by bleeding on probing and bone loss greater than 2 mm) had a probing depth of 6 mm or more. Among the implants classified as healthy (defined as absence of bleeding on probing) or diagnosed with mucositis (defined as bleeding on probing but no bone loss greater than 0.5 mm), 3% and 16% respectively exhibited a probing depth of 6 mm or more. It was observed that the prevalence of implants with a pocket depth (PD) of 6 mm or more increased as the severity of peri-implantitis increased.

Schwarz et al. conducted a cross-sectional examination on 238 patients (with a total of 512 implants) who had a median function time of 23 months (ranging from 1 to 80 months) [12]. At sites with peri-implant mucositis (as determined by bleeding on probing (BOP+) on at least one side of the implant), the frequency of BOP scores ranged mostly between 33% and 50%, with a maximum of 67% at sites with periimplantitis (defined as BOP+ and/or suppuration and changes in radiographic bone level compared to baseline). Implant sites affected by disease had higher occurrences of 4 to 6 mm probing depth (PD) compared to sites with a healthy and well-maintained preimplant mucosa. The distribution of PD measurements was similar for sites with mucositis and peri-implantitis. Peri-implantitis was detected in only one implant, which exhibited probing depth (PD) values of 7 mm or greater [12]. It is important to recognize that identifying a physiological peri-implant disease at implant sites is challenging in this particular scenario. An evaluation conducted recently revealed a significant range of variation in the vertical mucosal density assessed at healthy implant sites, with measurements ranging from 1.6 to 7.0 mm (i.e. the degree of mucosal margin to the crestal bone) [13]. A cross-sectional examination was conducted to assess and compare the horizontal mucosal thickness (hMT) at both healthy and sick implant locations. The median horizontal marginal thickness (hMT) was significantly increased at sick implant sites compared to healthy sites (1.1 mm). However, the hMT was similar at mucositis and peri-implantitis sites (1.7 mm and 1.6 mm, respectively). The values in all groups examined did not significantly vary based on the location of the implant (either in the upper or lower jaws) or its position (either in the front or back of the mouth) [14].

In addition to peri-implant infections in areas with increased probing depths, several case series have also documented the presence of periapical peri-implantitis lesions. The implants that were impacted were typically detected through a periapical radiographic radiolucency, with or without accompanying clinical signs of inflammation, such as redness, swelling, fistula formation, and/or abscess formation [15]. The presence of inflammation was observed using clinical and radiographic indicators within a range of 2 to 8 weeks and around 4 years after the implant was placed [15], [16], [17]. The majority of research studies have found a direct correlation between retrograde periimplantitis and the presence of periapical endodontic lesions in adjacent teeth [15], [16], [17].

Case reports have described various oral-mucosal lesions occurring at dental implants that can resemble peri-implant diseases. These lesions consist of primary malignant tumors, such as oral squamous cell carcinoma, as well as metastases, giant cell, and pyogenic granuloma [18-20]. Although these pathological diseases have many medical similarities to peri-implant sickness, they exhibit clear differences in terms of nonspecific inflammation at the histopathologic level [18-20].

To clean the implant, it is necessary to use devices that are less hard than titanium, such as polishing with a rubber cup and paste, flossing, using interdental brushes, or employing plastic scaling instruments. Studies have shown that metal and ultrasonic scalers roughen the implant surface area, while these do not [21]. The use of ultrasonic scalers with nonmetallic tips or resin/carbon fiber curettes can effectively minimize harm to the implant surface area. However, the presence of implant threads and roughness on the surface may hinder the ability to clean the area.

The research conducted by Karring et al. shown that performing sub-mucosal debridement using either an ultrasonic instrument or carbon fiber curettes alone is inadequate for effectively removing contaminants

from the surface areas of implants with peri-implant pockets measuring 5 mm or more and exposed implant threads [22]. It appears reasonable to suggest that relying just on mechanical or ultrasonic debridement may not be enough to effectively treat peri-implantitis.

