



HAL
open science

Prognostic value of contrast-enhanced MRI in Guillain–Barré syndrome in children

F. Althubaiti, C. Guiomard, Francois Rivier, Pierre Meyer, N. Leboucq

► **To cite this version:**

F. Althubaiti, C. Guiomard, Francois Rivier, Pierre Meyer, N. Leboucq. Prognostic value of contrast-enhanced MRI in Guillain–Barré syndrome in children. Archives de Pédiatrie, In press, 10.1016/j.arcped.2022.01.004 . hal-03552647

HAL Id: hal-03552647

<https://hal.science/hal-03552647>

Submitted on 2 Feb 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Prognostic value of contrast-enhanced MRI in Guillain–Barré syndrome in children

F. Althubaiti^{a,b,1,*}, C. Guiomard^a, F. Rivier^{a,c}, P. Meyer^{a,c}, N. Leboucq^d

^a Département de Neuropédiatrie, CHU de Montpellier, Hôpital Gui de Chauliac, 34295, Montpellier, France

^b King Abdulaziz University, Department of Pediatrics, 21589, Jeddah, Saudi Arabia

^c PhyMedExp, Université de Montpellier, INSERM, CNRS, Montpellier, France

^d Département de Neuroradiologie, CHU Gui de Chauliac, 34 295, Montpellier, France

A B S T R A C T

Background: : The aim of this retrospective study is to explore the prognostic value of different contrast enhancement imaging patterns in childhood Guillain–Barré syndrome by comparing the clinical, laboratory, and therapeutic outcomes.

Methods: : We included a total of 37 patients who were diagnosed and followed up by a pediatric neurology team at Montpellier University Hospital between 2000 and 2016. All images were reinterpreted by the first author and a senior pediatric neuroradiology staff member in two different sessions; in the case of disagreement, the expert's reading was considered.

Results: : The study group comprised 22 (59.5%) boys and 15 (40.5%) girls. The age ranged from 1.5 year to 14.8 years. Muscle weakness was present in 33 (89.2%) patients. Cranial nerves involvement was observed in 22 (59.5%) patients, while 29 (78.4%) patients had albuminocytological dissociation. In 27 (73%) patients, contrast enhancement or thickening of the lumbosacral nerve roots was found. Simultaneous spinal nerve root and cranial nerve enhancement was noted in five (17.2%) patients, while isolated cranial nerve enhancement was identified in three (10.3%) patients. Clinical and radiological cranial nerve involvement was found in seven (18.9%) patients, while isolated clinical cranial nerves involvement occurred in 13 (35.1%) patients. No significant correlation between different magnetic resonance imaging (MRI) enhancement patterns and short-term or long-term outcomes was found in our cohort.

Conclusion: : Contrast-enhanced brain and spinal MRI is a sensitive and recommended supportive test for diagnosing acute inflammatory polyradiculopathy in children. Its predictive value for clinical, and therapeutic outcomes in the short or long term has not yet been proved.

Keywords:

Guillain–Barré syndrome
Acute inflammatory polyradiculopathy
Miller–Fisher syndrome
Contrast-enhanced magnetic resonance imaging
Spinal nerve root enhancement

1. Introduction

Guillain–Barré syndrome (GBS) is an acute inflammatory polyradiculopathy that in its most typical form is characterized by acute-onset, progressive ascending and symmetrical weakness [1, 2], associated with areflexia and albuminocytological dissociation of the cerebrospinal fluid (CSF) [3]. The incidence rate is estimated at 0.8–1.9 per 100,000 people per year in the United States and Europe

with a male predominance [4]. A French nationwide epidemiological study based on hospital discharge data reported a crude incidence rate in children younger than 10 years of 0.9 per 100,000 per year with a peak of incidence at 2 years of age [5]. Nerve conduction studies (NCSs) have made it possible to classify acute polyradiculopathy into: acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN) [6, 7]. Antineuronal antibodies might be positive in certain cases [8]. Intravenous immunoglobulin and plasmapheresis are the main treatment modalities [9].

