

# Acute pancreatitis in SARS-CoV-2 infection: A case report from Tanzania

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## Abstract

The pandemic caused by the severe acute respiratory syndrome coronavirus 2 has mainly affected the respiratory system but has expanded to other systems, including the gastrointestinal system. We present an 80-year-old man with sharp epigastric pain and vomiting. Laboratory investigations revealed elevated pancreatic enzymes, and contrast-enhanced computed tomography of the abdomen suggested acute pancreatitis. He was undergoing treatment for acute pancreatitis when he developed respiratory compromise, leading to the use of oxygen. Computed tomography of the chest revealed bilateral pleural effusion. However, a positive nasopharyngeal swab suggested severe acute respiratory syndrome coronavirus 2 infection. He was treated for the viral infection with various medications until clinically stable before being self-isolated at home. His follow-up visits revealed a favorable outcome, with progressive resolve occurring 4 weeks after the onset. There is no specific conclusion regarding pancreatic involvement in severe acute respiratory syndrome coronavirus 2 infection. There are several confounding factors in the etiology of acute pancreatitis during concomitant severe acute respiratory syndrome coronavirus 2 infection. However, further research is warranted to evaluate whether pancreatic involvement is one of the clinical presentations or subsequent complications of severe acute respiratory syndrome coronavirus 2 infection.

## Keywords

COVID-19, SARS-CoV-2, acute pancreatitis, Tanzania

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## Introduction

The pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has allowed for significant literature publication regarding the clinical features and management of the disease.<sup>1</sup> Despite more frequent respiratory complaints in SARS-CoV-2, there has been a wide association of gastrointestinal symptoms in up to 79% of patients.<sup>2</sup> Multiple cases of acute pancreatitis and pancreatic injury caused by SARS-CoV-2 have been reported. Around 2% of non-severe and 17% of severe cases of SARS-CoV-2 show pancreatic injury, which may develop before the patient's admission.<sup>3</sup> We present an elderly male patient with SARS-CoV-2 infection who presented with epigastric pain and was diagnosed with acute pancreatitis.

## Case presentation

An 80-year-old male of Asian background presented to the hospital with sharp, non-radiating, epigastric pain for 1 day,

associated with nausea and vomiting. His medical history was positive for hypertension, hypothyroidism, and ischemic heart disease, with post-coronary artery stenting 10 years before admission. He denied a history of fever and chills. He reported no history of alcohol consumption or cigarette smoking. His regular oral medications were olmesartan

