The PHQ-2 as a Screening Tool for Clinical Depression

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The results reported herein point to a considerably higher prevalence of depression amongst optometric patients than might be suspected based on a survey of optometric practices that found 0.41% of patients with this condition.
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It was interesting and gratifying to see that optometrists make the list of the top 10 highest-paying, least stressful jobs in the USA. Among health professionals, apparently only orthodontists rate higher; the others in the top five are computer and information systems managers, law professors and physicists. Curiously, professional astronomers are ninth, so even in an alternate universe I might have enjoyed a less stressful career...

Stress is just one of the many possible causes of mental illness, and many of our patients and friends probably talk about how much they experience, and how they cope. There has been much in the news media lately about the importance of mental health – Bell Media’s annual “Let’s Talk” day is a good example of the push in Canadian society to remove the stigma of mental illness and encourage those affected to seek help.

As primary care practitioners, we optometrists can also help patients who are facing mental health issues. Our lead article by Drs. DellaBella, Schwartz and Nehmad discusses the efficacy of the PHQ-2 screening tool to identify patients who may benefit from a referral to a mental health specialist for clinical depression. The critical question is, do we have the time, inclination and professional duty to add this screening test to our examination routines, and if so, does it fit with our regulated scope of practice?

Our profession has changed immensely over the nearly four decades that I have been practicing. It is not inconceivable that screening for the mental health of our patients will become part of everyday activities.

REFERENCES

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Il est intéressant et gratifiant pour les optométristes de constater que leur métier figure sur la liste des 10 emplois les mieux rémunérés et les moins stressants aux États-Unis. Parmi les professionnels de la santé, seuls les orthodontistes semblent mieux se classer; les autres emplois dans les cinq premiers sont ceux des gestionnaires de systèmes informatiques, des professeurs de droit et des physiciens. Curieusement, les astronautes professionnels sont neuvièmes. Donc, même dans un univers alternatif, j’aurais peut-être pu avoir une carrière moins stressante...

Le stress n’est qu’une des nombreuses causes possibles de maladie mentale, et beaucoup de nos patients et amis parlent probablement de leur expérience et de la façon dont ils s’en sortent. Les médias ont beaucoup parlé récemment de l’importance de la santé mentale. La journée annuelle « Cause pour la cause » de Bell Média est un bon exemple des efforts déployés par la société canadienne pour éliminer la stigmatisation de la maladie mentale et encourager les personnes touchées à demander de l’aide.

En tant que praticiens des soins primaires, nous, les optométristes, pouvons aussi aider les patients qui ont des problèmes de santé mentale. Notre article principal des Drs DellaBella, Schwartz et Nehmad traite de l’efficacité de l’outil de dépistage PHQ-2 pour reconnaître les patients qui pourraient être adressés à un spécialiste de la santé mentale pour dépression clinique. La question essentielle est de savoir si nous avons le temps, le désir et le devoir professionnel d’ajouter ce test de dépistage à nos examens et, dans l’affirmative, s’il correspond à notre champ d’exercice réglementé?

Notre profession a énormément changé depuis le début de ma pratique, il y a près de quatre décennies. Il n’est pas inconcevable que le dépistage de problèmes de la santé mentale devienne partie intégrante de nos activités quotidiennes.

RÉFÉRENCES

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* In 2 of 4 clinical trials, Xiidra improved eye dryness in 12, 6, and as early as 2 weeks.

In OPUS-3 (Study 4; N = 711), a significant difference in mean change from baseline to Day 84 in EDS favouring Xiidra (−37.7) over vehicle (−30.5) was observed (p = 0.0007). Significant improvement in mean change from baseline of

