Caso clínico

Severe vitamin A deficiency after malabsorptive bariatric surgery

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Abstract

Vitamin A deficiency may occur after malabsorptive bariatric surgery. However, it rarely entails important functionally limiting symptoms. We present the case of a woman who underwent bariatric surgery and developed ocular dryness, xeroderma and hearing loss due to severe vitamin A deficiency. We illustrate an outstanding and exceptional case of the consequences of an excessive and uncontrolled malabsorption.


Abreviaturas

BMI: Body mass index.
%WL: Percentage weight loss.
RBP: Retinol binding protein.

Introduction

Nutrient and vitamin deficiencies may occur after bariatric surgery due to weight loss and malabsorption.1 Although it has been described that the prevalence of insufficient levels of fat-soluble vitamins may reach more than 60% after malabsorptive procedures,2 deficits are usually mild and it is frequently possible to overcome, and even prevent them, with adequate nutrient and multivitamin oral tablets.3 However, exceptionally, when excessive and uncontrolled malabsorption develops, deficiencies may entail important clinically limiting signs and symptoms. We present a case of severe vitamin A deficiency after malabsorptive bariatric surgery.

Case report

A 48-year old woman underwent Roux-en-Y gastric bypass (100 cm alimentary limb and 80 cm biliopancreatic limb) with a preoperative weight of 128 kg and body mass index (BMI) of 53.3 kg/m². Due to insufficient weight loss one year later (103 kg, BMI 42.9 kg/m², % weight loss [%WL] 19.5%), she underwent a second procedure in which a 100-cm longer Roux limb was defined, obtaining a total length of 200 cm. The patient followed correct diet and recommendations, and no postoperative complications occurred. However, despite repeated lifestyle modifications, she was still unable to achieve sufficient weight loss, and after six years, she had regained weight. She was willing to undergo a third bariatric surgery, so after careful and multidisciplinary consideration, she was programmed for a classic duodenal switch. The 200-cm alimentary limb was sectioned proximal to the jejuno-jejunal anastomosis and was reanastomosed 100 cm away from the caecum, leaving an alimentary + common limb of 300 cm and a common limb of 100 cm. The immediate
postoperative period was uneventful, the patient reached correct tolerance to normal diet in a few weeks, and routine oral nutrient and vitamin supplements were prescribed. Prior to the first bariatric procedure, fat-soluble vitamins and minerals were not routinely evaluated in our hospital; but before undergoing the third surgery, the patient presented normal levels of these parameters, as well of serum proteins (table I).

Subsequent to this third procedure, she started to lose weight at an increased rate, and she reached 79 kg (BMI 32.9 kg/m²) by 12 months, and 69 kg (BMI 28.7 kg/m²) at 2 years’ follow-up. At this time, she complained of increased vomiting, steatorrhea and diarrhea (8-10 times/day), without abdominal pain or anorexia, which partially responded to antibiotic prescription. She recalled progressive asthenia and generalized weakness and she also described the gradual development of lower limb edema, skin dryness, loss of hearing, dry and itchy eyes, and visual disturbances, especially evident at night. She acknowledged correct adherence to routine oral supplements, but dietary intake was limited in quantity, especially regarding proteins (less than 60 g/day).

Physical examination revealed generalized dryness of skin and mucosa. Erythematous desquamative patterns and eczematous plaques were remarked over legs, forearms, fingers, and lower back, and several excoriations due to scratching were evident (fig. 1). Abdominal examination revealed only a slight disturbance at palpation of the site of the surgical scar, and lower limb bilateral edema was evident. Biochemical and nutritional laboratory parameters at that time showed anemia, hypoproteinemia and vitamin-nutrient deficiencies (table I), particularly vitamin A, whose levels significantly decreased to 0.07 mg/L (0.3-1), and a ratio vitamin A/RBP of 0.25 mg/g RBP (0.8-1.2). Ophthalmological evaluation evidenced loss of visual acuity and bilateral keratoconjunctivitis sicca, which improved with preservative-free artificial tears. Dermatologic specific evaluation concluded that lesions mimicked xeroderma.

Increasing oral supplements of vitamins A and E to 50,000 IU/day and 400 mg/day, respectively, together with doubling doses of multivitamin and mineral...
supplements and optimizing protein intake, did not restore nutrient disturbances completely (table I): vitamin A 0.15 mg/L (0.3-1), ratio vitamin A/RBP 0.76 mg/g RBP (0.8-1.2). Clinical alterations persisted and she continued to lose weight (57 kg, BMI 23.7 kg/m²). Hospitalization was programmed for administration of total parenteral nutrition, including albumin, vitamins and minerals, in three different occasions, at 2-month intervals. With this intensive medical approach, vitamin and micronutrient deficiencies, as well as hypoproteinemia, were partially resolved, but only for a short period of several weeks. A slight amelioration of the patient’s generalized weakness, asthenia, increased bowel habit, cutaneous lesions and sensory symptoms was acknowledged, but clinical manifestations reappeared early after she was dismissed.

