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Case Report

Tackling the ST elevation in leptospirosis: A double-edged sword between bleeding and thrombosis – A case report

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Abstract

Although leptospirosis is a well-recognized zoonotic disease, the occurrence of ST-segment-elevation myocardial infarction (STEMI)-mimicking leptospiral myocarditis, accompanied by subsequent bleeding and thrombocytopenia is an exceptionally rare finding. The dual risks of bleeding and thrombosis further complicate the management of anticoagulation and thrombolytic therapy amidst competing risks. The aim of this study was to present leptospirosis complicated by myocarditis, which mimicked STEMI, followed by bleeding and thrombocytopenia. A 61-year-old male patient was referred from a community health center to the hospital with primary complaints of chest discomfort and diaphoresis, which had started 11 hours prior to admission. These symptoms were associated with a 12-day history of intermittent fever, nausea, and vomiting. Upon physical examination, the patient appeared lethargic, with a blood pressure of 86/63 mmHg, heart rate of 107 bpm, respiratory rate of 22 breaths per minute, and temperature of 39.8°C. Electrocardiography revealed widespread ST-segment elevation. Echocardiography showed global hypokinesia with a reduced ejection fraction of 48%. Laboratory tests confirmed the presence of IgM and IgG anti-*Leptospira* antibodies, along with elevated high-sensitivity cardiac troponin levels. The patient was diagnosed with Weil's disease (Faine's score 32), with leptospiral myocarditis and STEMI considered as differential diagnoses. Initial management involved a loading dose of dual antiplatelet therapy (aspirin 320 mg and clopidogrel 300 mg) due to the suspected diagnosis of STEMI. However, it was later discontinued on the second day of admission due to the development of severe thrombocytopenia and minor bleeding manifestations. Following the administration of ceftriaxone 2 g every 12 hours and doxycycline 100 mg every 12 hours, the patient's condition improved. This case highlights the importance of recognizing leptospirosis as a potential cause of myocarditis and thrombocytopenia, especially when clinical signs resemble those of STEMI. Early diagnosis and careful management, including the suspension of dual antiplatelet therapy and initiation of targeted antibiotic therapy, were pivotal in preventing further complications and improving the patient's outcomes.

Keywords: Leptospirosis, STEMI, myocarditis, anticoagulant, antiplatelet

Introduction

Leptospirosis is a well-recognized zoonotic disease caused by spirochetes belonging to the genus *Leptospira* [1]. This bacterial infection poses a significant public health burden, particularly in resource-limited populations [2]. Transmission to humans occurs through direct contact with the

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blood, tissues, or urine of infected animals, or indirectly via water or soil contaminated with the pathogen [3]. Rodents, particularly mouse-like species, serve as the primary reservoirs of infection [3]. The incubation period for leptospirosis typically ranges from 2 to 20 days [4]. The disease is predominantly reported in tropical regions, including East Sub-Saharan Africa, Southeast Asia, the Caribbean, and Oceania, which together account for approximately 73% of global cases [5]. The annual global mortality rate is estimated at 0.84 deaths per 100,000 individuals, with an incidence rate increasing during the rainy season [6].

Leptospirosis commonly follows a biphasic clinical course, characterized by an initial septicemic phase followed by an immune-mediated phase [7,8]. While the majority of cases are self-limiting, a subset of patients may develop severe complications, including jaundice, renal failure (commonly referred to as Weil's disease or icterohemorrhagic leptospirosis), pulmonary hemorrhage, and myocarditis [9]. Thrombocytopenia is frequently observed in leptospirosis and is associated with an increased risk of bleeding [10]. Despite these well-documented complications, the extent and mechanisms of cardiac involvement in leptospirosis remain inadequately understood.

ST-segment-elevation myocardial infarction (STEMI) is typically caused by the acute thrombotic occlusion of a coronary artery following the rupture of an atherosclerotic plaque [11]. This event triggers a cascade of platelet aggregation and activation of the coagulation pathways, resulting in myocardial ischemia and necrosis [11]. Leptospirosis, however, may mimic STEMI through several mechanisms, including microvascular endothelial injury, vasculitis, or direct myocardial inflammation [10,12]. Additionally, disseminated intravascular coagulation, a complication of severe leptospirosis, may lead to the formation of microthrombi, impairing both coronary and systemic circulation [10].

