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Chronic Nutritional Polyneuropathy After Bariatric Surgery: A Rare Case With A Common Presentation

Bariatrik Cerrahi Sonrası Gelişen Nütrisyonel Polinöropati: Alışılmış Bir Klinikle Ortaya Çıkan Nadir Bir Olgu

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Summary

With rising frequency of bariatric surgery, the common adverse effects of that procedure is more frequently encountered. Our patient unluckily developed pulmonary embolism after 3 months of bariatric surgery and that clinic picture was aggravated with proximal lower extremity weakness and numbness. Electromyography performed on admission revealed asymmetric polyradiculopathy with axonal degeneration, whereas somatosensory evoked potentials showed no tibial peripheric and cortical responses. Although we found no sign of malabsorption (except 25 OH vitamin D deficiency), the history of weight loss of 70 kg within 3 months after the surgery led us to the diagnosis of nutritional polyneuropathy. On the other hand, neuropathic pain in our patient responded well to pregabalin and, after strengthening of muscles via rehabilitation, the patient gained independent mobility again. *Turk J Phys Med Rehab 2012;58:151-3.*

Key Words: Bariatric surgery, polyneuropathy,neuropathic pain

Özet

Bariatrik cerrahilerin daha sık uygulanması sonucunda bu prosedürün yan etkilerine daha çok rastlanılmaktadır. Hastamızda talihsiz bir şekilde bariatrik cerrahiden 3 ay sonra pulmoner emboli gelişmiş ve bu klinik tablo proksimal alt ektstremitede güçsüzlük ve hissizlik ile ağırlaşmıştır. Yapılan elektromyografik inceleme sonucunda aksonal dejenerasyonun hakim olduğu asimetrik polinöropati tanısı konmuş, duysal uyandırılmış potansiyallerde tibial periferik ve kortikal cevaplar alınamamıştır. İncelemeler sonucunda 25 OH Vitamin D eksikliği dışında bir vitamin ya da mineral eksikliği saptanmazken, hastanın operasyon sonrası 3 ay içerisinde 70 kg kaybetmesi bizi nütrisyonel polinöropati tanısına yönlendirmiştir. Diğer yandan hastamızın nöropatik karakterdeki ağrısı pregabaline iyi yanıt vermiş ve kas güçlendirme eğitimi sonrasında hastamız mobilitede bağımsızlığa kavuşmuştur. *Türk Fiz Tıp Rehab Derg 2012;58:151-3.*

Anahtar Kelimeler: Bariatrik cerrahi, polinöropati, nöropatik ağrı

Introduction

The prevalence of overweight and obesity is increasing worldwide. Developed countries face the most devastating results of this health problem, but developing countries have dealt only with the tip of the iceberg so far. When conservative treatment modalities fail, bariatric surgery becomes a true option for morbidly obese patients like our patient. In our daily practice, we frequently encounter cases of polyneuropathy, but nutritional polyneuropathy after bariatric surgery is a rare case for physiatrists. In this case report, we will thoroughly discuss the good clinical outcomes of our patient.

When surgical and non-surgical interventions are compared, there is strong evidence that surgery lead to greater reduction in weight. Seven randomized controlled trials (RCTs) have reported an average reduction of 20% to 32% in initial body weight

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(depending on surgery technique) after 1-2 years of surgery (1). The Swedish Obese Subjects study (2) clearly showed that weight loss following surgery sustained 10 years post-surgery, whereas non-surgical treatment modalities did not result in long-term weight loss. Despite high percentages of weight loss, bariatric surgeries have been shown to result in many adverse events including neuropathies (3,4).

Case

A 41-year-old male with a body mass index (BMI) of 67,32 and meeting the National Institutes of Health criteria for weight loss surgery, underwent an uneventful antecolic antegastric laparoscopic Roux-en-Y gastric bypass (RYGBP). The bypassed small bowel was 150 cm long, approximately 1/3 of its original length. Before the surgery, he was under strict dietary control of the endocrinology department and he used orlistat 120 mg in combination with sibutramine HCL monohidrate 15 mg for 2 years. He lost only 10 kg with that treatment.

He lost 70 kg within 3 months after the surgery. After a bus trip lasting 12 hours he started to complain of right thigh pain, paresthesias, cough and dyspnea. He was diagnosed as having pulmonary embolism and was treated in the internal medicine inpatient unit. Shortly after his discharge, he felt weakness of both legs. As his weakness progressed, he was diagnosed as having asymmetric progressive paraparesis by neurologists and was hospitalized for further diagnostic tests.

Laboratory investigation was within normal range including complete blood count, creatine kinase, hepatic enzymes, renal function, total serum proteins, albumin, Vitamin B12, folate, serum iron, copper, seruloplasmin, vitamin A and E levels. Only 25 OH vitamin D levels were low (<5 ng/ml) and were replaced by Calcium 1000 mg and 880 IU vitamin D tablet and Calcitriol (Active vitamin D) 0.5 mcg tablet daily. No other nutrient or vitamin replacements were provided. In addition, blood sugar and thyroid function tests were within normal range.

