LEARNING OBJECTIVES

• Special Focus
A comprehensive overview on glaucoma in the setting of inflammatory diseases including incidence and prevalence, pre-dispositions and underlying diseases, pathophysiology and pathomechanisms.

• What’s New
A review of current principles of medical and non-medical management of glaucoma in dry eye patients including a review of the newest clinical data.

• Clinical Issues
A summary on detecting and diagnosing pediatric uveitic glaucoma, current treatment approaches and management of complications such as secondary glaucoma.

• Practical Tips
A practical lesson to differential diagnosis and treatment measures taken for specific entities such as Sjögren’s syndrome, Posner Schlossmann Syndrome and Fuchs Syndrome.

Main topic:
“GLAUCOMA AND CONCOMITANT EYE DISEASES”

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In the developed world, ocular inflammation is one of the common causes of preventable visual disability and blindness. Recent advances in the medical treatment of ocular inflammation has dramatically improved the visual outcome for an increasing number of patients. Glaucoma remains a major complication: visual prognosis depends more on the control of ocular pressure (IOP) than on the inflammation. The best way to avoid uncontrolled glaucoma is to prevent its occurrence, mainly by rapid and aggressive inflammation treatment and by minimizing the use of corticosteroids.

I- Incidence and prevalence

The epidemiology of inflammatory glaucoma is limited to studies, many of which are retrospective with missing details. Differences between transitory ocular hypertension (OHT) and glaucomatous optic neuropathy are rarely highlighted. Results from tertiary centers are biased by a higher proportion of more severe cases than in those managed in community-based ophthalmology practice.

Scleritis is a rare inflammatory eye disease\textsuperscript{1} associated with secondary glaucoma. Heinz et al. have reported on the incidence of OHT and glaucoma in patients with scleritis.\textsuperscript{2} 271 patients were included in this single-center, retrospective study. Even though the median follow-up was limited, IOP increased in 56 patients with an open angle with angle-closure in 4 patients. The necrotizing form was the main type of scleritis associated with high IOP, mainly observed during acute episodes of inflammation. The diagnosis of OHT was documented in 72\% of patients at referral but the rate decreased to 56\% at the end of the follow-up. The presence of anterior uveitis, peripheral ulcerative keratitis, posterior synechiae, and previous cataract surgery were the major risk factors for OHT and glaucoma in this cohort.

Uveitis is more common than scleritis. Macular alterations and glaucoma are two irreversible sight-threatening complications that appropriate management could prevent.\textsuperscript{3} Before the era of biologic agents, approximately 10\%-20\% of uveitis patients used to develop glaucoma in Western countries. In a series of 927 patients, we have identified a higher risk of OHT or secondary glaucoma in the subgroups of anterior or panuveitis.\textsuperscript{4} A more recent study from Thailand has shown a secondary glaucoma prevalence of 29\%.\textsuperscript{5} Age at onset of uveitis above 60 years and longer duration of ocular inflammation were two risk factors. The prevalence of patients with at least 1 blind eye was significantly higher in those who developed secondary glaucoma than in uveitis patients without glaucoma (P=0.001).

Other ocular inflammatory conditions have a lower prevalence of OHT. Topical cyclosporine significantly lowers the risk of steroid induced glaucoma in patients with ocular surface inflammation.

II- Pre-dispositions and underlying diseases (e.g. glaucoma as immunogenetic inflammatory component)
Uveitis and scleritis are the main entities to consider. B27-association is the most frequent etiology for anterior uveitis. Interestingly, in patients with unilateral acute anterior uveitis, IOP is usually lower than the fellow eye, perhaps related to a transient paralysis of the ciliary body. Many glaucoma-related uveitides are caused by infection. Their prevalence remains underestimated. Analysis of ocular fluids after anterior chamber tap or vitrectomy has dramatically modified the diagnostic and therapeutic management of patients with unilateral inflammatory OHT or secondary glaucoma.[6]

### A- Infectious uveitis

#### 1- Viral infections

Uveitis is unilateral in most of cases, even though 10% may affect both eyes. Anterior uveitis or keratouveitis are the principal presentations, but rarely panuveitis may also be associated with an acute retinal necrosis and OHT.

