Pasteurella ureae Endocarditis

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***Pasteurella ureae* is found in the normal human respiratory flora. We encountered a case of endocarditis caused by *Pasteurella ureae*. The patient was a 59-year-old man with a history of *Staphylococcus aureus* endocarditis. After treatment with antibiotics, blood cultures became negative, and the patient recovered completely. The incidence of endocarditis due to this organism is very rare; this case is the second clinically diagnosed case report. (Internal Medicine 32: 872–874, 1993)**

Key words: infective endocarditis, gram-negative coccobacillus, mitral valve

Introduction

Pasteurella ureae is a small gram-negative coccobacillus, which is part of the normal flora of the human nose and throat (1, 2), and which has rarely been identified as a pathogen in man. *Pasteurella ureae* endocarditis is particularly rare and only one clinically diagnosed case has appeared in the literature (3). Recently, we encountered a case of endocarditis due to this organism; this report constitutes the second clinically diagnosed case.

Case Report

A 59-year-old man was admitted to our hospital in August 1992, because of recurrent attacks of fever, malaise and pallor. He had a history of *Staphylococcus aureus* endocarditis in October 1985; two-dimensional echocardiography revealed moderate mitral regurgitation, but no vegetation, and he recovered with antibiotic therapy. Two weeks before the onset of recurrent attacks of fever, he had undergone a periodontal operation without the prophylactic use of antibiotics for endocarditis. He had no other noteworthy history such as recent overseas travel, use of intravenous drugs, or animal bites.

On physical examination, he was alert and ambulatory. His temperature was 38°C, pulse was 68 beats per minute, blood pressure was 122/78 mmHg and palpable. Cardiovascular examination revealed a pansystolic heart murmur at the apex. The respiratory system was unremarkable. The abdomen was soft and flat. Neither the liver nor spleen were palpable. Free fluid was not detectable in the abdomen. Skin lesions, such as Janeway lesions and splinter hemorrhages, conjunctival

petechiae, and Roth's spots were not detectable. The central nervous system appeared normal.

Laboratory analysis showed a white blood cell count of 8,100/mm³ with a shift to the left in the neutrophils, a red blood cell count of 3,700,000/mm³, hemoglobin 10.4 g/dl, hematocrit 31.7%, erythrocyte sedimentation rate (ESR) 98 mm/h, C-reactive protein (CRP) 12.18 mg/dl, serum urea 20 mg/dl, creatinine 1.0 mg/dl, sodium 140 mEq/l, potassium 4.3 mEq/l, chloride 104 mEq/l, calcium 4.2 mEq/l, iron 33 µg/ml, total protein 6.8 g/dl, glutamic oxalacetic transaminase (GOT) 15 mU/ml, glutamic pyruvic transaminase (GPT) 16 mU/ml, lactic dehydrogenase (LDH) 242 mU/ml, creatine phosphokinase (CPK) 10 mU/ml, total cholesterol 145 mg/dl and triglycerides 85 mg/dl. Proteinuria and hematuria were absent. An arterial blood gas analysis revealed pH of 7.42, PaO₂ of 84 mmHg, PaCO₂ of 42 mmHg, HCO₃ of 27 mmol/l, and O₂ saturation of 97%. Urine culture was sterile. Chest X-ray and electrocardiogram did not show any significant abnormalities.

Two-dimensional echocardiography on admission demonstrated a 11×2-mm vegetation on the posterior mitral valve leaflet (Fig. 1) with mild left ventricular enlargement. Color Doppler echocardiography revealed moderate mitral regurgitation. The size of the left ventricular cavity and the severity of mitral regurgitation remained unchanged compared with those recorded in 1985.

Bacteriology

All seven arterial blood cultures obtained on 3 consecutive days prior to antibiotic treatment grew colonies of bacteria; the isolates were identified as gram-negative, nonmotile coccobacilli that were catalase-positive and oxidase-positive, and fermented

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Fig. 1. Echocardiogram performed on admission shows a 11×2-mm vegetation on the posterior mitral valve leaflet. Ao: ascending aorta, LA: left atrium, LV: left ventricle.

glucose without gas production. Further, using the AP80 system (sensititre gram-negative autoidentification plate; SENSITITRE®, UK) and other standard biochemical methods, the isolates were identified as *Pasteurella urea*. Antibiotic testing *in vitro* demonstrated sensitivity to ampicillin, piperacillin, cefotiam hexetil hydrochloride, tetracycline, chloramphenicol, kanamycin, gentamicin, tobramycin and amikacin.

Clinical course (Fig. 2)

The patient was given intravenous piperacillin (2 g) every 8 hours and gentamicin (60 mg) every 12 hours. After one week

of treatment, the fever subsided and CRP levels fell to 3 mg/dl. CRP levels gradually decreased to within the normal range thereafter. ESR decreased to 30 mm/h 6 weeks after the start of treatment. Several blood cultures obtained during treatment became negative. The size of the vegetation remained unchanged during the hospital course. Gentamicin was withdrawn after 5 weeks due to a deterioration of renal function. Six weeks of intravenous piperacillin treatment was followed by cefotiam hexetil hydrochloride 600 mg/day per os, and the patient was discharged in good condition in October 1992.

Discussion

The genus *Pasteurella* includes the species *multocida*, *haemolytica*, *pneumotropica*, and *ureae*. Members of the genus *Pasteurella* have been rarely identified as pathogens in man, and endocarditis due to this organism is very rare (4, 5). Only a few cases of endocarditis caused by *multocida* (6–13), *haemolytica* (14, 15), *pneumotropica* (16) and *ureae* (3) have been reported. *Pasteurella multocida* and *haemolytica* are part of the normal flora of animals such as cattle, sheep, dogs, cats and fowl. Therefore, patients with endocarditis caused by these organisms typically have a history of exposure to animals, such as an animal bite or scratch. On the other hand, *Pasteurella ureae* is thought to be part of the normal human respiratory flora (1, 2), and person-to-person spread may occur; the infection appears to be due to opportunism rather than virulence. Previously Brass et al (3) reported the first case of *Pasteurella ureae* endocarditis associated with meningitis; the patient had no history of exposure to animals. In the present case, we speculate that *Pasteurella ureae* in the oral flora of the patient became a pathogen via a periodontal operation without antibiotic prophylaxis.

New episodes of endocarditis commonly occur in patients who have had infective endocarditis in the past (17). At least 6%

Fig. 2. Clinical course. PIPC: piperacillin, GM: gentamicin, CTM-HE: cefotiam hexetil hydrochloride, BT: body temperature, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate.

of patients with native valve endocarditis who are not drug abusers have one or more subsequent episodes of endocarditis (17); antibiotic prophylaxis is recommended for such patients (18). In the present case, the patient had a past history of *Staphylococcus aureus* endocarditis; therefore, at the peridontal operation he should have received prophylactic antibiotics for the prevention of infective endocarditis.

Pasteurella ureae is not particularly virulent and at present remains sensitive to penicillin, which is the drug of choice. *Pasteurella ureae* endocarditis probably occurs more frequently than is commonly appreciated. A high index of suspicion and early diagnosis, as well as prophylaxis, are warranted, particularly in patients with underlying predispositions.

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