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Asymmetric limb weakness in Guillain-Barré syndrome: Three case reports

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Abstract

BACKGROUND

Guillain-Barré syndrome (GBS) is an autoimmune-mediated peripheral neuropathy characterized by symmetric weakness. Asymmetric weakness in GBS is uncommon and may be easily confused with other differential diagnoses. We herein present three cases of asymmetric GBS and review the literature on this atypical subtype of GBS in order to describe the characteristics of asymmetric GBS and to provide experience for clinicians.

CASE SUMMARY

Different from patients in the previous reports, our patients showed persistent asymmetric limb weakness from the onset to recovery phase. All three patients were serologically positive for antecedent infections. Two of the three cases had IgG antibodies against ganglioside GM1. Two patients received immunotherapy including intravenous immunoglobulin and plasma exchange, while one patient received only supportive treatment. Autoantibodies against gangliosides, asymmetry of congenital development of blood-nerve barrier and limb use may contribute to the development of asymmetric limb weakness in GBS.

CONCLUSION

Asymmetric GBS may be a rare clinical variant and should be considered when a patient develops acute and progressive asymmetric limb weakness. The differences in clinical features and prognosis between asymmetric GBS and classic

GBS deserve further investigation in a large study.

Key Words: Guillain-Barré syndrome; Asymmetric limb weakness; Autoantibodies; Blood-nerve barrier; Case report

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Core Tip: Atypical Guillain-Barré syndrome (GBS) with asymmetric limb weakness was rarely reported, which is easily confusing the diagnosis by clinicians. We herein present three patients diagnosed as GBS who suffered persistent asymmetric limb weakness from onset to recovery phase, two of which received immunotherapy in timely and had a good prognosis. Asymmetric blood-nerve barrier, ganglioside distribution or limb use may cause asymmetric GBS. To differentiate asymmetric GBS is important for an early treatment, which can lead to a good patient outcome and early recovery.

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INTRODUCTION

Guillain-Barré syndrome (GBS) is one of the most common causes of acute flaccid paresis with significant clinical heterogeneity[1]. At disease onset, most patients present with typical features of GBS such as symmetric tetraparesis, while some patients only present with incomplete variants, such as paraparetic or pharyngeal-cervical-brachial forms[2,3]. Recently, some atypical forms, such as asymmetric cranial nerve palsies, have been reported in patients with GBS[4]. However, asymmetric limb weakness in GBS is uncommon and may be confused with other conditions such as stroke. We herein describe three patients diagnosed with GBS who suffered from asymmetric limb weakness, review the literature and hypothesize the possible underlying mechanisms.

CASE PRESENTATION

Chief complaints

Case 1: A 56-year-old male worker presented with distal weakness of right upper and lower limb for five days and distal weakness of left upper and lower limb for two days.

Case 2: A 59-year-old male farmer presented with right upper and lower limb weakness for five days and left upper and lower limb weakness for four days.

Case 3: A 36-year-old male farmer presented with right upper and lower limb weakness for two days and left upper and lower limb weakness for one day.

History of present illness

Case 1: The patient had a history of pneumonia two weeks previously. At disease onset, he complained of right distal upper and lower limb weakness, loss of pain and temperature sensation in the right hand and foot, and could not walk independently. Three days later, the limb weakness and numbness extended to the left distal upper and lower limb, which continued to worsen until day 5.

Case 2: The patient denied antecedent infections. His clinical symptoms started five days ago with right upper and lower limb weakness, which had been extended to the left upper and lower limb on day 2 after onset. He complained of significant loss of pain and temperature sensation in bilateral hands and feet.

Case 3: The patient had a history of gastroenteritis 10 d before the onset of limb weakness. Two days ago, he complained of right upper and lower limb weakness and numbness, which was initially diagnosed with cerebral infarction at another hospital. The following day, his clinical symptoms progressed and the limb weakness and numbness extended to the left right upper and lower limb.

History of past illness

The three patients had no particular past history.

Personal and family history

The three patients had no particular individual or family history.

Physical examination

Case 1: The neurological examination revealed normal cranial nerves, 5/5 Medical Research Council (MRC) grade on proximal four limbs, 3/5 MRC on the right distal limb and 4/5 on the left distal limb. The tendon reflex of the four limbs was absent.

Case 2: Neurological examination on admission revealed bilateral distal and proximal limb weakness (for the distal, 1/5 MRC on the right and 2/5 MRC on the left; for the proximal, 3/5 MRC on the right and 4/5 on the left). Sensory examinations showed significant loss of pain and temperature sensation in bilateral hands and feet. The tendon reflex of four limbs was absent.

