

## Commentary

Acute disseminated encephalomyelitis (ADEM) is an acute demyelinating disorder of the central nervous system (CNS).<sup>[1]</sup> It usually follows an antecedent infection or vaccination. A variety of microorganisms have been implicated as the causative agents. Among them, viruses are predominantly reported, including measles, mumps, rubella, varicella, herpes simplex, influenza A or B, hepatitis A or B, Epstein-Barr, cytomegalovirus, enterovirus, rotavirus and human immunodeficiency virus. Other agents include mycoplasmas, bacteria, rickettsiae and protozoans. These agents usually affect the hosts in an interval of two to 30 days before the development of widespread neurological disorders.

Among the diagnostic tools, neuroimaging is the most valuable in establishing the diagnosis of ADEM. Demyelinating lesions in the CNS are better recognized by MRI. These patchy lesions appeared on T2-weighted images and fluid attenuated inversion recovery sequence (FLAIR), usually exist throughout the white matters of the cerebrum, as well as cerebellum and brain stem. Deep-seated nuclei such as thalamus and basal ganglia may be also involved. Even in cortical areas, gray matter structures are not entirely spared. In Marchioni's series,<sup>[2]</sup> up to 60% of post-infectious inflammatory disorders in the CNS showed gray matter involvement in supratentorial regions.

In a previous study<sup>[3]</sup> of the etiologies of 50 pediatric ADEM cases during the period 1980 to 1997 in one medical center of northern Taiwan, the causative agents had shifted from common childhood infections (measles, mumps, rubella and chickenpox) to nonspecific respiratory tract infections or unknown origins, due to availability of vaccines. None did suffer from dengue infection, in that non-endemic area.

Though neurological complications of dengue infection are not uncommon,<sup>[4]</sup> ADEM following dengue fever have been rarely reported in the literature.<sup>[5-7]</sup>

Yamamoto *et al.*,<sup>[5]</sup> probably reported the first case of ADEM following dengue fever in the literature. Both the cases of Sundaram<sup>[6]</sup> and Gera<sup>[7]</sup> showed pathognomonic changes of demyelinating lesions with hemorrhagic foci in MRI, corresponding with thrombocytopenia clinically.

In this issue, Karoli *et al.*,<sup>[8]</sup> reported an interesting case "was it a case of acute disseminated encephalomyelitis? A rare association following the dengue fever". The possibility of dengue encephalitis in this case might be excluded from the biphasic pattern of clinical course three weeks apart. Some MRI lesions in this case are actually located in superficial gray matters. Though MRI lesions are not classical for ADEM, yet the fact of preceding dengue infection and quick response to pulse steroid therapy could support its postinfectious inflammatory nature. The MRI of this case did not show hemorrhagic lesions. Further investigations are needed to find out the characteristic MRI pictures of ADEM associated with dengue fever. Nevertheless, this article tells us that clinical awareness and appropriate MRI study in endemic areas of dengue fever may help to identify ADEM patients from those suffering from encephalopathy following dengue infection.

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