A 44-year-old woman was referred to our cardiology department because of septicemia caused by *Listeria monocytogenes* and sick sinus syndrome. The patient had a history of paresthesia of the limbs 7 years earlier with multiple sclerosis–like lesions on brain magnetic resonance imaging and positive tests for antinuclear antibodies and anti-double-stranded DNA antibodies. Full recovery occurred after a short course of glucocorticoids and hydroxychloroquine, and the patient did not experience any relapse.

Two weeks before hospitalization, the patient was admitted to a nearby hospital with a history of 1 week of fatigue, high-grade fever, mild chest pain, and an otherwise unremarkable physical examination. Baseline laboratory tests disclosed a C-reactive protein level of 103 mg/L, hemoglobin of 11.0 g/dL, leukocytosis of 11 350/mm³, plasma fibrinogen of 8.1 g/L, mild hepatic cytolytic and cholestatic changes, a normal serum gamma globulin level, and negative hepatitis B virus, hepatitis C virus, and HIV serology. Blood cultures and urine analysis were negative. Antinuclear antibody levels were 1:1600 (1:80), but no anti-double-stranded DNA or anti-extractable nuclear antigen antibodies were detectable. Thoracic and abdominal computed tomography scans, brain magnetic resonance imaging, and cerebrospinal fluid analysis were normal. Follow-up blood cultures grew *L monocytogenes* in 3 separate samples. Intravenous amoxicillin and gentamycin were started. A repeat ECG showed sick sinus node syndrome with both paroxysmal fast atrial fibrillation and sinus node exit block, and the patient was transferred to our intensive care department.

On admission, temperature was 38.5°C with an otherwise unremarkable physical examination except for arrhythmia. A repeat ECG showed sick sinus node syndrome with both paroxysmal fast atrial fibrillation and sinus node block. Transthoracic and transesophageal echocardiography revealed an abnormal atrial focal thickening in the right atrial free wall, sparing the tricuspid valvular apparatus, and a mild pericardial effusion (Figures 1A and 1B; online-only Data Supplement Movie I). Cardiac magnetic resonance allowed precise delineation of the right atrial mass extension (Figure 2; online-only Data Supplement Movie II) in the posterior and superior wall with no extension to the right coronary artery, tricuspid valve, or pericardium. The mass had an isosignal in T1- and T2-weighted images (Figures 2A through 2C) and no signal increase during first-pass perfusion (online-only Data Supplement Movie II).

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**Figure 1.** Transesophageal echocardiography images at 0° (A) and 97° (B) demonstrating the thickening of the right atrial (RA) free wall (8 mm) with a homogeneous and well-delimited mass located at the posterior and lateral RA wall. RV indicates right ventricle.
III). Delayed hyperenhancement images exhibited a slight peripheral hypersignal 10 minutes after gadolinium injection, with no enhancement of the mass itself (Figure 2D). 18F-fluorodeoxyglucose position emission tomography/computed tomography imaging showed an intense tracer uptake of the right atrial mass, which suggested an inflammatory process (Figures 3A and 3B); no other uptake locations were detected.

Five days after the beginning of antibiotic treatment, apyrexia was noted, and laboratory markers of inflammation had returned to subnormal values. Follow-up blood cultures were sterile. Because of the sick sinus node syndrome, including prolonged sinus pauses, continuous isoprenaline infusion was prescribed. After 2 weeks of treatment, surgery was scheduled because cardiac imaging remained unchanged. Intraoperative examination confirmed a pseudotumoral mass that exclusively involved the right atrial wall. The right atrial free wall was removed and rebuilt with a pericardial patch. An epicardial pacemaker was implanted. Pathological examination of the resected tissue revealed an abscess with parenchymal scarring suggestive of an infectious process (Figures 4A through 4D). Reverse transcription–polymerase chain reaction performed on a frozen tissue sample from this mass was positive for *L monocytogenes*. Postoperative recovery was uneventful, and antibiotics were discontinued after 2 weeks.

*L monocytogenes* is an aerobic, gram-positive coccobacillus that can result in severe sepsis, meningoencephalitis, and a wide variety of focal infections. This pathogen is found in food products (vegetable, raw milk, fish, poultry, and meat). It usually affects immunocompromised hosts. Cardiac involvement in *Listeria* infections is uncommon, generally affecting the left side of the heart, and has a dismal prognosis. There have been ≈70 cases of endocarditis described in the literature that affected native or prosthetic valves of the heart, but only a few cases described myocarditis or cardiac pseudotumor with wall abscesses without valvular involvement. The optimal treatment of these cardiac infections is not known; many antibiotics appear to be effective. It is generally agreed that *Listeria* endocarditis should be treated with ampicillin or penicillin plus aminoglycoside for at least 4 weeks in case of native valve infection and for 6 to 8 weeks in case of prosthetic valve infection, and the indications for surgery are the same as for other forms of endocarditis.

Cardiologists should be aware of the possibility of unusual pseudotumoral right atrial masses caused by *L monocytogenes* causing severe atrial arrhythmias.
Disclosures

None.

References


Unusual Pseudotumoral Right Atrial Involvement in Listeria monocytogenes Septicemia
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In the article by Berthelot-Gracias et al, “Unusual Pseudotumoral Right Atrial Involvement in *Listeria monocytogenes* Septicemia,” which published in the August 7, 2012, issue of the journal (*Circulation*. 2012;126:e66–e68), the first author’s name should have appeared as “E. Berthelot, MD.”

The correction has been made to the current online version of the article. The authors regret the error.