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Neurosurgical issues of bariatric surgery: A systematic review of the literature and principles of diagnosis and treatment

Daniele Bongetta[a,b,*], Cesare Zoia[a], Sabino Luzzi[a,c], Mattia Del Maestro[a,d], Andrea Peri[e], Germana Bichisao[f], Debora Sportiello[f], Isabella Canavero[g], Andrea Pietrabissa[b,e], Renato J. Galzio[a,b]

[a] Neurosurgery Unit, Fondazione IRCCS Policlinico San Matteo, Pavia, Italy
[b] Department of Clinical-Surgical, Diagnostic and Pediatric Sciences, University of Pavia, Italy
[c] Department of Emergency and Organ Transplantation, University of Bari "Aldo Moro", Italy
[d] Department of Life, Health and Environmental Sciences (MESVA), University of L'Aquila, Italy
[e] General Surgery Unit II, Fondazione IRCCS Policlinico San Matteo, Pavia, Italy
[f] Anesthesia and Intensive Care Unit I, Fondazione IRCCS Policlinico San Matteo, Pavia, Italy
[g] Emergency Neurology Unit, IRCCS Mondino Foundation, Pavia, Italy

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ABSTRACT

Bariatric surgery is gaining popularity as the treatment of choice of morbid obesity since this condition is constantly increasing over the last decades. Several complications have emerged as the number of surgeries and follow-up data increase. No systematic review of the neurosurgery-related potential complications has been performed to date. Objective of this work is to fill this gap. We reviewed the literature for bariatric surgery-related complications involving the neurosurgical practice. Moreover, we present explicative cases dealing with peri- and post-operative therapeutic precautions. Three pathological mechanisms emerged. The first is related to intracranial pressure alterations and may imply either intracranial hypertension or hypotension syndromes in the operative or post-operative periods. The second is the deficiency of macro- and micro-nutrients which are potential risk factors for neuro- or myelo-encephalopathies, fetal malformations and spine disorders. The third is a dysregulation of both autonomic and endocrine / pituitary control. Neurosurgeons must be aware of the several, multifactorial neurosurgery-related complications of bariatric surgery as their prevalence is likely to be higher in the next few years.

1. Introduction

The WHO reports that obesity is the fifth leading risk for mortality and its prevalence has nearly tripled since 1975. [1] In patients who have not responded to medical treatments, bariatric surgery (BS) is advised if BMI > 35–40 kg/m^2 [2]. The surgical aim is basically to restrict food intake with procedures such as the positioning of a gastric band around the upper stomach or the removal of the greater curvature of the stomach to create a sleeve-like pouch. More complex procedures also imply the division of the GI tract in order to alter the absorption of food. The mean weight loss is of 20-35% in a 2-3-year span. [3] Besides BS-specific complications, several reports have been published about systemic complications, some of which also involved the central and peripheral nervous systems. The analysis of the relationship between obesity and the neurosurgical practice reported controversial results [4–6]. Still, no study has systematically evaluated the relationship between BS and the neurosurgical practice. The aim of this study is to review the potential neurosurgery-related complications of BS focusing on etiopathological mechanisms. Preventive precautions and treatment caveats will be discussed also by means of explicative cases.

2. Materials and methods

In January 2018 we electronically searched PubMed/MEDLINE, EMBASE and The Cochrane Library to find articles dealing with the topic of neurosurgical complication of BS, with no time nor language restrictions. The original query yielded 489 articles. We used this search strategy in PubMed: ((((((((((csf hypotension) OR csf hypertension) OR

* Corresponding author at: UOC Neurochirurgia, Fondazione IRCCS Policlinico San Matteo, Viale Golgi 16, 27100 Pavia. Tel.: +393289672299; Fax: +390382502342.
E-mail address: danielebongetta@hotmail.com (D. Bongetta).

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Table 1
PRISMA guidelines flow diagram.

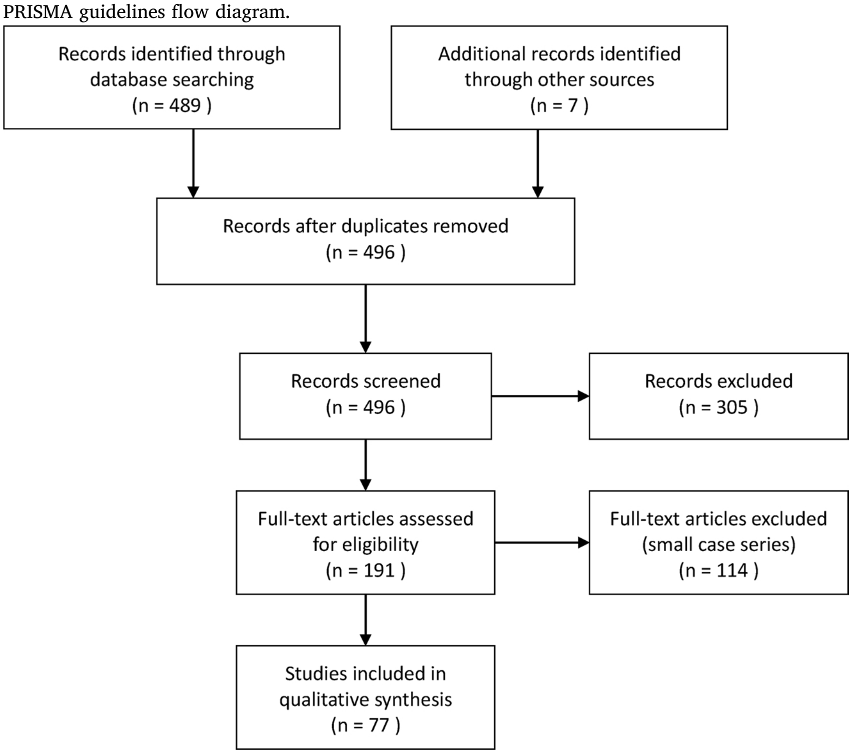

Table 2
Potential complications of BS divided by their etiopathological mechanism.

