CASE REPORT

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The management of acute pancreatitis among children on chronic ambulatory peritoneal dialysis during COVID-19 pandemic crisis in Indonesia: report of two cases

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ABSTRACT

Background: Children on dialysis seem to be at greater risk for COVID-19. Acute pancreatitis (AP) is an infrequent but severe complication of chronic peritoneal dialysis (PD). It contributes to morbidity and mortality rates of up to 25%. Patients with PD are exposed to a series of factors associated with AP risk. This report aimed to describe rare and interesting cases of acute pancreatitis in children with CAPD following PD-related peritonitis with a favorable response to conservative treatment.

Case report: We present two cases admitted to our emergency room (ER) with severe abdominal pain preceded by PD-related peritonitis. Poor adherence, lack of monitoring, and healthcare service restriction during the COVID-19 pandemic predisposed these patients to PD-related complications. Patients were diagnosed as AP based on the revised Atlanta criteria. Both met the criteria as they had abdominal pain, a threefold increase of pancreatic enzymes, and evidence of pancreatitis through ultrasonography (USG) investigation. Both patients presented a rapid resolution of AP after receiving conservative treatment, including fasting, total parenteral nutrition (TPN), prophylactic antibiotics, and analgesics. None of them experienced invasive intervention due to AP.

Conclusion: Diagnosing AP in children with CAPD may be challenging since the symptoms mimic other abdominal problems. Our cases are likely to be associated with PD-related peritonitis. This report may prove conservative treatment as a recommendation for managing AP in children with CAPD. The rapid development of innovative clinical management strategies in response to the COVID-19 pandemic is crucial to improving children's health care quality with CAPD.

Keywords: chronic kidney disease, peritoneal dialysis, acute pancreatitis, children.
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INTRODUCTION

Since COVID-19 was confirmed to spread to Indonesia in March 2020, several areas have implemented lockdown strategies. Health providers are advised to adjust their working hours, suspend non-emergency visits, and tighten health protocols to reduce the risk of SARS-CoV-2 transmission.[1] Lockdown strategies also limit patients' access to healthcare services. Moreover, many patients avoid hospital visits for fear of virus infection.

Poor adherence while handling PD components and lack of monitoring during the "stay at home" program could be risk factors for PD-related complications.[2,3] Over the last few months, there has been an increase in the incidence of CAPD complications in our pediatric nephrology unit. Acute pancreatitis occurred unexpectedly as an uncommon sequela of PD-related peritonitis cases.

Acute pancreatitis is an acute inflammatory process involving the pancreas. Patients on dialysis are at higher risk for AP than the general population. Meanwhile, the incidence of AP is significantly higher in PD patients than in HD patients. The morbidity and frequency of necrotizing pancreatitis are higher in those on PD. The mortality among end-stage kidney disease (ESKD) children in those who suffer from AP is as high as 25%.[4] Risk factors for the incidence of AP in dialysis patients remain unclear.[5] Acute pancreatitis in CAPD patients may be coexistence with peritonitis, which is clinically challenging to differentiate.[5,6] Here, we report two cases of AP in children with CAPD following PD-related peritonitis episodes.

CASE REPORT

Case 1

A 13-year-old Indonesian male with ESKD secondary to a multicystic dysplastic kidney had been undergoing CAPD for eight months. He used 1.5% dextrose solution (900 mL, five exchanges a day) as the dialysate. During an outbreak, the parents suspended the routine visit to the pediatric nephrology outpatient clinic for about two months. Afterward, the patient was admitted to the ER due

to abdominal pain and was diagnosed with PD-related peritonitis caused by *Streptococcus pyogenes*. He was treated with intraperitoneal Gentamicin 8 mg/L for the loading dose and continued with 4 mg/L five times a day for 14 days based on affluent peritoneal culture. Evaluation of peritoneal effluent culture yielded *Candida krusei*. We administered intravenous Micafungin 100 mg once a day for four weeks afterward until he underwent PD catheter removal and converted to HD.

