The Use of Tapered Implants in the Maxillae of Periodontally Susceptible Patients: 10-Year Outcomes

Zeev Ormianer, DMD[1]/Ady Palti, DDS[2]

Purpose: *This study is a retrospective assessment of the long-term efficacy of dental implant therapy in periodontally susceptible patients.* ***Materials and Methods:*** *A private-practice chart review was conducted to identify partially dentate subjects treated with implant-supported restorations that had been monitored annually for at least 9.5 years. Subjects were assigned to either a periodontal group or a control group according to their health histories. Data were entered into spreadsheets on a personal computer and analyzed statistically with dedicated software.* ***Results:*** *Thirty periodontal subjects were treated with 138 implants and 45 prostheses, and 16 control subjects were treated with 35 implants and 21 prostheses. The mean follow-up was 130 months. One implant failed before loading in the periodontal group. Cumulative 10-year survival rates were 99.3% (n = 137/138) for periodontal implants and 100% (n = 35/35) for control implants. Most surviving implants had no bone loss (n = 109/172, 63.4%). Most of the surviving implants with bone loss (n = 63/172, 36.6%) were concentrated in the periodontal cohort (90%, n = 57/63) and among women (60%, n = 15/25) regardless of cohort. Prosthesis failure was 25.2% (n = 16/66), with 12 porcelain fractures, 2 cement failures, and 2 framework fractures. In all cases, failed prostheses were immediately replaced and patients continued to function.* ***Conclusions:*** *Periodontal susceptibility resulted in increased bone loss but did not affect implant survival. The cause of greater bone loss in women could not be determined from the data but may have been related to the postmenopausal status of the subject population (mean age = 54 years).*
Int J Oral Maxillofac Implants 2012;27:442–448

Key words: *bone loss, dental implants, outcome, periodontitis*

Although an estimated 100 to 200 varieties of bacteria typically inhabit the human mouth at any given time,[1,2] most remain clinically benign below a certain count.[2–4] However, with unchecked reproduction, bacteria can organize into complex oral biofilms capable of interacting metabolically as a community[5] and triggering a sequence of gingival and periodontal diseases in the gingival sulcus regions of teeth. Infection is usually manifested as localized gingival inflammation, but continued release of inflammatory cytokines, chemokines, and other intracellular mediators from the biofilms can progressively lead to chronic gingivitis, periodontal attachment loss, bone resorption, and eventual tooth loss.[3,4] Periodontitis, by definition, is inflammation of the periodontal supporting tissues of the teeth, from the gingiva into the adjacent bone and ligament, usually with a progressive destruction change leading to loss of bone and the periodontal ligament.[6]

When exposed to the oral cavity through design or by attachment of a transmucosal abutment, dental implants are colonized by microbiota located on the surfaces and in the periodontal pockets of the surrounding dentition.[5] Peri-implant disease, by definition, affects the tissues associated with an oral implant and/or abutment, where bacteria play a major role in the etiology of peri-implant diseases, which can be restricted to soft tissue (mucositis) or progress to the supporting bone and induce its destruction (peri-implantitis).[6] The presence of both teeth and implants in periodontally susceptible patients creates a local environment in which subgingival pathogens around teeth can infect the peri-implant tissues.[7] The process of peri-implantitis typically begins as gingivitis after 10 to 14 days of plaque retention and triggers the same pathogen-induced inflammatory and infectious disease progression as periodontitis.[8–11] Destruction of the supporting host tissues has been reported to occur much faster in peri-implantitis than in periodontitis, in part because implants lack a natural soft tissue barrier, such as a connective tissue attachment or a predictable mucosal seal.[12]

[1]Lecturer, Department of Oral Rehabilitation, Maurice and Gabriela Goldschleger School of Dental Medicine, Tel Aviv University, Tel Aviv, Israel.
[2]Clinical Professor, New York University Dental School, Department of Periodontology and Implantology, New York, New York.

