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Clinical and electrophysiological features of pure sensory Guillain-Barré syndrome: retrospective analysis of 22 patients across 14 provinces in Southern China

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Abstract

Objective Currently, there are limited reports, both nationally and internationally, regarding Guillain-Barré Syndrome (GBS) that manifests solely with isolated sensory impairment. This study aims to explore the epidemiological and clinical features of GBS patients experiencing only paresthesia in southern China.

Methods We conducted a retrospective analysis of the medical records of GBS patients admitted to 31 hospitals across 14 provinces in southern China from January 1, 2013, to September 30, 2016.

Results A total of 1,056 patients diagnosed with GBS were identified from medical records, of whom 276 had paresthesia as their first symptom. Among these 276 patients, a total of 41 patients with GBS who exhibited only paresthesia were analyzed. Among them, 19 patients served as a control group and showed abnormal compound muscle action potential (CMAP). We identified 22 cases of pure sensory disturbances in GBS patients and named them "pure sensory GBS", characterized by normal CMAP. Comparative analysis revealed no statistically significant differences between the two groups in terms of age at onset, gender, residence, or antecedent events; however, the pure sensory GBS group demonstrated a higher incidence of onset during the spring. Electrophysiological evaluations revealed that the pure sensory GBS group had a lower likelihood of reduced amplitude in sensory nerve action potential (SNAP) compared to the control group. However, there were no significant differences between the two groups in sensory conduction latency, velocity, H-reflex, or F-wave detection. Additionally, no significant differences were observed in cerebrospinal fluid (CSF) studies, treatment modalities, discharge Hughes scores, or peak time. Notably, patients in the pure sensory GBS group had lower Hughes scores at admission and a shorter hospital stay, with these differences reaching statistical significance.

Conclusion Among GBS patients, those presenting solely with sensory disturbances are relatively uncommon, with only 22 cases. Compared to the control group, those patients are more frequently diagnosed in the spring,

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demonstrate a milder degree of reduction in amplitude of SNAP, present with milder symptoms at admission, and have shorter hospital stays.

Keywords Guillain-Barré syndrome, Paresthesia, Clinical features, Electrophysiological evaluations

Introduction

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated disorder affecting the peripheral nervous system. It is marked by rapidly progressive weakness, areflexia, and varying degrees of sensory involvement [1]. GBS is recognized as the most common cause of acute flaccid paralysis worldwide, with an incidence of 1 to 2 cases per 100,000 people each year [2, 3]. While motor deficits are the primary feature of GBS, sensory disturbances, particularly paresthesia, often appear as early symptoms, frequently occurring before the onset of muscle weakness [4]. Paresthesia, which includes abnormal sensations such as tingling, numbness, or burning, typically starts in the distal extremities and moves proximally [1]. Early identification of these symptoms is essential for the prompt diagnosis and treatment of GBS.

While sensory involvement in GBS is well documented, clinical and research efforts have predominantly focused on motor dysfunction. Sensory symptoms, such as paresthesia, are common in most patients but often take a backseat to the motor deficits that characterize the clinical progression of GBS. However, some individuals present with sensory-dominant or isolated sensory forms of GBS, where paresthesia is the primary or only symptom [5, 6]. These atypical presentations can create diagnostic challenges, as they differ from the classical motor-dominant for the GBS, which is more easily recognized in clinical settings [7].

The pathogenesis of GBS is linked to an aberrant immune response, often triggered by a preceding infection, such as Campylobacter jejuni, cytomegalovirus, or Epstein-Barr virus [8, 9]. This immune reaction results in an attack on peripheral nerves [10], causing demyelination, inflammation, or axonal damage. While motor neurons are frequently affected, sensory neurons can also be involved, leading to paresthesia and other sensory disturbances [11]. Understanding the nature of sensory involvement in GBS, especially in cases where sensory symptoms predominate, is crucial for enhancing diagnostic accuracy and informing appropriate treatment strategies.

Diagnosing GBS in patients with predominant sensory symptoms can be challenging, as these presentations may not align with the established diagnostic criteria, which primarily focus on motor weakness. Current guidelines, such as the Brighton criteria, may require reassessment to ensure they encompass the full range of GBS, including sensory-dominant variants. It is widely accepted that GBS is an immune-mediated peripheral neuropathy. Even in patients with the pure sensory variant, relevant laboratory tests, such as ganglioside antibody testing, can still provide strong diagnostic support. Early identification of paresthesia-dominant GBS is vital, as delays in diagnosis can lead to postponed treatment and potentially worse outcomes [4].

