GALLIUM-67 CITRATE SCINTIGRAPHY IN IDIOPATHIC PERICARDITIS

— Report of a Case —

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We report a case of idiopathic pericarditis in a 71-year-old woman without a friction rub or electrocardiographic changes suggestive of pericardial inflammation. She noticed dyspnea and palpitation on exertion about 40 days before admission. After admission, pericardial effusion and inflammatory reactions, such as elevated C-reactive protein, were found. Moreover, gallium-67 citrate scintigraphy revealed abnormal isotope accumulation over the cardiac silhouette. Therefore, she was diagnosed as having “active” pericarditis. The finding with the gallium scan became negative with steroid therapy. In this case, a gallium scan was very useful in assessing and monitoring the course of pericardial inflammation. (Jpn Circ J 1994; 58: 298–302)

IDIOPATHIC pericarditis is caused by pericardial inflammation of unknown etiology and is characterized by chest pain, a pericardial friction rub and serial electrocardiographic abnormalities! Pericardial effusion, which is usually detected by echocardiography? is also common. In a patient with idiopathic pericarditis who has these clinical features, other laboratory examinations may not be indicated. However, in a patient without characteristic manifestations strongly suggestive of pericardial inflammation, even with pericardial effusion, it is often difficult to decide whether specific treatments or further examinations including pericardiocentesis are required. Therefore, the available noninvasive techniques for demonstrating the activity of pericardial inflammation may be necessary in such a case. We report the usefulness of gallium-67 citrate, inflammation-avid isotope, scintigraphy for diagnosis and follow-up of idiopathic pericarditis in a patient with atypical clinical features of the disease.

CASE REPORT

A 71-year-old woman with a 5-year history of hypertension and angina pectoris was admitted to our hospital on September 7, 1992. Without a history of antecedent upper respiratory infection, she noticed palpitation and dyspnea on exertion approximately 40 days before admission. On admission, her body temperature was 36.6°C, systemic blood pressure 162/90 mmHg and pulse 120/min with regular rhythm. On auscultation, there was a systolic ejection murmur of grade 3/6 with a maximum point at the apex. Moist rales were audible over both lower lung fields. The liver was palpable at 2 and a half finger-breathths in the epigastric

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region. Mild central cyanosis and anemia were recognized. There was no jaundice or edema. The chest roentgenogram revealed bilateral pleural effusion and no pulmonary congestion (Fig. 1a). The electrocardiogram showed sinus tachycardia with a heart rate of 121/min (Fig 2; left). Blood tests on admission disclosed the following abnormal values: red blood cell count, 327×10^4/mm³; Hemoglobin, 9.5 g/dl; Hematocrit, 31.5%; C-reac-

Fig. 1 On chest roentgenography, bilateral pleural effusion was seen on admission (a), and decreased markedly on the 5th day after admission (b). Cardiomegaly with a cardio-thoracic ratio of 64% was also seen on day 5.

Fig. 2 Electrocardiogram recorded on admission (left) and on the 44th day after admission (right). Neither showed serial ST-T changes which might strongly suggest pericardial inflammation.

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tive protein (CRP), 5.7 mg/dl; and erythrocyte sedimentation rate (ESR), 26 mm/h. Cardiac enzymes such as creatinine phosphokinase and lactate dehydrogenase were normal. An arterial blood gas analysis on room air revealed a pH of 7.47, a PaO₂ of 49.4 mmHg, and a PaCO₂ of 34.5 mmHg. The echocardiogram revealed pericardial effusion approximately 10 mm wide behind the posterior left ventricular wall at the end-diastole. There were no echocardiographic signs of cardiac tamponade.

Pleurocentesis did not contribute to the diagnosis and pericardiocentesis was not performed because of the absence of cardiac tamponade. On the 5th day after admission, pleural effusion decreased markedly (Fig.1b) and PaO₂ in room air rose to 72.5 mmHg. CRP and ESR also showed a tendency to decrease, but the amount of pericardial effusion detected by 2-dimensional echocardiography remained unchanged. Moreover, a gallium-67 scintigraphic study performed on the 8th day after admission showed abnormal uptake by the heart (Fig.3a,b). The cause of pericarditis was not revealed, although several diagnostic tests, including seral neutralizing antibody titers for enteroviruses and Adenovirus, tuberculin skin test, antinuclear antibody titer, rheumatoid factor and computed tomogram of the chest and abdomen, were performed. Therefore, she was diagnosed her as having “active” idiopathic pericarditis. The patient was treated with 30 mg/day prednisolone for 14 days. Thereafter, laboratory signs of inflammation abated and pericardial effusion disappeared. Moreover, gallium-67 citrate uptake over the cardiac silhouette disappeared (Fig.3c). However, the electrocardiogram showed no significant changes during hospitalization, except for a decrease in heart rate and a rise in QRS voltage (Fig.2). Currently, recurrence of pericarditis or development of constrictive pericarditis is not evident.

