Development of Acute Hydrops in Eye with Infectious Keratitis: A case report

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Case Report

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Abstract

**Background:** There have been relatively many reports of cases of acute hydrops followed by infectious keratitis, there has not been a report of cases of infectious keratitis complicated by acute hydrops.

**Case presentation:** A 35-year-old man had undergone cataract surgery 7 years earlier and was being treated for atopic dermatitis by a neighborhood dermatologist. However, the dermatitis was poorly controlled. He came to our hospital with a complaint of pain and blurred vision in his left eye. The decimal best-correctly visual acuity (BCVA) of the left eye was 0.01. Slit-lamp microscopy showed conjunctival injection and a corneal opacity. The patient was diagnosed with infectious keratitis and treated with topical and systemic antibiotics. During his hospitalization, we noted that he frequently rubbed his eyes vigorously. Five days after the first visit, the cornea was noted to protrude markedly and the stroma surrounding an ulcerated area was edematous. These findings led to the diagnosis of acute hydrops and he underwent keratoplasty for the impending corneal perforation. Histopathological study of the excised cornea showed stromal edema, leucocyte infiltration, and a tear of Descemet membrane. Unfortunately, he developed endophthalmitis on the day after the surgery. We irrigated the anterior chamber with antibiotics and injected antibiotics into the vitreous. The endophthalmitis gradually subsided, and at two years after the surgery, his decimal BCVA had improved to 0.6.

**Conclusions:** Eye rubbing in cases of infectious keratitis can induce acute hydrops and timely surgical intervention is recommended.

Introduction

Acute corneal hydrops is one of the complications of corneal ectatic disorders, and it is associated with a tear in Descemet's membrane and endothelium leading to an influx of aqueous humor into the stroma causing corneal edema. It can cause eye pain and a reduction of vision. Most cases improve in a few months with conservative therapy. However, some cases will develop complications including corneal infections and corneal perforation (1). Although cases of acute hydrops followed by infectious keratitis have been reported, there has not been a report of cases of infectious keratitis complicated by acute hydrops.

We report our findings in a case of infectious keratitis complicated by acute hydrops that developed in a patient with poorly controlled atopic dermatitis. The patient developed endophthalmitis immediately after a therapeutic keratoplasty and was considered to have a poor visual prognosis, but early diagnosis and treatment enabled us to preserve vision.

Case Presentation

A 35-year-old man came to our hospital complaining of hyperemia and blurred vision in his left eye. He stated that he had been diagnosed with atopic dermatitis and had been treated by a local dermatologist. He had undergone cataract surgery bilaterally at 28-years-of-age and was being followed with atopic
keratoconjunctivitis by a local doctor. He had no other ocular problems, and his decimal visual acuity at the first visit was 1.0 with a correction of -1.00 DC ax 90° in the right eye and 0.01 (uncorrectable) in the left eye. Slit-lamp biomicroscopy showed a dense corneal opacity and an ulcer in the left eye (Fig. 1A). A smear of the corneal scraping showed no obvious microorganism, and cultures of the corneal abrasion was negative. Based on the slit-lamp findings, we suspected infectious keratitis induced by gram-negative bacteria. He was treated by topical 0.3% dibekacin and 1.5% levofloxacin 6/day. On the third day of treatment, a corneal ring infiltrate and abscess appeared in the anterior chamber (Fig. 1B). He was hospitalized and treated with hourly application of topical 0.3% dibekacin and 1.5% levofloxacin, and systemic infusion of imipenem. During his hospitalization, we noticed that he had a habit of rubbing the affected eye vigorously. On the fifth day of treatment, the abscess in the anterior chamber disappeared, but the cornea became markedly edematous. The cornea protruded and had an acute hydrops-like appearance (Figs. 1C, 1D).

Believing that the risk of developing a corneal perforation was high, a penetrating keratoplasty was performed using a fresh cornea. The day after the surgery, a vitreous opacity and bacterial mass-like opacity appeared on the intraocular lens. We diagnosed him with infectious endophthalmitis, and vancomycin and cefazidime were irrigated into the anterior chamber and also injected into the vitreous cavity. The endophthalmitis improved, and we tapered the topical antibiotics gradually. Two years after the surgery, his best-corrected visual acuity in the left eye was 0.6 with a correction of -6.00 DS = -2.25 DC Ax 82°.

Cultures of the aqueous humor collected at the time of surgery and of the excised cornea were negative. Histopathology of the excised cornea showed severe edema of the corneal stroma and the infiltration of leukocytes. There was also a tear in Descemet's membrane (Figs. 2A, 2B).

