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Case report: Guillain-Barré Syndrome presentation mimicking stroke. An atypical presentation

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ABSTRACT

We herein report an unusual case of a 58-year-old man with polycythemia presented with sudden right leg and right arm weakness. He was treated for a stroke but continued to worsen, leading to quadriparesis. He was referred to the emergency department after experiencing progressive weakness in all four limbs for five days. No prior history of fever, cough, chest pain, diarrhea, backache, or trauma was found. The patient had normal neurological examination, MRC scores, and bilateral weak hand grips. Sensory examination was normal. The patient had normal blood tests, electrolytes and brain MRI. CSF examination revealed protein an albumin-cytological dissociation pattern. An electrodiagnostic study showed evidence of acute motor axonal polyneuropathy. The patient received IVIg for five days, and symptoms improved significantly.

Keywords: GBS, polycythemia, albumin-cytological dissociation pattern, electrodiagnostic, polyneuropathy

INTRODUCTION

Guillain-Barré syndrome (GBS) is a diverse disorder defined by immune-mediated peripheral neuropathies with acute onset and fast progression of weakness, hypo, or areflexia [1]. It encompasses at least five different disorders that result in systemic motor paralysis and are distinguished by increased levels of protein in the cerebrospinal fluid but normal cell counts. Mostly the weakness is symmetrical, ascending type, starting distal to proximally from lower limb to upper limbs. These symptoms are frequently preceded by abnormal sensations, which often take the form of tingling in the feet or hands or even pain that typically begins in the back or legs [2]. In some situations, the syndrome may present non-classic symptoms that may challenge initial diagnosis and management.

Herein, we describe an unusual presentation of GBS showing a stroke-like manifestation.

CASE REPORT

A 58-year-old man with a history of polycythemia initially presented to a local hospital with sudden right leg weakness followed by right arm weakness. He was treated there as having a possible acute ischemic stroke and was admitted for a stroke workup, which came unremarkably. The patient's weakness continued to worsen, eventually leading to quadriparesis. The patient was referred to the emergency department of our hospital after experiencing progressive weakness in all four limbs for five days. Additional investigation and examination of the patient revealed no prior history of fever, cough, chest pain, diarrhea, backache, or trauma. He also had complete control of his sphincters. The mental status examination was normal on neurological examination with no cranial nerve palsy. Neck flexion and extension power were medical research council (MRC) scores of 5/5. He had bilateral weak hand grips, and feet dorsiflexion was MRC of (0/5), plantar flexion was (2/5), knee extension (3/5), and hip flexion/extension (5/5). Plantar reflexes were down go-

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ing bilaterally, and the deep tendon reflexes (DTRs) were generally 1. The sensory examination, including superficial and deep sensory (proprioception, joint position), was normal.

A routine blood test revealed a normal complete blood count (CBC), and electrolytes. The results of the brain MRI with DWI and the whole spinal MRI were normal. CSF examination revealed protein: 108.54 mg/dl, glucose: 4.72 mg/dl, and cells: 3 (an albumin-cytological dissociation pattern). The electrodiagnostic study showed evidence of acute motor axonal polyneuropathy (Table 1). A diagnosis of acute motor axonal polyneuropathy (AMAN) was made based on the abovementioned clinical, CSF, and electrodiagnostic investigations. In addition to supportive care and rehabilitation, the patient received IVIg at a dose of 0.4 gr/kg/day for five days, and his symptoms were determined to have significantly improved.

TABLE 1. Electrodiagnostic study of the patient

Sensory Nerve Conduction Study				
Nerve		**PL (ms)**	**Amp (μV)**	**NCV (m/s)**
Median L		4.1	47	49
Ulnar L		3.8	31	49
Sural R		3.5	14	50
SPN R		4.3	4	43
Sural L		3.5	24	47
SPN L		4.5	4	39
Motor Nerve Conduction Study				
Nerve		**Latency (ms)**	**Amp (mV)**	**NCV (m/s)**
Median L	Wrist	4.4	1.5	
	Elbow	9.4	1.6	50
Ulnar L	Wrist	5.5	0.4	
	Below Elbow	10.5	0.4	52
Peroneal R	Ankle	5.9	0.1	
	Fibula (head)	13.1	0.1	42
Tibial R	Ankle	8.9	0.2	
	Popliteal fossa	18.7	0.2	41
Peroneal L	Ankle	9.6	0.2	
	Fibula (head)	16.8	0.1	42
Tibial L	Ankle	5.1	0.3	
	Popliteal fossa	17.3	0.3	33

PL: proximal latency, Amp: amplitude, NCV: nerve conduction velocity, ms: milliseconds, μV: microvolt, mV: millivolt, m/s: meter per second

DISCUSSION

Classically, GBS often presents with weakness and areflexia within 1-4 weeks of the preceding illness. The main symptoms are hypo/areflexia, but normal reflexes have been observed in a few rare cases, usually in the AMAN variant. Acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), and acute inflammatory demyelinating polyradiculoneuropathy are three of those forms that predominantly involve the motor system (AIDP) [3]. The other types are Miller-Fisher syndrome and acute pandysautonomic neuropathy. For medical professionals, an unusual presentation presents a diagnostic problem.

Hemiparesis or hemiplegia is very rarely the first symptom experienced by GBS patients [4], and very few of them had symptoms that were thought to be acute stroke [5].

The traditional clinical indicators, an electrodiagnostic test, and an examination of the CSF fluid are used to make the diagnosis [6,7]. Identifying the precise subtype of the patient is crucial because axonal kinds (AMAN and AMSAN) often have worse prognoses. AMAN is purely motor and is more prevalent in Asian nations; these patients hardly ever have normal reflexes, and hemiplegia and paraplegia are unusual variations of the GBS that might occur [8]. Recently, a case of GBS, with the surprising diagnosis of acute hemiparesis was described by Castrodad-Molina et al. Likewise, a young man with a rare manifestation of GBS that mimicked a stroke was described by Mohamed Sheikh Hassan, et al [9]. Three cases of abrupt onset of acute stroke-like symptoms with a final diagnosis of hyperacute GBS were reported by de Montaudouin et al [10].

The precise etiology and mechanism of acute hemiplegia in GBS are unknown. In our case, the patient initially reported signs of hemiplegia, which resembled a cerebrovascular stroke. Acute neuropathy like GBS should always be suspected when stroke-like symptoms are present and brain imaging results are negative. After being ruled out by a brain MRI with diffusion sequence, CSF analysis, and nerve conduction studies, the progression to quadriplegia required further evaluation and inquiry, which eventually required the unexpected diagnosis of Acute Motor Axonal polyneuropathy.

CONCLUSION

In conclusion, when patients appear atypical, the diagnosis of GBS is commonly missed. We hope this case report will raise awareness of the possibility

that GBS may initially emerge with symptoms resembling a stroke. Clinicians must pay close attention and diagnose accurately as soon as they can.

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