Dental Pathophysiology of Odontogenic Sinusitis

Endodontic Infections

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KEYWORDS

• Apical periodontitis • Cone-beam computed tomography • Endodontics
• Maxillary sinus • Odontogenic maxillary sinusitis • Root canal treatment

KEY POINTS

- Odontogenic sinusitis (ODS), while previously underreported, is now recognized as a major cause of chronic maxillary sinusitis, with endodontic infections being one of the most common etiologies.
- Progression and severity of ODS due to endodontic infections can be explained by the unique microbial profile of pulpitis, pulpal necrosis, and apical periodontitis, as well as the innate and adaptive immune responses mounted against these infections.
- Pulpal and periapical infections commonly cause maxillary sinus mucositis (reactive mucosal edema without purulent infection), and this is distinct from purulent ODS. Mucositis is represented by mucosal thickening on computed tomography, whereas ODS will more often display partial to complete sinus opacification.
- Effective management requires a multidisciplinary approach, focused on accurately diagnosing the endodontic pathology, and eradicating the infection through targeted endodontic treatment, with or without subsequent sinus surgical interventions as needed.

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INTRODUCTION

Bacterial odontogenic sinusitis (ODS) involves inflammation and infection of the maxillary sinus that may spread to other sinuses. The infectious sinusitis can arise from a multitude of infectious dental problems in the nearby maxillary region or accidental trauma during dental procedures or other oral treatments.[1,2] Endodontic infections are one of the most common causes of ODS.[1] The complex relationship between endodontic infections and sinusitis has gained attention recently because of its clinical importance and the diagnostic challenges it presents. Historically, ODS has been overlooked, with earlier estimates suggesting that only 10% to 12% of sinusitis cases were linked to dental infections.[3,4] Recent research indicates a higher incidence of ODS, comprising approximately 25% to 40%[5,6] of all cases of chronic maxillary sinusitis,[5,6] and 45 to 75%[7–9] of unilateral maxillary sinus opacification observed on tomography scans.[7,9]

Endodontic issues comprise a range of conditions, including pulpal necrosis, root fracture, apical periodontitis, and periapical lesions such as abscesses, granulomas, or cysts. Bacterial ODS associated with endodontic infections arises from the significant interaction between distinct microbial factors and the close proximity of the posterior maxillary tooth root apices to the maxillary sinus mucosa. Furthermore, the immune responses activated by the host in response to endodontic infections are crucial in determining the advancement and severity of sinusitis. The interplay between dental pathogens and sinus tissues initiates complex inflammatory reactions, causing tissue damage and aiding in infectious spread to the sinuses. Understanding the exact mechanisms by which dental pathologies impact the development and progression of sinusitis is essential for optimal diagnostic and therapeutic strategies. Applying this understanding to one's clinical practice should not only prevent serious infectious complications but will also promote both oral and overall health in patients.

This study offers a comprehensive description of endodontic pathophysiology as it contributes to ODS development. By clarifying the links among microbial factors, dental and sinus anatomy, and immune responses, the goal is to enhance one's understanding of this intricate condition and lay the groundwork for more accurate diagnosis and successful treatment strategies.

ENDODONTIC PATHOPHYSIOLOGY AND ODONTOGENIC SINUSITIS

Understanding pulpal and periapical disorders in the context of ODS is important because they are one of the most common causes of ODS.[1,9] Appreciating pulpal and periapical disease origins and factors behind their progression directly impacts the development and progression of the potential sinusitis that can cause. Pulpal disorders like pulpitis and pulpal necrosis can act as sources of bacterial infection, creating an environment conducive to microbial growth within the root canal and periapical tissues. The inflammatory response of the sinus to periapical infection and other irritants originating from endodontic issues typically begins with localized inflammation and sinus mucosal edema (mucositis), which may progress to a fulminant purulent maxillary sinusitis (ODS).[10] However, whether mucosal inflammation always precedes purulent ODS remains to be determined.

