CASE REPORT

ACUTE PANCREATITIS AS A COMPLICATION OF DENGUE FEVER

Sayid Ridho, Syarif Hidayat, Marcel Simadibrata Kolopaking
Department of Internal Medicine, Medical Faculty, University of Indonesia/
Cipto Mangunkusumo National Centre Hospital, Jakarta

ABSTRACT

Acute pancreatitis is a rare complication of dengue fever that usually occurs without hyperglycemia. We report one patient of dengue fever with a complication of acute pancreatitis. A 28 year old male was referred from a private clinic to Dr. Cipto Mangunkusumo hospital, because of fever and abdominal pain since two days before admission. The physical examination showed slight fever and epigastric pain. The laboratory examination revealed leukocytosis, thrombocytopenia and increased levels of serum amylase and lipase. The diagnosis of dengue infection was made after the finding the IgM for dengue virus. After 12 days of hospitalization, the patient's clinical signs and symptoms and laboratory findings were within normal limits.

Key words: acute pancreatitis, dengue fever

INTRODUCTION

Acute pancreatitis is an acute inflammation reaction of the pancreas. The history of this disease varies greatly, from mild to severe accompanied by shock, renal and pulmonary disorders that may be fatal. Although the disease was first reported in the year 1700, the first systematic report was not published until 1889. It was a report by Reginal Fits. The incidence of pancreatitis varies greatly from one country to another and also from one area to another in the same country.

The frequency was reported to be around 0.14-15 or 10-15 patient for every 100 thousand people. There has not been many reports of the disease in Indonesia. The etiology of the disease also varies greatly, according to a research in Cipto Mangunkusumo hospital revealed that the most common causes of acute pancreatitis were leptospirosis (21.5%), gallstone (12.2%), typhoid fever (9.2%), dengue hemorrhagic fever (6.1%), and unknown causes (45.2%). Nurman reported the percentage of dengue hemorrhagic fever that caused acute pancreatitis is about 6.3%, however, Suyatmi and Sumanto reported it to be 10%. Nelwan reported 2 cases of dengue hemorrhagic fever that caused acute pancreatitis.

The finding of positive IgM is a sign that a patient is infected by the dengue virus. In Western countries, 90% of the cause were alcohol consumption (80-90% in men), gallstone(75% in women), and idiopathic, the rest were caused by trauma, virus, drugs, etc.

The pathophysiology of acute pancreatitis can be seen in scheme 1.

The diagnosis of acute pancreatitis is usually made if the patient feels sudden upper abdominal pain. The laboratory findings show that the serum amylase or lipase increased at least three times the normal value. The ultrasonography findings reveal acute pancreatitis, or operation/autopsy findings that confirm acute pancreatitis. Other complaints could be nausea and vomiting, gassiness, fever, and signs of local peritonitis.

The treatment of acute pancreatitis is aimed to stop the inflammation process and autodigestion, or at least to stabilize the clinical condition to give a chance for resolution. Basically, medical therapy consists of liquid and parenteral nutrition infusion, systemic complication therapy, symptomatic therapy particularly to overcome the pain. Sometimes pethidine is needed to overcome the pain. A nasogastric tube is placed for the pancreas and to overcome the paralytic ileus. Antibiotics for systemic complications can be given immediately, the right choice would be the new generation of cephalosporine.

If there is no improvement in the second week, the possibility of widespread necrosis must be kept in mind. If this happens, surgery must be considered. Some of the surgical operations that can be performed are pancreas...
loge exploration and drainage and also necrotic tissue debridement, gall duct decompression in the accumulating obstruction icterus, or pseudocyst and abscess drainage.6

CASE REPORT

Mr. K, 28 years old, was admitted to Cipto Mangunkusumo hospital with septic shock, acute pancreatitis, and acute renal failure as the last problems in the emergency unit. The patient’s major complaint was abdominal pain since 2 days before admission. The abdominal pain was felt like a sharp pain and a hot, irri-

tated feeling mostly in the epigastrium combined with nausea and 5 times vomiting. The patient also complained of having a feeling of fullness in the stomach that made him hard to breathe. He also complained of sudden fever 3 days before, mostly at night, accompanied by headache without preceding cough or sneezing. There was no complaint about urinating nor defecation. There was no history of spontaneous bleeding. The patient also had no history of jaundice, pancreas injury due to many causes

granulocyte leukocyte chemoattraction monocyte/macrophage

leukotriene

circulation effect

TNF, IL-1, IL-6, IL-8 platelet activation factor

liver: acute phase response endothel and neutrophil activation

blood proteolytic cascade activation

cell damage

multi organ failure lung, heart, kidney, shock

Scheme 1. The pathophysiology of acute pancreatitis

To determine the prognosis, the Ranson criteria is commonly used:

At first

Age > 55 years
Leukocyte > 16,000/mm³
Glucose > 200 mg/dl
LDH > 350 IU/l
AST > 250 U/l

In 48 hours

decreased hematocrit > 10%
decreased BUN > 5 mg/dl
Calcium < 8 mg/dl
pO₂ < 60 mmHg
Base deficit > 4 mEq/l

Score < 3 zero mortality
3-5 mortality 10 – 20 %
> 6 mortality > 50%, commonly connected with pancreatitis necroticans

Describe the systemic effect to target organs, such as lung and kidney

Describe the inflammation process
alcohol consumption, out of town travelling, or working in gutters. No other members of his family had the same illness as the patient.

