

The role of steroids in treating patients with *S. suis* infection remains unclear, although this case illustrates that the inflammation associated with this infection can be profound and can require prolonged steroid therapy. Since at least 2 cases of relapse have been reported after 2 and 4 weeks of treatment (1), prolonged therapy should be considered for infections caused by this pathogen. Hearing loss from *S. suis* meningitis occurs frequently and can be irreversible (1). Hawaii's swine industry is characterized by small herds and a high degree of concentration (9). This case of human *S. suis* meningitis in Hawaii emphasizes the need for these data to be generated and made available. Indeed, this bacterium is increasingly recognized as a significant zoonotic agent in Asia; although it remains a relatively rare cause of human infection elsewhere, persons in close occupational contact with pigs or pork products are at higher risk than others (1). Increasing awareness of this disease is expected to help counter human *S. suis* infections.

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References

1. Gottschalk M, Segura M, Xu J. *Streptococcus suis* infections in humans: the Chinese experience and the situation in North America. *Anim Health Res Rev.* 2007;8:29–45. DOI: 10.1017/S1466252307001247
2. Mai NT, Hoa NT, Nga TV, Linh LD, Chau TT, Sinh DX, et al. *Streptococcus suis* meningitis in adults in Vietnam. *Clin Infect Dis.* 2008;46:659–67. DOI: 10.1086/527385

3. Suankratay C, Intalaporn P, Nuntaphisud P, Arunyingmongkol K, Wilde H. *Streptococcus suis* meningitis in Thailand. *Southeast Asian J Trop Med Public Health.* 2004;35:868–76.
4. Yu H, Jing H, Chen Z, Zheng H, Zhu X, Wang H, et al. Human *Streptococcus suis* outbreak, Sichuan, China. *Emerg Infect Dis.* 2006;12:914–20.
5. Lee GT, Chiu CY, Haller BL, Denn PM, Hall CS, Gerberding JL. *Streptococcus suis* meningitis, United States. *Emerg Infect Dis.* 2008;14:183–5. DOI: 10.3201/eid1401.070930
6. Willenburg KS, Sentochnik DE, Zadzoks RN. Human *Streptococcus suis* meningitis in the United States. *N Engl J Med.* 2006;354:1325. DOI: 10.1056/NEJMe053089
7. Higgins R, Gottschalk M. An update on *Streptococcus suis* identification. *J Vet Diagn Invest.* 1990;2:249–52.
8. Vecht U, Wisselink HJ, Jellema ML, Smith HE. Identification of two proteins associated with virulence of *Streptococcus suis* type 2. *Infect Immun.* 1991;59:3156–62.
9. Sharma K, Leung PS, Zaleski H. Economic analysis of size and feed type of swine production in Hawaii. *Swine Health and Production.* 1997;5:103–10.

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have 1 Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

**Chorioamnionitis
and Neonatal
Sepsis from
Community-
associated MRSA**

To the Editor: Chorioamnionitis is a common cause of maternal and neonatal illness and death (1), but chorioamnionitis attributed to *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA), is reported infrequently (2–5). In the context of the rising incidence of community-associated MRSA (CA-MRSA) infections (6), we report an apparent case of CA-MRSA chorioamnionitis.

The patient, a 31-year-old woman with polycystic ovary syndrome and hypothyroidism, had 1 prior pregnancy but no viable offspring. After a clomiphene-assisted conception, routine ultrasound at 21 weeks' gestation identified a shortened cervix (5 mm). The patient declined amniocentesis for cerclage and was treated with pelvic rest and vaginal progesterone. Five days later, she arrived at the emergency department with foul-smelling vaginal discharge. At this time, the patient was afebrile and hemodynamically stable, had no abdominal pain, and had a leukocyte count of 9.5×10^3 cells/mm³.

Premature rupture of membranes was diagnosed, and the patient was admitted and administered intravenous ampicillin and azithromycin. Nine days into treatment, at 23 weeks' gestation, 210 hours after membrane rupture, a 415-g live-born girl was delivered spontaneously in footling breech with Apgar scores of 1 (1 min) and 5 (5 min). During admission, the mother was never febrile and did not complain of abdominal tenderness or chills. The highest leukocyte count was 12.4×10^3 cells/mm³. The mother was discharged the day after delivery without further complications. At 6-week follow-up, she remained well, with no signs of infection.

Pathologic examination of the placenta demonstrated focal acute funisitis, acute chorioamnionitis with fetal surface acute arteritis and acute decidualitis. Cultures from the maternal and fetal sides of the placenta grew predominantly MRSA and rare colonies of methicillin-susceptible *S. aureus*. The MRSA antimicrobial drug profile, including trimethoprim/sulfamethoxazole and clindamycin susceptibility, was characteristic of CA-MRSA (6).

