Prosthetic Valve Endocarditis Caused by Group Ve-1 Bacteria (Chromobacterium typhiflavum)

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A case of fatal prosthetic valve endocarditis was caused by group Ve-1 bacteria. Bacteriological characteristics and antibiotic susceptibilities are presented, as well as a brief discussion of the isolates of the Ve-1 organism in Ontario.

Group Ve-1 bacteria are yellow-pigmented, pseudomonaslike organisms (2) formerly designated *Chromobacterium typhiflavum* (3). Infections with these bacteria are rare, and most clinical isolates are from wounds or abscesses. This is the first reported occurrence of prosthetic valve endocarditis caused by group Ve-1 bacteria.

In November 1983, a 60-year-old man was admitted to hospital with severe heart failure secondary to partial dehiscence of an Omni-Science prosthetic aortic valve. There was no evidence of endocarditis, and preoperative blood cultures were sterile. The Omni-Science valve was replaced with a Bjork-Shiley prosthesis. Two weeks later the patient developed a fever, and a single blood culture grew a gramnegative, nonfermenting bacillus later identified as a member of group Ve-1. The origin of the bacteremia was not clear, and the patient was treated with a 2-week course of intravenous netilmicin and cefamandole. The organism was susceptible to both antibiotics, and two sets of blood cultures taken 48 h after treatment failed to grow any bacteria. The patient was then discharged home.

Two weeks later the patient returned in severe heart failure with a paravalvular leak. Preoperative blood cultures at this time grew group Ve-1 bacilli. At surgery an annular ring abscess was found. Both the Bjork-Shiley valve and a swab from the annular ring abscess grew group Ve-1 bacilli. Unfortunately, the patient had a cardiac arrest on postoperative day 1 and had no subsequent neurological function. He died 1 week later, and request for autopsy was refused.

The aerobic organism grown from the blood and prosthetic valve of the patient was a catalase-positive, oxidase-negative, gram-negative bacillus. On blood agar plates colonies were yellow pigmented and nonhemolytic, with a rough surface. API 20E reagent strips revealed positive reactions for *o*-nitrophenol- β -D-galactopyranoside, citrate, glucose, and arabinose, providing a code number of 1204002 which was designated as group Ve-1 by the API index. The organisms isolated on both admissions were confirmed by the Provincial Reference Laboratory as being CDC Ve-1 the microbiological characteristics listed in Table 1. Antibiotic susceptibility testing by the agar dilution method revealed that the organism was susceptible to tetracycline, tobramycin, netilmicin, and cefamandole, but resistant to ampicillin, cephalothin, and cotrimoxazole.

The Provincial Reference Laboratory for the Province of Ontario has recorded only seven isolates of group Ve-1 bacteria in the last 4 years (Dorothy Chang, personal communication). These isolates were recovered from urine, skin, burn, wound, and eye swabs. Our patient was the first whose

TABLE 1. Biochemical characteristics of group Ve-1 isolate

Test	Reaction"
Growth on MacConkey agar	+
Kligler agar slant, acid	-
Kligler agar butt, acid	-
Catalase	+
Oxidase	-
Indole	-
Motility	+
Simmons citrate	+
Nitrate reduction	+
Christensen urea	+
Lysine decarboxylase (Moeller)	-
Arginine dehydrolase	+
Ornithine decarboxylase	-
Esculin hydrolysis	+
ONPG ^{<i>b</i>}	+
H ₂ S production (Kligler)	-
Voges-Proskauer	-
Deoxyribonuclease	-
Phenylalanine deaminase	-
Pigment, yellow	+

^a +, Positive; -, negative.

^b ONPG, o-Nitrophenyl-β-D-galactopyranoside.

organism was isolated from a blood culture. Bacteremia caused by group Ve-1 organism secondary to a pancreatic abscess was recently reported by Berger et al. (1); the authors noted that this organism is rarely associated with significant clinical infection. To our knowledge, there are no previous reports in the English literature of these bacteria causing endocarditis.

This case supports the role of group Ve-1 bacilli as significant pathogens with the potential of producing blood stream infection and endocarditis. The two patients thus far described with systemic sepsis (bacteremia) due to group Ve-1 bacilli have had nosocomial infections. This suggests that this organism is more an opportunistic agent, with a greater propensity of invading human hosts in the presence of foreign bodies, intravenous catheters, and postsurgical states.

LITERATURE CITED

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