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NOTES

Abscess Associated with Rothia dentocariosa

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Rothia dentocariosa, an aerobic member of the Actinomycetaceae, was isolated from a pilonidal abscess. The clinical occurrence, bacteriological characteristics, and antimicrobial sensitivity pattern are presented.

Rothia was created as a genus in 1967 (3) because, although the organism resembles Actinomyces morphologically, it grows well aerobically and differs significantly from both Actinomyces and Nocardia in the constituents of its cell wall. The prototype organism, Rothia dentocariosa, was known previously as Actinomyces dentocariosus, Nocardia dentocariosus, and N. salivae. It may be found in the normal mouth and throat (2) as well as in inflammatory periodontal tissue (4). Although it appears to be a constituent of normal oral flora, the most recent edition of Bergey's Manual (2) states that no natural infections in humans or other animals have been reported. However, several other authors (1, 6) suggest that this bacterium can be the etiological agent in abscess formation. Here, we describe a human infection of a pilonidal cyst from which R. dentocariosa was isolated.

A 17-year-old female presented to a branch clinic of the Naval Regional Medical Center, Oakland, Calif., with an infected pilonidal cyst. Treated initially with sitz baths and analgesia, the abscess was surgically drained several days later. No Gram stain of the purulent material was done. A culture of the site produced a predominant growth of a gram-positive, gram-variable diphtheroid-like organism on 5% sheep blood agar. (Commercial media, California Laboratories Industries, North Hollywood, Calif., were used throughout this study.) It was later identified as R. dentocariosa. In addition, several colonies of a group F streptococcus were noted.

The *R. dentocariosa* isolate was aerobic to microaerophilic. Growth was significantly stimulated by 5% CO₂. Optimum growth occurred at

37°C. No growth occurred at 25°C. The organism grew well on 5% sheep blood agar and chocolate agar but not on MacConkey agar. Growth was apparent after 24 h as 0.5- to 1-mm circular, convex, entire, creamy white, smooth colonies. Their size increased to 2 to 3 mm after an additional 24 h of incubation without any visible morphological variation. They were adherent and formed very noticeable depressions in the agar. A Gram stain of the 24- and 48-h growth from the CO₂ blood agar plate revealed deep-staining, gram-positive coccobacillus а measuring approximately 1.0 by 1.5 μ m. The bacterium occurred mainly in pairs. A few short chains and clusters were also apparent. However, a Gram stain of the growth from parallel plates not incubated in CO₂ revealed a gramvariable bacterium. The predominant form was coccobacillary, but pleomorphic diphtheroidlike structures measuring approximately 1.0 by $3 \ \mu m$ were also noted. A Gram stain of the organism from the thioglycolate broth revealed gram-variable, bulbous-ended, diphtheroid-like filamentous structures. Very few coccobacillary structures were noted. Biochemically, the isolate was nitrate and catalase positive. The urease, indole, and litmus milk reactions were all negative. The bacterium produced acid from glucose, sucrose, maltose, and salicin but not from mannitol, glycerol, starch, arabinose, xylose, or inositol. A direct fluorescent-antibody test and biochemical reactions performed at the Center for Disease Control, Atlanta, Ga., confirmed R. dentocariosa.

A sensitivity pattern of the isolate to various antibiotics was performed, using the Kirby-Bauer disk diffusion technique. It was found to be uniformly sensitive to penicillin, cephalothin, erythrocmycin, tetracycline, chloramphenicol, clindamycin, trimethoprim-sulfamethoxazole,

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kanamycin, and gentamicin but resistant to colistin. No previous pattern could be found in the literature.

Brown-Brenn- and hematoxylin-eosin-stained cross sections of the pilonidal cyst were prepared for histological examination. The hematoxylineosin section revealed a hyperkeratotic squamous epithelial invagination projecting into the subcutaneous tissue. Keratotic debris and numerous bacteria were contained within the lumen of the invagination. Many polymorphonuclear leukocytes were noted percolating through the squamous epithelium of the invagination. Extensive granulation tissue surrounded the squamous invagination and proximal skin appendages. Numerous plasma cells, lymphocytes, fibroblasts, polymorphonuclear leukocytes, pigment-laden macrophages, and endothelial-lined spaces containing ervthrocytes were noted in the granulation tissue. Numerous gram-positive bacilli and coccobacilli forms were noted in the Brown-Brenn section.

Subsequent to the publication of the 8th edition of Bergey's Manual, Schafen (6), in 1975, described the only reported case of human infection due to R. dentocariosa. The report was of a periappendiceal abscess from which the organism grew, and a typical Gram stain of the purulent material was found. In addition to this single case, Brown et al. (1) described 50 isolates of R. dentocariosa derived from the Center for Disease Control collection and from several other sources. Of the cultures obtained from an identifiable source, only 7 of 46 were from locations which could be surmised to have been pathogenic, although no clinical data were available on any of the isolates. Most of the strains were from oral secretions or carious teeth. The isolates in question were obtained from cerebrospinal fluid, a chest abscess, postoperative wound, leg stump drainage, throat abscess, blood, and leg ulcer. Although potentially a pathogen in these settings, without clinical data and the lack of documented cases it is unclear whether *R. dentocariosa* was the etiological agent or a contaminant. This case report coupled with Schafen's case adds support to the supposition that *Rothia* may be able to form abscesses in humans. Roth and Flanagan (5) have investigated the pathogenicity of *Rothia* inoculated into mice. Localized abscesses were produced which slowly healed spontaneously after local injection. Schafen (6) also produced lesions in mice by intraperitoneal injection, which caused localized infection.

The absolute role that *R. dentocariosa* plays in abscess formation cannot be determined at this point. However, clinical microbiologists should be aware that the bacterium has been isolated from various sites as the predominant organism and should not necessarily pass it off as a contaminant. Further studies are needed to determine the true significance of this species in infective processes. Also, the role that other organisms play in combination with *R. dentocariosa*, such as group F streptococci, needs to be evaluated.

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