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Necrotizing pancreatitis with portal vein thrombosis in young patient with COVID-19

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ABSTRACT

The current pandemic of the novel coronavirus disease 2019 (COVID-19) originated in Wuhan, China in December 2019. The most common clinical manifestations are fever, fatigue, and cough. Other common symptoms include anorexia, chest tightness and shortness of breath. Extrapulmonary manifestations including gastrointestinal symptoms were also reported in patients with COVID-19 infection. It has been found that the ACE2 receptor of SARS-CoV-2 is expressed more in the pancreas than in the lungs. However, only few cases reported with pancreatic injury were caused by COVID-19. In this paper, we report a young patient presenting with acute necrotizing pancreatitis that is complicated with portal vein thrombosis and found to have COVID-19 infection.

Case presentation

A 15-year-old Saudi patient, who is medically free, presented to emergency department in June 2021 with severe epigastric pain for three days radiating to the back and associated with nausea, vomiting and fever. He reported no similar episodes before and had no respiratory symptoms. The patient denied any travel history, alcohol consumption, drug abuse or any medications. He did not receive any dose of COVID-19 vaccine and he reported a history of contact with positive COVID-19 cases in his family. On physical examination, he was in severe pain with the following vital signs: heart rate 120 beats per minute, respiratory rate 20 breath per min, blood pressure 130/80 mmHg, temperature 36.5 °C, and oxygen saturation 97% on room air. The abdomen was soft with severe epigastric tenderness. Laboratory workup on admission showed leukocytosis with raised inflammatory markers and his serum lipase was significantly elevated, as summarized on (Table 1).

Based on APACHE-II score for disease severity, our patient had severe pancreatitis with a score of 9. Accordingly, an abdominal Computed Tomography (CT) with contrast was performed to assess for complications of pancreatitis. The CT images revealed features of acute necrotizing pancreatitis at the neck and body of the pancreas with acute necrotic collection (non-organizing fluid collection measuring: 2.5 × 6.3 × 4.1 cm), however gallbladder and biliary tract showed no gross abnormality with no evidence of stones or congenital anomalies of the pancreas (Fig. 1).

In addition, nasopharyngeal swab for SARS-CoV-2 was taken with positive result, but his chest x-ray was unremarkable (Fig. 2).

He was treated conservatively with intravenous fluid and antibiotics. A full work up for autoimmune causes of pancreatitis was negative. Following a 9-day stay in the hospital, his abdominal pain was not improving and the repeated inflammatory markers were increasing despite upgrading the antibiotic. Accordingly, the abdominal CT with contrast was repeated and showed interval progression of the pancreatic well-defined fluid collection with multiple enhanced collection, the largest measuring (7.6 × 10.5 × 10.6 cm) and associated with filling defect in the portal vein (Fig. 3).

The patient underwent percutaneous drainage of the necrotic collection by interventional radiology; cultures including aerobe, anaerobe, and fungal organisms were sent to microbiology lab and were negative. He was managed with therapeutic low molecular weight heparin (Enoxaparin) for Portal Vein Thrombosis (PVT). Furthermore, thrombophilia workup including antiphospholipid antibodies was unremarkable. After drainage and starting him on anticoagulation, he was significantly improving clinically and radiologically, and was discharged on Rivaroxaban for total of three months.

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Table 1
Laboratory investigations on admission.

Laboratory test	Level	Normal range
WBC count	21.5	4–10 × 10^3 cells/μL
Hemoglobin	10.4	12–14 g/dL
Platelet count	356	150–450 × 10^3 cells/μL
Hematocrit	31.5	35–45%
AST	46	< 38 U/L
ALT	29	< 40 U/L
Alkaline phosphatase	250	98–279 U/L
Total bilirubin	1.6	0.1–1.2 mg/dL
Direct bilirubin	0.7	< 0.25 mg/dL
Albumin	3.6	3.5–5.5 g/L
Amylase	324	< 90 U/L
Lipase	281	< 60 U/L
Sodium level	127	135–145 mEq/L
Potassium level	5.1	3.5–5.1 mEq/L
Calcium level	8.0	8.5–10 mg/dL
Triglyceride	123	30–150 mg/dL
Blood urea nitrogen	23	7–26 mg/dL
Creatinine	0.77	0.6–1.3 mg/dL
C-reactive protein	30.99	< 10 mg/dL
ESR	36	0 – 20 mg/dL
D-Dimer	12.31	< 500 mg/dL
INR	1.19	
PT	17.6	13.1–16.5 s
PTT	37.7	25.5–42.3 s
pH	7.325	7.35–7.45

WBC: White Blood Count, AST: Aapartate Aminotransferase, ALT: Alanine Aminotransferase, ESR: Erythrocyte Sedimentation Rate, INR: International Normalized Ratio, PT: Prothrombin Time, PTT: Partial Thromboplastin Time.

