WEIL'S DISEASE WITH PNEUMONIA: A CASE REPORT

Evi Nurhayatun¹, Dhani Redhono Harioputro²
Internal Medicine Department Faculty of Medicine Sebelas Maret University, Surakarta, Indonesia¹,²
evi.nurhayatun@staff.uns.ac.id¹, dhani_redhono@staff.uns.ac.id²

ABSTRACT

Background: Leptospirosis is zoonotic infectious disease caused by the spirochaete Leptospira interrogans. It can usually be transmitted indirectly, per contaminated water, rarely directly, and through contact with infected animals. Leptospira bacteria commonly enter the body through the damaged skin or mucous membranes. The clinical syndromes may vary from a subclinical infection and mild febrile condition to severe clinical symptoms with jaundice and renal failure.

Case report: It is the case report from a woman 48 years old with Weil's disease with clinical manifestations included: jaundice, headache, pain in the calves, septic shock, pneumonia, renal failure, gastrointestinal bleeding and disturbances of consciousness. After the use of antibiotics, free-heparin hemodyalisis, symptomatic and substitution therapy, all symptoms resolved.

Discussion: Negative leptospirosis result that may caused due to antibody level have not been formed in the blood or antibody level still low because blood sample taken when disease onset less than 5 days while IgM level can be detected in the blood commonly after 5 days or will be better if more than 7 days. Patient has risk factors and clinical sign to severe leptospirosis with decreased consciousness, icteric, conjunctival suffusion, acute kidney failure that proved by kidney USG result not yet found chronic kidney disease and gastrointestinal bleeding

Conclusion: Mortality rate in severe leptospirosis or weil disease averages approximately 80%, with proper antibiotics and supportive treatment, and hemodialysis in patients will help reduce mortality in weil’s disease

Keywords: leptospirosis, Weil’s disease, pneumonia

I. INTRODUCTION

Leptospirosis is an acute zoonoticinfectious disease caused by the spirochaete Leptospira interrogans. Human leptospiral infection results primarily from exposure to the urine of infected rats.¹ Leptospires can contaminate humans through cuts and abrasions of the skin, through intact mucous membranes and the waterlogged skin. An incubation period of 5–14 days is typical. During this time, the spirochetes proliferate in the bloodstream and then disseminate hematogenously.

The clinical manifestations of leptospirosis in humans vary from a subclinical infection to severe illness with multiorgan dysfunction. The illness itself has a biphasic nature: an initial septicemia phase and consequent immune phase. During septicemia, patients will present with fever, headache, myalgia, conjunctival suffusion, and various non-specific findings such as mild cough, rash, lymphadenopathy, nausea, and vomiting.² Subsequently, patients may have a brief afebrile period of variable duration whereafter they develop organ derangements, most commonly of the liver and kidneys. Severe leptospirosis is the same with Weil's disease which is characterized by kidney and liver failure. Antibiotics should be as soon as the diagnosis is suspected. We showed such a severe case leptospirosis with complication and its management.

II. CASE REPORT

A 48-years-old woman came to the dr. Moewardi Hospital Emergency Room with a chief complaint of fever for five days. The fever has been felt suddenly, risen slowly and down not until normal usually in the morning. Fever
has improved with medication. The patient also complained diffuse myalgias, abdominal discomfort, nausea, jaundice, shortness of breath, cough, stabbing headache, and pain in the calves got worse when activity and has not decrease when the patient rest. At the time she denied seizure, shiver, cold sweat, diarrhoea, constipation, neither frequency nor urgency in urination. She works as a janitor. Her job is clean the warehouses and gutters with lots of rats. She denied history of recent travel especially to eastern Indonesia, visiting endemic malaria region, eating uncooked foods, and animal bites. The family and patient’s neighbors have not complaint like the patient. The patient had no history of jaundice, kidney disease, cardiovascular disease, hypertension and diabetes mellitus.

On exam, she was found to be tachycardic (102 beats per minute [bpm]), blood pressure of 120/60 mmHg, and febrile (39.6°C), scleral icteric, bilateral conjunctival suffusion, the skin presented jaundiced (yellow), petechie and gastrocnemius pain. The chest examination revealed crackles in both lungs. There was no finding hepatomegaly, and Murphy signs negative. Laboratory examination showed an increase leukocyte 15.6 x103/µL, neutrophils 84.60%, spartate aminotransferase (AST) of 46 u/L, total bilirubin total 15.96 mg/dL, ureum 205 mg/dL. Laboratory examination showed an decrease hemoglobin of 9.1 g/dL, erythrocyte 3.30 x 106/µL, limfosit 12.20%, albumin 2.6 g/dL, sodium 125 mmol/dL. Blood gas analysis showed PH 7.5, BE 2, l Mmol/L, PCO2 28,4 mmHg, PO2 49,2 mmHg, HCO3 24,7 Mmol/L, Laktat 1,40 Mmol/L. Sofa Score was 12 (Motality 40 – 75%). Ectrocardiography showed sinus takikardi at 102 beats/min normoaxis -10o transition zone V2 – V3. Chest Xray examination showedlung oedema with left pleura effusion, bilateral pneumonia.

Because of the initial presentation and the patient’s history, leptospirosis was treated with injection Ceftriaxone 2g per day, injection Metamizole 500 mg per 8 hours, and supportive therapy. The following day, serologic test results for Ig M leptospirosis was negative. Renal and abdominal ultrasonography results were normal. The diagnosis was made using microscopic aglutination test (MAT). Serum MAT finding elevated level serovar icterohaemorrhagiae, which confirmed the suspicion of leptospirosis. Sputum culture was found Pseudomonas aeruginosa. Antibiotic sensitivity test showed that Cefipime, Ciprofloxacine Ceftazidime Gentamicin were sensitive. Antibiotic therapy replaced by ceftazidime 1 gram per 8 hours.

