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LETTER TO THE EDITOR

Neuroimaging findings of Zika virus infection: emphasis of *congenital* versus *acquired* aspects

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Dear Editor,

We appreciate the interest on our article by Prof. Viroj Wiwanitkit [1]. We agree with Dr. Wiwanitkit that *acquired* Zika virus (ZIKV) infection is asymptomatic in up to 80% of the affected people, and it is estimated that only 20% of infected cases are symptomatic. Moreover, ZIKV causes a self-limited disease with fever, myalgia, arthralgia, maculopapular rash, and conjunctivitis in the majority of symptomatic adult patients. Our review article, however, focused mainly on neuroimaging findings of *congenital* ZIKV infection along with two autoimmune conditions that may complicate the acquired ZIKV infection [2].

The outbreak of Zika virus infection in Brazil in 2015 was associated with an increase in the cases of congenital microcephaly by a factor of 20. The suspected causal relationship between prenatal ZIKV infection and microcephaly has now been confirmed [3]. It is estimated that about 1–13% of pregnant women infected in the first trimester will have a child with microcephaly and other cerebral abnormalities [4]. These facts suggest that *congenital* ZIKV infection, which is vertically transmitted to the fetus from the infected mother, is much less benign than *acquired* infection. This issue has made congenital ZIKV infection a "Public Health Emergency of International Concern" (World Health Organization declaration, 1 February 2016).

We agree with Prof. Wiwanitkit that neuroimaging findings are most likely normal in the majority of patients with *acquired* Zika infection. There are a few exceptions, including adult patients complicated by ZIKV-related Guillain-Barré syndrome (GBS), which may be associated with post-contrast enhancement of the cranial nerves, conus medullaris, cauda equina nerve roots, and lumbar spinal ganglia on magnetic resonance imaging (MRI) as well as adult patients with ZIKV-related acute disseminated encephalomyelitis (ADEM) exerting brain and spinal cord changes on MRI study. On the other hand, the majority of fetuses/neonates with *congenital* Zika infection have abnormal neuroimaging findings including microcephaly, intracranial calcifications, malformations of cortical development (mostly polymicrogyria), cerebellar and brainstem hypoplasia, ventriculomegaly, agenesis/hypoplasia of the corpus callosum, absent cavum septum pellucidum, white matter changes most likely due to dysmyelination, and ocular abnormalities. These findings may be detected in the prenatal or neonatal imaging studies [2, 3, 5].

In conclusion, when discussing neuroimaging findings in Zika virus infection, it is extremely important to differentiate between *congenital* and *acquired* infection. Our review article focused mainly on congenital ZIKV infection, which is associated with abnormal neuroimaging findings

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in the majority of affected cases. It is crucial for radiologists and perinatologists to have a detailed knowledge of these imaging findings to make a correct diagnosis particularly in the prenatal screening of pregnant women residing in the endemic regions or those who have traveled to these areas. In addition, autoimmune complications (such as GBS and ADEM), although not frequently reported in the infected adults, should be investigated in any suspected or confirmed case of acquired ZIKV infection with severe neurological symptoms.

Compliance with ethical standards

Conflict of interest There is no conflict of interest.

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