# Clinical features and prognosis with Guillain-Barré syndrome

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## Abstract

**Background:** Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy commonly characterized by rapidly progressive, symmetric weakness and areflexia. **Materials and Methods:** We retrospectively assessed the clinical manifestations, results of electrodiagnostic tests, functional status and prognosis of 36 children diagnosed with GBS. **Results:** Based on clinical and electrophysiological findings, the patients were classified as having acute inflammatory demyelinating polyradiculoneuropathy (AIDP) ($n$ = 25), acute motor axonal neuropathy (AMAN) ($n$ = 10) and acute motor-sensory axonal neuropathy (AMSAN) ($n$ = 1). Twenty (55.5%) patients were males and 16 (44.5%) patients were females. The mean age of the 36 patients was 68.1 ± 45.01 months (range, 6–180 months). Five (13.8%) patients were younger than 2 years. The most common initial symptoms were limb weakness, which was documented in 34 (94.4%) patients. In our study, 18 patients (51.4%) showed albuminocytological dissociation (raised protein concentration without pleocytosis) on cerebrospinal fluid (CSF) examination. Three patients (8.3%) required mechanical ventilation therapy during hospitalization. Unfortunately, three (8.3%) patients died; one patient had AIDP and two patients had axonal involvement (one case was AMAN and another case was AMSAN). When we compared the cases of residual sequel/dead and cases of complete recovery for neural involvement type including AIDP, AMAN and AMSAN, we did not find a statistically significant difference between the groups ($P$ > 0.05). **Conclusion:** Our findings showed that cases of GBS was not uncommon in children younger than 2 years of age, and CSF protein level might be found high in the first week of the disease in about one half of the patients, with a higher rate of morbidity and mortality in patients with axonal involvement than in those with AIDP.

## Key Words

Children, Guillain-Barré syndrome, Electromyography

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## Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy most commonly characterized by a rapidly progressive, essentially symmetric, ascending flaccid paresis, weakness and areflexia.[1-3] The incidence of typical GBS has been reported to be relatively uniform between 0.6 and 4 cases per 100,000 per year throughout the world.[4] GBS has been frequently reported to be preceded by a nonspecific infection of variable type, usually a few weeks before the onset of neurological symptoms; other suggested triggering factors include trauma, surgery or vaccination.[5] Multifocal segmental demyelization is the main underlying pathology of the disease.[6]

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Based on clinical features, etiology, pathologic and electrophysiologic studies, GBS may be subclassified into several forms. These are acute inflammatory demyelinating polyradiculoneuropathy (AIDP); axonal forms of GBS, which include acute motor-sensory axonal neuropathy (AMSAN) and acute motor axonal neuropathy (AMAN); and Miller Fisher syndrome (MFS).[7,8]

In this article, the clinical and laboratory features of 36 GBS cases were reviewed to determine the prognostic factors in childhood GBS.

## Materials and Methods

The hospital records of children with GBS (age under 16 years) managed at Yuzuncu Yil University Faculty of Medicine, Department of Pediatrics between 2003 and 2009 were reviewed, retrospectively. The patients were examined for the following

parameters: Age, sex, personal history, seasonal preponderance, physical examination and laboratory findings, including cerebrospinal fluid (CSF) and electromyography (EMG), need for mechanical ventilation, applied specific treatment and prognosis. The diagnosis of acute GBS was based on the following criteria: An acute progressive symmetric weakness of the extremities with areflexia or hyporeflexia, CSF showing albuminocytological dissociation and electrophysiology revealing features of demyelinating/axonal neuropathy.[9] Albuminocytological dissociation was defined as CSF with raised protein and total cell count of ≤10/mm$^3$.

The studies of nerve conduction velocity were performed within 24–48 h of hospitalization in all cases of GBS. Needle EMG was also performed. At least one motor and one sensory nerve was tested on the upper and lower limbs. F response was recorded in all the extremities. Additionally, routine motor conduction studies were performed on the median, ulnar and tibial nerves using conventional procedures. Sensory nerve studies were performed on the median and sural nerves. The amplitude of the negative phase was measured for compound muscle action potentials and sensory nerve action potentials. In our study, the patients were classified into AIDP or AMAN based on the existing electrodiagnostic criteria.[10]

AMSAN was defined as the presence of AMAN pattern in motor nerve studies with sensory nerve action potential amplitude reduction more than 50% of the normal in two or more sensory nerves.

In all patients, neurological findings were recorded from medical records. Additionally, onset of weakness, associated or preceding events and progression of the disease were recorded. The patients' disabilities were evaluated using the functional grading scale of Hughes *et al.* [Table 1].[11]

Statistical analysis was performed using the commercial program SPSS 17. Mann-Whitney *U* test was used to compare the data of the groups. Correlation analysis was performed using the Spearman correlation test.

