



## Partial descemetorhexis for delayed Descemet membrane detachment following penetrating keratoplasty, suggestion of a pathomechanism

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### ABSTRACT

**Purpose:** to report a case of delayed Descemet membrane detachment (DMD) 45 years after penetrating keratoplasty (PK) for keratoconus and its management with a partial descemetorhexis after failed air/gas descemetopexy. A pathomechanism of DMD is proposed based on the anatomic appearance of the DMD and the success of descemetorhexis.

**Observations:** a 60-year old male presented with acute corneal edema of his left eye 45 years after successful PK for keratoconus. Anterior segment ocular coherence tomography (AS-OCT) revealed a wide area of DMD without a visible tear. Reattachment was tried using air and gas descemetopexy with only temporary success. A partial descemetorhexis was then performed just centrally to the graft-host interface and only in the detached area followed by injection of gas bubble. Complete reattachment of Descemet membrane (DM) on the 1st post-operative day was achieved. This anatomical success was maintained 3 months post-surgery and accompanied by decrease of central corneal thickness, however with uncomplete restoration of visual acuity.

**Conclusion:** delayed DMD following KP for keratoconus should be differentiated from acute graft rejection. It could be a result of Descemet tear, but in some cases and despite careful observation of AS-OCT no tear could be seen. In such cases, a tractional detachment of DM might be the underlying pathomechanism and descemetorhexis could help manage it. This new surgical approach might help avoid a re-keratoplasty.

**Importance:** This is the first case report describing success of partial descemetorhexis as a surgical management option for delayed DMD and suggesting a pathogenesis based on this success. This might help avoid re-keratoplasty as a management of this rare complication.

### 1. Introduction

Keratoconus is a progressive thinning and ectasia of the cornea which results in irregular corneal astigmatism and visual loss. Hydrops is an uncommon complication and leads to stromal edema as a result of a tear in the Descemet membrane.<sup>1</sup> Delayed Descemet membrane detachment (DMD) following penetrating keratoplasty (PK) for keratoconus is a rare late complication of PK and is scarcely described in the literature. Frequently, the delayed DMD was referred to as a hydrops although only a minority of these cases showed a tear of Descemet membrane (TDM).<sup>2</sup> In many cases of delayed DMD, the differentiation between presence/ absence of a tear was not possible<sup>3-5</sup> especially where anterior segment ocular coherence tomography (AS-OCT) or ultrasonography (UBM) were not available.

The etiology of delayed DMD is not known and many conservative and operative management options were suggested with variable

outcomes. In this case, we report the incidence of a delayed DMD without a TDM following keratoplasty for keratoconus which was managed with partial descemetorhexis after failed air/ Sulfur hexafluoride (SF<sub>6</sub>) descemetopexy. We suggest a pathophysiology of this rare complication based on the appearance of DMD in AS-OCT and the anatomical success of descemetorhexis.

### 2. Case report

A 60-year-old Caucasian male patient presented to our outpatient department with a history of blurry vision and photophobia in his left eye in the last two days.

The patient had a PK for the left eye 45 years ago. On the last visit 6 months before, he was achieving a visual acuity (VA) of 6/9 (0.63, Snellen Chart) using rigid gas permeable contact lenses (RGCL) with the corneal graft showing a mild degree of endothelial decompensation

