Retrograde Peri-implantitis: A Scoping Review of Current Understanding of a Rare Complication in Dental Implants

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Retrograde peri-implantitis is a rare complication of dental implants, characterized by symptomatic periapical lesions at the implant apex. Several etiological factors have been identified with an increased risk associated with implants placed near teeth with periapical lesions. The present study undertook a scoping review to discuss the etiological factors and recent classification of retrograde peri-implantitis and summarize current literature–based guidelines for its management. An online database search was performed to identify studies related to retrograde peri-implantitis using the following terms: retrograde peri-implantitis, implant apical lesions, and periapical implant lesion. A total of 167 studies were initially identified, of which 38 met the inclusion criteria. These comprised case reports and retrospective studies, including 14 case reports that presented various management strategies for retrograde peri-implantitis. Half of these reports describe adjacent teeth with failed root canal treatments or periapical infections. Four studies utilized nonsurgical treatment to manage the lesion, including antibiotics for root canal treatment of the adjacent teeth. The remaining cases involved surgical interventions, such as implant debridement, apical resection, implant removal, and bone grafting. This review discusses the etiological factors of retrograde peri-implantitis, presents the recent classification of the lesion, and summarizes current literature–based guidelines for its management.

Key Words: implant, retrograde peri-implantitis, regeneration, endodontically treated teeth, apical radiolucency

INTRODUCTION

Osseointegrated dental implants are the gold standard for replacing missing teeth in patients requiring partial or complete mouth rehabilitation. Longitudinal studies have reported a high survival rate of dental implants, making them predictable treatments.[1–3] In a meta-analysis by Pjetursson et al,[4] the survival rates of single implant-supported crowns and implant-supported fixed partial dentures were approximately 95%. However, the estimated survival decreases after the implants are functional for 10 years because of technical or biological complications.[4] The conventional protocol for implant placement includes 4–6 months of healing time before installation of the prosthesis. Immediate placement and loading have become feasible with the evolution of dental implant surgical techniques and microtextured implant surfaces.[5] Studies comparing immediate implant placement with conventional methods show that this protocol is safe and predictable with proper planning.[6,7] A retrospective analysis evaluating the success of immediate implants placed with and without bone grafts reveals high survival rates of 97% and 93%, respectively.[8]

The factors thought to affect the success of implants have been evaluated in several studies. These factors include primary implant stability, loading protocol, implant positioning, gingival biotype, and patient-related factors.[9,10] Biological complications around implants are common and are characterized by their inflammatory nature in response to bacterial plaque biofilm accumulation, which disturbs host-microbe hemostasis. Two types of peri-implant diseases have been identified: peri-implant mucositis and peri-implantitis. Whereas the inflammatory lesion in preimplant mucositis is confined to the peri-implant mucosa, it extends to the bone supporting the implant in peri-implantitis cases.[11,12]

Retrograde peri-implantitis (RPI) is a less common form of dental implant complication first reported by McAllister in 1992.[13] It is defined as a symptomatic radiographic radiolucency confined to the periapical area of the implant. The coronal portion of the implant demonstrates a normal implant–bone interface (Figure 1). The lesion can be detected immediately after implant placement from 2 to 8 weeks and up to 4 years with no significant difference between symptomatic and asymptomatic lesions. It has been documented that lesions associated with fistulas are diagnosed later than lesions without fistulas (33.1 weeks versus 18.9 weeks).[14] Before diagnosing an RPI lesion, a thorough evaluation and review of the case history are essential to rule out other implant periapical lesions that might be asymptomatic and require no intervention. Periapical radiolucency may appear on the radiograph when the implant is placed shorter than the drilled osteotomy site or as a result of aseptic bone necrosis caused by excessive heat during drilling.[15]

Clinically, active RPI presents with pain, tenderness, and swelling, and sometimes, a fistula may appear near the implant apex. The lesion may spread laterally and coronally; the implant may become mobile as it becomes extensive.[16] This scoping review discusses the prevalence of RPI and etiological

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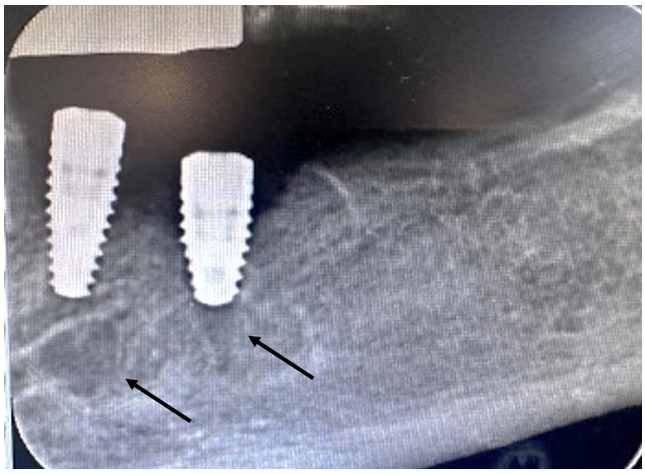

