Hyperglycemia, Hypertriglyceridemia, and Acute Pancreatitis in COVID-19 Infection
Clinical Implications

To the Editor:

Coronavirus disease 2019 (COVID-19), an emerging disease of variable severity caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is primarily a respiratory illness, but manifestations vary widely, affecting multiple organ systems resulting in management dilemmas.[1] Many aspects of the disease are just being studied. We report a case of severe acute pancreatitis (AP) in association with hypertriglyceridemia and COVID-19 infection and discuss clinical implications.

A 40-year-old obese (body mass index, 38.8 kg/m^2) male patient with no known comorbidity was admitted to the hospital with a 2-day history of severe epigastric pain radiating to back. Oxygen saturation on admission was 96% on room air. Laboratory tests showed blood glucose of 288 mg/dL, elevated serum lipase of 1544 U/L (reference range, 1–82 U/L), serum bicarbonate of 21 mmol/L (reference range, 21–33 mmol/L), and an anion gap of 14. A single view plain chest radiograph showed no acute cardiopulmonary process. Contrast-enhanced computed tomography scan of the abdomen and pelvis showed extensive peripancreatic stranding and fluid around the head and tail of the pancreas, confirming the diagnosis of AP. The patient did not consume alcohol regularly, and transabdominal ultrasound did not show cholelithiasis or choledocholithiasis. The serum triglyceride level was 4245 mg/dL (reference range, <150 mg/dL), and hemoglobin A1C was 9.7%. A diagnosis of new-onset type 2 diabetes mellitus (T2DM) and hypertriglyceridemic AP was made based on our previous reports.[2,3] Fluid resuscitation with lactated ringers at 200 mL per hour was initiated for the treatment of AP as per management recommendations of the American College of Gastroenterology.[4] The patient was admitted to the intensive care unit, and intravenous insulin infusion was given for the management of hypertriglyceridemia.

On intensive care unit day 1, the patient developed a fever and acute hypoxic respiratory failure requiring incremental amounts of supplemental oxygen therapy. Because of the respiratory failure amid the COVID pandemic, a nasopharyngeal swab for COVID-19 was done using real-time polymerase chain reaction assay for SARS-CoV-2, which returned positive. Other significant nonspecific laboratory markers for COVID-19 in this patient included serum ferritin of 1338 ng/mL (reference range, 18–464 ng/mL), lactate dehydrogenase of 502 U/L (reference range, 140–271 U/L), C-reactive protein of 300 mg/L (reference range, <5 mg/L), and D-dimer of 2697 ng/mL (reference range, <211 ng/mL). Bilateral venous Doppler ultrasound of lower extremities was negative for deep vein thrombosis. Because of worsening respiratory failure, despite 100% FiO2 via high-flow nasal cannula of 40 L/minute, support was escalated to noninvasive positive pressure ventilation using bilevel positive airway pressure machine. Fluid administration was stopped, and diuresis with intravenous boluses of furosemide was initiated. With these measures, the respiratory status improved and the patient was successfully transitioned to a nasal cannula oxygen. On hospital day 6, he was discharged home after resolution of hypoxemia and improvement of AP.

Whether COVID-19 played a role in the etiology of AP or a mere coincidence in a patient with a preexisting etiology for pancreatitis is debatable as the literature is meager. Recent report from China showed that up to 17% of hospitalized patients with COVID-19 had evidence of some pancreatic injury (elevated amylase and lipase) and hyperglycemia postulated to be due to β cell injury or as result of severe systemic illness.[5] In another report from South Korea of 2 patients, COVID-19 infection was implicated in severe acute hyperglycemic crises including diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state.[6]

In a case series of 100 patients with DKA, we previously reported the relationship between hyperglycemia, DKA, and AP.[2] Acidosis of any etiology is known to cause acinar cell injury, but in our patient, it was not an issue. In experimental studies, the extent of the acidosis is a major risk factor for AP.[6] Hypertriglyceridemia is a well-established etiology for AP and is typically seen in the setting of severe hyperglycemia.[7] A pathogenetic role for COVID is not clear. A mere coincidence of T2DM, hypertriglyceridemia, and COVID-19 cannot be excluded. To our knowledge, this is the first case of hypertriglyceridemic AP in the setting of COVID-19 infection.

The American College of Gastroenterology guidelines for AP recommend aggressive intravenous isotonic crystalloids administration of 250 to 500 mL per hour in the first 12 to 24 hours unless there are cardiovascular or renal comorbidities.[4] On the other hand, pulmonary manifestations of COVID-19 infection are similar to acute respiratory distress syndrome. Hence, the Society of Critical Care Medicine recommends the conservative rather than liberal fluid administration even in the setting of shock.[7] Uncontrolled T2DM and hypertriglyceridemia may be the consequence or coincidence in COVID-19 infection. Fluid replacement is a delicate issue and requires very close scrutiny of the respiratory status in patients with AP in the setting of COVID-19 infection.

ACKNOWLEDGMENT

The authors declare no conflict of interest.

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