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Myocarditis with Brugada Pattern: An Unusual Early Manifestation of Leptospirosis

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ABSTRACT

Leptospirosis is a zoonotic disease caused by Leptospira spirochaetes. Cardiac involvement in leptospirosis is frequent yet underdiagnosed. We describe a myocarditis case showing Brugada pattern Type 1, a rare leptospirosis cardiac complication. As myocarditis is an atypical cardiac manifestation of leptospirosis, it is important to investigate anything sign related to cardiac involvement in managing leptospirosis.

INTISARI

Leptospirosis adalah penyakit zoonosis yang disebabkan oleh bakteri Leptospira spirochaetes. Keterlibatan jantung merupakan komplikasi yang sering terjadi pada leptospirosis, namun tidak banyak terdiagnosis. Kami melaporkan kasus miokarditis yang menunjukkan pola Brugada Tipe 1, sebuah komplikasi jantung pada leptospirosis yang jarang terjadi. Oleh karena miokarditis adalah salah satu manifestasi jantung atipikal dari leptospirosis, segala tanda yang berhubungan dengan keterlibatan jantung harus diselidiki dalam penatalaksanaan leptospirosis.

INTRODUCTION

Leptospirosis is an infectious disease caused by pathogenic spirochetes of the genus Leptospira[1]. It can affect both animals and humans. It is the world's most common zoonosis and is resurfacing globally [2]. Leptospirosis is primarily found in tropical areas. Banten had the highest case fatality rate (CFR) for leptospirosis cases in 2016, at 60 percent, followed by DI Yogyakarta at 35.29 percent, and Central Java at 18.29 percent [3].

There are two classifications of leptospirosis clinical manifestations. They are (i) classic icteric leptospirosis, combined with renal and liver failure known as Weils's disease, and (ii) anicteric leptospirosis with various unspecific presentations in many organs[1]. In cases of cardiac manifestations, leptospirosis can cause arrhythmias, pulmonary edema, refractory shock, chest pain in patient with severe disease[4]. Physician should consider a cardiac involvement if the patient complaints shortness of breath, chest pain, palpitations, with physical examination showed tachycardia, basal crackles, or hypotension[5]. In addition of ECG abnormalities such as arrhythmias, ST/T changes, conduction abnormalities, wall motion abnormalities on echocardiography[5].

Thus far, cardiovascular involvement manifested as ECG (electrocardiogram) or transthoracic echocardiography abnormalities, is common but underdiagnosed[1,2,4–7]. Autopsy studies have revealed significant cardiac involvement in fatal leptospirosis[6]. Data shows that these patients have severe cardiac dysfunction, even though myocarditis with Brugada pattern is uncommon[7]

CASE PRESENTATION

A 46-year-old Indonesian man was admitted to our hospital after experiencing syncope, chest pain, fever, arthralgia, and severe myalgia in the previous day. There is no history of sudden death <45 years-old in his family, or similar symptoms before. Physical examination revealed anicteric sclera, tenderness of the abdominal, thigh and calf muscles. Thorax and lungs were normal vesicular without crackles. The cardiovascular examination showed regular, rhythmic heart sounds, tachycardia, no murmurs.

Blood biochemistry was initially shown as Table 1. The electrocardiogram (ECG) revealed sinus tachycardia, with "coved" with the ST-segment elevation that concaves down and inverted T waves suggestive Brugada Type-1 pattern in V1-V3 (Figure 1). Cardiac biomarkers were increased. We used a rapid chromatographic immunoassay for the qualitative detection of IgG and IgM antibodies to Leptospira interrogans in human's whole blood. Positive Leptospira IgM confirmed the diagnosis of leptospirosis.

TABLES

Hospitalization day	1st	2nd	5th	6th	13th
Hemoglobin (mg/dL)	12.5	12	11.4	-	-
White Blood Cells Count (10^3/µL)	7630	10700	13870	-	-
Segment Neutrophil (%)	95	94	94	-	-
Lymphocytes (%)	5	3	3	-	-
Platelets (10^3/µL)	173	110	197	-	-
Urea (mg/ dL)	45	-	-	179.1	115
Creatinine (mg/dL)	1.2	-	-	11	2.1
Potassium (mEq/L)	4.2	-	-	-	4.4
AST (U/L)	-	28	-	121	-
ALT (U/L)	32	32	-	89	-
Troponin I (pg/mL)	+ (0.10)	-	-	-	-

Based on sign and symptoms, physical examination, and diagnostic testing we concluded the patient has acute leptospirosis, myocarditis related to leptospirosis, and acute kidney injury. Treatment was started with doxycycline 2 x 100 mg orally, supportive care of ceftriaxone 2 x 1 g intravenously. In support of evaluation the myocarditis, patient had daily routine electrocardiography and trans thoracal echocardiography. The ECG showed no malignant arrhythmia during hospitalization. The echocardiography showed normal left ventricular function with no regional wall motion abnormalities

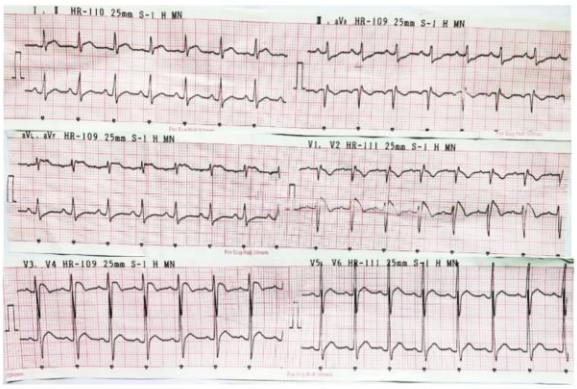

Figure 1. ECG on the admission in emergency room: sinus tachycardia, "coved" shaped ST elevation followed by T inversion in V1-V3 suggestive Brugada Type 1 Pattern

Significant improvements were seen after 13 days of treatment, showed by normal sinus rhythm and better kidney function biochemistry result. On the 14th day of hospitalization, the patient was discharged.

