Brief Clinical Note

Acute fulminant brachial plexopathy with good recovery: Electrophysiological features

Shoji Hemmi, M.D.*, Katsumi Kurokawa, M.D., Taiji Nagai, M.D.,
Nana Izawa, M.D., Tatsufumi Murakami, M.D. and Yoshihide Sunada, M.D.

Abstract: We report a case of fulminant brachial plexopathy with radicular involvement. A 25-year-old man developed acute total monoplegia in the left upper limb. Needle electromyography showed extensive acute denervation in the C5-T1 spinal segments, and peripheral sensory nerve conduction was normal, mimicking a preganglionic lesion. However, left median somatosensory evoked potentials revealed abnormal Erb's point potential, suggesting a brachial plexus lesion. Corticosteroid treatment resulted in good recovery. These findings suggest that the primary pathophysiology was conduction block and this can explain the good clinical recovery in this patient.

（臨床神経 2012;52:436-438）

Key words：brachial plexopathy, cervical radiculopathy, conduction block, sensory nerve action potential, somatosensory evoked potentials

Needle electromyography (EMG) and sensory conduction study (SCS) are commonly performed in the workup of plexopathy as well as radiculopathy. When radiculopathy cannot be differentiated clinically from plexopathy, EMG and SCS are indicated because they can identify plexopathy. It is well known that results of SCS are unequivocally normal in radiculopathy because the dorsal root ganglia (DRG) are located distal to the roots and are not affected by the radiculopathy[1]. We report here a unique case of fulminant brachial plexopathy and radicular involvement. Although it was clinically considered that the primary lesion was in the brachial plexus, EMG and SCS findings suggested that the lesion sites were the spinal roots. However, abnormal somatosensory evoked potentials (SEPs) clearly revealed the presence of brachial plexopathy.

Case Report

A 25-year-old man noticed severe weakness and dysesthesia of the left upper limb when he awoke in the morning. The patient came to our hospital three days after symptom onset. At the time of the first evaluation, he was unable to move his whole left arm up to the shoulder. He did not complain of any pain in the shoulder area. There was no history of antecedent infection.

Neurological examination revealed flaccid total paralysis of the left arm including the hand, deltoid, infraspinatus, and serratus anterior muscles. The strength of the trapezius was preserved. There were decreased pinprick and vibration sensations in the left upper limb. Left biceps, brachioradialis and triceps reflexes were absent. Neurological findings were normal in the other extremities. Clinically, fulminant brachial plexopathy was considered because flaccid total monoplegia in the whole upper limb developed within three days.

Sedimentation rate was normal. An extensive collagen disease workup showed nothing. Magnetic resonance images of the cervical spine confirmed that there was no abnormal signal intensity and that there were no gadolinium-enhanced lesions in the spinal roots and brachial plexus.

Eight days after symptom onset, motor and sensory nerve conduction studies (NCS) were performed in the bilateral median and ulnar nerves. Results of the NCS were normal except for the absence of F waves on the left side. Since sensory nerve action potentials (SNAPs) were normal, the possibility of cervical radiculopathy was considered electrophysiologically. To determine the presence of cervical radiculopathy, EMG was performed in the left C5-8 and T1 innervated muscles including serratus anterior and C6-8 paraspinal mus-

*Corresponding author: Department of Neurology, Kawasaki Medical School〔577 Matsushima, Kurashiki, Okayama, 701-0192 Japan〕
Department of Neurology, Kawasaki Medical School
（Received: 21 November 2011）

