Journal of the Neurological Sciences 351 (2015) 187–190

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Short communication

Cauda equina conduction time in Guillain-Barré syndrome

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ARTICLE INFO

Article history:
Received 23 December 2014
Received in revised form 4 February 2015
Accepted 27 February 2015
Available online 6 March 2015

Keywords:
Acute inflammatory demyelinating neuropathy (AIDP)
Acute motor axonal neuropathy (AMAN)
Magnetic stimulation
Motor-evoked potential
Spinal nerve
Cauda equina

ABSTRACT

The proximal segment of peripheral nerves is assumed to be involved in both demyelinating and axonal types of Guillain-Barré syndrome (GBS). However, electrophysiological examinations have not yet clarified if this segment is involved. We measured cauda equina conduction time (CECT) in nine demyelinating GBS and seven axonal GBS patients. Compound muscle action potentials (CMAPs) were recorded from the abductor hallucis muscle. Electrical stimulation was given at the ankle and the knee, and magnetic stimulation was given over the first sacral (S1) and first lumbar (L1) spinous processes using a magnetic augmented translumbosacral stimulation (MATS) coil. CECT was obtained by subtracting S1-level latency from L1-level latency. CECT was prolonged in all the patients with demyelinating GBS who had leg symptoms, whereas motor conduction velocity (MCV) at the peripheral nerve trunk was normal in all the patients. In all the patients with axonal GBS having leg symptoms, CECT and MCV were normal and no conduction blocks were detected between the ankle and the neuro-foramina. The cauda equina is much more frequently involved than the peripheral nerve trunk in demyelinating GBS. In axonal GBS, usually, CECT is normal and segmental lesions are absent between the ankle and the neuro-foramina. Therefore, the CECT measurement should be very useful for directly detecting demyelinating lesions in GBS.

1. Introduction

Guillain-Barré syndrome (GBS) is an acute peripheral neuropathy that usually follows a respiratory or intestinal infection; it reaches its nadir within 4 weeks and then the patients recover over weeks or months [1,2]. Some immune-mediated pathogenesis such as anti-ganglioside antibodies or some other circulating factors might be involved in the disease process [3,4]. Based on the electrophysiological and pathological findings, GBS is currently classified into demyelinating and axonal forms: demyelinating GBS (acute inflammatory demyelinating polyneuropathy: AIDP) and axonal GBS (acute motor axonal neuropathy: AMAN) [2,3]. In demyelinating GBS, nerve conduction studies (NCSs) more frequently show a prolonged distal latency of compound muscle action potentials (CMAPs) and a conduction block at the common site for entrapment neuropathy compared to the slowing of motor conduction velocity (MCV). The F-wave technique also often shows prolonged latency or unobtainable F-waves [5,6]. In axonal GBS, on the other hand, NCS does not show any severe conduction delays. A conduction block (reversible conduction failure) at the nerve terminal axon or a common site for entrapment neuropathy is observed [7,8]. As the mechanism of a conduction block in axonal GBS, sodium-channel dysfunction has been postulated. F-waves are also often unobtainable. On the basis of these electrophysiological findings, it is speculated that the sites vulnerable to the lack, destruction, or malfunction of the blood nerve barrier are preferentially involved rather than the peripheral nerve trunk in both types of GBS. As proximal lesions, the conduction block at the spinal nerve roots, including the cauda equina, prolonged refractoriness of the most proximal axon for backfiring, or decreased excitability of spinal motoneurons are assumed to produce F-wave abnormalities [8,9]. The disappearance of F-waves is frequently the sole abnormal finding in both types of GBS [10]. In such cases, we cannot accurately classify GBS into a demyelinating or axonal form. Even when F-waves are elicited, the F-wave technique does not localize the lesion sites in the peripheral nerves. Therefore, electrophysiological examinations have not yet clarified if the cauda equina is involved.

We have recently developed a novel magnetic stimulation method to measure cauda equina conduction time (CECT) using a specially devised powerful coil called a magnetic augmented translumbosacral stimulation (MATS) coil [11,12]. This enables us to activate the spinal nerves at both the proximal and distal sites of the cauda equina and to measure cauda equina conduction time (CECT) [13,14], which reflects the nerve conduction within the cauda equina. Furthermore, using the MATS coil, supramaximal stimulation can be achieved at the neuro-

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http://dx.doi.org/10.1016/j.jns.2015.02.049
0022-510X/

foramina (the most distal cauda equina), which provides us with information on the presence of a conduction block between the distal site and the neuro-foramina [11,13]. In this study, to investigate the involvement of the proximal segment of the peripheral nerves in GBS, we measured CECT and tried to detect a conduction block at the proximal parts of peripheral nerves in demyelinating and axonal GBS using MATS coil stimulation.

