INTRODUCTION

Fibroadipose degeneration of masticatory muscles is a rare form of myopathy characterized by an almost total substitution of the masticatory muscles with fatty tissue\(^1\). The consequence of the progressive degeneration is a progressive limitation of mouth opening, caused by the loss of muscle fiber, with fibro-adipose replacement of the contractile tissue and muscle impairment in generating force. This kind of degeneration is correctly diagnoses with computed tomography (CT) and magnetic resonance imaging (MRI). The etiology is still unclear. This case report presents a fatty degeneration of masticatory muscles in a patient with marked limitations of jaw movements.

CASE REPORT

A 63-year-old woman was referred to the Department of Gnathology of the Dental School of Turin, with pain and difficulty in chewing. On clinical examination mouth opening was reduced to 12 mm. There was 10 mm movement to the right but only 5mm to the left. The opening movement and protrusion deflected to the right. TMJs were painless on palpation and without joint noise on auscultation as well. The temporal, internal pterygoid and masseter muscles areas, were all painful to palpation.

MRI showed a marked atrophy of the muscles masseter and internal pterygoid of right side, with replacement of muscle tissue by fat.
CT showed fatty attenuation values in all the masticatory muscles, with only few residual strands of parenchymal density, mostly within the internal pterygoid muscle. Coronal T1 and axial proton density T2 MRI confirmed the presence of an almost total substitution of the right masseter, pterygoid and temporal muscles with fatty tissue (Figures 1 – 4). Comparison between CT and MRI showed a similar degree of fibroadipose substitution. Clinical neurological examination and electromyography were negative, excluding either an extension to other muscles or a systemic neuropathy.

3. DISCUSSION

This study describes, not only clinically but also through CT and MRI, a rare case of fibroadipose degeneration of masticatory muscles. The patient presented limitations of mouth opening and masticatory muscle pain as chief complaints. Fatty replacing of masticatory muscles was correctly diagnosed by CT and MRI, which is an important tool in the evaluation of skeletal muscle2,3. On MRI fatty replacement can be seen as hyperintense signal relative to muscle on T1-WI2,3. Increased signal intensity on T2-WI and isointensity on T1-WI are characteristic of an inflammatory process or of edema of the muscles3. Because of its relative long relaxation time, mainly in fast-spin echo techniques, fat is also seen as hyperintense signal on T2-WI, which makes it difficult to differentiate fat from edema or inflammation.

In existing Literature fatty degeneration of masticatory muscles is a very rare case and similar MRI features are reported only in other few previous papers. Pomatto et al.1 reported a similar case in a 43-years-old. Etiology was unknown, but symptoms improved thanks to myotomy of masseter and internal pterygoid muscles and coronoidotomy. Zanoteli et al4. described a bilateral involvement of the masticatory muscles due to centronuclear myopathy. The authors reported that The main abnormalities were centrally placed nuclei in 90% of the muscle fibers, type 1 fiber predominance, fiber size variability, a mild increase of endomysial connective tissue, and a marked increase of perimysial adipose tissue.

Pang et al5. reported a case of a patient affected by amyotrophic lateral sclerosis (ALS) with limitations of mouth opening and masticatory muscle pain. Diagnosis was based only on clinical examination and the case was initially misdiagnosed as temporo-mandibular disorder. Due to the severity and the persisting of disturbances, instrumental examination was performed. CT and MRI
revealed the presence of advanced fatty atrophy with reduced volume of both sides of the masseter, medial pterygoid, lateral pterygoid, and temporalis muscles.

Powers et al\textsuperscript{6} described severe jaw movement limitation in a patient affected by another congenital myopathy, the nemaline myopathy. TMJ was intact, but there was a marked atrophy and fibrosis of the masticatory muscles caused by the disease, leading to a TMJ pseudoankylosis. Fibrous ankyloses has been correlated with immobilization of the TMJ consequent to orthognathic surgery\textsuperscript{7}, it has been demonstrated that prolonged immobilization of the TMJ produced degenerative changes on the articular surface with development of fibrous adhesions\textsuperscript{8,9}.

Our case showed similar symptoms and similar MRI and CT features, but it's not possible to offer any hypothesis to explain the pathogenesis. In all the diseases presented, the most common feature is a bilateral muscle involvement. The adipose replacement of the masticatory muscles was, in our case, unilateral and could not be associated to any known etiological factor: both CT and MRI scans did not demonstrate any neurological alterations.

The case presented highlighted the need for a multidisciplinary approach and the importance of a correct differential diagnosis, based on clinical observation but also on CT and MRI which represent, nowadays, an essential instruments for the evaluation of TMJ abnormalities in patients with long-standing limitations of mouth-opening.

REFERENCES


