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Maxillofacial Tubercular Osteomyelitis: Clinical Manifestations, Radiological Characteristics, Diagnostic Challenges, and Multidisciplinary Dental Management-An Updated Review.

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Abstract

Background: Maxillofacial tubercular osteomyelitis is a rare manifestation of extrapulmonary tuberculosis that presents nonspecific clinical and radiological features. Its resemblance to odontogenic infections, pyogenic osteomyelitis, and malignant lesions frequently delays diagnosis and appropriate treatment.

Aim: This review examines the clinical manifestations, radiological characteristics, diagnostic challenges, and therapeutic considerations associated with maxillofacial tubercular osteomyelitis, with particular emphasis on the complementary roles of dentistry and radiology.

Methods: A narrative review of the available literature was conducted to evaluate reported clinical presentations, imaging findings, histopathological features, microbiological investigations, molecular diagnostic methods, and treatment approaches. Particular attention was given to diagnostic limitations and strategies for reducing delays in clinical management.

Results: Patients may present with persistent jaw swelling, pain, sinus formation, tooth mobility, and progressive bone destruction. Conventional radiography, computed tomography, cone-beam computed tomography, and magnetic resonance imaging help characterize lesion extent but cannot reliably distinguish tuberculosis from other infectious or neoplastic conditions. Histopathology provides important diagnostic evidence, although non-caseating granulomas and nonspecific inflammation may occur. Acid-fast bacilli staining and mycobacterial culture have limited sensitivity, supporting the use of molecular methods such as Xpert MTB/RIF and polymerase chain reaction. Treatment primarily involves antitubercular therapy, with surgical biopsy and debridement indicated in selected cases.

Conclusion: Early clinical suspicion coordinated radiological assessment, and combined histopathological, microbiological, and molecular investigations are essential for timely diagnosis. An integrated multidisciplinary approach can reduce diagnostic delays, prevent unnecessary surgery, and improve treatment outcomes.

Keywords: Maxillofacial tuberculosis; tubercular osteomyelitis; jaw osteomyelitis; dental radiology; molecular diagnosis; antitubercular therapy.

Introduction

Maxillofacial tuberculosis (TB) represents an uncommon manifestation of extrapulmonary tuberculosis and remains a diagnostic challenge in dental, maxillofacial, and radiological practice. Although tuberculosis predominantly affects the lungs, its ability to involve various extrapulmonary sites allows it to present in multiple anatomical forms, including the oral cavity, facial soft tissues, jaws, and temporomandibular joint (TMJ). Maxillofacial involvement is consistently reported as rare, which contributes to its limited consideration in the initial differential diagnosis of patients presenting with chronic jaw infections, destructive bone lesions, or persistent facial swelling [1,2,3,4,5]. Orofacial tuberculosis constitutes only a small proportion of all tuberculosis cases, while primary orofacial involvement, occurring without an identifiable

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pulmonary or systemic source, represents an even smaller percentage [6,7]. Similarly, tuberculous osteomyelitis accounts for a relatively minor proportion of skeletal tuberculosis, with involvement of the maxillary or mandibular bones being particularly uncommon [8,9]. Consequently, the limited clinical familiarity with this condition may contribute to delayed recognition, inappropriate initial treatment, and prolonged diagnostic investigations. Despite its low incidence, maxillofacial tubercular osteomyelitis has considerable clinical significance because of its ability to resemble several common infectious, inflammatory, and neoplastic conditions. The clinical manifestations are frequently nonspecific and may include persistent jaw pain, localized swelling, tenderness, restricted mouth opening, mucosal ulceration, draining sinuses, tooth mobility, and delayed wound healing. In some patients, the disease develops gradually and presents as a chronic inflammatory lesion without the classical constitutional manifestations of tuberculosis, such as fever, night sweats, weight loss, or persistent cough. This absence of characteristic systemic symptoms may lead clinicians to prioritize odontogenic infections, periodontal disease, chronic bacterial osteomyelitis, or malignant lesions over tuberculosis. Furthermore, mandibular involvement may resemble chronic suppurative osteomyelitis, infected odontogenic cysts, or a conventional dentoalveolar abscess, particularly when the patient presents with localized pain, swelling, and sinus tract formation [10,11].

