Immediate Placement of Implants into Infected Sites: A Systematic Review

Bruno Ramos Chrcanovic, DDS, MSc;* Maximiliano Delany Martins, PhD;† Ann Wennerberg, DDS, PhD‡

ABSTRACT

Background: Traditionally, before placing dental implants, the compromised teeth are removed and the extraction sockets are left to heal for several months. To preserve the alveolar bone level from the collapse caused by healing and to reduce treatment time in situations in which tooth extraction precedes implant placement, some clinicians began to install the implant immediately into the postextraction socket without waiting for the site to heal.

Purpose: The purpose of this study was to review the literature regarding treatment outcomes of immediate implant placement into sites exhibiting pathology after clinical procedures to perform the decontamination of the implant's site. The following questions were raised: Does the presence of periodontal or endodontic infection affect immediate implant placement success? What is suggested to address the infection in the socket prior to immediate placement?

Materials and Methods: An electronic search in PubMed (U.S. National Library of Medicine, Bethesda, MD, USA) was undertaken in March 2013. The titles and abstracts from these results were read to identify studies within the selection criteria. Eligibility criteria included both animal and human studies, and excluded any review and case reports articles. The publication's intervention had to have been implant placement into a site classified as having an infection (periapical, endodontic, perioendodontic, and periodontal).

Results: The search strategy initially yielded 706 references. Thirty-two studies were identified within the selection criteria, from which nine were case reports and review articles and were excluded. Additional hand-searching of the reference lists of selected studies yielded five additional papers.

Conclusions: The high survival rate obtained in several studies supports the hypothesis that implants may be successfully osseointegrated when placed immediately after extraction of teeth presenting endodontic and periodontal lesions, provided that appropriate clinical procedures are performed before the implant surgical procedure such as meticulous cleaning, socket curettage/debridement, and chlorhexidine 0.12% rinse. However, more randomized controlled clinical trials with a longer follow-up are required to confirm this procedure as a safe treatment. Moreover, the outcome measures were not related to the type of infection; the classification of infection was often vague and varied among the studies. The benefits of antibiotic solution irrigation and systemic antibiotic administration in such conditions are not yet proved and remain unclear.

KEY WORDS: dental implants, immediate implant placement, infected extraction sites, infection, tooth socket

INTRODUCTION

Traditionally, before placing dental implants, the compromised teeth are removed and the extraction sockets are left to heal from several months up to 1 year.[1] However, alveolar ridge resorption after tooth extraction may considerably reduce the residual bone volume and affect the favorable positioning of implants which is required to produce optimal restoration. This is even more pronounced in the anterior maxilla, where ridge resorption often creates an unfavorable palatolabial discrepancy between the implant and the prosthesis.[2] Horizontal reductions of up to 50% (5–7 mm) were observed during the first year following single tooth extractions.[3] To preserve the alveolar bone level from

*PhD student, Department of Prosthodontics, Faculty of Odontology, Malmö University, Malmö, Sweden; †professor, Laboratório de Nanoscopia – LabNano, Centro de Desenvolvimento da Tecnologia Nuclear – CDTN, Comissão Nacional de Energia Nuclear – CNEN, Belo Horizonte, Brazil; ‡professor and head, Department of Prosthodontics, Faculty of Odontology, Malmö University, Malmö, Sweden

Reprint requests: Dr. Bruno Ramos Chrcanovic, Department of Prosthodontics, Faculty of Odontology, Malmö University, Carl Gustafs väg 34, SE-205 06 Malmö, Sweden; e-mail: bruno.chrcanovic@mah.se; brunochrcanovic@hotmail.com

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the collapse caused by healing and to reduce treatment time in situations in which tooth extraction precedes implant placement, it is sometimes advisable to install the implant immediately into the postextraction socket, without waiting for the site to heal. Schulte and Heimke[4] first introduced this concept in 1976. Anneroth and colleagues[5] were the first to publish a study in an animal model (monkeys). In 1989, Lazzara[6] first reported immediate implant placement in an extraction socket in humans. Since then, this treatment modality has received much attention in the literature.[7]

Immediate implant placement has both social and economic advantages. The overall treatment time is reduced, a second surgical intervention is avoided, and there is a decrease in rehabilitation treatment time[8] because it minimizes the number of surgical procedures by combining extraction, implant placement, and bone grafting (if needed) into one appointment.[9] Less evident advantages comprise improved implant survival rates, enhanced hard and soft tissue maintenance, and there is the ability to place the fixture in an ideal axial position.[10]

Frequently, compromised teeth that are indicated for extraction are enveloped in infection, which conventionally contraindicate their immediate replacement with endosseous dental implants[8] because of the risk of microbial interference with the healing process.[11,12] Some studies on immediate implants suggest that this procedure should be avoided in the presence of periapical or periodontal pathosis,[10,13,14] and clinical reports have suggested that history of periodontal or endodontic infections is a predictive marker for implant infection and failure.[15,16] Alsaadi and colleagues,[17] in a large consecutive case study, noted a greater tendency toward implant failure in sites with apical lesions, especially with machined-surface implants. Additionally, cases of retrograde peri-implantitis have been thought to result from placement into such sites.[16,18] The presence of chronic periodontal disease has also been correlated with an increased risk of implant failure.[11,19–22] This clinical experience has led most clinicians to avoid the immediate placement of endosseous dental implants at infected sites and to consider infection a contraindication for immediate implantation.[8] A published systematic review[23] emphasized the paucity of available literature discussing this subject. It also stressed the need for studies incorporating designs that eliminate confounding variables, including implant placement immediately compared with placement in intact ridges, implant placement in sites with periapical pathology and in sites without periapical pathology, implant placement in sites with periapical pathology and sites without periapical pathology in similar areas of the mouth, and when comparing these two treatment modalities in the same patient.

The purpose of this article was to review the literature regarding treatment outcomes of immediate implant placement into sites exhibiting pathology after clinical procedures to perform the decontamination of the implant's site and provide treatment recommendations. It was discussed whether the presence of periodontal or endodontic infection compromises immediate implant placement success and whether it is advised to combat the socket infection prior to immediate placement.

MATERIALS AND METHODS

Objectives

The purpose of the present study was to review the literature regarding treatment outcomes of immediate implant placement into sites exhibiting pathology after clinical procedures to perform the decontamination of the implant's site and provide treatment recommendations. The following questions were raised and will be discussed: Does the presence of periodontal or endodontic infection compromise immediate implant placement success? What is advised to combat the socket infection prior to immediate placement?

