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Dental Abscess: Diagnosis And Management

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Abstract

Odontogenic infections, originating from teeth or their supporting structures, are among the most common infections encountered in clinical practice. These infections can remain localized or spread to adjacent or distant areas, potentially leading to severe complications. Periapical infections and pericoronitis are the most prevalent causes of odontogenic infections. Clinical presentations vary depending on the infection source and extent of spread, with pain, redness, and swelling being the hallmark signs. Ludwig's angina, a severe condition characterized by rapidly spreading cellulitis, can lead to life-threatening complications such as airway obstruction. The microbiology of odontogenic infections is polymicrobial, involving both facultative and strict anaerobes, with anaerobic bacteria outnumbering aerobes. Diagnosis is based on clinical history, examination, and radiographic findings, with cultures reserved for severe or unresponsive infections. Treatment primarily focuses on eliminating the infection source through dental interventions, with antibiotics serving as adjunctive therapy. Penicillin and clindamycin are the preferred antibiotics for odontogenic infections, while metronidazole, azithromycin, and moxifloxacin are effective alternatives in specific situations. Prompt diagnosis and appropriate management are crucial to prevent the progression of odontogenic infections and their potential complications.

Keywords: Dental Abscess, Diagnosis, Management, Oral Infection, Periapical Abscess, Periodontal Abscess.

Introduction

Odontogenic infections refer to infections involving the alveolus, jaws, or face that originate from a tooth or its supporting structures. These infections are among the most frequently encountered in clinical practice. The primary etiological factors include dental caries, deep restorations or failed endodontic treatments, pericoronitis, and periodontal disease. Typically, an odontogenic infection begins locally around a tooth and may remain confined to the originating region or spread to adjacent or distant areas. The progression of the infection is influenced by the bacterial virulence, the host's immune response, and regional anatomical factors. Periapical infection is the most common type of odontogenic infection and occurs when microorganisms invade the root canal system of the tooth. This acute apical condition involves concurrent infection of the root canal and the periradicular tissues, as the latter is an extension of the former. Once microorganisms infiltrate the periapical tissues through the apical foramen, they trigger an inflammatory response that may result in abscess formation. While such infections are typically localized intraorally, they can sometimes disseminate to distal areas and lead to severe complications, including sinusitis, airway obstruction, cavernous sinus thrombosis, brain abscess, or even mortality.

Pericoronitis is another prevalent cause of odontogenic infections. The condition primarily arises from the accumulation of bacteria and food debris trapped in the space between the overlapping gingiva of a partially erupted mandibular third molar and the tooth's crown. Most cases are chronic, manifesting as mild, persistent inflammation in the mandibular third molar area. However, pericoronitis can escalate into a severe infection characterized by fever, swelling, and abscess formation, which may spread if left untreated. In certain cases, symptoms can become severe due to rapid infection spread, necessitating hospitalization for intravenous (IV) antibiotic administration and potential surgical extraction of the affected tooth under general anesthesia. Given the proximity of the infection to the pharynx, airway obstruction becomes a significant concern.

Clinical Presentations

The clinical presentation of an odontogenic infection is highly variable and depends on factors such as the source of the infection (anterior teeth versus posterior teeth; maxillary versus mandibular teeth) and whether the infection is localized or disseminated. As with all infections, the hallmark clinical signs and symptoms include pain or tenderness, redness, and swelling. Patients with superficial dental infections often exhibit localized pain, cellulitis, and sensitivity to percussion and temperature. Conversely, patients with deep infections or abscesses that spread along fascial planes may present with swelling, fever, and sometimes difficulty swallowing, opening the mouth, or breathing. In cases involving single-space infections, the buccal space is most affected (60%), followed by the canine space (13%). In multi-space infections, the submandibular and buccal spaces are most frequently involved. Acute periapical inflammation or infection typically presents with symptoms such as pain and swelling. In contrast, chronic inflammatory responses are often asymptomatic and may lead to bone resorption around the root apex, visible on dental radiographs as periapical radiolucency. Chronic asymptomatic periapical pathology may flare up, presenting as an acute dental infection(Santosh et al., 2014).

