Nutritional deficiency after gastric bypass: diagnosis, prevention and treatment

C. Poitou Bernert*, C. Ciangura, M. Coupaye, S. Czernichow, J.L. Bouillot, A. Basdevant

Nutrition and Surgery Departments, Reference Clinic for Obesity, APHP, Hôtel-Dieu, 75181 Paris cedex 04, France

Received 9 July 2006; accepted 7 November 2006
Available online 26 January 2007

Abstract

In recent years, the recourse to obesity surgery to treat morbid obesities has grown. The number of “malabsorptive” interventions, such as the gastric bypass (RYGB: Roux-en-Y gastric bypass) increases each year. The RYGB, which combines two mechanisms promoting weight loss, restriction and malabsorption, has proven its effectiveness in term of weight loss and improvement of obesity-associated co-morbidities. However this intervention involves a profound change in digestive physiology and is the source of nutritional and metabolic complications. The deficits observed most frequently concern proteins, iron, calcium, vitamin B12 and vitamin D. The deficiencies in vitamin B1 are rare but potentially serious. Multidisciplinary follow-up is essential to ensure prevention, diagnosis and treatment of these complications. Based on an analysis of the literature, this article summarizes the various nutritional complications observed after RYGB and the means to diagnose it. It proposes practical recommendations for follow-up, preventive supplementation and treatment of these deficiencies, both generally and in the more specific case of a pregnancy after RYGB.

© 2007 Elsevier Masson SAS. All rights reserved.

Keywords: Roux-en-Y gastric bypass; Malabsorption; Vitamins; Nutritional deficiency; Bariatric surgery; Supplementation; Pregnancy; Review

Résumé

Carences nutritionnelles après by-pass gastrique : diagnostic, prévention et traitement.

 Ces dernières années, le recours à la chirurgie de l’obésité comme traitement des obésités morbides s’est amplifié. Le nombre d’interventions de type « malabsorptives », telles que le gastric bypass (RYGB : Roux-en-Y gastric bypass) augmente chaque année. Le RYGB qui associe deux mécanismes favorisant la perte de poids, la restriction et la malabsorption, a fait preuve de son efficacité en termes de perte de poids et d’amélioration des comorbidités. Cependant, cette intervention entraîne une modification profonde de la physiologie digestive à l’origine de complications nutritionnelles et métaboliques. Les déficits les plus fréquemment observés concernent les protéines, le fer, le calcium, la vitamine B12 et la vitamine D. Les carences en vitamine B1 sont rares mais potentiellement graves. Le suivi multidisciplinaire est indispensable pour assurer le dépistage, le diagnostic et la prise en charge de ces complications. Fondé sur une analyse de la littérature, cet article résume les différentes complications nutritionnelles observées après RYGB et les moyens pour en faire le diagnostic. Il propose des recommandations pratiques de suivi, de supplémentation préventive et de prise en charge des carences avérées, de façon générale et dans le cas plus spécifique d’une grossesse après RYGB.

© 2007 Elsevier Masson SAS. All rights reserved.

Mots clés : Bypass gastrique Roux-en-Y ; Malabsorption ; Vitamines ; Déficits nutritionnels ; Chirurgie bariatrique ; Suppléments ; Grossesse ; Revue générale

* Corresponding author. Nutrition Department, Hôpital-Dieu Hospital, 1, place du Parvis-Notre-Dame, 75004 Paris, France.
E-mail address: c.poitou.bernert@free.fr (C. Poitou Bernert).

1262-3636/$ - see front matter © 2007 Elsevier Masson SAS. All rights reserved.
doi:10.1016/j.diabet.2006.11.004

© 2019 Elsevier Masson SAS. Tous droits réservés. - Document téléchargé le 22/01/2019 Il est interdit et illicite de diffuser ce document.
For a better comprehension of the text, here are a few definitions:

Gastric bypass: gastro-jejunal short-circuit named in the literature as the Roux-en-Y Gastric Bypass (RYGB).

ANC: Apports Nutritionnels Conseillés (French advised nutritional intake). The ANC cover the needs of 97.5% of a given population as detailed in Table 3 [1]. Their values are very close from those of the recommended daily allowance (RDA) which are referred to in most publications. ANC will further used in the text.

1. Introduction

In recent years, surgical treatments of morbid obesity have greatly increased worldwide, notably in France where 17,000 operations a year were performed in 2001–2002, compared to only 2000 in 1995 [2,3]. Several surgical techniques are available, but in France, gastric banding is the most frequently used [2]. However, due to increasing “malabsorptive” operations, such as RYGB, experts were prompted to develop practical recommendations regarding these procedures [4,5].

