Causes Of Postoperative Complications After Dental Implantation (Literature Review)

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ABSTRACT

Biological complications affecting implant osseointegration are of great interest in modern dentistry. Such complications mainly refer to inflammatory conditions associated with bacterial contamination. Two clinical varieties can be distinguished: mucositis and peri-implantitis. Although the presence of an inflammatory lesion is a common feature of both conditions, only the latter form is manifested by loss of supporting bone. Mucositis is thought to precede peri-implantitis. Over the past two decades, the use of dental implants has become a common treatment to replace missing teeth.

Keywords: postoperative complications, dental implantation, overimplantitis

Introduction. According to industry projections, over 200000 implants were placed in the UK alone in 2013 [5]. With the increasing use of this treatment option, the incidence of complications is increasing. Peri-implantitis is a collective term for inflammatory reactions of the tissues surrounding implants, encompassing two main forms: mucositis and peri-implantitis. Initially, up to 0.2 mm of bone mass loss around implants in the first year, then 0.1 mm per year thereafter is considered an acceptable limit [1, 2]. However, as the technique improves, bone mass loss should decrease. Exceeding the acceptable bone mass loss threatens the success of the implant and therefore requires intervention. A search of PubMed, the US National Library of Medicine, the Excerpta Medica (Embase) database from Elsevier, and Web of Knowledge of Thomson Reuters databases revealed several relevant articles (i.e., experimental animal and human studies / observational studies, randomized / controlled clinical trials). studies, systematic reviews / meta-analyses, consensus reports) [7]. Peri-implantitis is a pathological condition occurring in the tissues around dental implants, characterized by inflammation of the mucosa around the implant and progressive loss of supporting bone.

Clinically, soft tissue inflammation is detected by probing (bleeding on probing, BOP), and progressive bone loss is detected on radiographs. Studies of peri-implantitis require case definitions and thresholds to distinguish 1) health from disease and 2) mucositis from peri-implantitis. It should be noted that although case definitions of peri-implantitis vary considerably between studies the definition of disease remains. Reflecting the progression of gingivitis to periodontitis, it is assumed that mucositis precedes peri-implantitis. At present, the signs or conditions that characterize the

conversion of mucositis to peri-implantitis have not been identified. The reactions of the soft tissue around the implant to plaque formation have been extensively studied in both animal and human studies.

Thus, plaque formation invariably resulted in soft tissue inflammation around the implant associated with clinical signs. inflammation such as redness and swelling. Zitzmann et al (2002) [6] examined human biopsies after a period of plaque formation lasting 21 days. Histologic analysis revealed the formation of an inflammatory cell infiltrate (ICI) with a predominance of B- and T-cells in the soft tissue lateral to the barrier epithelium, occupying an area of approximately 0.14 mm.

Similar results were obtained in animal studies demonstrating different apical extent of the inflammatory lesion [5]. In most of the implant sites studied, the lesion was located lateral to the barrier epithelium and separated from the crestal bone by an area of healthy connective tissue. However, at some sites in one study [8,12], the subepithelial connective tissue was infiltrated with inflammatory cells (i.e., CD 68+ cells), resulting in a reduced zone of healthy connective tissue over the peri-implant bone. After 16 weeks of plaque formation, the distance between the apical extension of the CPI and the crestal bone ranged from 1.0 to 1.9 mm. At only one implant site did the CPI reach the crestal bone.7 The exact histopathologic mechanisms leading to apical expansion of the CPI and associated crestal bone loss remain to be determined.Clinically, conversion of mucositis to peri-implantitis was evaluated in one retrospective observational study involving 80 patients [9] initially with peri-implant mucositis.17 Over 5 years, the incidence of peri-implantitis was lower in subjects enrolled in a program of regular supportive care (18%) than in patients without regular supportive care (43%). In the control group, "bone loss (BMD) + at> 50% of all implant sites" (OR (odds ratio 37) and "probing depth (PD) ≥4 mm at> 5% of sites" (OR 20) were associated with peri-implantitis. In the "no treatment" group, the associated factors were GZ (OR 26) and the presence of periodontitis (OR 11). In the entire group of patients, conversion to peri-implantitis was correlated with SCT (LS 18) and GZ (LS 16), lack of regular maintenance therapy (LS 6), and the presence of periodontitis (LS 9). The histopathologic and clinical conditions leading to the transformation of mucositis into peri-implantitis are not fully understood. The so-called "ligature model" is often used to study experimental peri-implantitis in animals [6]. The protocol includes a phase of active tissue destruction around the osseointegrated implants, including plaque formation and ligature placement in the submucosal position. The ligature destroys the implant mucosa and promotes the formation of a submucosal bacterial biofilm. The resulting inflammatory lesion causes tissue destruction, including loss of bone mass. Also, disease progression can occur after ligature removal and with persistent plaque formation. Thus, this model mimics natural peri-implantitis. Compared to experimentally induced periodontitis, lesions associated with experimental peri-implantitis show larger inflammatory cell infiltrates and more rapid and pronounced loss of bone mass [3]. Several weeks of plaque formation after ligature removal was associated with spontaneous progression of peri-implantitis. with severe inflammation and tissue destruction. Disease progression was influenced by implant surface characteristics with more severe destruction in implants with modified than unmodified surfaces.