Data Source and Search Strategies

This systematic review was made following to the PRISMA statement[24] suggestions. An electronic search without date or language restrictions was undertaken in March 2013 in the PubMed website (U.S. National Library of Medicine, National Institutes of Health). The following terms were used in the search strategy:

{Subject AND Adjective}
{*Subject*: (Immediate implant [text words])
AND
Adjective: (infected sites OR infected socket OR periapical lesion OR periodontitis OR periodontal lesion OR endodontic lesion OR pathology [text words])}

The publication had to be included in the electronic database to be considered in the review. All reference lists of the selected studies were then hand-searched for additional papers that might meet the eligibility criteria

for inclusion in this study. The titles and abstracts (when available) from these results were read by both authors for identifying studies meeting the eligibility criteria. For studies appearing to meet the inclusion criteria, or for which there were insufficient data in the title and abstract to make a clear decision, the full report was obtained and assessed. Contact with authors for possible missing data was not performed. Disagreement regarding the inclusion or exclusion of the retrieved articles was resolved by a discussion between reviewers.

Inclusion Criteria

Eligibility criteria included both animal and human studies. The publication's surgical intervention method had to have been implant placement into a site classified as having clinical and/or radiological signs of an infection, being periapical/endodontic (formation of a periapical abscess, pulpal necrosis, presence or not of an intraoral opening of a sinus tract, periapical radiolucency), perioendodontic (presence of acute inflammation of the periodontal ligament, pulpal necrosis, isolated deep pockets, and circumradicular/interradicular radiolucency, indicating an osseous defect along the periodontal ligament from apical to coronal), and/or periodontal (clinical signs include acute/chronic inflammation of the gingiva, periodontal attachment structures, and alveolar bone, periodontal pockets, periodontal abscess may or may not be present, loss of both the attachment of the periodontal ligament and bony support, decreased vertical height of the bone surrounding the affected teeth. Radiographic features include the presence of supragingival and subgingival calculus and loss of alveolar bone surrounding the teeth). The sites could not have been left to heal after teeth extraction and wound closure before implant placement. The implant(s) could not have placed after receiving active periodontal treatment, even though being placed immediately after teeth extraction.

Exclusion Criteria

Simple case report articles were not included. Review articles without original data were excluded, although references to potentially pertinent articles were noted for further follow-up.

Outcomes and Variables

For each of the identified articles included in this study, the following data were then obtained using a standard form (when available): year of publication, study design, number of patients, patients' age range and/or mean age, months of follow-up, type of infection, treatment success, number of placed implants, number of failed implants, percentage of success, mean marginal bone loss, type of implant used, region, and prosthetic conditions.

RESULTS

The study selection process is summarized in Figure 1. The search strategy resulted in 706 papers. The three reviewers independently screened the abstracts for those articles related to the focus questions. The initial screening of titles and abstracts resulted in 80 full-text papers; 48 were cited in more than one research of terms. Thus, 32 studies were identified without repetition. Of the 32 studies found, six were excluded for being case report articles[2,25–29] and three others for being review articles. Additional hand-searching of the reference lists of selected studies yielded five additional papers.[7,30–33] Table 1 summarizes the human and animal studies found. Twenty-one human studies case series were identified[7–9,30,32–48] along with seven animal model studies.[31,49–54]

Human Series

Twenty-one human series studies concerning immediate implant placement in infected sites were

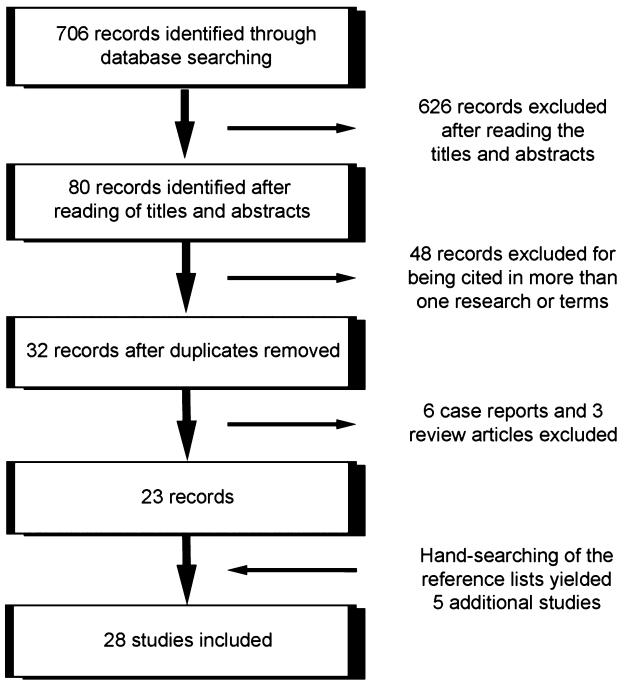

Figure 1 Study screening process.

TABLE 1 Immediate Placement of Implants into Infected Sites – Clinical Series and Animal Studies

Authors	Published	Study Design	Patients (*n*)	Patients' Age Range (Mean) (Years)	Follow-Up (Months)	Type of Infection	Treatment	Placed Implants (*n*)	Failed Implants (*n*)	Implant Success (%)	Mean Marginal Bone Loss (mm) (Range or SD)	Implant Type	Region Prosthetic Conditions
Pecora et al.[30]	1996	RA	7 (31)*	22–61 (41)	16.3*	Combined endodontic-periodontal involvement	Socket degranulation, placement with or without nonresorbable polytetrafluoroethylene (PTFE) membranes (GBR), postsurgical antibiotics for 7 days, postsurgical chlorhexidine for 8 weeks	7 (32)*	1 (1)*	85.7 (96.9)*	NM	Screw and cylinde-type plasma sprayed, Mini-Matic® (Minimatic Implant Technology, Inc., Boca Raton, FL, USA)	Maxilla, mandible Solitary implants
Novaes et al.,[31†]	1998	CCT	4	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Induced periradicular lesion versus healthy sockets	Debridement, rinse with tetracycline solution, 8 days of antibiotic coverage (beginning 1 day before the surgery)	28	0	100.0	NM	IMZ® (Dentsply, York, PA, USA)	Bilateral third and fourth mandibular premolars
Tripodakis[32]	2001	PS-NCG	13	NM	12–30	External root resorption, root fracture, periodontal or periapical lesion	10 days of antibiotic coverage, socket debridement, curettage, GBR in three cases, postsurgical chlorhexidine for 10 days	13	0	100.0	NM	Brånemark® (Nobel Biocare, Gothenburg, Sweden) Mk IV, Nobel Biocare®	Maxilla, frontal teeth, and premolars
Tripodakis[33]	2002	PS-NCG	6	NM	12–36	Periodontal lesion, periapical lesion with root fracture	10 days of antibiotic coverage, socket debridement, curettage, GBR in two cases, postsurgical chlorhexidine for 10 days	18	1	94.4	NM	Brånemark Mk IV, Nobel Biocare	Maxilla, incisors
Marcaccini et al.,[49†]	2003	CCT	5	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Ligature-induced periodontitis, bone loss, and furcation involvement	8 days of antibiotic coverage, socket debridement, curettage, antibiotic irrigation, fluorescein angiography	40 (20 NIS, 20 IS)[§]	NM	NM	NM	Frialit®-2 (Dentsply)	Mandibular first, second, third, and fourth premolar sites, using the contralateral teeth as controls
Novaes et al.,[50†‡]	2003	CCT	5	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Ligature-induced periodontitis, bone loss, and furcation involvement	8 days of antibiotic coverage, socket debridement, curettage, antibiotic irrigation, histomorphometric analysis	40 (20 NIS, 20 IS)[§]	5 (NIS) 3 (IS)[§]	75.0 (NIS) 85.0 (IS)[§]	NM	Frialit-2 (Dentsply)	Mandibular first, second, third, and fourth premolar sites, using the contralateral teeth as controls
Tehemar et al.,[51†]	2003	CCT	10 (5 test group, 5 control group)	Dogs were used as animal model	Dogs killed 6 months after placing the implants	Periodontal disease (gingival inflammation, accumulated plaque and calculus, pocket depths >6 mm, tooth mobility, alveolar bone loss of 30 to 50%)	Socket debridement by curettage, ampicillin IV before and after the surgery. Four groups: (1) implant alone; (2) implant + membrane; (3) implant + graft; (4) implant + graft + membrane	78 (80) two sites were not suitable for immediate implant insertion owing to severe inflammation of the gingiva and severe alveolar bone resorption	0	100.0	NM	Steri-Oss® (Nobel Biocare) hydroxyapatite (HA)-coated threaded root-form implants, Noble Biocare	Two premolars in each quadrant of the mandible and the maxilla (eight sites per dog)
Novaes et al.[52]	2004	RCT	6	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Ligature-induced periodontitis, bone loss, and furcation involvement	8 days of antibiotic coverage, socket debridement, curettage, histomorphometric analysis	36	0	100.0	NM	Frialit-2 (Dentsply)	Mandibular premolars

