Abstract

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A case report of COVID-19 inflammatory myositis: the role of Autoimmunity

Coppola MG*[1], Vitelli N [2], Landi G [2], D Errico T [3], Lugara M [1], Bologna C [1], Tirelli P [1], De Luca C [1], Guida A[1], De Sena A [1], Madonna P [1]

[1]Internal Medicine Unit, Ospedale Del Mare, ASL Napoli 1 Centro, Italy

[2]Postgraduate Specialization of Internal Medicine, Federico II University, Naples, Italy

[3]Rheumatology Clinic, PSI Napoli Est and Ospedale Del Mare, ASL Napoli 1 Centro, Italy

Background: During SARS-Cov2 pandemic, several evidences suggested a close relationship between virus infection and autoimmune disease, probably mediated by so-called cytokines storm. More than 10% of COVID-19 patients were reported to have musculoskeletal manifestations ranging from an asymptomatic elevation of Creatine kinase (CK) to severe rhabdomyolysis. Lasting effects are observed in some individuals at 6 months or even longer after recovery.

Case report: A 68 years-old-female presented to our hospital for chest pain, asthenia, myalgia and proximal weakness of lower and upper limbs. In the previous month the patient had been hospitalized for COVID-19 pneumonia treated with cortisone therapy and oxygen therapy. Her medical history included arterial hypertension, dyslipidaemia and hiatal hernia. She had used atorvastatin until a few months ago, but she had stopped due to muscle disturbances due to activity, nocturnal cramps and tendon soreness. Blood tests evidenced elevation of muscular enzymes (CK 2825 U/L n.v. 26-167, myoglobin 1601 ng/dl v.n. 21-72), mild hypertransaminasemia (GOT 89 U/L n.v. 4-32, GPT 104 U/L n.v. 4-33), increased cardiac necrosis (CK-MB 97,4 ng/ml n.v. 0,5, trop T 0,508 ng/ml n.v. 0,003-0,014) and inflammatory markers (VES 32 mm n.v.<12, CRP 1,37 n.v.<0.5). Normal findings at electrocardiogram, echocardiogram and cardiac CT excluded acute coronary syndrome. Chest computed tomography (CT) scan showed pulmonary ground glass opacity, consistent with recent COVID-19 pneumonia. Abdomen CT scan was negative. A mild muscle damage was present at electromyography .Infectious workup was negative for viral etiologies including viruses hepatitis B, C, cytomegalovirus, Epstein-Barr. The thyroid and renal function were normal. Autoimmune serology was positive for antinuclear antibody (ANA) (1:320, specked pattern). Complement levels were mildly elevated. The extractable nuclear antigen (ENA) and ant phospholipid antibodies were negative. She was started on intravenous fluid but despite this, her CK up-trended. The patient had refused the muscle biopsy. She was treated empirically for autoimmune myositis with 100 mg of prednisone daily. CK gradually down trended and prednisone was tapered to 50 mg daily. About 2 months after the start of steroids CK values were normal and the patient was asymptomatic for myalgia. Actually she is in the follow-up phase and the steroid therapy is gradually tapered off.

Discussion: In our case report all the possible causes that could trigger myositis like acute viral infections, endocrinopathies, Para neoplastic and statin were ruled out. The improvement of symptoms with high doses of corticosteroids can suggest an autoimmune etiology of myositis. COVID-19 induced myositis can present different manifestations in presentation, ranging from muscle weakness to the typical dermatomyositis filled with classic skin rashes. The pathophysiology of myositis in SARS-CoV-2 infection is not fully understood. SARS-COV 2 can cause muscle injury through direct mechanisms due to the expression of ACE2 receptors on skeletal muscles or indirect mechanisms due to the deposition of immune complexes in the muscles and the release of myotoxic cytokines. The full relationship between autoimmune phenomena and SARSCoV-2 infection is still unknown.