Listeria Monocytogenes Pericarditis in the Immune Compromised: A Case Report in a Newly Diagnosed Alpha Light Chain Cardiac Amyloidosis Patient

Itai Ghersin MD MHA[1], Omar Abu Alheija MD[2,3], Zaher S. Azzam MD[1,4] and Roni Nasser MD[1]

[1]Department of Internal Medicine "B", Rambam Health Care Campus, Haifa, Israel
[2]Department of Internal Medicine, Holy Family Hospital, Nazareth, Israel
[3]Faculty of Medicine in the Galilee, Bar-Ilan University, Safed, Israel
[4]Rappaport Faculty of Medicine, Technion–Israel Institute of Technology, Haifa,

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Purulent bacterial infections are an uncommon cause of pericarditis [1]. *Listeria monocytogenes* is a gram-positive, rod-shaped bacterium that can be found in soil, vegetation, water, and sewage, as well as in feces of humans and animals [2]. Listeria infection is associated with consumption of contaminated food products. It has a higher incidence among elderly, pregnant, and immunocompromised patients. Listeria pericarditis is a very rare disease, which is usually fatal [1]. Most reported cases of listeria pericarditis have been found in patients with significant underlying co-morbidities [1].

We reported the case of a patient who was diagnosed with cardiac amyloidosis, and subsequently developed fatal listeria pericarditis.

PATIENT DESCRIPTION

A 75-year-old male patient was admitted to our department with worsening dyspnea and leg edema.

Six months earlier the patient was admitted to a different hospital with similar symptoms. Transthoracic echocardiography (TTE) was performed, revealing moderate diastolic dysfunction with preserved ejection fraction. The patient was diagnosed with heart failure with preserved ejection fraction and treated with diuretics, which showed marked symptomatic improvement.

Prior to that hospitalization, the patient was healthy, without any cardiovascular risk factors and did not take medications on a regular basis. He was physically active and worked in a physically demanding job as a mechanic until his first hospital admission. At admission to our department, the patient's vital signs were: blood pressure 90/40 mmHg, pulse 78 beats per minute (regular), temperature 36.7°C, and saturation in room air 98%.

Physical examination revealed a cachexic patient who was awake and showed no signs of respiratory distress. Positive findings included increased jugular venous pressure (JVP), decreased air entry to the right lobe without crackles or wheezes, and a normal expirium and bilateral pitting edema +2.

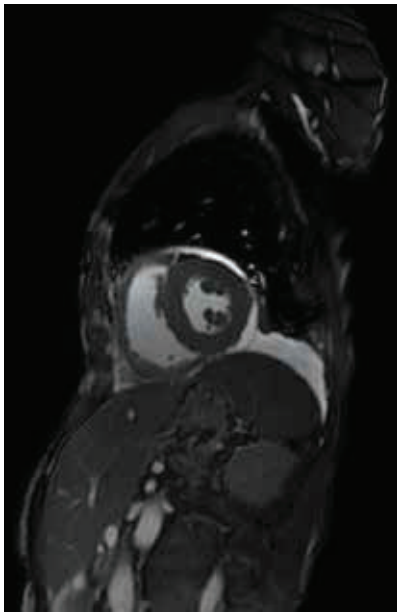
Complete blood count showed mild microcytic anemia of Hb 11.5 g/dl (lower normal limit 13 mg/dl) and mean corpuscular volume 78 fl (lower normal limit 79 fl) with high red cell distribution width (17%). White blood cell count and platelets were normal. Blood chemistry revealed kidney injury, creatinine 1.4 mg/dl (normal upper limit 1.3 mg/dl), and blood urea nitrogen (BUN) of 47 mg/dl (normal upper limit 25.7 mg/dl). The patient's baseline renal function was normal. Electrolytes were within normal range. In addition, mild direct hyperbilirubinemia was noted (total bilirubin -1.7 mg/dl, direct bilirubin -1 mg/dl) as were elevated alkaline phosphatase (252 U/L) and gamma-glutamyl transferase (90 U/L). Aspartate aminotransferase and alanine aminotransferase were normal. Electrocardiogram (ECG) showed sinus rhythm of low voltage, and a first-degree atrioventricular block without signs of ischemia. Chest X-ray disclosed a large right-sided pleural effusion.

A diagnosis of acute decompensated heart failure was made, with predominant right-sided manifestations, and treatment containing intravenous therapy diuretics, was initiated. During the patient's hospital stay, his symptoms and vital signs improved.

