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REVIEW

The Spectrum of Neurological Complications after Bariatric Surgery – Review of the Literature

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Abstract: *Bariatric surgery represents one of the most efficient treatments addressing obesity and its associated complications with an increasing trend. Even if the medical benefits for patients are huge, due to weight loss and despite technical improvements in bariatric procedures, it is still associated with diverse types of complications. Most of the complications appear due to nutrient deficiency, as absorption is compromised after surgical anatomy modification. Neurological complications after bariatric surgery are very diverse with an occurrence within days to years after surgery, most frequently in patients with gastrointestinal or surgical complications. Any segment of the nervous system can be affected with distinct types of manifestations ranging from peripheral axonal neuropathy, nerve palsy secondary to compartment syndrome, encephalopathy, and myelopathy, to psychiatric and demyelinating central nervous system diseases. Identifying patients at risk and systematic screening for nutrient deficiency with proper supplementation is essential for preventing neurological complications and lifelong sequelae. In this paper, we present the spectrum of neurological complications reported after bariatric surgery.*

Keywords: *bariatric surgery; nutrient deficiency; neurological complications*

INTRODUCTION

Bariatric surgery still represents one of the most efficient treatments addressing obesity and because the incidence of severe obesity is rising worldwide, more and more bariatric procedures are done annually in specialized medical centers. Even if the medical benefits for the patients are huge due to weight loss and despite the technical improvements for bariatric procedures, it is still associated with different types of complications.

Most of the complications are due to the modified anatomy which leads to impaired absorption of the nutrient, besides the inflammatory state and mechanical stress induced by surgical procedure. The most common complications are gastrointestinal and surgical complications and include but are not limited to reflux disease, vomiting, dumping syndrome, leaks with high mortality getting up to 15%, stenosis, twists and kinks causing dysphagia, bleeding, enteric perforations, internal hernias, bowel obstruction, ulcerations, and gallstones disease. Other complications seen following bariatric surgery are weight regain, pulmonary thromboembolism, metabolic and hematological disorders, such as anemia, leucopenia, and neuropsychiatric complications. [1]

MATERIALS AND METHODS

The purpose of our paper was to review the existing literature data regarding the neurological complications that occurred after bariatric surgery and the presence of nutrient impairment.

We performed a scoping review of the published literature, and we included original studies, reviews, and a series of cases by searching the Medline, PubMed, and Google Scholar databases. We included articles written in the English language.

We searched for the terms: “bariatric surgery”, “neurological complications and bariatric surgery”, “nutrient deficit and bariatric surgery”, “vitamin deficit and bariatric surgery”, “gastric sleeve”, “gastric banding”, “gastric bypass”, “vertical gastroplasty”, “obesity surgical treatment”.

We excluded papers with respect to bariatric surgery-related complications and pre-operative nutritional deficiencies.

NEUROLOGICAL COMPLICATIONS

Neurological complications after bariatric surgery were reported in 3-16% of cases and usually affected more than one component of the nervous system [2,3].

Neurological disorders are various and affect mainly the peripheral nervous system and less common central nervous system, but both are associated with a poorer quality of life and substantial morbidity. Neurological complications of these patients might occur days to years after surgery and include peripheral neuropathy, compartment syndrome, plexopathy, mononeuropathy, encephalopathy, myelopathy, ataxia, myopathy, myotonia, behavioral and psychiatric disorders [2,3,4].

The physiopathology of neurological impairment comprises nutritional deficiency, rapid weight loss, and compartment syndrome. The vitamin B12 shortage, thiamine deficit, and decreased levels of copper are the most commonly deficient nutrients responsible for neurological conditions development after bariatric surgery. Deficiency of riboflavin, pyridoxine, folate, vitamin D, E, phosphorus, calcium, selenium, zinc, and iodine can also contribute to neurologic dysfunction [3,5].

Impact Of Vitamin and Micronutrient Deficiency After Bariatric Surgery and Neurological Complications

Nutritional impairment after bariatric surgery is quite common and manifests a large spectrum of vitamin and micro-nutrient deficiencies affecting both, the water-soluble vitamins (thiamine, riboflavin, pyridoxine, folate, cyanocobalamin, vitamin C), and the liposoluble ones (A, D, E, K), alongside the fat malabsorption, loss of proteins, and minerals deficiencies (iron, zinc, copper, selenium). All these associate different clinical manifestations within a variable time frame of appearance and usually are overlooked by clinicians. Some bariatric procedures, such as Roux-en-Y gastric bypass and post-biliopancreatic diversion/duodenal switch put the patient at higher risk of developing multiple nutritional deficiencies since the absorptive surface is reduced or bypassed [5,6].

