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COVID-19 as potential aggravating factor for the natural course of new onset-dermatomyositis

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SUMMARY

Borges et al. have recently reported the first case of a dermatomyositis onset in close association with established coronavirus disease 2019 (COVID-19). Similarly, we report a patient who, on the contrary, had COVID-19 following early established dermatomyositis. We report prospectively the outcome of her disease.

Key words: *COVID-19, dermatomyositis, inflammatory myopathies, myositis, SARS-CoV-2.*

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CASE REPORT

A 59-year-old woman developed insidious and predominantly proximal muscle weakness in her four limbs and dysphagia. Three weeks later, she experienced cough, fever, and shortness of breath. Her oronasopharyngeal swab tested positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection by real-time polymerase chain reaction test (RT-PCR). Thoracic computer tomography images showed mild ground-glass opacities with a peripheral distribution. When she was admitted to our unit, she did not need orotracheal intubation, but progressed with worsening dysphagia and muscle weakness (upper limbs: proximal grade I and distal grade III; lower limbs: proximal and distal grade II). The patient also presented with heliotrope rash (Figure 1A), Gottron's sign with ulcerations (Figure 1B), cuticular hypertrophy (Figure 1C) since disease onset. Laboratory tests showed 231 U/L of aminotransferase aspartate, 127 U/L of aminotransferase alanine, 4812 U/L of creatine phosphokinase, 1131 U/L of lactic dehydrogenase; positivity for anti-Mi-2 and anti-SAE autoantibodies and negativity for anti-MDA-5, anti-Jo-1, anti-PL-7, anti-PL-12, anti-OJ, anti-EJ, anti-SRP anti-Ro52, anti-PM/Scl and anti-Ku antibodies (Myositis Profile Euroline blot test kit, Euroimmun, Lübeck, Germany) were seen as well as positive antinuclear antibodies with a homogenous nuclear pattern (1/640), and normal blood complement levels. Thigh muscle magnetic resonance showed intense muscle inflammation (Figure 1D) and absence of fat replacement. There was an intense area of myofibrillar necrosis with macrophage infiltrates in the vastus lateralis muscle biopsy (Figure 1E and F). Neoplastic and infectious causes different from COVID-19 were excluded. The dermatomyositis diagnosis was established, according to EULAR/ACR 2017 criteria for the classification of idiopathic inflammatory myopathies (1), and the patient received three monthly pulses of methylprednisolone (3 g) and two monthly cycles of intravenous immunoglobulin (2 g/kg/day), in addition to methotrexate (maximum dose of 25 mg/week) and prednisone (1 mg/kg/day). After three months, there was a significant improvement in the patient's clinical and laboratory status. The patient improved proximal grade IV strength, and distal grade V strength in both limbs and serum concentrations of muscle enzymes were already within the normal range. During follow-up, the patient had intolerance to methotrexate (alopecia), which was re-

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placed with azathioprine (2.5 mg/kg/day); she was already taking prednisone at a dose of 10 mg/day and was following a tapering scheme.

DISCUSSION

We report a case of definite dermatomyositis with coronavirus disease 2019 (COVID-19). Like in the case report by Borges et al. (2), our patient had typical dermatomyositis symptoms with positive specific-myositis autoantibodies. However, unlike them, we were able to document the additional dermatomyositis features through muscle magnetic resonance and muscle biopsy. We also present the patient's disease outcome.

Our hypothesis is that the insidious evolution of dermatomyositis could have been modified by the overlap with SARS-CoV-2 infection, which may have accelerated dermatomyositis onset and exacerbated its symptoms, leading for instance to a severe myopathic condition like in an immune-mediated necrotizing myopathy. Previous studies corroborating this hypothesis have already shown that the symptoms of autoimmune rheumatic diseases can be exacerbated or even evolve with other manifestations during COVID-19 infection (3, 4). Moreover, Lokineni et al. (5) described the first case of an immune-mediated necrotizing myopathy caused by COVID-19.

