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REVIEW ARTICLES

Foot Drop After Hip Replacement: Case Illustration of Iatrogenic Peroneal Nerve Injury and Review of Literature

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Abstract

Introduction: Iatrogenic peroneal nerve injuries are rare, serious complications of orthopedic procedures, especially hip arthroplasty, hip fracture fixation, or prosthetic implantation. These injuries cause disabling functional deficits if not promptly recognized and treated. Early neurophysiological assessment and referral to a neurosurgeon are critical for recovery. [1-3]

Case Illustration: We report the case of a 60-year-old female patient who sustained a right femoral neck fracture after a fall. She underwent hip arthroplasty with a titanium prosthesis five days post-injury. Postoperatively, she developed complete ipsilateral foot drop and neuropathic pain with electric-shock sensations radiating from the knee to the lateral toes. Immobilization lasted over six weeks. Initial physiotherapy and daily TENS were ineffective.

At two months, electromyography showed absent motor activity in the common peroneal nerve. Repeat ENMG studies confirmed a persistent deficit. MRI excluded spinal pathology. Nutritional neurotrophic supplementation (alpha-lipoic acid and gamma-linolenic acid) administered by a neurologist was ineffective. Given the plateau neurological state, neurosurgeon consultation was recommended, and within a month, the patient underwent peroneal nerve decompression.

Discussion: Iatrogenic peroneal nerve injuries after hip procedures may result from direct trauma, limb positioning, or traction during surgery. [4, 5] Prompt exploration and neurolysis, combined with structured physiotherapy, can enhance functional outcomes.[6] Delayed recognition and mislabeling as neurapraxia remain frequent, leading to disabling deficits. [7, 8, 9]

Conclusion: This case highlights the impact of delayed recognition of iatrogenic peroneal nerve injury after hip arthroplasty. Early suspicion, neurophysiological confirmation, and timely referral to peripheral nerve specialists are essential for optimizing neurological and functional outcomes.

Keywords: iatrogenic injury, peroneus nerve, hip arthroplasty, delayed recognition, foot drop

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Introduction

Iatrogenic peroneal nerve injuries are rare, serious complications of orthopedic procedures, especially hip arthroplasty, hip fracture fixation, or prosthetic implantation. Most diagnosed cases of foot drop caused by peroneal nerve injury are secondary to iatrogenic causes, mainly hip surgeries.[10]

The common peroneal nerve is a distal major branch of the sciatic nerve, originating from the L4–S2 nerve roots and coursing distally between the biceps femoris muscles until reaching the fibular head. At this level, the common peroneal nerve bifurcates into the superficial and deep branches—sensory and motor, respectively.

The superficial peroneal nerve supplies sensation to the posterolateral leg, the dorsum of the foot, and the first two toes; the deep peroneal nerve is responsible for dorsiflexion and innervates the tibialis anterior and extensor digitorum muscles. Therefore, peroneal nerve injuries typically present with tingling, pain, and electric-shock-like sensations in the corresponding dermatomes, or with gait impairment, such as steppage. [1, 18]

Between 8–25.4% of all peripheral nerve injuries are iatrogenic. Reported incidence ranges from 0.1–0.2% to 3.7%, whereas in hip arthroplasty it is estimated at 2–6%. Incidence of iatrogenic injury in revision arthroplasties is approximately 2–7%, with the risk of neural injury being three times higher compared with primary procedures. Iatrogenic factors are reported as the most common cause of sciatic nerve injuries (11%).

Neuropraxia of the external (motor) popliteal branch of the sciatic (peroneal) nerve, caused by traction, is the most frequently reported iatrogenic neurovascular injury in hip arthroscopy, with an estimated incidence of 0.05–1.9%. Dellon reported that 58% of patients with post-arthroplasty foot drop had peroneal neuropathy. Sunderland reported up to 2% transient nerve palsy following hip prosthesis implantation, with 0.5% resulting in permanent injury.

