Case Report

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Atrial flutter complicating severe leptospirosis: a case report

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ABSTRACT

Cardiac disturbances are relatively common and electrocardiographic abnormalities may be found in more than 70% of patients with leptospirosis. We report the case of a 68 year-old male with severe leptospirosis who developed atrial flutter. Effective treatment was done with amiodarone. The patient became clinical stable, with complete recovery. Rigorous clinical observation and continuous electrocardiogram (ECG) monitoring may facilitate the identification of rhythm disorders, and thus prevent a probable fatal outcome, in severe cases of leptospirosis.

Keywords: Leptospirosis. Weil syndrome. Arrhythmia. Atrial flutter. Electrocardiogram.

INTRODUCTION

Leptospirosis is a zoonotic disease of global distribution associated with a diverse clinical spectrum. It is caused by species of *Leptospira,* and transmitted to humans by direct contact with infected tissues or contaminated urine, or indirectly by contact with contaminated water or soil. Mammalian reservoirs are the main distributors, and although rodents are the most important reservoir, other animals, including livestock, wild and domestic animals may excrete leptospires in their urine and, therefore, be a potential source of infection[1-3]. Leptospirosis is associated with occupational and recreational exposure, usually occurring as endemic in rural areas with outbreaks associated with floods[1,3].

Clinical spectrum is broad in leptospirosis. Multiple clinical presentations may occur and a wide variety of diseases may mimic different stages of leptospirosis[1]. Most patients will present a febrile self-limited condition, often misdiagnosed as *influenza* or *dengue*[1,2,4]. Severe manifestations occur in approximately 5-10% of human infections and are characterized by the presence of jaundice, acute renal failure and hemorrhagic disorders, including severe pulmonary hemorrhage syndrome[1,2,5]. In severe forms the biphasic presentation becomes more evident. The first phase is commonly referred as septicemic; it lasts for 5 to 7 days and high fever, myalgia, headache, conjunctival suffusion, abdominal pain and vomiting are usually seen during this period. A brief afebrile period may follow this phase just before the immune phase of the disease begins. The latter is characterized by multiorgan involvement and a higher mortality rate[2,3,5,6].

Less common presentations may occur in a significant number of cases and lead to misdiagnosis. Pulmonary involvement may range from clinically undetectable to massive hemoptysis and respiratory failure, with severe pulmonary forms present in up to 74% of lethal cases, representing the main cause of death in Brazil[2,5]. Neurological manifestations such as symptomatic aseptic meningitis, myeloradiculopathy and myelopathy may be found in case-reports throughout the literature[4]. Pancreatitits is a rare complication, however asymptomatic hyperamylasemia is often present[2,4]. Cardiac disturbances are quite common and electrocardiographic abnormalities may be found in more than 70% of patients; nonetheless cardiac life-threatening conditions are unusual[7,8]. In recent study in Sri Lanka, during an outbreak in 2008, myocarditis was reported in 7.1% of cases and heart failure in 3.9%[9].

The objective of this article is to report a case of severe leptospirosis complicated by the occurrence of atrial flutter, an uncommon arrhythmia in the course of the disease.

CASE REPORT

A 68 year-old male agriculturist was brought by family members to the emergency department with a history of high fever (39°C, 40°C) that had started 10 days earlier. Two days after the initial symptom, the patient presented with the onset of diffuse myalgia, which was worst on abdomen and inferior limbs, shortness of breath, oliguria and bilateral conjuctival hemorrhage. Family members noted a deteriorating clinical condition and brought the patient, on the fifth day of the disease, to a previous hospital, where the patient was initially diagnosed with severe *Dengue.* Over the following days, there was persistence of the fever, onset of jaundice and continuous deterioration of the patient's condition, being than transferred to this hospital. His past medical history was unremarkable except

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for transurethral resection e subtotal prostatectomy for benign prostatic hypertrophy four years ago.

Upon admission the patient presented with a reduced consciousness, with a Glascow Coma Scale of 13 (verbal response: 6; ocular response: 3; motor response: 4), febrile to the touch, severe conjunctival hemorrhage in both eyes, taquicardic but with normal cardiac auscultation, breath sounds were unremarkable, liver and spleen were not palpable at the abdomen examination and bowel sound were present, severe tenderness of abdominal, thigh and calf muscles, painful even to superficial palpation and bilateral and symmetrical edema of inferior limbs.

