

Endocarditis Caused by Nonhemolytic Group B Streptococcus

CONSUELO MIRANDA, MARÍA-ISABEL GÁMEZ, JOSÉ-MARÍA NAVARRO,
AND MANUEL ROSA-FRAILE*

Microbiology Service, Virgen de las Nieves Hospital, 18014 Granada, Spain

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We report a case of bacterial endocarditis caused by nonhemolytic group B streptococcus (GBS) in a 67-year-old man with no predisposing risk factors. Nonhemolytic GBS strains rarely cause illness and are usually detected in perinatal infections. We believe this to be the first reported case of endocarditis caused by a nonhemolytic strain of GBS.

Cases of bacterial endocarditis caused by *Streptococcus agalactiae* (Lancefield group B streptococcus [GBS]) are rare, with fewer than 100 episodes reported in the literature in English (1, 4, 7, 8). More than half of all cases arise in patients with risk factors such as cardiac disease, diabetes, alcoholism, solid or hematological tumors, peripheral vascular disease, and nephropathy. Nonhemolytic GBS strains isolated in clinical microbiology laboratories make up 1 to 2% of all strains of this microorganism (3) and are considered less virulent than hemolytic strains (9). Although the former strains have been implicated in cases of neonatal infection (5), the number of infections in adults caused by these microorganisms is reportedly extremely small (2). To the best of our knowledge, the following is the first reported case of endocarditis caused by a nonhemolytic GBS.

A 67-year-old man came to the emergency service of a district hospital and was admitted for a systolic II/IV heart murmur with an aortic focus. The only antecedents of note were fever (more than 39°C) of 1 month's duration and a poor general condition. There were no antecedents of cardiovascular disease, hypertension, diabetes, or recent urinary or dental procedures. On day 2, blood cultures (BacTAlert system; Organon Teknika, Durham, N.C.) and urine cultures (cystine lactose electrolyte-deficient agar [CLED] medium) grew a nonhemolytic gram-positive coccus with streptococcal morphology, and imipenem and amikacin administration was started. On day 3, the patient developed severe aortic insufficiency and was transferred to our hospital for emergency valvular replacement. On admission, echocardiographic exploration revealed rupture of the papillary muscle, and a 2- by 2-cm vegetation on the aortic valve was detected. Three sets of aerobic and anaerobic FAN blood culture bottles (BacTAlert system) were then inoculated.

After valvular replacement, Gram staining of material from the valve showed abundant gram-positive cocci resembling streptococci, and antibiotic therapy was switched to vancomycin (500 mg/6 h given intravenously [i.v.]) plus amikacin (7.5 mg/kg of body weight/12 h given i.v.) until identification and susceptibility studies of the microorganism were available.

Slices of the native valve were homogenized in brain heart infusion broth and cultured in blood agar (Columbia agar base plus 5% sheep blood) aerobically and under an anaerobic atmosphere (7% CO₂, 10% H₂, 83% N₂) and in chocolate agar

(5% CO₂ atmosphere). A pure culture of grey, convex, creamy, nonhemolytic colonies developed in all culture media after 18 h of incubation at 37°C. Colonies were oxidase and catalase negative, and Gram staining showed gram-positive cocci in chains. In view of these preliminary findings, identification was oriented toward nonhemolytic streptococci. Growth was negative in both 6.5% sodium chloride broth and bile esculin agar. An API 20 Strep biochemistry strip (bioMérieux, Marcy l'Etoile, France) was inoculated, and the microorganism was identified as GBS, with profile 3463001 and excellent species level identification. In addition, both CAMP and sodium hippurate hydrolysis testing were positive. GBS antigen was detected with the Streptococcal Grouping Kit (Oxoid Diagnostic Reagent, Basingstoke, United Kingdom). Serotyping of the isolate by agglutination with antisera to GBS serotypes I to V (Dako A/S, Glostrup, Denmark) showed the strain to be type III. Repeated subcultures of the microorganism onto blood agar under aerobic and anaerobic conditions failed to produce beta-hemolysis. No pigment production was observed in Granada medium, a selective differential medium that enhances the production of the characteristic orange pigment of GBS hemolytic strains (6). All FAN blood culture bottles were negative after 7 days of incubation in the BacTAlert system incubator.

Antimicrobial susceptibility tests were carried out by the agar diffusion method on Mueller-Hinton agar according to National Committee for Clinical Laboratory Standards recommendations. The microorganism was susceptible to beta-lactam antibiotics and vancomycin. Penicillin and cefotaxime MICs were ≤ 0.016 $\mu\text{g/ml}$, as determined by the E-test method (AB Biodisks, Solna, Sweden). Treatment was changed to ceftriaxone (1 g/12 h given i.v. for 4 weeks) plus gentamicin (80 mg/8 h given i.v. for 2 weeks), and the patient made an uneventful recovery. At follow-up, 6 months after treatment was completed, he was asymptomatic.

Most cases of GBS endocarditis described in the literature produce embolism and evolve poorly. Mortality has been estimated at 45% of all cases in which only medical treatment was given (4, 7). In our case, no embolism formed and the patient progressed favorably with medical and surgical treatment. The case described is unusual in that a pathogenic strain of nonhemolytic and nonpigmented GBS was isolated against a background of severe invasive disease from an adult patient with no identified risk factors. However, the urinary tract may have been the focus, because previous urine cultures grew gram-positive cocci with streptococcal morphology.

Most experienced microbiologists can presumptively identify GBS on the basis of Gram morphology and a narrow beta-hemolysis zone around colonies in blood agar plates. Identifi-

* Corresponding author. Mailing address: Servicio de Microbiología, Hospital Virgen de las Nieves, 18014 Granada, Spain. Phone: 34-58-241109 or -241232. Fax: 34-58-241282 or -241245. E-mail: delarosa@cica.es.

cation can be confirmed by serogrouping and demonstrating one of the specific physiological characteristics of this group, e.g., pigment production, positive CAMP test, or hippurate hydrolysis. However, in this case the GBS strain isolated from the aortic valve was nonhemolytic and nonpigmented. These findings are not surprising, because there is evidence of a genetic relationship in GBS between hemolysis and pigment production (10), both of which are considered virulence factors (9).

Microbiologists often rely on beta-hemolysis for the initial allocation of streptococci as beta-hemolytic (mainly groups A and B) or non-beta-hemolytic (enterococci and viridans group). This approach to identification may make it difficult to correctly identify nonhemolytic strains of streptococcal species that usually show beta-hemolysis. If additional tests are not carried out, such strains can be misidentified and the number of patients infected with microorganisms such as nonhemolytic GBS may be underestimated.

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