■ Clinical commentary

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Epileptic Disorders

Status epilepticus after gastric bypass surgery

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ABSTRACT

Bariatric procedures are increasingly performed world-wide. They potentially have severe consequences for the nervous system. We report the case of a 39-year-old female who presented with status epilepticus after gastric bypass surgery. A diagnosis of multiple nutrient and vitamin deficiencies was made and she received vitamin supplementation with a good clinical response.

Key words: status epilepticus, bariatric surgery, vitamin deficiency, pyridoxine, thiamine

No fewer than 685,874 bariatric procedures were performed world-wide in 2016 [1]. Malnutrition represents a frequent complication of such procedures through malabsorption or reduced intake with potentially severe consequences for the nervous system, including encephalopathy, myelopathy, neuropathy and seizures [2]. Here, we describe the case of a patient with status epilepticus (SE) as a complication of gastric bypass surgery.

Case study

A 39-year-old female patient with no neurological history was transferred from another hospital to the intensive care unit (ICU) of our institution for the management of Grade IV hepatic encephalopathy due to non-alcoholic fatty liver disease. Her medical history was also notable for a gastric bypass surgery six months prior to admission, with reported lack of compliance to nutritional follow-up and supplementation. In addition to signs of hepatic encephalopathy, physical examination also revealed a diffuse erythematous rash (*figure 1A*), which was considered consistent with multiple vitamin and nutrient deficiencies. She was treated with continuous haemofiltration and parenteral nutrient supplementation.

She progressively became more responsive and was transferred to the neurology ward. There, she presented with a cluster of generalized tonic-clonic seizures, which evolved into refractory right hemispheric motor SE, then finally into non-convulsive SE (*figure 1B*). She was treated with intravenous lorazepam, lacosamide, levetiracetam and enteral topiramate. Seizure control was achieved three hours after treatment was initiated. Brain magnetic resonance imaging (MRI) revealed peri-ictal increased signal abnormalities in the right pulvinar on fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI) sequences (*figure 1C-D*). Results from lumbar puncture were unremarkable. Extensive additional blood tests confirmed severe malnutrition with numerous nutritional deficiencies (*table 1*) but normal ammonia levels (52 µg/dL; normal values: 19-87 µg/dL) and normal electrolyte levels. Dosage

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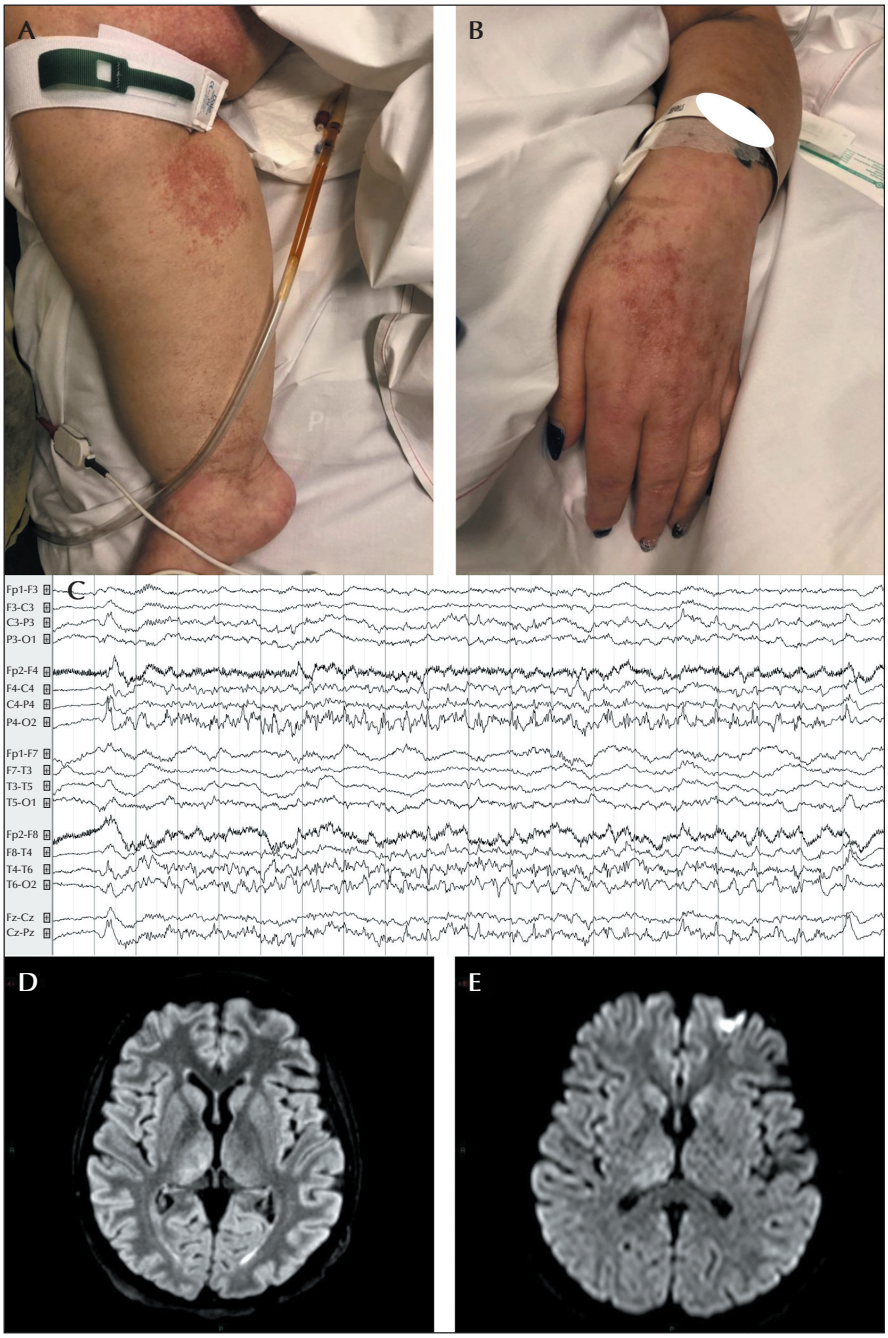

