Non-invasive monitoring of cyclodialysis cleft using anterior segment optical coherence tomography and its role in informing clinical treatment decisions

Thomas Andrew Berk a,⁎, Daniel Peretz a,⁎, Alaa Mofti a,b,c,d, Béatrice Des Marchais e, Hady Saheb a,b,e

a Department of Ophthalmology & Visual Sciences, McGill University, 5252, Boulevard de Maisonneuve West, 4th Floor Montreal, Québec, H4A 0A4, Canada
b Department of Ophthalmology, Ministry of the National Guard - Health Affairs, 2383, Al ‘Ullyaa Dist, JEDDAH, 22384 – 7864, Saudi Arabia
c King Abdullah International Medical Research Center, 7719, Al ‘Ullyaa Dist, JEDDAH, 22384 – 2565, Saudi Arabia
d King Saud Bin Abdulaziz University for Health Sciences, 2686, Al ‘Ullyaa Dist, JEDDAH, 22384 – 7607, Saudi Arabia
e Centre Universitaire d’Ophtalmologie, CHU de Québec-Université Laval, 1656, Chemin Ste-Foy, Québec City, Québec, G1S 4L8, Canada

⁎ Corresponding author.
E-mail addresses: thomas.berk@mail.mcgill.ca (T.A. Berk), daniel.peretz@mail.mcgill.ca (D. Peretz), alaa.mofti@mail.mcgill.ca (A. Mofti), beatrice.desmarchais@fmed.ulaval.ca (B.D. Marchais), hady.saheb@mcgill.ca (H. Saheb).

1 These authors contributed equally to this work and share co-first authorship.

1. Introduction

A cyclodialysis cleft represents the separation of the longitudinal fibres of the ciliary muscle from the scleral spur. This creates a direct channel from the anterior chamber to the suprachoroidal space leading to increased uveoscleral outflow as well as decreased aqueous production secondary to ciliary body detachment, both resulting in hypotony. Complications of cyclodialysis clefts include ciliochoroidal effusion, induced hyperopia, cataract, hypotony maculopathy, and optic disc edema.1–3

Cyclodialysis clefts are currently encountered almost exclusively in the context of blunt ocular trauma or as a complication of intraocular surgery.4 Gonioscopy is the gold standard for diagnosis but can be challenging due to shallowing of the anterior chamber and iris apposition, hazy media, and corneal folds upon indentation in hypotonous eyes.1,4,5 In these situations, ultrasound biomicroscopy (UBM) and anterior segment optical coherence tomography (AS-OCT) have been used to aid in the diagnosis.3,4,6 Smaller clefts have been reported to close spontaneously or with topical medical therapy alone, but larger and more persistent clefts may require more invasive approaches.
including laser and/or surgical treatments. Permanent structural sequelae such as macular scars or optic atrophy seem to be more predictive of the long term visual acuity prognosis in chronic clefts rather than the duration of hypotony maculopathy alone.

There are very few currently published descriptions of the use of AS-OCT in the diagnosis and management of cyclodialysis clefts. To our knowledge, none have reported on its usage to inform treatment decisions. We present a series of two patients in whom AS-OCT was used to diagnose and then monitor the progression of conservatively managed cyclodialysis clefts. Serial imaging directly informed our decision to defer more invasive therapeutic options over the course of each case’s treatment. The patient journeys and risk profiles were significantly altered by the information provided by AS-OCT.

2. Case series

2.1. Case 1

A 51-year-old Caucasian man with bilateral uncontrolled intraocular pressure (IOP) was referred for surgical consideration. His highest pressures on record were 36 mmHg and 34 mmHg in the right and left eyes, respectively. The referring ophthalmologist had recently performed bilateral selective laser trabeculoplasty (SLT) with minimal effect, with evidence of bilateral structural and functional progressive damage. On presentation to our clinic, his IOPs were 30 mmHg in the right eye and 27 mmHg in the left eye on four topical glaucoma agents bilaterally and oral acetazolamide. Iris transillumination defects were visible with open angles showing moderately pigmented trabecular meshwork and clear lenses in both eyes. A diagnosis of bilateral uncontrolled pigmentary glaucoma was made, and the patient was scheduled for gonioscopy-assisted transluminal trabeculotomy (GATT) in each eye.