Radiological assessment introduces additional diagnostic difficulties because the imaging characteristics of tuberculous osteomyelitis are not specific enough to establish the diagnosis independently. Conventional radiography may demonstrate ill-defined radiolucent areas, cortical destruction, irregular bone resorption, or mixed radiolucent and sclerotic changes. These findings may overlap with those observed in pyogenic osteomyelitis, odontogenic lesions, primary bone tumors, and metastatic disease. Computed tomography (CT) provides valuable information regarding cortical involvement, sequestrum formation, periosteal reactions, and the extent of osseous destruction, whereas magnetic resonance imaging (MRI) can help identify bone marrow abnormalities and associated soft-tissue extension. Cone-beam computed tomography (CBCT) also has an important role in dental and maxillofacial assessment because it provides detailed visualization of mandibular and maxillary structures. Nevertheless, none of these imaging modalities can reliably differentiate tuberculous infection from other inflammatory or neoplastic processes without clinical and laboratory correlation. Temporomandibular joint involvement represents another challenging presentation of maxillofacial tuberculosis. Patients may experience preauricular pain, joint swelling, tenderness, restricted mandibular movement, and difficulty during mastication. These manifestations may resemble degenerative joint disease, inflammatory arthritis, septic arthritis, primary TMJ osteomyelitis, or neoplastic disorders [2]. Consequently, radiologists and dental clinicians must consider tuberculosis when evaluating persistent or progressive TMJ abnormalities, particularly when conventional treatment fails to produce clinical improvement.

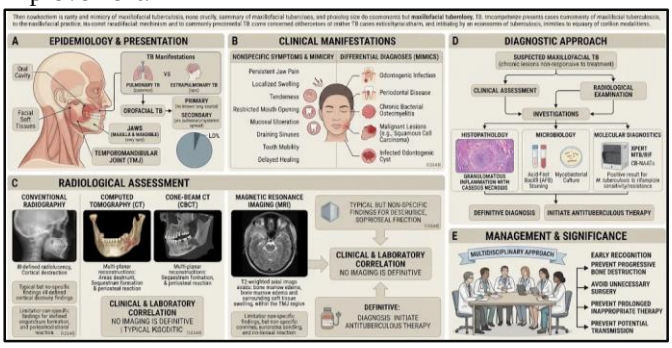


Figure-1: Maxillofacial tuberculosis (TB) Overview.

Definitive diagnosis requires an integrated approach combining clinical assessment, radiological examination, and microbiological or histopathological investigations. Fine-needle aspiration cytology (FNAC) may provide useful preliminary information, although inconclusive findings can occur in head and neck tuberculosis. Histopathological examination demonstrating granulomatous inflammation, particularly with caseous necrosis, can support the diagnosis. However, confirmation should be pursued through appropriate microbiological testing whenever possible. Acid-fast bacilli staining, mycobacterial culture, polymerase chain reaction (PCR), and molecular diagnostic techniques provide complementary information. In particular, Xpert MTB/RIF and other cartridge-based nucleic acid amplification tests (CB-NAATs) can facilitate the detection of Mycobacterium tuberculosis and identify rifampicin resistance, potentially reducing diagnostic delays and supporting timely treatment decisions [11,12,13]. Early recognition of maxillofacial tubercular osteomyelitis is important to prevent progressive bone destruction, unnecessary surgical interventions, prolonged inappropriate antimicrobial therapy, and potential transmission of infection. Effective management requires coordination between dentists, oral and maxillofacial surgeons, radiologists, pathologists, microbiologists, and physicians specializing in infectious diseases. This multidisciplinary approach facilitates accurate interpretation of clinical and imaging findings, appropriate selection of diagnostic specimens, confirmation of the underlying infection, and initiation of antituberculous therapy.

Clinical Presentation and Diagnostic Pitfalls

Maxillofacial tubercular osteomyelitis exhibits a broad range of clinical manifestations, although its most common presentation is characterized by a chronic inflammatory process involving the maxillary or mandibular bones. Patients typically present with persistent localized swelling, jaw pain, tenderness, tooth mobility, and the formation of draining sinus tracts. These manifestations may develop gradually and persist for weeks or months before the underlying cause is identified. In many cases, the clinical presentation closely resembles odontogenic infections, periodontal disease, dentoalveolar abscesses, or chronic pyogenic osteomyelitis, making clinical differentiation particularly challenging [9,10]. The absence of characteristic systemic manifestations of tuberculosis further complicates recognition, as some patients present with localized maxillofacial disease without fever, weight loss, night sweats, or respiratory symptoms. One of the most important diagnostic pitfalls is the tendency to attribute persistent jaw lesions to common dental conditions without considering less frequent infectious etiologies. This diagnostic anchoring may result in repeated prescriptions of conventional antibiotics, dental extraction, incision and drainage, or other routine interventions without addressing the underlying mycobacterial infection. Although these procedures may temporarily modify local inflammatory manifestations, they generally fail to achieve sustained resolution when tuberculosis remains untreated. Persistent symptoms despite appropriate conventional antibacterial therapy should therefore prompt clinicians to reconsider the initial diagnosis and investigate alternative causes, including tuberculous osteomyelitis [4,9]. Furthermore, patients may have no previously established diagnosis of tuberculosis or may not disclose a history of systemic infection. Consequently, the absence of a documented history of pulmonary TB should not be interpreted as evidence against maxillofacial tuberculosis [2,9,12,23].

