

# When General Surgeon Should Call Neurologist and What Neurologist Should Know About Obesity Surgery?

## Genel Cerrah Ne Zaman Nöroloğu Aramalı ve Nörolog Obezite Cerrahisi Hakkında Ne Bilmelidir?

<sup>id</sup> Serpil DEMİRCİ<sup>a</sup>, <sup>id</sup> Melike DOĞAN ÜNLÜ<sup>a</sup>

<sup>a</sup>Süleyman Demirel University Faculty of Medicine, Department of Neurology, Isparta, TURKEY

**ABSTRACT** Obesity is an ever-increasing problem all over the world and may associate with and cause many metabolic conditions. Bariatric surgery is recognized as the most effective procedure in losing weight, maintaining the weight loss over time and in reversing the associated comorbidities. However, surgery itself may cause some systemic complications due to limited food and micronutrient passage through the operated stomach. Up to 16% of the patients may develop complications involving any part of the nervous system. Neurological complications may develop immediately or long years after the surgery. Immediate post-operative complications are mononeuropathy/plexopathy and rhabdomyolysis. The common early complications are encephalopathy (in the form of Wernicke or Korsakoff encephalopathy), radiculoneuropathy mimicking Guillain-Barre syndrome and polyneuropathy. Optic neuropathy, myelopathy, peripheral polyneuropathy may develop over long-time course. Patients with these neurological complications are found to have isolated or multiple nutrient deficiencies of vitamins and minerals. Among all thiamine, B12 and copper deficiency ranks as the most common ones observed after surgery. Deficiencies of riboflavin, niacin, pyridoxine, folate, vitamin D, and vitamin E, iron, calcium, phosphorus, magnesium and trace elements such as zinc, iodine, and selenium have also been reported. Symptoms vary according to presentation time, anatomical location and injury mechanism. Occurrence of these neurological complications emphasizes the importance of patient education about adherence to nutritional suggestions and surveillance after surgery. Awareness and early intervention may prevent or heal these disabling and sometimes fatal complications.

**Keywords:** Obesity; bariatric surgery; central nervous system diseases; peripheral nervous system diseases

**ÖZET** Obezite tüm dünyada giderek artan bir sorundur ve birçok metabolik bozukluğa neden olur veya bunlarla birlikte bulunabilir. Bariatrik cerrahi kilo vermede, zaman içinde kilo kaybını korumada ve ilişkili komorbiditeleri tersine çevirmede en etkili yöntem olarak kabul edilir. Bununla birlikte, ameliyatın kendisi ameliyat edilen mideden sınırlı yiyecek ve mikro besin geçişi nedeniyle bazı sistemik komplikasyonlara neden olabilir. Hastaların %16 kadarında sinir sisteminin herhangi bir bölümünü etkileyen komplikasyonlar gelişebilir. Nörolojik komplikasyonlar cerrahiden hemen sonra ya da yıllar sonra ortaya çıkabilir. Cerrahi sonrası hemen gelişen komplikasyonlar mononöropati/pleksopati ve rabdomiyolizdir. Sık erken komplikasyonlar ensefalopati (Wernicke veya Korsakoff ensefalopatisi şeklinde), Guillain-Barre sendromunu taklit eden radikulonöropati ve polinöropatidir. Optik nöropati, miyelopati ve periferik polinöropati uzun dönemde gelişebilir. Bu nörolojik komplikasyonları olan hastalarda tek veya çoklu vitamin ve mineral eksiklikleri saptanabilir. Tiamin, B12 ve bakır eksikliği cerrahi sonrası en sık görülenlerdir. Riboflavin, niacin, piridoksin, folat, vitamin D ve vitamin E, demir, kalsiyum, fosfor, magnezyum ve çinko, iyot ve selenyum gibi eser elementlerin eksikliği de bildirilmiştir. Belirtiler ortaya çıkış zamanı, anatomik bölge ve hasar mekanizmasına göre değişebilir. Bu nörolojik komplikasyonların gelişimi cerrahi sonrası beslenme önerilerine uyum açısından hasta eğitimi ve gözetimin önemini vurgular. Farkındalık ve erken müdahale bu özürlendirici ve bazen ölümcül olabilen komplikasyonları önleyebilir veya iyileştirebilir.

