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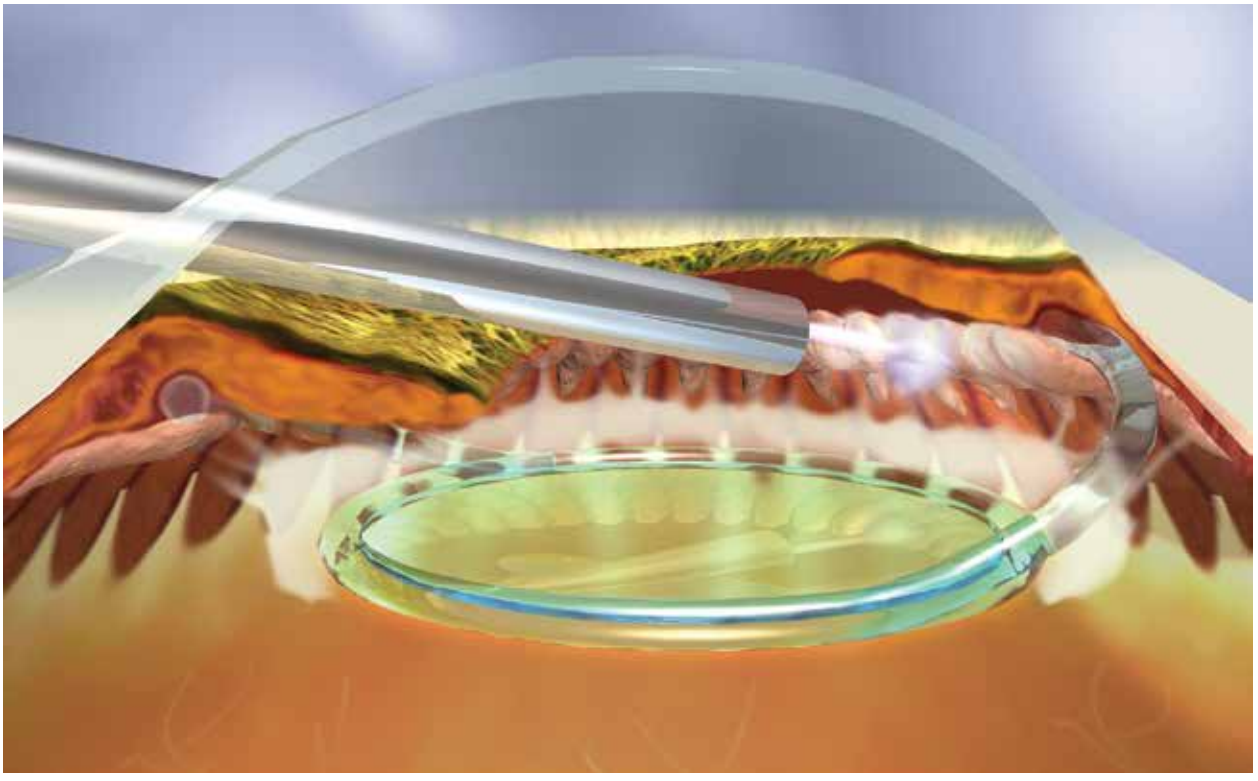

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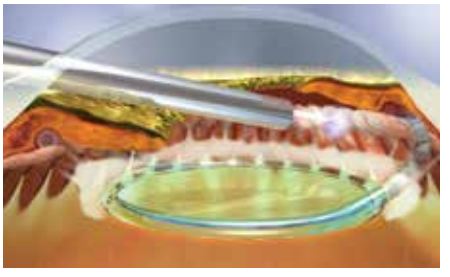

Endoscopic Cyclophotocoagulation of the Ciliary Processes.

Courtesy of Endo Optiks, Inc., Little Silver, New Jersey, USA.

The cover figure for this issue highlights one of the new surgical techniques for controlling glaucoma discussed by Drs. Heckler, Dorey and Damji. It reminded me of how technology has changed all aspects of eye care today.

In many optometric practices, the autorefractor has replaced retinoscopy. Corneal mappers and topographers have supplanted keratometers, and a vast array of imaging techniques has largely consigned direct ophthalmoscopy to a very limited role in assessment of the posterior segment. Recently, liquid crystal lenses have been introduced for occlusion therapy as an alternative to eye patches. The many different types of single vision and progressive addition lenses available to us belies the belief of one of my long-departed colleagues in ophthalmic optics that "optics will never change."

Safe reliable technology that saves chair time and enhances our ability to serve our patients' needs is always a welcome addition to practice, but for it to be used effectively requires the practitioner to understand its fundamentals and its limitations. The curriculum in our Schools of Optometry evolves to accommodate new technologies but at the same time has to retain fundamental knowledge and skills that are the basis of the new developments. It's always a matter of balance.

The Residents' Poster Session at the American Academy of Optometry's annual meeting in New Orleans showed how well the new generation of optometrists has adapted to new technologies. The report by Drs. Bitton and Dalton on the Canadian contributions to this meeting in this issue shows that our academic and research colleagues are leaders when it comes to extending the limits of the new technologies.

As 2015 draws to a close, I would like to take this opportunity on behalf of the CJO team to wish you and yours all the best for this holiday season and the New Year. Happy Christmas!

B. Ralph Chou, MSc, OD, FAAO
Editor-in-Chief

L'image de la couverture du présent numéro illustre l'une des nouvelles techniques chirurgicales de maîtrise du glaucome décrites par les Drs Heckler, Dorey et Damji. Elle me rappelle à quel point la technologie a transformé tous les aspects des soins de la vue dispensés aujourd'hui.

Dans beaucoup de cabinets d'optométrie, l'autoréfraction a remplacé la rétinoscopie. Les appareils de cartographie et de topographie cornéenne ont supplanté les kératomètres, et le vaste éventail de techniques d'imagerie a en grande partie relégué l'ophtalmoscopie directe à un rôle très restreint dans l'évaluation du segment postérieur. Depuis peu, on utilise des lentilles à cristaux liquides pour le traitement par occlusion au lieu de pansements oculaires. Les nombreux types de lentilles monovision et à addition progressive dont nous disposons font mentir l'un de mes collègues en optique ophtalmique, parti depuis longtemps, qui croyait que « l'optique ne changera jamais ».

Si les technologies fiables et sûres qui réduisent notre temps de consultation et augmentent notre capacité de combler les besoins de nos patients sont toujours accueillies favorablement dans la pratique, leur utilisation efficace suppose une bonne compréhension de leurs fondements et limites chez les praticiens. Le programme de nos écoles d'optométrie évolue en fonction des nouvelles technologies, mais l'enseignement des connaissances et compétences fondamentales qui constituent la base des innovations doit aussi être maintenu. C'est constamment une question de juste milieu.

La séance de présentations par affiches des résidents au Congrès annuel de l'American Academy of Optometry, à la Nouvelle-Orléans, a confirmé que la nouvelle génération d'optométristes s'est bien adaptée aux nouvelles technologies. Le compte rendu des Drs Bitton et Dalton sur les contributions canadiennes à ce congrès, dans ce numéro, démontre que nos collègues universitaires et chercheurs sont des chefs de file lorsqu'il s'agit de repousser les limites des nouvelles technologies.

Comme 2015 s'achève, j'en profite pour vous offrir, à vous et à vos proches, au nom de l'équipe de la RCO, nos meilleurs vœux pour la période des Fêtes et la nouvelle année. Joyeux Noël!

B. Ralph Chou, M. Sc., O.D., F.A.A.O
Éditeur en chef

New Surgical Options in Glaucoma

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Abstract

The treatment of glaucoma is undergoing constant change. In the last decade, there has been a surge of novel surgical options that aim to lower intraocular pressure while providing improved safety profiles compared to traditional incisional glaucoma surgery. This article summarizes four such options— trabectome, iStent, canaloplasty and endocyclophotocoagulation— including descriptions of the procedures and evidence behind them.

Key Words: Micro-invasive glaucoma surgery, *ab interno* trabeculectomy, trabecular micro-bypass stent, canaloplasty, endocyclophotocoagulation

Résumé

Le traitement du glaucome est en constante évolution. Au cours de la dernière décennie, on a observé un afflux de nouvelles options chirurgicales qui visent à abaisser la pression intraoculaire tout en présentant une innocuité accrue par rapport à la chirurgie classique par incision. Cet article décrit quatre de ces options – atrabéculectomie endoculaire au Trabectome®, mise en place du micro-implant iStent®, canaloplastie et cyclophotocoagulation endoscopique –, y compris leur déroulement et les données qui les étayent.

Key Words: Chirurgie micro-invasive du glaucome, trabéculectomie endoculaire, micro-implant de dérivation trabéculaire, canaloplastie, cyclophotocoagulation endoscopique

Introduction

Glaucoma is the second leading cause of blindness globally after cataract.[1] Treatment of glaucoma aims to preserve visual function and maintain overall quality of life.[2] In Canada, a model of interprofessional collaboration between ophthalmologists and optometrists has been suggested.[3] Management decisions are made based on the mechanism of glaucoma, the stage of disease, and the degree of intraocular pressure (IOP) elevation. Patients and care partners should be approached in an individualized manner, taking into account their biopsychosociospiritual (BPSS) profile and preferences.[4] This approach considers various components of health, including systemic biology such as life expectancy, psychological factors, socioeconomic considerations, and spiritual/cultural values.

The standard treatment algorithm has typically employed medications, laser treatment, and eventually surgery. Medications available in Canada include single agents and combination agents involving prostaglandin analogues, beta-blockers, alpha-agonists, carbonic anhydrase inhibitors, and miotics. Laser treatment options include argon laser trabeculoplasty, selective laser trabeculoplasty, and in more severe cases cyclophotocoagulation. This paper discusses surgical options for glaucoma, in particular several newer and less invasive techniques. We will be focusing on the trabectome, iStent, and endocyclophotocogulation, which are *ab interno* techniques, as well as canaloplasty, an *ab externo* technique. There are already several studies reviewing these techniques and any evidence supporting them, and we have referenced these works throughout this paper.[5–7]

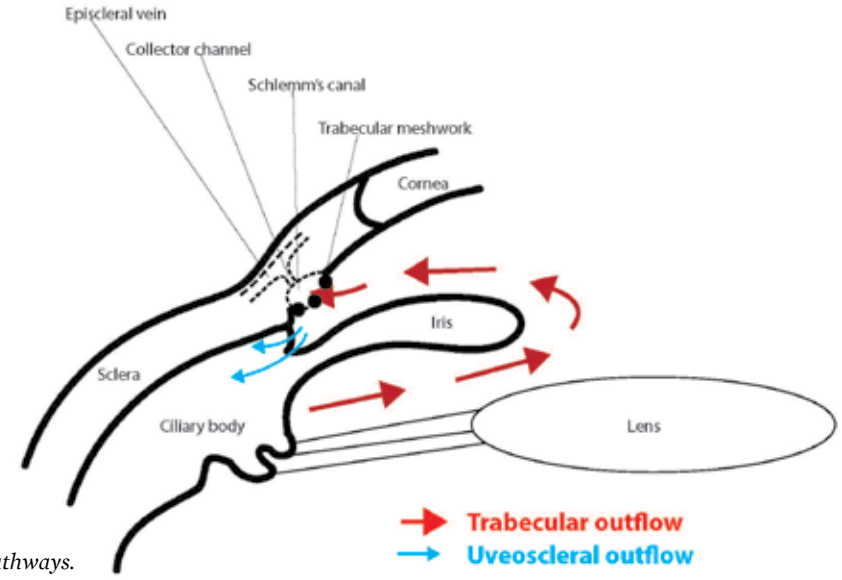

Figure 1. *Aqueous outflow pathways.*

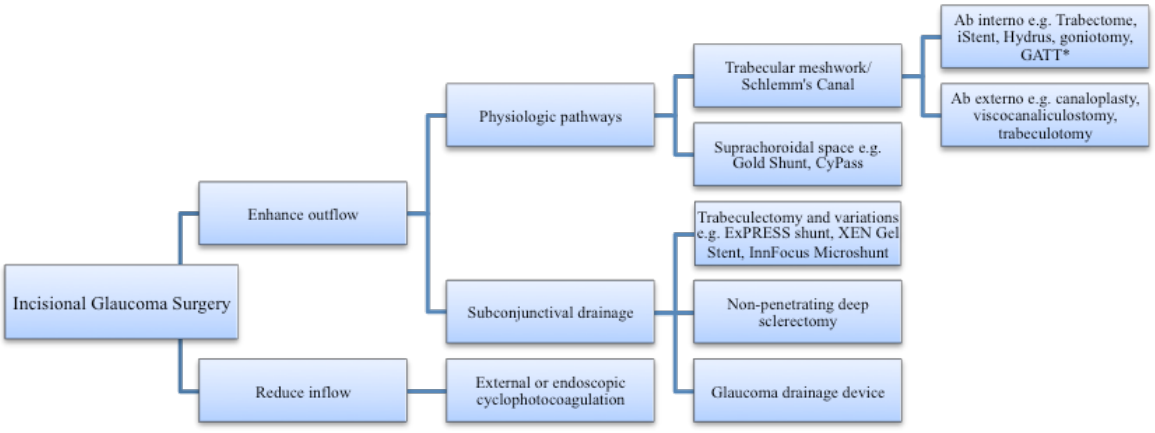

Figure 2. *Classification of Incisional Glaucoma Surgery. * Gonioscopy-assisted transluminal trabeculotomy*

Glaucoma Surgery: A Brief History

Since the 1950s, we have known that the major site of aqueous outflow resistance is the juxtacanalicular portion of the trabecular meshwork.[8] Efforts were made as early as the 1950s to perforate this tissue with energy from light sources to allow for improved outflow.[9] Moses studied microsurgical electro cautery techniques to the trabecular meshwork in enucleated eyes in 1971.[10] Shortly thereafter, laser treatment of the trabecular meshwork became well studied and employed. Surgery to the angle has been well established in the congenital glaucoma population, but it was not until the last fifteen years that angle surgery in adults has come into favour.[11]

A Review Of The Relevant Anatomy

The aqueous is produced by the ciliary processes and leaves the eye by two main pathways: the conventional or trabecular pathway, accounting for 70–95% of aqueous egress from the eye, and the unconventional or uveoscleral outflow, accounting for the other 5–30% (see Figure 1).[12] The techniques described here will be focusing on the aqueous production and trabecular outflow. This consists of the trabecular meshwork, Schlemm's canal, intrascleral channels, and the episcleral or conjunctival veins.

Classification Of Incisional Surgeries For Glaucoma

Incisional glaucoma surgery can be divided into procedures that reduce inflow of aqueous and procedures that enhance outflow (see Figure 2). Outflow procedures are the most common. Currently, trabeculectomy and glaucoma drainage devices remain the most frequently used techniques and several large-scale studies have demonstrated their efficacy.[13–15] Over the last ten years, there has been a shift towards

alternative glaucoma surgeries, largely due to the high rate of complications associated with traditional glaucoma surgeries. These newer techniques have become known as "micro-invasive glaucoma surgery," or MIGS, and play a different role in the glaucoma treatment algorithm.[5,7,11]

Enhancing the Outflow Pathways: *Trabectome, iStent, and Canaloplasty Trabectome*

Description of the Procedure

The Trabectome (Neomedix Corp.) is composed of a disposable hand piece that supplies irrigation and aspiration and a tip that delivers high-frequency electro cautery. The hand piece is advanced across the anterior chamber through a temporal corneal incision towards the nasal angle with the irrigating fluid on. Under gonioscopic visualization, the footplate is inserted into the trabecular meshwork. The cautery is activated, and the surgeon advances the instrument in a clockwise and counterclockwise manner, thereby removing a 3–4 clock-hour arc of trabecular meshwork and the inner wall of Schlemm's canal. This effectively creates a direct pathway for the aqueous to flow from the anterior chamber to the collector channels.[6] The procedure is generally combined with cataract extraction, although there may be certain subgroups who do well with trabectome alone, such as exfoliation syndrome,[16] and juvenile open angle glaucoma.[17]

The advantages of this procedure over traditional glaucoma surgeries include the faster recovery, due to the less invasive nature, the approach which leaves the conjunctiva intact for future surgeries, the lack of a filtering bleb, and the minimal added risk to the patient if combined with phacoemulsification.[18] The disadvantages are that IOP lowering is generally not as substantial as it is with traditional glaucoma surgery, and there are some potential complications, the most common one being hyphema. This usually clears in a few days. Other complications include damage to the cornea, iris, or lens, as well as intraocular pressure elevation, but serious complications such as hypotony and suphrachoroidal hemorrhage are very rare.[19] Delayed hyphema has also been reported,[20] where blood refluxes through the trabectome cleft, which usually resolves within a few weeks but can recur.