Although leptospirosis is a well-recognized zoonotic disease, the incidence of STEMI-mimicking leptospiral myocarditis accompanied by subsequent bleeding and thrombocytopenia is an exceptionally rare finding. The dual risks of bleeding and thrombosis further complicate the management of anticoagulation and thrombolytic therapy amidst competing risks, highlighting the critical need for prompt and accurate diagnosis to optimize patient outcomes and prevent potentially fatal complications. Therefore, the aim of this study was to present leptospirosis complicated by myocarditis, which mimicked STEMI, followed by bleeding and thrombocytopenia.

Case report

A 61-year-old male patient was referred from a primary healthcare facility to emergency department of Bethesda Hospital, Yogyakarta, Indonesia, with complaints of worsening chest pain, shortness of breath, and diaphoresis, which had begun 11 hours prior to hospital admission. The patient also reported nausea and vomiting earlier that morning. Twelve days prior to admission, the patient experienced a fluctuating fever, followed by episodes of diarrhea, which improved with oral paracetamol and loperamide. Additionally, the patient reported bilateral gastrocnemius pain. The patient denied any history of smoking, hypertension, or diabetes and there was no reported family history of coronary artery disease or other significant medical conditions. The patient denied any prior similar complaints or cardiac symptoms, including chest pain, palpitations, or shortness of breath. The patient worked as a farmer, and the rainy season was ongoing at the time.

The patient appeared lethargic, with vital signs indicating a blood pressure of 86/63 mmHg, a heart rate of 107 beats per minute, a respiratory rate of 22 breaths per minute, a body temperature of 39.8°C, and an oxygen saturation (SpO_2) of 95% on room air. Physical examination revealed anemic conjunctivae, icteric sclerae, and bilateral pulmonary crackles on auscultation. No cardiac murmurs or gallops were auscultated. The electrocardiogram (ECG) displayed global ST-segment elevation in all leads (**Figure 1**). Chest radiography showed increased bilateral pulmonary vascular markings.

Laboratory evaluation revealed significant hematological and biochemical abnormalities (**Table 1**). Initial hemoglobin levels were low (9.9 g/dL), indicating anemia, while the leukocyte count was markedly elevated (30×10^9/L), suggesting a severe infection. Thrombocytopenia was presented, with an initial platelet count of 131×10^9/L, which further decreased to 28×10^9/L by

day 2. Elevated blood urea nitrogen (71 mg/dL) and creatinine (2.3 mg/dL) levels indicated compromised renal function. Liver function tests showed elevated transaminases, with alanine aminotransferase (ALT) and aspartate transaminase (AST) levels of 93 U/L and 57 U/L, respectively, along with increased direct bilirubin (1.5 mg/dL) and indirect bilirubin (1.3 mg/dL). The high-sensitivity cardiac troponin (hs-cTn) assay revealed elevated levels, indicating myocardial injury. Urinalysis demonstrated proteinuria and electrolyte imbalances; hyponatremia (132 mEq/L), hypokalemia (3.1 mEq/L), and hypochloremia (80 mEq/L). Given the patient's clinical presentation and these laboratory findings, an IgM/IgG anti-leptospiral test was performed, yielding a positive result. A Faine's score of 32 further corroborated the diagnosis (**Table 2**).

