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CASE REPORT

COVID 19 induced acute pancreatitis in patients with renal impairment: report of five cases

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Abstract

COVID 19 infection is an ongoing pandemic that the world is facing currently. Though SARS-CoV2 infection mainly involves the lungs, it is known to affect other organs like kidneys, brain, heart, endocrine organs and gastrointestinal system. It is hypothesized that the ACE2 and transmembrane serine protease 2 which are expressed in the beta cells of the pancreas are the entry receptors for the SARS-CoV-2 virus, thus causing pancreatitis. A retrospective review of clinical records at our institution during the COVID 19 pandemic from 2019 to 2020 was carried out to find patients with COVID 19 infection presenting with acute pancreatitis. Additionally, a review of literature was conducted about COVID 19 patients presenting with pancreatitis in chronic kidney disease and renal transplantation recipients. Five patients with COVID 19 infection presented with acute pancreatitis during the 2019–2020 pandemic period. All patients were males and mean age of the patients was 48 ± 20 years. Out of 5 patients, 3 were chronic kidney disease patients, 2 were renal transplantation recipients. COVID 19 infection was the cause of acute pancreatitis in all 5 cases. Out of 5, 1 patient had acute necrotizing pancreatitis and the rest had mild to moderate severity pancreatitis. All patients recovered except the patient with acute necrotizing pancreatitis who succumbed to the illness. One patient with chronic kidney disease became dialysis dependent post recovery from pancreatitis. In all 5 patients, there was no correlation between the severity of COVID ARDS and the severity of pancreatitis. There was no correlation between the severity of pancreatitis and the elevation of inflammatory markers. In patients presenting with pancreatitis, we have to keep in mind COVID 19 infection along with other known aetiologies of acute pancreatitis.

Keywords Acute pancreatitis · COVID-19 infection · Chronic kidney disease · Renal transplantation

Introduction

SARS-CoV-2 though initially thought to involve only the lungs was eventually found to involve other organs also. There have been few case reports of COVID 19 infection-induced pancreatitis [1, 2]. Treating chronic kidney disease or renal transplantation recipients with COVID 19 infection and pancreatitis can be challenging especially due to associated comorbidities and impaired immunity. We present 5 patients with COVID 19 infection, who presented with acute pancreatitis.

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Case report

Patients

A retrospective review of records at Manipal hospitals, Bangalore between May 2020 to June 2021 during the COVID 19 pandemic period was done to identify COVID 19 infected patients presenting with acute pancreatitis. Five patients were identified to have COVID 19 infection and pancreatitis.

Clinical cases

Patient A

A 42 year-old gentleman, with hypertension, end-stage renal disease on maintenance hemodialysis presented to our unit with a history of fever, headache and pain abdomen of 3 days duration. His covid testing was positive through a naso-pharyngeal swab, detected by real-time reverse transcription

PCR. He was admitted in the isolation ward as per protocol. At admission, his pulse rate was 90/min, BP- 120/80 mmHg, saturation was 97% on room air. After admission, his HRCT thorax done showed a few patchy ground-glass opacities and organizing pneumonia with a CT severity score of 2/25. His laboratory investigations showed raised amylase, lipase with rise in inflammatory markers (Table 1). His CT abdomen with contrast showed that the entire pancreas was mildly bulky, particularly head and uncinate process. Mild peri-pancreatic fat stranding and minimal peri-pancreatic fluid with minimal ascites were present (Fig. 1). He was kept nil per oral initially and he was resuscitated with IV fluids. For COVID pneumonia, he was treated with i.v Remedesvir, i.v antibiotics, nebulization and other supportive care. On day 5, his pain abdomen reduced and he was allowed a liquid diet which he tolerated well. He improved symptomatically and was discharged after 10 days of hospitalization. At the time of discharge from the hospital, his laboratory investigation showed serum amylase-62 IU/L, serum lipase- 75 IU/L.