Case 3: On admission, neurological examination revealed weakness of both proximal and distal limbs, 3/5 MRC on bilateral proximal, 2/5 MRC on distal upper limbs, 2/5 MRC on the left distal lower limb and 1/5 on the right distal lower limb, deep tendon reflexes were decreased and he had loss of pain and temperature sensation in bilateral hands and feet.

Laboratory examinations

Case 1: Cerebrospinal fluid (CSF) analysis on day 10 showed normal cell counts (normal range, 0 to 8/mL) and increased protein concentration of 1.421 g/L (normal range, 0 to 0.45 g/L). Serological analysis of IgG antibodies against GM1, GM1b, GalNAc-GD1a, GD1a, GD1b, GQ1b, and GT1a were negative. Serological testing for IgM antibodies against influenza A virus and *Mycoplasma pneumoniae* were positive.

Case 2: CSF analysis on day 6 showed normal cell counts and increased protein levels (0.987g/L). IgG antibodies against GM1 were positive. Serological testing for IgM antibodies against influenza A virus was positive.

Case 3: CSF analysis showed normal cell counts and increased protein concentration of 0.483 g/L on day 3 after onset. IgG antibodies against GM1 were positive. Serological testing for IgM antibodies against *Campylobacter jejuni* was positive.

Imaging examinations

Case 1: A nerve conduction study (NCS) on day 10 showed that the distal latencies of the median, ulnar and peroneal nerves were significantly increased, conduction velocities of the median, ulnar and tibial nerves were reduced and there was temporal dispersion of the median and tibial nerves. NCS results suggested demyelination. Magnetic resonance imaging (MRI) of the brain was normal. The spinal MRI was not performed.

Case 2: MRI of the spine was normal. He refused to undergo a NCS.

Case 3: A NCS on day 3 showed that the amplitudes of the median, ulnar, tibial and peroneal nerves were significantly reduced.

FINAL DIAGNOSIS

The three cases were diagnosed with GBS.

TREATMENT

Case 1: The patient received intravenous immunoglobulin which was initiated on day 10 at 2 g/kg/day for 5 d.

Case 2: The patient opted for supportive treatment due to cost.

Case 3: He was treated with plasma exchange (PE) every two days.

OUTCOME AND FOLLOW-UP

Case 1: On day 16, his limb weakness and numbness started to improve. On day 17, he was discharged. A follow-up at six months after onset showed 5/5 MRC on proximal four limbs, residual mild weakness of distal upper limbs (5/5 MRC on the left, 4/5 MRC on the right) and lower limbs (4/5 MRC bilateral).

Case 2: His clinical symptoms progressed and reached the nadir on day 9 after onset and he developed urinary retention. The MRC on four proximal limbs was 2/5, left distal limb muscle strength was 1/5 and right distal limb muscle strength was 0/5. The patient was discharged and returned to a local hospital on day 11. Six months later, a follow-up of this patient showed 2/5 MRC on four proximal limbs and 2/5 MRC on the left distal limb and 1/5 MRC on the right distal limb.

Case 3: His clinical signs showed no response to the first session of PE and symptoms progressed on day 4 after onset. The neurological examination showed 2/5 MRC on the left proximal limb and 3/5 on the right proximal limb, 1/5 MRC on the left and 2/5 MRC on the right distal lower limbs, and 1/5 on distal bilateral lower limbs. His sensory deficits in bilateral feet rose to bilateral legs. PE treatment proceeded as planned. On day 10 after onset, his weakness and numbness started to improve. Following PE, he was transferred to the rehabilitation ward for a course of electrical stimulation and physical rehabilitation. He was discharged on day 28. A follow-up at six months after onset showed asymmetric weakness on upper (4/5 MRC on the right and 3/5 on the left) and lower limbs (3/5 MRC on the right and 2/5 on the left).

DISCUSSION

Typical GBS is a mono-phasic polyneuropathy with acute onset symmetric cranial or limb weakness[5]. Asymmetric deficits, especially asymmetric limb weakness is easily confused with other neurological conditions such as stroke, demyelinating myelopathy, acute-onset chronic inflammatory demyelinating polyneuropathy[6], and beriberi neuropathy[7]. Although a study from Israel reported that asymmetric weakness accounted for 23% of atypical clinical findings of GBS in children[8], atypical GBS in adults with asymmetric limb weakness is rarely reported. Weakness or paresthesia in GBS may begin with symptoms in one limb, but most of the asymmetric symptoms at onset are transient and will progress to symmetric weakness at nadir[9,10]. Chi *et al*[11] reported a patient with axonal GBS who presented with acute onset of left hemiplegia and left sublingual nerve palsy mimicking stroke, of which the hemiplegia further progressed to tetraparesis within a few days. A Dutch study reported four patients who had asymmetric GBS[12], and three of these patients progressed to symmetric GBS within one week. Being different, both the case presented by Logullo *et al*[13] and our patients showed persistent asymmetric limb weakness starting from the acute phase until the recovery phase. In this report, we review the literature and summarize the clinical presentation of asymmetric GBS, including a case reported previously[13] and our cases reported in this paper (Table 1).