## Results

Clinical and laboratory findings of the patients are described in Table 2. According to the clinical and electrophysiological findings, 25 (69.4%) patients manifested AIDP, 10 (27.8%) AMAN and one (2.8%) AMSAN. Twenty (55.5%) patients were males and 16 (44.5%) patients were females. The mean age of the 36 patients was 68.1 ± 45.01 months (range, 6–180 months). Five (13.8%) patients were younger than 2 years. Fifteen (41.6%) patients developed GBS during the summer (June to August) and nine (25%) during the fall (September to November).

**Table 1: Scale of Hughes**

| | |
|---|---|
| Grade 0 | Normal functional state |
| Grade 1 | Able to run with minor signs and symptoms |
| Grade 2 | Able to walk 5 m independently |
| Grade 3 | Able to walk 5 m with aid |
| Grade 4 | Bed- or chair-bound |
| Grade 5 | Requires assisted ventilation |

Duration between beginning of symptoms and admission to hospital was 5.6 ± 6.1 days (range, 1–30 days). Eleven (30.5%) patients had no identifiable preceding infection or event within 2 weeks prior to GBS onset, 13 (36.1%) had acute upper respiratory infections and 12 (33.3%) had acute gastroenteritis. The most common initial symptoms were limb weakness, which was documented in 34 (94.4%) patients. Other symptoms included muscle pain in eight patients, dysarthria in three, numbness in two, meningeal signs in two and facial palsy in one patient. Urinary dysfunction was noted in two (5.5%) patients with AIDP, whereas no patients had urinary dysfunction in both AMAN and AMSAN. Other autonomic dysfunction symptoms included hypertension in one patient and tachycardia in eight patients.

In 35 patients, a lumbar puncture was performed within the first day of admission. The CSF cell count was normal (<10 cells/mm$^3$) in all the patients. Duration between beginning of symptoms and admission was ≤7 days in 29 patients (3.3 ± 2 days) (range, 1–7 days) in those in whom the CSF protein level was 60.6 ± 54.3 mg/dL (range, 18–199 mg/dL). Of 29 patients, 14 (48.2%) patients had a high CSF protein level (>45 mg/dL). The admission duration was >7 days in six patients (17.2 ± 14.2 days) (range, 10–30 days) in whom the CSF protein level was 67 ± 55.2 mg/dL (range, 8–161 mg/dL). Of six patients, two patients had a low CSF protein level (<45 mg/dL). We did not find a statistically significant difference for CSF protein level between patients admitted ≤7 days and >7 days to the hospital ($P > 0.05$). While there was a positive correlation between CSF protein level and admission duration in the patient group admitted ≤7 days ($r = 0.486$, $P < 0.05$), we did not find a correlation in the patient group admitted >7 days ($r = 0.116$, $P > 0.05$).

The mean hospitalization period was 5.5 ± 3.6 days (range, 1–17 days). Three patients (8.3%) required mechanical ventilation therapy during hospitalization. In addition to supportive management, 34 (94.4%) patients received intravenous immunoglobulin (IVIG) alone and two patients received high-dose methylprednisolone alone because IVIG could not be obtained in Turkiye during that time. A good outcome with normal functional life was noted in 29 (80.5%) patients. Recovery from IVIG was noted between 3 and 12 days after initiation of treatment. Twenty-two children with AIDP and seven children with AMAN were able to walk independently by 6 months after onset. The remaining two children with AIDP and two children with AMAN were able to walk independently after 1 year from onset of GBS. In these four patients, the Hughes scale grade was 1 after 1 year, and persistent ataxia did remain as the residual sequel. A relapse was noted in a child successfully treated with IVIG with AIDP 2 months after the first attack. Unfortunately, three (8.3%) patients died; one patient had AIDP and two patients had axonal involvement (one case was AMAN and the other case was AMSAN). The causes of death included sudden cardiac arrest, respiratory insufficiency and pneumonia. Three (12%) patients with AIDP and four (40%) patients with axonal involvement resulted from residual sequel/death. When we compared the residual sequel/death and complete recovery cases for neural involvement type including AIDP, AMAN and AMSAN, we did not find a statistically significant difference between the groups ($P > 0.05$).