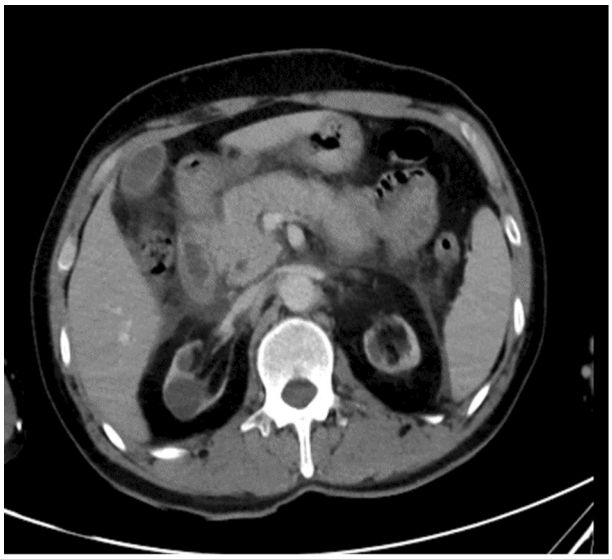

Fig. 1 Axial cuts of contrast-enhanced CT abdomen of patient A showing bulky pancreas

Patient B

A 71 year-old gentleman, with a medical history of hypertension and chronic kidney disease due to primary membranous nephropathy, with a nadir serum creatinine of 1.9 mg/dl, presented to our unit with a history of fever, cough, breathlessness and mild pain abdomen. His COVID testing was positive. At admission, his pulse rate was 90/min, BP-150/90 mmHg, Sp02- 88% on room air requiring 15l of oxygen support through non-rebreathing mask. On examination, his abdomen was distended with guarding present on palpation. He was admitted to the isolation ward as per protocol. His laboratory investigations done showed raised amylase, lipase levels with rise in inflammatory parameters (Table 1). His HRCT thorax showed bilateral extensive confluent peripheral and sub-pleural crazy paving opacity with interstitial thickening in a non-segmental distribution involving both lungs, all lobes, with a CT severity score of 16/25 (Fig. 2). CT abdomen showed extensive peri-pancreatic fat stranding and oedema extending along the para-renal fascia bilaterally with no localised collection (Fig. 3). He was kept nil per oral for 48 h and was treated with iv fluid resuscitations and his pancreatitis was managed conservatively. For his COVID ARDS, he was treated with i.v Remedesvir, i.v antibiotics, i.v dexamethasone 6 mg once daily, nebulization and other supportive care. His pain abdomen subsided after 4 days of admission and he was started orally which he tolerated well. His repeat enzymes done on day 4 of admission showed serum amylase- 78 IU/L, serum lipase- 79 IU/L. On day 6 of admission, his saturation dropped to 80% on room air with increase in oxygen requirement, so his steroids

Table 1 Laboratory investigations of all the patients at admission

At admission	Patient A	Patient B	Patient C	Patient D	Patient E
Haemoglobin (g/dl)	9	11.6	9.1	8.4	13.5
WBC count (cells/mm^3)	10,560	6190	6220	5880	4620
Platelet count (cells/mm^3)	226,000	197,000	119,000	110,000	188,000
Serum creatinine (mg/dl)	7.8	2.8	9.5	3.8	1.3
Serum amylase (IU/L)	136	93	139	204	834
Serum lipase (IU/L)	119	143	178	185	2851
Serum albumin (g/dl)	3.9	2.1	3.4	3.4	4.1
Serum AST/ ALT (IU/L)	15/69	107/55	21/15	14/16	23/24
Serum calcium (mg/dl)	9.3	6.7	9	7.8	6.5
IL6 (pg/ml)	32.9	78.1	56.5	8.1	10.7
CRP (mg/L)	44.1	28.3	49.1	40.8	1.7
Serum ferritin (ng/ml)	1015	2337	1073	2055	117.3
D dimer (mg/L)	8.85	0.19	1.04	0.6	0.05

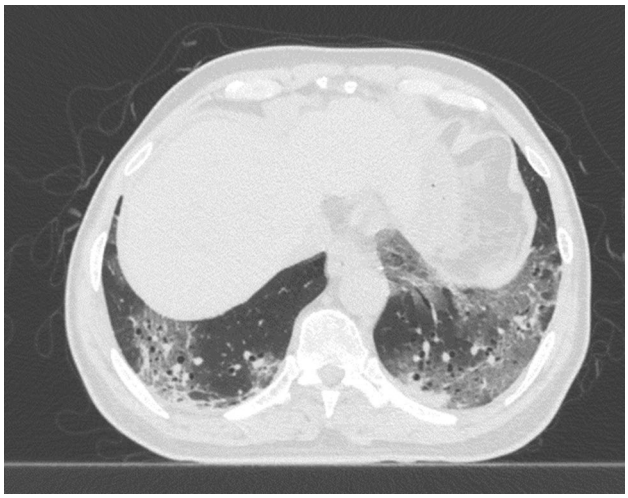

Fig. 2 Axial cut of HRCT thorax of patient B suggestive of extensive crazy paving opacities

