Temporomandibular Joint Dysfunction as the First Articular Sign in Psoriatic Arthritis: Case Report

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Abstract

Psoriatic arthritis (PA) is a chronic systemic disease that is difficult to recognize. The diagnosis is made primarily on clinical detection of psoriasis and inflammatory arthritis of the joints. In literature, many Authors have described the harmful effects of PA on the temporomandibular joint (TMJ), but no study has clearly indicated the TMJ as the first articulation to be involved in PA. This article presents a clinical case of PA that was diagnosed many years after a TMJ involvement, because no other signs and symptoms of disease apart from psoriasis were present. The missed early detection resulted in severe TMJ disorders. The TMJ can be the first affected joint in PA. For a right and early diagnosis of PA, an interdisciplinary approach between different dental and medical disciplines is very important.

Key words : Psoriatic arthritis, temporomandibular joint, erosion, sclerosis, MRI

I. Introduction

Psoriatic arthritis (PA) is a chronic disease characterized by an inflammatory arthritis associated with psoriasis. Alibert first described the association of psoriasis and arthritis in the early 19th century.[1] Although most patients with PA have a benign course, a subgroup of patients experiences a severe, mutilating form of arthritis. In some patients, the arthritic symptoms affect the small distal interphalangeal joints. In others, the symptoms affect the joints on one side of the body, but not on the other, while in yet another subgroup, the larger joints on both sides of the body are affected simultaneously, as in rheumatoid arthritis (RA). [2] Some patients with PA experience arthritis symptoms in the back and spine; in rare cases, the disease destroys the joints of the fingers and toes, causing bone fusion and leaving patients with gnarled, club-like hands and feet. [3-6]

The estimated prevalence of psoriasis is 1–2% of the general population, and is higher in North American and northern European whites. PA occurs in 5 to 10% of the psoriatic population, so its general prevalence is about 0.1%. Men and women are affected equally, although men tend to have more severe joint symptoms. [7,8] A genetic predisposition to PA has been demonstrated in family and human leukocyte antigen (HLA) studies. However, the association with the HLA system is not as strong as it is in ankylosing spondylitis (AS). The HLA-B27 gene marker is found in approximately 50% of patients with PA who have arthritis of the spine. Several other genes are also more common in patients with PA. Researchers believe genes that increase one's susceptibility to develop psoriasis are located on chromosomes 6 and 17.[9-11] Like psoriasis and other forms of arthritis, PA also appears to be an autoimmune disorder, triggered by an attack of the body's own immune system on itself.

Psoriasis often begins in the second and third decades, and the onset of PA is usually between the ages of 30 and 50 years. However, the mutilating form of PA may start before the age of 20 years.

In nearly 80% of patients, the symptoms of psoriasis precede the arthritis, while joint disease precedes the psoriasis in up to 15% of patients. If the arthritis precedes the skin disease by many years, the diagnosis of PA can be difficult. In fact, some patients have had arthritis for over 20 years before the psoriasis appeared. Conversely, patients can have psoriasis for over 20 years before developing joint disease, leading to the ultimate diagnosis of PA.[12,13]

The cause of PA remains unknown, although a combination of genetic, immune, and environmental factors is likely involved. Microbiologic agents are suspected of triggering the arthritis in genetically predisposed individuals. Synovitis is the basic pathologic lesion in the peripheral arthritis and is generally indistinguishable from that of RA.[14]

Many Authors have described in literature several classification criteria for PA. For clinical studies, the CASPAR

criteria recognized PA with at least 3 or more of the following features: current or history of psoriasis, dactylitis, juxta-articular new bone formation, rheumatoid factor negativity and nail dystrophy.[15,16]

Five different clinical PA groups have been recognized: symmetric polyarthritis (RA-like), asymmetric oligoarthritis, arthritis of the distal interphalangeal joints, spondylitis, and destructive (mutilans) arthritis.[17-19] The symmetric arthritis is much like RA, but is generally milder with less deformity. It usually affects multiple symmetric pairs of joints (i.e., it occurs in the same joints bilaterally) and can be disabling.

Asymmetric oligoarthritis can involve a few or many joints and does not occur in the same joints bilaterally. It can affect any joint, such as the knee, hip, ankle, or wrist. The hands and feet may develop enlarged, swollen digits that look like "sausages." The joints may also be warm, tender, and red. Individuals may experience periodic joint pain, which is usually responsive to medical therapy. This form is generally mild, although some people can develop disabling disease.