Three weeks after being discharged, he was readmitted to the ER due to severe abdominal pain, nausea, appetite loss, and vomiting. At presentation, his temperature was 36.7°C, heart rate of 96 beats/minute, respiratory rate of 24 breaths/minute, blood pressure of 100/60 mmHg, and body weight was 28 kgs. On physical examination, we noticed epigastric tenderness, and no mass was palpable per abdomen. The laboratory investigation yielded a low hemoglobin level of 7.5 g/dL; a normal white blood cell count of 9,750/µL; and a low platelet count of 57,000/µL. The amylase and lipase levels increased in the pancreatic enzyme to 521 U/L (reference range of 25-115 U/L) and 3,247 U/L (reference range of 79-393 U/L), respectively. The level of PTH was 1,145 pg/mL (reference range of 200-300 pg/mL for CKD G5D), 25(OH)D_3 was 23.8 ng/mL (reference > 30 ng/mL), total calcium serum was 10.2 mg/dL (reference range of 8.4-10.2 mg/dL), triglyceride serum level was 94 mg/dL (normal < 150 mg/dL), and blood glucose level was 100 mg/dL (reference range of 80-180 mg/dL). Gamma-glutamyltransferase and alkaline phosphatase levels were elevated, 481 u/L and 427 IU/L, respectively. Other serum electrolytes and liver function tests were within normal range. Abdominal USG revealed acute pancreatitis; acute cholecystitis; absence of gallstones, pancreatic calcification, and peripancreatic fluid collection. (**Figure 1**)

The patient was diagnosed with acute pancreatitis and cholecystitis. He was hospitalized, fasted and supported by TPN for 11 days, and then transitioned to oral liquid when the pain was well controlled. We administered intravenous Ceftriaxone 750 mg twice a day and Metronidazole 300 mg thrice a day with kidney adjusted dose for AP infection prophylaxis. We gave Acetaminophen 300 mg intravenously to relieve pain and transfused 1 unit of packed red blood cells. The patient was discharged after 24 days in improved clinical condition and decreased pancreatic enzyme levels in the normal range. Two weeks of follow-up after hospitalization, the amylase and lipase levels were 190 U/L and 223 U/L, respectively.

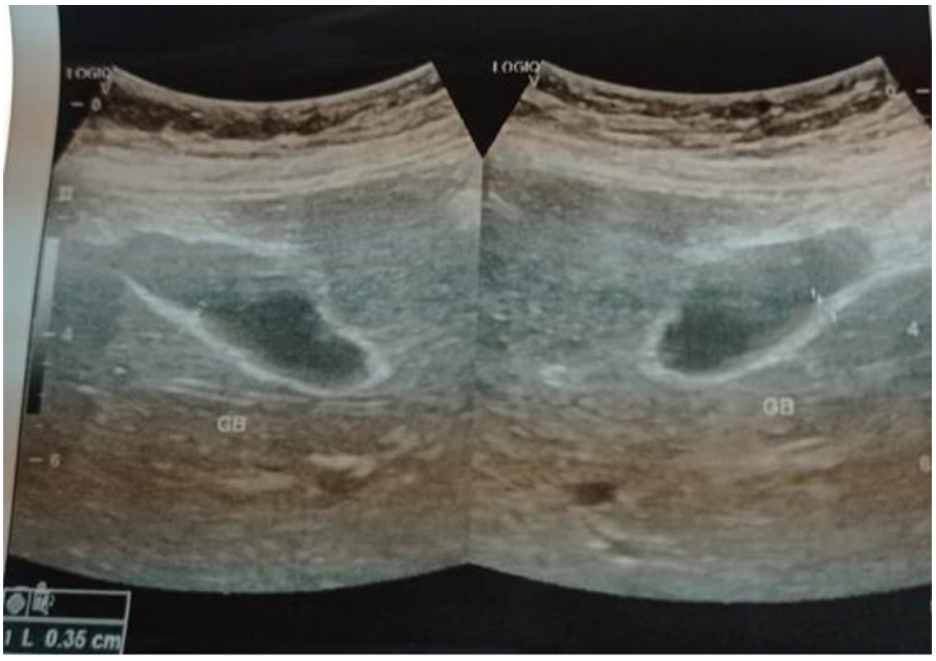

Figure 1. The abdominal USG of case 1 showed thickening of the gall bladder wall.