The cerebrospinal fluid was analyzed for a possible Guillain Barre Syndrome and was normal. On admission, EMG revealed asymmetric polyradiculopathy with axonal degeneration, whereas somatosensory evoked potentials showed no tibial periferic or cortical responses. Cranial, dorsal and lumbar magnetic resonance imaging indicated no spinal cord pathology explaining paraparesis.

The patient was referred to the department of physical medicine and rehabilitation and hospitalized for the treatment of nutritional polyneuropathy and paraparesis. Patient's BMI was calculated as 42,52 and was still at the level of obesity. At physical examination the upper extremity muscle strength was measured bilaterally as 4+/5 according to 5-point manual muscle strength test. Lower extremity muscle strength was: hip flexors 3-/5 at right and 4-/5 at left, hip extensors 4/5 at right and 4+/5 at left, hip abductors 4-/5 at right and 4/5 at left, knee flexors and extensors bilaterally 3/5, and muscle strength of dorsiflexors and plantarflexors of foot as well as extensor hallucis longus (EHL) strength bilaterally was 0/5.

Superficial sensation testing (pain, temperature and light touch) were normal up to the level T10. Below T10, all dermatomes revealed hypoesthesia and hypoalgesia. Vibration sense was absent at the right side but 10 seconds of vibration sence was measured at the left side. Proprioception and joint position sense at the left toe was normal, but no response was observed at the right side. Sacral sensation was intact and no urinary or fecal incontinence was noticed. Deep tendon reflexes were normal at the upper extremity, but no response appeared at the lower extremity, no pathologic reflex was found.

Neuromuscular electrical stimulation was carried out with Compex® (DJO firm, Surrey/UK) electrostimulation equipment and stimulation of the bilateral gluteus maximus, quadriceps, and the foot dorsiflexors and plantarflexors was performed. An exercise program consisting of lower extremity active-assistive range of motion exercises, isometric strengthening of hip muscles, push-ups, quadriceps setting exercises, balance and coordination exercises, and walking and transfer re-education was administered. One pair of solid ankle-foot orthoses was prescribed in order to assist the patient with walking.

As the patient complained of dysesthesia and paresthesia, pregabalin 75 mg twice daily was introduced with dose escalation to 300 mg daily. Complete resolution of neuropathic pain was noted after two weeks of treatment.

After 3 weeks, physical examination was conducted again. The upper extremity muscle strength was measured bilaterally as 5/5. At the lower extremities, hip flexors/extensors/abductors strength was 5/5 bilaterally. Knee flexors were 4/5 and knee extensors were 3/5 bilaterally. Muscle strength of dorsiflexors and EHL of the foot was still 0/5, but plantar flexors at right were 1+/5 and at left 3/5. At neurological examination, all dermatome tests (pain, temperature and light touch) were normal up to the level L2. Below L2, all dermatome tests revealed hypoesthesia and hypoalgesia. The rest of the neurological and locomotor system inspections revealed no changes.

Discussion

Although bariatric surgery is being performed more frequently due to increasing prevalence of morbid obesity, there are only a few studies reporting on the adverse events following this intervention. The large Swedish obese subjects study reported mortality of 0.25% in the surgical cohort (5/2010 patients within 90 days of surgery) (2). Major adverse events following surgery include anastomotic leakage, pneumonia, pulmonary embolism, band slippage and band erosion (5).

Some late post-operative complications such as nutritional imbalances, and neurological complications like encephalopathies, neuropathies, myelopathies have been reported (6). Among them, peripheral neuropathies are the most common complications and may accompany bariatric surgeries at a rate of 16% (7). Three patterns of neuropathy following bariatric surgery were defined. They include polyneuropathy, mononeuropathies, and radiculoplexoneuropathy.

There are many risk factors predisposing to neuropathies after bariatric surgery. Rapid weight loss, low postoperative BMI, low serum levels of albumin, prolonged postoperative gastrointestinal symptoms, prolonged nausea and vomiting are among them (8,9). In a study, Thaisetthawatkul et al. (7) compared the side effects of gastric bypass surgeries and other abdominal surgeries. The patients developed polyneuropathy more often after bariatric surgery than after cholesystectomy ($p<0.001$). Having jejunoileal bypass and postoperative surgical complications requiring hospitalization were important risk factors.

Some authors suggested that neurological complications following bariatric surgery were due to disturbed protein and vitamin metabolism (10). Deficiencies of iron, zinc, cobalamine (B12), thiamine (B1) and fat-soluble vitamins such as A, D, E, K were determined (10). Skroubis et al. (11) reported that, following RYGBP, iron deficiency increased from 26% to 39% after 4 years of surgery. In the same study, 33% of patients showed vitamin B12 deficiency after a 4-year follow-up. In another study, Brolin et al. (12) followed 298 patients who underwent distal RYGBP. After 2 years, 10% of patients had calcium deficiency and 51% of patients had Vitamin D deficiency. In our case, only 25 OH Vitamin D levels were low and were replaced. In a study of Chang et al. (3), 99 patients were followed and 40 of them had vitamin deficiency. These patients developed neuropathy after bariatric surgery. All neuropathies after procedure were called as "acute post-gastric reduction surgery neuropathy". No subtypes of neuropathies were mentioned. The types of bariatric surgeries were RYGBP, distal gastric bypass, duodenal switch and gastroplasty. The most encountered deficiencies were thiamine and vitamin B12.