Evidence

Several studies have evaluated the effectiveness of trabectome, the first ones being trabectome alone[21] and later ones being trabectome combined with phacoemulsification.[22] The first large study was a noncomparative prospective case series of 304 eyes with open-angle glaucoma undergoing trabectome combined with cataract extraction.[22] Mean baseline IOP of 20 mmHg decreased at 12 months to 15.5 mmHg, and medications decreased from 2.65 to 1.76. Secondary glaucoma procedures were performed in 9 patients. A retrospective review of 88 cases of trabectome alone and 158 cases of combined trabectome and cataract from the Mayo Clinic, found a reduction in mean IOP from 21.6 mmHg to 15.3 mmHg and a decrease in number of glaucoma medications from 3.1 to 1.9 at 2 years. However, subsequent glaucoma surgery was required in 66 patients (26.8%), an average of 10 months after surgery.[23] The weakness of these studies is the lack of a comparison (e.g. phacoemulsification alone) to prove the effectiveness of the procedure, since prior studies have demonstrated the IOP-lowering benefit of phacoemulsification alone.[24,25]

iStent

Description of the Procedure

The Glaukos micro-bypass trabecular iStent (Glaukos Corp.) is the smallest medical device to be implanted in the human body. It is made of nonferromagnetic heparin coated titanium and consists of an inlet ("snorkel") connected at a right angle to the implantable portion (see Figure 3).[6] The preloaded iStent inserter is advanced across the anterior chamber through a temporal corneal incision under gonioscopic visualization towards the nasal angle. The sharp tip of the iStent is used to engage and perforate the trabecular meshwork, and the stent then slides into the Schlemm canal. The device is then released from the inserter. Usage of the iStent has been studied in combination with cataract extractions. The advantages of the iStent are similar to the trabectome in terms of minimizing ocular tissue damage, and possibly lower risk of complications.[26]

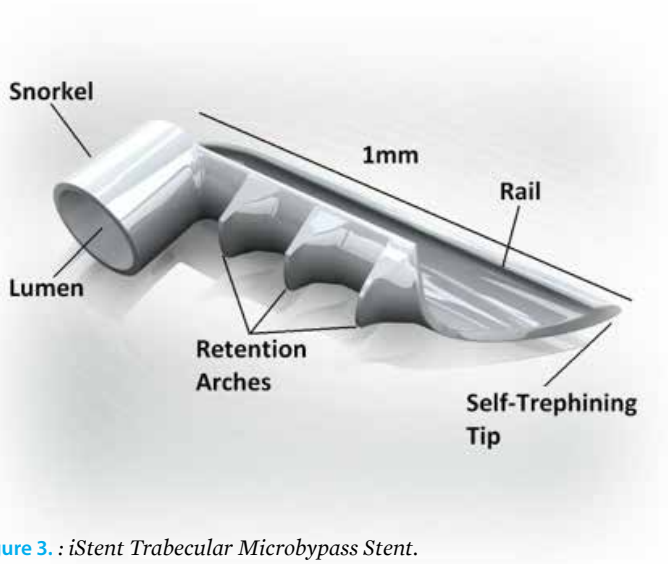

Figure 3. *: iStent Trabecular Microbypass Stent.*
Courtesy of Glaukos Corporation, Laguna Hills, California, USA.

Evidence

Initial studies reported the use of a single iStent per eye, though subsequent studies found multiple stents to be more effective.[27] Spiegal et al. reported the successful use of the iStent in 2008 in a prospective, multicenter noncomparative interventional trial.[28] Since then, further validation has arisen from randomized control trials comparing phacoemulsification alone to phacoemulsification with iStents.[29–31] In the two-year follow-up paper by the iStent Study Group, 240 eyes with mild to moderate glaucoma were randomized to receive one iStent combined with phacoemulsification or phacoemulsification alone. At both one and two years, the proportion of patients with unmedicated IOP of 21 or less was significantly higher in the iStent group.[31] Amount of hypotensive medications were lower in the stent group at one year, but this did not reach statistical significance at two years. Complication rate was similar between the two groups, and there were no stent-related adverse effects reported after two years. More recently, Ahmed et al. evaluated the use of two iStents combined with travaprost post-operatively, to improve both conventional trabecular and uveoscleral outflow in 39 open angle glaucoma patients uncontrolled on two medications. Mean IOP decreased from 22.2 mmHg to 11.8 mmHg at 18 months, from two medications pre-operatively to one medication post-operatively.[32]

Further expansion of the iStent's role and its long-term efficacy are yet to be determined. Some authors have suggested it may play a limited role in select patients with advanced disease or even prior filtering surgery.[33,34]

Canaloplasty

Description of the Procedure

This technique is similar in concept to viscocanalostomy, which involves the creation of a deep sclerectomy followed by injection of viscoelastic into Schlemm's canal. In canaloplasty, a superficial and then deeper scleral flap is created, so that Schlemm's canal can be reached. The canaloplasty device (Ellex) contains a fiber optic light catheter that is then inserted into one end of the open Schlemm's canal. This is threaded around the canal for 360 degrees, with the aid of the light, such that a false passage is not created, until it exits from the other opening in Schlemm's canal. A prolene suture is then attached to the distal end of the catheter, and the catheter is withdrawn, thereby threading the suture through the canal. The ends of the suture are tied to distend the trabecular meshwork.

The advantages of this procedure are that it does not require a filtering bleb, though occasionally one forms,[35] and it has less complications compared to conventional trabeculectomy. The disadvantages are that the learning curve is long, in some cases it is impossible to canulate Schlemm's canal, and the IOP-lowering effect is not as dramatic as trabeculectomy.[36] It is also more invasive than the MIGS procedures, as it disrupts conjunctiva, although *ab interno* approaches are now also being explored.

Evidence

In a nonrandomized multicentre trial, Lewis et al. reported the three-year results of 157 patients undergoing canaloplasty or combined canaloplasty-cataract surgery.[35] Baseline IOP of 28.5 mmHg reduced to 15.2 mmHg, and medications decreased from 1.8 to 0.8. Most complications occurred intraoperatively or in the early post-operative period, including partial suture extrusion through the trabecular meshwork, Descemet's detachment, hyphema, and IOP elevation.

Brüggemann et al. also compared canaloplasty to trabeculectomy.[37] In this study of 15 patients with prior trabeculectomy in one eye, the contralateral eye underwent canalopasty, and 6- and 12-month results were compared. Both procedures reduced the IOP significantly; however, the trabeculectomy group achieved lower IOP (11.64 vs. 13.21 mmHg), was on no medication, and required fewer additional interventions. However, they required longer initial hospitalization and more post-operative visits.

Reducing Inflow: Endoscopic Cyclophotocoagulation

Description of the Procedure

Cyclophotocoagulation was first used in the 1970s to lower intraocular pressure. It is usually done through a contact approach delivered over the area of the ciliary body, thereby reducing aqueous production. It is generally left as a last-resort to treat end-stage glaucoma due to its unpredictability and

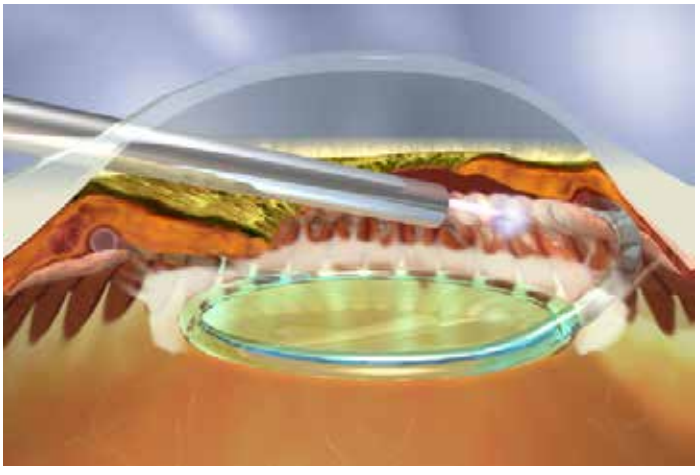

Figure 4. *: Endoscopic Cyclophotocoagulation of the Ciliary Processes. Courtesy of Endo Optiks, Inc., Little Silver, New Jersey, USA.*

possibility of complications like phthisis.[38] In 1992, however, Uram described the use of photocoagulation delivered to the ciliary body under direct visualization with endoscopy.[39]

Endoscopic cyclophotocoagulation (ECP) has been used in several different types of glaucoma, including primary open angle, neovascular glaucoma, chronic angle closure, uveitic, angle recession,[38] and a less destructive version in plateau iris syndrome.[40] The laser unit contains a probe to deliver the diode laser that has a built-in endoscope and attached camera monitor. When this is combined with phacoemulsification, the ECP is generally done before or after the lens is inserted. The probe is inserted through a corneal incision and directed behind the iris until the ciliary processes come into view on the camera monitor. The laser is then fired with a foot pedal and the desired outcome is a whitening of the ciliary processes (see Figure 4). This treatment can be done for a circumference of 90–360 degrees, and a second incision may be required.[38]

Evidence

A retrospective review of 368 eyes that underwent phacoemulsification combined with ECP found a 10.9 mmHg drop in IOP at two years post-operatively, with a mean decrease in number of medications of 1.1.[41] The largest published study, by the same group, was a retrospective review of 539 patients.[42] All eyes had at least one prior glaucoma surgery and IOP ≥ 35 on maximal medical therapy or advanced glaucoma with IOP above target. The mean IOP decreased from a baseline of 38 mmHg to 12.1 mmHg, and an average number of medications of 1.9, after 5 years. Prospective studies have also evaluated the use of ECP. In a randomized trial, Gayton et al. compared ECP to trabeculectomy and found a 29% reduction in IOP after ECP vs. 32% following trabeculectomy, with similar baseline IOP of 25 mmHg.[43] Francis et al. evaluated ECP use after failed aqueous shunts. Twent-five eyes received ECP for 360 degrees.[44] Success, defined as reduction in IOP of 3 mmHg and discontinuation of nontolerated glaucoma medications, was achieved in 88% of patients up to two years follow up. Murthy et al. studied 50 eyes of Indian patients with refractory glaucoma, including phakic, pseudophakicm and phakic patients, in a prospective non-comparative study.[45] IOP decreased from 32.58 mmHg to 13.96 mmHg, with an average follow up of about one year. The average number of glaucoma medications decreased from 2.51 to 1.09.

How To Choose Which Procedure To Use On On Each Patient?

The decision to proceed with glaucoma surgery is a complex one that should take into account different ocular and systemic factors. In the Canadian model, where optometrists and ophthalmologists work together in the best interest of patients, effective and timely communication is essential.[3,46] Treatment of glaucoma and glaucoma suspects is well summarized in the Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye.[2] Indications for surgery include situations where other methods of lowering IOP have been unsuccessful and the optic nerve is failing or likely to fail. Furthermore, surgery should be considered when patients are intolerant to and/or non-compliant with medications. Referral to an ophthalmologist when progression is suspected is crucial in preventing glaucoma-related blindness.

The indications for some of the novel glaucoma surgeries described here are currently in evolution. The procedures that act by increasing outflow (trabectome, iStent, canaloplasty) have primarily been studied in the context of mild to moderate glaucoma and are usually combined with cataract surgery. Since they generally do not lower IOP to a level as low as traditional surgery, the authors favour their use in situations where there is a visually significant cataract and early or moderate glaucoma where the IOP is above target. ECP, however, may be a good option in more advanced or refractory glaucoma based on the available literature.

Other factors that might encourage a surgeon to proceed with a MIGS over a trabeculectomy or tube shunt include poor conjunctival or scleral tissue, where a trabectome or iStents would be safer than an external surgery, as well as risk factors for over-filtration (e.g. myopia) or suprachoroidal hemorrhage (e.g. older age, presence of hypertension, use of anticoagulants). Fortunately, most MIGS procedures do not seem to affect the future outcome of traditional glaucoma surgery.[47] Finally, patients and care partners should be involved in the discussion and decision-making regarding treatment.

Post-Operative Care

Post-operative care for microinvasive glaucoma surgeries is similar to post-operative cataract care; however, there are several added complications that should be watched for. Uveitis may be more prominent due to the added intraocular manipulation, in particular with ECP.[38] This can usually be managed with steroid drops and/or non-steroidal agents, especially to reduce the risk of cystoid macular edema. Hyphema or microhyphema is not uncommon and can be treated conservatively with steroid drops, reduced level of activity, and elevation of the head. If a hyphema is large (i.e. over one-half of the anterior chamber), not clearing after two weeks, or associated with elevated intraocular pressure,

consideration should be given to a surgical washout.

Intraocular pressure elevation may occur, with early pressure rise likely due to inflammation and later rise more likely from steroid usage, particularly after trabectome (author's personal experience) or iStent surgery.[48] The management of these two situations, therefore, differs. If the inflammatory response seems prominent, steroids should be increased; however, if steroid-induced pressure elevation is suspected and inflammation is minimal, lower-potency steroids or non-steroidal agents should be used and rapidly tapered off.

Since some of these procedures include the insertion of a device, there is also the potential for such device-related complications as obstruction or malposition, though these risks are low.[5] Gonioscopy may be necessary to identify these issues. Finally, as is the case with all glaucoma surgeries, the pressure control may decrease or fail over time, at which point other methods must be employed (drops, laser, further surgery).

Conclusion And Summary

Surgical care of glaucoma is undergoing some major paradigm shifts. Larger scale randomized control trials will be important in better defining the role of MIGS in the treatment algorithms. Other MIGS procedures not discussed here have also been described, including Hydrus, CyPass, iStent inject (second-generation iStent), as well as other newer surgical treatments such as the Gold shunt. Finally, modifications of conventional surgeries have been studied and employed by many, including the Ex-PRESS Glaucoma Filtration Device (Alcon, Inc.), as an additive device to traditional trabeculectomy surgery. With continued collaboration among eye care specialists, individualized glaucoma care is becoming a reality.

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Reviewing Guidelines on Diabetic Retinopathy Screening in Children and Adolescents with Type 1 Diabetes: Is there consistency amongst practitioners?

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Abstract

Diabetic retinopathy (DR) is a common eye disease and a leading cause of visual impairment in patients with Type 1 diabetes (T1DM). Retinopathy screening for T1DM varies according to the age of disease onset and diabetes duration. Retinal screening varies from standard fundal examination to more advanced methods of screening. An online survey was conducted in February 2014. The purpose of this survey was to assess the frequency and methods of eye examinations routinely performed in children and adolescents with T1DM. Data on local practices were collected from a group of optometrists and ophthalmologists in the London-Middlesex area. One hundred and one surveys were e-mailed out and the response rate was 37.6%. Results indicated that different screening methods vary according to individual practices. These results may have an impact on the findings of retinopathy in this population. A review of utilized screening methods and comparisons to established guidelines will be highlighted.