Case 2

A 15-year-old Indonesian female patient had a history of ESKD secondary to lupus nephritis and was treated with intravenous Methylprednisolone (MP) 600 mg for six cycles plus oral Prednisone 30 mg reduced, Mycophenolic acid 360 mg twice a day, and Hydroxychloroquine 125 mg once a day. She had been undergoing CAPD with 1.5% dextrose solution (700 ml, five exchanges a day) as the dialysate for six months when she got the first episode of PD-related peritonitis caused by *Klebsiella ozaena*. She was hospitalized and treated with intraperitoneal Cefoperazone sulbactam 250 mg five times a day for 14 days based on affluent peritoneal culture. From the history taking, we found out that the patient tended to skip the required details of PD protocol.

Although the peritoneal effluent culture evaluation showed a sterile result, she complained of abdominal pain and had negative ultrafiltration. Two weeks after being discharged, she was readmitted to the ER since the symptoms became progressively worse and more painful. At presentation, her temperature was 38.8°C, heart rate of 100 beats/minute, respiratory rate of 28 breaths/minute, blood pressure of 106/70 mmHg, and her body weight was 22 kgs. We found abdominal tenderness in the periumbilical region and epigastrium. There were no signs of infection on the exit site of the PD catheter. Laboratory examination obtained a hemoglobin level of 8.8 g/dL; normal white blood cell count of 11,500/µL; and platelet count of 125,000/µL. The serum amylase and lipase levels were 894 U/L and 3,947 U/L, respectively. There was a high level of PTH (1300.8 pg/mL), low 25(OH)D_3 (17.4 ng/mL), normal level of total calcium serum (8.6 mg/dl), normal level of triglyceride serum (102 mg/dL), and blood glucose (90 mg/dL). Other serum electrolytes and liver function tests were normal. Dialysate peritoneal effluent analysis showed 4,166 cells with highly expressed polymorphonuclear (PMN) leukocytes, and peritoneal effluent culture yielded *Escherichia coli*. Abdominal USG showed acute pancreatitis, in which the pancreas head size was 3.5, absent of calcifications and fluid collection around the pancreatic. (**Figure 2**)

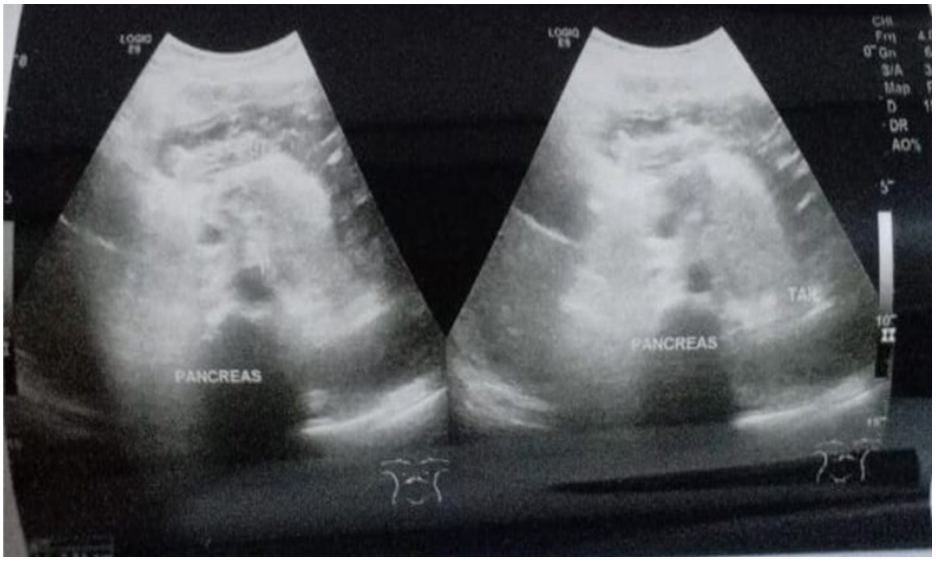

Figure 2. Enlargement of the pancreas head as the evidence of acute pancreatitis in case 2.

