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Case Report

Thrombosis in abdominal vessels associated with COVID-19 Infection: A report of three cases [☆]

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ABSTRACT

Hypercoagulability related to SARS-CoV-2 infection is one of the main extrapulmonary complications of COVID-19. We present three cases of intrabdominal thrombotic complications related to the state of hypercoagulability of COVID-19 and its tomographic features. Hypercoagulability state should be taking into account in the interpretation of radiological images in all infected patients with COVID-19.

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Introduction

The viral infection of the new coronavirus [SARS-CoV-2] can produce a variety of extra pulmonary complications, and the hypercoagulability state is one of the most important and serious sequela [1]. The physiopathology of this complication

is unknown, however, the suggested mechanism associated could be: endothelial injury by direct invasion of the virus to endothelial cells [2], activation of the intravascular complement system, and endothelia known as "cytokine storm" [3,4]. Other mechanisms could be increased coagulation factors such as factor VIII and fibrinogen, hyperviscosity of the blood [5], thrombotic microangiopathy, and venous stasis [6,7].

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D-dimer elevation and hypercoagulability are prognostic factors of morbidity [6]. In patients bearing these characteristics, arterial thrombosis risk appears to be lower than the risk of venous thrombosis.

Although percutaneous endovascular is considered the first choice of treatment in patients with mesenteric and/or visceral abdominal ischemia. In our experience, we have observed that patients with COVID-19 infection might have generalized or vascular access thrombosis, possibly because the endothelium manipulation enhances the demonstrated prothrombotic state.

This report presents three cases of intra-abdominal ischemic pathology associated with a prothrombotic state triggered by COVID-19 infection.

Clinical case 1

We attended in the urgency room a 62 year-old male patient with a history of high blood pressure with a 2 hour evolution and colic-type abdominal pain after food intake, along with several emetic episodes. Five days before admission, the patient had a molecular test positive for SARS-CoV-2, managed at home because he had only mild symptoms.

Clinical parameters at admission were cardiac frequency, 88 lats/min, respiratory frequency, 16/min, arterial pressure, 140/90 mm/Hg, temperature, 36.5°C, and 96% oxygen saturation. Heart sounds were rhythmic without murmurs, vesicular murmurs preserved without over grouped noises. Depressible soft abdomen, painful on palpation in the mesogastrium without signs of peritoneal irritation.

In the abdominal computed tomography (CT) with contrast, multiple peripheral frosted glass spotlights, compatible with viral pneumonia, were observed at both pulmonary bases. In addition, there was an absence of opacification of the superior mesenteric artery in its distal part, suggesting the presence of thrombus, likewise, dilation of the thin intestinal loops was identified (distal jejunum and proximal ileum) (Fig. 1).

The laboratory studies showed leukocytosis (20100 u/L), increased D-dimer (58608 ng/ml), LDH (534u/L), and serum ferritin (1543 u/L), while other studies were within normal limits.

The medical treatment given was anticoagulation with non-fractional heparin (HNF). Thrombectomy was not considered due to the distal location of the thrombus. The endovascular manipulation of the endothelium increases the risk of generalized thrombosis due to the hypercoagulability of the patients. At 48 hours after admission, the patient had increased pain and abdominal distention progressively increased his oxygen requirements and received mechanical ventilation.

Abdominal computed tomography angiography showed a sub-occlusive thrombus-filling defect in the distal third of the upper mesenteric artery and its distal branches. It was associated with the diffuse absence of enhancement in the ileum wall due to the evolving ischemic events. Adjacent free fluid was observed as a sign of loop distress, diffuse dilation of intestinal loops, and formation of air-fluid levels due to secondary ileums. (Fig. 2).

Due to the tomographic findings, we indicated an exploratory laparotomy and were observed that a moderate

amount of hemorrhagic fluid throughout the abdominal cavity. Areas of necrosis and segmental ischemia throughout the jejunum and ileum because of their wide extension did not benefit from the surgical resection. Subsequently, the patient presented a torpid evolution, given the intestinal ischemia associated with complications from COVID-19 infection.

