LETTERS

Methicillin-Resistant Staphylococcus aureus ST398, Italy

To the Editor: It has recently become apparent that livestock can constitute a new methicillin-resistant Staphylococcus aureus (MRSA) reservoir and be a source of a novel and rapidly emerging type of MRSA. These livestock-associated MRSA clones are nontypeable by use of pulsed-field gel electrophoresis with Smal and belong to sequence type (ST) 398 (1). MRSA ST398 clones account for 20% of all MRSA in the Netherlands (2), but the emergence of such clones has been described worldwide (3). Although ST398 transmission has been reported primarily between animals, persons with occupational exposure to livestock are at higher risk for MRSA carriage than the general population. Even though MRSA ST398 usually causes colonization, several cases of infections of variable clinical relevance, varying from skin and soft tissue infections (4) to endocarditis (5) and pneumonia (6), have been described over the past few years. Most instances of ST398 human carriers have been identified among persons who work at pig farms (7). Data regarding MRSA colonization of dairy farmers are less exhaustive and, to our knowledge, only 1 instance of direct transmission between cattle and humans has been proven. MRSA isolates from cows with subclinical mastitis in 2007 in Hungary were indistinguishable from MRSA isolates from the tonsil swab of a farmer who worked with these animals (8). We report a case of MRSA ST398 invasive disease in a cattle farmer, as well as a case of MRSA ST398 necrotizing fasciitis.

In early April 2008, a 52-year-old man was admitted to an intensive care unit in Manerbio, Italy, because of severe sepsis and a large ulcerative and
suppurative lesion on the right side of his neck. His medical history was unremarkable. He was a worker at a dairy farm, was obese, and did not report any previous contact with the healthcare system. At the time of hospital admission, he was oriented and cooperative. His temperature was 38.4°C, heart rate was 125 beats per minute, and blood pressure was 165/75 mm Hg. Arterial blood gas analysis showed hypoxemia and mild hypocapnia (PaO₂ 53 mm Hg and PaCO₂ 33.8 mm Hg on room air). Leukocyte count was 21,280 cells/μL (81.9% polymorphonuclear cells), and platelet count was 310,000 cells/μL. After blood samples were collected and aggressive surgical debridement of affected tissue was performed, empirical treatment with intravenous teicoplanin and imipenem was started. On the basis of histologic appearance of the intraoperative material and computed tomography scan images, necrotizing fasciitis was diagnosed. Culture of blood and necrotic tissue yielded MRSA. On day 3 after admission, antimicrobial drug therapy was changed to teicoplanin and clindamycin and, on day 7, to linezolid. Fever resolved in 3 days and the patient’s condition progressively improved. The patient was discharged after 31 days of antimicrobial drug therapy. The MRSA isolate was susceptible to all the non-β-lactam antimicrobial drugs tested (excluding tetracycline), carried the staphylococcal cassette chromosome mec type V, and was negative for Panton-Valentine leukocidin (PVL) genes. Multilocus sequence typing and sequence typing of the tandem repeat region of protein A gene (spa typing) showed that the isolate belonged to ST398 and spa type 899, respectively.

Some issues are of concern. Although the MRSA isolate was PVL negative, its virulence resembled that of PVL-positive strains. Furthermore, it was resistant to tetracycline, as we expected because oxytetracyclines are the antimicrobial drugs most frequently used in pig and cattle farming (3). The major limitation of our study was that data regarding MRSA colonization of the farm are missing, so cattle-to-human transmission cannot be proven. However, because our patient did not have any other potential risk factor, dairy cows were probably the source of the human infection. In countries where community-acquired MRSA is common, all patients with serious S. aureus infections should be treated for MRSA until antimicrobial susceptibilities are known. Our report suggests that even in countries where community-acquired MRSA is still rare, being a cattle farmer may be considered an indication for early treatment against MRSA.

The expanding knowledge of this zoonotic potential may undermine existing nosocomial MRSA control programs. In countries where a search and destroy policy (9) is adopted, such as the Netherlands, pig and cattle farmers may warrant screening and isolation at the time of hospital admission. Nevertheless, the first MRSA ST398 nosocomial outbreak has already been described (10).

It is difficult to prevent persons with constant exposure to MRSA in their work or home setting from becoming MRSA carriers. Revisiting policies for the use of antimicrobial drugs on livestock farms, as well as improving hygiene measures, may therefore be necessary in infection control programs. However, before final recommendations can be made, further investigation is needed to determine the prevalence of MRSA among livestock and their handlers.