The patient was diagnosed with PD-related peritonitis and acute pancreatitis. She received conservative treatment, including fasting and TPN for 14 days until her pain was reduced and immediately transitioned to oral liquid. We administered intraperitoneal Gentamicin 8 mg/L for the loading dose and continued with 4 mg/L five times a day. Unfortunately, she complained of colicky abdominal pain in every PD exchange. Therefore, we decided to suspend the PD and initiate HD. We administered intravenous Ampicillin sulbactam 1,000 mg twice a day and Metronidazole 250 mg thrice a day with kidney adjusted dose as prophylactic antibiotics for infectious complications. We gave the patient intravenous Acetaminophen 250 mg for fever and pain reliever. She was discharged after 20 days of admission and continues to be on HD after discharge. The pancreatic enzymes returned to normal serum amylase of 115 U/L and lipase level of 397 U/L, along with clinical improvement after six weeks of follow-up.

DISCUSSION

To our knowledge, this was the first time we encountered such a case in Indonesia. Both cases occurred during the coronavirus outbreak. Poor adherence, lack of monitoring, and limitations in providing adequate PD assistance and retraining in these patients could be risk factors for PD-related complications. These patients had severe abdominal pain, a threefold increase in pancreatic enzyme levels, and evidence of pancreatitis through USG investigation, which strongly suggested AP. They presented an episode of AP following PD-related peritonitis. All received prophylactic antibiotics, analgesics, and TPN support, while none of the patients required intensive care support or intervention. Both patients required a dialysis shift from PD to HD. Both children were hospitalized for about 3 weeks, and we are still under continuous observation for recurrent AP episodes, pancreatic pseudocyst formation, and necrotic complications for the next few months.

The prevalence of AP is higher in patients who are on dialysis than that of those in the general population.[4] Sharma et al. reported an incidence of 50 AP episodes per 1,000 patient-years among ESKD children in 8 years of observation.[7] The Italian Registry of Pediatric Chronic Dialysis reported an incidence rate of 9.5 AP episodes per 1,000 patient-years in PD and HD patients, with an incidence rate of 6.2 AP episodes per 1,000 patient-years in PD patients.[4] At the time of writing this report, we did not find any other studies in Indonesia that reported the incidence of AP in the pediatric ESKD population. This is the first report that describes two episodes of AP among children undergoing CAPD in our center, resulting in an incidence rate of 0.1 AP episodes per patient-year.

AP among patients with ESKD cannot be attributed only to one specific risk factor.[5] Some established factors suggest that PD patients are more prone to AP, including toxic substances in PD components, local hypercalcemia in the pancreas due to calcium in PD solution, PD-related peritonitis, and high intra-abdominal pressure caused by peritoneal fluid.[4–6] The concentration of glucose used in the dialysate may cause hyperglycemia and hypertriglyceridemia, which are associated with pancreatitis.[4,5] Children on dialysis are also frequently exposed to medications, including Valproic acid and ACE inhibitors known to be associated with AP. These drugs may increase bradykinin, a vasoactive substance that induces angioedema, which can cause pancreatic duct obstruction followed by enzyme leakage.[4] Both our patients used 1.5% dextrose solution as the dialysate. However, they had a normal level of triglycerides and blood glucose. They also had never been exposed to Valproic acid and ACE inhibitors.

Despite its rarity, hyperparathyroidism has been known for its association with pancreatitis incidence.[4,8–10] Hypercalcemia secondary to the secretion of parathyroid hormone (PTH) plays a major role in the pathogenesis, but other mechanisms may be involved. The elevation of PTH usually leads to hypocalcemia and hyperphosphatemia in CKD.[11] However, children on dialysis with the adynamic bone disease might develop severe hypercalcemia. Serum calcium levels can increase affected by dialytic calcium exposure and peak with high peritoneal dialysis fluid turnover.[11] Hypercalcemia leads to vascular calcification and calcium deposition in the soft tissue, thus increasing the risk of AP.[4,12] Both our patients had hyperparathyroidism suggested as a risk factor for AP. Nevertheless, it was not presented along with hypercalcemia. Thus, we propose that genetic factors or other mechanisms might play a role and therefore need further studies.

