ISSN: 0975-3583,0976-2833 VOL15, ISSUE 11, 2024

A POST COVID-19 ABDOMINAL CATASTROPHE

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ABSTRACT

COVID-19commonly manifests as a respiratory infection. However, several extrapulmonary complications have also been reported. Here, we report a case of post COVID-19 gastrointestinal complications in the form of acute pancreatitis and Ogilvie syndrome in the same patient which resulted in a catastrophic outcome with colonic perforation and peritonitis.

KEYWORDS

COVID-19, SARS-CoV-2, Ogilvie syndrome, acute pancreatitis

INTRODUCTION

The first confirmed case of COVID-19 was reported in December 2019 in Wuhan, China[1]. Since then, the world has seen its progression to a pandemic with over 774 million cases confirmed worldwide and over 7 million confirmed deaths as on October 2024. COVID-19 is caused by SARS-CoV-2, which is a highly transmissible and pathogenic novel beta coronavirus. It commonly manifests as a respiratory infection[2]. Common symptoms include fever, rhinitis, sore throat, cough and fatigue. Few cases progress to viral pneumonia with severe dyspnoea and lung infiltrates. However, significant extrapulmonary complications have been reported especially in severe COVID-19.

Gastrointestinal manifestations occur in up to 11.4- 61.1% of patients infected with SARS-CoV-2, and commonly present as anorexia, nausea, vomiting, diarrhoea, anosmia and dysgeusia[3].Complications such as acute liver injury, acute cholecystitis, paralytic ileus, acute pancreatitis, acute appendicitis, intestinal obstruction, mesenteric ischemia, hemoperitoneum or abdominal compartment syndrome have also been reported[4].

Here, we report two gastrointestinal complications of COVID-19, namely, acute pancreatitis and acute colonic pseudo-obstruction occurring concurrently in a patient, which have high rates of morbidity and mortality.

CASE REPORT

A twenty-eight-year-old pregnant woman (gravida 2, abortion 2) with 38 weeks of gestation presented with abdominal pain for five days. She had history of COVID-19, category B, three weeks ago which was managed with supportive care. Steroids or antivirals were not used during this period. At presentation, patient was conscious, oriented with mild diffuse abdominal tenderness. Her initial investigations showed mildly deranged liver function

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parameters.She had to undergo emergency lower segment Caesarean section for grade III meconium.

On post operative day 2, she was found to be tachypnoeic and had icterus, bilateral pedal oedema and a distended abdomen with absent bowel sounds. Repeat investigation reports revealed anaemia (haemoglobin- 9.1 g/dl), neutrophilic leucocytosis (total WBC count- 13, 900 cells/ mm^3, neutrophils- 82%, lymphocytes- 14%), deranged liver function parameters (total bilirubin- 8.09 mg/dl, direct bilirubin- 7.54 mg/dl, indirect bilirubin- 0.55 mg/dl, SGOT- 86 U/L, SGPT- 332 U/L, ALP-N318 U/L, total protein- 5 g/dl, albumin- 2.4g/dl, globulin- 2.6 g/dl, A/G ratio- 0.9, INR- 1.65) and elevated D- dimer (1000 mg/dl). Serum procalcitonin was mildly elevated- 2.1 ng/ml. Serum amylase and serum lipase were also elevated (767 U/L and 2698 U/L respectively). Ultrasonography of abdomen showed dilated, predominantly air filled small and large bowel loops till splenic flexure suggestive of colon cut off sign in acute pancreatitis, with moderate fluid in peritoneal cavity with diffuse bulky pancreatic parenchyma. Possibilities of post COVID-19 acute pancreatitis or an occult infection leading to sepsis and multiorgan dysfunction were considered. MIS-A, HELLP syndrome, cholestasis of pregnancy, acute fatty liver of pregnancy and mesenteric ischemia were also thought of. A contrast enhanced computed tomography scan (CECT) of abdomen was done which showed features of acute pancreatitis with modified CT severity index 6 and dilated predominantly air filled large and small bowel loops with no definite transition point, possibly intestinal pseudo-obstruction (Ogilvie syndrome).

Hence, a diagnosis of post COVID-19 acute pancreatitis and Ogilvie syndrome with impending bowel perforation was made. Patient was evaluated by surgical gastroenterologist and colonic decompression was doneusing neostigmine. Patient later developed recurrent episodes of fever. Blood culture yielded no growth. COVID-19 RT-PCR was negative. Echocardiography showed no evidence of infective endocarditis. A repeat CECT abdomen showed pneumoperitoneum with colonic perforation for which emergency exploratory laparotomy with peritoneal lavage and right hemicolectomy was done. Per operative findings included perforation in ascending colon, peritonitis with faecal contamination, hugely dilated entire colon with saponification in lesser sac and mesocolon. Empirical antibiotics were changed to tigecycline and meropenem. However, peritoneal fluid culture yielded growth of carbapenem resistant Klebsiella pneumoniae. Hence, patient was started on colistin. But patient had a further deteriorating clinical course leading to sepsis and multiorgan dysfunction and expired.