RYGB combines two methods, restriction and malabsorption (Fig. 1). The superior part of the stomach is cut to provide a small additional pocket of 20–30 ml, which in return leads to a quantitative decrease in nutritional intake. The inferior part of the stomach and the duodenum stay in place so they are of no more use to the crossing food, but they still secrete substances important for digestion (chlorhydric acid, pancreatic enzymes, biliary salts, hormones...). This is how a certain degree of food maldigestion and malabsorption contributes to weight loss.

The efficacy of the RYGB technique has been proven in terms of weight loss and improvement of comorbidities [6,7]. However this intervention is not without risk, and a frequent short-term and long-term follow-up is required for several reasons. Firstly, there are potential surgical complications (leaks, fistula, obstruction, anastomotic stenosis, internal hernia etc.) particularly in the case of patients with acute digestive symptoms. In addition, it is essential to follow-up on comorbidities, and to adapt treatments, in particular antidiabetic ones. Finally, the operation might have a deep psychological and social impact on patients, which then require a multidisciplinary support. In any case, the operation results in important changes in digestive physiology: for example, non acid reflux, exclusion of the duodenum and the proximal jejunum (1). The superior part of the stomach’s inferior part results in a decreased secretion of gastric acid, sometimes required to absorb vitamins and minerals (B12 and iron) (2). Dietary restrictions are also accompanied by duodeno-jejunal malabsorption related to the short-circuit (3). The duodenum is the main absorption site for calcium, iron and vitamin B1 (thiamin). In addition, asynergia occurs between the bolus and the bilo-pancreatic secretions in the common portion of the intestine (4).

In publications, the most frequent deficiencies after RYGB include iron, vitamin B1, folates (vitamin B9), vitamin D and calcium [8,12-15].

2. Nutritional deficiencies and physiopathology mechanisms

2.1. Macronutriments

2.1.1. Proteins

Although 50% of protein absorption takes place in the duodenum, the remaining proteins are absorbed in the small intestine [16]. Several mechanisms can lead to protein deficiency after RYGB. An intake deficiency might be caused by disgust for meat, difficulty to masticate or protein intolerance. A

Fig. 1. The physiopathological mechanisms which explain the deficiencies after realization of RYGB.

1/ Deficiency of dietary intake. 2/ Diminution of gastric secretions. 3/ Exclusion of the duodenum and the proximal jejunum. 4/ Asynergia between bolus and bilo-pancreatic secretions.
decrease in enzyme secretions, such as pepsinogen (precursor of pepsin) and the pancreatic enzymes, might also be involved [17]. Finally, the time of contact between pepsin secreted in the stomach or the other digestive enzymes, and the protein bolus is reduced, as is the intestinal absorption surface (with the exclusion of the duodenum).

This deficiency in proteins may cause important clinical repercussions: deterioration of general state of health, muscle weakness with loss of muscle mass, anomalies of the skin, mucosa and nails (alopecy, striated nails, dermatitis, hypopigmentation), edema.

Although cases of severe protein malabsorption have been described in bilio-pancreatic derivations such as duodenal switches and Scopinaro interventions [18–20], only few authors mention the protein deficit after RYGB [12], those deficits mostly being related to deficient intake [21]. As much as 13% of the patients with an IMC > 50 kg/m² experienced hypoalbuminemia within 2 years after distal RYGB, the severity of hypoalbuminemia sometimes requiring artificial nutrition. In comparison, no protein deficit was noted after RYGB in which the handle length did not exceed 150 cm [22,23]. This difference was confirmed in another study that showed a 5.8% rate of protein deficit after a distal RYGB, but none after standard RYGB [24]. A rate of 4.7% was also reported in a study on 237 patients after RYGB [25]. These deficits appear within the first 2 years following the intervention, and in more than half of the patients they appear during the regression phase of a complication (stenosis, vomiting), a related disease or an important inflammation. Skroubis et al. [26] reported a 1.3% frequency of hypoalbuminemia in study on 79 patients. In general, if protein deficiencies remain a rare event after RYGB, impaired by exogenic factors, they can be serious and significantly increase morbidity and mortality. Prevention as well as a nutritional follow-up is thus essential.

2.1.3. Lipids

After RYGB, biliary salt secretions and lipolytic enzymes are decreased because lipids pass directly in the jejunum [8]. There is then a reduction in the hydrolysis of the food lipids and a reduction in the formation of micelles leading to a reduction of lipid absorption. To our knowledge very few data are available on the specific consequences of RYGB on lipid malabsorption, contrary to the data concerning jejunoidal bypass or bilio-pancreatic derivations which show major lipid malabsorption, steatorrhea, invalidating chronic diarrhea and reduced liposoluble vitamins (A, D, E, K) absorption. A team studied the effect of gastrectomy (partial or total) followed by reconstruction of a Roux-en-Y loop and showed that for the majority patients, this intervention is accompanied by a pathological respiratory test with carbon14-marked triolein thus signing lipid malabsorption [28,29].

