

Temporomandibular joint alterations and their orofacial complications in patients with juvenile idiopathic arthritis

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ABSTRACT

Patients with juvenile idiopathic arthritis (JIA) can have alterations in bone metabolism and skeletal growth, as well as damage to the temporomandibular joint (TMJ), which can generate extra and/or intraoral alterations, resulting in craniofacial disorders. Our goal is to carry out a review of the literature on orofacial alterations in patients with JIA. Among the orofacial disorders in patients with JIA, alterations in mandibular growth, caused by dysfunctions in the TMJ region, seem highly prevalent in these patients. The most often found alterations are: retrognathia, micrognathia, anterior open bite, dental crowding, facial asymmetry and mouth opening limitation. Thus, the rheumatologist becomes a key agent in the early detection of these disorders, helping with patient referral to a dentist. The diagnosis, in turn, should be performed by the orthodontist, using clinical examination and imaging methods, allowing early treatment and a favorable prognosis. TMJ disorders should be treated by a multidisciplinary team, including pharmacological treatment for pain control and dental care through functional appliance and/or orthodontic therapy, physical therapy and sometimes, speech therapy. We conclude that among the orofacial disorders in patients with JIA, alterations in mandibular growth generated by dysfunctions in the TMJ region seem highly prevalent. Such dysfunctions can cause mainly open bite, mandibular retrusion, micrognathia, dental crowding and facial asymmetry. The rheumatologist can detect these alterations at an early stage, with immediate patient referral to a team that should preferably be a multidisciplinary one, consisting of an orthodontist, physical therapist and speech therapist, to reduce future occlusal and mandibular growth complications.

Keywords: juvenile rheumatoid arthritis, orthodontics, temporomandibular joint.

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INTRODUCTION

Juvenile idiopathic arthritis (JIA) is the most common chronic rheumatic disease in childhood and adolescence and comprises a group of distinct clinical entities of unknown etiology.¹ This disease is characterized by joint inflammation with symptoms persisting for more than six weeks and onset before 16 years of age.^{2,3} Currently, it is classified according to the International League of Associations for Rheumatology (ILAR) as: systemic arthritis, polyarthritis (RF negative), polyarthritis (RF positive),

oligoarthritis (persistent or extended), with enthesitis-related arthritis, psoriatic arthritis, and undifferentiated.⁴ A long-term complication of JIA is the decrease in bone mineral density at sites distant from the initial onset of the disease, causing alterations in bone growth and overall maturation.^{3,5} These alterations may lead to impaired mandibular growth, causing orofacial alterations that are significant in the adulthood of patients with JIA and may have direct impact on the quality of life of these individuals.⁵

Early diagnosis of facial growth disorders and temporomandibular joint (TMJ) dysfunctions seems to minimize future

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dental problems in JIA patients and increase the successful treatment of such alterations in these individuals.⁶ Thus, this article aims to provide a review of literature on TMJ involvement in JIA patients and its orofacial consequences.

PATHOGENESIS OF JUVENILE IDIOPATHIC ARTHRITIS

The articular and periarticular impairments observed in chronic arthritis begin with inflammatory alterations observed in the synovial membrane, called synovitis. Moreover, in the TMJ, the synovitis at the microscope can be characterized by an exudative phase, cell infiltration and, finally, by granulation tissue formation. These phases are interrelated with active phase of the disease and subsequent immunopathological events perpetuate this initial inflammatory reaction, which develops into the chronic disease process.⁷

During the active disease process, the released cytokines, especially IL-1 and TNF- α , promote the recruitment of more neutrophils, inducing edema and categorizing the joint effusion phase. These activated neutrophils are responsible for tissue destruction and release of enzymes that break collagen and other matrix proteins. At this moment, TCD4 lymphocytes will be attracted to the synovium, becoming the predominant cells in the synovial region. Such cells have the ability to clone and release mediators that stimulate inflammatory cells. These inflammatory cells, such as macrophages, release proinflammatory cytokines such as IL-1- β , TNF- α , IL-6, and IL-8, which in turn stimulate the release of proteases by other cells such as neutrophils, causing more damage to the joint tissue.⁷⁻⁹

