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# Neurological Uniqueness: A Case Study of Hepatitis A-Induced Acute Inflammatory Demyelinating Polyneuropathy

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### Abstract

Acute inflammatory Demyelinating Polyneuropathy (AIDP) with hepatitis A (HA) is more likely to affect men, develop at a younger age, and have a better prognosis overall. The progression of the Hepatitis A Virus (HAV)-caused liver inflammation and the neurological difficulties could lead to AIDP in the early stages of the hepatitis signs and symptoms. The World Health Organization (WHO) estimates 1.5 million clinical HAV cases annually. Extrahepatic complications of this disease are rare. The etiology of HA associated AIDP remains unclear, with cross-reactive HA epitopes between the peripheral nervous system and other authors have hypothesized that the presence of CSF antibodies reflects direct entry into the central nervous system. Our patient presentations favored AIDP most commonly in HA. A 22-year-old man, with no prior significant medical history, presented to neurology emergency with a 3-day history of acute onset, had been complaining of nausea, general weakness, yellowing of the sclera and history of fever for 5 days. Further investigation revealed marked elevation of liver enzymes in a pattern suggestive of hepatocellular processes. Serum titres of hepatitis B, C, and E were negative, but IgM anti-HAV was positive (enzyme immunoassay). Elevation of Cerebrospinal fluid (CSF) protein and myelitis transversa shown by magnetic resonance imaging were established. The diagnosis as AIDP was taken and treated by symptomatic and neurology treatment. However, the patient regained strength and underwent physiotherapy during two weeks. Approximately 3 months after discharged, the patient's gait had nearly returned to baseline at follow-up and the symptoms slowly improved.



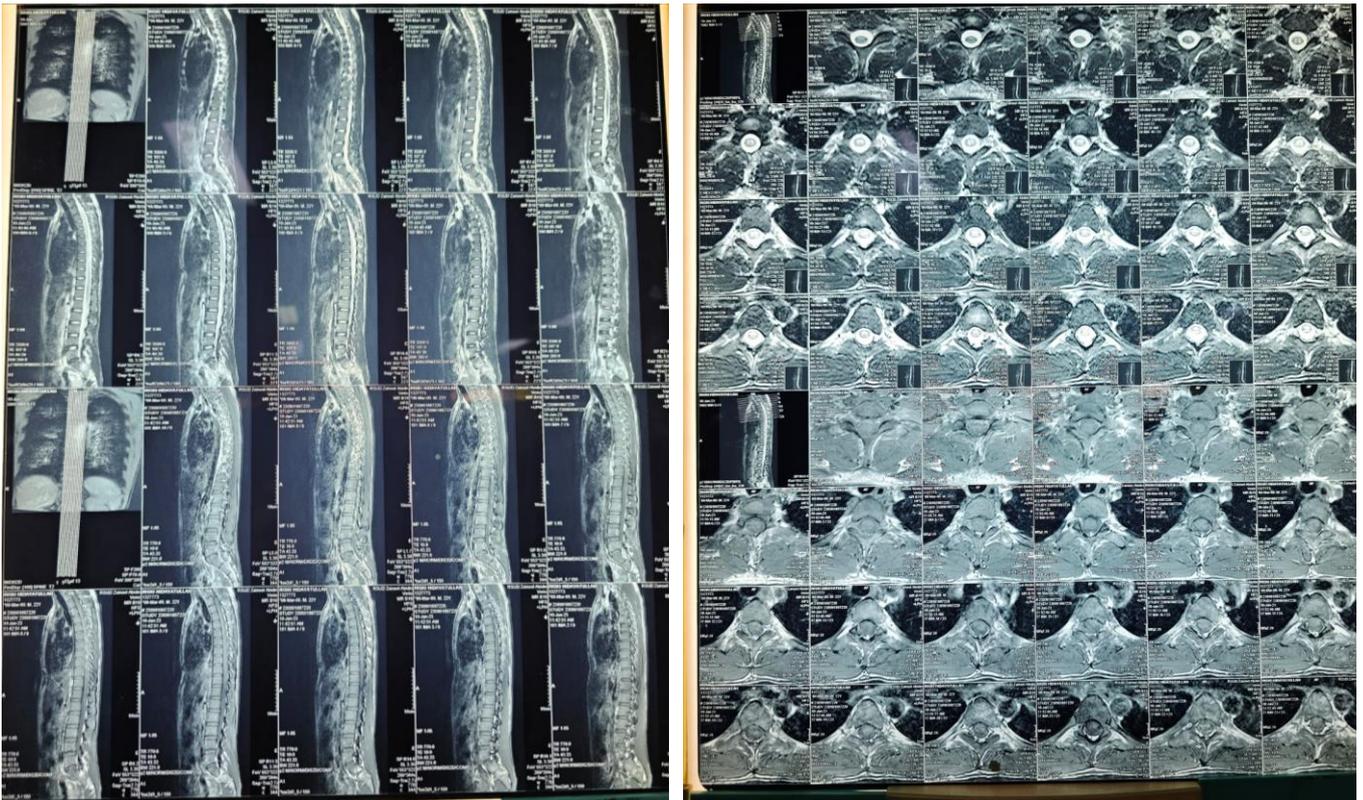
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## 1. Introduction