Figure 1. Radiographic representation of a retrograde peri-implantitis lesion affecting implants placed at sites #35 and #36. Additionally, both implants exhibit marginal bone loss with the middle third of the bone remaining intact. Note the proximity of the implant at #35 to the root surface of the adjacent endodontically treated tooth.

factors, presents a recent classification of the lesion, and summarizes current management guidelines from the literature.

Methodology

The following population, intervention, comparison, and outcome criteria were identified:

- Population: patients diagnosed with RPI
- Intervention:
  - Etiology focused: adjacent teeth with root canal treatment or periapical pathology
  - Treatment focused: surgical treatment
- Comparison:
  - Etiology focused: vital adjacent teeth with no periapical pathology
  - Treatment focused: nonsurgical treatment
- Outcome: resolution of the lesion clinically and radiographically and implant survival

Search strategy

A comprehensive literature search was performed in PubMed, Google Scholar, Web of Science, Midline, and Wiley online library databases in September 2024 to identify studies related to RPI. The search was performed using the following terms: retrograde peri-implantitis, implant apical lesions, and periapical implant lesions.

This scoping review included retrospective and prospective cohort studies, case-control studies, case series, and case reports published in English. Randomized controlled trials were not expected due to the condition's rarity but were included if identified. Exclusion criteria included animal studies, in vitro experiments, surveys assessing knowledge and attitude, and reviews without original patient data. All case reports and clinical trials aimed at determining the prevalence, etiology, and treating or salvaging implants diagnosed with RPI were included in the analysis. All treatment modalities were considered, including nonsurgical approaches, surgical debridement, and/or apicoectomy performed on the implant.

Results

A total of 167 studies were identified, and 38 studies were selected for this review. The selected studies comprised case reports, retrospective studies, and reviews that discussed the prevalence, classification, etiology, and management of this condition. Fourteen case reports were included in the management section. Those reports provided different approaches for treating RPI (4 studies used a nonsurgical approach, and the remaining 10 used different surgical treatments). The remaining 24 studies discussed the prevalence, etiologic factors, and classification of the lesion.

Discussion

Classification of the lesion

Numerous studies have attempted to classify RPI lesions based on various criteria. Sussman[17] proposes a classification system that includes 2 types based on the etiology of the lesion in relation to the adjacent teeth. Type 1, termed an implant-to-tooth lesion, occurs when implant placement leads to the devitalization of the adjoining tooth. In contrast, type 2, known as tooth-to-implant lesions, arises when a periapical lesion from an adjacent endodontically involved tooth contaminates the implant surface.[17] Additional causes of RPI are discussed in previous case reports. Smarst et al expands upon the previous classification by adding two additional classes: class 3, in which the implant is placed out of the alveolar bone housing by being angulated lingually or labially, and class 4, which includes implants placed in sites with residual infection.[18] Other studies describe lesions as infected or inactive. An infected lesion results from the placement of the implant in proximity to the infected tissue, whereas an inactive lesion represents apical scar tissue formation due to the placement of the implant that is shorter than the drilled osteotomy or overheating of the bone when drilling the osteotomy, resulting in aseptic bone necrosis.[15,19] In a recent systematic review, periapical lesions were classified based on their histopathological presentation rather than their etiology. In this classification, 5 distinct classes were proposed: class 1, implant periapical inflammation, which includes both implant nonsuppurated lesions and chronic granuloma; class 2, acute suppurated, chronic suppurated-fistulized; class 3, implant periapical cyst; class 4, foreign body reaction; and class 5, poor bone healing. The histopathological presentation significantly influences the signs and symptoms of the lesion and may affect management decisions.[20]

Prevalence and incidence

Several studies report this type of implant lesion, all showing a low prevalence. A retrospective study by Quirynen et al[16] evaluates the incidence of RPI in single-tooth implants before and at the abutment connection. The study found that 1.6% of the implants placed in the maxilla and 2.7% placed in the mandible were affected.[16] Another study by Di Murro et al examined 1749 implants placed more than 20 years ago, finding that

only 6 implants were affected by RPI (0.35%).[21] Alssum et al found that 2 implants affected 215 patients examined clinically and radiographically (0.9%), and both implants were placed next to root canal–treated teeth.[22] In a retrospective case-control study by Solomonov et al, the incidence was reported to be 3.7%, which correlated with endodontic treatment in teeth adjacent to the implant.[23]

