

HYPOPYON KERATITIS AS A COMPLICATION OF PRIMARY VARICELLA IN A 7-MONTH-OLD CHILD

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Abstract. Ocular complications associated with primary varicella zoster virus infection may involve any part of the eye. Unlike ophthalmic zoster in which the virus reactivates, ocular complications from primary varicella are rare. The aim is to present an interesting clinical case of a primary varicella keratitis overlapped by a bacterial infection. A seven-month-old boy was presented to an outpatient clinic 10 days (about 1 and a half weeks) after the beginning of a chickenpox rash with symptoms of bacterial keratitis of his right eye. He was admitted to a specialized clinic and successfully treated with a combination of topical quinolone treatment, washing of the conjunctival sac with iod-povidone 5% and two subconjunctival applications of Cefotaxime 5% and Atropine/Adrenalin 1% under general anesthesia. Twenty days after the initial complaints there were no signs of inflammation and only two small macules could still be observed. The child would be followed up for three months in case of recurrence. Primary varicella, even though considered benign in childhood, should not be underestimated. Some serious ocular complications can develop which if left untreated could lead to devastating visual results.

Key words: bacterial keratitis, chickenpox, ocular complications

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INTRODUCTION

Primary varicella infection, also called chickenpox, is a common benign illness caused by varicella-zoster virus (VZV), typically associated with fever and characteristic exanthematous vesicular skin rash that heals with scab formation. The varicella-zoster virus is a member of the Herpesviri-

dae family, and it occurs mostly in children younger than 10 years of age [1]. Unlike ophthalmic zoster in which a reactivation of latent VZV can cause ocular complications in up to 50% of the cases, severe ocular involvement is rare in varicella [2]. However, ocular complications associated with primary VZV infection can occur and may involve any part of the eye from the conjunctiva to the optic nerve. Ocular

disease may develop a few days to several weeks after the onset of a skin rash [3].

Ocular manifestations in varicella are diverse including lid lesions, conjunctivitis, conjunctival pock, keratitis, anterior uveitis, acute retinal necrosis [4], retinal vasculitis, preseptal cellulitis, chorioretinitis, optic neuritis, neuroretinitis [5], scleritis, and internal or external ophthalmoplegia [6]. Lid lesions and conjunctiva involvement have been described as the most common complications [5]. The conjunctival involvement may manifest as follicular conjunctivitis or commonly as a small erythematous pock-like lesion near the limbus. Ophthalmological involvement may be unilateral or bilateral.

Ophthalmic involvement may result from a direct viral replication or from an immune reaction to the systemic viral infection [3]. Ocular manifestations described during the acute phase include a palpebral cutaneous rash, conjunctivitis, superficial keratitis, sclerokeratitis, episcleritis, and uveitis. Superficial keratitis during the acute phase can have different manifestations. Some authors reported epithelial ulcerative keratitis [4], other bilateral recurrent geographic epithelial ulceration, mild punctate staining of the cornea, dendriform keratitis, and necrotizing keratitis with ulceration [7]. Stromal keratitis can occur after the acute phase, resulting from an immune response against viral antigens in the cornea. It may have a chronic course. The stromal keratitis can be nummular with multiple superficial stromal round infiltrates and a deeper and more diffuse stromal keratitis with infiltration and haze [8].

CASE PRESENTATION

A seven-month-old boy was presented to an outpatient clinic with blepharospasm, edema and hyperemia of the eyelids of the right eye. According to the parents the complaints started the previous day. They also noticed discharge from the eye. The patient suffered from chickenpox which started 10 days (about 1 and a half weeks) before the presentation of the ocular complaints with two days fever and a moderate rash. The child got infected from his older sister. The varicella infection had no other general complications up to this point. The child was born on term after an uncomplicated pregnancy and had normal intellectual and physical development. He did not suffer from any other accompanying diseases. At the time of the first ocular exam the skin rash had already begun to heal with crusts. When examining the conjunctiva, it was severely hyperemic with slight discharge. Due to the patient's young age, he was fussy and uncooperative, so the exam could not be thoroughly performed.

Topical quinolones and nonsteroidal anti-inflammatory drugs (NSAID) drops were prescribed. The same evening the parents noticed a white semicircle at the base of the cornea. The patient was referred immediately to a specialized Ophthalmic clinic.

