Isolated deep peroneal nerve palsy: Role of magnetic resonance imaging in localization

Samhita Panda, Mandaville Gourie-Devi, Ankkita Sharma, Aditi Sud[1]

Departments of Neurophysiology and [1]Radiology, Sir Ganga Ram Hospital, New Delhi, India

For correspondence:
Dr. Samhita Panda, Department of Neurophysiology, Sir Ganga Ram Hospital, New Delhi - 110 060, India.
E-mail: samhitapanda@yahoo.com

Ann Indian Acad Neurol 2015;18:451-453

Case Report

A 30-year-old gentleman presented with acute pain of right lower limb of moderate severity with difficulty in driving car for a month. There was no associated swelling or paresthesias. Pain subsided after few days and 2 weeks later, he had difficulty in walking and driving as he could not easily lift the right foot from accelerator to brake. There was no numbness in legs, weakness of other limbs, or craniobulbar muscles. There was no prior trauma, fracture of leg, or hypopigmented skin patches. He was neither diabetic nor suffering from any connective tissue disorder.

The patient was well-built with no significant abnormalities on systemic examination. Popliteal and dorsalis pedis pulses were palpable bilaterally and there was no redness, tightness, or swelling of affected limb. There was foot drop with significant weakness of right ankle dorsiflexors (Medical Research Council (MRC) Scale grade 1/5), toe dorsiflexors (grade 2/5), and mild weakness of right ankle evertors (grade 4-/5). There was no sensory loss or peripheral nerve thickening.

Blood sugar, lipid profile, and hematological examination were normal. Nerve conduction study (NCS) was normal and there was no conduction block along common peroneal nerve (CPN) on recording from extensor digitorum brevis (EDB). Electromyography (EMG) showed fibrillations and positive sharp waves with very few motor units and markedly reduced recruitment pattern in right tibialis anterior (TA); in EDB there were fibrillations and positive sharp waves, nearly normal motor units and slight reduction of interference pattern. No abnormality was noticed in right peroneus longus, medial gastrocnemius, and biceps femoris muscles [Table 1]. The findings suggested involvement of muscles innervated by right deep peroneal nerve (DPN). Magnetic resonance imaging (MRI) of right leg was performed using spin echo T1-weighted axial, coronal, and sagittal protocols. The CPN and DPN showed normal signal intensity with no extrinsic or intrinsic compression [Figure 1]. There was diffuse altered hyperintensity in right TA in its entire course in the leg as well as EDB with partial effacement of intermuscular fat planes [Figure 2a-d]. Normal signal intensity was noted in extensor hallucis longus, extensor digitorum longus, and peronei and tibialis posterior muscles.

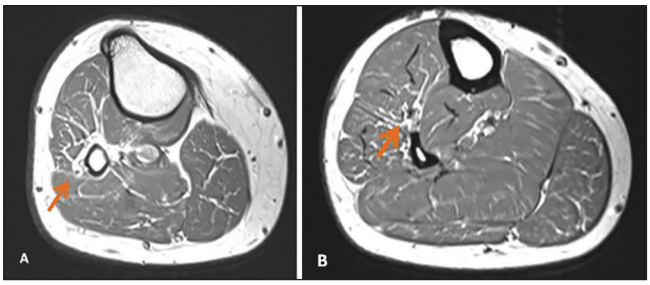

Figure 1: MRI of right leg (axial section) showing normal intensity in (a) common peroneal nerve and (b) deep peroneal nerve. MRI = Magnetic resonance imaging

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DOI: 10.4103/0972-2327.169646

How to cite this article: Panda S, Gourie-Devi M, Sharma A, Sud A. Isolated deep peroneal nerve palsy: Role of magnetic resonance imaging in localization. Ann Indian Acad Neurol 2015;18:451-3.
Received: 16-05-15, **Revised:** 25-06-15, **Accepted:** 01-07-15

Prednisolone 40 mg was started along with physiotherapy. Within 2 weeks, there was slight improvement of dorsiflexion of ankle and toes, which further improved and by 4 months, power of ankle and toe dorsiflexors was MRC 4+/5 and ankle eversion, MRC 4/5. Follow-up study showed normal NCS and EMG showed no spontaneous activity in the TA and EDB. High amplitude motor units were noted in right EDB suggesting reinnervation, while normal potentials were observed in right TA and peroneus longus. Mild reduction of interference pattern was seen in right TA, EDB, and peroneus longus [Table 1]. MRI of right leg now showed significant resolution of T2-hyperintensity in TA and EDB. Only mild edematous changes were seen in the subcutaneous fat planes of lower shin [Figure 2e-h].

