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Guillain-Barré Syndrome (GBS) in Children, the Experience of Senegal

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Abstract

Introduction: GBS is a rare condition in children. The risk factors for GBS are present in Africa; however, the majority of studies reported are case series. The objective of our work was to evaluate the epidemiological, clinical, paraclinical, therapeutic and evolutionary profile of GBS cases. **Material and Method:** This was a prospective longitudinal study, lasting 24 months between November 2019 and November 2021. All patients aged 2 - 18 years diagnosed with GBS according to the Brighton criteria (level 2) were included in our study. Patients with incomplete or unexploitable records were excluded. **Results:** Over a 24-month period, 16 cases of GBS were collected. These included 12 boys (75%) and 4 girls (25%). The demyelinating form (ADIP) was found in 9 children (43.75%) and the axonal form in 7 patients (37.5%), 5 of whom had pure motor involvement (AMAN) and 2 with sensory-motor involvement (AMSAN). Corticosteroid therapy was more effective in treating pain and in demyelinating forms. Three deaths were noted and all had AMAN. **Conclusion:** GBS poses a management problem in our context of a country with limited resources. Corticosteroid therapy has been shown to be effective in the treatment of pain and demyelinating forms of the disease and should be considered for use in GBS.

Keywords

GBS, Child, Senegal, Corticotherapy

1. Introduction

Acute polyradiculoneuritis is a heterogeneous group of peripheral nervous system disorders of which Guillain-Barré syndrome (GBS) represents the typical, most

common and best-characterized form. GBS is currently considered the most frequent cause of acute flaccid paralysis in children worldwide [1] [2]. It is a rare condition in children, with an annual incidence ranging from 0.34 to 1.34 per 100,000 person-years [3] [4] [5]. It is a serious pathology that can rapidly engage the vital prognosis of the child and requires consequent therapeutic means (polyvalent immunoglobulins, plasma exchange, pediatric neuro-resuscitation…). Most studies are conducted in Europe, Asia or North America [6]. The risk factors for GBS are present in Africa, but most of the studies reported are case series [7]. The current treatment of GBS is based on plasma exchange and high-dose polyvalent immunoglobulin. However, the use of glucocorticoids, which is very accessible, remains controversial [8] [9]. The difficulty in our context is the lack of an adequate therapeutic arsenal for better management. The objective of our work was to evaluate the epidemiological, clinical, paraclinical, therapeutic and evolutionary profile of GBS cases in the Albert Royer and Diamniadio University Children's Hospitals, Dakar/Senegal.

2. Patients and Methods

This was a prospective longitudinal study, lasting 24 months between November 2019 and November 2021. All patients aged 2 - 18 years diagnosed with GBS according to the Brighton criteria (level 2) **at the Albert Royer and Diamniadio University Children's Hospitals**, Dakar/Senegal were included in our study. Patients with incomplete or unusable records were excluded. The study of the records of the patients followed allowed us to obtain information on the following characteristics:

- Socio-demographics (age, gender).
- Clinical aspects (mode of onset, circumstances of onset, neurological and extra-neurological signs and clinical course).
- Paraclinical aspects: ENMG, cytological, bacteriological, viral and chemical analyses of the cerebrospinal fluid, blood count, CRP (C-reactive protein), retroviral serology, HBV, HCV and sedimentation rate (VS) were systematically requested. Other biological analyses such as the COVID-19 test were requested depending on the clinical context.

We used SPHINX DEMO 5 software for data entry and statistical analysis (calculation of means and frequencies) was performed with SPSS version 20.0 statistical software.

3. Results

Over a 24-month period, 16 cases of GBS meeting Brighton level 2 criteria were collected. The cases included 12 boys (75%) and 4 girls (25%). The mean age of the children was 7.56 years (range 2 and 16 years). The most representative age group was between 2 and 6 years (see **Table 1**). No specific background was found in our patients and 87.5% (14/16) of the patients had a history of an infectious episode before the occurrence of GBS. A history of influenza-like illness

Table 1. Distribution of patients by age group.

