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LETTER

Comment on: Zika virus and Guillain–Barré syndrome in Bangladesh

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Dear editor

We read with great interest the recent publication by Geurtsvan Kessel et al.[1] who, in a prospective study done between 2011 and 2015 in Bangladesh, sought to determine if the presence of ZIKV could be associated with GBS. After analyzing the presence of neutralizing antibodies in patients with GBS and healthy patients, it was concluded that there was no significant difference between cases and controls. Additionally, a subanalysis of two groups, patients with GBS and co-infection with ZIKV and *C. jejuni* and GBS associated with ZIKV alone was done. This analysis made it possible to observe that there are clinical outcomes and electromyographical differences between these two groups.

It is striking to find these results, due to the fact that during the different ZIKV outbreaks on Pacific islands and in Latin America, an increase in the incidence of GBS was observed parallel to it. It would be interesting to know if the health authorities of Bangladesh reported this phenomenon. On the other hand, although the association in the study was not found, GBS is an autoimmune disease, and its occurrence depends on multiple environmental and genetic determinants, which could influence its appearance. These unknown factors could bestow protection or risk factors aside from age and consanguinity.[2]

The presence of co-infections, on one hand, could influence the risk of developing GBS and, on the other, its clinical course. The presence of *C. jejuni* is known to trigger GBS and can be associated with greater disability and higher mortality; however, in this study, the co-infection with ZIKV did not worsen the clinical outcomes of the patients. Moreover, we observed that the presence of previous infection by *M. pneumoniae* was one of the main risk factors related to GBS associated with ZIKV in a Colombian population.[3] This could be due to a molecular mimicry between ZIKV antigens and host tissues bolstered by a previous infection.

Attention is drawn to the clinical differences observed between patients with GBS associated with ZIKV and GBS associated with a co-infection between ZIKV and *C. jejuni*. Firstly, like other reports, the association between GBS-ZIKV and a greater presentation of acute inflammatory demyelinating polyneuropathy, which, unlike the study in Polynesia, is comparable with other studies in Latin America. Finally, the high prevalence of autonomic symptoms reported in this study should be highlighted. In fact, we reported a possible association between autonomic symptoms and ZIKV without overt neurological complications.[4]

Conflict of Interest

The authors have no conflicts to declare

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