Correspondence to: Dr Zeev Ormianer, Department of Oral Rehabilitation, School of Dental Medicine, Tel Aviv University, Ramat Aviv, Tel Aviv, 69978 Israel. Fax: +972-03-6124226. Email: drzeev@ormianer.com

Nowzari and coworkers[13] evaluated the presence of periodontopathic bacteria and inflammatory cytokine levels around healthy teeth and dental implants. They found that counts of pathogenic bacteria were higher around teeth as compared to dental implants; conversely, higher concentrations of inflammatory cytokines were present around healthy dental implants in comparison to teeth. Cytokine levels increased around both teeth and implants in the presence of bacterial infection.[13] The finding that dental implants in a presumed state of health may exhibit a chronic, low-level inflammatory response without overt clinical manifestations may partially account for the aggressive tissue destruction often observed with peri-implantitis as compared to the level of tissue destruction that occurs with periodontitis.[8,12]

Patients with a documented history of periodontitis around one or more teeth may be susceptible to disease recurrence, but the degree of periodontal susceptibility can vary according to many different factors, such as personal lifestyle choices (eg, oral hygiene habits, alcohol consumption, drug use, smoking)[14–17]; concomitant medical conditions (eg, pregnancy, the use of certain medications)[18] or diseases (eg, diabetes, immune deficiencies)[19–22]; and genetic makeup (eg, patient sex, immune system health).[3,23–31] Diagnosis of periodontitis and peri-implantitis relies primarily on clinical and radiographic findings, since microbial diagnostic tests are limited in scope and genetic testing is still being developed for commercial use.[32]

The efficacy of placing dental implants into periodontitis-susceptible patients has been investigated in several long-term (≥ 5 years) studies, but outcomes have been inconsistent.[33–36] In a 10-year, prospective cohort study of 223 implants placed in 101 periodontally compromised and periodontally healthy patients, Roccuzzo et al[33] reported that patients with a history of periodontitis presented lower implant survival rates and exhibited a statistically significant increase in the number of sites with peri-implant bone loss. Similarly, Karoussis et al[11] conducted a 10-year prospective cohort study that evaluated the incidence of peri-implantitis in 53 patients. They found that patients with a history of periodontitis exhibited a 28.6% higher incidence of peri-implantitis than subjects without a similar history. In a separate study, Karoussis et al[34] conducted a systematic literature review of dental implant prognosis in periodontally compromised partially edentulous patients, but the authors were unable to perform a meta-analysis because the eight long-term and seven short-term prospective studies they identified differed greatly in methodologies. However, they concluded that patients with chronic periodontitis may exhibit, over the long term, significantly greater probing depths, marginal bone loss, and incidence of peri-implantitis than periodontally healthy patients.[34] Conversely, Schou et al[35] conducted a similar systematic review of the dental literature from 1986 to 2006 and identified two studies with 5- and 10-year follow-up periods. The studies reported no significant difference in implant survival rates but found that periodontally susceptible patients exhibited a significantly higher incidence of peri-implantitis and peri-implant bone loss. Lee and Wang[5] subsequently reported that recent systematic reviews were in agreement that a history of periodontitis represented an increased risk for implant failure, with odds ratios ranging between 2.3 and 4.7.[10,37,38] All studies concluded that more research is needed to determine the long-term outcomes of implants in periodontally susceptible patients.

The present retrospective investigation aimed to compare the long-term outcomes of dental implants placed in the maxillae of periodontally susceptible and periodontally healthy patients.

MATERIALS AND METHODS

This study was a retrospective review of patient treatment records from a single private practice setting. Study candidates were partially edentulous patients who presented with one or more missing or unsalvageable teeth and who were subsequently treated with implant-supported prosthetic restorations.

Study Inclusion Criteria

All patients who completed at least one annual hygiene prophylaxis and clinical evaluation appointment a minimum of 9.5 years after definitive prosthetic loading were admitted into the study. Any subjects who failed to meet these criteria were excluded from the study.

