Acute ST elevation myocardial infarction (STEMI) in a patient with leptospirosis; a therapeutical dilemma – A case report

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Abstract
A 45-year-old previously healthy farmer was seen at the Emergency Department with ischemic type chest pain. He had a history of an acute febrile illness for four days. The ECG showed an acute anterior myocardial infarction. His laboratory investigations revealed thrombocytopenia, a raised serum creatinine level and significantly raised high-sensitive cardiac Troponin I. A provisional diagnosis of acute STEMI with co-existing leptospirosis was made. The patient was managed conservatively with antibiotics, statins, and aspirin. Neither thrombolysis nor primary coronary intervention was attempted due to high risk of bleeding. Diagnosis of leptospirosis was confirmed later by markedly elevated leptospira antibodies. After recovery, he underwent coronary angiogram which revealed a totally occluded left anterior descending artery and is awaiting myocardial viability assessment with dobutamine stress echocardiography before PCI. We report this uncommon presentation of acute myocardial infarction in a patient with leptospirosis, as it causes a considerable therapeutic dilemma especially in a resource-poor setting.

Key words: leptospirosis, myocardial Infarction, ST- segment elevation

Introduction
Leptospirosis is one of the most widespread zoonotic diseases in the world caused by the pathogenic spirochetes of genus Leptospira. The disease is commonly found in Sri Lanka and other tropical countries.¹ Leptospirosis has a wide spectrum of clinical manifestations ranging from a self-limiting febrile illness to a severe fatal illness with multiorgan dysfunction. It causes a considerable degree of morbidity and mortality especially in resource-poor settings. The disease is known to affect many organ systems causing varying degrees of organ dysfunction and fatality.² Cardiac involvement in leptospirosis is an important complication that is underreported, poorly understood, and often predicts a poor outcome. The spectrum of cardiac involvement ranges from a variety of electrocardiographic (ECG) and echocardiographic changes to severe myocarditis causing overt heart failure.³ Several cases of leptospirosis with myocarditis mimicking ST-elevation myocardial infarction have been reported.⁴ Here we report a rare case of a patient who presented with ST-elevation myocardial infarction coexisting with leptospirosis which poses considerable challenges in diagnosis and treatment.

Case presentation
A 45-year-old, previously healthy man was transferred from a local hospital with a history of central chest pain for 4 hours duration. It was a tightening, non-radiating, retrosternal pain associated with sweating, nausea, and vomiting. He had a history of fever with chills and rigors, arthralgia, myalgia, abdominal pain, and loose stools for four days. His urine output was low, and urine was red in colour. He is a farmer who had worked in the paddy field the week before the onset of illness and had not been on prophylactic medication for leptospirosis. There were no significant cardiovascular risk factors identified.

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On examination, the patient was febrile with a temperature of 38.8°C and anicteric. He was hemodynamically stable with a blood pressure of 120/85 mmHg and a pulse rate of 96 bpm. The oxygen saturation was 96% on air. There was significant muscle tenderness over calves and thighs bilaterally. The rest of the physical examination was unremarkable.

On admission to the emergency department, his ECG showed significant ST elevations of 3mm on V1-V4 leads, and mild ST elevations on leads V5 and V6. With a provisional diagnosis of leptospirosis complicated with acute myocarditis or acute myocardial infarction, intravenous benzathine penicillin 2 MU six hourly was commenced, and oral doxycycline was added later. Intravenous morphine was given for pain relief.

Laboratory investigations revealed a white cell count of 3.8x10^9, platelet count of 99x10^9 and C-Reactive protein (CRP) levels of 237 mg/dL (< 5mg/dL). Liver transaminases levels were marginally elevated with SGPT was 61 U/L and SGOT was 78.5 U/L. Serum creatinine was 3.47mg/dL (0.7 -1.3mg/d) serum sodium was 140.6 mmol/L and serum potassium was 4.3 mmol/L. Dengue serological tests were negative. Chest X ray showed clear lung fields.

Dynamic changes were detected on serial ECGs. The initial high sensitivity Troponin I titer was 169.9ng/L (normal < 14ng) and it elevated to 40,000ng/L in six hours. An urgent bedside 2D echocardiogram revealed a LVEF of 40% with septal hypokinesia. Acute anterior ST elevation myocardial infarction coexisting with acute leptospirosis was made based on these findings. Myocarditis is thought to be less likely in the absence of significant haemodynamic instability and global hypokinesia in echocardiography.

Thrombolysis was not carried out due to the presence of thrombocytopenia and ongoing sepsis. Atorvastatin 40mg noxte, bisoprolol 2.5mg mane, aspirin 75mg noxte and subcutaneous enoxaparin 60mg daily were started following consultation with the cardiologist. Throughout the hospital stay, the patient remained haemodynamically stable with an adequate urine output, despite acute kidney injury (AKI) with increasing serum creatinine levels. AKI was managed conservatively. Considering the ongoing acute kidney injury and sepsis, primary percutaneous coronary intervention (PCI) was not carried out urgently.

Subcutaneous enoxaparin was continued for 5 days in conjunction with antibiotics and medicines mentioned above. The patient's condition improved gradually with marked improvements noted in his investigations and with defervescence.

