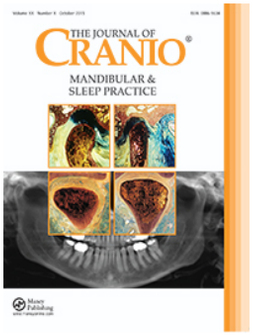

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CLINICAL PRACTICE

Infectious arthritis and the temporomandibular joint. A review

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ABSTRACT

Objective: Microorganisms can cause acute infectious arthritis, chronic infectious arthritis, or reactive inflammatory arthritis. The aim of this study is to perform a narrative review of the pathophysiology, etiology, and diagnostic features of infectious arthritis and TMJ infectious arthritis.

Methods: A search of the literature was performed using Medline, Scielo, Embase, and Google Scholar databases. The terms employed for the search were "Temporomandibular Joint Disorders" and "Infectious Arthritis"; or "Septic Arthritis"; or "Bacterial, Fungal, or Viral Arthritis." Over three hundred articles were screened for eligibility.

Results: The selected articles were utilized to perform a narrative review of the general aspects of infectious arthritis and infectious arthritis affecting the TMJ.

Conclusion: Infectious arthritis is a rare, yet very morbid, form of arthritis. Understanding general aspects of joint infections and specific features of TMJ infectious arthritis is imperative for an adequate diagnosis.

KEYWORDS

Temporomandibular disorders; temporomandibular joint; infectious arthritis; TMJ infectious arthritis; septic arthritis

Introduction

Arthritis is a term used to indicate inflammation of one or more joints, often accompanied by pain, swelling, stiffness, deformity, and limitation in range of motion [1]. Joint diseases are characterized by causing acute or progressive loss of functionality and the development of painful manifestations [2].

Based on biologic behavior, arthritic diseases affecting the temporomandibular joint (TMJ) can be separated into two different subsets: low-inflammatory and high-inflammatory arthritis [3].

TMJ osteoarthritis and post-traumatic arthritis are considered low-grade inflammatory arthritis. On the other hand, arthritic conditions, such as rheumatologic disorders, infectious arthritis, and spondyloarthropathies, are arthritic disorders mediated by high inflammatory responses [3].

The role of microorganisms in the pathophysiology of arthritis is complex and has been described since the times of Hippocrates [4].

Infectious arthritis (IA) is a generic term commonly used to describe any arthritic process caused by bacteria, fungi, viruses, or parasites, in which the ability of infectious agents in colonizing the joint will be dependent on host-related factors and the characteristics of the microorganisms [5].

In this article, the authors performed a narrative review providing a general overview of the pathophysiology, etiology, and diagnostic features of infectious arthritis to then analyze specific aspects of TMJ arthritic disease caused by microorganisms.

Materials and methods

The authors NPS and PAE-M performed a literature search in August 2017 using MEDLINE, *Scielo*, and Embase databases in 3 languages: English, Spanish, and French, without date restriction. The search strategy was carried out by using combinations of the following MeSH terms: "Temporomandibular Joint Disorders" or "TMJ" with "Septic Arthritis," or "Infectious Arthritis," or "Bacterial Infections," or "Viral Infections"/and "Fungal Infections." All studies matched during the search were considered for eligibility, including meta-analysis, systematic reviews, reviews, cohort studies, case-control studies, cross-sectional, and series of cases/case reports. In May 2019, the search was repeated and

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extended by all of the authors, using MEDLINE and Google Scholar databases. Additionally, a manual search was carried out in journals and textbooks of interest in oral and maxillofacial surgery, oral medicine and pathology, orofacial pain, rheumatology, and infectology. Over 300 articles were retrieved and screened for relevant information. The papers were first analyzed by their title and abstract; upon the authors' agreement, only the relevant papers were considered for a full-text revision and evaluated for inclusion.

Results

The articles included addressed different topics regarding the pathophysiologic mechanisms, etiologic agents, and clinical and demographic characteristics of infectious arthritis and TMJ infectious arthritis. Eighty-seven articles were selected and reviewed to perform the narrative review below, which was organized into two major topics: (1) General characteristics of infectious arthritis; and (2) IA of the TMJ, etiologic agents, diagnosis, and initial management.

Discussion

General characteristics of infectious arthritis

Dissemination mechanisms of infectious agents

Most infection processes on joints are related to the migration of microorganisms from a distant site, mainly through hematogenous spread [6]. Other mechanisms by which microorganisms can invade joints is by a contiguous focus of infection, i.e., sepsis or adjacent tissue infection, or through direct inoculation by a traumatic or iatrogenic event [7].