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References

1. Huijsdens X, van Dijke BJ, Spalburg E, van Santen-Verheuvel MG, Heck ME, Pluister GN, et al. Community-acquired MRSA and pig-farming. Ann Clin Microbiol. 2006;5:226. DOI: 10.1186/1469-7711-5-26
2. van Leeuwen, Huijsdens X, Tiemersma E, de Neeling A, van de Sande-Bruinsma N, Beuhaus M, et al. Emergence of methicillin-resistant Staphylococcus aureus of animal origin in humans. Emerg Infect Dis. 2007;13:1834–9.
3. Wulf M, Voss A. MRSA in livestock animals—an epidemic waiting to happen? Clin Microbiol Infect. 2008;14:519–21. DOI: 10.1111/j.1469-0691.2008.01970.x
4. Pan A, Battisti A, Zoncada A, Bernieri F, Boldini M, Franco A, et al. Community-acquired methicillin-resistant Staphylococcus aureus ST398 infection, Italy. Emerg Infect Dis. 2009;15:845–7. DOI: 10.3201/eid1505.081417
5. kkelenkamp MB, Sikkat M, Carpaj N, Troelstra A, Bonten MJ. Endocarditis due to methicillin-resistant Staphylococcus aureus originating from pigs [in Dutch]. Ned Tijdschr Geneeskd. 2006;150:2442–7.
6. van Rijen MM, van Keulen PH, Kluytmans JA. Increase in a Dutch hospital of methicillin-resistant Staphylococcus aureus related to animal farming. Clin Infect Dis. 2008;46:261–3. DOI: 10.1086/524672
7. Wulf MW, Sorm M, van Nes A, Skov R, Melchers WJ, Kluymans LJ, et al. Prevalence of methicillin-resistant Staphylococcus aureus among veterinarians: an international study. Clin Microbiol Infect. 2008;14:29–34. DOI: 10.1111/j.1469-0691.2007.01873.x
8. Juhász-Kaszanyitzky E, Janosi S, Somo- gyi P, Dan A, van der Graaf-van Bloois L, van Duijkeren E, et al. MRSA transmission between cows and humans. Emerg Infect Dis. 2007;13:630–2. DOI: 10.3201/eid1304.060833
9. Dutch Working Party on Infection Prevention. Policy for methicillin-resistant Staphylococcus aureus. 2007 [cited 2009 Dec 10]. http://www.wip.nl/UK/content/browser/onderwerp.asp
To the Editor: Neisseria meningitidis is a gram-negative bacterium found only in humans and is a major cause of serious invasive diseases. Before 2006, in the People’s Republic of China, all meningococcal diseases were caused by serogroups A, B, and C. However, there are ≥13 serogroups of this organism. Three cases of infection with N. meningitidis serogroup W135 were reported in China during 2006–2008. We describe these 3 meningitis patients and the N. meningitidis serogroup W135 strains isolated from these patients by genotyping methods.

Patient 1, a 36-year-old man, was seen at a local hospital in Fujian Province in January 2006. He became ill while on a business trip and was given a diagnosis by culture of an N. meningitidis infection. Patient 2, a 25-year-old man, was seen in Guangdong Province in May 2007. He had not traveled outside this area in the 10 days before becoming ill. Patient 3, a 14-year-old girl, was seen in Guangxi Province in February 2008. She was a middle school student and had toured the suburbs of this province with her classmates 2 days before becoming ill. Close contacts of all 3 patients were investigated; no additional N. meningitidis infections were detected. However, N. meningitidis was isolated from a throat swab specimen obtained from the younger cousin of patient 3.

N. meningitidis infection was confirmed for all 3 patients on the basis of clinical symptoms and laboratory results. All patients reported neck stiffness. Physical examinations showed Kernig signs, Brudzinski signs, and high temperatures (>38°C). Cerebrospinal fluid (CSF) samples were turbid with increased protein levels and pressure; leukocyte counts were increased (>5,000 cells/µL). CSF culture on chocolate agar grew N. meningitidis after 24 h. Isolates were identified as serogroup W135 by using specific antisera (Remel, Lenexa, KS, USA) at provincial Centers for Disease Control and Prevention (CDC) in China and confirmed at the Chinese CDC.

Patients were treated with antimicrobial drugs and recovered fully. An isolate from the cousin of patient 3 was also identified as W135. Etest strips and broth microdilution were used for antimicrobial drug susceptibility testing for the 4 W135 isolates. All isolates were susceptible to 12 antimicrobial drugs tested, which included therapeutic and prophylaxis agents used frequently in China.

Pulsed-field gel electrophoresis (PFGE), multilocus sequence typing, and outer membrane protein (porA) gene variant region subtyping were used to characterize the 4 case-related W135 N. meningitidis isolates and other isolates from asymptomatic carriers. Strain R29057 (from France) was used as a reference strain. The 4 case-related isolates showed similar PFGE patterns. These patterns were distinct from those of other W135 isolates obtained from asymptomatic carriers. Three invasive disease isolates and 1 from the close contact of patient 3 had the same multilocus sequence type (ST) and PorA subtype; all were ST11: P1.5, 2. This subtype was not detected among other tested isolates of W135 obtained from asymptomatic carriers (online Technical Appendix, www.cdc.gov/EID/content/16/2/348-Techapp.pdf).

ST11: P1.5, 2 N. meningitidis serogroup W135 was responsible for the epidemic of W135 meningococcal disease in 2000, which was associated with the Hajj pilgrimage in Saudi Arabia (1,2). The strain related to the Hajj pilgrimage was derived from clonal expansion within the ST11 complex/ET-37 complex (3). However, no epidemiologic data showed that the 3 cases in our study were linked to the Hajj pilgrimage. Since 2000, invasive diseases caused by W135 meningococci of ST11 have been reported in Africa, Asia, and the Middle East (4). ST11 W135 infections have been reported to cause invasive disease in Taiwan during 1996–2002 and were apparently introduced into Taiwan before the Hajj pilgrimage–associated outbreak because they were genotypically distinct from the Hajj-related W135 clone (5,6).

The 3 cases we report were observed in southeastern China near Taiwan (online Technical Appendix), but no direct epidemiologic links are known. Because of the lack of W135 strains from Hajj pilgrimages and Taiwan in this study, we could not provide a detailed and integrated genotypic relationship between the strains in China and those of Hajj pilgrimages and Taiwan. However, we can confirm that these 3 cases were caused by strains from the same hypervirulent clone characterized as ST11: P1.5, 2.

W135 strains have been isolated after vaccination with a bivalent meningococcal vaccine in Cameroon (7). In China, the bivalent meningococcal vaccine has been successfully introduced into the national expanded immunization program in response to an outbreak of N. meningitidis serogroup C during 2003–2004 (8).