Endodontic infection occurs when microorganisms and their by-products penetrate the dental pulp, which is typically composed of sterile loose connective tissue.[11] Normally, the dental pulp is enclosed within a space formed by mineralized tissues, offering significant mechanical support and protection from the oral environment.[12] However, if this protective barrier is compromised, the exposed pulp becomes susceptible to various harmful stimuli. Factors such as caries, trauma, dentinal microcracks, exposure

of lateral canals due to damaged cementum from severe gingival diseases, and defective restorations create pathways for microorganisms and their toxins to invade the pulp tissue.[12,13]

Inflammation is the initial reaction of the dental pulp to irritants, facilitated by its rich microcirculation and innervation. **Fig. 1**and **Table 1** describe and illustrate the stages of pulpal disease progression. Pulpitis, characterized by inflammation in response to microbial, chemical, or physical irritants, is considered reversible when the pulp can return to its normal state after the irritant is removed.[14] Short-term irritants typically prompt temporary acute inflammation, followed by tissue healing. Conversely, long-term irritants, like deep caries, gradually erode dentin and approach the pulp, triggering an intense inflammatory response.[15] Even before caries reach the pulp, bacterial toxins can penetrate dentin tubules, worsening the pulpal inflammatory process. Without treatment, inflammation progresses, causing severe damage to the pulp tissue and leading to irreversible pulpitis. This usually occurs when caries nearly reach the pulp or when caries bacterial biofilms directly contact the pulp. This interaction triggers intense inflammation that can advance to necrosis if the irritant overwhelms the pulp's defense mechanisms and disrupts its blood flow.[12,14] The progression

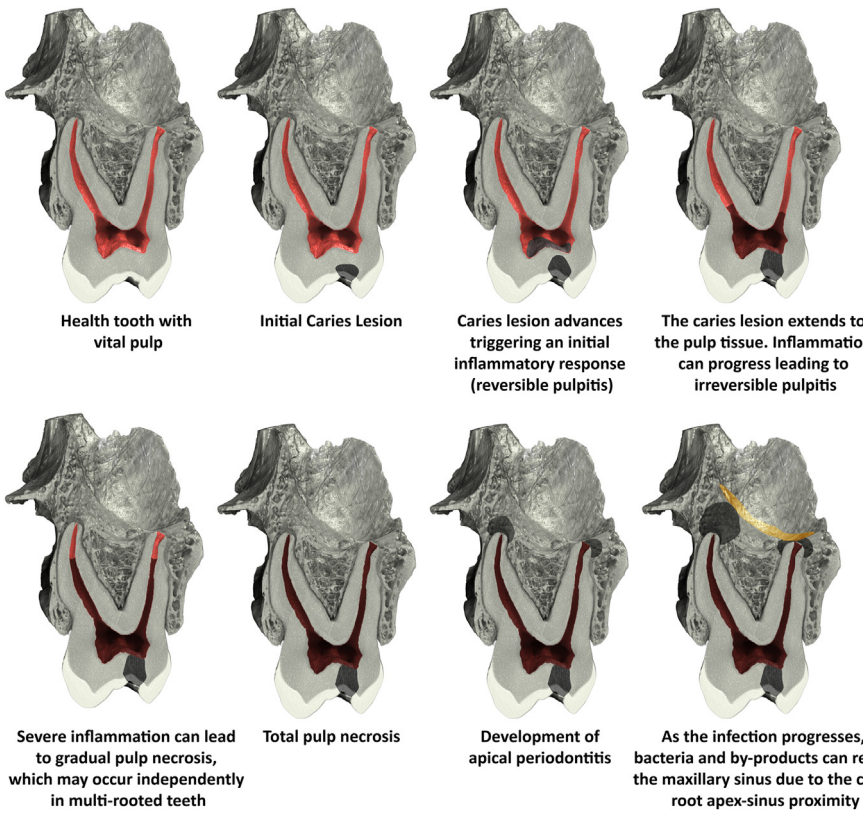

Fig. 1. Representative images illustrating the progression of endodontic infection until it reaches the maxillary sinus: (*A*) vital pulp; (*B*) initial caries lesion; (*C*) reversible pulpitis; (*D*) irreversible pulpitis; (*E*) partial pulp necrosis; (*F*) total pulp necrosis; (*G*) apical periodontitis; (*H*) maxillary sinus mucositis (*yellow color* area representing reactive mucosal thickening).

Table 1
Pulpal and periradicular pathologies and their main characteristics

Pulpal and Periradicular Diagnosis	Definition
Vital Pulp	Normal dental pulp is enclosed within a space formed by mineralized tissues and, if subjected to stimuli such as caries, the pulp can become inflamed.
Reversible Pulpitis	Mild pulp inflammation, but the pulp can return to its normal state after the removal of the stimulus. Often caused by short-term and superficial stimuli (eg, superficial caries and dentin exposure).
Irreversible Pulpitis	Severe pulp inflammation. The pulp cannot return to its normal state after the removal of the stimulus. Often caused by long-term and intense stimuli (eg, deep caries).
Pulp Necrosis	The infectious/inflammatory stimulus overwhelms the pulp's defense mechanisms and disrupts its blood flow. The pulp necrosis is a gradual process, moving through tissue compartments and gradually advancing toward the apex of the tooth. In multi-rooted teeth, pulp necrosis can occur independently in different roots.
Apical Periodontitis	Bacteria and their by-products can spread toward the surrounding alveolar bone through apical foramina, which are the exits of the root canal system situated near the tips of the root apices. This spread leads to periapical pathosis, characterized by an infectious process where periapical tissue responds to the microbial infection from the root canal. The periapical lesion may initially appear as a periapical abscess during the acute phase or as a periapical cyst or granuloma during the chronic phase or could exhibit mixtures of these features.

