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Considerations for Adding Minimally/Microinvasive Glaucoma Surgery (MIGS) to a Planned Cataract Surgery

Pushpinder Kanda, MD, PhD
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Introduction

Glaucoma is a progressive optic neuropathy defined by retinal ganglion cells loss and characteristic visual field loss. It is a leading cause of irreversible blindness and affects over 60 million people worldwide.[1] Its prevalence is estimated to increase to 111.8 million by 2040.[1] Intraocular pressure (IOP) is a major clinically modifiable risk factor for glaucoma. Thus, glaucoma therapy aims to reduce the IOP using medications, lasers (e.g., selective laser trabeculoplasty) or surgery. Historically, surgery has been reserved for advanced glaucoma and in cases with poorly controlled pressure despite medical and laser treatment. For decades, trabeculectomy and tube shunt devices have been the predominant surgical methods for lowering ocular pressure.[2] However, these traditional surgeries are invasive requiring significant manipulation of ocular tissue and have significant post-operative complication rates.[3] Many patients have fallen in the gap of needing more pressure lowering but not enough to justify a higher risk surgery. Fortunately, the landscape of glaucoma surgery has rapidly evolved over the past 20 years with the emergence of minimally/micro-invasive glaucoma surgery (MIGS).

MIGS is often performed as an adjunct to cataract surgery. As such, there is minimal added long-term risk if the procedure is done in the same space as the already planned cataract surgery. This represents a large group of patients, some of whom would not have been considered as glaucoma surgical candidates in the past. The clinician is now faced with the question, "Should I add MIGS to the cataract surgery?" In this paper, we suggest a series of questions to ask about each case in order to help make a patient-centred decision.

Minimally/Microinvasive Glaucoma Surgery (MIGS)

MIGS is defined as any device or a procedure with the following characteristics[4]: **1)** Good safety profile compared to traditional surgeries (e.g., fewer complications of hypotony, choroidal hemorrhage, or choroidal effusion); **2)** Less invasive with minimal trauma to ocular tissue; **3)** Typically performed as an internal approach through a small corneal incision (also called ab interno approach); **4)** Shorter operating time; **5)** Quicker post-operative recovery period and; **6)** Moderate IOP lowering effects (at least a 20% IOP reduction).

MIGS is typically indicated for patients with mild-to-moderate open angle glaucoma (OAG) who have failed IOP control despite medical management or laser treatment. In addition, it can be considered in patients with medication noncompliance, intolerance due to side effects, or a desire to decrease the number of medications used. It has also shown utility in secondary glaucoma (e.g., pseudoexfoliation or pigment dispersion glaucoma).[5,6]

MIGS is classified into five broad categories based on the anatomical target site (**Figure 1**)[2]: **1)** Trabecular meshwork bypass, in which aqueous humor is provided direct access to the Schlemm's canal using a stent or excising the trabecular meshwork tissue; **2)** Canaloplasty, where Schlemm's canal and collector channels are dilated to enhance outflow; **3)** Enhancing outflow through the uveoscleral pathway by placing a bypass device into the suprachoroidal space; **4)** Schlemm's canal bypass, where a filtering device directs outflow to the subconjunctival/sub-tenon space and forms

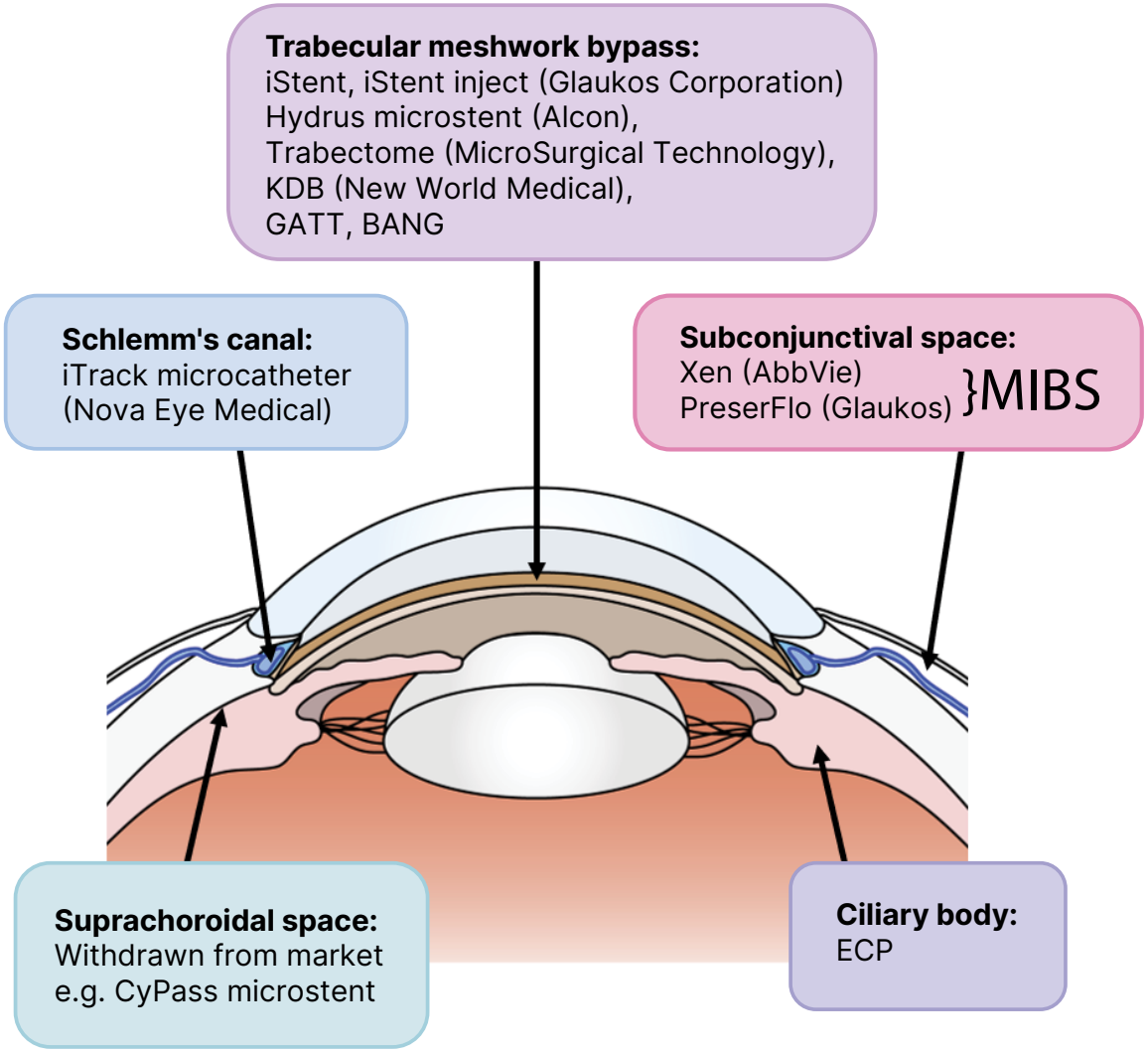

Figure 1. MIGS classification by anatomy; *courtesy of Pushpinder Kanda, MD, PhD and Garfield Miller, MD*.

Abbreviations: BANG: Bent ab interno needle goniectomy, **ECP:** Endoscopic cyclophotocoagulation, **GATT:** Gonioscopy-assisted transluminal trabeculotomy, **KDB:** Kahook dual blade, **MIBS:** minimally invasive bleb surgery

a bleb; **5)** Decreasing aqueous production by causing destruction of ciliary body epithelium.

Some authors highlight that the newer Schlemm's canal bypass surgeries (e.g., XEN gel stent or PreserFlo microshunt) do not strictly meet the criteria of MIGS due to the formation of a filtering bleb. Instead, the term minimally invasive bleb surgery (MIBS) has been adopted because it is less invasive and safer than traditional filtering surgery. Unlike MIGS, MIBS is more often used to treat moderate-to-advanced glaucoma as it can achieve low-teen to single-digit pressure. MIBS and suprachoroidal microstents are not covered in this review; however, coverage of them can be found elsewhere in detail.[2,7] Currently, there are no suprachoroidal microstents available in the North American market outside of investigational use. Last, endoscopic cyclophotocoagulation (ECP) may not always be categorized as conventional MIGS due to increased risk of complications such as inflammation and cystoid macular edema.[8,9]

Combined Cataract and MIGS Surgery (cMIGS)

MIGS can be performed alone or in combination with cataract surgery (cMIGS) for OAG.[10] Several studies have shown that cMIGS can improve IOP control and decrease the burden of glaucoma medications when compared to standalone surgery.[10-13] For example, the two-year pivotal iStent trial showed a 20% reduction in diurnal IOP from baseline in greater number of eyes treated with combined iStent in cataract surgery vs cataract surgery alone (75.8% of eyes for the combined group vs 61.9% for cataract surgery only, P=0.005).[14] Among the IOP responders, 84% of eyes with combined treatment and 67% with cataract surgery alone did not require glaucoma medication at 23 months. **Table 1** summarizes the results of various studies combining MIGS with cataract surgery for open angle or secondary glaucoma.

While lowering IOP and decreasing medication are desirable outcomes, ultimately preservation of vision is most important. Studies have shown that cMIGS can help decrease the need for incisional glaucoma filtering surgeries.[13] For example, the HORIZON trial showed that Hydrus® microstent combined with cataract surgery had less risk for needing major incisional glaucoma surgery at 5 years compared to cataract surgery alone (2.4% risk for combined surgery vs 6.2% for cataract alone, P=0.027).[13] In addition, this was one of the first studies to show that combined surgery resulted in slower progression of visual field loss (progression rate of -0.26dB/year [95% CI: -0.36 to -0.16] for the combined group vs to -0.49 dB/year [95% CI: -0.63 to -0.34] for cataract surgery alone, P=0.0138).[15]

An important consideration for most cMIGS is that studies have not shown an increase in serious complication rate compared to cataract surgery alone.[12,16] This makes cMIGS an attractive option for many glaucoma patients already being considered for cataract surgery.

Questions to Ask of Each Case

Is the glaucoma controlled?

Glaucoma is typically a chronic, slowly progressive disease. In the past, absolute IOP values were relied upon to decide if glaucoma was adequately treated. We now know that there is no universal pressure at which glaucoma can be considered controlled. Setting targets, then monitoring over time, typically with perimetry and structural analysis, is required to ensure stability.

In cataract cases where the glaucoma is early and controlled, cMIGS may not be required. In the early post-operative period following cataract surgery, IOPs can be variable with higher incidence of spikes in glaucoma patients.[30] Following the post-operative period, however, IOP generally tends to decrease slightly in OAG.[12,31] Craven RE *et al.* showed that patients with mild-to-moderate controlled OAG who underwent cataract surgery had little change in the visual fields despite the small increase in IOP after 2 years.[16] Thus, stable glaucoma patients who can tolerate some early lability in IOP, may opt for cataract surgery alone.

If glaucoma is uncontrolled, even in early glaucoma, MIGS should be considered in patients requiring cataract surgery. Iancu R *et al.* showed that cataract surgery alone decreased the IOP by 1.9±3.9 mmHg at 12 months in patients with uncontrolled primary open-angle glaucoma (POAG); however, 84.2% eventually needed glaucoma surgery by 11.6±4.18 months.[32]

The target IOP and the anticipated degree of IOP reduction are important factors to consider when choosing cMIGS. In cases where the cMIGS will likely place the patient within the target range, it is an excellent option, while minimizing risk. In cases where cMIGS is not possible or unlikely to be adequate for reaching target pressure, MIBS or traditional filtering surgeries should be considered.

Is the current treatment sustainable?

Even if glaucoma is controlled, the surgeon should consider whether or not the current therapy is sustainable. Adherence to medical therapy in glaucoma is notoriously poor with some studies reporting up to 30% non-adherence.[33] Barriers to adherence include cost, use of multiple drops, forgetfulness, psychiatric disorders (e.g,. depression), ocular side effects, and difficulty administering drops.[33] Medical or other life crises can result in prolonged periods with inconsistent medication use. Thus, reducing medication dependency with cMIGS may improve quality of life.[34]

It is important to consider that poor medication adherence leads to greater IOP fluctuations. Large diurnal fluctuation of IOP is an independent risk factor for glaucoma progression.[35] Therefore, these patients would benefit from cMIGS which has shown to provide more stable IOP reduction.[36]

Author (Year of Publication)	Type of Study	Type of Glaucoma	Type of Intervention	Main Outcomes
Samuelson TW *et al.* (2011)[12]	Prospective randomized controlled trial	Mild-to-moderate OAG with IOP ≤24 mmHg controlled on 1 to 3 medications	Treated group= iStent + cataract (n=111 eyes) Control group= cataract surgery alone (n=122 eyes)	• At 1 year, 72% of treated eyes achieved unmedicated IOP ≤21 mmHg vs 50% of control eyes (P<0.001) • 66% of treated eyes achieved 20% IOP reduction without medications vs 48% of control eyes (P<0.003) • Decrease in glaucoma medication was greater in treated group vs control (1.4±0.8 vs 1.0±0.8; P<0.005)
Craven RE *et al.* (2012)[16]	Prospective randomized controlled trial	Mild-to-moderate OAG with unmedicated IOP of ≥22 mmHg and ≤36 mmHg	Treated group= iStent + cataract (n=117 eyes) Control group= cataract surgery alone (n=123 eyes)	• At 2 years, 61% of treated eyes achieved unmedicated IOP ≤21 mmHg vs 50% of control eyes (P<0.036) • Trend in favour of treated group for achieving 20% IOP reduction without medications vs 44% of control eyes (53% treated vs 44% control eyes, P<0.09)
Wang SY *et al.* (2019)[6]	Retrospective, observational longitudinal cohort study	POAG (78.4%), Narrow angles (12.8%), Secondary OAG which included pigmentary glaucoma and PXG (8.8%)	Treated group= iStent + Cataract surgery (n=2971 subjects) Control group= Cataract surgery alone (n=1486 subjects)	• Treated group had a greater reduction in glaucoma drops (0.99 in treated vs 0.49 in control at month 20–24; P < 0.001) • Treated group had higher proportion receiving no glaucoma drops after surgery (73.5% in treated vs 55.3% in control at month 20-24; P <0.001)
Hengerer FH. *et al.* (2022)[17]	Prospective, longitudinal case series	POAG (74%), PXG (19%), Combined mechanism (5%), Pigmentary glaucoma (1%), NVG (1%)	iStent inject alone (n=44) iStent inject + cataract surgery (n=81)	• At 5 years, combined surgery reduced the mean IOP by 39% (22.6 to 13.8 mmHg, P<0.001) and medications by 69% (2.52 to 0.78, P<0.001) • Standalone surgery reduced the mean IOP by 42% (25.3 to 14.6 mmHg, P<0.001) and medications by 75% (2.98 to 0.74, P<0.001) • 83% of in the overall cohort achieved ≥20% IOP reduction