Contrast-enhanced magnetic resonance imaging (CE-MRI) is a valuable and validated supportive diagnostic tool for GBS in particular at an early presentation when electromyoneurography, blood and CSF analyses are not conclusive [10–12] (Figs. 1 and 2). In adult series of 474 patients [13], the concentration of CSF protein was strongly dependent on the timing of lumbar puncture. It was elevated in 48% of patients on day 1 after lumbar puncture, in 53% of patients on

Abbreviations: GBS, Guillain–Barré syndrome; NCSs, Nerve conduction studies; AIDP, Acute inflammatory demyelinating polyneuropathy; AMAN, Acute motor axonal neuropathy; AMSAN, Acute motor sensory axonal neuropathy; CE-MRI, Contrast-enhanced magnetic resonance imaging; CSF, Cerebrospinal fluid; CNs, Cranial nerves; ENMG, Electroneuromyography; PICU, Pediatric intensive care unit

* Corresponding author at: Centre Hospitalier Universitaire Montpellier, Hôpital Gui de Chauliac, 371, Avenue du Doyen Gaston Giraud, 34295 Montpellier Cedex 5, France.

1 Pediatric teaching assistant, Faculty of Medicine, King Abdulaziz University, Al Ehtifalat St, Jeddah, 21589, Saudi Arabia.

E-mail address: drfamt@gmail.com (F. Althubaiti).



Fig. 1. Post-contrast T1-weighted images. (A) Sagittal view with thickening and diffuse enhancement of anterior and posterior spinal nerve roots. (B) Axial view at cauda equina level with anterior and posterior intra-thecal thickening and enhancement of spinal nerve roots. Taken from patient no 8 in our series.

day 2, and in 88% of patients at 3 weeks. The results of nerve conduction studies in a subgroup of 180 (41%) patients of the same group did not meet the diagnostic criteria of demyelinating axonal or unexcitable neuropathy. Comparable results have been reported in a pediatric population [14–16]. The enhancement of nerve roots is explained by the rupture of the blood–nerve barrier as a result of the inflammatory process occurring [17,18]. An absence of this radiological phenomenon does not exclude its presence at the microscopic level. Although CE-MRI is sensitive for acute inflammatory polyradiculopathy, it is not specific since many other conditions might also have cranial nerve and/or spinal nerve root enhancement. Lyme disease is a frequently seen infectious meningo-radiculopathy that resembles GBS radiologically. Neuro-metabolic and neuro-neoplastic

diseases such as Krabbe disease, metachromatic leukodystrophy, and leptomeningeal carcinomatosis can also present with spinal root enhancement [19].

1.1. Study objectives

In this study we explored the prognostic value of CE-MRI in GBS by comparing the different radiological patterns with the clinical, laboratory, and therapeutic outcomes of this syndrome.

2. Study design, patients, and methods

2.1. Study design and patients

A retrospective multicenter cohort study was recently conducted by a group of colleagues, which included 110 patients from Montpellier and Toulouse neuropediatric services between 2000 and 2016. We exported the data of patients seen and followed up in the Montpellier neuropediatric service. A total of 42 GBS patients were diagnosed, 37 of whom were included in our study. The other four patients were excluded because two of them did not undergo MRI and the electronic files for the other two were not accessible. We included in our clinical analysis the GBS disability score for a more precise picture of the disease sequelae in the different groups. This score is mainly based on muscular deficits and graded from 0 to 6, as shown in Table 1 [20, 21].

2.2. Methods

Our CE-MRI protocol for GBS was as follows:

Spinal: Sagittal T2-weighted, T1-weighted with contrast, axial lumbosacral T1-weighted fat-saturation with contrast.
Brain: Axial T2-weighted, fluid-attenuated inversion recovery (FLAIR), diffusion, T2 *, three-dimensional T1-weighted with contrast.

The MRIs were re-interpreted by the first author. A third reading by a senior pediatric neuroradiologist was performed for all the series. In cases of incompatibility, the expert's reading was considered after discussion. All readings were conducted over a PACS high-resolution screen system. We divided patients into multiple groups based on the presence or absence of spinal nerve root and cranial nerve enhancement, as well as on clinical and radiological incidental cranial nerve involvement. We conducted between-group comparisons using a chi-square test for categorical variables, a Mann–Whitney *U* test for two groups, and the Kruskal–Wallis test for multiple quantitative variables.

