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CASE REPORT Open Access

Slimmer's palsy following an intermittent fasting diet

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Abstract

Background: Isolated common peroneal neuropathy (CPN) is the most common lower extremity mononeuropathy and the third most common mononeuropathy overall in adults. It often associated with multiple aetiologies, such as trauma, hereditary neuropathies and iatrogenic causes. We report a rare case of acute bilateral foot drop in a late adolescent female following a rapid weight loss attributable to intermittent fasting diet.

Case presentation: A 19-year-old female presented with worsening bilateral foot drop following a rapid intentional weight loss by intermittent fasting diet. She lost 20% of her baseline bodyweight within a period of 4 months. Systemic disease and metabolic screenings for neuropathy differentials were unremarkable. Electrophysiological studies revealed severe entrapment neuropathy involving bilateral common peroneal nerves (CPN). Her confounding risk factors for CPN injury include contact sport activities and habitual leg crossing during sitting. Following a dietary modification and physical therapy she achieved complete recovery of weakness and function on follow-up.

Conclusions: In a patient with bilateral PN rapid weight loss, prognosis is good and complete neurologic recovery can be attained through combination of cessation of extreme diet and physical therapy.

Keywords: Mononeuropathy, Common peroneal nerve, Nerve conduction studies, Electromyography

Background

Its relatively superficial anatomical course renders CPN to be susceptible to mechanical-related injuries and metabolic disturbances. Acute CPN that develops following rapid and excessive weight loss is rare and scarcely reported [1–4]. Partly due to social media influence to be physically aesthetics, weight reduction diets have been gaining popularity in recent time. Previous case reports [1–4] had described the existence of this condition among patients with a background history of rapid weight loss that classically presented with unilateral CPN. Through our extensive literature reviews, association of weight reduction diet-like intermittent fasting and bilateral CPN is exceptionally rare.

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Case presentation

Nineteen-year-old previously healthy female university student presented to neurology clinic for worsening of bilateral foot drop. The onset was first manifested following a skipping exercise session, whereby she developed sudden right sided foot weakness. Four months earlier, she participated in a strict dietary regimen involving prolonged intermittent fasting with a minimum of 16-h fasting window daily. The food intake consisted of very low carbohydrates and high protein diet that resulted her to lose 20 kg of weight within a period of 4 months. This is equivalent to 20% body weight reduction from her baseline weight of 102 kg with an average of 5-kg weight reduction per month.

On physical assessment, she was relatively tall southeast Asian adult female with a height of 174 cm, weighted at 82 kg with body mass index (BMI) of 27.2. She was noted to have high stepping gait and bilateral foot drop that was more prominent on the right side. There was no obvious

muscle wasting or physical deformities observed. Power assessment revealed marked bilateral ankle dorsiflexion weakness (Medical Research Council's [MRC] scale 2/5) and ankle eversion weakness (MRC scale 2/5). Power assessment over knee and hip joints were normal. Deep tendon reflexes were present, and sensation was intact.

On further questioning to determine possible predisposition factors she admitted involving with frequent squatting during sport activities and as well as having habitual tendency for leg-crossing. She denied having a previous history of physical trauma and was unaware of any inherited neurological disorders that run in her family.

Her metabolic screening and systemic disease screening tests which include fasting blood sugar, thyroid function test, thiamine, folate and vitamin B12 levels were