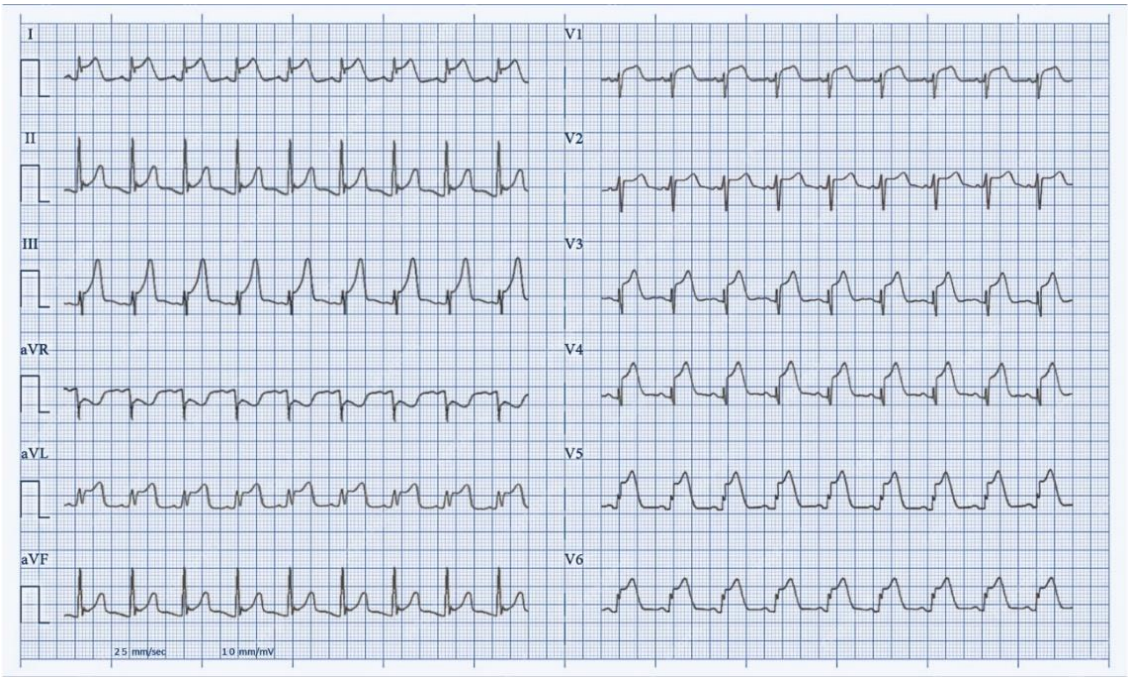

Figure 1. Electrocardiogram (ECG) examination with 12-lead showed sinus tachycardia and global ST elevation in the precordial and extremity leads.

Table 1. Laboratory results collected during hospitalization, detailing key diagnostic parameters and trends throughout the patient's stay

Test	Day 0	Day 2	Day 5	Day 11
Hemoglobin (Hb) (g/dL)	9.9	10.5	10.1	11.2
Leukocyte (×10⁹/L)	30	35	28	12
Thrombocyte (×10⁹/L)	131	28	42	107
Urea (mg/dL)	71	-	107	98
Creatinine (mg/dL)	2.3	-	3.1	1.6
Alanine aminotransferase (ALT) (U/L)	93	-	110	87
Aspartate transaminase (AST) (U/L)	57	-	131	90
Direct bilirubin (mg/dL)	-	1.5	-	-
Indirect bilirubin (mg/dL)	-	1.3		-
High-sensitivity cardiac troponin (hs-cTn) (ng/L)	93	135		-
IgM/IgG anti-leptospiral	Positive			
IgM/IgG anti-dengue	Negative			
Natrium (Na) (mEq/L)	132	-	140	-
Kalium (K) (mEq/L)	3.1	-	4.2	-
Chloride (Cl) (mEq/L)	80	-	89	-
Urinalysis	Proteinuria 1+			

On the second day of hospitalization, transthoracic echocardiography (TTE) revealed global hypokinesia and a reduced left ventricular ejection fraction (LVEF) by biplane of 48% (**Figure 2**). Mild left ventricular dilatation was observed, with an end-diastolic diameter of 5.6 cm. Right ventricular function was found to be normal. All cardiac valves appeared structurally and

functionally intact, with no evidence of regurgitation or stenosis. No thrombus or restrictive filling patterns were noted. The pericardium showed no signs of effusion (**Figure 2**).

