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Case Report

Can a Fibular Osteophyte Cause Foot Drop?

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Abstract:

Fibular neuropathy at the knee is the most common lower-extremity mononeuropathy and is typically caused by external compression or trauma. We report a case of recurrent left foot drop due to an intrinsic osseous lesion. A 57-year-old man presented with progressive left foot drop over two to three months, preceded by one year of intermittent, activity-dependent episodes. Examination revealed steppage gait, severe weakness of ankle dorsiflexion (anterior tibialis 2/5, extensor hallucis longus 0/5), and sensory loss in the superficial fibular nerve distribution, with sparing of deep fibular, sural, and plantar territories. Electrodiagnostic testing localized a fibular neuropathy at the knee, demonstrating focal slowing across the fibular head, absent or minimally recordable extensor digitorum brevis responses, and active denervation of fibular-innervated muscles distal to the knee. Knee radiographs identified a lateral fibular head osteophyte adjacent to the fibular nerve, suggesting intrinsic compression as the etiology. This case underscores the value of knee radiographs in patients with new-onset foot drop and electrodiagnostic evidence of fibular neuropathy localized at the knee.

Key words: electrodiagnosis; electromyography; peroneal neuropathy; osteophyte; foot drop

Abbreviations:

Ankle Foot Orthosis **(AFO),** Anterior Tibialis **(AT),** Electrodiagnostic **(EDX),** Electromyogram **(EMG),** Extensor Hallicus Longus **(EHL),** Multiple Exostosis Syndrome **(MES),** Nerve Conduction Studies (NCS)

Introduction:

Foot drop is a common neurological presentation resulting from central, radicular, or peripheral etiologies, most frequently involving the fibular nerve at the knee. Fibular neuropathy is the most common lower-extremity mononeuropathy due to the nerve's superficial course around the fibular head, where it is vulnerable to trauma, habitual compression, and iatrogenic injury (1). Electrodiagnostic studies are essential for localizing the lesion, distinguishing fibular neuropathy from lumbar radiculopathy, and characterizing the severity and chronicity of nerve injury. This case presentation involves a remote foot drop following proximal fibular injury with subsequent complete motor recovery. While external compression and trauma account for most cases, intrinsic structural causes such as osseous abnormalities are less commonly reported. Osteophyte-related compression of the fibular nerve is rare and may present with fluctuating or progressive motor deficits, potentially delaying diagnosis. Recurrent fibular neuropathy at the same anatomical site decades after an initial injury is also uncommon and may reflect altered local anatomy or predisposition to focal compression. The aim of this case report is to describe a rare cause of fibular neuropathy due to a lateral fibular head osteophyte, highlight the electrodiagnostic features of acute axonal loss superimposed on chronic reinnervation, and emphasize the diagnostic value of knee radiographs in patients with new-onset foot drop and electrodiagnostic localization of fibular neuropathy at the knee.

Case Report:

A 57-year-old male presented with progressive left foot drop, worsening over two to three months. He had a history of proximal posterolateral left calf laceration 34 years prior with associated foot drop at that time. The

area was originally surgically explored and imaged. At that time, the nerve was in continuity without evidence of a lateral fibular osteophyte. The initial foot drop resolved after nine months following the use of an ankle foot orthosis (AFO) and physical therapy. The patient reported normal gait without lower extremity weakness for over 32 years but reported permanent numbness in the distal anterolateral leg and dorsum of the foot. At clinical presentation, the patient was in good general health, without weight loss, diabetes, or generalized neuropathy history. Gradual onset of intermittent and activity dependent, temporary left foot drop that lasted less than an hour was reported over the course of one year prior to clinical presentation. These episodes of foot drop were associated with left anterolateral leg pain, brought on by walking, and improved with rest. The foot drop was not associated with low back pain or classical radicular complaints. Symptoms were not related to squatting, abnormal positioning of the left knee, or crossing the legs. Physical exam findings at initial evaluation included steppage gait, left foot drop. On manual muscle testing 0/5 extensor hallicus longus function (EHL), 2/5 anterior tibialis (AT), and 4/5 peroneus longus and brevis motor power. There was sensory loss reported in the superficial fibular nerve distribution. Sensation was spared to the first web space (deep fibular), posterolateral calf and heel (sural nerve) and the medial and lateral plantar regions on the sole of the foot and toes (medial and lateral plantar nerves). A left fibular Tinel's sign was present at the knee by the fibular head. The remainder of the physical examination was normal, and manual muscle testing was within normal limits in proximal and distal muscles of the upper and lower extremities. Normal bilateral biceps, triceps, quadriceps, and Achilles reflexes as well as vibratory sensation and proprioception were observed. The patient had good peripheral pulses and distal capillary refill. There was no intrinsic muscular atrophy in the hands or feet.

