ACUTE PANCREATITIS IN TYPE 2 DIABETES MELLITUS WITH MULTIPLE STONES IN THE CYSTIC DUCTUS

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ABSTRACT

Introduction: Severe upper abdominal pain is one of the symptoms of acute pancreatitis that often occurs in other diseases, such as peptic ulcer, acute hepatitis, cholangitis, and cholecystitis. Gallstone-induced acute pancreatitis has considerable morbidity and mortality.

Case illustration: A 50-year-old man came to the ED with a chief complaint of severe abdominal pain in the upper abdomen, VAS 8-9. His past medical history was unknown. On arrival, blood pressure was 141/102 mmHg, pulse was 98x/minute, and temperature was 36.4ºC. CT Scan of the abdomen showed pancreatitis and multiple stones in the cystic duct. Amylase and lipase serum results were 1897U/L and >3000 U/L.

Discussion: In acute pancreatitis caused by gallstones, stones usually get stuck in the branches of the pancreaticobiliary duct. Obstruction at this location causes reflux of bile into the pancreatic duct. Smaller gallstones are more likely to induce pancreatitis because they migrate more easily between bile ducts. This patient was suspected of having a previously unknown history of DM, which contributed to the patient's worsening condition.

Conclusion: Early management and treatment of triage, fluid resuscitation, and detection of local or systemic complications are essential. In pancreatitis due to gallstones, decisions about the need and timing of procedural intervention are critical.

Keywords: Gallstones; Pancreatitis; Type 2 Diabetes Mellitus.

INTRODUCTION

Acute pancreatitis is an inflammation of the pancreas whose causes vary due to infection and non-infection. This disease belongs to the class of common diseases that can begin with mild symptoms to have high mortality and morbidity. Acute pancreatitis is the leading cause of hospitalization for gastrointestinal disorders in the United States [1]. The most common etiologies of acute pancreatitis are hepatobiliary blockage and alcohol use. Gallstone-induced acute pancreatitis has considerable morbidity and mortality associated with its size, number and location [2].

Metabolic problems such as diabetes mellitus have a two-way relationship with the incidence of acute pancreatitis. Hyperglycemic conditions of diabetes mellitus can cause primary inflammation of the pancreatic parenchyma [3]. A history of diabetes mellitus also affects the mortality of acute pancreatitis associated with multiple organ damage [4]. Nonalcoholic fatty liver due to hyperlipidemia conditions showed significantly more severe features and worse clinical outcomes of acute pancreatitis than patients without fatty liver [5].

This case report discusses the incidence of severe acute pancreatitis with fatty liver, hepatobiliary blockage, and type 2 diabetes mellitus (DM) is still relatively rare. The condition of acute pancreatitis, with complicators, can undoubtedly increase the risk of mortality. Therefore, this case report aims to provide a picture of complaints of severe abdominal pain that can lead to acute pancreatitis after further examination. In this case, there are also comorbidities in the form of fatty liver, gallstones and type 2 DM, which causes the disease to develop severely. This case report referred the patient to a follow-up health facility for gastrointestinal surgery treatment.

CASE PRESENTATION

A 50-year-old man came to the emergency department complaining of severe abdominal pain in the upper abdomen three days before admission to the hospital, which felt increasingly severe. Patients deny complaints of fever, nausea, vomiting, and a history of trauma to the abdominal area. Complaints of yellow bodies are also denied. Patients have taken herbal medicine to reduce their complaints, but there has been no improvement. The history of the disease is unknown. Daily, patients often consume fatty and high-carbohydrate foods. History of alcohol consumption is denied.

The results of the initial physical examination when the patient entered obtained compos mentis awareness and vital signs: blood pressure at arrival 141/102 mmHg, pulse 98x/min, breathing rate 20x/min, temperature 36.4 C, oxygen saturation 98% with room air, and VAS pain score 8-9. The results of the abdominal examination found tenderness in the upper abdominal field, no muscular defence was found, and intestinal noise decreased.

Initial treatment in the emergency room, patients received ringer lactate infusion, antalgine, omeprazole and ondansetron injections while waiting for laboratory results, thorax x-rays, plain 3-position abdominal photos, and abdominal ultrasound examination. Patients still complained of pain with VAS 6-7, then got drip tramadol one ampoule in 500cc RL, ketorolac injection 30 mg.