Papalexiou et al.,[53¶]	2004	RCT	6	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Ligature-induced periodontitis, bone loss, and furcation involvement	8 days of antibiotic coverage, socket debridement, curettage, fluorescein angiography	36	0	100.0	NM	Frialit-2 (Dentsply)	Mandibular premolars
Villa and Rangert[7]	2005	PS-NCG	20	49–70 (NM)	>12 (15–44)	Residual teeth in the interforaminal area with clinical and radiographic evidence of advanced endodontic and periodontal lesions judged to be no longer recoverable and unable to support a fixed prosthesis	Amoxicillin from 1 day before surgery up to 5 days after surgery. Socket debridement, bone curettage, antibiotic irrigation, GBR in cases of voids to the implant larger than 1 mm, postsurgical chlorhexidine for 2 to 3 weeks	97	0	100.0	0.74 ± 1.19	Nobel Biocare	4 to 6 implants mandible. Partially or total bridge fixed prostheses. Full-arch immediate loading.
Lindeboom et al.,[9†]	2006	RCT	50	19–69 (39.7)	12	Radiographic signs of chronic periapical periodontitis	600 mg clindamycin 1 hour before surgery, socket degranulation, GBR, postsurgical chlorhexidine for 7 days	50 (25 IP, 25 DP)**	2 (IP) 0 (DP)**	92.0 (IP) 100.0 (DP)**	Mesial 0.49 ± 0.11 (IP) 0.52 ± 0.16 (DP) Distal 0.53 ± 0.12 (IP) 0.52 ± 0.14 (DP)**	Frialit-2 Synchro (Dentsply)	Maxilla (anterior and premolar locations). Solitary implants. Nonloaded healing period of 6 months
Rabel and Köhler[34]	2006	PS-NCG	59	20–81 (55)	12	Chronic adult periodontitis	GBR in 33% of cases	95	4	95.8	NM	Ankylos®/ Friadent® (Dentsply) and Camlog® (Camlog Biotechnologies AG, Basel, Switzerland) Root-Line	Maxilla, mandible
Casap et al.[8]	2007	PS-NCG	20	26–67 (43)	12–72	Subacute periodontal, chronic periapical, chronic perio-endodontic, chronic periodontal, periapical cyst	Antibiotics from 4 days before surgery up to 10 days after surgery, socket debridement, peripheral intrasocket ostectomy, GBR, primary closure	30	1	96.7	NM	NM	Maxilla, mandible Solitary and splinted implants
Siegenthaler et al.,[35†]	2007	CCT	29 (34)††	23–82 (49.9)	12	Periapical pathology with pain, radiolucency > 1 mm, fistula, suppuration, or a combination of these findings	Antibiotics 1 hour before surgery, chlorhexidine rinse, socket debridement, GBR, antibiotics 5 days postsurgery, postsurgical chlorhexidine for 2 weeks	29 (13 test, 16 control sites with no presence of infection)	0	100.0	Mesial 1.9 ± 1.4 (test) 1.8 ± 1.1 (control) Distal 1.7 ± 1.4 (test) 1.6 ± 1.1 (control)	Straumann® (Straumann, Basel, Switzerland)	Maxilla, mandible Solitary implants
Villa and Rangert[36]	2007	PS-NCG	33	NM (NM)	12	Advanced endodontic and periodontal lesions or root fracture judged to be no longer recoverable and unable to support a fixed prosthesis	Amoxicillin from 1 day before surgery up to 5 days after surgery. Socket debridement, bone curettage, antibiotic irrigation, GBR in cases of voids to the implant larger than 1 mm. After suture, cortisone injection into soft tissue, postsurgical chlorhexidine for 2 to 3 weeks	76	2	97.4	0.74 (flapless) 1.02 (flap)	Brånemark Mk III, Mk IV, NobelSpeedy®, (Nobel Biocare) TiUnite® (Nobel Biocare), Nobel Biocare	Maxilla. Single, partial, or complete prostheses. Definitive prostheses were inserted after 6 to 12 months
Chang et al.,[54†]	2009	CCT	4	Dogs were used as animal model	Dogs killed 12 weeks after placing the implants	Induced periradicular infection versus healthy sockets	Osteotomy to simulate periradicular surgery, curettage, placement with or without nonresorbable PTFE membranes (GBR), antibiotic coverage for 5 days	18	0	100.0	NM	Straumann	Root of lower second premolar of each dog (control group). Roots of bilateral lower third and fourth premolars (experimental groups). Absence of occlusal loading