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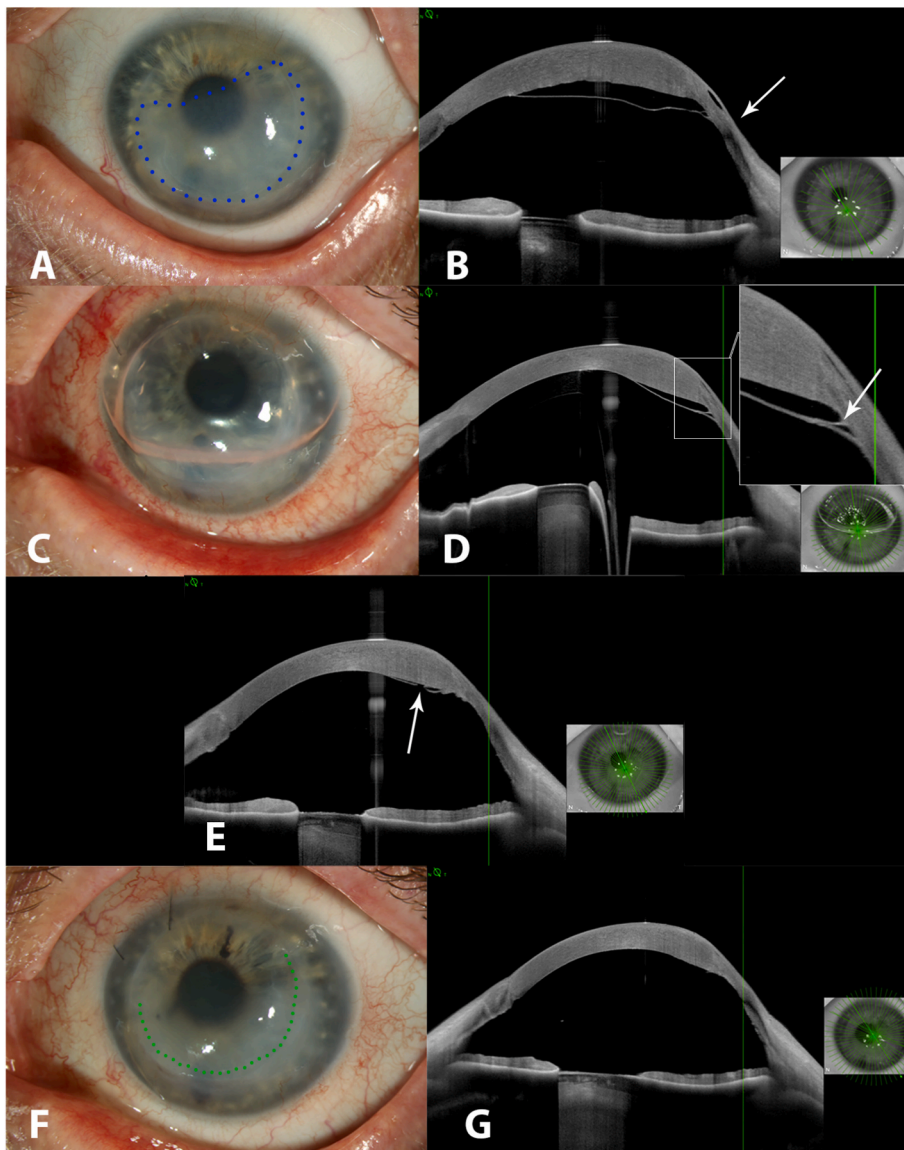
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presenting as localized sectoral subepithelial edema temporally and inferiorly but not involving the optic axis. This mild decompensation was stable through the last 2 years with a central corneal thickness (CCT) of 621 $\mu$ m. The right eye had a PK 25 years ago and showed a VA of 6/6 (1.0) using RGCL with a sectoral endothelial decompensation temporally and inferiorly sparing the visual axis.

Examination of the left eye on the day of presentation revealed a VA of hand motion (HM) and a mild conjunctival injection. The corneal graft showed severe stromal and epithelial edema in the inferior temporal quadrant being extensive in the host-donor interface and extending superio-nasally to involve the visual axis (Fig. 1 A, dotted blue line) and the host corneal side. CCT was 1077 $\mu$ m. There were no endothelial precipitates, no anterior chamber cells or flare. Fundus examination was within normal limits.

The differential diagnosis of the case included acute graft rejection, chronic endothelial decompensation and delayed DMD. Performing AS-OCT (Anterior, Heidelberg Engineering GmbH, Heidelberg, Germany) revealed a large area of DMD (Fig. 1 B) extending from the graft-host interface to the graft and involving about 7 clock hours (From 2 to 6 till 9) and about 70% of the cornea accompanied by an increased thickness of the graft tissue with subepithelial cysts. A thorough examination of multiple scans performed using the high resolution swept

source AS-OCT in different directions failed to show a rupture point but revealed an attached Descemet in the interface with a hyperreflective stroma implementing a scarred tissue (Fig. 1 B, white arrow). These findings confirmed the diagnosis of delayed DMD. The management plan included starting with a local steroid dose of Dexamethason eye drops 5 times a day with Cyclopentolate eye drops 3 times a day. As no improvement was observed in the first week and the DMD included a wide area, we didn't expect a spontaneous regression and proceeded with a surgical intervention with air descemetopexy. This was done under local anesthesia through a clear corneal incision superio-nasally without complications. Patient was instructed to maintain a strict supine position with eyes looking upwards. The examination on the 3rd post-op day revealed a significant regression of the corneal edema with the Descemet being attached centrally, but still detached in the para-interface area. The visual acuity improved to 6/38 (0.16) and the CCT decreased to 636 $\mu$ m. The resorption of the air bubble in the next days was however accompanied with recurrence of the DMD and increase of corneal edema which almost reached the primary situation. The VA deteriorated to HM. After one week, another try of re-attaching the Descemet membrane using 20% SF6 gas was also performed and showed a significant success achieving a VA of 6/240 (0.025), a temporary improvement was seen with attached Descemet membrane only



**Fig. 1.** A: AS photo on the day of presentation. Blue dotted line resembles area of DMD B: AS-OCT corresponding to A. white arrow points to hyperreflective scarred area C: AS photo after gas descemetopexy. D: AS-OCT corresponding to C. white arrow shows supposed tractional membranes E: AS-OCT after descemetorhexis. F: AS photo 3 months following descemetorhexis. Green dotted line shows extension of descemetorhexis G: AS-OCT corresponding to F. AS: anterior segment, AS-OCT: anterior segment ocular coherence tomography. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

