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Odontogenic Orofacial Infections in Pediatrics: Pathogenesis, Diagnostic Challenges, and Advanced Interdisciplinary Management in Clinical Practice

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Abstract

Background: Odontogenic orofacial infections in children are common, potentially serious conditions originating from dental caries, pulpal disease, or periodontal pathology. Historically linked to high mortality, they remain a frequent reason for emergency department visits. Pediatric patients, often first seen by non-dental clinicians, present unique diagnostic and management challenges.

Aim: This article provides a comprehensive review of the pathogenesis, clinical presentation, and evidence-based management of pediatric odontogenic infections, emphasizing an interdisciplinary approach for optimal outcomes.

Methods: A detailed narrative review synthesizes current literature on the microbial etiology, pathophysiological spread, and risk factors for severe infection. It evaluates diagnostic strategies, including clinical examination and imaging (e.g., panoramic radiograph, CT scan), and analyzes therapeutic protocols from major dental and medical guidelines.

Results: The pathophysiology involves polymicrobial flora spreading from necrotic pulp or periodontal pockets along fascial planes, risking deep neck space infections like Ludwig's angina. High-risk groups include children with diabetes, immunosuppression, or poor access to dental care. Diagnosis hinges on recognizing "red flag" symptoms (trismus, dysphagia, airway compromise). Management prioritizes definitive source control via drainage, endodontic therapy, or extraction. Antibiotics (e.g., amoxicillin) are adjunctive for systemic signs, not a substitute for dental intervention. Analgesia favors NSAID/acetaminophen combinations over opioids.

Conclusion: Successful management requires early recognition, accurate risk stratification, and prompt referral to dental specialists. A coordinated, interprofessional team approach is essential to prevent life-threatening complications and ensure effective treatment.

Keywords: Odontogenic infection, pediatric dentistry, deep neck space infection, dental abscess, antimicrobial stewardship, interdisciplinary management..

Introduction

Dental infections are pathological processes that arise within the tooth or its supporting periodontal apparatus and can subsequently extend into contiguous oral and maxillofacial tissues. When orofacial structures become involved, the infectious nidus typically originates from necrotic dental pulp, deep periodontal pockets, or inflammatory lesions

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associated with partially erupted teeth, such as pericoronitis. Historically, odontogenic infections were highly prevalent and carried a substantial risk of mortality. Archival data from the London Bills of Mortality in the 1600s indicate that infections of dental origin were listed as the fifth or sixth most common cause of death, underscoring their historical clinical significance.[1] Even in the early twentieth century, estimates suggested that odontogenic infections were associated with mortality rates ranging from 10% to 40%.[2] Advances in preventive dentistry, access to professional dental care, improved oral hygiene practices, and the advent of antimicrobial therapy have dramatically altered this landscape, rendering dental infections infrequently life-threatening in contemporary practice. Despite this progress, dental infections remain a frequent cause of presentation in medical environments, particularly in emergency departments. Among all dental-related visits, odontogenic abscesses constitute the predominant clinical entity, reflecting the central role of suppurative processes in advanced dental disease.[3] Patients often seek care in medical rather than dental settings for multiple reasons, including financial limitations, inadequate or absent dental insurance coverage, geographic or logistical barriers to accessing dental services, and high levels of dental anxiety. Misconceptions about the sufficiency of antibiotic therapy alone to resolve dental pathology further contribute to this pattern, as some patients assume that pharmacologic management can substitute for definitive dental intervention.[3]

Consequently, physicians and other non-dental clinicians are frequently required to evaluate and initiate management for dental infections, sometimes in the absence of immediate dental support. This situation is complicated by the fact that many medical practitioners receive limited formal training in oral and maxillofacial pathology, dental anatomy, and odontogenic infection pathways, which can make appropriate diagnosis and treatment planning challenging.[3] Mismanagement or delayed recognition of odontogenic infections may allow progression to deep space involvement, systemic sepsis, or airway compromise, particularly in high-risk patients. At the same time, medical practitioners—especially those in primary care and emergency medicine—occupy a critical position in the early detection and prevention of serious dental complications. They are often the first clinicians to encounter patients with early-stage odontogenic infections or associated systemic symptoms. This provides an important opportunity to recognize evolving dental disease, deliver foundational oral health education, initiate appropriate analgesic and antimicrobial regimens when indicated, and most importantly, facilitate timely referral to dental professionals for definitive treatment, such as drainage, endodontic therapy, or extraction. By integrating basic dental assessment into routine clinical evaluation and fostering collaborative relationships with dental colleagues, medical practitioners can contribute meaningfully to reducing the morbidity associated with oral and facial infections of dental origin and preventing their progression to severe, potentially life-threatening conditions.

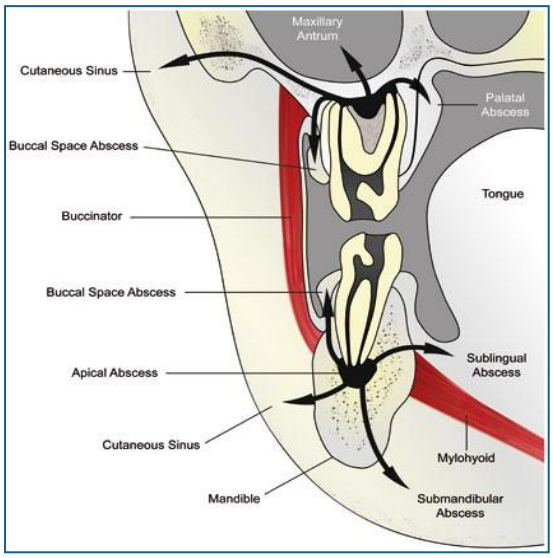

Fig. 1: Facial cutaneous sinuses of dental origin.

Etiology

Odontogenic infections arise from a spectrum of interrelated pathological processes involving the hard and soft tissues of the teeth and their supporting structures. The etiologic pathways typically begin with dental caries, progress through pulpal and periapical disease, and extend to periodontal involvement, with each stage representing a continuum of microbial, host, and environmental interactions. Understanding these mechanisms is essential for recognizing early disease, preventing progression to serious orofacial infections, and guiding appropriate referral and intervention. Dental caries represent the primary initiating factor for many odontogenic infections. Carious lesions develop through a dynamic interaction between susceptible tooth surfaces, microbial biofilm (dental plaque), and dietary fermentable carbohydrates, within the modifying context of salivary composition and flow, as well as individual genetic predisposition.[4] The dental biofilm is a structured microbial community adherent to the tooth surface, embedded within an extracellular matrix. When fermentable carbohydrates such as glucose, fructose, sucrose, and maltose are ingested, bacteria within this biofilm rapidly metabolize these substrates, producing organic acids, most notably lactic acid. The accumulation of these acids at the tooth surface leads to a transient lowering of pH within the plaque environment. When the pH drops below a critical threshold, mineral components—primarily calcium and phosphate—begin to dissolve from the hydroxyapatite crystals of enamel, initiating the process of demineralization.[4] Under physiologic

conditions, this demineralizing phase is counterbalanced by protective salivary mechanisms. As dietary sugars are cleared through swallowing, salivary dilution, and oral clearance, salivary buffering systems gradually neutralize the acidic environment, allowing the plaque pH to return toward neutrality. At this stage, saliva serves not only as a buffer but also as a reservoir of calcium, phosphate, and fluoride ions. These ions diffuse into the demineralized enamel and promote remineralization, thereby repairing early mineral loss and restoring enamel integrity.[4] This oscillating cycle of demineralization and remineralization occurs with each exposure to fermentable carbohydrates and, in a balanced state, may not result in clinically evident damage.

However, when this equilibrium is disrupted—for example, by frequent carbohydrate intake, prolonged exposure to sugary or acidic beverages, or impaired salivary flow—the conditions within the plaque microenvironment become persistently acidic. Reduced salivary flow, whether due to systemic disease, medications, or radiation therapy, diminishes both buffering capacity and mechanical clearance of food debris and bacteria, thereby increasing caries susceptibility. Repeated or continuous acid challenges favor the selection of more acidogenic and aciduric bacterial species within the biofilm, rendering it increasingly cariogenic.[4] When mineral loss chronically exceeds mineral replacement, the earliest clinically detectable manifestation is the so-called “white spot” lesion, or incipient carious lesion, characterized by subsurface enamel demineralization that remains intact on the surface. These white spot lesions are of particular etiologic and clinical significance because they represent a reversible stage of disease. If identified early—often by clinicians in medical or dental settings during routine examinations—such lesions can be managed noninvasively through targeted remineralization strategies and behavioral modifications. Topical fluoride therapies, improved oral hygiene practices, and reduction in frequency of sugar intake can halt or reverse the demineralization process, restoring enamel hardness and preventing cavitation.[4] Failure to intervene at this stage allows continued mineral loss, leading to enamel microcavities that eventually coalesce and collapse into overt cavitated lesions, creating niches that facilitate deeper bacterial invasion and subsequent pulpal involvement. Once caries progression breaches enamel and dentin to approximate or enter the pulp chamber, pulpal pathology ensues. Pulpitis, defined as inflammation of the dental pulp, may be triggered by bacterial toxins and metabolic by-products diffusing through dentinal tubules from carious lesions, by direct bacterial invasion through an existing restoration or tooth fracture, or by extension from a periodontal pocket via the apical foramen.[5] Non-microbial insults such as thermal, mechanical, or chemical irritation from restorative procedures can also precipitate pulpal inflammation.[6]