**- Herpetic anterior uveitis including Posner Schlossman syndrome**

Viral anterior uveitis is the second most common etiology (Table 1). Secondary glaucoma occurs in up to 54% of these patients.[7] Herpes simplex virus (HSV), varicella zoster virus (VZV) and cytomegaly virus (CMV) based anterior uveitides are usually acute, unilateral and granulomatous. Keratic precipitates (KP) may guide the diagnosis but they are non-specific (Figure 1). Sectoral iris atrophy (Figure 3) is classically found in all 3 entities even though early antiviral treatment may avoid its occurrence or extension. Posterior synechiae are absent in CMV anterior uveitis. Chronic anterior uveitis and Posner Schlossman syndrome are the two main presentations of CMV uveitis in immunocompetent patients (Figure 2).[8] More information on Posner Schlossman can be found in the Practical Tips Section. We have recently reported on the clinical characteristics and long-term outcome of CMV anterior uveitis.[9] In this series of 35 patients, keratic precipitates and iris atrophy were seen in 91.4% and 25.7% of cases, respectively. At baseline, mean IOP was 29 mmHg. Recurrences were reported in 73.5% of cases and glaucoma surgery was necessary in one out of four cases. Early initiation of antiviral therapy (<700 days) seemed to decrease the risk of glaucoma surgery. Viral scleritis is less frequently associated with OHT than uveitis. HSV and VZV are the main causative agents.

**- Rubella virus infection**

Fuchs’ uveitis, first described in 1906 represents 10 - 15% of all anterior uveitis. Anterior granulomatous uveitis with small diffuse stellate KP, iris heterochromia (Figure 4) and cataract are major diagnostic elements. Heterochromia is variable depending on the intensity of the inflammation, the initial iris color and amount of pigment in the iris pigment epithelium. Iris nodules and vitritis may be observed but posterior synechiae and macular edema are excluding criteria. Since the early 2000s, Fuchs’ uveitis has

<table>
<thead>
<tr>
<th></th>
<th>HSV</th>
<th>VZV</th>
<th>CMV</th>
<th>RUBELLA VIRUS</th>
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<tbody>
<tr>
<td>Previous keratitis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
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<tr>
<td>Scleritis</td>
<td>++</td>
<td>++</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Keratic Precipitates</td>
<td>Brown in Art’s triangle</td>
<td>Brown in Art’s triangle</td>
<td>White central in PSS or Brown in CAU</td>
<td>White-gray, Stellate, diffuse</td>
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<tr>
<td>Posterior synechiae</td>
<td>+</td>
<td>+</td>
<td>-</td>
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<td>Heterochromia</td>
<td>-</td>
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<tr>
<td>Iris atrophy</td>
<td>Sectoral</td>
<td>Sectoral</td>
<td>Sectoral (rare)</td>
<td>Diffuse</td>
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<td>Iris nodules</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
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<tr>
<td>OHT at presentation</td>
<td>++</td>
<td>++</td>
<td>+++</td>
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<td>Vitreous cells</td>
<td>+</td>
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<td>Chorioretinal scars</td>
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Table 1. Clinical characteristics of major viral ocular inflammatory entities involving the anterior segment and inducing OHT or glaucoma.
been strongly associated with rubella virus infection.[10-12] Topical corticosteroids have no efficacy on this viral-induced inflammation and are often the cause of OHT. Disease progression or misuse of topical corticosteroids may lead to glaucoma in 20 to 60% of cases. In the absence of anti-rubella drugs, the use of corticosteroids must be avoided. Molecular analysis of the aqueous humor and a few clinical signs may be predictive elements of secondary glaucoma. Additional information on the management of Fuchs’ corneal dystrophy may be found in the Practical Tips section.

