

CJORCO

CANADIAN JOURNAL OF OPTOMETRY | REVUE CANADIENNE D'OPTOMÉTRIE



VOL 74 NO 1 | 2012

A Paradigm Shift in Primary Open Angle Glaucoma

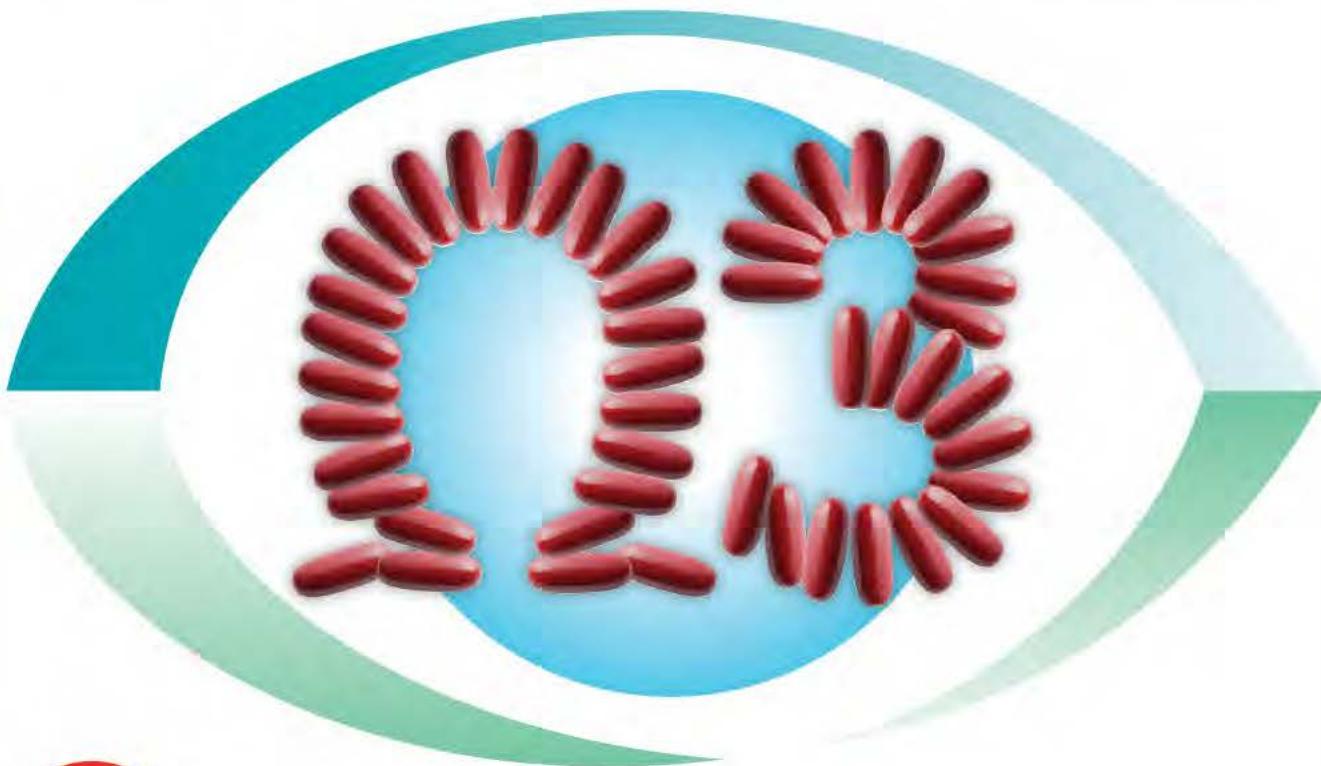
EYE SEE EYE LEARN

The Benefit of Comprehensive
Eye Examinations for Preschoolers

L'avantage des examens complets de
la vue chez les enfants d'âge préscolaire

AN ATYPICAL CASE OF HLA-B27-ASSOCIATED UVEITIS WITH HYPOPYON AND POSTERIOR SEGMENT INVOLVEMENT
CAS ATYPIQUE D'UVÉITE ASSOCIÉE À L'ANTIGÈNE HLA-B27 AVEC HYPOPYON ET ATTEINTE DU SEGMENT POSTÉRIEUR

New Advancements in Nutrition for Eye Health.



NEW!

For Adults With AMD

PreserVision®
Eye Vitamin and
Mineral Supplement
AREDS 2® formula

With **1000 mg** of Omega-3



For Adults Over 50

**OcuVite®
Adult 50+**
Eye Vitamin and Mineral
Supplement

Bausch & Lomb Canada Inc., Vaughan, Ontario, L4K 4B4

©Bausch & Lomb Incorporated

PreserVision and OcuVite are trademarks of Bausch & Lomb Incorporated or its affiliates.

® AREDS2 is a registered trademark of the United States Department of Health and Human Services.

*AREDS2® is an ongoing study.

†Euromonitor, Global Dollar Sales, Eye Health Supplements, 2010

BAUSCH + LOMB

The Global Leader in Ocular Vitamins.[†]



The Canadian Journal of Optometry is the official publication of the Canadian Association of Optometrists (CAO) / La Revue canadienne d'optométrie est la publication officielle de l'Association canadienne des optométristes (ACO) :
234 Argyle Avenue, Ottawa, ON, K2P 1B9. Phone 613 235-7924 /
888 263-4676, fax 613 235-2025, e-mail info@opto.ca,
website www.opto.ca. Publications Mail Registration No. 558206 /
Envoi de publication – Enregistrement no. 558206.
The Canadian Journal of Optometry / La Revue canadienne d'optométrie (USPS#0009-364) is published six times per year at CDN\$55, and CDN\$65 for subscriptions outside of Canada. Address changes should be sent to CAO, 234 Argyle Avenue, Ottawa, ON K2P 1B9.

The CJO*RCO is the official publication of the CAO. However, opinions and commentaries published in the CJO*RCO are not necessarily either the official opinion or policy of CAO unless specifically identified as such. Because legislation varies from province to province, CAO advises optometrists to consult with their provincial licensing authority before following any of the practice management advice offered in CJO*RCO.

The CJO*RCO welcomes new advertisers. In keeping with our goal of advancing awareness, education and professionalism of members of the CAO, any and all advertising may be submitted, prior to its publication, for review by the National Publications Committee of the CAO. CAO reserves the right to accept or reject any advertisement submitted for placement in the CJO*RCO.

La CJO*RCO est la publication officielle de l'ACO. Les avis et les commentaires publiés dans la CJO*RCO ne représentent toutefois pas nécessairement la position ou la politique officielle de l'ACO, à moins qu'il en soit précisé ainsi. Étant donné que les lois sont différentes d'une province à l'autre, l'ACO conseille aux optométristes de vérifier avec l'organisme provincial compétent qui les habilité avant de se conformer aux conseils de la CJO*RCO sur la gestion de leurs activités.

La CJO*RCO est prête à accueillir de nouveaux annonceurs. Dans l'esprit de l'objectif de la CJO*RCO visant à favoriser la sensibilisation, la formation et le professionnalisme des membres de l'ACO, on pourra soumettre tout matériel publicitaire avant publication pour examen par le Comité national des publications de l'ACO. L'ACO se réserve le droit d'accepter ou de refuser toute publicité dont on a demandé l'insertion dans la CJO*RCO.

Chair, National Publications Committee / Président,
Comité national des publications : Dr Paul Geneau

Academic Editors / Rédacteurs académiques :
University of Waterloo, Dr B. Ralph Chou
Université de Montréal, Dr Claude Glasson

Managing Editor / Rédactrice administrative ;
Advertising Coordinator / Coordonnatrice des publicités :
Graphic Design / Conception graphique
Leslie Laskarin

Editorial/Production Assistant / Adjoint de production et réviseur :
Tony Gibbs

Printing Consultant / Impression : Vurtur Communications

Translation / Traduction:
Tessier Translations / Les Traductions Tessier

Translation Editor / Réviseure des traductions :
Claudette Gagnon

President's Podium / Mot du président

- By/par *Lil Linton, OD* 4

Remembering Fred Kahn

- By *Scott Mundie, OD, LLDD, EA4AO* 6

2011 American Academy of Optometry (AAO) in Boston

- By *Etty Bitton, OD, MSc, EA4AO & Deborah Jones FCOptom, DipCLP, EA4AO* 11

Eye See Eye Learn—The Benefit of Comprehensive Eye Examinations for Preschoolers | L'avantage des examens complets de la vue chez les enfants d'âge préscolaire

- By/par *Deborah A. Jones FCOptom, DipCLP, EA4AO; Catherine A. Chiarelli OD, EA4AO;
Barbara E. Robinson OD, MPH, PhD, EA4AO; Karen E. MacDonald OD, EA4AO* 22

A Paradigm Shift in Primary Open Angle Glaucoma

- By/par *Mark Eltis, OD* 33

An atypical case of HLA-B27-associated uveitis with hypopyon and posterior segment involvement | Cas atypique d'uvéite associée à l'antigène HLA-B27 avec hypopyon et atteinte du segment postérieur

- By/par *Thomas Xie, OD & Etty Bitton, OD, MSc, EA4AO* 47

Uniform requirements for manuscripts: login to the member site at [opto.ca](#) or contact CAO.

Exigences uniformes pour les manuscrits: voir sur le site des membres à [opto.ca](#) ou contacter l'ACO.

99 reasons to visit morethanmedication.ca

1. The Ultimate Stretch for Flexibility routine.
2. Smart snacking tips for kids.
3. 12 organic foods to start buying now.
4. Tai Chi videos.
5. 5 keys to unlock difficult conversations.
6. Get a handle on clutter.



8. 21 days to power self esteem.
9. Stimulate your brain with interactive brain teasers.
10. Deep breathing exercises by Eli Bay.
11. How to outsmart your smartphone.
12. A free stress scale quizz.
13. Email etiquette made easy.
14. Crack the nutritional label code.
15. Are you a stress addict? Free quiz.
16. Easy ways to get more fibre in your diet.
17. Discover how to combat procrastination.
18. Pharmacist advice on herbal medicines.
19. Nutritious food that makes you feel full.
20. Learn why your doctor taps your back.
21. Dr. Ted Jablonski explains what doctors look for during check-ups.
22. How to travel with medication.



24. Safety checklist for seniors.
25. How to tell if you're properly hydrated.
26. How to find quality information on the internet.
27. Kick harmful habits
28. How to be in charge of your own health care.
29. Recognize the signs of stress.
30. How to stay energetic all day long.
31. Take a free healthy eating quiz.
32. How to practice active listening.
33. Test your snack IQ.
34. How tell the difference between stress, anxiety, and panic.
35. Hearing loss quick facts.

36. Learn the symptoms of a panic attack.
37. Smart snacking made easy.
38. Free audio meditation exercises.
39. Stop the "all-day sitting routine".
40. Tips for effective communication.
41. Watch Antoinette Giacobbe's video for achieving a positive way of thinking.
42. Find hope, strength, and inspiration through journaling.
43. Get perfect posture.
44. 10 essential steps to simplify your life.
45. How to de-clutter your home.



47. The ABCs of raising healthy, confident children.
48. How to visualize success.
49. Tips for co-parenting through a divorce or separation.
50. How to have "The Talk" with your teen.
51. How to stop saying "Yes" all the time.
52. What to do about emotional eating.
53. Strengthen your memory with free games.
54. Take our "What Inspires you?" poll.
55. Find support groups in your community.
56. How to de-clutter your mind of negative thoughts.
57. How to eat "mindfully".
58. Find out how much exercise kids need.
59. 11 activity ideas for families.



61. How to weight train with everyday items.
62. How to set a positive context for conversation.
63. Tips to boost your mood by getting physical.
64. What to do about excessive sweating.
65. Why you need to detoxify your relationships.
66. Get the low-down on artificial sweeteners.
67. Kick-start your metabolism.

68. Try the 3-Minute Work Desk Stretch.
69. Download a free health journal.
70. What you should know about bruises.
71. 5 easy ways to aid digestion.
72. Burn off calories with our Activity Calculator.
73. How your nutritional needs change as you age.
74. Learn everyday meditation techniques.
75. Find the best cavity-fighting foods.
76. Learn the benefits of Therapeutic Touch.
77. Stress-busting tactics for any workplace.
78. Get smidge, a free app that makes improving health fun and easy.
79. Seven easy ways to get more fruits and vegetables.
80. Just diagnosed? Here's what to do
81. How to beat the Restaurant Diet Sabotage Blues.
82. Check out a free video to improve your walking.
83. Learn about everyday portion control.



84. 7 steps to better sleep.
85. Are you getting enough vitamin D?
86. Where does it hurt? Use this interactive tool.
87. Take a free 5-Minute Memory Test.
88. How to be prepared for doctor's appointments.
89. Find healthier ways to satisfy your cravings.
90. Doctor's appointments from a doctor's POV. Free video.
91. How to work with your pharmacist for the good of your health.
92. How to cut down on salt without cutting down on taste.
93. What a pharmacist wants you to know.
94. How to energize yourself.
95. Turn your good (health) intentions into reality.
96. What one doctor wants you to know about hope.
97. 10 simple steps to eating healthy right now.
98. How to visualize inner strength.
99. Find lots more at morethanmedication.ca



Working together for a healthier world™

BY / PAR DR. LIL LINTON, OD

The national optometry incident report form (available in French and English – see links below) was launched in November 2011 and to date there have been 235 incidents reported.

This incident report form collects information to identify trends and issues to provide support for the value of comprehensive eye health examinations and optometric services. The form is intended to be completed by optometrists and/or their knowledgeable staff, to record incidents related to patients that present with:

- asymptomatic eye disease
- problems with contact lenses or eyeglasses purchased online or without a prescription
- a problem following a sight test
- a problem with cosmetic contact lenses

The keys to obtaining valuable information from the incident report initiative are widespread use from each province as well as the quality of information entered. To that end, the CAO and the provinces will be providing regular reminders to enter details of incidents you experience.

The national incident report form is an online form that should take 5 minutes or less to complete. It is simple to use, easily accessible online and will provide valuable information on eye health issues. The responses can provide a national aggregate picture or can provide information by province. The intent is to prepare an annual comprehensive report of the incidents collected and communicate those results to optometry and other stakeholders.

When the incident report initiative was first conceived, in response to the regulatory changes in British Columbia, CAO was cautioned that obtaining participation would be a challenge. Accordingly, a very conservative objective of 100 incident reports was established for the first year. The results in the first 5 months have far exceeded original expectations. However, CAO cannot emphasize enough the importance of continuing to report incidents that present themselves in your practice.

Who should enter the incidents is dependent on the structure of your practice. The CAO has and will continue to communicate with optometric assistants about the existence of the incident report and the need to report incidents. Depending on the practice, delegating a knowledgeable person within your office to take responsibility for reporting incidents can contribute positively to the success of this initiative.

Data obtained through incident reporting can be used in several ways, all of which can provide significant value to positioning optometry as the front line eye health profession. Information from incident reports can provide valuable evidence to influence the development of public policy. Aggregate data will also provide baseline measures that can help identify the impact of regulatory changes. Incident report information can be shared with regulators, provincial health departments and Health Canada to facilitate their decision making.

Incident report information can help identify potential examples of stories for media relations. If a patient's story is identified and desired to be used, the incident report form provides enough information that the optometrist can contact the patient to obtain permission. Obtaining patient permission on the incident report form was not deemed essential as only a small fraction of incidents would be needed or desired for marketing and communication purposes.

Incident reports represent a proactive initiative that provides information to support advocacy initiatives and to influence policy development. Recent history has shown how important it is to have access to this type of information when the need arises. A decade ago, Health Canada wanted evidence of the risk of harm from cosmetic contact lenses, but

there was no Canadian data. When the Government of British Columbia revised its regulations in 2010, local incidents relating to asymptomatic patients and internet purchases could have been invaluable. A substantive number of incidents for internet sales and cosmetic contact lenses could have supplemented efforts pertaining to the regulation of cosmetic contact lenses and the recent CBC Marketplace story.

The opportunities to use this information to support the value of optometric services will be ongoing. It is important that all CAO members place a priority on reporting incidents and contribute to the information being gathered.

An incident reporting system will certainly provide more information about the complications being attended to by Canadian optometrists than what currently exists. To be effective there has to be considerable effort initially to build awareness, increase use, and synthesize information. This is a required commitment for resources and support for the next 3-10 years.

Knowledge about eye health issues enables the optometric profession to proactively advocate for the eye health of Canadians and participate in the development of public policy. Your cooperation in the collection of this information is essential to influence government policy that regular eye health exams are important for early diagnosis and beneficial to reducing health care costs.

Le lancement du formulaire national de déclaration des incidents (en anglais et en français – voir les liens ci-après) a eu lieu en novembre 2011 et, jusqu'à maintenant, 235 incidents ont été déclarés. Ce formulaire d'incident sert à recueillir des renseignements destinés à

English - <http://www.surveymonkey.com/s/ODincidentreport>
 French - <http://www.surveymonkey.com/s/odrapportincident>

dégager les tendances et les enjeux ayant pour but de souligner l'importance de la valeur des examens complets de la santé oculovisuelle et des services optométriques. Sur ce formulaire, les optométristes ou leurs employés compétents y indiqueront les incidents liés à des patients qui :

- présentent une maladie oculaire asymptomatique
- éprouvent des problèmes à la suite de l'achat de lentilles cornéennes ou de verres en ligne OU sans ordonnance
- éprouvent un problème à la suite d'un test de la vue
- éprouvent un problème lié à des lentilles cornéennes à but esthétique

Pour obtenir des données utiles par le biais de cette initiative de déclaration, il est essentiel d'en généraliser l'utilisation dans chaque province et de recueillir des renseignements de qualité. À cette fin, l'ACO et les provinces diffuseront régulièrement des messages pour vous rappeler de consigner les incidents dont vous êtes témoin.

Le formulaire national de déclaration ne devrait pas prendre plus de cinq minutes à remplir en ligne. Ce formulaire simple d'utilisation et facilement accessible en ligne fournira de précieux renseignements sur les questions de santé oculovisuelle. Les réponses pourront fournir une image d'ensemble à l'échelle du pays ou encore des renseignements par province. Le but est de colliger un rapport annuel global des incidents consignés et d'en diffuser les résultats à l'optométrie et à d'autres intervenants.

Lors de la conception de cette initiative, en réponse à l'évolution de la réglementation en Colombie-Britannique, l'ACO avait été mise en garde qu'il serait difficile d'obtenir une participation. Par conséquent, on avait établi de façon très prudente un objectif de 100 rapports d'incidents pour la première année. Les résultats obtenus au cours des cinq premiers mois ont largement dépassé les espérances initiales. Cependant, l'ACO ne

peut souligner suffisamment l'importance de poursuivre la déclaration des incidents dont vous êtes témoin dans votre cabinet.

Les personnes responsables de la déclaration des incidents devraient être choisies selon la structure organisationnelle de votre cabinet. L'ACO continuera de faire connaître aux assistants optométriques l'existence du rapport d'incidents et le besoin de les déclarer. Selon votre cabinet, la délégation de la responsabilité de la déclaration des incidents à une personne qualifiée de votre bureau peut contribuer positivement au succès de cette initiative.

Les données obtenues par ce système de déclaration peuvent servir de plusieurs façons, mais toutes apporteront une valeur importante en aidant l'optométrie à se positionner en première ligne des professions de la santé. Les renseignements tirés des rapports d'incidents fourniront des données précieuses qui influeront l'élaboration de politiques publiques. Les données agrégées représenteront aussi des mesures de base qui pourront aider à connaître les répercussions des modifications réglementaires. Les renseignements du système de déclaration pourront être échangés avec les organismes de réglementation, les ministères provinciaux de la santé et Santé Canada pour faciliter la prise de décisions.

Les données de rapports d'incident pourront aussi aider à faire connaître les exemples potentiels susceptibles d'être diffusés par les médias. L'optométriste qui voudra utiliser l'histoire d'un patient pourra, grâce aux données suffisantes du rapport d'incident, communiquer avec le patient pour obtenir son autorisation. On n'a pas jugé essentiel d'obtenir la permission des patients lors de la déclaration d'un rapport d'incident puisque seul un faible pourcentage des incidents seront utilisés à des fins de marketing ou de communication.

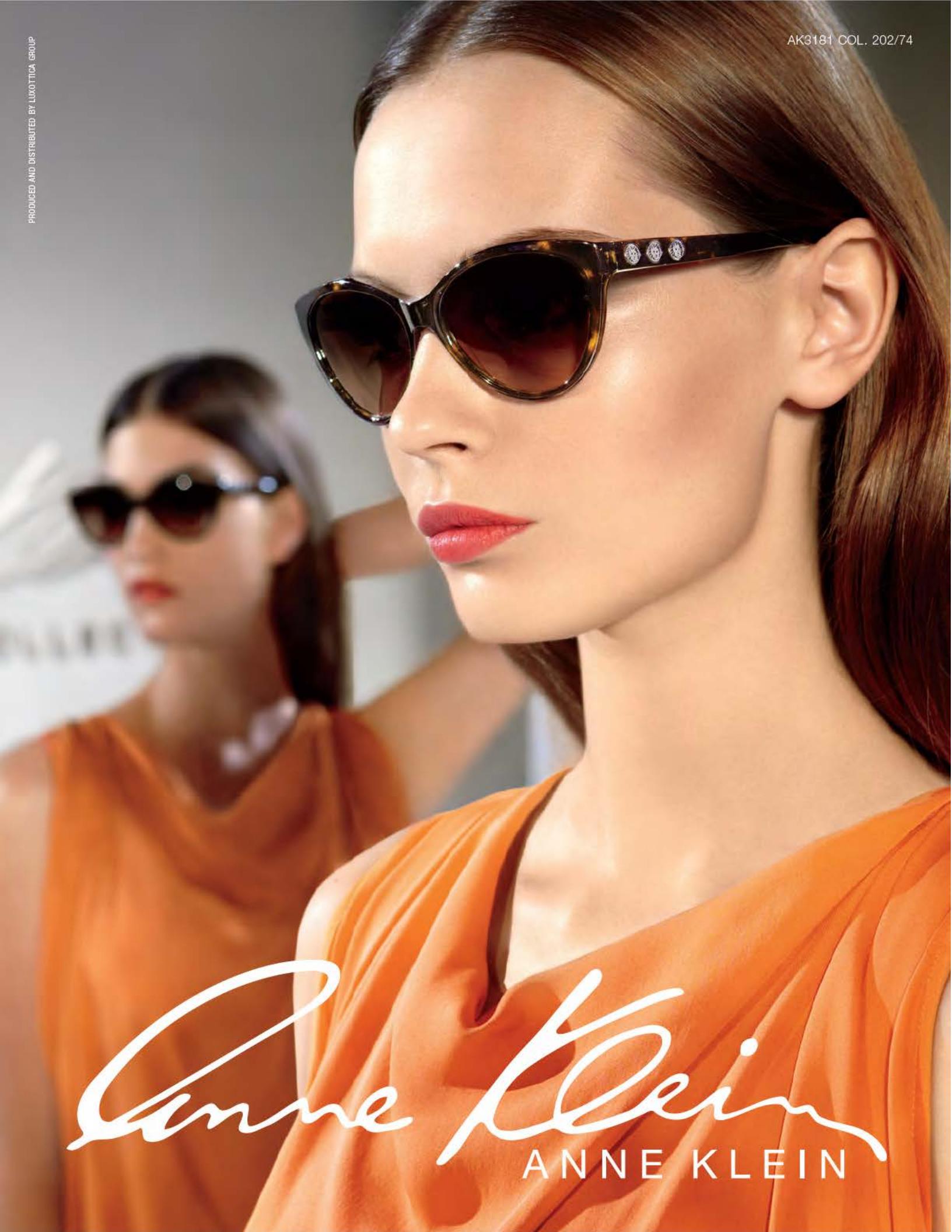
Les rapports d'incident représentent une initiative proactive qui servira à appuyer des initiatives de défense et à appuyer l'élaboration de politiques. Les faits récents nous ont révélé à quel point il est important d'avoir accès à ce type d'information lorsque le besoin se présente. Il y a une

dizaine d'années, Santé Canada désirait étoffer par des données les risques que comportaient les lentilles cornéennes à but esthétique, mais il n'existant aucune donnée à ce sujet au Canada. Lorsque le gouvernement de la Colombie-Britannique a revu son règlement en 2010, il lui aurait été très précieux de pouvoir disposer de données sur les incidents liés à des patients asymptomatiques et à des achats sur Internet. Un nombre élevé d'incidents provenant de la vente d'articles sur Internet et de lentilles cornéennes à but esthétique auraient pu compléter les efforts face à la réglementation des lentilles cornéennes à but esthétique de même qu'au récent reportage de Marketplace sur le réseau anglais de CBC.

Il ne cessera d'y avoir des occasions d'utiliser cette information pour appuyer la valeur des services optométriques. Il est important que tous les membres de l'ACO accordent la priorité à la déclaration des incidents et qu'ils contribuent aux renseignements qui seront recueillis.

Un système de déclaration des incidents fournira incontestablement plus de renseignements sur les complications que rencontrent les optométristes canadiens que ce qui existe actuellement. Pour qu'un tel système soit efficace, il faudra déployer initialement des efforts considérables pour bâtir la sensibilisation, pour accroître l'utilisation et pour synthétiser les données. Il faudra s'engager à disposer des ressources et de l'appui nécessaires pour les trois à dix prochaines années.

La connaissance des enjeux liés à la santé oculovisuelle permet à la profession optométrique de sensibiliser proactivelement les Canadiens à la santé oculo-visuelle et de participer à l'élaboration des politiques publiques. Votre collaboration à la collecte de ces renseignements est essentielle si nous voulons influencer des politiques gouvernementales qui appuient l'importance des examens réguliers de la santé oculovisuelle pour un diagnostic précoce et une diminution des coûts des soins de santé.



The image features two women in the background, both wearing dark sunglasses and bright orange sleeveless tops. The woman on the right is in sharp focus, looking slightly to her left with a neutral expression. Her hair is long and dark brown. The woman on the left is slightly out of focus, also wearing sunglasses and an orange top. The overall lighting is soft and warm.

Anne Klein
ANNE KLEIN

Fred Kahn

BY SCOTT MUNDLE, OD, LLD, FAAO

The year 2012 began with a loss in the ophthalmic field. It was not an economic loss. It was a deeper one. Fred Kahn passed away on January 10, 2012. If you graduated in the last decade or so you may not recognize that name. But for many of us who are getting longer in the tooth ourselves, Fred was a major figure in the early parts of our careers.

After graduating as a Civil Engineer in 1946, Fred joined the family business, Kahn Optical, and became its General Manager and ultimately Chief Operating Officer. In its day Kahn Optical was the epitome of the independent ophthalmic laboratory and frame distributor. It battled giants such as Imperial Optical, American Optical and Bausch and Lomb for its scrap of turf on the Canadian optical scene. But Fred was more than a lab owner. He travelled throughout Ontario and Western Canada selling his product and his philosophies on professional practice. He became personal friends with a great many of his clients. I considered him a mentor in many ways.

When Fred's company succumbed to the growing trend of bigger business he decided to follow one of his passions — creating thoughtful, high-end optometric offices across the country. Having visited many hundreds of offices and talking to practitioners for so many years, he understood the needs of grass roots optometry better than almost anyone. He and his interior designer associate, Larry Funston, who actually engineered and designed Fred's concepts and oversaw the construction, worked out of Zeiss Canada's Toronto quarters. Together they changed the face of optometric offices for almost two decades.

Fred's gently persuasive, reasoned manner and his love of language made him a natural communicator. He not only came up with innovative office designs that presented optometry in a powerful and professional manner, he wrote about it. He published numerous articles in professional journals

in both Canada and the United States. Ultimately he authored a book, "Maximizing the Potential of Your Ophthalmic Office", that was hailed as a bible in optometric office design. But even more important from my standpoint, Fred Kahn understood our profession, recognized its evolution and worked with many of its leaders to move it forward from its mercantile roots to the respected primary care health profession it has become. When others clung to the tried and true, wary of displacing the long held emphasis on being dispensers with a diagnostic bonus attraction, Fred got it. While remaining proud of its dispensing background, optometry had to bring its ever increasing diagnostic and treatment capabilities to the fore. He understood and championed the unique value that optometry could offer. "Unified service" was a philosophy he believed in, promoted and it was the hallmark of his designs. Gone were the little refracting lanes in the back of optical dispensaries. Across the country, optometrists who practiced in a Kahn-inspired office were seen as leading edge by their patients and their peers.

Fred's office designs evolved with the profession. In fact, it could be argued that Fred didn't follow the evolution of the profession in Canada, he was one of its leaders. When optometry was emerging as a fully recognized health care profession in the mid-sixties, Fred saw that it had to downplay its mercantile beginnings. Fred began advocating "dispensing rooms" with frames in drawers, which he eventually designed and manufactured, so that the dispenser (often the optometrist) completely controlled the frame selection process. It tied in with the fee for service concept that distinguished optometric offices from optical shops. Once the profession gained confidence and firmly established its professional status he began to advocate a whole new approach to the dispensary. It could still be controlled but fashion display became a key element and made getting glasses a pleasurable experience for the involved patient. Delegation of a number of tasks in the optometric office,



Fred Kahn passed away on January 10, 2012

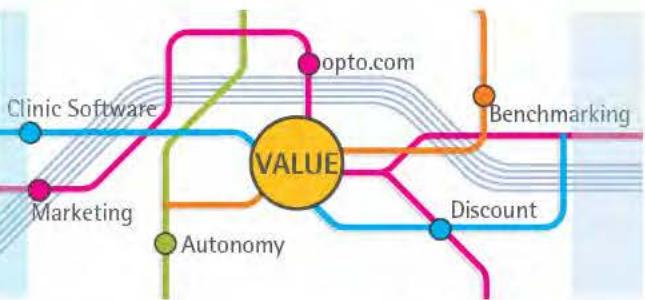
including frame selection, and eventually diagnostic technical tasks, were assumed in the new designs. Over the years, Fred fortified not only the unified service concept of optometry but helped make the health related/medical aspects of optometric care more efficient and effective. Diagnostic pods radiating from a central core were seen more and more across the country. These designs reflected his firm belief that optometrists had strong choices in their mode of practice. A non-dispensing model was certainly valid but neither superior nor inferior to a unified service mode with an exciting high fashion eyewear boutique as an integral part of a state of the art diagnostic centre. In this context many would say he was a visionary.

Our profession has continued to evolve since Fred finally slowed down and rested after a lifetime of working with passion and love for our field. There are many beautiful new offices and our confidence is at an all time high. But I wonder if we would have been where we are today had it not been for the likes of Fred Kahn. His last book was a thick, comprehensive, delightful chronicle of his family and his life. He finished it at age 88. He gave me a copy when we visited him in his seniors' apartment in Toronto last year. I will cherish it and I just might try to do the same thing myself someday. He inspired me personally in many ways. He challenged, enriched and loved our profession. I will miss him.



Partner Profile

Profile de partenaire



Optometric Services Inc. (OSI), generating value for Optometrists

Services Optométriques Inc. (SOI), générer de la valeur aux optométristes

OSI was founded in 1983 and is owned exclusively by independent Optometrists who purchase shares in the company. At present more than 1,500 Optometrists owning and operating more than 750 clinics make up the OSI network. OSI offers a multitude of essential services that allow independent Optometrists to be more competitive in their respective markets.

These services include:

- Centralized billing
- Advantageous supplier discounts
- Marketing advertising and web services
- Clinic management software
- "In-Field" Regional Account Managers
- Equipment financing
- Exclusive private labels
- Training seminars with CE credits
- And much more...

Since its inception, OSI has consistently welcomed new Optometrists to the network. Those Optometrists benefit rapidly from considerable discounts on their purchasing and see tangible results on their operations when using one or several of the aforementioned services provided by the network.

With all its benefits, and without any membership fees, OSI is indisputably the most performing tool when it comes to generate value for the Optometrists. Help strengthen the Canadian Independent Optometry and join Canada's Largest Network of Optometrists right away. Please contact us at (800) 363-4096 or info@opto.com.

Fondée en 1983, SOI compte plus de 750 cliniques membres, dans lesquelles plus de 1 500 optométristes pratiquent. Appartenant exclusivement aux optométristes membres, le regroupement offre une multitude de services essentiels qui permettent aux optométristes indépendants d'être plus concurrentiels sur leurs marchés respectifs.

En effet, les membres bénéficient, entre autres :

- facturation centrale
- taux d'escampes avantageux
- services marketing, publicité et web
- logiciel de gestion de clinique
- financement d'équipement
- marques privées exclusives
- séminaires de formation
- et bien plus encore...

Depuis sa fondation, SOI recrute régulièrement de nouvelles cliniques qui veulent prendre leur juste place dans un environnement de plus en plus compétitif. Ces cliniques réalisent en peu de temps des économies considérables sur leurs achats et voient des résultats tangibles sur leurs opérations en utilisant un ou plusieurs services offerts par le regroupement.

Avec tous ses atouts, et sans frais d'adhésion, SOI est sans contredit l'outil le plus performant lorsqu'il s'agit de générer de la valeur pour les optométristes. Joignez la force d'un réseau d'optométristes propriétaires dès maintenant en nous contactant au 514-762-2020, 1-800-363-4096 ou info@opto.com.



Canada's Largest Network of Optometrists
Le plus grand réseau d'optométristes au Canada
www.opto.com

Follow us on /Suivez-nous sur :





OPEN YOUR EYES
SILVER
PARTNER 2012

EYE HEALTH COUNCIL OF CANADA | LE CONSEIL CANADIEN DE LA SANTÉ DE L'OEIL



Centennial adds LOGIC® contact lenses

Centennial Optical is pleased to announce that it has acquired the Logic® line of contact lenses from Professional Contact Lens Inc., effective January 1, 2012. Centennial President, Steve de Pinto, stated: "We are also pleased to have Gerry Griffin, the former President of PCL, renew his association with Centennial Optical as sales consultant for contact lenses."

The Logic contact lens line includes Logic Enhanced View and Logic 1-Day single vision lenses, plus Logic CT Colours and Logic 1-Day Colours single vision lenses in a wide range of cosmetic colour options.

Centennial Optical is committed to maintaining, as closely as possible, the programs, pricing and policies that PCL had been offering. For Logic customers, the transition should prove to be a virtually seamless one, with the added benefits of the support of larger sales and customer service teams and the availability of a wider product offering.

Logic contact lenses, as with all of the contact lens products distributed by Centennial Optical, are available exclusively to Eye Care Professionals. Commenting on the company's ECP-only distribution policy, Mike Jones, Director of Sales for Lenses and Contact Lenses said,

"It's only fair that the ECP, who performs the examinations and patient trials, creating the market for this product, be provided the tools to retain the business they've created."

