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Case Report

Guillain-Barre Syndrome Mimicked by Spinal Stenosis in A Case of Chronic Prolapsed Intervertebral Disc: A Case Report

Yi Xuan Lee.[1]

Abstract

Background: Guillain-Barre Syndrome (GBS) is a rare acute autoimmune polyneuropathy, usually preceded by infections. It can be difficult to diagnose, especially in patients with underlying neurological comorbidities. **Case:** A 54-year-old male, with a long history of a prolapsed intervertebral disc, presented with progressive and asymmetrical onset tetraparesthesia for 4 weeks, which was associated with progressive paraparesis for 2 weeks. The diagnosis of GBS was initially missed due to a lack of relevant history of prior infection, atypical presentation (asymmetrical limb weakness), and radiological evidence of spinal stenosis. Nerve conduction study, cerebrospinal fluid analysis, and antiganglioside antibodies later confirmed the diagnosis of GBS. The patient was started on intravenous immunoglobulin and achieved significant improvement. He was discharged a week later and transferred to a rehabilitation hospital. **Conclusion:** GBS should not be excluded prior to diagnostic tests and lab work in neurological patients. Physicians should avoid over-reliance on radiological findings to conclude a diagnosis. Comprehensive history and examinations to understand the development of patients' presentations should be prioritized when establishing a diagnosis.

Key Words: Guillain-Barre Syndrome; Polyradiculoneuropathy; Intervertebral Disc Displacement; Spinal Stenosis (Source: MeSH-NLM).

Introduction

Guillain-Barre Syndrome (GBS) is a rare acute autoimmune polyneuropathy, with a global prevalence of only 0.0019% based on the Global Burden of Disease Study 2019.[1] However, around 20-30% of cases develop life-threatening respiratory failure due to severe generalized paralysis.[2] GBS-related years lost due to disability peak among children aged 5-9 years and elderly aged 60–64 years. Across all age groups, males are more likely to develop GBS than females.[3] GBS is usually precipitated by gastrointestinal or respiratory infections and, rarely, systemic illnesses, post-surgery, trauma, and pregnancy.[4] Several systemic diseases, such as Hodgkin's disease, lung cancer, thyroid disease, and systemic lupus erythematosus, were also reported to be related to GBS.[4] Common presentations of GBS include progressive limb weakness with reduced tendon reflexes, muscle or radicular pain, ataxia, sensory symptoms, and autonomic dysfunctions.[2]

In contrast, spinal stenosis is the narrowing of the spinal canal (central canal or neural foramina), that causes central cord or nerve roots compression. The most common etiology of spinal stenosis is degenerative spine diseases, including disc herniation, facet joint hypertrophy, ligamentum flavum hypertrophy, osteophyte formation, and spondylolisthesis.[5] Lumbar spinal stenosis is classically presented as lower back and leg pain, lower limb weakness, paresthesia, and loss of balance. In severe cases like cauda equina syndrome, bowel or bladder incontinence can occur due to lumbar-sacral nerve root compression.[5,6]

Highlights:

- Guillain-Barre Syndrome (GBS) is a rare acute autoimmune polyneuropathy that can cause severe complications.
- Broad differentials of neurological signs and symptoms increase the difficulty of diagnosing GBS.
- Comprehensive investigations should be established to rule out GBS, especially in patients with coexisting neurological comorbidities.

Although acute ascending sensorimotor polyneuropathy is a distinctive feature of GBS, it can be sometimes challenging for physicians to diagnose GBS when there are atypical presentations, rare variants, and heterogeneous manifestations. Broad differentials, lacking high sensitivity and specificity diagnostic tools, further complicate the diagnosis of GBS.[7] This report describes a case of GBS initially misdiagnosed as spinal stenosis in a patient with a long history of a prolapsed intervertebral disc.

[1] MD. The National University of Malaysia, Kuala Lumpur, Malaysia.

About the Author: *Yi Xuan is a medical graduate from The National University of Malaysia. She is a passionate volunteer in healthcare and human rights.*

Correspondence:
Yi Xuan Lee
Address: Universiti Kebangsaan Malaysia, 43600 Bangi, Selangor, Malaysia
Email: yixuan005@gmail.com