The x-ray results of the abdomen in 3 positions obtained the impression of *preperitoneal fat* clouding, and the x-ray results of the thorax impressed bronchitis. At the same time, the results of 2-dimensional abdominal ultrasound found the presence of *grade 2 fatty liver* and free fluid on the *Morison pouch*. No abnormalities were found from the electrocardiography results, and the blood laboratory results obtained an increase in leukocytes, erythrocytes and hematocrit, respectively 13.06 103 / uL, 5.75 106 / uL, and 48.4%. There was also *left shifting* with lymphocytes at 8.1%, monocytes at 7.2% and neutrophils at 84.4%. Creatinine slightly increased by 1.12 mg/dL, hypocalcemia 0.95mmol/L and hyperglycemia 230mg/dL. Significant improvement in liver function examination is SGOT 417 U / L and SGPT 502 U / L.

During treatment in the room, patients continue to complain of pain with VAS 6-7 and complain of reduced urine and tea-coloured urine. CT-Scan of the abdomen was then performed, and the results of pancreatitis and multiple stones in the cystic duct (average size 0.574 cm) were obtained. The results of serum amylase and lipase examination were very high. Namely, 1897U / L and >3000 U / L. Total bilirubin levels of 1.94 mg / dL, boiled bilirubin 0.65 mg / dL and indirect 1.29 mg / dL all obtained an increase. Triglyceride levels increased by 193 mg/dL. The patient was diagnosed with acute pancreatitis and multiple cystic duct stones. Patients were then given rehydration therapy loading ringer lactate 1500cc, meropenem injection, lansoprazole injection 30mg / 12 hours, ondansetron injection 4 mg / 8 hours, hydrocortisone injection 100mg / 8 hours, paracetamol injection 1gr / 8 hours, fentanyl injection 150 mcg in 500 ccs asering drip, SNMC injection one ampoule / 12 hours, novorapid 3x8 IU, Levemir 0-0-6 IU, Sucralfat 3x15 mL, ursodeoxycholic acid 3x1, spasminal 3x1, Curcuma 3x1. The patient had experienced hypotension and complained of tightness. Once the patient's condition stabilizes, the patient is referred to a digestive surgery tertiary health facility for follow-up management on day 2 of treatment.

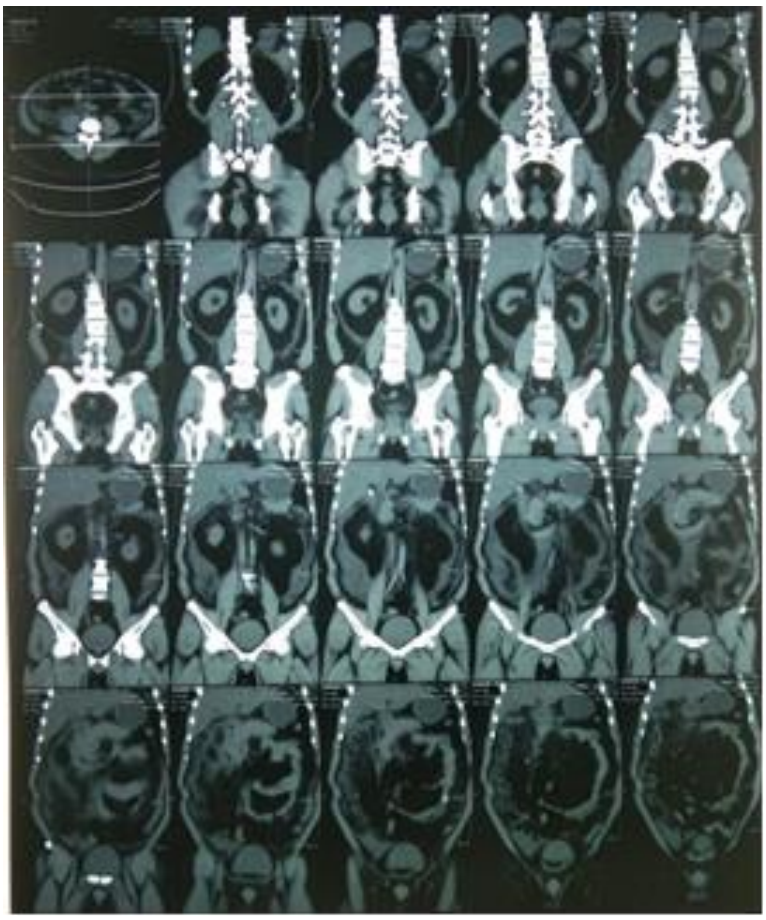

Figure 1. Abdominal CT Scan Results

DISCUSSION

Intense upper abdominal pain is one of the symptoms of acute pancreatitis that often appears in other diseases such as peptic ulcer, acute hepatitis, cholangitis, and cholecystitis· A good, directed history and physical examination can rule out other differential diagnoses [6]. According to the 2012 Atlanta criteria, the severity varied from mild degrees requiring conservative treatment to severe with high morbidity and mortality [1]. In this case, patients are included in severe degrees accompanied by

comorbidities in the form of type 2 DM, suspected to be unknown to the patient. Type 2 diabetes and *nonalcoholic fatty liver disease* (NAFLD) are commonly found together. These two things are considered manifestations of metabolic syndrome. NAFLD in type 2 DM patients has a prevalence of 70% [7]. According to Kvit et al., acute pancreatitis patients do not always progress to DM. In acute lesions of the pancreatic gland, islet cells are often found in good condition despite severe damage to pancreatic acinar cells [3].