Etiological factors

Different etiological factors have been associated with RPI. However, preexisting inflammation is considered the most common cause. The source of inflammation can be bacteria or inflammatory cells from the remaining cyst or granuloma, which can lead to an active periapical implant lesion.[24] Several studies document that the periapical area of an implant is colonized by different bacterial species when RPIs is diagnosed. One retrospective study found that RPI lesions harbored more anaerobic bacteria, including Porphyromonas gingivalis, Corynebacterium, Streptococcus, and Klebsiella pneumonia.[25] A case report of an implant that developed mesial radiolucency 1.5 years after placement detected the presence of sulfur granules when the granulation tissue was evaluated histologically. These granules were identified as actinomycotic colonies.[26] Conversely, Romanos et al[27] detected bacteria in 1 out of 4 cases of RPI. The remaining 3 patients presented aseptic bone necrosis.[27] Another study demonstrated many plasma cells and lymphocytes in the biopsy of a lesion adjacent to a tooth with insufficient root canal treatment, consistent with inflammatory granuloma.[28] One case report identified a potential link between RPI and HIV infection. In this instance, the lesion appeared 3 months after implant insertion. Histopathological examination revealed acute inflammatory changes in the presence of fibrous connective tissues. The authors suggested that the effect of HIV on bone resorption and formation could explain the observed histopathological features. Nevertheless, it is essential to acknowledge the current scarcity of literature on implant complications in patients with HIV.[29]

In most case reports, active implant periapical lesions were found adjacent to teeth with unresolved periapical pathologies, contaminating the implant apex area. In a study by Zhou et al,[30] the incidence of RPI in implants placed next to endodontically treated teeth was 7.8%. The incidence increased when the implants were placed within a month of the completion of endodontic treatment of the adjacent teeth. Additionally, a positive correlation was found between the incidence of RPI and the distance between the implant and the adjacent teeth (Figure 2a).[30] Lefver et al report a high chance of developing an implant periapical lesion when the adjoining tooth had a clear periapical pathology (odds ratio = 8), whereas this chance dropped to 1% when the teeth did not show signs of endodontic pathology regardless of endodontic treatment.[31] One systemic review that included 7 clinical studies evaluating the success of implants placed next to endodontically treated teeth or teeth with periapical pathology concluded that the present evidence suggests an association between the endodontic status of teeth adjacent to dental implants and the success/survival of the implant. However, the results of these

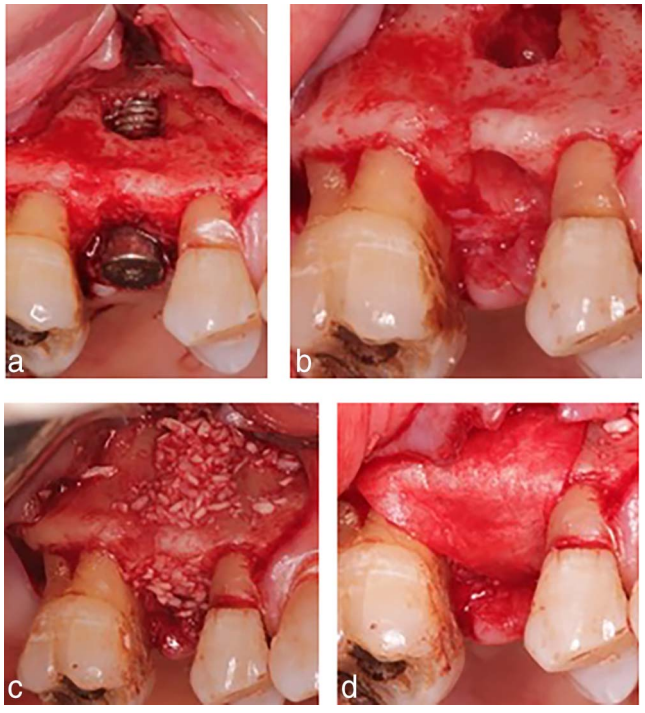

Figure 2. A symptomatic implant periapical lesion appeared 2 months following the immediate placement of an implant at site #15. The implant was positioned near the root tip of tooth #14, leading to its devitalization (a). The implant was mobile and subsequently removed, leaving a well-contained defect at the apical area (b). The defect was grafted with demineralized freeze-dried bone allograft and covered with a collagen membrane (c and d).

