Asymptomatic Nasal Carriage of Mupirocin-Resistant, Methicillin-Resistant *Staphylococcus aureus* (MRSA) in a Pet Dog Associated with MRSA Infection in Household Contacts

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Recurrent methicillin-resistant *Staphylococcus aureus* (MRSA) infection in a patient with diabetes and in his wife is described. Culture of nares samples from the family dog grew mupirocin-resistant (minimum inhibitory concentration >1024 μg/mL) MRSA that had a pulsed-field gel electrophoresis chromosomal pattern identical to the MRSA isolated from the patient’s nares and his wife’s wound. Further recurrence of MRSA infection and nasal colonization in the couple was prevented only after successful eradication of MRSA from the family dog’s nares.

Methicillin-resistant *Staphylococcus aureus* (MRSA) is an important cause of nosocomial—and, increasingly, community-acquired—infection [1]. Person-to-person spread of MRSA in the household setting has been reported previously [2–4]. Eradication of MRSA colonization from nares has been complicated by the emergence of mupirocin resistance [5]. I report the isolation of mupirocin-resistant MRSA from the nares of an apparently healthy dog belonging to a patient who, along with the patient’s wife, repeatedly had bouts of soft-tissue MRSA infections. The relatedness of the MRSA isolates from the dog and household contacts was demonstrated by PFGE of chromosomal DNA.

**Case report.** A 48-year-old man with diabetes mellitus and chronic renal insufficiency was hospitalized on 30 January 2001 for treatment of a right leg-stump infection. Previously, on 1 December 2000, the patient had been admitted for right below-knee amputation because of a nonhealing wound, and readmitted on 8 December 2000 for a culture-negative surgical-site infection; for the latter, he was treated with intravenous vancomycin. On 25 January 2001, a culture of stump drainage fluid grew MRSA that was resistant in vitro to most antibiotics tested (including trimethoprim-sulfamethoxazole, erythromycin, gentamicin, clindamycin, and tetracycline) but was susceptible to vancomycin and rifampin; culture of nares samples also grew MRSA with an identical susceptibility pattern. The patient was treated with intravenous vancomycin, a 10-day course of daily chlorhexidine baths, and topical mupirocin ointment applied twice per day to the nares. Despite resolution of cellulitis and completion of decolonization therapy, cultures indicated the presence of MRSA in the patient’s nares on 26 April, 18 May, and 11 June 2001. Attempts at decolonization with a 2-week course of oral rifampin and topical mupirocin in May 2001 were again unsuccessful, despite in vitro susceptibility of the isolate to rifampin. On 12 April and 27 July 2001, the patient’s wife, a kidney transplant recipient with diabetes, had MRSA cellulitis diagnosed (on the basis of wound cultures and clinical exam) after biopsies of seemingly minor skin lesions. In both instances, the antibiogram of the isolates was identical to that of the patient’s, and nares cultures failed to grow MRSA.

On 4 August 2001, because of concern over an unidentified source of MRSA within the household, nares cultures were performed for the only other household member, a healthy 18-month-old female Dalmatian. The culture grew MRSA with a susceptibility pattern identical to that of the previous isolates from the patient and his wife. A second culture of samples from the dog’s nares obtained 9 days later confirmed the presence of MRSA. A previous MRSA isolate from the patient’s nares and the original isolate from the dog’s nares were both tested against mupirocin by Etest (AB Biodisk) and found to be highly resistant to this drug (MIC >1024 μg/mL)[5]. Genomic DNA typing by PFGE of chromosomal DNA of the same isolates from the patient and the dog, as well as an isolate from the wife’s earlier wound cultures, revealed indistinguishable profiles; another previous isolate from the patient’s wound was closely related (2-band differences) to these 3 isolates (figure 1). On further questioning, the couple reported that the dog routinely slept in their bed and frequently licked their faces. Application of vancomycin ointment (5%) to the nares 2 times/day for 10 days was prescribed for the patient, his wife, and the dog. For the dog, a cotton swab laden with the ointment was inserted 1–2 cm into each nostril and twirled several times. Avoidance of intimate contact with the dog was also urged. On
11 September 2001 (2 weeks after completion of decolonization therapy), culture of samples from the nares of the patient failed to grow MRSA from the nares of the patient, but the dog’s nares remained culture-positive. Concurrent culture of nares samples from the patient’s wife grew MRSA for the first time. Another course of vancomycin ointment to the nares, similar to the previous regimen, was prescribed for all 3 household members. On 11 October 2001, nares cultures for the patient and the dog grew MRSA, but the result of nares culture for the patient’s wife was negative. The 10-day vancomycin ointment regimen was repeated for all household members.

On 30 October 2001, a few days after the completion of decolonization therapy, the patient developed another bout of MRSA infection in his leg stump, despite a negative nares culture result; his wife had no open wounds or evidence of skin infection at that time. Another course of vancomycin ointment, similar to the prior regimen but for a longer duration (2 weeks), was prescribed for the patient, his wife, and the dog. Cultures of nares samples from the couple and the dog obtained 2 weeks after completion of therapy were negative for MRSA.