We believe that the histopathological finding compatible with necrotizing myopathy in our case report is associated with the viral infection. Due to the severity of the clinical onset, our patient received pulse therapy with methylprednisolone and intravenous immunoglobulin. This is a therapeutic scheme used and recommended in newly diagnosed cases of patients with systemic autoimmune myopathies, especially in cases of immune-mediated necrotizing myopathy (6, 7). In this study, the patient improved with prompt remission of the disease, even allowing for early weaning from oral corticosteroid therapy, thus minimizing the unwanted effects of using this medication in the long term.

In conclusion, the possibility of potentially more severe clinical cases with atypical manifestations in patients with systemic autoimmune myopathies infected with SARS-CoV-2 infection must be considered.

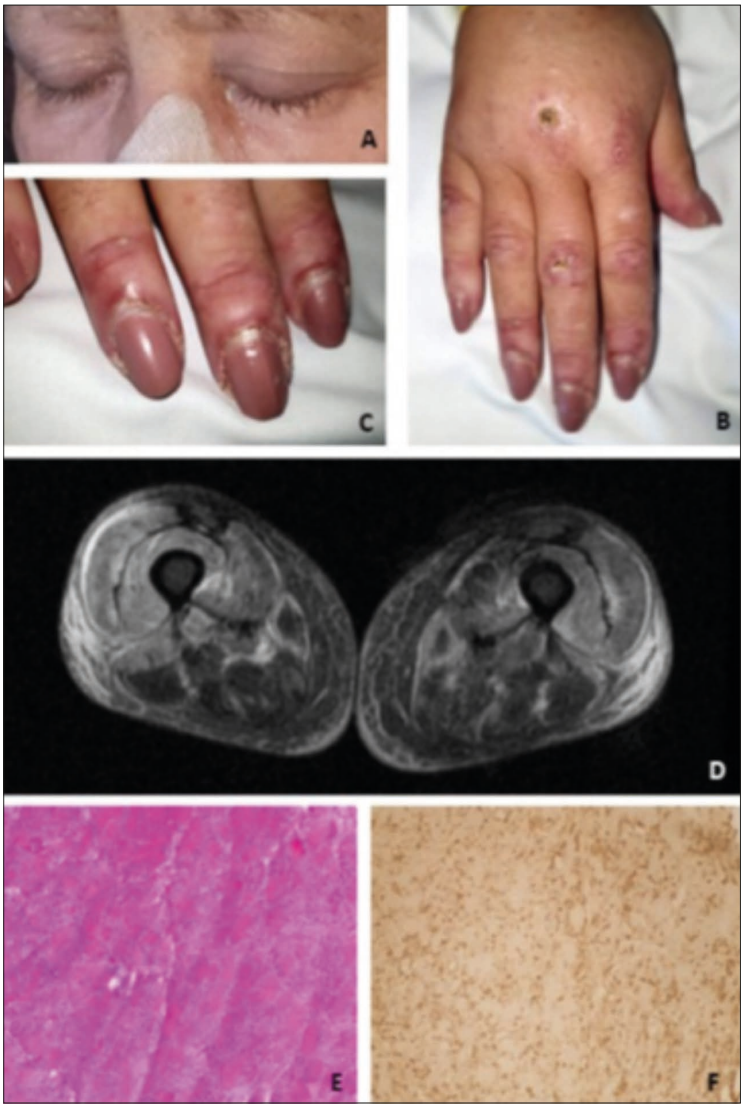

Figure 1 - General features of the patient with dermatomyositis. A) Heliotrope rash; B) Gottron's sign with ulcerations; C) Cuticular hypertrophy; D) Thigh muscle magnetic resonance (STIR sequence) showing intense muscle inflammation; E) Muscle biopsy with an intense area of myofibrillar necrosis (hematoxylin and eosin stain), 10x; F) Muscle biopsy with macrophage infiltrates (immunohistochemical reaction - CD68), 10x.

Conflict of interest

The authors declare that they have no conflict of interest.

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