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MRI Correlation of Electromyographic Findings in Common Peroneal Neuropathy at the Fibular Head: A Case Report

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Abstract

Fibular (peroneal) neuropathy is the most common mononeuropathy of the lower limb and a frequent cause of foot drop, most often occurring at the fibular head. While electrodiagnostic studies remain the reference standard for functional assessment, Magnetic Resonance Neurography (MRN) plays an important role in localizing the site of nerve injury and identifying underlying structural causes. We report the case of a 37-year-old male presenting with progressive right foot drop without a history of trauma. Electrodiagnostic studies suggested compression of the common peroneal nerve at the fibular head with signs of active denervation. MRI of the knee demonstrated focal thickening and T2 hyperintensity of the common peroneal nerve at the fibular neck. No macroscopic compressive lesion was identified. This case highlights the complementary role of MRI in confirming electrodiagnostic findings, detecting denervation changes, and excluding structural causes in fibular neuropathy.

Introduction

Fibular (peroneal) neuropathy is the most frequent mononeuropathy encountered in the lower limb and the third most common focal neuropathy overall [1,2]. It typically presents with "foot drop" due to ankle dorsiflexion weakness, with the most common site of injury being at the fibular head.

While diagnosis traditionally relies on clinical examination and electrodiagnostic studies, Magnetic Resonance Neurography (MRN) has an increasing role in defining the precise site and extent of the disorder [1,3]. High-field MRI is particularly valuable for identifying compressive causes, assessing nerve continuity, and detecting secondary muscle denervation patterns to distinguish the condition from mimics like L5 radiculopathy [4]. This case report briefly outlines a patient presenting with foot drop where high-resolution imaging was essential in localizing the site of nerve insult and guiding management.

Case presentation

A 37-year-old male with no particular history of trauma presented with a one-month history of progressive right foot drop. The patient reported frequently crossing his legs. Neurological examination revealed a steppage gait and distal weakness of the right lower limb: tibialis anterior 3/5, extensor digitorum brevis 3/5, tibialis posterior 5/5, and gluteus medius 5/5. No hypoesthesia was noted. Electromyography demonstrated findings consistent with compression of the right common peroneal nerve at the level of the fibular head, associated with signs of active denervation. To further investigate the underlying cause, Magnetic Resonance Imaging (MRI) of the right knee was performed. MRI revealed focal thickening of the common peroneal nerve measuring approximately 4.5 mm in diameter, associated with increased T2-weighted signal intensity extending over a length of approximately 32 mm. No abnormal enhancement was observed following gadolinium administration. The signal abnormality was localized at the level of the fibular head, where the nerve courses around the fibular neck. No abnormalities were detected in the deep or superficial peroneal nerves (Figures 1 and 2).

A mild subcutaneous edema was noted adjacent to the fibular head. No perineural cyst was identified. There were no osseous abnormalities of the fibular head, no proximal tibiofibular joint arthropathy, and no proximal tibiofibular synovial cyst. Based on these findings, a diagnosis of focal neuropathy of the common peroneal nerve at the level of the fibular neck was established, without any identifiable

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macroscopic compressive cause on MRI. The imaging findings were associated with subtle signs of denervation in the muscles of the anterolateral leg compartment.

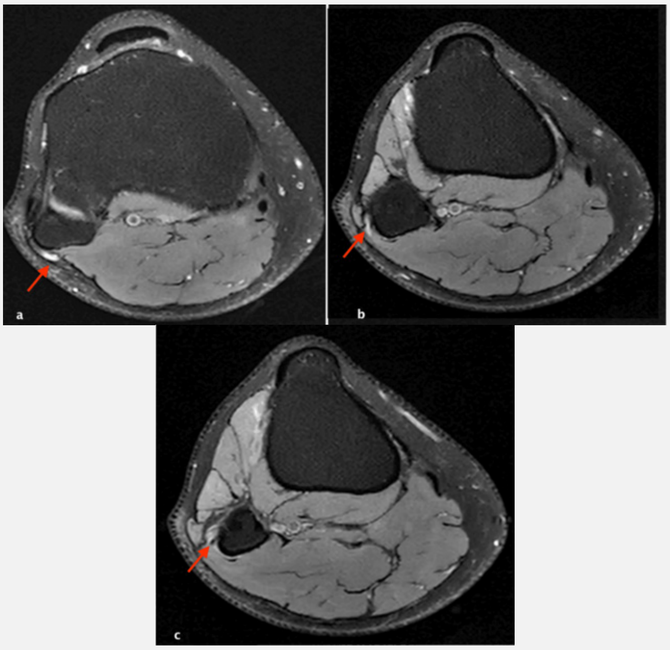

Figure 1: Axial proton density with fat saturation sequences showing the increased T2-weighted signal intensity in the common peroneal nerve (red arrows).

