Case Report

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Guillain-Barré syndrome and Low back pain: two cases and literature review

Abstract: Purpose. To describe the clinical, electrophysiological, and lumbar magnetic resonance imaging (MRI) features of two cases of atypical Guillain-Barré syndrome (GBS). Methods We reported two GBS variant cases with initial and prominent symptoms of low back pain. We analysed their clinical, electrophysiological, and lumbar MRI features. Results Two patients with GBS reported low back pain as the initial and prominent symptom, which was not accompanied by limb weakness. The electrophysiological study showed abnormal F-waves in the common peroneal and tibial nerves, and acute polyradiculoneuropathy in the cauda equina. Examination of the cerebrospinal fluid (CSF) showed albuminocytologic dissociation. Serum was positive for GQ1b-IgM antibodies. Lumbar MRI showed gadolinium enhancement of the nerve roots and cauda equina. A standard regime of intravenous immunoglobulin markedly alleviated the low back pain. Conclusions Low back pain caused by GBS should be differentiated from other diseases. This initial or early prominent symptom may delay the diagnosis of GBS; therefore, it is important to conduct a detailed electrophysiological, CSF, and gadolinium-enhanced lumbar MRI analysis.

1 Introduction

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy typically following an upper respiratory tract or gastrointestinal infection. A diagnosis of GBS is made based on the clinical presentation of perineuropathy, albuminocytologic dissociation of the cerebrospinal fluid (CSF), and electrodiagnostic findings of polyradiculoneuropathy. Pathological and electrophysiological features can be used to classify GBS into acute inflammatory demyelinating polyradiculoneuropathy (AIDP) [1, 2], acute motor axonal neuropathy (AMAN) [3–5], or acute motor and sensory axonal neuropathy (AMSAN) [6]. Other distinctive variants include Fisher's syndrome, radicular neuritis, and facial diplegia [7–9].

Over 50% of GBS patients experience severe pain; however, this symptom is often overlooked because the most attention is given to limb weakness. Various types of pain have been described in GBS [10–17]. Among them, low back pain is a common symptom but it can cause diagnostic and therapeutic difficulties when patients experience severe pain in the absence of limb weakness at the early stage of the disease. Here, we present two GBS cases with low back pain as an initial and prominent symptom.

2 Case report

2.1 Case 1

A 64-year-old woman was admitted to our hospital because of severe pain in the low back and extremities, and bladder dysfunction for about 20 days. Upon physical examination, the patient was alert with normal cranial

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nerves. She had reduced tendon reflexes in the lower extremities and reduced perianal touch sensation associated with hyperalgesia at both soles of foot. Lasègue's signs were positive; however, Kernig's sign was negative. Muscle power and vibration sensations were intact in the upper and lower extremities. She denied fever, infections, or diarrhoea during the previous weeks. She reported lumbar spondylosis in her past medical history.

Lumbar puncture was performed at day 21 after onset. CSF analysis revealed albuminocytologic dissociation: the nuclear cell count was 22/µl (normal range 5–10), total protein was 825 mg/L (normal range 0–450), and microorganism test was negative. The serum anti-ganglioside antibody profile was assessed using the immunodot assay [18], which revealed the presence of serum GQ1b-IgM antibodies. Lumbar magnetic resonance imaging (MRI) showed moderate gadolinium enhancement of the nerve roots and cauda equina (Fig. 1).

An electromyogram (EMG; Dantec Keypoint) was obtained at day 25 after onset. Motor nerve conduction velocity (NCV) data were obtained from the bilateral ulnar, median, and peroneal nerves. Sensory NCV data were obtained from median, sural, and peroneal nerves. F-waves were elicited in bilateral ulnar, peroneal and tibial nerves using supramaximal stimuli at the wrist and ankle for ten consecutive trials, respectively. The results are summarized in Table 1 and Table 2. F-waves were not elicited in the tibial nerve (Table 1). A needle electrophysiological study showed widespread spontaneous activity in paravertebral muscles of the lumbosacral region and anal sphincter, and neurogenic changes in the lower limb muscles (Table 2). However, NCV showed no abnormalities in upper and lower limbs (Table 1) and the needle electrophysiological study and F-waves were normal in the upper limbs. The electrophysiological study showed proximal axonal conduction failure and diffuse neurogenic changes in the cauda equina, indicating acute polyradiculoneuropathy.

