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Acute Motor and Sensory Axonal Neuropathy: A Case Report

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Abstract
One of the subtypes of Guillain – Barre syndrome is Acute motor and sensory axonal neuropathy (AMSAN) which is characterized by acute onset of distal weakness, loss of deep tendon reflexes, and sensory symptoms. A 65-yr-old male came to our hospital due to respiration difficulties and progressive weakness since 10 days. Nerve conduction studies showed reduced amplitudes of compound muscle action potentials in bilateral peroneal, and posterior tibial nerves with decreased sensory nerve conduction velocity, amplitude and delayed latency in left ulnar nerve. Absent sensory conduction in bilateral median, right ulnar in upper limb and bilateral superficial peroneal and sural nerves in loer limbs. Based on clinical features, laboratory findings, and electrophysiologic investigation, the patient was diagnosed as AMSAN. The patient was treated with symptomatic treatment and physiotherapy.

INTRODUCTION

The four most common subtypes of Guillain – Barre syndrome are : acute inflammatory demyelinating polyradiculoneuropathy (AIDP) – most common , the acute motor axonal neuropathy (AMAN), the acute motor and sensory axonal neuropathy (AMSAN) and Miller Fisher's syndrome [(1)]. Amongst these, AMSAN is considered to be a rare variant of GBS, and one that usually has a more serious clinical course and slower recovery than the classic demyelinating form of GBS [(2)]. Many immunological mechanisms and infectious agents play a role in pathogenesis of Guillain-Barre syndrome (GBS). The pathogenesis of AMSAN has been associated with antecedent *Campylobacter jejuni* (*C. jejuni*) infection [(2)]. We present a case of GBS-subtype AMSAN who had presented with respiratory difficulties, limb weakness and had history of hepatitis since 10 days .