2. Patients and methods

2.1. Subjects

We studied 16 GBS patients (12 men and 4 women) whose diagnosis was made according to the established diagnostic criteria [1]. The mean ± standard deviation (SD) age and body height of the patients were 44.1 ± 15.9 (range 27–65) years and 170.0 ± 7.2 (158–183) cm, respectively. These patients were classified into the two types of GBS according to the electrodiagnostic criteria of Hadden et al. [2]: 9 demyelinating GBS and 7 axonal GBS. The 9 demyelinating GBS and 7 axonal GBS also fulfilled the electrodiagnostic criteria of Ho et al. [3]: 9 AIDP and 7 AMAN. In all demyelinating GBS patients, the CMAP distal latency or F-wave latency was prolonged in at least two peripheral nerves. On the other hand, in all axonal GBS patients, the CMAP amplitude was decreased but all latencies were not severely prolonged. Patients in whom reliable CMAPs were not obtained by electrical stimulation were excluded from this study. If patients were not classified into the two types of GBS (equivocal GBS in the electrodiagnostic criteria of Hadden et al. [2] or unclassified GBS in Ho et al. [3]), NCS was repeatedly performed for the classification. The clinical profile of the studied patients is summarized in Table 1. Their disabilities were assessed using the Hughes functional grading scale (grade 6 = dead, grade 5 = requires assisted respiration, grade 4 = bed bound, grade 3 = able to walk 5 m with aid, grade 2 = ambulates independently, grade 1 = minimal signs and symptoms, able to run, and grade 0 = normal) [15].

Informed consent to participate in this study was obtained from all subjects. The procedure was approved by the Ethics Committee of the University of Tokyo and the study was conducted in accordance with the ethical standards of the Declaration of Helsinki.

2.2. Stimulation, recording, and analysis

During the examination, patients lay comfortably on a bed in the prone position. CMAPs were recorded from the abductor hallucis muscle (AH) on the more affected side. Disposable silver–silver chloride disc electrodes of 9 mm diameter were placed in a belly-tendon montage over AH. Signals were amplified with filters set at 20 Hz and 3 kHz and recorded by a computer (Neuropack MEB-2306; Nihon Kohden Corporation, Tokyo, Japan). The skin temperature was maintained at around 32 °C to 33 °C.

For NCS at a distal segment, the posterior tibial nerve was stimulated at the posterior medial malleolus of the ankle and the popliteal fossa using a conventional electrical stimulator (Neuropack MEB-2306; Nihon Kohden Corporation, Tokyo, Japan). MCV was calculated by dividing the ankle–knee length by the latency difference. To measure CECT, magnetic stimulation was performed with a monophasic stimulator, Magstim 200^2 (The Magstim Co. Ltd., Whitland, UK) and a MATS coil (diameter 20 cm, 0.98 T; The Magstim Co. Ltd., Whitland, UK) [11,12]. For the most distal cauda equina stimulation at the neuro-foramina, the edge of the MATS coil was positioned over the first sacral (S1) spinous process, which induces eddy currents to flow along the spinal nerves at their exit site from the spinal canal [11,13,14]. Stimulus intensity was gradually increased, and if possible, supramaximal CMAPs were obtained at the most distal cauda equina (neuro-foramina). For the most proximal cauda equina stimulation, the edge of the MATS coil

Table 1
Clinical profile and results of 16 GBS patients.

Case	Age	Gender	Experimental date	Hughes scale	MCV (m/s)	CECT (ms)	F-wave persistence (%)	F-wave latency (ms)	Anti-ganglioside antibodies	Muscular weakness
Demyelinating GBS										
D1	29	M	2 days	4	42	7.2 ↑	ND	ND	GD1b-IgG	Diffuse
D2	54	F	2 days	4	40	5.9 ↑	100	53.1 ↑	ND	Diffuse
D3	49	F	3 days	4	49	3.0	100	48.3	GQ1b-IgG GD1b-IgG GT1a-IgG GT1b-IgG	Diffuse
D4	65	M	3 days	3	44	8.6 ↑	100	58.9 ↑	ND	Diffuse
D5	58	F	8 days	1	45	6.2 ↑	100	50.0	ND	Distal dominant
D6	31	M	13 days	3	42	6.5 ↑	ND	ND	GM1-IgG GM2-IgG GalNAc-GD1a-IgM	Distal dominant
D7	56	M	14 days	4	40	5.8 ↑	100	54.3 ↑	GD1a-IgG	Diffuse
D8	58	M	15 days	1	39	6.2 ↑	100	50.0	NE	Distal dominant
D9	37	M	1 month	1	40	5.7 ↑	100	55.3 ↑	NE	Diffuse
Axonal GBS										
A1	38	M	5 days	2	45	4.0	93.8	43.0	GM1-IgG GM1-IgM	Distal dominant
A2	40	M	9 days	1	47	5.1	100	53.0 ↑	GM1-IgG GM1-IgM GA1-IgG	Distal dominant
A3	28	M	12 days	1	42	2.7	100	51.3	NE	Distal dominant
A4	58	F	13 days	1	51	5.3	100	46.9	ND	Proximal dominant
A5	27	M	16 days	3	41	2.9	87.5	52.6 ↑	NE	Distal dominant
A6	44	M	3 months	2	52	4.2	100	47.9	NE	Distal dominant
A7	33	M	3 months	4	49	4.1	100	45.4	ND	Diffuse
Normal values (mean ± SD, n = 20 normal subjects)					49.3 ± 4.4	3.7 ± 0.8		44.6 ± 3.0		
Lower limit or upper limit (mean − or + 2.5 SD)					38.3	5.7		52.1		

MCV: motor conduction velocity, CECT: cauda equina conduction time, SD: standard deviation, ↑: abnormal increment.
ND: not detected, NE: not examined.
Note: CECT and F-waves were measured at the same time (but not always at the time that the types of GBS were classified).