The most common deficiencies reported after bariatric surgery are iron deficiency (which was found in more than 30% of patients) [5], cobalamin and folate deficiency in up to 35%, respectively 39% after five years [6], and thiamine deficiency in 49% of patients [7].

Reports from a French cohort study showed a high rate of overall vitamin B deficiency (74%), with thiamine deficit in more than 47% of the cases [8]. A significant number of patients, up to 30%, already had micronutrient deficiency before bariatric surgery which increased the risk of new complications [9]. Hypovitaminosis D after bariatric surgery was reported in 25-70% of the patients, with calcium deficiency in 10%, but it should be considered that obesity is associated with suboptimal 25-hydroxy-vitamin D and 30% of obese patients could have vitamin D deficiency [10,11]. Vitamin E deficiency was reported up to 65.7% in a recent systematic review, and vitamin A in 5-69% in 2-4 years following surgery. Clinical manifestations of vitamin E deficiency are usually seen after Roux-en-Y gastric bypass, being mainly neurological (ataxia, peripheral neuropathy), but also hemolytic anemia, impaired immune response, ophthalmological disorders, infertility, and nystagmus can occur [12]. Although rarely clinically manifested, deficiency of vitamin C and vitamin K was reported in almost half of the patients [6,12].

Low zinc level after bariatric surgery was associated with high inflammatory status. There are reports of necrolytic migratory erythema with severe bacteremia and optic neuropathy due to zinc depletion [13,14]. The zinc repletion therapy should be done cautiously because it can induce copper deficiency. The copper deficiency occurs mostly after gastric bypass as its absorption occurs in the stomach and duodenum. Many cases of copper deficiency had been reported up till 9 years after bariatric surgery with capricious clinical expression, the majority mimicking vitamin B12 deficiency, consisting of myelopathy, ataxia, peripheral neuropathy, optic neuropathy, blindness, muscle weakness, anemia, and neutropenia [15,16, 17,18].

The diagnosis of copper deficiency usually requires the measurement of serum levels associated with the ceruloplasmin level under 20 mg/dl and C reactive protein because ceruloplasmin is an acute phase protein and levels could be misinterpreted in inflammatory states. MRI study with T2 hyperintensity in the posterior dorsal column of the spinal cord can suggest the diagnosis [19,20].

Most of the neurological complications are often seen in patients with surgical complications, longer hospitalization time, or those with gastrointestinal symptoms, especially protracted vomiting. Moreover, these patients underwent a surgical reintervention in 8-32% of the cases [8,21].

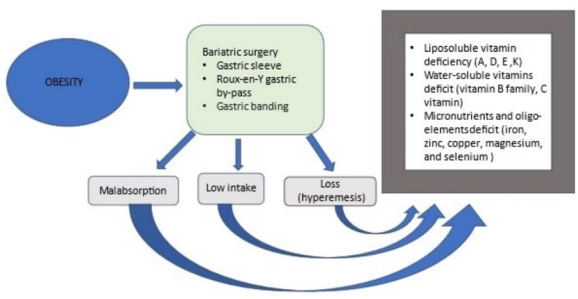

Figure 1: Pathophysiology mechanisms of neurological complications after bariatric surgery and types of nutrient deficiencies

Peripheral nervous system complications

Neuropathy is the most frequent neurological complication described after bariatric surgery and has been reported in 16% of patients after this type of procedure [22]. Its occurrence is induced by vitamin deficiency, especially thiamine, and cobalamin, which are involved in myelin synthesis, but also riboflavin, pyridoxine, and copper deficiency [21].

Another mechanism of occurrence is attributed to entrapment and mechanical compression. Rapid weight loss with subcutaneous protective fat tissue disappearance and inaccurate leg postures can cause compression neuropathy, frequently unilateral. Involvement is usually sensory predominant with late motor progression, chronic and symmetrical, mostly affecting distal limbs, but also small fiber neuropathies and bilateral neuropathies were several times described, contrary to plexopathy which was rarely seen [23,24].

