

Bacteremia Due to *Cedecea neteri* sp. nov.

J. J. FARMER III,^{1*} NEELA K. SHETH,^{2,3} JUNE A. HUDZINSKI,² HAROLD D. ROSE,^{3,4} AND MARY F. ASBURY¹

*Nosocomial Infections Laboratory Branch, Hospital Infections Program, Center for Infectious Diseases, Centers for Disease Control, Atlanta, Georgia 30333*¹; *Microbiology Section, Laboratory Service,*² *and Infectious Disease Section, Medical Service,*⁴ *Veterans Administration Center, Wood (Milwaukee), Wisconsin 53193;* and *Departments of Pathology and Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin 53226*³

Received 5 April 1982/Accepted 18 June 1982

Three of four blood cultures from a patient with possible endocarditis were positive for a new species of *Enterobacteriaceae* which is named *Cedecea neteri*. This is the first reported case of bacteremia caused by a strain of *Cedecea*.

The name *Cedecea* was recently proposed for a unique new genus in the family *Enterobacteriaceae* previously known as Enteric Group 15 (4). Strains of *Cedecea* resemble strains of *Serratia* because they are lipase (corn oil) positive and are resistant to cephalothin and colistin. However, unlike *Serratia* strains, *Cedecea* strains do not hydrolyze gelatin or DNA. Two species were originally named in *Cedecea*, *C. davisae* and *C. lapagei*, but DNA-DNA hybridization indicated that three other groups were distinct and should be considered as three additional species (4). These latter three groups were not given scientific names because only one strain was available for each. Instead, the names "Cedecea species 3," "Cedecea species 4," and "Cedecea species 5" were used (4). None of the original 17 strains of *Cedecea* were from blood or spinal fluid, so there was doubt whether *Cedecea* would prove to be clinically significant. Similarly, Bae and co-workers (1) recently reported two isolates of *C. davisae* that were from sputum, but the role of these isolates in causing pneumonia was doubtful. Since the original description of *Cedecea* species 4, three additional strains have been studied. Because the isolate of *Cedecea* species 4 in the present case report was clinically significant, we propose the scientific name *Cedecea neteri* sp. nov. to facilitate communication about this new species.

A 62-year-old white man with a history of valvular heart disease was admitted to the Veterans Administration Hospital, Wood (Milwaukee), Wis., on 30 May 1981 because of recurrent fever and shaking chills of 4 days' duration. The illness began abruptly and was accompanied by painful urination. Four weeks before the patient's admission, a dentist had prescribed an oral antimicrobial agent for 2 days before filling a cavity. The patient's history was not signifi-

cant except for a heart murmur since the age of about 22. In 1976, aortic valvular stenosis and insufficiency were found when cardiac catheterization was done.

Physical examination indicated that the man was acutely ill but alert. The temperature was 101.8°F (38.8°C), the pulse rate was 80 beats per min, and the blood pressure was 120/80. There was a harsh grade III holosystolic murmur at the base of the heart and a high-pitched grade I diastolic murmur along the left sternal border. There were no signs of congestive heart failure, and there were no extracardiac findings of bacterial endocarditis or costovertebral angle tenderness. An examination done on hospital day 2 indicated that the prostate was firm and not tender.

Laboratory findings included a hemoglobin level of 13.3 g per 100 ml, a hematocrit reading of 40%, and a leukocyte count of 12,700, with a left shift of the differential cell count. Urinalysis was normal except for 3+ protein (Chemstrip 7; Bio-Dynamics, Indianapolis, Ind.). Microscopic examination of urine on hospital day 1 (before antibiotics) indicated 0 to 4 leukocytes and 0 to 2 erythrocytes per high-power field. The results were similar on hospital days 2 and 15. The rheumatoid arthritis latex test was negative. A chest X ray showed only minimal enlargement of the heart, but an electrocardiogram indicated left ventricular hypertrophy (enlargement of the myocardium of the ventricle). X rays of the gastrointestinal tract and ultrasonography of the genitourinary tract failed to show a structural abnormality. Three urine cultures taken on hospital days 1 and 2 (one was taken before antibiotics were started) were sterile. A sputum culture taken on hospital day 2 grew normal flora only. On hospital day 1, before antibiotics were started, four blood culture sets were drawn, each set

