JACC: CARDIOVASCULAR IMAGING
VOL. 3, NO. 8, 2010

ISSN 1936-878X/$36.00
PUBLISHED BY ELSEVIER INC.

iMAIL

LETTERS TO THE EDITOR

Streptococcal Pharyngitis-Associated Myocarditis Mimicking Acute STEMI

Nonrheumatic streptococcal pharyngitis-associated myocarditis (SPAM) is infrequently reported, and its pathogenesis remains unclear. We report 8 young individuals (age 20 to 35 years, 7 men) who presented with SPAM mimicking acute ST-segment elevation myocardial infarction (STEMI) and characterized clinically with nonpleuritic chest pain, focal electrocardiographic (ECG) ST-segment elevation, and biochemical evidence of myocyte necrosis. In each case, coronary arteries were angiographically normal and cardiac magnetic resonance (CMR) imaging showed subepicardial late gadolinium enhancement (LGE). Seven patients had no prior history of cardiovascular disease, whereas 1 had an episode of acute SPAM 6 years prior to the recent presentation. All patients had evidence of recent streptococcal pharyngitis, within 3 to 7 days prior to presentation, and were treated with antibiotics; none satisfied the revised Jones criteria for diagnosis of acute rheumatic fever. Admission ECG showed focal (5 anterolateral, 3 inferior) ST-segment elevation (1 to 2 mm) that resolved over the course of the illness and was followed by T-wave abnormalities without development of Q waves. Transthoracic echocardiography showed regional (2 to 4 segments) left ventricular (LV) wall motion abnormality in all patients (wall motion index 1.12 to 1.24, mean 1.16 ± 0.06, using a 16-segment model); trivial-to-mild mitral regurgita-

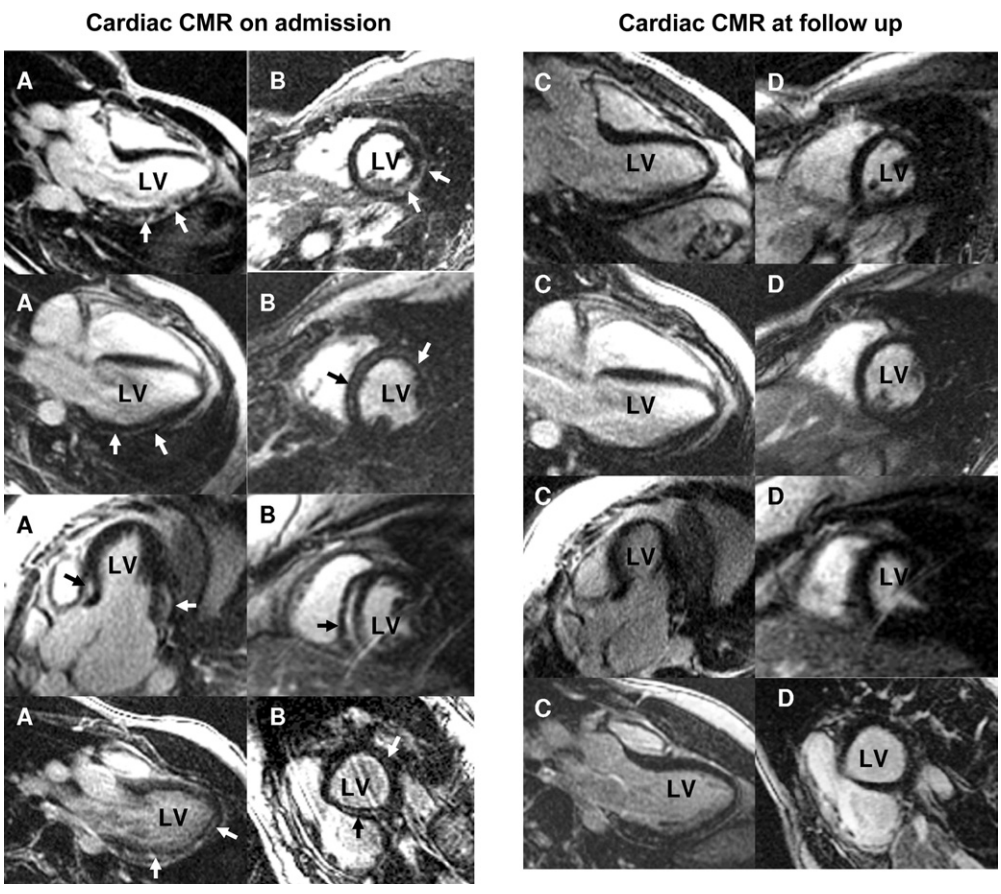

Figure 1. CMR Images of 4 Young Adults With SPAM Acutely and at Follow-Up

(A, B) Cardiac magnetic resonance (CMR) images of 4 young adults with acute streptococcal pharyngitis-associated myocarditis (SPAM) showing left ventricular (LV) long- **(A)** and short- **(B)** axis views obtained at index admission showing focal subepicardial late gadolinium enhancement **(bright signals, arrows)**. **(C, D)** Follow-up CMR images of the same patients as in **A and B** showing reduced number of segments with late gadolinium enhancement in left ventricular long- **(C)** and short- **(D)** axis views.

