Rashad et al. The Egyptian Journal of Neurology, Psychiatry and Neurosurgery (2019) 55:22
https://doi.org/10.1186/s41983-019-0064-0

The Egyptian Journal of Neurology, Psychiatry and Neurosurgery

RESEARCH Open Access

Post-bariatric surgery peripheral neuropathies: Kuwaiti experience

Hanaa M. Rashad[1,2], Doaa Youssry[1,3], Dina F. Mansour[4,5], Ayman Kilany[1,6*], Jasem Y. Al-Hashel[1,7], Adnan J. Khuraibet[1], Walaa A. Kamel[1,8] and Rossen T. Rousseff[1]

Abstract

Background: Obesity is a major global health problem. Kuwait has a very high prevalence of obesity, and consequently, the number of bariatric surgeries is rising.

Objectives: The aim of this study is to analyze the clinical presentation and electrodiagnostic features of peripheral nerve complications following bariatric surgery.

Subjects and methods: We retrospectively involved a convenience sample of patients presenting at a tertiary referral center and analyzed the patterns and frequency of peripheral nerve involvement, correlations with operative techniques, perioperative complications, nutritional status, possible risk factors, and functional impairment.

Results: Among the 58 cases, 23 presented with chronic distal symmetrical sensorimotor neuropathy, 10 suffered from small fiber neuropathy, 22 had mononeuropathies, 2 patients had acute axonal sensorimotor neuropathy, and only 1 patient had lumbar plexopathy. In 22 patients, we observed mononeuropathies (10 cases of carpal tunnel syndrome, 7 cases of peroneal compression at the knee, 4 cases of ulnar neuropathies at the elbow, and 1 case of meralgia paresthetica). Rapid weight loss and protracted postoperative vomiting tended to correlate with generalized neuropathies, while focal compression with loss of the protective subcutaneous tissue pad was associated with mononeuropathies. All patients suffered from a deficiency of at least 1 micronutrient. Compliance with supplementary therapy was poor. Some post-bariatric neuropathies interfere severely with patients' functional status.

Conclusion: Prevention by close follow-up, nutritional intervention, and patient education to avoid habitual postures related to nerve compression is appropriate.

Keywords: Peripheral neuropathy, Neuromuscular complications, Bariatric surgery, Weight loss

Introduction

Obesity has reached epidemic proportions in recent decades, becoming a major global health problem through its negative impact on morbidity, mortality, quality of life, and related healthcare costs [1, 2]. Its prevalence in the Arabian Gulf countries is very high. Kuwait is reportedly the fourth "fattest" country in the world with prevalence rates of obesity up to 45.3% [3].

Bariatric surgery (BS) has proven effective for sustained weight loss and superior to non-surgical obesity treatments [4, 5]. It reduces food intake, alters the route of food absorption, and modulates metabolism [6]. BS is recommended in morbidly obese patients by national and international guidelines [5]. The number of bariatric procedures has soared over the last decade; in Kuwait, 6682 bariatric surgeries were performed in 2012, which is the highest number as the percentage of the population in the world [7].

BS procedures have been perfected and are associated with low morbidity and mortality [8]. Still, BS is not free from complications caused by malnutrition, micronutrient deficiencies, and loss of the protective fat tissue pad [9, 10]. They may involve any part of the nervous system, including the peripheral division [10, 11]. The aim of our study is to analyze peripheral neuropathies complicating BS in a cohort of Kuwaiti patients.

* Correspondence: elkilany7000@yahoo.com
[1]Neurology Department, Ibn Sina Hospital, Kuwait City, Kuwait
[6]National Research Centre, Children with Special Needs, Cairo, Egypt
Full list of author information is available at the end of the article

Subjects and methods

We performed a retrospective analysis of a convenience series of 58 post-bariatric surgery patients who were referred between May 2014 and April 2015 with a clinical diagnosis of peripheral nerve disorder to the neurophysiology unit of a tertiary care hospital in Kuwait.

An informed written formal consent to participate in the study was obtained from every patient. Patients with a history predating their surgery and patients referred for generalized neuropathy, presenting with a well-documented cause of peripheral neuropathy (as long-term diabetes, alcoholism, and toxin exposure), were not included in the study.

