Case Report

Slimmer's paralysis: a case report

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ABSTRACT

Isolated common peroneal neuropathy (CPN) is the most common lower extremity mononeuropathy and the third most common mononeuropathy in adults. It is often associated with multiple causes including trauma, hereditary neuropathies and iatrogenic causes. We report a case of acute bilateral foot drop in a young male following a rapid weight loss attributable to tirzepatide therapy. This case highlights the importance of monitoring for neurological side effects in patients receiving novel glucagon like peptide-1 plus gastric inhibitory polypeptide receptor agonist.

Key words: *tirzepatide, foot drop, peroneal neuropathy.*

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INTRODUCTION

Common peroneal neuropathy (CPN) is a functionally debilitating mononeuropathy affecting the deep and superficial peroneal nerve branches and is often considered as the commonest mononeuropathy of the lower extremity.[1] Its relatively superficial anatomical course renders common peroneal nerve to be susceptible to mechanical-related injuries and metabolic disturbances. Acute CPN that develops following rapid and excessive weight loss is rare and occasionaly reported.[2-5] Common presenting symptoms of CPN include foot drop due to weakness in ankle dorsiflexion and eversion without affecting inversion of the ankles.[6] Partly due to social media influence to be physically aesthetics, weight reduction medicines and diets have been gaining popularity in recent time.

Tirzepatide, a novel glucagon like peptide-1 and gastric inhibitory polypeptide receptor agonist, is widely used for weight loss and glycemic control in type 2 diabetes mellitus and prediabetes. While common adverse effects are gastrointestinal, emerging evidence suggests a possible link with rare neurological complications. This case explores a possible adverse effect profile in a young male.

CASE REPORT

A 21-year-old previously healthy male university student, known case of psoriasis for three years (managed intermittently with topical corticosteroids and vitamin D analogues), metabolic (dysfunction) associated fatty liver disease (MAFLD), presented with tingling sensation in both lower limbs for two weeks. The onset was first manifested following a skipping exercise session, whereby he developed sudden right sided foot weakness. Six weeks prior to symptom onset, he was initiated on tirzepatide therapy (2.5 mg once weekly for four weeks, then increased to 5 mg once weekly for two weeks) for elevated alanine aminotransferase (85 U/L) and obesity (initial weight 103 kg, body mass index 33.3 kg/m^2). Since treatment initiation, he achieved 8 kg weight loss in one and half months.

On physical assessment, he was relatively tall Bangladeshi adult male with a height of 169 cm, weighted at 95 kg with body mass index of 33.3 kg/m^2. He had 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. Strength in proximal lower extremities were 5/5 but there

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was weakness at the bilateral ankles with dorsiflexion (0/5 on right foot, 1/5 on left foot) and eversion (1/5 on right foot, 2/5 on left foot). Power assessment over knee and hip joints were normal. Deep tendon reflexes were absent and sensory deficit over the dorsum of the right foot and lateral aspect of the leg.

Extensive laboratory testing, including complete blood count, comprehensive metabolic panel (blood glucose,

Motor Nerve Conduction Study

Site	Latency (ms)	Amplitude	Area	Segment	Distance (mm)	Interval (ms)	NCV (m/s)	NC
Median, L								
Wrist	3.14ms	15.94mV	40.89mVms	Wrist		3.14ms		
Elbow	7.94ms	12.51mV	32.47mVms	Wrist - Elbow	245mm	4.80ms	51.0m/s	
Ulnar, L								
Wrist	1.86ms	13.28mV	30.56mVms	Wrist		1.86ms		
Elbow	6.72ms	13.18mV	29.00mVms	Wrist - Elbow	255mm	4.86ms	52.5m/s	
Axilla	7.38ms	14.48mV	26.19mVms	Elbow - Axilla	50mm	0.66ms	75.8m/s	
Median, R								
Wrist	3.08ms	14.69mV	36.88mVms	Wrist		3.08ms		
Elbow	7.4ms	13.21mV	33.32mVms	Wrist - Elbow	245mm	4.32ms	56.7m/s	
Ulnar, R								
Wrist	2.38ms	16.15mV	34.08mVms	Wrist		2.38ms		
Elbow	6.74ms	16.56mV	34.12mVms	Wrist - Elbow	250mm	4.36ms	57.3m/s	
Axilla	7.68ms	16.55mV	32.99mVms	Elbow - Axilla	50mm	0.94ms	53.2m/s	
Tibial, R								
Ankle	4.55ms	15.57mV	25.83mVms	Ankle		4.55ms		
Popliteal	13.75ms	9.90mV	18.18mVms	Ankle - Popliteal	405mm	9.20ms	44.0m/s	
Peroneal, R								
Ankle	6.75ms	140.00uV	46.08uVms	Ankle		6.75ms		
Head of fibula	15.05ms	250.00uV	416.50uVms	Ankle - Head of fibula	330mm	8.30ms	42.2m/s	
Popliteal	15.9ms	90.00uV	147.80uVms	Head of fibula - Popliteal	50mm	0.85ms	58.8m/s	

