ΜΕΤΑΠΤΥΧΙΑΚΟ ΠΡΟΓΡΑΜΜΑ ΣΠΟΥΔΩΝ:
‘‘ΕΛΑΧΙΣΤΑ ΕΠΕΜΒΑΤΙΚΗ ΧΕΙΡΟΥΡΓΙΚΗ,
ΡΟΜΠΟΤΙΚΗ ΧΕΙΡΟΥΡΓΙΚΗ ΚΑΙ ΤΗΛΕΧΕΙΡΟΥΡΓΙΚΗ’’

ΕΘΝΙΚΟ ΚΑΙ ΚΑΠΟΔΙΣΤΡΙΑΚΟ ΠΑΝΕΠΙΣΤΗΜΙΟ ΑΘΗΝΩΝ
ΙΑΤΡΙΚΗ ΣΧΟΛΗ

ΔΙΠΛΩΜΑΤΙΚΗ ΕΡΓΑΣΙΑ

ΘΕΜΑ:

Wernicke encephalopathy after sleeve gastrectomy.

A review of the literature

ΜΕΤΑΠΤΥΧΙΑΚΟΣ ΦΟΙΤΗΤΗΣ:
ΑΓΓΕΛΟΥ ΑΝΑΣΤΑΣΙΟΣ
Α.Μ.:20120726

ΑΘΗΝΑ, ΙΑΝΟΥΑΡΙΟΣ 2015

ΠΡΑΚΤΙΚΟ ΚΡΙΣΕΩΣ
ΤΗΣ ΣΥΝΕΔΡΙΑΣΗΣ ΤΗΣ ΤΡΙΜΕΛΟΥΣ ΕΞΕΤΑΣΤΙΚΗΣ ΕΠΙΤΡΟΠΗΣ
ΓΙΑ ΤΗΝ ΑΞΙΟΛΟΓΗΣΗ ΤΗΣ ΔΙΠΛΩΜΑΤΙΚΗΣ ΕΡΓΑΣΙΑΣ
Του Μεταπτυχιακού Φοιτητή Αγγέλου Αναστασίου

Εξεταστική Επιτροπή

- Ανδρέας Αλεξάνδρου, Λέκτορας Χειρουργικής, **Επιβλέπων**
- Χρήστος Π. Τσιγκρής, Καθηγητής Χειρουργικής & Επιστημονικός Υπεύθυνος του Π.Μ.Σ.
- Θεόδωρος Διαμαντής, Καθηγητής Χειρουργικής

Η Τριμελής Εξεταστική Επιτροπή η οποία ορίσθηκε από την ΓΣΕΣ της Ιατρικής Σχολής του Παν. Αθηνών Συνεδρίαση της.....ης ..... 20.... για την αξιολόγηση και εξέταση του υποψηφίου κ. Αγγέλου Αναστάσιου, συνεδρίασε σήμερα .../.../....

Η Επιτροπή διαπίστωσε ότι η Διπλωματική Εργασία του Κ. Αγγέλου Αναστάσιου με τίτλο «Wernicke encephalopathy after sleeve gastrectomy. A review of the literature» είναι πρωτότυπη, επιστημονικά και τεχνικά άρτια και η βιβλιογραφική πληροφορία ολοκληρωμένη και εμπεριστατωμένη.

Η εξεταστική επιτροπή αφού έλαβε υπ' όψιν το περιεχόμενο της εργασίας και τη συμβολή της στην επιστήμη, με ψήφους ..... προτείνει την απονομή του Μεταπτυχιακού Διπλώματος Ειδίκευσης (Master's Degree), στον παραπάνω Μεταπτυχιακό Φοιτητή.

Στην ψηφοφορία για την βαθμολογία ο υποψήφιος έλαβε για τον βαθμό «ΑΡΙΣΤΑ» ψήφους ....., για τον βαθμό «ΛΙΑΝ ΚΑΛΩΣ» ψήφους ....., και για τον βαθμό «ΚΑΛΩΣ» ψήφους ..... Κατά συνέπεια, απονέμεται ο βαθμός «.....».

