Neurological Complications After Bariatric Surgery

13

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All answers lie in the neurons.

— Abhijit Naskar

13.1 Introduction

The term "complications" implies any deviation from the normal postoperative course. The incidence of complications is often used as an indicator for the quality of surgery. The occurrence of complications in bariatric surgery is not accepted by the patient, family, and the society because obesity is usually not perceived to be a disease. Though early postoperative complications like bleed, leak, etc. are commonly discussed, long-term issues like neurological complications are rarely discussed in bariatric forums. Mostly associated with nutritional deficiencies, neurological complications are often diagnosed late underscoring the importance of increasing awareness of this complication.

Patients with obesity suffer from malnutrition with 20–30% presenting with micro-nutrient deficiencies before surgery [1]. After surgery, these deficiencies can worsen because of inadequate intake, vomiting episodes, mal-absorption, lack of balanced diet, bacterial overgrowth, and reduced gastric acid. Despite guidelines for postoperative micro-nutrient supplementation, variations are prevalent in dietary practice followed by patients and even amongst bariatric clinicians. As more diversionary procedures are being performed, this requirement gains additional significance.

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It is probably important to mention here that the systematic review and meta-analysis published in 2019 showed an overall improvement of peripheral neuropathy in patients undergoing bariatric surgery [2].

Epidemiology A large longitudinal bariatric series reported an incidence of 1.3% (133/9996) of neurological complications [3]. Abarbanel et al. [4] described that neurological complications developed 3–20 months after restrictive procedures in 4.6% of their 500 patients. The highest incidence, however, was recorded in a study from the Mayo Clinic that described 16% of peripheral neuropathy among patients undergoing bariatric surgery [5]. The incidence of neurological complications in another study of 592 sleeve gastrectomy was 1.18% after 4.6 ± 3.4 months [6]. There are no large scale studies reporting post-bariatric neurological complications from India.

Neurological Complications

- Encephalopathy
- Behavioral abnormalities
- Seizures
- Cranial nerve palsies
- Ataxia
- Myelopathy
- Plexopathies
- Peripheral neuropathy
- Mono-neuropathies: Carpal tunnel syndrome, Meralgia paresthetica
- Compartment syndromes
- Myopathy
- Myotonia

Based on the duration of onset after surgery, they can be classified as:

1. Early (within 1 year)
   - Compression injury of peripheral nerve/muscle: Brachial plexus injury, meralgia paresthetica
   - Encephalopathy: Wernicke's encephalopathy, Korsakoff's psychosis
   - Polyradiculopathy: Acute Guillian-Barre syndrome
2. Late (after 1 year)
   - Optic neuropathy.
   - Myelopathy: Subacute combined degeneration of the spinal cord
   - Peripheral neuropathy: Burning feet syndrome, Carpel tunnel syndrome
   - Myopathy

Pathophysiology of Neurological Complications

1. Mechanical injury to peripheral nerves and muscles: This risk is higher in patients with obesity during surgical positioning (steep head up) due to inadequate padding of pressure points, hyperextension of joints (brachial plexus

injury), injury during arterial line access, or mechanical retractors. There is also a risk of fatal rhabdomyolysis and myopathies associated with compression injury [5]. Additionally, loss of fat makes nerves more susceptible to compression in the postoperative period.

2. Neural inflammation and immunological mechanisms: There have been reports of inflammatory infiltrates in nerve biopsies from patients who had neurological complications after surgery (acute/subacute polyneuropathy or radiculoneuropathy) [3, 5]. In a setting of severe weight loss, a cachexia-like state with associated nutritional deficiencies may lead to inflammatory and immune-mediated neuropathy [5].
3. Nutritional: The most accepted etio-pathogenesis of neurological complications is vitamin and mineral deficiency due to inadequate intake (surgical restriction), hyperemesis, or reduced absorption from the intestine (surgical bypass) [5].

Proposed Mechanisms of Nutritional Deficiency

- Stomach: Loss of gastric acid and intrinsic factor, inadequate meal size, repeated emesis.
- Intestine: Reduced absorptive surface (duodenum is the main absorption site of calcium, iron, and vitamin B1), bacterial overgrowth.
- Others: Intolerance to certain foods (milk, rice, meats, fiber).

Specific Deficiencies

1. Thiamine (vitamin B1): Thiamine deficiency may occur within 3–6 months postoperatively but has been reported as early as 6 weeks [7, 8]. Vitamin B1 is obtained mainly from vegetables, whole-grain, and supplemented foods. The storage of vitamin B1 is only about 25–30 mg; thus, its stores can be depleted within 2–3 weeks The typical history is insufficient oral intake and repeated episodes of vomiting. As thiamine is mainly absorbed in the duodenum, deficiency can occur, even in restrictive procedures like sleeve gastrectomy, despite oral supplementation if emesis is present. Vitamin B1 deficiency can lead to depression of transketolase activity, reduced oxygen uptake, and lactic acidosis [9]. Impairment of cellular oxidative metabolism and reduction in thiamine-dependent enzymes results in selective neuronal cell death [10].