The median nerve compression, the peroneal nerve palsy, and the ulnar nerve palsy were the commonest nerves affected after bariatric surgery. Peroneal nerve palsy is usually unilateral, but cases with bilateral affection were described.

Rapid weight loss with a mean of 45-50 kg reduction in several months is the main risk factor identified for peroneal nerve injury as the nerve's superficial anatomic localization makes it susceptible to impingement [24]. One case of meralgia paresthetica after bariatric surgery was described in an Asiatic report [25]. Rare cases of dry beriberi disease characterized initially by nausea, vomiting, constipation, severe weakness and then progressing to feet tingling and burning were described in the literature with late diagnosis, which was only suggested after performing electromyography [26].

A few cases of acute axonal polyneuropathy resembling Guillain Barre syndrome, rapidly progressive were described in the medical literature after bariatric surgery with good response to vitamin B1 and B12 high dose replacement [27]. There are also recently reported cases of mixed polyneuropathy after bariatric surgery both axonal and demyelinating after five to 24 months post-surgery and some case reports of Guillain Barre syndrome, raising the suspicion that bariatric surgery could induce immunological changes which predispose to autoimmunity [14,28]. Cases of plexopathies affecting the lumbar plexus were reported, manifesting with unilateral severe pain with sensory loss in the hip and thigh region, muscle wasting, patellar reflex loss, and extensive denervation on electromyography study [25].

Several other risk factors for predicting neuropathy development, despite rapid weight loss, were previously reported, such as low albumin and transferrin levels, gastrointestinal complications, surgical complications with long hospitalization, jejunoileal bypass, and lack of nutritional assessment after bariatric surgery [22]. Symptoms of neuropathies include pain, paresthesia, burning feet in small fiber neuropathy, usually progressive weakness, and rarely acute, foot drop, slapping gait, loss of reflection, and objective sensory loss. The diagnosis is confirmed by electromyography studies indicating distal axonal neuropathies. Biopsies of the affected nerves show axonal degeneration and peri-vascular inflammation [24,29].

Treatment includes a high dose of vitamin B12 dose of 1000 ug intramuscular once daily for a week and then weekly, vitamin B1 500 mg daily, intravenous for three days, and afterward 100 mg oral route and acid folic supplementation 5 mg per day, with good response and resolution of symptoms. Vitamin supplementation should be done for long periods and started as soon as possible when diagnosing neuropathy in post-bariatric surgery patients without waiting for confirmation of vitamin deficiency tests since vitamin levels can be normal. Nerve decompression was cited as the most efficient therapy for peroneal nerve entrapment after bariatric surgery [23]. Intravenous immunoglobulin is the treatment of choice for Guillain Barre syndrome, with a good response. Physical therapy can be used as a complementary treatment to vitamin replacement when treating neuropathy [24,29].

Functional impairment is usually mild, but it can be debilitating when the neuropathy becomes chronic or when the patient experiences weakness and foot drop. Also, small fiber neuropathy can evolve into distal neuropathic chronic pain syndrome in all extremities, deteriorating life quality [24,29].

Central nervous system complications (CNC)

The most reported CNS complications after bariatric surgery are encephalopathy, myelopathy, neuropsychiatric disorders, and optic neuropathy [30].

Encephalopathy

The most common type of encephalopathy seen after bariatric surgery is Wernicke encephalopathy secondary to thiamin deficiency, but also different types of encephalopathy are being described, such as hyperammonemia encephalopathy due to high plasmatic levels of glutamine and ammonia, zinc deficiency and urea cycle disorders. Episodic encephalopathy associated with D lactic acidosis is another type of encephalopathy encountered after bariatric surgery, usually precipitated by high carbohydrate intake, and hardly diagnosed by clinicians [31,32].

