JOURNAL OF CLINICAL MICROBIOLOGY, Mar. 1997, p. 777–779 Vol. 35, No. 3
0095-1137/97/$04.00+0

Septicemia Caused by *Streptococcus canis* in a Human

F. BERT* AND N. LAMBERT-ZECHOVSKY

Service de Microbiologie, Hopital Beaujon, Clichy, France

Received 2 August 1996/Returned for modification 1 November 1996/Accepted 6 December 1996

We describe a case of septicemia due to *Streptococcus canis* in a 77-year-old man. The organism was presumably transmitted from a domestic animal. Ulcers of the lower limbs were the likely portals of entry. The differentiation between *Streptococcus canis* and *Streptococcus dysgalactiae* was based on biochemical properties and DNA macrorestriction analysis by pulsed-field gel electrophoresis.

Large-colony-forming group G streptococci can be divided into human strains belonging to the species *Streptococcus dysgalactiae* (10) and animal strains belonging to the species *Streptococcus canis* (7). Human and animal strains can be differentiated on the basis of their biochemical and enzymatic profiles (6, 9) and by DNA-DNA hybridization studies (7). Group G streptococci have been associated with a variety of serious human infections, including bacteremia, endocarditis, septic arthritis, puerperal sepsis, and soft-tissue infections (1, 11, 20). Animal group G *Streptococcus* has been recognized as the cause of bovine mastitis (12, 16), genital and wound infections in dogs (3), and epizootics in cats (13, 19). To our knowledge, *S. canis* has not been reported as the agent of human infections, with the exception of a recent case report of meningitis (14) in which the identification of the strain was questionable since the biochemical characteristics of the isolate were not specified. The present report describes a case of septicemia due to a group G *Streptococcus*, subsequently identified as *S. canis* on the basis of its biochemical properties and a macrorestriction analysis of its DNA.

A 77-year-old man was referred by his general practitioner to the emergency department of our hospital because of malaise, fever, and tachycardia. The patient had a medical history of hypertension and continuous arrhythmia. On examination, he was found to be dyspneic and to have varicose ulcers and edema of the lower limbs. His temperature was 40°C, his pulse was 150 beats/min, and his blood pressure was 120/80 mm Hg. A chest X ray revealed bilateral opacities consistent with pulmonary edema. Laboratory studies disclosed a leukocyte count of 20,000/mm^3 with 67% neutrophils, a hemoglobin level of 10.4 g/dl, a platelet count of 191,000/mm^3, and an erythrocyte sedimentation rate of 34 mm. A urinalysis showed numerous polymorphonuclear leukocytes and gram-negative bacilli. Three blood cultures (Hemoline; BioMérieux, Marcy l'Etoile, France) were taken, and empiric therapy with josamycin (3 g/day) was started. The patient was admitted to the cardiology department. He became afebrile after 24 h of antibiotics, and his pulmonary edema regressed after treatment with furosemide.

The three blood cultures grew gram-positive cocci in chains after 48 h of incubation at 37°C. Subcultures onto sheep blood agar incubated aerobically and anaerobically yielded beta-hemolytic large colonies of streptococci belonging to Lancefield group G (streptococcal grouping kit; Oxoïd). By susceptibility testing on Mueller-Hinton blood agar by the disk diffusion method, the organism was found to be susceptible to benzylpenicillin, amoxicillin, cefalotin, erythromycin, clindamycin, pristinamycin, rifampin, and vancomycin and resistant to tetracycline. After the results of blood cultures were available, josamycin was switched to intravenous amoxicillin (6 g/day). Pefloxacin (800 mg/day) was added to the regimen after a urine culture yielded amoxicillin-resistant *Escherichia coli*. Searches for a primary focus of infection were unavailing. Echocardiography revealed a hypertrophic cardiopathy but no endocarditis. Dental panoramic and sinus X rays were unremarkable. There was no pulmonary or abdominal infection. The varicose ulcers of the lower limbs, although apparently healed, were considered to be the likely portals of entry for the septicemia. The search for a history of exposure to domestic animals revealed that the patient had a dog. The patient recovered routinely and was discharged after 2 weeks. Oral amoxicillin (3 g/day) was continued after discharge for 10 days.