Patient Evaluations

Patients were routinely subjected to a preliminary evaluation, which included careful review of medical and dental histories, detailed clinical and radiographic examinations, evaluations of oral hygiene, and assessment of the ability to commit to hygiene prophylaxis and clinical monitoring annually at a minimum. A diagnostic workup was performed to evaluate the volume and location of available bone and the esthetic and functional needs of the case relative to the expressed desire of the patient. A study cast was fabricated and mounted on a semiadjustable articulator utilizing a facebow transfer and vertical registration to determine the jaw relationships, available occlusal dimension, proposed implant position(s), crown-root ratio(s), and potential complications. This allowed the creation of a prosthetic wax-up and fabrication of a surgical template to guide placement of the implants relative

to the planned prosthesis. The treatment plan and alternative options were discussed, and signed informed consent was obtained from each patient prior to implant treatment.

Surgical Treatment

Patients were instructed in the use of chlorhexidine digluconate for the chemical control of plaque, which commenced 3 days prior to surgery and continued for 10 days postoperative. Antibiotic prophylaxis involved daily administration of 2 g of amoxicillin/clavulanic acid beginning 2 hours before surgery and continuing for 5 days afterward. On the day of surgery, the patient was anesthetized via local infiltration in the maxilla, inferior alveolar block in the mandible, or general sedation, depending on the desires of the patient and preferences of the clinician. In some cases, midcrestal and terminal vertical releasing incisions were made, followed by elevation of a mucoperiosteal flap, which was kept as small as possible to preserve the periosteal vascular supply. In other cases, osteotomy preparation was performed directly through the soft tissue without incisions or flap elevation to facilitate healing; minimize invasion, pain, edema, bleeding, and hematoma associated with conventional implant placement; and preserve the existing vascular network and soft tissue architecture. For patients requiring extractions, a gentle avulsion technique was used to minimize trauma to the surrounding tissues, and the sockets were thoroughly debrided. Osteotomies were prepared with the aid of a surgical template, and implants with microtextured surfaces (Tapered Screw-Vent, Zimmer Dental) were placed in accordance with the manufacturer's protocol. In low-density bone, an osteocompressive surgical technique was used, which prepared an osteotomy that was 0.5 mm smaller in diameter than the maximum diameter of the tapered implant. When implants were placed into fresh extraction sites, coronal gaps greater than 1 mm were grafted with autogenous bone or beta-tricalcium phosphate mixed with blood and covered with a resorbable barrier membrane (BioMend, Zimmer Dental). Some implants were subjected to delayed loading after a conventional submerged healing period, while other implants were immediately provisionalized with nonoccluding restorations.

Annual Hygiene Prophylaxis and Monitoring

Patients were seen at least once annually for hygiene prophylaxis and monitoring of implant health. Marginal bone changes were calculated from the crest of the ridge to the first implant thread utilizing standardized radiographs taken at implant placement (baseline) and during annual follow-up. A transparent implant template with a 1.0-mm grid, enlarged 25% to help compensate for radiologic distortion, was placed over each radiograph to calculate marginal bone changes relative to the prosthetic platform of the implant. Because of the difficulty in measuring slight variations and an inability to control for exact radiologic distortion with this technique, bone loss was recorded as none (0 mm), less than 0.5 mm, 0.5 to 1 mm, 1 to 1.5 mm, 1.5 to 2 mm, or greater than 2 mm.

Plaque, gingival depths, and probing depths were recorded as references for monitoring the health of the peri-implant mucosa. Crevicular depth measurements were taken on the mesial, distal, lingual, and buccal using a periodontal probe (Hu-Friedy). Implant-related problems were treated, and failed implants were removed and recorded as failures. Patients were subsequently treated for the failed implants.

Data Collection

All patient records in the practice were examined to identify subjects who met the inclusion criteria. Data from each record were entered into spreadsheets (Excel, Microsoft) on a personal computer (Windows XP operating system, Microsoft). For purposes of analysis, patients were assigned to the periodontally susceptible (periodontal) group if they had a history of periodontitis or to the periodontally healthy (control) group if they had no history of periodontitis at the time of implant surgery.

Survival Criteria

Implant survival was defined as implant immobility during manual testing, absence of peri-implant radiolucency, no irresolvable clinical symptoms or mechanical problems, clinical function, and fulfillment of prosthodontic purpose. Because of the retrospective nature of the study, all data were derived from patient records and accompanying periapical radiographs. Clinically failed implants that were removed and subsequently replaced were recorded as failures in the spreadsheet. Roos et al[39] postulated that expected bone loss should be less than 2.8 mm after 10 years of functional loading.