This incidence is expected to rise due to population aging and the increasing number of hip replacements [3, 10, 12, 13, 16]

The orthopedic community classifies nerve damage into three subgroups. Grade I nerve injury involves damage to nerve continuity during surgery, even though the nerve was not a surgical target—this is the most common iatrogenic injury. Grade II injury involves fascicular or continuity damage when the nerve itself is the target of surgery. Grade III injury refers to intentional iatrogenic nerve damage, such as nerve harvesting for transplantation. [3]

Sunderland and Seddon's classifications provide a practical framework for identifying the level and severity of nerve injury, guiding preoperative planning. According to *Sunderland:*

- *Grade I* – Neuropraxia involves transient motor conduction block or decreased CMAP due to localized compression, causing demyelination and temporary interruption of orthograde axonal transport. SCV may be altered, presenting as tingling, electric-shock-like pain, paresthesia, and motor deficits. Nerve continuity and distal excitability are preserved, allowing spontaneous recovery within three months.
- *Grade II* – Axonotmesis involves axonal disruption with preservation of endoneurial tubes. It is characterized by Wallerian degeneration. ENMG shows sharp waves, polyphasia, or fibrillations within the first 21 days, or absence of CMAP/SCV, depending on lesion length. Recovery is slower and depends on the length of the affected segment. Neuropathic pain and neuroma formation may occur.
- *Grade III* – Neurotmesis is a complete loss of nerve continuity, producing flaccid paralysis with loss of vasomotor and sudomotor function, dry skin, and loss of sensation and proprioception. Early diagnosis and intervention yield recovery rates of up to 85%. [1, 3, 12, 13, 16, 18]

These injuries cause significant functional impairment if not promptly recognized and treated. Neurovascular injuries are among the most common iatrogenic complications in orthopedic surgery, particularly arthroscopy (the second most common). Although hip arthroplasty is widely practiced (accounting for 80% of orthopedic procedures in the UK over the past decade), minimally invasive techniques remain less familiar and are associated with higher rates of nerve injury. Less common approaches expose nerves to greater iatrogenic risk. Early neurophysiological assessment and neurosurgical referral are essential. [1-3, 12, 18]

Case Illustration

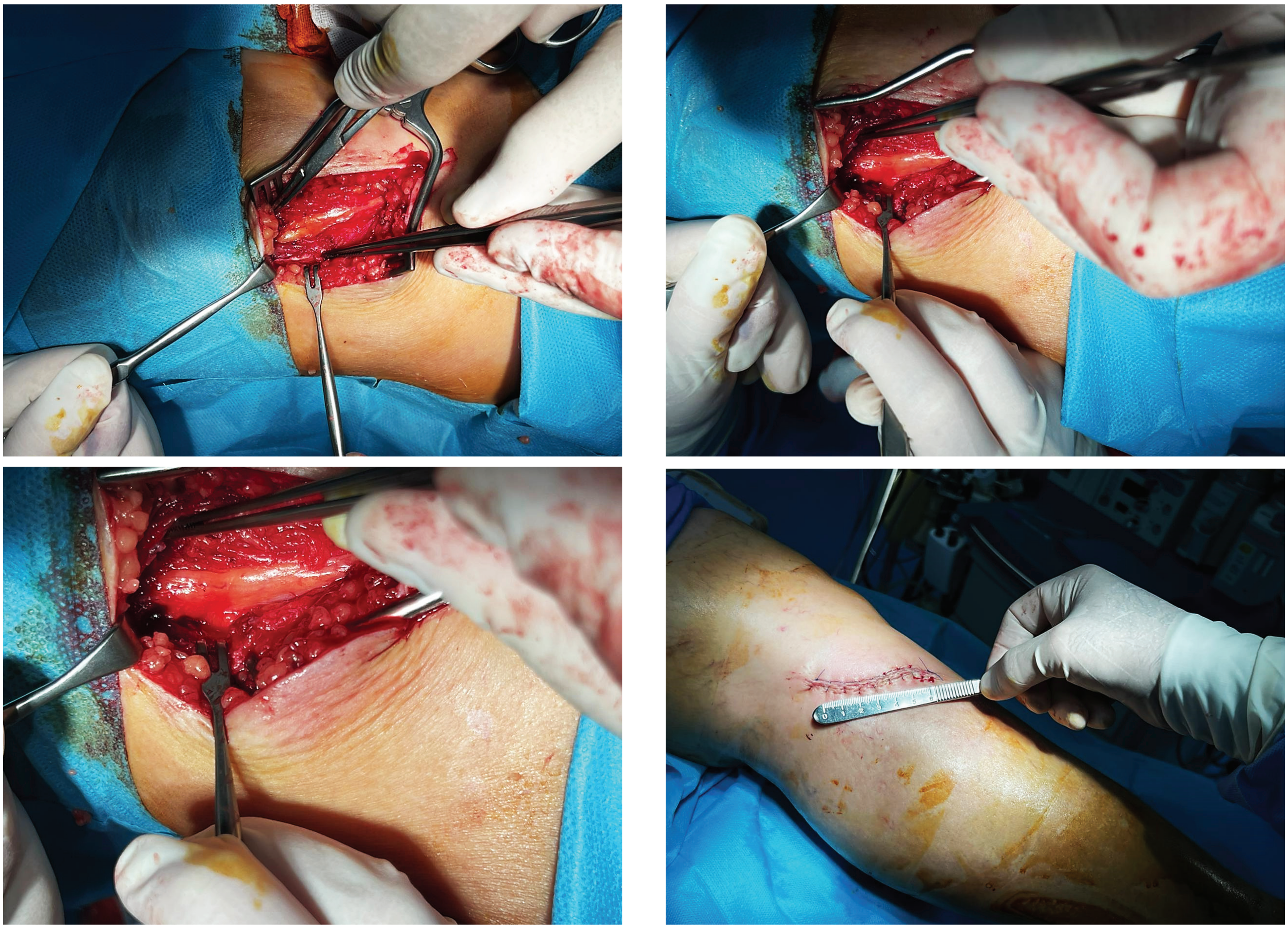
We report the case of a 60-year-old female who sustained a right femoral neck fracture after falling from a chair, landing directly onto her right thigh. She presented with persistent pain and functional impairment for seven months.

