Pancreatic Pseudocyst with Colonic Perforation Complication

Angga Pramudita, Marcellus Simadibrata**, Achmad Fauzi**, Nisan Soeheri**

* Department of Internal Medicine, Faculty of Medicine, University of Indonesia
Dr. Cipto Mangunkusumo General National Hospital, Jakarta
** Division of Gastroenterology, Department of Internal Medicine, Faculty of Medicine
University of Indonesia/Dr. Cipto Mangunkusumo General National Hospital, Jakarta

ABSTRACT

Pancreatic pseudocyst is one of complications of both acute and chronic pancreatitis. It is a rare clinical condition. The incidence is low ranging between 1.6 and 4.5%, or 0.5-1 per 100,000 adults annually. The clinical manifestations range from asymptomatic to severe acute abdomen due to complications. Acute complications may include bleeding, infection, rupture and perforation of the gastrointestinal tract; while chronic complications are gastric and biliary obstruction as well as thrombosis of portal vein.

We present a case report of a 38-year-old male with complaints of abdominal pain, fatigue, nausea and vomiting containing undigested food and yellow liquid. On clilnical examination, the patient was found to be fatigue, having enlarged abdomen, unpalpable liver and spleen, no signs of shifting dullness was detected. We found an abdominal mass in the left upper and lower quadrant sized 20 x 10 cm accompanied with epigastric pain on palpation. Abdominal ultrasonography revealed a cystic lesion on the head of pancreas with differential diagnosis of pseudocyst. The abdominal computed tomography (CT-scan) showed a lesion arising from pancreas, extending into abdominal cavity and part of left groint and attaching to left intestinal in the abdomen, part of gastric region and left diaphragm. A diagnosis of pancreatitis was suspected with differential diagnosis of pancreatic mass and peritonitis. It is a case report of pancreatic pseudocyst with acute complication of colonic perforation.

Keywords: *pseudocyst, pancreatitis, pancreatitis complication, colonic perforation*

ABSTRAK

Pseudokista pankreas merupakan salah satu komplikasi dari pankreatitis akut maupun kronik. Pseudokista merupakan suatu kondisi klinis yang jarang terjadi. Angka kejadian pseudokista pankreas rendah hanya berkisar 1,6-4,5%, atau 0,5-1 per 100.000 orang dewasa per tahun. Gambaran klinis pseudokista pankreas dapat berkisar dari asimtomatik sampai akut abdomen yang berat akibat komplikasi. Komplikasi akut yang terjadi antara lain perdarahan, infeksi, ruptur dan perforasi ke dalam saluran gastrointestinal, sedangkan komplikasi kronis antara lain obstruksi gaster, obstruksi bilier, dan trombosis pada vena porta.

Pada laporan kasus ini dipaparkan seorang laki-laki 38 tahun dengan keluhan nyeri perut, lemas, mual, dan muntah berisi makanan dengan cairan berwarna kuning. Pada pemeriksaan fisik menunjukkan abdomen buncit, lemas, hepar dan lien tidak teraba, tidak terdapat shifting dullness, teraba masa pada abdomen kuadran kiri atas dan bawah ukuran 20 x 10 cm dengan nyeri tekan pada ulu hati. Hasil ultrasonografi (USG) abdomen menunjukkan adanya kista di kaput pankreas dengan diagnosa banding pseudokista. Hasil computed tomography scan (CT-scan) abdomen terlihat lesi yang berasal dari pankreas meluas mengisi rongga abdomen dan sebagian pelvis sisi kiri, perlekatan dengan usus-usus di sisi kiri abdomen serta sebagian gaster dan diafragma kiri suspek pankreatitis dengan diagnosa banding massa di pankreas dengan peritonitis. Pada kasus ini terjadi pseudokista pankreas dengan komplikasi akut perforasi kolon.

Kata kunci: *pseudokista, pankreatitis, komplikasi pankreatitis, perforasi kolon*

INTRODUCTION

Pancreatic pseudocyst is one of complications of both acute and chronic pancreatitis.[1] It is a rare clinical condition. The incidence is relatively low ranging between 1.6 and 4.5%, or 0.5 - 1 per 100,000 adults annually.