Author (Year of Publication)	Type of Study	Type of Glaucoma	Type of Intervention	Main Outcomes
Samuelson TW. *et al.* (2019)[14]	Prospective, randomized controlled trial	Mild-to-moderate POAG with IOP ≤24 mmHg on 1 to 3 medication, unmedicated diurnal IOP 21–36 mmHg	Treated group= iStent inject + cataract surgery (n=387 eyes) Control= Cataract surgery alone (n=118 eyes)	• At 24 months, the treated group had greater proportion of eyes which had ≥20% reduction in unmedicated diurnal IOP from baseline compared to control group (75.8% treated vs 61.9% control, P=0.005) • The mean reduction in unmedicated diurnal IOP from baseline was greater in treated eyes (7.0±4.0 mmHg treated vs 5.4±3.7 mmHg control; P<0.001)
Ahmed IIK *et al.* (2022)[13]	Prospective, randomized controlled trial	Mild-to-moderate POAG with washed-out diurnal IOP of 22–34 mmHg	Treated group= Hydrus microstent + cataract surgery (n=369 eyes) Control= Cataract surgery alone (n=187 eyes)	• At 5 years, the treated group had higher proportion of eyes with IOP ≤18 mmHg without medications (49.5% treated vs 33.8% control, P=0.003) • Greater proportion of treated eyes had ≥20% IOP reduction (54.2% treated vs 32.8% control, P<0.001) • Greater reduction in medications in treated group (0.5±0.9 treated vs 0.9±0.9 control, P<0.001) • Greater proportion of treated eyes were drop free (66% treated vs 46% control, p<0.001) • Cumulative risk of needing incisional glaucoma surgery was lower in treated group (2.4% treated vs 6.2% control, P=0.027)
Esfandiari H. *et al.* (2019)[5]	Retrospective case series	POAG (62.3%), PXG (14.8%), Pigmentary glaucoma (6.6%), PACG (8.2%), Others secondary cause (8.2%) Glaucoma was mild (34.4%), moderate (26.3%) and severe (39.3%)	Trabectome + cataract surgery (n=61 eyes)	• Success was defined as IOP >5mmHg and ≤ 21 mmHg, ≥ 20% IOP reduction from baseline at two consecutive visits, no need for further glaucoma surgery, and no loss of light perception. • At 5 years, the cumulative success was 67.5%. • IOP decreased from 20±5.6 mmHg at baseline to 15.6±4.6 mmHg (P=0.001) • Trend toward decreasing glaucoma medication compared to baseline (1.8±1.2 at baseline and 1.0±1.2 at 5 years) • Risk factors for failure were lower baseline IOP, younger age, and higher central corneal thickness • Exfoliative glaucoma was associated with higher success rate.

Author (Year of Publication)	Type of Study	Type of Glaucoma	Type of Intervention	Main Outcomes
Tojo N. *et al.* (2020)[18]	Observational, retrospective study	Subjects with low (<18 mmHg), moderate (18–26 mmHg) or high (>26 mmHg) IOP glaucoma Glaucoma included POAG, PXG, PACG, other secondary glaucoma	A total of 204 eyes had trabectome surgery of which n=105 had simultaneous trabectome + cataract surgery	• At 2 years, trabectome surgeries decreased IOP from baseline (23.0±7.2 mmHg baseline to 13.6±3.6 mmHg at 2 years, and 13.2±4.0 mmHg at 5 years) • Thin central corneal thickness and simultaneous cataract surgery were associated with better surgical outcomes with cutoff IOP ≤21 mmHg and ≤15 mmHg
Kuerten D. *et al.* (2023)[19]	Prospective case series	POAG, NTG	KDB + cataract surgery (n= 55 eyes with POAG and n= 14 eyes with NTG)	• At 12 months, IOP was lowered from 19.7±4.7 mmHg at baseline to 16.1±3.2 (P<0.05) in POAG • At 12 months, there was a trend towards reduction of IOP in NTG group (15.1±2.5 mmHg baseline to 13.6±1.8 mmHg, P>0.08) • 64% of all subjects achieved IOP <21 mmHg without need for glaucoma drops
Dorairaj SK. *et al.* (2018)[20]	Prospective case series	POAG (84.6%), Pigmentary glaucoma (7.7%), NTG (3.9%)	KDB + cataract surgery (n= 52 eyes)	• At 12 months, the mean IOP was reduced from 16.8±0.6 mmHg at to 12.4±0.3 mmHg (P<0.001) • A 50% reduction of glaucoma medication was achieved after surgery (1.6±0.2 baseline vs 0.8±0.1 at 12 months, P<0.05) • ≥57.7% of eyes had ≥20% IOP reduction form baseline
Ventura-Abreu N. *et al.* (2021)[21]	Randomized controlled trial	Mild-to-moderate OAG, OHT	Treatment= KDB + cataract (n= 21 eyes) Control= cataract surgery alone (n= 21 eyes)	• At 12-months, there was no significant difference in the reduction of IOP and glaucoma medications between groups. • Both groups showed similar safety profile
DeVience E. *et al.* (2024)[22]	Retrospective case control series	POAG, OHT	Treatment = needle goniotomy + cataract surgery (n=46 eyes) Control= cataract surgery alone (n=115 eyes)	• At 6 months, treated group showed a 28% reduction of IOP (-6.3±6.5 mmHg) compared to 1% IOP reduction (-0.3±2.9 mmHg) for control group (P<0.05). • 23.1% of control group showed an incidence of early IOP spike vs 6.0% of treated group (Odds Ratio=4.5, P<0.05)

Author (Year of Publication)	Type of Study	Type of Glaucoma	Type of Intervention	Main Outcomes
Eslami Y. *et al.* (2022)[23]	Case series	POAG, PXG, OHT	Needle goniotomy + cataract surgery (n=32 eyes)	• At 6 months, there was a 32.1% IOP reduction (21.8±4.6 mmHg at baseline to 14.8±3.9 mmHg after treatment, P<0.001) • There was a 50.0% reduction in medications (1.2±1.5 at baseline to 0.6±1.1 after treatment, P<0.048)
Wan Y. *et al.* (2022)[24]	Consecutive case series	POAG	Microcatheter-assisted GATT only (n=66 eyes) GATT + cataract surgery (n=58 eyes)	• At 24 months, IOP was reduced from 26.40±6.37 mmHg at baseline to 16.08±2.38 mmHg with combined surgery. • Medication was reduced from 3.12±0.80 to 0.45±0.96 with combined surgery • There was no significant difference between combined surgery and GATT only group for IOP and medication reduction. • The incidence of hyphema did not differ between the two groups • Combined surgery had less post-operative IOP spikes (17.2% eyes) vs GATT only group (54.5% eyes), P<0.0001) at 24 months
Williamson BK. *et al.* (2023)[25]	Retrospective, stratified observational study	Mild-to-moderate POAG, PXG, Pigment dispersion glaucoma	Both Group 1 (>18mmgHg) and Group 2 (≤ 18mmHg) had subjects which underwent: Canaloplasty/ trabeculotomy (Omni system) + cataract surgery Canaloplasty/ trabeculotomy (Omni system) surgery alone	• At 2 years, both combined surgery and standalone surgery decreased the IOP and reduced the medication from baseline. • 75% of all subjects had ≥20% IOP reduction, or between 6-18 mmHg, and nor increase in secondary surgical intervention
Greenwood MD. *et al.* (2023)[26]	Prospective, single-arm, intervention study	Mild-to-moderate OAG with IOP ≤33 mmHg, on 1 to 4 medications, and unmedicated post-washout diurnal IOP ≥21 mmHg and ≤36 mmHg POAG (96%), PXG (4%)	Canaloplasty/ trabeculotomy (OMNI system) + cataract surgery (n= 66 subjects)	• At 36 months, subjects experienced a mean reduction of IOP by 6.9±3.4 mmHg (P<0.00001 vs baseline) • 78% of eyes had ≥20% IOP reduction. • Treatment reduced glaucoma medications from 1.7 at baseline to 0.3 at 36 months (P<0.00001 vs baseline). • About 74% of subjects were medication free at 36 months

Author (Year of Publication)	Type of Study	Type of Glaucoma	Type of Intervention	Main Outcomes
Gallardo MJ. *et al.* (2018)[27]	Retrospective, comparative case series	POAG which was mild (37.3%), moderate (16.0%) or, severe (38.7%)	ABiC alone (iTrack surgical system) (n=41 eyes) ABiC + cataract surgery (n=34 eyes)	• At 12 months, combined treatment reduced IOP from 19.4±3.7 mmHg at baseline to 13.0±1.8 mmHg (P<0.001) and medications from 2.6±1.0 at baseline to 0.8±0.2 (P<0.001). • At 12 months, standalone treatment reduced IOP from 21.2±5.3 mmHg at baseline to 13.7±1.9 mmHg (P=0.001) and medications from 3.0±0.7 at baseline to 1.3±1.1 (P<0.001). • 40% of eyes were medication free
Koerber N. *et al.* (2024)[28]	Retrospective consecutive case series	POAG, PXG	ABiC (iTrack surgical system) alone (n=4 eyes) ABiC + cataract surgery (n=23 eyes)	• At 6 years, ABiC (standalone or combined surgery) reduced IOP from 19.9±5.2 mmHg at baseline to 14.6±3.3 (P<0.001) and medications from 1.9±1 at baseline to 0.9±0.9 (P=0.005) • There was statistical different between standalone or combined group and between different types of glaucoma.
Yap TE. *et al.* (2022)[9]	Retrospective case series	POAG	ECP + cataract surgery (n=83 eyes)	• At 3 years, surgery reduced IOP (18.4±5.2 at baseline to 13.6±3.7 mmHg, P<-0.0001) and medications (2.7±0.9 at baseline to 1.8±1.3, P<0.0001). • At 3 years, 45% of patients did not achieve failure (defined as one or more of **(1)** IOP >21 mmHg or <20% reduction from baseline at two consecutive visits, **(2)** IOP < 5 mmHg at any visit or, **(3)** needing further IOP lowering surgery)
Smith M. *et al.* (2018)[29]	Retrospective case series	Uncontrolled glaucoma POAG/NTG (85%), PXG (8%), PACG (7%)	ECP + cataract surgery (n= 84 eyes)	• At 3 years, surgery reduced IOP (18.7 at baseline to 14.0 mmHg). • The number of medications pre- and post-surgery was similar. • The failure rate at 3 years was 58.3% (defined as 1 of 2 criteria **(1)** IOP >21 or <6 mmHg, or not reduced by 20% from baseline, **(2)** further need for laser or surgery at any timepoint

Table 1. Summary of studies combining MIGS with cataract surgery; *courtesy of Pushpinder Kanda, MD, PhD and Garfield Miller, MD.*

Abbreviations: ABiC: Ab interno canaloplasty, **ECP:** Endoscopic cyclophotocoagulation, **GATT:** Gonioscopy-assisted transluminal trabeculotomy, **KDB:** Kahook dual blade, **NTG:** Normal tension glaucoma, **OAG:** Open angle glaucoma, **OHT:** Ocular hypertension, **IOP:** Intraocular pressure, **PACG:** Primary angle closure glaucoma, **POAG:** Primary open angle glaucoma, **PXG:** Pseudoexfoliation glaucoma

Chronic ocular surface inflammation, allergic reactions and systemic side effects can all impact the long-term viability of topical glaucoma treatment. Studies have shown that preservatives in most topical therapies can lead to morbidity due to dry eye and increase the risk of failure for any future filtering glaucoma surgeries.[37,38] Medications such as brimonidine have shown high rates of allergic reaction even years after uneventful use.[39] Overall, decreasing glaucoma medication will benefit patients who might require traditional filtering surgery or MIBS in the future.

Last, progression of glaucoma is associated with both increased treatment cost and worsening quality of life. Thus, decreasing the pharmacotherapy with MIGS can not only improve quality of life but also lead to cost-saving. A cost-effective analysis by Sood S *et al.* showed that iStent and Hydrus microstent combined with cataract surgery were more cost effective and accumulated higher quality-adjusted life year compared to cataract surgery alone.[40]

Overall, cMIGS represents an opportunity to decrease topical drops in stable patients already planning cataract surgery.

Is the angle open or closed?