Keywords: diabetic retinopathy; type 1 diabetes; children; microvascular complications.

Résumé

La rétinopathie diabétique est une maladie oculaire répandue et une cause importante de déficience visuelle chez les personnes atteintes de diabète de type 1. Son dépistage varie suivant l'âge du patient quand le diabète est diagnostiqué et l'ancienneté de cette maladie. Les méthodes de dépistage vont du simple examen du fond d'œil à des techniques plus poussées. En février 2014, nous avons mené un sondage en ligne, afin d'évaluer la fréquence et la nature des examens oculaires menés systématiquement chez les enfants et les adolescents atteints de diabète de type 1. Les données sur les pratiques locales ont été recueillies chez des optométristes et des ophtalmologistes de la région de London-Middlesex (Ontario). Le taux de réponse aux 101 formulaires envoyés par courriel s'est établi à 37,6 %. Les résultats obtenus témoignent de la diversité des méthodes de dépistage appliquées par les praticiens. Cette diversité pourrait avoir une incidence sur le diagnostic de la rétinopathie chez les enfants et les adolescents diabétiques. Cet article expose les méthodes de dépistage appliquées, en les mettant en parallèle avec les méthodes recommandées dans les lignes directrices établies.

Mots clés : rétinopathie diabétique; diabète de type 1; enfants; complications microvasculaires.

Introduction

In 2008/09, more than 3,000 new cases of diabetes (Type 1 and Type 2) were reported among Canadian children and youth aged one to 19 years, bringing the number of prevalent cases to just fewer than 26,000. The rate of T1DM diabetes among one to nine year olds has increased, from 0.1% (3,726 cases) in 1998/99 to 0.2% (5,201 cases) in 2008/09.[1] Despite the increase in incidence, the prevalence of diabetic retinopathy (DR) has decreased globally, which is mainly attributed to improved management of diabetes control.[2] In a 20-year Australian study of 1604 adolescents with T1DM, it was found that the prevalence of DR was approximately 50% in the early 1990s, and decreased to approximately 12% in 2009.[3]

The Diabetes Control and Complications Trial (DCCT, 1983-1993)[4] provided unequivocal evidence that intensive diabetes treatment and improved glycemic control conferred a significant risk reduction for microvascular complications compared with conventional treatment. In the adolescent cohort, intensive treatment compared with conventional treatment reduced the risk and progression of background retinopathy by 53 %.[4] Diabetic retinopathy rarely develops in children with Type 1 diabetes <10 years of age regardless of the duration of diabetes. Among patients <15 years of age, irrespective of age of diabetes onset, the prevalence of mild nonproliferative retinopathy was 2% with no reported sight-threatening diabetic retinopathy.[2] However, the prevalence rate increases sharply after 5 years' duration of diabetes in post pubertal individuals with Type 1 diabetes.[2] Early identification and treatment of DR can decrease the risk of vision loss in affected patients. Therefore, it is imperative to screen for early signs of this complication in the pediatric T1DM population.

There are three distinct forms of DR: i) macular edema, ii) nonproliferative and proliferative DR, and iii) retinal capillary closure. Macular edema involves focal or diffuse vascular leakage at the site of the macula. Nonproliferative DR is the progressive accumulation of blood vessel change that includes microaneurysms, intraretinal hemorrhage, vascular tortuosity and vascular malformation (together known as nonproliferative diabetic retinopathy) that ultimately leads to abnormal vessel growth (proliferative diabetic retinopathy). Retinal capillary closure is a form of vascular change detected on fluorescein angiography, which is also well recognized as a potentially blinding complication of diabetes but currently has no treatment options.[5] Severe nonproliferative DR, proliferative DR, and clinically significant macular edema are considered sight-threatening DR.

The aim of this survey was to explore the practices for DR screening in patients with T1DM aged less than 18 years assessed in London Middlesex County, Ontario. Screening methods in relation to recommended guidelines will be discussed.

Methods

The online survey was approved by the Ethics Review Board of Western University (#HSREB 104566). Questionnaires were distributed by e-mail to optometrists and ophthalmologists in London and Middlesex County listed in the Optometry Association of Ontario. This was conducted via Survey Monkey from February 10th, 2014 to March 1st, 2014. Participants were advised that by opening the electronic survey and completing it, they were providing their consent to be involved in the study.

Seven open-ended questions were administered to all participants Table 1. Questions on the survey were related to the frequency and technique of eye examinations routinely performed in T1D children. Responses were collected for review and analysis.

Results

Responses were received from 38 of 101 surveys sent (response rate 37.6%), which included 31 optometrists and 7 ophthalmologists (81.6% and 18.4%, respectively). All responding optometrists and 6 out of 7 ophthalmologists examine children or adolescents aged less than 18 years with T1DM for DR. From the optometrist group, the majority (64.5 %) examine less than 10 children and adolescents per year. From the ophthalmology group, the majority (57%) examine more than 10 patients per year.

Respondents use different methods to screen for DR as shown in Figure 1. A subgroup of respondents (28%) examines for DR using multiple methods Figure 2.

The majority of optometrists (96.8%) and ophthalmologists (71.4%) recommend annual eye exams for children and adolescents with T1DM if their exam is normal. One optometrist (3.2%) recommends exams every two years, and two ophthalmologists (28.6%) recommend exams every 6 months.

In terms of referring patients for an ophthalmologist assessment, a higher percentage of optometrists (54.8%) refer those with signs of moderate non-proliferative diabetic retinopathy (NPDR). Two (6.5%) ophthalmologists refer with findings of mild NPDR and six (19.4%) refer only if evidence of severe NPDR. 19.3% refer children with T1DM only with evidence of proliferative diabetic retinopathy (either early or high risk).

All ophthalmologists and 53% of optometrists send reports to both the family doctor and the endocrinologist; 38% of optometrists send reports to the family doctor only and 3% do not send any reports.

Table 1. Eye Survey Questions

1. Do you assess patients with diabetes for diabetes retinopathy? Y/N

2. Do you exam children/adolescents aged less than 18 years with **Type 1 diabetes**? Y/N

3. Approximately, how many children/ adolescents aged less than 18 years with type 1 diabetes do you exam **per year**?
(Age groups: <5, 5-10, 10-15, 15-20)

4. What is the method of screening used for the population with type 1diabetes?
a) Ophthalmoscopy without dilated fundi
b) Ophthalmoscopy on dilated fundi
c) 7-field stereoscopic photography with pupil dilation
d) 4-field wide-angle stereoscopic photography with pupil dilation
e) Digital imaging (3-field) with no dilation
f) Other method, please specify:_____

5. If the eye exam is **NORMAL**, how frequently do you recommend eye exam for children and adolescents with type 1 diabetes?
a) 6 months
b) 12 months
c) 24 months
d) > 24 months

6. **If you are not an ophthalmologist**, what abnormalities according to Airlie House classification for diabetic retinopathy do you refer children/ adolescents with type 1 diabetes to an ophthalmologist?
a) Mild nonproliferative
b) Moderate nonproliferative
c) Severe nonproliferative
d) Early proliferative retinopathy
e) High risk proliferative retinopathy

7. To whom do you send the eye examination reports?
a) To Family doctor
b) To Endocrinologist
c) To both Family doctor and endocrinologist
d) To family only
e) Reports not sent

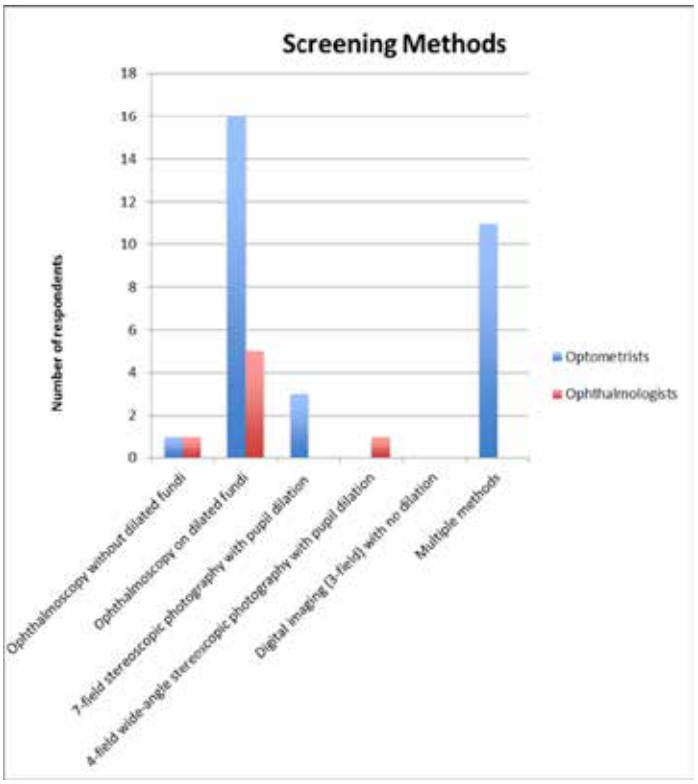

Figure 1. Methods for assessment of diabetic retinopathy (DR) used among children and adolescents with type 1 diabetes (T1DM).

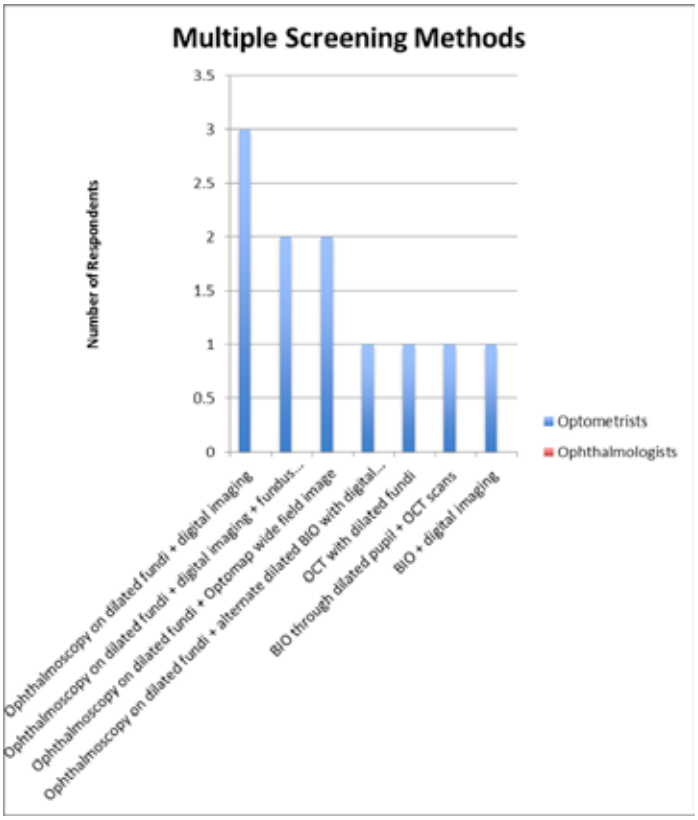

Figure 2. Distribution of multiple screening methods used in examining children and adolescents with type 1 diabetes (T1DM).

Table 2. Summary of screening methods for diabetic retinopathy

Screening Method	Description	Sensitivity	Specificity
Methods done with pupil dilation			
7-field stereoscopic photography	Film photographs of the retina taken at 30°-35° fields. Gold standard for documenting diabetic retinopathy as described in the Early Treatment Diabetic Retinopathy Study.[12]		
4-field wide-angle stereoscopic photography	Digital coloured images of the retina taken at 45°-60° fields.[13]	94% for detecting severe NPDR or better	96% for detecting severe NPDR or better
Ophthalmoscopy with pupil dilation	Clinical examination of the retina using ophthalmoscope through dilated pupil.	65% (95% CI: 51-79%)	97% (95% CI: 95- 99%)
Fundus biomicroscopy	Slit-lamp examination with use of biomicroscope to provide stereoscopic, highly magnified examination of the ocular fundus and vitreous with a large field of view. It should be considered the standard clinical technique for stereoscopic examination of the posterior pole of the eye.[14]	76% (95% CI: 70-81%) for detecting sight threatening DR	95% (95% CI: 95-96%) for detecting sight threatening DR
Methods done without pupil dilation			
Ophthalmoscopy without pupil dilation	Clinical examination of the retina using ophthalmoscope without dilation of pupil.	Not available	Not available
Digital imaging (3-field) without pupil dilation	Three 45° field stereoscopic fundus images obtained at the optic disc and macula, superotemporal to the optic disc, and nasal to the optic disc.[15]	98%	86%
Binocular indirect ophthalmoscopy (BIO)	Headband or Slit-lamp examination using a BIO lens. The slit lamp microscope binocular indirect method allows for assessment of the depth of retinal vascular lesions, disc evaluation and macular oedema. The head-band indirect ophthalmoscopy method allows for views into the far periphery, past the equator.[16]	76%	95%
Optomap wide field image	One stereoscopic digital image taken at up to 200° (82%) of the retina, using scanning laser ophthalmoscope technology combined with a large ellipsoidal mirror.[17]	DR: 99% DR: 99%	DR: 100% PDR: 99%

CI, confidence interval

Discussion

Diabetic retinopathy rarely develops in children with Type 1 diabetes <10 years of age regardless of the duration of diabetes.[2] In the Wisconsin Epidemiology Study of Diabetic Retinopathy 4-year incidence study, no person <17 years of age developed proliferative retinopathy or macular edema.[2] In the United Kingdom Prospective Diabetes Study (UKPDS), few patients without retinopathy at diagnosis of diabetes had disease progression to the point of requiring retinal photocoagulation (laser treatment) in the following 3 to 6 years.[6]

The Liverpool Diabetic Eye Study reported the 1-year cumulative incidence of sight-threatening diabetic retinopathy in individuals with Type 1 or Type 2 diabetes who, at baseline, had no diabetic retinopathy, had background retinopathy or had mild pre-proliferative retinopathy. In people with Type 1 diabetes, the incidence in these groups was 0.3%, 3.6% and 13.5%, respectively.[7]

In the pediatric population with Type 1 diabetes, others have reported a decline in retinopathy supporting current guidelines that recommend lower glycemic targets and the use of intensive diabetes management in children and adolescents with T1DM.[3]

In a cross-sectional study, Kubin *et al* (2011) examined the prevalence of DR through fundus photographs in children and adolescents diagnosed with T1DM. The overall prevalence of DR was 11.8% showing no decrease in the past 17 years.[8]

The largest prospective studies to date by Porta *et al* (2014) support the hypotheses that DR may appear later in patients

Table 3. Summary of recommended guidelines for Diabetic Retinopathy screening

Guideline recommendations	Initiate screening	Frequency of screening	Method of screening
ISPAD/IDF[9-10]	Start screening for retinopathy at age 11 and after 2 years of type 1 diabetes duration.	Screen annually after 2 to 5 years' diabetes duration and more frequently if indicated. For those with less than 10 years of diabetes duration, with reasonable glycemic control may assess biennially by fundal photography.	Minimum retinopathy assessment should be by ophthalmoscopy through dilated pupils by an experienced observer. In countries with ample resources, retinopathy assessment should be by fundal photography with or without mydriasis.
CDA 2013[6]	Start screening for retinopathy 5 years after type 1 diagnosis in all individuals 15 years and older	Five years after diagnosis of type 1 diabetes in all individuals ≥15 years, rescreen annually.	Seven-standard field, stereoscopic colour fundus photography with interpretation by a trained reader Direct ophthalmoscopy or indirect slit-lamp fundoscopy through dilated pupil, or Digital fundus photography
ADA 2014[11]	Consider initial dilated and comprehensive eye examination at start of puberty or at age 10, whichever is earlier, and after 5 years of type 1 diabetes duration.	Annual screening. May increase to biennial eye exams if no findings on screening. If evidence of retinopathy is seen, then resume annual exams.	High-quality fundus photographs.