2.2. Micronutrients

2.2.1. Vitamin B12

Vitamin B12 or cobalamin is constitutionally linked to food proteins of animal origin. Digestion starts with the separation of B12 and food by an acid mechanism of hydrolysis. Vitamin B12 binds then to gastric (R) and salivary (Rs) protein. The pancreatic enzymes dissociate the connection vitamin B12-Rs in the duodenum. Then vitamin B12 is transported thanks to its connection with intrinsic factor FI (secreted by the parietal cells of the stomach, mainly in the antrum). Finally the B12-FI complex is fixed on the receiver at the end of the ileon. After RYGB the secretion of hydrochloric acid in the small gastric pocket is weak. This creates a defect of cleavage between vitamin B12 and its food protein support. The exclusion of the stomach and the duodenum prevents the link between FI and B12. The authors nevertheless described that the Schilling test is normal after RYGB, suggesting that the secretion of FI persists [30].

In the literature, the prevalence of vitamin B12 deficit is estimated between 12% and 70%, and occurs most of the time after the first year following RYGB [31–36]. Overall, in studies comprising 957 patients, the estimated frequency is 25% in the first 2 years [37]. The incidence increases later on, to about 36–70% [22,26,38,39]. The lower frequency of a deficit in the first years can be explained by the importance of the bodies reserves (2000 μg) compared with the daily needs (2–3 μg/day). Nevertheless in certain situations, in particular when RYGB is performed after a first bariatric surgery (gastric banding for example), it is possible to observe earlier deficits in B12.

Some cases of megaloblastic anemia were reported but are rare [40–42]. Out of approximately 350 patients, only 0.8% of the patients presented macrocytosis without anemia and none developed megaloblastic anemia [31]. To our knowledge no cases of neurological symptoms due to a deficit of B12 after RYGB have been reported.

2.2.2. Vitamin B9 (folates)

Deficits in folates are explained primarily by a deficiency of dietary intake (fruits and vegetables), because even if physiological absorption takes place in the proximal small intestine, folates may be absorbed throughout the whole intestine.

Folate deficiencies seem less frequent than deficiencies of vitamin B12. This prevalence is variable from one study to
another [31,39,43,44], about 20% at the end of 1 year on average [37].

2.2.3. Vitamin B1 (thiamin)

The food sources of B1 are cereals, meats (in particular pork), fish and eggs, then vegetables and fruits and finally dairy products. B1 is absorbed in the duodenum’s acid medium. The deficits observed are due to the combination of a reduction in gastric acid secretion and food intake, and are worsened by vomiting [37,45–49].

The prevalence is weak, about 1% at the end of 1 year on average [37]. On the whole of bariatric surgeries (more than 168,000), only 29 cases of deficiency in symptomatic B1 were found (0.0002%) [50]. In a study among patients treated with a multivitamin providing 100% of the RDA (or 1–1.3 μg/day), no case of deficiency was reported [51].

The consequences of a deficiency in B1 are serious (Beriberi). Irreversible polyneuropathy, and Gayet-Wernicke’s encephalopathy have been reported [37,50,52]. Gayet-Wernicke’s encephalopathy is recognizable by the traditional triad of ophthalmologic abnormalities with nystagmus, a deterioration of cerebral functions, and cerebellar ataxia. Other less specific symptoms can also be present: anorexia, vomiting, nausea, apathy, psychomotor deceleration, marked asthenia, bilateral paraesthesia and muscular weakness. No cases of cardiac Beriberi have been described after RYGB.

Vitamin B1 deficiency must be suspected if given even the slightest clinical doubt. In all cases, suspected or proven by B1 measurement, the administration of parenteral B1 (50–100 mg) corrects the deficit.

2.2.4. Liposoluble vitamins (A, E, K)

In theory, the absorption of liposoluble vitamins would be lower after RYGB because of poor lipid absorption. In fact, the frequency of deficits in vitamins A, E and K is very low after RYGB [51,53–57] and the clinical consequences are not significant.

On the other hand, 4 years after a bilio-pancreatic diversion, the prevalence of vitamin A deficit in was estimated at 69%, vitamin K at 68%, and vitamin E at 4% [58]. Ophthalmologic complications (night eye trouble, xerophthalmia) due to deficiencies in vitamin A were described after malabsorptive interventions such as bilio-pancreatic derivations. Only one case of vitamin K deficit has been reported after bilio-pancreatic diversion [58]. Prudence recommends that patients taking anticoagulants (antivitamin K) must be closely monitored after RYGB.

2.2.5. Calcium and vitamin D

Calcium deficits are explained by a deficit of dietary intake (aggravated by lactose intolerance), and reduction in the absorption which usually takes place in the duodenum and the proximal jejunum. Vitamin D is liposoluble and its absorption in the small intestine (jejunum and ileon) is decreased because of lipid malabsorption [59]. However after RYGB, deficiencies in calcium and vitamin D are less frequent and less severe than after bilio-pancreatic derivations [60].