■ **Figure 1.** Clinical, EEG and imaging findings. (A) Dermatological examination showed confluent erythematous macules, symmetrically distributed on the inner side of the thighs and legs; although the lesions blanched on pressure, there was a slight delay in capillary refill. (B) These macules were also present on the upper limbs, to a lesser degree; some fine scales were noted on the dorsa of the hands and medial aspect of the elbows, and ecchymosis was present at pressure points on the upper limbs with livedo reticularis on the feet. Examination of the mucous membranes and nails was unremarkable. (C) Continuous EEG recording revealed nearly continuous ictal activity over the right temporo-parietal region (longitudinal bipolar montage; 20 seconds per page; high-frequency filter cut-off: 70 Hz; low-frequency filter cut-off: 0.5Hz; notch filter off). (D, E) Brain magnetic resonance imaging (MRI) revealed peri-ictal increased signal abnormalities in the right pulvinar on fluid-attenuated inversion recovery (FLAIR) (D) and diffusion-weighted imaging (DWI) (E) sequences.

▼ **Table 1.** Nutrients blood levels.

Nutrients	Patient's values	Normal values
Zinc	35 µg/dL*	80-120 µg/dL
Copper	51 µg/dL*	80-155 µg/dL
Iron (TIBC)	33 µg/dL*	250-400 µg/dL
Vitamin A	0.1 mg/L*	0.2-1.0 mg/L
Vitamin D	18 µg/L*	30-80 µg/L
Vitamin E	9.2 mg/L	5-20 mg/L
Folate (B9)	10.3 µg/L	> 4.6 µg/L
Hydroxy-cobalamine (B12)	1676 ng/L*	197-771ng/L
Albumin	19 g/L*	40-49 g/L
Prealbumin	< 0.05 g/L*	0.2-0.4 g/L
Azotemia	7.0 mg/dL*	16.6-48.5 mg/dL

*Abnormal values

of thiamine, niacin and pyridoxine levels were unavailable. Cerebrospinal fluid studies, including screening for autoantibodies, were unremarkable.
A diagnosis of multiple vitamin and nutrient deficiency was made and she received further parenteral supplementation. She was discharged from the ICU upon SE control and admitted to a neurological rehabilitation unit. She was discharged home from the hospital after 38 days, after having progressed to a fully ambulant state with persistent mild executive dysfunction. Anti-seizure medications were discontinued 10 months after the episode of SE and she has remained seizure-free for more than a year since then. She now undergoes a thorough nutritional follow-up with no evidence of malnutrition.

Discussion

Severe malnutrition and its potential life-threatening complications represent a major burden among patients undergoing bariatric surgery. As illustrated in this case, bariatric surgery induces malnutrition of which the severity depends on surgical technique and adhesion of patients to nutrient supplementation. Lack of compliance may cause deficiencies and neurological complications. In adults, seizures and SE due to malnutrition or vitamin deficiency have rarely been reported. Deficiency in pyridoxine [3, 4], cobalamin [5] and niacin [6] have mostly been incriminated. Pyridoxine deficiency-induced seizures can occur during pregnancy, with chronic or acute liver disease, after isoniazid intoxication or in patients receiving levodopa/carbidopa intestinal gel infusion for advanced Parkinson disease [3]. A history of pyridoxine-dependent epilepsy during childhood is sometimes reported. The mechanisms include reduced synthesis of gamma amino butyric acid (GABA) [4]. The mechanisms through which cobalamin and niacin deficiency might lead to seizures are unclear. Acute hepatic failure was suggested as a potential cause for SE in this case. Indeed, SE may occur during Grade III-IV hepatic encephalopathy and acute liver failure [7]. In our case, normalization of liver function tests and ammonia levels were obtained before SE onset, suggesting an alternative diagnosis. Further, liver failure may also be a complication of malnutrition due to bariatric surgery [8]. It is also possible that haemofiltration for the alleged hepatic encephalopathy may have worsened such deficiencies. In fact, haemofiltration is suspected to induce micronutrient loss through direct elimination in effluent [9] and may worsen pre-existing nutrient deficiencies.

Conclusion

Bariatric surgery can lead to life-threatening complications, including SE. The mechanisms are unclear but likely involve multiple nutrient and vitamin deficiencies, possibly predominantly pyridoxine. ■

Supplementary material.
Summary slides accompanying the manuscript are available at www.epilepticdisorders.com.

Disclosures.
The authors have no conflicts of interest to declare.

Consent.
Signed consent authorising the publication has been obtained.

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TEST YOURSELF

(1) Which of the following deficiencies is associated with seizures (more than one answer may apply?
A. Niacin deficiency
B. Vitamin B6 deficiency
C. Folic acid deficiency
D. Vitamin B12

(2) Production of which of the following is reduced in pyridoxine deficiency-induced seizures?
A. Gamma amino butyric acid (GABA)
B. N-methyl-D-aspartate (NMDA)
C. Amino-hydroxy-methyl-isoxazolepropionic acid (AMPA)
D. Adenosine

(3) Which of the following clinical entities may be caused by malnutrition (more than one answer may apply)?
A. Encephalopathy
B. Polyneuropathy
C. Neuromuscular junction disorders
D. Seizures

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com.