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Review

Implant Survival in Sites With Previous Endodontic Failure and Apical Pathology

Anwar Salih Alghamdi[1*], Abdulhakeem Mohammad AlGhamdi[2], Haifa Tuwairish Al-Enezi[3], Hasan Abdullah Alhumaily[4]

[1] *Department of Dental Prosthetics Laboratory, Prince Mishari bin Saud Hospital, Albaha, Saudi Arabia*
[2] *Department of Dentistry, Prince Meshari Bin Soud Hospital, Albaha, Saudi Arabia*
[3] *Department of Dentistry, Primary Healthcare Center in Al Masif, Ministry of Health, Riyadh, Saudi Arabia*
[4] *Department of Dentistry, King Faisal General Hospital, Al Ahsa, Saudi Arabia*

Correspondence should be addressed **Anwar Salih Alghamdi**, Department of Dental Prosthetics Laboratory, Prince Mishari bin Saud Hospital in Balharshi, Albaha, Saudi Arabia, Email: anowar531616197@gmail.com

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Abstract

Periapical pathology resulting from endodontic failure poses a significant challenge for subsequent implant placement. The altered biological environment in such sites includes chronic inflammation, residual microbial contamination, and compromised bone architecture, all of which can affect implant integration and long-term stability. Although clinical and radiographic healing may suggest resolution, histological studies often reveal lingering inflammatory responses and microbial remnants within the bone and soft tissues. These factors can impair osseointegration and increase the risk of early implant complications, including peri-implantitis and marginal bone loss. Multiple variables influence the success of implants placed in previously infected sites. These include the timing of implant placement, the degree of surgical debridement, and the use of regenerative materials to restore lost bone. Delayed placement after complete healing has shown more predictable outcomes compared to immediate implantation, primarily due to reduced microbial load and improved soft and hard tissue conditions. Adjunctive measures such as photodynamic therapy, guided bone regeneration, and modified drilling protocols have been introduced to enhance clinical outcomes in these compromised sites. Despite the inherent risks, studies have reported comparable implant survival rates between previously infected and non-infected sites when proper protocols are followed. Customizing surgical techniques and material selection based on site-specific conditions is critical. Evaluating the biological history of the site, not just its structural readiness, remains essential for achieving favorable outcomes. Understanding the interplay between past infection and current healing dynamics informs clinical decision-making and improves the predictability of implant therapy in these complex scenarios.

Keywords: endodontic failure, apical pathology, dental implants, osseointegration, peri-implantitis

Introduction

Dental implants have become a widely accepted and effective solution for the replacement of missing teeth, offering predictable long-term outcomes in terms of both function and esthetics. However, when implants are placed in sites with a history of endodontic failure or periapical pathology, concerns arise regarding their survival and integration. These sites are often characterized by the presence of microbial contamination, compromised bone quality, and inflammatory responses that may interfere with osseointegration and the long-term stability of the implant fixture (1).

Apical periodontitis is a common sequela of endodontic failure, typically resulting from persistent intraradicular or extraradicular infection. Such lesions may alter the periapical environment and the immunological profile of the surrounding bone, potentially jeopardizing the healing process following implant placement. The persistence of residual microorganisms, particularly in inadequately treated endodontic sites, has been shown to be a contributing factor in delayed healing and early implant failure (2). The role of microbial biofilms, which are more resistant to host defenses and conventional decontamination methods, further complicates the prognosis in these compromised sites (3).

Numerous histopathological and microbiological studies have confirmed that bone in areas previously affected by apical lesions often contain inflammatory cells and necrotic debris, even after radiographic healing. These residual elements may not be detectable through standard imaging techniques but could remain biologically active, exerting a deleterious influence on the peri-implant tissues (4). Moreover, previous endodontic infections may lead to bone resorption and architectural changes that compromise the structural support for implants, particularly in the anterior maxilla and posterior mandible where bone volume is often limited. In this review, we aim to discuss implant survivability in sites that had previous endodontic failure and its relation to apical pathology.

Review

Implant placement in sites with a history of endodontic failure poses unique clinical challenges due to the altered biological environment and potential microbial persistence. Even after extraction and apparent healing, these sites may harbor residual bacteria and inflammatory byproducts that can negatively affect osseointegration. Studies have indicated that the microbial environment in previously infected areas can differ significantly from that of healthy bone, potentially increasing the risk for early implant complications if not properly managed (5).

