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Case Report

INTRAMUSCULAR OEDEMA AFTER TRUNCULAR ANALGESIA DIAGNOSED BY MRI: A CASE REPORT AND DIFFERENTIAL DIAGNOSIS OF MOUTH OPENING LIMITATION

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ABSTRACT

Mouth opening limitation (MOL) is an important clinical sign generally referred to as temporomandibular disorders (TMD) but MOL can also be due to other pathologies as neoformations. The first level's radiological exam is an orthopantomography that helps the clinician choose the most appropriate second-level exam. MRI is the gold standard for TMD, while multislices CT generally investigates maxillofacial pathologies. Cone beam CT (CBCT) with an appropriate FoV is recommended if a contrast agent is not indicated. Muscle contractures frequently cause MOL as prolonged mouth opening during dental treatment, antalgic contractures due to any infectious-inflammatory process, or after surgery. An MRI investigation of a case of intramuscular oedema after troncular analgesia has never been documented in the literature.

A 36-year-old female with MOL (21 mm) with midline deflection to the left side since the last 15 days is presented. The patient reported that she had endodontic therapy on 4.6 after troncular analgesia at the ipsilateral ascending mandibular ramus just before the onset of symptoms. She had never experienced symptoms of TMJ dysfunction a before T2/DP-weighted TMJ MRI with T1/T2-weighted scans of the pterygoid muscles in axial and coronal planes with a dedicated coil was performed. The images demonstrate the presence of a reducing disc displacement in the right TMJ and a non-reducing disc displacement in the left TMJ with a limitation of condyle translation. An inhomogeneous area of isointense in T1 and hyperintense in T2, referable to intramuscular oedema resulting from troncular analgesia, was appreciated At the right internal pterygoid muscle, at the level of the spine of Spix.

MRI is an essential method in the differential diagnosis of MOL and allows targeted and resolutive therapy.

KEYWORDS: *MRI, pterygoid muscles, temporomandibular disorders*

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INTRODUCTION

During oral examination (OE) mouth opening limitation (MOL) is one of the most important clinical signs to be detected. In most cases, MOL is related to chronic trauma of the temporomandibular joints (TMJ). More rarely, the cause is related to specific arthritis, malformations, trauma outcomes, or neoformations.

Functional alterations may result from intraarticular adhesions (1) or extraarticular causes that determine a limitation of mandibular movements (2).

Intraarticular causes could be a joint lock, neoformations, chronic arthritis (post-traumatic, infectious, specific arthritis), intracapsular fracture outcomes, and ankylosis. In contrast, extraarticular ones are neoformations, dysplastic lesions, malformations or fractures of the facial bones that can cause an impediment to mandibular movements, muscular contractures (post-traumatic, infectious, antalgic, and in dysfunctional patients), scarring (post-traumatic or following burns), progressive systemic sclerosis, connective tissue diseases and outcomes of demolition resective surgery with reconstruction of the jaws tissues and systemic diseases leading to a deficit of the neuromuscular system. Therefore, the differential diagnosis of MOL is mainly performed by anamnestic investigation and oral examination and completed by imaging.

The first level's radiological exam is an orthopantomography (OPT) of the dental arches, which helps the clinician choose the most appropriate second-level exam.

MRI is the gold standard for temporomandibular disorders (TMD), while multislices CT generally investigates maxillofacial pathologies (fractures, malformations, dysplastic and neoplastic lesions). CBCT with an appropriate FoV is recommended if a contrast agent is not indicated. Among the intraarticular causes are the TMJ lock, usually caused by chronic inflammation resulting from a non-reducible disc dislocation clinically manifested by a limitation of the mouth opening (1). The lock is defined as chronic when condylar hypomobility remains after several episodes of remission and exacerbation of symptoms. The mandibular kinetics shows a limitation of mouth opening of less than 30mm. Clinically, non-specific symptoms such as cervicalgia, headache, tinnitus, and vertigo may also be present. On OE, there is a deviation of the mandible towards the locked TMJ both during opening and protrusion. On palpation of the locked joint, no clicks can be appreciated, and rarely can a crackling be perceived. The joint compression test causes vivid pain. The end-feel test does not increase the mouth opening and causes severe pain in the locked joint. MRI shows a condylar hypomobility with the articular disc displaced anteriorly. It is also easy to find T2W hyperintense intra-articular joint effusion, retrodiscal fibrosis, and remodeling of the articular surface of the condyle (3). CBCT is only useful in cases of gross remodelling of the condyle. Here a case of intramuscular oedema resulting from troncular analgesia is reported, and the pertinent literature is discussed.

