

## Case report: patient with Weil's disease, chest pain, hepatitis b, hepatorenal syndrome, and electrolyte imbalance

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**Abstract.** A 48-years-old man was hospitalized due to febrile for 5 days accompanied by headache and left chest pain radiating into the back. Pain calf, abdominal pain and tea color urine has occurred since 1 week before admission. Physical examination revealed temperature 38.5° C, conjunctival suffusion, jaundice sclera, irregular heart sound, hepatomegaly, gastrocnemius tenderness, Faine score 23. Laboratory tests showed leukocytosis, thrombocytopenia, elevated transaminase enzyme, ureum 181 mg/dl, creatinine 4.3 mg/dl, albumin 2.4 g/dl, sodium 110 mmol/L, potassium 2.3 mmol/L, reactive HbsAg, CKMB 68.06 ng/ml, increased HBV-DNA, negative IgM anti Leptospira. MAT demonstrated 4 positive serovar. Electrocardiography revealed AF rapid ventrikel response. Chest x-ray showed cardiomegaly. Abdominal ultrasound revealed hepatomegaly with chronic parenchymal liver disease, renal insufficiency. Fibroscan showed severe fibrosis. Patient was diagnosed with Weil's disease and hepatorenal syndrome as complication. Patient was given clavulanate amoxicillin injections. This patient was not dialysis, only fluid balance monitoring and checked ureum-creatinine per 3 days, and there was improvement of ureum-creatinine. Hepatorenal syndrome in Weil's disease contributed to electrolyte imbalance. In addition, chronic hepatitis B on this patient was treated with tenofovir.

### 1. Introduction

Weil's disease is a severe leptospirosis disease, caused by a bacterial infection of the genus *Leptospira icterohemorrhagiae* that can be transmitted from animals to humans. Weil's disease is the most widespread zoonosis in the world, especially the tropics. This is a redundancy from abstract, find another sentence. The disease was first reported in 1886 by Adolf Weil with high fever accompanied by neuro disorders as well as enlarged liver and spleen. The disease with these symptoms by Goldsmith (1887) is referred to as "Weil's Disease". In 1915 Inada successfully proved that Weil's Disease is caused by the bacterium *Leptospira icterohemorrhagiae*.<sup>1</sup>

Weil's disease is a problem in the world, the incidence rates are low in most countries, because of difficulties to establish diagnosis, so the true events are unknown, but in the tropics the estimated case of Weil's disease is 10-30 per 100,000 population per year.<sup>2</sup>

Risk factors that are thought to play a role in Weil's disease are physical environmental factors, biological environmental factors, and individual factors. Physical environmental factors are environmental conditions both inside and outside the home that do not include health requirements. Biological environmental factors such as the presence of livestock, pets and mice can be a source of



leptospirosis transmission. While individual factors are contact with water, soil (mud), plants that have been littered with urine Weil's disease patient, walking barefoot outside the home.<sup>3</sup>

Symptoms of this disease vary widely ranging from fever, jaundice, hemoglobinuria, in pregnant animals can occur abortus and dead birth fetus, can even cause death of the patient. The degree of Weil's disease depends on the serovar leptospira and the infected animal species in a particular area. Serological tests are widely used to confirm the diagnosis of this disease.<sup>4</sup>

Hepatorenal syndrome is a condition of acute renal failure and acute hepatic failure. Hepatorenal syndrome is potentially reversible but involves highly complex pathogenetic mechanisms and equally complex clinical and therapeutic management. Once Hepatorenal syndrome developed, it has a very poor prognosis.<sup>5</sup>

Infectious diseases caused by hepatitis B virus (VHB) is still a global health problem, because it can lead to serious liver disease ranging from fulminant hepatitis to hepatocellular carcinoma. It is estimated that around 2 billion of the world's population have been infected with hepatitis B virus, and 360 million people as HBsAg and 220 million (78%) of whom are in Asia. Five hundred thousand to 750 thousand people are thought to die of hepatic cirrhosis or develop liver cancer.<sup>6,7</sup>

## 2. Findings

Patient had fever since 5 days before admission. Fever increased at night. Fever reduced when given drugs. Fever was accompanied by headache from the middle to the back. Headache felt intermitten. Headache increased with activity and decreased with rest. There was also left chest pain radiating to the back. Chest pain increased with activity and decreased with rest.

Patient also complained of pain in both calves since 1 week before admission. Pain in the calf was felt continuously, increased when to walk or activity and decreased with rest and drug administration. Pain in the legs lately makes the patient difficult to walk.

