JOURNAL OF CLINICAL MICROBIOLOGY, June 1992, p. 1583–1584
0095-1137/92/061583-02$02.00/0

Vol. 30, No. 6

Pseudomonas mendocina, an Environmental Bacterium Isolated from a Patient with Human Infective Endocarditis

MARIA DEL ROSARIO ARAGONE,[1]* DIEGO MARCELO MAURIZI,[2] LILIANA OFELIA CLARA,[2] JOSE LUIS NAVARRO ESTRADA,[3] AND AMANDA ASCIONE[1]

Bacteriology Section, Central Laboratory,[1] Infectious Diseases Section, Internal Medicine Division,[2] and Division of Cardiology,[3] Hospital Italiano, Gascon 450, 1181 Capital Federal, Argentina

Received 25 November 1991/Accepted 16 March 1992

***Pseudomonas mendocina* has been isolated from soil and water samples. Although it has been recovered from some human clinical samples, its pathogenic role has not yet been documented. We report the first known case of endocarditis in humans due to *P. mendocina*.**

Pseudomonas mendocina was first isolated by N. J. Palleroni from soil and water samples collected in the province of Mendoza, Argentina (4). Its recovery from human clinical specimens other than urine and leg ulcers has not been documented, and its association with human infections is otherwise unknown (3). We have recently isolated a strain of *P. mendocina* from several blood samples from a man with a probable endocarditis according to Fordham von Reyn's criteria (2).

Case report. A 63-year-old man was admitted to our hospital on 17 April 1991, owing to high fever. He had been well until 5 days before admission, when his temperature had risen to 39 to 39.5°C, accompanied by shivering. His previous medical history includes poliomyelitis at the age of 2 years, type II diabetes, aortic valve replacement with mechanical prosthesis in 1987 owing to calcified aortic stenosis, and implantation of a permanent pacemaker due to complete atrioventricular block in January 1990. The patient is a florist. He is not an endovenous drug addict. Petechiae were observed on both legs, and a hemorrhage was observed beneath the nail of the first right toe. He had small erythematous lesions on the fingertips of both hands, which were attributed to thorn pricks. Auscultation confirmed a systolic aortic murmur reported in previous medical records (1987 to 1990) and revealed a new systolic murmur audible at the apex.

Six sets of blood cultures were obtained between 17 and 19 April 1991; 48 h after admission, treatment with cephalothin (12 g/day, intravenously [i.v.]) and gentamicin (160 mg/day, i.v.) was begun, with remarkable improvement within 24 h. A transthoracic echocardiogram revealed a thickened native mitral valve but no abnormalities on the prosthetic valve; a transesophageal color Doppler echocardiogram performed 10 days later showed a vegetation on the anterior mitral valve with mitral insufficiency. All blood cultures grown in tryptic soy broth yielded gram-negative bacilli within 72 h. Therapy was consequently changed to ceftriaxone (2 g/day, i.v.) and gentamicin (160 mg/day, i.v.); the serum trough and peak bactericidal levels were 1:128 and 1:256, respectively, by using a macrodilution method with cation-supplemented Mueller-Hinton broth as the diluent (6). Because the patient had a favorable course and because clinical and echocardiographic examinations failed to reveal abnormalities on the prosthetic valve, surgery was not considered and i.v. antibiotic treatment continued for 6 weeks, followed by 2 weeks of ciprofloxacin (1.5 g/day, per os). One month after completing antibiotic treatment, the patient was doing well, control blood cultures were negative, and a transesophageal echocardiogram showed a 5-mm vegetation on the anterior mitral valve with moderate mitral insufficiency and a normal prosthetic aortic valve. A new control at 6 months showed that the patient was still asymptomatic, with normal laboratory data and negative blood cultures.

Microbiological features. Colonies were flat, smooth, butyrous, nonwrinkled, and of a brown-yellow color on 5%

TABLE 1. Biochemical characteristics of the isolated strain

Pigment	Slightly yellow
Action on blood	Lysis
Oxidase	+
Triple sugar iron agar activity	
Slant, acid	−
Butt, acid	−
Butt, H_2S	−
Motility, flagella	+ (monotrichous polar)
Catalase	+
Acid from (carbohydrate base, O-F medium):	
Glucose	+
Xylose	+
Mannitol	−
Lactose	−
Sucrose	−
Maltose	−
Starch	−
Indole	−
Growth at or in:	
25°C	+
35°C	+
42°C	+
Nutrient broth, 6% NaCl	+
Salmonella-shigella agar	+
Simmons citrate utilization	+
Nitrate reduction	+
Gas from nitrate	+
H_2S (Pb acetate paper)	+
Lysine decarboxylase	−
Ornithine decarboxylase	−
Arginine dihydrolase	+
Acetamide assimilation	−
Gelatin hydrolysis	−
Esculin hydrolysis	−

* Corresponding author.

TABLE 2. Minimum useful characteristics to differentiate *P. mendocina* from other *Pseudomonas* spp.[a]

Organism	Flagellar insertion	Pigment type	Acid from (in O-F medium):		Gas from nitrate	Acetamide	Arginine dihydrolase	Growth at 42°C
			Maltose	Starch				
P. mendocina	Monotrichous polar	Carotenoid	−	−	+	−	+	+
P. stutzeri	Monotrichous polar	Carotenoid	+	+	+	−	−	Variable
P. aeruginosa	Monotrichous polar	Fluorescent phenazine	−		+	+	+	+
P. fluorescens	Tuft polar	Fluorescent	−		−	−	+	−

[a] References 4 and 7.