The diagnostic complexity extends beyond the clinical manifestations of bone infection because tuberculosis can involve adjacent anatomical structures and produce findings that resemble diseases originating from neighboring tissues. Mandibular tuberculosis, for example, may present with swelling around the angle of the mandible or the preauricular region, creating the impression of parotid gland pathology. In some reported cases, tuberculous lesions have been mistaken for parotid abscesses, salivary gland tumors, or persistent parotid fistulas. Such misinterpretation may expose patients to unnecessary surgical procedures, including superficial parotidectomy, before the infectious etiology is recognized [12,24]. These observations emphasize the importance of assessing the anatomical relationship between the lesion, the mandibular bone, surrounding soft tissues, and salivary structures during clinical and radiological evaluation. Temporomandibular joint (TMJ) and mandibular condyle involvement represent particularly challenging presentations because their symptoms are frequently nonspecific. Patients may report preauricular pain, tenderness, swelling, restricted mouth opening, difficulty chewing, and reduced mandibular movement. These findings may resemble temporomandibular disorders, inflammatory arthritis, septic arthritis, degenerative joint disease, or malignant involvement of the joint and adjacent bone. In addition, patients may initially seek consultation from otolaryngologists because of ear pain or symptoms resembling chronic otitis, resulting in diagnostic investigations that do not initially focus on the mandibular condyle or TMJ [2,3]. The possibility of tuberculous involvement should therefore be considered when persistent joint symptoms are associated with progressive bone abnormalities or fail to respond to conventional treatment.

Radiological findings may contribute to these diagnostic difficulties because the appearance of tuberculous lesions varies according to disease duration, anatomical location, and the extent of bone and soft-tissue involvement. Areas of osteolysis, irregular cortical destruction, poorly defined bone margins, and associated soft-tissue abnormalities may suggest chronic osteomyelitis or an aggressive neoplastic process. Therefore, imaging findings should be interpreted alongside the clinical history, laboratory investigations, and tissue examination rather than being considered independently diagnostic of tuberculosis. A high index of clinical suspicion is particularly important in patients presenting with chronic jaw swelling, unexplained tooth mobility, persistent draining sinuses, recurrent maxillofacial abscesses, or destructive lesions that remain unresolved despite conventional dental and antimicrobial interventions. Additional attention is warranted when imaging demonstrates progressive bone destruction without a convincing odontogenic source. In such circumstances, obtaining appropriate tissue or aspirated specimens for histopathological examination, acid-fast bacilli detection, mycobacterial culture, and molecular testing may help establish the diagnosis. Early consideration of tuberculosis can reduce unnecessary procedures, prevent prolonged ineffective treatment, and facilitate timely initiation of appropriate antituberculous therapy.

Radiological Findings and Limitations

Radiological assessment plays a fundamental role in the evaluation of maxillofacial tubercular osteomyelitis, providing essential information about the anatomical distribution, extent, and severity of osseous involvement. Imaging techniques also help identify associated soft-tissue abnormalities, evaluate adjacent anatomical structures, guide tissue sampling, and support

treatment planning. Despite these advantages, radiological findings in tuberculous osteomyelitis are generally nonspecific and cannot independently establish the infectious etiology. The imaging appearance frequently overlaps with that of chronic pyogenic osteomyelitis, odontogenic infections, inflammatory bone disorders, and malignant lesions, creating considerable diagnostic uncertainty [2,7]. Conventional radiography remains an initial investigation in patients presenting with suspected jaw pathology. Panoramic radiographs may demonstrate poorly defined radiolucent areas, diffuse bone rarefaction, alterations in the normal trabecular architecture, and varying degrees of cortical destruction. These abnormalities reflect progressive inflammatory involvement and bone resorption but do not represent characteristic findings exclusive to tuberculosis. In some patients, the radiographic appearance may resemble suppurative osteomyelitis or an aggressive malignant process, particularly when extensive osteolysis and irregular bone margins are present [4,9]. Furthermore, early disease may produce limited or subtle radiographic abnormalities, making conventional imaging insufficient for excluding tuberculous involvement when clinical suspicion remains high.

