Commentary

Guillain-Barré syndrome (GBS) is an acute postinfectious polyradiculoneuropathy with an incidence of 0.6-4.0/100,000 person/year worldwide.^[1] In its classical form is demyelinating in nature, presented with rapidly progressive generalized weakness, minor sensory deficits, and hyporeflexia or areflexia. In addition, elevated protein level in the cerebrospinal fluid and abnormalities of conduction velocity in the electrophysiology testing after the first week of illness are ancillary laboratory findings that greatly contribute to the diagnosis of GBS.^[2] Respiratory muscles weakness and autonomic nervous system involvement are serious manifestations, which if occurred, should be promptly addressed. Treatment with intravenous immunoglobulin (04 g/kg per day for 5 days) or plasma exchange sessions at the early stages, management of cardiac arrhythmias, and support of respiratory function in intensive care unit have lowered the permanent disability to less than 20% and mortality to 5% of all cases.^[3,4]

According to the prevailing view, GBS is due to a transient autoimmune response induced by an exogenous trigger. Indeed, antecedent upper respiratory tract infection or gastroenteritis as precipitating events are documented in two-thirds of GBS cases, albeit the responsible microorganism is rarely identified.^[2] The most frequently encountered infectious agents are campylobacter jejuni, cytomegalovirus, Epstein-Barr virus, mycoplasma pneumonia, borrelia, *Haemophilus influenzae*.^[5] One might argue that no investigation for the responsible agent in patients with GBS is required, since irrespective to the prior infection, management depends on clinical severity of GBS. However, GBS variants and therefore, prognosis may indeed depend on the causative agent. For example, campylobacter jejuni gastroenteritis has been associated with a specific type of GBS known as acute motor axonal neuropathy (AMAN), which has worse prognosis than the classical demyelinating type.^[1] Several reports have linked viral hepatitis with the development of GBS. Hepatitis A, B, C, D, and E viruses have been implicated in single GBS cases.^[6] Specifically, GBS associated with hepatitis A virus (HAV) appeared to be of the classical demyelinating type, but as described in case report presented in this issue,^[7] less common variants such as acute motor and sensory axonal neuropathy (AMSAN) may occur.

Awareness of a potential association between HAV infection and GBS may prove useful for medical doctors,

who are not accustomed to neurological diseases. The diagnosis of GBS can be challenging for these doctors who treat patients with acute hepatitis, since neuromuscular manifestations like myalgia and fatigue are symptoms of hepatitis itself. Taking into account that hepatitis A occurs in tens of millions of persons per year worldwide,^[8] its potential complications, even the rare ones, are clinically significant and even more so conditions like GBS, which is potentially treatable.

Elisabeth Chroni

Department of Neurology, School of Medicine, University of Patras,
Rio-Patras 26504, Greece

Address for correspondence:

Prof. Elisabeth Chroni,
Department of Neurology, School of Medicine,
University of Patras, Rio-Patras 26504, Greece.
E-mail: echroni@yahoo.com

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