3. Results

3.1. Patient characteristics

A total of 37 patients were included: 22 (59.5%) males and 15 (40.5%) females. Their age ranged from 1.5 year to 14.8 years with a median of 5.3 years. Overall, 29 (78.4%) cases were preceded by infections, mainly oronasopharyngeal infections in 12 (32.4%) patients, followed by gastrointestinal infections in nine (24.3%) patients, isolated fever in five (13.5%) patients, and varicella in two patients (5.4%). Muscle weakness was present in 33 (89.2%) patients. Tendon reflexes were absent in 27 (73%) patients. Ataxia was present in 20 (54.1%) patients, paresthesia in 10 (27%) patients, and pain in 28 (75.7%) patients. The minimum pain duration was 2 days and the maximum was 90 days with a median of 39 days for 16 out of 28 patients. Cranial nerves (CNs) were involved in 22 (59.5%) patients. A GBS disability score of grade 1 (GBS1) was noted in three (8.1%)

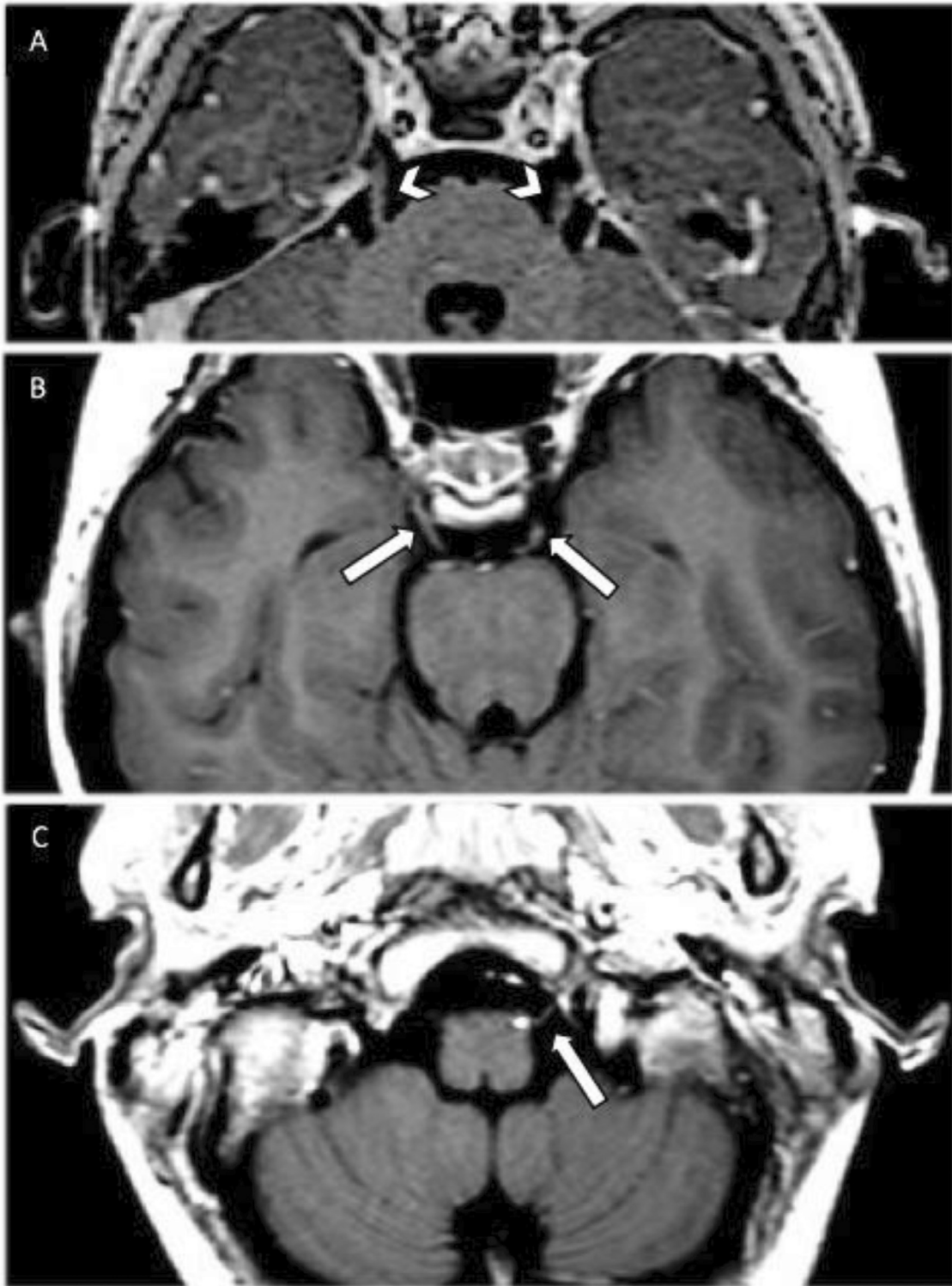


Fig. 2. Post-contrast axial T1-weighted images. (A) Bilateral fifth nerve enhancement (arrowheads). (B) Bilateral third nerve enhancement with right side predominance (solid arrows). (C) Left 12th nerve enhancement. Taken from patients no. 10, 35, and 37 in our series.

