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Guillain Barre syndrome in childhood: a case series

Çocukluk çağında Guillain Barre sendromu: Bir olgu serisi

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ABSTRACT

Guillain Barre Syndrome (GBS) is an acute inflammatory polyneuropathy characterized by rapidly progressive, ascending, symmetric weakness and areflexia. It is the most common cause of acute flask paralysis in childhood. It is frequently characterized by progressive weakness, associated mild sensory symptoms, and albuminocytologic dissociation emerged in the cerebrospinal fluid often occurs within several days or weeks afternon specific infection. The neurological deficit progresses in days and months.

In this study, a total of seven GBS cases with different clinical manifestations admitted to our clinic between 2016 and 2018 were presented. Two patients were diagnosed with acute motor axonalneuropathy (AMAN), two patient with acute inflammatory demyelinating polyneuropathy, one patient with Miller Fischer syndrome, one patient with Bickerstaff's brainstemencephalitis and one patient with atypical Guillain Barre syndrome. The cases with AMAN necessitated respiratory support and ventilator-associated pneumonia and tension pneumothorax developed in one of them. All cases healed with out any sequels.

Keywords: Guillain-Barré Syndrome, childhood, AMAN, AIDP, MFS, BBE

ÖZET

GuillainBarre Sendromu (GBS) hızla ilerleyen, asendan yerleşimli, simetrik zayıflık ve arefleksi ile karakterize akut inflamatuar bir polinöropatidir. Çocukluk çağında akut flask paralizinin en sık nedenidir. Sıklıkla ilerleyici güçsüzlük, ilişkili hafif duyusal semptomlar ve spesifik olmayan enfeksiyondan birkaç gün veya hafta sonra beyin omurilik sıvısında ortaya çıkan albüminositolojik ayrışma ile karakterizedir. Nörolojik defisit günler ve aylar içinde ilerler.

Bu çalışmada 2016 ila 2018 yılları arasında kliniğimize başvuran farklı klinik bulgulara sahip toplam 7 GBS olgusu sunuldu. İki hastada akut motor aksonalnöropati (AMAN), iki hastada akut inflamatuar demiyelinizan polinöropati, bir hastada Miller Fischer sendromu, bir hastada Bickerstaff beyin sapı ensefaliti ve bir hastada atipik GBS tanısı konuldu. AMAN' lı olgularda solunum desteği gerekti ve bunlardan birinde ventilatör ilişkili pnömoni ve tansiyon pnömotoraks gelişti. Tüm olgular sekelsiz iyileşti.

Anahtar kelimeler: Guillain-Barré Sendromu, çocukluk, AMAN, AIDP, MFS, BBE

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INTRODUCTION

Guillain-Barré Syndrome (GBS) is defined as polyradiculoneuropathy characterized by symmetrical ascending paralysis. Most studies that estimate incidence rates of GBS were done in Europe and North America and showed a similar range of 0.8-1.9 (median 1.1) cases, per 100,000 people per year, and the rate of GBS increased exponentially with age [1]. Guillain-Barré Syndrome incidence in children (0-15 years old) between 0.34 and 1.34/100,000 [2]. Two-thirds of adult and pediatric patients have antecedent respiratory or gastrointestinal tract infection within four weeks of onset of weakness [3,4]. Acute myelin damage (acute inflammatory demyelinating polyradiculoneuropathy) or axonal damage (acute motor axonal neuropathy or acute motor sensoryaxonal neuropathy) mediated by the immunological mechanisms triggered by infections like Ebstein-Barr virus (EBV), Cytomegalovirus (CMV), Mycoplasma pneumonia, Campylobacter jejuni is responsible for the pathogenesis [5]. Although antibodies target myelin proteins, in some cases axonal structures become the primary target of immune-mediated damage. Typical GBS involves damages of peripheral nerves' myeline insulation where as atypical GBS was considered in case of localized or regional involvement of motor and sensoryaxons of peripheral nerves and autonomicnervous system [6].

During diagnosis process, cerebrospinal fluid (CSF) examination and electrophysiological procedures are important. Protein increase in CSF without cells (albuminocytological dissociation) is observed. Demyelinisation and axonal damages can be shown in electrophysiological studies [7].