1 Comparative clinical significance has not been established.


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EDS was seen in the Xiidra group over vehicle for key secondary endpoints at Day 14 (−22.7 vs. −14.9, p < 0.0001) and Day 42 (−33.0 vs. −23.7, p < 0.0001).\textsuperscript{1,2} In OPUS-2 (Study 3; N = 718), a statistically significant difference in mean change from baseline to Day 84 in EDS (co-primary symptoms endpoint) favouring Xiidra (−35.3) over vehicle (−22.8) was observed (p < 0.0001). A post hoc analysis of mean change from baseline in EDS for secondary endpoints showed a treatment effect as early as Day 14 for Xiidra over vehicle (−19.7 vs. −13.1) and at Day 42 (−28.3 vs. −18.2).\textsuperscript{1,3} In OPUS-2, Xiidra treatment did not result in a statistically significant difference for the co-primary sign endpoint (ICSS).\textsuperscript{1,3}

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The PHQ-2 as a Screening Tool for Clinical Depression in a Primary Eye-Care Clinic

ABSTRACT

Purpose: Screening tests for clinical depression, a highly prevalent and often disabling condition, have not been investigated in primary-care eye settings. The purpose of the present study was to determine the percent of patients in an urban primary-care eye clinic who fail the PHQ-2 screening tool. The PHQ-2 is an ultra-short screener consisting of 2 items regarding mood and anhedonia.

Methods: The two-question PHQ-2 was administered (as part of a larger questionnaire that included data on gender, age, and ethnicity) to patients seated in the Primary Care Clinic of the SUNY College of Optometry [University Eye Center] in Manhattan, NY. A total of 739 surveys were completed over a two-month period, with a completion rate of 69%. All surveys were completed anonymously, and unfinished surveys were not included in the final data set.

Results: The demographics collected in this study mirror those of the population that this clinic serves; overall very diverse, with good representation from each age group. Thirteen percent of the sample received a score of 3 or higher, the standard cutoff score for failure of the PHQ-2.

Conclusions: The failure rate on the PHQ-2 in a primary eye-care, urban population approaches that found in general medical practice, suggesting similar rates of clinical depression. Thus, the PHQ-2 may be a beneficial tool for screening for depression, however, it is important to follow-up with a referral to a mental health specialist.

KEYWORDS

depression, dysthymia, PHQ-2, screening, primary eye care

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The lifetime prevalence of major depression may be as high as 16%.1 About 5 – 10% of patients in primary care settings suffer from major depression, while dysthymia, a chronic low-grade depression, affects 2 to 4% of this population.2 These conditions result in significant suffering, morbidity and mortality.3,4 Major depression is a leading cause of disability in adults, and is expected to soon rank second only to heart disease worldwide.5,6 The only chronic condition that is more prevalent in general medical practice is hypertension.7,8 Depressed patients are more likely to attempt or commit suicide.9

Despite the increasing availability of effective treatments,10,11 both medical and psychological, it is estimated that as few as 22% of patients with major depressive disorder receive appropriate care.1 Since symptoms may not be apparent to the practitioner during routine primary-care medical encounters, there is great interest in developing screening surveys that can be employed in such settings to effectively screen for this disease.5,10,15 To encourage the use of these screening tools in primary-care medical practice, where time constraints make efficiency a major consideration, the trend has been toward the use of shorter instruments, including ultra-short (one-, two-, three- and four-item) surveys.5,6,10,18,22

The most widely studied of the ultra-short screening surveys, the PHQ-2, consists of the first two items of the longer nine-item Patient Health Questionnaire (PHQ-9).6,17 The two questions, which are based on symptoms specified by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), concern mood and anhedonia (Table 1).19 The PHQ-2 has been studied in various clinical populations, including primary care, geriatric, cardiology, obstetrics-gynecology and general medical.6,10,18,27,29,32 The most detailed studies with primary-care populations found sensitivities ranging from 79 – 83% and specificity ranging from 86 – 92%.6,23,25 These findings point to its possible utility as a tool for screening patients seeking primary care services.25

Table 1: Patient Health Questionnaire-2 (PHQ-2)

<table>
<thead>
<tr>
<th>Over the last 2 weeks, how often have you been bothered by any of the following problems?</th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Little interest or pleasure in doing things</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>b. Feeling down, depressed, or hopeless</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>

PHQ-2 Copyright© 1999 Pfizer Inc. All rights reserved. Reproduced with permission.

