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Pseudomonas paucimobilis Bacteremia

PAUL M. SOUTHERN, JR.[1,2]* AND ANNA E. KUTSCHER[2]

Department of Pathology, The University of Texas Health Science Center at Dallas,[1] and Clinical Microbiology Laboratory, Parkland Memorial Hospital,[2] Dallas, Texas 75235

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Pseudomonas paucimobilis was isolated from the blood of a man after surgery for occlusive vascular disease of his lower extremities. Circumstances suggest that the infection was hospital associated and was possibly caused by an organism present in the surroundings of this particularly susceptible host. An environmental source was not found. The isolate was susceptible in vitro to carbenicillin, chloramphenicol, tetracycline, and trimethoprim-sulfamethoxazole and was moderately susceptible to amikacin and ampicillin. This case represents the fourth reported incidence of infection due to *P. paucimobilis*.

Pseudomonas paucimobilis has only recently been described as having human clinical significance. This organism was previously listed by Tatum and colleagues at the Centers for Disease Control as belonging to the "*Pseudomonas*-like" group of bacteria and was designated group IIk-1 (6). Holmes et al. made a further study of the group and proposed the name *P. paucimobilis* (3). This epithet has been generally accepted since then. Both the group at the Centers for Disease Control and the one in London have reported isolating the organism from environmental sources (including hospitals) and human clinical specimens, but neither could be certain as to the significance of the organism as a pathogen. However, *P. paucimobilis* has since been isolated in pure culture from a leg ulcer (4), from the blood of a septicemic man (5), and from the cerebrospinal fluid of a man with acute meningitis (2). This report documents the isolation of *P. paucimobilis* from the blood of a septicemic man in a setting suggesting hospital-associated infection.

CASE REPORT

A 61-year-old man with a history of ethanol abuse and probable narcotic addiction was admitted to Parkland Memorial Hospital in August 1979 for evaluation of intermittent claudication. He also had a history of recurrent bronchitis and hypertension and had been thought to have peripheral neuropathy in early 1979. Outpatient evaluations in early 1979 which showed sclerodactyly and an antinuclear antibody titer of 1:25,000, speckled, also suggested the possibility of a collagen or vascular disease. Upon admission to the hospital, he had a blood pressure of 160/90 mm Hg (21.33/12 kPa), a regular pulse rate of 92, a normal temperature, spider telangiectasias, moderate hepatic enlargement, nodular scars over deltoid areas from prior injections, sclerodactyly, and no palpable pulses below the femoral arteries in either lower extremity. Aortography revealed abdominal aortic atherosclerosis, occlusion of the right common iliac artery, and severe compromise of the left iliac artery. On day 3 after admission, he underwent an aortobifemoral bypass operation with insertion of a Dacron graft. Beginning 12 h after surgery and continuing for 30 days, the patient was pyrexial almost continuously, with peak temperatures reaching from 38.1 to 39.5°C daily. During this time, his clinical course was complicated by the development of pneumonitis and basilar pulmonary atelectasis, a gradual loss of circulation to the left foot and lower leg, mental disorientation, a purpuric rash over the trunk and thighs, rectal bleeding, and thrombocytopenia (platelet count, 34,000 per μl). The latter was thought to be heparin induced; therefore, therapy with coumadin was substituted. However, because of continued hemorrhagic phenomena, all anticoagulants were discontinued 15 days after surgery. Shortly thereafter, he developed hemorrhagic vesicles on his left hip that were thought to represent either ecthyma gangrenosum or vasculitis due to an underlying disorder or to be related to coumadin or antibiotic therapy. Blood cultures taken at that time (17 days after surgery) ultimately yielded *P. paucimobilis*. Because of continued compromise of the circulation, a partial amputation of the left foot was performed 18 days after the first operation. Thereafter, several extensive surgical debridements of the necrotic hip lesion were required, and ultimately the area necessitated skin grafting. Both the graft site and the donor site became purulent but eventually healed after aggressive surgical care. The amputation stump healed very slowly; a delayed closure was accomplished 4 weeks after the necrotic front of the foot was removed.

