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Caso clínico

Dermatitis and optic neuropathy due to zinc deficiency after malabsortive bariatric surgery

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Abstract

We present a patient who underwent successful classic duodenal switch and developed a marked dermatitis with a significant functional limitation. This is an unusual complication, despite the relatively moderate prevalence of nutrient deficiency after this type of bariatric surgery. We discuss possible pathogenic mechanisms and emphasize the importance of an appropriate nutritional management.

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Key words: *Bariatric surgery. Dermatitis. Zinc deficiency. Nutrient deficiency. Duodenal switch. Optic neuropathy.*

DERMOPATÍA Y NEUROPATÍA ÓPTICA POR DÉFICIT DE ZINC TRAS CIRUGÍA BARIÁTRICA MALABSORTIVA

Resumen

Se presenta el caso de una mujer sometida a cirugía bariátrica malabsortiva que desarrolló una llamativa dermopatía y neuropatía secundarias al déficit de zinc. Se trata de una complicación muy poco frecuente, a pesar de la prevalencia de déficit de micronutrientes que se ha observado tras este tipo de intervención. Se discute la patogenia y se recalca el papel del correcto manejo nutricional como tratamiento eficaz.

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Palabras clave: *Cirugía bariátrica. Dermopatía. Dermatitis. Déficit de zinc. Déficit de nutrientes. Cruce duodenal. Neuropatía óptica.*

Abreviaturas

BMI: Body mass index.
%WL: Percentage weight loss.
MRI: Magnetic resonance imaging.

Introduction

Nutritional disturbances after bariatric surgery may occur due to excessive malabsorption and weight loss[1]. Micronutrient deficiencies such as iron, folic acid, calcium, vitamin D, vitamin B12 and thiamine, may result in anemia, neurological alterations and osteopenia. However, even though insufficient levels of zinc and other micronutrients have also been described, its clinical relevance is unusual[2]. We describe a patient who developed important clinical manifestations due to zinc deficiency early after bariatric surgery.

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Case report

A 32-year old woman, with a long-term history of obesity and no associated comorbidities, except for irregular menses, underwent classic duodenal switch with a body weight of 143 kg and body mass index (BMI) of 50.7 kg/m^2, with no immediate postoperative complications. After surgery, routine vitamin and mineral oral supplements were introduced. She adequately progressed during the follow-up period and reached correct tolerance to normal solid diet by 5 months. At this moment, she weighed 106 kg (BMI 37.6 kg/m^2; % weight loss (%WL) 25.9%). Occasional vomiting and increase in bowel habit did not limit her activities of daily living.

At 10 months' follow-up, she had achieved a body weight of 85 kg (BMI 30.1 kg/m^2; %WL 40.5%) and increased bowel habit and vomiting persisted. She complained because of gradual appearance of cuta-

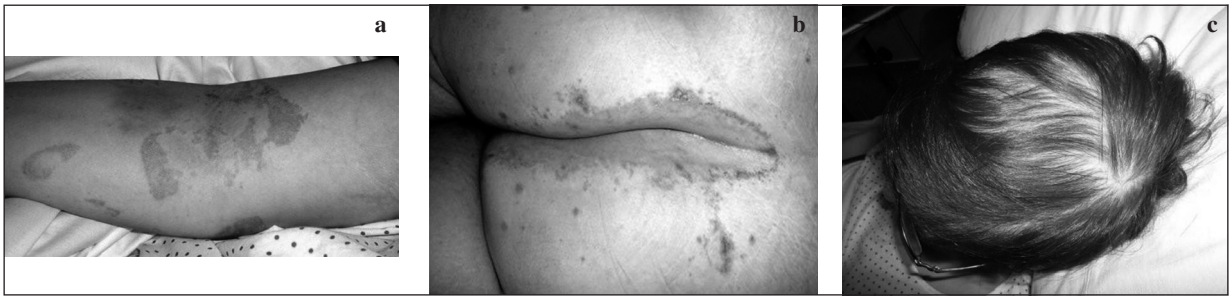

Fig. 1.—Erythematous desquamative cutaneous lesions and eczematous plaques over upper limbs and cutaneous folds (a) and perianal areas (b), with excoriations due to scratching; depigmentation of hair and non-androgenic alopecia (c).