TABLE 1 *Continued*

Authors	Published	Study Design	Patients (*n*)	Patients' Age Range (Mean) (Years)	Follow-Up (Months)	Type of Infection	Treatment	Placed Implants (*n*)	Failed Implants (*n*)	Implant Success (%)	Mean Marginal Bone Loss (mm) (Range or SD)	Implant Type	Region Prosthetic Conditions
Del Fabbro et al.[37]	2009	PS-NCG	30	31–75 (55.8)	18.5 (10–24)	Chronic periapical lesion of endodontic or endoperiodontal origin	Socket degranulation, plasma rich in growth factors coating of implant, surgical reentry procedure was performed after 3 to 4 months of healing	61	1	98.4	0.41 ± 0.22 (1 year)	BTI Biotechnology Institute® (BTI Biotechnology Institute, Vitoria-Gasteiz, Spain)	Maxilla, mandible 14 partial prostheses and 26 single tooth restorations
Crespi et al.,[38†]	2010	CCT	37	32–71 (52.5)	48	Endodontic, periodontal, root fracture	Amoxicillin from 1 hour before surgery up to 7 days after surgery, debridement, saline rinse, postsurgical chlorhexidine for 15 days	275 (78-NIS, 197-IS)[§]	0 (NIS)/2 (IS)[§]	100.0 (NIS)/ 98.9 (IS)[§]	0.78 ± 0.38 (NIS) 0.79 ± 0.38 (IS)[§]	Titanium Plasma Spray® Sweden & Martina, Due Carrare, Italy), Sweden & Martina® (Sweden & Martina)	Maxilla, mandible Single, partial, or complete prostheses Immediate temporary prosthetic restorations
Crespi et al.,[39†]	2010	CCT	30	34–71 (51.2)	24	Periapical pathology and periapical radiolucencies and no signs of pain, fistulas, or suppuration.	Amoxicillin from 1 hour before surgery up to 7 days after surgery, debridement, saline rinse, postsurgical chlorhexidine for 15 days	30 (15 TG/15 CG)[‡‡]	0	100.0	0.82 ± 0.52 (CG) 0.86 ± 0.54 (TG)	Seven® (Sweden & Martina), Sweden & Martina	Maxilla, mandible, incisors, canines, premolars Solitary implants
Bell et al.,[40†]	2011	RA	256 (655)[§§]	NM (58.4)[§§]	3–93 (19.75)	Chronic periapical pathology	Preoperative chlorhexidine rinse, preoperative intravenous antibiotics, socket debridement, saline rinse, platelet-rich plasma + bone graft	285 (922)[§§]	7 (15)[§§]	97.5 (98.4)[§§]	NM	Straumann SLA	Implants loaded after a 3-month healing period
Kusek[41]	2011	PS-NCG	10	43–61 (50.6)	>12	Chronic infection or inflammation in the treatment area, root fractures, failed apicoectomy, incomplete root canal fill, internal resorption	Antibiotic and corticoid IV right before the surgery, laser treatment of the socket after the extraction, GBR	10	0	100.0	NM	NM	Maxilla, mandible Solitary implants
Truninger et al.,[42†,¶¶]	2011	CCT	29 (34)[¶¶]	23–82 (49.9)	36	Periapical pathology with pain, radiolucency > 1 mm, fistula, suppuration, or a combination of these findings	Antibiotics 1 hour before surgery, chlorhexidine rinse, socket debridement, GBR, antibiotics 5 days postsurgery, postsurgical chlorhexidine for 2 weeks	29 (13 test, 16 control sites with no presence of infection)	0	100.0	Mesial 1.54 ± 0.88 (test) 1.57 ± 0.57 (control) Distal 1.69 ± 0.92 (test) 1.59 ± 0.8 (control)	Straumann	Maxilla, mandible Solitary implants
Fugazzotto[43]	2012	RA	432	18–73 (NM)	24–204 (mean 67.3)	Periapical pathology	Amoxicillin 500 mg 3×/day for 10 days. Etodolac 400 mg 3×/day for 5 days. Curettage of periapical lesions, debridement of soft tissues, placement of autologous bone of particulate materials and covering membranes when needed	418***	5	98.8	NM	Titanium plasma-sprayed surfaces, sandblasted/ acid-etched surfaces, or SLActive surfaces	Maxilla, mandible Solitary implants restored 3 to 7 months postinsertion with abutments and individual crowns
Fugazzotto[44]	2012	RA	64	21–71 (46)	≤117 (mean 64)	Periapical pathology	Amoxicillin 500 mg 3×/day for 10 days. Etodolac 400 mg 3×/day for 5 days. Curettage of periapical lesions, debridement of soft tissues, placement of autologous bone of particulate materials and covering membranes when needed.	64 (128)[†††]	3 (4)[‡‡‡]	95.3[§§§]	NM	NM	Maxillary anterior region Solitary implants restored 3 to 7 months postinsertion with abutments and individual crowns

Jofre et al.[45]	2012	PS-NCG	31	19–84 (48)	6–297 (mean 15)	Acute or chronic endodontic or periodontal disease, chronic apical lesions	3 days before surgery: dental prophylaxis, drainage of abscess and irrigation with chlorhexidine 0.12%, and antibiotics + chlorhexidine 0.12% rinse twice a day. At the surgery: curettage/ degranulation of sockets, irrigation with chlorhexidine 0.12%. Postoperative: Antibiotics and chlorhexidine for 7 days	At least 31	0	100.0	NM	NM	Maxilla, mandible Solitary implants + provisional restoration
Jung et al.[46]	2012	CCT	27 (34)¶¶¶	23–82 (49.9)	60	Periapical pathology with pain, radiolucency > 1 mm, fistula, suppuration, or a combination of these findings	Antibiotics 1 hour before surgery, chlorhexidine rinse, socket debridement, GBR, antibiotics 5 days postsurgery, postsurgical chlorhexidine for 2 weeks	27 (12 test, 15 control sites with no presence of infection)	0	100.0	Mesial 1.5 ± 0.8 (test) 1.4 ± 0.5 (control) Distal 1.7 ± 0.7 (test) 1.5 ± 0.6 (control)	Straumann	Maxilla, mandible Solitary implants
Meltzer[47]	2012	RA	63	NM (NM)	3–24	Active periodontal or endodontic lesions	Amoxicillin 500 mg starting 48 hours before surgery and continued for 8 days, curettage of sockets, irrigation with chlorhexidine 0.12% followed by saline, postsurgical rinse twice a day with chlorhexidine 0.12%	77	1	98.7	NM	NanoTite, Biomet 3i® (Biomet 3i, Warsaw, IN, USA)	Maxilla, mandible Solitary implants and 2-implant splinted constructions, immediate provisional restoration. Definitive restoration after 3 to 4 months
Marconcini et al.[48]	2013	PS-NCG	13	24–65 (NM)	12	Tooth fracture and presence of an acute infection, acute endodontic failure, or chronic endodontic failure	Amoxicillin 2 g, 1 hour before surgery, thereafter, 1 g twice daily for 5 days, socket curettage, postsurgical chlorhexidine	20	0	100.0	0.5	NM	Maxilla, mandible Solitary implants. Definitive prostheses were inserted after 4 months

*Thirty-two teeth in 31 patients were included in the study with the following diagnoses: 13 teeth with vertical root fractures, eight teeth with horizontal root fractures, four teeth with root perforations, and seven teeth with combined endodontic-periodontal involvement. Only one implant from the 32 failed to integrate, and this implant was placed in a fresh extraction socket of a tooth with combined endodontic-periodontal involvement. Thus, the implant success was 85.7 for this group of seven implants with endodontic–periodontal involvement, and 96.9 for the whole group of 32 implants. The follow-up period was considered only after prosthodontic restoration (after 4–6 months of osseointegration).

