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ORIGINAL ARTICLE/ARTICLE ORIGINAL

Acute motor conduction block neuropathy pattern occurring in the course of an acute inflammatory demyelinating polyradiculoneuropathy

Neuropathie motrice aiguë démyélinisante avec blocs de conduction apparaissant dans le décours d'une polyradiculonévrite inflammatoire aiguë démyélinisante

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KEYWORDS
Acute motor conduction block neuropathy;
Multifocal motor neuropathy;
Acute inflammatory demyelinating polyradiculoneuropathy;
Guillain—Barré syndrome;
Conduction block

Summary
Objective. — To describe the case of a young woman with the diagnosis of acute inflammatory demyelinating polyradiculoneuropathy (AIDP), who during the course of the disease developed an electrophysiologic pattern of acute motor conduction block neuropathy (AMCBN).
Methods. — Electrophysiologic techniques including needle EMG, standard motor and sensory nerve conductions studies, and somatosensory evoked potentials were carried out over the four months after symptom onset.
Results. — The results of four neurophysiological studies, performed on Days 14, 26, 35 and 125 after symptomatic onset are reported. All immunological determinations including antiganglioside antibodies (GM1, GM2, GM3, asialoGM1, GD1a, GD1b, GD3, GQ1b and GT1b) were negative. The patient had a favorable evolution following treatment with intravenous immunoglobulins (IVIg).

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Conclusions. — We conclude that the electrophysiologic hallmark of AMCBN may occur in the course of AIDP. Serial investigation including proximal, intermediate and distal segments of all nerves from upper and lower limbs is essential for its detection.

MOTS CLÉS
Neuropathie motrice aiguë avec blocs de conduction ;
Neuropathie motrice multifocale ;
Polyradiculonévrite inflammatoire aiguë démyélinisante ;
Syndrome de Guillain-Barré ;
Bloc de conduction

Résumé
But. — Présentation du cas d'une jeune femme présentant un diagnostic de polyradiculonévrite inflammatoire aiguë démyélinisante (Piad) et qui, dans le décours de la maladie, développa un pattern électrophysiologique de neuropathie motrice aiguë démyélinisante avec blocs de conduction (NMADBC).
Méthodes. — Suivi électrophysiologique (incluant l'EMG à l'aiguille, les études standard des conductions motrices et sensitives, les potentiels évoqués somesthésiques) durant les quatre mois suivant la survenue des symptômes.
Résultats. — Nous rapportons les résultats des examens neurophysiologiques réalisés aux jours 14, 26, 35 et 125. Tous les tests immunologiques (anticorps antigangliosides : GM1, GM2, GM3, asialoGM1, GD1a, GD1b, GD3, GQ1b et GT1b) se sont avérés négatifs. La patiente évolua favorablement après traitement par immunoglobulines intraveineuses.
Conclusions. — Des caractéristiques électrophysiologiques de NMADBC peuvent apparaître dans le décours d'une Piad. Leur détection nécessite des évaluations répétées des conductions proximales, intermédiaires et distales de tous les nerfs des membres supérieurs et inférieurs.

Introduction

Acute motor conduction block neuropathy (AMCBN) can correspond to an unusual presentation of two different pathological conditions: Guillain—Barré syndrome (GBS) or multifocal motor neuropathy with persistent conduction blocks (MMNpCBs) [2,5]. Both entities can be distinguished on electrophysiologic grounds. Thus, while in GBS all CBs resolve in a few weeks or months, in parallel with clinical improvement, in the case of MMNpCB, CBs persist for more than three months in the same nerve trunks, independently of the clinical evolution. The most representative papers were published by Capasso et al. [2] for the former situation (AMCBN as variant of GBS) and by Lefaucheur et al. [5] for the latter one (AMCBN as an acute onset of MMNpCBs).

We report the case of a young woman who presented with clinical and electrophysiologic features compatible with the diagnosis of acute inflammatory demyelinating polyradiculoneuropathy (AIDP). During the course of the disease, she developed an electrophysiologic pattern resembling that described for AMCBN. We will discuss the pathophysiological, diagnostic and nosologic implications of our findings.

Patient and methods

Clinical case

A previously healthy 31-year-old Brazilian woman was admitted to our hospital because of progressive ''symmetric'' weakness and paresthesias of the four limbs. Fourteen days before, she had noted progressive tingling and numbness of the lower extremities. Three days before admission, she developed progressive bilateral leg weakness and numbness in both hands. Neurologic examination showed diffuse areflexia and mild stocking hypoesthesia. Strength was normal in upper limbs and decreased in lower limbs (MRC 4/5 in proximal muscles). All systemic laboratory tests were within normal limits. Cerebrospinal fluid revealed values for proteins and glucose within normal range. Serological tests were negative for *Campylobacter jejuni*, mycoplasma, borrelia, HIV, and hepatitis B and C viruses. All immunological determinations including antiganglioside antibodies (GM1, GM2, GM3, asialoGM1, GD1a, GD1b, GD3, GQ1b and GT1b) were also negative. An initial electrophysiologic study revealed results in keeping with the diagnosis of AIDP (see below). The patient was treated with intravenous immunoglobulins (IVIg) (0.4 g/kg per day for five days). On the next 12 days, she experienced a slight motor worsening in both lower limbs (MRC 3/5 in both quadriceps muscles). Then, a second electrophysiologic exam was performed. Over the next nine days, she experienced a discrete clinical improvement. On neurologic examination, tendon reflexes reappeared in upper limbs and stocking hypoesthesia was no longer observed, though a mild weakness (MRC 4/5) persisted in the proximal lower limb muscles. On Day 35 after symptom onset a five-day cycle of IVIg was administered. Later on, the patient noticed a slowly progressive clinical improvement and on Day 125 after the symptom onset, neurologic examination showed normal strength, lower-limb areflexia and normal sensory functions.

Electrophysiologic studies

Electrophysiologic techniques have been thoroughly described elsewhere [1]. They included needle EMG, standard motor and sensory nerve conductions studies (SNCSs), and somatosensory evoked potentials (SEPs). EMG was recorded with disposable concentric needles from tibialis anterior (TA), extensor digitorum brevis (EDB) and first interosseous dorsal (FID) muscles. Motor conduction velocity (MCV), distal motor latency (DML), compound muscle action potential (CMAP) amplitude and area from the baseline to negative peak were measured in the median, ulnar, peroneal and tibial nerves. F-waves were elicited by supramaximal stimulation at the wrist and ankle, and the minimum and maximum latency for 20 consecutive responses was