The deficiencies in vitamin D and calcium after RYGB can cause hyperparathyroidism [61–64]. Goode et al. [59] showed that hyperparathyroidism was more frequent in post menopause patients. The consequence of hyperparathyroidism, particularly in the long run, is an increase in bone remodeling and a decrease in bone mass [62,64–67] which can lead to severe forms of osteomalacia [68]. Coates et al. [65] compared 25 patients 1 year after RYGB who had not been operated on, with obese patients. Signs of bone remodeling were significantly higher, with an increase in the bone resorption, associated with a reduction in the bone mineral density (BMD) in the hip among operated patients.

For these reasons, monitoring of phosphocalcic metabolism (calcium, phosphorus, PTH, alkaline phosphatase, 25-OH vitamin D) is essential after RYGB.

2.2.6. Iron

An iron deficit results from several mechanisms. There is very often a deficiency of dietary intake (red meat). The reduction hydrochloric acid secretion accounts for the low transformation of ferric form (Fe(3+)) to ferrous form (Fe(2+)), which is the absorbable form. Moreover, iron is primarily absorbed in the duodenum. Finally, in menstruating women, iron reserves are limited. Iron deficiencies are the most frequent deficiencies after RYGB. According to the literature, the frequency in the first 2 years varies from 15 to 60% [36,39,44,69], for an average to 33% [70] but is generally higher than 50% among women of childbearing age. The risk of deficiency persists in the long run [26], beyond 7 years after surgery [71]. All these studies also show that iron deficiencies can appear despite regular use of a multivitamin and multimineral supplementation.

The consequence of an iron deficit is ferricprive anemia whose clinical signs are usually: asthenia, dyspnea, paleness, tinnitus, hair loss. On average, the frequency of deficiency anemias whatever the type of deficiency (vitamin B12, iron, folates), is estimated at 30% [70]. In a study of approximately 350 patients, microcytic anemia was observed in 63% of patients with an iron deficit [31], and was confirmed in another study [72].

The clearest sign of iron deficit is a reduction in ferritin, followed by a reduction in serum iron. Worse deficiencies lead to microcytosis (although average globular volume can be distorted by associated deficiencies in B12 and folates), and finally to a reduction in hemoglobin.

2.2.7. Potassium and magnesium

One study described an incidence of hypokalemia raised by 56%. In the majority of cases, there was a diuretic treatment for arterial hypertension [44]. In another study on 150 patients, 6.3% of the subjects presented marked hypokalemia (< 3 mmol/l) [39]. It is thus necessary to use diuretics carefully after the intervention (clinical monitoring of blood pressure, hydration and measurement of plasmatic Na⁺ and K⁺).
Hypomagnesemia was reported by Halverson in 34% of patients [44] while in the study of Amaral et al., no cases were found [39]. To date, no clinical consequence of hypomagnesemia has been described.

The deficiencies of potassium and magnesium were, in all cases, easy to correct by adapted supplements.

2.2.8. Zinc and selenium

The absorption of zinc is dependent on the absorption of lipids which is reduced after RYGB. Low zinc concentrations were observed after biliopancreatic diversion [58]. Moreover deficiencies in dietary intake can appear after restrictive surgery [73]. A study carried out in 14 subjects showed that the plasmatic zinc rates were not modified 6 and 12 weeks after RYGB [74]. Recently Madan et al. [57], showed abnormal zinc levels in 30% of the patients evaluated before RYGB and 36% in the patients after 1-year. In 24 morbidly obese patients studied before and 2 months after RYGBP, it has been shown that the main change was in zinc erythrocyte and urinary concentrations [75]. Long-term evolution of zinc plasmatic rates and the clinical consequences related to zinc deficit have not been studied.

In practice, moderate hair loss is frequently observed among women between 3 and 6 months after the RYGB. The most frequently mechanisms reported are iron, protein and zinc deficiencies, post surgical stress and significant weight loss. However only one study described an improvement of alopecia after treatment with high zinc sulfate supplements after restrictive surgery [76]. The responsibility of a zinc deficit in alopecia after RYGB remains under discussion.

In a study comparing biliopancreatic derivations with or without duodenal switch, the authors found a selenium deficit (< 0.7 μmol/l) in 14.5% of the cases, without any clinical consequence described [77]. Madan et al. [57] described a deficit in selenium for 3% of the patients 1-year after surgery. No study described the possible clinical repercussion, in particular in the long run, of such a deficit.

3. Diagnosis of the deficiencies and nutritional follow-up

Taking into account the frequency of the nutritional deficits among obese patients (vitamin D, B12, folates, iron), it is imperative to carry out a complete nutritional assessment before surgery. After surgery, we propose a nutritional check-up after 3 months, then every 6 months in the first 2 years, then at least once per year after 2 years.

