



Case Report: Acute Necrotizing Pancreatitis Following SARS-CoV-2 Infection

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Abstract

Acute pancreatitis is a well-recognized clinical entity, but necrotizing pancreatitis in the absence of known risk factors such as alcohol use, gallstones, or hypertriglyceridemia is rare. With emerging evidence of pancreatic involvement in SARS-CoV-2 infections, this case highlights a unique presentation of acute necrotizing pancreatitis in a previously healthy, non-comorbid individual with recent COVID-19 positivity.

Keywords: SARS-CoV-2, COVID-19, Acute Pancreatitis, Necrotizing Pancreatitis, Pleural Effusion

Introduction

SARS-CoV-2, the virus responsible for COVID-19, has been associated with multi-organ involvement beyond the lungs, including gastrointestinal and pancreatic systems. Pancreatic injury is infrequent and necrotizing pancreatitis even more so, especially in the absence of traditional risk factors. This case outlines such a rare presentation, adding to the growing body of evidence suggesting SARS-CoV-2 may play a direct or immune-mediated role in pancreatic pathology.

Case Presentation

A 45-year-old male, a professional painter, with no prior comorbidities or significant past medical history,

presented with severe epigastric pain and vomiting for one day. The pain was sudden in onset, persistent, piercing in nature, and radiated to the back. It was aggravated by food intake and partially relieved by bending forward. He reported 8-10 episodes of non-projectile vomiting, initially containing food particles and later bile-stained.

There was no history of alcohol use, recent trauma, drug intake, hypercalcemia, or prior similar episodes. Examination revealed stable vitals and epigastric tenderness. Laboratory investigations showed elevated serum amylase (854 U/L), lipase (479 U/L), and CRP (120 mg/L). USG revealed a bulky pancreas and mild free fluid. He was diagnosed with acute pancreatitis and managed conservatively. Ranson's score was 3 and BISAP score was 3.

Further workup revealed no gallstones, normal triglycerides, GGT, and calcium. Triple serology was negative but SARS-CoV-2 PCR was positive.

On day two, he developed mild respiratory distress with bilateral pleural effusion (left > right). Pleural fluid analysis was exudative and negative for infection or malignancy. CECT showed necrotizing pancreatitis (CTSI 6/10) with ascites and bilateral effusion. He improved with conservative treatment. Repeat CRP was 62, serum amylase 39, and lipase 38.

Discussion

The absence of classical risk factors and the temporal association with COVID-19 infection suggests a possible viral etiology in this case of necrotizing pancreatitis. SARS-CoV-2 binds to ACE2 receptors, which are expressed in pancreatic cells, leading to direct or indirect injury. Similar cases have been reported, but necrotizing transformation remains uncommon. Pleural effusion was minimal and sterile. Conservative management yielded

successful resolution without the need for ICU admission or interventions.

Conclusion

Acute necrotizing pancreatitis should be considered in patients presenting with epigastric pain post-COVID, especially in the absence of typical risk factors. Early diagnosis, supportive care, and thorough etiological evaluation are critical to prevent complications and improve outcomes.

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