Guillain-Barré Syndrome may pose a life threatening situation because of the developing inabi-lity in respiratory muscles after rapid paralysis and severe arrhytmias due to autonomic involve-ment [7]. Treatment consists of supportive therapies and physical therapy approaches together with immunomodulatory therapies. Intravenous immunoglobulin (IVIG) and Plasma Exchange (PE) are known to accelerate healing only when supportive care is applied [8, 9].

In this case series, seven children and adolescents diagnozed with GBS were discussed in the light of recently reported literature.

MATERIAL and METHOD

This case series was aimed to reveal seven children with GBS and their clinical and electrophysiological features. Clinical, electrophysiological and CSF findings and treatment and follow-up of seven patients who presented to our clinic with various clinical findings between 2016-2018 were evaluated retrospectively. Motor conduction studies were performed on the median, ulnar, tibial, and peroneal nerves. Sensory nerve studies were performed on the median, ulnar, and sural nerves. The amplitude of the negative phase was measured for compound muscle action potentials (CMAPs) and sensory nerve action potentials (SNAPs). F-waves were recorded from the median, ulnar, tibial and peroneal nerves. We used diagnostic criteria which were described by Rajabally et al. 10 (seeTable 1).

Table 1. Rajabally'scriteria of GBS

Rajabally'scriteria
1.Acute inflammatory demyelinating polyneuropathy (AIDP)
At leastone of thefollowing in at leasttwonerves
° MCV <70% LLN
° DML >150% ULN
° F-response latency>120% ULN, or>150% ULN (if distal CMAP <50% of LLN)
OR
° The F wave absence in two nerves with dCMAP ≥20% LLN, with an additional parameter, in one other nerve
OR
° pCMAP/dCMAP amplitüde ratio<0.7 (excluding the tibial nerve), in two nerves with an additional parameter, in one other nerve
2. Axonal GBS (includingin excitable forms)
None of the above features of demyelination in any nerve (except one demyelinating feature allowed in one nerve if dCMAP<10% LLN), and at least one of the following:
° dCMAP<80% LLN in two nerves
° F-wave absence in two nerves with distal CMAP ≥20% LLN, in absence of any demyelinating feature in any nerve
° pCMAP/dCMAP amplitüde ratio<0.7, in two nerves (excluding the tibial nerve)
° F-wave absence in one nerve with distal CMAP ≥20% LLN
OR
° pCMAP/d CMAP amplitüde ratio<0.7 (excluding the tibial nerve), in one nerve; with, **IN ADDITION**, dCMAP<80% LLN in one other nerve
Inexcitable
° If dCMAP absent in all nerves (or present in only one nerve with dCMAP<10% LLN)
3. Equivocal
Abnormal range findings how ever not fitting criteria for any other group

MCV: motor conduction velocity, DML: distal motor latency, ULN: upper limit of normal, CMAP: compound muscle action potential, pCMAP: proximal compound muscle action potential, dCMAP: distal compound muscle action potential, LLN: lower limit of normal

CASES

Case 1

A 14-year-old boy was admitted to the hospital after fever and abdominal pain, complaints of the patient disappeared after receiving antibiotic treatment, but 10 days later he complained of foot pain and not to be able t o walk. He admitted to our clinic by swallowing difficulties and unable to close his eyes. Physical examination revealed facial asymmetry, effacement of left nasolabial fold, not fully closed of left eye, right wards liding of mouth, positive corneal reflex, negative gag reflex, hypoactive patellar and Achill reflexes, bilaterally flexor plantar reflexes, with muscle strength of 3/5 of distal lower extremities. He was hospitalized with preliminary diagnosis of GBS. Electromyograph (EMG) revealed not prolonged normal F response. Transversemyelitis was excluded in the patient with normal cervical, thoracic, and lumbar magnetic resonance imaging (MRI). There was albuminocytologic dissociation (ACD) in CSF (Cell negative, protein: 1660 mg/dL). Infectious causes have been ruled out. Antibodies against ganglioside GQ1B were negative. Bickerstaff's encephalitis was considered because of there was facial peripheral paralysis, accompanied by the possibility of swallowing. IVIG was given to the patient for four days at 0.5 gr/kg/day. In the follow-up, since there was bilaterally facial paralysis and no improvement in neurological findings with IVIG treatment, plasmapheresis was performed 7 times to the patient. Acute demyelinating polyneuropathic involvement was detected in the EMG carried out 12 days later. In the fourth session of the plasmapheresis, retching reflex and deep tendon reflexes (bilaterally in the lower extremities) were obtained. Slight numbness in the right eyelid was considered to be meaningful for brainstem involvement. Plasmaferesis was completed in 7 sessions and IVIG was repeated at a dose of 0.5 mg/kg/day for four days. The patient was followed for 15 days, the neurological findings disappeared and the patient was discharged. There was no neurological deficit in the patient evaluated five months after discharge.