†Placement of implants in infected and noninfected sites.

‡The same study group published another article[49] analyzing the same five dogs used by this article. However, instead of doing histomorphometric analyses, fluorescein angiography was performed.

§IS, periodontally infected sites; NIS, noninfected sites.

¶The same study group published another article[52] analyzing the same six dogs used by this article. However, instead of doing fluorescein angiography, two histomorphometric analyses were performed: percentage of bone/implant contact and analyses of the bone density in adjacent and distant areas from the implant surface.

**IP, immediately placed into infected sites, DP, delayed placement after 3 months.

††Five patients (four test and one control) had to be withdrawn from the study because of the inability to obtain primary implant stability owing to unfavorable bone morphology (early exit).

‡‡The control group (CG) included 15 patients without periapical lesions but with root caries and root fractures. The test group (TG) included 15 patients with periapical lesions, periapical radiolucencies, and no signs of pain, fistulas, or suppuration.

§§In total, 922 implants were placed in 655 patients. A total of 477 patients had 637 implants placed into extraction sites that were not affected by periapical radiolucencies. In 256 patients, a total of 285 implants were placed into extraction sites with periapical pathology. Eight implants failed from sites not affected by periapical radiolucencies, and seven failed from sites affected by periapical radiolucencies. The mean age was 58.4 years in the study group and 60.1 years in the control group.

¶¶All the patients from the study of Truninger et al.[42] were part of a former study evaluating the early events and the 1-year follow-up.[35]

***The author tried to place 475 implants in 432 patients, but only 418 implants were effectively placed. Here the number of implants is smaller than the number of the patients for a reason. In 57 instances, no implant could be placed following tooth extraction and defect debridement due to inadequate bone present following tooth extraction and defect debridement for ideal positioning of an implant of the desired dimensions as the result of an inability to achieve primary implant stability.

†††Sixty-four implants were immediately placed at the time of extraction of a tooth that demonstrated radiographic evidence of a periapical lesion; other 64 implants were placed in a "pristine" site.

‡‡‡Of the four implants lost, three were immediately placed in infected sites.

§§§Considering only the implants placed in the infected sites.

¶¶¶All the patients from the study of Jung and colleagues[46] were part of two former studies evaluating the early events at the 1-year[35] and 3-year follow-up.[42] But now, only 27 of the 29 attending the 1-year visit could be recruited for the 5-year follow-up visit (12 in the test group and 15 in the control group).

CCT, controlled clinical trial; GBR, guided bone regeneration; NM, not mentioned; PS-NCG, prospective study with no control group; RA, retrospective analysis; RCT, randomized controlled trial.

published.[7–9,30,32–48] Detailed data of these studies are presented in Table 1; the important points are presented below.

Pecora and colleagues[30] published the first human case series in 1996. In their study, 32 titanium alloy implants were inserted immediately after the extraction of teeth that were diagnosed during endodontic surgery as having root fractures, perforations, or endodontic–periodontal complications. Only one implant out of the 32 failed to integrate, and this implant was placed in a fresh extraction socket of a tooth with combined endodontic–periodontal infection.

Tripodakis[32] placed 13 immediate implants into fresh extraction sockets of upper frontal teeth and premolars with external root resorption, root fracture, or periodontal or periapical lesion. All implants osseointegrated. In a study published 1 year later, the same author[33] placed 18 immediate implants in fresh extraction sockets of upper incisors with periapical or periodontal lesions; only one implant was lost.

Twenty patients in need of mandibular implant treatment and with teeth showing signs of infection in the interforaminal area were included in the study of Villa and Rangert.[7] The patients received four to six implants in or close to the fresh extraction sockets and received a provisional prosthesis within 3 days. The implant survival rate was 100% ($n = 97$), and a mean marginal bone loss of 0.7 mm (SD 1.2 mm) was registered (the patients were followed for a minimum of 1 year; range 15 to 44 months).

In a prospective human study conducted by Lindeboom and colleagues,[9] 25 implants were immediately placed after extraction of teeth with radiographic signs of chronic periapical periodontitis, and 25 implants were placed after a 3-month healing period. Only two implants of the immediate group were lost. However, mean Implant Stability Quotient, gingival aesthetics and radiographic bone resorption, and periapical cultures were not significantly different between the two groups.

Rabel and Köhler[34] investigated the prevalence of periodontal marker organisms and specific interleukin-1 (IL-1) gene polymorphisms (which show a close association with periodontitis) and their effect on the success of immediate implant placement postextraction in the patient with periodontal disease. A group of 59 patients with chronic adult periodontitis was treated with a total of 95 immediate dental implants placed into extraction sites. After 1 year, four failures were observed; all of these patients were smokers. No association was observed between failures and the IL-1 gene polymorphisms or pathogens, showing that periodontally infected sites do not seem to be a contraindication for immediate implantation.

In the study of Casap and colleagues,[8] 30 implants were immediately placed into debrided infected sites in 20 patients. Only one implant failed to osseointegrate (follow-up of 12 to 72 months). In the prospective and controlled clinical trial conducted by Siegenthaler and colleagues,[35] immediate implant placement in sites with or without periapical pathology did not lead to an increased rate of complications, more interproximal bone loss, or worse clinical parameters.

In the study of Villa and Rangert,[36] 33 patients with advanced endodontic and periodontal lesions or root fracture judged to be no longer recoverable and unable to support a fixed prosthesis were included in the study. Seventy-six implants were placed directly in extraction sockets of infected teeth and only two were lost. A total of 30 partially edentulous patients with teeth requiring extraction and chronic periapical lesions were included in the study of Del Fabbro and colleagues,[37] and 61 implants were installed immediately after extraction and debridement combined with plasma rich in growth factors (PRGF) placement into the socket. Only one implant was lost. The authors suggested that the use of PRGF in association with immediate implant placement could be a viable therapeutic option for the rehabilitation of postextraction sockets.

Crespi and colleagues[38] compared the outcomes of immediate loading of implants in replacing teeth with and without chronic periodontal lesions. From a total of 275 implants placed and immediately loaded in extraction sockets, 197 were placed in periodontally infected sites and 78 in noninfected sites. Two implants of the infected sites were lost. However, implants that were placed in periodontally infected sockets showed no significant differences compared with implants placed in uninfected sites (48 months of follow-up). Thirty patients requiring a single-tooth extraction of a monoradicular or premolar tooth were included in the study of the same research group, published in the same year.[39] Half of the patients were considered the control group (without periapical lesions) and the other half the test group (with periapical lesions). A survival rate of 100% was reported for all implants at the 24-month follow-up.