2- Other infectious diseases

Toxoplasmic retinochoroiditis may induce OHT. The peripheral location of lesions and their size are more frequently associated with a severe panuveitis and OHT. Ocular pressure is rapidly normalized with antibiotics and systemic corticosteroids. Tuberculous uveitis or scleritis may involve the anterior segment with large KP, extensive posterior synechiae and different types of granulomas on the iris or the trabeculum. OHT is commonly observed in severe anterior inflammation from tuberculosis (TB), due to different pathophysiologic mechanisms, with significant ocular morbidity.[13] Syphilitic uveitis is another granulomatous anterior uveitis in the secondary or tertiary phase of infection. All three conditions require a prompt diagnosis before specific therapy. Topical corticosteroids may be helpful in association with specific antibiotics. Their short-term use explains the absence of OHT or glaucoma secondary to steroids.

3- Two special entities: BADI and BAIT

Bilateral acute depigmentation of the iris (BADI) and bilateral acute iris transillumination (BAIT) are two recently described entities with anterior segment inflammation and OHT. BADI is characterized by acute onset of depigmentation of the iris stroma. Iris pigment is deposited heavily in the iridocorneal angle, decreasing aqueous outflow.[14] OHT is usually transient. A case of simultaneous BADI in 2 siblings has been recently reported.[15] On the other hand, BAIT patients present with iris transillumination and a mydriatic atonic pupil, even though the pathophysiology of OHT and secondary glaucoma remains similar. Recent systemic antibiotic (fluoroquinolone) use or a prior upper respiratory tract infection are frequently reported prior to BAIT onset. Damages are more frequent in BAIT patients with a higher rate of secondary glaucoma.

B- Noninfectious uveitis

1- Juvenile idiopathic arthritis-associated uveitis (JIAU)

Secondary glaucoma is one of the most severe complications in children with JIAU.[16] More information on
epidemiology, etiology and treatment of pediatric uveitic glaucoma may be found in the Clinical Issues section. It should be added that laser flare photometry is useful for the management of severe cases as it may provide a more quantitative and reproducible evaluation of anterior chamber flare, avoiding overtreatment with topical steroids.

2- Sarcoidosis

Both uveitis and scleritis may be associated with sarcoidosis. Ocular disease may occur without detectable systemic involvement. Prevalence ranges widely in different geographic areas.[17] Diagnostic criteria have been recently revised.[18] Granulomatous nodules on the trabecular meshwork and/or tent-shaped peripheral anterior synechiae may explain OHT and secondary glaucoma. In a series of 88 biopsy-proven sarcoid patients, ocular disease was present in 36.4% of cases and the prevalence of glaucoma has been estimated at 5.7%.[19]

III- Pathophysiology and pathomechanisms

Understanding the pathophysiology of OHT or secondary glaucoma in patients with ocular inflammation is challenging but remains necessary to determine an appropriate therapeutic strategy.

Angle closure glaucoma may be missed, masquerading as an acute anterior uveitis with OHT. It is vital to examine the angle and use tests like anterior segment ultrasound, if necessary. Posterior synechiae can point to previous episodes of angle closure. Appropriate laser or surgical treatment may avoid irreversible damage.

Uveitic glaucoma is caused by four principal mechanisms. Open angle glaucoma is the most common form. Trabecular meshwork blockade due to inflammatory cells, fibrin, proteins and other debris is the main reason for increased resistance to aqueous outflow.[20] Protein accumulation in the aqueous humour (AH) follows a disrupted blood-aqueous barrier. Permanent damage to trabeculum varies among patients and depends on the type of uveitis.

A second mechanism for decreased aqueous outflow is steroid responsiveness, aggravating other damage. Corticosteroids response occurs in up to one third of patients;[21] while it usually develops 2 - 6 weeks after initiating therapy, it may occur later in the course of the disease. Prevalence of steroid responsiveness seems higher in the group of patients with uveitis compared with the general population. Perhaps this is due to an already altered trabecular meshwork and a cumulative effect. Extremes of ages are more frequent steroid responders. The type of steroid molecule and its dosage are relevant.