Clinically, the pulpal inflammatory response evolves from a reversible to an irreversible process. Reversible pulpitis is characterized by transient, mild to moderate pain often provoked by thermal stimuli, particularly cold, and relieved promptly upon removal of the stimulus. At this stage, the pulp retains its vitality and capacity for repair, and conservative treatment aimed at eliminating the irritant—most commonly removal of carious tissue and placement of an appropriate restoration—can resolve the inflammation.[6] In contrast, irreversible pulpitis reflects a sustained inflammatory state in which pulpal damage has progressed beyond the capacity for self-repair. Patients typically experience spontaneous, persistent, and often poorly localized pain, which may be exacerbated by thermal stimuli but no longer subsides quickly. This type of pain is frequently severe and is one of the most common reasons patients seek urgent or emergency care.[7] In irreversible pulpitis, the standard of care is endodontic intervention involving partial or complete removal of inflamed pulp tissue (pulpotomy or pulpectomy) and subsequent root canal therapy, or extraction if the tooth cannot be restored.[6] Importantly, antibiotics do not have a therapeutic role in the management of irreversible pulpitis in the absence of overt infection. Clinical signs such as localized swelling, purulence, or systemic symptoms are typically absent in this condition.[7] The underlying pathology is confined to inflamed but not yet infected pulp tissue, and pain arises from increased intra-pulpal pressure and nerve sensitization rather than from active spreading infection. Evidence indicates that systemic antibiotics do not significantly reduce pain or decrease analgesic requirements in patients with untreated teeth affected by irreversible pulpitis.[8] Hence, antimicrobial therapy should not be initiated solely for pulpal pain but reserved for situations where infection has clearly extended beyond the tooth into surrounding tissues. Without definitive treatment, irreversible pulpitis may progress to pulpal ischemia and necrosis. Paradoxically, once the pulp becomes necrotic, the tooth may become asymptomatic despite ongoing microbial colonization of the root canal system.

Pulp necrosis sets the stage for periapical pathology, including the development of an apical abscess. Terms such as apical abscess, dentoalveolar abscess, and odontogenic abscess are often used interchangeably to describe a localized collection of pus at or around the apex of a non-vital tooth root.[9] This occurs when bacteria and their products traverse the apical foramen, inciting an inflammatory reaction in the periapical tissues. Depending on host response and microbial virulence, acute or chronic forms of periapical abscess may develop. Acute apical

abscesses present with rapid onset of pain and swelling. The affected tooth is typically extremely tender to percussion or palpation, reflecting periapical inflammation and pressure.[9] Swelling is most commonly intraoral initially but may extend extraorally as the infection progresses. Systemic manifestations such as fever, malaise, and regional lymphadenopathy can accompany acute apical abscesses, particularly when the infection is more advanced. Anatomic location influences the pattern of spread. In the maxilla, acute apical abscesses usually drain through the buccal or palatal bone into the buccal vestibule or adjacent soft tissues. In the mandible, drainage patterns can be more variable: infection may perforate the buccal or lingual cortical plates and drain intraorally, or it may extend into deeper fascial spaces of the neck, leading to cellulitis and potential airway compromise.[9] Mandibular molar infections are of particular concern because their root apices lie in close proximity to the sublingual, submandibular, and submental spaces. Spread of infection into these interconnected compartments can result in Ludwig angina, a rapidly progressive, bilateral cellulitis of the submandibular space. This condition can elevate and displace the tongue posteriorly, leading to airway obstruction and posing an immediate threat to life if not promptly recognized and treated.[10] Thus, what begins as a localized periapical process can, in the absence of early intervention, evolve into a severe deep neck space infection.

In its uncomplicated form, however, an apical abscess remains a localized infection that does not automatically warrant antimicrobial therapy prior to definitive dental management.[5] The principal treatment is surgical: drainage of purulent material through the root canal system via endodontic access, incision and drainage of fluctuant soft tissue collections when present, and eventual root canal therapy to disinfect and seal the canal system, thereby preserving the tooth. When the tooth is non-restorable or the patient's circumstances preclude endodontic therapy, extraction eliminates the source of infection. Antibiotics are reserved for cases where there is evidence of systemic involvement or spreading infection, such as fever, diffuse facial swelling, or cellulitis.[5] In such situations, broad-spectrum coverage targeting common odontogenic pathogens is indicated, with amoxicillin/clavulanate frequently used as a first-line agent due to its expanded spectrum against β-lactamase–producing organisms.[11] Alongside caries and pulpal pathology, periodontal disease constitutes a major etiologic pathway for oral infections of dental origin. Periodontal infections typically begin as gingivitis, a reversible inflammatory condition of the gingival tissues provoked by plaque accumulation at the gingival margin. If plaque is not disrupted by effective oral hygiene, it mineralizes into calculus, which further facilitates plaque retention. In susceptible individuals, this process progresses to periodontitis, characterized by irreversible destruction of the periodontal ligament, alveolar bone resorption, and formation of periodontal pockets.[12]

The pathogenesis of periodontitis is multifactorial. While bacterial biofilm is the initiating factor, the clinical course and severity of periodontal disease are largely determined by the host's inflammatory and immune response, modulated by environmental, systemic, and genetic factors.[12][13] Tobacco smoking is one of the most important modifiable risk factors, associated with increased prevalence, severity, and progression of periodontitis. Smoking alters vascular and immune responses, impairs neutrophil function, and affects the composition of the subgingival microbiota, cumulatively exacerbating tissue destruction. Diabetes mellitus is another major risk factor, with a bidirectional relationship: poorly controlled diabetes increases susceptibility to periodontitis, while severe periodontal disease may adversely affect glycemic control. Hyperglycemia leads to formation of advanced glycation end-products, altered collagen metabolism, and dysregulated inflammatory mediator production, all of which contribute to periodontal breakdown.[12][13] Other systemic conditions, medications causing xerostomia, psychosocial stress, and certain genetic polymorphisms further modify an individual's risk profile. The net result is that two individuals with similar plaque levels may exhibit markedly different periodontal outcomes, reflecting host susceptibility. Left untreated, periodontitis can lead to tooth mobility, tooth loss, and chronic inflammatory burden that may serve as a nidus for recurrent orofacial infection. Deep periodontal pockets can communicate with the pulpal space or periapical region through accessory canals or apical foramina, creating combined endodontic-periodontal lesions and additional pathways for infection spread. In summary, the etiology of oral and facial infection of dental origin is rooted in a continuum from dental caries and pulpal disease to apical abscess formation and periodontal breakdown, shaped by microbial, host, and environmental factors. Early recognition of these processes—whether through detection of white spot lesions, diagnosis of reversible or irreversible pulpitis, identification of apical abscesses, or assessment of periodontal status—provides critical opportunities for intervention, prevention of progression, and reduction of the risk of severe odontogenic infections with systemic implications.[4][5][6][7][8][9][10][11][12][13]

Epidemiology

Oral and facial infections of dental origin are highly prevalent worldwide and represent a substantial public health burden, with significant clinical and socioeconomic implications. In contemporary populations, dental infections and toothache remain common reasons for seeking both dental and medical care. Data suggest that

approximately 13% of adults pursue professional evaluation for dental infections or tooth pain within a 4-year period, illustrating the persistent frequency of odontogenic pathology in the general population.[14][15] Although many such presentations are managed in outpatient dental settings, a measurable subset progress to severity requiring hospital-level care. In the United States, it is estimated that approximately 1 in 2,600 individuals will require hospitalization as a direct consequence of dental infection, reflecting the potential for seemingly localized pathology to escalate into serious systemic illness.[14][15] The underlying disease burden that predisposes to odontogenic infections is considerable. More than 20% of the population lives with untreated dental caries, while nearly three-quarters of adults have undergone at least one dental restoration, signifying previous carious disease.[16][17] Periodontitis likewise affects a large segment of the population, with epidemiologic studies estimating a prevalence of approximately 35% among Americans aged 30 to 90 years, highlighting that a substantial proportion of adults experience chronic inflammatory destruction of periodontal tissues.[16] These conditions—caries and periodontitis—are the principal precursors of dental abscesses, linking their high prevalence to the frequent occurrence of odontogenic infections.

Socioeconomic status exerts a profound influence on the distribution of dental disease and, consequently, on the risk of odontogenic infection. Individuals residing at or below 100% of the federal poverty level experience untreated dental caries at rates exceeding 41.9%, which is more than 2.5 times higher than the 16.6% observed among those living at or above 200% of the federal poverty threshold.[17] This disparity underscores the role of structural inequities in access to preventive and restorative dental services. Overall, dental caries affect approximately 90% of adults and 42% of children between 6 and 19 years of age, with the prevalence of caries experience remaining relatively high and stable across age groups.[17] Importantly, adolescents aged 12 to 19 tend to have lower rates of untreated caries than younger children aged 5 to 11, likely reflecting differences in access to care, exposure to preventive interventions such as sealants and fluoride, and evolving oral health policies targeting school-aged populations.[17] Certain clinical populations are at particularly elevated risk for developing odontogenic abscesses. Individuals with type 1 or type 2 diabetes mellitus demonstrate a significantly greater prevalence and severity of dental abscesses compared with nondiabetic controls, even after adjusting for comorbid periodontal and cardiovascular disease.[18][19][20] Hyperglycemia and impaired host immune response in diabetes predispose to both caries and periodontitis, which in turn provide the substrate for pulpal necrosis and apical abscess formation. Similarly, patients with periodontal disease are independently predisposed to acute periapical abscesses. Studies have reported substantially increased odds ratios for periapical abscess among those with advanced periodontitis, even after controlling for diabetes status and tobacco use, supporting a strong link between chronic periodontal breakdown and acute odontogenic infections.[18][19][20]

Obesity has also emerged as a risk marker for odontogenic abscesses. Obese individuals exhibit a higher prevalence of periapical abscess compared with individuals of normal weight, and this association remains statistically significant even after adjusting for confounding factors such as diabetes, smoking, and periodontal status.[21][22] The mechanisms may involve systemic low-grade inflammation, altered immune function, and dietary patterns that promote caries formation. Older adults constitute another high-risk group, due to the convergence of multiple predisposing factors: higher cumulative exposure to caries and periodontal disease, increased rates of systemic comorbidities, polypharmacy including medications that cause xerostomia, functional limitations that impair oral hygiene, and logistical or financial barriers to accessing dental care. Beyond clinical comorbidities, a complex network of social determinants of health strongly shapes the epidemiology of odontogenic infections. Individuals with poor oral hygiene practices, low socioeconomic status, tobacco use, harmful alcohol consumption, or limited contact with dental services are more likely to develop both caries and periodontal disease, and thus face a higher risk of progressing to dental abscesses.[21][22] Populations at increased risk specifically due to socioeconomic constraints include those living in poverty, individuals without dental insurance or those reliant on public insurance, people experiencing homelessness, residents of rural or urban underserved areas, and communities with documented shortages of dental professionals. For these groups, affordability, availability, and accessibility of dental services are substantial barriers, leading to high rates of untreated disease.