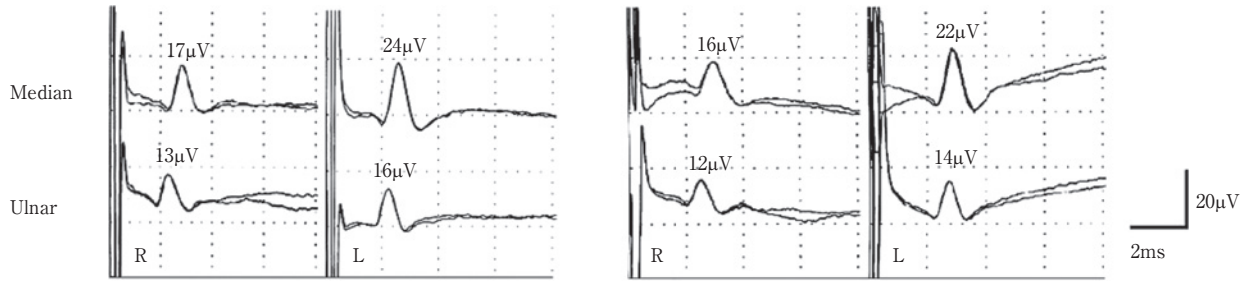

Fig. 1 Median and ulnar SNAPs obtained eight days after onset (left) and 22 days after onset (right). The number above each SNAP represents amplitude of the SNAP. SNAPs: sensory nerve action potentials.

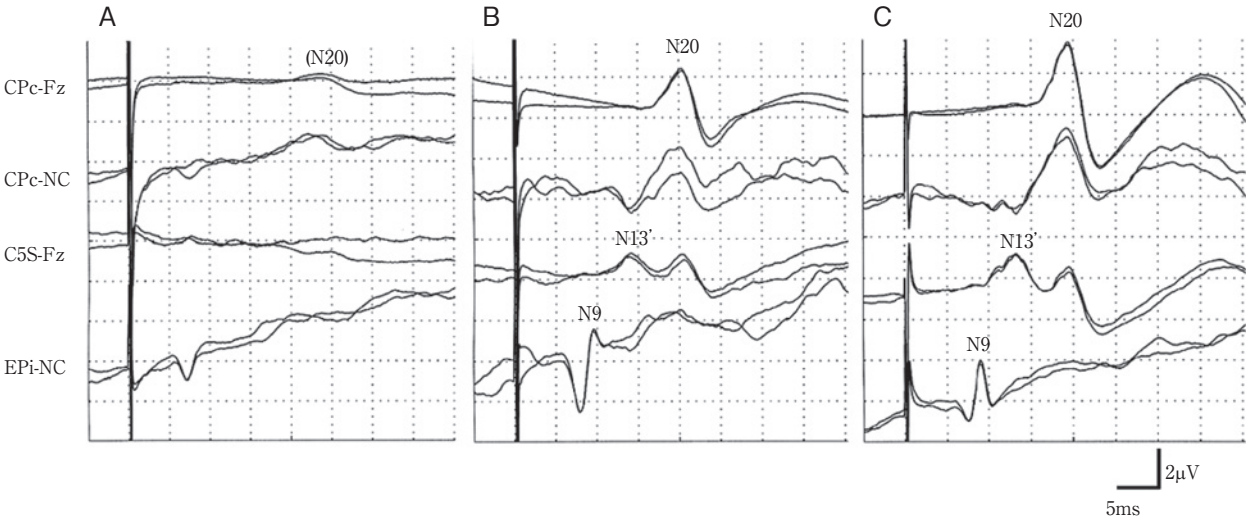

Fig. 2 Left median nerve SEPs obtained eight days after onset (A), 39 days after onset (B), and 75 days after onset (C). SEPs: somatosensory evoked potentials, CPc: centroparietal electrode contralateral to the stimulation, NC: non-cephalic reference electrode, EPi: Erb's point electrode ipsilateral to the stimulation.

cles. Increased insertion activity with +2 fibrillation potentials and positive sharp waves was present in all tested muscles, and the degree of spontaneous activities was not different in the muscles. No motor unit potential was generated with maximal attempt. Normal SNAPs were reconfirmed two weeks after the first evaluation (Fig. 1). EMG and NCS showed the presence of left C5-8 and T1 polyradiculopathy. However, abnormal Erb's point potential (N9) and no reproducible cervical (N13') or cortical (N20) potentials were shown in the left median nerve SEPs (Fig. 2A). The SEP findings were indicative of left brachial plexopathy.