Since the patient's history was inconclusive for the etiology of heart failure, a clinical workup was performed and included a thorough history taking and physical examination that were unrevealing. TTE results showed a normal left ventricle (LV) systolic function with abnormal diastolic function, and mildly increased LV wall thickness, with a sparkling appearance of myocardium. Mild enlargement of the left atrium and moderate enlargement of the right atrium were also noted.

Based on these findings, a suspicion of restrictive infiltrative cardiomyopathy, including cardiac amyloidosis was raised; therefore, cardiac magnetic resonance imaging was ordered. The results disclosed diffuse heterogenous subendocardial

Figure 1. Cardiac magnetic resonance imaging revealed diffuse heterogeneous subendocardial delayed enhancement in a pattern that was pathognomonic for cardiac amyloidosis



delayed enhancement, in a pattern pathognomonic for cardiac amyloidosis [Figure 1]. Serum protein electrophoresis showed a monoclonal peak of kappa light chains.

Based on these results, a diagnosis of cardiac amyloidosis, most likely amyloid light-chain amyloidosis. Due to his marked clinical improvement, the patient was discharged with oral diuretics, with a recommendation to undergo bone marrow and fat pad biopsies on an outpatient basis.

One month later, the patient was admitted to a different hospital due to worsening dyspnea and weakness. His vital signs at admission were: blood pressure 60/40 mmHg, pulse 150 beats per minute, respiratory rate 25/minute, temperature 36.8°C, and saturation in room air 89%. Physical examination revealed a tachypneic patient with increased JVP, decreased heart sounds, and pulsus paradoxus. Complete blood count showed leukocytosis (17900/µl) with neutrophilia (69%). Red blood cell count and platelets were normal. Blood chemistry revealed acute kidney injury, with creatinine of 2.9 mg/dl (normal upper limit 1.3 mg/dl) and BUN of 70 mg/dl (normal upper limit 25.7 mg/dl). ECG revealed sinus tachycardia with low voltage. Chest X-ray demonstrated an enlarged heart with a large right-sided pleural effusion.

Bedside ECG revealed a large pericardial effusion, with classical manifestations of tamponade. Echocardiography-guided pericardiocentesis was performed, and 200 cc of sero-bloody effusion was drained. The fluid was sent to cultures and cytology.

Despite pericardiocentesis, the patient remained in the state of shock. He was mechanically ventilated and hemodynamically supported with high-dose vasopressors. Unfortunately, resuscitation efforts failed and patient was pronounced dead.

Later, cultures from the pericardial effusion were positive for *Listeria monocytogenes*.

COMMENT

Cardiac amyloidosis is a challenging diagnosis to make; and therefore, it is vastly under diagnosed [3]. This disorder should be suspected in patients with diastolic heart failure that cannot be attributed to other causes [4]. Additional clinical clues include hypotension and low ECG voltage, which are very uncommon in heart failure due to other etiologies [4].

The prognosis of AL amyloidosis is grave, as our case demonstrates, with a median survival of 11 months according to one study [5]. Transthyretin amyloidosis appears to have a slower disease course and better prognosis [5].

Bacterial purulent pericarditis is a rare entity. It accounts for approximately 1% of all pericarditis cases [1]. The most frequently isolated bacteria reported are *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis*. Listeria pericarditis is extremely rare [1]. The outcome of purulent bacterial pericarditis is poor, with an approximate mortality rate of 40%. Based on a recent review of literature [1], it seems that the prognosis of listeria pericarditis is even worse. Six of the nine reported cases resulted in death. Mortality of this condition is estimated at approximately 70% when adding our case to the tally.

Most patients with listeria pericarditis had a significant underlying disease, such as cirrhosis, hemochromatosis, or human immunodeficiency viruses [1]. Our patient was no different, presenting with AL cardiac amyloidosis, which probably put him at a predisposition to develop this fatal infection. To the best of our knowledge, this case the first reported case of listeria pericarditis in a patient with a plasma cell dyscrasia or cardiac amyloidosis.

CONCLUSIONS

Our case highlights the importance of suspecting cardiac amyloidosis in patients presenting with heart failure with atypical manifestations, such as hypotension and low ECG voltage. It also provides knowledge of listeria pericarditis, a rare and generally fatal diagnosis. To the best of our knowledge, our case is the first ever case reported of listeria pericarditis complicating cardiac amyloidosis.

Correspondence
Dr. I. Ghersin
Dept. of Internal Medicine B, Rambam Health Care Campus, Haifa, 31096 Israel
Phone: (972-4) 777-1976
Fax: (972-4) 777-3252
email: ighersin@gmail.com

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