

Case Report of Recurrent Xerophthalmia Associated with Vitamin A Deficiency Secondary to Lipid Malabsorption Following Extensive Colon Resection

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Abstract

Purpose: To describe the successful utilization of intensive ocular surface lubrication and anti-inflammatory therapy following the United States Institute of Surgical Research (ISR) ophthalmic burn protocol in the treatment of a case of recurrent xerophthalmia associated with Vitamin A deficiency secondary to lipid malabsorption.

Methods: The patient is a 55-year-old female with a history of Vitamin A deficiency secondary to lipid malabsorption following extensive bowel resection presenting with blurred vision, pain, and nyctalopia in both eyes. On exam, her best corrected visual acuity was 20/70 in both eyes. She was noted to have Bitot spots, diffuse epithelial erosions, and microcystic edema in both eyes. The United States Institute of Surgical Research ophthalmic burn protocol was initiated, which includes ocular surface lubricants, topical moxifloxacin, oral doxycycline, topical prednisolone, topical acetylcysteine, oral vitamin C, PROKERA®, and temporary tarsorrhaphy.

Results: The patient's ocular surface was successfully maintained utilizing the ISR Burn protocol to protect the ocular surface affected by xerophthalmia while awaiting Vitamin A supplementation. More invasive surgical procedures were not required. By day 24 following Vitamin A supplementation, the patient's best corrected visual acuity was 20/20 in both eyes with a benign ocular surface on exam.

Conclusion: The visual morbidity associated with xerophthalmia secondary to Vitamin A deficiency can be devastating. The authors demonstrate the success of aggressive therapy to protect the ocular surface while awaiting supplemental Vitamin A in a patient with recurrent xerophthalmia secondary to lipid malabsorption. Additionally, the importance of continued follow-up, coordination of care, and maintenance of Vitamin A supplementation in patients with lipid malabsorption are demonstrated.

Keywords: Vitamin A; Xerosis; Keratomalacia; Xerophthalmia; Nyctalopia

Introduction

A common cause of blindness in developing countries, Vitamin A deficiency is estimated to affect more than 250 million children worldwide, with nearly 3 million suffering xerophthalmia [1]. A fat-soluble vitamin, 90% of Vitamin A is stored in the liver [2]. Its prevalence in the developed world is expected to continue to increase as a result of the rising frequency of bariatric surgery, cirrhosis, and extensive bowel resections [3,4]. The expected increase has significant implications in terms of the ocular health amongst the aforementioned patient populations.

Vitamin A is critical to the normal function of the retina, as it is required for the synthesis of visual pigment within the rods and cones and is key to photo signal transduction. Additionally, vitamin A plays a major role in the maintenance of the corneal and conjunctival epithelium [1,2]. Vitamin A deficiency results in a loss of mucus production by goblet cells, which leads to decreased wetting and a compromised ocular surface. Damaged epithelium is replaced by keratinized epithelium with atypical organization, which leads to the

development of superficial punctate keratopathy with conjunctival/corneal xerosis, and ultimately keratomalacia resulting from liquefactive necrosis [4].

The authors present a case of recurrent xerophthalmia associated with Vitamin A deficiency due to lipid malabsorption in a patient following significant colon resection. The authors demonstrate the successful utilization of the United States Institute of Surgical Research Ophthalmic Burn protocol to protect the ocular surface while awaiting Vitamin A supplementation.

Case and Methods

The patient is a 55-year-old African American female who presents to the Acute Care Eye Service with three weeks of progressive blurring of vision, pain, and nyctalopia bilaterally. She has an ocular history of Vitamin A deficiency World Health Organization (WHO) Grade X3 (Figure 1) secondary to lipid malabsorption. Her medical history also includes colon cancer status-post a right hemicolectomy in 2008, and a pancreatic mucinous neoplasm status-post Whipple procedure in 2010. The patient had been followed by the Surgical Oncology service and Ophthalmology service, but was lost to follow-up approximately one year prior to presentation. She was also followed by the Nutritional

medicine service and was receiving Vitamin B12 injections, but denied any Vitamin A supplementation.

