Multiple arterial thrombosis in a patient with COVID-19

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DESCRIPTION

A 61-year-old man presented to the emergency department with complaints of asthenia, dyspnoea and dry cough for the past 4 days. He had a medical history of type 2 diabetes and dyslipidaemia. On chest CT, an extensive bilateral SARS-CoV-2 pneumonia was diagnosed, and pulmonary embolism was ruled out. Dexamethasone (6 mg once daily) as well as enoxaparin (1 mg/kg per day) were initiated.

Five days later, due to severe respiratory failure, the patient was admitted to the Intensive Care Unit. At the time, there was no clinical or analytical evidence of coinfection.

Twenty-four hours after admission, he complained of intense pain on the lower limbs. On physical examination, both femoral pulses were palpable, but there was pulselessness, pallor and poikilothermia of the distal extremities, suggestive of arterial ischaemia (figure 1). Due to worsening of the respiratory failure, the patient was intubated.

Blood tests showed new onset hyperlactataemia (4.87 mmol/L), coagulopathy (elevated INR-1.32, aPTT-49 seconds, d-dimer and fibrinogen), leucocytosis (14.8x10⁹/L) and elevated inflammatory markers (C reactive protein: 162.4 mg/dl; procalcitonin: 2.41 mg/dl; ferritin: 5961 µg/L; lactate dehydrogenase 817 mmol/L).

A CT angiography was performed (video 1) and revealed an intramural aortic thrombosis with more than 50% stenosis of the descending thoracic aorta, as well as a complete occlusion of the lower abdominal aorta with extension to both common iliac arteries. A left renal infarction as well as a pulmonary right lower lobe consolidation suggestive of coinfection are shown.

A total of 100 mg of alteplase were infused over 2 hours. Blood cultures and tracheal aspirate were obtained, and intravenous antibiotics were started.

In spite of these therapeutic measures, the patient was refractory to all supportive care and ended up dying.

A complete blood panel was available a few days later and showed no signs suggestive of an acquired or congenital thrombophilia.

This case report intends to reinforce the difficulty to manage multiple arterial thrombotic events in a patient with COVID-19, despite prophylactic measures as well as pulmonary consolidation suggestive of coinfection. There was no evidence of pulmonary thromboembolism or aortic dissection.

Learning points

- SARS-CoV-2 infection can associate with multiple arterial thrombotic events, despite prophylactic measures.
- Critical patients with COVID-19 have a higher risk to develop thromboembolic events.
- Prophylaxis as well as a prompt diagnosis and initiation of directed therapy are the mainstay to manage these patients.
status with elevation of fibrinogen and d-dimer. These factors, alongside prolonged immobilisation in a critical patient, promote thrombotic events, even in the absence of previous history of atherosclerosis.

Distinct from other viral infections, SARS-CoV-2 infection promotes arterial thrombotic events. Prophylaxis as well as a prompt diagnosis and initiation of directed therapy are the mainstay to manage these patients.

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