Multiple studies have shown that standalone cataracts can lower IOP in patients with OAG, leading some to question whether or not cMIGS is necessary at all.[12,16] Although IOP reduction has been shown with cataract surgery alone, visual field stabilization has not been demonstrated in OAG.[41] One study using the Corvis ST tonometer showed that the cornea biomechanics change following cataract surgery.[42] It was suggested that a component of the measured IOP lowering seen postoperatively may be due to biomechanical changes as opposed to a true lowering of IOP. Ultimately, the goal of glaucoma treatment is preventing progression and cMIGS has been shown not only to lower pressure, but also help in the stabilization of the disease.[15]

In contrast, cataract surgery alone in angle closure glaucoma (ACG) has demonstrated both IOP reduction and visual field stabilization.[41] In cases where there is peripheral anterior synechiae (PAS) and ACG, goniosynechialysis can result in additional lowering of pressure.[43] However, the likelihood of significant IOP lowering may decrease with the chronicity of PAS.[44] Furthermore, patients with chronic ACG may also have underlying trabecular meshwork (TM) dysfunction.[45] In these cases, removal of anatomical closure with cataract surgery alone may only provide partial benefit since TM-dysfunction still needs to be addressed. In these cases, cMIGS has shown to benefit patients.[45] Various studies have shown cMIGS can significantly decrease IOP in ACG.[46,47] For example, a case series by Hernstadt DJ *et al.* showed that combined iStent with cataract surgery in primary angle closure or primary angle closure glaucoma (PACG) was effective in lowering IOP and reducing the number of glaucoma medications in 89.2% of the eyes at 1 year.[46]

In addition, narrow angles are not synonymous with angle closure. For example, a 2018 study by Xu BY *et al.* used anterior segment OCT to demonstrate that only patients with angle narrowing below a certain anatomical threshold had an association with increased IOP.[48] Similarly, Porporato N *et al.* showed that only patients with iridocorneal touch greater than ~60% seen on anterior segment OCT or an anterior chamber depth of less than 2.5 mm were associated with increased IOP.[49] Considering these findings, we should be cautious about attributing glaucoma or an elevated IOP solely to clinically narrow angles determined by gonioscopy. It may be prudent to consider cMIGS in scenarios where angles just meet the gonioscopic criteria for "narrow" or where secondary causes such as pseudoexfoliation are identified.

Some cases of primary angle closure have a significant component of plateau iris. At times, angles may remain very narrow or closed following cataract surgery.[50] ECP is MIGS procedure with a specific application in these difficult cases. Often referred to as endocycloplasty when used to treat plateau iris, it can be combined with cataract surgery to significantly open the angle.[51] This involves the application of a diode laser via endoscope to the anterior ciliary processes in the sulcus. The endpoint is the shrinkage and retraction of the ciliary processes, directly treating the mechanism of angle closure.

What is the stage of glaucoma?

While pivotal trials generally indicated MIGS for mild-to-moderate glaucoma, some studies have shown that it may be an option for some moderate-to-advance cases.[52-54] One study compared multi-iStent to trabeculectomy in patients with moderate-to-severe glaucoma and showed that both procedures reduced IOP, but the reduction was less pronounced in the multi-stent group (mean post-operative IOP of 14.2 mmHg or 31% reduction in multi-stent group and 12.5 mmHg

or 43% reduction in trabeculectomy group).[52] Patients with multi-stent required one additional medication compared to the trabeculectomy group, but still had a 51% reduction compared to baseline. However, patients with multi-stent benefited from a more favourable safety profile and improved quality of life. Similarly, the Hydrus microstent has also shown to reduce IOP and medications in advanced glaucoma but its effects were less marked compared to those in milder glaucoma.[54] Gonioscopy-assisted transluminal trabeculotomy (GATT) combined with cataract surgery has also shown to be effective for at least 76.67% of advanced POAG cases.[24] Overall, MIGS can occasionally be an option for advanced glaucoma patients, especially if they are not good candidates for bleb-forming filtering surgeries. However, closer follow-up is mandated to ensure adequate glaucoma control and monitoring for post-operative pressure spike secondary to hyphemia, steroid response or long-term IOP elevation due to PAS.[24,25] If MIGS does not lower IOP as expected, the surgeon must have options available for more traditional surgeries soon after. In addition, longevity of effect needs to be considered carefully as long-term studies for MIGS are still limited.

Is it a refractive cataract surgery case?

Glaucoma patients are shown to have a higher incidence of refractive surprise following cataract surgery.[55] Nonetheless, they should be offered the opportunity to obtain the best refractive outcomes that they can safely achieve, paying special attention to contrast sensitivity and potential for future disease progression. Optimization of refractive outcomes through advances in diagnostics, planning software, lens options, and femtosecond laser are increasingly being offered to glaucoma patients. Numerous publications reviewing various cMIGS have demonstrated no significant effect on refractive outcome by the MIGS component.[56,57]

There are a few specific considerations in refractive cases. Post-operative visual recovery can be prolonged in many cMIGS with hyphema being one of the main transient complications. A thorough pre-operative consent process should include a discussion about these complications in order to set appropriate expectations.

Femtosecond laser can be a helpful adjunct in some refractive cataract surgery cases, but may pose some risk in certain cMIGS. Chang E. *et al.* published a case report in 2021 highlighting the risk of intractable hyphema with trabecular meshwork ablation following femtosecond laser.[58] Increased episcleral venous pressure from the docking and vacuum process was believed to be the cause. iStent following femtosecond laser has been reviewed in the literature and does not appear to have a significant hyphema risk.[57,59]

ECP has been shown to affect refractive outcomes. Wang JC *et al.* showed that patients with ACG undergoing cataract surgery combined with ECP had decreased predictability of postoperative refractive error.[60] Overall, there is a tendency toward a small myopic surprise.

Conclusion

MIGS has changed the landscape of glaucoma management. When glaucoma patients already require cataract surgery, it is an opportune time to consider adding MIGS. The risk/benefit ratio is improved as most MIGS's have not been shown to increase the risk of serious complication further than the cataract surgery itself. In the decision-making process, one should consider the degree of stability/control, the sustainability of the current treatment, whether the angle is open or closed, the stage of the glaucoma, and whether refractive cataract surgery options are being considered. Cost and local availability of MIGS options are additional factors beyond the scope of this paper. Considering the mounting evidence of its safety and benefits, cMIGS is an important component of the discussion with glaucoma patients requiring cataract surgery.

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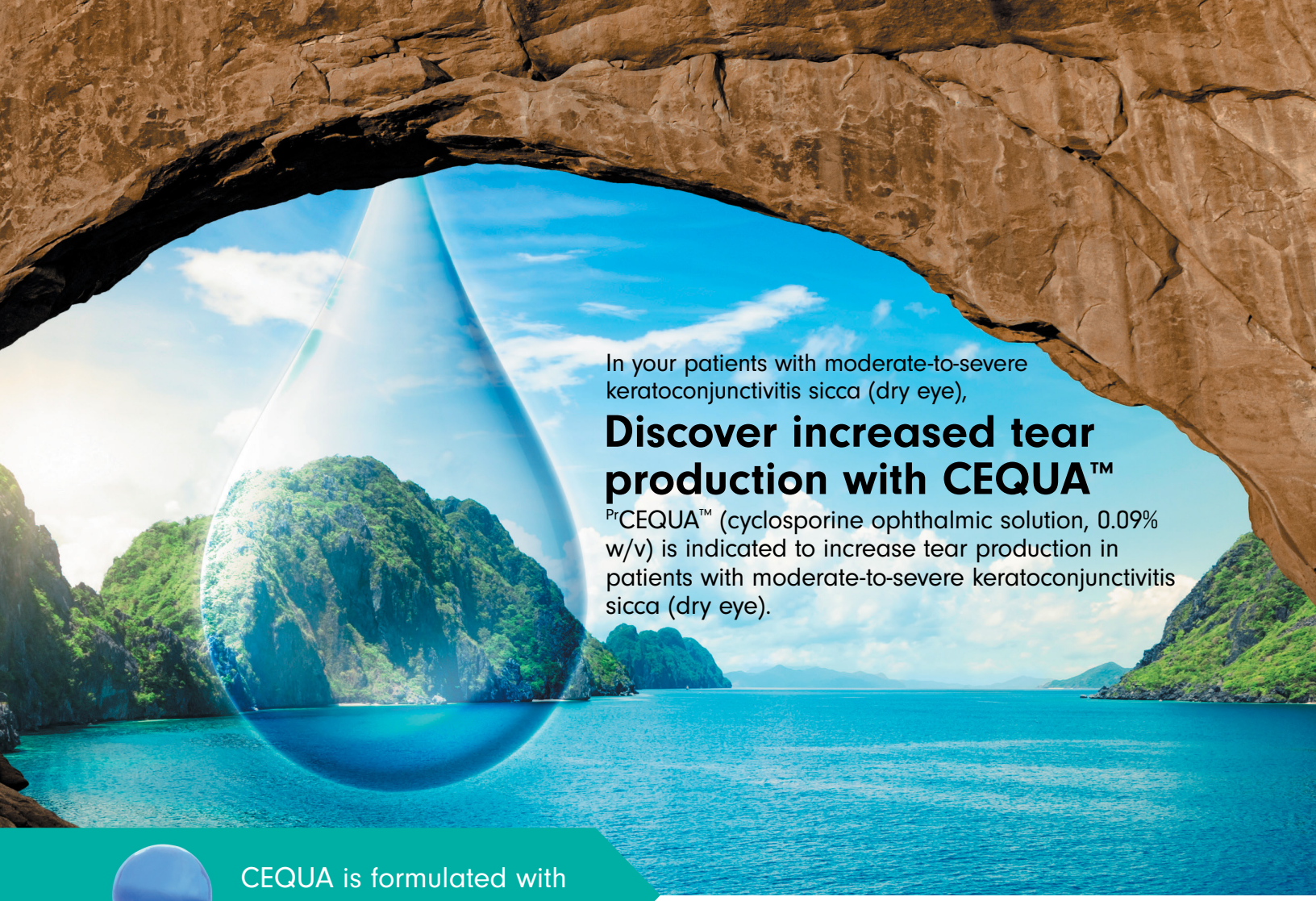

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- Hypersensitivity reactions
- The effect of CEQUA has not been studied in patients with renal or hepatic impairment
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Update on Giant Cell Arteritis: Essential Information for Ophthalmologists

Laura Donaldson, MD, PhD

Introduction

Giant cell arteritis (GCA) is an important cause of irreversible vision loss in the elderly population. For any physician, making this diagnosis can be difficult due to the highly variable clinical presentation of this large-vessel vasculitis. The 1990 American College of Rheumatology (ACR) classification criteria for GCA[1] are frequently used, however, they were developed to classify GCA patients vs those with other forms of vasculitis and are not true diagnostic criteria. Despite the high predilection of GCA for ocular circulations, the original 1990 criteria did not include any signs or symptoms related to vision. The classification criteria were updated by the ACR and European Alliance of Associations for Rheumatology (EULAR) in 2022[2] with the significant inclusion of "Sudden vision loss" (**Table 1**).

The Importance of the Ophthalmologist in Diagnosis and Management of Giant Cell Arteritis

Sudden vision loss has a broad differential and as ophthalmologists, we provide the expertise to assess these patients and determine whether vision changes are related to vasculitis. Patients with a suspected or known diagnosis of GCA are often referred for evaluation of undifferentiated vision changes. Ocular findings can confirm a diagnosis or recurrence and guide therapy including corticosteroid dose and duration. This review will focus primarily on the scenario where the ophthalmologist makes the initial diagnosis of GCA. Approximately 20% of GCA cases with ocular involvement are classified as occult,[3,4] where patients do not present with any systemic symptoms but still have elevated erythrocyte sedimentation rate (ESR) and C-reactive protein

ACR 1990	ACR/EULAR 2022	
Presence of 3 criteria classifies as GCA	Score ≥ 6 classifies as GCA Age ≥ 50 not a criterion but an absolute requirement for classification	
Clinical Criteria		
• Age at onset ≥ 50 • New headache • Abnormal temporal artery	• Morning stiffness in shoulders/neck • Sudden vision loss • Jaw or tongue claudication • New temporal headache • Scalp tenderness • Abnormal temporal artery	**+2** **+3** **+2** **+2** **+2** **+2**
Laboratory, Imaging, and Biopsy Criteria		
• ESR ≥ 50 mm/hr • Positive TAB	• Max ESR ≥50 mm/hr or CRP ≥10 mg/L • Positive TAB or TAUS • Bilateral axillary artery involvement • FDG-PET activity throughout aorta	**+3** **+5** **+2** **+2**

Table 1. Classification criteria for giant cell arteritis; *courtesy of Laura Donaldson, MD, PhD.*

Abbreviations: ACR: American College of Rheumatology, **CRP:** C-reactive protein, **ESR:** Erythrocyte sedimentation rate, **EULAR:** European Alliance of Associations for Rheumatology, **FDG-PET:** fluorodeoxyglucose positron emission tomography, **GCA:** giant cell arteritis, **TAB:** temporal artery biopsy, **TAUS:** temporal artery ultrasound

(CRP). In these cases, the ophthalmologist is likely the first or only provider seen and has the responsibility to recognize GCA and prevent further permanent vision loss and other serious sequelae of this disease.

Diagnostic key: Involvement of multiple circulations should immediately cause concern for giant cell arteritis.

- Bilateral, simultaneous, or immediately sequential anterior ischemic optic neuropathy
- Anterior ischemic optic neuropathy in combination with CRAO, cilioretinal artery occlusion, or cotton wool spots outside of the peripapillary region
- Ischemic vision loss in combination with cranial nerve palsy

Ocular Manifestations of Giant Cell Arteritis

Permanent ischemic vision loss occurs most commonly through one of three mechanisms.[3,5-9]

1. Anterior ischemic optic neuropathy (70–90%)
2. Central retinal artery or cilioretinal artery occlusion (~15%)
3. Posterior ischemic optic neuropathy (~5%)

1. Anterior ischemic optic neuropathy

Anterior ischemic optic neuropathy (AION) results from non-perfusion of the short posterior ciliary arteries supplying the optic nerve head. Approximately 90% of cases of AION neuropathy are non-arteritic (NAION), with the arteritic form (AAION) comprising the minority of cases. Ophthalmologists assessing a patient with AION

cannot consider a negative inquiry for GCA symptoms and the presence of vascular risk factors including diabetes and hypertension to be sufficient to rule out GCA. All cases of AION in a patient over age 50 must have urgent ESR and CRP measured, regardless of whether or not any GCA symptoms are reported. Other clinical features affect the pre-test probability for AAION versus NAION, however, they do not negate the need for this testing.

