Prolonged use of hydroxychloroquine (HCQ) has been implicated in the development of conduction disturbances and myocardial dysfunction.1 We report a case of cardiomyopathy after 10 years of HCQ therapy in a 66-year-old woman with systemic lupus erythematosus (SLE).

Case Presentation
A 66-year-old white woman with a 24-year history of SLE presented to our institution with decompensated heart failure. She had no cardiac history until 8 weeks before admission, when she presented to an outside hospital for new-onset heart failure. Other significant medical history included gout, dyslipidemia, hypertension, and SLE first diagnosed in 1986 complicated by World Health Organization class 4 lupus nephritis, diagnosed in 2001. She was taking HCQ (400 mg daily), prednisone (<5 mg/d), and azathioprine for treatment of SLE. The evaluation for new-onset heart failure at the outside facility consisted of an echocardiogram, which showed a left ventricular ejection fraction of 35% and coronary angiogram that revealed mild, nonobstructive coronary artery disease. The ECG showed new-onset, 3rd-degree atrioventricular block. She underwent placement of a biven-
tricular pacemaker and implantable cardioverter-defibrillator. Cardiac therapy was begun and consisted of carvedilol, hydralazine, isordil, and torsemide.

On admission, a transthoracic echocardiogram was performed and showed moderate to severe biatrial enlargement, an ejection fraction of 25%, and increased biventricular wall thickness. The left ventricular internal diastolic diameter was 4.4 cm. A right heart catheterization was performed and showed restrictive physiology as evidenced by equalization of pressures in the right atrium and pulmonary capillary wedge pressure recordings, steep y descent, and rapid diastolic filling (Figure 1A and 1B). She underwent endomyocardial biopsy for further evaluation of suspected restrictive cardiomyopathy. Hematoxylin and eosin staining showed myocytes of normal size with numerous clear vacuoles and absence of inflammatory cells, consistent with findings described in HCQ toxicity. HCQ was discontinued and the patient was treated with furosemide and milrinone, responding well with normalization of hemodynamics on repeat catheterization 1 week later. However, 2 months after discharge, a repeat echocardiogram showed decline in left ventricular ejection fraction to 15% to 20%. She was readmitted with multiorgan failure and ultimately died within 2 months of her initial diagnosis.

**Discussion**

Echocardiographic findings of HCQ cardiomyopathy are variable, ranging from increased ventricular wall thickness with or without dilatation and systolic or diastolic dysfunction and can mimic other causes of myocardial dysfunction in SLE patients such as myocarditis, amyloidosis, ischemic heart disease, other toxic drugs (eg, amiodarone), and rarely, Fabry disease or other storage disorders. Cardiac conduction disturbances including atrioventricular block and bundle-branch block have been previously described in such cases and were present in our patient. Histological evaluation with an endomyocardial biopsy is useful to exclude above mentioned competing diagnoses. Biopsy findings in myocarditis are nonspecific and characterized by perivascular mononuclear infiltrates and features of myocyte injury, whereas HCQ toxicity is associated with distinct features such as absence of myocyte injury with vacuolization, as seen in this case. We report this case to highlight the significance of HCQ-induced cardiomyopathy and the role of endomyocardial biopsy in establishing a diagnosis in patients who present with new atrioventricular heart block and subsequent heart failure.

**Disclosures**

None.

**References**


**KEY WORDS:** cardiomyopathy ▪ heart failure ▪ drug toxicity ▪ hydroxychloroquine ▪ cardiomyopathy
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Circ Heart Fail. 2011;4:e7-e8
doi: 10.1161/CIRCHEARTFAILURE.110.959916
Circulation: Heart Failure is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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