The aetiology of pancreatitis that is often found is gallstones and alcohol, and is identified in 70% of cases [2]. Acute pancreatitis of any aetiology is diagnosed if it meets two of the following three criteria: 1. consistent upper abdominal or back pain, 2. increased levels of pancreatic enzymes (lipase and amylase) > 3 times the upper limit of normal, and 3. inflammation of the pancreas on *imaging examination* (*computed tomography* [CT] or *magnetic resonance imaging*) [8].

The pathophysiology of pancreatitis is a combination of local damage in the pancreas and a systemic inflammatory response involving a complex inflammatory cascade process. The most frequently put forward theory today states that pancreatitis is caused by injury or disruption of the pancreatic sinus system, resulting in leakage of pancreatic enzymes (trypsin, chymotrypsin, and elastase) into pancreatic tissue. The leaking enzyme becomes active in the tissues, which triggers the process of autodigestion and acute pancreatitis [9].

Activated proteases (trypsin and elastase) and lipase break down tissues and cell membranes, causing oedema, vascular damage, bleeding and necrosis. This inflammatory cascade is responsible for the systemic manifestations of acute pancreatitis. It can ultimately lead to increased capillary permeability and endothelial damage by microvascular thrombosis leading to multiple organ dysfunction syndrome (MODS), the leading cause of morbidity and mortality in acute pancreatitis [9].

In acute pancreatitis caused by gallstones, stones usually come out of the gallbladder through the cystic duct and then get stuck in the branching of the pancreaticobiliary duct or Vater's ampulla. Obstruction at the site causes reflux of bile into the pancreatic ducts. The risk of pancreatitis by gallstones increases when the gallstone size is smaller, allowing the stone to move more easily between the bile ducts across the ampulla [10]. Acute pancreatitis caused by gallstones has characteristics such as symptoms of biliary obstruction, including dark-coloured urine, pale stools, icteric sclera, excoriation from pruritus and jaundice. An increase in liver tests of alanine transaminases more than three times the upper limit of normal, followed by a more significant increase in aspartate aminotransferase, is a relatively specific indication of biliary aetiology [2]. DM is one disease that can increase the severity of acute pancreatitis. DM with hyperglycemia conditions, plus factors affecting insulin resistance (TNF-α, NFk B, amylin), leads to increased formation of reactive oxygen species (ROS) in pancreatic acinar cells. Elevated levels of amylin and CGRP (calcitonin gene-related peptide) in DM are thought to play a role in acute pancreatitis through excessive stimulation of pancreatic acinar cells. In addition, high CGRP and amylin levels can reduce pancreatic blood flow, causing further damage to the pancreas [11].

Initial evaluation of suspected acute pancreatitis involves laboratory abnormalities that lead to biliary cholestasis, hypercalcemia, or severe hyperlipidemia. Abdominal ultrasound is recommended to assess choledocolitiasis and bile duct dilatation in all patients. In the state where the diagnosis of pancreatitis is still in doubt, computed tomography (CT) with intravenous contrast can establish or rule out the diagnosis [8].

The basis of management for acute pancreatitis is aggressive early fluid resuscitation after the diagnosis is established. Ringer lactate solution is the recommended liquid with an initial dose of 15 to 20 mL/kg and a further 3 mL/kg per hour (usually around 250 to 500 mL per hour) during the first 24 hours if there are no contraindications [12]. Empirical antibiotics may be given pending culture results if there is suspicion of infection. Antibiotic regimens that can be given include carbapenems or combinations with

quinolones, ceftazidime, or cefepime with metronidazole [13]. Further management is adjusted to the aetiology of pancreatitis. In pancreatitis due to gallstones, early cholecystectomy is highly recommended. Early ERCP within 24 hours is recommended for cholangitis or biliary obstruction [14].

CONCLUSION

Anamnesis and physical examination, and sound and directed supporting examinations can establish the diagnosis of acute pancreatitis in cases of acute pain in the abdominal region. Examination of amylase and lipase enzymes, abdominal ultrasound, and abdominal CT scan are recommended to establish or rule out the diagnosis of acute pancreatitis. Establishing a proper early diagnosis will help good management, including resuscitation and procedural interventions in cases of acute pancreatitis.

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CONFLICT OF INTEREST

None

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