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LETTER TO THE EDITOR

Foot drop of central origin: a misleading alteration of nerve conduction study

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Dear Professor Federico,

We present a case of foot drop of central origin in a 45-year-old woman who was diagnosed a malignant brain tumor in the motor area. Extensive neurophysiological assessment with nerve conduction study and electromyography (EMG) was performed during the diagnostic process and showed abnormalities which challenged us towards a comprehensive clinical and neurophysiological evaluation.

Foot drop is commonly considered a consequence of peripheral nervous system injury ranging from peroneal nerve palsy to L5 radiculopathy [1]. However, literature reports few cases of foot drop of central origin in which the paucity of upper motor neuron symptoms hindered the correct etiological diagnosis [2]. To our knowledge none of these reports includes extensive neurophysiological assessment (i.e., nerve conduction study and EMG). F wave is a late motor response produced by antidromic activation of motor neurons characterized by low-amplitude, high variability and ubiquity. F-wave latencies are a measure of the peripheral motor conduction time from the site of stimulation to the spinal cord and then return to the site of peripheral recording, e.g., target muscle. F-wave persistence, mean amplitude, area and duration might be influenced by affections of the central nervous system such as stroke [3], reflecting changes in central motor neuron excitability [4]. Currently no report has been made on alteration of F-wave parameters in the occurrence of brain tumors.

We present the case of a 45-year-old female who experienced an acute and progressive weakness in right foot dorsiflexion. Her familiar medical history was negative for neurological diseases as well as her personal clinical history. In August 2012 she attended a neurological outpatients clinic and underwent neurophysiological evaluation which showed absence of F-wave in the right peroneal nerve and was otherwise normal. Therefore she underwent lumbosacral neuroimaging in the hypothesis of disc protrusion, which resulted negative. In November 2012 the patient attended the outpatient clinic of our Department complaining progression of the symptoms. She underwent neurological examination, neurophysiological evaluation and neuroimaging. The neurological examination showed weakness in the right foot dorsiflexion with residual muscle strength 3/5, normal muscular tone and normal deep tendon reflexes in upper and lower extremities, with presence of the extensor hallucis longus deep tendon reflex, no sensory impairment, right plantar reflex in extension, left plantar reflex in flexion. The nerve conduction study was normal except for absence of F wave in the right peroneal nerve (see Table 1). Electromyographic examination was performed in vastus lateralis, tibialis anterior, gastrocnemius and extensor hallucis longus bilaterally; no abnormalities were observed in the explored segments except for a deficit of maximal effort recruitment in the right extensor hallucis longus. Neurophysiological and clinical findings suggested a weakness in foot dorsiflexion with foot drop of central origin. Cerebral MRI was performed in the hypothesis of a cerebral lesion and showed a tumour in the primary motor area (see Fig. 1).

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The patient was hospitalized immediately and underwent neurosurgery. At histological examination, the lesion proved to be a Glioblastoma Multiforme. The patient underwent subsequent radiotherapy and chemotherapy with temozolomide, and died in December 2013 after being hospitalized for brain tumour relapse.

Intracranial cause of isolated unilateral drop foot is rare; most of the cases reported in the literature are due to brain tumours. The diagnosis of the central etiology might be delayed in the absence of clear signs of upper motor neuron involvement, and in cases of concomitant lumbar spinal disease [2]. In our case careful neurological examination prompted us to suspect central disease (right Babinski sign).

However, given the more common peripheral etiology [1], neurophysiological examination is often performed early in the diagnostic process. Our patient presented with flaccid drop foot and showed alteration of nerve conduction study (F wave absence) which lead our colleagues to investigate for peripheral causes.