Ludwig's Angina

Ludwig's angina is a severe, potentially life-threatening condition characterized by brawny, board-like swelling caused by rapidly spreading cellulitis, typically without lymphatic involvement or abscess formation. This condition affects the sublingual, submental, and submandibular spaces, leading to tongue elevation, edema, drooling, and airway obstruction. The infection is most associated with infected lower molars or pericoronitis(Kim et al., 2012).

Signs of an impending airway catastrophe include hoarseness, stridor, respiratory distress, decreased air movement, cyanosis, and the "sniffing" position. The sniffing position is characterized by an upright posture with the neck extended forward and the chin elevated, often adopted by patients with imminent upper airway compromise. Beyond airway compromise, Ludwig's angina is associated with other complications, such as carotid sheath infection and arterial rupture, suppurative thrombophlebitis of the internal jugular vein, mediastinitis, empyema, pericardial and/or pleural effusion, osteomyelitis of the mandible, subphrenic abscess, and aspiration pneumonia.

Spread of Infection

Odontogenic infections are generally mild and tend to remain localized within the alveolar ridge or adjacent tissues, such as the buccal, labial, or lingual vestibule. However, if untreated, infections originating at the apex of a tooth can erode the surrounding bone, typically at its thinnest region, and spread to adjacent tissues. In the mandible, the lingual aspect of the molar region is most susceptible, while in the maxilla, the thin buccal plate is the most vulnerable. The direction of spread is determined by the relationship between the muscle attachment and the point of infection perforation. Most odontogenic infections perforate the bone in such a manner that they form vestibular abscesses. However, when infections spread beyond muscle attachments, they invade fascial spaces, leading to more severe conditions. Occasionally, infection may spread beyond fascial space barriers, resulting in life-threatening complications such as cavernous sinus thrombosis, brain abscess, airway obstruction, mediastinitis, or endocarditis.

Infections affecting the fascial planes of the head and neck typically spread downward along the cervical fascia due to factors like gravity, respiration, and negative intrathoracic pressure. While the spread pattern may vary between patients, a consistent trend in the distribution of infections across these spaces is observed(Mihos et al., 2004).

Besides fascial spread, infections may disseminate via hematogenous or lymphatic routes. Bacteria from odontogenic infections can enter the bloodstream, leading to bacteremia, which can have systemic effects. In immunocompromised individuals, bacteremia may progress to septicemia, a more severe bloodstream infection characterized by symptoms such as chills, high fever, rapid heart rate, severe nausea, vomiting, and altered mental state. Hematogenous spread may also occur via valveless veins, including the facial, angular, and ophthalmic veins, allowing retrograde flow into the cavernous sinus and cranial cavity. This can result in cavernous sinus thrombosis, although dental infections account for less than 10% of such cases. Most cases are related to maxillary infections, and the incidence has significantly decreased due to effective antimicrobial therapies.

Lymphatic spread of odontogenic infections occurs when microorganisms enter the lymphatic system and travel from a primary node near the infection site to a secondary node at a distant site. Lymphatic fluid travels through tubules interspersed with lymph nodes, ultimately draining into the venous system at the junction of the internal jugular and subclavian veins in the neck. Lymph nodes filter lymph and produce lymphocytes to combat infection. If the infection is contained in the primary node, it will not spread further. However, severe infections can spread through primary nodes to subsequent groups of nodes. Each group of nodes has the potential to contain the infection if it is not too severe.

