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Leptospira Myocarditis mimicking ST-elevation Myocardial Infarction - a case report.

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Abstract:
Wide variability in the presentation of leptospirosis often makes it difficult to diagnose, especially in an emergency setup when EKG findings mimic STEMI. We describe a 55-year-old patient, who presented to the emergency room with complaints of typical compressing substernal chest pain for 4 hours duration. He is a chronic smoker and alcoholic with no co-morbidities. Upon general examination, the patient was febrile, not icteric with pulse= 120/minute, BP= 100/70 mm Hg. Other observations were normal. EKG showed significant ST elevations in leads V1- V6, I, aVL. Echocardiography findings showed global hypokinesia with EF: 38%. We suspected extensive anterior wall myocardial infarction and as a general rule in non-PCI-capable hospitals, thrombolysis was done. Meanwhile, routine blood investigations showed leukocytosis, increased total bilirubin (2.8 mg/dl), and platelet count of 90000/mm3. These lab findings along with persistent ST elevation in EKG made us suspect leptospira myocarditis. A positive Leptospiral IgM and Microscopic Agglutination test (MAT) confirmed the diagnosis. After starting penicillin injection, the patient's condition started to improve. The ejection fraction returned to 52% on day 5. Myocarditis in leptospirosis can lead to fatal cardiac dysfunction. Moreover, thrombolysis can be fatal in some patients. In conclusion, we can say that an early diagnosis and treatment of leptospira myocarditis is crucial for the prognosis, and a high index of clinical suspicion is needed to diagnose leptospira myocarditis.

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I. Introduction

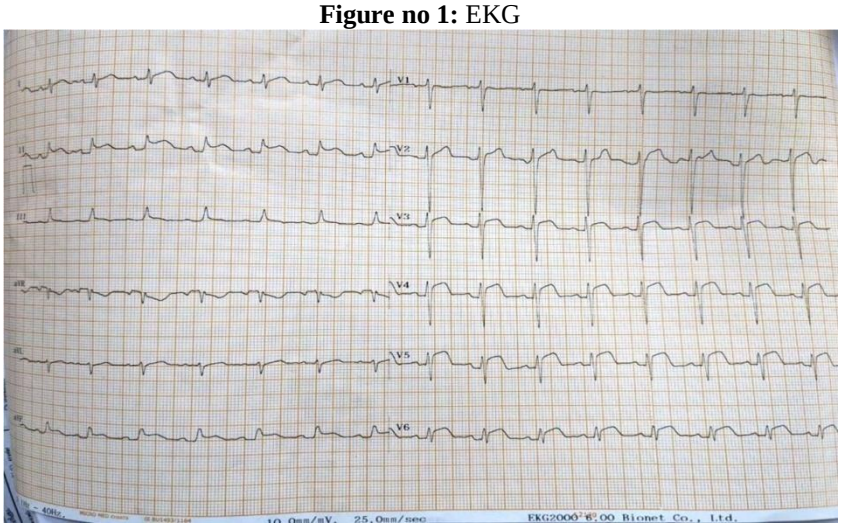
In clinical practice, we see a lot of patients coming to the emergency department with typical chest pain. EKG would show ST elevation, suggestive of an acute coronary syndrome. Depending on the window period, affordability, and other factors, the patient would undergo a reperfusion procedure in the form of PCI/ thrombolysis. But at times, diagnosing a case as acute coronary syndrome just based on EKG findings might turn out to be wrong. Here is one such case. Leptospirosis can present as myocarditis mimicking ST-elevation myocardial infarction. Leptospirosis shows a broad spectrum of clinical manifestations making it difficult to diagnose, especially the anicteric form. The frequency and extent of cardiac involvement in leptospirosis are under-reported and poorly understood; the majority of the cases are asymptomatic[15]. A high index of clinical suspicion is needed to diagnose leptospira myocarditis.

II. Case Report

A 55-year-old patient presented to the emergency room with complaints of typical compressing substernal chest pain for 4 hours duration. The patient had no co-morbidities. The patient was a chronic smoker, alcoholic and was working as a manual laborer at a construction site. Upon general examination, the patient was febrile, not icteric, pulse= 120/minute, BP= 100/70 mm Hg. Respiratory and per abdominal examination were unremarkable.

EKG showed significant ST elevation in leads V1-V6, I, aVL.

Figure no 1: EKG



A provisional diagnosis of extensive anterior wall myocardial infarction was made and a loading dose of anti-platelets was given. The patient was then transferred to an Intensive Coronary Care Unit.