Secondary angle-closure glaucoma is rarer but significant mechanism. Pupillary seclusion or block happens when chronic insidious inflammation has not been diagnosed rapidly and/or not treated appropriately. Sarcoidosis, tuberculosis, JIAU and chronic Vogt-Koyanagi-Harada disease are at high risk of pupillary seclusion, block and iris bombé (Figure 5). Inflammatory induced anterior
Synechiae can also result in total closure of the angle. Finally, transient anatomic alterations observed during Vogt-Koyanagi-Harada disease such as ciliary body rotation may induce non-pupillary block angle closure glaucoma. Posterior scleritis may be associated with angle closure glaucoma. Diagnosis is easily made due to presence of choroidal thickening, sub-Tenon effusion and the classical "T" sign observed on ultrasonography.

Finally, neovascular glaucoma may occur in uveitis patients with episodes of extensive retinal vein occlusion associated with different types of disease such as severe Behçet's disease, sarcoidosis and idiopathic retinal vasculitis. Careful examination of the iris and chamber angle is mandatory; it guides developing an appropriate therapeutic approach.

IV - Conclusion

Inflammatory diseases have multiple causes and mechanisms, which may change during the course of management. A multi-disciplinary approach to diagnosis and treatment achieves optimal management strategies for the underlying inflammation and the superimposed glaucoma.

References:


What’s New:

Principles of medical and non-medical management of glaucoma in dry eye patients

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Core Concepts

- The ocular surface is frequently damaged in glaucoma patients on long term topical medical treatment
- Active compound, preservative and individual patient sensitivities may interact to cause or aggravate ocular surface disease (OSD)
- The preservative benzalkonium chloride (BAK) may play an important role in dry eye through its own chemical properties
- The number of BAK-containing eyedrops instilled each day is a significant risk factor for failure in glaucoma surgery
- In patients with OSD it is advisable to use preservative-free or at least BAK-free eye drops
- Removal of the causative compound(s) is more effective than adding eye drops to counteract OSD (subtractive preferred to additive strategy)

I- Ocular surface changes in glaucoma

Both basic science and clinical research demonstrate frequent and significant ocular surface changes from long-term eye drop use.1 In patients treated chronically with topical glaucoma medications, signs of low-grade, chronic inflammation occur, with various clinical manifestations. Such medications, often used for decades in clinical practice, may cause and/or exacerbate pre-existing ocular surface disease (OSD), such as dry eye, meibomian gland dysfunction, and chronic allergy, further depressing quality of life, adherence, and surgical outcomes. Several consistent observational studies on the ocular surface in glaucoma patients highlight a much higher prevalence of OSD than that found in the general population.2-4 Signs and symptoms of OSD are observed in 35–50% of glaucoma patients, according to the criteria analyzed, which is substantially more common than could be expected from dry eye prevalence.5 An observational study confirmed a prevalence of OSD in glaucoma patients reaching 35-40%, with 35% of patients repeatedly using tear substitutes adjunctively. Interestingly, this study also evaluated those patients suffering from OSD (dry eye or allergy) prior to the initiation of antiglaucoma treatment. As expected, the prevalence was 10 to 15%, which is consistent with dry eye diagnosis in the general population.6 The difference is therefore that induced by glaucoma therapy, which points out the importance of drug-induced OSD. In 2017 The Dry Eye WorkShop II emphasized the role of medical or surgical interventions in inducing dry eye, and dedicated a chapter to iatrogenic causes of dry eye.7 The importance of topical treatments, and especially the role of preservatives in inducing OSD deserved special attention (Figure 1).

II- The role of preservatives in ocular surface disease

Several studies have correlated signs and symptoms of OSD with the number of concomitantly used eye drops containing benzalkonium chloride (BAK), the most frequently used preservative in eye drops.2,3 Signs and symptoms significantly improved with discontinuation of the BAK-preserved drops and substitution with non-preserved drops.2,4 BAK impairs the tear film because of its surfactant properties (Figure 2). It also has proinflammatory, and cytotoxic properties for epithelial and goblet cells1,9 and may cause increased tear hyperosmolarity.10 Its use in dry eye patients therefore may aggravate ocular surface disorders. This is why most tear substitutes available worldwide have been developed as preservative-free or BAK-free formulations.
Interestingly, this can be modeled in vitro, where BAK’s deleterious effects were significantly increased when conjunctival cells were stressed osmotically.11 This process may mimic the enhanced toxicity of the preservative in dry eye patients, compared with normals.

