**TITLE: INTRACAMERAL C3F8 IN ACUTE HYDROPS POST PENETRATING KERATOPLASTY**

**ABSTRACT**

Purpose: To present a case of spontaneous Descemet membrane detachment (DMD) presenting as acute hydrops in recurrent keratoconus in a corneal graft managed with intracameral C3F8.

Method: A young male diagnosed with acute hydrops post penetrating keratoplasty for keratoconus was treated with intracameral C3F8 which led to clearing of the corneal graft and obviating the need for a repeat corneal transplant.

Result: To the best of our knowledge, the use of intracameral C3F8 for the management acute hydrops post penetrating keratoplasty has not been reported.

Conclusion: We advocate meticulous slit lamp examination and use of anterior segment imaging to accurately diagnose such a patient and timely intervention with intracameral gas tamponade to avoid the sequalae of acute hydrops.

Keywords:Recurrent keratoconus, Descemet’s detachment, penetrating keratoplasty, acute hydrops, Gas tamponade.

**INTRODUCTION**

Keratoconus is a non-inflammatory ectatic disease of the cornea, with an onset generally at puberty. Acute hydrops in keratoconus, results from rupture of Descemet membrane with aqueous entry into corneal stroma leading to marked stromal and epithelial edema. This is often accompanied with severe loss of vision. Injection of intracameral C3F8 has been reported to be successful in sealing the Descemet membrane breaks and detachment with faster recovery in cases with hydrops in advanced keratoconus.1

There exist anecdotal reports of acute hydrops after penetrating keratoplasty (PK) for keratoconus.2,3 No intervention has however been reported in these cases for the management of hydrops. Most of these cases underwent penetrating corneal graft at a later stage with histological confirmation of the diagnosis. We report a case of recurrence of keratoconus and spontaneous DMD with acute hydrops after PK, successfully managed using intracameral C3F8.

**CASE REPORT**

This study adheres to the tenets of tenets of the Declaration of Helsinki. The surgical case and pictures contain no personal identification information. However, a signed informed consent was obtained prior to the procedure.

A 29-year-old male patient with bilateral keratoconus presented with sudden onset diminution of vision, pain and mild redness in his right eye (OD). He had undergone penetrating keratoplasty with a trephine size of 8.5mm for donor and host cornea in his right eye for Krumeich stage 4 keratoconus and corneal collagen crosslinking for Krumeich stage 1 keratoconus in his left eye a few years later. 10 years later, the patient was diagnosed to have recurrence of keratoconus with bulging of clear graft. The central keratometry (K) reading was 56.7D and maximum K was 78.5D on scheimpflug imaging. The thinnest pachymetry was 380µ at 4 O’clock adjacent to the graft-host junction. There was no history of trauma. He was maintaining a BCVA of 6/6 on Snellen’s visual acuity chart with scleral contact lenses in both eyes.

He presented with a drop of vision one day, the BCVA was 2/60 (OD) and 6/6 (OS). Slit lamp examination revealed the presence of localized inferior graft edema, more at 4 O’clock area with Descemet membrane detachment (DMD). Severe inferonasal corneal thinning was seen and faintly seen Descemet membrane seemed to have slipped from the inferior host graft junction (FIG 1A, FIG 1B). No keratic precipitates or cells were seen in the anterior chamber. The intraocular pressure was 20 and 16 mmHg with tonopen. Left eye had only mild central epithelial haze. Anterior segment OCT (RTVue,OPTOVUE) showed marked inferior stromal edema with localized Descemet’s detachment (FIG 2A). No tear or cleft in the DM was appreciated. A diagnosis of acute hydrops was made and the patient was started on topical hyperosmotics (Hypersol5,JAWApharmaceuticals,INDIA).

The patient was planned for intracameral Isoexpansile 14% perfluoropropane (C3F8) injection in the right eye following partial paracentesis under aseptic conditions at presentation. On the first postoperative day there was slight reduction in the graft edema with air bubble in the anterior chamber (FIG 3A). In addition a small air bubble was trapped between the stoma and DM of the graft inferiorly at 4 O’ clock which was confirmed on ASOCT (FIG 2B).

Post- gas injection 1% predacetate (Predforte ,ALLERGAN), timolol 0.5% bd (Iotim,FDC ltd) and intravenous mannitol 350cc in addition to the topical hyperosmotics were added and patient was advised to maintain a supine position with foot end elevated. At 2 weeks postgas injection the graft edema reduced significantly and predescemetic gas bubble partially resorbed (FIG 3B, FIG 2C). The patient had a BCVA of 6/36. At 3 weeks there was complete resolution of graft edema and resorption of predescemtic air bubble and the DM was attached completely with no areas of separation or scarring (FIG 3C, FIG 3D). This was further confirmed on ASOCT (FIG 2D). The patient regained BCVA of 6/9 with a minimal anterior subcapsular lenticular opacification at 8 weeks. The endothelial count being 1360 cells/mm2.