Pseudocyst is more likely due to chronic rather than acute pancreatitis. The incidence of pancreatic pseudocysts caused by chronic pancreatitis is approximately 30-40%; a study in Scotland reported that of all pancreatic pseudocysts cases, about 7-10% cases are complications of acute pancreatitis. Pancreatic pseudocyst is the most common pancreatic cystic lesion with incidence of 70-80%.[1,2]

The clinical manifestations range from asymptomatic to severe acute abdomen due to complications. Acute complications may include bleeding, infection, rupture and perforation of the gastrointestinal tract; while chronic complications are gastric and biliary obstruction as well as thrombosis of portal vein.[1,3]

Initial diagnosis is established by using computed tomography scan (CT-scan) or magnetic resonance imaging (MRI).[1,4] Aspiration endoscopy may be performed to differ pseudocyst from other pancreatic cystic lesion. Most of pseudocysts may have spontaneous regression. The size of pseudocyst may not be an indicator of resolution or complication.[1]

CASE REPORT

A 38-year-old male came with abdominal pain since seven days before hospital admission. The abdominal pain was stabbing on all abdominal quadrants and spread to the left groin. The patient was also found to be fatigue, had nausea and vomiting as many as two times containing undigested food an yellow liquid.

Since two months before admission, he had epigastric pain and bloating. The abdominal pain worsened on the seventh day before admission. He admitted to have constipation, i.e. only had one defecation in a day, but since seven day before admission, he had 2-3 times defecation daily. He had normal urination with adequate amount of urine and no pain. However, since seven days before admission, he had infrequent urination. There was no fever, chest pain and short of breath.

The patient gave history of diabetes mellitus (DM) and hypertension, which had been diagnosed since 5 years ago. The disease was uncontrolled and no regular treatment was taken. On admission at the emergency room of Cipto Mangunkusumo hospital, he had a blood glucose level 57 mg/dL with no signs of cold sweat and fatigue. He gave no history of pulmonary or cardiac disease. He has been smoking 2 packs daily for 15 years. He was alcoholic with drinking habit 1-2 bottle(s) of beer daily. His father also had hypertension. There was no diabetes mellitus, hypercholesterolemia, heart disease or sudden death in his family.

On clinical examination, the patient was found to be fully alert with moderate illness. His vital signs were blood pressure 150/80 mmHg, pulse rate 92 times/minute, temperature 37.3ºC, respiratory rate 24 times/minute. Abdominal examination revealed soft, enlarged abdomen, unpalpable liver and spleen, no shifting dullness and an abdominal mass in the left upper and lower quadrant sized 20 x 10 cm with epigastric pain on palpation. There were no evidences of enlarged lymph nodes either on suboccipital, preauricular, submandibular, submental, colli, supra- and infra-clavicula as well as on the axilla and inguinal nodes. The extremities were warm, no edema and had good peripheral perfusion.

Laboratory investigations revealed a hemoglobin level 10.7 g/dL, mean corpuscular volume 31 fL, mean corpuscular hemoglobin 29%, hematocrit 30%, total leukocyte count 12,400/mm^3, platelet count 362,000/mm^3, random blood glucose level 57 mg/dL, aspartate aminotransferase/alanine aminotransferase (AST/ALT) level of 23/20 U/L, natrium level of 135 mmol/L, potassium level 4.2 mmol/L, chloride level 99 mmol/L, ureum level 124 mg/dL and creatinine level 7.1 g/dL.

The patient was treated with soft diet for diabetic, i.e. 1,800 kcal, 60 g protein and low-fat food. Medication were administered including rectal suppository of ketoprofen, omeprazole 1 x 40 mg dose intravenously, sucralfate suspension 4 x 15 cc dose, furosemide 1 x 40 mg dose intravenously, ceftriaxone 1 x 2 g dose intravenously, azithromycin on dose 1 x 500 mg by oral route, ambroxol suspension 3 x 15 cc, acetyl salicylic acid 1 x 80 mg and insulin drip 1 unit/hour dose (corrected dose).

On the 2nd day of hospitalization, the patient had a fever, worsened abdominal pain, nausea and vomiting containing green fluid. Clinical examination revealed blood pressure 130/80 mmHg, febrile condition (38.7˚C), tachypnea with respiratory rate 30 times/minute, tachycardia with pulse rate 108 times/minute. Laboratory investigation showed high value of amilase and lipase levels, i.e. 324.2 U/L and 226 U/L, respectively. The patient was diagnosed with sepsis due to acute pancreatitis. A nasogastric tube (NGT) was inserted revealing green colored fluid. The patient was given total parenteral nutrition (triofusin E: aminovel/12 hours, NaCl/8 hours). On the third day, a serial examinations of ultrasonography, abdominal CT-scan and emergent hemodialysis was performed. Abdominal ultrasonography showed a cystic lesion on the head of pancreas with a differential diagnosis of pseudocyst. The possibility of pancreatitis could not be excluded. There were

dilatation and intestinal thickening which were the characteristics of obstruction. There was hepatomegaly and fatty liver along with a diagnosis of chronic liver disease (CLD); cutaneous/subcutaneous edema was evident on the left back; while both kidneys were in good condition (Figure 1).

