CLINICAL VIGNETTE

A Case of COVID-19 Presenting as Acute Pancreatitis

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Introduction

Coronavirus disease-2019 (COVID-19), which is due to infection with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has been found to cause a myriad of symptoms and multi-organ complications. We present a patient who presented primarily with gastrointestinal symptoms, and developed acute pancreatitis in the setting of newly diagnosed SARS-CoV-2 infection.

Case Presentation

A 72-year-old female presented to the emergency department with a two-day history of intractable nausea, vomiting, diarrhea, and abdominal pain. She had past medical history significant for hypertension, hyperlipidemia, gout, chronic kidney disease (unknown baseline renal function), and hypopituitarism on chronic thyroid and steroid replacement. The patient reported that she was in her usual state of health when she developed sharp, mid-epigastric, non-radiating abdominal pain, worsened when sitting up in a chair. She also complained of nausea with non-bloody, non-bilious emesis, as well as non-bloody, watery diarrhea. She denied any new foods recent travel or sick contacts. Her home medications were allopurinol, amlodipine, hydralazine, losartan, levothyroxine, and prednisone, which she had been taking for many years. She denied tobacco, alcohol, or recreational drug use. The patient also noted a new mild non-productive cough during the onset of her gastrointestinal symptoms, but denied any fever, chest pain, or shortness of breath. Because of the COVID-19 pandemic, she was following public health guidelines of staying at home. She lived with her daughter who left their home only for essential trips.

In the emergency department, the patient's initial vitals: Temperature 39.4ºF, Blood Pressure 139/51, Heart Rate 97, Respiratory Rate 26, and Oxygen Saturation of 88% on room air, with improvement with 2L supplemental oxygen. She did not have labored breathing and her physical exam was significant for tenderness to palpation in the mid-epigastrum. Initial laboratory results were notable for creatinine 2.4 mg/dL, white blood cell 11.7 g/dL, lipase 436 U/L, lactate 1.2 mmol/L, lactate dehydrogenase 857 U/L, aspartate transaminase 93 U/L, alanine transaminase 53 U/L, total bilirubin 0.5 mg/dL, direct bilirubin 0.1 mg/dL, procalcitonin 0.79 ng/mL, thyroid stimulating hormone 0.4 mIU/mL, free T4 0.3 ng/dL, and C-reactive protein 128 mg/L. CXR showed bibasilar atelectasis without focal consolidation or opacities. CT Abdomen/Pelvis showed nodular opacities in the lung bases concerning for an infectious or inflammatory process and a 14-mm low-density focus in the pancreas representing either a small pancreatic cyst, IPMN, or low-grade cystic neoplasm. There were other radiographic findings to explain the patient's acute abdominal pain. Blood cultures were obtained, and the patient was started on ceftriaxone and azithromycin for the pulmonary opacities seen on imaging. Metronidazole was added for possible intra-abdominal infection. Subsequent CT Chest showed bilateral lower lobe peripheral opacities concerning for a viral pneumonia such as COVID-19. A SARS-CoV-2 PCR test was ordered and she was admitted to the hospitalist service for further management.

The patient was initially kept NPO given the acute pancreatitis. Intravenous fluids were given temporarily and cautiously given the concern for COVID-19 pneumonia. A right upper quadrant ultrasound was done to further visualize the gallbladder and rule out possible gallstone pancreatitis. However, no cholelithiasis was found, and the biliary system was unremarkable. Lipid panel returned with triglycerides mildly elevated at 184 mg/dL, thus making hypertriglyceridemia a less likely etiology of the acute pancreatitis. Her diet was eventually advanced to a clear liquid diet, though she still did not have much of an appetite. She continued to have intermittent fevers, diarrhea, and non-productive cough, but her diarrhea appeared to have slightly improved in frequency. Clostridium difficile test was negative. On hospital day 2, the SARS-CoV-2 test returned positive, and the patient and her daughter were made aware of the results. Per the daughter, no one else at home had symptoms, but they were instructed to quarantine at home per public health guidelines. On this same day, the patient's oxygen requirement increased from 2L to 4L nasal cannula due to persistent hypoxia, but she denied any respiratory distress. The ICU team was made aware of the patient in case the hypoxia continued to worsen. On hospital day 4, the patient's oxygen requirements increased to 5L NC, but she otherwise remained hemodynamically stable. Furosemide was given intermittently for diuresis, given the patient had previously received intravenous fluids for the acute pancreatitis. On hospital day 5. She was transferred to the Intensive Care Unit for hypoxia despite 6L oxygen for high-flow nasal cannula (HFNC). Later that evening, the patient's hypoxia worsened significantly despite maximum support on HFNC, and she was intubated and placed on mechanical ventilation. The patient's condition continued to worsen, and on hospital day 6, the patient went into septic shock requiring vasopressor support and escalation to broad-spectrum anti-

biotics, as well as worsening renal failure requiring initiation of dialysis. Stress-dose steroids were also administered given the history of hypopituitarism. On hospital day 7, the ICU team spoke to the patient's family about her guarded prognosis, and decision was made to continue aggressive medical management but not to perform cardiac resuscitative measures if she went into cardiac arrest. The patient's condition continued to decline despite maximum ventilator settings, and ultimately the patient expired.

Discussion

COVID-19 was first declared a pandemic by the World Health Organization on March 11, 2020. There have been over 90 million cases confirmed worldwide and over 1.9 million deaths.[1] COVID-19 has been found to cause a myriad of symptoms as well as multi-organ complications, such as acute respiratory distress syndrome, myocardial injury, and acute renal failure.

Gastrointestinal disease, such as acute liver injury, has also been well documented. There have been several case reports of COVID-19 presenting as acute pancreatitis in patients who would otherwise have no other risk factors for pancreatic injury. In a case series of 52 patients hospitalized with COVID-19, 17% were found to have evidence of acute pancreatic injury, defined by abnormal elevation in amylase or lipase.[2] These patients had more severe COVID-19, but compared to patients without evidence of pancreatitis, there was no difference in rate of progression of disease requiring mechanical ventilation. The patient in the above case presentation did unfortunately go on to develop critical illness that required mechanical ventilation.

There are several explanations of the etiology of pancreatic injury in COVID-19, and one potential etiology is perhaps related to direct viral-induced injury. SARS-CoV-2, the virus that causes COVID-19, is known to bind to angiotensin-converting enzyme-2 (ACE2) receptors on host cells via its spike protein. ACE2 receptors are known to be highly expressed in various organs, including pancreatic islet cells.[3] Damage to the pancreas can lead to complications such as acute respiratory distress syndrome, which is already seen in severe cases of COVID-19. This may explain why patients who had evidence of pancreatitis were found to have more severe disease in this case series.

Our patient had no other risk factors for acute pancreatitis. There was no alcohol use, no cholelithiasis on imaging, and triglyceride level was also not significantly elevated. She was taking losartan at home which has been implicated in acute pancreatitis, but she had been on this medication for many years. She did have evidence of a pancreatic cyst or possible pancreatic neoplasm, but it is unlikely that these entities would cause acute pancreatic injury. Thus, we suspect that acute pancreatitis in this patient was due to COVID-19.

Conclusion

COVID-19 has been found to present in a variety of ways. Though most patients present with flu-like symptoms such as fever and cough, patients may present with primarily gastro-intestinal symptoms. Patient presenting with gastrointestinal symptoms with laboratory evidence of acute pancreatitis without risk factors, should consider SARS-CoV-2 infection.

REFERENCES

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