

Methicillin-Resistant *Staphylococcus aureus* USA400 Clone, Italy

To the Editor: In the past 30 years, methicillin-resistant *Staphylococcus aureus* (MRSA) has been the leading cause of nosocomial infections throughout the world. Healthcare-associated MRSA (HA-MRSA) isolates are resistant to multiple antimicrobial drugs. This resistance severely hampers treatment options. During the past decade, MRSA isolates have also emerged as major pathogens in the community, first in the United States and later worldwide. Community-associated MRSA (CA-MRSA) isolates are usually more susceptible to antimicrobial drugs but are more virulent than HA-MRSA isolates. Among various determinants involved in the pathogenesis of CA-MRSA infections, special attention has been focused on the Panton-Valentine leukocidin (PVL), which has a strong epidemiologic link with CA-MRSA clones (1).

It has been suggested that CA-MRSA might move to healthcare settings, blurring the line between HA- and CA-MRSA (2). Nevertheless, CA-MRSA isolates are increasingly being reported as pathogens in the general population in persons with no risk factors for HA-MRSA acquisition. These pathogens are generally associated with skin and soft tissue infections, but also with more severe infections such as necrotizing pneumonia or septicemia. CA-MRSA strains usually harbor a staphylococcal cassette chromosome (SCC) *mec* (type IV or V) that is smaller than the type I–III SCC*mec* elements commonly found in HA-MRSA strains. To date, 5 major CA-MRSA clonal lineages from diverse genetic backgrounds have been recognized by pulsed-field gel electrophoresis and multilocus sequence

typing; certain clones predominate in specific areas of the world (1).

The most common lineages in the United States are sequence type (ST) 1 (USA400) and ST8 (USA300), which usually carry type IV SCC*mec* and PVL-encoding genes. Over the past few years, ST8 (USA300) has become predominant in the United States (3), also emerging as a major cause of nosocomial infections (4). In Europe, data are more limited, but the situation appears to be more varied: the predominant CA-MRSA clonal lineage is ST80 (5), although single cases or small clusters caused by ST8 (USA300) have increasingly been reported (6–8). In contrast, the ST1 (USA400) clone is still rare in Europe (9,10). We describe the importation of ST1 (USA400) into Italy and its isolation in the country. The organism was isolated from an Italian woman with a skin infection that she contracted in the United States.

In late November 2007, a 36-year-old Italian woman was seen at Pordenone Hospital (northeastern Italy) for spider-bite-like skin lesions on the face, characterized by rapid evolution to furuncles and small abscesses. The infection had started ≈1 month earlier in California, where she had spent several months on business (wine import-export), and where she had been treated empirically with amoxicillin/clavulanate for 10 days (1 g, 3×/day), with no clinical improvement.

Culture of the pus from the abscesses yielded an MRSA isolate that was resistant to oxacillin and susceptible to all non-β-lactam antimicrobial drugs tested by Vitek 2 AST-P536 card (bioMérieux, Marcy l’Etoile, France). Such a particular susceptibility pattern and the community origin of the infection prompted molecular investigation and typing by established methods, which confirmed the isolate to be CA-MRSA and identified it as belonging to the USA400 clone (ST1, type IVa SCC*mec*, presence of PVL genes,

agr type III, *spa* type t128). Notably, t128 is the *spa* type found in MW2, the highly virulent prototype strain of USA400. Treatment with oral levofloxacin for 7 days (500 mg, 1×/day) led to complete resolution of the infection. After more than a year, the patient has experienced no recurrences.

All 3 previously reported cases of CA-MRSA infection in Italy were caused by type IV SCC*mec*, PVL-positive strains, none of which, however, belonged to the ST80 clonal lineage that predominates in Europe (7). The first case (in 2005) was a necrotizing pneumonia caused by an ST30 isolate; the 2 other cases (2006) were severe invasive sepsis and a neck abscess, both caused by ST8 (USA300) isolates. The case we note here documents the importation of a US pathogen into a country in Europe, from an area where the pathogen is widespread and has been highly virulent since the late 1990s, to an area where its penetration in the past has been poor.

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References

- Diep BA, Otto M. The role of virulence determinants in community-associated MRSA pathogenesis. *Trends Microbiol.* 2008;16:361–9. DOI: 10.1016/j.tim.2008.05.002
- Tenover F. Community-associated methicillin-resistant *Staphylococcus aureus*: it's not just in communities anymore. *Clin Microbiol Newslett.* 2006;28:33–6. DOI: 10.1016/j.clinmicnews.2006.02.001
- King MD, Humphrey BJ, Wang YF, Kourbatova EV, Ray SM, Blumberg HM. Emergence of community-acquired methicillin-resistant *Staphylococcus aureus* USA 300 clone as the predominant cause of skin and soft tissue infections. *Ann Intern Med.* 2006;144:309–17.

