

# Chronic hypotony management using endoscopy-assisted vitrectomy after severe ocular trauma or vitrectomy

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## Abstract

• **AIM:** To report outcomes of endoscopy-assisted vitrectomy (EAV) in patients with chronic hypotony following severe ocular trauma or vitrectomy.

• **METHODS:** This was a retrospective, noncomparative case series. Ciliary bodies were evaluated using ultrasound biomicroscopy pre-operatively and direct visualisation intraoperatively. All selected individuals (seven patients/seven eyes) underwent EAV. Removal of ciliary membrane and traction, gas/silicone oil tamponade (GT/SOT), and scleral buckling (SB) were performed in selected eyes. Outcome measurements mainly included intraocular pressure (IOP) and best-corrected visual acuity (BCVA).

• **RESULTS:** Seven eyes from 7 male aphakic patients with a mean age of 45 (range, 20-68)y were included in this study; the average follow-up time was 12 (9-15)mo. GT was performed in 2 eyes; membrane peeling (MP) and SOT in 2 eyes; and MP, SOT, and SB in 3 eyes. The mean pre- and post-operative IOP were 4.5 (range, 4.0±0.11 to 4.8±0.2) mm Hg and 9.9 (range, 5.6±0.17 to 12.1±0.2) mm Hg at 52wk (12mo), respectively. BCVA improved in six eyes; one eye still showed light perception, and no bulbi phthisis was observed.

• **CONCLUSION:** Endoscopy offers improved judgment and recognition and has an improved prognosis for chronic hypotony. Therefore, endoscopy can be an effective and promising operative technique for chronic traumatic hypotony management.

• **KEYWORDS:** endoscopy-assisted vitrectomy; chronic hypotony; anterior proliferative vitreoretinopathy; anterior vitreous segment; trauma

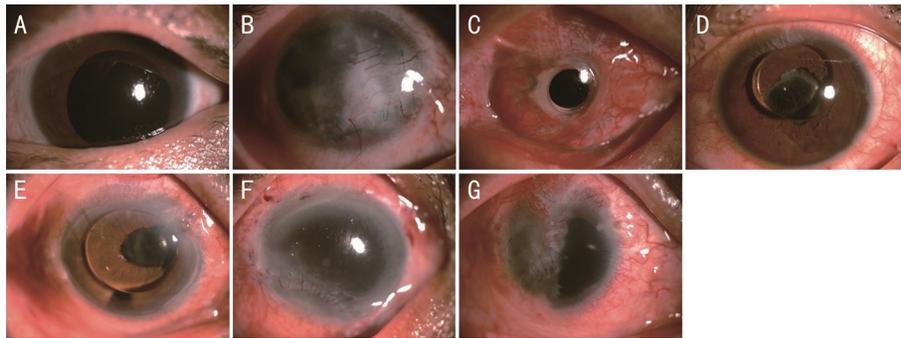
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## INTRODUCTION

Hypotony is defined as an intraocular pressure (IOP) of less than 5 mm Hg<sup>[1-2]</sup>. Chronic hypotony is usually attributed to glaucoma surgery, retinal detachment (RD) or choroidal detachment, ocular trauma, proliferative vitreoretinopathy (PVR), or chronic uveitis<sup>[2-3]</sup>. Prolonged ocular hypotony can result in pain and an unacceptable aesthetic appearance of the eye, which could result in permanent and devastating consequences, including poor vision, maculopathy, optic neuropathy, or bulbi phthisis.

Certain methods for treating chronic ocular hypotony include ciliary body condensation adhesion, intraocular corticosteroid therapy, injection of viscoelastic material, and pars plana vitrectomy (PPV) with gas tamponade (GT) or silicone oil tamponade (SOT)<sup>[2-4]</sup>. Recently, foldable capsular vitreous body (FCVB) implantation has been used to restore the ocular structure and maintain the IOP<sup>[5-7]</sup>, but there are concerns about some complications, such as infections, glaucoma, sacculus displacement, and severe inflammation. Additionally, a capsular tension ring placed in the iridocorneal angle to treat chronic hypotony has been proposed<sup>[8]</sup>, although its application is limited. Of note, none of these procedures has shown great long-term benefits.



