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Case Report: Open Access

# A Rare Case of Corneal Perforation Presenting As Spontaneous Expulsion of Lens in a Neonate: Case Report

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## ABSTRACT

Spontaneous corneal perforation in premature infants not due to birth trauma is a rare event, with only 8 cases reported in the literature. We are reporting a neonate 7days old who has presented to us with spontaneous expulsion of lens due to corneal perforation.

**Keywords:** Perforation, Neonate, Expulsion

## INTRODUCTION

Corneal perforation in the perinatal period is rare. Risk factors are low birth weight, systemic infection, birth trauma and vitamin A deficiency. Keratomalacia [1] is an ocular condition usually affecting both eyes that results from severe deficiency of vitamin A. That deficiency may be dietary or metabolic. Vitamin A is essential for normal vision as well as proper bone growth, healthy skin and protection of the mucous membranes of the digestive, respiratory and urinary tracts against infection.

Early symptoms may include night blindness and xerophthalmia followed by keratomalacia. Without adequate treatment [2] increasing softening of the corneas may lead to corneal infection, perforation and degenerative tissue changes resulting in blindness.

## CASE REPORT

We report a case of neonate 7 days old presenting as auto extraction of lens. According to the mother she was opening her eyes for the first three days of life, then it developed a yellowish color discharge from both the eyes and since then she was unable to open her right eyes only. The patient had not taken any treatment. Thereafter on 7$^{th}$ day pediatrician found an oval yellowish glistening structure lying on the right side of the eye on the bed on routine morning checkup. Then they called ophthalmologist. They diagnosed a case of keratomalacia presenting as auto extraction of lens. There is no history of watering and pain and redness. One of the significant findings in the family history was that the mother had history of night blindness and was also having night blindness at the time of admission in the ward. Serum retinol concentration was 14 µg/dl.

The weight of the neonate is 2500 g born at 34 weeks prematurely. APGAR score at 0, 1, 5 min was 8, 9, 9. Cause of premature delivery was uncontrolled hypertension.

On ocular examination left eye eyelids are sticky. Conjunctiva is keratinized. Cornea has full thickness perforation limbus to limbus. Margins of perforation are sharp, edematous and yellowish in color. Cornea is melted. Fundal glow is absent. Right eye has conjunctival keratinazion. Cornea is clear. Anterior segment is within normal limit. Fundus is within normal limit. As per WHO classification R/E comes under XN and L/E under X3b category. Patient was given antibiotic drop and patched the left eye. Right eye was given lubricant ointment. As a treatment the patient was given 50,000 I.U. of vitamin A I.M. daily for two days. Simultaneously the mother was also treated for night blindness with parental doses of vitamin A. Antibiotic ointment and lubricant gel was prescribed three times a day. Then patient was sent to higher centre for corneal repair but patient was expired on next day due to septicemia, so further management could not be done **(Figures 1-3)**.

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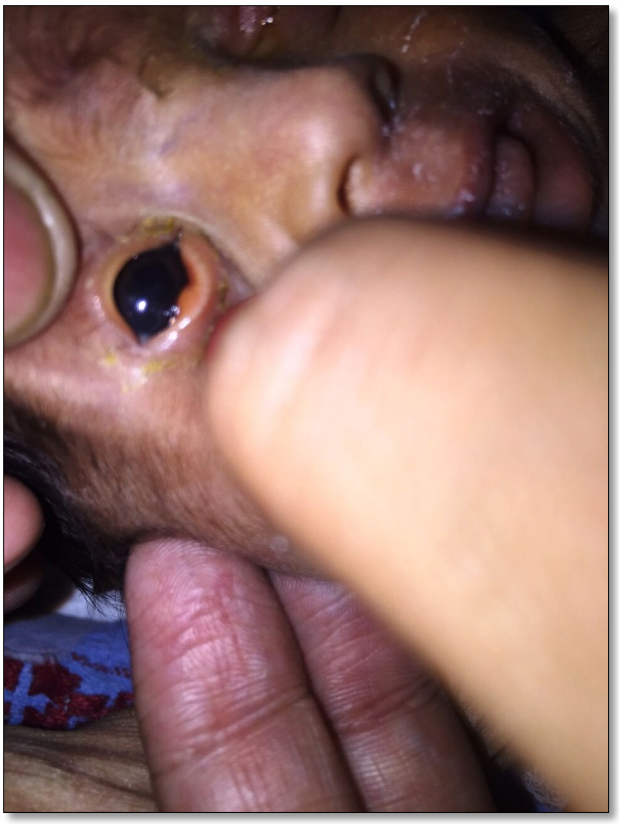