F wave abnormalities have to be interpreted at the light of clinical neurological signs (muscle tone and strenght, deep tendon reflexes and Babinski sign). Absence of F waves is usually interpreted as a sign of proximal demyelination, but a decreased motoneuron excitability might also be produced through an inhibition of descending supraspinal pathways. Indeed, F wave persistence, mean amplitude, area and duration are reduced in patients with acute upper motor neuron injury [4] also showing correlation with weakness severity, muscle tone impairment and the decrease in deep tendon reflexes. Later on in the disease progression, when patients present an increased muscle tone, we can find F-wave increased persistence, amplitude and duration [5]. These abnormalities are due to the activation of great motoneurons through altered interaction of inhibitory and excitatory mechanisms on segmental level: the disinhibition of descending supraspinal pathways produces an increased motoneuron excitability.

In conclusion, we report the case of F wave absence in a patient affected by Glioblastoma Multiforme of the primary motor area; we hypothesize that the absence of F wave was a result of reduced motor neuron excitability at spinal level caused by the focal corticospinal disruption; the reduced cortical output was also confirmed by the reduced maximal effort recruitment at EMG examination.

Table 1 Nerve conduction study, November 2012: the table shows nerve conduction study parameters recorded at our unit in November 2012

Motor					
Nerve	Latency (ms)	Amplitude (mV)	Morphology	F-Wave Latency (ms)	MNCV (ms)
Post tibial R	4.5	12.3	Normal	44.0	50
Post tibial L	4.8	11.3	Normal	42.3	48
Peroneal R	3.6	6.4	Normal	NV	53
Peroneal L	3.8	7.0	Normal	43.5	52

Sensory			
Nerve	Latency (ms)	Amplitude (uV)	SNCV (m/s)
Sural R	2.4	15.3	56
Sural L	2.7	17.0	54

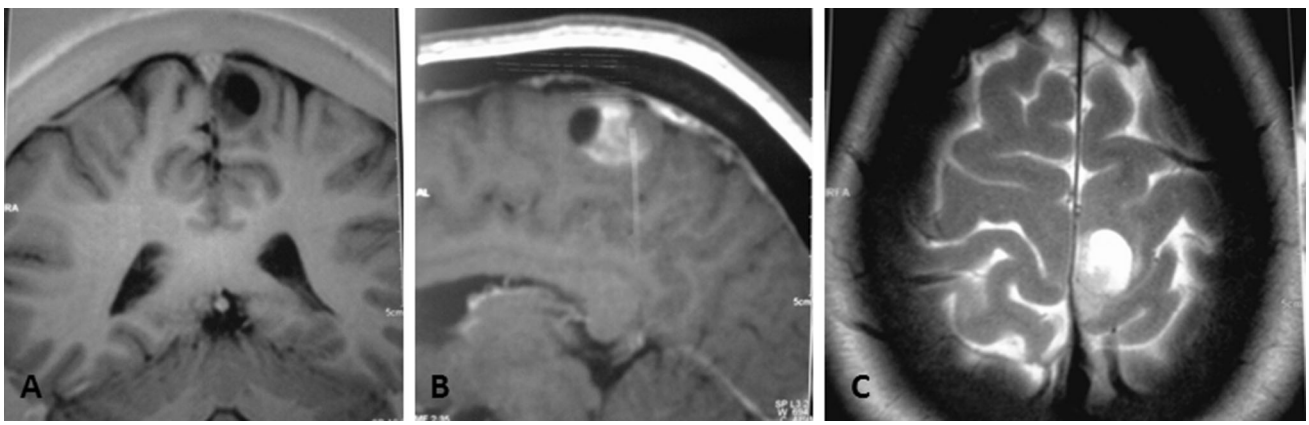

Fig. 1 MRI scan images of the patient in November 2012. **a** T1; **b** T1-gadolinium; **c** T2

This case reveals the primary role of the anamnesis (e.g., lack of pain and sensory symptoms) and challenges the neurophysiologist to carefully interpret nerve conduction parameters such as F-wave in the light of a careful clinical evaluation (e.g., Babinski sign). Moreover, a careful interpretation of maximal effort recruitment at EMG is necessary to discern central from peripheral nervous system involvement; in this context, quantitative EMG techniques could serve to evaluate frequency of motor unit action potential discharges.

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Compliance with ethical standards

Conflict of interest Nothing to report.

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