**Figure 1 Anterior segment photographs of the patients** A: Patient 1 had severe blunt ocular trauma, with a transparent cornea and absence of the posterior capsule and aphakia. B: Patient 2 had ruptured globe injury, with an irregular corneal scar, leukoma, and aphakia; the other posterior segments could not be clearly observed. C: Patient 3 had severe alkali burns with keratoprosthesis, absence of the posterior capsule, and aphakia. D and E: Patients 4 and 5 had retinal detachment post-vitrectomy with a transparent cornea; however, the small pupil was caused by synechiae and posterior capsular opacification and aphakia. Silicone oil tamponade was applied in the vitreous body. A silicone oil bubble could be seen in the anterior chamber. F: Patient 6 had a severe ruptured globe injury, with a corneal scar in the inferotemporal region, absence of a posterior capsule, and aphakia. G: Patient 7 had a severe explosion injury and post-vitrectomy, with a corneal scar in the temporal region, absence of a posterior capsule, and aphakia.

PVR, especially anterior proliferative vitreoretinopathy (aPVR), occurs in cases of severe ocular trauma and/or multiple vitrectomies. aPVR often proliferates over the anterior vitreous segment (AVS), including the ciliary body, leading to a structural disorder and chronic hypotony, which may ultimately result in bulbi phthisis<sup>[9-10]</sup>. As aPVR is difficult to identify and visualise in the affected eyes, it is difficult to address the AVS. Even with ultrasound biomicroscopy (UBM) or anterior segment optical coherence tomography, it is difficult to evaluate the correct tissues of the AVS behind the iris for structural disorders and media opacity.

Endoscopy-assisted ocular surgery is a relatively old technique that is increasingly being recognised for its application in cases of vitreoretinal diseases. It was first introduced in 1934 when Thorpe<sup>[11]</sup> removed an intraocular foreign body using integrated forceps. The endoscope must cross the anterior segment to capture images using the distal tip. Therefore, an endoscope allows us to observe any part of the retina without limitations. It is a useful tool for undetectable RD repair (especially for undetectable breaks in the peripheral retina), PVR, especially aPVR, neovascularisation glaucoma, removal of retinal intraocular foreign bodies, endophthalmitis, subluxed intraocular lens, and ocular trauma<sup>[12-14]</sup>. Obviously, the endoscope enables visualisation of the operative field in cases of media opacity or microcornea, and the AVS, including the anterior vitreous body, ciliary body, and ciliary process without scleral indentation can be easily viewed<sup>[14-15]</sup>.

Chronic hypotony is an intractable problem that remains to be solved. There have been sporadic case reports of endoscopy-assisted ophthalmologic surgery for chronic hypotony treatment<sup>[16-19]</sup>. Thus, in our case series, we describe surgical outcomes associated with ophthalmological endoscopy to

examine AVS in patients with chronic hypotony with opaque media or microcornea following severe ocular trauma or vitreoretinal surgery; moreover, we verified the possible causes of hypotony and suggested a promising method for treatment.

## SUBJECTS AND METHODS

**Ethical Approval** The study followed the tenets of the Declaration of Helsinki and complied with the Healthy Insurance Portability and Accountability Act of 1996. We obtained Institutional Review Board (IRB) approval of General Hospital of Southern Theater Command (approval number: NZLLKZ2022194). Informed consent was obtained for endoscopy-assisted PPV from all patients.

A retrospective chart review was conducted for all patients treated with endoscopy-assisted vitrectomy (EAV) for chronic hypotony by one vitreoretinal surgeon (Zou YP) at the Department of Ophthalmology, General Hospital of Southern Theater Command of PLA, between January 2014 and December 2018. Hypotony was defined as an IOP<5 mm Hg for two consecutive visits within at least 1mo or an IOP<5 mm Hg at one visit with the presence of hypotony maculopathy or choroidal effusions upon examination. The exclusion criteria were less 5mo of follow-up, no history of hypotony, giant tear-related RD, proliferative diabetic retinopathy, or an inoperable eye (*e.g.*, closed funnel RD) at the time of surgery. The patients' demographic characteristics are summarised in Table 1, and the anterior segment photographs and UBM examinations before surgery are shown in Figures 1 and 2, respectively.