During the first 24 hours after the injury, she remained immobilized due to severe pain. At 48 hours post-trauma, her pain worsened, prompting evaluation in the Emergency Department, where radiographs confirmed a right femoral neck fracture. She underwent titanium cocco-femoral prosthetic replacement five days later.

Immediately postoperatively, she developed complete ipsilateral foot drop, accompanied by electric shock–like paresthesia, dysesthesia, and radiating neuropathic pain from the right knee to the anterolateral leg and digits II–V, worsened by weight-bearing. Due to symptom severity, she remained bedridden for approximately 1.5 months. A 10-day course of physiotherapy and twice-daily TENS sessions produced no improvement.

Two months following surgery, persistent symptoms with plateau evolution led to neurological consultation. ENMG demonstrated an absent motor response (0 mA) of the right common peroneal nerve, and a lumbar spine MRI was performed due to associated radicular-type pain. A repeat ENMG one month later confirmed unchanged findings. She was started on alpha-lipoic acid and gamma-linoleic acid supplements and referred for neurosurgical evaluation.

Neurological examination showed: Steppage gait, limping walk, complete right foot drop (FM 0/5), deficient digital flexion/extension excursions, Hypoesthesia of digits II–V, impaired toe abduction, Positive Tinel sign at the right fibular head (common peroneal nerve) and at the tarsal tunnel (posterior tibial nerve). ENMG classified the lesion as severe right peroneal neuropathy.



Photos 1, 2, 3, 4. - Intraoperative photos courtesy of Prof. Dr. Ridvan Alimehmeti showing a 6cm incision revealing the decompressed peroneal nerve.

At the Neurosurgery Service, decompression of the right common peroneal nerve was performed under local anesthesia (lidocaine–bicarbonate 4:1). A curvilinear incision at the level of the lateral gastrocnemius extending proximally toward the fibular head allowed identification of the nerve. Dense fibrotic adhesions surrounding the nerve were released. Three anterior and distal lateral fascial compartments of the superficial peroneal nerve were also decompressed. Extensive adhesiolysis was completed, hemostasis was achieved, and the wound was closed in layers.

The patient was discharged the same day with instructions for wound care and electrostimulation of the tibialis anterior and extensor digitorum muscles.

Discussions

Iatrogenic nerve damage is most commonly caused by extreme intraoperative joint positioning for prolonged periods, direct surgical trauma (misidentification of nerve vs. tendons or vessels), neurotoxic injections, prolonged traction, laceration, stretching, mechanical pressure from retractors, improper trocar placement, anatomical distortion (e.g., dysplasia, dislocation), tissue swelling, tourniquets, fixators, intraoperative bleeding, thermal injury, misplaced hardware, entrapment within fractures, ischemia, and compression. Approximately 94% of injuries result from direct mechanisms, including suturing, penetration, osteosynthesis removal, cuts, elongation, puncture, avulsion, drilling, or crushing. Indirect mechanisms include inadequate surgical exposure. Postoperative dressings, compartment syndrome, radiation, compression, or hematoma may also cause Grade I injury. [2-4, 11-19]