Pathophysiology of infectious articular destruction

Articular tissue destruction often progresses rapidly (as soon as 8 hrs after infection) [8], by the following pathogenic mechanisms:

a) The rapid proliferation of microorganisms within the synovial fluid and synovial lining, activating phagocytosis of macrophages, synoviocytes, and polymorphonuclear neutrophils. Also, the accumulation of high levels of proinflammatory cytokines and chemotactic molecules lead to vasodilatation and release of catabolic enzymes causing damage and degradation of the articular surfaces [9,10].

b) Binding capability to sialoproteins and glycoproteins of joint tissues and the release of proteases that enhance the cartilage breakdown [11].

c) Antigen-antibody complexes form in response to the polymers present in the cellular walls of microbial agents, e.g., lipopolysaccharide (LPS) of the Neissaria gonorrhea (N. gonorrhea), generating a profuse inflammatory response) [12].

d) The similarity between self-antigens and microbial molecules may also be a mechanism triggering immune T cell response and cytotoxicity [13].

Etiologic agents and general features of infectious arthritis

Infectious arthritis (IA) is a generic term commonly used to describe any arthritic process caused by bacteria, fungi, viruses, or parasites [5]. In the search strategy performed by the authors, no articles were found describing the role of fungal, viral, or parasitic agents in TMJ infectious arthritis. Consequently, the etiologic and general characteristics of these forms of infectious arthritis were not included.

Bacterial arthritis

Bacterial infections can cause acute or chronic articular infections. Septic arthritis (SA) is often used to describe acute bacterial infections affecting the joint. Septic arthritis refers to acute pyogenic bacterial infection of a joint. Acute intra-articular infections are rare and recognized as rheumatological emergencies due to their potential mortality (ranging from 10–20%) and significant morbidity [14,15,16].

Information regarding the epidemiology of SA is scarce due to its very low prevalence. SA often affects large weight-bearing joints, such as the knee or hip, being far less prevalent in smaller joints, such as the temporomandibular joint (TMJ) [17]. In fact, only a few series of cases with more than 50 patients are published in medical and dental literature [18].

Etiologic agents in SA can be divided into two distinct groups: gonococcal and non-gonococcal infections [19,20].

Non-gonococcal SA

The most commonly isolated agents in each group are Neisseria gonorrhoeae (gonococcal infections) and staphylococcus aureus (S. aureus) (non-gonococcal infections) [21].

Staphylococci or streptococci account for 91% of the isolated microorganisms in non-gonococcal SA [22–25], probably due to their adherence mechanisms during bacteremia [26].

Staphylococcus aureus is found in approximately 37–56% of SA cases [24]. Methicillin-resistant S. aureus is isolated in 25% of the SA, more predominantly in elderly patients, intravenous drug abusers, and patients with prosthetic joints [27,28].

Bacteriology of SA in children is similar to that found in adults, as S. aureus is the most common isolated pathogen in pediatric SA [10].

Gonococcal septic arthritis

Gonococcal arthritis occurs in approximately 42–85% of patients with disseminated gonococcal infection (DGI).

Disseminated gonococcal infection is a non-purulent form of polyarthritis that often affect wrists, knees, ankles, and may also compromise smaller joints. Purulent forms of DGI are uncommon, producing mono or polyarticular SA [29]. Two cases of TMJ infectious arthritis produced by DGI have been reported in the literature [30,31].

Chronic bacterial arthritis

Chronic bacterial arthritis is associated with two infectious pathogens: Treponema pallidum (syphilis) and Mycobacterium tuberculosis (M. tuberculosis). Treponema pallidum is known to cause polyarthritis, affecting large and smaller joints in late syphilitic stages [32]. M. tuberculosis is capable of causing monoarticular granulomatous infection, most predominantly in weight-bearing joints, such as the knee or coxofemoral joint. All patients having articular infections caused by tuberculosis or syphilis should be tested for human immunodeficiency virus (HIV), as these diseases are usually comorbid [33,34].

Lyme disease

Lyme borreliosis, or Lyme disease (LD), is caused by bacteria that belong to the family of spirochaetes "Borrelia burgdorferi," transmitted by the bite of an infected tick (lxodes ssp) endemic from North America and Eurasia, often found in small mammals, such as mice and voles. The articular manifestations of Lyme disease, or the clinical features, are often migratory polyarthritis with distinctive grades of severity and rigidity [35].