sheep blood agar. The organism grew on MacConkey agar and was oxidase positive; triple sugar iron agar showed no change, and hydrogen sulfide was not produced. It was motile; the Leifson's stain made from an agar slant showed a polar monotrichous flagellum and several lateral ones of shorter wavelength. On 5% sheep blood agar, there was a diffuse lysis of erythrocytes extending out from heavy growth. It was positive for catalase production, growth on salmonella-shigella agar, growth at 42°C and in nutrient broth with 6.5% NaCl, utilization of citrate (Simmons), nitrate reduction with gas production, arginine dihydrolase, hydrogen sulfide production with the papers trip test, and acid from glucose and xylose in oxidation-fermentation (O-F) medium. It was negative for indole production, gelatin and esculin hydrolysis, acetamide assimilation, lysine and ornithine decarboxylase, and acid from mannitol, lactose, sucrose, maltose, and starch in O-F medium (Table 1). On the basis of these reactions, the isolate was identified as *P. mendocina* (3, 7). Confirmation of this identification was made by the Special Bacteriology Section of the Centers for Disease Control, Atlanta, Ga.

The organism was susceptible to amikacin, gentamicin, pefloxacin, piperacillin, ceftazidime, trimethoprim-sulfamethoxazole, ceftriaxone, colistin, netilmicin, and ciprofloxacin and was resistant to ampicillin and cephalothin in a standard disk assay (1). The MICs and MBCs, respectively, for the following antimicrobial agents by broth macrodilution (5) were as follows (in micrograms per milliliter): amikacin, 0.5 and 1.0; gentamicin, 0.25 and 0.50; trimethoprim-sulfamethoxazole, ≤0.25/4.75 and ≤0.25/4.75; ciprofloxacin, 0.125 and 0.125; piperacillin, 0.62 and 0.62; ceftazidime, 1.0 and 1.0; and ceftriaxone, 4.0 and 4.0.

P. mendocina shares phenotypic characteristics with *Pseudomonas stutzeri* and members of the fluorescent pseudomonad group (particularly *Pseudomonas aeruginosa*) but possesses some features that are unique. It can be distinguished from *P. stutzeri* by differences in the colonial morphology (*P. stutzeri* usually forms dry and wrinkled colonies) and in some nutritional respects: it fails to use starch and maltose and possesses arginine dihydrolase. It differs from *P. aeruginosa* in its inability to assimilate acetamide and in its pigmentation (neither fluorescent nor phenazine pigments are produced). Finally, it also differs from *Pseudomonas fluorescens* in its pigmentation, in the presence of a polar monotrichous flagellum, and in its ability to grow at 42°C and to grow with abundant gas production in media that contain sufficient nitrate (Table 2) (4, 7).

As far as we know, *P. mendocina* has not yet been isolated from humans as an infectious agent. It has been recovered from human urine and leg ulcers, but its pathogenic role remains unknown (3). This is the first reported case in which *P. mendocina* has been isolated from human blood samples. All of the clinical, echocardiographic, and microbiological evidence strongly suggests that this patient's endocarditis was of the native mitral valve and was due to *P. mendocina*. Curiously enough, there was no evidence to suggest involvement of the prosthetic valve or its adjacent tissue; the reason for this paradoxical behavior (that is, preference for a native valve rather than a prosthetic valve) remains unclear. It is surprising that an endocarditis due to a *Pseudomonas* strain should have been resolved without surgery; very likely its particular antimicrobial susceptibility explains this fact.

Because the species has been isolated from soil and water (4), we think that the bacteria entered the bloodstream through thorn pricks and the handling of damp earth in view of this man's occupation. We believe that from now on *P. mendocina* should be regarded as a potential human pathogen.

We thank R. E. Weaver of the Special Bacteriology Reference Laboratory, Meningitis and Special Pathogens Branch, Division of Bacterial Diseases, Centers for Disease Control, Atlanta, Ga., for his approval of the identification of the bacterium.

REFERENCES

1. **Barry, A. L., and C. Thornsberry.** 1990. Susceptibility tests: diffusion test procedures, p. 1117–1125. *In* A. Balows, W. J. Hausler, Jr., K. L. Herrmann, H. D. Isenberg, and H. J. Shadomy (ed.), Manual of clinical microbiology, 5th ed. American Society for Microbiology, Washington, D.C.
2. **Fordham von Reyn, C., B. S. Levy, R. D. Arbeit, et al.** 1981. Infective endocarditis: an analysis based on strict case definition. Ann. Intern. Med. **94(part 1)**:505–518.
3. **Giraldi, G. L.** 1990. *Pseudomonas* and related genera, p. 429–441. *In* A. Balows, W. J. Hausler, Jr., K. L. Herrmann, H. D. Isenberg, and H. J. Shadomy (ed.), Manual of clinical microbiology, 5th ed. American Society for Microbiology, Washington, D.C.
4. **Palleroni, N. J., M. Doudoroff, and R. Y. Stainer.** 1970. Taxonomy of the aerobic pseudomonas: the properties of the Pseudomonas stutzeri group. J. Gen. Microbiol. **60**:215–231.
5. **Sahn, D. F., and J. A. Washington II.** 1990. Antibacterial susceptibility tests: dilution methods, p. 1105–1116. *In* A. Balows, W. J. Hausler, Jr., K. L. Herrmann, H. D. Isenberg, and H. J. Shadomy (ed.), Manual of clinical microbiology, 5th ed. American Society for Microbiology, Washington, D.C.
6. **Schoenknecht, F. D., L. D. Sabath, and C. Thornsberry.** 1985. Special susceptibility tests, p. 1239–1249. *In* E. H. Lennette, A. Balows, W. J. Hausler, Jr., and H. J. Shadomy (ed.), Manual of clinical microbiology, 4th ed. (Spanish translation). American Society for Microbiology, Washington, D.C.
7. **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.