**Anahtar Kelimeler:** Obezite; bariatrik cerrahi; santral sinir sistemi hastalıkları; periferik sinir sistemi hastalıkları

Obesity is becoming an important and increasing health problem all over the world and is now listed among the leading causes of premature death. Obesity

may associate with many metabolic conditions like diabetes, hypertension, vascular problems, and also with increased risk for some cancers. According to 2008

**Correspondence:** Serpil DEMİRCİ

Süleyman Demirel University Faculty of Medicine, Department of Neurology, Isparta, TURKEY/TÜRKİYE

**E-mail:** serpildemirci@hotmail.com



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WHO estimates, approximately 2 in three adults (>20 years old) in Turkey were overweight and one in three were obese; and these numbers are predicted to increase in future.<sup>1</sup>

The first step in management of obese patients is dietary restriction, increased exercise, and weight loss. However, failure of these conservative treatments in many has led the surgical procedures to be used much more commonly especially for those who have a high body mass index (BMI) with no obesity-related comorbidities, or for those with associating comorbid conditions.<sup>2</sup> The most effective procedure among all in losing weight, maintaining the weight loss over time and in reversing the associated comorbidities has been noted as bariatric surgery.<sup>3</sup> Bariatric surgical procedures can be grouped as restrictive and restrictive with malabsorptive properties. Restrictive ones (eg, gastric band, gastric sleeve) reduce the proximal stomach volume capacity, and the latter one (eg, Roux-en-Y, biliopancreatic diversion) interferes the normal digestion and absorption of food.<sup>3,4</sup> Among all, the most commonly preferred and performed one all over the world is the Roux-en-Y gastric bypass surgery.<sup>3,4</sup> Though surgery is recommended to control and prevent obesity-related medical problems, they themselves are not exempt from complications. Besides perioperative or postoperative surgical complications, some patients are also prone to systemic complications due to limited food and micronutrient passage through the operated stomach. Restrictive procedures interfere with the passage of food through the stomach and carry a low risk for the development of clinically significant nutritional deficiency; however, when stomach bypassed surgically, iatrogenic malabsorption of micronutrients, including vitamins and metals, can ensue.<sup>2,4</sup> This iatrogenic malabsorption may lead to immediate or late neurological complications involving any part of the nervous system. Given the ever-increasing problem of obesity, it is not surprising that surgeons are more likely to perform surgical procedures. As a result of this preference, the neurologists are also more likely to confront bariatric surgery-related neurological complications. The rate of neurological complications affecting central or peripheral nervous system or both is reported to range from 4.6

to 16%.<sup>5-11</sup> Most commonly observed complications are encephalopathy, optic neuropathy, myelopathy, poly-radiculoneuropathy, polyneuropathy, and mononeuropathy.

These complications are most commonly seen after nutritional deficiency of thiamine, B<sub>12</sub> and copper.<sup>11,12</sup> Though their precise neurologic significance is not clear, deficiencies of riboflavin, niacin, pyridoxine, folate, vitamin D, and vitamin E is also detected.<sup>11</sup> Although the frequency of the incidence of these vitamin deficiencies varies considerably between studies, the published ratios may give an idea on the frequency of complications that may develop. Among all, vitamin B<sub>12</sub> deficiency ranks the first with a ratio of 64%.<sup>8,13</sup> This is followed by deficiencies of vitamin D (51%), folate (38%), vitamin C (34%), thiamine (18%), vitamin B<sub>6</sub> (18%), riboflavin (14%), and vitamin A (11%).<sup>13</sup> Iron deficiency is not uncommon among patients undergone bariatric surgery but has not been related directly with neurological complications, other minerals such as calcium, phosphorus, magnesium and trace elements such as zinc, iodine, and selenium have not been studied in detail.<sup>11,14</sup> Multiple nutrient deficiencies may also coexist.