**Table 2: Clinical and laboratory findings of the patients**

| Parameter | AIDP (*n* = 25) | Axonal involvement (*n* = 11) | |
|---|---|---|---|
| | | AMA (*n* = 10) | AMSAN (*n* = 1) |
| Gender | 14 male<br>11 female | 6 male<br>4 female | 1 female |
| Age (year) | 0.5–15 | 1.5–10 | 4.5 |
| Application season | | | |
| Summer | 10 | 5 | - |
| Fall | 5 | 4 | - |
| Winter | 2 | 1 | 1 |
| Spring | 8 | - | - |
| Duration between and admission onset of weakness (day) | 1–20 | 1–30 | 10 |
| Antecedent infection | | | |
| Upper respiratory tract infection | 9 | 4 | - |
| Acute gastroenteritis | 8 | 3 | 1 |
| Initial symptoms/signs | | | |
| Limb weakness | 23 | 10 | 1 |
| Muscle pain | 6 | 2 | - |
| Numbness | 2 | - | - |
| Facial palsy | 1 | - | - |
| Dysarthria | 2 | 1 | - |
| Meningeal signs | 1 | - | 1 |
| Areflexia or reduced tendon reflexes | 24 | 10 | 1 |
| Autonomic dysfunction | | | |
| Hypertension | 1 | - | - |
| Micturition disorder | 1 | - | - |
| Tachycardia | 4 | 3 | 1 |
| CSF findings | | | |
| Proteins (mg/dL) | 12.5–199 | 19–199 | 8 |
| Cell count | 0–1 | - | - |
| Treatment | | | |
| IVIG | 24 | 9 | 1 |
| HDMP | 1 | 1 | - |
| Required mechanical ventilation | 1 | 1 | 1 |
| Duration of hospitalization (day) | 3–17 | 1–5 | 1 |
| Prognosis | | | |
| Complete recovery | 21 | 7 | - |
| Residual deficit | 2 | 2 | - |
| Relapse | 1 | - | - |
| Death | 1 | 1 | 1 |

HDMP = High-dose methylprednisolone; IVIG = Intravenous immunoglobulin; AIDP = Acute inlammatory demyelinating polyradiculoneuropathy; AMSAN = Acute motor-sensory axonal neuropathy; AMAN = Acute motor axonal neuropathy.

## Discussion

Although the occurrence of GBS in children is relatively rare, it is the most common cause for the development of acute flaccid paralysis among infants and children.[7] Since the first report of GBS in childhood by Mannier-Vinard in 1925, showing an incidence of 0.24–1.26 per 100,000 children under 15 years of age,[9] the age-specific incidence was 1.26 in 100,000 in the 1–4 years age group and 0.24 in 100,000 in the 5–9 years age group.[12] GBS has a worldwide distribution and affects all races and all ages, including the newborn.[13] Gender ratios in individual reports in the literature vary from 1.5 to 2.7 males for one female.[13] The gender ratio in our series was 1.3 in favor of males. The occurrence of GBS in children increases with age, and it is quite rare in children younger than 2 years of age.[7] In our study, five cases were younger than 2 years of age. Of five cases, three and two cases were AMAN and AIDP, respectively.

GBS is frequently associated with a preceding illness, such as upper respiratory infection or acute enterocolitis. Although respiratory illness suggests seasonal predominance, the relationship between incidence of GBS and seasonal change was not determined.[14] In a study from Northern China, a seasonal predominance was evident in the summer months among children and young adults.[15] Our study revealed a seasonal predominance in summer and spring, accounting for 41.6% and 22.2% respectively of the episodes, especially AIDP appeared more common in that period.

Limb weakness, especially in the distal part of the lower extremities, was the most prevalent symptom associated

with hospitalization.[14] We found that limb weakness was the initial symptom in 34 (94.4%) of 36 patients, a relatively high percentage.

CSF is characteristically acellular. Protein levels may be normal during the first week of the illness, but the majority will have an increase in protein if measured 2 or 3 weeks later. Elevated CSF protein concentration in GBS has been mainly associated with increased permeability of the blood–CSF barrier.[13,16] In our series, 18 (51.4%) patients showed albuminocytological dissociation (raised protein concentration without pleocytosis) on CSF examination. We did not find a statistically significant difference for CSF protein level between patients admitted ≤7 days and >7 days to the hospital ($P > 0.05$). However, we found a positive correlation between CSF protein level and admission duration in the patient group admitted ≤7 days ($r = 0.486$, $P < 0.05$).

