

DHF WITH COMPLICATION OF ACUTE PANCREATITIS RELATED HYPERGLYCEMIA : A CASE REPORT

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Abstract. DHF is endemic in Indonesia, with incidence of 9.72/100,000 population and CFR of 2.5 %. Acute pancreatitis is a rare complication in DHF, usually without hyperglycemia. We report here 1 patient of DHF grade II with complication of acute pancreatitis, and hyperglycemia which occurred as a result of pancreatitis. A 24 years old female was referred from Santa Jusuf Hospital, with 5 days of fever and hematemesis. On physical examination we found slight fever and hematoma on her left leg. Laboratory examination revealed Hb 13.4 g%, WBC 8,500/mm³, Ht 42%, platelets 22,500 /mm³, amylase 317 U/l, lipase 1,198 U/l and blood glucose 397 mg%. CT scan result of pancreas was consistent with acute pancreatitis. Diagnosis of dengue infection was made after the finding of positive IgM and IgG for dengue virus. After 18 days clinical symptoms and signs and laboratory results returned to normal.

INTRODUCTION

Dengue infection is still one of the major public health problems in Indonesia. The incidence of the disease is still increasing (10.2/100,000 population), but the mortality decreased from 41.3 % in 1968 to 2.7% in 1996 (Suroso, 1997). In Bandung where this case presented, an outbreak occurred in 1986. The problems are vector control, education of the population, early diagnosis and appropriate treatment. WHO clinical criteria with Indonesian modification was used to establish diagnosis of DHF, and confirmed by serologic test.

Encephalopathy, renal impairment, liver injury, ARDS, DSS as complications of DHF have been reported. Acute pancreatitis is a rare complication of DHF and hyperglycemia as a consequence of acute pancreatitis in DHF is unusual. We report here one patient of DHF in adult with acute hepatitis and pancreatitis, and hyperglycemia occurred as a result from acute pancreatitis.

CASE REPORT

A female aged 25 was referred to the hospital on January 23, 1997; with 5 days history of fever. She also complained of hematemesis, epistaxis, myalgia and epigastric pain. On physical examination the patient was unconscious, blood pressure 130/80

mmHg, pulse rate 84/minute, respiration rate 20/minute, temperature 37.2°C, icteric, heart and lung normal, liver just palpable, spleen not palpable and a hematoma was found on the left leg. Laboratory examination revealed Hb 13.4 g%, WBC 8,500/mm³, platelets 22,500/mm³, Ht 42%, bleeding time 2', clotting time 8', PT 12" (control normal 11"), APPT 28" (control normal 27"), fibrinogen 230 g% D-Dimer negative, prickle cell positive, creatinine 2.1 mg%, urea N 60 mg% GOT 41.2 U/l, GPT 240.8 U/l, GGT 970 U/l, bilirubin total 6.5 mg% (direct 5.9 mg%), alkaline phosphatase 309 U/l, amylase 317 U/l, lipase 1,198 U/l, blood glucose 397 mg% and positive reduction of urine.

Chest X rays and ECG were normal; plain X rays of abdomen showed no calcification of pancreas, air overdistribution inside the gut and colon cut off appearance that is consistent with acute pancreatitis. The problem was the cause of hyperglycemia.

Diagnosis of viral hemorrhagic fever with the first possibility of DHF grade II was suspected, accompanied by complication of acute hepatitis and pancreatitis.

Treatment was given as follows: Ringer's lactate infusion, insulin 4 U tid, gastric aspiration through nasogastric tube, metoclopramide 1 amp/day. On day 2, the patient became conscious, diuresis 1,500 cc/17 hours, CVP was kept between 10 and 12 cm H₂O. But Hb decreased to 8.3 mg%, Hct to 29%, platelets 102,000 /mm³. Transfusion

was administered to replace blood loss.