Clinical case 2

A 22-year-old female patient, without significant past medical history, presented a 20-days history of abdominal pain mainly located in the mesogastrium. The patient had multiple admissions to the emergency room. In her last visit, 8 days before admittance, she presented with an increase in appendix size. It was shown in the abdominal CT the patient underwent an appendectomy. The pain reappeared on the first postoperative day and was associated with fever for 3 days. The patient was readmitted without gastrointestinal symptoms. Moreover, the patient referred she had close contact with relatives with respiratory symptoms who had not been tested for COVID-19.

In the physical examination, at admission, the patient had O₂ Sat 96%, soft depressive abdomen, healthy appendectomy, surgical wound without signs of infection, with pain on palpation of the transverse and descending colic frame without signs of peritoneal irritation. The abdominal angiography was indicated and acute and/or subacute thrombosis of the superior mesenteric vein with edematous changes of the adjacent fat was visualized (Fig. 3).

Thrombophilia and autoimmune pathology were excluded. The management was carried out with anticoagulation, analgesia, and antibiotic therapy due to the patient presenting feverish peaks during hospitalization. She had a favorable evolution.

Clinical case 3

A 65 year-old female patient came to our center with a history of left nephrectomy, 15 day history of respiratory symptoms, a positive PCR study for COVID-19 (8 days before admission), 7 days of evolution of abdominal pain predominantly in the upper hemiabdomen, with back irradiation, associated with hyporexia.

In the physical examination, at admittance, we found FC: 86/min, FR: 15/min, PA: 162/97, T: 36°C, Sat O₂: 94%, visual analog pain scale: 8/10. Rhythmic heart sounds, non-aggregated ventilated lungs, depressible soft abdomen, pain on palpation of the upper hemiabdomen, and no signs of peritoneal irritation.

Contrast abdominal tomography was performed, where we found thrombi in the left jejunal artery branch with infarction of the corresponding jejunal loops. In addition, we observed edema of the mesenteric fat and hypo attenuation of the intestinal wall with greater dilatation of the compromised intestinal segment. It showed signs of splenic infarction and pleural parenchymal bands in both lung bases (Fig. 4).

The patient underwent an exploratory laparotomy with an intestinal resection of the compromised segment, intraperitoneal collection, drainage, and cavity lavage were performed.