Table 2. Patient's Faine's score assessment for leptospirosis diagnosis

Assessment	Score
Part A: Clinical data	
Headache	2
Fever*	2
If fever, temperature 39°C or more*	2
Conjunctival suffusion (bilateral)	4
Meningism	4
Muscle pain (especially calf muscle)*	4
Conjunctival suffusion + meningism + muscle pain	10
Jaundice	1
Albuminuria or nitrogen retention	2
Part B: Epidemiological factors	
Rainfall*	5
Contact with contaminated environment*	4
Animal contact	1
Part C: Bacteriological and laboratory findings	
Isolation of *Leptospira* on culture	Diagnosis certain
Enzyme-linked immunosorbent assay (ELISA) IgM positive*; slide agglutination test positive; microscopic agglutination test (MAT) single high titer	15
MAT rising titer (paired sera)	25

*Findings in the patient

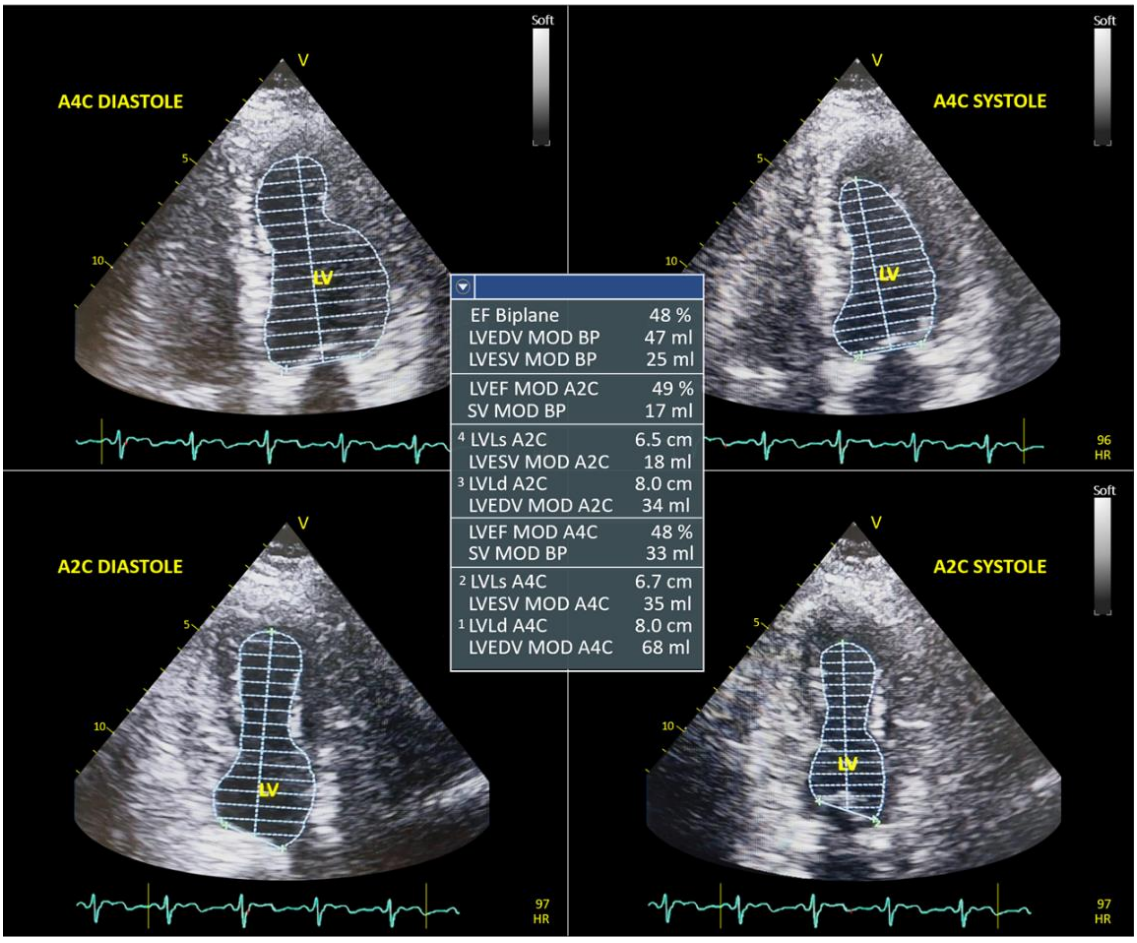

Figure 2. Transthoracic echocardiography (TTE) examination showed reduced left ventricular ejection fraction (LVEF) by biplane of 48%.

Upon admission to the emergency department, the patient was initially diagnosed with STEMI, with leptospirosis considered as a differential diagnosis. However, subsequent laboratory findings and TTE confirmed the diagnosis of Weil's disease complicated by cardiogenic shock secondary to leptospiral myocarditis.