Laboratory Findings:

Electrodiagnostic (EDX) evaluation was performed (Table 1). Common fibular motor conduction to the AT showed significant slowing at the fibular head, with normal amplitude, and without conduction block. The fibular motor response at the extensor digitorum brevis was barely recordable and no response was found proximal or distal to the knee. Needle electromyogram (EMG) revealed fibrillations limited to the left EHL and AT musculature. Motor unit analysis of the AT revealed large amplitude, long duration motor units without polyphasia, representing remote injury and reinnervation. Additionally, many AT motor units had variable amplitudes, durations, and degrees of polyphasia, and were considered to represent acute/subacute motor unit reinnervation consistent with the fluctuating clinical foot drop presentation. The abundant fibrillations combined with a predominance of small amplitude polyphasic motor units over the less frequent larger, more mature motor units evident in the AT indicated to the authors that the axonal loss was acute superimposed upon a remote denervation and reinnervation process. Muscles proximal to the fibular head in the fibular nerve distribution (biceps short head) as well as other L4, L5, and S1 myotome muscles and lumbar paraspinals were normal on needle EMG evaluation, ruling out a lumbar radiculopathy. We did not study the fibularis longus or brevis nor perform NCS of the superficial fibular nerve. Thus, we did not document involvement of the superficial fibular nerve, however the sensory symptoms on the dorsum of the foot make this likely.

Side	**Nerve**	**Stim Site**	**Seg (cm)**	**Onset Latency** (milliseconds)	**Norm** (milliseconds)	**Amplitude** (millivolts)	**Norm** (millivolts)	**Conduction Velocity** (meters/sec)	**Norm** (meters/sec)
Left	Right Tibial	**Ankle**	8	**4.1**	<6	**10.1**	>3.0		
		Popliteal	43	**13.4**		**7.9**		**46**	>41
Right	Left Tibial	**Ankle**	8	**4.6**	<6	**8.9**	>3.0		
		Popliteal	47	**13.6**		**8.7**		**47**	>41
Left	Left Deep Fibular	Ankle	9	**6.2**	<6.1	**0.2**	>2.2		
		Fibular Head	37	**NR**		**NR**		**NR**	>41
Right	Right Deep Fibular	Ankle	9	**4.4**	<6.1	**2.8**	>2.2		
		Fibular Head	37	**11.6**		**2.5**		**51**	>41
Left	Left Common Fibular	**Fibular Head**	9	**4.3**		**2.5**			
		Popliteal	16	**9.5**		**2.4**		**31**	

Table 1: (a) Nerve conduction data with sensory nerve conduction studies (NCS) to bilateral sural and left medial and lateral plantar nerves. (b) Nerve conduction data with deep fibular motor NCS to extensor digitorum brevis and common fibular motor NCS to anterior tibialis. The right side NCS is provided for comparison. Tibial motor responses are recorded from the abductor hallicus.

Table Abbreviations: Nerve conduction studies (NCS) A radiograph taken at the time of clinical presentation of the left knee revealed an osteophytic spur on the lateral proximal fibula in the transition area from the fibular head to the neck (Figure 1)