Few data are available on the prevalence of clinical depression among patients seeking eye care. In a survey of optometric practices, Soroka et al. determined that only 0.41% of optometric patients had a diagnosis of depression, considerably below what would be expected based on the condition’s prevalence in the general population and primary-care medical settings.23,25 This value, which is based on case histories, does not take into account undiagnosed patients or those unwilling to reveal a diagnosis of depression.

The current study was undertaken to determine the failure rate for the PHQ-2 when administered in a large urban primary-care eye clinic. While the results obtained with a screening device do not indicate the prevalence of clinical depression, they provide a basis for comparison with the findings in other primary-care settings.

METHODS

Subjects and Procedure

Patients seated in the waiting area of the Primary Care Clinic of the SUNY, College of Optometry, University Eye Center (UCE) were individually asked by one of the investigators if they would be interested in completing a short survey. The UEC, which is located in midtown Manhattan, provides eye-care services to a diverse urban population.

If the patient agreed, he/she was given the survey and a consent statement along with an envelope in which to place the survey once it was completed. Both the top and bottom of the one-page survey displayed the statement “DO NOT WRITE OR SIGN YOUR NAME ON THIS FORM.” A statement asking the subject to read the accompanying consent prior to completing the survey was also printed on the survey form, as were statements that “answers to the questions on the survey are anonymous” and “no one, including the researchers, will know how you answered the survey questions.” The envelopes containing the surveys were subsequently collected by the investigator, who placed them in a bag in the patient’s presence. Minimum age for participation in the study was 18 years. The experimental protocol was approved by the SUNY State College of Optometry Institutional Review Board.
A total of 739 surveys were completed over a period of about two months. Based on the final 797 potential subjects who were approached, the survey completion rate was 69%. About 16% refused to take the survey, and 2% of the returned surveys were incomplete. The remaining potential subjects could not complete the survey due to language barriers (6%), disability (1%), because they were called in for their exam (3%) or because they had been dilated and were unable to clearly see the survey items (3%).

Survey Instrument
The survey consisted of 10 items, with the first two from the PHQ-2 (Table 1). Included in the remaining items were questions related to demographics (age, gender and ethnicity). A Likert scale was used for the PHQ-2. Data for each completed survey were entered into an SPSS database for analysis.

RESULTS
Table 2 summarizes the demographic characteristics of our sample, which appear to be representative of the population served by the clinic. Results of the PHQ-2 are given in Table 3, which shows that cutoff values of 2, 3, and 4 gave failure rates of 0.29, 0.13 and 0.07 respectively. A score of 3 or higher is normally considered a failing score.23

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Sample (n, %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td></td>
</tr>
<tr>
<td>18-30</td>
<td>198 (26.8%)</td>
</tr>
<tr>
<td>31-45</td>
<td>156 (21.1%)</td>
</tr>
<tr>
<td>46-60</td>
<td>230 (31.1%)</td>
</tr>
<tr>
<td>61-75</td>
<td>132 (17.9%)</td>
</tr>
<tr>
<td>76 or older</td>
<td>23 (3.1%)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>264 (35.7%)</td>
</tr>
<tr>
<td>Female</td>
<td>474 (64.1%)</td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>69 (9.3%)</td>
</tr>
<tr>
<td>Black</td>
<td>227 (30.7%)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>278 (37.6%)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>117 (15.8%)</td>
</tr>
<tr>
<td>Native American</td>
<td>5 (0.7%)</td>
</tr>
<tr>
<td>Mixed</td>
<td>43 (5.8%)</td>
</tr>
</tbody>
</table>

Table 2: Sample demographics

<table>
<thead>
<tr>
<th>PHQ-2 Total Score</th>
<th>Frequency</th>
<th>Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>12</td>
<td>1.6</td>
<td>1.6</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>1.1</td>
<td>2.7</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>4.6</td>
<td>7.3</td>
</tr>
<tr>
<td>3*</td>
<td>40</td>
<td>5.4</td>
<td>12.7</td>
</tr>
<tr>
<td>2</td>
<td>118</td>
<td>16.0</td>
<td>28.7</td>
</tr>
<tr>
<td>1</td>
<td>89</td>
<td>12.0</td>
<td>40.7</td>
</tr>
<tr>
<td>0</td>
<td>438</td>
<td>59.3</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table 3: Frequency distribution of PHQ-2 scores

