The Clinical Features of Asymmetry Weakness in Childhood Guillain-Barre syndrome: The Outcome and 5-year Follow-up

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Research article

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Abstract

Objectives To compare the clinical profile and long-term outcome of children with asymmetry weakness and symmetry weakness in Guillain-Barre syndrome (GBS).

Methods We retrospectively analyzed the clinical parameters, auxiliary examinations and long-term outcome between asymmetry weakness and symmetry weakness in childhood GBS.

Results A total of 72 children were included, 12 children had asymmetry weakness. Six children were transient asymmetry weakness and six children were persistent asymmetry weakness. Compared to symmetry weakness children, asymmetry weakness had more preschool children (75% vs 25%, \( p = 0.005 \)), longer days on hospital (26.5(15-37) days vs 11(9-15) days, \( p = 0.000 \)), more mechanical ventilation in children (50% vs 8.33%, \( p = 0.000 \)), higher Disease severity score (DSS) at nadir of disease (4(3-5) vs 3(1-4), \( p = 0.010 \)), more axonal subtypes (50% vs 15%, \( p = 0.013 \)) and more complications (58.33% vs 8.33%, \( p = 0.000 \)). Eight children had sequelae and sixty-four children had good recovery. Compared to good recovery group, sequelae group had more axonal subtypes (62.5% vs 15.63%, \( p = 0.002 \)) and more persistent asymmetry weakness (62.5% vs 4.69%, \( p = 0.000 \)).

Conclusions In conclusion, asymmetry weakness had two types in GBS, namely transient and persistent asymmetry weakness. Asymmetry weakness in GBS indicated more complex condition during disease than symmetry weakness. Persistent asymmetry weakness and axonal subtypes in GBS related with sequelae. Anterior horn cells in the spinal cord involvement may be the possible function in persistent asymmetry weakness combined with axonal subtypes in GBS.

1 Introduction

Guillain-Barre syndrome (GBS) is an acute inflammatory polyneuropathy typically characterized by progressive symmetry weakness and areflexia. GBS is currently the most frequent cause of acute flaccid paralysis in children \([1]\), with an estimated incidence of 0.5:100,000 to 1.5:100,000 individuals in the under 18-year age group \([2]\). It has usually been considered as an immune-mediated polyneuropathy clinically characterized by acute symmetry muscle weakness and areflexia. Symmetry weakness is the core feature in GBS, but asymmetry weakness was also found in some cases. In pediatric GBS, asymmetry weakness was the atypical clinical feature of GBS and it occurred in a significant proportion of children \([3]\). A case report of asymmetry GBS described a patient who developed an acute motor-sensory neuropathy with a more prolonged and incomplete type of recovery in more weakness limb \([4]\). Until now, the clinical feature of asymmetry GBS was only reported in case reports, and its function was unclear. Indeed, these cases report raised issues on diagnosis and features in asymmetry GBS. The aim of this paper is to describe features in asymmetry GBS, make a comparison between asymmetry weakness and asymmetry weakness in GBS and discuss the possible pathophysiology in this type of GBS.

2. Materials And Methods

Children

We retrospectively reviewed our database of children with a diagnosis of GBS at the neurology department of Wuhan children hospital between 2010 and 2018. The diagnosis was made in each case in accordance with the diagnostic criteria of Asbury and Cornblath \([5]\). Recurrent GBS was excluded. We focus on GBS with limbs weakness. The study was approved by the ethical committee of Wuhan Children's Hospital. Written informed consent was obtained from their parents of patient in our study.

Methods

We systematically collected datas on children's demographics, physical examination, nerve conduction results, CSF, and other related test results. Peak weakness was defined as the nadir of disease. Disease severity score (DSS) was determined by Hughes’ functional scale score \([6]\) or upper limb disability grading scale \([7]\) (when the muscle weakness was more prominent in upper limbs). When upper limbs was asymmetry weakness, DSS was evaluated on the more prominent weakness limb. Recovery was defined as a return to normal life with a DSS of 0. At least one year should be given to follow-up.