CASE REPORT

A 36-year-old female patient presented with MOL (21 mm) with midline deviation to the left side since the last 15 days. At the time of her medical history, she reported that she had undergone endodontic therapy on 4.6 after troncular analgesia at the ipsilateral ascending mandibular ramus just before the onset of symptoms. She had never experienced symptoms of TMJ dysfunction before the MOL had started after dental treatment and had progressively worsened in about 10 days.

She reported pain in the right temporomandibular region, at the level of the right internal pterygoid muscle and right masseter, which were painful on palpation (80 VAS), and limited pain in the left temporomandibular area (40 VAS). The end-feel test did not allow an increase in maximum mouth opening and caused pain in the left TMJ. The patient presented an occlusal class I. She also reported frequent teeth clenching. Rx OPT was negative.

A TMJ MRI with T1/T2-weighted scans of the pterygoid muscles in axial and coronal planes with a dedicated coil was performed. The images demonstrate the presence of a reducing disc displacement in the right TMJ and a non-reducing disc displacement in the left TMJ with a limitation of condyle translation. Furthermore, at the right internal pterygoid muscle, at the level of the spine of Spix, an inhomogeneous area of isointense in T1 and hyperintense in T2 scans referable to intramuscular oedema probably resulting from troncular analgesia, was detected (Fig. 1-3).

DISCUSSION

Frequently, dental therapies requiring very long sessions, such as endodontic treatment of a molar, result in muscle spasms and/or antalgic contractures that resolve spontaneously or with appropriate medical therapy within a couple of weeks. In some cases, these muscle spasms can lead to joint dysfunction or worsen the prognosis of an already pre-

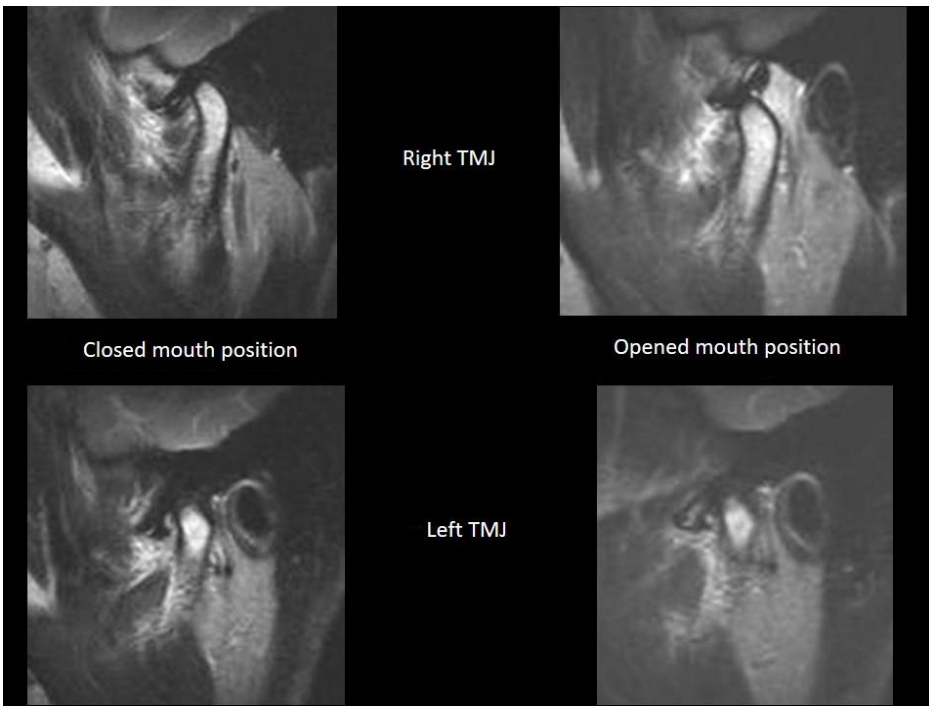

Fig. 1. *Turbo spin-echo T2-weighted MRI with parasagittal scans: top images of the right TMJ, bottom images of the left TMJ; left with mouth closed and right with mouth open.*

