Sterile Corneal Perforation Occurring Several Years After Biliopancreatic Diversion

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Abstract

Background To report the first two cases of sterile corneal perforation secondary to vitamin A deficiency after biliopancreatic diversion with duodenal switch (BPD/SW).

Methods Observational case series.

Results Two patients with a history of BPD/SW presented with corneal perforation associated with conjunctival xerosis and keratopathy. In both cases, serum vitamin A level dosage revealed a marked deficit, and the patients admitted poor compliance with vitamin supplementation. Oral therapy with vitamin A was started immediately, and in one case ocular surgery was performed to preserve the integrity of the globe.

Conclusions Ophthalmologists should carefully examine the ocular surface of patients undergone bariatric surgery in order to promptly recognize the signs of vitamin A deficiency and avoid serious sight-threatening complications.

Keywords Biliopancreatic diversion · Vitamin A · Xerophthalmia · Corneal perforation

Introduction

Vitamin A is an essential fat-soluble vitamin that is necessary for ocular surface epithelial integrity as well as for retinal phototransduction. Retinol (preformed vitamin A) is the most active form and is found in animal products, while carotenoids are precursors that are present in vegetables and fruits. In developing countries, vitamin A deficiency represents the leading causes of preventable blindness [1]. Conversely, in developed countries, this deficiency is rare and may occur in the setting of psychiatric eating disorders or malabsorption diseases such as cystic fibrosis, chronic pancreatitis, chronic liver disease, and inflammatory bowel disease [2].

Also patients undergoing bariatric surgery are at risk for vitamin and mineral deficiencies because of decreased intake, change in gastrointestinal anatomy, digestion, rate of transit, and absorption [3]. Vitamin A is mainly absorbed via micelles by the mucosa of the proximal jejunum. Relative bile acid deficiency as well as bile acid deconjugation due to upper intestinal bacterial overgrowth is responsible for vitamin A deficiency after bariatric surgery [4]. Low levels of vitamin A were reported in 61–69% of patients after biliopancreatic diversion with duodenal switch (BPD/SW) and in 8–11% after Roux-en-Y gastric bypass (RYGB) [4]. Clinical manifestations of vitamin A deficiency include nyctalopia (night blindness), xerophthalmia, dry skin and hair, and reduced resistance to infections.

We report herein the first two cases of sterile corneal perforation occurring several years after bariatric surgery due to severe vitamin A deficiency.

Report of Cases

Patient 1 A 68-year-old woman was referred to our center for a chronic corneal ulcer in her right eye (RE). Patient's past medical history was significant for obesity that was treated in 2005 with BPD/SW. The postoperative course of the following years was complicated by iron-deficiency anemia that required iron supplementation and periodic blood transfusions.

Upon examination, the patient presented a large central corneal perforation with iris prolapse in RE (Fig. 1a and b),

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while conjunctival xerosis with superficial punctate keratitis (SPK) was detected in left eye (LE) (Fig. 1c). On questioning, the patient admitted noncompliance with vitamins' supplementation prescribed after the bariatric surgery. Serum vitamin A level dosage revealed a marked deficit (0.04 mg/L vs normal range of 0.3–0.7 mg/L). Gundersen conjunctival flap was used to restore the integrity of the compromised corneal surface (Fig. 1d). Postoperatively, the patient was treated with vitamin A ointment applied topically every 2 h, and 100,000 IU of vitamin A was administered intramuscularly for 3 days followed by 50,000 IU for 2 weeks, as recommended by the American Society for Metabolic and Bariatric Surgery [5]. Despite the poor/null vision in RE (light perception), the ocular surface health improved in the following weeks in both eyes.

Patient 2 A 46-year-old woman was referred to our center for severe pain and light sensitivity in LE. She reported being treated without success with topical antibiotics and tear substitutes over the last weeks for a recalcitrant corneal ulcer.