On 6 February 2002, the patient presented to the infectious disease clinic with a new pressure ulcer above the right leg stump. Although there was no evidence of infection around the ulcer, a wound swab was obtained; it showed no evidence of MRSA. Four months after decolonization therapy of the entire family ended, the patient and his wife remained free of MRSA infection. The dog remained asymptomatic.

**Discussion.** To the best of my knowledge, this is the first reported case of isolation of mupirocin-resistant MRSA from an animal, a finding with potentially significant ramifications, given the frequent use of mupirocin as the first-line treatment for nasal colonization in humans. It is also the first report of MRSA isolation from an animal in which the relatedness of the isolate to that of its human contacts was proved by PFGE.

There has been only one other report of MRSA isolation from a dog, but that case differs from the present one in that the dog had apparently been symptomatic (it had an eye infection), mupirocin susceptibility testing was not performed on the isolate, and only phage typing was done to prove the isolate’s relatedness to the isolates from the household contacts [6]. Compared with PFGE, phage typing has been shown to have inferior discriminatory capability in distinguishing MRSA isolates from different sources [7].

The carriage of MRSA by the dog might have played an important epidemiological role in the recurrent infections and colonizations in the patient and his wife. There are several reasons to suspect this. First, the antibiogram and PFGE patterns of the MRSA isolate from the dog were identical to those of the isolates from the patient’s nares samples and from the patient’s wife’s wound; they were also closely related to those of the isolate from the patient’s wound, on the basis of its PFGE pattern. Second, despite the apparent eradication of MRSA from the patient’s nares, samples from his wife’s nares became culture positive, attendant with the persistence of MRSA in the dog’s nares (in September 2001). Third, despite the eradication of MRSA from the nares of the patient and a recent negative nares culture result for his wife, he acquired another MRSA leg-stump infection (October 2001). Last, termination of the infection and colonization cycle from the patient and his wife was only possible after the successful eradication of MRSA from the dog’s nares.

It is likely that the dog (which had not been ill or hospitalized or received antibiotics previously) initially became colonized by MRSA through contact (often intimate) with the patient, who had previously been hospitalized. In turn, the dog likely served as a source of reinfection or recolonization for both the patient and his wife. Direct person-to-person transmission of MRSA between the patient and his wife might have also occurred, but this possibility would have been less likely in October 2001, when the patient experienced another bout of wound infection in the absence of MRSA in the couple’s nares.

In 1959, Mann [8, pp. 469–70] reported the isolation of *S. aureus* from the nostrils of 23 of 100 dogs and proposed that “the common house pet can serve as an important reservoir or carrier of staphylococci infective for man.” Subsequent studies reported that, compared with many other animals, dogs seem to be particularly susceptible to nasal carriage of *S. aureus* [9–12], with some studies reporting up to a 70%–88% carriage rate in the canine population [10, 11]. In 1961, Live and Nichols [13] reported a high rate of nasal carriage of antibiotic-resistant,
coagulase-positive staphylococci among fourth-year veterinary students, many of whom also had skin infections caused by the same organism. The high prevalence of antibiotic-resistant staphylococci among hospitalized dogs, who often received antibiotics as prophylaxis and therapy, was thought to have contributed to colonization and disease in these students. The authors concluded that “antibiotic-resistant staphylococci have become established in veterinary hospitals, which may act as a source of the organisms for human beings associated with them as well as for hospitalized animals” [13, p. 203]. Experimental cross-transmission of S. aureus from dogs to kennel attendants has also been demonstrated [11].

Despite these published reports, the potential for dogs to serve as a source of S. aureus infections in humans has gone relatively unnoticed. Indeed, none of the 4 commonly referenced textbooks of infectious diseases even mention the possibility of S. aureus carriage in pet animals serving as a source of infection in humans [14–17]. One possible explanation of the general disregard for the findings of the aforementioned studies may be that the relatedness of canine isolates to isolates from the human contacts was never definitively demonstrated, because of the inadequate discriminatory abilities of antibiotic and phage typing for distinguishing different strains of S. aureus, compared with PFGE. With the increasing prevalence of MRSA in the population and its attendant higher rate of infection following colonization, as compared with methicillin-sensitive S. aureus [18], investigation into asymptomatic MRSA carriage in canine household members may need serious consideration for patients with recurrent community-acquired MRSA infections. Furthermore, the increasing popularity of pet therapy, particularly in health-care facilities [19, 20], where MRSA may be endemic, also raises questions about the possibility of asymptomatic canine acquisition and the subsequent spread of MRSA in such settings; further studies are needed to explore this possibility. Although the treatment of MRSA carriage by the application of topical antimicrobial agents to dog nares has not been studied systematically, my experience with the case described here suggests that it may be more difficult to eradicate MRSA carriage from dog nares than from human nares, given that several attempts were required before decolonization was successful. Last, although topical vancomycin therapy was used because of the mupirocin resistance of the MRSA isolates from the dog and the patient, this treatment cannot be routinely recommended because of increasing resistance to vancomycin among S. aureus isolates [21].

In conclusion, until further studies are performed, the evaluation of patients with recurrent MRSA colonization or infection that does not have an obvious source should prompt queries regarding any regular contact with pet dogs, particularly in household settings. Any investigation aimed at identifying hidden carriers of MRSA among close patient contacts should not overlook “man’s best friend.”

References