In summary, paresthesia is a frequent and often early symptom of GBS, but when it appears as the predominant or sole symptom in certain patients, it can present diagnostic challenges. Recognizing sensory-dominant variants of GBS is critical for ensuring timely diagnosis and appropriate management. As the range of GBS presentations broadens, a deeper understanding of sensory involvement, particularly paresthesia, is essential for refining diagnostic criteria, developing targeted therapies, and enhancing patient outcomes. Further research is necessary to investigate the epidemiological and clinical characteristics of sensory-dominant GBS and to uncover the underlying mechanisms driving this subtype.

Methods

Patient ascertainment

From January 1, 2013, to September 30, 2016, we gathered medical records for 1,056 patients diagnosed with GBS from 31 hospitals across 14 provinces in southern China. Based on the patient's symptoms, we selected individuals who experienced paresthesia, which refers to abnormal sensations, such as tingling or numbness, in a specific part of the body without external stimuli. During the physical examination, these patients may or may not exhibit detectable sensory impairment, which includes hypersensitivity, reduced sensation, or absent sensation to stimuli such as pain, temperature, or vibration. Among these 1056 patients, 276 patients presented with paresthesia firstly, furthermore, we screened 41 patients who exhibited only paresthesia. Then, based on the nerve conduction studies (NCS) results, we identified 22 cases of pure sensory disturbances in GBS patients. A detailed flowchart of this process is shown in Fig. 1.

Information collection and classification

The collected patient information included age, gender, residence, season of onset, antecedent events, clinical symptoms and signs, results of NCS, cerebrospinal fluid (CSF) analysis, treatment methods, peak time, and duration of hospitalization.

The seasons in this study are categorized as follows: spring (March to May), summer (June to August), autumn (September to November), and winter

