Clinical, microbiological, physicochemical and biochemical analysis of failing dental implants with peri-implantitis

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Abstract

(Objective) To investigate the pathophysiology of peri-implantitis with failing implants.

(Materials and Methods) A total of 10 patients with 20 failing implants were recruited. Probing pocket depths (PPD), gingival index (GI) or modified bleeding index (mBI), plaque index (PlI) or modified plaque index (mPlI) were assessed. Plaque samples were collected and real-time quantitative polymerase chain reaction (qPCR) was carried out for measuring the number of total bacteria and of the target pathogens. X-ray fluorescence (XRF) analysis was performed to analyze the elements of removed implant surfaces. Real-time qPCR was performed using the granulation tissues obtained at the time of implant removals. The target mRNA expressions were analyzed by the comparative cycle threshold (Ct) method. In the *in vitro* part of the study, real-time qPCR and enzyme-linked immunosorbent assay (ELISA) were performed.

(Results) The mean PPD, the mean GI or mBI and the mean PlI or mPlI were significantly larger around the peri-implantitis sites compared to the healthy gingiva sites, the periodontitis sites, and the healthy peri-implant tissue sites. The mean total bacterial numbers obtained from the locations with ≥7 mm in depth of the failing implant was significantly larger than those from the healthy tooth sulcus, the intact implant sulcus, deepest periodontal pocket of the remaining tooth, ≤3 mm depth, and 4–6 mm depth of the failing implant. The amount of *Porphyromonas gingivalis* (*P. gingivalis*), *Treponema denticola* (*T. denticola*), *Tannerella forsythia* (*T. forsythia*), *Fusobacterium nucleatum* (*F. nucleatum*), and *Prevotella intermedia* (*P. intermedia*) were significantly larger at the failing implants with peri-implantitis than those without. Zinc was detected from 15 removed implant surfaces, most of which were found in the subjects with multiple implant failures. Significantly shorter function time was observed in the removed implants with the presence of zinc than without. The mRNA expressions of matrix metalloproteinase-9 (MMP-9) and tissue inhibitor of metalloproteinase-1 (TIMP-1) were significantly up-regulated in granulation tissues around the failing implants with the presence of zinc than without. *In vitro* study demonstrated that MMP-9 and TIMP-1 mRNA expressions in macrophage-like cells were significantly up-regulated by lipopolysaccharide from *P. gingivalis* (*P. gingivalis* LPS) and/or $ZnSO_4$ stimulation and that *P. gingivalis* LPS and $ZnSO_4$ co-stimulation significantly increased TIMP-1 production on macrophage-like cells.

(Conclusion and Discussion) *P. gingivalis*, *T. denticola*, *T. forsythia*, *F. nucleatum* and *P. intermedia* were more frequently detected at the failing implants with peri-implantitis and zinc was detected in the attached substances on the removed implant surfaces of the subjects with multiple implant failures. In *in vitro* study, co-existence of bacteria and zinc affected macrophages provoking the break-down of the balance between MMP-9/TIMP-1 and increased TIMP-1 production. TIMP-1 is known to promote osteoclast formation. Function time of the implants with the presence of periodontopathic bacteria and zinc was thus hypothesized to be shortened.