Acute Pancreatitis in a Young Patient with Hepatoma and Recurrent Hematemesis-Melena

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INTRODUCTION

It is estimated that worldwide 300 million individuals are chronically infected by the Hepatitis B Virus (HBV), a cause for a great number of mortality due to chronic liver disease. It is also estimated that at least 30% of chronic hepatitis B patients will suffer from liver cirrhosis. Young patients, who are infected by HBV during infancy or childhood, have a higher incidence rate of cirrhosis compared to those who were infected as adults.[1]

Additionally, chronic HBV infection has also been acknowledged as the most important causative factor for hepatocellular carcinoma (HCC), especially in areas with an epidemiologically high incidence rate of HBV infection. Beasley et al found that adult males with chronic HBV infection have a relative risk for HCC 200 times that of uninfected individuals.[1]

Portal hypertension is one of the complications of advanced liver cirrhosis with or without liver carcinoma. What follows is varices in the esophagus or other sites, as well as ascites. Patients often die due to bleeding from uncontrollable or recurrent esophageal varices rupture or due to spontaneous bacterial peritonitis (SBP), sepsis, coma, and renal failure.[2]

If an individual is diagnosed with esophageal varices, the possibility of bleeding within 2 years is approximately 30%, while the mortality risk for each incidence of variceal bleeding is 20-30%. Large varices, varices in patients with severe liver dysfunction, and unremitting alcoholism increase the risk of bleeding. Poor nutritional status, very high levels of bilirubin or alkaline phosphatase, very low serum albumin and blood coagulation factors II, VII, and X, could cause severe or recurrent bleeding, even death. Varices at the gastric fundus and congestive gastropathy caused an even higher risk than esophageal varices, and are more difficult to manage.[2]

Chronic HBV infection has several extra-hepatic manifestations, such as vasculitic rash, sero-negative arthritis or arthralgia, glomerulonephritis, polyarteritis nodosa, and the nephrotic syndrome. The pathogenesis of these manifestations are yet unclear, but it is suspected that they are caused by deposition of immune complexes on the arterioles of the affected organs.[1]

Acute pancreatitis is a gastrointestinal emergency that is often clinically encountered with varying symptoms. Thirty percent of all cases of acute pancreatitis have no known cause, while the rest are due to viral infection (such as parotitis or dengue), or bacterial infection, billiary tract stone, alcoholism or the use of certain drugs (steroids, tiazid diuretics), immunological reactions (such as that occuring in LE), protein deficiency, circulation disturbances, metabolic acidosis, and obstruction of the pancreatic duct.[3] The association between HBC infection and pancreatitis is rarely mentioned in literature. A study conducted at Cipto Mangunkusumo Hospital in 1996 found the cause of acute pancreatitis to be leptospirosis (21.5%), gall stones (12.2%), typhoid fever (9.2%), dengue hemorrhagic fever (6.1%), and idiopathic in 45.2%. Iatrogenic acute pancreatitis may result after ERCP procedure.[3]

The diagnosis of acute pancreatitis is often difficult and is established based on clinical findings confirmed by relatively unspecific laboratory and radiology findings. Complaints of sustained epigastric discomfort (for

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more than 5 hours), accompanied by nausea or vomiting, hypogastric discomfort, periumbilical discomfort, temperature of over 37.5 degree Celsius, pulse rate of over 90 times per minute, and leukocytosis of over 10,000 support the diagnosis of pancreatitis especially if accompanied by abdominal distention, tenderness, and increased blood amilase and lipase levels at least three times the normal levels.[5] Important examinations to confirm the diagnosis as well as to determine the severity of pancreatitis consist of abdominal ultrasound examination and CT-scan.

The management of acute pancreatitis is mostly supportive while preventing and managing complications that might occur. Antibiotics are given as prophylaxis or if the cause is suspected to be bacterial infection. Over 80% of patients with acute pancreatitis recover rapidly without any complication.[5]

CASE REPORT

A male patient, 28 years of age, was brought to the hospital due to vomiting approximately 1500 cc of dark brown blood. According to the history, for the past year the patient had been suffering from frequent nausea and bloating, he was easily fatigued, weak, and had a poor appetite. The patient had often visited the general practitioner, to no avail. Since he felt unhealthy and lacked energy, regularly took traditional concoctions (*jamu*) from street vendors. One of the ingredients of the concoction he took was wine. The patient recalled having had "yellow fever" (hepatitis) for a significant period of time when he was 8 years old, even though he was never hospitalized. Other than that and the problem he was suffering at that moment, he had never suffered from any serious illness. The patient rarely took any medication (except for traditional concoctions) without a physician's recommendation. Four months before the patient had been hospitalized in another hospital with a diagnosis of chronic hepatitis B. During hospitalization, ultrasound findings were appropriate for liver cirrhosis with a tendency towards hepatoma. CT-scan examination on the 19th of August 1999 demonstrated findings of hepatoma with metastatic lesions, splenomegaly, enlargement of para-aortal nodes, and ascites. His pancreas, kidneys, and gall bladder were normal.

