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References

1. Marlet MVL, Sang DK, Ritmeijer K, Muga RO, Onsongo J, Davidson RN. Emergence or re-emergence of visceral leishmaniasis in areas of Somalia, northeastern Kenya, and south-eastern Ethiopia in 2000–01. Trans R Soc Trop Med Hyg. 2003;97:515–8.
2. Dereure J, El-Safi SH, Bucheton B, Boni M, Kheir MM, Davoust B, et al. Visceral leishmaniasis in eastern Sudan: parasite identification in humans and dogs; host-parasite relationships. Microbes Infect. 2003;5:1103–8.
3. Mukhtar MM, Sharief AH, el Saffi SH, Harith AE, Higazzi TB, Adam AM, et al. Detection of antibodies to *Leishmania donovani* in animals in a kala-azar endemic region in eastern Sudan: a preliminary report. Trans R Soc Trop Med Hyg. 2000;94:33–6.
4. Mutero CM, Mutinga MJ, Ngindu AM, Kenya PR, Amimo FA. Visceral leishmaniasis and malaria prevalence in West Pokot District, Kenya. East Afr Med J. 1992;69:3–8.
5. Wykoff DE, Barnley GR, Winn MM. Studies on kala-azar in Uganda—entomological observations. East Afr Med J. 1969;46:204–7.
6. Chappuis F, Mueller Y, Nguimfack A, Rwakimari JB, Couffignal S, Boelaert M, et al. Diagnostic accuracy of two rK39 antigen-based dipsticks and the formol gel test for rapid diagnosis of visceral leishmaniasis in northeastern Uganda. J Clin Microbiol. 2005;43:5973–7.
7. Bern C, Joshi AB, Jha SN, Das ML, Hightower A, Thakur GD, et al. Factors associated with visceral leishmaniasis in Nepal: bednet use is strongly protective. Am J Trop Med Hyg. 2000;63:184–8.
8. Collin SM, Coleman PG, Ritmeijer K, Davidson RN. Unseen Kala-azar deaths in south Sudan (1999–2002). Trop Med Int Health. 2006;11:509–12.
9. Ryan JR, Mbui J, Rashid JR, Wasunna MK, Kirigi G, Magiri C, et al. Spatial clustering and epidemiological aspects of visceral leishmaniasis in two endemic villages, Baringo District, Kenya. Am J Trop Med Hyg. 2006;74:308–17.

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Chikungunya Virus Infection in Traveler to Australia

To the Editor: Chikungunya is a mosquitoborne alphavirus in the family *Togaviridae*. Recently, a chikungunya virus epidemic that affected thousands of persons occurred in islands in the southwestern Indian Ocean, including Mauritius and Reunion (*1*). An outbreak is ongoing in India, and cases are being exported to many other countries (*2–4*) The likelihood of importation of exotic infectious agents into Australia increased during events such as the March 2006 Commonwealth Games in Melbourne. Urgent diagnosis of rarely seen infections is a travel health challenge, particularly when serologic tests used for diagnosis in areas with high prevalence are not locally available. Only 1 previously diagnosed case of chikungunya virus infection has been reported in Australia; it involved importation of the virus from Indonesia to Darwin in 1989 (*5*). We report a second case of infection with this virus.

A 59-year-old man came to a hospital emergency department in North Melbourne, Australia, on March 12, 2006, 5 days after traveling from Mauritius for the Commonwealth Games. He reported a 2-day history of acute swelling and erythema of the left leg and associated malaise. Twenty-four hours earlier, severe lumbar back pain and arthralgias that involved the lower limbs had developed, with associated fevers, rigors, and headache.

The patient had a temperature of 39°C, bilateral conjunctivitis, tender and markedly swollen Achilles tendons, a swollen left ankle, and a maculopapular rash that involved the left forefoot and anterior portion of the shin. Initial blood examinations showed leukopenia (leukocyte count 1.8×10^9/L, lymphocyte count 0.2×10^9/L), mild thrombocytopenia (platelet count 105×10^9/L), and abnormal liver function test results (alanine aminotransferase 133 IU/μL, γ-glutamyl transpeptidase 141 IU/μL, bilirubin 7 mmol/L). Malaria blood films and dengue serologic results were negative. A validated, in-house, generic alphavirus reverse transcription–PCR (RT-PCR) showed positive results 24 hours after collection of blood when the patient was admitted.

The patient received supportive treatment and was discharged from the hospital 3 days after admission, at which time leukopenia and thrombocytopenia had improved. The patient had fully recovered on review 1 week after discharge.

For virus isolation, plasma and leukocyte fractions were placed onto Vero E6 cells and incubated at 37°C for 5 days. Cells were observed daily for virus-specific cytopathic effects. A virus isolate was obtained after 4 days of cell culture. A 10-mL volume of supernatant from infected cells was applied to a carbon-coated grid, stained with phototungstic acid, and examined by electron microscopy. This procedure showed virus with morphology similar to Togavirus (data not shown).