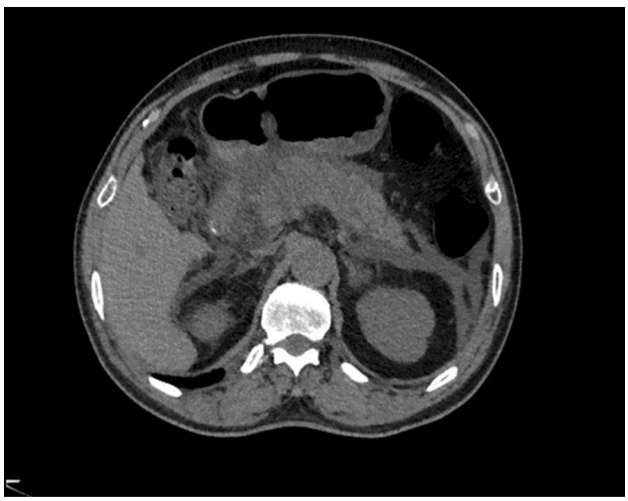

Fig. 3 Axial cut of plain CT abdomen of patient B suggestive of peripancreatic fat stranding

were increased to i.v methylprednisolone 40 mg once daily. His serum creatinine worsened gradually from 2.8 mg/dl at admission to 5.9 mg/dl on day 6 of admission with the development of uremic symptoms needing initiation of hemodialysis through a temporary dialysis catheter. He required a few sessions of dialysis due to acute on chronic kidney disease following which his dialysis was stopped. His steroids were gradually tapered and stopped. He improved symptomatically but continued to require 2l of oxygen support. Since he continued to require oxygen support, his repeat HRCT thorax was done on day 20 of admission, which showed mild bronchiectasis and small cystic spaces in both lungs. He was discharged on home oxygen support of 2 l/min on day 32 of admission.

Patient C

A 63 year-old male with a medical history of diabetes mellitus, hypertension, ischemic heart disease, diabetic nephropathy, chronic kidney disease- stage 5 not on dialysis had a baseline creatinine of 6 mg/dl. He presented to the emergency unit with complaints of fever, cough, breathlessness, pain abdomen and multiple episodes of vomiting since 5 days. On examination- his oxygen saturation on room air was low—90%, requiring oxygen support. His pulse rate was 110/min, BP- 130/80 mmHg. His COVID testing was positive, so he was admitted to the isolation ICU. His laboratory investigations done showed BUN-67 mg/dl, serum creatinine-9.5 mg/dl, serum potassium-6 mmol/L, serum bicarbonate- 16 mmol/L. His HRCT thorax done showed multiple areas of ground-glass opacities noted in all lobes of both lungs predominantly in peripheral and peri-broncho vascular distribution with CT severity of 18/25 (Fig. 4). CT abdomen showed mild perinephric fat stranding. Because of pain abdomen and persistent vomiting episodes, he was kept nil per oral and resuscitated with IV fluids. For his COVID ARDS, he was treated with IV antibiotics, IV Remedesvir 5 doses, IV dexamethasone 6 mg and supportive care. In view of high serum creatinine and high potassium levels, he was initiated on hemodialysis. His pain abdomen reduced by the next day and he was started with oral feeds which he tolerated well. His laboratory tests done on day 5 of admission showed serum amylase-62 IU/L, lipase-53 IU/L. His oxygen saturation improved gradually and his oxygen support was tapered and stopped by day 10. He became dialysis dependent and was continued on dialysis after discharge from the hospital.

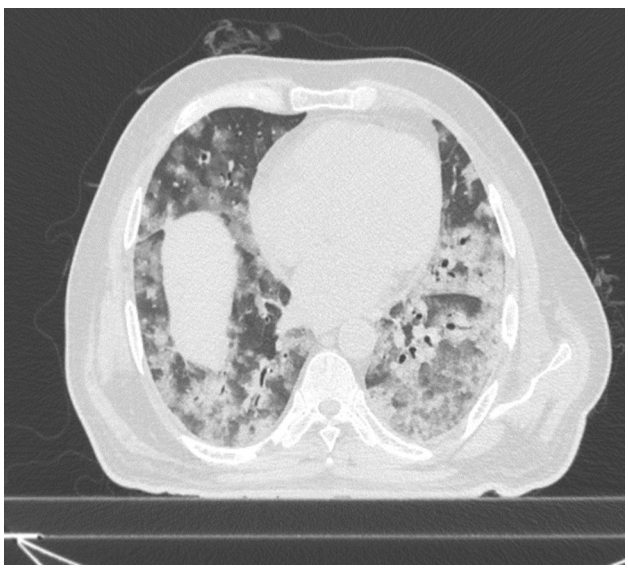

Fig. 4 Axial cut of HRCT thorax of patient C showing bilateral extensive ground glassing