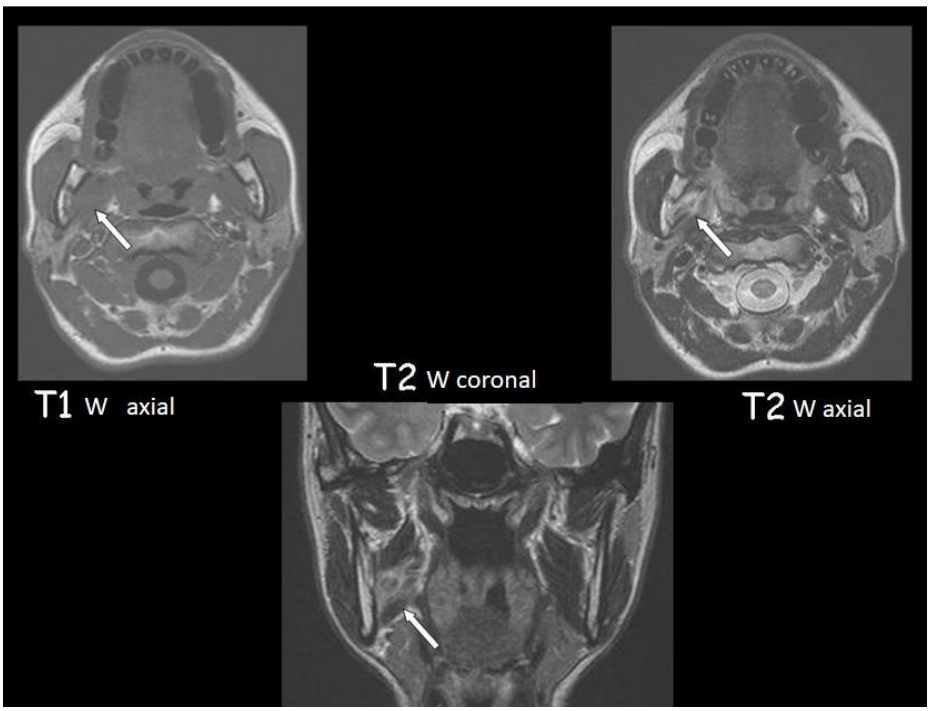

Fig. 2. *MRI of the maxillofacial structures with axial T1- and T2-weighted and coronal T2-weighted scans. The white arrow indicates the lesion inside the right internal pterygoid muscle which is isointense in T1 and hyperintense in T2.*

existing dysfunction. Troncular analgesia alone may also lead to muscle contracture due to oedema at the injection site and, thus, to MOL. In order to make a proper diagnosis, an MRI scan to evaluate both the muscles and the TMJ is needed. In the MRI request the clinician must to point out the suspicion of a lesion in a specific muscle to allow the radiologist to perform an adequate MRI examination. To perform the correct diagnosis several pathological condition has to be excluded in differential diagnosis. In addition, MOL can be due to several conditions.

A benign neoformation (4) such as a chondroma, osteoma or osteochondroma and synovial chondromatosis (5) can cause inflammation and alteration of the joint tissues such as adhesions and thus functional limitations but is unlikely to cause MOL on its own.

Regarding chronic arthritis, the patient often reports a previous trauma (fracture, whiplash) leading to osteoarthritis and thus to chronic inflammation, or, more rarely, recurrent infections (in debilitated, immunocompromised patients) or with psoriasis, rheumatoid arthritis and other autoimmune diseases.

