Retrospective Analysis of Implant Survival and the Influence of Periodontal Disease and Immediate Placement on Long-term Results

Cyril I. Evian, BDS, HDD, DMD[1]/Robert Emling, MS, EdD[2]/Edwin S. Rosenberg, DMD[2]/
Jonathan A. Waasdorp, BS[3]/Wendy Halpern, DMD[4]/Shalin Shah, BS[3]/Marela Garcia, DDS[5]

Purpose: *The purpose of this study was to investigate the cumulative survival rates of dental implants placed in a private periodontal practice and the effects of periodontal disease and immediate placement on implant survival.* ***Materials and Methods:*** *A retrospective chart review was conducted on 149 consecutive patients. Each patient had a single implant placed. For the purpose of analysis, patients were divided into 2 groups: those who were periodontally healthy and those who had periodontal disease. Implants were placed into available bone either immediately or after a healing period. All failed implants were removed and recorded. The effects of periodontal status and placement time on implant survival were evaluated using Cox proportional hazards regression and log-rank tests.* ***Results:*** *Of the 149 implants in the study, 22 failed during the observation period. The 127 censored cases (ie, implants that had not failed at the end of the observational period) were observed for a mean of 943 days (SD 932, range 35 to 4,030). Failed implants were observed for a mean of 722 days (SD 1,026, range 18 to 3,548). The presence of periodontal disease appeared to be associated with a greater failure rate, but there was no observed effect associated with time of placement. The percentages of censored immediate placement cases and delayed placement cases were nearly identical. Among the 77 implants associated with periodontal disease, placement time was not strongly associated with percentage censored. Forty-three of the 55 immediately placed implants (78.18%) and 18 of the 22 implants (81.18%) whose placement was delayed were censored. Both Cox proportional hazards regression and log-rank tests established that survival was adversely affected by periodontal disease (*$P < .05$*) but unaffected by time of placement (*$P > .50$*). The lower 1-sided 95% confidence limit for median survival time was 3,548 days for patients without periodontal disease and 1,799 days for patients with disease.* ***Discussion and Conclusion:*** *Implant survival was compromised by a history of periodontitis but not affected by immediate or delayed placement.* INT J ORAL MAXILLOFAC IMPLANTS 2004;19:393–398

Key words: *dental implants, extraction sockets, periodontal susceptibility, survival rates*

During the developmental period of modern implant dentistry, researchers[1] focused primarily on the phenomenon of osseointegration and the best techniques for its achievement and maintenance. When hopeless teeth were extracted, empirical evidence at the time suggested that a healing period of 9 to 12 months was necessary to allow for the formation and maturation of new bone within the socket prior to implantation.[1,2] Patients who suffered from severe periodontitis were required to undergo an extended healing period before implantation.[1]

Periodontitis comprises a variety of pathologic conditions that affect the health of the periodontium.[3] In general, patients tend to exhibit gingival inflammation and loss of the connective tissue attachment to teeth.[3,4] Loss of the periodontal ligament, disruption of its attachment to the cementum, and resorption of the alveolar bone can also occur.[3] Along the root surface, there may be migration of the epithelial attachment and resorption of

[1]Clinical Professor, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania; Private Practice Limited to Periodontics, TMD, and Cosmetic Dentistry, King of Prussia, Pennsylvania.
[2]Clinical Professor, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania.
[3]First-year Dental Student, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania.
[4]Implant Fellow, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania.
[5]Lecturer in Periodontics, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania.

Correspondence to: Dr Cyril I. Evian, 1000 Valley Forge Towers, Suite 112, King of Prussia, PA 19406. Fax: +610 783 7829. E-mmail: cevian@msn.com

Table 1 Comparison of Reported Implant Survival Rates in Fresh and Healed Extraction Sites