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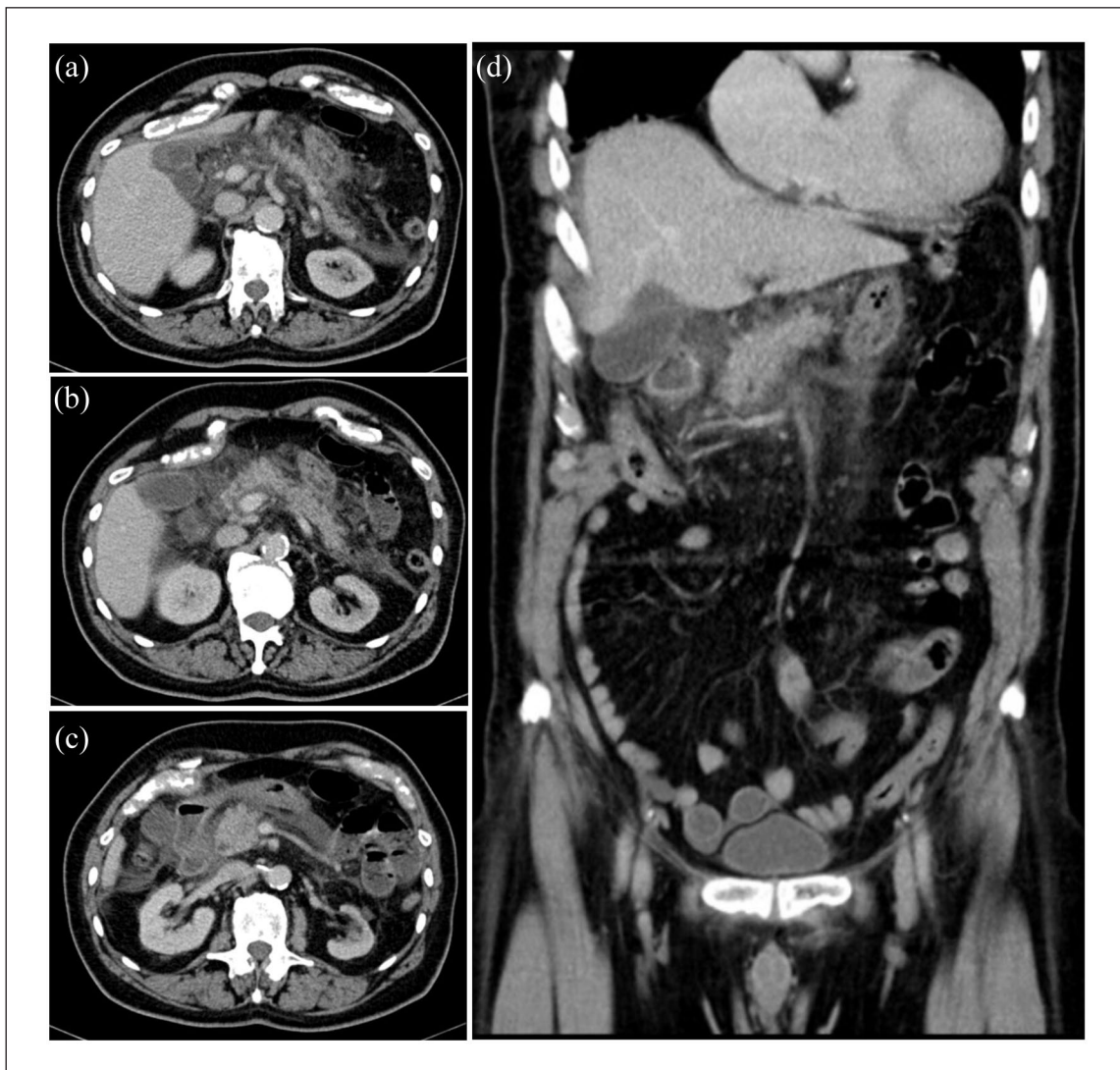
20 mg once daily, atenolol 50 mg once daily, isosorbide mononitrate 10 mg once daily, amlodipine 5 mg once daily, aspirin 75 mg once daily, atorvastatin 20 mg once daily, and levothyroxine 50 mcg once daily. He had been taking these medications daily for more than 12 months.

On examination, he was afebrile and mildly dehydrated. His blood pressure was 105/60 mmHg, his pulse rate was 62 beats per minute, and his oxygen saturation was 92% in room air. He had an obese abdomen that was soft on palpation with severe epigastric tenderness. He was tachypneic, but his chest X-ray was normal. An abdominal ultrasound showed no liver or gallbladder pathology. Laboratory tests revealed blood glucose of 5.6 mmol/L, a raised lactate dehydrogenase of 1998.0 U/L, erythrocyte sedimentation rate (ESR) of 73 mm/h, serum amylase of 2340.5 U/L, serum lipase of 645 U/L, serum cholesterol of 4.1 mmol/L, serum

triglyceride of 1.2 mmol/L, serum calcium of 2.3 mmol/L, and creatine kinase of 1107.0 U/L.

A contrast-enhanced computed tomography of the abdomen showed a heterogeneously enlarged pancreas with diffuse peripancreatic fat stranding and fluid collection in keeping with acute interstitial edematous pancreatitis (Figure 1), with a Balthazar score of 5/10. He was admitted to the surgical intensive care unit and initially treated conservatively with bowel rest, fluid replacement, intravenous pantoprazole 40 mg once daily, intravenous metoclopramide 10 mg thrice daily, and intramuscular tramadol 50 mg thrice daily.

The following day, his oxygen saturation dropped to 82% in room air and oxygen therapy was initiated via a non-rebreather face mask. A nasopharyngeal swab specimen was collected and tested positive for SARS-CoV-2 by reverse transcriptase polymerase chain reaction. A computed



**Figure 1.** Axial (a–c) and coronal (d) contrast-enhanced computed tomography of the abdomen at the level of the pancreas revealing a heterogeneously enlarged pancreas with diffuse peripancreatic fat stranding and fluid collection in keeping with acute interstitial edematous pancreatitis.

tomography of the chest revealed bilateral minimal pleural effusion with partial atelectasis of the left lower lobe. The patient was initiated on a cocktail of medications, including intravenous dexamethasone 4 mg twice daily, intravenous ceftriaxone 1 g once daily, oral azithromycin 500 mg once daily, subcutaneous enoxaparin 60 mg once daily, oral vitamin C 1000 mg once daily, oral vitamin D 5000 units once daily, and oral zinc 40 mg once daily, in addition to his regular medications. He was also isolated and monitored for any clinical deterioration.