With the joint tissue destruction, granulation tissue is formed and replaces the damaged tissue. This tissue, in turn, is capable of penetrating the joint cartilage and subchondral bone, forming the *pannus*. Finally, the hyaline cartilage can be replaced by dense fibrous tissue, and thus, by penetrating the subchondral bone it may or may not generate ankylosis. In JIA, ankylosis is, in most cases, fibrous ankylosis and rarely the bony type.⁹

TMJ IMPAIRMENT AND OROFACIAL EFFECTS

JIA can cause, in addition to functional impairment of the initially affected joint, juxta-articular or periarticular bone lesions. Studies show that patients with JIA may have overall growth impairment,¹⁰⁻¹² which can affect 10%–20% of patients, with a higher incidence in certain subtypes of the disease, such as systemic and polyarticular subtypes.¹³ Although the etiology of this growth retardation is unknown, several factors

have been implicated, such as the presence of high levels of proinflammatory cytokines, chronic use of corticosteroids to control the disease, malnutrition, and immobilization.^{13,14} The use of corticoids and the marked increase in proinflammatory cytokines cause an alteration in growth hormone (GH) release.¹⁴ Simultaneous to the previous fact, patients with JIA, particularly those with the polyarticular and systemic subtypes, clearly show a decrease in muscle mass which, one believes, also causes a decrease in the bone mass of these individuals.¹⁴ These facts impair not only the overall growth of the patient, but also the mandibular growth.

Furthermore, in the case of the mandibular bone, in addition to GH release deficiency there are two other facts: a) erosive lesions in the condylar region, which have very high prevalence in patients with JIA (around 75%),¹⁵⁻¹⁷ being caused by the inflammatory process of JIA^{5,10,13,16-18} and b) premature closing of the epiphyseal bone, due to the prolonged use of corticosteroids during the growth phase.¹⁸ Such actions cause mandibular growth impairment, as the condylar region is known to be the main growth center in this region. These alterations seem to become more evident in patients aged 9 to 12 years,¹⁸ a period when there is a growth spurt and during which jaw growth is expected to be accelerated. As a result, it is not uncommon for JIA patients to have micrognathia and/or posterior rotation of the mandible^{13,18} (which may be observed in the patient's profile, with the chin region being too posterior when compared to the skull base), as well as an open bite, in which the anterior teeth have no contact during occlusion. If only one TMJ is affected by the disease, facial asymmetry occurs, as in this case only one area of mandibular growth is compromised, while the opposite will show normal growth.^{13,18}

During the clinical examination in JIA, it is very common to detect a marked mouth opening limitation, especially if the disease is in the active stage.^{18,19} A mouth opening measurement < 29.5 mm can be considered limited in children up to 3 years old; 34.5 mm in children up to 6 years old, and 39.5 mm for those older than 7 years.²⁰ This movement restriction also influences mandibular growth, as the bone growth stimulation of muscle traction becomes almost nonexistent.

The JIA subtype also influences the severity of orofacial alterations. In the polyarticular type, these alterations are more prominent when compared to other subtypes.^{21,22} The time of disease onset also has a strong influence on abnormal mandibular growth,²³ as the earlier the disease starts, the greater the likelihood of occurrence of adverse effects caused by the treatment and the degenerative effects of the underlying disease on the normal development of the mandible, as described above.

Patients with JIA, when compared to healthy subjects, have decreased jaw and body dimensions, as well as decreased mandibular branch height.¹⁶ A physiological compensation for this deficiency results in the protrusion of the upper and lower incisors, causing a decrease in the interincisal angle (angle between the tips of the upper and lower incisors).¹⁷ This picture can be observed clinically, and the patient has a convex profile, caused by the biprotrusion.

The posterior rotation of the mandible and subsequent decrease in the mandibular dimensions in patients with JIA can lead to an anterior open bite.^{6,17-19} This open bite, in addition to the esthetic aspect, promotes a mastication overload to the posterior elements, which may cause from mastication discomfort to the most intense pain in the TMJ region. However, the compensatory vertical growth occurs often in the anterior mandibular alveolar bone, making the lower and upper incisors undergo overeruption, which can clinically mask an installed open bite.⁹

According to studies, the prevalence of TMJ dysfunction in patients with JIA can vary between 25%–75%,^{1,2,12,17} and this discrepancy can be explained by the fact that the studies cover different ages, JIA subtypes and duration of disease.¹² According to Ringold et al.,¹ although there is a high prevalence, 69% of patients diagnosed with temporomandibular disorders (TMD) are asymptomatic.