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The Case

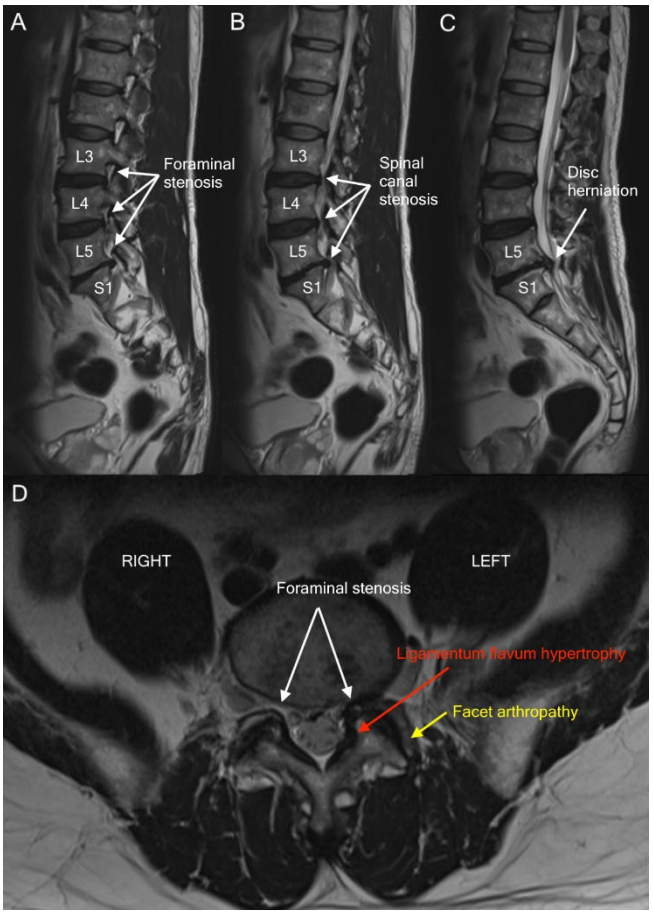
A 54-year-old gentleman with a long history of a prolapsed intervertebral disc presented to a tertiary hospital in Kuala Lumpur, Malaysia in June 2022, complaining of progressively worsening bilateral upper and lower limb numbness for the past 5 weeks, associated with bilateral lower limb weakness in the last 3 weeks. He was diagnosed with a slipped disc at the L4-L5 vertebral level 21 years ago. No surgical intervention was done at the time, and he had been asymptomatic since, only requiring physiotherapy.

5 weeks before admission, the patient had a gradual onset of numbness in his right foot, which progressed proximally from his lower limbs to the waist bilaterally, and was associated with bilateral hand numbness up to the elbow. 3 weeks prior to admission, he began to experience bilateral lower limb weakness that worsened rapidly over a span of 2 weeks, until he was wheelchair-bound. At this point, he was also unable to hold a spoon firmly nor button up his shirt. He went to a private hospital and a Magnetic Resonance Imaging (MRI) thoracolumbar spine was done, revealing varying degrees of foramen stenosis due to multiple degenerative changes at L3 to S1 discs (*Figure 1*). Therefore, he was diagnosed with spinal stenosis and surgical intervention was proposed. However, the patient did not consent for the surgery and opted for medical treatment. He was then given Pregabalin capsules 75mg twice daily, Eperisone tablet 50mg once daily, and Vitamin B-complex tablet once daily.

Despite the medications, the patient's symptoms persisted. One week after the initiation of medical therapy, he visited our center for a second opinion. Upon thorough questioning, the patient recalled having 3 days of mild cough 2 weeks prior to the onset of his symptoms. Otherwise, he had no history of trauma, back pain, radiating pain, and bowel or urinary incontinence. He also had no diplopia, slurred speech, dysphagia, or dyspnea. On upper limb examination, the tonus was normal and sensation was intact. The power of his upper limb was reduced (Medical Research Council scale 4/5) with absent deep tendon reflexes. Upper limb proprioception and finger-to-nose test were unremarkable, with no dysdiadochokinesia. The grip-and-release test was negative, but the finger escape test was positive. On lower limb examination, sensation was intact. Hypotonia was present in the lower limbs and power was reduced at 4/5 over the hip and knee, 1/5 for dorsiflexion, and 2/5 for plantar flexion. Babinski's sign was equivocal with no clonus response. Lower limb proprioception was impaired, and the patient was unable to perform the heel to shin test. All upper and lower limbs findings were bilaterally symmetrical.

MRI of the cervical and thoracic spine was done, revealing no obvious spinal cord stenosis or lesions at the cervical and thoracic level. Nerve conduction studies (NCS) showed electrophysiological evidence of bilateral sural sparing, generalized, predominantly demyelinating polyneuropathy, suggestive of Guillain-Barre Syndrome, particularly the acute inflammatory demyelinating polyneuropathy (AIDP) subtype (*Table 1*). The diagnosis was further confirmed by cerebrospinal fluid analysis, which showed cytoalbuminologic dissociation: normal white cell count (0/mm3) and elevated total protein (1842mg/dL). Antiganglioside antibodies were also positive. The Modified Erasmus GBS Outcome Score (mEGOS) calculated for this patient was 3, representing a 41% probability of an inability to walk after 4 weeks and 18% after 3 months.