Case 2

A 10-year and 10-month-old boy admitted to our hospital three days later when he admitted for loss of strength and imbalance following swallowing impairment. The treatment of upper respiratory tract infection was given to the patient. He suffered from difficulty in swallowing and complained that the staff that he ate had come from the nose. There was non specific issue in his or family history. There was no facial asymmetry and no gag reflex. Deep tendon reflexes were hypoactive. Reflexes were normal and muscle strength was 4/5 on both upper and lower extremities. The patient's brain computerized tomography (CT) was normal. CSF was revealed no leukocytes and the protein was in normal range. There was no ACD finding. On follow-up bilateral rales, reduction in oxygen saturation, consolidation in chest X-ray were detected and pediatric aspiration pneumonia /GBS/ brainstem encephalitis were diagnosed and intensive care and antibiotics started. From the patient's CSF samples, anti-AChRres antibody and ganglioside panel were studied in an external laboratory and all resulted in negative results. EMG showed asymmetrically reduced F responses. Considering GBS, the patient was given IVIG for two days at a dose of 1 g/kg/day. Cranial MRI and diffusion MRI were normal. At the first day of the hospitalization, he was intubated for having respiratory complaints. Plasmapheresis was administered to the patient after IVIG. After the second plasmapheresis, IVIG was repeated. On the 9th day of the follow-up, the patient was extubated. Antibiotic therapy was completed in 10 days and then discontinued. At the 10th day of the follow-up, full oral feeding was started. Seven plasmapheresis sessions were performed with there commendation of Hematology and Neurology. IVIG was administered to the patient 6 times. Patient's CSF examination was performed resulting in no leukocytes and protein: 149 mg/dL. On the 15th day of follow-up, the patient's physiotherapy clinic was transferred to a natural neurological examination except for mild dysarthria.

Case 3

A 17 years old Syrian boy, admitted to our clinic with complaints of fever, weakness starting from feet and going upwards, and not being able to walk five days. The brain CT was normal. There was no facial asymmetry, there was no Achill reflex and patellar reflex, upper deep tendon reflexes were normoactive, muscle strength was 3/5, plantar reflex was bilaterally flexor. The patient was admitted with the diagnosis of GBS. Electromyography revealed acute motor axonal polyneuropathic involvement (AMAN) findings. Albuminocytologic dissociation was detected in CSF examination. Contrast enhancement was observed in all spinal MRI-caudafibers, which may be interpreted as significant in terms of viral GBS. The patient was given 2 g/kg IVIG. The neurological findings of the patient were regressed and muscle strength was increased. Physiotherapy and exercise were carried out. On the 10th day of the follow-up,

the patient was taken over by the physical therapy and rehabilitati on clinic.

Case 4

A 4-year and half-old boy was admitted to the external center due to fever and sore throat two weeks earlier. Antibiotics were given but his complaints were not improved. The patient admitted to our clinics with the complaint of difficulty in walking, and headache continuing for five days. There was a third-degree relation between his parents, uncledied from rheumatic disease, another uncle died due to aplastic anemia. Physical examination revealed no facial asymmetry, normoactive reflexes. There was slight weakness in the lower extremity, slight weakness in walking but could walk with out assistance. EMG showed findings that "it is compatible with the sensory-motor demyelinating polyneuropathic involvement, supporting the diagnosis of acute inflammatory demyelinating polyneuropathicinvolvement (AIDP). There was albuminocytologic dissociation in the CSF, no leukocytes, protein: 457 mg/dL. IVIG treatment was given to the patient for four days at a dose of 2 g/kg. The patient was discharged on the 7th day when the neurological findings were dismissed, the walking strength improved, and the patient was discharged.