The respiratory symptoms gradually resolved within 5 days. His serum amylase and lipase levels gradually decreased with time. On day 5, his control serum amylase and lipase were 272.0 and 179.5 U/L, respectively. He was discharged home after he was clinically stable and had no respiratory or gastrointestinal complaints. He reported significant improvement on follow-up 2 weeks after discharge. The serum pancreatic enzymes, amylase of 84.0 U/L and lipase of 91.0 U/L, had normalized 4 weeks later.

## Discussion

Acute pancreatitis is a reversible inflammation of the exocrine pancreas that is most commonly caused by gallstones, alcohol consumption, or idiopathic causes. Intrapaneatic protease activation from acinar cell injury and impaired zymogen secretion is the basis of this disease.<sup>4</sup> Acute pancreatitis requires two of the three criteria for diagnosis: relevant history, elevated serum pancreatic enzymes (amylase or lipase) more than three times the upper limit of normal, and suggestive imaging findings.<sup>4</sup>

Apart from gallstones and alcohol, various microorganisms may cause acute pancreatitis, including bacteria (mycoplasma, legionella, salmonella, and leptospira); viruses (viral hepatitis, Coxsackie virus, cytomegalovirus, human immunodeficiency virus, herpes virus, mumps, and other viruses); parasites (toxoplasmosis, cryptosporidium, and ascaris); and fungi (*Aspergillus*).<sup>5</sup> Although the exact mechanism of viruses causing acute pancreatitis is not known, a possible mechanism includes viral replication in pancreatic acinar cells resulting in protease leakage and activation, in addition to ampullary edema and cholangiopathy.<sup>6</sup>

SARS-CoV-2 enters host cells via its spike protein, which binds to angiotensin-converting enzyme 2 (ACE2). However, the expression of ACE2 in the normal pancreas (exocrine glands and islets of the pancreas) was slightly higher than that in the lungs, suggesting the possibility of pancreatic injury in SARS-CoV-2-infected patients.<sup>7</sup> However, it remains controversial whether SARS-CoV-2 infections increase acute pancreatitis.<sup>8</sup> Although the pathophysiology is multifactorial, the proposed mechanisms include direct tissue invasion, inflammation-mediated damage, and microvascular injury, as observed in the intestines<sup>9</sup> and ear.<sup>1</sup> However, SARS-CoV-2 outside the lungs may be due to residual viral RNA, as seen in the pancreas.<sup>10</sup>

The acute respiratory distress syndrome and multi-organ failure seen in severe SARS-CoV-2 infection resemble the lipotoxicity process in severe acute pancreatitis. Therefore, it has been suggested that acute pancreatitis may be caused by either systemic inflammation of SARS-CoV-2 or viral invasion of the pancreas.<sup>10</sup> This was confirmed when a study demonstrated that SARS-CoV-2 can directly infect human-induced pluripotent stem cell-derived pancreatic cells with the presence of the virus in confirmed post-mortem human pancreatic tissue.<sup>11</sup>

From the patient's medication list, aspirin, atorvastatin, olmesartan, and atenolol, although rare, may have caused drug-induced pancreatitis.<sup>12</sup> However, the various therapies used during the SARS-CoV-2 infection—ranging from antibiotics, antivirals, steroids, and monoclonal antibodies—may have been linked to causing acute pancreatitis as an adverse effect.<sup>13</sup> Since the respiratory symptoms exacerbated during hospitalization, there is a possibility that the patient may have acquired SARS-CoV-2 infection during hospitalization.

## Conclusion

This case report highlights one of many cases reported of acute pancreatitis in SARS-CoV-2 infection, with a favorable outcome. From the current literature, it is evident that SARS-CoV-2 directly invades the pancreatic cells, causing injury. However, there are several confounding factors in the etiology of acute pancreatitis during concomitant SARS-CoV-2 infection. Although SARS-CoV-2 was identified but unproven as a causal factor in this case, raising the suspicion in patients with acute pancreatitis may reduce delay in the diagnosis and treatment of SARS-CoV-2. However, further research is warranted to evaluate whether pancreatic involvement is one of the clinical presentations or subsequent complications of SARS-CoV-2 infection.

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## Author contributions

J.S. and J.L. conceptualized and drafted the manuscript. E.S., A.S., S.U., D.M., and S.C. have reviewed the medical records. A.S. has read and reported the radiology images. A.S. and J.L. edited the manuscript. All the authors have read and approved the final manuscript.

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## Ethical approval

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## Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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