Bell and colleagues[40] immediately placed 285 implants into sockets that had chronic periapical infections (with seven failures) and 637 implants into extraction sites that were not affected by periapical radiolucencies (with eight failures). The difference between the control group and the group with periapical radiolucencies was not statistically significant. Kusek[41] immediately placed 10 implants into sockets after extraction of teeth having root fractures, failed apicoectomy, incomplete root canal fill, and internal resorption. The results showed a noticeable reduction of bacteria in the extraction sites after the use, just before the implant placement, of an erbium laser to reduce the bacteria in the sockets. No implants were lost.

All the patients from a more recent study[42] with a follow-up of 3 years were part of a previously mentioned study[35] evaluating the early events and the 1-year follow-up. The clinical and radiological parameters showed no statistically significant difference between the test and the control group at 3 years. Implant survival was 100% at 3 years, and between the 1- and 3-year visits, the bone-implant contact increased in both groups significantly on one side of the implant. Moreover, in both groups, no periapical radiolucencies were found, which was also observed by two other studies.[8,35]

Fugazzotto[43] retrospectively evaluated 418 immediately placed implants in sites with teeth presenting periapical pathology. After a mean follow-up period of 67.3 months, only five implants were lost. The author concluded that implants placed with this procedure have comparable survival rates than to those implants placed immediately into sites without periapical pathology.

Fugazzotto[44] assessed implant survival rates when implants are placed in sites with periapical pathology and sites without periapical pathology in the same patient. The author observed that both treatments yielded comparable results, and that difference in survival rates was not statistically significant.

Jofre and colleagues[45] reported a series of 31 cases treated according to the protocol of antisepsis after extraction of infected teeth, and immediate implant placement and provisionalization. No implant was lost after a mean follow-up of 15 months. The authors stated that implants can be immediately placed in infected sites with high rates of success when following a protocol that includes antibiotic therapy, debridement, antisepsis of the compromised tissue, and high primary implant stability.

Jung and colleagues[46] compared the outcome of immediately placed implants in sockets with or without periapical pathology 5 years after placement. All the patients from this study were part of two former studies evaluating the early events at the 1-year[35] and 3-year follow-up.[42] But now, only 27 of the 29 attending the 1-year visit could be recruited for the 5-year follow-up visit (12 in the test group and 15 in the control group). They concluded that this technique can be a successful treatment modality with no disadvantages in clinical, aesthetical, and radiological parameters to immediately placed implants into healthy sockets.

Meltzer[47] evaluated the primary stability and the reverse torque testing at 3 to 4 months postoperatively of 77 implants placed in fresh extraction sockets with active periodontal or endodontic lesions. Only one implant did not osseointegrate. The authors concluded that this is a successful technique if adequate care is taken, that is, the socket is thoroughly debrided in conjunction with an oral antibiotic regimen, and the immediate implant is nonocclusally loaded.

Marconcini and colleagues[48] evaluated the clinical success of implants placed in fresh extraction sockets that showed clinical signs of periodontal disease. All the implants were osseointegrated, and at the end of the 12-month follow-up period, patients were asymptomatic and showed no signs of infection or bleeding when probed. The authors stated that this may be a valid operative technique that leads to predictable results if adequate preoperative and postoperative care is taken.

Animal Studies

Seven experimental studies in animals have corroborated the clinical experience in humans and have shown that socket debridement and prophylactic antibiotics create adequate conditions for the bone remodeling process around immediate implants placed into infected sites.[31,49–54] Detailed data of these studies are presented in Table 1; the important points are presented below.

Some of these seven animal studies were conducted in the same animals (i.e., the same study). Marcaccini and colleagues[49] and Novaes and colleagues[50] analyzed the same samples from the same dogs. However, one article published a histomorphometric study of BIC,[50] and the other one a fluorescence microscopy study.[49] This is also true for two other studies.[52,53] Novaes and colleagues[52] performed two histomorphometric analyses (percentage of BIC and analyses of the bone density

in adjacent and distant areas from the implant surface), and Papalexiou and colleagues,[53] fluorescein angiography.

In the first animal study published,[31] implants were immediately placed in fresh sockets with periapical infections. The crowns of the teeth were cut with burs at the cementoenamel junction and removed, exposing the roots and root canals. The pulpal tissue was removed, and the roots were gently instrumented with endodontic files without care to avoid contamination of the canals. Radiographs were taken every 3 months to evaluate the size of the developing periapical lesions, and only after 9 months were the lesions large enough to proceed to the placement of implants. Twelve weeks later, all implants were successfully osseointegrated, and no signs of inflammation or exudation were observed during the healing period. Histomorphometric analysis revealed no significant difference in the percentage of BIC at the periapically infected sites (28.6%) compared with healthy sites (38.7%).

Novaes and colleagues[50] evaluated the percentage of BIC of immediate implants placed in periodontally infected sites in five dogs. Periodontitis was induced using nonresorbable silk suture placed and left in place for 3 months into infrabony pockets of approximately 1 mm in depth, which were created around each premolar after dissection of the marginal periodontium. After repositioning of the periodontal flaps, the wound was closed with resorbable sutures. After the 3 months, the implants were placed, and after more 3 months, the dogs were euthanized. Histologic observations showed that the BIC had mineralized bone matrix in intimate contact with the implant surface. Histomorphometric analysis revealed no significant difference in mean percentage of direct BIC around the middle third of the experimental/infected sites implants ($66.0 \pm 19.6\%$) compared with control/noninfected sites implants ($62.4 \pm 19.6\%$).

Marcaccini and colleagues[49] observed a short delay in the first stages of immediate implant healing in periodontally infected sites, but final osseointegration was not affected as verified by fluorescence analysis used to determine the rate and extension of bone formation.

In the study of Tehemar and colleagues,[51] four treatment modalities associated with the immediate placement of implants into extraction sockets of healthy and periodontally diseased teeth were tested in dogs; the implants were either inserted alone, surrounded only by a membrane, surrounded only by grafted material, or by a combination of grafted material and membrane. In the group with the membrane without graft material, epithelial migration downward along the neck of the implants was prevented, and the space was maintained under the membranes. There was no noted improvement of the BIC in control and test 3-month implants in the maxilla and mandible. However, the 6-month implants showed a significant improvement in the BIC under the membrane, suggesting that the fact of leaving the membrane for a longer time may improve bone regeneration. Moreover, the graft material significantly enhanced the BIC in control and test implants at 3 months, and the enhancement was maintained at 6 months.