Electrodiagnostic findings have been used to classify childhood GBS according to types as well as to determine the relationship of each with the severity of clinical features or long-term functional status. There have been a number of reports on the incidence of AMAN. The incidence of the AMAN form of GBS varies considerably, from <10% of the patients with GBS in Western countries to over 40% in East Asia and, in these countries, it frequently affects children.[1,17,18] AMAN is characterized by rapidly progressive weakness, often with respiratory failure and usually good recovery, while AMSAN is generally associated with slow and incomplete recovery.[19] The severity of residual impairment has been found to be related to the degree of AMAN, as shown by electrodiagnostic tests, and to the severity of the clinical symptoms at nadir.[20] Electrodiagnostic criteria predictive of poor outcomes in adults, such as low amplitudes of motor responses and abnormal spontaneous activities, are relatively common and have a limited prognostic value in pediatric GBS.[21] In a Japanese study, the proportion of children with AIDP (35%) was similar to that of children with AMAN (48%), and recovery was generally favorable in both subtypes.[22] When pediatric GBS patients were classified according to axonal or myelin involvement, their functional status 12 months after onset did not differ and was good in both, and a similar study showed that the duration of ventilatory support did not differ significantly.[23,24] We found that 69.4% of the patients was AIDP type, 27.8% AMAN type and 2.8% AMSAN type. Our results showed that the AIDP form was more frequent than the AMAN and AMSAN forms. There have been a number of reports on the incidence of AIDP and AMAN in childhood GBS. The AMAN pattern was found in 65–86% in China,[25,26] 30% in Argentina[27] and 35% in Turkiye.[23] In contrast, a number of reports showed that in North America, most GBS children appear to have AIDP, although these reports did not employ the same electrodiagnostic criteria for AIDP or AMAN as used in this study.[22] These findings suggest that the incidence of AMAN in childhood GBS varies considerably among countries, as it does in the adult population. The reason is unknown, but preceding infectious agents and host factors may be responsible, as postulated for adult GBS.[22]

The autonomic nervous system was reported to be involved in 25% of the GBS patients, usually manifesting as blood pressure instability, sinus tachycardia, pupillary abnormality or sweating abnormality.[28] Urinary dysfunction was found in 21% of the patients with classic GBS but in 50% of those with axonal GBS, but was very rare in MFS and AMSAN.[14] In our series, urinary dysfunction was noted in two (5.5%) patients with AIDP. The mechanism of urinary dysfunction was not well known. Bladder areflexia and disturbed bladder sensation are considered to be common patterns, but nonrelaxing urethral sphincter with neurogenic change is also possible.[29]

Optimal management and treatment of GBS is critically important because the stakes are life or death. Although many patients with GBS are desperately ill and paralyzed, their chances of a full recovery are high if they can overcome the acute stages. Thus, an important aspect of treatment is to provide maximum supportive care during the acute stages. A recent large, multicenter, randomized trial made a comparison between plasma exchange, intravenous exchange and combined treatment. Its final analysis revealed that there was no significant difference in efficacy between these three therapeutic regimens.[7] Furthermore, a retrospective multicenter study found that intravenous immunoglobulin accelerated recovery in children with GBS who were unable to walk.[30] Haass, *et al*.[31] reported improvement of GBS with the use of high-dose methylprednisolone and deterioration with a low dose. In our series, 34 (94.4%) patients received IVIG and the reminder two patients received high-dose methylprednisolone.

The only new observation with patients treated with IVIG was acute relapse in 11.9% of the patients. A relapse rate ranging from 1.4 to 46.7 was reported with use of IVIG.[32,33] In our study, the relapse ratio was 2.7%.

Studies of GBS that focused on both children and adults together found that respiratory support was required in about 20–30% of the patients.[34,35] Case fatality rates requiring mechanical ventilation for respiratory failure were estimated to be 15–30%.[7] Childhood GBS in about one-third of all patients needed ventilatory support for respiratory muscle paralysis, and about 10% of the patients died of the disease and its complications.[36] In our study, three (8.3%) patients who required ventilatory support died because of sudden cardiac arrest, respiratory insufficiency and pneumonia.

GBS in children has a shorter course and is associated with a more complete recovery than GBS in adult patients. Despite modern treatment regimens, about 10–20% of adult GBS patients continued to be disabled.[14,17,37] Moreover, older age at onset was significantly associated with a poorer outcome at 1 year.[14] In a retrospective study including adult patients in Taiwan, 12.5% of the patients remained at Hughes scale grade 4–6 after 1 year.[19] In contrast, although approximately 40% of the children became nonambulant during their illness and 15–20% required ventilatory support, more than 90% recovered fully, with a small minority showing minimal residual impairment, such as weakness of the ankle dorsiflexor 1–4 months after onset, but were able to walk unaided.[21,30] After 1 year, only 14.3% of the pediatric GBS patients needed assistance in walking.[7] Moreover, about 72% of the children with GBS could walk independently 1 year after onset, more than twice the percentage of adults.[38] In our study, all patients achieved independent ambulation by 1 year after onset of the disease.

As in most retrospective studies, our study also has some limitations, which are as follows. In our series, the number of

patients is not high. Therefore, we could not statistically compare the subgroups of GBS. Secondly, it would be beter if the patients who were admited early (admission duration was <7 days) had performed repeated lumbar puncture. None of the patients had any electrophysiological examination during their follow-up.

In conclusion, our findings showed that cases of GBS was not uncommon in children younger than 2 years of age, and CSF protein level might be found high in the first week of the disease in about one half of the patients. Patients with axonal involvement showed more severe clinical progression than those with AIDP.

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