Study	Maximum follow-up (y)	Extraction site	Implants			
			No. placed	No. censored*	No. removed	Survival rate (%)
Gomez-Roman and associates[5]	6	Fresh	124	13	4	97.00
Cosci and Cosci[7]	7	Fresh	423	0	2	99.53
Becker and associates[27]	8	Fresh	134	0	9	93.30
Tolman and Keller[28]	6	Fresh	303	10	2	99.30
Wagenberg and Ginsburg[29]	11	Fresh	1,081	0	54	95.00
Mensdorff-Pouilly and associates[30]	1	Fresh	93	8	7	91.80
Schwartz-Arad and Chaushu[31]	7	Fresh	95	0	9	94.70
De Leonardis and associates[32]	1	Healed	100	0	0	100.00
Schwartz-Arad and Dolev[33]	5	Healed	87	0	4	95.40
Lekholm and associates[34]	10	Healed	461	123	34	89.90
ten Bruggenkate and associates[35]	7	Healed	253	28	7	96.90
Ahlqvist and associates[36]	2	Healed	269	0	14	94.80

*Withdrawn or lost to follow-up.

the supporting bone.[3] It is widely held that the initiation and progression of periodontitis are dependent on the presence of microorganisms capable of causing the disease.[3] According to some studies, dental implants placed in patients with a history of periodontal disease have demonstrated survival rates comparable to those for implants placed in patients with no history of the disease.[5–7]

Peri-implantitis and its retrograde form are periodontal infections associated with dental implants. The disease consists of inflammation or infection of the tissues that surround and support the implant. Conventional peri-implantitis originates in the soft tissue sulcus around the implant. In the retrograde form of the disease, lesions develop in the periapical region of the implant. Bacterial contamination is the primary etiology of both infections; it may be caused by plaque-induced inflammation[8–10] or seeding from extracted or adjacent endodontically infected teeth.[10–16] Retrograde peri-implantitis has also been attributed to excessive heating of the bone during the creation of the osteotomy site,[14,17,18] residual bone cavities created by the placement of implants that are shorter than the prepared surgical sites,[14,19] and microfractures in the bone caused by overloading, premature loading, or excessive lateral forces.[10,14]

As the disease progresses, the infection can advance along the surface of the implant and cause resorption of the hard tissue. If left untreated, peri-implant infections can ultimately lead to implant loss. Studies have shown that the bacteria associated with peri-implantitis are similar to those that cause periodontitis[20,21] but destroy tissues around implants much more rapidly than around natural dentition.[10] Some question whether implants with hydroxyapatite (HA) coatings are more susceptible to periodontal infection. To date, this concern has not been validated by any controlled, prospective clinical research. One prospective, 3-year multicenter study of more than 2,900 HA-coated and non-HA-coated implants found no clinical difference in adverse periodontal responses between coated and uncoated implants after 36 months of clinical follow-up.[22] The interval studied was too short to allow researchers to come to a decisive conclusion about HA-coated implants. Other research has demonstrated that it is possible to detoxify both grit-blasted and HA-coated implant surfaces when peri-implantitis occurs.[23,24] At the present time, it is unknown whether textured implant surfaces may be more vulnerable to infection than machined implant surfaces in patients with past or present periodontal disease.

The timing of implant placement after extraction can vary according to the clinical circumstances of the case. Immediate placement involves placing the implant into a prepared socket immediately following tooth extraction, whereas delayed placement allows an interval for the extraction site to heal prior to implant placement.[25,26] Recent clinical studies have reported that dental implants placed directly into the prepared sockets of freshly extracted teeth[5,7,27–31] achieved clinical results that were comparable to those achieved by placing implants into available mature bone[32–36] (Table 1). Several studies have reported the successful placement of dental implants directly into the extraction sockets of patients with a history of periodontal disease,[37–39] but very little has been reported on the long-term survival of these implants.

The purpose of this investigation was to determine long-term survival rates of dental implants

Table 2 Distribution of Failed and Censored Implants

Variable/group	Total no. of cases	No. of failures	No. of censored	% censored
Periodontal disease				
No	72	6	66	91.67
Yes	77	16	61	79.22
Time of placement				
Immediate	100	15	85	85.00
Delayed	49	7	42	85.71

placed in a private periodontal practice and to analyze the effects of periodontal disease and immediate placement after tooth extraction on long-term implant survival.

