Endocarditis due to *Cardiobacterium hominis*

R. S. JOBANPUTRA¹ AND JEFFERY MOYSEY

From the Departments of Microbiology and Cardiology, Northwick Park Hospital and Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ, UK

SUMMARY A case of bacterial endocarditis was caused by *Cardiobacterium hominis* in a 41-year-old man with mitral and aortic incompetence due to a previous episode of rheumatic fever. The main distinguishing characteristics of *C. hominis* are described, and the incidence of endocarditis is reviewed. After six weeks of treatment with effective bactericidal chemotherapy a microbiological cure was achieved and the patient successfully underwent mitral valve replacement and aortic valve plication.

Cardiobacterium hominis was first recognised as a causative organism in human infection by Tucker and associates in 1962. They described four cases of endocarditis caused by a group of organisms resembling *Pasteurella* and named at that time as 'group II D'. In 1964 Slotnick and Dougherty studied the cultural characteristics, biochemical reactions, and antigenic relationships of this organism and proposed the name *Cardiobacterium hominis*. There have been only two cases of endocarditis due to *C. hominis* reported in the United Kingdom.

Case report

A 41-year-old man who lived alone, presented to Northwick Park Hospital outpatient department on 4 August 1976, giving a history of rheumatic fever at 12 years of age and describing a rash, swelling and pain in the ankles, knees, and hands, and involuntary movements. At the time he had been confined to bed for many months and advised to abstain from school sports. His only attendance at the doctor's surgery was for an attack of loin pain. Otherwise he claimed that he was keeping extremely well, although he did admit to a weight loss of 9.5 kg over a period of six weeks, increasing breathlessness, ankle swelling, and malaise. He worked as a supervisor with a local paper manufacturer and smoked 25 cigarettes per day.

On examination he looked thin and had gross gingivitis. There were no splinter haemorrhages or Roth's spots. The jugular venous pressure was raised by 2 cm and the cardiac apex was palpable in the sixth intercostal space in the anterior axillary line. He had an apical pansystolic murmur grade 4/4 and a soft early diastolic murmur grade 2/4 at the left sternal edge. The liver was smoothly enlarged 2 cm below the costal margin, but there was no splenomegaly.

Although afebrile on admission, over the next two days he had several pyrexial episodes. His haemoglobin was 8.9 g/dl with indices of a normochromic, normocytic anaemia and a 3.9% reticulocyte response. He had an ESR of 133 mm/h (Westergren), haematuria lasting for 11 days of his illness, and an electrocardiogram indicating left ventricular hypertrophy. His chest x-ray showed a cardiothoracic diameter of 17:29, and the appearance of the lung fields was that of pulmonary oedema. A presumptive diagnosis of infective endocarditis on incompetent mitral and aortic valves was made. Serial venous blood cultures produced a growth of *C. hominis*, and the patient was started on intravenous penicillin G, 3 mega units six-hourly. An echocardiogram showed gross left atrial and ventricular enlargement, with increased mitral valve excursion and a reduced ejection fraction. Immediately after admission to hospital he was noted to have a raised total serum protein (97 g/l) and serum globulin (70 g/l) and a reduced serum albumin (27 g/l). Urinary Bence Jones protein was negative and plasma electrophoresis and immunoelectrophoresis revealed a polyclonal increase in IgG at 54.5 g/l (normal 5.9 to 17.9 g/l).

¹Present address: Public Health Laboratory, St. John's Hospital, Thorpe Road, Peterborough, Cambs.

