can produce similar airway cast formation in humans; severe respiratory distress reflects extensive obstruction of the respiratory system.

Healthcare providers should be aware of the possibility of bronchial casts when examining children with influenza (H1N1) infection accompanied by atelectasis. Steroids can be administered early in infection to avoid cast formation, and antiviral drug therapy and respiratory support can be used for influenza (H1N1)–infected children in whom airway casts have developed.

Acknowledgments

We thank Naoko Chiba and Akiko Ono for assistance with manuscript preparation.

This work was supported in part by a Grant-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology of Japan (no. 21390306) to T.T.

Maki Hasegawa, Yasuji Inamo, Tatsuo Fuchigami, Koji Hashimoto, Miyuki Morozumi, Kimiko Ubukata, Haruo Watanabe, and Takashi Takahashi

Author affiliations: Nihon University Nerima-Hikarigaoka Hospital, Tokyo, Japan (M. Hasegawa, Y. Inamo, T. Fuchigami, K. Hashimoto); Graduate School of Infection Control Sciences, Kitasato University, Tokyo (M. Morozumi, K. Ubukata, T. Takahashi); and National Institute of Infectious Diseases, Tokyo (H. Watanabe)

DOI: 10.3201/eid1602.091607

References

1. Shinde V, Bridges CB, Uyeki TM, Shu B, Balish A, Xu X, et al. Triple-reassortant swine influenza A (H1) in humans in the United States, 2005–2009. N Engl J Med. 2009;360:2616–25. DOI: 10.1056/NEJMoa0903812
2. Novel Swine-Origin Influenza A (H1N1) Virus Investigation Team, Dawood FS, Jain S, Finelli L, Shaw MW, Lindstrom S, et al. Emergence of a novel swine-origin influenza A (H1N1) virus in humans. N Engl J Med. 2009;360:2605–15. DOI: 10.1056/NEJMoa0903810
3. Madsen P, Shah SA, Rubin BK. Plastic bronchitis: new insights and a classification scheme. Paediatr Respir Rev. 2005;6:292–300. DOI: 10.1016/j.prrv.2005.09.001
4. Kruger J, Shpringer C, Picard E, Kerem E. Thoracic air leakage in the presentation of cast bronchitis. Chest. 2009;136:615–7. DOI: 10.1378/chest.08-0383
5. Hasegawa M, Hashimoto K, Morozumi M, Ubukata K, Takahashi T, Inamo Y. Spontaneous pneumomediastinum complicating pneumonia in children infected with 2009 pandemic influenza A(H1N1) v virus. Clin Microbiol Infect. 2009 Oct; [Epub ahead of print]. DOI: 10.1111/j.1469-0691.2009.03086.x
6. Hamano-Hasegawa K, Morozumi M, Nakayama E, Chiba N, Murayama SY, Takayanagi R, et al. Comprehensive detection of causative pathogens using real-time PCR to diagnose pediatric community-acquired pneumonia. J Infect Chemother. 2008;14:424–32. DOI: 10.1007/s10156-008-0648-6
7. Noizet O, Leclerc F, Leteurtre S, Brichet A, Pouessel G, Dorkenoo A, et al. Plastic bronchitis mimicking foreign body aspiration that needs a specific diagnostic procedure. Intensive Care Med. 2003;29:329–31. DOI: 10.1007/s00134-002-1610-1
8. Nayar S, Parmar R, Kulkarni S, Cherian KM. Treatment of plastic bronchitis. Ann Thorac Surg. 2007;83:1884–6. DOI: 10.1016/j.athoracsur.2006.12.027
9. Nili H, Asasi K. Natural cases and an experimental study of H9N2 avian influenza in commercial broiler chickens of Iran. Avian Pathol. 2002;31:247–52. DOI: 10.1080/03079450220136567

Addresses for correspondence: Yasuji Inamo, Department of General Pediatrics, Nihon University Nerima-Hikarigaoka Hospital, Nihon University School of Medicine, 2-11-1, Hikarigaoka, Nerima-ku, Tokyo, 179-0072, Japan; email: y-inamo@pb3.so-net.ne.jp; or Takashi Takahashi, Laboratory of Infectious Diseases, Graduate School of Infection Control Sciences, Kitasato University, 5-9-1 Shirokane, Minato-ku, Tokyo 108-8641, Japan; email: taka2si@lisci.kitasato-u.ac.jp

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 *Sma*I 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 (PaO_2 53 mm Hg and $PaCO_2$ 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.

Laura Soavi, Roberto Stellini, Liana Signorini, Benvenuto Antonini, Palmino Pedroni, Livio Zanetti, Bruno Milanesi, Annalisa Pantosti, Alberto Matteelli, Angelo Pan, and Giampiero Carosi

Author affiliations: University of Brescia, Brescia, Italy (L. Soavi, R. Stellini, L. Signorini, A. Matteelli, G. Carosi); Presidio Ospedaliero di Manerbio, Manerbio, Italy (B. Antonini, P. Pedroni, L. Zanetti, B. Milanesi); Istituto Superiore di Sanità, Rome, Italy (A. Pantosti); and Istituti Ospitalieri di Cremona, Cremona, Italy (A. Pan)

DOI: 10.3201/eid1602.091478

References

1. Huijsdens XW, van Dijke BJ, Spalburg E, van Santen-Verheuvel MG, Heck ME, Pluister GN, et al. Community-acquired MRSA and pig-farming. Ann Clin Microb Antimicrob. 2006;5:26. DOI: 10.1186/1476-0711-5-26
2. van Loo I, Huijsdens X, Tiemersma E, de Neeling A, van de Sande-Bruinsma N, Beaujean D, 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, Sekkat M, Carpaij 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, Sorum M, van Nes A, Skov R, Melchers WJ, Klaassen CH, 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. Juhasz-Kaszanyitzky E, Janosi S, Somogyi 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/onderwerpsort.asp

10. Wulf MW, Markestein A, van der Linden FT, Voss A, Klaassen C, Verduin CM. First outbreak of methicillin-resistant *Staphylococcus aureus* ST398 in a Dutch hospital, June 2007. Euro Surveill. 2008;13(9):pii 8051.

Address for correspondence: Laura Soavi, Institute of Infectious and Tropical Diseases, Spedali Civili, Piazzale Spedali Civili 1, 25123 Brescia, Italy; email: laura.soavi@tiscali.it

Neisseria meningitidis Serogroup W135, China

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 antiserum (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 (*por*A) 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*).