Increased gastrointestinal hormones such as cholecystokinin, gastric inhibitory

polypeptide, and glucagon in patients with ESKD can stimulate hypersecretion of pancreatic enzymes, especially trypsin which can contribute to the pancreatic morphological changes as well as functions and predispose AP occurrence.[4,6] In addition, recurrent PD-related peritonitis and intraperitoneal administration of antibiotics or anticoagulants may contribute to pancreatic autodigestion, leading to AP.[13] The suspicion of pancreatitis or other intra-abdominal pathology is high in patients with severe or persistent PD-related peritonitis.[4] Peritonitis secondary to fungal infection is infrequent, and the prognosis is generally poor in children with PD.[14] Catheter removal is considered the key therapy in PD-related peritonitis due to fungal infection.[14,15] Sato et al. reported that acute pancreatitis was associated with micafungin.[16] However, no other studies or clinical experiences of AP are associated with micafungin. Our patient in case 1 had a history of fungal peritonitis due to *Candida krusei,* which was treated with intravenous micafungin. Therefore, we suspected that micafungin might increase the risk of AP in this patient. While in case 2, we presumed that the occurrence of AP was due to unspecified abdominal symptoms that may overlap with other intraabdominal problems and her history of recurrent PD-related peritonitis. Moreover, her laboratory and radiology findings were highly suggestive of AP.

The common risk factors of AP in children are gallstones and autoimmune disorders such as Systemic Lupus Erythematosus (SLE).[17,18] A retrospective cohort study showed SLE patients who received MP pulse had a 2.39 times higher risk for infection than those who did not get MP pulse (p=.017, 95%CI 1.19-4.82). The onset of infection occurred < 6 months in 50% patients and > 1 year in 36.7% patients. The use of very high dose steroids (300-1000mg/day) can activate nongenomic pathways that cause immunosuppression and anti-inflammatory effects.[19,20] Regarding the infective complication, SLE patients with CAPD, are more susceptible to peritonitis than other CAPD patients.[19,20] Peritonitis can induce exacerbations of the disease and may contribute to the development of other serious complications. Therefore, these indicate that PD may not be the first choice for kidney replacement therapy in lupus patients undergoing immunosuppressive therapy.[19,21] The patient in case 2 was diagnosed with Lupus nephritis. She had received an intravenous MP pulse for six cycles and undergone CAPD for six months when she got the first episode of PD-related peritonitis. Two weeks after discharge, she was readmitted with a second episode of peritonitis coexistence with AP. This patient tends to be susceptible to infection. Thus, we consider switching the dialysis modality from PD to HD permanently.

The clinical manifestations of acute pancreatitis in dialysis patients do not differ from those in the general population.[5] The diagnosis of AP is generally based on the revised Atlanta classification in which it must fulfill two of the following three features: (a) abdominal pain; (b) elevation of serum amylase and lipase levels over three folds greater than the upper limit of normal; and (c) radiological evidence of AP by USG, CT scan, or MRI. Other signs and symptoms like nausea, vomiting and abdominal rebound tenderness may present.[22] Abdominal CT scan or MRI imaging is the gold standard for AP diagnosis. At the same time, USG is more widely available than other modalities since it is safe, non-invasive, and does not utilize radiation. However, USG accuracy is limited and often tricky due to the retroperitoneal organ location, overlying bowel gas, or an obese patient. The typical USG images of AP are decreasing in echogenicity and increasing in volume as the pancreatic body may exceed 2.4 cm in diameter with marked anterior bowing and irregular surface.[4,23] Our patients met all AP criteria as they had abdominal pain, threefold pancreatic enzyme elevation, and signs of AP confirmed by USG examination. We did not perform a CT scan or MRI since USG had revealed AP and did not show evidence of other pancreatic anomalies which could predispose to AP or other complications.