Through the loss of its protective properties, an impaired tear film provokes dry eye symptoms with corneal damage and convey cytotoxic inflammatory mediators across the ocular surface. Tear film alterations may even stimulate a series of biological changes in the ocular surface, leading to subsequent neurogenic inflammation and further impairment of the tear film, actually creating a vicious circle.12

BAK has also been shown to be neurotoxic13, causing corneal hypoesthesia and nerve damage. In a study comparing the effect of various antiglaucoma treatments on corneal nerves, abnormal nerve morphology and corneal hypoesthesia were consistently found in patients receiving preserved compared with unpreserved medications.14 BAK’s nerve-damaging effects could contribute to the overall false comfort of patients receiving BAK-containing eyedrops, with corneal hypoesthesia masking corneal damage and explaining a misleading apparently “good” tolerance. Indeed tolerance of glaucoma therapy is usually considered as quite good, despite a high rate of dry eye disease, tear instability and meibomian gland dysfunction in glaucoma patients. BAK was already identified as delaying corneal wound healing and should thus be avoided in patients with damaged cornea.15,16

### III- Glaucoma in dry eye patients

Dry eye disease is quite prevalent in the elderly population, frequency ranging between 10 and 30%, depending on the criteria considered.3 Therefore glaucoma may occur in significant numbers of patients suffering from OSD: a retrospective case series showed the incidence of glaucoma was 20.4%, ranging from 13.6% to 60% in patients with coinciding OSD.17 In these patients, it is advisable either to avoid preservative-containing IOP-lowering agents or to limit their use.

When dry eye has progressively developed over time with antiglaucoma treatments, switching to preservative-free medications should be the first choice, rather than adding tear substitutes or anti-inflammatory agents. This would emphasize a “subtractive strategy”, which is likely more effective, because it targets the origin of the disease or one of its co-factors, rather than an “additive strategy” consisting of adding medications to counteract the iatrogenic effects of other drugs. Data from studies where such switches have taken place suggest reversible drug-induced ocular surface changes, which may improve when the causative or aggravating factor is removed3 or even just decreased.3 However, removal of an aggravating factor may not be sufficient to treat dry eye associated with glaucoma. Specific dry eye therapy may be necessary, such as preservative-free tear substitutes, osmoprotectants or topical cyclosporine, to counteract inflammatory reactions often associated with dry eye. In aqueous-deficient dry eye, punctual plugs may be helpful, but it is advisable to minimize or remove potentially toxic compounds that would be retained on the ocular surface. In addition, surface inflammation as well as toxic compounds may diffuse from the surface into deeper ocular structures, like subconjunctival tissue, which may negatively influence the outcome of glaucoma surgery, as suggested by Broadway et al. as early as in the 90s, and confirmed by the PESO study that clearly identified the number of BAK-containing eyedrops instilled each day as a significant risk factor of failure.18,19 Moreover BAK may penetrate to the trabecular meshwork and there exert toxic and/or proinflammatory effects.20 Specifically treating the ocular surface and removing toxic compounds not only reduced surface inflammation but also reduced intraocular pressure; such inflammation could cause an iatrogenic trabeculitis.21,22

### IV- Which patients may benefit most from preservative-free drugs and how to identify them?

Not all patients are sensitive to preservatives and not all side effects observed with antiglaucoma medications are induced by preservatives. Three factors must in fact be considered: the active compound, the preservative, and the...
patient’s ocular surface. Patients with existing OSD when glaucoma treatment is initiated, as well as those developing dry eye or ocular irritation during glaucoma therapy, should be identified and receive particular attention. Simple clinical tests may help the clinician detect ocular surface disease, such as assessment of symptoms of irritation or dryness, eyelid margin redness, positive corneal and conjunctival fluorescein staining, and rapid tear film break-up time.

In such cases, quality of life, adherence, surgical outcome, and overall glaucoma care may be adversely affected. Consider treatment alternatives, such as removing the preservative when possible or at least decreasing the number of preserved eyedrops by using fixed combinations or using medications with preservatives other than BAK, treating the ocular surface with unpreserved tear substitutes, and considering laser trabeculoplasty or surgery to decrease the number of eyedrops.

