

Lupus Myocarditis: Case Report

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Although gallium-67 (^{67}Ga) accumulates in both neoplastic and inflammatory tissues, indium-111 (^{111}In) labeled leukocytes are seen only in inflammatory cells. Indium-111-labeled leukocytes therefore are a useful agent in the noninvasive differentiation of inflammatory tissue from neoplastic tissue. This case is an interesting example of the use of ^{111}In -labeled leukocytes to make that differentiation.

CASE REPORT

A 39-yr-old white female was admitted for evaluation of valvular heart disease. She had been treated in the past for Liebman-Sachs endocarditis resulting in aortic, tricuspid, and mitral regurgitation. The patient had a history of progressive shortness of breath over the past 6–12 mo. She denied paroxysmal nocturnal dyspnea, orthopnea, or ankle edema. The patient described atypical chest discomfort not associated with exertion. She had a known history of systemic lupus erythematosus and had mild to moderate pericardial effusions diagnosed by echocardiography in the past. She had Raynaud's phenomenon and a rash with arthritis. She was diagnosed as having Hodgkin's disease in 1963 for which she had a regional nodal resection with local radiation. She underwent a left mastectomy in 1978 for breast cancer and received tangential beam radiation with a total dose of 2,400 Rads.

The patient had a 25 pack a year history of cigarette smoking. Pulmonary function tests demonstrated moderately severe obstructive changes. She denied alcohol abuse, diabetes, hypertension, myocardial infarction, or rheumatic fever.

On physical examination the blood pressure was 116/80, pulse 96, respirations 20. She was afebrile. An erythematous malar rash was present. There was no neck vein distention. Carotid pulses were equal bilaterally with radiation of bruits and normal upstroke. There was a grade II/IV harsh systolic ejection murmur along the left sternal border and a grade II/VI diastolic blowing murmur at the left sternal border. S4 was present and loudest at the apex. The lungs were clear. Extremities were without edema, cyanosis or clubbing and the distal pulses were intact. The electrocardiogram demonstrated sinus tachycardia, QRS axis $+65^\circ$, PR interval 0.16, heart rate 105 per minute with left ventricular hypertrophy. The heart size was borderline enlarged on chest x-ray with some left ventricular rounding and normal pulmonary vascularity (Fig. 1). The

vectorcardiogram showed left ventricular hypertrophy. Cine-CT demonstrated thickening of the pericardium with a small amount of fluid around the heart (Fig. 2). The end diastolic diameter of the left ventricle was increased. The resting ^{201}Tl myocardial perfusion scan demonstrated uniform myocardial perfusion. No defects were noted. There was persistent right ventricular activity in keeping with right ventricular pressure or volume overload (Fig. 3). A resting first-pass radionuclide angiogram in the 20° RAO projection demonstrated the right ventricular ejection fraction to be 44%. The left ventricular ejection fraction was 56% with a left ventricular end diastolic volume of 166 cc/beat, end diastolic volume index of 91.8 cc/beat/ m^2 , cardiac output 8.6 l/min, cardiac index 4.7 l/min/ m^2 . There were no segmental wall motion abnormalities identified.

An ^{111}In leukocyte scan was performed. Following the i.v. administration of 500 μCi of ^{111}In autologous labeled leukocytes, 24- and 48- hr whole body (Fig. 4A) and detailed mediastinal images (Fig. 4B) were obtained. The study demonstrated diffusely increased activity in the mediastinum (Fig. 4). Differential includes an inflammatory nonneoplastic process of the mediastinum. In addition, there was a solitary focus of increased isotope concentration in the right supraclavicular

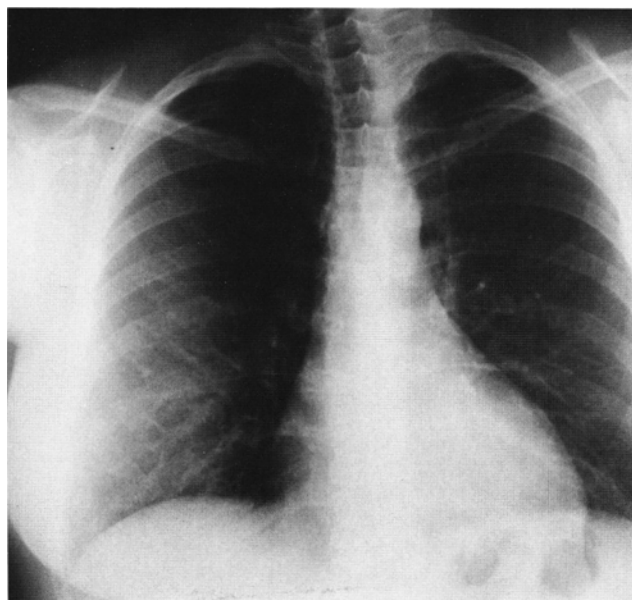


FIG. 1. The heart size was borderline enlarged on chest x-ray with some left ventricular rounding and normal pulmonary vascularity.

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FIG. 2. Cine-CT demonstrating thickening of the pericardium with small amount of fluid around the heart.