Τα Μέλη της Εξεταστικής Επιτροπής

- Ανδρέας Αλεξάνδρου , **Επιβλέπων** (Υπογραφή) _____
- Χρήστος Π. Τσιγκρής (Υπογραφή) _____
- Θεόδωρος Διαμαντής (Υπογραφή) _____

Contents **Page**

Wernicke encephalopathy -after sleeve gastrectomy. A review of the literature

1. INTRODUCTION

Bariatric surgery is currently the most effective treatment modality to induce weight loss and ameliorate the various comorbidities of obesity. However, bariatric interventions induce major changes in the anatomy and physiology of the gastrointestinal tract, thus can be responsible for quite a range of side-effects, including nutritional deficiencies.

Bariatric operations traditionally are divided to restrictive, malabsorptive or combined according to the mechanism they induce weight-loss. The vertical banded gastroplasty,the laparoscopic adjustable gastric band and the laparoscopic sleeve gastrectomy are considered restrictive procedures, deriving their efficacy from decreasing the gastriccapacity, thus restricting the ability to ingest food. The various configurations of biliopancreatic diversion (Scopinaro, duodenal switch, etc) are considered malabsorptive procedures, as they bypass various parts of the small intestine, thus reducing the actual absorptive ability of the alimentary tract, creating in this way a permanent partial food malabsorption, when this reduced absorptive capacity is overtaken by increased food ingestion. (1). Last but not least Roux-en-Y gastric bypass combines both mechanisms by reducing drastically the gastric capacity with the creation of a small gastric pouch and inducing malabsorption by bypassing a various portion of the proximal jejunum used for the creation of the biliopancreatic and the alimentary loop, thus allowing only the common loop for food digestion.

Forseeingly, the various bariatric surgical procedures have been associated with nutritional deficiencies, but their prevalence depend on the type of surgery. Sleeve gastrectomy being a restrictive operation leads less frequently to nutritional

deficiencies than the Roux-en-Y gastric bypass, which having also a malabsorptive component is associated with a higher prevalence of nutritional deficiencies. Biliopancreatic diversion is a procedure in which a large section of small intestine is bypassed, and hence it carries the highest risk for nutritional deficiencies. (2) (3) (4)

In this paper, one particular deficiency complicating bariatric surgery, with potential grave consequences, the thiamine (vitamin B1) deficiency and wernicke encephalopathy is summarized, with a focus on clinical management aspects.

2. MATERIALS AND METHODS

2.1 Literature search strategies

We searched MEDLINE, Ovid, Embase, ISI (Science Citation Index), and Google Scholar independently and in duplicate, using the MeSH terms [Wernicke encephalopathy and bariatric surgery or obesity surgery or sleeve gastrectomy]. We reviewed references of all reports for additional cases. We used the related articles link and searched the citations of reports in

the ISI Science Citation Index to identify additional reports.

2.2 Inclusion and Exclusion criteria

Included were case reports, case series, or cohort studies describing Wernicke encephalopathy after sleeve gastrectomy. Cases included were those that met the operational case definition of Wernicke encephalopathy, comprising two of the following four symptoms: 1) dietary deficiencies, 2) oculomotor abnormalities, 3) cerebellar dysfunction, and 4) altered mental state or mild memory impairment. We independently reviewed the full text of these articles and abstracted data on age, sex,

duration of onset, type of surgery, severity of weight loss, predisposing factors, radiographic findings, unique neurologic features, and outcomes.

2.3 Result of the literature search

By using the above search strategy a total of 7 studies, including 7 patients were analyzed.

