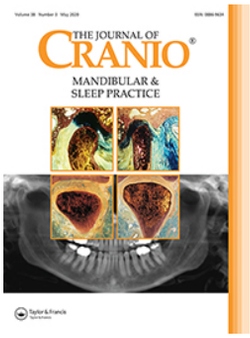

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CASE REPORT

Destructive psoriatic arthritis of the temporomandibular joint: a clinical case, an overview of the pathophysiology and its differential diagnoses

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ABSTRACT

Background: Clinical assessments and uniplanar images in temporomandibular disorders are not always entirely reliable. This predicament is especially important when clinicians need to determine the nature of temporomandibular joint disease, particularly when clinical features are not helpful in determining the diagnosis.

Clinical presentation: A 63-year-old female patient presented with mild pain in her right TMJ. During routine imaging exams, a destructive monoarticular arthritis was noticed, producing multiple erosions of the mandibular and temporal condyles. In addition, attrition of the ceiling of the glenoid fossa was observed, generating a communication with the endocranium. Only the presumptive biological behavior revealed on TMJ imaging and the appearance of the psoriatic plaques later during follow-up helped the authors to narrow the differential diagnosis.

Conclusion: The clinical case presented illustrates the difficulties in diagnosing an erosive, seronegative TMJ destruction, suggestive of a systemic arthritis.

KEYWORDS
TMJ; temporomandibular joint diseases; psoriatic arthritis; TMJ arthritis; erosive osteoarthritis

Introduction

Temporomandibular disorders (TMDs) are a group of musculoskeletal conditions that produce dysfunction and painful sensations in the orofacial masticatory apparatus. The prevalence varies from 10–15% in adults to 4–7% in adolescents, exhibiting a gender ratio of 2:1 (female/male) [1]. Findings in the Orofacial Pain Prospective Evaluation and Risk Assessment Cohort Study reported an annual incidence of painful TMD diagnosis of 3.9% annually, and almost 49% of the incident cases persisted with painful TMDs in the six months longitudinal follow-up [2]. Furthermore, Manfredini et al. [3], in a systematic review, accounted for a total of 3463 subjects and observed an overall prevalence of 30.1% for arthralgic- and joint disease-related TMDs.

Recent data recognized that arthralgic and myalgic TMDs are a part of a complex multifactorial model, disregarding old mechanistic beliefs and occlusion-based explanatory models [4]. Moreover, with the advent of systematic and taxonomic research in the field, advancements in imaging techniques, and better understanding of the pathophysiology, rheumatology, and immunology of articular disease enable elucidation of essential aspects of the biology of joint destructive processes.

The recently adopted criteria for TMDs, the Diagnostic Research Criteria for Temporomandibular Disorders (DC/TMD) gathers under "Joint Disease" a variety of diagnostic categories, including Degenerative Joint Disease, Systemic Arthritides, Idiopathic Condylitis, and other less frequent presentations [5]. The vast number of diagnoses under the umbrella of joint disease involving different etiologies makes it difficult to comprehend every category individually. Therefore, it may be more practical to address joint diseases in terms of the biological behavior of arthritic disorders affecting the TMJ. Mercuri et al. [6] proposed to divide TMJ arthritic disorders into two distinct subgroups: low-inflammatory and high-inflammatory joint

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diseases, based on the intensity of the biological response. Degenerative joint disease, i.e., osteoarthritis/osteoarthrosis, and post-traumatic arthritis are considered arthritic conditions with low-grade inflammatory responses. Conversely, systemic arthritides, such as rheumatologic disorders, mixed connective tissues disorders, spondyloarthropathies, crystal-induced arthropathies or infectious arthritis, are mediated by high inflammatory responses [6].