centrally with persistent detachment peripherally (Fig. 1 C and D) and reduction of CCT to 999 $\mu$ m. After gas resorption on day 14 a total recurrence occurred. The loss of a visible break of the DM, consistent detachment peripherally despite Descemetopexy and recurrence following resorption of air and gas raised our suspicion of another pathomechanism. After thorough explanation and discussion of the risks and management options, an informed consent was obtained and a partial descemetorhexis was performed in the detached area from 2 to 6 till 9 o'clock slightly centrally to the interface (Fig. 1 E white arrow) using a Descemet incision hook (Geuder AG, Heidelberg, Germany). The procedure was performed under local anesthesia as known in the first steps of Descemet membrane endothelial keratoplasty with sparing the non-detached Descemet by 9–12 and 2 o'clock (Fig. 1 E, dotted green line). SF6 was injected and the patient was told to maintain strict supine body position with eyes looking upwards. The post-operative examination on day 1 showed a complete resolution of the DMD and an improvement of corneal edema (CCT of 814 $\mu$ m) which was stable on the last follow up after 3 months achieving a VA of 6/60 (0.1) and a CCT of 796 $\mu$ m (Fig. 1 F and G).

### 3. Discussion

A few cases of corneal hydrops and DMD following PK were described in the literature. Most of them occurring in eyes previously having severe keratoconus<sup>2–10</sup> but also in non-keratoconic eyes.<sup>11,13</sup> The presence of TDM was not seen in all cases. In some cases where tears were described, no AS-OCT or UBM were performed which makes it very difficult to confirm/exclude the presence of Descemet rupture. Some authors depend in their diagnosis on the presence of a break in Descemet membrane seen in the extracted corneal graft after performing PK.<sup>3,11</sup> Such a tear is indeed inevitable in case of a DMD if performing a PK even when no tear was present preoperatively. The pathomechanism of DMD in the absence of a TDM is not known. Matthew Gorski et al. described the absence of TDM in AS-OCT in two cases and suggested two mechanisms: a retrocorneal membrane or a progressive keratoconus on the host side.<sup>5</sup> Mechanical traction through anatomical changes of the graft interface or stretching and progressive ectasia was also proposed as a pathomechanism.<sup>2</sup> Treatment of hydrops cases with DMD ranged from observation which resulted in scarring and regression of edema after several months<sup>4,6</sup> or in no improvement.<sup>6</sup> Surgical intervention with air/gas (SF6) descemetopexy was reported to succeed in some cases but not in all.<sup>9</sup> Some cases needed a lamellar<sup>10</sup> or a penetrating re-keratoplasty.<sup>3,11</sup>

In our case, we had a large DMD of about 7 clock hours extending from the graft-host interface beyond the center of the graft. AS-OCT showed a wide DMD without a tear. This was very unlikely to respond to conservative management. Two attempts to reattach the Descemet membrane with air and SF6 gas were unsuccessful. A failed reattachment after air/ gas descemetopexy suggests a different etiology than TDM. A retrocorneal membrane being a possible mechanism was excluded previously<sup>5</sup> although such membranes were also present in our case (Figure-1 D Inset, white arrow). Observing the AS-OCT images and the clinical picture of the cornea, we suggest a tractional mechanism to be the cause of DMD. This traction might occur through progression of keratoconus over many years on the host side of the cornea leading to no-balance situation on the two sides of interface where Descemet membrane is firmly attached in the scarred area and causing a tractional force and detachment on either side of the interface as the elasticity of Descemet membrane and the posterior stroma are very different.<sup>12</sup> Another possibility could be the remodeling process in the interface area over the years which might result in thinning on one side more than the other causing this traction followed by a detachment. Such a traction mechanism was proposed by Vivienne Kit et al.<sup>2</sup> "Releasing" this traction seems to be crucial to reattach Descemet membrane. To do so, the descemetorhexis should "overcome" the source of traction which seems to be at the interface. A similar principle is used in retinal surgery in

cases of detachment related to proliferative vitreoretinopathy. Performing a retinotomy/ retinectomy here is essential to release the traction and allow retinal reattachment. Performing a partial descemetorhexis in the detached area and just centrally to the interface resulted in our case in complete resolution on the first post-operative day. The incision directly on the interface would not have released this traction and was unsuccessful in relieving the detachment in other study.<sup>5</sup>

The anatomical success of our case was accompanied with an uncomplete clinical improvement (VA improved from HM to 6/60 (0.1), CCT from 1077 to 796 $\mu$ m). This could be the result of already partially decompensated endothelium seen before the DMD. This might have decompensated after 3 surgical interventions in a relatively short period of time and through the mechanical manipulation on the detached Descemet membrane through the air/SF6 descemetopexy and during the descemetorhexis. A re-KP was still an option for our patient to improve vision.

### 4. Conclusion

Delayed DMD following KP could have different pathomechanisms and should be differentiated from hydrops of naïve keratoconic eyes and acute graft rejection. Performing AS-OCT in these cases may be helpful to lead management plan. The presence of a TDM might suggest improvement using air/gas descemetopexy. However, the absence of a TDM and resistance to air/gas descemetopexy suggest a tractional detachment which may only respond to a partial descemetorhexis and thus help to avoid re-keratoplasty.

### Patient consent

This report does not contain any personal information that could lead to the identification of the patient.

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