

Endocarditis caused by *Rothia dentocariosa*

SA BROEREN, MM PEEL*

From the Pathology Department, Central Gippsland Hospital, Princes Highway, Traralgon, Victoria, 3844, and the *Microbiological Diagnostic Unit, University of Melbourne, Parkville, Victoria, 3052, Australia

SUMMARY A case of infective endocarditis caused by *Rothia dentocariosa* is described in a 53 year old man with a history of rheumatic fever. *R dentocariosa* is a component of the oral microbiota and has only rarely and recently been recognised as a human pathogen. In this patient the oral flora was the probable source of infection, with a broken molar tooth providing the probable avenue for infection.

In 1949, Onishi¹ described the isolation of a Gram positive branching bacterium from carious dentine, which he named *Actinomyces dentocariosus*. Roth² later placed the bacterium in the genus *Nocardia*, as *Nocardia dentocariosus*, because of its preference for aerobic conditions of growth. The same organism was described by Davis and Freer³ as *Nocardia salivae*, although these authors showed that it differed from *Nocardia* spp in cell wall composition. In 1967, Georg and Brown⁴ proposed the creation of a new genus *Rothia*, within the family *Actinomycetaceae*, to accommodate this bacterium, the characteristics of which did not conform to those of either *Actinomyces* or *Nocardia*.

R dentocariosa is a common inhabitant of the nose and throat.⁵ It has been isolated from many clinical sources but has rarely been reported as a cause of clinical infection.⁶ We describe a case of infective endocarditis caused by *R dentocariosa*.

Case report

A 53 year old man was admitted to hospital with a two week history of intermittent sweating, generalised pains, and stiffness. He was known to have impaired function of the mitral valve as a consequence of previous attacks of rheumatic fever. On examination he had a temperature of 38°C, a pansystolic heart murmur, and old splinter haemorrhages on both thumbs. Laboratory tests showed a haemoglobin concentration of 13.8 g/dl and a raised erythrocyte sedimentation rate (ESR) of 42 mm in the first hour (Westergren). During the next two weeks, the ESR increased to 100 mm in the first hour and the leucocyte count peaked at $17.6 \times 10^9/l$.

Three weeks after admission symptoms of intermittent myalgia and arthralgia developed; his haemoglobin concentration was 12.4 g/dl. By this time, Osler's nodes had appeared on the index fingers of both hands, and splinter haemorrhages subsequently developed under the fingernails.

Blood cultures taken two days after admission grew a Gram positive, branching, aerobic bacterium after incubation for eight days at 37°C. The isolate was initially thought to be a species of *Nocardia* and treatment with co-trimoxazole was started, with rifampicin being added later. *Mycobacterium* spp were excluded by the Mycobacterium Reference Laboratory of Fairfield Hospital, Fairfield, Victoria. An echocardiogram suggested the presence of small vegetations on the anterior leaflet of the mitral valve. Pleuritic pain developed on the right side of the chest and more Osler's nodes developed on the right hand. The ESR was 85 mm in the first hour. Nine sets of blood cultures were taken over the three week period after admission. The isolate from these cultures was identified as *Rothia dentocariosa* by the Microbiological Diagnostic Unit at the University of Melbourne. Antibiotic treatment was altered to include penicillin and gentamicin with the rifampicin, and co-trimoxazole was discontinued.

The patient gradually became afebrile and his symptoms resolved. Treatment with gentamicin and rifampicin was continued until six weeks after admission. He was discharged at eight weeks after admission on a regimen of oral penicillin V. On discharge, his ESR was stable at 26 mm in the first hour. The patient reported breaking a molar tooth while chewing on a bone about two months before admission. He received no dental treatment until about two months after admission to hospital, when the tooth was extracted and the presence of other carious teeth was noted.