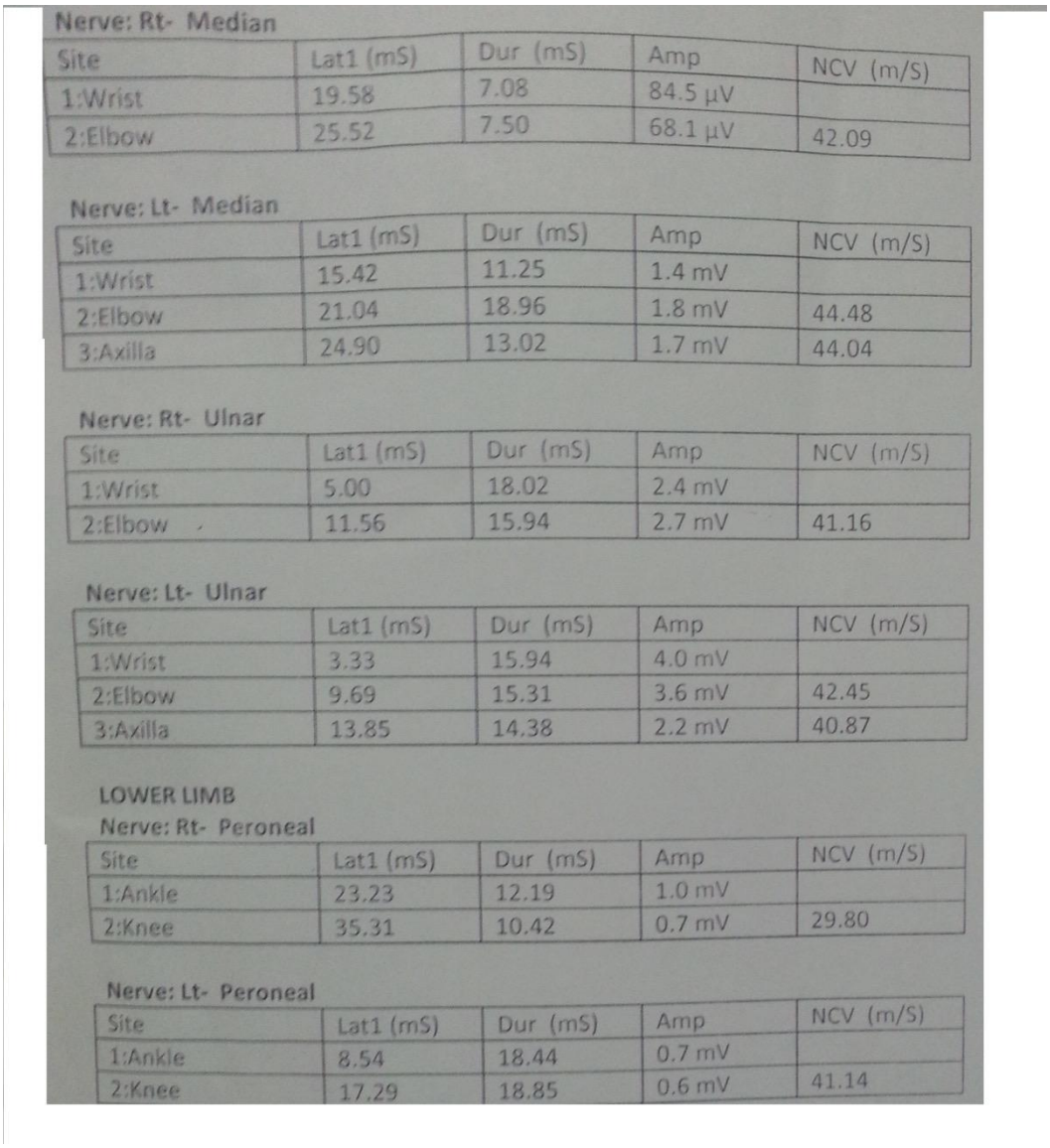
CASE DESCRIPTION

A 65-yr-old male came to our hospital due to respiration difficulties and progressive weakness from a local hospital where the patient was being treated since 10-days for acute viral hepatitis. Patient gave history of decreased appetite, yellowish discoloration of eyes and generalized weakness since 10 days. He did not have any past history of respiratory or gastrointestinal infection in the past 1 month before onset of progressive weakness. On Admission, he was conscious oriented, vitals were stable, patient had Icterus. On abdominal palpation he did not have any organomegaly. Single breathe count was 20. On

neurological examination, the mental status of the patient was alert. By the Medical Research Council (MRC) grading, the patient had weakness with areflexia in both proximal upper limbs (MRC grading 4), both distal upper limbs (MRC grading 2), both proximal lower limbs (MRC grading 4), and both distal lower limbs (MRC grading 2). Bilateral Planters were absent. Pin-prick, vibratory, and joint position senses were reduced in glove and stocking distribution.

Laboratory tests revealed a Total Bilirubin of 4.2 mg/dl, Indirect 2.1 mg/dl . SGOT was 83, SGPT 54. Viral markers for Hepatitis A virus were Negative. CSF analysis showed protein, 92 mg/dL; glucose, 64 mg/dL, with no pleocytosis.

Nerve conduction studies (NCSs) were performed at 2 weeks after the onset of weakness. The findings of motor NCSs showed markedly reduced amplitudes and conduction velocities in bilateral median and ulnar nerve in upper limb and bilateral peroneal, and posterior tibial nerves, without evidence of demyelination. The findings of sensory NCSs revealed decreased sensory nerve conduction velocity, amplitude and delayed latency in left ulnar nerve. Absent sensory conduction in bilateral median, right ulnar in upper limb and bilateral superficial peroneal and sural nerves in lower limbs. F-wave response was normal in bilateral tibial nerves.



Nerve: Rt- Median

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	19.58	7.08	84.5 µV	
2:Elbow	25.52	7.50	68.1 µV	42.09

Nerve: Lt- Median

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	15.42	11.25	1.4 mV	
2:Elbow	21.04	18.96	1.8 mV	44.48
3:Axilla	24.90	13.02	1.7 mV	44.04

Nerve: Rt- Ulnar

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	5.00	18.02	2.4 mV	
2:Elbow	11.56	15.94	2.7 mV	41.16

Nerve: Lt- Ulnar

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	3.33	15.94	4.0 mV	
2:Elbow	9.69	15.31	3.6 mV	42.45
3:Axilla	13.85	14.38	2.2 mV	40.87