3. RESULTS

3.1 Indications for sleeve gastrectomy

The indications for sleeve gastrectomy are referred to Table 2 (5)

3.2 Patient's demographics and clinical characteristics (Table 1)

We identified 7 case reports. Patients ranged in age from 24 to 43 years; 3 patients were aged 21 to 30 years, 3 patients were aged 31 to 40 years, and 1 patients were aged 41 to 50 years. Most patients were women (n=5). The onset of Wernicke encephalopathy occurred as early as 3 weeks after surgery to as late as 60 weeks after surgery. When symptoms of Wernicke encephalopathy developed, patients had lost from 19 kg of body weight up to 60 kg of body weight.

The first case reported by Cerutti et al.(6), described the manifestation of central and peripheral neurological deficits and psychogenic anorexia in a 33-year-old man, who presented 18 weeks after SG. Because of the patient's history, the clinical examination and findings with brain magnetic resonance imaging, he was diagnosed with Wernicke's encephalopathy. The patient improved with thiamine, antidepressant drugs and atypical neuroleptics. The second case reported by Jeong et al.(7) described the manifestations of diplopia and dysarthria in a 24-year-old man who presented 4 months after SG. He had been experiencing these symptoms for 3 months

after surgery, but failed to report them to his physician. His symptoms partially improved with thiamine treatment; however mild gait disturbances and dysarthria failed to completely resolve. In the third case by Makarewicz et al.(8), a 38 year old morbidly obese patient underwent SG, complicated by gastric wall edema, dietary noncompliance and recurrent vomiting; in addition to the restrictive nature of her surgery, these factors precipitated Wernicke's encephalopathy 1 month after her surgery. Thiamine supplementation was administered, with gradual improvement over 6 months after the diagnosis. No permanent cognitive impairment was reported.

In 2012, Moizé et al.(9) published a case of a 35 year old obese female presenting to the emergency department 2.5 months after SG, complaining of repeated vomiting and 11.3% weight loss as compared to her preoperative status. As workup failed to demonstrate an etiology, she was diagnosed with anxiety and discharged home, only to be readmitted a month later with visual disturbances. A nystagmus was elicited by neurological exam, and a thiamine level confirmed the diagnosis of Wernicke's encephalopathy. The fifth case by Scarano et al(10), describes a 37-year-old woman with a late development of Wernicke's encephalopathy and severe polyneuropathy 3 months after SG. About 6 months after onset of neurological picture, her symptoms partially improved with thiamine treatment. In the next case by Sharabi and Bisharat(11), a 43-year-old female patient underwent SG and suffered from Wernicke's encephalopathy and paralytic ileus. Thiamine supplementation was administered with gradual improvement in her encephalopathy; however, she was readmitted three months later with a profound ileus, developed multi-system organ failure and died. The last case by Saab et al.(12)refers to a 27 year-old female that she had undergone SG for management of morbid obesity 3 weeks prior to her admission. Given her recent bariatric surgery and high index of suspicion, a quick neurological

examination was done, demonstrating a left direction-changing horizontal nystagmus, normal visual fields and no diplopia. She had short-term memory loss, and was poorly cooperative with the sensory examination. There was weakness in all extremities, with a decreased knee-jerk reflex bilaterally.

Vomiting was a predisposing factor in most patients (n=5). IV glucose without thiamine and parenteral nutrition were other predisposing factors. The triad of Wernicke encephalopathy (confusion, ataxia, and nystagmus) was present in the majority of patients. Common findings on brain MRI were hyperintense signals in the dorsal medial thalamic nucleus, periaqueductal gray area, third/fourth ventricles, and caudate nucleus and putamen (n=5). Serum thiamine levels were low in 5 patients. Information about thiamine supplementation was unavailable in most reports.

Most patients were treated with parenteral thiamine and made a complete recovery (n=6). Some patients had residual neurologic defects, including Korsakoff psychosis, persistent amnestic state with antegrade amnesia and retrograde memory loss, memory problems, persistent ataxia, nystagmus, and neuropathy(n=2). One died during hospitalization from septic shock.

Table 1.