The distinctive biological behavioral differences between low and high inflammatory arthritis are also represented in imaging findings. For example, osteoarthritis often presents prototypic findings, especially in late osteoarthritis, in which there is coexistence of progressive destruction and adaptive-reparative changes, well-described by Ahmad et al. [7]. By contrast, imaging findings in high inflammatory arthritis often correspond to more aggressive erosive articular destruction with seropositive laboratory blood tests or with a diagnosed rheumatologic disease. Nevertheless, there are clinical cases in which symptoms and signs may indicate low inflammatory arthritic conditions, but imaging findings suggest a more aggressive destructive arthritis, consistent with a high inflammatory disease. The authors will present a clinical case of a monoarticular, highly destructive arthritis affecting the TMJ, in which the uncharacteristic clinical features and the indecisiveness of the serological testing made it challenging to establish a definitive diagnosis.

Case report

A 63-year-old, edentulous female patient was referred to an orofacial pain private center in Santiago, Chile, presenting with mild pain in her right TMJ. An experienced Orofacial Pain Fellow (NSP) performed a full orofacial pain history and a complete clinical examination following DC/TMD criteria. At the time of examination, the patient presented with mild controlled hypertension, hypercholesterolemia, and osteopenia, with no history of rheumatological diseases. At the time of examination, she was taking Captopril, Atorvastatin, and calcium/vitamin D supplements.

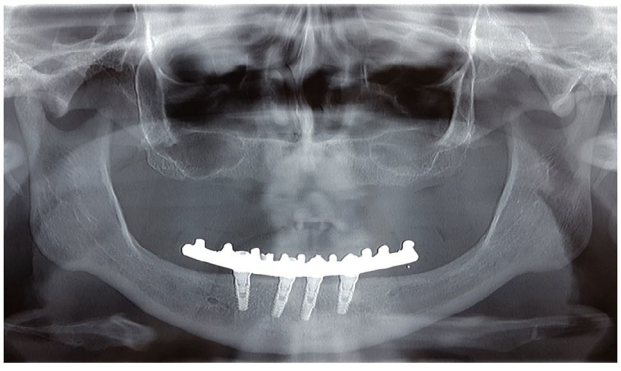

Figure 1. Orthopantomography showing the irregularities of the superficial surface of the right condyle and the right glenoid fossa.

