Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Paraparesis due to aortic thromboembolism as a rare complication of COVID-19 infection

In the context of the coronavirus disease 2019 (COVID-19) pandemic, a recent report by Naudin et al[1] sought to explain arterial thrombosis as a presentation of COVID-19 infection. Their patient was admitted with acute monoparesis and sensory loss of the left lower limb as a consequence of aortoiliac thrombosis and limb ischemia during COVID-19 infection.[1] However, we want to emphasize that, in addition to limb ischemia, spinal cord ischemia should be considered in patients with acute limb paresis and COVID-19 infection.

We read very interesting article from Naudin et al while we were finishing a case series including very similar cases about two male patients with paraparesis and COVID-19 infection. The first patient was admitted with sudden paraplegia, urinary retention, and a pain and temperature sensory level of T6, with intact proprioceptive senses. His cremasteric and bulbocavernosus reflexes were absent. Dorsalis pedis pulses were present. A full spinal cord magnetic resonance imaging was normal. The next day, signs of limb ischemia were developed. Color Doppler ultrasound examination and computed tomography angiography revealed an infrarenal abdominal aorta thrombosis, with extension to both common iliac arteries.

The second patient, with known multiple sclerosis, was admitted with urinary incontinence and acute paraparesis. In addition, he had diminished deep tendon reflexes of the lower limbs and hypoesthesia in the right leg. Distal pulses in the lower extremities were present. The next day, he developed signs of limb ischemia. Color Doppler ultrasound examination and computed tomography angiography revealed complete thrombosis of the infrarenal abdominal aorta with extension to both common iliac arteries.

The results of COVID-19 tests were positive for both patients. It is plausible that the paraparesis in both patients was a sign of spinal cord ischemia as a consequence of aortic thrombosis in the context of COVID-19 infection. Spinal cord ischemia is a rare disease with a prevalence of 1.2% of all strokes.[2] In patients with COVID-19, who are more vulnerable to thromboembolic events,[3] such rare conditions may occur more frequently. Therefore, in patients with any acute neurologic deficit, different ischemic disorders, including spinal cord ischemia, as a consequence of thrombotic complication of COVID-19 should be considered and evaluated.

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Acute paraplegia with or without coronavirus disease 2019 infection: Decision-making algorithm

We thank our colleagues for their letter. Acute paraplegia should always suggest two urgent diagnoses that require immediate specific treatment: acute aorta occlusion (AAO), favored by coagulation disorders in those with coronavirus disease 2019 (COVID-19) infection, and spinal cord compression, with COVID-19 as a coexisting condition.

AAO can be easy to diagnose in the presence of acute pain, pallor and coldness of the lower limbs, mottling, and the absence of femoral pulses. However, in most cases, the diagnosis will remain difficult, and the patients will often be referred to a neurologist or neurosurgeon. The specificity of pulse palpation is very low,[1] and the use of the ankle brachial index must be systematic. The use of pulsed wave Doppler ultrasound by well-trained emergency physicians could identify AAO; however, urgent computed tomography remains the reference standard.

AAO is a life-threatening event with mortality of near 30%. The main determinants of mortality are the interval until revascularization and the neurologic status of the extremities.[2] In the case of AAO, the neurologic symptoms can be caused by ischemia of the peripheral nerves or spinal cord ischemia. Almost all the blood supply to the sacral roots and plexus derives from the internal iliac arteries.[3] The physiopathology of spinal cord ischemia in those with AAO is not totally understood. The blood supply of the terminal cord can depend on the lower lumbar

or sacral radicular arteries, when the Adamkiewicz artery originates abnormally high or is chronically occluded.[3] The variable intensity of sensorimotor deficits might be explained by the variable collateralization of the spinal arterial supply.[4] However, urgent revascularization is always required for both causes. The neurologic recovery will be better in the case of peripheral ischemia.

However, patients can also have spinal cord compression with no relationship to COVID-19 infection. The presence of COVID-19 infection can lead us astray, and we can miss the urgent diagnosis requiring medullar magnetic resonance imaging studies and surgical decompression.[5] Finally, the diagnosis might be acute myelopathy associated with COVID-19 infection[6]; however, this must remain a diagnosis of elimination, in particular, because no specific treatment is available. Thus, with or without COVID-19 infection, patient with acute paraplegia must very quickly undergo computed tomography and, if the findings are negative, medullar magnetic resonance imaging.

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COVID-19 thromboembolic complications: Deepening immunoinflammatory features

It was the end of 2019 when severe acute respiratory syndrome coronavirus-2, the respiratory pathogen capable of binding to human angiotensin-converting enzyme 2 receptors and responsible for coronavirus disease 2019 (COVID-19), was isolated in Wuhan, China; since then, COVID-19 has been affecting the worldwide population for its many clinicopathologic aspects, including vasculocoagulative ones.[1,2] The interesting study by Indes et al[1] correlates the high incidence of arterial thromboembolic events with COVID-19 severity, outlining, as key factors, an abnormal immune/inflammatory reaction, and a hypercoagulative state.[1] Deepening their pathophysiologic features helps to better understand this complication.

The lymphocytic endotheliitis, which is expressed early with endothelial cells damage, and a consequent accelerated pyroptosis can evolve toward a leukocytoclastic vasculitis, a frank acute necrotizing vasculitis owing to immune complexes and complement fraction depositions as well as activation in the vascular walls, characterized by the infiltration of neutrophils into the media and adventitial layers of small and medium arteries and the occurrence of fibrinoid necrosis.[3,4] This diffuse endothelial damage is followed by an increase in the circulating endothelial cells and a type III hypersensitive reaction.[4,5]

The severity of COVID-19 includes a progressively higher production of cytokines, inducing, among other complications, a hypercoagulative state. In particular, IL-6 correlates with an augmented megakaryocytopoiesis, proven by histologic study of bone marrow and lungs with a high number of denuded megakaryocytes.[6] This is followed by a greater release of circulating platelets, already active in the earlier stages of this disease in repairing damaged vascular endothelia, and, according to their myeloid origin, in performing covercytosis against virions.[6] In the meantime, from an immunologic perspective, a