The group G streptococcal isolate from the blood cultures was further identified on the basis of its biochemical properties. Acid was produced from glucose, sucrose, lactose, maltose, and ribose but not from trehalose, sorbitol, raffinose, mannitol, or arabinose. Hyaluronidase and fibrinolysin activities, tested as previously described (7), were negative. The isolate was positive for α-galactosidase, β-galactosidase, arginine dehydrolase, and leucine aminopeptidase, negative for aesculin hydrolysis, pyrrolidone aminopeptidase, and β-glucuronidase, and negative by the Voges-Proskauer test, as determined by API test strips (API, La Balme Les Grottes, France). It was identified as *S. canis* by the API 20 Strep system (profile, 0373405; identification, 99.9%) and the Rapid ID 32 Strep system (profile, 16116041150; identification, 99.9%).

The strain was further characterized by chromosomal DNA macrorestriction analysis by using the rare-cutting enzyme *Sma*I coupled with pulsed-field gel electrophoresis, as described elsewhere (2). Eight other strains were studied, including type strain *S. canis* DSM 20715, type strain *S. dysgalactiae* NCDO 2023, two *S. canis* isolates from dogs, and four human group G streptococcal isolates. The degree of similarity of the strains was determined by the Dice coefficient. The dendrogram constructed showed that the isolate clustered with reference strain DSM 20715 and the canine isolates and was clearly differentiated from reference strain NCDO 2023 and the human group G isolates (Fig. 1 and 2).

The term *S. canis* was first proposed by Stafseth et al. (18) for canine beta-hemolytic streptococci fermenting lactose but not trehalose and sorbitol. Subsequently, it has been widely used in veterinary textbooks to designate animal group G streptococci (4, 5). These strains are commonly isolated from the skin and urogenital tracts of dogs (6) and are responsible for bovine mastitis (12, 16) and epizootics in closed animal

* Corresponding author. Mailing address: Hopital Beaujon, service de Microbiologie, 100 boulevard du Général Leclerc, 92110, Clichy, France. Phone: 33 1 40.87.54.42. Fax: 33 1 40.87.05.50.

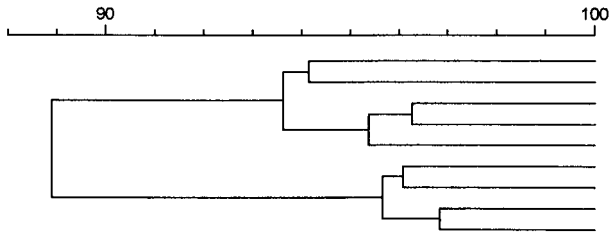

FIG. 1. Dendrogram obtained from the chromosomal DNA macrorestriction analysis of nine group G streptococcal strains. Type 1, reference strain NCDO 2023 (*S. dysgalactiae*); types 2 to 5, group G streptococcal isolates of human origin; type 6, reference strain DSM 20715 (*S. canis*); type 7, isolate from the present case report; types 8 to 9, group G streptococcal isolates of canine origin. The scale indicates percent genetic similarity.

