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Clinical Evaluation and Localisation of Foot drop in an Adolescent Girl with History of Substance Abuse

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Abstract

Mononeuropathy in children and adolescents may be due to post-infectious, infectious, metabolic-endocrine, injuries, pressure from outside or at compression sites. Otherwise it may be idiopathic.

We describe here- in an adolescent girl with history of substance abuse who presented with pressure induced mononeuropathy of distal sciatic nerve about 6 months after her deaddiction and rehabilitation. This followed her being in a friendly gathering at one of her friends' house where she spent 2 hours in cross-legged posture. She had an acute onset left sided foot drop immediately after standing up from the prolonged cross-legged posture with inability to raise her forefoot and numbness over whole leg and foot with inability to stand without support. Electrodiagnostic study of her lower limbs revealed axonal motor and sensory neuropathy of peroneal (fibular) and tibial nerves. She was treated conservatively with physical therapy and avoiding sitting cross-legged posture and a short course of steroid. She showed improvement in 3 wks. Her follow up has been satisfactory so far.

Keywords: Compression neuropathy; Sciatic nerve; Nerve conduction velocity.

INTRODUCTION

Mononeuropathy at or outside the usual compression or entrapment sites are commonly found in clinical practice. Adolescent children with or without history of trauma do suffer from pressure induced mononeuropathy. In addition, mononeuropathy can be caused by post-infectious 'neuritis'. Detailed history of illness, temporal progression, and judicious clinical examination are essential for diagnosis of mononeuropathy. Neurophysiological evaluation with nerve conduction study and electromyography are the cornerstone for localisation and management of such illness.

In this clinical review of we tried to evaluate the localisation of mononeuropathy in the context of an adolescent girl who presented in out patient clinic with acute onset unilateral foot drop.

CASE REPORT

An 18 yr old girl presented with history of sudden weakness and inability to stand on her left foot without support. This started about a week ago when she stood up after sitting for about 2 hrs in a friendly gathering. She was earlier released from a de-addiction center about 6 months ago after recovering from her habit of substance abuse in

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the form of intravenously administered solution of 'brown sugar' mixed with anti allergic medications which she had been taking from her age of 16 yrs. She also had pain in her left hip region without any difficulty in hip or knee movements. She denied having taken any drugs in that gathering or any other time after her de-addiction. She admitted that she sat in the gathering for about 2 hours at a stretch in a cross-legged position with her thighs externally rotated and legs flexed on each other with each foot pressing behind the opposite thigh as in 'asana' posture. She could walk with support on her left side by one of her family members.

On general examination, she was well built with no pallor, jaundice, edema, rash or lymphadenopathy.

Neurological examination revealed normal muscle bulk, tone, power MRC grade 5/5 and 2/2 deep tendon reflexes in both upper and right lower limbs with normal sensory examination.

On her left lower limb, muscle bulk was normal, muscle power was 4/5 proximal (knee flexon) and 3/5 distal with inability to dorsiflex her foot and toe (foot drop) and step page gait. She also had difficulty in foot eversion and inversion.

There was hypotonia and absent ankle jerk (0/2), normal knee jerk (2/2).

Sensory examination of left lower limb showed absent touch, vibration and position sensations on lateral leg and dorsum and plantar aspects foot with decreased sensations over medial foot and leg.