Data were anonymously extracted from patients' records, including demographic data: history directed at the amount and rate of weight loss, gastrointestinal disturbances after surgery (vomiting, abdominal pain), compliance with nutritional supplement intake, and specific complaints related to peripheral nerve involvement. Results of detailed neurological examination, results of comprehensive motor and sensory nerve conduction study (NCS), sympathetic skin responses, and blood levels of vitamins were tabulated. Thiamine was assessed by measuring erythrocyte transketolase (thiamine deficiency is defined as blood thiamine lower than 74 nmol/l), serum level of vitamin B_{12} (B_{12} deficiency is defined as serum B_{12} lower than 150 pg/ml), folate (folate deficiency is defined as serum folic acid lower than 1.8 ng/ml), vitamin D (vitamin D deficiency is defined as serum level of 25-hydroxycholecalciferol lower than 20 ng/ml), and copper were investigated. EMG was carried out whenever the clinical presentation is that of a muscle disease or to detect radicular affection as most of the cases with root affection have normal motor and sensory NCS especially early in the course of the disease. CTS was diagnosed according to the guidelines and practice parameters by Jablecki and colleagues [12].

The functional status of the patients was assessed using the modified Rankin Scale for Neurologic Disabilities [13]. While such a scale is not designed specifically for use in peripheral nerve pathology, it provides an excellent tool for overall disability and we considered it best for our non-homogeneous patient cohort.

NCS (using Deymed, TrueTrace, Czech Republic) were performed by the same qualified technologists under the supervision of a clinical neurophysiology specialist according to standard techniques [14]. The median, ulnar, peroneal, tibial, and sural nerves were routinely studied, with temperature maintained over 33 °C in the hands and over 30 °C at the malleoli. Where necessary, needle EMG was performed.

The final diagnosis rested on the consensus between a neurologist and a clinical neurophysiologist among the authors after discussing the clinical, laboratory, and electrodiagnostic findings in each case.

The study was descriptive and approved by the institutional ethical committee. Statistical analysis was performed using Statgraphics© Online. Descriptive statistics, ANOVA for continuous data, and Fisher exact probability test for non-parametric variables, at accepted level of significance $P < 0.05$.

Results

Our series included 58 patients, 33 (56.8 %) were female and 25 (43.2%) were male. Their age ranged from 19 to 52 years (median age of 32 years). The weight loss ranged from 25 to 93 kg (mean value 47.8 ± 17.2 kg) over a period from 3 to 60 months (median period of 16 months). The onset of symptoms was between 4 and 52 months after BS (median interval of 20 months). The pattern of peripheral nervous system affection included 35 patients with generalized neuropathy, 22 patients with mononeuropathy, and 1 patient with lumbar plexopathy. The nosological distribution, demographic profile, and details on the pattern of weight loss are presented in Table 1.

Weight loss had a tendency to be more rapid in generalized neuropathy cases, but this was not statistically significant. The majority of patients with generalized neuropathy presented with chronic distal symmetrical sensory predominant neuropathy (23 cases). Sensory complaints (pain, paraesthesia) followed by objective sensory loss, areflexia, and gradually progressive weakness were typical in this group, while weakness in the feet and legs as an initial symptom was reported only in 3 patients.

At the time of diagnosis, only 7 patients had significant weakness in the lower leg and 7 had preserved reflexes. Only 3 patients had sensory symptoms in the arms.

Electrodiagnostic assessment confirmed distal axonal neuropathy in all patients. In 10 patients, we observed isolated small fiber neuropathy ("burning feet syndrome") without objective findings on clinical examination and routine NCS; in 6 of them, the sympathetic skin responses were absent.

Two cases of acute sensorimotor neuropathy with weakness involving also the thighs and upper limbs were observed in a 40-year-old patient (6 months after the surgery, prominent vomiting, drastic weight loss of 60 kg over 6 months, and poor compliance with prescribed nutritional supplementation). The other case was 24-year-old female patient came with a picture of Wernicke encephalopathy associated with sensorimotor neuropathy 8 months after the surgery, and she did not receive any nutritional supplement after the operation; also, she developed persistent vomiting due to gastric issue.

Neurophysiology was compatible with generalized axonal motor-sensory neuropathy. These two patients recovered gradually and incompletely. Among mononeuropathies, cases of carpal tunnel syndrome (CTS) had mostly insidious (five patients) or subacute (five patients) course.

