Guillain-Barré Syndrome after Chikungunya Infection

To the Editor: Chikungunya virus is an RNA alphavirus (group A arbovirus) in the family Togaviridae. The known vectors are *Aedes aegypti* and *Ae. albopictus* mosquitoes. Chikungunya infection, after an incubation period of 2–10 days, has the main clinical manifestations of fever, polyarthralgia, and rash. Treatment consists of rest and medication for pain. Outcome is marked by incapacitating arthralgia, which can persist for several weeks or months (1). Complications are rare and consist of mild hemorrhage, myocarditis, and hepatitis (2). Neurologic manifestations are less well known (3). Infection is confirmed by the identification of genomic products in acute-phase blood specimens, (reverse transcription–PCR (RT-PCR)) or, more recently, by serum immunoglobulin (Ig) M or a 4-fold increase in other antibodies. In 2006, chikungunya virus was found on Réunion Island; seroprevalence on the island was estimated to be 38.2% among 785,000 inhabitants (95% confidence interval 35.9%–40.6%) (4).

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy; incidence worldwide is 0.6–4/100,000 persons/year. In two thirds of patients, neuropathic GBS occurs after an infection (5,6).

Cases of GBS have been described in association with the arboviruses dengue and West Nile but not with chikungunya virus. We report 2 cases of acute and severe GBS related to infection with chikungunya virus.

The first patient was a 51-year-old woman who in 2006 was admitted to an intensive care unit in Réunion Island’s Centre Hospitalier Departmental for treatment of polyradiculoneuropathy. Her medical history consisted of poorly treated type 2 diabetes and hypertension. Three weeks before hospital admission, she had had fever, arthralgia, rash, and diarrhea. One week later, rapidly progressing motor weakness and sensory disturbances developed, e.g., tingling in all limbs. She had facial palsy, and her tendon reflexes were absent. Cerebrospinal fluid (CSF) contained increased protein (1.44 g/L) but not increased leukocytes (1/mm³). Electromyography displayed typical signs of demyelinating sensorimotor neuropathy with increased distal motor latency and reduced motor conduction velocity. Sensory nerve action potential was absent. Antichikungunya IgM was found in serum at 15 days after onset of signs and symptoms. This seroconversion confirms an acute infection by an alphavirus. Serum genomic product (RT-PCR, TaqMan method) (7) was negative for chikungunya virus. Anti-chikungunya IgM and IgG were also found in CSF.

The patient’s respiration rapidly deteriorated, and she required tracheal intubation and mechanical ventilation for 9 days. After receiving intravenous immunoglobulin for 5 days, she recovered quickly. Return of a productive cough and satisfactory muscle tone enabled her to be removed from mechanical ventilation on day 9.

For the 2 patients reported here, GBS diagnosis was based on a typical clinical acute motor and sensory polyradiculoneuropathy, which evolved in 3 characteristic stages: rapid deterioration, plateau, and slow recovery (6). Also typical of GBS are normal CSF counts, increased CSF proteins, and electromyography data (peripheral neuropathy, conduction block). The widespread screening for organisms known to be associated with GBS produced negative results. However, antichikungunya IgM was found in serum and CSF, although genomic products in serum and CSF were negative, which was not surprising, given the brief period (4–5 days) of viremia (8). These findings strongly supported a disseminated acute chikungunya infection and enabled us to conclude that chikungunya virus was probably responsible for the GBS.

Epidemiologic data also support a causal relationship between chikungunya infection and GBS. The incidence rate of GBS increased ≈22% in 2006 (26/787,000 [3.3/100,000] persons) over the rate in 2005 (21/775,000 [2.7/10,000] persons) and then declined to a rate closer to baseline in 2007 (23/800,000 [2.87/100,000] persons).

These 2 cases of GBS on Réunion Island were related to an acute and documented chikungunya infection. In the absence of an effective treatment, patients with these suspected infections should receive supportive care for classic GBS.
Gaëtan Lebrun, Karim Chadda, Anne-Hélène Reboux, Olivier Martinet, and Bernard-Alex Gaüzère
Author affiliation: Centre Hospitalier Felix Guyon, Saint-Denis, La Réunion, France

DOI: 10.3201/eid1503.071482

References


Address for correspondence: Gaëtan Lebrun, Hôpital Européen Georges Pompidou, 20 rue Leblanc, 75908 Paris CEDEX 15, France; email: gaetan.lebrun@egp.aphp.fr

LETTERS

Cockroaches (Ectobius vittiventris) in an Intensive Care Unit, Switzerland

To the Editor: Ectobius vittiventris (Costa) is a field-dwelling cockroach and 1 of 4,000 cockroach species worldwide (1). We describe a cockroach infestation of an intensive care unit (ICU). Successful management required knowledge of the ecology of cockroaches and highlighted the need for species-level identification to tailor control strategies.

The University of Geneva Hospitals are a 2,200-bed tertiary healthcare center. The 18-bed medical ICU is located on the ground floor next to an outdoor recreational area and admits ≈1,400 patients/year. Smoking inside hospital buildings by patients and healthcare workers (HCWs) is strictly prohibited. On August 25, 2006, ≈30 cockroaches were observed in the ICU hiding inside oxygen masks, moving around on the light panels below the ceilings, or dropping onto intubated patients during the night.

An outbreak investigation was initiated. All work areas, including sinks and material stock areas, were thoroughly searched for cockroaches. External pest control experts identified only 1 species, E. vittiventris, which had presumably entered the ICU through windows facing the outdoor recreational area. The investigation showed that despite verbal recommendations and being repeatedly forbidden to do so, HCWs had opened the windows secretly with screwdrivers so that they could smoke during night shifts. The infestation was halted within 3 days after information regarding the infestation was provided to HCWs and all windows were bolted shut. In contrast to measures required to deal with a reported infestation in a neonatal ICU (2), no other measures such as use of insecticides, review of the air circulation system, or changes in architectural structures were necessary to stop the infestation reported here.

Cockroaches can cause 2 potentially serious health problems. First, they may provoke allergic reactions (3). Second, they have been suggested as possible vectors of multidrug-resistant pathogens. In particular, cockroaches that live and breed in hospitals have higher bacterial loads than cockroaches in the community (4–6). Up to 98% of these “nosocomial” cockroaches may carry medically important microorganisms on their external surfaces or in their alimentary tracts (4–9) and may disseminate these microorganisms by fecal–oral transmission.

Cockroaches are capable of harboring Escherichia coli (6,7), Enterobacter spp. (6,8,9), Klebsiella spp. (6,7,9), Pseudomonas aeruginosa (6,9), Acinetobacter baumannii (2), other nonfermentative bacteria (7,9), Serratia marcescens (7,9), Shigella spp. (6), Staphylococcus aureus (6,7), group A streptococci (6,7,9), Enterococcus spp. (6,7), Bacillus spp. (7), various fungi (6–8), and parasites and their cysts (6). An outbreak of extended-spectrum β-lactamase–producing Klebsiella pneumoniae in a neonatal unit was attributed to cockroaches (2). Pulsed-field gel electrophoresis did not distinguish organisms from the insects or from those colonizing infants or causing clinical disease (2). Unlike other investigators, we did not cultivate the cockroaches (6,9).

E. vittiventris cockroaches are easily confused with Blattella germanica (Linnaeus) (the German or croton cockroach), which is probably the most important cockroach pest worldwide (1,9). In contrast to B. germanica (6,9) and other species (online Technical Appendix, available from www.cdc.gov/EID/content/15/3/496-