Epicardial Lipomatous Hypertrophy Mimicking Pericardial Effusion: Characterization With Cardiovascular Magnetic Resonance

Christopher A. Miller and Matthias Schmitt

_Circ Cardiovasc Imaging_ 2011;4;77-78;
DOI: 10.1161/CIRCIMAGING.110.957498

Circulation: Cardiovascular Imaging is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 75251
Copyright © 2011 American Heart Association. All rights reserved. Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/4/1/77.full
A 62-year-old man with no history of cardiac disease was referred because of exertional dyspnea. His body mass index was elevated at 29 kg/m², and a large cutaneous lipoma was present on his abdominal wall. Transthoracic echocardiography was performed and initially reported to demonstrate a moderate-sized global pericardial effusion (Figure 1 and Movies 1 and 2). Consideration was given to pericardiocentesis; however, subsequent review suggested that the appearances may have been due to pericardial thickening (Movie 3). Cardiovascular magnetic resonance (CMR) imaging was performed for clarification.

A thick layer of epicardial tissue, measuring up to 29 mm deep, was seen to surround the myocardium on balanced steady-state free precession (SSFP) cine images (Figure 2 and Movie 4). On both SSFP and half-Fourier single-shot fast spin-echo images, signal intensity was high, indeed identical to that from subcutaneous fat. Using a spatial modulation of magnetization sequence ("tagging"), the epicardial tissue appeared to be adherent to the myocardium (Movie 5). The interatrial septum was also markedly thickened (23 mm), with sparing of the fossa ovalis, and had the same high signal intensity (Figure 2C). Fast spin-echo images with a fat-saturation inversion recovery prepulse (which significantly reduces, or “nulls,” the signal from fat) confirmed the epicardial and interatrial septal tissue to be fat (Figure 3). A diagnosis of epicardial lipomatous hypertrophy with concomitant lipomatous hypertrophy of the interatrial septum was made. The pericardium itself was thin and of normal appearance, with no evidence of pericardial effusion; indeed, the contrast provided by the fat allowed for unusually good delineation of the pericardium, highlighting its cranial extension.

Cardiac lipomatosis is characterized by the accumulation of nonencapsulated mature adipose tissue caused by hyperplasia of lipocytes. The etiology is unknown, but it may be associated with obesity and advancing age.1 The most frequent manifestation is lipomatous hypertrophy of the interatrial septum. Massive epicardial lipomatous hypertrophy is less well documented. Although histologically benign, it has been reported to cause cardiac tamponade, requiring decompressive pericardiectomy.2 In the presented case, cine imaging demonstrated normal right heart and caval appearances, phase contrast imaging with velocity encoding demonstrated normal systemic venous inflow, and on real-time, free-breathing imaging, ventricular septal motion was seen to be normal, all of which suggested reassuring cardiac filling physiology.

The case highlights the possibility of mistaking epicardial lipomatous hypertrophy for pericardial effusion on transthoracic echocardiography. The tissue characterization provided by CMR allowed the diagnosis to be made, avoiding the need for invasive investigation or unnecessary intervention. The functional data

Figure 1. Echocardiographic images. Parasternal long-axis (A) and short-axis (B) views showing the echolucent zone surrounding the heart that was mistaken for a pericardial effusion (asterisk, dashed line). LV indicates left ventricle; RV, right ventricle; LA, left atrium; and Ao, aorta.
provided by CMR suggested that the epicardial lipomatous hypertrophy was not affecting cardiac function.

**Disclosures**

None.

---

**References**