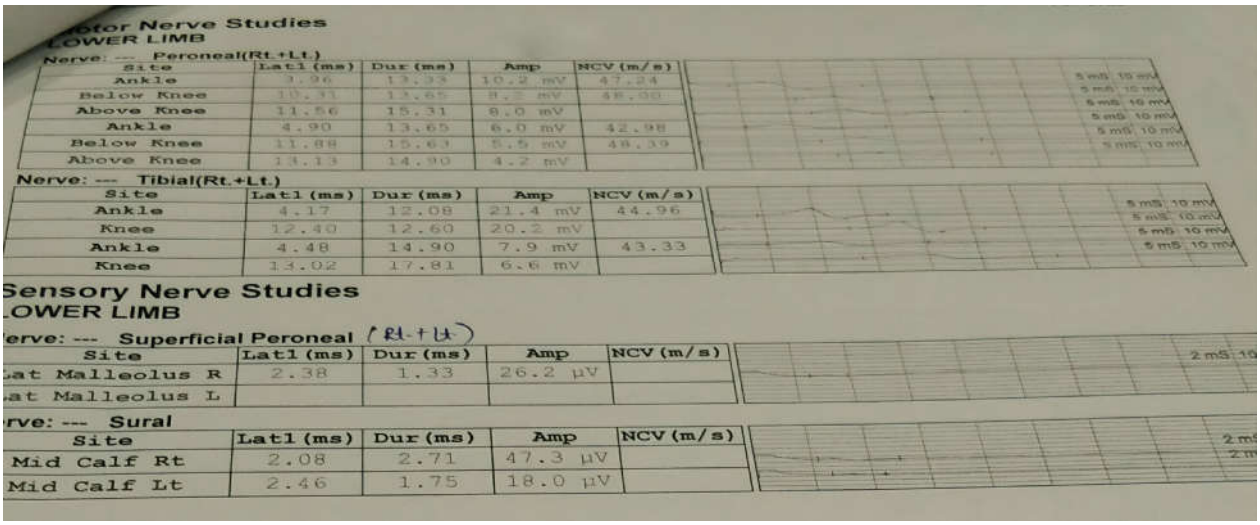
Cerebellar signs were absent bilaterally.

There was no palpable nerve on any probable site.

Her serological test was negative for HIV, HBV and HVC (done earlier during her rehabilitation).

Serum vitamin B12 was within normal range.

Electrophysiology (Nerve Conduction Velocity) (Fig. 1-8) showed:



Motor Nerve Studies
LOWER LIMB

Nerve: --- Peroneal(Rt.+Lt.)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Ankle	3.96	13.23	10.2 mV	47.24
Below Knee	10.11	11.85	8.7 mV	48.00
Above Knee	11.56	15.31	8.0 mV	
Ankle	4.90	13.65	6.0 mV	42.98
Below Knee	11.88	15.61	5.5 mV	48.19
Above Knee	13.13	14.90	4.2 mV	

Nerve: --- Tibial(Rt.+Lt.)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Ankle	4.17	12.08	21.4 mV	44.96
Knee	12.40	12.60	20.2 mV	
Ankle	4.48	14.90	7.9 mV	43.33
Knee	14.02	17.81	6.6 mV	

Sensory Nerve Studies
LOWER LIMB

erve: --- Superficial Peroneal (Rt+Lt)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
at Malleolus R	2.38	1.33	26.2 µV	
at Malleolus L				

rve: --- Sural

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Mid Calf Rt	2.08	2.71	47.3 µV	
Mid Calf Lt	2.46	1.75	18.0 µV	