Racial and ethnic disparities further compound these inequities. Minority populations—including Asian, Black, Hispanic/Latino, Native American/Alaska Native, and Native Hawaiian/Pacific Islander groups—often experience higher burdens of untreated dental caries and periodontitis, reflecting broader patterns of healthcare inequity, discrimination, and unequal access to preventive and restorative care. Adults with physical, cognitive, or developmental disabilities, and individuals living in institutional settings such as long-term care facilities or correctional institutions, also experience elevated risk, owing to dependence on caregivers for oral hygiene, limited autonomy in

seeking dental services, and competing healthcare priorities. The United States Preventive Services Task Force has emphasized that these patterns of disparity are principally driven by social determinants of health, including low income, lack of health and dental insurance, and geographic isolation, all of which restrict access to preventive and restorative dental services and increase the risk of severe oral infections.[23] Economic pressures, including cost-of-living crises and food insecurity, may further worsen oral health by limiting the ability to purchase toothbrushes, toothpaste, and fluoridated products, while simultaneously increasing reliance on inexpensive, highly processed foods rich in fermentable sugars. This combination amplifies the risk of both caries and periodontal disease and, by extension, odontogenic abscess formation.

Socioeconomic gradients in dental health are consistently observed across populations. Lower levels of education, reduced income, and lower occupational status correlate strongly with a higher prevalence of dental problems and diminished utilization of dental care, even after controlling for behavioral and psychosocial variables.[24][25][26] These gradients highlight that individual behaviors alone cannot account for the observed patterns; structural factors such as healthcare coverage, neighborhood resources, transportation, and culturally appropriate services play a central role. Additional high-risk groups for dental abscesses include individuals with a history of dental trauma, failed or incomplete endodontic treatment, or existing complex restorations that predispose to recurrent infection. Patients with underlying immunocompromising conditions—such as HIV infection, malignancy, organ transplantation, or long-term corticosteroid or immunosuppressive therapy—are particularly susceptible to rapid progression of odontogenic infections and may experience more severe systemic sequelae. Some epidemiologic analyses have also linked specific demographic factors, including female sex and African American race in certain cohorts, to higher observed prevalence of dental abscesses, though these patterns often intersect with broader socioeconomic and access-related disparities. Taken together, the highest-risk groups for oral and facial infections of dental origin include individuals with diabetes mellitus, significant periodontal disease, obesity, and advanced age, particularly when these factors coexist with poor oral hygiene, tobacco use, alcohol misuse, or limited access to dental care. In these populations, the cumulative effect of biological vulnerability and social disadvantage produces a disproportionate burden of disease. Recognizing these epidemiologic patterns is crucial for clinicians and public health planners, as it underscores the need for targeted screening, preventive interventions, and improved access to dental and medical services. Early identification of dental disease, timely management of caries and periodontal pathology, and strategic outreach to vulnerable communities are key to reducing the incidence of odontogenic abscesses and mitigating their impact on overall health and healthcare systems.[14][15][16][17][18][19].

Pathophysiology

The pathophysiology of odontogenic infections reflects a dynamic interplay between microbial colonization, host immune responses, and the anatomical structure of the teeth and surrounding fascial planes. These infections typically originate within the dental pulp or periodontal tissues and evolve according to the balance between bacterial virulence factors and host defenses. While many infections remain localized when treated promptly, disruptions in this balance can allow the infection to spread beyond its initial confines, posing significant risks to adjacent anatomical spaces and systemic health. Odontogenic infections are most commonly polymicrobial, dominated by a mixture of aerobic and anaerobic organisms found within the normal oral microbiota. Caries-induced pulp exposure or periodontal pocketing provides a portal for bacterial invasion into deeper tissues. Within the pulp chamber, restricted vascular supply limits the tissue's ability to mount an effective immune response. As bacteria proliferate, the confined environment results in elevation of intrapulpal pressure, vascular collapse, and tissue ischemia. This ischemic milieu accelerates pulpal necrosis, transitioning the environment from inflamed but vital pulp to a necrotic, bacteria-rich space ideal for anaerobic proliferation. Once necrosis occurs, microorganisms and their toxic metabolic by-products can migrate through the apical foramen into the periapical tissues. The host responds with an inflammatory cascade involving neutrophils, macrophages, and cytokines. Neutrophil infiltration leads to liquefaction necrosis and the accumulation of purulent exudate—hallmarks of an acute apical abscess. The expanding collection of pus increases periapical pressure, causing severe pain and promoting cortical bone erosion. Once bone is breached, infection tracks along the path of least resistance, guided by muscular attachments and fascial planes [18][19[20].

The pattern of spread varies with tooth position and anatomical relationships. Maxillary infections may erode through the thin buccal or palatal cortical plates, leading to vestibular abscesses, canine space infections, or orbital complications. Mandibular infections have a greater tendency to spread into deep fascial spaces due to thicker cortical bone and proximity to major anatomical compartments. Infection originating from mandibular molars may extend into the submandibular, sublingual, or submental spaces. When these spaces become simultaneously involved, the condition can progress to Ludwig angina, a rapidly spreading cellulitis characterized by bilateral swelling, elevation of the tongue, and impending airway obstruction.

Infections that extend deeper into the neck can involve the parapharyngeal, retropharyngeal, or danger spaces. These regions have direct continuity with the mediastinum, providing a pathway for potentially fatal descending necrotizing mediastinitis. Hematogenous spread of microorganisms from untreated odontogenic infections can also precipitate systemic sepsis or metastatic infections such as cavernous sinus thrombosis or brain abscesses. The host immune response plays a key role in determining infection severity. Although inflammation initially serves a protective function, excessive or dysregulated immune activity contributes to tissue destruction, edema, and increased intratissue pressure. Immunocompromised individuals—including those with diabetes, HIV, malignancy, or medication-induced immunosuppression—are at heightened risk for rapidly progressive infections due to impaired leukocyte function and diminished vascular response [18][19][20].

The pathophysiologic process is further influenced by the bacterial species involved. Facultative anaerobes initiate the infection, consuming available oxygen and creating a hypoxic environment conducive to the proliferation of obligate anaerobes such as *Prevotella*, *Fusobacterium*, and *Porphyromonas* species. These anaerobes produce proteolytic enzymes, collagenases, hyaluronidases, and other virulence factors that degrade connective tissue, facilitate spread along fascial planes, and exacerbate regional inflammation. Overall, the pathophysiology of dental infections illustrates how a localized microbial insult can, in the absence of timely intervention, progress into a severe and potentially life-threatening condition. Early recognition of the anatomical pathways of spread and the clinical signs of deep or systemic involvement is crucial. Prompt dental or surgical management—including drainage, removal of the infectious nidus, and appropriate antimicrobial therapy—remains essential to prevent complications such as deep neck space infections, airway compromise, or septicemia [16][17][20].

History and Physical

Assessment of oral and facial infection of dental origin begins with careful clinical observation of dental caries and pulpal status. Carious lesions may present along a spectrum from acute, rapidly progressing destruction to more chronic, slowly evolving defects. Clinically, caries can appear as brown-yellow soft cavities, indicative of active demineralization, or as black, hard lesions that may represent arrested or long-standing disease. In advanced cases, frank pulp exposure may be visible within the cavity when the carious process has penetrated enamel and dentin. Early carious changes are more subtle. Incipient caries represent the first clinical manifestation and often pose a diagnostic challenge, especially for clinicians less accustomed to oral examination. These lesions typically appear as white, opaque, irregular spots, reflecting subsurface enamel demineralization. They tend to occur in areas of plaque retention, such as along the gingival margin or at stagnation sites where cleansing is inadequate, and can be easily overlooked without deliberate inspection. The patient's symptomatology provides important insight into the state of the dental pulp. In reversible pulpitis, individuals usually report toothache that is clearly stimulus-dependent, triggered by hot or cold food and beverages or occasionally by sweet substances. The pain is typically transient, resolving shortly after the stimulus is removed, and is often localized by the patient to a specific tooth. In contrast, irreversible pulpitis is characterized by spontaneous, often severe and throbbing pain that may be poorly localized and frequently worsens at night, disrupting sleep. The pain is less clearly associated with an external trigger and may linger or intensify following thermal challenge. As disease progresses, periapical involvement may result in a localized swelling adjacent to the affected tooth, and a periapical abscess may become palpable either intraorally or, in more advanced cases, extraorally. A dental abscess may originate from pulpal infection, periodontal structures, or a combination of both, producing complex endodontic–periodontal involvement. More serious clinical manifestations, including fever, facial edema, trismus, dysphagia, and dysphonia, signal the possibility that infection has extended beyond the confines of the alveolar bone into the deep fascial spaces of the neck. When this occurs, patients may present in respiratory distress or with hemodynamic instability and sepsis, necessitating urgent intervention.[27]

Periodontal disease contributes significantly to the clinical spectrum of odontogenic infections. Patients with gingivitis or periodontitis frequently describe persistent halitosis and report bleeding during toothbrushing or flossing. In many cases, pain is not a prominent symptom, which may delay presentation and contribute to disease progression, as asymptomatic patients do not perceive an urgent need for care. One notable exception is necrotizing periodontal disease, in which intense pain, spontaneous bleeding, and ulcerative lesions of the gingiva are key features. Gingivitis itself is defined as localized inflammation of the gingival tissues primarily initiated by the accumulation of dental plaque at the tooth surface.[28] Clinically, the gums appear swollen, erythematous, and prone to bleeding on gentle probing or brushing. Importantly, gingivitis is not associated with attachment loss; the gingival sulcus depth remains within normal limits, and there is no radiographic evidence of alveolar bone loss.[29] Once gingival inflammation progresses to involve the deeper periodontal apparatus, periodontitis develops.