The neonate, who died on day 16, was culture-positive for CA-MRSA from blood, 2 umbilical swabs, and a tracheal aspirate. The antibiogram of these isolates was identical to the placental cultures, including absence of inducible clindamycin resistance. Postmortem examination showed hemorrhagic necrotizing pneumonia and gram-negative bacilli. Culture of lung tissue grew *Escherichia coli*. Isolates from the placenta and neonate were identified phenotypically, without molecular testing.

Maternal complications of chorioamnionitis include endometritis, bacteremia, hemorrhage, and cesarean delivery (1). Clinically, chorioamnionitis can be diagnosed by maternal fever (>38°C) and 2 of the following: maternal leukocytosis (>15 × 10³ cells/mm³), maternal tachycardia (>100 bpm), fetal tachycardia (>160 bpm), uterine tenderness, and foul-smelling amniotic fluid (1). This patient had none of these signs, except foul-smelling amniotic fluid, and fetal tachycardia was absent. In this case, chorioamnionitis was diagnosed by histology.

Amniotic fluid cultures from pregnancies complicated by chorioamnionitis have shown multiple organisms from the vaginal flora, such as *Streptococcus agalactiae*, *Gardnerella vaginalis*, *Mycoplasma hominis*, *Ureaplasma urealyticum*, anaerobes, and *E. coli* (1). Chorioamnionitis associated with *S. aureus* is uncommon (2,3), and MRSA chorioamnionitis is rare (4,5). The first 2 reports of MRSA chorioamnionitis appeared in 1998

(4) and 2002 (5). In both instances, the patients worked in the healthcare industry, and the authors considered the MRSA to have been nosocomial strains. The patient in our report was a restaurant manager, had no prior recorded hospital admissions, and was not previously known to be colonized by MRSA.

CA-MRSA strains are epidemiologically and clonally unrelated to hospital-associated MRSA (HA-MRSA) strains and can be differentiated by the presence of staphylococcal cassette chromosome *mec* type IV and the absence of multidrug resistance seen with HA-MRSA (6). Recently, the incidence of CA-MRSA infections increased in community settings, including outbreaks in settings in which CA-MRSA is endemic, with manifestations ranging from skin and soft tissue infections to necrotizing pneumonia (6). Genital colonization with MRSA recently has been reported with a frequency of 0.5%–3.5% in pregnant women (7,8). In 1 study, most (93%) of these isolates were CA-MRSA (7).

Eckhardt et al. described a patient with chorioamnionitis in whom CA-MRSA bacteremia developed (9). However, this descriptor was used to specify multidrug-resistant MRSA not acquired in a hospital. Moreover, neither placental nor amniotic fluid cultures were described. Laibl et al. reported 2 patients with CA-MRSA infections in whom chorioamnionitis developed (10). Again, placental and amniotic fluid culture results were not reported, nor was chorioamnionitis listed as an infection caused by CA-MRSA in their cohort. However, these latter 2 patients might represent additional cases of CA-MRSA chorioamnionitis.

Although the incidence of CA-MRSA infections continues to increase, CA-MRSA chorioamnionitis appears to remain rare. Nevertheless, the prevalence of MRSA genital colonization among pregnant women cre-

ates an opportunity for this agent to cause ascending gestational infection. This finding is meaningful because recommended empirical antimicrobial drug treatments may not cover CA-MRSA, increasing the likelihood of infectious complications (1). However, culture results when available can provide therapeutic guidance. We hope this report raises awareness of the possibility of CA-MRSA chorioamnionitis and encourages reports from other authors so this entity can be better established, characterized, and monitored.

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References

1. Newton ER. Preterm labor, preterm premature rupture of membranes, and chorioamnionitis. *Clin Perinatol*. 2005;32:571–600. DOI: 10.1016/j.clp.2005.05.001
2. Ben-David Y, Hallak M, Evans MI, Abramovici H. Amnionitis and premature delivery with intact amniotic membranes involving *Staphylococcus aureus*. A case report. *J Reprod Med*. 1995;40:485–6.
3. Negishi H, Matsuda T, Okuyama K, Sutoh S, Fujioka Y, Fujimoto S. *Staphylococcus aureus* causing chorioamnionitis and fetal death with intact membranes at term. A case report. *J Reprod Med*. 1998;43:397–400.
4. Geisler JP, Horlander KM, Hiatt AK. Methicillin resistant *Staphylococcus aureus* as a cause of chorioamnionitis. *Clin Exp Obstet Gynecol*. 1998;25:119–20. DOI: 10.1016/S0889-8545(05)70361-2
5. Fowler P. Methicillin-resistant *Staphylococcus aureus* chorioamnionitis: a rare cause of fetal death in our community. *Aust N Z J Obstet Gynaecol*. 2002;42:97–8. DOI: 10.1111/j.0004-8666.2002.00109.x
6. Palavecino E. Community-acquired methicillin-resistant *Staphylococcus aureus* infections. *Clin Lab Med*. 2004;24:403–18. DOI: 10.1016/j.cll.2004.03.007
7. Chen KT, Huard RC, Della-Latta P, Saiman L. Prevalence of methicillin-sensitive and methicillin-resistant *Staphylococcus aureus* in pregnant women. *Obstet Gynecol*. 2006;108:482–7.

