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Acute Axonal Polyneuropathy in a Chronic Alcoholic Patient: A Rare Presentation

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Abstract

Guillain Barre Syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy of autoimmune nature, characterized by rapidly progressive, symmetric weakness and areflexia. Acute motor sensory axonal neuropathy is a variant of GBS with motor paralysis, hyporeflexia/areflexia, and sensory loss. Alcohol produces central nervous system toxicity as its principal side effects. Alcoholic neuropathy is a mixed sensory and motor disorder affecting large and small fibers to varying degrees. In contrast to the classic, slowly progressive polyneuropathy in alcoholic patients, acute forms, clinically mimicking GBS, are rare. This is a case of a 37 year old chronic alcoholic lady presenting with subacute onset weakness of both lower limbs whose laboratory data indicate neuropathy of acute motor sensory axonal pattern. We emphasize that acute alcoholic neuropathy has to be distinguished from GBS and other forms of acute polyneuropathy by using clinical, laboratory, and electrophysiological data for therapeutic and prognostic concerns.

Keywords: Acute Motor Sensory Axonal Neuropathy, Acute Polyneuropathy, Alcoholic Neuropathy, Guillain Barre Syndrome
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1. Introduction

Alcoholic neuropathy is one of the most common peripheral neuropathy seen in clinical practice. Chronic alcoholic neuropathy typically presents as a sensorimotor, axonal polyneuropathy numbness, weakness, and sensory ataxia. Electrophysiologic and histopathological studies demonstrated that axonal degeneration was the primary pathophysiological mechanism in chronic alcoholic polyneuropathy.[1] Acute axonal neuropathy in chronic alcoholics, as described in this case report, have been reported as rare case scenarios.[2-4]

2. Case Report

A 37 year old homemaker presented with subacute onset, symmetric flaccid paraparesis involving both proximal and distal muscle groups without any abnormal movements, wasting, or fasciculations, which reached to its maximum severity over 2 weeks. It was associated with tingling and burning paresthesias in all 4 limbs predominantly distal parts without any objective sensory loss, truncal, or neck weakness. There was no history suggestive of higher mental function or cranial nerve involvement.

The patient was nondiabetic, nonhypertensive, not on any long term medication. There was no history of any chronic illness or recent diarrhea, upper respiratory tract infection, and vaccination in preceding 1 month.

But the patient was addicted to alcohol since last 15 years and used to take about 100 g of ethanol per day. She was also chronic smoker since last 15 years. However, there was no recent history of binge drinking or recurrent vomiting.

Examination revealed her vitals were within normal limits, and there was no sign of malnutrition. There was hypotonia in both lower limbs and grade 3/5 power distal and 2/5 in the proximal group of muscles according to Medical Research Council. Hand grips were weak, more so on the left side. Plantars were bilaterally nonresponsive.

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Deep tendon reflexes were absent in lower limbs and sluggish in upper limbs. There was no objective loss of pain, temperature sensation, or sphincter involvement. Vibration sense was absent up to anterior superior iliac spines in lower limbs and up to elbows in upper limbs. Joint position sense was also impaired distally. There was moderate hepatomegaly.

Blood picture revealed mild macrocytic anemia (hemoglobin 10 g/dl and mean corpuscular volume 107 fl). Liver function test showed normal bilirubin with raised aspartate aminotransferase (test: 120 U/L, normal: 12–38 U/L), alanine aminotransferase (test: 60 U/L, normal: 7–41 U/L), raised alkaline phosphatase (376 U/L), and low albumin (1.9 g/dl). Sodium was 144 meq/L, and potassium was 3.6 meq/L. Human immunodeficiency virus 1 and 2 were nonreactive, and thyroid function tests were normal. Serum level of vitaminB12 (test: 306 pg/ml, normal: 279– 996 pg/ml), folic acid (test: 20 ng/ml, normal: 2–20 ng/ml), thiamine (test 70 nmol/L, normal: 70–180 nmol/L), and methylmalonic acid (test: 4 μg/dl, normal: 0–4.7 μg/dl) were within normal limits. Ultrasonography of the whole abdomen revealed enlarged liver (span 17 cm) with moderate to severe fatty changes.

