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A sensory-predominant Guillain-Barré syndrome spectrum disorder with preserved tendon reflexes: a case report

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Case Report

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Abstract

Background

Guillain-Barré syndrome (GBS) is an acute immune-mediated peripheral neuropathy that usually causes progressive limb weakness with reduced or absent tendon reflexes. Some patients, however, present mainly with sensory symptoms, mild weakness, or preserved reflexes, which can make early recognition more difficult.

Case presentation

A 71-year-old Han Chinese man presented with a 10-day history of distal paresthesia that became worse 2 days after diarrhea. He developed glove-and-sock sensory loss, lower-limb heaviness, gait difficulty, and mild distal weakness. Neurological examination showed reduced distal superficial sensation, preserved tendon reflexes, and no pathological reflexes. Brain MRI was unremarkable, and spinal MRI did not identify a central lesion that could explain the presentation. Cerebrospinal fluid findings showed albuminocytologic dissociation. Because neurophysiological testing was not completed during hospitalization, the working diagnosis was a clinically suspected sensory-predominant GBS-spectrum disorder. The patient received intravenous immunoglobulin together with supportive care and other adjunctive treatments, improved during hospitalization, and regained independent walking. At approximately 12-month telephone follow-up, the symptoms had largely resolved and no recurrence was reported.

Conclusions

When patients present after gastrointestinal infection with symmetrical distal sensory disturbance and albuminocytologic dissociation, GBS-spectrum disorders should still be considered even if weakness is mild and tendon reflexes are preserved. This case shows how difficult an atypical sensory-predominant GBS-spectrum presentation can be to recognize. Because neurophysiological testing was not completed during hospitalization and several interventions were given during the same period, treatment-specific effects cannot be determined.

Background

Guillain-Barré syndrome (GBS) refers to a group of acute immune-mediated peripheral neuropathies that often occur after an infection[1]. Typical clinical features include symmetrical limb weakness, reduced or absent tendon reflexes, and sensory symptoms of varying severity[2–4]. Many patients report a preceding respiratory or gastrointestinal infection, and the diagnosis is often supported by cerebrospinal fluid protein-cell dissociation together with neurophysiological abnormalities[5]. Because the disease can progress quickly and may lead to marked motor impairment or respiratory failure, early recognition and treatment are important[6].

GBS does not always present in the usual way[7]. In a small subset of patients, sensory symptoms predominate, weakness is relatively mild, and tendon reflexes may remain intact early in the course[8]. Such sensory-predominant presentations may be mistaken for spinal cord disease, degenerative spine disease, diabetic peripheral neuropathy, nutritional neuropathy, or other sensory neuropathies, which can delay diagnosis. In this setting, a case report is more useful for showing the diagnostic reasoning process than for assigning improvement to any single concurrent intervention. Single case reports are better suited to raising clinical questions and illustrating diagnostic difficulties than to proving treatment effects. We therefore report a case of clinically suspected sensory-predominant GBS-spectrum disorder with preceding diarrhea, glove-and-stocking sensory impairment, albuminocytologic dissociation, preserved tendon reflexes, and incomplete neurophysiological assessment. Our purpose is to show how the diagnosis was reached in an atypical presentation and to document the clinical course. This case report was prepared in accordance with the CARE case report guideline and its explanation and elaboration paper[9, 10].

Case presentation

A 71-year-old Han Chinese man was admitted to the Department of Neurology on June 12, 2025, because of numbness in the distal extremities that had lasted 10 days and had become more severe over the preceding 2 days. Ten days before admission, he developed diarrhea after cold exposure, followed by tingling in the fingers and toes. The symptoms persisted and gradually spread proximally in a glove-and-sock distribution. Two days before admission, the numbness worsened and was accompanied by heaviness in the lower limbs, difficulty walking, and discomfort in the shoulders and back. He did not develop facial weakness, coughing while drinking, dysphagia, or obvious urinary or bowel dysfunction.

