CASE REPORT

Guillain-Barré Syndrome and Acute Hepatitis E: A Rare Association

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Abstract

A 35-year-old male presented with acute onset ascending, areflexic paralysis of lower limbs, upper limbs, and weakness of respiratory muscles. He had anorexia, jaundice, and loose motions for the last 20 days. Nerve conduction studies revealed reduced compound muscle action potential (CMAP), markedly reduced nerve conduction velocity (NCV), and prolonged distal latencies. Sensory nerve action potential (SNAP) and F wave could not be recorded. These features were suggestive of acute motor sensory axonal neuropathy (AMSAN), a variety of Guillain-Barré syndrome (GBS). Patient had elevated liver enzymes and definite serological evidence of infection with acute hepatitis E. He was managed with intravenous immunoglobulin (IV-IG) and ventilatory support. Patient’s muscle power improved significantly within 5 days and liver enzymes returned to normal after 2 weeks.

Key Words

Guillain-Barré syndrome, Hepatitis E.

Case report

A 35-year-old male presented with a history of rapidly developing ascending weakness involving lower limbs, upper limbs, and the respiratory muscles—all of 3 days duration. Patient also had jaundice for the last 20 days. He had anorexia and loose motions at the onset of illness, which subsided within a week. There was no history of blood transfusion, needle injury, vaccination, or drug intake. On general examination, he had a pulse rate of 64/minute, blood pressure—120/84 mmHg, respiratory rate—18/minute, and the single breath count of 14. He was afebrile, and icterus was present. Nervous system examination on day 1 revealed normal higher mental functions, muscle power grade—0/V in lower limbs, III/V in upper limbs, and plantars were bilateral flexor. Deep tendon jerks were absent in lower limbs and sluggish in upper limbs. On day 2, his muscle power was grade—O/V in all the limbs, all deep tendon reflexes were absent, and the single breath count had reduced to 6. Rest of the central nervous system and other systemic examination were normal.

Laboratory investigation revealed normal complete blood count, serum electrolytes, and arterial blood gas analysis. Liver enzymes were elevated—an aspartate aminotransferase (AST)—976 U/L, alanine aminotransferase (ALT)—752 U/L, and alkaline phosphatase—239 U/L. Serum bilirubin was 91.8 mol/L. IgM antibody against hepatitis E virus was positive in serum and markers for hepatitis A, B, C, D were absent. ELISA for HIV was negative. Electrophysiological studies revealed reduced amplitude of compound motor action potential, markedly reduced nerve conduction velocity, and prolonged distal latencies (Fig. 1, 2). F-wave and sensory nerve action potential were absent. CSF study was not done as patient had weakness of 3 days duration. Ultrasound abdomen showed altered echotexture of liver parenchyma. Clinical, serological, and electrophysiological studies suggested a diagnosis of acute motor sensory axonal variety of GBS with concomitant acute hepatitis E virus infection.

Patient was managed in the intensive care unit. He was given ventilatory support for 2 days and IV-IG (intravenous immunoglobulin) in the dose of 0.4 gm/Kg/day for 5 days. He recovered his
power to grade IV/V within 5 days. Liver enzymes and muscle power returned to normal after 2 weeks of follow-up.

Discussion

GBS is an acquired motor dominating, areflexic, acute inflammatory polyradiculoneuropathy. It is an autoimmune disorder of heterogeneous group of pathological and clinical entities. Based on the electrophysiology there are three subtypes of GBS: a) acute inflammatory demyelinating polyradiculoneuropathy (AIDP) characterised by segmental demyelination and subsequent remyelination associated with recovery, b) acute motor axonal neuropathy (AMAN) which causes rapidly progressive weakness often with respiratory failure but has a good prognosis, c) acute motor-sensory axonal neuropathy (AMSAN) which has fulminant course with slow and incomplete recovery.

Campylobacter jejuni is the commonest and cytomegalovirus is the second most common antecedent infection reported. Other causes are Mycoplasma pneumoniae, Haemophilus influenzae, Influenza A and B viruses, parainfluenza type 1, adenovirus, Varicella zoster virus, HIV, and Epstein-Barr virus infection. Vaccines like oral polio, mumps/measles/rubella (MMR), tetanus toxoid, and hepatitis B have also been reported to cause GBS.

Murthy reported fifteen cases of GBS following specific infections (varicella-7, infective hepatitis-4, measles-2, and mumps-2). GBS has been reported with acute viral hepatitis A, hepatitis B, acute hepatitis C, and hepatitis D infections. Neurological manifestations of hepatitis E virus infection are rare; however Yadav et al have documented a case of oculomotor palsy associated with hepatitis E.
The exact pathogenesis of hepatitis causing GBS is not known. Antecedent infections are thought to trigger an immune response causing demyelination and axonal degeneration. There is possibility of molecular mimicry between antigens of hepatotrophic virus and components of myelin of peripheral nerves. Most of the investigators are of the opinion that GBS occurs due to host's immune attack on the nervous system triggered by antecedent viral hepatitis. The antibodies develop and lymphocyte sensitisation takes place, which is directed against the myelin of nervous system. Tsukada et al found immune complexes containing HbsAg in the serum and CSF of GBS associated with hepatitis B infection. IV-IG currently remains the treatment of choice and should be given within a week of illness at the dose of 0.4 gm/Kg/day for 5 days. There is no significant difference in efficacy between plasma exchange and IV-IG therapy.

Till date only one report of acute viral hepatitis E with GBS could be found in the literature. However, in this case, electrophysiological studies did not classify the type of neuropathy. The patient had shown spontaneous recovery over a period of two months without specific treatment. Our patient had acute motor-sensory variety of GBS, which has fulminant course with slow recovery. But our patient showed excellent improvement with IV-IG therapy. This is probably the first case of acute motor sensory type of Guillain Barré syndrome with concomitant acute hepatitis E - virus infection.

References