Case Report

Acute pancreatitis in a patient with COVID-19: a case report

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Abstract: The global pandemic of coronavirus disease-2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is predominantly a respiratory illness, but gastrointestinal (GI) manifestations of variable severity have been reported. In patients with COVID-19 pneumonia, observational studies have demonstrated the elevation of pancreatic enzymes as surrogate markers for pancreatic injury without evidence of acute pancreatitis (AP). We report a case of AP in a patient with COVID-19 with SARS-CoV-2 as possible etiological agent with imaging evidence of pancreatitis. We hypothesize a causal relationship of SARS-CoV-2 in this patient with an otherwise unexplained presentation of AP after excluding the common causes. We postulate that AP in COVID-19 could be related to the abundant expression of angiotensin converting enzyme 2 (ACE 2) receptors in the pancreas which serve as viral entry binding receptors for SARS-CoV-2 or due to direct viral involvement of the pancreas. Although there seems to be an association between diabetes and AP, the available data regarding the etiological role of diabetes in causing AP is very limited. We also propose that imaging studies such as computerized tomography (CT) scan of the abdomen should be considered in the diagnosis of AP in patients with COVID-19 infection to exclude the false positive amylase and lipase.

Keywords: Acute pancreatitis (AP); coronavirus disease-2019 (COVID-19); severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2); structural damage; case report

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Introduction

Gastrointestinal (GI) manifestations of coronavirus disease-2019 (COVID-19) have been widely recognized, and they include ageusia, anorexia, nausea and vomiting, abdominal pain, diarrhea and elevated liver enzymes (1,2). The full clinical spectrum of this emerging disease, especially the involvement of the pancreas and other GI organs is still being studied and several aspects remain unknown. Earlier in the pandemic, there were some reports suggestive of pancreatic injury in patients with COVID-19 with indirect evidence of abnormal biochemical tests (elevated amylase and lipase) (3,4). While elevated amylase and lipase are suggestive of acute pancreatitis (AP), such elevations can occur in other conditions unrelated to AP raising the questions about accuracy of the diagnosis and over reporting of AP in these reports. Here, we report a case of AP with evidence of structural damage to the pancreas (imaging evidence) in a COVID-19 patient in addition to biochemical injury and without other known etiology of AP.

We present the following article in accordance with the CARE reporting checklist (available at http://dx.doi.org/10.21037/tgh-20-234).

Case presentation

A 74-year-old Caucasian woman with a medical history of type 2 diabetes mellitus (T2DM) and prior cholecystectomy

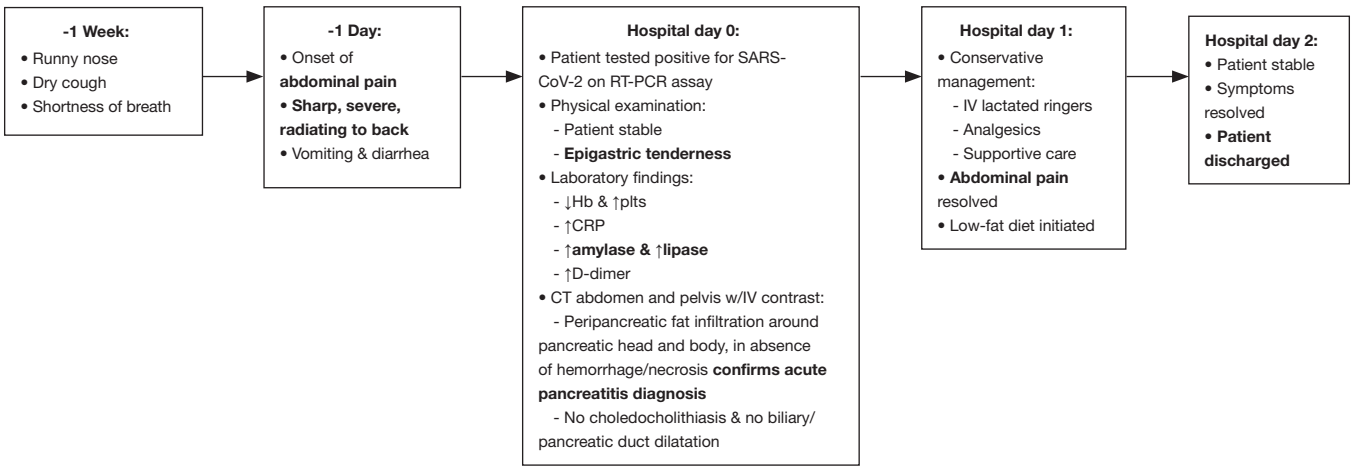

Figure 1 Case—AP in COVID-19 infection timeline. AP, acute pancreatitis; COVID-19, coronavirus disease-2019.