Reactive arthritis (ReA)

ReA is a painful form of inflammatory arthritis (spondyloarthropathy) reactive to certain bacteria or bacterial antigens. The term ReA describes a painful inflammatory arthritis that occurs in reaction to a distant mucosal infection derived from the gastrointestinal, genitourinary, or respiratory tracts, with a detectable presence of replicant bacteria or bacterial antigens within the synovial lining or fluid [36,37]. The actual prevalence of ReA is not known, and it is mostly dependent on a specific epidemiological outbreak of the causative pathogen. Musculoskeletal signs emerge within 1–2 weeks after the infection but can begin even after 4 weeks from the initial infection [38]. Clinically, ReA is characterized by asymmetric oligoarthritis, enthesitis, tendinitis, conjunctivitis, urethritis, and dysuria [36].

Infectious arthritis and the temporomandibular joint

TMJ septic arthritis

Infectious arthritis affecting the TMJ is an uncommon form of arthritis with an estimated incidence of 2–10 cases per 100,000 [39]. The majority of the literature makes reference to adult and pediatric SA affecting the TMJ [40].

Hematogenous dissemination through the TMJ seems to be the most common migration mechanism [41], followed by the direct spread of the bacteria from adjacent structures, e.g., ear infections, otomastoiditis, odontogenic and pharyngeal infections, parotid gland, or distant maxillofacial fractures, and direct inoculation of bacteria via penetrating trauma or iatrogenic causes [42,43]. Jeon et al. [44] proved that bacteria could spread hematogenously and invade TMJ synovial fluid after maxillofacial surgical procedures. The authors found that S. aureus produced the highest bacteremia rates in the bloodstream (68.7%) and were also the only microorganisms measured in the synovial fluid (over 50% of cases). Kim et al. [45], in a case and control study, found that S. aureus was the only bacteria that were significantly more detected in the disc displacement samples, leading the authors to infer that these microorganisms were likely to invade the TMJ with internal derangement more easily. Also, a correlation was found between the number of bacteria present and several clinical manifestations. For example, mycoplasma genitalium (M. genitalium) was associated with 3.80 times more joint pain, and mycoplasma fermentans/oralis (M. fermentans/oralis) was 2.80 times more frequent in patients with limited opening [45].

Clinical diagnosis

TMJ SA often starts as monoarticular infection (90% of cases), producing erythema, swelling, pain, and loss of range of movement on the affected joint. Typically, SA frequently occurs with moderate fever; in only 30–40% of cases, it begins with temperatures higher than 39°C (102.2°F) [23]. Latency usually takes over two weeks upon manifestation. However, the development of arthritis is relative to the virulence of the microorganisms [46].

Early diagnosis is essential to predict a successful prognosis and therapeutic outcome, mainly because after the consolidation of the septic infection, the destruction of the articular tissues begins almost immediately [47].

The clinical manifestations appear with severe and spontaneous unilateral localized pain, tenderness, increased temperature, redness, and swelling of the TMJ. The pain often increases with mandibular movement and a limited opening, as well as severe ipsilateral malocclusion [43,48,49,50,51]. Acute occlusal changes, such as anterior or ipsilateral open bite, may be due to osteolysis of the joint surfaces or intra-articular effusion and edema [52].

When the aforementioned clinical symptoms have a temporal relation with fever, SA should be immediately suspected. Other signs that may lead to suspicion of SA are poor general conditions, chills, fatigue, weakness, and sweating [22].

Clinically, the diagnosis should include a complete imaging study, laboratory testing, and the aspiration of the synovial fluid for analysis, especially when SA is suspected [43].

Synovial fluid analysis

Synovial fluid extraction for analysis is considered essential to confirm SA diagnosis, allowing the identification of over 87% of involved bacteria in the joint infection. Intra-articular aspiration or lavage of the septic content improves prognosis, decreases the bacterial load, and limits joint damage [53]. Ideally, the sample of the aspirated synovial fluid must be analyzed within 1–6 hours post-extraction to avoid altering its composition or contact with artifacts that may reduce diagnostic efficacy [24,54]. The synovial analysis must include gross appearance documentation and cytological, microbiological, and crystallographic analysis [55,56] (Table 1).