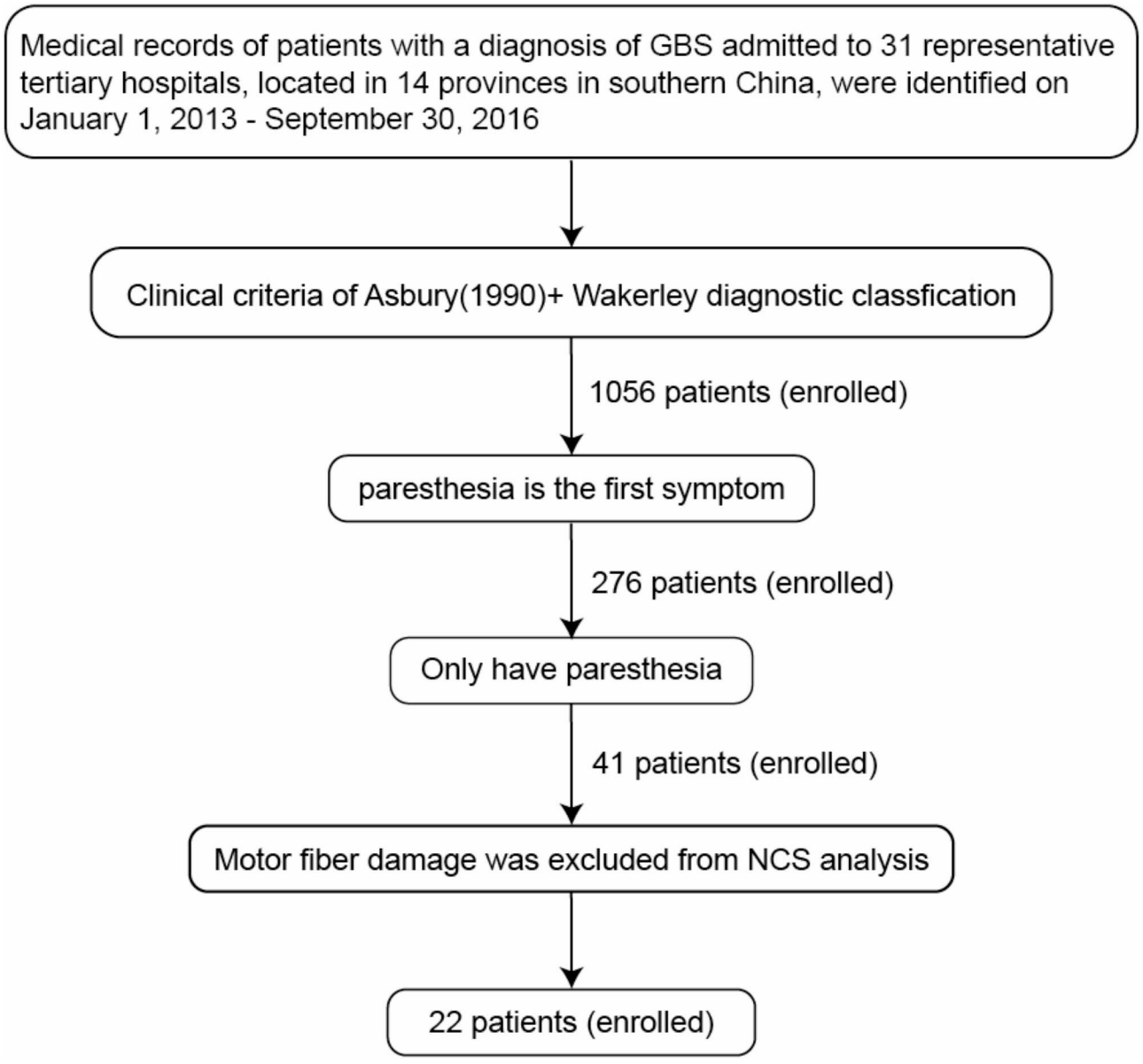

Fig. 1 The specific flowchart for patient ascertainment

(December to February). Antecedent events primarily included respiratory and gastrointestinal infections, with the onset of GBS symptoms within one month of these events being considered indicative of an antecedent event.

The albuminocytological dissociation in CSF was characterized by a protein concentration > 450 mg/L and a cell count < 50/μL. The treatments administered to patients were also recorded, which included intravenous immunoglobulin (IVIG) at a dose of 0.4 g/kg/day for 5 days, glucocorticoid (GCs) therapy, a combination of IVIG and GCs, and purely supportive treatment (NO). The clinical status of GBS patients was evaluated using the Hughes Functional Grading Scale [12], which comprises six grades: Grade 6 indicates death; Grade 5 signifies the need for assisted ventilation; Grade 4 denotes being bedridden; Grade 3 indicates the ability to walk with assistance; Grade 2 indicates the ability to walk independently; Grade 1 represents mild signs and symptoms that allow for engagement in manual labor; and Grade 0 indicates normal functioning. The peak time refers to the interval from the onset of symptoms to their maximum severity, while the hospital stay is defined as the period from admission to discharge.

Electrophysiological classification

NCS were conducted to evaluate the median, ulnar, peroneal, and tibial nerves for motor nerve conduction,

and the median, ulnar, radial, and sural nerves for sensory nerve conduction. Additionally, nerve root damage was assessed through F-wave and H-reflex evaluations. Abnormal findings in NCS were defined as follows [13]: motor NCS showed prolonged distal latencies, slowed conduction velocities, and decreased amplitudes of the compound muscle action potential (CMAP). For sensory NCS of the extremities, abnormalities included reduced sensory nerve action potential (SNAP) amplitude, often accompanied by disproportionate distal latency prolongation or slowed sensory conduction velocities (SCV). F-wave abnormalities were characterized by prolonged or absent F-waves, while the absence of H-reflex was also classified as abnormal. The normal reference values for NCS used in our study were based on those from our previous research [14]. We also took into account the effect of age on NCS results and applied age correction accordingly.

Statistical analyses

Data comparisons were performed using GraphPad Prism software (version 8.0, GraphPad Prism software Corp., USA). Results are presented as mean ± standard error of the mean (SEM). The Mann-Whitney U test was utilized for comparing count data between two groups. Categorical data are expressed as percentages. The $\chi 2$ test was employed to assess differences between the two groups, while the Fisher's exact test was used when the total sample size was less than 40.