Distal interphalangeal predominant (DIP) arthritis, although the "classic" type, occurs in only about 5% of those with PA. Primarily, it involves the distal joints of the fingers and toes (the joint closest to the nail) and is sometimes confused with osteoarthritis, although nail changes are usually prominent.[6,20]

Spondylitis is inflammation of the spinal column and is the predominant symptom in about 5% of individuals with PA. Inflammation with stiffness of the neck, lower back, sacroiliac joint, or spinal vertebrae is common in a large number of patients, making motion painful and difficult. Peripheral disease can be present in the hands, arms, hips, legs, and feet.

Arthritis mutilans is a severe, deforming, destructive arthritis that affects fewer than 5% of those with PA. It principally affects the small joints of the hands and feet, although it is frequently associated with neck or lower back pain.

Moreover, the patterns of PA are not permanent; more than 60% of patients experience a change from their initial pattern, resulting in a heterogenous combination of joint diseases.[6]

Radiographically, PA is characterized by bony ankylosis, destruction of the joints with narrowing of the joint spaces, bony proliferation, and erosion. Erosion in combination with tapering of the proximal phalanx and bony proliferation of the distal phalanx results in the typical "pencil-in-cup" deformity.[20] In addition to the psoriatic skin changes, several extra-articular features may be observed in patients with PA.

Inflammation of the tendons behind the heel (Achilles tendinitis) can lead to pain on walking or climbing stairs; inflammation of the chest wall and costal cartilage can cause chest pain, as seen in costochondritis; inflammation can affect the eyes (iritis and conjunctivitis); inflammation in and around the lungs can lead to chest pain, especially on deep breathing, as well as shortness of breath; inflammation of the aorta (aortitis) can lead to leakage of the aortic valve. PA can also be aggravated by the concomitant occurrence of other diseases, such as obesity, type 2 diabetes, hypertension, and depression.[21,22]

PA affecting the TMJ was first described by Lundberg and Franks in 1965.[23,24] Radiographic TMJ involvement in patients with PA is reported to range from 24 to 82%, with clinical involvement of the masticatory system in about half of the patients with PA. The variation depends on the differences in the populations studied and the examination techniques used. Könönen compared 110 individuals with PA with a healthy control group matched for age, sex, and occlusal support and concluded that PA affects the masticatory system directly in 20 to 30% of patients.[25-28]

The most common masticatory symptom in patients with PA is pain in the TMJ area during function. Some patients report difficulty opening the mouth wide, but this is not typical in an unselected PA population. Typical clinical signs frequently found in patients with PA are tenderness of the TMJ and masticatory muscles to palpation and TMJ crepitations. Major occlusal changes are absent in individuals with PA. [28,29]

Radiographic changes in the TMJ are observed frequently, and the most common finding is erosion of the cortical outline, which is a typical sign of inflammatory TMJ involvement. Flattening of the mandibular condyle, which is often found in patients with PA, is also a common finding in those with RA, AS, and osteoarthrosis.[30-32]

In patients with PA, significant associations have been found between the radiographic changes in the TMJ (especially cortical erosion) and the subjective symptoms and clinical signs, which also correlate with the severity and extent of the joint disorder. The masticatory symptoms start, on average, 7 years after the onset of PA.[19]

II. CASE REPORT

A 36-year-old, white woman with psoriasis was referred to the Orthodontics and Gnathology Department of the University of Milan by her rheumatologist in May 2007. She had persistent pain over the temporomandibular area that increased on talking and biting, concomitant with occasional locking and TMJ sounds. She first noticed TMJ problems at the beginning of 2000 (Figs. 1 and 2). Since 2003, she has used a bite appliance to reduce the pain, which diminished but did not disappear. She continued this therapy until 2007, when she had an acute episode of temporomandibular pain that was so severe she visited a rheumatologist.

On clinical examination, the patient showed facial asymmetry, particularly evident on opening movement with deviation toward the left. The maximal interincisal distance was reduced to 20 mm. The left TMJ was tender to palpation, with marked crepitus, and the capsule was swollen.

The profile analysis showed a good relationship between the

maxilla and the nose and the nose–labial angle was normal. The mandibular zone was posterior relative to the maxilla at the level of the lower lip and chin. The angle between the lip and chin was acute.