Clinical manifestation of Wernicke encephalopathy consists of acute confusion, ataxia, nystagmus, sometimes associated with cranial nerve palsies (third and sixth), seizures, dysarthria,

myoclonus, asterixis, papilledema, vestibular symptoms, blurred vision, and polyneuropathy. Almost 80% of patients surviving encephalopathy develop Korsakov syndrome defined by amnesia (retrograde and anterograde), confabulation, and loss of insight, being more common in alcohol consumers. Most cases of Wernicke encephalopathy occur between 14 days to 1-2 years, mostly in women, and are usually precipitated by several factors. Vomit-ing is the most common gastrointestinal symptom precipitating Wernicke encephalopathy, usually preceding thiamine (Gloy VL) deficiency manifestations [8,12,33]. Other factors include rapid weight loss, food avoidance, poor appetite, and lack of vitamin supplementation, as thiamine reserves are depleted in less than 3 weeks without supplementation. In the absence of rapid diagnosis and appropriate treatment, the disease is fatal. Diagnosis of Wernicke encephalopathy is usually clinical, supported by laboratory and IRM studies. Tissue thiamine levels are accurately determined by red blood assays of specific enzymes (erythrocyte transketolase activity coefficient since blood and urine levels are not reliable [34].

IRM changes in Wernicke encephalopathy include symmetrical increased signal on T2/FLAIR phase in dorsomedial thalami, mamillary bodies, tectum plate, periaqueductal area, and around the third ventricle. Treatment of Wernicke encephalopathy consists of intravenous/intramuscular of thiamine 100 mg for 3-5 days minimum, followed by 30 mg twice orally for several months and correction of hypo-magnesemia since magnesium is a cofactor in thiamine-dependent metabolism. Prevention is mandatory and patients at risk should get thiamine supplementation of 100 mg intramuscularly followed by 50 orally once daily. Recovery in Wernicke encephalopathy induced by bariatric surgery after optimum thiamine supplementation is seen in more than half of the cases, the others remaining with cognitive deficiencies, gait disturbances, nystagmus, psychosis, and rarely death [15,30,33].

Hyperammonemia encephalopathy represents a particular type of encephalopathy recognized in the past years in patients who have undergone Roux-en-Y gastric bypass with a high mortality rate, characterized by a high level of ammonia in the absence of liver disease [31]. In a review from Andrew Z. Fenves et all in 2015 a mortality rate of 50% was reported in these patients with a predominance of women as the conditions were most often seen in this category of patients [35].

Laboratory findings include mild to moderate increased levels of ammonia, high levels of plasma glutamine, low albumin and zinc levels, essential amino acids deficiency, and reactive hypoglycemia [36].

The precise mechanism of onset is not yet understood, but several factors were until now described in the hyperammonemia state after bariatric surgery. Most of the lethal cases of hyperammonemia encephalopathy showed genetic predisposition caused by urea cycle abnormalities with a partial ornithine transcarbamylase deficiency or a mild carbamyl phosphate synthetase (CPS) deficiency [32].

One of the predisposing factors for hyperammonemia is the presence of huge nitrogenous residues in the liver. Intake reduction triggers a high catabolic state with protein breakdown. Ammonia level is also increased by altered gut microbiota that produces more urea lithic strains because of small bowel pouches created by bariatric surgery [31,36]. Case reports from patients with this neurological complication after bariatric surgery reported lower citrulline levels in the intestine affecting ureagenic capacity. Moreover, other predisposing conditions as the presence of a splenorenal shunt should be suspected and ruled out in patients developing hyperammonemia [32,35].

Therapeutic options include protein intake reduction, zinc and amino acid repletion, parenteral glucose, lipid supplementation, and oral lactulose. In refractory cases, hemodialysis and setting back of surgical procedures could be an option [37].

D-lactic acidosis encephalopathy is a rare condition reported mostly in short bowel syndrome, but also described years after bariatric surgery, especially after jejunum-ileostomy, and should be taken into consideration in a patient with anatomic changes of the digestive tract who develops confusion syndrome [38]. Confusion episode is associated with behavioral changes, dysarthria, ataxia, lethargy, and weakness, and is usually triggered by high carbohydrate intake. D lactate serum elevation is a result of carbohydrate fermentation in the small bowel or colon and is hardly diagnosed as serum and urine measurement of D lactate is not commonly done, L lactate being the only form that is most frequently checked when assessing lactic acidosis. A level over 3 mmol/l is diagnostic for this pathology and is often associated with a high serum anion gap [38]. Treatment involves metabolic acidosis correction with intravenous bicarbonate, fasting reducing carbohydrate levels, and intravenous amino acids, but also modulating intestinal microbiota with antibiotics which improve neurological symptomatology [39].

