Case Reports

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Unusual Presentation of Listerial Myocarditis

and the Diagnostic Value of Cardiac Magnetic Resonance

Listeria monocytogenes is an infrequent cause of bacterial myocarditis. Myocarditis without evidence of endocarditis is even rarer. Management in such cases involves early diagnosis, antibiotic therapy, and emergency treatment of arrhythmias.

We report the case of a 47-year-old man who presented with features of acute STsegment-elevation myocardial infarction complicated by ventricular tachycardia that necessitated urgent electrical cardioversion. Contrast-enhanced cardiac magnetic resonance images revealed hypertrophy, necrosis, and a mass that was determined to be an abscess caused by L. monocytogenes. Antibiotic treatment led to resolution of the listerial myocarditis.

In addition to reporting our patient's case, we discuss the comparative advantages of cardiac magnetic resonance versus transthoracic echocardiography in characterizing myocarditis, upon presentation and in follow-up evaluation. **(Tex Heart Inst J 2015;42(3):255-8)**

isteria monocytogenes, a foodborne intracellular pathogen, is known to cause infections among neonates, the elderly, immunosuppressed individuals, and pregnant women. This pathogen is a cause of invasive central nervous system involvement such as cerebritis, meningoencephalitis, and abscesses of the spine and brain, and it can cause a flu-like syndrome in pregnant patients that can result in fetal infection or death. Very few cases of listerial endocarditis have been reported, and myocardial involvement is almost unheard of. It is unclear how listeria seeds the myocardium, and investigation is necessary to identify particular strains that might exhibit tissue tropism for the myocardium. Strong suspicion and rapid diagnosis of listerial myocarditis are essential to avoiding a fatal outcome.

We present an unusual case of listerial myocarditis, note its treatment with antibiotics, and highlight the value of gadolinium-enhanced cardiac magnetic resonance (CMR) in the diagnosis and monitoring of this rare condition.

Case Report

In January 2012, a 47-year-old man presented with diaphoresis, progressively worsening shortness of breath, and generalized weakness, a nonproductive cough, and a low-grade fever of 4 weeks' duration. He had been unable to get out of bed and called emergency medical services. The responders found him to have a wide-complex, monomorphic ventricular tachycardia (heart rate, 252 beats/min) (Fig. 1A). Immediate electrical cardioversion promptly restored sinus rhythm. A 12-lead electrocardiogram (ECG) revealed ST-segment elevations in leads V_1 and V_2 (Fig. 1B). The patient's cardiac troponin level was mildly elevated at 0.97 ng/mL. Laboratory results included a white blood cell count of 18.4 mm³, a normal creatinine kinase level, and normal liver and renal functions. Emergency coronary angiograms revealed no significant stenosis. A left ventricular (LV) angiogram showed hypokinesis of the diaphragmatic and anterobasal segments (estimated ejection fraction, 0.35). After the angiography, the patient's course was stable. A transthoracic echocardiogram (TTE) showed asymmetric LV hypertrophy with marked hypertrophy of the basal interventricular septum (Fig. 2A). Moderate hypokinesis of the basal septum and anterior wall was seen. The TTE findings were consistent with nonobstructive hypertrophic cardiomyopathy.

The patient underwent contrast-enhanced CMR for further evaluation. The images showed a large but poorly defined complex mass compatible with an abscess,



Fig. 1 The electrocardiographic rhythm strip shows widecomplex regular tachycardia, suggesting ventricular tachycardia. Below the strip, the 12-lead electrocardiogram shows STsegment elevation in leads V_1 and V_2 , and reciprocal ST-segment depression in leads II, III, aVF, and V_4 through V_6 .

and necrosis involving the interventricular septum (Fig. 2B). The mass extended into the mid anteroseptal and mid anterior segments and into the proximal portion of the right ventricular outflow tract. Prominent myocardial edema was seen in these regions, and a perfusion sequence revealed less vascularity than in the myocardium. Late gadolinium enhancement indicated necrosis (Fig. 2C). No myocardial infarction was observed beyond the area affected by the mass, and there was no phenotypic evidence of hypertrophic cardiomyopathy. The patient's LV ejection fraction was 0.44. One of 4 blood cultures began growing gram-positive rods, identified as Listeria monocytogenes, and the abscess was judged to be from listerial infection. The patient was started on intravenous ampicillin and gentamicin to treat listerial myocarditis. Transesophageal echocardiography, performed to rule out endocarditis, showed severe hypertrophy of the interventricular septum (thick-



