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DOI: 10.17235/reed.2021.8099/2021
Link: PubMed (Epub ahead of print)

Please cite this article as:

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Portal thrombosis in a patient with SARS-CoV-2 infection

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Received: 17/05/2021
Accepted: 14/06/2021
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Keywords: SARS-CoV-2. COVID. Portal thrombosis.

Conflicts of interest: the authors declare none.

CASE PRESENTATION
A 72-year-old female with diabetes and Parkinson’s disease came to the Emergency Department due to fever and dyspnea. On arrival, she presented a basal oxygen saturation of 76 %. On physical examination, bibasal crackles on auscultation and pain in the right upper quadrant with positive Murphy’s sign on abdominal palpation were found. Laboratory tests showed leukocytosis at 25,500 cells/µL (normal values (NV): 3000-12,000/µL), platelets at 677,000/µL (NV: 120,000-450,000/µL), D-dimer at 34.56 µg/mL (NV: 0-0.5 µg/mL), and fibrinogen (Clauss) at 865 mg/dL (NV: 150-450 mg/dL), as well as a positive SARS-CoV-2 PCR. Chest X-rays showed bilateral pneumonia and a thoracoabdominal CT scan ruled out pulmonary thromboembolism, although a filling defect in the bifurcation of the portal vein compatible with portal thrombosis was
found (Fig. 1). Congestion of tributary mesenteric vessels was also observed, without signs of intestinal distress (Fig. 2). There were no findings suggestive of an abdominal infectious process.

The patient was admitted to the COVID Unit and received ventilatory support, dexamethasone, antibiotic treatment, and anticoagulation with low molecular weight heparin at a dose of 60 IU every 12 hours, with an excellent clinical response. Given the favorable evolution, she was discharged with prolonged anticoagulant treatment. After six months, the patient remains asymptomatic with a partial resolution of her portal thrombosis on ultrasonography.

DISCUSSION

The inflammatory effect of SARS-CoV-2 infection triggers a state of hypercoagulability, platelet activation, and endothelial dysfunction that increases the risk of thromboembolic events. This risk can be monitored with laboratory parameters such as elevation of D-dimer, fibrinogen, and platelet levels. Endothelial damage occurs by direct infection of the vascular cells by the virus (1).

The receptor for the angiotensin converting enzyme 2 (ACE-2) seems to play a fundamental role in the entry of the virus into the body. Although the most common route of infection is the respiratory tract, all organs with ACE-2 receptors can be gateways for SARS-CoV-2 particles into the body, including the small intestine. In patients in whom entry takes place via the digestive tract, digestive symptoms are more frequent. After the virus enters intestinal cells through endocytosis, migration occurs to the rest of the body. The portal system plays an important role in this dissemination. Thus, the appearance of venous thromboembolic events at the portal level could be more frequent in these patients (2).

REFERENCES


Fig. 1. Abdominal CT scan in the portal phase. The red arrow indicates the portal bifurcation where the thrombosis is located.
Fig. 2. Abdominal CT scan in the arterial phase. The orange circle indicates congestion of the tributary mesenteric vessels. Preserved intestinal wall uptake.