The intraoral examination revealed large amalgam restorations on the upper left and lower right first molars and a ceramic crown on the lower left first molar. The occlusion analysis showed a good relationship between the upper and lower arches, with bilateral class I molar and canine relationships. In the anterior zone, marked dental crowding occurred in both the upper and lower arches with consequent rotation of all teeth. Marked gingival recession with clinical extension of all crowns was observed.

Panoramic, lateral, and posteroanterior radiographs were requested for a three-dimensional cephalometric analysis (Fig. 3, Table 1). The panoramic radiograph showed no serious dental abnormalities, except an impacted third right

Table 1) Steiner's analysis

Measurement	Units	Value	Mean	Difference	Deviation	
Skeletal Analysis						
SNA	Degrees	77.2	82.0 ± 2	-4.8	-xx	Maxillary retrusion
SNB	Degrees	74.8	80.0 ± 2	-5.2	-xx	Mandibular retrusion
ANB	Degrees	2.4	3.0 ± 2	-0.6		Class 1
SND	Degrees	72.2	76.0 ± 2	-3.8	-x	Mandibular retrusion
Length SE	mm	16.9	22.0 ± 2	-5.1	-xx	Reduced
Length SL	mm	35.2	51.0 ± 2	-15.8	-xxxxx	Reduced
Angle occlusal plane (Occl to SN)	Degrees	17.9	14.0 ± 2	3.9	x	Increased
Angle mandibular plane (GoGN-SN)	Degrees	40.3	32.0 ± 2	8.3	xx	Increased
Dental Analysis						
Upper incisor position (I/ to NA)	mm	6.9	4.0 ± 1.0	2.9	xx	Protrusion
Lower incisor position (I/ to NB)	mm	6.9	4.0 ± 1.0	2.9	xx	Protrusion
Upper incisor angle (I/ to NA)	Degrees	118.1	131.0 ± 6.0	-12.9	-xx	Protrusion
Lower incisor angle (I/ to NB)	Degrees	30.5	22.0 ± 2.0	8.5	xxxx	Proinclined
Lower incisor angle (I/ to NB)	Degrees	29.0	25.0 ± 2.0	4.0	xx	Proinclined
Soft Tissues Analysis						
Upper lip	mm	-2.0	0.0 ± 0.0	-2.0	-x	Labial retrusion
Lower lip	mm	-1.0	0.0 ± 0.0	-1.0	-x	Labial retrusion

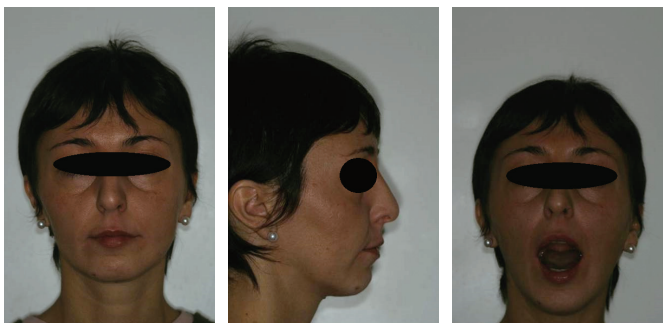

Fig. 1) The (a) frontal, (b) lateral, and (c) maximum opening facial views obtained before therapy

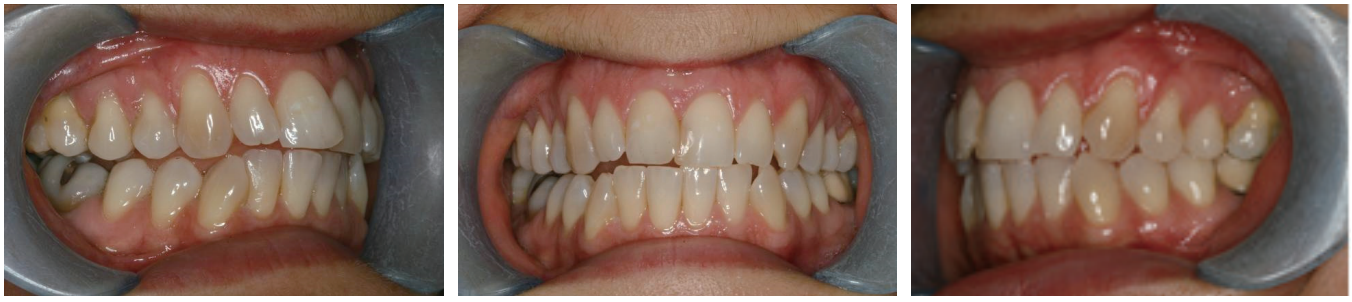

Fig. 2) The (a) lateral right, (b) frontal, and (c) lateral left intraoral views obtained before therapy