Microbiology

The pathogenesis of odontogenic infections is polymicrobial, involving facultative anaerobes such as the Streptococcus viridans group and Streptococcus anginosus group, as well as strict anaerobes, including anaerobic cocci, Prevotella spp., and Fusobacterium spp(Robertson & Smith, 2009). Recent advancements in microbiological techniques, including strict anaerobic culturing methods, 16S rRNA gene sequencing, and polymerase chain reaction, have identified numerous hard-to-culture organisms, broadening the understanding of the microbial flora associated with dental infections(Riggio et al., 2007). Studies utilizing culture and molecular methods have identified over 460 unique bacterial taxa from 100 genera and nine phyla in various types of endodontic infections(Siqueira & Rôças, 2009). The predominant isolates are strictly anaerobic gram-negative rods and gram-positive cocci, with facultative and microaerophilic streptococci. Anaerobic bacteria outnumber aerobes by a ratio of 3:1. Culture and molecular studies confirm that apical abscess microbiota is mixed, with anaerobes dominating the population. Common anaerobes (75%) include Peptostreptococcus, Bacteroides, Prevotella spp., and Fusobacterium nucleatum. Aerobic species (25%) primarily consist of α-hemolytic streptococci.

Bacterial species frequently isolated in dental infections belong to seven bacterial phyla, with Firmicutes and Bacteroidetes accounting for over 70% of the species found in dental abscesses(Siqueira & Rôças, 2013):

1. **Firmicutes**: Streptococcus, Dialister, Filifactor, Pseudoramibacter
2. **Bacteroidetes**: Prevotella, Porphyromonas, Tannerella
3. **Fusobacteria**: Fusobacterium, Leptotrichia
4. **Actinobacteria**: Actinomyces, Propionibacterium
5. **Spirochaetes**: Treponema
6. **Synergistetes**: Pyramidobacter
7. **Proteobacteria**: Campylobacter, Eikenella

Studies have identified various bacterial mixtures within apical abscesses. One report noted that in acute infections, the predominant phyla were Firmicutes (52%), Fusobacteria (17%), Bacteroidetes (13%), and other mixed species (18%). Conversely, in chronic asymptomatic apical periodontitis, Firmicutes (59%), Bacteroidetes (14%), and Actinobacteria (10%) were dominant. Fusobacteria species were more prevalent in acute infections but decreased significantly in chronic cases. Acute abscesses exhibit greater bacterial diversity compared to chronic infections(Santos et al., 2011).

Among the various species, Prevotella spp. have been reported as the most frequently isolated, found in 10% to 87% of dentoalveolar abscesses (Shweta & Prakash, 2013). While clinical practice often seeks to identify specific pathogens or groups of pathogens associated with acute odontogenic infections, research has shown that no single pathogen is responsible. Instead, multispecies biofilm communities are involved, and the causative organisms are heterogeneous(Machado De Oliveira et al., 2007). The bacterial community profile, characterized by species richness and clinical relevance, provides insights into the dominant species within these communities. Culture studies report a mean number of 2 to 8.5 bacterial species per pus specimen.

Bacterial Load and Its Clinical Relevance

In clinical infections, the bacterial load, in addition to the type of bacteria, plays a crucial role. A significant bacterial load can lead to an overwhelming bacterial burden that may compromise the host's defense mechanisms. The total bacterial load in abscess cases has been reported to range between 10^4 and 10^9 cells[14]. A higher bacterial load is associated with increased diversity of organisms, potentially resulting in numerous synergistic interactions within the microbial community and an escalation in virulence factor production. Interactions among community members can modify the production of virulence factors. In polymicrobial infections, even species typically considered avirulent or present in low numbers may influence the virulence of other community members(Peters et al., 2012). The presence of a virulent strain in significant quantities can elevate the virulence of the entire community, leading to more severe infections. Clinical approaches such as tooth extraction, root canal therapy, incision and drainage, mechanical debridement, and thorough irrigation aim to reduce the bacterial load, thereby mitigating the infectious burden on the host.