On the third day, patient’s decrease onsciousness and getting worse, serum creatinine was 6,1 mg/dL. The patient was also diagnosed with Weil’s disease, hyperbilirubin with comorbid septic shock, pneumonia, acute kidney injury, gastrointestinal bleeding, anemia and received a transfusion 4 kolf packed red cell and free heparin-free hemodialysis. During treatment for 26 days, patients experience improvement conditions. One week after hospitalization, the patient went to policlinic with the chief complaint headache, jaundice and conjunctival suffusion were improvement. Then chest Xray performed bronchopneumonia cardiomegaly (LV) with aorta elongation.

III. DISCUSSION

In this patient, we had complaint of fever since 5 days before admission. The patient also complained diffuse myalgias, abdominal discomfort, nausea, jaundice, shortness of breath, cough, stabbing headache, and pain in the calves. Then from the lab result we found thrombocytopenia, hyperbilirubinemia and azotemia. Therefore, we diagnosed the patient with Probable Leptospirosis based on WHO SEARO criteria. In our patient, we found negative leptospirosis result that may caused due to antibody level have not been formed in the blood or antibod level still low because blood sample taken when disease onset less than 5 days while IgM level can be detected in the blood commonly after 5 days or will be better if more than 7 days. Patient has risk factors and clinical sign to severe leptospirosis with decreased consciousness, icteric, conjunctival suffusion, acute kidney failure that proved by kidney USG result not yet found chronic kidney disease and gastrointestinal bleeding.

We obtain WHO criteria and modified faine score in patient which leads to leptospirosis. Therefore, we conduct gold standard test to detect antibody against Leptospia interrogans which is Microscopic Agglutination Test (MAT) using living organism. In general, agglutination test will not positive until first week of infection, peak level of antibody will be 3-4 weeks after symptom onset and settle for several years although the concentration will subsequently decline. MAT test detects antibody at serovar level which can be used to identify Leptospira strain in human and gold animal. MAT become gold standard for diagnosing leptospirosis due to it has high sensitivity. MAT stated positive if there is seroconversion in titer increment 4 times or more than 1:320.4 In this patient, increment occurred almost in all serovar with highest titer at type of serovar hardjo 1:10240, javanica 1:10240, Sejroe 1:10240, Shermani 1:10240, Icterohaemorrhagiae 1:5120 and Tarassovi 1:20480. Serovar
Icteric haemorrhage often associated with leptosira infection in mice.\(^5\) We diagnose patient with confirmed leptospirosis or Weil disease due to MAT positive.

Leptospirosis is an acute infection disease that can attack human through animals (zoonosis) due to leptospirosis interrogans bacteria, it is spiral aerobic bacteria with very small size just 0.1 µm x 0.6 µm until 0.1 µm x 20 µm. The life span of leptospirosis approximately one month in fresh water, but it will die quickly in sea water, sewage water, and undiluted urine because of their sensitivity to acids.\(^5\) In animal body leptospiroa will settle and form colony, it will multiply in renal tubules epitel and will flow continuously in urine filtrate. Transmission from animal to human will be through water or soil that contaminated with fluid or tissue from infected animal. We suspect the infection occurred in the patient because the patient has risk factor due to working as a janitor. there are lots of rats and patient habit that working without footwear protection and glove. Therefore, bacteria can get in through skin abrasion on hands and feet or through water splash that get into eye mucosa or oronasopharyngeal mucosa.

After entering the body, leptospiroa infiltrate various organ especially liver and kidney. Complication that often occurred on leptospirosis patient is acute renal failure, characterized by oliguria or polyuria which can occur 4-10 days after symptom on leptospirosis appear. Renal failure at leptospirosis patient occurred through 3 mechanisms: Invasion/direct nephrotoxicity from leptospire, immunological reaction, non-specific reaction to infection such as other infections.\(^6\) In this patient, acute renal failure found with increasing level of ureum: 205 mg/dL and creatinin: 6.6 mg/dL. Dialysis can be done on patient with leptospirosis if there is azotemia/uremia. It complies with Kidney Disease Improving Global Outcome (KIDGO) criteria which drafted guideline for Acute Renal Disorder. Another complication in the liver is necrosis centrilobular with proliferation of Kupffer cells and cholestasis. Jaundice in leptospirosis is caused by several things, including damage to liver cells, impaired kidney function will reduce the excretion of bilirubin in the blood, bleeding in the tissues and intravascular hemolysis will increase bilirubin levels, Typically this disease is biphasic, which has a leptospiremic / septicemic phase and an immune phase.\(^7\)

### IV. CONCLUSION

It is a case report of a 48 years old woman with Weil’s disease. Weil’s disease represents the most severe form of leptospirosis, weil’s with complicated by gastrointestinal bleeding, acute renal failure and hyperbilirubin with comorbid septic shock, pneumonia and pulmonary edema. Patien was treated for 20 days in ICU and received antibiotic treatment, and hemodialysis. Mortality rate in severe leptospirosis or Weil disease averages approximately 80%, with proper antibiotics and supportive treatment, and hemodialysis in patients will help reduce mortality in Weil’s disease.

### REFERENCES


www.turkiphysiotherrehabil.org