All children included had undergone electrophysiological study of at least 4 motor and 2 sensory nerves within 3 weeks from symptom-onset. The electrophysiology abnormalities were categorised as acute inflammatory demyelinating polyradiculoneuropathy (AIDP), and axonal type: acute motor axonal neuropathy (AMAN) acute motor and sensory axonal neuropathy (AMSAN) or unclassifiable (equivocal) based on the single study approach in accordance to Hadden et al.’s and Rajabally et al.’s electrodiagnostic criteria \([8]\). If children had more than two electrophysiological studies, subtype was categorized based on last electrophysiological result.
Asymmetry weakness was defined as at least 1 score difference by Medical Research Council (MRC) grade between the right- versus left-side muscles in children at admission[9].

Statistical Analysis

Statistical analyses were performed with SPSS version 11.0 software. Categorical data are presented as proportions, and differences in proportions were tested using the $\chi^2$ test. Continuous data with a normal distribution are presented as the means ± SDs, and these variables were tested using Student t test or analysis of variance. Continuous variables with a skewed distribution are presented as medians (M) and interquartile range (IQR) ([25th – 75th]), and these variables were tested by the Mann-Whitney U test. A test level $\alpha = 0.05$ or $p < 0.05$ indicated that the difference was significant.

3 Results

3.1 Demographics

In total, we identified 72 children with clinical diagnosis of classic GBS. Among these, 42 were males, 30 were females. 24 children were under 6-year-old. The median age at time of diagnosis was 91.86±37.94 months. Antecedent events were found in 49 of children. 7 children had diarrhoea, 14 children had upper respiratory tract infections (URI), 21 children had fever only, 2 children had vaccination, 2 children had Chicken pox and 3 children had herpes. The average days at hospital stay was 12(8-18) days. Ten children need MV during the disease. Twelve children had sensory nerve deficit and sixteen had cranial nerve involvement. Signs of dysautonomia were present in 14 children (35%), 5 of whom had arterial hypertension (58%), 6 had sinus tachycardia (29%), 2 had urinary problems (29%) and 1 had vasomotor disorders (8%). The mean GBS disability score in nadir was 3(2-4). 18 children had positive ganglioside antibodies in serum and 47 had albuminocytologic dissociation on the cerebrospinal fluid (CSF). 15 children were classified as axonal type of GBS (13 AMAN and 2 AMSAN), 45 were classified as AIDP and 12 were classified as equivocal type. One children had abnormality in cranial MRI. Complications: 12 children had complications, including central nerve system (CNS) infection (abnormal electroencephalogram with limit impaired or normal consciousness), liver function impairment, gastrointestinal bleeding, electrolyte disturbance, cardiac arrest, bronchopneumonia, II respiratory failure, atelectasis, severe protein-energy malnutrition, closed craniocerebral injury. The most common pathogen during disease was mycoplasma pneumonia infection. Intravenous immunoglobulin was the first-line therapy, and some children need plasma exchange or glucocorticoid therapy.

3.2 Clinical feature of asymmetry weakness.

Twelve children had asymmetry weakness. Nine children were male, three were female. Nine children were under 6-year-old. The median age at time of diagnosis was 51.25±33.54 months. Antecedent infections were found in 6 children. The mean length of the hospital stay was 26.5(15-37) days. Six children need MV. One child had sensory nerves deficit, and four children had cranial nerves deficit. The nadir of disease in DSS were 4(3-5). Three children had dysautonomia. Six children had persistently asymmetry weakness and six children had transit asymmetry weakness (Table 1). Among six children with persistently asymmetry weakness, one child had good recovery. The characteristics of the asymmetry weakness children or those asymmetry weakness are shown in Table 1. In six children with transit asymmetry weakness, 5 children (5/6, 83.33%) were AIDP, while in persistent asymmetry weakness, 5 children (5/6, 83.33%) were axonal GBS. AIDP were more reported in transit asymmetry weakness while axonal GBS were more reported in persistent asymmetry weakness.