1. Features Supporting a Diagnosis of AAION:

a) Preceding transient monocular vision loss

Transient monocular vision loss (TMVL) is often a precursor to AAION. One case series reported 32% of patients with AAION had a preceding history of TMVL, vs 2.5% of NAION patients.[10] Partial occlusion of posterior ciliary arteries by inflammatory infiltrates decreases optic nerve head perfusion and can result in TMVL with minor increases in intraocular pressure.

b) Bilateral simultaneous or closely sequential AION

Bilateral simultaneous NAION is rare, and usually occurs in the setting of severe arterial hypotension such as in shock or during hemodialysis.[11] In AAION, bilateral involvement is common[12] and the risk of closely sequential AION increases with the interval between the time of first eye involvement and initiation of steroid treatment.

c) Absence of a crowded "disc at risk" in the fellow, unaffected eye.

The pathophysiology of NAION is thought to involve development of a compartment syndrome at the optic nerve head, where ganglion cell axons travel through small fenestrations in the rigid lamina cribrosa. Eyes that develop NAION almost always have minimal or no cupping of the optic nerve producing significant crowding of axons.[13,14] Absence of this feature should strongly raise suspicion for AAION.

d) Pallid optic disc edema

Waxy pallor of the optic disc in the acute phase is seen in approximately half to two-thirds of AAION.[12,15] In NAION, typically there is diffuse or segmental disc hyperemia evolving to pallor over weeks with resolution of disc swelling.

e) Severe central vision loss

Central visual acuity at presentation is usually worse in AAION than in NAION, with a higher proportion of arteritic cases presenting with vision of hand motions or worse. This cannot be used as a reliable predictor of AAION, however, as a minority of cases are consistently reported with vision 20/50 or better in affected eyes.[5,15]

2. Central Retinal or Cilioretinal Artery Occlusion

Approximately 5% of central retinal artery occlusion (CRAO) is arteritic and all patients over age 50 without a visible retinal embolus require urgent measurement of ESR and CRP. Reperfusion and spontaneous vision improvement may occur in embolic CRAO but this is very rare in the arteritic form.[16]

3. Posterior Ischemic Optic Neuropathy

Posterior ischemic optic neuropathy (PION) is due to vasculitic involvement of pial vessel and ophthalmic artery branches supplying the retrobulbar optic nerve.[17] The optic nerve head appears normal in the acute phase. PION is rare and almost exclusively occurs in one of three scenarios: severe systemic hypotension, in the peri-operative setting (most frequently with spinal surgery) and in GCA.

Other Ocular Manifestations of Giant Cell Arteritis

Focal Retinal Ischemia

Cotton wool spots, or focal areas of inner retinal ischemia, are common in patients with ocular involvement of GCA and may be an early sign of vasculitis.[12,18] Cotton wool spots at the optic nerve head may be seen in other causes of ischemic optic neuropathy, but if seen outside of the peripapillary region they indicate involvement of the retinal circulation. Focal middle and outer retinal ischemic changes, paracentral acute middle maculopathy or acute macular neuroretinopathy are also increasingly being recognized in GCA due to widespread availability of spectral domain and swept-source optical coherence tomography (OCT).[19]

Optic Perineuritis

GCA is an important cause of optic perineuritis in older individuals. These patients often present with evidence of optic neuropathy but relatively spared central acuity, as ischemic

changes primarily affect peripheral optic nerve axons supplied by small pial branches.[20,21] The hallmark of optic perineuritis is optic nerve sheath enhancement on MRI of the orbits with contrast.

Diplopia

Diplopia in GCA is usually caused by 3rd, 4th, or 6th nerve palsies. More rarely, brainstem ischemia can affect these cranial nerve nuclei, their fascicles or other central pathways controlling vision. Approximately 6–10% of patients with ocular involvement have diplopia and older patients with ischemic vision loss and acute oculomotor nerve palsy should be presumed to have GCA until proven otherwise.[12,22,23]

Uncommon Ocular Signs of GCA

- Hemianopia secondary to posterior circulation stroke
- Horner's syndrome
- Anterior segment ischemia and ocular ischemic syndrome
- Orbital inflammatory syndrome

Work-up of GCA: To Biopsy or Not

Temporal artery biopsy (TAB) is considered the gold standard for diagnosis of GCA and is still the first choice of neuro-ophthalmologists, particularly in North America.[24] Biopsy also has the advantage of identifying other pathology that can mimic GCA,[25] including small vessel vasculitis, amyloidosis, lymphoma, and sarcoidosis.

The sensitivity of unilateral TAB is likely around 77–87%[26,27] with very high specificity. The ACR recommends TAB performed within two weeks of corticosteroid initiation and with a specimen at least 1 cm in length, with TAB preferred over temporal artery ultrasound (TAUS) and other imaging modalities.[28]

The use of imaging as an alternative to TAB for diagnosis of GCA is becoming increasingly common. Updated EULAR guidelines for imaging in GCA were published in 2023[29] and recommend consideration of TAUS, high resolution MRI, and fluorodeoxyglucose positron emission tomography (FDG-PET) as first-line options over TAB. FDG-PET has more limited availability and is used more often in suspected extracranial GCA.

TAUS, looking for a "halo sign" and temporal artery compressibility, has advantages of being non-invasive and inexpensive. The bilateral temporal arteries and axillary arteries can also be simultaneously assessed for evidence of vasculitis. TAUS sensitivity in the hands of an experienced ultrasonographer is likely similar to TAB, but with lower specificity.[30,31] A major limitation is that false negatives occur quickly after steroid initiation, in as little as 2 days.[32]

MRI angiography also has the advantage of being less invasive, and allows for simultaneous assessment of all major cranial vessels. Alternate pathology including stroke, sinusitis and TMJ disease can also be identified.[33] Standardized imaging protocols on high resolution (at least 3T) machines read by radiologists experienced with this specific procedure can give high sensitivity,[34] but currently this is difficult to achieve outside of tertiary centres. Like TAUS, MRI findings of GCA can also normalize quickly following administration of corticosteroids.[35]

If TAUS or MRI with vasculitis protocol can be performed quickly and by experienced operators, these modalities are good options to rule in GCA when pre-test probability is high.

Treatment of GCA

The mainstay of GCA treatment is high-dose corticosteroids to prevent onset or worsening of permanent vision loss and to induce remission. Vision loss from GCA is a true ophthalmologic emergency and initiating treatment immediately is key. In the pre-steroid era, the prevalence of permanent vision loss from giant cell arteritis was probably around 40%.[36] Currently, the rate of irreversible ischemic vision loss in at least one eye is approximately 8–17%, with longer time to treatment associated with higher risk.[5,37-39]

Most patients with bilateral ischemic vision loss from GCA lose vision simultaneously, or sequentially before the diagnosis of GCA is made.[40] After initiation of corticosteroids the risk of vision loss in a fellow, unaffected eye is highest within the first two days, with the longest interval in a recent systematic review reported to be 12 days.[41] Progression of vision loss in an already affected eye is similarly rare with highest risk in the first few days of treatment.[42]

There is ongoing debate about whether to treat new onset GCA with intravenous (IV) or oral corticosteroids. In a recent survey of neuro-ophthalmologists, 52% routinely

treat GCA patients with vision loss with IV methylprednisolone, and only 3% routinely initiate IV treatment in GCA patients without vision loss.[43] Both the ACR and EULAR do conditionally recommend IV corticosteroids for patients with ischemic vision loss.[28,44] However, the quality of evidence guiding this decision is low and there are no randomized trials to determine whether or not IV corticosteroids can prevent further deterioration or increase the likelihood of vision improvement in these patients.[45] High-dose oral prednisone at a dosage of approximately 1 mg/kg/day can be an acceptable option for patients with ischemic vision loss from GCA, particularly if organizing IV corticosteroids would delay treatment.

Early referral to rheumatology should be made for consideration of adjuvant therapies. Tocilizumab has shown clear benefit in reducing total corticosteroid requirement in GCA[46] and is now routinely used early in the disease course.[28] Methotrexate is also a common option as a steroid-sparing agent.[44]

Conclusion

Approximately 20% of patients with ocular GCA lack systemic symptoms and a result usually present first to ophthalmology. Recognition of how GCA manifests differently than other causes of ocular ischemia is key for early diagnosis and immediate initiation of high-dose systemic corticosteroids. TAB remains the gold standard for confirming a diagnosis; TAUS and MRI are likely to play greater roles in the future as standardized protocols are adopted and expertise in interpretation becomes more widespread.

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Ocular Fundus Tumours: A Simplified Clinical Classification

Hatem Krema, MD, MSc, FRCS, FICO (Hon.)

Introduction

The diagnosis of the most common ocular fundus tumours can be achieved according to clinical features including their malignant potential, anatomical location within the eye, and relation to systemic disease, as well as imaging features. The majority of these tumours can be classified into four major categories according to their clinical presentation.

I- Melanotic Tumours

The source of melanin in the ocular fundus is either the choroidal melanocytes or the melanin in the retinal pigment epithelium (RPE). According to the melanin content within their cells, the melanotic tumours present with varying shades of brown colour.

1) Choroidal Melanotic Tumours:

a) Choroidal nevus: It is a small, flat, or slightly raised area of circumscribed choroidal

melanocytic proliferation. It grows very slowly over the years and eventually stabilizes, with an overall prevalence of about 2%. A chronic nevus demonstrates retinal degenerative changes on its surface due to chronic nutrition deprivation of the RPE by the nevus mass, which compresses and displaces choriocapillaries (**Figure 1A**). These degenerative changes manifest as RPE atrophy, RPE metaplasia into a grey collagenous membrane, RPE migration and proliferation around nearby blood vessels, drusen formation, and later intraretinal cysts or subretinal neovascular membrane (SRNVM) formation. A nevus in the macular area may lead to eventual central vision loss from gradual attrition of the RPE and subsequent degeneration of the overlying neuroretina.[1]

b) Uveal melanoma: This is the most common primary intraocular tumour in adults, with an annual incidence of 4–6/million in the population. Choroidal melanoma constitutes 85% of uveal melanoma, ciliary body melanoma is 10%, and iris melanoma is 5%. Choroidal melanoma typically presents as an elevated dome-shaped subretinal mass, which may be associated with subretinal fluid (SRF), lipofuscin deposits (orange pigment), or hemorrhage on its surface (**Figure 1B**).

The tumour may perforate through Bruch's membrane, in which case it assumes a collar button or mushroom configuration and may rarely invade the retina, causing pigment dispersion within the vitreous. Choroidal melanoma can be non-pigmented (amelanotic) or partially melanotic; it typically reveals low internal reflectivity and choroidal excavation in ultrasonography and dual circulation in fluorescein angiography (FA).[2,3]

c) Indeterminate melanocytic lesion (IML): This term describes a small choroidal lesion with mixed features between a nevus and a small melanoma, the biological nature of which cannot be ascertained by a single clinical exam (**Figure 1C**). The management is periodic observation every 3–4 months to detect progressive growth and, if noticed, the lesion is treated as a small melanoma. The clinical features suggestive of eventual growth in a small IML include tumour thickness >2 mm, subretinal fluid, visual symptoms, orange pigment, echogenic hollowness, and diameter >5 mm. The presence of 3 of these features predicts growth in 1/3 of the lesions.[4,5]

d) Choroidal melanocytoma: This is a form of nevus, which is characterized by larger cells that accommodate more melanin-filled melanosomes in their cytoplasm. Thus, it appears densely dark with feathery edges and surrounding pigment dispersion.

e) Pseudomelanoma: These are fundus lesions that are not of melanocytic origin but may simulate choroidal melanocytic lesions. These include subretinal hematoma of various causes, hemorrhagic pigment epithelium detachment, dilated ampulla of the vortex vein, uveal effusion that may simulate ring melanoma of the ciliary body, and scleral or orbital mass indenting the choroid.