Orthopedic surgeons often place non-absorbable sutures around nerves suspected of intraoperative injury to facilitate later identification, but this can cause painful neuroma and fibrotic entrapment. This practice should cease. Any suspicion of nerve damage should prompt immediate exploration and repair, ideally by a neurosurgeon.[18]

Early diagnosis is crucial, yet underdiagnosis persists due to poor understanding, inadequate examination, medical negligence, failure to recognize clinical signs, and challenges in distinguishing degenerative from non-degenerative lesions. In the early 1990s, some UK cases were diagnosed as late as 40 months post-injury, while a decade later, delays of up to eight years continued. New Zealand reported that one-third of all iatrogenic nerve injuries originated from orthopedic procedures. A Royal College of Surgeons study found that 80% of 100 orthopedic patients treated for iatrogenic injuries showed reinnervation. [2, 6, 18]

Delayed diagnosis derives from inadequate neurological examination and poor understanding of nerve injury pathophysiology, resulting in overdiagnosis of neuropraxia, giving inadequate follow-up, and deepening nerve injury. Delayed diagnosis contributes to complex regional pain syndrome, which includes cortical hypersensitivity, neuropathic pain, trophic skin changes, sweating abnormalities, sensory loss, increased infection risk, ulceration, muscle atrophy, joint contracture, limb deformity, and musculotendinous shortening [18]

TENS (Transcutaneous electrical nerve stimulation) therapy enhances reinnervation when applied twice daily, five days per week, beginning immediately after suture removal; however, stimulation should not exceed one hour per day. LLT (Low-Level Laser Therapy) promotes neural sprouting and Schwann cell proliferation. [1, 13]

A comprehensive review by the Grünsburg Neurosurgical Department (1990–2012) found that 25% of iatrogenic sciatic nerve injuries were Grade I or II. Based on this, an algorithm was developed recommending immediate intraoperative exploration and microsurgical repair when nerve injury is suspected. Among 340 iatrogenic injuries, seven involved hip procedures. [4]

The Texas Nerve and Paralysis Institute's 17-year review of 28 iatrogenic foot-drop cases identified seven caused by hip replacement, typically due to traction injuries extending to the myoneural junction. [10]

A four-year Spanish retrospective study found a 0.65% incidence of iatrogenic neurovascular injury, with orthopedic procedures as the second leading cause. Among nerve injuries associated with hip surgery, all involved the external popliteal division of the sciatic nerve. [13]

Intraoperative monitoring (IOM) and intraoperative ENMG can help prevent iatrogenic injuries, though prognosis remains guarded in ischemic or severe axonal injuries. Preventive strategies include repeated position checks, proper retractor placement, limiting lithotomy position, monitoring tourniquet duration, and careful postoperative neurological exams. [10, 12, 14]

Surgeons, of any specialty, should know how to recognize, acknowledge, mitigate, and apologize (RAMA). [3]

After repair, nerves regenerate at approximately 1 mm per day in Grade III injuries and 2–3 mm per day in Grade I–II injuries, though tension at the repair site halts regeneration. In cases of irreversible paralysis resulting from delayed diagnosis, tendon transfer can restore partial motor function. [3]

Postoperative rehabilitation often requires mirror therapy, psychological support, cognitive therapies, meditation, and sometimes tricyclic antidepressants. [3]

Iatrogenic neurovascular injuries remain rare but underrecognized complications with significant medicolegal implications. The fibular head's superficial anatomy exposes the peroneal nerve to increased risk from compression and stretch. Nevertheless, the literature indicates that 83% of patients undergoing decompression, neurolysis, or nerve transfer achieve improved dorsiflexion. [10, 12, 13, 14, 18]

Conclusion

Iatrogenic peroneal nerve injuries after hip procedures may result from direct trauma, limb positioning, or traction. [4, 5] Prompt exploration and neurolysis, combined with structured physiotherapy, can improve functional outcomes. [6]

Neurolysis reduces neuropathic pain.[2] Delayed recognition and mislabeling as neurapraxia are common and lead to significant disability. [7-9]

Repeated postoperative neurological examinations are essential for identifying iatrogenic injury and preventing paralysis.[3] This case highlights the consequences of delayed diagnosis of iatrogenic peroneal nerve injury following hip arthroplasty. Early suspicion, neurophysiological confirmation, and timely referral to peripheral nerve specialists are key to optimizing neurological and functional recovery.

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