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

Reed D* Brundridge W, Gallagher C, Santamaria J and Legault G
San Antonio Military Medical Center, Texas, USA

*Corresponding author: Reed D, San Antonio Military Medical Center, 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234, USA, Tel: 6159433222; E-mail: donovan.s.reed@gmail.com

Received date: July 08, 2018; Accepted date: July 31, 2018; Published date: August 08, 2018

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.

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.

On 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.
difficulties maintaining 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

The views expressed herein are those of the authors and do not reflect the official policy or position of Brooke Army Medical Center, the U.S. Army Medical Department, the U.S. Army Office of the Surgeon General, the Department of the Air Force, the Department of the Army, Department of Defense, the Uniformed Services University of the Health Sciences or any other agency of the U.S. Government.

References