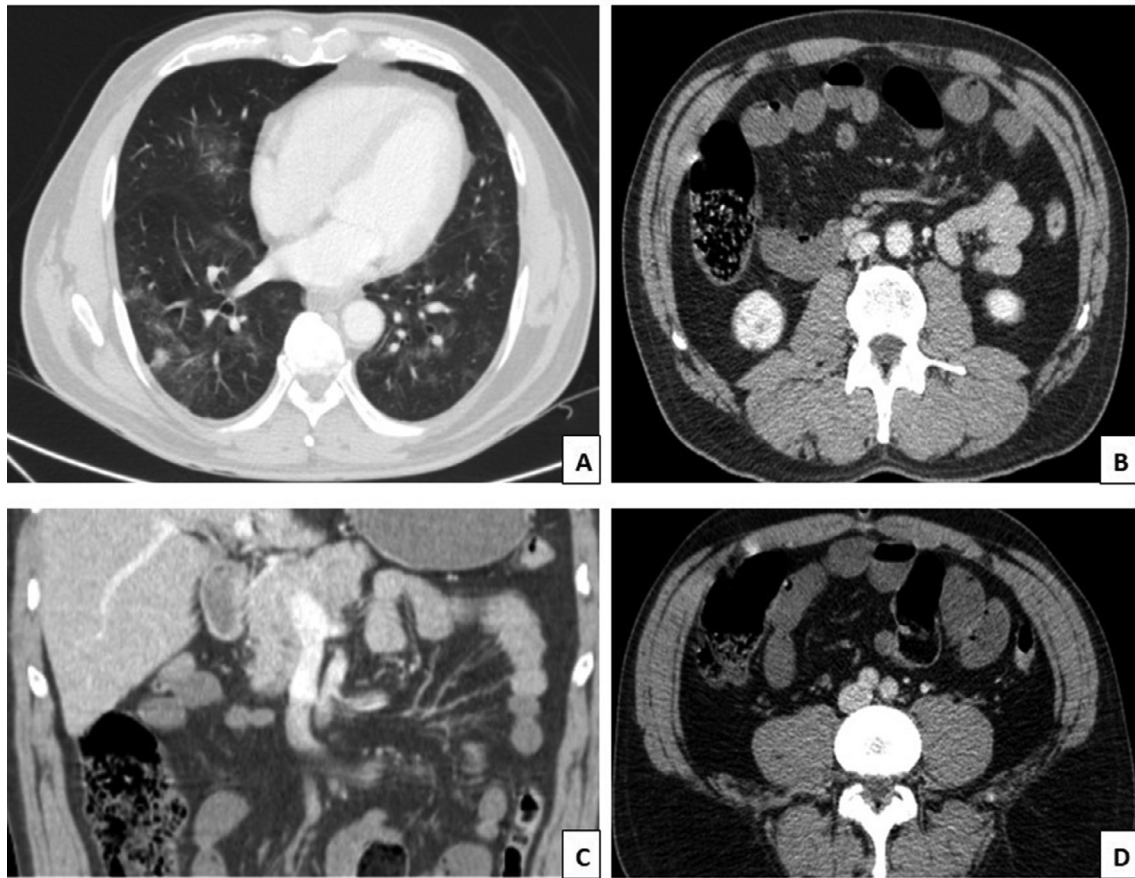


Fig. 1 – (A) Lung bases with multiple peripheral ground glass foci compatible with viral pneumonia. (B and C) Distal thrombus in the superior mesenteric artery with the occlusion of this and its branches. (D) Dilatation of thin intestinal loops

Discussion

Currently, tested computed tomography is considered as the imaging study of choice for the evaluation of patients with clinical suspicion of arterial or venous thrombosis. It allows direct visualization of the thrombus as a defect within the affected vessel. Due to its high sensitivity (93.3%) and specificity (95.9%), it has displaced angiography [8–10]. In addition, it allows characterizing the complications derived such as mesenteric ischemia, splenic infarction, liver, renal, portal thrombosis, Bud Chiari, among others.

For the evaluation of intra-abdominal thrombotic pathology, it suggested to perform an angiotomography with an iodinated contrast volume of 80 to 100 ml (320–350) through a 20-gauge antecubital intravenous catheter, at an injection speed of 4 ml/s, with a slice thickness of 0.625 mm and multiplanar reconstructions that allow adequate characterization of the findings [8].

The most common intra-abdominal thrombotic complications are described below.

Mesenteric ischemia

Within abdominal vessel thrombosis, the most common presentation is mesenteric ischemia, which usually appears

acutely in up to 95% of cases. Its etiology corresponds to arterial obstruction, both embolic and thrombotic in 60% to 70% of cases, venous obstruction in 5% to 10%, and non-occlusive associated with pathologies such as acute myocardial infarction, low cardiac output, and hypovolemia in 20% [9].

Acute mesenteric ischemia has classically presented in adults over 60 years of age due to comorbidities such as atrial fibrillation, atherosclerosis, arterial hypertension, heart failure, valve disease, and portal hypertension. However, in the current context of the COVID-19 pandemic, both acute mesenteric ischemia and complications associated with thrombosis of abdominal visceral vessels are a cause that must rule out in patients infected by the virus, who present with acute abdominal pain, regardless of age group.

The severity of mesenteric ischemia varies from mild, where generally transient superficial changes of the intestinal mucosa occur, to severe, producing transmural necrosis of the intestinal wall, more dangerous and potentially fatal [10].