toward pulp necrosis is gradual, moving through tissue compartments and gradually advancing toward the root apices.[14,16] In multi-rooted teeth, like maxillary premolars and molars, necrosis can occur independently in different roots. Therefore, one root may have pulp necrosis, while others maintain vital pulp, showing varying stages of tissue integrity.[17] It is important to recognize that in some cases, significant trauma that disrupts the pulp's neurovascular bundle can lead to immediate pulp necrosis.[12]

When vital pulp tissue is absent, bacteria and their by-products can spread toward the surrounding alveolar bone through foramina, which are the exits of the root canal system situated near the root apices. This spread leads to periapical pathosis, characterized by an infectious process where periapical tissue responds to the microbial infection from the root canal.[11,13] As the periapical infection and inflammation advance, infection spreads through the alveolar bone marrow along blood vessel and lymphatic pathways. The progression of the endodontic lesion typically involves an acute phase, initiated by the activation of the innate immune system, followed by a chronic phase, marked by an adaptive immune response.[13,18] Consequently, a periapical lesion (radiolucency on radiographic imaging) may represent a periapical abscess during the acute phase, or a periapical cyst or granuloma during the chronic phase.[18] However, a chronic infection can also transition into an acute phase, termed a phoenix abscess, due to an imbalance between microbial virulence and host immune factors.[19]

Half of the global population has at least 1 tooth with a periapical lesion.[20] Research suggests that periapical infections in maxillary posterior teeth or canines are associated

with either maxillary sinus mucositis or ODS in 50% or more of cases.[21–23] ODS originating from endodontic problems typically occurs when periapical lesions spread to the maxillary sinus. This can happen through inflammation and possibly erosion of the overlying bone and sinus mucosa (ie, the Schneiderian membrane), thrombophlebitic spread of bacteria through intact bone and sinus mucosa via venous and lymphatic channels, displacement of endodontic materials into the sinus cavity, perforation of the sinus mucosa during endodontic procedures, or dental traumas like vertical root fractures.[24–26]

Infectious spread from teeth to the maxillary sinus is made easier by their close proximity. Among maxillary teeth, second molars most closely approximate the maxillary sinus, followed by first molars, premolars, and canines.[26,27] On average, the distance between maxillary molar and premolar roots and the maxillary sinus is approximately 1.97 mm. Root tips may also protrude into the sinus floor mucosa, causing minor elevations or prominences in the sinus floor.[27] The ongoing expansion and pneumatization of the maxillary sinus overtime may lead to the inferior displacement of the sinus floor toward the maxillary posterior teeth roots, sometimes leaving the sinus mucosa directly apposing the tooth roots without intervening periapical bone.[25,28] While some bacterial species from endodontic infections may directly invade through sinus mucosa, others express virulence factors and toxins that can induce an intense mucosal innate and adaptive immune response seen in both the periapical lesion and sinus mucosa.[29–31]

Vertical root fractures are another possible cause of ODS, as they provide pathways for various inflammation-inducing agents like bacteria and their by-products and necrotic pulp products, to spread through the lateral aspect of roots to periapical tissues. Bacteria at the fracture site can also originate from communication with the gingival sulcus, as vertical root fractures often lead to periodontal breakdown and deep probing defects. This type of fracture is more commonly associated with teeth that have undergone root canal treatment and are less prevalent in vital teeth.[32] Excessive removal of dentin during root canal or post space preparations and excessive forces during root filling or post placement, are common causes of vertical root fractures.[32] Additionally, previously treated teeth may exhibit persistent apical periodontitis due to challenges in treating the root canal system, coronal restoration leakage, or untreated canals.[33] Furthermore, a chronic periapical infection can potentially lead to an oroantral fistula, a communication from the sinus lumen to the oral cavity via infected periapical and periodontal spaces.[34]