Patient complained nausea since last 1 week. Nausea is felt continuously, sometimes accompanied by vomiting 3-4 times / day, vomiting containing food and drink, no blood. Nausea and vomiting did not decrease with rest and increased if the patient was late to eat.

Patient complained of right-sided abdominal pain since 1 week before admission. Abdominal pain felt intermitten. Abdominal pain was felt like being stabbed. Abdominal pain increased when activity and did not decrease with feeding or rest. Patient also complaint of yellow eyes. Initially the patient did not realize it but the last 3 days the patient's eyes looked more yellow than usual.

Patient complained of tea-colored urine since last 1 week. Urine reduced 5-6 times daily  $\frac{1}{2}$  -  $\frac{3}{4}$  glass and there was no pain. Defecation was within normal limits 1-2 times a day, soft consistency, yellowish brown color.

Patient had a hypertension history since 3 years ago but patient did not control and take medication routinely. Patient have no history of diabetes, liver disease, and heart disease. too many repetition words: patient, fever...find another way to explain.

On physical examination, blood pressure 130/80 mmHg, pulse rate 105 times/min, irregular, respiratory rate 26 times/min, 38.5°C temperature, VAS: 4 (in feet). There was conjunctival suffusion, jaundice sclera, I-II heart sound, interval varied, irregular, no heart murmur, palpable liver 2 cm below arcus costa dextra, 1 cm below xiphoid process, gastrocnemius tenderness, and Faine score 23.

Laboratory results showed Hemoglobin 11 g/dl, leukocyte 17000/ul, platelet 22000/ul, AST 357 U/L, ALT 214 U/L, total bilirubin 13.2 mg/dl, ureum 181 mg/dl, creatinine 4.3 mg/dl, albumin 2.6 g/dl, sodium 110 mmol/L, potassium 2.3 mmol/L, chloride 75 mmol/L, reactive HBsAg, CKMB 68.06 ng/ml, increased HBV-DNA, negative IgM anti Leptospira, serovar Icterohaemorrhagiae 1:400, Bataviae 1:400, Javanica 1:100, Tarassovi 1:100.

ECG examination revealed AF rapid ventricular response and chest X-ray showed cardiomegaly with congestive pulmonum. Abdominal ultrasound revealed hepatomegaly with chronic parenchymal liver disease and renal insufficiency. Fibroscan examination showed fibrotization F2-F3, severe fibrosis. your case is lacking the picture, either patient symptom or lab result eq.ecg

### 3. Discussion

In this case a 48-years-old man came with the chief complaint fever since 5 days before admission. From the history, patient got fever, headache, yellow eyes, calf pain, nausea, vomit, abdominal pain, and tea-colored urine. From the physical examination the patient was fully alert, febris, jaundice sclera, conjunctival suffusion, gastrocnemius pain. Laboratory tests showed azotemia, hypoalbuminemia, increased transaminase enzyme, bilirubinemia, hyponatremia, and hypokalemia. Assessment of Weil's disease based on the Faine criteria (WHO), the amount of A + B + C = 38 (A + B + C > 25 = Weil's disease) with serovar *Ichterohaemorrhagiae* 1:400, *Javanica* 1:100, *Bataviae* 1:400, *Tarassofi* 1:100, so we gave therapy based on WHO with Peniciline G 1.5 million units every 6 hours but due to the medicine supply was empty, then patient was given amoxicillin clavulanate 1 gr/12 hours intravenously because drug of choice besides penicillin is ampicillin, amoxicillin, and doxycycline. Doxycycline is recommended for mild cases and prophylaxis. Ampicillin and amoxicillin are also recommended for mild cases, penicillin G is indicated for severe Weil's disease. Penicillin has a very good effect when given on days 1 to 3, giving on day 4 to 6 are not good, and giving on day 7 is not useful. The type of penicillin given was penicillin G, 600,000 units every 4 hours. If the disease is more severe, the dose can be increased to 8-12 million units per day. Patient who come at day 7 or more, WHO recommend penicillin G 6-12 million units.

Patient had left-sided chest pain that penetrated to the back and the pain decreased slightly with rest as a manifestation of Weil's disease. Blood pressure of patient was 130/80 mmHg, ECG examination did not show either ST elevation or ST depression disorder, but AF rapid ventricular response. CKMB and troponin I increased. Risk factor in these patients is hypertension for 3 years.