populations (19). *S. canis* was recognized as an official species in 1986 (7). DNA-DNA hybridization data have shown that these strains are genetically distinct from human group G streptococci (7) which, along with group C beta-hemolytic *Streptococcus equisimilis*, group C alpha-hemolytic *S. dysgalactiae*, and group L strains, correspond to the emended description of *S. dysgalactiae* (10). The possible role of *S. canis* in human infections has not been recognized so far, and it was thought that there was a species specificity in the disease-producing capability of group G streptococci (6). T antigens of human isolates do not cross-react with T antigens of animal isolates, a fact that explains the possible failure of animal strains to colonize humans (8). The absence of hyaluronidase and fibrinolysin activities in *S. canis* may account for its decreased virulence for humans when compared with that of *S. dysgalactiae* (6). Moreover, M protein genes, which are a major virulence factor in the human pathogen *Streptococcus pyogenes*, are found in human group G streptococci but not in animal group G streptococci (17). A fatal case of meningitis and septicemia caused by *S. canis* in a 75-year-old woman has been recently reported (14), but the biochemical characteristics of the strains were not specified and the identification of the strain was not confirmed by a reference center; moreover, the authors stated that *S. canis* accounts for the majority of group G streptococci isolated from humans, indicating a confusion

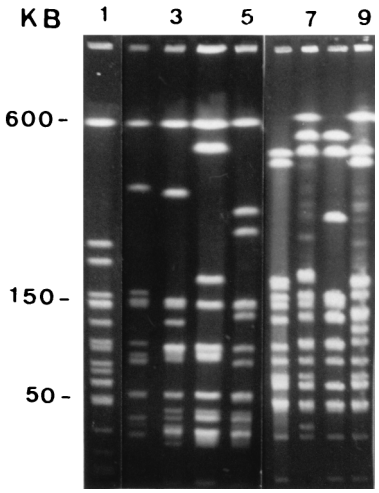

FIG. 2. Pulsed-field gel electrophoresis patterns of group G streptococcal isolates. Lane 1, *S. dysgalactiae* reference strain; lanes 2 to 5, *S. dysgalactiae* human isolates; lane 6, *S. canis* reference strain; lanes 7 to 8, *S. canis* canine isolates; lane 9, isolate from the present case report.

TABLE 1. Biochemical properties useful for differentiation of human and animal group G streptococci[a]

Activity or production	Result[b] for:	
	Human strains (*S. dysgalactiae*)	Animal strains (*S. canis*)
Hyaluronidase	+	−
Fibrinolysin	+	−
α-Galactosidase	−	+
β-Galactosidase	−	+
β-Glucuronidase	+	−
Acid produced from trehalose	+	−
Acid produced from lactose	±	+
Methyl-D-glucopyranoside	±	+

[a] Adapted from references 6, 7, and 9.
[b] +, ≥85% of strains positive; ±, 16 to 84% of strains positive; −, ≤15% of strains positive.

between *S. canis* and *S. dysgalactiae*. We found in the literature a single case of human group G streptococcal infection possibly due to *S. canis*: a two-year-old girl developed primary peritonitis caused by a group G *Streptococcus* following the contamination of an orofacial burn with infected urine of the family dog (15). However, the isolates recovered from the patient and the dog's urine were not identified to the species level.

The present report establishes the possible invasive capacity of *S. canis* for humans. The organism was presumably transmitted from the family dog and colonized the varicose ulcers of the patient, subsequently causing a systemic infection. The isolate was easily differentiated from *S. dysgalactiae* on the basis of its biochemical properties (Table 1), and DNA analysis confirmed that it belonged to the species *S. canis*. Although uncommon, human infections due to this organism may be underestimated because the routine identification of beta-hemolytic streptococci is based only on Lancefield's serological grouping, which is often falsely considered to be species specific. The occurrence of such infections probably requires close contact between a domestic animal and a skin wound such as a burn or ulcers. The recovery of a group G streptococcal isolate from a human infection should lead investigators to look for a history of exposure to animals, and, if necessary, the strain should be identified to the species level.