Other contact lens brands distributed exclusively to Eye Care Professionals by Centennial Optical include Sauflon Clariti and Bioclear, Extreme H2O and Definition AC. For more information, please contact your Centennial Optical lens representative.

For more information, contact:
Rick Leroux
Director, Marketing & Communications
Lens Division, Centennial Optical Ltd.

Phone: 416-739-5458 / 1-800-561-0681
Fax: 416-739-6504
Email: rleroux@centennialoptical.com





One in seven Canadians will develop a serious eye disease in their lifetime.

May is Vision Health Month, and CNIB is challenging Canadians to take action to maintain their vision health so that we can eliminate avoidable sight loss in Canada.

Join the cause. Spread the message.
Learn more at eyesareforlife.ca today.



CNIB National Vision Health Month Partner



The Canadian
Association of
Optometrists

OPTOMETRYGIVINGSIGHT

Transforming lives through the gift of vision



**Optometry raising
funds to...**

Train



local eye care
professionals

Photo courtesy of ICSE

Establish



vision centres for
sustainability

Photo courtesy of ICSE

Deliver



eye care and low
cost glasses

Optometry Giving Sight funds sustainable eye care services for people who are blind or vision impaired due to uncorrected refractive error.

Please become a donor today:

www.givingsight.org or call **1-800-585-8265 ext 4**



Follow us on Facebook
facebook.com/optometrygivingsight



Follow us on Twitter
[@givingsightorg](https://twitter.com/givingsightorg)

2011 American Academy of Optometry (AAO) in Boston



Alex Hui (JW) received the Ezell Travel Fellowship from the American Optometric Foundation (AOF) at the AAO meeting

**BY ETTY BITTON, OD, MSc, FAAO
& DEBORAH JONES FCOptom, DipCLP, FAAO**

Another year, another meeting! Why do they seem to come around more quickly from year to year? The two Canadian schools once again were well represented at this key optometry meeting, held last October in Boston. The meeting attracted a total of 5693 attendees, the second largest of all time, and a record-breaking, 1094 students. Optometrists, vision scientists, professors, residents and students congregated for the 4-day meeting packed full of information. Conferences, scientific presentations (paper and poster presentations), workshops, special symposiums, and the latest technologies and products in the exhibit hall were available for the meeting participants.

Faculty, graduate and optometry students from both Canadian schools also participated in the meeting with contributions to lectures and scientific presentations, as shown in Table 1 and 2.

Table 1: École d'optométrie, Université de Montréal participation at the 2011 AAO Meeting

FACULTY	
AUTHOR	TITLE
Bitton E	A CLOSER LOOK AT THE OCULAR TEAR FILM (conference 1 hr)
Bitton E, Landreville MP, Forget MC	INFLUENCE OF EYE POSITION ON THE SCHIRMER TEAR TEST (paper)
Brûlé J, Tousignant B	EVIDENCE-BASED TOOL TO TRAIN EDUCATORS IN MULTIPLE-CHOICE QUESTION WRITING IN RESOURCE-LIMITED SETTINGS: A PILOT STUDY (poster)
Brûlé J, Duguay C, Larouche J	AWARENESS OF MODALITIES OF EYE EXAM IN QUEBEC, CANADA (poster)
Brûlé J, Larouche J, Duguay C	IDENTIFYING THE NEEDS FOR OCULAR HEALTH EDUCATION IN QUEBEC, CANADA (poster)
Faubert J	PERCEPTUAL-COGNITIVE SPEED TRAINING WITH PROFESSIONAL ATHLETES (poster)
Frenette B, Michaud L	IMPROVEMENT OF CONTACT LENS COMFORT WITH THE USE OF A SODIUM HYALURONATE (SH) DROP ON THE LENS BEFORE INSERTION (poster)
Kergoat H, Lovasik JV, Kergoat MJ, Racine N, Parent M	RETINAL METABOLIC REGULATION WITH AGE (poster)
Michaud L	MODERN RGP LENS DESIGNS FOR DAY-TO-DAY PRACTICE: A CLINICAL GRAND ROUND (conference 1 hr)
Michaud L, Bitton E, et al. Michaud L	GRAND ROUNDS I: First Report of Ocular Dryness as Related to Maple Syrup Urine Disease (MSUD) (conference 2 hr) TOPS: A NEW CLINICAL FINDING RELATED TO FABRY'S DISEASE (poster)
Overbury O	A NEW PhD PROGRAM IN VISION SCIENCE AT THE UNIVERSITY OF MONTREAL (poster)
Renaud J, Overbury O, Durand MJ	WHAT EXPLAINS GOOD SUBJECTIVE QUALITY OF LIFE OF OLDER ADULTS WITH VISUAL IMPAIRMENT? (poster)

OPTOMETRY STUDENTS	
AUTHOR	TITLE
Bachir V, Rubino L, Warde R, Lovasik JV, Kergoat H	THE EFFECTS OF PANRETINAL PHOTOCOAGULATION ON NEUROVASCULAR COUPLING IN THE HUMAN DIABETIC EYE (poster)
Bédard E, Michaud L, Brazeau D, Pop M	COMPARATIVE STUDY OF 3 MODES OF TREATMENT FOR KERATOCONUS (poster)
Deschambault E, Abboud C, Brûlé J	SMOKING CESSION COUNSELLING: PRACTICES OF QUÉBEC OPTOMETRISTS (poster)
Hong Y, Bitton E	ACQUIRED FACIAL NERVE PALSY FOLLOWED BY HERPES ZOSTER INFECTION: MANAGEMENT OF OCULAR SYMPTOMOLOGY (poster)
Samaha D, Lafleur G, Michaud L	HYDROGEN PEROXIDE AS A BETTER ALTERNATIVE CARE REGIMEN FOR RGP AND RIGID CONTACT LENSES (paper)
Xie T, Bitton E	AN ATYPICAL CASE OF HLA-B27- ASSOCIATED UVEITIS WITH HYPOPYON AND POSTERIOR SEGMENT INVOLVEMENT (poster)

Continued

Several awards were handed out at the AAO meeting including the Carl Zeiss Vision Fellowship to optometry students Claudine Courey (UM) and Mariam Nahal (UW). William C. Ezell Fellowships for graduate work was awarded to Jean-Marie Hanssens (UM) and Alex Hui (UW). Several travel fellowships were also available to help optometry and graduate students attend the meeting. These included the Essilor Student Travel Fellowship awarded to Samar Farhat (UM) and Tyler Anderson (UW); The Vision Care Institute™, LLC Travel Fellowship was awarded to Vanessa Bachir, Estefania Chriqui, Reza Abbas Farishta (UM) and Alan Ng, Salsabeel Jadi and Subam Basuthka (UW). The Brazelton Low Vision Student Travel Fellowship was awarded to Lea Gagnon (UM), the Irvin Borish Student Travel Fellowship was awarded to Judith Renaud (UM) and the Section on Cornea, Contact Lenses & Refractive Technologies Resident Travel Fellowship was awarded to Richard Warde (UM). Dr. Thom Freddo (UW professor) was appointed to the Board of Regents of Beta Sigma Kappa.

The annual meeting is also the time where new Fellows of the AAO (FAAO) get inducted. This process is a peer-recognition whereby an optometrist or vision scientist demonstrates their knowledge, contributions and leadership to a committee. The annual meeting culminates this effort with a final interview process and those that successfully complete the process are awarded a FAAO at the meeting. This year 194 new Fellows were inducted with six Canadians including Dr. Walter Wittich (UM and presently at the MAB-MacKay Rehabilitation Center), Dr. Doerte Luensmann (UW-CCLR), Dr Marc Schultze (UW-CCLR), Dr Sara Maciver (UW), Dr Mark Eltis (Toronto, Ontario) and Dr David Schwirtz (Surrey, British Columbia). Congratulations to all the new Fellows !

Next year the meeting will be held in Phoenix, Arizona on October 24-27, 2012. Looking forward to seeing you there !

Table 1: École d'optométrie, Université de Montréal (*continued from p.11*)

GRADUATE STUDENTS	
AUTHOR	TITLE
Abbas R	EFFECTS ON THE VISUAL CORTEX OF ELECTRICAL AND VISUAL STIMULATION OF THALAMIC NUCLEI REVEALED BY VSD IMAGING (poster) RESPONSES OF THE STRIATE AND EXTRA STRIATE CORTEX TO PULVINAR AND LGN STIMULATION IN THREE SHREWS (poster)
Chriqui E, Kergoat MJ, Champoux N, Leclerc BS, Kergoat H	ASSESSMENT OF VISUAL ACUITY OF COGNITIVELY IMPAIRED OLDER INDIVIDUALS RESIDING IN LONG-TERM CARE FACILITY (poster)
Gagnon L, Aumond S, Huppe A, Ptito, M	HOW TO IMPROVE TACTILE MAPS: LESSONS FROM BLINDNESS! (poster)
Graudet G, Miconi C, Hanssens JM, Faubert J	ROLE OF BINOCULAR DISPARITIES IN THE VISUAL CONTROL OF QUIET STANCE (poster)
Hanssens JM, Graudet G, Allard R, Faubert J	MODULATING SOMATO-SENSORY INPUT INCREASES VISUAL DEPENDENCY FOR POSTURAL CONTROL (poster)



Student attendees from Montreal at the AAO meeting



Master's student Dr. Estefania Chriqui and supervisor Dr. Hélène Kergoat (UM) presenting at the scientific section of the meeting.



Dr. Lyndon Jones flanked by new AAO Fellows, Dr. Doerte Luensmann and Dr. Marc Schultze (UW)

Table 2: School of Optometry, University of Waterloo participation at the 2011 AAO Meeting

AUTHOR	TITLE	AUTHOR	TITLE
Alex Hui* (first author) Heather Sheardown, Lyndon Jones	MOLECULAR IMPRINTED SILICONE HYDROGEL MATERIALS FOR CIPROFLOXACIN DRUG DELIVERY (paper)	Elizabeth Irving (first author) Carolyn Machan, Andrea Mittles-taadt, Patricia Hryncak	DIFFERENCE BETWEEN PRESENTING AND BEST CORRECTED VISUAL ACUITY AS A FUNCTION OF AGE (poster)
Kathy Dumbleton (first author) Craig Woods, Lyndon Jones, Desmond Fonn	A SURVEY TO INVESTIGATE LAPSED CONTACT LENS WEARERS IN CANADA (paper)	Elizabeth Irving (first author) Carolyn Machan, Patricia Hryncak	ASSOCIATIONS BETWEEN REFRACTIVE ERROR, NEAR PHORIA AND AGE (poster)
Kathy Dumbleton (first author) Craig Woods, Lyndon Jones, Desmond Fonn	COMPARING CONTACT LENS COMPLIANCE AND COMPLICATIONS IN A UNIVERSITY CLINIC WITH PRIVATE OPTOMETRY OFFICES (paper)	B. Ralph Chou (first author) Jeffery K. Hovis	OCULAR HAZARD OF A THERMAL LANCE (poster)
Mike Woods (first author) Kathy Dumbleton, Craig Woods, Lyndon Jones, Desmond Fonn	DO CONTACT LENS WEARERS REMEMBER WHAT PRODUCTS THEY ARE USING? (poster)	Craig Woods (first author) Nancy Keir, Doerte Luensmann, Desmond Fonn	VISUAL PERFORMANCE OF MULTIFOCAL CONTACT LENSES (PAPER)
Doerte Luensmann (first author) Nancy Keir, Megan Despres, Doris Richter, Craig Woods, Desmond Fonn	IN VIVO WETTABILITY CHANGES OVER 3 DAYS USING DAILY DISPOSABLE CONTACT LENSES (poster)	Sruthi Srinivasan (first author) Kara Menzies, Luigina Sorbara, Lyndon W Jones	IMAGING MEIBOMIAN GLAND STRUCTURES USING THE OCULUS KERATOGRAPH (poster)
Alan Ng (first author) Miriam Heynen, Lyndon Jones	THE IMPACT OF LACTOFERRIN AND LIPIDS ON KINETIC LYSOZYME DEPOSITION ON CONTACT LENSES (paper)	Chalmers, R. L.; Keay, L.; Kern, J.; Jansen, M.; Lam, D.; Kinoshita, B.; Wagner, H.; Sorbara, L.; Bullimore, M.; Shovlin, J.; Szczotka-Flynn, L.	CHARACTERIZING CORNEAL INFILTRATES FROM A 2010 CASE CONTROL STUDY OF SOFT CONTACT LENS WEARERS (paper)
Sarah Guthrie (first author) Jill Woods, Nancy Keir, Vivian Choh, Sally Dellehay, Mark Tyson, Richard Griffin, Lyndon Jones, Elizabeth Irving	CONTROLLING LENS INDUCED MYOPIA IN CHICKENS WITH PERIPHERAL LENS DESIGN (paper)	Sarah MacIver (first author) Bass S., Sherman J. (SUNY State College of Optometry)	VASO-OBLITERATION IN A CASE OF IDIO-PATHIC RETINAL VASCULITIS, ANEURYSMS AND NEURORETINITIS (poster)
Subam Basuthkar (first author) Trefford Simpson	DOES DEFOCUS CONTRIBUTE TO OCULAR DISCOMFORT? (paper)	Sarah MacIver (first author) Madonna R., Slotnik S., Sherman J. (SUNY State College of Optometry)	SD-OCT IMAGES THE RECOVERY OF PHOTORECEPTOR FUNCTION IN CASE OF COMMOTIO RETINAE OF THE MACULA (poster)
Marc Schulze (first author) Trefford Simpson, Ping Situ, Kara Menzies, Hendrik Walther, Lyndon Jones	EFFECTS OF MAGNIFICATION ON TEAR MENISCUS PARAMETERS USING OPTICAL COHERENCE TOMOGRAPHY (OCT) IMAGES (poster)	Jill Woods (first author) Ping Situ, Nancy Keir, Craig Woods, Desmond Fonn	HOW DOES READING ADDITION INFLUENCE THE PERFORMANCE AND ACCEPTANCE OF SOFT MULTIFOCAL LENSES? (poster)
Patricia Hryncak (first author) Carolyn Machan, Elizabeth Irving	NEAR PHORIA AND STRABISMUS AS A FUNCTION OF AGE IN A CLINIC POPULATION (poster)	Jill Woods (first author) Ping Situ, Craig Woods, Desmond Fonn	MEDIUM- ADDITION, CENTRE- NEAR,SILICONE HYDROGEL MULTIFOCAL LENS COMPARED TO MONOVISION: PERFORMANCE, ADAPTATION AND PREFERENCE (poster)
Norris Lam (first author) Alison Leong, Susan Leat	LOW VISION SERVICE PROVISION BY OPTOMETRISTS – A CANADIAN SURVEY (Poster)	Lyndon Jones (first author) Mark Willcox, Loretta Szczotka-Flynn, Joe Shovlin, Jason Nichols	BIOFILMS AND BIODEPOSITS: CLINICAL IMPLICATIONS (paper)
Balsam Alabdulkader (first author) Susan Leat	THE EFFECTIVENESS OF READING ADDI-TIONS FOR CHILDREN AND YOUNG ADULTS WITH LOW VISION (paper)	Amir Moezzi (first author) Ping Situ, Doerte Luensmann, Desmond Fonn, Craig Woods, John McNally, Lyndon Jones	DOES COMFORT WITH AGING SILICONE HYDROGEL LENSES RELATE TO CHANGES IN LENS FIT AND CONJUNCTIVAL STAINING? (poster)
Susan Leat	PEDIATRIC LOW VISION MANAGEMENT – MAKING A START – CE lecture chair and co-founder of this group (inaugural meeting this year)	Nancy Keir (first author) D Richter, CA Woods, P Bergenske, M Fahmy, D Luensmann, M Despres, D Fonn	THE EFFECT OF MASKING ON SUBJECTIVE RESULTS WITH DAILY DISPOSABLE CONTACT LENSES (paper)
Susan Leat	VISION IN AGING SIG – CE lecture chair and co-founder of this group (inaugural meeting this year)	Ping Situ (first author) Trefford Simpson, Marc Schulze, Kara Menzies, Hendrik Walther, Lyndon Jones	INTRA- AND INTER-OPERATOR VARIABILITY OF MERIDIONAL CORNEAL AND EPITHELIAL THICKNESS MEASUREMENTS OBTAINED USING OPTICAL COHERENCE TOMOGRAPHY (OCT) (poster)
Salsabeel Jadi (first author) Miriam Heynen, Doerte Luensmann, Lyndon Jones	INCUBATION SOLUTION COMPOSITION IMPACTS IN VITRO PROTEIN UPTAKE TO SILICONE HYDROGEL CONTACT LENSES (paper)	Lindsay Paquette (first author) Debbie Jones, Megan Despres, Krithika Nandakumar, Craig Woods	EASE OF CONTACT LENS FITTING AND TRAINING IN A CHILD AND YOUTH POPULATION (poster)
Carolyn Machan (first author) Patricia Hryncak, Elizabeth Irving	STATIN USE, TYPE 2 DIABETES AND AGE- RELATED CATARACT: WATERLOO EYE STUDY (paper)		
Thomas Freddo	THE MEDICAL WORK-UP OF THE RED EYE – CE(TQ) Lecture		
Thomas Freddo	CONJUNCTIVAL LESIONS: LINKS TO SYSTEMIC DISEASE – CE(TQ) Lecture		

Eye See Eye Learn

The Benefit of Comprehensive Eye Examinations for Preschoolers

ABSTRACT

Objective: Undetected vision problems in children can lead to permanent vision loss, a condition known as amblyopia. Early detection and treatment of the causes of amblyopia may prevent this vision loss. The objective of this paper is to look for evidence that comprehensive eye examinations upon entry to junior Kindergarten are an effective way to identify and treat vision problems early.

Methods: Relevant peer-reviewed publications on amblyopia and the importance of comprehensive eye examinations were reviewed. Specific areas investigated include: the prevalence and causes of amblyopia; impact of vision problems on child development and education; impact of amblyopia and/or strabismus on quality of life; and the cost effectiveness of treating amblyopia. The validity of vision screening compared to a comprehensive eye examination was also reviewed.

Synthesis: The review suggests that without a complete eye examination many eye or vision problems remain undetected at school entry. Left uncorrected these problems negatively impact child development, education and quality of life. Reduced vision due to amblyopia also restricts future employment opportunities and increases the risk of bilateral visual impairment in adulthood. Examination procedures with high sensitivity and specificity are required to accurately detect these problems. Studies show that amblyopia treatment initiated at an early age is one of the most cost-effective of all health interventions.

Conclusion: There is good evidence in the literature that a full eye examination is critical to detect all cases of amblyopia. This and other visual problems can be detected and managed at an early age, which leads to better visual quality of life and economical outcomes. The *Eye See Eye Learn* program offers the "gold standard" of eye care.

BY DEBORAH A. JONES FCOptom, DipCLP, FAAO;
CATHERINE A. CHIARELLI OD, FAAO;
BARBARA E. ROBINSON OD, MPH, PhD, FAAO;
KAREN E. MACDONALD OD, FAAO

Currently, there are many eye care initiatives underway for the paediatric and preschool population, including vision screenings and comprehensive eye examinations. Although both programs are valuable in facilitating early detection and subsequent treatment of vision problems, their relative effectiveness differs. This paper is an evidence-based literature review conducted to determine which testing strategy provides the best visual and social outcomes in the most cost effective manner. It was written with the intention of appropriately guiding the decisions of health and education policy-makers.

Introduction

Should children have a comprehensive eye examination upon entry to junior Kindergarten? This question has received much attention in the literature. This paper is intended to provide a review of the current literature reporting the benefits for children undergoing a comprehensive eye examination with an optometrist during their entry year into school. The *Eye See Eye Learn* (ESEL) program was developed to raise awareness among parents of the importance of identifying and treating vision problems early. The program provides comprehensive eye examinations by local optometrists to junior Kindergarten kids in participating school regions. This paper

will report on scientific data and expert opinion as evidence that the *Eye See Eye Learn* program is of benefit to children. Papers included in this review are not limited to the visual implications of this program but also include both the short and long-term social ramifications for children.

Eye See Eye Learn Background

In 2003, the Alberta Ministry of Children's Services, the Alberta Association of Optometrists, the Alberta Public Health divisions of Capital and East Central Health Authorities, and the Elk Island Public School Board formed a partnership and created the *Eye See Eye Learn* program. As a result of the program the percentage of junior

Kindergarten children receiving an eye exam rose from 14% to 45%. Of the children examined, 12% were found to have a previously undiagnosed eye or vision problem: 6% required eyeglasses, 4% had eye coordination issues and 2.5% had amblyopia. Based on the successful outcome of the Elk Island ESEL pilot, the program was expanded to be province-wide in Alberta in 2007. Subsequently, the ESEL pilot programs began in 2008 in Saskatchewan involving the Saskatoon school board and in 2009 in Ontario involving the Hamilton school board. In both of these provinces ESEL has expanded and most recently in Ontario in 2010, government support of \$200,000 per year for 5 years will allow the program to further expand. Currently, talks are underway in Manitoba and New Brunswick to adopt the ESEL program.

Visual Benefits of ESEL

The *Eye See Eye Learn* program provides comprehensive eye examinations for children entering school. This involves assessment of visual acuity, refractive error, eye coordination and eye health.

It could be argued that if a child has no vision problems at all then it really makes little difference whether they have a vision screening or a full examination. However the argument becomes very significant when children with vision problems are considered. Undetected vision problems can lead to permanent vision loss (amblyopia).

There have been many definitions of amblyopia in textbooks and in publications. One of the

more simple statements originated from Von Graefe who described amblyopia as the condition “in which the observer sees nothing and the patient very little” cited by Grounds.¹ Amblyopia typically develops when the image in one or both eyes is blurred or obscured and is a significant cause of unilateral vision loss, and is one of the most common causes of persistent unilateral vision impairment in adulthood. Prevalence has been estimated to be between 2% and 5% in children²⁻⁴ and between 0.35% and 3.2% in adults.⁵⁻⁷

Amblyopia occurs in childhood and, if treated while the visual system is still maturing, may be reversible resulting in normal vision. There are many causes, the two most common are strabismus and anisometropia (a difference in refractive error between the two eyes). In a population sample of 3-6 year olds with amblyopia, 38% was found to be associated with strabismus and 37% with anisometropia.⁸ This is significantly different to a cohort of children, younger than three years-of-age, in which strabismus is the primary cause of amblyopia (82%).⁹ Children with strabismus tend to be referred to an eye care professional at an earlier age as parents and/or family physicians at routine health checks often detect strabismus. Another common cause of amblyopia is uncorrected astigmatism or uncorrected high refractive error of any kind.¹⁰

The main steps in the diagnosis of amblyopia are: measurement of vision in each eye; measurement of refractive error; evaluation of eye alignment and movement;

examination of the health of the eyes to rule out pathology (eye disease); and rechecking the vision with eyeglasses, as required. These steps can only be accurately carried out by a trained eye care professional and are the components of a full eye examination.

In most cases amblyopia occurs in one eye only so that even severe amblyopia may go unnoticed by the child or their caregiver. In everyday life unilateral amblyopia results in poor depth perception. Reduced depth perception has an adverse effect on many tasks for young children that involve good hand-eye coordination, such as penmanship and dexterity with scissors.^{11,12} Children with reduced depth perception can be challenged by ball sports and do not perceive the effects of modern 3-D movies.

Amblyopia is a preventable and treatable condition. There are many forms of treatment for amblyopia. The type of amblyopia dictates the treatment modality. For amblyopia related to a large refractive error, eyeglasses may be all that is required. The results of a comparative case series indicated that children aged five to seven years with astigmatism who had been provided with spectacles prior to Kindergarten showed significantly better corrected visual acuity than did children of similar ages who had not received their glasses prior to entering Kindergarten.¹³

Amblyopia that is not fully treated with eyeglasses alone, and amblyopia that is related to strabismus, is treated by occlusion (patching) of the non-amblyopic eye. This

usually is done a few hours per day for several months. After amblyopia treatment is complete, some children also need vision training or surgery to correct strabismus.

The evidence that early detection of amblyopia is vital to the success of treatment is compelling. It is well documented that younger children respond better to treatment than older children and although there is evidence that amblyopia can be treated even in adult years there is little evidence to suggest that normal visual acuity may be achievable.

A recently published study looked at the effect of age on response to amblyopia treatment in children. The results of the study demonstrated that children aged 7-13 years were significantly less responsive to treatment compared with younger subjects (aged three to seven years). Older children did show improvement in vision with treatment but the amount of improvement was less than in the younger children. There is a greater chance of obtaining normal vision in an eye with amblyopia if treatment is initiated before the age of seven.¹⁴

The Canadian Association of Optometrists recommends that a child's first eye examination be at six months-of-age and then again at three years. There is good evidence to support this recommendation. It can be shown from Ontario Health Insurance Plan (OHIP) evidence that children are not having their eyes examined and in fact less than 25% of children in Ontario have an annual eye examination by an optometrist.

Many authors have suggested pre-school vision screening or eye examinations would be beneficial in order to detect and treat amblyopia. Holmes in 2006 stated, "Based on the current evidence, if one screening session is used, screening at school entry could be the most reasonable time."¹⁵ The *Eye See Eye Learn* program provides full comprehensive eye examinations at school entry age for all children.

Developmental Benefits of ESEL

The impact of uncorrected vision problems may be seen in many areas of child development. Effects on the development of motor skills, behaviour and attention, learning skills and reading ability, and general quality-of-life, have been studied extensively. The goal of ESEL is to identify and treat vision problems in early childhood, thus minimizing such secondary consequences.

Motor skills are influenced by visual input. Amblyopia causes reduced vision and eye coordination, and can affect the development of motor skills. Children with amblyopia demonstrate reduced fine motor skills, especially for tasks requiring speed and accuracy. This is especially characteristic of children with amblyopia related to strabismus.¹⁶

Children with amblyopia also demonstrate reduced reach-to-grasp performance.¹⁷ The importance of accurate eye coordination in developing precise hand-eye coordination increases, as children

grow older. The successful treatment of amblyopia can improve hand action control.¹⁷ Children with uncorrected vision problems may demonstrate behavioural, emotional or attention problems when confronted with visual tasks.¹⁸ Increases in misconduct, hyperactivity and aggressiveness have been reported.¹⁹ This is of special significance in children with learning difficulties, who may be unable to effectively communicate their visual symptoms.²⁰

Children with symptomatic convergence insufficiency and/or weak accommodation have been shown to have higher scores on surveys of their behaviour related to deficiencies in school performance (including inattention, avoidance, opposition, hyperactivity).^{21,22}

Educational Benefits of ESEL

Vision is considered the most important sense for learning and it is estimated that 80% of what children learn in primary grades is gained through visual input.²³ Students spend 30-60% of the school day on sustained reading, writing and other near point tasks. Uncorrected vision problems can create strain or distraction during these activities, forcing children to work harder to perform well.¹⁸

The relationship between visual function and academic performance has been highlighted in a policy statement from the American Optometric Association (AOA) / American Academy of Optometry (AAO),²⁴ in which it is

stated that identification and treatment of vision problems enhances learning potential.

Early vision evaluation should be part of a multi-disciplinary approach to ensuring that children reach their full learning potential.²⁵⁻²⁷ Most professionals agree that, although vision problems are not the single cause of learning difficulties in most cases, they can be a relevant factor that influences a child's ability to perform required academic tasks and to use vision to access the curriculum effectively.²⁵⁻²⁷

There are numerous publications that report on how vision problems affect academic achievement. These are summarized below:

- Reduced visual acuity, especially at near, has been reported to be more common in children with learning difficulties.^{28,29}
- Hyperopia has been shown to be strongly linked with reduced literacy skills.²⁹⁻³³ Correction of hyperopia has been shown to result in improved reading achievement.³¹⁻³³
- Anisometropia also has been demonstrated to be more common in children with poor reading skills.^{29,33}
- Eye coordination skills allow accurate, efficient and comfortable input of visual information. Weakness in eye coordination skills results in discomfort, reduced concentration and slower processing speed.^{29,34-36}
- Weakness in eye coordination skills also may interfere with phonetic or eidetic decoding and spelling.^{37,38} In different studies,

reading deficiencies have been correlated with unstable eye coordination,^{18,44,45} poor eye movements,⁴⁶⁻⁴⁹ reduced vergence,^{28,29,34,39-42} reduced accommodation^{28,43-47} and reduced depth perception.⁴¹

- In preschool children, reduced depth perception and reduced accommodation were found to be predictors of reading performance in Kindergarten and Grade One.⁴⁸
- The number of people suffering from amblyopia who complete higher university degrees is considerably fewer than those without amblyopia.⁴⁹

Quality of Life Benefits of ESEL

A number of studies have investigated the impact of amblyopia and/or strabismus on Quality-of-Life (QoL). Different QoL surveys have been administered to children with these conditions, their caregivers, adults who were treated for these conditions in childhood, and adults with residual amblyopia and/or strabismus.^{11,19,49-56}

For individuals with amblyopia, many QoL issues are related to the impact of treatment (i.e. patching therapy) rather than the condition itself. Individuals may develop low self-esteem and a negative self-image,^{51,54} and may experience feelings of depression, frustration, embarrassment,^{54,55} or shame.¹⁹ Many become distressed about their appearance⁵⁹ and worry about losing their eyesight in the future.²⁸ Children with amblyopia are 37%

more likely to be the object of bullying or discrimination.^{19,52,55} They often perceive a lower social acceptance⁵¹ and sometimes avoid social events⁵⁵ because of how they feel about their condition. Amblyopia treatment impacts family life, causing increased stress and anxiety for the caregiver and altering the caregiver-child relationship and other family relationships.¹⁹ This highlights the importance of early intervention, since early identification and treatment of amblyopia are associated with shorter treatment times and more successful outcomes. In addition, the social consequences of amblyopia treatment may have less impact on younger children.⁵²

Untreated amblyopia affects the ability to complete daily tasks. In one study, 55% of individuals with amblyopia reported that it affected their performance in school, 48% reported that it interfered with their work and 50% felt that it influenced their general lifestyle.⁶¹ Reduced vision due to amblyopia also restricts certain employment opportunities that have specific vision standards, such as the armed forces.¹¹

Individuals with amblyopia also are at greater risk for future vision loss. They are more prone to ocular injury and have an increased five-year incident risk of visual loss in the better eye.⁴⁹

Overall, persons with amblyopia have a lifetime risk of vision loss that is almost double that for persons without amblyopia (18% vs 10%), and typically suffer a longer duration of bilateral visual impairment in their lifetime.^{57,58}

A survey in the UK in 2002 found that only 35% of people who lost the vision in their non-amblyopic eye were able to continue in paid employment.⁵⁹ The early identification and treatment of amblyopia in childhood can prevent such tragic visual impairment in adulthood.

Individuals with strabismus also report significant QoL concerns.^{19,52,60-64} They report discomfort when driving, difficulty maintaining eye contact, and anxiety about their appearance and social acceptance. This may affect emotional self-esteem and personal relationships. This highlights the need for early identification and treatment of strabismus, to avoid life-long consequences.

Advantages of ESEL over other Children's Vision Programs

Children deserve our best effort to help them maximize their vision, general development, education and quality of life by accurately identifying and treating vision problems early.

ESEL is a program offering full eye examinations in an optometrist's office, unlike a vision screening which is an assessment of specific aspects of visual function carried out in a location of convenience. This distinction is critical as there are many implicit advantages to having the child visit the optometrist's office. At the optometrist's office the assessment is carried out by a team of professionals comprised of the optometrist and trained support staff,

while at a vision screening the tests are often performed by lay volunteers. An optometrist evaluates all aspects of vision and eye health. Vision screenings often isolate single tests such as visual acuity and depth perception tests or incorporate automated testers that can give false readings that are difficult for screeners to interpret. On-site, at the optometrist's office, specialized instrumentation is available to aid in the accurate evaluation of a young patient. Vision screenings, by definition, necessitate portability, which implies significant limitations to what testing can be performed – many essential tests are simply not possible.

Although vision screenings have been effective at raising awareness for the need for paediatric visual assessments by a convenient mechanism, there are numerous reports in the literature that point to validity problems. In a 1992-4 study, 3,434 Oxford County preschoolers in Ontario underwent a vision screening. Of the 1,017 preschoolers who failed the screening only 384 (38%) actually were found to have a vision problem.⁶⁵ In this study the sensitivity (ability to accurately identify children with vision problems) and specificity (ability to accurately identify children without vision problems) were both low (60.4% and 79.7%) so the vision screening did not do a good job of identifying children with and without vision problems. When a vision problem is detected during a vision screening there is no guarantee that follow-up care will be sought. It has been shown that

40% of children who fail an initial vision screening do not receive the appropriate follow-up care.⁶⁶

The Enhanced Vision Screening Program (EVSP) assessed the negative predictive value (percentage of children who pass the screening who do not have any vision problems) in vision screenings involving 11,734 children and reached a similar conclusion.⁶⁷ The main goal of these vision screenings was to detect amblyopia, strabismus and high refractive error. Of the children who passed the screening, 200 were randomly selected to undergo the “gold standard” – a strictly defined eye examination. The results showed a negative predictive value of 97.6% and the authors conclude:

Because the negative predictive value of the EVSP is not 100%, some children with amblyopia, strabismus or refractive errors are missed...parents should be aware of this.'

The Vision in Preschoolers Study (VIP study) was a multi-phased, multi-centre, interdisciplinary, clinical study to evaluate the accuracy of screening tests used to identify preschool-aged children in need of further evaluation for vision disorders.⁶⁸ The gold standard against which the screenings were tested was a comprehensive eye examination (100% sensitivity and specificity). In the phase II conclusions it was noted that:

'The best performing tests had high testability whether performed by trained eye care professionals, nurses or lay screeners but detection of strabismus was improved by the use of cover test by doctors...'

And from the Preschool Vision Screening: Rationale, Methodology and Outcome, came:

'The relatively low prevalence of amblyopia makes it difficult to achieve a high screening yield in terms of predictive value...unless a 'supertest' can be devised, with very high sensitivity and specificity, health policy decisions will be required to determine which of these two characteristics should be emphasized.'

As indicated by these authors, vision screenings often fail to correctly identify the 2-5% of children with amblyopia and in the process give false reassurances to parents that their child's vision is normal. A comprehensive eye examination with an optometrist could be considered to be the "supertest".

Cost Effectiveness of ESEL

While there has not been a direct analysis of the cost-effectiveness of ESEL the benefit of such a program may be inferred from the literature. The cost-effectiveness of screening and treatment of amblyopia has been examined in the United States and in Europe.

A cost-benefit analysis of five vision screening programs in the United States showed that the greater the sensitivity of the screening method the more beneficial the program.⁶⁹ This study also found that the highest net benefit was for children three to four years-of-age. A study in Germany calculated that the cost-effectiveness of vision screening in children three years-of-age was 727 euros per case detected.⁷⁰ The measure of effectiveness was determined by the number of

newly diagnosed cases of amblyopia as well as cases of strabismus and refractive errors likely to cause amblyopia.