Patient D

A 36 year-old gentleman with a medical history of hypertension, live related renal transplantation done in 2019 with native kidney disease being chronic interstitial nephritis had a nadir serum creatinine of 3.1 mg/dl. His immunosuppression comprised of mycophenolate mofetil and prednisolone. He presented to our unit with fever, cough, persistent vomiting and pain abdomen. His COVID testing was positive. At admission, his pulse rate was 80/min, BP- 120/90 mmHg, Sp02- 95% on room air. On examination- he had tenderness in the umbilical region, but there was no guarding or rigidity of the abdomen. He was admitted to the isolation ward as per the protocol. His laboratory investigations done showed raised amylase and lipase levels (Table 1). His HRCT thorax done showed bilateral sub-pleural ground glassing with CT severity of 6/25. CT abdomen showed evidence of acute pancreatitis. He was kept nil per oral because of pain abdomen with repeated episodes of vomiting and was resuscitated with IV fluids. For COVID ARDS, he was treated with IV Remedesvir, IV antibiotics, nebulization and other supportive care. His mycophenolate mofetil was stopped temporarily due to infection, his prednisolone was stopped and IV Dexamethasone 6 mg once daily dose was added. He was treated conservatively for pancreatitis, and he was symptomatically better by day 4 of admission and he was started on oral feeds which he tolerated well. His serum lipase was 124 IU/L on day 7 of admission.

Patient E

A 28 year-old gentleman, had a live-related renal transplantation in 2014 with his mother as a kidney donor. His native kidney disease was Alport's syndrome. His nadir creatinine was 1.1 mg/dl. He presented to our unit, with a history of fever, cough, persistent pain abdomen and multiple episodes of vomiting of 5 days duration. At admission, his pulse rate was 100/min, BP- 110/80 mmHg, SpO2 was 98% on room air. On examination of his abdomen- he had severe tenderness in the umbilical region with guarding and rigidity. His COVID testing was positive. He was admitted to the isolation ward as per protocol. His laboratory investigation showed raised inflammatory markers and raised amylase, lipase levels (Table 1). After admission, his HRCT thorax showed bilateral small areas of alveolitis typical for SARS Cov 2 pneumonia. Contrast-enhanced CT abdomen showed that most of the pancreas was non-enhancing, except pancreatic tissue in the head uncinate process and distal tail region. There was also a significant interval increase in the peripancreatic, retroperitoneal, omental fat stranding. A focal thrombus was seen in the main trunk of the superior mesenteric vein for a length of 16 mm causing severe narrowing of caliber (S1). He was kept nil per oral and resuscitated with IV fluids. For COVID ARDS, he was treated with i.v Remedesvir, i.v Piperacillin Tazobactum, nebulization and supportive care. His serum calcium was low, so i.v calcium correction was given. Mycophenolate mofetil was stopped due to infection, prednisolone was changed to i.v dexamethasone, tacrolimus was stopped since the patient was NPO and i.v cyclosporine was added. i.v Enoxaparin was given since his CT abdomen was suggestive of SMV thrombosis and also keeping in mind the coagulopathy associated with COVID infection. Gastroenterology team opinion was sought and was advised continuation of i.v fluid resuscitation, antibiotics and supportive care. His pain abdomen persisted and on day 14 he had spikes of fever with raise in WBC counts. Repeat contrast-enhanced CT showed that the entire pancreas was necrotic sparing part of the head and tail. Extensive peri-pancreatic oedema with fat stranding, necrosis in the mesenteric fat and moderate ascites was present (S2). In view of fever spikes and increasing WBC counts, he was shifted to ICU for further care. His repeat blood cultures were sterile. His antibiotics were escalated to i.v Meropenem after discussing with microbiologists. On day 15, he developed hypotension requiring inotropes and his general health deteriorated. He developed multi-organ dysfunction and refractory shock. He succumbed to illness on day 15 of illness.

Discussion

COVID 19 infection, first detected in Wuhan, China is highly variable in its presentation. Though initially thought to involve only the lungs, eventually it was realized that the virus affects other organs like the heart, kidney, gastrointestinal system and pancreas [3]. Glycosylated-spike (S) protein expressed by the virus, binds to the ACE2 receptor, leading to cell invasion. A recent study has shown that upto 40% of patients affected with COVID-19 infection present with gastrointestinal symptoms [4]. A study from Wuhan, China showed that out of 52 patients suffering from COVID 19 infection, 17% of patients had pancreatic injury [5]. SARS-CoV-2 can manifest as pancreatitis in 32.5% of critically ill patients and with raised amylase, lipase levels in 7.5%-17% of the patients [6].