Based on these diagnostic findings, a GBS variant was postulated. A standard regime of intravenous immunoglobulin was administered (0.4 g/kg/day for 5 days). The treatment markedly alleviated the low back pain and radicular pain in lower limbs. In addition, there was an improvement in bladder function. She had no symptoms on follow-up.
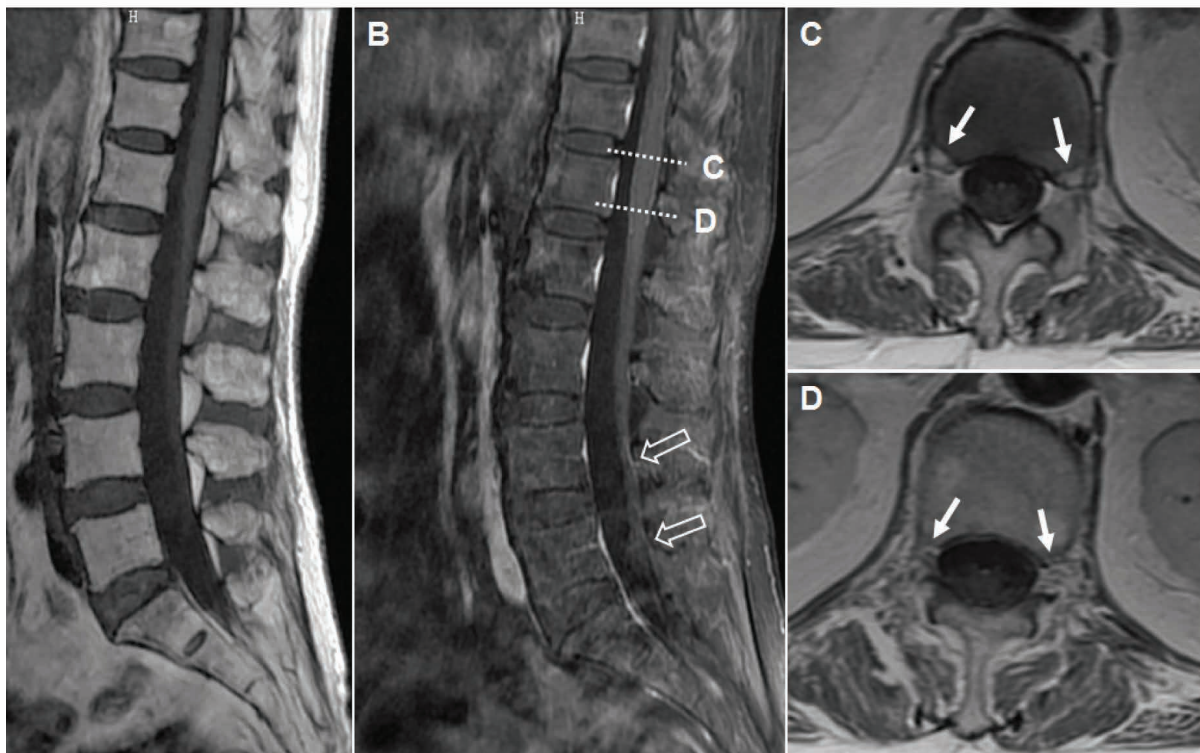

Figure 1: Lumbar magnetic resonance imaging (MRI) results of a 64-year-old woman with Guillain-Barré syndrome. Pre-contrast sagittal SE T1-weighted MRI demonstrated no abnormality of the cauda equina, conus medullaris, and dural sac (A). Contrast-enhanced fat suppressed sagittal T1-weighted MRI (B) and contrast-enhanced axial T1-weighted MRI (C-D) showed moderate enhancement of the nerve roots (solid arrows) and cauda equina (open arrows). The levels of figures C-D were shown as the dotted lines in figure B.

