Case Report

Splenic infarction and spontaneous rectus sheath hematomas in COVID-19 patient

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ABSTRACT

Multiple studies and reports have suggested that coronavirus disease-19 (COVID-19) promotes arterial and venous thrombotic events in multiple organ systems, although the mechanism leading to a hypercoagulable state is still unknown. Few cases of splenic infarction associated with COVID-19 have been reported, of which half were found incidentally upon autopsy. This may be due to a clinically silent presentation or the symptoms being wrongfully attributed to pain caused by the effects of COVID-19. Due to the rarity of the condition and its lack of consistent symptomatology, splenic thromboembolism can be difficult to diagnose. Awareness of the condition and high clinical suspicion will help the clinician identify and manage the problem. Hemorrhage in patients with COVID-19 is uncommon in the hypercoagulable state that threatens thrombus formation in patients with COVID-19 infection. Despite prophylactic treatment with anticoagulation therapies, patients are more prone to developing clots. It is also well-known that therapeutic anticoagulation can place patients at a higher risk of bleeding. Thus, this unique population is at risk of developing both thrombotic and hemorrhagic events. We report a rare case of splenic infarction in a patient with confirmed COVID-19 infection despite prophylactic treatment with low-molecular-weight heparin which was found incidentally during workup for 2 other rare conditions: spontaneous rectus sheath hematoma and microhemorrhage or thrombus of the mesenteric vessels.

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Introduction

In December 2019, the first cases of COVID-19 caused by the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) were reported in Wuhan, China. Since, there have been 95,612,831 confirmed cases of COVID-19, including 2,066,176 deaths, reported to the World Health Organization [1]. Multiple studies have been done to examine clinical and epidemiologic aspects of the disease [2-7]. It is well established that COVID-19 has a myriad of presentations, ranging from asymptomatic to severe illness; the most severe cases being observed in the elderly and those with comorbid conditions [8,9]. COVID-19 predominantly attacks the pulmonary system, and causes acute lung injury and diffuse alveolar damage, but it also has been shown to affect multiple systems in patients with and without comorbidities. Literature suggests that infection with COVID-19 provokes arterial and venous thrombotic events [10], and could be contributing to the multisystem damage. In an autopsy study done by Falasca et al histopathological hallmarks of widespread vascular injury were found in the liver, kidney, bone marrow, and spleen [3]. The exact mechanism causing these findings is not yet understood, but it is theorized to be a distinct process unique to the Sars-CoV-2 virus [10]. The current postulations attribute the pathophysiologic mechanisms of COVID-19-related hypercoagulopathy to systemic inflammatory response syndrome (SIRS) precipitated by cytokine storm or activation of the coagulation cascade due to cellular activation triggered by the virus, or both [11]. These pathologies are quantified by measurement of the D-dimer and fibrin degradation product levels which have been shown to correlate with the severity of disease and patient prognosis [7].

Interestingly, the patient we present suffered one confirmed and one possible hemorrhagic event as a result of being anticoagulated to prevent further thrombus formation. 80 mg prophylactic LMWH was being administered twice daily, but the measured D-dimer remained markedly elevated. COVID-19-related hypercoagulopathy dictates that clinically overt bleeding is uncommon in the setting of COVID-19 [12]. However, bleeding is a risk of therapeutic anticoagulation. Our patient spontaneously developed bilateral rectus sheath hematomas (RSH) as well as mesenteric vessel microhemorrhage. Rectus sheath hematoma is an uncommon cause of acute abdomen and can be benign, but in the setting of a patient receiving anticoagulation agents, can quickly progress to a life-threatening event. To date, there is only one other case reported of rectus sheath hematoma in a patient with COVID-19 [13]. Splenic infarct, mesenteric vessel infarct, and rectus sheath hematoma are rare in COVID-19 patients and providers should be aware of these potential complications. We present a case where all 3 are concurrently present in the same patient.