F-wave Study

Nerve		Stim.Site	F-Lat.	F-Lat. N.D.	M Lat.	F-M Lat.	F-Occurr.	Distance	FWCV
Median	R	Wrist	26.65ms		3.5ms	23.15ms	7/16,44%		
Median	L	Wrist	28.35ms		3.4ms	24.95ms	11/16,69%		
Tibial	R	Ankle	50.3ms		5.2ms	45.1ms	7/16,44%		

Sensory Nerve Conduction Study

Site	Latency (ms)	Amplitude	Area	Segment	Distance (mm)	Interval (ms)	NCV (m/s)	NC
Median, L								
Wrist	2.42ms	37.30uV	2.47uVms	Wrist	135mm	2.42ms	55.8m/s	
Ulnar, L								
Wrist	2.34ms	17.60uV	5.04uVms	Wrist	125mm	2.34ms	53.4m/s	
Median, R								
Wrist	2.4ms	27.10uV	2.10uVms	Wrist	140mm	2.40ms	58.3m/s	
Ulnar, R								
Wrist	2.14ms	19.30uV	1.03uVms	Wrist	125mm	2.14ms	58.4m/s	
Sural, R								
Sural	2.82ms	17.50uV	1.14uVms	Sural	135mm	2.82ms	47.9m/s	

EMG Findings Summary

Muscle Side		Ins. Act	Fibs.	Pos. Wave	Fasc.	MYO. Disch.	Normal MUP	Poly	Low Amp.	High Amp.	Dur	Recruit
Tibialis Anterior	R	Normal	0	0	0	0	0	++	0	0	Normal	Reduce
Gastroc. Lateral H	R	Normal	0	0	0	0	+2	N	0	0	Normal	Full
Quadriceps	R	Normal	0	0	0	0	+3	N	0	0	Normal	Full
1st Dorsal Inter.	R	Normal	0	0	0	0	+2	N	0	0	Normal	Full

Figure 1. Nerve conduction study and electromyography of the patient

sodium, potassium, bicarbonate, chloride, calcium, total protein, albumin, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, bilirubin, creatinine, blood urea nitrogen), thyroid-stimulating hormone, serum protein electrophoresis, vitamin B1 and B6, heavy metals such as arsenic, lead, mercury, treponema pallidium antibodies, connective tissue screening which includes anti cyclic citrullinated peptide antibody, anti nuclear antibody, complement levels and anti-double-stranded DNA was all negative. Vitamin B12 level was 298 pg/ml. HbA1c was 6.2%. Nerve conduction study (NCS) demonstrated peroneal neuropathy (axonal) affecting lower limbs and electromyography showed mild re-innervation changes in lower limbs (Figure 1). Magnetic resonance imaging (MRI) of the lumbosacral spine and cervical spine revealed straightening of the normal lordotic curvature but no disc herniation, spinal cord compression or other abnormalities (Figure 2). Cerebrospinal fluid analysis was normal, excluding infectious, inflammatory or demyelinating causes.

Patient was eventually diagnosed as slimmer's paralysis secondary to tirzepatide-induced rapid weight loss. He is currently under evaluation for surgical release and is being managed with ankle foot orthotic, physical therapy and discontinuation of tirzepatide.

Figure 2. Magnetic resonance imaging of lumbosacral spine of the patient

DISCUSSION

Slimmer's paralysis was a term initially coined by Soteniemi in 1984[3] documenting a case of peroneal nerve neuropathy after rapid weight loss. This effect has continued to be well documented over the years. Most notably, there are multiple studies looking at this effect following bariatric surgery.[7-9] The mechanism seems to be loss of a fat pad that protects the peroneal nerve.[10] With this loss of protection, even leg crossing can cause injury to the nerve.[11-12] Slimmer's paralysis is a classic but infrequent complication of rapid weight loss. In this case, tirzepatide-induced rapid weight loss likely precipitated the condition. Although glucagon like peptide-1 plus gastric inhibitory polypeptide receptor agonists like tirzepatide are not directly neurotoxic, the associated fat pad loss and mechanical vulnerability can trigger entrapment neuropathies. Patients with autoimmune diseases (e.g., psoriasis), metabolic dysfunction and hepatic abnormalities may be at greater risk. Exclusion of other differential diagnoses, timely diagnosis with nerve conduction studies and electromyography, discontinuation of the offending agent and supportive care are essential for recovery.

In conclusion, slimmer's paralysis is not a new phenomenon but its incidence is certain to increase as more effective weight loss medications like tirzepatide become available. It is important for us as providers to be aware of these dangers so we can adjust medication titration, counsel regarding these risks and efficiently manage these complications when they occur. Peroneal nerve neuropathy, resulting in foot drop, is one of these complications that we need to be aware of and try to prevent, but quickly recognize and treat when it occurs.

Authors' contribution: MRH managed the case, did literature review and drafted the manuscript. KSS, AMA and AIA helped in literature review and drafting manuscript. NCK supervised managing the case and revised the manuscript. All authors read the final manuscript and approved it.

Consent: Informed written consent was taken from patient for publication of these case reports and any accompanying images.

Conflict of interest: Nothing to declare.

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