Statistical Methods

As stated, study variables were summarized by creating two subgroups of subjects for analytic purposes: patients with a history of periodontitis (periodontally susceptible) were assigned to a periodontal group, and those without a history of periodontitis (periodontally healthy) were assigned to a control group. For each analysis group, categorical study endpoints were summarized as frequencies and percentages for each level of the variable. Continuous variables were summarized using descriptive statistics (n, mean, median, standard deviation, minimum, and maximum). Between-group comparisons of categorical endpoints were made using the Fisher exact test (dichotomous endpoints) or

Table 1 Summary of Implant Data

	Control (n = 35)	Periodontal (n = 138)	All (n = 173)	P*
Implant length (mm)				.9926
10	5	19	24	
13	25	100	125	
16	5	19	24	
Implant diameter (mm)				.0000
3.7	16	113	129	
4.7	19	25	44	
No. of implants placed/patient				.2750
1	7	4	11	
2	4	5	9	
3	3	5	8	
4	0	0	0	
5	1	2	3	
6	1	8	9	
7	0	2	2	
8	0	1	1	
9	0	2	2	
10	0	0	0	
11	0	1	1	
Location of implants				.0420
Central incisor	5	31	36	
Lateral incisor	8	23	31	
Canine	9	27	36	
First premolar	2	5	7	
Second premolar	7	10	7	
First molar	4	18	22	
Second molar	0	24	24	
Prosthesis type				.0000
Single tooth	11	5	16	
Fixed partial denture	24	133 (including 1 failure)	157 (including 1 failure)	

*Fisher exact test (2 × 2); likelihood ratio chi-square (r^2); Student *t* test (pooled sample variance); *F* test (folded, equal sample variances); Satterthwaite *t* test; and Wilcoxon nonparametric tests when sample variances were unequal (sig folded *F* test).

the likelihood ratio chi-square test (polychotomous endpoints). Between-group comparisons of continuous variables were made using the Student *t* test, with sample variances pooled. A (folded) *F* test was used to assess the equality of sample variances between the groups. If the assumption of equal sample variances in the pooled *t* test was unmet, then the Satterthwaite *t* test and Wilcoxon rank sum test were performed. Statistical significance was inferred at the nominal level of type I (alpha) error of .05. Significance levels were not adjusted for multiplicity. All analyses were performed using statistical software (SAS) on a personal computer (Windows XP operating system, Microsoft).

RESULTS

Study Subjects and Treatment Data

Of 60 patients identified as having completed at least 9.5 years of clinical follow-up, 14 were excluded because of inconsistent follow-up patterns (n = 7), poor oral hygiene practices (n = 4), or late development of concomitant medical conditions that could have affected implant outcomes (long-term corticosteroid use, n = 1; uncontrolled type 1 diabetes, n = 1; jawbone irradiation for oral cancer, n = 1). The final study population therefore consisted of 46 subjects (19 men, 47 women) assigned to either the control (periodontally healthy) (n = 16) or the periodontal (periodontally susceptible) (n = 30) cohort. The mean age of subjects at surgery was 51 years. In all, 173 implants were placed (35 in control subjects to support 21 prostheses and 138 in periodontal subjects to support 45 prostheses), with lengths of 10, 13, or 16 mm and diameters of 3.7 or 4.7 mm (Table 1).

