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Case Report

Discrepancy between clinical symptoms and electrodiagnostic examination in Guillain Barre syndrome: a case report

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ABSTRACT

Guillain-Barré syndrome (GBS) is an autoimmune disease that targets the peripheral nerves. This case report presents an atypical GBS case with purely motor symptoms but mixed axonal and demyelinating lesions. A 35-year-old man presented with complaints of weakness in four extremities for 2 days before being admitted to the hospital. Four days prior, patient vomited. Motor strength in upper and lower extremities was 3334/4333 and 2334/4332, respectively. Physiological and pathological reflexes were absent in all four extremities. Sensibility was within normal limits. Cerebrospinal fluid (CSF) analysis revealed albumin-cytologic dissociation. Nerve conduction velocity (NCV) examination indicated motor polyneuropathy in 3 extremities, with axonal and demyelinating lesions. Patient was diagnosed with GBS, acute inflammatory demyelinating polyneuropathy (AIDP) subtype. AIDP is the most common subtype of GBS. The patient in this case only had motor symptoms which could occasionally happen in AIDP. However, NCV studies revealed axonal and demyelinating lesions. This discrepancy could be due to secondary axonal degeneration that had been reported in demyelinating neuropathies. Initial mixed pattern had also been reported to evolve into other subtypes during serial electrodiagnostic evaluations. The discrepancy can complicate the diagnosis and further observation may be needed. Patient in this case report improved spontaneously with supportive treatment. GBS is a complex disease to diagnose and manage due to its heterogeneous clinical presentation and variable prognosis. This case report presents an AIDP-subtype GBS patient with purely motor symptoms but mixed demyelinating and axonal lesions. Further electrodiagnostic examination might be needed to establish a diagnosis.

Keywords: Guillain Barre syndrome, Electrodiagnostic, AIDP

INTRODUCTION

Guillain-Barré syndrome (GBS) is an autoimmune disease that targets the peripheral nerves, characterized by symptoms of motor polyneuropathy.[1,2] Globally, the incidence rate is approximately 0.4 to 2 per 100,000 people.[2] GBS primarily affects the myelin sheath and causes symptoms such as muscle weakness, tingling sensation, and, in severe cases, paralysis. There are numerous GBS-related infections, predominantly respiratory and gastrointestinal diseases. Approximately 70% of patients report a history of an infectious disease occurring anywhere from 1 week up to 6 months before the manifestation of GBS symptoms. CSF analysis typically reveals albumin-cytologic dissociation in 90% cases.[1-3] This case report presents an atypical GBS case with purely motor symptoms but mixed axonal and demyelinating lesions in NCV studies.

CASE REPORT

A 35-year-old man of Javanese descent presented with complaints of weakness in four extremities for 2 days before being admitted to the hospital. The weakness began in the toes when patient was working and progressively ascended to limbs and arms. Weakness in limbs and arms

occurred simultaneously and did not start from the limbs. Four days prior admission to the hospital, patient experienced vomiting of fluid and some food. The weakness was not exacerbated by activity, and there was no fluctuation in its severity. Other symptoms such as radiating pain, headache, dizziness, fever, sore throat, cough, runny nose, diarrhea, tingling sensation, and numbness were all denied. Similar symptoms occurred in 2005, which improved spontaneously within 1 week.

Physical examination revealed a moderately ill general appearance, with a Glasgow coma scale (GCS) of E4V5M6. His vital signs were: blood pressure 120/80 mmHg, heart rate 77×/minute, respiratory rate 20x/minute, temperature 36.8°C, oxygen saturation 98% on room air. A general physical examination revealed no abnormalities. Neurological examination discovered no signs of meningeal irritation or cranial nerves palsy. Motor strength in upper and lower extremities was 3334/4333 and 2334/4332, respectively. Physiological reflexes were absent in all four extremities, with no pathologic reflexes observed. Sensibility was within normal limits.

Initial laboratory tests discovered a normal sinus rhythm on ECG. The complete blood count was: hemoglobin 15.5 g/dL, hematocrit 46.8%, leucocyte: 8,450/µL, platelet: 246,000/µL, erythrocyte: 5.61×10^6/µL and random blood glucose 117 mg/dL. CSF analysis revealed albumin-cytologic dissociation. NCV examination indicated motor polyneuropathy in 3 extremities, with both axonal and demyelinating lesions, characterized by conduction block and temporal dispersion (Table 1 and Figure 1).

Based on patient's history, physical exam and supporting diagnostic results, diagnosis of GBS was made, AIDP subtype with suspected secondary axonal involvement.

