2. Tigas LA, Van Vuren DH, Sauvajot RM. Behavioral responses of bobcats and coyotes to habitat fragmentation and corridors in an urban environment. Biological Conservation. 2002;108:299–306. DOI: 10.1016/S0006-3207(02)00120-9
3. Wang X, Werner BG, Konomi R, Hennigan D, Fadden D, Caten E, et al. Animal rabies in Massachusetts, 1985–2006. J Wildl Dis. 2009;45:375–87.
4. Centers for Disease Control and Prevention. Protocol for postmortem diagnosis of rabies in animals by direct fluorescent antibody testing: a minimum standard for rabies diagnosis in the United States [cited 2009 Mar 31]. http://www.cdc.gov/rabies/docs/RabiesDFASPv2.pdf
5. Blanton JD, Hanlon CA, Rupprecht CE. Rabies surveillance in the United States during 2007. J Am Vet Med Assoc. 2007;231:540–56.
6. Ballantyne EE. Symptoms noted in rabid coyotes in Alberta. Can J Comp Med. 1958;22:107–9.

Address for correspondence: Xingtai Wang, Division of Molecular Diagnostics and Virology, Massachusetts Department of Public Health William A. Hinton State Laboratory Institute, 305 South St, Boston, MA 02130, USA; email: xingtai.wang@state.ma.us

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 *Nhe*I, (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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Cecilia Fazio, Stefania Starnino, Marina Dal Soldà, Tonino Sofia, Arianna Neri, Paola Mastrantonio, and Paola Stefanelli

Author affiliations: Istituto Superiore di Sanità, Rome, Italy (C. Fazio, S. Starnino, T. Sofia, A. Neri, P. Mastrantonio, P. Stefanelli); and Azienda Sanitaria, Locale, Cremona, Italy (M. Dal Soldà)

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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, Pang XH, et al. A first meningococcal meningitis case caused by serogroup X *Neisseria meningitidis* strains in China. Chin Med J. 2008;127:664–6.
4. Boisier P, Nicolas P, Djibo S, Taha M-K, Jeanne I, Maïnassara HB, et al. Meningococcal meningitis: unprecedented incidence of serogroup X-related cases in 2006 in Niger. Clin Infect Dis. 2007;44:657–63. DOI: 10.1086/511646
5. Gagneux SP, Hodgson A, Smith TA, Wirth T, Ehrhard I, Morelli G, et al. Prospective study of a serogroup X *Neisseria meningitidis* outbreak in northern Ghana. J Infect Dis. 2002;185:618–26. DOI: 10.1086/339010
6. Mutonga DM, Pimentel G, Muindi J, Nzioka C, Mutiso J, Klena JD, et al. Epidemiology and risk factors for serogroup X meningococcal meningitis during an outbreak in western Kenya. Am J Trop Med Hyg. 2009;80:619–24.
7. Clinical and Laboratory Standard Institute. Performance standards for antimicrobial susceptibility testing, 18th informational supplement. M100–S18. Wayne (PA): The Institute; 2008.
8. Abdillahi H, Poolman JT. Whole-cell ELISA for typing *Neisseria meningitidis* with monoclonal antibodies. FEMS Microbiol Lett. 1987;48:367–71. DOI: 10.1111/j.1574-6968.1987.tb02626.x
9. Hartstein A, Phelps C, Lemonte A. 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

Address for correspondence: Paola Mastrantonio, Department of Infectious, Parasitic and Immune-mediated Diseases, Istituto Superiore di Sanità, Viale Regina Elena, 299, 00161, Rome, Italy; email: paola.mastrantonio@iss.it

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, pharyingitis, 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 acetaminophen, but he was readmitted 1 week later with recurring fever, pleuritic chest pain, and shortness of breath. He was febrile (38.5ºC), 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 D-dimer was 3,414 μg/L (reference range 68–494 IU/L). Fibrinogen levels, prothrombin time, and partial thromboplastin time were normal. Chest radiographs showed a small area of pleural effusion on the left side. A computed tomographic scan of the chest showed multiple pulmonary emboli with areas of parenchymal infarction.

Antibodies against phospholipids (PLs) and β_2-glycoprotein I (β_2GPI) 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), IgG anti-β_2GPI + 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, oxygen, 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.