Case Report

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Rare Clinical Presentation of Acute Liver Injury and Symptomatic Seizure in Leptospirosis Infection: A Case Report

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ABSTRACT

Background: Leptospirosis is a zoonotic disease caused by bacteria from the genus Leptospira. It has a wide variety of clinical manifestations and could cause high morbidity if not diagnosed correctly. According to the Indonesian Health Profile Report in 2022, there were 1,419 cases of leptospirosis reported, with 139 deaths. ***Case Presentation:*** This study reports a case of leptospirosis in a 48-year-old male who presented with the main complaint of seizure. Prior medical history showed that he had a prolonged fever for more than 7 days. He worked as a farmer and lived in flood-prone areas. Physical examination showed icteric sclerae, tenderness in the upper right abdominal region, and pain in both calves. Laboratories showed acute liver injury, with highly elevated liver enzymes (ALT: 1408 U/dL, AST: 595 U/dL), without other severe manifestations, such as acute kidney injury or hemorrhage manifestations. The serology test showed a positive IgM antibody to Leptospira. The patient was treated with cefotaxime intravenously and other supportive therapies. After 10 days, the patient was discharged with improvement in clinical symptoms and laboratory parameters. ***Conclusion:*** Given its unusual presentations, diagnosing leptospirosis can be challenging, especially in tropical regions. This report aims to describe unusual findings in leptospirosis and to raise awareness of its possible etiology in a patient with acute liver injury, particularly in a high-risk individual in a tropical region.

INTRODUCTION

Leptospirosis is a zoonotic disease caused by infection with bacteria of the genus Leptospira [1]. The bacteria mainly come from rodents, the primary carriers, and are excreted into the environment via urine, where they may infect other organisms [1,2]. This organism infects both wild and domestic mammals, making it an occupational hazard for people who regularly handle animals, such as livestock farmers and animal control workers [1,3]. This infection can occur when animal urine or contaminated water or soil enters through a wound in

the skin and comes into contact with the mucosa [1,3]. Leptospirosis occurs worldwide, but it is especially present in tropical and subtropical areas with heavy rainfall [4]. Commonly, Leptospirosis can present in 2 distinct clinical syndromes, icteric or anicteric, where anicteric is rarely fatal and represents approximately 90% of documented Leptospirosis cases. The icteric phase of leptospirosis is the severe form of leptospirosis, with prominent manifestations including renal failure, jaundice, hemorrhage, and respiratory distress; it may also involve the heart, central nervous system, and muscles [5]. The severity of leptospirosis is not predictable; it is influenced by host-related factors and the pathogenicity of the microorganisms [6].

According to the Indonesian Health Profile Report in 2022, there were 1,419 cases of leptospirosis reported, with 139 deaths [7]. It is also reported in the 2019 World Health Organization (WHO) report that the number reported was an underestimate of leptospirosis occurrence in Indonesia [4]. There are a lot of reports and studies of Leptospirosis involving liver injury from mild with slightly to moderately elevated liver enzymes to severe manifestations and unusual complications such as Weil's Disease, liver Abscess, and failure with multi-organ dysfunction [8-11]. There are also very few reported cases of leptospirosis and the occurrence of symptomatic seizures related to neuro-leptospirosis [12,13]. While most reported cases showed only slight to moderate elevations in baseline liver enzyme levels in mild and severe Leptospirosis Infection, these levels were not as high as in our cases.

This case presented a leptospirosis patient with clinical signs of prolonged fever, icteric sclera and skin, slightly palpable liver, highly elevated liver enzymes (ALT: 1408 U/dL, AST: 595 U/dL), and a neurological sign, such as symptomatic seizure, but without Weil's or other severe manifestations. It is concerning that the small reported number of cases is due to wide clinical variation and a lack of awareness among patients with leptospirosis. This case report purpose is to describe unusual findings in leptospirosis infection with highly elevated liver enzymes without develop any disorders to multi-organ failure, such as acute kidney injury, or hemorrhage manifestations with the occurance of neurological manifestasion such as symptomatic seizure, and to increase awareness of possible etiology of leptospirosis in a patient with acute liver injury, especially in a person with a high-risk factor of leptospirosis in tropical region.

