Thiamine deficiency is a neurologic emergency that requires prompt diagnosis and treatment. Typical presentations include patients with alcoholism or history of gastric bypass; however, poor nutritional status due to reduced intake, decreased absorption, increased metabolic requirements, or increased losses of defective transport can also lead to thiamine deficiency [1].

We present a case of thiamine deficiency causing both Wernicke’s encephalopathy, and dry beriberi in the setting of rapid intentional weight loss while on a tomato soup diet. The MRI findings demonstrated in this patient are objective proof that if thiamine deficiency is diagnosed and treated promptly, abnormal brain findings can be reversible.

A 24-year-old woman presented with confusion, lateral gaze-provoked nystagmus, rapidly progressive weakness, and hyperalgesic pain in her arms and legs. Her condition developed over 6 months and coincided with intentional weight loss of 30 kg, which she accomplished by drinking tomato soup as her only source of calories. On exam, she had lateral gaze-provoked nystagmus, distal weakness with hyperalgesic pain of her extremities, and absent reflexes. She was unable to stand or ambulate. Magnetic resonance imaging of the (MRI) brain showed hyper-intense T2 FLAIR changes in the bilateral thalami and periventricular white matter (Figure 1A). Demyelinating disease was considered so a lumbar puncture was performed. Cerebrospinal fluid had no signs of inflammation. Electrodiagnostic testing was consistent with an acute axonal sensorimotor polyneuropathy in combination with a myopathy.

The presentation, MRI, and electrodiagnostic testing were concerning for Wernicke’s encephalopathy and dry beriberi, and serum testing confirmed a low thiamine level of 60 nmol/L (reference range 78 nmol/L to 185 nmol/L) with no evidence of other nutritional deficiencies. Treatment was initiated with high-dose intravenous thiamine (500 mg daily) prior to receiving the thiamine level. Repeat MRI was completed 5 days after treatment initiation, and revealed substantial improvement of the T2 hyper-intensities in the bilateral thalami (Figure 1B). The patient was discharged to post-acute inpatient rehab. Eleven weeks later, she regained the ability to walk, nystagmus resolved, and mental status returned to her baseline.

The atypical presentation in this case stresses the consideration of nutritional deficiencies when there are neurological findings and a history of profound weight loss. The rapid improvement of the MRI FLAIR changes emphasizes the importance of prompt treatment.

Reference