ISPAD, International Society of Pediatric and Adolescent Diabetes; IDF, International Diabetes Federation; CDA, Canadian Diabetes Association; ADA, American Diabetes Association.

who develop diabetes before puberty; however the long term pre-pubertal years add to its cumulative prevalence. DR is generally infrequent and mild during childhood and may be related to the shorter length of time of glycemic exposure.[9]

In terms of screening methods, ADA, CDA and IDF/ISPAD guidelines[5,10,11] support fundus photography, with or without pupil dilation, as the method of screening for retinopathy. Nevertheless, ISPAD/IDF recognizes that this method may only be available in countries with ample resources, and thus it accepts dilated ophthalmoscopy as the minimum assessment.[10] CDA specifies 7-field stereoscopic fundal photography as the gold standard for DR screening.[5] (Table 2)

Moreover, ISPAD/IDF advocates the onset of screening at age 11 years while CDA promotes later initiation of screening at 15 years of age and after 5 years of diabetes duration.[10] Table 3

This present survey highlights the lack of consistency both in the methods applied for retinopathy assessment as well as the time-intervals recommended by different specialists.

The divergent responses of this survey are a reflection of the different practices recommended by different expert groups. For example, ADA, CDA and ISPAD/IDF guidelines suggest annual DR screening, although there is no agreement on the age and duration of diabetes at initiation of screening.[7] (Table 3)

In our survey, 94.7% of respondents, met the minimum recommendations and provided at least dilated ophthalmoscopy or fundal photography. Fundal photography was used by 34.2% of respondents, 9 respondents used it in combination with dilated ophthalmoscopy. Dilated ophthalmoscopy alone was used by 55.3%.

Only less than 1% of our participants, all of whom were optometrists, apply the gold standard screening of 7-field fundal photography. Although it is not possible to confirm participant location, one explanation could be that these participants are linked to tertiary centres where higher resources are available.

The main limitation of this study is a low survey response rate which may not have captured the true prevalence of methods used for retinopathy screening and the communication gap between professionals.

Caring for children and adolescents with T1DM requires a multidisciplinary approach. Communication between healthcare members is crucial for optimal diabetes care and prevention of acute and chronic complications. CDA guidelines have no clear recommendations in relation to communication between specialists. ADA suggests that results of eye examinations should be documented and transmitted to

the referring health care professional. Of note, almost 40% of optometrists in this survey report only to family doctors with no routine communication with the endocrinologist. Also, 3% of the participants do not report to any physician involved in the care of these children.

In summary, this local survey demonstrated a broad range of screening methods for assessment of diabetic retinopathy with no consistency in relation to age of onset or frequency of eye examinations. It also identified a gap in communication between healthcare providers involved in the care of children and adolescents with T1DM. This highlights the need for improved communication in a timely manner in order to prevent the development of diabetes-related complications.

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Vernal Keratoconjunctivitis and its Management Challenges

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Abstract

AbstractVernal keratoconjunctivitis (VKC) is an allergic, bilateral, recurrent inflammatory disorder of the conjunctiva and the cornea that has a seasonal incidence. It affects young males more than females in age bracket of 3 to 16. In the majority of cases, symptoms resolve at puberty, but some may run into adulthood. Diagnosis is based typically on clinical signs and symptoms. The cause of the disease is not clearly known, but it is often associated with atopic diseases such as asthma or eczema and is probably due to a longstanding allergic reaction.

The disease consists clinically of the palpebral, limbal and mixed types. Its management has been a great challenge to eye care providers because of its recurrent nature, the large number of individuals who are affected, wrong diagnoses, and lack of judicious drug administration. The disease has the potential of producing serious vision-threatening complications if not adequately managed. This case report also reviews the diagnosis and management options for patients with mixed VKC and demonstrates the importance of the clinician's role in taking a careful case history and in modifying treatment when necessary throughout care.

Keywords: Shield ulcer, giant papillae, superficial keratopathy, supratarsal injection, local concoction.

Résumé

La kératoconjonctivite vernale (KCV) est une inflammation allergique, bilatérale et récurrente de la conjonctive et de la cornée dont l'incidence est saisonnière. Elle sévit plus chez les hommes que chez les femmes dans la tranche d'âge trois à seize ans chez les jeunes. Dans la majorité des cas, les symptômes disparaissent à la puberté, mais dans certains cas, ils peuvent persister jusqu'à l'âge adulte. Le diagnostic repose normalement sur les signes et symptômes cliniques. La cause de cette affection n'est pas clairement établie, mais elle se trouve souvent liée à des affections atopiques comme l'asthme ou l'eczéma et est probablement imputable à une réaction allergique de vieille date. Elle est cliniquement des types palpébral, limbique et mixte. Sa prise en charge a présenté tout un défi pour les soignants oculovisuels à cause de son caractère récurrent, du grand nombre de sujets touchés, des erreurs de diagnostic et de l'absence de posologie judicieuse. L'affection risque d'engendrer de graves complications qui viendront menacer la vue si la maladie n'est pas convenablement prise en charge. Dans cette étude de cas, nous passerons aussi en revue les options de diagnostic et de gestion dans le cas des patients atteints d'une KCV du type mixte et nous démontrerons l'importance du rôle du clinicien qui doit relever soigneusement les antécédents et modifier le traitement si nécessaire tout au long des soins.

Mots clés : Ulcère de la cornée, papilles géantes, kératite superficielle, injection supratarsienne, préparation locale.

Introduction

Allergic conjunctivitis is the inflammation of the conjunctiva due to allergens. The inflammation caused by the allergens may be seasonal and involve both the conjunctiva and the cornea, hence the name vernal keratoconjunctivitis (VKC). Other clinical forms are included in the classification of ocular allergy from which VKC must be distinguished: seasonal/intermittent allergic conjunctivitis (SAC), perennial/persistent allergic conjunctivitis (PAC), atopic keratoconjunctivitis (AKC), giant papillary conjunctivitis (GPC) and drug-induced dermato-conjunctivitis. Amongst these, AKC is closest in appearance to VKC.[1,2,17]

This review focuses on the management challenges of VKC, a seasonal, recurrent, bilateral, chronic allergic inflammation of the conjunctiva and the cornea.[2,6] The allergic response is triggered by an over-reaction of the body's immune system to allergens or foreign bodies and tends to run a more severe course.

It is also known as 'seasonal conjunctivitis,' 'spring catarrh' or 'warm weather conjunctivitis.'.[1] It is the only ocular disease to involve solely Type 1 hypersensitivity response mediated by IgE antibodies bound to the membrane of the mast cell in the conjunctival substantia propria.[3,14] Attachment of the antigen to the IgE antibodies results in the release of pro-inflammatory mediators like histamines, leukotrienes, prostaglandins, tryptase, chymase, platelet activating factor and other chemo-attractants. This results in increased vascular permeability and attraction of eosinophils and neutrophils.[3,7,12]

These pro-inflammatory mediators are responsible for the clinical pictures of the conjunctivitis.[3] However, cytologic, biohumoral, immunohistologic and molecular biologic studies have suggested the involvement of T helper cell type 2 (Th-2) lymphocyte mediated allergic reaction with additional hypersensitivity responses that are often ill-defined.[7,12,13] Vernal keratoconjunctivitis is usually bilateral, but may occasionally be asymmetrical. It accounts for roughly 0.10 to 0.50% of patients with ocular diseases worldwide.[8,18] However, unilateral VKC is rare.

The disease affects children between 3 to 16 years of age, though it may appear earlier than that and continue into adulthood. In some cases, it is often self-limiting and the symptoms resolve around puberty.[6] It is more common among boys than in girls probably due to their persistent outdoor activities.[1] Its predominance in male children, resolution at puberty and positive staining for oestrogen and progesterone receptors in conjunctiva of patients with VKC also suggest a hormonal component in the development of VKC.[7,9,16] In addition, increased serum levels of interleukin-17 (IL-17) and antinuclear antibodies, together with a high association with familial history of autoimmune disorders suggest additional mechanisms involved in the development of VKC.[7,11,13] Multiple factors can contribute to the aetiology of VKC including environmental allergens, climate and genetic predisposition.[7] However, the suggestion of a hereditary association still remains unclear as no clear direct correlation with specific genetic loci has been made.[5] The disease is minimal in temperate climates compared to warm climates, and almost nonexistent in cold climates. It is severe in spring, summer and incidence falls in the winter. It is more common in Sub-Saharan Africa and the Middle East.[1]

Though the name vernal implies a seasonal occurrence, the disease may be perennial (persisting throughout the year) or with acute exacerbations.[18] Severe VKC greatly affects a child's academic performance and ultimately impairs his quality of life. The corneal involvement in the disease known as vernal keratitis may be severe enough to interrupt a child's education and ultimately affect his future potential. Besides, it can lead to permanent visual impairment if not adequately managed.[15] Vernal keratitis is characterized by a combination of punctate epithelial erosions and keratitis.[3] Punctate epithelial erosions are areas of absent epithelium that stain with fluorescein. Punctate epithelial keratitis is caused by an influx of white cells into the corneal epithelium. The epithelium in this area stains with Rose Bengal, which stains devitalized cells.[2,9] Keratitis epithelialis of Tobgy is the early phase of vernal keratitis, consisting of minute white dots in the epithelium which when coalesce, forming vernal ulcers known as shield ulcers.[7,16] Shield ulcers are distinctly indolent, oval in shape and most often found in the upper half of the cornea. The pathogenesis of these shield ulcers is believed to involve a combination of mechanical damage to the corneal epithelium by giant papillae and toxic epitheliopathy from inflammatory mediators secreted by eosinophils and mast cells.[5,15] These ulcers are sterile but may, by chance, be infiltrated by bacterial pathogens giving rise to bacterial corneal ulcers. These ulcers can be differentiated from vernal shield ulcers, which are indolent and often have a plaque at the base. Shield ulcers occur in approximately 3% to 20% of patients with VKC.[7,15,16]

Causes

The aetiology and pathophysiology of VKC remain unclear, but may be due to a longstanding allergic reaction of the body's immune system to allergens.[4] It is common in people who have other signs of allergic diseases such as hay fever, asthma, and eczema.[4,6] Prominent causative allergens include pollen from trees, grasses and ragweed; animal dander; dust; smoke;

mould spores; hairs; dust mites; wool and feathers; perfumes; cosmetics; eye drops; and skin medicines.[1,8]

Symptoms

The symptoms are due to the release of histamines and other active substances by the mast cells, which stimulate dilation of blood vessels, irritate nerve endings and increase secretion of tears.[7]

Common symptoms include severe itching, tearing, photophobia (sensitivity to light), pain, eyelid swelling, irritation, blepharospasm, burning and foreign body sensation. Blurring of vision may occur when the cornea is involved. These complaints, especially itching, are made worse when the patient is exposed to a dusty, warm humid atmosphere, wind and bright light, or physical exertion associated with sweating, possibly due to increase in allergen count.[3,6,8,16]

Signs

The disease is characterized by milky appearance of the conjunctiva, fine papillae in the lower tarsal conjunctiva and giant, flat-topped papillae resembling a 'cobblestones' in the superior tarsal conjunctiva (which is the hallmark of the disease).[1,7] Tenacious mucus may form and adhere to the cobblestones. The conjunctiva may be swollen and hyperaemic. Thick, ropy strands of mucus or filamentary keratitis are present (mainly in severe VKC).[16] Microscopic examination of the conjunctival biopsies reveal large numbers of scattered eosinophils and eosinophilic granules but these are less common on conjunctival smears. Basophils may also be present in the biopsy but not in the same proportion as eosinophils. High levels of proteins secreted by eosinophils (e.g. eosinophil cationic protein) have been found in the tears of VKC patients as well as atopic and allergic conjunctivitis. There are also low proportions of mononuclear and polymorphonuclear inflammatory cells in the exudates.[1]

Clinically, vernal keratoconjunctivitis consists of the palpebral, the limbal and the mixed types.[3,6]

The palpebral form of VKC is characterised by giant, flat-topped papillae on the superior tarsal conjunctiva that gives a "cobblestones" appearance.[6] Giant papillae consist of papillary conjunctival masses greater than 1mm in size on the upper tarsal conjunctiva, with a proliferation of collagen underneath the conjunctival epithelium. Its mechanism of formation is mainly epithelial thickening and fibroblast proliferation.[5,7] The presence of giant papillae signifies prolonged chronic inflammation. It may also indicate poor prognosis. The palpebral VKC is also characterized by sticky exudates and tightly packed papillae in the active phase of the disease which get more separated as the inflammation abates. Approximately 50% of the patients with the palpebral form of VKC have corneal involvement which may range from superficial punctate keratopathy to well-demarcated sterile corneal shield-like ulcer (frank epithelial loss) located superiorly.[6] This may heal leaving behind a vascularised scar. Frank epithelial loss (shield ulcer) results from the abrasion of the underlying cornea from the keratinised epithelium of the giant papillae. It also gives rise to the complaint of foreign body sensation.[11] A fine fibrinous pseudomembrane that is enhanced by heat may accumulate on giant papillae and is known as Maxwell-Lyon's sign.[1] (A pseudomembrane is an aggregate of coagulated exudates that is attached to an inflamed conjunctiva which when removed leaves behind an intact epithelium). A pseudoptosis, mostly unilateral, may be noted. The cause is quite unclear, but could be as a result of palpebral thickening, heavy giant papillae, chronic eye rubbing or inflammatory insult to the levator palpebrae superioris muscle.

Limbal VKC is prominent in black races and females.[6] It has a better prognosis.3 The conjunctiva is thick, oedematous and hyperaemic. The papillae in this form are found at the limbus (corneal-scleral junction) mainly superiorly. At the top of each papilla are gelatinous white clumps of degenerated epithelial cells and eosinophils, called Horner-Trantas dots, seen mainly during the active phase of the disease.[1,6] The conjunctival changes include hyperpigmentation, subconjunctival fibrosis, keratinisation and symblepharon. A pseudogerontoxon (arcus senilis-like haze) is often noted in the cornea adjacent to the limbal papillae.[1,16] It is often seen as a waxing and waning grey-white lipid deposit in the peripheral, superficial stroma. Pseudogerontoxon is clinically important because it is an indication of previous allergic eye disease. Papillae can be differentiated from follicles by their red centres which consist of dilated blood vessels at the core of the papillae, surrounded by inflammatory cells including lymphocytes, plasma cells and eosinophils. Papillae consist of hyperplastic conjunctival epithelium thrown into numerous folds and can only develop in the palpebral and bulbar conjunctiva at the corneo-scleral junction (limbus). Follicles, on the other hand, are most prominent in the forniceal conjunctiva and consist of hyperplasia of lymphoid tissue within the stroma. They are clinically seen as multiple, discrete, slightly elevated lesions reminiscent of small grains of rice surrounded by tiny blood vessels.[3] Follicles are commonly seen in viral, chlamydial infections, Parinaud oculoglandular syndrome and hypersensitivity to topical medications.

The mixed form of VKC has the characteristic features of both the palpebral and the limbal forms.

