Intracorneal blood removal six weeks after canaloplasty

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In a 71-year-old patient with bilateral open-angle glaucoma, intracorneal blood was found after a canaloplasty procedure in the right eye. Six weeks after surgery on ultrasound biomicroscopy examination, liquified blood and blood clots could be observed nasally in the deep corneal stroma close to the Descemet’s membrane. The intracorneal blood was washed out with balanced saline solution following deep corneal incision and lamellar dissection. Descemet’s membrane was reattached with air injection into the anterior chamber. Two months later, visual acuity improved to 20/50, intraocular pressure was 16 mm Hg without medication and confocal microscopy showed deep stromal folds and limited endothelial cell loss. Viscoelastic entering the cornea at Schwalbe’s line and reflex of blood from the collector channels to Schlemm’s canal account for corneal hematoma. Even six weeks after canaloplasty, successful blood removal could be fulfilled without rupturing the Descemet’s membrane.

Key words: Canaloplasty, confocal microscopy, intracorneal blood removal, ultrasound biomicroscopy

Intracorneal hemorrhage is an uncommon event. Mostly single case reports show that it occurred after cataract surgery, corneal grafting, traumatic hyphema, corneal ulcers, chemical burns and viscocanalostomy.[1,2] Canaloplasty is a recent non-penetrating glaucoma surgical procedure, similar to viscocanalostomy,[3] but for the placement of a circumferential suture within the Schlemm’s canal. Complete success rate of 77.5% at 36 months with minimal intraoperative and postoperative complications,[4,5] including one intracorneal hematoma,[6] were reported after canaloplasty.

Case Report

In a 71-year-old patient with end-stage bilateral open-angle glaucoma, the best-corrected visual acuity was 20/40 and 20/20 in the right and left eye, respectively. Intraocular pressure (IOP) was 24 mmHg in the right and 19 mmHg in the left eye.
with 0.03% bimatoprost plus 0.5% timolol. Bilateral nuclear cataract and posterior synechiae were recorded. Canaloplasty was performed in the right eye uneventfully; 1.4% sodium hyaluronate was used as viscoelastic. On day 1, postoperative intracorneal blood was misdiagnosed as a retrocorneal hemorrhage. The patient has been followed elsewhere. After six weeks, the visual acuity had decreased to count fingers, IOP was 18 mmHg without medication and dark intracorneal blood involving the upper nasal quadrant and the visual axis was evident at slit-lamp examination [Fig. 1a]. The patient had no bleeding disorders. Ultrasound biomicroscopy (UBM Paradigm P45, Salt Lake, UT, USA) evidenced low (liquefied blood) and high (blood clots) reflective areas in the predisecemetic space [Fig. 1b], large intrascleral lake and moderate suture indentation within Schlemm’s canal.

A limbal partial-thickness incision was performed nasally with a 30-degree knife, followed by deepening and dissection with a crescent knife to reach the predisecemetic space, where balanced saline solution was delivered by a 27-gauge cannula to wash out the blood. This maneuver caused central Descemet’s membrane detachment, and an air bubble was injected at normal pressure into the shallow anterior chamber. Peripheral iridectomy was performed superotemporally to avoid pupillary block. The self-sealing corneal incisions were not sutured.

After two months visual acuity recovered to 20/50 and IOP was 16 mmHg without medication. Corneal stromal edema slowly faded, but a nasal peripheral scar at the limbic incision was still evident [Fig. 1c]. Confocal microscopy showed endothelial cell polymegathism, increased Descemet’s membrane reflectivity (223 IU), deep stromal folds, anterior stromal edema and rare activated keratocytes [Fig. 1d]. Central endothelial cell count was 1473/mm² in the right and 1852 mm² in the left eye. No noteworthy changes in IOP, visual acuity and anterior segment of the right eye were recorded in the following 12 months.

Discussion

The essentials of canaloplasty include the insertion of a microcatheter (iScience Interventional) into Schlemm’s canal with the aid of high-molecular weight viscoelastic, thus stretching the trabecular meshwork, the openings of the collector channels and dilating Schlemm’s canal. The microcatheter is withdrawn and only a tightened 10-0 Prolene suture is left within the canal. After suturing the outer flap with 10-0 Vicryl, viscoelastic is injected into the scleral lake under the flap to test for water tightness. If too much viscoelastic is injected during the entire surgical procedure, this may rupture the Descemet’s membrane.

A limited lysis of Descemet’s membrane during the introduction of sodium hyaluronate under the superficial scleral flap was believed to have caused intracorneal hemorrhage after viscocanalostomy in one single case, which was successfully managed with an intentional break of Descemet’s membrane and injection of sulfur hexafluoride gas into the anterior chamber. The same complication was observed in our patient where, in our opinion, viscoelastic entering the cornea at Schwalbe’s line and reflux of blood from the collector channels into Schlemm’s canal occurring close to the scleral lake. Distinguishing between postoperative intracorneal and retrocorneal blood requires very careful slit-lamp examination, and unfortunately in our patient the intracorneal blood was misdiagnosed.

Our technique was similar to that reported both in a case of intraocular lens explant with deep corneal vessels ingrowth and a quite recent case where the canaloplasty microcatheter encountered an obstacle at the 6 o’clock position. In both patients, a hemorrhagic detachment of Descemet’s membrane occurred and the blood was soon removed without incising the Descemet’s membrane by making a partial-thickness paracentesis and injecting an air bubble into the anterior chamber with a Rycroft cannula.

In our patient two months after corneal blood removal, the cornea was transparent with no blood staining and moderate endothelial cell loss, but in vivo confocal microscopy showed increased reflectivity of anterior stroma and Descemet’s membrane together with deep stromal folds. In our case six weeks after canaloplasty, UBM could show the presence of intrascleral lake and suture tension within Schlemm’s canal, both indicators of a successful canaloplasty, the extent and details of blood clots and liquefied blood in the predisecemetic space. Similarly on UBM, Kachi et al. detected an internal low-echogenic (liquefied blood) cyst 10 months after onset of a spontaneous intracorneal hematoma which was almost resolved but still visible even 20 months later. Actually corneal blood staining may take up to three years to clear spontaneously, but, in order to prevent possible further endothelial cell loss, it is best to aspirate it as soon as possible, especially when the hematoma covers the pupillary area.

In conclusion, in our patient viscoelastic entering the cornea and reflux of blood from Schlemm’s canal during canaloplasty resulted in a corneal hematoma, which even six weeks later was successfully removed without rupturing Descemet’s membrane. Our simple surgical technique could be useful, should this rare complication occur after canaloplasty.

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