

Case Report

Late-Onset Inadvertent Bleb Formation following Pars Plana M3 Molteno Implant Tube Obstruction

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Keywords

Inadvertent bleb · Glaucoma · Optical coherence tomography · Molteno implant

Abstract

Purpose: To report a case of inadvertent bleb formation presenting 18 months after pars plana M3 Molteno implant tube obstruction in a patient with mixed mechanism glaucoma.

Materials and Methods: An 84-year-old Caucasian male with mixed mechanism glaucoma underwent slit-lamp examination, gonioscopy, colour anterior segment photography and anterior segment optical coherence tomography (AS-OCT). **Results:** An inadvertent bleb developed 18 months after pars plana implant tube re-positioning with a 6/0 Vicryl tie ligature. The bleb was located in the area anterior to the implant plate; it was characterised by a thin, transparent, avascular and multi-cystic wall, with a visible stoma at the posterior edge of the bleb. The bleb was functioning as demonstrated by an intraocular pressure of 6 mm Hg at presentation and a punctate fluorescein uptake pattern of the bleb wall. The bleb over the plate of the Molteno implant was non-functioning, likely secondary to tube obstruction by vitreous in the early postoperative period. AS-OCT showed a tract from the anterior chamber commencing at an entry wound through a corneal tunnel to the posterior stoma at the base of the inadvertent bleb. **Conclusions:** We hypothesise that the pathophysiologic factors resulting in an inadvertent bleb are a result of a combination of apoptosis, late-onset wound dehiscence and internal gaping of a centrally placed corneal wound. In addition, aqueous hydrodynamic factors may play a role.

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Introduction

Inadvertent filtering blebs have been reported following cataract surgery [1] and secondary to corneal wick syndrome [2] and pterygium surgery [3]. Complications include hypotony, macular oedema, shallow anterior chamber, choroidal detachment, dellen formation and blebitis that can progress to sight-threatening endophthalmitis [1].

We present a case of inadvertent filtering bleb 18 months after tube obstruction following pars plana Molteno implant tube re-positioning for mixed mechanism glaucoma.

Case Report

An 84-year-old Caucasian male was referred to the Ophthalmology Department with recent deterioration in vision in his left eye after an admission for an acute cerebrovascular accident. His past ocular history included bilateral age-related macular degeneration resulting in a blind right eye secondary to a disciform scar and treated left choroidal neovascularisation, as well as previous bilateral YAG peripheral iridotomies for mixed mechanism glaucoma. He was on topical treatment with Combigan BD and Latanoprost nocte OU. An M3 175 mm² Molteno implant was inserted in the left eye 5 years prior to presentation. It was noted in the immediate postoperative period that the tube position was through a peripheral corneal tunnel. This was followed by uncomplicated left phacoemulsification cataract extraction and posterior chamber intraocular lens implant 2 years later. Pars plana vitrectomy, implant tube repositioning into the pars plana and 6/0 Vicryl tie ligature were undertaken 1 year later due to early corneal decompensation, noted by the presence of a deep stromal corneal opacity opposite the tube lumen. Failure of bleb functioning was noted in the early postoperative period, evidenced by a failure of bleb formation at the expected Vicryl tie released 6–8 weeks postoperatively; this was likely to be secondary to vitreous incarceration in the tube. A decision was made to manage the complication conservatively and resume topical treatment.

On examination at the current presentation, visual acuity was 6/30 and intraocular pressure was 6 mm Hg OS. Slit-lamp examination of the anterior segment of the left eye revealed a multi-cystic bleb located in the region anterior to the implant plate (Fig. 1a). The bleb wall was thin and transparent with a visible stoma in the base at the posterior edge of the bleb (Fig. 1b). There was a characteristic punctate fluorescein uptake pattern over the bleb wall indicative of microcyst formation. There were 2 patent superior peripheral iridotomies and a superior temporal corneal scar at the previous tube insertion site. Cup/disc ratio was 0.85; there was retinal pigment epithelial disturbance at the macula.

Anterior segment optical coherence tomography (AS-OCT) of the left eye revealed a tapering 5.3-mm tract extending between the anterior chamber and the inadvertent bleb, connecting the limbal stomal entry wound to the posterior stoma at the base of the inadvertent bleb. The subconjunctival region appeared diaphanous, and the multi-cystic nature of the bleb was noted (Fig. 2). AS-OCT of the internal wound demonstrated gaping “fish-mouthing” of the internal wound lip (Fig. 3)

On re-assessment 2 weeks after ceasing topical treatment, intraocular pressure was 8 mm Hg, and there was improvement of visual acuity to 6/21 OS. The ocular condition remains stable at 6 months of follow-up.

