Anti-GQ1b antibody syndrome in Korea

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New data and information in the field of stroke have been reported last year. Immediate and acute blood pressure lowering was attempted for acute management of ischemic and hemorrhagic stroke. In terms of the medications, new oral anticoagulant, edoxaban, for the primary and secondary prevention in non-valvular atrial fibrillation was reported and dual antiplatelet therapy in acute stage of ischemic stroke has accumulated the evidence for clinical use. In this review, all the results and information about primary prevention, acute management and secondary management will be discussed in the order named.

Key Words: Stroke, Prevention, Acute management

After discovering immunoglobulin G (IgG) anti-GQ1b antibodies in Fisher syndrome (FS) patients in 1992, serologically positivity for the IgG anti-GQ1b antibodies was a common finding in a series of clinical spectrum such as Miller Fisher syndrome (MFS), Guillain-Barre syndrome (GBS) with ophthalmoplegia, Bickerstaff brainstem encephalitis (BBE), and acute ophthalmoplegia without ataxia (AO). From 83% to 99% cases of typical MFS, atypical MFS, and GBS with ophthalmoplegia and 68% of cases of BBE show abnormal elevations in the anti-GQ1b antibody acutely. More recently, this phenomenon led to the clinical spectrum that is referred to as the 'Fisher–Bickerstaff syndrome'. Anyhow, a more inclusive nomenclature such as 'anti-GQ1b antibody syndrome' could be used to include the common serological profile when referring to the clinical syndromes as we described before.

In Korea, there have been several nation-wide clinical reports regarding anti-GQ1b antibody syndrome. The one report in 2008 analyzed retrospectively 34 patients diagnosed with anti-GQ1b antibody syndrome focusing on ophthalmoplegia. 31 patients had ophthalmoplegia and they were classified into MFS (n = 13), AO (n = 11), GBS with ophthalmoplegia (n = 6), and BBE (n = 1). In AO patients (32.4%, 11/34), external ophthalmoparesis was present in all the patients and included mixed horizontal-vertical (n = 7), pure horizontal (n = 3), and pure vertical gaze palsy (n = 1). Binocular involvement was common, but unilateral ophthalmoparesis was also observed in 27.3%. Other findings included ptosis (n = 5, 45.5%) and internal ophthalmoplegia (n = 6, 54.5%). Other anti-GQ1b antibody syndromes other than AO had prominent neurologic signs including ataxia, weakness, and facial palsy in addition to ophthalmoplegia. The patterns of neuro-ophthalmologic findings did not differ between AO and other anti-GQ1b antibody syndromes with ophthalmoplegia.

Another nation-wide study, even though it was not focusing on anti-GQ1b antibody syndrome, analyzed acute phase of GBS with IgG or IgM antibodies against any type of ganglioside. IgG anti-GQ1b antibody was positive in 10 of total 60 patients with acute GBS, and was always associated with anti-GT1a antibody. Ophthalmoplegia was strongly correlated with coexisting IgG anti-GQ1b antibody positivity. However, four of the ten IgG anti-GQ1b

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antibody-positive cases had no oculomotor palsy during the disease course.

It still remains to be elucidated why diverse phenotypes are caused within anti-GQ1b antibody syndrome. One explanation may be that different effects of the patient's serum on the blood–brain barrier (BBB) distinguish the PNS and CNS phenotypes. Another explanation is the diverse fine specificities of anti-GQ1b antibodies. Further assessment of anti-GQ1b antibodies in the CNS disorders should be done in future.