pISSN 1738-6586 / eISSN 2005-5013 / J Clin Neurol 2023;19(2):204-206 / https://doi.org/10.3988/jcn.2022.0315

Polyneuropathy After Rapid and Massive Weight Loss

Rae On Kim
Eun Ji Lee
Moo young Ahn
Hakjae Roh

Department of Neurology, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul, Korea

Dear Editor,

Various types of neuropathies related to weight loss have been reported since the first report of peroneal neuropathy after massive weight loss. Neurological complications after weight loss such as encephalopathy, myelopathy, and polyneuropathy have mostly been studied through bariatric surgery.[1] A recent study found polyneuropathy incidence of about 6%.[2] Reports of polyneuropathy following rapid and massive weight loss regardless of bariatric surgery are extremely rare. We report the two cases of polyneuropathy that developed acutely after rapid and massive weight loss due to dietary restrictions and exercise.

A 23-year-old male (Case 1) with weakness and paresthesia in both feet and a 21-year-old male (Case 2) with weakness in both lower extremities visited the hospital. Both had developed acute symptoms without progression. Before symptom onset, Case 1 consumed a small amount of porridge twice daily and exercised, and Case 2 cut his daily meals in half and exercised. The bodyweight of Case 1 reduced from 110 kg to 68 kg in 3 weeks, while that of Case 2 reduced from 92 kg to 78 kg in 4 weeks (Table 1). They had no medical, alcohol abuse, or family history of neuropathy. A neurological examination revealed that Case 1 had Medical Research Council (MRC) scale grade 4 for left plantar flexion with hypesthesia below the knees, while Case 2 had MRC grade 4 in both dorsiflexion and plantar flexion without sensory deficits. Motor-dominant axonal polyneuropathy affecting the bilateral peroneal and tibial nerves was detected in nerve conduction studies of both cases (Table 1). Extensive laboratory tests produced normal findings except for a reduced folate level (3.60 ng/mL) in Case 1 and elevated aldolase (10.1 U/L) in Case 2. Lumbosacral and brain MRI produced normal findings in both cases. Both patients received rehabilitation therapy and multivitamin supplements that contained folic acid, thiamine, and cyanocobalamin. The nutritionist also provided advice on a better dietary plan for weight loss. After about 1 year, nerve conduction study results had normalized, and the weakness and sensory symptoms had disappeared.

Polyneuropathy following weight loss has been documented much less than peroneal neuropathy. Both polyneuropathy and peroneal neuropathy are mostly attributed to nutritional deficiencies following bariatric surgery[2] or anorexia nervosa.[3] However, our cases were distinctive as they did not present these predisposing factors.

We first clinically considered classic Guillain-Barré syndrome (GBS) or its variants based on the acute onset of weakness and areflexia. However, it was differentiated by the lack of clear preceding infectious illness, non progressive weakness, negativity for antiganglioside antibodies, and normal CSF findings. There was therefore insufficient evidence to diagnose GBS, causing us to consider other diagnoses.

Both patients lost weight over a short period of time through only dietary restrictions and exercise. Besides malnutrition, lower post surgery body mass index (BMI) and rapid weight loss are also risk factors for neuropathy after bariatric surgery.[2,4] Rapid weight loss is defined as a reduction of body weight at least 5% over 5 weeks,[5] and massive weight loss is defined as a reduction of at least 50% excess weight (actual body weight minus ideal body weight),[6] and so it was reasonable to consider that our cases had experienced rapid and

Received August 24, 2022
Revised November 3, 2022
Accepted November 10, 2022

Correspondence
Hakjae Roh, MD, PhD
Department of Neurology, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, 59 Daesagwan-ro, Yongsan-gu, Seoul 04401, Korea
Tel +82-2-709-9224
Fax +82-2-795-3687
E-mail newroh@schmc.ac.kr

Table 1. Clinical characteristics of the two patients

	Case 1				Case 2				Reference range
Age, years	23				21				
Height, cm	166.7				182.4				
IBW, kg/m^2	62.9				77.2				
WL									
	Pre-WL		Post-WL		Pre-WL		Post-WL		
BW, kg	110		68		90		78		
EBW, kg	47.1		5.1		12.8		0.8		
BMI, BW/height in kg/m^2	39.6		24.5		27.1		23.4		
CSF									
RBC	0				0				0
WBC	0				0				0
Protein, mg/dL	33				32				15–45
Glucose (CSF/blood), mg/dL	62/122				59/111				40–70
LDH, mmol/L	13				22				115–125
Nutrients									
Thiamine, nmol/L	-				341.7				66.1–200.6
Folate, ng/mL	3.60				7.71				3.89–26.8
Vitamin B12, pg/mL	489				482				232–1,245
Aldolase, U/L	-				10.1				0–7.6
Nerve conduction study									
	Initial		1 year		Initial		1 year		
Nerve/sites	Right	Left	Right	Left	Right	Left	Right	Left	
Peroneal nerve									
CMAP, mV	3.5	3.2	5.2	4.9	4.0	2.0	7.2	6.4	≥4
CV, m/s	44.1	41.1	45.3	44.8	48	44	48.5	46.2	≥42
Tibial nerve									
CMAP, mV	4.3	2.9	8.1	7.7	5.5	1.1	7.1	6.8	≥5
CV, m/s	40.3	40.5	48	49	47	41	49	47	≥41
Superficial peroneal nerve									
SNAP, μV	13.7	14.1	17	14	11	12	13	16	≥6
CV, m/s	46.8	45.7	47.1	46.4	39	39	45	46	≥40
Sural nerve									
SNAP, μV	15	13.4	22	21	15	9	19	14	≥6
CV, m/s	43.6	50.5	42.2	47.8	41	40	42	43	≥35