*Standard cut-off point

DISCUSSION
A limitation of the current study is that the actual prevalence of clinical depression in the sample was not determined. To do so would have required a structured diagnostic clinical interview of all subjects. The most frequently used of these is the Structured Clinical Interview for DSM-IV (SCID), a lengthy and tedious process that was neither feasible nor appropriate with our sample.26 The PHQ-2 failure rate, however, has been determined in patient samples with a known prevalence of clinical depression. These data may be used to infer the prevalence of clinical depression when the PHQ-2 failure rate is known.24,25
A cutoff score of 3, the standard cutoff score for the PHQ-2, resulted in a 13% failure rate in our sample taken from an urban primary-care eye clinic.21,25 In a sample derived from primary-care medical and obstetrics-gynecology clinics that had a 7% prevalence of depression as determined with structured interviews, Kroenke et al. found that 15.2% scored 3 or higher on the PHQ-2.23 The PHQ-2 failure rates for the primary-care eye sample in the current study and medical/obstetrics-gynecological samples in previous studies are comparable, suggesting a similar prevalence of clinical depression.

The results reported herein point to a considerably higher prevalence of depression amongst optometric patients than might be suspected based on a survey of optometric practices that found 0.41% of patients with this condition.24,26 This latter figure reflects reliance on case history to determine if depression is present. The 13% PHQ-2 failure rate found in our sample is similar to that in primary-care medical practices, where 7% of the patients were diagnosed with depression, leading one to suspect that the prevalence in primary-care optometric practices, particularly urban practices with demographics similar to ours, approaches 7%.27

The practical application of these findings to eye-care is complex. Meta-analysis of two- and three-question screening instruments revealed a negative predictive value as low as 93%, indicating that up to 7% of subjects who pass the test are clinically depressed.28 Of greater practical import is that, despite its relatively high specificity, most of the patients who fail the PHQ-2 will not meet the diagnostic criteria for major depression or dysthymia.23 Two- and three- question screeners have a positive predictive value of about 0.4, meaning that only four of ten patients who fail the screener are clinically depressed.23,28

If the PHQ-2 was used in isolation to screen to depression, without follow-up, it would result in an unjustifiably high over-referral rate. For this reason, it has been recommended that ultra-short screening instruments should only be administered when failing scores can be followed-up with a diagnostic interview or longer survey of higher specificity, such as the PHQ-9, which has additional items specific to the DSM-IV diagnostic criteria, including items related to suicidal ideations.23,28 Patients who fail the more comprehensive screening can then be referred for a mental health evaluation. This two-stage screening may be practicable in eye clinics situated in multidisciplinary settings.

In summary, results with the PHQ-2 screening instrument suggest that the prevalence of clinical depression in the primary-care patient population of an urban eye-care clinic may approach that of medical primary-care settings. The availability of appropriate follow-up, however, is of upmost importance when using this screening tool in eye-care practices.

ACKNOWLEDGEMENTS
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Le PHQ-2 comme outil de dépistage de la dépression clinique dans une clinique de soins oculovisuels de première ligne

RÉSUMÉ

Objet : Les tests de dépistage de la dépression clinique, une maladie très répandue et souvent invalidante, n’ont pas été étudiés dans les établissements de soins oculovisuels de première ligne. L’objet de la présente étude était de déterminer le pourcentage de patients d’une clinique de soins oculovisuels de première ligne en milieu urbain qui répondent positivement à l’outil de dépistage PHQ-2. Le PHQ-2 est un outil de dépistage ultra-court composé de deux questions concernant l’humeur et l’anhédolie.