Micronutrient deficiencies have been reported to be present in 20-30% of obese patients even before surgery, yielding obesity as a preexisting risk factor for malnutrition.<sup>2,10</sup> After surgery, protracted vomiting, dietary changes, colonic flora changes and loss of absorptive surface, gastric acid and intrinsic factor may worsen the nutritional status, and hence may lead to malnutrition.<sup>2,6,10</sup> Rapid and too much weight loss, protracted vomiting and diarrhea, postsurgical complications, preoperative subclinical vitamin and micronutrient deficiency, neglect of nutritional follow-up and failure to obey nutritional recommendations, food avoidance, reduced serum albumin and transferrin, and type of surgical procedure are reported as the risk factors for neurological complications.<sup>6,11,12,15</sup>

The neural injury developing after bariatric surgery has been explained with different mechanisms. Structural changes along with subcutaneous tissue and protective fat pad loss due to rapid weight

loss render nerves more vulnerable to pressure and compression and increase the risk for compression and entrapment neuropathy development.<sup>10</sup> Likewise, some immediate complications may result from mal-positioning of the patients and from mechanical retractors or radial or ulnar catheters used during surgery.<sup>10</sup> The presence of mononuclear inflammatory infiltrates in sural nerve biopsies, increased intrathecal immunoglobulin G synthesis from patients who had polyneuropathy or radiculoneuropathy after bariatric surgery led some authors to bring forward a possible immune-mediated or inflammatory basis for the development of neurological complications.<sup>6,7,15,16</sup> But the leading cause for the occurrence of neurological complications is the deficiency of vitamins and micronutrients.<sup>11,12</sup> Neurological complications can be classified according to presentation time, anatomical location and injury mechanism. In this review, potential neurological complications are compiled based on time of presentation: immediate, early and late complications.

## IMMEDIATE COMPLICATIONS

**Plexopathy/Mononeuropathy:** Peripheral neuropathies may present either as mononeuropathy or plexopathy and do not have a rare occurrence after surgery. Mononeuropathies present more acutely and unilaterally. One study reported 50 cases with neurological complications among a total of 96 patients undergone bariatric surgery; 18 had mononeuropathies, which included 17 with meralgia paresthetica and 1 with foot drop.<sup>8</sup> Thaisethawatkul et al. reported mononeuropathy in 9% and radiculoplexus neuropathy in 1% of a retrospective study group of 435 patients.<sup>6</sup> Brachial plexus stretch injury and ulnar mononeuropathy are reported as the common ones, even with examples of bilateral plexopathy or ulnar neuritis.<sup>10,16-18</sup> Plexopathy may affect both the lumbosacral and brachial plexus. Presenting symptoms are numbness and weakness of the involved limb, usually accompanied with pain. Cases with gluteal compartment syndrome resulting in sciatic nerve damage have been reported.<sup>19,20</sup> With compression of the lateral cutaneous nerve meralgia paresthetica, varying in degree from mild pruritus to frank anesthesia in the lateral thigh may develop.<sup>2,6,10</sup> Mechanical traction forces,

intra-abdominal pressure increase and anatomical variants are reported as the factors increasing the risk of occurrence of this condition.<sup>2</sup> Meralgia paresthetica is treated conservatively and reported to resolve spontaneously.<sup>2,10</sup> Vagal nerve injury, though rare, has also been reported to occur.<sup>2,10</sup>