Panoramic imaging and cone-beam computed tomography (CBCT) have particular relevance in dental and maxillofacial practice. Although panoramic radiography provides an overall assessment of the jaws and dentoalveolar structures, CBCT offers three-dimensional visualization of the affected bone. This capability allows more detailed assessment of the anatomical distribution of osteolytic lesions, cortical integrity, trabecular alterations, and the relationship between the lesion and adjacent teeth. CBCT may also demonstrate the presence of cortical perforation, irregular bone destruction, and sequestration, which can influence decisions regarding biopsy and surgical intervention. In mandibular tuberculous osteomyelitis, diffuse bone rarefaction may be evident; however, such changes lack a distinctive radiological pattern that reliably differentiates tuberculosis from other inflammatory conditions [2,9,12]. Computed tomography (CT) provides additional diagnostic information, particularly when the infection extends beyond the mandibular or maxillary bone into surrounding soft tissues. It is useful for demonstrating cortical erosion, osseous destruction, periosteal abnormalities, and associated inflammatory collections. In patients with temporomandibular joint (TMJ) or mandibular condyle involvement, CT may reveal extensive erosive changes, joint destruction, and adjacent abscess formation. Contrast-enhanced CT can further demonstrate enhancing inflammatory collections extending into neighboring muscles and other soft-tissue compartments. Nevertheless, these findings may closely resemble those of conventional bacterial osteomyelitis, and the radiological appearance alone may lead to an incorrect initial diagnosis of pyogenic infection [3,7]. Therefore, CT findings should be interpreted in conjunction with the patient's clinical presentation and microbiological investigations.

Magnetic resonance imaging (MRI) represents a valuable complementary technique, particularly in patients with suspected early disease, inconclusive conventional radiographs, or extensive soft-tissue involvement. Unlike radiography and CBCT, which primarily demonstrate mineralized bone structures, MRI provides superior visualization of bone marrow abnormalities and surrounding soft tissues. Tuberculous infection may produce altered marrow signal intensity, inflammatory changes, and associated soft-tissue abnormalities. MRI can also help determine the extent of disease involving the masticatory muscles, temporomandibular joint, and adjacent anatomical compartments. Its ability to identify early marrow changes makes it particularly useful when conventional radiographs appear normal or demonstrate only minimal abnormalities [15,25]. Despite the availability of advanced imaging modalities, several limitations remain important in clinical practice. First, no single radiological feature is sufficiently specific to confirm maxillofacial tuberculosis. Second, different imaging techniques demonstrate overlapping abnormalities among infectious, inflammatory, and neoplastic disorders. Third, radiological interpretation may be influenced by the anatomical location and stage of the disease, potentially resulting in variable findings between patients. In some cases, the imaging appearance may suggest odontogenic tumors, primary bone malignancies, or metastatic lesions without providing sufficient evidence to establish the underlying diagnosis [2,7].

Accordingly, radiological investigations should be considered an essential component of a multidisciplinary diagnostic process rather than a substitute for histopathological and microbiological confirmation. The selection of imaging modality should depend on the clinical presentation, suspected anatomical involvement, and diagnostic objectives. Conventional radiography and panoramic imaging may provide an initial overview, whereas CBCT offers detailed assessment of osseous destruction. CT is particularly valuable for evaluating cortical abnormalities and associated collections, while MRI contributes to the assessment of early marrow involvement and soft-tissue extension. Ultimately, correlation of imaging findings with histopathological examination, acid-fast bacilli detection, mycobacterial culture, and molecular diagnostic testing is necessary to distinguish tuberculous osteomyelitis from other maxillofacial diseases and establish an appropriate treatment strategy.

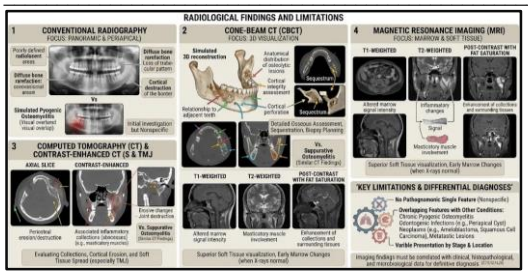


Figure-2: Radiological Examination of Maxillofacial TB.

Histopathological Challenges

Histopathological examination represents a fundamental component of the diagnostic evaluation of maxillofacial tubercular osteomyelitis, particularly in cases where clinical manifestations and radiological findings overlap with those of other infectious and neoplastic conditions. The distinction between tuberculous and pyogenic osteomyelitis may be difficult to establish through clinical assessment and imaging alone. Consequently, tissue examination provides valuable information about the underlying inflammatory process and may identify characteristic granulomatous changes that support a diagnosis of tuberculosis. Histopathological evaluation is particularly important in patients presenting with persistent jaw lesions, unexplained bone destruction, recurrent abscesses, or lesions that fail to respond to conventional antibacterial treatment [10]. The classical histopathological appearance of tuberculous infection is characterized by granulomatous inflammation, consisting of epithelioid histiocytes, lymphocytes, and multinucleated Langhans-type giant cells. Caseous necrosis may be present within the center of the granulomas and represents a characteristic finding associated with tuberculosis. However, the histological appearance is not consistent across all patients, and the absence of caseous necrosis should not be considered sufficient evidence to exclude tuberculous infection. Some specimens demonstrate non-caseating granulomas containing epithelioid cells and Langhans-type giant cells without the typical necrotic component. This variability may complicate interpretation, particularly when the clinical history does not initially suggest tuberculosis [8].