On physical examination, the patient was moderately ill, mentally alert, pale, demonstrated no signs of jaundice, and had a nasogastric tube (NGT) inserted, with brownish blood flowing out at a steady rate. His blood pressure was 100/70 mmHg, pulse rate 100 times per minute, temperature 37 degrees Celcius, respiration rate 32 times per minute, and jugular venous pressure 5-2 cm H_2O. Lung and heart examination revealed no abnormalities. His abdomen was distended. There was no muscular defence. There was minimal ascites. His liver was palpable 2 fingers below the costal arc, and had an irregular surface, rounded edge, supple to solid consistency, and tenderness. His spleen was palpable at S1, with a rubbery consistency. His bowel sounds were normal. His extremities were warm, with minimal edema, palmar erythema and clapping tremor unclear. There was no spider nevi.

Laboratory results on admission were as follows: hemoglobin level 9.8 g%, leukocyte count 9,800/mm^3, platelet count 136,000/mm^3, erythrocyte count 3.7 million/mm^3, and hematocrit 30%. The results of laboratory specimens taken in the ward were as follows: hemoglobin level 6.9 g%, leukocyte count 5,500/mm^3, platelet count 112,000/mm^3, albumin 3.1%, globulin 2.3%, alkaline phosphatase 609, cholinesterase 193, LDH 368, SGOT 294, SGPT 52, AFP 76.8, total bilirubin 1.1 mg%, HbsAg positive, anti-HCV negative, ureum 25 mg%, creatinine 0.8 mg%, bleeding time 4'30", clotting time 11', APTT 26,80', fibrinogen 262. Esophago-gastroduodenoscopy found grade III-IV esophageal varices with signs of previous bleeding, severe portal hypertension gastropathy, and moderate portal hypertension duodenopathy.

During hospitalization the patient suffered from recurrent hematemesis-melena with compensated DIC. The patient received transfusion, total parenteral nutrition, unalog somatostatin, propranolol, ranitidine, Duphalac®, neomycin, aldactone, vitamin K, gastric lavage with ice water 2 to 3 times daily, and enemas in the mornings and afternoons.

On the tenth day, the patient had a sudden attack of severe, continuous, stomach ache, nausea, gassiness, abdominal distention with tenderness of almost the whole abdomen and lower bowel sounds, without muscular defense nor palpation-release pain, and was suddenly restless/apathetic. His blood pressure was 90/70 mmHg, his pulse rate was 120 times per minute, his temperature was 37.5 degrees Celsius. Laboratory findings were as follows: hemoglobin level 9 g%, leukocyte count 31,600/mm^3, platelet count 229,000/mm^3, amilase level 433 (normally less than 120), lipase level 504 (normal <190).

The patient also had acute pancreatitis. The following actions were taken: NGT retained, total parenteral nutrition administered, with the addition of 2 x 1 g of ceftriaxone.

After several days of hospitalization, the patient's

condition improved, his abdominal discomfort, abdominal distention and tenderness were reduced. Serum amilase and lipase levels dropped on the 15th day, to 181 and 205 respectively, and on the 21st day, to 205 and 142 respectively.

Even though the patient had suffered from acute pancreatitis and had recovered, his esophageal varices caused recurrent bleeding, and the patient underwent ligation therapy on the 22nd of September. His bleeding was under control.

DISCUSSION

The patient was a young male who was infected by Hepatitis B during childhood. His condition developed into liver cirrhosis. The patient complained of frequent nausea, gassiness, loss of appetite, and fatigue for the past year prior to hospital admission. During the final months, his cirrhosis transformed into hepatoma with severe portal hypertension, accompanied by gastropathy and duodenopathy with esophageal varices.

As a consequence of increased hepatic resistance, the patient's portal tension was increased further, causing the outgrowth of collateral blood vessels that connected the portal system and the azygos system or systemic circulation localized in the lower esophagus, cardia, upper minor gastric curvature, fundus, umbillicus, rectum, and on the abdominal wall, which could then result in varices.

The haemodynamic system of patients with bleeding varices must first be stabilized, and then undergo sclerotherapy or endoscopic ligation. Bleeding could then be apprehended and recurrent bleeding prevented. Somatostatin or octreotide were proven to be effective in the management of bleeding, and are thus alternative treatments if sclerotherapy or ligation could not be conducted immediately.

