

SHORT REPORT

## Acute Motor Axonal Neuropathy Developing During *Helicobacter pylori* Infection

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Guillain-Barré Syndrome (GBS) is an acute demyelinating neuropathy of immunological origin caused by antibodies formed against nerve antigens. GBS has an acute onset and its incidence in children under 15 is 0.8/100,000 (1,2). GBS was classified in recent neurophysiologic studies as acute inflammatory demyelinating polyradiculopathy (AIDP), acute motor axonal neuropathy (AMAN) and Fisher syndrome (3).

Most GBS patients may have a history of bacterial or viral infection in the previous few weeks. *Campylobacter jejuni*, Cytomegalovirus, Epstein-Barr virus and *Mycoplasma pneumoniae* are the agents most commonly held responsible for this syndrome (3-6). *Helicobacter pylori* is the most frequently encountered cause of gastroduodenal diseases. Mucosal damage is generally attributed to antigenic similarity between *H. pylori* and the host (7). Antibodies formed against *H. pylori* can also be identified in extragastric tissues (8,9). A putative pathogenetic role has been ascribed to *H. pylori* in several extradigestive neurological disorders, including Parkinson's disease, anterior optic ischemic neuropathy, primary headache and sudden infant death syndrome (10). A relation was established between *H. pylori* infection and GBS, especially the axonal form of it, and a high rate of antibodies formed against *H. pylori* was found in the cerebrospinal fluids of these patients (11).

This paper presents a 14-year-old GBS patient who had had gastrointestinal complaints for a year and whose serum and cerebrospinal fluid (CSF) samples contained a

high concentration of IgG antibodies against *H. pylori*. Thus, the aim of this paper is to emphasize that weakness in patients with gastrointestinal complaints may possibly be associated with GBS of *H. pylori* origin.

### Case Report

A 14-year-old male patient presented with weakness in his feet for the previous month and being unable to walk for a week. It was learned that the patient had had indigestion, abdominal pain and occasional nausea after meals for a year, had been diagnosed with a peptic ulcer and had been receiving treatment since then. Neurological examination of the patient showed that deep tendon reflexes were hypoactive in the upper extremities and absent in the lower extremities, while muscle strength was normal in the upper extremities, but 3/5 in the proximal and 2/5 in the distal parts of the lower extremities. Other system examinations were normal. IgG antibody levels against *H. pylori* were highly positive in method place serum and CSF samples, and protein (70 mg/dl) was high. Nerve conduction studies were performed on the median, ulnar, tibial, and peroneal nerves using conventional procedures including F wave analyses. These showed signs compatible with diffuse motor axonal neuropathy (reduced compound muscle action potential with normal conduction and sensory conduction velocities). The patient was therefore diagnosed as having acute motor axonal neuropathy type GBS, and intravenous immunoglobulin (400 mg/kg per day, 5 days) treatment

was given. After the treatment, partial recovery (4/5) was observed in muscle strength, which was totally restored 1 month after treatment. Thereafter, peptic ulcer treatment for a 6 week period (amoxicillin, metronidazole, omeprazole) was started. Treatment is still continuing.

GBS is the most common cause of acute flaccid paralysis in countries where poliomyelitis has been eradicated (12). It can affect people at any age, including neonates. Its incidence is 1.5-2.7 times higher in males than in females (13,14).

*H. pylori* infection may result in protean complications (chronic gastritis, gastric and duodenal ulcers, gastric cancer, mucosal B-cell lymphoma and vascular disorders), but it rarely causes neurological complications (11). *H. pylori* is the agent held responsible for the etiology of the axonal form of GBS in particular, and it is found at high levels in the cerebrospinal fluid of these patients (11,15). However, whether there is a relation between *H. pylori* antibodies and GBS prognosis is not known for sure. Chiba et Al. (10,15) found a high rate of anti-*H. pylori* IgG in CSF in GBS patients, particularly those with the motor axonal type. Our patient had also had gastrointestinal complaints for a long time and had been treated for *H. pylori*-associated peptic ulcer for the previous 2 months. The weakness that arose in the meantime was determined to be related to GBS by neurological examination, CSF and EMG findings. Moreover, peptic ulcer treatment had been given in addition to IVIG treatment, and it was thought that recovery from GBS was very fast because of the eradication of *H. pylori*. Under these conditions, it can be said that our patient had been suffering

for a long time from a disease possibly associated with *H. pylori*. It is known that extragastrintestinal findings of *H. pylori* infection (cardiovascular, cutaneous, autoimmune, allergic, liver, diabetes mellitus, growth retardation etc.) can be of immunological origin, and GBS is one of the leading findings (8,11,16,17). In addition, it has been reported that *H. pylori* causes some vascular lesions (such as atherosclerosis, ischemic heart disease and primary Raynaud's phenomenon). It has also been shown that toxins are accumulated in neural tissues in experimental studies(18). Late manifestation of GBS (thought to be caused by *H. pylori* in our patient) may be explained by the fact that the timing of the appearance of these complications depends on the effects of toxins and vascularity on the neural tissue. However, in order to support such a hypothesis, vascular and toxic neural damages should be demonstrated in *H. pylori*-induced experimental gastric studies. Therefore, all patients with peptic disease should be examined in terms of all possible and treatable agents. Thus, possible complications of peptic disease resulting from *H. pylori* can be prevented or treated by early diagnosis.

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