The right eye’s intra- and postoperative course was uneventful, with a stable IOP in the mid-teens achieved at 6 weeks without medication. The right eye’s GATT was likewise routine, however examination on postoperative day 4 revealed a slight relative shallowing of the anterior chamber and an IOP of 2 mmHg. AS-OCT imaging using the CASIA SS-1000 system (Tomey Corporation, Nagoya, Japan) revealed a nasal cyclodialysis cleft spanning 39° of arc with adjacent ciliochoroidal effusions measuring 623 μm in maximal height (Fig. 1A). Attempted gonioscopy was unsuccessful, as the shallow anterior chamber made visualization challenging and corneal striae developed when indentation was performed in this hypotonous eye.

Topical medical treatment was initiated by reducing the patient’s routine postoperative steroid regimen and adding twice-daily 1% atropine sulfate. Despite this, the anterior chamber had shallowed further at 3 weeks postoperatively with a best-corrected visual acuity (BCVA) of 20/25 and an IOP of 2 mmHg. No hypotony maculopathy was present, and repeat AS-OCT imaging displayed a smaller cleft (35°) and a reduction in maximum effusion height (493 μm) (Fig. 1B). It was therefore decided to continue conservative medical management.

Early superficial macular folds were evident at 5 weeks postoperatively with a worsened BCVA of 20/50 and IOP stable at 2 mmHg. Medical therapy was modified by stopping the topical steroids entirely and the 1% atropine was continued twice a day. 10 weeks postoperatively found a further reduction in BCVA (20/70) and deeper, more significant macular folds as well as optic disc edema, with a slightly improved IOP of 5 mmHg. Given the steadily decreasing BCVA and the worsening hypotony maculopathy, serious consideration was given to escalating therapy to more invasive laser and/or surgical treatment options. However, repeat AS-OCT imaging once again showed anatomical improvement, with the cleft now spanning 14° of arc and the effusion height now measuring 174 μm (Fig. 1C). This objective and quantifiable imaging evidence of anatomical improvement directly informed our decision to continue with conservative management, and saved the patient a more invasive intervention at this point.

A cataract was noted to be forming by this time and significant macular folds remained. Cataract surgery was performed with the goals of improving visual acuity, stimulating an anterior segment inflammatory reaction to promote continued closure of the cyclodialysis cleft, as well as to improve gonioscopic visualization of the angle structures should laser gonioplasty be needed as a subsequent therapeutic modality. 1 week post-phacoemulsification an IOP spike occurred, signifying likely closure of the cleft. Three topical glaucoma medications were temporarily prescribed to mitigate the spike and were gradually discontinued soon after. 6 months following phacoemulsification, AS-OCT displayed complete closure of the cyclodialysis cleft and resolution of the ciliochoroidal effusions (Fig. 1D; see also Table 1). Hypotony maculopathy had resolved, IOP was 11 mmHg on no medications, and uncorrected visual acuity (UCVA) was 20/20.

2.2. Case 2

A 29-year-old man was referred by the retina service for uncontrolled IOP (30 mmHg on 4 agents) in the right eye despite pars plana vitrectomy, membrane peeling and removal of recently injected intravitreal

Fig. 1. A-D, Sequential postoperative anterior segment optical coherence tomography images displaying cyclodialysis cleft and ciliochoroidal effusion progression in Case 1. A, Postoperative day 4; cleft arc of 39° and effusion height of 623 μm. B, Postoperative week 3; cleft arc of 35° and effusion height of 493 μm. C, Postoperative week 10; cleft arc of 14° and effusion height of 174 μm. D, Postoperative month 6; resolution of cleft and effusions.
steroid. He had a history of a traumatic outer retinal disturbance involving the macula, an epiretinal membrane, an injection of triamcinolone acetonide and a subsequent elevation of IOP. The patient then underwent SLT which controlled his IOP for 19 months, at which time the IOP was found to be 22 mmHg on 2 topical agents. Given the development of a visually significant cataract, a combined phacoemulsification and GATT surgery was recommended to the patient.

GATT was complicated intraoperatively by the creation of a nasal cyclodialysis cleft thought to possibly represent the reopening of a previously healed undiagnosed traumatic cleft. Postoperative day 1 IOP was 9 mmHg, and 1% atropine sulfate was started. At 6 days post-operatively, BCVA was 20/60 and IOP 3 mmHg. AS-OCT imaging revealed a nasal cyclodialysis cleft spanning 41° of arc and adjacent ciliochoroidal effusions measuring 555 μm in maximal height (Fig. 2A). Medical management was maintained.

At postoperative week 3, BCVA worsened to 20/200, IOP was measured at 5 mmHg, and chorioretinal folds were apparent on macular OCT. The cyclodialysis cleft was no longer visible on gonioscopy, and AS-OCT showed a shallow cleft reduced in size (16°), but a slight increase in effusion height was noted (600 μm; Fig. 2B). Despite evidence of hypotony maculopathy, objective and quantifiable evidence of anatomical improvement of cleft size on serial imaging supported our decision to continue with conservative management at this time.