1 INTRACRANIAL PRESSURE ALTERATIONS
1.1 Intracranial hypertension
1.2 Overdrainage syndrome
1.3 CSF fistula
2 MACRO- AND MICRO-NUTRIENTS DEFICIENCIES
2.1 Peripheral Neuropathy
2.2 Myelo-encephalopathy
2.3 Neural tube defects
2.4 Spine disorders
3 AUTONOMIC AND ENDOCRINE CONTROL ALTERATIONS
3.1 Orthostatic intolerance
3.2 Pituitary gland alterations

csf) OR neurosurgery) OR brain) OR pseudotumor) OR vision) OR blindness) OR neuropathy) OR spina bifida) OR dysraphism) AND bariatric. A flow diagram according to PRISMA guidelines about the selection the articles discussed is reported on Table 1. Two independent researchers conducted literature searches to assess the effective relevance of a given article, if questions arose, consensus was reached through discussion with the senior author. Excluding redundant papers and preferring larger datasets for incidence reports we analyzed 77 papers. Three pathological mechanisms emerged: intracranial pressure alterations, macro- and micro-nutrients deficiencies and autonomic and endocrine control alterations. The results of discussed studies are briefly summarised in Table 2 and reported accordingly. No funding is to be reported.

Given the consent of the patients, we present two cases treated at our Institution with a neurosurgery-related complication of BS.

3. Results and discussion

3.1. Intracranial pressure alterations

Obesity has long been known to be a relevant risk factor for raised ICP and related conditions such as idiopathic intracranial hypertension (IIH) and empty sella syndrome (ESS). Different plausible pathological mechanisms have been proposed to explain this correlation: firstly, the increased abdominal pressure has been assumed to impair cranial and spinal venous outflow, with a consequent raise in venous pressure, determining CSF outflow impairment. Moreover, there is some evidence about the presence of occult cerebral sinovenous obstruction in obese patients, thanks to a hypercoagulable state. Moreover, the involvement of hormonal factors and obstructive sleep apnea syndrome has been suggested. On the basis of these assumptions alone or combined with other CSF diversion techniques, BS has then been reported as a treatment option of both obesity and IIH in selected patients. [7–9] Neurosurgical issues may then arise in operating on patients with a high intracranial pressure, especially if laparoscopic procedures are involved.

3.1.1. Intracranial hypertension

The main concern in dealing with BS candidates is to avoid potential sources of ICP increase during the anesthesiological procedures. In particular hypercarbia, hypoxia, hypertension and hyperthermia need to be avoided. [10] When dealing with hypercarbia, the exogenous CO_2 needed for laparoscopic procedures may be absorbed by the peritoneum leading to an end-tidal CO_2 raise of up to 30%. As for hypertension, a decreased abdominal and chest-wall compliance due to pneumoperitoneum may increase the systemic venous and arterial resistance. [11] On the pharmacological side, volatile agents, in particular Halotane and Nitrous oxide, may raise ICP, whereas IV anesthetic agents generally decrease it, with the exception of Ketamine. Depolarizing muscle relaxants should be avoided because they may rise ICP through muscle fasciculation by increasing the venous pressure [12]. In cases where the ICP value is known to be already above 20 mmHg, CSF drain may be considered before surgery in symptomatic patients. In this regard, we propose to temporarily set in place a lumbar drain as explained in case 2. We preferred this strategy in order to both avoid sudden CSF withdrawals and to be able to monitor ICP throughout the intervention. Moreover, we advocate against the prophylactic use of Dexametasone or osmotic diuretics because of the chronic, non-edema-related ICP raise.

3.1.2. Overdrainage syndrome

Several patients undergoing BS have already been treated by means of different CSF diversion techniques because of concomitant raised ICP. Even though it has been reported as unsuccessful in ESS, BS has been reported to be able to revert some already treated IIH patients to shunt-free clinical course. [13,14] In this regard, the way BS exerts its effect on CSF diversion may be twofold: firstly, it could revert the aforementioned mechanisms responsible for IIH; secondly, the pressure gradient across the shunting may be raised because the abdominal pressure diminishes [15]. Hence, if the combination of these mechanisms prevails over the actual need for shunting, an overdrainage syndrome may occur. The clinical manifestation is usually with orthostatic headaches but may imply potential life-threatening conditions such as cerebellar tonsillar herniation with distorsion of the brainstem [16]. Overdrainage syndromes have been reported for both lumboperitoneal (LP) and ventriculoperitoneal (VP) shunts. Nevertheless, there have been reports of dramatic improvement of the overdrainage syndrome after shifting a LP shunt to a VP one, thus suggesting that pressure dynamics may act separately in relation to obesity and IIH [15]. Either case, we advocate for a frequent clinical and radiological follow-up evaluation in shunted patients undergoing BS.

3.1.3. CSF fistula

The effects of BS on ICP may be relevant also in patient without previous disturbances of ICP. In particular manifestations similar to a spontaneous intracranial hypotension syndrome (IHS) may occur, as reported in our first case. Franzini et al thoroughly analyzed the potential etiology of IHS: besides the supposed risk factors such as trivial trauma and congenital weakness of the dural sac, they interestingly pointed out the role of the reduction of pressure in the inferior vena cava, which in turn may increase the venous drainage from the epidural plexuses of Bateson fostering CSF outflow. [17] Specifically, this mechanism may also be responsible for the developing of a hypotension syndrome even without the neuroradiological evidence of a definite CSF leak. As a matter of fact, the negative gradient may justify an aspirating force acting diffusely on the dural surface an at the origin of spinal roots with a considerable outflow of CSF from the subarachnoid space to the radicular veins [17]. Interestingly, Schievink et al reported 11 patients out of a consecutive series of 338 HIS cases (3.3%) who underwent previous BS procedures [18]. 7 of them had a demonstrated CSF leak whereas 4 had multiple thoracic arachnoid cysts. When analyzing this relationship, three other speculative mechanism may be suggested besides the pressure gradient explanations. Firstly, the abrupt thinning of the epidural fat may withdraw a pre-existing epidural tamponade. Secondly, the pre-existing ICP raise may have somewhat thinned the dural layer, thus making it prone to ruptures e.g. in the skull base. Lastly, the post-BS frailty of the dural sac may be related both to macro- and micro- nutrient deficiencies and to structural changes of the dura, in particular those related to the collagen composition of its extracellular matrix [19].