Table 1 Distribution of final diagnosis and their relation to demographic data and weight loss characteristics

Variables	Mononeuropathy, *N* = 22 (37.5%)	Polyneuropathy, *N* = 35 (60.4%)	*P**
Continuous data			
Age (years)	Mean ± SD, 33 ± 9.5 Range, 22–50	Mean ± SD, 32.7 ± 7 Range, 19–52	0.90
Amount of weight loss (kg)	Mean ± SD, 48.4 ± 16.3 Range, 25–91	Mean ± SD, 48.7 ± 16.0 Range, 25–93	0.91
Time to maximal weight loss (months)	Mean ± SD, 20.2 ± 16.5 Range, 6–60	Mean ± SD, 14.0 ± 9.2 Range, 4–36	0.09
Time to disease onset (months)	Mean ± SD, 25.8 ± 20.1 Range, 3–25	Mean ± SD, 23.4 ± 15.3 Range, 4–52	0.64
Non-parametric data			
Gender, female	*N* = 22 Percent = 54.5%	*N* = 35 Percent = 57.7%	1.0
Postoperative complications	*N* = 22 Yes = 2 Percent = 9.1%	*N* = 35 Yes = 5 Percent = 14.2%	0.6
Prolonged vomiting	*N* = 22 Yes = 3 Percent = 13.5%	*N* = 35 Yes = 11 Percent = 31.0%	0.2

A single patient with lumbosacral radiculoplexus neuropathy is not included in this table
*ANOVA for means for continuous variables; Fisher contingency tables for non-parametric

Patients with ulnar neuropathy at the elbow presented subacutely. Cases of peroneal compression and meralgia paraesthetica presented with acute onset of the corresponding individual peripheral nerve syndrome.

The patient with lumbar plexopathy had a typical unilateral presentation with severe pain in the thigh and hip, sensory loss along the anterior aspect of the thigh and shin, wasting in the thigh muscles, lost patellar reflex, and widespread denervation on needle EMG study.

The nutritional status of the patients was unsatisfactory. By history, only 12 patients (20.6%) were compliant with nutritional supplement intake, while 29 (50%) took it rarely and another 17 (29.3%) neglected treatment at all. Vitamin D deficiency (defined as serum level of 25-hydroxycholecalciferol lower than 20 ng/ml) was present in 40 patients (69%), thiamine deficiency (defined as blood thiamine lower than 74 nmol/l) in 32 (55%), vitamin B_{12} deficiency (defined as serum B_{12} lower than 150 pg/ml) in 22 (38%), and folate deficiency (defined as serum level below 1.8 ng/ml) in 12 (20.6%). Copper level was normal in all screened patients. The distribution of patients by diagnoses compared with nutritional status is presented in Table 2. No significant difference between the polyneuropathy and mononeuropathy patients regarding their nutrition compliance with treatment could be established.

Measurement of whole blood thiamine by erythrocyte transketolase activity showed significant difference between the polyneuropathy (85 ± 14 nmol/l) and mononeuropathy (110 ± 21 nmol/l) patients ($P < 0.05$). Also, serum level of vitamin B_{12} showed nearly the same significant difference between the polyneuropathy (220 ± 37 pg/ml) and mononeuropathy (325 ± 54 pg/ml), this difference is illustrated in Fig. 1. There was no statistically significant difference between the mono- and polyneuropathy groups regarding vitamin D and folate.

The impact of peripheral nerve complications on the functional status of our patients is summarized in Table 3. While some of the complications in our patients are apparently mild, a significant number were disabling either through weakness (advanced generalized neuropathy, bilateral foot drop, severe unilateral foot drop) or through neuropathic pain syndrome (small fiber neuropathy, cases of sensory neuropathy, cases of carpal tunnel syndrome). The inability to drive safely or to drive at all was a major factor for higher disability, as driving independently is the major means of transportation in Kuwait.

Discussion

We observed various patterns of peripheral nerve complications following BS in our cohort. Micronutrient deficiencies and non-compliance with treatment were quite frequent in our patients. However, nutritional deficiencies after BS seem not the only preventable factor leading to peripheral neuropathies. We recognize a number of weaknesses in our study. It is retrospective and not controlled so that conclusions regarding the risk factors or possible correlates of the peripheral nerve disorders observed are less reliable or impossible. Nevertheless, we believe our observations make a positive contribution to the knowledge of different post-bariatric peripheral nerve complications that may be better discussed separately.