Optometrists in Ontario provide a "gold standard" eye examination that will provide better case finding than the methods described in either of these two papers.

Systematic reviews of the effectiveness of screening preschool children for amblyopia have reported insufficient evidence due to lack of randomized controlled trials conducted in this area.^{71,72} However other reviews have noted that treatment of strabismus and amblyopia can improve visual outcomes. The U.S. Preventive Services Task Force (USPSTF) states that it is important to detect amblyopia, strabismus and defects in visual acuity in children younger than 5 years-of-age.⁷³

Studies based on U.S. and German data concluded that treatment for amblyopia is likely to be very cost effective.^{74,75} In both studies the cost effectiveness was based on Quality-Adjusted Life-Years (QALYs). The U.S. study showed that amblyopia therapy initiated at four years-of-age, including both surgical and nonsurgical treatment, yields a \$/QALY gained of \$2,281.⁷⁴ The authors state that "interventions with a \$/QALY gained of <\$20,000 are especially cost-effective".

The study based on German data found that treatment for amblyopia starting at three years of age was more favourable than many other health care interventions.⁷⁵ They found the

incremental cost effectiveness ratio of treatment was 2,369 euros/QALY. It is interesting that although each study used a different model for analysis both found similar results.

Conclusion

In conclusion there is good evidence in the literature that a full eye examination at school entry age is beneficial. Amblyopia and other visual problems can be detected and managed at an early age, which leads to better visual, quality-of-life and economical outcomes. The *Eye See Eye Learn* program offers the "gold standard" of eye care.

Author Affiliations

Deborah A. Jones FCOptom, DipCLP, FAAO *Lecturer, School of Optometry, University of Waterloo*

Catherine Chiarelli OD, FAAO *Vision Institute of Canada*

Barbara Robinson OD, MPH, PhD, FAAO *Associate Professor, School of Optometry, University of Waterloo*

Karen MacDonald OD, FAAO *Children's Vision Committee Chair, Ontario Association of Optometrists*

References

1. Barnard N. Edgar D. Pediatric Eye Care: Blackwell Science; 1995.
2. Robaei D, Rose K, Ojaimi E, Kifley A, Huynh S, Mitchell P. Visual acuity and the causes of visual loss in a population-based sample of 6-year-old Australian children. Ophthalmology. Jul 2005;112(7):1275-1282.
3. Thompson JR, Woodruff G, Hiscox FA, Strong N, Minshull C. The incidence and prevalence of amblyopia detected in childhood. Public Health. Nov 1991;105(6):455-462.
4. Ross E MA, Stead S. Prevalence of amblyopia in grade 1 schoolchildren

- in Saskatoon. Can J Public Health. 1997;68:491-493.
5. Rosman M, Wong TY, Koh CL, Tan DT. Prevalence and causes of amblyopia in a population-based study of young adult men in Singapore. Am J Ophthalmol. Sep 2005;140(3):551-552.
 6. Brown SA, Weih LM, Fu CL, Dimitrov P, Taylor HR, McCarty CA. Prevalence of amblyopia and associated refractive errors in an adult population in Victoria, Australia. Ophthalmic Epidemiol. Dec 2000;7(4):249-258.
 7. Attebo K, Mitchell P, Cumming R, Smith W, Jolly N, Sparkes R. Prevalence and causes of amblyopia in an adult population. Ophthalmology. Jan 1998;105(1):154-159.
 8. PEDIG. The clinical profile of moderate amblyopia in children younger than 7 years. Arch Ophthalmol. 2002;120(3):281-287.
 9. Birch EE, Holmes JM. The clinical profile of amblyopia in children younger than 3 years of age. J Aapos. Dec 2010;14(6):494-497.
 10. Harvey EM, Dobson V, Miller JM. Prevalence of high astigmatism, eyeglass wear, and poor visual acuity among Native American grade school children. Optom Vis Sci. Apr 2006;83(4):206-212.
 11. Webber AL, Wood J. Amblyopia: prevalence, natural history, functional effects and treatment. Clin Exp Optom. Nov 2005;88(6):365-375.
 12. Fielder AR, Moseley MJ. Does stereopsis matter in humans? Eye (Lond). 1996;10 (Pt 2):233-238.
 13. Dobson V, Clifford-Donaldson CE, Green TK, Miller JM, Harvey EM. Optical treatment reduces amblyopia in astigmatic children who receive spectacles before kindergarten. Ophthalmology. May 2009;116(5):1002-1008.
 14. Holmes JM, Lazar EL, Melia BM, et al. Effect of Age on Response to Amblyopia Treatment in Children. Arch Ophthalmol. Jul 11 2011.
 15. Holmes JM, Clarke MP. Amblyopia. Lancet. Apr 22 2006;367(9519):1343-1351.
 16. Webber AL, Wood JM, Gole GA, Brown B. The effect of amblyopia on fine motor skills in children. Invest Ophthalmol Vis Sci. Feb 2008;49(2):594-603.
 17. Suttie CM, Melmoth DR, Finlay AL, Sloper JJ, Grant S. Eye-hand coordination skills in children with and without amblyopia. Invest Ophthalmol Vis Sci. Mar 2011;52(3):1851-1864.
 18. Goldstand S, Koslowe KC, Parush S. Vision, visual-information processing, and academic performance among seventh-grade schoolchildren: a more significant relationship than we thought? Am J Occup Ther. Jul-Aug 2005;59(4):377-389.
 19. Koklanis K, Abel LA, Aroni R. Psychosocial impact of amblyopia and its treatment: a multidisciplinary study. Clin Experiment Ophthalmol. Nov 2006;34(8):743-750.
 20. Pilling R. Learning disability: challenging behaviour. Br J Ophthalmol. Oct 2008;92(10):1436.
 21. Rouse M, Borsting E, Mitchell GL, et al. Academic behaviors in children with convergence insufficiency with and without parent-reported ADHD. Optom Vis Sci. Oct 2009;86(10):1169-1177.
 22. Borsting E, Rouse M, Chu R. Measuring ADHD behaviors in children with symptomatic accommodative dysfunction or convergence insufficiency: a preliminary study. Optometry. Oct 2005;76(10):588-592.
 23. Moore J. The visual system and engagement in occupation. Journal of Occupational Science: Australia. 1996;3(1):16-17.
 24. American Academy of Optometry AOA. Vision, Learning and Dyslexia. A joint organizational policy statement. Optometry and Vision Science 1997;74(10):868-870.
 25. Solan HA. Dyslexia and learning disabilities: epilogue. Optom Vis Sci. May 1993;70(5):392-393.
 26. Levine MD. Reading disability: do the eyes have it? Pediatrics. Jun 1984;73(6):869-870.
 27. Handler SM, Fierson WM, Section on O. Learning disabilities, dyslexia, and vision. Pediatrics. Mar 2011;127(3):e818-856.
 28. Grisham D, Powers M, Riles P. Visual skills of poor readers in high school. Optometry. Oct 2007;78(10):542-549.
 29. Garzia RP, Nicholson SB. Visual function and reading disability: an optometric viewpoint. J Am Optom Assoc. Feb 1990;61(2):88-97.
 30. Williams WR, Latif AH, Hannington L, Watkins DR. Hyperopia and educational attainment in a primary school cohort. Arch Dis Child. Feb 2005;90(2):150-153.
 31. Association APH. Policy statement Improving early Childhood Eyecare. 2001.
 32. American Academy of Pediatrics Committee on Practice and Ambulatory Medicine: Vision screening and eye examination in children. Pediatrics. Jun 1986;77(6):918-919.
 33. Grisham JD, Simons HD. Refractive error and the reading process: a literature analysis. J Am Optom Assoc. Jan 1986;57(1):44-55.
 34. Williams G. New opportunities in vision therapy. Optometry. Dec 2009;80(12):717-720.
 35. Rawstron JA, Burley CD, Elder MJ. A systematic review of the applicability and efficacy of eye exercises. J Pediatr Ophthalmol Strabismus. Mar-Apr 2005;42(2):82-88.
 36. Satyan HS. Management of children with reading difficulties: a multidisciplinary approach. J Learn Disabil. Oct 1980;13(8):435-439.
 37. Cornelissen P, Bradley L, Fowler S, Stein J. Covering one eye affects how some children read. Dev Med Child Neurol. Apr 1992;34(4):296-304.
 38. Cornelissen P, Bradley L, Fowler S, Stein J. What children see affects how they spell. Dev Med Child Neurol. Aug 1994;36(8):716-726.
 39. Ygge J, Lennerstrand G. Visual impairment and dyslexia in childhood. Curr Opin Ophthalmol. Oct 1997;8(5):40-44.
 40. Kirkby JA, Webster LA, Blythe HI, Liveredge SP. Binocular coordination during reading and non-reading tasks. Psychol Bull. Sep 2008;134(5):742-763.
 41. Palomo-Alvarez C, Puell MC. Binocular function in school children with reading difficulties. Graefes Arch Clin Exp Ophthalmol. Jun 2010;248(6):885-892.
 42. Buzzelli AR. Stereopsis, accommodative and vergence facility: do they relate to dyslexia? Optom Vis Sci. Nov 1991;68(11):842-846.
 43. Evans BJ. The underachieving child. Ophthalmic Physiol Opt. Mar 1998;18(2):153-159.
 44. Kaye G. Vision and learning to read. Clin Exp Optom. Mar 2002;85(2):111.
 45. Palomo-Alvarez C, Puell MC. Accommodative function in school children with reading difficulties. Graefes Arch Clin Exp Ophthalmol. Dec 2008;246(12):1769-1774.
 46. Maples WC. Visual factors that significantly impact academic performance. Optometry. Jan 2003;74(1):35-49.

47. Shin HS, Park SC, Park CM. Relationship between accommodative and vergence dysfunctions and academic achievement for primary school children. *Ophthalmic Physiol Opt*. Nov 2009;29(6):615-624.
48. Solan HA. Visual deficits and dyslexia. *J Learn Disabil*. Jul-Aug 1999;32(4):282-283.
49. Chua B, Mitchell P. Consequences of amblyopia on education, occupation, and long term vision loss. *Br J Ophthalmol*. Sep 2004;88(9):1119-1121.
50. Carlton J, Kaltenthaler E. Amblyopia and quality of life: a systematic review. *Eye (Lond)*. Apr 2011;25(4):403-413.
51. Webber AL, Wood JM, Gole GA, Brown B. Effect of amblyopia on self-esteem in children. *Optom Vis Sci*. Nov 2008;85(11):1074-1081.
52. Williams C, Harrad R. Amblyopia: contemporary clinical issues. *Strabismus*. Mar 2006;14(1):43-50.
53. Damji KF. Vision screening programs in children. *Can Fam Physician*. May 1988;34:1133-1139.
54. Packwood EA, Cruz OA, Rychwalski PJ, Keech RV. The psychosocial effects of amblyopia study. *J Aapos*. Feb 1999;3(1):15-17.
55. Sabri K, Knapp CM, Thompson JR, Gottlob I. The VF-14 and psychological impact of amblyopia and strabismus. *Invest Ophthalmol Vis Sci*. Oct 2006;47(10):4386-4392.
56. Rahi JS, Cumberland PM, Peckham CS. Does amblyopia affect educational, health, and social outcomes? Findings from 1958 British birth cohort. *Bmj*. Apr 8 2006;332(7545):820-825.
57. Nilsson J. The negative impact of amblyopia from a population perspective: untreated amblyopia almost doubles the lifetime risk of bilateral visual impairment. *Br J Ophthalmol*. Nov 2007;91(11):1417-1418.
58. van Leeuwen R, Eijkemans MJ, Vingerling JR, Hofman A, de Jong PT, Simonsz HJ. Risk of bilateral visual impairment in individuals with amblyopia: the Rotterdam study. *Br J Ophthalmol*. Nov 2007;91(11):1450-1451.
59. Rahi J, Logan S, Timms C, Russell-Egitt I, Taylor D. Risk, causes, and outcomes of visual impairment after loss of vision in the non-amblyopic eye: a population-based study. *Lancet*. Aug 24 2002;360(9333):597-602.
60. Hatt SR, Leske DA, Kirgis PA, Bradley EA, Holmes JM. The effects of strabismus on quality of life in adults. *Am J Ophthalmol*. Nov 2007;144(5):643-647.
61. Durmian JM, Owen ME, Marsh IB. The psychosocial aspects of strabismus: correlation between the AS-20 and DAS59 quality-of-life questionnaires. *J Aapos*. Oct 2009;13(5):477-480.
62. Hatt SR, Leske DA, Adams WE, Kirgis PA, Bradley EA, Holmes JM. Quality of life in intermittent exotropia: child and parent concerns. *Arch Ophthalmol*. Nov 2008;126(11):1525-1529.
63. Hatt SR, Leske DA, Yamada T, Bradley EA, Cole SR, Holmes JM. Development and initial validation of quality-of-life questionnaires for intermittent exotropia. *Br J Ophthalmol*. Nov 2007;91(11):1412-1416.
64. Hatt SR, Leske DA, Holmes JM. Comparison of quality-of-life instruments in childhood intermittent exotropia. *J Aapos*. Jun 2010;14(3):221-226.
65. Robinson B, Bobier WR, Martin E, Bryant L. Measurement of the validity of a preschool vision screening program. *Am J Public Health*. Feb 1999;89(2):193-198.
66. Donahue SP, Johnson TM, Leonard-Martin TC. Screening for amblyogenic factors using a volunteer lay network and the MTT photoscreener. Initial results from 15,000 preschool children in a statewide effort. *Ophthalmology*. Sep 2000;107(9):1637-1644; discussion 1645-1636.
67. De Becker I, MacPherson HJ, LaRoche GR, et al. Negative predictive value of a population-based preschool vision screening program. *Ophthalmology*. Jun 1992;99(6):998-1003.
68. Kulp MT. Findings from the Vision in Preschoolers (VIP) Study. *Optom Vis Sci*. Jun 2009;86(6):619-623.
69. Joish VN, Malone DC, Miller JM. A cost-benefit analysis of vision screening methods for preschoolers and school-age children. *J Aapos*. Aug 2003;7(4):283-290.
70. Konig HH, Barry JC, Leidl R, Zrenner E. Cost-effectiveness of orthoptic screening in kindergarten: a decision-analytic model. *Strabismus*. Jun 2000;8(2):79-90.
71. Schmucker C, Grosselfinger R, Riemsma R, et al. Effectiveness of screening preschool children for amblyopia: a systematic review. *BMC Ophthalmol*. 2009;9:3.
72. Powell C, Porooshani H, Bohorquez MC, Richardson S. Screening for amblyopia in childhood. *Cochrane Database Syst Rev*. 2005(3):CD005020.
73. Force UPST. Screening for Visual Impairment in Children Younger than 5 years: Recommendation Statement. *Ann Fam Med*. 2004;2:263-266.
74. Membreno JH, Brown MM, Brown GC, Sharma S, Beauchamp GR. A cost-utility analysis of therapy for amblyopia. *Ophthalmology*. Dec 2002;109(12):2265-2271.
75. Konig HH, Barry JC. Cost effectiveness of treatment for amblyopia: an analysis based on a probabilistic Markov model. *Br J Ophthalmol*. May 2004;88(5):606-612.

Brown, blue or green? Eye disease likes all colours.

As an optometrist, you know that a complete eye exam is a great way to detect many serious eye diseases that can lead to vision loss.

Now it's time the rest of the country knows it too.

This May, join CNIB and the CAO for Vision Health Month.

cnib.ca/visionhealthmonth



Eye See Eye Learn

L'avantage des examens complets de la vue chez les enfants d'âge préscolaire

PAR DEBORAH A. JONES FCOptom, DipCLP, FAAO;

CATHERINE A. CHIARELLI OD, FAAO;

BARBARA E. ROBINSON OD, MPH, PhD, FAAO;

KAREN E. MACDONALD OD, FAAO

De nombreuses initiatives en cours portent sur les soins oculovisuels en pédiatrie et chez les enfants d'âge préscolaire, y compris les tests de dépistage et les examens complets de la vue. Même si les deux programmes aident à faciliter le dépistage précoce et le traitement subséquent des problèmes de vision, leur efficacité relative diffère. Cette analyse documentaire factuelle vise à déterminer la stratégie d'examen qui produit les meilleurs résultats visuels et sociaux de la façon la plus rentable. Elle vise à guider comme il se doit les décisions des responsables des politiques sur la santé et l'éducation.

RÉSUMÉ

Objectifs : Chez les enfants, les problèmes de vision non détectés peuvent entraîner une perte permanente de la vision, problème appelé amblyopie. La détection et le traitement précoce des causes de l'amblyopie peuvent éviter cette perte de vision. Cette communication vise à chercher des éléments probants démontrant que les examens complets de la vue au moment de l'entrée en prématernelle constituent un moyen efficace de repérer et de traiter rapidement les problèmes de vision.

Méthodes : Nous avons étudié des publications pertinentes critiquées par des pairs portant sur l'amblyopie et sur l'importance des examens complets de la vue. Les aspects précis étudiés comprennent la prévalence et les causes de l'amblyopie, l'effet des problèmes de vision sur le développement et l'éducation de l'enfant, l'effet de l'amblyopie ou du strabisme sur la qualité de vie et la rentabilité du traitement de l'amblyopie. Nous avons évalué aussi la validité de ces tests de dépistage comparativement à l'examen complet de la vue.

Résumé : L'étude indique que sans examen complet de la vue, beaucoup de problèmes oculovisuels ne sont pas détectés à l'arrivée de l'enfant à l'école. S'ils ne sont pas corrigés, ces problèmes ont un effet négatif sur le développement, l'éducation et la qualité de vie de l'enfant. La baisse de la vue causée par l'amblyopie limite aussi les possibilités d'emploi futures et accroît le risque de déficience visuelle bilatérale chez l'adulte. Des méthodes d'examen très sensibles et spécifiques s'imposent pour repérer ces problèmes avec précision. Des études montrent que le traitement de l'amblyopie à un jeune âge constitue une des interventions les plus rentables en santé.

Conclusion : Les publications contiennent de bons éléments de preuve indiquant qu'un examen complet de la vue joue un rôle crucial dans la détection de tous les cas d'amblyopie. Il est possible de détecter et de traiter l'amblyopie et d'autres problèmes de vision à un jeune âge, ce qui améliore les résultats reliés à la vue, à la qualité de vie et à l'économie. Le programme *Eye See Eye Learn* offre « l'étonnant » des soins oculovisuels.

Introduction

Les enfants devraient-ils subir un examen complet de la vue à leur arrivée à la prématernelle? Cette question attire beaucoup d'attention dans les publications. Ce document présente une étude des publications courantes sur les avantages pour les enfants de se soumettre à un examen complet de la vue chez un optométriste pendant l'année de leur arrivée à l'école. Le programme *Eye See Eye Learn* vise à sensibiliser les parents à l'importance de repérer et de traiter rapidement les problèmes de vision. Le programme offre aux enfants de la prématernelle des régions scolaires participantes des examens complets de la vue effectués par des optométristes locaux. Ce document présente un rapport sur les données scientifiques et les avis d'experts au sujet des éléments de preuve indiquant que le programme *Eye See Eye Learn* est bénéfique pour les enfants. Les documents étudiés ne sont pas limités aux répercussions du programme sur la vision, mais ils traitent aussi des ramifications sociales à court et à long termes pour les enfants.

Antécédents du programme *Eye See Eye Learn*

En 2003, le ministère des Services à l'enfance de l'Alberta, l'Association des optométristes de l'Alberta, les divisions de la santé

publique des régies sanitaires Capital et East Central de l'Alberta et le Conseil des écoles publiques d'Elk Island ont créé un partenariat et lancé le programme *Eye See Eye Learn* (ESEL). Le programme a fait passer de 14 % à 45 % le pourcentage des enfants de la prématernelle qui subissent un examen de la vue. Sur les enfants examinés, on a constaté que 12 % avaient un problème oculaire ou visuel non diagnostiqué : 6 % avaient besoin de lunettes, 4 % avaient des problèmes de coordination des yeux et 2,5 % avaient une amblyopie. À la suite du succès qu'a connu le programme pilote *Eye See Eye Learn* d'Elk Island, on l'a étendu à l'Alberta au complet en 2007. Par la suite, des programmes pilotes ESEL ont fait leur apparition en 2008 en Saskatchewan, où le conseil scolaire de Saskatoon a été mis à contribution, et en 2009 en Ontario, avec la collaboration du conseil scolaire de Hamilton. Dans ces deux provinces, le programme ESEL a pris de l'ampleur et le gouvernement de l'Ontario a annoncé récemment, soit en 2010, un appui de 200 000 \$ par année pendant cinq ans afin de permettre au programme de prendre encore de l'expansion. Des pourparlers en cours au Manitoba et au Nouveau-Brunswick visent à lancer le programme ESEL dans ces provinces.

Avantages du programme ESEL pour la vue

Le programme *Eye See Eye Learn* fournit des examens de la vue complets aux enfants qui entrent à l'école. Ces examens comportent une évaluation de l'acuité visuelle,

de l'erreur de réfraction, de la spécificité, de la coordination des yeux et de la santé de l'œil.

On pourrait soutenir que si un enfant n'a absolument aucun problème de vision, qu'il se soumette à un test de dépistage ou à un examen complet, cela ne fait pas grand différence en réalité. L'argument devient toutefois très important lorsque l'on tient compte des enfants qui ont des problèmes de vision. Les problèmes de vision non détectés peuvent aboutir à une perte permanente de la vision (amblyopie).

Les manuels et les publications contiennent de nombreuses définitions de l'amblyopie. Un des énoncés les plus simples provient de Von Graefe, qui décrivait l'amblyopie comme la situation « où l'observateur ne voit rien et le patient voit très peu », cité par Grounds.¹ L'amblyopie fait habituellement son apparition lorsque l'image dans un des deux yeux ou dans les deux est floue ou obscurcie et constitue une cause importante de perte de vision unilatérale, une des causes les plus courantes de déficit persistant de la vision unilatérale chez les adultes. La prévalence en est estimée entre 2 et 5 % chez les enfants²⁻⁴ et entre 0,35 % et 3,2 % chez les adultes.⁵⁻⁷

L'amblyopie fait son apparition au cours de l'enfance et si elle est traitée avant que le système visuel soit parvenu à maturité, elle peut être réversible et aboutir quand même à une vision normale. Les causes sont nombreuses. Les deux plus courantes sont le strabisme et l'anisométropie (une erreur de réfraction diffé-

rente entre les deux yeux). Dans un échantillon d'enfants de 3 à 6 ans atteints d'amblyopie, on a constaté qu'il y avait un lien avec le strabisme dans 38 % des cas et avec l'anisométropie dans 37 %.⁸ Il s'agit là d'une différence importante par rapport à une cohorte d'enfants de moins de trois ans chez lesquels le strabisme constitue la principale cause d'amblyopie (82 %).⁹ Les enfants qui ont un strabisme ont tendance à être référés plus jeunes à un professionnel des soins oculovisuels, car les parents ou les médecins de famille détectent souvent le strabisme au cours d'exams médicaux de routine. L'astigmatisme non corrigé, ou la réfraction élevée non corrigée de quelque type que ce soit, constitue une autre cause courante d'amblyopie.¹⁰

Les principales étapes du diagnostic de l'amblyopie sont les suivantes : mesure de la vision dans chaque œil; mesure de l'erreur de réfraction; évaluation de l'alignement et du mouvement des yeux, examen de la santé des yeux pour exclure toute réfraction pathologique (maladie des yeux); nouvelle vérification de la vision avec des lunettes au besoin. Seul un professionnel des soins oculovisuels qui a reçu une formation peut effectuer avec précision les évaluations qui constituent les éléments d'un examen complet de la vue.

Dans la plupart des cas, l'amblyopie atteint un œil seulement et c'est pourquoi il se peut que même une amblyopie grave ne soit pas remarquée par l'enfant ou son proche. Dans la vie de tous les jours,

l'amblyopie unilatérale entraîne une mauvaise perception de la profondeur. La baisse de la perception de la profondeur a un effet indésirable sur beaucoup de tâches des jeunes enfants qui nécessitent une bonne coordination oculo-manielle comme la calligraphie et la dextérité avec des ciseaux.^{11,12} Les sports de balle peuvent poser un défi à cause d'une baisse de la perception de la profondeur et les enfants qui ont une mauvaise perception de la profondeur ne perçoivent pas les effets des films modernes en trois dimensions.

L'amblyopie est un problème qu'il est possible d'éviter et de traiter. Il y a de nombreuses façons de traiter l'amblyopie. La méthode de traitement dépend du type d'amblyopie. Dans le cas de l'amblyopie causée par une importante erreur de réfraction, des lunettes peuvent suffire. Les résultats d'une série de cas de comparaison indiquent que les enfants de 5 à 7 ans atteints d'astigmatisme à qui l'on a fourni des lunettes avant la maternelle avaient une acuité visuelle beaucoup mieux corrigée que les enfants du même âge qui n'avaient pas reçu leurs lunettes avant leur arrivée à la maternelle.¹³

L'amblyopie qui n'est pas traitée entièrement par des lunettes seulement et celle qui est reliée au strabisme sont traitées par occlusion (cache-œil) de l'œil qui n'a pas d'amblyopie. Le cache-œil est habituellement porté quelques heures par jour pendant plusieurs mois. Une fois le traitement de l'amblyopie terminé, des enfants doivent aussi faire de la thérapie ou se soumettre à une

intervention chirurgicale pour corriger le strabisme.

Les éléments probants indiquant que le dépistage précoce d'une amblyopie est vital pour que le traitement réussisse sont convaincants. Il est bien reconnu que les enfants plus jeunes répondent mieux aux traitements que les enfants plus âgés et que même si des éléments de preuve démontrent qu'il est possible de traiter l'amblyopie même chez l'adulte, il y a peu de données qui indiquent qu'il peut être possible de rétablir une acuité visuelle normale. Une étude publiée récemment traitait de l'effet de l'âge sur la réaction au traitement de l'amblyopie chez les enfants. Les résultats de l'étude ont démontré que les enfants de 7 à 13 ans répondaient beaucoup moins au traitement que les sujets plus jeunes (âgés de 3 à 7 ans). Le traitement a amélioré la vision des enfants plus âgés mais moins que chez les enfants plus jeunes. Il y a plus de chance de rétablir la vision normale d'un œil atteint d'amblyopie si le traitement commence avant l'âge de 7 ans.¹⁴

L'Association canadienne des optométristes recommande qu'un enfant subisse son premier examen de la vue à 6 mois et en subisse ensuite un autre à 3 ans. De bons éléments de preuve appuient cette recommandation. Les statistiques du RAMO aident à démontrer que les enfants ne subissent pas d'examen de la vue et qu'en fait moins de 25 % des enfants de l'Ontario subissent un examen annuel de la vue effectué par un optométriste.

Beaucoup d'auteurs ont indiqué que les tests de dépistage ou les examens de la vue au niveau préscolaire aideraient à détecter et à traiter l'amblyopie. En 2006, Holmes a affirmé que « Compte tenu des éléments de preuve courants, si l'on utilise une séance de dépistage, c'est probablement à l'arrivée en milieu scolaire qu'il serait le plus raisonnable de le faire.¹⁵ Le programme *Eye See Eye Learn* offre à tous les enfants des examens complets de la vue à leur arrivée en milieu scolaire.

Bienfaits du programme ESEL pour le développement

Les problèmes de vision non corrigés peuvent avoir une incidence sur de nombreux aspects du développement de l'enfant. On a étudié en détail leurs effets sur le développement de la motricité, le comportement et l'attention, les techniques d'apprentissage, l'apprentissage de la lecture et la qualité de vie en général. Le programme ESEL vise à repérer et traiter les problèmes de vision au cours de la petite enfance et à minimiser ainsi de telles répercussions secondaires.

Les intrants visuels ont des répercussions sur la motricité. L'amblyopie fait baisser la vue et réduit la coordination des yeux, ce qui peut avoir un effet sur l'acquisition de la motricité. Les enfants atteints d'amblyopie montrent une baisse de la motricité fine, surtout dans le cas des tâches à exécuter rapidement et avec précision. C'est particulièrement caractéristique

chez les enfants dont l'ambylopie est reliée au strabisme.¹⁶

Les enfants atteints d'ambylopie obtiennent aussi de moins bons résultats lorsqu'ils cherchent à atteindre quelque chose.¹⁷ La coordination exacte des yeux prend de l'importance dans l'acquisition d'une coordination oculo-manuelle précise à mesure que les enfants grandissent. Le traitement réussi de l'ambylopie peut améliorer le contrôle du mouvement de la main.¹⁷

Les enfants qui ont des problèmes de vision non corrigés peuvent avoir des problèmes de comportement, d'affectivité ou d'attention face à des tâches visuelles.¹⁸ On a signalé des aggravations de l'inconduite, de l'hyperactivité et de l'agressivité.¹⁹ C'est particulièrement important chez les enfants qui ont des troubles d'apprentissage, qui peuvent être incapables de faire connaître efficacement leurs symptômes visuels.²⁰

Les enfants qui ont une insuffisance de la convergence symptomatique ou une faiblesse de l'accommodation obtiennent de mauvais résultats à des questionnaires portant sur leur comportement relié aux résultats scolaires (y compris inattention, évitement, opposition, hyperactivité).^{21,22}

Bienfaits du programme ESEL pour l'éducation

La vue est considérée comme le sens le plus important pour apprendre et l'on estime que les enfants acquièrent sous forme de signaux visuels 80 % de ce qu'ils apprennent au primaire.²³ Les élèves passent de 30 à 60 % de

la journée scolaire à lire, à écrire et à effectuer d'autres tâches au proximum de convergence. Les problèmes de vision non corrigés peuvent causer de la fatigue oculaire ou des distractions pendant ces activités et obliger les enfants à travailler plus fort pour obtenir de bons résultats.¹⁸

L'American Optometric Association (AOA) et l'American Academy of Optometry (AAO)²⁴ ont mis en évidence, dans un énoncé de politique, le lien entre la fonction visuelle et les résultats scolaires. Dans cet énoncé, on affirme que la détermination et le traitement des problèmes de vision améliorent le potentiel d'apprentissage.

L'évaluation précoce de la vue doit faire partie d'une approche multidisciplinaire visant à garantir que les enfants atteignent leur plein potentiel d'apprentissage.^{25,27} La plupart des professionnels reconnaissent que même si les problèmes de vision ne sont pas la seule cause des troubles d'apprentissage dans la plupart des cas, ils peuvent constituer un facteur pertinent qui joue sur la capacité d'un enfant d'exécuter des tâches scolaires obligatoires et d'utiliser sa vision pour avoir accès efficacement au programme d'études.²⁵⁻²⁷

De nombreuses publications décrivent l'effet des problèmes de vision sur les résultats scolaires. Nous les résumons ci-dessous :

- On a signalé que la baisse de l'acuité visuelle, surtout de près, est plus courante chez les enfants qui ont des troubles d'apprentissage.^{28,29}

■ Il existe un lien solide entre l'hypermétropie et la baisse des compétences en lecture et en écriture.²⁹⁻³³ Il est démontré que la correction de l'hypermétropie améliore les résultats en lecture.^{31,33}

■ Il a aussi été démontré que l'anisométropie est plus courante chez les enfants qui ont de la difficulté à lire.^{29,33}

■ Les techniques de coordination des yeux permettent de saisir l'information visuelle avec précision, efficience et confort. La faiblesse de la coordination des yeux cause de l'inconfort, fait baisser la concentration et ralentit la vitesse de traitement.^{29,34,36}

■ La faiblesse de la coordination des yeux peut aussi nuire au décodage phonétique ou eidétique et à l'orthographe.^{37,38} Différentes études ont établi un lien entre les déficits de la lecture et l'instabilité de la coordination des yeux,^{18,44,45} des mouvements médiocres des yeux,^{46,49} la réduction de la vergence,^{28,29,34,39-42} la baisse de l'accommodation,^{28,43,47} et une diminution de la perception de la profondeur.⁴¹

■ Chez les enfants d'âge préscolaire, on a constaté une diminution de la perception de la profondeur et de l'accommodation, qui constitue un des prédicteurs des résultats de la lecture au jardin et en première année.⁴⁸

■ Les personnes qui ont une ambylopie et terminent des études universitaires au niveau supérieur sont beaucoup moins nombreuses que celles qui n'ont pas d'ambylopie.⁴⁹

Bienfaits du programme ESEL pour la qualité de vie

Des études ont traité de l'effet de l'ambylopie ou du strabisme sur la Qualité de vie (QDV). Différents questionnaires sur la QDV ont été administrés à des enfants qui ont ces problèmes, à leurs aidants naturels, à des adultes qui ont été traités pour ces problèmes au cours de l'enfance et à des adultes qui ont une ambylopie ou un strabisme résiduel.^{11,19,49-56}

Pour les personnes qui ont une ambylopie, beaucoup de problèmes de QDV sont reliés à l'effet du traitement (c.-à-d. à l'utilisation du cache-œil) plutôt qu'au problème même. Les personnes en cause peuvent avoir une faible estime d'elles-mêmes et une image d'elles-mêmes négative^{51,54} et ressentir de la dépression, de la frustration, de l'embarras^{54,55} ou de la honte.¹⁹ Leur apparence est une cause de détresse pour beaucoup de personnes⁵⁹ qui craignent de perdre la vue un jour.²⁸ Les enfants atteints d'ambylopie sont 37 % plus susceptibles d'être victimes d'intimidation ou de discrimination.^{19,52,55} Ils se croient souvent moins acceptés sur le plan social⁵¹ et évitent parfois les activités sociales⁵⁵ à cause de ce qu'ils pensent de leur problème. Le traitement de l'ambylopie a des répercussions sur la vie familiale, alourdit le stress et l'anxiété pour l'aidant naturel et modifie la relation aidant-enfant et d'autres relations familiales.¹⁹ Ces phénomènes mettent en évidence l'importance de l'intervention rapide puisqu'on établit un lien entre la détermination et le traitement précoce de l'ambylopie et

des traitements de moyenne durée et des résultats plus fructueux. En outre, les conséquences sociales du traitement de l'ambylopie peuvent avoir moins d'effet sur les enfants plus jeunes.⁵²

L'ambylopie non traitée a des répercussions sur la capacité d'effectuer des tâches quotidiennes. Au cours d'une étude, 55 % des personnes qui avaient une ambylopie ont déclaré que leur problème avait un effet sur leurs résultats scolaires, 48 % ont affirmé qu'elle nuisait à leur travail et 50 % pensaient qu'elle avait un effet sur leurs habitudes de vie générales.⁶¹ La baisse de la vision attribuable à l'ambylopie limite aussi certaines possibilités d'emploi qui ont des normes individuelles précises comme dans les forces armées.¹¹

Les personnes atteintes d'ambylopie risquent aussi davantage de perdre la vue à l'avenir. Elles sont plus vulnérables aux traumatismes oculaires et présentent un risque accru d'incidents à après 5 ans de perdre la vue dans leur meilleur œil⁴⁹

Dans l'ensemble, les personnes qui ont une ambylopie risquent presque deux fois plus de perdre la vue (18 % c. 10 %) et ont habituellement un déficit visuel bilatéral de plus longue durée pendant leur vie.^{57,58} Un sondage réalisé au Royaume-Uni en 2002 a révélé que 35 % seulement des personnes qui ont perdu la vue de leur œil non amblyopique ont pu garder un emploi rémunéré.⁵⁹ La détermination et le traitement précoce de l'ambylopie chez les enfants peuvent éviter des déficits visuels aussi tragiques à l'âge adulte.