Another important diagnostic limitation involves the possibility of false-negative or inconclusive biopsy findings. Initial incisional biopsies may demonstrate only nonspecific chronic inflammatory changes, including lymphocytic infiltration, inflammatory granulation tissue, and areas of tissue destruction, without identifiable granulomatous inflammation. Such findings may lead clinicians to favor alternative diagnoses and postpone further investigations for tuberculosis. Sampling errors may contribute to these outcomes because the selected tissue may not contain the active granulomatous component of the lesion. Furthermore, variations in disease progression, lesion distribution, and the presence of secondary bacterial infection may influence the histological characteristics of the collected specimens [9,11]. The selection of an appropriate biopsy site is therefore an important consideration in the diagnostic process. Whenever possible, tissue should be obtained from representative areas of the lesion, particularly regions demonstrating active inflammatory changes on clinical examination or radiological assessment. In patients with extensive bone involvement, sampling only superficial necrotic tissue may provide insufficient diagnostic information. Coordinating biopsy procedures with radiological findings can improve specimen selection and increase the likelihood of obtaining representative material. In addition, adequate tissue samples should be allocated for both histopathological examination and microbiological investigations because placing the entire specimen in formalin may prevent subsequent mycobacterial culture or certain molecular diagnostic procedures.

Fine-needle aspiration cytology (FNAC) provides a relatively accessible and cost-effective preliminary diagnostic approach, particularly in healthcare settings where advanced diagnostic facilities are limited. It may be useful for evaluating patients with localized swelling, suspected abscess formation, or associated soft-tissue lesions. Cytological examination may reveal epithelioid histiocytes, multinucleated giant cells, and lymphocytic infiltration, suggesting granulomatous inflammation. Nevertheless, these findings are not specific to tuberculosis and may also occur in other infectious and inflammatory disorders. Furthermore, aspirated material may contain predominantly inflammatory cells, necrotic debris, or histiocytes without demonstrating well-formed granulomas, thereby limiting diagnostic certainty [3]. Examination of pus or aspirated material may occasionally provide additional diagnostic clues. However, the identification of epithelioid cells or histiocytes without characteristic granulomatous organization should not be interpreted as definitive evidence for or against tuberculosis. In such circumstances, persistent clinical suspicion warrants further investigation through an incisional or excisional tissue biopsy. This staged diagnostic approach helps minimize premature diagnostic conclusions and reduces the possibility of overlooking an uncommon mycobacterial infection [26]. Importantly, histopathological findings should be correlated with microbiological and molecular investigations. Ziehl–Neelsen staining or other appropriate acid-fast staining techniques may demonstrate acid-fast bacilli, although negative staining does not exclude tuberculosis because the number of organisms in extrapulmonary lesions may be limited. Mycobacterial culture provides an opportunity to identify the causative organism and assess drug susceptibility, while polymerase chain reaction (PCR) and cartridge-based nucleic acid

amplification tests can support more rapid detection of Mycobacterium tuberculosis. Therefore, the diagnosis of maxillofacial tubercular osteomyelitis should not depend exclusively on a single histopathological finding. A combination of representative tissue sampling, cytological or histological assessment, microbiological testing, molecular investigations, and radiological correlation provides a more reliable diagnostic strategy. Recognizing the limitations of biopsy and FNAC is particularly important in patients with chronic, progressive, or treatment-resistant jaw lesions, where repeated or complementary investigations may be necessary to establish the diagnosis and initiate appropriate antituberculous treatment.

Conventional Microbiology and Sensitivity Limitations

Conventional microbiological investigations play an important role in confirming maxillofacial tuberculosis; however, their diagnostic performance is affected by several limitations, particularly the low bacterial burden frequently encountered in extrapulmonary lesions. Tuberculous osteomyelitis of the jaws is often paucibacillary, meaning that infected tissues may contain relatively small numbers of Mycobacterium tuberculosis organisms. This characteristic reduces the likelihood of detecting the pathogen through routine microbiological methods and contributes to false-negative results. Acid-fast bacilli (AFB) smear microscopy, although widely available and relatively inexpensive, has limited sensitivity in extrapulmonary specimens, with reported detection rates ranging from approximately 0% to 40%. Consequently, a negative smear result cannot reliably exclude maxillofacial tuberculosis, particularly when clinical, radiological, or histopathological findings remain suggestive of the disease [9]. An important diagnostic pitfall occurs when clinicians interpret negative routine microbiological investigations as sufficient evidence against tuberculosis. Patients with maxillofacial involvement may have negative AFB smears and normal chest radiographs, particularly when the disease is confined to extrapulmonary sites. These findings should not discourage further investigation when persistent jaw lesions, granulomatous inflammation, or unexplained bone destruction raise suspicion of tuberculosis. Reliance on conventional screening results without considering the clinical presentation may delay definitive diagnosis and appropriate treatment [3].