Table 1 clinical feature in 12 children with asymmetry weakness (n=12)
|                        | Case 1                          | Case 2                          | Case 3                          | Case 4                          | Case 5                          | Case 6                          |
|------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Age/sex(years)         | 9/M                             | 2/M                             | 1/M                             | 7/M                             | 3/M                             | 4/F                             |
| Antecedent infections  | N                               | URI                             | N                               | N                               | N                               | N                               |
| Days on hospital       | 21                              | 16                              | 15                              | 18                              | 13                              | 14                              |
| Nerve studies          | AIDP                            | AMAN                            | AIDP                            | AIDP                            | AIDP                            | AIDP                            |
| Mechanical ventilation | N                               | N                               | N                               | P                               | N                               | N                               |
| Sensory nerves deficit | N                               | N                               | N                               | P                               | N                               | N                               |
| The muscle strength at admission (MRC) | left upper limb:3/5 right upper limb and bilateral limbs:5/5 | left lower limb:2/5, right lower limb:4/5, bilateral upper limbs:5/5 | right upper limb:2/5, left upper limb:3/5, bilateral lower limbs:2/5 | bilateral upper limbs:3'/5, left upper limb:2/5, right limb lower:3/5 | right upper limb:2/5, left upper limb:4/5, bilateral lower limbs:2/5 | bilateral upper limbs:5/5, right limb:4/5 |
| Duration time on asymmetry weakness | 2 days                          | 5 days                          | 4 days                          | 12 days                         | 7 days                          | 4 days                          |
| Cranial nerve deficit  | P                               | N                               | N                               | P                               | N                               | N                               |
| Dysautonomia           | N                               | N                               | N                               | P                               | N                               | N                               |
| Ganglioside antibodies | N                               | Anti-GM1                        | N                               | N                               | N                               | N                               |
| Complications          | N                               | N                               | N                               | gastrointestinal bleeding       | electrolyte disturbance         | N                               |
| ICU therapy days       | N                               | N                               | N                               | 5 days                          | N                               | N                               |
| Cranial and spinal MRI | N                               | N                               | N                               | Bilateral frontal white matter: slightly higher signal intensity. | N                               | N                               |
| Pathogen detection results | N                               | mycoplasma pneumoniae           | N                               | N                               | klebsiella pneumoniae enterobacter cholerae enterobacter cloacae | mycoplasma pneumoniae           |
| Albuminocytologic dissociation on the CSF | N                               | N                               | P                               | N                               | Not done                        | P                               |
| Therapy                | G                               | IVIG                            | IVIG                            | IVIG                            | IVIG                            | N                               |
| Recovery time/follow-up time | 39 days                        | 41 days                        | 47 days                        | 11 months                      | 8 months                       | 55 days                        |
| Sequelae               | N                               | N                               | N                               | N                               | N                               | N                               |

P=positive,N=negative,CNS=central nerve system, The CSF=cerebrospinal fluid, MRC= Medical Research Council grade, URI= upper respiratory tract infections, G=glucocorticoid, IVIG= intravenous immunoglobulin, AIDP= acute inflammatory demyelinating poluradiculoeneuropathy, AMAN= acute motor axonal neuropathy, AMSAN= acute motor and sensory axonal neuropathy.

Table 1 clinical feature in 12 children with asymmetry weakness (sequels)(n=12)
| Case | Age/sex(years) | Antecedent infections | Days on hospital | Nerve studies | Mechanical ventilation | Sensory nerves deficit | The muscle strength at admission(MRC) | Duration time on asymmetry weakness | Cranial nerve deficit | Dysautonomia | Complications | Pathogen detection results | Albuminocytologic dissociation on the CSF | Therapy | Recovery time/follow-up time | Sequelae |
|------|----------------|-----------------------|------------------|--------------|-----------------------|----------------------|-------------------------------------|----------------------------------|-----------------|-------------|-------------|-----------------------------|-------------------------|---------|-----------------------------|---------|
| 7    | 1/F            | fever                 | 41               | equivocal    | P                     | N                    | right upper limbs: 3+/5, left upper limbs: 2+/5, right lower limb: 1/5, left lower limb: 1+5/5 | persistent asymmetry weakness | N                | N           | N           | N                           | N                      | IVIG+G  | 4 years                     | Right upper limbs: 5/5, left upper limbs: 3+/5, right lower limb: 3/5, left lower limb: 4/5 |
| 8    | 4/M            | fever                 | 37               | AMSAN        | P                     | N                    | left upper limb: 3/5, right upper limb: 5/5, bilateral lower limbs: 3/5 | persistent asymmetry weakness | P                | N           | N           | N                           | N                      | IVIG+PE | 19 months                   | Left limbs: 3/5, right limbs: 5/5 |
| 9    | 5/M            | Fever                 | 32               | AMAN         | P                     | N                    | left limbs: 2/5, right limbs: 3+/5 | persistent asymmetry weakness | N                | N           | Anti-GD1b    | Mycoplasma pneumoniae       | N                      | IVIG+PE | 37 months                   | Bilateral upper limbs: 3/5, bilateral lower limbs: 4/5 |
| 10   | 7/F            | Fever,cough           | 34               | AMAN         | P                     | N                    | bilateral upper limbs: 2/5, left lower limb: 1/5, right lower limb: 3+/5, right lower limb: 5/5 | persistent asymmetry weakness | N                | N           | Anti-GM1     | Cytomegalovirus             | N                      | IVIG+G  | 28 months                   | Bilateral upper limbs: 3/5, bilateral lower limbs: 4/5 |
| 11   | 4/M            | fever                 | 84               | AMAN         | P                     | N                    | bilateral upper limbs: 2/5, left lower limb: 0/5, right lower limb: 3+/5, left lower limbs: 4/5 | persistent asymmetry weakness | N                | N           | Anti-GM1     | CNS infection, bronchopneumonia | N                      | IVIG+PE+G | 16 months                   | Right upper limb: 4/5, left upper limb: 2/5, right lower limb: 4+/5, left lower limbs: 5/5 |
| 12   | 2m#/M          |                       | 36               | AMAN         | P                     | N                    | right upper limb: 1/5, left upper limb: 0/5, right lower limb: 3+/5, left lower limbs: 4/5 | persistent asymmetry weakness | N                | N           | Two circle of IVIG         |                           |         | 5 years                     |                                     |