Ultrasonography examination of pancreas, liver and gall bladder did not show any abnormalities. CT scan with contrast of abdomen revealed hepatosplenomegaly, gall bladder wall thickening and pancreas feature that is consistent with acute pancreatitis. Serologic test results : IgG, IgM for dengue were positive; HbsAg, IgM-Anti HBc, Anti-HCV were negative. Endocrinologist was consulted for hyperglycemia. Acute pancreatitis was likely to be the cause of hyperglycemia and diabetes mellitus was excluded by normal finding of HbA1c (7.4%).

From day 3 to day 7 : The patients was still conscious, hemodynamically stable, diuresis was between 2,300 ml and 3,000 ml/day. Hb increased to 10.3 g%, hematocrit 83%, platelets 105,000 - 114,000/mm³, creatinine 0.8 g%, urea N 18.2 mg%, blood glucose decreased to 139 mg%, amylase 324 U/l, lipase 2,094 U/l. Enteral nutrition started on day 3.

From day 8 to day 13 : Platelets became normal (172,000/mm³), bilirubin became normal (0.92 mg%), GOT 57.9 U/l, GPT 103 U/l, GGT 151 U/l, alkaline phosphatase 174 U/l, amylase 204 U/l, lipase 622 U/l. Insulin was discontinued on day 11. On day 13 the patient was discharged without any medication, and on day 18 there were no abnormalities (clinically and laboratory).

DISCUSSION

Hyperglycemia may be caused by pancreatitis or pre-existing diabetes mellitus. Diabetes mellitus was excluded by the finding of normal HbA1c since high HbA1c level indicates hyperglycemia during the last 3 months. This was also supported by the absence of history of polydipsia, polyphagia and polyuria and blood glucose was still normal after insulin was discontinued. Chronic pancreatitis was excluded by the absence of pancreatic calcification, history of steatorrhea, alcoholism by the CT scan finding. Destruction of exocrine and endocrine tissue of the pancreas may lead to increase of transaminase and blood glucose. So the acute pancreatitis in this patient was likely to be responsible for hyperglycemia.

Fever less than 7 days with bleeding abnormality and multi-organ involvement including acute

pancreatitis may be caused by infection. Viruses that could be responsible for this include dengue virus, hanta virus, herpes virus, hepatitis virus. Dengue hemorrhagic fever was established in this patient on the basis of WHO clinical criteria (Indonesian modification: platelets below 150,000/mm³) and confirmed by the finding of positive IgG, IgM for dengue virus. DHF grade II was confirmed by the absence of circulatory failure and the presence of spontaneous bleeding. Hepatitis A, B and C were excluded by serologic tests. Serologic tests for other viruses were not done, because of positive results of the serologic test for dengue virus. IgG, IgM for dengue virus were tested on day 6 of illness. Positive results may suggest secondary infection of dengue, since IgG may be positive after 2 weeks illness following the primary infection. Isolation of dengue virus and serotype tests were not done.

DHF is distinguished from classic DF by the presence of hemoconcentration thrombocytopenia and abnormal hemostasis (Nimmannitya, 1996). In this patient hemoconcentration was absent because the patient had been given adequate IV fluid administration from Santa Jusuf Hospital.

Immune mechanisms are responsible for capillary leakage and organ involvement in DHF (Simpson, 1996). Complications of DHF may include shock (DSS) encephalopathy, respiratory distress (Monath, 1995). According to Kitayaporn (1994) 12.5% of DHF patients have jaundice from hepatic failure. Pancreatitis is a rare complication of DHF. A previous study in Hasan Sadikin Hospital from 1995-1996 showed that of 55 patients of DHF 2 patients were DSS, and no patient had pancreatic complications (Jusuf, 1996).

Treatment of DHF is supportive and symptomatic. The fluid used in DHF should be an isotonic solution. Fluid replacement is important to avoid hypovolemia caused by plasma leakage, the rate of infusion must be adjusted according to the rate of plasma leakage (Nimmannitya, 1996). We used here Ringer's lactate. Hemoglobin and hematocrit must be monitored to avoid hypovolemia or overloading. We also administer CVP in severe illness. Reduction of Hb and Hct without high CVP indicated blood loss, so transfusion was given in this patient.

In conclusion, DHF may have acute pancreatitis as a complication and unusual hyperglycemia may be the result of this acute pancreatitis.

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