Generalized neuropathy was observed in 35 of our patients, 23 of whom developed chronic distal symmetrical polyneuropathy. It was purely sensory or sensory

Table 2 Nutritional status and compliance with treatment among the patients

	Mononeuropathy, *n* = 22		Polyneuropathy, *n* = 35		*P*
Thiamine, deficient vs non-deficient (*n*)	9 (41%) vs 13 (59%)		20 (57%) vs 15 (43%)		0.24*
Vitamin B_{12}, deficient vs non-deficient (*n*)	7 (31.8%) vs 15 (69.2%)		15 (43%) vs 20 (57%)		0.53*
Folate, deficient vs non-deficient (*n*)	5 (22.7%) vs 17 (77.3%)		12 (34.2%) vs 23 (65.7%)		0.23*
Vitamin D, deficient vs non-deficient	6 (27.2%) vs 16 (72.8%)		6 (17%) vs 29 (83%)		0.27*
Compliance with treatment	*n*	Percent	*n*	Percent	
Compliant	8	36.4	7	20.7	0.62*
Poorly compliant	11	50.0	16	48.3	
Non-compliant	3	13.6	12	31.0	

*Fisher exact probability test for 2 × 2 and 2 × 3 contingency tables

predominant but tended to progress and involve the motor function. Our findings coincide with previous descriptions of this most common post-bariatric distal chronic neuropathy [10, 11, 15, 16]. Acute axonal radiculoneuropathies or neuropathies with prominent weakness have also been reported [15–17]. We encountered 2 patients with acute axonal sensorimotor neuropathy. Such cases may represent either an early [17] or a late [18] complication of BS, often combined with Wernicke's encephalopathy [19]. As these generalized neuropathies seem mostly due to thiamine deficiency resulting from malabsorption and postoperative vomiting [15, 20], postoperative care for patients with refractory vomiting should be very close, with adequate thiamine replacement.

Small fiber painful presentation ("burning feet" that in some patients evolved into distal neuropathic pain syndrome in all extremities) was seen in 10 cases. Small fiber neuropathy seems relatively frequent in our series, but other authors have also turned the attention to this variety that may be responsive to thiamine [15, 21, 22]. Early pain control is also necessary to prevent the development of chronic neuropathic pain. Patients with various isolated mononeuropathies have been described after BS [10, 11, 15]. Some lesions were directly related to compression or stretch during surgery [22], a pattern that we did not observe.

CTS was the most frequent mononeuropathy in a large controlled cohort study [11] and also in our patients (10 out of 22). The causal relation between weight