**Rhabdomyolysis:** Rhabdomyolysis may be asymptomatic or in some cases may threaten life with resulting electrolyte abnormalities, compartment syndrome, cardiac arrhythmias, disseminated intravascular coagulation, acute renal failure and multisystem organ failure. It has been reported to have a rare occurrence. However, in some case series and prospective studies, its frequency was found to range from 1.4 to 77%, depending on the diagnostic criteria.<sup>20-25</sup> Male gender, age >40 years, BMI >55 kg/m<sup>2</sup>, presence of hypertension, diabetes, sleep apnea, use of statins, prolonged immobilization during surgery, use of propofol and American Society of Anesthesiologists (ASA) physical status >2 are listed as the risk factors increasing the probability of rhabdomyolysis in bariatric surgery.<sup>22-25</sup> Since early diagnosis may have significant impact on outcome, physicians should have a high suspicion of rhabdomyolysis after surgery. The symptoms and signs most commonly encountered are muscular pain, numbness and muscle weakness. Mostly affected muscle groups are gluteal and lumbosacral muscles, rarely shoulder and leg muscles may be compromised.<sup>20,22,25</sup> Elevated serum creatine phosphokinase and serum and urine myoglobin are diagnostic.

**Early complications:** Early complications occur less than one year after surgery. The most common early complications are polyradiculoneuropathy and encephalopathy.

**Polyradiculoneuropathy:** This is a rapidly progressive state, presenting like Guillain-Barre syndrome (GBS). However, there are some distinctive features of polyradiculoneuropathy developing after bariatric surgery from the classical GBS. In recent years, acute postgastric reduction surgery neuropathy (APGARS neuropathy) term was introduced to cover this clinical disorder featured by protracted post-operative vomiting, hyporeflexia and limb weakness.<sup>26</sup> It can occur temporally as early as one week after the surgery or as late as 2 years.<sup>27</sup> Occurrence of

polyradiculoneuropathy is relatively rare, however, in some series the occurrence rate varying between 1-17% has been reported.<sup>6-8,15</sup> As in GBS, patients present with lower limbs pain, ascending parasthesia and progressive limb weakness, ataxia, areflexia and vibratory sensory loss. Weight loss is significant and precedes symptoms. Likewise, a persistent, protracted vomiting in the first months after surgery is remarkable in the personal history of the patients.<sup>28</sup> Along with limb weakness patients may complain from pain, incontinence, diplopia and visual impairment. Less commonly, patients may complain from hearing loss and dysphonia, and in some cases encephalopathy symptoms may coexist.<sup>28</sup> Examination reveals a proximal symmetric lower extremity weakness interfering the ambulation. Cerebrospinal fluid analysis yields normal protein level. Diagnosis is confirmed with electroneuromyography which shows axonal sensorimotor polyradiculoneuropathy characterized by axonal degeneration with no demyelination.<sup>27</sup> It has been reported to occur due to vitamin B<sub>1</sub> deficiency; vitamin B<sub>12</sub>, B<sub>6</sub>, and folate deficiency was also noted in some case reports.<sup>27</sup> Besides micronutrients, deficiencies secondary to malabsorption, immune and inflammatory processes also have been presumed to play a role in the pathogenesis of neuronal injury.<sup>10,11,27</sup> Presence of inflammatory cell infiltrates in sural nerve biopsies of patients with neuropathies or radiculoneuropathies and the presence of anti-ganglioside antibodies in serum of patients supports this hypothesis.<sup>10,15,27</sup> Symptoms and findings may resolve with parenteral thiamine (100 mg/d) supplementation.<sup>29</sup> Intravenous immunoglobulins are recommended to be considered because of an immune-inflammatory pathogenesis.<sup>27</sup> Patients fare better when early treatment is instituted. Recovery of walking function may improve however ataxia and distal weakness in lower extremities may persist in the long-term.<sup>15</sup>