**V- Conclusion**

Definitively glaucoma should be considered a high risk for ocular surface disease that deserves specific attention so as to manage safely and effectively this sight-threatening disease for the long-term.

**References:**


I- Introduction
Paediatric uveitis is most commonly idiopathic, followed closely by uveitis associated with juvenile idiopathic arthritis (JIA)\(^1\). Approximately 12-38% of children with JIA develop uveitis (JIA-U)\(^2\). There are a number of arthritides associated with paediatric uveitis and subsequent secondary uveitic glaucoma. These are highlighted in Tables 1 and 2. Initial screening of a child diagnosed with these subclasses should be within 6 weeks of referral followed by two monthly intervals from onset of arthritis for 6 months. Then all children should be screened until 11 years of age irrespective of the subclass of diagnosis\(^3\).

Management of paediatric uveitis poses many challenges. Despite severe inflammation, children may remain asymptomatic or have difficulty expressing their symptoms. They often present with advanced disease, which can lead to a lifetime of visual impairment and amblyopia\(^4,5\). Vision loss in paediatric uveitis is more common than in adults. Structural complications from the disease include inflammatory mediated (cystoid macular oedema and optic disc swelling) and non-inflammatory mediated (elevated IOP/glaucoma/glaucomatous optic neuropathy/cataract/band keratopathy/posterior synechiae/hypotony). These can be

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### Core Concepts
- Paediatric uveitis is rare, 5-10% of the uveitis burden
- Although many cases are idiopathic, 41–67% of uveitis in children is linked with Juvenile Idiopathic Arthritis (JIA) and 3–6% with Sarcoidosis
- Systemic management of the intraocular inflammation is crucial to reduce the vision threatening complications of paediatric uveitic glaucoma
- While screening guidelines remain the same the 2018 EULAR guidelines and the Sycamore trial highlight multidisciplinary care and use of biologics to control disease to reduce the incidence of secondary uveitic glaucoma
Table 2 - AAO Paediatric guidelines for screening eye examinations

<table>
<thead>
<tr>
<th>JIA</th>
<th>Subtype risk of iritis</th>
<th>Examination frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oligoarticular or polyarticular, onset &lt; 7 years of age and ANA +ve</td>
<td>High risk</td>
<td>Every 3-4 months</td>
</tr>
<tr>
<td>Oligoarticular or polyarticular, onset &lt; 7 years of age and ANA +ve</td>
<td>Medium risk</td>
<td>Every 6 months</td>
</tr>
<tr>
<td>Oligoarticular or polyarticular, onset &lt; 7 years of age and ANA +ve</td>
<td>Medium risk</td>
<td>Every 6 months</td>
</tr>
<tr>
<td>Oligoarticular or polyarticular, onset &lt; 7 years of age and ANA +ve</td>
<td>Low risk</td>
<td>Every 12 months</td>
</tr>
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</table>

Figure 1 - An approach to the management of Paediatric uveitic glaucoma adapted from Joos et al 2013.
present at diagnosis or develop during the chronic course of the disease. Children appear to be more susceptible to these structural complications and up to 20% develop glaucoma.

Paediatric uveitic glaucoma is challenging because of the multifactorial nature of the elevated IOP (Figure 1). Medical treatment should be commenced early with any elevated IOP, even if the optic discs are normal. Glaucoma surgery is often required: options include trabeculectomy, glaucoma drainage devices and cyclotherapy (rarely, as it is pro-inflammatory). To reduce complications, it is important to operate on a ‘quiet eye’. Surgical risks include: increased inflammation, cataract, corneal decompensation, ciliary body “shutdown”, retinal detachment, cystoid macular edema, choroidal effusion, flat anterior chamber, encapsulated bleb, extruded glaucoma drainage device and infection. Hypotony is a common problem in paediatric uveitic patients post glaucoma surgery even in the absence of over-drainage.

II- What is new?

1. Recent EULAR guidelines and randomised control trials highlight the need for multi-disciplinary clinics, with open communication between ophthalmologists (paediatric, glaucoma and uveitis) and the paediatric rheumatologist concerning changes in disease activity, adjustments or escalation of any treatment.