His initial blood work, related to the eighth day of the disease, showed a hemoglobin of 13.2mg/dL, a white blood cell count of 12,900/mm^3, with 67% of neutrophils and 23% of lymphocytes, platelets of 42,500/mm^3, a creatinin level of 6.0mg/dL, an aspartate aminotransferase of 80U/L, an alanine aminotransferase of 76U/L, with 2.0g/dL of albumin and creatine phosphokinase (CPK) of 89 U/L. Total bilirubin was 7.3mg/dL with a direct bilirubin of 6.82mg/dL and amylase was 221U/L. Kidney function continued to decrease over the next days, with blood urea level of 214mg/dL, creatinine of 7.2mg/dL, a metabolic acidosis (pH of 7.26, pCO_2 of 31 and HCO_3^- of 14.6) and hyperkalemia of 6.0mEq/L.

Upon admission to this hospital, intravenous ceftriaxone as initiated along with supportive measures, including vigorous hydration, to which there was rapid response with diuresis. On the tenth day of the disease a central venous catheter was obtained for emergency hemodialysis. The day after the patient's pulse rate increased to 180/min and an electrocardiogram revealed an *atrial flutter*, with abnormal F waves **(Figure 1)**, a 2:1 atrium-ventricular block and an atrial frequence of 360/min. The patient showed no signs of hemodynamic instability; his blood pressure maintained at a baseline level of 110 over 90mmHg, there was no worsening of his consciousness level, no acute onset of dyspnea or precordial pain. An attack dose of amiodarone was then administered and reverted the *atrial flutter* to a sinus tachycardia with a pulse rate of 120/min. The patient was transferred to an intensive care unit bed for further monitoring.

He remained in the intensive care unit (ICU) for four days. During that period he remained performing daily hemodialysis, he showed no newer arrhythmias or hemodynamic instability and his consciousness level improved. Bilirubin and CPK levels increased initially as well as his white blood count (WBC) count.

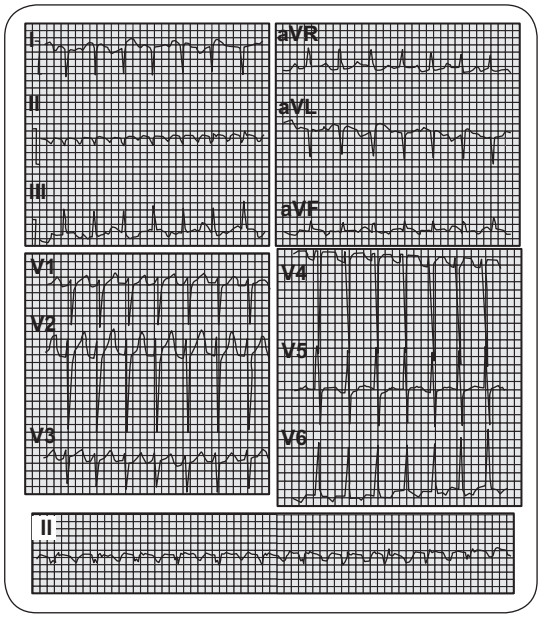

FIGURE 1 - Abnormal F waves with a 2:1 atrium-ventricular block and an atrial frequence of 360/min.

TABLE 1 - Laboratorial tests during hospital stay

Days of disease	8th Day	9th Day	11th Day	12th Day	13th Day	16th Day	17th Day
Hemoglobin (mg/dL)	13.5	12.8	12.8	12.2	10.3	8.27	7.96
White Blood Cells Count (/mm^3)	12,900	21,600	13,700	14,400	12,500	9,610	8,240
Neutrophil (%)	67.0	94.0	85.0	84.0	83.0	78.9	70.0
Lymphocytes (%)	23.0	3.2	13.0	0.7	6.5	4.1	13.3
Platelets (/mm^3)	42,000	59,000	123,000	145,000	161,000	504,000	652,000
Urea (mg/dL)	-	214	235	206	130	87	56
Creatinin (mg/dL)	6.0	7.4	5.3	5.0	3.3	-	1.4
Potassium (mEq/L)	-	6.0	5.2	4.7		-	3.5
AST (IU/L)	76	19	17	30	39	75	-
ALT (IU/L)	80	33	38	60	66	64	-
Total Bilirubin (mg/dL)	-	7.23	11.37	13.41	15.3	3.82	-
Direct Bilirubin (mg/dL)	-	6.82	7.59	8.98	9.77	3.77	-
CPK (IU/L)	-	89	377	636	486	-	62
Amylase (IU/L)	-	221	250	261	362	-	-

AST: aspartate aminotransferase; ALT: alanine aminotransferase; CPK: creatine phosphokinase.