The child was admitted at the clinic two days later due to logistic problems. A consultation with an infectious diseases specialist was done before admission for exclusion of an epidemiological risk and other general complications from the chickenpox. The infectious diseases specialist confirmed that the child did not pose a threat to other patients and was in a good general state except for slight hyperemia of the pharynx. A blood sample was taken on the day of admission and the results showed Hgb – 116 g/l, RBC – $5.03 \times 10^{12}/L$, WBC – $9.84 \times 10^9/L$, PLT – $681 \times 10^9/L$. A consultation with a pediatrician was conducted and oral Azithromycin was prescribed.

The child was examined under general anesthesia using an eyelid speculum (Fig. 1). Since topical antibiotic drops had already been administered, the team decided that taking a corneal scrape would be pointless and risky for the patient. There was a severe ciliary injection and a limbal pock which was inflamed at one o'clock. A paracentral ulcer was observed with a severe hypopyon (3 mm). The pupil was constricted but synechiae were not observed. The posterior eye segment was impossible to assess at that time due to corneal and anterior chamber changes. The most probable hypothesis of the team for these complications was a primary replication of the virus through the limbal pock into the cornea which caused epithelial changes. An epithelial lesion caused by a lid margin crust scratching towards the cornea of the child was also discussed. The patient's inability to verbally express his complaints due to his young age left the problem unnoticed by the parents. This led to bacterial superinfection and an ulcer formation with hypopyon. It was impossible to know for certain the exact form of the initial changes because of the late diagnosis.

Treatment was started on the spot which was empirical due to the lack of antibiogram. First, the conjunctival sac was washed with Povidone-iod 5%, then subconjunctival Cefotaxime 5% combined with Atropine 1% and Adrenalin 1% was applied. A topical treatment with Moxifloxacin o.d., Bromfenac t.i.d., Tropicamide 1% t.i.d., Phenylephrine 2,5% t.i.d. and a gel consisting of Dexpanthenol and Hyaluronic acid (Rogodex gel) o.d. was started.

After three days (Fig. 2 A), the ciliary injection was significantly reduced, the cornea was still edematous and there was some infiltration of the ulcer and the limbal pock. A second application of antibiotic plus cycloplegic agent was performed subconjunctivally.

Ten days after the beginning of treatment the inflammation and edema of the cornea had completely resolved, and the ulcer had already epithelized. However, a scar had begun to form (Fig. 2 B). A local corticosteroid was included to increase cornea transparency and block possible immune reactions and neovascular formation. Twenty days after the initial symptoms only two small macules remain without signs of inflammation or neovascularization. Every other therapy was stopped except for the epithelizing gel. The child would be carefully followed up for the next three months for recurrence of the corneal problems. The team decided not to start oral Acyclovir for prophylaxis due to lack of indications.

DISCUSSION

Even though primary varicella is considered a benign and self-limiting disease, in a minority of cases it may

lead to complications such as pneumonia, encephalitis, skin superinfections and invasive bacterial infections. In a study by Bozolla et al. 42.6% of children with primary varicella underwent antibiotic therapy, of which 90% for a complication and 10.0% for the fear of developing complications [9]. Even though bacterial complications from primary varicella are common, bacterial keratitis has rarely been described in the literature. Pedro-Edge et al. presented a clinical case of an adult with suppurative keratitis as a complication of primary varicella infection [10]. Garg et al. described chickenpox as a risk factor for *Moraxella* keratitis [11]. There are no other reports in the literature of hypopyon keratitis following primary varicella in such a young patient up to our knowledge.