CASE PRESENTATION

A 48-year-old male patient was admitted to a hospital in Samarinda, East Kalimantan Province, Indonesia, in October 2024 with a seizure of approximately 40 minutes before arriving at the hospital. During the treatment in the Emergency Room, the seizure occurred again and lasted for less than a minute. The seizure had the same characteristics as the previous episode. It was said that the patient had a prolonged fever for more than 7 days, and was not relieved with paracetamol. The patient noticed that his skin color changed to yellow, and he felt pain in the

upper right abdominal region, nausea, and vomiting. The patient also experienced generalized fatigue and myalgia, and a very painful sensation, especially in both of his calves. It was known that the patient lived on a farm in Malinau, North Kalimantan Province, and worked as a farmer. He had poultry in his barn and often went to the forest. His house often flooded easily when it rained.

On physical examination (1st day), the patient was conscious, with a Glasgow Coma Scale (GCS) score of 15 (E4V5M6), and moderately ill. His blood pressure was measured at 113/72 mmHg, with pulse rate 64x/minute, respiratory rate 20x/minute, temperature 38.50 C, and SpO2 96%. On general physical examination, the patient had slightly icteric sclerae bilaterally. Chest examination revealed no abnormalities. On abdominal examination, the liver was slightly palpable; tenderness and pain occurred with pressure. The spleen was not palpable. Skin examination showed a visible tattoo on his left arm, no rashes, ecchymosis, or any signs of hemorrhage. In the lower extremities, both calves were tender. Neurological examination, including neck stiffness and the meningeal irritation test, was negative.

Further examination of the complete blood count and electrolyte showed a low erythrocyte (4.46×10^{6}/uL), hemoglobin (12.3 gr/dl), platelet count (85×10^{3}/uL), and natrium level (129 mmol/L). The patient had a previous result of abdominal ultrasound from another hospital, which showed chronic gastritis without abnormality of another abdominal organ. Chest X-ray showed no abnormalities. Initially, the patient was given a normal saline infusion (NaCl 0,9%), a 10 mg diazepam suppository, a 500 mg paracetamol tablet, and a 100 mg phenytoin tablet every 8 hours.

Patient examinations were conducted daily in the ward. On the 3rd day, the patient continues to complain of the same manifestations. Temperature still fluctuated, liver still palpable, and pain reported, but no seizure reported. Phenytoin tablet was stopped and switched to oral levetiracetam 500 mg twice a day for seizure management. The patient was also given cefotaxime 1 g intravenously every 8 hours. The electroencephalogram (EEG) showed no abnormalities (Figure 1). Laboratory findings showed markedly elevated liver enzymes on the 3rd day (ALT: 1408 U/dL, AST: 595 U/dL). The patient had negative results on the 4th day for anti-HAV, HBsAg, and malaria serology. On the 5th day, it also showed a negative anti-HCV serology test, but had a positive result for Leptospira IgM antibody (Table 1). Thus, the patient was diagnosed with leptospirosis infection with acute liver injury and acute symptomatic seizure. The leukocyte level was also within a normal range in this case (Table 1).

The treatment was the same after the result showed positive leptospira antibody. Daily follow-up showed normal vital signs, with a fluctuating fever from the 1st to the 6th day. Seizure was an absence during admission to the ward. Upper abdominal discomfort, including palpable liver, myalgia, and pain in both calves, lasted until the 9th day. After that, the patient had no significant complaints, only generalized weakness. He was allowed to leave the hospital on the

11th day with stable vital signs and hemodynamics, and improved liver and Bilirubin parameters (Table 1).