Mycobacterial culture remains an important reference method for confirming tuberculosis and evaluating antimicrobial susceptibility. Löwenstein–Jensen medium is commonly used for the isolation of Mycobacterium tuberculosis from clinical specimens. However, culture-based diagnosis has important practical limitations, particularly the prolonged time required for bacterial growth. Depending on the specimen, organism burden, and laboratory conditions, culture results may require approximately 15 days to five weeks or longer. Furthermore, cultures may remain negative despite the presence of infection, especially when specimens contain insufficient viable organisms or are inadequately collected or transported [11]. These limitations have important implications for the management of patients with suspected maxillofacial tubercular osteomyelitis. Waiting exclusively for conventional culture results may prolong diagnostic uncertainty and postpone appropriate therapeutic decisions. Therefore, microbiological investigations should be incorporated into a broader diagnostic strategy that includes histopathological examination and molecular techniques, such as polymerase chain reaction and Xpert MTB/RIF. Combining these approaches can improve diagnostic yield, facilitate earlier identification of tuberculosis, and provide information regarding drug resistance. Ultimately, negative smear microscopy or culture findings should always be interpreted in the context of clinical suspicion and other available diagnostic evidence.

Molecular Diagnostics

Molecular diagnostic techniques have become important tools in the investigation of maxillofacial tubercular osteomyelitis, particularly because conventional microbiological methods frequently fail to detect Mycobacterium tuberculosis in paucibacillary extrapulmonary lesions. Among these techniques, Xpert MTB/RIF, a cartridge-based nucleic acid amplification test (CB-NAAT), offers a rapid approach for detecting mycobacterial genetic material and identifying mutations associated with rifampicin resistance. Unlike conventional acid-fast bacilli microscopy, which depends on the presence of sufficient numbers of organisms in the examined specimen, molecular testing can detect specific bacterial DNA, thereby improving the opportunity for microbiological confirmation in patients with suspected extrapulmonary tuberculosis [9,27]. Xpert MTB/RIF provides clinically relevant information by identifying the presence of Mycobacterium tuberculosis and simultaneously assessing rifampicin resistance through the detection of mutations in the rpoB gene. This information is particularly important because rifampicin resistance may indicate drug-resistant tuberculosis requiring additional investigations and modifications to the therapeutic regimen. The relatively short processing time also represents a major advantage over conventional mycobacterial culture, which may require several weeks before providing definitive results. Reported pooled diagnostic performance for extrapulmonary tuberculosis includes a sensitivity of approximately 83.1% and a specificity of 98.7%, although these estimates vary according to specimen type, disease location, and the reference standard used [27]. The World Health Organization (WHO) recognizes rapid

molecular diagnostic techniques as important components of contemporary tuberculosis diagnostic pathways [28]. Quantitative polymerase chain reaction (qPCR) represents another valuable molecular approach for detecting Mycobacterium tuberculosis DNA in tissue specimens obtained from patients with suspected maxillofacial infection. Depending on the assay and laboratory procedures, molecular amplification and analysis may provide diagnostic information within several hours. In selected settings, qPCR-based investigations have been reported to establish a diagnosis in approximately eight hours, offering an opportunity to reduce delays associated with conventional culture methods [11]. These techniques are particularly useful when histopathological examination demonstrates granulomatous inflammation but fails to identify acid-fast bacilli.