2) Retinal Melanotic Tumours:

a) Congenital hypertrophy of the retinal pigment epithelium (CHRPE): It presents as single or multiple flat, grey-to-black lesions with well-demarcated edges. It may show lacunae devoid of RPE pigment (**Figure 1D**). Multiple CHRPE-like lesions of tadpoles-like morphology may be a manifestation of Gardner's syndrome. Torpedo maculopathy describes a paramacular albinotic patch of RPE that causes disruption of the overlying outer retina.[6]

b) Optic disc melanocytoma: Similar to uveal melanocytoma, it presents as a grey to black mass involving the optic disc, arising from melanocytes at the lamina cribrosa, and may be associated with a choroidal component (**Figure 1E**). It may display minimal growth in 10–15% of cases, but malignant transformation is exceedingly rare. It is typically asymptomatic but may show an afferent pupillary defect, enlargement of the blind spot, or arcuate field defect as compressive symptoms.[7]

c) Combined hamartoma of the retina and RPE: It manifests as a posterior pole area of retinal thickening with distorted vessels and traction of the surrounding retinal vessels. It has a grey hue from the RPE component. Fluorescein angiography highlights the abnormal vasculature, and optical coherence tomography (OCT) is diagnostic.[8] It is typically sporadic but could be a manifestation of neurofibromatosis type II.

d) RPE hamartoma: It is a localized small area of dark-coloured RPE thickening without impact on the overlying retina (**Figure 1F**).

e) RPE adenoma/ adenocarcinoma: This appears as a central or peripheral darkly pigmented, abruptly elevated mass (Derby-hat configuration) surrounded by SRF and hard exudates, with a feeder artery and a draining vein. Adenocarcinoma is locally aggressive but seldom metastasizes.[8,9]

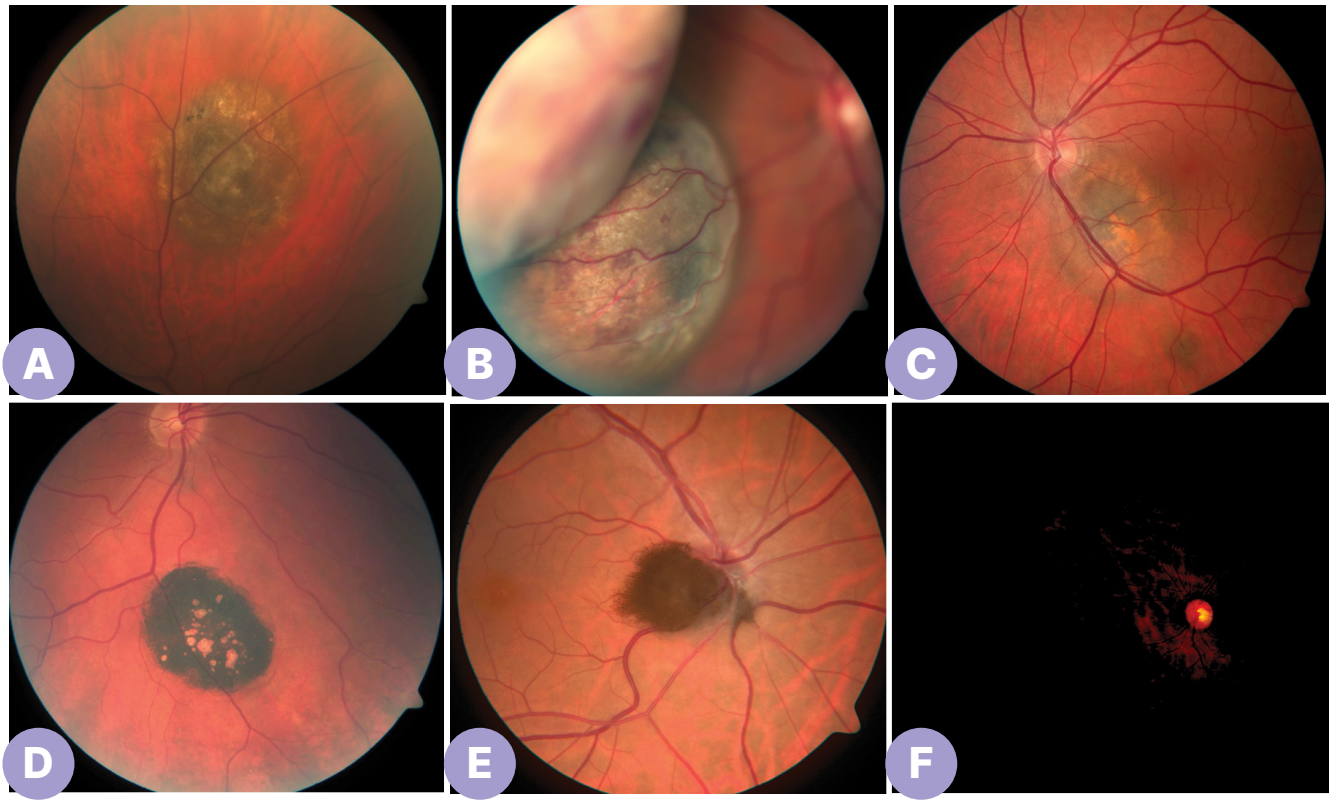

Figure 1. Melanotic fundus tumors; **A)** choroidal naevus appears flat with surface drusen; **B)** choroidal melanoma showing as choroidal pigmented mass with adjacent retinal detachment; **C)** Indeterminate melanocytic lesion (IML) appears slightly thicker with surface orange pigment; **D)** Congenital hypertrophy of the retina and RPE (CHRPE) appears as a well-demarcated flat dark lesion with amelanotic lacunae; **E)** Optic disc melanocytoma with feathery edge and pigment dispersion; **F)** RPE hamartoma appears as a dark small circumscribed lesion; *courtesy of Hatem Krema, MD, MSc, FRCS, FICO (Hon.)*.

f) Pigmented gliotic scar: This appears as a variegated retinal lesion resulting from RPE proliferation within a nonpigmented mass of gliosis.

g) Metastatic cutaneous melanoma mostly involves the retina and vitreous system, as well as diffuse perivascular pigmented clumps and dark vitreous debris.

II- Amelanotic Tumours

This is a group of non-pigmented tumours that are neither vascular nor calcified; they typically present as white to creamy yellow lesions.

1) Choroidal metastasis: Typically present in a patient with a history of systemic cancer, but 20% of patients are unaware of their systemic cancer. The most common primary site is the lung or breast. The most frequent presentation is a unilateral unifocal lesion, but metastases may be multifocal and bilateral. It differs from amelanotic choroidal melanoma in being rapidly growing, usually with significant SRF and "leopard skin" appearance from significant surface deposition of lipofuscin from the irritated RPE (**Figure 2A**). Unlike melanoma, metastasis displays a medium to high internal reflectivity in ultrasonography, a mountain-like "lumpy bumpy" profile in OCT, and an absence of dual circulation in FA. Diagnosis is usually clinically based, particularly with a history of systemic cancer, although a needle biopsy may be needed in a few cases.[10]

2) Amelanotic melanoma: This represents less than 20% of choroidal melanomas. It may show intrinsic vascularization or surface hemorrhage and may assume a configuration similar to pigmented melanoma (**Figure 2B**).

3) Intraocular lymphoma: It can be broadly classified into:

a) Vitreoretinal lymphoma of large B-cell lymphoma with +/- CNS involvement. This

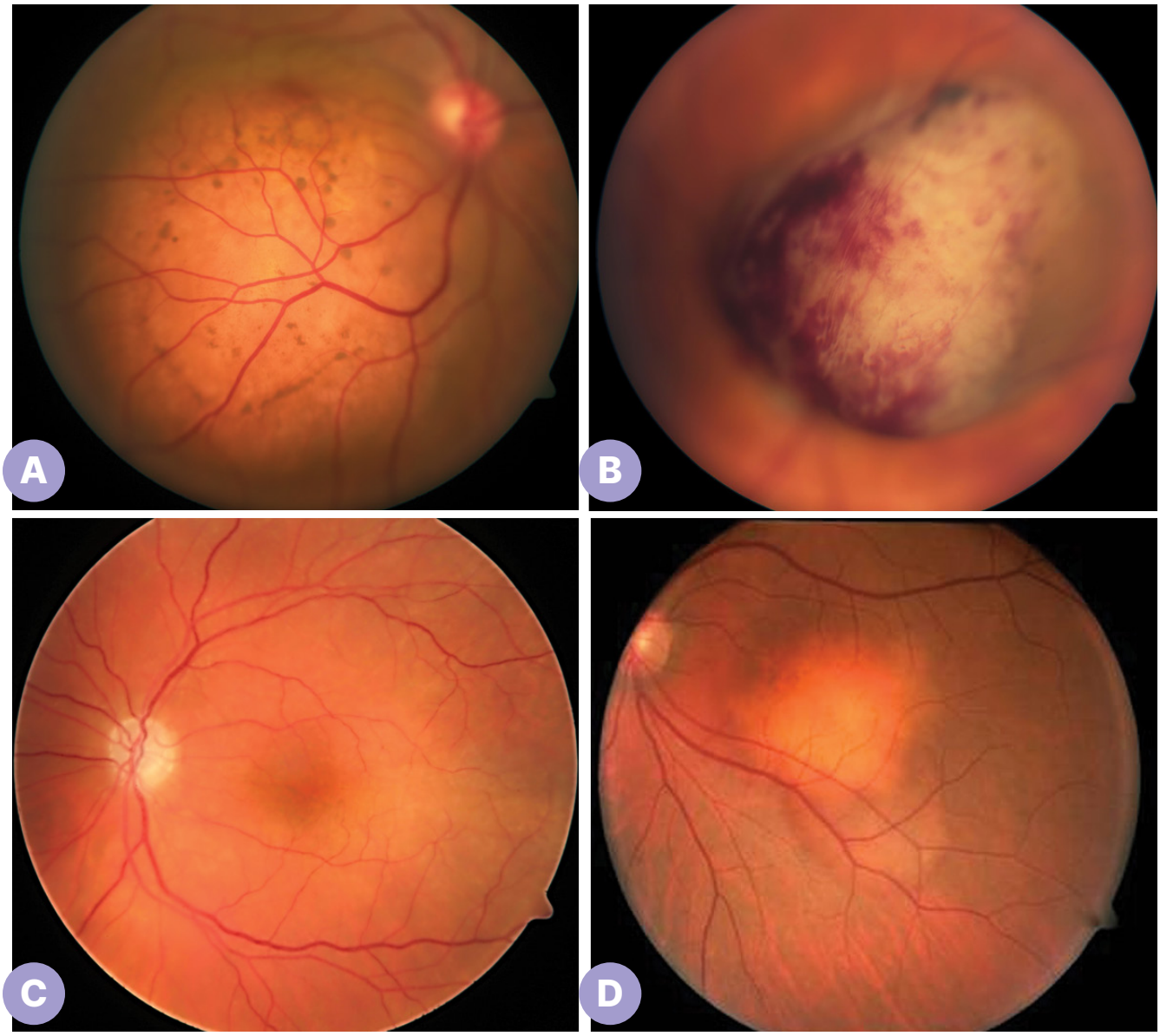

Figure 2. Amelanotic fundus tumours; **A)** Amelanotic melanoma with collar-button configuration and surface subretinal hemorrhage; **B)** Choroidal metastasis appears as an amelanotic choroidal mass with surface changes showing leopard-skin appearance; **C)** Choroidal lymphoma appears as ill-defined amelanotic choroidal infiltration; **D)** Choroidal granuloma of sarcoidosis appears as an amelanotic flat lesion with irregular margin with an adjacent pocket of subretinal fluid; *courtesy of Hatem Krema, MD, MSc, FRCS, FICO (Hon.)*.

manifests as steroid-resistant vitritis with patches of retinal infiltrates simulating retinitis. It runs an aggressive course with a high risk of recurrences and mortality.

b) Uveal and adnexal lymphoma: This manifests as single or multiple, unilateral or bilateral, choroidal infiltrates of non-Hodgkin's small B-cell lymphoma, forming diffuse thickening of the choroid or discrete masses (**Figure 2C**). These lesions may be associated with co-involvement of adnexal structures, such as a conjunctival salmon patch, or diffuse swelling in adjacent orbital structures. This lymphoma runs a less aggressive course.[11]

4) Choroidal granuloma: This manifests as single or multiple choroidal masses of irregular borders, adjacent satellite lesions, and local vitritis (**Figure 2D**). The nature of such lesions

may include sarcoidosis, tuberculosis, less likely toxoplasmosis, toxocariasis, cat-scratch disease, or other specific or non-specific inflammations. Uveitis work-up may lead to diagnosis, but a choroidal biopsy may be required in some cases.[12]

5) Retinal amelanotic tumours: These include rare tumours of the retinal supportive elements such as retinal schwannoma or medulloepithelioma of the nonpigmented epithelium of the ciliary body.

III- Vascular Tumours

These can be diagnosed in fundoscopy by their colour, which ranges from orange to bright red.

1) Circumscribed choroidal hemangioma: This presents as a solitary orange-pink circumscribed mass that may be associated with SRF (**Figure 3A**). It is sporadic but should be differentiated from vascular orange-colored solitary metastasis of renal, neuroendocrine and thyroid cancers and highly vascularized amelanotic melanoma. Hemangioma exhibits progressive fluorescence in the early phases of FA and "the ring sign" in the late phases of ICG.[13]

*2) Diffuse choroidal hemangioma: I*t is a manifestation of Sturge-Weber syndrome or Phacomatosis Pigmentovascularis.[14] It presents as an ill-defined orange-pink diffuse lesion that may involve most of the choroid, with thickened areas, termed "tomato ketchup fundus." It may be associated with significant transudative retinal detachment (**Figure 3B**).

3) Retinal hemangioblastoma: It is a manifestation of Von Hipple- Lindau disease. It presents as single or multiple, unilateral or bilateral, bright red lesions surrounded by SRF and hard exudates. Peripheral larger lesions may have a feeding artery and a draining vein, which are lacking in sizable lesions at the optic disc (**Figure 3C**). Acquired sporadic retinal capillary or cavernous hemangiomas are rare and not associated with systemic diseases.[15]

4) Vasoproliferative tumour of the ocular fundus (VPTOF): It presents as a peripheral retinal grey-pink growth, surrounded by SRF and hard exudates, but without the feeder vessels observed in hemangioblastoma (**Figure 3D**). The VPTOF may be multiple or bilateral but has no systemic association. Macular cysts and epiretinal membrane formation are frequently present due to VEGF secretion by the tumour. Secondary VPTOF may be associated with retinal disease and thought of as a reactive gliotic response.[16]

IV- Calcified Tumours

These tumours contain foci of calcification, detectable with ultrasonography as highly reflective areas within the tumour that cast an orbital shadow. In doubtful cases, a CT scan can confirm calcification.