The natural evolution of the disease leads to successive complications that begin with bacterial proliferation in the intestinal wall (pneumatosis of the wall) favored by its necrosis, subsequently, the gas passes to the mesenteric vessels and from there to the portal vein (portal pneumatosis). Finally, sepsis and/or intestinal perforation are commonly the causes of death. It is important to mention that ischemia of thrombotic origin can lead to reactive mesenteric vasoconstriction, reduc-

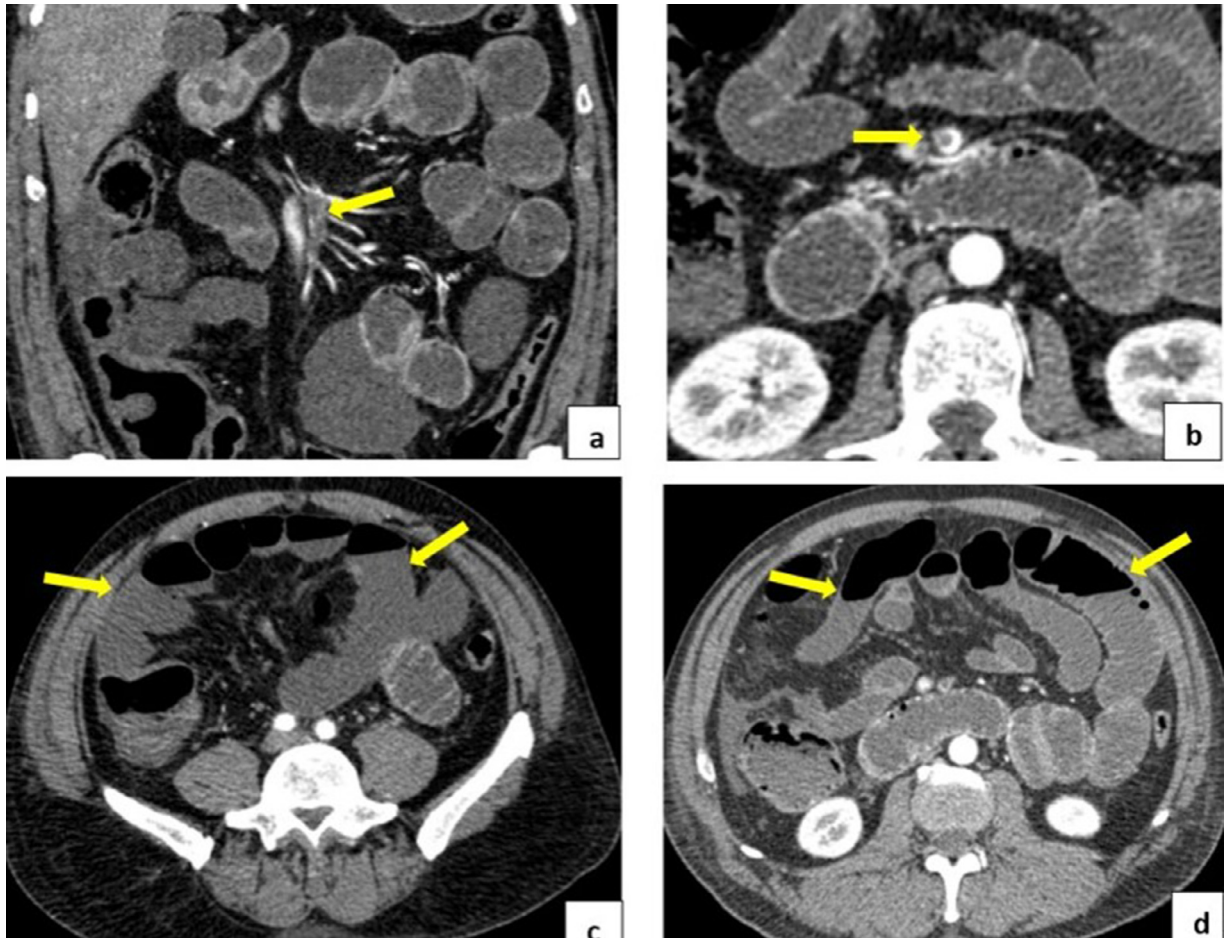


Fig. 2 – (A and B) Subocclusive thrombus in the distal third of the superior mesenteric artery. Absence of enhancement in the walls of thin intestinal loops in the relationship of signs of evolving ischemia. (D) Diffuse dilation of intestinal loops and presence of air-fluid levels