Visual acuity, IOP, and examination findings were recorded at pre-operative and post-operative visits. All IOP measurements were performed using a Goldman slit lamp-mounted tonometer (Haag-Streit International, USA), and the average of three

**Table 1 Pre-operative and post-operative characteristics of patients**

Patient (eye)	Age (y)/sex	Cause	Duration of hypotony	Number of prior surgeries	Primary diagnosis	Surgical technique (assisted by endoscopy)	Media opacity, state of the cornea and lens	Pre-operation (UBM)	Intraoperation (endoscope)	Complication
Patient 1 (L)	45/male	Severe blunt ocular trauma	>24mo	6	Blunt ocular trauma	Vitreotomy, photocoagulation, GT	Cornea is transparent, aphakic eye	CB detachment in one quadrant	Velvet vitreous body, invisible vitreous gel could be seen, CB was white and finger-shaped, CP was almost normal, RD slightly in polar peripheral region	No
Patient 2 (R)	44/male	Severe ocular trauma	6mo	4	Ruptured globe injury	Vitreotomy, photocoagulation, GT	Severe corneal scar and leukoma, aphakic eye	CB detachment in two quadrants, suspected membrane covered the CB	Velvet vitreous body, invisible vitreous gel could be seen, CB was white and finger-shaped, CP was normal, membrane slightly covered on the CB, slight RD	No
Patient 3 (L)	42/male	Severe alkali burn	>24mo	6	Ocular alkali burn	Vitreotomy, MP, photocoagulation, SOT	Keratoprosthesis, aphakic eye	Eye did not undergo UBM	CP depigmentation, sheets of pigmented tissue, stiff vitreous fibres and membrane traction, scattered atrophy CP, the retina was attached in the posterior area while detached slightly in the peripheral area	No
Patient 4 (L)	36/male	Post-vitreotomy for RD	>24mo	3	RD, post-vitreotomy	Vitreotomy, MP, photocoagulation, SOR, SOT	Cornea is transparent aphakic eye, small pupil with synechiae and PCO, SOT in the vitreous body	CB detachment and cleft, membrane covered the CB		No
Patient 5 (R)	68/male	Post-vitreotomy for RD	>24mo	5	RD, post-vitreotomy	Vitreotomy, MP, photocoagulation, SOR, SOT, SB	Corneal is almost transparent, aphakic eye, small pupil with synechiae and PCO, SOT in the vitreous body	CB detachment, other disordered structures of the AVS	Cyclitic membrane covered on the CB, pigmented granulations, some CP atrophy, CB was covered by membrane, the retina was attached in the posterior area while detached slightly in the peripheral area	No
Patient 6 (R)	58/male	Severe ocular trauma	>24mo	2	Ruptured globe injury	Vitreotomy, MP, photocoagulation, SOT, SB	Scar in the inferotemporal cortex, aphakic eye	Eye did not undergo UBM	Stiff and wrinkling membrane covering CB and leading to traction, vitreous gel or vitreous fibers could be seen, the retina was attached in the posterior area while detached slightly in the peripheral area	No
Patient 7 (R)	20/male	Severe explosion injury	24mo	3	Explosion injury, post-vitreotomy	Vitreotomy, MP, photocoagulation, SOR, SOT, SB	Scar in the temporal cortex, aphakic eye, SOT in the vitreous body	CB detachment, disordered structures of the AVS		No

R: Right eye; L: Left eye; RD: Retinal detachment; PVR: Proliferative vitreoretinopathy; GT: Gas tamponade; CB: Ciliary body; CP: Ciliary process; MP: Membrane peeling; SOR: Silicone oil removal; SOT: Silicone oil tamponade; PCO: Posterior capsular opacification; SB: Scleral buckling; AVS: Anterior vitreous segment.

measurements was calculated. IOP was measured at roughly the same time during the day. Best-corrected visual acuity (BCVA) was measured pre-operatively and at each post-operative visit. A standard projected Snellen chart was used. All the patients underwent anterior segment photography, and two patients missed UBM before vitrectomy (Patients 3 and 6).