Amniotic membrane graft	Promotes epithelialization
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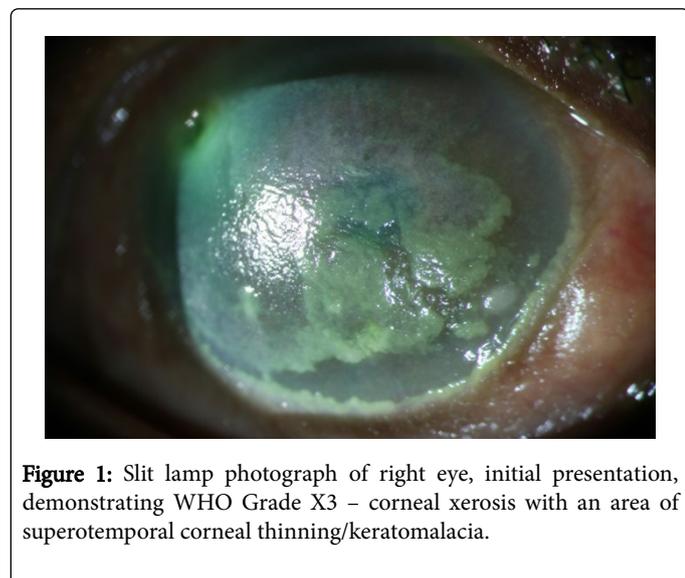


Figure 1: Slit lamp photograph of right eye, initial presentation, demonstrating WHO Grade X3 – corneal xerosis with an area of superotemporal corneal thinning/keratomalacia.

On exam, the patient’s best corrected visual acuity (BCVA) was 20/70 in both eyes. She had no appreciable tear lake in either eye, and the tear break-up time was approximately two seconds. On slit lamp exam she was noted to have perilimbal chemosis with chalasis and Bitot spots in both eyes (OU). A superotemporal scar was present in the right eye with approximately 60% thinning. Additionally, diffuse punctate epithelial erosions, microcystic edema, and Descemet folds were present in both eyes, but no corneal melt observed in either eye. The patient was diagnosed with recurrent Vitamin A deficiency, WHO Grade X2.

At this time, given the extent of her ocular surface disease, the United States Institute of Surgical Research (USISR) ophthalmic burn protocol (Table 1) was initiated. Initial management included a laboratory evaluation of serum Vitamin A level, in addition to preservative free tears every hour, one drop ophthalmic moxifloxacin 0.5% solution four times per day, one drop ophthalmic prednisolone 1% suspension eight times daily, ophthalmic 10% acetylcysteine solution drops twice daily, and Refresh Lacri-lube® ointment nightly in both eyes.

Treatment	Mechanism
Artificial tears and lubricants	Increases wetting of the ocular surface
Moxifloxacin topical	Prevents secondary bacterial infection of the exposed surface
Doxycycline, oral	Decreases MMPs resulting in decreased inflammation
Prednisolone, topical	Anti-inflammatory
Acetylcysteine, topical	Collagenase inhibitor
Vitamin C, oral	Antioxidant and cofactor in rate-limiting-step of collagen synthesis
Temporary tarsorrhaphy	Limits exposure keratopathy

Table 1: Institute of Surgical Research (ISR) Ophthalmic Burn protocol components.

Additionally, oral doxycycline 100 mg twice daily and Vitamin C was initiated. Furthermore, a PROKERA® was placed in the both eyes. The Internal Medicine service was consulted and an urgent referral placed to schedule Vitamin A intramuscular injections for the patient as quickly as possible.

The following day, the patient was noted to have a stable exam with bilateral PROKERA® intact, but persistent diffuse corneal haze. Celluvisc® four times daily was added to the treatment regimen in both eyes. On day four, the stromal edema had improved. The PROKERA® was noted to be absent in both eyes. The PROKERA® rings were removed, and a temporary temporal tarsorrhaphy was performed in both eyes. Celluvisc® ophthalmic 0.05% solution every 30 minutes in addition to cyclosporine 0.05% ophthalmic emulsion (Restasis®) three times daily and over the counter Vitamin A ophthalmic ointment were added to the treatment regimen. Despite attempts to coordinate with the Nutritional medicine and Internal medicine service to ensure urgent Vitamin A supplementation, the patient had not received any supplements due to an apparent lack of communication between services and/or understanding of the patient. The patient was again encouraged to follow-up with the Nutritional medicine service for administration of Vitamin A intramuscularly. The patient was lost to follow-up but did ultimately undergo Vitamin A supplementation following the protocol presented by Rocha Lima and colleagues [5]. On day 10, the Vitamin A level returned at 2 mcg/dL (normal=20-65 mcg/dL). She received 100,000 IU Oleovitamin A intramuscularly for 3 days starting on Day 10 after presentation, followed by 50,000 IU for eleven days. The patient was then transitioned to daily oral doses of 25,000 IU and followed clinically.