Fig. 2 A) Transthoracic echocardiogram (parasternal long-axis view) shows marked hypertrophy of the basal interventricular septum (arrow). B) Cardiac magnetic resonance T2-weighted image shows a mass (arrow) that involves the basal anterior, anteroseptal, and anterolateral segments of the hypertrophied interventricular septum. C) Late gadolinium enhancement on cardiac magnetic resonance indicates necrosis (arrow). D) Transthoracic echocardiogram (parasternal long-axis view) shows thinning of the interventricular septum and anterior wall (arrow).

ness, 2.7 cm) and anterobasal wall (thickness, 2.9 cm); the inferior, posterior, lateral, and apical walls were of normal thickness. There was severe hypokinesis of the septal and anterobasal walls, and no valvular vegetation or intraventricular thrombus.

The patient's 3-week antibiotic regimen yielded improvement in his fever and leukocytosis. After 2.5 weeks of this therapy, ECG, TTE, and CMR were repeated. On ECG, the ST-segment elevations in the anteroseptal leads had reverted to baseline. The TTE showed regression (thinning) of the septum and anterior wall (Fig. 2D), and both regions exhibited severe hypokinesia. The patient's LV ejection fraction had decreased to 0.34, and he had an increased LV end-diastolic volume. A T2-weighted CMR image showed a thinwalled, 3-mm-thick aneurysm that had replaced the previously infiltrating myocardial abscess in the basal anterior and anteroseptal wall. One cm of nonenhancing residual necrotic myocardium was seen in the mid inferoseptum at the interface of the aneurysm and the normal myocardium, with surrounding enhancement but without tissue edema (Fig. 3). The patient's blood



Fig. 3 Cardiac magnetic resonance T2-weighted image, obtained after the patient completed the antibiotic regimen, shows a thin-walled aneurysm (arrow) at the former site of the abscess in the basal anterior and anteroseptal wall. Non-enhancing residual thinned, fibrotic myocardium is seen in the mid inferoseptum at the interface of the aneurysm and normal myocardium (arrowhead), with surrounding enhancement but without tissue edema.

cultures were consistently negative. An electrophysiologic study revealed inducible ventricular arrhythmia, so an automated implantable cardioverter-defibrillator was placed for secondary prevention. A repeat TTE in June 2012 showed a dilated LV with apical and septal akinesia (ejection fraction, 0.35–0.40). As of January 2015, the patient's LV ejection fraction had improved to 0.45, and no arrhythmias had recurred.

Discussion

Listeria monocytogenes is an aerobic, gram-positive coccobacillus. It is ubiquitous and is found in animals, animal products, soil, water, and sewage. It is an infrequent cause of human disease. Listeria is transmitted to human beings in contaminated food, such as vegetables, raw milk, fish, poultry, and meat. Risk factors for infection include age extremes (<1 mo and >60 yr), pregnancy, organ transplantation, steroid therapy, human immunodeficiency virus, and hematologic and lymphoid malignancies. Several forms of listerial infection have been recognized in human beings: meningoencephalitis, oculoglandular listeriasis, cervicoglandular infections, pneumonia, and cutaneous and perinatal infant septicemia. Listerial endocarditis affecting native and prosthetic heart valves has been reported in approximately 70 instances and is associated with a 37% mortality rate.1 Myocarditis, a potential sequela of listerial endocarditis, is universally fatal if untreated. However, reports of listerial myocarditis or cardiac-wall abscess without valvular involvement in immunocompetent patients are few.²⁻⁴ Listerial myocarditis has mimicked graft rejection in patients who have undergone cardiac transplantation.⁵ Our patient's case is remarkable with regard to the clinical presentation, site of disease, and excellent resolution upon treatment. Also unusual in this case is the lack of any of the aforementioned risk factors for listerial infection. However, listerial infection from contaminated cantaloupes had been reported in our patient's geographic area around the same time as his illness and might have been the culprit.

As in our patient's case, contrast-enhanced CMR has proved to be even more valuable than TTE in the evaluation of patients with myocarditis.⁶ It has been recommended as a highly sensitive, nonoperator-dependent, versatile mode of investigation for suspected myocarditis. The identification of tissue edema helps to characterize the disease as "active" myocarditis.⁷ Late gadolinium enhancement can indicate necrosis.⁸ We found a high diagnostic usefulness of contrast-enhanced CMR, and we think that CMR will continue to serve as the definitive mode of imaging for myocarditis.

We recommend awareness that pseudotumoral masses in the ventricular septal wall or free wall might be caused by *L. monocytogenes*, which in turn might be causing ventricular arrhythmias.

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