**DISCUSSION**

Acute hydrops in keratoconus results from tears in descemet’s membrane and manifest as acute severe edema of the corneal epithelium and stroma. This complication can occur spontaneously or after trauma and is seen in 2%to 3% of the patients.4 Corneal edema secondary to acute hydrops is usually self-limiting and improves within 6 to 10 weeks leaving a stromal scar. Cycloplegics, topical steroids, non-steroidal anti-inflammatory agents and hypertonic sodium chloride eye drop or ointment have been advised in the management of this condition.1,5

Intracameral gas/air injection seals the cleft in descemet’s membrane and prevents further seepage of aqueous into corneal stroma resulting in quicker recovery and improvement ofvisual acuity.6

The development of acute hydrops in keratoconic eyes undergoing penetrating keratoplasty has been attributed to the failure to excise the cone completely which can lead to progression of keratoconus in the host with possible involvement of the donor tissue.7,8 Another proposed mechanism is the use of donor tissue with undiagnosed keratoconus or the recurrence of keratoconus in donor corneas, which typically occurs 2 decades after the keratoplasty.9 This condition presents as progressive thinning of the host cornea, usually inferiorly, with secondary astigmatism.

In our case large 8.5mm graft was transplanted on 8.5mm excised ectatic cornea. Progressive bulging of donor cornea and increase in the keratometric readings noted 10 years after the first corneal graft suggested recurrence of keratoconus post penetrating keratoplasty. Sudden onset of pain and graft edema leading to graft opacification and absence of anterior chamber reaction or keratic precipitates suggested acute hydrops.

Acute hydrops in corneal graft differs from that occurring in the advanced keratoconus as the Descemet’s membrane separates from the graft host junction as opposed to a break in the DM in the central cornea as seen in the later.5 Our case also had spontaneous DM detachment from the graft host junction at 4 O’clock area.

The similarity in presentation between certain cases of acute hydrops and the acute endothelial rejection, may lead to patients being treated unnecessarily with intensive, long-term, highly potent topical steroids. This can be avoided like with meticulous slit lamp examination and use of anterior segment imaging to help make an accurate diagnosis like in our case**.**

Review of literature reveals that management of acute hydrops post keratoplasty has largely been conservative and most of the reports had graft failures necessitating repeat corneal grafts. To the best of our knowledge the use of intracameral gas to treat acute hydrops occurring post penetrating keratoplasty has not been reported. C3F8 being the longest acting among all the intraocular gases provides tamponading effect for longer period and usually repeat injections are not required.1 C3F8 acted by unrolling the torn ends of ruptured DM and maintained the tamponade. The entrapped predescemetic gas bubble further attested the sealing of slipped descemet’s membrane from graft host junction at 4 O’ clock by intracameral gas bubble. This facilitated early reattachment of descemet’s membrane and preserved the endothelial function.

**TAKE HOME MESSAGE**

We recommend early intervention with intracameral gas tamponade in post corneal transplant cases presenting with spontaneous Descemet detachment and acute hydrops as it hastens the recovery, shortens the period of edema and loss of endothelial cell function thus preventing the further sequelae of corneal scaring which may obviate the need for a further re-graft in these patients.

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**FIGURE LEGENDS**

**FIG 1A**: Clinical photograph of the patient at presentation with localized inferior graft edema. **FIG 1B**: White arrow shows the Descemet membrane detachment (DMD).

**FIG** **2A**: ASOCT at presentation showing corneal edema with DMD (white arrow). **FIG 2B**: post op day 1 ASOCT showing trapped pre-descemetic air bubble (white arrow). **FIG 2C**: 2 weeks post op ASOCT showing partially absorbed air bubble (white arrow). **FIG 2D**: ASOCT at 3 weeks reveals that DM was attached throughout with no areas of separation or scarring.

**FIG 3A**: Clinical photograph post-op day 1 showing air bubble in the anterior chamber with a small trapped pre-descemetic air bubble between the stroma and DM of the graft inferiorly (white arrow). **FIG 3B**: 2 weeks post op showing marked reduction in corneal edema with partially absorbed air bubble. **FIG 3C, FIG 3D**: Clinical photograph of the patient at 3 weeks post-op with complete resolution of graft edema and an attached DM