The patient had a history of prostatitis and Helicobacter pylori infection, but no history of hypertension, diabetes mellitus, coronary artery disease, hepatitis, or tuberculosis. He had undergone hemorrhoidectomy about 20 years earlier. He did not smoke or drink alcohol. The family history was unremarkable. On admission, his temperature was 36.6°C, pulse was 95 beats per minute, and respiratory rate was 18 breaths per minute. He was alert, spoke fluently, and showed no abnormality in higher cortical function. Cranial nerve examination was normal. Muscle tone was normal in all limbs. Proximal muscle strength was grade 5, and distal muscle strength was grade 5-. Superficial sensation was reduced in the distal extremities in a glove-and-sock pattern, whereas deep sensation was preserved. Biceps, triceps, knee, and ankle reflexes were symmetrically preserved, and no pathological reflexes were elicited.

A peripheral nervous system disorder was initially suspected, and supportive care was started while the diagnostic work-up continued. Laboratory testing on June 13, 2025, showed no clear abnormality in the complete blood count, urine analysis, or stool analysis. Liver function, renal function, coagulation indices, and thyroid function were within or close to the normal range. Fasting blood glucose was 5.65 mmol/L, glycated hemoglobin was 5.8%, serum vitamin B12 was 382 pg/mL, and folate was 8.94 ng/mL. Serological tests for hepatitis B, hepatitis C, syphilis, and HIV were negative. Plasma homocysteine was

23.10 μmol/L. Electrocardiography showed sinus rhythm, sinus tachycardia, and intermittent atrial premature contractions.

Subsequent evaluation showed no relevant abnormality on non-contrast brain MRI. MRI of the cervical, thoracic, and lumbar spine demonstrated multilevel disc herniation or bulging with degenerative changes. No definite spinal cord morphological or signal abnormality was identified, and no parenchymal spinal lesion was found that could explain the current symptoms. Representative imaging findings are shown in Fig. 1. Cervical vascular ultrasonography was essentially unremarkable. Abdominal ultrasound showed fatty liver and a left renal cyst. Chest CT showed fibrotic foci in the lingular segment of the left upper lobe and the medial segment of the right middle lobe, together with a small mass in the right middle lobe that required follow-up. Orthopedic consultation concluded that the spinal imaging findings were insufficient to explain the symmetrical distal sensory deficit and recommended further neurophysiological testing. Because of practical limitations, electromyography and nerve conduction studies were not performed during hospitalization.

Lumbar puncture was performed on June 16, 2025. The cerebrospinal fluid (CSF) was clear and colourless. Routine examination showed a white blood cell count of 0.001 × 10^6/L and a red blood cell count of 0.001 × 10^6/L, and the Pandy test was negative. Biochemical testing showed a protein concentration of 590 mg/L, a glucose level of 4.27 mmol/L, and a chloride concentration of 133.3 mmol/L. Cerebrospinal fluid culture showed no bacterial growth after 48 hours. The combination of elevated CSF protein and a normal cell count was consistent with albuminocytologic dissociation. Essential CSF findings are summarized in Table 1. Considering the acute-to-subacute onset, preceding diarrhea, symmetrical distal sensory impairment, albuminocytologic dissociation, and the absence of a definite organic central nervous system lesion on imaging, the most reasonable working diagnosis was a clinically suspected sensory-predominant Guillain-Barré syndrome (GBS) spectrum disorder[11]. Because nerve conduction studies and electromyography were not completed during hospitalization, diagnostic certainty remained limited, and the case was not presented as a definitive diagnosis of classic GBS.

Table 1
Cerebrospinal fluid findings

Cerebrospinal fluid parameter	Result	Reference range
Biochemical examination		
Protein (mg/L)	590	150–450
Chloride (mmol/L)	133.3	120–132
Glucose (mmol/L)	4.27	2.5–4.5
Routine examination		
Appearance	Colorless	Colorless
Transparency	Clear	Clear
Coagulation	No clot	No clot
Pandy test	Negative	Negative
Red blood cell count (×10^6/L)	0.001	0
White blood cell count (×10^6/L)	0.001	0–8
Microbiological examination		
CSF culture	No bacterial growth after 2 days	Not applicable

In the differential diagnosis, although MRI of the cervical, thoracic, and lumbar spine showed degenerative changes, no definite spinal cord parenchymal lesion was identified that could explain the acute symmetrical distal sensory symptoms. The patient also had no evidence of diabetes mellitus. Vitamin B12 and folate levels were normal, and the available test results did not support nutritional or metabolic peripheral neuropathy. Taken together, these findings favored a GBS-spectrum disorder over the main alternative diagnoses.