Outcome Variables

The study groups did not differ significantly ($P = .1761$) in the frequencies of prosthesis-related adverse events, of which porcelain fracture was the most prevalent (control = 1; periodontal = 11) (Table 2). There was one report of cement failure and one incidence of frame-

Table 2 Summary of Results

	Control (n = 35)	Periodontal (n = 138)	All (n = 173)	P*
Non–failure-related adverse events				.1761
None	32 (91.4%)	1 (0.7%)	2 (1.2%)	
Cement failure	1 (2.9%	11 (8.0%)	12 (7.0%)	
Porcelain fracture	1 (2.9%)	1 (0.75)	2 (1.2%)	
Framework fracture	1 (2.9%)			
Implant survival				> .9999
Surviving	35 (100%)	137 (99.3%)	172 (99.4%)	
Failed	0 (0.0%)	1 (0.7%)	1 (0.6%)	
Bone loss (mm)				.1008
0	30 (85.7%)	85 (62.0%)	115 (66.9%)	
0.5	2 (5.7%)	30 (21.9%)	32 (18.6%)	
1.5	3 (8.6%)	18 (13.1%)	21 (12.2%)	
2.5	0 (0.0%)	3 (2.2%)	3 (1.7%)	
3.5+	0 (0.0%)	1 (0.7%)	1 (0.6%)	

*Fisher exact test (2 × 2); likelihood ratio chi-square (r^2); Student *t* test (pooled sample variance); *F* test (folded, equal sample variances); Satterthwaite *t* test; and Wilcoxon nonparametric tests when sample variances unequal (sig folded *F* test).

work fracture in each group. Consequently, the cumulative prosthesis survival rate was 90.9% (n = 60/66). In all cases, failed prostheses were replaced immediately and implants continued to function without problems.

The majority of study implants (66.9%) had no bone loss (Table 2). The 61 implants with documented bone loss were overwhelmingly clustered in the periodontal cohort and among women as compared to men regardless of cohort. When implants were categorized as either having or not having bone loss, there was a significant difference (P = .0085) between control and periodontal implants. When bone loss data were grouped into millimeter segments and analyzed, there was no statistically significant difference (P = .1008) between groups. By implant diameter, the rate of bone loss was 2.625 times lower for 4.7-mm implants (n = 7/44, 16%) as compared to 3.7-mm implants (n = 54/129, 42%). By prosthesis type, bone loss occurred in 64% of the fixed partial dentures (n = 32/50; 10 control and 40 periodontal prostheses) and in 31.3% of single-tooth replacements (n = 5/16; 3 control and 2 periodontal prostheses). Only one surviving implant (n = 1/172), located in the periodontal cohort, met the definition of pathologic bone loss (ie, > 2.8 mm after 10 years of function).[39] The patient, a 50-year-old woman treated with fixed partial dentures supported by 11 maxillary implants, sustained 7.5 mm of bone loss around one canine implant. While five other implants in this patient also lost 1.5 mm of bone after 124 months of functioning, this bone loss was considered nonpathologic according to the criteria of Roos et al.[39] Implants in this patient continued to function without problem, and the patient declined subsequent guided bone regeneration to repair the bone loss around the canine. No other cases of pathologic bone loss were found in either cohort.

The cumulative implant survival rates over 9.5 years were 99.4% (n = 172/173) for all implants placed, 99.3% (n = 137/138) for all periodontal implants, and 100% (n = 35/35) for all control implants. A single implant (3.7 × 10 mm) failed to osseointegrate in the maxillary right first premolar area of a 72-year-old female periodontal patient who developed a localized infection shortly after placement. The patient was treated for the infection, and seven adjacent implants osseointegrated successfully and adequately supported the planned prostheses. The patient was restored with two fixed partial dentures and experienced no other complications during 133 months of clinical monitoring.

DISCUSSION

The finding that bone loss rates were predominantly clustered among female patients (62.5%), regardless of periodontal status, could not be explained by the present data. It is surmised, however, that this loss may have been influenced, in part, by the natural decline in estrogen levels in this segment of the subject population, whose mean age was 54 years (range = 35 to 72 years, mode = 55 years) at the time of implant placement. The median age of menopause onset globally has been reported to range from a low of 42.1 years in Asia to a high of 53 years in Latin America.[40] The rapid decline in endogenous estrogen production that occurs in menopause has been associated with significant bone loss, increased bone fragility, and elevated risks of bone fracture and/or tooth loss.[41,42] Sex steroid levels can also reportedly exert profound effects on multiple immunologic parameters regulating both the amplification and resolution of inflammation.[31,42,43] There is strong evidence to support the concept of

sexual dimorphisms in both innate and acquired immunity.[31,43] Injury and infection have been associated with higher levels of inflammatory cytokines (eg, interleukin-1β, tumor necrosis factor-α) in men than in women, which parallels reported sex-specific differences in periodontitis infections.[42,43] Bone loss in this group occurred late in the follow-up period, around 114 months, which may be linked to a gradual decline in estrogen levels. Further investigations in this area would be of clinical interest.

