

Zika Virus Infection Complicated by Guillain-Barré Syndrome: What Really is True?

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Guillain-Barré syndrome (GBS) is clinically defined as an acute peripheral neuropathy causing limb weakness that progresses over a time period of days or, at most, up to several weeks [1]. Increased GBS incidence has been reported in countries with local transmission of Zika virus (ZIKV), a flavivirus transmitted primarily by certain *Aedes* species mosquitoes. The current epidemic outbreak due to ZIKV began in 2015 and since then it has been reported in 31 countries and territories in America.

In view of the above, it is necessary to identify enough evidence for its causality. PubMed manuscripts reporting original clinical, laboratory, and electrodiagnostic data on ZIKV and GBS present controversial results. Most studies have robust evidence for such an association, but many others lack a more qualified methodology. "We may not believe that any type of motor paralysis post-infection by ZIKV, is considered a GBS case". The differential diagnosis includes infection by the Dengue virus, Chikungunya fever, respiratory and gastrointestinal infections, immunizations and other diseases.

We should always characterize the nature of a polyneuropathy and exclude others neuropathies, such as plexopathy, neuronopathy, or multiple mononeuropathies. In view of this, all "possible" cases of GBS should be rigorously assessed by thorough neurological, electrophysiological and serological examination. Unfortunately, many studies do not present this basic screening for diagnostic confirmation. The distinction between GBS cases and other flaccid paralysis has serious financial and social implications, which may not only burden health care expenditures, but also lead to despair among the population.

We know the existence of "Mimics and chameleons, GBS and Miller Fisher syndromes". Countries affected by the ZIKV should be prepared for immediate care of "confirmed GBS cases" with intravenous human immunoglobulin. Until pathogenesis is established, new cases of ZIKV related weakness be termed "Zika virus acute flaccid paralysis," rather than attributed to variants of GBS [2].

World Health Organization (WHO) stated in 2016 that the mosquito-borne Zika virus was a cause of the neurological disorders, such as Guillain-Barré syndrome (GBS) [3]. In Brazil, a growing number of severe neurological complications among adults is related to the spread of ZIKV, according to some researches, was observed an increase in admissions of patients with inflammatory complications such as GBS, myelitis and encephalopathy.

Future studies should find out how this ZIKV neurotropism occurs and increases awareness of its neurological complications. On the surface, these neurological pathologies appear to be new features of ZIKV infection, however, its causality has not yet been established. Severe neurological diseases associated with ZIKV may, therefore, not be a new feature for the virus, but may have been ignored due to

previous outbreaks [4]. The ZIKV epidemic in the Americas is a serious situation and decisions based on solid scientific evidence - not hyped media speculations - are required for effective outbreak response.

Undoubtedly, the number of GBS cases increased substantially in the year 2016 [5]. However, we may not forget the climatic conditions of the population, especially mosquitoes that carry other types of virus as Yellow fever, Dengue virus, Chikungunya fever. All these factors must be taken into account. Compared with a similar seasonal period prior to the first documented ZIKV case, hospital admissions for GBS in the period from 2015 to 2016 showed significant increases of 1.0 to 5.6 cases per month [5].

Thus, we do not know if all patients diagnosed with GBS had positive serology for ZIKV; cerebrospinal fluid, electroneuromyography and if they have undergone a thorough neurological examination.

The GBS has been reported in many articles as a possible complication of acute Chikungunya and Dengue infection [6,7]. A systematic review identified the occurrence of auto-inflammatory diseases related to 17D vaccine administration of Yellow fever. Six studies were identified describing 13 possible cases. The diseases were GBS, multiple sclerosis, multiple points evanescent syndrome, acute disseminated encephalomyelitis, autoimmune hepatitis, and Kawasaki disease. The data suggest that 17D vaccination may play a role in the mechanism of self-tolerance loss [8].

It is very important to note that all these viruses were circulating during the GBS epidemic; accordingly, only a certainty diagnosis, composed of physical and complementary examination would be able to conclude GBS and ZIKV cases - many studies did not follow this methodology. Unfortunately, the vast majority of our health services are not fully prepared for a correct certainty diagnosis facility, so it is important not to confuse GBS with other types of neuropathies, considering them as a single entity. It would also be negligent to oppose the data presented in the current literature.

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