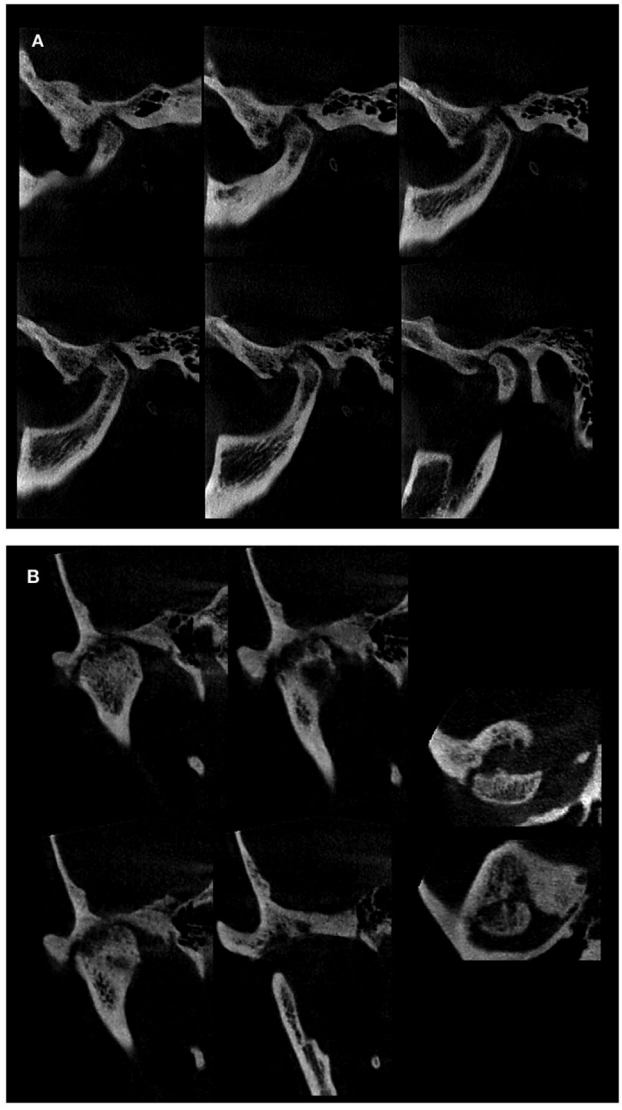

Figure 2. (A) Sagittal CBCT slides. (B) Coronal and axial CBCT slides. In the series of cone beam computed tomography (CBCT) images, deformation of the condylar head was observed due to massive erosion compromising the superior and lateral surface of the right temporomandibular joint (TMJ) and the posterior surface of the temporal condyle. The presence of subchondral bone sclerosis and focused bone formation near the erosion was observed. The glenoid fossa presented a punched-out pattern of erosive destruction, also affecting the most superior articular fossa, producing cortical attrition and communication with the endocranium.

On DC/TMD axis II assessment, the pain drawing showed six areas of bodily pain located in joints of the right hip, left knee, both ankles, metacarpophalangeal and distal interphalangeal joints.

The patient was diagnosed with local myalgia in the right superficial masseter, osteoarthritis in the right

TMJ, and osteoarthrosis in the left TMJ. Considering the clinical characteristics and the low importance that the patient attributed to her symptoms, the primary diagnostic hypothesis was polyarticular osteoarthritis, for which there was ordered an orthopantomography X-ray (OPG) and cone beam computerized tomography (CBCT).

Orthopantomography revealed cortical irregularities on the surface of the right condyle and the right glenoid fossa (Figure 1). On CBCT, massive erosion compromising the superior surface on the right TMJ and posterior surface temporal condyle was observed. The left TMJ showed findings consistent with degenerative joint disease (Figures 2 and 3). Owing to findings of the OPG and the CBCT, Magnetic Resonance Imaging (MRI) was performed, revealing images consistent with a systemic arthritis in the right TMJ and degenerative changes in the left TMJ, as detailed in Figures 4 and 5.

A full set of laboratory tests was solicited, including exams for bone metabolism and rheumatic diseases. The patient was referred to a rheumatologist and endocrinologist to rule out systemic arthritides, bone metastatic tumor or brown tumor, with no conclusive results being reached. Due to the dissonance between imaging studies and clinical symptoms, treatment started with conservative management based on topical and systemic medication. More invasive options were proposed to obtain a histopathological analysis, but the patient did not consent to treatment.

The treatment was effective in reducing symptomatology, and the patient remained asymptomatic during follow-up. At the six-month control, the patient presented for follow-up with an abrupt onset of red inflamed skin patches partially covered by silvery-white scaly skin, producing itching sensations in her elbows, ankles, and scalp (Figure 6). Considering the appearance of skin patches, psoriasis was suspected, leading the authors to consider psoriatic arthritis of the TMJ, for which the CASPAR criteria (Classification Criteria for Psoriatic Arthritis) was applied [8]. A dermatologist and rheumatologist later confirmed the diagnosis.

Discussion

The TMJ is a synovial joint, and consequently, it is not excluded from biological phenomena affecting other synovial joints of the locomotor system. This property means that the biological response of the TMJ should be similar to that of other joints in high or low inflammatory-mediated conditions. Likewise, when clinical features suggest a degenerative joint disease, but the imaging findings show aggressive articular destruction, the clinician should suspect that particular biological behavior and aggressiveness do not correspond to the gradual progression observed in TMJ osteoarthritis [9].

