Heart Failure From Gouty Myocarditis: A Case Report

Background: Myocarditis is an important cause of heart failure, and successful treatment depends on identifying the underlying cause and mechanism. Infectious and autoimmune diseases are the most common causes, although myocarditis can also be caused by endocrine disorders, such as pheochromocytoma (1); infiltrative disorders, such as amyloidosis (2); and metabolic disorders. Gout is a metabolic disorder characterized by increased uric acid in the blood and the deposition of amorphous urate crystals that cause a vigorous inflammatory reaction in the joints, kidneys, and soft tissues. Urate crystals have been found in coronary vessels and cardiac valves but, to our knowledge, not in the myocardium (3).

Objective: To report what we believe to be previously unreported myocardial damage from the deposition of urate crystals in the heart.

Case Report: A 49-year-old man with severe gouty polyarthritis and soft tissue tophi was hospitalized because of dyspnea and edema of his lower limbs. He had stopped taking allopurinol and colchicine for several months before admission because of gastric discomfort and diarrhea. On physical examination during admission, blood pressure was normal and tachycardia of 120 beats/min with a gallop rhythm was found. His echocardiogram showed sinus rhythm with left anterior fascicular block and left atrial and ventricular overload. On routine laboratory tests, renal function was normal but he had elevated levels of C-reactive protein (28.57 nmol/L; normal value, <0.5 nmol/L), serum uric acid (832.8 μmol/L; normal value, <416.4 μmol/L), troponin T (0.054 μg/L; normal value, <0.014 μg/L), and pro-B-type natriuretic peptide (14 890 ng/L; normal value, <308 ng/L). In addition, he had leukocytosis (leukocyte count, 0.00149 x 10⁹ cells/L with 88% neutrophils).

The patient’s echocardiogram showed biventricular dilation with a left ventricular ejection fraction of 30% and a right ventricular ejection fraction of 39%. Cardiac magnetic resonance imaging confirmed severely reduced left ventricular function. It also identified multiple foci of late gadolinium enhancement, mild absorption of fluid with expansion of the left ventricular myocardium (edematous imbibition), and predominant septal involvement. Coronary angiography found normal coronary arteries. Left ventricular biopsy provided endomyocardial samples that were processed for histology and electron microscopy. Histology revealed extensive lymphocytic myocarditis with inflammatory infiltrates associated with areas of focal necrosis involving adjacent cardiomyocytes, which contained inclusion bodies (Figure, A). Electron microscopy using semifine (Figure, B) and ultrastructural (Figure, C)
and D) sections found that the inclusion bodies contained amorphous crystals, and these crystals had negative birefringence when exposed to polarized light. Microcrystals or paracrystals of different sizes but the same shape were present in the cytosol (Figure, C), in membrane-bound vacuoles, and inside the Golgi apparatus (Figure, D). In addition, we used real-time polymerase chain reaction to test 2 biopsy fragments for the presence of the following major cardiotropic viruses: adenovirus, Epstein–Barr virus, herpesvirus, parvovirus B19, cytomegalovirus, enterovirus, influenza A or B virus, and hepatitis C virus; these tests found no evidence of the viruses.

We treated the patient with allopurinol (300 mg/d) and prednisone (1 mg/kg of body weight daily). After 4 weeks of treatment, his cardiovascular condition improved and his left ventricular ejection fraction measured with 2-dimensional echocardiography had increased from 30% to 45%.

Discussion: Many studies have found that hyperuricemia is associated with increased risk for coronary artery disease, heart failure, and sudden death. The mechanism for this association remains uncertain, and to the best of our knowledge myocardial damage from urate crystal deposition has not been reported. We identified monosodium urate crystals by histology, electron microscopy, and negative birefringence on exposure of a frozen myocardial sample to polarized light.

On the basis of our experience with this patient, we conclude that gout can cause myocarditis with cardiac dilatation, cardiac dysfunction, and heart failure, particularly in patients with tophaceous and untreated gout. The mechanism seems to be deposition of amorphous urate crystals inside cardiomyocytes, which induces a strong inflammatory reaction and cell death. In our patient, treatment with allopurinol and steroids was followed by alleviated symptoms and cardiac contractility. Endomyocardial biopsy, which has infrequent and usually transient complications (4), is necessary to exclude other possible causes and confirm the diagnosis.

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References