MATERIALS AND METHODS

This study was a retrospective chart review of consecutive patients treated in a private periodontal practice by a single clinician. Charts were analyzed for all patients who had implants exposed and loaded for at least 1 year. Investigators (excluding the treating clinician) completed data collection forms for each chart, and the information was entered into a computer database (Microsoft Access; Microsoft, Redmond, WA). A total of 149 dental implants (Paragon; Zimmer Dental, Carlsbad, CA) placed in 149 patients (Table 2) were entered into the database. Data were recorded for patients who received a single implant. Any patient who received multiple implants was excluded from the study. Periodontal disease was diagnosed if probing depths were 5 mm or greater and associated with radiographic signs of bone loss. Periodontal disease was not diagnosed if a fractured tooth or endodontic lesion could have been the cause of the bone loss. Patients who exhibited 1 or more teeth with periodontal disease, or who originally lost their teeth as a result of periodontitis, were considered to have periodontal disease, while those with no history or current clinical manifestations of periodontal disease were considered healthy.

Preoperative documentation was recorded for each patient. A medical history was obtained, and any necessary medical issues were resolved prior to surgery. Patients with contraindicating diseases, such as uncontrolled endocrine disorders, were excluded from implant therapy. Patients with diabetes were treated with implant therapy only if their physicians certified that their disease was under control. All other patients who received implant therapy were included in the study. Endodontic therapy was performed prior to implant placement if a periapical lesion was present in the region of the future implant site. Periodontal treatment was performed prior to or in conjunction with implant placement. In all cases, signed informed patient consent was obtained prior to treatment.

All implants were placed according to a conventional 2-stage surgical procedure. Implants were mainly screw type; a small percentage were a combination of screw and press-fit type. Implant length ranged from 10 to 18 mm, and diameter ranged from 3.3 to 6.0 mm. Numerous implants had HA coatings; the remainder were pure titanium. Sutures were generally removed at 1 week. Implants either were placed immediately into extraction sockets (immediate placement) or into available bone sometime after extraction (delayed placement). In cases where extraction was necessary, implants were not immediately placed if acute infection was present. For situations in which a large opening was present as a result of extraction, a free gingival graft was sutured over the opening. In cases where primary closure was not achievable, small openings were left to granulate in. The majority of implants were exposed between 4 and 6 months after placement. Healing collars were attached to the implants at that time and remained in place until the tissues matured and the patients returned to their restorative dentists for prosthetic restoration. Many different referring dentists performed the restorative procedures.

Patients were instructed to return to the surgical office for regular follow-up maintenance. At each follow-up appointment, data were recorded on how the implants were performing. Implant-related problems were treated, and failed implants were removed and recorded. Implants were considered survivors if they continued to support a load-bearing restoration and were free from irresolvable clinical complaints (eg, peri-implant radiolucency, chronic pain, implant mobility, progressive bone loss). Implants that exhibited advanced bone loss, acute infection, pain, or irresolvable discomfort were removed. Problems with implants were

recorded at each occurrence and, if lesions or problems were severe and the implants were removed, those implants were listed as failures.

Statistical Analysis

The effects of periodontal status and placement time on implant survival were evaluated using Cox proportional hazards regression and log-rank tests.

RESULTS

Of the 149 implants in the study, 22 failed during the observation period. The 127 censored cases (ie, implants that had not failed at the end of the observational period) were observed for a mean of 943 days (SD 932, range 35 to 4,030 days). Failed implants were observed for a mean of 722 days (SD 1,026, range 18 to 3,548 days).

Table 2 shows the distribution of failures and censored observations within the binary categories defined for periodontal disease and time of placement. The presence of periodontal disease appeared to be associated with a greater failure rate, but there was no observed effect associated with time of placement (the percent of censored cases was nearly identical for immediate and delayed placement). Among the 77 implants associated with periodontal disease, placement time was also not strongly associated with percentage censored. Forty-three of the 55 immediately placed implants (78.18%) and 18 of the 22 implants (81.18%) whose placement was delayed were censored. However, these apparent effects on survival rates need to be addressed with the following statistical techniques, which are appropriate for censored observations.