3.2. Macro- and micro- nutrients deficiencies

Obesity is, counter-intuitively, a risk factor for malnutrition, as it has been reported that 20–30% of obese patients have in fact micronutrient deficiencies. [20] Moreover, in the post-BS period, vomiting, altered dietary patterns and bacterial overgrowth could lead to relevant nutrient deficiencies, the most common being vitamin B12, vitamin B9 and thiamine [21]. These deficiencies have been deemed responsible for a 5–10% incidence of sensorimotor deficits in the post-BS period [22]. The relative incidence has been estimated to consist of about 60% peripheral neuropathies, 30% encephalopathies and 8% optic nerve lesions [21]. Rarely, nutrient deficiencies have also been related to spinal disorders and potential fetal malformations or undergrowth.

3.2.1. Peripheral neuropathy

Peripheral neuropathy after BS is the most common neurological complication and may be characterized by different onset timing, neurologic deficit pattern and etiology. All the different peripheral damages to the nerves share a potential etiology in relative or absolute deficiency of one or more nutrient, which may explain the protean manifestation of the damage; the definition of the specific neurological implication of a given micronutrient deficiency is better reviewed elsewhere. [23,24] Moreover, peripheral nerves of morbidly obese patients may already be more prone to injury even before BS also because of concomitant metabolic diseases (i.e. diabetes) and mechanical stress [25]. Hence, BS may foster an immediate onset of post-operative neuropathy which is potentially related to patient positioning. In this regard, both brachial plexus and ulnar nerve have been commonly reported, especially in lengthy procedures, but there have also been reports of sciatic nerve damage because of gluteal compartment syndrome [26,27]. Interestingly, there have also been reports of sciatic neuropathy due to body contouring surgery after massive weight loss [28]. Moreover, Meralgia parestethica has also been related to both morbid obesity and immediate post-BS period [29]. The common etiology of the onset timing of these two conditions may reside in an impingement of the femoral cutaneous nerve due to pannicular traction at the inguinal ligament in a chronic fashion. This is mainly due to obesity and also to abdominal retractors or laparoscopic insufflation at a more acute stage.

According to the territories involved, sub-acute and late peripheral neuropathies have been classified as sensory-predominant polyneuropathy, mononeuropathy and radiculoplexus neuropathy; the lagest cohort studied has reported relative incidences of 55, 38 and 7% respectively. [30]

Sensory-predominant polyneuropathy is usually related to various micronutrient deficiencies and should be prevented and treated accordingly. [31] Notably, post BS neuropathic pain has multifactorial etiologies and has diverse manifestations ranging from a focal pain (e.g. burning feet syndrome) to a central component like in painful myelopathy. Its treatment should not then be based only on dietary supplements, it also has to rely on tailored pharmacological treatments taking into account the pharmacokinetic alterations after BS [32]. As for mononeuropathy, a dual mechanism has been proposed: firstly, a rapid weight loss may imply a relevant loss of subcutaneous tissue and protective fat pads, thus leading to structural changes that in their turn make the nerves more susceptible to compressions; secondly, metabolic changes including low albumin with low oncotic pressure or possibly intracellular formation of sorbitol from glucose causing osmotic stress may damage both myelin and neurons [33]. The most frequent mononeuropathies reported in literature are carpal tunnel syndrome and the aforementioned meralgia parestethica but there have also been several reports of peroneal neuropathy-related foot drop, which have resolved after simple supplementary therapy alone or in combination with focal decompression surgery [34–37]. Post-BS motor deficit related to Guillan-Barré syndrome and myopathies have also been reported, thus implying the fundamental role of a thorough electrophysiological study for a correct diagnosis [38,39].

3.2.2. Myelo-encephalopathy

There are many reports about the beneficial effects of BS on cognitive and motor performances. In particular, it has emerged that BS improves neural plasticity of the limbic system and prefrontal areas. [40–42] Sadly, not all the patients experience an improvement since some of them may develop neurological deficits. After the first series reported in 1987, several reports have been recently published on the topic [43–47]. A new term, i.e. acute post-gastric reduction surgery neuropathy (APGARS), has even been introduced to describe a syndrome characterized by a combination of vomiting, hypo / hyperreflexia, weakness, confusion psychosis, ophtalmoplegia and gait disturbances [48]. APGARS is most likely a combination of various neurological disorders related to deficiencies in micro-nutrients such as the aforementioned peripheral neuropathy, Wernicke-Korsakoff syndrome and posterolateral myelopathy. Wernicke-Korsakoff is an acute encephalopathy usually related to deficiency in vitamin B1, characterized by the triad ophthalmoplegia / nystagmus, motor ataxia, and confusion. Most cases have an onset within 6 months post BS, but it may be earlier in case of frequent vomiting [49]. Brain MRI could show hyperintense areas on T2 (periventricular zone, mammillary bodies, medial thalamus). Although most neurologic disorders enlisted are reversible with vitamin replacements, persistent residual neurologic symptoms are unfortunately common in patients with Wernicke-Korsakoff [50]. Posterolateral myelopathy, has instead been reported to occur an average of 9 years (range 0.5–18 years) after surgery [51]. Its clinical signs are a disabling gait ataxia with spastic legs with hyperreflexia, loss of proprioception and vibratory sensation. It has been mostly related to vitamin B12 and copper deficiencies and even in this case a spinal MRI could be helpful, whereas the CSF study has been reported as useless.[51] Since its onset may be late from BS, we advocate for a thorough anamnestic evaluation of patients affected by a myelopathy with no overt radiological signs of spinal cord compression at the imaging studies.

3.2.3. Neural tube defects

Pregnant and postpartum BS patients are at particular risk of micronutrient deficiencies leading to various potential fetal / neonatal complications. Dealing specifically with neurulation and neural tube growth, both folic acid, vitamin B12 and vitamin A are fundamental. Several cases of fetal neural malformations related to BS have been reported from the late '80 s. [52–56] Pre- and post-conception assessment of the various micro nutrient statuses should be recommended and constantly monitored in women of childbearing age [57–59].