   Wernicke's encephalopathy and Korsakoff's syndrome have been associated with thiamine deficiency. The diagnostic criteria for Wernicke's encephalopathy require two of the following four features:

   (a) Dietary deficiency
   (b) Oculomotor abnormality (nystagmus)
   (c) Cerebellar dysfunction (ataxia) and
   (d) Confusion or mild memory impairment

   Peripheral neuropathy may also accompany Wernicke encephalopathy and has been referred to as "bariatric beriberi." It may occur earlier than Wernicke's encephalopathy and mainly affects the lower limbs with both sensory and motor deficits. Sometimes, it may progress rapidly over a few days mimicking Guillain-Barré syndrome. Permanent impairment of recent memory with con-

fabulations, known as Korsakoff's syndrome, is also associated with thiamine deficiency.

Deficiency can be confirmed by measuring the vitamin B1 pyrophosphate effect in erythrocyte transketolase studies. A typical clinical picture with the features of encephalopathy, coupled with the response to administration of parenteral vitamin B1, is sufficiently diagnostic. Sometimes, a magnetic resonance imaging (MRI) of the brain may be required, which shows characteristic abnormalities-hyperintense signal abnormalities on T2-weighted images in the dorso-medial thalamic nuclei, periventricular gray matter, and mammillary bodies. Treatment consists in intravenous (IV) administration: 500 mg IV three times a day for 3 days, followed by 250 mg IV daily until improvement stops. This dose must be followed by an oral dose of 50–100 mg daily [10, 11]. Patients with history of bariatric surgery and repeated postoperative vomiting should be given preventive thiamine supplementation by 250 mg IM once a day. Ophthalmoplegia usually resolves in a few hours and confusion improves within a few days. Complete recovery is possible within 3–6 months of initiation of therapy if the symptoms are recognized early [12]. If thiamine deficiency is not discovered early, patients may be left with permanent deficits and mental status changes.

2. Cobalamin (vitamin B12): Vitamin B12 deficiency may appear several years after bariatric surgery, since sufficient liver stores compensate for the initial dietary insufficiency [13]. The absorption of vitamin B12 requires the intrinsic factor derived from gastric parietal cells, acidic gastric pH, and its absorption in the ileum, any of which may be affected by bariatric surgery [14]. Vitamin B12 plays a role in the synthesis of the myelin sheath and in methylation of RNA which slows RNA degradation, explaining the involvement of long axons in vitamin B12 deficiency. Subacute combined degeneration is seen, in which the peripheral nerves and posterior columns of the spinal cord are chiefly affected (demyelinated). Common neurologic symptoms include paresthesias, weakness, hyporeflexia, spasticity, ataxia, loss of position and vibratory sense, incontinence, loss of vision from optic neuropathy, dementia, psychosis, and altered mood [15]. Severe autonomic symptoms may also rarely occur [16]. The initiation of vitamin B12 supplementation within 6 months postoperatively is recommended. Oral crystalline vitamin B12 at a dose of at least 350 mcg/day has been shown to maintain normal plasma vitamin B12 levels [17]. Optimal dosing of oral, sublingual, or intranasal forms of B12 supplementation has not been well studied.
3. Other deficiencies: Low plasma folate levels are seen in upto 42% of patients undergoing gastric bypass surgery after 3 years. Folate deficiency with an accompanying peripheral neuropathy is a known phenomenon [18]. Oral folic acid supplementation (400–500 μg/day; recommended 1 mg daily) has been shown to be effective in maintaining levels within the reference range [17, 19]. However, isolated folate supplementation can lead to masking of an underlying B12 deficiency, and may cause progression of neurological damage.

Niacin deficiency or pellagra have been reported with restrictive procedures like vertical gastroplasty [20]. This syndrome is characterized by symmetrical rash on sun-exposed areas (casal collar) with desquamation. Other features include diarrhea, photosensitivity, glossitis, dementia, hallucinations, and encephalopathy.

Vitamin D deficiency after gastric bypass may present with symptomatic hypocalcemia. In a report by Marinella [21], patients developed carpo-pedal spasms, intermittent facial twitching, and ophthalmoplegia in association with hypocalcemia many years after gastric bypass surgery. This patient responded well to calcium supplementation. Myopathy has been linked to vitamin D deficiency and has been shown to improve with treatment (400–800 units per day) [22]. Higher daily dose (1000–2000 units) have also been frequently used today [22]. These doses may be recommended in more malabsorptive procedures like OAGB or DS with longer bypassed limbs. Reduced vitamin D levels are common in the general population and all patients undergoing bariatric procedures should be screened pre-operatively with 25-hydroxyvitamin D levels. They can subsequently be modified based on serum levels and bone densitometry. Recommended doses of elemental calcium after bariatric surgery range from 1200 to 2000 mg daily, along with supplementary vitamin D [17, 19].