The effects of periodontal status and time of placement on survival time were evaluated using the Cox proportional hazards model. While the likelihood ratio test for the 2-predictor model was statistically significant ($P = .0241$), only periodontal status made a significant contribution to the model (using Wald chi-square, $P = .0122$ for periodontal status versus $P > .5$ for time of placement).

The log-rank test for equality of the distribution of event times across groups was statistically significant for periodontal status ($P = .0213$) but not for time of placement ($P > .5$; Mantel-Cox) and confirmed the Cox proportional hazards regression findings. The lower 1-sided 95% confidence limit for median survival time was 3,548 days for patients without periodontal disease and 1,799 days for patients with disease.

DISCUSSION

Since the patients in this study were treated in a periodontal office, the study population consisted of a large group of patients with past or present periodontal disease, as well as a large number of periodontally healthy patients who sought solely implant therapy. The presence or absence of periodontal disease and immediate versus delayed implant placement were the only variables studied. The patients in the study were partially edentulous, which increased the possibility of implant infection from pathogens associated with the natural teeth in the mouth.

Some studies have reported survival rates for dental implants placed in patients with a history of periodontal disease comparable to survival rates for implants placed in patients with no history of the disease.[5–7] One study evaluated only implants placed in anterior positions[5] while the other study was a series of case reports describing factors affecting immediate implant placement.[7] The present study, however, indicates a significant difference in long-term survival between patients with periodontal disease and patients without it, regardless of whether placement was immediate or delayed.

Since only the surgical phase of implant therapy was conducted in the authors' office, and the patients were referred back to their dentists for restoration, some of the patients did not return for routine maintenance or annual follow-up and presented only when problems occurred. The influence of the missing data cannot be known and could affect the findings. However, the survival rate of 91.67% for the implants in the healthy group is similar to implant survival rates in other studies reported in the literature.[40–42]

HA coatings may be associated with greater susceptibility to periodontal infections. Short-term data have indicated no difference between machined implants and HA-coated implants.[22,43] To date, this concern has not been validated by any controlled, long-term prospective clinical research. At the present time, it is unknown whether textured implant surfaces may be more vulnerable to infection than machined implant surfaces in patients with past or present periodontal disease. Numerous implants in the present study had HA coatings and this factor may have influenced the outcome, especially in the periodontally diseased group. The remainder of the implants were pure titanium with machined, etched, or roughened surfaces. Analysis of these data by surface properties will be the subject of a future study.

Endodontic problems associated with teeth to be extracted may affect the long-term survival of implants immediately placed into the same sites. Adjacent endodontically involved teeth have been shown to involve implants with retrograde periimplantitis.[14,44] Teeth with endodontic problems, treated or untreated, may also influence long-term survival in immediate-placement situations.

Numerous implants in this investigation were placed at the same time as periodontal surgical procedures were being carried out. The influence of this cotherapy on implant contamination during the procedure has not been investigated but is a subject for a future study. Primary closure over immediately placed implants is another variable that could influence the outcome of the present study. Although gingival grafts were placed over the socket if primary closure was not achieved, in numerous cases the socket may have had exposure to the oral environment during healing.[45] Therefore, factors such as soft tissue management[46] and the previously discussed variables may have influenced this outcome.

Based on the findings of this study, the investigators suggest that a history of periodontal disease does not preclude placement of implants directly into extraction sockets.

CONCLUSIONS

Past or present periodontal disease compromised implant survival in this patient population. Immediate placement into extraction sockets in patients with periodontal disease or a history thereof altered the outcome. Present or past periodontal disease appeared to increase the risk of implant failure.

ACKNOWLEDGMENTS

The authors thank Michael M. Warner, MA (Zimmer Dental), for his assistance in preparing the manuscript.