Upon examination, the patient presented a corneal perforation with shallow anterior chamber and anterior synechiae in LE (Fig. 2a and b), while conjunctival xerosis, Bitot's spots, and SPK were detected in RE (Fig. 2c and d, Parts C-D). On further questioning, the patient admitted her history of BPD/SW performed in 2006 that was initially not disclosed. She reported to have interrupted vitamin supplementation on her own initiative due to limited efficacy and high cost. Serum vitamin A dosage confirmed a severe deficiency (0.06 mg/L). Botulinum toxin was injected in the levator superioris palpebrae to induce temporary ptosis to protect the eye globe. The patient was treated with vitamin A ointment applied topically every 2 h, and 100,000 IU of vitamin A was administered intramuscularly for 3 days followed by 50,000 IU for 2 weeks. Although in the following weeks corneal perforation healed and ocular surface status gradually improved in both eyes, vision is still poor in LE (20/400), and surgery will be required for rehabilitation.

Discussion

The incidence of vitamin A deficiency is predicted to rise over the next decades because of the increasing prevalence of obesity and the popularity of bariatric surgery [6]. Nyctalopia represents the earliest and most common symptom of vitamin A deficiency and was previously reported in patients who have undergone both RYGB and BPD [7, 8]. Usually, the night vision rapidly recovers with the initiation of vitamin supplementation without sequelae [7]. Conversely, in case of long-term and severe deficiency, the ocular surface may become involved, with the development of conjunctival xerosis, superficial punctate keratitis, and corneal ulceration that may impair vision at a various extent [8].

Although vitamin A deficiency following bariatric surgery is common, to the best of our knowledge, only one case of corneal ulceration has been reported in this setting [9]. In that case, the patient underwent RYGB and subsequently laser in

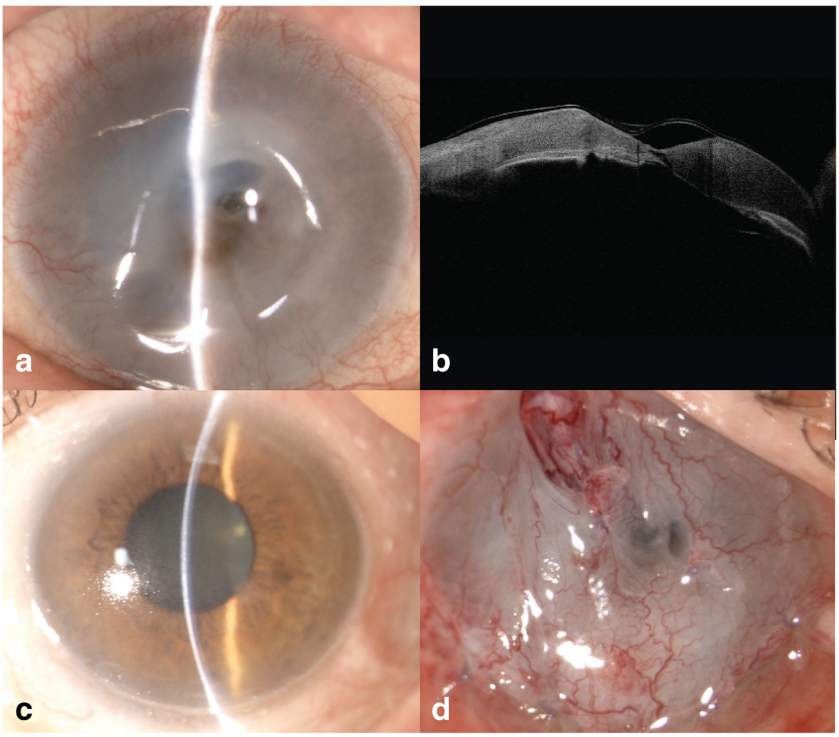

Fig. 1 Patient 1. **a** Slit-lamp photograph of right eye (RE) showing a large central corneal perforation with iris prolapse. A soft contact lens was put to cover the corneal surface. **b** Anterior segment optical coherence tomography of RE showing the contact lens covering the corneal perforation and the complete adhesion between iris and cornea. **c** Slit-lamp photograph of the left eye showing conjunctival xerosis and corneal epitheliopathy. **d** Slit-lamp photograph of RE 1 week after Gundersen conjunctival flap