In addition to vitamin D, patients undergoing Roux-en-Y gastric bypass may also be at risk of deficiency of another fat-soluble vitamin, vitamin E which is also associated with neurological manifestations. Low vitamin E levels have been found 6–12 months after surgery but may not be apparent for 5–10 years [21]. Common findings in patients with vitamin E deficiency include hyporeflexia, proximal myopathy, limb, and truncal ataxia, reduced vibration and position sense, ophthalmoplegia, ptosis, and dysarthria [23]. Saccadic eye movements (following a moving object) may be slow, and progressive gaze impairment may be noticed. Some patients with ophthalmoplegia also have medial rectus dysfunction and an associated nystagmus. Severe vitamin E deficiency has also been linked with polyradiculopathies, peripheral neuropathies, and sensory axonopathy. The recommended dose of vitamin E is 400 IU daily. The proposed supplementation in asymptomatic patients post-surgery is a standard multi-vitamin formulation rich in vitamin E [24].

Copper deficiency may lead to myelopathy after bariatric surgery that is clinically indistinguishable from vitamin B12 deficiency. Kumar [25] described a 49-year-old lady who developed myelopathy characterized by spastic ataxia, symmetrical lower limb hyper-reflexia, and loss of vibratory perception and of pinprick and touch sensations in feet 24 years after bariatric surgery. As vitamin B12 levels were normal, serum copper and ceruloplasmin levels were measured and were found to be reduced. Patients improved clinically after intravenous administration of copper (as cupric sulfate). Other features found in patients with copper deficiency include peripheral neuropathy, optic neuropathy, myopathy, demyelination, and myelo-opticoneuropathy (Tables 13.1 and 13.2).

Table 13.1 Neurological complication, etiology, and symptoms

Complication	Etiology/Deficiency	Symptoms
Wernicke's encephalopathy	Thiamine	Ophthalmoplegia or nystagmus, ataxia, and mental status changes
Korsakoff's psychosis	Thiamine	Severe memory loss, anterograde amnesia, retrograde amnesia, confabulations
Polyradiculoneuropathies	Thiamine	Pain in the lower limbs, weakness, ascending paralysis, areflexia
Optic neuropathy	Vitamin A, vitamin B6, B12, and copper	Blurred vision with central scotoma, night blindness
Myelopathy/subacute combined degeneration	Vitamin B12, vitamin E, copper, rarely folate	Disabling gait ataxia, spasticity in the legs, paresthesias, loss of proprioception and vibratory sensations in legs, limb weakness
Polyneuropathy	Vitamin B1, B12, B6 (pyridoxine), vitamin E, copper, and possibly vitamin D and folate	Symmetric, distal, sometimes painful paresthesias ("burning feet syndrome"), sometimes weakness, gait ataxia, loss of pinprick, vibratory, and temperature sensation
Mononeuropathy	Same as polyneuropathy	Carpal tunnel syndrome, ulnar neuropathy, radial neuropathy, peroneal neuropathy [foot drop] lateral femoral cutaneous neuropathy (meralgia paresthetica) Restless leg syndrome
Myopathy	Protein, copper, selenium, magnesium, Calcium & Vitamin D	Weakness, vague muscle pains

Table 13.2 Studies reporting neurological complications after bariatric surgery and their findings

Study	N	Procedures	Complication	Deficiency
Koffman et al. [3]	96	RYGB, VBG, adjustable band, BPD-DS	Peripheral neuropathy = 60 Encephalopathy = 30 Myopathy = 7	Thiamine in 27 (28.1%)
Abarbanel et al. [4]	23/500 (4.6%)	RYGB, VBG	Polyneuropathy = 12 Wernicke's = 2	Thiamine in 3(12%)
Thaisetthawatkul et al. [5]	71/435 (16%)	Restrictive and malabsorptive	Polyneuropathy = 27 Mononeuropathy = 39 Radiculopathy = 5	–
Tabbara et al. [6]	7/592 [1.2%]	Sleeve gastrectomy	Polyneuropathy = 5 Wernicke's = 2	Thiamine 2 (29%)
Landais A [12]	2	Sleeve gastrectomy	Severe axonal neuropathy = 1 Wernicke's = 1	Thiamine, Vit. B6
Juhasz-Pocsine et al. [13]	26	Restrictive and malabsorptive	Myelopathy, encephalopathy, and polyradiculopathy	Vit. B12, copper
Sen et al. [26]	7/635	Sleeve gastrectomy	Peroneal nerve entrapment neuropathy	–

Overview of Treatment of Neurological Complications

It is important to detect and treat neurological complications early as delayed treatment may lead to irreversibility and residual neurodeficit has also been reported [27].

It is prudent to start empirical therapeutic supplementation even before the test results are available since the vitamin B complex does not carry toxic effects.

13.2 Conclusion

Neurological complications after bariatric surgery are rare. They are often preceded by gastrointestinal symptoms like vomiting of prolonged duration, rapid weight loss, and a failure of compliance with vitamin supplementation and dietary follow-up. Re-hospitalization may be necessary, and a multidisciplinary team management including a bariatric physician and nutritionist is crucial to establish the diagnosis, provide appropriate treatment, and halter the progression of these neurological complications.

Key Points

- Neurological complications after bariatric surgery are preventable and rare but are often diagnosed late.
- They are often preceded by gastrointestinal symptoms.
- The most common cause of neurological complications are nutritional deficiencies.
- Hospitalization may sometimes be necessary.
- Multidisciplinary team management is crucial.

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