Hepatitis A virus (HAV) is a global infectious agent responsible for acute hepatitis. The primary mode of transmission for hepatitis A (HA) is typically via the oral-faecal route, involving exposure to contaminated food, water, or close contact with an infected individual. Notably, infection rates are generally low in developed nations, as stated by the World Health Organization (WHO). Nevertheless, populations at a higher risk of infection encompass drug users, men who engage in

sexual activity with men, individuals traveling to regions where the disease is prevalent, as well as secluded communities. Instances of extrahepatic complications stemming from this illness are infrequent [1].

The four most common subtypes of Guillain-Barré syndrome (GBS) are acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy, acute motor and sensory axonal neuropathy, and Miller-Fischer syndrome. The source of HA-associated GBS remains unclear, with cross-reactive HA epitopes



**Figure 1.** Magnetic resonance imaging of spine with contrast and without contrast.

between the peripheral nervous system and other authors have hypothesized that the presence of cerebrospinal fluid antibodies reflects direct entry into the central nervous system [2]. The incidence of acute inflammatory demyelinating polyneuropathy (AIDP), also known as GBS, acute inflammatory polyneuropathy, or postinfectious polyneuropathy, is approximately 2 per 100,000 people. AIDP presents with progressive muscle weakness, usually symmetry, loss of reflexes, decreased distal sensation, frequent evidence of autonomic dysregulation, and elevation of cerebrospinal fluid (CSF) proteins without cellular increases. AIDP is a motor-dominant neuropathy, but early symptoms are often characterized by paresthesia. Diffuse back and muscle pain are also common during the course of the disease but usually cause no symptoms [3].

AIDP represents the most prevalent variant of GBS. It is distinguished by a combination of diminished reflexes, progressive muscle weakness that starts in the lower extremities and moves upwards, involvement of cranial nerves, and subtle sensory alterations as per classical descriptions. This disorder is of autoimmune origin. Annually, around 3000 to 6000 cases are identified in the United States. Generally, symptoms emerge within 1 to 6 weeks after a preceding upper respiratory or gastrointestinal infection. Hospitalization in the intensive care unit is frequently required. These patients are prone

to experiencing rapid-onset weakness, which might lead to respiratory failure (observed in 20% of AIDP cases), as well as dysfunction of the autonomic nervous system. Timely recognition, proper management, and targeted therapy for AIDP play a pivotal role in enhancing patient outcomes and averting complications. Consequently, it becomes imperative for emergency medical practitioners to cultivate an extensive array of potential diagnoses when assessing patients who present with neurological complaints and observable physical manifestations [4].