Motor nerve conduction studies (NCS) in upper limbs showed normal distal latency in both median and ulnar nerve with normal conduction velocity. In left median, compound muscle action potential amplitude was significantly reduced. Motor NCS in lower limbs revealed both peroneal and posterior tibial nerves were inexcitable. Sensory NCS showed normal distal latency but reduced sensory nerve action potential (SNAP) amplitude in both median nerves and absence of SNAPs in both sural nerves [Tables 1-3]. Hence, NCS in overall revealed sensory motor neuropathy (axonopathy) predominantly affecting lower limbs. The cerebrospinal fluid (CSF) study was normal except for mild elevation of protein (67 mg/dl). Magnetic resonance imaging brain showed mild widening of cortical sulci with periventricular hyperintensities, slightly prominent lateral ventricles [Figure 1].

Antinuclear antibody, antineutrophil cytoplasmic autoantibody, perinuclear, antineutrophil cytoplasmic autoantibody, cytoplasmic, and anti-GM1 ganglioside antibodies were found to be negative in her serum.

The patient was treated conservatively and as there was no progressive deterioration intravenous immunoglobulin (IVIg) was not considered. Daily physiotherapy was given during admission and was continued after discharge. She was kept under deaddiction program. After 1 month, she regained her ability to walk.

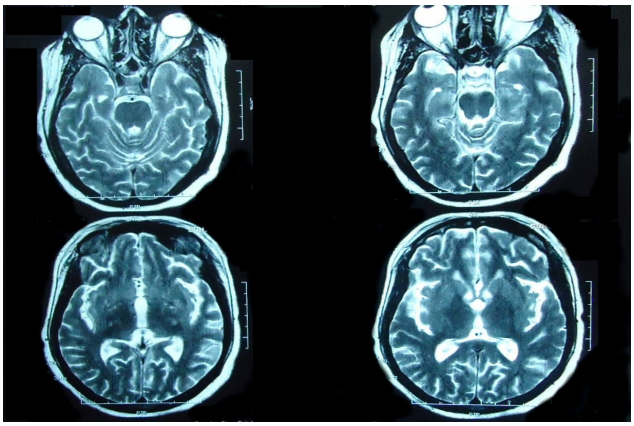


Figure 1. MRI brain showed mild widening of cortical sulci with periventricular hyperintensities, slightly prominent lateral ventricles

Table 1. Nerve Conduction Study : Motor

MOTOR	DISTAL LATENCY (mS)	AMPLITUDE (mV)	CV (m/s)
RIGHT MEDIAN	2.88 (N<3.5)*	10.37 (N>10)*	68.57 (N>45)*
RIGHT ULNAR	1.88 (N<2.5)*	9.49 (N>10)*	69.52 (N>45)*
LEFT MEDIAN	3.25 (N<3.5)*	3.74 (N>10)*	62.02 (N>45)*
LEFT ULNAR	1.88 (N<2.5)*	6.54 (N>10)*	61.32 (N>45)*
RIGHT POSTERIOR TIBIAL NERVE	INEXCITABLE	INEXCITABLE	INEXCITABLE
RIGHT COMMON PERONEAL NERVE	INEXCITABLE	INEXCITABLE	INEXCITABLE
LEFT POSTERIOR TIBIAL NERVE	INEXCITABLE	INEXCITABLE	INEXCITABLE
LEFT COMMON PERONEAL NERVE	INEXCITABLE	INEXCITABLE	INEXCITABLE

mS : milli second, mV: milli volt, CV: Conduction Velocity, m/s: meter per second

* The normal reference values used in the Nerve Conduction Study are as per the normative data used in our Electrophysiology Laboratory