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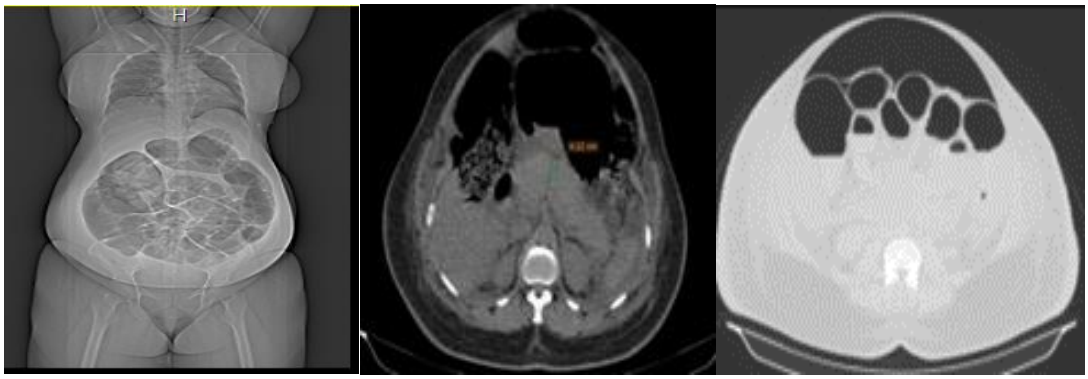

A B C

Figure 1A. Dilated air filled small and large bowel loops. 1B. Diffuse bulky pancreas. 1C. Pneumoperitoneum

A B C

Figure 2A. Dilated caecum and ascending colon. 2B. Perforation in ascending colon. 2C. Saponification of colonic serosal fat

DISCUSSION

COVID-19 is predominantly an airborne infection transmitted through respiratory droplets and aerosols[5]. SARS-CoV-2 mainly affects the respiratory system. Around 10-20% of infected patients progress to severe illness. However, COVID-19 is also found to produce gastrointestinal, neurological, cardiovascular, hepatic and dermatological manifestations. Gastrointestinal manifestations are often the initial or the only manifestations of the disease. In severe COVID-19, gastrointestinal complications can be encountered in the form of acute liver injury, acute cholecystitis, paralytic ileus, acute pancreatitis, acute appendicitis, intestinal obstruction, mesenteric ischemia, hemoperitoneum or abdominal compartment syndrome.

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Ogilvie syndrome, also known as acute colonic pseudo-obstruction is a functional disorder characterised by acute dilatation of the colon in the absence of obstructive anatomic lesions[6]. Risk factors include male gender, age over fifty years, critical illness, neurodegenerative diseases, post operative states, electrolyte abnormalities, viral infections and drugs[7].

SARS-CoV-2 enters type 2 alveolar cells of the lungs by binding to ACE-2 receptors using its spike proteins and then gets activated by human proteases[8]. Similarly, it uses the same receptors to bind to colonocytes, oesophageal keratinocytes, epithelial cells of stomach, ileum and rectum, exocrine glands and islets of pancreas leading to gastrointestinal manifestations[9]. Corroborative evidence includes detection of SARS-CoV-2 viral load in faecal samples of patients with COVID-19 presenting with gastrointestinal manifestations. SARS-CoV-2 viral ribonucleic acid (RNA) has also been detected in stools samples of COVID-19 patients even though respiratory samples showed presence of no infection. This also reveals the potential faecal-oral transmission of SARS-CoV-2[10].

Ogilvie's syndrome can be managed conservatively with electrolyte replacement, bowel rest and nasogastric suction. Patients who do not respond to these measures should undergo interventions like neostigmine (acetyl cholinesterase) therapy and colonic decompression. However, surgery may be required in certain cases

Acute pancreatitis is another gastrointestinal complication of COVID-19. The clinical manifestations of COVID-19 acute pancreatitis could be heterogeneous. Some patients may present with symptoms such as abdominal pain and vomiting at the beginning of SARS-CoV2 infection, while others may develop acute pancreatitis after several days of onset of infection. The severity of pancreatic injury is also strongly associated with the viral load and the multiple organ dysfunction in these patients[11]. SARS-CoV-2 causes pancreatic injury by its direct cytopathic effect as well as by causing indirect injury as a result of systemic immunological response leading to endothelial inflammation with micro-thrombosis[12]. A pooled prevalence of acute pancreatitis among patients with COVID-19 was found to be 3.1% according to a meta-analysis from China, USA and European countries[13]. However, the true prevalence is not known.

The pancreas expresses high levels of ACE2 receptor and transmembrane protease serine 2 (TMPRSS2), which facilitate the entry of SARS-CoV-2 into the islets as well as the exocrine cells of the pancreas[14].Other factors, such as dehydration,cytokine storm, and multiple organ failurecould also lead to development of acute pancreatitis in COVID-19 patients[15]. Many studies have demonstrated thatSARS-CoV2 infection causes the activation of the coagulation cascade, which is another pathogenetic mechanism leading to pancreatic injury[16]. COVID-19 also causes profound inflammation which results in endothelial dysfunction and platelet activation which lead to venous thromboembolism. According to a study from Warzecha et al.,the severity of COVID-19 acute pancreatitis strongly correlates with the hemostatic imbalance. It was demonstrated through experimental models that inhibition of the coagulation process decreased the severity of the acute pancreatitis[17].

CONCLUSION

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COVID-19 may present as a variety of gastrointestinal symptoms, ranging from mild to fatal complications which may precede or not be accompanied by respiratory symptoms. This case report highlights the importance of considering SARS-CoV-2 as an etiological agent of acute viral pancreatitis. It is also important to recognise Ogilvie syndrome as a potential complication of COVID-19 and promptly intervene to prevent bowel perforation and ischemia. Moreover, only very few cases of both these complications occurring together in a post COVID-19 patient has been reported.

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