International Journal of Research in Medical Sciences
Kapardar BK et al. Int J Res Med Sci. 2021 Sep;9(9):2841-2844
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20213229

Case Report

A rare case report of acute necrotizing pancreatitis being the sole extra-pulmonary manifestation of COVID-19

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Received: 30 July 2021
Revised: 12 August 2021
Accepted: 13 August 2021

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ABSTRACT

The current pandemic of Corona virus disease (COVID) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Commonly the disease presents as symptoms suggestive of upper respiratory tract infection to severe acute respiratory infection syndrome (SARI). However various extra-pulmonary manifestations related to gastrointestinal system and hepatobiliary system including acute pancreatitis have been reported quite frequently. Here we report a case of COVID-19 who presented with acute necrotizing pancreatitis without any respiratory symptoms.

Keywords: COVID-19, Acute necrotizing pancreatitis, Viral pancreatitis, Extra-pulmonary manifestation

INTRODUCTION

Ever since the first case of novel Corona virus disease (COVID-19) pandemic originated from Wuhan, China in the month of December 2019 more than 220 countries and territories around the world have reported more than 196 million of confirmed cases of COVID-19 and more than 4 million deaths have been reported due to the same disease. India has been struck hard by the second wave of COVID19 pandemic with daily positive cases and deaths being reported as more than 400,000 cases and 4,000 deaths, respectively. Commonly COVID-19 causes symptoms related to respiratory system ranging from upper respiratory tract infection to lower respiratory tract infection including severe acute respiratory infection. However extra-pulmonary symptoms related to gastrointestinal system and hepatobiliary system have been reported quite frequently since the onset of first wave of COVID-19 pandemic.[1] Patient can present with features of acute abdomen including acute pancreatitis as reported in various cases since the first wave of COVID-19.[1,2] Here we report a case of acute necrotizing pancreatitis as the only manifestation of COVID-19 without any symptoms of respiratory system. Proper history taking and relevant investigations excluded other possible causes of pancreatitis in our case. So, it is imperative not to rely only on respiratory symptoms but to screen all patients presenting with acute pancreatitis for SARS-CoV-2 so that the risk of possible transmission to the attending interventionist can be excluded altogether.

CASE REPORT

A 44 years old man presented to the emergency department at all India institute of medical sciences, Bhubaneswar with chief complaints of sudden onset severe stabbing abdominal pain in the left upper quadrant with history of radiation of pain to the back and associated with severe nausea and recurrent vomiting of 1 day duration. The abdominal pain was increasing in intensity on lying down posture and decreasing on sitting and leaning forward posture. On careful history taking he had mild upper abdominal pain since last 5 days which was burning sensation in nature, associated with nausea and occasionally radiating to the back. For the same complaints he was treated for acid peptic disease from

local primary care hospital at his village, but without any relief. Later on, the course after two days he developed acute constipation and the abdominal pain started increasing in intensity until when it was unbearable the patient was referred to our tertiary care centre for further evaluation and management. The patient was not an alcoholic, neither he was addicted to any other substance abuse. Considering the second wave of current pandemic of COVID-19 on further history taking he revealed that he had recent contact history with a laboratory proven COVID-19 positive case in his village ten days back. However, he did not develop any of the symptoms of fever, myalgia, cough or shortness of breath prior to the onset of abdominal pain. On past history and treatment history he was a known case of type-2 diabetes mellitus and on regular oral anti-diabetic medication with metformin 500 mg twice daily since last three months. He was not taking any other medication on regular basis.

On clinical examination the patient had sign of dehydration in the form of dry tongue and his vitals on presentation were normal body temperature, pulse rate of 125 beats per minute, regular and normal volume, blood pressure was 156/100 mmHg and oxygen saturation were 95% on room air. On abdominal examination there was mild abdominal distension and severe tenderness in epigastrium and left hypochondrium area, on auscultation there was sluggish bowel sound and on digital rectal examination showed collapsed rectum and stool-stained finger. His immediate work-up in the emergency department revealed his random blood sugar of 358 mg/dl, his Acid blood gas analysis showed normal pH, but mild hypokalaemia of 2.8 mmol/l and other parameters being normal, Urine for ketone body-negative, his ECG showed sinus tachycardia, his chest X-ray (P-A view) was of normal study, his X-ray abdomen (erect view) showed dilated transverse colon, descending colon and sigmoid colon and air in pelvic bowel loops and his emergency Ultrasound scan of whole abdomen showed normal gall bladder, distended bowel loops with bulky head of pancreas and further details of pancreas could not be commented as the field was obscured by bowel gas and no features of intestinal obstruction. His immediate laboratory parameters (Table 1) showed raised total leucocyte count, raised serum amylase and lipase level (greater than three times of normal value), normal renal function test and liver function test, mild hypokalaemia and other electrolytes being normal, lipid profile showed normal triglycerides level. His nasal swab reverse transcriptase polymerase chain reaction test for SARS-CoV-2 came positive. After general surgery and gastroenterology consultation diagnosis was made as acute pancreatitis with paralytic ileus with uncontrolled type-2 diabetes mellitus with mild COVID. He was admitted in COVID isolation ward and management was started as per protocol. He was managed conservatively with bowel rest, intravenous crystalloid fluid resuscitation, analgesia and basal-bolus insulin regimen and correction of hypokalaemia. As the pain was persistent on third day of admission Contrast enhanced CT scan of abdomen (Figure 1, 2 and 3) was performed which showed diffusely bulky pancreas with fuzzy outline, more than 30% necrosis in neck, the body and tail of pancreas in the form of an ill-defined necrotizing collection along neck, body and tail of pancreas extending into lesser sac measuring 1.7×8.7×1.8 cm (AP-TR-CC), diffuse inflammatory changes in the form of peri-pancreatic, mesenteric fat stranding, thickening of left para-renal fascia as well as thickening of second part of duodenum. No evidence of pseudoaneurysm or thrombosis was seen. There was minimal ascites noted in the abdomen and pelvis. So, the final diagnosis was changed to acute necrotizing pancreatitis according to modified Atlanta criteria. Following this empirical antibiotic with piperacillin-tazobactam and escalation of analgesia were added to the management. His laboratory tests were performed on daily basis. After 2-3 days his abdominal symptoms decreased in intensity and he tolerated liquid diet. After about seven days of management his abdominal pain subsided completely and the laboratory reports (Table 1) also came out to be within normal limits. After a total of twelve days of hospitalisation he was discharged in a hemodynamically stable condition. After fourteen days on follow-up visit, he was absolutely normal without any fresh complaints and he was on his normal daily diet.