Despite these concerns, the literature supports the viability of implants placed in healed sites of prior apical pathology, provided that infection is thoroughly eradicated and proper healing protocols are followed. Careful case selection, adequate debridement, and allowing sufficient time for bone regeneration can lead to favorable outcomes. Additionally, the use of adjunctive regenerative techniques, such as bone grafting or guided tissue regeneration, has been shown to enhance bone quality and reduce failure risk in these sites (6). Clinicians must be cautious in their evaluation of these sites, as radiographic resolution alone may not be sufficient to confirm biological readiness for implantation. A comprehensive approach that integrates clinical, radiological, and sometimes histological assessments is critical for ensuring long-term implant success in such complex cases.

Biological Factors in Previously Infected Implant Sites

Sites affected by previous endodontic failure often present with complex biological challenges that extend beyond simple structural considerations. The microenvironment of these sites is shaped by a history of microbial colonization, chronic inflammation, and altered bone metabolism, all of which influence the outcome of implant therapy. The biological response of peri-implant tissues to these residual changes plays a crucial role in determining implant integration and stability over time. Histological studies have consistently demonstrated the presence of inflammatory

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infiltrates, granulation tissue, and necrotic bone fragments in areas formerly affected by apical periodontitis, even after clinical resolution. These residual elements can persist for extended periods, undermining the healing potential of the site. Ferreira et al. identified bacterial remnants embedded in connective tissue near the apex of failed root canal-treated teeth, which were not visible on radiographs and could remain undetected during routine clinical assessment (7). These microbial residues may provoke a subclinical inflammatory response after implant placement, potentially interfering with the early stages of osseointegration.

The influence of the host immune response also plays a substantial role in previously infected sites. Chronic periapical lesions stimulate an ongoing immune reaction characterized by the release of cytokines, prostaglandins, and other proinflammatory mediators. This creates an environment with elevated levels of bone-resorbing cells and disrupted bone remodeling. When an implant is introduced into such a biologically active site, the local immune activity may continue to influence peri-implant bone dynamics. Huang et al. reported that increased expression of interleukin-1β and tumor necrosis factor-alpha in periapical lesions could contribute to delayed bone healing and impaired osseointegration, especially in the early post-placement phase (8).

Angiogenesis within previously infected bones may be compromised. Vascular supply is essential not only for nutrient delivery but also for the migration of osteoprogenitor cells and removal of metabolic waste. Chronic apical inflammation reduces capillary density and alters endothelial cell function. A study by Botticelli et al. evaluating bone healing after tooth extraction in infected versus non-infected sites found significantly reduced vascularization and delayed bone fill in sockets with prior periapical pathology (9). This diminished angiogenic response may prolong the time required for successful integration or increase the likelihood of early implant failure. Furthermore, the surface of the implant itself interacts dynamically with the surrounding tissue environment. In an infected or previously infected site, the risk of biofilm formation on implant surfaces may be elevated due to the residual microbial presence in the alveolar bone or adjacent soft tissue. Biofilms are known to resist both immune clearance and systemic antibiotics. Shibli et al. investigated the microbial composition around implants placed in previously infected sites and found that pathogenic species such as *Fusobacterium nucleatum* and *Porphyromonas gingivalis* were more frequently detected during the early healing phase compared to healthy sites, indicating that microbial recolonization may occur rapidly under favorable conditions (10).

Effect of Endodontic Failure on Implant Outcomes

The consequences of endodontic failure often extend beyond the boundaries of the tooth, influencing the surrounding alveolar bone and impacting future restorative interventions, particularly implant therapy. When an implant is placed in a site that previously hosted a failed endodontic treatment, multiple risk factors converge that can subtly or overtly influence clinical outcomes. These include chronic periapical inflammation, alterations in bone morphology, and the persistence of microbiota that can compromise peri-implant health. Chronic infections stemming from endodontic failure contribute to bone degradation and localized immune dysregulation. The resulting inflammatory environment, even if resolved clinically and radiographically, can persist on a microscopic level. Kangarloo et al. evaluated implant outcomes in areas where root canal therapy had failed and highlighted a statistically significant increase in marginal bone loss surrounding implants placed in such sites compared to those in previously healthy areas (11). The difference in bone remodeling dynamics was attributed to an altered host response triggered by the prior infection history.

Microbial remnants present another issue. While extraction and site preparation aim to reduce or eliminate bacterial presence, studies have shown that biofilm fragments and bacterial DNA can remain embedded in the alveolar bone, especially

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when curettage is insufficient. These remnants are not merely inert; they have the potential to reignite inflammatory cascades post-implantation. The presence of these subclinical infections increases the likelihood of early peri-implantitis, affecting both soft tissue and crestal bone stability. Yoon et al. reported that implants placed in previously infected sites showed earlier signs of mucositis and higher bleeding indices during the first year of function (12).