patients, GBS2 in five (13.5%) patients, GBS3 in 10 (27%) patients, GBS4 in 14 (37.8%) patients, and GBS5 in four (10.8%) patients. Six (16.2%) patients had respiratory difficulties, three (8.1%) of them were intubated. Five (13.5%) patients were admitted to the pediatric intensive care unit. Dysautonomia occurred in 15 (40.5%) patients.

3.2. Laboratory work-up

In total, 29 (78.4%) patients were found to have albuminocytological dissociation. Antineuronal antibodies were detected in six (16.2%) cases. Nine (24.3%) patients had positive serology test results. The

Table 1
Guillain–Barré syndrome disability scale [24].

Score	Description
0	Healthy state
1	Minor symptoms and capable of running
2	Able to walk 10 m or more without assistance but unable to run
3	Able to walk 10 m across an open space with help
4	Bedridden or chairbound
5	Requiring assisted ventilation for at least part of the day
6	Dead

electroneuromyography (ENMG) was normal in six (16.2%) cases. AIDP was the predominant pattern in our series, diagnosed in 27 (73%) patients; AMAN was diagnosed in two (5.4%) patients, ASMAN in one (2.7%) patient, and one case was not classifiable.

3.3. Treatment analysis

Seven (18.9%) patients received one course of intravenous immunoglobulins (T1), while 10 (27%) patients were treated with two courses of intravenous immunoglobulins (T2). Seven (18.9%) patients received more than two courses of intravenous IgG (T3). Eight (21.6%) patients were treated with more than two courses of intravenous IgG and plasmapheresis (T4). Four (10.8%) patients were treated with more than two courses of intravenous IgG and corticoids (T5). The recovery period ranged from 35 to 3897 days with a median of 193 days. Four patients had GBS sequelae interfering with daily activities. Five patients were lost to follow-up, while the minimum follow-up was 35 days after diagnosis.

3.4. Magnetic resonance imaging

A total of 27 (73%) patients had contrast enhancement or thickening of the lumbosacral nerve roots compared to 10 (27%) patients who had neither contrast enhancement nor thickening (Table 2). Eight (21.6%) patients had an enhancement or thickening of the anterior lumbosacral nerve roots, seven (18.9%) patients had posterior

nerve root enhancement or thickening, and 12 (32.4%) patients had diffuse nerve root enhancement or thickening. Brain MRI was not performed for two (5.4%) patients. Simultaneous spinal nerve root and CN enhancement was noted in five (17.2%) patients, while isolated CN enhancement was identified in three (10.3%) patients. Clinical and radiological CN involvement was found in seven (18.9%) patients, while isolated clinical CN involvement occurred in 13 (35.1%) patients. One patient (2.7%) had oculomotor, trigeminal, and abducens nerve enhancement with no associated clinical manifestations. Serial MRI studies were performed for seven patients, and one of them had transverse myelitis associated with GBS.

3.5. Time course

The period between the appearance of symptoms and performing the MRI ranged from 1 to 173 days in 35 patients, while two patients were excluded because the exact dates were not available. Two MRI studies were performed with a delay of 153 and 173 days after the appearance of symptoms, for which they were excluded from the analysis. Therefore, the mean time of performing CE-MRI was 10.6 days. No significant difference was found between the group who had CE-MRI of the spinal nerve roots and those who did not (two-sided $p = 0.57$).

4. Discussion

The sensitivity of contrast-enhanced MRI to pediatric GBS in our series was 72.97% less than that observed in other studies (83–95%) [11]. The contrast enhancement group had a higher incidence of triggering factors (75.9%), pain (71.4%), muscle weakness (72.7%), ataxia (70%), gait disturbance (73.5%), loss of tendon reflexes (70.4%), albuminocytological dissociation (79.3%), and positive serological test results (88.9%). None of these factors had a significant correlation. There were more pediatric intensive care admissions in the non-enhancement group, with a significant difference of $p = 0.014$, where all patients admitted to the intensive care unit from the non-enhancement group had cranial nerve involvement and dysautonomia. When comparing cranial nerve involvement and dysautonomia

Table 2
Clinical features and statistical significance of patients with lumbosacral nerve root enhancement or thickening versus patients without lumbosacral nerve root enhancement or thickening.