**Figure 1.** Right eye showing keratomalacia.

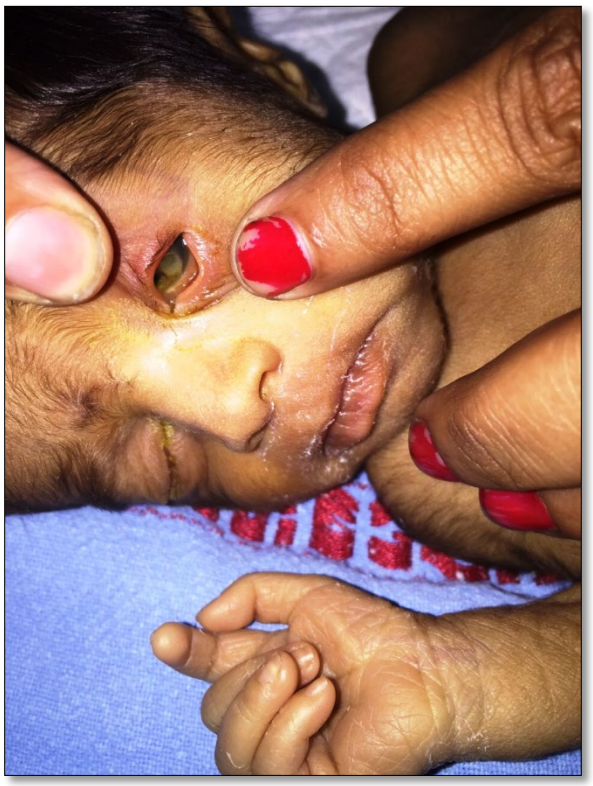

**Figure 2.** Image showing corneal melting.

**Figure 3.** Yellowish color discharge extracted from the eyes.

## DISCUSSION

Vitamin A deficiency is the leading cause of childhood blindness in the developing world. Xerophthalmia [3] is a term used to describe the spectrum of ocular disease that can arise from vitamin A deficiency. These changes include xerosis, corneal ulceration and melting, night blindness and retinopathy. Vitamin A is also essential for immune function and affected children are more susceptible to severe infections, such as measles.

Vitamin A [4] is ingested in the form of retinaldehyde from milk, meat, fish, liver and eggs. It is also ingested as carotene from green leafy vegetables, yellow fruits and red palm oil. These compounds are stored in the liver in the form of retinyl pamitate. The aldehyde form of vitamin A, retinal or retinaldehyde, combines with the protein opsin in the rods to create rhodopsin, which is a photosensitive pigment. A similar process takes place in the cones. During photo transduction, some retinal is lost so a constant supply of vitamin A is needed. Vitamin A deficiency can therefore lead to night blindness with associated visual field changes and a depressed ERG. Vitamin A [5] is also necessary for the maintenance of specialized epithelial surfaces. In the conjunctiva, loss of goblet cells and squamous cell metaplasia leads to dryness or xerosis. Bitot's spots are perilimbal gray plaque. A full-thickness liquefactive necrosis of the cornea that is keratomalacia can also occur. Finally, retinopathy in the form of yellow or white punctuate dots can be seen in the retinal periphery.

In developing countries [6] vitamin A deficiency is associated with general malnutrition and protein deficiency with high mortality and morbidity rates. In one study [7] subclinical vitamin A deficiency is a problem during the pregnancy. Serum concentration of retinol <20 µg/dl appears to indicate a deficient status and is associated with an increased risk of preterm delivery and increased susceptibility of infection. It is hypothesized that maternal malnutrition and defective corneal morphogenesis may have contributed in present case.

## CONCLUSION

Use of prophylactic anti glaucoma medication in premature neonates is proposed and needs analysis. Awareness in pediatricians of this potential complication is vital to prevent blindness.

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