Anaerobes

Anaerobic bacteria are implicated in nearly all dental infections. These organisms require anaerobic conditions to initiate and sustain growth. Typically, they lack catalase, although some can produce superoxide dismutase, which offers protection against oxygen. Strict anaerobes cannot grow in environments where oxygen

exceeds 0.5%, whereas moderate anaerobes can grow in conditions with 2% to 8% oxygen. Facultative anaerobes can grow in both aerobic conditions and anaerobic environments, though they proliferate more rapidly in the latter(Mosby, 2012). The microbial composition of primary odontogenic infections reflects the normal oral microbiota and the infection's dissemination pathway. Over time, ecological interactions and environmental conditions typically result in oral anaerobes becoming the predominant group in endodontic and periapical infections(Gaetti-Jardim et al., 2012). The presence of gram-negative anaerobic bacilli and anaerobic gram-positive cocci is often associated with acute symptoms, such as pain, sensitivity to pressure, and cellulitis. Anaerobic infections are characterized by abscess formation, foul-smelling pus, and tissue destruction.

Strict anaerobes alone cause about 20% of dental abscesses. While most infections are mixed, strict anaerobes usually outnumber facultative anaerobes by a ratio ranging from 1.5:1 to 3:1. Among these, strictly anaerobic gram-negative rods tend to be more pathogenic than facultative or strictly anaerobic gram-positive cocci.

Facultative Anaerobes

Facultative anaerobes primarily belong to the Streptococcus viridans group and the Streptococcus anginosus group. Staphylococcus aureus has also been identified in cases of acute dental abscesses.

Aerobes

Purely aerobic bacterial infections likely account for less than 5% of odontogenic infections. Aerobic bacteria involved in these infections are predominantly invasive Streptococcus species that form part of the normal endogenous flora. These aerobic streptococci are typically observed in the early stages of infection, resulting in cellulitic reactions. However, this phase rapidly transitions into a mixed infection dominated by anaerobic bacteria. Aerobic bacteria primarily serve as initiators of the infection, altering the local environment to facilitate anaerobic bacterial invasion as the affected tissue becomes more hypoxic, favoring anaerobic growth.

Diagnosis

Oral infections rank among the most common reasons for seeking healthcare worldwide. Diagnosing odontogenic infections is based on the chief complaint, the history of the current issue, clinical signs and symptoms, radiographic findings, and, when needed, obtaining specimens for culture. Most odontogenic infections can be diagnosed based on clinical history alongside physical and radiographic examination. Pain, often accompanied by swelling, is typically the primary complaint. The affected tooth is generally tender during acute infections. The history and examination should focus on the pain's location, type, frequency, duration, onset, and triggers, such as responses to heat or cold. Dental caries is the leading cause of odontogenic infections, with bacteria penetrating the pulp chamber, progressing through the root canal system, and reaching periradicular tissues. Examination may reveal a carious or nonvital tooth or an impacted tooth. Patients with superficial infections often report localized pain and sensitivity to pressure, percussion, and temperature. Conversely, individuals with deep infections or abscesses spreading to fascial planes may exhibit fever, trismus, and complaints of difficulty swallowing or breathing.

Acute Apical Abscess

Acute apical abscess refers to an inflammatory response to pulpal infection and necrosis. It is marked by rapid onset, spontaneous pain, extreme tenderness of the affected tooth to pressure, pus formation, and swelling of associated tissues. Radiographic evidence of destruction may be absent initially, but patients often present with malaise, fever, and lymphadenopathy.

Chronic Apical Abscess

Chronic apical abscess is an inflammatory response to pulpal infection and necrosis, characterized by a gradual onset, minimal discomfort, and intermittent pus discharge through a sinus tract. Radiographic signs of osseous destruction, such as radiolucency, are typically present. Identifying the source of a draining sinus tract involves placing a gutta-percha cone into the stoma until resistance is met, followed by radiographic imaging.

Radiographic Imaging

The choice of imaging technique depends on the clinical presentation. Panoramic radiographs of the jaws are considered the most suitable initial imaging method for odontogenic infections. Radiographs often reveal signs of infection in the supporting bone, such as periapical abscesses or impacted third molars in cases of pericoronitis. For severe infections involving deep spaces, computed tomography (CT) is the preferred modality. Magnetic resonance imaging (MRI), like CT, is effective for localizing deep fascial space infections in the head and neck. For conditions such as Ludwig's angina or infections spreading into the neck, posterior-anterior and lateral neck radiographs may assist in identifying tracheal compression, deviation, or gas presence within soft tissues. Similar information can be obtained via CT scans, though interpretation may be more challenging for practitioners less familiar with CT imaging.