Variables	Lumbosacral nerve root enhancement/thickening ($n = 27$)	Lumbosacral nerve root non-enhancement/thickening ($n = 10$)	p
Sex	16 (72.7%) Males 11 (73.3%) Females	6 (27.3%) Males 4 (26.7%) Females	1
Triggering factors	22 (75.9%)	7 (24.1%)	0.635
Paresthesia	4 (40%)	6 (60%)	0.025
Dysautonomia	9 (60%)	6 (40%)	0.260
Pain	20 (71.4%)	8 (28.6%)	1
Cranial nerve involvement	14 (63.6%)	8 (36.4%)	0.153
Ataxia	14 (70%)	6 (30%)	1
Loss of walking	13 (68.4%)	6 (31.6%)	0.717
Loss of tendon reflexes	19 (70.4%)	8 (29.6%)	1
Intubation	1 (33.3%)	2 (66.7%)	0.172
Albuminocytological dissociation	23 (79.3%)	6 (20.7%)	0.333
Antineuronal antibodies	3 (50%)	3 (50%)	0.320
Serology	8 (88.9%)	1 (11.1%)	0.386
Age (years)	(1.2–13.2) - M: 3.8	(2–14.8) - M: 8	0.55
Recovery period (days)	(35–3897) - M: 193	(117–424) - M: 196	0.911
GBS disability score	GBS1 (2, 5.6%) GBS2 (5, 13.9%) GBS3 (6, 16.7%) GBS4 (12, 33.3%) GBS5 (1, 2.7%)	GBS1 (1, 2.7%) GBS2 (0) GBS3 (4, 11.1%) GBS4 (2, 5.6%) GBS5 (3, 8.3%)	0.277
ENMG	AIDP (19, 73.1%), AMAN (1, 3.8%)	AIDP (8, 30.8%), AMAN (1, 3.8%), AMSAN (1, 3.8%)	0.792
Treatment	T1 (5, 13.5%), T2 (8, 21.6%), T3 (6, 16.2%), T4 (5, 13.5%), T5 (3, 8.1%)	T1 (2, 5.4%), T2 (2, 5.4%), T3 (1, 2.7%), T4 (4, 10.8%), T5 (1, 2.7%)	0.736

One course of intravenous IgG (T1). Two courses of intravenous IgG (T2). More than two courses of intravenous IgG (T3). More than two courses of intravenous IgG and plasmapheresis (T4). More than two courses of intravenous IgG and corticoids (T5).

GBS: Guillain–Barré syndrome; AIDP: acute inflammatory demyelinating polyneuropathy; AMAN: acute motor axonal neuropathy; AMSAN: acute motor sensory axonal neuropathy; ENMG: electroneuromyography.

in the two groups, we did not find any association. The non-enhancement group had a higher incidence of paresthesia (60%), with a significant difference of $p = 0.025$. The disability score, nerve conduction studies, and treatment modality did not show any significant correlation. The duration of hospital stay and the recovery period also did not demonstrate any correlation in this cohort.