Narrative

A male in his 70s with a past medical history of hypertension, benign prostatic hypertrophy, gastroesophageal reflux disease, and depression presented to the emergency department with a chief complaint of worsening dyspnea. Seven days prior, the patient developed a dry cough with sore throat, and subsequently tested positive for COVID-19 which he had been managing at home on an outpatient basis. Since his COVID-19 diagnosis, his symptoms continued to worsen, and upon arrival to the emergency department he had dyspnea at rest, nonproductive cough, fevers, nausea, decreased appetite, and weakness. He was found to be in acute hypoxic respiratory failure with oxygen saturation (O2 SAT) of 68% on room air. His O2 SAT increased to 88%-92% on a 15% non rebreather mask. He was also tachypneic at 22 breaths per minute and afebrile at 37.6 °C. Chest X-ray revealed new extensive patchy consolidative opacities about the lungs, favored to represent multifocal pneumonia. Laboratory evaluation was significant for elevated D-dimer, fibrinogen, and ferritin at 14.41 mg/L, 620 mg/dL and 1973 mg/mL, respectively. Additionally, lactate acid and white blood cell count were both elevated at 2.6 mmol/L and 14,900 cells/μL, respectively. Blood and urine cultures were negative. Treatment with enoxaparin, dexamethasone, and remdesivir was initiated as the patient was admitted to the intensive care unit for inpatient management of sepsis, viral pneumonia, severe COVID-19 infection, and acute hypoxic respiratory failure.

On hospital admission day 2, repeat D-dimer continued to be elevated at 5.56 ng/mL. A duplex ultrasound examination of the bilateral lower extremities was performed which revealed no evidence of intraluminal thrombus. Over the course of the next few days, the patient reported improved symptomatology and resolved dyspnea. His acute hypoxic respiratory failure was improving; he was reduced from 90% FiO2 on BiPAP to 45% FiO2, high-flow nasal cannula/BiPAP. His care was transferred from intensive care to intermediate care. His D-dimer continued to be elevated at 4.20, 3.78, and 4.58 ng/mL on hospital admission days 3, 4, and 5, respectively.

On the morning of hospital admission day 6, the patient continued to endorse improvement of symptoms, including no nausea, vomiting, fever, or chills, but he began to complain of constipation. To manage said constipation, the patient was given 30 mL oral lactulose for symptomatic relief.

Later that evening, the patient had a large bowel movement. Subsequently, the patient experienced acute onset of severe left lower quadrant abdominal pain. CT scan of the abdomen and pelvis with intravenous contrast revealed several findings. A 4 cm well-demarcated area of nonenhancement within the anterior superior spleen (Fig. 1) consistent with acute infarct was seen. Additionally, there was inflammation within fat surrounding the mesenteric vessels in the left upper quadrant (Fig. 2) which was suspected to be microhemorrhage or thrombosis. There were also large hematomas within the bilateral rectus muscles (Figs. 3–5) beginning just above the umbilicus extending down to the pubis measuring 5.6 cm × 18 cm on the left (Fig. 4) and 7 cm × 4 cm × 10 cm on the right (Figs. 4 and 5). Contrast within the hematomas suggested active bleeding at the time of imaging (Fig. 3). Hemoglobin was tested and was found to have decreased from 13.7 g/dL at the time of admission to 10.7 g/dL shortly after the time of CT scan. The patient’s full dose enoxaparin, which was initially started due to significantly elevated D-dimer, was subsequently discontinued due to the presence of actively bleeding
hematomas, despite the presence of a splenic infarct. When measured earlier that morning, his D-dimer continued to be elevated at 3.90 mg/mL.

At this time, the decision was made to transfer the patient by helicopter to a tertiary care center for possible interventional radiology embolization as well as management of the large rectus hematomas. At this point, the patient had received 6 doses of IV remdesivir out of 10 as well as 6 days of IV dexamethasone. The patient was stable at the time of transfer.

After 2 days of in-patient care at the tertiary center, it was determined that the active bleeding had stopped, and the hematomas remained stable. The patient’s COVID-19 symptoms had also improved, and he was able to maintain adequate oxygenation levels while ambulating. The patient was discharged to complete his recovery at home. His recovery course has gone well without complications and as of 2 months following discharge he has been able to safely return to normal activity.