Blood cultures taken on days 15, 26, and 34 (two sets per day) after the first operation were sterile. Cultures of the necrotic hip lesion taken on days 16, 20, and 24 after the first operation and multiple cultures of other draining wounds and operative sites yielded only skin-associated organisms (*Staphylococcus aureus*, *Staphylococcus epidermidis*, *Corynebacterium* sp., and an enterococcus). *P. paucimobilis* was

TABLE 1. *Characteristics of isolates from blood compared with those of P. paucimobilis*[a]

Characteristic	Reaction of isolates from:	
	Blood	*P. paucimobilis*
Action on blood		
Lysis	−	− to indeterminate
Color	Slight green	Slight green
CO_2 required	−	−
Pigment in colony	Deep yellow	Deep yellow
Flagella	1 polar	1 polar
Enrichment required	None	None
Triple sugar iron agar	Alk./Alk.	N/N or Alk./N
Oxidase (tetramethyl-*p*-phenylenediamine dihydrochloride)	+	+ (75%)
Catalase	+	+
Growth on		
MacConkey agar	(+, slight at 4 days)	d (4 to 19%, delayed)
SS agar	−	−
Cetrimide agar	−	−
H_2S		
Triple sugar iron agar	−	−
Lead acetate paper	Tr	(+)
Oxidative or fermentative	O	O or F
Urea (Christensen agar)	−	− or (+)
Indole	−	−
MR/V-P	−	−
Citrate (Simmons)	−	− or (+)
Motility (hanging drop and Gard plate)	−	− or (+)
Gelatin	−	− or (+)
Moeller decarboxylase		
Lysine	−	−
Arginine	−	−
Ornithine	−	−
Litmus milk (peptonization)	−	− or (+)
Nitrate reduction	−	− or (+)
Nitrate reduction to gas	−	−
Carbohydrates utilized (King O-F basal medium)		
Glucose	(+, 7 days)	+
Xylose	(+, 4 days)	+
Mannitol	−	−
Lactose	(+, 7 days)	+
Sucrose	(+, 7 days)	+
Maltose	(+, 7 days)	+
Esculin	(+, 2 days)	+ or (+)
Growth at (°C):		
25	+	+
37	+	+
42	−	Variable

[a] As described by Tatum et al. (6). Symbols: −, no reaction, negative, +, positive reaction; (+), delayed reaction; Alk., alkaline; N, neutral; O, oxidative; F, fermentative; MR/V-P, methyl red or Voges-Proskauer; d, different reactions.

isolated from only two separate sets of blood cultures taken on day 17 after the first operation. The patient received a wide array of antimicrobial agents, including cefamandole, cephalexin, cephalothin, erythromycin, and tobramycin, in various doses and combinations. At the time of his positive blood cultures, he was receiving cephalothin and erythromycin. Tobramycin was begun 3 days later and was continued for 8 days. Erythromycin was continued until 8 days after the positive blood cultures. Various cephalosporin agents were administered later for approximately 6 weeks.

The patient was discharged from the hospital in December 1979 and was seen only once thereafter in follow-up by a consultant in clinical rheumatology. The consultant thought the patient probably had either scleroderma or a mixed connective tissue disorder. At that time, the patient appeared to be free of infection.

RESULTS

Yellow-pigmented, gram-negative, nonfermentative bacilli were isolated from two separate sets of blood cultures as described above. The blood was inoculated into 100-ml bottles of tryptic digest of soy broth with 0.025% polyanethol sulfonate added. Two bottles per set were then inoculated with approximately 10 ml of blood each. In each set, one bottle was vented, and the other remained unvented. The original isolation was obtained from both bottles in each set after being subcultured onto chocolatized sheep blood agar plates that were incubated at 35°C in 5% CO_2 in air (subcultures from the bottles were performed during the first 24 h in the laboratory). Growth on the chocolate agar plates was evident within 24 h after the subculture was performed. Visible growth in the original blood culture bottles was evident within 48 h after inoculation. Biochemical and other characteristics of the organisms are given in Table 1. In vitro antimicrobial susceptibility testing was carried out with an agar plate dilution method

TABLE 2. *In vitro antimicrobial susceptibility of P. paucimobilis isolated from blood of a septicemic patient*