DIAGNOSIS OF ALTERATIONS IN THE TMJ

A correct diagnosis is important to distinguish between dental malocclusion and bone discrepancy,⁶ thus differentiating the normal growth trajectory from an abnormal growth caused by a pathological condition. There is a strong need for criteria to facilitate the early diagnosis of TMJ involvement in order to establish an early treatment. It is highly recommended to follow the masticatory function of children with JIA, as well as the TMJ functional capacity.⁶

It is essential that rheumatologists understand their role in the early detection and referral of patients with JIA, including examination of this joint in clinical practice. The following should be considered as parameters: limited mouth opening, pain, mandibular deviation upon opening and/or closing, snapping, cracking, and¹² sharp mandibular retrusion. Such retrusion can be clinically characterized by evaluating the patient profile and observing whether the position of the mental region is too posterior to the skull base.

As an aid to diagnosis, panoramic or transcranial radiographs can be used, as well as ultrasound and magnetic resonance imaging of the joint region. MRI is considered the best

form of early diagnosis, as it can detect alterations in muscle, synovial cartilage, and bone, even in early phases.^{24,21}

The only disadvantage of MRI is the fact that very young patients need to be sedated for the examination.^{12,22,25} Panoramic radiography has advantages such as low cost, low radiation, and reliability, but it only allows the visualization of lesions at a later phase, when the condylar erosions are already established. Ultrasonography is quite limited by the anatomy of the TMJ region, thus being rarely used.²¹

DISCUSSION

Bilateral TMJ involvement is more common in patients with JIA, corresponding to a prevalence of 74%,^{1,17,11} which can lead to growth deficiency of the entire mandibular region, causing all previously described orofacial alterations. The unilateral involvement can cause clinically detectable facial asymmetry, explained by the asymmetric growth of the jawbone.^{17,26} Furthermore, studies have shown that corticoid use, together with marked increase in inflammatory interleukins, directly interfere with GH release during the course of the disease, in addition to stimulating the production of osteoclasts and osteoblast suppression.^{11,13,14} Early diagnosis of disease activity on the TMJ is more difficult than in other joints affected by JIA, as clinical symptoms can be very subtle in children.⁶ This diagnosis becomes easier when there are lesions in the condylar region, observed on radiographs and magnetic resonance imaging.^{5,6} Patients with TMJ affected by the disease also have short masseter muscle. The presence of this characteristic is observed most often in very young patients diagnosed with JIA and TMD, as pain caused by the latter inhibits the development of the masseter muscle, which leads to mandibular bone growth arrest.²⁴ When the masseter growth deficiency is clinically established, the healthcare professional can observe that the patient, when attempting full opening of the mouth, has a deviation toward the side of the shortened muscle.⁶

In a longitudinal study that followed patients from the diagnostic phase of the disease (childhood) to adulthood, it was observed that there was no growth impairment regarding mandibular size, but a change in the direction of this growth.² However, a later study that evaluated 100 patients with different AIJ subtypes concluded that mandibular dimensions were decreased.¹⁶ Regardless of the type of alteration, patients will have a clinical picture of micrognathia.

A recent study that assessed 60 patients with a diagnosis of JIA that was made 27 years before, it was concluded that there was some sort of mandibular growth disorder; however

a direct association with TMJ involvement caused by JIA was not demonstrated. Micrognathia showed to have a direct association with bilateral involvement of the TMJ.²⁷

In a cross-sectional study carried out for 12 months on the incidence of TMJ involvement by JIA detected by panoramic radiography, it was concluded that 66% of patients showed improvement of temporomandibular dysfunction in relation to improvement of JIA signs.²⁸

CONCLUSION

Among the orofacial disorders in patients with JIA, alterations in mandibular growth generated by dysfunctions in the TMJ

region seem to be highly prevalent. These dysfunctions may result mainly in open bite, mandibular retrusion, micrognathia, dental crowding, and facial asymmetry. The rheumatologist can detect these changes at an early stage, referring the patient to a team, which should preferably be a multidisciplinary one, consisting of an orthodontist, a physical therapist, and a speech therapist, aiming to reduce future occlusal and mandibular growth complications.

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