ENDODONTIC INFECTIONS

While various microorganisms colonize the root canal system, bacteria emerge as the primary etiologic agents of endodontic infections.[35–37] The formation of biofilms in these infections is a complex process that enables microorganisms to survive and persist in the hostile environment of the root canal system. These biofilms not only protect bacteria from external threats but also contribute to their resistance against antimicrobial agents, presenting challenges for complete infection eradication.[37,38] Numerous in situ studies have shown apical periodontitis being associated with biofilms formed both intraradicularly[39–41] and along the external root surfaces.[36]

Endodontic infections have been classified based on the timing of infection establishment within the root canal system (primary, secondary, or persistent) and their location (intraradicular or extraradicular; **Fig. 2** and **Table 2**). Primary intraradicular endodontic infections begin with the colonization of necrotic pulp tissue. Secondary intraradicular endodontic infections are characterized by the presence of microorganisms that were

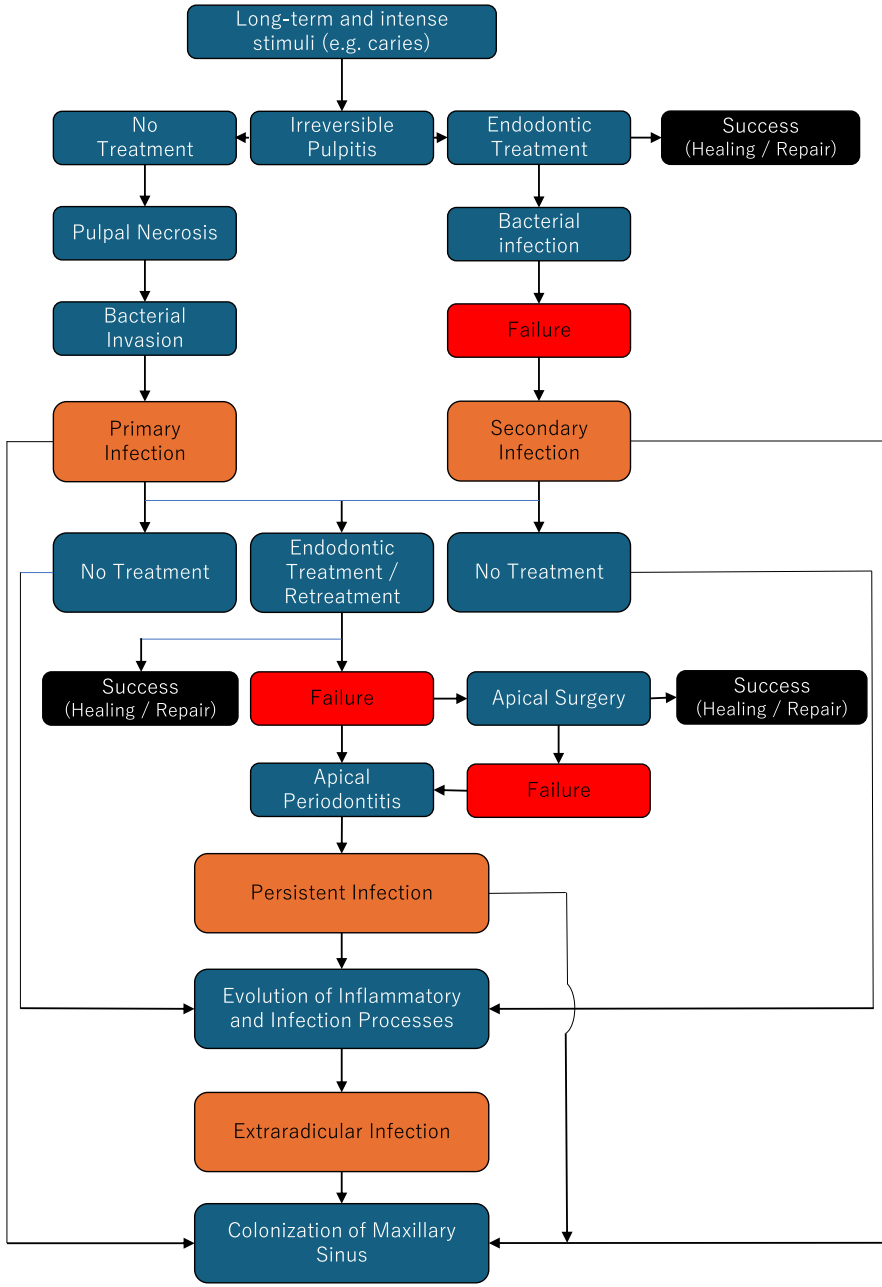

Fig. 2. Schematic of pulpal and periapical pathologies before and after endodontic treatments.