Cultures

Due to the well-documented microbiology and antibiotic sensitivity of dental infections, initiating treatment with a known effective antibiotic without conducting cultures is considered reasonable. Performing cultures for all simple odontogenic infections would incur substantial economic costs. Additionally, there is

insufficient evidence to support the routine culturing of uncomplicated dental infections, as empiric antibiotic treatment often proves curative. In general, culturing is unnecessary for simple infections confined to the alveolar area unless the infection fails to respond to initial empiric therapy. For infections that extend to adjacent fascial spaces or occur in immunocompromised patients, obtaining needle aspirates or swab cultures for both aerobic and anaerobic analysis is recommended. Swab cultures, however, may be less effective for anaerobic organisms, as cotton fibers can damage these bacteria. Anaerobic culture specimens should be stored in specialized anaerobic collection tubes, often containing oxygen-free prereduced culture media and designed with airtight seals to maintain anaerobic conditions. For intraoral lesion cultures, contamination with resident oral flora is inevitable. Thus, needle aspiration of pus from an abscess cavity, preferably via an extraoral approach, is preferred where possible.

Treatment

The management of odontogenic infections depends on the infection's source, severity, and the patient's immune status. The primary goal in treatment is the elimination of the infection source, achieved through extraction of the affected tooth, surgical removal of diseased tissue, or extirpation of necrotic pulpal tissue followed by endodontic therapy. For acute abscesses, incision and drainage are necessary to remove purulent material containing bacteria. This procedure involves disrupting all loculations within the abscess cavity, evacuating pus, and using copious irrigation to reduce the bacterial population. For simple periapical abscesses, diseased tissue is removed from the pulp chamber, allowing drainage through the root canal.

Odontogenic infections can progress rapidly; therefore, prompt treatment is critical. Signs indicating the need for intervention by an oral and maxillofacial surgeon include:

- Midface swelling that causes eyelid closure
- Swelling extending below the mandibular border into the submandibular and submental spaces
- Significant swelling in the floor of the mouth
- Interincisal opening reduced to less than 20 mm
- Difficulty swallowing or breathing
- Neck swelling or erythema
- Headache or stiff neck
- Fever of 102°F (38.9°C) or higher
- Generalized weakness

Patients with airway compromise, systemic illness (fever, tachycardia, malaise), severe submandibular or submental swelling, severe trismus, or inability to tolerate oral fluids or medications require hospital admission. Such cases may necessitate intravenous (IV) antibiotics, abscess incision and drainage with possible external drain placement, and tooth extraction. Airway management through prolonged nasal intubation or tracheostomy may also be required.

Patients with compromised immune defenses exhibit an impaired response to infections. These conditions, such as diabetes, can exacerbate infection progression, alter microbiology, and increase susceptibility to invasive fungal infections, complicating antimicrobial treatment. Among such conditions, diabetes is the most encountered by dentists. The prevalence of type 2 diabetes has quadrupled globally between 1980 and 2015. Patients with diabetes are more susceptible to infections, and dental infections can worsen glycemic control, creating a hyperglycemic environment that impairs immune function and neutrophil activity. Furthermore, diabetes-related metabolic disturbances may facilitate and accelerate infections. Aggressive management of both hyperglycemia and infection is often necessary. Long-standing diabetes may lead to microvascular and macrovascular diseases, impairing tissue perfusion, which limits phagocytic cell access and hinders antibiotic efficacy.

In immunocompromised patients, underlying diseases may obscure clinical presentations. Careful monitoring of fever is essential as these patients are more prone to bacteremia, potentially leading to septicemia. Collaboration with specialists, such as internists, hematologists, endocrinologists, oncologists, and infectious disease experts, may be necessary. Surgical or endodontic interventions, in combination with antibiotics, are critical for treating such patients. IV antibiotics targeting oral flora may be required in certain cases.