After meticulous evaluation once again, the patient underwent surgery for reconversion of her previous malabsorptive technique, shaping a longer common limb, with an unremarkable immediate postoperative period. She then followed oral supplementation with vitamin A, E, B-complex and micronutrients at doses 3-fold higher than what is routinely prescribed. She regained weight to reach 62 kg (BMI 25.8 kg/m²) after 6 months, and signs and symptoms of nutrient deficiencies disappeared completely, and laboratory data normalized, including levels of fat-soluble vitamins (table I). Multivitamin and mineral oral supplements were prescribed again at the usual doses, and laboratory data have maintained stable since then.

Discussion

Vitamin A plays an important role in several human functions, such as visual acuity, immunological activity, and cellular proliferation and differentiation. Vitamin A complex includes retinols, beta-carotens and carotenoids, and pool status is usually determined by measuring retinol serum levels, although better estimates are obtained if serum levels of its transporter, i.e. the retinol binding protein (RBP), are also measured, and the ratio vitamin A (retinol)/RBP is calculated. Since vitamin A is transported also by other proteins such as prealbumin, the ratio vitamin A/prealbumin could be equally valid for store estimates.

It has been described that up to 12.5% of patients scheduled for bariatric surgery present vitamin A deficiency due to several factors, including non-alcoholic steatohepatitis and intake of high caloric foods with relatively less quantity of vitamins and minerals. Additionally, after bariatric procedures, vitamin A deficiency may develop due to malabsorption related to exclusion of gastrointestinal segments, decrease in intake, and avoidance of certain types of food, including fat and other sources for vitamin A. It may also be aggravated by other micronutrient deficiencies, especially zinc and iron, which have a prominent interrelationship with vitamin A metabolism, and in fact, combined deficiency of vitamin A and zinc has been associated to inflammation and increased surgical risk.

This is especially relevant in cases of malabsorptive procedures; several studies have reported vitamin A deficiency rates of 60-70% after biliopancreatic diversion, while lower rates around 10% have been reported after Roux-en-Y gastric bypass. The majority of studies describing vitamin deficiencies did not report associated clinically evident manifestations, yet it is possible that they were overestimated. Vitamin levels are generally given as absolute values, rather than in relation to its binding protein, and this would only be useful and trustworthy in the absence of hypoproteinemia. If levels of vitamin A, prealbumin or RBP are decreased, interpretation of retinol levels may be falsely reduced and should, therefore, be considered with caution. However, in the case here presented, both absolute and relative values of vitamin A were clearly under the normal reference range during the period of time in which she experienced symptoms of malabsorption, but they were within the normal range prior to the third surgical approach as well as after bariatric reconversion.

Furthermore, even though there are no standardized recommendations, prophylactic daily use of routine nutrient and vitamin oral supplement tablets that contain around 800 ug of vitamin A generally avoids clinically relevant complications, and manifestations like the ones this patient developed are rarely seen to such an extent. There have been some reports of rare visual symptoms (nyctalopia) or skin disturbances following bariatric surgery, in which nutrient and vitamin deficiencies were observed. But dermatologic lesions consisting of hyperkeratosis and desquamative plaques are exceptionally associated to other systemic symptoms in the bariatric postoperative setting. Recently, Ocon Breton et al. described a patient who progressively developed follicular hyperkeratosis located on the extensor surfaces of extremities and xeroftalmia one year after undergoing biliopancreatic diversion. Clinical manifestations were ascribed to vitamin A insufficiency and after intensive treatment, disturbances disappeared.

However, in our patient, multiple symptoms of deficiency developed acutely and vitamin A reached almost undetectable levels despite intensive supplement treatment. The fact that a common limb of only 100 cm for nutrient assimilation was created in the third bariatric procedure, although effective for weight loss, was followed by the counterpart of excessive malabsorption; this could be corroborated with laboratory data, where severe hypoproteinemia, fat malabsorption, and associated nutrient deficiencies including zinc, selenium, copper and vitamin D were observed. Persistent severe malabsorption occurring in our patient over a prolonged period of time determined such a significant reduction of vitamin A stores as to result in the clinical manifestations here reported.

Although it has been described that vitamin A deficiency can be treated by an approximate three-fold
increase in oral supplements, this was not enough in the case that we present. Neither was total parenteral nutrition sufficient; this patient presented such degree of malabsorption that deficiencies reappeared when parenteral supplements were discontinued. Reconversion of the bariatric procedure to achieve a longer, and therefore less malabsorptive, common limb was necessary for final improvement of the clinical spectrum.

In our experience, not all patients undergoing malabsorptive bariatric techniques develop the same degree of nutrient and vitamin insufficiencies. However, a few patients may respond with uncontrolled insufficient nutrient assimilation despite adequate supplements and follow-up and, in these cases, bariatric reconversion may be a reasonable alternative approach.

Another alternative that could have been considered in this patient instead of surgery would have been periodic intramuscular administration of vitamin A. This has been reported to be effective for improvement of dark blindness in patients with chronic liver disease11 and may be pondered in cases with severe malabsorption not responding to massive vitamin A supplements, and in whom repeated surgery is not feasible. Careful monitoring should, however, be performed subsequently.

We emphasize the severe consequences this patient endured due to poor bowel adaptation to biliopancreatic diversion, which determined an excessive and uncontrolled malabsorption despite adequate adherence to oral supplements and scheduled outpatient monitoring. Reconversion techniques may be useful in severe cases not responding to intensive medical treatment.

References