Case 5

A 2-year and 11-month-old boy admitted to the hospital for fever one day ago. The patient was started to antibiotics, but afterward, both eyelids fell and the up-view gaze was restricted. The patient first applied to the eye doctor and then to ourclinic. There was no specific issue in his and family history. Physical examination revealed the restriction of up-view gaze. There was no facial asymmetry, deep tendon reflexes were hypoactive in the lower extremities. EMG was consisted with GBS. He was hospitalized with the diagnosis of Miller Fisher syndrome based on his physical examination. IVIG was administered to the patient in 4 sessions at a dose of 2 g/kg. There was no ACD in the LP. The patient was discharged on the basis of the neurological findings regressed after the IVIG treatment and was followed in the out patient clinic. Following-up in the out patient clinic, the restriction of the up-view of the patient was diminished but still continued. His upper and lower extremities of the deep tendon reflexes (DTR)were longer. He was consulted to the eye policlinic for his strabismus. In the follow-up of the nine-month outpatient clinic, botox was performed in the patient who septosis did not recover.

Case 6

A 14 and half-year-old boy was admitted to our hospital with complaints of weakness in the feet and hands with a history of upper respiratory tract infection three weeks before and playing basketball and falling on the pelvis one week before. Physical examination was normal except for deep tendon reflexes were not to be taken in the lower extremities, upper extremities' muscle strength was 2/5, lower extremities' muscle strength was 3/5. The patient was admitted with the diagnosis of GBS. EMG revealed at the upper right; F response was long, H reflex latencies at the top were prolonged, and the patient's left leg and the right arm involvement were excessive, thus this finding was interpreted as "atypical" due to excessive asymmetric involvement. LP was carried out and the CSF protein was found to be slightly elevated. The treatment consisted of 5 sessions of IVIG at a dose of 2 g/kg and marked improvement was detected in the muscle strength. The brain and whole spinal MRI were normal. At the 10th day of follow-up, EMG was re-examined and it was found to be markedly improved. The patient whose neurological findings were regressed was discharged.

Case 7

A 15-year-old boy was admitted to our hospital with abdominal pain, diarrhea, sluggishness, difficulty in walking, speaking and swallowing, with continuing for four days. Medical examination was normal except for there was no gag reflex, upper extremities' muscle strength was 3/5, lower extremities' muscle strength was 2/5, DTR were not able to obtain in upper extremities, while it was normal in upper extremities. Plantar reflexes were bilaterally flexor. Brain CT was normal and the patient was hospitalized with GBS diagnosis. LP findings of CSF was normal, there was no albuminocytologic dissociation. Electromyography was interpreted to be consistent with motor AMAN. IVIG treatment was given at a dose of 2 g/kg. On the first day of the hospitalization, the patient was intubated because he suffered from respiratory distress, with decreasing oxygen saturation. Antibiotic treatment was started due to pneumonia stemming from the collapse of the left upper lobe in the chest X-ray. Hypertension emerged and it was thought to be a secondary to autonomic dysfunction. Five sessions of plasmapheresis were performed after the patient's IVIG, and then IVIG was given again. At the 7th day of follow-up, pneumothorax and pneumomediastinum developed in the patient. Underwater drainage was performed by pediatric surgery. Bedside fiber-optic bronchoscopy was performed by thoracic surgery. Pneumomediastinum was planned to follow without surgical intervention. By intermittent washing via fiber optic bronchoscope, atelectasis and chronic lung development were prevented. Tracheostomy was performed on follow-up. When the swallowing function returned, oral feeding began. Pneumomediastinum, pneumothorax was strained and chest tube removed. On the 17th day of follow-up, he left from the ventilator and tracheotomy was closed. The patient whose neurological findings regressed was transferred to physiotherapy.

DISCUSSION

Accordingto electrodiagnostic study (EDx) and clinical signs patients divided into AIDP (5 patients) and AMAN (2 patients) group. One patient diagnosed as Miller Fisher syndrome and one patient diagnosed as Bickerstaff's brainstem encephalitis, two of them were in the AIDP group. All 7 cases' clinical and laboratory findings were summarized in theTable 2.

