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Original Research Paper

Surgery

COVID-19 INDUCED PANCREATITIS, IN A CASE OF SITUS INVERSUS AND POLYSPLENIA

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ABSTRACT SARS-CoV-2 usually presents with respiratory system manifestations. Atypical presentations affecting the gastrointestinal and hepatobiliary systems can also occur. This is a case report of a 25 year old male with COVID-19 induced pancreatitis along with findings of situs inversus and polysplenia on imaging. COVID-19 infection, though classically affects the respiratory system, has a great potential to attack the pancreas. It is essential to have clinical and imaging evidence along with laboratory indicators, excluding all other known causes, for attributing COVID-19 as the trigger for acute pancreatitis.

KEYWORDS : COVID-19; Pancreatitis; Situs inversus; Polysplenia

INTRODUCTION

Coronavirus disease (COVID-19), discovered in Wuhan, China, has been declared a pandemic by the World Health Organization. The condition caused by Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2), usually presents with respiratory system manifestations. Atypical presentations affecting the gastrointestinal and hepatobiliary systems can also occur.[1,2] We report a rare case of SARS-CoV-2 induced pancreatitis with incidentally identified developmental anomalies.

Case Report

A 25-year-old male presented to the surgical emergency with complaints of abdominal pain and vomiting since 2 days. There was history of radiation of pain towards back region. He was a known case of pancreatitis, with previous acute episodes in 2011 and 2016. There was no history of alcoholism, smoking or drug abuse.

He had presented to the medical emergency 10 days ago, with generalized weakness, headache and loss of taste sensation and was diagnosed with SARS-CoV-2 infection by Real-Time Polymerase Chain Reaction (RT-PCR) test. He was then advised home isolation with standard care of treatment protocol, to which he had responded favorably within 4 days.

On examination, the patient was febrile, pulse rate was 86 / min, blood pressure of 120/80 mmHg and an oxygen saturation of 96% on room air. There was no evidence of guarding and rigidity over abdomen, but extreme tenderness in epigastric region. There was no evidence of organomegaly. Bowel sounds were sluggish. Laboratory analysis revealed normal blood counts, liver and renal function tests. Serum lipase was significantly elevated (>1400 U/L) and D-dimer was >4000 ng/ml. Serum amylase was mildly raised.

X ray chest revealed dextrocardia with normal lung fields, confirmed on 2D-echo study. Ultrasound study of abdomen revealed findings of situs inversus, along with hypoechoic echotexture of pancreas. Computed Tomography (CT) scan of the abdomen showed an edematous and bulky pancreas with significant peri-pancreatic fat stranding.

There were tiny foci of parenchymal calcifications in the head region and uncinate process. Pockets of fluid collection were evident in peri-pancreatic region along the transverse mesocolon (Fig.1, 2). The modified CT severity index was 4. The stomach was on the right side, duodenum and duodeno-jejunal junction on left, most of small bowel loops on left side, ileocaecal junction on right, with the appendix in the midline. Multiple spleniculi were noted in the right hypochondrium. CT Thorax confirmed findings of Dextrocardia (Fig. 3)

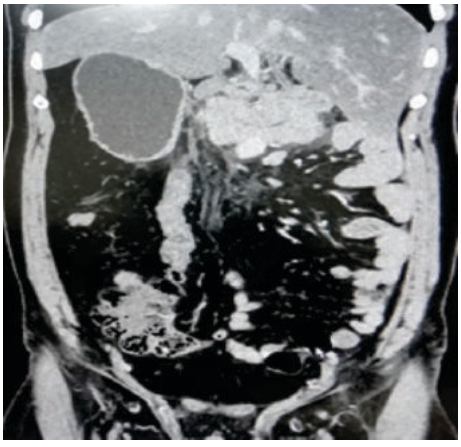

Fig.1: Post-contrast coronal image showing Liver on left side, Stomach on right side, central oedematous bulky Pancreas with mesenteric fat stranding

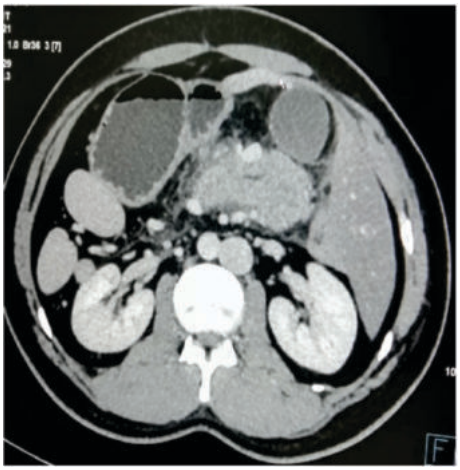

Fig.2: Axial post-contrast image showing Aorta on right side, Inferior Vena Cava on left side, with multiple Spleniculi on right side