1) Choroidal osteoma: It typically manifests in middle-aged females as a unilateral, slowly progressive juxtapapillary lesion, which is rather flat and vascularized with an irregular, rugged surface (**Figure 4A**). It may lead to significant vision loss from the attrition of the RPE in the macular area or the formation of SRNVM.[17]

2) Idiopathic sclerochoroidal calcification: It typically presents as ill-defined subretinal yellow lesions near the equator, mostly multifocal and bilateral. (**Figure 4B**). Some deeper lesions may not be observable by fundus exam and could be detected with ultrasonography of the equator. OCT shows subretinal lesions with an irregular profile, indenting the overlying normal retina. It may be associated with abnormalities in serum calcium, phosphorus, or potassium levels.[18]

3) Retinal Astrocytic Hamartoma: It is a benign growth of retinal glial cells that may present as a unilateral unifocal lesion or as multiple or bilateral lesions in association with tuberous sclerosis complex. Reactive astrocytic gliosis has been associated with NF1, retinitis pigmentosa, Stargardt's disease, and gyrate atrophy. Morphologically, the astrocytic hamartomas are classified into three types. **Type 1 (most common):** relatively flat, smooth, semitransparent lesions without calcification; **Type 2:** raised, multinodular ("mulberry-like"), opaque, totally calcified lesions; **Type 3:** lesions with mixed features of Types 1 and 2 (**Figure 4C**). Astrocytoma is a neoplastic growth. Giant astrocytoma with aggressive behaviour has been rarely reported.[19]

4) Retinoblastoma (RB): It is the most common pediatric intraocular cancer. It presents as leukocoria in half of the patients and strabismus in one-third. Sporadic Rb is unilateral and unifocal, but germline-mutation RB may be unifocal or multifocal and bilateral. Endophytic RB may simulate astrocytic hamartoma, while exophytic RB may simulate Coats' disease, which presents with abnormal peripheral retinal vessels and copious

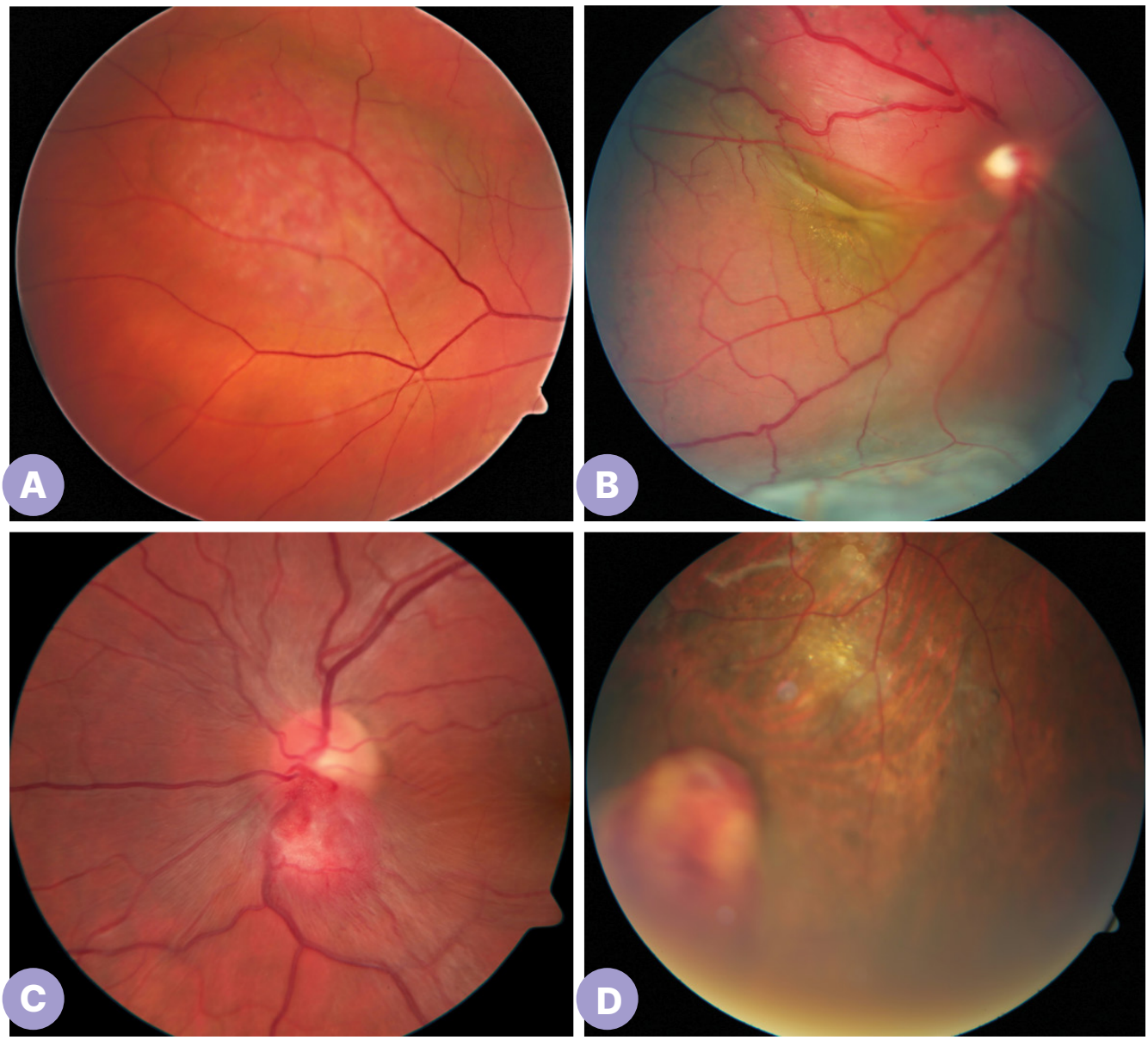

Figure 3. Vascular fundus tumours: **A)** Circumscribed capillary hemangioma; **B)** Diffuse capillary hemangioma with overlying retinal detachment; **C)** Retinal hemangioblastoma at the inferior edge of the optic disc; **D)** Vasoproliferative tumour with associated hard exudates; *courtesy of Hatem Krema, MD, MSc, FRCS, FICO (Hon.).*

creamy yellow exudation; nevertheless, clinical differentiation may not be possible in some cases (**Figure 4D**). Other causes of leukocoria include retinopathy of prematurity, retinal dysplasia, and coloboma, uveitis, and persistent hyperplastic primary vitreous. Adult aggressive RB is rare, yet a retinoma may be incidentally discovered in an adult as a dormant lesion.[20]

Conclusion

The diagnosis of the most frequent ocular fundus tumours depends mainly on clinical and imaging features without the need for a diagnostic biopsy. The presented simplified classification herein can help clinicians distinguish between the majority of the common fundus tumours encountered in their practice.

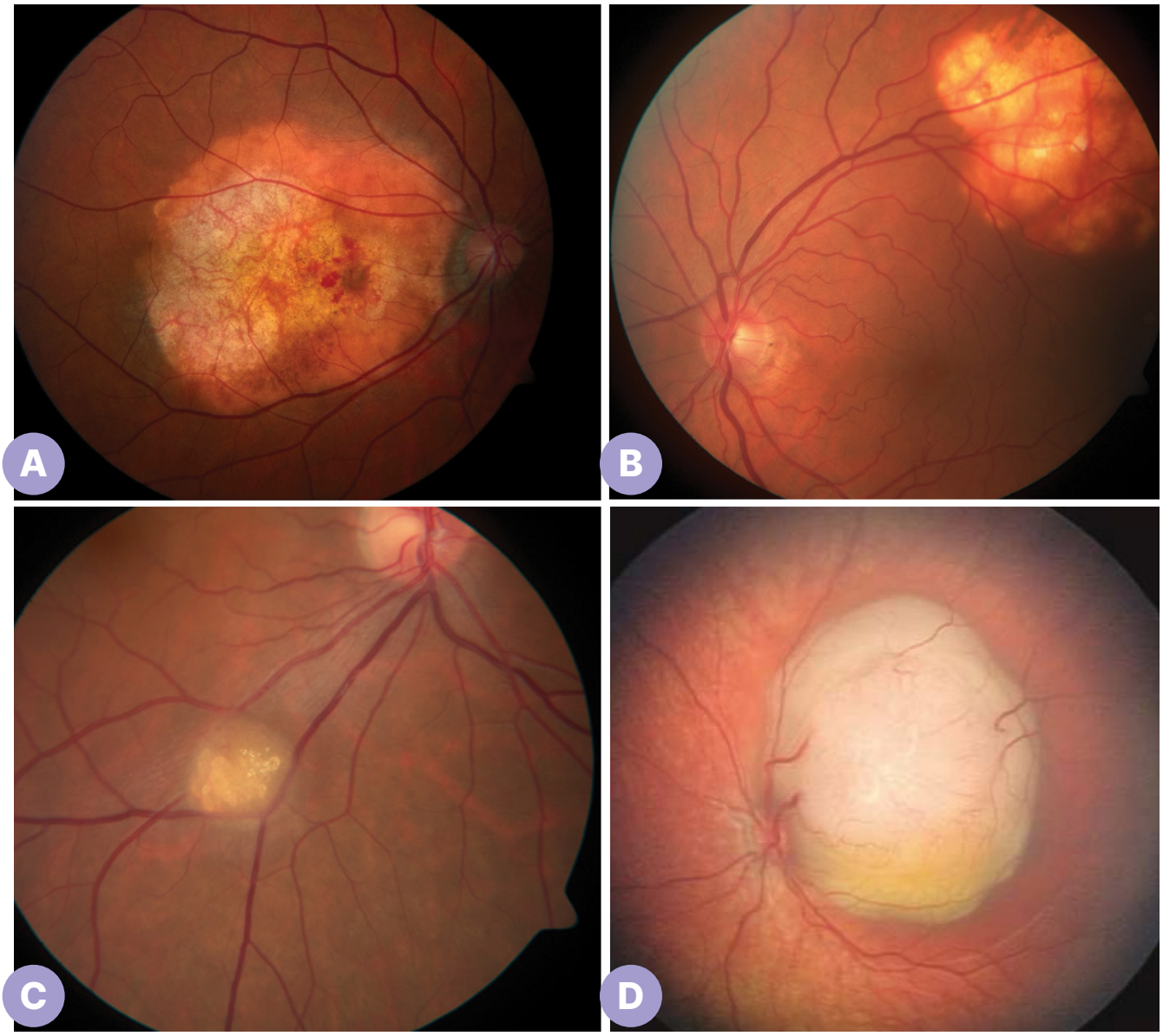

Figure 4. Calcified fundus tumors: **A)** Choroidal osteoma appears as a rugged surface posterior pole lesion with intrinsic vessels; **B)** Idiopathic sclerochoroidal calcification appears as a choroidal glistening irregular mass at the fundus mid periphery under the arcades; **C)** Retinal astrocytic hamartoma appears as retinal mass gelatinous mass with foci of calcifications; **D)** Retinoblastoma, endophytic type, presenting as a white mass with feeder retinal vessels and glistening intrinsic calcification; *courtesy of Hatem Krema, MD, MSc, FRCS, FICO (Hon.)*.

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Standard of Care for First-line Therapy in Newly Diagnosed Glaucoma and Update on SLT from the LiGHT Trial

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Introduction

Glaucoma is a progressive, multifactorial disease marked by ganglion cell loss, optic nerve damage and progressive vision loss, which can result in blindness if not treated. Glaucoma accounts for 11% of registrations for blindness.[1] The disease is closely linked to increased intraocular pressure (IOP) and reducing this pressure is the sole available treatment to slow disease progression.[2] The epidemiology of glaucoma presents a significant public health challenge, with primary open-angle glaucoma (POAG) being the most common form, affecting approximately 2–3% of adults over the age of forty.[3] Many patients can be initially managed with medications; however, the treatment has significant limitations. Issues such as complications, side effects, adherence, nonresponse, reduced effectiveness over time (tachyphylaxis), and financial costs pose challenges to controlling IOP with eye drops. The global burden of glaucoma is expected to increase as the population ages, highlighting the urgency for effective management strategies. The landmark LiGHT (Laser in Glaucoma and Ocular Hypertension) trial, published in 2019, with an initial 36 months of follow-up,[4] later extended to 72 months of follow-up,[5] has conceptually influenced the management of POAG and Ocular hypertension (OHT). By demonstrating the efficacy and safety of selective laser trabeculoplasty (SLT), a “dropless” and “knifeless” alternative as a first-line treatment option, the LiGHT trial challenged the conventional treatment paradigm. The six-year results further consolidate SLT’s role as a fundamental treatment option, indicating its long-term effectiveness and durability in managing glaucoma, potentially redefining standard care protocols.

Therapeutic Options for Glaucoma: Historical Overview

The management of glaucoma has traditionally centered on the reduction of IOP to halt or slow down the progression of optic nerve damage. Therapeutic options include medications (mainly topical eye drops), laser treatments and surgical interventions. Each treatment modality aims to decrease eye pressure, either by improving aqueous humour outflow or reducing its production. Until recent years, the paradigm for treating glaucoma followed a linear and sequential approach. Initially, therapy would commence with the prescription of topical eye drops, aiming at lowering IOP to target levels. As the condition progressed or if initial treatments proved insufficient, the strategy involved the addition of other eye drops, each with a different mechanism of action, to enhance the IOP-lowering effect. If this pharmacological approach did not achieve the desired outcomes, laser trabeculoplasty (LTP) treatment was considered as the next step in the treatment paradigm. Ultimately, if both medical and laser therapies failed to control the disease adequately, traditional glaucoma surgeries were employed as the final resort. This approach was primarily guided by the principle of avoiding the potentially devastating adverse events associated with traditional filtering procedures.

IOP lowering eye drops, the primary treatment for many patients, provide a non-invasive method to manage IOP. Nonetheless, their efficacy is compromised by several challenges. Non-adherence to treatment regimens, which is reported to be as high as 50%,[6,7] significantly undermines treatment outcomes. Patients may also experience ocular

surface irritation, including symptoms such as burning, dryness, conjunctival hyperemia and a sensation of a foreign body in the eye.[8] Over time, the effectiveness of these medications can decrease, further complicating treatment efforts. Additionally, the impact on patients' quality of life[9] combined with the requirement for lifelong daily administration and the risk of potential systemic side effects, frequently results in suboptimal therapeutic success.

