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Case Report: Acute Abdominal Pain as Presentation of Pneumonia and Acute Pancreatitis in a Pediatric Patient With COVID-19

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Abstract: Abdominal pain, nausea, and vomiting are known gastrointestinal symptoms of symptomatic SARS-CoV-2 infection (COVID-19 disease) in pediatric patients.[1] There is little literature regarding pancreatitis in COVID-19. We describe a 16-year-old male diagnosed with acute pancreatitis in the setting of a SARS-COV-2 infection and associated fluid balance considerations.

Key Words: SARS-CoV-2, acute pancreatitis, pediatrics

An estimated 4% of SARS-CoV-2 patients are less than 18 years old in the United States as of April 2, 2020.[2] In an Italian cohort of 100 children, 10% presented with nausea or vomiting, 9% with diarrhea, and 4% with abdominal pain.[1] SARS-CoV-2 RNA has been detected in the stool, indicating active disease, gastrointestinal shedding, or both.[3]

Acute pancreatitis (AP) in COVID-19 patients is not well described. A retrospective study from the Zhongnan hospital in Wuhan, China, reported 17% of COVID-19 patients had elevated amylase or lipase.[4] A case series from Denmark reported 2 of 3 family members with COVID-19 then developing severe AP.[5] Other case reports describing AP and COVID-19 include a 59-year-old patient, and 3 different 36-year-old females, one of whom was pregnant.[6–9] Our case involves a 16-year-old male who was admitted to the hospital with bibasilar pneumonia, AP, COVID-19, and concern for multisystem inflammatory syndrome. Cross-sectional imaging supports the diagnosis of AP in this patient.

CASE REPORT

A 16-year-old (weight 91.8 kg; BMI = 31 kg/m^2) obese Hispanic male without previous medical problems presented with 3 days of nausea, nonbloody, nonbilious emesis, and epigastric pain. No previous fevers, respiratory symptoms, sick contacts, or alcohol use. Vital signs revealed fever (100.6 F), tachycardia (110/min), tachypnea (28/min), and borderline oxygen saturation (SpO_2 93%). He appeared ill, lungs were clear with diminished breath sounds at the bases. Abdomen was soft but tender to light palpation, worse in the epigastrium. Admission labs (Table 1) showed elevated lipase >4× the upper limit of normal. Nasal swab for SARS-CoV-2 utilizing reverse transcriptase PCR methodology was positive, done as part of routine screening. Abdominal ultrasound did not visualize the pancreas. CT scan of abdomen/pelvis demonstrated findings consistent with AP (peripancreatic and retroperitoneal edema without necrosis) as well as peripheral ground glass and nodular opacities in the lung bases, Figure 1. His CT scan of chest also demonstrated the bilateral lung consolidations (Figure, Supplemental Digital Content 1, http://links.lww.com/PG9/A2). He was diagnosed with AP (epigastric abdominal pain, elevated lipase, and CT scan findings consistent with AP), COVID-19, and pneumonia and admitted to the pediatric gastroenterology service.

Initial management included intravenous fluid support with a 1 L 0.9% normal saline (NS) bolus followed by 0.9% NS at 150 mL/hr, pain management, and no dietary restriction. He was placed on 1 L/min supplemental oxygen. Infectious disease was consulted to assist with evaluation and management of COVID-19 and pneumonia. Infectious work up was negative for an alternative etiology for his pneumonia (Table, Supplemental Digital Content 1, http://links.lww.com/PG9/A3); therefore, SARS-CoV-2 was the suspected etiology of his pneumonia. He had no previous history of pancreatitis and no identified risk-factors including medication exposure, family history, trauma, alcohol, or gallstones.

Over 24 hours, his urine output was noted to be poor (UOP = 0.17 mL/kg/hr). Further evaluation (Table 1) revealed a high-urine specific gravity, evidence of acute kidney injury (AKI), mild hypoalbuminemia, and elevated inflammatory markers. Examination revealed tachycardia (121/min), tachypnea (29/min), and SpO_2 92% on 2L nasal cannula. His weight was up 16.2 kg (obtained by 2 scales, second by bed scale). His fluid balance was 3.9 L positive. Lungs had rales in bases bilaterally and bilateral pedal edema. His abdomen remained tender to light palpation and felt full. Chest x-ray demonstrated increased bibasilar opacities and suspected trace left pleural effusion. Repeat CT scan of abdomen/pelvis revealed new peripancreatic fluid collections with continued pancreatic and retroperitoneal edema, Figure 2. He was diagnosed with fluid overload with third spacing secondary to capillary leak causing pulmonary edema and AKI. He required oxygen of 3L/min by cannula. He was given several doses of 25% albumin followed by intravenous furosemide with an increase in urine output.

He was monitored closely for progression to multisystem inflammatory syndrome in children (MIS-C). He was started on intravenous remdesivir for acute treatment of COVID-19 in view of pulse-oximetry <94% and supplemental oxygen requirement as a part of compassionate use from Gilead Sciences, Inc. (Foster City, CA). He received 6 days of treatment and improved. His weight at discharge was 8.5 kg over admission weight (obtained via bed scale).