Table 1. Laboratory Examination

Parameters	Results					
	Day 1 5/10/24	**Day 2 6/10/24**	**Day 3 7/10/24**	**Day 4 8/10/24**	**Day 6 10/10/24**	**Day 10 14/10/24**
Hemoglobin (g/dL)	12.3	13.3	13.6	13.3	**-**	**-**
Erithrocyte (10^6/uL)	4.46	4.52	4.51	4.56	-	-
Leukocyte (10^3/uL)	4.89	6.57	5.62	7.29	-	-
Monocyte (%)	2%	0%	7%	-	-	-
Thrombocyte (10^3/uL)	**85**	**114**	**100**	**127**	-	-
Ureum (mg/dL)	31.5	-	-	25.6	-	-
Creatinine (mg/dL)	0.7	-	-	0.7	-	-
ALT (U/L)	-	-	**1408**	-	**466**	**212**
AST (U/L)	-	-	**595**	-	**62**	**43**
Direct Bilirubin (mg/dL)	-	-	-	8.0	8.8	3.6
Indirect Bilirubin (mg/dL)	-	-	-	1.3	0.5	1.1
Total Bilirubin (mg/dL)	-	-	-	9.3	9.3	4.7
Na (mmol/L)	129	-	-	-	-	-
Anti HAV	-	-	-	Non-reactive	-	-
HbsAg	-	-	-	Non-Reactive	-	-
Anti HCV	-	-	-	-	Non-Reactive	-
Malaria	-	-	-	Negative	-	-
IgM anti-leptospira	-	-	-	-	**Reactive**	-

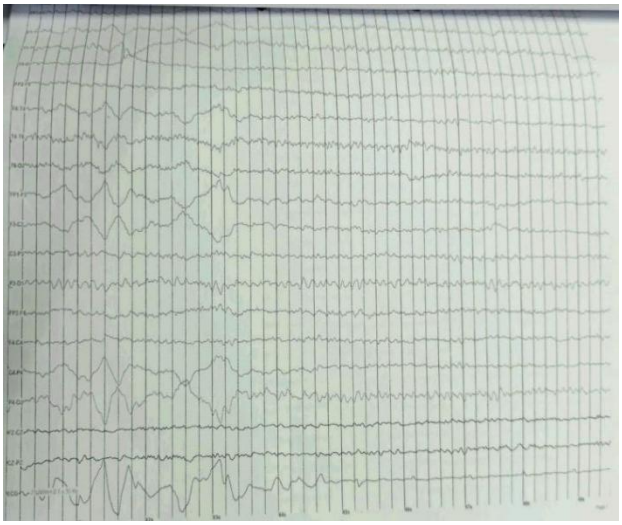

Figure 1. The electroencephalogram showed no abnormalities.

DISCUSSION

In this report, we presented a case of leptospirosis with acute liver injury and acute symptomatic seizure without multiple organ failure. Leptospirosis is diagnosed on clinical suspicion and confirmed by molecular testing, serology, culture, and antigen detection. The clinical presentations range from undifferentiated febrile illness to severe and multi-organ failure, involving kidney and pulmonary manifestations. It usually consists of the triad of hemorrhage, jaundice, and acute kidney injury, also known as Weil's Disease [3,14].

Acute liver injury is a common clinical condition faced by clinicians. The most common infectious agents causing hepatic injury are hepatotropic viruses, including hepatitis A, B, C, D, and E [3]. In this case, the diagnostic challenge was the initial seizure presentation, the potential source of infection, and the history. We checked the most likely parameters, including anti-HAV, HBsAg, and anti-HCV for viral hepatitis, malaria, and, finally, antibodies for leptospira, which were reactive on the 6th day. Leptospira penetrates the host and reaches the bloodstream, promoting bacteremia. This process can infect and colonize many organs, such as the spleen, liver, lungs, and kidneys. In the liver, leptospirosis disrupts hepatocytes, disrupts intracellular junctions, and leads to elevated liver enzymes and direct bilirubin cholestasis, resulting in jaundice. It could also colonize in the renal tubules and cause bleeding disorders [15]. This patient had developed icteric leptospirosis with highly elevated liver enzymes; however, during hospitalization, this patient did not develop any disorders or multi-organ failure, such as acute kidney injury or hemorrhage manifestations. Several reports of Leptospirosis stated mildly to moderately elevated liver enzymes. One of the reports described derangements in liver function tests among patients with Leptospirosis in a health care center, with a mean baseline AST of 105.8±72.9 and ALT of 55.9±28.9 in 100 patients who were IgM lepto-antibody-positive [8]. There were also reports of