Received for publication 6 April 1977

Twenty-two days into his illness he became acutely dyspnoeic with a tachycardia and increased signs of cardiac failure. After this increase, the signs of aortic incompetence became more prominent and he developed a third heart sound. Despite treatment his ESR remained unacceptably high and seven days of intravenous gentamicin, 80 mg eight-hourly, was given concurrently with the penicillin. Seven weeks after admission and one week after cessation of antibiotic therapy venous blood cultures were negative. His condition continued to improve and he was discharged home on 9 October 1976. Subsequently he underwent total dental clearance and then cardiac catheterisation which confirmed gross mitral and aortic incompetence. At surgery on 7 December 1976 the chordae tendineae of the mitral valve were found to be ruptured and calcified, allowing gross prolapse of the anterior cusp (Fig. 1). The valve was replaced with a sclerosed pig (Hancock) valve. The aortic valve was incompetent due to a thickened and shortened left coronary cusp, and plication significantly reduced the degree of regurgitation. Postoperatively atrial fibrillation developed for the first time but was controlled with digoxin, and he was finally discharged home on

Fig. 1 Surgically excised mitral valve showing the ruptured chordae tendineae to the anterior cusp (courtesy of the Photography Department, St. George's Hospital, London).

21 December 1976. A summary of his clinical course is outlined in Figure 2.

Microbiological findings

Three successive venous blood cultures were taken over the first 24 hours after admission. On each occasion 5 ml of blood were inoculated into liquid broth (10 ml) and thioglycollate broth (100 ml). Liquid broth was incubated in air with 10% CO₂ at 37°C. Thioglycollate broth (anaerobic bottle) was incubated at 37°C. Each broth was subcultured to horse blood agar and incubated under the same conditions as the primary cultures. The organism isolated from the blood of the patient was first apparent after 48 hours' incubation in liquid broth (CO₂ bottle). On subculture to horse blood agar the organism grew slowly and colonies were 1-2 mm in size after 48 hours. All three CO₂ bottles yielded pleomorphic Gram-negative rods which were non-motile. No growth was observed in three anaerobic bottles. The biochemical properties of the organism were as follows: catalase negative; oxidase positive;

Fig. 2 A summary of the clinical course.

indole positive; nitrates not reduced; urease negative; ONPG negative; H₂S negative; acid, without gas was produced from glucose, maltose, mannitol, fructose, mannose, and sorbitol; acid not formed from lactose and sucrose. On the basis of these findings the organism was identified as *C. hominis*. The identity of the organism was confirmed by the National Collection of Type Cultures, Central Public Health Laboratory, Colindale, London. By disc test, the organism was sensitive to penicillin, ampicillin, cephaloridine, tetracycline, co-trimoxazole, and gentamicin.

The minimal inhibitory (MIC) and bactericidal (MBC) concentrations of penicillin against the isolate were 0.06 mg/l. A sample of the patient's serum collected on the 28th day after admission was examined for agglutinins to *C. hominis* isolated from the blood. Suspension of the organism heated to 100°C showed an agglutination titre of 1280. Assay of the bactericidal activity of the patient's serum against the homologous isolate was undertaken on the sixth day of penicillin therapy. The tube dilution method was used with an inoculum of 10⁶ organisms per ml in serum broth. The patient's serum was bactericidal in dilutions up to 256.

Discussion

Cardiobacterium hominis appears to be a rare cause of endocarditis. Slotnick *et al.* (1964) have isolated *C. hominis* from the respiratory tract in 68% of normal individuals. The investigation was carried out using specific fluorescent antibody technique and bacteriological culture. None of the persons examined in this study showed any evidence of past or current history of rheumatic heart disease, endocarditis or related heart disease. There were no circulating antibodies in persons harbouring *C. hominis* and he concluded that *C. hominis* is a member of indigenous commensal flora. We consider that dental sepsis was the most likely origin of infection in this case. The raised serum globulin may be due

to a phase of immunological reaction to infective endocarditis.

Reviewing the literature, 10 cases of endocarditis due to *C. hominis* are reported (Table). In seven cases there were pre-existing cardiac lesions and in three cases there was no evidence of previous cardiac lesions. In one case, a femoral artery mycotic aneurysm developed after antibiotic therapy (Perdue *et al.*, 1968). A 58-year-old man responded satisfactorily to intravenous penicillin treatment but a severe degree of aortic incompetence remained and subsequently required aortic valve replacement (Midgley *et al.*, 1970). In another case major cardiac damage required valvular replacement and the patient had a cerebral mycotic aneurysm (Laguna *et al.*, 1975).

