**CASE REPORT**

Guillain-Barré syndrome associated with central nervous system lesions

A Okumura, H Ushida, K Maruyama, K Itomi, Y Ishiguro, M Takahashi, A Osuga, T Negoro, K Watanabe

We report the clinical course, and neurophysiological and neuroimaging findings of a patient with Guillain-Barré syndrome associated with central nervous system lesions. During a course of intravenous immunoglobulin therapy, she had headache with meningism. Cerebral magnetic resonance imaging showed lesions in both frontal and right occipital lobes. Cerebrospinal fluid showed a raised protein concentration accompanied by mild pleocytosis. Her symptoms resolved within two months. Subsequent magnetic resonance imaging revealed cavity formation in the deep white matter and atrophic changes in the right occipital lobes.

Guillain-Barré syndrome (GBS) is an acute demyelinating polyneuropathy presumably related to immunological mechanisms. The central nervous system (CNS) is usually intact in patients with GBS. However, there have been some reports of an association of GBS with CNS involvement.

**LITERATURE REVIEW**

Guillain-Barré syndrome associated with central nervous system lesions has been reported in children and adults. Gamstorp reported an 8-year-old girl with GBS by with unconsciousness, oscillating eye movement, and convulsions, and proposed the term GBS associated with CNS manifestations has been described in children, as well as adults. The syndrome can be accompanied by meningism, headache, and ocular motility disorders.

**CONCLUSION**

GBS is regarded as a predominantly motor neuropathy with few sensory features. Although the CNS is rarely involved, GBS associated with CNS manifestations has been described in children, as well as adults. Gamstorp reported an 8-year-old girl with GBS by with unconsciousness, oscillating eye movement, and convulsions, and proposed the term...
“encephalomyeloradiculoneuropathy”. Amit et al described a 10 year old girl with GBS associated with deep coma. Contrast enhanced CT displayed multifocal enhancement of the white matter. In our patient, MRI showed multiple CNS lesions, not only in the periventricular white matter, but also in the occipital cortex and subcortex. It is interesting that CNS manifestations were not evident in our patient, although the previously reported patients always had CNS symptoms, such as reduced consciousness, seizure, or brain stem impairment. This implies that an association of CNS involvement in patients with GBS could be underestimated because some lesions can be clinically silent.

Mild pleocytosis was observed in the second lumbar tap in our patient. We consider this pleocytosis attributable to intravenous immunoglobulin. Aseptic meningitis is a common complication of intravenous immunoglobulin therapy. We consider her headache with meningitis was attributable to intravenous immunoglobulin, although these symptoms can be a clinical manifestation of GBS itself. Another explanation is that pleocytosis may be related to CNS lesions. CSF pleocytosis is often seen in patients with demyelinating CNS disorders such as multiple sclerosis.

There are some possible explanations for the pathogenesis of CNS lesions in our patient, including watershed infarction, demyelination, reversible posterior leuoencephalopathy, and adverse effects of immunoglobulin. With regard to watershed infarction, there was no clinical event that could cause ischemic brain damage. Given that extensive CNS lesions seen in our patient were watershed infarction, it is not likely to be clinically silent. Raised CSF myelin basic protein indicated the demyelinating nature of CNS lesions in our patient. Some authors have discussed the possibility of shared pathogenic central and peripheral nervous system epitope. Several animal studies reported peripheral nervous system lesions in experimental allergic encephalitis, as well as central nervous injury induced by peripheral nerve antigen.

Reversible posterior leuoencephalopathy may be another explanation for CNS lesions in our patient. Reversible posterior encephalopathy is often associated with a condition in which blood pressure rises acutely. Patients with this syndrome often have seizures, consciousness loss, or visual disturbance. On the other hand, our patient lacked apparent hypertension or CNS symptoms, and MRI lesions were asymmetric. Intravenous immunoglobulin itself could have been a cause of CNS lesions in our patient. There have been previous reports of encephalopathy associated with intravenous immunoglobulin treatment in patients with GBS. Although hyperviscosity or vasospasm have been suggested to be related to the development of encephalopathy in those patients, its pathogenesis has not been clarified.

In summary, we report a patient with GBS associated clinically silent CNS lesions. Such cases have been reported rarely, but our experience suggests that the association of CNS lesions with GBS may be underestimated.

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