UNSUSPECTED Q FEVER ENDOCARDITIS — A CASE REPORT

by

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Q fever endocarditis is usually characterized by a history of contact with animals or animal products, subsequent weight loss, pyrexia and night sweats. Clinical features almost invariably include valvular involvement, hepatosplenomegaly and haematuria.[1] We report a case of Q fever endocarditis in an elderly woman who, despite high titres of antibody, initially had no features apart from valvular involvement.

CASE REPORT

A seventy year old woman presented with tiredness and dyspnoea. There was no history of weight loss, sweating, anorexia or rash. Clinical examination revealed the signs of aortic stenosis and incompetence; there was no pyrexia, tachycardia, anaemia or hepatosplenomegaly. Routine haematological studies were normal and there was no haematuria, but the ESR was 43 mm/hr, the IgG level was raised at 23.2 g/l and Q fever complement fixing antibody titres, checked routinely in this unit, were elevated (Phase 1—1,600, Phase 2—400). There was no history of direct contact with animals though the patient lived in an area where her house was fronted by a road frequently used by cattle lorries. Cardiac catheterization confirmed fairly severe calcific aortic stenosis with mild incompetence. Neither the left ventricular angiogram nor echocardiogram showed vegetations on the aortic valve. In the absence of clinical features of Q fever endocarditis, antibiotics were initially withheld and over the next two months she remained well with normal haematological screening apart from a few abnormal mononuclear cells in the blood films. However, both phase 1 and 2 Q fever antibody titres rose to 3,200 and the IgG level rose to 34.32 g/l. There was still no anaemia, pyrexia or hepatomegaly but the tip of the spleen became palpable and the aortic diastolic murmur more pronounced.

Rifampicin 600 mg daily and co-trimoxazole two tablets twice daily was started but the co-trimoxazole was discontinued after three weeks, because of a skin rash which resolved on cessation of this drug.

During the next three months the antibody titres and immunoglobulin level remained relatively unchanged but the haemoglobin fell from 12.8 to 10.9 g/dl. With the patient becoming increasingly dyspnoeic on effort and at rest aortic valve replacement was considered necessary.

At operation the aortic valve was bicuspid and heavily calcified with low grade inflammatory process at the insertion of the valve leaflets. A size 23 aortic St. Jude valve was sutured in place.

Guinea pig inoculation with a suspension of the homogenised aortic valve in buffered saline was carried out at the Special Pathogens Reference Laboratory at Porton Down. Blood samples from all four guinea pigs before inoculation and after

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22 days showed a rise in Q fever phase 2 antibody titre from < 10 to > 640, confirming active infection.

Six months after valve replacement she was asymptomatic with no signs of cardiac failure or aortic incompetence and the spleen was not palpable. Both phase 1 and 2 Q fever antibody titres had fallen to 200. Tetracycline treatment, which had been commenced post-operatively, was continued.

DISCUSSION

For twelve weeks our patient with aortic valve disease had high antibody titres to *Coxiella burnetii* without typical clinical features. Previous reports have usually stressed a history of exposure, though this may be limited to loose straw in the neighbourhood[2] as possibly in this case. All eight cases reported by Varma and his colleagues had a history of exposure, pyrexia, night sweats and enlargement of liver or spleen, or both organs.[3] In one patient with infection of a ventricular aneurysm there was an absence of symptoms and signs.[4]

In this case elevated and rising antibody titres in the presence of valve disease indicated active infection, despite absence of the usual features. Earlier antibiotic therapy may have prevented the necessity for surgical intervention though valve replacement would eventually have been required. Furthermore, since Q fever may be a surgical hazard we suggest that Q fever antibody titres should be estimated routinely before valve surgery is undertaken.

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REFERENCES

1 Turck WPG, Howitt G, Turnberg LA, Fox W, Matthews MB, Das Gupta R. Chronic Q Fever. *Q J Med* 1976; **45**: 193-217.

2 Salmon MM, Howels B, Glencross EJG, Evans AD, Palmer SR. Q Fever in an urban area. *Lancet* 1982; **I**: 1002-1006.

3 Varma MPS, Adgey AAJ, Connolly JH. Chronic Q fever endocarditis. *Br Heart J* 1980; **43**: 695-699.

4 Willey RF, Matthews MB, Peutherer JF, Marmion BP. Chronic cryptic Q fever infection of the heart. *Lancet* 1979; **II**: 270-272.