Patient's demographics and clinical characteristics

Paper	sex /age	Onset (wk)	c/a/n	Neurologic deficits	Outcome	Therapy b1	Thiamine levels	BMI (weight loss-kg)	Comorbidities	Sleeve complication
CERRUTI et al.(6)	m/ 33	18	c/a/n	Paresthesia, dysarthria	Improvement	1500mg/iv-500mg orally	-	47(60)	-	-
HEO JUN et al.(7)	m/ 24	60	c/a/n	dysarthria	Improvement	Iv 100mg for 10d then orally	15nmol/L (20-50)	47,1(60)	DM, HTN	Anastomotic line leakage
MAKAREWICZ et al.(8)	f/ 38	4	c/a/n	Paresis of left tibial nerve, loss of sensation	Improvement	Iv 50mg per day	-	62	DM, HTN	Edema of gastric wall, functional stenosis of the stomach
MOIZE et al.(9)	F/ 35	8	c/a/n	-	Resolution	100mg x3 +5mg folic orally	11ng(35-91)	49,8 (19,5)	DM, HTN	Laceration of the mucosa of GEJ
SCARANO et al.(10)	f/ 27	12	c/a/n	Lower limb paresthesia	Improvement	1800mcg im/w, iv 500 mg x3 then orally 100mg x1	Urine 22mg (40-90)	43,7	-	-
SHARABI et al.(11)	f/ 43	8	c/a/n	-	Death	Iv 600 mg x1	17 ng(28-85)	-	-	Inguinal abscess-paralytic ileus
SAAB et al.(12)	f/ 27	3	c/a/n	-	Improvement	Iv 100 mg-orally 100 x3	43(70-180)	-	-	-

Table 2.

Indications for Primary Laparoscopic Sleeve Gastrectomy in the Morbidly Obese(5)

Procedure	**Characteristics**
Two-stage procedure	
First step in super–super morbidly obese patient	Followed by RYGB or BPD
First step to a non -bariatric second procedure	Low BMI of 35–40 Followed by hip replacement, recurrent incisional hernia, pull through procedure for ulcerative colitis, renal/liver transplantation
Single-stage procedure	
Final step in ASA IV Morbidly Obese Patient	Low EF, Heart/Liver/Kidney transplant recipient
Final step in poor candidate for LRYGB or BPD-DS	Smoker Warfarin
Final step in extremes of age	Adolescents Elderly age ≥70 yrs
Final step in a high risk stomach	Chile, Colombia, Japan: high incidence of gastric cancer
Final step in Crohn's disease **Patient preference** **Low BMI of 35–40 with comorbidity** **BMI 30–35 with the metabolic syndrome**	

5.DISCUSSION

Wernicke Encephalopathy (WE) was first described by Carl Wernicke in 1881 as "superior acute hemorrhagic poliencephalitis". Despite the fact that WE was described so early, it took more than half a century to recognize the relation between the disease and the thiamine deficiency(20). Thiamine is a water-soluble vitamin of the B complex. It Is involved with several enzymes in the Krebs cycle and pentose phosphate pathway and plays a central role in cerebral metabolism. Thiamine deficiency in turn instigates brain injury by inhibiting metabolism in regions of the brain with high metabolic demand. The most frequently affected regions are the medial thalamus and periventricular region of the third ventricle, periaqueductal area, mammillary bodies and midbrain tectum (superior and inferior colliculi). Often, there is selective loss of the Purkinje cells in the cerebellum. Although alcoholic patients account for more than 90% of both acute and chronic WE in developed countries, acute WE can occur also in cases of malnutrition (prolonged fasting, parenteral nutrition without the addition of thiamine) or even in cases of prolonged vomiting(13, 14). Bariatric surgery can imitate or be complicated by all these conditions. Suddenly occurring in its most common form, WE is classically characterized by a clinical triad including reduced consciousness, a cerebellar syndrome (in particular ataxia and dysarthria) as well as oculomotor disorders (ophthaloplegia, nystagmus). The classic triad of WE was present in only 16% of patients in a retrospective autopsy review(15). According to the Caine criteria (14), the diagnostic criteria for Wernicke encephalopathy require 2 of the following 4 features: (1) dietary deficiency, (2) oculomotor abnormality, (3) cerebellar dysfunction, and (4) confusion or mild memory impairment. These criteria have a very high interrater reliability for the diagnosis.