Discussion

External fistulisation connecting the ocular aqueous compartment to either the ocular surface or the subconjunctival space is a known complication of filtration surgery and aqueous shunts [4]. We postulate that the inadvertent bleb in our case was a result of a combination of 3 main factors: (1) factors related to apoptosis; (2) factors related to corneoscleral wound healing; and (3) factors related to aqueous hydrodynamics.

Factors Related to Apoptosis

The results of the Otago Glaucoma Surgery Outcome Study, through histological, immunohistochemical and electron microscopy studies, demonstrated that the normal life cycle of capsules in both primary and secondary glaucoma includes an outer fibroproliferative zone characterised by collagen synthesis, fibroblasts, tissue macrophages and capillaries [5]. Renewal of this layer is balanced by an inner fibrodegenerative surface disintegration, being associated with apoptosis and breakdown of tissue matrix components, which becomes more marked over time, mediated by internal receptor ligand interactions and perhaps accelerated by ischaemia from a compromised systemic circulatory state in this case [6, 7].

Factors Related to Corneoscleral Wound Healing

The cornea and sclera are connective tissues consisting of collagen fibrils (mainly type I) embedded in a proteoglycan-rich extrafibrillar matrix. The collagen forms multi-layered lamellae stabilized by covalent cross-links; this arrangement provides the mechanical strength in these tissues [8].

The corneal stroma comprises about 90% of the total corneal thickness and is responsible for most of the corneal tensile strength [9]. Because of its avascularity, healing of corneal stromal wounds is slower than in other connective tissues. Ongoing histopathological changes in human corneal laceration wounds have been observed years after injury, despite meticulous original suture closure, and dehiscence can occur spontaneously [9].

The normal radial stress forces in the cornea as a result of intraocular pressure can be mathematically approximated assuming the cornea to be a spherical surface [8]:

$$\sigma = (p \times r)/2t,$$

where σ = radial stress within the surface plane resulting from the internal pressure, p = intraocular pressure (Pa), r = radius of curvature (m), and t = thickness of the cornea (m). The relationship between these factors is demonstrated in Figure 3. For our patient, from the preoperative biometric assessment with $r = 8.07$ mm, the average corneal thickness at the wound site is $978 \mu\text{m}$ as determined by AS-OCT, giving a theoretical mean radial stress in the corneal wound of $52.92 \times 10^2 \text{ N/m}$ ($539.6 \times 10^3 \text{ g/mm}$) at 10 mm Hg.

In a study by Gasset et al. [10] on the tensile strength of central and limbal corneal wounds in an animal model, the mean tensile strength in an unwounded limbal cornea was observed to be $1,742 \pm 284 \text{ g/5 mm}$ of cornea. During the first 12 postoperative days after the limbal wound, no tensile strength could be measured; slow healing began afterwards; the mean tensile strength ranged from 224 ± 108 to $198 \pm 60 \text{ g/5 mm}$ corneal wound. Therefore, wound dehiscence occurs when the radial stress forces exceed the tensile strength of the wound or if disruption of the collagenous lamellae reduces the tensile strength. Common pathophysiologic mechanisms include trauma, suture malfunction, scleral melt, obesity, old age and prolonged steroid use [1, 11]. Delayed wound dehiscence can occur due to abnormal

collagen structure in systemic disorders, such as in Marfan syndrome [12] and diabetes [13]. A number of growth factors and their associated receptors, including epidermal growth factor, transforming growth factor- β , keratinocyte growth factor, hepatocyte growth factor, fibroblast growth factor and platelet-derived growth factor, regulate functions including mitosis, differentiation, motility and apoptosis, which play a vital role in corneal wound healing, mediating the proliferation of epithelial and stromal tissue and affecting the remodelling of the extracellular matrix [13]. The internal shape of the wound has been known to influence the occurrence of inadvertent blebs. Gimbel et al. [14] highlighted the long-term influence of internal wound gape, which is termed “fish-mouthing.” If left uncorrected, internal wound gape has been known to cause delay in primary wound healing with aqueous egress to the deep portion of the tunnel and subconjunctival space [14].