Administration of AIDP is interdisciplinary. Both plasmapheresis and intravenous immunoglobulin are equally effective in reducing disease severity and neuropathy. Once medically stable, patients can be managed at a general medical/neurological level, but continued vigilance is important to prevent respiratory, cardiovascular, and other medical complications. Patients with significant functional limitations may require transfer to an inpatient rehabilitation facility. Ongoing care is also necessary to minimize issues related to immobility, nervous bowel and bladder, and pain. Early involvement of medical staff is recommended. Early detection and treatment of GBS may be important for long-term prognosis, especially in patients with advanced age, rapidly progressing course, and poor clinical outcome, such as a history of diarrhea. Immunomodulatory treatments have been used to

hasten recovery. Intravenous immunoglobulin (IVIG) and plasmapheresis have been shown to be equally effective [4]. Here in, we reported a case of AIDP following HA.

## 2. Cases

A 22-year-old man, with no prior significant medical history, presented to neurology emergency with a 3-day history of acute onset, had been complaining of nausea, general weakness, and yellowish of the sclera. He had history of fever for 5 days. A week prior to this, the patient had a fever with a dry cough that lasted for 5 days, for which the patient took over-the-counter medications. There was no history of canned food consumption, loose bowels, exanthem, recent vaccination, or dog or snake bite. There was no family history of a similar illness. Further investigation revealed marked elevation of liver enzymes in a pattern suggestive of hepatocellular processes. Serum titres of hepatitis B, C, and E were negative, but IgM anti-HAV was positive (enzyme immunoassay). Malaria antigen was negative. The patient was received symptomatic treatment.

Over the next 15 days, there was an acute onset of progressive asymmetric (left>right) weakness in both upper extremities that developed. This weakness then progressed and affected the lower extremities over the following 2 days. The weakness did not further progress, and there were no associated dysphagia, nasal insufficiency, neck weakness, facial weakness, shortness of breath, bladder-bowel involvement, or cardiac arrhythmias.

On examination, jaundice and normal respiratory rate of were pronounced. Bilateral fundus examination and all cranial nerves were normal. Motor system examination showed normal nutrition and muscle tone, strength was MRC grade 4/5 in both proximal and distal lower extremities, left upper extremity strength was 3/5 distal, proximal It was 4/5 in position and right side.

Notably, the deep tendon reflexes exhibited activity, and the soles displayed lateral stretching. Surface reflexes, encompassing abdominal reflexes, remained intact. Sensory examination yielded normal results, and Romberg's sign was negative. No indications of cerebellar or meningeal irritation were observed. The neurological assessment revealed full alertness, absence of bilateral ptosis, unimpaired extraocular movements with appropriate pupillary light response, absence of jaw weakness, bilateral facial weakness characteristic of lower motor neuron involvement, as well as the absence of cough and gag reflexes. Around 4 weeks following symptom onset, the patient demonstrated signs of improvement, consistent with the typical progression of AIDP cases. Conversely, the reported symptoms of

general malaise, joint pain, chills, and nausea, which led to the prescription of antibiotics, were likely linked to HAV infection.

An elevation in cerebrospinal fluid (CSF) protein to 65 milligrams per deciliter (mg/dL) (within a normal reference range of 12-60 mg/dL) was observed, along with the presence of two nucleated cells and three erythrocytes. Nonetheless, the patient regained strength and underwent a two-week course of physiotherapy. Approximately 3 months after discharge, the patient's gait had nearly returned to baseline during follow-up, and symptoms showed gradual improvement. Magnetic resonance imaging of the spine, both with and without contrast, revealed myelitis transversa, characterized by enlargement of the 1st to 7th nerve roots in the cervical spine as seen in AIDP.

## 3. Discussions

AIDP has been characterized as T cell-mediated disorder affecting nerves. Infiltration of nerves by lymphocytes is a common occurrence. Recognizing the potential role of antibodies in the pathogenesis of various GBS subtypes has prompted investigations into antibody-mediated mechanisms in AIDP. There have been reports of antibodies targeting gangliosides, basal lamina components, and other proteins in a limited number of AIDP cases. The absence of a singular antigen has raised doubts about the causal role of antibodies in AIDP's development. Nevertheless, clear correlations seem to exist between the presence of antiganglioside antibodies and the clinical manifestations of AIDP. For instance, GM1 antibodies are detected in approximately 15%–20% of AIDP cases and are associated with distinct motor involvement and particularly severe disease often involving nerves. Studies suggest prolonged recovery in AIDP patients who test positive for GM1 antibodies [5].

