Extensive Subepicardial Fibrosis in a Patient With Apical Hypertrophic Cardiomyopathy With Persistent ST-Segment Elevation Simulating Acute Myocardial Infarction

Jong-Won Ha, Byoung-Wook Choi, Se-Joong Rim, Seok-Min Kang, Yangsoo Jang, Namsik Chung, Won-Heum Shim and Seung-Yun Cho

Circulation. 2005;112:e49-e50
doi: 10.1161/CIRCULATIONAHA.104.479386
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539

Extensive Subepicardial Fibrosis in a Patient With Apical Hypertrophic Cardiomyopathy With Persistent ST-Segment Elevation Simulating Acute Myocardial Infarction

Jong-Won Ha, MD, PhD; Byoung-Wook Choi, MD; Se-Joong Rim, MD, PhD; Seok-Min Kang, MD, PhD; Yangsoo Jang, MD; Namsik Chung, MD, PhD; Won-Heum Shim, MD, PhD; Seung-Yun Cho, MD, PhD

Apical hypertrophic cardiomyopathy is a unique form of hypertrophic cardiomyopathy (HCM), in which the hypertrophy of myocardium predominantly involves the apex of the left ventricle. The ECG in apical HCM typically shows repolarization changes in the anterolateral leads and sometimes giant negative T waves. Previous reports have shown that apical HCM may mimic myocardial infarction, although its underlying mechanism is unclear. This case report demonstrates typical echocardiographic and MRI features of apical HCM, but with unusual electrocardiographic features characterized by chronic ST-segment elevation in the precordial leads. Contrast-enhanced MRI showed unusual extensive subepicardial delayed hyperenhancement at the left ventricular apex, suggesting that subepicardial fibrosis may be a possible cause for this unusual ECG abnormality in patients with apical HCM. Because this condition can be misdiagnosed as acute infarction, resulting in unwarranted thrombolytic therapy or emergency angiography, we believe this case is important as a reminder that acute infarction is not the only cause of ST-segment elevation.

In March 2003, a 44-year-old man presented with exertional dyspnea. He was normotensive and had never experienced chest pain or other symptoms suggestive of heart disease. A routine 12-lead ECG (Figure 1) revealed an ST-segment elevation in V_3 to V_6 precordial leads, which made us suspect possible acute myocardial infarction. Physical examination revealed no abnormal findings, and cardiac biomarkers were negative. Echocardiography and MRI (Figure 2 and Movies) revealed a severe hypertrophy confined to the left ventricular apex, suggesting apical HCM. Coronary angiography by MRI showed no significant luminal narrowing (Figure 3). A contrast-enhanced image obtained by MRI showed prominent delayed hyperenhancement localized at the subepicardial area of the left ventricular apex (Figure 4). Follow-up ECG obtained 1 year later showed persistent ST elevation in precordial leads without interval change (Figure 5).

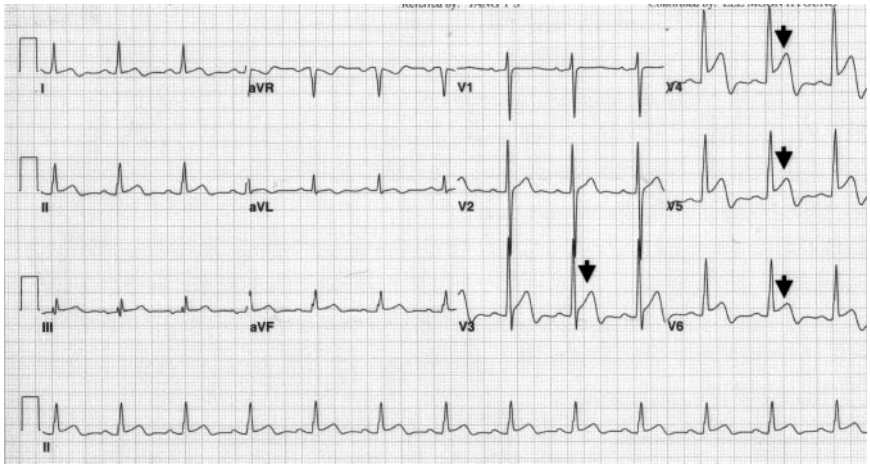

Figure 1. Initial ECG showing ST-segment elevation in V_3 to V_6 precordial leads, indicative of possible acute myocardial infarction.

From Cardiology Division, Yonsei University College of Medicine, Yonsei Cardiovascular Center, Seoul, Korea.

The Data Supplement is available at http://www.circ.ahajournals.org/cgi/content/full/112/3/e49/DC1.