There was chronic hepatitis B based on fever, yellow eyes, abdominal pain, and tea-colored urine. Physical examination got febrile and jaundice sclera. Laboratory examination showed increased transaminase enzyme, reactive HBsAg, increased HBV DNA, and fibroscan lead to chronic hepatitis B. The patient was treated with tenofovir 300 mg/24 h.

This patient got hepatorenal syndrome with Ur: 181 Cr: 4.3. Patient with Weil's disease who had severe azotemia/uremia, dialysis may be performed, based on the Kidney Disease Improving Global Outcomes (KDIGO) criteria. This patient was not dialysis, only fluid balance monitoring and checked ureum-creatinine per 3 days, and there was improvement of ureum-creatinine. Hepatorenal syndrome in Weil's disease contributed to electrolyte imbalance. These are still case presentation not discussion, there is no comparison with other research or literature.

Serum electrolyte levels (sodium, chloride, potassium, and calcium ion) in leptospirosis patient are known to be altered but not significant due to acute renal failure in leptospirosis.

Kidney disorders are provoked by hypoxia and toxins. From the research note that the increase of sodium content in distal tubules resulted in decreased glomerular filtration rate. Low sodium levels due to reabsorption of sodium and oxygen-dependent chloride in the loop of Henle. Hypoxia and toxins damage the tubular cells. Decreasing ATP synthesis in tubular cells results in elevated levels of sodium and chloride resulting in cellular edema. Decreased sodium reabsorption stimulates the osmotic receptor in the macula densa to activate the renin-angiotensin system as well as the glomerular arteriolar spasm.<sup>8</sup> Damage to the intracellular structure of the tubular cells decreases the susceptibility of protein reabsorption. The presence of oxidative damage results in increased calcium resulting in acute tubular necrosis. When tubular cell regeneration occurs, sodium reabsorption returns to normal and sodium levels in the dense macula also decrease. The presence of acute renal failure in leptospirosis contributes to increased potassium secretion caused by increased sodium due to sodium reabsorption disorders.<sup>9,10</sup>

### 4. Conclusion

Weil's disease can be cured with non pharmacology and pharmacology especially antibiotic without dialysis and the outcome was good.

**References**

- [1] Zein U 2006 Leptospirosis *Buku ajar ilmu penyakit dalam edisi keempat* vol 3, ed A Sudoyo, B Setiyohadi, *et al.* (Jakarta: Pusat Penerbitan Departemen Ilmu Penyakit Dalam Fakultas Kedokteran Universitas Indonesia) pp 1845-8
- [2] Riyanto B, Gasem M and Sofro M 1999 *Kumpulan makalah simposium leptospirosis* (Semarang: Badan Penerbit Universitas Diponegoro) pp 27-8
- [3] Soeharyo 2008 Leptospirosis: management of critical illness in disaster *Kumpulan makalah national symposium: the 2<sup>nd</sup> Indonesian SEPSIS forum* pp 1-9
- [4] Gasseem H 2000 Gambaran klinik dan diagnosis leptospirosis pada manusia *Kumpulan makalah simposium leptospirosis* (Semarang: Badan Penerbit Universitas Diponegoro) pp 17-27
- [5] Speelman P 2001 Leptospirosis *Harrison's principles of internal medicine 15<sup>th</sup> edition* vol 1, ed Braunwald, Fauci, *et al.* (New York: McGraw-Hill Medical Publishing Division) pp 1055-8
- [6] Roffi M, Patrono C, Collet J P, Mueller C, Valgimigli M, Andreotti F, *et al.* 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation *Eur. Heart J.* **10** 1-59
- [7] Gani A R, Hasan I, Djumhana A and Setiawan P B 2012 Konsensus nasional penatalaksanaan hepatitis B di Indonesia *PPHI*
- [8] Markum H 2006 Gagal ginjal akut *Buku ajar ilmu penyakit dalam edisi keempat* vol 1, ed A Sudoyo, B Setiyohadi, *et al.* (Jakarta: Pusat Penerbitan Departemen Ilmu Penyakit Dalam Fakultas Kedokteran Universitas Indonesia) pp 585-9
- [9] Hugh R, Brady and Barry M B 2000 Gagal ginjal akut *Harrison prinsip-prinsip ilmu penyakit dalam edisi 13* vol 3, ed Isselbacher, Braunwald, *et al.* (Penerbit buku kedokteran EGC) pp 1425-34
- [10] Alper 2010 The kidney *Robin and cotran pathologic basis of diseases 8<sup>th</sup> edition* ed Kumar, Abbas, *et al.* (Elsevier) pp 921-9