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Case Report

Acute Pancreatitis in Type 2 Diabetes Mellitus

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Abstract

Introduction: Severe 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. Acute gallstone-induced Pancreatitis has considerable morbidity and mortality. A good anamnesis and physical examination can help establish a more precise diagnosis and management.

Case: A 50-year-old male came to the emergency room with a significant complaint of severe abdominal pain in the right upper abdomen for three days that was getting worse. VAS score 8-9. The history of the disease is previously unknown. Blood pressure when coming 141/102 mmHg, pulse 98 x/ min, temperature 36.4ºC. Abdominal CT scan results obtained the results of images of Pancreatitis and multiple stones in the cystic duct. Serum amylase and lipase results were very high at 1897 U/L and > 3000 U/L. Patients were then referred to the digestive surgery department on the 2nd day of treatment for further treatment.

Discussion: Acute pancreatitis caused by gallstones is usually caused because stones get stuck in the branching of the pancreatic duct. Obstruction at the site causes bile reflux into the pancreatic duct. Small gallstones increase the risk of Pancreatitis because they move quickly between the bile ducts. In this patient, it is suspected to have had a previously unknown history of DM, which is one of the causes of worsening the condition.

Conclusion: Early diagnosis enforcement is essential in acute pancreatitis cases because it will affect patients' initial management of emergencies. Initial management includes fluid resuscitation and detection of local and systemic complications, and decision-making for procedural intervention measures.

Keywords: Acute Pancreatitis; Cholecystitis; Diabetes Mellitus Type 2; Cystic Duct; Gallstones

Abbreviations

DM: Diabetes Mellitus; CT: Computed Tomography

Introduction

Acute Pancreatitis is an inflammation of the pancreas whose causes are diverse due to infection and non-infection. This disease belongs to the group of common conditions that can start with mild symptoms and have high mortality and morbidity. Acute Pancreatitis is the leading cause of hospitalization in gastrointestinal disorders in the United States [1]. The most common etiology of acute Pancreatitis is hepatobiliary obstruction and alcohol use. Acute Pancreatitis induced gallstones have considerable morbidity and mortality, associated with their size, number and location [2].

Metabolic problems such as diabetes mellitus have a two-way relationship with acute pancreatitis incidence. The hyperglycemic condition of diabetes mellitus can cause primary inflammation of the pancreatic parenchyma [3]. A history of diabetes mellitus is also known to affect the mortality of acute pancreatitis associated with the presence of multiple organ damage [4]. Nonalcoholic fatty liver due to hyperlipidemia conditions significantly showed a more severe picture and worse clinical results of acute Pancreatitis than patients without fatty liver [5].

This case report discusses the incidence of severe acute Pancreatitis with fatty liver, hepatobiliary obstruction, and type 2 diabetes mellitus (DM) is still relatively rare. Acute pancreatitis conditions with complications can undoubtedly increase the risk of mortality. Therefore, this case report aims to provide an overview 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 to severe degrees. The patient was referred to a follow-up health facility for digestive surgery treatment in this case report.

Case Reports

A 50-year-old man came to the emergency installation with a significant complaint of severe abdominal pain in the upper abdomen from 3 days before entering the hospital, which was getting worse. The patient denies complaints of fever, nausea, vomiting, and a history of trauma to the abdominal area. Yellow body complaints are also refuted. The patient has taken herbal medicine to reduce his complaints, but there has been no improvement. The history of the disease is previously unknown. Patients daily often consume fatty and high-carbohydrate foods. The history of alcohol consumption is refuted.

The results of the initial physical examination when the patient entered obtained awareness of compos mentis, vital signs: blood pressure when coming 141/102 mmHg, pulse 98 x/min, breath rate 20 x/ 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 er, the patient received an infusion of lactate ringer, antalgin injection, omeprazole and ondansetron while waiting for laboratory results, thorax X-ray, a plain photo of the abdomen in three positions, and abdominal ultrasound examination. Patients still complain of pain with VAS 6-7, then get a drip of tramadol ketorolac injection of 30 mg.

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

During the treatment in the room, the patient continues to complain of pain with VAS 6-7 and complains of reduced urine and tea-like colour. Abdominal CT scans were then performed, and the results of the description of Pancreatitis and multiple stones were obtained in the cystic ducts (average size 0.574 cm). The serum amylase and lipase examination results were very high, namely 1897U/L and > 3000 U/L. Total bilirubin levels of 1.94 mg/dL, direct bilirubin 0.65 mg/dL and indirect 1.29 mg/dL were all obtained an increase. Triglyceride levels increased by 193 mg/dL. The patient was diagnosed with acute Pancreatitis and multiple stones in the cystic duct. The patient was then given rehydration therapy loading ringer lactate 1500cc, meropenem injection, lansoprazole injection 30mg/ 12 hours, ondansetron injection 4 mg/8 hours, hydrocortisone injection 100mg/8hours, paracetamol injection 1gr/8 hours, fentanyl injection 150 mcg, SNMC injection one ampoule/12 hours, rapid insulin 3 x 8 IU, detemir 0-0-6 IU, Sucralfat 3 x 15 mL, ursodeoxycholic acid 3 x 1, spasminal 3 x 1, Curcuma 3 x 1. 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 treatment on the 2nd day.