Les personnes atteintes de strabisme signalent aussi d'importantes préoccupations sur le plan de la QDV.^{19,52,60-64} Elles se sentent mal à l'aise au volant, ont de la difficulté à maintenir le contact visuel et leur apparence et leur acceptation dans la société les rendent anxiées. Ces facteurs peuvent avoir une incidence sur leur estime de soi affective et sur leurs relations personnelles. C'est pourquoi la détermination et le traitement précoce du strabisme s'imposent pour éviter des conséquences qui dureront toute la vie.

Avantages du programme ESEL sur d'autres programmes de protection de la vision chez les enfants

Les enfants méritent notre meilleur effort lorsqu'il s'agit de les aider à maximiser leur vision, leur développement général, leur éducation et leur qualité de vie en déterminant avec précision leurs problèmes de vision et en les traitant rapidement.

Le programme ESEL offre des examens complets de la vue dans le bureau d'un optométriste, contrairement à un test de dépistage qui constitue une évaluation d'aspects précis de la fonction visuelle effectuée à un endroit commode. La distinction est cruciale, car la visite de l'enfant au cabinet de l'optométriste comporte de nombreux avantages impliqués. L'évaluation y est effectuée par une équipe de professionnels constituée de l'optométriste et d'auxiliaires qui ont reçu une

formation, tandis qu'à un centre de dépistage, les tests sont souvent effectués par des bénévoles non initiés. Un optométriste évalue tous les aspects de la vision et de la santé de l'œil. Le dépistage des problèmes visuels isole souvent certains tests en particulier comme les tests d'acuité visuelle et de perception de la profondeur ou utilise des appareils automatisés qui peuvent donner de fausses mesures difficiles à interpréter. Les instruments spécialisés disponibles au cabinet de l'optométriste aident à évaluer un jeune patient avec précision. Les tests de dépistage doivent par définition être portables, ce qui sous-entend que les tests qu'il est possible d'effectuer sont très limités – il y a beaucoup de tests essentiels qu'il est tout simplement impossible d'effectuer.

Même si les tests de dépistage ont réussi à faire mieux connaître la nécessité des évaluations de la vue chez les enfants effectuées par un moyen commode, de nombreux rapports dans les publications signalent des problèmes de validité. Au cours d'une étude réalisée de 1992 à 1994, 3 434 enfants d'âge préscolaire du comté d'Oxford en Ontario se sont soumis à un test de dépistage. Sur les 1 017 enfants d'âge préscolaire qui ont échoué au test, 384 (38 %) seulement avaient en réalité un problème de vision.⁶⁵ Cette étude a révélé que la sensibilité (capacité de repérer exactement les enfants qui ont des problèmes de vision) et la spécificité (capacité de déterminer avec précision les enfants qui n'ont pas de problème de vision) étaient toutes deux faibles (60,4 % et 79,7 %) et que

le test de dépistage n'a donc pas bien réussi à repérer les enfants qui avaient des problèmes de vision et ceux qui n'en avaient pas. Lorsqu'un problème de vision est détecté au cours d'un test de dépistage, rien ne garantit que l'on cherchera à obtenir des soins de suivi. Il a été démontré que 40 % des enfants qui échouent à un premier test de dépistage ne reçoivent pas les soins de suivi appropriés.⁶⁶

Le programme de dépistage amélioré des problèmes de vision (EVSP) a déterminé la valeur prédictive négative (pourcentage des enfants qui réussissent le test et n'ont pas de problèmes de vision) au cours de tests de dépistage portant sur 11 734 enfants et a atteint une conclusion semblable.⁶⁷ Ces dépistages visaient principalement à détecter l'amblyopie, le strabisme et l'erreur de réfraction élevée. Parmi les enfants qui ont réussi le test, on en a choisi 200 au hasard pour les soumettre à l'étau-or – un examen de la vue défini rigoureusement. Les résultats ont révélé une valeur prédictive négative de 97,6 % et les auteurs concluent que :

« Comme la valeur prédictive négative du programme ESVP n'atteint pas 100 %, on ne détecte pas l'amblyopie, le strabisme ou l'erreur de la réfraction chez quelques enfants – et les parents devraient en être conscients ».

L'étude clinique interdisciplinaire multicentrique et à volets multiples sur la vision chez les enfants d'âge préscolaire (essai VIP) devait évaluer la précision des tests de dépistage utilisés pour repérer les enfants d'âge préscolaire qui ont besoin d'une évaluation plus

poussée des troubles de la vision.⁶⁸ L'examen complet de la vue (sensibilité et spécificité de 100 %) constituait l'étau-or en fonction duquel on a vérifié les tests de dépistage. Dans les conclusions de la phase II, on a signalé que :

« Les tests les plus performants présentent une testabilité élevée, qu'ils soient effectués par des professionnels des soins oculovisuels qui ont reçu une formation, des infirmières ou des dépisteurs non formés, mais le test de l'écran effectué par des médecins a amélioré la détection du strabisme... »

On tire aussi la conclusion suivante de l'étude *Preschool Vision Screening: Rationale, Methodology and Outcome* :

« À cause de la prévalence relativement faible de l'amblyopie, il est difficile d'obtenir un rendement élevé au dépistage sur le plan de la valeur prédictive... sauf si l'on peut mettre au point un « super-test » très sensible et spécifique, il y a aura des décisions à prendre sur les politiques de la santé pour déterminer sur laquelle de ces deux caractéristiques il faudrait mettre l'accent ».

Comme l'indiquent ces auteurs, il arrive souvent que les tests de dépistage ne réussissent pas à identifier correctement les 2 à 5 % d'enfants atteints d'amblyopie et donnent aux parents de fausses assurances en leur faisant croire que la vision de leur enfant est normale. On pourrait considérer qu'un examen complet des yeux effectué par un optométriste constitue le « supertest ».

Rentabilité du programme ESEL

Il n'y a pas eu d'analyse directe de la rentabilité du programme ESEL, mais il est possible de déduire des publications l'avantage qu'offre un tel programme. La rentabilité du dépistage et du traitement de l'amblyopie a été étudiée aux États-Unis et en Europe.

Une analyse coûts-avantages portant sur cinq programmes de dépistage aux États-Unis a montré que plus la méthode de dépistage est sensible, plus le programme est bénéfique.⁶⁹ Cette étude a aussi révélé que ce sont les enfants de 3 à 4 ans qui en tirent le plus grand avantage net. Au cours d'une étude réalisée en Allemagne, on a calculé que la rentabilité du dépistage chez les enfants de 3 ans s'établissait à 727 euros par cas détecté.⁷⁰ La mesure d'efficacité a été établie en fonction du nombre des cas nouvellement diagnostiqués d'amblyopie, ainsi que des cas de strabisme et d'erreurs de la réfraction susceptibles de causer l'amblyopie. Les optométristes de l'Ontario offrent un examen de la vue qui constitue l'étalon-or et qui permettra de mieux repérer les cas que les méthodes décrites dans l'un ou l'autre de ces deux documents.

Les critiques systématiques portant sur l'efficacité du dépistage de l'amblyopie chez les enfants d'âge préscolaire n'ont pas produit suffisamment d'éléments de preuve à cause du manque d'essais contrôlés randomisés réalisés dans ce domaine.^{71,72} D'autres critiques ont toutefois signalé que le traitement du strabisme et de l'amblyopie peut améliorer les résultats pour

la vision. Le groupe de travail sur les services de prévention des États-Unis (USPSTF) affirme qu'il importe de détecter l'amblyopie, le strabisme et les défauts de l'acuité visuelle chez les enfants de moins de 5 ans.⁷³

Des études basées sur des données américaines et allemandes ont conclu que le traitement de l'amblyopie sera probablement très rentable.^{74,75} Dans le cadre de ces deux études, l'efficacité des coûts était basée sur les années de vie ajustées par la qualité (AVAQ). L'étude américaine a montré que le traitement de l'amblyopie commencé à 4 ans, ce qui inclut le traitement chirurgical et non chirurgical, produit un gain de 2 281 \$/AVAQ.⁷⁴ Les auteurs affirment que « les interventions qui produisent un résultat de <20 000 \$/AVAQ sont particulièrement rentables ».

L'étude basée sur des données allemandes a révélé que le traitement de l'amblyopie qui commence à 3 ans était plus favorable que beaucoup d'autres interventions en soins de santé.⁷⁵ Les chercheurs ont constaté que l'augmentation du ratio coût-éfficacité du traitement s'établissait à 2 369 euros/AVAQ. Il est intéressant de noter que même si chaque étude a utilisé un modèle d'analyse différent, les deux ont abouti au même résultat.

Conclusion

En conclusion, les publications contiennent de solides éléments probants indiquant que l'examen complet de la vue à l'arrivée dans le système scolaire est bénéfique.

L'amblyopie et d'autres problèmes de vision peuvent être détectés et traités chez les enfants jeunes, ce qui améliore les résultats pour la vision, la qualité de vie et l'économie. Le programme *Eye See Eye Learn* offre « l'étalon-or » des soins oculovisuels.

Affiliations des auteurs

Deborah A. Jones, FCOptom,
DipCLP, chargée de cours FAAO, École
d'optométrie, Université de Waterloo

Catherine Chiarelli, OD, FAAO,
Vision Institute of Canada

Barbara Robinson, OD, MPH, PhD
FAAO, professeure adjointe à l'École
d'optométrie de l'Université de Waterloo

Karen MacDonald, OD, FAAO,
présidente du Comité de la vision pour les
enfants, Association des optométristes de
l'Ontario

Références

1. Barnard N, Edgar D. *Pediatric Eye Care*: Blackwell Science; 1995.
2. Robaei D, Rose K, Ojaimi E, Kifley A, Huynh S, Mitchell P. Visual acuity and the causes of visual loss in a population-based sample of 6-year-old Australian children. *Ophthalmology*. Juil. 2005;112(7):1275-1282.
3. Thompson JR, Woodruff G, Hiscox FA, Strong N, Minshull C. The incidence and prevalence of amblyopia detected in childhood. *Public Health*. Nov. 1991;105(6):455-462.
4. Ross E MA, Stead S. Prevalence of amblyopia in grade 1 schoolchildren in Saskatoon. *Revue canadienne de santé publique*. 1997;68:491-493.
5. Rosman M, Wong TY, Koh CL, Tan DT. Prevalence and causes of amblyopia in a population-based study of young adult men in Singapore. *Am J Ophthalmol*. Sept. 2005;140(3):551-552.
6. Brown SA, Weih LM, Fu CL, Dimitrov P, Taylor HR, McCarty CA. Prevalence of amblyopia and associated refractive errors in an adult population in Victoria, Australia. *Ophthalmic Epidemiol*. Déc. 2000;7(4):249-258.

7. Attebo K, Mitchell P, Cumming R, Smith W, Jolly N, Sparkes R. Prevalence and causes of amblyopia in an adult population. *Ophthalmology*. Janv. 1998;105(1):154-159.
8. PEDIG. The clinical profile of moderate amblyopia in children younger than 7 years. *Arch Ophthalmol*. 2002;120(3):281-287.
9. Birch EE, Holmes JM. The clinical profile of amblyopia in children younger than 3 years of age. *J Aapos*. Déc. 2010;14(6):494-497.
10. Harvey EM, Dobson V, Miller JM. Prevalence of high astigmatism, eyeglass wear, and poor visual acuity among Native American grade school children. *Optom Vis Sci*. Avril 2006;83(4):206-212.
11. Webber AL, Wood J. Amblyopia: prevalence, natural history, functional effects and treatment. *Clin Exp Optom*. Nov. 2005;88(6):365-375.
12. Fielder AR, Moseley MJ. Does stereopsis matter in humans? *Eye (Lond)*. 1996;10 (Pt 2):233-238.
13. Dobson V, Clifford-Donaldson CE, Green TK, Miller JM, Harvey EM. Optical treatment reduces amblyopia in astigmatic children who receive spectacles before kindergarten. *Ophthalmology*. Mai 2009;116(5):1002-1008.
14. Holmes JM, Lazar EL, Melia BM, et al. Effect of Age on Response to Amblyopia Treatment in Children. *Arch Ophthalmol*. 11 juil. 2011.
15. Holmes JM, Clarke MP. Amblyopia. *Lancet*. 22 avril 2006;367(9519):1343-1351.
16. Webber AL, Wood JM, Gole GA, Brown B. The effect of amblyopia on fine motor skills in children. *Invest Ophthalmol Vis Sci*. Févr. 2008;49(2):594-603.
17. Suttle CM, Melmoth DR, Finlay AL, Sloper JJ, Grant S. Eye-hand coordination skills in children with and without amblyopia. *Invest Ophthalmol Vis Sci*. Mars 2011;52(3):1851-1864.
18. Goldstand S, Koslowe KC, Parush S. Vision, visual-information processing, and academic performance among seventh-grade schoolchildren: a more significant relationship than we thought? *Am J Occup Ther*. Juil.-août 2005;59(4):377-389.
19. Koklanis K, Abel LA, Aroni R. Psychosocial impact of amblyopia and its treatment: a multidisciplinary study. *Clin Experiment Ophthalmol*. Nov. 2006;34(8):743-750.
20. Pilling R. Learning disability: challenging behaviour. *Br J Ophthalmol*. Oct. 2008;92(10):1436.
21. Rouse M, Borsting E, Mitchell GL, et al. Academic behaviors in children with convergence insufficiency with and without parent-reported ADHD. *Optom Vis Sci*. Oct. 2009;86(10):1169-1177.
22. Borsting E, Rouse M, Chu R. Measuring ADHD behaviors in children with symptomatic accommodative dysfunction or convergence insufficiency: a preliminary study. *Optometry*. Oct. 2005;76(10):588-592.
23. Moore J. The visual system and engagement in occupation. *Journal of Occupationsl Science*: Australia. 1996;3(1):16-17.
24. American Academy of Optometry AOA. Vision, Learning and Dyslexia. A joint organizational policy statement. *Optometry and Vision Science* 1997;74(10):868-870.
25. Solan HA. Dyslexia and learning disabilities: epilogue. *Optom Vis Sci*. Mai 1993;70(5):392-393.
26. Levine MD. Reading disability: do the eyes have it? *Pediatrics*. Juin 1984;73(6):869-870.
27. Handler SM, Fierson WM, Section on O. Learning disabilities, dyslexia, and vision. *Pediatrics*. Mars 2011;127(3):e818-856.
28. Grisham D, Powers M, Riles P. Visual skills of poor readers in high school. *Optometry*. Oct. 2007;78(10):542-549.
29. Garzia RP, Nicholson SB. Visual function and reading disability: an optometric viewpoint. *J Am Optom Assoc*. Févr. 1990;61(2):88-97.
30. Williams WR, Latif AH, Hannington L, Watkins DR. Hyperopia and educational attainment in a primary school cohort. *Arch Dis Child*. Févr. 2005;90(2):150-153.
31. Association APH. Policy statement Improving early Childhood Eyecare. 2001.
32. American Academy of Pediatrics Committee on Practice and Ambulatory Medicine: Vision screening and eye examination in children. *Pediatrics*. Juin 1986;77(6):918-919.
33. Grisham JD, Simons HD. Refractive error and the reading process: a literature analysis. *J Am Optom Assoc*. Janv. 1986;57(1):44-55.
34. Williams G. New opportunities in vision therapy. *Optometry*. Déc. 2009;80(12):717-720.
35. Rawstron JA, Burley CD, Elder MJ. A systematic review of the applicability and efficacy of eye exercises. *J Pediatr Ophthalmol Strabismus*. Mars-avril 2005;42(2):82-88.
36. Satyan HS. Management of children with reading difficulties: a multidisciplinary approach. *J Learn Disabil*. Oct. 1980;13(8):435-439.
37. Cornelissen P, Bradley L, Fowler S, Stein J. Covering one eye affects how some children read. *Dev Med Child Neurol*. Avril 1992;34(4):296-304.
38. Cornelissen P, Bradley L, Fowler S, Stein J. What children see affects how they spell. *Dev Med Child Neurol*. Août 1994;36(8):716-726.
39. Ygge J, Lennerstrand G. Visual impairment and dyslexia in childhood. *Curr Opin Ophthalmol*. Oct. 1997;8(5):40-44.
40. Kirkby JA, Webster LA, Blythe HI, Liversedge SP. Binocular coordination during reading and non-reading tasks. *Psychol Bull*. Sept. 2008;134(5):742-763.
41. Palomo-Alvarez C, Puell MC. Binocular function in school children with reading difficulties. *Graefes Arch Clin Exp Ophthalmol*. Juin 2010;248(6):885-892.
42. Buzzelli AR. Stereopsis, accommodative and vergence facility: do they relate to dyslexia? *Optom Vis Sci*. Nov. 1991;68(11):842-846.
43. Evans BJ. The underachieving child. *Ophthalmic Physiol Opt*. Mars 1998;18(2):153-159.
44. Kaye G. Vision and learning to read. *Clin Exp Optom*. Mars 2002;85(2):111.
45. Palomo-Alvarez C, Puell MC. Accommodative function in school children with reading difficulties. *Graefes Arch Clin Exp Ophthalmol*. Déc. 2008;246(12):1769-1774.
46. Maples WC. Visual factors that significantly impact academic performance. *Optometry*. Janv. 2003;74(1):35-49.
47. Shin HS, Park SC, Park CM. Relationship between accommodative and vergence dysfunctions and academic achievement for primary school children. *Ophthalmic Physiol Opt*. Nov. 2009;29(6):615-624.
48. Solan HA. Visual deficits and dyslexia. *J Learn Disabil*. Juil.-août 1999;32(4):282-283.
49. Chua B, Mitchell P. Consequences of amblyopia on education, occupation, and long term vision loss. *Br J Ophthalmol*. Sept. 2004;88(9):1119-1121.

50. Carlton J, Kaltenbacher E. Amblyopia and quality of life: a systematic review. *Eye (Lond)*. Avril 2011;25(4):403-413.
51. Webber AL, Wood JM, Gole GA, Brown B. Effect of amblyopia on self-esteem in children. *Optom Vis Sci*. Nov. 2008;85(11):1074-1081.
52. Williams C, Harrad R. Amblyopia: contemporary clinical issues. *Strabismus*. Mars 2006;14(1):43-50.
53. Damji KF. Vision screening programs in children. *Le médecin de famille canadien*. Mai 1988;34:1133-1139.
54. Packwood EA, Cruz OA, Rychwalski PJ, Keech RV. The psychosocial effects of amblyopia study. *J Aapos*. Févr. 1999;3(1):15-17.
55. Sabri K, Knapp CM, Thompson JR, Gottlob I. The VF-14 and psychological impact of amblyopia and strabismus. *Invest Ophthalmol Vis Sci*. Oct. 2006;47(10):4386-4392.
56. Rahi JS, Cumberland PM, Peckham CS. Does amblyopia affect educational, health, and social outcomes? Findings from 1958 British birth cohort. *Bmj*. 8 avril 2006;332(7545):820-825.
57. Nilsson J. The negative impact of amblyopia from a population perspective: untreated amblyopia almost doubles the lifetime risk of bilateral visual impairment. *Br J Ophthalmol*. Nov. 2007;91(11):1417-1418.
58. van Leeuwen R, Eijkemans MJ, Vingerling JR, Hofman A, de Jong PT, Simonsz HJ. Risk of bilateral visual impairment in individuals with amblyopia: the Rotterdam study. *Br J Ophthalmol*. Nov. 2007;91(11):1450-1451.
59. Rahi J, Logan S, Timms C, Russell-Eggett I, Taylor D. Risk, causes, and outcomes of visual impairment after loss of vision in the non-amblyopic eye: a population-based study. *Lancet*. 24 août 2002;360(9333):597-602.
60. Hatt SR, Leske DA, Kirgis PA, Bradley EA, Holmes JM. The effects of strabismus on quality of life in adults. *Am J Ophthalmol*. Nov. 2007;144(5):643-647.
61. Durmian JM, Owen ME, Marsh IB. The psychosocial aspects of strabismus: correlation between the AS-20 and DAS59 quality-of-life questionnaires. *J Aapos*. Oct. 2009;13(5):477-480.
62. Hatt SR, Leske DA, Adams WE, Kirgis PA, Bradley EA, Holmes JM. Quality of life in intermittent exotropia: child and parent concerns. *Arch Ophthalmol*. Nov. 2008;126(11):1525-1529.
63. Hatt SR, Leske DA, Yamada T, Bradley EA, Cole SR, Holmes JM. Development and initial validation of quality-of-life questionnaires for intermittent exotropia. *Ophthalmology*. Janv. 2010;117(1):163-168 e161.
64. Hatt SR, Leske DA, Holmes JM. Comparison of quality-of-life instruments in childhood intermittent exotropia. *J Aapos*. Juin 2010;14(3):221-226.
65. Robinson B, Bobier WR, Martin E, Bryant L. Measurement of the validity of a preschool vision screening program. *Am J Public Health*. Févr. 1999;89(2):193-198.
66. Donahue SP, Johnson TM, Leonard-Martin TC. Screening for amblyogenic factors using a volunteer lay network and the MTI photoscreener. Initial results from 15,000 preschool children in a statewide effort. *Ophthalmology*. Sept. 2000;107(9):1637-1644; discussion 1645-1636.
67. De Becker I, MacPherson HJ, LaRoche GR, et al. Negative predictive value of a population-based preschool vision screening program. *Ophthalmology*. Juin 1992;99(6):998-1003.
68. Kulp MT. Findings from the Vision in Preschoolers (VIP) Study. *Optom Vis Sci*. Juin 2009;86(6):619-623.
69. Joish VN, Malone DC, Miller JM. A cost-benefit analysis of vision screening methods for preschoolers and school-age children. *J Aapos*. Août 2003;7(4):283-290.
70. Konig HH, Barry JC, Leidl R, Zrenner E. Cost-effectiveness of orthoptic screening in kindergarten: a decision-analytic model. *Strabismus*. Juin 2000;8(2):79-90.
71. Schmucker C, Grosselfinger R, Riemsma R, et al. Effectiveness of screening preschool children for amblyopia: a systematic review. *BMC Ophthalmol*. 2009;9:3.
72. Powell C, Porooshani H, Bohorquez MC, Richardson S. Screening for amblyopia in childhood. *Cochrane Database Syst Rev*. 2005(3):CD005020.
73. Force UPST. Screening for Visual Impairment in Children Younger than 5 years: Recommendation Statement. *Ann Fam Med*. 2004;2:263-266.
74. Membreño JH, Brown MM, Brown GC, Sharma S, Beauchamp GR. A cost-utility analysis of therapy for amblyopia. *Ophthalmology*. Déc. 2002;109(12):2265-2271.
75. Konig HH, Barry JC. Cost effectiveness of treatment for amblyopia: an analysis based on a probabilistic Markov model. *Br J Ophthalmol*. Mai 2004;88(5):606-612.

Yeux bruns, bleus ou verts?
Les maladies oculaires aiment toutes les couleurs.

Vous êtes optométriste, vous savez donc qu'un examen oculaire complet pratiqué par un optométriste est une excellente façon de dépister les nombreuses maladies oculaires qui peuvent entraîner une perte de vision. Il est maintenant temps que le reste du pays le sache aussi.

Au mois de mai, joignez-vous à INCA et à l'ACO pour le Mois de la santé visuelle.
inca.ca/moisdelasantevisuelle

INCA
 Institut national de la chambre des optométristes
 Partenaire médical du Mois de la santé visuelle ©INCA

BESIVANCE®

Demonstrated efficacy and an excellent safety profile

BESIVANCE® is indicated for the treatment of patients one year of age and older with bacterial conjunctivitis caused by susceptible strains of the following organisms:

Aerobic, Gram-Positive

- CDC coryneform group G
- *Staphylococcus aureus*
- *Staphylococcus epidermidis*
- *Streptococcus mitis*
- *Streptococcus oralis*
- *Streptococcus pneumoniae*

Aerobic, Gram-Negative

- *Haemophilus influenzae*

BESIVANCE® is contraindicated in patients with known hypersensitivity to this drug, to other quinolones, or to any ingredient in the formulation or component of the container.

**NOT FOR INJECTION INTO THE EYE.
FOR TOPICAL OPHTHALMIC USE ONLY.**

In three safety and efficacy trials, no serious adverse reactions related to Besivance® were reported. The most frequently reported treatment-emergent ocular adverse events in the study eye were blurred vision (2.1%), eye pain (1.9%), and eye irritation (1.4%).



BAUSCH + LOMB

Bausch & Lomb Canada Inc., Vaughan, Ontario L4K 4B4
©/™ denotes trademark of Bausch & Lomb Incorporated
© Bausch & Lomb Incorporated

Pr Besivance®
besifloxacin ophthalmic suspension, 0.6%

Pr Besivance®

Besifloxacin ophthalmic suspension, 0.6%



Prescribing Summary



Patient Selection Criteria

THERAPEUTIC CLASSIFICATION

Antibacterial (ophthalmic)

INDICATIONS AND CLINICAL USE

BESIVANCE® is indicated for the treatment of patients one year of age and older with bacterial conjunctivitis caused susceptible strains of the following organisms:

Aerobic, Gram-Positive

- *CDC coryneform group G*
- *Staphylococcus epidermidis*
- *Streptococcus oralis*
- *Staphylococcus aureus*
- *Streptococcus mitis*
- *Streptococcus pneumoniae*

Aerobic, Gram-Negative

- *Haemophilus influenzae*

CONTRAINDICATIONS

BESIVANCE® is contraindicated in patients with known hypersensitivity to this drug, to other quinolones, or to any ingredient in the formulation or component of the container.

Special Populations

Geriatrics (> 60 years of age): No overall differences in safety and effectiveness have been observed between elderly and younger patients.

Pediatrics (< 1 years of age): The safety and effectiveness of BESIVANCE® in infants less than 1 year of age have not been established.

Pregnant Women: BESIVANCE® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Women: Caution should be exercised when BESIVANCE® is administered to a nursing mother.



Safety Information

WARNINGS AND PRECAUTIONS

General

NOT FOR INJECTION INTO THE EYE. FOR TOPICAL OPHTHALMIC USE ONLY.

BESIVANCE® is a sterile suspension for topical ophthalmic use only, and should not be injected subconjunctivally, nor should it be introduced directly into the anterior chamber of the eye. There are no data to support use of BESIVANCE® in patients with concomitant corneal injury/damage.

Contact Lenses: Patients should be advised not to wear contact lenses if they have signs and symptoms of bacterial conjunctivitis or during the course of therapy with BESIVANCE®.

Growth of Resistant Organisms with Prolonged Use: As with other anti-infectives, prolonged use of BESIVANCE® may result in overgrowth of non-susceptible organisms, including fungi. If super-infection occurs, discontinue use and institute alternative therapy. Whenever clinical judgment dictates, the patient should be examined with the aid of magnification, such as slit-lamp biomicroscopy and, where appropriate, fluorescein staining.

Carcinogenesis and Mutagenesis

Long-term studies in animals to determine the carcinogenic potential of besifloxacin have not been performed.

ADVERSE REACTIONS

Adverse Drug Reaction Overview

In three safety and efficacy trials with 2377 patients enrolled, no serious adverse reactions related to BESIVANCE® were reported. The most frequently reported treatment-emergent ocular adverse events in the study eye were blurred vision (2.1%), eye pain (1.9%), and eye irritation (1.4%).

To report an adverse event, contact your Regional Adverse Reaction Monitoring Office at 1-866-234-2345 or Bausch + Lomb at 1-888-459-5000



Administration

Instill one drop in the affected eye(s) 3 times a day for 7 days. If a dose of this medication has been missed, it should be taken as soon as possible. However, if it is almost time for the next dose, the missed dose should be skipped and return to the regular dosing schedule. Do not double dose.

Patients should be advised to thoroughly wash hands prior to using BESIVANCE®. Patients should be advised to avoid contaminating the applicator tip with material from the eye, fingers or other source.

Patients should be instructed to invert closed bottle (upside down) and shake once before use. Remove cap with bottle still in the inverted position. Tilt head back, and with bottle inverted, gently squeeze bottle to instill one drop into the affected eye(s).

SUPPLEMENTAL PRODUCT INFORMATION

WARNINGS AND PRECAUTIONS

Immune

Anaphylaxis and Hypersensitivity:

Besifloxacin is only commercially available for topical ophthalmic administration. While anaphylaxis or other hypersensitivity reactions have not been observed with topical ophthalmic use of besifloxacin in humans, the potential for such reactions should be considered since patients with known hypersensitivity to fluoroquinolones were excluded from clinical trials.

In patients receiving systemically administered quinolones, serious and occasionally fatal hypersensitivity (anaphylactic) reactions have been reported, some following the first dose. Some reactions were accompanied by cardiovascular collapse, loss of consciousness, angioedema (including laryngeal, pharyngeal or facial edema), airway obstruction, dyspnea, urticaria, and itching. If any allergic reaction occurs, BESIVANCE® should be discontinued and appropriate therapy should be administered as clinically indicated.

Bacterial Conjunctivitis Trials

The rates of the most common treatment-emergent ocular adverse events irrespective of causality observed in eyes treated with BESIVANCE® during the three bacterial conjunctivitis clinical trials are displayed in Table 1.

Table 1 - Incidence (%) of Treatment-Emergent Adverse Events Irrespective of Causality that Occurred in ≥ 1% of Study Eyes/Patients Treated with BESIVANCE® or Vehicle in Bacterial Conjunctivitis Studies (Population: Safety1)

Adverse Events	Besifloxacin n=1187 (%)	Vehicle n= 614 (%)
Eye Disorders		
Vision Blurred	25 (2.1%)	24 (3.9%)
Eye Irritation	17 (1.4%)	18 (2.9%)
Eye Pain	22 (1.9%)	11 (1.8%)
Conjunctivitis	14 (1.2%)	15 (2.4%)
Eye Pruritus	13 (1.1%)	10 (1.6%)
Conjunctivitis Bacterial	7 (0.6%)	9 (1.5%)
Nervous System Disorders		
Headache	21 (1.8%)	11 (1.8%)

1 Safety population includes subjects treated for bacterial conjunctivitis that were randomized and received at least one dose of the study drug in the three safety and efficacy studies. BESIVANCE® was tested in all three studies, while the vehicle was tested in only two of the studies.

Less Common Clinical Trial Adverse Drug Reactions (<1%)

Treatment-related adverse events (possibly, probably or definitely related) reported in 0.1 to 1.0% of eyes receiving BESIVANCE® included:

Eye Disorders: eye pruritus, dry eye, conjunctivitis, conjunctivitis bacterial, punctate keratitis, conjunctival oedema, eye discharge, corneal infiltrates, corneal staining, eyelid margin crusting, keratoconjunctivitis sicca, foreign body sensation in eyes, conjunctival follicles, dry skin, eye disorder, instillation site pain, photophobia, visual disturbance.

Nervous System Disorders: headache

SYMPOTMS AND TREATMENT OF OVERDOSE

No information is available on overdosage of BESIVANCE®. A topical overdose of BESIVANCE® may be flushed from the eye(s) with warm tap water.

Full Product Monograph available for health professionals at: <http://www.bausch.ca>

BAUSCH + LOMB



Bausch & Lomb Canada Inc., Vaughan, Ontario L4K 4B4
®/™ denotes trademark of Bausch & Lomb Incorporated
© Bausch & Lomb Incorporated

A Paradigm Shift in Primary Open Angle Glaucoma

BY MARK ELTIS, OD, FAAO

Introduction

Glaucoma is a chronic irreversible neurodegenerative disease characterized by destructive changes in the optic nerve head and retinal nerve fibre layer due to loss of retinal ganglion cells and their axons.¹⁻¹⁴

For years glaucoma was considered to be a disease of ocular hypertension (greater than 21 mmHg).¹⁵⁻¹⁷ Over the last decade the disease has been redefined as an optic neuropathy with field loss resulting from IOP unacceptably high for the optic nerve head.^{6,8,18-21} Therefore, the simplistic cut-off point of 21 mmHg seems outdated and invalid.^{8,19,22}

Despite the prevalence of glaucoma, a universal definition of the disease is still absent.²³⁻²⁷

Glaucoma is a leading cause of blindness worldwide and the leading cause of vision loss in the United States.^{4,6,15,16,25} Approximately 2% of the U.S. population older than 40 have glaucoma and, with the aging of the population, the number of patients with the disease is expected to increase.^{2,15,16,25} Glucomatous neuropathy is generally an insidious disease with no symptoms until the advanced stages.^{2,14,28} There is a large body of evidence to suggest that an estimated 50% of those with glaucoma have not been diagnosed.^{6,28}

Primary open angle glaucoma (POAG), the most common glaucoma^{29,30} (representing 90-95% of

ABSTRACT

The definition, diagnosis and management of glaucoma have changed radically over the last decade. This paper reviews recent advances in primary open angle glaucoma and how they were applied to the case of a 68-year-old patient.

Key words: *pachymetry, neurodegenerative, prostaglandin analogue*

cases),² is defined by optic neuropathy in the absence of an identifiable secondary cause.^{1,2,4,6,21} POAG is a bilateral condition, but disease progression may be asymmetric.^{2,16} POAG can be divided into high tension glaucoma (HTG) and normal tension glaucoma (NTG).³⁰⁻³²

Although it is frequently accompanied by increased IOP, POAG can exist in patients with normal IOP.^{2,15,16} Up to 50% of patients with glaucoma never have an IOP above the statistical norm.^{22,31,33} Therefore, IOP alone is not reliable for glaucoma screening.^{30,33} However, IOP remains the only modifiable risk factor.^{12,22} Early detection is critical to prevent permanent structural damage and irreversible vision loss.^{2,3,28,34} Over the past decade, the diagnosis and management of glaucoma have changed dramatically.^{15,16} Numerous advances, such as the diagnostic role of pachymetry,^{35,36} imaging devices,³⁷⁻⁴¹ advanced perimeters⁴²⁻⁴⁴ and new therapeutic options,⁴⁵⁻⁴⁷ have revolutionized the diagnosis and management of the disease.³

Case Report

A 68-year-old female retiree of Hispanic descent presented to our office on July 8, 2009 for a routine exam. She was not satisfied with the quality of her old glasses and wanted to update her prescription. She was not taking any medication and had no history of health problems either in general or specifically of the eye. The patient had neither worn contact lenses nor had any corrective surgery. The patient had no known allergies and was not aware of health conditions in her family, was neither a smoker nor a drinker and did not engage in recreational drug use.