In our case, old age, systemic circulatory compromise combined with the fish-mouth configuration of the internal wound and the centrally shifted tube entry wound through a corneoscleral tunnel, encouraged by a relatively shallow anterior chamber at the initial surgery, likely contributed to this clinical picture.

Factors Related to Aqueous Hydrodynamics

The effect of shear stress on the wall of a fistula tract is a well-known physiologic phenomenon. Several strategies are used in clinical practice to modify this force and increase the patency of prosthetic arterial grafts, assist in the design of graft anastomoses and promote the adhesion of endothelial cells to an arterial graft wall. Furthermore, this shear stress may result in the synthesis and secretion of factors implicated in the pathogenesis of vascular aneurysms [15].

We postulate that both laminar and turbulent aqueous flow currents generate shear stress forces acting along the fistulous tract originating at the corneal wound. The estimated average shear stress force on the fistula wall could be represented by the equation valid for either turbulent or laminar flow [16]:

$$\tau_w = (D \times \Delta p) / 4l,$$

where D = diameter of the fistula ($\approx 168.29 \mu\text{m}$ averaged measurement over 7 sections along the fistula length measured on an anterior segment OCT), Δp = pressure gradient ($\approx 1.999835 \text{ kPa}$ assuming 10 mm Hg of intraocular pressure gradient), and l = length of the fistula ($\approx 5,297 \mu\text{m}$ as measured by anterior segment OCT):

$$\begin{aligned} \tau_w &= (168.29 \times 10^{-6} \times 1.3332 \times 10^3) / 4 \times 5,297 \times 10^{-6} \\ &= 10.59 \text{ Nm}^{-2} = 1.059 \times 10^{-5} \text{ N.mm}^{-2} \end{aligned}$$

These shear stress forces may play a role in initiating and maintaining the patency of the fistulous tract.

Conclusions

We hypothesise that the pathophysiologic factors resulting in an inadvertent bleb are a result of a combination of apoptosis, late-onset wound dehiscence and internal gapping of a centrally placed corneal wound. In addition, aqueous hydrodynamic factors may play a role.

Statement of Ethics

The patient's informed consent has been obtained.

Disclosure Statement

The co-author is the inventor of the Molteno implant.

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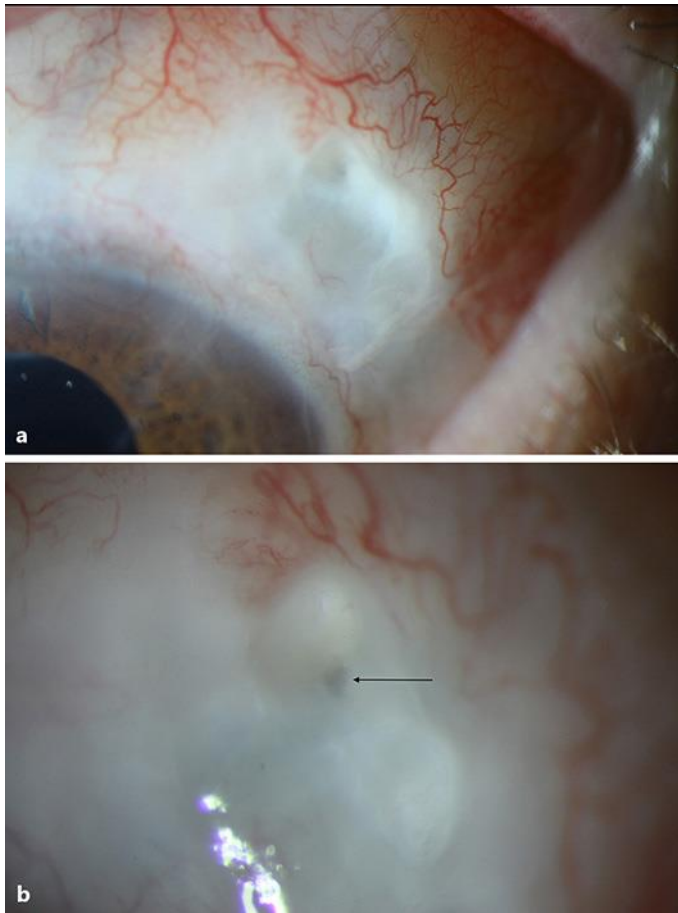


Fig. 1. Colour anterior segment photograph of the left eye. **a** There is a multi-cystic, avascular thin-walled bleb anterior to the non-functioning superior temporal M3 Molteno implant. **b** Arrow indicating stoma at the base of the inadvertent bleb.