Table 1: Serial laboratory tests values.

Tests	Day 0	Day 3	Day 7
Hb (g/dl)	11.4	11.6	10.3
TLC (cumm)	14170	13540	9440
Neutrophil (%)	92.4	83	83.2
Lymphocyte (%)	5.1	13.3	11.9
TPC (lakh/cumm)	2.57	1.6	2.03
S. Urea (mg/dl)	48	26	13
S. creatinine (mg/dl)	1.0	0.6	0.8
RBS (mg/dl)	358	188	146
S. Amylase (U/l)	277	217	36
S. Lipase (U/l)	396	182	45
TB (mg/dl)	1.3	0.9	1.1
DB (mg/dl)	0.5	0.2	0.4
IB (mg/dl)	0.8	0.7	0.7
AST (U/l)	27	20	21
ALT (U/l)	20	15	13
ALP (U/l)	67	60	66
S. Sodium (mEq/l)	141	138	138
S. Potassium (mEq/l)	2.9	3.8	3.56
S. Calcium (mmol/l)	1.12	1.15	1.14
Triglycerides (mg/dl)	94	108	102
PT (sec)	25.9	24.6	25.2
aPTT (sec)	14.	16.2	12.4
INR	1.30	1.18	1.12

Hb-Hemoglobin, TLC-Total leukocytes, TPC-Total platelet count, RBS-Random blood sugar, TB-Total bilirubin, DB-Direct bilirubin, IB-Indirect bilirubin, AST-Aspartate transaminase, ALT-Alanine transaminase, ALP-Alkaline phosphatase, PT-Prothrombin time, aPTT-activated partial thromboplastin time, INR-International normalised ratio.

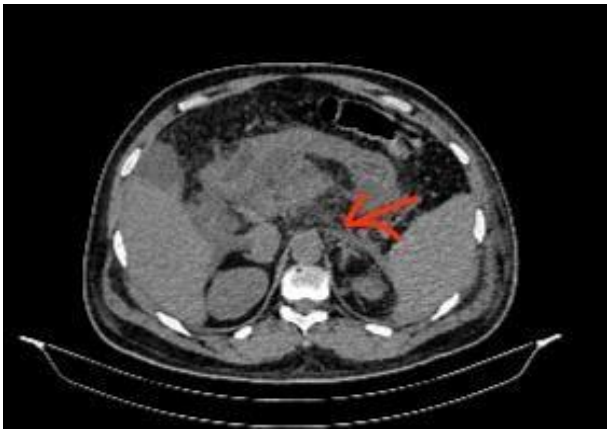

Figure 1: Contrast enhanced CT scan abdomen (axial view) showing areas of nonenhancement consistent with development of necrosis and thickening of left para-renal fascia (arrow mark).

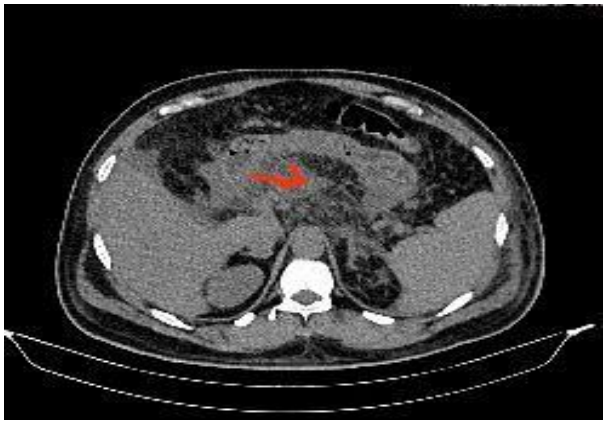

Figure 2: Contrast enhanced CT scan of abdomen (axial view) showing decreased enhancement (arrow mark) in the neck/body of the pancreas suggestive of necrotizing pancreatitis.