upper molar and some teeth with previous restorative and prosthetic treatments. The lateral radiograph was used for a cephalometric analysis according to Steiner. The analysis underlined a class I skeletal deformity with mandibular and maxillary retrusion and a long face. The upper and lower incisors were protruded, while the soft tissues of the upper and lower lips were retruded. The posteroanterior radiograph showed marked asymmetry between the right and left sides. The left mandibular ramus was much shorter than the right and deeper antegonial notching was observed.

Magnetic resonance imaging (MRI) showed a normal right TMJ, but also several abnormalities of the left TMJ (Fig. 4), including a visibly irregular superior profile of the mandibular condyle with an ulcer-like appearance that the radiologist attributed to degenerative changes. The left articular disc was positioned forward and medially, compared to the mandibular condyle. Moreover, reactive thickening of the bilaminar zone was observed.

The analysis of mouth opening showed a reduction in mandibular condyle excursion with respect to the eminence of the temporal bone, and the articular disc remained ahead of the mandibular condyle.

The temporomandibular function analysis showed reduced opening and noises during opening. Electromyography demonstrated an increase in masseter and anterior temporal muscular tone bilaterally with spasms (Fig. 5).

The computed mandibular scans and mandibular kinesiology evidenced a serious limitation in opening movement (18.9 mm). Moreover, during opening, deviation toward the left was observed. Lateral movement to the right was very limited, while movement to the left was normal (Fig. 6).

The kinesiographic study was repeated after applying transcutaneous electrical nerve stimulation (TENS) for 45 minutes, and was unchanged. The kinesiographic analysis of the muscles demonstrated that the disorder was specifically temporomandibular, and it was not occlusal.