Figure 1. T2 Weighted MRI of the Lumbar Spine.



Legend: A) Sagittal Image Shows Foraminal Stenosis (white arrows) at L3-L4, L4-L5, and L5-S1 levels. (B) Sagittal Image Shows Lumbar Spinal Stenosis (White Arrows) at L3-L4, L4-L5, and L5-S1 Levels. (C) Sagittal Image Shows Disc Herniation (White Arrow) at L5-S1 Level. (D) Axial Image at L5-S1 Level Shows Bilateral Foraminal Stenosis (left>right, White Arrows), Ligamentum Flavum Hypertrophy (Red Arrow) and Facet Joint Arthropathy (Yellow Arrow).

The patient received intravenous immunoglobulin (IVIg) 0.4g/kg daily for 5 days and inpatient physiotherapy. His physiotherapy exercises focused on limb muscle strengthening. Upper limb proprioceptive neuromuscular facilitation was done on days 1 and 2 of admission, while other exercises were performed 3 times a day until discharge. These exercises included bilateral leg bridging with a 10-second hold, straight leg raise of 45 degrees with a 5-second hold, hip abduction and adduction with isometric exercises, and triceps strengthening exercises with 250g weights. All exercises were repeated 10 times in each training session.

Discussion

The clinical course and severity of GBS vary due to the condition's different pathophysiological processes. In general, the disease progresses through several phases.[2] About two-thirds of patients report respiratory or gastrointestinal symptoms within a period of 4 weeks prior to the onset of GBS symptoms. This is followed by progressive worsening of symptoms (progressive phase) that peaks at 2-4 weeks. Subsequently, the symptoms plateau from 2 days to 6 months (with median of 7 days), before entering the recovery phase, where 80% of patients are able to walk independently within 6 months.[8] In the case described, the patient presented to us 5 weeks after the onset of symptoms, which was during the plateau phase, following 2 weeks of tetraparesthesia and 2 weeks of progressive paraparesis.

Table 1. Nerve Conduction Studies (NCS) Results.

Motor					
Nerve/ Site	**Latency (ms)**	**Amplitude (mV)**	**Conduction Velocity (m/s)**	**F wave (ms)**	**Interpretation**
Median – Abductor Pollicis Brevis (Left/ Right)					
Wrist	5.65/10.35	3.5/3.9	-	38.31/37.78	DML: severely prolonged CMAP: reduced MCV: reduced F-wave: prolonged
Elbow	11.60/16.80	3.0/2.5	40.3/40.3		
Reference	<4.7	>4.2	>47	-	
Ulnar – Abductor Digiti Minimi (Left/ Right)					
Wrist	7.60/5.15	3.3/2.6	-	45.08/40.50	DML: severely prolonged CMAP: reduced MCV: reduced F-wave: prolonged
Elbow	12.20/12.00	2.1/1.7	45.7/32.1		
Ant. elbow	16.70/17.00	1.6/1.0	31.1/30.0		
Reference	<3.7	>7.9	>52	-	
Peroneal – Extensor Digitorum Brevis (Left/ Right)					
Ankle	8.30/8.40	1.2/0.8	-	NR	DML: severely prolonged CMAP: reduced MCV: reduced F-wave: prolonged
Fib. head	NR/NR	NR/NR	0/0		
Knee	NR/NR	NR/NR	0/0		
Reference	<6.5	>1.1	>42	-	
Tibial – Abductor Hallucis (Left/ Right)					
Ankle	7.75/8.20	1.6/1.0	-	68.5/68.83	DML: severely prolonged CMAP: reduced with conduction block MCV: reduced F-wave: prolonged
Knee	23.00/25.75	0.5/0.1	27.5/23.9		
Reference	<6.1	>5.3	>37	-	
Sensory					
Nerve/ Site	**Rec. Site**	**Latency (ms)**	**Peak-Peak Amplitude. (µV)**	**Velocity (m/s)**	**Interpretation**
Median – Digit II (Left/ Right)					
Digit II	Wrist	NR/NR	NR/NR	0/0	DSL: absent SNAP: absent
Reference		<3.3	>15	-	
Ulnar – Digit V (Left/ Right)					
Digit V	Wrist	NR/NR	NR/NR	0/0	DSL: absent SNAP: absent
Reference		<3.1	>13	-	
Sural – Lateral Malleolus (Left/ Right)					
Calf	Lat. malleolus	2.10/1.95	2.0/2.5	66.7/71.8	DSL: normal SNAP: reduced
Reference*		<3.6	>4	-	

Legend: DML, distal motor latency; CMAP, compound muscle action potential; MCV, motor conduction velocity; DSL, distal sensory latency; SNAP, sensory nerve action potential amplitude.