studies are inconsistent and may be subject to bias.[32] A case report presented 3 cases of RPI in implants placed next to asymptomatic endodontically treated teeth that had no clinical or radiographic symptoms. Due to an incomplete seal or inadequate obturation, the authors suggest that persistent bacteria can contaminate the implant surface and cause infection at the implant apex.[33] The correlation between the radiographic appearance of endodontically treated teeth and histological findings showed that teeth classified as successfully treated based on radiographs presented different reactions, ranging from normal and uninflamed to mild inflammation.[34] Moreover, radiographic studies reveal that the validity of periapical radiographs, commonly used to assess the success of endodontic treatment, is questionable. Some cases were documented as endodontically healthy because of the absence of periapical radiolucency on periapical radiographs that showed evidence of apical periodontitis on cone beam computerized tomography images.[35] This limitation should be considered when assessing teeth undergoing root canal treatment next to future implant sites.

Despite the success of immediate implant placement in extraction sockets with or without apical pathology, several case reports document different forms of early implant failure with immediate placement. Apical pathology before tooth extraction is considered a form of preexisting inflammation that may increase the risk of RPI. A histopathological study of

alveolar bone cores obtained from osteotomies during implant placement in sites that had been edentulous for a minimum of 1 year showed regions of nonviable bone and bacterial biofilms within the bone marrow spaces.[36] Drilling implant osteotomies may reactivate residual bacteria in the healed socket, contaminating the implant surface.[21] Therefore, the dental history of the implant site may affect the prognosis and risk of developing RPI after implant insertion. The risk of developing periapical lesions increases when dental implants are inserted to replace teeth with a history of periapical lesions even if endodontic treatment is administered (odds ratio = 7.23).[18] The chance of developing RPI reached 20% when implants were placed at sites with previous apical surgeries.[27]

The root tip near the implant is a risk factor for implant success. Evidence regarding the placement of dental implants in contact with dental tissue remains controversial. A prospective study revealed the uneventful healing of implants placed in contact with fragments of ankylosed teeth over 12–24 months.[37] A case series of 6 patients who received dental implants encroached through a root fragment demonstrated clinically and radiographically stable implants.[38] However, other case reports document RPI in implants adjacent to root tips.[39,40] Park et al[39] report a case in which the lesion developed 22 days after implant placement, causing pain, swelling, and a fistula at the buccal site. One week after removing the root tip and replacing the existing implant with a wider one, the patient's symptoms disappeared, and further complications did not appear at follow-up.[39]

Management

To date, the treatment of RPI remains empirical as there is no clear consensus in the literature regarding its management. However, clinicians should address the causative factors and lesions when developing a treatment plan. Sarmast et al[41] propose a decision tree for managing RPI lesions based on a literature review of 20 case reports of successfully treated cases. The decision tree addresses symptomatic and asymptomatic lesions and vital or nonvital adjacent teeth. The authors recommend antibiotic treatment for asymptomatic lesions that appear after implant placement. Surgical intervention was required if no signs of improvement were observed during follow-up. However, an endodontic assessment of the adjacent teeth is necessary when the lesions are symptomatic. Teeth with necrotic pulp should be treated first, and if root canal treatment resolves the lesion, no further treatment is needed. If no resolution is observed, apicectomy and surgical debridement of the implant lesions should be performed. Lesions that do not respond to the former treatment can be managed by implant apicectomy or guided bone regeneration (GBR). Conversely, surgical debridement of the lesion, with or without GBR, is performed when the pulp of the adjacent teeth is vital. Follow-up visits will help to determine the need for further treatment. Implant removal is indicated in cases of implant mobility or when surgical management fails to resolve lesions or symptoms (Table).[41]