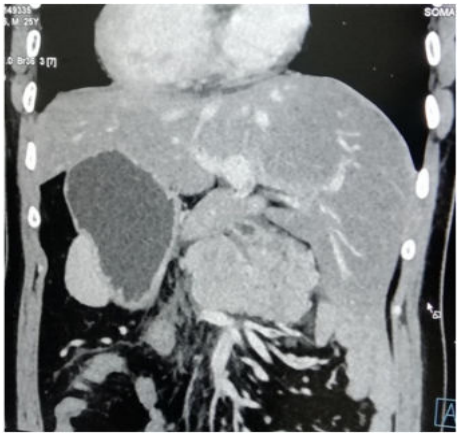

Fig. 3: Post-contrast coronal image showing apex of Heart on right side and left Liver lobe morphology on right side

He was admitted with a diagnosis of acute on chronic pancreatitis due to COVID-19 after ruling out other causes. He was kept nil by mouth and started with intravenous fluids, parenteral antibiotics (Cefoperazone + Sulbactum 1.5 gm TDS, Metronidazole 500 mg 8 hourly), analgesics (Inj Tramadol 50 mg BD, Diclofenac 2 cc IM BD), antacid (Inj Pantoprazole 40 mg BD) and anticoagulant therapy (Inj Enoxaparin 0.4cc SC BD). A further increase in serum lipase level (1405 ng /ml) was noted on second day of admission. Subsequently there was improvement in his clinical condition and was discharged from the hospital after 6 days, with normal laboratory investigations.

DISCUSSION

Though COVID-19 has been identified particularly as a respiratory infection manifesting as fever, cough and shortness of breath, various atypical presentations are being reported.[3] 50% cases in various studies have shown occurrence of gastrointestinal symptoms like vomiting, diarrhea and pain in abdomen.[4] Pancreatic involvement evident in form of deranged enzyme levels was noted in a study for 17.3% cases.[5] COVID-19 induced, clinically symptomatic pancreatitis is extremely rare. The revised Atlanta Criteria for diagnosis of acute pancreatitis include at least two out of three features: a) acute onset epigastric pain, usually radiating to the back), b) Serum Lipase or Amylase level> 3 times the upper limit of normal and c) imaging findings of acute pancreatitis.[6] Our case showed presence of all three features on the sixth day after detection of COVID-19 infection by RT-PCR test. All risk factors including alcohol, gall stone disease, use of steroids for COVID-19 infection, were absent in our case.

The host immune response is initiated by the glycosylated-spike (S) protein that is encoded by the coronavirus genome. This protein attaches itself to the Angiotensin-Converting Enzyme 2 (ACE2) receptors, which are present on the lung alveolar cells as well as on pancreatic islet cells.[7,8,9] Significant expression of these receptors on pancreatic islet cells help the virus to have cellular entry and exert a direct cytopathic effect. Li *et al*[10] have described the phenomenon of 'viral sepsis' comprising of an immune-mediated response, systemic inflammatory response and endothelial dysfunction. Meireles *et al*[11] noted that pancreatic injury was seen in COVID-19 patients in due course or during recovery of viral pneumonia, thereby postulating that pancreatic affection is due to an immune-mediated inflammatory reaction. Anand *et al*[12] have reported acute pancreatitis in a COVID-19 pneumonia recovered patient, five days after discharge, based on clinical and imaging findings. Studies from USA and Spain have reported a prevalence of 0.27% and 0.07% respectively for acute pancreatitis in hospitalized patients with COVID-19 infection.[13]

Situs inversus is a rare anomaly, with an incidence of 1 in 10,000 live births, in which the normal positions of the organs are reversed as a mirror image of the normal anatomy. Heterotaxy involves abnormal symmetry and malposition of the thoracic and abdominal organs and vasculature, including cardiac and extra-cardiac anomalies.[14] Polysplenia, initially described by Helwig in 1929, is defined as presence of more than one spleen and is found in 20% cases of situs inversus.[15] A case of acute pancreatitis in heterotaxy syndrome with polysplenia has been described by Kavuturu *et al*,[14] highlighting the vulnerability of these patients with anomalies of biliary tree, duodenum and / or pancreas for development of pancreas-related complications. Our case was a rare combination of COVID-19 infection induced pancreatitis along with situs inversus and polysplenia.

CONCLUSION

The case report brings to light, an emerging etiology for acute viral pancreatitis. COVID-19 infection, though classically affects the respiratory system, has a great potential to attack the pancreas. It is essential to have clinical and imaging evidence along with laboratory indicators, excluding all other known causes, for attributing COVID-19 as the trigger for acute pancreatitis.

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