Antibiotics

Antibiotics play a vital role in resolving dental infections but should not be considered a substitute for dental intervention. Removing the infection source remains the primary treatment objective, with antibiotics serving as adjunctive therapy. Oral and maxillofacial surgeons are trained to administer IV antibiotics in hospital settings, but general dental practitioners typically prescribe oral antibiotics, which are the focus here.

Given the established microbiology and antibiotic sensitivity of odontogenic pathogens, empiric treatment with effective antibiotics is a rational approach. Antimicrobial susceptibility tests for anaerobes often require extended timeframes, delaying treatment and increasing costs. In the early stages of clinical symptoms (3–4 days), facultative streptococci predominate; however, gram-negative obligate anaerobes increase in numbers in untreated cases exceeding 4 days. Accordingly, penicillin V is the preferred antibiotic during the initial 3–4 days, while clindamycin becomes the antibiotic of choice thereafter(Flynn et al., 1999).

Common orally administered antibiotics effective against odontogenic infections include:

- Penicillin
- Amoxicillin
- Clindamycin
- Metronidazole
- Azithromycin
- Moxifloxacin

Penicillin

Penicillin remains the preferred antibiotic for managing odontogenic infections due to its efficacy against gram-positive aerobes and intraoral anaerobes, both of which are commonly present in alveolar abscesses. It is effective against both aerobic and anaerobic microorganisms.

Amoxicillin

Amoxicillin, a semisynthetic antibiotic from the penicillin group, exhibits a broad spectrum of bactericidal activity against numerous gram-positive and gram-negative bacteria. While its spectrum of activity is broader than that of penicillin V, this does not necessarily translate to improved efficacy in treating odontogenic infections. Its dosing regimen, which allows administration twice daily (every 12 hours) or three times daily (every 8 hours), and its compatibility with food improve patient compliance. Therefore, amoxicillin is considered particularly suitable for noncompliant individuals.

Clindamycin

Clindamycin provides excellent coverage for gram-positive cocci and anaerobic bacteria, making it the antibiotic of choice for patients allergic to penicillin and for cases involving penicillin-resistant organisms. It functions by inhibiting bacterial protein synthesis and is bactericidal at higher doses (300 mg). Notably, the Sanford Guide to Antimicrobial Therapy has replaced penicillin with clindamycin as the recommended treatment for odontogenic infections(M. D. Gilbert et al., 2008, p. 20).

Metronidazole

Metronidazole disrupts DNA synthesis in microbial cells by inhibiting nucleic acid synthesis, a process that occurs only in anaerobic bacteria where the drug is partially reduced. As a result, metronidazole exhibits no activity against aerobic bacteria. Despite this limitation, it is highly effective against anaerobic organisms, which constitute the majority of bacteria in odontogenic infections. Clinically, it is particularly useful when combined with penicillin for treating severe acute odontogenic infections. Some studies suggest that the combination of penicillin and metronidazole should be regarded as the first-line therapy for odontogenic infections due to its excellent coverage of the mixed bacterial populations typically isolated from dental abscesses(Gregoire, 2010).

Azithromycin

Azithromycin, a macrolide antibiotic derived from erythromycin, represents a second-generation formulation with improved tolerance and tissue penetration. It is bacteriostatic and inhibits RNA-dependent bacterial protein synthesis. Azithromycin’s lipophilic nature facilitates high tissue penetration, particularly in inflamed areas, maintaining effective tissue concentrations for seven days after a three-day treatment regimen. It demonstrates good efficacy against aerobic and facultative gram-positive microorganisms (e.g., Staphylococci and Streptococci), anaerobes, and various atypical or rapidly growing pyogenic bacteria. This makes azithromycin effective for dental infections and a viable option for patients with type I hypersensitivity to penicillin. It is particularly useful when other antibiotics prove ineffective, for patients intolerant to penicillin, or for infections involving the sinuses. While its once-daily dosing improves compliance, azithromycin is not recommended as a first-line treatment for odontogenic infections. However, for pediatric infections, its short treatment duration, once-daily dosing, and acceptable taste make it an appealing choice.