ing compensatory collateral flow, worsening ischemic damage [11].

Regardless of the clinic, abdominal pain is a common characteristic in patients with this pathology. Usually of rapid onset and high intensity could be associated with nausea, vomiting, abdominal distension, and diarrhea [9,10].

The findings observed in tomography include identification of the arterial and/or venous vascular filling defect. In addition, visceral findings such as parietal thickening, a more marked mural of the affected loop wall with a “target” appearance, dilation of intestinal loops, and hydro levels in the airways. In more severe cases, when there is intestinal infarction, thinning, and hypo enhancement of the intestinal wall with or without pneumatosis, the air in the mesenteric and/or portal veins. Other findings include diffuse increased attenuation of mesenteric fat, interested fluid, and pneumoperitoneum [10–13].

In any case, the success of the treatment is obtained by influencing the reversible phase of ischemia to avoid complete necrosis of the intestinal loop. On the other, endovascular management is based on quickly restoring blood flow in the affected vessel using techniques such as aspiration, thrombectomy, thrombolysis, and angioplasty with or without

stenting, surgical exploration of the intestine, and resection of necrotic intestinal segments, and anticoagulation [14].

Portal thrombosis

It is a rare complication in the general population with a prevalence of 1%, relative to most cases due to hepatic cirrhosis. However, it can also appear as a primarily vascular disorder, which is a hereditary or acquired prothrombotic state, associated with tumor invasion of the carrier or pylephlebitis [15,16]. It can also spread to the splenic vein and upper mesenteric vein [17].

Tomographic findings are the filling defect that partially or completely occludes the light of the vessel and where enhances the edge of the vessel wall, alterations, and heterogeneity in the liver parenchyma highlight this pattern. In chronic cases, there can be seen a decreased diameter of the carrier with collateral circulation around it, which can in some cases be seen as a solid mass in the liver hilum with progressive enhancement, where it is not possible to individualize the national veins, which is known as “portalcavernomatosis” [18]. When thrombosis is acute, a higher attenuation content