By definition, retrospective studies use existing data, such as those found in patient medical records, that have been recorded for reasons other than research.[44] Although the retrospective study design is often discouraged when prospective research is feasible, such studies can serve an important pilot function by helping to formulate appropriate study questions, clarify hypotheses, and identify issues and required sample sizes for evaluation in prospective studies.[44] A major limitation with all retrospective studies, including the present one, is the lack of an overriding research protocol, which allows for unintentional bias and skewing of variables that can affect the outcome. For example, the method of radiographic analysis (ie, placing periapical radiographs on a light box and measuring crestal bone changes with a transparent overlay grid) precluded the ability to detect small, incremental changes in bone height because of radiographic distortion and thereby a margin of error in changes of less than 1 mm.[45] While study variables may be dampened or skewed by inconsistencies in patient selection or treatment regimens, it is not known whether these differences were minimized in the present study, in which all patients were treated by the same clinician. The influence of clinician experience in treating periodontally susceptible patients with dental implants would be an interesting topic for future prospective research.

Comparisons with data from other 10-year implant studies were difficult because of differences in focus and the finding that most retrospective studies tended to be short-term analyses. In a 10-year retrospective clinical and radiographic study, Bonde et al[46] reported 94% implant and prosthesis survival rates for single-tooth replacements restored by dental students. In the present study, there were 2 cases of framework breakage, 2 crowns with cement failure, and 12 cases of porcelain fracture. In comparison, Bonde et al[46] reported 5 technical complications, 5 episodes of peri-implant inflammation caused by excess cement, and 2 implant fistulas after a 10-year follow-up of single-tooth prostheses restored by dental students. Lekholm et al[47] conducted a 10-year prospective study of implants placed in partially edentulous patients and reported cumulative implant survival rates of 90.2% for the maxilla and 93.7% for the mandible.

The present study only included data on maxillary implants because no patients with mandibular implants met the study's minimum 9.5-year follow-up inclusion criterion. The cumulative implant survival rates of 99.4% for all implants placed, 99.3% for implants in the periodontal cohort, and 100% for implants placed in the control cohort surpassed the 90.2% 10-year survival rate of maxillary implants reported by Lekholm et al.[47] Further research should be conducted to determine whether the combination of osteocompressive surgical technique and textured implant surface reported by in vitro studies,[48–50] thought to be beneficial in stabilizing implants in low-density bone, helped to achieve the high implant survival rates in this study. The present study did, however, affirm the findings of Schou et al[35] that implants placed in periodontally susceptible patients were not necessarily more prone to failure, but that periodontally susceptible patients exhibited a significantly higher incidence of peri-implant bone loss.

CONCLUSION

Periodontal susceptibility resulted in increased bone loss but did not affect implant survival in the present retrospective cohort.

ACKNOWLEDGMENTS

The authors thank Christopher T. Mesman, MS, Zimmer Inc (Orthopedics), for statistical support, and Hai Bo Wen, PhD, and Michael M. Warner, MA, Zimmer Dental Inc, for scientific consultation and manuscript assistance, respectively.

REFERENCES

1. Cohan FM. What are bacterial species? Annu Rev Microbiol 2002; 56:457–458.
2. Siqueira JF Jr, Rocas IN. The oral microbiota: General overview, taxonomy, and nucleic acid techniques. Methods Mol Biol 2010;666:55–69.
3. Nunn ME. Understanding the etiology of periodontitis: An overview of periodontal risk factors. Periodontology 2000 2003;32:11–23.
4. Kaufman E. The new classification system of periodontal diseases and conditions. Dent Today 2001;20:102–105.
5. Lee A, Wang HL. Biofilm related to dental implants. Implant Dent 2010;19:387–393.
6. Laney WR (ed). Glossary of Oral and Maxillofacial Implants. Chicago: Quintessence, 2007:122–124.
7. Gouvoussis J, Sindhusake D, Yeung S. Cross-infection from periodontitis sites to failing implant sites in the same mouth. Int J Oral Maxillofac Implants 1997;12:666–673.
8. Meffert RM. What causes peri-implantitis? J Calif Dent Assoc 1991; 19:53–57.
9. Renvert S, Roos-Jansaker AM, Lindahl C, Renvert H, Persson GR. Infection at titanium implants with or without a clinical diagnosis of inflammation. Clin Oral Implants Res 2007;18:509–516.