BMI, body mass index; BW, body weight; CMAP, compound muscle action potential; CSF, cerebrospinal fluid; CV, conduction velocity; EBW, excessive body weight (actual BW-IBW); IBW, ideal body weight (50+0.91×[height in centimeters-152.4]); LDH, lactate dehydrogenase; RBC, red blood cells; SNAP, sensory nerve action potential; WBC, white blood cells; WL, weight loss.

massive weight loss. Since the BMIs after weight loss and serum nutrient levels of our patients were normal, we inferred that rapid weight loss might have contributed to the development of neuropathy. Axonal polyneuropathy in our cases also suggested that metabolic disturbances were predominantly involved.

The most-deficient nutrients after weight loss following bariatric surgery are thiamine, cyanocobalamin vitamins E, and D, and copper.[7] No such deficiencies were observed in our cases, but a reduced folate level was observed in Case 1. Folate deficiency can cause slowly progressive, length-dependent, sensory-dominant subacute polyneuropathy, although acute-onset cases have also been reported.[8] The folate deficiency in Case 1 might have contributed to the neuropathy.

Nonalcoholic thiamine deficiency can manifest length-dependent and motor-dominant polyneuropathy.[9] In Case 2, thiamine deficiency was clinically suspected but a normal thiamine level and elevated aldolase were detected. Since aldolase increases in thiamine deficiency, an increased serum aldolase level and motor-dominant axonal polyneuropathy indicate the possibility of thiamine deficiency at the cellular level or decreased thiamine activity.[10]

While the exact pathogenesis of polyneuropathy after weight loss has not yet been clarified, nutritional deficiency may induce the immune mechanisms of neuropathy,[2] and multiple factors including nutritional and metabolic disturbances might be involved. The two cases reported here warn that going on an extreme diet can cause neuropathy, and it is important to avoid losing weight too quickly and severely, while maintaining a nutritional balance.

Ethics Statement

This study was approved by the Institutional Review Board (IRB No. 2022-10-008).

Availability of Data and Material

All data generated or analyzed during the study are included in this published article.

ORCID iDs

Rae On Kim https://orcid.org/0000-0002-7859-6209
Eun Ji Lee https://orcid.org/0000-0001-6690-5783
Moo young Ahn https://orcid.org/0000-0003-0329-5855
Hakjae Roh https://orcid.org/0000-0002-0979-4279

Author Contributions

Conceptualization: Rae On Kim, Hakjae Roh. Data curation: Rae On Kim, Hakjae Roh. Formal analysis: Hakjae Roh, Eun Ji Lee, Moo young Ahn. Investigation: Rae On Kim. Methodology: Hakjae Roh. Project administration: Hakjae Roh. Resources: Hakjae Roh. Supervision: Hakjae Roh, Moo young Ahn. Writing—original draft: Rae On Kim, Hakjae Roh, Moo young Ahn. Writing—review & editing: all authors.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Funding Statement

None

REFERENCES

1. Kumar N. Neurologic complications of bariatric surgery. *Continuum (Minneap Minn)* 2014;20:580-597.
2. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. *Neurology* 2004;63:1462-1470.
3. Teixeira AL, Junho BT, Barros JL, Gomez RS. Anorexia nervosa presenting as a subacute sensory-motor axonal polyneuropathy. *Braz J Psychiatry* 2016;38:180.
4. Weyns FJ, Beckers F, Vanormelingen L, Vandersteen M, Niville E. Foot drop as a complication of weight loss after bariatric surgery: is it preventable? *Obes Surg* 2007;17:1209-1212.
5. Ashtary-Larky D, Ghanavati M, Lamuchi-Deli N, Payami SA, Alavi-Rad S, Boustaninejad M, et al. Rapid weight loss vs. slow weight loss: which is more effective on body composition and metabolic risk factors? *Int J Endocrinol Metab* 2017;15:e13249.
6. Shermak MA, Chang D, Magnuson TH, Schweitzer MA. An outcomes analysis of patients undergoing body contouring surgery after massive weight loss. *Plast Reconstr Surg* 2006;118:1026-1031.
7. Becker DA, Balcer LJ, Galetta SL. The neurological complications of nutritional deficiency following bariatric surgery. *J Obes* 2012;2012:608534.
8. Hammond N, Wang Y, Dimachkie MM, Barohn RJ. Nutritional neuropathies. *Neurol Clin* 2013;31:477-489.
9. Koike H, Iijima M, Sugiura M, Mori K, Hattori N, Ito H, et al. Alcoholic neuropathy is clinicopathologically distinct from thiamine-deficiency neuropathy. *Ann Neurol* 2003;54:19-29.
10. Dhir S, Tarasenko M, Napoli E, Giulivi C. Neurological, psychiatric, and biochemical aspects of thiamine deficiency in children and adults. *Front Psychiatry* 2019;10:207.