Cardiogenic shock with severe myocardial injury: A rare presentation of Leptospirosis

¹Perera IA, ¹Lakshman P, ²Kumanan T, ²Suganthan N

¹Teaching Hospital Jaffna, Sri Lanka, ² Faculty of Medicine, Jaffna

Abstract

Leptospirosis is a common zoonotic disease of the tropics, caused by pathogenic spirochetes of the genus Leptospira. Though infection tends to be mild and self limiting in most cases, it is known to cause life threatening complications such as acute kidney injury, pulmonary haemorrhages and acute respiratory distress syndrome, myocarditis and rhabdomyolysis. Severe myocarditis leading to cardiogenic shock with circulatory collapse in leptospirosis is a rare clinical entity. Here we describe a young healthy male, who presented with extensive myocardial injury with cardiogenic shock, and made an uneventful recovery with timely intervention and care.

Key words

Cardiogenic shock, myocardial injury, Leptospirosis, Zoonosis, Acute kidney injury

Background

Leptospirosis, is a globally widespread zoonotic disease, encountered both in the tropics as well as in the temperate climates, however the incidence being significantly higher in the tropics. Cardiac involvement in leptospirosis is also recognized, though not frequently reported. The usual clinical evidence of myocardial injury is in the form electrocardiographic abnormalities, reduced cardiac ejection fraction in 2D echocardiogram and raised cardiac enzymes. The development of arrhythmias, repolarization abnormalities on electrocardiogram are considered to carry a poor prognosis.

Case presentation

A 23-year-old, previously healthy male, was admitted to the emergency treatment unit of the tertiary care hospital in Northern Sri Lanka, with a history of fever for 7 days, associated with constitutional symptoms such as myalgia, arthralgia and loss of appetite. He had abnormal behavior in the form of irrelevant speech, excessive somnolence and fluctuating level of mental state during the last 6 hours and it was associated with reduced urine output. He had no history suggestive of either respiratory or urinary tract infection and there was no history of photophobia and symptoms suggestive of focal neurological deficits or fits. There was no history of alcohol ingestion or withdrawal.

On examination, he was found to be conscious, but confused. He was febrile (temperature -38.6° C), diaphoretic and icteric. Cardiovascular system examination showed a pulse rate of 140 beats/min, regular and a blood pressure of 70/40 mmHg. His lungs had bi-basal fine inspiratory crackles with few wheezes. He had no signs of focal neurological deficit nor neck stiffness. Rest of the clinical examination was unremarkable including the optic fundi.

Initial resuscitation with fluid boluses of intravenous 0.9% saline was carried out, but as the blood pressure failed to rise beyond 80mmHg systolic, inotropes were initiated. Blood pressure could be brought up to 116/80 mmHg after optimal doses of inotropes. Subsequently, a central venous catheter was placed in-situ.

Furthermore, history of an exposure to river water during an excursion, 10 days prior to the febrile illness was mentioned by the family members. Clinically a diagnosis of Leptospirosis was entertained and he was started on intravenous crystalline penicillin 2MU 6 hourly. In addition, a three days course of intravenous methylprednisolone (500mg daily) was initiated considering the severity of the illness and followed by an oral course.

An ECG showed sinus tachycardia with diffuse ST segment depression, and a 2D echocardiogram confirmed a globally dilated, hypokinetic heart with a Left ventricular EF of 25%, compatible with

Corresponding Author: Suganthan N. Email: drn.suganthan@yahoo.com bttps://orcid.org/0000-0001-7905-6709 Submitted April 2020, Accepted June 2020



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severe myocarditis. Troponin I was significantly elevated (29ng/ml).

After initial stabilization, he developed worsening of shortness of breath, with type 1 respiratory failure on the same day evening, requiring invasive ventilation for a period of 48 hours. A chest radiograph revealed bilateral diffuse alveolar shadow compatible with severe pulmonary edema.

He developed acute kidney injury and it was managed with continuous renal replacement therapy initially, and thereafter with intermittent hemodialysis for which he responded favorably. The serum transaminases, which were raised up to five-fold of the upper limit of normal on presentation, normalized by day 16, along with the moderate thrombocytopenia. An echocardiogram performed just prior to discharge showed an improving Left ventricular EF of 50%. Leptospira antigen was positive on day 8 of the illness. On a subsequent follow up visit at a month, the findings on assessment by clinical examination and the laboratory investigations revealed no abnormalities.

Discussion

Leptospirosis, a widesparead zoonotic disease, continues to be a major health burden and a diagnostic challenge to the clinician, due to its non specific and variable presentations. Weil's disease, the severe form of this infectious disease, is often associated with multi-organ involvement and carries a mortality rate of approximately 5-15%(1).

Well known life-threatening complications are pulmonary haemorrhages with acute respiratory distress syndrome, jaundice, renal injury, myocarditis, meningitis and uveitis. The most lethal of all is pulmonary haemorrhage, which has been described extensively in medical literature (2). Though cardiac involvement is a frequent accompaniment of severe leptospirosis, severe degree of myocarditis and left ventricular dysfunction is rare, and if occurs, often tends to be fatal (3). Cardiac injury in leptospirosis, which carries a significant risk of mortality, may present in the form of chest pain, arrhythmias, pulmonary edema or refractory cardiogenic shock. A range of electrocardiographic changes are known to depict myocardial injury in the form of atrial fibrillation, Atrio ventricular conduction blocks, and nonspecific ventricular repolarization abnormalities (4). Cardiovascular collapse can develop rapidly secondary to myocarditis, and without prompt circulatory support can be fatal (5).

Conclusion

Cardiogenic shock and multi-organ dysfunction often ensue in majority of systemic infection leading to severe sepsis. Though myocarditis is listed as one of the complications of leptospirosis, extensive myocardial injury leading to cardiogenic shock as a presenting clinical entity is rarely encountered in clinical practice and should be considered as an important differential diagnosis in a patient presenting with febrile illness and shock.

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