Osteoarthritis is considered a "whole joint disease," determined by a series of molecular processes that converge into a characteristic clinical phenotype [10]. Systemic and local factors, such as aging, low estrogen levels, genetic susceptibility, mechanical overload, joint instability, external trauma, and misalignment of the joint can act as precipitating or predisposing factors in disrupting the balance between catabolism and anabolism in joint tissues [11]. The current understanding of osteoarthritis

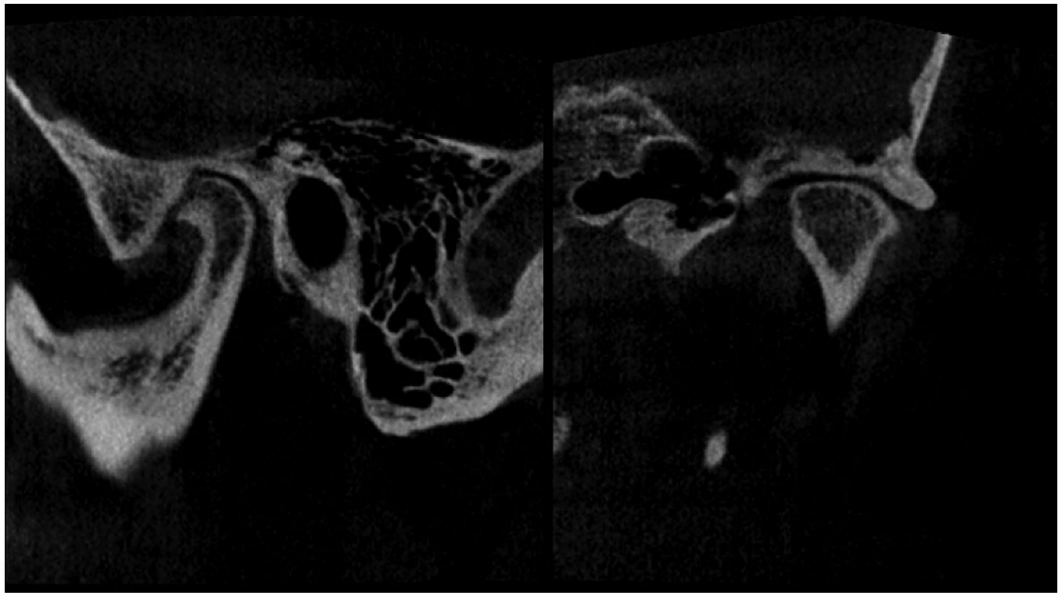

Figure 3. Left temporomandibular joint (TMJ) showed a more typical degenerative joint disease, with diminished joint space, the presence of an osteophyte, cortical attrition, subchondral cysts, and subchondral sclerosis.