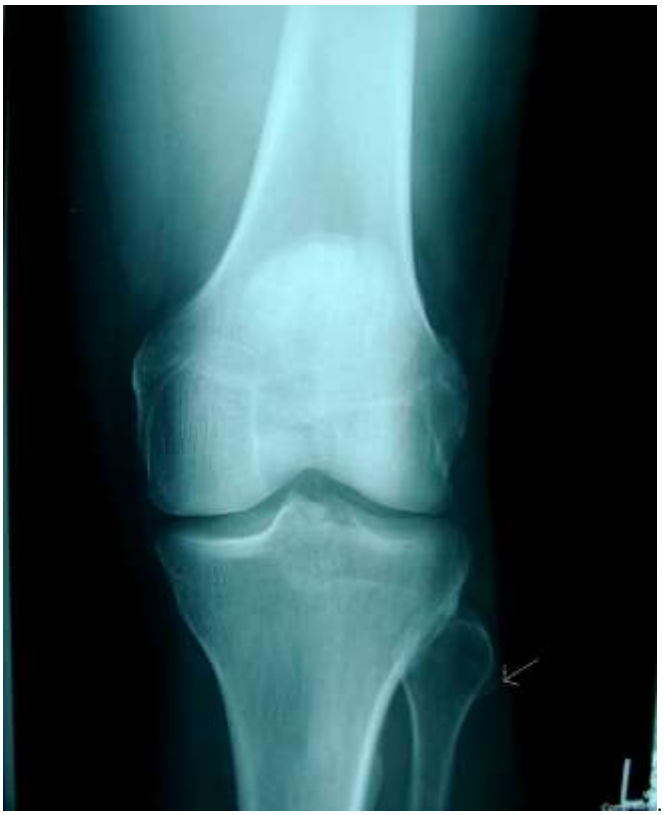

Figure 1: Patient's left knee radiograph depicting a 1cm lateral fibular osteophyte (arrow).

The patient was offered surgery but chose not to have surgical exploration and went on to have complete permanent foot drop over six months, requiring ongoing use of an AFO.

Discussion:

The fibular nerve is vulnerable to trauma due to its fixation as it courses around the fibular neck. The fibular nerve derives its arterial supply from small vessels branching off the popliteal artery. Compression of the fibular nerve at the fibular head can result in varying degrees of paralysis depending upon the nature of the compression or trauma. The differential diagnosis of "foot drop" is broad, including central nervous system etiologies, radiculopathies, as well as and not limited to generalized neuropathies [2]. Localized fibular injury at the fibular head is associated with osteochondromas (exostoses), subluxations of the knee, lateral compartmental capsule-ligamentous lesions, supination distortions, neoplasms, cysts, osteoid osteomas, as well as fracture, direct fibular laceration, gunshot wounds, traction, or direct surgical injury [1, 3, 4, 5]. Acute fibular lesions are most commonly associated with prolonged knee flexion, perioperative compression and subacute lesions with significant weight loss and recent prolonged hospitalization [3].

Fibular neuropathy due to an osteophyte is rarely reported. Fibular neuropathy associated osteophytes have been described at various locations on the fibula and tibia [5, 9]. We present a case with a unique location on the fibula in the transition area from the fibular head to the neck. We found only two case reports of fibular neuropathy due to a fibular head osteophyte. [1, 10]. We present the only case report with detailed EDX data. Our patient not only has a rare cause of fibular neuropathy but a rare location and EDX findings.

In pediatric patients there are case reports of resolution of foot drop with excision of associated fibular osteophytes [1]. It is our opinion that adult patients with foot drop correlating with a fibular head osteophyte may benefit from fibular nerve exploration and osteophyte excision. Our literature review revealed a 15-year-old with an osteophyte that closely resembles the location of the osteophyte in our case and is pictured in Flores' article [1]. Osteophytic formation in tubular bones has the potential to compress surrounding peripheral nerves [4]. The difference between simple exostoses and osteochondromas is histologic. There are two main histologic categories of benign osteophyte-like lesions that originate from the head of the fibula; a simple exostosis is a bony growth projecting outward, while an osteochondroma is a bony exostosis of proliferative bone capped by cartilage [1]. No epidemiological data exists currently to describe exostosis prevalence at the fibular head or the frequency of the exostosis subtypes. Radiographic appearance of these lesions is described as lateral extrusions of portions of the growth plate that cause an eccentric, cartilage capped bone prominence [11]. Purely cartilaginous exostoses are reported to occur less frequently than bony exostoses [1] and would not be visualized on plain radiographic imaging. MRI in this situation could visualize such a lesion. Only a few cases of cartilaginous exostoses causing fibular neuropathy have been described [5].

The most common reported etiology of osteophyte associated fibular neuropathy is multiple exostosis syndrome (MES) [1]. This autosomal dominant disease begins in childhood and is cited to cause peripheral nerve entrapments [11, 12]. Nerve compression of any peripheral nerve is rare in childhood MES, described as occurring in less than 1% of all cases [13]. The fibular bone exostoses occur in 8.2% of childhood MES and associated nerve compression is rare [14]. The frequency of exostosis involvement of the fibular head in adults has not been previously described and its association with fibular neuropathy is reported in this case. This case describes an osteophyte protruding from the fibular head area (Figure 1) as a probable contributing cause of fibular neural injury. The presentation is complicated because of remote fibular nerve trauma 34 years earlier. The patient reported complete motor recovery and normal AT function without foot drop for over 32 years.