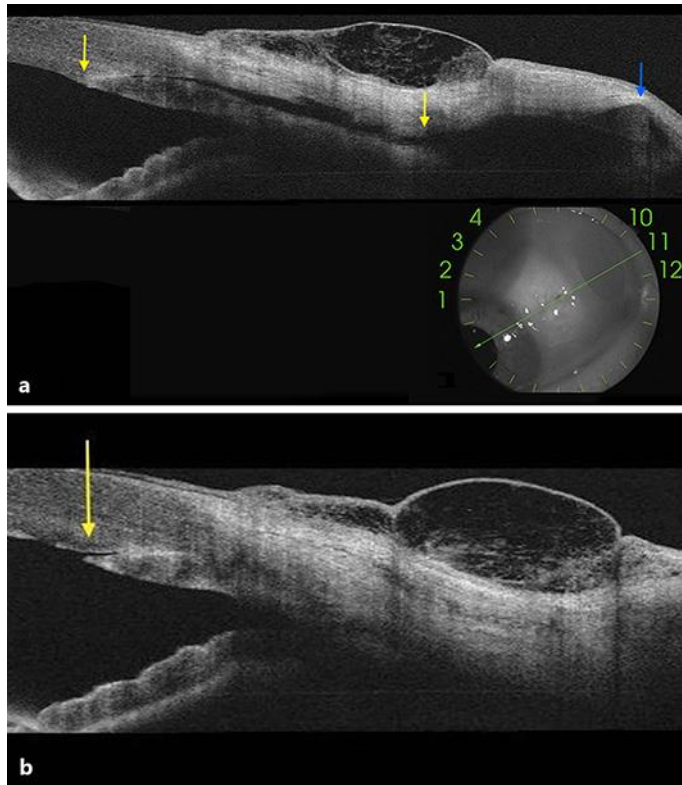


Fig. 2. Anterior segment DRI Triton swept source optical coherence tomography of the left eye. **a** The fistulous tract extends from the corneal tunnel to the subconjunctival space anterior to the implant (yellow arrows). The anterior ridge of the Molteno implant M3 plate is seen just behind a multi-cystic thin-walled bleb (blue arrow). **Inset** showing the orientation of the radial scan coinciding with the original tube pathway. **b** Fish-mouthing of the internal wound measuring 57 μm in separation (yellow arrow).

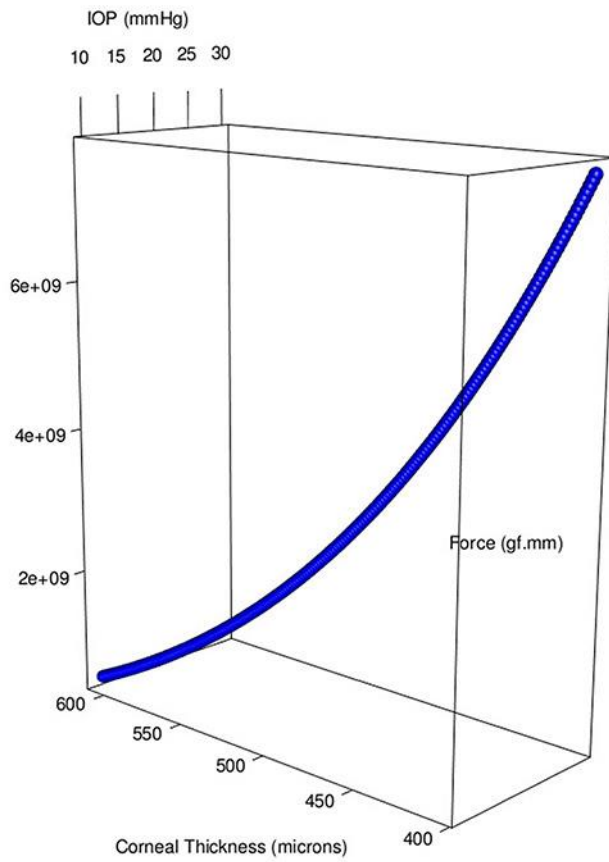


Fig. 3. Theoretical normal radial tension forces in the cornea as a function of corneal thickness and intraocular pressure (IOP).