

Guillain-Barré Syndrome Associated with Central Nervous System Lesions – A Case Report

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Abstract: Guillain-Barré syndrome (GBS) is defined as heterogeneous group of autoimmune polyradiculopathies, involving sensory, motor and autonomic nerves. It is the most common cause of rapidly progressive flaccid paralysis. Radiological studies are ordered to exclude other causes and in cases where nerve conduction studies and CSF examination are equivocal. In the brain the facial nerve (CN VII) is the most commonly affected. Brain parenchyma findings in MRI are usually not encountered. We present a case of Guillain Barre Syndrome with incidental findings on MRI brain.

Keywords: Guillain Barre Syndrome, AIDP, MRI brain

1. Introduction

The Guillain-Barré syndrome, which is characterized by acute areflexic paralysis with albuminocytologic dissociation (i.e., high levels of protein in the cerebrospinal fluid and normal cell counts), was described in 1916. It is usually diagnosed on the basis of clinical history, CSF findings, nerve conduction studies, and if required, MRI of the spine, which usually shows gadolinium enhancement of the cauda equina roots. The diagnosis of GBS is straightforward, and rarely is the MRI of the brain ordered or required. 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.¹⁻⁷ We report the clinical course, and neurophysiological and neuroimaging findings of a patient with GBS associated with distinctive CNS lesions.

2. Case Report

A 28 year old male presented to our outdoor patient department (OPD) with complaint of progressive ascending paralysis since 5 days. It started as heaviness of both legs, as evidenced by difficulty in moving the limbs, progressing to difficulty in moving limbs while walking over a period of 5 days. He did not have any urinary complaints, any history of diarrhoea, any sensory disturbances, any history of trauma, nor any history of fever. On examination, all deep tendon reflexes were absent, with intact sensory system. He was conscious, but could not get up and move his legs without support. His vitals were normal, and all routine investigations normal. He was admitted to our hospital for further management. On the 3rd day of admission, he developed weakness of grip, and was unable to grasp objects with his hands tightly. An MRI of the brain, a routine CSF examination, and nerve conduction studies were ordered. MRI Brain showed the picture given below, with T2 weighted image (lower) showing high intensity areas in the deep white matter in the bilateral frontal lobes, and in the cortex and subcortex in the right occipital lobe. T1 weighted

image (upper) showed low intensity areas in the corresponding place.

The CSF examination revealed a protein level of 110 mg/dL, and a cell count of 50/mm³. The nerve conduction study revealed the picture of mixed axonal and sensory type of demyelinating neuropathy. A neurology consult was taken, and the patient was diagnosed as Guillain Barre Syndrome. He was kept under observation, and physiotherapy and rehabilitation were started. We initially planned to start IVIg therapy for the patient, however, due to financial problems, the same could not be done. 3 weeks post admission, the patient showed symptomatic improvement, and was able to walk without support, with a normal grip. The patient was then discharged, and continued to be asymptomatic on follow up.

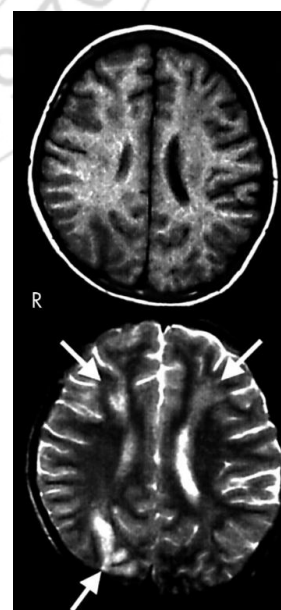


Figure 1: T2 weighted image (lower) showed high intensity areas in the deep white matter in the bilateral frontal lobes, and in the cortex and subcortex in the right occipital lobe (arrows).

3. Discussion

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,^{1,2,4} as well as adults.^{3,5-7} Gamstorp reported an 8 year old girl with GBS by with unconsciousness, oscillating eye movement, and convulsions, and proposed the term "encephalomyeloneuropathy".¹ Amit et al described a 10 year old girl with GBS associated with deep coma.⁴ 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.^{1,4-7} This implies that an association of CNS involvement in patients with GBS could be underestimated because some lesions can be clinically silent.

There are some possible explanations for the pathogenesis of CNS lesions in our patient, including watershed infarction, demyelination, and reversible posterior leucoencephalopathy. With regard to watershed infarction, there was no clinical event that could cause ischaemic 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.^{3,6} Several animal studies reported peripheral nervous system lesions in experimental allergic encephalitis,^{9,10} as well as central nervous injury induced by peripheral nerve antigen.^{11,12} Reversible posterior leucoencephalopathy 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.^{13,14} 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. There have been previous reports of encephalopathy associated with intravenous immunoglobulin treatment in patients with GBS, although our patient did not receive the same.^{7,15-17} 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. A detailed evaluation of the patient, with more frequent neuroimaging may help find a correlation between GBS and positive neuroimaging findings. The suspicion and confirmation of GBS must always be clinical, with neurological imaging only to help it, not to stamp it. All neuroimaging findings must be correlated clinically for a more accurate diagnosis.

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