Nosocomial Dengue by Mucocutaneous Transmission

To the Editor: Wagner and colleagues report nosocomial dengue transmitted by needlestick and note that it is the fourth case of nosocomial dengue to their knowledge (1). In the same issue of Emerging Infectious Diseases, Nemes and colleagues report a separate case of nosocomial dengue also transmitted by needlestick (2). Three other cases of nosocomial dengue transmission by needlestick have previously been published (3–5).

We have recently published a case of nosocomial dengue infection that was transmitted by mucocutaneous exposure to blood from a febrile traveler who had recently returned from Peru (6). During phlebotomy, a healthcare worker was splashed in the face with the traveler’s blood. Both the traveler and the healthcare worker were subsequently found to have dengue fever with dengue virus type 3. This route of infection is biologically plausible because infection through mucosal surfaces (intranasal and oral routes) has been shown possible for arboviruses (7). In our review of the literature, we also found a report of dengue virus transmission by bone marrow transplantation (8). Other cases of transmission of dengue virus without a mosquito vector have occurred in 5 reported instances of infection in newborns as a result of intrapartum or vertical transmission from mother to child (9–12).

We agree that nosocomial transmission may become more common in temperate areas as more travelers return home with acute dengue fever. As Wagner and colleagues pointed out, travelers visiting Southeast Asia have the greatest risk of acquiring dengue infections because of the high endemicity of these viruses there. Our report further illustrates the occurrence of dengue infection in the Americas (13) and the risk for dengue to travelers visiting this region. Among 33 returned travelers with dengue infection reported in the United States in 1999 and 2000, 20 had acquired infection in the Caribbean islands (12 cases) or Central or South America (8 cases) (14). Clinicians and laboratorians should be alert to the possibility of acquiring infection with a dengue virus after needlestick or mucocutaneous blood exposure. The magnitude of nosocomial transmission in dengue-endemic areas is unknown and more difficult to assess because healthcare workers may be exposed to dengue virus–infected mosquitoes outside the clinical setting.

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of new or unusual infections (2,3). This information is essential for public health and medical interventions. As outlined by Louie et al., hospital autopsy rates have dropped to single digits, and concerns by pathologists about occupational risks and biosafety have likely contributed to this decline. Currently, the last stronghold of autopsy expertise is forensic pathology (4). However, the medicolegal death investigative system does not have jurisdiction over all potential infectious disease deaths nor is it adequately supported to assume the cases that are missed by our present hospital autopsy system. Additionally, many medicolegal and hospital autopsy facilities with outdated or poorly-designed air flow systems are ill suited to handle autopsies when infectious disease is suspected (5). Air-handling systems can be expensive to fix.

Reference centers such as the National Prion Disease Pathology Surveillance Center, while providing diagnostic expertise, fail to surmount the biosafety obstacles (real and perceived) that prevent pathologists from enthusiastically performing autopsies on those who died of potential infectious diseases, including prion diseases. One potential solution is the creation of regional centers of excellence for infectious disease autopsies that could operate in conjunction with a mobile containment autopsy facility (5,6). Such centers could provide diagnostic expertise as well as biosafety capacity.

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References

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Q Fever Wildlife Reservoir

To the Editor: To the list of zoonotic infections with wildlife sources reported by Kruse et al. (1), I would add Coxiella burnetii infection because of its global extent, extensive presence in the animal kingdom, and potential for use as an agent of bioterrorism (2). C. burnetii causes Q fever, a self-limited disease that usually appears as undifferentiated fever, pneumonia, or hepatitis, but which may progress into chronic disease, especially endocarditis, among susceptible persons. Q fever is endemic worldwide in domestic mammals, especially ungulates (cattle, sheep, and goats), but also has been found in wild mammals, birds, and arthropods. The transmission of Q fever to humans from wild rabbits was documented in the 1980s (3). More recently, a study showed seroprevalence of Q fever ranging from 7% to 53% in brown rats (Rattus norvegicus) in Oxfordshire, which suggests that they are a possible reservoir for C. burnetii in the United Kingdom. The study also speculated why cats, as frequent predators of rats, are important in maintaining the transmission cycle of the disease (4).

A case-control study published in 2001 (5) attempted to define the risk factors for an increase in the incidence of Q fever in French Guiana in 1996. The study found no link between Q fever and domestic ungulates, the usual source of outbreaks. The role of pets, basically dogs and cats, as a reservoir was also excluded. Multivariate analysis showed that living in close proximity to the forest, exposure to wild animals (including bats), and working in public trade or public works were all associated with infection. A strong correlation between large amounts of rainfall and higher incidence of Q fever was found also. All of these findings suggested a wild reservoir as a potential source of the epidemics, although the researchers could not identify a particular species as the specific source.

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References