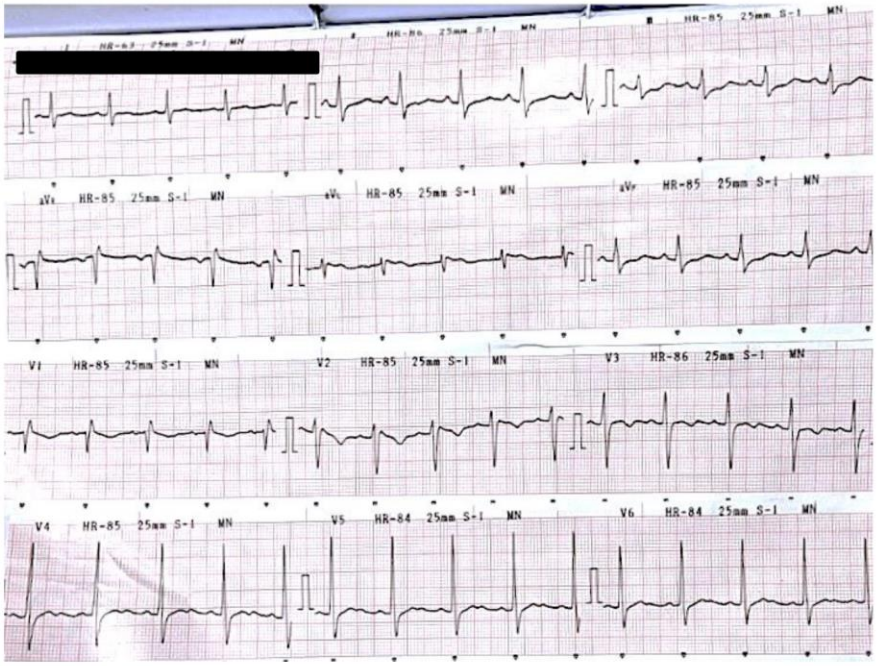

Figure 2. ECG on the 13th day of hospitalization: normal sinus rhythm without ST elevation

DISCUSSION

Early phase is leptospiremia or septicemic phase when non-specific acute febrile illness occurring for three to nine days. The following phase is immune phase, where IgM antibodies detected in the blood. It's settled in higher concentrations in the organs, therefore serious manifestations occur during this phase[5] .

We assumed our patient was in immune phase leptospirosis, shown by the presents of kidney injury, IgM reactive antibody, and myocarditis. Chest pain, tachycardia, "coved" shaped (ST segment elevation followed by T

inversion) in ECG suggestive of type 1 Brugada Pattern, and elevated cardiac markers were indicating the myocarditis. Myocarditis usually grows on the fifth to seventh day of leptospiral infection[6]. Because of normal ECG after complete treatment achieved and there was no sign of ventricular arrhythmia during hospital stay, we didn't treat the patient as Brugada syndrome. However, as the present of syncope history, further electrophysiology study may be necessary.

We consider that the transient Brugada pattern in the patient ECG, may be elicited by the febrile state. A febrile person (T>38o C) is 20 times more common to have Type I Brugada than the afebrile[8], known as fever induced Brugada syndrome. Nonetheless, as the complete treatment accomplished associated with the ECG changes, the mechanism of leptospirosis related myocarditis is inescapable. A pathological study from 24 postmortem hearts of leptospirosis patients showed myocarditis (96%) and half of the cases (50%) have endocardial inflammation[9]. Increasing level of troponin indicated cardiac injury which led to peri-myocarditis or cardiac hypoxemia.

The hallmarks of acute myocarditis are sign of injury and death of cardiomyocytes and immune cells infiltration in the tissue10. It is not described clearly yet, however TLR (Toll Like Receptor) maybe involved in the mechanism[7]. Infiltration of lymphocytes and plasma cells, mononuclear infiltration, and petechial hemorrhage especially in epicardium, or coronary arteritis was presented in leptospirosis[11].

Electrolyte abnormalities are likely to contribute to electrocardiographic changes. Renal tubular electrolyte transport mechanisms were disturbed during leptospirosis infection[4]. Data suggest that an outer membrane protein of Leptospira inhibits the Na+ - K+ -Clcotransporter activity in the thick ascending limb of Henle, resulting hypokalemia and sodium wasting[12].

CONCLUSION

We describe a rare and unusual early leptospirosis complication. The ECG abnormalities are possibly caused by the direct effect of leptospirosis or non-specific result of a febrile infection or metabolic and electrolyte abnormalities. Cardiac manifestation such as myocarditis should be investigated in treating patient with leptospirosis. As shown in this case, it is important to bear in mind that transient Brugada pattern ECG changes during fever could trigger syncope.

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Disclosures and Ethics

Conflicts of interest: the authors have no relevant financial or non-financial interests to disclose.

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