Papalexiou and colleagues[53] evaluated, by confocal laser scanning microscope, the quantity and the chronological influence of two surface treatments, titanium plasma spray and grit blasted/acid etched, of implants placed in periodontally diseased sites on the remodeling activity of newly formed bone in six dogs. Periodontitis was induced in the same manner as Novaes and colleagues.[50] Their results showed that there were no significant statistical differences in bone formation between groups from 3 days to 12 weeks. However, covariance analysis showed that the percentage of marked bone was statistically greater for the grit-blasted/acid-etched surface group when compared with the titanium plasma spray group between the 3-day and 8-week periods of evaluation. The same study group published another article[52] analyzing the same samples used by this article.[53] The grit-blasted/acid-etched surface, although not statistically significant, had a slightly better performance when compared with the titanium plasma spray surface for all the parameters studied.[52]

Chang and colleagues[54] compared in dogs the osseointegration of immediate implants in infection-free sites (control group) and in sites with periradicular lesions and osseous defects made by periradicular surgery (implant placement with [experimental group 1] or without [experimental group 2] nonresorbable polytetrafluoroethylene [PTFE] membranes). Periradicular lesions were induced by the following method: The coronal portions of the teeth were removed with burs at the level of the cementoenamel junctions to expose the pulpal cavities under local anesthesia. A periradicular infection was induced by placing dental plaque harvested from the adjacent teeth into the pulpal

cavities. The pulpal cavities were then sealed with zinc oxide and eugenol. After 3 months, complete debridement was performed and the teeth extracted. The control group showed a significantly higher BIC (76%; $p < .05$) than experimental groups 1 (59%; $p = .0280$) and 2 (48%; $p = .0044$). There was no significant difference in the BIC between experimental groups 1 and 2. However, the implants in both control and experimental groups were clinically not mobile and showed no sign of infection at the time of euthanization. The radiographs taken 12 weeks after the placement of implants showed complete resolution of periradicular lesions in both control and experimental groups.

Treatment Protocol

Considering only the human case series, the treatment protocol of most studies enclosed in this review included socket debridement, curettage, the use of systemic antibiotics, and postsurgical chlorhexidine rinses varying from 1 to 8 weeks. Many performed GBR procedures.[7–9,30,32–36,41–44,46] Some studies included peripheral intrasocket ostectomy,[8] PRGF coating of implant,[37] combination of bone, xenograft and platelet-rich plasma,[40] antibiotic solution irrigation of the socket,[7,36] socket irrigation with chlorhexidine 0.12%,[45,47] and the use of an erbium laser using photoacoustics to reduce the bacteria in osteotomy sites that were infected by apical pathology.[41]

DISCUSSION

Does the Presence of Periodontal or Endodontic Infection Compromise Immediate Implant Placement Success?

The disadvantage of the placement of implants into the sockets of teeth with periapical lesions is the potential for implant contamination during the initial healing period because of remnants of the infection.[12,16,18,55,56] *Bacteroides* species can inhabit tooth periapical lesions[57] while being encapsulated in a polysaccharide that promotes its virulence, survival, and importance in mixed infections. *Bacteroides forsythus* has been shown to persist in asymptomatic periradicular endodontic lesions and may survive in bone in an encapsulated form after extraction and subsequently infect an implant.[56] Ayangco and Sheridan[16] reported three patients who had a history of failed endodontic and apicetomy procedures, which finally led to extraction of the involved teeth and subsequent placement of implants after sufficient healing time. It would appear that even after thorough and vigorous debridement and irrigation of the extraction sockets and the passing of sufficient healing time, bacteria (or cyst/granuloma) had remained in the bone, which led to the initiation of retrograde peri-implantitis. Brisman and colleagues[55] reported that even asymptomatic endodontically treated teeth with a normal periapical radiographic appearance could be the cause of an implant failure. They suggested that microorganisms might persist, even though the endodontic treatment is considered radiographically successful, because of inadequate obturation of an incomplete seal. In a study to investigate whether extraradicular infection can persist in apparently healed alveolar bone, Nelson and Thomas[58] found that bacteria may persist in healed alveolar bone remodeled after teeth with apical pathosis have been removed by surgical debridement, which may be reactivated to an infection during clinical implant therapy. Kassolis and colleagues'[59] histopathologic findings provided evidence that the edentulous jaw can contain regions of bacterial biofilm formation and nonviable alveolar bone for 1 year or more following tooth extraction and mucosal healing. The authors suggested that such regions of subclinical infection and necrotic bone may represent a significant risk factor for early dental implant failure. A review made by Quirynen and colleagues[12] indicated that sites of neighboring teeth with an endodontic pathology or extraction sites from teeth with a history of failed endodontic and apicetomy procedures constitute a risk for successful implant insertion. The term *retrograde peri-implantitis* was introduced as radiolucencies around the most apical part of an osseointegrated implant; they might be provoked by the remaining scar or granulomatous tissue after immediate implant placement into extraction sockets.[18]

In contrast to these findings, studies have shown in animal experiments that implants placed in artificially induced periapical lesions osseointegrate as well as implants placed in healthy sites.[31,49,50,53] There are also several reports on the immediate placement of implants in humans after the extraction of endodontically compromised teeth. Two studies[9,35] showed that the immediate placement of a dental implant in an extraction socket with a periradicular infection does not have a higher rate of complication than one placed in an uninfected site. Del Fabbro and colleagues[37] evaluated in a prospective study the clinical outcome of implants immediately placed into fresh extraction sockets of teeth

affected by chronic periapical pathologic features using PRGF as an adjunct during the surgical procedure, showing a high success rate, preservation of hard and soft tissues, and general high patient satisfaction. In the study of Bell and colleagues[40] who evaluated the success of dental implants placed immediately into extraction sites in the presence of chronic periapical pathology, the only variable that significantly affected the outcome was the presence of periapical pathology in retained teeth adjacent to the implant being placed. Adjacent lucencies have previously been found to increase implant failure.[55] Thus, Bell and colleagues[40] suggested that endodontic treatment of teeth adjacent to implant sites, especially if those implant sites have teeth with lucencies, should be seriously considered.

Fugazzotto[44] conducted the only study comparing implants immediately placed into sites with periapical pathology with those immediately placed into sites without periapical pathology in the same patient, thus helping to control a number of interpatient variables and render the results more directly clinically applicable. It was observed that both treatments yielded comparable results with no statistically significant difference in survival rates.

The high success rate of fresh-socket implants placed in chronic and acute lesions may be explained by the behavior of endodontic infections because they are mixed infections dominated by anaerobic bacteria (*Fusobacterium*, *Prevotella*, *Porphyromonas*, *Actinomyces*, *Streptococcus*, and *Peptostreptococcus*) commonly restricted in the infected root canal.[60] Extraction of the involved tooth generally leads to eradication of the cultured microorganisms.[9]

No implant failures were observed in most of the animal models studies.[31,51–54] However, it is important to consider that in these studies, the dogs were euthanized 12 to 24 weeks after placing the implants. One study[50] reported eight failures (of a total of 40 implants). Another study[49] did not report if any implant did fail. Because these two studies[49,50] analyzed the same samples from the same dogs, the number of failed implants must be the same. Animal studies showed that the presence of periodontal or endodontic infections, even in active phase, did not compromise the osseointegration of immediately placed implants and did not reduce the BIC after the healing phase.