Presenting distance visual acuities through her glasses (+3.25 - 2.00 × 065 OD and +3.25 - 2.00 × 120 OS) were 20/40 OD, 20/20 OS and 20/20 OU. She was wearing progressive addition lenses, and her vision through her +2.25 reading addition was 20/30 OD, 20/20 OS and 20/20 OU (40 cm working distance).

Extraocular muscles were unrestricted in all gazes. Pupils were round and reactive to light and accommodation with negative Marcus Gunn. Confrontation visual fields were full to finger count in both eyes, and near point of convergence was 8 cm. Cover test was ortho in the distance and at near.

Subjective refraction was OD +2.25 - 2.00 × 061 20/25 and OS +3.25 - 2.00 × 120 20/20. Her new add was +2.50.

Slit lamp examination revealed normal lids and lashes. The conjunctivas of both eyes were clear. Both corneas had arcus senilis present but were otherwise clear and did not stain with fluorescein. Irises were brown, and transillumination was absent in both eyes. Both crystalline lenses had grade 1 cortical spoking and nuclear sclerosis. (There was no pseudoexfoliation in either eye.) Anterior chambers were without either cells or flare and were estimated by Van Herick grading to be grade 3 in both eyes. Intraocular pressure was 16 mmHg in both eyes at 3:30 p.m. using Perkins applanation tonometry. Dilated fundus examination was performed. Cup-to-disc ratios were 0.8/0.8 OD and 0.7/0.7 OS. The arterial-venous ratio was 2/3 in OU, and the retinal vessels appeared normal. Maculas were clear in both eyes although a foveal reflex was absent. There were neither holes, tears nor instances of retinal detachment in either eye. The patient was counselled that a glaucoma work-up would be in order. She said there was no family history of glaucoma, but she was asked to inquire further to be sure. The patient was asked to book a follow-up for a check of IOP, for gonioscopy and for another look at the fundi. The patient declined to be sent for visual field tests, pachymetry, OCT and HRT. She was also informed about the early cataracts and arcus. (The patient said she had just had her cholesterol checked and that it was normal.)

Follow-up #1

After initially declining all further testing and then taking an extended vacation overseas, the patient

returned for a follow-up on September 19, 2009. She stated that her brother was in fact being treated for glaucoma. Presenting distance visual acuities through her glasses (+3.25 - 2.00 × 065 OD and +3.25 - 2.00 × 120 OS) were 20/40 OD, 20/20 OS and 20/20 OU. Intraocular pressure was 16 mmHg in both eyes at 1:15 p.m. using Perkins applanation tonometry. Gonioscopy (Goldmann) was performed and did not reveal any pathology. Scleral spur was identified in all four quadrants of both eyes. Anterior chambers were without either cells or flare. Dilated fundus examination revealed that cup-to-disc ratios were 0.8/0.8 OD and 0.7/0.7 OS. There was no indication of either optic nerve drusen or nerve pallor in either eye. The arterial-venous ratio was 2/3 in OU, and the retinal vessels appeared normal. Maculas were clear in both eyes although a foveal reflex was absent. The patient was scheduled for pachymetry, OCT, HRT and a visual field test.

Follow-up #2

The patient rescheduled her follow-up several times but finally had testing done on January 6, 2010.

Visual field testing showed a large inferior arcuate defect OU which corresponded to the NFL superior defect on both the OCT and HRT. The visual field test was very reliable, and GHT was outside normal limits in both eyes. The signal strength of the OCT was a less than ideal 6 in each eye, probably due to media opacities. Central corneal thickness (CCT) was 510 µm OD and 515 µm OS. The patient was informed that she was a glaucoma suspect and was

scheduled for a consult with a glaucoma specialist.

Follow-up #3

The patient failed to appear for her follow-up but finally returned for a visual field at the glaucoma specialist's office on July 29, 2010, prior to the glaucoma consult. A repeatable arcuate defect was confirmed in each eye. At that visit the patient's corrected visual acuity was 20/40⁺ OD and 20/30 OS. Her IOP was 16 mmHg in each eye at 11 a.m. using Goldmann tonometry.

Follow-up #4

The patient was then seen by the glaucoma specialist on August 3, 2010. IOP at 12 p.m. was 16 mmHg OU. Gonioscopy was performed, and angles were found to be "open" in both eyes. Cup-to-disc ratios were 0.8/0.6 OD and 0.8/0.6 OS and a "superior rim notch" was noted OU. OCT was repeated and confirmed superior NFL damage in both eyes. An "arcuate defect" in each eye was identified based on the 24-2 Humphrey visual field test (performed July 29, 2010), which was consistent with the area of damage on OCT.

The following differential diagnosis was considered in this case:

- 1. Physiological cupping** has neither field loss, progression of cupping nor damage to NFL.^{18,31} Although cupping may be large, the neuroretinal rim is healthy.¹⁰
- 2. Congenital anomaly of the disc** is characterized by non-progressive visual field defects.^{10,31} Examples include tilted discs, colobomas and optic nerve pits.¹⁸

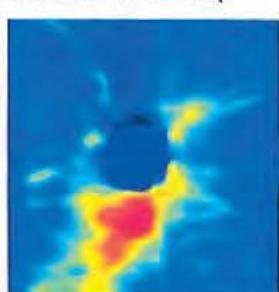
Name: OD OS
 ID: Exam Date: 8/3/2010 8/3/2010
 DOB: Exam Time: 9:18 AM 9:19 AM
 Gender: Female Technician:
 Doctor: Signal Strength: 8/10 6/10



RNFL Thickness Analysis: Optic Disc Cube 200x200

OD ● ● OS

RNFL Thickness Map



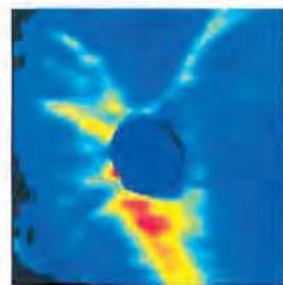
350
175
0 μm

Average Thickness

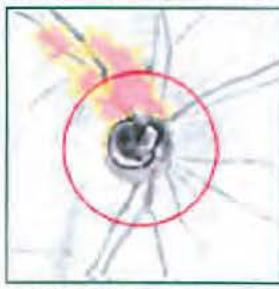
87 77
65 69
76 80 83
124 106
52 53 67
63 93 70
58 72 97
145 143 83

Quadrants

Clock Hours

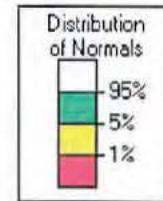
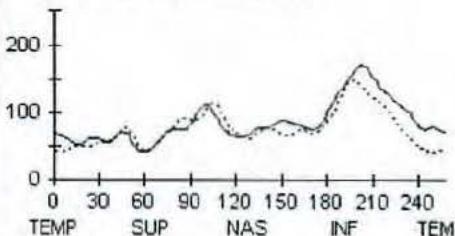


RNFL Thickness Deviation



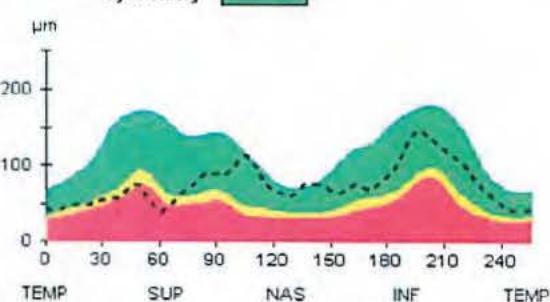
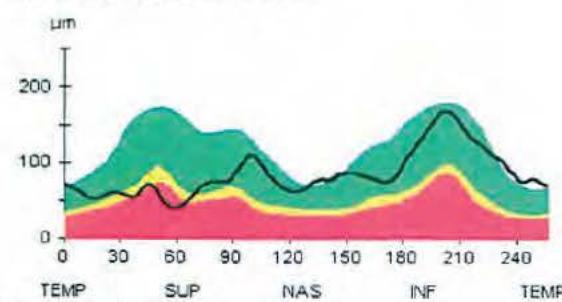
μm — OD ----- OS

Offset (-0.12; -0.03) mm



Offset (-0.06; -0.24) mm

RNFL TSNIT Normative Data



Symmetry

88%

Extracted RNFL Tomogram

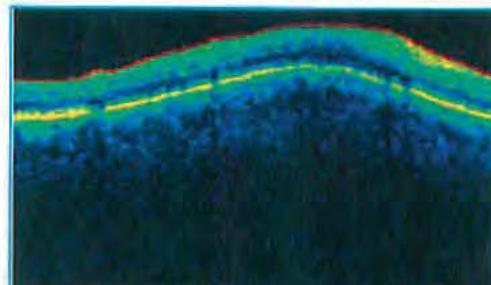
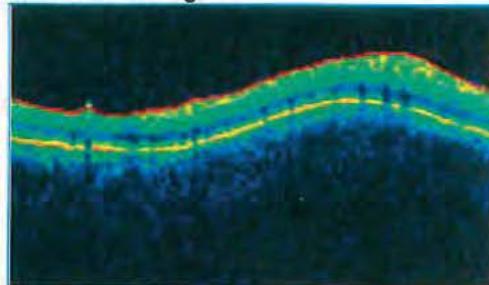


Figure 1 – OCT showing superior RNFL damage in both eyes. Exam date: August 3, 2010.

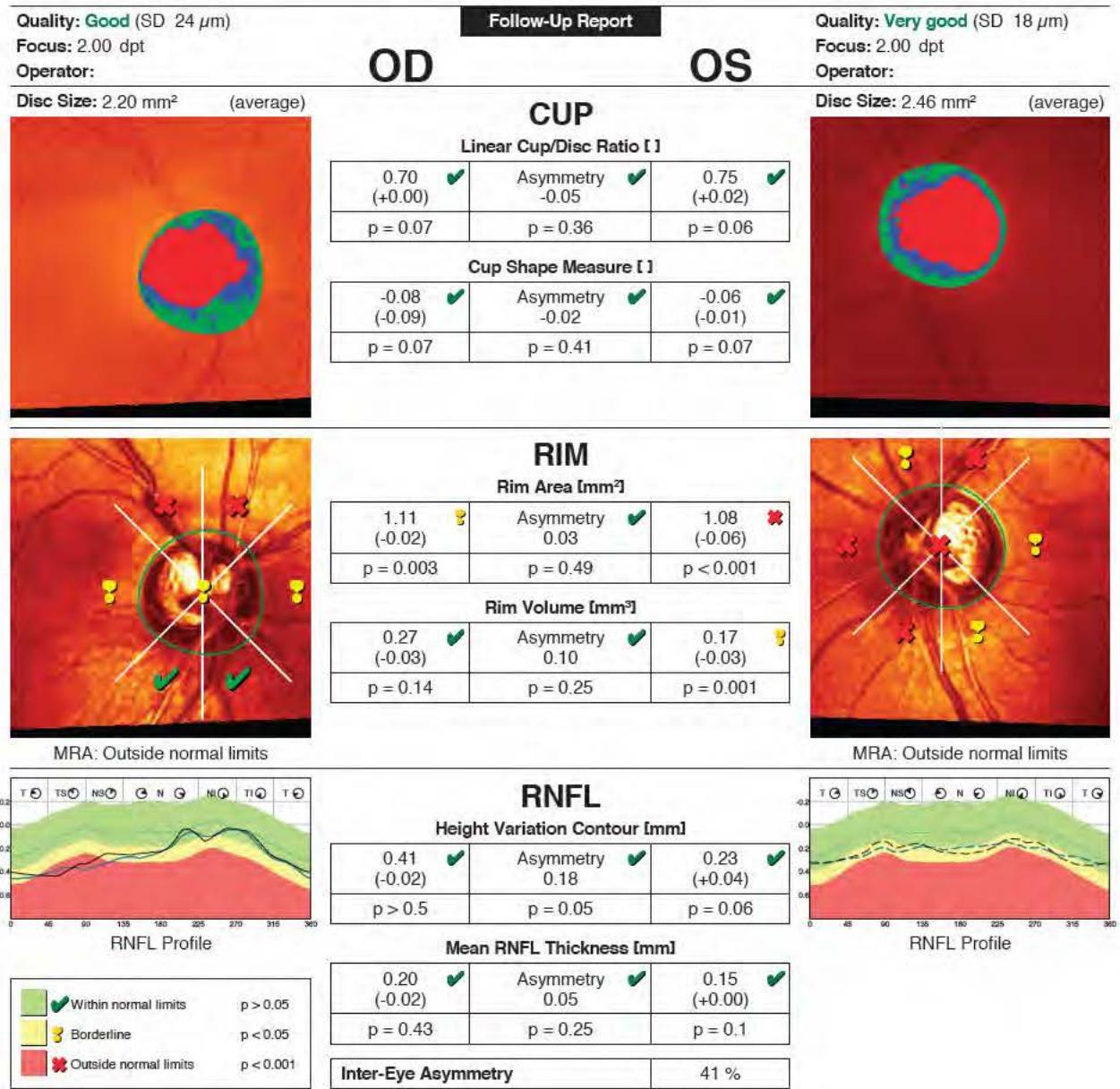


Figure 2 – HRT. Exam date: October 13, 2010.

- 3. Optic atrophy** is characterized by more optic nerve pallor than cupping.¹⁰ There may be a reduction in the small vessels on the disc surface (Kestenbaum sign).¹³ Central vision is generally decreased.¹⁸ The numerous causes of optic atrophy include tumours and

either vascular or degenerative disease.¹⁸ Visual field defects that respect the vertical midline are typical of intracranial lesions.¹³

- 4. Optic nerve drusen** are hyaline-like, may be either superficial or buried¹³ and are associated with retinitis pigmentosa.¹⁰ With

drusen, an absence of cupping may be visible.^{18,31} Lesions can be seen on B-scan¹⁸ and demonstrate autofluorescence with fluorescein filters through a fundus camera.¹⁰ Drusen may cause arcuate scotomas to develop.¹⁰ Several ONH drusen may be visible as

elevated chunky refractile nodules.¹⁰ Buried drusen obscure the physiological cup,^{18,31} and exposed drusen emerge during the teen years and are visible.¹³

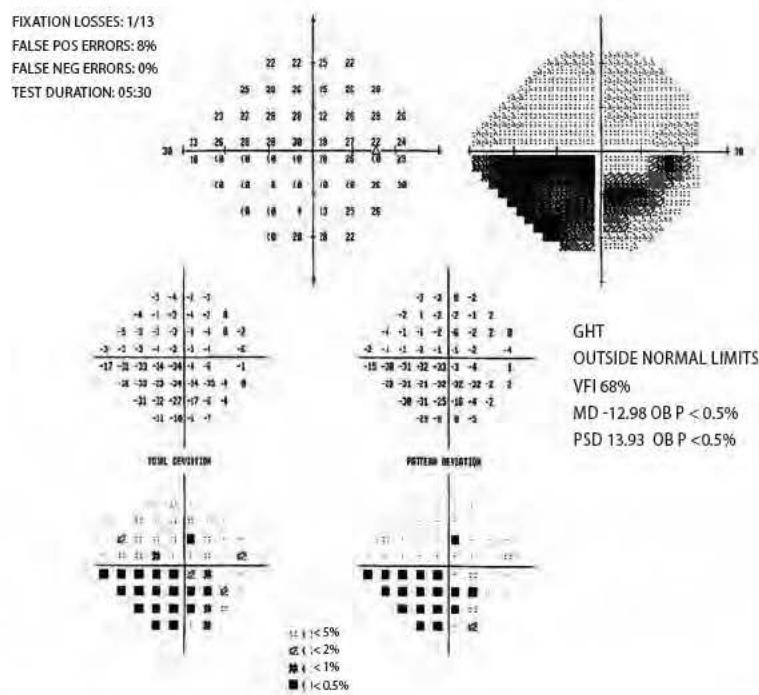
5. POAG (divided into NTG and HTG) is optic neuropathy with IOP too high for the optic nerve.³⁰⁻³² POAG by definition has an open anterior chamber (confirmed with gonioscopy) and no identifiable cause for the elevated IOP.¹⁶ Cupping, notching of the optic nerve along with corresponding visual field loss is present.¹³ POAG is generally bilateral but may be asymmetric.¹⁸ Visual field defects cross the vertical midline, and central scotomas are absent early in the disease.³¹

6. Secondary glaucoma is a root cause for the elevated IOP. Either optic nerve damage or field loss must be present. Causes include pigment dispersion syndrome, pseudoexfoliation of the lens, steroid use and trauma.¹⁶ Secondary glaucoma can be ruled out through case history and SLE examination with gonioscopy.^{10,16}

Our patient had nerve fibre damage with corresponding areas of field loss. The damage was bilateral and consistent with glaucomatous loss. Her central corneal thickness was thin in each eye. There was no history of steroid use or previous glaucoma episodes. No optic nerve drusen were seen, and the cupping was large rather than obliterated. Pallor of the optic nerves was also absent.

Neuro-ophthalmological examination did not seem indicated³¹ considering the bilateral cupping and the type of visual field loss

RIGHT EYE



LEFT EYE

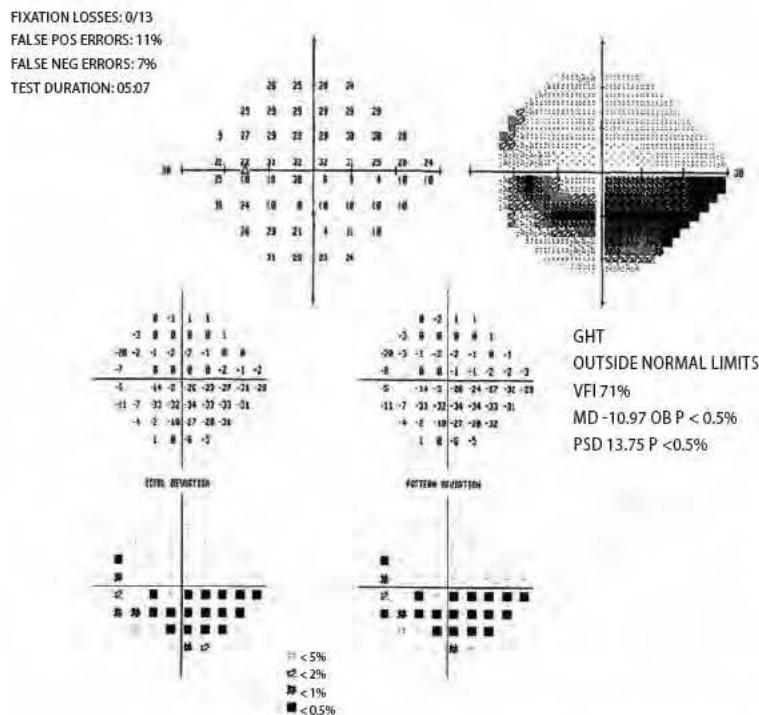


Figure 3 – 24-2 Humphrey visual field test. Exam date: October 13, 2010.

which crossed the vertical midline. Gonioscopy and slit lamp examination did not reveal a secondary cause for the suspected glaucoma. Therefore, the tentative diagnosis was primary open angle glaucoma likely of the NTG subgroup because the pressure reading was within the statistical norm. However, it is possible that, if multiple readings of IOP had been taken over a longer period and particularly at night, there may either have been times when the patient's IOP was elevated above 21 mmHg or have been large diurnal fluctuations.

The glaucoma specialist prescribed Travatan Z: 1 drop once per day OU for our patient. She was to return for a follow-up in two months.

Follow-Up #5

The patient repeated HRT and 24-2 visual fields on October 13, 2010 at the glaucoma specialist's office. Her presenting visual acuities were 20/30³ OD and 20/25² OS with glasses. IOP was 10 mmHg OD and 14 mmHg OS at 4:08 p.m. using Goldmann tonometry.

Follow-Up #6

The patient was subsequently seen in the glaucoma specialist's office on October 19, 2010 for a consult. The patient's IOP was noted as 12 mmHg in both eyes at 3 p.m. Pachymetry was 510 µm OD and 515 µm OS. "Adequate IOP control" was recorded in the assessment, and a follow-up appointment was scheduled for 6 months.

Follow-Up #7

The patient was scheduled for follow-up with the glaucoma specialist on April 20, 2011. She did not appear for her appointment and did not return either my office's phone calls or the specialist's despite numerous attempts.

Discussion

Epidemiology

Glaucoma affects 80 million people¹⁴ and is the second leading cause of blindness worldwide.^{21,48} The prevalence of glaucoma varies widely around the globe, with the lowest rate found among the First Nations of Alaska (0.006%) and the highest among Caribbean people of African ancestry (7.1-8.8%).⁴⁹

Two million people have glaucoma in the U.S.,² and the number is expected to rise significantly.⁴ Rates are higher among Latinos than among Caucasians, in comparison with whom African Americans are 3-4 times more at risk.¹⁶ POAG is the leading cause of blindness in African Americans.² Normal tension glaucoma rates (among those affected with glaucoma) appear highest in Japan.³⁰

Risk Factors

Glaucoma is a group of optic neuropathies with multiple risk factors,²¹ including increased cup-to-disc ratio, thin central corneal thickness, either African or Hispanic descent and the presence of an optic disc haemorrhage.^{2,13,15,36} Elevated IOP remains the most significant risk factor for glaucomatous progression.^{1,17} Optic disc haemorrhage is critical since it may precede visual field loss and further optic nerve damage.¹⁶

Positive family history is also an important risk actor.^{2,13,36,49} The familial nature of glaucoma has been recognized for decades with up to 50% of patients having a positive family history.⁴ The prevalence of glaucoma increases significantly in those 60 and older.²

The roles of diabetes^{15,16} and hypertension in glaucoma remain unclear.^{50,51} Controversially, some recent studies suggest that, in patients with either pre-existing diabetes or cardiovascular disease, the daily use of cholesterol drugs such as statins may reduce the risk of POAG development.²

The relationship between glaucoma and myopia is also still under investigation,^{2,16} with some evidence pointing to an increased risk of glaucoma among myopes.^{19,36} The LALES study suggested an independent relationship between increased axial length and glaucoma due to weaker scleral support at the optic nerve.¹⁶ There may be an association between NTG and Raynaud's phenomenon, migraines and hypotension.^{10,31,39} There are no environmental factors that are definitively associated with POAG.⁴⁹ IOP remains the only known risk factor that can be modified to decrease chances of disease progression.²

Genetic Factors

Clearly, there are genetic factors related to glaucoma.¹⁰ Less clear is the full extent of the relationship; although some genes have been identified, over 90% remain a mystery.⁴ To date, 22 genetic loci have been linked to POAG, and 3 genes have been identified for POAG from the reported loci.^{21,30} Mabuchi

et al recently concluded that SRBD1 gene polymorphism (a non-IOP-related genetic factor) is associated with both development of NTG and HTG POAG.³⁰ Genes and some environmental factors may affect the rate of ganglion cell apoptosis.⁴⁹

Which Changes Happen First: Structural Or Functional?

Structural change precedes functional change in some glaucomatous eyes but follows it in others.¹⁶ That some ganglion cells may begin to malfunction before dying results in decreased sensitivity without structural change.³ In other cases, where ganglion cell redundancy is high, retinal nerve fibre layer (RNFL) loss may precede functional damage.^{11,38,39,42} In the OHTS study, 60% of patients who converted to glaucoma had optic disc changes before field loss.^{23,35,36}

Both OHTS and EGPS showed that, in many patients, structural defects discovered with stereo photos are detectable before repeatable SAP (standard automated perimetry) functional defects.³

When SAP damage is detected first, it may be that the patient started out with more structure available to be lost and therefore that the patient continues to be within the norm for structure.³ Functional glaucoma deficits can occur before structural ones, but, more commonly, structural damage occurs before functional.^{5,11} The exact mechanism of damage is not yet fully understood.^{7,23,52}

Diagnosis and management of glaucoma are shifting from disease staging to evidence-based risk assessment of the patient.⁵¹ Risk factor calculators, such as the one that merged data from OHTS and

EGPS (<http://ohts.wustl.edu/risk>) to create an online risk calculator for clinicians, now help to predict a patient's individual risk of developing glaucoma in the next five years.⁵³

Because retinal ganglion cell dysfunction and death characterize glaucoma,^{20,54} detection is based on identification of either abnormalities or changes to either the ONH or the RNFL, either structural or functional.^{26,54} As there is no true gold standard for glaucoma diagnosis, progressive optic neuropathy has been suggested as a definition for use alongside functional testing.^{3,23}

A purely functional view of glaucoma may miss early glaucomatous patients.³ The disease may more comprehensively be defined according to both functional and structural criteria.^{26,27}

Neuroprotection in Glaucoma

Recent studies suggest the involvement of the immune system in glaucoma,^{12,20} but the neuroprotective and neurodestructive potencies of the immune system make its role unclear and sometimes contradictory.^{8,14} T-cell – mediated immune response which is initially neuroprotective but goes unchecked¹² may become autoimmune neurodegeneration.⁸

Damage to retinal ganglion cells could potentially be prevented by targeting the mechanism behind neuronal destruction.²⁰ Neuroprotective agents used in neurodegenerative diseases such as ALS (amyotrophic lateral sclerosis) and Alzheimer's disease are currently being evaluated for use in glaucoma.¹⁴

Several antibodies have been associated with glaucoma but no definitive specific antibody has been identified to screen for glaucoma.⁸

A recent study found that BDNF (brain-derived neurotrophic factor), a polypeptide growth-factor known to develop and to preserve neurons,²⁰ may be a useful biochemical marker for early detection of glaucoma.⁶ BDNF crosses the blood-brain barrier and is found in lower concentrations in the tears of those with glaucoma.⁶ This may also become a reliable, time-efficient and cost-effective method for the screening and management of the disease.⁶ Vaccinations which boost the immune system such as ones being used to enhance neuroprotection in multiple sclerosis may become a promising therapy for glaucoma.¹²

Ocular Hypertension and High Tension Glaucoma

Ocular hypertension (OH) is IOP elevated above the normal range.¹³ OH can exist without causing either damage to the nerve or vision loss but is a key risk factor for glaucoma.¹ If left untreated, roughly 1 in 10 patients with OH will develop glaucoma in 5 years.¹ The only proven strategy to prevent POAG progression is the use of ocular hypotensives among people with OH.^{16,36,49} The decreasing of IOP lowers the rate of conversion from OH to POAG and slows its progression.^{2,15,36,50}

In a glaucoma suspect, optic nerve deterioration (confirmed by either stereophoto, nerve fibre analysis or visual field glaucomatous changes) should be considered to indicate a conversion to POAG.⁵⁴

This is why the establishment of a baseline through photography, imaging and fields is critical.¹⁶

Normal Tension Glaucoma

Normal tension glaucoma (NTG) is a form of POAG^{10,13,18} where optic nerve damage and corresponding visual field defects occur^{29,30} despite apparently normal IOP.⁵⁵ With NTG, the etiological trigger – the pathogenic process – simply takes place at a lower IOP.^{31,33} Whenever IOP in the individual is high enough to initiate the disease process, the pathophysiologic steps are the same as in HTG.³¹ Chinese glaucoma specialists reached a consensus in 2008 that NTG is a subtype of POAG and the dividing line was IOP greater than 21 mmHg.³²

A neuro exam and MRI may be useful⁵⁶ to differentiate between glaucoma and other types of optic neuropathy.⁵⁵ However, neuro-ophthalmic evaluation with neuroimaging does not seem to be necessary for all cases of suspected NTG.³¹

NTG is fundamentally the same disease as POAG³¹, and the treatment is the same,³³ but some features may be more prevalent in NTG than HTG^{31,32} (splinter haemorrhages for example and CCT typically around 510 to 520 microns).¹⁸

The collaborative NTG study showed that lowering the IOP by 30% did change the course of the disease in a majority of patients.^{10,31,33}

It is believed that non-IOP-related factors are more important in NTG,³⁰ but recent findings may put that reasoning into question. The SRBD1 gene, which was considered to be a non-IOP-related genetic

factor, is implicated in both NTG and HTG.³⁰ That HTG and NTG run in the same families suggests they either are the same or are related conditions.³¹ A large proportion of glaucoma patients in Japan suffer from NTG.³⁷ (In a Japanese study in the city of Tajimi, 92% of POAG sufferers had NTG.)³³ There is no global consensus on the question of whether NTG and HTG are simply subtypes of POAG.^{32,56}

Diagnostic Techniques

Stereoscopic ONH photography is a simple low-cost three-dimensional image of the ONH.¹⁰ In practice, it is an accepted way of documenting structural damage in glaucoma suspects.¹⁵

An advantage of subjective assessment over quantitative analysis is the inclusion of some parameters of the ONH (pallor and haemorrhages) which contribute to a comprehensive evaluation and are not quantifiable.^{3,38} The ONH has a wide range of normal variations; qualitative variables have been shown to have higher specificity than quantitative parameters in separating glaucomatous eyes from normal.³

Assessment of the iridocorneal angle by gonioscopy is key to ruling out a secondary cause.^{2,15,16,57}

For decades, Goldmann tonometry has been the most accepted way of measuring IOP.¹⁹ Goldmann assumed average corneal thickness of 0.52 mm.¹⁹ That assumption may lead to either under- or overestimations of IOP with either excessively thin or excessively thick corneas.^{10,19,31}

Advances in Diagnostic Techniques – CCT

OHTS's discovery that lower than average CCT was a risk factor for glaucoma converted pachymetry into a routine procedure in glaucoma assessment.^{2,19,35,36,50}

In both OHTS and EGPS, CCT was the most important predictive factor for conversion from OH to glaucoma.^{35,36,47,58} CCT is no less important in glaucoma suspects with “normal” IOP. In those cases, factors unrelated to pressure are important determinants of progression.¹⁹

CCT as a risk factor is independent of IOP and possibly based on biomechanical characteristics of the eye.⁴⁷ CCT is not only important because of the artefact of Goldmann's assumption^{10,31} but because patients with thinner corneas also have thinner nerve fibre layers.⁴⁷

Conversion to the “true” pressure based on pachymetry is no longer advocated because there is no consensus on the conversion formula.^{16,47} (Conversion formulae are widely believed to be oversimplifications of a complex non-linear multifactorial relationship.)^{19,50} Instead of converting, we now classify: either thick, average or thin.⁵⁰ Average CCT is approximately 545 µm.^{16,47} African Americans generally have thinner corneas,^{19,50} Caucasians' corneas tend to be of average thickness, and Asians' corneas tend to be thicker.^{36,47} The OHTS study highlighted that, after adjustments were made for larger baseline cup-to-disk ratios, central corneal thickness and not race was a statistically significant predictor.³⁶

Recent studies have found that there is considerable variation in the pachymetry readings measured by trained observers.^{35,47} Therefore, pachymetry should be repeated twice, and three times if the first two readings are different.^{35,47} CCT also changes over the course of the day and over the course of a lifetime;³⁵ the cornea is thickest in the morning due to corneal oedema and also thins with age.³⁵

Imaging Technology and Retinal Nerve Fibre Analysis

A new generation of objective (quantitative) imaging technologies have been developed to measure RNFL that go beyond photography, which generally requires subjective interpretation.^{11,37,39,54} To help the clinician in the evaluation of visual function and structure, computer-based imaging devices such as scanning laser ophthalmoscopy (HRT), scanning laser polarimetry (GDx) and optical coherence tomography (OCT) provide quantitative assessment of structural damage to the optic disk and retinal nerve fibre layer.^{3,7,9,10,11,15,16,37,41,59,60}

HRT (Heidelberg retina tomograph) is the commercially available form of confocal scanning laser ophthalmoscopy.^{13,38} HRT uses a laser to scan the retina at multiple focal planes and creates a stack of 64 coronal planes.³⁸ This provides the examiner with optic disc topography,³⁸ a three-dimensional composite image of the ONH and posterior segment.^{3,9,38,59} HRT can superimpose baseline and follow-up images for automated detection of ONH changes, and existing machines may easily be upgraded with newer software.³⁹ The

new generation has a higher scan rate and resolution⁵⁹ and eliminates a subjective component by eliminating the need for a contour line drawn by the examiner.^{3,38,59} Scanning laser polarimetry (GDx) uses polarized light to measure the retinal nerve fibre layer birefringence in order to estimate tissue thickness.^{7,54,59} Media opacities decreases its reliability but the newer versions can compensate for this more effectively.³ OCT employs the principles of low-coherence interferometry and is analogous to ultrasound B-mode imaging but using light – not sound – to acquire high-resolution images of ocular structures.^{7,11,54,59} The main limitations of time-domain OCT are low resolution and slow scan rate.⁴⁰ The newest OCT technology – spectral domain⁴⁰ – collects all backscattered light frequencies simultaneously.⁵⁴ That results in a faster scan rate⁴⁰ and better resolution,⁴⁰ with each frequency of light representing a tissue depth.⁵⁴ A new parameter was also introduced for the OCT: signal strength (seven or greater is needed for good quality).⁵⁹ Ocular opacities can decrease the reliability of OCT as well.³

Multiple studies have found structural imaging technology to be at least as good as stereo photos in their ability to help clinicians differentiate between glaucomatous and normal eyes.^{3,37,38,59,60} Studies have not demonstrated the clear superiority of one imaging technique over another.^{3,41}

It is important to note that the definition of “normal” varies with each technology (HRT, OCT, GDx).⁵⁹ In one study, both GDx VCC and stratus OCT had similar correlations at each clock-hour

segment, and both were useful in early detection for patients with preperimetric glaucoma.⁷

In another study, the diagnostic imaging techniques outperformed subjective assessment of the optic nerve by general ophthalmologists (but not by glaucoma specialists, whose expertise outperformed objective techniques).⁵⁹ All scans had better sensitivity and specificity than general ophthalmologists’ subjective photo assessment of ONH.⁵⁹

In several studies, HRT was as effective as stereophotos in estimating risk of developing POAG in ocular hypertensive subjects.^{3,38,60} Therefore, HRT can be used to successfully differentiate ocular hypertensives, normals and eyes with glaucoma.^{3,9,38,60}

The use of scanning technology in concert with subjective assessment by either a general ophthalmologist or a glaucoma expert improved identification of glaucoma patients.⁵⁹ This suggests that, particularly for general ophthalmologists, measurement of RNFL provides an important degree of additive information when combined with subjective assessment.^{41,54,59} Scanning technology should not replace observation of the optic nerve (or perimetry) but complement it.^{11,39,54,59}

Visual Field Testing

Despite advances in glaucoma diagnosis, visual field (VF) testing is still important.^{3,5,34,43,44} It is critical to include measurements both of structure and of function in the evaluation of glaucoma although they may not correspond in the early stages of the disease.^{3,26,34} White-on-white static automated perimetry is still the

most commonly used method to clinically diagnose visual field loss (and monitor progression).^{5,16,37,39,42} It is now suggested that, due to the high variability of results, VF testing be repeated three times to confirm area of loss.³ In OHTS, the majority of VF abnormalities initially detected were not repeated on follow-up testing.³ SITA-SAP (Swedish interactive threshold algorithm – standard automated perimetry) has become the standard for clinical use with the Humphrey visual field analyzer.^{3,34} SITA is a testing strategy that has decreased testing time to roughly five minutes with no loss of accuracy.^{3,42} GHT (glaucoma hemifield test) analyzes 24-2 and 30-2 visual fields for patterns typically found in glaucoma (such as vertical asymmetry).¹³

Newer technologies like FDT (frequency doubling technology) and SWAP (short wavelength automated perimetry) may be helpful for early detection of disease when SAP is normal but glaucoma is nevertheless suspected.^{37,42,43}

FDT perimetry seems to have promise as an effective and efficient method of detecting visual field loss.^{7,16,43,44} It is portable, easier to use and faster than SAP.⁴² FDT loses reliability with cataracts but has lower test-retest variability compared to SAP and SWAP.^{3,37} SWAP is selective and highly sensitive to early damage (up to 5 years earlier than SAP⁴²).^{3,16} However, like FDT, it is vulnerable to media opacities.³

Studies suggested that a combination of field tests may increase sensitivity to early damage without a drop in specificity.⁴² Results of tests can be repeated either within a test or across tests to look for evidence

of damage to the same area;^{3,43} SITA-SAP may be combined with SWAP, for example.