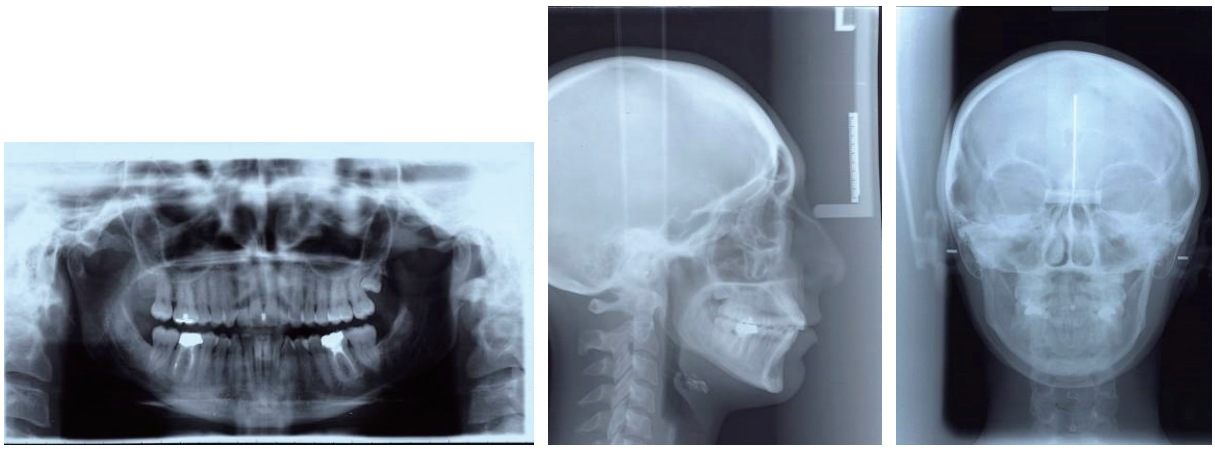

Fig. 3) (a) Panoramic, (b) lateral, and (c) posteroanterior radiographs

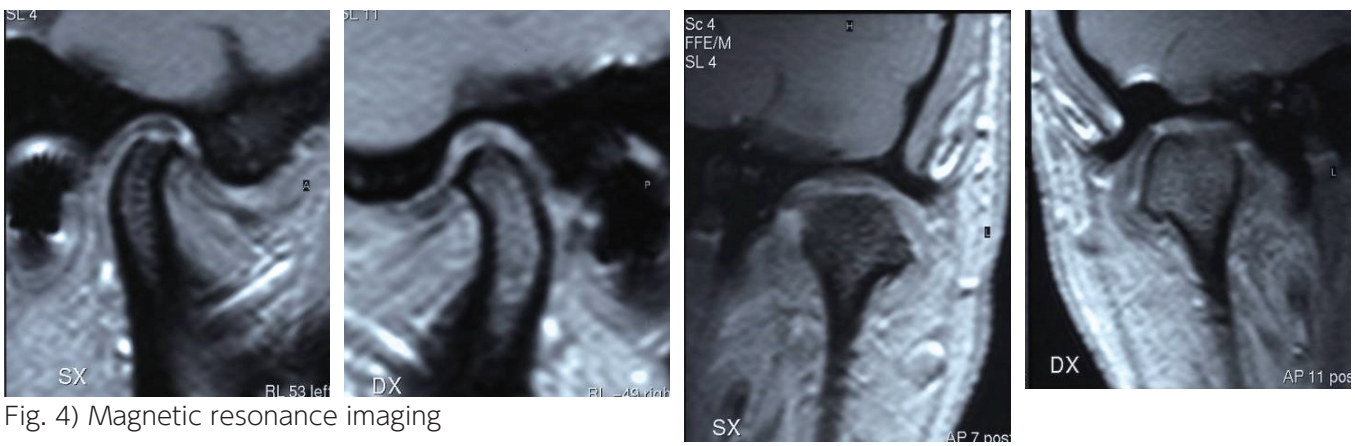

Fig. 4) Magnetic resonance imaging

The patient has had psoriasis of the skin since adolescence, without it affecting the nails or joints.

Although the temporomandibular findings in the patient were similar to those in PA, the diagnosis of PA had never been made.

Functional therapy to reduce the pain was proposed, which consisted of an anterior bite plane appliance and daily myofunctional exercises (Fig. 7). After 1 month of therapy, the left TMJ pain disappeared (Figs. 8 and 9). Four months later, the patient complained of mild foot pain. Since PA was now strongly suspected, the patient was sent to a rheumatologist to confirm the diagnosis, which was made after another 3 months. The rheumatologist detected asymmetric PA with calcaneal enthesitis (juxta-articular inflammation of the foot), which also clearly explained the previous temporomandibular disorder.