This condition is characterized by apical migration of the junctional epithelium and formation of periodontal pockets, which harbor pathogenic biofilm and perpetuate inflammation. Over time, clinical signs may include gingival recession, papillary blunting or detachment, tooth mobility, and fremitus, with radiographs revealing varying degrees of alveolar bone resorption.[30] The extent and pattern of destruction are influenced by individual risk factors such as smoking, diabetes, genetic susceptibility, and oral hygiene behaviors. These changes create an environment in which pathogens can access the root surface and, in advanced disease, potentially communicate with the pulpal space, thereby increasing the risk of abscess formation.

Pericoronitis represents a distinct clinical entity and an important cause of odontogenic infection. It involves inflammation of the soft tissue surrounding the crown of a partially erupted tooth, most commonly the mandibular third molars. Food debris and bacteria accumulate beneath the pericoronal flap (operculum), leading to localized pain, swelling, and difficulty in mastication. In mild cases, symptoms remain localized; however, in more severe presentations, pericoronitis can be a portal for spread to adjacent fascial spaces, contributing to significant facial swelling, trismus, and systemic signs of infection. Because of its anatomic location, infection originating from mandibular third molars can readily extend into the submandibular, submasseteric, or pterygomandibular spaces, with potentially serious consequences. A thorough and focused clinical history is crucial for understanding the nature, extent, and potential severity of an odontogenic infection. The history of present illness should begin with a clear articulation of the chief complaint, followed by a detailed characterization of pain, including precise location, onset, progression, duration, quality (sharp, throbbing, dull), radiation, and factors that exacerbate or alleviate symptoms. Inquiry regarding swelling should address when it was first noticed, its rate of progression, associated tenderness, and whether the patient perceives fluctuation or firmness. The presence of systemic symptoms such as fever, malaise, or chills may indicate that the infection has progressed beyond a localized process. Symptoms such as difficulty chewing, dysphagia, slurred or muffled speech, trismus, foul taste, or persistent bad breath may signal accumulating pus, fistula formation, or deep space involvement [27][29][30].

The past dental history should document recent dental procedures—such as extractions, root canal therapy, restorations, or trauma—that may predispose to infection. A history of recurrent dental infections, untreated caries, or partially erupted third molars should be specifically sought. The medical history must include systemic conditions known to affect host response, particularly diabetes mellitus and other causes of immunosuppression, including HIV infection, malignancy, systemic corticosteroid use, or chemotherapy. A history of previous facial cellulitis, abscess formation, or deep neck infection is also significant, as it may signal an underlying odontogenic source or structural vulnerability. Physical examination begins with a careful extraoral evaluation. The clinician should inspect the face and neck for asymmetry, swelling, erythema, and areas of induration or fluctuance. Palpation may reveal tender, warm, or fluctuant regions consistent with abscess, or firm, indurated areas suggestive of cellulitis. Palpation of regional lymph nodes, particularly in the submandibular and cervical chains, can reveal lymphadenopathy, which often accompanies more advanced or systemic involvement. Signs of fascial space infection, such as fullness in the submandibular, buccal, or submental regions, may be apparent even before overt airway symptoms occur. Airway evaluation is paramount, particularly in patients with facial swelling or systemic signs of infection. The clinician should listen for voice changes, such as a “hot potato” voice, that may suggest oropharyngeal or supraglottic involvement, and observe for drooling or difficulty handling oral secretions. The presence of stridor, tachypnea, use of accessory muscles, or any respiratory distress must be regarded as an ominous sign of potential airway compromise, requiring prompt escalation of care. Intraoral examination should be systematic and thorough. The dentition should be inspected for visible caries, defective restorations, fractures, discoloration, or increased mobility of individual teeth. Percussion and palpation of suspect teeth can help localize periapical tenderness. The gingiva should be evaluated for erythema, edema, bleeding, recession, and the presence of periodontal pockets or gingival abscesses. The vestibules, floor of the mouth, and palate should be examined for swelling, induration, or fluctuance, as well as for draining sinus tracts or fistulae that may indicate chronic abscess drainage. In cases of pericoronitis, an operculum overlying a partially erupted tooth, often with erythema, tenderness, and accumulation of debris, may be observed [30].

Measurement of mouth opening, typically by assessing the interincisal distance, is important for detecting trismus, which may signal involvement of the masticator or pterygomandibular spaces. Clinicians must also distinguish between cellulitis, in which tissues feel firm and indurated, and abscess, characterized by localized fluctuance. This distinction influences both diagnostic imaging and the decision to pursue surgical drainage. Risk stratification is an essential component of the history and physical examination, guiding decisions about outpatient management versus urgent referral or hospitalization. Mild to moderate infections are generally localized, with pain and limited swelling, no systemic symptoms, and preserved ability to eat, drink, and maintain airway function. These cases often permit

dental referral and outpatient management if reliable follow-up is ensured. By contrast, severe infections are distinguished by "red flag" signs, including systemic toxicity—such as fever exceeding 38.5 °C, tachycardia, or hypotension—combined with evidence of deep fascial space involvement, rapidly spreading cellulitis, or significant trismus. Dysphagia, odynophagia, drooling, dysphonia, and any sign of respiratory compromise are particularly concerning. Immunocompromised patients, even with apparently modest swelling, warrant a lower threshold for aggressive management. In such contexts, timely recognition based on meticulous history and physical examination is critical to preventing progression to airway obstruction, mediastinal spread, or systemic sepsis, and to initiating appropriate surgical and medical intervention without delay.[27][28][29][30]

Evaluation

The evaluation of a patient presenting with an odontogenic infection requires a structured, methodical, and time-sensitive approach, as these infections possess the capacity to progress rapidly and unpredictably. While many dental infections remain localized, others can disseminate along the interconnected fascial planes of the head and neck, leading to deep space involvement, airway obstruction, or systemic sepsis. For this reason, clinicians—particularly those in emergency, urgent care, and primary care settings—must be equipped to identify the severity of the infection, differentiate uncomplicated dental disease from more serious pathology, and determine when immediate intervention or referral is necessary.[31] A comprehensive assessment rests on integrating findings from patient history, physical examination, appropriate imaging, and targeted laboratory investigations. The initial evaluation begins with a focused history and physical assessment that aims to determine the origin and extent of the infection. Clinicians should inquire about the evolution of symptoms, including the onset and progression of pain, swelling, difficulty chewing, drooling, fever, or trismus. Particular attention should be given to symptoms suggestive of deep neck space involvement, such as dysphagia, odynophagia, restricted mouth opening, voice changes, dyspnea, or inability to handle secretions. These features warrant heightened concern for impending airway compromise. Additionally, the patient's medical history—particularly the presence of diabetes, immunosuppressive conditions, prior infections, or recent dental interventions—helps identify those at elevated risk for rapid disease progression [31].

Physical examination is equally critical and should begin with an extraoral assessment, including inspection and palpation of facial symmetry, swelling, erythema, lymphadenopathy, and fluctuation or induration. Airway evaluation is mandatory in all patients with significant swelling. Intraoral examination should identify signs of active dental disease such as caries, pulp exposure, periodontal abscesses, pericoronal inflammation, pus discharge, draining fistulae, or localized vestibular swelling. Examination of the floor of the mouth and posterior oropharynx is essential, as elevation of the tongue or swelling encroaching upon the airway may indicate early Ludwig angina or parapharyngeal extension. Diagnostic imaging plays a pivotal role when the infection is suspected to extend beyond the alveolar bone or when there is clinical uncertainty about the presence of abscess formation. A panoramic radiograph (orthopantomogram) is often the first-line modality in dental settings, as it provides a broad view of the teeth, jaws, and adjacent structures, allowing for detection of periapical radiolucencies, dental caries, impacted third molars, or other structural abnormalities. Periapical radiographs offer more detailed imaging of individual teeth, assisting in the diagnosis of localized apical pathology. However, in medical settings where deep-space infection or extensive swelling is suspected, contrast-enhanced computed tomography (CT) of the neck and face is considered the gold standard. CT imaging allows for precise differentiation between cellulitis and abscess, identification of fascial space involvement, assessment of airway narrowing, and recognition of complications such as mediastinal spread. CT is particularly valuable in patients with severe trismus or extensive extraoral swelling, as it provides superior visualization of deep structures [31].