REFERENCES

1. Brånemark P-I, Hansson BO, Adell R, et al. Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10-year period. Scand J Plast Reconstr Surg 1977; 111(16 suppl):1–132.
2. Adell R, Lekholm U, Brånemark P-I. Surgical procedures. In: Brånemark P-I, Zarb GA, Albrektsson T (eds). Tissue-Integrated Prostheses. Osseointegration in Clinical Dentistry. Chicago: Quintessence, 1985:211–232.
3. American Academy of Periodontology. The pathogenesis of periodontal diseases. J Periodontol 1999;70:457–470.
4. Listgarten MA. Pathogenesis of periodontitis. J Clin Periodontol 1986;13:418–430.
5. Gomez-Roman G, Kruppenbacher M, Weber H, Schulte W. Immediate postextraction implant placement with root-analog stepped implants: Surgical procedure and statistical outcome after 6 years. Int J Oral Maxillofac Implants 2001; 16:503–513.
6. Becker W, Becker BE, Hujoel P. Retrospective case series analysis of the factors determining immediate implant placement. Compend Contin Educ Dent 2000;21:805–817.
7. Cosci F, Cosci B. A 7-year retrospective study of 423 immediate implants. Compend Contin Educ Dent 1997;18: 940–950.
8. Bauman GR, Mills M, Rapley JW, Hallmon WW. Plaque-induced inflammation around implants. Int J Oral Maxillofac Implants 1992;7:330–337.
9. Warrer K, Buser D, Lang NP, Karring T. Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa. An experimental study in monkeys. Clin Oral Implants Res 1995;6:131–138.
10. Meffert R. Periodontitis and periimplantitis: One and the same? Pract Periodontics Aesthet Dent 1993;5:79–82.
11. Shaffer MD, Juruaz DA, Haggerty PC. The effect of periradicular endodontic pathosis on the apical region of adjacent implants. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:578–581.
12. Mombelli A. Etiology, diagnosis and treatment considerations in peri-implantitis. Curr Opin Periodontol 1997;4:127–136.
13. McAllister BS, Masters D, Meffert RM. Treatment of implants demonstrating perioapical radiolucencies. Pract Periodontics Aesthet Dent 1992;4(9):37–41.
14. Ayangco L, Sheridan PJ. Development and treatment of retrograde peri-implantitis involving a site with a history of failed endodontic and apicoectomy procedures: A series of reports. Int J Oral Maxillofac Implants 2001;16:412–417.
15. 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.
16. Sussman HI. Endodontic pathology leading to implant failure—A case report. J Oral Implantol 1997;23:112–115.
17. Eriksson RA, Adell R. Temperatures during drilling for the placement of implants using the osseointegration technique. J Oral Maxillofac Surg 1986;44:4–7.
18. Watanabe F, Tawada Y, Komatsu S, Hata Y. Heat distribution in bone during preparation of implant sites: Heat analysis by real-time thermography. Int J Oral Maxillofac Implants 1992;7:212–219.
19. Reiser GM, Nevins M. The implant periapical lesion: Etiology, prevention and treatment. Compend Contin Educ Dent 1995;16:768–777.
20. Listgarten MA, Lai C-H. Comparative microbiological characteristics of failing implants and periodontally diseased teeth. J Periodontol 1999;70:431–437.
21. Eke PI, Braswell LD, Fritz ME. Microbiota associated with experimental peri-implantitis and periodontitis in adult *Macaca mulatta* monkeys. J Periodontol 1998;69:190–194.
22. Morris HF, Ochi S, Spray JR, Olson JW. Periodontal-type measurements associated with hydroxyapatite-coated and non-coated implants: Uncovering to 36 months. Ann Periodontol 2000;5(1):56–67.
23. Zablotsky MH, Diedrich DL, Meffert RM. Detoxification of endotoxin-contaminated titanium and hydroxylapatite-coated surfaces utilizing various chemotherapeutic and mechanical modalities. Implant Dent 1992;1:154–158.