**Discussion**

COVID-19 infection is well known to cause hypercoagulability with pulmonary emboli being the most common presentation, splenic artery embolism and splenic infarction are rarely reported [14–16]. Splenic infarct is a rare cause of abdominal pain, often secondary to a hypercoagulable state [7]. COVID-19 hypercoagulability has been proposed to occur due to elevation of proinflammatory cytokines, including IL-6 [14–16]. Additionally, elevated D-dimer and fibrinogen degradation products are associated with poorer disease prognosis, potentially related to risk of disseminated intravascular coagulation [16]. Thus, treatment of the hypercoagulable state with antithrombotic agents is appropriate.

In a brief literature review, we identified 5 case studies consisting of 6 patients with involvement of splenic thromboembolism secondary to COVID-19 infection, including one case of atraumatic splenic rupture, and one case of hemoperitoneum [7,10,17–19]. Additionally, multiple post-mortem autopsies have identified splenic involvement secondary to COVID-19 infection [2–6]. There have also been 2 cases of psoas hematoma [20,21]. As of yet, there has been only one prior report of rectus sheath hematoma secondary to COVID-19 coagulopathy [13].

Abdominal scans are not routinely performed in COVID-19 patients as primary symptomatology involves the respiratory tract. Thus, only symptomatic splenic infarctions or those found incidentally on CT scans of the chest extending into the abdomen may be identified. Evidence of splenic involvement has been noted on autopsy in patients known to have had COVID-19 [2–6]. This suggests that the presence of splenic involvement due to COVID-19 hypercoagulability may be higher than reported. While most cases of splenic infarct may be asymptomatic, atraumatic splenic rupture can be a devastating complication and necessitates a high clinical index of suspicion for patients with abdominal pain and concurrent or prior COVID-19 infection.

Rectus sheath hematoma is an uncommon complication of anticoagulation therapy. Other risk factors include old age, female gender, history of abdominal surgery/trauma/injections, cancer, coagulopathies, and renal impairment [22]. We hypothesize that the development of RSH in our patient was due to shearing of the epigastric vessels caused by the combina-
tion of anticoagulation therapy and trauma caused by straining and/or coughing. Early diagnosis and intervention are key to improving patient mortality and morbidity. CT scan with IV contrast is considered the gold standard [22], for identification of bleeding and for differentiating between arterial and venous bleeds [13]. In cases where conservative management is unsuccessful, or the patient presents with severe clinical criteria, CT angiography can be utilized to identify active bleeding and help in staging for interventional radiology treatment [23]. In patients who have contraindication to contrast, Doppler ultrasound and red cell scintigraphy can be used. The required treatment should be dictated by the severity of the RSH and status of the patient. Conservative therapy including a binder, rest and analgesics may be adequate in the
stable patient, whereas patients with hemodynamic instability may require resuscitation using IV fluids and blood products. In patients who have been anticoagulated, intravascular coil embolization or rarely surgery to ligate the epigastic vessels, may be required to achieve adequate hemostatic control.

Conclusion

Splenic infarction in COVID-19 infection is rarely reported and may go undetected if symptoms are vague or obscured by other ailments such as constipation, placing patients at risk of splenic rupture. Likewise, SRSH are also rare, a source for abdominal pain and a potentially serious condition especially in the anticoagulated patient, necessitating emergent treatment and the need to weigh the risks of hemorrhage versus thrombus when considering anticoagulation reversal. Despite their rarity, these conditions are treatable and should be considered in COVID-19 patients with abdominal pain and radiologists should monitor for thrombosis to aid in early diagnosis. Additionally, further research or reports on acute hemorrhage in COVID-19 patients receiving anticoagulation is recommended.

Learning points

• COVID-19 is well known to predispose patients for hypercoagulable events and complications, including thrombosis of unusual locations such as splenic artery branches.
• Anticoagulant management of acute thrombotic events in COVID-19 is critical in prevention of morbidity and mortality.
• Full anticoagulation predisposes patients to adverse effects related to bleeding and hemorrhage, including rectus sheath hematoma.
• There are no clear guidelines on management of hemorrhagic events in patients on therapeutic anticoagulation as treatment of ischemia due to thrombus, the risks of hemorrhage versus thrombosis must be weighed.
• Further research or reports on acute hemorrhage in COVID-19 patients receiving anticoagulation is recommended.

Patient consent statement

We are using entirely anonymized images from CT scans. These do not contain any identifying marks and are not accompanied by text that might identify the individual concerned.

Credit author statement

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