DISCUSSION

Our patient noticed dyspnea and palpitation, which are occasionally chief complaints

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of patients with acute pericarditis, but felt no chest pain. Moreover, no friction rub was heard and no diagnostic electrocardiographic abnormalities were found. We thought that her symptoms were related primarily to respiratory failure due to associated pleurisy because she became symptom-free after pleural effusion disappeared, and that the reason for the lack of characteristic findings was in part because she visited our hospital about 40 days after the onset of the symptoms. In addition, Ueda and Sugiura reported that chest pain compatible with pericardial pain or typical electrocardiographic changes in acute pericarditis were less common in elderly patients than in younger cases.

In patients who are definitely diagnosed as having pericarditis, echocardiography is a very effective laboratory technique for detecting pericardial fluid, complication of myocarditis, and development of cardiac tamponade or constrictive pericarditis. However, echocardiography does not usually record the nature of pericardial fluid, i.e., whether the inflammation of the pericardium is active. In addition, this procedure sometimes discloses chronic pericardial effusion of unknown origin incidentally in asymptomatic patients, and in such cases no specific treatment is often required. On the other hand, unresolved pericardial inflammation may occur and result in persistence of pericardial effusion or constrictive pericarditis, even though idiopathic pericarditis is usually a self-limited illness which lasts only a few weeks! Other laboratory findings, such as elevation of CRP and leukocytosis, are suggestive of inflammation but not are diagnostic of pericarditis. Therefore, a noninvasive technique for detecting pericardial inflammation may be necessary, particularly in patients without characteristic manifestations and with pericardial effusion, such as the patient described here. In this instance, gallium-67 citrate scintigraphy was very useful, with the aid of echocardiography, in assessing a pericardial inflammatory activity and in monitoring the response to steroid therapy.

Initially described as a bone-scanning agent, gallium is recognized as an effective noninvasive localizer of tumors and of either acute or chronic inflammatory regions. It has also been used successfully to image heart diseases including pericarditis. However, its sensitivity and specificity when scanning for idiopathic pericarditis have not been clearly established. Moreover, it is unclear when such scanning should be used. Based on the results of the present study, gallium-67 citrate scanning was considered to be an effective modality of diagnosis and follow-up of pericardial inflammation even when the patient revealed atypical clinical course of the disease. In addition, we would recommend that gallium scan should be applied in such cases.

Histopathologically, idiopathic pericarditis causes inflammation of the visceral and parietal pericardial membranes with infiltration first of polymorphonuclear leukocytes and then of lymphocytes around small vessels! Fibrin is deposited in the pericardial space. The inflammation may result in a serous, serofibrous, supplicative, or hemorrhagic effusion with a predominance of lymphocytes. There is still no general agreement on the exact mechanism of gallium-67 localization in inflammatory lesions. However, it has been speculated that the accumulation of gallium in inflammatory lesions is the result of fixation on leukocytic membrane! lysosomes, lactoferrin and transferrin which are present in high concentrations in inflammatory lesions. It has been proposed that hyperpermeability due to inflammation also influences gallium-67 uptake.

Indium-111-oxime-labeled leukocytes or technetium-99m-hexamethylpropylene-amine oxide-labeled leukocytes has also been used as inflammation-avid isotopes. Scintigraphy by these methods is useful for localization of infection under conditions in which the foci of infection attract leukocytes via chemotaxis. Since most of the cells that are labeled in their scannings are usually neutrophils, inflammatory conditions in which the predominant cellular response is other than neutrophilic, such as idiopathic pericarditis, particularly with sustained continuous clinical course, may result in false negative studies.

In summary, we found that gallium-67 citrate scanning might be useful in estimating the activity and the extent of idiopathic pericarditis, particularly in a patient with a rather atypical clinical course. The result
obtained by such scanning may lead to specific therapies or further examinations.

REFERENCES