After starting the patient on prednisone therapy (20 mg/day), his arm strength gradually improved and he was able to raise his arm completely within three months. His left hand grip recovered from 0 to 40 Kg. At 39 days after symptom onset, N9, N13' and N20 appeared (Fig. 2B). At 75 days after symptom onset, the SEP findings were completely normalized (Fig. 2C).

Discussion

The electrophysiological findings showed the existence of widespread lesions from the brachial plexus to the spinal roots. The abnormal Erb's point potential established that the primary lesion site was brachial plexus. If SEPs had not been obtained, we could not have demonstrated the presence of a brachial plexus lesion electrophysiologically. A SCS can show normal results despite a sensory defect in the following conditions: 1) central nervous system lesion, 2) lesion proximal to the DRG (radiculopathy), 3) lesion of the DRG (ganglionopathy) or axonal degeneration distal to the DRG (axonopathy) in the acute phase, and 4) conduction block distal to the DRG. Ganglionopathy or axonopathy can be discriminated from other conditions by a follow-up SCS after

the acute phase. The follow-up SCS will show reduced or absent SNAP in the case of ganglionopathy or axonopathy. In the present case, follow-up SCS showed no change in the SNAP amplitude, and we therefore excluded the possibility of ganglionopathy or axonopathy. Judging from the EMG and SCS findings that suggested radiculopathy, we could not have concluded that the lesion site was distal to the DRG. The presence of plexopathy was established electrophysiologically only after SEPs had revealed the conduction block to be distal to the DRG. In electrophysiological evaluation, SEPs are useful for distinguishing this condition from pure radiculopathy.

Recently, the clinical usefulness of magnetic stimulation for evaluating conduction block in the proximal regions of peripheral nerves has been reported[2]. Magnetic stimulation has rarely been used in the diagnosis of conduction block because supramaximal responses cannot always be evoked by using a conventional stimulator. Supramaximal responses could be obtained in most normal subjects by using a custom-built stimulator that was about 1.4-times more powerful than a commercially available stimulator. Proximal conduction block was thereby demonstrated by magnetic cervical motor root stimulation in a patient with neuralgic amyotrophy. We did not perform magnetic stimulation in the present case because a high-power stimulator was not available in our hospital. High-power stimulators are not available in most hospitals, whereas SEPs are much more convenient to use.

The present case differed from neuralgic amyotrophy since 94.9%[3] to 96.3%[4] of neuralgic amyotrophy patients have experienced severe neuropathic pain during their attacks. A similar case, radiculoplexopathy with conduction block caused by Epstein-Barr virus infection, has been reported[5]. The authors proposed that the conduction block was caused by immune-mediated focal demyelination. Although there was no history of antecedent infection, electrophysiological findings and prognosis in our patient resemble those of radiculoplexopathy caused by Epstein-Barr virus infection. The etiology of the present case was unclear, but this condition might have resulted from a postinfectious process. The finding of proximal conduction block indicated that focal demyelination was the primary underlying pathology. The clinical features suggest an immune-mediate mechanism because of a good response to steroid therapy.

When conduction block is the etiology of a plexus lesion, good clinical recovery is expected even if there is fulminant total paralysis of an upper limb. Because of the good prognosis of conduction block, differentiation between conduction block and axonal degeneration is critical in the management of patients. In this sense, SEPs and SCS are extremely helpful.

※The authors declare there is no conflict of interest relevant to this article.

References

1) Oh SJ. Principles of Clinical Electromyography Case Studies. Baltimore: Williams&Wilkins; 1998. p. 215-221.
2) Matsumoto L, Hanajima R, Matsumoto H, et al. Supramaximal responses can be elicited in hand muscles by magnetic stimulation of the cervical motor roots. Brain Stimul 2010;3:153-160.
3) Favero KJ, Hawkins RH, Jones MW. Neuralgic amyotrophy. J Bone Joint Surg Br 1987;69:195-198.
4) van Alfen N, van Engelen BG. The clinical spectrum of neuralgic amyotrophy in 246 cases. Brain 2006;129:438-450.
5) Vucic S, Palmer W, Cros D. Radiculoplexopathy with conduction block caused by acute Epstein-Barr virus infection. Neurology 2005;64:530-532.