Chikungunya virus was identified by a heminested RT-PCR for the non-

structural protein 4 gene. First-round primers were AlphaF1 (GenBank accession no. NC004162, nt 6942–6961, 5′-CSATGATGAARTC HGGHATG-3′) and AlphaR (nt 7121–7141, 5′-CTATTTAGGACCRC CGTASAG-3′). Second-round primers were AlphaF2 (nt 7480–7501) (5′-TGGNTBAAYATGGAGGTIAAG-3′) and AlphaR. Sequencing of the second-round product identified the virus. A 339-bp fragment (GenBank accession no. DQ678928) had 97% identity with the African prototype strain S27 isolated in Tanzania (Tanganyika) in 1953 (AF369024) and 100% identity with viral sequences from Reunion Island in 2006 (DQ443544).

Knowledge of distant epidemics aids clinical recognition of infections not commonly seen in Australia. Websites and electronic bulletins (e.g., Promed) are a conduit of information. On the basis of this case-patient, those in Australia became more aware of the chikungunya virus epidemics affecting the islands of the southwestern Indian Ocean.

Laboratory diagnosis of chikungunya virus infection is usually serologic. However, alphavirus infections not endemic to Australia are unlikely to be diagnosed serologically because specific assays are generally available only for those viruses known to circulate in Australia. Because viremia of alphaviruses is brief, success of RT-PCR depends on early admission and clinical recognition of infection (6).

Rapid establishment of a definitive diagnosis had substantial benefits that included management of the febrile patient, reduced need for further investigations, and better prognosis. Infection caused by an introduced arbovirus may have important public health implications in Australia. Because immunity in the Australian population is unlikely, consideration must be given to the potential for transmission of the virus to caregivers and the local community. chikungun-

ya virus is commonly spread by mosquitoes of the genera *Aedes*, including *Aedes aegypti*, *Ae. furcifer-taylori*, *Ae. luteocephalus*, *Ae. albopictus*, and *Ae. dalzieli* (7). The Australian Ross River and Barmah Forest alphaviruses are spread by many species of *Aedes* and *Culex* (8). Because some local species could transmit chikungunya virus, necessary steps should be taken to ensure containment when a patient is viremic.

This case highlights the potential for exotic viruses to be introduced into Australia by visitors or returning travelers and the utility of molecular testing for their rapid detection. The generic nature of the RT-PCR enabled detection of an alphavirus with subsequent specific identification by sequencing. Rapid identification and differentiation in a public health setting minimized the potential for spread of the virus.

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References

1. Schuffenecker I, Iteman I, Michault A, Murri S, Frangeul L, Vaney MC, et al. Genome microevolution of Chikungunya viruses causing the Indian Ocean outbreak. PLoS Med. 2006;3:e263.
2. Chikungunya – Indian Ocean update (11): islands, India. Archive no. 20060330.0961. 2006 Mar 30. [cited 2006 Dec 13]. Available from www.promedmail.org
3. Chikungunya – China (Hong Kong) ex Mauritius: conf. Archive no. 20060402. 0989. 2006 Apr 2. [cited 2006 Dec 13]. Available from www.promedmail.org
4. Chikungunya – Indian Ocean update (17): spread to France. Archive no. 20060421. 1166. 2006 Apr 21. [cited 2006 Dec 13]. Available from www.promedmail.org
5. Harnett GB, Bucens MR. Isolation of Chikungunya virus in Australia. Med J Aust. 1990;152:328–9.
6. Sellner LN, Coelen RJ, Mackenzie JS. Detection of Ross River virus in clinical samples using a nested reverse transcription-polymerase chain reaction. Clin Diagn Virol. 1995;4:257–67.
7. Diallo M, Thonnon J, Traore-Lamizana M, Fontenille D. Vectors of Chikungunya virus in Senegal: current data and transmission cycles. Am J Trop Med Hyg. 1999;60:281–6.
8. Dale PE, Ritchie SA, Territo BM, Morris CD, Muhar A, Kay BH. An overview of remote sensing and GIS for surveillance of mosquito habitats and risk assessment. J Vector Ecol. 1998;23:54–61.

Avian Influenza A (H5N1) Age Distribution in Humans

To the Editor: A total of 229 confirmed human cases of avian influenza A (H5N1) were reported to the World Health Organization (WHO) from 10 countries of Africa, Asia, and Europe in the 30 months leading up to July 4, 2006 (1). WHO has highlighted the skewed age distribution of these confirmed cases toward children and young adults, with relatively few cases in older age categories (2). An explanation for this age bias is currently lacking, although a range of behavioral, biological, demographic, and data-related factors may account for the observed pattern (2,3).

To determine whether the statistical parameters of the case distribution can shed any light on the issue, we reviewed the age profile of patients with confirmed avian influenza A