**Wernicke Encephalopathy (WE):** Exact prevalence of WE is not certain. Besides case reports, it is also reported in case series. In one study 4 out of 50 patients (8%) developed WE after surgery, and in another series of 23 patients (among a sample of 500 patients) with neurologic complications after obesity surgery 2 (8.7%) patients had WE.<sup>28,29</sup> Encephalopa-

thy mostly occurs from 4 to 12 weeks after surgery. However, it may ensue as early as 2 weeks to as late as 78 weeks after surgery.<sup>11,12,30</sup> Vomiting, parenteral nutrition, IV glucose without thiamine, increased alcohol intake stood out as the predisposing factors.<sup>2,10-12,30</sup> When WE symptoms developed, patients had lost 8-45% of body weight.<sup>30</sup> Besides classical WE triad (confusion, ataxia and nystagmus), which is found only in 30% of cases, patients with hypothermia, third and sixth nerve palsy, polyneuropathy, dysarthria, dysmetria, myoclonus, asterixis, seizures, papilledema, hearing loss, psychosis and eating avoidance were also reported.<sup>10,11,30</sup> Serum thiamine and erythrocyte transketolase levels confirms the diagnosis, however, it has a low specificity and non-available at many centers and also takes too long time to be used in an acute clinical setting. Magnetic resonance imaging of the brain, with a sensitivity of 53% and specificity of 93%, is the most helpful method to establish the diagnosis of WE.<sup>10</sup> The typical findings are bilateral and symmetrical increased T2 signal, sometimes with gadolinium enhancement in T-1 weighted sequences, in the diencephalon, mammillary bodies, mesencephalic tectum and periaqueductal region. Lesions may be hyperintense on diffusion weighted images and reduced, normal, or increased on ADC (apparent diffusion coefficient).<sup>31</sup> Hemorrhages in diencephalon and thalamic nuclei have also been reported.<sup>10</sup> The typical MRI lesions were observed in 58% of the patients with WE.<sup>31</sup> Once the diagnosis is given, the patient must be commenced on thiamine, 500 mg tid for 3 days, then followed by 250 mg/d until patient shows no more improvement and then continued orally with a dose of 50-100 mg daily.<sup>32</sup> As a preventive measure, daily 250 mg intramuscular thiamine is recommended for the patients with protracted vomiting.<sup>32</sup> Resolution of clinical symptoms and findings may take 3 to 6 months after management even when the diagnosis is made early; and a full recovery is reported 51% of the patients.<sup>2,10</sup> Untreated WE may lead to death or to development of Korsakoff syndrome characterized with anterograde and retrograde amnesia and confabulation.<sup>10</sup> Some patients may suffer from permanent neurological deficits, such as ataxia, nystagmus, neuropathy and Korsakoff psychosis.<sup>30</sup>

**Late complications:** May develop over years.

**Optic neuropathy:** Optic neuropathy (ON), may develop acutely or 1.5-3 years after surgery.<sup>8-11,15</sup> In systematic reviews or case series, ON is reported in 3.6% to 8% of the cases.<sup>8,15</sup> Symptoms include impaired vision with central scotoma, optic atrophy or nyctalopia. It is more frequently developed following malabsorptive procedures and associated with deficiencies of vitamins A, B<sub>1</sub>, B<sub>12</sub>, E and copper.<sup>15</sup> Loss of visual acuity and nyctalopia is remarkable for vitamin A deficiency, whereas vitamin E deficiency may cause visual symptoms related to retinopathy. In thiamine deficiency, persistent blurred vision with nystagmus or difficulty focusing vision may also be present.<sup>15,33</sup> Copper deficiency usually takes decades to manifest, however, a case with acute and bilateral optic neuropathy has also been reported.<sup>34</sup> Some degree of vision improvement was observed in some cases once B<sub>12</sub> injections were resumed.<sup>15</sup>