This table summarizes the clinical characteristics of 12 cases, including age, sex, antecedent infections, days on hospital, nerve studies, mechanical ventilation, sensory nerves deficit, the muscle strength at admission, duration time on asymmetry weakness, cranial nerve deficit, dysautonomia, complications, pathogen detection results, albuminocytologic dissociation on the CSF, therapy, recovery time/follow-up time, and sequelae.
P=positive,N=negative,CNS=central nerve system, The CSF=cerebrospinal fluid, 2m²=2 months, MRC= Medical Research Council grade,PE=plasma exchange, G=glucocorticoid, IVIG= intravenous immunoglobulin. MRC= Medical Research Council grade. AIDP= acute inflammatory demyelinating poluradiculoneuropathy, AMAN= acute motor axonal neuropathy, AMSAN= acute motor and sensory axonal neuropathy.

Table 1 clinical feature in 12 children with asymmetry weakness (n=12)

3.3 Comparative between children with asymmetry weakness and symmetry weakness

12 children(12/72,16.67%) had asymmetry weakness. Compared to symmetry weakness group, asymmetry weakness had more preschool children, longer days on hospital, more MV, higher DSS, more axonal type, more complications and sequelae (P<0.05)(Table 2).

Table 2 clinical feature of asymmetrical weakness and symmetrical weakness(n=72)

|                      | Asymmetrical weakness(n=12) | symmetrical weakness(n=60) | T/χ² | P    |
|----------------------|-----------------------------|----------------------------|------|------|
| Median age(month)    | 51.25±33.54                 | 99.98±33.46                | 4.604| 0.000|
| Male:female          | 9:3                         | 36.24                      | 0.947| 0.331|
| Preschool children   | 9(75%)                      | 15(25.00%)                 | 7.887| 0.005|
| Antecedent events    | 6(50.00%)                   | 43(71.65%)                 | 2.129| 0.145|
| Days on hospital     | 26.5(15-37)                 | 11(9-15)                   | 4.393| 0.000|
| Mechanical ventilation| 6(50.00%)                  | 4(8.33%)                   | -6.079| 0.000|
| Sensory nerves deficit| 1(1.67%)                   | 11(18.33%)                 | 0.710| 0.399|
| Cranial nerves deficit| 4(33.33%)                  | 12(20%)                    | 1.014| 0.314|
| Dysautonomia         | 3(25.00%)                   | 11(18.33%)                 | 0.280| 0.597|
| AIDP                 | 5(41.67%)                  | 40(66.67%)                 | 2.565| 0.010|
| Axonal type(AMAN,AMSAN)| 6(50.00%)                  | 9 (15.00%)                 | 6.166| 0.013|
| Equivocal type       | 1(8.33%)                    | 11 (18.33%)                | 0.710| 0.399|
| ganglioside antibody | 4(33.33%)                   | 14(23.33%)                 | 0.526| 0.468|
| Complication         | 7(58.33%)                   | 5(8.33%)                   | 17.750| 0.000|
| therapy              |                             |                            |      |      |
| Monotherapy          | 7(58.33%)                   | 44(73.33%)                 |      |      |
| Combination therapy  | 5(41.67%)                  | 6(10.00%)                  | 5.742| 0.017|
| Sequelae             | 5(41.67%)                  | 3(5.00%)                   | 13.423| 0.000|

AIDP= acute inflammatory demyelinating poluradiculoneuropathy, DSS=Disease severity score.