Advances in Therapeutic Options

Medical treatment with ocular hypotensives is the mainstay of therapy, particularly in the early stages of the disease.^{1,48} Beta-blockers, CAIs and alpha-2 adrenergic agonists decrease formation of aqueous fluid while prostaglandin analogues improve outflow through the uveoscleral pathway.^{2,45,47}

Prostaglandin analogues (PGAs), the safest and most effective drugs to date,^{1,47,61} have become the preferred first-line agents for glaucoma management.^{10,46,47} Once daily dosing optimizes convenience and increases compliance.⁶² Patients respond differently to each prostaglandin, none of which is universally better than another.⁴⁷

Hyperaemia, which is the most common adverse effect of PGAs (and which has been found to decrease compliance) occurs less frequently in latanoprost.⁶² Timolol (beta-blocker) monotherapy produces far less hyperaemia but is less effective than latanoprost and bimatoprost.¹ Out of the PGAs, latanoprost (which is associated with less hyperaemia) may achieve the best balance of IOP efficacy and tolerability.^{1,47} There is no clear choice of adjunctive therapy with prostaglandins. Other classes are relatively ineffectual with them.⁶¹

Contrary to the widely held belief, IOP is actually highest at night.^{33,47} This is likely due to elevated episcleral venous pressure which causes a backup in the drainage system.⁴⁷ Prostaglandins are better at prevent-

ing nocturnal spikes (due to mechanism of action) while beta-blockers probably have poor IOP control at night.⁴⁷

Studies have shown Travatan's and Travatan Z's duration of action exceeds their 24-hour dosing schedule (up to 84 hours).⁴⁶ The incidence of IOP varying with missed doses, which has been associated with glaucoma progression, thus decreases.^{16,46,48}

Travatan Z is the only PGA that contains SofZia and not BAK as a preservative.^{46,47} BAK reduces TBUT and decreases epithelial cell integrity, and that causes dry eye and ocular surface inflammation.^{46,47} One study found ocular surface disease is a problem in almost half of all glaucoma patients. The reduction of BAK may be important in glaucoma management.^{46,47} The chronic subclinical inflammation can cause surgical failure in those who have filtration surgery.^{46,47} BAK-free Travatan was found to be at least as effective as original Travatan but better for ocular surface disease.⁴⁶

Management

The goal of glaucoma management is to protect the patient's vision and quality of life.³⁴

IOP reduction should be at least 20 to 30% lower than where damage occurred.^{1,16,22} The greater the initial IOP, the greater the target reduction.¹ Achieving these objectives may require several medications, particularly when reduction over 20% is required.⁵⁰

Determining the effectiveness of IOP-lowering drugs is complicated by a diurnal fluctuation of up to 5 mmHg in normal populations with no evidence of pathology.¹ Greater

IOP fluctuations means greater risk of progression.^{16,34} The more the IOP is lowered, the more likely that the glaucoma progression will be stopped.¹⁶

When pharmacological therapy either fails or is not an option, various surgical procedures are available either to increase outflow or decrease inflow.^{10,63} Laser trabeculoplasty (incision-free) stimulates the trabecular meshwork into functioning more efficiently.^{2,13,16} Laser trabeculoplasty was found to be at least as efficacious as initial treatment with topical medication in the Glaucoma Laser Trial.⁶³

Trabeculectomy is used when non-incisional techniques fail.^{2,16} Drainage implants, which are alternatives to incisional techniques, shunt aqueous humor to the subconjunctival space (usually reserved for complicated cases when trabeculectomy fails).^{2,16} Filtration surgery with cataract removal is not as effective as filtration surgery alone. Patients who need both may want to delay cataract removal.¹⁶

Adherence

Adherence to medications is a serious problem,^{15,16} with rates of non-compliance with glaucoma meds sometimes soaring to 80% of cases in the literature.^{1,48} Lack of consistency in dosing can lead to either sustained increases or fluctuations in levels of IOP.¹⁶ In either case, the risk of glaucoma progression is increased.^{16,46} Non-compliance may occur for many reasons. The cost of medication may be prohibitive for the patient; the patient may not be able to properly administer dosage.^{16,48} Side effects may dissuade a patient, and a patient may simply fail to understand the importance

of treatment and quit.^{16,48} The most common reason cited is forgetfulness.⁴⁸ Up to 50% of patients discontinue chronic medications during the first five months of therapy despite its importance in stopping the progression of glaucoma.⁶²

The GAPS study showed that 37% of patients experiencing hyperaemia with PGA will discontinue treatment.¹ Uninterrupted use of glaucoma medication is relatively rare but is better with latanoprost than with other PGAs.⁶²

Electronic monitoring is currently the most objective method to track patient adherence.⁴⁸

The Future of POAG Diagnosis and Management

A broadly accepted definition of POAG^{24,26,27} in addition to a universal and cost-effective screening protocol is clearly needed to improve diagnosis and treatment.⁶ Detailed studies on the time-frame from disease onset to significant vision loss and blindness need further investigation.⁵¹

Most definitions of glaucoma are based on visual field loss but recently a definition incorporating progressive optic disk change has been suggested.^{3,27}

Better hardware and software are needed to improve the diagnostic value of imaging technologies¹¹ and automated perimetry.⁴³ Future imaging technology includes polarization-sensitive OCT, which will allow more precise measurement of the RNFL.^{16,54} The next generation of OCT, swept source (SS-OCT), will be even faster than spectral domain and will minimize motion artefacts.⁵⁴ The high cost of imaging technology promotes the economic feasibility of general

ophthalmologists receiving ongoing training in optic disc glaucomatous signs, particularly in developing countries.⁵⁹ Current tonometers supply merely a snapshot, but future models may be able to deliver, via implant in the eye, 24 hours of data (as with a Holter monitor).²² A new front in the fight against glaucomatous damage may be opened by drugs working through an alternative to IOP-reduction.^{33,61} In particular, drugs which offer neuroprotection (which shield the optic nerve from damage) would be revolutionary.^{12,14,20,61}

Communication between optometrists and ophthalmologists needs to be improved; research shows that both the letters of referral to specialists and the responses to ODs leave much to be desired.²⁸ A recent study showed that alarms, both audio and visual, boast improved compliance and a decreased proportion of missed doses.⁴⁸ Activities consistent with overall health (such as exercise, a diet high in fruits and vegetables and the avoidance of smoking) may be suggested despite the present lack of conclusive evidence.⁴⁹ Lifestyle and environment are factors requiring further study. They may modify IOP-risk and decrease the economic and visual consequences of POAG.^{49,51}

Conclusion

While much progress has occurred in the diagnosis and management of POAG, the exact pathogenesis and a cure remain elusive. This case highlights the importance of looking beyond IOP and emphasizes the application of modern technology and therapy in the management of this disease.

References

1. Orme M, Collins S, Dakin H, Kelly S, Loftus J. Mixed treatment comparison and meta-regression of the efficacy and safety of prostaglandin analogues and comparators for primary open-angle glaucoma and ocular hypertension. *Curr Med Res Opin.* 2010 Mar;26(3):511-28.
2. Hazin R, Hendrick AM, Kahook MY. Primary open-angle glaucoma: diagnostic approaches and management. *J Natl Med Assoc.* 2009 Jan;101(1):46-50.
3. Sharma P, Sample PA, Zangwill LM, Schuman JS. Diagnostic tools for glaucoma detection and management. *Surv Ophthalmol.* 2008 Nov;53 Suppl:S17-32.
4. Allingham RR, Liu Y, Rhee DJ. The genetics of primary open-angle glaucoma: a review. *Exp Eye Res.* 2009 Apr;88(4):837-44.
5. Carreras FJ, Rica R, Delgado AV. Modeling the patterns of visual field loss in glaucoma. *Optom Vis Sci.* 2011 Jan;88(1):63-79.
6. Ghaffariyeh A, Honarpisheh N, Heidari MH, Puyan S, Abasov F. Brain-derived neurotrophic factor as a biomarker in primary open-angle glaucoma. *Optom Vis Sci.* 2011 Jan;88(1):80-5.
7. Kim HG, Heo H, Park SW. Comparison of scanning laser polarimetry and optical coherence tomography in preperimetric glaucoma. *Optom Vis Sci.* 2011 Jan;88(1):124-9.
8. Lee KJ, Jeong SM, Hoehn BD, Hong YJ, Lee SH. Valosin-containing protein is a novel autoantigen in patients with glaucoma. *Optom Vis Sci.* 2011 Jan;88(1):164-72.
9. Balasubramanian M, Bowd C, Weinreb RN, Zangwill LM. Agreement between the Heidelberg Retina Tomograph (HRT) stereometric parameters estimated using HRT-I and HRT-II. *Optom Vis Sci.* 2011 Jan;88(1):140-9.
10. Friedman NJ, Kaiser PK, Pineda R. The Massachusetts eye and ear infirmary illustrated manual of ophthalmology. 3rd Ed. Philadelphia: Elsevier; 2009:483-523.
11. Yoo YC, Park KH. Comparison of optical coherence tomography and scanning laser polarimetry for detection of localized retinal nerve fiber layer defects. *J Glaucoma.* 2010 Apr-May;19(4):229-36.
12. Cheung W, Guo L, Cordeiro MF. Neuroprotection in glaucoma: drug-based approaches. *Optom Vis Sci.* 2008 Jun;85(6):406-16.
13. Kanski JJ. Clinical ophthalmology: a systemic approach 7th Ed. Oxford: Elsevier; 2011: 311-399, 785-811.
14. Baltmr A, Duggan J, Nizari S, Salt TE, Cordeiro MF. Neuroprotection in glaucoma - Is there a future role? *Exp Eye Res.* 2010 Nov;91(5):554-66.
15. American Academy of Ophthalmology Glaucoma Panel. Preferred Practice Pattern® Guidelines. Primary Open-Angle Glaucoma Suspect. San Francisco, CA: American Academy of Ophthalmology; 2010. Available at: www.aao.org/ppp.
16. American Academy of Ophthalmology Glaucoma Panel. Preferred Practice Pattern® Guidelines. Primary Open-Angle Glaucoma. San Francisco, CA: American Academy of Ophthalmology; 2010. Available at: www.aao.org/ppp.
17. Kniestedt C, Punjabi O, Lin S, Stamper RL. Tonometry through the ages. *Surv Ophthalmol.* 2008 Nov-Dec;53(6):568-91.
18. Ehlers JP, Shah CP. The Wills Eye Manual: office and emergency room diagnosis and treatment of eye disease. 5th Ed. Baltimore: Lippincott Williams & Wilkins, 2008: 191-225.
19. Lester M, Mete M, Figus M, Frezzotti P. Incorporating corneal pachymetry into the management of glaucoma. *J Cataract Refract Surg.* 2009 Sep;35(9):1623-8.
20. Danesh-Meyer HV. Neuroprotection in glaucoma: recent and future directions. *Curr Opin Ophthalmol.* 2011 Mar;22(2):78-86.
21. Jia LY, Tam PO, Chiang SW, Ding N, Chen LJ, Yam GH, Pang CP, Wang NL. Multiple gene polymorphisms analysis revealed a different profile of genetic polymorphisms of primary open-angle glaucoma in northern Chinese. *Mol Vis.* 2009;15:89-98.
22. Stamper RL. A history of intraocular pressure and its measurement. *Optom Vis Sci.* 2011 Jan;88(1):16-28.
23. Denniss J, Echendu D, Henson DB, Artes PH. Discuss: investigating subjective judgment of optic disc damage. *Optom Vis Sci.* 2011 Jan;88(1):93-101.
24. Miglior S, Guareschi M, Romanazzi F, Albe E, Torri V, Orzalesi N. the impact of definition of primary open-angle glaucoma on the cross-sectional assessment of diagnostic validity of Heidelberg retinal tomography. *Am J Ophthalmol.* 2005 May;139(5):878-87.
25. Swanson MW. The 97.5th and 99.5th percentile of vertical cup disc ratio in the United States. *Optom Vis Sci.* 2011 Jan;88(1):86-92.
26. Boland MV, Quigley HA. Evaluation of a combined index of optic nerve structure and function for glaucoma diagnosis. *BMC Ophthalmol.* 2011 Feb 11;11:6.
27. Harwerth RS, Wheat JL, Fredette MJ, Anderson DR. Linking structure and function in glaucoma. *Prog Retin Eye Res.* 2010 Jul;29(4):249-71.
28. Lockwood AJ, Kirwan JF, Ashleigh Z. Optometrists referrals for glaucoma assessment: a prospective survey of clinical data and outcomes. *Eye (Lond).* 2010 Sep;24(9):1515-9.
29. Asrani S, Samuels B, Thakur M, Santiago C, Kuchibhatla M. Clinical Profiles of Primary Open Angle Glaucoma versus Normal Tension Glaucoma Patients: A Pilot Study. *Curr Eye Res.* 2011 May;36(5):429-35.
30. Mabuchi F, Sakurada Y, Kashiwagi K, Yamagata Z, Iijima H, Tsukahara S. Association between SRBD1 and ELOVL5 gene polymorphisms and primary open angle glaucoma. *Invest Ophthalmol Vis Sci.* 2011 Jun 28;52(7):4626-9.
31. Anderson DR. Normal-tension glaucoma (Low-tension glaucoma). *Indian J Ophthalmol.* 2011 Jan;59 Suppl:S97-101.
32. Zhang L, Zhang YQ, Xu L, Yang H, Wu XS. [Is normal-tension glaucoma different from primary open-angle glaucoma]. *Zhonghua Yan Ke Za Zhi.* 2011 Feb;47(2):105-108.
33. Shields MB. Normal-tension glaucoma: is it different from primary open-angle

- glaucoma? *Curr Opin Ophthalmol.* 2008 Mar;19(2):85-8.
34. Gardiner SK, Demirel S, Johnson CA. Perimetric indices as predictors of future glaucomatous functional change. *Optom Vis Sci.* 2011 Jan;88(1):56-62.
35. Brandt JD, Gordon MO, Beiser JA, Lin SC, Alexander MY, Kass MA; Ocular Hypertension Treatment Study Group. Changes in central corneal thickness over time: the ocular hypertension treatment study. *Ophthalmology.* 2008 Sep;115(9):1550-1556.
36. Gordon MO, Beiser JA, Brandt JD, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP, Parrish RK 2nd, Wilson MR, Kass MA. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. *Arch Ophthalmol.* 2002 Jun;120(6):714-20; discussion 829-30.
37. Nomoto H, Matsumoto C, Takada S, Hashimoto S, Arimura E, Okuyama S, Shimomura Y. Detectability of glaucomatous changes using SAP, FDT, flicker perimetry, and OCT. *J Glaucoma.* 2009 Feb;18(2):165-71.
38. Alencar LM, Bowd C, Weinreb RN, Zangwill LM, Sample PA, Medeiros FA. Comparison of HRT-3 glaucoma probability score and subjective stereophotograph assessment for prediction of progression in glaucoma. *Invest Ophthalmol Vis Sci.* 2008 May;49(5):1898-906.
39. Alencar LM, Zangwill LM, Weinreb RN, Bowd C, Vizzeri G, Sample PA, Susanna R Jr, Medeiros FA. Agreement for detecting glaucoma progression with the GDx guidedprogression analysis, automated perimetry, and optic disc photography. *Ophthalmology.* 2010 Mar;117(3):462-70.
40. Rao HL, Zangwill LM, Weinreb RN, Sample PA, Alencar LM, Medeiros FA. Comparison of different spectral domain optical coherence tomography scanning areas for glaucoma diagnosis. *Ophthalmology.* 2010 Sep;117(9):1692-9.
41. Pueyo V, Polo V, Larrosa JM, Ferreras A, Alias E, Honrubia FM. Ability of different optical imaging devices to discriminate between healthy and glaucomatous eyes. *Ann Ophthalmol (Skokie).* 2009 Summer;41(2):102-8.
42. Alencar LM, Medeiros FA. The role of standard automated perimetry and newer functional methods for glaucoma diagnosis and follow-up. *Indian J Ophthalmol.* 2011 Jan;59 Suppl:S53-8.
43. Fogagnolo P, Rossetti L, Ranno S, Ferreras A, Orzalesi N. Short-wavelength automated perimetry and frequency-doubling technology perimetry in glaucoma. *Prog Brain Res.* 2008;173:101-24.
44. Johnson CA, Wall M, Thompson HS. A history of perimetry and visual field testing. *Optom Vis Sci.* 2011 Jan;88(1):8-15.
45. Susanna R Jr, Medeiros FA. The pros and cons of different prostanooids in the medical management of glaucoma. *Curr Opin Ophthalmol.* 2001 Apr;12(2):149-56.
46. Gross RL, Peace JH, Smith SE, Walters TR, Dubiner HB, Weiss MJ, Ochsner KI. Duration of IOP reduction with travoprost BAK-free solution. *J Glaucoma.* 2008 Apr-May;17(3):217-22.
47. Sowka J. The four Ps of glaucoma. *Rev Optom* 2010 Nov 15:66-71.
48. Ho LY, Camejo L, Kahook MY, Noecker R. Effect of audible and visual reminders on adherence in glaucoma patients using a commercially available dosing aid. *Clin Ophthalmol.* 2008 Dec;2(4):769-72.
49. Pasquale LR, Kang JH. Lifestyle, nutrition, and glaucoma. *J Glaucoma.* 2009 Aug;18(6):423-8.
50. Rhee DJ. Preventing glaucoma in a high-risk population: impact and observations of the Ocular Hypertension Treatment Study. *Arch Ophthalmol.* 2009 Feb;127(2):216-8.
51. Weinreb RN, Medeiros F. Risk assessment for glaucoma. *Open Ophthalmol J.* 2009 Sep 17;3:30-1.
52. Shafiq A, Swanson WH, Dul MW. Structure and function in patients with glaucomatous defects near fixation. *Optom Vis Sci.* 2011 Jan;88(1):130-9.
53. Ocular Hypertension Treatment Study Group; European Glaucoma Prevention Study Group, Gordon MO, Torri V, Miglior S, Beiser JA, Floriani I, Miller JP, Gao F, Adamsons I, Poli D, D'Agostino RB, Kass MA. Validated prediction model for the development of primary open-angle glaucoma in individuals with ocular hypertension. *Ophthalmology.* 2007 Jan;114(1):10-9.
54. Townsend KA, Wollstein G, Schuman JS. Imaging of the retinal nerve fibre layer for glaucoma. *Br J Ophthalmol.* 2009 Feb;93(2):139-43.
55. Dumitrica DM, Stefan C. [Normotensive glaucoma]. *Oftalmologia.* 2008;52(1):31-5.
56. Potop V, Dumitraci M, Ciocalteu A. [Normal tension glaucoma]. *Oftalmologia.* 2010;54(2):11-4.
57. Alward WL. A history of gonioscopy. *Optom Vis Sci.* 2011 Jan;88(1):29-35.
58. Elsheikh A, Alhasso D, Gunvant P, Garway-Heath D. Multiparameter correction equation for Goldmann applanation tonometry. *Optom Vis Sci.* 2011 Jan;88(1):102-12.
59. Vessani RM, Moritz R, Batis L, Zagui RB, Bernardoni S, Susanna R. Comparison of quantitative imaging devices and subjective optic nerve head assessment by general ophthalmologists to differentiate normal from glaucomatous eyes. *J Glaucoma.* 2009 Mar;18(3):253-61.
60. Weinreb RN, Zangwill LM, Jain S, Becerra LM, Dirkes K, Piltz-Seymour JR, Cioffi GA, Trick GL, Coleman AL, Brandt JD, Liebmann JM, Gordon MO, Kass MA; OHITS CSLO Ancillary Study Group. Predicting the onset of glaucoma: the confocal scanning laser ophthalmoscopy ancillary study to the Ocular Hypertension Treatment Study. *Ophthalmology.* 2010 Sep;117(9):1674-83.
61. Realini T. A history of glaucoma pharmacology. *Optom Vis Sci.* 2011 Jan;88(1):36-8.
62. Hahn SR, Kotak S, Tan J, Kim E. Physicians' treatment decisions, patient persistence, and interruptions in the continuous use of prostaglandin therapy in glaucoma. *Curr Med Res Opin.* 2010 Apr;26(4):957-63.
63. Razeghinejad MR, Spaeth GL. A history of the surgical management of glaucoma. *Optom Vis Sci.* 2011 Jan;88(1):39-47.



NEW KR-1

The Next Generation of Refractive Care

AUTO KERATO-REFRACTOMETRY
at a touch with 360° freedom



- Easy to use colour touch screen
- 360 degree rotatable monitor
- Fully automated
- Speedy, accurate, and repeatable refraction and keratometry measurements
- Ergonomic and comfortable design for operator and patients
- Flexible layout and space saving design

TOPCON CANADA INC.



Exclusive Canadian distributor for:
Topcon, Amtek, Welch Allyn, Paradigm (Dicon), Gulden,
M&S Technologies, Tinsley (Selected Products), Icare

www.topcon.ca

e-mail: info@topcon.ca

Eastern Canada • 1-800-361-3515

Ontario • 1-800-387-6768

Western Canada • 1-800-661-8349

CONNECTING VISIONS

An atypical case of HLA-B27-associated uveitis with hypopyon and posterior segment involvement

BY THOMAS XIE, OD & ETTY BITTON, OD, MSc, FAAO

ABSTRACT

The presence of a hypopyon and posterior segment involvement are uncommon clinical findings in HLA-B27-associated uveitis. Furthermore, first-time attacks rarely occur in the elderly. This report highlights an atypical uveitis case involving an older patient, an evident hypopyon and severe intermediate uveitis in one eye.

The 60-year-old Caucasian male was admitted for a painful, red eye with a sudden decreased vision to hand motion, in the affected eye. Ocular and systemic history were unremarkable. An anterior chamber examination of the eye revealed extensive cells and flare with a conspicuous hypopyon. An evaluation of the posterior segment revealed significant vitreous haze, obstructing all view to the retina. Despite the age and the atypical ocular findings of the patient, a diagnosis of HLA-B27-associated uveitis was made following an extensive clinical and laboratory evaluation. The inflammatory condition was successfully managed with a combination of intravenous, topical and oral corticosteroids tapered over the course of a few weeks, and visual acuity recovered to 20/30.

This case is an important reminder that atypical signs, such as a hypopyon or intermediate uveitis, can occur and may be a significant sign of HLA-B27-associated uveitis. Clinicians should be aware of the diverse manifestations of HLA-B27-associated uveitis and be careful to include a comprehensive assessment of both the anterior and posterior segments of any presenting painful, red eye.

Keywords: *anterior uveitis, intermediate uveitis, HLA-B27, hypopyon*

Introduction

Uveitis, the most common form of inflammatory eye disease, is an important public health concern. It accounts for a significant percentage (estimated at 10–15%) of prevalent cases of legal blindness in the United States.¹ The most frequent subtype is anterior uveitis, representing up to 92% of total cases in community-based ophthalmic practices.² HLA (human leukocyte antigen)-B27 positivity, a human major histocompatibility complex (MHC), is the most common identifiable cause of anterior uveitis and accounts for about 50% of the cases in different populations.^{3,4} HLA-B27-associated uveitis is characterized by recurrent alternating acute unilateral attacks of intraocular inflammation of the anterior segment of the eye, and typically affects young male adults.⁵ In uveitis related to HLA-B27, the presence of a hypopyon – a layer of white blood cells in the anterior chamber – and posterior segment involvement of the eye are uncommon.

A hypopyon suggests severe anterior segment intraocular inflammation and is a rare occurrence in patients with uveitis, occurring in less than 1% of all uveitis patients.⁶ Posterior segment involvement, namely intermediate and/or posterior uveitis, is also infrequent and has been reported in up to 25% of HLA-B27-associated uveitis

cases.^{7–10} Furthermore, anterior and intermediate uveitis cases have a lower risk of hypopyon compared to patients with only anterior uveitis.⁶

This report highlights an atypical case of HLA-B27-associated uveitis that presented with both a hypopyon and severe intermediate uveitis in an elderly man.

Case report

A 60-year-old Caucasian male was seen in the eye clinic of a hospital reporting a red, painful right eye with decreased vision. The patient was in fact seen three days earlier for pain and inflammation from a right shoulder injury for which he was put on a narcotic analgesic (oxycodone 5 mg and acetaminophen 325 mg marketed as Percocet, one tablet every four hours as needed). The onset of his ocular symptoms coincided with the introduction of Percocet so he discontinued the drug after one day, however, his vision continued to worsen. Ocular history was unremarkable with no reports of trauma, surgery, inflammation or infection. Medication was limited to the recent use of Percocet for the shoulder and the occasional nonsteroidal anti-inflammatory drug (naproxen) for nonspecific pain in the body with no reported allergies to any medication. Review of all systems revealed episodes described as podagra (i.e. inflammation on the big toe related to episodes of gout)¹¹

within the past year (although he was never officially diagnosed with gout) and a history of papular rashes on his forehead and both his shins with mild erythema and some excoriation (abraded areas where the skin is torn or worn off). Upon further questioning, the patient reported no history of ulcers, sores, irritable bowel disease, bloody stool, urination difficulties or shortness of breath. The patient's family history was unremarkable. The patient also denied excessive nicotine or alcohol use, and had not engaged in any sexual activity recently. The patient was oriented to time, place and person, and was lucid at the time of the examination.

Upon ocular examination, visual acuity (VA) was hand motion OD (without improvement with pinhole) and 20/30 OS. Slit lamp examination of the right eye revealed 2+ injection of the bulbar conjunctiva. Fine keratic precipitates (KPs) were widely distributed throughout the cornea, although no corneal thinning was noted. Anterior chamber (AC) examination of the right eye revealed 4+ cells and flare (without convection current), fibrin production with a 1.5 mm hypopyon in the lower quadrant as seen in *Figure 1*. Dilated funduscopic examination (DFE) revealed severe vitreous haze (grade 4+ vitreous cells), obstructing all view to the retina. B-scan ultrasound revealed extensive vitreous debris. The left eye revealed only an early nuclear sclerotic cataract with no evidence of active or past inflammation. A summary of the ocular findings is shown in *Table 1*.

Given the B-scan, the patient was diagnosed with acute unilateral

Table 1: Clinical findings at initial presentation

	RIGHT EYE (OD)	LEFT EYE (OS)
Visual acuity	Hand motion	20/30
Pupil reactions	Normal	Normal
Extraocular movements	Normal	Normal
Tonometry (Tonopen)	21 mmHg	22 mmHg
Bulbar conjunctiva	2+ injection	Unremarkable
Cornea	Fine keratic precipitates widely distributed	
Anterior chamber	4+ cells and flare, fibrin production with a 1.5 mm high hypopyon, grade IV Van Herick angle	
Crystalline lens	1+ nuclear sclerotic cataract without posterior synechiae	
Posterior segment	Severe vitreous haze (grade 4+ vitreous cell) with extensive vitreous debris	

nongranulomatous anterior and intermediate hypopyon uveitis, with a tentative HLA-B27 association. The patient received 125 mg of intravenous anti-inflammatory glucocorticoid (methylprednisolone sodium succinate) coupled with a topical anti-inflammatory corticosteroid (1% prednisolone acetate *qb*, with a loading dose before bedtime and upon awakening), a cycloplegic/mydriatic agent (atropine 1% *tid*), and an oral anti-inflammatory corticosteroid (prednisone 80 mg daily). The patient was then sent to the laboratory to have his blood drawn for further analysis.

A subsequent review of his laboratory examination revealed an elevation of ESR, CRP and white blood cells. Laboratory results were positive for the following markers: HLA-B27, HSV IgG and HSV IgM. (See *Table 2*) The remainder of the work-up, including ACE, Toxoplasma, FTA-ABS, RPR and VZV titre, was negative. The etiology of the uveitis was thus confirmed as HLA-

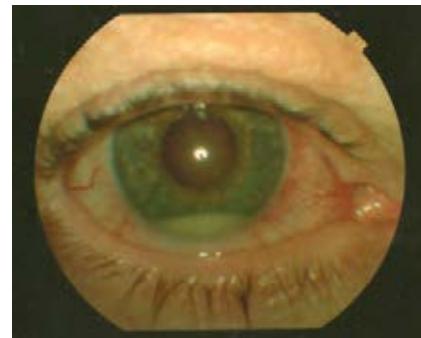


Figure 1 – A hypopyon (height of 1.5mm) seen at the lower quadrant of the anterior chamber

B27 positivity. The patient did not have a primary care provider, so a consultation with a rheumatologist was recommended.

The patient responded well to therapy. Ten days after treatment was initiated, VA of the right eye improved to 20/60, IOP was 16 mmHg, anterior segment revealed few fine KPs inferiorly, 1+ cells and flare in the AC and a <0.5 mm hypopyon. A DFE demonstrated 1+ anterior vitreous cells, snowbanks and snowballs resting

Table 2: Clinical laboratory results

	PURPOSE	NORMAL VALUES	RESULT
Erythrocyte Sedimentation Rate (ESR)	Inflammation	$\leq 30 \text{ mm/hr}$	Elevated (110 mm/hr)
C-Reactive Protein (CRP)	Inflammation	$< 6 \text{ mg/L}$	Elevated (19.35 mg/L)
White blood cells	Inflammation	$4 \times 10^9 \text{ to } 1.1 \times 10^{10}/\text{L}$	Mild leukocytosis ($13.1 \times 10^9/\text{L}$)
Human Leukocyte Antigen B27 (HLA-B27)	Specific protein strongly associated with spondyloarthropathies	Negative	Positive
Herpes Simplex Virus (HSV) IgG	Herpes simplex virus-specific antibody	Negative	Positive
Herpes Simplex Virus (HSV) IgM	Herpes simplex virus-specific antibody	Negative	Positive
Angiotensin-Converting Enzyme (ACE)	Sarcoidosis	Negative	Negative
Toxoplasma	Toxoplasmosis	Negative	Negative
Treponema Pallidum Antibody (FTA-ABS)	Syphilis	Negative	Negative
Rapid Plasma Regain (RPR)	Syphilis	Negative	Negative
Varicella-Zoster Virus (VZV) titer	Varicella zoster virus antibodies	Negative	Negative

inferiorly. The macula, optic disc and the rest of the retina were unremarkable. Due to the marked subjective and objective improvement, tapering of oral prednisone was initiated (60 mg for 5 days, 40 mg for 5 days, 30 mg for 5 days, 20 mg for 5 days, 10 mg for 5 days, 5 mg for 5 days, then discontinued).

At week 3, clinical evolution was favourable with VA at 20/30². The KPs and hypopyon disappeared (*Figure 2*), however grade 0.5+ cells and flare remained in the AC, grade 0.5+ cells in the anterior vitreous, and persistent snowbanks and snowballs. The left eye remained stable and quiet throughout the episode. At his last follow up at week nine, the patient had already discontinued both systemic and topical medication a week prior. His VA was maintained at 20/30¹, IOP was 16 mmHg,

anterior segment was unremarkable, and a posterior segment examination revealed grade 0.5+ vitreous cells, inferior snowballs from five to eight o'clock. The patient was to return for regular monitoring in one month.

Discussion

The differential diagnosis for this patient included infectious and non-infectious etiologies for the uveitis, which includes, HLA-B27 positivity, Herpes simplex virus (HSV), Behcet's disease, sarcoidosis, toxoplasmosis, Varicella-zoster virus (VZV), syphilis and tuberculosis. Multiple sclerosis, Lyme disease and Bartonella, although less probable culprits, could also have been on the list of differentials. A brief description of each can be found in *Table 3*.

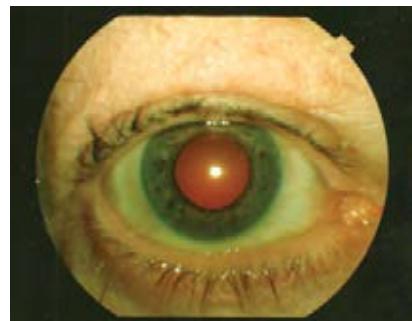


Figure 2: Anterior segment photo illustrating the resolved hypopyon and a clear anterior chamber

The patient was initially diagnosed with an acute nongranulomatous uveitis with a tentative HLA-B27 association, however, the hypopyon and intermediate uveitis were atypical. Ramsay and Lightman (2001) classified the causes of hypopyon into non-infectious causes, infectious agents, neoplasms, and corneal disorders.²⁶ *Table 4* shows the most common differential diagnosis for hypopyon, anterior uveitis and intermediate uveitis.

In both intraocular infection and inflammation, hypopyon consists largely of tissue debris, fibrin, inflammatory by-products and leukocytes, and signifies severe anterior segment intraocular inflammation.⁶ A study by Zaidi et al,⁶ indicates that hypopyon is an uncommon finding in patients with uveitis, occurring in around 8.57 patients per 1000 person-years (0.86%), even in tertiary uveitis practices. This retrospective study indicated that hypopyon was more common among patients with uveitis limited to the anterior chamber than in patients who also had intermediate uveitis as a part of their diagnosis, but was nearly

WHAT ELSE CAN YOU DO IN 10 SECONDS?



