PATIENTS WITH ACUTE PANCREATITIS SUSPECTED OF ABDOMINAL ABSCESS

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ABSTRACT

Acute pancreatitis has high morbidity and mortality. We report the case of a 62-year-old woman with acute pancreatitis suspected to be caused by an abdominal abscess. The diagnosis is made based on history, physical examination, laboratory examination, and radiological examination. From the anamnesis, the patient complains of upper right abdominal pain, the pain gets worse when lying on his back, decreases when he bends down and bends the knee. Laboratory tests showed elevated serum amylase and lipase levels. Abdominal ultrasound examination (Ultrasonography) obtained thick fluid in the abdominal cavity, MSCT (Multislice Computerized Tomography) with a diagnosis of multiple intra-abdominal abscesses. Patient with a history of gallstone surgery 1 week ago. The prognosis for Dubia is ad bonam.

Keywords—Acute pancreatitis, abdominal abscess, Acute pancreatitis management

I. INTRODUCTION

Acute pancreatitis is the leading cause of gastrointestinal hospitalization in the United States. Its incidence has increased over the last few decades and is estimated at 38 per 100,000 population per year.¹ Acute pancreatitis is mostly caused by gallstones by 45% and alcohol by 35%, hypertriglycerides about 1-4%, while in 10% of other causes such as infection, hypercalcemia, etc.² Pancreatitis due to infection can be caused by viruses, bacteria, fungi, parasites. Infection in pancreatitis is a secondary event. Evidence suggests that bacteria from the gastrointestinal tract translocate to necrotic tissue infections of the pancreas (necrotic pancreas) and extra pancreas (cholangitis, catheter-acquired infections, bacteremia, urinary tract infections and pneumonia). The mechanism of pancreatitis caused by infection is not known with certainty.³

We report a patient with acute pancreatitis thought to be due to an abdominal abscess

II. CASE DESCRIPTION

A 62-year-old woman was referred to Dr. Soetomo's hospital with complaints of upper right abdominal pain since 1 week. The pain was getting worse 3 days before admission to the hospital, the patient thought it was pain after gallbladder surgery. The pain is like stabbing and is felt continuously for the last 3 days. Pain is not related to activity, nor does it improve with rest. The pain gets worse when the patient lies supine, the patient is more comfortable if the position is bent and the knee is bent. The pain is constant with nausea, flatulence and sometimes vomiting. The patient also complained of fever.

Based on physical examination, general condition is weak, consciousness is good, and vital signs are normal. On examination of the head and neck, there was no anemia, slight icterus of the conjunctiva sclera, no cyanosis, no shortness of breath, no increase in jugular venous pressure and enlarged lymph nodes in the neck. On chest examination, no heart and lung abnormalities were found. On abdominal examination, there were surgical scars covered with sterile gauze, no pus, no hyperemia, no Cullen sign and Turner sign, normal bowel sounds, soft palpable, and no organomegaly.

Laboratory examination HB 11.7 g/dL; HCT 35.1%; WBC 24,000/µl, PMN 77.7%; PLT 390,000/µl; CRP 294.7 mg/L; Amylase 114 U/L; Lipase 485 U/L; AST 22 U/L; ALT 11 U/L; LED 40 mm/hour; Total Bilirubin 3.8
mg/dl; Direct Bilirubin 2.8 mg/dl; Indirect Bilirubin 1 mg/dl; albumin 2.8 g/dl; BUN 10 mg/dl; Creatinine 0.83 mg/dl; Sodium 137 mmol/L; Potassium 4.1 mmol/l; Chloride 101 mmol/L; random blood sugar 160 mg/dL; HBsAg is non-reactive.

BOF (Buich Over Sich) photo showed gloomy right lower abdomen suspicious of thick fluid, abdominal ultrasound: Ductus choledochus was not dilated, fluid in the intestine/duodenum was perivesical and seen fluid entering the proximal CBD (Common Bild Duct), there was loculated fluid collection intensity of thick echo fluid in the abdominal cavity; No signs of obstructive ileus, Gall bladder not visualized (post cholecystectomy), Pancreas normal. MSCT abdomen: CBD looks dilated and looks to empties into the duodenum, no stones visible, Pancreas: normal size and contour, normal parenchyma, no mass/cyst, normal pancreatic duct, Abscess formation of abdominal cavity, pelvic cavity, suspect signs of impending peritonitis, right abdominal wall abscess. The patient was referred with a diagnosis of multiple intra-abdominal abscesses.