presented to the hospital with epigastric pain of 1-day duration. She described the pain as sharp, severe, radiating to the back, and associated with episodes of vomiting and diarrhea. She has no prior history of pancreatitis, does not consume alcohol or use recreational drugs. Her home medication regimen includes glimepiride, iron sulfate, aspirin, atorvastatin and esomeprazole. One week prior to the hospitalization, the patient developed symptoms of runny nose, dry cough and shortness of breath. She was tested positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on nasopharyngeal swab-based reverse transcriptase-polymerase chain reaction assay (RT-PCR). The timeline of this episode of care is shown in *Figure 1*.

In the emergency room, she had stable vital signs. Abdominal examination revealed epigastric tenderness. Laboratory tests showed hemoglobin of 8.4 g/dL, white blood cell count of 8,600 per cu mm, platelet count of 462,000 per cu mm, blood urea nitrogen of 23 mg/dL, creatinine of 0.75 mg/dL, serum calcium 8.2 mg/dL, amylase of 229 (normal, 30–151) U/L, and lipase of 7,550 (normal, 11–82) U/L. Serum glucose level was 112 g/dL with normal anion gap and normal serum bicarbonate level. Liver tests showed normal aspartate aminotransferase (AST) of 31 (normal, 14–36) U/L, alanine aminotransferase (ALT) of 24 (normal, 9–52) U/L, alkaline phosphatase of 74 (normal, 53–141) U/L and total bilirubin of 0.4 g/dL. D-dimer 1,608 (normal less than 211) ng/mL and C-reactive protein 34 (normal less than 5) mg/L were elevated. The serum triglyceride level was 188 (normal less than 150) mg/dL. Computerized tomography (CT) of the abdomen and pelvis with intravenous contrast demonstrated peripancreatic fat infiltration around the pancreatic head and body without hemorrhage or necrosis confirming the diagnosis of AP (*Figure 2*). There was no choledocholithiasis, biliary or pancreatic ductal dilatation. The patient was managed conservatively with intravenous lactated ringers, analgesics, and supportive care. On hospital day 1, the patient's abdominal pain resolved, and a low-fat diet was initiated. She was discharged home on hospital day 2 in a stable condition after resolution of her symptoms. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institution and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient.

Discussion

The presence of COVID-19 and AP in our patient could be a mere coincidence. However, in a recent case series of 52 hospitalized patients with COVID-19 pneumonia in a single center in China, 17% had biochemical evidence of pancreatic injury (elevated amylase and lipase) (4), but structural damage to the pancreas, such as edema, pancreatic necrosis, peripancreatic fat stranding or fluid collection, is not established as imaging studies were not available in this observational study. Thus, authors postulated that pancreatic injury in patients with COVID-19 pneumonia could be due to direct viral involvement of the pancreas or from secondary enzyme abnormalities due to severe systemic illness without substantial pancreatic injury. Furthermore, it is established that certain viral illnesses

