

Letter to the Editor

Diagnosing SARS-CoV-2 associated myositis should not rely on the profile of myositis-specific antibodies alone

Dear Editor,

With interest we read the article by Sacchi et al¹ about a 77yo female with moderate COVID-19 disease who could not be weaned from continuous positive airway pressure (CPAP) ventilation after 15 days, being attributed to myositis of the respiratory muscles. Myositis was diagnosed upon the clinical presentation (respiratory insufficiency) and positivity for anti-nuclear antibodies (ANA) and anti-cytoplasmic antibodies (ANCA)¹. The patient received steroids but experienced diffuse muscle weakness and weight loss¹. We have the following comments and concerns.

The main shortcoming of the study is that myositis was diagnosed solely upon the clinical presentation and the antibody profile. Diagnosing myositis requires not only clinical assessment and determination of myositis-specific antibodies (anti-MDA5, NXP2, anti-Ku, anti-MI2 β etc.), but also needle electromyography (EMG), muscle MRI with contrast medium, and muscle biopsy. Diagnosing myositis upon the antibody profile can be misleading, as several of these antibodies can be non-specifically elevated in diseases other than myositis as well. Anti-MDA5 antibodies have been associated with interstitial lung disease² or neuromyelitis optica³. Anti-Ku antibodies have been associated with lupus erythematosus⁴.

Another shortcoming is that the clinical presentation on admission and during hospitalisation is not well described. Failure of weaning from CPAP can be multifactorial and not necessarily due to isolated myositis of the respiratory muscles. Since the patient complained about diffuse muscle weakness 15d after admission, we should be informed if muscle weakness on admission included only the respiratory muscles or extra-ocular, axial, bulbar, facial, and limb muscles as well. We should know the results of the clinical neurologic exam on admission and at hospital day 15.

The patient received chloroquine, which is ineffective for the SARS-CoV-2 infection but may exhibit various side effects. We should know for how long chloroquine was given, in which dosage, and if muscle weakness could be due to muscular side effects of chloroquine. From chloroquine it is well appreciated that it can be myotoxic and can cause permanent myopathy⁵.

Since SARS-CoV-2 may go along with affection of the central nervous system (CNS) we should be informed about the results of cerebral imaging, particularly if viral encephalitis, immune encephalitis, cerebral vasculitis, acute, hemorrhagic, necrotising encephalopathy (AHNE), and acute disseminated encephalomyelitis (ADEM) were excluded as causes of central respiratory insufficiency.

Elevated ANA not only suggest myositis but also vasculitis. We should know if cerebral vasculitis, previously reported as a manifestation of a SARS-CoV-2 infection⁶, was excluded by cerebral MRI with contrast medium and cerebral MRA.

It is unclear if the patient was a female or a male. In the first sentence of the case report the patient is a female but the first sentence of paragraph 4 starts with "he".

Overall, the interesting report has a number of limitations, which should be met before diagnosing myositis. SARS-CoV-2 associated myositis should not be diagnosed upon the profile of specific myositis antibodies alone.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Authors' Contribution

JF: design, literature search, discussion, first draft, critical comments.

Informed Consent

The informed consent was obtained.

Approval

The study was approved by the institutional review board.

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