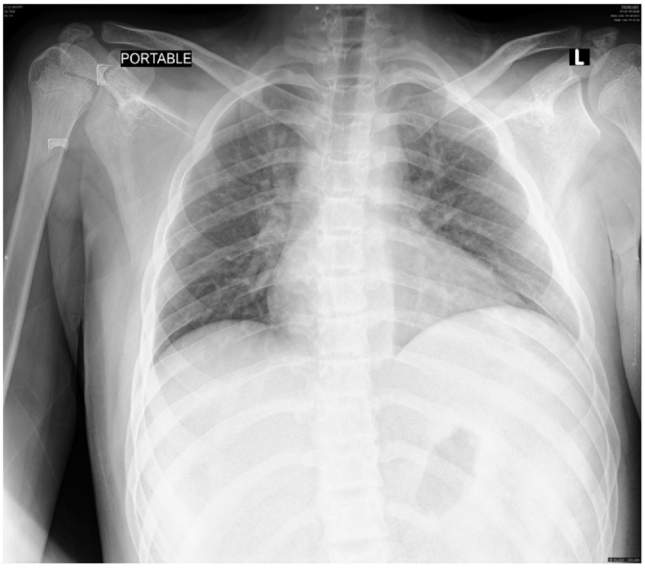

Fig. 2. Chest X-ray.

Discussion

Corona virus disease-19 (COVID-19) had been advocated pandemic in March 2020 [1]. It mainly affects the respiratory system with a variable clinical presentation, ranging from a flu-like syndrome with mild respiratory symptoms, viral pneumonia with acute respiratory failure, and multiple organ dysfunction [2]. However, extra pulmonary manifestations have been reported in illustrative studies from China, such as gastrointestinal symptoms which can be as high as 50% [3] These symptoms include nausea (17.3%), diarrhea (12.9%), anorexia (12.2%), abdominal pain (5.8%), belching (5%) and emesis (5%) [4]. Some articles have reported acute necrotizing pancreatitis in many patients due to COVID-19 infection. Wang et al. were the first to describe nine patients with acute pancreatitis in a case series of 52 patients with COVID-19 [5]. Although the exact pathogenesis of pancreatitis caused by COVID-19 is still unclear with many proposed theories, it has been reported in one study that ACE2 receptor is expressed more in the pancreas than the lungs [6]. This renders the pancreas to be a target for SARS-CoV-2, which leads to pancreatic injury from the direct cytopathic effect mediated by the local replication of SARS-CoV-2 [7].

A rapidly accumulating data suggests that COVID-19 is a multisystemic disease and can present with extra pulmonary manifestations, such as hypercoagulability leading to arterial and venous thrombosis. Many reports have shown increasing concerns about hypercoagulability in COVID-19 patients with a higher incidence of thrombotic complications have been observed in COVID-19 patients admitted in Intensive Care Unit (ICU) reaching 28% whereas in non-ICU setting the incidence was 10% [8,9].

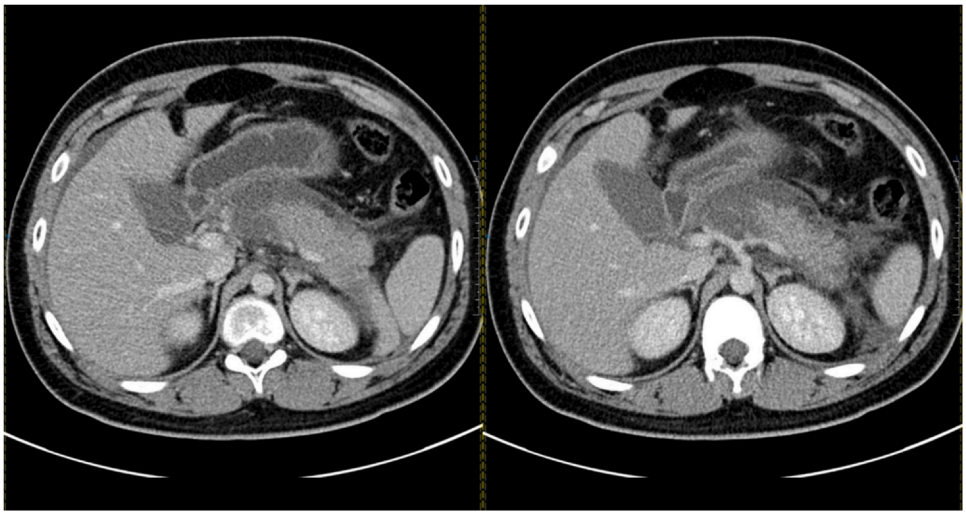

Fig. 1. Contrast-enhanced abdominal computed tomography (CT) showing non-enhancing low attenuation areas at the neck and body of the pancreas constituting 20–30% of pancreatic tissue, in keeping with acute necrotizing pancreatitis.