Results

Classification of GBS patients with paresthesia

A total of 276 cases of GBS exhibited symptoms of paresthesia, which were categorized based on their characteristics. Out of these, 201 patients (72.83%) reported numbness, 33 patients (11.96%) experienced pain, and 17 patients (6.16%) presented with both pain and numbness. Additionally, 25 patients (9.06%) either did not specify their symptoms or showed sensory deficits or hypersensitivity (Fig. 2A). Further classification based on the presence of additional symptoms indicated that 126 patients (45.65%) experienced paresthesia alongside weakness, while 109 patients (39.49%) had paresthesia and cranial nerve or bulbar paralysis. Notably, the smallest group comprised 41 patients (14.86%) who exhibited only paresthesia (Fig. 2B).

General characteristics of GBS patients with paresthesia symptoms only

41 patients diagnosed with GBS who presented solely with paresthesia were divided into two groups based on CMAP abnormalities: 19 patients (46.34%) showed abnormal CMAP results, served as the control group; while 22 patients (53.66%) had normal CMAP, regarded as pure sensory GBS. Table 1 illustrates a comparative analysis of the general characteristics of these groups. The study found no statistically significant differences in terms of age at onset, gender, geographic residence, or antecedent events. However, it was noted that GBS patients with pure sensory disturbances were more commonly observed in the spring, a finding that reached statistical significance ($P = 0.0418$).

NCS characteristics of GBS patients with only paresthesia symptoms

Table 2 provides an analysis of the NCS results for the 41 patients. These 41 patients had NCS test about two weeks after onset of symptom (13.61 ± 9.36, 15.00 ± 10.76). In the control group, 2 patients (10.53%) were unable to elicit SNAP, while 5 patients (22.73%) in the pure sensory GBS group also did not elicit SNAP. A review of the NCS reports for patients who were able to elicit SNAP

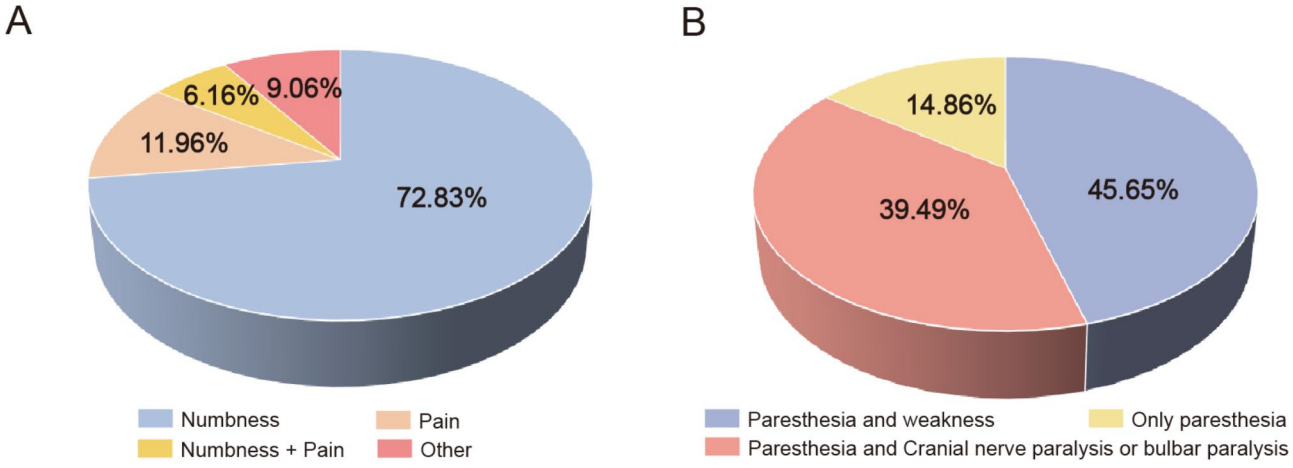

Fig. 2 Classification of GBS patients with paresthesia. **A**: Classification according to the nature of paresthesia. **B**: Depending on whether paresthesia is combined with other symptoms

