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## Letter to the Editor

# Arrhythmias in leptospirosis-associated acute kidney injury: a case series



Dear Editor,

Leptospirosis is a zoonotic disease of global importance and a major public health problem, which is transmitted to humans by contact with mammals' contaminated urine, most often rats.<sup>1</sup> Its severe form manifests as jaundice, acute kidney injury (AKI), and hemorrhage, particularly in the lungs.<sup>2</sup> Arrhythmias are the most important cardiac manifestations of this disease, and they have been described as risk factors for death.<sup>3</sup> Herein we present clinical, demographic, and laboratory characteristics from a series of patients with leptospirosis admitted to a tertiary hospital in Fortaleza, Ceará, Brazil, from January 1985 to December 2015 who presented AKI and electrocardiographic (ECG) abnormalities. Among 488 leptospirosis patients, 24 (4.91%) presented arrhythmias, 23 acute atrial fibrillations and one atrial flutter. Most of them were young females (17–70.8%). All patients presented severe AKI, 16 (66.7%) needed hemodialysis, and nine patients (37.5%) died. They evidenced thrombocytopenia and high levels of AST, ALT and bilirubin, as summarized in [Table 1](#). The patients were dehydrated on admission, due to vomiting, diarrhea, low fluid intake, polyuria, and excessive transpiration. In addition, hypokalemia, hypocalcemia and metabolic acidosis were noticed. These electrolyte abnormalities may have been a key point in the onset of arrhythmias, since they have been related to ECG abnormalities in leptospirosis, even when there is no myocardial dysfunction.<sup>4</sup> Even though serum magnesium values were not available in patients' charts, we believe that hypomagnesaemia may have also contributed to arrhythmias, as previously reported.<sup>5</sup> In summary, dehydration and electrolyte disturbances were crucial to trigger arrhythmias in patients with leptospirosis. Therefore, these factors must be avoided or treated in order to prevent such life-threatening complications.

**Table 1 – Demographic, clinical and laboratory data of leptospirosis patients with arrhythmias (n = 24).**

	Mean ± SD/median/ number (%)	Range
<b>Demographic and clinical data</b>		
Age (years)	46.2 ± 13.4	(11–69)
Time onset of symptoms to admission (days)	7.1 ± 1.7	(5–10)
Hospitalization time (days)	10.6 ± 6.2	(1–24)
<b>Gender</b>		
Males	7 (29.2%)	
Females	17 (70.8%)	
Hemodialysis	16 (66.7%)	
Death	9 (37.5%)	
<b>Laboratory data</b>		
Hemoglobin (g/dL)	10.1 ± 1.6	6.5–12.3
Hematocrit (%)	31.3 ± 4.8	22–38
Leukocytes (10 <sup>3</sup> /mm <sup>3</sup> )	16.83 ± 10.07	4.5–52.6
Platelets (10 <sup>3</sup> /mm <sup>3</sup> )	58.56 ± 43.58	7.0–145.0
Urea (mg/dL)	204.7 ± 59.7	108–347
Creatinine (mg/dL)	6.9 ± 2.3	3.4–13.3
Sodium (mEq/L)	132.0 ± 6.0	124–144
Potassium (mEq/L)	3.2 ± 0.68	2.3–5.0
Calcium (mEq/L)	7.5 ± 2.4	6.4–11.0
AST (U/L)	115.7 ± 69.1	27–240
ALT (U/L)	64.8 ± 30.0	12–130
Direct bilirubin (mg/dL)	16.0 ± 7.9	4.3–31.0
LDH (U/L)	714.2 ± 214.4	380–1044
CPK (U/L)	451.5 ± 552.8	27–1609
pH	7.36 ± 0.06	7.25–7.48
HCO <sub>3</sub> (mEq/L)	18.6 ± 4.6	12.8–28.9

AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CPK, creatine phosphokinase; HCO<sub>3</sub>, serum bicarbonate.

Variables were expressed as mean ± standard deviation.

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## Conflicts of interest

The authors declare no conflicts of interest.

## REFERENCES

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1. Charan J, Saxena D, Mulla S, Yadav P. Antibiotics for the treatment of leptospirosis. Systematic review and meta-analysis of controlled trials. *Int J Prev Med.* 2013;4:501–10.
2. Daher EF, Soares DS, de Menezes Fernandes ATB, et al. Risk factors for intensive care unit admission in patients with severe leptospirosis: a comparative study according to patients' severity. *BMC Infect Dis.* 2016;16:1–7.
3. Dupont H, Dupont-Perdrizet D, Perie JL, Zehner-Hansen S, Jarrige B, Daijardin JB. Leptospirosis: prognostic factors associated with mortality. *Clin Infect Dis.* 1999;25:720–4.
4. Navinan MR, Rajapakse S. Cardiac involvement in leptospirosis. *Trans R Soc Trop Med Hygiene.* 2012;106:515–20.
5. Khositseth S, Sudjaritjan N, Tananchai P, Ong-ajyuth S, Sitprija V, Thongboonkerd V. Renal magnesium wasting and tubular dysfunction in leptospirosis. *Nephrol Dial Transplant.* 2008;23:952–8.

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