Endocarditis caused by Rothia dentocariosa

BACTERIOLOGY

Blood samples were cultured in biphasic modified brain heart infusion medium (Gibco Laboratories, Cat No M06800) and fluid thioglycollate medium USP (Gibco Laboratories, Cat No M21200). After 24 and 48 h of incubation at 37°C, subcultures were made on to two plates of 5% horse blood agar, one of which was incubated aerobically and the other anaerobically, and a plate of MacConkey agar, which was incubated aerobically. Gram stained smears of the blood culture broths were also prepared and examined. Thereafter, blood cultures were examined daily and subcultured regularly. The Gram positive branching bacterium was generally detected in the brain heart infusion medium at the eighth day of incubation. It did not grow in the fluid thioglycollate medium.

After incubation aerobically on horse blood agar for 24 h at 37°C, isolated colonies were about 1 mm in diameter, creamy white, convex, and smooth with entire edges. After incubation for 72 h under the same conditions they reached a diameter of about 3 mm and became rough with concentric and radial ridges and irregular edges. Microscopically, Gram positive branching filaments, often with clavate ends, predominated in early cultures on horse blood agar, but Gram positive coccoid and coccobacillary forms and short rod shaped forms were predominant after incubation for 72 h at 37°C.

The isolate grew under aerobic and microaerophilic conditions. Growth under anaerobic conditions was minimal. Growth at 30°C was poor compared with that at 37°C. The bacterium grew on horse blood agar, nutrient agar, Hoyle's tellurite medium, and Lowenstein-Jensen medium but not on Sabouraud's dextrose agar. It was weakly acid fast and non-motile. The biochemical characteristics are given in the Table. The enzyme profile as determined by the API ZYM system (API system SA,

Biochemical characteristics of the isolate of Rothia dentocariosa

| Test | Results |
|--|-----------|
| Acid from carbohydrates (PWS)*: | |
| Positive for glucose, glycerol, maltose, salicin, sucrose, trehalose | |
| Negative for lactose, mannitol, raffinose, sorbitol, xylose | |
| Aesculin hydrolysis | + |
| Catalase activity | + |
| Casein digestion | - |
| Gelatin liquefaction | + |
| Growth in Simmons' citrate | - |
| Hugh and Leifson's OF test | F |
| Indole production | - |
| Nitrate reduction | + |
| Starch hydrolysis | - |
| Triple sugar iron (slant/butt) | Acid/acid |
| Urease production | - |
| Voges-Proskauer reaction | + |

*PWS = peptone water sugars
OF = oxidation/fermentation

38390 Montalieu, Vercieu, France) was consistent with that reported for *R dentocariosa* by Kilian⁷ except for a weakly positive reaction for phosphamidase. In particular, the isolate gave a strongly positive reaction for valine aminopeptidase, a characteristic of importance for differentiating it from *Bacterionema matruchotii*, the enzyme profile of which is otherwise indistinguishable. On the basis of the morphological and biochemical characteristics, the isolate was identified as *Rothia dentocariosa*.⁵⁻⁸

Minimal inhibitory and bactericidal concentrations for antimicrobial agents and the serum bactericidal concentrations for the therapeutic regimens were determined at the Microbiology Department of the Royal Melbourne Hospital, Parkville, Victoria. Minimal inhibitory and bactericidal concentrations (in mg/l) were: penicillin, ≤0.12, 1.0; gentamicin, ≤2.0, ≤2.0; rifampicin, ≤0.5, ≤0.5; sulphamethoxazole, >160, >160; trimethoprim, >8, >8. Pre-dose and post-dose serum bactericidal levels during treatment were 1:125 to 1:>256 and 1:>256, respectively, on two determinations.

Discussion

Although a common inhabitant of the normal mouth and throat,⁵ *R dentocariosa* has also been associated with carious teeth^{1 6} and periodontal disease.⁹ The bacterium has been isolated from a wide variety of clinical sources, including throats, cerebrospinal fluids, sputum samples, and blood,⁶ but it has only rarely been incriminated in clinical infections. One of the major manuals of clinical microbiology, published in 1980, makes no mention of this bacterium.¹⁰ The first report of *R dentocariosa* as a primary pathogen was published as recently as 1975.¹¹ Since then, of the infections attributed to *R dentocariosa* only two have been cases of endocarditis,^{12 13} and in neither of these was there any indication of the likely source of infection.

