

Short report

Slimmer's paralysis—peroneal neuropathy during weight reduction

KA SOTANIEMI

From the Department of Neurology, University of Oulu, Oulu, Finland

SUMMARY Ten patients developed peripheral neuropathy while on a reducing diet. One of the patients sustained a severe polyneuropathy attributable to thiamine deficiency. Nine developed unilateral peroneal paralysis. Electromyography revealed bilateral abnormalities in three of these patients. The neuropathy could not be attributed to any factor other than weight reduction. In contrast to previous reports suggesting a compressive aetiology, the present observations indicate that metabolic disturbances are the cause of the disorder.

Weight reducing diets introduced and practised for medical requirements, as well as for the timely trends of high-fashion and body-building, have become very popular in recent years. Both prolonged total starvation¹ and supplemented fasting,² in which a protein supplement is employed, have been prescribed as a treatment of obesity. In addition, people construct dietary regimens by themselves and may even follow extremely radical reducing diets without medical surveillance. Although supplemented fasting is now recognised as an effective means of achieving substantial weight loss safely with a minimum of patient discomfort,² slimming procedures may also cause several problems, including complaints such as headache, nausea, dizziness, fatigability and muscle cramps,^{2,3} and a number of metabolic disturbances.^{3,4} Unfortunately, weight reduction also is occasionally followed by neurological complications,^{5–8} which may sometimes mar an otherwise successful outcome of therapeutic or cosmetic slimming. In particular, rapid and self-planned weight reduction may be disastrous.⁹ We draw attention to the features associated with the occasional neurological consequences of dieting. This report presents ten cases, nine of whom developed peroneal paralysis and one sustained a severe generalized polyneuropathy during a self-planned weight reducing diet.

Address for reprint requests: Dr KA Sotaniemi, MD, Dept of Neurology, University of Oulu, Oulu, Finland.

Received 1 January 1982 and in final revised form 20 January 1984

Case reports

The cases are presented in the table. All the patients underwent thorough clinical neurological examinations, radiology of the spine and of the lower extremities, and extensive laboratory investigations to exclude known causes of peripheral neuropathy and local or systemic diseases. The laboratory investigations (erythrocyte sedimentation rate, blood cell count, glucose tolerance test, liver enzymes, thyroid hormone, uric acid, electrolytes, serum total proteins, serum albumin, serum creatine, urinalysis, B₁₂ vitamin, serum folate, collagen disease screening performed in all the cases and cerebrospinal fluid analysis performed in most of them) were normal. No other abnormalities except those given in the table were encountered. None of the patients showed muco-cutaneous signs attributable to nutritional deficiency. All the patients had been neurologically asymptomatic before the onset of their symptoms. None of them had taken any medication for several months prior to the weight reduction period and none used slimming pills. Two patients (Cases 1 and 2) had used multivitamins during the diet. Only one of the slimmers (Case 1) increased his physical activities, starting jogging and weight-lifting while dieting. None reported excessive use of alcohol prior to the weight reduction period, and during the period alcohol was particularly avoided because of its supposed appetising effect.

The slimming regimen was self-planned in all of the cases, not being under doctor's surveillance. The diets were composed either of vegetables alone or of a reduced amount of usual food with as little fat and carbohydrate as possible. Case 4 had an extreme diet: she took only one fruit (apple, tomato, orange etc.) a day. This strict diet was followed by thiamine deficiency resulting in polyneuropathy which made the patient bed-ridden for two weeks and caused marked disability for half a year.