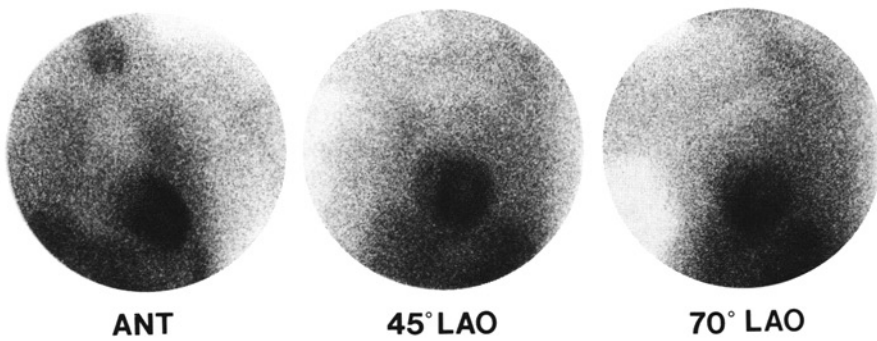


FIG. 3. Resting thallium study demonstrating abnormal right heart activity in keeping with right ventricular pressure or volume overload. There are no perfusion defects identified.

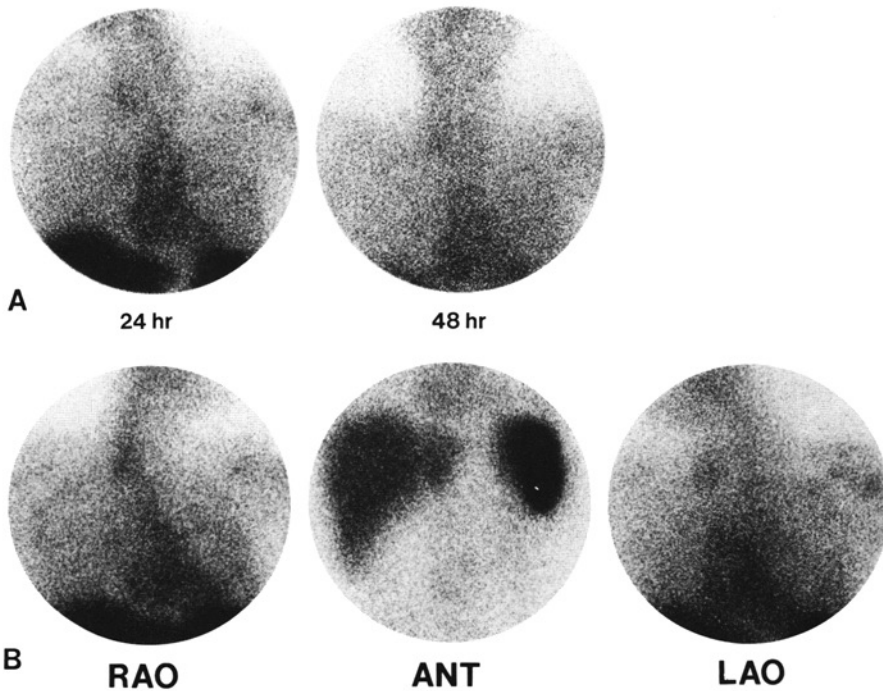


FIG. 4. Indium-111 leukocyte images. (A) 24- and 48-hr whole body images and (B) demonstration of diffuse increased activity in the mediastinum.

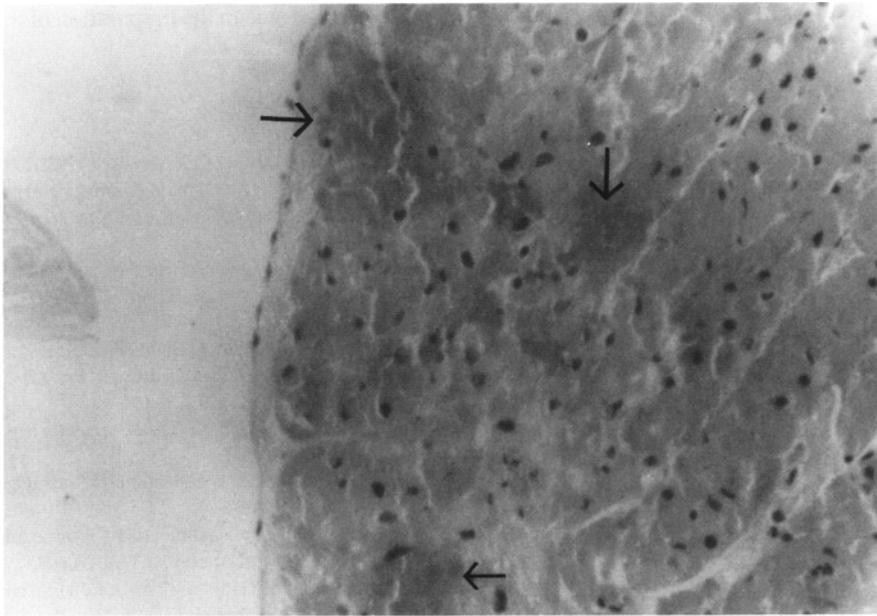


FIG. 5. H and E 10X—arrows indicate a smudged appearance of cytoplasm. A few polys are noted. These changes are secondary to blood infiltrate.