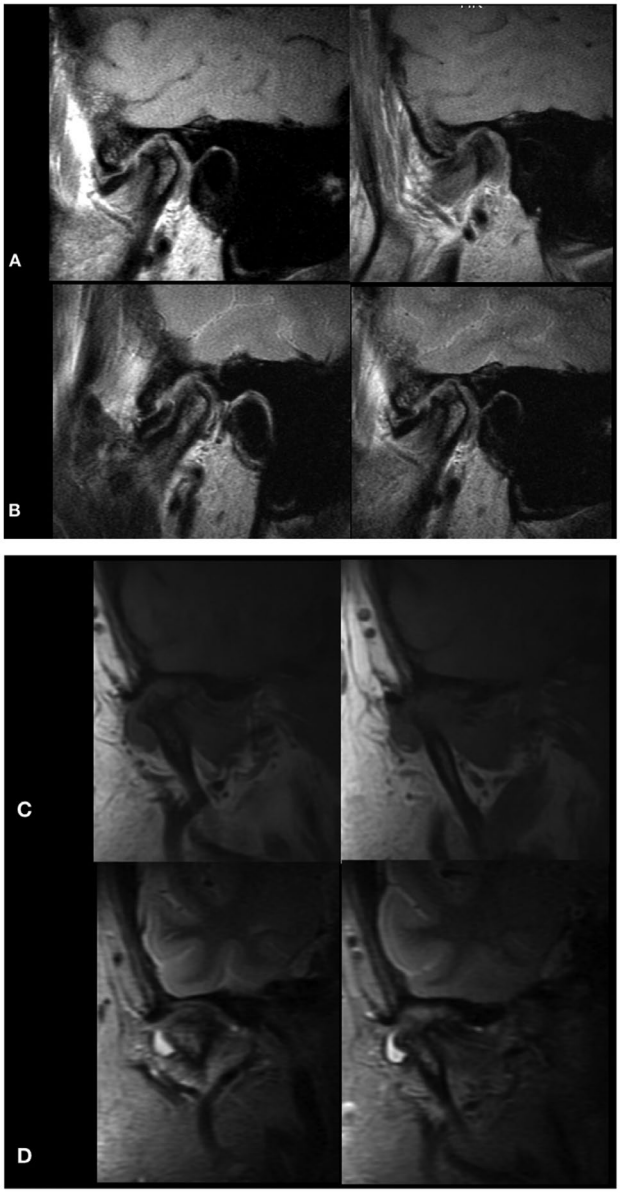

Figure 4. (A) On Magnetic Resonance Imaging (MRI), protonic density acquisition showed that both discs were anteriorly displaced and were not reduced during the opening, most noticeable in the right temporomandibular joint (TMJ), which presented a more displaced and deformed disc. (B) Enhanced synovial membrane signal/pannus in the right TMJ in pre- and post-contrast T1 acquisition was found, being more marked in the erosion areas of the temporal and mandibular condyle. (C) In addition, the mandibular condyle surface presented mixed zones of erosion and sclerosis, and the posterior surface presented mostly erosive destruction with bone marrow edema. (D) The coronal MRI images showed lateral displacement of the articular disc and effusion in the supradiscal and infradiscal articular space.

is built on the role of innate immunity and inflammation in cartilage and subchondral bone deterioration. The lesioned joint releases extracellular matrix debris, plasma proteins, and apatite crystals into the synovial fluid. This phenomenon induces innate immune activation and the sensitization of extra-immune cells (synovial fibroblasts, synovial macrophages, and chondrocytes) and immune cells (macrophages, dendritic cells, and monocytes) [12, 13]. The sensitized cells amplify an inflammatory response through the expression of pro-inflammatory cytokines, chemokines, complement effectors, and catabolic enzymes, promoting a pro-catabolic state. Whereas, many of the expressed molecules are proinflammatory mediators, certain synovial cells will express anti-inflammatory mediators, bone morphogenic protein (BMP), and Transforming Growth Factor (TGF) with the intent to enhance anabolism of the lesioned cartilage [14].

By contrast, the destructive mechanisms in high inflammatory arthritis involve the sensitization and proliferation of fibroblast-like synoviocytes through recognition of auto-antigens and sequential activation of systemic autoimmunity, initiating an intense inflammatory against the articular tissues [15]. This unequal biological behavior between low and high inflammatory arthritis is also represented in the imaging findings. For example, osteoarthritis often presents largely prototypic findings, exhibiting, especially in late osteoarthritis, a coexistence of progressive destruction and adaptive-reparative changes, well-described by Ahmad et al. [7]. In contrast, high inflammatory arthritis often initiates with flares of acute inflammatory symptoms, showing imaging signs of aggressive articular destruction and positive serology [16]. There are clinical variants that may present as chronical progressive joint disease with mild to moderate symptomatology and seronegative blood tests, however, with imaging findings more consistent with high inflammatory arthritis. These less-common clinical presentations are well-established in other joints and are often related to cases of erosive osteoarthritis or some subsets of seronegative rheumatoid arthritis, juvenile idiopathic arthritis, psoriatic arthritis, or spondyloarthropathies. In the clinical case presented, the imaging findings and dissonant symptomatology raised the suspicion of high inflammatory joint disease.