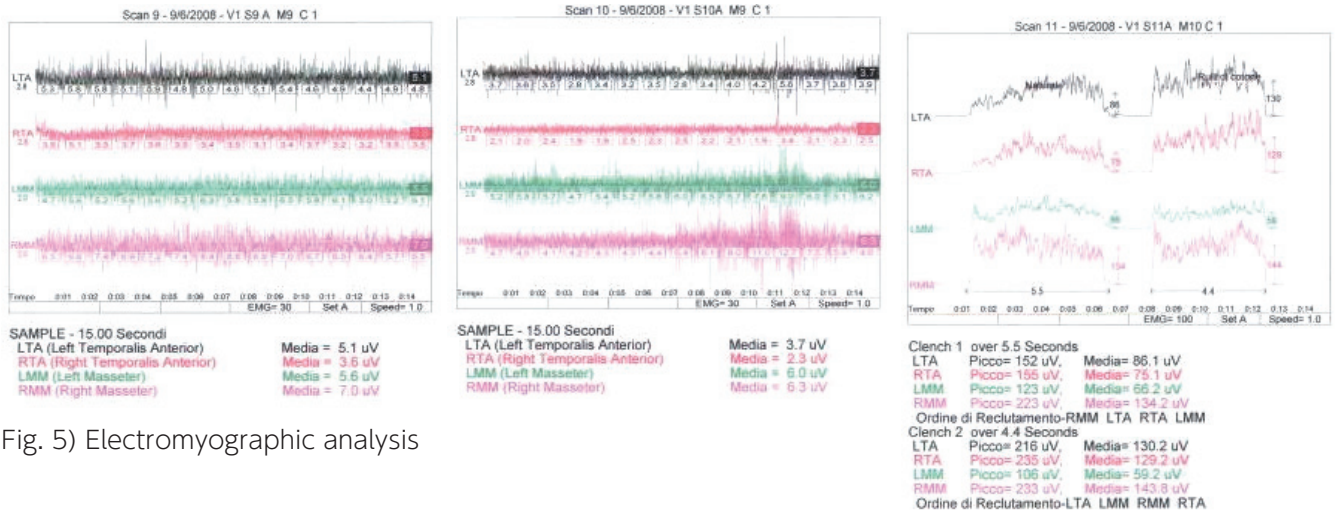

Fig. 5) Electromyographic analysis

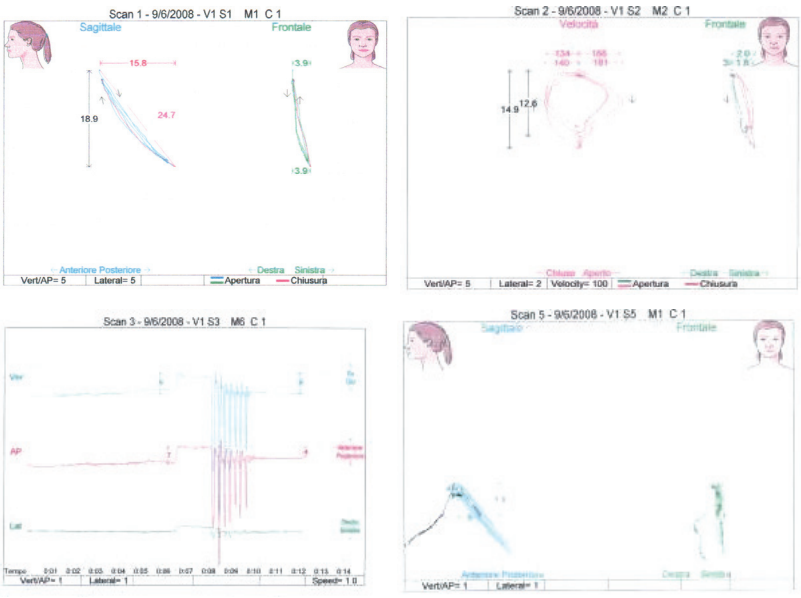

Fig. 6) Kinesiographic analysis

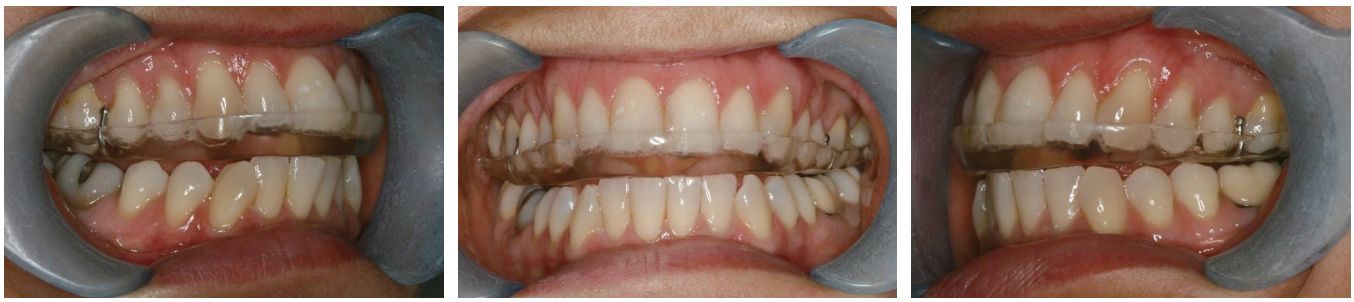

Fig. 7) The maxillary bite plane appliance, seen from the (a) lateral right, (b) frontal, and (c) lateral left intraoral views

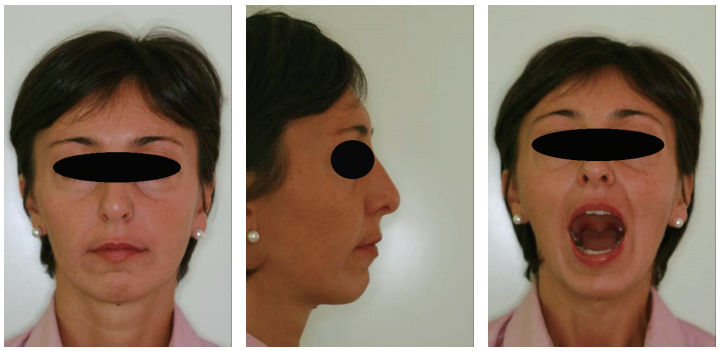

Fig. 8) The (a) frontal, (b) lateral, and (c) maximum opening facial views during gnathologic therapy

