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Ultrasound and neurophysiological correlation in common fibular nerve conduction block at fibular head

Hiroshi Tsukamoto [a,*,1], Giuseppe Granata [a,1], Daniele Coraci [b], Ilaria Paolasso [b], Luca Padua [a,b]

[a] Institute of Neurology, Catholic University of Sacred Heart, Rome, Italy
[b] Don Carlo Gnocchi Onlus Foundation, Milan, Italy

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HIGHLIGHTS

- The study is focused on the occurrence of nerve abnormalities (size and echotexture) at ultrasonographic examination in fibular nerve conduction block and relationship with neurophysiological findings.
- The study shows that a normal ultrasound is usually present in a fibular nerve conduction block, but in case of additional axonal damage is associated with an increase of cross sectional area.
- The importance of our study is the possible diagnostic/prognostic value of the ultrasound in cases of fibular nerve conduction block: normal CSA might indicate a better prognosis.

ABSTRACT

Objective: Ultrasound (US) and neurophysiological examination are useful tools in the evaluation of common fibular mononeuropathy. There is only a report comparing US and electrophysiological parameters in patients with common fibular nerve (CFN) conduction block at fibular head. We investigated the correlation between US and neurophysiologic findings in this condition.
Methods: We retrospectively reviewed patients with CFN assessed in our lab during last 2 years. Each patient underwent to clinical, neurophysiological and ultrasound evaluations. Cross sectional area (CSA) of CFN at fibular head was assessed.
Results: Twenty-four patients were included. Motor nerve conduction study showed a reduction of distal compound muscle action potential (CMAP) amplitude in 10 patients (mean 1.3 mV). US showed an increased CSA in 10 patients. Statistical analysis revealed a strong correlation between the increased CSA and the CMAP reduction of CFN.
Conclusion: Our data suggest that usually US examination is normal in CFN conduction block at fibular head. However the association with axonal damage is frequently accompanied by an increase of CSA.
Significance: Ultrasound evaluation may represent a powerful diagnostic/prognostic tool in cases with CPN conduction block at fibular head because it usually shows normal pattern in pure conduction block and increase of CSA in associated axonal damage.

1. Introduction

Common fibular mononeuropathy is the most frequent mononeuropathy at lower limbs. The common fibular nerve (CFN) arises from sciatic nerve at popliteal fossa and runs around fibular neck where it divides into two terminal branches. At the fibular head the nerve is usually very superficial and not well protected by muscles and soft tissues, so at this level CFN is highly vulnerable for

* Corresponding author. Address: Institute of Neurology, Università Cattolica del Sacro Cuore, Largo F. Vito 1, 00168 Rome, Italy. Tel.: +39 06 30154435; fax: +39 06 35501909.
E-mail address: piroty@gmail.com (H. Tsukamoto).
[1] H. Tsukamoto and G. Granata contributed equally to this work.

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anatomical reasons (Katirji, 1999). Common fibular mononeuropathy may be due to several causes among which prolonged posture, surgical intervention, weight loss, trauma, bedridden condition, external compression from cast, arthrogenic cyst at the fibula, venous aneurysm, diabetes mellitus and idiopathic (Padua et al. 2002; Aprile et al., 2005; Jang, 2009). The diagnosis is based on clinical symptoms and signs and electrophysiological examinations, including electromyography (EMG) and nerve conduction study (NCS). Electrodiagnostic evaluation is extremely useful (1) to confirm the clinical diagnosis, (2) to establish the site of the fibular nerve lesion, (3) to assess the type of nerve damage, and (4) to predict the prognosis (Katirji, 1999). However electrophysiological study is not able to show the cause of nerve injury and to obtain anatomical information on nerve and surrounding tissues. High frequency probes have made ultrasound (US) useful in assessing nerve pathology and a growing body of literature supports the use of US in nerve assessment (Martinoli et al., 2000; Bianchi et al. 2003, 2004; Chiou et al., 2003; Reynolds et al., 2004; Padua and Martinoli, 2008). It is now well accepted that in nerve diseases, especially in nerve mononeuropathies, the combination of morphological data (obtained through US) and functional data (obtained through electrophysiology) is the best way to reach a correct diagnosis and obtain complete information for prognosis and therapeutic approach (Martinoli et al., 2004). Nerve conduction block is associated with a good prognosis and it is a common feature in common fibular mononeuropathy. In a previous study Aprile et al. reported that conduction block was present in 54% of patients with common fibular mononeuropathy (Aprile et al., 2005). Although some reports described US findings in common fibular mononeuropathy (Martinoli et al., 2000), to the best of our knowledge, there is only a report in literature comparing US and electrophysiological findings in cases of fibular nerve conduction block at fibular head (Visser et al., 2013). The aim of our study was to investigate the correlation between US and neurophysiologic findings in patients with CFN conduction block at fibular head.