Laboratory when the patient arrives at the hospital, Dr. Soetomo Hb 10.7 g/dL; WBC 25,300 /µl; HCT 35.1%; PLT 390,000/µl; PMN 82.9%; CRP 311.31mg/L; amylase 296 U/L; lipase 2,348 U/L; AST 34 u/L; ALT 16 u/L; Bilirubin Total 3 mg/dl; Direct Bilirubin 1.59 mg/dl; albumin 2.7 g/dl; PPT 11 (control12,7); APTT 21.1 (control 24.7); BUN 7 mg/dl; Creatinine 0.5 mg/dl; Sodium 132 mmol/L; Potassium 4.9mmol/L; Chloride 101mmol/L; ALP 277 IU/L; LDH 394 U/L. From the Thorax Photo: the heart and lungs did not show any abnormalities. The patient was diagnosed with multiple intra-abdominal abscess + acute pancreatitis + hypoalbumin. Urgent exploratory laparotomy + drainage, general condition improvement, O2 nasal 3 liter/minute, insert NGT (Nasogastric tube) and temporary fasting, rehydration with Ringer lactate 1000 cc, then maintenance with Nacl infusion 0.9% : RD5 2:2, Meropenem injection 1 g/8 hour intravenously, antrain 1 g/8 hour intravenously. Diagnostic planning: Blood sensitivity culture, pus culture, serial lipase amylase

Day 3 of treatment, complaints of pain, general condition is weak, Vital signs: BP 120/80 mmHg; HR 102 bpm; respiratory 20 cycles/minute, temperature 36.9°C; Laboratory examination HB 10 g/dl; HCT 30.5%; WBC 23,030/µl; PLT 380,000/µl; PMN 83.4%; random blood sugar 163 mg/dl; total cholesterol 162 mg/dl; HDL 13 mg/d; LDL 108 mg/dl; TG 121 mg/dl; AST 29 u/l, ALT 24 u/l, BUN 8 mg/dl; creatinin 0.48 mg/dl; sodium 134 mmol/l; potassium 4.6 mmol/l; chloride 100 mmol/l; albumin 2.7 g/dl; amylase 132 U/L, lipase 1420 U/L, PH 7.42, PCO2 38, PO2 76, HCO3 24, 6, TCO2 25.8, BE 0.1, SO2 95. Continued therapy and performed exploratory laparotomy surgery + abscess drainage (An abscess with necrotic tissue of the rectal cavity was found, associated with localized by connective tissue, peritoneum difficult to identify, Pus 400 cc The results of blood culture showed no bacterial formation, the results of gram pus culture, aerobic Escherichia Coli ESBL (+) was found. Planing antibiotic therapy remained sensitive, meropenem plus metronidazole 500 mg/8 hours intravenously. On the 10th day of treatment the patient had no complaints, vital signs stable, good general condition, dry wound, laboratory HB 10,4 g/dL, WBC 11,000/µl, Amylase 49 U/L, lipase 105 U/L. The patient was discharged with Amoxicillin 500 mg/8 hours, metronidazole 500 mg/8 hours and paracetamol 500 mg/8 hours.

III. DISCUSSION

Acute pancreatitis is an inflammatory disease of the pancreas with various clinical manifestations, ranging from mild to severe to often lead to death. Clinically, pancreatitis is characterized by acute abdominal pain accompanied by an increase in enzymes in the blood and urine. Based on the recommendation from the ACG in 2013, the diagnosis of acute pancreatitis is made if there are 2 of 3 criteria: 1). The presence of consistent abdominal pain, 2). Increased serum amylase and lipase >3x the upper limit of normal, 3). There are findings on imaging examinations (USG/CT Scan or MRI) that show the characteristics of acute pancreatitis 4.

In this case, a 62-year-old woman with complaints of sudden abdominal pain, felt intermittent, fever, jaundice and found that serum amylase and lipase increased more than 3 times above normal. This patient met two of the
three criteria for pancreatitis, according to the 2013 ACG guidelines, the patient was diagnosed with acute pancreatitis.

Radiological examinations to establish the diagnosis of acute pancreatitis include BOF, abdominal ultrasound, MSCT and Magnetic Resonance Imaging (MRI). BOF is an inexpensive screening test and can demonstrate pancreatic classification. Abdominal ultrasound is an important examination to determine the etiology of pancreatitis, which has a sensitivity of about 95% for detecting the presence of choledolithiasis, and 50% for detecting the presence of choledocholithiasis. MRCT is used to assess the severity of pancreatitis and assess the presence of complications of pancreatitis using the Balthazar score. MRI can be performed to detect the presence of biliary tract stones and other abnormalities in the biliary tract. Ultrasound and MSCT results show an abdominal abscess suspected of causing acute pancreatitis.

Once the diagnosis of pancreatitis is established, the next important thing to do is risk stratification to predict the severity of pancreatitis and the complications that may arise. Assessing the severity of acute pancreatitis at the patient's initial presentation is important for determining the prognosis, initial clinical management and determining which room the patient should be admitted to. The Atlanta criteria (2013) divide pancreatitis into mild, moderate and severe pancreatitis, based on the presence or absence of organ failure and local or systemic complications. Ranson's criteria can predict the patient's mortality score. Ranson's criteria assesses 5 parameters when the patient is admitted, namely age > 55 years, leukocytes > 16,000/ul, glucose > 200 mg/dl, serum LDH > 350u/L and AST > 250u/L. Meanwhile, 6 other criteria were assessed after 48 hours of treatment, namely hematocrit decreased by more than 10 points, BUN > 5 mg/dL, arterial PO2 < 60 mmHg, Serum calcium < 8 mg/dL, Base deficit 4 mEq/L, and fluid sequestration was estimated to be higher of 6L. Ranson's criteria with a value of more than 3 indicates severe pancreatitis.