Although the exact mechanism of asymmetric deficits in GBS remains unclear, current evidence suggests a potential association between anti-ganglioside antibodies and asymmetric deficits in GBS. Stork *et al*[14] previously suggested that auto-antibodies against asialo-GM1 were associated with more pronounced motor deficits and asymmetry. Kim and Yuki[15] reported an unusual patient with hemiparetic GBS who had preceding gastroenteritis and anti-GD1a antibodies. In our patients, two out of three had autoantibodies against GM1. Anti-GM1 IgG antibodies binding to the nodal axolemma, which locates the GM1 and voltage-gated sodium channels, leads to the formation of membrane attack complex and disruption of sodium channels, which causes axonal impairment and nerve-conduction failure[16]. Binding of circulating antibodies onto the peripheral nerve myelin or axolemma relies on a rich blood supply and discontinuity of the blood-nerve barrier (BNB). Generally, the BNB along the nerve fiber is inhomogeneous and the distal and proximal are selectively more vulnerable to injury[17]. Manousakis *et al*[18] reported a patient with melanoma who developed multifocal and asymmetric polyradiculoneuropathy after ipilimumab treatment. The asymmetry of congenital development of BNB including the vascular endothelium permeability or asymmetric disruption of the BNB may be one of the underlying mechanisms leading to asymmetric limb weakness in GBS and antibodies-related peripheral neuropathy. Furthermore, the asymmetric distribution of the gangliosides may also cause the difference in limb involvement on both sides of patients with GBS. Finally, body position or limb use has also been suggested as a possible cause, which influences the initial distribution of weakness during crucial moments of influx of inflammatory factors into nerves[19]. All of our three patients were right-handed and their first clinical presentation at onset was all right-sided weakness. At nadir, the first two patients had more serious limb weakness on the right while the third patient had more serious limb weakness on the left. Our cases suggest a possible correlation between limb use and asymmetric GBS, which deserves further investigation.

Table 1 Clinical features of patients with asymmetric Guillain-Barré syndrome

Characteristics	Report by Logullo <i>et al</i> [13]	Present report		
	Case 1	Case 1	Case 2	Case 3
Age (yr)/sex	14/M	56/M	59/M	36/M
Antecedent illness	Gastroenteritis	Pneumonia	-	Gastroenteritis
At entry				
GBS disability score	3	2	4	4
MRC sum-score				
Left limb weakness	NA	26	16	15
Right limb weakness	NA	22	10	12
Sensory deficits	+	+	+	+
Tendon reflex	Normal	Areflexia	Areflexia	Hyporeflexia
Bulbar palsy	-	-	-	-
Ataxia	-	-	-	-
Cranial nerve involvement	-	-	-	-
Albuminocytological dissociation in CSF	+	+	+	+
Cerebral magnetic resonance imaging	None	-	-	-
IgG antibodies to	GM1	-	GM1	GM1
Nerve conduction studies	Axonal	Demyelination	NA	Axonal
Treatment	IVIg	IVIg	ST	PE
At nadir				
Days between onset and nadir	16	5	9	4
GBS disability score	4	2	4	4
MRC sum-score				
Left limb weakness	NA	26	8	8
Right limb weakness	NA	22	4	12
Sensory deficits	+	+	+	+
Tendon reflex	Areflexia	Areflexia	Areflexia	Areflexia
At six months after onset				
GBS disability score	3	2	4	4
MRC sum-score				
Left limb weakness	NA	29	12	15
Right limb weakness	NA	26	8	21
Sensory deficits	+	+	+	+
Tendon reflex	NA	Hyporeflexia	Areflexia	Hyporeflexia

GBS: Guillain-Barré syndrome; MRC: Medical Research Council; CSF: Cerebrospinal fluid; IVIg: Intravenous immunoglobulin; PE: Plasma exchange; ST: Supportive treatment; NA: Not available.

CONCLUSION

To sum up, asymmetric GBS is challenging the diagnosis criteria of GBS. To differentiate asymmetric GBS, especially the hemiplegia at onset with other similar conditions such as stroke, is important for an early treatment of IVIg or PE, which can lead to a good patient outcome and early recovery. Additionally, further study is necessary to investigate whether asymmetric GBS can be taken as a variant of GBS.

FOOTNOTES

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