Case report

Cardiac tamponade in acute rheumatic carditis

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SUMMARY In patients with valvular heart disease, fever, and cardiomegaly echocardiography is an invaluable noninvasive tool. In this report we describe a young female presenting with cardiac tamponade due to acute rheumatic carditis. Echocardiography showed an exudative pericardial effusion which was haemorrhagic on pericardiocentesis. She responded to steroid therapy with resolution of carditis and pericardial effusion.

Case report

A 12-year-old Malay schoolgirl was admitted to another hospital with a 3-week history of fever, sore throat, transient arthritis of the right knee, and progressive exertional dyspnoea. There were no previous major medical illnesses of note.

On admission the patient was pale, orthopnoeic, and febrile at 39°C. The respiratory rate was 30 per minute and the heart rate was 120 per minute. Blood pressure was 120/70 mmHg. On sitting, the jugular veins were distended up to the angle of the jaw. She had cardiomegaly, mitral and aortic regurgitation, and a pericardial rub. The liver and spleen were enlarged.

Initial investigations revealed a haemoglobin of 6.2 g/dl and a white cell count of $14.4 \times 10^9/l$ with neutrophilia. The erythrocyte sedimentation rate was 95 mm in the first hour. Repeated urine microscopy showed no haematuria. The electrocardiogram showed sinus tachycardia with electrical alternans in the praecordial leads. The chest x-ray showed a grossly globular heart and bilateral pleural effusions. The antistreptococcal O lysin titre (ASOT) was 716 Todd units. Blood and urine cultures were taken, but they grew no pathogenic organisms.

Echocardiography showed the presence of a large pericardial effusion with 'strands' extending across the pericardial space (Figs 1, 2). The mitral valve was thickened, but the aortic valve was normal. No vegetations were seen. The left ventricle was 'swinging' in the pericardial space. The patient was treated with salicylates, ampicillin, cloxacillin, penicillin, and gen-

tamicin for one week without any improvement. As she deteriorated despite therapy, she was transferred to our Cardiac Intensive Care Unit for further management.

On admission she was found to be in cardiac tamponade. The respiratory rate was 40 per minute with heart rate of 150 per minute. The blood pressure was 100/70 mmHg with a paradox of 30 mmHg. Her jugular veins remained distended to the angle of the jaw. Emergency pericardiocentesis was done and 500 ml of evenly blood-stained fluid obtained. The patient immediately became more comfortable. The

Fig. 1 2D echocardiogram (subxiphoid 4-chamber view) showing the presence of a pericardial effusion and intrapericardial fibrinous deposits (arrows). RA = right atrium, LA = left atrium, LV = left ventricle, PE = pericardial effusion.

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Fig. 2 2D echocardiogram (parasternal long axis view) showing the presence of a pericardial (PE) and pleural effusion (PL). The intrapericardial deposits are shown by the arrows. LV = left ventricle, LA = left atrium.

pulse rate fell to 120 per minute and the blood pressure increased to 120/55 mmHg with a reduced paradox of 12 mmHg. The pericardial fluid was sent for pyogenic and mycobacterial cultures, which proved to be negative. Two days after pericardiocentesis, a repeat echocardiogram showed persistence of the pericardial effusion. Haemoglobin electrophoresis subsequently showed the patient to have haemoglobin-E disease. Rheumatoid arthritis factor, antinuclear antibodies and lupus erythematosus cell factor were all negative. The patient remained ill despite receiving antibiotics, salicylates and treatment for heart failure.

At that time it was not possible to come to a definitive diagnosis. The most likely diagnosis was thought to be acute rheumatic pancarditis. The possibility of tuberculous pericarditis could not be excluded.

As she remained seriously ill, steroids were given in conjunction with antimycobacterial drugs. Dramatically, within 48 hours of steroid therapy, defervescence of the fever occurred without recurrence. Heart failure improved and a repeat echocardiogram done one week after pericardiocentesis showed resorption of the pericardial effusion. The ASOT fell to 232 Todd Units. Serial chest radiographs showed the dramatic reduction in heart size. The cardiac murmurs remained unchanged. The patient was discharged after one month's convalescence in hospital and has remained well at follow-up 6 months later without need for any antibiotic therapy.

Discussion

Critically ill patients with fever, valvular heart disease, and cardiomegaly frequently pose diagnostic and therapeutic problems. In such a situation echocardiography is invaluable, since it can differentiate between ventricular dilatation and pericardial effusion as the cause of cardiomegaly. Identification of dense intrapericardial 'strands' on 2-D echocardiogram¹ and subsequent pericardiocentesis established the presence of an exudative pericardial effusion in our patient.

The clinical presentation and initial investigations indicated that the most likely diagnosis was acute rheumatic pericarditis with a massive pericardial effusion. In the less developed areas in the tropics rheumatic fever is more severe and intense than in developed countries in the West.² However, in this region tuberculous pericarditis and acute infective endocarditis had to be excluded. The patient had not responded to salicylates, and steroids had to be given on the presumptive clinical diagnosis of acute rheumatic pancarditis. The patient was too ill to await the results of more definitive tests, and steroids would have aggravated her condition if she had uncontrolled pyogenic or mycobacterial infection. She was, hence, treated with these other possibilities in mind. Subsequent investigations and the dramatic clinical response to steroids confirmed the initial diagnosis of acute rheumatic pancarditis with a massive haemorrhagic pericardial effusion.

The presence of an exudative pericardial effusion was demonstrated by pericardiocentesis and echocardiography. The pathology of rheumatic pericarditis is well documented.^{3,4} It consists of an intensive inflammation with extensive shaggy adhesions.³ Despite extensive fibrinous deposits the pericardial effusion usually resolves without any chronic sequelae.⁵

During the acute stage the echocardiogram in our patient showed the presence of extensive intrapericardial fibrinous deposits. After steroid therapy repeat echocardiograms showed almost complete resorption of the effusion. Echocardiographic recording of the acute exudative pericardial effusion and its subsequent resorption in acute rheumatic pancarditis has, we believe, not been reported before.

Acute rheumatic pericarditis has been said rarely, if ever, to produce cardiac tamponade owing to the slow rate of pericardial fluid accumulation which allows the pericardium to stretch.⁴ Our patient had clinical evidence of cardiac tamponade on admission, namely, hypotension, raised venous pressures, tachycardia, tachypnoea, and pulsus paradoxus in excess of 10 mmHg. There was immediate relief on pericardiocentesis.

Large pericardial effusions have been reported in rheumatic fever.³⁻⁶ These patients have a poorer prognosis and a poorer response to salicylates.⁶ Though usually serosanguinous, haemorrhagic pericardial effusions have been reported—and were fatal.³⁻⁶ It is conceivable that the size of the effusion might depend on the intensity of the inflammation. Rapid accumulation of fluid in the pericardium could result in cardiac tamponade, as in our patient. Urgent pericardiocentesis would then be life-saving.

References

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