

Title:

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

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CASE PRESENTATION:

A 72-year-old woman with diabetes and Parkinson's disease came to the Emergency Department presenting fever and dyspnoea. At arrival she presented 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 detected. Analytically she presented leucocytosis of 25500/uL (Normal Values (NV) 3000-12000/uL), platelets 677.000/uL (NV 120000-450000/uL, D-Dimer of 34.56ug/mL (NV 0-0.5 ug / mL) and fibrinogen (Clauss) 865mg/dL (NV 150-450mg / dL). Positive SARS-CoV-2 CRP. A chest X-ray showed bilateral pneumonia. A thoracoabdominal CT which ruled out pulmonary thromboembolism was performed, visualizing a filling defect in the bifurcation of portal vein compatible with portal thrombosis [Figure 1]. A congestion of tributary mesenteric vessels was also observed, without signs of intestinal distress [Figure 2] No findings suggestive of 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 60IU every 12 hours with excellent clinical response. Given the favourable evolution, she was discharged with prolonged anticoagulant treatment.

After six months, the patient remains asymptomatic with partial ultrasound resolution of the portal thrombosis.

DISCUSSION:

The inflammatory effect of SARS-CoV-2 infection triggers a state of hypercoagulability, platelet activation, and endothelial dysfunction which increases the risk of thromboembolic events. This risk can be monitored with analytical parameters such as elevation of D-Dimer, fibrinogen, and platelets. 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 an ACE-2 receptor can be gateways for SARS-CoV-2 particles into the body, including the small intestine. In patients in whom the entrance door is the digestive tract, digestive symptoms are more frequent. After the virus enters the intestinal cells through endocytosis, migration occurs to the rest of the body. The portal system plays an important role in this dissemination. For this reason, the appearance of venous thromboembolic events at the portal level could be more frequent in these patients [2].

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Figure 1: Abdominal CT in portal phase. The red arrow indicates the portal bifurcation where the thrombosis is located.

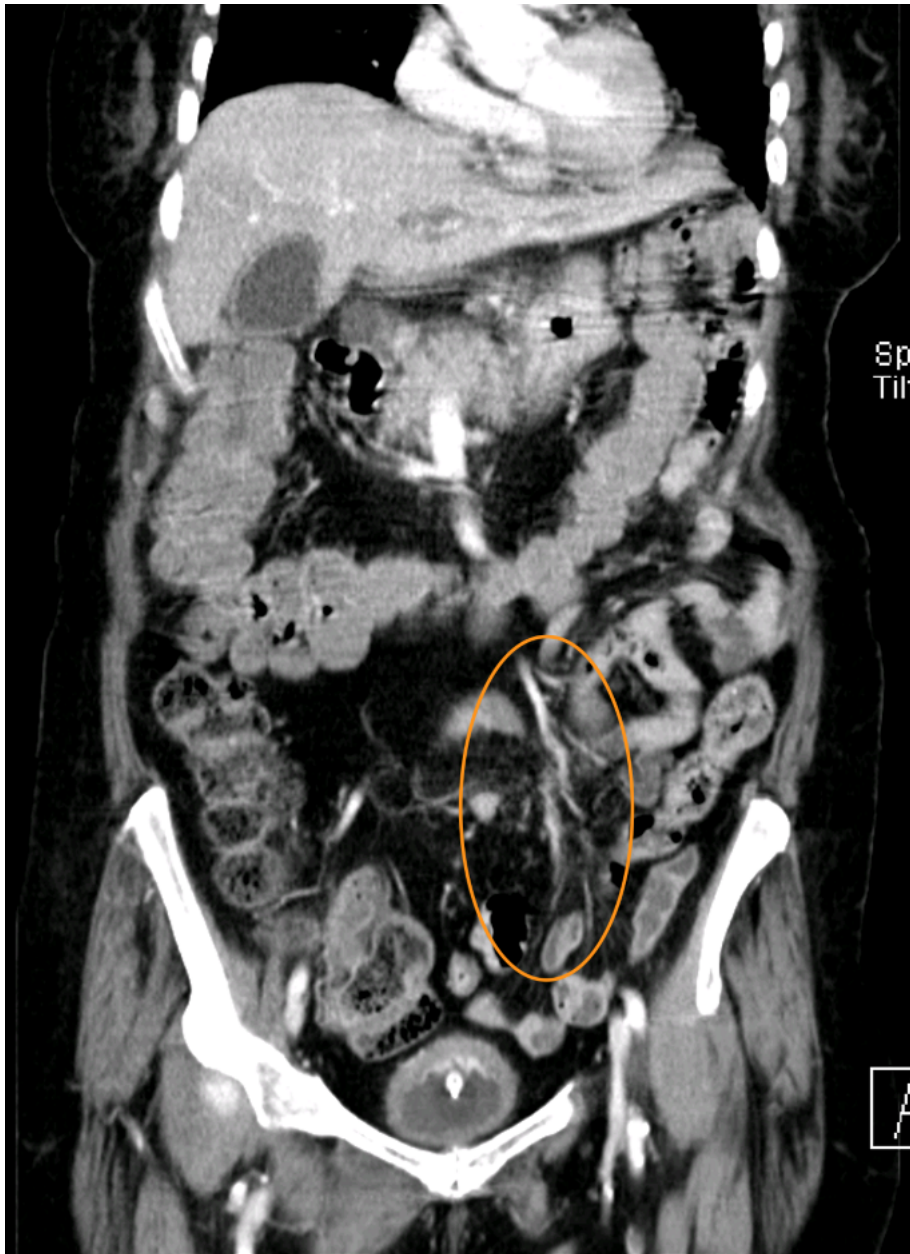


Figure 2: Abdominal CT in arterial phase. The orange circle indicates congestion of the tributary mesenteric vessels. Preserved intestinal wall uptake.