In the case of outcomes of intracapsular fracture, imaging is essential to understand the cause of the MOL. OPT may show remodelling of the condylar head, but CBCT scan is the elective study; dynamic MRI is equally essential to evaluate the condylar-discal complex and to exclude the presence of other pathologies (6, 7).

TMJ ankylosis is the last stage of diseases such as TMJ dysfunction, condyle fractures and chronic arthritis that evolve into fibrosis and then bony ankylosis. The patient's clinical history will differ depending on the etiopathogenesis of the ankylosis and a simple OPT could be sufficient to make the diagnosis. A CBCT scan of TMJ will be used to describe the extent of the ankylotic block and to plan TMJ surgery (8).

Among the extraarticular causes that could lead to MOL we find dysplastic alterations as fibrous dysplasia (9), ossifying fibroma, cherubism (10) and leontiasis.

Malignant tumours can cause a MOL either directly by causing adhesions with the mandible (parotid lodge) or a mechanical block to mandibular movements (infratemporal fossa) or indirectly by involving the masticatory elevator muscles (temporalis, masseter and internal pterygoid) causing trismus (11).

In our report, clinical history shows a sudden onset of MOL without a previous history of joint dysfunction. Generally, the patient may be asymptomatic or report pain; in addition to MOL , signs such as intra- and/or extra-oral swellings, swelling of the cervico-facial lymph nodes, erythro/leukoplasic lesions (even ulcerated) in the oral cavity in particular on the retromolar trigone, soft palate and tonsillar lodge, epistaxis, sudden tooth mobility, visual changes and more generally neurological symptoms related to cranial nerve or central nervous system alterations and signs of endocranial hypertension can be appreciated.

The OPT may be negative or show the absence of rear wall of the maxillary sinus (12) or alterations of the ascending

Fig. 3. *Maximum mouth opening and lateral right movement, protrusive movement, and lateral left movement.*

branch of the mandible. Multislices CT with contrast agent and MRI (13) are indicated.

In case of maxillofacial fractures (14) which cause an impairment of mandibular movements, CBCT are used for diagnosis and to plan surgical therapy.

The hypertrophy of the coronoid processes cause an increase in the height of the coronoid processes, which impacts against the posterior wall of the zygoma, resulting in a MOL (15). An OPT or a CBCT is sufficient for a diagnosis.

Muscle contractures can be caused by a prolonged opening of the mouth such as during dental treatment, antalgic contractures due to any infectious-inflammatory process (abscesses and odontogenic phlegmons involving the masseter or internal pterygoid), the outcome of trauma with skin ecchymosis, intramuscular hemangioma (16) and intramuscular oedema, surgery such as the extraction of a third molar (17);

Scarring outcomes as a result of facial skin burns, can limited the mouth opening as well as progressive systemic sclerosis (18) and connectivopathies result in a progressive sclerosis of the connective tissues, particularly the perioral tissues (tobacco bag mouth) that reduces the mouth opening. The diagnosis is clinical and a referral to a specialist is recommended for a general assessment of the patient's health.

Among the connectivopathies, scleroderma and in particular the Parry-Romberg syndrome, especially when it occurs during development, determines a localised atrophy of connective tissues (cutaneous and muscular) that blocks skeletal growth resulting in facial asymmetry and, in severe cases, even MOL.

Pedicled or revascularized flaps in maxillofacial surgery can lead to scar retractions that, together with submandibular muscle deficits caused by laterocervical emptying, can lead to MOL.

Systemic diseases leading to a deficit of the neuromuscular system can give rise to a spastic paresis of the masticatory muscles or a deficit of the trigeminal efferences. Hypercalcemia can also lead to 'tetanic' trismus of the masticatory muscles.

CONCLUSION

MRI is an essential method in the differential diagnosis of MOL and by highlighting intramuscular and articular lesions, it allows targeted and resolutive therapy.

For the first time, an MR imaging of a case with a MOL caused by intramuscular oedema at the injection site after mandibular nerve troncular analgesia is presented.

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