**Discussion**

Acute hydrops is a rare complication that occurs in approximately 2% of keratoconus cases (2). A tear in Descemet's membrane is associated with a structural weakening of the Descemet's membrane and surrounding cornea. It is believed that repetitive application of pressure to the cornea that exceeds the corneal resistance to them will lead to a rupture of Descemet's membrane (3). The eye rubbing behavior seen in patients with atopic dermatitis may cause a significant increase in the IOP to about 300–400 mmHg, and the increase may increase the risk of developing acute hydrops(4). One of the complications of acute hydrops is infectious keratitis, and it has been reported that acute hydrops occurs in <2% of bacterial keratitis cases (1). It is generally believed that corneal stromal edema and rupture of Descemet's membrane may be factors that predispose the eye to infectious keratitis after the acute hydrops. Because corneal stromal edema causes a widening and irregularity of the collagen layer of the corneal stroma and a reduction of the barrier function of the corneal epithelium, these changes may facilitate the movement of microorganisms into the cornea. This would then induce the rapid progression of bacterial keratitis.
In our case, there was no history of keratoconus, and the acute hydrops developed during the clinical course of the keratitis. Histopathology of the incised cornea showed a rupture of Descemet's membrane, loss of the normal collagen layer, and invasion of inflammatory cells into the cornea. Because similar findings have been observed in reports of acute hydrops complicated by infectious keratitis(5), we considered that the acute hydrops had also developed in our case. We believed that the reason why the patient without keratoconus developed acute hydrops was the intense rubbing of the eye while the cornea was structurally weakened due to the infectious keratitis. We also suggest that these changes, and the rapid expansion of the posterior cornea by the formation of large Descemetocelle caused the tear in the Descemet's membrane which lacks elasticity.

In Asia, therapeutic corneal transplantation has been performed in 9.4% of the cases of infectious keratitis(6). In a study of the National Transplant Registry data of the United Kingdom, the incidence of endophthalmitis after penetrating keratoplasty (PKP) was 0.67%(7), and the risk factors for endophthalmitis after PKP was infectious keratitis, corneal trauma, corneal ulcer, corneal perforation, and a preceding of ophthalmological surgery. In our case, the size of the infectious infiltration was large at 8 mm, and there was a rupture of Descemet's membrane, which is close to a corneal perforation. These factors can explain why this case developed endophthalmitis after the PKP. In cases of therapeutic corneal transplantation at the acute phase of infectious keratitis, the use of fresh comeas is desirable from the perspective of enabling screening for postoperative endophthalmitis.

The prevalence of *Staphylococcus aureus* carriage in the skin, eyelids, and conjunctival sacs in patients with atopic dermatitis has been reported to be significantly higher than in healthy controls(8). The presence of *S. aureus* increases the expression of inflammatory cytokines(9) which can disrupt the barrier function of the epidermis and the corneal epithelium leading to an increased risk of infection from the ocular surface. Furthermore, the eye rubbing behavior commonly observed in patients with atopic dermatitis damages the barrier function of the ocular surface and makes the cornea more vulnerable to infections. Thus, patients with atopic dermatitis are more likely to develop ocular surface infections and may be at a higher risk of developing endophthalmitis as a postoperative complication. Thus, a good control of the atopic dermatitis is important for the prevention of infection and perioperative management.

There was at least one limitation. We cannot deny the possibility of undiagnosed keratoconus preceding the corneal infection because we don't have the data of corneal topography before the onset of corneal infection. However, the affected eye had a good visual acuity before the onset of corneal infection and the patient was unaware of the difference in visual acuity from the other eye, it is unlikely that the patient had keratoconus.

In summary, we report a case that developed acute hydrops at the acute phase of infectious keratitis. The eye rubbing behavior, which is common in patients with atopic dermatitis, should be noted as it can exacerbate the corneal infection and enhance the development of acute hydrops. In addition, if infectious
keratitis is complicated by acute hydrops, the possibility of corneal perforation or endophthalmitis should be considered. Thus, arrangements should be made for a therapeutic corneal transplant.

**Abbreviations**

IOP, intraocular pressure

**Declarations**

**Ethics Approval and Informed Consents:** Ethical approval by the Ethics Committee of Mie University Hospital was waived due to the retrospective nature of the study considering that no personal identifying information of the patients was available from the records and the study was limited to the collection of clinical data.

**Consent for publication:** Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

**Availability of data and materials:** The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

**Competing Interest:** Y Kashima, None; K. Kato, None; M. Takeuchi, None; Y. Yonekawa, None; Y Takashima, None; K. Hirano, None; M. Kondo, None.

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**Authors’ contributions:** KK and YK contributed to the design of the work. YK wrote the original manuscript and KK, MK and KH reviewed it. KK, MT, YY, YK collected the data. All authors reviewed the manuscript.

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**References**


Figures
Figure 1

Slit-lamp images of a patient with infectious keratitis who developed acute hydops.

A: Slit-lamp image at the initial visit. A central corneal opacity and edema of the entire cornea can be seen.

B: Three days after the initial visit. The slit-lamp image shows markedly exacerbated opacity with a ring infiltrate and hypopyon.

C: Diffuse illumination image at five days after the initial visit. The corneal opacity in the central part of the cornea has improved with increased transparency in the peripheral part of the cornea.
D: Direct focal illumination image taken 5 days after the initial visit. The central part of the cornea is edematous and is markedly protruded.

Figure 2

Histopathological findings of the corneal button removed during penetrating keratoplasty.

A: Histopathological image showing a severely swollen corneal stroma. Hematoxylin-eosin staining, original magnification ×100.

B: High magnified view of the squared area in Figure 2A. A tear of Descemet's membrane and infiltration of leucocytes into the surrounding area can be seen. original magnification ×400.