Endocarditis as a manifestation of *Chlamydia B* infection (psittacosis)

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A case of Chlamydia B (*psittacosis*) endocarditis is described in a patient with no known previous valve disease. After mitral valve replacement a fall in Chlamydia B antibody titre occurred. At repeat mitral valve replacement five months later for a paravalve leak no evidence of continuing endocarditis was present. Direct confirmation of infection in the patient's apparently healthy budgerigar was obtained. Aspects of the pathogenesis, diagnosis, and treatment are discussed.

Endocarditis caused by *Chlamydia B* is rare. We are aware of only two previous well-authenticated reports in which the patient was diagnosed and treated by valve replacement (Birkhead and Apostolov, 1974; Ward et al., 1975). We report a further case in which the diagnosis was established by histology and serology and in which mitral valve replacement was performed.

**Case report**

A 43-year-old housewife was admitted with a history of tiredness, vague left-sided chest discomfort, and weight loss.

There was no history of previous rheumatic fever or other major illness. A soft apical systolic murmur had been noted one year previously but not subsequently confirmed.

Examination revealed left basal atelectasis, a soft apical systolic murmur as previously noted, and a short diastolic murmur at the left sternal edge. Mild pyrexia was subsequently noted on two occasions during this admission.

Chest x-ray film showed a normal cardiac contour, with left lower lobe atelectasis. Haemoglobin was 8.3 g/dl; white count 5.4 x 10⁹/l; blood film normochromic and normocytic; and erythrocyte sedimentation rate (ESR) 112 mm in the first hour. Repeated blood cultures (six) were negative.

*Haemophilus influenzae* was isolated in the sputum. She was treated with ampicillin and improved, being discharged after 13 days.

Three months later she was readmitted because of exertional dyspnoea and orthopnoea. She also complained of retrosternal chest pain and a 'loud' heart beat.

She was afebrile, but now had an apical systolic thrill with a grade 5/6 pansystolic murmur conducted to the axilla. Fine crepitations were heard at the lung bases. The spleen was palpable.

Haemoglobin was 11.5 g/dl and ESR 24 mm in the first hour.

Chest x-ray film showed gross cardiac enlargement with pulmonary venous congestion.

The electrocardiogram was normal on admission but T wave inversion in leads III aVF, V2-V3 and ST segment depression in leads V4-V6 developed over the ensuing weeks.

Blood cultures (six) were again repeatedly negative but *Chlamydia B* complement-fixing antibody titre was 1:128. A presumptive diagnosis of *Chlamydia B* endocarditis was, therefore, made.

Further inquiry revealed that the patient had kept an apparently healthy budgerigar for five years and she had been the only member of the household to clean the cage.

Subsequently serology on the other members of the family proved negative. The budgerigar was sacrificed and necropsy showed *Chlamydia B* in stained smears of the liver.

The patient was treated with tetracycline for six weeks but mitral regurgitation was clinically so severe that mitral valve replacement was required.

At operation severe pulmonary arterial and
left atrial hypertension were present. The mitral valve was grossly damaged, having two large perforations of the anterior leaflet, ruptured anterior leaflet chordae, and anterior leaflet prolapse. There were no obvious vegetations and the remaining leaflet tissue was only a little thickened. A glutaraldehyde-processed porcine xenograft (Hancock) valve was used as replacement.

The patient made an uneventful recovery. At the time of discharge from hospital *Chlamydia B* complement-fixing antibody titre had fallen to 1:32.

**Histology and serology**

Microscopically, the valve had lost its normal architecture. The leaflets were thickened and fibrosed, with focal heavy inflammatory infiltration of polymorphs, macrophages, and lymphocytes. Areas of necrosis and haemorrhage were present with perforation at one point. Thrombus at varying stages of organization was present on the leaflet surface.

Modified Giemsa staining of cryostat sections of valve revealed numerous basophilic coccoid bodies (less than 1 μ in diameter) closely resembling typical chlamydial inclusions. Most were present within macrophages, but some were lying free.

Using Macchiavello’s method these coccoid bodies were again evident.