2. Methods

The study was approved by local Ethics Committee of Università Cattolica del Sacro Cuore, Rome, Lazio, Italy. All participants provided informed consent.

We retrospectively reviewed all patients with common fibular mononeuropathy assessed in our EMG lab from January 2011 to May 2013. Inclusion criteria were:

(1) CFN conduction block, defined according the American Academy of Electro-diagnostic Medicine criteria: a drop in compound motor action potential (CMAP) amplitude of more than 50% and in the CMAP area of more than 40% stimulating distally to the fibular head respect to the proximal stimulation (American Association of Electrodiagnostic Medicine and Olney, 1999).

(2) Concomitant US evaluation of fibular nerve (US evaluation performed in the same day as the neurophysiologic study).

(3) Time elapsed from the symptoms onset more than 14 days, to exclude a pseudo-conduction block (the situation in which there is axonal damage but the distal axons are still excitable mimicking the conduction block; this situation can be present from the moment of nerve injury until approximately the following 10 days).

We excluded patients with known diagnosis causing nerve hypertrophy (hereditary neuropathy with liability to pressure palsy, chronic inflammatory demyelinating polyradiculoneuropathy, multifocal motor neuropathy, Guillain–Barré syndrome, Charcot-Marie-Tooth disease, neurofibromatosis), polyneuropathy (as diabetic neuropathy) or previous damage to the affected fibular nerve.

Neurophysiological evaluation consisted in the CFN motor nerve conduction evaluation and it was performed bilaterally. Neurophysiological examination was performed using an Oxford Synergy (Surrey, England) equipment. Skin temperature was controlled during NCS and maintained always at 32 °C or above. Motor nerve conduction studies were performed using our lab standard methods. Surface recordings were made from the extensor digitorum brevis. The fibular nerve stimulation was performed at the ankle, 2 cm below the fibular head, and above the fibular head at the lateral popliteal fossa. The popliteal fossa-fibular head segment must be $\geqslant$7 cm. Nerve conduction velocities, distal motor latencies (DML), and amplitude (negative phase) of all CMAPs were measured. We used as normal values those of our lab: DML <6.0 ms, motor nerve conduction velocity (MNCV) fibular head – ankle tract >40 m/s, MNCV popliteal fossa-fibular head tract >40 m/s, CMAP >1.5 mV. We considered also pathological CMAP lower than 50% of the contralateral side. We considered pure nerve conduction block the cases with normal distal CMAP. We considered mixed damage (axonal involvement plus conduction block) cases with reduced CMAP.

Strength tibialis anterior (TA), extensor hallucis longus (EHL), peroneus longus (PL) was scored with the Medical Research Council (MRC) scale. Sensory deficits were evaluated through a cotton wool.

After clinical and neurophysiological evaluation, US of the fibular nerve was performed. An Esaote MyLab 70 equipped with broadband linear transducers (frequency band 6–10 and 10–18 MHz) was used for US examination. US criteria for nerve identification were based on detection of the fascicular echotexture and anatomical landmarks. The two anatomical landmarks were the medial aspect of biceps femorii short head, proximally, and the lateral aspect of the fibular head, distally. The course of CFN was assessed from the fibular head to the popliteal fossa where the nerve splits from the sciatic nerve. We defined the fibular head tract as the portion included between 2 cm before the fibular head (proximally) and the splitting point in the terminal branches (distally). The remaining tract was considered as popliteal fossa. We measured and recorded the maximum CFN cross-sectional area (CSA) (Cartwright et al., 2008), using the ellipse method (Martinoli et al., 2000). Echogenicity changes, presence and localization of nerve abnormality, presence of extrinsic compression and anatomical relationship of the nerve with surrounding structures were also recorded. As normal CSA values we used our lab reference values (Padua et al., 2012): maximum CSA 12 mm^2 at fibular head and 8 mm^2 at popliteal fossa.

Statistical analysis was performed using SigmaStat 3.5. Spearman rank correlation test was used in order to evaluate the relationship between percentage of conduction block and maximal CSA value (max-CSA), and between max-CSA and distal CMAP value. Fisher's exact test was used for analysis of relationship between the presence of normal/reduced distal CMAP and the presence of normal/increased CSA at fibular head. The threshold of significance was defined as $p < 0.05$.

3. Results

We evaluated 65 patients with CFN. Twenty-four of them 14 men and 10 women fulfilled the inclusion criteria and were included in the study. Exclusion of 41 patients was due to the absence of conduction block or because of US and NCS was not performed the same day. Patients mean age was 41.7 years (range 12–80) and the mean interval between neurophysiological evaluation and onset of common fibular mononeuropathy was 2.6 months (range 15 days–12 months).