not initially present in the primary infection or those that colonize the root canal following endodontic intervention, either after the completion of endodontic treatment or between sessions. Persistent intraradicular endodontic infections involve microorganisms capable of resisting the disinfection procedures employed during endodontic

Table 2
Types of endodontic infections

Infection Types		Definition
Intraradicular	Primary	It denotes infected necrotic pulp tissue, comprising pioneer species that invade and colonize the necrotic pulp environment.
	Secondary	It arises from secondary bacterial invasion, precipitated by coronal leakage or a disruption in the aseptic chain during prior treatment.
	Persistent	It entails microorganisms resilient to the disinfection procedures performed during endodontic treatment or retreatment, representing a significant contributor to treatment failure.
Extraradicular		It comprises microorganisms derived from preexisting intraradicular infections, possessing the ability to evade root canal system and colonize periradicular tissues.

treatment.[42] Extraradicular infections arise from microorganisms originating in preexisting infections that spread to and colonize periradicular tissues.[43,44] Although extraradicular infections are rare,[41] they primarily manifest as acute periapical abscesses. In such cases, microorganisms effectively evade host defenses and colonize the extraradicular surface. If left untreated and allowed to progress, these infections can lead to systemic complications.[45] It is reasonable to assume that both extraradicular and intraradicular infections lead to ODS through periapical infection.

Each type of infection presents distinctive characteristics in its microbial profile. Primary endodontic infections are characterized by a predominance of anaerobic bacteria, including species like *Prevotella* spp., *Porphyromonas* spp., *Fusobacterium* spp., *Treponema* spp., and *Actinomyces* spp.[46–51] Secondary and persistent infections also exhibit a diverse mixed profile, with species such as *Enterococcus faecalis*, *Parvimonas micra*, *Porphyromonas endodontalis*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Dialister invisus*, *Propionibacterium acnes*, *Tannerella forsythia*, and *Treponema denticola* being among the most prevalent.[52–55] Additionally, the genus *Actinomyces* may play a significant role in secondary/persistent infections and may be associated with cases of extraradicular biofilm.[56,57]

INTERPLAY BETWEEN ENDODONTIC AND PERIODONTAL INFECTIONS

While not the focus of this study, it is important to appreciate that infectious periodontal disease can also lead to ODS, termed periodontitis or marginal periodontitis.[58] This is distinct from endodontic disease in that it begins primarily in the periodontium, the tissues supporting the dentition (periodontal ligament, cementum, gingiva, alveolar bone). In advanced stages, it may also impact the periapical region.[59] Periodontal infections exhibit a microbial profile similar to that of endodontic disease, with genera such as *Porphyromonas*, *Treponema*, *Prevotella*, *Filifactor*, *Tannerella*, and *Fusobacterium* commonly being isolated.[59] However, the pathogenesis between endodontic and periodontal infections differs significantly. While endodontic infections arise from microbial invasion of pulp tissue within the root canal, periodontal disease occurs as a consequence of dysbiosis in the oral microbiome.[60]

The periodontal and pulp tissues are connected anatomically through 3 pathways: dentinal tubules, lateral and accessory canals, and the apical foramen.[61] When dental pulp and periodontal tissues undergo inflammation and/or infection, these pathways

allow for pathologic infectious spread between the spaces.[61] Such pathologic spread can lead to endodontic-periodontal ("endo-perio") lesions, where the inflammatory/infectious process affects both pulpal and periodontal tissues.[62] Recent advancements in understanding endo-perio lesions have led to a new classification, moving away from the idea of primary endodontic or primary periodontal conditions. This modern approach emphasizes the interrelationship between endodontic and periodontal diseases, recognizing their complex interplay in lesion development and progression.[62] Only 1 study to date has shown the potential of endo-perio lesions to cause ODS.[63] When infections from both the inside of the tooth (endodontic) and the surrounding supporting structures (periodontal) extend into the sinus area, it is crucial to determine the appropriate treatment approach. The dental treatment may need to focus only on the root canal if the infection is solely endodontic in origin, or it may require combined root canal and periodontal treatments if both areas are diseased.