Many studies have assessed the diagnostic value of CE-MRI in GBS but few studies have been conducted to evaluate its predictive prognostic value. Yikilmaz et al. in 2010 [22] carried out a retrospective study of a pediatric population and examined the correlation between different radiological patterns of contrast enhancement and clinical outcomes. They included 40 patients, 38 of whom demonstrated contrast enhancement; these patients were then divided into four groups (no enhancement, anterior root enhancement > posterior root enhancement, anterior root enhancement = posterior root enhancement, and only anterior root enhancement). They did not find any correlation between contrast enhancement and clinical type of GBS, and they also reported no significant correlation between the intensity of nerve root enhancement and the clinical severity. Although the study grouping is different to that in our study, their results are in concordance with what we observed, i.e., that contrast enhancement has no correlation with the type or severity of GBS. Byun et al. [23] included eight patients suspected of having GBS, five of whom were children, and classified them according to their enhancement pattern into group 1 with anterior and posterior spinal nerve root enhancement, and group 2 with only anterior spinal nerve root enhancement. All cases had muscle weakness of the extremities and hyporeflexia or areflexia. They reported sensory changes only in the first group, with no sensory findings in the second group and normal sensory nerve studies. Abnormal motor conduction study results were found in both groups. Byun concluded that isolated anterior nerve root involvement is highly suggestive of GBS. In our study we observed that the dominant type is diffused involvement of anterior and posterior nerve roots (32.4%). We did not find any sensory manifestations significantly correlated with different patterns of contrast enhancement. By contrast, paresthesia was found to be significantly correlated with GBS with no nerve root enhancement. Gorson et al. [11] in a prospective study of an adult population evaluated gadolinium enhancement of the lumbosacral nerve roots in 24 patients. Cases were categorized according to spinal nerve root enhancement into absent, mild, and prominent. They found that patients with prominent lumbosacral enhancement had more severe back or leg pain, a higher GBS disability grade, and significantly fewer patients were able to walk independently at 2 months. Pain and disability scores are sometimes hard to evaluate in children especially those younger than 3 years. These parameters were not show a significant correlation in our cohort. We found simultaneous enhancement of CNs and spinal nerves roots in five (17.2%) patients, and when comparing this group with patients who had isolated CN enhancement (three patients; 10.3%) we did not find any significant differences. Radio-clinical discordance of CN involvement has been reported in other studies. Seven (18.9%) patients in our series had clinical and radiological CN involvement compared with 13 (35.1%) patients who had only clinical CN involvement. Facial nerve palsy occurred in 13 (35.1%) patients, although only four patients had facial nerve enhancement, which is comparable to what is reported by Zuccoli et al. [24]: Trigeminal nerve enhancement occurred in five patients although clinically only one patient had trigeminal nerve involvement. Our comparison between these two groups did not reveal any significant clinical association. Four patients in our series had GBS sequelae interfering with their autonomy in daily activities, three of whom had spinal root enhancement. Myelo-polyradiculopathy was diagnosed in one patient. When comparing the sequelae in the two groups, we did not find any correlation. Repeated MRI studies were performed for seven patients, with an average of two MRI examinations per patient. One patient diagnosed with myelo-polyradiculopathy had two MRI studies after diagnosis at 7 and 15 months,

which demonstrated the disappearance of spinal nerve root enhancement and persistence of spinal cord heterogeneity on STIR sequences. Another patient diagnosed with chronic inflammatory demyelinating polyneuropathy (CIDP) who had an initial presentation of GBS had four follow-up MRI examinations at 3, 15, 21, and 25 months. Spinal nerve root enhancement disappeared at 25 months with an absence also of brachial plexus enhancement and T2-weighted high-intensity signals. In a group of 23 patients diagnosed with CIDP for whom a high resolution ultrasound (HRUS) and MRI of the brachial plexus have been done as part of the diagnostic workup, six of 23 patients (26%) had normal MRIs [25]. In these particular patients, NCSs were more contributive in the diagnosis and follow-up than CE-MRI. Our study is limited by its retrospective design and some inconsistencies in our CE-MRIs protocol. Eight patients did not have cerebral three-dimensional T1-weighted sequences, and 21 patients did not have axial spinal T1-weighted fat-saturated sequences.

5. Conclusion

Contrast-enhanced brain and spinal MRI is a sensitive and recommended supportive test for diagnosing GBS in children, especially in cases with equivocal clinical and laboratory investigations or cases with an atypical clinical presentation. It is currently part of the standard of care for patients suspected of having acute polyradiculoneuritis. The predictive value of CE-MRI for acute inflammatory polyradiculopathy outcomes has not proved. Repeated CE-MRI might be of interest in cases resistant to treatment when nerve conduction studies are not conclusive or when a GBS-associated pathology is suspected. This decision should be balanced by the risk of anesthesia when needed and the risk of contrast exposure.

Declaration of interest

This work was authorized by the research committee of Montpellier University Hospital.

Conflict of interest

None.