Table 1: Electrophysiological findings of nerve conduction examination

Case	Nerve	CMAP			SNAP			F-wave response
		Terminal latency, ms	Amplitude, mV	Conduction velocity, m/s	Terminal latency, ms	Amplitude, mV	Conduction velocity, m/s	
1	Peroneus	9.5	4.0	46.2	3.1	17	54.2	No
	Suralis				3.2	11	64	
2	Peroneus	10.8	1.5	50.0	2.9	17	56.8	Prolonged latency
	Suralis				3.7	4.5	48.3	

CMAP compound motor action potential, SNAP sensory nerve action potential

Table 2: Needle electromyography findings

Case	Parameter	Tibialis anterior		Gastroc caput median		Vastus lateralis		Add magnus		Iliopsoas	
		Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
1	Duration, ms	19.0	21.0	18.2	17.8	16.8	17.8	18.6	20.5	20.3	19.8
	Amplitude, μV	4093	1295	1339	988	1155	1475	831	1639	1654	783
2	Duration, ms	11.4	13.2	14.9	11.4	22.8	20.3	13.3	14.5	14.8	16.6
	Amplitude, μV	404	858	815	674	1451	920	368	830	1159	1320

2.2 Case 2

A 76-year-old woman presented with severe low back and extremity pain for approximately 10 days, accompanied by bladder dysfunction. She reported diarrhoea without fever before the onset of pain. On admission, neurological examination revealed a reduction in the Achilles tendon reflex associated with positive Lasègue's sign. In addition, perianal touch sensation was reduced; however, the muscle power of the upper and lower limbs was intact, with normal cranial nerves and vibration sensations. She reported ischialgia in her past medical history.

Lumbar puncture was performed at day 12 after onset. CSF results showed a mild elevated nuclear cell count of 50/μl, moderately elevated protein level of 1430 mg/l, and a negative microorganism test. The stool culture was negative. The anti-ganglioside antibody profile revealed that the serum was positive for GQ1b-IgM antibodies. Lumbar MRI examination showed moderate to marked gadolinium enhancement of the nerve roots and cauda equina (Fig. 2).

An electrophysiological study was performed at day 13 after onset. The EMG procedure was the same as that used in case 1. The results are summarized in Table 1 and Table 2. F-waves showed a prolonged latency in the common peroneal and tibial nerves (51.2 ms) (Table 1). The needle electrophysiological results showed widespread spontaneous activity in the paravertebral muscles of the lumbosacral region, and neurogenic changes in the lower limb muscles (Table 2). Two weeks later, a repeat NCV showed low amplitude compound motor action potentials (CMAP) in the left tibial nerve (Table 1). No conduction blocks were detected; however, sensory NCV showed no abnormalities (Table 1) and the needle electrophysiological study and F-waves were normal in the upper limbs. The electrophysiological study showed proximal axonal conduction abnormalities and diffuse denervation of the cauda equina, indicating acute polyradiculoneuropathy.

Based on these diagnostic findings, a GBS variant was postulated. A standard regime of intravenous immunoglobulin was administered (0.4 g/kg/day for 5 days). The treatment markedly alleviated the low back pain and radicular pain. The bladder dysfunction also obviously improved. She had no symptoms during follow-up.