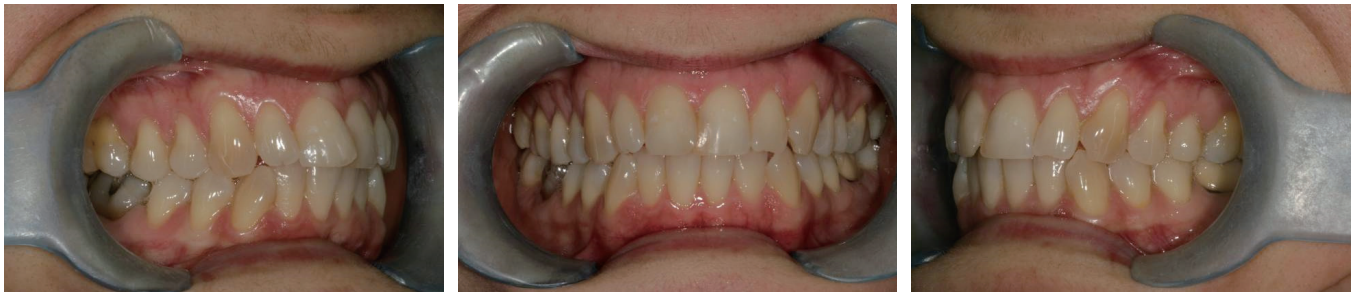

Fig. 9) The (a) lateral right, (b) frontal, and (c) lateral left intraoral views during gnathologic therapy

III. DISCUSSION

PA can develop slowly with mild symptoms, or it can develop quickly and be severe. The cause is probably multifactorial, and histocompatibility studies suggest that the condition is associated with several other systemic conditions, including Crohn's disease, Reiter's, Behçet's, and Sjögren's syndromes, and aortic incompetence. Heredity and a history of trauma are important factors in the development of PA.[19]

Despite the prevalence of PA in other joints, fewer than 35 cases of PA of the TMJ have been reported in the English literature.[33-35] However, some studies suggest that TMJ involvement in PA is more common and could be as great as 20%. When PA affects the TMJ, the onset of symptoms occurs by the fourth decade of life and is usually associated with a history of trauma.[36,37]

The arthritic changes of PA are mostly limited to the distal interphalangeal and metacarpophalangeal joints, but may occasionally extend to the proximal interphalangeal joints, producing characteristic sausage-shaped digits.[38]

The systemic symptoms of PA include fever, generalized fatigue, myalgia, weight loss, fatigue, and malaise. Joint symptoms include morning stiffness and pain, swelling, and limitation and reduced range of motion with tendonitis and

bursitis. Other associated symptoms include nail dystrophy in the form of pitting and onycholysis in 80% of patients and eye symptoms including redness and pain, with conjunctivitis, iritis, and uveitis. Mitral valve prolapse is more frequent in patients with PA than in the general population.[39]

PA of the TMJ is often unilateral, with a sudden onset. Moreover, it is episodic in nature and may undergo spontaneous remission. Symptoms include pain and tenderness of the joint area and the muscles of mastication, morning stiffness, tiredness in the jaws, joint crepitation, occasional painful swelling of the TMJ capsule and painful mandibular movements associated with a progressive decrease in the interincisal opening. In severe cases, ankylosis of the TMJ may occur.[34]

Reduced opening, deviation, and a newly developed anterior open bite may indicate the degree of joint involvement. In addition, noting whether the disease is unilateral or bilateral, and if bilateral condylar resorption occurs, is important. Joint crepitation is generally an indication of advanced joint disease, whereas clicking is usually indicative of disc deformity or disc position abnormalities.[40,41]

Radiographic changes associated with PA may be seen in as many as 82% of the patients with affected TMJ and include erosion, flattening, osteoporosis, loss of joint mobility, and extreme joint space narrowing. Subchondral bone cysts, subluxation, and ankylosis are also occasionally observed. The radiographic changes are nonspecific and cannot be easily distinguished from those of other types of arthritis, particularly RA and AS.[42]

