A 69-year-old woman presented with symptoms and examination findings consistent with congestive heart failure. She had history of hypertension, mild renal insufficiency, and bilateral hip replacements. Transthoracic echocardiogram showed left ventricular ejection fraction of 25% to 30%, normal left ventricular size and left ventricular wall thickness, and moderate pericardial effusion (Figures 1 and 2; Movie I in the Data Supplement). Pericardiocentesis removed 650 mL of straw-colored fluid, which was negative for infection or malignant cells. Coronary angiogram showed normal coronary arteries.

She was discharged on low-dose beta blocker, angiotensin-converting enzyme inhibitor, and diuretics. Over the next 4 months, her biventricular function declined significantly, leading to multiple hospitalizations for congestive heart failure or symptomatic hypotension.

Cardiac MRI revealed normal left ventricular size and wall thickness with severely reduced left ventricular ejection fraction of 14%. Right ventricle was normal in size with severely reduced systolic function (Movie II in the Data Supplement). T2 images were suggestive of edema with the T2 signal intensity normalized to skeletal muscle tissue of 2.4 (Figure 3). There was prominent enhancement of the subepicardial lateral walls. In addition, enhancement of left and right atrial walls was noted (Figures 4 and 5).

On obtaining further history, she mentioned that her hip prosthesis (DePuy, Johnson & Johnson, +2 mm metal-on-metal liner) had been squeaking for the last 2 years, and there were tentative plans for its removal owing to its recall status. Given the delayed enhancement pattern on her cardiac MRI and elevated serum cobalt and chromium levels, a right heart catheterization with endomyocardial biopsy was performed. Histopathology of myocardium showed hypertrophic myocytes with increased interstitial fibrosis and histological features consistent with a dilated cardiomyopathy probably caused by elevated levels of cobalt (Figure 6). No interstitial inflammatory cell infiltrate, iron deposition, or amyloid was seen. The electron microscopy showed nonspecific increased lipid and degenerated lipid deposits with minimal mitochondrial changes, which is also consistent with a diagnosis of cobalt cardiomyopathy, though myocardial cobalt levels could not be obtained.

The patient progressed to cardiogenic shock and was transferred to a tertiary care hospital for advanced heart failure support and prosthetic hip removal with the diagnosis of cobalt cardiomyopathy. The patient was stabilized and nonhypertrophied ventricle associated with pericardial effusion have been reported by echocardiography in other cases of cobalt cardiomyopathy. Recently, there have been several case reports of cobalt-induced cardiomyopathies related to failure of metal-on-metal hip prosthesis. These cardiomyopathies associated with high circulating level of heavy metals usually tend reverse with removal of the offending prosthesis. Chelation and phlebotomy in addition to treatment of heart failure has been suggested by some authors, though experience with these therapies is limited.

The findings of decreased systolic function in a nondilated and nonhypertrophied ventricle associated with pericardial effusion have been reported by echocardiography in other cases of cobalt cardiomyopathy. Recently, biventricular hypertrophy in transmural pattern sparing basal and mid septum in late gadolinium enhancement images was described in another reported case; we feel that the presence
of edema on T2 images and prominent enhancement of the subepicardial lateral walls would represent another pattern to be expected on contrast-enhanced MRI with cobalt cardiomyopathy. Thus, we suggest that in addition to obtaining a good history (including type of metal-on-metal implant) in patients with suspected cobalt cardiomyopathy, workup should also include serum cobalt level, echocardiogram with strain imaging, cardiac MRI, and obtaining myocardial cobalt concentration if possible.

Disclosures

None.

References


Key Words: cobalt ❖ cardiomyopathy ❖ heart failure ❖ magnetic resonance imaging

Figure 1. Echocardiographic image showing restriction by mitral inflow.

Figure 2. Echocardiographic images showing tissue Doppler imaging.

Figure 3. T2 mid ventricular short axis slice suggesting myocardial edema with a T2 SI ratio to skeletal muscle of 2.4.

Figure 4. Post-contrast delayed gadolinium enhancement 4 chamber image showing prominent late gadolinium enhancement of the lateral epicardial wall.
Figure 5. Post-contrast delayed gadolinium enhancement basal short axis image showing prominent late gadolinium enhancement of the lateral and inferior epicardial walls.

Figure 6. Hematoxylin and eosin stain (200×) showing markedly hypertrophic myocytes with cytoplasmic vacuolization and degeneration of myocytes. There is a marked increase in interstitial fibrosis as well.

Figure 7. Electron microscopy (12000×) image of myocardial biopsy showing increased degenerated lipid deposits with minimal mitochondrial changes within cardiac myocytes.
Unusual Case of Congestive Heart Failure: Cardiac Magnetic Resonance Imaging and Histopathologic Findings in Cobalt Cardiomyopathy
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**Video legend:**

Video 1: Four chamber echocardiogram showing pericardial effusion and reduced LVEF

Video 2: Four chamber cine SSFP showing biventricular dysfunction and a large circumferential pericardial effusion with evidence of increase pericardial pressure.