

Guillain-Barré Syndrome outbreak associated with Zika virus

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Summary

Background

Between October, 2013, and April, 2014, French Polynesia experienced the largest Zika virus outbreak ever described at that time. During the same period, an increase in Guillain-Barré syndrome was reported, suggesting a possible association between Zika virus and Guillain-Barré syndrome. We aimed to assess the role of Zika virus and dengue virus infection in developing Guillain-Barré syndrome.

Methods

In this case-control study, cases were patients with Guillain-Barré syndrome diagnosed at the Centre Hospitalier de Polynésie Française (Papeete, Tahiti, French Polynesia) during the outbreak period. Controls were age-matched, sex-matched, and residence-matched patients who presented at the hospital with a non-febrile illness (control group 1; n=98) and age-matched patients with acute Zika virus disease and no neurological symptoms (control group 2; n=70). Virological investigations included RT-PCR for Zika virus, and both microsphere immunofluorescent and seroneutralisation assays for Zika virus and dengue virus. Anti-glycolipid reactivity was studied in patients with Guillain-Barré syndrome using both ELISA and combinatorial microarrays.

Findings

42 patients were diagnosed with Guillain-Barré syndrome during the study period. 41 (98%) patients with Guillain-Barré syndrome had anti-Zika virus IgM or IgG, and all (100%) had neutralising antibodies against Zika virus compared with 54 (56%) of 98 in control group 1 ($p<0.0001$). 39 (93%) patients with Guillain-Barré syndrome had Zika virus IgM and 37 (88%) had experienced a transient illness in a median of 6 days (IQR 4–10) before the onset of neurological symptoms, suggesting recent Zika virus infection. Patients with Guillain-Barré syndrome had electrophysiological findings compatible with acute motor axonal neuropathy (AMAN) type, and had rapid evolution of disease (median duration of the installation and plateau phases was 6 [IQR 4–9] and 4 days [3–10], respectively). 12 (29%) patients required respiratory assistance. No patients died. Anti-glycolipid antibody activity was found in 13 (31%) patients, and notably against GA1 in eight (19%) patients, by ELISA and 19 (46%) of 41 by glycoarray at admission. The typical AMAN-associated anti-ganglioside antibodies were rarely present. Past dengue virus history did not differ significantly between patients with Guillain-Barré syndrome and those in the two control groups (95%, 89%, and 83%, respectively).

Interpretation

This is the first study providing evidence for Zika virus infection causing Guillain-Barré syndrome. Because Zika virus is spreading rapidly across the Americas, at risk countries need to prepare for adequate intensive care beds capacity to manage patients with Guillain-Barré syndrome.

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