Ultrasound serves as an additional imaging tool for evaluating superficial soft-tissue abscesses, especially in the buccal, submandibular, or perioral regions. It is noninvasive, portable, and avoids radiation exposure, making it suitable for bedside assessment. However, its utility is limited in deeper or more complex infections. Laboratory investigations are generally reserved for patients with moderate to severe infections, systemic symptoms, or suspected complications. A complete blood count (CBC) is useful for detecting leukocytosis or other abnormalities in white blood cell count that may indicate systemic response to infection. Inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) help gauge the degree of systemic inflammation and are often elevated in deep space infections. Blood cultures should be obtained when sepsis is suspected or when the patient presents with high-grade fever, hypotension, or unexplained systemic toxicity. Because hyperglycemia is a known contributor to poor outcomes, delayed healing, and increased infection severity, targeted evaluation of blood glucose levels is essential, particularly in patients with known diabetes or clinical signs suggestive of previously undiagnosed insulin resistance. In

summary, the evaluation of dental infections requires more than simple inspection of the oral cavity. It demands a nuanced approach that combines detailed history-taking, meticulous physical examination, strategic use of imaging modalities, and targeted laboratory studies. Early and accurate identification of high-risk features enables timely intervention and reduces the likelihood of progression to serious complications such as deep neck space infections, airway compromise, or systemic sepsis. Such a comprehensive evaluative framework ensures that clinicians can differentiate routine dental pathology from true medical emergencies, facilitating safe and effective patient care [30][31].

Treatment / Management

Management of oral and facial infections of dental origin requires a careful, stepwise approach that addresses both the acute clinical presentation and the underlying source of infection. Patients frequently present to emergency departments or primary care settings with acute dental pain, most commonly arising from pulpitis or an acute periapical abscess. The clinician's initial responsibility is to rapidly determine whether the process remains localized within the oral cavity or has extended into deep fascial spaces or produced systemic illness. This distinction is crucial, as odontogenic infections have the potential to spread through contiguous tissue planes, resulting in airway compromise, mediastinal extension, or sepsis if not promptly recognized and managed.[32] At the onset of evaluation, particular attention must be directed to the airway. Any suspicion of impending compromise mandates immediate escalation of care to a facility capable of advanced airway management, with early notification of oral and maxillofacial surgery, anesthesiology, or otolaryngology teams. Clinical features that raise concern for advanced or rapidly spreading infection include significant trismus, dysphagia, drooling, reduced tongue mobility, swelling or firmness of the floor of the mouth, and the classic "hot potato" voice, all of which suggest deep space involvement and potential Ludwig angina.[33] Patients with Ludwig angina represent a true maxillofacial emergency: they should be admitted urgently, undergo airway stabilization—often requiring awake fiberoptic intubation or tracheostomy in severe cases—and receive broad-spectrum intravenous antibiotic therapy alongside surgical drainage where indicated.[33]

In the absence of red flag signs or systemic instability, many odontogenic infections may be managed on an outpatient basis; however, referral to a dental practitioner for definitive care remains mandatory.[32] The central tenet of treatment is removal or neutralization of the infection's source rather than reliance on pharmacologic therapy alone. Only dental professionals possess the training and scope of practice to perform procedures such as root canal therapy, surgical drainage, or tooth extraction, which are the definitive interventions in these conditions.[32]

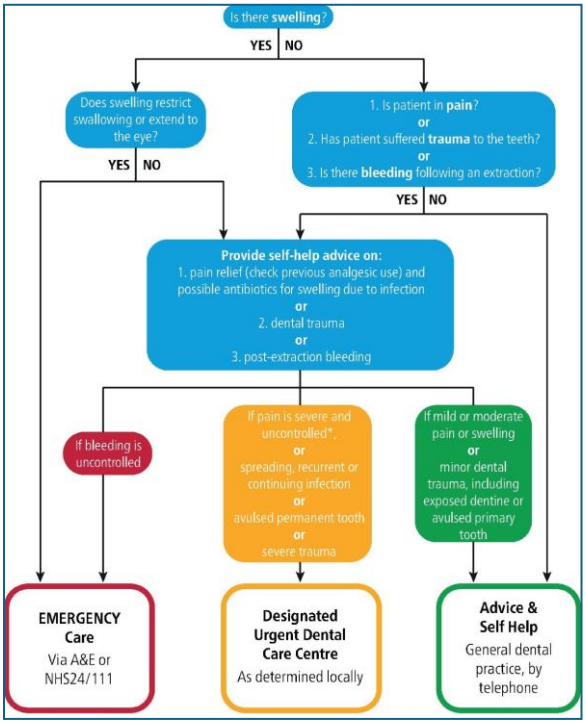

Fig. 2: Management of orofacial infections.

In cases of irreversible pulpitis, root canal therapy is considered the treatment of choice, as it removes inflamed or necrotic pulp tissue, disinfects the root canal system, and allows the tooth to be preserved. For an acute periapical abscess, the initial management generally consists of establishing drainage of purulent material, either by creating access through the occlusal surface into the pulp chamber or via incision and drainage when soft-tissue fluctuation is present. Subsequently, the tooth may undergo root canal treatment to eradicate infection and restore function; if the tooth is structurally unsalvageable or the patient's circumstances preclude definitive restorative care, extraction is indicated to eliminate the infectious focus. Antibiotic therapy occupies a secondary, adjunctive role rather than serving as a stand-alone solution. In localized odontogenic infections without systemic features or spreading cellulitis, timely dental intervention alone is often sufficient. Antibiotics are reserved for scenarios where there is evidence of local extension or systemic involvement, such as facial cellulitis, regional lymphadenopathy, fever, malaise, or fatigue. In such cases, prescribing antibiotics prior to or alongside dental referral is appropriate to limit further dissemination and reduce systemic burden.[32]

Evidence-based guidelines from the American Dental Association identify amoxicillin as the preferred first-line antibiotic for most uncomplicated dental infections, due to its favorable antimicrobial spectrum against common oral flora, good absorption, and relatively low incidence of

adverse effects. Standard adult dosing regimens include 500 mg every 8 hours or 875 mg every 12 hours. Penicillin VK is an acceptable alternative; however, amoxicillin is generally favored because of its broader coverage and improved gastrointestinal tolerability. Antibiotic courses should not be unnecessarily prolonged; current guidance recommends discontinuation approximately 24 hours after resolution of signs and symptoms, with clinical reassessment within about 3 days of therapy initiation to confirm response and ensure that definitive dental treatment has been or will be performed [34]. In patients with a documented, true IgE-mediated or severe delayed hypersensitivity to penicillins, alternative regimens must be considered. The American Dental Association suggests clindamycin 300 mg every 8 hours, cephalexin 500 mg every 6 hours, or azithromycin 500 mg on day 1 followed by 250 mg daily for an additional 4 days as potential alternatives in appropriate clinical contexts.C:\Users\akk29\Downloads\A1 [34] Nonetheless, each of these agents carries limitations. Clindamycin is strongly associated with *Clostridioides difficile* colitis and should be prescribed only when clearly indicated and with careful monitoring. Azithromycin, while convenient, has higher rates of resistance among oral pathogens in some regions. Cephalexin is not appropriate in true anaphylactic-type β-lactam allergies and may have limited anaerobic coverage. For these reasons, antibiotic selection in penicillin-allergic patients should be individualized, balancing efficacy, allergy profile, and risk of adverse events.

In severe or refractory infections, or when a significant anaerobic component is suspected—such as in deep space infections, recurrent abscesses, or in immunocompromised hosts—therapeutic regimens may require expansion. Metronidazole is often combined with amoxicillin to augment anaerobic coverage, or amoxicillin/clavulanate is chosen to enhance activity against β-lactamase–producing organisms. Hospitalized patients with severe odontogenic infections often respond to intravenous regimens such as ampicillin/sulbactam or second- or third-generation cephalosporins, tailored based on local resistance patterns and patient-specific factors.[34][35] In individuals with serious β-lactam allergy, fluoroquinolones such as moxifloxacin or combination regimens including trimethoprim-sulfamethoxazole may be considered, although these options require careful evaluation of cardiac, renal, and other comorbid risks.[34][35] Regardless of antibiotic choice, it must be emphasized that antimicrobial therapy alone is not curative. Without drainage or removal of the infectious nidus, bacteria can persist within necrotic pulp, closed abscess cavities, or periodontal pockets, promoting relapse and resistance. Definitive dental intervention—whether through endodontic therapy, extraction, incision and drainage, or periodontal treatment—remains the cornerstone of effective management.[36] Early coordination with dental professionals is therefore central to optimal outcomes.

Pain management is another critical component of treatment. Acute dental pain can be severe and debilitating, and effective analgesia improves patient comfort, facilitates oral intake, and enables adherence to definitive care. Current recommendations from the American Dental Association and the United States Centers for Disease Control and Prevention endorse nonopioid analgesics as first-line therapy for dental pain.[1][4][7][4] Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen 400 mg every 6 to 8 hours (up to a maximum of 2,400 mg/day) or naproxen sodium 440 mg every 8 to 12 hours (maximum 1,100 mg/day) are preferred, either alone or in combination with acetaminophen 500 to 1,000 mg every 6 hours (maximum 4,000 mg/day). Multiple studies demonstrate that the combination of ibuprofen and acetaminophen provides superior analgesia compared to opioid-containing regimens and achieves the highest rates of maximal pain relief with a more favorable safety profile.[37][38][39]. When NSAIDs are contraindicated—such as in patients with significant gastrointestinal ulcer disease, advanced renal impairment, or certain cardiovascular conditions—acetaminophen at full therapeutic doses may be used as monotherapy.[37] Opioid analgesics, including hydrocodone- or oxycodone-acetaminophen combinations, should be reserved for exceptional circumstances in which nonopioid regimens have failed to provide adequate relief or are contraindicated. When opioids are prescribed, they should be used at the lowest effective dose and for the shortest possible duration, typically not exceeding three days, with explicit counseling on risks of dependence, sedation, and misuse—particularly in adolescents and young adults.[37][38][39] Fixed-dose opioid combinations such as acetaminophen-codeine offer no clear advantage over nonopioid regimens and carry the same opioid-related hazards, and thus should be used sparingly, if at all.[38][40].