LOWER LIMB

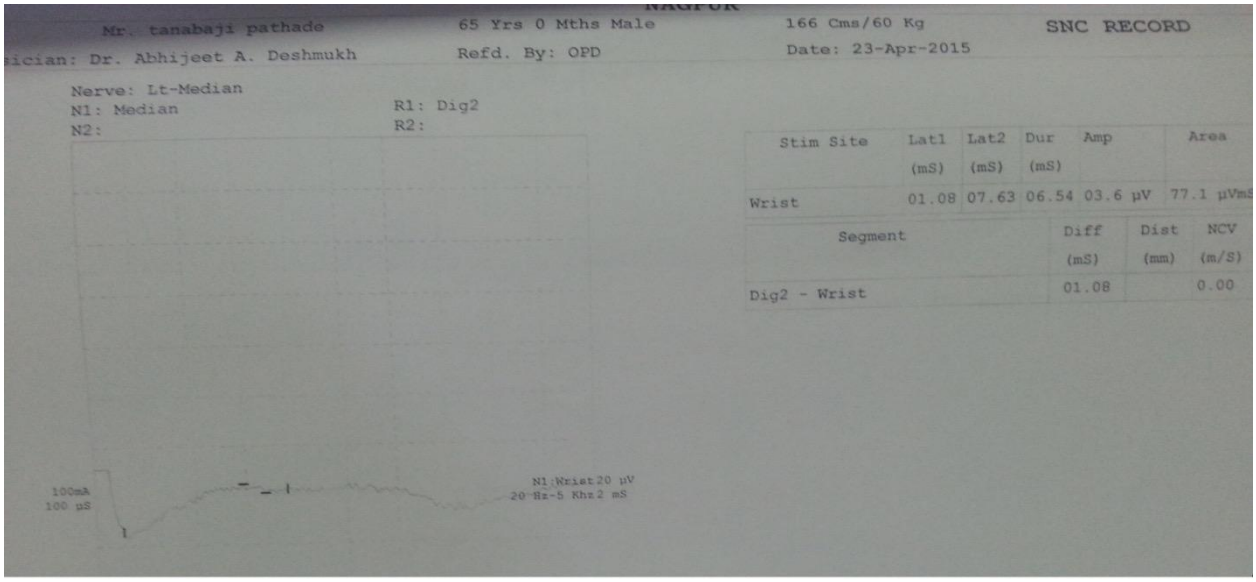
Nerve: Rt- Peroneal

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Ankle	23.23	12.19	1.0 mV	
2:Knee	35.31	10.42	0.7 mV	29.80

Nerve: Lt- Peroneal

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Ankle	8.54	18.44	0.7 mV	
2:Knee	17.29	18.85	0.6 mV	41.14

Pic : Motor Nerve conduction Velocity



Pic 2: Sensory Nerve Conduction Velocity Of left Median Nerve

Based on clinical features with motor and sensory involvement, laboratory findings and electrophysiologic investigation, this patient was diagnosed with AMSAN subtype of GBS (4). Since the patient presented to us after two weeks of progression of symptoms and his SBC (Single Breathe Count) was 20 he was not given IV Immunoglobulin and treated conservatively.

DISCUSSION

Acute motor and sensory axonal neuropathy (AMSAN) is characterized by acute onset of distal weakness, loss of deep tendon reflexes, and sensory symptoms. AMAN and AMSAN, axonal subtypes of GBS, can be preceded by *C. jejuni* enteritis [2].The pathological features that differentiate AIDP from AMAN and AMSAN is that the macrophages invade the space between the Schwann cell and axon, leaving the myelin sheath intact [2]. Griffin et al. [5] proposed the attractive hypothesis that AMAN and AMSAN are part of the spectrum of a single type of immune attack on the axon, but the relationship between AMAN and AMSAN has yet to be clarified. The AMSAN cases differ from the AMAN pattern of GBS in terms of slow recovery, in addition to sensory fiber involvement, but the pathologies are very similar [6]. The number of GBS-subtype AMSAN cases is very small (< 10% of AMAN cases) [7]. In this case, clinical, serological, and electrophysiological studies suggested a diagnosis of GBS-subtype AMSAN.

The patient was treated symptomatically and the patient improved gradually. At present the patient is doing well and is under our follow up.

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