COVID 19 infection and renal impairment

Chronic kidney disease and renal transplantation recipients are at increased risk of severe COVID infection due to associated comorbidities and impaired immunity [7, 8]. The exact effect of COVID 19 infection on CKD and renal transplantation recipients is not fully understood and need further studies in the future. Renal dysfunction due to COVID 19 is multifactorial. COVID 19 can lead to acute kidney injury

(AKI), glomerular diseases, tubulo-interstitial disease or renal vascular dysfunction.

AKI—AKI has been documented among COVID 19 infected recipients due to various reasons. AKI due to fluid depletion, hypotension-induced acute tubular necrosis or ischemic nephropathy, acute interstitial nephritis (due to COVID 19 infection or antivirals/ antibiotics employed), complement or cytokine-mediated injury have all been reported so far. Rhabdomyolysis-induced cast nephropathy and COVID 19-induced autoimmune haemolytic anaemia producing hemoglobinuria are two further possible causes of AKI.

There has been various hypothesis for AKI in COVID 19 infected patients. The cytopathic effect of COVID 19 infection on kidneys is thought to be caused by increased ACE2 expression on podocytes, proximal tubular cells, and bladder cells. The activation of Toll-like receptors (TLR) by ACE2 results in increased production of cytokines and chemokines such as IL-6, TNF-alpha, and others leading to renal injury. Higher levels of FGF2 and Smad7 have been linked to renal cell apoptosis in COVID 19 infected patients, but more research is needed to confirm this. In COVID 19 infected patients, male gender, advanced age, diabetes, underlying cardiovascular disease, hypertension, low baseline glomerular filtration rate, obesity, and hypotension are all independent risk factors for AKI [9, 10].

Glomerular disease—COVID 19 can aggravate infection in patients on immunosuppressive medicines and worsen renal function in patients with underlying glomerular disease.

In patients of African descent with the APOL1 genotype, COVID 19 has been associated with collapsing focal segmental glomerulonephritis (FSGS). COVID 19 has been linked to IgA nephropathy, anti-GBM disease, and ANCA vasculitis in some patients. In few cases, the COVID vaccine has been observed to promote relapse of underlying glomerular disease in persons who had been in remission previously [11, 12].

Tubulointerstitial diseases—COVID 19 can lead to acute tubular necrosis, acute interstitial nephritis, acute cortical necrosis, etc. In patients with pre-existing chronic interstitial nephritis, it can cause further worsening of renal functions.

Vascular lesion—COVID 19 can induce thrombotic microangiopathy, which can be triggered by hypercoagulability, microthrombi formation, persistent inflammation, cytokines, endothelial injury, and activation of the alternative complement pathway, etc.

End-stage kidney disease (ESKD)—Due to their immunocompromised state and accompanying comorbidities, ESKD patients are at a higher risk of contracting COVID infection. ESKD patients have a higher incidence of severe disease, need for mechanical ventilation, and mortality when compared to the general population.

Renal transplant recipients—Many studies, including Nair et al., Banerjee et al., and Elhadedy et al. [13–15], have reported severe COVID infection in renal transplant recipients. Additionally, managing immunosuppressive medications in these patients during infection is difficult. Those with the mild disease rarely require immunosuppression dose adjustments, whereas patients with moderate to severe disease require immunosuppressive medicine modifications. Antimetabolites/anti-proliferative medications are frequently withheld briefly or their dose is lowered, whereas calcineurin inhibitors are usually continued. Steroids are continued, and if necessary, the dose is raised. The risk of rejection must be considered while changing immunosuppressive medicines. The general consensus says that renal transplantation patients are at increased risk of COVID 19 infection due to their impaired immunity state. A study conducted in 2020, with a cohort of 36 renal transplant recipients infected with COVID 19 showed that patients with renal transplantation have a more rapid clinical progression and high mortality rate [16, 17].

Pancreatitis in patients with renal impairment

Avram et al. identified significant pancreatic pathology in 56% of uremic patients on hemodialysis, compared to 11.8% in those without renal insufficiency, in a study of 21 uremic patients on hemodialysis [18]. Patients on peritoneal dialysis have a higher rate of pancreatitis and its related morbidity than those on hemodialysis [19]. It can be caused by peritoneal dialysate's low pH, hypertonicity, and elevated glucose concentrations, or in patients who have had multiple episodes of peritonitis [20]. Kroner et al. discovered that hypercalcemia was the most common cause of acute pancreatitis among CKD-5 patients, independent of whether or not they needed dialysis, and that viral infections were the most common cause of acute pancreatitis among renal transplant recipients, followed by drugs. Furthermore, compared to the non-CKD group, CKD and transplant patients had higher associated morbidity and mortality [21].