The metabolic deficiencies following bariatric surgery are not the only causes of neuropathies (12). Another possible cause of neuropathy is peripheral nerve inflammation following surgery. A recent study carried by Staff et al. (13) suggests that neuropathies following surgeries may have an inflammatory etiology. Nerve biopsies of 21 patients showed increased epineurial perivascular lymphocytic inflammation. Patients treated with immunotherapy improved regarding median neuropathy impairment score. On the other hand, Thaisetthawatkul et al. (7) described abnormal accumulation of mononuclear cells around nerve epineurium and endoneurium in the absence of vasculitis. These findings indicate a multifactorial etiology.

While treating nutritional neuropathy by replacing deficient vitamins or elements, medical therapy to control the neuropathic pain is also important for the quality of life. Attal et al. (14) recommended tricyclic antidepressants and anticonvulsants such as gabapentin and pregabalin as the first-line treatment of neuropathic pain secondary to polyneuropathies. In our case, neuropathic pain in our patient disappeared completely after using pregabalin 300 mg daily.

Commonly anticipated complications after bariatric surgery such as pulmonary embolism and deep vein thrombosis are rare, however, neurological complications due to nutritional deficiencies seem to be pretty common and not easily treatable. Even grave neurologic deficits such as Wernicke-Korsakoff Syndrome may follow such surgical procedures (15). Our patient had an unhappy coincidence of a pulmonary embolism and chronic polyneuropathy. Physicians should consider neuropathic complications stemming from nutritional deficiencies following bariatric surgery in the appropriate clinical situation. This is important for both informing the patient prior to surgery and planning a rehabilitation program.

Conflict of Interest:

Authors reported no conflicts of interest.

References

1. Karmali S, Johnson Stoklossa C, Sharma A, Stadnyk J, Christiansen S, Cottreau D, et al. Bariatric surgery: a primer. Can Fam Physician 2010;56:873-9.
2. Sjöström L. Bariatric surgery and reduction in morbidity and mortality: experiences from the SOS study. Int J Obes (Lond) 2008;32:93-7.
3. Chang CG, Adams-Huet B, Provost DA. Acute post-gastric reduction surgery (APGARS) neuropathy. Obes Surg 2004;14:182-9.
4. Abarbanel JM, Berginer VM, Osimani A, Solomon H, Charuzi I. Neurologic complications after gastric restriction surgery for morbid obesity. Neurology 1987;37:196-200.
5. NIH Conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. Ann Intern Med 1991;115:956-61.
6. Koffman BM, Greenfield LJ, Ali II, Pirzada NA. Neurologic complications after surgery for obesity. Muscle Nerve 2006;33:166-76.
7. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. Neurology 2004;63:1462-70.
8. Maryniak O. Severe peripheral neuropathy following gastric bypass surgery for morbid obesity. Can Med Assoc J 1984;131:119-20.
9. Feit H, Glasberg M, Ireton C, Rosenberg RN, Thal E. Peripheral neuropathy and starvation after gastric partitioning for morbid obesity. Ann Intern Med 1982;96:453-5.
10. Bloomberg RD, Fleishman A, Nalle JE, Herron DM, Kini S. Nutritional deficiencies following bariatric surgery: what have we learned? Obes Surg 2005;15:145-54.
11. Skroubis G, Sakellaropoulos G, Pouggouras K, Mead N, Nikiforidis G, Kalfarentzos F. Comparison of nutritional deficiencies after Roux-en-Y gastric bypass and after biliopancreatic diversion with Roux-en-Y gastric bypass. Obes Surg 2002;12:551-8.
12. Brolin RE, LaMarca LB, Kenler HA, Cody RP. Malabsorptive gastric bypass in patients with superobesity. J Gastrointest Surg 2002;6:195-203.
13. Staff NP, Engelstad J, Klein CJ, Amrami KK, Spinner RJ, Dyck PJ et al. Post-surgical inflammatory neuropathy. Brain 2010;133:2866-80.
14. Attal N, Cruccu G, Haanpää M, Hansson P, Jensen TS, Nurmikko T, et al. EFNS guidelines on pharmacological treatment of neuropathic pain. Eur J Neurol 2006;13:1153-69.
15. Chaves LC, Faintuch J, Kahwage S, Alencar Fde A. Acluster of polyneuropathy and Wernicke - Korsakoff syndrome in a bariatric unit. Obes Surg 2002;12:328-34.