Prospective studies evaluating the occurrence and progression of natural peri-implantitis cannot be identified and for obvious ethical reasons are not feasible. However, retrospective observational studies using multilevel growth curve models have provided statistical estimates of the onset and pattern of bone mass loss associated with peri-implantitis [9]. Fransson et al. [3] examined 182 patients with 419 implants (machined / turned surfaces, no bone grafting, fixed restorations) who had progressive bone mass loss. For these implants, bone levels were assessed using intraoral radiographs obtained between 1 year. examination and a follow-up period of 5 to 23 years (mean 11.1 years). The mean bone mass loss was 1.7 mm, and the cumulative percentage of implants with bone mass loss ≥1 mm, ≥2 mm,

or ≥3 mm was 68%, 32%, and 10%, respectively. The multilevel growth curve model showed that the pattern of bone mass loss was nonlinear, accelerated, and exhibited increased variance over time, which was associated with subject heterogeneity. This was confirmed in a retrospective analysis by Derks et al [2, 9]. The results indicated that the onset of peri-implantitis may occur earlier, as most implants showed the first signs of bone mass loss (> 0.5 mm) already after the second (52%) and third year (66%) of service. At the subject level, these estimates were 70% and 81%, respectively.

When evaluating the aforementioned studies, it should be kept in mind that the onset of peri-implantitis was evaluated only on the basis of radiologic bone loss, without taking into account other clinical parameters. Nevertheless, these analyses suggest that peri-implantitis may begin. at the beginning of the follow-up period and that peri-implantitis progresses more rapidly than periodontitis. The concept of a potentially early onset of peri-implantitis is further supported by studies evaluating peri-implant status already after relatively short follow-up periods (≤2 years) [8]. A cross-sectional analysis of 238 patients with a total of 512 implants showed that peri-implantitis (case definition: SCT+ and radiographic bone level changes from baseline) was frequently reported in all implant age groups studied. At the implant level, its incidence was n = 18 after 1-12 months of follow-up, n = 34 after 12-48 months and n = 12 after>48 months, respectively. For the diagnosis of mucositis, the number of affected implants in the respective age groups was n = 25, n = 157 and n = 32 respectively.Becker et al. [4] recently studied the incidence of biological complications with zirconium dioxide implants over a 2-year period in 52 patients. The SCT values increased significantly from 21% at baseline (i.e., 10-12 weeks after implant placement) to 38% and 64% at 6 and 12 years. months, respectively. Based on this case description (SCT + and changes in radiologic bone level compared to baseline), 18 patients were diagnosed with initial peri-implantitis between 12 and 24 months [10]. The histopathologic features of natural peri-implantitis lesions have been extensively studied in human biopsy material [7].

Compared to mucositis, lesions at peri-implantitis sites (case definition: PKT+, suppuration, radiologic bone loss) contain more neutrophilic granulocytes and a greater "proportion of B-cells (CD19+)." Similar to periodontitis, lesions at peri-implantitis sites were also dominated by plasma cells and lymphocytes but characterized by a higher proportion of polymorphonuclear leukocytes and macrophages. It has also been recently shown [12] that the size of peri-implantitis foci (case definition : interdental implant sites with SCT + and GZ ≥7 mm) was more than twice as large as that observed in periodontitis sites (3.5 mm2 vs. 1.5 mm2). Moreover, peri-implantitis lesions were characterized by larger proportions of area, number and density of plasma cells, macrophages and neutrophils, and higher density of vascular structures outside and lateral to the cellular infiltrate. Another study [11] using immunohistochemical analysis of collected soft tissue biopsies showed that IL-1α was the dominant cytokine activating osteoclasts at peri-implantitis sites. It should be emphasized that the aforementioned analyses of human peri-implant tissue biopsies did not include the bony component of these sites for ethical reasons. By conducting microbiologic and immunologic studies and using conventional DNA probe and culture assays for this purpose, common periodontopathogenic bacteria were isolated in both healthy and affected implant sites and the distribution of detected species did not differ significantly according to the clinical status of the implant (i.e., healthy, mucositis, peri-implantitis) [6]. However, compared to healthy implant sites alone, peri-implantitis was associated with higher bacterial counts of 19 species, including Porphyromonas gingivalis and Tannerella forsythia [1]. Moreover, observational studies [8] showed that peri-implantitis was more frequently associated with opportunistic microorganisms. 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 (e.g., human cytomegalovirus) , Epstein-Barr virus), indicating a rather complex and heterogeneous infection. It should be emphasized that the submucosal microbiota of peri-implant lesions has not been extensively studied using culture-independent methods. Thus, the microbial picture of peri-implantitis should be considered incomplete.

In conclusion, the prognosis of implant lesions will depend on early detection and treatment of mucositis and peri-implantitis. Conclusions: Although the studies on the different treatments for peri-implantitis are not comparable, a general pattern of some clinical improvement emerges with the use of anti-infective treatments in terms of resolution of inflammation and bone healing. This observation, combined with our knowledge of the undisputed role of periodontal pathogens in the etiology of peri-implantitis, indicates that some form of anti-infectious therapy should be combined with any other strategy to address this problem.

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