Table 1: Nerve conduction studies.

Site	L Lat (ms)	R Lat (ms)	L-R Lat (ms)	L Amp (µV)	R Amp (µV)	L-R Amp (%)	Site 1	Site 2	L Vel (m/s)	R Vel (m/s)	L-R Vel (m/s)
Anti sensory left/right comparison											
Median anti sensory (2nd Digit)											
Wrist		3.0			48.1		Wrist	2nd digit		50	
Saphenous anti sensory (Ant Med Mall)											
14 cm		3.4			6.4		14 cm	Ant Med Mall		36	
Sup peron anti sensory (Ant Lat Mall)											
14 cm	3.9			7.8			14 cm	Ant Lat Mall	29		
Ulnar anti sensory (5th digit)											
Wrist		2.6			83.7		Wrist	5th digit		42	
Ortho sensory left/right comparison											
Sural ortho sensory (Lat Mall)											
Calf	2.8	3.0	0.2	12.7	4.3	66.1	Calf	Lat Mall	41	39	2
Motor left/right comparison											
Median motor (Abd Poll Brev)											
Wrist	13.8	6.2	7.6	0,0	2,5	100.0					
Peroneal motor (Ext Dig Brev)											
Ankle	11.9	8.4	3.5	1.1	3,5	68.6	B Fib	Ankle	15	23	8
B Fib	37.2	24.1	13.1	0.7	0.3	57.1					
Tibial Motor (Abd Hall Brev)											
Ankle	11.6	11.3	0.3	1.0	0.8	20.0	Knee	Anke	16	25	9
Knee	37.2	29.4	7.8	0.4	0.1	75.0					
Ulnar motor (Abd Dig Minimi)											
Wrist		4.5			1.3		B elbow	Wrist		32	
B elbow		12.7			0.6		A elbow	B elbow		22	
A elbow		18.1			0.1						

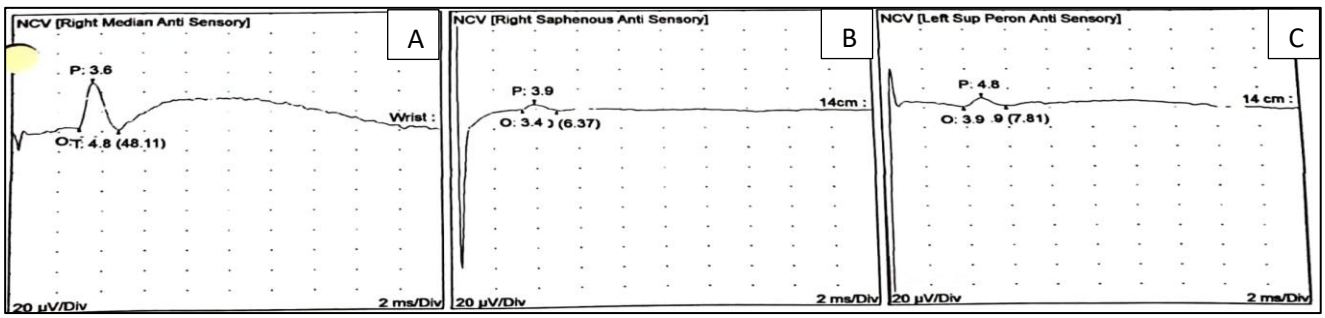

Figure 1 (A-C): Nerve conduction velocity.

DISCUSSION

GBS is an autoimmune polyneuropathy that causes involving progressive, symmetrical, ascending muscle weakness affecting more than 2 extremities. It is often areflexic, with or without sensory, autonomic, and brain stem involvement. Weakness tends to be more prominent in the limb muscles than in arm muscles, and there is typically no fever during early stages of nerve symptoms. Cranial nerve involvement may affect the airway, facial muscles, eye movements, and swallowing. Symptoms typically begin with numbness and tingling sensations in the feet.[1-4] GBS can be classified into various subtypes (Table 2), with the most common subtype being AIDP variant.[1,2]

Table 2: GBS classification.