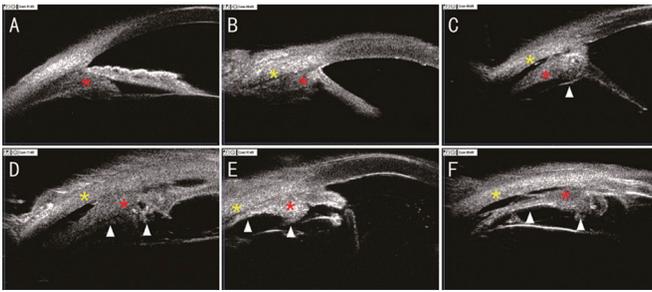
**Surgical Technique** The patients had severe ocular trauma or post-vitreotomy, and conventional three-port PPV was performed using a 23-gauge trocar-cannula system in all the patients. Corneal limbal tunnel incisions were performed at the 9 o'clock to 3 o'clock meridian if needed. An endoscopy system (Poly-Diagnost, Germany) was used to visualise the AVS without scleral depression and to assist in the vitrectomy; the AVS mainly included the anterior chamber angle, ciliary body, and anterior vitreous body. Endoscopy was used to assist in the manipulation of the anterior proliferative vitreous membrane.

Using endoscopy, the specific conditions of the fundus and AVS could be visualised in each patient and different surgical strategies could be applied for specific situations. Vitrectomy was considered in every patient to clear up the vitreous body and retinal membrane, cyclitic membrane, or other proliferative membranes. In addition, retinal photocoagulation was conducted in every patient to ensure tight attachment of the retina. Alternatively, GT was applied to ciliary body traction and with/without slightly peripheral RD. Membrane peeling (MP) was performed for cases with obvious membrane involvement, such as retinal membrane or cyclitic membrane, and severe membrane traction between them. SOT was used for patients with extensive RD with a rigid retina and ciliary body detachment, with scales >1 and 2, and GT could not help to flat retina and the ciliary body. For cases with severe vitreous proliferation or aPVR, more extensively stiff and rigid retina, or inferior retinal breaks, scleral buckling was applied. Further, silicone oil removal (SOR) was considered when the vitreous cavity was filled with silicone oil.

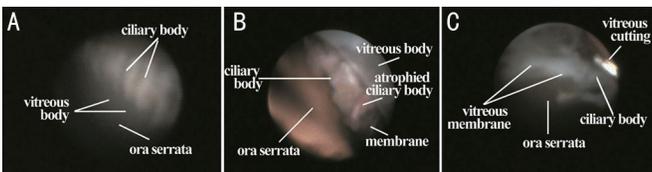
**RESULTS**

The average patient age was 45y (range, 20-68y). Seven patients were males. Visual acuity, IOP, and the examination findings, including UBM results, anterior segment photographs, and direct visualisation *via* an endoscope, were recorded at the pre-operative and post-operative visits. The median follow-up duration was 12mo, with a range of 9-15mo.

**Pre-operative UBM Scans** UBM in Patient 1 revealed single-quadrant ciliary body detachment. UBM in Patient 2 revealed that approximately two quadrants had ciliary body detachment and suspected slight membrane covering on the ciliary body. UBM in Patient 4 revealed ciliary body detachment and cleft in one quadrant, and the membrane appeared to cover the



**Figure 2 Anterior segment shown using UBM** A: Normal structure of the anterior segment. The chamber angle, ciliary body, scleral spur, and sclera can be recognised. Red asterisks show the ciliary body. B-F: Anterior segments of Patients 1, 2, 4, 5, and 7, respectively; yellow asterisks reveal the ciliary body detachment, and white triangles indicate the cyclitic membrane on the ciliary body. UBM: Ultrasound biomicroscopy.



**Figure 3 The view of the anterior vitreous segment shown using endoscopy** A: Normal structure of the ciliary body, velvet-like vitreous body, and ora serrata. B: AVS of Patient 4, the vitreous body, ora serrata, and normal and atrophied ciliary body could be seen, and a membrane covered the ciliary body. C: AVS of Patient 7, the vitreous cutting and ora serrata could be seen. While the ciliary body was difficult to find, a cotton-like membrane gathered on the surface of the ciliary body and led to traction. AVS: Anterior vitreous segment.

ciliary body. UBM in Patient 5 revealed ciliary detachment and disordered structures in the AVS. UBM in Patient 7 revealed ciliary detachment and disordered structures in the AVS (Figure 2).

**Direct Vision of the Anterior Vitreous Segment by Endoscopy** The AVS could be clearly detected upon examination using endoscopy (Figure 3), which could help select the appropriate surgical method.