**Peripheral neuropathy:** Peripheral nerve complications after bariatric surgery may present clinically in different patterns: sensory, motor, sensory-motor polyneuropathy, mononeuropathy and radiculoneuropathy or plexopathy. Temporally it has an insidious onset and chronic course. However, as noted in early complications, polyradiculopathies and plexopathies mostly occur acutely or subacutely; mononeuropathies can develop at any time in the postoperative period. As mononeuropathy carpal tunnel syndrome and the meralgia paresthetica are the most common. Besides, cases with involvement of radial, superficial radial, ulnar, great occipital nerve, truncal, peroneal and sciatic nerves have been reported.<sup>6,8</sup> Though frequently unilateral, a bilateral mononeuropathy may be seen, especially in cases of carpal tunnel syndrome. Though commonly reviewed as an early complication, a late and slowly progressive polyneuropathy may develop. Patients may present with symmetric, distal, painful paresthesias. Neurological examination may yield weakness and gait ataxia and pinprick, vibratory and temperature sensation loss may also be found.<sup>10</sup> Cramps and autonomic symptoms may be noted. In a controlled retrospective study comprised of 435 patients, 71(16%) have been reported to have peripheral neuropathy; 27 (38%) of these cases presented with a sensory pre-

dominant polyneuropathy.<sup>6</sup> In another study of 393 patients, the same authors identified 26 (7%) cases with peripheral neuropathy at a median time of 16 months (ranging between 1 to 108 months); sensory neuropathy comprising the 8% of cases.<sup>7</sup> Frago et al. noted 10 (38.5%) cases of polyneuropathy among 26 patients, Koffman et al. 27 cases (28%) among 96 patients, and Juhasz-Pocsine 5 cases (19 %) among 26 patients.<sup>5,8,15</sup> Risk factors for the development of polyneuropathy are listed as a rapid weight loss, protracted vomiting and diarrhea, not attending nutritional recommendations, low serum albumin and transferrin after surgery, postoperative surgical complications and presence of jejunoileal bypass.<sup>6,7</sup> Polyneuropathy is thought to result from a combination of multiple vitamin and micronutrient deficiencies by many authors. The most commonly encountered ones are thiamine, niacin, pyridoxine, vitamin B<sub>12</sub>, vitamin E, vitamin D, folate and copper.<sup>8,9,35</sup> Electromyography reveals sensorimotor axonal neuropathy. Prominent degeneration and perivascular inflammation can be shown in sural nerve biopsies.<sup>10</sup> Nutritional supplementation may partially improve symptoms.<sup>8</sup> Thaisethawatkul et al. emphasize the importance of a systematic and multidisciplinary approach of intensive nutritional management and recommends screening of vitamins and micronutrients before and after surgery to decrease the occurrence of peripheral neuropathy especially sensory polyneuropathy.<sup>6,7</sup>

**Myelopathy** (Subacute combined degeneration): Posterolateral myelopathy with an insidious onset has been reported with a mean latency of 9.9 years after surgery.<sup>15</sup> Symptoms and findings of patients presenting with myelopathy were noted as gait ataxia, limb weakness, spasticity of lower limbs, hyperreflexia, paresthesias, loss of proprioceptive and vibratory sensation in lower extremities.<sup>10</sup> Pain and temperature sensation loss along with urinary urgency and incontinence have also been reported.<sup>10,15</sup> Imaging of the spinal cord may reveal lesions with increased signal intensity on T2-weighted sequences in majority of cases.<sup>10</sup> Juhasz-Pocsine et al. described 12 patients with myelopathy among 26 cases with neurological complications after surgery. In patients with myelopathy, varying degrees of vitamin B<sub>12</sub>, vi-

tamin B<sub>6</sub>, vitamin E, folate and copper deficiencies have been noted.<sup>8,9,15,35</sup> Some degree of improvement have been reported with nutritional supplementation and increased caloric intake, however, most of the patients are reported to have gait ataxia after a mean follow-up of 3 years.<sup>10,15</sup>

