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Circulation. 2006;113:e4-e5

doi: 10.1161/CIRCULATIONAHA.105.554139

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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

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Assessment of Pericardial Inflammation in a Patient With Tuberculous Effusive Constrictive Pericarditis With ^{18}F -2-Deoxyglucose Positron Emission Tomography

Jong-Won Ha, MD, PhD; Jong-Doo Lee, MD, PhD; Young-Gook Ko, MD; Mijin Yun, MD; Se-Joong Rim, MD, PhD; Namsik Chung, MD, PhD; Sang-Ho Cho, MD, PhD

Constrictive pericarditis is an uncommon but treatable cause of heart failure that results from a variety of acute inflammatory processes. Although complete surgical pericardiectomy remains the only definitive treatment, complete resection may not be easy in the presence of residual inflammation and friable pericardium. However, no reliable diagnostic test is available to accurately evaluate the inflammation of the pericardium. This case illustrates that a noninvasive imaging modality, ^{18}F -2-deoxyglucose (FDG) positron emission tomography, may be useful for the assessment of pericardial inflammation.

A 75-year-old woman was evaluated for progressively worsening exertional dyspnea. On physical examination, her jugular vein was distended, and her liver was enlarged. An ECG showed sinus tachycardia with low-voltage QRS. Echocardiography showed a moderate amount of pericardial effusion without significant hemodynamic compromise. However, abnormal septal motion (septal bouncing) was noted, suggestive of constrictive physiology. FDG positron emission

tomography showed prominent uptake in the visceral and parietal pericardium, suggesting active inflammation (Figure 1, left panel). Closed pericardiostomy with biopsy was performed. After pericardiostomy with drainage, echocardiographic features of constrictive physiology persisted and thus verified the presence of effusive constrictive pericarditis. Pathology showed chronic granulomatous inflammation with caseous necrosis consistent with tuberculosis (AFB stain: positive; Figure 2). Antituberculosis medication was started (isoniazid 400 mg, ethambutol 800 mg, rifampin 600 mg, pyrazinamide 1500 mg/d). Follow-up FDG positron emission tomography 12 months after administration of antituberculosis medication showed no visible uptake in the visceral and parietal pericardium, suggesting resolved inflammation (Figure 1, right panel).

Disclosures

None.

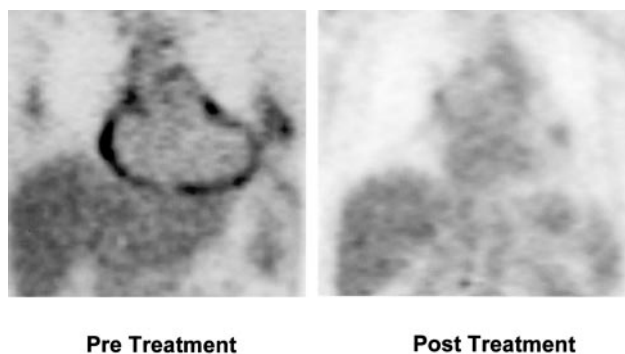


Figure 1. Left, PET scan showed homogenous uptake in the visceral and parietal pericardium, suggesting active inflammation. Right, Follow-up FDG positron emission tomography 12 months after administration of antituberculosis medication showed no visible uptake in the visceral and parietal pericardium, suggesting resolved inflammation.

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Circulation is available at <http://www.circulationaha.org>

DOI: 10.1161/CIRCULATIONAHA.105.554139

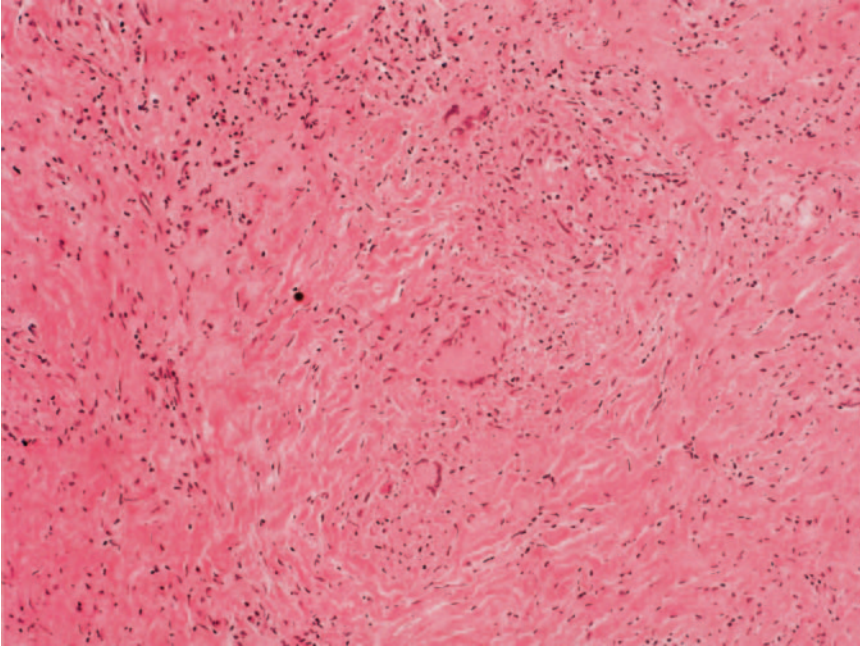


Figure 2. Pathology showed chronic granulomatous inflammation with caseous necrosis consistent with tuberculosis.