Age range	Numbers	Percentage (%)
≤2 ans	2	12.5
]2; 6 ans]	6	37.5
]6; 9 ans]	3	18.75
]9; 12 ans]	3	18.75
]12; 17 ans]	2	12.5

was found in 10 patients (62.5%), on average 9 days before the onset of neurological signs with extremes of 7 and 21 days. Four patients (25%) had presented an episode of acute gastroenteritis on average 12 days before the onset of neurological signs, with extremes of 8 and 16 days. The clinical symptoms were paresthesias of the four limbs in 9 children (56.25%) and in 3 children (18.75%) these paresthesias were associated with diffuse myalgias. These symptoms preceded the onset of motor deficits by an average of 3.5 days, with extremes of one to five days. The clinical manifestations were dominated by a predominantly proximal motor deficit, observed in all the children and which had settled in an ascending manner, on average 12.18 days with extremes of 5 and 25 days. Fourteen patients (87.5%) had tetraparesis and two patients (12.5%) had tetraplegia. Osteotendinous reflexes were abolished in 14 patients and decreased in 2 patients. Hypotonia was observed in all children; it involved all four limbs in 11 children and was generalized in 5 patients. Six patients had cranial nerve damage, including 2 with bulbar nerve damage (X and XI), one patient had oculomotor nerve damage (IV and VI), 2 patients had facial diplegia and one patient had peripheral facial palsy. The neurovegetative manifestations were tachycardia in 2 patients and cardiac arrhythmia in one patient. A biological inflammatory syndrome associating C-reactive protein positivity and hyperleukocytosis with a predominance of neutrophils was detected in 2 patients. COVID-19 serology was positive in 2 patients (IgG), Campylobacter Jejuni serology in 1 patient, and hepatitis B antigen in 1 patient without signs of active hepatitis. Retroviral serology (HIV) was negative in all children. Lumbar puncture revealed albuminous-cytological dissociation in 12 patients (75%) and viral, bacteriological and parasitic analysis of CSF was unremarkable. In these 12 patients, the lumbar puncture was performed on average 7.5 days after the onset of motor deficits with extremes of 2 and 16 days. In the 4 patients with normal lumbar puncture, it was performed on average 12.5 days after the motor deficit.

The electroneuromyogram (ENMG) showed a demyelinating form (ADIP) in 9 patients (43.75%). An axonal form in 7 patients (37.5%) of which 5 with pure motor involvement (AMAN) and 2 with sensory-motor involvement (AMSAN). The axonal form affected more infants, early and middle childhood with extremes of 2 and 6 years, for an average age of 4.16 years. The demyelinating form was

more frequent in adolescents and older children with a mean age of 10.22 years (extremes of 4 and 16 years).

All patients received corticosteroid therapy, prednisone 1 mg/kg/day for 4 weeks with a gradual stop over 4 to 6 weeks. Only two patients with bulbar signs had received an initial five-day bolus of methylprednisolone 1 g/1.73m^2. For the treatment of myalgia, in addition to corticosteroid therapy, analgesic treatment based on Paracetamol (15 mg/kg/6h) was used and the evolution was marked by the improvement of the pain in an average of 5.66 days. In addition to corticosteroid therapy, carbamazepine (10 mg/kg/D) was used in those with paresthesias and the pain improved on average 5.44 days. Two patients had received antibiotic therapy with ceftriaxone 100 mg/kg/D and two patients were intubated after respiratory distress. The duration of hospitalization varied between 3 and 102 days with an average of 25 days. It was much shorter in children with demyelinating GBS with a mean of 17.55 days and extremes of 3 and 42 days.

Children with axonal GBS had a mean hospital stay of 34.57 days with extremes of 8 and 102 days. After two years of follow-up, a complete recovery was noted in the 9 patients (64.28%) who had demyelinating GBS and the average recovery time was 22.7 days with extremes of 15 and 45 days. Motor sequelae were noted in 4 patients (AMAN (2 children) and AMSAN (2 children)). The 3 patients who died (18.75%) had presented an AMAN form. The first death occurred after 4 months of evolution following a cardiac arrest and the other two patients after 18 and 22 days of hospitalization in a respiratory distress of unknown cause.

The clinical, electrophysiological, therapeutic and evolutionary aspects of each patient are summarised in (**Table 2**).