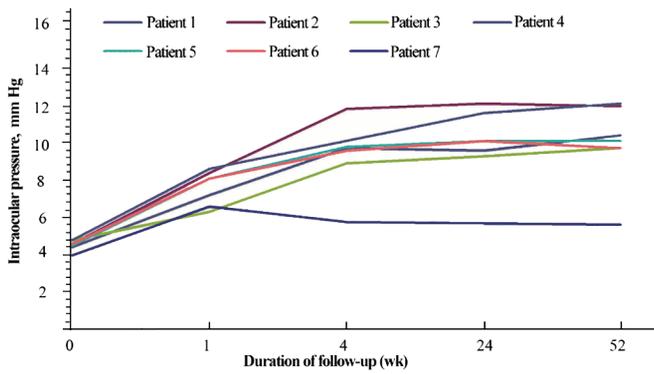
Patients 1 and 2 (two eyes): endoscopy revealed a ciliary body that was white and finger-shaped; the ciliary processes were normal, the vitreous body appeared velvet-like and invisible with vitreous gel, and pigmented granulations clustered in the AVS. While endoscopy did not reveal membrane covering or membrane traction on the ciliary body in Patient 1, some slight membrane covering on the ciliary body, but not membrane traction, was detected in Patient 2. The fundus in both eyes were detached slightly; especially, the retina was detached in the polar peripheral region in Patient 1, which could not be detected by direct fundus examination. The two eyes underwent EAV combined with photocoagulation and GT.

Patients 3 and 4 (two eyes): endoscopy revealed that anterior

proliferative vitreous membranes covered the ciliary body closely in nearly all the quadrants, resulting in an aPVR. The ciliary body presented depigmentation or white granulation in the anterior vitreous body; sheets of pigmented tissue and stiff vitreous fibres were observed, and slight membrane traction was observed in certain areas of the ciliary body. The ciliary processes were covered by membranes; the structures of most of them were normal, but certain scattered atrophied ciliary processes were observed. Using endoscopy, the vitreous body for Patient 4 was filled with silicone oil; in both eyes, the retina was attached in the posterior area but slightly detached in the peripheral area. As the fundus in both eyes were detached slightly, the eyes underwent EAV combined with epiciliary MP and SOT, while Patient 4 underwent an additional operation involving SOR.

Patients 5, 6, and 7 (three eyes): in Patients 5 and 6, endoscopy revealed cyclitic membranes that closely covered the ciliary bodies in nearly all the quadrants, resulting in ciliary body traction and ciliary body detachment, as well as ciliary body cleft in certain quadrants. In addition to ciliary body process depigmentation, pigmented granulations in the anterior vitreous base were detected. The ciliary processes were covered by membranes; the structures of most of them were normal, but some scattered atrophied ciliary processes were observed. Overall, the conditions of these patients were worse than those of Patients 3 and 4. In Patient 7, the eye had severe ciliary body detachment and severe aPVR-affected membranes owing to severe ocular trauma and a history of several vitrectomy operations. Endoscopy revealed ciliary body traction caused by stiff and wrinkling cyclitic membranes, as well as high-density vitreous gel or fibrous vitreous membranes. Most ciliary processes were atrophied, with structural integrity defects or depigmentation, or some sheets of pigment tissues or scars were observed. As the fundus in two eyes (Patients 5 and 7) were filled with silicone oil, the retina in all the three eyes were attached in the posterior area but slightly detached in the peripheral area, which were observed using endoscopy. Additionally, certain focal retina and uvea defects in the temporal middle peripheral region were found intraoperatively using endoscopy. Then, the three eyes underwent EAV, combined epiciliary MP, SOT, and scleral buckling. Finally, the ciliary body was exposed completely, and Patients 5 and 7 underwent an additional operation involving SOR.

The fundus of all the eyes revealed varying degrees of RD in certain quadrants intraoperatively by endoscopy; much more severe proliferative membrane covered the retina surface in Patients 6 and 7. The retina was flat following all the surgeries. During the follow-up time, silicone oil was still retained in the vitreous cavity in Patients 3-7, and band-shaped corneal degeneration has not occurred yet in the patients' eyes.



**Figure 4 Pre- and post-operative average intraocular pressure (IOP) in individual patients** Six patients showed an increase in IOP, and only one eye of one patient maintained hypotony.