TABLE 1. Biochemical reactions of *C. neteri* and the type strain

Test	Cumulative % of four strains positive at:			Reaction ^a of:	
	24 h	48 h	7 days	Type strain (ATCC 33855)	Patient isolate (1238-81)
Indole production	— ^b	0	—	—	—
Methyl red	—	100	—	+	+
Voges-Proskauer	—	100	—	+	+
Citrate (Simmons') utilization	100	100	100	+	+
H ₂ S production (triple sugar iron agar)	0	0	0	—	—
Urea (Christensen's)	0	0	0	—	—
Phenylalanine deaminase	0	—	—	—	—
Lysine decarboxylase (Moeller's)	0	0	0	—	—
Arginine dihydrolase (Moeller's)	100	100	100	+	+
Ornithine decarboxylase (Moeller's)	0	0	0	—	—
Motility	100	100	100	+	+
Gelatin liquefaction at 22°C	0	0	0	—	—
Growth in KCN	0	75	75	—	+ ²
Malonate utilization	100	100	100	+	+
D-Glucose-acid production	100	100	100	+	+
D-Glucose-gas production	100	100	100	+	+
Acid production from:					
Lactose	0	50	100	+ ⁴	+ ²
Sucrose	100	100	100	+	+
D-Mannitol	100	100	100	+	+
Dulcitol	0	0	0	—	—
Salicin	100	100	100	+	+
Adonitol	0	0	0	—	—
<i>i</i> -(<i>myo</i>)Inositol	0	0	0	—	—
D-Sorbitol	100	100	100	+	+
L-Arabinose	0	0	0	—	—
Raffinose	0	0	0	—	—
L-Rhamnose	0	0	0	—	—
Maltose	100	100	100	+	+
D-Xylose	75	100	100	+ ²	+
Trehalose	100	100	100	+	+
Cellobiose	100	100	100	+	+
α-Methyl-D-glucoside	0	0	100	+ ⁴	+ ⁴
Erythritol	0	0	0	—	—
Melibiose	0	0	0	—	—
D-Arabitol	100	100	100	+	+
Glycerol	0	0	75	+ ⁷	—
D-Mannose	100	100	100	+	+
D-Galactose	100	100	100	+	+
Mucate-acid production	0	0	0	—	—
Tartrate (Jordan's)	0	0	0	—	—
Esculin hydrolysis	75	100	100	+	+ ²
Acetate utilization	0	0	0	—	—
Citrate (Christensen's)	100	100	100	+	+
Lipase (corn oil)	100	100	100	+	+
DNase at 25 or 36°C	0	0	0	—	—
Oxidase (Kovács')	0	—	—	—	—
NO ₃ ⁻ → NO ₂ ⁻	100	—	—	+	+
ONPG ^c test	100	100	100	+	+
Pectate hydrolysis	0	0	0	—	—
Pigment production	0	0	0	—	—
Tyrosine clearing	0	0	0	—	—

^a +, Positive at 24 h; —, negative at the end of the incubation period; superscript numbers indicate the day the reaction became positive.

^b —, Not done.

^c ONPG, *o*-Nitrophenyl-β-D-galactopyranoside.

consisting of an aerobic blood culture flask containing 75 ml of tryptic soy broth (Difco Laboratories, Detroit, Mich.) with 0.3 sodium citrate, to which 7 to 10 ml of blood was added, and an anaerobic blood culture bottle containing thiol broth with sodium polyanetholesulfonate and CO₂ (Difco), to which 5 ml of blood was added. The sets were taken at 5:15 p.m. (the resultant blood cultures are referred to as aerobic 1 and anaerobic 1), 6:15 p.m. (aerobic 2 and anaerobic 2), 9:45 p.m. (aerobic 3 and anaerobic 3), and 11:30 p.m. (aerobic 4 and anaerobic 4). After incubation, five of the eight blood culture bottles (aerobic 1, aerobic 3, anaerobic 3, aerobic 4, and anaerobic 4) were positive. The time necessary for the bottles to become turbid was recorded in two instances; aerobic bottle 1 became positive at 14 h, and anaerobic bottle 4 became positive at 36 h. The positive bottles yielded a gram-negative, oxidase-negative rod which had a biochemical profile number of 3 205 721 when tested by the API 20E system (Analytab Products, Plainview, N.Y.). The most likely identifications given by the API computer identification service were Enteric Group 15 (*Cedecea*) and *Enterobacter agglomerans*. The organism was susceptible, as tested by the Kirby-Bauer disk method, to cefamandole, chloramphenicol, tetracycline, gentamicin, tobramycin, and amikacin but was resistant to colistin, cephalothin, and ampicillin.

The patient was started on nafcillin and tobramycin initially on the night of hospital day 1 for possible endocarditis. On hospital day 3, the antibiotics were changed to intravenously administered cefamandole, 2.0 g every 6 h, and gentamicin, 100 mg every 8 h. There was some adjustment in the gentamicin dosage due to increasing nephrotoxicity. His temperature peaked to 103°F (39.4°C) for 4 days after admission but was normal thereafter. The findings from physical examination and repeated echocardiograms did not change. No vegetations on

heart valves were seen on the echocardiograms. However, antimicrobial therapy was continued for 4 weeks for possible bacterial endocarditis. On hospital day 10, the minimal bacteriocidal concentrations in the patient's serum (against his blood isolate) drawn 30 min before and 30 min after an infusion of cefamandole were 1:4 and 1:32, respectively. At this time, the patient was also receiving gentamicin, 130 mg intravenously every 18 h, but the exact timing of the gentamicin on this particular day was not clear from the chart.