In this particular patient, recurrent bleeding was unremitting, possibly due to thrombocytopenia (and low levels of coagulation factors due to severe liver dysfunction such as due to cirrhosis or carcinoma). Furthermore, gastro and duodenopathy such as that demonstrated in endoscopic examination, history of regular alcoholism that was even continued after the development of symptoms, which make it more difficult to stop bleeding.

The diagnosis of pancreatitis in this case was based on clinical symptoms that support and increase amilase and lipase levels as well as leukocytosis. Unspecific symptoms and increased serum amilase levels could be encountered under conditions such as gastric ulcer perforation, paralytic illeus, acute cholecystitis, parotitis, renal failure and liver cirrhosis itself.[6] The levels were not highly increased. In this particular case the clinical diagnosis of mild acute pancreatitis could still be established. The cause of acute pancreatitis in this patient was unclear. Possible factors that predispose include alcoholism, circulation disturbance due to bleeding of the varices, hemostatic disturbance and immune complex reaction. Hepatitis B virus infection had no direct association, bearing in mind that it is hepatothropic. The indirect possibility of vasculation as a complication of malignancy (hepatoma) cannot be ruled out.

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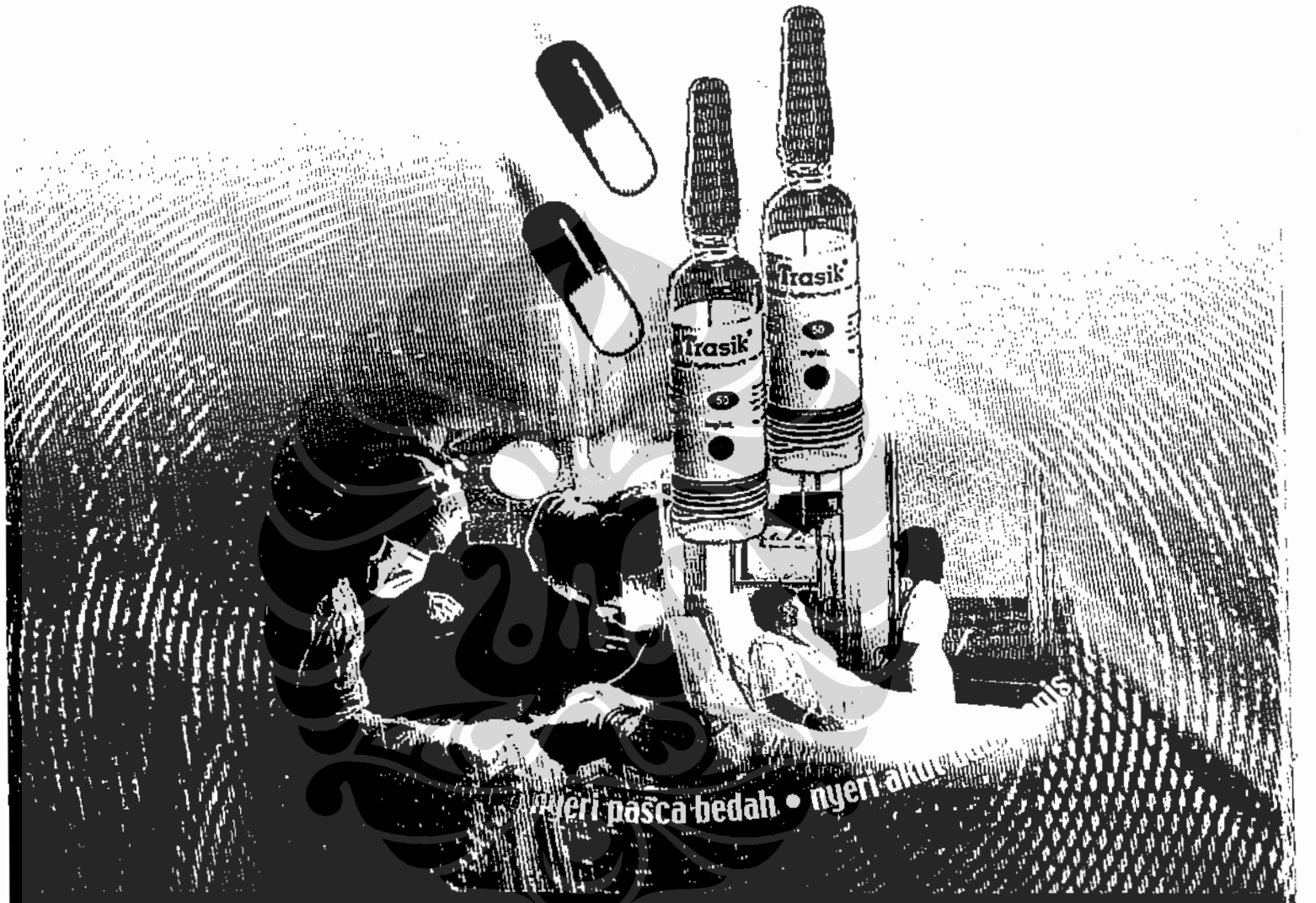