2. The Sycamore study was a randomised control trial comparing 90 children with JIA uveitis treated with either Adalimumab and Methotrexate versus Methotrexate alone. The trial was stopped after one year of recruitment with a difference in relapse rate 27% Adalimumab and Methotrexate versus 60% Methotrexate alone. Biologics are the next step being increasingly used when uveitis is poorly responsive to systemic immunomodulating drug therapy.

3. Glaucoma surgery should be conducted prior to cataract surgery, as there is often increased inflammation after cataract surgery, requiring increased short-term steroid that subsequently can increase the IOP. If trabeculectomy is performed, tight scleral flap sutures are mandatory. Non-flow restricted glaucoma drainage devices (GDD) should have an intraluminal stent in addition to an extraluminal occlusive suture (e.g. 6/0 Coated polyglactin 910 Ethicon USA), to reduce the risk of post-operative hypotony. We recommend a Baerveldt tube 350mm (Johnson & Johnson Vision USA) independent of age to reduce the risk of the child outgrowing the tube and a 3.0 nylon intraluminal stent to reduce flow and risk of hypotony (see Figure 2).

4. We advocate for an IOL during cataract surgery especially when there is adequate systemic immunosuppression and the child has been free from inflammation for >3 months. This reduces the risks of both amblyopia and aphakic glaucoma. An intravitreal triamcinolone injection (2mg) at time of surgery improves post-operative inflammatory control without the need for additional systemic steroids. Heparin 1 Unit per ml of Balanced Salt Solution (BSS) in the infusion line further reduces inflammation in the early post-operative period.

III- Conclusion

Managing uveitic glaucoma in children is challenging. Adequate immunosuppression is crucial in the management of these children. This means early recognition of poor control with topical agents with institution of systemic steroid sparing agents.

References:


5. Jabs DA, Nussenblatt RB, Rosenbaum JT. Standardization of uveitis nomenclature for reporting clinical data. Results of the First


Practical Tips:
Glaucoma and concomitant eye diseases
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Core Concepts
• In ocular surface disease such as Sjogren’s syndrome the treatment of glaucoma can further compromise a patient’s quality of life
• To improve outcomes for your patient with ocular surface disease and glaucoma, avoid preservatives in and reduce the number of topical medications
• Ocular inflammation and/or its treatment with steroids, can increase intraocular pressure leading to glaucoma
• Patient’s with Posner Schlossman syndrome, suffer with recurrent attacks of increased intraocular pressure and inflammation, long-term monitoring is needed due to the risk of glaucoma
• In Fuch’s corneal dystrophy eventual failure of the corneal endothelium is most commonly managed with an endothelial keratoplasty. This procedure can be performed in patients with glaucoma, including those with drainage devices, but in the long-term re-grafting maybe needed

I- Introduction
Glaucoma is the most frequent cause of irreversible preventable blindness worldwide; with increased prevalence in the elderly. Due to its prevalence, other ocular conditions can co-exist with glaucoma or be directly caused or exacerbated by glaucoma or its treatment. When there is co-existent ocular disease, such as dry eye, the patient’s quality of life maybe further reduced. Eye care practitioners need to be aware of the interaction between glaucoma and other ocular conditions to guide management.

II- Sjogren’s Syndrome
In Sjorgen’s syndrome, aqueous tear production is reduced from autoimmune-mediated inflammation of the lacrimal gland. It is typically accompanied by dry mouth, but dry skin and nose may occur. Patients may suffer with fatigue, muscle and joint pains. For the eye, there is a thin marginal tear strip and the effects on the ocular surface are similar to other forms of dry eye with punctate corneal erosions typically present in the palpebral fissure (Figure 1) and inflammation. Due to the presence of ocular surface disease in Sjogren’s syndrome clinicians, when treating co-existent glaucoma, should avoid preservatives in topical therapy and reduce the number of topical glaucoma medications daily, as well as minimize oral Diamox, which can have a drying effect. It may be appropriate to consider non-medical approaches to treatment such as selective laser trabeculotomy, MIGS or drainage surgery.

