Cervical spinal calcinosis in systemic sclerosis presenting with foraminal stenosis, radiculopathy and vertebral artery displacement

Sirs,

We have read with great interest the recent letter published by Capparelli *et al*. describing the use of filgotinib for cervical spine calcinosis with peri-odontoid inflammatory changes in diffuse cutaneous systemic sclerosis (DcSSc) (1). Calcinosis affects nearly one third of SSc patients and leads to a significant morbidity (2). Their report illustrated an exceptionally rare and clinically severe manifestation of calcinosis; axial involvement. While distal extremities are the most common sites, involvement of the axial skeleton remains exceptional and is increasingly recognised as a potential source of pain, functional impairment, and even neurological or vascular compromise (3-5). In this context, we would like to contribute with a complementary case that further broadens the clinical spectrum of this entity.

We report the case of a 48-year-old woman diagnosed with DcSSc in May 2017. In addition to the skin thickening and Raynaud's phenomenon, our patient also presented with non-specific interstitial pneumonia (NSIP) at the time of diagnosis. Autoimmunity tests showed positive antinuclear antibodies at a 1/1280 titre, with positive anti-Ro and anti-topoisomerase-1 antibodies. She was started on mycophenolate mofetil and nifedipine, with no evident disease progression during the following year.

In November 2018, she developed progressive cervical pain of new onset, predominantly nocturnal and exacerbated by movement, accompanied by increasing limitation of left shoulder abduction. On examination, discrete proximal weakness of the left arm was noted, raising the differential diagnosis between a Parsonage-Turner syndrome (idiopathic brachial neuritis) and a plexoradiculopathy of mechanical or inflammatory origin. Initial investigations, including shoulder ultrasonography and routine laboratory testing, showed no evidence of structural abnormalities or elevation of acute-phase reactants.

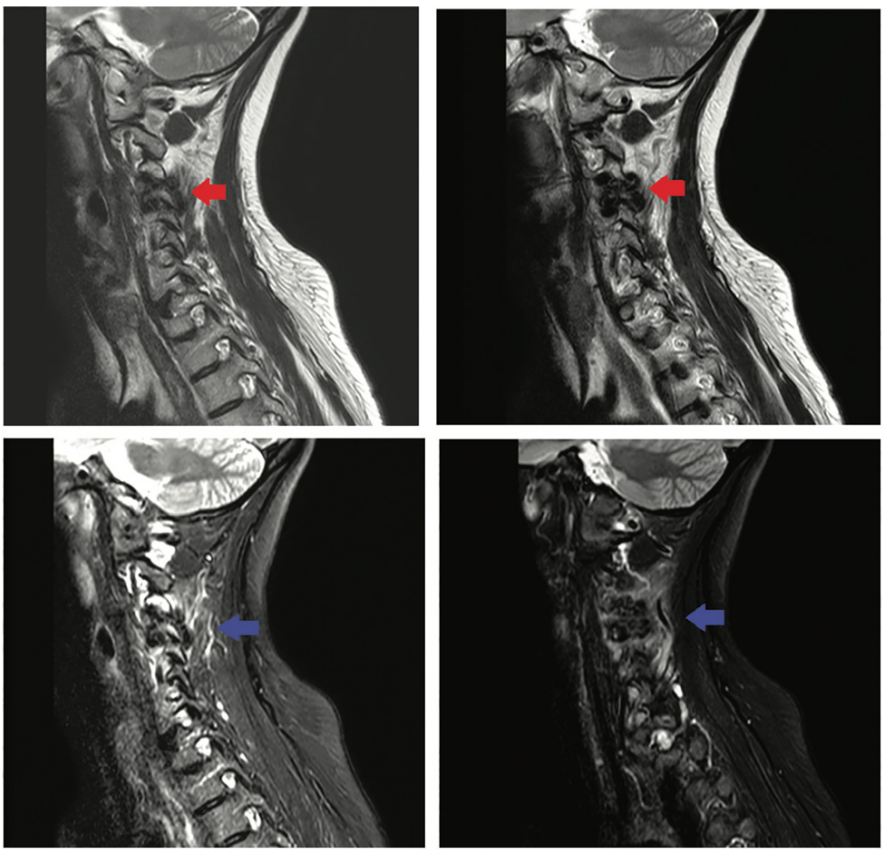


Fig. 1. Magnetic resonance imaging (MRI) of the cervical spine. Red arrows indicate the calcinosis lesions on the T2-weighted sequence and blue arrows in the STIR sequence.

Nuclear magnetic resonance imaging revealed severe left C3/4 and C4/5 foraminal stenosis due to nodular calcific deposits involving the facet joints, associated with erosive changes and marked inflammatory signal. The deposits extended to the transverse processes and laminae, encasing the articular facets and compressing the exiting nerve roots. Importantly, they also reached the transverse foramina, where they contacted and displaced the left vertebral artery, which appeared reduced in calibre compared with the contralateral side (Fig. 1). Computed tomography confirmed the nodular calcific morphology, compatible with hydroxyapatite or carbonate apatite deposition in the context of SSc calcinosis (Fig. 2).

The patient received a single dose of intravenous methylprednisolone (100 mg) together with colchicine 1mg per day. She achieved notable clinical improvement, with complete strength recovery and progressive pain mitigation, without recurrence of cervicobrachial symptoms up to this day. Since July 2021, she is on periodic rituximab due to progression of her lung involve-



Fig. 2. Computed tomography (CT) scan of the cervical spine. Yellow arrows indicate calcinosis deposits extending along paravertebral soft tissues and invading the cervical canal.

ment, and no further axial calcinosis flares have occurred.

Our case differs from that reported by Capparelli *et al*. (1) in several important aspects. Whereas their patient presented with peri-odontoid calcinosis complicated by erosive inflammatory changes and responded to filgotinib, our patient developed a facet-driven foraminal phenotype with radiculopathy and, notably, vertebral artery displacement. Axial calcinosis in SSc has been described only in isolated reports involving the cervical (6, 7), thoracic (3), and lumbar spine (8), with presentations ranging from axial pain to radiculopathy and even central cord syndrome (6, 9). A review of 35 paravertebral cases since 1974 confirmed a female predominance and frequent cervical involvement, sometimes necessitating surgical decompression (3, 10). However, vascular compromise has rarely been emphasised, making our observation particularly relevant. Although our patient developed mild proximal weakness, the reduction in vertebral artery calibre fortunately did not result in neurological events.

Therapeutic approaches to spinal calcinosis in SSc remain largely empirical. Surgical excision is generally considered the most reliable intervention in advanced compressive disease, but it carries significant perioperative risk (9, 10). Pharmacological strategies have shown heterogeneous efficacy, and recent evidence points to the potential utility of JAK inhibitors in inflammatory phenotypes (1, 4, 11). Our case is noteworthy in this context: despite the absence of systemic inflammatory markers, a single corticosteroid pulse combined with colchicine, achieved complete and durable clinical remission, thereby avoiding surgical intervention. This outcome, in contrast to the peri-odontoid case successfully treated with filgotinib (1), suggests that the addition of short-course glucocorticoid and colchicine therapy may represent a pragmatic- anti-inflammatory, less invasive and more cost-effective therapeutic option in selected patients with facet-related, radicular and inflammatory spinal calcinosis. These findings emphasise the heterogeneity of this complication and highlight the need for systematic studies to better define predictors of therapeutic response and to guide personalised management strategies.

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