4. Discussion

The incidence of GBS remains unknown in our context, in Africa the majority of studies are on case series. In Denmark the incidence of GBS was 0.69/100,000 children, with an average age of 8 years [4]. A study conducted in the United States in children under 9 years of age showed an incidence of GBS of 0.62/100,000. In 24 months, we collected 16 cases of GBS and the mean age was 7.56 years. Several studies have shown a male predominance of GBS in children [4] [8] [10], which is similar to our study where the male predominance was estimated at 75%.

In our study, influenza-like illness preceded the clinical picture in 62.5% of cases and 25% of children presented with an episode of acute gastroenteritis. According to the literature, GBS is preceded by an infectious, respiratory or digestive episode in 80% of cases [11] [12].

The pathogens frequently incriminated are Campylobacter Jejuni, Cytomegalovirus, Epstein-Barr virus and Mycoplasma [13] [14]. Other factors such as vaccination and surgery have also been implicated [15]. Two children had a positive COVID-19 serology, this result is justified by the COVID-19 pandemic context during our study.

Table 2. Summary table of clinical, electrophysiological, therapeutic and evolutionary data of patients.

Patients	Age	Sex	Symptoms	ENMG	Treatments	Evolution	
						Short and medium term	Long term (2 ans)
1	2	M	TPG (HS: 5) + P + M + BNI	AMAN	C + CBZ + PARA	RP (7j); DH (102j)	D
2	2	F	TPG (HS: 4)	AMAN	C	DH (18j)	D
3	5	M	TPS (HS: 2) + P	AMAN	C + CBZ	RP (5j); DH (32j)	HS: 2
4	4	F	TPG (HS: 4) + P + PFP	AMAN	C + CBZ	RP (8j); DH (18j)	HS: 3
5	6	M	TPG (HS: 4) + P + ANO	AMAN	C + CBZ	RP (8j); DH (22j)	D
6	5	M	TPG (HS: 4) + BPFP	AMSAN	C+	DH (18j)	HS: 4
7	5	M	TPG (HS: 4) + P	AMSAN	C + CBZ	RP (3j); DH (32j)	HS: 3
8	16	M	TPG (HS: 4) + P + M	ADIP	C + CBZ + PARA	RP (5j); DH (15j)	HS: 0
9	10	M	TPG (HS: 4) + BPFP	ADIP	C+	DH (33j)	HS: 0
10	14	F	TPG (HS: 4) + P	ADIP	C + CBZ	RP (5j); DH (16j)	HS: 0
11	4	M	TPG (HS: 5) + BNI	ADIP	C	DH (42j)	HS: 0
12	8	M	TPS (HS: 3)	ADIP	C+	DH (10j)	HS: 1
13	12	M	TPG (HS: 4) + P + M	ADIP	C + CBZ + PARA	RP (5j); DH (16j)	HS: 0
14	7	F	TPG (HS: 4) + PFP	ADIP	C+	DH (8j)	HS: 0
15	12	M	TPG (HS: 4) + P	ADIP	C + CBZ	RP (3j); DH (15j)	HS: 0
16	9	M	TPG (HS: 4)	ADIP	C+	DH (3j)	HS: 1

TPG: tetraplegia; HS: Hughes score; P: paresthesias; BNI: bulbar nerve impairment; M: myalgias; PF: peripheral facial palsy; BPFP: bilateral peripheral facial palsy; AMAN: acute axonal motor neuropathy; AMSAN: acute axonal motor and sensory neuropathy; ADIP: acute inflammatory demyelinating polyradiculoneuropathy; C: corticosteroid therapy; CBZ: carbamazepine; PARA: paracetamol; PR: pain relief; DH: duration of hospitalization; D: death.

In our study, 56.25% of the children reported prodromes (paresthesias and myalgias) before the onset of the motor deficit, which was ascending and symmetrical with different levels of severity (tetraparesis and tetraplegia). Our results are in agreement with the data in the literature [16] [17].

Autonomic dysfunction is frequent but often unrecognized in pediatric GBS [4]. In our study, we observed autonomic nervous system involvement in 3 children (18.75%).

