Case of Q Fever Endocarditis at Site of Aortic Valve Prosthesis

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Bacterial and fungal infections are well-recognized complications of open-heart surgery. Rickettsial infections might also be expected to occur but have not, as far as we are aware, been described. We report here a case of Q fever endocarditis involving the tissues around an aortic valve prosthesis.

Case Report

A 40-year-old floor layer was admitted to the Exeter City Hospital in July 1967. He gave a history of rheumatic fever twice in childhood and had developed severe aortic incompetence requiring the replacement of his aortic valve by a Starr-Edwards prosthesis at the Bristol Royal Infirmary in July 1966. The day after operation the patient had a cardiac arrest and a few days later a myocardial infarction, but despite these complications he made a good recovery, and by February 1967 was able to return to work. He continued working until a week before his admission when he started to feel ill, developed chest pain and night sweats, and became breathless.

On examination he looked ill and feverish. He was sweating and pale but not cyanosed. Finger clubbing was conspicuous. The pulse was regular and the blood pressure was 170/80 mm. Hg. He had a thrusting apex beat felt in the fifth intercostal space and displaced to the anterior axillary line. There was a loud midsystolic murmur maximal in the aortic area and conducted into the neck. For the first time since operation, a short early diastolic murmur was heard. There were no signs of heart failure and no embolic manifestations. Neither the liver nor the spleen was palpable.

Investigation showed that the haemoglobin had fallen from 14.3 g./100 ml. three weeks before admission, to 12.9 g./100 ml.; the erythrocyte sedimentation rate (Wintrobe) had risen from 13 mm. to 45 mm. in the first hour. The blood urea was 24 mg./100 ml., and occasional red and white cells and a trace of albumin were found in the urine. Total serum proteins were 7.1 g./100 ml., albumin 3.0 g./100 ml. and globulin 4.1 g./100 ml., with a slightly raised γ -globulin fraction. The

electrocardiogram showed a prolonged P-R interval (Fig. 1) indicating first degree heart block. On x-ray the cardiothoracic ratio had remained unaltered since before his operation (7:11.5), but congestive changes had appeared in the lung fields.

A diagnosis of endocarditis was made and this was strengthened by the development of Osler's nodes on two fingers on the day after admission. Though blood cultures were sterile, intensive antibacterial therapy with penicillin and streptomycin was started. As there was no response to this treatment and further blood cultures were sterile, the possibility of Q fever endocarditis was considered. Complement-fixation tests against *Rickettsia burnetii* antigens done four weeks after admission gave raised titres—phase 1, 1/640; phase 2, 1/1280. Therapy was changed to tetracycline without any clinical improvement. A few days later the patient had a pulmonary embolus, and despite treatment he developed congestive cardiac failure and died two weeks after starting tetracycline therapy.

Pathology. Necropsy was performed 72 hours after death. In view of the diagnosis of Q fever endocarditis and the known risk of infection by aerosol spread (Marmion and Stoker, 1950), a limited necropsy examination confined to the removal of the heart was performed.

The heart showed biventricular dilatation and hypertrophy. The pericardium was thickened and adherent especially in the area where it had been incised at operation. The Starr-Edwards valve was intact. On the ventricular side there was a vegetation measuring approximately 1 cm. $\times$ 1 cm. which extended into the ventricular cavity (Fig. 2). The base of the vegetation was at the ventricular suture line of the valve. Its position was such that it would have prevented the complete return of the ball into the valve ring, thus allowing diastolic reflux.

The mitral orifice admitted one and a half fingertips. The valve cusps were hard and thickened, a finding consistent with old rheumatic heart disease. There was a line of friable vegetations on both valve cusps along

FIG. 1.—(A) Before operation—P-R interval 0.20 sec. (B) Three months after operation—P-R interval 0.20 sec. (C) On admission—P-R interval 0.30 sec.

FIG. 2.—Ventricular aspect of Starr-Edwards valve showing vegetation. Inset: Section through vegetation showing rickettsiae. ($\times 1200$.)

their line of closure. The pulmonary and tricuspid valves appeared normal.

Sections of the aortic vegetation showed mainly fibrinous material with, in parts, early signs of organization, and in others, areas of acute inflammation. No microorganisms were found on routine examination. Dr. D. E. B. Powell, using the methods reported by Evans, Powell, and Burrell (1959), showed rickettsiae in this lesion. Sections of the mitral valve showed old fibrous thickening with extensive calcification and a fibrinous vegetation along the line of closure. The heart muscle showed a few areas of scarring but there was no evidence of acute infection.

Microbiology. Specimens of blood taken before death and post-mortem cardiac tissue were inoculated intraperitoneally into guinea-pigs (Table). The tissues were ground in Medium 199 (Morgan, Morton, and Parker, 1950) without antibiotics; 1 ml. of dilutions (w/v) of 10^{-1} and 10^{-4} were inoculated to distinguish surface or blood-borne infection from tissue infection (Andrews and Marmion, 1959), the guinea-pigs were bled by cardiac puncture before inoculation and again 6 to 8 weeks later. The sera were examined for complement-fixing antibodies to *R. burnetii* phase 2 antigen and the results are given in the Table. They show that a rickettsaemia

was present both before and during treatment with tetracycline. In the heart tissue infection with *R. burnetii* was localized to the aortic vegetation.

Comment

Kristinsson and Bental (1967) have shown that some cases of Q fever endocarditis can be success-

TABLE
RESULTS OF COMPLEMENT-FIXATION TESTS
AGAINST *R. BURNETII* PHASE 2 ON GUINEA-PIG
SERA

Specimen	Complement-fixing titre	
	Pre-inoculation serum	Post-inoculation serum
Blood before tetracycline therapy	< 5	> 80
Blood during tetracycline therapy	< 5	20
Aortic vegetation 10^{-1}	< 5	> 80
10^{-4}	< 5	> 80
Mitral valve 10^{-1}	< 5	> 80
10^{-4}	< 5	> 10
Myocardium 10^{-1}	< 5	40
10^{-4}	< 5	> 10
Pulmonary valve 10^{-1}	< 5	> 10
10^{-4}	< 5	> 10

fully treated by replacing the infected valve by a prosthesis. Our case appears to be the first reported where Q fever endocarditis developed in relation to an artificial valve. A large vegetation arose from the base of the prosthesis interfering with its function, in contrast to the small vegetations or destructive lesions from infection by *R. burnetii* in other reports.

The presence of first degree heart block may indicate direct involvement of the conducting system by rickettsiae. In the series of Kristinsson and Bentall, surgical treatment was undertaken primarily for haemodynamic reasons, but in future operations may be used solely to remove infected tissue. First-degree heart block may indicate that such excision would require to be extensive.

Treatment with antibiotics has generally been disappointing and in our case did not influence the course of the disease. We were able to show that a rickettsiaemia was still present after one week's treatment with tetracycline.

In view of the poor prognosis, consideration should be given to the immunization of patients at risk from *R. burnetii*.

Summary

A man of 40 with an aortic valve prosthesis developed Q fever endocarditis localized to the tissues around it. The granulations rendered the valve incompetent, and in spite of treatment the patient died.

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