References

- [1] Ropper AH. The Guillain-Barré syndrome. *N Engl J Med* 1992;326:1130–6.
- [2] Yuki N, Hartung HP. Guillain-Barré Syndrome. *N Engl J Med* 2012;366:2294–304.
- [3] Hughes RAC, Cornblath DR, Willison HJ. Guillain-Barré syndrome in the 100 years since its description by Guillain, Barré and Strohl. *Brain* 2016;139:3041–7.
- [4] Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology* 2011;36:123–33.
- [5] Delannoy A, Rudant J, Chaingot C, et al. Guillain-Barré syndrome in France: a nationwide epidemiological analysis based on hospital discharge data (2008–2013). *J Peripher Nerv Syst* 2017;22:51–8.
- [6] McKhann GM, Cornblath DR, Griffin JW, et al. Acute motor axonal neuropathy: a frequent cause of acute flaccid paralysis in China. *Ann Neurol* 1993;33:333–42.
- [7] Paradiso G, Tripoli J, Galicchio S, et al. Epidemiological, clinical, and electrodiagnostic findings in childhood Guillain-Barré syndrome: a reappraisal. *Ann Neurol* 1999;46:701–7.
- [8] Schessl J, Koga M, Funakoshi K, et al. Prospective study on anti-ganglioside antibodies in childhood Guillain-Barré syndrome. *Arch Dis Child* 2007;92:48–52.
- [9] Doets AY, Jacobs BC, van Doorn PA. Advances in management of Guillain-Barré syndrome. *Curr Opin Neurol* 2018;31:541–50.
- [10] Crino PB, Zimmerman R, Laskowitz D, et al. Magnetic resonance imaging of the cauda equina in Guillain-Barré syndrome. *Neurol* 1994;44:1334–6.
- [11] Gorson KC, Ropper AH, Muriello MA, et al. Prospective evaluation of MRI lumbosacral nerve root enhancement in acute Guillain-Barré syndrome. *Neurol* 1996;47:813–7.
- [12] Coskun A, Kumandas S, Pac A, et al. Childhood Guillain-Barre syndrome. MR imaging in diagnosis and follow-up. *Acta Radiol* 2003;44:230–5.
- [13] Fokke C, van den Berg B, Drenthen J, et al. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. *Brain* 2014;137:33–43.
- [14] Korinthenberg R, Schessl J, Kirschner J. Clinical presentation and course of childhood Guillain-Barré syndrome: a prospective multicentre study. *Neuropediatrics* 2007;38:10–7.

- [15] Tang J, Dai Y, Li M, et al. Guillain-Barré syndrome in Chinese children: a retrospective analysis. *Pediatr Neurol* 2011;45:233–7.
- [16] Varkal MA, Uzunhan TA, Aydınli N, et al. Pediatric Guillain-Barré syndrome: indicators for a severe course. *Ann Indian Acad Neurol* 2015;18:24–8.
- [17] Nguyen C, Haughton VM, Ho KC, et al. Contrast enhancement in spinal nerve roots: an experimental study. *AJNR Am J Neuroradiol* 1995;16:265–8.
- [18] Nolte I, Pham M, Bendszus M. Experimental nerve imaging at 1.5-T. *Methods* 2007;43:21–8.
- [19] Kontzialis M, Poretti A, Michell H, et al. Spinal Nerve Root Enhancement on MRI Scans in Children: a Review. *J Neuroimaging* 2016;26:169–79.
- [20] Hughes RAC, Newsom-Davis JM, Perkin GD, et al. Controlled trial of prednisolone in acute polyneuropathy. *Lancet* 1978;312:750–3.
- [21] van Koningsveld R, Steyerberg EW, Hughes RA, et al. A clinical prognostic scoring system for Guillain-Barré syndrome. *Lancet Neurol* 2007;6:589–94.
- [22] Yikilmaz A, Doganay S, Gumus H, et al. Magnetic resonance imaging of childhood Guillain-Barre syndrome. *Childs Nerv Syst* 2010;26:1103–8.
- [23] Byun WM, Park WK, Park BH, et al. Guillain-Barré syndrome: MR imaging findings of the spine in eight patients. *Radiology* 1998;208:137–41.
- [24] Zuccoli G, Panigrahy A, Bailey A, et al. Redefining the Guillain-Barré spectrum in children: neuroimaging findings of cranial nerve involvement. *AJNR Am J Neuroradiol* 2011;32:639–42.
- [25] Goedee HS, Jongbloed BA, van Asseldonk JTH, et al. A comparative study of brachial plexus sonography and magnetic resonance imaging in chronic inflammatory demyelinating neuropathy and multifocal motor neuropathy. *Eur J Neurol* 2017;24:1307–13.