Dual antiplatelet therapy, consisting of a loading dose of aspirin 320 mg and clopidogrel 300 mg orally, was administered in the emergency department. However, following subsequent laboratory results revealing severe thrombocytopenia (**Table 1**) and the patient showing gum bleeding and hematuria, the administration of dual antiplatelet therapy was discontinued on day 2 of hospitalization. Empiric antibiotic therapy with a dose of doxycycline 100 mg every 12 hours orally and ceftriaxone 2 g every 12 hours intravenously was initiated upon admission after the laboratory results confirmed a positive anti-leptospiral test. Supportive treatments included paracetamol 1 g every eight hours intravenously, methylprednisolone 20 mg every eight hours intravenously, esomeprazole 40 mg daily intravenously, oral ondansetron 4 mg every 12 hours, and oral potassium chloride 600 mg three times daily. The patient's condition gradually improved, and was discharged after a 15-day hospitalization. One week post-discharge, during a follow-up visit, the patient was in stable condition, with no recurrence of symptoms. Improvement in LVEF to 62% was observed, along with normal global left ventricular wall motion and cardiac chamber dimensions within normal limits.

Discussion

Leptospirosis is a zoonotic disease which can lead to systemic infection and multi-organ involvement. The bacteria enter the body through abrasions, open wounds, or mucous membranes [3,13]. Once inoculated, *Leptospira* enters the bloodstream and disseminates to various organs, including the liver, spleen, lungs, and kidneys, a stage known as leptospiremia [10,14]. Cardiovascular manifestations in leptospirosis result from endotoxins and the interaction between *Leptospira* and the host's immune system [15]. During leptospiremia, circulating *Leptospira* concentrations can be exceedingly high due to the failure of the human innate immune system, particularly pattern recognition receptors such as Toll-like receptor 4, to recognize *Leptospira* lipopolysaccharides [16]. Instead, Toll-like receptor 2 activates the immune response by triggering intracellular signaling pathways, leading to the activation of nuclear factor kappa B (NF-κB) and activator protein-1. These processes stimulate the secretion of prostaglandins, nitric oxide, and pro-inflammatory cytokines, including interleukin (IL)-6, IL-2, IL-1β, tumor necrosis factor alpha, and interferon gamma (**Figure 3**) [4,16].

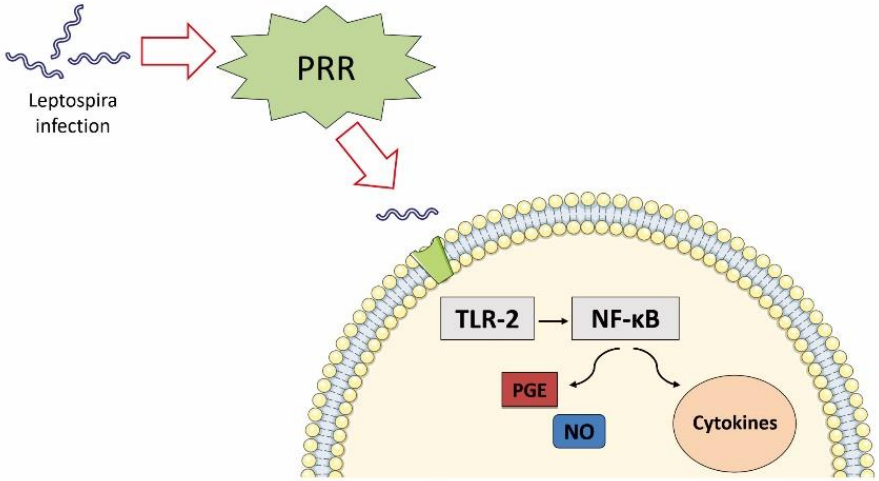

Figure 3. Immune response against *Leptospira* infection. NF-κB: nuclear factor kappa B; NO: nitric oxide; PGE: prostaglandin; PRR: pattern recognition receptor; TLR-2: toll-like receptor 2.