Micropannus is often common in both palpebral and limbal VKC, but gross pannus is unusual.[1] Neovascularisation of the cornea may follow.[6] Conjunctival scarring usually does not occur unless the patient is treated with cryotherapy, surgical removal of papillae, irradiation or other damaging procedures.[1] Impairment of vision is common in cases with shield ulcers and corneal plaques. The disease may be associated with ectatic corneal diseases such as pellucid marginal degeneration and keratoconus.[1,3] Amblyopia seen among patients with VKC may be caused by corneal opacity, irregular astigmatism and keratoconus.[16]

The palpebral form of VKC is more prevalent In Europe and the Americas, whereas the limbal and the mixed forms are common in Africa and Asia respectively, with some geographical variation.[5,7]

Challenges

Allergic eye diseases and vernal keratoconjunctivitis, in particular, have been a thorn in the flesh of eye care providers and patients in warm climates. This ugly scenario has been attributed to the large percentage of the populace affected by the disease.[6] Khaksar and Bagheri, in an epidemiological study of VKC in Kashan (in Iran), stated that approximately 3% of the patients seeking eye care services in outpatient clinic of the Ophthalmology Department BVH, Bahawalpur had VKC and more than 80% are below 18 years of age.[10] Also, Hall and Shilio of the Ophthalmology Department, Kilimanjaro Christian Medical Centre, Moshi, Tanzania stated that over 25% of the total children seen at a tertiary referral paediatric eye clinic in East Africa had VKC and more of the children came flocking to screening clinics complaining of itchy eyes.[6]

The unstable and unpredictable global weather conditions have increased the vulnerability of affected individuals who are unable to predict effectively exposure periods and take adequate precautionary measures.

The management is time-consuming and frustrating both to the patients and the eye care providers, in that patients keep coming with the same complaint often without appreciable remedy.[6] The patients often seek different eye clinics and medicine dealers in search of cure when symptoms become unbearable. Sometimes, they may have suffered the affliction many years before being properly diagnosed.

The morbidity rate of blindness from the disease is high. Some patients that develop corneal ulcers may go blind due to corneal opacification.[6] Other visual impairments such as glaucoma, dry eye syndrome, or cataract may result from uncontrolled use of steroids.[16]

Some continents like Sub-Saharan Africa and the Middle East, where the disease is more prevalent, are populated by individuals living in abject poverty and unhygienic environment. The high costs of the potent topical medications for the disease are beyond the means of the populace. This compounds the problem, leaving the patients with no option but resort to the use of local concoctions, which may give rise to diverse ocular complications and blindness. On the other hand, some of these potent ocular medications are not readily available. When available, some are adulterated, thereby making disease response inadequate. The available effective drugs may not be judiciously used by some patients as prescribed, thus limiting management.

Inadequate man power to cater for the ever-increasing number of patients with ocular allergy is another problem. This has led to patronization of quacks with the resultant effect of visual impairment.

It is often said that he who is not informed is deformed. Lack of awareness of the disease by the patient, and the inability of some eye care providers to provide the patients with firsthand information of the nature, course and predisposing factors associated with their ailment constitutes another setback. This leaves the patient with the option of seeking information from the wrong channel which may even aggravate their condition.

The lack of standardized diagnostic criteria, absence of an established gold standard treatment, and lack of common language among physicians regarding the severity of VKC renders the disease more difficult to diagnose and treat.[16]

In order to tackle these challenges directly, eye care providers should have first-hand information of the nature of the disease, recognition of its clinical features and potential blinding complications.[6]

Given the chronic nature of the disease, regular and adequate treatment through proper patient education regarding what to expect and the pit falls of therapy as well as long term follow-up is essential.[18] This can be backed up by patient information leaflets as a way of breaking the cycle of inadequate treatment, misuse of medication, noncompliance, and resulting frustration.[6] Discouragement of self-medication through effective enlightenment campaigns by eye care providers and provision of potent low cost ocular medications will go a long way toward ameliorating the problem. However, psychological support may be necessary in severe cases.

Training and re-training of eye care professionals is of utmost importance to equip them with the latest developments in eye care profession. The use of community health care providers in reaching out to the vast populace in the rural areas far from health facilities should be encouraged. The public should be advised about the benefits of personal and

environmental hygiene. Government and non-governmental organizations should collaborate in bringing heath care resources closer to the people for prompt and effective dispensing of treatment.

Medical outreach programmes can be organized by the eye care professionals. Policy makers should endeavour to incorporate physical and health education in the school curriculum from primary to tertiary level to raise awareness of common ocular and other diseases.

Government and non-governmental agencies should support medical research institutes and researchers with readily available funds and research grants. This will permit in-depth study of the disease and other ailments, with the aim of establishing standardized diagnostic criteria and a universally acceptable model of treatment.

Treatment

Vernal keratoconjunctivitis treatment involves alleviating the severity of the symptoms and preventing serious sight threatening complications. Permanent visual impairment can result from improper management of the disease.

The first step in the management of vernal keratoconjunctivitis is patient counselling. This is aimed at educating the patients on the nature, course and predisposing factors associated with the diseases.[6] This will go a long way toward good adherence to the symptomatic management and avoidance of those environmental factors that may exacerbate the disease.

Topical mast cell stabilizers such as Alomide (Lodoxamide Tromethamine 0.1%), Alocril (Nedocromil Sodium 2%), and Crolom (Cromolyn sodium 4%) can be given in mild to moderate cases. They should be used regularly 3-4 times daily even when the symptoms have subsided to prevent the release of histamines from the mast cells. However, their use after the symptoms have occurred is of no value because their action is not immediate. They can be used for a long time, for they do not have the side effects of steroids. They are not usually adequate when the eyes are severely inflamed, when keratitis is present, or when there is a vernal ulcer. In these cases, topical steroids should be used to control the inflammation and mast cell stabilizers added as adjunctive therapy to give room for steroid tapering.[3,5,6,7]

Topical mast cell stabilizing- antihistamines such as livostin (Levocabastine hydrochloride 0.05%), patanol (Olapadine Hydrochloride 0.1%), zaditor (Ketotifen Fumarate 0.025%) and optivar (azelastine 0.05%) are good in managing mild to moderate cases.[1,14] They act by reducing eosinophil activation and cytokine release.[1] Oral antihistamines like Benadryl 25mg three times daily are also effective in mild to moderate cases in suppressing other symptoms of allergy but are of limited benefit in the eye.[3]

Topical decongestants such as epinephrine, naphazoline, and phenylephrine may play an effective role in mild to moderate VKC by producing vasoconstriction of superficial vessels, reducing hyperaemia, chemosis and other symptoms and by retarding the release of chemical mediators.[11,17] Cold compresses, ice packs, preservative free artificial tears and ointments may play a helpful role in diluting the antigens, thereby reducing the severity of the symptoms, but are irrelevant in managing onset of keratitis. Sleeping (if possible also working) in a cool, air conditioned room can keep the patient reasonably comfortable. Probably the best remedy of all is to move to a cool moist climate.[1,8]

Topical non-steroidal anti-inflammatory drugs (NSAID) such as Acular (Keteorolac Tromethamine 0.5%), Diclofenac (voltaren) may provide significant symptomatic relief in moderate cases, but may retard the re-epithelialization of a shield ulcer.[16] These agents act primarily by blocking the synthesis/release of prostaglandin through inhibition of cyclooxygenase, the enzyme that catalyses the conversion of arachidonic acid to prostaglandins.[1] However, a careful follow-up is required as corneal melting has been reported after instillation of several types of NSAID.[5]

A short term course of systemic and or topical steroids such as Pred Forte, Lotemax (loteprednol etabonate 0.5%), Alrex or Pred Mild (prednisolone 0.12%, Allergan) and Efemoline followed by cold packs and histamine blocking agents can be used in severe cases as well as in those presenting with corneal involvement.[7,14]

Steroids should be avoided as the first line of defence in the treatment of mild to moderate VKC because of their long term side effects.[16] In using steroids, those with low intraocular absorption such as Fluorometholne, Lotoprednol, or Efemoline should be used first as they have less or no effect in intraocular pressure (IOP). Stronger steroids such as Prednisolone, Dexamethasone, or betamethasone should be used with adequate supervision only when the above-mentioned first choice steroids have proven ineffective.

Steroids inhibit mediator biosynthesis and disrupt intercellular communication by preventing the release of lymphokines. They are the most effective and best proved treatment for VKC especially in active keratitis.[5,17]

Steroids should be used frequently initially and tapered to a stop once the acute stage of the disease is stabilized (usually a few weeks). Prolonged use of steroids should be avoided since it is too often followed by herpes simplex keratitis, increase

in IOP, cataract, glaucoma, delayed wound healing, dry eye syndrome, fungal and other opportunistic corneal ulcers.[6,9,16]

Supratarsal injections of both long acting steroids (e.g. Triamcinolone acetonide), and short acting steroids (e.g. Dexamethasone sodium succinate) may be used for those not responding to conventional treatment.[6,10]

Steroid-antibiotic combination eye drops should be avoided as VKC is an allergic inflammation rather than an infection. In cases of secondary bacterial infection, topical antibiotic eye drops and/or ointment should be used to provide adequate therapeutic dose to combat the infection.

Topical Cyclosporine A (CsA) drops (1-5%) e.g. Restasis, in olive oil or castor oil 4 times a day can be used as an alternative to steroid injection in severe cases.[6,7,12] Cyclosporin A is a calcineurin inhibitor that binds to cyclophilin, an intracellular protein, which in turn prevents the formation of interleukin-2 and the subsequent recruitment of activated T cells. It also inhibits histamine release from mast cells. Through reduction of IL-5 production it may reduce the recruitment and the effect of eosinophils on the conjunctiva.[12,16,19] It has been used successfully in several studies to treat severe cases of VKC. It does not cause an increase in IOP. It is a potent immunosuppressive agent used in the prevention of transplant rejections. It plays an important role in the healing of vernal shield ulcers but recurrences may occur at lower concentrations, which may mandate increasing the concentration. Its use in VKC management is limited due to its high cost and unavailability of commercial preparation of topical CsA in higher concentrations.[13,16,19]

Tacrolimus is a potent drug, similar to CsA in its mode of action, but chemically distinct. Case series and placebo controlled randomized control clinical trial has shown a beneficial effect of tacrolimus ointment (0.1% to 0.3%) and drops (0.005% and 0.1%) in severe VKC. However, it irritates the eyes and makes patients susceptible to opportunistic infections and herpes simplex keratitis. It has a limited use in the developing world due to its high cost.[21]

Tenacious mucus adhering to the cobblestones in particular, thick, ropy strands of mucus as well as filamentary keratitis can be treated with Acetylcysteine. It is known to break the disulfide bonds, thereby dissolving the mucus.[16] It is formulated from commercially available Mucomyst, diluted to a 5% or 10% solution with artificial tears, and is applied four times a day. It is also effective in treatment of shield ulcer in conjunction with aggressive cycloplegia (homatropine 5% or scopolamine 0.25%, bid) and topical antibiotic drops-Ciloxan (ciprofloxacin, Alcon); Ocuflox (ofloxacin, Allergan) or Quixin (levofloxacin, Santen).[11,16,19]

Short-term, low-dose, topical mitomycin-C 0.01% has been considered for treating acute exacerbations in patients with severe VKC refractory to conventional treatment. It inhibits fibroblast proliferation and decreases mucus discharge, conjunctival hyperaemia and limbal oedema. However, unavailability of commercial topical preparations, limited comparative studies, and the lack of data on the safety profile and long-term outcomes are major limitations in recommending mitomycin for the treatment of VKC.[16,20]

Debridement/lamellar keratectomy of early mucus plaques at the base of the vernal ulcer and intensive topical steroids may speed up the re-epithelialization process. Also, low-water-content bandage contact lenses are helpful in the treatment of these defects as well as reducing the interaction between the lid and cornea.[5,6,16]

Free autologous conjunctival graft resection of giant papillae facilitates the re-epithelialization of non-healing shield ulcer. Persistent corneal epithelial defects can be treated by amniotic membrane transplantation and limbal epithelial cell transplantation. Vision improves significantly after transplant. Corneal epithelial cell transplants could be beneficial when amniotic membrane transplant is not sufficient to restore the ocular surface.[3,12,16]

Other general measures in the management of VKC include: dark goggles (sunglasses), visors and caps to prevent photophobia. Identification of the non-specific triggering agents which could aggravate symptoms such as strong wind, dust, air pollutants and strong sunlight and avoiding them are of optimum importance but this is often impractical. Frequent hand and face washing with clean cool water or rinsing the eye with an adequate amount of cool normal saline is also important to reduce the severity of the symptoms and avoid secondary infection.

Case Presentation

A 13-year-old male patient presented to the clinic with complaints of severe itching, discharge, blurry distance vision with object distortion and discomfort with light in both eyes. The problem started about two and a half months earlier and he had been on chloramphenicol eye drops to no avail. Further probing into his case revealed that his condition was usually made worse whenever he visited their poultry house. He had been known to have seasonal allergies right from birth and had never had an eye examination before. There is also a family history of eczema.

Clinical examination revealed the following: unaided visual acuity OD: 20/60 (6/18), PH: $20/40^{-2}$ ($6/12^{-2}$). OS: $20/60^{+2}$ ($6/18^{+2}$), PH: 20/40 (6/12). The patient was moderately

photophobic with no relative afferent papillary defect (RAPD) in both eyes. The intraocular pressure (IOP) was OD: 16mmHg and OS: 17mmHg. Slit lamp examination revealed dense papillary reaction with some thick strands of mucus adhering on the palpebral surface of the upper lids. There was also moderate papillary reaction of the limbal conjunctiva superiorly and a dark brownish colouration of the conjunctiva around the limbus, more profound at the palpebral fissure in both eyes. Dilated fundus examination revealed normal disc, macula, vessels and peripheral fundus in both eyes. Refraction before dilated funduscopy gave the following results.

Objective refraction (Autorefractor):
OD: +0.25 - 3.50 x 045 VA 20/40 (6/12); OS: +0.75 – 5.50 x 130 VA $20/40^{+2}$ ($6/12^{+2}$)

Subjective refraction:
OD: Pl – 2.00 x 15 VA 20/40 (6/12); OS: +0.25 – 3.50 x 115 VA $20/40^{+2}$ ($6/12^{+2}$)

The patient was diagnosed with mixed vernal keratoconjunctivitis; animal danders were suspected as the triggering agent. He was started on topical steroid Lotemax (Loteprednol etabonate 0.5%) twice daily due to the severity of the disease and topical antihistamine/mast cell stabilizer-Zaditor (Ketotifen fumarate 0.025%) twice daily. He was advised against visiting their poultry house or other animal farms outside their home, coming in contact with pets, and to discontinue the application of chloramphenicol eye drops. He was also advised on the need for proper personal hygiene, regular washing of hands and face with cool clean water, scrubbing of the eyelash with cotton tipped applicator soaked in normal saline to remove the dry exudates. The subjective refraction result was not dispensed due to the suspected compromise in the topography of the corneal surface due to the active nature of the disease. On his follow-up visit two weeks later, there was an improvement of the limbal papillae but the superior tarsal conjunctival papillae persisted together with the dark limbal colouration. He was advised to continue with the two drugs and to return to clinic after one week. On his next visit one week later, there was a complete resolution of the limbal papillae and some resolution of the dark limbal colouration but the superior tarsal papillae persisted with only moderate clearance of mucus adherence. He was then given a supratarsal injection of 0.5ml triamcinolone under local anaesthetic drops as follows: The conjunctiva was anaesthetized with 4% lidocaine hydrochloride drops instilled to both eyes every minute for five minutes, after the course of treatment has been explained and his consent obtained. The upper eyelid was everted. 4% lidocaine hydrochloride soaked cotton tipped applicator was dabbed over it for about 1 minute. The patient was asked to look down and a 26 gauge needle of 1ml syringe containing 0.5ml of triamcinolone acetonide (40mg/ml) was inserted through the conjunctiva into the supratarsal space between the conjunctiva and the Mueller's muscle. Adequate care was taken to avoid the marginal blood vessels. 0.5ml of triamcinolone acetonide was injected after ensuring proper placement of the needle. The potential space between the conjunctiva and Mueller's muscle was ballooned indicating successful placement of the injection. Both eyes were injected and a pressure pad was applied for 2 to 3 minutes to reduce the ballooning of the lid and help spread the injection evenly. He was maintained on Zaditor after the injection but discontinued topical steroid after tapering it one drop daily for 1 week. On his next visit 2 weeks later there was a total resolution of the papillae and the limbal brownish colouration could hardly be noticed. There was no significant increase in the IOP as it stood at 17 mmHg in both eyes. His final optical prescription after resolution was OD: Pl -1.25 x 35 VA $20/30^{+2}$ ($6/9^{+2}$) and OS: Pl – 2.00 x 145 VA $20/20^{-3}$ ($6/6^{-3}$) with VA in both eyes as $20/20^{-2}$ ($6/6^{-2}$). The spectacle prescription was dispensed to him in photochromic form to enhance his vision (as well as eliminating image distortion) and reduce the effect of light. He was placed on regular use of Zaditor to avoid sudden reoccurrence. He was counselled on the nature of his disease and the need to avoid coming in contact with the suspected predisposing agent and the importance of regular clinical checks despite resolution.