Moxifloxacin

Moxifloxacin, a fourth-generation synthetic fluoroquinolone, has a broad spectrum of activity and is particularly effective against Eikenella, Bacteroides, Prevotella, and other β-lactamase-producing bacterial strains. This makes it an excellent option for treating infections unresponsive to penicillins. Moxifloxacin has been reported to demonstrate the highest bacterial susceptibility rates among all antibiotics, including penicillin and clindamycin, for odontogenic infections(Kuriyama et al., 2007; Warnke et al., 2008). Despite its efficacy, it is generally considered a second-line treatment option after penicillin, clindamycin, and metronidazole, primarily due to its cost (approximately $140 for a seven-day course). Additionally, moxifloxacin may pose risks to bones, joints, and surrounding tissues in pediatric patients and is contraindicated for children under 18 years of age.

Conclusion

Odontogenic infections, though common, encompass a wide spectrum of clinical presentations and potential complications that require timely and effective management. From localized abscesses to life-threatening conditions such as Ludwig's angina, understanding the pathogenesis, microbiology, and mechanisms of infection spread is paramount. The interplay between bacterial virulence, host defense mechanisms, and anatomical factors underscores the complexity of these infections.

Effective treatment begins with addressing the primary source of infection through surgical intervention or endodontic therapy, complemented by appropriate antibiotic therapy. While penicillin remains a cornerstone of treatment, alternatives such as clindamycin, metronidazole, and amoxicillin play critical roles, particularly in cases of penicillin resistance or allergy. Empiric antibiotic therapy, guided by established microbiological profiles, has proven effective in most cases, though culture and sensitivity testing are crucial in refractory or severe infections.

For immunocompromised patients, the challenges of managing odontogenic infections are amplified, necessitating multidisciplinary collaboration and aggressive intervention to mitigate systemic complications. Advances in imaging and molecular diagnostics continue to enhance the understanding and management of these infections, contributing to improved patient outcomes.

Ultimately, the successful management of odontogenic infections hinges on a comprehensive approach that integrates clinical expertise, timely intervention, and patient-specific considerations. As research continues to evolve, the dental and medical community must remain vigilant in adopting evidence-based practices to combat these infections effectively and minimize their associated risks

References

Flynn, T., Wiltz, M., Adamo, A., Levy, M., McKitrick, J., Freeman, K., & Trieger, N. (1999). Predicting length of hospital stay and pencillin failure in severe odontogenic infections. *International Journal of Oral and Maxillofacial Surgery*, *28*, 48. https://doi.org/10.1016/S0901-5027(99)80799-1

Gaetti-Jardim, E., Landucci, L. F., De Oliveira, K. L., Costa, I., Ranieri, R. V., Okamoto, A. C., & Schweitzer, C. M. (2012). Microbiota associated with infections of the jaws. *International Journal of Dentistry*. Scopus. https://doi.org/10.1155/2012/369751

Gregoire, C. (2010). Point of care: How are odontogenic infections best managed? *Journal of the Canadian Dental Association*, *76*(2), 114–116. Scopus.

Kim, M. K., Allareddy, V., Nalliah, R. P., Kim, J. E., & Allareddy, V. (2012). Burden of facial cellulitis: Estimates from the Nationwide Emergency Department Sample. *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology*, *114*(3), 312–317. https://doi.org/10.1016/j.tripleo.2011.07.043

Kuriyama, T., Williams, D. W., Yanagisawa, M., Iwahara, K., Shimizu, C., Nakagawa, K., Yamamoto, E., & Karasawa, T. (2007). Antimicrobial susceptibility of 800 anaerobic isolates from patients with dentoalveolar infection to 13 oral antibiotics. *Oral Microbiology and Immunology*, *22*(4), 285–288. Scopus. https://doi.org/10.1111/j.1399-302X.2007.00365.x

M. D. Gilbert, D. N., M. D. Moellering, R. C., Eliopoulos, G. M., & Sande, M. A. (2008). *The Sanford Guide to Antimicrobial Therapy, 2008* (38th edition). Antimicrobial Therapy.