Ethics Statement: The study has been complied with all the relevant national regulations, institutional policies and the tenets of the Helsinki Declaration, and has been approved by the ethics committee of Sir Run Run Shaw Hospital, School of Medicine, Zhejiang University. Informed consent has been obtained from all individuals included in this study.
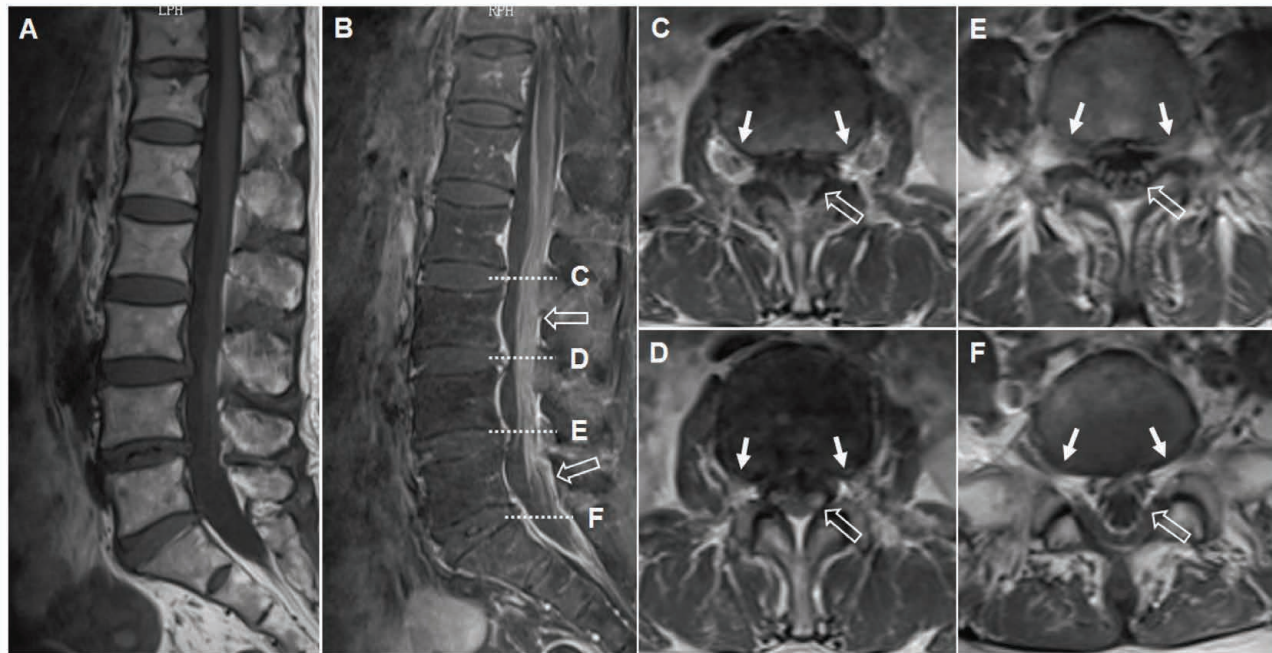
Figure 2: Lumbar magnetic resonance imaging (MRI) results of a 76-year-old woman with Guillain-Barré syndrome. Pre-contrast sagittal SE T1-weighted MRI demonstrated no abnormality of the cauda equina, conus medullaris, and dural sac (A). Contrast-enhanced fat suppressed sagittal T1-weighted MRI (B) and contrast-enhanced axial T1-weighted MRI (C-F) shows moderate to marked enhancement of the nerve roots (solid arrows) and cauda equina (open arrows). The levels of figures C-F were shown as the dotted lines in figure B.

3 Discussion

In this report, we presented two cases of low back pain in a variant of GBS where initial diagnosis was made based on neurological features; EMG features showing polyradiculoneuropathy; MRI findings showing the enhancement of the cauda equina and nerve roots; CSF findings showing albuminocytologic dissociation; and positive serum GQ1b-IgM antibodies. The severe low back pain and bladder dysfunction were markedly ameliorated following a standard regime of immunoglobulin treatment. Both patients lacked characteristic limb weakness; however, their radicular neuritis should be considered as atypical GBS. The spectrum of GBS variants may manifest as pure sensory GBS, bilateral lumbar nerve root disease, such as radicular neuritis, or may not be accompanied by limb weakness.

Moderate to severe pain is common in GBS, and is reported in 85% of patients with GBS [11]. Various types of pain have been described in GBS, including muscle, low back, radicular, and joint pain. Low back pain is pervasive in GBS and its frequency ranges from 13–62% (Table 3). The pathophysiology of low back pain in GBS has not been established and is likely to be multifactorial. One proposed explanation is entrapment neuropathy [15, 17]. Acute pain is mainly nociceptive because of the nerve root

and peripheral nerve inflammation which may activate nociceptors [15]. Pain can precede, be simultaneous with, and persist after the resolution of weakness in GBS. It is reported that pain would precede weakness in approximately one third of GBS patients [11]. Both our GBS cases showed severe low back pain as the initial and prominent symptom, without limb weakness.

Low back pain can be overlooked during the diagnostic process identifying GBS because most attention is directed towards the progression of weakness. Subsequently, low back pain, as an initial or early prominent symptom in GBS, remains a diagnostic challenge. Such cases may mimic acute spinal cord injury or nerve root compression syndrome on initial presentation and receive inappropriate spine surgery treatment and perhaps report GBS as a rare complication of spine surgery. Therefore, GBS should be taken into consideration in the differential diagnosis of low back pain.

