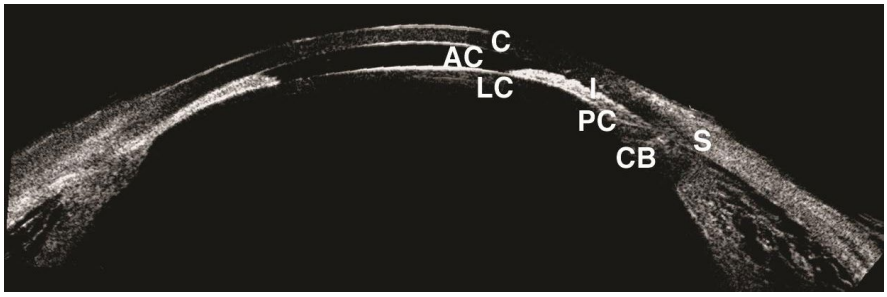
Overview of Post-Op Aqueous Misdirection



EVIDENCE ON UBM. *In this eye, the lens-iris diaphragm is pushed forward, causing a uniform shallowing of the anterior chamber (AC). The central portion of the anterior lens capsule (LC) is nearly in contact with the cornea (C). Note ciliary body (CB) detachment, which is commonly seen in malignant glaucoma. I = iris; PC = posterior chamber; S = sclera.*

Courtesy of Robert Ritch, MD

Aqueous misdirection (AM) is a rare and challenging form of secondary angle closure characterized by uniform shallowing of the anterior chamber in the absence of pupillary block, frequently with elevated IOP.[1] AM is also known as malignant glaucoma and has been known by a variety of names—including ciliary block, ciliolenticular block, and posterior aqueous diversion syndrome. The condition was originally described by von Graefe in 1869 as a malignant process owing to its aggressive course and poor response to medical therapy.

The pathogenesis of AM remains incompletely understood, and consensus on its treatment and nomenclature has not yet been achieved. While AM can present both intraoperatively (e.g., intraoperative rock-hard eye syndrome[1]) and postoperatively, this review will focus on recognizing and treating AM in the postoperative setting.

Prevalence and Risk Factors

Incidence. Given its relative rarity, the true incidence of AM is difficult to study. Published research estimates an incidence ranging from 0.06% to 2% in postprocedural contexts, but its incidence is likely underreported.[2]

Surgical risk factors. AM typically presents in the early postoperative period following incisional glaucoma surgery. It is traditionally most common after trabeculectomy or combined trabeculectomy and cataract surgery (collectively accounting for upward of two-thirds of reported cases[3]) but has also been reported after bleb needling/revision, cataract surgery, penetrating keratoplasty, and laser iridotomy.[2,3]

Anatomic risk factors. A number of predisposing anatomic risk factors for AM have been suggested, including short axial length, nanophthalmos, plateau iris configuration, anatomically narrow angles, and primary angle closure (PAC) and primary angle-closure glaucoma (PACG).[2,3] Other proposed risk factors include pseudoexfoliation and female sex.[3,4]

Although the majority of documented cases of AM have been in patients with short axial lengths or a diagnosis of PAC or PACG, it is important to note that eyes of any dimension can develop AM postoperatively.[5] Furthermore, AM is not necessarily limited to the immediate postoperative period and may occur after laser suture lysis or following discontinuation of postoperative topical medications.[5]

Pathophysiology

Although the precise pathogenesis is still a matter of debate, AM results from a fundamentally abnormal anatomic relationship between the ciliary body, lens, and anterior vitreous; this results in anterior displacement of the lens-iris diaphragm.

AM was originally described as a malignant process, wherein the pressure in the posterior chamber increases and aqueous outflow is disrupted in a self-amplifying cycle. One of the original theories, first described by Shaffer, proposed a valve-like mechanism in which aqueous tracks posteriorly behind a vitreous detachment or accumu-

BY KIAN M. MADJEDI, MD, ANDREW C. CRICHTON, MD, ANTHONY P. KHAWAJA, PHD, FRCOPHTH, AND PAUL J. FOSTER, PHD, FRCOPHTH.
EDITED BY JEREMIAH P. TAO, MD, FACS.

lates in Berger space or pockets in the vitreous. Thus, this theory holds that this misdirected aqueous pushes the vitreous forward and anteriorly displaces the ciliary body, lens, and iris.[4]

Other more recent theories challenge the existence of a valve-based mechanism in the eye and suggest that the mechanism is related to choroidal expansion (and not true misdirection of aqueous). Breakdown of the blood-aqueous barrier in the intra- or postoperative setting (e.g., overfiltration and hypotony) leads to loss of normal osmotic fluid forces in the choroidal vessels, and it can result in choroidal volume expansion. It has been proposed that this leads to an increase in the absolute pressure of each compartment and a compensatory increase in aqueous outflow moving along this pressure gradient.

The resulting creation of a pathologic posteroanterior pressure gradient leads to more aqueous outflow and fluid from the vitreous moving more anteriorly, thus leading to further anterior displacement of the lens-iris diaphragm.[6] This process can also occur in aphakic eyes, with the anterior hyaloid exerting posterior pressure, thus causing the ciliary body and iris to move forward. Of note, this hypothesis would suggest that, as a term, aqueous misdirection is a misnomer, as the proposed pathophysiology may not in fact involve any true misdirection of aqueous.

Clinical Features and Presentation

AM is largely a diagnosis of exclusion. Conditions that must be considered when evaluating a patient with possible AM include choroidal hemorrhage or effusion, pupillary block, and other secondary angle-closure conditions.