The systemic inflammatory response in leptospirosis can also affect the heart, leading to myocardial involvement. Leptospirosis-induced myocarditis often manifests during the immunogenic phase (around days 5 to 7) [7]. This phase is marked by hyperactivation of the immune system, leading to a sepsis-like syndrome and systemic vasculitis which can cause

myocardial inflammation [7]. Histopathological findings commonly reveal epicardial mononuclear infiltration and interstitial myocarditis, with some studies suggesting endothelial proliferation without atherosclerosis or infarction, indicating possible coronary artery endotheliitis [12,17]. These observations imply that the ST-elevation pattern seen in leptospirosis may not always signify myocardial infarction.

In this patient, the ECG revealed an ST-elevation pattern and elevated cardiac troponin levels, both characteristic of STEMI but also observed in myocarditis. However, there are notable differences that distinguish ST-elevation in STEMI and myocarditis. In STEMI, ST-elevation is typically localized to a specific coronary distribution, often accompanied by reciprocal changes, a tombstone or coved shape, and a sharp, significant rise in troponin levels [18,19]. In contrast, ST-elevations in myocarditis, including that induced by *Leptospira*, are generally diffuse and concave, without reciprocal changes, and troponin levels increase more gradually [18,19]. Other ECG changes observed in leptospirosis-induced myocarditis include bundle branch block, atrial fibrillation, various degrees of AV block, ventricular and supraventricular extrasystole, and low-voltage QRS complexes [20]. In this patient, the ECG demonstrated ST-segment-elevation without reciprocal changes, a pattern consistent with myocarditis. Therefore, no other arrhythmic abnormalities were detected.

Elevated troponin levels do not always indicate acute coronary syndrome, as these levels can also rise in conditions such as perimyocarditis and sepsis [21]. Sepsis can cause myocardial and microvascular dysfunction, leading to ST elevation and troponin release in the absence of significant coronary artery obstruction [21]. While this incidence is rare and the precise mechanism remains unclear, potential explanations include transient hypoperfusion, coronary vasospasm, and localized endothelial injury triggered by the systemic inflammatory response seen in sepsis [21,22].

Leptospirosis can lead to thrombocytopenia, as observed in this patient. Thrombocytopenia may occur during the early stages of dengue fever or as a late complication of severe leptospirosis [23]. However, a diagnosis of dengue was excluded in this case due to negative anti-dengue IgM/IgG test. The reduction in platelet count may be attributed to several mechanisms, including pre-adherence detachment, platelet disruption, and fragmentation mediated by leptospiral toxins, or the pathological processes associated with disseminated intravascular coagulation [10]. *Leptospira* bacteria replicate within macrophages and non-phagocytic cells, utilizing virulence factors that facilitate adhesion, endocytosis, and replication [12]. Extramembrane proteins such as OmpL1 and LigB play a critical role in this process, promoting vascular leakage by increasing endothelial permeability [12]. *Leptospira* adhesins bind to extracellular matrix components, including type IV collagen, laminin, plasminogen, fibronectin, collagen, fibrinogen, and elastin, thereby triggering the coagulation cascade; this cascade leads to the formation of a fibrin mesh and impaired coagulation, further contributing to thrombocytopenia [10,24].

This case report highlights the diagnostic and therapeutic challenges associated with leptospirosis complicated by cardiac involvement, specifically myocarditis mimicking STEMI. Initially, the patient was suspected of having STEMI, which necessitated consideration of antiplatelet or anticoagulant therapy and potential reperfusion strategies [25]. However, such approaches require caution due to the elevated risk of bleeding in leptospirosis. Alternative management strategies tailored to the patient's condition must be considered, particularly when the diagnosis may not be myocardial infarction, but rather myocarditis, which can present with similar symptoms and ECG findings. Careful interpretation of clinical findings, laboratory results, and imaging is crucial to differentiate myocarditis from STEMI, thereby preventing potentially harmful interventions, such as unnecessary dual antiplatelet therapy. Although dual antiplatelet therapy loading was initially administered based on the suspicion of STEMI, it was later discontinued due to the development of severe thrombocytopenia and minor bleeding manifestations. In this case, the initial ST-elevation on the ECG and clinical presentation strongly suggested acute coronary syndrome, which was STEMI, subsequently prompting the administration of antiplatelet therapy to prevent further thrombosis. The use of multimodal diagnostic tools—including clinical scoring (Faine's criteria), serological confirmation, and imaging modalities—is essential to distinguish between myocardial infarction and leptospiral myocarditis, which may present with similar ECG patterns. This case highlights the importance

of cautious diagnostic and therapeutic approaches, especially in endemic regions, to avoid misdiagnosis and inappropriate treatment.

