References

1. Soumahoro MK, Fontenille D, Turbelin C, Pelat C, Boyd A, Flahault A, et al. Imported chikungunya virus infection. Emerg Infect Dis. 2010;16:162–3. http://dx.doi.org/10.3201/eid1601.080776
2. Institut De Veille Sanitaire. Alerte chikungunya dans les Antilles. 2013 Dec. No 2 [cited 2014 Oct 6]. http://www.ars.guadeloupe. sante.fr/Le-point epidemiologique.173867.0.html
3. Caribbean Public Health Agency. Laboratory country report. Port of Spain (Trinidad): The Agency; 2014.
4. Pan American Health Organization, Centers for Disease Control and Prevention. Preparedness and response for chikungunya virus introduction in the Americas. 2011 [cited 2014 Nov 12]. http://stacks.cdc.gov/view/cdc/21188
5. Lam SK, Chua KB, Hooi PS, Rahimah MA, Kumari S, Tharmaratnam M, et al. Chikungunya infection: an emerging disease in Malaysia. Southeast Asian J Trop Med Public Health. 2001;32:447–51.
6. Laras K, Sukri NC, Larasati RP, Bangs MJ, Kosim R, Djauzi XX, et al. Tracking the re-emergence of epidemic chikungunya virus in Indonesia. Trans R Soc Trop Med Hyg. 2005;99:128–41. http://dx.doi.org/10.1016/j.trstmh.2004.03.013
7. Renault P, Solet JL, Sissoko D, Balleydier E, Larrieu S, Filleul L, et al. A major epidemic of chikungunya virus infection on Reunion Island, France 2005–2006. Am J Trop Med Hyg. 2007;77:727–31.
8. Lanciotti, RS, Valadere AM. Transcontinental movement of Asian genotype chikungunya virus [letter]. Emerg Infect Dis. 2014;20:1400–2.
9. Leparc-Goffart I, Nougairede A, Cassadou S, Prat C, de Lamballerie X. Chikungunya in the Americas. Lancet. 2014;383:514. http://dx.doi.org/10.1016/S0140-6736(14)60185-9

Acute Zika Virus Infection after Travel to Malaysian Borneo, September 2014

Dennis Tappe,[1] Stephan Nachtigall,[1] Annette Kapaun,[1] Paul Schnitzler, Stephan Günther, Jonas Schmidt-Chanasit

Author affiliations: Bernhard Nocht Institute for Tropical Medicine/World Health Organization Collaborating Centre for Arbovirus and Haemorrhagic Fever Reference and Research, Hamburg, Germany (D. Tappe, S. Günther, J. Schmidt-Chanasit); University Medical Center Heidelberg, Heidelberg, Germany (S. Nachtigall, A. Kapaun); University of Heidelberg, Heidelberg (P. Schnitzler); German Centre for Infection Research, Hamburg (S. Günther, J. Schmidt-Chanasit)

To the Editor: Zika virus (ZIKV), a mosquito-borne flavivirus, causes Zika fever, a self-limiting febrile and exanthematic arthralgia syndrome closely resembling dengue fever. Most often, signs and symptoms are maculopapular rash, fever, arthralgia, myalgia, headache, and conjunctivitis; edema, sore throat, cough, and vomiting occur less frequently (1). The virus, which was initially isolated from a rhesus monkey (*Macaca mulatta*) in 1947 in Uganda, has come to attention recently after a large outbreak occurred in the western Pacific region, including French Polynesia, New Caledonia, Easter Island, and the Cook Islands (2). Travel-related imported infections have thus been increasingly reported from the western Pacific and sporadically also in travelers to other regions of the world, including Thailand, Indonesia, and Senegal (2,3). ZIKV is transmitted by different *Aedes* mosquito species, and nonhuman primates play a role as reservoirs (1). After the beginning of the ZIKV epidemic in late 2013, a 20-fold increase of Guillain-Barré syndrome incidence was noted in French Polynesia; 1 patient was infected a week before neurologic symptoms started (4). We report an acute ZIKV infection in a traveler returning from Malaysian Borneo who experienced bilateral hearing difficulties during the course of illness.