In our patient, the bacterium probably gained access to the blood stream through trauma to his carious teeth. Dental manipulations are often implicated as the causal event leading to endocarditis in patients with damaged heart valves; in this instance dental intervention with appropriate antibiotic cover may well have prevented infection.

R dentocariosa is sensitive to many antimicrobial agents. It has been uniformly susceptible when tested against penicillin, which is probably the antibiotic of choice for treatment.^{12 13}

Characteristics of value for differentiating *R dentocariosa* from other members of the family *Actinomycetaceae* have been described.^{4 6-8} Promi-

ment among those useful for differentiating *R dentocariosa* from species of *Nocardia* are its inability to grow on Sabouraud's dextrose agar, lack of aerial mycelium, and fermentative action on carbohydrates. Separation from *Actinomyces* and *Arachnia* is based mainly on the inability of the species of these genera to grow aerobically. Detection of the enzyme valine aminopeptidase provides a means of distinguishing *R dentocariosa* and *B matrucotii*.⁷ Increased awareness of the characteristics and potential pathogenicity of *R dentocariosa* should lead to the more frequent identification of this bacterium and an even better appreciation of its role as a human pathogen.

We thank Dr David Ogilvy and Dr Derek Wong for the clinical details; Dr ER Pavillard and Mr PB Ward of the Royal Melbourne Hospital for the antimicrobial sensitivity testing; and Dr JRL Forsyth for reviewing the manuscript.

References

- ¹ Onishi M. Study on the Actinomyces isolated from the deeper layer of carious dentine. *Shikagaku Zasshi* 1949;6:273-82.
- ² Roth GD. Proteolytic organisms of the carious lesion. *Oral Surg* 1957;10:1105-17.
- ³ Davis GHG, Freer JH. Studies upon an oral aerobic actinomy-
- cete. *J Gen Microbiol* 1960;23:163-78.
- ⁴ Georg LK, Brown JM. *Rothia*, gen. nov. an aerobic genus of the family Actinomycetaceae. *Int J Syst Bacteriol* 1967;17:79-88.
- ⁵ Georg LK. *Rothia*. In: Buchanan RE, Gibbons NE, eds. *Bergey's manual of determinative bacteriology*. 8th ed. Baltimore: Williams and Wilkins, 1974:679-81.
- ⁶ Brown JM, Georg LK, Waters LC. Laboratory identification of *Rothia dentocariosa* and its occurrence in human clinical materials. *Appl Microbiol* 1969;17:150-6.
- ⁷ Kilian M. Rapid identification of Actinomycetaceae and related bacteria. *J Clin Microbiol* 1978;8:127-33.
- ⁸ Holmberg K, Hallander HO. Numerical taxonomy and laboratory identification of *Bacterionema matruchotii*, *Rothia dentocariosa*, *Actinomyces naeslundii*, *Actinomyces viscosus*, and some related bacteria. *J Gen Microbiol* 1973;76:43-63.
- ⁹ Leshner RJ, Gerencser VF, Morrison DJ. Presence of *Rothia dentocariosa* strain 477 serotype 2 in gingiva of patients with inflammatory periodontal disease. *J Dent Res* 1977;56:189.
- ¹⁰ Lennette EH, Balows A, Hausler WJ Jr, Truant JP, eds. *Manual of clinical microbiology*. 3rd ed. Washington, DC: American Society for Microbiology, 1980.
- ¹¹ Scharfen J. Untraditional glucose fermenting actinomycetes as human pathogens. Part II: *Rothia dentocariosa* as a cause of abdominal actinomycosis and a pathogen for mice. *Zentralbl Bakteriol Mikrobiol Hyg [A]* 1975;233:80-92.
- ¹² Pape J, Singer C, Kiehn TE, Lee BJ, Armstrong D. Infective endocarditis caused by *Rothia dentocariosa*. *Ann Intern Med* 1979;91:746-7.
- ¹³ Schafer FJ, Wing EJ, Norden CW. Infectious endocarditis caused by *Rothia dentocariosa*. *Ann Intern Med* 1979;91:747-8.

Requests for reprints to: SA Broeren, Pathology Department, Central Gippsland Hospital, Princes Highway, Traralgon, Victoria, 3844, Australia