The risk of WE evolvement depends on the type of bariatric surgery performed. Purely restrictive operations, such as laparoscopic adjustable gastric banding,sleeve gastrectomy and vertical banded gastroplasty, tend to cause fewer deficiencies than do malabsorptive procedures such as laparoscopic Roux-en-Y gastric bypass and biliopancreatic diversion(16). In morbidly obese patients following LSG, several factors, including the restriction in energy intake, prolonged vomiting, rapid and excessive weight lost, and medication noncompliance, have been associated to the expression of WE(17). In particular, vomiting represents a well-known complication of LSG which may be due to surgery-related complications because of the presence of gastric stricture, leak and staple-line bleeding.

Despite the availability of different diagnostic modalities, including laboratory evaluation, electroencephalography, and brain imaging, WE remains a clinical diagnosis, requiring a high index of suspicion. The presumptive diagnosis may be confirmed by measuring of thiamine and its phosphate esters in human blood by high-performance liquid chromatography (HPLC) or by measuring the activity of the enzyme transketolase in erythrocytes(18). However, because of technical difficulty and lack of specificity, and also take several days to perform and will not aid in decisionmaking, these tests are limited. Neuroimaging studies are powerful tools in supporting the diagnosis of WE. MRI offers the best way to make a definitive diagnosis for WE, despite a low sensitivity of 53%, but a 93% specificity to rule out the disorder(19). Common brain MRI findings are hyperintense signals in the dorsal medial thalamic nuclei, periaqueductal gray area, and the third and fourth ventricle(17). The acute stage may show mammillary bod y enhancement, whereas the chronic stage may show mammillary body atrophy(19).

The mainstay of treatment of WE is the administration of thiamine. The standard recommendation is 200 mg, 3 times per day. The intravenous route is preferred instead of the intramuscular one, while the oral route in the acute state is not recommended due to decreased gastrointestinal absorption. However, several authors state that the dose should be increased to 500 mg thiamine 3 times per day(14).

The contribution of LSG to the evolution of bariatric surgery is unquestionable. However, LSG like any other type of surgery is not without complications. The impact of WE after bariatric surgery is significantly underestimated. Due to its increasing clinical application, even if Wernicke's encephalopathy was considered as an extremely rare complication that can occur after LSG, more and more relevant literature references come to light. Therefore, physicians should be highly suspicious with the diagnosis of Wernicke's encephalopathy in cases of rapidly altered mental status occurring in patients with malnutrition or malabsorption as a consequence of prolonged vomiting after bariatric surgery.

6. CONCLUSIONS

In summary, this study reaffirms that only a small number of patient suffered from B1 deficiency around the world after LSG for morbid obesity. Physicians should be highly suspicious with the diagnosis of Wernicke's encephalopathy in cases of rapidly altered mental status occurring in patients with malnutrition or malabsorption as a consequence of prolonged vomiting after SG. Preoperative nutritional assessment and rigorous postoperative follow-up plan with administration of multi-vitamin supplements and assessment of serum levels is recommended in all patients.

ABSTRACT

Background: Bariatric surgical procedures are increasingly and successfully applied in the treatment of morbid obesity. Nutrient deficiencies constitute the most important long-term complications of bariatric interventions, as they may lead to haematological, metabolic and especially neurological disorders which are not always reversible. Thiamine (vitamin B1) deficiency has been reported in patients after bariatric surgery.

Objective: The aim of this study is to review all cases that underwent Sleeve gastrectomy and presented with Wernicke Encephalopathy, as a complication of the procedure.