Classification	Other Variants
Acute inflammatory demyelinating	Miller Fisher syndrome
Polyneuropathy (AIDP)	Bickerstaff Brainstem encephalopathy
Acute motor axonal neuropathy (AMAN)	Pharyngeal-cervical-brachial variant
Acute motor and sensory axonal neuropathy (AMSAN)	Multiple cranial neuropathy variant
	Facial diplegia with paresthesia
	Acute paraparesis
	Acute pandysautonomia

A diagnosis of AIDP requires at least the following signs and symptoms: (1) progressive weakness in both arms and legs (often beginning in the lower extremities); (2) hyporeflexia or areflexia; and (3) other additional criteria that further support the diagnosis: worsening of symptoms reaching their peak <28 days, a relatively symmetric neurological deficit pattern, cranial nerve palsy (especially bilateral facial muscle weakness), autonomic nerve dysfunction, a history of an upper respiratory tract infection or gastrointestinal disturbance, albumin-cytologic dissociation in CSF (increase in protein without corresponding increase in cell count, <10 cell/mm^3), and typical electrodiagnostic findings matching GBS criteria. In this case, the patient presented with bilateral extremities weakness that was progressive, symmetrical, and ascending. Decrease in motor strength and areflexia were observed in all extremities without sensory disturbances. CSF examination revealed albumin-cytologic dissociation. Based on these findings, AIDP was deemed to be the most likely diagnosis.

NCV studies were performed by placing the recording electrodes on specific muscles (for motor NCV) or nerves (for sensory NCV), while electrode stimulator was positioned over the examined peripheral nerve. This stimulation generates an action potential, referred to as the compound muscle action potential (CMAP) in motor nerves and sensory nerve action potential (SNAP) in sensory nerves.[5,6] CMAP typically has a biphasic wave pattern, starting with a negative deflection above the baseline. Generally, nerve conduction pattern in polyneuropathy can be categorized into axonal, demyelination, or a mix of both. In a demyelinating lesion, there is a decrease in amplitude due to conduction block.[5] SNAP reflects dorsal ganglion and all sensory axon function. In lesions involving dorsal ganglion and sensory nerve axons, there will be a decrease or loss of SNAP amplitude. Although the patient complained of sensory disturbance symptoms, SNAP would remain normal in central or radiculopathy lesion. SNAP examination will show abnormalities in ganglionopathy, plexopathy, or axonal neuropathy.[6]

In this case, NCV studies revealed motor polyneuropathy with both axonal and demyelinating lesions, as well as conduction block and temporal dispersion. These results meet the electrodiagnostic criteria for AIDP, including motor NCV<70% of the lower normal limit and distal motor latency>130% of the upper normal limit in more than 2 nerves. Additional supporting signs include distal CMAP duration>120% of the upper normal limit, a proximal-to-distal CMAP duration ratio>130%, and F wave latency>120% of the upper normal limit.[1] Notably, aside from the prominent demyelinating results, results also indicated features of axonal lesion. The distal CMAP amplitude was ≤120% of the lower normal limit, indicating conduction block. The patient in this case only had motor symptoms which could occasionally happen in AIDP. However, AIDP normally only involves demyelinating lesions while NCV studies revealed both axonal and demyelinating lesions. This discrepancy could be due to secondary axonal degeneration that had been reported in demyelinating neuropathies.[7] Initial mixed pattern (both demyelinating and axonal lesions) had also been reported to evolve into other subtypes during serial electrodiagnostic evaluations.[8] Hence, further evaluation might be warranted.

The treatment of GBS is determined by its disability scoring. In general, poorer prognosis are associated with individuals older than 40 years, a history of diarrhea, *Campylobacter jejuni* infection, rapid and severe disease progression, severe disability at the peak of progression, and axonal types, particularly AMSAN.[1] In this patient, disability score was 3, indicating the patient's ability to walk up to 5 meters using the assistance of tools. The patient did not experience severe respiratory distress, so he was treated and observed for worsening symptoms in a normal room. According to the GBS treatment algorithm, the patient should have received pharmacologic treatment with intravenous immunoglobulin (IVIG) at 0.4 g/kg body weight for 5 days or plasmapheresis 200-250 ml/kg body weight per session (5 sessions in 2 weeks).[1] However, the patient in this case report improved spontaneously without immunotherapy. The use of immunotherapy in patients with limited mobility remains controversial but may be considered if the patient experiences rapid and severe

disease progression including autonomic dysfunction, bulbar paralysis, and respiratory insufficiency.[9-12]

CONCLUSION

GBS is a complex disease to diagnose and manage due to its heterogeneous clinical presentation and variable prognosis. Therefore, it would be best not to postpone treatment. While the prognosis is generally good, some patients may experience mild to moderate residual symptoms or even persistent neurological deficits. This case report presents an AIDP-subtype GBS patient with purely motor symptoms but mixed demyelinating and axonal lesions. The discrepancy can complicate the diagnosis and further electrodiagnostic examination may be needed.

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