**Table 2 Intraocular pressure in patients following surgery for chronic hypotony**

Patient	Pre-operation (mm Hg)	Post-operation (mm Hg)			
		1wk	1mo	6mo	12mo
Patient 1	4.4±0.10	7.2±0.23	9.7±0.20	9.6±0.15	10.4±0.11
Patient 2	4.6±0.15	8.4±0.15	11.8±0.15	12.1±0.20	12±0.15
Patient 3	4.8±0.20	6.3±0.26	8.9±0.10	9.3±0.10	9.7±0.11
Patient 4	4.8±0.10	8.6±0.17	10.1±0.17	11.6±0.15	12.1±0.20
Patient 5	4.5±0.17	8.1±0.15	9.8±0.15	10.1±0.20	10.1±0.11
Patient 6	4.6±0.10	8.1±0.21	9.6±0.15	10.1±0.20	9.7±0.11
Patient 7	4±0.11	6.6±0.10	5.8±0.15	5.7±0.15	5.6±0.17

**Intraocular Pressure Outcomes** EAV was performed within an appropriate time period in all the 7 patients. The median pre-operative IOP was 4.5 mm Hg ranging from 4.0±0.11 to 4.8±0.2 mm Hg. Post-operatively, all the cases demonstrated an immediate increase in the IOP, and the average IOP was 7.6 mm Hg (range, 6.6±0.1 to 8.6±0.17 mm Hg) at 1wk after the operation. The average IOP was 9.4 mm Hg (range, 5.8±0.15 to 11.8±0.15 mm Hg) at 4wk (1mo) after the operation. During the follow-up, the IOP remained stable at 24wk (6mo) following the operation, with an average of 9.8 mm Hg (range, 5.7±0.15 to 12.1±0.2 mm Hg), and the IOP was stable at 52wk (12mo) following the operation, with an average of 9.9 mm Hg (range, 5.6±0.17 to 12.1±0.2 mm Hg). Among the 7 eyes, only one eye maintained hypotony with 5.6±0.17 mm Hg at 52wk (12mo), even with SOT (Patient 7); this eye has not developed bulbi phthisis thus far (Table 2, Figure 4). Figure 4 shows the IOP changes of the 7 patients.

**Visual Acuity** One eye (Patient 1) was transparent without any media opacity, and the others showed media opacity. One eye had keratoprosthesis (Patient 3), three eyes had corneal scars and leukomas (Patients 2, 6, and 7), and two eyes had small pupils attributed to synechiae and posterior capsular opacification (Patients 4 and 5). At the 24-week (6-month) visit, all the eyes maintained stable visual acuity, and six eyes showed obviously increased BCVA. Pre- and post-operative

**Table 3 Best-corrected visual acuity in patients pre- and post-operation**

Patient	Pre-operation	Post-operation			
		1wk	1mo	6mo	12mo
Patient 1	HM	20/2000	20/500	20/500	20/200
Patient 2	HM	HM	CF	CF	CF
Patient 3	LP	HM	20/400	20/400	20/250
Patient 4	LP	HM	20/200	20/100	20/100
Patient 5	HM	CF	20/1000	20/1000	20/1000
Patient 6	LP	HM	20/1000	20/500	20/500
Patient 7	LP	LP	LP	LP	LP

CF: Counting figure; HM: Hand motion; LP: Light perception.

BCVA in all the patients was hand motion (HM) and 20/500, HM and counting figure (CF), light perception (LP) and 20/500, LP and 20/100, HM and 20/1000, LP and 20/500, and LP and LP in Patients 1, 2, 3, 4, 5, 6, and 7, respectively. At 52wk (12mo), BCVA was 20/20, CF, 20/250, 20/100, 20/1000, 20/500, and LP in Patients 1, 2, 3, 4, 5, 6, and 7, respectively. Only one of seven eyes (Patient 7) did not maintain stable visual acuity; the other eyes revealed no visual loss. There were no severe complications in any of the eyes (Table 3).

## DISCUSSION

This case series suggests that EAV could be useful for chronic hypotony management following severe ocular trauma or vitrectomy. Endoscopy can clearly reveal the fundus, the severity of the membrane or fibrous traction of the ciliary body and the anterior vitreous body, and the quantity and severity of ciliary process damage or atrophy, even if the media (cornea in most cases) are opaque. In our cases, three eyes had severe opaque cornea (Patients 2 and 7) or keratoprosthesis (Patient 3) and two eyes had severe iris posterior synechia (Patients 4 and 5); therefore, it was difficult to observe all the intraocular structures in these eyes. Using endoscopy, the retina, peripheral retina, and AVS could be observed easily. Although the media were transparent in Patient 1, it would be helpful to find the essential cause of hypotony in RD in the polar peripheral region, which could be missed by routine examination, and ciliary body detachment in one quadrant through endoscopic direct vision and to make decisions on the selection of surgical procedures.