At 4 weeks postoperatively, IOP persisted at 20/80, and AS-OCT showed a further decrease in the extent of the cleft (9°) as well as a reduction in the adjacent ciliochoroidal effusion height (516 μm). By 6 weeks postoperatively, BCVA had improved to 20/70 and IOP had risen to 12 mmHg. AS-OCT revealed resolution of the cyclodialysis cleft and further shallowing of the ciliochoroidal effusions with a maximal height of 484 μm (Fig. 2C). These effusions had resolved on imaging 1 week later (Fig. 2D), and topical steroids and atropine were both tapered at this point. Following closure of the cyclodialysis cleft, an IOP spike ensued which was managed with topical medication and occasional temporizing anterior chamber paracentesis. At 2 years post-combined surgery, the patient’s UCVA is 20/25 and IOP is 17 mmHg with no drops (see Table 1).

### Table 1
Clinical progression of cyclodialysis cleft arc & ciliochoroidal effusion height with associated visual acuities and intraocular pressures.

| Case 1 | Case 2 |
|--------|--------|
| **POD 4** | **POD 6** |
| **POW 3** | **POW 4** |
| **POW 10** | **POW 5** |
| **POM 6** | **POW 7** |
| **POY 1** | **POY 2** |
| **Distance Visual Acuity** | **IOP (mmHg)** | **Cleft Arc (°)** | **Effusion Height (μm)** |
| 20/80 | 20/60 | 39 | 623 |
| 20/25 | 20/200 | 35 | 493 |
| 20/70 | 20/80 | 14 | 174 |
| 20/20 | 20/70 | 0 | 0 |
| 20/20 | 20/50 | 0 | 0 |
| **IOP (mmHg)** | **Distance Visual Acuity** | **Cleft Arc (°)** | **Effusion Height (μm)** |
| 4 | 20/80 | 11 | 0 |
| 5 | 20/20 | 5 | 0 |
| 11 | 20/20 | 11 | 555 |
| 11 | 20/20 | 3 | 16 |
| 3 | 20/70 | 5 | 16 |
| 5 | 20/50 | 5 | 9 |
| 12 | 20/25 | 12 | 0 |
| 26 | 20/25 | 17 | 0 |

POD = postoperative day; POW = postoperative week; POM = postoperative month; POY = postoperative year; IOP = intraocular pressure.

3. Discussion

The goal of cyclodialysis cleft treatment is to promote cleft closure and normalize the IOP. Management typically begins with a trial of topical medical therapy using strong cycloplegic agents such as atropine sulfate to relax the ciliary muscle in an attempt to reappose it to the scleral spur. If this fails, laser photocoagulation techniques have been described using the argon, yttrium aluminum garnet, and transcleral diode platforms, as well as an ab-externo cryotherapy approach. These modalities all aim to create a relatively controlled, localized inflammatory reaction to increase tissue adhesion and encourage cleft closure. Success with these nonsurgical techniques is generally seen in clefts < 4 clock hours (<120°) in size, with a variety of incisional surgical approaches being employed in more extensive cases. The increasingly invasive and potentially risky nature of each escalation in this stepwise therapeutic algorithm highlight the value of a non-invasive imaging method of monitoring smaller clefts during conservative therapy. When one also considers AS-OCT’s ability to potentially diagnose clefts that would otherwise not be clinically visible on slit lamp gonioscopy, the value and utility of this modality in cyclodialysis management is highlighted even further.

This case series illustrates these very points. In Case 1, AS-OCT diagnosed a cyclodialysis cleft and thus directed appropriate treatment initiation at a point when gonioscopic visualization was inadequate. In both cases, the objective and quantifiable serial AS-OCT measurements of the cyclodialysis cleft arc and ciliochoroidal effusion height demonstrated clear anatomical improvement and supported a continuation of conservative management at times when worsening hypotony maculopathy, decreasing BCVA, and progressive anterior chamber shallowing...
suggested that treatment escalation might be warranted. Ultimately, AS-OCT saved both of these patients the potential risks and decreased quality of life associated with a more invasive laser or surgical treatment.

A number of studies have reported the use of AS-OCT to aid in the identification and diagnosis of cyclodialysis clefts. Mateo-Montoya and Dreifuss report the use of AS-OCT as an adjunct to diagnosis of a cyclodialysis cleft in a 38 year old patient following blunt trauma. Selvan et al. report the case of a 12 year-old boy who developed a cyclodialysis cleft following penetrating ocular trauma. Gonioscopy revealed a cleft extending only 2 clock hours while AS-OCT revealed 5 clock hours thus expediting surgical repair. Prata et al. report the case of an 8 year-old boy presenting with a cyclodialysis cleft following blunt trauma. Given a limited gonioscopic exam due to poor patient cooperation and corneal folds, AS-OCT and UBM were performed on this patient identifying the location of the cleft as well as 360° of choroidal effusion.