Erosive osteoarthritis (EOA) is a rare and aggressive form of osteoarthritis, sharing degenerative joint disease and inflammatory arthritis features. It affects the interphalangeal joints, starting in 40- to 50-year-old female patients around menopause. Clinically, EOA progresses as episodes of painful and deforming arthritis, having acute exacerbations of swelling, allodynia, and even paresthesia [17]. Histologically, EOA is characterized by presenting foci of proliferative synovitis related to articular surface erosions that are almost indistinguishable from the synovial proliferation of rheumatoid arthritis [18]. However, it also presents some histologic characteristics typically found in osteoarthritis, including subchondral bone condensation and sclerosis, subintimal layer fibrosis, and the

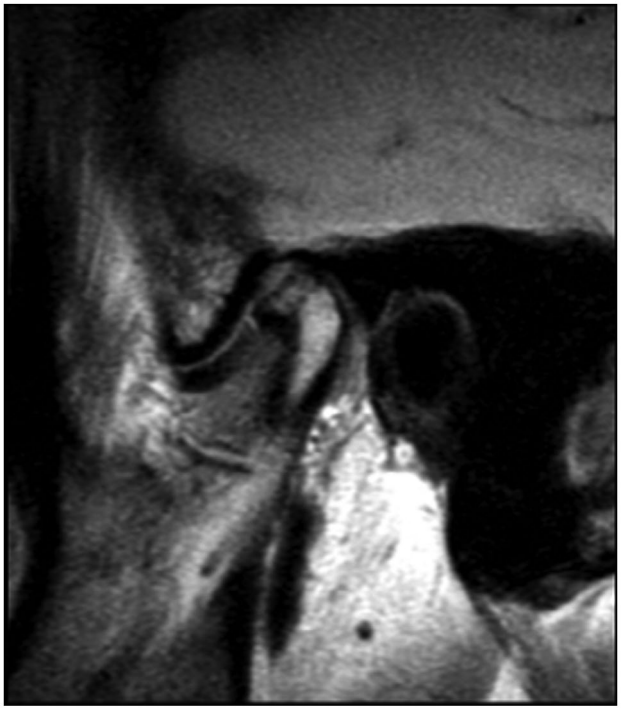

Figure 5. Left temporomandibular joint (TMJ) magnetic resonance imaging (MRI) presented imaging findings more consistent with osteoarthritis.

formation of marginal osteophytes. EOA can be associated with several of the following conditions: Endocrine conditions (hypothyroidism and hyperthyroidism); microcrystal-induced (calcium pyrophosphate dehydrate, apatite deposition diseases, recurrent calcic periarthritis); autoimmune (scleroderma, Sjögren's syndrome, chronic autoimmune thyroiditis); and chronic renal disease (azotemic, amyloid or hemodialysis-associated arthropathy) [19]. The fact that EOA on the TMJ has not been described in the literature is probably due to unawareness of the condition or mislabeling with diagnoses such as idiopathic condylar resorption. In this clinical case, certain characteristics presented by the patient did not fit with EOA, primarily because the patient did not suffer from acute inflammatory exacerbations or episodes of severe articular pain [18].

Spondyloarthropathies may produce erosive articular destruction in the TMJ with seronegative laboratory findings that may produce similar clinical symptomatology and a destructive articular pattern [9]. Nonetheless, HLA-B27 antigen testing was negative and was ruled out by the rheumatologist.

It was not until the patient exhibited psoriatic plaques in her elbow and scalp during the follow-up period that the authors could arrive at a working diagnostic hypothesis.

Psoriatic arthritis is prevalent in patients with psoriasis, having an estimated prevalence of 5–30% [20]. The pathophysiological mechanisms are not well understood, but evidence suggests that psoriatic arthritis may share similar histologic, immunologic and pathogenic features with psoriatic skin lesions [8]. Histologically, the synovial response is less hyperplasic than that of rheumatoid arthritis, but with marked hypervascularization and abundant lymphocytic, neutrophilic, and activated-macrophages cellular infiltration [21].