The frequency of GBS subtypes varies considerably between regions. It should be noted that AIDP, which corresponds to the classic demyelinating form, represents the vast majority (70% - 90%) of GBS cases in Western Europe and the United States, whereas the AMAN form represents the dominant form in Asia, accounting for 65% of cases in China [1] [18]. In our study, the demyelinating form was the most frequent (56.25% of patients).

The use of glucocorticoid (GC) in GBS patients is controversial [8] [9]. Clinical trials in Europe and North America have not found significant efficacy of

GCs alone. Investigators such as Hughes have suggested that GBS patients with conduction block respond well to GCs, whereas the use of GCs in patients with denervation delays GBS recovery [19]. Linzhuo Ma *et al.* [20] had shown in a multicenter study that the Hughes score at discharge and after 3 months was significantly lower in patients with the ADIP form treated with GCs than in patients who had the AMAN form. Further analysis of this study revealed that in patients with ADIP, the high-dose group (250 to 1000 mg/d of methylprednisolone for 3 to 5 days) had shorter hospital days and a nadir Hughes score, at hospital discharge and 3 months after disease onset, significantly lower than in the low-dose group (40 - 120 mg/d methylprednisolone for 3 - 5 days or prednisolone 1 mg/kg/d for 1 week, tapering to 2 months). However, among AMAN patients, the short-term outcome in the high-dose group was not significantly different from that in the low-dose group [20]. The effect of corticosteroid therapy in GBS lies more in its effectiveness in treating pain [21]. In our study, its combination with CARBAMAZEPINE and PARACETAMOL allowed us to have an amendment of the pain in an average of 5.44 days for the patients who had paresthesias alone and 5.66 days for the patients who had paresthesias associated with myalgias.

The limitations of our work are: the size of our sample and the lack of comparison between patients on corticosteroids and those who are not. The unavailability of some treatments, such as plasma exchange in order to make the comparison with children on corticosteroid alone or corticosteroid plus plasma exchange.

5. Conclusion

In our study, the axonal form affected more infants, early and middle childhood with extremes and the demyelinating form was more encountered in adolescents and older children. The frequent recourse to corticotherapy was related to the inaccessibility of intravenous immunoglobulin and plasma exchange. However, the favorable evolution of the children, the improvement of pain (paresthesias and myalgias) and the inaccessibility of certain drugs in our context should push us to be more interested in the effectiveness of corticotherapy in GBS.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Asbury, A.K. (2000) New Concepts of Guillain-Barré Syndrome. *Journal of Child Neurology*, **15**, 183-191. https://doi.org/10.1177/088307380001500308

[2] Asbury, A.K. and Cornblath, D.R. (1990) Assessment of Current Diagnostic Criteria for Guillain-Barré Syndrome. *Annals of Neurology*, **27**, S21-S24. https://doi.org/10.1002/ana.410270707

[3] Hafsteinsdóttir, B., Ólafsson, E. and Jakobsson, F. (2018) Incidence and Outcome of

Guillain-Barré Syndrome in Iceland: A Population-Based Study. *Acta Neurologica Scandinavica*, **138**, 454-458. https://doi.org/10.1111/ane.13000

[4] Levison, L.S., Thomsen, R.W., Markvardsen, L.K., *et al.* (2009) Pediatric Guillain-Barré Syndrome in a 30-Year Nationwide Cohort. *Pediatric Neurology*, **107**, 57-63. https://doi.org/10.1016/j.pediatrneurol.2020.01.017

[5] McGrogan, A., Madle, G.C., Seaman, H.E., *et al.* (2009) The Epidemiology of Guillain-Barré Syndrome Worldwide. *Neuroepidemiology*, **32**, 150-163. https://doi.org/10.1159/000184748

[6] Willison, J., Van Doorn, H.J. and Bart, P. (2016) Guillain-Barré Syndrome. *The Lancet*, **388**, 717-727. https://doi.org/10.1016/S0140-6736(16)00339-1

[7] Akolly, D., *et al.* (2016) Syndrome De Guillain-Barré Chez L'enfant Dans Un Contexte A Ressources Limitées: A Propos De Deux Observations Au CHU Sylvanus Olympio De Lomé—TOGO. *European Scientific Journal*, **12**, 182-188. https://doi.org/10.19044/esj.2016.v12n36p182

