Rothia dentocariosa **Endocarditis:**

An Especially Rare Case in a Previously Healthy Man

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Rothia dentocariosa is a rare gram-positive bacterial organism, one of the group of microbes that normally resides in the mouth and respiratory tract. *R. dentocariosa* rarely causes disease. Documented cases occur chiefly in patients with valvular or dental disease, or both. We report the case of a previously healthy 58-year-old man who presented with evidence of bacterial endocarditis caused by this organism—which originated from an elusive source. His endocarditis was successfully treated with mitral valve replacement and the administration of antibiotic agents. (*Tex Heart Inst J* 2016;43(3):255-7)

Key words: Anti-bacterial agents/therapeutic use; embolism, septic; endocarditis, bacterial/drug therapy; endophthalmitis, endogenous; heart valve diseases/drug therapy/surgery; mitral regurgitation; *Rothia dentocariosa*/gram-positive; sepsis/etiology

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R*othia dentocariosa*, a normal commensal of the oral cavity, is a bacterium considered to be of low virulence. Usually associated with dental caries and periodontal disease, *R. dentocariosa* has a variety of presentations, including septic arthritis, pneumonia, septicemias in renal transplant patients, and arteriovenous infections.¹⁻⁵ It is noted that only a few cases of *R. dentocariosa* endocarditis have been reported to date, almost exclusively in patients with an underlying heart condition as the predisposing risk factor.⁶ We present a case of a previously healthy patient with endocarditis caused by this rare organism.

Case Report

In September 2011, a 58-year-old man presented to his primary care physician (PCP) with a one-month history of fatigue, chills, night sweats, and fevers up to 102 °F. The patient also had a rash on his lower-right extremity. He was diagnosed with cellulitis and was treated with amoxicillin and clavulanate (875 mg/125 mg, 2×/d) for 10 days and showed subsequent improvement in his symptoms. An incidental murmur was thought to be insignificant at the time. Two weeks later, the patient noticed that his fatigue, fevers, chills, and night sweats had reappeared. When the patient returned to his PCP, a more pronounced murmur was noted. The PCP diagnosed the patient with presumptive endocarditis and admitted him to a hospital for further evaluation. A transthoracic echocardiogram revealed moderate-to-severe mitral regurgitation, a thickened and myxomatous mitral valve, and a flail posterior mitral leaflet with no definite vegetations. A transesophageal echocardiogram confirmed these results. Blood cultures grew *R. dentocariosa* sensitive to penicillin G. A dental examination and radiographs showed no evidence of an oral cause. On his last day of admission, the patient reported decreased sensations in his right cheek, as well as floaters in his right eye. Neurologic evaluation, magnetic resonance imaging (MRI), and magnetic resonance angiography did not reveal any pathologic condition. The patient was discharged on daily penicillin G (21 million U) via continuous pump.

Two weeks after discharge from the hospital, the patient woke up with pain, erythema, floaters, and complete loss of vision in his right eye. He presented on an emergency basis to an ophthalmologist, who diagnosed him with endogenous endophthalmitis. A gram stain revealed no organisms and few neutrophils. He received intravitreal vancomycin and ceftazidime empirically, was started on ofloxacin eyedrops, and was asked to return in 3 days. On his return, he agreed that his symptoms had improved. Cultures taken earlier remained negative. During this follow-up period, the ophthalmologist recommended that the patient be admitted to the hospital for further evaluation.

The patient was admitted to our institution with subacute bacterial endocarditis and secondary endogenous endophthalmitis. Pertinent findings included right-eyelid edema, conjunctival injection, right-sided facial numbness, right 3rd-digit splinter

hemorrhage, and a holosystolic murmur heard at the apex, with radiation to the axilla.

A computed tomographic scan of the head showed evidence of cerebral infarct, with a small amount of intracranial hemorrhaging. Subsequent MRI findings suggested a septic embolism. A diagnostic angiogram ruled out a cerebral mycotic aneurysm. Definitive cardiothoracic surgical treatment followed. When the mitral valve was exposed, pertinent findings correlated well with the prior echocardiograms. There was also evidence for infection at P2, which infection extended to the annulus. Because a valve repair would clearly have left residual areas of infection, we decided to replace the valve. The patient had an uneventful postoperative period and was discharged from the hospital with 2 weeks of further intravenous penicillin G and antibiotic eyedrops.

Discussion

Only a few cases of *R. dentocariosa* endocarditis have been reported to date. Boudewijns and colleagues⁶ reviewed all cases reported in the literature up to 2002, totaling 20 patients. Since then, only 3 other cases of *R. dentocariosa* endocarditis have been reported.⁷⁻⁹ In these 23 patients, underlying heart conditions predominated as the predisposing risk factor: only 2 of the 23 had no known prior heart conditions. Seven had no concomitant risk factors—usually identified as dental in origin (for example, dental caries or recent oral procedures). Of note, all 7 patients had valvular disease.

Our patient's case is remarkable because he was a previously healthy man with no recent dental manipulation, or dental or periodontal disease. We speculate that our patient either had an occult oral source of *R. dentocariosa* or that his incidental murmur arose from undiagnosed mitral disease of that same bacterial origin.

This report is one of few pertaining to *R. dentocariosa* as a pathogen responsible for endocarditis, with embolic sequelae that provide further evidence of the organism's invasive potential.⁶⁻¹¹ *R. dentocariosa* has occasionally been reported to cause infection such as endocarditis, and has even been isolated as a cause of peritonitis, septic arthritis, and pneumonia.^{2,3,6,12} It most frequently manifests itself in patients who have prior heart conditions with a clinical course that is typically subacute but accompanied by a high rate of sequelae that necessitate prompt surgical intervention. In particular, the reported incidence of aneurysms is as high as 25%.⁶

Because of the rarity of pathogenic *R. dentocariosa*, risk factors for invasive infection are not well defined. Similar uncertainty surrounds the subject of treatment regimens. No definite guidelines are available, but distinct antibiotic agents have been recognized. A variety of agents have been described: of these, penicillin is the treatment of choice, and ceftriaxone has had similar success. Most patients are treated for at least 4 to 6 weeks.^{6,8}

It is of interest that 2 case reports^{13,14} question the usefulness of vancomycin despite (in one of these cases) documentation of in vitro susceptibility.

Up to 85% of infective endocarditis cases are attributed to staphylococci, streptococci, and enterococci. However, clinicians should be aware of the organisms responsible for the remaining 15% of cases.¹⁵ Infective endocarditis caused by *R. dentocariosa* is mostly a subacute process with low rates of mortality, but with frequent cardiac and noncardiac sequelae. The universal susceptibility to penicillin makes this antibiotic agent the first choice for therapy, but additional sequelae might necessitate prompt surgical intervention.

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