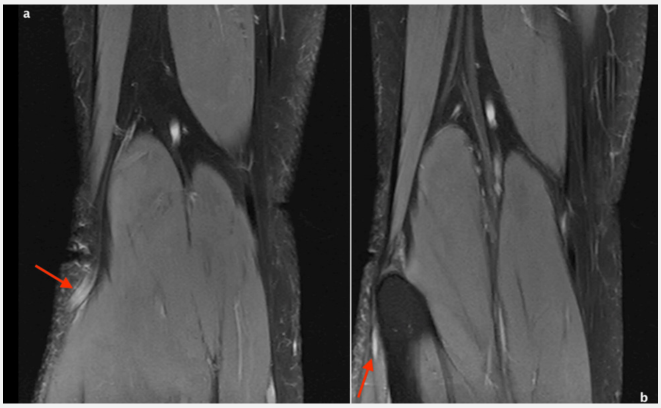

Figure 2: Coronal proton density with fat saturation sequences showing the increased T2-weighted signal intensity in the common peroneal nerve (red arrows).

Discussion

Fibular (peroneal) neuropathy is the most frequent mononeuropathy encountered in the lower limb and the third most common focal neuropathy overall, following median and ulnar neuropathies [1,2,5]. The most common site of injury is at the fibular head, where the nerve is superficial and relatively fixed, making it highly susceptible to external pressure or stretch [1]. Reported causes are diverse, ranging from acute trauma-such as knee dislocations, fractures, or lacerations-to chronic compression resulting from habitual leg crossing, improperly fitted casts, or significant rapid weight loss, a phenomenon often referred to as “Slimmer’s paralysis” [6]. In our case, the 37-year-old patient presented with a progressive onset without a history of trauma, which is characteristic of nontraumatic entrapment or habitual compression [7].

The hallmark clinical presentation of fibular neuropathy is “foot drop”, characterized by weakness in ankle dorsiflexion and great toe extension, which causes the toe to catch during ambulation and leads to a steppage gait [5]. Depending on whether the deep or superficial branch is more involved, patients may also exhibit weakness of eversion or sensory loss over the foot dorsum and the first web space [6]. However, sensory symptoms may occasionally be absent, as seen in some focal compression cases [1]. Our patient specifically demonstrated a classic steppage gait, highlighting the functional impact of the nerve compromise on the anterior compartment muscles.

The Role of EMG Electrodiagnostic (EDX) studies, including electromyography and nerve conduction studies, remain the traditional reference standard for evaluation [8]. They are essential for localizing the site of injury, determining the type of lesion (axonal vs. demyelinating), and assessing the potential for recovery [6]. In this case, the EMG was instrumental in confirming compression at the fibular head and identifying signs of active denervation, which provided the necessary clinical justification to proceed with targeted imaging. EDX studies are particularly useful to narrow the “diagnostic gap,” though they often require 2 to 3 weeks for denervation potentials to become evident [8].

While EDX assesses nerve function, Magnetic Resonance Neurography (MRN) is increasingly utilized to define the precise site, type, and extent of the disorder [3]. High-field 3-Tesla scanners allow for the visualization of nerves at the fascicular level, offering superior soft-tissue contrast [4]. Typical MRI findings in peroneal neuropathy include:

Nerve signal and caliber changes: Affected nerves typically show focal thickening (caliber increase) and increased T2 signal intensity. In our patient, the nerve measured 4.5 mm in diameter with hyperintensity extending over 32 mm at the fibular neck [2].

Muscle denervation patterns: MRI can detect secondary muscle changes, such as T2/STIR hyperintensity (edema) in the acute/subacute phase, which was observed in our patient’s tibialis anterior, EHL, and EDL muscles [4].

Differential diagnosis: This imaging pattern is crucial for distinguishing fibular neuropathy from mimics like L5 radiculopathy, which would additionally involve the posterior tibial and popliteus muscles [9].

Exclusion of mass lesions: MRI is vital for identifying space-occupying causes such as intraneural ganglia, tumors, or synovial cysts. In this case, the absence of any macroscopic compressive cause on MRI confirms a focal entrapment neuropathy likely related to localized fascial or positional factors [5].

Beyond MRI, high-resolution ultrasonography is a valuable, cost-effective tool for dynamic imaging, allowing for the detection of nerve thickening and real-time assessment of nerve excursion [2]. CT scans may be indicated if there is suspicion of underlying bony abnormalities or fractures [1].

Management depends on the cause and severity of the deficits. Conservative treatment including the removal of external compression (e.g., avoiding leg crossing), physical therapy, and the use of an Ankle-Foot Orthosis (AFO)-is often successful for nontraumatic cases [1,6]. Surgical intervention (neurolysis or decompression) is reserved for persistent symptoms, severe axonal loss, or identifiable mass lesions [1]. Regarding prognosis, the presence of any Compound Muscle Action Potential (CMAP) on motor nerve conduction studies is a strong predictor of a good long-term outcome [7].

Conclusions

In conclusion, while clinical examination and electromyography are the primary tools for diagnosing peroneal neuropathy, high-resolution MRI/MRN serves as an essential supplement. It provides a detailed "road map" by precisely localizing the nerve insult, assessing the severity through muscle denervation patterns, and ruling out macroscopic compressive causes or radicular mimics, thereby ensuring optimal clinical management.

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