The management of AP consists of conservative and interventional treatments. Conventional treatment includes fasting, TPN, prophylactic antibiotics, and analgesic.[4,24] Fasting is the first step to resting the pancreas, especially if the patient presents vomiting. By resting the bowel, the pancreas allows it to rest. Thus, it heals fast. Delivering nutrition via parenteral support using TPN is recommended. Typically, patients can be transitioned to clear oral liquid when pain is well controlled, then to low-fat full liquid and a regular low-fat diet.[24] In severe cases, an analgesic can be given to relieve pain.[24,25] Antibiotic prophylaxis was needed to decrease the risk of severe pancreatic infections. The early use of antibiotic prophylaxis known to penetrate necrotic tissue such as Carbapenems, Quinolones, and Metronidazole is recommended for AP to reduce mortality and lower septic complications.[4,22,24,25] The use of somatostatin and pancreatic protease inhibitors for AP did not find consistent clinical benefits in children.[25,26] Both our patients received conservative treatment. Fasting and TPN for several days successfully decreased amylase and lipase serum in these patients. Intravenous fluid administration helped maintain adequate fluid status and urine output. We administered prophylactic antibiotics for our patients based on updated data on trends of organism incidence and susceptibility to antimicrobial agents in our setting since there are no guidelines stating specific antibiotic prophylaxis for AP in children with CAPD.

Immediate intervention is not required for patients with asymptomatic fluid collection or necrosis.[25] Percutaneous CT-guided aspiration or necrosectomy is necessary for patients with severe pancreatitis and infected necrosis or persistent fluid collection. In adults, surgical management is indicated for those who develop pancreatic abscess or necrosis or for the diagnostic approach.[26] Pediatric literature regarding the indications for surgery is limited. These patients did not need further intervention, including interventional radiology, endoscopy, or pancreatic surgery, since AP improved by conservative treatment.

Acute pancreatitis can vary from a mild mortality rate of less than 1% and typically resolves in several days to severe with a mortality rate up to 30%.[22] The progression to a severe state is caused by the alteration

in the microcirculation of the pancreas resulting from hypovolemia, increased capillary permeability, and microthrombi formation. The Italian Registry of Pediatric Chronic Dialysis reported that all PD patients with AP were hospitalized for a median time of 20 days, and the mortality rate was high.[4] Mortality rates are highest in patients with hemorrhagic pancreatitis, necrotizing pancreatitis, and multiorgan dysfunction.[22,26] We did not find evidence of hemorrhagic and necrotizing pancreatitis in our patient.

Moreover, we did not obtain any signs of multiorgan dysfunction. Both of our patients recovered uneventfully and were discharged after three weeks of hospitalization. We continue life-long evaluation of recurrent AP episodes, pancreatic pseudocyst formation, and necrotic complications.

During the period of prolonged lockdown, it became evident that the potential harms of COVID-19 to pediatric CAPD patients are not only from the virus itself but also from being kept at home and away from healthcare services. Information, support, and monitoring needs for patients and their parents during this period are crucial. Relevant strategies need to be formulated and implemented in response to the COVID-19 pandemic, including providing patients and caregivers with adequate PD care guidelines, assistance, and robust telehealth.[27–29] Further development regarding the retraining procedure is needed.[30] An observational survey revealed that hospitals that conduct retraining for PD patients had lower complication rates than those that did not provide retraining.[31] Before the pandemic, PD nurse home visits has significant benefits. The home visiting program needs to adapt its strategy from traditional to virtual home visits.[32] Since both our cases occurred during the COVID-19 outbreak, we suspected these conditions were due to healthcare service restriction and lack of monitoring affecting the patient's adherence to PD protocol. Hence, we should propose innovative clinical management strategies to ensure proper patient treatment and reduce the risk of SARS-CoV-2 transmission.

CONCLUSION

Acute pancreatitis is an infrequent but severe complication in chronic PD patients. In our patient, recurrent PD-related peritonitis was likely a major contributing factor. Being exposed to micafungin in case 1 and having an autoimmune disorder in case 2 heightened their risk for AP. Moreover, poor adherence, lack of monitoring, and healthcare service restriction may also contribute to increased AP occurrence. Management for AP, including conservative treatment and therapy for any underlying cause, successfully treated both patients. Furthermore, innovative clinical management strategies for pediatric patients undergoing CAPD in response to the COVID-19 pandemic crisis are urgently needed to prevent severe complications and improve patient care quality.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

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ETHICS APPROVAL

The patient's family gave permission for patient information to be published in scientific journals anonymously.

AUTHOR CONTRIBUTION

All authors contributed equally in the writing of this case report

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