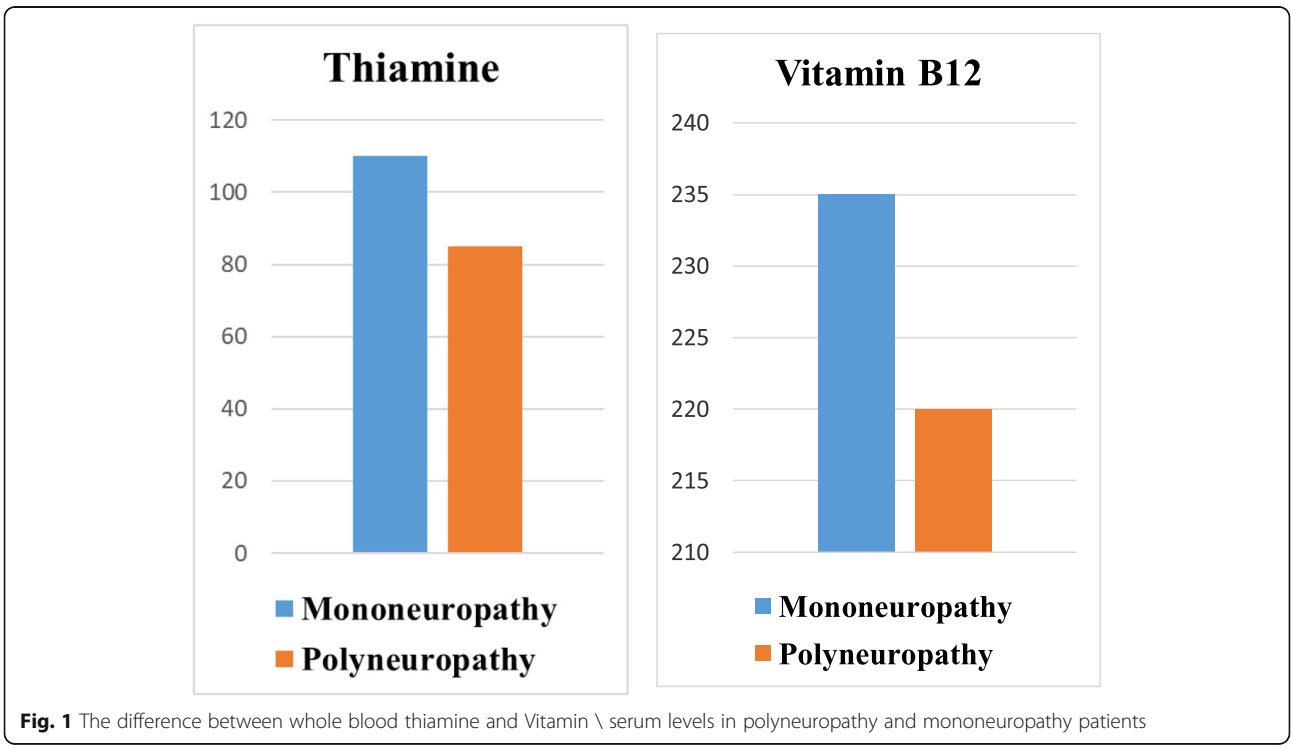

Fig. 1 The difference between whole blood thiamine and Vitamin \ serum levels in polyneuropathy and mononeuropathy patients

Table 3 Functional impairment of patients with different peripheral nerve complications

	Modified Rankin Scale			
	Score 1	Score 2	Score 3	Score 4
Mononeuropathy				
Carpal tunnel syndrome	5	5		
Bilateral peroneal palsy			2	
Unilateral peroneal palsy		2	3	
Ulnar neuropathy at the elbow	2	2		
Meralgia paresthetica	1			
Polyneuropathy				
Distal symmetrical sensorimotor polyneuropathy	8	10	5	
Acute several axonal neuropathy				2
Small fiber neuropathy	2	4	4	
Lumbar plexopathy			1	

Number of patients distributed by score. There were no patients with a score of 0.5 or 6

loss and CTS is difficult to understand. Obesity is in fact a risk factor for CTS, so mechanical rather than metabolic factors may be at play [11]. Peroneal and ulnar nerve involvement at the predilection sites seems easier to explain after weight loss, as the reduction of protective subcutaneous fat together with increased nerve vulnerability in malnutrition results in easier development of compression [20, 23, 24]. We observed 2 patients with bilateral peroneal palsy among 22 mononeuropathies, while the literature reports on bilateral peroneal palsy after BS are few [25, 26]. Unilateral peroneal neuropathy was also frequent in our patients (5 out of 58 cases) while described in only 2 patients among 71 in a previous series [11]. This higher occurrence of peroneal palsy in our patients is likely due to cultural differences. In Asia, squatting or sitting "taylor" style with legs crossed is quite common, causing peroneal nerve compromise [27]. Peroneal palsy, especially bilateral, is disabling [28] while entirely preventable by avoiding the offending postures. The same considerations are valid in cases of ulnar nerve compression, of which we observed 4 cases. We observed a single case of meralgia paraesthetica while others have described this complication far more frequently [29], mostly as an early complication related to intraoperative trauma of the nerve. A single case of lumbar plexopathy was observed. In other series, this complication was more frequent [11]. This entity is presumably autoimmune or inflammatory, and the direct relation to BS seems elusive [20].

In our study, there are two patterns of neurological complications after bariatric surgery, generalized polyneuropathy that can be explained by malabsorption and prolonged vomiting that leads to neurologic dysfunction in different levels of the neuraxis. This mechanism explains why whole blood level of thiamine and serum level of vitamin B_{12} are more affected in these patients than mononeuropathy patients that their pathology can be explained by rapid weight loss that may make the nerves more susceptible to compression through loss of subcutaneous tissue, loss of protective fat pads, or structural changes. Sometimes, injury from mechanical retractors, patients' malpositioning, and use of radial or ulnar catheter at the wrist during surgery may participate in immediate complications after BS [11].

Our data indicate that poor nutritional status and non-compliance with treatment in patients with post-bariatric neuropathy are quite frequent. The study is not controlled and does not allow us to indicate particular factors, but this clustering of malnutrition among cases of peripheral complications is well-described and likely multifactorial (malabsorption, vomiting, rapid weight loss, poor compliance) [11, 15, 20]. While some peripheral complications of BS are mild, others may be disabling and irreversible [20, 30]. Our brief assessment of patients' functional status turns attention to such cases (sensorimotor neuropathy, foot drop, or severe neuropathic pain in poly- or mononeuropathies) and underlines the need for prevention through close perioperative care, regular follow-up, adequate supplementation, and educating the patients to avoid habitual postures causing nerve compression.