INTERPLAY BETWEEN ENDODONTIC AND SINUS INFECTIONS

The sinus microbiome consists of complex communities of bacteria commonly found in the upper respiratory tract, including species like *Corynebacterium*, *Staphylococcus*, *Streptococcus*, *Haemophilus*, *Moraxella*, *Propionibacterium*, and *Pseudomonas*.[64] These bacteria are part of the normal microbial community in the sinuses and usually do not cause illness unless there is a disruption in the sinus environment. On the other hand, the oral microbiome includes bacteria such as *Actinomyces*, *Porphyromonas*, *Prevotella*, *Fusobacterium*, *Treponema*, *Lactobacillus*, *Enterococcus*, *Peptostreptococcus*, and *Streptococcus* species.[65] While these bacteria contribute to maintaining oral health, they can lead to dental caries, periodontal disease, and other oral infections when their balance is disturbed. Species like *Streptococcus*, *Prevotella*, *Porphyromonas*, *Peptostreptococcus*, and *Fusobacterium* have the ability to migrate through pulp chambers and the periapical tissues and ultimately penetrate the sinuses to cause ODS.[25] This migration highlights the intimate relationship between oral and sinus ecosystems, where imbalances in one environment can significantly affect the other.

ODS is typically a polymicrobial infection involving bacteria from both the oral and upper respiratory tracts, though oral bacteria tend to predominate.[27,62,66,67] Multiple studies have demonstrated the microbial profile in ODS to be distinct from that of non-odontogenic rhinosinusitis. Patients with ODS exhibit a higher prevalence of anaerobes and alpha-hemolytic streptococci (especially *Streptococcus intermedius*, *Streptococcus anginosus*, and *Streptococcus constellatus*), and less frequently bacteria cultured in acute and chronic rhinosinusitis like *Haemophilus influenzae, Streptococcus pneumonia,* and *Moraxella catarrhalis*. The microbial profile of ODS closely resembles that of endodontic infections (eg, *Peptostreptococcus* spp., *Prevotella* spp., and *Fusobacterium* spp.).[1,25,68–71] While more research is necessary to understand the pathophysiologic spread of bacteria from periapical tissues to the maxillary sinus, these microbiologic studies provide a strong foundation from which one can support a causal relationship between endodontic infections and ODS development. It should also be noted that no evidence to date supports that purulent maxillary sinusitis spreads from the sinus to the teeth, though future research could explore whether this occurs.

INTEGRATING PATHOPHYSIOLOGY WITH CLINICAL CARE

Overview of Endodontic Diagnosis and Treatment

Managing ODS due to endodontic disease starts with a thorough diagnostic assessment to confirm the infectious dental origin. The endodontic examination entails

evaluating pulp vitality and periapical disease. Cold pulp testing is widely regarded as the gold standard for detecting pulpal necrosis, with a lack of tooth response to the cold stimulus suggesting necrosis. Additionally, prior endodontic treatments must be evaluated for potential issues such as leakage, inadequate root canal fillings, or untreated canal anatomy. Conventional periapical radiography provides 2 dimensional images that can reveal periapical changes, bone destruction, and other signs of endodontic pathology. However, it may not always provide sufficient detail, especially for complex anatomy and treatments. In these cases, cone beam computed tomography (CBCT) offers 3 dimensional imaging with higher resolution and accuracy compared to conventional radiography. It provides detailed views of the tooth, surrounding bone, and adjacent structures, allowing for better visualization of periapical lesions, root canal anatomy, other potential sources of infection, as well as sinus pathology. Thus, radiographic and/or tomographic assessments are essential for corroborating the clinical diagnosis and aiding in the formulation of an effective treatment plan.

Regarding endodontic treatment of ODS, the primary objective is to eradicate the pulpal infectious source and allow the maxillary sinus's mucosal inflammatory response plus mucociliary clearance to eliminate the purulent sinusitis. From an endodontic perspective, this typically involves either root canal treatment, root canal retreatment, or endodontic microsurgery (apicoectomy), and these will be detailed in a later study in this issue. In brief, root canal treatment or retreatment aims to remove infected or necrotic pulp tissue from the root canal system, disinfecting the root canal chambers, then filling the canals with biocompatible material to prevent reinfection, and this usually leads to resolution of the periapical disease. If root canal treatments fail to resolve the periapical lesion, sometimes an apicoectomy is necessary. Apicoectomy involves the surgical removal of the infected periapical tissue along with the tip(s) of the involved root(s), which should eliminate any remaining infection and promote periapical tissue healing. If these treatments fail, infected teeth may require extraction. Establishing close collaborative relationships among endodontists, otolaryngologists, and other dental specialists is essential for comprehensive ODS management, ensuring successful oral and sinusitis outcomes.