10. Heydenrijk K, Meijer HJA, van der Riijden WA, Raghoebar GM, Vissink A, Stegenga B. Microbiota around root-form endosseous implants: A review of the literature. Int J Oral Maxillofac Implants 2002;17:829–838.
11. Karoussis IK, Muller S, Salvi GE, Heitz-Mayfield LJA, Bragger U, Lang NP. Association between periodontal and peri-implant conditions: A 10-year prospective study. Clin Oral Implants Res 2004;15:1–7.
12. Meffert RM. Periodontitis and periimplantitis: One and the same? Pract Periodontics Aesthet Dent 1993;5:79–82.
13. Nowzari H, Botero JE, DeGiacomo M, Villacres MC, Rich SK. Microbiology and cytokine levels around healthy dental implants and teeth. Clin Implant Dent Relat Res 2008;10:166–173.
14. Ryder MI. The influence of smoking on host responses in periodontal infections. Periodontology 2000 2007;43:267–277.
15. Kapila YL, Kashani H. Cocaine-associated rapid gingival recession and dental erosion. A case report. J Periodontol 1997;68:485–488.
16. Khocht A, Janal M, Schleifer S, Keller S. The influence of gingival margin recession on loss of clinical attachment in alcohol-dependent patients without medical disorders. J Periodontol 2003;74:485–493.
17. Turkyilmaz I. Oral manifestations of "meth mouth": A case report. J Contemp Dent Pract 2010;11:e073–080.
18. Gürsoy M, Könönen E, Gürsoy UK, Tervahartiala T, Pajukanta R, Sorsa T. Periodontal status and neutrophilic enzyme levels in gingival crevicular fluid during pregnancy and post-partum. J Periodontol 2010;81:1790–1796.
19. Lemos SS, Oliveira FA, Vencio EF. Periodontal disease and oral hygiene benefits in HIV seropositive and AIDS patients. Med Oral Patol Oral Cir Bucal 2010;15:e417–421.
20. Nagasawa T, Noda M, Katagiri S, et al. Relationship between periodontitis and diabetes—Importance of a clinical study to prove the vicious cycle. Intern Med 2010;49:881–885.
21. Suzuki JI, Aoyama N, Ogawa M, et al. Periodontitis and cardiovascular diseases [review]. Expert Opin Ther Targets 2010;14:1023–1027.
22. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: A systematic review and meta-analysis. J Periodontol 2010;81:1708–1724.
23. Baker PJ, Roopenian DC. Genetic susceptibility to chronic periodontal disease. Microbes Infect 2002;4:1157–1167.
24. Nares S. The genetic relationship to periodontal disease. Periodontology 2000 2003;32:36–49.
25. Hacker BM, Roberts FA. Periodontal disease pathogenesis: Genetic risk factors and paradigm shift. Pract Proced Aesthet Dent 2005; 17:97–102.
26. Jingjin L, Zemin G, Xin M, et al. Correlation between an interlukin-6-572C/g polymorphism and chronic periodontitis. Int J Periodontics Restorative Dent 2010;30:301–305.
27. Soga Y, Nishimura F, Ohyama H, Maeda H, Takashiba S, Murayama Y. Tumor necrosis factor-alpha gene (TNF-α) -1031/ -863, -857 single-nucleotide polymorphisms (SNPs) are associated with severe adult periodontitis in Japanese. J Clin Periodontol 2003;30:524–531.
28. Ikezawa I, Tai H, Shimada Y, Komatsu Y, Galicia JC, Yoshie H. Imbalance between soluble tumour necrosis factor receptors type 1 and 2 in chronic periodontitis. J Clin Periodontol 2005;32:1047–1054.
29. American Academy of Periodontology. The pathogenesis of periodontal diseases. J Periodontol 1999;70:457–470.
30. Listgarten MA. Pathogenesis of periodontitis. J Clin Periodontol 1986;13:418–430.