Paradigm Shift: The Rise of Interventional Glaucoma

In recent years, as understanding of disease progression mechanisms deepens, along with recognition of the critical need for early and effective IOP reduction and the challenges of medical treatment, the field of glaucoma has witnessed a paradigm shift toward "Interventional Glaucoma"—a concept that emphasizes early intervention (whether invasive or none) over traditional pharmacotherapy. Procedures such as minimally invasive glaucoma surgeries (MIGS) and minimally invasive bleb-based surgeries (MIBS) have revolutionized treatment by offering safer, effective alternatives to lower IOP, with fewer complications and a more favourable impact on the patient's lifestyle compared to lifelong medication use.

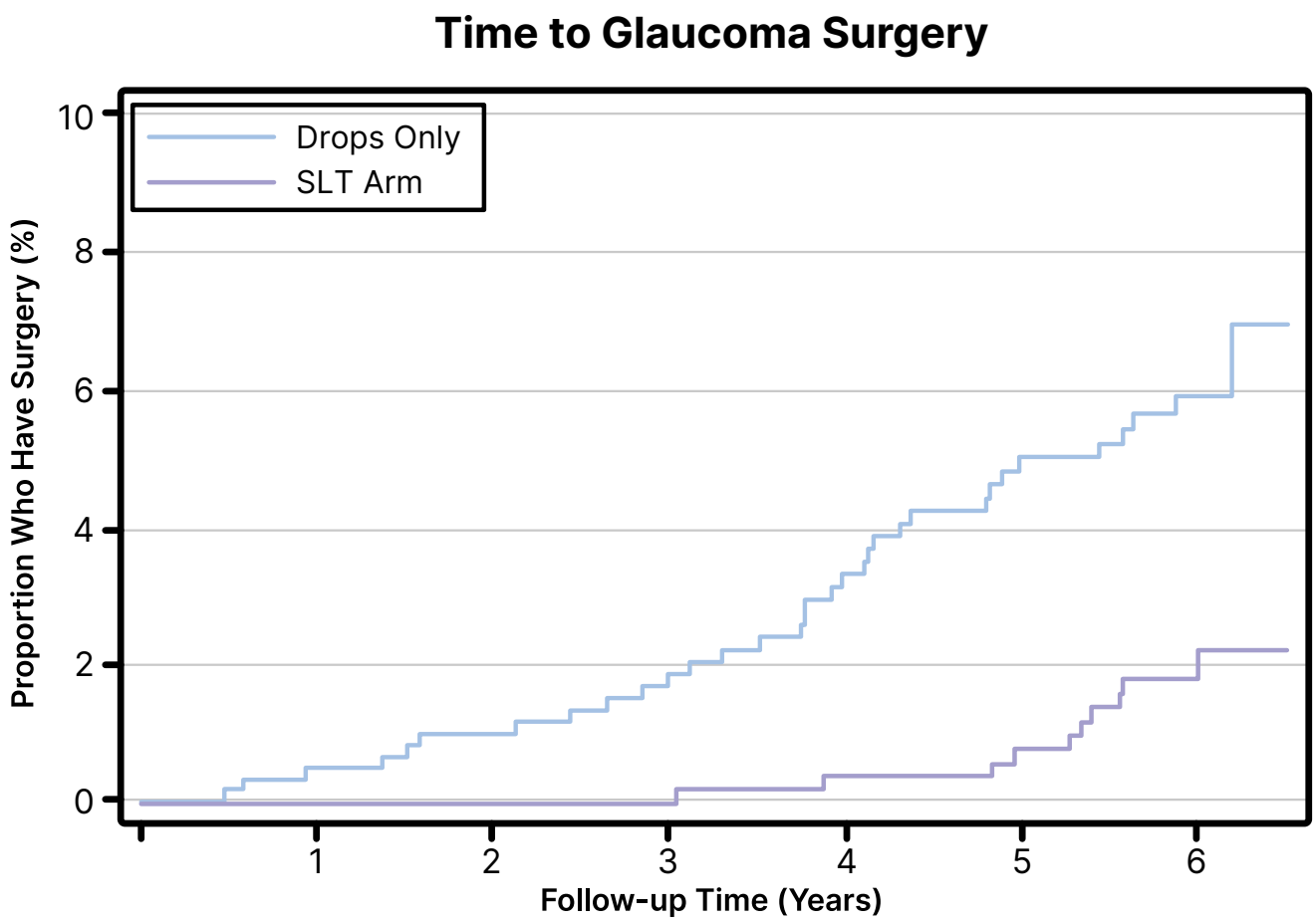
SLT, a noninvasive glaucoma intervention, has emerged as a pivotal treatment modality, particularly for patients with POAG in 1995 and was approved by the FDA in 2001.[10] SLT utilizes nanosecond low-energy light, undertaken in single-shot mode, directed at the angle to selectively target the pigmented trabecular meshwork elements. The enhanced outflow facility and reduced IOP typically manifest within 4 to 6 weeks, although the mechanism behind the improvement of outflow facility remains uncertain. Randomized controlled trials (RCTs) have demonstrated a decrease in the number of IOP medications required for IOP control in patients with POAG and OHT.[11,12] Additionally, there is increasing evidence supporting satisfactory outcomes in terms of repeatability.[13,14]

The LiGHT trial, a multicentre RCT conducted in the United Kingdom, enrolled 718 patients with POAG or OHT. The study compared SLT treatment with IOP-lowering medications, employing a stepwise strategy for adding medications or SLT as required to achieve target IOP. The results demonstrated that SLT was as effective as medications in controlling IOP. At 36 months, 78.2% of patients receiving SLT achieved their target IOP without the need for additional medication, in contrast to 64.6% in the medication group who were on a single medication. By 72 months, 69.8% of the SLT group had maintained their target IOP without requiring additional medications or surgeries. At 36 months, SLT was also found to be more cost-effective as an initial therapy compared to medications within the United Kingdom's healthcare system. Another significant outcome of this study was that while the 36-month results of the LiGHT trial indicated no measurable improvement in quality of life with SLT compared to medications, according to various quality of life surveys; yet the extended follow-up at 72 months showed improved Glaucoma Symptom Scores for the SLT group.

Nevertheless, one of the most impactful findings of this study was that at 72 months, fewer eyes in the SLT group experienced glaucoma progression (19.6% vs 26.8%), and fewer required trabeculectomy (13 eyes vs 32 eyes) (**Figure 1**). This might suggest that using SLT as a first-line treatment could not only successfully lower IOP, but also potentially alter the course of the disease. The reduced likelihood of undergoing filtration surgery and avoiding the well-known and not uncommon complications of filtration surgery is of great significance. This outcome suggests that SLT could offer a substantial benefit in reducing the progression of glaucoma, thereby decreasing the need for more invasive interventions.

The Transformative Impact of the LiGHT Trial

Smaller trials leading up to LiGHT showed similar results with SLT: It worked as well as IOP lowering drops as a first-line therapy to lower pressure with minimal side effects; still there was little movement away from drops. The Medicare billing study showed that SLT was performed in less than 5% of people with glaucoma.[15] As mentioned, the LiGHT study is the largest RCT with the longest follow-up period to date, comparing SLT as an initial treatment with IOP-lowering drops. It has already initiated a shift and is likely to continue influencing the transition from medication to laser as the first-line management approach. The study has shed clear light on previously undetermined issues. It confirmed the efficacy and safety of SLT, the potential benefits of repeated treatments and its cost-effectiveness superiority.



Number of Patients at Risk/Years	0	1	2	3	4	5	6*
Drops Arm	361	352	242	334	307	291	266
SLT Arm	355	351	345	328	304	287	270

Figure 1. Failure plot indicating time to glaucoma surgery from baseline by treatment arm (P < 0.001, log-rank test) based on intention-to-treat analysis (y-axis on a scale of 0-10%; the unit of analyses is the eye). The number at risk at 6 years includes the patients whose last visit was +/- 6 months; *adapted from Gazzard, Gus et al, 2023; doi:10.1016/j.ophtha.2022.09.009; https://creativecommons.org/licenses/by/4.0/*

Abbreviations: SLT: selective laser trabeculoplasty

Additionally, it has unveiled intriguing insights into possible disease mechanisms. The early utilization of SLT has shown significant decrease in disease progression and the need of subsequent glaucoma surgeries. This potential alteration in the course of the disease suggests that early intervention targeting the pathophysiological tissue involved could help prevent future fibrosis and stiffness in the more distal outflow system.

From a public health perspective, the results of the study would suggest laser for everybody with early-to-moderate open-angle glaucoma, and indeed, primary SLT at diagnosis is now recommended as the preferred treatment by the UK National Institute of Health and Care Excellence (NICE), and as an equivalent alternative in the European Glaucoma Society Treatment Guidelines and the American Academy of Ophthalmology Preferred Practice Patterns.

Nonetheless, like everything in life, the implementation of new guidelines is not without its challenges and barriers, requiring time, effort and perseverance to overcome these. One significant challenge is overcoming clinical inertia and tradition, as many ophthalmologists are accustomed to initiating treatment with topical medications, a practice deeply rooted in decades of clinical experience. Additionally, not all ophthalmologists may be trained or have experience in performing SLT, limiting their ability to offer this treatment option, especially in areas where access to training or laser equipment is scarce. Patient's acceptance and perception also play a crucial role; many might prefer starting with what they perceive as less invasive options like eye drops, necessitating thorough patient education about the benefits of SLT. Access to the necessary laser equipment can be a barrier, particularly in under-resourced or rural areas. Furthermore, the initial cost of SLT, compared to that of topical medications, and potential reimbursement issues could make it financially unfeasible for some patients. Last, there is a lack of awareness among healthcare providers and patients about the latest evidence supporting SLT as a first-line treatment option. To address many of the challenges outlined previously, a key solution lies in education. This education is targeted not only at glaucoma specialists but also at primary care physicians, general ophthalmology specialists, optometrists, and patients themselves. For instance, NICE has recognized these barriers and has put forth initiatives to support healthcare professionals. These initiatives include providing comprehensive training on the suitability and safety of SLT, along with its benefits and risks, and enhancing the skills needed to effectively discuss these factors with patients.

Conclusion

By addressing the drawbacks of traditional treatments and presenting a viable, less invasive alternative, the LiGHT trial has set a precedent in the glaucoma treatment landscape, advocating for a shift toward more patient-friendly and efficacious management strategies. This long-term data provided a stronger case for the adoption of SLT as a preferable first-line treatment in glaucoma management, underscoring its benefits not just in controlling IOP but also in enhancing patients' overall well-being by changing the course of their disease.

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Preventing Work-related Musculoskeletal Injuries

Rookaya Mather, MD, FRCSC

Introduction

"... the primary focus in healthcare has been on immediate patient care rather than the long-term well-being of healthcare workers. This has often led to ergonomic considerations being overlooked." [1]

Work-related musculoskeletal disorders (WMSD) are very common among ophthalmologists. The literature suggests that anywhere from 35 to 93% of ophthalmologists experience WMSDs. These disorders and injuries predominantly affect the neck, lower back and shoulders.[2] As discussed in past issues of *Canadian Eye Care Today*, WMSDs are associated with suboptimal ergonomics related to the slit lamp and operating microscope, both of which promote the adoption of a forward head posture (FHP).

Implications and Prevention

The most prevalent WMSD affecting ophthalmologists is cervical spine disease. Over time, the repetitive loading of the spine leads to chronic and permanent degenerative changes in the C-spine.[3,4] According to the literature, WMSDs can lead to career-ending and career-interrupting injuries, reduced work productivity, surgical practice restriction, and early retirement.[5,6] Those with smaller anthropometrics, female ophthalmologists, and any ophthalmologists with a high degree of patient care and surgical volumes are at increased risk for WMSD.[5,7] Additional research is needed to develop a systems-based approach to modify WMSD risk factors in ophthalmology.

WMSD prevention requires an understanding of the contributing factors. The iceberg model is an effective representation of work-related injury in the context of "hidden" factors that lead to injury (**Figure 1**). On the surface, we see the consequences of WMSDs: time away from work, cancelled clinics and surgeries, disruption to on-call and teaching duties, and work modifications related to WMSD symptoms. What we don't see is the

contribution of underlying factors, professional culture, limitations of the healthcare system, and the equipment we use on a daily basis. Individual factors that predispose to injury include a lack of awareness of WMSDs. Cultural norms in medicine contribute to our reluctance to discuss pain and injury. As a result, ophthalmologists have not advocated for themselves or educated their peers and trainees about this threat to career longevity. Our healthcare institutions and systems have not been resourced to support physician wellbeing. The equipment and devices we use are not conducive to comfort and neutral working postures. The industry has lacked the input of ophthalmologists regarding ergonomic design engineering.

WMSD Prevention Strategies

Whether the goal is to prevent WMSDs or to facilitate a healthy and sustainable return-to-work following injury, the strategies are similar (**Figure 2**). The individual's personal efforts must be supported by the workplace and institution leadership. Ergonomic assessments and guidance from a professional ergonomist are central to injury prevention and successful return-to-work. Adoption of ergonomic accommodations and best practices enable clinicians to be productive, work with less pain and reduce injury risk. Normalizing discussion related to wellbeing and injury prevention encourages advocacy, education and collaboration with healthcare systems and industry partners to promote the health of the entire ophthalmology workforce. When developing primary or secondary WMSD prevention strategies, supportive measures that foster healthy workplaces must be part of the strategy.

Ergonomics, Injury Prevention and Return to Work after Injury: My Personal Prescription

"Ergonomics is the process of designing or arranging workplaces, products and systems so that they suit the professionals who use them. This is in sharp contrast to the typical hospital design that assumes that one size fits all." [8]

Having had some time to reflect on my own injury and return to work experience, I have conceptualized a three-step prescription for WMSD prevention.