Conclusions

Peripheral nerve complications after BS may be classified into generalized neuropathies (chronic axonal distal symmetric sensory or sensorimotor neuropathy, acute polyradiculoneuropathy, and small fiber neuropathy), mononeuropathies, and plexopathy. These complications may overlap in the same person either by chance or by common causal factors. The clinical picture of every individual patient or similar group of patients will differ according to the relative frequency of individual complications and their correlation with nutritional deficits, local compression, and possible inflammatory factors. Prevention is possible and essential to avoid peripheral nerve complications of BS.

The current study has faced certain limitation related to its nature, being a descriptive retrospective analysis. One of these limitations is that we cannot precisely estimate the rate of weight loss as well as the actual compliance to vitamin supplement. Another limitation is the small number of cases which limits the statistical power of analysis. The correlation between the type of neuropathy and the clinical variable could be found to be significant in a larger group of study.

Abbreviations
ANOVA: Analysis of variance; BS: Bariatric surgery; CTS: Carpal tunnel syndrome; EMG: Electromyography; NCS: Nerve conduction study

Acknowledgements
Not applicable.

Funding
No funding related to this study has been received by any and all of the Authors.

Availability of data and materials
All data are available at the hospital archive within the patients files but can not be shared publicly.

Authors' contributions
HR, AJK, and RR carried out the neurophysiology work-up and contributed to the case selection and recruitment. DY, DM, JA, WK, and AyK contributed in the case selection, evaluation, and diagnoses. DY and HR contributed to the idea of the research. AyK and DY contributed to the drafting of the manuscript and its revision. AyK contributed to the statistical analysis and carried out the submission process. All authors read and approved the final manuscript.

Ethics approval and consent to participate
The study has been approved, on January 2014, by the ethical approval committee of Ibn Sina Hospital, Kuwait.
A legal written consent has been taken from every case regarding agreement on participation in the study. Proper explanation was given to patients regarding investigation and results of the study. An appropriate time was given for discussion and we admit that patients understood all the points.

Consent for publication
not applicable.

Competing interests
The authors declare that they have no competing interests.

Publisher's Note

Author details
[1]Neurology Department, Ibn Sina Hospital, Kuwait City, Kuwait. [2]Clinical Neurophysiology Department, Faculty of Medicine, Kasr El Aini Hospital, Cairo University, Giza, Egypt. [3]Neurology Department, Faculty of Medicine, Kasr El Aini Hospital, Cairo University, Giza, Egypt. [4]Neurology Department, Faculty of Medicine, Minia University, Minya, Egypt. [5]Neurology Unit, Farwaniya Hospital, Kuwait City, Kuwait. [6]National Research Centre, Children with Special Needs, Cairo, Egypt. [7]Health Sciences Center, Kuwait University, Kuwait City, Kuwait. [8]Department of Neurology, Faculty of medicine, Beni-Suef University, Beni-Suef, Egypt.

Received: 23 November 2017 Accepted: 5 February 2019
Published online: 12 March 2019

