

surveillance of antimicrobial drug susceptibility patterns of *V. cholerae*. Providing early information on antimicrobial drug susceptibility to practitioners in affected areas will lessen the illness and death from this devastating disease.

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DOI: 10.3201/eid1703.101176

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Neisseria meningitidis Strain of Unknown Serogroup, China

To the Editor: *Neisseria meningitidis* is a major public health hazard in many parts of the world. This organism is classified into 13 serogroups, and most meningococcal disease is caused by strains that express 1 of the 5 types of capsular polysaccharides (A, B, C, Y, and W135). In the natural reservoir of the human nasopharynx, strains of *N. meningitidis* that do not fit into 1 of the 13 serogroups and are presumably unencapsulated are common. By contrast, rare meningococcal diseases are caused by these nonserogroupable

strains. In this article, we describe a case of *N. meningitidis* infection caused by a nonserogroupable strain in the People's Republic of China and the genotype characteristics of this strain.

The patient was a 6-month-old boy who was admitted to a local hospital in Beijing in May 2009. The infection started suddenly with high fever (39°C). *N. meningitidis* infection was confirmed on the basis of the clinical signs and results of laboratory examination. Nausea, vomiting, and neck stiffness developed, and the patient lost consciousness. Physical examination showed a positive Kernig sign and negative Brudzinski sign. The patient's cerebrospinal fluid sample was injected into chocolate agar, in which microbial growth was observed after 24 hours. The API NH system (bioMérieux, Marcy-Etoile, France) showed that the isolate was *N. meningitidis*. However, this strain could not be placed in a serogroup, even after specific antiserum (Remel, Lenexa, KS, USA) was used. No other disease with complement deficiency was detected in the patient. The patient's infection was treated with antimicrobial drugs, and he recovered completely.

We investigated this nonserogroupable *N. meningitidis* strain by multilocus sequence typing (MLST), pulsed-field gel electrophoresis (PFGE), and subtyping of the variable regions of the genes (*porA*, *porB*, and *fetA*) encoding the outer membrane proteins. MLST indicated that the *fumC* was a new allele with a new number 482. The allele numbers for *abcZ*, *adk*, *fumC*, *gdh*, *pdhC*, and *pgm* were 222, 3, 58, 386, 18, and 77, respectively. This strain was assigned a new sequence type number, ST7962.

Among the 44 complexes designed in the MLST database, ST7962 was most similar to the ST4821 complex with 3 identical loci. The PFGE pattern of this strain was compared with the PFGE

patterns in the reference database of *N. meningitidis* from China by using BioNumerics version 5.10 software (Applied Maths, Kortrijk, Belgium). At the time of comparison, the database contained 618 isolates of *N. meningitidis* and 243 PFGE patterns. This strain had a single pattern that was clustered together with the ST4821 complex strains in the cluster tree based on the PFGE patterns. The PorA genotype of the strain was determined to be P1.7–2, 14, which was a genotype associated with the ST4821 complex serogroup C strains that caused outbreaks in China in 2003 (1). The *porB* and *fetA* alleles of this strain were 3–18 and F4–21, respectively.

The genetic basis for the reason that this strain was nonserogroupable was studied by PCR and sequencing. PCR showed that this strain had intact capsule genetic islands of *ctrA-D*, *synA-C*, *lipA*, and *lipB*, and contained *synD*, encoding the serogroup B polysialyltransferase. The capsular gene clusters were sequenced entirely, and a missense mutation within *synD* was identified (Figure). The mechanism underlying the capsule phase variation of *N. meningitidis* serogroup B involves a variation caused by slipped-strand mispairing in the polyC tract at the 5' end of *synD* (2–4). A tract of 7 C residues encodes capsular expression, and an insertion or deletion of 1 C results in a missense mutation within *synD*, thereby leading

to nonexpression of the capsule. Nucleotide sequencing of *synD* of our isolate revealed an insertion of 1 C within the polyC tract. Thus, slipped-strand mispairing within *synD* was predicted to be the mechanism underlying the nonserogroupability of this strain.

Few reports have described invasive meningococcal disease caused by nonserogroupable *N. meningitidis* strains; the lack of such reports suggests that complement deficiency might be a predisposing factor, and all the reported isolates were determined to be capsule null locus (*cnl*) strains, which lacked the genetic islands encoding the entire capsule (5–7). However, the patient described here was not found to have a complement deficiency, and the disease-associated nonserogroupable *N. meningitidis* strain in this study contained the genetic locus of an intact capsule.

In China, meningococcal polysaccharide vaccines A and C have been used for routine immunization. In many countries in Africa, repeated vaccination against *N. meningitidis* serogroups A and C have likely led to a selective increase in the incidence of meningococci of other serogroups, thereby resulting in a changed profile of meningococcal disease (8). In recent years, invasive disease caused by *N. meningitidis* serogroup W135 and serogroup X strains has emerged in China (9,10). Therefore,

meningococcal disease caused by serogroups other than A and C as well as nonserogroupable *N. meningitidis* strains appears to be an emerging problem and should be investigated epidemiologically.

This study was supported by grants from the Ministry of Health and the Ministry of Science and Technology (2008ZX10004-008), People's Republic of China.

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DOI: 10.3201/eid1703.101329

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Strain	<i>synD</i>	Capsule
MC58 (wild-type)	atg ... tat ctt acc ccc cca cgt aac aat ... taa ... Y L T P P R N N ...	On
100924 (missense mutation)	atg ... tat ctt acc ccc cca acg taa ... Y L T P P T . stop	Off

Figure. Genetic basis for the *Neisseria meningitidis* strain that cannot be placed in a known serogroup. A predicted slipped-strand mispairing occurred within *synD*, which encodes the serogroup B sialyltransferase. In wild-type *N. meningitidis* serogroup B (MC58), the *synD* polyC tract contains 7 C residues, and capsule is expressed. When an insertion (as in isolate 100924) of 1 C residue occurs, a result of local denaturation and mispairing followed by replication or repair, a premature stop codon is generated, and the capsule is not expressed.

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etymologia

Pseudoterranova azarasi

[sü-dō-'ter-ə-nō-və a-zär-a-sē]

From the Greek for false, Latin for earth and new, and Japanese for sea lion. First identified in 1878 as a parasite in pinnipeds by Danish scientist Harald Krabbe, who suggested the name *Ascaris decipiens*, the taxonomic designation for these nematodes changed as knowledge of the life cycles and morphologic features of members of the order Ascaridida expanded. In 1998, molecular examination found *Pseudoterranova decipiens*, long thought to be a monotype, consisted of genetically distinct sibling species. Mattiucci et al. proposed *Pseudoterranova azarasi* for 1 of the 5 sibling species, incorporating part of the name *Porrocaecum azarasi*, previously considered a synonym for *Pseudoterranova decipiens*.

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