Traditionally, synovial fluid white blood cell count (WBC), over 50,000 per mm3, is considered a useful marker of joint infection, but it may overlap with other forms of arthritides or acute crystalline joint disease. Likewise, gonococcal and granulomatous SA may have WBC count below 50,000 per mm3 [20]. The obtained samples from the affected TMJ must be analyzed for crystal analysis, Gram staining, culture, and tested for sensitivity [57]. Cytological analyses throwing over 75% of polymorphonuclear neutrophils (PMN) and WBC over 50,000 cu/mm are correlated with SA [58]. Bacterial DNA testing, or PCR for specific bacteria should be considered when the empirical antibiotic treatment is ineffective [59] (Figure 1).

Table 1. Characteristics of the synovial fluid [56].

Type of Synovial Fluid	Macroscopic aspect	White Blood Cell Count/ mm3	% PMN
Normal	Transparent, pale-yellowish, low aspirated volume, viscous.	0–200	<10
Non-Inflammatory or Low Inflammatory	Yellowish, slightly turbid, more aspirated volume, less viscous	200–2000	<20
Inflammatory	Yellow, turbid, xanthochromic, more aspirated volume, non-viscous	2000–50000	20–75
Septic	Turbid, purulent, yellow-green pus with hematic content, much more aspirated volume.	>50000	>75

PMN: Polymorphonuclear neutrophils.

Laboratory tests

Leukocytosis, over 11,000 per mm3, has 75% sensitivity and 55% specificity in the diagnosis of SA [60]. Acute inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), are often helpful in determining an ongoing SA [61]. However, patients with confirmed SA may present normal CRP and ESR levels. Cultures of blood samples may be useful in identifying the causative organism when the synovial fluid analysis has failed to do so [55].

Imaging in TMJ septic arthritis

Imaging studies are complementary and can help confirm the diagnosis but should not be used as a sole diagnostic method. Initial studies can include uniplanar X-rays, such as an orthopantomography or a transcranial oblique lateral radiography. These techniques may be useful in detecting changes in the joint space, limitation of condylar movement, or substantial articular damage [41].

Ultrasonography is a valuable diagnostic tool in detecting joint effusion and small amounts of septic collections. It may also serve as a guide for the aspiration of the TMJ. Septic joints exhibit an echo-free-joint effusion (hypoechoic fluid) characteristic of clotted septic/hemorrhagic collections [62,63].

The MRI T2 displays good resolution for early detection of joint effusion and purulent accumulations, allowing evaluation of the state of the bone surfaces with a reasonable resolution and other structures, such as the indemnity of the articular disc and adjacent soft tissues [64,65].

Computed tomography (CT) provides fewer anatomical details and spatial resolution than magnetic resonance imaging (MRI). However, it displays a better diagnostic performance in identifying bony structures and has less interference with metal implants. Cone-beam computed tomography (CBCT) or medical CTs are advantageous in