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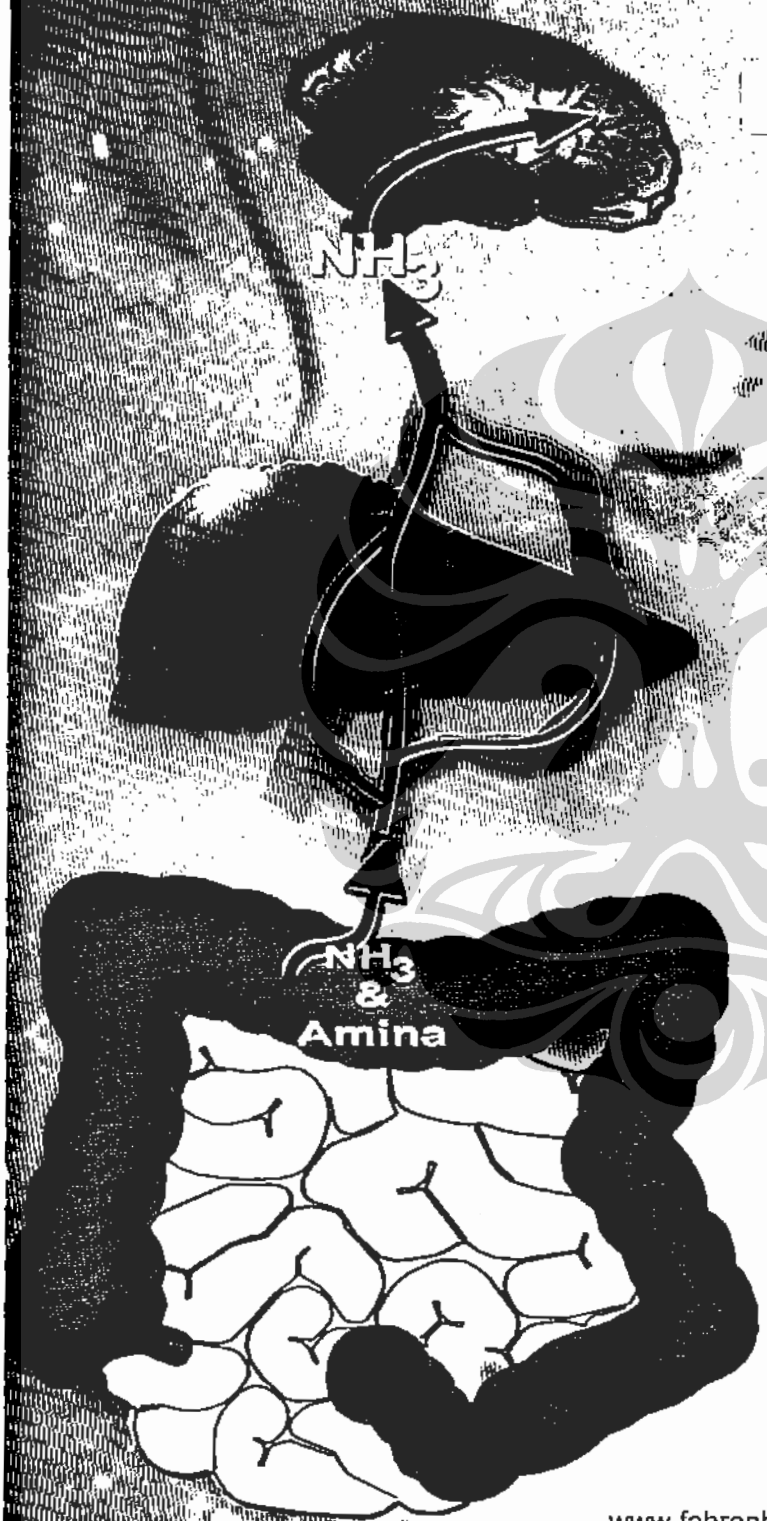

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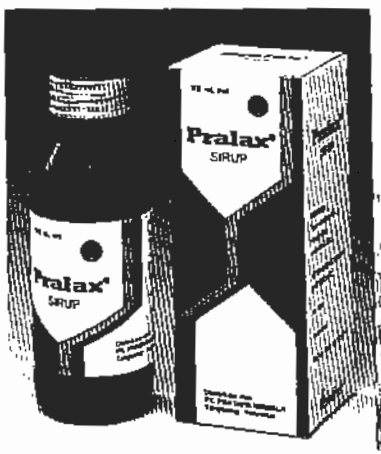

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