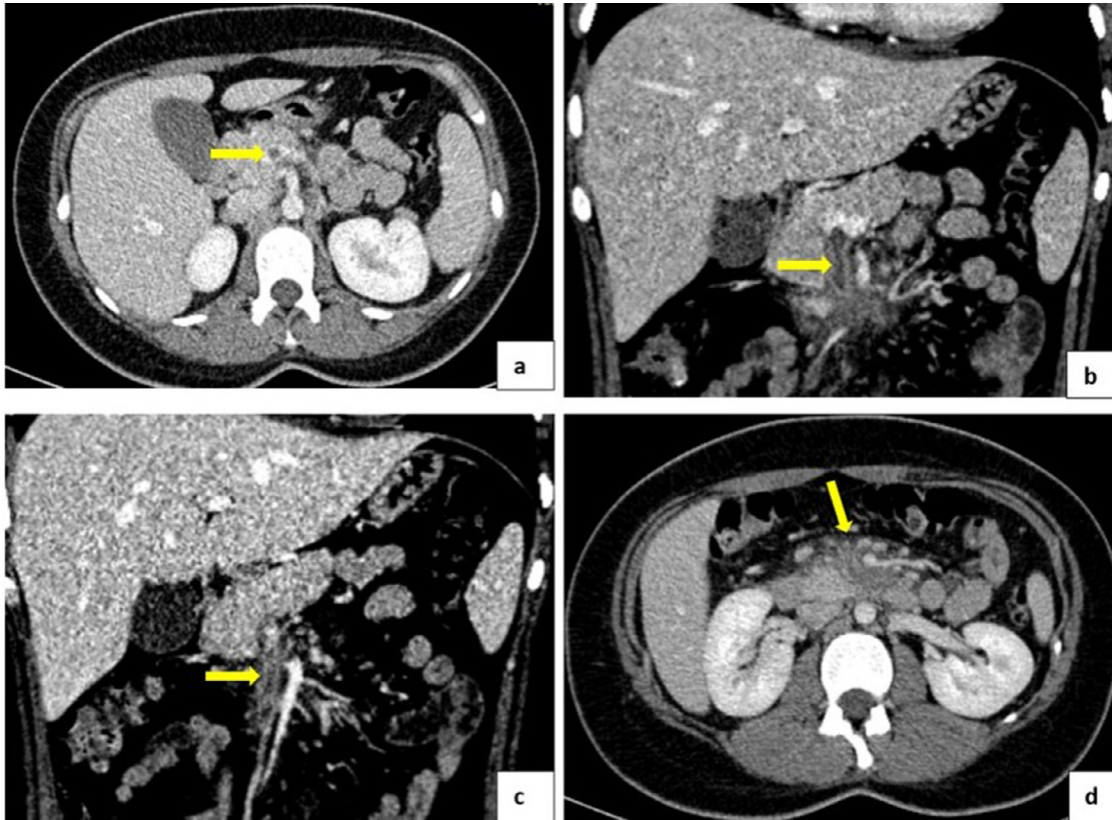


Fig. 3 – (A, B, and C) Obstructive thrombus is evidenced in the superior mesenteric vein. (D) Diffuse increase in the density of the fatty planes of the root of the mesentery to inflammatory changes

can sometimes be identified inside the portal vein and an increase in cup caliber.

Splenic infarction

For the evaluation, it is recommended to carry out the tomographic study in the venous phase, to avoid confusion with the “brindle” the enhancement characteristic of the splenic parenchyma in the arterial phase. Wedge hypodensity is one of the typical characteristics of this pathology, as well as multiple hypodense or hypo enhanced areas of the entire spleen, in cases of multiple infarcts or global infarction respectively [19,20].

It should be taken into account that in the hyperacute phase there may be hyperdense areas in the simple phase due to hemorrhagic infarcts. While in the chronic phase, it is common to find areas of fibrotic retraction of the parenchyma or cystic areas due to tissue liquefaction [20, 21]. Associated complications are rupture and abscess [22].

Bud Chiari syndrome

It comprises a group of conditions characterized by a partial or complete blockage of the outlet flow of the hepatic veins.

If the blockage is severe and not corrected early, it can lead to necrosis of hepatocytes and progress to liver fibrosis and cirrhosis [23,24].

The most common cause is an underlying hypercoagulability or prothrombotic state. Clinical presentation ranges from mild symptoms to fulminant hepatic impairment, and most patients manifest as a chronic liver disease [24]. Acutely, patients have predominant pain in the hemiabdomen, ascites, hepatomegaly, renal failure, and jaundice [25].

In tomography, the most common direct sign is the absence of visualization of the hepatic veins. If thrombosis is acute, a filling defect will be evident inside the hepatic veins, which are usually distended, with the presence of a hypodense thrombus in light [26].

There are also indirect tomographic findings characterized by morphological changes in the patterns of parenchymal enhancer and collateral circulation. In the acute stage, there is uniform hepatomegaly with a smooth contour of the liver, a heterogeneous liver enhanced in the portal phase, and hypodense areas setting up a mosaic pattern, due to venous congestion. More than in subacute and chronic stages, when intrahepatic collateral has developed and hepatic mass fibrosis has been established, it is common to find a liver with nodular contour is, with volume redistribution to signs of cirrhosis and differences in the pattern of energy become more subtle [25,26].

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