On the sixteenth day of the disease he was readmitted to the infirmary with significant improvement of the mylagia, disappearing of conjunctival hemorrhage, normal ECG **(Figure 2)** and an improved kidney function (**Table 1).** His treatment was concluded after twenty six days of disease and the patient was discharged from the hospital.

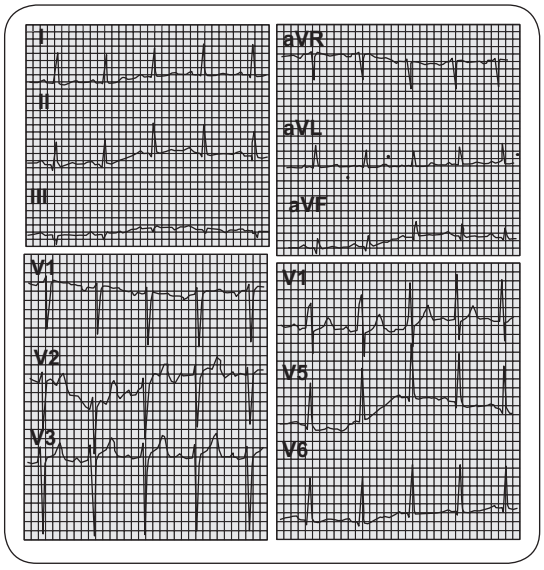

FIGURE 2 - Normal electrocardiogram.

DISCUSSION

Cardiac involvement is usually subclinical, in milder forms, or masked by pulmonary hemorrhage, in severe leptospirosis[8]. Most of the reports have been clinical and tachycardia, electrocardiogram (ECG) abnormalities and refractory hypotension are the usual clinical manifestations[1,10-12]. Pericardial effusion has been reported in patients with leptospirosis and advanced renal failure, suggesting a possible role for uremia in such patients[12].

The mechanism of cardiac involvement is poorly elucidated but may, as for kidney damage, result from toxic mechanisms associated with endotoxins, immunoallergic phenomena or direct damage by leptospira[1]. Disseminated intravascular coagulation can also play an important role in leptospirosis-associated cardiac involvement[9]. Autopsy studies have shown a high prevalence of cardiac involvement in severe leptospirosis, with up to 96% of patients dying from such disease[8,11]. The autopsy findings are many; cardiomegaly, pericardic hemorrhage, perivascular inflammation, endocarditis and intimitis of coronary arteries and of the aorta were among the features of cardiac damage[8,11]. The predominant histophatological finding was an interstitial myocarditis, present in up to 96% of the studies mentioned and highly associated with inflammation of the conduction system[8,11].

Electrocardiographic abnormalities may be found in numerous patients undergoing hospitalization for leptospirosis treatment. Some series report these changes in 48% to 80% of patients[7,8,10]. These results are probably overestimated since most of the series analyze only hospitalized patients and, therefore, more likely to have severe leptospirosis, a clinical scenario where cardiac involvement becomes more evident[4,7]. Changes in ECG are the most commonly described cardiovascular abnormality in leptospirosis, but they may have low specificity for myocardial involvement and can reflect electrolyte abnormalities[9]. ECG changes includes sinus tachycardia, relative and marked bradycardia, bundle branch blocks, changes in the P-QRS-T complexes, low voltage QRS complexes, ST-T wave disturbances, intraventricular conduction disturbances, non-specific ventricular repolarization disturbances, ventricular and supraventricular extrasystoles, first-degree and third-degree heart block and atrial fibrillation[9]. In a study with 157 patients with serologic proven leptospirosis performed at Salvador, Bahia, Brazil, 107 exhibited ECG abnormalities. Alteration of ventricular repolarization and disorders of condution (including bundle branch block and atrioventricular block) were the commonest disturbances found, followed by atrial fibriliation in 17 patients and extrasystoles in 9[7].

Atrial flutter is a rare arrhythmia in the course of the leptospirosis. Patient's clinical condition was not significantly altered by the presence of such condition, however more persistent and refractory arrhythmias may lead to severe hemodynamic instability. Atrial fibrillation and first degree atrioventricular block are the most common arrhythmias found [7,10,12].

Cardiac involvement is a factor associated with a poor prognosis and, therefore, a higher mortality rate[1,9]. Rigorous clinical observation and continuous ECG monitoring may facilitate the identification of rhythm disorders, and thus prevent escalation to a fatal outcome.

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