The assessment should attempt to seek clinical signs of deficiency (Table 1). An example of the complementary examina-

<table>
<thead>
<tr>
<th>Vitamins/minerals</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>Eyes: Night blindness; Keratomalacia</td>
</tr>
<tr>
<td></td>
<td>Skin: Hyperkeratosis (thickening and roughness of skin); Xerosis, acne, dry hair; Fatigue insomnia</td>
</tr>
<tr>
<td>Vitamin B1 (thiamin)</td>
<td>Cardiovascular system: Congestive heart failure; Nervous system: Confusion, irritability, memory loss, nervousness, numbness of hands and feet, pain sensitivity, poor coordination, weakness</td>
</tr>
<tr>
<td></td>
<td>Intestinal tract: Constipation, intestinal disturbances, loss of appetite</td>
</tr>
<tr>
<td>Vitamin B6 (pyridoxine)</td>
<td>Skin and mucosa: Loss of hair, mouth lesions, facial oiliness, eye inflammation, acne; Other: Depression, dizziness, fatigue, impaired wound healing, irritability, loss of appetite, nausea</td>
</tr>
<tr>
<td>Vitamin B9 (folic acid)</td>
<td>Hematopoiesis: Anemia; Nervous system: Apathy, fatigue, headaches, insomnia, neural tube defects in fetus, weakness</td>
</tr>
<tr>
<td></td>
<td>Intestinal tract: Diarrhea, loss of appetite</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>Hematopoiesis: Macrocytosis anemia; Nervous system: Fatigue headaches, irritability, loss of vibration sensation, spinal cord degeneration, numbness, paresthesia</td>
</tr>
<tr>
<td></td>
<td>Intestinal tract: Diarrhea, constipation</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>Skin and mucosa: Bleeding gums, easy bruising impaired wound healing, joint pains, loose teeth; Other: Irritability, malaise, tiredness, depression</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>Bone: Osteomalacia, osteoporosis, joint and bone pains</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Hemostasi: Bleeding disorders</td>
</tr>
<tr>
<td>Vitamin PP (niacin)</td>
<td>Skin: Dermatitis; Intestinal tract: Diarrhea; Nervous system: Dementia</td>
</tr>
<tr>
<td>Iron</td>
<td>Hematopoiesis: Anemia, microcytosis; Skin and mucosa: Brittle nails, inflamed tongue, mouth lesions, pruritus; Intestinal tract: Constipation, Other: Confusion, depression, dizziness, fatigue, headaches</td>
</tr>
<tr>
<td>Zinc</td>
<td>Skin: Acne, eczema, white spots on nails, brittle nails, hair loss; Taste: Loss of sense of taste; Other: Depression, fatigue, immune impairment, anemia, irritability, lethargy, male infertility</td>
</tr>
<tr>
<td>Selenium</td>
<td>Increased incidence of cancer, pancreatic insufficiency (inability to secrete adequate amounts of digestive enzymes), immune impairment, liver impairment, male sterility</td>
</tr>
</tbody>
</table>
Follow-up after RYGB

<table>
<thead>
<tr>
<th>Before surgery</th>
<th>3 months</th>
<th>6 months</th>
<th>12 months</th>
<th>18 months</th>
<th>24 months</th>
<th>Annually</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, symptoms of vitamins/minerals deficiency</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Blood ionogram, calcemia</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Blood cell count</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Iron, ferritin</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Albumin</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Vitamins B1, B9, B12</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Vitamin D, alkaline phosphatase, PTH</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Osteodensitometry</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

Supplementary measurements should be realized: at 3 months and at 6 months in case of deficit observed before surgery, 3 months after supplementation start in the case of specific deficit and in the case of pregnancy project.

- If weight loss > 10%.
- If results are abnormal, evaluation must be realized annually.

4. Prevention and treatment of the nutritional deficiencies after RYGB

No controlled trial exists to determine the type of supplements and the dosages to be prescribed after RYGB. The majority of the reviews published on post-RYGB deficiencies recommend a multivitamin supplement providing 100% of the ANC (Table 3) [8,51].

However, in France, no marketed supplement available covers all the requirements. In addition, one does not know the proportion of each vitamin or minerals introduced which is really absorbed, the principal site of absorption remaining the excluded duodenum. It is thus important to know the composition of the available multivitamins (Table 3), and to adapt prescription according to plasmatic measurements carried out during the follow-up and to the compliance of the patient to the galenic form (tablets, syrup, drinkable solution).

It should be noted that multivitamin supplements are not refunded and represent a considerable monthly cost. This information must be given to the patient before the intervention.

Considering proteins, the patients should consume approximately 0.8 g/kg of proteins daily. The nutritional level is evaluated by the food consumption survey, but also by the evolution of specific biological markers (albumin, prealbumin). In the event of insufficient dietary intake, it is possible to prescribe a hyperprotidic supplement (Table 5).