8. Andrews WW, Schelonka R, Waites K, Stamm A, Cliver SP, Moser S. Genital tract methicillin-resistant *Staphylococcus aureus*: risk of vertical transmission in pregnant women. *Obstet Gynecol*. 2008;111:113–8.
9. Eckhardt C, Halvosa JS, Ray SM, Blumberg HM. Transmission of methicillin-resistant *Staphylococcus aureus* in the neonatal intensive care unit from a patient with community-acquired disease. *Infect Control Hosp Epidemiol*. 2003;24:460–1. DOI: 10.1086/502234
10. Laibl VR, Sheffield JS, Roberts S, McIntire DD, Trevino S, Wendel GD Jr. Clinical presentation of community-acquired methicillin-resistant *Staphylococcus aureus* in pregnancy. *Obstet Gynecol*. 2005;106:461–5.

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Methicillin-Resistant *Staphylococcus aureus* in Marine Mammals

To the Editor: Methicillin-resistant *Staphylococcus aureus* (MRSA) is emerging as an important cause of illness and death in animals and has been found in an impressive variety of species. However, to date, only 2

studies have reported the isolation of MRSA from marine mammals, 1 seal (1) and 3 bottlenose dolphins (2). We describe an investigation that was conducted after MRSA was isolated from a dolphin at a marine park in North America.

In November 2006, a 20-year-old, male, captive, bottlenose dolphin, suspected of having pneumonia, was treated empirically with ciprofloxacin and itraconazole. Despite treatment, the dolphin died in December 2006. A necropsy was performed, and a culture swab specimen of the blowhole was submitted for bacteriologic examination; MRSA was then isolated. The clinical relevance of this finding was unclear. Pulsed-field gel electrophoresis (PFGE) was conducted (3), and results indicated that the MRSA strain isolated was the Canadian epidemic MRSA (CMRSA)2 (USA100) strain, the predominant hospital- and community-associated MRSA strain found in persons in Canada (4). To determine the extent of MRSA colonization in this marine park, blowhole swab specimens were collected from dolphins, orcas, and beluga whales, and nasal swab specimens were collected from walruses, sea lions, harbor seals, gray seals, and park personnel, excluding 4 employees in January 2007. Selective culture for MRSA was performed, and strains were typed with PFGE (3) and *spa* typing (5). All MRSA strains were investigated for the Pantone-Valentine leukocidin (PVL) toxin genes (6).

In January 2007, MRSA was not isolated from personnel (0/22), sea

lions (0/12), harbor seals (0/2), gray seals (0/2), orcas (0/4), or beluga whales (0/23); it was isolated from dolphins (2/6, 33.3%) and a walrus (1/6, 16.7%). To reduce the risk for MRSA transmission among the marine mammals and to personnel, the following steps were recommended: colonized animals were isolated, contact with colonized animals was restricted, all park personnel were required to wear gloves and masks when handling colonized animals, and routine hand hygiene was emphasized. Colonized walruses were isolated in a separate facility until May 2007. Because of space limitations, colonized dolphins could not be isolated. Although the park instituted a strict policy that required personnel to wear gloves and masks, this policy ceased during the summer months due to the park's exhibition schedule.

Because we knew from our observations of other animal species that natural decolonization with MRSA is common, as well as lacking information about antimicrobial drug efficacy for MRSA decolonization in marine mammals, and had concerns regarding the emergence of further antimicrobial drug resistance, we recommended that no attempt be made to decolonize the animals with antimicrobial agents. After these recommendations were made and implemented, follow-up testing for MRSA colonization was performed on the dolphins and walruses throughout 2007 and 2008 (Table). In October 2007, testing conducted on all sea lions, harbor seals, gray seals, orcas,

Table. MRSA colonization status of dolphins and walruses during 2007–2008*

Date	No. (%) dolphins MRSA positive	Identification nos. of MRSA-positive dolphins	No. (%) walruses MRSA positive	Identification nos. of MRSA-positive walruses
2007 Jan	2/6 (33.3)	2, 3	1/6 (16.7)	1
2007 Feb	2/6 (33.3)	2, 4	2/5 (40)	2, 3
2007 Apr	2/5† (40)	3, 5	0/6 (0)	NA
2007 May	2/3 (66.7)	3, 5	0/6	NA
2007 Oct	1/5 (20)	3	0/5	NA
2008 May	1/5 (20)	3	NT	NA
2008 Jul	0/5	NA	NT	NA
2008 Oct	0/5	NA	NT	NA

*MRSA, methicillin-resistant *Staphylococcus aureus*; NA, not applicable; NT, not tested.

†Dolphin 2 died due to unknown circumstances.