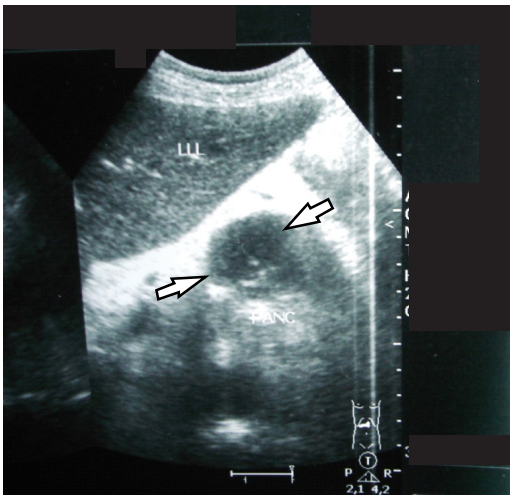

Figure 1. Abdominal ultrasonography showed hypoechoic lesion on the head of pancreas

Barium enema evaluation showing dullness on lower abdominal quadrants with suspected intra-abdominal mass (Figure 2). On figure 3, the abdominal CT-scan revealed a lesion arising from pancreas, extending into abdominal cavity and part of left groint, attaching to left intestinal in the abdomen, part of gastric region and left diaphragm. A diagnosis of pancreatitis was suspected with differential diagnosis of pancreatic mass and peritonitis. There were bilateral pleural efusion and suspected pneumonia of the left lung. The pathology anatomy examination of the pancreatic tissue showed necrotic tissue caused by the pancreatic enzymes.

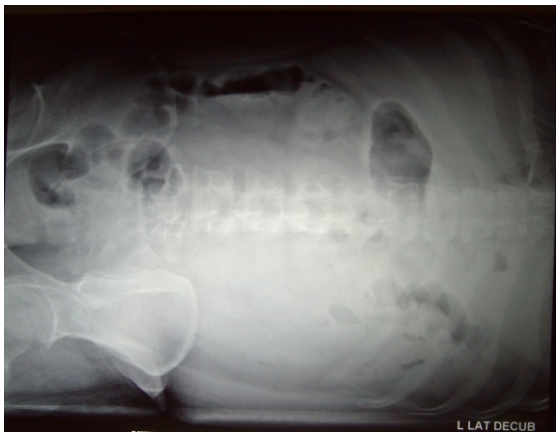

Figure 2. Barium enema evaluation showed dullness on lower abdominal quadrants with suspected intra-abdominal mass

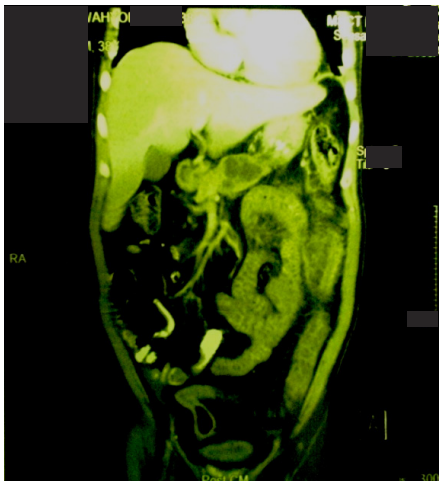

Figure 3. Abdominal scan showed a lesion arising from pancreas

On the 17th day of hospitalization, we repeated the ultrasonography and investigated the amilase and lipase levels. The ultrasonography revealed a hypoechoic lesion on the head of pancreas sized 2.9 x 2.4 cm and left pleural efusion. Other intra-abdominal organs were within the normal range.

On the 20th day of hospitalization, the patient had short of breath and fever. The chest X-ray demonstrated cardiomegaly, lung edema and left pleural efusion (Figure 4). The patient had 2 ampules extra doses of furosemide injection.