In these studies, AS-OCT is highlighted as a valuable diagnostic tool which can identify and quantify the extent of a cleft potentially under-estimated or missed entirely on gonioscopy. It is shown to be useful in situations where the gonioscopic exam is limited by cooperation or an obscured view. It also offers an advantage over UBM in that it does not require a high level of technical expertise or contact with the ocular surface.

In addition to the diagnostic capabilities of AS-OCT, our cases are the first to discuss the potential role of AS-OCT in monitoring the progression of cyclodialysis clefts during conservative management, directly informing the deferral of more invasive treatment in some cases. The 360° imaging capacity of the CASIA swept source AS-OCT system (Tomey Corporation, Nagoya, Japan) allows for reproducible measurements of the extent of the cyclodialysis cleft arc as well as the maximal effusion height. Further study is needed with a larger number of patients, preferably with clefts arising from a variety of etiologies, before more definitive conclusions can be drawn.

4. Patient consent

Formal written consent to publish these cases was not obtained from the two patients presented in this series. However, this report does not contain any personal information that could be used to identify the patients.

Credit author statement

Thomas Andrew Berk: Investigation, Writing – Original Draft, Visualization. Daniel Perez: Investigation, Writing – Original Draft, Visualization. Alaa Mofti: Investigation, Writing – Review & Editing. Béatrice Des Marchais: Resources, Writing – Review & Editing. Hady Saheb: Conceptualization, Investigation, Resources, Writing – Review & Editing, Supervision, Project Administration.

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Declaration of competing interest

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References

1. Agrawal P, Shah P. Long-term outcomes following the surgical repair of traumatic cyclodialysis clefts. Eye. 2013;27:1147–1152.
2. Ioannidis AS, Barton K. Cyclodialysis cleft: causes and repair. Curr Opin Ophthalmol. 2010;21:150–154.
3. Mateo-Montoya A, Dreifuss S. Anterior segment optical coherence tomography as a diagnostic tool for cyclodialysis clefts. Arch Ophthalmol. 2009;127:109–110.
4. González-Martín-Moro J, Contreras-Martín I, Muñoz-Negrete FJ, Gómez-Sanz F, Zarallo-Gallardo J. Cyclodialysis: an update. Int Ophthalmol. 2016;37:441–457.
5. Selvan H, Yadav S, Gupta V, Gupta S. Case report: cyclodialysis cleft in a case of open-globe injury and role of swept-source anterior segment optical coherence tomography in diagnosis. Optom Vis Sci. 2020;97:395–399.
6. Prata TS, Palmiero PM, De Moraes CGV, et al. Imaging of a traumatic cyclodialysis cleft in a child using slit-lamp-adapted optical coherence tomography. Eye. 2009;23:1618–1619.
7. Hwang JM, Ahn K, Kim C, Park KA, Kee C. Ultrasonic biomicroscopic evaluation of cyclodialysis before and after direct cycloexy. Arch Ophthalmol. 2008;126:1222–1225.
8. Küchle M, Naumann GO. Direct cycloexy for traumatic cyclodialysis with persisting hypotony. Report in 29 consecutive patients. Ophthalmology. 1995;102:322–333.
9. Berk TA, An JA, Ahmed IIK. Inadvertent cyclodialysis cleft and hypotony following ab-interno trabeculotomy using the Trabectome device requiring surgical repair. J Glaucoma. 2017;26:742–746.
10. Ormerod LD, Baerveldt G, Sunalp MA, Riekhof FT. Management of the hypotonic cyclodialysis cleft. Ophthalmology. 1995;98:1384–1393.
11. Harbin Jr TS. Treatment of cyclodialysis clefts with argon laser photocoeagulation. Ophthalmology. 1982;89:1082–1085.
12. Brooks AM, Troski M, Gilles WE. Noninvasive closure of a persistent cyclodialysis cleft. Ophthalmology. 1996;103:1943–1945.
13. Amini H, Razeghinejad MR. Trans scleral diode laser therapy for cyclodialysis cleft induced hypotony. Clin Exp Ophthalmol. 2005;33:348–350.
14. Krohn J. Cryotherapy in the treatment of cyclodialysis cleft induced hypotony. Acta Ophthalmol Scand. 1997;75:96–98.