neous lesions distributed over legs, perineal and perianal areas, and she referred increased asthenia, loss of appetite, hair loss, brittle nails, depigmentation, and slight decrease in visual acuity. Physical examination revealed skin pallor, depigmentation of hair, eyelashes and eyebrows, non-androgenic alopecia, subtle and non-painful smooth hepatomegaly and bilateral and symmetric edema in lower limbs. Additionally, erythematous desquamative patterns and eczematous plaques were remarked over abdomen, upper and lower limbs, perineal area, cutaneous folds, and feet soles, and several excoriations due to scratching were evident (fig. 1).

Laboratory data at this time were: erythrocites 3,590,000/mm^3, hemoglobin 11.9 g/dL, hematocrit 34.7%, mean corpuscular volume 96.5 fL, leukocytes 6,100/mm^3, lymphocytes 2,200/mm^3, prothrombin time (PT) 79%, activated partial thromboplastin time (aPTT) 32.2 s, international normalized ratio (INR) 1.1, iron 78 ug/dL (40-145), ferritin 177 ng/mL (10-180), folic acid 3.97 ng/mL (2.3-20), vitamin B_{12} 1402 pg/mL (180-914), serum total proteins 5 g/dL, serum albumin 2.2 g/dL, prealbumin 7 mg/dL, alanine aminotransferase (ALT) 54 U/L, aspartate aminotransferase (AST) 52 U/L, gamma glutamyltransferase (GGT) 163 U/L, alkaline phosphatase (FA) 117 U/L, total bilirrubin 2.7 mg/dL, total cholesterol 177 mg/dL, triglycerides 361 mg/dL. Micronutrient deficiency was observed: copper 51 ug/dL (80-155), zinc 41 ug/dL (60-150), selenium 50 ug/L (60-120) and ceruloplasmin 19.7 mg/dL (22-60). Routine serological tests, including hepatitis, human immunodeficiency virus and toxoplasma, were negative. An esophago-gastroduodenal transit showed no postoperative complications and abdominal ultrasound evidenced a mild hepatic steatosis and gallstones.

Dermatologic specific evaluation concluded that cutaneous lesions were due to nutrient deficiency. Neurologic examination did not observe loss of sensitivity, motor or reflex functions of lower limbs, but revealed bilateral optic disc pallor. A cerebral magnetic resonance imaging (MRI) was performed with unremarkable findings. Evoked visual potentials showed a decrease in conduction velocity in both optic nerves. The diagnosis of deficiency optic neuropathy was established.

Total parenteral nutrition, including vitamins and micronutrients, was started, and topic glucocorticoids were prescribed. Two weeks later, cutaneous lesions had disappeared, the patient's unspecific symptoms were improved, and laboratory measures returned to normality: serum proteins 5.8 g/dL, serum albumin 3.5 g/dL, prealbumin 22.3 mg/dL, copper 83 ug/dL (80-155), zinc 71 ug/dL (60-150), selenium 68 ug/L (60-120), ceruloplasmin 27.8 mg/dL (22-60). The patient was discharged with daily supplements of protein, multivitamins and zinc sulfate (60 mg/day), without reappearance of lesions nor visual deficits, and remaining clinically asymptomatic.

