**CASE REPORT**

**AMAN with Ophthalmoparesis: A Rare Presentation**

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**ABSTRACT**

Acute motor axonal neuropathy (AMAN) is a variant of Guillain–Barré syndrome (GBS), characterized by acute areflexic flaccid quadriparesis with motor axonal changes and absence of demyelinating findings in electrophysiological studies. A 30-year-old man presented with acute onset flaccid type of weakness involving all four limbs, along with drooping of eyelids. Examination revealed ptosis with restricted horizontal and vertical eye movements. Spinomotor system examination revealed acute flaccid areflexic quadriparesis. Nerve conduction studies (NCS) showed features suggestive of motor axonal neuropathy changes. Cerebrospinal fluid (CSF) revealed albuminocytological dissociation. The diagnosis of AMAN was made, and the patient was treated with intravenous immunoglobulin (IVIg). His weakness gradually improved over 1 month, with partial improvement in ptosis and eye movements. This case highlights the occurrence of ophthalmoparesis in the AMAN variant of GBS. The presence of ophthalmoparesis and areflexia makes it necessary to exclude Miller-Fisher syndrome. But, the presence of axonal changes in nerve conduction study and the profound weakness with negative serum anti-GQ1b antibody profile, supports the diagnosis of AMAN.

**INTRODUCTION**

Acute motor axonal neuropathy (AMAN) is characterized clinically by a pure motor syndrome without sensory involvement. The electrophysiological findings show a decreased amplitude of compound muscle action potential (CMAP) without any evidence of demyelination or changes in sensory nerve action potential (SNAP). We report a case of the AMAN variant presenting with external ophthalmoparesis and symmetric proximal and distal weakness without sensory abnormalities.

**CASE DESCRIPTION**

A 30-year-old man without any comorbidity presented with acute onset flaccid-type weakness of both lower limbs. Within 1 day, the weakness progressed to involve both upper limbs. The next day, he developed drooping of both eyelids. The patient had a history of fever 3 days before these symptoms. There was no history of neck pain, sensory disturbances, urinary retention, ileus, or respiratory compromise.

Examination revealed ptosis (Fig. 1) with restricted horizontal and vertical eye movements, initially in the left eye followed by the right eye. He also had bilateral lower motor neuron (LMN) facial paresis (Fig. 2). Spinomotor system examination revealed hypotonia and areflexia of all limbs. Muscle power examination showed bilateral symmetrical proximal and distal weakness, with the lower limbs being affected more than the upper limbs (Table 1). Plantar reflex showed bilateral flexor response. Sensory and cerebellar examinations were normal.

Complete hemogram, random blood sugar, liver function tests, renal function tests, serum electrolytes, thyroid function test, urine analysis, and vasculitic workup were normal. Serology tests for human immunodeficiency virus, hepatitis B, and hepatitis C were negative. Magnetic resonance imaging scans of the brain and spine were normal. Cerebrospinal fluid (CSF) analysis revealed elevated protein levels with albuminocytological dissociation (Table 2). The serum ganglioside GQ1b antibody was negative. Nerve conduction studies (NCS) done on the day of admission showed normal findings (Table 3), with no denervation potential and motor unit potentials detected in needle electromyography (EMG) (Table 4).

The patient’s clinical findings did not show improvement at the end of 2 weeks. On the 14th day, repeat electrophysiological studies revealed a marked reduction in CMAP amplitude in all four limbs, with normal distal latencies and conduction velocities. Sensory nerve conduction velocities and sensory

**Table 1:** Grading of muscle power on admission, discharge, and follow-up

<table>
<thead>
<tr>
<th>Power</th>
<th>On admission (day 01)</th>
<th>On admission (day 14)</th>
<th>Discharge (day 45)</th>
<th>Follow-up (day 72)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UL*—proximal</td>
<td>4/5</td>
<td>3/5</td>
<td>4/5</td>
<td>4+/5</td>
</tr>
<tr>
<td>UL—distal</td>
<td>4/5</td>
<td>3/5</td>
<td>4/5</td>
<td>4+/5</td>
</tr>
<tr>
<td>LL*—proximal</td>
<td>1/5</td>
<td>0/5</td>
<td>3/5</td>
<td>4−/5</td>
</tr>
<tr>
<td>LL—distal</td>
<td>1/5</td>
<td>0/5</td>
<td>3/5</td>
<td>4−/5</td>
</tr>
</tbody>
</table>

UL, upper limbs; LL, lower limbs

**Fig. 1:** Ptosis of both eyes (left > right)

**Fig. 2:** Absence of both nasolabial folds

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action potentials were normal. F-waves were impersistent in the bilateral median and peroneal nerves (Table 3).

The diagnosis of the AMAN variant of Guillain–Barre syndrome (GBS) was made based on clinical presentation, which was further supported by NCS findings. The patient was treated with 0.4 gm/kg/day intravenous immunoglobulin (IVIg) for 5 days. His weakness gradually improved during the next 4 weeks, with partial improvement in ptosis and eye movements.

**Discussion**

Acute motor axonal neuropathy (AMAN), characterized by decreased CMAP and the absence of demyelinating findings in electrophysiological studies, is a variant of GBS. Patients diagnosed with AMAN typically experience a more rapid progression of weakness, reaching an earlier nadir compared to AIDP, resulting in prolonged paralysis and respiratory failure over a few days. In our case, the patient had a rapid worsening of symptoms with prolonged motor weakness, without any respiratory failure.

**Conclusion**

This case highlights the occurrence of ophthalmoparesis in the AMAN variant of GBS. To the best of our knowledge and literature search, no case reports are available yet for the same. The presence of ophthalmoparesis and areflexia makes it necessary to exclude Miller–Fisher syndrome. However, the presence of axonal changes in nerve conduction study and profound weakness, with the absence of ataxia and negative serum anti-GQ1 antibody profile favors the diagnosis of AMAN more likely.

**References**