On September 1, 2014, a 45-year-old woman was seen in an outpatient clinic in Heidelberg, Germany for fever of up to 39°C and maculopapular rash covering her trunk, arms, and legs. Fever had started on August 30, which was 6 days after she had returned from a 3-week vacation to peninsular Malaysia and Sabah, Malaysian Borneo. Laboratory analyses showed a slightly elevated C-reactive protein level of 5.2 mg/L (reference range <5.0), but liver function test and complete blood count results were within reference range. During the next 3 days, the fever subsided, but the patient experienced a sore throat, bilateral conjunctivitis, and a burning sensation of the palms and soles. These symptoms were accompanied by swelling of the hands and increasing arthralgia of the wrists, palms, and fingers. There was no lymphadenopathy. An indirect immunofluorescence assay for ZIKV (3) demonstrated an IgM titer of 1:640 and an IgG titer of 1:320 (cutoff <1:20) on day 6 of illness (Figure). An indirect immunofluorescence assay for dengue virus demonstrated an IgG titer of 1:80 and no IgM (cutoff <1:20).

Two days later, the patient experienced sudden bilateral dull and metallic hearing; in her left ear, she experienced a very short delay between a sound and her perception of the sound. Follow-up ZIKV serologic testing on day 11 of illness showed a decreased IgM titer of 1:160 and an increased IgG titer of 1:2,560 (Figure). Viral neutralization testing (3) of the same sample demonstrated the presence of ZIKV-specific neutralizing antibodies. Chikungunya virus serology results were negative. An archived serum sample from day 3 of illness studied by ZIKV serology and a ZIKV-specific real-time reverse transcription PCR (3) was negative (Figure). Hearing difficulties lasted for 10 days and resolved gradually (Figure).

IgM < 1:20
IgG < 1:20
RT-PCR negative

IgM 1:640
IgG 1:320
RT-PCR negative

IgM 1:160
IgG 1:2,560

fever

exanthema

arthralgia

hearing problems

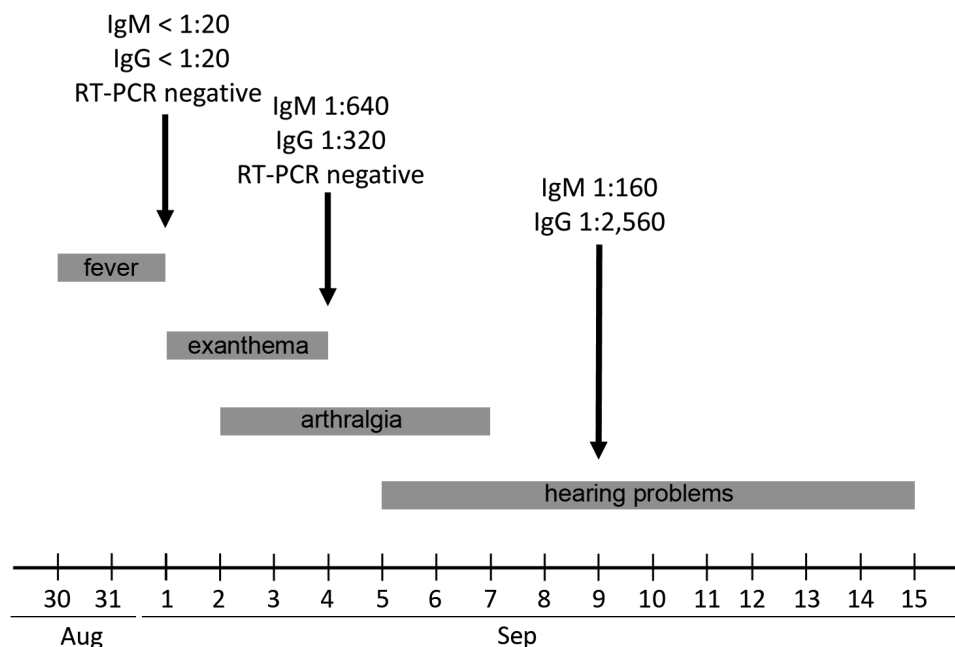| 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |

Aug   Sep

Figure. Clinical course and laboratory results (reverse transcription-PCR [RT-PCR]) for a patient with Zika virus (ZIKV) infection acquired from Malaysian Borneo. Length of gray box indicates duration of symptom.