Discussion

Vernal keratoconjunctivitis is a seasonal, bilateral, recurrent allergic inflammation of the conjunctiva and the cornea.[2,6] The disease affects children between 3 and years of age, though it may appear earlier than that and continue into adulthood. In some cases, it is often self-limiting and the symptoms resolve around puberty.[6] It is more common among males than females.[1] It is characterized by the presence of bilateral palpebral and/or bulbar conjunctival papillae, photophobia, tearing, burning, thick, ropy, yellow mucoid discharge and mild to severe itching.[3,6,7] It has a high incidence in the warm, dry climates with fewer incidences in cooler climates.[1] It is associated with a positive family history for atopic diseases such as asthma, rhinitis, hay fever and eczema.[4,6,7] In VKC, an allergic response is triggered by an over-reaction of the body's immune system to allergy or foreign bodies. This leads to the release of histamines and other mediators by the immune

system resulting in itching, burning and runny eyes that may become red and inflamed. Classically, it is thought of as a type 1 IgE-mediated hypersensitivity reaction. However, it has been suggested that there is a cell-mediated Th-2 involvement.[3,7,13] Laboratory testing of the conjunctival scrapings reveals higher numbers of eosinophils.[1] Finding eosinophilic granules can aid the diagnosis but is not necessary, except in difficult cases, as VKC is easily diagnosed clinically based on history, signs and symptoms.[14,16]

The main idea in the management of VKC is to minimize inflammation as well as to reduce complications until the disease subsides. Topical treatment is preferable and more effective than systemic. Topical mast cell stabilizers and antihistamines or a combination of both are the first line of choice in the management.[14,16] The drugs are effective for mild to moderate cases and can be used for a long period without adverse effect. Severe and unresponsive cases are treated with steroids and cyclosporine. Non-steroidal anti-inflammatory eye drops are also useful in mild cases. However, detection and avoidance of the triggering agents (allergens), cold compresses, and moving to a cooler climate is a good strategy in ameliorating the severity of the disease.[1,6,14]

In this case, the blurry distance vision resulted from astigmatism brought about by the compromise of the corneal surface due to the activeness of the disease. (This manifests from the discharge, limbal papillae, tarsal palpebral papillae pressing on the eyeball and constant rubbing of the eye due to intense itching which may slightly alter the topography of the cornea). The photophobia results from the painful contraction of an inflamed iris or the excitation of the many pain fibres of the cornea. The discharge comes from the migration of inflammatory cells (e.g. eosinophils) from the conjunctival stroma through the epithelium to the surface. Here they combine with fibrin and mucus from the goblets cells to form the conjunctival exudates[1] Topical mast cell stabilizer (Zaditor) and topical steroid (Lotemax) were prescribed due to the severity of the disease. These produced a good response in resolving the limbal component of the disease. The unresponsive tarsal palpebral component was taken care of by supratarsal injection of 0.5mL triamcinolone acetonide which gave a complete resolution and disappearance of the brownish colouration. The Zaditor was continued after the resolution mainly to avoid a relapse. There was no significant change in the IOP of the patient due to adequate monitoring and close supervision of the drug administration. The refraction result obtained before the resolution of the disease was not dispensed due to its unreliability brought about by the compromise of the corneal surface as a result of the active nature of the disease. However, the result obtained after complete resolution which gave a significant visual improvement was dispensed in photochromic form to reduce the patient's sensitivity to light. Eliminating exposure to the identified triggering agent (animal dander) accelerated the early resolution and should prevent recurrence.

Differential diagnosis includes giant papillary conjunctivitis due to retained foreign body, exposed sutures and contact lens related problems, chlamydial and gonococcal conjunctivitis, chronic blepharitis, atopic keratoconjunctivitis, viral and toxic conjunctivitis, SAC, PAC, phlyctenulosis, band keratopathy and floppy eyelid syndrome.[2,8,14] This is possible through a comprehensive clinical history and ocular examination. AKC rarely occurs in childhood and patients tend to complain of a watery discharge (mucus discharge in VKC).

Conjunctival scraping reveals eosinophils to a greater degree in VKC, the inferior palpebral conjunctiva is more involved (usually superior in VKC), and there is often an associated blepharitis (less likely in VKC).[1,3] Differentiating VKC from the other allergic conditions is usually fairly straightforward: corneal changes are rarely seen in SAC and PAC; in GPC, the ocular irritation is associated with contact lens wear; and in drug induced dermato-conjunctivitis, the offending agent can usually be identified. Symptoms of infective conjunctivitis are normally worse first thing in the morning and the eyelids are often gummed together on awakening due to discharge. In viral conjunctivitis, small conjunctival haemorrhages are common. Tenderness and swelling of the preauricular nodes is indicative of viral conjunctivitis. Bacterial conjunctivitis is characterised by purulent discharge.[1,4,17]

In conclusion, since a gold standard treatment for VKC has not been established, understanding of the pathogenesis of the disease by eye care providers may lead to better therapy for the patients with this unfortunate disease.

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Etty Bitton, OD, MSc, FAAO, FBCLA
AAO Faculty-Student Liaison for Montreal and Committee Chair

Kristine Dalton, OD, PhD, FAAO
AAO Faculty-Student Liaison for Waterloo

Just when you thought the annual meeting of the American Academy of Optometry (AAO) could not get any bigger, this year's meeting in New Orleans, broke the all-time record for the AAO at a whopping 7489 attendees, 448 of which were Canadian. At total of 4368 optometrists attended, along with almost 1400 students, breaking even more records.

The Canadian schools, once again, contributed significantly to the program, both in the continuing education and the scientific programs; Tables 1-4 tabulate the contributions of the Canadian schools to the meeting. (Figure 1)

Figure 1. Waterloo Optometry Students Markio Hirano (left) and Raymond Ho (right) presented their first-ever posters at this year's AAO meeting in New Orleans (pictured with Kristine Dalton, Waterloo Faculty.

For the first time this year, the University of Waterloo, the Université de Montréal and the Centre for Contact Lens Research teamed up to host the Canadian Schools Alumni & Friends Reception, bringing together alumni, friends, faculty and students from around the world. Dr. Paul Murphy (Director of Waterloo) and Dr. Julie-Andrée Marinier (Secretary of Montreal) welcomed the attendees and gave a brief overview of some of the advancements of their institutions. Over 200 people attended the inaugural event and the formidable attendance reinforced the need to make this an annual event moving forward! (Figure 2, 3)

Figure 2. A packed house for the first ever Canadian Schools Alumni & Friends Reception at the AAO in New Orleans

Figure 3: Gina Sorbara (Waterloo) and Etty Bitton (Montreal) at the first joint Canadian school reception

Another mainstay of the annual meeting is the completion of requirements to attain the Fellow of the AAO (FAAO). Candidates who have accumulated 50 points (consult the URL for more details, http://www.aaopt.org/becoming/efellowship) are considered for fellowship and once all the written requirements are completed, they are invited for a peer-review interview process at the annual meeting. This year, 240 new fellows were inducted at the annual banquet representing 15 different countries, with 9 Canadians completing their FAAO.

Once fellowship has been obtained a member can join any of the numerous sections that the AAO offers, such as Anterior Segment, Cornea and Contact Lens, Low Vision, Optometric Education, etc. When Fellows meet requirements specific to a section and demonstrate both knowledge and expertise in their specific topic area, they can be invited to become a "Diplomate" of that section. We are thrilled to congratulate Drs. Catherine Chiarelli (Toronto, ON) and Ronald Gall (Oakville, ON) who both successfully completed their Diplomate of the Binocular Vision, Perception & Pediatric Optometry (BVPPO) Section. The 2015 class of Canadian FAAOs and Diplomates can be found in Table 5. Congratulations to all!

Figure 5. Dr Raiju Babu (Waterloo) congratulated on his FAAO by Drs Lyndon and Debbie Jones (left) and Dr William Ngo (right)

Figure 6. Drs. Catherine Chiarelli (Toronto) and Ronald Gall (Oakville) receiving their Diplomate of the Binocular Vision, Perception & Pediatric Optometry (BVPPO) Section

Figure 4. Dr. Caroline Faucher (Montreal) receiving her FAAO

In addition to the numerous new fellows and diplomates from Canada this year, there were a number of new student fellows from both optometry schools. The student fellowship program was created by the AAO in 2011 to encourage optometry students to be come more involved in the meeting and experience all of the events the Academy has to offer. This year 32 students from Montreal and 4 from Waterloo completed their student fellowships. Congratulations students!

As always, the AAO and the AOF continue to provide numerous grants, scholarships, and travel fellowships, through the support of industry and the generous donations of individuals. These awards support and encourage faculty, as well as optometry and vision science graduate students to participate in the meeting by lessening its costs, and recognizing their dedication and hard work. There were a number of recipients from both Schools at this year's meeting, as listed on right. Congratulations!

AOF –VSP/FYi Doctors Scholarship
Carolyn Perugino (4th yr student, Montreal)
Wylie Tan (4th yr student, Waterloo)
Morgan Wellburn (4th yr student, Waterloo)

Essilor Student Travel Fellowship
Maxime MacGregor (3rd yr student, Montreal)
Roxanne Côté-Castonguay (3rd yr student, Montreal)
Lor Sildiryan (3rd yr student, Waterloo)
Jessica Yang (4th yr student, Waterloo)

Section on Cornea, Contact Lenses & Refractive Technologies-Resident Travel Fellowships
Stephanie Britton (Cornea & Contact Lens resident, Waterloo)

Student Travel Fellowship funded by an educational grant from The Vision Care Institute
Vivek Labishetty (PhD candidate, Waterloo)
Lacey Haines (PhD candidate, Waterloo)

Ezell Fellowship
Dr. William Ngo (PhD candidate, Waterloo)

AOF Innovation in Education Grant
Dr. Nadine Furtado (Faculty, Waterloo)

Finally, every year the AAO takes time to honour outstanding clinicians, educators and researchers. This year, a Waterloo alumnus, Dr. Austin Roorda and his colleagues, were awarded the Garland W Clay Award as the authors of the manuscript published in Optometry & Vision Science that received the most citations in the world in the previous five years. Dr. Roorda completed his PhD in 1996 under the supervision of Drs. Bill Bobier and Beth Irving. Congratulations Austin and colleagues!

Next year's meeting promises to be as popular as ever and is set for Nov 9-12, 2016 in Anaheim, California. Mark your calendars!

Table 1. Presentations by faculty of the École d'optométrie, Université de Montréal

Author (s)	Title of presentation	Type of presentation
Bitton, E	Daily Disposables: Taking it one day at a time	CE Conference (1 hr)
Bitton E, Kronish S, Jones L, Bouchard JF	The impact of hand rinsing time on soap residue left on the surface of silicone hydrogel CL	Poster
Bachir V, Bitton E	Treatment of Demodex with Hypochlorous acid: A Case report	Poster
Kergoat H, Chriqui E, Law C, Kergoat MJ, Leclerc BS.	Visual impairment in older institutionalized adults with neurocognitive disorder.	Poster
Marinier JA, Camirand-Larue E, Morgan T, Overbury O	Evaluation of answers to Massof's Activities Inventory and health perception by visually impaired patients, aged at least 65 years old living independantly at home	Poster
Michaud L, Morency J, Giasson C.	Evaluation of oxygen under scleral lenses fitted with different fluid reservoir thickness	Poster
Michaud L. Diaconu V. Garon ML Forcier P	Optic Nerve Capillaries Blood oxygenation Investigation in Fabry's patients	Poster
Michaud L	Contact Lenses and Dry eye : Cause or Remedy?	CE Conference (1 hr)
Michaud L. Brazeau D	It is all about scleral lenses: Advanced Tips for Practitioners	CE Conference (2 hrs)
Tan B, Yuen T, Moy A, Zhou Y, Graham A, Michaud L, Lin M	Pilot study of post-lens fluorogram with scleral lenses of various clearances	Paper
Yuen T, Tan B, Moy A, Graham A, Michaud L, Lin M	Peripheral tear mixing under scleral lenses fitted with various clearances	Paper
Wittich W	Validation of the Purdue Pegboard for Older Adults with Low Vision	Poster

Table 2. Presentations by faculty of the School of Optometry and Vision Science, University of Waterloo

Author (s)	Title of presentation	Type of presentation
Christian LW, Nandakumar K	Visual status in school children with reading problems in Canada	Poster
Christian LW, Bobier WR	Photographic leukocoria	Poster
Mitchell GL, Benavente-Perez A, Handford P, Oechslin T, Christian LW, Irving EL, Jenewein E, Kulp MT, Patel R, Schnell PH	Validity of an online, self-administered symptom questionnaire in patients with accommodative dysfunction	Paper
Dalton K	Establishment of a new sports vision clinical service at the University of Waterloo School of Optometry & Vision Science	Poster
Dalton K	Ezell Fellows Present: Tackling Traumatic Brain Injury - Can our eyes be the window to the brain?	Symposium Lecture
Dalton K, Willms A	Establishment of standard measures of visuomotor reaction time	Poster
Freddo T	Understanding granulomatous disease in optometric practice	CE Conference (1 hr)
Bitton E, Kronish S, Bouchard JF, Jones L	The impact of hand rinsing time on soap residue left on the surface of silicone hydrogel CL	Poster
Guthrie S, Woods J, Dumbleton K, Fonn D, Jones L	Contact lens discomfort management strategies of ECPs	Poster
McCabe K, Piper S, Jones L, Papinski D, Fadli Z	In-vitro bacterial adhesion to silicone hydrogel contact lenses: Does a surface coating inhibit bacterial adhesion?	Poster
McCabe K, Piper S, Jones L, Papinski D, Fadli Z	Bacterial adhesion to silicone hydrogel and conventional hydrogel lenses	Paper
Panjwani F, Papinski D, Woods J, Jones L	In-vivo dehydration of omafilcon A and delefilcon A	Poster
Panjwani F, Papinski D, Varikooty J, Woods J, Jones L	In-vivo dehydration of stenfilcon A and delefilcon A silicone hydrogel materials	Poster
Schulze M, Luensmann D, Ng A, Panjwani F, Srinivasan S, Jones L	The relationship between the positioning of multifocal contact lens optics and satisfaction with vision	Poster
Subbaraman L, Heynen M, McCanna D, Omali N, Jansen M, Fadli Z, Toubouti Y, Coles-Brennan C, Jones L	Impact of pigment presence in etafilcon A on in vitro interaction of lysozyme and impact on inflammatory biomarker release	Paper
van Doorn K, Subbaraman L, Lemp J, Maissa C, Jones L	Reversibility of pollen adhesion to contact lenses	Paper
Leat SJ, Lewis, A, Werner J, Figuero M Flannagan M	Vision in Aging SIG and Public Health and Environmental Vision Section symposium: Aging in the International Year of Light	Symposium Moderator
Cheong AMY, Lam HY, Siong KH, Chan HHL, Tsang WWN, Leat SJ	Effects of eye movements on postural sway in community dwelling older adults	Poster
Cheong AMY, Lam HY, Siong KH, Ting PWK, Tsang WWN, Leat SJ	Dynamic grating acuity in community dwelling older adults	Paper
MacIver S	Ellerbrock Presents: Grand Rounds VIII: Thickening of the ELM on SD-OCT: a novel method of detecting early Stargardt's disease and prognosis of the disease	Symposium Lecture
MacIver S, Furtado N, Reed K, Ruskin D, Chous P, Harrison W, et al.	Vitamins and other nutrition factors found to improve macular pigment optical density and contrast sensitivity in optometry convention attendees	Poster
Situ P, Begley C, Simpson T, Li Y, Becker LE, Thibos L	Schlieren optics to visualize and cabilrate a novel pneumatic esthesioemeter - a preliminary report	Poster