Indirect fluorescent antibody staining with the positive human serum confirmed the presence of chlamydiae within the inclusions. Small amounts of nonspecific fluorescence were present throughout the section, but the characteristic bright yellow-green positive fluorescence with the chlamydiae was easily distinguished. Sections stained with the negative control serum were clearly negative.

Direct staining with antihuman Ig:FITC conjugated serum also showed reasonably bright fluorescence, though this was brightest in the envelope and less brilliant with the individual chlamydiae within the inclusion. Rabbit anti guinea-pig: FITC conjugate did not show any fluorescing material either in the envelope or chlamydiae within the inclusion. Nonspecific fluorescence was not present.

**Subsequent clinical course**

Tetracycline was given for four weeks after operation.

About a month after discharge dyspnoea on effort recurred and progressive signs of mitral regurgitation became apparent. Though recurrent chlamydial endocarditis was suspected the antibody titre remained low at 1:28.

Cardiac catheterization 5 months after the initial operation showed severe pulmonary hypertension and raised pulmonary wedge pressure, with prominent ‘V’ wave. Left ventriculography confirmed fairly severe mitral regurgitation, but it was not clear whether this was central or peripheral to the valve.

At second operation 6 months after the first operation a paravalvar leak was found, caused by tearing out of sutures from the posterior annulus. Though the xenograft valve appeared normal it was removed and replaced by a Bjerk-Shiley prosthesis. Recovery from this operation was uneventful and she was discharged on long-term prophylactic tetracycline. Antibody titre has since shown a further fall to 1 in 8.

Pathological and serological examination of the excised xenograft valve was repeated as previously described and showed no evidence of infection.

**Discussion**

The incidence of *Chlamydia B* infection in man has shown a steady increase in recent years in the United Kingdom (Communicable Diseases Scotland Weekly Report, 1975). Part of this increase may be attributable to the lifting of restrictions on the importation of psittacine birds in 1966. *Chlamydia B* endocarditis may well be emerging as a more recognized manifestation of the infection.

In previous reports of *Chlamydia B* endocarditis the affected valve has usually shown a congenital abnormality or evidence of previous rheumatic heart disease (Birkhead and Apostolov, 1974; Grist and McLean, 1964; Levison et al., 1971). In the case reported here there was no history to suggest valve disease and at operation no firm evidence of previous valve abnormality was seen. Direct demonstration of *Chlamydia B* infection in the patient’s apparently healthy budgerigar confirms the previous suspicion that the domestic budgerigar can be a reservoir of infection for man. Transmission of the disease to our patient probably occurred by inhalation of infected droppings. We now question the wisdom of patients with known valvular heart disease (a group at risk from endocarditis) keeping domestic birds which may be infected with *Chlamydia B*.

*Chlamydia B* must now be recognized as an additional cause of infective endocarditis. In such cases serological examination for *Chlamydia B* and other unusual organisms such as coxiella should be performed, especially in the presence of negative blood cultures. Though endocarditis was suspected at the time of the initial presentation it is possible that the diagnosis of chlamydial B endocarditis was missed because of failure to appreciate this point. Whether psittacosis infection has a role in the aetio-
logy of chronic valvular heart disease, as Ward (1971) has suggested, is still uncertain.

This case report again raises doubts over the efficacy of present methods of treatment. Though tetracycline in a dose of 1 g/day was administered over a six-week period numerous organisms could be seen in the excised valve. Our experience is similar to that of Birkhead and Apostolov (1974) who failed to eradicate the organism from the aortic valve despite four months' treatment with tetracycline and rifampicin. Recognizing the limitations of present drugs, Oakley (1974) has suggested that tetracycline should be continued indefinitely to prevent relapse.

Valve replacement surgery was performed in this case and postoperatively clinical recovery occurred and was accompanied by a significant fall in Chlamydia B complement-fixation antibody titre. This is similar to the experience in the two previous cases described (Birkhead and Apostolov, 1974; Ward et al., 1975). The ideal choice of a suitable valve replacement in such a case is not clear. Though we chose to use a prosthetic valve at the second operation because of doubts regarding infection of the tissue valve, no such infection was demonstrable. Thus a combination of valve replacement surgery and long-term prophylactic chemotherapy may well be the treatment of choice in this manifestation of Chlamydia B infection.

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