

Atrial flutter as a rare manifestation of leptospirosis

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DESCRIPTION

A 50-year-old healthy man presented with fever and chills after returning from Vietnam. While running around an urban water reservoir, he fell and sustained abrasions to his knee and elbow. He cleaned his injuries with dew and reservoir water. Approximately 14 days later, he began experiencing fever, chills, headache, dizziness and severe body aches. After two syncopal episodes, he presented to an urgent care clinic which promptly referred him to our clinic. Physical examination revealed a temperature of 39.1°C, bradycardia, an irregular rhythm and right upper quadrant tenderness. Laboratory results included a low white cell count of $2.7 \times 10^9/L$ (normal value $3.7\text{--}10.5 \times 10^9/L$), a low platelet count of $121 \times 10^9/L$ (normal value $150\text{--}400 \times 10^9/L$), high aspartate aminotransferase 313 U/L (10–40), high alanine aminotransferase 223 U/L (10–41), high alkaline phosphatase 172 U/L (40–129), and normal bilirubin and creatinine. An abdominal ultrasound revealed an edematous gallbladder wall without evidence of cholelithiasis. ECG revealed new onset atrial flutter and variable atrioventricular nodal conduction with heart rate ranging from 35 to 55/min (figure 1). Leptospirosis IgM antibody by dot blot and chikungunya, rickettsia and Lyme disease serologies were negative. A *Leptospira* microscopic agglutination test (MAT) from the Centers for Disease Control and Prevention was also negative at less than 1:100. The patient was started on doxycycline empirically, given a high suspicion for leptospirosis. He underwent successful cardioversion. His symptoms continued to improve with doxycycline and he completed a 10-day course. A follow-up leptospirosis IgM titre obtained 2 weeks later was positive. Repeat MAT was positive at 1:3200 (32-fold increase). During a 16-month follow-up visit, the patient was asymptomatic and he was in sinus rhythm.

Leptospirosis is a widespread zoonotic disease caused by pathogens of the genus *Leptospira*. More than 1 million cases are estimated to occur

Learning points

- ▶ Leptospirosis, which is caused by pathogens of the genus *Leptospira*, can range in severity from a mild febrile illness to severe, life-threatening disease.
- ▶ Leptospirosis rarely causes cardiac manifestations including various arrhythmias, myocarditis, pericarditis and endocarditis.
- ▶ Paired acute and convalescent antibodies should be obtained to diagnose leptospirosis.

worldwide each year.¹ Transmission occurs seasonally, most commonly during the rainy season, via direct contact with urine, blood or tissue of infected animals or via contact with contaminated freshwater.² Infection may range from asymptomatic to life-threatening. Symptoms typically arise 7–12 days following exposure but can develop from 3 days to 1 month after exposure.³ While a non-specific febrile illness is the most common manifestation, a small percentage of patients develops severe disease involving multiple organ systems. Although rare, leptospirosis can also affect the heart. Cardiac manifestations range from non-specific electrocardiographic changes and arrhythmias to myocarditis, pericarditis, endocarditis and cardiogenic shock.^{4–6} The electrocardiographic changes generally resolve completely after successful treatment and clinical recovery.⁷ Early diagnosis requires a high degree of suspicion as symptoms can be non-specific. A leptospirosis diagnosis guideline has not been published. Nevertheless, this infection is generally diagnosed by isolating *Leptospira* spp. from a normally sterile site or documenting a fourfold increase in the MAT titre from the acute-phase to the convalescent-phase serum sample or a single MAT titre of $\geq 1:400$.⁸ Since the MAT is not readily available, alternative rapid tests are often used including IgM ELISA or molecular tests such as PCR. While serology is the most common initial method for diagnosing leptospirosis, the usefulness of serologic testing for the diagnosis of acutely ill patients is limited by delayed antibody appearance (day 5 to 7 after the onset of illness) among naive persons and by background seropositivity in endemic areas.⁹ Therefore, paired acute and convalescent samples should be obtained as we did for our patient. Molecular testing, such as PCR, provides results faster than serology and does not require paired acute and convalescent samples. The sensitivity of PCR is only 53% while the specificity is 97%.⁸ Early initiation of antimicrobial therapy may prevent some patients from progressing to severe disease.² Mild disease is usually treated with 7 days of doxycycline or penicillin, whereas



Figure 1 ECG showed new atrial flutter and variable atrioventricular nodal conduction with heart rate ranging from 35 to 55/min.



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severe disease is typically treated with parenteral penicillin or a third-generation cephalosporin.

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