3.2.4. Spine disorders

The effect of BS on the spine health of obese patients may be dual: it is generally beneficial on spine pain scores of patients but its effect on calcium metabolism triggers some warnings. [60–63] In particular, obese patients may have low levels of vitamin D even before surgery because of bad nutritional habits, sequestration in the adipous tissue and sedentary lifestyle. This deficit may worsen after surgery because of malabsorption and diminished intake leading to hypocalcemia and secondary hyperparathyroidism, thus weakening bony structures [64]. In a recent review, a trend of increased fracture risk in BS patients compared to controls at 24 months post-surgery (RR = 1.24) is reported [62].

3.3. Autonomic and Endocrine Control Alterations

The relationship between the obesity and CNS is characterized by a feedback mechanism so integrated and complex that obesity has been defined as a "chronic subcortical brain disease". [65] This integration is so deep-rooted that the thalamus / hypothalamus region has well defined relationships with food intake. Lesions in that area could lead to secondary obesity, especially in young patients, whereas the DBS modulation of ventromedial hypothalamus, lateral hypothalamic area and nucleus accumbens have already been tested for the treatment of morbid obesity [66,67]. On the other hand, BS alters the incretion of gut hormones which can, in turn, modulate autonomic nerve tone and its response to stimuli [68]. Eventually, the pituitary function is related to the systemic adipose deposits both in physiological and pathological conditions. Hence, an abrupt modification of these multidirectional feedbacks, as in a dramatic weight loss, could lead to some pathological alterations.

3.3.1. Orthostatic intolerance

Gut hormones such as ghrelin, gastrin, GLP-1, CCK insulin and leptin are released systemically and have been reported to exert their function on the vagus nerve too. [69] Their incretion is actually correlated to an exaggerated vagal tone which could be the cause of pathological vasovagal syndromes. Several cases of clinically relevant orthostatic hypotension have been reported recently, with an onset of symptoms varying from few months to years after [70–73]. A post-BS, autonomic neuropathy primarily affecting the lower thoracic and lumbar sympathetic neurons has also been proposed [74]. Although this hypothesis cannot be completely ruled out, it is unlikely that a neuropathy spares other districts focusing only on autonomic fibers. Instead, it is to be considered that most obese patients may have had metabolic syndromes with various degrees of insulin-resistance which could have led to a chronic microvascular neuropathy. Even a small amount of vagal tone variation may thus exert a relevant clinical effect on an already challenged autonomic system. A neurosurgical differential diagnosis of syncope in BS patients must include potential cerebrovascular accidents, seizures or derangement of the CSF dynamics which however tends to crop up at an earlier post-operative stage [75].

3.3.2. Pituitary gland alterations

Besides the empty sella syndrome already discussed previously, it is well-known that some endocrinological disorders in the pituitary region may be related to obesity. This raises various concerns: the potential misdiagnosis of the obesity cause, the role of BS on pre-existing hormonal deficiency and the potential influence of BS on the absorption of the hormone therapy replacement therapy. As for the cause of obesity, a study on 783 obese patients undergoing BS showed the prevalence of pituitary disease in 1.9% of the patients and specifically a prevalence of Cushing's disease of 0,8%, greatly higher than the figures for the general population. [76] Since the presence of such dysfunctions may require specific therapies other than BS which, in turn, may be even contraindicated, we advocate for a careful endocrinological evaluation of all the patients undergoing BS. In particular, as far as Cushing's syndrome is concerned, at least an overnight 1 mg dexamethasone test should be recommended to avoid unnecessary treatments [77,78]. Endocrinological follow-up evaluations should also be planned since already reported recurrences of Cushing's syndrome after both pituitary and BS have already reported [79]. On the other hand, the role of BS on pre-existing hormonal deficiencies should be evaluated on the basis of the interactions between the adipose tissue and the pituitary gland. As for GH incretion, many factors have to be taken into account. Firstly, GH secretion drops by up to 6% per each unit increase in BMI. Moreover, obesity-related conditions such as sleep apnea syndrome and type-2 diabetes notably infer the GH-IGF-1 axis. The abrupt resolution of the obesity may, then, exert a function in augmenting GH secretion as reported in an acromegalic patient whose GH values showed a constant increase in the post-operative course after BS [80]. Similarly, the secretion rates of the gonadotropin hormone show augmented incretion values after BS, whereas TSH values do not seem to be relevantly altered by BS [81,82]. Dealing with the concern of an altered absorption of hormone replacement therapy in BS patients, recent reports excluded any significant impairment [83,84].

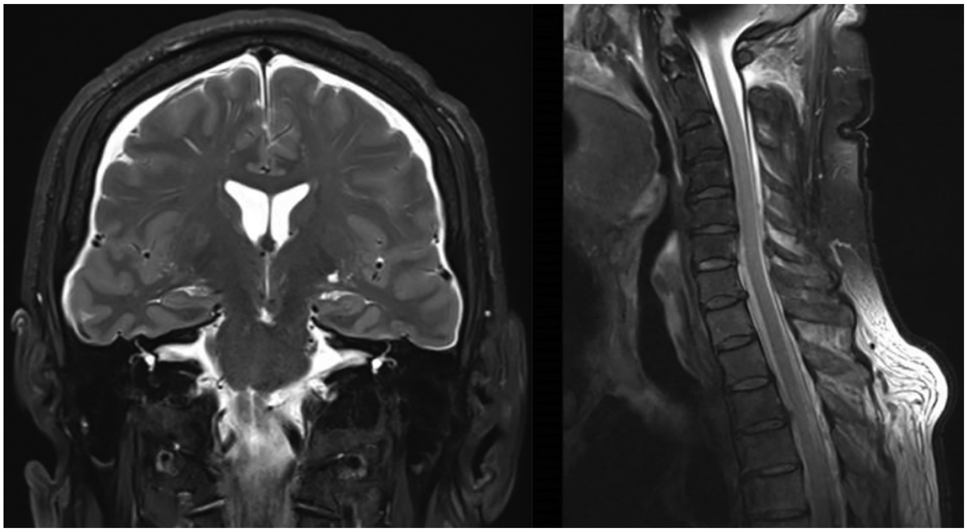

Fig. 1. MRI T2 scans showing thin fronto-parietal, bilateral chronic subdural hematomas and detachment of the dura from the bony margins of the cervical spinal canal with hyperintensity signal in the interspinous soft tissues as of CSF leakage.