Leptospirosis with complications such as acute kidney injury with elevated AST (207 U/L) and ALT (102 U/L) [3]. At the same time, other studies reveal acute liver and kidney failure with elevated AST (91 U/L) and ALT (113 U/L) [9]. In contrast, it was slightly elevated and caused other organ damage; our case didn't progress to multi-organ failure, although AST and ALT were highly elevated. The presence of the seizure showed that the patient had a neurological manifestation of leptospirosis. The common and usual neurological symptoms include encephalitis, seizure, cerebellitis, movement disorders, myelitis, flaccid paraplegia, neuralgia, polymyositis, acute disseminated encephalomyelitis, altered sensorium, deeply comatose state, and acute symptomatic seizure [12,16]. As far as we know, there were only a few reports about the acute symptomatic seizures in leptospirosis infection. This report concluded from a total of 31 patients, where eleven (35.5%) had acute symptomatic seizures at the time of presentation [12]. Neurological forms may arise from the CNS via leptospira or from an immune-mediated response to the bacterium [13]. We defined acute symptomatic seizure in this case as one that occurs in relation to an acute CNS insult, which may be metabolic, toxic, structural, infectious, or inflammatory. The seizures in this case were most likely caused by leptospirosis infection itself or as a result of metabolic disorders (hyponatremia). It is also different from an epileptic seizure, which happens without any apparent precipitating factors or conditions [17]. These clinical manifestations make it a distinct and unique clinical variant of leptospirosis compared to its usual presentation.

It remains unclear which factors determine the severity of leptospirosis. Host immune responses, with extensive release of inflammatory cytokines such as interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor-α (TNF-α), contribute to clinical manifestations and pathogenesis. High leptospiral load and different Leptospira serogroups, which have different leptospiral lipopolysaccharides (LPS) and hemolysin, may be other factors contributing to disease severity [18].

Leptospirosis is detected by detecting the presence of the organism or its metabolites in body fluids or tissues, using serological tests and culture. It is observed that leptospirosis antibodies develop only 3–10 days after symptom onset. The gold standard test for leptospirosis is the microscopic agglutination test (MAT). Other diagnostic tests include enzyme-linked immunosorbent assay (ELISA), IgM, Immuno DOT, and lateral flow tests [19]. In this case, the patient's diagnosis was supported by a positive result on the IgM anti-leptospira serology test using the immunochromatographic method. One study showed that a rapid diagnostic test using the immunochromatographic assay method had 97.4% of sensitivity and 94.5% of specificity for diagnosing acute leptospirosis [20]. In the meta-analysis report, rapid diagnostic tests showed 75% and 85% sensitivity and 87% and 79% specificity when MAT and ELISA were used as reference tests, indicating a moderate level of acceptance for detecting acute leptospirosis [21].

CONCLUSION

Acute liver injury, marked by highly elevated liver enzymes, without multi-organ damage, along with the presence of seizures, represents a rare clinical presentation of leptospirosis and should be considered in suspected cases. Careful and precise clinical evaluation, along with appropriate diagnostic testing, is essential to identify and exclude other possible etiologies of acute liver injury and seizures.

DECLARATIONS

Ethics approval

This study was approved by the Health Research Ethics Committee of Abdoel Wahab Sjahranie General Hospital, Samarinda.

Conflict of interest.

The authors declare no conflict of interest

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