Local anesthetic techniques can further enhance pain control. Short-acting agents such as 2% lidocaine with 1:100,000 epinephrine or 4% articaine with 1:100,000 epinephrine can provide rapid relief through nerve blocks or local infiltration, which is particularly valuable in acute pulpitis or abscess drainage procedures. Long-acting anesthetics such as 0.5% bupivacaine with 1:200,000 epinephrine may be selected when prolonged postoperative analgesia is desirable.[37] For minor mucosal discomfort, topical anesthetics such as benzocaine 10% to 20% may afford short-term symptom relief.[37] In pediatric and special-needs populations, nonopioid analgesics remain the mainstay, with careful weight-

based dosing and attention to potential interactions with chronic medications. Across all age groups, analgesic choices must be individualized according to comorbid conditions, concomitant therapies, and contraindications. Ultimately, successful treatment of oral and facial infections of dental origin hinges on an integrated approach that combines early recognition of severity, appropriate triage, prompt surgical management of the source, judicious use of antibiotics, and safe, effective pain control.

Differential Diagnosis

The differential diagnosis of oral and facial pain or swelling suggestive of a dental infection is broad and requires careful clinical discrimination, as several non-odontogenic conditions can present with overlapping signs and symptoms. Localized odontogenic infections, particularly those associated with periapical abscesses or periodontal pathology, may be mistaken for salivary gland disorders, including sialadenitis, sialolithiasis, and benign or malignant salivary gland neoplasms. Acute sialadenitis typically presents with localized painful swelling, erythema, and tenderness over the affected gland, often exacerbated by eating due to stimulated salivary flow. Sialolithiasis manifests with similar features, particularly postprandial pain and intermittent swelling, and may be confused with localized dental abscess or cellulitis of odontogenic origin when the swelling overlies the submandibular or parotid regions. Salivary gland tumors, especially in early stages, may present as a unilateral, painless facial or intraoral mass; as they enlarge or become secondarily inflamed, tenderness and functional impairment may ensue, further complicating differentiation. Paranasal sinus disease, particularly maxillary sinusitis, also features prominently in the differential diagnosis of dental pain and facial swelling. Patients with maxillary sinusitis may report facial pressure, pain in the distribution of the maxillary teeth, and warmth or erythema overlying the maxillary sinus. The close anatomical relationship between the apices of maxillary posterior teeth and the sinus floor can result in referred pain, leading patients or clinicians to suspect a dental origin. Conversely, odontogenic infections originating from these teeth may penetrate the sinus floor, producing sinusitis of dental origin. Distinguishing between primary sinus disease and dental pathology therefore requires detailed history, careful intraoral and extraoral examination, and, when necessary, radiographic or cross-sectional imaging [37][40]. Other conditions that may mimic dental infection include temporomandibular disorders, neuropathic facial pain, facial cellulitis of cutaneous origin, lymphadenitis, and neoplastic processes of the jaws or soft tissues. In children, teething discomfort, eruptive cysts, or viral stomatitis can also be confused with early odontogenic infection. Accurate differentiation hinges on identifying a clearly diseased tooth or periodontal structure, the presence and character of swelling, the relationship of pain to mastication or thermal stimuli, and the presence of systemic signs. Misdiagnosis may delay appropriate treatment, expose patients to unnecessary antibiotics, and increase the risk of complications, underscoring the importance of a systematic diagnostic approach [40].

Prognosis

The prognosis of dental infections is closely tied to their extent, rapidity of recognition, and timeliness of definitive management. For uncomplicated odontogenic infections confined to the tooth and adjacent alveolar tissues, outcomes are generally excellent when prompt dental intervention—such as drainage, root canal therapy, or extraction—is undertaken. These localized infections usually resolve fully with appropriate treatment and, in otherwise healthy individuals, rarely lead to long-term morbidity. Early detection of pulpitis, incipient abscess formation, or localized periodontal involvement further improves prognosis by preventing progression to more extensive disease. However, when dental infections extend beyond the alveolar process into deep neck spaces, prognosis becomes more guarded and the risk of serious sequelae increases substantially. Deep neck infections associated with odontogenic sources carry significant morbidity and mortality, particularly when diagnosis is delayed or when patients have underlying comorbidities such as diabetes, immunosuppression, or advanced age. Reported mortality rates for deep neck infections range widely, from approximately 1% to 25%, reflecting variations in patient populations, healthcare access, and the presence of complications such as airway obstruction, septic shock, or mediastinal spread.[41][42] Early recognition of red flag signs—such as trismus, dysphagia, odynophagia, and respiratory compromise—and rapid escalation to specialist care are therefore critical determinants of outcome.

Mediastinitis represents one of the most severe complications of descending odontogenic infection and is associated with particularly high mortality rates, reported to reach up to 40% despite advances in critical care, surgery, and antimicrobial therapy.[41][42] The need for aggressive surgical drainage, prolonged hospitalization, and intensive care support contributes to both the clinical and economic burden of these cases. Outcomes are further influenced by the speed of diagnosis and the initial adequacy of source control. In general, patients who receive definitive dental treatment early, alongside judicious use of antibiotics when indicated, have excellent prognoses with low risk of recurrence. In contrast, reliance on analgesics and antibiotics alone, without addressing the underlying source, often results in persistent or recurrent infection and increases the likelihood of progression to serious complications. Thus, from a prognostic perspective, the critical elements include early recognition,

appropriate triage based on risk stratification, timely definitive dental intervention, and effective management of underlying systemic conditions that may impair host defenses [41][42].

Complications

Dental infections, though often initially localized, have the potential to give rise to serious and sometimes life-threatening complications as they spread along the potential spaces and fascial planes of the head and neck. The routes of dissemination are determined by tooth location, root anatomy, cortical bone thickness, and the relationship of apices to muscle attachments. Once infection breaches the confines of the alveolar bone, it can extend into adjacent soft tissues and deep spaces. One important complication is osteomyelitis of the jaws, which may arise when infection spreads contiguously from an infected tooth into the surrounding cortical and cancellous bone. Osteomyelitis is characterized by progressive bone destruction, sequestrum formation, and chronic draining sinus tracts, and may require prolonged antimicrobial therapy and surgical debridement. Infections originating from lower second and third molars are particularly concerning, as their root apices lie close to the sublingual, submandibular, and submental spaces. Spread into these spaces can result in Ludwig angina, a rapidly progressive, bilateral cellulitis of the submandibular space that elevates the tongue and floor of the mouth, threatening airway patency and necessitating urgent airway management and surgical drainage. In pediatric populations, odontogenic infections may spread posteriorly into the retropharyngeal or parapharyngeal spaces, giving rise to retropharyngeal or parapharyngeal abscesses, respectively. These conditions can manifest with neck stiffness, torticollis, dysphagia, fever, and respiratory symptoms, and may progress to airway compromise or mediastinal involvement if not promptly treated. Descending necrotizing mediastinitis represents a particularly severe outcome, in which infection tracks inferiorly through deep and superficial fascial planes into the mediastinum, resulting in a fulminant, life-threatening process [43].

Further cranial spread of dental infections can, in rare instances, lead to cavernous sinus thrombosis, particularly when maxillary or canine space infections involve veins that communicate with the cavernous sinus.[43] Clinical manifestations may include severe headache, cranial nerve deficits, ophthalmoplegia, and visual disturbances. Very rarely, dental infections have been implicated in the development of meningitis or subdural empyema, especially in cases of maxillary sinus involvement or direct extension from infected maxillary teeth.[44] Systemic hematogenous dissemination constitutes another significant complication. Dental infections and invasive dental procedures such as extractions or endodontic treatment can cause transient bacteremia, which in susceptible individuals may seed damaged or prosthetic heart valves, resulting in infective endocarditis.[27] This risk is heightened in patients with structural heart disease, prosthetic valves, or certain congenital heart conditions, for whom prophylactic antibiotics are recommended under specific guideline-directed circumstances. Collectively, these complications underscore the importance of early and definitive treatment of dental infections and the need for clinicians to maintain a high index of suspicion when evaluating patients with facial or neck swelling, systemic symptoms, or neurologic or cardiopulmonary changes [43][44].

Patient Education

Prevention of dental infections—and by extension, their potentially serious complications—rests largely on effective patient education and reinforcement of sound oral hygiene practices. Counseling should emphasize that odontogenic infections are largely preventable conditions, arising from cumulative caries, periodontal disease, and failure to address early dental pathology. Patients must understand that daily oral hygiene behaviors, including thorough toothbrushing with fluoridated toothpaste at least twice daily and regular flossing or interdental cleaning, are fundamental in reducing plaque accumulation and preventing both dental caries and gingival inflammation. Dietary counseling is equally important, as frequent consumption of sugar-containing foods and beverages fuels the cariogenic activity of oral biofilm. Patients benefit from specific guidance on limiting sugary snacks, reducing intake of sweetened drinks, and avoiding prolonged sipping or grazing patterns that expose teeth to repeated acid attacks. Education should also address the role of fluoridated water and topical fluoride products in enhancing enamel resistance to demineralization, particularly for individuals at elevated caries risk. In addition to personal hygiene and diet, patients should be informed about the importance of regular professional dental care. Routine dental checkups enable early detection of incipient caries, reversible gingivitis, and minor restorations before they progress to pulpitis, abscesses, or advanced periodontitis. Many patients seek emergency or medical care only when pain becomes severe, unaware that earlier intervention would have been simpler, less invasive, and less costly. Clarifying this connection can motivate more consistent engagement with preventive dental services [44].