Of note, hypotony occurs under conditions of increasing aqueous outflow or decreased aqueous humour production. The possible reasons are as follows. First, rhegmatogenous or giant RD, extensive choroid exposure, or chorioidectomy could increase the aqueous outflow to the absorbing compartment of the retinal pigment epithelium and the choriocapillaris, or suprachoroidal effusions could occur. Second, cyclodialysis cleft or ciliary body detachment could increase the aqueous outflow. Third, ciliary body injury attributed to chemical or mechanical factors could result in reduced ciliary body secretion and increased aqueous uveoscleral outflow<sup>[16,20-21]</sup>. In

addition, some researchers have proposed that chronic traction of the AVS and aPVR could also result in hypotony<sup>[17-18]</sup>. Chronic hypotony occurs when the influencing factors persist. In our cases, severe ocular trauma or multiple vitrectomies caused intractable chronic hypotony. We presume that ocular injury directly results in ciliary body detachment or ciliary body atrophy, scar tissue formation or proliferative membrane damage, causing ciliochoroidal detachment, or ciliary body disorder.

The management of chronic hypotony is limited and challenging. Cryopexy and GT were performed in simple cases, while scleral buckling, PPV, and SOT were considered in complex situations. Helbig and Foerster<sup>[22]</sup> first reported pars plana cryopexy and GT for detached ciliary body; these procedures could reattach the cyclodialysis and increase the IOP. When encountering chronic hypotony secondary to complicated chronic uveitis, complicated RD, and/or aPVR, intravitreal silicone oil injection combined with PPV or scleral buckling should be performed. Lewis and Verdaguer<sup>[19]</sup> conducted a prospective study of 17 eyes of consecutive patients with chronic hypotony and reported dissection of the epicyliary membrane tissue using the pars plana approach. Lee *et al*<sup>[17]</sup> found that EAV with the removal of aPVR from the ciliary body is an effective treatment for chronic hypotony. Recently, some new surgical approaches have been proposed. Some researchers found that FCVB was effective and safe for eyes with severe ocular trauma or silicone oil-dependent eyes with severe RD, but these were cases of fresh eye injury or fresh RD without rigid or stiff retina or retina membrane<sup>[5,6,23]</sup>. Zhang *et al*<sup>[24]</sup> considered that vitrectomy combined with silicone oil endotamponade results in better short-term improvement in the treatment of no LP caused by severe ocular trauma than silicone oil-filled FCVB. In this regard, FCVB can only be applied in limited cases. Besides, the surgery could cause infections, glaucoma, sacculus displacement, severe inflammation, FCVB rupture, and silicone oil leakage.

We employed a traditional microscope to perform operations through clear anterior media in the series of patients; however, accessing the AVS was challenging. Although scleral depression can be used, it may distort the normal anatomy of the AVS, it is unsafe and difficult to perform vitrectomy or peel the ciliary body membrane, and it is difficult to visualise the operative zone adequately<sup>[13-14]</sup>. In cases of ocular trauma, nearly all the media are opaque, and accessing the intraocular area using conventional microscopy is challenging; however, intraocular endoscopy provides a high magnification with panoramic, unobstructed, and undistorted views of the AVS behind the iris<sup>[12,14]</sup>. Boscher and Kuhn<sup>[25]</sup> observed 2000 consecutive eyes that underwent vitrectomy for RD with aPVR; they indicated that endoscopy provided

a unique evaluation and therapeutic tool for anterior vitreous proliferation. Hammer and Grizzard<sup>[18]</sup> indicated that traction elongation of ciliary processes was associated with ocular hypotony, and proliferative membranes or fibrosis around the ciliary body could be evaluated and visualised using intraocular endoscopy.

