Diagnosis and Treatment of Leptospirosis with Complications

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ABSTRACT

Leptospirosis is a zoonotic disease cause by a microorganisme leptospira, with animals especially rats acting as the reservoir host. Humans are infected by direct contact with urine or tissues of the infected animals and indirectly by contaminated water, soil, and vegetation leptospira. Infection can result in acute renal failure, or liver damage and pancreas.

In this case patient with the problem is leptospirosis, acute pancreatitis and acute renal failure. Serologic test resulted in positive leptospira with titer 400 for serovar Hardjo and titer 100 for serovar Bataviae.

Keywords: Leptospirosis, Acute Pancreatitis, Acute Renal Failure

INTRODUCTION

Leptospirosis is a zoonotic disease caused by the microorganism, *Leptospira*, with animals especially rats acting as the reservoir host. *Leptospira* is carried in the kidney or urine of the animals. Humans are exposed to it through the animal's urine. Currently, there are serotypes that can cause diseases with severe clinical syndromes. Moreover, the disease may be fatal, such as that caused by *L. icterohaemorrhagiae*. However, there are some serotypes that can cause mild clinical syndromes, such as *L. autumnalis*, *L. bataviae*.[1,2]

This disease is spread throughout the world, prominently in the tropics. It can infect men and women of all ages. Most of the cases involve young men; 50% of the cases involve persons between the ages of 10 to 39 years, and 30% are men.[2]

Humans are infected by direct contact with the urine or tissues of the infected animals and indirectly through contaminated water, soil, and vegetation. *Leptospira* enters the human body through abraded skin, mostly of the feet, conjunctival, nasal and oral mucous membranes. Then it multiplies and spreads to human organs and tissues. In the kidney, *Leptospira* will reach the convulated tubules and form colonies in the lumen walls. Finally, it enters the ureter.[3]

Leptospira infection can result in acute renal failure, or damage of the liver and pancreas. In the pancreas, ischemia of the organ appears to transform mild edematous pancreatitis into severe hemorrhagic necrotizing forms of the disease. The mechanism of change in the organs is still unclear. There is suspicion that the changes may be due to nephrotoxin, immunological reaction, renal ischemia, haemolysis, or direct invasion of the microorganism to the organs.[3,6]

Clinical manifestations begin with mild symptoms such as mild fever, chills, nausea, vomiting, headache, muscle ache and tenderness, especially of the gastrocnemius muscles, thighs, and lumbar areas. Reduced consciousness is found in 25% of cases.

Relative bradycardia and infected conjunctiva appear on the third and fourth days after the infection. On the skin, macular and maculopopular rash or urticaria is found spreading all over the body. Splenomegaly and hepatomegaly are detected on the examination of the abdomen.[1,2,7,8]

The final diagnosis is confirmed upon detection of *Leptospira* in the blood, urine or positive result on serologic test of *Leptospira*.

Treatment of *Leptospira* includes bed rest, administration of antibiotics and analgesics, and rehydration. With effective antibiotics, there should be improvements within 7 to 10 days after the infection.

The prognosis of the disease depends on the general condition of the patient, the patient's age, and the virulence of the *Leptospira*. Death usually results from secondary factors such as renal failure liver failure or bleeding.[1,2,3,9,10]

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CASE REPORT

Mr. K., a 45-year old driver, was admitted to dr. Cipto Mangunkusumo National General Hospital from November 2nd 2000 to December 1st 2000, with a chief complaint of weakness three days prior to hospital admission.

From the present history, we found that he had had a high fever since 9 days prior to admission. Sometimes, he had chills, especially at night. He also complained of stomachache and epigastric pain, nausea, and vomiting of stomach contents resulting in a decreased appetite. He had headaches and muscles fatigue particularly in the legs. He complained of having yellowish eyes and skin. His urine was yellow like the color of tea, and the quantity was decreased.

Three days prior to hospital admission, his temperature increased. He went to a general practitioner who said he suffered from a liver disease, and was referred to dr. Cipto Mangunkusumo National General Hospital.

From the past history, we found that this was the first time that he suffered from a disease like this. He denied of ever having hepatitis, kidney disease, heart disease or gastritis.

He worked as a driver and lived in a very crowded area on the riverbank where there were so many rats. When he is at home, he was often exposed to dirty water.

On physical examination, his general condition was weak, he looked very sick, apathetic, with a blood pressure of 130/80 mmHg, a pulse rate of 92 times/minute, a temperature of 38°C, a respiration rate of 20 times/minute. Cilliary injection was found in both eyes; his sclera was icteric; the lymph nodes of the neck were impalpable, and there was no neck stiffness. Spider nervi was not found on the thorax, his first and second heart sounds were clear, without murmur or gallop. His lungs were sonor, vesicular, with no rales or wheezing. His abdomen was supple and flat, and ascites was not found. His liver was palpable 2 fingers below the rib arc while the spleen was impalpable. There was pain on palpation throughout the abdomen. Acute abdomen was not found, bowel sounds was decreased, there was sufficient turgor, there was no pain when the costovertebrae angle was tapped, there was no palmar erythema in the extremities, his acrals were warm, and there was pain on palpation of musculus gastronemius of both lower extremities.