Machado De Oliveira, J. C., Siqueira Jr., J. F., Rôças, I. N., Baumgartner, J. C., Xia, T., Peixoto, R. S., & Rosado, A. S. (2007). Bacterial community profiles of endodontic abscesses from Brazilian and USA subjects as compared by denaturing gradient gel electrophoresis analysis. *Oral Microbiology and Immunology*, *22*(1), 14–18. Scopus. https://doi.org/10.1111/j.1399-302X.2007.00311.x

Mihos, P., Potaris, K., Gakidis, I., Papadakis, D., & Rallis, G. (2004). Management of descending necrotizing mediastinitis. *Journal of Oral and Maxillofacial Surgery*, *62*(8), 966–972. https://doi.org/10.1016/j.joms.2003.08.039

Mosby. (2012). *Mosby's Medical Dictionary - E-Book: Mosby's Medical Dictionary - E-Book*. Elsevier Health Sciences.

Peters, B. M., Jabra-Rizk, M. A., O'May, G. A., William Costerton, J., & Shirtliff, M. E. (2012). Polymicrobial interactions: Impact on pathogenesis and human disease. *Clinical Microbiology Reviews*, *25*(1), 193–213. Scopus. https://doi.org/10.1128/CMR.00013-11

Riggio, M. P., Aga, H., Murray, C. A., Jackson, M. S., Lennon, A., Hammersley, N., & Bagg, J. (2007). Identification of bacteria associated with spreading odontogenic infections by 16S rRNA gene sequencing. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, *103*(5), 610–617. https://doi.org/10.1016/j.tripleo.2006.08.009

Robertson, D., & Smith, A. J. (2009). The microbiology of the acute dental abscess. *Journal of Medical Microbiology*, *58*(2), 155–162. https://doi.org/10.1099/jmm.0.003517-0

Santos, A. L., Siqueira, J. F., Rôças, I. N., Jesus, E. C., Rosado, A. S., & Tiedje, J. M. (2011). Comparing the bacterial diversity of acute and chronic dental root canal infections. *PloS One*, *6*(11), e28088. https://doi.org/10.1371/journal.pone.0028088

Santosh, A. N., Viresh, A. N., & Sharmada, B. K. (2014). Microbiology and Antibiotic Sensitivity of Odontogenic Space Infection. *International Journal of Medical and Dental Sciences*, *3*(1), 303. https://doi.org/10.19056/ijmdsjssmes/2014/v3i1/80675

Shweta, & Prakash, S. K. (2013). Dental abscess: A microbiological review. *Dental Research Journal*, *10*(5), 585–591.

Siqueira, J. F., & Rôças, I. N. (2009). Diversity of Endodontic Microbiota Revisited. *Journal of Dental Research*, *88*(11), 969–981. https://doi.org/10.1177/0022034509346549

Siqueira, J. F., & Rôças, I. N. (2013). Microbiology and treatment of acute apical abscesses. *Clinical Microbiology Reviews*, *26*(2), 255–273. Scopus. https://doi.org/10.1128/CMR.00082-12

Warnke, P. H., Becker, S. T., Springer, I. N. G., Haerle, F., Ullmann, U., Russo, P. A. J., Wiltfang, J., Fickenscher, H., & Schubert, S. (2008). Penicillin compared with other advanced broad spectrum antibiotics regarding antibacterial activity against oral pathogens isolated from odontogenic abscesses. *Journal of Cranio-Maxillofacial Surgery*, *36*(8), 462–467. https://doi.org/10.1016/j.jcms.2008.07.001