tion was present in 4 patients. Total serum creatine kinase (range 176 to 1,573 U/l, mean: 700 ± 449 U/l, normal <225 U/l), creatine kinase MB fraction (range 15.1 to 174 ng/ml, mean 61 ± 53 ng/ml, normal <9.0 ng/ml), and serum troponin-T (range 0.46 to 3.23 ng/ml, mean 1.401 ± 0.95 ng/ml, normal <0.1 ng/ml) levels were elevated in 7, 8, and 8 patients, respectively. Evidence for recent streptococcal infection was obtained from antistreptolysin O titer in 7 patients (range 392 to 2,364 IU/ml, mean 851 ± 745 IU/ml, normal <200 IU/ml), positive throat culture in 3 patients, and positive rapid streptococcal antigen test in 1 patient. Emergent invasive (n = 6) or computed tomographic (n = 1) angiography showed normal epicardial coronary arteries. Coronary angiography was not performed in a 20-year-old man with recurrent SPAM in whom normal coronary arteries had been demonstrated during a similar episode 6 years earlier. CMR was performed within 2 days of presentation in 7 patients. All patients showed a characteristic subepicardial LGE (3 to 8 segments, mean 5 ± 1.6 of 17 LV segments) (Figs. 1A and 1B); LV ejection fraction ranged from 40% to 64% (mean 53 ± 8%) and was mildly reduced in 4 patients. Resting myocardial perfusion was normal in all. Follow-up CMR was performed 12 ± 7 months after initial presentation in 6 patients. In 5 patients, LV systolic function improved, but in aggregate, LV function improvement was not statistically significant (LV ejection fraction: 61 ± 6 vs. 55 ± 5 at baseline, 95% confidence interval [CI]: −2.5 to 13.2; p = 0.14) and in all patients end-diastolic diameter showed a trend to improvement (50 ± 5 mm vs. 53 ± 5 mm at baseline, 95% CI: −6.3 to 0.3; p = 0.06). Follow-up CMR showed fewer LV segments with LGE (2 ± 1 vs. 5 ± 1.6 segment at baseline, 95% CI: −2.1 to −3.9; p = 0.0004) (Figs. 1C and 1D). Patients received antibiotics and nonsteroidal anti-inflammatory agents; beta-adrenergic blocking agents were given to 5 patients with LV systolic dysfunction. All patients showed clinical improvement during 2 to 3 (mean 2.4 ± 0.7) days of hospitalization and had complete resolution of clinical symptoms at follow-up. This report is limited by the fact that T2-weighted CMR imaging was not performed.

This report provides clinical, ECG, laboratory, coronary angiographic, and echocardiographic features of SPAM, wherein myocarditis was verified by CMR tissue characterization. Despite the small number of patients, the present report emphasizes several important clinical considerations. SPAM may not be as infrequent as previously recognized. It may remain a consideration in the differential diagnosis of acute STEMI, especially in young patients without traditional risk factors for coronary artery disease (1,2). Furthermore, this small series suggests that nonrheumatic presentation of SPAM may resolve upon antibiotic therapy. However, it is not clear whether chronic antibiotic prophylaxis would be necessary in patients who recover from SPAM, especially in those with frequent exposure to streptococcal infection. It is also not clear whether such an initial interaction is a prelude to development of rheumatic fever, especially since the binding of group A streptococci (GAS) to collagen has been proposed, similar to a well-conserved virulence trait of *Staphylococcus aureus*.

Rasoul Mokabberi, MD, Jamshid Shirani, MD, Afsaneh Haftbaradaran M., B. Dennis Go, MD, William Schiavone, DO*

*Geisinger Medical Center, Department of Cardiology, 100 North Academy Avenue, Danville, Pennsylvania 17822-2775.
E-mail: waschiavone@geisinger.edu

doi:10.1016/j.jcmg.2010.05.012

REFERENCES

1. Narula J, Khaw BA, Dec GW Jr., et al. Recognition of acute myocarditis masquerading as acute myocardial infarction. N Engl J Med 1993;328:100–4.
2. Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation 2004;109:1250–8.

Lipid-Rich Obstructive Coronary Lesions

Is Plaque Characterization Important?

We read with interest the high-risk nature of atherosclerotic plaques harboring circumferential necrotic cores (termed *napkin-ring* necrotic cores [1]), their possible association with optical coherence tomography-verified thin fibrous caps (2), and the likelihood of poor outcomes after coronary intervention (3). We, by using multimodality imaging, present the importance of clinical recognition of such plaques, especially if an intervention is planned.

An 85-year-old woman with hypertension and hyperlipidemia on pharmacologic therapy underwent an exercise stress test after reporting atypical chest pain. The stress test showed normal electrocardiographic response to exercise, normal ventricular function, and a small apical perfusion defect. The coronary computed tomography angiography (CTA) revealed a calcium score of zero and a significant stenosis in the mid-left anterior descending artery, subsequently confirmed by selective coronary angiography. CTA demonstrated a positively remodeled (PR) (remodeling index = 1.7), low attenuation (20 ± 10 Hounsfield units) plaque (LAP) (Fig. 1). Intravascular ultrasound demonstrated a minimal luminal area of 2.5 mm^2 and a plaque burden of 79% (Fig. 2A and 2B). Near-infrared spectroscopy (NIRS) (4) confirmed a circumferential lipid-rich lesion (Fig. 2C). The patient underwent percutaneous intervention with a drug-eluting stent that was complicated by transient no-reflow and a periprocedural myocardial infarction (peak troponin, 8.1 ng/ml) (Fig. 3). This case highlights the complementary findings suggestive of a high-risk coronary plaque obtained from different imaging modalities. PR and LAP on CTA, a minimal luminal area <4 mm^2 or >70% cross-sectional plaque burden, and napkin-ring lipid-rich lesion on NIRS usually associated with high-risk plaques also demonstrate the increased risk of distal embolization and no-reflow after intervention (5). The subsequent hospital course was uneventful, and the patient was discharged 3 days after the procedure. Now that we are able to define the composition of plaques by noninvasive and intravascular imaging modalities, it becomes necessary to design a clinical