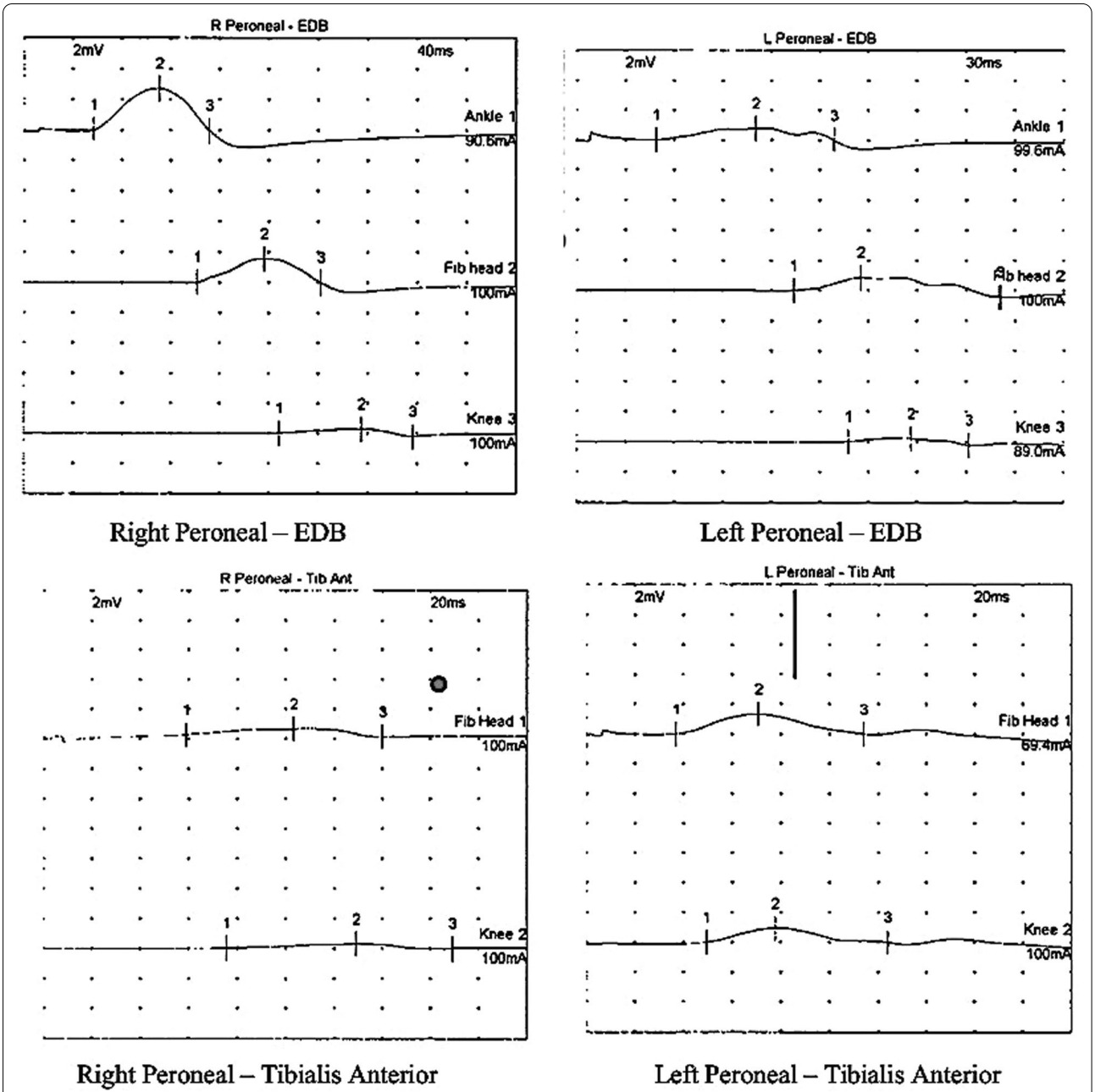

Fig. 1 Graphs of compound motor action potentials (CMAPs) of common peroneal nerves with recordings over extensor digitorum brevis (EDB) and anterior tibialis. Severe reduction in CMAP amplitude seen over bilateral peroneal nerves at the fibular head and knee levels

within normal limits. Her inflammatory markers were not raised, while connective tissue screening which includes ANA, complement levels and anti-double-stranded DNA was negative.

Nerve conduction studies (NCS) with recordings over extensor digitorum brevis and anterior tibialis revealed severe reduction in compound muscle action potential (CMAP) amplitudes (Fig. 1), coupled with moderate to severe (right more than left) slowing of motor conduction velocities (MCV) of peroneal nerves at the fibular head and knee levels (results and normal values are shown in Fig. 2 and Table 1 respectively). Distal stimulations near ankle levels revealed predominantly axonal neuropathic patterns as evidenced by reduced CMAP amplitudes and relative preserved MCV. Sensorimotor nerve studies of upper limbs and tibial nerves otherwise normal. EMG showed denervation changes as evidenced by scanty fibrillation potentials and discrete activated motor unit action potentials (MUAPs) in muscles innervated by CPN, while EMG were normal for other tested muscles.

She was eventually diagnosed with bilateral CPN with electrophysiological evidence of entrapment neuropathy at fibular head levels. She was referred to physiotherapist for physical therapy and dietician for dietary modification. Upon reassessment at 3 months, her bilateral foot drop resolved, and she regained her full lower limb muscle strength.

Discussion

CPN is one of two terminal branches of the sciatic nerve. It passes the anterior compartment of the lower leg through the fibular canal formed by fibres of the peroneus longus muscle. Its superficial course over the fibular head which is only covered by subcutaneous tissue and skin makes the CPN susceptible to surrounding compressive insults.

Isolated bilateral CPN following rapid weight loss is relatively rare with up to 20% can be associated with extreme dieting. Few theories have been offered to explain the link between extreme dieting with weight loss and propensity to develop entrapment CPN. The most likely explanation is that extreme dieting in general promotes calorie deficits that eventually triggers significant loss of subcutaneous tissue [1–5]. This theory supported by a review of multiple case series of patients with CPN and significant weight reduction by which neurophysiological studies in majority of cases revealed conduction block consistent with entrapment neuropathy [3, 6] at the level of fibula head.