[8] Hughes, R.A. (2004) Treatment of Guillain-Barré Syndrome with Corticosteroids: Lack of Benefit? *The Lancet*, **363**, 181-182. https://doi.org/10.1016/S0140-6736(03)15367-6

[9] Mastaglia, F.L. (2005) Neuromuscular Disorders: Molecular and Therapeutic Insights. *The Lancet Neurology*, **4**, 6-7. https://doi.org/10.1016/S1474-4422(04)00946-9

[10] Nagasawa, K., Kuwabara, S., Misawa, S., *et al.* (2006) Electrophysiological Subtypes and Prognosis of Childhood Guillain-Barré Syndrome in Japan. *Muscle & Nerve*, **33**, 766-770. https://doi.org/10.1002/mus.20520

[11] Jacobs, B.C., van Doorn, P.A., Schmitz, P.I., Tio-Gillen, A.P., Herbrink, P., Visser, L.H., *et al.* (1996) *Campylobacter jejuni* et anticorps anti-GM1 dans le syndrome de Guillain-Barré. *Annals of Neurology*, **40**, 181-187. https://doi.org/10.1002/ana.410400209

[12] Sharshar, T., Siami, S. and Orlikowski, D. (2007) Does This Patient Have Guillain-Barré Syndrome? *Resuscitation*, **16**, 504-510. https://doi.org/10.1016/j.reaurg.2007.09.006

[13] Bradshaw, D.Y. and Jones, H.R. (1992) Guillain-Barré Syndrome in Children: Clinical Course, Electrodiagnosis, and Prognosis. *Muscle & Nerve*, **15**, 500-506. https://doi.org/10.1002/mus.880150415

[14] Goodfellow, J.A. and Willison, H.J. (2016) Guillain-Barré Syndrome: A Century of Progress. *Nature Reviews Neurology*, **12**, 723-731. https://doi.org/10.1038/nrneurol.2016.172

[15] Webb, A.J.S., Brain, S.A.E., Wood, R., *et al.* (2015) Seasonal Variation in Guillain-Barré Syndrome: A Systematic Review, Meta-Analysis and Oxfordshire Cohort Study. *Journal of Neurology, Neurosurgery and Psychiatry*, **86**, 1196-1201. https://doi.org/10.1136/jnnp-2014-309056

[16] Kuwabara, S. and Yuki, N. (2013) Axonal Guillain-Barré Syndrome: Concepts and Controversies. *The Lancet Neurology*, **12**, 1180-1188. https://doi.org/10.1016/S1474-4422(13)70215-1

[17] Wu, X., Li, C., Zhang, B., *et al.* (2015) Predictors for Mechanical Ventilation and Short-Term Prognosis in Patients with Guillain-Barré Syndrome. *Critical Care*, **19**, Article No. 310. https://doi.org/10.1186/s13054-015-1037-z

[18] Devos, D., Magot, A., Perrier-Boeswillwald, J., *et al.* (2013) Guillain-Barré Syndrome during Childhood: Particular Clinical and Electrophysiological Features: GBS in Childhood. *Muscle & Nerve*, **48**, 247-251. https://doi.org/10.1002/mus.23749

[19] Hughes, R.A., Brassington, R., Gunn, A.A. and van Doorn, P.A. (2016) Corticoste-

roids for Guillain-Barré Syndrome. *Cochrane Database of Systematic Reviews*, **10**, CD001446. https://doi.org/10.1002/14651858.CD001446.pub5

[20] Ma, L.Z., Liu, S.P., Xiao, Z.M., *et al.* (2022) Comparison of the Effects of Different Doses of Glucocorticoids on Distinct Subtypes of Guillain-Barré Syndrome in South China. *BMC Neurology*, **22**, Article No. 46. https://doi.org/10.1186/s12883-022-02567-8

[21] Kabore, R., Magy, L., Boukhris, S., *et al.* (2004) The Value of Corticosteroid Therapy in the Treatment of Pain in the Acute Phase of Guillain-Barré Syndrome. *Revue Neurologique* (*Paris*), **160**, 821-823. https://doi.org/10.1016/S0035-3787(04)71037-1