Patients with specific risk factors—such as diabetes, immunosuppression, xerostomia-inducing medications, tobacco or alcohol use, and a history of recurrent dental infections—require tailored education about their heightened susceptibility and the need for more frequent dental surveillance. Addressing common misconceptions is also important; for instance, patients should be informed

that antibiotics alone cannot cure most dental infections and that definitive dental treatment is always necessary to eradicate the source. Public health efforts can complement individual counseling by promoting oral health literacy, advocating for fluoridation programs, and improving access to affordable dental care. Educational campaigns in schools, community centers, and primary care settings can reinforce messages about brushing, flossing, diet, and regular dental visits. By integrating oral health education into routine medical encounters, especially in high-risk or underserved populations, clinicians can contribute to reducing the incidence of odontogenic infections and minimizing their associated complications over the long term [44].

Other Issues

Several key principles help guide the recognition and management of dental infections in clinical practice. First, it is essential to remember that odontogenic infections originate within the tooth or its supporting structures—namely the pulp, periodontal ligament, gingiva, and alveolar bone—and may subsequently spread into adjacent tissues if not promptly addressed. The most common pathway of infection involves bacterial invasion of the dental pulp, typically via carious lesions or trauma, with subsequent extension into periapical tissues. Infections may also arise primarily from the gingiva and periodontal structures, where plaque-induced inflammation progresses from gingivitis to destructive periodontitis if left untreated. Second, clinicians should recognize that seemingly modest complaints, such as localized tooth pain or swelling, may mask evolving deep-space processes. More serious symptoms—including fever, facial edema, trismus, dysphagia, or dysphonia—often indicate that infection has extended beyond the alveolar process into deep neck spaces, necessitating urgent evaluation and potential hospital admission. These signs should always be treated as red flags, prompting thorough airway assessment and, where indicated, consultation with surgical specialists. Imaging with contrast-enhanced computed tomography plays a central role when fascial space involvement is suspected. CT with contrast permits detailed visualization of soft tissues, differentiation between diffuse cellulitis and localized abscess, evaluation of the extent of disease, and identification of potential complications such as mediastinal spread or vascular involvement. This information is critical for planning surgical drainage and guiding the scope of intervention [33][44].

From a therapeutic standpoint, the most important step in treating dental infections is drainage and removal of the source of infection. Whether via root canal therapy, tooth extraction, or surgical drainage of an abscess, definitive source control is necessary for durable resolution. Antibiotics, while often helpful as adjunctive therapy in the presence of systemic signs or spreading cellulitis, cannot substitute for procedural management. Severe complications, although relatively rare in the context of modern dental and medical care, remain clinically important. These include osteomyelitis of the jaws, Ludwig angina, retropharyngeal and parapharyngeal abscesses, necrotizing mediastinitis, cavernous sinus thrombosis, meningitis, and subdural empyema. Awareness of these potential sequelae should inform both risk stratification and patient counseling. Ultimately, clinicians must maintain a high index of suspicion, adopt a proactive approach to diagnostic imaging and referral when indicated, and emphasize definitive dental treatment to minimize the risk of progression to these life-threatening conditions [44].

Enhancing Healthcare Team Outcomes

Optimizing outcomes in patients with dental infections requires a coordinated, interprofessional approach that leverages the expertise of physicians, dentists, advanced practitioners, nurses, pharmacists, and other allied health professionals. Physicians and advanced practice providers, particularly in emergency and primary care settings, frequently serve as the first point of contact for individuals presenting with acute dental pain or facial swelling. Their understanding of the etiology, natural history, and potential complications of odontogenic infections is critical for appropriate triage and early decision-making. Conditions such as pulpitis and periapical abscess often prompt medical consultations; however, definitive resolution depends on eliminating the source of infection through dental procedures such as drainage, endodontic therapy, or extraction. Over-reliance on antibiotics in place of dental referral not only fails to cure the infection but also fosters recurrent disease and antimicrobial resistance. Enhancing education in oral and dental pathology during medical training would substantially improve clinicians' ability to recognize odontogenic disease, distinguish it from mimics, and prioritize timely referral for definitive care. Nurses are central to patient assessment and ongoing monitoring. They are often the first to detect red flag symptoms such as deteriorating airway function, escalating pain, fever, or hemodynamic instability. By promptly communicating these changes to the medical team, nurses help ensure timely escalation of care. They also play an important role in patient education, reinforcing instructions on medication adherence, follow-up with dental providers, and recognition of warning signs that should prompt urgent reevaluation [27][35][43][44].

Pharmacists contribute by ensuring that prescribed antibiotics are appropriate for suspected oral pathogens, dosed correctly, and safe in the context of the patient's comorbidities and concomitant medications. They can counsel against unnecessary or prolonged antibiotic courses and assist in mitigating risks such as *Clostridioides difficile* infection or drug interactions. Effective collaboration between medical and dental professionals is essential to coordinate diagnostic and

therapeutic plans. This may involve establishing referral pathways, shared protocols for high-risk presentations, and communication channels that facilitate rapid consultation, especially for patients with severe or complex infections. Interprofessional case discussions, joint educational initiatives, and integration of oral health into broader healthcare quality agendas can further strengthen team performance. By fostering mutual understanding of roles and responsibilities, maintaining open and timely communication, and aligning strategies around evidence-based care, the healthcare team can reduce delays in definitive treatment, minimize unnecessary antibiotic use, and lower the risk of serious complications. Ultimately, such collaborative, patient-centered care improves clinical outcomes and enhances the overall quality and safety of management for individuals with dental infections [44].

Conclusion:

In conclusion, pediatric odontogenic infections represent a significant clinical entity where timely and appropriate management is crucial to prevent progression from localized dental disease to life-threatening deep neck space infections. The cornerstone of successful treatment is a clear understanding that definitive care requires dental intervention—such as drainage, root canal therapy, or extraction—to eliminate the infectious nidus. Antibiotic therapy plays only an adjunctive role and should be reserved for cases with evidence of systemic spread or cellulitis, not for uncomplicated pulpitis or localized abscess. Effective management hinges on early recognition and risk stratification by the first-contact clinician, often in a medical setting. A thorough history and physical exam are vital to identify “red flag” symptoms like trismus, dysphagia, or airway compromise that necessitate urgent escalation. Imaging, particularly CT scanning for suspected deep space involvement, is critical for surgical planning. An interdisciplinary approach, integrating emergency physicians, pediatricians, dentists, oral surgeons, and pharmacists, is paramount. This collaboration ensures rapid diagnosis, appropriate antimicrobial stewardship, effective pain management prioritizing non-opioid regimens, and seamless referral for definitive dental care. Furthermore, patient and family education on oral hygiene, dietary habits, and the importance of regular dental visits is essential for long-term prevention. Ultimately, by adhering to evidence-based guidelines that emphasize source control over pharmacologic management alone and by fostering robust interprofessional teamwork, clinicians can significantly reduce the morbidity and potential mortality associated with these common yet potentially severe pediatric infections.

References:

1. Robertson D, Smith AJ. The microbiology of the acute dental abscess. Journal of medical microbiology. 2009 Feb:58(Pt 2):155-162. doi: 10.1099/jmm.0.003517-0.
2. Thomas TT. III. Ludwig's Angina (Part II): An Anatomical, Clinical and Statistical Study. Annals of surgery. 1908 Mar:47(3):335-73
3. Vytla S, Gebauer D. Clinical guideline for the management of odontogenic infections in the tertiary setting. Australian dental journal. 2017 Dec:62(4):464-470. doi: 10.1111/adj.12538.
4. Pitts NB, Zero DT, Marsh PD, Ekstrand K, Weintraub JA, Ramos-Gomez F, Tagami J, Twetman S, Tsakos G, Ismail A. Dental caries. Nature reviews. Disease primers. 2017 May 25:3():17030. doi: 10.1038/nrdp.2017.30.
5. Nguyen DH, Martin JT. Common dental infections in the primary care setting. American family physician. 2008 Mar 15:77(6):797-802
6. Ricucci D, Loghin S, Siqueira JF Jr. Correlation between clinical and histologic pulp diagnoses. Journal of endodontics. 2014 Dec:40(12):1932-9. doi: 10.1016/j.joen.2014.08.010.
7. Agnihotry A, Fedorowicz Z, van Zuuren EJ, Farman AG, Al-Langawi JH. Antibiotic use for irreversible pulpitis. The Cochrane database of systematic reviews. 2016 Feb 17:2():CD004969. doi: 10.1002/14651858.CD004969.pub4.
8. Nagle D, Reader A, Beck M, Weaver J. Effect of systemic penicillin on pain in untreated irreversible pulpitis. Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics. 2000 Nov:90(5):636-40
9. Siqueira JF Jr, Rôças IN. Microbiology and treatment of acute apical abscesses. Clinical microbiology reviews. 2013 Apr:26(2):255-73. doi: 10.1128/CMR.00082-12.
10. Candamourty R, Venkatachalam S, Babu MR, Kumar GS. Ludwig's Angina - An emergency: A case report with literature review. Journal of natural science, biology, and medicine. 2012 Jul:3(2):206-8. doi: 10.4103/0976-9668.101932.
11. Bascones Martínez A, Aguirre Urízar JM, Bermejo Fenoll A, Blanco Carrión A, Gay-Escoda C, González-Moles MA, Gutiérrez Pérez JL, Jiménez Soriano Y, Liébana Ureña J, López Marcos JF, Maestre Vera JR, Perea Pérez EJ, Prieto Prieto J, de Vicente Rodríguez JC. Consensus statement on antimicrobial treatment of odontogenic bacterial infections. Medicina oral, patologia oral y cirugia bucal. 2004 Nov-Dec:9(5):369-76; 363-9
12. Bartold PM. Lifestyle and periodontitis: The emergence of personalized periodontics. Periodontology 2000. 2018 Oct:78(1):7-11. doi: 10.1111/prd.12237.
13. Yekani M, Dastgir M, Fattahi S, Shahi S, Maleki Dizaj S, Memar MY. Microbiological and