Agent	Minimum inhibitory concn (μg/ml)
Amikacin	16
Ampicillin	16
Carbenicillin	64
Cefamandole	>32
Cefoxitin	>32
Cephalothin	32
Chloramphenicol	8
Colistimethate	>16
Gentamicin	16
Kanamycin	>32
Tetracycline	8
Tobramycin	>16
Trimethoprim-sulfamethoxazole	5.25[a]

[a] Represents 0.26 μg of trimethoprim and 4.99 μg of sulfamethoxazole.

similar to that described by Washington and Sutter (7). The results of this test are presented in Table 2. Identification of the organism as *P. paucimobilis* was confirmed in the bacteriology laboratory of R. E. Weaver at the Centers for Disease Control.

DISCUSSION

Originally designated as group IIk-1, the organism now known as *P. paucimobilis* was first isolated before 1979 from numerous human clinical specimens, the hospital environment, and other environmental sources without much documentation of its potential pathogenicity (3, 6). Holmes and colleagues characterized 47 strains of yellow-pigmented, nonfermentative, gram-negative bacilli from various sources and proposed the name *P. paucimobilis* for the species (3). This epithet has been recognized generally since that time. The legitimacy of this designation was strengthened by the work of Dees et al., who showed that the cellular fatty acid composition of *P. paucimobilis* group IIk-1 was readily distinguishable from that of groups IIk-2, Ve-1, and Ve-2 (1). In 1979, the first three reports ascribing human clinical significance to *P. paucimobilis* appeared in the literature. Peel et al. isolated the organism in pure culture from an infected leg ulcer on a seaman in a hospital in Australia (4). In England, Hajiroussou et al. later isolated the organism from the cerebrospinal fluid of a man with meningitis and a background of chronic epilepsy (2). Finally, Slotnick et al. in Los Angeles isolated *P. paucimobilis* from the blood of a man with fever, chest pain, pulmonary embolization, and a background of chronic atrial fibrillation (5). The patient herein described represents a fourth clinically significant case of infection due to *P. paucimobilis*. Although it has been suggested that this organism might easily be implicated in hospital-acquired infection, our patient is the first to be reported in whom this was a likely possibility. Earlier reports have noted the isolation of the organism from hospital sources but have failed to make a direct link in a specific patient (3, 5, 6). The present case was complicated, and the sequence of events suggested that the infection was hospital associated. A limited environmental search was made for a similar organism in the surroundings of the patient, but none was found. It was not, however, an exhaustive, systematic, or timely search.

Previous reports have stated that clinical isolates of *P. paucimobilis* were susceptible in vitro to tetracycline, kanamycin, gentamicin, sulfamethoxazole, chloramphenicol, and tobramycin (4); to ampicillin, carbenicillin, chloramphenicol, gentamicin, tobramycin, kanamycin, tetracycline, and co-trimoxazole (5); and to rifampin and, by implication (data not shown), to carbenicillin and gentamicin (2). We report results of susceptibility testing by agar dilution. The isolates were susceptible in vitro to carbenicillin, chloramphenicol, tetracycline, trimethoprim-sulfamethoxazole, and probably to amikacin and ampicillin (Table 2). Because our patient did not receive antimicrobial agents to which the organisms were susceptible in vitro, it is likely that his survival of infection was owing mainly to aggressive surgical treatment of the presumed local source, i.e., the necrotic ulcer on his hip.

We emphasize the potential of this organism for causing nosocomial infections in susceptible hosts. Our patient had severely compromised circulation to his lower extremities, probable chronic problems due to ethanol and narcotics abuse, and perhaps an underlying collagen or vascular disease. These factors, when compounded by vascular surgery, qualified him as a susceptible host. In such patients, the development of in-hospital infection due to a yellow-pigmented, nonfermentative, gram-negative bacillus gives rise to the possibility that *P. paucimobilis* is responsible. The confirmation of this hypothesis will require extensive biochemical characterization. On the basis of this and other reported cases of infection by this organism, it appears that the antimicrobial susceptibility of *P. paucimobilus* may be somewhat variable but may include chloramphenicol, tetracycline, and trimethoprim-sulfamethoxazole. Rational antibiotic therapy will require in vitro susceptibility testing on each clinical isolate.

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