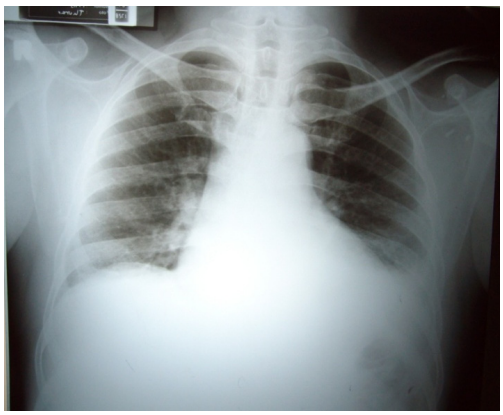

Figure 4. The chest X-ray showed infiltrate and cardiomegaly

In sputum culture, we found *Klebsiella pneumoniae*, which was sensitive to gentamicin, amikacin, meropenem, and imipenem. We found no pathogenic microorganism in urine culture. Echocardiography revealed concentric hypertrophy, hypokinetic at the anterolateral of basal/apical segement, grade 2 diastolic dysfunction, normal systolic function of left ventricle with 54% ejection fraction and minimal pericardial efusion.

On the 31st day, an explorative laparotomy was performed since the patient had generalized peritonitis and suspected visceral perforation. By laparotomy,

we found intestinal adhesion which then was managed by intestinal adhesiolysis. An exploration was later performed by tracing the edge of drainage and we found perforation of descendent colon > 50% of intestinal lumen circumference. A resection was performed on the perforated descendent colon and stoma was made on the transversal colon. The patient was discharged from hospital by his own request on the 53rd day of hospitalization. However, we still monitored the patient even he had been home. The patient reported that there was self-loosening drain and no liquid production. Based on results of consultation from outpatient clinic of the Department of Surgery and Endocrinology, he was suggested to be treated at the Kidney Hypertension Outpatient Clinic.

DISCUSSION

Pancreatic pseudocysts are localized fluid collection containing amylase and other pancreatic enzymes. They are surrounded by fibrous tissue that has non-epithelialized lining.[3] Pseudocysts are connected to the pancreatic ductal system, both directly and indirectly through pancreatic parenchyma. Such connections are caused by damage of pancreatic duct due to increased pressure. The pressure is resulting from stenosis, stone or protein collection that obstruct the main pancreatic duct or it may be caused by pancreatic necrosis following the acute pancreatitis attack.[1]

Pseudocysts occur in parallel with pancreatitis. The etiology of pseudocyst is similar to the cause of pancreatitis and chronic pancreatitis is more common than acute pancreatitis. Pseudocysts are also more common in patients with alcohol-induced pancreatitis than the non-alcoholic patients. Pancreatitis associated with alcohol is a major cause in 59-78% of pseudocysts overall.[1] A study conducted by Whitcomb reported that there are some causative factors in pancreatic pseudocysts including alcohol (70%), biliary tract disease (8%), blunt trauma (5%), penetrating trauma (1%), deep operative trauma (0.3%), and idiopathic (16%). Most patients were in the idiopathic group and were thought to be associated with alcohol, but no definite evidence was documented.[5]

In the present case, we assume that the cause of pancreatic pseudocyst is due to alcohol consumption. The patient has been consuming alcohol 1-2 bottle(s) of beer daily since he was 18-years old. Alcohol exerts a number of effects causing abnormal sphincter of Oddi motility, direct toxic effect on pancreas and duct obstruction by protein plug formation.[6] The patient had complained about epigastric pain since 2 months before admission. He did not visit any doctor and therefore the cause of such symptom could not be revealed. Thus, acute or chronic pancreatitis could not be excluded as the causative factors of pseudocyst in the patient. In addition, the ultrasonography revealed that there was no evidence of biliary stone, which may become a risk factor of pancreatitis.

The pathogenesis of pseudocysts seems to stem from disruptions of the pancreatic duct due to pancreatitis or trauma followed by extravasation of pancreatic secretion. Two thirds of patients with pseudocysts have demonstrable connections to the pancreatic duct. In the other third, an inflammatory reaction is supposed to have sealed the connection so that it is not demonstrable.[1] Pseudocyst develops after acute pancreatitis, when the collection of fluid has last more than 4-6 weeks. If it has been surrounded by granulation and fibrous tissue, we could say that acute pseudocyst has occurred. Pseudocyst usually contains enzymatic material and necrotic tissue.[3]