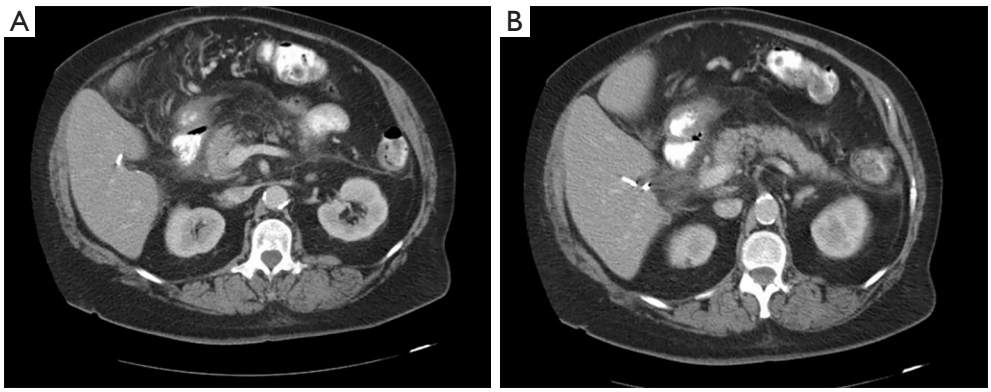

Figure 2 Cross sectional images of contrast enhanced computerized tomography (CT) scan of the abdomen demonstrating peri pancreatic fat stranding (A) and pancreatitis of head and body (B).

such as mumps, coxsackie, Epstein-Barr virus, hepatitis A and E are known to cause AP (5,6). While it is difficult to establish the etiological role of SARS-CoV-2 in AP in our patient, prior studies have demonstrated the expression of angiotensin converting enzyme 2 (ACE 2) in the pancreatic tissue, which also serve as viral entry binding receptors for SARS-CoV-2 (7,8). In a recent case report of a patient with severe COVID-19 pneumonia with ARDS, authors reported AP based on elevated lipase and epigastric pain (3). However, the imaging evidence of AP was absent in this case whereas our patient had clear changes of AP with peripancreatic fat stranding on CT scan. In another case, although authors reported a case of AP in a 24-year-old patient with COVID-19, the possibility of biliary pancreatitis could not be excluded especially in the absence of liver test results (9). Similar cases have been reported recently with COVID-19 as a potential cause of AP, however our case is unique because of our patient had prior cholecystectomy and normal liver enzymes which eliminate possibility of biliary pancreatitis beyond reasonable doubt (10-12).

Some observational studies have reported modest increase in incidence of AP in diabetic patients. In a large population based nested case control study, incidence of AP in T2DM was 54.0 per 100,000 person-years compared to 30.1 in general population (13). The adjusted incidence ratio of AP in this study was 1.77 (95% CI: 1.46–2.15) in T2DM patients compared to general population. However, after adjusting demographic and lifestyle variables, drug exposure and comorbidities, overall risk of AP in T2DM patients was non-significant (adjusted OR 1.37, 95% CI: 0.99–1.89). Several reasons for increased AP in T2DM have been postulated such as increasing incidence of obesity perhaps causing increased gallstones or role of some anti-diabetic medications especially glucagon like peptide-1 (GLP-1) analogues. In this study, although there was increased risk of AP with increasing age in overall cohort, there was significant trend towards decreasing association between AP and diabetes and increasing age (P=0.019). Association between hyperglycemia, diabetic ketoacidosis (DKA) and AP has been previously reported perhaps due to severe underlying acidemia causing pancreatic acinar cell injury (14). In our elderly diabetic patient, in the absence of DKA, culpable anti-diabetic medications and no clear association between increasing age and diabetes with AP, etiological role of T2DM in AP is highly unlikely.

In conclusion, it is unclear whether AP is a coincidence or consequence of COVID-19 infection. However, in the absence of alternative etiology and due to the aforementioned factors, the role of COVID-19 in causing AP should be considered. AP can be a rare manifestation of pancreatic cancer (15). In certain cases, endoscopic ultrasound may be required to rule out a small tumor, however, in our patient, contrast enhanced CT scan did not show pancreatic cancer. Many aspects of COVID-19 are still being investigated, and larger case series or cohort studies in future may provide insight into the etiological role of SARS-CoV-2 in AP. Nevertheless, it appears that AP could be one among the myriad of GI manifestations caused by COVID-19 albeit less common. Available evidence is meager regarding association of COVID-19 infection, AP and T2DM in the absence of DKA. Nevertheless, factors such as older age and T2DM might have contributed

to the development of AP in the setting of COVID-19 infection, which is impossible to establish in a single case. However, we recommend high index of suspicion for AP in older COVID-19 patients presenting with abdominal pain especially with underlying T2DM. Due to the non-specific nature of elevated amylase and lipase in the diagnosis of AP, we propose that cross sectional imaging studies should be considered to confirm the diagnosis of AP in patients with COVID-19 infection.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at http://dx.doi.org/10.21037/tgh-20-234

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/tgh-20-234). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institution and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient.

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