31. Shiau HJ, Reynolds MA. Sex differences in destructive periodontal disease: Exploring the biological basis. J Periodontol 2010;81: 1505–1517.
32. Khalili J. Genetic test and periodontal disease. Lik Sprava 2010;1–2: 91–95.
33. Roccuzzo M, De Angelis N, Bonino L, Aglietta M. Ten-year results of a three-arm prospective cohort study on implants in periodontally compromised patients. Part I: Implant loss and radiographic bone loss. Clin Oral Implants Res 2010;21:490–496.
34. Karoussis IK, Kotsovilis S, Fourmousis I. A comprehensive and critical review of dental implant prognosis in periodontally compromised partially edentulous patients. Clin Oral Implants Res 2007;18:669–679.
35. Schou S, Holmstrup P, Worthington HV, Esposito M. Outcome of implant therapy in patients with previous tooth loss due to periodontitis. Clin Oral Implants Res 2006;17(suppl 2):104–123.
36. Van der Weijden GA, van Bemmel KM, Renvert S. Implant therapy in partially edentulous periodontally compromised patients: A review. J Clin Periodontol 2005;32:506–511.
37. Ong CT, Ivanovski S, Needleman IG, et al. Systematic review of implant outcomes in treated periodontitis subjects. J Clin Periodontol 2008;35:438–462.
38. Heitz-Mayfield LJ, Lang NP. Comparative biology of chronic and aggressive periodontitis vs peri-implantitis. Periodontol 2000 2010; 53:167–181.
39. Roos J, Sennerby L, Lekholm U, Jemt T, Gröndahl K, Albrektsson T. A qualitative and quantitative method for evaluating implant success: A 5-year retrospective analysis of the Brånemark implant. Int J Oral Maxillofac Implants 1997;12:504–514.
40. Palacios S, Henderson VW, Siseles N, et al. Age of menopause and impact of climacteric symptoms by geographical region. Climacteric 2010;13:419–428.
41. Sacco SM, Ward WE. Revisiting estrogen: Efficacy and safety for postmenopausal bone health. J Osteoporos 2010;22:708931.
42. Meisel P, Reifenberger J, Haase R, et al. Women are periodontally healthier than men, but why don't they have more teeth than men? Menopause 2008;15:270–275.
43. Shiau HJ, Reynolds MA. Sex differences in destructive periodontal disease: A systematic review. J Periodontol 2010;81:1379–1389.
44. Hess DR. Retrospective studies and chart reviews. Respir Care 2004; 49:1171–1174.
45. Ormianer Z, Palti A. Retrospective clinical evaluation of tapered Screw-Vent implants: Results after up to eight years of clinical function. J Oral Implantol 2008;34:150–160.
46. Bonde MJ, Stokholm R, Isidor F, Schou S. Outcome of implant-supported single-tooth replacements performed by dental students. A 10-year clinical and radiographic retrospective study. Eur J Oral Implantol 2010;3:37–46.
47. Lekholm U, Gunne J, Henry P, et al. Survival of the Brånemark implant in partially edentulous jaws: A 10-year prospective multicenter study. Int J Oral Implantol 1999;14:639–645.
48. Shalabi MM, Wolke JGC, de Ruijter AJE, et al. A mechanical evaluation of implants placed with different surgical techniques into the trabecular bone of goats. J Oral Implantol 2007;33:51–58.
49. Skalak R, Zhao Y. Interaction of force-fitting and surface roughness of implants. Clin Implant Dent Relat Res 2000;2:219–224.
50. Shalabi MM, Wolke JGC, Jansen JA. The effects of implant surface roughness and surgical technique on implant fixation in an in vitro model. Clin Oral Implants Res 2006;17:172–178.