The diagnostic yield of molecular investigations is strongly influenced by specimen selection and collection techniques. Pus samples and representative tissue biopsies may provide better diagnostic opportunities than specimens containing limited numbers of aspirated cells. In particular, insufficient cellular material or low bacterial concentrations may contribute to false-negative results in aspirated specimens. Therefore, lesion-directed sampling is essential to improve the probability of detecting mycobacterial DNA. The selection of an appropriate biopsy site should be guided by clinical examination and radiological findings, with particular attention to areas demonstrating active inflammation or progressive bone destruction [13]. Additional technical considerations are relevant when performing molecular testing on bone specimens. Bone mineral components, tissue processing procedures, and formalin fixation may interfere with DNA extraction or amplification, potentially reducing assay performance. Short-segment amplification strategies, including the use of approximately 90-base-pair targets, have been suggested to improve the detection of mycobacterial DNA in challenging bone specimens and partially address amplification difficulties associated with calcium-containing tissues or paraffin-embedded material [11]. Despite their advantages, molecular diagnostic methods should not be considered completely independent of clinical and histopathological assessment. False-negative results may occur because of inadequate sampling, low organism burden, or technical limitations. Furthermore, detection of bacterial DNA does not necessarily establish the presence of viable organisms. Accordingly, combining Xpert MTB/RIF or qPCR with histopathological examination, mycobacterial culture, and radiological assessment provides a more comprehensive diagnostic approach. This integrated strategy can facilitate earlier recognition of maxillofacial tuberculosis, identify potential rifampicin resistance, and support timely initiation of appropriate antituberculous treatment.

Immunological Tests

Immunological investigations, including the Mantoux tuberculin skin test and interferon-gamma release assays (IGRAs), may provide supportive evidence of exposure to Mycobacterium tuberculosis in patients presenting with suspected maxillofacial tuberculosis. However, these tests cannot independently confirm active tuberculous osteomyelitis or distinguish localized infection from latent tuberculosis. Chest radiography is also useful for evaluating possible pulmonary involvement and identifying additional evidence of systemic disease. Nevertheless, patients with isolated maxillofacial tuberculosis may demonstrate normal chest radiographs and negative immunological screening results despite subsequently confirmed infection [2,8]. These limitations highlight the importance of interpreting immunological findings within the broader clinical context. A negative Mantoux test or IGRA should not be considered sufficient to exclude tuberculosis when clinical manifestations, radiological abnormalities, or histopathological findings remain suspicious. Accordingly, persistent diagnostic suspicion should prompt further investigations, including tissue biopsy, mycobacterial culture, and molecular testing, to establish the diagnosis and guide treatment [3,9].

Discussion

Maxillofacial tubercular osteomyelitis represents a diagnostic challenge because its clinical and radiological manifestations are nonspecific, while conventional microbiological investigations have limited sensitivity and prolonged turnaround times [2,3,4]. Its rarity contributes to a low index of clinical suspicion, often resulting in delayed etiological confirmation after repeated treatment for presumed odontogenic infections. Persistent symptoms, recurrent swelling, or failure to respond to antibiotics and drainage should therefore prompt reconsideration of the initial diagnosis and escalation to tissue sampling and molecular investigations [4,22]. The central diagnostic difficulty is not simply recognizing osteomyelitis but identifying its underlying cause when conventional treatment fails. Imaging findings, including bone destruction, rarefaction, and cortical involvement, cannot reliably distinguish tuberculosis from pyogenic osteomyelitis or malignant lesions. Radiological examinations should therefore guide lesion assessment and biopsy planning rather than establish the definitive diagnosis [10]. This limitation is particularly relevant to temporomandibular joint (TMJ) involvement, which may resemble arthritis, pyogenic infection, or malignancy and lead to diagnostic investigations outside the maxillofacial tuberculosis pathway [2,3]. The differential diagnosis includes pyogenic osteomyelitis, oral squamous cell carcinoma, and salivary gland disorders [2,4,10]. Less common conditions, including Langerhans cell

histiocytosis and chronic recurrent multifocal osteomyelitis, should also be considered when lesions demonstrate persistent inflammation, resistance to treatment, or tumor-like radiological characteristics [2,9,16–19,25,29]. Accurate differentiation requires integration of clinical findings, imaging, histopathology, and laboratory investigations rather than reliance on a single diagnostic feature.

Several interacting factors contribute to diagnostic delay and disease progression. Clinical and radiological nonspecificity may lead to provisional diagnoses of dentoalveolar abscess or pyogenic osteomyelitis, resulting in prolonged antibiotic treatment without addressing the underlying infection [2,4,9,30–34]. Negative acid-fast bacilli (AFB) smears, cultures, and chest radiographs may provide false reassurance, particularly in patients with localized or paucibacillary disease [2,8]. Patients may also conceal or fail to report a previous history of tuberculosis, further complicating clinical assessment [3,29]. Delayed treatment can permit progressive bone destruction and extension into adjacent craniofacial structures [29].