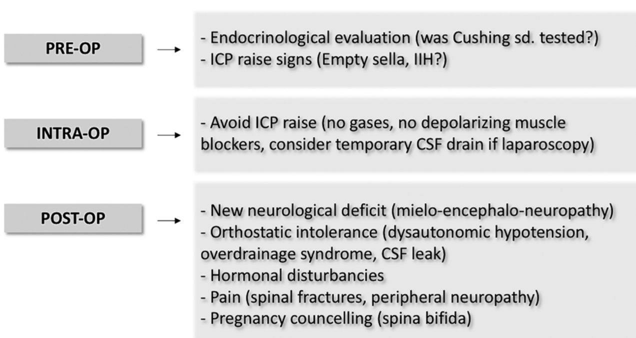

Fig. 2. Neurosurgical issues of BS divided in relation to the surgical timing.

Case 1. The patient was a 57-year-old male who underwent gastric bypass surgery 2 years before losing a total of 96 kg (height: 1.84 m, BMI = from 63.2 to 34.9). During the week prior admission, he complained of orthostatic cephalalgia, no other focal neurological signs were present and he reported no traumas. CT and MRI scans showed very thin fronto-parietal, bilateral chronic subdural hematomas. A whole-spine MRI study showed a detachment of the dura from the bony margins of the spinal canal, associated to a diffuse T2 hyperintensity signal in the interspinous soft tissues as of CSF leakage (Fig. 1). A diagnosis of spontaneous cervical CSF fistula with related intracranial hypotension syndrome was made. A blood patch procedure was planned but the symptoms disappeared after 1 week of forced clinostatism and sustained hydration. Cephalalgia was never reported at follow-up evaluations and subdural hematomas spontaneously reabsorbed.

Case 2. The patient was a 33-year-old woman whose BMI was of 52, so a videolaparoscopic sleeve gastrectomy procedure was proposed. Her intracranial pressure was of 29.4 mmHg (40 cmH_2O) with no clinical response to an Acetazolomide treatment (1250 mg per day). At the time of surgery, we collegially decided to set a lumbar external drain with continuous ICP measurement. Specifically, after the system was set in place, 30 ml of CSF were drained and ICP dropped to 26 mmHg. We then set the height of the device to allow little-to-no CSF drain. The insufflation of the peritoneal cavity was performed at a low pressure (10 instead of 15 mmHg) and we registered the drain of further 15 ml of CSF throughout the intervention. The patient underwent an uneventful awakening and the drain was removed immediately after surgery. At the last follow-up (24 months) the patient lost a total of 83 kg (1.58 m, BMI = from 52.1–29.9) and her neurological symptoms greatly improved.

4. Conclusions

BS is an elective procedure which implies complex pre-operative evaluations, careful intraoperative managing and a long-lasting follow-up based on outpatient visits (Fig. 2). Neurosurgeons must be aware of the several, multifactorial, neurosurgery-related complications of BS since their prevalence is likely to be higher in the next years. In the neurosurgical practice, many of these complications may act as confounders in the diagnosis of other, more frequent, neurosurgical pathologies e.g. CSF pressure alterations, myelo-radiculopathies or pituitary alterations.

Delay in the diagnosis of some of these complications may worsen their long-term sequelae. A multidisciplinary team management is advised, providing careful neurological and nutritional monitoring at regular intervals.

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None

References

[1] WHO, Obesity and overweight, Key facts (http://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight [Accessed September 11, 2018].

[2] American College of Cardiology/American Heart Association Task Force on Practice Guidelines, Obesity Expert Panel, Executive summary: guidelines (2013) for the management of overweight and obesity in adults: a report of the american college of Cardiology/American heart association task force on practice guidelines and the obesity society published by the obesity society and american college of Cardiology/American heart association task force on practice guidelines. Based on a systematic review from the the obesity expert panel, 2013, Obesity Silver Spring (Silver Spring) 22 (Suppl. 2) (2013) S5-39, 2014.

[3] C.W. le Roux, H.M. Heneghan, Bariatric surgery for obesity, Med. Clin. North Am. 102 (1) (2018) 165–182.

[4] H.H. Dasenbrock, M.O. Nguyen, K.U. Frerichs, D. Guttieres, W.B. Gormley, M. Ali Aziz-Sultan, et al., The impact of body habitus on outcomes after aneurysmal subarachnoid hemorrhage: a Nationwide Inpatient Sample analysis, J. Neurosurg. 127 (1) (2018) 36–46.

[5] J.D. Hughes, M. Samarage, A.M. Burrows, G. Lanzino, A.A. Rabinstein, Body mass index and aneurysmal subarachnoid hemorrhage: decreasing mortality with increasing body mass index, World Neurosurg. 84 (6) (2018) 1598–1604.

[6] K.E. Schultheiss, Y.G. Jang, R.N. Yanowitch, J. Tolentino, D.J. Curry, J. Lüders, F. Asgarzadie-Gadim, R.L. Macdonald, Fat and neurosurgery: does obesity affect outcome after intracranial surgery? Neurosurgery 64 (2) (2009) 316–326.