4. Seybold U, Kourbatova EV, Johnson JG, Halvosa SJ, Wang YF, King MD, et al. Emergence of community-associated methicillin-resistant *Staphylococcus aureus* USA300 genotype as a major cause of health care-associated blood stream infections. *Clin Infect Dis.* 2006;42:647–56. DOI: 10.1086/499815
5. Deurenberg RH, Vink C, Kalenic S, Friedrich AW, Bruggeman CA, Stobberingh EE. The molecular evolution of methicillin-resistant *Staphylococcus aureus*. *Clin Microbiol Infect.* 2007;13:222–35. DOI: 10.1111/j.1469-0691.2006.01573.x
6. Witte W, Strommenger B, Cuny C, Heuck D, Nuebel U. Methicillin-resistant *Staphylococcus aureus* containing the Panton-Valentine leucocidin gene in Germany in 2005 and 2006. *J Antimicrob Chemother.* 2007;60:1258–63. DOI: 10.1093/jac/dkm384
7. Tinelli M, Pantosti A, Lusardi C, Vimercati M, Monaco M. First detected case of community-acquired methicillin-resistant *Staphylococcus aureus* skin and soft tissue infection in Italy. *Euro Surveill.* 2007;12:E070412.1.
8. Ruppitsch W, Stoger A, Schmid D, Fretz R, Indra A, Allerberger F, et al. Occurrence of the USA300 community-acquired *Staphylococcus aureus* clone in Austria. *Euro Surveill.* 2007;12:E071025.1.
9. Witte W, Braulke C, Cuny C, Strommenger B, Werner G, Heuck D, et al. Emergence of methicillin-resistant *Staphylococcus aureus* with Panton-Valentine leukocidin genes in central Europe. *Eur J Clin Microbiol Infect Dis.* 2005;24:1–5. DOI: 10.1007/s10096-004-1262-x
10. Harbarth S, François P, Schrenzel J, Fankhauser-Rodriguez C, Hugonnet S, Koessler T, et al. Community-associated methicillin-resistant *Staphylococcus aureus*, Switzerland. *Emerg Infect Dis.* 2005;11:962–5.

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Meningitis and Radiculomyelitis Caused by *Angiostrongylus cantonensis*

To the Editor: *Angiostrongylus cantonensis* infection is endemic in regions such as Southeast Asia, China, the Pacific Basin, and the Caribbean, but international travel has spread the disease elsewhere, including Europe (1–10). Dissemination of the parasite to many regions has also occurred because of the ship-borne international migration of rats and the diversity of potential intermediate hosts. The target organ in humans is the central nervous system in which an eosinophilic reaction develops in response to dying larvae. We report a case of eosinophilic meningitis and lumbosacral myeloradiculopathy caused by *A. cantonensis* and present a review of cases of *A. cantonensis* infections from Europe.

A 47-year-old merchant seaman was admitted to the University Hospital of Infectious Diseases, Zagreb, Croatia, in March, 2006 on the 17th day of illness because of fever, headache, vomiting, and constipation. At the end of the first week of illness, paresthesias developed in his feet; on the 10th day of illness, he also noticed difficulties with urination. He had returned from a 1-month trip to Southeast Asia (Malaysia and Singapore) 35 days before the onset of symptoms and recalled eating vegetables and salads. He also consumed shrimp, but he believed that they were from salt water. On physical examination, we noticed increased muscle tone, tremor of the tongue and upper limbs, and decreased deep tendon reflexes of the lower limbs. He experienced urinary retention, and catheterization was required. Saddle anesthesia was observed. There was no neck stiffness, and the results of the rest of the physical examination were normal.

His blood leukocyte count was $11.5 \times 10^9/\text{L}$ with 80% neutrophils, 12% lymphocytes, 4% monocytes, 2% basophils, and 2% eosinophils. Cerebrospinal fluid (CSF) analysis showed 320 cells/ μL with 6.5% eosinophils (21 eosinophils/ μL). Results of CSF testing by PCR for herpes simplex virus 1 (HSV-1) and HSV-2 DNA were negative, as were cultures for bacteria, mycobacteria, and fungi. Results of serum and CSF antibody tests for *Borrelia burgdorferi*, *Treponema pallidum*, HSV-1, HSV-2, tick-borne encephalitis virus, *Toxoplasma gondii*, *Taenia solium*, *Toxocara* spp., and *Trichinella* spp. were also negative. Results of stool examination for *Ascaris lumbricoides*, *Trichuris trichiura*, *Taenia* spp., *Giardia intestinalis*, *Strongyloides* spp., and *Entamoeba histolytica* were negative. The patient was also negative for HIV by ELISA. Magnetic resonance imaging scans of the brain and spine were unremarkable. *A. cantonensis* infection was diagnosed by immunoblot testing at the Department of Helminthology, Faculty of Tropical Medicine, Mahidol University, Bangkok. Antibodies against *A. cantonensis* 31-kDa antigen were detected in serum and CSF of the patient; antibodies against *Gnathostoma spinigerum* were not detected.

Treatment was symptomatic; to lessen the headache, 4 lumbar punctures were performed. After 1 month, the patient's general condition was greatly improved; however, minor symptoms such as diminished concentration, slow thinking, and mild headache persisted. Urinary retention lasted for 38 days, and the patient had occasional mild headaches and paresthesia in his feet for the next 5 months.

Because large numbers of persons from Europe travel to destinations where angiostrongyliasis is endemic, it is somewhat surprising that the infection has been rarely described in Europe. In a Google and Medline