The pathogenesis of pseudocyst formation in chronic pancreatitis is more difficult to be understtod, but at least two mechanisms may be involved. Pseudocyst may develop as a result of an acute exacerbation of the underlying disease; or it may occur due to obstruction of a major branch of a pancreatic duct by protein plug, calculus or fibrotic tissue.[1]

The clinical manifestations of pancreatic pseudocyst may range from asymptomatic to abdominal disorder due to complications. According to literatures, there is no pathognomonic symptom of pancreatic pseudocyst. The usual signs and symptoms include persistent abdominal pain more than 3 weeks that occur in 80-90% patients. Other symptoms are nausea, vomiting and weight loss in 40-50% patients. Other signs such as jaundice and pruritus may develop when there is an obstruction in biliary duct. Complication of pseudocysts may occur in 9% patients.[1,3,6]

In the present case, the patient had already suffered from epigastric pain and bloating since two months before admission. He had complained abdominal pain since 7 days before admission. The pain was accompanied with nausea and fatigue. He also had vomiting as many as two times containing undigested food an yellow liquid. One day before admission, the complaints worsened, his abdomen became more painful, enlarged and had tense abdominal distention.

There are no specific physical findings for pancreatic pseudocysts. On palpation, 50% patients would have a mass in upper abdominal quadrants.[4] Peritoneal signs indicates the occurrence of ruptured pseudocyst or infection. Other findings include fever, jaundiced sclera or pleural efusion.[1]

On clinical examination, we found a mass measuring 20 x 20 cm with indistinct border in the left abdominal region. The patient denied any tenderness on palpation. The abdomen was distended. We found

reduced vesicular sound on the left lung and rales on the basal area of the lung, but no wheezing was found. The chest X-ray revealed cardiomegaly, lung edema and left pleural efusion.

Laboratory investigations may include unspecific findings for pancreatic pseudocyst. Amilase and lipase level may sometimes increase, but usually still within the normal range. Serum bilirubin level and liver function test may increase when there is duct obstruction by stone or pressure of pancreatic pseudocyst or due to underlying liver disease such as hepatitis. Some laboratory investigations may provide some clues of underlying cause in pancreatitis, i.e. the increased triglyceride or calcium level. Elevated levels of blood chemistry tests may increase our suspicion on the occurrence of biliary pancreatitis.[1] In the present case, the laboratory results indicated elevated amilase and lipase levels although the patient had no jaundice. The trigylceride level was not relatively high. There was signs of infection and the AST/ALT level were normal.

The abdominal ultrasonography revealed anechoic image with distal acoustic enhancement. The pseudocysts are round or oval and contained within wall. At the initial phase of its developement, pseudocyst may appear more complex with various internal echoic level. It may even seem more complex if there is any bleeding or infection of pseudocyst. Color doppler or duplex scanning should be always performed for a cystic lesion in order to confirm that the lesion is not a giant pseudoaneurysm. USG has high sensitivity of 75-90% in detection of pseudocyst. However, it is lower than CT scan 90-100% sensitivity and 98% specificity.[1,4]

CT-scan typical findings for pancreatic pseudocysts in patients with history of pancreatitis may reveal a round mass containing fluid with thick wall. Any CT-scan finding which is consistent with the characteristic of pseudocyst may not need other investigation for confirmation. There is a major weakness of CT-scan. It cannot differentiate a pseudocyst from neoplasm, particularly mucinous cystadenoma and intraductal papillary mucinous neoplasm (IPMN). The contrast agents given during CT-scan may impair renal function. MRI and magnetic resonance cholangiopancreatography (MRCP) are sensitive procedures for pancreatic pseudocysts. Such procedures are usually not routinely performed since CT-scan may have already provide the necessary diagnostic information.

The MRI should be performed in sub-acute pancreatitis cases to prevent any complication of infection such as tissue debris or necrotic tissue.[7] Endoscopic retrograde cholangiopancreatography (ERCP) may not be necessary in establishing diagnosis of pseudocyst; however, it may become definitive treatment in some cases. It should be performed before any surgery.[1,4]

Abdominal ultrasonography in the present case revealed a cystic lesion on the head of pancreas, which suggested the diagnosis of pseudocyst and there was no findings of stone in the gall bladder or biliary duct. The abdominal CT-scan showed a lesion arising from pancreas, extending into abdominal cavity and part of left groint and attaching to left intestinal in the abdomen, part of gastric region and left diaphragm. A diagnosis of pancreatitis was suspected with differential diagnosis of pancreatic mass and peritonitis.There were also bilateral pleural efusion and suspected left pneumonia.

