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Case report

Constrictive pericarditis, pyopericardium, and tamponade with rheumatoid arthritis

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SUMMARY Constrictive pericarditis and pyopericardium with tamponade in a patient with rheumatoid arthritis is described. No fever was recorded despite a litre of pus in the pericardial sac, perhaps because of previous treatment with hydrocortisone. Echocardiography is a diagnostic advance.

Pericarditis is the commonest cardiac manifestation of rheumatoid arthritis. It may present as 'dry' pericarditis with a friction rub and only rarely with tamponade (Kirk and Cosh, 1969). Pyopericardium is extremely rare, and we can find no previous account of pyopericardium with tamponade in association with rheumatoid arthritis.

Case report

A 53-year-old man presented with a history of progressive shortness of breath and oedema from the ankles to the umbilicus. A diagnosis of rheumatoid arthritis had been made 20 years previously. Proximal interphalangeal, metacarpalphalangeal, wrist, ankle, and knee joints were involved. The Rose-Waaler test was positive, with negative tests for LE cells and antinuclear factor. A synovectomy had been performed on the right knee in 1972, and in the past year he had had several intra-articular injections of hydrocortisone, the last injection into the left knee 6 weeks before admission. He was also treated with aspirin and indomethacin.

Despite his disability he had worked as a tree feller until serious breathlessness developed 5 months before his attendance at this hospital.

On examination he had gross oedema of the legs, ascites, and bilateral pleural effusions, and the jugular venous pressure was raised above the angle of the jaw. The heart sounds were inaudible, the rate 120 per minute in sinus rhythm, with intermittent supraventricular tachycardia. Systolic blood pressure was 100 mmHg, and 40 mm of arterial systolic paradox could be demonstrated at the brachial pulse. He was, and remained, apyrexial.

The pericardial effusion was demonstrated by echocardiography (Fig. 1), and right heart catheterisation with angiography confirmed the clinical diagnosis of constriction and showed tamponade (Fig. 2). The pericardium was aspirated by the subxiphisternal approach. A small amount of pus was withdrawn. Gram stain and culture showed the presence of Staphylococcus aureus.

The patient was treated with frusemide and cloxacillin, and 24 hours later emergency surgery was undertaken on account of his deteriorating condition.

At operation both the right and left atrial pressures were 30 mmHg. The parietal pericardium was very tense, and when it was opened 1 litre of thick creamy pus was removed. The parietal pleura was not attached to the heart and was almost completely removed. The visceral pericardium was white, thickened, and intimately adherent to the ventricles, causing constriction. This layer was removed, with release of both ventricles; the venous pressure and left atrial pressure fell dramatically to normal.

Early recovery from the operation was satisfactory, with a stable cardiovascular system, but on the sixth day the patient collapsed and died of a massive pulmonary embolism, which was proved at necropsy. Histological examination of the pericardium showed marked fibrous thickening. It was lined with an inflammatory exudate, which was becoming organised. There was no evidence of tuberculosis or calcification.

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Discussion

The reported complications of rheumatoid arthritis include pericarditis, aortic incompetence, heart block, arteritis involving the small vessels, and rarely myocarditis (Cosh, 1972). The diagnosis of pericarditis is infrequently made in adults. However, when Kirk and Cosh (1969) examined 100 consecutive patients with rheumatoid arthritis specifically for this complication, it was found in 10. The overall incidence from necropsy studies in 8 major series was 30% (Kirk and Cosh, 1969). The most usual finding was adherent or obliterative pericarditis. Bacon and Gibson (1972) detected pericardial effusions by echocardiography in as many as 50% of patients with chronic nodular rheumatoid arthritis. Despite this figure clinical presentation with pericardial constriction or effusion with tamponade is rare. Constriction usually develops within 5 years of the onset of joint disease, but 1 of the patients reported by Harrold (1968) had a 28-year history, and our patient had a 20-year history of rheumatoid arthritis.

Pyopericardium has not been reported in association with constrictive pericarditis due to rheumatoid arthritis. It was of particular interest that no site of entry of infection was found and that, throughout his illness and despite the presence of 11

Fig. 1 Echocardiogram demonstrating the pericardial effusion.

Fig. 2 Angiogram showing the increased distance between the right border of the cardiopericardial silhouette and contrast medium in the right atrium confirming effusion. The outward concave contour of the contrast medium suggests tamponade.
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of pus in the pericardial sac, the patient remained afebrile. It may be relevant that he had recently had an intra-articular injection of hydrocortisone. Other reported causes of pyopericardium are secondary infection from pneumococcal pneumonia or staphylococcal osteomyelitis and septicaemia. These usually occur in children.

It seems that the diagnosis of constrictive pericarditis should be considered in any patient with rheumatoid arthritis who develops severe cardiac failure. The clue is the finding of pulse paradoxus. This case illustrates the coexistence of pyopericardium with cardiac tamponade and illustrates the use of echocardiography in this situation.

Surgery is the treatment for this condition. It is essential not only that the tamponade is cured by the drainage of pus, but that the diseased visceral pericardium is removed to relieve ventricular constriction.

References


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