Table 1 General characteristics of GBS patients with paresthesia symptoms only

Characteristic	Control (*n* = 19)	Pure sensory GBS (*n* = 22)	*P* value
Age at onset, years	41.89 ± 15.33	44.50 ± 16.65	0.6835
Gender			0.0673
Male	14 (73.68)	10 (45.45)	
Female	5 (26.32)	12 (54.55)	
Residence	15	16	0.2890
Urban	9 (60.00)	6 (37.50)	
Rural	6 (40.00)	10 (62.50)	
Season			
Spring	3 (15.79)	10 (45.45)	**0.0418**
Summer	8 (42.11)	5 (22.73)	0.1836
Autumn	3 (15.79)	4 (18.18)	0.8391
Winter	5 (26.32)	3 (13.64)	0.3070
Antecedent events	7	10	
Gastrointestinal infections	3 (42.86)	1 (10.00)	0.2263
Respiratory infections	4 (57.14)	9 (90.00)	0.1730

Data are presented as *n* (%) or mean ± standard deviation

Statistically significant results are indicated in bold

Table 2 NCS characteristics of GBS patients with paresthesia symptoms only

Characteristic	Control (*n* = 19)	Pure sensory GBS (*n* = 22)	*P* value
Duration from symptom onset to NCS test performing, day	13.61 ± 9.36	15.00 ± 10.76	0.4258
Unelicited SNAP	2 (10.53)	5 (22.73)	0.3005
Elicited SNAP	17 (89.47)	17 (77.27)	
Normal	5 (29.41)	8 (47.06)	0.4813
Prolonged latencies	1 (5.88)	0 (0.00)	0.9999
Slowed SCV	10 (58.82)	5 (29.41)	0.1663
Decreased amplitude	12 (70.59)	5 (29.41)	**0.0381**
Conduction block	0 (0.00)	0 (0.00)	0.9999
Abnormal H-reflex	6 (31.58)	7 (31.82)	0.9869
Abnormal F-wave	6 (31.58)	9 (40.91)	0.5362
Normal NCS	0 (0.00)	4 (18.18)	0.1179

Data are presented as *n* (%) or mean ± standard deviation

SNAP, sensory nerve action potential; SCV, sensory conduction velocities; NCS, nerve conduction studies

Statistically significant results are indicated in bold

showed no statistically significant differences between the two groups in terms of latency of SNAP, SCV, H-reflex, and F-wave parameters. However, the pure sensory GBS group exhibited a significantly lower likelihood of decreased amplitude of SNAP compared to the control group, indicating a statistically significant difference ($P = 0.0381$).

Diagnosis, treatment plan, and course of GBS in patients with paresthesia symptoms only

In the CSF analysis, 12 patients (85.71%) in the control group displayed albuminocytologic dissociation, compared to 17 patients (89.47%) in the pure sensory GBS group. Regarding treatment options, the majority of patients in both groups received IVIG (68.42% in the control group and 72.73% in the pure sensory GBS group), while others chose GCs or a combination of IVIG. Notably, one patient in the control group received only supportive treatment. A comparison of the Hughes scores at admission revealed a lower proportion of patients in the pure sensory GBS group with scores ≥ 3, which was statistically significant ($P = 0.0452$). Furthermore, the length of hospital stay was shorter in the pure sensory GBS group compared to the control group ($P = 0.0339$). However, no significant differences were observed between the groups regarding Hughes scores at discharge or the peak time.

Discussion

Paresthesia is frequently observed in patients with GBS and is part of the diagnostic criteria [3]. In this study, we examined data from 276 GBS cases that included paresthesia. The majority of patients reported numbness only

(72.83% in Fig. 2A), while a smaller percentage reported pain or other sensations. Additionally, most patients displayed paresthesia with weakness or cranial nerve involvement (85.14% in Fig. 2B). Importantly, a small portion of the cases presented solely paresthesia (14.86% in Fig. 2B), underscoring the need for heightened awareness to prevent misdiagnosis in clinical settings.

Our study examined the clinical characteristics of 41 GBS patients who presented with only paresthesia. Although these patients did not show motor deficits, we discovered that 19 patients had abnormal CMAP (control group). This finding underscores the necessity of regularly monitoring both symptoms and CMAP changes, especially during the early stages of the condition. Abnormal CMAP may indicate underlying nerve damage, and timely surveillance can assist clinicians in modifying treatment plans to prevent further deterioration.

