

Soft tissue reactions to dental implants in man

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Background

Healthy peri-implant soft tissues are a prerequisite for the long-term success of dental implants (Brånemark et al. 1985). Morphology and pathology of these tissues were almost exclusively analysed in animal studies (Lindhe & Berglundh 1998). Little is known about the histological response of human peri-implant soft tissues to plaqueaccumulation, about the morphology of the tissues, the distribution of inflammatory infiltrates and the biologic width. The aim of the present investigation was to evaluate the healthy and inflamed peri-implant soft and hard tissues surrounding dual acid-etched dental titanium implants using histological, histometrical, and histomorphometrical techniques.

Materials and Methods

Twelve fully edentulous subjects (7 females and 5 males, 37-67 years of age) were selected. The study protocol was approved by the Ethics Committee of the Medical School, University of Freiburg, Germany. All subjects gave informed consent. Two (3.25 mm x 4 mm or 6 mm) custom-made screw implants (Osseotite®, 3i, Palm Beach Gardens, FL, USA) were inserted in the region of the former first molar, one in each lower quadrant (24 implants). The implant shoulder was placed at the level of the alveolar bone. Abutment connection was performed after three months of healing. One month of soft tissue healing was allowed to establish clinically healthy conditions of the peri-implant mucosa. Then, plaque control was discontinued randomly at one of the custom-made implants (plaque accumulation = AC) and continued by the patient at the other implant (plaque control = PC). The implants and the surrounding tissues were harvested different timepoints (7, 21, or 90 days). The biopsies were processed for light microscopy.

The following histological analyses were performed: a) location and extent of inflammatory infiltrates, b) distance between gingival margin (GM) and first bone-implant-(fBIC) (= implanto-mucosal contact complex), c) distance between GM and the most apical point of the junctional epithelium (aJE), d) distance between aJE and fBIC (connective tissue attachment), d) distance between microgap (MG) and aJE and e) distance between MG and fBIC (= bone loss).

Statistics

For descriptive analysis, means and standard deviations were calculated. Further analysis was carried out using ANOVA. The level of significance was set at $\alpha < 5\%$.

Results

Two implants did not integrate and 2 implants could not be successfully processed. The (histo-) pathology of the 20 control-implants remaining testand basically differed not. Test- and controlimplants of all groups showed slightly to severely infiltrated soft tissues in the region of the GM and lateral to the MG (Fig. 1). A progression of the histological inflammation could not be observed over time. Morphological features were: (i) apical migration of the epithelium to the bone around some implants (Fig. 2), (ii) zonal arrangement of the infiltrated areas with fibrotic transformation of collagen fibres in the outer zone (Fig. 3), (iii) immature structure of the connective tissue in vertical bone craters (Fig. 4), (iv) diffuse infiltrates in the connective tissue.







Fig. 2 AC 90: Migration junctional epithelium to the first bone-implant-contact



Fig. 3 AC 90: Severe reduction of collagen fibres lateral to microgap; fibrotic transformation of collagen fibers in an outer zone

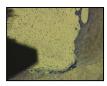


Fig. 4 PC 7: Bone crater with immature connective tissue: large amount of fibrocytes and small vessels. no collagen bundles; osteoclasts at the bone margin

The average height of the implanto-mucosal complex was 2.9 mm, 2.2 mm for the sulcus + epithelial attachment, and 0.8 mm for the connective tissue attachment. The aJE was located 1.1 mm apically to the microgap. The bone loss amounted to 1.8 mm (Tab.1).

Tab.1: Histometric analyses

Groups	AC 7 (n = 1)	AC 21 (n = 2)	AC 90 (n = 3)	PC 7 (n = 3)	PC 21 (n = 2)	PC 90 (n = 3)	All
G M -fBIC	2.46 ± 0.00	2.75 ± 0.15	353 ± 0.44	2.73 ± 0.84	2.99 ± 0.17	2.98 ± 0.86	2.9
GM-aJE	2.14 ± 0.00	1.79 ± 0.44	2.88 ± 0.74	2.03 ± 0.78	1.94 ± 0.25	2.22 ± 0.91	2.2
A JE-fBIC	0.32 ± 0.00	0.96 ± 0.60	0.65 ± 0.31	0.70 ± 0.26	1.06 ± 0.43	0.85 ± 0.42	0.8
V D I-e K I K	1.42 ± 0.00	2.52 ± 0.16	1.77 ± 0.45	1.77 ± 0.89	2.03 ± 0.64	1.15 ± 0.43	1.8
VDI-aSE	1.10 ± 0.00	1.56 ± 0.43	1.12 ± 0.22	1.07 ± 0.82	0.98 ± 1.06	0.33 ± 0.82	1.1

ANOVA: all variables p > 0.05

The lack of (patho-) histological differences and statistically significant histometrical changes was probably due to the small number of subjects and inadequate compliance regarding plaque control. A migration of the junctional epithelium to the bone, as found in this investigation, was not reported in numerous studies in dogs (Abrahamsson et al. 1996). However such a migration was observed in monkeys (Hashimoto et al. 1989, Krekeler 1997) and in a case report (Piatelli et al. 1996). Lack of cementum and inflammatory lesions (Sanavi et al. 1998) may be a cause for apical migration of the junctional epithelium. Zonal arrangement of the infiltrated areas with fibrotic transformation was also described in monkeys (Krekeler 1997) and around human teeth (Page & Schroeder 1976), but not in dogs (Ericsson et al. 1995). The observed inflammatory infiltrates were not as clearly circumscribed and localised as described around implants in dogs (Ericsson et al. 1995). The evaluated average dimensions of components ٥f the biologic width/implanto-mucosal complex were similar those found in animal studies (Abrahamsson et al. 1996).

Conclusions

From the results of the present investigation we can conclude that: 1) short time periods regarding plaque control or accumulation do not seem to lead to differences in the histologic appearance of the different tissues, 2) some aspects of soft tissue histology and pathology are different to those in dogs; 3) histometric soft tissue measurements (biologic with) coincide with animal experiments.

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