In this report, a unique clue was the EMG finding. There were abnormal F-waves in the tibial nerve; widespread spontaneous activity in the muscles of the lumbosacral region; and neurogenic changes in the lower limb muscles in both cases. These findings revealed proximal axonal conduction failure with widespread denervation of the cauda equina, indicating acute polyradicu-

Table 3: Summary of studies on Guillain-Barré syndrome (GBS) and pain

Author	Number of patients	Subtype of pain	Pain ratio (%)
Moulin et al. [11]	55	Back and leg pain	62
		Dysesthetic extremity pain	49
		Myalgic-rheumatic extremity pain	35
		Pressure palsy (ulnar nerve)	2
		Visceral pain	20
		Headache caused by dysautonomia	2
Wilmshurst et al. [12]	27	Low back pain and/or radicular pain	33
		Neck pain	15
		Muscle pain	56
		Joint pain	15
Green et al. [13]	12	Low back pain	17
		Periarticular pain or more diffuse pain	42
Korinthenberg et al. [14]	95	Neuropathic pain	79
Ruts et al. [15][a]	39	Backache	33
		Interscapular pain	28
		Muscle pain/cramps	24
		Radicular pain	18
		Joint pain	5
		Painful par-/dysaesthesiae	18
		Visceral pain	5
Ruts et al. [16][b]	151	Low back pain	13
		Interscapular pain	10
		Extremity pain	25
		Neck pain	10
		Trunk pain	4
		Muscle pain	19
		Radicular pain	8
		Arthralgia	2
		Painful par-/dysaesthesiae	11
		Meningism	1

[a] The number of patients and the ratio for the subtype of pain are for patients with GBS who had pain within 4 weeks before randomization in the retrospective study.

[b] The number of patients and the ratio for the subtype of pain are for patients with GBS who had pain maximum of 2 weeks before onset of weakness.

loneuropathy rather than nerve root compression due to lumbar spine disease.

Diagnosing GBS with atypical clinical features or electrophysiological abnormalities remains a diagnostic problem. A gadolinium-enhanced MRI of the spine can be a helpful method in diagnosing these GBS variants.

Several reports have shown that gadolinium-enhanced MRI of the lumbar spine has demonstrated the enhancement of the cauda equina and/or nerve roots in GBS. A prospective study by Gorson KC et al [19] obtained gadolinium-enhanced lumbosacral spine MRIs in 24 consecutive patients with acute GBS. They found that 20 patients had cauda equina or nerve root enhancement with gadolinium on lumbosacral MRI, and prominent cauda equina or nerve root enhancement correlates with pain, GBS disability grade, and duration of recovery. In a series of 9 GBS patients [12], Wilmshurst JM et al found an enhancement of the cauda equina in all 4 cases examined by spine MRI. It has been postulated that gadolinium enhancement reflects disruption of the blood-nerve barrier due to inflammatory changes of the spinal nerve roots, characterized by mononuclear cell infiltrates, edema, and segmental demyelination [20–22]. They argued that MRI should be performed as part of the routine or emergency "work up" of the patients presenting with low back pain because a positive enhancement of the cauda equina reinforces the diagnosis of GBS. In our study, both cases demonstrated moderate or conspicuous cauda equine and nerve root enhancement on lumbar MRI, consistent with the distribution of pain and abnormal EMG findings.

Therefore, MRI is clinically useful in selected GBS patients with atypical EMG or clinical features.

4 Conclusions

The typical clinical features of GBS include progressive weakness and diminished or absent reflexes. However, the spectrum of GBS variants may manifest as pure sensory GBS, bilateral lumbar nerve root disease, or not accompanied by limb weakness. Over 50% of patients experience severe pain, and low back pain is a common symptom of GBS; however, this presentation as an initial or early prominent symptom may delay the diagnosis of GBS. Both cases in this study presented low back pain without characteristic limb weakness, resulting in the difficulties in diagnosis at the early stage. Taken together, this report shows that it is important to provide a detailed assessment of electrophysiological studies and CSF analysis. Furthermore, enhancement of the cauda equina in gadolinium-enhanced MRI of the lumbar spine is a helpful diagnostic tool for recognising atypical GBS.

Conflict of interest: The authors have no conflict of interest to declare.

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