Table 2. con't

Author (s)	Title of presentation	Type of presentation
Chalmers, RL, Wagner, H, Sorbara, L, Mitchel GL, Kinoshita BT, Lam DY, Richdale K, Zimmerman A, Collier S, Cope J, MacGurn MM, Rao M, Beach M, Yoder J. (Collaboration between Centers for Disease Control and Prevention and CLAY Study Group)	Is purchasing lenses from the prescriber associated with better soft lens wearers habits?	Poster
Zimmerman, A, Richdale, K, Mitchell, GL, Kinoshita, B, Lam, D, Wagner, H, Sorbara, L, Chalmers R, Collier S, Cope J, MacGurn A, Rao MM, Beach M, Yoder J. (Collaboration between Centers for Disease Control and Prevention and CLAY Study Group)	Contact lens wear and water exposures	Poster
Subbaraman, L	Contact Lens and Ocular Physiology Papers	Session Moderator
Thompson B	ARVO/AAO Joint Symposium sponsored by the Binocular Vision, Perception and Pediatric Optometry Section: Recovery of visual function in adults with amblyopia	Symposium Lecture
Anstice N, Paudel N, Denny S, Shea K, Thompson B, Jacobs R	Effect of astigmatic defocus on visual acuity measured with Cardiff acuity cards, Lea symbols, and Sloan letters in adults	Poster
Babu R, Raveendran R, Erkelens I, Chow A, Bobier W, Thompson B	Saccadic eye movement latencies in adults with amblyopia: the role of suppression	Paper
Gao T, Babu R, Dai S, Bobier W, Anstice N, Black J, Thompson B	Treatment of Amblyopia in Adults with Refractive Correction Alone	Paper

Table 3. Presentations by optometry students from Canadian schools

Author (s)	Title of presentation	Type of presentation
Gagné S, Labelle M, Marinier JA, Faucher C (Montreal)	The assessment of clinical reasoning development in low vision using the script concordance test	Poster
Hirano M, Hutchings N, Simpson T, Dalton K (Waterloo)	Investigation of the validity and repeatability of a novel system designed for the measurement of dynamic visual acuity	Poster
Hirano M, Hutchings N, Simpson T, Dalton K (Waterloo)	The Effect of Optotype Velocity and Trajectory on Dynamic Visual Acuity	Paper
Ho R, Babu R, Thompson B, Dalton K (Waterloo)	Ocular Dominance Varies With Test Distance	Poster
Perugino C, Charette S, Bitton E (Montreal)	Tear lubricant osmolalities	Hybrid poster (paper/poster)
Sayah D, Asaad K, Hanssens JM, Giraudet G, Faubert J (Montreal)	Differences in visually induced postural responses between myopes and emmetropes	Poster
Lampasona V, Langevin C, El-Zoghbi N, Hanssens JM, Bouchard JF (Montreal)	Evaluation of the preservation period of diagnostic eye drops	Poster

Table 4. Presentations by Residents and Graduate students from Canadian Schools

Author (s)	Title of presentation	Type of presentation
Defalque H, Michaels J, Kapusta M, Overbury O (Montreal)	Fixation patterns after macular hole surgery: structural and functional recovery	Poster
Erkelens I, Bobier WR (Waterloo)	Vergence adaptation shows main sequence effect.	Poster
Haines L, Sorbara L (Waterloo)	Comparison of sagittal height of keratoconus and normal eyes using a novel eye surface profiler and Visante OCT	Poster
Labhishetty V, Bobier WR (Waterloo)	Are high lags of accommodation in myopic children due to motor deficits?	Poster
Ngo W, Srinivasan S, Jones L (Waterloo)	The impact of an eyelid warming device in the management of meibomian gland dysfunction	Paper
Ngo W, Srinivasan S, Jones L, Bitton E (Montreal/Waterloo)	Enhancement of clinical observation of *Demodex folliculorum*	Poster
Plourde M, Lebeau S, Corbeil ME, de Guise D (Montreal)	A case of ocular myasthenia gravis combined with a tonic pupil mistaken for partial third nerve palsy	Poster

Table 5. New Fellows and Diplomates from Canada in 2015

Name	Region	Status
Raiju Babu	Waterloo, ON	FAAO
Catherine Chiarelli	Toronto, ON	Diplomate, Pediatrics/BV
Stacy Chong	Toronto, ON	FAAO
Angela Di Marco	Toronto, ON	FAAO
Caroline Faucher	Montreal, QC	FAAO
Ronald Gall	Oakville, ON	Diplomate, Pediatrics/BV
Nalisha Kassam	Calgary, AB	FAAO
Sung (Kelly) Lee	Toronto, ON	FAAO
Sandeep Randawa	Waterloo, ON	FAAO
Jessica Steen	Regina, SK	FAAO
Glenn Wicks	Victoria, BC	FAAO

How to Retain Eyewear Sales and Boost Revenue

Pauline Blachford

Pauline Blachford consults optometrists on how to reduce unbooked appointments, increase eyewear sales, and improve employee productivity. She has abundant experience in the eye health industry, including 17 years at White Rock Optometry in BC. Pauline frequently presents at optometry conferences and is a regular columnist for the CJO. For more information, visit paulineblachford.com.

You and your team know your patients' unique eyewear needs, and have the expertise to provide them with the perfect product. This alone, however, isn't necessarily enough to ensure your patients buy their glasses and contact lenses from you, their trusted optometry team.

Whether they're chasing affordability or a specific style, one out of three eyeglass patients fill their prescriptions elsewhere.[1] In fact, in the U.S. market, the top four companies in the eyewear industry are strictly retailers, and collectively account for 45% of the market's revenue.[2] That figure doesn't even include Walmart, Costco or 1-800 Contacts.

Despite being a highly competitive and increasingly concentrated market, eyewear sales are worth the bother, and on average, anywhere from 40-60% of an independent optometrist's bottom line. An American optometry practice with an annual gross revenue of $750,000 sees about $160,000 of potential eyewear revenue walk out the door each year.[3]

To retain current eyewear sales and reclaim some of the business that goes elsewhere, here are several strategies small and medium-sized practices can implement to maximize their strengths and boost their bottom line.

Trust and personality

Loyal customers buy more, buy more frequently and buy higher-end products than any other type of customer.[4] In short, it's worth it to your practice to invest in strengthening your patient relationships.

Research suggests that patient loyalty is developed through continuity of care, and a greater personalization of service.[5] When customers had the opportunity to create a positive, emotional bond with service representatives in the retail-banking sector, 85% of customers purchased or invested more.[6] And generally speaking, 70% of buying experiences are based on how a customer feels they are being treated,[7] and as many as 81% of customers would pay extra for better service.[8]

One way to provide continuity of care and personalized service is to extend the excellent eye care you deliver in the examination room to the level of customer service you provide in the optical department. I wrote about the impact that telephone recalling has on increasing trust in a past *CJO* issue[8] and, obviously, a great handoff after the eye exam is key. Some optometrists I work with have their optician join them in the exam room to discuss the type of lenses being prescribed to the patient. Others introduce the patient to their optician after an exam, and reiterate their eyewear recommendations. Both methods show the patient you will go over-and-above to ensure they get the right eyewear for their unique needs. They also begin to expand doctor-patient trust and loyalty to include your optician.

Patient convenience

Research shows that purchasing directly from their optometrist right after an eye exam is a convenience patients prefer, 10 and smart recalling is the most effective way to ensure a patient comes to your clinic ready to buy.

When booking patients, have your recaller invite them to set aside at least an hour for their visit to your clinic. Clients often underestimate how long it takes to receive great service and have to rush out the door to get to their next appointment before having the chance to shop for their eyewear.

You can also have your recaller ask clients to bring in all their eyewear, including glasses, contact lenses and sunglasses. This small act may uncover that the patient has lost their glasses, or are out of contact lenses. By making the appropriate notes, your recaller can ensure all optometry staff are prepared

to meet each patient's eyewear needs. It also makes patients feel more prepared to make a purchase when they can compare what they will be getting to what they had.

Following the exam, a few small initiatives can help further increase convenience. For example, have your staff encourage your contact lens wearers to purchase a year's supply of lenses and solution. Give them a discount if they do so. This won't have a dramatic influence on your profits, but it will establish that your clinic is a one-stop shop for all your clients' eyewear needs. Also, have your staff complete and mail your clients' insurance rebate forms.

Increasing revenue per patient and closing the sale

From LensCrafters to Walmart, retailers in the eyewear industry are in the business of selling. They are experts in upselling customers and pros at attracting new ones with deals. When independent optometry practices pair their eye care services with effective sales strategies, they too can reap the rewards enjoyed by the bigger eyewear chains.

The opticians I meet when I'm consulting and speaking at conferences always amaze me with their eyewear knowledge and technical skills. But they often confide that they aren't comfortable selling the products. The research confirms this. According to a report prepared by the *Review of Optometric Business*, most optometrists capture only a portion of the existing eyewear and contact lens demand within their patient bases.[11]

It isn't enough to merely explain to patients the features and benefits of the lenses you offer, and at worst, inundating patients with information may turn them off a sale. Having your optician complement their eyewear knowledge with sales training will give them the skills to close a sale, including how to ask the right questions, and how to listen to a patient's needs. Plus, the opticians I've trained in sales strategies always report feeling more confident, which is half the battle when it comes to sales.

Every practice is unique, but when independent practices exploit their strengths - particularly when it comes to providing excellent service - optometrists can not only avoid losing sales to bigger retailers, but they can boost their eyewear sales across the board.

To learn how you can develop an effective business strategy to increase eyewear sales or for sales training for your opticians, please email me at info@paulineblachford.com or visit paulineblachford.com.

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10 Leasing Tips for Optometry Tenants

By Jeff Grandfield and Dale Willerton – The Lease Coach

Dale Willerton is the founder of The Lease Coach and Jeff Grandfield recently joined him as partner. Dale and Jeff are commercial lease consultants who work exclusively for tenants, and are also professional speakers and co-authors of Negotiating Commercial Leases and Renewals For Dummies. *Got a leasing question? Need help with your new lease or renewal?*

Call 1-800-738-9202, email DaleWillerton@TheLeaseCoach.com, or visit www.TheLeaseCoach.com. For a copy of our free CD, Leasing Dos & Don'ts for Commercial Tenants, please email your request to DaleWillerton@TheLeaseCoach.com.

New and existing optometry tenants will specialize in proper patient care, but may fall short in knowing about their own commercial leases. Many optometry tenants in fact will either blindly sign a commercial lease document or a lease renewal without completely reading it, trust the landlord to charge a 'fair rental rate,' and/or fear asking what may seem like too many questions. As we emphasize in our new book, Negotiating Commercial Leases & Renewals For Dummies, the commercial lease is a binding legal document and not fully knowing or understanding what you are signing (and agreeing to …) can be a serious mistake as you may sign for an inappropriate lease term, miss out on valuable tenant inducements offered by the landlord, and agree to pay too much monthly rent.

Before you sign a commercial lease or a renewal, know what you are getting into and look before you leap. Here are some proven tips to help get you the best commercial lease deal possible:

Negotiate to Win: This should be the goal of the optometry tenant. Why? Because this is the goal of the landlord, the landlord's property manager, and the landlord's real estate agents. The landlord is not necessarily looking for a "win-win" lease deal (thereby benefitting all parties …). Instead, a typical landlord charges the tenants as much rent as possible and who would expect anything less?

Optometry tenants may mistakenly think that landlords set their rental rate based on what they think the tenants can afford to pay. But landlords really set rental rates based on the cash flow they need to service their mortgage, manage the property, and make a profit. The problem is rather than negotiating to win, most tenants are simply negotiating not to lose. This happens for several reasons including a lack of experience, knowledge, and time. Tenants may often face a fear of rejection (with asking for what they want) as well.

Whether you are negotiating your first commercial lease or a lease renewal, don't rely on the landlord to give you what you deserve. With effective negotiation, many benefits may be available to optometry tenants – these include the lowest possible monthly rental rate, the longest free rent period, the most signage and/or parking, and the biggest tenant allowance.

Negotiate All Lease Terms at Once: Resist the urge to look at your lease as a list of individual points that must be all negotiated separately. All the business terms contained in your lease are connected and must be negotiated collectively. For example, don't agree to the rental rate until you agree to the length of the lease term.

Don't Telegraph Your Intentions or Give Buying Signals: What we mean here is to check your emotions and what you say when dealing in commercial real estate. To explain, if you're planning to open another location or relocate your practice and your prospective new landlord (or his real estate agent) asks you what you're paying now in your current location, be aware that this is a loaded question. If you're paying $28.00 per square foot in your current location but they were only going to ask $23.00 per square foot, they could well increase that rental rate because they think that you can afford to pay it. Also, if a tenant reveals somehow that emotionally or mentally he's already decided to renew his current lease – or, if during a space showing, your business partner is gushing over how nice it all is and how perfect the space would be for you, then get ready to pay a higher rent.

Protect Yourself by Incorporating: There are a couple of good reasons to incorporate or form a limited liability company (LLC) if you're about to go into business and lease a commercial location. Businesses fail, often through no fault of the business owner, and incorporation can help protect you.

If you allow the landlord to put your personal name as the tenant on the lease agreement (or even the letter of intent or offer to lease), then you are personally responsible for rent payments and all other terms of the lease agreement. By incorporating, you are generally off the hook personally and transfer these obligations to the tenant corporation.

Change the Day Your Rent is Due: For many small business-owners, the end of the month is not a pleasant time. Staff payroll, loan payments, and commercial rent are all due. Sometimes having a few days' grace period to make the monthly rent payment can make a world of difference. The Lease Coach often negotiates with the landlord to change the tenant's rental payment date (from month end to perhaps the fifth or tenth day of the following month). If you're successful getting the landlord to agree to this proposal, make sure you get it in writing in the lease or a proper lease-amending agreement stating it.

Creatively Build on Your Relationship with the Landlord: Making deposits to your relationship with your landlord can be invaluable. When you think about it, the tenant is the landlord's customer and the landlord should be trying to initiate or sustain a good relationship with the tenant. But because you may only have one landlord, and the landlord may have hundreds of tenants, it often falls to the tenant to build and foster these relationships. As The Lease Coach, we've always leased commercial office space for our business and found doing this can be extremely effective – offer a bottle of cold water on a hot day, wrap up a bottle of "holiday cheer" at Christmas time, and extend plenty of thanks and appreciation for their work.

Ask the Property Manager or Landlord for a Favor: Have you ever agreed to do something you really didn't want to do, but knew the other person would be beholden to you if did it? We have, both personally and professionally, and even in commercial real estate. Simply asking someone for something in which there is no promise of return or benefit to the other party seldom seems to work. But if you ask for a favor and say those magic words, "I owe you one" or promise a specific future benefit, you're more likely to get your favor. Remember to pause after you ask and let them respond. The tendency is to justify or over-explain. Sometimes asking by e-mail is fine, but doing it verbally gives you a chance to provide more details if the property manager doesn't fully understand your request (or initially turns you down).

