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Neisseria lactamica Septicemia in an Immunocompromised Patient

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Neisseria lactamica was isolated from the blood of a 7-year-old girl who was immunosuppressed from chemotherapy for acute lymphocytic leukemia. She was receiving trimethoprim-sulfamethoxazole prophylactically. The isolate was resistant to trimethoprim-sulfamethoxazole and sensitive to penicillin. The patient responded to intravenous penicillin therapy. The organism did not produce immunoglobulin A1 protease.

Neisseria lactamica rarely causes serious infections (3). It most commonly is found in the nasopharynx of children (1) and less often has been recovered from lung, vagina, amniotic fluid, and sputum (4). The clinical significance of these isolates is unclear. A few cases of *N. lactamica* meningitis are documented (2, 5), and there is one report of sepsis in a patient with otitis media presumably caused by the same organism (8). This report describes an immunosuppressed patient with sepsis due to a strain of *N. lactamica* that is resistant to trimethoprim-sulfamethoxazole.

Case report and microbiological findings. A 7-year-old girl was admitted to the Arizona Health Sciences Center with recent onset of fatigue and fever. She had recently been treated for relapse of acute lymphocytic leukemia and was receiving prophylactic trimethoprim-sulfamethoxazole.

On admission her temperature was 39.5°C, her pulse was 140/min, and her respiration was 24/min. Physical findings were positive for multiple small petechiae and nasal congestion. Her blood contained 400 white cells per μl with 10% neutrophils and 90% mature lymphocytes. Her platelet count was 5,000/μl. Two blood cultures were obtained on the day of admission, and she was treated with methicillin, gentamicin, and granulocyte transfusions. On the following day the blood culture was positive and grew oxidase-positive, gram-negative diplococci. An attempt to serotype the organism with meningococcal antisera failed because of autoagglutination. The isolate was identified as *N. lactamica* on the following day, based on biochemical and growth characteristics. The organism formed acid from glucose, maltose, and lactose, but not from sucrose, on cysteine-tryptic soy agar. It also gave a positive ONPG test for β-D-galactosidase and grew on modified Thayer-Martin medium. It was susceptible to penicillin, cephalothin, erythromycin, tetracycline, and chloramphenicol and resistant to sulfadiazine and trimethoprim-sulfamethoxazole by disk diffusion testing. The sulfadiazine minimum inhibitory concentration was 64 μg/ml when tested by agar dilution. The patient's therapy was changed to intravenous penicillin. She became afebrile on the following day and was discharged 10 days after admission on oral penicillin therapy. Andrew G. Plaut (New England Medical Center Hospital, Boston, Mass.) kindly tested the isolate for the production of immunoglobulin A1 (IgA1) protease. The result was negative.

Discussion. *N. lactamica* is a common commensal in the nasopharynx of children and generally is considered to be nonpathogenic (1, 3, 4). However, a few cases of meningitis due to *N. lactamica* have been reported (2, 5). In addition, Wilson and Overman (8) have described a case of *N. lactamica* sepsis in a 23-month-old girl in which otitis media was considered the primary infection. The patient had chromosomal defects but apparently was not immunosuppressed, and the infection responded to ampicillin.

The patient described in the present report was immunosuppressed and was receiving prophylactic trimethoprim-sulfamethoxazole. Her *N. lactamica* blood isolate was resistant to sulfadiazine and trimethoprim-sulfamethoxazole. Sulfadiazine resistance in *N. lactamica* is unusual. Hollis et al. (4) tested 100 strains of *N. lactamica* and found that 90% were inhibited by sulfadiazine at 10 μg/ml and all were inhibited at 30 μg/ml. The isolate from our patient had a sulfadiazine minimum inhibitory concentration of 64 μg/ml and was resistant to trimethoprim-sulfamethoxazole by disk diffusion testing. The mechanism of resistance was not investigated. It is likely that prophylactic antibiotic therapy provided a selective advantage for the infectivity of

this strain.

Previous reports of serious *N. lactamica* infection suggest that the upper respiratory tract is the initial site of infection (5, 8). Although a focus of infection was not identified, our patient had nasal congestion on admission, and it is possible that mucosal inflammation in this leukopenic patient provided an avenue for the organism to enter the blood and cause serious infection.

Plaut and co-workers (6, 7) have shown that pathogenic neisseriae (*N. meningitidis* and *N. gonorrhoeae*) uniformly produce an IgA1 protease which cleaves human IgA1 into F_c and F_{ab} fragments. They suggest that this specific and selective inactivation of the secretory immune system may be an important pathogenic mechanism in these organisms. *N. lactamica* generally is not pathogenic, and no strain has yet been found that produces IgA1 protease. The isolate from the present case was tested for IgA1 protease production to examine a property that would account for its unusual pathogenicity. IgA1 protease was not detected. It is likely that the immunocompromised state of the patient and the resistance of the organism to trimethoprim-sulfamethoxazole were the two most important factors accounting for this infection.

LITERATURE CITED

1. **Gold, R., I. Goldschneider, M. L. Lepow, and T. F. Draper, and M. Randolf.** 1978. Carriage of *Neisseria meningitidis* and *Neisseria lactamica* in infants and children. J. Infect. Dis. **137**:112–121.
2. **Hansman, D.** 1978. Meningitis caused by *Neisseria lactamica*. N. Engl. J. Med. **299**:491.
3. **Herbert, D. A., and J. Rusken.** 1981. Are the "nonpathogenic" neisseriae pathogenic? Am. J. Clin. Pathol. **75**:739–743.
4. **Hollis, D. G., G. L. Wiggins, R. E. Weaver, and J. H. Shubert.** 1970. Current status of lactose-fermenting neisseria. Ann. N.Y. Acad. Sci. **174**:444–449.
5. **Laver, B. A., and C. E. Fisher.** 1976. *Neisseria lactamica* meningitis. Am. J. Dis. Child. **130**:198–199.
6. **Mulks, M. H., and A. G. Plaut.** 1978. IgA protease production as a characteristic distinguishing pathogenic from harmless *Neisseriaceae*. N. Engl. J. Med. **299**:973–976.
7. **Plaut, A. G., J. V. Gilbert, M. S. Artenstein, and J. D. Capra.** 1975. *Neisseria gonorrhoeae* and *Neisseria meningitidis*: extracellular enzyme cleaves human immunoglobulin A. Science **190**:1103–1105.
8. **Wilson, H. D., and T. L. Overman.** 1976. Septicemia due to *Neisseria lactamica*. J. Clin. Microbiol. **4**:214–215.