Clinical vignette

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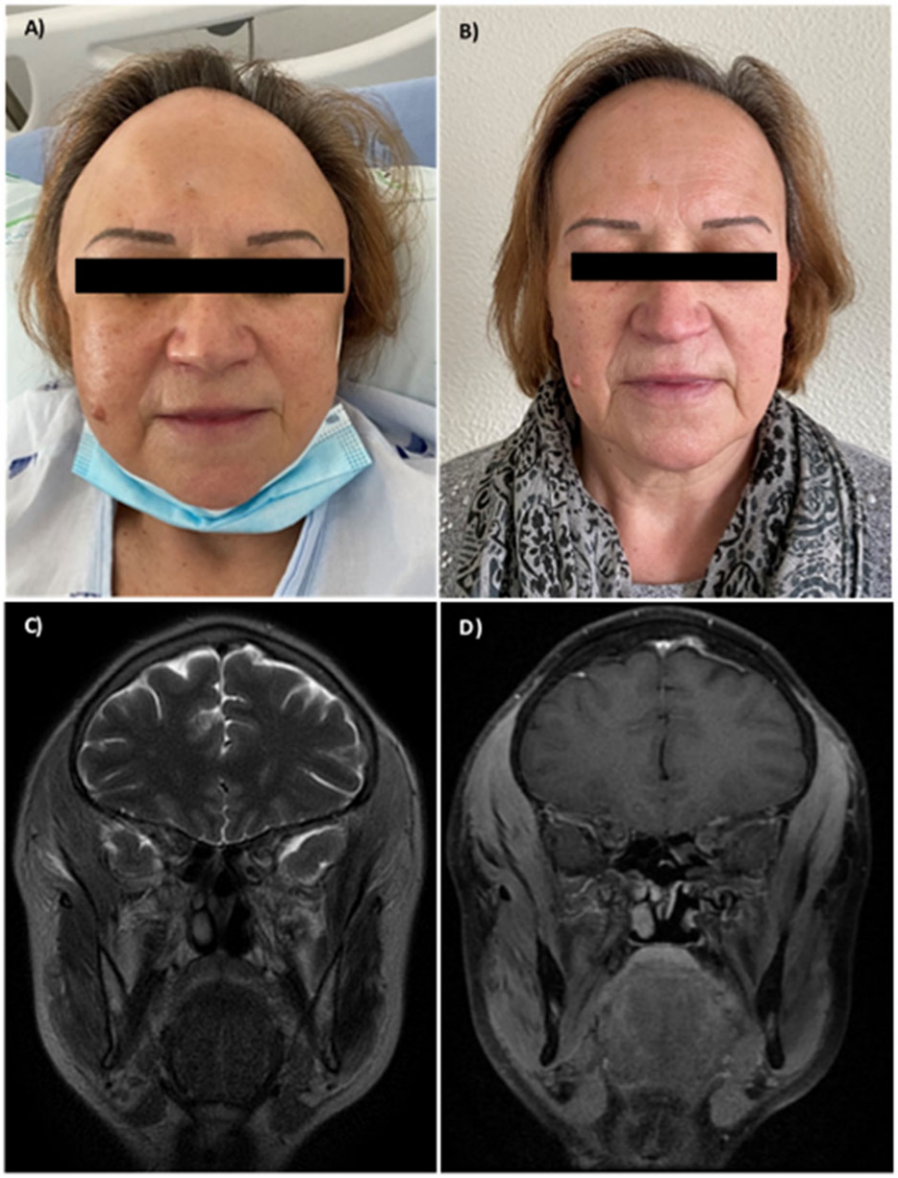
An atypical case of focal myositis

A 64-year-old woman presented with a 10-day history of painful progressive bilateral temporal and right retroauricular region tumefaction (Fig. 1A), dysphonia and a body temperature of 37.5°C. She had been previously prescribed with antibiotics assuming an oral infection, with no benefit. She had no other complaints and her past medical history and clinical exam was otherwise unremarkable. Her labs revealed high levels of CRP (11.7 mg/dl), ESR (52 mm/h), creatine kinase (CK 623 U/l), myoglobin (83 U/l), aspartate transaminase (56 U/l) and alanine transaminase (69 U/l). Serologies were negative for *Treponema pallidum*, human immunodeficiency,

Fig. 1 Images of the patient before (**A**) and after (**B**) treatment with prednisolone and facial MRI: T2-weighted MRI (**C**) and fat-saturated T1-weighted MRI with gadolinium (**D**)



hepatitis B and C, and Epstein–Barr viruses; blood cultures and immunological study, including anti-nuclear antibodies and antibodies associated with inflammatory myopathies, were negative. A facial MRI revealed thickening and T2/FLAIR hypersignal of temporal, masseter and pterygoids muscles with signal intensification after gadolinium injection, suggesting myositis (Fig. 1C and D). A temporal muscle biopsy showed marked lymphocyte infiltration (predominantly lymphocyte T CD3), as well as marking of multiple fibres with major histocompatibility complex class I products, which supported the diagnosis. Prednisolone 0.5 mg/kg/day (30 mg) was started, with marked clinical improvement (Fig. 1B) and normalization of CRP, ESR and CK.

Focal myositis typically is localized to a single skeletal muscle, commonly in the lower limbs; however, we present a case of focal myositis involving the masticatory muscles, rarely documented [1]. A distinct embryological origin has been recognized for striated craniofacial and limb muscles [2], raising the hypothesis that a different disease mechanism may be involved in masticatory muscle myositis.

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Data availability statement

Data are available upon reasonable request by any qualified researchers who engage in rigorous, independent scientific research, and will be provided following review and approval of a research proposal and Statistical Analysis Plan (SAP) and execution of a Data Sharing Agreement (DSA). All data relevant to the study are included in the article.

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References

1 Gallay L, Hot A, Petiot P *et al.* Focal myositis: new insights on diagnosis and pathology. Neurology 2018;90: e1013–20.

2 Yu F, Stål P, Thornell LE, Larsson L. Human single masseter muscle fibers contain unique combinations of myosin and myosin binding protein C isoforms. J Muscle Res Cell Motil 2002;23:317–26.