Table Details of cases

Case identification M = man F = woman	Previous weight kg	Weight reduction				Neurological findings	Recovery
		Weight lost by the time when complications appeared	Months of dieting before the appearance of neurological symptoms	Daily diet	Multivitamin substitution use		
1. M, 28 years	86	16	3	vegetables only	yes	R peroneal paralysis	complete, after 6 months
2. M, 35 years	96	14	2,5	vegetables only	yes	R peroneal paralysis	essential in 4 months, slight resid. symptoms
3. M, 50 years	104	20	3	fat-free meat, one piece of bread		L peroneal paralysis	complete in 2 months
4. F, 16 years	104	44	4	one small fruit	no	severe generalised polyneuropathy	disabled for 6 months, permanent peroneal weakness
5. M, 34 years	95	17	3	reduced amount of usual food	no	R peroneal paralysis	complete in 2 months
6. M, 32 years	101	25	4	reduced amount of usual food	no	R peroneal paralysis	complete in 3 months
7. M, 39 years	92	16	4	reduced amount of usual food	no	R peroneal paralysis	persistent peroneal weakness
8. F, 18 years	54	10	2	tea and fruits only	no	L peroneal paralysis	complete in 3 months
9. F, 32 years	74	12	3	vegetables, fruits	no	R peroneal paralysis	persistent peroneal weakness
10. M, 48 years	86	16	4	reduced amount of usual food	no	L peroneal paralysis	complete in 3 months

Her serum thiamine concentration was 0.006 µg/ml (normal values over 0.01 µg/ml). This case has been described in detail earlier.⁹

Peroneal paralysis developed gradually over a few days in all the cases. Two patients (Cases 3 and 7) had only motor signs while the others also showed sensory peroneal abnormalities.

Routine electroneuromyography (ENMG) showed signs of denervation in the muscles innervated by the clinically affected peroneal nerve but, in addition, three patients displayed denervation activity also on the clinically asymptomatic side. In Case 4, the ENMG revealed polyneuropathy due to axonal degeneration, peroneal abnormalities being the most severe. Two years after the acute phase there was still marked peroneal neuropathy but no other ENMG findings were present.

The mean motor conduction velocity of the clinically affected peroneal nerve was 46.1 m/s (range 39.5–50.8 m/s), the mean distal latency was 5.0 ms (range 4.2–5.3 ms). The respective values for the clinically non-affected peroneal nerves were 51.1 m/s (range 44.5–54.0 m/s) and 4.3 ms (range 3.6–5.0 ms).

Intensive physiotherapy including electrical stimulation of the peroneal nerve was prescribed as treatment in most of the cases. No other specific therapy was practised except intensive vitamin B treatment in Case 4. Six patients recovered completely in 2 to 6 months, the remaining ones having slight residual peroneal weakness.

Discussion

Peroneal paralysis is known to be associated with a

number of conditions and illnesses (nerve compression, alcoholism, chronic infection, thyreotoxicosis, paraneoplastic conditions, vitamin B depletion, haemorrhagic diathesis).^{6,10} However, these and other known causes of neuropathic manifestations could be ruled out in the present patients (except Case 4). Follow-up for at least one year in all the cases has not revealed any additional or relapsing symptoms. The ENMG also failed to reveal peroneal nerve entrapment which is commonly seen in, for instance, peroneal nerve compression at the level of the knee. Polyneuropathy could not be found, except in Case 4, the only case with an apparent cause of neurological signs. In this patient the complications were attributable to a verified thiamine deficiency due to prolonged inadequate nutrition. Although thiamine determinations were not performed in the other patients, the nature of their diets made thiamine deficiency less likely. Moreover, thiamine stores built up with normal nutrition are considered to be sufficient for several months.⁶ However, it is noteworthy that the clinical signs of thiamine deficiency may be delayed in starvation when the need of the vitamin is minimal due to low carbohydrate intake.¹¹ Signs may appear rapidly when carbohydrate is given after a period of malnutrition,¹² as occurred in the present Case 4 which sustained polyneuropathy a few days after turning to a high carbohydrate diet after having

fasted for 4 months.

Compressive mononeuropathy is one of the most common causes of peroneal paralysis. It has been emphasised that susceptibility to mechanical irritation at the proximal head of the fibula increases after the amount of fat around the peroneal nerve has decreased due to weight loss.^{5 10 13} Habitual excessive leg-crossing has also been claimed^{5 10} to favour the development of pressure neuropathy in inactive persons on reducing diets. In the present patients, however, this kind of involvement seemed unlikely. Although we paid particular attention to the patients' sitting and lying postures, none of them reported any conditions supporting a compressive aetiology. Nor could any postural or toxic occupational correlates be established. Furthermore, the patients were young (mean age 31 years), active and healthy without any factors predisposing them to neurological manifestations. Local aetiology with mechanical nerve irritation cannot be absolutely excluded in the patient who increased his physical activities but even he did not show any localisable clinical or ENMG findings.