III- Posner Schlossman syndrome
Posner Schlossman syndrome, also known as glaucomatocyclitic crisis, is a relatively uncommon condition in which acute, recurrent attacks of raised
intraocular pressure and anterior chamber inflammation typically occur in one eye. It should be considered in patients, typically middle-aged Asian men, with repeat episodes of unilateral non-granulomatous anterior uveitis and raised intraocular pressure. It is important to identify as chronic secondary glaucoma can follow and require management, most often with topical therapy, but in some cases systemic therapy or possibly surgery are needed. The diagnosis however is usually clinical although aqueous or blood investigations may be undertaken if infection is suspected. Patients with Posner Schlossman syndrome require long term follow up to identify and manage chronic glaucoma as well as ocular inflammation.3

IV- Fuchs’ corneal dystrophy

Endothelial keratoplasty (EK) has revolutionized the management of corneal decompensation in Fuchs’ corneal dystrophy (Figure 2). As in Fuchs’ corneal dystrophy, corneal oedema occurs gradually due to endothelial cell loss. It is usually bilateral and characterized by deposits on Descemet’s membrane known as guttate. EK offers faster and easier visual rehabilitation, reduced risk of graft rejection, and avoidance of suture related complications such as microbial keratitis, that can follow penetrating keratoplasty. In eyes with a glaucoma drainage device, EK can still be performed with simple adjustments to the surgical technique and acceptable clinical results. Patients should be advised that re-grafting maybe required.4 Nonetheless, repeated EK has a reduced burden of treatment compared with repeat penetrating keratoplasty. If a repeat EK is required, there is emerging evidence that the presence of glaucoma or prior glaucoma surgery may not affect visual outcomes. 5

V- Conclusion

In summary, glaucoma is a burden for many people but it is not just those with glaucoma who may suffer with its consequences. In Sjögren’s syndrome, ocular surface disease can be exacerbated by glaucoma treatments and for patients with Posner Schlossman they may develop glaucoma over time and require monitoring. Lastly, in corneal disease such as Fuch’s corneal dystrophy when surgery is required , glaucoma may develop or require adjustments to the procedure.

References:


Figure 2: Fuchs’ corneal dystrophy is characterized by central corneal guttate and can cause corneal oedema. Note that the endothelium has a typical ‘beaten metal’ appearance.
In order to maximize the learning effect, participants have the opportunity to register at our website and to answer a number of multiple choice questions for each of the four sections covering the key points of each section. Shortly after test completion, participants receive electronic feedback on successful accomplishment or failure. In case of failure the participant is encouraged to review articles and retake the test. A successful test will earn the participant valuable CME credits needed for their continuous medical education efforts.

ACCREDITATIONS
Individual issues of Glaucoma Now are accredited for Continuing Medical Education (CME) by the Physicians’ Chamber of Baden-Württemberg, Germany (Local Medical Responsible: Andreas Buchholz, MD, PhD, R0ph). This accreditation automatically implies acceptance of credits throughout the European Union and associated countries.

Glaucoma Now is also recognized by the Royal Australian and New Zealand College of Ophthalmologists as a valid Continuing Professional Development activity.

Since 2013 the program is recognized by the Brazilian Council of Ophthalmology. Brazilian physicians successfully taking CME tests on our website are automatically awarded CME points by CBO.

STATEMENT OF NEED AND PROGRAM DESCRIPTION
Recent months and years have seen significant advances in our understanding of glaucoma. Much has been learned, not only about damage mechanisms and pathogenesis, but also about diagnosis and management. Treatment options – both medical and surgical – continue to expand. This program will review this new knowledge with an emphasis on incorporating recent insights into day-to-day practice.

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DISCLAIMER
Participants have an implied responsibility to use newly acquired information to enhance patient outcomes and professional development.

The information presented in this activity is not meant to serve as a guideline for patient care. Any procedures, medications, or other courses of diagnosis or treatment discussed or suggested in this activity should not be used by clinicians without evaluation of their patient’s conditions and possible contraindications or dangers in use, applicable manufacturer’s product information, and comparison with recommendations of other authorities.

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