Dear Editor,

We report a 15-year-old girl who presented to us with a history of progressive weakness of both upper and lower limbs for 2 days before. On examination, the power of lower and upper limbs were 2/5 and 4/5, respectively. She had decreased tone and areflexic quadriparesis with mute plantar. The patient was clinically diagnosed to have Guillain-Barre syndrome (GBS). Nerve conduction studies confirmed GBS with both axonal and demyelinating polyradiculoneuropathy. Cerebrospinal fluid (CSF) analysis showed albuminocytological dissociations. Over the next 2 days, her weakness rapidly progressed to respiratory failure, and she was intubated and ventilated and was closely monitored. Her blood pressure (BP) fluctuated between 190/130 mmHg to 140/90 mmHg and labetalol was used intermittently to reduce BP. She was started on IVIG 20 gms/day for 5 days; however, on day 14 of admission she had 2 episodes of generalized seizures and was treated with antiepileptics. She was weaned from ventilator after 4 weeks. Although her muscle power improved, she complained of light perception vision loss in both eyes. In the clinical evaluation, her direct and indirect pupillary reflexes, fundus examination using direct ophthalmoscopy and extraocular movements were normal. Magnetic resonance imaging (MRI) of the brain revealed bilateral parieto-occipital T2/FLAIR hyperintensity and diagnosis of posterior reversible encephalopathy syndrome (PRES) was made [Figure 1a and b]. The patient gradually improved over the next month to regain complete vision and most of the muscle power with conservative treatment.

Vision loss in GBS is rarely reported in the literature. Association of papilledema with GBS is well known. It is usually asymptomatic, but can rarely induce field defects and complete visual loss due to secondary optic atrophy comparable to idiopathic intracranial hypertension. These patients have elevated CSF proteins which were postulated to cause a defect in the proper absorption of CSF at the arachnoid villi, giving rise to raised intracranial pressure and even rarely hydrocephalus.[1] Lolekha and Phanthumchinda reported a case of Miller-Fisher syndrome (MFS) with vision loss due to optic neuritis occurring as a CNS manifestation. [2] Güngör et al in a study of visual evoked potentials in GBS concluded that out of 32 cases with GBS, 16% had P100 prolongation further suggesting the involvement of CNS in GBS and its variants.[3]

Cortical blindness in GBS is a very rare manifestation and may occur due to PRES or hypoxic ischaemic encephalopathy. In our case, there was no documented hypotension to suspect hypoxia and imaging features were consistent with PRES. PRES is a clinico-radiologic entity described first by Hinchey et al in 1996.[4]

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Figure 1. (a and b) Magnetic resonance imaging brain fluid attenuation inversion recovery shows hyperintense signal changes in bilateral parieto-occipital areas.
Recognition of PRES has increased over the years along with increasing availability of MRI. PRES is typically reversible once the cause is removed. In GBS with dysautonomia, an acute increase in BP causes the release of pro-inflammatory cytokines to break the blood-brain barrier and cause vasogenic edema. PRES can occur concurrently with weakness or during the course of GBS. In Indian literature, we found only one case report which presented both GBS and PRES.[5]

In conclusion, vision loss in GBS is an uncommon complication. GBS with dysautonomia can be considered as an independent risk factor for PRES.

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Conflicts of Interest
There are no conflicts of interest.

REFERENCES


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