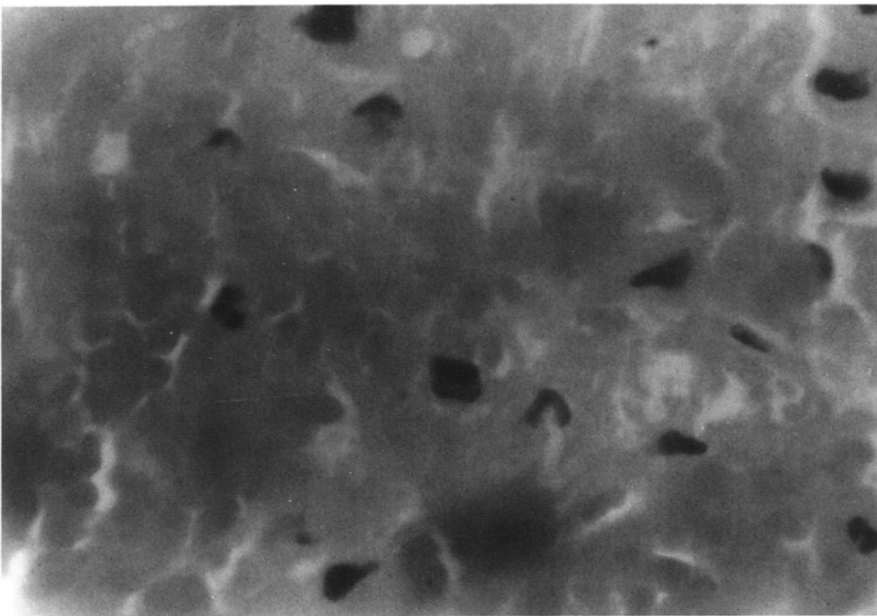


FIG. 6. H and L 40X high power view demonstrating myocytic necrosis with the invasion of polys and pyknosis.

region corresponding to the port of entry for the endomyocardial biopsy site. Examination of the remainder of the thorax was unremarkable. There was normal ^{111}In concentration in the liver and spleen. No abnormal foci were identified in the abdomen or pelvis.

The patient underwent a cardiac catheterization. There was moderate aortic regurgitation, mild mitral regurgitation, and moderate to significant tricuspid regurgitation. A minimal right coronary artery lesion was seen. The end diastolic pressures of the right ventricle and left ventricle were slightly increased without a constrictive pattern. Pulmonary artery pressures were elevated—mean = 18 mmHg, V wave = 25 mmHg. A right ventricular endomyocardial biopsy was performed

(Figs. 5 and 6). The endocardium was slightly thickened but on average was unremarkable. The largest of three fragments demonstrated broad focal changes characterized by the local extravasation of red blood cells resulting in a pressure degenerative process to myocytes as well as a ground-glass appearance with indistinct cellular borders. In addition, there was dissolution of the cytoplasm with marked vacuolization. There was absence of cross striations. A few polymorphonuclear leukocytes were present. There was marked increase in the nuclear cytoplasmic ratios. The above changes were consistent with idiopathic hypertrophic cardiomyopathy and systemic lupus erythematosus but not diagnostic of the latter. The patient developed a pericardial rub most likely as a result of

diminution in pericardial fluid, but surgical intervention was not warranted at this time. The patient was discharged on nonsteroidal anti-inflammatory medication and diuretics.

DISCUSSION

Gallium-67 is known to accumulate in both inflammatory and neoplastic cells. Lin et al. (1) provided a gamut of known causes of ⁶⁷Ga cardiac uptake. These causes include tuberculous (2), purulent (3) or acute nonspecific (4) pericarditis, myocarditis (5), inflammatory cardiomyopathy (6,7), bacterial endocarditis (2,8) malignant neoplastic involvement of the pericardium and/or myocardium (2,9,10), sarcoidosis (11), acute myocardial infarction (2, 12), myocardial abscess (2,13), luetic myocarditis (14), Kawasaki disease (2,15), and hypersensitivity angitis (5).

Increased ¹¹¹In leukocyte concentration within the cardiac silhouette may occur in silent myocardial infarction but acute infarcts are often missed (16,17). Increased uptake is also seen in myocarditis. Indium-111 images are not usually positive in cases of subacute bacterial endocarditis because the lesions are too small to be detected (18). The images may be improved with the use of tomographic scanning. The positive ¹¹¹In study in this case suggested an inflammatory nonneoplastic process involving the myocardium. This suspicion was confirmed by the endomyocardial biopsy which was consistent with idiopathic hypertrophic cardiomyopathy and systemic lupus erythematosus. The addition of ¹¹¹In labeled-leukocyte scanning was helpful in narrowing the differential diagnosis in this patient who had both neoplastic and inflammatory possibilities in the differential diagnosis of the pericardial and myocardial disease.

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