Prosthetic Valve Endocarditis Caused by Corynebacterium diphtheriae in a Patient with Pemphigus Vulgaris

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The clinical and bacteriological findings in a case of prosthetic aortic valve endocarditis caused by Corynebacterium diphtheriae are presented. The patient died despite adequate medical therapy. This appears to be the first report of endocarditis caused by this species in a prosthetic aortic valve.

Corynebacterium diphtheriae is a well-known pathogen of the localized respiratory tract disease diphtheria (16, 22), which can be complicated by myocarditis and neuritis, caused by systemic effects of the absorbed exotoxin (6). Transmission is usually by way of infected droplets or nasopharyngeal secretions (23), infective skin exudates (2), animals, fomites, or milk. The incidence of skin infections due to C. diphtheriae is high in the tropics and subtropics (1, 18), which serve as reservoirs for the acquisition and transmission of this organism, thereby instituting respiratory tract diphtheria in tropic and temperate climates. Carriers of toxigenic C. diphtheriae in the nasopharynx or on the skin are potential sources of diphtheria for their close associates (4); they can only be detected by culture of the nasopharynx or skin (7). Diphtheria is controlled by immunization of susceptible persons with diphtheria toxoid, identification of the diphtheria carrier, and termination of the carrier state by antibiotic treatment. Heroin addiction, immunosuppression, and cardiovascular surgery have been associated with endocarditis caused by other Corynebacterium species (27), whereas nontoxigenic strains of C. diphtheriae have been associated with relatively few life-threatening infections such as bacteremia in faucial diphtheria (20, 31), endocarditis (8, 11, 19), and abscesses of the liver and spleen (14). We describe the first case of a patient with pemphigus vulgaris and a prosthetic aortic valve who developed fatal prosthetic valve endocarditis (PVE) caused by a nontoxigenic strain of C. diphtheriae.

The patient, a 55-year-old Saudi man, had a prosthetic aortic valve (St. Jude Medical, Inc., St. Paul, Minn.) replacement in October 1983 for aortic stenosis and regurgitation. Dipyridamole and aspirin were prescribed to prevent thrombus formation. In November 1985 he was admitted to our hospital with a 3-week history of flaccid bullous lesions in the nasal and oral mucous membranes and on the face, trunk, knees, and lower legs. The clinical diagnosis of pemphigus vulgaris was made and confirmed by light microscopic examination of tissue from a skin biopsy and the detection by immunofluorescence of the immunoglobulin G autoantibodies specific for epidermal intercellular cement. On examination he appeared in good health, afebrile, and with normal ear, nose, throat, and cardiovascular systems. The results of laboratory tests were as follows: hemoglobin, 13.8 g/dl; leukocyte count, 5,800/mm³, with 46% polymorphonuclear cells, 44% lymphocytes, 3% monocytes, and 7% eosinophils; erythrocyte sedimentation rate, 50 mm/h (Westergren); platelet count, 132,000/mm³; glutamic-oxaloacetic transaminase, 65 IU/liter (normal range, 0 to 31); glutamic-pyruvic transaminase, 59 IU/liter (normal range, 0 to 31); total creatine phosphokinase, 50 IU/liter (normal range, 24 to 170); lactate dehydrogenase, 2,155 IU/liter (normal range, 230 to 460); α-hydroxybutyrate dehydrogenase, 591 IU/liter (normal range, 50 to 140). Serum electrolytes, blood sugar, coagulation activity, total proteins and gamma globulins, urine analysis, and electrocardiograms were within normal limits. He was treated with dipyridamole (eight hourly 100-mg doses), aspirin (one 600-mg dose), propanolol (10 mg every 6 h), and predinosolone (starting with 75 mg/day in divided doses and gradually decreasing to 4 mg/day over a 13-day period). On his tenth day in the hospital he developed a fever of 37.5°C and a leukocyte count of 16,000/mm³, with 80% polymorphonuclear cells, 18% lymphocytes, 1% monocytes, and 1% eosinophils. Six blood culture sets (vented aerobic bottle with brain heart infusion broth, unvented anaerobic bottle with thiglycolate broth, and vented aerobic bottle with nutrient broth [Difco Laboratories, East Molesey, United Kingdom]; all bottles were supplemented with sodium polyanethol sulphonate) were obtained under sterile conditions from both antecubital fossae over a period of 6 h. The electrocardiogram was normal. Empirical antibiotic therapy was started intravenously with penicillin G (6 × 10⁹ U every 6 h), cloxacillin (500 mg every 6 h), and gentamicin (80 mg every 8 h). After overnight incubation, all blood cultures yielded small gram-positive cocci and rods typical of diphtheroids (subsequently identified as nontoxigenic C. diphtheriae by L. R. Hill, Central Public Health Laboratory, Colindale, London, United Kingdom). The isolate was identified as C. diphtheriae on the basis of its cultural characteristics on Dorset egg medium (Oxoid Ltd., Basingstoke, Hampshire, England) as a gram-positive bacillus containing metachromatic granules on Albert’s stain. It was nonhemolytic on horse blood-tellurite agar and produced a brown halo on Tinsdale medium (Oxoid). The organism produced acid only in 1% glucose and in 1% maltose. Glycogen, dextrin, starch, and sucrose were not fermented. Nitrate was reduced, and catalase was positive. Urea was not hydrolyzed. The organism was non-toxin producing by the Elek method (10). The C. diphtheriae isolate was susceptible to penicillin, ampicillin, erythromycin, and gentamicin and was resistant to cloxacillin by the disk-agar diffusion method (30). MICs were determined by the agar dilution method, as described

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by Waterworth (33). The MICs were read after 24 h of incubation at 37°C and were 0.125, 0.125, 0.0625, 0.25, 0.125, 2, and 32 μg/ml for penicillin, ampicillin, erythromycin, vancomycin, methicillin, gentamicin, and cloxacillin, respectively. Levels in serum (29) determined with samples obtained on days 2, 4, and 10, while the patient was receiving penicillin, cloxacillin, and gentamicin, were bactericidal and the antibiotics were given (trough) and 1/8 and before the initial values. The electrocardiogram disclosed normal cerebrospinal fluid showed no bacteria, and the fluid was cells respectively. Levels in serum (29) determined with samples respectively, 1 h after the antibiotics were given (peak).

There was no history of previous immunization for diphtheria. The renal function of the patient deteriorated, and on day 14 a fever of 39°C, hematuria, pyuria, shock, and coma developed.

The peripheral leukocyte count was 24,000/mm³, with 95% polymorphonuclear cells, 4% lymphocytes, and 1% monocytes. The cerebrospinal fluid was cloudy and contained a leukocyte count of 14,000/mm³, with 96% polymorphonuclear cells and 4% lymphocytes. A Gram stain of the cerebrospinal fluid showed no bacteria, and the fluid was sterile on culture. Levels of cardiac enzymes were similar to the initial values. The electrocardiogram disclosed normal sinus rhythm with only occasional supraventricular prema-

tures; otherwise, the electrocardiogram was normal. An echocardiogram (2D technique) disclosed evi-

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C. diphtheriae, early diagnosis is important, and any patient with fever should have blood taken for culture and suscep-

bility testing and 2D echocardiography should be per-

formed. The patient should be managed in a center in which a multidisciplinary team (cardiologist, cardiac surgeon, micro-

biologist, and infectious disease physician) is on hand for immediate surgical management should the clinical situation require it.

LITERATURE CITED


American Society for Microbiology, Washington, D.C.