Laboratory findings when admitted to the hospital were as follows: hemoglobin level 10.4 g/dl, haematocryte 27, leukocyte count 25,800, platelet count 363.000/ul, ureum level 120 mg/dl, creatinine level 6,5mg/dl, blood sugar level 156, amylase level 1948 u/l, lipase 1326 u/l. ECG results were within normal limits. Chest x-ray demonstrated a CTR of <50%, and no infiltrate.

It was concluded that the problems in this patient were as follows: Leptospirosis, acute pancreatitis and acute renal failure.

Leptospirosis was diagnosed based on these findings: high fever, chills, headache, jaundice, pain on palpation of musculus gastronemius, and cilliary injection of both eyes. The patient lived in a crowded area where there were rats everywhere and he was often exposed to dirty water. The patient was treated with 1 x 2 g of Ceftriaxone. On the fourth day, his temperature had not decreased, and an itchy erythema was found on the chest, stomach and arms. The new problem was drug eruption due to cephalosporin. Two x 1 vials of intravenous Avil and dexamethasone were administered. The antibiotic was switched to 2 x 500 mg of oral ciprofloxacin (according to the recommendation of the subdivision of allergy-immunology and tropical infection).

On the twelfth day, the patient's fever was falling and the patient's general condition also demonstrated clinical improvements. Serologic test resulted in positive *Leptospira* with a titer of 400 for Hardjo serovar and a titer of 100 for Bataviae serovar. Ultrasound examination demonstrated hepatomegaly and acute pancreatitis. After seventeen days of treatment, the patient's jaundice was reduced, with a direct bilirubin level of 3.9 and an indirect bilirubin level of 1.5, reduced from a direct bilirubin level of 21.6 and an indirect bilirubin level of 19.5.

The problem of acute prancreatitis was based on nausea, vomiting, epigastric pain and pain throughout the abdomen, an amylase level of 1948 u/l and a lipase level of 1326. This was thought to be a complication of leptospirosis. The abdominal ultrasound examination showed peripancreatic edema. The patient was treated using a nasogastric tube, and the patient was requested to fast and received parenteral nutrition of Triofusin E 1000: 12 hours/kolf and 10% dextrose, 0.9% NaCl/6 hours, and 2 x 1 vials of ranitidine. Amylase and lipase levels decreased to 50 g/uL and 900/uL respectively on the fifteenth day, and there was clinical improvement. The patient was given a liquid diet of 3 x 100 cc. It was gradually increased to a pancreas diet until the twenty-fifth day of treatment. On the thirtieth day of treatment, the patient's amylase and lipase levels decreased to 47/ul and 795/ul respectively.

Acute renal failure was confirmed from a history of decreased amount of urine, and a ureum and creatinine level of 120m/dl and 6.5 mg/dl respectively. Acute renal failure was thought to be due to leptospirosis infection

and dehydration due to vomiting. During treatment there was improvement of the renal function, shown by increased urine production. The patient's ureum level increased to 46 mg/dl, and his creatinine level to 1.1 mg/dl. The patient was under observation for liquid and electrolyte balance.

DISCUSSION

Leptospirosis is a common infectious disease. It is a zoonotic disease with various clinical manifestations. However, the most frightening thing about it is the complications, which can result in mortality if treatment is inadequate.[1,2]

In the case study above, the complication was acute pancreatitis with clinical manifestations of nausea, vomiting, increased temperature, and increased amylase and lipase levels. From abdominal ultrasound examination, peripancreatic edema was found.[2,9]

From the reference, it is stated that the diagnosis of pancreatitis can be confirmed if the level of amylase and lipase were 3 times greater than normal. In this case study, pancreatitis was the result of *Leptospira* infection, confirmed from positive serologic testings for *Leptospira* (400 for serovar Hardjo and 100 for serovar Bataviae). The mechanism of acute pancreatitis in leptospirosis is still unknown. The treatment is conservative directed to limit the inflammation process and autodigestion by reducing the secretion of pancreatic enzymes by means of fasting and installing a nasogastric tube and maintaining liquid and electrolyte balance, with administration of parenteral nutrition with adequate calories. If the patient's condition improves, oral nutrition may be given gradually.[1,2,3,9]

The complication of acute renal failure is caused by *Leptospira* that reach the convulated tubules, possibly releasing nephrotoxin or causing an immunological reaction and ischemia; even though the mechanism for this is still unclear. The patient is treated with antibiotics to cure the infection, while liquid and electrolyte balance is maintained.[1,2,7]

The antibiotic treatment originally used in this case study was 1 x 2 g of Ceftriaxone, which then caused generalized erythema. This was thought to be a drug eruption, but it might also be a manifestation of *Leptospira* on the skin, which is macular, maculopopular rash or urticaria spreading on the body.

The prognosis of this case was good because the general condition of the patient was good, he was relatively young, and adequate antibiotic was given early.

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