[7] A.V. Kalyvas, M. Hughes, C. Koutsarnakis, D. Moris, F. Liakos, D.E. Sakas, et al., Efficacy, complications and cost of surgical interventions for idiopathic intracranial hypertension: a systematic review of the literature, Acta Neurochir. (Wien) 159 (1) (2017) 33–49.
[8] J.H. Manfield, K.K. Yu, E. Efthimiou, A. Darzi, T. Athanasiou, H. Ashrafian, Bariatric surgery or non-surgical weight loss for idiopathic intracranial hypertension? A systematic review and comparison of meta-analyses, Obes. Surg. 27 (2) (2017) 513–521.
[9] J. Fridley, R. Foroozan, V. Sherman, M.L. Brandt, D. Yoshor, Bariatric surgery for the treatment of idiopathic intracranial hypertension, J. Neurosurg. 114 (1) (2011) 34–39.
[10] J.E. Cottrell, Patel P. Cottrell and Patel's Neuroanesthesia, sixth edition, Elsevier, 2016.
[11] D.E. Longnecker, M.F. Newman, W.M. Zapol, W. Sandberg, S. Mackey, Anesthesiology, third edition, McGraw Hill Professional, 2017.
[12] T.H. Kamine, N.Y. Elmadhun, E.M. Kasper, E. Papavassiliou, B.E. Schneider, Abdominal insufflation for laparoscopy increases intracranial and intrathoracic pressure in human subjects, Surg. Endosc. 30 (9) (2016) 4029–4032.
[13] Q.G. D'Alessandris, N. Montano, F. Bianchi, F. Doglietto, E. Fernandez, R. Pallini, et al., Persistence of primary empty sella syndrome despite obesity surgery: report of two unusual cases, Br. J. Neurosurg. 26 (6) (2016) 875–876.
[14] K.B. Hoang, K.G. Hooten, C.R. Muh, Shunt freedom and clinical resolution of idiopathic intracranial hypertension after bariatric surgery in the pediatric population: report of 3 cases, J. Neurosurg. Pediatr. 20 (6) (2016) 511–516.
[15] J. Roth, S. Constantini, A. Kesler, Over-drainage and persistent shunt-dependency in patients with idiopathic intracranial hypertension treated with shunts and bariatric surgery, Surg. Neurol. Int. 6 (Suppl. 27) (2015) S655–60.
[16] J.J. Graber, R. Racela, K. Henry, Cerebellar tonsillar herniation after weight loss in a patient with idiopathic intracranial hypertension, Headache 50 (1) (2010) 146–148.
[17] A. Franzini, G. Messina, V. Nazzi, E. Mea, M. Leone, L. Chiapparini, et al., Spontaneous intracranial hypotension syndrome: a novel speculative physiopathological hypothesis and a novel patch method in a series of 28 consecutive patients, J. Neurosurg. 112 (2) (2010) 300–306.
[18] W.I. Schievink, A. Goseland, S. Cunneen, Bariatric surgery as a possible risk factor for spontaneous intracranial hypotension, Neurology 83 (20) (2014) 1819–1822 11.
[19] I.H. Prist, A.G. Salles, T.M. de Lima, M.L. Modolin, R. Gemperli, H.P. Souza, Extracellular matrix remodeling derangement in ex-obese patients, Mol. Cell. Biochem. 425 (1-2) (2017) 1–7.
[20] D.J. Frantz, Neurologic complications of bariatric surgery: involvement of central, peripheral, and enteric nervous systems, Curr. Gastroenterol. Rep. 14 (4) (2012) 367–372.
[21] B.M. Koffman, L.J. Greenfield, I.I. Ali, N.A. Pirzada, Neurologic complications after surgery for obesity, Muscle Nerve 33 (2) (2006) 166–176.
[22] J.R. Berger, The neurological complications of bariatric surgery, Arch. Neurol. 61 (8) (2004) 1185–1189.
[23] N. Kumar, Neurologic presentations of nutritional deficiencies, Neurol. Clin. 28 (1) (2010) 107–170.
[24] A. Kazemi, T. Frazier, M. Cave, Micronutrient-related neurologic complications following bariatric surgery, Curr. Gastroenterol. Rep. 12 (4) (2010) 288–295.
[25] A. O'Connor, B. McNamara, B.J. Sweeney, Diabetic lumbosacral radiculoplexus neuropathy after bariatric surgery, Surg. Obes. Relat. Dis. 13 (2) (2017) e9–e11.
[26] J.J. Rodríguez Uranga, A.J. Uclès Sánchez, J.M. Pérez Díaz, Neuropathy of common sciatic nerve secondary to compartment syndrome as a complication after bariatric surgery, Neurologia 20 (2) (2005) 94–97.
[27] M.L. Benevides, R.J. Nochi Júnior, Rhabdomyolysis secondary to gluteal compartment syndrome after bariatric surgery: case report, Rev. Bras. Anestesiol. 56 (4) (2006) 408–412.
[28] D. Kiermeir, A. Banic, K. Rösler, D. Erni, Sciatic neuropathy after body contouring surgery in massive weight loss patients, J. Plast. Reconstr. Aesthet. Surg. 63 (5) (2010) e454–7.
[29] A.M. Macgregor, E.K. Thoburn, Meralgia paresthetica following bariatric surgery, Obes. Surg. 9 (4) (1999) 364–368.
[30] P. Thaisetthawatkul, M.L. Collazo-Clavell, M.G. Sarr, J.E. Norell, P.J. Dyck, A controlled study of peripheral neuropathy after bariatric surgery, Neurology 63 (8) (2004) 1462–1470 26.
[31] S.A. Rudnicki, Prevention and treatment of peripheral neuropathy after bariatric surgery, Curr. Treat. Options Neurol. 12 (1) (2010) 29–36.
[32] V. Kattalai Kailasam, C. DeCastro, C. Macaluso, A. Kleiman, Postbariatric surgery neuropathic pain (PBSNP): case report, literature review, and treatment options, Pain Med. 16 (2) (2015) 374–382.
[33] F.J. Weyns, F. Beckers, L. Vanormelingen, M. Vandersteen, E. Niville, Foot drop as a complication of weight loss after bariatric surgery: is it preventable? Obes. Surg. 17 (9) (2015) 1209–1212.
[34] M.S. Menezes, K.O. Harada, G. Alvarez, Painful peripheral polyneuropathy after bariatric surgery. Case reports, Rev. Bras. Anestesiol. 58 (3) (2008) 252–259.
[35] E.T. Aasheim, D. Hofsø, J. Hjelmesaeth, R. Sandbu, Peripheral neuropathy and severe malnutrition following duodenal switch, Obes. Surg. 18 (12) (2008) 1640–1643.
[36] W.J. Elias, N. Pouratian, R.J. Oskouian, B. Schirmer, T. Burns, Peroneal neuropathy following successful bariatric surgery. Case report and review of the literature, J. Neurosurg. 105 (4) (2006) 631–635.
[37] J. Hamel, E.L. Logigian, Acute nutritional axonal neuropathy, Muscle Nerve 57 (1) (2018) 33–39.
[38] N. Ishaque, B.A. Khealani, A.H. Shariff, M. Wasay, Guillain-Barré syndrome (demyelinating) six weeks after bariatricsurgery: a case report and literature review, Obes. Res. Clin. Pract. 9 (4) (2015) 416–419.