- Auto Refraction
- Keratometry
- Placido Disc Topography
- 11,880 Corneal Data Points
- Wavefront Aberrometry
- 2,520 Wavefront Data Points
- SA Cornea for Aspheric IOL selection
- Lenticular- Residual Astigmatism
- Angle Kappa
- Pre/Post Toric IOL Measurements
- Mesopic/Photopic Pupil Size
- Point Spread Function
- Zernike Graphs
- Corneal Refractive Power Map
- IOL Tilt/ Decentration
- Difference Maps-Pre/Post Healing
- WF Optimized Rx
- Day/Night Rx
- RMS Values
- Customized Colour Maps
- EMR Compatibility
- Network Integration
- Viewing software available for Exam Lane
- Pathology Discernment

ALL IN 10 SECONDS

NIDEK OPD SCAN 3

Table 3: Possible etiologies for uveitis

	GENERAL DESCRIPTION	OCULAR MANIFESTATIONS
HLA-B27 positivity ⁵	Genetic marker associated with spondyloarthropathies Most common identifiable cause of anterior uveitis (50%)	Anterior uveitis
Herpes simplex uveitis ^{12, 13, 35-38}	Uveitis caused by Herpes simplex virus Secondary to herpetic keratitis Absence of corneal disease in 15%	Corneal scarring Unilateral recurrent anterior uveitis Iris atrophy, elevated IOP, KPs and synechiae Hypopyon possible Posterior segment inflammation in 15%
Behçet's disease ¹⁴	Systemic vasculitis disorder of unknown etiology Relapsing episodes of oral ulcers, genital ulcers and skin lesions	Sudden attacks of anterior uveitis with hypopyon
Sarcoidosis ^{15, 16, 17}	Chronic non-caseating granulomatous systemic disease of unknown etiology A third of patients have ocular involvement	Chronic bilateral anterior uveitis Iris nodules, posterior synechiae, KPs and peripheral anterior synechiae Posterior segment involvement possible
Toxoplasmosis ^{2, 18, 19}	Infection caused by the parasite Toxoplasma gondii Most common infectious cause of intraocular inflammation in immunocompetent patients	Can cause anterior uveitis and retinal vasculitis
Herpes zoster ophthalmicus (ocular shingles) ²⁰	Reactivation of the Varicella-zoster virus 50% develop ocular complications, with uveitis occurring in 43%	High incidence of secondary glaucoma Uveitis is uniphasic and short-lived
Syphilis ²¹⁻²⁴	Sexually-transmitted infectious disease by Treponema pallidum Many people do not have symptoms Can initially present as anterior uveitis	Can mimic many ocular conditions Uveitis can be unilateral or bilateral, acute or chronic Can cause hypopyon
Tuberculous uveitis ²⁵	Rare Caused by Mycobacterium tuberculosis 2% of active tuberculosis	Chronic granulomatous anterior uveitis Disseminated choroiditis with vitritis Cystoid macular oedema

as frequent among patients with posterior or panuveitis. The most common risk factors for hypopyon are Behçet's disease and HLA-B27 positivity, conferring respectively an approximate five-fold and two-fold increased risk of hypopyon. In fact, HLA-B27 hypopyon uveitis occurs in 5.7% of all uveitis cases and is more common among Caucasians.²⁷ Even though hypopyon is an indicator of remarkably severe inflammation, eyes that develop hypopyon do not appear to have adverse visual outcomes more often than eyes without it. A previous study on Behçet's disease showed that patients who developed hypopyon were more likely to gain three lines of vision at any point during follow-up,

probably because the haze associated with a hypopyon was a reversible cause of vision loss.²⁸

Intermediate uveitis is diagnosed when intraocular inflammation primarily involves the vitreous, peripheral retina and pars plana ciliaris.²⁹ It is the type of uveitis with the longest clinical duration.³⁰ The syndrome is more frequent in the third and fourth decade.³¹ Intermediate uveitis has been reported to make up 1.4 – 22% of all uveitis cases.³² Although the majority of cases are of unknown etiology, a significant association between intermediate uveitis and multiple sclerosis, sarcoidosis and Lyme disease has been reported.^{33, 34} Intermediate uveitis is bilateral 80% of the time. Main

clinical features are vitreous cells, with or without snowballs and snow banking. The intermediate uveitis found in this case was atypical in that it was monocular and not associated with the aforementioned diseases.

In the case reported here, the patient's laboratory results were positive for HLA-B27, HSV IgG and HSV IgM, which pointed towards an etiology of either an HLA-B27-associated uveitis or Herpes simplex uveitis.

Acute HSV uveitis is typically secondary to herpetic keratitis, although 15% of patients may not experience corneal involvement.^{35, 39} Clinical signs of HSV uveitis include corneal scarring, focal or patchy iris atrophy, iris transillumination defects, KP,

Table 4: Most common differential diagnosis of hypopyon, anterior uveitis and intermediate uveitis

HYPOPYON	ANTERIOR UVEITIS	INTERMEDIATE UVEITIS
<ul style="list-style-type: none"> ● Non-infectious <ul style="list-style-type: none"> HLA-B27 Behçet's disease Spondyloarthropathy Iatrogenic/Neoplasm/Trauma ● Infectious <ul style="list-style-type: none"> Endogenous endophthalmitis Toxoplasmosis Syphilis Hansen's disease Brucellosis HSV Keratitis ● Other 	<ul style="list-style-type: none"> ● Idiopathic ● HLA-B27 ● Reactive arthritis (Reiter's syndrome) ● Ankylosing spondylitis ● Sarcoidosis ● Syphilis ● HSV ● Trauma ● Posner-Schlossman Syndrome ● Neoplasm <ul style="list-style-type: none"> Lymphoma, leukemia, retinoblastoma ● Other 	<ul style="list-style-type: none"> ● Idiopathic ● Multiple sclerosis ● Sarcoidosis ● Lyme disease

posterior synechiae, and elevated IOP.³⁹⁻⁴¹ A unilateral anterior uveitis coupled with an elevated IOP point to HSV uveitis. The presence of patchy iris atrophy and transillumination defects corroborates the diagnosis. Since the clinical presentation of this case did not involve the iris or an elevated IOP, HSV uveitis was rejected as a potential diagnosis and HLA-B27 positivity was favoured as the main etiology.

Despite the atypical presentation, the prognosis was positive for this patient, like in most HLA-B27-associated uveitides. With only topical and systemic steroids, the clinical progression was favourable and the patient regained VA to 20/30¹ within 9 weeks. The patient suffered from inflammation of his right shoulder just before the onset of his uveitis attack. It is unclear at this time whether the shoulder inflammation and the uveitis are two separate inflammatory events, or whether they are part of a single systemic problem. HLA-B27-associated anterior uveitis patients with concomitant posterior segment manifestations have a significantly higher incidence of associated

systemic diseases, namely ankylosing spondylitis, inflammatory bowel diseases or reactive arthritis (formerly referred to as Reiter's syndrome).⁴² Thus, a referral to rheumatology was recommended to rule these out. The patient has been educated that recurrences are highly possible, and that this process may be part of a systemic inflammatory condition, which may require steroids and/or chronic immunosuppressive therapy. However, the patient was not seen in a follow-up after the referral.

HLA-B27-associated uveitis

As the most common specific uveitis diagnosis, HLA-B27-associated uveitis accounts for approximately 13–17% of all uveitis cases.^{2,43} About 50% of patients suffering from acute anterior uveitis are HLA-B27 positive.⁵ HLA-B27-associated uveitis is three times more common in males.⁴⁴ The average age of onset of the disease is 35, although cases have been reported in children (10% of cases begin prior to 20 years old) and late adulthood (5% after 55 years of age).¹⁰

The classic presentation of HLA-B27-associated ocular disease is acute anterior uveitis (AAU).¹⁰ The onset is typically abrupt and symptoms include photophobia, ocular pain, epiphora, ocular redness, and mild-to-severe visual blurring. Cases are generally unilateral but a recurrent attack may affect the contralateral eye. Although the inflammation is usually nongranulomatous, it may be severe enough to cause a hypopyon or a plasmoid aqueous. HLA-B27-associated AAU is in fact the most common cause of hypopyon uveitis in North America.²⁷ Uncommon cases (less than 25.1%) cases may involve the posterior segment.⁷ Such posterior segment involvement is recognized as vitritis, cystoid macular oedema, papillitis and retinal vasculitis, and is thought to be secondary to anterior segment inflammation.^{10,45} Thus, HLA-B27-associated uveitis may be unusually severe and may cause a panuveitis, which is an under-recognized phenomenon.⁴⁶ During an acute attack, IOP is generally lowered due to the shutdown of the ciliary body. Nevertheless, increased IOP and secondary glaucoma is a

well-recognized complication due to iris bombé or synechial angle closure. Uveitis attacks are normally short-lived and resolve within three months. Recurrent episodes are common, but the frequency varies between multiple attacks per year to single attacks separated by one or more decades.⁴⁶

There is a stepladder approach to the management of uveitis.⁴⁷ The immediate goal is to control the inflammation and ciliary spasm, with a long-term goal of addressing the underlying cause of the uveitis. First-time occurrences and uncomplicated recurrences can be treated with topical corticosteroid, such as prednisolone acetate 1%, which has a moderate potency and is appropriate for many cases. Although the risk of systemic side effects is low, 1% of patients experience increased IOP, in which case a milder formulation such as rimexolone and lotoprednol can be utilized.⁴⁸ The concomitant use of a cycloplegic/mydriatic drop, such as atropine sulphate 1%, reduces pain from ciliary spasm and may break or prevent posterior synechiae.

More severe cases may require a combination of oral, topical, periocular or intravitreal treatments. Systemic corticosteroid administration is required in 24% of patients. A typical oral starting dose is 1 mg/kg of prednisone daily. Side effects resulting from a short course of systemic steroids are infrequent but include sleep disturbances, weight gain, increased appetite, mood imbalance and more.⁴⁹ In extreme cases of uveitis involving the posterior segment, periocular or intravitreal corticosteroids may be considered.⁵⁰

Periocular injections (transseptally, in the sub-tenon's space or subconjunctivally) are designed as depot injections (e.g. triamcinolone acetate) and therefore, are effective for an extended period of time.⁵¹ Intravitreal injections are effective for three to six months, minimize systemic side effects, and have the benefit of treating macular oedema caused by posterior uveitis.⁵² On the other hand, ocular complications (e.g. cataracts and increased IOP) are more common with intravitreal injections than with systemic steroids.

Extreme sight-threatening cases may require pulse intravenous (IV) steroids to bring the inflammation under control more quickly and to prevent irreparable damage. The recommended regimen for such cases is methylprednisolone 1 g IV per day for 3 days, with subsequent transition to oral therapy, starting at 1 mg/kg per day.^{47, 53, 54} For recalcitrant cases, steroid-free strategies can be considered, such as oral NSAID therapy or immunosuppressive therapy.^{55, 56, 47}

Patients on topical or systemic steroids for an extended period of time (i.e. over two weeks) should be tapered off over the course of several weeks to avoid rebound inflammation after topical use, or inducing adrenal crisis from abrupt stoppage of oral corticosteroid use. Patients requiring immunosuppressive therapy should remain on their regimen without dose reductions to prevent any recurrences, which may be difficult to control.

HLA-B27 testing in patients with uveitis is useful because it may help to identify a previously undiagnosed systemic disease. Among patients

with HLA-B27-associated uveitis, around 70% will have an associated (rheumatoid factor) seronegative spondyloarthropathy of which approximately 50% will not have been diagnosed or will have been misdiagnosed. Seronegative spondyloarthropathy includes ankylosing spondylitis, reactive arthritis (Reiter's syndrome), psoriatic arthropathy, and arthritis associated with inflammatory bowel disease. Patients with posterior segment manifestations have a significantly higher incidence of such systemic diseases.^{42, 57} In addition, around 30–90% of patients with HLA-B27-associated uveitis suffer from associated joint disease.⁵⁸ As a result, HLA-B27 testing can be beneficial in improving management of the patient's overall systemic health.

Summary

In this case report, a final diagnosis of HLA-B27-associated uveitis was made following an extensive clinical and laboratory evaluation despite the atypical presentation. The hypopyon and its associated anterior and intermediate uveitis were successfully treated with topical and systemic steroids. This case is an important reminder that, although uncommon, hypopyon and posterior segment involvement may be present in an HLA-B27-associated uveitis, and can even affect the elderly. Furthermore, it is important to include a comprehensive assessment of both the anterior and posterior segments of any presenting painful, red eye for a full clinical appreciation.

Acknowledgement

We would like to thank Gerald Abruñez, OD from the Syracuse Veterans Affairs Medical Center for his help and guidance.

This paper was presented in part, as a poster at the American Academy of Optometry's Annual meeting in Boston on October 14, 2011.

References

1. Rothova A, Suttorp-van Schulten MS, Frits Treffers W, Kijlstra A. Causes and frequency of blindness in patients with intraocular inflammatory disease. *Br J Ophthalmol* 1996;80(4):332-6.
2. McCannel CA, Holland GN, Helm CJ, et al. Causes of uveitis in the general practice of ophthalmology. UCLA Community-Based Uveitis Study Group. *Am J Ophthalmol* 1996;121(1):35-46.
3. Brewerton DA, Caffrey M, Nicholls A, et al. Acute anterior uveitis and HLA-A 27. *Lancet* 1973;302(7836):994-6.
4. Feltkamp TE. Ophthalmological significance of HLA associated uveitis. *Eye (Lond)* 1990;4 (Pt 6):839-44.
5. Wakefield D, Chang JH, Amjadi S, et al. What is new HLA-B27 acute anterior uveitis? *Ocul Immunol Inflamm* 2011;19(2):139-44.
6. Zaidi AA, Ying GS, Daniel E, et al. Hypopyon in patients with uveitis. *Ophthalmology* 2010;117(2):366-72.
7. Kase S, Namba K, Horie Y, et al. Repeated exacerbations of ocular inflammation with vitreous hemorrhage in a patient with HLA-B27 associated uveitis. *J Med Invest* 2007;54(3-4):350-3.
8. Dodds EM, Lowder CY, Meisler DM. Posterior segment inflammation in HLA-B27+ acute anterior uveitis: clinical characteristics. *Ocul Immunol Inflamm* 1999;7(2):85-92.
9. Rothova A. Comment on 'Posterior segment inflammation in HLA-B27+ acute anterior uveitis: clinical characteristics'. *Ocul Immunol Inflamm* 2000;8(1):73-5.
10. Tay-Kearney ML, Schwam BL, Lowder C, et al. Clinical features and associated systemic diseases of HLA-B27 uveitis. *Am J Ophthalmol* 1996;121(1):47-56.
11. Schumacher HR, Jr. The pathogenesis of gout. *Cleve Clin J Med* 2008;75 Suppl 5:S2-4.
12. Dawson CR, Togni B. Herpes simplex eye infections: clinical manifestations, pathogenesis and management. *Surv Ophthalmol* 1976;21(2):121-35.
13. Santos C. Herpes simplex uveitis. *Bol Asoc Med P R* 2004;96(2):71-4, 7-83.
14. Mendes D, Correia M, Barbedo M, et al. Behcet's disease--a contemporary review. *J Autoimmun* 2009;32(3-4):178-88.
15. Cozier YC, Berman JS, Palmer JR, et al. Sarcoidosis in black women in the United States: data from the Black Women's Health Study. *Chest* 2011;139(1):144-50.
16. Uyama M. Uveitis in sarcoidosis. *Int Ophthalmol Clin* 2002;42(1):143-50.
17. Jones NP. Sarcoidosis and uveitis. *Ophthalmol Clin North Am* 2002;15(3):319-26, vi.
18. Park YH, Han JH, Nam HW. Clinical features of ocular toxoplasmosis in Korean patients. *Korean J Parasitol* 2011;49(2):167-71.
19. Bornand JE, de Gottrau P. Uveitis: is ocular toxoplasmosis only a clinical diagnosis? *Ophthalmologica* 1997;211(2):87-9.
20. Thean JH, Hall AJ, Stawell RJ. Uveitis in Herpes zoster ophthalmicus. *Clin Experiment Ophthalmol* 2001;29(6):406-10.
21. Hong MC, Sheu SJ, Wu TT, Chuang CT. Ocular uveitis as the initial presentation of syphilis. *J Chin Med Assoc* 2007;70(7):274-80.
22. Reddy S, Cubillan LD, Hovakimyan A, Cunningham ET, Jr. Inflammatory ocular hypertension syndrome (IOHS) in patients with syphilitic uveitis. *Br J Ophthalmol* 2007;91(12):1610-2.
23. Tucker JD, Li JZ, Robbins GK, et al. Ocular syphilis among HIV-infected patients: a systematic analysis of the literature. *Sex Transm Infect* 2011;87(1):4-8.
24. Lutchman C, Weisbrod DJ, Schwartz CE. Diagnosis and management of syphilis after unique ocular presentation. *Can Fam Physician* 2011;57(8):896-9.
25. Varma D, Anand S, Reddy AR, et al. Tuberculosis: an under-diagnosed etiological agent in uveitis with an effective treatment. *Eye (Lond)* 2006;20(9):1068-73.
26. Ramsay A, Lightman S. Hypopyon uveitis. *Surv Ophthalmol* 2001;46(1):1-18.
27. D'Alessandro LP, Forster DJ, Rao NA. Anterior uveitis and hypopyon. *Am J Ophthalmol* 1991;112(3):317-21.
28. Nussenblatt RB. Uveitis in Behcet's disease. *Int Rev Immunol* 1997;14(1):67-79.
29. Jabs DA, Nussenblatt RB, Rosenbaum JT. Standardization of uveitis nomenclature for reporting clinical data. Results of the First International Workshop. *Am J Ophthalmol* 2005;140(3):509-16.
30. Bloch-Michel E. Opening address: intermediate uveitis. *Dev Ophthalmol* 1992;23:1-2.
31. Chan SM, Hudson M, Weis E. Anterior and intermediate uveitis cases referred to a tertiary centre in Alberta. *Can J Ophthalmol* 2007;42(6):860-4.
32. Babu BM, Rathinam SR. Intermediate uveitis. *Indian J Ophthalmol* 2010;58(1):21-7.
33. Zierhut M, Foster CS. Multiple sclerosis, sarcoidosis and other diseases in patients with pars planitis. *Dev Ophthalmol* 1992;23:41-7.
34. Breeveld J, Rothova A, Kuiper H. Intermediate uveitis and Lyme borreliosis. *Br J Ophthalmol* 1992;76(3):181-2.
35. Miserocchi E, Waheed NK, Dios E, et al. Visual outcome in herpes simplex virus and varicella zoster virus uveitis: a clinical evaluation and comparison. *Ophthalmology* 2002;109(8):1532-7.

36. Liesegang TJ. Ocular herpes simplex infection: pathogenesis and current therapy. *Mayo Clin Proc* 1988;63(11):1092-105.
37. Schacher S, Garweg JG, Russ C, Bohnke M. [Diagnosis of herpetic uveitis and keratouveitis]. *Klin Monbl Augenheilkd* 1998;212(5):359-62.
38. Rodriguez A, Power WJ, Neves RA, Foster CS. Recurrence rate of herpetic uveitis in patients on long-term oral acyclovir. *Doc Ophthalmol* 1995;90(4):331-40.
39. Liesegang TJ. Herpes simplex virus epidemiology and ocular importance. *Cornea* 2001;20(1):1-13.
40. Kaye S, Choudhary A. Herpes simplex keratitis. *Prog Retin Eye Res* 2006;25(4):355-80.
41. Liesegang TJ. Classification of herpes simplex virus keratitis and anterior uveitis. *Cornea* 1999;18(2):127-43.
42. Kataria RK, Brent LH. Spondyloarthropathies. *Am Fam Physician* 2004;69(12):2853-60.
43. Rodriguez A, Calonge M, Pedroza-Seres M, et al. Referral patterns of uveitis in a tertiary eye care center. *Arch Ophthalmol* 1996;114(5):593-9.
44. Power WJ, Rodriguez A, Pedroza-Seres M, Foster CS. Outcomes in anterior uveitis associated with the HLA-B27 haplotype. *Ophthalmology* 1998;105(9):1646-51.
45. Rodriguez A, Akova YA, Pedroza-Seres M, Foster CS. Posterior segment ocular manifestations in patients with HLA-B27-associated uveitis. *Ophthalmology* 1994;101(7):1267-74.
46. Smith JR. HLA-B27-associated uveitis. *Ophthalmol Clin North Am* 2002;15(3):297-307.
47. Lee FF, Foster CS. Pharmacotherapy of uveitis. *Expert Opin Pharmacother* 2010;11(7):1135-46.
48. Tripathi RC, Parapuram SK, Tripathi BJ, et al. Corticosteroids and glaucoma risk. *Drugs Aging* 1999;15(6):439-50.
49. Noone T. An overview of steroid use and its potential side-effects. *Nurs Times* 2006;102(17):24-7.
50. Ferrante P, Ramsey A, Bunce C, Lightman S. Clinical trial to compare efficacy and side-effects of injection of posterior sub-Tenon triamcinolone versus orbital floor methylprednisolone in the management of posterior uveitis. *Clin Experiment Ophthalmol* 2004;32(6):563-8.
51. Riordan-Eva P, Lightman S. Orbital floor steroid injections in the treatment of uveitis. *Eye (Lond)* 1994;8 (Pt 1): 66-9.
52. Antcliff RJ, Spalton DJ, Stanford MR, et al. Intravitreal triamcinolone for uveitic cystoid macular edema: an optical coherence tomography study. *Ophthalmology* 2001;108(4):765-72.
53. Yalcindag FN, Can E, Ozdemir O. Intravenous methylprednisolone pulse therapy for acute posterior segment uveitis attacks in Behcet's disease. *Ann Ophthalmol (Skokie)* 2007;39(3):194-7.
54. Wakefield D, McCluskey P, Penny R. Intravenous pulse methylprednisolone therapy in severe inflammatory eye disease. *Arch Ophthalmol* 1986;104(6):847-51.
55. Okada AA. Immunomodulatory therapy for ocular inflammatory disease: a basic manual and review of the literature. *Ocul Immunol Inflamm* 2005;13(5):335-51.
56. Bom S, Zamiri P, Lightman S. Use of methotrexate in the management of sight-threatening uveitis. *Ocul Immunol Inflamm* 2001;9(1):35-40.
57. Monnet D, Breban M, Hudry C, et al. Ophthalmic findings and frequency of extraocular manifestations in patients with HLA-B27 uveitis: a study of 175 cases. *Ophthalmology* 2004;111(4): 802-9.
58. Rosenbaum JT. Acute anterior uveitis and spondyloarthropathies. *Rheum Dis Clin North Am* 1992;18(1):143-51.

A vision for the future • Une vision pour l'avenir



CRA Registered Charity / Numéro d'organisme de bienfaisance de l'ARC #118834852

The Canadian Optometric Education Trust Fund (COETF) advances growth, research and education in Canada. This is our charity—a trust fund for the profession, supported by the profession.

Invest in optometry's future. Make a charitable donation today.

opto.ca/coetc

Grâce à votre don de bienfaisance, vous favorisez la recherche, la croissance, l'éducation et le développement de la profession optométrique au Canada.

Faire un don de bienfaisance aujourd'hui.

opto.ca/ffoce

CANADIAN
OPTOMETRIC
EDUCATION
TRUST FUND



FONDS DE FIDUCIE DES
OPTOMÉTRISTES CANADIENS
POUR L'ÉDUCATION

Cas atypique d'uvéite associée à l'antigène HLA-B27 avec hypopyon et atteinte du segment postérieur

PAR THOMAS XIE, OD & ETTY BITTON, OD, MSc, FAAO

RÉSUMÉ

La présence d'un hypopyon et l'atteinte du segment postérieur sont des signes cliniques peu fréquents dans les cas d'uvéite associée à l'antigène HLA-B27. De plus, les premières attaques sont rares chez les personnes âgées. Ce rapport décrit un cas atypique d'uvéite chez une personne âgée qui s'est présentée à la fois avec un hypopyon visible et une uvéite intermédiaire sévère dans un œil.

Un homme caucasien de 60 ans a consulté à cause d'un œil rouge et douloureux avec baisse subite de la vision au niveau de la perception de la main dans l'œil atteint. Ses antécédents oculaires et systémiques ne présentaient rien de remarquable. Un examen de la chambre antérieure de l'œil a révélé la présence de cellules inflammatoires importantes et un hypopyon flagrant. Un examen du segment postérieur a révélé la présence d'une vaste hyalite, entravant toute visibilité de la rétine. En dépit de l'âge et des signes oculaires atypiques du patient, un diagnostic d'une uvéite liée à l'antigène HLA-B27 à la suite d'une évaluation approfondie des signes cliniques et des résultats d'analyses de laboratoire a été proposé. L'inflammation a été traitée avec succès au moyen d'une combinaison de corticostéroïdes administrés par voies intraveineuse, topique et orale dont la dose a diminué progressivement en quelques semaines. L'acuité visuelle s'est rétablie à 20/30.

Ce cas constitue un rappel important du fait que des signes atypiques comme un hypopyon ou une uvéite intermédiaire peuvent apparaître et constituer un signe important d'uvéite liée à l'antigène HLA-B27. Les cliniciens doivent connaître les différentes manifestations de l'uvéite associée à l'antigène HLA-B27 et agir avec prudence en incluant une évaluation complète des segments antérieur et postérieur face à un œil rouge et douloureux associé à une baisse de vision.

Mots clés : uvéite antérieure, uvéite intermédiaire, HLA-B27, hypopyon

Introduction

L'uvéite, la forme la plus courante d'inflammation oculaire, constitue une importante préoccupation en santé publique. Elle cause un pourcentage important (estimé de 10 à 15 %) des cas prévalents de cécité légale aux États-Unis.¹ Le sous-type le plus répandu est l'uvéite antérieure, qui représente jusqu'à 92 % du total des cas dans les pratiques ophtalmiques communautaires². La positivité au HLA (antigène leucocytaire humain) – B27, complexe majeur d'histocompatibilité (CMH) des humains, constitue la cause identifiée la plus courante de l'uvéite antérieure et cause environ 50 % des cas dans des populations différentes.^{3,4} L'uvéite associée au HLA-B27 est caractérisée par des poussées unilatérales aiguës en alternance et récidivantes d'inflammation intraoculaire du segment antérieur de l'œil et touche habituellement les jeunes hommes adultes.⁵ Dans les cas d'uvéite reliée au HLA-B27, la présence d'un hypopyon – amas de globules blancs dans la chambre antérieure – et l'atteinte du segment postérieur de l'œil ne sont pas courantes.

Un hypopyon indique la présence d'une inflammation intraoculaire sévère du segment antérieur et il est rare chez les patients qui ont une uvéite, faisant

son apparition dans moins de 1 % des cas.⁶ L'atteinte du segment postérieur, appelée uvéite intermédiaire ou postérieure, est peu fréquente elle aussi et elle varie de 0 à 25 % des cas d'uvéite associée au HLA-B27.⁷⁻¹⁰ De plus, les cas d'uvéite antérieure et intermédiaire présentent un risque plus faible d'hypopyon comparativement aux patients qui ont seulement une uvéite antérieure.⁶

Le présent rapport décrit un cas atypique d'uvéite associée au HLA-B27 chez un homme âgé qui avait à la fois un hypopyon et une uvéite intermédiaire sévère.

Rapport de cas

Un homme caucasien de 60 ans s'est présenté à la clinique oculaire d'un hôpital en se plaignant d'avoir l'œil droit douloureux, rouge, avec une baisse de la vision. Le patient avait en fait été vu trois jours plus tôt à cause d'une douleur et d'une inflammation causées par un traumatisme à l'épaule droite pour lesquelles on lui avait prescrit un analgésique narcotique (oxy-codone 5 mg et acétaminophène 325 mg vendu sous la marque Percocet, un comprimé aux quatre heures au besoin). Les symptômes oculaires sont apparus lorsqu'il a commencé à prendre le Percocet, et c'est pourquoi il a cessé de prendre le médicament après une journée, mais sa vision a continué

Tableau 1 : Présentation clinique initiale

	ŒIL DROIT (OD)	ŒIL GAUCHE (OS)
Acuité visuelle	Mouvement de la main	20/30
Réflexes des pupilles	Normaux	Normaux
Mouvements extraoculaires	Normaux	Normaux
Tonométrie (TonoPén)	21 mmHg	22 mmHg
Conjonctive bulbaire	Injection 2+	
Cornée	Précipités kératiques fins généralisés	
Chambre antérieure	Cellules 4+ et « flare », production de fibrines et hypopyon de 1,5 mm de hauteur, angle Van Herick grade IV	Rien de remarquable
Cristallin	Sclérose nucléaire 1+ sans synéchies postérieures	
Segment postérieur	Cellules et débris inflammatoires vitréens prononcés (grade 4+)	

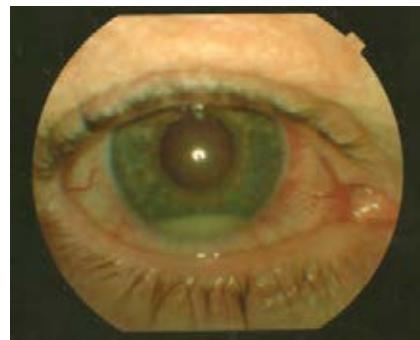


Figure 1 – Hypopyon (hauteur de 1,5 mm) dans le quadrant inférieur de la chambre antérieure.

à se détériorer. Ses antécédents oculaires ne présentaient rien de remarquable et ne comportaient aucun traumatisme, ni chirurgie, inflammation ou infection. Comme médicaments, il prenait seulement le Percocet pour sa douleur à l'épaule depuis peu et, à l'occasion, un anti-inflammatoire non stéroïdien (naproxen) pour une douleur non spécifique dans le corps. Il n'avait aucune allergie déclarée à aucun médicament. L'examen de tous les systèmes a révélé des poussées appelées podagre (c.-à-d. inflammation du gros orteil liée à des récidives de goutte)¹¹ au cours de la dernière année (même si l'on n'a jamais diagnostiqué officiellement qu'il avait la goutte) et des antécédents d'éruptions papulaires au front et aux deux tibias avec érythème bénin et un peu d'excoriation. Suite à un questionnement plus approfondi, le patient n'a signalé aucun

antécédent d'ulcère, de lésion, de syndrome du côlon irritable, de selles sanguinolentes, de difficulté à uriner ou d'essoufflement. Les antécédents familiaux du patient ne présentaient rien de remarquable. Le patient a aussi affirmé ne pas abuser de la nicotine et de l'alcool et ne pas avoir eu de rapport sexuel récent. Le patient avait conscience du temps, de l'espace et de sa personne, et il était lucide au moment de l'examen.

L'examen oculaire a révélé que l'acuité visuelle (AV) était limitée au mouvement de la main OD (sans amélioration au trou sténopéique) et s'établissait à 20/30 OS. L'examen biomicroscopique de l'œil droit a révélé une injection 2+ de la conjonctive bulbaire. Des précipités kératiques fins étaient distribués généralement sur toute la cornée. Aucun amincissement n'a été constaté. L'examen de la chambre antérieure (CA) de l'œil

droit a révélé la présence de cellules 4+ et un effet Tyndall (sans courant de convection), la production de fibrine et un hypopyon d'une hauteur de 1,5 mm dans le quadrant inférieur comme le montre la *Figure 1*. L'examen du fond d'œil avec dilatation pupillaire a révélé la présence d'un brouillard sévère du vitré (cellules inflammatoires grade 4+) bloquant la vue de la rétine. Une ultrasonographie de l'œil (B-Scan) a révélé la présence de nombreux débris au vitré. L'examen de l'œil gauche a révélé seulement la présence d'une sclérose nucléaire précoce sans signe d'inflammation active ou antérieure. Le *Tableau 1* résume les observations cliniques.

Après l'échographie B, on a diagnostiqué chez le patient une uvéite à hypopyon antérieure et intermédiaire non granulomateuse unilatérale et aiguë en lien présumé avec l'anticorps HLA-B27. Le patient a reçu par voie intraveineuse 125 mg de glucocorticoïde anti-inflammatoire (succinate sodique de méthylprednisolone) jumelé à un corticostéroïde anti-inflammatoire topique (acétate de

Tableau 2 : Résultats de laboratoire cliniques

	OBJET	VALEURS NORMALES	RÉSULTAT
Taux de sédimentation	Inflammation	≤ 30 mm/h	Élévation (110 mm/h)
Protéine C-réactive	Inflammation	<6 mg/L	Élévation (19,35 mg/L)
Globules blanches	Inflammation	4 × 10 ⁹ à 1,1 × 10 ¹⁰ /L	Légère leucocytose (13,1 × 10 ⁹ /L)
Antigène leucocytaire humain B27 (HLA-B27)	Protéine spécifique fortement liée aux spondyloarthropathies	Négatif	Positif
IgG du virus de l'herpès simplex	Anticorps spécifiques du virus de l'herpès simplex	Négatif	Positif
IgM du virus de l'herpès simplex	Anticorps spécifiques du virus de l'herpès simplex	Négatif	Positif
Enzyme de conversion de l'angiotensine	Sarcoïdose	Négatif	Négatif
Toxoplasma	Toxoplasmose	Négatif	Négatif
Anticorps du Treponema pallidum (FTA-ABS)	Syphilis	Négatif	Négatif
Anticorps réaginique (RPR)	Syphilis	Négatif	Négatif
Dosage du virus zona varicelle (VZV)	Anticorps du virus zona varicelle	Négatif	Négatif

prednisolone à 1 % *qb*, avec dose d'attaque avant le coucher et au réveil), un agent cycloplégique/ mydiatrique (atropine à 1 % *tid*) et un corticostéroïde anti-inflammatoire à prendre par voie orale (prednisone 80 mg tous les jours). Le patient a ensuite été envoyé au laboratoire pour une prise de sang devant servir à une analyse plus poussée.

Une étude subséquente des résultats du bilan sanguin (*Tableau 2*) a révélé une élévation de taux de sédimentation, protéine C-réactif et des globules blancs. Les résultats de laboratoire étaient positifs pour les marqueurs suivants : HLA-B27, IgG et IgM du virus de l'Herpès. Les autres

résultats de l'analyse, y compris ACE, Toxoplasma, FTA-ABS, RPR et titrage VZV, étaient négatifs. L'étiologie de l'uvéite a donc été confirmée comme positive au HLA-B27. Le patient n'avait pas de médecin généraliste et c'est pourquoi on a recommandé une consultation avec un rhumatologue.