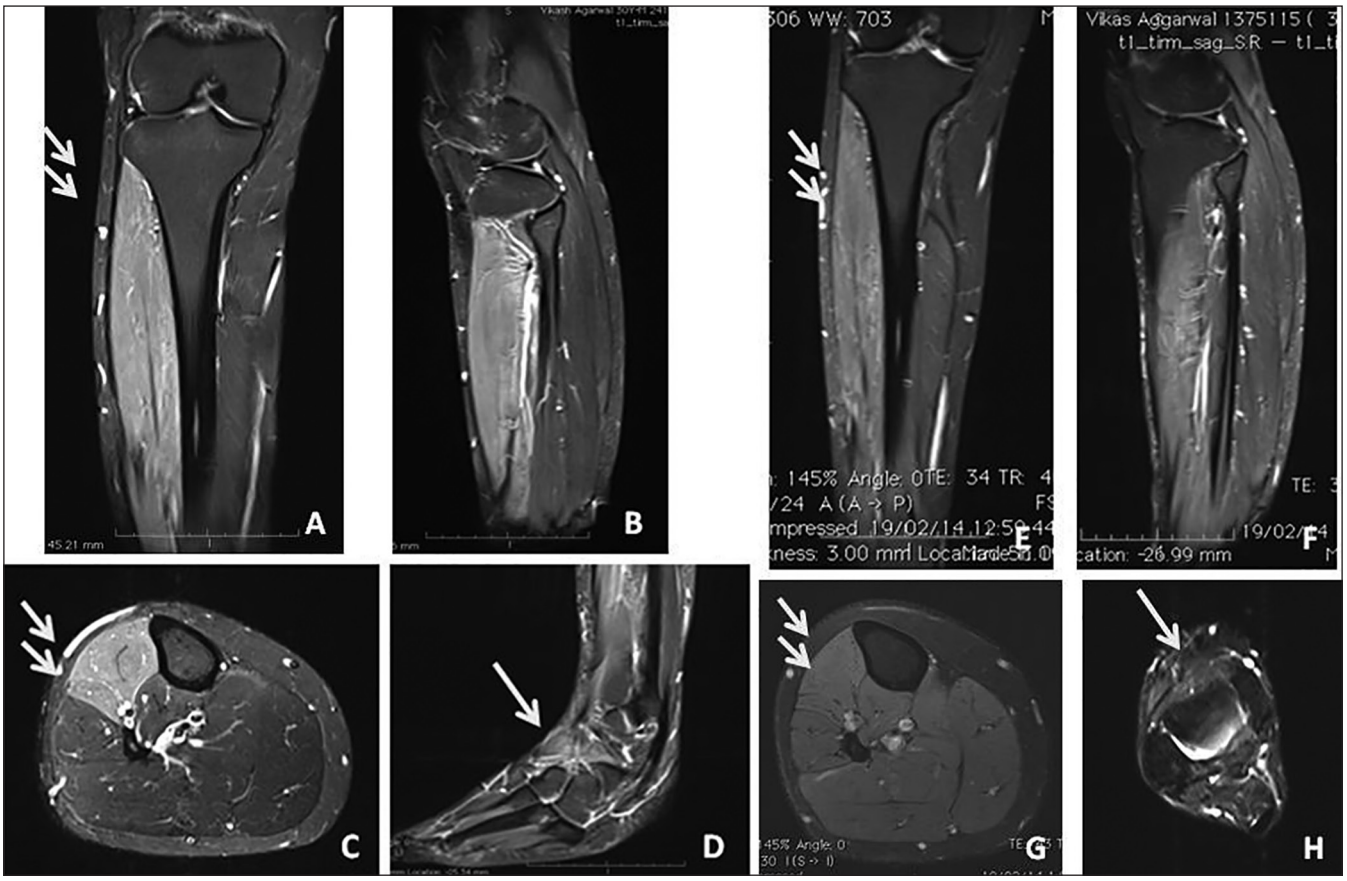

Figure 2: (a-d) MRI of right leg at onset shows diffuse altered hyperintensity in right TA (double arrows) and EDB (single arrow on sagittal FAT-SAT image) with partial effacement of intermuscular fat planes. (a) Coronal FAT-SAT image, (b) sagittal FAT-SAT image, and (c) axial TIRM FAT-SAT image. (e-h) MRI of right leg repeated 4 months later shows significant regression of altered signal intensity in TA (double arrows on (a) coronal FAT-SAT image, (b) sagittal FAT-SAT image, and (c) axial TIRM FAT-SAT image) and EDB (single arrow on axial FAT SAT image). TA = Tibialis anterior, EDB = extensor digitorum brevis, FAT-SAT = fat saturated, TIRM= Turbo inversion recovery magnitude