It is recommended to prescribe supplements in the form of 1200 mg calcium and 8 μg (that is to say 320 UI) of vitamin D per day for 6 months, did not correct hyperparathyroidism and BMD, compared to a control group of 13 women not taking supplements.

It thus appears important to evaluate the bone osteodensitometry before surgery in order to identify patients at risk for aggravation of osteopenia. Those patients should receive a reinforced supplement and a specialized follow-up.

Multivitamin and multimineral supplements alone are not sufficient to meet the iron requirements. It is thus recommended to prescribe additional iron. The amount is about 50–100 mg/day. In menstruating women, an even higher amount is recommended [69,72,80]. A galenic form with vitamin C added which increases the absorption of iron can be given [72]. It should be noted that orally taken iron is sometimes limited by intestinal disorders (abdominal pains, diarrheas). When hemoglobin concentration is below 10 g/dl, it is possible to give the supplements parenterally. The iron and multivitamins must be taken at different times, so the iron does not interfere with the absorption of calcium, magnesium and zinc.

The recommended daily allowance (ANC) of folates is 300–330 μg (Table 3). For Boylan et al., no case of deficiency appeared after a year of taking supplements providing 100% of the ANC. The majority of the multivitamins contain a sufficient quantity of folates (Table 3) to ensure a normal plasmatic level [31,81]. A dose of 1 mg/day seems sufficient to treat existing deficiencies [51].

For vitamin B12, the multivitamin supplements contain approximately 100% of the ANC, that is to say only 2–3 μg/ day. The majority of the studies showing a frequency of deficit in B12 higher than 30%, were carried out using this type of supplement. Provenzale et al. [82] estimated a deficit in B12 of approximately 30% of the patients despite increasing the supplement’s quantity to 10 μg/day. There are thus arguments for saying that a standard multivitamin supplement (Table 3) is not sufficient to maintain a normal plasmatic concentration of B12. Other authors noted that the patients treated with 100 μg/day did not develop a deficiency [51] and that in the majority of the cases, an amount higher than 350 μg/day corrected the plasmatic rates of vitamin B12 [19,32,79].

The need for a preventive treatment is under discussion, and when it is carried out, the methods of administration (monthly...
or daily) and the amounts prescribed are variable from one study and team to another. Some authors recommended giving B12 in addition to one multivitamin supplement either in injectable form, or in crystalline oral form able to be absorbed after RYGB [35,83]. In a study that looked at more than 1500 RYGB over 20 years, who received multivitamins as the only preventive treatment, the team of Brolin identified no ascribable notable consequences of a B12 deficit such clinical signs, and megaloblastic anemia. It thus proposes carrying out a check-up every 6 months the first 2 years, then annually, and to supplement orally or parenterally, only in the event of a proven deficit [84].

According to these studies and our own experience, several courses of action appear possible to us:

- in the absence of a deficit (Table 4):
  - to systematically supplement B12 when it is in the low range of the normal values, or when RYGB follows gastric banding, because the reserves are very often lowered due to the prior deficiency of dietary intake;
  - not to supplement in the other cases, but to monitor the level of B12 (Table 2);
- in the event of a deficit (Table 5):

### Table 3
The main forms of multivitamin supplements

<table>
<thead>
<tr>
<th>Vitamins</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANC Pregnant Women</td>
</tr>
<tr>
<td>Beta carotene = provitamin A (mg)</td>
</tr>
<tr>
<td>A (retinol) (µg)</td>
</tr>
<tr>
<td>B1 (thiamin) (mg)</td>
</tr>
<tr>
<td>B2 (riboflavin) (mg)</td>
</tr>
<tr>
<td>B5 (pantothenic acid) (mg)</td>
</tr>
<tr>
<td>B6 (pyridoxine) (mg)</td>
</tr>
<tr>
<td>B8 (biotin) (µg)</td>
</tr>
<tr>
<td>B9 (folic acid) (mg)</td>
</tr>
<tr>
<td>B12 (cyanocobalamin) (µg)</td>
</tr>
<tr>
<td>PP (B3) (nicotinamide) (mg)</td>
</tr>
<tr>
<td>E (α tocopherol) (mg = UI)</td>
</tr>
<tr>
<td>C (ascorbic acid) (mg)</td>
</tr>
<tr>
<td>H (mg)</td>
</tr>
<tr>
<td>D3 (cholecalciferol) (µg (UI))</td>
</tr>
</tbody>
</table>

