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Dengue or Japanese Encephalitis - An Emerging Diagnostic Dilemma for Tropical World Clinicians

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Editorial

Viruses are notorious culprits causing encephalitis globally, even more notorious than bacteria, fungi or protozoa. In spite of upgrading the diagnostic armamentarium with newer PCR techniques, in several cases especially those from tropical Africa, Asia and Latin America due to co-existence of closely related viruses and immunological cross reactivity among their genomic products during tests, often a definitive diagnosis of the specific virus causing the neurological catastrophe cannot be made [1].

Japanese encephalitis (JE) is a known virus belonging to *flaviviridae* family causing epidemics of encephalitis in several parts of the world like south eastern Asia. To prevent mortality as well as the high incidence of post-encephalitis neurosequelae, several countries (like India) have started dedicated programs for routine immunizations against JE in affected regions.

Dengue virus is another single-stranded RNA virus belonging to family *flaviviridae*. Previously neurological features during dengue fever or dengue hemorrhagic fever were thought to be due to the combined effects of coagulopathy, hemoconcentration and vascular leakiness, cerebral edema, hemorrhage, toxic cytokine storm etc. [2]. Dengue virus, for a long time, has been considered a non-neurotropic one [3]. Even WHO in their 2009 guidelines [4] has proposed to include all the cases of dengue fever with neurological features under the classification of "severe dengue", without differentiating dengue encephalopathy and dengue encephalitis.

Since the beginning of this millennia, several case reports, series and even studies started to accumulate which have suggested that dengue has the properties of a neurotropic virus directly causing neuroinvasion and resulting in viral encephalitis [2,5,6]. Presence of viral antigens and antibodies in the cerebrospinal fluid and involvement of brain parenchyma in magnetic resonance imaging (MRI) have strengthened this hypothesis [5,7-10].

JE has the classical feature of involvement of bilateral deeply situated grey matter of the brain like basal ganglia, thalamus etc. on magnetic resonance imaging (MRI) and for long this pattern of involvement was accepted to be the signature of this disease.

In 2011, Borawake et al. reported a case of viral encephalitis where MRI showed bilateral involvement of thalami just like JE but the CSF study came out to be positive for dengue IgM [10]. Liyanage et al. reported similar 2 cases in 2016 where diagnosed dengue cases showed radiological features of brain similar to JE [11]. There is an extensive retrospective study by Singh et al. where 129 out of 1410 patients with acute encephalitis syndrome and thrombocytopenia showed positive results for both JE and dengue IgM. When PCR was done to detect viral RNA 8 patients showed positivity to both (2 having both serum and CSF positive, 6 positive results in serum only). Highest dual positivity was noticed in the month of September when both the diseases are nearly at their peak in this geographical region [12]. Similar cases have been reported from children as well as adult populations.

A-nuegoonpipat et al. in their study found that 13% serum samples and 11% CSF samples from JE patients were positive for anti-dengue IgM and 9% of serum samples from dengue patients were positive for anti JE IgM [13]. So only doing antibody study is not enough as per this study due to the reason that both are from same family of virus and antibody responses have cross-reactivity. Ideally a PCR study using both serum and CSF to find out viral RNA may be more definitive in diagnosing the virus.

There is dire need of establishing the clear cut discrimination between dengue encephalopathy and encephalitis and also between dengue encephalitis and JE. Further researches in the fields of immunology and infectious disease may hold the answer. Till then clinicians of the tropical regions should treat the cases cautiously keeping both the etiologies in mind and not relying solely on MRI findings or serological studies as definitive proof of one form of encephalitis or the other. That is indeed a tough clinical dilemma as one disease has higher chances of mortality with no long term sequelae and the other, a high chance of lifelong morbidity.

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