She presented again on day 17, and a repeat vitamin A laboratory evaluation was performed, demonstrating a level of 67 mcg/dL. The temporary tarsorrhaphy remained in place in both eyes. The patient’s BCVA had improved to 20/40 in both eyes. There were persistent diffuse punctate epithelial erosions, but the corneal edema had significantly improved. A prednisolone taper was initiated at that time. By day 24 following Vitamin A supplementation, the patient’s BCVA was 20/20 bilaterally with a benign ocular surface on exam as demonstrated in Figure 2.

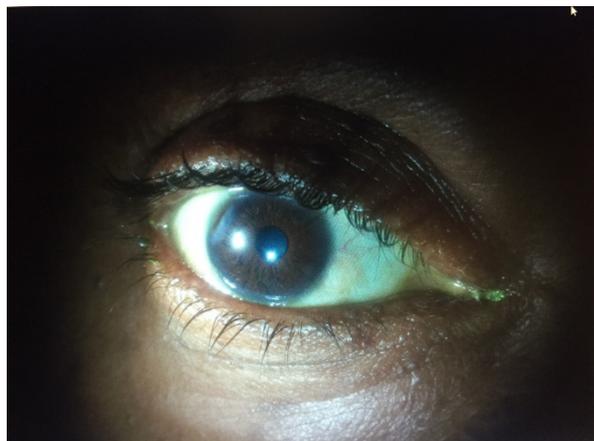


Figure 2: Slit lamp photograph of right eye with a superotemporal corneal scar but overall benign ocular surface, second presentation after ocular surface treatment and Vitamin A supplementation.

The temporary tarsorrhaphy in both eyes was discontinued. The next week, the patient's BCVA remained 20/20 in both eyes, with significantly improved PEE and edema. Her treatment regimen was switched to artificial tears every 3-4 hours as needed, Refresh Lacri-lube® ointment four times daily, and Restasis® twice daily. At follow-up day 45, the patient's BCVA was stable at 20/20 in both eyes, with a benign ocular surface on exam. A timeline of therapeutic interventions following diagnosis is presented in Table 2.

Time from presentation (days)	Intervention
1	USISR burn protocol initiated
2	Celluvisc added
4	Temporary temporal tarsorrhaphy placed
10	Vitamin A supplementation initiated
24	Temporary tarsorrhaphy discontinued
31	Ocular surface drop regimen tapered

Table 2: Timeline of therapeutic interventions following diagnosis.

Discussion

Visual outcomes of unrecognized and untreated xerophthalmia secondary to vitamin A deficiency can be devastating. With early recognition, aggressive ocular surface therapy, and Vitamin A supplementation, excellent visual acuity can be maintained. Clinical suspicion of Vitamin A deficiency in certain patient populations such as those having undergone bariatric surgery or significant colon resection is warranted. Additionally, recent case reports of psychogenic Vitamin A deficiency and self-induced Vitamin A deficiency related to fad dieting practices should alert providers to the possibility of xerophthalmia with ocular complaints in such patients [6,7].

Given the inability of adequate tear production secondary to the lack of available Vitamin A, the utilization of an aggressive ocular surface lubrication and anti-inflammatory regimen for patients with

difficulty maintaining an adequate ocular surface is intuitive. At present, no specific protocol for ocular surface lubrication and preservation in xerophthalmia secondary to Vitamin A deficiency has been proposed. Extensive research in the field of ocular burns has demonstrated the importance of the elements listed within the above protocol in terms of surface wound healing for patients with thermal and chemical burns, limbal stem cell deficiency, and Stevens-Johnson syndrome [8,9].

Conclusion

The case presented demonstrates the successful utilization of an ocular surface lubrication and anti-inflammatory regimen following the ISR Ophthalmic Burn protocol to protect the ocular surface prior to Vitamin A supplementation in a patient with recurrent xerophthalmia due to Vitamin A deficiency. This case also highlights the importance of effective communication across specialty services in coordinating the care of patients with nutrient deficiencies and ocular disease. The patient's final best-corrected visual acuity following eventual supplementation of Vitamin A remained at baseline, being Snellen 20/20 in both eyes at the conclusion of treatment. The importance of continued follow-up and maintenance of Vitamin A supplementation in patients with lipid malabsorption are also demonstrated. Successful coordination of care between ophthalmic providers, primary care physicians, and nutritionists is crucial to the overall management and preservation of vision in patients with xerophthalmia secondary to Vitamin A deficiency.

Disclaimer

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