REVUE

Fibular nerve palsy after hip replacement: Not only surgeon responsibility. Hereditary neuropathy with liability to pressure palsies (HNPP) a rare cause of nerve liability

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Abstract

Mononeuropathy after surgery may occur and hereditary neuropathy with liability to pressure palsies is a possible pathological condition related to paresis after hip surgery. We present a case of 66-year-old man presenting severe weakness at inferior limb muscles after hip prosthesis revision. Clinic and electrophysiology showed severe right fibular nerve damage and ultrasound found a marked enlargement of the same nerve, associated with focal enlargements in other nerves. A diagnosis of hereditary neuropathy with liability to pressure palsies was suspected and confirmed by genetic test. The patient gradually recovered returning to a normal daily active life. Ultrasound was crucial for diagnosis. The suspicion and diagnosis of latent neuropathy, which can occur after surgical intervention, may lead to a better understand of the risks of the surgery, specific for the patient, and avoid the wrong attribution to surgical malpractice.

1 Introduction

Nerve lesions after orthopaedic surgery are not uncommon [1 2] and may be due to several well-known causes: direct damage by the surgeon, distractors, postures, ischemia and surgical instruments. The mononeuropathy arising after surgery may require weeks or months to recover, often incompletely [3] . Sometimes, although high attention is paid, nerve lesions occur without an ascertainable cause [4] and surgeons and anaesthetists are considered ethically and legally responsible for the damage. In some cases, an overlooked clinical condition may predispose to postoperative complications. Hereditary neuropathy with liability to pressure palsy (HNPP) is a possible disease related to paresis after orthopaedic intervention [5] . HNPP is an autosomal dominant hereditary demyelinating neuropathy caused by microdeletion of *PMP22* gene locates on the 17p11.2 [6 7] . The abnormal myelin in HNPP increases susceptibility to peripheral nerve damages by

involuntary compressions of nervous trunks, even during surgery. Due to the insidious features of HNPP, the incidence is still underestimation and we suspect that HNPP is not as unusual as it could be assumed (approximately 16/100,000) [8] .

We present a case of posterolateral approached hip replacement [9] in which an unrecognized HNPP played a crucial role for a post-surgical complication.

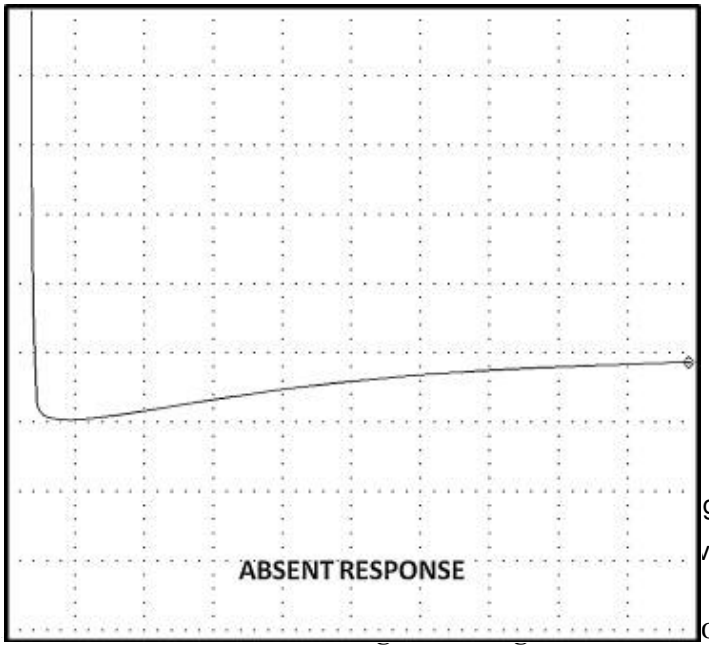
2 Observation

A 66-year-old Caucasian male affected by bilateral hip osteoarthritis, underwent primary left total hip replacement (THR) in 1998 and right THR in 1999 without complications using the same type of implant, a primary cementless prosthesis. A two-stage posterolateral approached revision was performed on the left side in January 2008 for septic loosening using a revision trabecular metal acetabular cup and a revision femoral stem with a complete functional recovering.

In 2013, the patient underwent to revision of the liner and head of the right side for an impending proximal aseptic loosening, due to polyethylene liner wear. During the latter procedure a peri-prosthetic greater trochanter osteolysis with a stable femoral stem was also observed, therefore a bioactive granular ceramic bone substitute was used to fill the area of bone resorption. No intraoperative complications were observed during the revision procedure on the right side, but the patient postoperatively, showed right fibular nerve palsy.

All the described surgical procedures were performed under general anesthesia by an expert hip surgeon using a posterolateral approach and preserving all the neurological structures (avoiding compressions at the fibular head level). No peripheral nerve block anesthesia was performed peri- or postoperatively. The patient immediately after surgery had severe weakness at tibialis anterior muscle (Medical Research Council, MRC, score 1/5) and extensor hallucis longus muscle (MRC score 2/5). He also showed sensory impairment to light touch in deep fibular nerve distribution with right-leg-localized numbness. Bilateral patellar and Achilles reflexes were reduced. No other neurological alterations were found.