Methods: A review of the literature was conducted to evaluate all sleeve gastrectomies performed till today and caused Wernicke encephalopathy to the patients. Demographic data, operative parameters,, postoperative outcomes were collected and assessed.

Results: A total of 7 studies, including 7 patients were analyzed. Patients ranged in age from 24 to 43 years. The onset of Wernicke encephalopathy occurred as early as 3 weeks after surgery to as late as 60 weeks after surgery. When symptoms of Wernicke encephalopathy developed, patients had lost from 19 kg of body weight up to 60 kg of body weight. As this condition may lead to potentially irreversible neurological manifestations, clinical alertness and prompt adequate treatment in case of deficiency are required.

Conclusions: Micronutrient deficiencies and particularly B1 deficiency can occur after LSG, although rarely. Preoperative nutritional assessment and postoperative

follow-up plan, should signs of Wernicke's encephalopathy be traced, is recommended in all patients.

ΠΕΡΙΛΗΨΗ

Εισαγωγή: Οι βαριατρικές επεμβάσεις είναι διαρκώς αυξανόμενες και χρησιμοποιούνται με επιτυχία στη θεραπεία της νοσογόνου παχυσαρκίας. Η ανεπάρκεια βασικών θρεπτικών συστατικών αποτελεί μια σοβαρή μακροπρόθεσμη επιπλοκή των επεμβάσεων αυτών, καθώς μπορεί να προκαλέσει σοβαρές αιματολογικές, μεταβολικές και ειδικά νευρολογικές διαταραχές που πολλές φορές είναι μη αναστρέψιμες. Η έλλειψη Β1 (θειαμίνης) έχει περιγραφεί σε ασθενείς που έχουν υποβληθεί σε επέμβαση για νοσογόνο παχυσαρκία.

Σκοπός: Ο σκοπός αυτής της ανασκόπησης είναι η συλλογή των ασθενών που υποβλήθηκαν σε επιμήκη γαστρεκτομή και εμφάνισαν στην μετεγχειρητική τους πορεία εγκεφαλοπάθεια Wernicke, ως πιθανή επιπλοκή της επέμβασης.

Μέθοδοι: Η ανασκόπηση της βιβλιογραφίας πραγματοποιήθηκε με σκοπό να εκτιμήσει το ποσοστό των επιμηκών γαστρεκτομών, οι οποίες είχαν ως επιπλοκή την εμφάνιση εγκεφαλοπάθειας Wernicke στους ασθενείς. Δημογραφικά δεδομένα, διεγχειρητικά στοιχεία και μετεγχειρητικά αποτελέσματα συγκεντρώθηκαν και αναλύθηκαν.

Αποτελέσματα: Ένα σύνολο 7 μελετών που περιελάμβαναν 7 ασθενείς αναλύθηκαν. Οι ασθενείς είχαν ηλικία από 24-43 έτη. Η εμφάνιση των συμπτωμάτων της εγκεφαλοπάθειας Wernicke συνέβη από την 3^{η} ΜΤΧ εβδομάδα έως και 60 εβδομάδες μετά το χειρουργείο. Όταν εμφανίστηκαν τα συμπτώματα οι ασθενείς είχαν χάσει από

19-60 κιλά σωματικού βάρους. Αυτή η κατάσταση οδηγεί σε πιθανές μη αναστρέψιμες νευρολογικές βλάβες και απαιτεί την εγρήγορση των χειρουργών.

Συμπεράσματα: Βασικές μεταβολικές διαταραχές και ειδικά η ανεπάρκεια Β1 (θειαμίνης) είναι δυνατόν να συμβούν μετά από επιμήκη γαστρεκτομή. Η προεγχειρητική εκτίμηση θρέψης των ασθενών και ο μετεγχειρητικός επανέλεγχος συστήνεται για τους ασθενείς με νοσογόνο παχυσαρκία.