Chickenpox eye complications are rare. Yat et al. reported that an estimated 1 in 27 000 cases of primary varicella would require active treatment for ocular complications. Most of the patients with

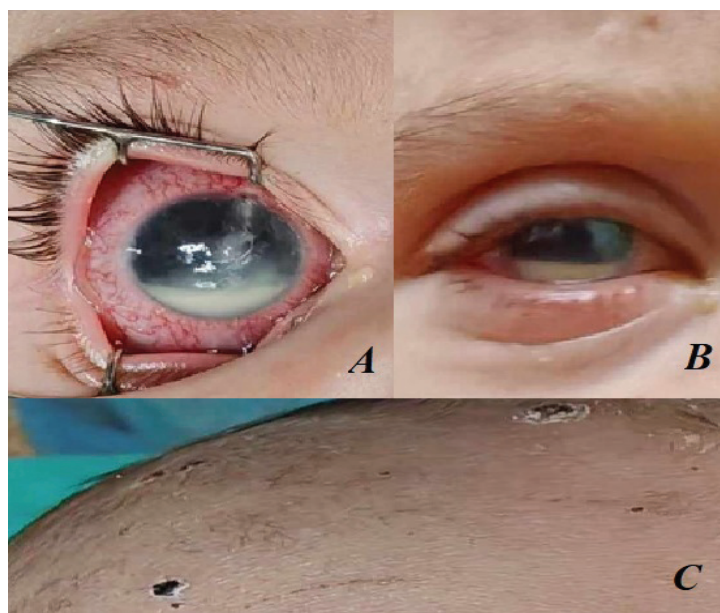


Fig. 1. The patient on the day of hospital admission

- A)** Examination with an eyelid speculum showing ciliary injection, limbal pock and paracentral ulcer both inflamed as well as severe hypopyon
- B)** Hyperemic and edematous eyelid and blepharospasm. The hypopyon can also be observed
- C)** The varicella rash in a phase of healing

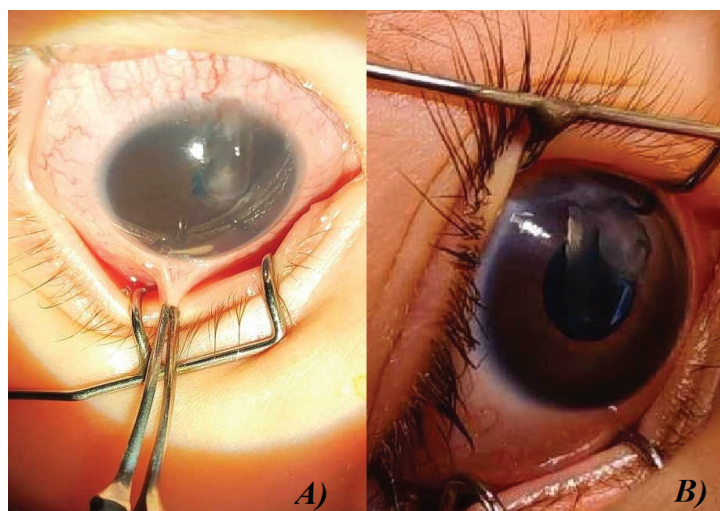


Fig. 2. Follow-up examinations of the patient

- A)** On the day of the second subconjunctival antibiotic plus cycloplegic application three days after the first. The cornea is still edematous, but the infiltration is partially resolved, and the ciliary injection is significantly reduced
- B)** Two small macules remain 10 days (about 1 and a half weeks) after the beginning of treatment

eye complaints had conjunctivitis and lid lesions which settled with only observation [5]. However, ocular complications like acute retinal necrosis and retinal vasculitis from primary varicella infection can sometimes be severe and permanent [12]. Prompt diagnosis is critical to the outcome in some cases.

Even with the slight delay in the diagnosis of our patient, his vision recovered after appropriate treatment with only minor sequelae. Corneal melting and perforation and development of leucoma with neovascularization have been reported in Nigerian patients lacking compliance and follow-up [10]. In some cases of varicella stromal keratitis, a chronic relapsing course is observed with up to six months dependence on corticosteroids [8]. They are caused by an immune reaction which we do not suspect in our case. However, we will follow our patient for at least three months after the cessation of treatment.

CONCLUSION

Even though primary varicella is a relatively benign condition in childhood its possible complications should not be underestimated. Patients with recent varicella infection and ocular complaints should be kept under high suspicion for complications. Pediatricians and primary care clinicians should refer to an ophthalmologist all patients with eye pain and redness, floaters, or blurred vision following primary varicella infection, especially if the complaints persist. Late diagnosis in some cases can lead to devastating visual results. Varicella zoster virus vaccination should be more widely recommended in our country and future implementation of mandatory vaccination should be considered.

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