C. hominis should be distinguished from other closely related organisms such as *Haemophilus aphrophilus*, *Actinobacillus actinomycetemcomitans*, and *Streptobacillus moniliformis*. All these species require CO₂ and high humidity for their growth. The main characteristics of *C. hominis* distinguishing it from other closely related organisms are: (1) absence of catalase activity; (2) positive oxidase reaction; (3) production of indole; and (4) absence of nitrate reduction. The production of hydrogen sulphide differs among different strains of the species. Midgley *et al.* have shown that three strains of *C. hominis*, including one strain NCTC 10427 from Dr Slotnick, failed to produce hydrogen sulphide.

We thank Dr E. B. Raftery and Dr R. Blowers for permission to publish this report, and Mr D. J. Parker, consultant cardiothoracic surgeon, St. George's Hospital, London, for the surgical notes. Our thanks are also due to Mr John A. Taylor, National Collection of Type Cultures, for confirming the identity of the isolate.

References

Laguna, J., Derby, B. M., and Chase, R. (1975). *Cardiobacterium hominis* endocarditis with cerebral mycotic

Table Data of published cases of endocarditis due to *Cardiobacterium hominis*

Reference	Age	Sex	Pre-existing cardiac lesions	Antibiotic treatment
1962 Tucker and others	39	F	Yes	Penicillin
	57	M	Yes	Penicillin and streptomycin
	50	F	Yes	Penicillin and streptomycin
	43	F	No	Penicillin, streptomycin, and tetracycline
1968 Perdue and others	33	M	Yes	Penicillin, streptomycin, and chloramphenicol
1969 Snyder and Ellner	53	F	No	Penicillin and streptomycin
1970 Midgley and others	35	F	Yes	Penicillin and streptomycin
	58	M	Yes	Penicillin, ampicillin, and streptomycin
1975 Weiner and Werthamer	55	F	Yes	Penicillin
	65	M	No	Penicillin and streptomycin
Laguna and others				Penicillin and streptomycin

- aneurysm. *Archives of Neurology*, **32**, 638-639.
- Midgley, J., Lapage, S. P., Jenkins, B. A. G., Barrow, G. I., Roberts, M. E., and Buck, A. G. (1970). *Cardiobacterium hominis* endocarditis. *Journal of Medical Microbiology*, **3**, 91-98.
- Perdue, G. D., Dorney, E. R., and Ferrier, F. (1968). Embolomycotic aneurysm associated with bacterial endocarditis due to *Cardiobacterium hominis*. *American Surgeon*, **34**, 901-904.
- Slotnick, I. J., and Dougherty, M. (1964). Further characterisation of an unclassified group of bacteria causing endocarditis in man: *Cardiobacterium hominis* ge.et sp.n. *Antonie van Leeuwenhoek*, **30**, 261-272.
- Slotnick, I. J., Mertz, J. A., and Dougherty, M. (1964). Fluorescent antibody detection of human occurrence of an unclassified bacterial group causing endocarditis. *Journal of Infectious Diseases*, **114**, 503-505.
- Snyder, A. I., and Ellner, P. D. (1969). *Cardiobacterium hominis* endocarditis. *New York State Journal of Medicine*, **69**, 704-705.
- Tucker, D. N., Slotnick, I. J., King, E. O., Tynes, B., Nicholson, J., and Crevasse, L. (1962). Endocarditis caused by a Pasteurella-like organism. Report of four cases. *New England Journal of Medicine*, **267**, 913-916.
- Weiner, M., and Werthamer, S. (1975). *Cardiobacterium hominis* endocarditis: characterisation of the unusual organisms and review of the literature. *American Journal of Clinical Pathology*, **63**, 131-134.