Clinical Cases

Case 1

A 52 year old man sought dental care complaining of pain in the upper left region of his mouth (**Fig. 3**A–L). Upon examination, it was observed that left upper second molar had a large composite filling and did not respond to cold pulp testing, indicating possible pulp necrosis. Radiographs and CBCT scans showed no periapical lesion, but complete opacification of the portion of the maxillary sinus seen, adjacent to the infected molar (see **Fig. 3**A-C). This likely represented ODS, even though there was no definitive confirmation of sinus purulence (ie, no nasal endoscopy or sinus surgery was performed). Given the diagnosis of pulp necrosis, the patient underwent endodontic treatment of the tooth in a single session without post-procedural systemic antibiotics. The procedure involved the use of 2.5% sodium hypochlorite and 17% ethylenediaminetetraacetic acid for irrigation. Follow-up clinical examinations and CBCT scans were performed at 1 and 4 years after the initial treatment. These assessments revealed successful root canal filling, normal periapical tissues, and resolution of the maxillary sinus opacification (see **Fig. 3**G-L).

Case 2

A 16 year old girl presented with intense pain in her upper left molar area and swelling of the alveolar mucosa (**Fig. 4**A–I). Clinical and radiographic assessments

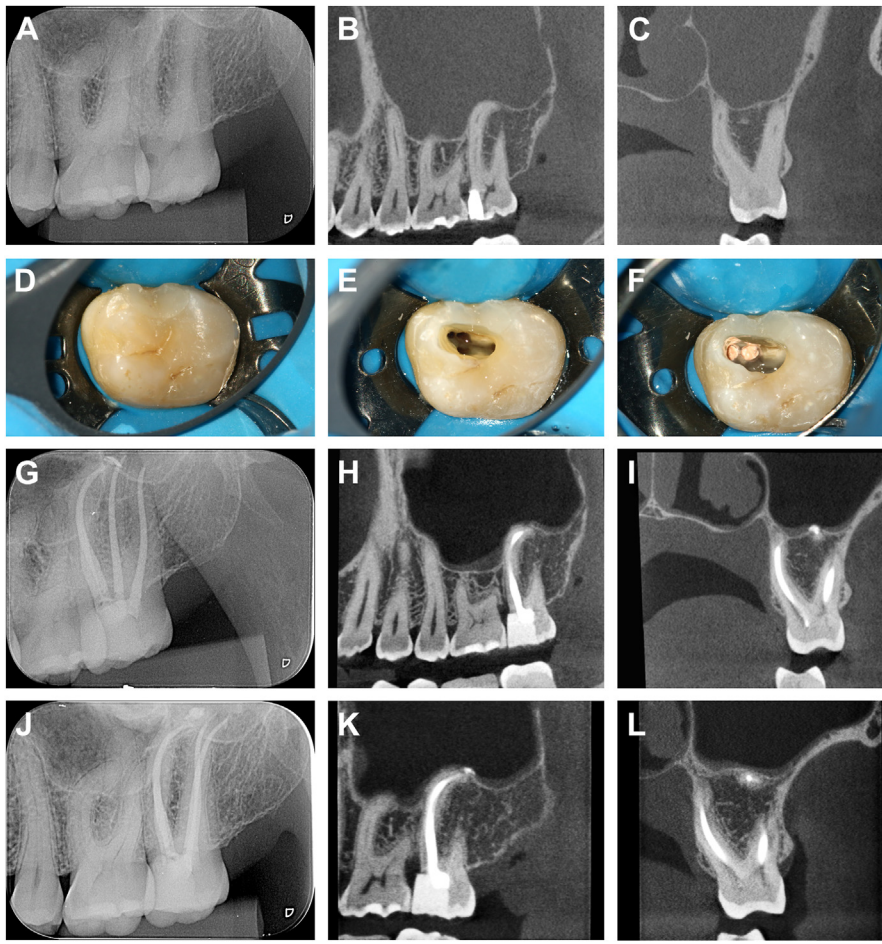

Fig. 3. (*A*) Periapical radiograph, (*B*) sagittal, and (*C*) coronal CBCT showing the left maxillary second molar with a large composite filling and complete opacification of the portion of the maxillary sinus seen. (*D*) Clinical appearance of the upper second molar before root canal treatment, (*E*) after access cavity and root canal preparation, and (*F*) after root canal filling procedures. (*G*) Periapical radiograph and (*H, I*) CBCT taken 1 year after endodontic treatment showing complete root canal filling and resolution of the sinus opacification. (*J*) Periapical radiograph and (*K, L*) CBCT taken 4 years after endodontic treatment showing stable resolution of the sinus opacification and no periapical lesion development. (*Courtesy of* Prof. Dr. Leandro Pereira, Campinas, Brazil.)

