# BilateralCorneal UlcerationDueTo Pancreatitis

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**Abstract:** Purpose: Vitamin A deficiency can lead to a variety of ocular changes from xerosis and xerophthalmia to corneal ulcer and perforation. The treatment of this devastating disease is simple and inexpensive. It is therefore important to recognize and treat accordingly, especially in the event of ulcers unresponsive totreatment or in the presence of severe malnutrition/malabsorption syndromes. Hereby we report a case of bilateral corneal ulceration who is a known alcoholic diagnosed with pancreatitis. Initially ulcers were unresponsive to conventional treatment for immunogenic ulcers, but resolved on vitamin A supplementation. The purpose of this case report is to remind physicians of the potentially devastating effects of vitamin Adeficiency on the eyes and to demonstrate outcomes after vitamin A treatment.

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#### I. Introduction

Vitamin A deficiency is the leading cause of preventable childhood blindness in the developing world<sup>1</sup>. Vitamin A deficiency has been known to occur as a result of poor dietary intake, liver diseases, and gastrointestinal malabsorption<sup>2-5</sup>. Vitamin A is a fat-soluble vitamin ingested in the diet in two forms: as retinol itself from animal sources, such as milk, meat, fish, liver, and eggs, or as the provitamin carotene from plant sources, such as green leafy vegetables, yellow fruits, and red palm oil<sup>6</sup>. On the ocular surface, retinol is necessary for epithelial cell RNA and glycoprotein synthesis. As a result, vitamin A deficiency has a wide range of ocular manifestations including conjunctival and corneal xerosis, keratomalacia, retinopathy, visual loss, and nyctalopia, also called night blindness, which is the earliest and most common symptom<sup>7</sup>. We report a case of bilateralcorneal ulceration in a patient with severe vitamin A deficiency due to pancreatitis.

## II. Case Report

32 year old male presented to our unit with complaints of diminution of vision in both eyessince 1 week associated with pain. Patient was recently diagnosed with pancreatitis for which he is on pancreatic enzymes, insulin and fat free diet. Patient is a known alcoholic.

Acuity without correction was 6/60 OD, improved by pinhole to 6/36, and 6/60 OS not improved by pinhole. The intraocular pressure was 19 mm Hg OD and 21 mm Hg OS by NCT.

Slit-lamp examination showed temporal wrinkling of the conjunctiva in both eyes, withloss of transparency (figure 1). The corneas appeared dull with superficial punctate keratopathy. Epithelial defects were seen in corneas of both eyes overlying a partial-thickness ulcer with approximately 50% thinning and adjacent limbitis (figure 2). In both eyes, the anterior chambers were quiet and lens were clear. No abnormality was detected on funds examination.





Figure 1: conjunctival xerosisFigure 2: corneal ulceration with limbitis

### III. Result

Considering peripheral ulcerative keratitis, patient was started on topical steroids, topical antibiotics and preservative free artificial tears in both eyes. A vasculitic workup (C-reactive protein, erythrocyte sedimentation rate, antinuclear antibody, antineutrophil cytoplasmic antibody, rheumatoid factor, syphilis serology) was unremarkable.

On review after 1 week, condition was worsening with further drop in vision. Suspecting vitamin A deficiency in view of pancreatitis, Serum was sent for analysis of vitamin A and retinol-binding protein (RBP) levels. The results of his serum vitamin A levels revealed that the levels were significantly decreased to 0.5 µmol/l (normal range 0.9–2.5). The RBP level was also reduced to 12 mg/l (normal 30–92). A diagnosis of vitamin A deficiency with subsequent corneal ulceration was established. The patient was treated with IV Multivitamin (containing 10,000 IU vitamin A, C, D, E and B complex) for 5 consecutive days while in hospital, and then with oral vitamin A 200,000 IU once a week for 2 weeks. The corneal ulcersin both eyes were resolved. Similarly, vitamin A and RBP levels normalized after 6 weeks of treatment.

### IV. Discussion

We present a rare case of vitamin A deficiency secondary tomalnutrition caused by pancreatitis leading to bilateral xerosis and sequential corneal ulceration. As our initial treatment with topical steroids, topical antibiotics and tear supplements considering peripheral ulcerative keratitis worsened the condition. The alcoholic status of patient and diagnosis of pancreatitis inclined us to test the serum Vitamin A levels, which were markedly low. Pancreatitis causes indigestion leading to malabsorption and malnutrition. The goal of the therapy is the replenishment of vitamin A stores. The oral dosage regimen is 200,000 IU vitamin A in oil, followed the next day with an additional dose of 200,000 IU<sup>8</sup>. If patients have severe corneal disease or malabsorption, the preferred dose is 100,000 IU watermiscible vitamin A administered intramuscularly<sup>9</sup>. It is important to note that if vitamin A deficiency is associated with a protein-energy malnutrition syndrome, protein levels must be corrected at the same time as vitamin supplementation. If this is not done, symptoms will recur within weeks after therapy.

#### V. Conclusion

Our case serves as a reminder to corneal specialistsof the potentially devastating effects of vitamin A deficiency on the eye. Vitamin A deficiency should be suspected in any practice in the presence of corneal ulceration in combination with malabsorption and malnutrition syndromes.

#### References

- [1]. Thylefors B, Négrel AD, Pararajasegaram R, Dadzie KY: Global data on blindness. Bull World Health Organ 1995;73:115–121.
- [2]. Bishara S, Merin S, Cooper M, Azizi E, Delpre G, Deckelbaum RJ: Combined vitamin A and E therapy prevents retinal electrophysiological deterioration in abetalipoproteinaemia. Br J Ophthalmol 1982;66:767–770.
- [3]. Russell RM, Morrison SA, Smith FR, Oaks EV, Carney EA: Vitamin-A reversal of abnormal dark adaptation in cirrhosis. Study of effects on the plasma retinol transport system. Ann Intern Med 1978;88:622–626.
- [4]. Rogers EL, Douglass W, Russell RM, Bushman L, Hubbard TB, Iber FL: Deficiency of fat soluble vitamins after jejunoileal bypass surgery for morbid obesity. Am J ClinNutr 1980;33:1208–1214.
- [5]. Eckert MJ, Perry JT, Sohn VY, Boden J, Martin MJ, Rush RM, Steele SR: Incidence of low vitamin A levels and ocular symptoms after Roux-en-Y gastric bypass. SurgObesRelat Dis 2010;6:653–657.
- [6]. Combs G: The Vitamins: Fundamental Aspects in Nutrition and Health, ed 2. San Diego, Academic Press, 1998.
- [7]. Smith J, Steinemann TL: Vitamin A deficiency and the eye. IntOphthalmolClin 2000;40:83–91.
- [8]. Rustigi AK MD: Vitamin deficiencies; in Albert DM, Jackobiac FA (eds): Principles and Practice of Ophthalmology. Philadelphia, Saunders, 1996, vol 5, pp 2981–2982.
- [9]. 6 A, Tarwotjo I: Protein deficiency and treatment of xerophthalmia. Arch Ophthalmol 1982;100:785–787.

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