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# Case Report

# Leptospirosis presenting with myopericarditis and cardiogenic shock- a case report

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## Introduction

Leptospirosis is a zoonotic infection endemic in tropical countries. Infection results from exposure to infected reservoir animals who release pathogenic leptospirae in urine. Even though a wide variety of domestic and wild species serve as reservoir hosts, the brown rat (*Rattus norvegicus*) is the most significant source of human infection. A majority of patients present with non-specific symptoms, including fever, headache and myalgia, which can progress to multi-organ failure involving the kidneys, brain, heart and lungs. Haemorrhagic complications are commonly reported, associated with coagulation abnormalities [1].

We report a case of an elderly male presenting with leptospirosis complicated with multi-organ failure, from Sri Lanka.

## Case presentation

A 65-year-old, previously healthy farmer presented with sudden onset pleuritic chest pain lasting for 3 hours which was relieved when seated and bending forwards. He had associated sweating, vomiting and faintishness. He complained of fever of 4 days duration, along with arthralgia and myalgia. He had mild shortness of breath and his urine output was 0.3 ml/kg/hr. He denied any respiratory, urinary or gastro-intestinal symptoms.

On examination, he was febrile and icteric. He was not pale. Glasgow Coma Scale was 15/15. His peripheries were cold. Pulse was weak and thready with a rate of 54 bpm. Blood pressure was 70/50mmHg. Saturation on air was 98%. Respiratory rate was 24 breaths per minute and auscultation of lung bases revealed bi-basal fine end-inspiratory crepitations. Abdomen was soft.

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Since there was exposure to leptospirosis, he was managed as for leptospirosis with cardiac involvement with cardiogenic shock and acute kidney injury. He was started on intra-venous [IV] ceftriaxone 2g daily along with IV noradrenaline 0.4micrograms/kg/min. He was given IV methylprednisolone 1mg/kg, which was continued for 3 days.

Initial electro-cardiogram (ECG) revealed sinus bradycardia. Full blood count showed a white cell count of 8.2 x $10^9$/L (neutrophils- 84.8%, lymphocytes- 9.8%), haemoglobin of 13.6g/dL and a platelet count of 42 x$10^9$/L. Blood picture was reported as severe thrombocytopenia due to sepsis with liver pathology. C-reactive protein was 227.8mg/L. Erythrocyte sedimentation rate was 24mm/hr. High sensitivity troponin I was 2984.6ng/L. Liver function tests revealed a alanine transaminase of 53.5U/L, aspartate transaminase of 91.9U/L, total protein of 5.8g/dL, total bilirubin of 59.07micromol/L, direct bilirubin of 45.98micromol/L, gamma-glutamyl transferase 0f 79.4U/L, albumin of 3.2 g/dL and globulin of 2.6 mg/L. Serum creatinine was 242 µmol/L. Serum sodium was 136mmol/L and serum potassium was 3.8mmol/L. Urine full report revealed 10-12/ hpf pus cells and 140 to 150/hpf red cells. Ultra-sound scan of kidneys, ureters and bladder revealed bilateral acute kidney injury. Chest X-ray postero-anterior was normal. 2 D echocardiogram revealed mild global hypokinesia with an ejection fraction 50–55% suggestive of myocarditis. NS1- antigen and dengue IgM and IgG serology were negative. Leptospira IgM antibody performed on day 6 of illness came as positive. IgG antibody was negative.

He continued to have fever spikes despite being on IV antibiotics. Two days later, he developed confusion and agitation. Electroencephalogram was suggestive of encephalitis. His confusion persisted for two days and gradually settled thereafter. ECG taken on day two revealed widespread saddle shaped ST segment elevation in the infero-lateral leads along with PR segment depression suggestive of pericarditis (Fig. 1). Fever spikes gradually settled and on day 8 following admission he was asymptomatic. IV noradrenaline was tailed off. On examination pulse rate was 78bpm, blood pressure was 130/80mmHg without IV noradrenaline and lungs were clear. Repeat ECG was normal. IV ceftriaxone was continued and full blood count, renal function tests and C-reactive protein normalized by day 10.