The electrophysiological test, performed two weeks after the surgery, showed absence of motor and sensory response of fibular nerve and a diffuse slow conduction velocities associated with low action potential in the other nerves: sensory conduction velocity of right median nerve (at the third digit) 27 m/s (normal value, n.v., > 44 m/s) with an amplitude of 2 μV (n.v. > 4 μV); distal motor latency of right median nerve (registering from abductor pollicis brevis) 8.4 ms (n.v. < 4.0 m/s) (Fig. 1 (fig0005)).



gistering from extensor digiti brevis muscle
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on neurophysiologic assessment indicating a concomitant sensorimotor, mainly demyelinating, neuropathy. Nerve ultrasound (US) showed a bilateral marked enlargement of fibular nerve at fibular head (cross sectional area [CSA] 18 mm ^{2} at right side, 13 mm ^{2} at left side; n.v. at fibular head 12 mm ^{2}) and bilateral increased CSA of tibial nerve (37 mm ^{2}; n.v at popliteal fossa 10 mm ^{2}) (Fig. 2 (fig0010)). Because of clinical, neurophysiological and ultrasonographic findings, we suspected hereditary neuropathy with liability to pressure palsies (HNPP), and US evaluation of median and ulnar nerves were performed: the nerves presented focal enlargements at wrist and elbow level, respectively.

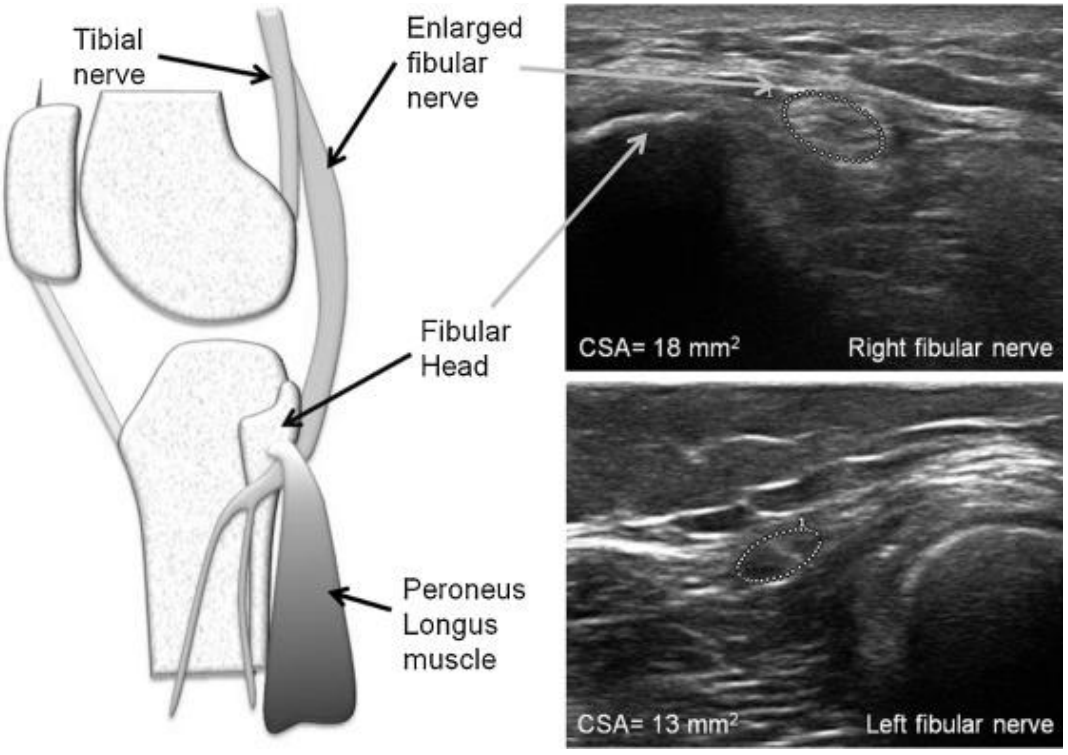

Fig. 2

Ultrasound (US) scan of fibular nerve. On the left side of figure, schematic representation of enlarged right fibular nerve with its anatomic relationships. On the right side, US evaluation of enlarged right and normal left fibular nerves.

After 3 weeks, needle electromyographic evaluation showed intense fibrillation in right tibialis anterior and right peroneus longus muscles.

Genetic test requested on the basis of neurophysiological and US findings confirmed the 17p11.2 microdeletion.

The patient gradually began to walk with sticks since 4 days after surgery and after 3 months, he returned to a normal daily active life. At 2 years follow-up, the patient is completely autonomous and able to walk even for long routes without any strut or pain but wearing a “Codivilla” spring.

3 Discussion

Our case suggests that suspecting and excluding diseases, such as HNPP before surgery, is extremely important to plan appropriate surgery and inform the patient about the possible risks of the operation. In fact, patients with diagnosis of HNPP may have an increased risk of nerve damage after surgery [5] . Furthermore, this risk is likely independent by intrinsic difficulties of the surgical procedure. At present, a real prevention is not always possible. However, knowing this condition may allow surgeons correctly informing the patients about possible postoperative complications and adopting specific precautions to prevent dangerous cases (for example, reducing compression of nerves for immobilization or manoeuvres). Clinical history assessment, focused on familiar and personal neurological elements, as past transient sensorimotor deficit related to posture or frequent history of transient paresthesia, results useful to select patients with latent HNPP. Moreover, performing a comprehensive neurological evaluation, focused on possible previous mild nerve palsies should be considered before a surgical intervention, in particular for orthopaedic surgery and can allow to screen patients needing further investigations.

These patients should be studied with accurate, fast and relatively inexpensive techniques: neurophysiological tests and US, as our case shows, may confirm or exclude a possible nerve liability to post-surgical complication [10 11] . The diagnosis of a latent neuropathy, which can occur after surgical intervention, may avoid the wrong attribution to surgical malpractice.

Disclosure of interest

The authors declare that they have no competing interest.

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