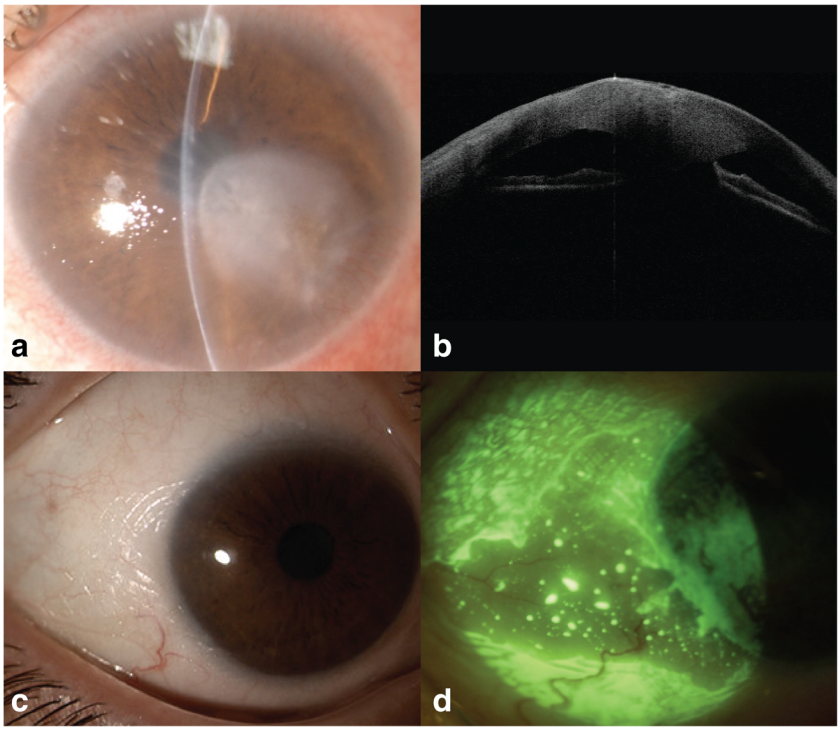

Fig. 2 Patient 2. **a** Slit-lamp photograph of left eye (LE) showing infero-temporal corneal perforation with surrounding corneal edema. **b** Anterior segment optical coherence tomography of LE showing corneal edema in the contest of previous perforation and anterior synechiae. **c** Slit-lamp photograph of right eye (RE) showing the typical appearance of conjunctival xerosis. **d** Slit-lamp photograph of RE after fluorescein staining showing Bitot's spots located in the temporal conjunctiva

situ keratomileusis surgery for the correction of myopia. It is reasonable to hypothesize that the corneal surgery may have worsened a pre-existing dry eye, thus determining the onset of the corneal ulceration.

We described herein the first case series of sterile corneal perforation secondary to vitamin A deficiency occurring in two patients several years after bariatric surgery. Unlike ulceration, corneal perforations represent a surgical emergency with potentially devastating consequences, including not only blindness but also anatomical loss of the eyeball [10]. The first goal is to re-establish the structural integrity of the globe, avoiding infection. Then, it is crucial to restore vitamin A levels and ocular surface health, necessary for maximizing outcomes of subsequent ocular surgery, when visual rehabilitation is needed.

Lifelong multivitamin supplementation is required after bariatric surgery; in addition, regular metabolic and nutritional monitoring is highly recommended. Both our patients reported non-adherence to vitamin supplementation. Overall, poor adherence to post-bariatric surgery nutritional recommendation is common and is associated with higher rates of micronutrient deficiency. In a recent survey, more than half of bariatric surgery patients reported having trouble taking all their supplements. Common reasons for poor adherence were difficulty in remembering, high number of tablets, side effects, and cost [10].

On one hand, all patients undergoing bariatric surgery should be educated regarding the possibility of irreversible eye damage from vitamin A deficiency. The adherence with proper vitamin supplementation should be stressed to prevent potentially vision-threatening complications. On the other hand, ophthalmologists should promptly recognize the ocular hallmarks of vitamin A deficiency and questioning patients about their past medical history. In fact, a careful examination of the ocular surface may allow early diagnosis of milder cases that can be managed with success, avoiding devastating complications typical of severe cases.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval For this type of study, formal consent is not required.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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