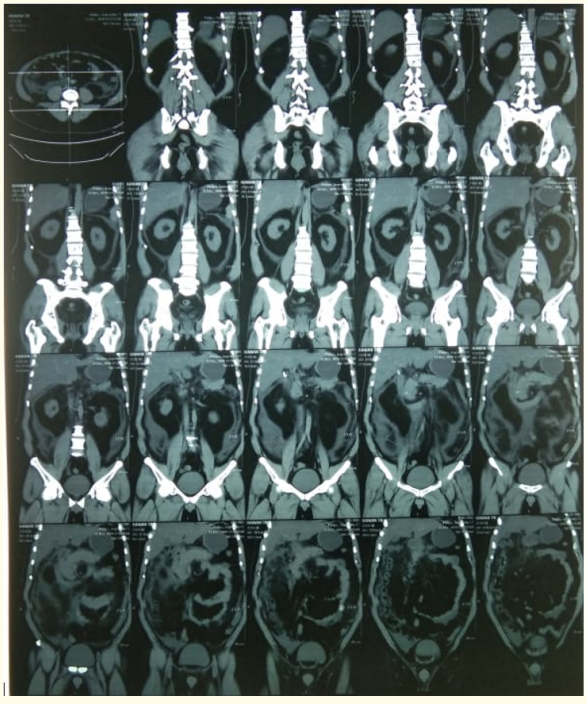

Figure 1: Abdominal CT Scan results of the description of Pancreatitis and multiple stones were obtained in the cystic ducts (average size 0.574 cm).

Discussion

Severe 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. An excellent and directed anamnesis and physical examination can rule out a differential diagnosis that is lain [6]. According to the Atlanta 2012 criterion, the degree of severity varies, from mild degrees requiring conservative treatment to severe degrees with high morbidity and mortality [1]. The patient, in this case, belongs to an extreme degree accompanied by comorbidities in the form of type 2 DM, which is suspected to be unknown to the patient in advance. Dm type 2 and non-alcoholic 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 develop into DM. In acute lesions of the pancreatic gland, islet cells are often found in good condition despite severe damage to pancreatic three acinar cells.

The frequently found etiology of Pancreatitis is gallstones and alcohol and is identified in 70% of cases2. Acute Pancreatitis of any etiology 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 of the normal upper limit, 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 an injury or disruption to the acinous system of the pancreas, resulting in the leakage of pancreatic enzymes (trypsin, chymotrypsin, and elastase) into the tissues of the pancreas. 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, damage to blood vessels, bleeding and necrosis. This inflammatory cascade is responsible for the systemic manifestations of acute Pancreatitis. It can ultimately increase capillary permeability and endothelial damage with microvascular thrombosis that causes multiple organ dysfunction syndromes (MODS), the main 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 pancreatic duct and vater ampules. Obstruction at the site causes bile reflux into the pancreatic duct. The risk of Pancreatitis by gallstones increases when the gallstones are smaller in size, as they allow the stones to move more easily between the bile ducts across the ampullar [10].

Acute Pancreatitis caused by gallstones has symptoms of biliary obstruction, including dark-coloured urine, pale stools, jaundicular sclera, excoriation of pruritus and jaundice. An increase in the transaminase alanine liver test more than three times the upper limit of normal, followed by a greater reduction in aminotransferase aspartate, is a relatively specific indication of biliary etiology [2].

DM is one of the diseases that can increase the severity of acute Pancreatitis. DM with hyperglycemia conditions, plus factors affecting insulin resistance (TNF-α, NFk B, amylin), leads to an increase in reactive oxygen species (ROS) formation 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 the over-stimulation of pancreatic acinar cells. In addition, high levels of CGRP and amylin can reduce pancreatic blood flow, causing further damage to the pancreas [11].

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

The basis of acute pancreatitis management is aggressive early fluid resuscitation after the diagnosis has been established. Ringer lactate solution is a 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 no12 contraindications. Empirical antibiotics can be given while waiting for the results of the culture if there is a suspicion of infection. Antibiotic regimens that can be given include carbapenem or a combination with quinolones, seftazidim, or cephrayme with metronidazole[13]. Further management is adapted to the etiology of Pancreatitis. In Pancreatitis due to gallstones, early cholecystectomy is highly recommended. Early ERCP within 24 hours is recommended in cases of cholangitis as well as biliary obstruction [14].

Conclusion

Anamnesis and physical examination and a good and directed supporting test can establish a diagnosis of acute Pancreatitis in cases of acute pain in the abdominal region. Analysis of the enzyme amylase and lipase, abdominal ultrasound, and an abdominal CT scan is recommended to establish or rule out the diagnosis of acute Pancreatitis. Proper enforcement of early diagnosis will help with good management, including resuscitation and procedural intervention in acute pancreatitis cases.

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Conflict of Interest

Declare if any financial interest or any conflict of interest exists.

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