Letters to the Editor

Reversible Dry Beri-Beri Postintragastric Botulinum Toxin Injection for Weight Reduction

Dear Sir,

Post bariatric surgery or gastrectomy dry Beri-Beri mimicking Guillain-Barre syndrome, secondary to thiamine deficiency contributed by concomitant risk factors such as vomiting, weight loss, anorexia and alcohol consumption is well documented in the literature.^[1-3] With a recommended dietary intake of 1.1–1.4 mg/day, thiamine stores can be completely depleted from the body within two weeks of initiation of a thiaminedeficient diet. Thiamine pyrophosphate, the active form of thiamine plays a vital role in Krebs's cycle, in turn being essential for the utilization of energy substrates maintaining neuromuscular transmission.^[4] Hence, thiamine deficiency contributes to neuronal loss and neuromuscular dysfunction.^[4] Intragastric botulinum toxin injection is a functional bariatric procedure proven effective for weight reduction by delaying gastric emptying and inducing early satiety.^[5] Anorexia, weight loss and vomiting can precipitate nutritional deficiencies even after functional bariatric procedures, thereby consolidating the need for prophylactic vitamin supplements.^[1,2,4] We report a case of reversible dry Beri-Beri postintragastric botulinum toxin injection who had a remarkable improvement with thiamine therapy.

A 27-year-old obese female with no other medical co-morbidities, without a history of alcohol consumption, underwent intragastric botulinum toxin injection for weight reduction. Over the next

eight weeks, she had a weight loss of 20 kg along with nausea and vomiting. She presented to our facility with subacute onset ascending sensorimotor symptoms involving the lower limbs and diplopia. Her neurological examination showed incomplete right sixth cranial nerve palsy, distal predominant weakness of the lower limbs and pan-sensory loss below the midcalf region with absent ankle jerks bilaterally without encephalopathy or ataxia. A possibility of acute inflammatory polyradiculoneuropathy was considered. Nerve conduction study (NCS) and electromyography (EMG) showed reduced bilateral peroneal, tibial and right ulnar compound muscle action potential (CMAP) amplitudes along with reduced bilateral superficial peroneal sensory nerve action potential (SNAP) amplitudes and reduced motor conduction velocity of right peroneal and tibial nerves [Table 1]. Concentric needle EMG showed active neurogenic changes in the right tibialis anterior, medial gastrocnemius and tibialis posterior muscles. The above findings were consistent with acute sensorimotor axonal polyneuropathy. A lumbar puncture was recommended; however, the patient declined it. On account of history of bariatric procedure with rapid weight loss, nausea and vomiting her serum vitamin levels were checked which showed reduced thiamine levels (32.7; range 66.5–220 nmol/L) with normal pyridoxine, cobalamin, copper and folic acid levels. Contrast-enhanced magnetic resonance imaging (MRI) of the brain and spine was normal [Figure 1]. She was started on parenteral thiamine replacement (500 mg intravenous three times daily for three days followed by 250 mg daily for the next three days)^[5] following which she started experiencing improvement of her symptoms. Postdischarge she was maintained on oral thiamine supplements (100 mg twice daily) with normalization

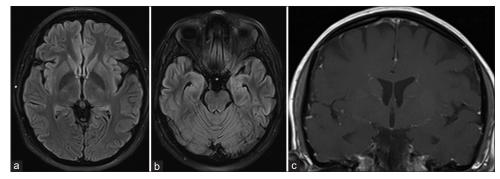
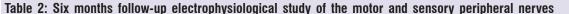


Figure 1: MRI brain axial T2 FLAIR (fluid attenuated inversion recovery sequences) images showing no abnormal signal intensities in the thalamus, mamillary bodies (a), or periaqueductal grey matter (b). Axial contrast enhanced T1 sequences depict no abnormal contrast enhancement in the mamillary bodies (c)

Nerve tested (Right)	Motor nerves			Sensory nerves	
	MCV (Controls) (m/sec)	DL (Controls) (millisec)	CMAP (Controls) (mV)	SCV (Controls) (m/sec)	SNAP (Controls) (μV)
Ulnar	62 (>51)	2.3 (<3.0)	6.2 (>8)	58 (>51)	87 (>18)
Tibial	40 (>41)	2.9 (<5.8)	6.2 (>8)		
Peroneal	39 (>41)	2.9 (<5.5)	1.3 (>3)	45 (>41)	5 (>6)
Sural				52 (>41)	15 (>6)



Nerve tested (Right)	Motor nerves			Sensory nerves	
	MCV (Controls) (m/sec)	DL (Controls) (millisec)	CMAP (Controls) (mV)	SCV (Controls) (m/sec)	SNAP (Controls) (µV)
Median	64 (>51)	2.9 (<3.9)	8.9 (>6)	48 (>51)	88 (>18)
Ulnar	71 (>51)	1.85 (<3.0)	8.5 (>8)	42 (>51)	98 (>18)
Tibial	50 (>41)	4.3 (<5.8)	12 (>8)		
Peroneal	47 (>41)	4.0 (<5.5)	6.5 (>3)	44 (>41)	23 (>6)
Sural				42 (>41)	14 (>6)

of thiamine levels at three months (156.2 nmol/L). She improved remarkably on follow-up with only residual symptoms being numbness involving the soles of feet while maintained on oral thiamine 100 mg once daily. Her six months follow-up NCS demonstrated complete recovery of the sensory and motor parameters [Table 2]. This case highlights the importance of considering acute nutritional deficiency even in the background of functional weight reduction procedures. Even though post-bariatric surgery neurological complications occur within 6 weeks to 20 months after the surgery affecting 4.6-8.6% of patients, with a wide spectrum encompassing both central (Wernicke's encephalopathy, seizures, ataxia, cranial nerve palsies, myelopathy) and peripheral manifestations (acute/subacute/chronic peripheral neuropathy, small fiber neuropathy, mononeuropathy, plexopathy, myopathy, myotonia), data are sparse regarding neurological complications post-functional gastric procedures like intragastric botulinum toxin injection.^[4,6] Albeit rare, other micronutrient deficiencies have also been reported to result in post-bariatric neuropathy such as pyridoxine (mild sensory neuropathy), folate (mild chronic large fiber neuropathy), cobalamin (myeloneuropathy), and rarely copper deficiency (myeloneuropathy and hematological abnormalities).^[1] High index of suspicion of acute nutritional neuropathy in appropriate clinical scenario with prompt institution of therapy plays a pivotal role in improving the overall functional outcomes as demonstrated in our case.

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Conflicts of interest

There are no conflicts of interest.

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