Differential diagnosis. A general approach to differential diagnosis first considers whether the AC is shallow or deep, whether there has been recent filtering surgery, and whether the IOP is high or low. The differential diagnosis of shallow AC with high IOP would include pupillary block, suprachoroidal hemorrhage, and aqueous misdirection. The diagnosis of AM requires, at minimum, axial shallowing of the AC, a patent iridectomy/iridotomy, if one is present, and the absence of suprachoroidal fluid or blood.[5]

The configuration of the peripheral iris can be helpful in differentiating AM from other forms of angle closure: in pupillary block, the peripheral iris will be bowed while the central AC is normal, while AM often presents with a more uniformly shallowed AC. If there is an existing iridotomy, its patency must be assessed, especially as ciliary processes or vitreous can block a previously patent iridotomy.

In general, AM tends to be progressive and often refractory to initial medical management in comparison to the other conditions on the differential diagnosis.

Presentation. Clinical symptoms are nonspecific and can include pain, conjunctival injection, corneal edema, and myopic shift.[7] The physical examination may show corneal edema, unilateral, uniform shallowing/flattening of both the peripheral and central AC, and anterior displacement of the lens-iris diaphragm. IOP is typically elevated, but in the presence of a functional trabeculectomy or tube shunt, it may be normal or low.

In some cases, ciliary body processes in apposition to the crystalline lens may be visible through a prior iridotomy. In posttrabeculectomy patients, it is important to differentiate between AM and a shallow chamber caused by an overfiltering bleb or wound leak—even while recognizing that postoperative hypotony may be an inciting event for the pathogenesis of AM.

Diagnostic Testing

Indirect ophthalmoscopy should be performed to exclude choroidal detachment and hemorrhage. In the absence of clear media, a B-scan should be performed to exclude these pathologies. Ultrasound biomicroscopy (UBM) and OCT may also be used to assess and quantitatively measure anterior segment structures, and they can be particularly helpful in assessing the patency of an iridotomy.

Features of AM on UBM include anterior rotation of the ciliary body, anteriorization of the lens-iris diaphragm, and iridocorneal contact. Inability to locate the posterior chamber on UBM has been suggested as a key imaging feature of AM.[2]

Treatment

Although good evidence on management of AM is lacking, the mainstay of treatment involves IOP reduction through medical or surgical means.

Medical approaches. Medical treatment with cycloplegic agents, aqueous suppressants, and/or hyperosmotics in patients without contraindications (and depending on institutional preference) should be promptly employed.

Cycloplegic agents may help improve aqueous flow, increasing zonular tension and helping pull the lens-iris diaphragm more posteriorly. Miotics are generally contraindicated as they can further anteriorize the lens-iris diaphragm and worsen the condition.[4] Hyperosmotic agents such as mannitol can reduce the volume of the vitreous by drawing water out through the choroidal vessels, thereby reducing posterior pressure; however, these agents can have systemic contraindications and should be used cautiously.

Surgical approaches. In many cases medical treatment alone is insufficient, and surgical and/or laser interventions are required.

A peripheral Nd:YAG laser may be performed in pseudophakic eyes to create an anterior hyaloidotomy to relieve the pressure gradient between the chambers.[4] Laser cyclophotocoagulation (CPC) of the ciliary body with argon laser and transscleral diode laser CPC also have been used to facilitate posterior rotation of the ciliary body and to decrease cilio-hyaloidal apposition, although these are not commonly employed.[8]

The mainstay of surgical intervention is pars plana vitrectomy (PPV) with or without iridozonulohyaloidectomy (IZH). This latter procedure purposefully disrupts the anterior hyaloid face and creates a unicameral eye.[9] Vitrectomy and a patent iridectomy should theoretically remedy the underlying pathogenesis of AM, but there have been reported instances of recurrence following PPV plus IZH,[10] which sug-

gests that much remains to be understood about this complex condition.

Conclusion

Having a low threshold of suspicion for AM—particularly in postoperative contexts with an elevated IOP and uniformly shallow eye with preexisting risk factors—should help alert the clinician to prompt recognition and treatment of this complex condition.

1 Lau OC et al. *J Cataract Refract Surg.* 2014; 40(5):799-804.
2 Kaplowitz K et al. *Surv Ophthalmol.* 2015;60(3): 229-241.
3 Halenda KM et al. *Curr Ophthalmol Rep.* 2019; 7(2):150-159.
4 Fischer NA et al. *Curr Ophthalmol Rep.* 2020; 8:51-61.
5 Foreman-Larkin J et al. *J Ophthalmol.* Published online Nov. 23, 2025. Article ID 283707.
6 Quigley HA et al. *J Glaucoma.* 2003;12(2):167-180.
7 Moinul P et al. *Can J Ophth.* 2017;52(4):e146-e180.
8 Stumpf TH et al. *Ophthalmology.* 2008;115(11): 2058-2061.
9 Al Bin Ali GY et al. *Retina.* 2017;37(10):1916-1922.
10 Francis BA et al. *Ophthalmology.* 2000;107(7): 1220-1221.

Dr. Madjedi is an ophthalmology resident at the University of Calgary in Alberta, Canada; **Dr. Crichton** is Clinical Professor at the University of Calgary; **Dr. Khawaja** is Associate Professor at the UCL Institute of Ophthalmology in London; and **Dr. Foster** is Professor of Ophthalmic Epidemiology and Glaucoma Studies at the UCL Institute of Ophthalmology. *Relevant financial disclosures: None.*

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