**Myopathy:** Myopathy has a rare occurrence. It was observed only in 2 cases among 139 patients with bariatric surgery-related neurological complications.<sup>15</sup> Patients present with weakness and diffuse pain and electromyography yields a myopathic pattern. Symptoms resolve with appropriate treatment. Myopathy has been reported most commonly with global protein, vitamin D, copper deficiencies; but also related with thiamine, vitamin E and mineral (calcium, phosphate, magnesium) deficiencies.<sup>8,9,35,36</sup> Global protein deficiency mostly occurs in surgical complications such as stenosis or fistulas. In case of sleeve gastrectomy or gastric banding, a rapid and significant weight loss may lead to protein deficiency. Treatment responsive myopathy due to vitamin D deficiency, hypocalcemia and secondary hyperparathyroidism has been reported in three of 106 patients after a gastric surgery.<sup>8,10,11,12</sup>

**Very Rare Complications:** Episodic encephalopathy: Patients with short bowel syndrome may develop a rare form of lactic acidosis; D-lactic acidosis with encephalopathy. In humans, lactic acidosis is mainly due to L-lactic acid, however, in this rare condition the gut flora produces D-lactic acid enantiomer of the lactic acid.<sup>37</sup> High carbohydrate load in the diet, alteration of colonic microbiota, diminished colonic motility and impaired physiological D-lactate metabolism play a significant role in the production of D-lactate.<sup>4,37</sup> History of short bowel syndrome, Roux-en-Y gastric bypass surgery, antibiotic or probiotic overuse, presence of thiamine deficiency, diabetes and renal failure are the conditions increasing the risk for D-lactic acidosis.<sup>37</sup> Clinical presentation is characterized by episodic encephalopathy and metabolic acidosis. Besides, patient may present with slurred speech, ataxia, weakness, headache, nystagmus, blurry vision, aggressive or bizarre behavior, psychosis, lethargy, delirium or even coma.<sup>37</sup> Diagnosis depends on a high index of clinical suspicion in a patient with appropriate risk factors presenting

with metabolic acidosis, elevated anion gap, normal L-lactate level, and characteristic neurological features.<sup>4,37</sup> Electroencephalogram is noninformative, however abnormalities may coincide with D-lactate levels in acute state. Elevated D-lactate levels can be found in serum, urine and stool. The mainstays of long-term management are carbohydrate restriction, hydration, and suppression of the D-lactic acid producing flora with antibiotics.<sup>4,37</sup> A previously asymptomatic patient presenting with episodic encephalopathy associated with recurrent hyperammonemia was diagnosed to have functional ornithine transcarbamylase deficiency. It is postulated that dietary change, the metabolic stress of high protein diet, following bariatric surgery unmasked the condition.<sup>38</sup> One another case presenting with progressive encephalopathy has been found to have hyperammonemia along with zinc, essential and nonessential amino acid deficiencies.<sup>39</sup> Four cases of parkinsonism developed acutely or lately following bariatric surgery have been identified.<sup>40,41</sup> An 18-years old female experiencing generalized seizure and ischemic stroke 4 months after surgery have been reported.<sup>42</sup>

## CONCLUSION

With ever-increasing prevalence of obesity, the surgical procedures are becoming increasingly common because surgery has been proved to be a favorable option in the treatment of obesity. A recent meta-analysis of 771 studies favors beneficial effects of bariatric surgery in morbidly obese patients.<sup>43</sup> However, besides operative complications, patients carry a risk for a number of neurological complications some of which are irreversible. Prevention of these sometimes fatal complications demands that patients are well informed and educated of these potential adverse events. In the meantime, neurologists should have a high index of suspicion for neurological complications, even in patients with vague symptoms such as numbness and muscle pain. They also need to recognize the time to presentation, injury mechanisms and anatomical distribution of these possible complications which appear years to decades after bariatric surgery and are amenable to correction.

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### Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

### Authorship Contributions

All authors contributed equally while this study preparing.

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