Table 2 clinical feature of asymmetry weakness and asymmetry weakness(n=72)

3.4 Comparative between children with sequelae and good prognosis group

Eight children had sequelae and sixty-four children had good prognosis. Compared to good prognosis group, sequelae group had younger age at the onset of disease, longer days on hospital, more MV, more axonal subtype, more persistent asymmetry weakness and complications(P<0.05)(Table 3).

Table 3 clinical feature between children with sequelae and good prognosis group (n=72)
Axonal type at least as a late-stage feature with poor outcome was suggested to be responsible for prolonged motor symptoms, persistent asymmetry weakness and the predominantly inflammatory cell infiltrates comprising lymphocytes and macrophages in the spinal cord of GBS. Axonal GBS had positive ganglioside antibodies or involvement of sensory nerves which favor GBS diagnosis. Secondly, there were increased axonal GBS and involvement of anterior horn cells in the spinal cord were hardly be distinguished from each other sometimes. In our cases, axonal type in nerve conduction, persistent asymmetry weakness and long-time or incomplete recovery. In addition, it should be recognized that hypothesis, there was a virus infection which can bring involvement of peripheral nerves and anterior horn cells in the spinal cord, causing asymmetry weakness, reduced compound muscle amplitude potentials in motor nerve conduction, and long-time or incomplete recovery. In our cases, anterior horn cells involvement and persistent asymmetry weakness in our axonal cases: central nerves infectious, persistent asymmetry weakness had more serious and complex conditions during disease. Monotherapy had limited effect and combination therapy was necessary to improve prognosis. We also made a comparative between GBS with or without sequelae which indicated persistent asymmetry weakness was related to sequelae.

We also compare clinical features and auxiliary examinations between asymmetry weakness and asymmetry weakness in GBS. Preschool children had more asymmetry weakness than school-age children. The diagnosis of GBS should be carefully evaluated in preschool children as more atypical clinical presentations were reported. MV, axonal type as well as combination therapy were more reported in asymmetry weakness. Longer days on hospital, higher DSS in motor function, more complications and sequelae were also reported in asymmetry weakness. Our datas indicated that asymmetry weakness had more serious and complex conditions during disease. Monotherapy had limited effect and combination therapy was necessary to improve prognosis. We also made a comparative between GBS with or without sequelae group which indicated persistent asymmetry weakness was related to sequelae.

Also, 5 patients (5/6, 83.33%) with persistent asymmetry weakness were axonal type. There were several possible interpretations of persistent asymmetry weakness and sequelae in axonal type. First of all, virus infection could cause direct or indirect damage in anterior horn cells in the spinal cord which was the most common asymmetry weakness in neuromuscular disease. There were several common features between anterior horn cells in the spinal cord involvement and persistent asymmetry weakness in our axonal cases: central nerves infectious, persistent asymmetry weakness, reduced compound muscle amplitude potentials in motor nerve conduction, and long-time or incomplete recovery. In our hypothesis, there was a virus infection which can bring involvement of peripheral nerves and anterior horn cells in the spinal cord, causing axonal type in nerve conduction, persistent asymmetry weakness and long-time or incomplete recovery. In addition, it should be recognized that axonal GBS and involvement of anterior horn cells in the spinal cord were hardly be distinguished from each other sometimes. In our cases, axonal GBS had positive ganglioside antibodies or involvement of sensory nerves which favor GBS diagnosis. Secondly, there were increased inflammatory cell infiltrates comprising lymphocytes and macrophages in the spinal cord of GBS. Impairment of the spinal anterior horn cells with their axons was suggested to be responsible for prolonged motor symptoms, persistent asymmetry weakness and the predominantly axonal type at least as a late-stage feature with poor outcome.

