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Letter to Editor

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Novel coronavirus infection and rhabdomyolysis

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Dear editor,

First novel coronavirus infection was seen in Wuhan, China at the end of 2019, as pneumonia cases of unknown etiology. This new type of virus was defined as a new type of coronavirus (2019-nCoV) that has not been seen in humans before on January 7, 2020 (1). Later, the name of the 2019-nCoV disease was named as Coronavirus Disease-2019 (COVID-19), and the virus was named as SARS-CoV-2 due to its close resemblance to the Coronavirus (SARS CoV) related to Severe Acute Respiratory Syndrome (2). The first detected case of the COVID-19 epidemic in Turkey, which has spread around the world, was announced by the Ministry of Health on 11 March. The first death due to the virus in Turkey took place on March 15, 2020 (3).

Rhabdomyolysis is the release of toxic muscle contents into the circulation because of damage to striated muscles due to traumatic or non-traumatic causes and destruction of muscle tissue. Rhabdomyolysis was first described in the 1940s in patients exposed to trauma in the form of crushing under destroyed houses during the war. Toxic substances can cause crush syndrome and acute renal failure, which is one of the most important clinical problems related to this condition (4). Rhabdomyolysis is a clinical and biochemical syndrome caused by acute necrosis of skeletal muscle fibers and leakage of cellular contents into the circulation. In general, the most common causes of rhabdomyolysis include alcohol and substance use, drugs, muscle diseases, trauma, disasters, neuroleptic malignant syndrome, seizures, immobility, infection, heavy physical activity, myositis, and heat-related diseases (5). The blood creatine kinase (CK) value is a significant test for the diagnosis of rhabdomyolysis. Serum CK levels begin to increase 2-12 hours after kidney injury, peak in 1-3 days, and then decrease within 3-5 days. A five-fold increase in serum CK is sufficient for the diagnosis of rhabdomyolysis (6).

Influenza A virus subtype is associated with several viruses, including H1N1 and SARS-CoV-1. It has been reported in the literature that some patients with severe acute respiratory syndrome or H1N1 infection show mild to moderate increases in serum CK levels (7). Similarly, in many case reports in the literature, a case of SARS-CoV-2 associated rhabdomyolysis has been reported (8). Haroun et al. showed that rhabdomyolysis is associated with increased mortality in patients with COVID-19 (9). Gang et al. reported that rhabdomyolysis was associated with in-hospital mortality in their study in hospitalized patients (10). The mechanism of muscle damage in viral infections, especially SARS-CoV-2, is not fully understood. Possible explanations suggested including direct and indirect mechanisms. The most important possible explanation is that the virus directly causes muscle damage. The presence of angiotensin converting enzyme 2, which is defined as the functional receptor for SARS-CoV and SARS-CoV-2, in skeletal muscles is one of the data supporting this theory in the literature (11). However, in postmortem examinations SARS-CoV was not detectable in skeletal muscle, and it is therefore unclear whether SARS-CoV-2 directly infects muscle (12) The second plausible explanation postulates that the cytokine storm-like immune response may lead to skeletal muscle damage (13). The muscle pain seen in patients during viremia and the accompanying high CK levels support the first explanation. Late rhabdomyolysis seen in hospitalized patients support the second explanation.

As a result, rhabdomyolysis can be seen in SARS-CoV-2 infected patients and the mechanism is not clear. Researchers should do new research on this subject and the mechanism should be clearly revealed.

Conflict of interest
None to declare.

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