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Letter to the Editor

Weight loss per se does not cause polyneuropathy but accompanying metabolic changes do

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We read with interest the article by Kim *et al*. on two patients (patient-1, 23yo male, patient-2, 21yo male) who were diagnosed with axonal polyneuropathy due to rapid and massive weight loss (patient-1 42kg in 3 weeks, patient-2 14kg in 4 weeks) [1]. A detailed examination did not clarify the cause of the polyneuropathy, which is why it was attributed to the voluntary weight loss [1]. Clinical and electrophysiologic abnormalities completely disappeared in both patients after one year [1]. The study is compelling but has limitations that should be discussed.

We disagree with the statement that the polyneuropathy in the two index patients was solely due to weight loss [1]. Weight loss in itself does not cause polyneuropathy. Weight loss may uncover a pre-existing polyneuropathy for example in patients with subclinical hereditary neuropathy with liability to pressure palsies (HNPP) when peripheral nerves become more vulnerable due to the reduction of protecting fat and muscle.

However, weight loss, when achieved through starvation, can be complicated by malnutrition particularly vitamin deficiencies, which can secondarily cause polyneuropathies [2]. Thus, a possible cause of the polyneuropathy in the two index patients is malnutrition, as already suspected by the authors. However, an argument against malnutrition is that patient-2 reduced food intake by only 50% for 4 weeks, which many people world-wide do to lose weight but without developing polyneuropathy. Patient-2 did not alter the composition of the diet, suggesting that the lack of essential dietary components may have not been so severe as to constitute malnutrition. Patient-1 ate porridge twice a day, suggesting at least no B-vitamin deficiency. Porridge consists of grains cooked in liquid or other ingredients with a viscous to semi-solid consistency.

It is also conceivable that the polyneuropathy existed before starvation began, but was subclinical and thus independent of hunger and malnutrition. We should therefore know if there were any symptoms of polyneuropathy before the two patients began to starve. There are many people who starve but do not develop polyneuropathy.

We should also be informed about the dietary habits of the two patients before they began to starve. Did they eat junk food? Is it conceivable that malnutrition was already present before the onset of polyneuropathy and starvation? Even if you are overweight, you may still be malnoutrished.

Since polyneuropathy is often multi-causal, it is also conceivable that not just a single cause, but several different causes have contributed to the development of the polyneuropathy. In addition to vitamin deficiency, intoxication, paraneoplasia, neoplasia, immunological disease, and hereditary causes should be thoroughly ruled out.

A limitation of the study is that thiamine was not measured in patient-1. Because thiamine deficiency can be complicated by polyneuropathy, it is important to know whether or not patient-1 was thiamine- deficient. Did patient-1 present with symptoms of Wernicke encephalopathy, manifested by the triad of gait ataxia, confusion, and visual disturbances?

Another limitation of the study is that cerebrospinal fluid (CSF) studies were limited. Not reported were CSF immunoglobulines, oligoclonal bands, the virus panel, including SARS-CoV-2, and CSF lactate. In the case of polyneuropathy of unknown etiology, the exclusion of Borreliosis, a viral infection (virus panel), an immunological disease, and vasculitis is imperative. If CSF tests are non-informative, a hereditary cause should be considered and panel investigations should have been performed to exclude the most common causes of hereditary neuropathy. Since a normal CSF protein does not rule out Guillain-Barre syndrome (GBS), it would be interesting to know whether F-wave parameters were within normal range or were abnormal, suggesting proximal neuropathy.

Overall, the interesting study has limitations that put the results and their interpretation into perspective. Addressing these issues would strengthen the conclusions and could improve the status of the study. Before attributing polyneuropathy to weight loss, various alternative causes need to be thoroughly ruled out.

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