A 57-year-old male of Central American origin was referred due to acute lower-back and abdominal pain with nausea. One week before the patient described flu-like symptoms, primarily represented by headache. Upon admission, the patient was afebrile and hypertensive (158/87 mm Hg) with otherwise normal vital parameters and electrocardiography. White blood cell count was 10.9 G/L and C-reactive protein was 5.6 mg/L (reference: <5 mg/L). The clinical examination indicated a possible renal genesis of the symptoms. Consequently, an angio-computed tomography (angio-CT) was done on the same day and showed a stenosis of the proximal splenic artery and splenic infarctions (►Fig. 1A). In addition, an enlargement of the truncus coeliacus (diameter of 14 mm) was noted (►Fig. 1B). The presence of minimal bibasilar opacities was consistent with a positive SARS-CoV2 PCR-based test done upon admission. Potential sources of embolization were excluded. Common viral infections were excluded. Autoimmunity tests orienting toward a rheumatologic genesis of the disease were negative or normal.

A few days later, despite therapeutic-dose heparin and antiplatelet treatment in the suspicion of antiphospholipid syndrome and a steroid therapy in the suspicion of atypical panarteritis nodosa, a novel imaging test showed progression of the splenic infarcts, novel bilateral kidney infarcts (►Fig. 1C–F), and a spontaneous dissection of the mesenteric artery and collaterals (►Fig. 2A–C). We observed a concomitant dramatic increase of the inflammatory and necrosis parameters, notably C-reactive protein (5.6–266 mg/L) and LDH (423–1,241 U/L), in concomitance of the worsening of renal function (estimated glomerular filtration rate from 103 to 37 mL/min), indicating the acute nature of this process. No inherited or acquired thrombophilia was identified, leaving coronavirus disease 2019 (COVID-19) as the sole identifiable risk factor. Steroid therapy was then immediately stopped due to the low probability of a rheumatologic cause. During hospitalization, the patient showed an improvement of symptoms with progressive normalization of laboratory parameters. After 6 weeks of anticoagulation, a secondary prevention with antiplatelet agent was suggested, which was continued for 3 months.

The clinical spectrum of patients with COVID-19 ranges from asymptomatic cases to severe pneumonia with acute respiratory distress syndrome. COVID-19 is associated with an increased risk of thromboembolic complications, notably pulmonary embolism and deep vein thrombosis. Arterial cardiovascular complications and myocarditis have also been described in association with COVID-19, but appear to be less prevalent. In this report of a 57-year-old man with multiple splanchnic infarctions, arterial dissections and COVID-19 as the sole potential trigger, we describe a novel type of complications and put it in the context of a growing literature on this topic.
respiratory distress syndrome. COVID-19 is associated with a hypercoagulable state, endotheliitis, and a generalized inflammatory response, which ultimately lead to an increased risk for venous thromboembolic complications and high mortality.\(^1,2\)

Our otherwise healthy patient developed acute symptomatic splanchnic thrombosis, infarctions, and progressive arterial dissections despite mild COVID-19 not associated with hyperinflammation or hypercoagulability and, during the course of hospitalization, despite anticoagulant and antiplatelet therapies. This clinical picture resembled only partly that of segmental arterial mediolysis. Over the past weeks, an increasing number of reports concerning COVID-19 patients indicated that acute spontaneous arterial dissections and multiple infarctions may occur and do not appear to be associated with an apparent source of embolization.\(^3\)–\(^5\) These reports contribute to raise awareness of this complication, the pathophysiology of which remains to be studied.

**Note**

The present work was conducted at the Clinic of Angiology of the University Hospital Zurich (Switzerland) and was not funded.
Conflict of Interest
The authors declare that they have no conflict of interest.

References

Fig. 2 (A–C) Contrast-enhanced CT shows the dissection of the mesenteric artery and its collaterals (white arrows).