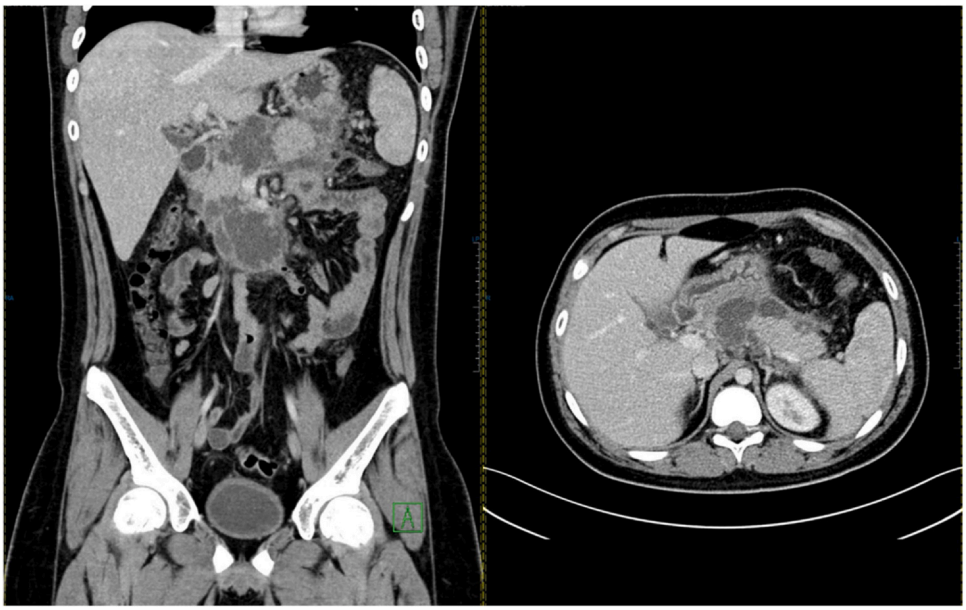

Fig. 3. Contrast-enhanced abdominal computed tomography (CT) with evidence of thrombosis involving branches of the anterior and posterior segments of the right portal vein.

Another contributing factor for thrombosis in patients with COVID-19 is hyperglycemia secondary to β cell destruction caused by SARS-CoV-2, which could lead to a new onset diabetes or sustained in-hospital hyperglycemia [10]. However, hyperglycemia and insulin resistance were found to be predisposing factors for prothrombotic state secondary to endothelial dysfunction, activation of coagulation and platelet hyperactivity, which increase the risk of thrombosis [11].

Taking into consideration that venous thromboembolism (VTE) is the most thrombotic complication in patients with COVID-19, Intra-abdominal thrombosis including portal vein and superior mesenteric vein thrombosis whish are unusual site thrombosis are rare in COVID-19 infection [12]. The pathophysiology of thromboembolism in COVID-19 may be more platelet-dependent and related to initial binding of SARS-CoV-2 to type II pneumocytes within the alveoli resulting in mass innate immune cells infiltration. Subsequent cytokine release, from these immune cells, contributes to a hypercoagulable state through various proposed mechanisms [8–13]. Portal vein thrombosis usually occurs in association with liver cirrhosis, hepatic malignancy or in patients with inherited or acquired thrombophilia, and it is rarely due to viral infection [14]. Additionally, portal vein thrombosis can present as a complication of severe pancreatitis particularly in cases with pancreatic necrosis. Intra-abdominal inflammation associated with acute pancreatitis may cause transient hypercoagulable state, but it is uncommon complication [15], with a reported incidence of 1–2% [16]. In a two-year,single institution retrospective study, a total of 20 (15.7%) patients out of 127 who were admitted with acute pancreatitis developed splanchnic vein thrombosis and all cases were observed in severe pancreatitis associated with pancreatic necrosis and peripancreatic collection. Furthermore, in these reported cases, splenic vein was the most commonly involved vessel followed by portal and the superior mesenteric veins. Moreover, there was a clear association between the site of necrosis, peripancreatic collection and the vessels thrombosed [17].

However, we are reporting this case due to its unique presentation with both acute necrotizing pancreatitis and portal vein thrombosis in association with COVID − 19 infection which has not been reported previously in the literature.

Conclusion

Acute necrotizing pancreatitis is a rare complication of COVID-19 and VTE is a well-known manifestation of it, however their co-occurrence has not been reported in the previous literature. A high clinical suspicion for unusual site thrombosis such as splanchnic vein thrombosis should be raised while investigating for gastrointestinal manifestations of SARS-COV 2 infection.

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