**Severe Acute Axonal Polyneuropathy in the Setting of Nutritional Deficiency**

Johanna Hamel and Eric Logigian

**OBJECTIVE:** Retrospective analysis of patients presenting with acute axonal neuropathy and nutritional deficiency resembling Guillain Barré syndrome (GBS*)*.

**BACKGROUND:** A GBS-like syndrome has been described in patients presenting with acute axonal neuropathy and normal CSF associated with protracted emesis and weight loss after bariatric surgery, alcohol exposure and anorexia. The nutritional mechanism is unclear.

**METHODS**: Retrospective description and analysis of patients presenting with acute axonal neuropathy, vomiting and nutritional deficiency.

**RESULTS:** 10 patients (mean age 35.6 years) presented with a severe, progressive, symmetric, often painful, sensory or sensorimotor polyneuropathy over 3-12 weeks with sensory ataxia, areflexia, variable muscle weakness but no ophthalmoparesis or encephalopathy. All patients had poor nutritional status and prolonged vomiting with weight loss. 7 patients were consuming alcohol extensively; 1 had undergone bariatric surgery and 2 were anorexic. Electrodiagnostic testing revealed a severe sensory or sensorimotor axonal neuropathy. CSF obtained in 7 patients was normal. Laboratory work-up revealed decreased prealbumin (n=5), low vitamin B6 levels (n=5), elevated MCV (n=6). Vitamin B1 was low in 2 patients. Folate and B12 were normal in all patients. Other metabolic derangements included hyponatremia, hypochloremia, hypokalemia, elevated liver function tests, and metabolic acidosis, due to dehydration and vomiting. Nerve biopsy in 2 patients confirmed axonal loss without demyelination or vasculitis. Most patients were treated with thiamine (n=8), multi-vitamin tablets (n=6), copper supplementation (n=1), vitamin B6 (n=3) and immunoglobulin (n=2).

**CONCLUSIONS**: We describe a GBS-like, acute, severe, axonal sensory or sensorimotor polyneuropathy in association with prolonged vomiting, weight loss, and poor nutritional status. A single causal vitamin deficiency was not identified. Vitamin B1 (thiamine) deficiency remains a hypothetical cause, but unfortunately B1 levels were generally tested after supplementation. Vitamin B6 was low in the majority of cases but is unlikely the only deficiency. We suggest that acute axonal neuropathy in patients with severe vomiting and weight loss previously described in the setting of alcohol abuse, gastric bypass or bulimia is one syndrome, likely caused by nutritional deficiencies. Awareness of this syndrome will reduce delay in targeted treatment of nutritional deficiencies (e.g., vitamin supplementation), and avoid immunotherapies when not indicated.