Based on the Atlanta criteria, this patient had moderate-severe pancreatitis, where there was no organ failure but systemic symptoms such as sepsis. Meanwhile, with the Ranson criteria, the results obtained ranson 3 (aged 62 years, leukocytes 25,300 and LDH 394), so it includes severe pancreatitis with a mortality of 16%.

Trauma from sharp or blunt injuries to the pancreas can result in acute pancreatitis which has a high mortality rate. Trauma that occurs can result in rupture of the duct system in the pancreas which can result in the seepage of fluid (pancreatic juice). Parasitic infections (ascariasis, clonorchiasis), viruses (HIV, Hep A, B), bacteria (mycoplasma, tuberculosis, leptospira) can also cause acute pancreatitis. In this patient on ultrasound and MRCP examination, multiple intra-abdominal abscesses were found, on pus culture, abscess, necrotic tissue found Escherichia coli ESBL (+) which may be the cause of pancreatitis. And a history of abdominal surgery is very likely a factor that increases the risk of developing pancreatitis.

Conservative measures for the management of acute pancreatitis include: resting the pancreas by fasting; administration of strong analgesics; administration of total parenteral nutrition in the form of electrolyte fluid, nutrition, plasma protein fluid; and suctioning of gastric juices to reduce gastrin release from the stomach and prevent gastric contents from entering the duodenum to reduce stimulation of the pancreas. In accordance with the guidelines of the 2013 ACG, in the initial management of patients with acute pancreatitis, aggressive hydration should be undertaken once the diagnosis of acute pancreatitis is established. Hydrate with 250-500 ml of crystalloid solution per hour in all patients, except in patients with comorbid cardiac and renal disease. Aggressive hydration is beneficial in the first 12-24 hours. Strong analgesics such as pethidine are recommended, while morphine is not recommended because of side effects such as sphincter of Oddi's spasm. Patients are advised to fast completely to rest the pancreas. In moderate acute pancreatitis, oral feedings can be started as soon as possible when complaints of nausea, vomiting and abdominal pain subside, starting with a low-fat soft diet. Meanwhile, in severe acute pancreatitis, enteral nutrition is recommended to prevent infectious complications, with administration through a nasogastric tube or nasojejunum. Long-term parenteral nutrition should be avoided unless enteral nutrition is not tolerated or does not meet the required caloric requirements. Oral nutrition restriction (fasting) in cases of pancreatitis aims to rest the pancreas. The old paradigm of fasting until there is complete resolution of the pancreas has been abandoned. Recent studies suggest that prolonged fasting can cause atrophy of the intestinal mucosa and increase infectious complications. Due to the translocation of bacteria from the intestine. Early oral nutrition in patients with acute pancreatitis has been shown to reduce length of stay, reduce infection, reduce morbidity and mortality. Although the exact timing of starting oral nutrition is controversial, several studies suggest that it is safe to administer oral nutrition immediately to patients with moderate pancreatitis. The low-fat diet is said to be safe and provides more calories than the diet in liquid.
Routine use of antibiotics for prophylactic purposes in patients with severe acute pancreatitis is not recommended, only given to extra-pancreatic infections such as cholangitis, infections due to catheter insertion, bacteremia, urinary tract infections and pneumonia. Antibiotics that have been widely studied and penetrated into the pancreas are carbapenem, quinolones, metronidazole and high doses of cephalosporins. Based on research data, the most effective antibiotic is imipenem which is given at a dose of 0.5 g/8 hours intravenously. In this patient, the initial management was according to the guidelines, namely hydration, antibiotics and oral nutrition was given as soon as the complaints improved.

Interventional measures for pancreatitis are performed in cases of infected necrotizing pancreatitis, sterile necrotizing pancreatitis, with complications (e.g. bile duct obstruction, gastric outlet obstruction), multiple organ failure that does not improve with intensive therapy, symptomatic pancreatic pseudocyst, acute biliary pancreatitis with cholangitis and in cases of acute pancreatitis with gallstones. Cholecystectomy is useful when the stone in question is clearly visible on imaging, but in cases with sludge or microlithiasis the benefit is unclear. Meanwhile, open surgery is only performed if the facilities and resources are not available for minimally invasive procedures. Stable patients with infected necrosis should delay surgical, radiological, and/or endoscopic drainage interventions, preferably for four weeks. This allows time for the wall around the necrosis to develop. No need for intervention for asymptomatic pancreas and/or extrahepatic necrosis and/or pseudikist, regardless of size and location. This patient underwent exploratory laparotomy because there were multiple intra-abdominal abscesses causing acute pancreatitis.

IV. CONCLUSION

It was reported that a 62-year-old woman came with complaints of sudden severe abdominal pain, especially in the pit of the stomach. Laboratory examinations showed an increase in amylase and lipase enzymes and the presence of leukocytosis. The results of BOF, USG, MRCP showed the presence of multiple intra-abdominal abscesses. Previous patient with history of gallbladder surgery. The patient was treated for pancreatitis and given antibiotics according to the most likely cause of bacteria in the abdomen.

REFERENCES


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