Table 1: Electromyographic features of right lower limb at presentation and 4 months after treatment

Muscle tested	Spontaneous activity	Motor units	Recruitment
At presentation			
Tibialis anterior	Fibrillations, positive sharp waves	Few nascent potentials	Reduced
Medial gastrocnemius	Nil	Normal	Full
Peroneus longus	Nil	Normal	Full
Extensor digitorum brevis	Fibrillations, positive sharp waves	Normal	Reduced
Short head of biceps femoris	Nil	Normal	Full
After 4 months			
Tibialis anterior	Nil	Normal	Reduced
Medial gastrocnemius	Nil	Normal	Full
Peroneus longus	Nil	Normal	Reduced
Extensor digitorum brevis	Nil	Neurogenic	Reduced
Short head of biceps femoris	Nil	Normal	Full

Discussion

Compression of CPN at fibular neck is the commonest cause of foot drop and DPN lesion is relatively rare.[1] In this patient; clinical, neurophysiological tests, and imaging, which is a recent addition to the evaluation of peripheral nerve lesions, assisted in confirming the diagnosis of DPN palsy and in monitoring the progress.

Common peroneal mononeuropathy is due to a variety of causes including compression at the neck of fibula, trauma, metabolic disorders (diabetes mellitus), infections (leprosy), vasculitis, and immune-mediated disorders.[1,2] On the contrary, isolated deep peroneal neuropathy is rarely encountered, and can be seen after knee surgery, anterior compartment syndrome, and trauma.[3] In the present case, the site of lesion is likely to be in the segment of DPN proximal to origin of branch to TA. Anterior compartment syndrome of leg was excluded since there was no history of severe pain and limb swelling with preservation of dorsalis pedis pulse.[3]

Interestingly, electrophysiological assessment, done as per the American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM) practice parameters[1,4] a month after clinical onset of symptoms, revealed normal nerve conduction with no evidence of conduction block. However, EMG showed significant denervation in muscles innervated by DPN with sparing of superficial peroneal nerve territory. Presence of normal CPN conduction despite significant foot drop and electromyographic changes in CPN innervated muscles may be explained by the fact that NCS was done by recording from EDB, which showed only mild electromyographic changes. Recording from TA, which showed severe involvement on EMG and imaging, might have shown conduction abnormality of CPN.[5]

MRI is being increasingly utilized as an adjunct in evaluation of mononeuropathies.[6] Intrinsic and extrinsic lesions of peripheral nerves and features of denervation of corresponding muscles can be identified.[7] The nerve normally shows low signal intensity within high intensity fat on T1- and T2-weighted sequences.[6] Abnormal bulbous enlargement of nerve is seen at the site of compression, with increased signal on T1- and T2-weighted sequences.[6] MR neurography of CPN has been found to be useful in unexplained footdrop.[8] MRI of normal skeletal muscle shows an intermediate signal intensity (higher than water) on T1-weighted sequence and lower signal intensity on T2-weighted sequence.[7] In acute muscle denervation, usually normal T1- and T2- signal pattern may be retained up to 1 month, however, increased short-tau inversion recovery (STIR) signal intensity may be reported within 4 days[7] and gadolinium enhancement as early as 24 h after denervation.[9] These changes may antedate EMG evidence of denervation. In subacute denervation (1-12 months) increased signal intensity on T2-weighted and STIR images and normal on T1-weighted images have been observed.[10] In chronic denervation with muscle atrophy and fat replacement, MRI shows decreased muscle volume with high signal intensity on T1-weighted sequences.[7,10] Following adequate reinnervation, muscle signal intensity returns to normal.[10]

Although electrophysiologically, the deep peroneal component of the CPN was involved, MRI did not reveal any involvement of the nerve. Therefore, the distribution of the signal changes in the TA and EDB muscles, sparing the lateral and posterior leg compartments, confirmed the involvement of DPN. This case adequately illustrates the usefulness of MRI in localizing the lesion to specific nerve distribution.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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