The patient received intravenous immunoglobulin (IVIG) from June 18 to June 20, 2025. Based on a body weight of 92.5 kg, the planned total dose was approximately 2 g/kg. Owing to the medication formulation, infusion schedule, and patient tolerance, the full dose was administered as divided infusions over three days, with prophylactic dexamethasone 5 mg given before the first infusion. No infusion-related adverse event occurred during treatment, and serial monitoring of renal function and coagulation markers showed no major abnormality. Adjunctive medications included methylcobalamin, vitamin B1, vitamin B6, vitamin C, and citicoline.

During hospitalization, the patient also received several integrative medicine interventions during the same period, including oral modified Ermiao San, acupuncture, lower-back cupping, topical herbal application, and herbal washing therapy. Detailed acupuncture parameters are provided in Additional file 1: Table S1, and the herbal prescription and administration details are provided in Additional file 1: Table

S2. These measures were completed as planned, and no treatment was stopped because of tolerability concerns. Because IVIG, supportive care, and several integrative medicine interventions were given together, the specific contribution of any single intervention cannot be determined.

After treatment, numbness in the hands and feet decreased, and lower-limb weakness together with shoulder and back discomfort improved. The patient regained independent walking, and sleep, bowel function, and urinary function were satisfactory. He was discharged on June 26, 2025. Figure 2 summarizes the clinical course and the main diagnostic and therapeutic milestones. Telephone follow-up was performed approximately 12 months after discharge. The patient reported overall improvement compared with the initial stage of illness. Numbness in both feet had resolved, with only mild residual sensory abnormality in the left index finger. He rated symptom severity as approximately 1–2 points, compared with a peak of 6 points before hospitalization, and reported independent walking without major difficulty during stair climbing or long-distance walking. No definite recurrence, new motor impairment, cranial nerve involvement, dyspnea, or major autonomic dysfunction was reported. Nerve conduction studies were reportedly performed at another institution, but the detailed results were unavailable to the authors. Because follow-up information was obtained mainly by telephone, no standardized functional scale was used, complete follow-up neurophysiological data were unavailable, and several therapies continued after discharge, the long-term outcome should be interpreted cautiously and should not be regarded as evidence of treatment-specific efficacy.

Discussion

The main clinical message of this case is that preserved tendon reflexes do not rule out a Guillain-Barré syndrome (GBS) spectrum disorder in a patient with an acute sensory-predominant neuropathic syndrome after infection. Previous reports suggest that sensory-predominant, pure sensory, or sensory-ataxic phenotypes can fall within the GBS spectrum, and some patients do not show the typical pattern of flaccid paralysis at the initial stage[12]. In the present case, bilateral distal numbness, a glove-and-stocking distribution, mild distal weakness, and the absence of cranial nerve or respiratory involvement were all compatible with an atypical GBS-spectrum presentation[13].

Several findings made GBS a reasonable diagnostic consideration. The preceding diarrhea supported a post-infectious immune-mediated process. The acute-to-subacute progression, bilateral symmetrical distal distribution, and cerebrospinal fluid protein-cell dissociation were also consistent with immune-mediated peripheral neuropathy[14]. In addition, imaging did not reveal a definite central nervous system lesion that could explain the symptoms, which made a GBS-spectrum disorder more likely.

One notable feature of this case was the preservation of tendon reflexes. Reduced or absent reflexes are classically associated with Guillain-Barré syndrome (GBS), but previous reviews and case reports have shown that some patients, particularly those with atypical subtypes, may have normal or even exaggerated tendon reflexes[15, 16]. For that reason, preserved reflexes alone should not exclude

Guillain-Barré syndrome in a patient with preceding infection, symmetrical distal sensory impairment, and cerebrospinal fluid protein-cell dissociation.

The lack of neurophysiological testing during hospitalization is a major limitation of this case. The Brighton framework and later validation studies show that the completeness of cerebrospinal fluid and electrophysiological data affects diagnostic confidence[11]. Because nerve conduction studies and electromyography were not completed during hospitalization, the case could not be classified further as a demyelinating or axonal subtype, and diagnostic certainty remained limited[17]. Accordingly, the available evidence supports a clinically suspected sensory-predominant GBS-spectrum disorder rather than a definitive diagnosis of classic GBS.