[39] A.F. Landais, Rare neurologic complication of bariatric surgery: acute motor axonal neuropathy (AMAN), a severe motor axonal form of the Guillain Barré syndrome, Surg. Obes. Relat. Dis. 10 (6) (2014) e85-7.
[40] H. Eickhoff, Central modulation of energy homeostasis and cognitive performance after bariatric surgery, Adv. Neurobiol. 19 (2017) 213–236.
[41] M. Rullmann, S. Preusser, S. Poppitz, S. Heba, J. Hoyer, T. Schütz, et al., Gastric-bypass surgery induced widespread neural plasticity of the obese human brain, Neuroimage 15 (172) (2018) 853–863.
[42] A.L. Pearce, E. Mackey, J.B.C. Cherry, A. Olson, X. You, S.N. Magge, et al., Effect of adolescent bariatric surgery on the brain and cognition: a pilot study, Obesity Silver Spring (Silver Spring) 25 (11) (2017) 1852–1860.
[43] J.M. Abarbanel, V.M. Berginer, A. Osimani, H. Solomon, I. Charuzi, Neurologic complications after gastric restriction surgery for morbid obesity, Neurology 37 (2) (1987) 196–200.
[44] M. Tabbara, S. Carandina, M. Bossi, C. Polliand, L. Genser, C. Barrat, Rare neurological complications after sleeve gastrectomy, Obes. Surg. 26 (12) (2016) 2843–2848.
[45] A. Landais, Neurological complications of bariatric surgery, Obes. Surg. 24 (10) (2014) 1800–1807.
[46] D.A. Becker, L.J. Balcer, S.L. Galetta, The neurological complications of nutritional deficiency following bariatric surgery, J Obes 2012 (2012) 608534.
[47] J.C. Goodman, Neurological complications of bariatric surgery, Curr. Neurol. Neurosci. Rep. 15 (12) (2015) 79.
[48] C.G. Chang, B. Adams-Huet, D.A. Provost, Acute post-gastric reduction surgery (APGARS) neuropathy, Obes. Surg. 14 (2) (2004) 182–189.
[49] F. Pardo-Aranda, N. Perez-Romero, J. Osorio, J. Rodriguez-Santiago, E. Muñoz, N. Puértolas, et al., Wernicke's encephalopathy after sleeve gastrectomy: literature review, Int. J. Surg. Case Rep. 20 (2016) 92–95.
[50] S. Punchai, Z.N. Hanipah, K.M. Meister, P.R. Schauer, S.A. Brethauer, A. Aminian, Neurologic manifestations of vitamin B deficiency after bariatric surgery, Obes. Surg. 27 (8) (2017) 2079–2082.
[51] K. Juhasz-Pocsine, S.A. Rudnicki, R.L. Archer, S.I. Harik, Neurologic complications of gastric bypass surgery for morbid obesity, Neurology 68 (21) (2007) 1843–1850 22.
[52] J.E. Haddow, L.E. Hill, E.M. Kloza, D. Thanhauser, Neural tube defects after gastric bypass, Lancet 1 (8493) (1986) 1330 7.
[53] L. Martin, G.F. Chavez, M.J. Adams Jr, E.E. Mason, J.W. Hanson, J.E. Haddow, et al., Gastric bypass surgery as maternal risk factor for neural tube defects, Lancet 1 (8586) (1988) 640–641 19.
[54] J.A. Moliterno, Luna M.L. Di, S. Sood, K.E. Roberts, C.C. Duncan, Gastric bypass: a risk factor for neural tube defects? Case report, J. Neurosurg. Pediatr. 1 (5) (2008) 406–409.
[55] G. Pelizzo, G. Nakib, A. Alfei, A. Lasci, H. Cena, D. Locatelli, et al., Fetal neural tube defects in pregnant women previously submitted to bariatric surgery: more attention to a new emerging entity, Prenat. Diagn. 33 (2013) 196–197.
[56] G. Pelizzo, V. Calcaterra, M. Fusillo, G. Nakib, A.M. Ierullo, A. Alfei, et al., Malnutrition in pregnancy following bariatric surgery: three clinical cases of fetal neural defects, Nutr. J. 14 (13) (2014) 59.
[57] L. Kaska, J. Kobiela, A. Abacjew-Chmylko, L. Chmylko, M. Wojanowska-Pindel, P. Kobiela, et al., Nutrition and pregnancy after bariatric surgery, ISRN Obes. 30 (2013) 492060 2013.
[58] C.B. Chagas, C. Saunders, S. Pereira, J. Silva, C. Saboya, A. Ramalho, Vitamin a deficiency in pregnancy: perspectives after bariatric surgery, Obes. Surg. 23 (2) (2013) 249–254.
[59] M. Medeiros, C. Saunders, C.B. Chagas, S.E. Pereira, C. Saboya, A. Ramalho, Vitamin D deficiency in pregnancy after bariatric surgery, Obes. Surg. 23 (10) (2013) 1679–1684.
[60] N.E. Epstein, Bariatric bypasses contribute to loss of bone mineral density but reduce axial back pain in morbidly obese patients considering spine surgery, Surg. Neurol. Int. 19 (8) (2017) 13.
[61] T. Çakır, M.T. Oruç, A. Aslaner, F. Duygun, E.C. Yardımcı, B. Mayir, et al., The effects of laparoscopic sleeve gastrectomy on head, neck, shoulder, low back and knee pain of female patients, Int. J. Clin. Exp. Med. 8 (2) (2015) 2668–2673 15.
[62] A. Kalani, H. Bami, M. Tiboni, R. Jaeschke, J.D. Adachi, A.N. Lau, The effect of bariatric surgery on serum 25-OH vitamin D levels: a systematic review and meta-analysis, Obes. Sci. Pract. 3 (3) (2017) 319–332 27.
[63] Z. Lidar, E. Behrbalk, G.J. Regev, K. Salame, O. Keynan, C. Schweiger, et al., Intervertebral disc height changes after weight reduction in morbidly obese patients and its effect on quality of life and radicular and low back pain, Spine (Phila Pa 1976) 37 (23) (2012) 1947–1952 1.
[64] S. Panda, K. Sharma, Osteomalacia induced peripheral neuropathy after obesity reduction surgery, Ann. Indian Acad. Neurol. 16 (4) (2013) 690–692.
[65] P. Sinclair, N. Docherty, C.W. le Roux, Metabolic effects of bariatric surgery, Clin. Chem. 64 (1) (2018) 72–81.
[66] K.W. Wang, M. Valencia, L. Banfield, R. Chau, A. Fleming, S.K. Singh, et al., The effectiveness of interventions to treat obesity in survivors of childhood brain tumors: a systematic review protocol, Syst. Rev. 5 (1) (2016) 101 14.
[67] D.A. Dupré, N. Tomycz, M.Y. Oh, D. Whiting, Deep brain stimulation for obesity: past, present, and future targets, Neurosurg. Focus 38 (6) (2015) E7.
[68] C.N. Ochner, C. Gibson, M. Shanik, V. Goel, A. Geliebter, Changes in neurohormonal gut peptides following bariatric surgery, Int. J. Obes. (Lond) 35 (2) (2011) 153–166.
[69] E. Valassi, M. Scacchi, F. Cavagnini, Neuroendocrine control of food intake, Nutr. Metab. Cardiovasc. Dis. 18 (2) (2008) 158–168.
[70] R. Rubinshtein, M. Ciubotaru, H. Elad, H. Bitterman, Severe orthostatic hypotension