Supportive care is routinely provided for patients with pseudocyst including intravenous fluid, analgesics and antiemetics. Patients who are still able to have oral intake are suggested to have low fat diet. For patients who could not have oral intake, the diet is given through NGT or TPN. Pseudocyst may resolve with supportive care. Some studies showed that the size of cyst and the range duration of symptoms are not helpful indicators in predicting resolution or complication of pseducysts.[1,3,8]

Drainage procedure should be performed when there is any symptom of complication including infection, bleeding, gastric outlet or biliary obstruction. Until now, no study has been conducted to directly observe the best drainage technique for pseudocysts. It may be percutaneous, surgical or endoscopic. Based on literatures, endoscopic drainage is the most common approach.[1]

During the hospitalization, the patient was treated with soft diet for diabetic, i.e. 1,800 kcal, 60 gram of protein and low-fat food. The patient had IVFD, i.e. NaCl 0.9% every 8 hours, rectal suppository of ketoprofen, omeprazole 1 x 40 mg dose intravenously, sucralfate suspension 4 x 15 cc, furosemide 1 x 40 mg dose intravenously, ceftriaxone 1 x 2 g dose intravenously, azithromycin on dose 1 x 500 mg by oral route, ambroxol suspension 3 x 15 cc, acetyl salicylic acid 1 x 80 mg.

On the second day of hospitalizaiton, the patient was diagnosed with acute pancreatitis. The patient was suggested to fast and NGT was inserted. Total parenteral nutrition was administered subsequently. On the 10th day, the patient went on laparatomy to have debridement procedure surround the pancreatic pseudocyst and drainage was performed due to worsen clinical condition.

Complications of pancreatic pseudocysts include spleen complication such as massive bleeding, sepsis and thrombosis, ruptured pseudocyst, bleeding due

to vascular erosion of the pseudocyst, infection, biliary complication (obstructive jaundice) and portal hypertension due to compression or obstruction of portal vein. When there is any spleen complication, resection of pseudocyst is performed as a treatment by splenectomy and distal pancreatectomy. For bleeding case, radiologic imaging may be helpful to determine the source of bleeding. Surgery is the treatment of choice for such case.[3,9,10]

In the present case, there was acute pancreatitis and complication of infected pseudocyst with colonic perforation. The literatures reveal that colonic perforation in patients with infected pancreatic pseudocyst is very rare.[8,11] Our data analysis demonstrates that the incidence of colonic perforation in acute pancreatitis is about 3.3 to 15%. Little has been known about the complication; therefore, the diagnosis and treatment of similar cases are still controversial among the experts.[12]

Although there have been several theories on how pancreatic pseudocysts rupture into the colon, the exact pathogenesis remains unknown. The first theory is about perforation at the splenic flexure resulted from direct pressure exerted by a giant pseudocyst. The second theory suggests that there is mesenteric ischemia due to intravascular volume depletion caused by severe acute pancreatitis in patients who developed shock. It may contribute to colonic infarction and perforation. Aldridge et al, reported that colonic complications due to severe acute pancreatitis are more common at the splenic flexure, where it closely abuts the pancreatic tail. They also reported that in addition of ischemia at the splenic flexure, there is also direct enzyme activity from the dispersion of ruptured pancreatic pseudocyst causing inflammation and colonic necrosis due to thrombosis of mesenteric and submucosal vessels. The hypothesis is based on presumption that inflammation and edema in mesocolon are worsened by systemic hypotension which induces thrombosis leading to retardation of blood flow in the mesenteric vessels.[11,12]

CT-scan is utilized to monitor the progression of disease as well as the complications, both systemic and local.[13] In the present case, the CT scan showed complication of colonic perforation, which assumed resulting from dispersion of ruptured pancreatic pseudocyst (Figure 1). To confirm the diagnosis, we conduct pathology anatomy examination, which revealed tissue necrosis caused by pancreatic enzyme.[14] Colonic perforation had also manifested as peritonitis and therefore, explorative laparatomy was performed.

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Correspondence:
Marcellus Simadibrata
Division of Gastroenterology, Department of Internal Medicine
Dr. Cipto Mamgunkusumo General National Hospital
Jl. Diponegoro No. 71 Jakarta 10430 Indonesia
Phone: +62-21-3153957 Facsimile: +62-21-3142454
E-mail: marcellus_sk@yahoo.com