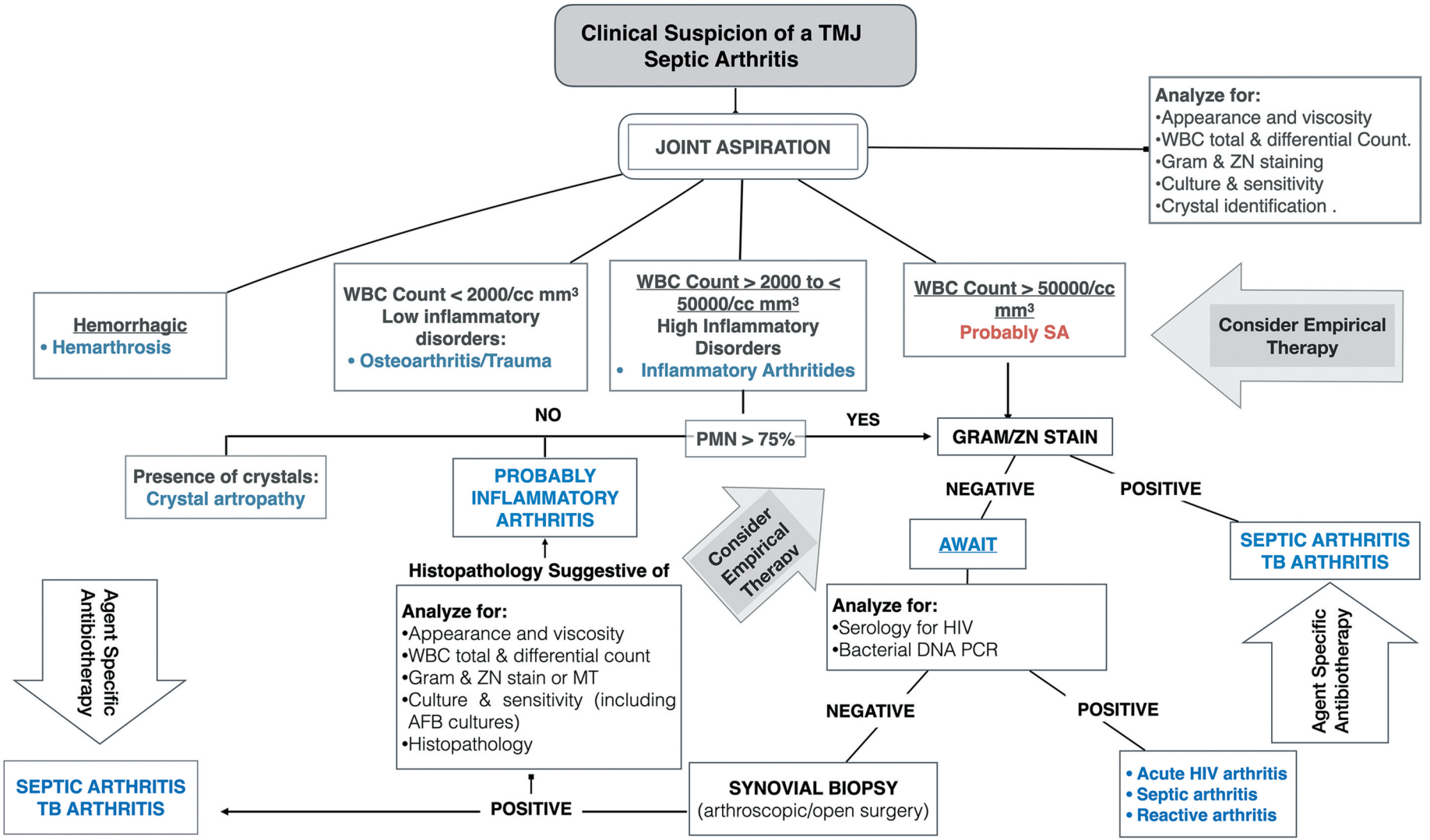

Figure 1. Suggested diagnostic approach to temporomandibular joint septic arthritis (TMJ SA). Figure depicts the diagnostic workup after synovial fluid aspiration. WBC: White Blood Count; ZN stain: Ziehl-Neelsen stain; AFB cultures: Acid-fast bacilli; MT: Mantoux test; cc: cell count; PMN: Polymorphonuclear neutrophils.Figure adapted from Mathew and Ravindran [24].
Note: Septic arthritis should only be diagnosed by a specialist.

diagnosing detrimental bone changes, such as erosion and changes in the cortical and cancellous bones, effectively allowing the visualization of sequelae in the TMJ [66,67].

Treatment

Empirical treatment should begin as soon as possible, using broad-spectrum antimicrobials, mostly preferring third generation cephalosporins [19]. Cai et al. [41], in a retrospective series of cases, reported that empirical therapies, using third generation cephalosporins and metronidazole were adequate in most cases.

Stengel et al. [68], in a systematic review and meta-analysis, reported there were no significant clinical advantages in choosing one antibiotic regimen over another.

After draining the purulent content of the joint, immobilization or maintaining a jaw resting position is recommended to prevent joint pain; once the infectious process remits, controlled jaw exercises may be advisable. The most frequent clinical sequels are intracapsular adhesion, disc perforation, ankylosis, fibrosis, and bone and cartilage destruction [54].

Chronic bacterial arthritis and the TMJ

Chronic bacterial infections in the TMJ have been linked mostly to extrapulmonary tuberculosis. Extrapulmonary tuberculosis represents an enormous clinical challenge when its onset is in the absence of classic pulmonary symptoms [33]. Approximately 50% of HIV-positive patients with tuberculosis manifest the extrapulmonary form of the disease [69].

Current evidence suggests that about 10% of extrapulmonary manifestations can affect craniocervical structures, most commonly affecting cervical lymph nodes, followed by bony facial structures [70].

