

Myositis as a manifestation of SARS-CoV-2

We read with great interest the paper from Monti *et al* describing clinical course of coronavirus disease 2019 (covid-19) in patients with chronic arthritis. We would like to emphasise that symptoms mimicking connective tissue disease can occur at the early phase of covid-19 infection.¹

Despite the fact that myalgia has been already reported in several cohorts of patients with covid-19 infection,² myositis was not described in these studies. We report a case of a MRI-documented myositis secondary to covid-19 in a patient. The patient was not under medication prior to the illness. Symptoms appeared suddenly on waking with diffuse myalgias and proximal lower limb muscle weakness, causing him to fall. On arrival at the hospital, the patient was afebrile and did not present any upper or lower airway symptoms. Motor testing revealed a bilateral hip flexion deficit graded at 3/5 on the Medical Research Council (MRC) muscle scale. Initial blood work-up revealed creatine kinase (CK) at 25 384 IU/L (n <195 IU/L), C reactive protein at 54 mg/L and a lymphocytopenia. Initial management consisted of administration of intravenous fluids.

On day 4 after the appearance of symptoms, the patient presented with fever at 39°C. Blood and urine cultures were negative, and nasopharyngeal swab multiplex PCR for respiratory viruses, not including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was also negative. A chest CT scan on day 5 showed bilateral lower lobe ground-glass opacities. On day 7, the patient desaturated and required oxygen at 1L/min, and a SARS-CoV-2 nasopharyngeal swab was negative. A proximal lower limb MRI showed bilateral external obturator muscle and quadriceps oedema (figure 1), compatible with bilateral myositis. Specific overlap myositis, dermatomyositis, immune-mediated necrotising myositis and antinuclear antibody testing were negative. On day 10, the patient's respiratory status worsened and a ventral chest CT scan showed worsening of bilateral ground-glass opacities. A second specific SARS-CoV-2 nasopharyngeal swab still remained negative. The patient was transferred to the intensive care unit on day 11, where bronchoalveolar lavage fluid was finally positive for SARS-CoV-2. The patient still remains in critical condition.

The prevalence of myalgia varies between 11% and 50% in different studies²⁻⁵ and muscle weakness related to covid-19 has been reported; however to our knowledge, this is the first MRI documentation of such myositis.

In the Guan *et al* study, two patients had rhabdomyolysis (0.2%) and the CK levels were elevated in 13.7% patients.³ One study showed statistical association between elevated CK levels and mortality.⁶ As observed in autoimmune myositis, an association between myositis and myocarditis could explain this excess in

mortality. Indeed, some studies reported elevation of N-terminal pro-brain natriuretic peptide (NT-pro-BNP) and troponin.⁷

In our patient, the subsequent association of myositis followed by interstitial pneumonitis led to the hypothesis of autoimmune myositis but all the immunological tests looking for any forms of myositis were negative.

In conclusion, covid-19 manifestations, although frequently limited to upper and lower airways, can, as shown in our case, reveal itself by acute myositis. Since the association of muscle inflammation with interstitial pneumonia can be seen in either covid-19 or autoimmune myositis, this differential diagnosis should be known by clinicians.

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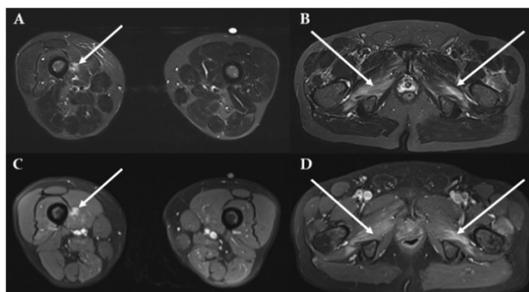


Figure 1 Pelvic and thigh MRI. (A) Thigh MRI in T2 STIR sequence showing oedema of the right vastus medialis (arrow). (B) Pelvic MRI in T2 STIR (short TI inversion recovery) sequence showing bilateral oedema of external obturator muscles (arrows). (C and D) T1 sequences revealing enhancement of muscle lesions after gadolinium infusion (arrows).