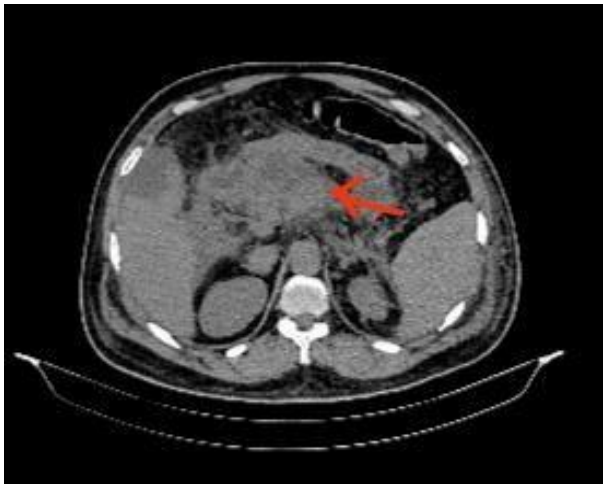

Figure 3: Contrast enhanced CT scan of abdomen (axial view) showing decreased enhancement (arrow mark) in the neck/body of the pancreas suggestive of necrotizing pancreatitis.

DISCUSSION

Coronaviruses (CoVs) are a large family of single-stranded RNA viruses, with infectivity in humans and animals, causing a myriad of symptoms.[3] Acute pancreatitis is caused most commonly by gallstones and alcohol consumption. Diagnosis of acute pancreatitis requires two out of three criteria that include typical history, elevated serum amylase or lipase more than 3 times the upper limit of normal for the laboratory reference range and suggestive imaging findings.[4] Acinar cell injury and impaired zymogen secretion leading to intra-pancreatic protease activation is the pathogenesis of this disease. Other common causes of acute pancreatitis include hypertriglyceridemia, any trauma, surgical procedure, invasive investigation like endoscopic retrograde cholangiopancreatography (ERCP), Anatomical pancreatic anomalies, any similar complaints in the past suggestive of chronicity of disease, drug induced like etc. Several viruses reported in the etiology of acute pancreatitis include cytomegalovirus, Epstein-Barr virus, hepatitis AE viruses, herpes simplex virus, varicella zoster virus, mumps, measles and coxsackie virus etc. The possible mechanisms include viral replication in pancreatic acinar cells resulting in protease leakage and activation, in addition to cholangiopathy and ampullary oedema.[5] SARSCoV2 enters host cells via its spike (S) protein which binds to angiotensin-converting enzyme 2 (ACE2). In the initial phase of the disease, infection of the upper respiratory tract ACE2expressing nasal epithelial cells occurs in asymptomatic individuals. Later in the disease course, infection of lower respiratory tract ACE2-expressing cells causes pneumonitis.[6] The virus mainly causes pulmonary disease; however, extra-pulmonary manifestations affecting gastrointestinal and hepatobiliary systems have also been reported. Pathophysiology of COVID-19 can be multifactorial. Suggested mechanisms include direct tissue damage, inflammation-mediated damage and microvascular injury as observed in the small bowel.[1] The microvascular injury includes diffuse severe endotheliitis of the submucosal vessels occurring in several anatomical sites that in turn, cause diffuse micro-ischemic disease. Similar ischemic damage could occur in the pancreas.[7] In a study by Liu et al expression of ACE2 in normal pancreases was found to be slightly higher than that of the lungs and it was expressed in both the exocrine glands and islets of the pancreas.[8] Schepis et al reported the detection of SARS-CoV-2 RNA in a fluid sample obtained from a pancreatic pseudocyst in a patient with COVID-19 pneumonia and acute pancreatitis.[9] Gonzalo-Voltas et al reported a case of pancreatitis from Spain in the absence of the most common risk factors: alcohol and gallstones.[10] Reports from Wuhan, China provide great insight into this possible association. Among 52 patients with COVID-19 pneumonia, 9 patients (17%) were found to have pancreatic injury.[11] A study from the USA revealed a point prevalence of 0.27% of acute pancreatitis in hospitalized patients with COVID-19.[3] Clinical history and relevant investigations in our case ruled out these

common causes of pancreatitis. So, there can be a temporal causal relationship of acute pancreatitis and its complications like acute necrotizing pancreatitis with SARS-CoV-2 virus.

CONCLUSION

It should be kept in mind that acute abdomen can be the sole clinical presentation of COVID-19 without any respiratory system involvement. Moreover, acute pancreatitis and its complications like acute necrotizing pancreatitis can be considered as the differential diagnosis of acute abdomen in a case of COVID-19. Proper clinical history taking and relevant investigations and prompt management of the case can have a better outcome in such cases.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Kapardar BK, Pradhan SR. A rare case report of acute necrotizing pancreatitis being the sole extra-pulmonary manifestation of COVID-19. Int J Res Med Sci 2021;9:2841-4.