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Letter to the Editor

# Concurrent Guillain-Barré syndrome, transverse myelitis and encephalitis post-Zika: A case report and review of the pathogenic role of multiple arboviral immunity

We thank Yasri and Wiwanitkit for their comments on our review article of Zika virus (ZIKV) neurotropism [1,2]. They indicate that in their geographic region in South East Asia (Thailand) —a ZIKV endemic area— the concurrent immunoreactivity to ZIKV and other arboviruses is very common, particularly with dengue virus (DENV) and Chikungunya virus (CHIKV) [3], but there are no reports of neurological complications resulting from co-reactivity between ZIKV and other arboviruses.

In fact, as of June 2018, only 4 confirmed cases of congenital ZIKV infection with microcephaly have been reported in South East Asia (3 Thailand, 1 Vietnam) [4], but in only one case the Asian lineage was confirmed. Wongsurawat et al. [4] demonstrated that the ZIKV circulating in Asia could cause microcephaly, just like the American strains, particularly when the mother is infected before week 21 of gestation or in the first trimester. Regarding ZIKV-associated Guillain-Barré syndrome (GBS), by August 2017 the number of patients with ZIKV infection in Thailand reached 1417 with only 2 reported patients with GBS (0.14%) [5].

Therefore, the number of neurological complications appears to be minimal despite the fact that ZIKV has been circulating in South East Asia longer than in other parts of the tropical world. This is in clear contrast with the epidemic of ZIKV-associated microcephaly and GBS observed in French Polynesia, Brazil, Colombia and the Caribbean. Moreover, as summarized in Table 2 of our review [2], a number of ZIKV-associated central nervous system (CNS) complications were reported from Colombia (8 cases), Brazil (6 cases), Dominican Republic (3 cases) and 1 case each from Puerto Rico and Guadeloupe (French Antilles). No such CNS complications have been reported in Thailand suggesting that the ZIKV of Asian lineage is less neurotropic than the American lineage.

Surveillance is complicated by a problem of correct diagnosis that arises when attempting to determine the viral load given the cross-reactivity of DENV and ZIKV [6]. In Colombia, a case-control study by Anaya et al. [7] compared 29 patients with ZIKV-GBS and 74 matched-controls with ZIKV infection alone and found that all GBS patients were positive for IgG antibodies against both ZIKV and DENV, and 69% were positive also for CHIKV [7]. Previous infection with *Mycoplasma pneumoniae* increased 4-fold the risk of GBS (OR: 3.95; 95% CI = 1.44–13.01; $p$ = .006). No differences in antibody levels against *Campylobacter jejuni,* Epstein B virus (EBV) and Cytomegalovirus (CMV) were observed. Unfortunately, similar case-control studies have not been performed elsewhere. Finally, the possibility of "original antigenic sin" [8] could be invoked to explain some of the ZIKV-associated neurological complications. Methods such virus-specific PCR for accurate diagnosis plus geographical information systems (GIS) [9] should provide information on the true prevalence of ZIKV-associated neurological complications in South East Asia.

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