It was concluded from human studies[7,9,35,36,38,45,47] that for those implants with primary stability, the immediate placement into infected sites did not lead to an increased rate of complications and rendered an equally favorable type of tissue integration of the implants, if appropriate clinical procedures like antibiotic administration, meticulous cleaning, and alveolar debridement are performed before the surgical procedure. Extraction of the involved teeth with socket degranulation and an appropriate antibiotic prophylaxis leads to the eradication of the cultured microorganisms[7,9,39] and might reduce the inflammatory response and the bone-resorption process.[39] Moreover, immediate implant placement may be beneficial in maintaining the integrity of the extraction sockets and contribute to the maintenance of the interdental papillae around implant restorations.[61]

All studies here reviewed[7–9,30–54] demonstrated that immediate implant placement in infected extraction sockets can be successful, provided that thorough preoperative care is given (for details, see Table 1).

What Is Advised to Combat the Socket Infection Prior to Immediate Placement?

Regarding the treatment protocol, appropriate clinical procedures to perform the decontamination of the implant's site, such as antibiotic administration, meticulous cleaning, and alveolar debridement, combined with GBR with or without bone grafting,[9,31,35] is suggested to create adequate conditions for bone regeneration and osseointegration despite the previous contamination.[8]

The natural healing process after tooth extraction normally manages residual infection, but as an infection increases inflammatory activity, infection may result in increased bone resorption and a higher risk of implant stability loss and failure. The presence of granulation tissue in the socket of an infected tooth must be considered as an inflammatory response to bacteria. This reactive tissue protects bone from direct bacterial aggression and, if carefully removed, will reveal healthy bone. Therefore, infected tooth extraction and conventional granulation tissue removal, as well as an early onset of antibiotic treatment, may be effective in reducing the inflammatory response and the consequent bone resorption activity.[7,36] Dent and colleagues[62] reported a tendency to reduced implant failure when antibiotics were used preoperatively and in appropriate doses. However, a systematic review[63] suggested that the benefits of antibiotic prophylaxis in noninfected sites remain unclear and may not be needed. Because all

human studies reviewed here implemented systemic antibiotics, the success of this protocol and low rate of infections may be related to their use. However, until now, no study compared the immediate placement of implants in infected sites conducting a careful debridement with and without the use of systemic antibiotics. Thus, more research is needed concerning this issue.

Since all the four studies[7,31,36,49] that used antibiotic solution irrigation of the socket also implemented systemic antibiotics, it seems difficult to define a clear advantage of the method. Moreover, two of these studies were conducted in an animal model[31,49] and the two others in humans,[7,36] which make comparisons between them difficult. There were also no control groups in these studies. As the use of an erbium laser using photoacoustics to reduce the bacteria in osteotomy sites that were infected by apical pathology was applied only in one study with 10 patients without a control group,[41] more research is needed concerning this issue.

In addition, it is reported that local administration of glucocorticoid dexamethasone reduces bone resorption processes by preventing macrophage and osteoclast activation.[64] Therefore, local delivery of an anti-inflammatory drug at the implant site may reduce potential loss of implant stability during healing. However, no study has proved, with a control and a test group, the benefits of local delivery of anti-inflammatory drugs after the immediate insertion of implants in infected sites.

Regarding the chlorhexidine rinse, its use may be indicated as a preventive method as it may reduce microbial complications when used at least in the immediate perioperative period in implant surgery. Lambert and colleagues[65] examined the effect of perioperative chlorhexidine on the frequency of infectious complications through stage II. With 0.12% chlorhexidine rinse, there was a significant reduction in the number of infectious complications (4.1% vs 8.7%).

In the presence of periapical pathology, a decision has to be taken whether an immediate or a delayed implant placement strategy is to be preferred.[35] One problem in immediate implant placement in chronically infected sites may be an incongruity between the implant diameter and the morphology of the alveolus that is worsened by the presence of a bone defect because of the periapical infection. Some authors stated that a minimum of residual apical bone of 3 to 5 mm in a vertical dimension is required.[10,13] When respecting these recommendations, many sites do not qualify for immediate implant placement. Another critical aspect is the diameter of the periapical lesion. If it exceeds the diameter of the planned implant, then there may be a need to obtain implant stabilization more apically. Conversely, if the implant diameter is larger than the diameter of the periapical lesion, initial stability may be sufficient without extending 3 to 5 mm apically to the extraction socket.[35] In the study of Lindeboom and colleagues,[9] in the immediate placement group, larger diameter implants were used more frequently than in the delay-placed group (3 months after extraction). But, this is not completely a disadvantage when sufficient bone is available. Another study has shown that wide-diameter implants are associated with increased removal torque, and that the load on cortical bone decreases with increasing implant diameter.[66] Moreover, according to Truninger and colleagues,[42] the axis of an implant placed in the anterior area to match esthetic expectations differs from the axis of the tooth that is extracted. It can therefore be assumed that even though the diameter of the implant might be bigger than that of the periapical pathology, the buccal part of the former pathology would remain intact despite the drilling sequence. Thus, the main criteria for immediate placement of an implant into a socket with periapical pathology should be the achievement of primary implant stability after debridement and not the size of the pathology itself.

CONCLUSIONS

The findings from the studies reviewed here should be interpreted cautiously because of a great variability among the studies in terms of type of implant used, area of implant placement, type of infection present, criteria for patient selection, and loading protocol. However, the high survival rate and the normal marginal bone changes obtained in several studies support the hypothesis that implants may be successfully osseointegrated when placed immediately after extraction of teeth presenting endodontic and periodontal lesions, provided that appropriate clinical procedures are performed before the implant surgical procedure. These procedures include meticulous cleaning and alveolar debridement. Chlorhexidine 0.12% rinse may be indicated at least in the immediate perioperative period. The benefits of antibiotic solution irrigation of the socket and systemic antibiotic administration in such conditions have not yet been proven and remain unclear. So far, no study

compared the immediate placement of implants in infected sites conducting a careful debridement with and without the use of systemic antibiotics, but it is important to consider that most of these studies performed a short-term follow-up. Therefore, more randomized controlled clinical trials with a longer follow-up are required to confirm this procedure as a safe treatment. Moreover, the outcome measures were not related to the type of infection; the classification of infection was often vague and varied among the studies. Thus, a clear classification system needs to be implemented with clinical evaluation related to a more specific pathology. Animal studies showed that artificially induced periodontal or endodontic infections, even in active phase, did not compromise the osseointegration of immediately placed implants and did not reduce the BIC after the healing phase.

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