### Minerals

<table>
<thead>
<tr>
<th>Minerals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium (mg)</td>
</tr>
<tr>
<td>Iodine (µg)</td>
</tr>
<tr>
<td>Magnesium (mg)</td>
</tr>
<tr>
<td>Zinc (mg)</td>
</tr>
<tr>
<td>Iron (mg)</td>
</tr>
<tr>
<td>Copper (mg)</td>
</tr>
<tr>
<td>Potassium (mg)</td>
</tr>
<tr>
<td>Manganese (mg)</td>
</tr>
<tr>
<td>Molybdenum (µg)</td>
</tr>
<tr>
<td>Selenium (µg)</td>
</tr>
<tr>
<td>Chromium (µg)</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
</tr>
</tbody>
</table>

### Galenic presentation

<table>
<thead>
<tr>
<th>Box</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
</tr>
<tr>
<td>Tablet</td>
</tr>
<tr>
<td>Drop</td>
</tr>
<tr>
<td>Syrup</td>
</tr>
<tr>
<td>Capsule</td>
</tr>
</tbody>
</table>

### Cost/month (euros)

| 8–10 | 12–14 | 13–15 | 13–14 | 15–17 |
### Table 4
Examples of supplementation after RYGB

<table>
<thead>
<tr>
<th>Before surgery</th>
<th>After surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>If specific deficiency: cf. Table 5</td>
<td></td>
</tr>
<tr>
<td>Polyvitamin: Vivamyne Multi® 1 tablet per day or Azinc optimal 2 capsules per day</td>
<td>If tablets can not be swallowed</td>
</tr>
<tr>
<td>Tardyferon® (ferrous sulfate + ascorbic acid): 1 tablet per day except for menstruating women (2 per day)</td>
<td>Alvityl® 3 tablets per day to suck or Alvityl Syrup® 3 tea spoons per day or Hydrosol polyvitamine® minimum 25 drops per day</td>
</tr>
<tr>
<td>Calculrat® 1000 mg (calcium carbonate): 1 tablet per day to suck or Ostram® 1200 mg (calcium carbonate): 1 tablet per day</td>
<td>Orocal D3® (calcium carbonate 500 mg + vitamin D 400 UI): 2 tablets per day</td>
</tr>
<tr>
<td>Uvedose® (colecalciferol) 100,000 UI to be swallowed once. Repeat after 3–6 months</td>
<td></td>
</tr>
<tr>
<td>Vitamin B12®: 1 tablet (250 μg)/day if plasmatic measurement is in the low end of normal range</td>
<td></td>
</tr>
<tr>
<td>If tablets can not be swallowed</td>
<td></td>
</tr>
</tbody>
</table>

### Table 5
Management of deficiencies after RYGB

<table>
<thead>
<tr>
<th>Protein</th>
<th>铁</th>
<th>硒</th>
<th>益生元</th>
<th>口服补充</th>
<th>注射补充</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fortimel® 200 ml (20g of proteins, 260 kcal) or Resource Nutridoral® or Protein+® 200 ml (19 g of proteins, 250 kcal). Daily quantity depends on dietary questionnaire</td>
<td>Ferro-Grad® (ferrous sulfate) 105 mg of iron and 500 mg of vitamin C per tablet</td>
<td>Ferrostrane® (sodium feredetate) 34 mg per teaspoon</td>
<td>Zymad® (colecalciferol) 200,000 UI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream: crème Resource HP® (12.5 g of proteins, 167 kcal)</td>
<td>Tardyferon® (ferrous sulfate) 80 mg per tablet</td>
<td>Venofer® (ferrous sucrose): one perfusion IV of 100–200 mg/month until deficiency correction</td>
<td>Vitamine D3 BON® (colecalciferol) 200,000 UI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>Fumafer® (ferrous fumarate) 66 mg per tablet</td>
<td>if 25OHD3 &lt; 20 ng/ml and/or if increased PTH</td>
<td>Uvedose® (colecalciferol) 100,000 UI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>Tadyferon B9® (ferrous sulfate + folic acid): 50 mg of iron + 350 μg of B9 per tablet</td>
<td></td>
<td>Benerva® or Bevitine® (thiamin) 250 mg: 2 tablets per day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>Ferrostrane® (sodium feredetate) 34 mg per teaspoon</td>
<td>if the deficit is not corrected after 3 months of oral supplements</td>
<td>Vitamin B12® 250 μg: 2 tablets per day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>Venofer® (ferrous sucrose): one perfusion IV of 100–200 mg/month until deficiency correction</td>
<td>* anemia or macrocytosis is present</td>
<td>Vitamin B12® 1000 μg 1 injection IM/month until deficiency correction then a dose (orally) between 250 and 500 μg/day (1–2 tablets) if</td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>if the deficit is not corrected after 3 months of oral supplements</td>
<td>* anemia or macrocytosis is present</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>if the patient does not appreciate the liquid consistence, it could be proposed as a cream:</td>
<td>* patient refuses oral form</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
It is often associated with a vitamin B5 (example Bepanthène®) prescription a multivitamin containing zinc (Table 3). In practice, 2 per day) and Cystine® B6 2 per day, and a local treatment.