Pathophysiology of acute pancreatitis in CKD

Acute pancreatitis in patients with chronic kidney disease is complex. Uremia, hypercalcemia with secondary hyperparathyroidism, and contrast agents are the usual causes. The pancreas is harmed by decreased renal clearance of gastrointestinal hormones such as glucagon, cholecystokinin, and gastric inhibitory peptide. In addition to the foregoing, dialysis patients face additional risks, such as hypotension during hemodialysis, which can lead to ischemia–reperfusion injury. Repeated peritonitis episodes put peritoneal dialysis patients at risk, and peritoneal dialysis fluid components

including calcium and glucose predispose them to pancreatitis [22].

Pathophysiology of acute pancreatitis in renal transplant recipients

The immunosuppressive drugs including steroids, calcineurin inhibitors, mTOR inhibitors, and antiproliferative therapies like azathioprine are all risk factors for pancreatitis. Because renal transplant recipients are immunocompromised, they are more vulnerable to viral infections such as CMV, EBV, and varicella zoster, which can cause viral pancreatitis [23]. Other variables, such as hyperglycemia and hypertriglyceridemia, might cause pancreatitis in kidney transplant recipients.

Hence, treating renal impairment patients with COVID 19 infection is challenging, more so if they have pulmonary and extra pulmonary manifestations of COVID 19 infection. Our 5 patients presented to the Nephrology unit with symptoms suggestive of pancreatitis which was later proven by laboratory tests like serum amylase and lipase or through radiological modality like CT abdomen. All 5 patients were male and mean age was 48 ± 20 years. All 5 patients were tested positive for COVID infection through a nasopharyngeal swab, detected by real-time reverse transcription PCR. In all 5 patients, there was no history of alcohol consumption, hypertriglyceridemia, history of any drug intake other than their regular prescribed medication for their comorbidities. None of the patients had a history of diabetes mellitus and their sugars were within normal limits. All of the patients' biliary enzymes, including GGT, were normal. On ultrasound or CT scans of the abdomen, no gall stones were found in any of the patients. Patients A–D had no EUS or MRCP done because there was no suspicion of biliary pancreatitis, however patient E had an MRCP, which was normal with no signs of CBD stone.

Of the 5 patients, 2 patients had chronic kidney disease, 1 patient had end-stage renal disease, 2 patients were post-renal transplantation on immunosuppressive drugs. All 5 patients presented with fever, pain abdomen and vomiting. According to the revised Atlanta classification, for the diagnosis of acute pancreatitis, at least 2 of the following are needed- pain abdomen suggestive of pancreatitis, raised amylase and lipase levels more than 3 times the normal levels or imaging findings suggestive of acute pancreatitis [24]. All 5 patients fulfilled the Atlanta criteria of acute pancreatitis. While 4 patients had mild pancreatitis, 1 patient had acute necrotizing pancreatitis (Table 2). COVID 19 induced pancreatitis on imaging usually shows features like increased bulkiness, increased peripancreatic fluid and edema, peripancreatic streaking, and severe cases show evidence of pancreatic necrosis, as described by studies such as Ahmet et al. and Naren et al. [25, 26]. Even all our patients had similar features on imaging with 1 patient having features of necrotizing pancreatitis. On imaging, autoimmune pancreatitis can include pancreatic duct or biliary duct dilatations or strictures, as well as peripancreatic lymph node enlargement, which our patients did not exhibit.

In our patients, lung CT severity score did not correlate with the severity of pancreatitis (Table 2). D dimer levels or inflammatory marker levels did not correlate with the severity of COVID ARDS or the severity of pancreatitis in our patients. The patient with SMV and splenic vein thrombosis did not have raised D dimer levels. 1 patient with CKD-stage 5 progressed to end-stage renal disease after recovery from pancreatitis, requiring maintenance dialysis. 1 patient with acute necrotizing pancreatitis succumbed to illness. Proteolytic enzyme inhibitors such as nafamostat were not used in any of our patients.