In our cases, nearly all the eyes had slight RD. Besides, we found that nearly all the eyes revealed structural abnormality of the ciliary body and ciliary process. Two eyes (Patients 1 and 2) had only ciliary body detachment in one quadrant, while the ciliary processes were normal without covered or tractional membrane in one eye (Patient 1). In addition, one eye (Patient 2) had slightly cyclitic membrane covering the ciliary body. However, 5 eyes (Patients 3, 4, 5, 6, and 7) had proliferative membranes covering the ciliary body, accompanied by ciliary detachment and/or ciliary body cleft or ciliary process atrophy. Three eyes (Patients 5, 6, and 7) had severe ciliary body detachment and membrane traction on the ciliary body and the anterior vitreous body, with ciliary process atrophy in certain orientations, while one eye (Patient 7) had severe aPVR and severe ciliary process atrophy extensively. Therefore, EAV and other operative techniques were applied for specific situations as mentioned before. There was a significant increase in the IOP following surgery in 6 eyes (Patients 1-6), and the IOP remained stable at the follow-up examinations. Visual acuity improved obviously in 6 eyes (Patients 1-6), while just one eye (Patient 7) maintained an IOP lower than 5 mm Hg with no changes in visual acuity.

The study by Lee *et al*<sup>[17]</sup> showed that IOP improved in patients who tended to be younger and who had fewer previous intraocular surgeries, while our study showed persistent hypotony in one eye (Patient 7). In this previous study by Lee *et al*<sup>[17]</sup>, the outcomes of patients with hypotony secondary to aPVR after previous RD repair were observed, and IOP improved because of the exposure of the ciliary body after aPVR stripping. Thus, we could infer that the function of the ciliary process is normal in people without aPVR. Unlike RD patients in the study by Lee *et al*<sup>[17]</sup>, Patient 7 in the present study underwent AVS detection and membrane stripping and releasing using EAV. The patient had a severe explosion injury with a cornea debridement surgery and history of several vitrectomy surgeries. In addition, we observed severe ciliary body detachment, severe aPVR, stiff and wrinkling cyclitic membranes, and atrophied ciliary processes with structural integrity defects. The IOP was still lower than 5 mm Hg after management. Although the patient was a young man, and SOT is beneficial for retinal reattachment and IOP maintenance, the reason for the low IOP was possibly his ocular dysfunction following severe ocular trauma, especially the state of the atrophied ciliary process with structural integrity defects or

scars. We inferred that, first, severe ocular trauma damaged the ciliary body function by mechanical trauma, which led to the atrophied ciliary process and reduction of the aqueous fluid content. Second, the ocular trauma caused inflammation that altered membrane development and led to RD and ciliary body traction and atrophy, which lasted for a long duration. Third, since ocular repair had been performed following the severe ocular trauma, certain focal retina and uvea defects in the temporal middle peripheral region observed *via* intraoperative endoscopy might have also contributed to the aqueous fluid outflow.

Few studies exist on the mechanisms and pathology of chronic hypotony, especially hypotony following severe ocular trauma or vitrectomy. Recognising all the initial factors involved in chronic hypotony is often challenging, and it is identified by experience in most cases. O'Connell<sup>[26]</sup> suggested that ciliary process dysfunction is caused by tractional or other damage, which results in the misdirection of the aqueous fluid into the suprachoroidal space. Kim *et al*<sup>[9]</sup> developed a model of chronic hypotony in pigmented rabbits by injecting cultured rabbit dermal fibroblasts in the epiciliary area following vitrectomy. This model could be useful in elucidating the pathophysiology of hypotony and identifying an optional treatment, such as epiciliary membrane dissection. In our cases, not all the eyes had normal IOPs, and we inferred that ciliary process atrophy, membrane traction, and direct traumatic damage to the ciliary body decreased aqueous production, resulting in persistent hypotony. Other eyes had positive outcomes concerning IOP and visual acuity, perhaps because in cases of slight ciliary body damage or dysfunction and membrane or fibrous traction, the structure or function of the ciliary body can still maintain a balance between aqueous inflow and outflow. One limitation was the small sample size. Further studies over long periods should be performed to investigate the long-term sufficiency of the technique.

In summary, chronic hypotony management is a challenging avenue. Our study suggests that endoscopy may be an appropriate method, as it offers improved judgement and recognition and has an improved expected prognosis for chronic hypotony; thus, patients may show better recovery of ocular function and IOP than those treated using standard methods. Additionally, we suspect that chronic hypotony is closely related to the state of the ciliary body, membrane traction, and ciliary body detachment; however, the sample size was small in this series. The pathophysiology of chronic hypotony is complicated, and further research is warranted.

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