From a treatment perspective, once a GBS-spectrum disorder became the working diagnosis, the patient received IVIG together with supportive care and multiple adjunctive interventions, followed by gradual symptomatic improvement. This case should not be interpreted as evidence for the efficacy of any single adjunctive intervention. Current guidelines support IVIG as a standard disease-modifying treatment for GBS, whereas the value of additional adjunctive interventions remains uncertain[18]. The present report should therefore be understood primarily as a description of clinical management and diagnostic reasoning rather than as evidence of treatment-specific efficacy.

The value of this case lies mainly in showing how clinicians recognized and reassessed an atypical Guillain-Barré syndrome (GBS) spectrum presentation. The emphasis is not on proving the efficacy of any specific adjunctive therapy, but on how the medical history, neurological examination, cerebrospinal fluid (CSF) findings, and imaging results were integrated to reach a working diagnosis when the presentation differed from classic GBS. This case also indicates that preserved reflexes should not, by themselves, remove a GBS-spectrum disorder from consideration in the appropriate clinical setting.

In addition, the patient did not undergo systematic longitudinal follow-up at prespecified intervals after discharge. The 12-month outcome data were obtained mainly by telephone, no standardized functional scale was used, complete follow-up neurophysiological data were unavailable, and several therapies continued after discharge. Therefore, the long-term outcome should be interpreted cautiously, and these follow-up findings should not be regarded as evidence of treatment-specific efficacy.

Conclusion

This case report describes a clinically suspected sensory-predominant Guillain-Barré syndrome (GBS) spectrum disorder with preceding diarrhea, symmetrical distal sensory impairment, albuminocytologic dissociation, mild muscle weakness, and preserved tendon reflexes. In patients with atypical acute peripheral neuropathy, when imaging does not show a definite central lesion and metabolic or nutritional causes are not supported, clinicians should integrate the history, neurological examination, and cerebrospinal fluid findings carefully while keeping GBS-spectrum disorders in the differential diagnosis. The absence of comprehensive neurophysiological testing and the concurrent use of multiple therapies prevent treatment-specific conclusions.

Patient perspective

The patient reported that the most bothersome symptoms during hospitalization were distal limb numbness and difficulty walking. After treatment, these symptoms gradually improved, and his ability to perform daily activities became better than at admission. He also reported no obvious discomfort during treatment and felt that all therapies administered during hospitalization were well tolerated.

Abbreviations

CSF
cerebrospinal fluid
GBS
Guillain-Barré syndrome
IVIG
intravenous immunoglobulin
MRI
magnetic resonance imaging
TCM
traditional Chinese medicine

Declarations

Ethics and consent to participate

Ethical approval was waived by the Ethics Committee of Shaanxi Provincial Hospital of Traditional Chinese Medicine because this study was a retrospective single-patient case report and, in accordance with institutional policy, formal ethical approval was not required.

Consent to publish

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Availability of data and materials

All data generated and analyzed during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Xiaofeng Min drafted the initial manuscript, created all figures and tables, and coordinated manuscript revisions. Wei Liu collected the clinical history, performed the neurological physical examinations, and drafted the Case Presentation section. Runfang Wang conducted the literature search and cross-verified all clinical data. Xiaorui Yang completed the post-discharge telephone follow-up and maintained regular communication with the patient. Guanyu Zhang screened the retrieved literature for relevance and standardized the reference formatting. Ting Ke organized the clinical data collection and assisted with the endocrine-related differential diagnosis. Weihua Zhang assisted in interpreting the examination findings and critically reviewed the manuscript for clinical accuracy. Xinrong Guo conceived the study design and substantially revised the manuscript for important intellectual content. Yang Xiao coordinated patient care and clinical data collection and revised the manuscript for clinical accuracy. All authors read and approved the final manuscript.

