

Endoscopic pericardial fenestration for a patient with sustained lupus pericarditis

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ABSTRACT

A 57-year-old woman was diagnosed in January 1982 with SLE based on ANA 1:640, positive LE cell preparation, proteinuria (3+), and pericarditis. In 1984, 1994, and 1997, the pericardial effusion was noted to have increased without signs of disease exacerbation or cardiac tamponade, and pericardial drainage was repeated to control the effusion. A massive pericardial effusion developed in August 1997. After tuberculosis, hypothyroidism, neoplasm, and progression of SLE were ruled out, we decided to perform pericardial fenestration. A safe and minimally invasive pericardial fenestration was successfully completed endoscopically. Pathologic study of the specimen revealed chronic pericarditis. We consider endoscopic pericardial fenestration to be useful for at risk patients with pericarditis to control the effusion and establish a differential diagnosis.

Introduction

Patients with systemic lupus erythematosus (SLE) can develop pericarditis, and the prevalence has been shown to be between 20% and 50% (1). Because corticosteroids suppress pericardial effusion, the effusion seen in such patients rarely becomes a problem; i.e., it rarely leads to cardiac tamponade (2-5). Even when disease activity fails to be controlled, pericarditis appears to improve without additional treatment in the vast majority of SLE patients. However, there remain some who develop massive pericardial effusion necessitating pericardial drainage or the use of immunosuppressive agents (2-5).

Progress in endoscopic treatment approaches led us to perform various endoscopic procedures safely and with minimal invasiveness. We here report an SLE patient with sustained/massive pericardial effusion that was finally resolved by endoscopic pericardial fenestration.

Case report

A 57-year-old woman had complained of eye-lid edema in January 1982 and was diagnosed with SLE. Her ANA titer was 640 dil. and her LE cell prepara-

tion was positive. She showed proteinuria (3+) and pericarditis. She began to receive methylprednisolone 48 mg/day with sufficient outcome except for control of the pericardial effusion. As she never complained of signs of cardiac tamponade despite a large amount of pericardial effusion, further treatments were withheld. In 1984, 1994, and 1997, pericardial effusions were noted to have increased without signs of disease exacerbation. At these times, the effusions were controlled by pericardial puncture or drainage.

A massive pericardial effusion developed in August 1997, and the patient was admitted to our hospital to control the effusion (Fig. 1). Initially, pericardial drainage was performed with minimum improvement, whereas some further disease activity was suggested: CH50 21 U/mL, anti-DNA 17 IU/mL, ESR 55 mm/hr, and fever. Tuberculous pericarditis was ruled out; i.e., culture and detection of DNA using PCR in the pericardial fluids were negative. Pathologic findings of the fluids were incompatible with neoplastic proliferation. Laboratory findings failed to reveal hypothyroidism.

Cyclophosphamide (CYC:75 mg/day) was prescribed to ameliorate disease activity and to control the effusion. This treatment improved the hypocomplementemia, but the sustained pericardial effusions remained uninfluenced. After discussion with the patient, we finally decided to perform pericardial fenestration endoscopically, and the operation was carried out in November. The clinical course thereafter was uneventful, and the patient was discharged four weeks later. The pericardial effusion rapidly decreased, although pleural effusions developed transiently thereafter, probably due to leakage from the pericardium. The pleural effusion improved slowly and had almost disappeared without additional effusions in the pericardium by October 1998. Pathologic findings of the pericardial specimen were compatible with chronic inflammation without granuloma, vasculitis, or neoplastic proliferations.

Discussion

We report here the first case of an SLE patient with massive/sustained pericar-



Fig. 1. Chest x-ray taken in August 1997.



Fig. 2. Chest x-ray taken in October 1998.

dial effusion who underwent endoscopic pericardial fenestration. Pericardial fenestration is useful for the differential diagnosis and for controlling massive pericardial effusions (6, 7). However, the procedure has been performed only in a limited number of cases, because the procedure is considered to be risky for immunocompromised patients such as

those with SLE or other immunologic disorders. Thus, pericardial fenestration is usually reserved for patients with malignant pericardial effusion or thoracic trauma in order to ameliorate the life-threatening tamponade (7, 8). Progress in endoscopic treatments has accelerated in the last decade. As a result, even radical surgery for lung can-

cer is being done endoscopically around the world, especially in aged patients. This minimally invasive treatment is now being applied to various diseases of the thorax. We attempted, with success, to treat our SLE patient with endoscopic pericardial fenestration in the interests of minimal invasion. As a result, we were able to diagnose chronic pericarditis, differentiating it from tuberculous pericarditis, vasculitis, and neoplasm. We thus avoided the excessive use of corticosteroids against the possible exacerbation of SLE activity. Endoscopic pericardial fenestration was useful to establish the diagnosis and to control the massive pericardial effusions in this case. We believe that pericardial fenestration should be performed endoscopically in more patients with massive pericardial effusion, especially in the elderly or those with rheumatic diseases, cardiovascular diseases, diabetes or other high risk conditions.

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