Dear Editor, Coronavirus disease 2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Clusters were first identified in December 2019 in Wuhan, China; subsequently, rapid spread has resulted in an ongoing pandemic. We present the case of a 72-year-old man who attended the Emergency Department during the first peak of the UK epidemic with a 3-week history of unexplained flu-like symptoms.

The patient reported fever, non-drenching sweats, dry cough, arthralgia, myalgia, bilateral pleuritic chest pains, shortness of breath and unremitting fatigue. His past medical history comprised mild chronic obstructive pulmonary disease, OA and hyperlipidaemia, for which he was taking Simvastatin, Losartan, Omeprazole, a combination inhaler containing fluticasone furoate/umeclidinium/vilanterol, diclofenac 1.16% gel and co-codamol 15/500. He denied any weight loss. He was an ex-smoker. Initial observations demonstrated peripheral oxygen saturation 89% in room air, respiratory rate 36 breaths/min, blood pressure 112/73 mmHg, heart rate 121 beats per min and temperature 38.0°C. No further fevers were documented throughout his admission. Skin and mucous membranes were unremarkable. Chest radiograph showed bilateral, mainly left lower lobe, areas of opacification, as well as minor right and mild left pleural effusions, the findings of which were thought to represent infection. The patient was treated with ward-based oxygen, i.v. antibiotics and diuretics, later followed by prednisolone 30mg daily; however, he failed to improve and developed worsening digital vasculitis with necrotic fingertips and respiratory compromise (Fig. 1A and B). High-resolution CT depicted bilateral subpleural consolidation and developing fibrosis within the areas of consolidation, which raised the possibility of organizing pneumonia (Fig. 1C).

On day 8, the patient underwent a trans-oesophageal echocardiogram, which confirmed normal biventricular size and systolic function, no significant valve dysfunction and no echocardiographic features of endocarditis. Twelve days into admission, myositis-specific antibody screening confirmed the presence of anti-PL-7 antibodies. Consequently, the patient received urgent treatment with i.v. methylprednisolone and CYC 10 mg/kg (2-weekly for first three, then 3-weekly for six), in conjunction with mesna, co-trimoxazole and nystatin. He was also given aspirin 75 mg daily, HCQ 200 mg daily, prednisolone reducing regime and sildenafil 25 mg three times daily. After 3 months, the patient had made a rapid recovery, with no further symptoms, and repeat high-resolution CT showed near-complete resolution of earlier changes.

Myalgia, fatigue and weakness are frequently encountered with viral pathogens, not least with the coronavirus family. The Lancet published a report of 41 patients hospitalized with pneumonia: 33% of them showed CK elevation and that number increased up to 46% in intensive care unit patients [1]. Antisynthetase syndrome (ASS) is a rare autoimmune disease characterized by interstitial lung disease (ILD) and/or inflammatory myositis, with positive antisynthetase antibodies (anti-Jo-1, anti-PL-7, anti-PL-12, anti-ZO, anti-OJ, anti-KE or anti-KS).
Other symptoms described include arthritis, fever, RP and mechanic’s hands [2]. A large retrospective study revealed a high prevalence of ILD (100%) and relatively low prevalence of other symptoms, including myositis (50%), findings of which corroborated previous anti-PL-7 series [3].

In conclusion, it is imperative when considering flu-like or multisystem presentations in the current climate, to be aware of one’s cognitive biases and maintain a sense of skepticism, so that important diagnoses do not go amiss. Emerging reports highlight missed or delayed treatment for infections such as *Pneumocystis jiroveci* pneumonia that imitate COVID-19; however, there is little reference in the literature to autoimmune mimics of COVID-19 [4–6]. ASS is a rare clinical entity that is, even at the best of times, difficult to recognize; diagnosis requires a thorough multidisciplinary approach, synthesizing rheumatology and pulmonary assessments, along with serology, radiology, and occasionally muscle and/or lung biopsy results. It shows high heterogeneity between patients with respect to clinical phenotype and disease severity. Moreover, there is often a variable temporal relationship between the onset of ILD and that of myositis or other ASS-specific features. Early diagnosis followed by immunosuppressive therapy can significantly increase both the quality of life and life expectancy of patients, largely by reversibility of ILD [2]. Patients with anti-PL-7 or anti-PL-12 often have severe ILD, frequently without myositis. A meta-analysis of 27 studies on ASS, found that arthralgia and ILD predominate in ASS, as compared with the other idiopathic inflammatory myopathies, and patients with Jo-1 have more myositis and a better prognosis compared with patients with PL-7 or PL-12 [7].

Our case highlights the importance of a thorough multisystem assessment of patients presenting with COVID-19-like features and raised creatine kinase, and greater awareness of the clinical features of ASS in acute settings, in order that earlier recognition and appropriate treatment may improve outcomes. One should consider, according to local availability, formal nailfold videocapillaroscopy and a Myositis Panel for the detection of myositis antibodies in the diagnostic work-up.

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