Prasad et al. [71] estimated that only 1 out of 165 extrapulmonary tuberculosis cases affecting facial and cervical structures could affect the TMJ. Extrapulmonary mycobacterium infection can be disseminated into a joint using a hematogenous pathway through the cancellous bone. These spreading forms of colonization on the joint are also known as osteoarticular tuberculosis [72]. Osteoarticular tuberculosis often has an insidious onset, and tubercular involvement is likely to affect the TMJ through the cancellous portion of the mandibular condyle [73].

Clinical manifestations are similar to the clinical findings in TMJ SA. Swelling, redness, tenderness, local increase in temperature, and limitation of function in a granulomatous joint infection are almost indistinguishable from a TMJ SA [72,73,74,75]. All patients with TMJ SA, who are not responsive to empirical antibiotics, should be tested for M. tuberculosis and HIV. The Mantoux test is helpful to rapidly determine a Mycobacterium tuberculosis infection in patients who do not have tuberculosis. Once the diagnosis is confirmed, anti-tubercular chemotherapy should be started immediately [76].

Lyme disease and the TMJ

The TMJ is the fourth most commonly affected joint in Lyme disease (LD) [77]. Arthritis secondary to Lyme often occurs in the initial stages of the disease and may present itself as an earache, TMJ arthralgia, temporomandibular sounds or crepitation, or masticatory myofascial pain, acoustic symptoms, and limited opening. The histopathological analysis may detect the presence of spirochaetes, fibrinous deposits, vascular obliteration, and polymorphonuclear neutrophils depositions [78]. Early diagnosis and therapy often prevent the persistence of Borrelia within the synovial lining and chronic arthralgia [79].

Arthroscopic studies reported that patients with Lyme disease often present more synovial inflammation and hypertrophy, vascular dysfunction, adhesions, disc pathology, and degenerative changes than controls [78].

Reactive arthritis and the TMJ

Few reports establish the relation between TMJ arthritis and ReA. As mentioned previously, ReA is a form of painful inflammatory arthritis, which is a consequence of a distant bacterial infection from gastrointestinal or genitourinary tracts. Könönen [80] evaluated the muscular and articular dysfunction in patients with ReA; his results demonstrated that patients with ReA had a more severe Helkimo Index than healthy controls, presenting more pain at mandibular motion, palpation, and jaw opening. He also found more muscular symptoms in ReA patients.

In another study, Könönen et al. [81] compared the radiographic findings of orthopantomography in 49 ReA patients versus 49 healthy controls, reporting radiographic findings in 33% of ReA patients versus 10% in the control group.

Henry et al. [82] showed the presence of Chlamydia trachomatis (C. trachomatis) and M. genitalium by immunostaining and PCR screening of retrodiscal tissue extracted from disc repositioning surgery. Another study of internal derangement patients who underwent TMJ surgery found that TMJ retrodiscal tissues that tested positive for C. trachomatis were significantly associated with the expression TNF–α and IL-6 [83].

Several studies have discussed the possible causative role of C. trachomatis in TMJ pathology. While some studies have confirmed the presence of C. trachomatis in patients with arthralgia and TMJ internal derangement, other studies have failed to identify these microorganisms

[84,85,86,87]. The assumption of such an association requires further clarification.

Conclusion

Infectious arthritis is a rare and highly morbid form of arthritis. Several microorganisms have been associated with different forms of TMJ arthritic manifestations. However, the potential role of infectious agents in the development of TMJ pathology has not been thoroughly clarified in the dental literature.

Understanding general aspects of joint infections and how they correlate to TMJ arthritis seems imperative to correctly identify, diagnose, and manage these conditions. Therefore, in the context of a red, swollen, and painful TMJ, dental practitioners who specialize in TMJ pathology must be able to discern plausible etiologic factors and its possible course of progression. Moreover, a prompt referral to an emergency or rheumatology service can be quintessential to reduce morbidity and ensure a positive outcome. Synovial fluid aspiration and characterization seems to be essential in defining the diagnosis and therapeutic strategy and improving the prognosis.

Although the literature on incidence and prevalence is scarce, it appears that the most prevalent subtypes of IA affecting the TMJ are TMJ SA and TMJ chronic bacterial infection linked to extrapulmonary tuberculosis. The role that microorganisms, such as C. trachomatis, play concerning reactive inflammatory responses within the TMJ structures needs further research. Currently, the etiologic role of C. trachomatis cannot be discarded as a plausible cause of TMJ reactive arthritis.

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Competing interest

None of the authors have a conflict of interest to declare.

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