Cardiac tamponade in rheumatoid arthritis

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Summary

A 56-year-old woman with definite rheumatoid arthritis developed cardiac tamponade while her disease was both clinically and serologically inactive. To our knowledge such a complication has been reported only once previously.

KEY WORDS: pleural effusion, prednisolone, echocardiography.

Introduction

Although 40-50% of patients with rheumatoid arthritis show evidence of pericarditis on post-mortem examination or by means of echocardiography, a very low incidence of pericarditis can be detected clinically (Bonfiglio and Atwater, 1969; Franco, Levine and Hall, 1972; Thadani, Iveson and Wright, 1975; Bacon and Gibson, 1972).

We recently saw a patient with definite rheumatoid arthritis in whom cardiac tamponade suddenly developed when her disease was clinically inactive and serologically negative. To our knowledge a serious pericardial complication with inactive rheumatic disease has been described only once previously (Grivaux *et al.*, 1979).

Case report

A 56-year-old woman came with a 1-year history of arthralgia with swelling and morning stiffness in both wrists and metacarpophalangeal joints. Physical examination showed swelling in both wrists and metacarpophalangeal joints with motion tenderness affecting all of them. Blood pressure was 140/80 mmHg. Investigations: erythrocyte sedimentation rate (ESR) 48 mm/hr (Westergren method). Latex test was ++, Waaler-Rose 1/160 and antinuclear

antibodies were positive to a dilution of 1/640 with speckled pattern. The LE phenomenon and anti-DNA antibodies (*Chritidia lucillae* test) were negative. Radiological examination of the chest was normal. In both hands, subcondral osteoporosis, soft tissue swelling and cortical erosions were detected.

One month after the diagnosis of definite rheumatoid arthritis, stiffness, swelling and tenderness developed in the left ankle. Chrysotherapy was begun and this treatment was continued until a dosage of 1 g was reached, with clinical remission and improvement in the laboratory tests 1 month before the end of the course.

Ten months after diagnosis and 2½ months after the course of chrysotherapy she became suddenly dyspnoeic. On examination blood pressure was 80/55 mmHg, respirations 36/min and heart rate 120/min. Jugular veins were full without discernible pulse at 45°. Neither Kussmaul's sign nor pulsus paradoxus were observed. Cardiac examination showed diminished heart sounds without murmurs or rubs. A chest X-ray (Fig. 1) revealed right pleural effusion and a huge cardiac silhouette. The electrocardiogram showed sinus tachycardia and diminished amplitude with non-specific ST-T changes. Central venous pressure was 24 cm H₂O. An echocardiogram revealed grade IV pericardial effusion.

Pericardiocentesis yielded 600 ml of serofibrinous fluid and the patient improved rapidly. A drainage tube was left in the pericardial cavity obtaining 1200 ml of fluid in 48 hr. Cardiomegaly and echocardiographic abnormalities disappeared and the patient became asymptomatic. Investigations showed normochromic normocytic anaemia with haemoglobin of 11.2 g/dl, with normal ESR and negative latex test; the pericardial fluid showed $55 \times 10^6/1$ leucocytes with 89% granulocytes, a glucose level below 10 mg/dl,

FIG. 1. Chest X-ray revealing huge cardiomegaly and right pleural effusion.

negative latex and Waaler-Rose tests, absence of acid-fast bacilli and negative Gram stain. Cultures were negative and cytological examination did not show abnormal cells. After general and intrapericardial methylprednisolone therapy during the acute episode, prednisone (60 mg/day p.o.) was administered for 2 weeks. Tapering of the dose was then begun as the patient remained asymptomatic. Five months after the pericardiocentesis she remains asymptomatic both from the articular and pericardial disease on a prednisone dosage of 5 mg/day. Rheumatoid factor is negative and ESR and gamma-globulin level are within normal range but anti-nuclear antibodies are still positive at 1/40 dilution.

Discussion

Pericardial involvement has been detected in a high percentage of rheumatoid patients in necropsy and echocardiographic studies, but neither cardiac tamponade nor constrictive pericarditis are common complications of the disease (Nomeir *et al.*, 1973). The rapid development of serious pericardial disease, as in our case, is even less frequent (John, Hough and Sargent, 1979). When the pericardium is affected clinically in the course of rheumatoid arthritis, rheumatoid factor is positive in 95% of the cases and

subcutaneous nodules are found in 35% of the patients (Franco *et al.*, 1972). In our case, however, subcutaneous nodules were never present and when the cardiac tamponade developed her rheumatoid factor was negative and the disease was clinically inactive, a situation not previously reported to our knowledge although Grivaux *et al.* (1979) described a patient with constrictive pericarditis in inactive rheumatoid disease. Although the probability was thus high that the exudative pericarditis was independent of her rheumatoid arthritis, the characteristics of the pericardial exudate were highly suggestive of a rheumatoid effusion: pleocytosis of neutrophils, an extremely low glucose level and negative microbiological and cytological examinations. We are not aware of follow-up studies on pericardial rheumatoid disease; our patient has done well with corticosteroids but the ultimate prognosis of this complication remains to be established.

This case stresses the systemic nature of the rheumatoid disease and the possibility of the appearance of organ involvement even when the process seems to be inactive.

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