Both the rate and the prolongation of the reducing diet seem to be involved in the occurrence of neuropathy, since in the present as well as in the previously reported⁷ cases the signs appeared only after at least two or three months' dieting. In our patients, the rate of weight loss exceeded 5 kg per month and dieting lasted for at least two months.

From the arguments and observations discussed above it is concluded that peroneal paralysis appearing in association with weight reduction by dieting is unlikely to be attributable to mechanical causes alone, such as excessive leg-crossing or the related compressive factors previously suggested. It seems more probable that neuropathy is due to factors of metabolic origin or to metabolic, mechanical or microcirculatory disturbances operating alone, simultaneously or successively. There are, indeed, a number of reports on obesity and weight reduction which mention disturbances in, for example, lipoproteins,¹⁴ catecholamines,¹⁵ hormonal activity¹⁶ and electrolytes,^{3 17} and all of these disturbances could both be associated with and contribute to nerve dysfunction. The fact that the harmful manifestations appear first in the peroneal nerve shows the predisposing nature of the anatomical conditions. However, the elucidation of the pathogenesis requires more knowledge of the metabolic conditions both in obesity and in weight reduction.

To conclude, the clear risk of peripheral neuropathy should always be remembered both in

therapeutic fasting and when counselling about a slimming regimen. Special care should be taken when the weight loss exceeds 5 kg a month and is prolonged over several months. Furthermore, slimming persons known to have conditions predisposing them to neurological disturbances should be kept in particular surveillance. And finally, it is worth noting that the use of multivitamins is not necessarily effective in preventing slimmer's peroneal paralysis.

References

- Drenick EJ, Swendseid ME, Bland WH, Tuttle SG. Prolonged starvation as treatment for severe obesity. *JAMA* 1964;**187**:100-5.
- Vertes V, Genuth SM, Hazelton IM. Supplemented fasting as a large-scale outpatient program. *JAMA* 1977;**238**:2151-3.
- Lawlor T, Wells DG. Metabolic hazards of fasting. *Am J Clin Nutr* 1969;**22**:1142-9.
- Editorial. Fasting and obesity. *Lancet* 1977;*i*:673.
- Sproffkin BE. Peroneal paralysis—a hazard of weight reduction. *Arch Int Med* 1958;**102**:82-7.
- Victor M. Polyneuropathy due to nutritional deficiency and alcoholism. In: Dyck PJ, Thomas PK, Lambert EH, eds. *Peripheral Neuropathy*. Philadelphia: W B Saunders 1975: 1030-66.
- Sherman DG, Easton JD. Dieting and peroneal nerve palsy. *JAMA* 1977;**238**:230-1.
- Scobie IN, Durward WF, MacCuish AC. Proximal myopathy after prolonged total therapeutic starvation. *Br Med J* 1980;**280**:1212-3.
- Sotaniemi KA, Kaarela K. Dry beriberi in a slimmer. *Br Med J* 1977;**2**:1634-5.
- Marwak V. Compression of the lateral popliteal (common peroneal) nerve. *Lancet* 1964;**2**:1367-9.
- Farmer TW. Vitamin B₁₂ deficiency. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Amsterdam: North Holland Publishing Company 1976:49-57.
- Sibley WA. Polyneuritis. *Med Clin N Amer* 1972;**56**:1299-319.
- Nagler SH, Rangell L. Peroneal palsy caused by crossing the legs. *JAMA* 1947;**133**:755-61.
- Streja DA, Boyko E, Rabkin SW. Changes in plasma high-density lipoprotein cholesterol concentration after weight reduction in grossly obese subjects. *Br Med J* 1980;**281**:770-2.
- Jung RT, Shetty PS, Barrand M, Callingham BA, James WPT. Role of catecholamines in hypotensive response to dieting. *Br Med J* 1979;**1**:12-3.
- Kopelman PG, Pilkington TRE, White J, Jeffcoate SL. Impaired hypothalamic control of prolactin secretion in human obesity. *Lancet* 1979;**1**:747-9.
- De Luise M, Blackburn GL, Flier JS. Reduced activity of the red-cell sodium-potassium pump in human obesity. *N Engl J Med* 1980;**303**:1017-22.