Myelopathy is usually seen years after bariatric surgery being one of the most disabling complications. It commonly affects posterior columns with the dorsal ganglia root and can be also associated with neuropathy (myeloneuropathy) or optic neuropathy. Manifestations consist of vibration and

proprioception loss, gait ataxia, hyperreflexia, and Babinsky sign, sometimes accompanied by sphincter dysfunction and limb weakness [40].

When neuropathy occurs sensory changes in glove and stocking patterns are also seen in these patients after bariatric surgery. This complication is caused primarily by cobalamin deficiency but is also attributed to other deficient nutrients such as copper, vitamin E, and pyridoxine. Correction of these micronutrients initially intravenously and then long-term orally or intramuscularly for vitamin B12 improves neurologic symptomatology but without full recovery in some patients. For optimum management copper levels should be regularly checked and supplemented as quickly as needed initially intravenous and then oral administration [41].

Optic neuropathy affecting one or both side nerves is associated with bariatric procedures because of nutritional deficiencies in vitamin B12 and vitamin A, copper, and rarely zinc, causing malfunction of nerve fibers. The onset can vary during a large period and can be acute or progressive causing blindness. As optic neuropathy pathogenesis is linked to demyelination, permanent blindness may not be overcome [22,42].

Central nervous system demyelination presenting as multiple sclerosis which fulfilled the McDonald criteria for diagnosis was reported in a series of cases after various types of bariatric surgery with relapsing-remitting forms [4,43,44], without understanding the mechanism of appearance or the exact relationship between the events, but it might be attributed to rapid and substantial weight loss. Other previous case reports also described multiple sclerosis onset and myasthenia gravis after rapid weight loss induced by bariatric surgery or highly restrictive diets [45]. Two cases of monophasic central nervous system demyelination were also reported by Falah et al in a series of patients with neurological complications after bariatric surgery, and in a Brazilian study [44].

NEUROPSYCHIATRIC DISORDERS

Neuropsychiatric disorders are seen after bariatric surgery, usually consisting of eating disorders (anorexia and bulimia nervosa, binge eating, avoidance eating (post-surgical avoidance eating disorder), reports of acute mania [46], and higher risk of suicide. These disorders frequently appear in preexisting psychiatric conditions, making psychiatric evaluation before surgery mandatory to find patients at risk who need to be followed. Several cases of cognitive complaints were described following bariatric surgery in Roux-en-Y Gastric Bypass induced by focal brain atrophy in the bilateral posterior thalamus [47] but need to be further studied.

Other conditions described after bariatric surgery with higher incidence are stroke, seizures [48,49], spontaneous intracerebral hypotension linked to vitamin A, and D deficiency, and spontaneous leakage of cerebrospinal fluid, but the exact relationship needs to be established in future studies [30].

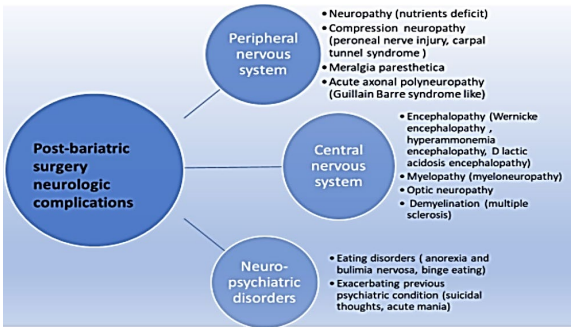

Figure 2: The spectrum of neurological complications described after bariatric surgery. From peripheral to central nervous complications, to neuropsychiatric disorders.

CONCLUSIONS

Complications after bariatric surgery have a broad spectrum of manifestations involving both the peripheral and central nervous system and should be suspected and diagnosed as soon as possible to prevent long-term sequelae. The risk of developing central nervous system demyelination or stroke events after bariatric surgery should be further evaluated in future studies. Patients with rapid weight loss and those who develop gastrointestinal symptoms and surgical complications are considered at high risk for developing neurological complications, which is why nutritional deficiency assessment and supplementation should be done as early as possible.

Conflicts of interest and sources of funding

The authors declare no conflict of interest.

Authors' contribution

Conceptualization: FIR; Methodology: MJ, RSC, PN; Writing - original draft preparation: MC; writing-review and editing SB, MC; Supervising: FIR. All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate

Not applicable.

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