In several case reports, a nonsurgical approach was used to treat RPI lesions, such as root canal treatment or extraction of the adjacent teeth, with or without systemic antibiotics.[42] In a case series by Sarmast et al,[43] resolution of the implant periapical lesion was observed after nonsurgical root canal treatment of necrotic neighboring teeth. The treatment resulted in the clinical and radiographic resolution of the implant periapical lesion during a 2-year follow-up period.[43] Gong et al report a case in which radiographic evidence of healing of the RPI lesion was observed following extraction of the adjacent tooth, which was affected by a periapical infection.[28] Chang et al[44] document the successful management of an implant periapical lesion that developed 1 month after implant placement. Initial treatment with amoxicillin and acetaminophen did not yield improvement, but substituting prednisolone, augmentin, and mefenamic acid resulted in complete healing, as evidenced by radiographs, with no recurrence of symptoms during a 2-year follow-up.[44] Wassdorp et al[45] report a case of an extensive periapical lesion involving the apical third of an implant placed in the mandibular incisor. The lesion developed 4 months after implant placement and was managed with a course of systemic antibiotics, resulting in the resolution of the lesion after 9 months without further intervention.[45] Despite these few case reports, most authors agree that surgical exposure of the apical part of the implant should be considered to ensure proper debridement and removal of the infected tissue. Defects in the apical area can be managed either by GBR or resection of the apical part of the implant.

Several case reports propose surgical debridement of RPI lesions and resection of the apical portion of the implant as treatment methods. Using the resection approach, Dahlin et al[46] treated 2 cases of periapical lesions around implants that had been functional for 1 and 2 years. Following surgery, bone fill was noticed on radiographs, and the implants were stable.[46] Another retrospective study of 39 implants that were clinically and radiographically identified to exhibit periapical lesions and treated with apicectomies showed that all except these implants remained stable and functioned over a 4.5- to 15-year follow-up period.[47] Shaffer et al presented a case in which an implant with a periapical lesion was surgically saved by resection of the exposed apical part of the implant and grafting of the defect with demineralized freeze-dried bone allograft (DFDBA) and tetracycline powder.[48]

The most used method for surgically managing RPI lesions is GBR. After exposing the apical part of the affected implant, the infected granulation tissue is removed, the exposed implant surface is debrided, and the lesion is grafted.[49] Graft materials are used to fill the cavity after debridement, which helps to stabilize blood clots and maintains space for optimal healing and complete bone regeneration. However, decontaminating the implant surface through mechanical debridement is a crucial step in ensuring the success of the bone graft. In a case study by Soldatos et al,[50] an Er, Cr:YSGG laser was used in conjunction with mechanical debridement for the surgical management of implants diagnosed as RPIs. After degranulation, the lesion was grafted with FDBA and covered with a collagen membrane. The patient healed uneventfully, resolving clinical symptoms and evidence of bone fill on radiographs taken at 6 and 13 months.[50] Sgarbanti and Mauro[51] demonstrated the successful management of periapical lesions using

Table

Summary of case reports demonstrating different management approaches for retrograde peri-implantitis lesion

			Nonsurgical Management		Surgical Management			
Study	Etiology	Time to Diagnosis	RCT of Adjacent Teeth	Pharmacological	Implant Debridement	Implant Resection or Removal	Bone Graft	Follow-up
Shaffer et al (1998)	All cases with failed RCT in adjacent teeth	Case 1: 2 months Case 2: 3 months Case 3: 7 months Case 4: 2 months	Yes	Case 4: Cephalosporin (250 mg, 4 times a day)	—	Case 1: implants were removed Case 2: resection Case 3: implant removed Case 4: 2 implants were removed	Case 2: DFDBA with tetracycline powder and a membrane Case 4: remaining implants DFDA	—
Chaffe et al (2001)	Pulpitis in the adjacent tooth after implant insertion	35 days		Amoxicillin (500 mg, 3 times daily)			Curettage and grafting with DFDBA covered with barrier membrane	Improvement was noticed after 2 years
Brisman et al (2001)	All cases with RCT in adjacent teeth	Case 1: 6 weeks Case 2: 4 weeks Case 3: 4 months Case 4: —	Case 1: apicectomy of adjacent tooth	Case 4: f clindamycin 300 mg, four times a day for 2 days then 150 g for a week)	Case 4: Implant debridement	Case 1: implant was removed Case 2: implant was removed and adjacent tooth with RCT was extracted Case 3: implant was removed		
Flanagan (2002)	Residual infection of the failed endodontic treatment	10 weeks		Penicillin, 500 mg, 4 times a day for 7 days	Implant surface was not treated	—	Paste of calcium hydroxide and water without membrane	11 months
Atullah et al (2006)	—	2 months	—	—	Irrigation with copious saline and chlorhexidine	—	BioOss and collagen membrane	3 months at stage 2
Balshi et al (2007)		1.6 years	—	Yes	—	Implants apicectomies	TCN paste, bone graft with membrane	4.5–15 months
Chang et al (2011)		1 month	—	Prednisolone, augmentin & mefenamic acid	—	—	—	2 years
Waasdorp et al (2010)		4 months	—	Systemic antibiotic	—	—	—	12 months
Soldatos et al (2018)	Previously infected site		—	Clindamycin 300 mg for a week	Er, Cr:YSGG laser	—	FDBA and collagen membrane	13 months
Di Murro (2021)	—	Range (5–165 days)	—	Amoxicillin 500 mg & metronidazole 250 mg starting 1 day before surgery	- Ultrasonic devices and carborundum bur - Chlorhexidine gluconate 0.2% for 2 min and saline	—	BioOSs without membrane	3 to 20 years