REFERENCES

1.Baptista V, Wassef W. Bariatric procedures: an update on techniques, outcomes and complications. Current opinion in gastroenterology. 2013;29(6):684-93.
2.Saltzman E, Karl JP. Nutrient deficiencies after gastric bypass surgery. Annual review of nutrition. 2013;33:183-203.
3.Hammer HF. Medical complications of bariatric surgery: focus on malabsorption and dumping syndrome. Digestive diseases. 2012;30(2):182-6.
4.Levinson R, Silverman JB, Catella JG, Rybak I, Jolin H, Isom K. Pharmacotherapy prevention and management of nutritional deficiencies post Roux-en-Y gastric bypass. Obesity surgery. 2013;23(7):992-1000.
5.Tucker ON, Szomstein S, Rosenthal RJ. Indications for sleeve gastrectomy as a primary procedure for weight loss in the morbidly obese. Journal of gastrointestinal surgery : official journal of the Society for Surgery of the Alimentary Tract. 2008;12(4):662-7.
6.Cerutti D, Guilpain P, Schiffmann A, Riviere S, Carra C, Labauge P, et al. [Serious clinical manifestations of vitamin deficiency after a "sleeve" gastrectomy: role of psychogenic anorexia]. La Revue de medecine interne / fondee par la Societe nationale francaise de medecine interne. 2014;35(9):609-12.
7.Jeong HJ, Park JW, Kim YJ, Lee YG, Jang YW, Seo JW. Wernicke's Encephalopathy after Sleeve Gastrectomy for Morbid Obesity - A Case Report. Annals of rehabilitation medicine. 2011;35(4):583-6.
8.Makarewicz W, Kaska L, Kobiela J, Stefaniak T, Krajewski J, Stankiewicz M, et al. Wernicke's syndrome after sleeve gastrectomy. Obesity surgery. 2007;17(5):704-6.
9.Moize V, Ibarzabal A, Sanchez Dalmau B, Flores L, Andreu A, Lacy A, et al. Nystagmus: an uncommon neurological manifestation of thiamine deficiency as a serious complication of sleeve gastrectomy. Nutrition in clinical practice : official publication of the American Society for Parenteral and Enteral Nutrition. 2012;27(6):788-92.
10.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. European journal of clinical nutrition. 2012;66(5):645-7.
11.Sharabi S, Bisharat N. Wernicke encephalopathy after sleeve gastrectomy. The Israel Medical Association journal : IMAJ. 2012;14(11):708-9.
12.Saab R, El Khoury M, Farhat S. Wernicke's encephalopathy three weeks after sleeve gastrectomy. Surgery for obesity and related diseases : official journal of the American Society for Bariatric Surgery. 2014;10(5):992-4.
13.Skeen MB. Neurologic manifestations of gastrointestinal disease. Neurologic clinics. 2002;20(1):195-225, vii.
14.Chaves LC, Faintuch J, Kahwage S, Alencar Fde A. A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. Obesity surgery. 2002;12(3):328-34.
15.Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. Journal of neurology, neurosurgery, and psychiatry. 1986;49(4):341-5.
16.Giusti V, Suter M, Heraief E, Gaillard RC, Burckhardt P. Effects of laparoscopic gastric banding on body composition, metabolic profile and nutritional status of obese women: 12-months follow-up. Obesity surgery. 2004;14(2):239-45.

17.Watson WD, Verma A, Lenart MJ, Quast TM, Gauerke SJ, McKenna GJ. MRI in acute Wernicke's encephalopathy. Neurology. 2003;61(4):527.
18.Talwar D, Davidson H, Cooney J, St JRD. Vitamin B(1) status assessed by direct measurement of thiamin pyrophosphate in erythrocytes or whole blood by HPLC: comparison with erythrocyte transketolase activation assay. Clinical chemistry. 2000;46(5):704-10.
19.Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. AJR American journal of roentgenology. 1998;171(4):1131-7.