Nerve / Sites	Muscle	Latency ms	Amplitude mV	Amp % %	Duration ms	Segments	Distance cm	Lat Diff ms	Velocity m/s
R Peroneal - EDB									
Ankle	EDB	**5.68**	**2.7**	100	9.48	Ankle - EDB	8		
Fib head	EDB	14.17	1.6	57.1	10.05	Fib head - Ankle	27	8.49	32
Knee	EDB	20.83	0.2	9.16	10.83	Knee - Fib head	8	6.67	12
L Peroneal - EDB									
Ankle	EDB	4.90	**0.8**	100	10.99	Ankle - EDB	8		
Fib head	EDB	13.39	0.9	111	12.71	Fib head - Ankle	40	8.49	47
Knee	EDB	16.77	0.2	21	7.40	Knee - Fib head	10	3.39	30
R Tibial - AH									
Ankle	AH	5.31	12.4	100	7.24	Ankle - AH	8		
Pop fossa	AH	14.64	10.1	81.6	7.92	Pop fossa - Ankle	40	9.32	43
L Tibial - AH									
Ankle	AH	8.13	11.5	100	6.82	Ankle - AH	8		
Pop fossa	AH	15.16	10.6	92.2	9.53	Pop fossa - Ankle	40	7.03	57
R Peroneal - Tib Ant									
Fib Head	Tib Ant	5.89	0.4	100	8.13	Fib Head - Tib Ant			
Knee	Tib Ant	7.55	0.3	69.4	9.37	Knee - Fib Head	8	1.67	48
L Peroneal - Tib Ant									
Fib Head	Tib Ant	3.70	1.3	100	7.71	Fib Head - Tib Ant			
Knee	Tib Ant	4.95	1.0	71.7	7.45	Knee - Fib Head	8	1.25	64

Fig. 2 Lower limb motor nerve conduction study results showing bilateral entrapment neuropathy at knee level characterised by severe reduction in CMAP amplitudes and reduced motor conduction velocities (MCVs)

Table 1 Reference range for motor nerve conduction studies of lower limbs

Nerve/sites	Latency ms	Amplitude mV	Distance cm	Velocity m/s
R Peroneal—EDB				
Ankle	< 5.5	> 3		> 40
Fibula head	< 5.5	> 3	28	> 40
Knee	< 5.5	> 3	10	> 40
L Peroneal—EDB				
Ankle	< 5.5	> 3		
Fibula head	< 5.5	> 3	29	> 40
Knee	< 5.5	> 3	9	> 40
R Tibial—AH				
Ankle	< 6	> 4		
Knee	< 6	> 4	35	> 40
L Tibial—AH				
Ankle	< 6	> 4		
Knee	< 6	> 4	36	> 40

Duration of weight reduction diet and the rate of weight loss has been associated with the development of PN [2]. A weight loss that exceeds five kilogram per month and persists for several months following has been the strongest risk factor for the development of CPN. Some authors postulated the pathophysiology may also be explained by metabolic and hormonal disturbances following rapid weight loss rather than just mere mechanical insults [2].

Entrapment neuropathy in this lady likely been exacerbated by her habitual leg-crossing tendency and her contact sport activities as observed in previous case series [7, 8]. After reverting to normal balanced diet and avoidance of further insults, she regained complete recovery of her bilateral foot weakness within 3 months. The prognosis of CPN in the context of extreme weight loss is generally good, with observed recovery rates between 78% and 90% [1, 3]. Poor recovery and persistent limb weakness should trigger other alternative diagnoses such as hereditary neuropathy with liability to pressure palsies (HNPP) [1] that sometimes unmasked by extreme weight loss mechanism. HNPP may be the possible differential if NCS reveals polyneuropathy injuries in the absence of primary systemic disorders.

Conclusions

In a patient with bilateral CPN rapid weight loss, prognosis is good and complete neurologic recovery can be attained through cessation of extreme diet. Given the growing popularity of weight reducing diets in recent time, we anticipate this rare but reversible neurological disorder will increase in prevalence in near future. Therefore, clinicians must be aware of pathophysiologic processes underlying this condition and its favourable outcome if recognized earlier.

Abbreviations

ANA: Anti-nuclear antibody; CMAPs: Cumulative motor action potentials; CPN: Common peroneal nerve; DNA: Deoxyribonucleic acid; EMG: Electromyography; HNPP: Hereditary neuropathy with liability to pressure palsies; MCV: Motor conduction velocity; MRC: Medical Research Council; MUAPs: Motor unit action potentials; NCS: Neuro-conduction study; PN: Peroneal neuropathy.

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Author contributions

JB and AHKYK diagnosed the patient and collected clinical data. JB, AHKYK, AMAR, LWC, and AI carried out the literature search, review, and manuscript preparation. WAWS and LNIM collected data and preparing review on neuroconduction studies. FKH and HB reviewed the manuscript and approved the final draft. All authors read and approved the final manuscript.

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Availability of data and materials

The data sets supporting the conclusion of this article are included within the article.

Declarations

Ethics approval and consent to participate

We confirm that ethical clearance was not required for publication of this case report.

Consent for publication

Written informed consent to publish this information was obtained from study participant.

Competing interests

Authors confirm that they have no competing interests.

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