Echocardiography findings:

- global hypokinesia of Left ventricle with Ejection fraction 38%
- normal chambers
- no pericardial effusion
- descending aorta normal

After getting informed consent and ruling out absolute contraindications, the patient underwent intravenous thrombolysis with streptokinase. Meanwhile, routine blood investigations showed leukocytosis, increased total bilirubin (2.8 mg/dl), and platelet count of 90000/mm3. The findings are listed below:

Table no 1: Complete blood count

Hemoglobin	12.3 g/dl
RBC	4.1 million/mm3
HCT	40%
MCV	85 fL
MCHC	34.4 g/dl
WBC	12,000 cells/mm3
Neutrophils	91%
Lymphocytes	5.7%
Platelet count	90,000 cells/mm3

Table no 2: Renal function test

Random blood sugar	124 mg/dl
Urea	64 mg/dl
Creatinine	1.6 mg/dl
Sodium	132 mEq/L
Potassium	3.5 mEq/L

Table no 3: Liver function test

Serum bilirubin Direct Indirect	2.6 mg/dl 1.6 mg/dl 1.0 mg/dl
SGOT	147 U/L

SGPT	88 U/L
Serum ALP	73 U/L
Total protein	5.5 g/dl
Albumin	3.0 g/dl
Globulin	2.5 g/dl

Table no 4: Other investigations

PT INR	11 seconds, 0.94
IgM dengue	Negative

Post Streptokinase EKG showed persistent ST elevation, tachycardia, and the patient was still febrile and complaining of chest pain. When asked in detail about the fever, the patient told he has been having a fever for the last one week with myalgia. This history along with EKG findings and echocardiography showing global hypokinesia (and not an artery territory) prompted us to think of other causes of ST elevation including myocarditis and pericarditis. Serial LFTs showed rising bilirubin and increasing total count. Leptospirosis IgM, Microscopic Agglutination test (MAT), and dengue IgM were sent.

Leptospirosis IgM was strongly positive followed by a positive Microscopic Agglutination test (MAT). The patient was diagnosed to have leptospira myocarditis based on Modified Faine's criteria and was started with injection crystalline penicillin. The patient's total bilirubin and total count started to decrease and he was symptomatically better. Repeat echocardiogram on day 5 of admission showed Ejection fraction=52%. The patient was referred to our parent hospital for a coronary angiogram which showed normal coronaries.

III. Discussion

Leptospirosis is a zoonosis of ubiquitous distribution, caused by infection with pathogenic Leptospira species[1]. Frequently exposure occurs from contact with contaminated water sources. Occupational exposure, recreational activities, and weather events like floods are important risk factors [2].

Leptospirosis is a biphasic illness characterized by an early septicaemic phase lasting about a week and a delayed immune phase [2], [3].The majority of the patients develop complications during the immune phase, particularly the second week. It is very important to monitor the patients during the immune phase because they can decompensate after the febrile phase and even after receiving specific antibiotics; also, these two stages get confused and many times become one when patients are predisposed to get complicated [4]. Symptoms usually begin several days to several weeks after exposure and range from a mild febrile illness to overt Weil's syndrome with azotemia, jaundice, confusion, anemia, thrombocytopenia, and high temperatures [5]. The disease is frequently overlooked especially the anicteric form. However, an early diagnosis and treatment are crucial for the prognosis of the disease. Jaundice and renal failure ("Weil's disease"), pulmonary hemorrhage, acute respiratory distress syndrome (ARDS), uveitis, optic neuritis, peripheral neuropathy, myocarditis, and rhabdomyolysis are well-known complications [6].

Cardiac manifestations range from non-specific electrocardiographic changes and arrhythmias to myocarditis, pericarditis, endocarditis, and cardiogenic shock [7]. Myocarditis usually occurs by the end of the first week. The Commonest ECG changes are conduction defects followed by ST/T wave changes and atrial arrhythmias [8].

But the mechanism behind it is not well understood and the magnitude of the problem is under-reported. It is probably secondary to toxin-mediated vascular damage, leading to intimal layer, perivascular, and subendocardial inflammation [10]. The severity of leptospirosis could be related to the intensity of the immune response [9].

Given the increased prevalence of coronary artery disease and lifestyle habits, most often we label patients with primary ST-T changes as myocardial infarction. In an emergency setup, differentiating myocardial infarction from myocarditis/pericarditis based on the morphology of the ST segment is often difficult.

The European Society of Cardiology working group on myocardial and pericardial diseases has developed clinical and diagnostic criteria, when present myocarditis should be suspected [12]. However, a definitive diagnosis of myocarditis ideally should be established by myocardial biopsy. This is not practical in most settings. Due to wide variability in presentation and non-specific clinical findings, many cases of myocarditis are likely to go undetected [13].

Here comes the role of good history taking. History taking, although underrated, is still the most powerful tool of a physician. In this case, the patient had a fever with constitutional symptoms which evolved into anginal chest pain. Echocardiogram findings of global hypokinesia rather than regional wall abnormality always suggest a non-vascular cause.

Regarding treatment, studies show there are potential benefits in early initiation of appropriate antimicrobials and early supportive treatment such as VA-ECMO support, including consideration of high-dose pulse steroids where appropriate (especially in the immune phase) [14].

Extensive research on leptospirosis should be carried out to aid us in attaining a more holistic understanding of the complexity between Leptospira and humans and for a better design of improvised therapeutic regimens in the future.

Leptospira myocarditis is very rare but should always be thought of in a clinical scenario like this. Thrombolysis can be fatal. Therefore, thorough clinical assessment and EKG analysis is vital before thrombolysis in non-PCI-capable centers. Accurate diagnosis is life-saving in such conditions.

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