Le patient a bien répondu au traitement. Dix jours après le début du traitement, l'acuité visuelle de l'œil droit s'était améliorée à 20/60, la PIO s'établissait à 16 mmHg, le segment antérieur a révélé la présence de quelques précipités kératiques dans la partie inférieure, des cellules 1+ et des cellules inflammatoires dans la

CA, de même qu'un hypopyon de <0,5 mm. Un examen du fond de l'œil avec dilatation pupillaire a démontré la présence de cellules vitréennes antérieures 1+ et des accumulations d'xsudats étendues (« snowbanks ») et limitées (« snowballs ») à la partie inférieure. La macula, le nerf optique et le reste de la rétine ne présentaient rien de remarquable. À cause de l'amélioration subjective et objective prononcées, la dose de prednisone a été progressivement réduite (60mg × 5 jours, 40 mg × 5 jrs, 30 mg × 5 jrs, 20 mg × 5 jrs, 10 mg × 5 jrs, 5 mg × 5 jrs et ensuite un arrêt du médicament).

Au cours de la troisième semaine, l'évolution clinique était favorable et l'AV s'établissait à 20/30². Les précipités et l'hypopyon ont disparu (*Figure 2*), mais des cellules inflammatoires dans la CA de grade 0,5+ sont demeurés, ainsi que des cellules au vitré antérieur de 0,5+ et une persistance d'xsudat à la rétine. L'œil gauche est demeuré stable et sans manifestation durant l'épisode. Au dernier suivi à neuf semaines, le patient avait déjà cessé de prendre les médicaments systémiques et topiques une semaine plus tôt. Son AV s'était maintenue à 20/30¹, la PIO était de 16 mmHg, le segment antérieur ne présentait rien de remarquable et un examen du segment postérieur a révélé la présence de cellules vitréennes de grade 0,5+ et d'xsudats limités à la partie inférieure, entre 5 à 8 heures. Le patient devait revenir pour un suivi périodique un mois plus tard.

Tableau 3 : Étiologies possibles de l'uvéite

	DESCRIPTION GÉNÉRALE	MANIFESTATIONS OCULAIRES
Positivité au HLA-B27 ⁵	Marqueur génétique associé aux spondyloarthropathies Cause identifiable la plus courante de l'uvéite antérieure (50 %)	Uvéite antérieure
Uvéite à herpès simplex ^{12, 13, 35-38}	Uvéite causée par le virus de l'herpès simplex Secondaire à une kératite herpétique Aucune atteinte cornéenne dans 15 % des cas	Cicatrisation cornéenne Uvéite antérieure récidivante unilatérale Atrophie de l'iris, élévation de la PIO, précipités et synéchies Hypopyon possible Inflammation du segment postérieur dans 15 % des cas
Maladie de Behçet ¹⁴	Vasculite systémique d'étiologie inconnue Récidive d'ulcères dans la bouche, d'ulcères génitaux et de lésions cutanées	Crise subite d'uvéite antérieure avec hypopyon
Sarcoïdose ^{15, 16, 17}	Maladie chronique systémique granulomateuse sans caséification d'étiologie inconnue Atteinte oculaire chez le tiers des patients	Uvéite antérieure bilatérale chronique Nodules iridiens, synéchies postérieures, précipités et synéchies antérieures périphériques Atteinte possible du segment postérieur
Toxoplasmose ^{2, 18, 19}	Infection causée par le parasite Toxoplasma gondii Cause infectieuse la plus courante d'inflammation intraoculaire chez les patients immunocompétents	Peut causer l'uvéite antérieure et la vasculite rétinienne
Ophthalmicus à herpès zoster (zona oculaire) ²⁰	Réactivation du virus zona de la varicelle Complications oculaires chez 50 % des patients avec uvéite dans 43 % des cas	Incidence élevée de glaucome secondaire Uvéite à phase unique et de brève durée
Syphilis ²¹⁻²⁴	Infection à Treponema pallidum transmise sexuellement Plusieurs personnes n'ont pas de symptômes Peut se manifester au début sous forme d'uvéite antérieure	Peut imiter de nombreuses conditions oculaires. L'uvéite peut être unilatérale ou bilatérale, aiguë ou chronique Peut causer l'apparition d'un hypopyon
Uvéite tuberculeuse ²⁵	Rare Causée par Mycobacterium tuberculosis 2 % des cas de tuberculose active	Uvéite antérieure chronique granulomateuse Choroïdite disséminée avec vitrite Œdème maculaire kystoïde



Figure 2: Photo du segment antérieur illustrant l'hypopyon résorbé et une chambre antérieure claire.

Discussion

Le diagnostic différentiel dans le cas de ce patient a inclus des étiologies infectieuses et non infectieuses de l'uvéite, ce qui comprend la positivité à l'antigène HLA-B27, le virus de l'herpès simplex, la maladie de Behçet, la sarcoïdose, la toxoplasmose, le virus zona de la varicelle (VZV), la syphilis et la tuberculose. Même si elles sont moins probables, la sclérose en plaques, la maladie de Lyme et Bartonella auraient aussi pu faire partie de la liste des diagnostics différentiels. Le Tableau 3

présente une brève description de chaque étiologie.

Le patient a été diagnostiqué initialement avec une uvéite non-granulomateuse liée tentativement au HLA-B27, mais l'hypopyon et l'uvéite intermédiaire étaient atypiques. Ramsay et Lightman (2001) ont classifié les causes de l'hypopyon en causes non infectieuses, agents infectieux, néoplasmes et troubles de la cornée.²⁶ Le Tableau 4 présente le diagnostic différentiel le plus courant de l'hypopyon, de l'uvéite antérieure et de l'uvéite intermédiaire.

Tableau 4 : Diagnostic différentiel le plus courant d'hypopyon, d'uvéite antérieure et d'uvéite intermédiaire

HYPOPYON	UVÉITE ANTÉRIEURE	UVÉITE INTERMÉDIAIRE
<ul style="list-style-type: none"> ● Non infectieux <ul style="list-style-type: none"> HLA-B27 Maladie de Behçet Spondyloarthropathie Iatrogène/Néoplasme/Traumatisme ● Infectieux <ul style="list-style-type: none"> Endophthalmitis endogène Toxoplasmose Syphilis Maladie de Hansen Brucellose Virus de l'herpès simplex Kératite ● Autre 	<ul style="list-style-type: none"> ● Idiopathique ● HLA-B27 ● Arthrite réactionnelle (Reiter) ● Spondylite ankylosante ● Sarcoïdose ● Syphilis ● Virus de l'herpès simplex ● Traumatisme ● Syndrome de Posner-Schlossman ● Néoplasme <ul style="list-style-type: none"> Lymphome, leucémie, rétinoblastome ● Autre 	<ul style="list-style-type: none"> ● Idiopathique ● Sclérose en plaques ● Sarcoïdose ● Maladie de Lyme

Dans les cas d'infections et d'inflammations intraoculaires, l'hypopyon est constitué en grande partie de débris de tissus, de fibrine, de sous-produits de l'inflammation et de leucocytes, et signifie qu'il y a une inflammation intraoculaire sévère du segment antérieur.⁶ Une étude de Zaidi et al indique que la présence d'hypopyon est peu courante dans les cas d'uvéite, représentant seulement 8,57 patients qui ont une uvéite et fait son apparition chez quelque 8,57 patients par 1 000 années-personne (0,86 %) même dans les pratiques de soins tertiaires de l'uvéite. Cette étude rétrospective a indiqué que l'hypopyon était plus courant chez les patients atteints d'une uvéite limitée à la chambre antérieure que chez ceux qui avaient également une uvéite intermédiaire diagnostiquée, mais presque aussi fréquent chez les patients qui avaient une uvéite postérieure ou une panuvéite. Les facteurs de risque les plus courants d'hypopyon sont la maladie de Behçet et la positivité au HLA-B27, ce qui

quintuple et double environ le risque d'hypopyon respectivement. En fait, l'uvéite à hypopyon liée au HLA-B27 se manifeste dans 5,7 % de tous les cas d'uvéite et est plus courante chez les personnes caucasienes.²⁷ Même si l'hypopyon est un indicateur d'une inflammation sévère, les yeux qui le développent ne sont pas plus à risque pour des troubles visuels que ceux qui n'en développent pas. Une étude antérieure portant sur la maladie de Behçet a montré que les patients chez lesquels un hypopyon faisait son apparition étaient plus susceptibles de regagner trois lignes d'acuité visuelle lors du suivi, probablement parce que l'embrouillage associé à l'hypopyon constituait une cause réversible de perte de vision.²⁸

On diagnostique une uvéite intermédiaire lorsque l'inflammation intraoculaire atteint principalement le vitré, la région périphérique de la rétine et la pars plana ciliaris.²⁹ Il s'agit du type d'uvéite dont le suivi de rétablissement est le plus long.³⁰ Le syndrome est plus fréquent au cours de la troisième

et de la quatrième décennie.³¹ On a signalé que l'uvéite intermédiaire constitue de 1,4 à 22 % de tous les cas d'uvéite.³² Même si la majorité des cas sont d'étiologie inconnue, on a signalé un lien important entre l'uvéite intermédiaire et la sclérose en plaques, la sarcoïdose et la maladie de Lyme.^{33,34} L'uvéite intermédiaire est bilatérale dans 80 % des cas. Les principales caractéristiques cliniques sont les cellules de vitré, avec ou sans accumulation limitée ou étendue d'exsudats. L'uvéite intermédiaire constatée dans le cas décrit était atypique, car elle était monoculaire et ne présentait aucun lien avec les maladies susmentionnées.

Dans le rapport de cas décrit ici, les résultats de laboratoire du patient étaient positifs pour le HLA-B27, IgG et IgM du virus de l'herpès, ce qui indiquait une étiologie d'uvéite associée au HLA-B27 ou d'uvéite liée à l'herpès simplex.

L'uvéite aigüe liée au virus de l'herpès est habituellement secondaire à la kératite herpétique, par contre il se peut que 15% des patients n'ont pas d'atteinte cornéenne.^{35,39}

Les signes cliniques d'uvéite au virus de l'herpès comprennent les cicatrices cornéennes, l'atrophie de l'iris focalisée ou en région, les défauts de transillumination de l'iris, la présence de précipités, de synéchies postérieures, et une hausse de la PIO.³⁹⁻⁴¹ Une uvéite antérieure unilatérale combinée à une hausse de PIO est indicative d'une uvéite reliée au virus de l'herpès. Une atrophie de l'iris ainsi qu'une transillumination raffirme le diagnostic. Dans le cas décrit plus haut, ni l'iris ni la PIO étaient affectés, alors un diagnostic d'uvéite reliée à l'herpès n'était pas retenu et celui relié à une positivité de HLA-B27 était favorisé comme étiologie.

En dépit d'une présentation atypique, le pronostic était positif dans le cas de ce patient, comme dans celui de la plupart des uvéites associées au HLA-B27. L'administration de stéroïdes topiques et systémiques a permis une évolution clinique favorable et le patient a retrouvé son AV à 20/30¹ en moins de neuf semaines. Le patient avait une inflammation de l'épaule droite immédiatement avant l'apparition de son uvéite. On ne sait pas trop pour le moment si l'inflammation de l'épaule et l'uvéite constituent deux inflammations distinctes ou si elles font partie d'un seul problème systémique. Les patients qui ont une uvéite antérieure associée au HLA-B27 et des symptômes simultanés au segment postérieur présentent une incidence beaucoup plus élevée de maladies systémiques connexes, comme la spondylite ankylosante, les maladies intestinales inflammatoires ou l'arthrite réactionnelle (auparavant

appelée syndrome de Reiter).⁴² On a donc recommandé de référer le patient en rhumatologie pour exclure ces possibilités. On l'a informé que des récidives sont probables et que le processus peut faire partie d'un problème inflammatoire systémique qui peut l'obliger à prendre des stéroïdes ou à suivre une thérapie immunosuppressive chronique. Le patient n'a toutefois pas été vu au cours d'un suivi après la référence.

Uvéite associée au HLA-B27

Comme diagnostic d'uvéite spécifique le plus répandu, l'uvéite associée au HLA-B27 représente environ 13 à 17% de tous les cas d'uvéite.^{2,43} Environ 50 % des patients qui ont une uvéite antérieure aigüe sont positifs pour le HLA-B27. L'uvéite associée au HLA-B27 est trois fois plus fréquente chez les hommes.⁴⁴ La maladie fait son apparition vers l'âge de 35 ans en moyenne, même l'on en a signalé des cas chez des enfants (10 % des cas se manifestent avant l'âge de 20 ans) et chez des adultes d'âge mûr (5 % après 55 ans).¹⁰

L'uvéite antérieure aigüe (UAA) constitue la manifestation classique d'une maladie oculaire associée au HLA-B27.¹⁰ L'apparition est habituellement subite et les symptômes comprennent la photophobie, la douleur oculaire, l'épiphorie, la rougeur oculaire et des troubles de vision variés. Les cas sont en général unilatéraux, mais une récidive peut atteindre l'œil contralatérale. Même si l'inflammation est habituellement non granulomateuse, elle peut être assez sévère pour causer l'apparition d'un hypopyon ou rendre l'humeur aqueuse plasmoïde. L'UAA associée

au HLA-B27 est en fait la cause la plus courante d'uvéite à hypopyon en Amérique du Nord.²⁷ Dans les cas peu courants (moins que 25,1 %), le segment postérieur peut être atteint.⁷ Cette atteinte du segment postérieur peut inclure une vitrite, un œdème maculaire cystoïde, une papillite, une vasculite rétinienne et l'on croit qu'elle est secondaire à une inflammation du segment antérieur.^{10,45} C'est pourquoi l'uvéite associée au HLA-B27 peut être d'une gravité inhabituelle et provoquer une panuvéite, un phénomène sous-évalué.⁴⁶ Au cours d'une attaque aigüe, la PIO peut être diminuée à cause d'une baisse de production de l'humeur aqueuse du corps ciliaire.

L'augmentation de la PIO et le glaucome secondaire constituent néanmoins des complications bien reconnues à cause de la présence d'un iris bombé ou la fermeture de l'angle relié aux synéchies. Les crises d'uvéite sont en général de courte durée et disparaissent dans les trois mois. Les récidives sont courantes, mais la fréquence varie entre de multiples crises par année et des crises périodiques à une décennie ou plus d'intervalle.⁴⁶

Le traitement de l'uvéite est graduel.⁴⁷ Il vise dans l'immédiat à contrôler l'inflammation et les spasmes ciliaires et, à long terme, à s'adresser à la cause sous-jacente de l'uvéite. Il est possible de traiter les premières occurrences et les récidives sans complication au moyen de corticostéroïdes topiques comme l'acétate de predni-solone à 1 %, de teneur modérée, qui convient à de nombreux cas. Même si le risque d'effets secondaires systémiques est faible, il est possible de retrouver une

augmentation de la PIO chez 1% des patients. On peut alors utiliser une formulation plus douce comme la rimexolone et le lotoprednol.⁴⁸ L'utilisation simultanée d'un cycloplégique/mydriatique comme le sulfate d'atropine à 1 % atténue la douleur causée par le spasme ciliaire et peut dissocier les synéchies postérieures ou en prévenir l'apparition.

Les cas plus sévères peuvent obliger à combiner les traitements oraux, topiques, périoculaires ou intravitréens. Dans 24% des patients, il faut administrer un corticostéroïde systémique. Une dose de départ habituellement administrée par voie orale est de 1 mg/kg de prednisone à chaque jour. Les effets secondaires découlant d'un bref traitement aux stéroïdes systémiques sont peu fréquents mais comprennent les troubles du sommeil, la prise de poids, l'augmentation de l'appétit, des déséquilibres de comportement et autres.⁴⁹ Dans les cas extrêmes d'uvéite atteignant le segment postérieur, on peut envisager d'administrer des corticostéroïdes périoculaires ou intravitréens.⁵⁰ Les injections périoculaires (par voie transseptale, dans l'espace sous-tenonien ou par voie sous-conjonctivale) sont conçues comme des injections à effet prolongé (p. ex., acétate de triamcinolone) et sont donc efficaces pendant une période plus longue.⁵¹ Les injections intravitréennes sont efficaces pendant trois à six mois, réduisent au minimum les effets secondaires systémiques et peuvent traiter l'œdème maculaire causé par l'uvéite postérieure.⁵² Par contre, les complications oculaires (p. ex., cataractes et augmentation de la PIO) sont plus courantes avec les

injections intravitréennes qu'avec les stéroïdes systémiques.

Des cas extrêmes qui mettent la vue en danger peuvent nécessiter une intervention intermittente de stéroïdes par voie intraveineuse (IV) afin de contrôler l'inflammation plus rapidement et prévenir des dommages irréparables. Le traitement recommandé dans de tels cas est la méthylprednisolone 1 g IV par jour pendant trois jours. On passe ensuite à une thérapie orale commençant à 1 mg/kg par jour.^{47, 53, 54} Dans le cas des infections récalcitrantes, on peut envisager des stratégies sans stéroïdes comme une thérapie aux AINS (anti-inflammatoire non-stéroïdien) par voie orale ou une thérapie immunosuppressive.^{55, 56, 47}

Les patients qui prennent des stéroïdes topiques ou systémiques pendant une période prolongée (c.-à-d. pendant plus de deux semaines) doivent être sevrés sur plusieurs semaines de façon à éviter l'inflammation de rebond après l'usage topique, ou pour éviter de provoquer une crise surrénale à cause de l'arrêt brutal de l'usage de corticostéroïdes oraux. Les patients qui ont besoin d'une thérapie immunosuppressive devraient suivre leur traitement sans réduire la dose afin d'éviter toute récidive, qui peut être difficile à contrôler.

L'évaluation d'une présence du facteur de HLA-B27 chez les patients souffrant d'uvéite peut être utile, afin d'identifier les cas reliés à des maladies systémiques. Environ 70 % des patients qui ont une uvéite associée au HLA-B27 auront une spondyloarthropathie séronégative (facteur rhumatoïde) associée et l'on n'aura pas posé de diagnostic

chez environ 50 % d'entre eux, ou le diagnostic aurait été erroné. La spondyloarthropathie négative inclut la spondylite ankylosante, l'arthrite réactionnelle (syndrome de Reiter), le rhumatisme psoriasique et l'arthrite associée à la maladie intestinale inflammatoire. L'incidence de ces maladies systémiques est beaucoup plus élevée chez les patients qui ont des manifestations au segment postérieur.^{42, 57} En outre, de 30 à 90 % des patients qui ont une uvéite associée au HLA-B27 ont aussi une maladie articulaire associée.⁵⁸ Par conséquence, il est important d'évaluer la présence du facteur HLA-B27 afin de pouvoir améliorer la prise en charge globale du patient.

Résumé

Dans ce rapport de cas, on a posé un diagnostic final d'uvéite associée au HLA-B27 après une évaluation clinique et des analyses de laboratoire détaillées en dépit de la manifestation atypique. L'hypopyon et l'uvéite antérieure et intermédiaire associée ont été traités avec succès au moyen de stéroïdes topiques et systémiques. Ce cas constitue un rappel important : même s'ils sont peu courants, l'hypopyon et l'atteinte du segment postérieur peuvent être présents dans un cas d'uvéite associée au HLA-B27 et peuvent même atteindre les personnes âgées. De plus, il importe d'inclure une évaluation détaillée des segments antérieurs et postérieurs devant toute présentation d'œil rouge douloureux afin d'avoir un profil clinique plus complet.

Remerciements

Nous remercions Gerald Abruzzese, OD, du Syracuse Veteran's Medical Center, de son aide et de ses conseils.

Ce rapport était présenté en partie au congrès annuel de l'Académie américaine en optométrie (AAO) à Boston le 14 octobre 2011

Références

1. Rothova A, Suttorp-van Schulten MS, Frits Treffers W, Kijlstra A. Causes and frequency of blindness in patients with intraocular inflammatory disease. *Br J Ophthalmol* 1996;80(4):332-6.
2. McCannel CA, Holland GN, Helm CJ, et al. Causes of uveitis in the general practice of ophthalmology. UCLA Community-Based Uveitis Study Group. *Am J Ophthalmol* 1996;121(1):35-46.
3. Brewerton DA, Caffrey M, Nicholls A, et al. Acute anterior uveitis and HLA-A 27. *Lancet* 1973;302(7836):994-6.
4. Feltkamp TE. Ophthalmological significance of HLA associated uveitis. *Eye (Lond)* 1990;4 (Pt 6):839-44.
5. Wakefield D, Chang JH, Amjadi S, et al. What is new HLA-B27 acute anterior uveitis? *Ocul Immunol Inflamm* 2011;19(2):139-44.
6. Zaidi AA, Ying GS, Daniel E, et al. Hypopyon in patients with uveitis. *Ophthalmology* 2010;117(2):366-72.
7. Kase S, Namba K, Horie Y, et al. Repeated exacerbations of ocular inflammation with vitreous hemorrhage in a patient with HLA-B27 associated uveitis. *J Med Invest* 2007;54(3-4):350-3.
8. Dodds EM, Lowder CY, Meisler DM. Posterior segment inflammation in HLA-B27+ acute anterior uveitis: clinical characteristics. *Ocul Immunol Inflamm* 1999;7(2):85-92.
9. Rothova A. Comment on 'Posterior segment inflammation in HLA-B27+ acute anterior uveitis: clinical characteristics'. *Ocul Immunol Inflamm* 2000;8(1):73-5.
10. Tay-Kearney ML, Schwam BL, Lowder C, et al. Clinical features and associated systemic diseases of HLA-B27 uveitis. *Am J Ophthalmol* 1996;121(1):47-56.
11. Schumacher HR, Jr. The pathogenesis of gout. *Cleve Clin J Med* 2008;75 Suppl 5:S2-4.
12. Dawson CR, Togni B. Herpes simplex eye infections: clinical manifestations, pathogenesis and management. *Surv Ophthalmol* 1976;21(2):121-35.
13. Santos C. Herpes simplex uveitis. *Bol Asoc Med P R* 2004;96(2):71-4, 7-83.
14. Mendes D, Correia M, Barbedo M, et al. Behcet's disease--a contemporary review. *J Autoimmun* 2009;32(3-4):178-88.
15. Cozier YC, Berman JS, Palmer JR, et al. Sarcoidosis in black women in the United States: data from the Black Women's Health Study. *Chest* 2011;139(1):144-50.
16. Uyama M. Uveitis in sarcoidosis. *Int Ophthalmol Clin* 2002;42(1):143-50.
17. Jones NP. Sarcoidosis and uveitis. *Ophthalmol Clin North Am* 2002;15(3):319-26, vi.
18. Park YH, Han JH, Nam HW. Clinical features of ocular toxoplasmosis in Korean patients. *Korean J Parasitol* 2011;49(2):167-71.
19. Bornand JE, de Gottrau P. Uveitis: is ocular toxoplasmosis only a clinical diagnosis? *Ophthalmologica* 1997;211(2):87-9.
20. Thean JH, Hall AJ, Stawell RJ. Uveitis in Herpes zoster ophthalmicus. *Clin Experiment Ophthalmol* 2001;29(6):406-10.
21. Hong MC, Sheu SJ, Wu TT, Chuang CT. Ocular uveitis as the initial presentation of syphilis. *J Chin Med Assoc* 2007;70(7):274-80.
22. Reddy S, Cubillan LD, Hovakimyan A, Cunningham ET, Jr. Inflammatory ocular hypertension syndrome (IOHS) in patients with syphilitic uveitis. *Br J Ophthalmol* 2007;91(12):1610-2.
23. Tucker JD, Li JZ, Robbins GK, et al. Ocular syphilis among HIV-infected patients: a systematic analysis of the literature. *Sex Transm Infect* 2011;87(1):4-8.
24. Lutchman C, Weisbrod DJ, Schwartz CE. Diagnosis and management of syphilis after unique ocular presentation.
- Le Médecin de famille canadien 2011;57(8):896-9.
25. Varma D, Anand S, Reddy AR, et al. Tuberculosis: an under-diagnosed etiological agent in uveitis with an effective treatment. *Eye (Lond)* 2006;20(9):1068-73.
26. Ramsay A, Lightman S. Hypopyon uveitis. *Surv Ophthalmol* 2001;46(1):1-18.
27. D'Alessandro LP, Forster DJ, Rao NA. Anterior uveitis and hypopyon. *Am J Ophthalmol* 1991;112(3):317-21.
28. Nussenblatt RB. Uveitis in Behcet's disease. *Int Rev Immunol* 1997;14(1):67-79.
29. Jabs DA, Nussenblatt RB, Rosenbaum JT. Standardization of uveitis nomenclature for reporting clinical data. Results of the First International Workshop. *Am J Ophthalmol* 2005;140(3):509-16.
30. Bloch-Michel E. Opening address: intermediate uveitis. *Dev Ophthalmol* 1992;23:1-2.
31. Chan SM, Hudson M, Weis E. Anterior and intermediate uveitis cases referred to a tertiary centre in Alberta. *Journal canadien d'ophtalmologie* 2007;42(6):860-4.
32. Babu BM, Rathinam SR. Intermediate uveitis. *Indian J Ophthalmol* 2010;58(1):21-7.
33. Zierhut M, Foster CS. Multiple sclerosis, sarcoidosis and other diseases in patients with pars planitis. *Dev Ophthalmol* 1992;23:41-7.
34. Breeveld J, Rothova A, Kuiper H. Intermediate uveitis and Lyme borreliosis. *Br J Ophthalmol* 1992;76(3):181-2.
35. Miserocchi E, Waheed NK, Dios E, et al. Visual outcome in herpes simplex virus and varicella zoster virus uveitis: a clinical evaluation and comparison. *Ophthalmology* 2002;109(8):1532-7.
36. Liesegang TJ. Ocular herpes simplex infection: pathogenesis and current therapy. *Mayo Clin Proc* 1988;63(11):1092-105.
37. Schacher S, Garweg JG, Russ C, Bohnke M. [Diagnosis of herpetic uveitis and keratouveitis]. *Klin Monbl Augenheilkd* 1998;212(5):359-62.
38. Rodriguez A, Power WJ, Neves RA, Foster CS. Recurrence rate of

- herpetic uveitis in patients on long-term oral acyclovir. Doc Ophthalmol 1995;90(4):331-40.
39. Liesegang TJ. Herpes simplex virus epidemiology and ocular importance. Cornea 2001;20(1):1-13.
40. Kaye S, Choudhary A. Herpes simplex keratitis. Prog Retin Eye Res 2006;25(4):355-80.
41. Liesegang TJ. Classification of herpes simplex virus keratitis and anterior uveitis. Cornea 1999;18(2):127-43.
42. Kataria RK, Brent LH. Spondyloarthropathies. Am Fam Physician 2004;69(12):2853-60.
43. Rodriguez A, Calonge M, Pedroza-Seres M, et al. Referral patterns of uveitis in a tertiary eye care center. Arch Ophthalmol 1996;114(5):593-9.
44. Power WJ, Rodriguez A, Pedroza-Seres M, Foster CS. Outcomes in anterior uveitis associated with the HLA-B27 haplotype. Ophthalmology 1998;105(9):1646-51.
45. Rodriguez A, Akova YA, Pedroza-Seres M, Foster CS. Posterior segment ocular manifestations in patients with HLA-B27-associated uveitis. Ophthalmology 1994;101(7):1267-74.
46. Smith JR. HLA-B27-associated uveitis. Ophthalmol Clin North Am 2002;15(3):297-307.
47. Lee FF, Foster CS. Pharmacotherapy of uveitis. Expert Opin Pharmacother 2010;11(7):1135-46.
48. Tripathi RC, Parapuram SK, Tripathi BJ, et al. Corticosteroids and glaucoma risk. Drugs Aging 1999;15(6):439-50.
49. Noone T. An overview of steroid use and its potential side-effects. Nurs Times 2006;102(17):24-7.
50. Ferrante P, Ramsey A, Bunce C, Lightman S. Clinical trial to compare efficacy and side-effects of injection of posterior sub-Tenon triamcinolone versus orbital floor methylprednisolone in the management of posterior uveitis. Clin Experiment Ophthalmol 2004;32(6):563-8.
51. Riordan-Eva P, Lightman S. Orbital floor steroid injections in the treatment of uveitis. Eye (Lond) 1994;8 (Pt 1): 66-9.
52. Antcliff RJ, Spalton DJ, Stanford MR, et al. Intravitreal triamcinolone for uveitic cystoid macular edema: an optical coherence tomography study. Ophthalmology 2001;108(4):765-72.
53. Yalcindag FN, Can E, Ozdemir O. Intravenous methylprednisolone pulse therapy for acute posterior segment uveitis attacks in Behcet's disease. Ann Ophthalmol (Skokie) 2007;39(3):194-7.
54. Wakefield D, McCluskey P, Penny R. Intravenous pulse methylprednisolone therapy in severe inflammatory eye disease. Arch Ophthalmol 1986;104(6):847-51.
55. Okada AA. Immunomodulatory therapy for ocular inflammatory disease: a basic manual and review of the literature. Ocul Immunol Inflamm 2005;13(5): 335-51.
56. Bom S, Zamiri P, Lightman S. Use of methotrexate in the management of sight-threatening uveitis. Ocul Immunol Inflamm 2001;9(1):35-40.
57. Monnet D, Breban M, Hudry C, et al. Ophthalmic findings and frequency of extraocular manifestations in patients with HLA-B27 uveitis: a study of 175 cases. Ophthalmology 2004;111(4): 802-9.
58. Rosenbaum JT. Acute anterior uveitis and spondyloarthropathies. Rheum Dis Clin North Am 1992;18(1):143-51.

NOVA SOUTHEASTERN UNIVERSITY
College of Optometry, Office of Continuing Education

Last Call TPA

BOARD REVIEW COURSE

Subject: 100 Hour

THERAPEUTIC PHARMACEUTICAL AGENTS

Certification Course

Dates: July 8 -18, 2012

Hours can be customized
for state/province requirements
or board certification review.

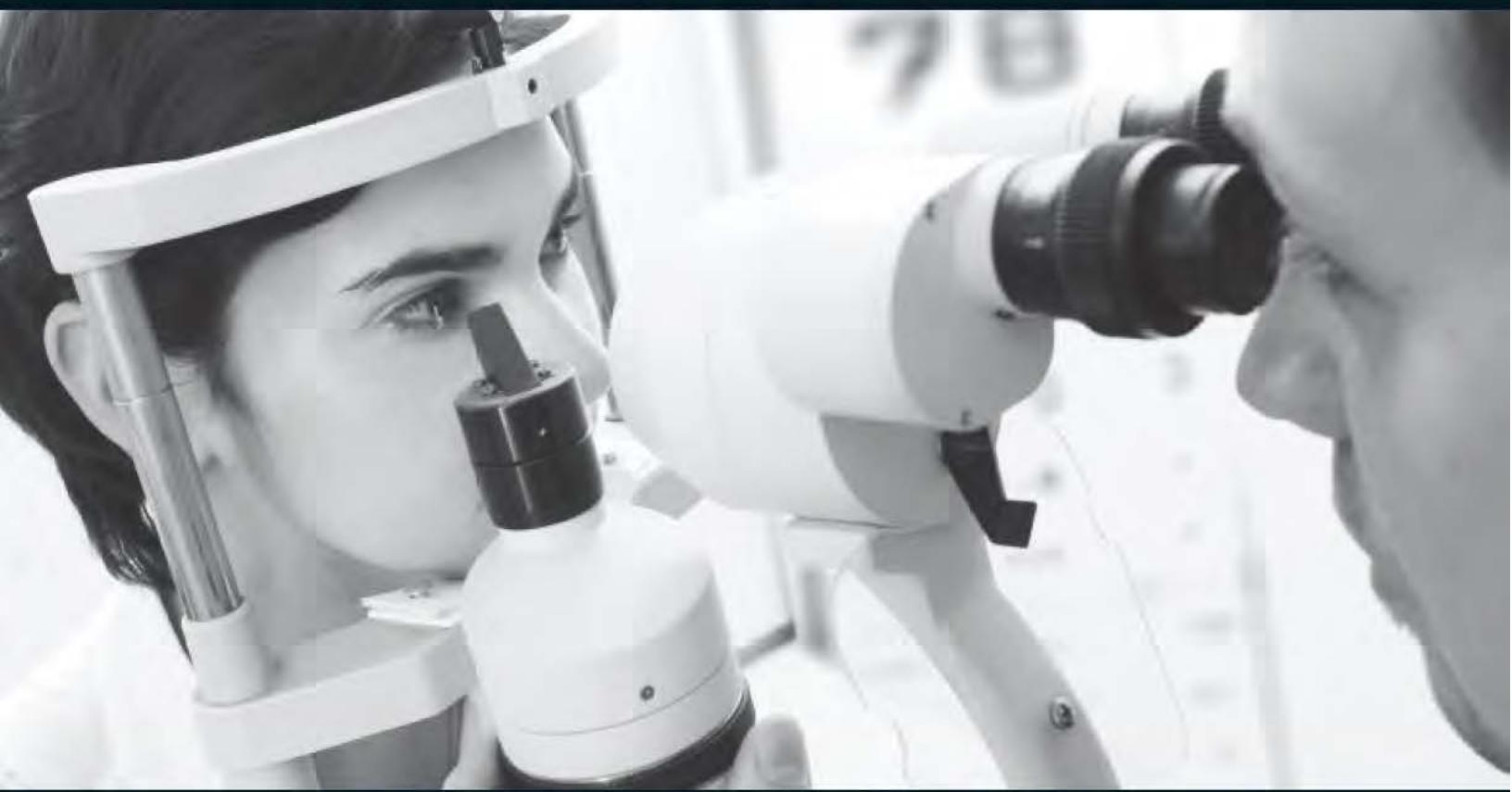
For further information and to register:
Web: optometry.nova.edu/ce Tel: (954) 262-4224

Notice of Accreditation/Nondiscrimination
Nova Southeastern University admits students of any age, race, color, sexual orientation, pregnancy status, religion or creed, nondisqualifying disability, and national or ethnic origin. Nova Southeastern University is accredited by the Commission on Colleges of the Southern Association of Colleges and Schools (1866 Southern Lane, Decatur, Georgia, 30033-4097; telephone number 404-679-4501) to award associate's, bachelor's, master's, educational specialist, and doctoral degrees.

COPE APPROVAL PENDING

NOVA SOUTHEASTERN UNIVERSITY
College of Optometry

The majority of people who use tobacco **want to quit.**



In less than 3 minutes you can make a difference in your patient's health.

Follow the 5As:

Ask your patient if he/she uses tobacco.

Advise your patient to quit.

Assess your patient's readiness to quit.

Assist your patient to quit.

Arrange a follow-up with Smokers' Helpline.

For free materials and training on the 5As visit **YouCanMakeItHappen.ca**

smokers' helpline

CONNECT TO QUIT
smokershelpline.ca
1 877 513-5333

Contact Your
Local Public
Health Unit for
materials and
support

Join Your
Local Cessation
Community
of Practice
to network
with others

**SMOKE
FREE
ONTARIO**



The boxfish inspired a more aerodynamic car.



The butterfly inspired more efficient LED technology.



The eye inspired the latest advance in lens care.

Bio-inspiration.
Nature's best ideas.

BAUSCH + LOMB