

Rhabdomyolysis as Potential Late Complication Associated with COVID-19

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To the Editor: We provide follow-up information on a case discussed in Emerging Infectious Diseases of a man with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection who reportedly had late-onset rhabdomyolysis with lower limb pain and fatigue (1). After the patient was stabilized, he was transferred to Wuhan Union Hospital with medical support by Fujian Provincial Hospital, where he disclosed symmetric weakness (Medical Research Council grade 4/5) in both lower limbs with weakened deep tendon reflexes and decreased sensation to light touch and pinprick distally. Because weakness and paresthesia persisted after biochemistries normalized, we feel that these observations are not explained solely by rhabdomyolysis.

The patient was discharged 43 days after admission and was able to walk normally but with reduced endurance. Electromyography (day 120) showed motor and sensory fiber involvement in both lower extremities, presenting as axonal injury accompanied by demyelination (Tables 1, 2). Despite his 10-year history of diabetes, the patient reported no history of paresthesia or reduced motor endurance, which ruled

against preexisting diabetic neuropathy or myopathy. We believe he developed peripheral neuropathy during his COVID-19 illness, which may have been missed during the acute phase. We are unsure what caused this neuropathy. In addition to hematologic or lymphatic spread, coronaviruses may directly invade the peripheral nerve terminals and interfere with subsequent synaptic transfer (2). Indirect causes, such as cytokine-mediated damage, should also be considered in this patient (3). Finally, thromboembolism has the potential to cause peripheral nerve ischemia and necrosis (4). However, the coagulation indices, including fibrinogen (7.95 g/L, reference 2–4 g/L), D-dimer (>20 mg/L, reference <0.5mg/L), and fibrinogen degradation products (80 µg/mL, reference <5 µg/mL), were at the highest level at the onset of rhabdomyolysis and gradually decreased with enoxaparin treatment. We cannot offer a definitive diagnosis and were limited by the lack of muscle biopsies and complete electromyography; however, several factors may have caused his peripheral neuropathy.

About the Authors

Dr. He is a neurologist and Dr. Chen is an emergency physician in Fujian Provincial Hospital, Fujian, China. During the COVID-19 outbreak, they worked as a support medical team in the isolation ward of Union Hospital, Tongji Medical College, Wuhan, China.

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Table 1. Motor nerve conduction studies on a patient with rhabdomyolysis after severe acute respiratory syndrome coronavirus 2 infection, China

Location	Distal latency, ms	Amplitude, mV	Conduction velocity, m/s	F latency, ms
Left tibial nerve				
Ankle-abductor hallucis brevis	6.5 (reference ≤5.1)	0.825 (reference ≥4)	38 (reference ≥40)	51.4 (reference ≤56)
Popliteal fossa	15.4	0.755		
Right tibial nerve				
Ankle-abductor hallucis brevis	6.3 (reference ≤5.1)	5.4 (reference ≥4)	39 (reference ≥40)	49.4 (reference ≤56)
Popliteal fossa	15.0	4.46		
Left peroneal nerve				
Ankle-extensor digitorum brevis	5.1 (reference ≤5.5)	1.061 (reference ≥2)	35 (reference ≥42)	Not tested
Below fibula	11.6	1.022		
Right peroneal nerve				
Ankle-extensor digitorum brevis	3.8 (reference ≤5.5)	1.947 (reference ≥2)	33 (reference ≥42)	Not tested
Below fibula	10.7	1.328		

Table 2. Antidromic sensory nerve conduction studies on a patient with rhabdomyolysis after severe acute respiratory syndrome coronavirus 2 infection, China

Location	Amplitude, µV	Conduction velocity, m/s
Left superficial fibular nerve: lateral calf–lateral ankle	Absent	Absent
Right superficial fibular nerve: lateral calf–lateral ankle	5.879 (reference ≥6)	37 (reference ≥40)
Left sural nerve: calf–posterior ankle	Absent	Absent
Right sural nerve: calf–posterior ankle	4.225 (reference ≥6)	37 (reference ≥40)

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COVID-19 Outbreak Associated with Air Conditioning in Restaurant, Guangzhou, China, 2020

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To the Editor: Lu et al. (1) describe the indoor airborne spread of COVID-19 (coronavirus disease) facilitated by a type of standard, wall-mounted, ductless air conditioner (AC) used in most countries. These units are low-cost in comparison to ducted AC units, which can cost 3 times as much to purchase, install, and operate. Ductless units cool and dehumidify indoor air but have little ability to filter or remove airborne contaminants.

A wall-mounted ductless system blows air directly onto those closest to it, potentially disseminating infectious droplets or aerosols along the airflow. Lu et al. use arrows to point out the airflows emanating from and returning to the AC unit, delineating a possible trajectory of putative airborne droplets.

This trajectory coincides with the seating distribution of other persons at the restaurant who later became ill (1). We agree that the AC probably contributed to the upstream and downstream airborne spread of the virus.

The type of AC system required to mitigate airborne transmission is neither affordable nor architecturally feasible for many buildings or regions. To prevent the spread of coronavirus disease in indoor spaces, we need work-around solutions in addition to distancing and fresh air exchange. Viable, low-cost possibilities might include operating AC on low fan settings and installing units near the ceiling, which would channel airflow towards the ceiling instead of directly onto patrons. Other methods might include installing high-efficiency particulate air filters, ultraviolet germicidal irradiation (which can disinfect some airborne coronaviruses such as mouse hepatitis virus and Middle Eastern respiratory syndrome coronavirus) (2), or a combination of these methods.

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Nonpharmaceutical Measures for Pandemic Influenza in Nonhealthcare Settings—International Travel-Related Measures

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