Screening for cardiovascular risk factors revealed fasting blood glucose 95mg/dL, total cholesterol 190mg/ dL, LDL100mg/dL and serum triglycerides of 135mg/dL A detailed 2D echocardiogram revealed anterior left ventricular wall and septal hypokinesia with moderately impaired LV systolic function of 45%. Leptospira microscopic agglutination test (MAT) done on the sixth day of the illness reported a titer of ≥1: 2560 confirming the diagnosis.

The patient was subsequently discharged home with aspirin 75mg noxte, bisoprolol 2.5mg daily, atorvastatin 40mg noxte and a short course of oral doxycycline after a total stay of 6 days in hospital. He was referred to a cardiology center for a coronary angiogram and percutaneous intervention (PCI). One month later he underwent a coronary angiogram and was found to have a totally occluded mid left anterior descending artery (LAD) and a PCI was planned after testing for myocardial viability.

Discussion

Leptospirosis is a highly prevalent zoonotic disease in the tropics caused by the pathogenic spirochetes of Genus *Leptospira*. Clinical manifestations of leptospirosis are diverse, and most patients have an uncomplicated febrile illness. About 10% of the cases develop severe leptospirosis with unusual manifestations and multiorgan involvement, which is often fatal.

Cardiac involvement usually occurs in severe leptospirosis and clinically manifests with chest pain, palpitations, and hemodynamic instability. ECG abnormalities occur early in the course of the disease and atrial fibrillation, atrioventricular conduction blocks, and non-specific ventricular repolarization changes are commonly found. ECG changes in leptospirosis can be due to direct myocardial involvement or deranged homeostasis. However, left ventricular function assessed by echocardiography is usually normal. Autopsy studies have reported that interstitial myocarditis and vasculitis as the more predominant lesions.

Several case reports have been published where patients with leptospirosis developed ST elevation in ECG mimicking acute myocardial infarction. However, these ECG changes rapidly normalized and were believed to be due to myocarditis or severe leptospirosis causing vasculitis. Our patient had acute myocardial infarction in LAD territory (demonstrated by coronary angiography-CA) with coexisting severe leptospirosis with acute kidney injury and liver involvement. He might have had an underlying...
asymptomatic coronary artery disease although no cardiac risk factors were reported. The increased cardiac workload, coronary hypoperfusion and oxidative stress during the acute infection would have triggered plaque rupture leading to acute MI. Myocarditis secondary to leptospirosis was considered in the differential diagnosis. Cardiac magnetic resonance scan (Cardiac MRI) or endomyocardial biopsy is needed for definitive diagnosis of myocarditis. Both investigations are not readily available in Sri Lanka. However, considering ischemic type chest pain, ST segment elevation in ECG, significantly raised troponin titer, regional wall motion abnormality (antero-septal) in echocardiography, we adopted a tentative diagnosis as acute MI. Later CA revealed the occluded vascular territory corresponding to the ST segment elevation in ECG.

We faced several therapeutic dilemmas in the management of this patient. Firstly, the administration of empiric loading doses of antiplatelets for a patient with thrombocytopenia and acute febrile infection in regions where leptospirosis and dengue fever are prevalent is a challenging decision. Due to the increased risk of bleeding, we decided not to administer the loading doses of aspirin and clopidogrel. High doses of statins were prescribed. Secondly, decision has to be made whether to perform thrombolysis in the background of thrombocytopenia and acute febrile illness. Considering the potential risk of pulmonary hemorrhage, thrombolysis was not carried out. Although there are multiple case series and studies on performing PCI in patients with thrombocytopenia and malignancies, no case studies or case reports could be located to justify the benefit of PCI for acute MI, in the setting of ongoing sepsis and thrombocytopenia. Primary PCI was not attempted taking the ongoing acute kidney impairment and sepsis into account. Furthermore, PCI facilities are not readily available in Sri Lanka where resources are minimal. The other thought-provoking consideration was the usage of dual antiplatelets in the setting of acute MI, thrombocytopenia, and sepsis. In literature, aspirin has been administered to patients with cancer and thrombocytopenia without any complications but there are no reports on the benefits or possible adverse effects of aspirin in the presence of acute sepsis with thrombocytopenia. However, after excluding the possibility of dengue (clinically and serologically), we decided to start the patient on aspirin as a single antiplatelet agent. Clopidogrel was not considered due to the higher possibility of bleeding.

Conclusions

Leptospirosis with cardiac involvement presents many diagnostic and therapeutic dilemmas to the internist. ST segment elevation in the ECG in acute leptospirosis is rare but represents serious underlying pathology, either myocarditis or MI, causing significant diagnostic and therapeutic difficulties in clinical practice. Careful interpretation of clinical and biochemical picture of the patient, coupled with advanced imaging studies are necessary for optimal care of the patient.

Author declaration

Consent to participate

Written and informed consent was taken from the patient for publication.

Competing interests

No competing interest.

Author contributions

All authors involved in the management of the patient. GP and PR wrote the case description. YUK researched and wrote the introduction, discussion and corrected the final draft. NM contributed to the discussion. All authors read and approved the final manuscript.

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