Four methods of purifying implant surfaces were evaluated in a monkey model: (1) using an air-powder abrasive technique followed by citric acid application, (2) using only an air-powder abrasive technique, (3) using gauze soaked in saline followed by citric acid application, and (4) using gauze soaked simultaneously in 0.1% chlorhexidine and saline [23]. The medical criteria, radiography (specifically quantitative electronic subtraction radiography), histology, and stereology did not identify significant differences among any of the procedures employed. The results of a laboratory investigation combining the use of toluidine blue solution and gentle laser irradiation have shown that it is possible to eliminate germs from various titanium surfaces without altering the surface of the implant [23].

Photodynamic therapy is a non-invasive method that can be utilized to reduce bacteria in peri-implantitis [26]. Topical antiseptics such as 2% chlorhexidine or 3% hydrogen peroxide can be used. The purification of implants that have been affected by contamination can be effectively achieved by using gauze soaked in a combination of chlorhexidine and saline solution. This method is particularly easy and efficient when dealing with implants that have titanium plasma-sprayed or sandblasted/acid-etched surfaces [21].

The utilization of an erbium-doped: yttrium, aluminum, and garnet (Er: YAG) laser in the non-surgical treatment of peri-implantitis lesions resulted in reduced levels of F. Nucleatum was observed one month following treatment [26]. Based on Schwarz et al.'s findings, both the Er: YAG laser and the combination of mechanical debridement and chlorhexidine are equally effective in significantly improving peri-implant probing pocket depth and clinical attachment level after 6 months of treatment. However, the use of the Er: YAG laser results in a much greater reduction in bleeding on probing compared to the additional use of chlorhexidine [24]. However, a further research study conducted by Schwarz et al. found that the effectiveness of the Er: YAG laser was only observed for a period of 6 months, specifically for advanced peri-implantitis lesions [25]. It was suggested that using the Er: YAG laser alone may not be enough to effectively treat peri-implantitis. Additional restorative procedures, such as using the Er: YAG laser again or using osseous regenerative treatments afterwards, may be necessary.

Accurate microbiological data regarding the existence of potential pathogens is crucial for making an informed decision regarding systemic or local antibiotic treatment. The composition of the subgingival microbial component is crucial in determining the appropriate medication. Additionally, the oral distribution patterns of potential microorganisms play a significant role in establishing whether an antibiotic agent should be administered locally or systemically. In order to do this work, medical professionals must take into account the periodontal state of the remaining teeth.

The study conducted by Schwarz et al. showed that treating peri-implant infection through mechanical debridement using plastic curettes combined with antiseptic treatment (0.2% chlorhexidine) resulted in significant improvements in bleeding on probing, peri-implant probing pocket depth, and clinical attachment level after 6 months, as compared to the initial measurements [24]. A study conducted by Renvert et al. shown that the inclusion of disinfectant therapy alongside mechanical debridement does not offer additional benefits in treating shallow peri-implant sores with a mean probing pocket depth of less than 4 mm [27]. Thus, it seems that incorporating antibacterial treatment alongside mechanical debridement does not provide additional benefits in shallow peri-implant sores with an average pocket probing depth of less than 4 mm. However, it does appear to offer extra clinical improvements in deep peri-implant lesions with an average pocket probing depth of greater than 4 mm, and particularly in those with an average pocket probing depth of greater than 5 mm. Patients who have specific peri-implant problems without any additional infections may be suitable for therapy with local drug-delivery devices. Topical administration of antibiotics through the placement of tetracycline fibers for a duration of 10 days can deliver a consistently high concentration of the antimicrobial agent directly to the affected area for an extended period of time [28]. Utilizing minocycline microspheres in conjunction with mechanical therapy seems advantageous in treating peri-implant lesions, however it may require replication [28]. The study conducted by Renvert et al. demonstrated that the additional advantages resulting from the inclusion of the antibiotic minocycline to mechanical debridement are generally superior, albeit to a limited degree, compared to the benefits achieved through the combined use of a disinfectant (chlorhexidine) and mechanical debridement [27]. The improvements in peri-implant probing depths achieved with the

additional application of minocycline can be maintained for a period of 12 months. According to