In AIDP patients who show serological indications of cytomegalovirus infection, roughly 50% of cases reveal the presence of GM2 antibodies. These individuals often display more marked clinical manifestations when contrasted with AIDP patients lacking GM2 antibodies. Furthermore, antibodies directed at different gangliosides, such as GM1b, GalNAc-GD1a, and LM1, have been pinpointed in AIDP patients. The mechanisms underlying the emergence of these antibodies remain elusive.

In a cohort of Dutch AIDP patients, there was a prevalence of IgM antiganglioside antibodies, whereas Japanese AIDP patients exhibited a greater prominence of IgG antibodies. Moreover, there have been reports of antibodies targeting an array of nerve and myelin proteins beyond gangliosides, potentially playing a role in

the pathogenesis of specific AIDP cases. Koski, utilizing a highly sensitive approach involving the C1 fixation and exchange assay, identified circulating IgM antibodies against peripheral nerve myelin in over 90% of AIDP cases.

The demographic and clinical characteristics of patients with Acute-onset Chronic Inflammatory Demyelinating Polyneuropathy (A-CIDP) and those with AIDP in the initial 8 weeks are highly similar. However, alterations in proprioception are more commonly observed in A-CIDP patients. The underlying pathophysiological mechanisms of A-CIDP remain elusive. From a phenotype and diagnostic standpoint, distinguishing between AIDP and A-CIDP may not be necessary. Nevertheless, identifying risk factors associated with relapse or further progression could offer valuable insights for prognostic and therapeutic considerations [6].

In spite of the fact that neurologic signs related with hepatitis are moderately uncommon, it would be typically developed within the setting of different extrahepatic appearances of viral hepatitis. The fringe and central nervous system can be included amid viral hepatitis infections. Peripheral anxious framework appearances of viral hepatitis diseases incorporate an intense fiery AIDP that mirrors Guillain-Barré disorder, a constant

AIDP has been commonly detailed to be gone before with intense viral hepatitis B, C, D, and once in a while with HA. Be that as it may, within the estimation of HA widespread in Shanghai between January and Walk 1988, out of 2,92,301 cases of HA, eight were analyzed with AIDP and they concluded that activating AIDP post HA disease was not rare.

#### 4. Conclusions

The conclusions section should summarize the key findings of the report and their significance. It should also reiterate the purpose of the report and its potential impact on clinical practice. The conclusion should be clear and concise, leaving the reader with a clear understanding of the case and its implications. Finally, it may be appropriate to include any final thoughts or recommendations for future research.

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incendiary demyelinating polyneuropathy (CIDP), a disconnected mononeuritis or a mononeuritis multiplex disorder, a symmetric sensorimotor polyneuropathy, and a subacute fiery myopathy.

AIDP could be an uncommon neurological clutter which predominantly among both and females and distinctive age bunches. In this clutter, resistant cells assault myelinated neurons, driving to demyelination and intense axonal degeneration. Consequently, it is tallied among immune system infections. Dynamic muscle shortcoming, shivering sensation is regularly watched in appendages, and deadness are found in a normal AIDP understanding. Lumbar cut is regularly found related in all cases of suspected AIDP.

In AIDP the genders are similarly influenced. A preparatory disease goes before the neurologic indications by five days to three weeks and facial nerve paralysis is watched in more than 50% of the cases. A few patients have stamped lessening within the recognition of joint position and/or vibration, and ideal recuperation happens over a period of weeks or months. Two-thirds of all cases of GBS take after a contamination. Upper respiratory disease and enterocolitis are common antecedents. In an arrangement of 1,100 cases of AIDP, as it were eleven taken after viral hepatitis.

**Informed Consent Statement:** The patient who participated in this study has provided informed consent, including approval for the release of all data, and has agreed to it verbally.

**Data Availability Statement:** The article encompasses all essential data required to substantiate the findings.

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