Table

Continued

Study	Etiology	Time to Diagnosis	Nonsurgical Management: RCT of Adjacent Teeth	Nonsurgical Management: Pharmacological	Surgical Management: Implant Debridement	Surgical Management: Implant Resection or Removal	Surgical Management: Bone Graft	Follow-up
Luongo et al (2022)	Case 1: root fragment Case 2: RCT in adjacent tooth	Case 1: started at day 1 till 1 month	—	Case 1 & 2: Augmentin started 1 day prior to surgery	—	Case 1: Implant was removed and replaced by another Case 2: resection of the implant apex and the RCT treated tooth	—	Case 1: 3 months and implant was loaded after Case 2: —
Sgarbanti et al (2023)	Adjacent nonvital tooth	14 months	Yes, and allowed to heal for 2 months before implant surgery	Amoxicillin 500 mg thrice daily for 7 days before endodontic treatment	- Mechanical instrumentation with titanium brush - Detoxification with tetracycline	—	Allograft particulate bone graft and collagen membrane	6 months
Gong et al (2023)	Case 1: Inappropriate RCT and close proximity of implant to the root Case 2: apical periodontitis	Case 1: — Case 2: at the second stage implant surgery	Case1: Ext of the adjacent tooth Case 2: Yes	Case 1: gentamicin solution Case 2: —	—	—	—	Case1: 12 months Case 2: 6 months
Oh S-L & Tordik (2024)	Apical periodontitis of adjacent teeth	16 years	Yes. With apical surgery to debride the apical defect	—	—	—	—	2 years

RCT, root canal treatment; DFDBA, demineralized freeze-dried bone allograft; TCN paste, tetracycline hydrochloride paste; FDBA, freeze-dried bone allograft.

DFDBA allografts and collagen membranes. In this case report, the patient presented with slight discomfort at the buccal surface of an implant placed 14 months previously, and a fistula developed at the implant apex and adjacent tooth. Radiography revealed a periapical lesion at the implant apex, proximal to the neighboring tooth. The patient was initially treated with antibiotics (amoxicillin, 500 mg thrice daily for 7 days); however, no improvement was observed. The vitality of adjacent teeth was assessed as nonvital. Endodontic treatment was performed, and GBR surgery was performed after 2 months. The implant surface was brushed with a titanium brush at a low oscillating speed, after which the area was decontaminated with tetracycline.[51] Xenografts alone or in combination with platelet-rich fibrin were used in another case report to treat an implant periapical lesion 4 months after the immediate placement of an implant at the upper lateral incisor site. The implant was restored 4 months later and remained stable at the 1-year follow-up visit.[52,53] One case report documented the successful management of implant periapical lesions using a calcium hydroxide paste as a graft material without a barrier membrane.[54]

Most studies investigating the management of RPI demonstrate implant stability after treatment over a specified follow-up period. However, implant removal is required in cases in which the osseointegration of the affected implant is compromised, and the implant is mobile (Figure 2b through d).[48,55]

Limitations

The low prevalence of this type of implant lesion has resulted in a lack of randomized controlled clinical trials addressing this problem, and most studies are case reports. The studies included in this review presented various management approaches. Most studies used surgical treatment with some exclusively employing nonsurgical treatment. Additionally, variation exists among the studies regarding surgical protocols with no definitive guidelines on the appropriate indications for each method. Comparison between nonsurgical and surgical treatments is not feasible as the cases included vary in clinical presentation and underlying etiology. Further prospective clinical research is needed to better understand the etiology of RPI and evaluate the efficacy of different treatment protocols.

Conclusion

The rare peri-implant disease RPI specifically affects the apical portion of the implant. Diagnosis relies on both clinical and

radiographic assessments. Data from case reports suggest that successful management depends on eliminating causative factors and surgical debridement and, in some cases, bone grafting to support regeneration. Further clinical research is needed to better understand its etiology and evaluate treatment efficacy. Long-term data are limited, and current management approaches largely depend on clinician experience.

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