If the condition is widespread, comprehensive microbiological data is collected, and antibiotics are administered systemically. Lang et al. suggest the following antibiotic regimens: either taking systemic ornidazole 500 mg twice a day for 10 days, or metronidazole 250 mg three times a day for 10 days, or a once daily combination of metronidazole 500 mg and amoxicillin 375 mg for 10 days [28]. If peri-implantitis is associated with chronic periodontal disease, then both problems must be addressed. In this scenario, the further use of systemic antibiotics may be considered. Currently, there are no ongoing clinical trials investigating the use of systemic antibiotics for the treatment of peri-implantitis.

When mechanical and antibacterial treatments are followed before starting antibiotic therapy, it appears that superficial peri-implant infection can be adequately managed using antibiotics [1]. Nevertheless, it remains uncertain if more extensive peri-implant lesions can be effectively treated without surgery using a combination of a localized antibiotic and mechanical debridement.

Surgical methodology

Surgical excision is often limited to implants located in areas that are not aesthetically important. A surgical flap facilitates the thorough removal of dead tissue and cleansing of the affected implant. The surgical procedure involved the use of autogenous bone grafts covered by membrane layers, autogenous bone grafts alone, membranes alone, and a control access flap treatment. The results showed that defects treated with membrane-covered autogenous bone had significantly greater amounts of bone regrowth and reosseointegration compared to the other three treatments [21]. However, membrane exposure commonly occurs following these treatments. Exposing porous e-PTFE membrane layers can potentially result in bacterial infiltration and subsequent infection [21].

Currently, there are no randomized controlled medical trials available for the use of access flap surgical procedure (open-flap debridement) as a standalone therapy for periimplantitis. A randomized comparative clinical trial conducted by Romeo et al. concluded that the combination of respective surgical procedures and implantoplasty can positively impact the survival rates of rough-surfaced implants affected by peri-implantitis, as well as improve peri-implant clinical parameters such as pocket-probing depth, suppuration, Renvert et al.'s study, the observed bone loss did not exceed three implant threads [27].

and sulcus bleeding [29]. The research conducted by Schwarz et al. shown that both nanocrystalline hydroxyapatite and directed bone regeneration resulted in significant clinical improvements after 6 months of non-submerged healing, as indicated by clinical specifications [30]. The 2-year results from Schwarz et al.'s clinical study once again showed that both treatment methods were effective in significantly reducing pocket-probing depth and increasing clinical attachment level. However, the use of a combination of natural bone mineral and collagen membrane was associated with greater improvements in these clinical measures and, consequently, led to a more predictable and improved healing outcome [31]. Regrettably, the limited sample size of the research study (22 patients) prevented a reliable comparative analysis of the effectiveness of both therapeutic interventions. Collecting additional data on various regenerative techniques to address peri-implantitis is necessary.

CONCLUSION:

Various treatments for peri-implantitis, such as non-surgical therapies and respective and regenerative surgical therapies, have been documented. Often, a synthesis of methods is employed. The findings from research on different treatment approaches for peri-implantitis are inconclusive and subject to significant debate, making it now unfeasible to construct a universally accepted treatment program. Implantoplasty is a clinical technique used to make the exposed threads of an implant smoother, creating a more desirable location where the implant meets the surrounding tissue. The mechanical alteration of the implant surface promotes a decrease in bacterial attachment and facilitates the adjustment of the surrounding soft tissue throughout the healing phase. The objective of this study was to evaluate the effectiveness of implantoplasty in enhancing peri-implant health, based on previous research.

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