Based on the results of motor nerve conduction studies, the 41 patients were categorized into two groups: one with paresthesia and abnormal CMAP served as the control group, and the other with paresthesia and normal CMAP, the latter referred to as pure sensory GBS. While comparing the overall characteristics of these groups, we found that the mean age of onset was predominantly among young to middle-aged individuals, with an average age of 41.89 ± 15.33 years in the control group and 44.50 ± 16.65 years in the pure sensory GBS group. The male-to-female ratio was 2.8 (14:5) in the control group, compared to 0.83 (10:12) in the pure sensory GBS group, with no significant statistical differences noted regarding residential areas.

In terms of seasonal onset, about 45.45% of pure sensory GBS presented in the spring, and 9 of the 22 pure sensory GBS patients (40.91%) had respiratory infections as antecedent events (Table 1). Seasonal factors may appear to influence the incidence of GBS, fluctuating temperatures and increased rates of respiratory infections in the spring may act as potential triggers. Therefore, there may be some respiratory viruses involved in the occurrence and development of pure sensory disorders GBS. Angiotensin-converting enzyme 2 (ACE2) mRNA is co-expressed in nociceptors alongside other key proteins involved in pain signal transduction, and plays a role in the formation of nerve endings. Recent studies have demonstrated that ACE2 serves as a receptor for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), facilitating viral entry into the dorsal root ganglion and contributing to peripheral sensitization. Given the discovery that small fibers bear ACE2 epitopes, SARS-CoV-2 could trigger corresponding small-fiber autoimmunity [15]. In addition, respiratory pathogens associated with the onset of GBS mainly include Epstein-Barr virus, cytomegalovirus, Mycoplasma pneumonia, Haemophilus influenzae, and influenza A virus. Whether they are involved in the pathogenesis of pure sensory GBS needs further study and verification [16].

In our analysis of sensory nerve conduction in the 41 patients (Table 2), we observed that some individuals were unable to elicit SNAP, this finding was more prevalent in the pure sensory GBS group (22.73%). Additionally, four patients (18.18%) in the pure sensory GBS group exhibited normal NCS despite experiencing paresthesia. This inconsistency may be due to factors such as measurement errors, variability among operators, and the limited sample size. Our results indicate that, compared to the control group, the pure sensory GBS group showed a lower likelihood of reduced amplitude of SNAP, with a statistically significant difference ($P = 0.0381$). This difference may suggest variations in the underlying pathophysiological mechanisms associated with the different forms of GBS.

During our analysis of CSF (Table 3), we found that the majority of patients exhibited albuminocytologic dissociation (85.71%, 89.47%), although a small proportion did not. This finding underscores the importance of vigilance in clinical practice to prevent misdiagnosis [17]. In terms of treatment, there were no significant statistical differences between the two groups. Most patients received IVIG therapy (68.42%, 72.73%), while some were treated with GCs alone (21.05%, 22.73%), and a few (5.26%, 4.55%) received a combination of both treatments. One patient underwent only supportive care.

The Hughes scores upon admission (Table 3) indicated that patients with pure sensory GBS generally presented with milder symptoms ($P = 0.0452$) and shorter hospital stays ($P = 0.0339$). These findings underscore the importance of implementing stratified management for GBS patients. Early identification and appropriate intervention can lead to improved prognoses, optimize the allocation of healthcare resources, and enhance patients' quality of life [18, 19]. As shown in Table 3, a total of 13 patients (9 + 4) in these two groups had a Hughes score of ≥ 3 at admission, despite only presenting with paresthesia. The intensity and distribution of sensory impairment varied among patients, leading to differing degrees of discomfort or activity limitations. For example, pain could prevent them from walking independently, as the discomfort restricted their ability and willingness to move. Additionally, some patients may have had sensory impairments affecting proprioception, further hindering their ability to maintain balance, which contributed to mobility difficulties. In addition, 9 of these 13 patients exhibited abnormal CMAP findings. As discussed earlier, this suggests the possibility of underlying motor nerve involvement. These patients require longer-term monitoring to avoid misdiagnosis as other types of GBS (such as acute inflammatory demyelinating polyradiculoneuropathy or acute motor-sensory axonal neuropathy) or

Table 3 Diagnosis and treatment plan and course of GBS in patients with paresthesia symptoms only