Fig. 1: Values

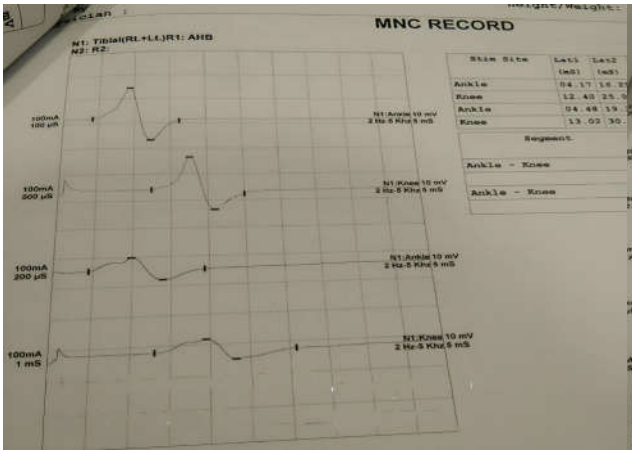


Fig. 2: Lt tibial CMAP amplitude decreased compared to R

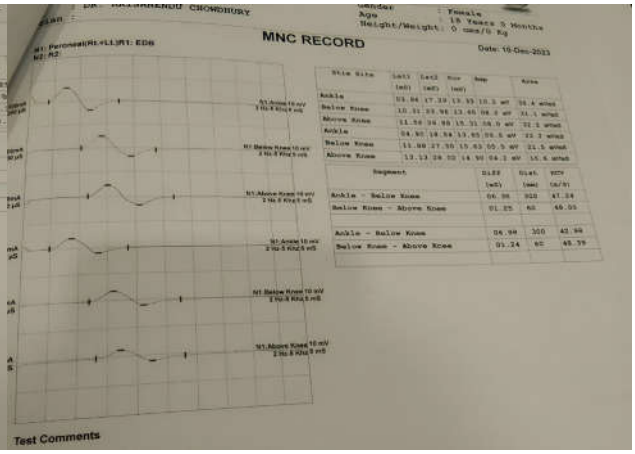


Fig. 3: Lt personal CMAP decreased compared to right

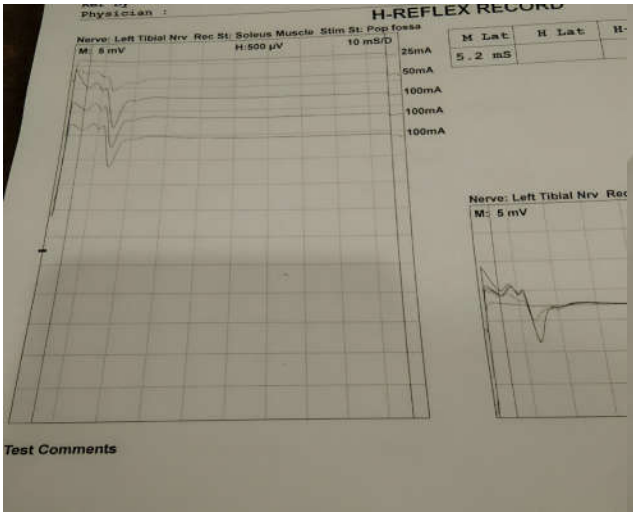


Fig. 4: Lt. H-reflex absent

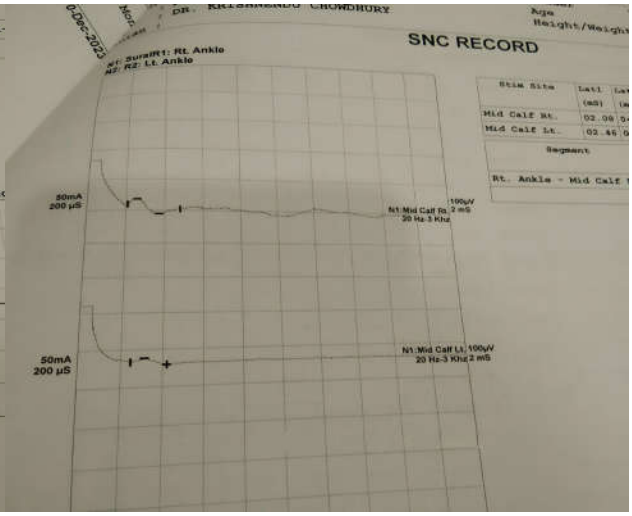


Fig. 5: Lt sup person SNAP absent

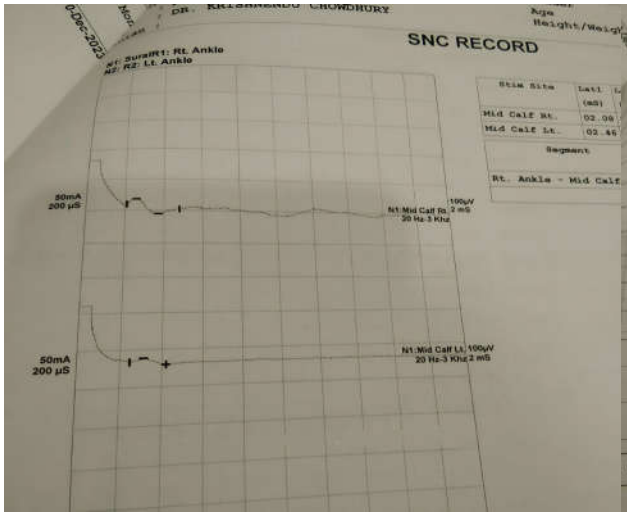


Fig. 6: Lt sural SNAP low

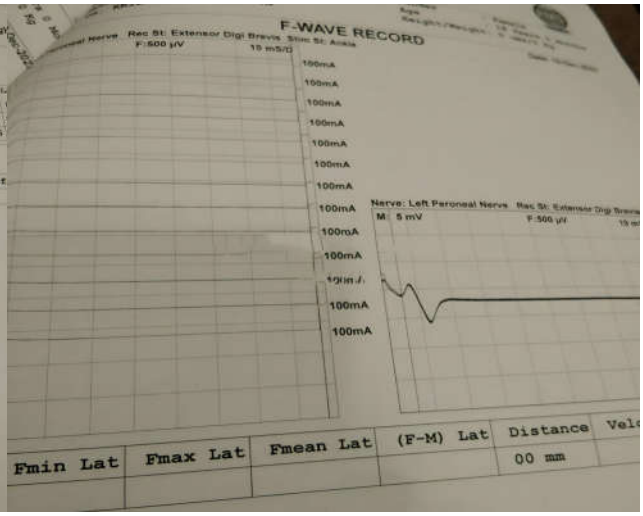


Fig. 7: Lt fibular F - wave absent

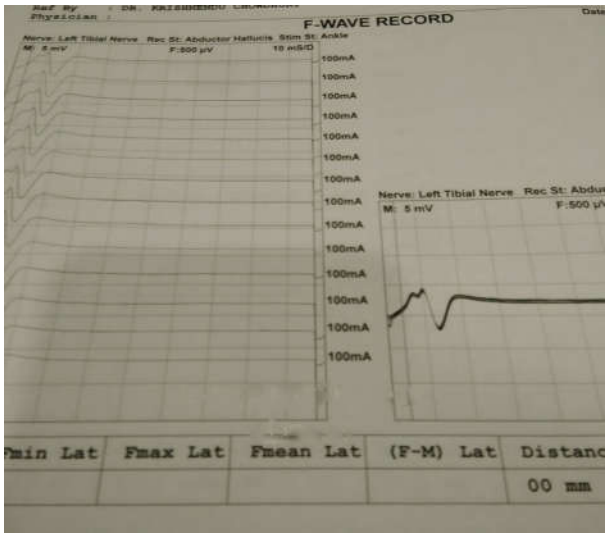


Fig. 8: Left tibial F-wave absent

Motor

Left tibial, peroneal CMAP amplitudes decreased with absent F-waves.

Sensory

Left superficial peroneal SNAP inexcitable.

Left sural SNAP amplitude decreased.

Left H-reflex absent.

So, left sided peroneal and tibial (motor-sensory) axonal neuropathy consistent with distal sciatic neuropathy was postulated.

Electromyography, MRI lumbar spine and genetic testing were denied by patient.

She was advised physical therapy to improve power of her left leg and foot muscles and a short course of oral steroid for 7 days.

She was advised to avoid pressure over the posterior thigh and buttock which would be important to allow healing.

She regained power of her left lower limb and could walk normally without support in next 3 weeks.

Further it was postulated and reassured to the parents that her earlier problem of substance abuse did not have any impact on her present illness.

DISCUSSION

Sciatic neuropathy is the second most common mononeuropathy of the lower extremity and typically presents with foot drop.[1] Nontraumatic pediatric sciatic neuropathies are most commonly due to compressive lesions secondary to long leg and body casts or braces, abnormal lower extremity positioning, and presence of cystic lesions over the popliteal fossa.[1,2]

