Case report

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 understanding of the risks of the surgery, specific for the patient, and avoid the wrong attribution to surgical malpractice.

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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 post-operative 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 postsurgical 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 sub-
stitute 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 posterolat-
eral 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 distri-
bution 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 associ-
ated 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).

A severe fibular nerve damage was diagnosed based on neu-
rophysiological 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 mm² at right side, 13 mm² at left
side; n.v. at fibular head 12 mm²) and bilateral increased CSA
of tibial nerve (37 mm²; n.v at popliteal fossa 10 mm²) (Fig. 2).
Because of clinical, neurophysiological and ultrasonographic find-
ings, we suspected hereditary neuropathy with liability to pressure
palsies (HNPP), and US evaluation of median and ulnar nerves were

![Fig. 1. Motor nerve conduction study of fibular nerve, performed registering from extensor digiti brevis muscle and stimulating at ankle. The study showed absence of nerve response.](image)

performed: the nerves presented focal enlargements at wrist and
elbow level, respectively.

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.

![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.](image)
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