Case Reports

Cardiac Tamponade in Rheumatoid Arthritis

Successful Treatment with Intrapericardial Steroid Administration

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SUMMARY

Cardiac tamponade complicated by classic rheumatoid arthritis was markedly alleviated by pericardiocentesis and intrapericardial administration of steroid. For the following 2 years, no recurrence of cardiac tamponade or constrictive pericarditis was observed. According to the literature, only one other patient with cardiac tamponade complicating rheumatoid arthritis (RA) had been successfully treated patient by intrapericardial steroid administration.

Additional Indexing Words:

Rheumatoid arthritis Cardiac tamponade Steroid therapy

PERICARDITIS, a frequent complication of RA observed in 30–50% of patients at autopsy,¹⁾ is rarely regarded as a major clinical problem since most therapeutic attention is diverted from this seldom symptomatic disorder²⁾ to more common symptoms of RA such as severe arthralgia. However, this condition may in rare instances lead to cardiac tamponade and constrictive pericarditis. Although cardiac tamponade complicating RA has been commonly treated by epicardiectomy, the condition was markedly alleviated in 1 patient by pericardial drainage and intrapericardial steroid treatment.

CASE REPORT

The patient was a 24-year-old male working as a clerk. In 1967, when

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he was 9 years old, the patient presented with arthralgia of both knees, and was treated by a local physician under a diagnosis of juvenile rheumatoid arthritis. However, the arthralgia then spread symmetrically to the elbow, hand and foot joints, accompanied by fever, limitation of movement and morning stiffness. With the recurrence of fever and arthralgia in May 1982, the patient was admitted to Saiseikai Matsuzaka Hospital for close examination.

On admission, the patient was 168 cm tall and weighed 50 kg. The blood pressure was 125/82 mmHg and the pulse was regular at 120 beats/min. The temperature was 39.0°C. A Levine II degree ejection murmur was heard at the left margin of the sternum over the fourth intercostal space, but pericardial friction rubs were absent. Dilatation of the jugular veins was not observed. Fusiform deformity of the finger joints, a rheumatoid nodule on the ulnar side of the left forearm, and tenderness and swelling of the elbow, knee and foot joints were noted.

The CTR was 46% on chest X-ray, white blood cell count was 17,100/mm³, CRP was 6+, serum gamma-globulin was 27.5% and rheumatoid factors were positive. Anti-DNA antibody and anti-nuclear antibody were, however, negative.

As fever persisted and arthralgia increased in spite of oral indomethacin, prednisolone (40 mg/day) was initiated on the 14th hospital day. The CTR showed a gradual increase to 67%, and dyspnea and dilatation of the jugular veins appeared on the 21st hospital day. Paradoxical pulses (27 mmHg) (Fig. 1) were observed and a diagnosis of cardiac tamponade was made. Pericardiocentesis was immediately performed, draining 700 ml of pericardial effusion, and prednisolone (20 mg) was then administered into the pericardial cavity. As determined by cardiac catheterization, arterial pressure improved

Fig. 1. Brachial artery pressure tracing prior to pericardiocentesis shows a paradoxical pulse of 27 mmHg.

T.N. 24y.o. male

Fig. 2. M-mode echocardiogram before and after pericardiocentesis. upper: before, middle: immediately after, lower: 9 days after pericardiocentesis. PE=pericardial effusion; AO=aorta; IVS=interventricular septum; LV=left ventricle; LA=left atrium.

from 95/58 mmHg before drainage to 110/52 mmHg after drainage, pulmonary artery pressure improved from 31/19 to 21/10 mmHg, right atrial pressure from 12 to 6 mmHg and cardiac index from 2.6 to 4.5 L/min/m². Paradoxical pulses disappeared. The pericardial effusion was yellowish and contained 4.6 g/dl of protein, 18 mg/dl of glucose and 4,740 U/dl of LDH; RA was negative, gamma-globulin was 42% and the culture was negative for bacteria. The CTR decreased from 74% before drainage to 62% after drainage. Improvements in electrocardiogram were observed at low voltages. B mode echocardiography showed a pendulum motion, and M mode (Fig. 2) showed a marked decrease in the pericardial effusion after drainage. The effusion completely disappeared 9 days after the procedure. Prednisolone was reduced and then discontinued after about 1 month. No recurrence of pericarditis or reaccumulation of pericardial effusion has been observed thereafter, and the patient has remained free of constrictive pericarditis for 2 years.

DISCUSSION

The present patient, showing morning stiffness, pain on motion of several joints, tenderness, symmetric joint swelling, radiographic abnormalities and positive RA factors, fulfilled 6 items of the diagnostic criteria for RA of the American Rheumatology Association. The incidence of pericarditis in RA is reported to be 11–50% by autopsy^{3),4)} but less than 10% by clinical observation.⁵⁾ This wide discrepancy may be due to the scarcity of clinical signs and symptoms of this disorder as well as the difficulty in detecting it by conventional chest X-ray studies or electrocardiography. In fact, Bacon⁶⁾ and Bernstein et al⁷⁾ observed accumulation of pericardial effusion in 50% and 36% of their patients, respectively, and these frequencies are nearly comparable to those reported in autopsy studies. According to Berger,⁸⁾ pericarditis complicating RA is usually either transient or readily manageable by steroid therapy. However, cardiac tamponade or constrictive pericarditis may develop secondary to it in a limited number of patients, most commonly those with typical RA.¹⁾ The incidence of cardiac tamponade complicating RA is very low, but the condition is fatal in 90% of cases without proper treatment.⁹⁾

Several treatments^{10)–12),14)} have been proposed for cardiac tamponade complicating RA. Thadani et al⁹⁾ considered drainage to be difficult due to the thickness of the pericardial fluid and the frequent follicular formation, and suggested pericardiectomy as the best treatment. Majeed et al,¹³⁾ however, advocated pericardiocentesis as the first treatment to be attempted, contending that the difficulties suggested by Thadani et al can be overcome by using a larger aspiration needle. Although these authors failed to provide detailed descriptions of the condition of their patients, the approaches of both of these authors are considered to be appropriate by current standards. The patients of Thadani et al showed thick pericardial fluid and follicular formation, and the symptoms were not relieved by pericardiocentesis. Furthermore, thickening of the pericardium was noted at surgery. These observations suggest that they had effusive constrictive pericarditis¹⁶⁾ showing characteristics of both cardiac tamponade and constrictive pericarditis. The patient of Majeed et al,¹³⁾ on the other hand, is considered to have had typical cardiac tamponade since pericardiocentesis promptly relieved symptoms. At present, differentiation of these two conditions and selection of appropriate treatments are possible by careful evaluation of echocardiograms and cardiac catheterization studies. Majeed et al¹³⁾ stated that systemic administration of steroid was effective, but the steroid therapy initiated in our patient 7 days before the onset of cardiac tamponade was of no prophylactic value. Scharf et al¹⁵⁾

reported dramatic clinical improvement after pericardiocentesis and intrapericardial administration of methylprednisolone acetate (80 mg) as in our patients. Although intrapericardial steroid administration has not been established as a treatment of cardiac tamponade, it was markedly effective in this patient as well as in ours.

The relative value of various treatments of cardiac tamponade complicating RA has yet to be established, partly due to the rarity of the condition. However, treatment consisting of pericardiocentesis and local steroid administration, as in this case, is less invasive and of greater benefit than surgical approaches.

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