|                              | Sequele cases (n=8) | Recovery cases (n=64) | T/χ² | P   |
|------------------------------|---------------------|-----------------------|------|-----|
| Median age(month)            | 67.25±35.10         | 94.94±37.40           | 1.986| 0.051|
| Male:female                  | 5:3                 | 37.27                 | 0.063| 0.801|
| Preschool children           | 5/62.5%            | 19/29.69%            | 3.397| 0.065|
| Antecedent events            | 5/62.5%            | 44/68.75%            | 0.126| 0.723|
| Days on hospital             | 33(15-41)           | 12(9-15)              | 3.572| 0.000|
| Mechanical ventilation       | 6/75%              | 4/6.25%              | 28.623| 0.000|
| Cranial nerves deficit       | 4(50.00%)          | 12(18.75%)           | 3.962| 0.047|
| Dysautonomia                 | 3 (37.5%)          | 11(17.19%)           | 1.847| 0.174|
| Axonal type                  | 5/62.5%            | 10/15.63%            | 9.432| 0.002|
| Persist asymmetrical weakness| 5/62.5%            | 3(4.69%)             | 23.730| 0.000|
| Complications                | 4(50.00%)          | 8(12.50%)            | 7.100| 0.008|

Table 3 clinical feature between children with sequelae and good prognosis group (n=72)

Discussion

In our study, 16.67% (12/72) children had asymmetry weakness on admission and this data was lower than previous report which included chronic inflammatory demyelinating polyradiculoneuropathy. GBS is a generalised but unevenly distributed illness. In six cases, asymmetry weakness reappeared within 4 weeks, indicating transient asymmetry weakness at admission. In early stage of disease, demyelination was patchy in motor nerves which indicated normal to various abnormalities in electrophysiology. Also, demyelination parameters and motor nerves involvement were in varying degrees. Distal motor latency and conduction block were the most common abnormal parameters in AIDP and median nerve was the most frequently affected nerve. In axonal GBS, reduced motor amplitudes was the most common parameter, and the most frequently abnormal nerve was tibial nerve. In another investigation, ulnar, peroneal nerve abnormal and sural-sparing pattern were detected at very early stage. Several early and sensitive parameters in AIDP, such as medial plantar sensory response or erb's point stimulation method, can be used in the early stage of GBS, as parasthesias often involved the medial plantar nerve or proximal nerve was the first involvement place. The neurophysiological and pathological findings of AIDP indicated that demyelination of motor nerves is focal and patchy and most of transit asymmetry weakness were AIDP. Persistent asymmetry is one of the features casting doubt or excluding other possible diagnosis. As no reliable serological marker of GBS is currently available, long-time follow-up is necessary to exclude other possible diagnosis. In our asymmetry weakness, at least one year follow-up time was given to exclude other diagnosis.

We also compare clinical features and auxiliary examinations between asymmetry weakness and asymmetry weakness in GBS. Preschool children had more asymmetry weakness than school-age children. The diagnosis of GBS should be carefully evaluated in preschool children as more atypical clinical presentations were reported. MV, axonal type as well as combination therapy were more reported in asymmetry weakness. Longer days on hospital, higher DSS in motor function, more complications and sequelae were also reported in asymmetry weakness. Our datas indicated that asymmetry weakness had more serious and complex conditions during disease. Monotherapy had limited effect and combination therapy was necessary to improve prognosis. We also made a comparative between GBS with or without sequelae group which indicated persistent asymmetry weakness was related to sequelae.
As we all known, with deeper and further investigations, many atypical features and variants in GBS were reported. More and more investigations had found that central nerve and peripheral nerves can both involved in GBS, such as encephalopathy, seizure concurrent with GBS\textsuperscript{[18,19]}. Also, peripheral nerves involvement could progress to anterior horn cells in spinal cord in GBS, either by inflammatory cells infiltrated or virus infection.

In conclusion, asymmetry weakness had two types in GBS, including transient and persistent asymmetry weakness. Asymmetry weakness in GBS indicated more complex condition during disease. Persistent asymmetry weakness in GBS had related with sequelae. Anterior horn cells in the spinal cord involvement may be the possible function in persistent asymmetry weakness in GBS.

**Abbreviations**

GBS = Guillain-Barré syndrome  
DSS = Disease severity score  
AIDP = acute inflammatory demyelinating polyradiculoneuropathy  
AMAN = acute motor axonal neuropathy  
AMSAN = acute motor and sensory axonal neuropathy  
MRC = Medical Research Council  
URI = upper respiratory tract infections  
CSF = cerebrospinal fluid  
CNS = central nerve system

** Declarations**

Ethics approval and consent to participate

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. We had ethics approval and consent to participate.

The study was approved by the ethical committee of Wuhan Children's Hospital. Written informed consent was obtained from their parents of patient in our study.