Prolonged sitting in a position putting pressure on a particular nerve can lead to pressure palsy of the nerve which is usually of small duration, say for hours, but it can continue for days to weeks in susceptible individuals. Patients with genetic cause of such palsy usually presents with repeated episodes starting early or late childhood as seen in PMP 22 gene deletion. This type of hereditary neuropathy with liability to pressure palsy (HNPP) happens to be demyelinating type rather than axonal.[1,2]

In our case this could be linked to her prolonged sitting with compression of her left posterior thigh with her right foot leading to external pressure on distal part of the sciatic nerve including the origin of fibular and tibial nerves. Her left knee flexion was also impaired (4/5), most likely due to simultaneous compression on the nerve to short head of biceps femoris as the twig originates from the common peroneal nerve near its origin.

NCV study of the patient showed axonal type of neuropathy involving the tibial and fibular nerves at knee with changes in their motor as well as sensory components.[2,3] Absence of conduction block with normal latencies and conduction velocities excluded demyelination. Left L-5 radiculopathy (pre-ganglionic lesion) was excluded by impaired sensory potential from the sural and superficial peroneal components.

Rest, physical therapy and avoidance of pressure on the nerve pathway and a short course of steroid empirically could possibly prompted her recovery.[4]

CONCLUSION

Non-traumatic compressive neuropathy can occur on non-entrapment site from external pressure from postural misadjustment. Nerve conduction study is essential to know the site of lesion and type of neuropathy whether axonal or demyelinating. Avoidance of prolonged sitting with cross-legged position is the cornerstone of managing such disorder like distal sciatic neuropathy.

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(No conflict of Interest)

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