References
1. Stevens GA, Singh GM, Lu Y, Danaei G, Lin JK, Finucane MM, et al. National, regional, and global trends in adult overweight and obesity prevalences. Popul Health Metrics. 2012;10(1):22.
2. Allender S, Rayner M. The burden of overweight and obesity-related ill health in the UK. Obes Rev. 2007;8(5):467–73.
3. WHO Global Health Observatory data repository, http://apps.who.int/gho/data/node.main.A900A?lang=en. Accessed online 14 Dec 2015.
4. Colquitt JL, Pickett K, Loveman E, Frampton GK. Surgery for weight loss in adults. Cochrane Database Syst Rev. 2014 Aug 8;8:CD003641.
5. Yumuk V, Tsigos C, Fried M, Schindler K, Busetto L, Micic D, et al. European Guidelines for Obesity Management in Adults. Obes Facts. 2015;8(6):402–24.
6. Corcelles R, Daigle CR, Schauer PR. Metabolic effects of bariatric surgery. Eur J Endocrinol. 2016;174(1):R19–28 Epub 2015 Sep 4.
7. Haskins O. Bariatrics in Kuwait: Dr Salman al-Sabah. http://www.bariatricnews.net/?q=news/111249/bariatrics-kuwait-dr-salman-alsabah. Accessed 14 Dec 2015.
8. Chang SH, Stoll CR, Song J, Varela JE, Eagon CJ, Colditz GA. The effectiveness and risks of bariatric surgery: an updated systematic review and meta-analysis, 2003–2012. JAMA Surg. 2014;149(3):275–87.
9. Malinowski SS. Nutritional and metabolic complications of bariatric surgery. Am J Med Sci. 2006;331(4):219–25.
10. Koffman BM, Greenfield LJ, Ali II, Pirzada NA. Neurologic complications after surgery for obesity. Muscle Nerve. 2006;33(2):166–76.
11. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. Neurology. 2004;63(8):1462–70.
12. Jablecki CK, Andary MT, Floeter MK, Miller RG, Quartly CA, Vennix MJ, et al. Practice parameter: electrodiagnostic studies in carpal tunnel syndrome. Neurology. 2002;58:1589–92.
13. van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J. Interobserver agreement for the assessment of handicap in stroke patients. Stroke. 1988;19(5):604–7.
14. Kinura J. Assessment of individual nerves. In: Kimura J, editor. Electrodiagnosis in diseases of nerve and muscle: principles and practice. 4th ed. NY: Oxford University Press; 2013. p. 99–138.
15. Abarbanel JM, Berginer VM, Osimani A, Solomon H, Charuzi I. Neurologic complications after gastric restriction surgery for morbid obesity. Neurology. 1987;37(2):196–200.
16. Chaudhry V, Umapathi T, Ravich WJ. Neuromuscular diseases and disorders of the alimentary system. Muscle Nerve. 2002;25(6):768–84.
17. Chang CG, Adams-Huet B, Provost DA. Acute post-gastric reduction surgery (APGARS) neuropathy. Obes Surg. 2004;14(2):182–9.
18. Scarano V, Milone M, Di Minno MN, Panariello G, Bertogliatti S, Terracciano M, et al. Late micronutrient deficiency and neurological dysfunction after laparoscopic sleeve gastrectomy: a case report. Eur J Clin Nutr. 2012;66(5):645–7.
19. Samanta D. Dry beriberi preceded Wernicke's encephalopathy: thiamine deficiency after laparoscopic sleeve gastrectomy. J Pediatr Neurosci. 2015;10(3):297–9.
20. Berger JR, Singhal D. The neurologic complications of bariatric surgery. Handb lin Neurol. 2014;120:587–94.
21. Menezes MS, Harada KO, Alvarez G. Painful peripheral polyneuropathy after bariatric surgery. Case reports. Rev Bras Anestesiol. 2008;58(3):252–9.
22. Philippi N, Vinzio S, Collongues N, Vix M, Boehm N, Tranchant C, et al. Peripheral neuropathies after bariatric surgery. Rev Neurol (Paris). 2011;167(8–9):607–14.
23. Cruz Martínez A. Slimmer's paralysis: electrophysiological evidence of compressive lesion. Eur Neurol. 1987;26(3):189–92.
24. Frantz DJ. Neurologic complications of bariatric surgery: involvement of central, peripheral and enteric nervous system. Curr Gastroenterol Rep. 2012;14(4):367–72.
25. Elias WJ, Pouratian N, Oskouian RJ, Schirmer B, Burns T. Peroneal neuropathy following successful bariatric surgery. J Neurosurg. 2006;105(4):631–5.
26. Milants C, Lempereur S, Dubuisson A. Bilateral peroneal neuropathy following bariatric surgery. Neurochirurgie. 2013;59(1):50–2.
27. Yu JK, Yang JS, Kang SH, Cho YJ. Clinical characteristics of peroneal nerve palsy by posture. J Korean Neurosurg Soc. 2013;53(5):269–73.
28. Aprile I, Tonali P, Caliandro P, Pazzaglia C, Foschini M, Di Stasio E, et al. Italian multicentre study of peroneal mononeuropathy: multiperspective follow-up. Neurol Sci. 2009;30(1):37–44.
29. Macgregor AM, Thoburn EK. Meralgia paresthetica following bariatric surgery. Obes Surg. 1999;9(4):364–8.
30. Juhasz-Pocsine K, Rudnicki SA, Archer RL, Harik SI. Neurologic complications of gastric bypass surgery for morbid obesity. Neurology. 2007;68(21):1842–50.