Correspondence to Jong-Won Ha, MD, PhD, Cardiology Division, Yonsei University College of Medicine, Yonsei Cardiovascular Center, CPO Box 8044, Seoul, Korea. E-mail jwha@yumc.yonsei.ac.kr

(*Circulation.* **2005;112:e49-e50.**)

Circulation **is available at http://www.circulationaha.org** **DOI: 10.1161/CIRCULATIONAHA.104.479386**

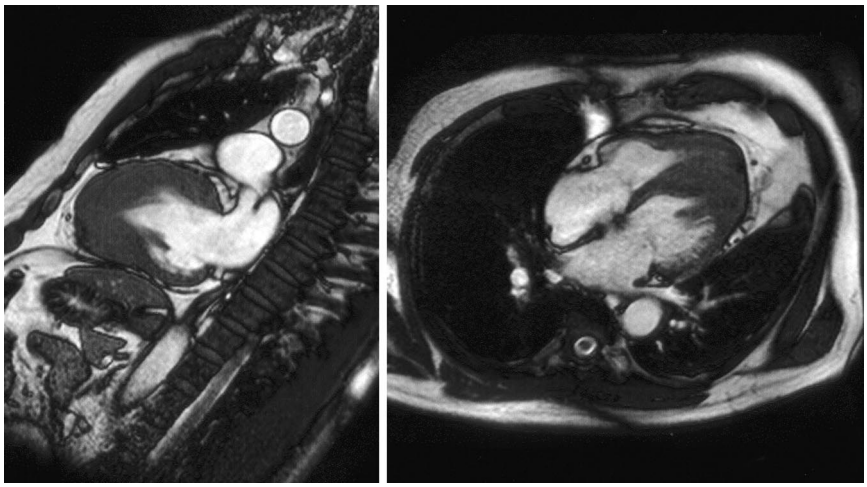

Figure 2. MRI revealed severe hypertrophy confined to left ventricular apex, suggesting apical HCM.

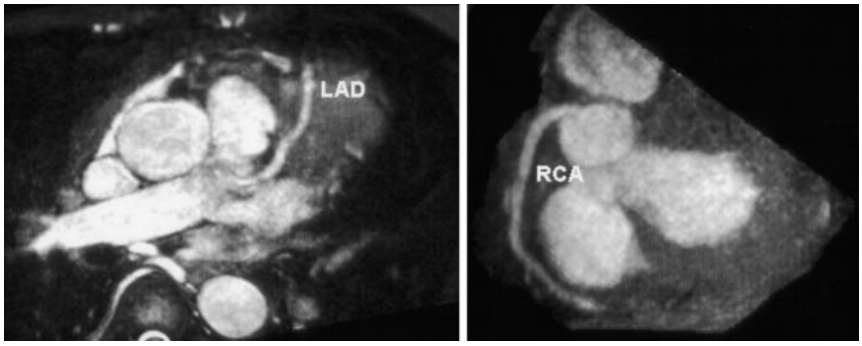

Figure 3. Coronary angiography by MRI showed no significant luminal narrowing.

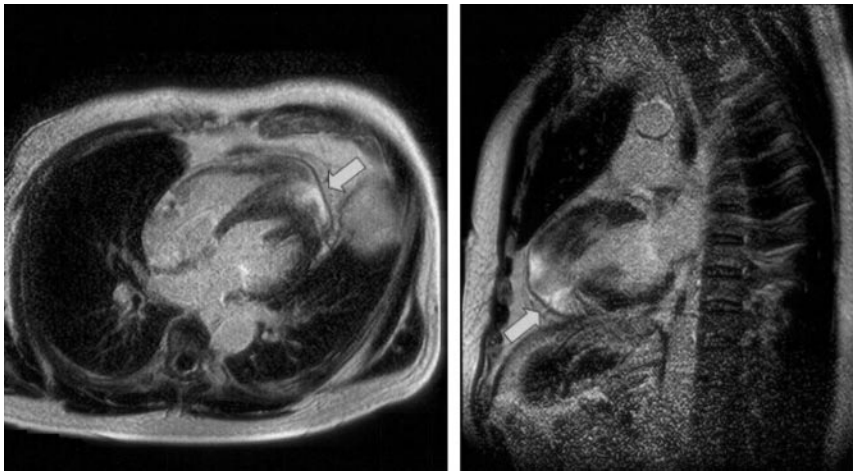

Figure 4. Contrast-enhanced image obtained by MRI of 4-chamber view (left) and 2-chamber view (right) showed prominent delayed hyperenhancement localized at subepicardial area of left ventricular apex.

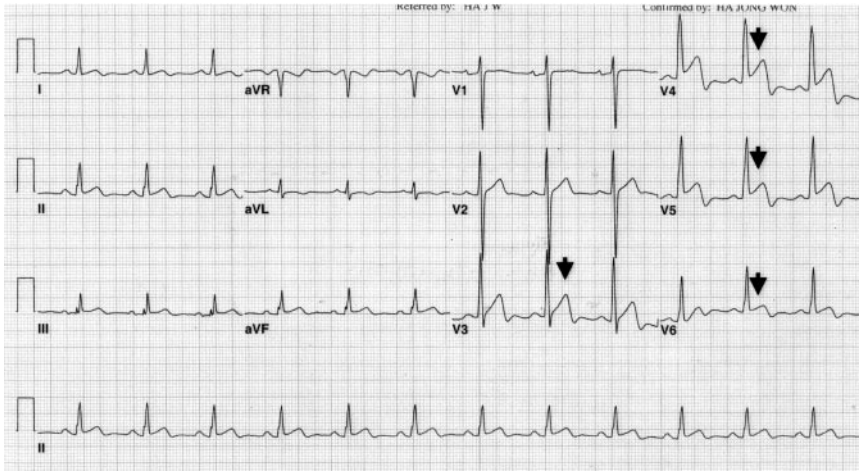

Figure 5. Follow-up ECG 1 y later showed persistent ST elevation in precordial leads without interval change.