The patient with acute necrotizing pancreatitis developed septic shock and succumbed to illness. Necrotizing pancreatitis is associated with high morbidity and mortality [27]. His CT abdomen showed SMV and splenic vein thrombosis which can occur as a complication of pancreatitis due to the inflammatory process [28]. Various studies have shown that early necrosectomy is associated with high mortality compared to conservative treatment in patients with acute necrotizing pancreatitis. A study compared patients with severe necrotizing pancreatitis treated with late necrosectomy after 12 days versus early necrosectomy. The study showed high mortality in patients with early necrosectomy (56% in early necrosectomy versus 27% in patients with late necrosectomy) [29]. Our patient with necrotizing pancreatitis was treated with IV fluid resuscitation, pain management, nutritional support, iv antibiotics. He developed sepsis, multiorgan dysfunction and succumbed to illness.

In both our renal transplantation patients, mycophenolate mofetil was temporarily stopped due to on-going infection. In patient with necrotizing pancreatitis, since he was kept nil per oral, his oral tacrolimus was stopped and i.v cyclosporine was added. The choice of calcineurin inhibitor, an integral part of renal transplant immunosuppression, may also play a role in COVID 19 infection treatment. Cyclosporine A is hypothesized to have an inhibitory effect on coronavirus invitro due to its action on cyclophilin A and B [30, 31].

Conclusion

Treating COVID 19 infection is challenging when it presents with both pulmonary and extra-pulmonary symptoms. Patients with chronic kidney disease or renal transplantation can present with severe COVID infection. Through this case series, we emphasize that COVID 19 infection can present as pancreatitis and hence, it should be suspected in patients

Table 2 Demographics and clinical features of the patients

	Patient A	Patient B	Patient C	Patient D	Patient E
Age (years)	42	71	64	36	28
Sex	Male	Male	Male	Male	Male
BMI (kg/m^2)	22.9	20.6	21.4	19.8	20.1
Comorbidities	Hypertension, end-stage renal disease on dialysis	Chronic kidney disease, hypertension	Chronic kidney disease, hypertension, diabetes mellitus, ischemic heart disease	s/p renal transplantation, hypertension	s/p renal transplantation, hypertension
HRCT thorax severity	2/25	16/25	18/25	6/25	1/25
Presenting symptoms	Fever, headache, pain abdomen	Fever, cough, breathlessness, pain abdomen	Fever, cough, breathlessness, vomiting, pain abdomen	Fever, cough, vomiting, pain abdomen	Fever, cough, vomiting, pain abdomen
Serum amylase at admission (IU/L)	136	93	139	204	834
Serum lipase at admission (IU/L)	119	143	178	165	2851
Radiological evidence of pancreatitis	Yes	Yes	Yes	Yes	Yes
Onset of pancreatitis	Prior to pneumonia	Onset after pneumonia	Onset after pneumonia	Onset after pneumonia	Prior to pneumonia
Atlanta severity of pneumonia	Mild	Severe	Mild	Mild	Severe
Oxygen requirement	None	15 L through non-rebreathing mask	15 L through non-rebreathing mask	None	Initially no oxygen requirement, later intubated on day 14
Treatment	IV ceftriaxone, IV Remedesvir, IVenoxaparin, nebulization	IV piperacillin tazobactum, IV Remedesvir, IV enoxaparin, IV dexamethasone, nebulization	IV piperacillin tazobactum, IV Remedesvir, IV enoxaparain, IV dexamethasone, nebulization	IV ceftriaxone, IV Remedesvir, IV enoxaparin, nebulization, I.V dexamethasone	IV meropenem, IV metronidazole, IV remedesvir, Iv dexamethasone, IV enoxaparin, nebulization, ventilatory support
Complications	None	None	Dialysis dependent	None	Sepsis
Outcome	Improved	Improved	Improved	Improved	Death

presenting with pain abdomen or other symptoms suggestive of pancreatitis.

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Declarations

Conflict of interest There are no conflicts of interests.

Statement of human and animal rights This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent Informed consent is not required for this study.

