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Case Report

Severe leptospirosis complicated with atrial fibrillation

[1]Sarannija E, [1]De Abrew S.T.N, [1]Amarasekara AADS

[1]North Colombo Teaching Hospital, Sri Lanka.

Abstract

Cardiac rhythm abnormality is a common finding in patients with leptospirosis. Relative bradycardia, atrial fibrillation, atrial flutter and ventricular premature beats are the common arrhythmias seen in leptospirosis. In the present case, the patient presented with septic shock and had atrial fibrillation. Further evaluation revealed hypokalemic acute kidney injury and severe metabolic acidosis. The atrial fibrillation reverted to sinus rhythm after correction of hypokalemia, acidosis, septic shock and with medical cardioversion. This case revealed that early detection of rhythm abnormalities and other forms of cardiac involvement and correction of precipitating factors is important in preventing fatal outcomes in leptospirosis

Key words

Leptospirosis, Relative bradycardia, atrial fibrillation, atrial flutter

Introduction

Most infections of leptospirosis are asymptomatic or mildly symptomatic. However, a small number of cases can develop the severe form of illness with multi organ failure. The presence of cardiac involvement by performing a 2DEcho or clinically tends to predict a poor outcome in leptospirosis. (1,2)

Case report

A previously healthy 45yrs old male farmer admitted with fever of one week. The fever was high grade and remittent, which responded to paracetamol. It was associated with chills, rigors, arthralgia and myalgia. He also had dry cough from the onset of fever. Two days prior to the admission he observed reduction in urine output despite adequate intake. He also experienced mild shortness of breath. There was no history of dysuria, hematuria or frothy urine. His latest muddy water exposure was one week prior to the onset of symptoms. Before one hour of admission he developed palpitations and increased severity of shortness of breath which brought him to the hospital.

On admission, the patient was febrile, dyspneic and unwell. He had conjunctival suffusion and was icteric. He was conscious and rational. His room air oxygen saturation was 93% and pulse rate was 140bpm which was irregularly irregular. The blood pressure was 80/50 mmhg. His abdominal and neurology examinations were normal and there was bilateral bibasal fine occasional crepitations in the lung fields.

A clinical diagnosis of leptospirosis with septic shock was made. The blood pressure picked up to 110/70 mmhg after 1500ml of crystalloid boluses. Patient was given intravenous ceftriaxone after taking blood and urine cultures. An urgent ECG showed an atrial fibrillation with the rate of 146 bpm. The ABG revealed severe metabolic acidosis with high lactate levels. The ABG potassium level was 2.74 mmol/l. The potassium level was rapidly corrected with 30mmol IV potassium chloride over 30 minutes. After the potassium replacement the ABG potassium level was 3.2mmol/l. The heart rate dropped to 120bpm after the correction of potassium and shock but the rhythm remained as atrial fibrillation. The patient was given loading dose of intravenous amiodarone and started on maintenance dose with careful monitoring of vitals.

His initial investigations are given below (table1.1).

Table 1.1: Initial investigations of the patient

Complete blood count	
White blood cells (*103/µL)	13.8
Neutrophils %	74.5
Lymphocytes %	18.4
Haemoglobin (g/dl)	13
Platelets (*103/µL)	11
ABG	
pH	7.2
HCO 3- (mmol/l)	16.7
Potassium (mmol/l)	2.6

Corresponding Author: E Sarannija, Email: sarannija1990@hotmail.com https://orcid.org/0000-0001-5495-6487, Submitted November 2021, Accepted December 2021

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lactate (mmol/l)	6
Serum potassium (mmol/l)	2.6
Serum sodium (mmol/l)	142
Serum calcium (mmol/l)	2.2
Serum magnesium (mmol/l)	0.9
Serum phosphate (mmol/l)	1.5
Serum creatinine (µmol/l)	677
Blood urea (mg/ dl)	261
Liver function test	
AST (U/L)	26
ALT (U/L)	19
Total bilirubin (µmol/ L)	227
Direct bilirubin (µmol/ L)	221
CRP (mg/l)	342
CPK (U/L)	96
LDH (U/L)	490
Troponin I	negative
UFR	pus cells – 8, red cells – 10, protein +
Blood and urine cultures	no growth
Coagulation profile	normal

The chest x-ray on admission revealed no evidence of pulmonary haemorrhages. The bedside echocardiogram did not reveal any pericardial effusion or left ventricular dysfunction. The bedside ultrasound scan abdomen revealed mild hepatomegaly with acute renal parenchymal changes. Urgent nephrology opinion was taken for oliguric acute kidney injury with metabolic acidosis. The patient was offered an urgent haemodialysis and transferred to ICU.

The maintenance dose of amiodarone was continued for 24 hrs and then changed to oral amiodarone. The oral amiodarone slowly tailed off and omitted over the next four days. The potassium levels were closely monitored and corrected during the ICU stay. He returned to sinus rhythm after 36 hours of admission and did not develop any further episodes of atrial fibrillation.

A proper 2D echocardiography revealed ejection fraction of 60% without any evidence of myocarditis. The Lepto MAT which was sent on admission was positive with the titre of >1:320.

His urine output, creatinine levels and bilirubin levels slowly improved. He was transferred to medical ward after 1 week of ICU stay and discharged after 10 days of admission. A review was planned after 5 days of discharge with complete blood count, creatinine, bilirubin levels and ECG.

Discussion

Cardiac injury in leptospirosis results from toxic damage associated with endotoxins, immunoallergic phenomena or direct damage by Leptospira. Disseminated intravascular coagulation and electrolyte imbalances also play an important role in leptospirosis-associated cardiac involvement. (3)

In our case the patient developed atrial fibrillation without any evidence of myocarditis. Hypokalemia, acidosis and septic shock were thought to be the contributary factors for the development of atrial fibrillation.

Electrical cardioversion is the ideal management option for atrial fibrillation in a haemodynamically unstable patient. As our patient was in septic shock, we immediately restored the blood pressure with crystalloid boluses. We rapidly corrected the hypokalemia which was a major contributing factor to the arrhythmia. With the correction of the septic shock and hypokalemia the heart rate improved but the rhythm was not restored to sinus rhythm. Then we decided for medical cardioversion with amiodarone. The acidosis also corrected with haemodialysis. The atrial fibrillation in our patient reverted to sinus rhythm with careful monitoring of electrolytes, fluid management, correction of metabolic acidosis and with medical cardioversion.(4,5)

Conclusion

Cardiac involvement in leptospirosis is associated with poor outcome. Close monitoring and early detection of rhythm abnormalities and other forms of cardiac involvement and correction of precipitating factors is important in preventing fatal outcomes.

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