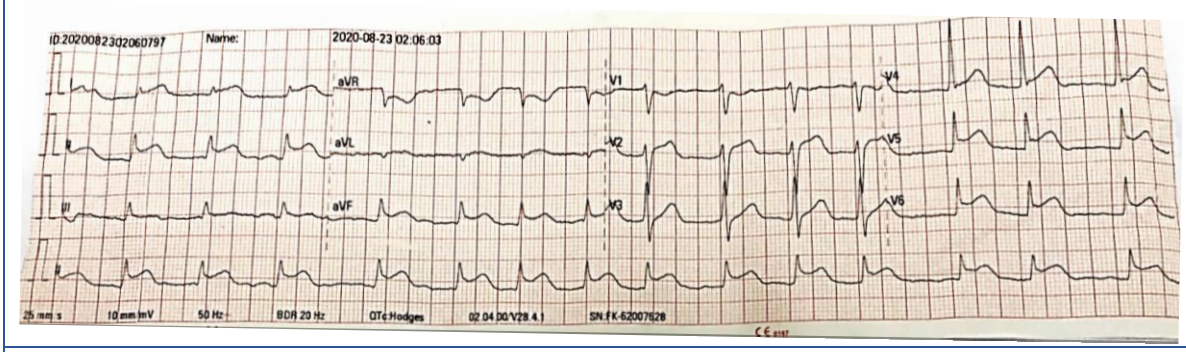


Figure 1: ECG on day 2 following admission

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## Discussion

Leptospirosis is a zoonotic disease caused by a spirochaete of genus Leptospira. This is also known as Weil's disease, canicola fever, water-borne fever, rice field fever, mud fever and swamp fever. Common farm animals that transmit the disease include pigs, cattle and horses. Individuals residing in slum environments with poor housing and inadequate sanitation are at high risk of exposure. Majority of human leptospiral infections are mild or asymptomatic, especially in endemic areas. Outcome of disease depends on host factors, epidemiological conditions and virulence of pathogen [1].

Leptospirosis usually occurs during the rainy season in the tropics and in early fall and late summer in temperate countries. Incidence is 10 times more in the tropics than in temperate climates. The World Health Organization reports that there are 873,000 cases annually with over 40,000 deaths [2]. Organisms shed in infected animal's urine survive in fresh water for 16 days and in soil for 24 days. The leptospira invade mucous membranes and intact skin following contact with contaminated soil or water [1].

Severe leptospirosis can cause multi-organ failure including kidneys, liver, heart and lungs. Cardiac manifestations of leptospirosis include non-specific electrocardiographic changes, arrhythmias, myocarditis, pericarditis, endocarditis, acute coronary arteritis, aortitis and cardiogenic shock [3]. The incidence and exact pathophysiology of cardiac manifestations of leptospirosis is unknown. A study conducted in India reported that 56% of patients with leptospirosis had cardiac involvement and 52% of them had ECG changes [4]. A retrospective analysis of autopsied cases of leptospirosis revealed that 41 of 44 cases had cardiovascular involvement of which myocarditis was found histologically in 100% of cases, endocardial involvement in 39%, valvular in 36% of cases, coronary artery disease in 51% and aortic involvement in 56% [5].

According to the European Society of Cardiology guidelines on pericardial diseases, the diagnosis of pericarditis requires the presence of at least two of four criteria which include typical chest pain, pericardial friction rub, suggestive ECG changes and new or worsening pericardial effusion [6]. Our patient had typical chest pain and ECG changes diagnostic of pericarditis.

According to recent literature, shock in severe leptospirosis is caused by dysregulation of inflammatory mechanisms leading to sepsis and systemic inflammatory response syndrome [7]. Therefore, pure cardiac contribution to shock is difficult to diagnose. Hence, it is important to assess the type of shock by history, examination and investigation findings. Our patient had cold peripheries with a weak thready pulse favouring cardiogenic shock.

There are several case reports of leptospirosis complicated by myocarditis and pericarditis but cases of myopericarditis and cardiogenic shock are rare in the available literature. Cavalcanti S, L Lerena V and Gomez C et al. in 2018, reported a case of leptospirosis with myopericarditis from Peru [8]. Two similar cases of myopericarditis

were reported in 1996, in France [9,10]. No specific therapies are available to treat cardiac complications of leptospirosis. Supportive measures along with correction of coagulopathy is included in current management. Pericarditis should be managed with non-steroidal anti-inflammatory drugs, colchicine and low dose corticosteroids [11].

Even though myopericarditis is a rare complication of leptospirosis, it should be considered in a patient presenting with fever and chest pain. Early clinical suspicion and appropriate therapy minimize complications.

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