JOURNAL OF CLINICAL MICROBIOLOGY, Sept. 1986, p. 501–502
0095-1137/86/090501-02$02.00/0

Vol. 24, No. 3

Endocarditis with *Moraxella*-Like M-6 after Cardiac Catheterization

RAMON E. PEREZ

Department of Infectious Disease, University of California, Irvine, Medical Center, Orange, California 92668

Received 4 March 1986/Accepted 3 June 1986

A patient developed bacteremia with CDC group M-6, a *Moraxella*-like bacterium, after a complicated heart catheterization. He was treated with tobramycin and ampicillin. The aortic valve was later replaced and did not show any signs of infection. The slow growth of M-6 can delay diagnosis and give misleading antibiotic susceptibility results. Penicillin is not always active against this organism.

A patient developed bacteremia with CDC group M-6 (a *Moraxella*-like bacterium) following heart catheterization. These organisms have been recovered from wounds, urine, sputum, and blood (1). They grow slowly in liquid media. This characteristic property can lead to erroneous results in antibiotic susceptibility tests. These bacteria can be detected within 24 h by the BACTEC automated blood culture system (Johnston Laboratories, Inc., Towson, Md.). However, their growth in subculture media is much slower. Not all of the strains are susceptible to penicillin. There is a previous report of endocarditis with M-6 (4).

Case report. A 57-year-old man developed palpitations and orthostatic dizziness lasting 24 h on 1 February 1982. He had rheumatic fever as a child, and a murmur of his heart was first noted in 1946. He received a 6-week treatment with intravenous penicillin for streptococcal endocarditis in 1969. Aortic insufficiency with a 30 mm Hg gradient was diagnosed in 1972. The patient remained well until presenting symptoms on 1 February 1982.

On 1 March 1982, the patient had a difficult cardiac catheterization showing a dilated left ventricle and a calcified tricuspid aortic valve with 3+ insufficiency. By 4 March 1982 he had developed generalized weakness and fever. He was hospitalized on 5 March 1982 with a temperature of 38.8°C, aortic valve murmurs (3/6 systolic and 1/6 diastolic), and bilateral femoral hematomas. His leukocyte count was 6,200, with 77% neutrophils, 3% bands, and 15% lymphocytes; his hematocrit was 44.4%, and hemoglobin was 14.7 g/dl. Mild cardiomegaly was noted by chest X ray, and his electrocardiogram showed left ventricular hypertrophy and left atrial enlargement.

Eleven sets of blood cultures were obtained over a period of 23 h between 5 and 6 March 1982. All cultures became positive within 24 h in the BACTEC system containing tryptic soy broth. Identification was made by the method of King (5), as later revised by her successors (6). The API 20E was ineffective because of slow metabolic activity and poor growth of the organism. It took 10 days from the time of detection of the organism by BACTEC to the time of its identification. Muller-Hinton agar, oxidation-fermentation broth media, and blood agar were supplied by Calscott Laboratories, Carson, Calif.

The results were corroborated by the State of California Microbial Diseases Laboratory at Berkeley (Table 1). The organism was nonmotile, gram negative, and coccobacillary and completely reduced nitrates and nitrites without gas production. It was resistant to penicillin but susceptible to

TABLE 1. Characteristics of CDC group M-6 isolate

Characteristic	Reaction or observation
Morphology	Coccobacillus
Gram reaction	−
Motility	−
Catalase	−
Oxidase	+
Oxidation-fermentation-glucose	
Open	−
Closed	−
Indole	−
Urea	−
Nitrate reduced to:	
Nitrite	−
Gas	−
Gelatin	−
Litmus milk, digested	−
Simmons citrate	−
Esculin hydrolysis	−
Triple sugar iron agar	
Slant, acid	−
Butt, acid	−
Butt, H_2S	−
Growth:	
25°C	+
35°C	+
42°C	−
Air	+
Candle jar	+
Nutrient broth	+
Nutrient broth + 6% NaCl	−
MacConkey agar	−
Salmonella-shigella agar	−
Penicillin, susceptible	−

polymyxin, aminoglycosides, and ampicillin. Antibiotic susceptibilities of the bacterium were demonstrated on blood agar plates by disk diffusion because of slow growth on standard media.

The patient was treated with tobramycin from 7 March 1982 to 2 April 1982, and with 12 g of ampicillin daily from 7 March 1982 to 19 April 1982. Tobramycin was discontinued because of nephrotoxicity, in spite of peak serum levels between 4 and 6 μg/ml. The serum trough bactericidal level read ≥1:320 while the patient was on ampicillin only. The aortic valve was replaced on 12 April 1982 for hemodynamic reasons. No bacteria were found on tissue stains of the valve, and cultures were negative. The patient has remained asymptomatic with a normally functioning prosthetic valve for the past 4 years.

The bacteria recovered from this patient resemble CDC group M-6 previously described by Tatum and co-workers (5). There are some unexplained discrepancies in recent reports of M-6. Initially, 1 of 33 strains (3%) was susceptible to penicillin (1). Subsequently, penicillin susceptibility was reported for 32 of 40 strains (88%) (2).

The initial choice of therapy for group M-6 must take into account the variable susceptibility to penicillin. Slow-growing organisms show unreliable reactions to antibiotics in liquid media. These reactions occur in tests for MICs and serum bactericidal levels (7) of antibiotics. The blood agar disk diffusion method may be more reliable than the MIC.

Routine heart catheterization usually does not require prophylaxis (3). Since this patient did not have any bacteriologic proof of endocarditis at surgery, only catheterization could be implicated in his bacteremia.

LITERATURE CITED

1. **Rubin, S. J., P. A. Granato, and B. L. Wasilauskas.** 1980. Glucose-nonfermenting gram-negative bacteria, p. 284–286. *In* E. H. Lennette, A. Balows, W. J. Hausler, Jr., and J. P. Truant (ed.), Manual of clinical microbiology, 3rd ed. American Society for Microbiology, Washington, D.C.
2. **Rubin, S. J., P. A. Granato, and B. L. Wasilauskas.** 1985. Glucose-nonfermenting gram-negative bacteria, p. 347–348. *In* E. H. Lennette, A. Balows, W. J. Hausler, Jr., and H. J. Shadomy (ed.), Manual of clinical microbiology, 4th ed. American Society for Microbiology, Washington, D.C.
3. **Shulman, S. T., D. P. Amren, A. L. Bisno, A. S. Dajani, D. T. Durack, M. A. Gerber, E. L. Kaplan, H. D. Millard, W. E. Sanders, R. H. Schwartz, and C. Watanakunakorn.** 1984. Prevention of bacterial endocarditis. Circulation **70**:1123A–1127A.
4. **Simor, A. E. and I. E. Salit.** 1983. Endocarditis caused by M-6. J. Clin. Microbiol. **17**:931–33.
5. **Tatum, H. W., W. H. Ewing, and R. E. Weaver.** 1974. Miscellaneous gram negative bacteria, p. 270–294. *In* E. H. Lennette, E. H. Spaulding, and J. P. Truant (ed.), Manual of clinical microbiology, 2nd ed. American Society for Microbiology, Washington, D.C.
6. **Weaver, R. E., D. G. Hollis, W. A. Clark, and P. Riley.** 1983. The identification of unusual pathogenic gram negative bacteria. Centers for Disease Control, Atlanta.
7. **Wolfson, J. S., and M. N. Swartz.** 1985. Drug therapy: serum bactericidal activity as a monitor of antibiotic therapy. N. Engl. J. Med. **312**:968–975.