molecular aspects of periodontitis pathogenesis: an infection-induced inflammatory condition. Frontiers in cellular and infection microbiology. 2025:15():1533658. doi: 10.3389/fcimb.2025.1533658.
14. Boykin MJ, Gilbert GH, Tilashalski KR, Shelton BJ. Incidence of endodontic treatment: a 48-month prospective study. Journal of endodontics. 2003 Dec:29(12):806-9
15. Wang J, Ahani A, Pogrel MA. A five-year retrospective study of odontogenic maxillofacial infections in a large urban public hospital. International journal of oral and maxillofacial surgery. 2005 Sep:34(6):646-9
16. Dye BA, Li X, Beltran-Aguilar ED. Selected oral health indicators in the United States, 2005-2008. NCHS data brief. 2012 May:(96):1-8
17. Beltrán-Aguilar ED, Barker LK, Canto MT, Dye BA, Gooch BF, Griffin SO, Hyman J, Jaramillo F, Kingman A, Nowjack-Raymer R, Selwitz RH, Wu T, Centers for Disease Control and Prevention (CDC). Surveillance for dental caries, dental sealants, tooth retention, edentulism, and enamel fluorosis--United States, 1988-1994 and 1999-2002. Morbidity and mortality weekly report. Surveillance summaries (Washington, D.C. : 2002). 2005 Aug 26:54(3):1-43
18. Rahimi-Nedjat RK, Sagheb K, Sagheb K, Hormes M, Walter C, Al-Nawas B. The role of diabetes mellitus on the formation of severe odontogenic abscesses-a retrospective study. Clinical oral investigations. 2021 Nov:25(11):6279-6285. doi: 10.1007/s00784-021-03926-4.
19. Saleh W, Xue W, Katz J. Diabetes Mellitus and Periapical Abscess: A Cross-sectional Study. Journal of endodontics. 2020 Nov:46(11):1605-1609. doi: 10.1016/j.joen.2020.08.015.
20. Rotstein I, Katz J. Association of periodontal disease and the prevalence of acute periapical abscesses. Journal of the American Dental Association (1939). 2025 Mar:156(3):234-238. doi: 10.1016/j.adaj.2024.12.010.
21. Katz J, Rotstein I. Acute periapical abscesses and obese patients. American journal of dentistry. 2024 Aug:37(4):197-200
22. Shay K. Infectious complications of dental and periodontal diseases in the elderly population. Clinical infectious diseases : an official publication of the Infectious Diseases Society of America. 2002 May 1:34(9):1215-23
23. Cope AL, Chestnutt IG. The implications of a cost-of-living crisis for oral health and dental care. British dental journal. 2023 Apr:234(7):501-504. doi: 10.1038/s41415-023-5685-0.
24. Steele J, Shen J, Tsakos G, Fuller E, Morris S, Watt R, Guarnizo-Herreño C, Wildman J. The Interplay between socioeconomic inequalities and clinical oral health. Journal of dental research. 2015 Jan:94(1):19-26. doi: 10.1177/0022034514553978.
25. Kailembo A, Quiñonez C, Lopez Mitnik GV, Weintraub JA, Stewart Williams J, Preet R, Iafolla T, Dye BA. Income and wealth as correlates of socioeconomic disparity in dentist visits among adults aged 20 years and over in the United States, 2011-2014. BMC oral health. 2018 Aug 23:18(1):147. doi: 10.1186/s12903-018-0613-4.
26. Northridge ME, Kumar A, Kaur R. Disparities in Access to Oral Health Care. Annual review of public health. 2020 Apr 2:41():513-535. doi: 10.1146/annurev-publhealth-040119-094318.
27. Jevon P, Abdelrahman A, Pigadas N. Management of odontogenic infections and sepsis: an update. British dental journal. 2020 Sep:229(6):363-370. doi: 10.1038/s41415-020-2114-5.
28. James P, Worthington HV, Parnell C, Harding M, Lamont T, Cheung A, Whelton H, Riley P. Chlorhexidine mouthrinse as an adjunctive treatment for gingival health. The Cochrane database of systematic reviews. 2017 Mar 31:3(3):CD008676. doi: 10.1002/14651858.CD008676.pub2.
29. Bosshardt DD. The periodontal pocket: pathogenesis, histopathology and consequences. Periodontology 2000. 2018 Feb:76(1):43-50. doi: 10.1111/prd.12153.
30. Trombelli L, Farina R, Silva CO, Tatakis DN. Plaque-induced gingivitis: Case definition and diagnostic considerations. Journal of periodontology. 2018 Jun:89 Suppl 1():S46-S73. doi: 10.1002/JPER.17-0576.
31. Mardini S, Gohel A. Imaging of Odontogenic Infections. Radiologic clinics of North America. 2018 Jan:56(1):31-44. doi: 10.1016/j.rcl.2017.08.003.
32. Beech N, Goh R, Lynham A. Management of dental infections by medical practitioners. Australian family physician. 2014 May:43(5):289-91
33. Miah MR, Ali AS. Ludwig's angina. British dental journal. 2020 Sep:229(5):268. doi: 10.1038/s41415-020-2132-3.
34. Lockhart PB, Tampi MP, Abt E, Aminoshariae A, Durkin MJ, Fouad AF, Gopal P, Hatten BW, Kennedy E, Lang MS, Patton LL, Paumier T, Suda KJ, Pilcher L, Urquhart O, O'Brien KK, Carrasco-Labra A. Evidence-based clinical practice guideline on antibiotic use for the urgent management of pulpal- and periapical-related dental pain and intraoral swelling: A report from the American Dental Association. Journal of the American Dental Association (1939). 2019 Nov:150(11):906-921.e12. doi: 10.1016/j.adaj.2019.08.020.
35. Zirk M, Buller J, Goeddertz P, Rothamel D, Dreiseidler T, Zöller JE, Kreppel M. Empiric

systemic antibiotics for hospitalized patients with severe odontogenic infections. Journal of cranio-maxillo-facial surgery : official publication of the European Association for Cranio-Maxillo-Facial Surgery. 2016 Aug:44(8):1081-8. doi: 10.1016/j.jcms.2016.05.019.

36. Segura-Egea JJ, Gould K, Şen BH, Jonasson P, Cotti E, Mazzoni A, Sunay H, Tjäderhane L, Dummer PMH. Antibiotics in Endodontics: a review. International endodontic journal. 2017 Dec:50(12):1169-1184. doi: 10.1111/iej.12741.
37. Carrasco-Labra A, Polk DE, Urquhart O, Aghaloo T, Claytor JW Jr, Dhar V, Dionne RA, Espinoza L, Gordon SM, Hersh EV, Law AS, Li BS, Schwartz PJ, Suda KJ, Turturro MA, Wright ML, Dawson T, Miroshnychenko A, Pahlke S, Pilcher L, Shirey M, Tampi M, Moore PA. Evidence-based clinical practice guideline for the pharmacologic management of acute dental pain in adolescents, adults, and older adults: A report from the American Dental Association Science and Research Institute, the University of Pittsburgh, and the University of Pennsylvania. Journal of the American Dental Association (1939). 2024 Feb:155(2):102-117.e9. doi: 10.1016/j.adaj.2023.10.009.
38. Dowell D, Ragan KR, Jones CM, Baldwin GT, Chou R. CDC Clinical Practice Guideline for Prescribing Opioids for Pain - United States, 2022. MMWR. Recommendations and reports : Morbidity and mortality weekly report. Recommendations and reports. 2022 Nov 4:71(3):1-95. doi: 10.15585/mmwr.rr7103a1.
39. Moore PA, Ziegler KM, Lipman RD, Aminoshariae A, Carrasco-Labra A, Mariotti A. Benefits and harms associated with analgesic medications used in the management of acute dental pain: An overview of systematic reviews. Journal of the American Dental Association (1939). 2018 Apr:149(4):256-265.e3. doi: 10.1016/j.adaj.2018.02.012.
40. da Silva PB, Mendes AT, Cardoso MBF, da Rosa RA, do Nascimento AL, Pereira JR, Só MVR. Comparison between isolated and associated with codeine acetaminophen in pain control of acute apical abscess: a randomized clinical trial. Clinical oral investigations. 2021 Mar:25(3):875-882. doi: 10.1007/s00784-020-03374-6.
41. Almuqamam M, Gonzalez FJ, Sharma S, Kondamudi NP. Deep Neck Infections. StatPearls. 2025
42. Sanders JL, Houck RC. Dental Abscess. StatPearls. 2025 Jan
43. Yeo GS, Kim HY, Kwak EJ, Jung YS, Park HS, Jung HD. Cavernous sinus thrombosis caused by a dental infection: a case report. Journal of the Korean Association of Oral and Maxillofacial Surgeons. 2014 Aug:40(4):195-8. doi: 10.5125/jkaoms.2014.40.4.195.
44. Cariati P, Cabello-Serrano A, Monsalve-Iglesias F, Roman-Ramos M, Garcia-Medina B. Meningitis and subdural empyema as complication of pterygomandibular space abscess upon tooth extraction. Journal of clinical and experimental dentistry. 2016 Oct:8(4):e469-e472.