During her journey to several cities and villages in Sabah, Malaysian Borneo, the patient had noticed several mosquito bites even though she had used repellents. She had stayed in hotels, private homes, and remote church homes under various conditions (online Technical Appendix, http://wwwnc.cdc.gov/EID/article/21/5/14-1960-Techapp1.pdf).

In Asia, Zika fever has been described sporadically in Cambodia, Thailand, and Indonesia (Java and Lombok) (1,3,5,6). On the basis of the incubation time of ≈6 days in returning travelers (2,3), we assumed that the patient became infected in Keningau or surrounding villages, in northern Borneo. Although ZIKV was detected in *Ae. aegypti* mosquitoes in peninsular Malaysia in 1969 (7) and antibodies against ZIKV were demonstrated in serum samples from 15 of 79 patients on peninsular Malaysia and 9 of 50 patients in Borneo in 1953 (8), Zika fever in peninsular Malaysia or Borneo has not been reported. In 2001, ZIKV seropositivity was demonstrated in a native Bornean, 2 migrants to Borneo, and 2 Bornean orangutans (*Pongo pygmaeus*) (9). A later study found an additional 8 Bornean orangutans to be seropositive for antibodies against ZIKV (10). Thus, in Borneo, either the virus only rarely infects humans or the disease is mistaken for dengue fever.

Neurologic complications of ZIKV infections had previously been reported only as Guillain-Barré syndrome, and hearing difficulties in Zika fever patients have not been reported. Because this symptom resolved spontaneously, no audiometry or auditory brainstem response testing was performed, and the cause of the disorder remains unclear. Because of increasing travel and migration and heightened clinical and laboratory awareness, more ZIKV infections are likely to be diagnosed outside of epidemic events.

References

1. Hayes EB. Zika virus outside Africa. Emerg Infect Dis. 2009;15:1347–50. http://dx.doi.org/10.3201/eid1509.090442
2. Wæhre T, Maagard A, Tappe D, Cadar D, Schmidt-Chanasit J. Zika virus infection after travel to Tahiti, December 2013. Emerg Infect Dis. 2014;20:1412–4. http://dx.doi.org/10.3201/eid2008.140302
3. Tappe D, Rissland J, Gabriel M, Emmerich P, Gunther S, Held G, et al. First case of laboratory-confirmed Zika virus infection imported into Europe, November 2013. Euro Surveill. 2014;19:20685.
4. Oehler E, Watrin L, Larre P, Leparc-Goffart I, Lastere S, Valour F, et al. Zika virus infection complicated by Guillain Barré syndrome—case report, French Polynesia. Euro Surveill. 2014;19:20720.
5. Olson JG, Ksiazek TG, Suhandiman, Triwibowo. Zika virus, a cause of fever in Central Java, Indonesia. Trans R Soc Trop Med Hyg. 1981;75:389–93. http://dx.doi.org/10.1016/0035-9203(81)90100-0
6. Olson JG, Ksiazek TG, Gubler DJ, Lubis SI, Simanjuntak G, Lee VH, et al. A survey for arboviral antibodies in sera of humans and animals in Lombok, Republic of Indonesia. Ann Trop Med Parasitol. 1983;77:131–7.
7. Marchette NJ, Garcia R, Rudnick A. Isolation of Zika virus from *Aedes aegypti* mosquitoes in Malaysia. Am J Trop Med Hyg. 1969;18:411–5.
8. Smithburn KC. Neutralizing antibodies against arthropod-borne viruses in the sera of long-time residents of Malaya and Borneo. Am J Hyg. 1954;59:157–63.
9. Wolfe ND, Kilbourn AM, Karesh WB, Rahman HA, Bosi EJ, Cropp BC, et al. Sylvatic transmission of arboviruses among Bornean orangutans. Am J Trop Med Hyg. 2001;64:310–6.
10. Kilbourn AM, Karesh WB, Wolfe ND, Bosi EJ, Cook RA, Andau M. Health evaluation of free-ranging and semi-captive orangutans (*Pongo pygmaeus pygmaeus*) in Sabah, Malaysia. J Wildl Dis. 2003;39:73–83. http://dx.doi.org/10.7589/0090-3558-39.1.73