**Competing interests**

None of the authors have any conflicts of interest to disclose.

**Availability of data and material**

The datas in report had availability of data and material.

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**Authors' contributions**

Sun Rui-di designed the study, performed the research, and wrote the paper.

Jiang Jun analysed data,

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**Consent for Publication**
Images and clinical details of patient were all in compromise anonymity and they consent for the publication of identifying that. A statement of consent was obtained from the patients’ guardians to agree to use images or other personal/clinical details.

Reference

[1] Yuki N, Hartung HP. Guillain-Barre´ syndrome. N Engl J Med 2012;366:2294-2304.
[2] Rabie M, Nevo Y. Childhood acute and chronic immunemediated polyradiculoneuropathies..

Eur J Paediatr Neurol 2009;13:209-218

[3] Yosha-Orpaz N, Aharoni S, Rabie M, Nevo Y. Atypical Clinical Presentations of Pediatric Acute Immune-Mediated Polyneuropathy. J Child Neurol 2019;34:268-276.

[4] Logullo F, Manicone M, Di Bella P, Provinciali L. Asymmetry Guillain-Barré syndrome. Neurol Sci . 2006;27:355-359.

[5] Asbury AK, Comblath DR. Assessment of Current Diagnostic Criteria for Guillain-Barre Syndrome. Ann Neurol 1990;27:S21-24.

[6] Greenwood RJ, Newsom-Davis J, Hughes RA, Aslan S, Bowden AN, Chadwick DW et al. Controlled trial prednisolone in acute polynuropathy. Lancet 1978:750-753.

[7] Samadi M, Kazemi B, Golzari Oskoui S, Barzegar M. Assessment of Autonomic Dysfunction in Childhood Guillain-Barré Syndrome. J Cardiovasc Thorac Res 2013;5:81-85.

[8] Rajabally YA, Durand MC, Mitchell J, Orlikowski D, Nicolas G. Electrophysiological diagnosis of Guillain-Barré syndrome subtype: could a single study suffice?. J Neurol Neurosurg Psychiatry. 2015;86:115-9.

[9] Roodbol J, de Wit MY, van den Berg B, Kahlmann V, Drenthen J, Catsman-Berrevoets CE, et al. Diagnosis of Guillain–Barre´ syndrome in children and validation of the Brighton criteria. J Neurol 2017;264:856-861.

[10] Brown WF, Snow R. Patterns and severity of conduction abnormalities in Guillain-Barré syndrome. J Neurol Neurosurgery Psychiatry 1991;54:768–774.

[11] Rajabally YA, Hiew FL. Optimizing electrodiagnosis for Guillain-Barré syndrome: clues from clinical practice. Muscle Nerve 2017;55:748-751.

[12] Jin J, Hu F, Qin X, Liu X, Li M, Dang Y, et al. Very Early Neuropysiological Study in Guillain-Barre Syndrome. Eur Neurol 2018;80:100-105.

[13] Ahdab R, Ayache S, Noureldine MHA, Nordin T, Lefaucheur JP. The medial plantar sensory response: A sensitive marker of acute inflammatory demyelinating polyneuropathy. Clin Neurophysiol. 2017;128:2122-2124.

[14] Ye Y, Zhu D, Liu L, Wang K, Huang K, Hou C. Electrophysiological measurement at Erb's point during the early stage of Guillain-Barre syndrome. J Clin Neurosci 2014;21: 786-789.

[15] Roodbol J, de Wit MC, Walsgaard C, Hoog M, Catsman-Berrevoets CE, Jacobs BC. Recognizing Guillain-Barré syndrome in preschool children. Neurology 2011;76:807-10.

[16] Leis AA, Stokic DS. Neuromuscular manifestation of West Nile virus infection. Front Neurol. 2012;21:37.

[17] Müller HD, Beckmann A, Schröder JM. Inflammatory infiltrates in the spinal cord of children with Guillain-Barré syndrome. Acta Neuropathol 2003;106:509-17.

[18] Panda PK, Sharawat IK. Seizure in a child with Guillain-Barre syndrome: Association or coincidence. Indian Pediatr 2020; 57(1):79.

[19] Rigamonti A, Basso F, Scaccabarozzi C, Lauria G. Posterior reversible encephalopathy as the initial manifestation of a Guillain-Barre syndrome. Neuromuscul Disord 2009;19:709-71