Neisseria meningitidis
Serogroup X
Sequence Type 2888, Italy

To the Editor: Neisseria meningitidis serogroup X was first described in the 1960s and has been found to be responsible of rare cases of invasive meningococcal diseases, in particular, meningitis, in North America, Europe, Australia, Africa, and the People’s Republic of China (1–3). This serogroup has recently emerged in Africa as an increasing cause of meningitis; unfortunately, it is not covered by current vaccine programs. Serogroup X outbreaks have been reported in Niger, Ghana, and Kenya (4–6). In particular, in Niger during January–June 2006, N. meningitidis serogroup X represented 51% of confirmed cases of meningitis (4).

To investigate the population structure of serogroup X meningococci isolated during recent decades in Africa, Europe, and North America, Gagneux et al. (1) compared the molecular characteristics among them. That study highlighted a low genetic variability between African serogroup X strains, which contrasts with higher genetic variability among isolates from Europe and the United States (1).

We describe a case of invasive meningococcal disease caused by a serogroup X N. meningitidis strain isolated in Italy. The patient was a 55-year-old Italian woman with no immune deficiency. The onset of disease started quickly with high fever (39°C) on June 1, 2009. No contacts with persons coming from abroad were reported. This case was diagnosed on the basis of clinical signs and symptoms and results of laboratory confirmatory tests, including blood culture. The patient received ceftriaxone (2 g/day) for 7 days with a favorable outcome.

The strain was susceptible to penicillin G, rifampin, ciprofloxacin, and ceftriaxone, as determined by Etest method (bioMérieux, Florence, Italy). The breakpoints were those recommended by the Clinical and Laboratory Standards Institute (7). Serogroup was determined by serum agglutination, and serotype/subtype, NT:P1.15, 19 were determined by standard whole-cell ELISA with monoclonal antibodies (obtained from the National Institute for Biological Standards and Control, South Mimms, UK) (8).

PorA variable regions, FetA, and multilocus sequence typing analyses were performed according to standard procedures from the Neisseria Multi Locus Sequence Typing Web site (http://pubmlst.org/neisseria). The isolate from Italy had the pattern PorA VR1–19, VR2–15, and VR3–36; F5–5 and sequence type (ST)-2888. The same ST was already described in Greece in 2002 but in a noninvasive strain (http://pubmlst.org/neisseria).

The pattern obtained by pulsed-field gel electrophoresis (9), using the rare-cutting enzyme NheI, (data not shown), was identical to patterns found among meningococci X strains isolated in United Kingdom and belonging to ST-750, clonal group X-II (1). In particular, ST-2888 resembles, except for gdh gene sequence, ST-2317, which was found among the X meningococci isolated in the United Kingdom in 2002 with phenotype X:4:P1.7 (http://pubmlst.org/neisseria).

Our data document a rare case of invasive meningococcal meningitis in Italy, caused by N. meningitidis serogroup X ST-2888. Future surveillance data may be able to determine epidemiologic influences, likely emanating from nearby countries, on the spread of such a strain into Italy.

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References

1. Gagneux S, Wirth T, Hodgson A, Ehrhard I, Morelli G, Kriz P, et al. Clonal groupings in serogroup X Neisseria meningitidis. Emerg Infect Dis. 2002;8:462–6.

2. del Castillo CM, Vázquez JA, Romero J, Pascual A. Infections by Neisseria meningitidis serogroup X in Spain. Clin Microbiol Infect. 2003;9:964–5. DOI: 10.1046/j.1469-0691.2003.00685.x

3. Chen C, Zhang TG, Wu J, Chen LJ, Liu JF, Deng XH, et al. Typing of sequential bacterial isolates by pulsed-field gel electrophoresis. Diagn Microbiol Infect Dis. 1995;22:309–14. DOI: 10.1016/0732-8893(95)00139-8

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Antiphospholipid Syndrome and Acute HIV Infection

To the Editor: Patients with acute HIV infection frequently experience a syndrome characterized by fever, sore throat, lymphadenopathy, maculopapular rash, and lymphomonocytosis, which mimics acute infectious mononucleosis, 3–6 weeks after primary infection (1). Aseptic meningitis, encephalitis, and peripheral neuropathy are the most commonly observed features. In contrast, antiphospholipid syndrome complicated with pulmonary emboli is not commonly associated with acute retroviral syndrome. The following case should prompt clinicians to consider an expanded clinical scope of initial signs and symptoms for acute HIV infection.

A 28-year-old homosexual man was admitted to a hospital in Madrid, Spain, on June 22, 2009, with fever, pharyngitis, and myalgias. Generalized lymphadenopathy was found on examination. Lymphomonocytosis and mild elevation of serum aspartate aminotransferase and serum alanine aminotransferase levels were found. Chest radiographs showed no abnormalities. Results of a commercial ELISA for HIV-1 and HIV-2 were negative. Results of a p24 antigen-capture assay were positive, and viral load measured by reverse transcription–PCR (RT-PCR, Amplicor; Roche Molecular Diagnostics, Pleasanton, CA, USA) was 2,600,000 copies RNA HIV/mL. CD4+ T-cell count was 297 cells/μL.

The patient was discharged with instructions to take acetylsalicylic acid, but he was readmitted 1 week later with recurring fever, pleuritic chest pain, and shortness of breath. He was febrile (38.5°C), tachycardic, and tachypneic and had a blood pressure of 155/72 mm Hg and generalized lymphadenopathy. Blood tests showed a hemoglobin level of 10.6 g/dL, leukocyte count of 5,160 cells/μL, and thrombocyte count of 293 cells/μL. Results of renal function tests were within normal limits as were serum aminotransferase levels. Lactate dehydrogenase level was 698 IU/L (reference range 211–423 IU/L) and γ-glutamyl transpeptidase level was 1210 IU/mL (reference range 5–50 IU/mL). Ferritin level was 2,000 μg/L (reference range 10–200 μg/L), and C-reactive protein level was 10.6 mg/dL (reference range 0–5 mg/dL).

Antibodies against phospholipids (PLs) and β2-glycoprotein I (β2GPI) measured by ELISA were detected at high titers: immunoglobulin (Ig) M anticardiolipin > 72 U MPL/mL (positive at >20 U MPL/mL), IgG anticardiolipin > 158 U GLP/mL (positive at >20 U GLP/mL), and IgA anticardiolipin > 210 U/mL (positive at >10). Results of screening tests for thrombophilia and other autoantibodies were within normal limits.

The patient was treated with low molecular weight heparin, oxyzen, and analgesics. His fever subsided, and he was discharged a few days later while continuing to receive acenocoumarol, an oral coumarin anticoagulant. Results of a repeated HIV ELISA were then positive. Western blot assay confirmed the presence of antibodies to p24, gp41, and gp120/160.