24. Zablotsky MH. A retrospective analysis of the management of ailing and failing endosseous dental implants. Implant Dent 1998;7:185–191.
25. Grunder U, Polizzi G, Goené R, et al. A 3-year prospective multicenter follow-up report on the immediate and delayed-immediate placement of implants. Int J Oral Maxillofac Implants 1999;14:210–216.
26. Nemcovsky CE, Artzi Z, Moses O, Gelernter I. Healing of dehiscence defects at delayed-immediate implant sites primarily closed by a rotated palatal flap following extraction. Int J Oral Maxillofac Implants 2000;15:550–558.
27. Becker BE, Becker W, Ricci A, Geurs N. A prospective clinical trial of endosseous screw-shaped implants placed at the time of tooth extraction without augmentation. J Periodontol 1998;69:920–926.
28. Tolman DE, Keller EE. Endosseous implant placement immediately following dental extraction and alveoloplasty: Preliminary report with 6-year follow-up. Int J Oral Maxillofac Implants 1991;6:24–28.
29. Wagenberg BD, Ginsburg TR. Immediate implant placement on removal of the natural tooth: Retrospective analysis of 1,081 implants. Compend Contin Educ Dent 2001;22: 399–409.
30. Mensdorff-Pouilly N, Haas R, Mailath G, Watzek G. The immediate implant: A retrospective study comparing the different types of immediate implantation. Int J Oral Maxillofac Implants 1994;9:571–578.
31. Schwartz-Arad D, Chaushu G. Placement of implants into fresh extraction sites: 4 to 7 years retrospective evaluation of 95 immediate implants. J Periodontol 1997;68:1110–1116.
32. De Leonardis D, Garg AK, Pecora GE, Andreana S. Osseointegration of rough acid-etched implants: One-year follow-up of placement of 100 Minimatic implants. Int J Oral Maxillofac Implants 1997;12:65–73.
33. Schwartz-Arad D, Dolev E. The challenge of endosseous implants placed in the posterior partially edentulous maxilla: A clinical report. Int J Oral Maxillofac Implants 2000;15: 261–264.
34. 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 Maxillofac Implants 1999;14:639–645.
35. ten Bruggenkate CM, Asikainen P, Foitzik C, Krekeler G, Sutter F. Short (6-mm) nonsubmerged dental implants: Results of a multicenter clinical trial of 1 to 7 years. Int J Oral Maxillofac Implants 1998;13:791–798.
36. Ahlqvist J, Borg K, Gunne J, Nilson H, Olsson M, Åstrand P. Osseointegrated implants in edentulous jaws: A 2-year longitudinal study. Int J Oral Maxillofac Implants 1990;5: 155–163.
37. Novaes AB Jr, Vidigal GM, Novaes AB, Grisi MFM, Polloini S, Rosa A. Immediate implants placed into infected sites: A histomorphometric study in dogs. Int J Oral Maxillofac Implants 1998;13:422–427.
38. Becker BE, Becker W, Ricci A, Geurs N. A prospective clinical trial of endosseous screw-shaped implants placed at the time of tooth extraction without augmentation. J Periodontol 1998;69:920–926.
39. Babbush CA. Immediate implant placement in fresh extraction sites. Dent Surg Products 1998;3(2):32–36.
40. Nevins M, Langer B. The successful application of osseointegrated implants to the posterior jaw: A long-term retrospective study. Int J Oral Maxillofac Implants 1993;8:428–432.
41. Lekholm U, van Steenberghe D, Herrmann I, Bolender C, Folmer T, Gunne J. Osseointegrated implants in the treatment of partially edentulous jaws: A prospective 5-year multicenter report. Int J Oral Maxillofac Implants 1994;9: 627–635.
42. Jemt T, Lekholm U. Oral implant treatment in posterior partially edentulous jaws: A five-year follow-up report. Int J Oral Maxillofac Implants 1993;8:635–640.
43. Evian CI. Comparison of hydroxylapatite-coated Micro-Vent and pure titanium Swede-Vent implants. Int J Oral Maxillofac Implants 1996;11:639–644.
44. Jalbout ZN, Tarnow DP. The implant periapical lesion: Four case reports and review of the literature. Pract Proced Aesthet Dent 2001;13(2):107–112.
45. Evian CI, Cutler SA. Autogenous gingival grafts as epithelial barriers for immediate implants: Case reports. J Periodontol 1994;65:201–210.
46. Rosenquist B. A comparison of various methods of soft tissue management following the immediate placement of implants into extraction sites. Int J Oral Maxillofac Implants 1997;12:43–51.