led to the diagnosis of pulp necrosis and apical periodontitis in the upper left first molar, along with an acute apical abscess in the neighboring second molar. Note that a CBCT revealed reactive maxillary sinus mucositis (sinus floor mucosal thickening) adjacent to the underlying infected molars. Note there was periapical bone erosion, but there was intact bone between the periapical lesion and sinus mucosa (see **Fig. 4**A–C). To address the endodontic conditions, root canal treatments were performed on both molars in a single appointment without post-procedural systemic antibiotics. The procedure involved the use of 2.5% sodium hypochlorite and 17% ethylenediaminetetraacetic acid for irrigation. Follow-up examinations

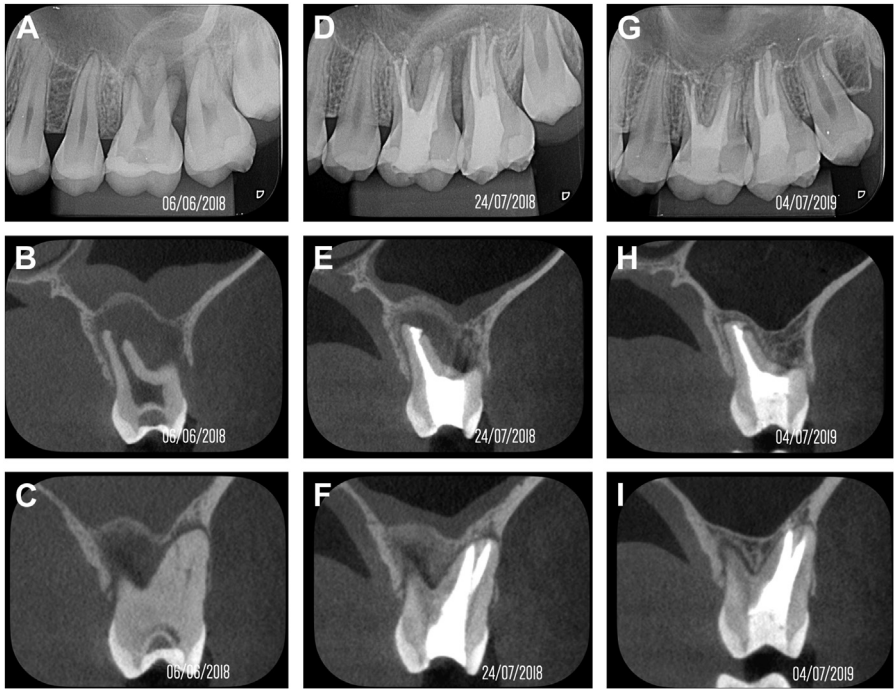

Fig. 4. (*A*) Periapical radiograph, and (*B*, *C*) CBCT showing the left maxillary first and second molars with apical periodontitis and the presence of mucositis in the maxillary sinus (sinus floor reactive mucosal thickening). (*D*) Periapical radiograph and (*E*, *F*) CBCT taken 1 month after endodontic treatment showing partial resolution of apical periodontitis and reduction of the sinus mucosal thickening. (*G*) Periapical radiograph and (*H*, *I*) CBCT taken 10 months after endodontic treatment showing complete resolution of apical periodontitis and the sinus mucosal thickening. Note this was not ODS, but important to know that dental treatment alone is all that is usually necessary in these scenarios. (*Courtesy of* Prof. Dr. Leandro Pereira, Campinas, Brazil.)

were conducted 1 month later to monitor healing progress. These assessments revealed partial improvement in the apical periodontitis and a reduction in the sinus mucosal thickening (see **Fig. 4**D–F). Nearly 1 year posttreatment, a new CBCT demonstrated normal periapical tissues and complete resolution of the maxillary sinus mucositis (see **Fig. 4**G–I).

These cases highlight the potential effectiveness of endodontic interventions in resolving maxillary sinus inflammation and infection due to endodontic infections.

CLINICS CARE POINTS

- Endodontic infection is one of the most common causes of ODS, generally stemming from pulp necrosis leading to apical periodontitis with or without periapical abscess formations.
- Pulpal and periapical infections commonly cause reactive maxillary sinus mucositis, and this is distinct from purulent ODS. Mucositis is represented by mucosal thickening on CT, whereas ODS will more often display partial to complete sinus opacification.

- Effective management of ODS due to endodontic disease benefits from a multidisciplinary approach, focused on accurately diagnosing the endodontic pathology, and eradicating the infection through targeted endodontic treatment, with or without subsequent sinus surgical interventions as needed.

DISCLOSURES

The authors do not have any conflicts of interest or financial disclosures to declare.

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