STEP 1: Acknowledge the ergonomic challenges

STEP 2: Consult experts

STEP 3: Adopt ergonomic best practices and advocate for ergonomic improvements

STEP 1:

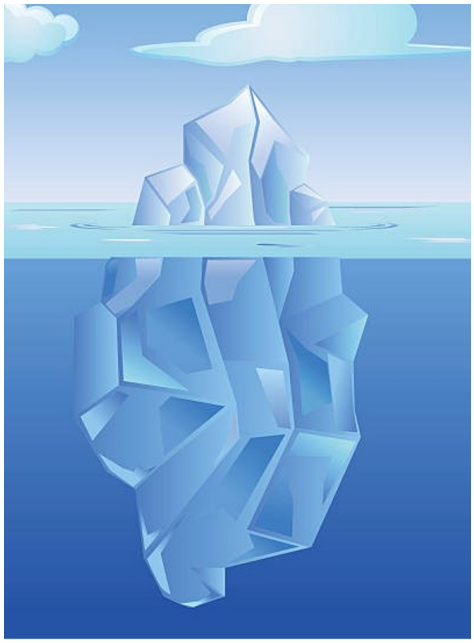
First, we need to stop accepting pain and acknowledge the ergonomic challenges inherent in ophthalmology practice. Awkward postures and discomfort associated with work tasks tend to be ignored because we are hyper-focused on efficiency. From the literature, we know this type of work ethic is not sustainable as up to 93% of us will experience WMSD. Once we acknowledge WMSD risk, we can benefit from the experts who have the knowledge and skills to help us avert injury and perform at our best.

STEP 2:

A professional ergonomist will identify high-risk postures and movements that may lead to injury. The ergonomist will recommend strategies to correct maladaptive postures and work practices. In my case, the ergonomist identified that I was adopting an extreme FHP while performing slit lamp examinations. She explained how FHP limits range of motion, leading to muscle tension and compromised strength, motor function, grip and dexterity.

With the guidance of the ergonomist, I learned how best to position myself and the patient for slit lamp examination. The ideal posture is to sit upright, as close as possible to the patient. The ergonomist suggested that I sit upright at the slit lamp first, then ask the patient sit at the edge of their chair and lean forward into the slit lamp. This scenario prevents the ophthalmologist from leaning forward to reach the oculars. The ergonomist also noted that the footrest on the patient chair extended outward and prevented my stool from getting close enough to the patient. Flipping the footrest up allows the ophthalmologist's stool to roll closer to the patient, promoting a more upright working posture.

Ergonomists provide individualized strategies to prevent injury, including postural modifications, workflow improvements and better placement of equipment in the workspace. Recommendations regarding adjustable desks and chairs, workstations, and padded elbow and wrists rests



Injury
Disability, unplanned absence(s), cancelled ORs, cancelled clinics, changes in practice profile, diminished capacity to take on extra patient volume

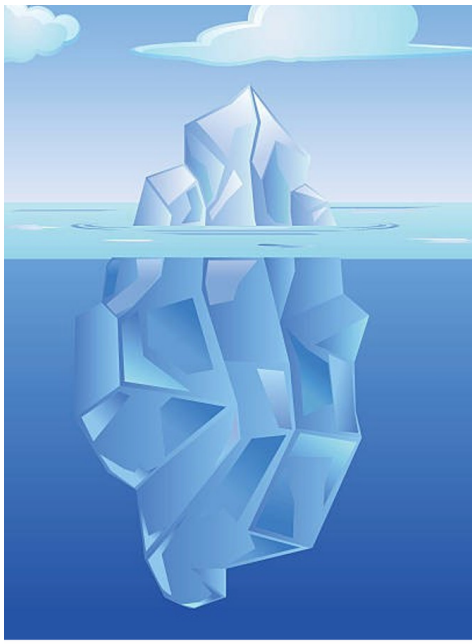
Individual Factors: lack of awareness about injury risk & prevention anthropometrics, genetic predisposition, practice profile, prior MSK injury

Medical Culture: "pain is just a part of the job", hidden curriculum discourages open discussion, lack of formal education on ergonomics & injury prevention

Healthcare System: lack of investment in ergonomic optimization, human resource shortages, wait list pressures, healthcare systems do not prioritize physician wellness

Medical Equipment And Devices: not ergonomically optimized

Figure 1. Factors leading to work-related injury; *courtesy of Rookaya Mather, MD, FRCSC.*



Return to Work & Injury Prevention

Individual Efforts: treatment, rehabilitation, graduated return to work & long-term efforts to optimize endurance & **avoid re-injury**

Culture Shift: psychological safety to discuss ergonomic challenges, workplaces that encourage gradual return to work & facilitate ergonomic accommodations & support injury prevention

Healthcare Systems: investment in ergonomic optimization & human resource planning, ergonomic accommodations for injured physicians, regular ergonomic assessments & equipment upgrades

Equipment Industry: user-centric equipment designed with input from ophthalmologists

Figure 2. Return to work and injury prevention; *courtesy of Rookaya Mather, MD, FRCSC.*

are often indicated to reduce muscle strain, fatigue and nerve compression while working.

Most hospital occupational health departments employ professional ergonomists or occupational therapists who perform ergonomic assessments of physicians and trainees at no cost. Privately hired ergonomists can be engaged by physicians to conduct such assessments in the clinic, office and the OR. The value of an ergonomic assessment is that you are assessed while you are working and interfacing with patients and equipment. You will learn how to adopt ergonomic best practices as it pertains to your own workflow and anthropometric characteristics. Ergonomist consultations are also valuable to organizations wishing to improve safety and work efficiency. The adoption of ergonomic best practices and ergonomically optimized equipment in ophthalmology can improve productivity, safety and quality of care.

Equipment specialists are experts in equipment options, accessories and modifications. These experts can determine how existing equipment can support better working postures as determined by the ergonomist. In my case, the ergonomist identified extreme FHP during slit lamp examinations. She suggested longer oculars to avoid leaning forward and craning my head to look through the eyepieces. Unfortunately, longer oculars are not available. My slit lamp representative suggested two options that would help reduce FHP: a beam splitter (**Figure 3**); or a 20-degree inclined eyepiece adaptor (**Figure 4**). Both options add approximately 5 cm to the length of the oculars, thereby reducing FHP and improving neck posture. The 20-degree inclined adaptor requires the user to raise their stool height to view through the oculars. Viewing downward through the oculars places the head and neck in a more neutral posture. The beam splitter, on the other hand, reduces FHP without inclining the oculars so one can sit without having to raise the stool height. Both of these are effective options. For the operating microscope, the biomedical equipment specialist at my hospital suggested I use longer and more adjustable oculars for surgery. He sourced and ordered adjustable oculars, which I trialed with the ergonomist present. The ergonomist determined that my posture was significantly better, and I felt more comfortable at the end of my surgery day. Finally, investing the time to adjust equipment before use can seem time-consuming and inefficient; however, the three seconds it takes to become comfortable has reduced the daily strain for me.

Physiotherapists address maladaptive postures and help develop postural awareness through physical conditioning. After injury, the goal of rehabilitation is to restore functional ability and quality of life, while "prehabilitation" involves maintaining optimal functioning and performance to prevent injury and disability. Both involve promoting neutral body posture through strength and flexibility training and stretch therapy to offset work-related musculoskeletal (MSK) strain. Targeted neck and core strengthening can help to relieve posture-related fatigue and strain by stabilizing muscles. Both rehabilitation and prehabilitation can protect career longevity and reduce the risk of (further) MSK injuries. Engaging in a 20-minute supervised resistance training program performed three times a week can reduce pain and improve performance in as little as one month. Other forms of exercise such as pilates, yoga, dance and targeted stretching can counteract maladaptive postures. As well, the literature supports adopting stretch breaks during clinic and the OR to offset muscle fatigue and pain.[9] Physiatrists and family physicians are important experts to consult, particularly when physiotherapy does not alleviate symptoms or when neurological deficits and symptoms require further referrals.

STEP 3:

Adopting ergonomic best practices includes all measures that benefit your wellbeing as you deliver the best possible patient care. Measures that increase comfort, reduce strain and fatigue include:

- Optimizing equipment and workflows
- Optimizing postures and engaging in regular physical conditioning
- Integrating ergonomic "time-out" in the OR and microbreaks to stretch between patient examinations and procedures

The process of ergonomic optimization is iterative and requires multiple trials and assessments. Even simple changes to equipment, postures and workflows may require a substantial learning curve as old habits are difficult to change. **Figure 5** presents an ergonomic guide for ophthalmologists developed by Dr. Rishi Gupta, serving as a visual cue to correct posture, move, stretch and breathe.

Figure 3. A beam splitter effectively "extends" the oculars toward the user; *photo sourced from Haag Streit USA; https://innovamed.com/products/haag-streit-50-50-beam-splitter-bq/*

Adjusting the viewing angle 20° on the slit lamp allows the physician to maintain a more natural neck position.

Figure 4. Split lamp inclined eyepiece adaptor; *photo sourced from https://products.haag-streit-usa.com/wp-content/uploads/2020/05/HS-ErgoWhitePaper.pdf*

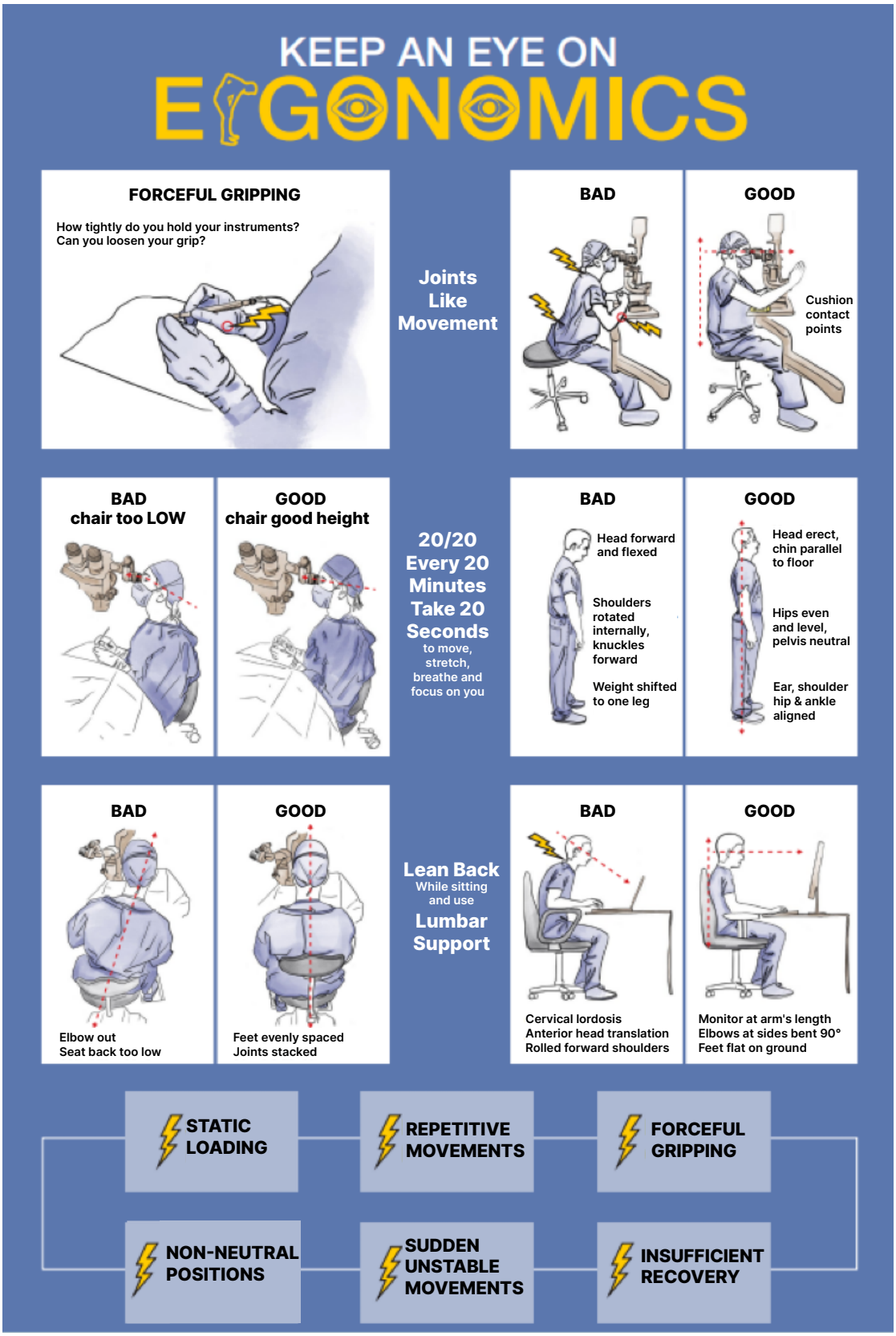


Figure 5. Ergonomic best practice guide for ophthalmologists; *courtesy of R. Rishi Gupta, MD.*

Canadian Ophthalmological Society Ergonomics Working Group

Advocating for ergonomic improvements involves engaging with colleagues, trainees, leaders and professional organizations. Raising awareness about injury risk and ergonomics is the first step in bringing about systems changes. The recently established Canadian Ophthalmological Society (COS) Ergonomics Working Group seeks to educate every Canadian ophthalmologist on ergonomics and injury prevention, as depicted above. The Ergonomics Working Group is currently developing a major initiative to promote ergonomic awareness and injury prevention at the COS conference in 2025.

Conclusion

We all can be ergonomics champions in our practices, clinical departments and professional societies to advocate for institution- and system-level ergonomic improvements to enhance quality and safety for all.

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