The patient had an uneventful recovery and was discharged from the hospital on day 32. He was doing well when examined in the outpatient clinic 4 weeks after discharge, and blood cultures (both aerobic and anaerobic) taken in the outpatient clinic on days 34, 52, 87, and 105 were negative. This appeared to be a community-acquired rather than a nosocomial infection, since the patient was admitted with fever and other symptoms and had no known exposure to the hospital in the preceding 4.5 years. This was the first documented isolate of *Cedecea* at the hospital.

The isolate from one of the patient's blood cultures was sent to the Nosocomial Infections Laboratory Branch, Hospital Infections Program, Centers for Disease Control, Atlanta, Ga., where it was given the number 1238-81 (deposited in the American Type Culture Collection as ATCC 33856) and identified biochemically (2, 3) as a typical strain of *Cedecea* species 4, which is named *C. neteri* sp. nov. in this report. The following is a description of *C. neteri*, as required by the *Bacteriological Code* (5). *C. neteri* (pronunciation: suh dee' see ah [or C'D'C ah] knee' ter eye) conforms to the description of the genus *Cedecea* as given by Grimont et al. (4). It is a gram-negative, motile rod which is lipase (corn oil) positive and resistant to colistin and cephalothin. It is positive for methyl red, Voges-Proskauer (very weak), citrate, arginine "dihy-

TABLE 2. Biochemical tests that differentiate the three named and two unnamed species of *Cedecea*.

Test	Reaction ^a of:				
	<i>C. davisae</i>	<i>C. lapagei</i>	<i>C. neteri</i>	<i>Cedecea</i> species 3	<i>Cedecea</i> species 5
Ornithine decarboxylase (Moeller's)	+	-	-	-	v
Fermentation of:					
Sucrose	+	-	+	v	+
D-Sorbitol	-	-	+	-	+
Raffinose	-	-	-	+	+
D-Xylose	+	-	+	+	+
Melibiose	-	-	-	+	+
Malonate utilization	+	+	+	-	-
Growth in media without thiamine	-	+	+	+	+

^a +, 90 to 100% positive (all data are for 48 h and 36°C); -, 0 to 10% positive; v, 26 to 74% positive.

drolyase" (Moeller's), malonate, and nitrate reduction to nitrite. *C. neteri* is negative for indole production, H₂S production on triple sugar iron agar, urea hydrolysis, phenylalanine "deaminase," lysine and ornithine "decarboxylase" (Moeller's), and oxidase. A more complete description based on all four strains is given in Table 1. The type strain (holotype) is designated as ATCC 33855 (CDC 0621-75; strain 002 of Grimont et al. [4]). The type strain was from a human foot wound in California. One additional strain was deposited in the American Type Culture Collection; 1751-80 (ATCC 33857) was from human sputum and came from Virginia. The species name "*neteri*" is a neo (modern) Latin genitive (masculine) form of Neter, coined to honor Erwin Neter, the American physician-microbiologist, who has made many contributions to our knowledge of the family *Enterobacteriaceae*, particularly the role of this family in human disease.

Table 2 lists the biochemical reactions that can be used to differentiate the five species (groups) of *Cedecea*. We hope this report will

stimulate others to isolate and identify strains of *Cedecea* (particularly *C. neteri*, *Cedecea* species 3, and *Cedecea* species 5), so that their ecological niches and role in human disease can be better defined.

LITERATURE CITED

1. Bae, B. H. C., S. B. Sureka, and J. A. Ajamy. 1981. Enteric group 15 (*Enterobacteriaceae*) associated with pneumonia. *J. Clin. Microbiol.* **14**:596-597.
2. Edwards, P. R., and W. H. Ewing. 1972. Identification of *Enterobacteriaceae*, 3rd ed. Burgess Publishing Co., Minneapolis.
3. Farmer, J. J., III, M. A. Asbury, F. W. Hickman, D. J. Brenner, and The *Enterobacteriaceae* Study Group. 1980. *Enterobacter sakazakii*: a new species of "*Enterobacteriaceae*" isolated from clinical specimens. *Int. J. Syst. Bacteriol.* **30**:569-584.
4. Grimont, P. A. D., F. Grimont, J. J. Farmer III, and M. A. Asbury. 1981. *Cedecea davisae* gen. nov., sp. nov. and *Cedecea lapagei* sp. nov., new *Enterobacteriaceae* from clinical specimens. *Int. J. Syst. Bacteriol.* **31**:317-326.
5. Lapage, S. P., P. H. A. Sneath, E. F. Lessel, V. B. D. Skerman, H. P. R. Seeliger, and W. A. Clark (ed.). 1975. International code of nomenclature of bacteria. 1975 Revision. Published for the International Association of Microbiological Societies by the American Society for Microbiology, Washington, D.C.