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TABLE 1. Laboratory Evaluation

	Day of Admission	Upon Clinical Worsening 17 Hours After Admission	Inflammatory Markers—Obtained Within 24 Hours of Admission	
BUN (mg/dL)	16	22	C-reactive protein (mg/dL)	36.4
Creatinine (mg/dL)	0.94	1.1	Erythrocyte sedimentation rate (mm/hr)	24
Glucose (mg/dL)	185	130	Ferritin	375
Calcium (mg/dL)	8.4	8.7	Fibrinogen (mg/dL)	623
Total protein (g/dL)	6.9	6.4	D-dimer (mcg/mL)	7.60
Albumin (g/dL)	3.5	3.1	IL-10 (pg/mL)	6.0 (<2.8)
AST (U/L)	43	39	IL-6 (pg/mL)—obtained 36 hours after admission	31.6 (<2.0)
ALT (U/L)	63	54		
Alkaline phosphatase (U/L)	87	83		
Total bilirubin (mg/dL)	1.3	1.9		
Direct bilirubin (mg/dL)	0.6	0.9		
Lipase	961			
GGT (U/L)		197		
CBC				
Hemoglobin (g/dL)	16.4	12.5		
Hematocrit (%)	47.4	37.3		
Platelets (per mm^3)	291	234		
WBC (per mm^3)	7.91	13.0		
Lymphocytes	38.1%	2.8%		
Urine Specific Gravity		1.039		

ALT indicates alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CBC, complete blood count; GGT, gamma-glutamyl transferase.

DISCUSSION

To date, the relationship between SARS-CoV-2 infection and AP is unclear. Our case demonstrates a pediatric patient with COVID-19, AP, pneumonia, and AKI providing emphasis on fluid management. The standard of care for AP in pediatrics includes pain control and crystalloid intravenous fluid repletion at 1.5–2× maintenance rate.[10] However, recently published guidelines for critically ill adults with COVID-19, conservative fluid strategy is recommended.[11] This suggests that patients with AP and COVID-19 need thoughtful decision-making regarding fluid choices and monitoring of their fluid balance.

AP can lead to systemic inflammatory response syndrome (SIRS) thus increasing the risk for capillary permeability.[12,13] COVID-19 is a highly inflammatory disease. In mouse models, SARS-CoV-2 enters cells by attaching to angiotensin converting enzyme-2 (ACE-2) receptor. Upon cell entry, it downregulates ACE-2 expression leading to elevated levels of angiotensin-2, which contributes to lung inflammation by stimulating NADPH oxidase activity and IL-17A production from T cells.[14,15] This process increases pulmonary vascular permeability thus increasing the risk for capillary leak.[16,17]

Our patient's acute, profound weight gain was disproportionate to the amount of fluid he received. We hypothesize that the

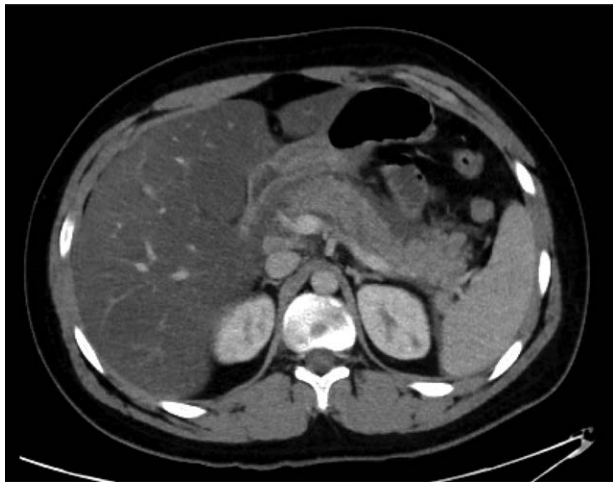

FIGURE 1. Initial CT scan findings demonstrating acute pancreatitis with peripancreatic and retroperitoneal edema. No evidence of necrosis or vascular complication.

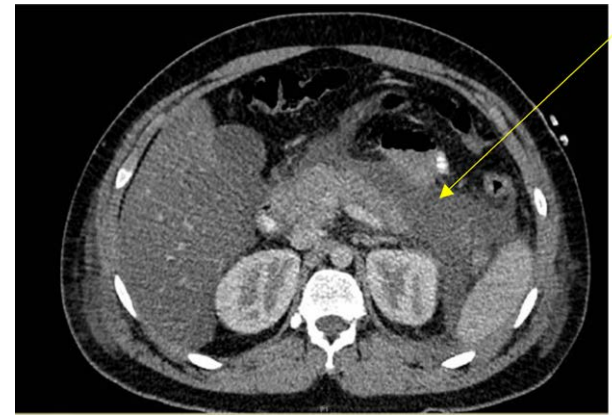

FIGURE 2. CT scan findings 2 days later demonstrating peripancreatic edema, fat stranding, peripancreatic fluid collection medial to spleen. Arrow demonstrating acute peripancreatic fluid collection.

combination of AP and COVID-19 increased his risk for developing capillary leak and volume overload. Notably, the risk of volume overload was likely heightened by AKI in his case.

It is unknown if AP is directly caused by SARS-CoV-2 or if it is a consequence of an inflammatory cascade triggered by the virus. ACE2 receptors are expressed in pancreatic islet cells, where the expression may be higher than in alveolar epithelial cells suggesting a mechanism that SARS-CoV-2 may induce pancreatic damage.[18] Alternatively, AP may be secondary to the profound systemic inflammatory response and cytokine storm that occurs in SARS-CoV-2 infection. Our patient had biochemical evidence of significant systemic inflammation with elevations in ESR, CRP, D-dimer, fibrinogen, IL-6, and IL-10. Elevations in these parameters along with pulmonary, pancreatic, and renal dysfunction meet the CDC case definition of MIS-C.

This case highlights the need for prudent evaluation of patients with COVID-19 for the possibility of AP. This is the first reported pediatric case of a patient with symptomatic COVID-19 manifested by fever, oxygen desaturations, and pneumonia, with true radiographic pancreatic findings consistent with AP. This demonstrates the need for investigation into the relationship between SARS-CoV-2 infection and AP and the unique fluid balance considerations for these patients.

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