References

1. Kataria S, Sharif A, Rehman AU et al. COVID-19 induced acute pancreatitis: a case report and literature review. Cureus. 2020;12.
2. AlHarmi RA, Fateel T, Adnan JS, et al. Acute pancreatitis in a patient with COVID-19. BMJ Case Rep CP. 2021;14:e239656.
3. Lai CC, Ko WC, Lee PI, et al. Extra-respiratory manifestations of COVID-19. Int J Antimicrob Agents. 2020;56:106024.
4. Zhang JJ, Dong X, Cao YY, et al. Clinical characteristics of 140 patients infected with SARS-CoV-2 in Wuhan, China. Allergy. 2020;75:1730.
5. Wang F, Wang H, Fan J, et al. Pancreatic injury patterns in patients with coronavirus disease 19 pneumonia. Gastroenterology. 2020;159:367.
6. Liu F, Long X, Zhang B, et al. ACE2 expression in pancreas may cause pancreatic damage after SARS-CoV-2 infection. Clin Gastroenterol Hepatol. 2020;18:2128.
7. Wu J, Li J, Zhu G, Zhang Y, et al. Clinical features of maintenance hemodialysis patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. Clin J Am Soc Nephrol. 2020;15:1139–45.
8. Alberici F, Delbarba E, Manenti C, et al. A single center observational study of the clinical characteristics and short-term outcome of 20 kidney transplant patients admitted for SARS-CoV2 pneumonia. Kidney Int. 2020;97:1083–8.
9. McAdams M, Ostrosky-Frid M, Rajora N, et al. Effect of COVID-19 on kidney disease incidence and management. Kidney. 2020;2:141.
10. Legrand M, Bell S, Forni L, et al. Pathophysiology of COVID-19-associated acute kidney injury. Nat Rev Nephrol. 2021;17:751–64.
11. Shetty AA, Tawhari I, Safar-Boueri L, et al. COVID-19–associated glomerular disease. J Am Soc Nephrol. 2021;32:33–40.
12. Gulati S, Prasad N, Sahay M, et al. Glomerular diseases with reference to COVID-19. Indian J Nephrol. 2020;30:158.
13. Nair V, Jandovitz N, Hirsch JS, et al. COVID-19 in kidney transplant recipients. Am J Transplant. 2020;20:1819–25.
14. Banerjee D, Popoola J, Shah S, et al. COVID-19 infection in kidney transplant recipients. Kidney Int. 2020;97:1076–82.
15. Elhadedy MA, Marie Y, Halawa A. COVID-19 in renal transplant recipients: case series and a brief review of current evidence. Nephron. 2021;145:192–8.
16. Banerjee D, Popoola J, Shah S, Ster IC, et al. COVID-19 infection in kidney transplant recipients. Kidney Int. 2020;97:1076–82.
17. Akalin E, Azzi Y, Bartash R, et al. Covid-19 and kidney transplantation. N Engl J Med. 2020;382:2475–7.
18. Avram MM. High prevalence of pancreatic disease in chronic renal failure. Nephron. 1977;18:68–71.
19. Lankisch PG, Weber-Dany B, Maisonneuve P, et al. Frequency and severity of acute pancreatitis in chronic dialysis patients. Nephrol Dial Transplant. 2008;23:1401–5.
20. Joglar FM, Saade M. Outcome of pancreatitis in CAPD and HD patients. Perit Dial Int. 1995;15:264–6.
21. Kroner PT, Mareth K, Raimondo M, et al. Acute pancreatitis in advanced chronic kidney disease and kidney transplant recipients: results of a US nationwide analysis. Mayo Clin Proc. 2019;3:160–8.
22. Barbara M, Tsen A, Rosenkranz L. Acute pancreatitis in chronic dialysis patients. Pancreas. 2018;47:946–51.
23. Graham D, Ito T, Busuttil R, et al. Pancreatitis in solid organ transplant patients: a review of the literature. OBM Hepatol Gastroenterol. 2019;3:1.
24. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102–11.
25. Bozdag A, Eroglu Y, Tartar AS et al. Pancreatic damage and radiological changes in patients with COVID-19. Cureus 2021;13.
26. Kumaran NK, Karmakar BK, Taylor OM. Coronavirus disease-19 (COVID-19) associated with acute necrotising pancreatitis (ANP). BMJ Case Rep CP. 2020;13:e237903.
27. Werge M, Novovic S, Schmidt PN, et al. Infection increases mortality in necrotizing pancreatitis: a systematic review and meta-analysis. Pancreatology. 2016;16:698–707.
28. Türkvatan A, Erden A, Türkoğlu MA, et al. Imaging of acute pancreatitis and its complications. Part 2: complications of acute pancreatitis. Diagn Interventional Imagin. 2015;96:161–9.
29. Mier J, Luque-de León E, Castillo A, et al. Early versus late necrosectomy in severe necrotizing pancreatitis. Am J Surg. 1997;173:71–5.
30. de Wilde AH, Zevenhoven-Dobbe JC, van der Meer Y, et al. Cyclosporin A inhibits the replication of diverse coronaviruses. J Gen Virol. 2011;92:2542.
31. Tanaka Y, Sato Y, Sasaki T. Suppression of coronavirus replication by cyclophilin inhibitors. Viruses. 2013;5:1250–60.