Table2. Clinical and laboratory findings of children and adolescents with GBS

	Cases						
	Case 1	**Case 2**	**Case 3**	**Case 4**	**Case 5**	**Case 6**	**Case 7**
Age (years)	14	11 y	17	4.5	3	14.5	15
Gender	Boy	Boy	Boy	Boy	Boy	Boy	Boy
EMG	Not prolonged F response	Asymmetrical reduced F	AMAN findings	AIDP findings	GBS findings	Prolonged F and H	AMAN findings
CMRI	Normal	Normal	Abnormal	NA	NA	NA	NA
ACD in CSF	Yes	No	Yes	Yes	No	Yes	No
CCT	NA	Normal	Normal	NA	NA	NA	Normal
Anti-GQ1B	Negative	NA	NA	NA	NA	NA	NA
Anti-ChR	NA	Negative	NA	NA	NA	NA	NA
Diagnosis	Bickerstaff's encephalitis	Brainstem encephalitis	Viral GBS	GBS	Miller Fisher	Atypical GBS	GBS
Treatment	IVIG	IVIG plus plasmapheresis	IVIG	IVIG	IVIG	IVIG	IVIG
Hospitalization (day)	15	15	10	7	4	10	17
Prognosis	Good	Mild dysarthria	Good	Good	Ptosis	Good	Good

One of them (Case 6) had atypical GBS features.

Kocaman et al (2005) reported in their 9 GBS cases similar with ourseries [10]. Of 9 cases with GBS, a girl and eight boys, mean age was 6.2 years and six cases of them AMAN whereas each one of the rest of was AIDP, AMSAN and Miller Fisher Syndrome, respectively. Four cases had ACD and except one children, who had AMAN, 8 of casesfully recovered from disease with in three months [11]. The time between the onset of symptoms and hospital admission was 1-13 days. In case 4 there were antecedent infections which were upper respiratory tract infection in two patients and abdominal pain in two patients. All of the patients were boy. All patients except one had the weakness. In the 5th case, there was restricted upward gaze and ptosis bilaterally. The three out of four patients with cranial nerve involvement had no gag reflex, and two of them needed intubation. The age range of the patients was 2 years 11 months-17 years.

All of the patients had EDx abnormalities (case 1's first EDx was normal, these condinvestigation showed nerve demyelination) (seeTable 3).

Table 3. Summary of electrophysiological data

Abnormal parameters	Number of patients
Distal motor latency (DML)	3/7
Distal CMAP amplitüde decrease	2/7
F-wave latency prolongation & absent	5/7 & 2/7
Conduction block	0/7
Normal values:	
Median distal motor latency (DML) <3.8 ms; ulnar DML <3.3 ms; peroneal DML <6.0 ms; tibial DML <6.0 ms; Upper limb motor nerve conduction velocity (MNCV) >50 m/s; lower limb MNCV >40 m/s; Upper limb F-wave<30 ms; lower limb F-wave<55 ms; CMAPs: median>5 mV; ulnar>5 mV; peroneal>2 mV; tibial>4 mV	

CMAP: compound muscle action potential

LP was performed in all of the patients, only four patients had albuminocytologic dissociation. IVIG treatment was administered in all of the patients and three patients were treated with plasma exchange additionally. One patient developed hypertension and was considered as autonomic involvement.

Clinical characteristics in pediatric GBS differed from that in adult ones. The incidence of facial nervedeficit in children was lower and the incidence of bulbar dysfunction in children was hig-her than that in adults [4]. The cranial nerve involvement more frequently in pediatric GBS [12,13]. In our case series, four out of seven patient had cranial nerve involvement (57.1%). In children, pain and walking difficulty are the most common symptom [14]. In our series two patient had foot pain and 6 patients had difficulty in walking. AIDP is the most common form of GBS in child-hood [15,16]. AIDP does not differ in electrophysiological findings and prognosis in children and adults. Response to IVIG treatment in AMAN is not different from adults. The response to IVIG treatment in AIDP is not as effective compared to adults [15]. In a study fromTurkey, a girl/boy ratio is 1/4 in AIDP while 1/1 in axonal form [17].

In the management of the situation, the period of the disease is very important. Since it is the first four weeks of onset and progression, it should always be kept in mind that the symptoms may progress during this period and lead to respiratory insufficiency. Therefore, the child should definitely be hospitalized and closely monitored. Weakness and hypotonia are initially mild, and sometimes it is necessary to predict and treat fatal autonomic or respiratory complications [18]. In addition to close monitoring of respiratory and autonomic complications, it is important to avoid complications such as constipation, pressure injuries, contractures and renal stones due to pain management and immobility. Vital signs and respiratory capacity should be closely monitored [19]. The treatment of GBS is directed at two main reasons. On the one hand, there is life support with intensive care treatment in the presence of respiratory insufficiency, prevention of secondary complications and intensive physical therapy, and on the other hand, the autoimmune process that causes the demyelination of peripheral myelins heaths is to stop with early immunomodulatory therapy. Aggressive supportive therapy alone provides a dramatic reduction in mortality. With intensive care treatment mortality decreased from 50% to 10%. Immunomodulator treatment shortens the duration of treatment but does not significantly affect mortality [20].