Characteristic	Control (*n* = 19)	Pure sensory GBS (*n* = 22)	*P* value
CSF Albuminocytological dissociation	12*(85.71)	17**(89.47)	0.9999
Treatment			0.7510
IVIG	13 (68.42)	16 (72.73)	
GCs	4 (21.05)	5 (22.73)	
IVIG + GCs	1 (5.26)	1 (4.55)	
NO	1 (5.26)	0 (0.00)	
Hughes score on admission			
≥ 3	9 (47.37)	4 (18.18)	**0.0452**
<3	10 (52.63)	18 (81.82)	
Hughes score at discharge	0.89 ± 0.66	1.00 ± 0.76	0.7028
Peak time, day	8.92 ± 5.28	6.71 ± 5.61	0.1347
Hospital stay, day	16.00 ± 7.67	12.91 ± 5.51	**0.0339**

Data are presented as *n* (%) or mean ± standard deviation

*14 patients in the abnormal CMAP group underwent lumbar puncture; **19 patients in the normal CMAP group underwent lumbar puncture

CSF, cerebrospinal fluid; IVIG, intravenous immunoglobulin; GCs, glucocorticoids; NO, purely supportive treatment

Statistically significant results are indicated in bold

other conditions (such as spinal cord disorders or toxic neuropathies).

The Hughes scores at discharge (Table 3) indicated that both groups had relatively mild disease, with scores (0.89 ± 0.66 and 1.00 ± 0.76). This supports the view that GBS is a self-limiting condition that responds well to IVIG treatment and typically results in favorable outcomes [20–22]. In comparing the disease progression and severity between the two groups, we found that the average peak time in the control group was 8.92 days, which was significantly longer than the 6.71 days observed in the pure sensory GBS group.

In conclusion, the clinical presentation of GBS is both complex and diverse, exhibiting notable differences in symptoms and underlying pathophysiological mechanisms. The spring season sees a peak in cases of pure sensory GBS, prompting clinicians to maintain heightened vigilance to prevent misdiagnosis. Moreover, while these patients may only exhibit sensory abnormalities, it is crucial to monitor and follow up on CMAP as well. Future research should further investigate the pathogenic mechanisms of the various forms of GBS and their associations with seasonal variations, aiming to provide more precise guidance for clinical practice.

However, our study has certain limitations. First, as a retrospective analysis, it presents some challenges with follow-up and may have a selection bias in case inclusion. Second, the sample size for cases with purely sensory disturbances is relatively small, with the potential for sample loss due to incomplete medical records.

Future clinical studies should aim for a larger sample size and multi-center participation, with a more comprehensive recording of additional biomarkers and pathological features. At the same time, efforts should be made to strengthen the scope and intensity of long-term follow-up, in order to address these issues more comprehensively.

Conclusion

Among GBS patients, those presenting solely with sensory disturbances are relatively uncommon, with only 22 such cases identified in this study. This type of GBS patient tends to experience a relatively mild condition and shorter hospital stays, suggesting that early recognition and intervention may lead to more effective management. Our findings provide valuable insights into this rare manifestation of GBS, and our study contributes to the growing literature on sensory-only GBS.

Abbreviations

GBS	Guillain-Barré Syndrome
CMAP	Compound muscle action potential
SNAP	Sensory nerve action potential
CSF	Cerebrospinal fluid
NCS	Nerve conduction studies
IVIG	Intravenous immunoglobulin
GCs	Glucocorticoid
NO	Purely supportive treatment
SCV	Sensory conduction velocities
SEM	Standard error of the mean
ACE2	Angiotensin-converting enzyme 2
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2

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Author contributions

Database organization and establishment: Z.L. Conceptualization: C.W., S.L. Data collection and organization: C.W., S.L., H.C., B.P., Y.Z. Diagrams drawing:

S.L., B.P., J.L. Manuscript writing: C.W., S.L. All authors contributed to the article and approved the submitted version.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This retrospective study was approved by the ethics committee of the Renmin Hospital of Wuhan University and conducted in accordance with the principles of the Declaration of Helsinki. The requirement for informed consent was waived by the ethics committee of the Renmin Hospital of Wuhan University because the analysis was retrospective.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. van Doorn PA, Van den Bergh PY, K, Hadden RDM, et al. European academy of neurology/peripheral nerve society guideline on diagnosis and treatment of Guillain-Barré syndrome [J]. Eur J Neurol. 2023;30(12):3646–74.
2. McGrogan A, Madle GC, Seaman HE, et al. The epidemiology of Guillain-Barré syndrome worldwide. Syst Literature Rev [J] Neuroepidemiology. 2009;32(2):150–63.
3. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome [J]. Lancet. 2021;397(10280):1214–28.
4. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré Syndrome [J] Lancet. 2016;388(10045):717–27.
5. Packiyarajah P, Sundaresan KT, Nusair MSM. Rare sensory ataxic variant of Guillain-Barre syndrome: A case report [J]. Cureus. 2023;15(6):e40920.
6. Singh A, Jain E, Sharma V, et al. Guillain-Barré syndrome presenting as painful weakness and edema of the legs: A case report [J]. Cureus. 2023;15(6):e40641.
7. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome [J]. Ann Neurol, 1990, 27 Suppl: S21-S4.
8. Finsterer J. Triggers of Guillain-Barré syndrome: Campylobacter jejuni predominates [J]. Int J Mol Sci, 2022, 23(22).
9. Kong Q-X, Gao Z-K, Liu Y, et al. Explore genetic susceptibility association between viral infections and Guillain-Barré syndrome risk using two-sample Mendelian randomization [J]. J Transl Med. 2024;22(1):890.
10. Liu J, Pan R. Causal effects of systemic inflammatory proteins on Guillain-Barre syndrome: insights from genome-wide Mendelian randomization, single-cell RNA sequencing analysis, and network Pharmacology [J]. Front Immunol. 2024;15:1456663.
11. Kuwabara S, Yuki N. Axonal Guillain-Barré syndrome: concepts and controversies [J]. Lancet Neurol. 2013;12(12):1180–8.
12. Hughes RA, Newsom-Davis JM, Perkin GD, et al. Controlled trial prednisolone in acute polyneuropathy [J]. Lancet. 1978;2(8093):750–3.
13. Arends S, Drenthen J, de Koning L, et al. Electrodiagnostic subtyping in Guillain-Barré syndrome patients in the international Guillain-Barré outcome study [J]. Eur J Neurol. 2024;31(9):e16335.
14. Liu S, Xiao Z, Lou M, et al. Guillain-Barré syndrome in Southern China: retrospective analysis of hospitalised patients from 14 provinces in the area South of the Huaihe river [J]. J Neurol Neurosurg Psychiatry. 2018;89(6):618–26.
15. Shiers S, Ray PR, Wangzhou A, et al. ACE2 and SCARF expression in human dorsal root ganglion nociceptors: implications for SARS-CoV-2 virus neurological effects [J]. Pain. 2020;161(11):2494–501.
16. Bellanti R, Rinaldi S. Guillain-Barré syndrome: a comprehensive review [J]. Eur J Neurol. 2024;31(8):e16365.
17. Al-Hakem H, Doets AY, Stino AM, et al. CSF findings in relation to clinical characteristics, subtype, and disease course in patients with Guillain-Barré syndrome [J]. Neurology. 2023;100(23):e2386–97.
18. McKenzie ED, Kromm JA, Mobach T, et al. Risk stratification and management of acute respiratory failure in patients with neuromuscular disease [J]. Crit Care Med. 2024;52(11):1781–9.
19. Kushner DS, Johnson-Greene D, Felix ER, et al. Predictors of discharge to home/community following inpatient-rehabilitation in a US National sample of Guillain-Barre-Syndrome patients [J]. PLoS ONE. 2023;18(5):e0286296.
20. Guo J, Zhang R, Dong R, et al. Interpretable machine learning model for predicting the prognosis of Guillain-Barré syndrome patients [J]. J Inflamm Res. 2024;17:5901–13.
21. Sprenger-Svačina A, Svačina MKR, Gao T, et al. Emerging treatment landscape for Guillain-Barré syndrome (GBS): what's new? [J]. Expert Opin Investig Drugs. 2024;33(9):881–6.
22. Hughes RAC, Swan AV, van Doorn PA. Intravenous Immunoglobulin for Guillain-Barré syndrome [J]. Cochrane Database Syst Rev. 2014;2014(9):CD002063.

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