Table 2. Nerve Conduction Study : Sensory

SENSORY	DISTAL LATENCY (mS)	AMPLITUDE (μV)
RIGHT MEDIAN	2.3 (N<3.5)*	5.14 (N>10)*
RIGHT ULNAR	2 (N<2.5)*	15.46 (N>10)*
LEFT MEDIAN	2.65 (N<3.5)*	4.23 (N>10)*
LEFT ULNAR	2.15 (N<2.5)*	11.09 (N>10)*
RIGHT SURAL	ABSENT	ABSENT
LEFT SURAL	ABSENT	ABSENT

mS: milli second, μV: micro volt

* The normal reference values used in the Nerve Conduction Study are as per the normative data used in our Electrophysiology Laboratory

Table 3. Nerve Conduction Study : F Wave

NERVE	F Minimum Latency (mS)	F Maximum Latency (mS)	Chrono Dispersion (mS)
RIGHT MEDIAN	24.62 (N<28)*	27.5 (N<32)*	2.88 (N<4)*
RIGHT ULNAR	24.88 (N<28)*	27.5 (N<32)*	2.62 (N<4)*
LEFT MEDIAN	25 (N<28)*	27.5 (N<32)*	2.5 (N<4)*
LEFT ULNAR	25 (N<28)*	27.5 (N<32)*	2.5 (N<4)*

* The normal reference values used in the Nerve Conduction Study are as per the normative data used in our Electrophysiology Laboratory

3. Discussion

Wöhrle *et al.* and Tabaraud *et al.* described alcohol related acute axonal polyneuropathy in five and eight chronic alcoholics, respectively.[2,3] In their descriptions, CSF were almost normal. But CSF albuminocytological dissociation and improvement after IVIg administration was mentioned by Sahin *et al.* in their case report.[5]

The patient described here had fulfilled the major diagnostic criteria of Guillain Barre syndrome (GBS).[6] Routine blood investigations and ultrasound abdomen were pointing chronic alcohol intoxication. NCS clearly indicated acute axonal neuropathy as the cause of weakness in this patient.

Alcoholic cerebral atrophy, though not a well-defined entity, may be found in young alcoholics with or without symptoms of the cerebral disease, with enlarged cerebral ventricles and widened sulci mainly of the frontal lobe.[7]

An association of *Campylobacter jejuni* infection with GBS and positivity of GM1 ganglioside antibody was confirmed in previous studies.[8,9] Our patient was negative for anti-GM1 antibody and she had no history of preceding diarrhea. But *Campylobacter* antibody titre were not determined.

The pathophysiological mechanism of polyneuropathy in patients with chronic alcohol abuse are still a matter of debate. Animal studies gave an evidence for a direct toxic effect of alcohol.[10] Patients with alcoholic neuropathy with regular intake of >100 g of ethanol for at least 10 years before the onset of neuropathic symptoms and normal thiamine levels were found to have slowly progressive, painful, and predominantly sensory symptoms with relatively selective small fiber axonal loss. Large fiber axonal loss is predominant in the thiamine deficient group, and the patients are found to have acutely progressive, motor dominant symptoms with impairment of superficial and deep sensation. Patients with alcoholic neuropathy and thiamine deficiency had characteristics of both the alcoholic neuropathy and thiamine deficient neuropathy.[11]

Thiamine status of this patient was normal. The preexistance of subclinical chronic polyneuropathy due to chronic alcoholism cannot be methodologically ruled out. But clinical features, electrodiagnostic test, and

laboratory features in this patient indicate probably it was due to the toxic effect of alcohol.

4. Conclusion

The diagnosis of alcohol related acute axonal polyneuropathy was established in this patient based on the clinical, laboratory, and electrophysiological studies. As the serum vitamin levels were normal, we propose this event to be directly related mostly to the toxic effect of alcohol. This case was unique in the sense that it mimics GBS in an alcoholic patient and has normal serum vitamin levels. The present case is important because it is a reminder that atypical acute polyneuropathy can be a complication of alcoholism and its distinction from classic demyelinating GBS and axonal variant of GBS is clinically important because of different management.

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