Discussion

Bariatric surgery is an effective treatment for morbidly obese patients, which allows significant and durable weight loss, as well as resolution of several comorbidities.[3] However, it may potentially develop a variety of nutritional and metabolic complications due to anatomical changes of the gastrointestinal tract.[1] This is especially remarkable in malabsortive procedures such as Roux-en-Y gastric bypass (RYGB) and biliopancreatic diversions, in comparison to the merely restrictive ones.[4]

The most common micronutrient deficiencies are iron, vitamin B12, calcium and vitamin D,[5] which can result in anemia, neurologic affections and osteopenia. In general, adequate oral multiple supplements may prevent them, but there are no controlled trials that establish the best type and dosage required for patients after bariatric surgery.[6] Less frequent deficiencies include vitamins A and K, zinc and copper.[2]

Zinc deficiency has been observed in obese patients prior to bariatric surgery, and is worsened afterwards.[7,8] Levels are not always monitored, and, thus, prevalence of its postoperative deficiency is difficult to infer[2], but several reports have observed rates ranging from 10-74%, and differences have been observed between Roux-en Y gastric bypass and biliopancreatic diversion techniques.[7] Nevertheless, most deficiencies are marginal and clinically asymptomatic, and there are only few reports[9] that address its impact illustrating clinical manifestations such as the patient here

presented. Moreover, these subtle subnormal micronutrient levels are rarely specifically targeted, since they are usually associated to low intake of red meat and other proteins, and, they are, therefore, frequently resolved when diet is improved.

Because of its role in numerous biochemical pathways, insufficient zinc levels may affect several organ systems, including integumentary, immune and central nervous system, leading to mental lethargy, diarrhea, poor appetite, weight loss, immune dysfunction, alopecia, delayed wound healing and dermatitis.[10] However, clinical manifestations are rarely relevant.

The hypothesized mechanism for the development of these alterations is that cells with a rapid turnover rate are highly sensitive to nutritional deficiencies[9]. Skin signs include those observed in our patient, but histopathological examination is not specific, so biopsy of lesions was not deemed necessary to establish the diagnosis, since they were characteristic and micronutrient levels were found to be decreased; thus, other etiologies for dermatitis were reasonably ruled out. Rapid weight loss and malabsorption inherent to bypassing of duodenum and proximal jejunum (important sites for zinc absorption), would have been the main reasons why nutrient deficiency developed in this patient. Additionally, protein malnutrition and routine iron and calcium supplements may have contributed to insufficient zinc assimilation[11]. Zinc absorption capacity is jeopardized even despite doubling of oral supplements, so current and empirical reference intakes that regular multivitamin tablets provide (10-15 mg/day) deem insufficient for recovery of body zinc stores, and higher intakes are required (40-60 mg/day),[12] as it occurred in this patient.

Regarding neurological symptoms, optic neuropathies have been associated to vitamin B12 deficiency. However, except for low vitamin B12 and copper in patients with posterolateral myelopathy, and low thiamin in those with acute encephalopathy and polyradiculoneuropathy, a correlation of a specific nutritional deficiency and a particular neurologic syndrome cannot always be assured.[13] Insufficient zinc levels have also been observed in this context and, therefore, in our patient, a plausible explanation for development of optic alterations is its deficiency, although disturbances of other elements such as copper cannot be disregarded. However, the latter are more frequently associated to demyelinating neuropathies and hematological alterations in cases of long-lasting and severe deficits,[2] which were not present in this patient. Bilateral and progressive non-acute affection and the improvement of clinical symptoms after parenteral supplementation support the hypothesis of zinc insufficiency as the main etiology. Furthermore, the normality of serologic tests and MRI findings, and the absence of toxic use or family history, reasonably ruled out other possible etiologies at this age, such as infection, demyelination, toxics or familiar alterations.

The impressive and functionally limiting affection this patient developed due to nutrient deficiency is not frequently observed. Zinc status was evaluated according to plasma levels, which is the most widely used biochemical indicator and the only one for which reference parameters are available.[12] However, plasma zinc represents only 0.1% of total body stores. Zinc is mainly found in skeletal muscle, bone and liver[10] and, in blood, the majority of this element is found in erythrocytes (with plasma levels representing only 10-20%).[11] Protein deficiency in this patient may have determined hypotrophy of both enterocytes and red cells[9], contributing to reduction of intestinal zinc absorption and total pool levels, respectively, even though plasma values would not be as equally affected.

We illustrate and emphasize the need for intensively supplementing zinc after bariatric surgery, especially after malabsortive procedures, as requirements of this element are probably underestimated.

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