We propose that this fibular neuropathy is due to 3 factors: a) osteophyte formation at the fibular head superimposed on b) the old fibular nerve

injury from 34 years earlier and c) aging [1, 15]. We interpreted the clinical presentation of activity related waxing and waning AT strength as neurogenic weakness due to remote and acute axonal loss. Age related axonal drop out was considered as one potential contributor to the foot drop. We considered this as the least contributing factor because he was not elderly, the weakness progressed relatively rapidly over 2-4 months, and there were profuse fibrillations, (more than that expected from normal aging). In individuals with prior nerve injury, it would be expected that the reduced number of alpha motor neurons that remain viable would innervate more muscle fibers with an expanded muscle fiber number and area, and large rapid firing motor units would be seen on recruitment with needle EMG [16]. These findings of large motor units were seen in the AT but was not the predominant finding evident in multiple areas of the AT muscle. In this case, fibrillations (most consistent with acute denervation) were evident, and the denervation potentials were of normal amplitude suggesting acute denervation of myocytes due to new injury to alpha motor axons in the fibular nerve that were unaffected by the prior trauma. The lateral fibular osteophyte in this case was considered to play a role in the reoccurrence of foot drop because of the timing with which foot drop progressed, advancing rapidly in the last two to three months prior to presentation. No evidence of polyneuropathy or a more proximal nerve lesion was found to be a contributing factor in this clinical presentation. From a practical clinical perspective, nothing could be done to reverse age related axonal loss. However, if the fibular neural functional decline was due to the fibular osteophyte, then it was considered that the patient might benefit from surgical exploration and potential osteophyte removal. This case is presented because the removal of the fibular osteophyte might have prevented complete loss of fibular motor function and improved the clinical foot drop. An additional benefit of this case presentation is that it presents a clinical diagnostic dilemma that involves consideration of a variety of factors when determining causation and potential treatment options of foot drop. The fibular osteophyte presented in this case represents a simple exostosis. Simple exostoses are common in skeletally mature adults and frequently seen in the elderly, but no case reports were found describing an association with fibular neuropathy distal to the fibular head in the adult population.

The evaluation of fibular neuropathy at the fibular head does not typically include a radiograph of the knee. A radiograph should be considered in this situation because it may provide an option for treatment. In this case no surgery was performed so we cannot be certain if decompression would have helped. Surgery to remove fibular exostosis in children has successfully resulted in resolution of foot drop [1]. No known report discussing recovery of fibular function after excision of exostoses in the adult population was found. If a proximal fibular osteophyte is associated with foot drop in an adult, we propose surgical excision may be a reasonable consideration.

Conclusions:

This case illustrates a rare cause of recurrent fibular neuropathy due to intrinsic osseous compression from a lateral fibular head osteophyte in an adult patient, occurring decades after a prior traumatic nerve injury. Although osseous causes of fibular neuropathy are more commonly described in pediatric or adolescent populations, adult presentations remain under-reported and may be overlooked in the diagnostic evaluation of foot drop. The combination of fluctuating clinical symptoms and electrodiagnostic evidence of acute axonal loss superimposed on chronic reinnervation enabled precise localization of the lesion to the fibular head and distinction from lumbar radiculopathy. This report emphasizes the importance of correlating electrodiagnostic findings with targeted imaging and supports consideration of knee radiographs in adult patients presenting with new-onset foot drop and electrodiagnostic localization of fibular neuropathy at the knee. Early recognition of structural etiologies may facilitate timely intervention and improve functional outcomes.

Clinical Implications and Limitations

This case highlights an under-recognized cause of recurrent fibular neuropathy in adults due to intrinsic osseous compression at the fibular head. The correlation of fluctuating clinical symptoms with electrodiagnostic evidence of acute axonal loss superimposed on chronic reinnervation allowed precise localization of the lesion and distinction from lumbar radiculopathy. Clinically, this supports consideration of targeted knee imaging, including plain radiographs, in adult patients presenting with new-onset or recurrent foot drop localized to the fibular nerve at the knee. Limitations include the single-patient design and limited generalizability; however, the rarity of this presentation in adults supports the value of this report in raising clinical awareness.

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