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Figures

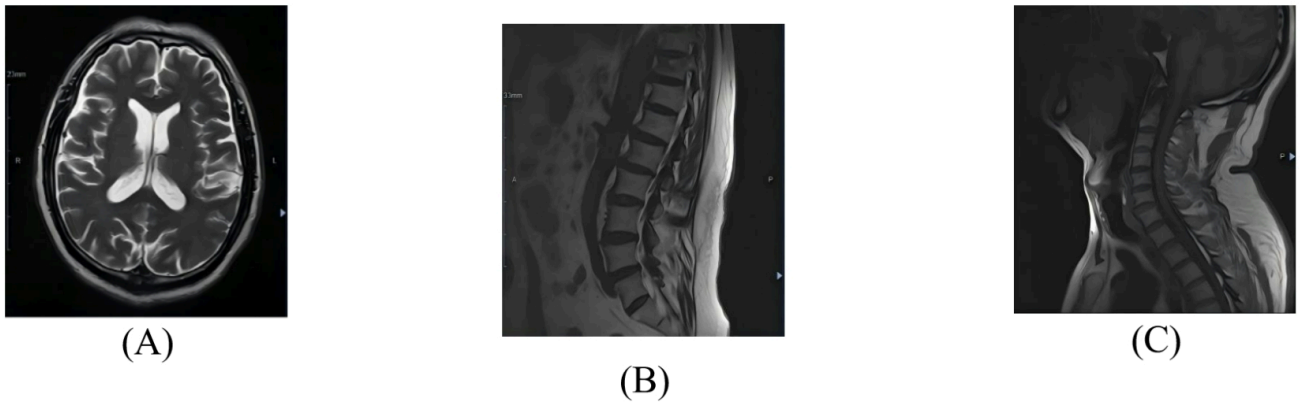


Figure 1

Representative neuroimaging findings.

(A) Brain MRI showed no definite acute intracranial abnormality.

(B) Lumbar MRI showed degenerative changes and disc bulging without a clear central lesion explaining the patient's symptoms.

(C) Cervical MRI showed degenerative disc-related changes without definite spinal cord parenchymal lesion.

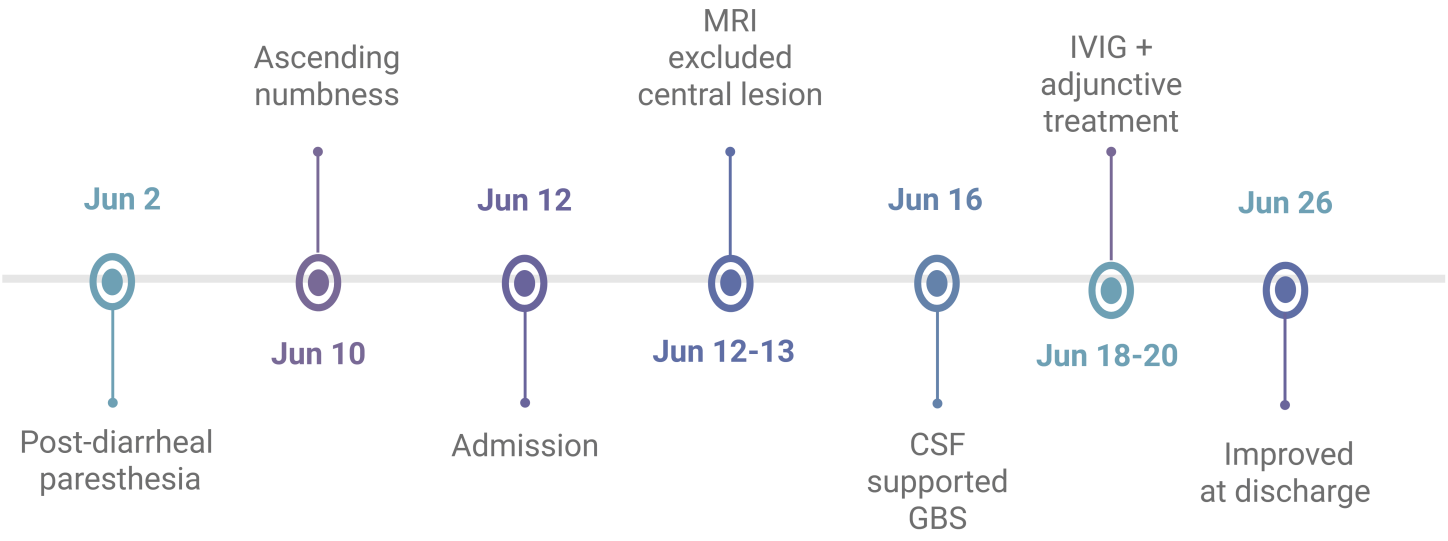


Figure 2

Timeline summarizing the patient's clinical course and the main diagnostic and therapeutic events.

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