Microscopic findings in psoriatic arthritis show pronounced clonal expansion of TCD4 in the synovial lining, an increase of TCD8 lymphocytes in the synovial fluid, producing a relevant Th1 response and a less pronounced marked Th17 response [22]. The chronic inflammatory response produces erosive destruction of the fibrocartilage and articular surfaces. In contrast to rheumatoid

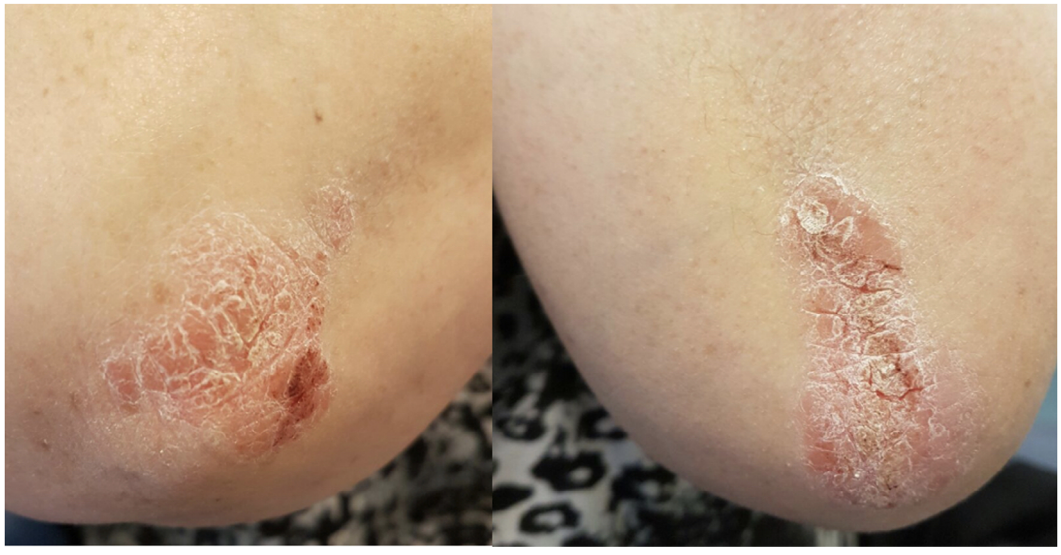

Figure 6. Psoriatic plaques in the elbows, first presented during follow up.

arthritis, psoriatic arthritis shows irregular destructive patterns due to enhanced reactivity of the subchondral response, generating reactive osteoclastogenesis and bone formation [23].

Imaging findings from CBCT or MRI are not precise enough to make a differential diagnosis among different types of highly destructive arthritides. Erosive lesions with multiple foci of bone proliferation may help to direct diagnosis toward psoriatic arthritis, ankylosing spondylitis, or other spondyloarthropathies. By contrast, rheumatoid arthritis patients may present with articular erosions with a more punched-out appearance [9].

MRI images can be useful to determine osseous changes, disc derangement, joint effusion, abnormal bone marrow signals, synovial proliferation, and parotid lymph node swelling [24]. Despite the fact that many of these findings are not disease-specific synovial proliferations, synovial hypervascularization enhanced with gadolinium, augmentation in bone marrow signal and lymphatic swelling are more characteristic of high inflammatory arthritis [25].

In this case, the only clinical sign that helped guide the diagnosis, other than imaging, was the appearance of the psoriatic plaques during continuous follow-up.

Conclusion

In the clinical case presented by the authors, clinical symptoms were not relevant to determine the diagnosis; imaging findings suggested a destructive process that is more compatible with high inflammatory arthritis, and laboratory testing was inconclusive. Only the onset of the psoriatic skin lesions oriented the clinicians to a definitive diagnosis. Regardless of inconclusive clinical symptoms or negative serology, images revealing erosive destructive arthritis of the TMJ should make the clinician suspect the presence of high inflammatory arthritis. Rigorous follow-up may be imperative to narrow the diagnosis, primarily because serology may become manifest, and new clinical symptoms may appear that contribute to the diagnosis.

Disclosure statement

No potential conflict of interest was reported by the authors.

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