Address for correspondence: Jonas Schmidt-Chanasit, Bernhard Nocht Institute for Tropical Medicine/World Health Organization Collaborating Centre for Arbovirus and Haemorrhagic Fever Reference and Research, Bernhard-Nocht-Strasse 74, 20359 Hamburg, Germany; email: jonassi@gmx.de

Ebola and Psychological Stress of Health Care Professionals

Marco Lehmann,[1] Christian A. Bruenahl,[1] Bernd Löwe, Marylyn M. Addo, Stefan Schmiedel, Ansgar W. Lohse, Christoph Schramm

Author affiliations: University Medical Center Hamburg-Eppendorf, Hamburg, Germany (M. Lehmann, C.A. Bruenahl, B. Löwe, M.M. Addo, S. Schmiedel, A.W. Lohse, C. Schramm); Schön Klinik Hamburg-Eilbek, Hamburg (M. Lehmann, C.A. Bruenahl, B. Löwe); German Center for Infection Research, Hamburg-Lübeck-Borstel, Germany (M.M. Addo)

To the Editor: Providing medical care for Ebola virus–infected patients entails physical and psychological stress, extended shift times, and risk for infection. In addition, the wearing of personal protective equipment impairs communication and performance of diagnostic and therapeutic procedures. Lessons learned from outbreaks of other infectious diseases indicate that such challenging treatment environments require the monitoring of health care professionals for psychological distress (e.g., anxiety, depression, fatigue, and social isolation) to prevent personal exhaustion and reduced job performance (1).

In August 2014, the first patient in Germany known to have Ebola virus disease was admitted to the University Medical Center Hamburg-Eppendorf (2) and received treatment in the isolation facility for 18 days. We hypothesized that health care professionals working in the isolation unit who had direct contact with the Ebola patient would show more signs of psychological distress than those not working in the isolation unit.

To test our hypothesis, we conducted a cross-sectional controlled study by using validated self-report scales (1,3–5) and open-response questions. Seven days after the Ebola patient was admitted, we distributed questionnaires to the 46 health care professionals (17 physicians, 29 nurses) who had direct contact with the patient (Table).

Of the 46 health care professionals, 30 participated in the study. During patient contact, these staff members wore

Astro-Protect pressurized suits (Asatex, Bergheim, Germany). As a control group, 40 health care professionals from other wards in the same department were recruited and participated in the study. Providers in the control group cared for terminally ill patients and for patients with reduced consciousness, but they had no direct contact with the Ebola patient. The control participants were not recruited from intensive care units because, at the time of the study, the patient was not receiving intensive care treatment. The 2 groups were balanced with respect to age and occupational characteristics (Table). There was no special psychological support service for health care workers in this hospital. Staff members had received mandatory biweekly training, which included decontamination procedures, technical aspects of diagnostic procedures, and emergency care.

In contrast to our hypothesis, no significant differences emerged between the 2 groups with respect to the severity of somatic symptoms, anxiety, depression, and fatigue (Table). Moreover, mean total scores for both groups were at a comparable level to mean scores for the general population (3–5). However, health care professionals who had direct contact with the Ebola patient reported significantly greater social isolation and felt significantly more need for shorter shift hours. The open responses of participants who experienced social isolation suggested that their spouses, children, and other relatives had infection-related concerns. Additionally, half of the participants who did not have direct patient contact reported feeling a need for psychological preparation (Table). Nevertheless, almost all health care professionals (97% of those with direct patient contact, 93% of those without direct patient contact) believed that the health care facilities of the hospital were safe.

Our investigation of the psychological stress of health care professionals in a Western tertiary care center showed that a well-trained and dedicated team can cope well with the stress of caring for a severely ill Ebola patient. Of note, the direct patient contact group tended to comprise more male participants and more participants living with partners, which may have influenced the experience of psychological stress. No staff member refused to participate in the treatment of the Ebola patient, which underlines the high level of motivation within the team and may render direct comparison to other centers difficult.

While the patient was in the isolation unit, working shifts lasted up to 12 hours, consisting of 2 periods with 3–4 hours of work while wearing personal protective equipment in addition to time spent disinfecting. Most respondents felt that these shifts were too long. We therefore suggest that shift durations should be decreased to 8 hours comprising 2 blocks of 2 hours each for direct patient contact. Shorter shifts should improve staff satisfaction with the working conditions and potentially increase the personal safety of all health care personnel involved in direct patient contact.