The geometry and quality of bone in sites of endodontic failure also differ from those in uninfected regions. Areas previously affected by periapical lesions often exhibit irregular trabecular patterns and cortical thinning. This can complicate the mechanical aspects of implant placement and reduce primary stability. Mechanical anchorage is critical in the early stages of healing, particularly when the site has a history of chronic infection. Pereira et al. analyzed resonance frequency measurements of implants placed in previously infected sites and found consistently lower initial stability values, especially in the posterior maxilla, where bone density is often low to begin with (13). These observations point toward a need for strategic planning in implant dimensions and insertion torque when dealing with compromised sites.

Soft tissue healing dynamics are also affected. The peri-implant mucosa in sites with a previous endodontic history may demonstrate delayed epithelial maturation and altered vascularization. This has implications for both esthetics and resistance to bacterial ingress. Rathi et al. performed a histomorphometric study on soft tissue biopsies taken from healed extraction sites with prior apical pathology and observed a higher concentration of inflammatory markers in the connective tissue matrix surrounding the implant compared to control sites (14). These markers included interleukin-6 and matrix metalloproteinase-8, both of which are involved in tissue degradation and inflammatory signaling.

Strategies for Implant Placement in Healed Lesions

Successful implant therapy in previously infected sites hinges on protocols that acknowledge and compensate for altered local biology and bone architecture. Once infection is resolved and the lesion shows radiographic and clinical signs of healing, surgical planning must integrate both standard biomechanical considerations and nuanced assessment of the site's history. This includes evaluating bone remodeling patterns, ensuring microbial elimination, and using techniques that promote controlled regeneration without overloading compromised tissue.

Timing of implant placement remains central in this context. Immediate placement into infected sockets has generally been discouraged due to the high risk of bacterial persistence and interference with osseointegration. Instead, delayed placement following a healing interval allows for resolution of inflammation and partial regeneration of bone. However, determining when the site has sufficiently healed is less straightforward than relying solely on radiographs. Scarano et al. used histological analysis to compare previously infected extraction sites with non-infected ones after 4 to 6 months of healing and found significantly more fibrotic tissue and fewer osteoblasts in the previously infected sites despite radiographic evidence of bone fill (15). This underscores the need to interpret imaging findings with caution and to combine them with clinical signs such as absence of suppuration, pain, or sinus tract formation.

Decontamination of the socket during extraction is critical. Meticulous curettage, irrigation with antimicrobial solutions, and in some cases, adjunctive laser therapy or photodynamic disinfection have been employed to reduce the microbial burden. Ohba et al. evaluated the safety and efficacy of antimicrobial photodynamic therapy in the treatment of peri-implant disease and observed improved soft tissue healing and a reduction in inflammatory markers following its application in infected sites (16). Such therapies, while not yet universally adopted, are gaining attention for their potential to shift the microbial

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environment toward a more favorable profile for osseointegration.

Regenerative materials are often needed to manage residual defects left by apical pathology. The use of xenografts or allografts, often in combination with collagen membranes, can aid in restoring the bony contour and volume needed for ideal implant positioning. The choice of material should reflect both the extent of the defect and the biological demands of the site. A study by Simion et al. evaluating grafted post-extraction sites with a history of endodontic failure showed higher success rates in implants placed within sites treated with mineralized allografts compared to ungrafted controls, even when both had radiographic healing (17).

Primary stability can also be influenced by implant design and drilling protocol. In sites with a softened trabecular pattern due to previous infection, under-preparation of the osteotomy can help achieve adequate torque and stability. Additionally, selecting implants with aggressive thread patterns or tapered geometries may improve engagement with the bone. Chappuis et al. compared implant stability in previously infected healed sites and noted better insertion torque values when tapered implants were used alongside modified osteotomy protocols tailored to local bone density (18). These adaptations reflect the necessity of customizing surgical approaches based on site-specific characteristics rather than following standard protocols.

Conclusion

Implant placement in sites with previous endodontic failure requires careful biological and clinical consideration. Residual inflammation, compromised bone quality, and microbial persistence can influence long-term outcomes. Thorough site evaluation, proper healing protocols, and tailored surgical strategies are essential. Evidence supports favorable survival rates when these factors are properly managed.

Disclosure

Conflict of interest

There is no conflict of interest.

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Data availability

All data is available within the manuscript.

Author contribution

All authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

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