In 2011, the Brighton Collaboration GBS Working Group refined the original diagnostic criteria for GBS from the National Institute of Neurological Disorders and Stroke by providing the diagnostic level of different symptoms and ancillary examinations.[9-11] It is crucial to diagnose GBS timely due to its rapid progression and high mortality (3-10%), particularly once patients develop autonomic dysregulation, resulting in respiratory failure, arrhythmia, and blood pressure instability.[7] In our patient's case, the cough was an important hint for GBS, but was missed during the first hospital visit, despite it being the most common presenting complaint in primary care.[12] Other common complaints from GBS include back pain, abdominal symptoms, pharyngitis, dermatitis, fever, headache, and fatigue. Even though the presenting complaint may initially seem irrelevant to the chief complaint, physicians should always conduct a review of the systems and screen for related symptoms.

AIDP was the first type of GBS discovered and is also the most common type. The two terms were used interchangeably until the axonal forms of GBS were discovered, namely acute motor axonal neuropathy and acute motor-sensory axonal neuropathy.[4] Since then, more variants were classified under the spectrum of GBS, including paraparesis, pharyngeal–cervical brachial, bilateral facial palsy with paresthesia, pure sensory, Miller Fisher syndrome, and Bickerstaff brainstem.[7] Although some variants share similar clinical presentations like flaccid weakness and areflexia, they can be differentiated by respective distinctive pathological characteristics. Diagnosis of GBS and its subtypes can be confirmed by electrodiagnostic studies, cerebrospinal fluid analysis, and serum antiganglioside antibodies analysis.[4,7,8] NCS, in this case, revealed classic findings of AIDP: prolonged F-waves, reduced compound muscle action potential with motor conduction blocks, reduced motor conduction velocities, prolonged distal sensory latency, and reduced sensory nerve action potential. Another important finding in sensory NCS which differentiates demyelination from axonal polyneuropathy is the "sural-sparing pattern", where the sensory action potential of the sural nerve is normal or relatively preserved compared to a total absence of median and ulnar sensory nerve action potentials. This is the most specific sensory abnormality in AIDP.[3]

Management of GBS patients requires a multidisciplinary approach and patients often require intensive care unit admission due to respiratory failure, paralysis, bulbar and autonomic cardiovascular dysfunction.[4] Common complications like atelectasis, aspiration, nosocomial infections, deep venous thrombosis, and pulmonary embolism should be monitored closely to improve the overall outcome and prognosis.4 Immunomodulatory therapy is best started within 2 weeks of symptom onset to achieve maximum efficacy. IVIg, plasma exchange, or combination therapy are all proven equally effective in treating GBS; though IVIg is often chosen over plasma exchange due to easier administration, wide availability, and lesser adverse effects.[2,7] Most patients show substantial recovery and resume walking ability within 6 months.[2,4,7]

The mEGOS is a validated tool used to predict the prognosis of GBS patients.[13,14] A lower score may prompt physicians to utilise more aggressive treatment approaches in the early phase of diagnosis, such as lower threshold ICU admission and a higher dose of IVIg treatment.[4,13] However, a second IVIg course should not be given as it does not benefit patients and causes more severe adverse events instead, as suggested by the Netherlands SID-GBS RCT trial.[15] The I-SID-GBS study, a global counterpart of the Netherlands study, is currently ongoing as part of the International Guillain-Barré Syndrome Outcome Study and is expected to complete in September 2024, elucidating further guidance on adequate treatment.[16]

GBS can be difficult to diagnose, especially in patients with co-existing neurological comorbidities. Established diagnostic criteria of GBS should be assessed when patients with limb weakness have symptoms atypical for their current diagnosis. In this case, leg pain is absent, despite the fact that it is the most common symptom of spinal stenosis.[4,5] Also, physicians should refrain from over-relying on radiological findings to conclude a diagnosis. Comprehensive history and examination to discover reasons behind patients' presentations should be the mainstay when making a diagnosis.

Summary – Accelerating Translation

Guillain-Barre Syndrome (GBS) is a rare neurological disease. Its complications can range from limb weakness, muscle pain, loss of balance, and abnormal sensations, to life-threatening ones in more severe cases, such as autonomic dysfunctions. Diagnosing GBS in patients with existing neurological conditions can be challenging due to overlapping symptoms and signs. When developing a diagnosis, comprehensive history and examination to discover reasons behind patients' presentations should be prioritized. Also, GBS should not be excluded before diagnostic tests and laboratory investigations are completed. Careful evaluation and management of GBS patients using multidisciplinary care should be prioritized to ensure optimal patient recovery.

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