In contrast to previous case reports [15,26], this case highlights the potential role of cytokine storm and endotheliitis in inducing platelet dysfunction, leading to thrombocytopenia and bleeding. These complications may have been exacerbated by the administration of thrombolytic or anticoagulant therapy, given the overlapping clinical manifestations with STEMI. Reperfusion therapy, including fibrinolysis, was initially considered but deferred due to the patient's clinical presentation of bleeding. Although the patient remained within the potential fibrinolysis window of 11 hours following the onset of chest pain, further investigations were necessary to confirm leptospirosis, as the clinical presentation strongly suggested the disease. While awaiting laboratory confirmation, fibrinolysis was withheld due to the significant bleeding risk associated with this intervention. Primary percutaneous coronary intervention (PPCI) was not performed due to financial constraints. Additionally, the rainy season and the patient's occupation as a farmer placed him at high risk of exposure to *Leptospira* through contact with contaminated soil or water. The combination of occupational and environmental exposures, clinical presentation, and laboratory findings was instrumental in establishing the diagnosis of leptospirosis.

TTE was employed to assist in diagnosis and monitor the patient's condition. Initially, it revealed a reduced LVEF, which subsequently normalized as the patient's condition improved following the resolution of the leptospirosis infection. This dynamic recovery emphasizes the importance of serial TTE assessments to evaluate cardiac improvement during the management of leptospiral myocarditis. TTE findings were consistent with previous case series [27] indicating that cardiac involvement in leptospirosis may result in transient electrocardiographic abnormalities, elevated Hs-cTn, and echocardiographic dysfunction. Compared with similar case reports [27], TTE in this case demonstrated global hypokinesia with reduced LVEF, followed by significant improvement in cardiac function after the initiation of targeted antibiotic therapy and supportive care. This finding emphasizes the potential for recovery in leptospiral myocarditis despite initial hemodynamic instability. Furthermore, the observed improvement aids in distinguishing leptospiral myocarditis from STEMI.

This case report has some limitations. Notably, the microscopic agglutination test (MAT), which is considered the gold standard for diagnosing leptospirosis [28], was not performed in this case. Instead, IgM/IgG anti-*Leptospira* test was utilized, which has moderate sensitivity and specificity compared to MAT [29]. Additionally, advanced imaging techniques such as cardiovascular magnetic resonance, the non-invasive gold standard for diagnosing myocarditis [30], were not conducted due to equipment availability and the relatively high cost associated with this method.

Although leptospiral myocarditis is rare, it should be considered in such clinical scenarios, as inappropriate thrombolysis can be fatal. Accurate diagnosis necessitates a comprehensive clinical evaluation and careful interpretation of the ECG. Although angiography was not performed in this case, the absence of localized ST-elevation, reciprocal changes, and the rapid rise and fall of troponin levels supported myocarditis as a more plausible diagnosis than acute coronary syndrome. Longitudinal studies exploring the long-term cardiac outcomes of patients with leptospiral myocarditis are critical for informing management strategies. Such research will enhance understanding of the pathophysiology of leptospirosis and improve patient care in endemic regions.

Conclusion

This study highlights the importance of recognizing leptospirosis as a potential cause of myocarditis and thrombocytopenia, especially when clinical signs mimic those of STEMI. The patient's condition presented unique challenges, including the dual risk of thrombosis and bleeding, which required early discontinuation of dual antiplatelet therapy and close monitoring for complications. The dynamic improvement in cardiac function, as evidenced by ECG findings, indicated the potential for recovery with targeted antibiotic therapy and supportive care. This case emphasizes the need for a comprehensive clinical evaluation to differentiate leptospirosis from other conditions with similar presentations, guiding appropriate therapeutic interventions.

Ethics approval

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

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None.

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

All data underlying the results are available as part of the article and no additional source data are required.

Declaration of artificial intelligence use

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

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