Histopathological examination remains central to diagnosis but should be interpreted alongside microbiological and molecular findings. The absence of caseating granulomas does not exclude tuberculosis, as specimens may demonstrate non-caseating granulomas or nonspecific chronic inflammation. When clinical suspicion persists despite inconclusive histology, repeat lesion-directed sampling and adjunct molecular testing should be considered [8,11]. A practical diagnostic approach should prioritize early tissue sampling and simultaneous histopathological, microbiological, and molecular evaluation [3,9,11]. The proposed algorithm involves six steps: (1) suspect tuberculosis in chronic or treatment-resistant jaw lesions [2,4]; (2) use CT or CBCT to assess the extent of bone destruction [2,28]; (3) obtain an incisional biopsy or site-specific aspiration [3,28]; (4) request histopathology, AFB staining, and mycobacterial culture simultaneously [28]; (5) perform Xpert MTB/RIF (CB-NAAT) or PCR for rapid molecular confirmation [11,27,28]; and (6) assess patients for concurrent pulmonary involvement [28]. This proposed sequence is derived from the available evidence and clinical reasoning. It has not yet undergone prospective validation and should be regarded as a practical diagnostic framework rather than a validated clinical guideline.

Therapeutic Perspectives and Considerations

Once maxillofacial tubercular osteomyelitis is confirmed, prompt initiation of antitubercular therapy (ATT) is essential and is generally associated with substantial clinical improvement [3,29] (Supplementary Table 3). Several reported cases have demonstrated marked improvement within four weeks of treatment initiation [8]. ATT remains the cornerstone of management, while surgical intervention serves as an important adjunct for obtaining diagnostic tissue and controlling localized disease [30]. Accurate diagnosis also has important therapeutic implications because it can prevent unnecessary procedures, including superficial parotidectomy performed for lesions incorrectly suspected to represent salivary gland neoplasms [4,12]. Treatment planning should consider disease extent, anatomical involvement, microbiological findings, and potential drug resistance. Clinicians should remain alert to multidrug-resistant tuberculosis (MDR-TB) and extensively drug-resistant tuberculosis (XDR-TB), particularly in patients with previous treatment exposure or inadequate therapeutic response [35,36]. Although rapid molecular assays can identify tuberculosis and detect rifampicin resistance, mycobacterial culture and drug susceptibility testing remain necessary for comprehensive resistance assessment and treatment selection [35,36]. Adjunctive corticosteroids are recommended in selected complications of tuberculosis, particularly meningitis and pericarditis, rather than as routine treatment for uncomplicated maxillofacial disease [35,36].

Despite its diagnostic value, implementing Xpert MTB/RIF may be challenging in resource-limited settings. The costs of instruments, cartridges, maintenance, and laboratory infrastructure can restrict accessibility, particularly in low- and middle-income countries. Reliable cartridge supplies, trained personnel, and equipment maintenance are also necessary for sustainable implementation. Although economic evaluations have generally supported its cost-effectiveness compared with conventional diagnostic pathways, initial investment and operational constraints remain important barriers [28,37,38]. Furthermore, Xpert MTB/RIF may produce false-negative results in paucibacillary disease, including extrapulmonary and HIV-associated tuberculosis, or when specimens are inadequate or poorly collected. Consequently, a negative molecular result should not exclude tuberculosis when clinical, radiological, or histopathological findings remain suspicious. Repeat sampling, culture, and further clinical evaluation may be required [37,38]. Surgical intervention has both diagnostic and therapeutic indications. Diagnostic surgery primarily involves lesion-directed tissue acquisition, particularly when initial smears or aspirates are negative despite persistent clinical suspicion. Incisional biopsy provides material for histopathological examination, mycobacterial culture, and molecular testing, while helping distinguish tuberculosis from malignancy and other granulomatous disorders. Therapeutically, debridement may be indicated as an adjunct to ATT in patients with extensive osteolytic destruction, persistent abscesses, necrotic tissue, or progressive

involvement of adjacent anatomical structures. Early intervention may help control local disease and limit further damage to vital craniofacial structures, including the orbit. Nevertheless, surgery should complement rather than replace ATT, with its extent determined by clinical findings and disease progression.

Conclusion:

Maxillofacial tubercular osteomyelitis remains a diagnostic challenge because its clinical and radiological manifestations frequently resemble odontogenic infections, pyogenic osteomyelitis, and malignant lesions. No single investigation provides sufficient diagnostic certainty in all cases. Histopathology, mycobacterial culture, and molecular assays should therefore be used together when tuberculosis is suspected. Dental clinicians and radiologists have complementary roles in recognizing atypical lesions, assessing bone destruction, and identifying patients who require tissue sampling. Early initiation of antitubercular therapy is essential, while surgery should be reserved for diagnostic sampling and selected therapeutic indications. Greater clinical awareness and access to rapid molecular testing may reduce diagnostic delays, prevent unnecessary surgical procedures, and limit progressive craniofacial destruction. Prospective studies are needed to establish standardized diagnostic pathways and evaluate their effectiveness in clinical practice.

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