ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY FOLLOWING ACUTE HEPATITIS A INFECTION

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Guillain-Barre syndrome (GBS) subtype-acute motor and sensory axonal neuropathy (AMSAN) is considered to be a rare variant of GBS. Acute viral hepatitis A (HA), as an antecedent infection of AMSAN, has not been reported. We present a extremely rare case of GBS-subtype AMSAN following acute HA infection.

A 21-year-old male was transferred to our hospital due to respiration difficulties and progressive weakness from a local hospital. Physical examination upon referral to our hospital showed the patient presented with anorexia, jaundice, and elevated transaminase levels. IgM antibodies against HA were detected in blood and cerebrospinal fluid (CSF) samples. CSF analysis showed protein, 156 mg/dl; glucose, 61 mg/dl, with no pleocytosis. Results of serological and CSF tests for C. jejuni, cytomegalovirus, Epstein-Barr virus and H.influenzae infection were negative. The findings of motor and sensory NCSs showed an electrophysiologic pattern of axonal involvement without evidence of demyelination. In our case, clinical features and electrophysiological studies were consistent with AMSAN following acute hepatitis A virus infection.

A variety of neurological syndromes including GBS have been reported in serologically confirmed hepatitis A. A cross-reaction between Schwann cells, myelin or other peripheral nerve antigens remains a possibility, but any molecular resemblance between hepatotropic viruses and structural components of peripheral nerves has not been explored. Taking into account these facts, we presume that the epitope of HA virus and the axonal component of peripheral neural tissue might have molecular mimicry in our case. Further studies are needed to ascertain this exact mechanism.