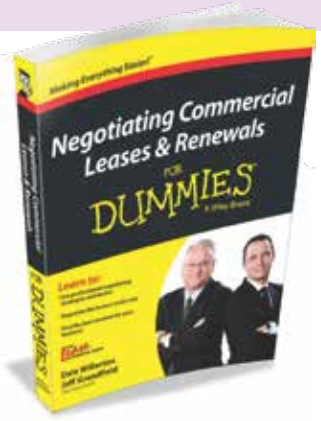
Prepare for Murphy's Law: Anything that can go wrong will go wrong – and at the worst possible time. Emotionally and financially, it pays to plan ahead and try to envision what will go wrong. Say the contractor doesn't get your new space built out in time and you're going to open for business five weeks late. Do you want to be paying rent while you're not open for business? Of course not. In this case, The Lease Coach will often negotiate wording into a tenant's lease agreement so the rent doesn't start until the tenant actually opens for business.

Get Professional Help with the Leasing Process: We've spoken with commercial tenants who were relying on their brother, sister, mother, father, spouse, and/or residential real estate uncle to help them negotiate their leases. Granted, these people may know more about leasing than the average tenant; however, they surely are no experts! A professional lease consultant with a proven track record who earns a living serving commercial tenants is your better choice. This so important that it can be the deciding factor in whether your business succeeds.

For a copy of our free CD, Leasing Dos & Don'ts for Commercial Tenants, please e-mail your request to DaleWillerton@TheLeaseCoach.com.

Dale Willerton and Jeff Grandfield - The Lease Coach are Commercial Lease Consultants who work exclusively for tenants. Dale and Jeff are professional speakers and co-authors of Negotiating Commercial Leases & Renewals For Dummies (Wiley, 2013). Got a leasing question? Need help with your new lease or renewal?

Call 1-800-738-9202,
e-mail DaleWillerton@TheLeaseCoach.com
or visit www.TheLeaseCoach.com.



Mieux vaut prévenir pour le bien de votre pratique

Par Maggie Green et Brian Gomes

Brian Gomes est président et directeur général de BMS Canada Risk Services Ltd. (groupe BMS). Il travaille avec bon nombre des plus grandes associations de professionnels de la santé du pays pour concevoir et offrir des régimes d'assurance personnalisés à plus de 300 000 membres de professions de la santé réglementées au Canada. Brian possède plus de 10 ans de connaissances et d'expérience de l'industrie et on lui demande souvent de donner des exposés ou des conférences sur la responsabilité et les structures de risque pour les groupes. Titulaire de plusieurs titres professionnels, il est considéré comme un expert national et mondial dans les domaines de la faute professionnelle médicale et de la responsabilité professionnelle.

Maggie Green
Professionnelle de la santé autorisée et titulaire d'une maîtrise en qualité, risque et sécurité des soins de santé, Maggie possède plus de 10 ans d'expérience dans le secteur des soins de santé. Elle est souvent invitée à titre de conférencière sur la responsabilité professionnelle, la gestion des risques, la sécurité des patients et les tendances médico-légales dans l'exercice de la profession. À titre de chef nationale de l'exercice de la profession au groupe BMS, Maggie offre des services personnalisés de gestion des réclamations et des risques aux organisations de soins de santé et à leurs membres.

À titre d'optométriste, vous avez besoin d'une assurance responsabilité professionnelle pour vous inscrire auprès de votre ordre de réglementation. Nous le savons tous. Toutefois, lorsque vous souscrivez une assurance, pensez-vous à la raison pour laquelle vous en avez besoin et à quel point elle est importante pour l'avenir de votre pratique? Connaissez-vous les détails de l'assurance que vous souscrivez? Êtes-vous certain d'être suffisamment protégé et d'avoir les limites voulues pour répondre à une réclamation?

L'assurance et la responsabilité sont des sujets complexes, tout comme les offres et les polices qui les appuient. Il est important que vous compreniez les différences entre les différentes options d'assurance qui s'offrent à vous afin de choisir la plus appropriée pour les conditions de votre pratique. Nous espérons tous ne jamais avoir besoin de nos assurances, mais nous nous devons de nous assurer que nous avons la bonne protection avant que nous en ayons effectivement besoin.

L'assurance responsabilité professionnelle :

Le contact régulier avec les patients fait partie de la réalité de toute pratique optométrique et constitue un fait quotidien. Cela dit, vous pourriez être confronté à des situations où vos patients cherchent à vous tenir responsable de blessures qu'ils estiment avoir subies.

L'assurance responsabilité professionnelle vous protège contre une responsabilité ou allégation de responsabilité à l'égard de tout préjudice ou dommage découlant d'une négligence, erreur, omission ou faute professionnelle dans le cadre de vos fonctions à titre d'optométriste. Les ordres de réglementation de l'optométrie exigent que tout optométriste agréé qui offre des services, que ce soit contre rémunération ou bénévolement, soit couvert par ce genre d'assurance. L'assurance responsabilité professionnelle vous protège en tant qu'optométriste en garantissant que votre défense devant les tribunaux sera coordonnée et payée en cas de poursuite. L'assurance couvre aussi le coût des indemnisations ou dommages-intérêts accordés aux patients. De plus, vos patients sont aussi protégés parce qu'une assurance responsabilité professionnelle signifie qu'il y a de l'argent pour les indemniser si leur poursuite est fondée.

Si votre ordre exige que vous déteniez une assurance responsabilité professionnelle, il y a également d'autres facteurs à considérer. Êtes-vous propriétaire d'entreprise? Avez-vous des employés? Avez-vous du contenu à protéger? Dans l'affirmative, vous devriez penser à ajouter une protection supplémentaire à votre assurance responsabilité professionnelle individuelle afin de protéger le nom, la propriété et le contenu de votre entreprise.

L'assurance responsabilité professionnelle d'entreprise :

L'assurance responsabilité professionnelle d'entreprise offre une limite de responsabilité distincte pour protéger votre actif commercial advenant le cas où le nom de votre entreprise est mentionné dans une demande introductive d'instance ou une poursuite en justice. Si un incident se produit, il est fort probable que le patient précise non seulement le nom de l'optométriste, mais également le nom de votre entreprise en sa qualité de fournisseur de services. Les propriétaires d'entreprise devraient envisager de souscrire une assurance responsabilité professionnelle d'entreprise si d'autres optométristes (par exemple, copropriétaires, employés, associés) facturent des services sous le nom de l'entreprise. Veuillez noter qu'une seule personne devrait souscrire cette assurance au nom de tous les propriétaires de l'entreprise, les employés et l'entité commerciale.

Si vous êtes travailleur autonome ou propriétaire unique n'ayant pas d'autres optométristes qui facturent des services sous votre nom d'entreprise, votre police d'assurance responsabilité professionnelle de l'ACO protègera automatiquement votre nom d'entreprise.

Mentionnons qu'aux fins de la police de l'ACO, vous n'avez pas besoin de souscrire une assurance responsabilité professionnelle d'entreprise si votre personnel membre d'une profession de la santé réglementée est composé exclusivement d'opticiens et vous n'avez pas non plus à souscrire cette assurance pour votre personnel administratif ou de soutien.

L'assurance commerciale de responsabilité civile générale :

L'assurance commerciale de responsabilité civile générale vous protège contre les réclamations découlant de blessures corporelles ou de dommages aux biens que vous (ou votre entreprise, y compris vos employés) pourriez causer à une autre personne dans le cadre de vos activités ou dans votre lieu d'affaires. Par exemple, un patient pourrait faire une chute sur un plancher mouillé et se blesser dans vos bureaux, puis tenir vous ou votre entreprise responsable (ils ont subi une lésion corporelle à cause de votre lieu d'affaires). Voilà pourquoi on fait souvent référence à l'assurance commerciale de responsabilité civile générale comme étant l'assurance « glissade-chute ».

L'assurance commerciale de responsabilité civile générale est également recommandée pour les optométristes qui possèdent ou exploitent une clinique. Cette protection est également recommandée pour les praticiens indépendants qui offrent des services en sous-traitance ou facturent des services sous le nom de leur entreprise. À titre d'exemple, si vous êtes un praticien indépendant et que vous causez accidentellement des dommages à la propriété dans laquelle vous travaillez, le propriétaire des lieux peut chercher à vous tenir responsable de lui rembourser le coût des réparations. Votre police d'assurance commerciale de responsabilité civile générale interviendrait dans ces circonstances.

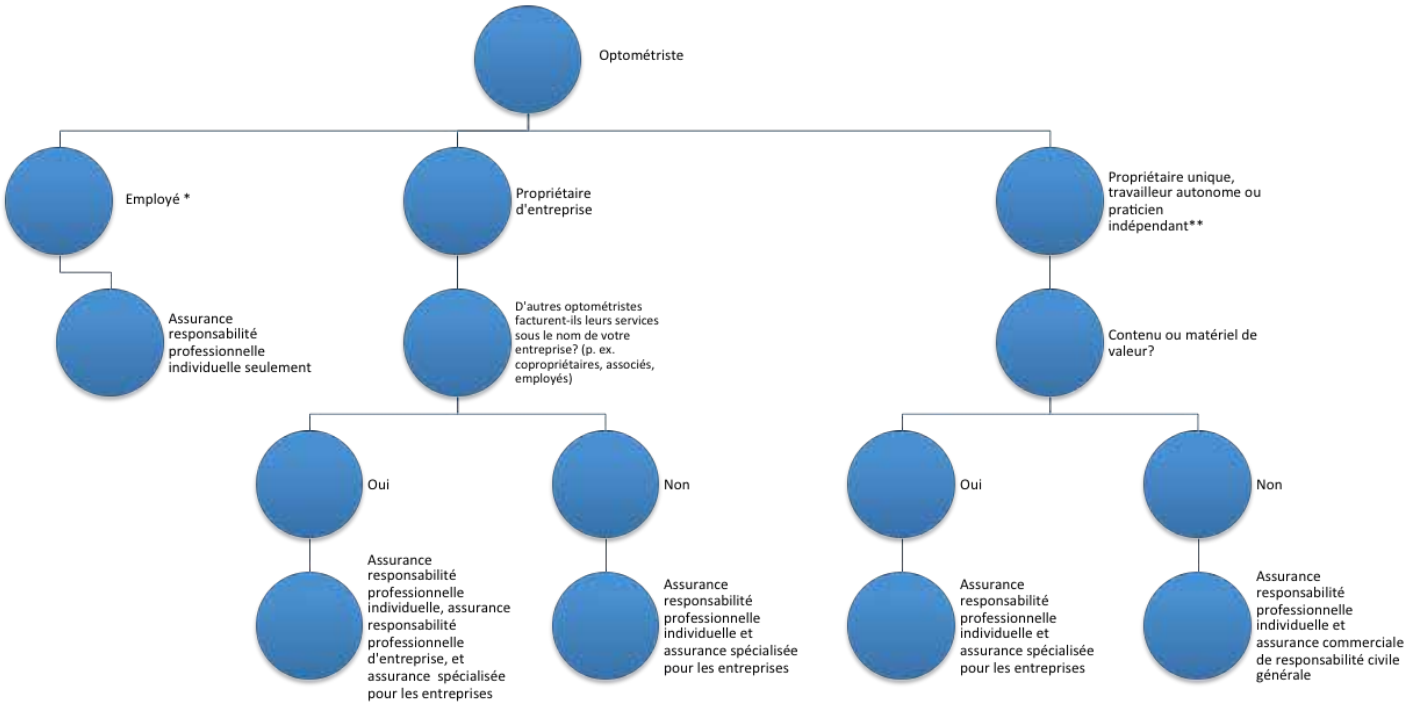
L'assurance de biens, la protection contre les actes criminels et l'assurance spécialisée pour les cliniques et entreprises :

L'assurance de biens et la protection contre les actes criminels protègent votre entreprise et son contenu contre les pertes associées aux dommages à la propriété (comme un incendie) et aux actes criminels. L'assurance de biens vous protège contre les dommages à la propriété, y compris l'équipement professionnel, et les pertes de revenu causées par une interruption des activités commerciales découlant d'un sinistre assuré. La protection contre les actes criminels vous protège contre la malhonnêteté, la fraude ou le vol d'argent, de valeurs mobilières ou de biens appartenant à l'entreprise ou au bureau.

Le régime de l'ACO offre une assurance spécialisée pour les cliniques et les entreprises, qui comprend l'assurance de biens, la protection contre les actes criminels et l'assurance commerciale de responsabilité civile générale. Les membres peuvent également souscrire l'assurance commerciale de responsabilité civile générale seulement.

Maintenant que vous connaissez bien la responsabilité professionnelle et les options d'assurance commerciale qui s'offrent à vous, vous pouvez utiliser l'organigramme qui suit pour vous aider à déterminer quelle assurance convient le mieux à vos conditions de pratique.

Veuillez noter que cet organigramme ne donne qu'un aperçu des scénarios de pratique courants et ne comprend pas toutes les structures commerciales et professionnelles possibles. Il constitue un premier cadre pour la prise de décisions, mais ne devrait pas être considéré au même titre que des conseils complets d'un courtier, ni utilisé comme tel. **Vous devriez toujours parler à un professionnel des assurances du groupe BMS pour déterminer l'assurance qui convient le mieux à vos conditions de pratique particulières.**



* *facture ses services sous le nom d'entreprise de son employeur.*

** *facture ses services sous votre nom d'entreprise; aucun autre optométriste ne facture ses services sous votre nom d'entreprise.*

Do you have more questions surrounding your professional liability and business insurance protection?

That's a good thing!

Professional liability protection and insurance are complex areas.

This is why we invite you to contact BMS Group at 1-844-517-1371 or cao.insurance@bmsgroup.com if you have any questions about the CAO insurance program, or to discuss your individual liability or business protection needs.

Find out more about the different insurance coverages here: www.cao.bmsgroup.com.

OPTOMETRYGIVINGSIGHT

Transforming lives through the gift of vision

Give the gift of vision this holiday season.

Photo Courtesy: Cielo Pictures

A donation of just $100 this holiday season could help fund a school screening in an underserved community

Haiti is the poorest country in the Americas. There are only 3 optometrists and 6 ophthalmologists in the public sector servicing a country of 10 million. This makes access to eye health services difficult for over 70% of Haiti's population. For children like Ralph (pictured), this can significantly reduce their ability to do well at school.

Please donate today at www.givingsight.org

or call 1-800-585-8265 ext 4

Donate

OPTOMETRYGIVINGSIGHT

Transformer des vies en donnant la vue

Faites le don de la vue pendant la période des fêtes.

Photo gracieuseté de Cielo Pictures

Un simple don de 100 $ pendant la période des fêtes pourrait aider à financer des examens de la vue dans les écoles d'une localité mal desservie.

Haïti est le pays le plus pauvre des Amériques. Dans ce pays de dix millions d'habitants, il n'y a que trois optométristes et six ophtalmologistes au sein du secteur public. Par conséquent, plus de 70 pour cent des Haïtiens peuvent difficilement avoir accès à des soins oculaires. Pour les enfants comme Ralph (sur la photo), cela peut avoir un effet néfaste sur la réussite scolaire.

Nous vous prions de faire un don aujourd'hui à www.givingsight.org

ou au 1-800-585-8265 poste 4

Faites un don

ARE YOU PAYING TOO MUCH FOR YOUR BUSINESS INSURANCE?

NEW! Optometrists now have access to group discounted business insurance coverage.

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CAO
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Find out how members have increased coverage while saving an average of 40% on their insurance costs.

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