In Table 4, Examples of treatments for the proven deficits are given in Table 5. It is interesting to note that there can be no overdose of vitamin B1, B9, and B12. On the other hand, for vitamins A and D, there is a toxic risk for amounts higher than 10 times the ANC, causing digestive and neurological disorders for vitamin A, and hypercalcemia for vitamin D.

5. The case of pregnancy

5.1. Specific nutritional needs during pregnancy and breast feeding [11]

The specificANCs for pregnant women are detailed in Table 3. The additional protein contribution necessary for foeto-placental growth is estimated at 11 g/day in the third trimester compared to 1.3 g/day in the first trimester.

The consequences of certain specific deficits deserve to be detailed:

- in the event of maternal ferrorrivate anemia, the risks of prematurity and low-weight birth are increased [85,86];
- there is a relation between a maternal deficit in vitamin D and the occurrence of neonatal hypocalcemia, infantile rickets [87], and weak osseous mineralization in childhood [88,89]. Women with the most severe deficits can develop a symptomatic osteomalacia during pregnancy. The supplements recommended are 10 mg/day (400 UI) if begun at pregnancy, or 25 mg/day (1000 UI) if begun in the third trimester, or a single amount of 100,000 UI taken once in the sixth or seventh month;
- the requirements of iodine are increased by 50 μg/day. Iodine deficiencies can cause thyroid hyperplasy, eventually resulting in maternal and fetal goiter. A reduction in the intellectual coefficient was noted in children whose mothers had iodine deficiencies [90];
- the role of folic acid deficit in terms of malformations (neural tube, cleft palate) is well-known [91].

Consequently, during pregnancy, the three priorities are preventing iron, calcium/vitamin D, and folic acid (in particular around the time of conception) deficiencies. It is recommended to systematically supplement women with vitamin D in the sixth or seventh month of pregnancy, with folates (400 μg) in the month preceding conception until the second month of pregnancy, and to increase iodine rich food intake (milk, eggs, fish, salt enriched with iodine). On the other hand there is no benefit, and no recommendation in France for a systematic iron or calcium supplement in the absence of a deficit.

5.2. Pregnancy and RYGB

Generally, it is recommended that women wait approximately at least 18 months after surgery before beginning a pregnancy. This period is only a suggestion, since weight stabilization and correction of the various deficits are more important than the length of time. There are few cases in literature concerning specific deficiencies during pregnancy after RYGB.

A case of B12 deficiency in a children exclusively breastfed by a women having had a RYGB has been reported [92]. In the event of anemia due to proven iron deficiency, orally taken iron can be insufficient, making intravenous administration necessary, or even blood transfusions [93]. In two reported cases, intravenous feeding was necessary for women after jejunoileal bypass: a 24-year-old diabetic [94], and a 33-year-old woman having twins [95]. An increase in cases of malformations of the neural tube was reported [96–98]. Even if no study evaluated the risks of iodine deficiencies in RYGB patients, it is advisable to monitor it using the 24 hour urinary excretion test. Indeed, the absorption of iodine takes place in the stomach and the small intestine and hypochlorydria decreases iodine absorption.

Some observations carried out in the general population on a small number of subjects suggested that deficiencies in certain nutrients and vitamins (zinc, selenium, vitamin A, vitamin B6) played a role in causing fetal complications [99]. There is no data on the monitoring and the supplements to be prescribed for pregnant women after RYGB. By prudence, we propose a broad evaluation of the deficits at the beginning of pregnancy among RYGB women and a systematic reinforcement of supplements (Table 4).

The pre-pregnancy assessment proposed is as follows:

- blood cell count;
- iron, ferritin;
- vitamins B1, B6, B12, B9;
- calcium and vitamin D;
- vitamins A and E;
- zinc, selenium, magnesium;
- 24 h urinary excretion of iodine;
- albumin.

If there is a proven deficit, a targeted treatment must be prescribed and must be monitored monthly, to check for overdoses and to adapt doses. In the absence of a deficiency, we propose monitoring the blood cell count, iron, ferritin, calcemia and vitamin D every trimester.
6. Conclusion

The large increase in the number of gastric surgeries and in particular malabsorptive techniques such as RYGb, as well as the nutritional deficiencies observed after this type of surgery require rigorous medical follow-up and multi-disciplinary collaboration between nutritionists, surgeons, dieticians and obstetricians.

Based on the data in the medical literature, this synthesis suggests practical guidelines for vitamin supplements and post-operative follow-up, but well-designed randomized tests are necessary to validate these practices.

References


Coates PS, Fernstrom JD, Fernstrom MH, Schauer PR, Greenspan SL. Gastric bypass surgery for morbid obesity leads to an increase in bone turnover and a decrease in bone mass. J Clin Endocrinol Metab 2004;89:1061–5.