The physical examination showed that the patient looked seriously ill. He was alert with blood pressure of 90/60 mmHg, pulse rate 110 times/minute, respiration rate 18 times/minute, body temperature 37.8 °C. The head examination showed a hyperemic conjunctiva, the sclera was not icteric. There was a nasogastric tube showing greenish fluid and an oxygen tube on the patient’s nose. The tongue was dry, dirty, not hyperemic. There was no abnormality of the tonsils and pharynx. The neck examination showed no increase in the jugular vein pressure, no lymph nor thyroid gland enlargement.

The chest examination showed no abnormality.

The abdomen was distended, the patient felt pain when the epigastrium was pressed. The liver and the spleen were not palpable. There were normal bowel sounds, with no muscular defense nor ascites.

In the extremities, the acrals were warm, there was no cyanosis, and the tourniquet test was positive. There was no gastrocnemius pain on palpation. There was sufficient turgor and no leg edema.

Laboratory examination showed a hemoglobin level of 12.5 g/dl, hematocrit 37%, leukocyte 14,100/ml, thrombocyte 40000/ml, erythrocyte 4.1 million/ml, ureum 98 mg/dl, creatinine 3.5 mg, blood glucose 119 mg/dl, amylase 1453 U/l, lipase 1606 U/l. Blood glucose analysis showed pH 7.415, pCO2 27.5 mmHg, pO2 162.7 mmHg, HCO3 17.6 mmol/l, BE 5.6 mmol/l, oxygen saturation 99.2%. Blood sodium 138 mEq/l, and blood potassium 3.8 mEq/l.

Abdominal ultrasonography examination showed no abnormality.

The patient was treated with problems of acute pancreatitis, acute renal failure, and improving septic shock. The patient was put on a fast and given ranitidine and ceftiraxone intravenously. He was given intravenous fluid of lactic ringer in line I, and parenteral nutrition Ivelip. The patient was treated as a dengue hemorrhagic fever patient. On the fourth day of treatment the patient’s condition ameliorated, epigastric pain on palpation had disappeared, there was adequate fluid and electrolyte balance, and the fever had subsided. The patient was gradually given liquid food from the nasogastric tube combined with parenteral feeding. On the following days, thrombocyte level rose to normal value. On the sixth day the nasogastric tube was removed, and the patient was given food more frequently. On the tenth day, the patient’s condition was back to normal, there was no fever, no abdominal complaint, normal thrombocyte, ureum, and creatinine levels. The antibiotics were no longer administered. Two days later, the patient was returned home in a good condition, and was suggested to go to the Gastroenterology and Tropical Infection subdepartment for further examination.

DISCUSSION

The working diagnosis of acute pancreatitis was based on complaints of sharp abdominal pain, feeling of hotness in the epigastrium accompanied with nausea and vomiting, epigastric pain on palpation, and high levels of amylase and lipase. It was thought that it was due to Dengue Hemorrhagic Fever virus infection because there was sudden fever 3 days before admission, thrombocytopenia, positive tourniquet test.

The working diagnosis of acute renal failure was based on ureum 98 mg/dl, creatinine 3.5 mg/dl, and analysis of blood gases that indicated compensated metabolic acidosis. The cause was thought to be pre-renal, as a result of hypoperfusion due to shock in acute pancreatitis involving the kidney.

The working diagnosis of improving septic shock was based on blood pressure of 90/60 mmHg, warm acrals, and still high body temperature. These were thought to be a complication of acute pancreatitis, because of the finding of leukocyte 14,100/ml. In the emergency unit the patient had already suffered from multi organ failure because he had low blood pressure, high ureum and creatinine levels, and blood gas analysis showed metabolic acidosis. Nevertheless, there was no pulmonary involvement because of high pO2. Referring to the Ranson criteria, if the LDH and AST were high, then the highest score would be 2. This meant a good prognosis with zero mortality. It was proved by the patient’s recovery after 12 days of treatment.

The cause was concluded to be dengue virus infection because of the positive IgM for dengue. This was in accordance with some of the references that mentioned that virus infection could cause acute pancreatitis. \(^4,5,11\)
The patient was treated with conservative therapy. Besides, he was given antibiotics for the sepsis, with a good result in accordance with reference.5

REFERENCES