following weight reduction surgery, Arch. Intern. Med. 161 (17) (2001) 2145–2147.
[71] S.R. Billakanty, M.D. Kligman, Y.M. Kanjwal, D.J. Kosinski, G.T. Maly, B. Karabin, et al., New-onset orthostatic intolerance following bariatric surgery, Pacing Clin. Electrophysiol. 31 (7) (2008) 884–888.
[72] V. Ponnusamy, A.P. Owens, S. Purkayastha, V. Iodice, C.J. Mathias, Orthostatic intolerance and autonomic dysfunction following bariatric surgery: a retrospective study and review of the literature, Auton. Neurosci. 198 (2016) 1–7.
[73] K.P. Loh, O. Ogunneye, Malignant cardioinhibitory vasovagal syncope - an uncommon cardiovascular complication of Roux-en-Y gastric bypass surgery: the fainting syndrome!, Int J Cardiol 15 164 (3) (2013) e38-9.
[74] R.D. Hoeldtke, G.E. Dworkin, S.R. Gaspar, B.C. Israel, Sympathotonic orthostatic hypotension: a report of four cases, Neurology 39 (1) (1989) 34–40.
[75] J.Y. Choi, T.K. Scarborough, Stroke and seizure following a recent laparoscopic Roux-en-Y gastric bypass, Obes. Surg. 14 (6) (2004) 857–860.
[76] P. Fierabracci, A. Pinchera, S. Martinelli, G. Scartabelli, G. Salvetti, M. Giannetti, et al., Prevalence of endocrine diseases in morbidly obese patients scheduled for bariatric surgery: beyond diabetes, Obes. Surg. 21 (1) (2011) 54–60.
[77] M. Fleseriu, W.H. Ludlam, S.H. Teh, C.G. Yedinak, C. Deveney, B.C. Sheppard, Cushing's syndrome might be underappreciated in patients seeking bariatric surgery: a plea for screening, Surg. Obes. Relat. Dis. 5 (1) (2009) 116–119.
[78] L. Borsoi, B. Ludvik, G. Prager, A. Luger, M. Riedl, Cushing's syndrome in a morbidly obese patient undergoing evaluation before bariatric surgery, Obes. Facts 7 (3) (2014) 191–196.
[79] B.R. Javorsky, T.B. Carroll, N.A. Tritos, R. Salvatori, A.P. Heaney, M. Fleseriu, et al., Discovery of Cushing's syndrome after bariatric surgery: multicenter series of 16 patients, Obes. Surg. 25 (12) (2015) 2306–2313.
[80] A. Lecube, R. Vilallonga, G. Sturniolo, G. Obiols, J.M. Fort, Renaissance of acromegaly after bariatric surgery, Endocrine 43 (1) (2013) 239–241.
[81] A. Rosenblatt, J. Faintuch, I. Cecconello, Abnormalities of reproductive function in male obesity before and after bariatric Surgery-A comprehensive review, Obes. Surg. 25 (7) (2015) 1281–1292.
[82] B. Guan, Y. Chen, J. Yang, W. Yang, C. Wang, Effect of bariatric surgery on thyroid function in obese patients: a systematic review and meta-analysis, Obes. Surg. 27 (12) (2017) 3292–3305.
[83] P. Wolf, Y. Winhofer, S. Smajis, R. Kruschitz, K. Schindler, A. Gessl, et al., Hormone substitution after gastric bypass surgery in patients with hypopituitarism secondary to craniopharyngioma, Endocr. Pract. 22 (5) (2016) 595–601.
[84] M. Trotta, J. Da Broi, A. Salerno, R.M. Testa, G.M. Marinari, Sleeve gastrectomy leads to easy management of hormone replacement therapy and good weight loss in patients treated for craniopharyngioma, Updates Surg. 69 (1) (2017) 95–99.