If bony changes are seen on an initial screening view, such as a panoramic radiograph, additional imaging studies should be carried out. These radiologic examinations may include MRI, Cone Beam CT, radionuclide bone scans, and ultrasonography. These imaging studies provide more detailed information about any joint involvement. MRI can be used to detect early arthritic changes, determine disc position, and note joint effusions or other fluid collections.[43] Radionuclide scans and Cone Beam CT are quite sensitive for detecting reactive osteoblastic processes, even in the absence of conventional radiographic changes. In addition, ultrasonography can be used to diagnose certain pathological changes in the TMJ, as in RA and PA.[44]

Since the symptoms of TMJ involvement are generally nonspecific, the diagnosis of PA of the TMJ is difficult and is based mainly on the systemic presentation of the disease. In general, the diagnosis is based on the triad of psoriasis, radiographic evidence of erosive polyarthritis, and a negative serologic test for rheumatoid factor (RF). However, even in the presence of a skin rash, the diagnosis of PA cannot be confirmed absolutely. The differential diagnosis of PA should always include RA, Reiter's syndrome, AS, and gout. No definitive test for PA exists. The diagnosis is made mostly on a clinical basis and by a process of elimination.[1,32,33]

The patient's medical history, physical examination, laboratory tests, and imaging studies may be used to diagnose PA. The symptoms of PA are similar to those of other arthritic diseases. RA generally involves joints symmetrically on both sides of the body, and it may produce bumps under the skin, which are not present in PA. However, some forms of PA look very similar. The simultaneous presence of psoriasis on the skin and nail changes supports a diagnosis of PA.

Specific antibodies, the RF (70%-80%) and the anti-cyclic citrullinated peptide or anti-CCP (78.5-97.8%), are normally present in RA, while they are not usually found in the blood of patients with PA.[45,46]

Gout has a characteristic involvement of the great toe, causing pain and swelling. Fluid drawn from the inflamed joints by arthrocentesis reveals serum uric acid, which is typically elevated in gouty arthritis, although PA and AS are also occasionally associated with elevated levels. Distinguishing between these two forms of arthritis is important because they are treated with different medications. Conversely, many of the treatments for PA and RA overlap. Fever is most frequently associated with infectious arthritis. Red patches on the face, loss of demarcation of the vermilion border, restricted mouth opening, and pitted nails are all signs of scleroderma and other collagen diseases. Certain laboratory tests occasionally provide information that may lead to confirmation of the diagnosis. However, laboratory tests should be used with care in the diagnosis of arthritic disease because of their lack of specificity, the degree of overlap in the test results among these diseases, and the possibility of false-positive results.[19,25]

The most commonly used tests after RF and uric acid are the antinuclear antibody (ANA) test, which is positive in 50% of patients with RA and most patients with collagen diseases, C-reactive protein, which is elevated mostly in RA, Reiter's syndrome, and collagen diseases, and the erythrocyte sedimentation rate (ESR), which is elevated in various inflammatory states, including RA, although it is nonspecific.[19]

Generally, the treatment of PA involves a combination of anti-inflammatory medications (specifically, nonsteroidal anti-inflammatory drugs, or NSAIDs) and exercise. If progressive inflammation and joint destruction occur despite NSAIDs treatment, more potent medications are used, such as methotrexate, corticosteroids, and antimalarial medications. In arthritis, exercises are performed to improve strength and to maintain or improve joint range of motion. NSAIDs treatment helps to decrease joint inflammation, pain, and stiffness, but frequently causes side effects. Newer NSAIDs called Cox-2 inhibitors cause gastrointestinal problems less frequently.[6,19] Disease-modifying antirheumatic drugs (DMARDs) may relieve the more severe symptoms and attempt to slow or stop joint and tissue damage and the progression of PA. Biological drugs are also considered DMARDs. They are highly selective agents that target specific internal events in the body that cause psoriasis and PA.[32,33]

Infliximab adalimumab etanercept are monoclonal antibodies, specific for TNF-alpha. They are effective and well tolerated in patients who are not responsive to DMARDs. [47,48]

IV. CONCLUSION

The early detection, diagnosis, and treatment of PA can relieve pain and inflammation and might help to prevent progressive joint involvement and damage. A holistic, multidisciplinary approach to PA treatment is very important as it highlights the many factors that affect disease management. The orthodontist should recommend treatments based on the type of PA, its severity, and the patient's response to therapy. In addition to exercise and local pain treatment, an occlusal splint may be used to help keep the TMJ working properly, improve function, relieve pain, reduce swelling, and prevent further severe TMJ damage. Given the complexity of managing PA, the interdisciplinary approach should focus on health education and promotion as means of prevention.

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