REFERENCES

1. **Auckenthaler, R., P. E. Hermans, and J. A. Washington.** 1983. Group G streptococcal bacteraemia: clinical study and review of the literature. Rev. Infect. Dis. **5:**196–204.
2. **Bert, F., C. Branger, and N. Lambert-Zechovsky.** Pulsed-field gel electrophoresis is more discriminating than multilocus enzyme electrophoresis and random amplified polymorphic DNA analysis for typing pyogenic streptococci. Curr. Microbiol., in press.
3. **Biberstein, E. L., C. Brown, and T. Smith.** 1980. Serogroups and biotypes among β-hemolytic streptococci of canine origin. J. Clin. Microbiol. **11:**558–561.
4. **Buxton, A., and G. Fraser.** 1977. Animal microbiology, p. 169. Blackwell Scientific Publications, Oxford, United Kingdom.
5. **Carter, G. R.** 1976. Essentials of veterinary bacteriology and mycology, p. 101. Michigan State University Press, East Lansing, Mich.
6. **Clark, R. B., J. F. Berrafati, J. M. Janda, and E. J. Bottone.** 1984. Biotyping and exoenzyme profiling as an aid in the differentiation of human from bovine group G streptococci. J. Clin. Microbiol. **20:**706–710.
7. **Devriese, L. A., J. Hommez, R. Kilpper-Bälz, and K.-H. Schleifer.** 1986. *Streptococcus canis* sp. nov.: a species of group G streptococci from animals. Int. J. Syst. Bacteriol. **36:**422–425.
8. **Efstratiou, A.** 1983. The serotyping of hospital strains of streptococci belonging to group C and group G. J. Hyg. **90:**71–80.
9. **Efstratiou, A., G. Colman, G. Hahn, J. F. Timoney, J. M. Boeufgras, and D. Monget.** 1994. Biochemical differences among human and animal streptococci of Lancefield group C or group G. J. Med. Microbiol. **41:**145–148.
10. **Farrow, J. A. E., and M. D. Collins.** 1984. Taxonomic studies on streptococci

of serological groups C, G and L and possibly related taxa. System. Appl. Microbiol. **5:**483–493.
11. **Gaunt, P. N., and D. V. Seal.** 1987. Group G streptococcal infections. J. Infect. **15:**5–20.
12. **Hamilton, C. A., and D. M. Stark.** 1970. Occurrence and characterization of Lancefield group G streptococci in bovine mastitis. Am. J. Vet. Res. **31:**397–398.
13. **Iglauer, F., I. Kunstyr, R. Mörstedt, H. Farouq, W. Wullenweber, and S. Damsch.** 1991. *Streptococcus canis* in a cat breeding colony. J. Exp. Anim. Sci. **34:**59–65.
14. **Jacobs, J. A., M. C. T. de Krom, J. T. C. Kellens, and E. E. Stobberingh.** 1993. Meningitis and sepsis due to group G streptococcus. Eur. J. Clin. Microbiol. Infect. Dis. **12:**224–225.
15. **Khan, J. A., H. E. Evans, M. R. Macabuhay, Y.-E. Lee, and R. Werner.** 1975. Primary peritonitis due to group G *Streptococcus*: a case report. Pediatrics **56:**1078–1079.
16. **Miller, W. T., and J. H. Heishman.** 1940. Bovine mastitis caused by unusual types of streptococci. Cornell Vet. **30:**310–318.
17. **Simpson, W. J., J. C. Robbins, and P. P. Cleary.** 1987. Evidence for group A-related M protein genes in human but not animal-associated group G streptococcal strains. Microb. Pathog. **3:**339–350.
18. **Stafseth, H. J., W. W. Thompson, and L. Neu.** 1937. Streptococcic infections in dogs. Acid milk, arthritis, and postvaccination abscesses. J. Am. Vet. Med. Assoc. **90:**769–781.
19. **Tillman, P. C., N. D. Dodson, and M. Indiveri.** 1982. Group G streptococcal epizootic in a closed cat colony. J. Clin. Microbiol. **16:**1057–1060.
20. **Vartian, C., P. I. Lerner, D. M. Shleas, and K. V. Gopalakrishna.** 1985. Infections due to Lancefield group G streptococci. Medicine (Baltimore) **64:**75–88.