This is a case series. Ethics committee approval is not required. Written informed consent was obtained from all cases.

REFERENCES

1.Sejvar JJ, Baughman AL, Wise M and Morgan OW. Population incidence of Guillain-Barre syndrome: a systematic review and meta-analysis. Neuroepidemiology. 2011; 36: 123-33.

2.McGrogan A, Madle GC, Seaman HE and de Vries CS. The epidemiology of Guillain-Barre syndrome worldwide. A systematic literature review. Neuroepidemiology. 2009; 32: 150-63.

3.Jacobs BC, Rothbarth PH, van der Meche FG, et al. The spectrum of antecedent infections in Guillain-Barre syndrome: a case-control study. Neurology. 1998; 51: 1110-5.

4.Wu X, Shen D, Li T, et al. Distinct clinical characteristics of pediatric Guillain-Barre Syndrome: A comparative study between children and adults in Northeast China. PLoS One. 2016; 11: e0151611.

5.Wijdicks EF and Klein CJ. Guillain-Barre Syndrome. Mayo Clin Proc. 2017; 92: 467-79.

6.Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet. 2016;388(10045):717-27. doi: 10.1016/S0140-6736(16)00339-1.

7.Varkal MA, Yıldız E, Yıldızİ, Aydınlı N, Ünüvar E. Çocukluk çağında Guillain-Barré Sendromu. Çocuk Dergisi. 2015; 15: 89-98. doi: 10.5222/j.child.2015.089).

8.Hughes RA, Swan AV and van Doorn PA. Intravenous immunoglobulin for Guillain-Barre syndrome. Cochrane Database Syst Rev. 2014: CD002063.

9.Raphael JC, Chevret S, Hughes RA and Annane D. Plasma exchange for Guillain-Barre syndrome. Cochrane Database Syst Rev. 2012: CD001798.

10.Rajabally YA, Durand MC, Mitchell J, Orlikowski D and Nicolas G. Electrophysiological diagnosis of Guillain-Barre syndrome subtype: could a single study suffice? Journal of neurology, neurosurgery, and psychiatry. 2015; 86: 115-9.

11.Kocaman C, Yıldırım Yesinel S, Yesinel S, Aksüyek E, Arslan G. Guillain Barre Sendromlu çocukların klinik ve elektrofizyolojik özellikleri. İzmir Tepecik Hast Derg 2005;1.5:911.-97.

12.Lee JH, Sung IY and Rew IS. Clinical presentation and prognosis of childhood Guillain-Barre syndrome. J Paediatr Child Health. 2008; 44: 449-54.

13.Linden V, da Paz JA, Casella EB and Marques-Dias MJ. Guillain-Barre syndrome in children: clinic, laboratorial and epidemiologic study of 61 patients. Arquivos de Neuro-psiquiatria. 2010; 68: 12-7.

14.Devos D, Magot A, Perrier-Boeswillwald J, et al. Guillain-Barre syndrome during childhood: particular clinical and electrophysiological features. Muscle Nerve. 2013; 48: 247-51.

15.Ye YQ, Wang KR, Sun L and Wang Z. Clinical and electrophysiologic features of childhood Guillain-Barre syndrome in Northeast China. JFormosMedAssoc.2014; 113: 634-9.

16.Gupta PK, Singhi P, Singhi S, Kasinathan A and Sankhyan N. How Different is AMAN from AIDP in Childhood GBS? A Prospective Study from North India. Indian J Pediatr. 2019; 86: 329-34.

17.Tekgul H, Serdaroglu G and Tutuncuoglu S. Outcome of axonal and demyelinating forms of Guillain-Barre syndrome in children. Pediatric Neurol. 2003; 28: 295-9.

18.Bradshaw DY, Jones HR. Guillain-Barre syndrome in children: clinical course, electrodiagnosis, and prognosis. Muscle Nerve. 1992; 15: 500-6.

19.Ropper AH and Kehne SM. Guillain-Barre syndrome: management of respiratory failure. Neurology. 1985; 35: 1662-5.

20.Haupt WF. Recent advances of therapeutic apheresis in Guillain-Barre syndrome. Ther Apher. 2000; 4: 271-4.