Méthodes : Les deux questions du PHQ-2 ont été posées (dans le cadre d’un questionnaire plus vaste qui comprenait des données sur le sexe, l’âge et l’origine ethnique) aux patients assis à la clinique de soins de première ligne du College of Optometry de la SUNY [University Eye Center] à Manhattan (New York). Au total, 739 questionnaires ont été remplis sur une période de deux mois, avec un pourcentage de réponse de 69 %. Tous les questionnaires ont été remplis de façon anonyme, et les questionnaires inachevés n’ont pas été inclus dans l’ensemble de données final.

Résultats : Les données démographiques recueillies dans le cadre de cette étude reflètent celles de la population desservie par cette clinique ; très diversifiée dans l’ensemble, avec une bonne représentation de chaque groupe d’âge. Treize pour cent de l’échantillon a reçu une cote de 3 ou plus, soit le seuil standard pour un résultat positif au PHQ-2.

Conclusions : Le taux de résultats positifs au PHQ-2 obtenu chez la population urbaine en soins oculovisuels de première ligne s’approche de celui que l’on trouve en médecine générale, ce qui suggère des taux semblables de dépression clinique. Par conséquent, le PHQ-2 peut être un outil utile pour le dépistage de la dépression, mais il est important de faire un suivi en aiguillant les personnes vers un spécialiste de la santé mentale.

MOTS CLÉS
dépression, dysthymie, PHQ-2, dépistage, soins oculovisuels de première ligne

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La prévalence à vie de la dépression majeure peut atteindre 16 %.1 Environ 5 à 10 % des patients en contexte de soins de première ligne souffrent de dépression majeure, tandis que la dysthymie, une dépression chronique moins grave, touche de 2 à 4 % de cette population.2 Ces maladies entraînent une souffrance, une morbidité et une mortalité importantes.3 La dépression majeure est l’une des principales causes d’invalidité chez les adultes et devrait bientôt se classer au deuxième rang derrière les maladies cardiaques dans le monde.4,5 L’hypertension est le seul problème chronique plus répandu en médecine générale.6 Les patients déprimés sont plus susceptibles de tenter de se suicider ou d’y parvenir.7

Malgré la disponibilité croissante de traitements efficaces,8 à la fois médicaux et psychologiques, on estime que seulement 22 % des patients atteints d’un trouble dépressif majeur reçoivent des soins appropriés.3 Étant donné que les symptômes peuvent ne pas être évidents pour le praticien pendant les rencontres médicales de routine en soins de première ligne, on s’intéresse beaucoup à l’élaboration d’enquêtes de dépistage qui peuvent être utilisées dans de tels contextes pour dépister efficacement cette maladie.9,10,11 Afin d’encourager l’utilisation de ces outils de dépistage dans les soins médicaux de première ligne, lorsque les contraintes de temps font de l’efficacité une considération majeure, la tendance a été à l’utilisation d’instruments plus courts, y compris des outils de dépistage ultracourts (une, deux, trois et quatre questions).11,12,13,14,15

Le questionnaire de dépistage ultracourt le plus souvent étudié, le PHQ-2, comprend les deux premières questions du Questionnaire sur la santé du patient (Patient Health Questionnaire; PHQ-9).16,17 Les deux questions, qui sont fondées sur les symptômes précisés dans le Manuel diagnostique et statistique des troubles mentaux, quatrième édition (DSM-IV), portent sur l’humeur et l’anhedonie (tableau 1).18 Le PHQ-2 a été étudié dans diverses populations cliniques, y compris les soins de première ligne, la gériatrie, la cardiologie, l’obstétrique-gynécologie et la médecine générale.19,20,21,22,23,24 Les études les plus détaillées auprès des populations recevant des soins de première ligne ont révélé une sensibilité allant de 79 à 83 % et une spécificité variant de 86 à 92 %.19,20,21,22 Ces constatations indiquent son utilité possible comme outil de dépistage des patients demandant des services de soins de première ligne.25

Tableau 1 : Patient Health Questionnaire-2 (PHQ-2)

<table>
<thead>
<tr>
<th>Au cours des deux dernières semaines, à quelle fréquence avez-vous été dérangé par l’un ou l’autre des problèmes suivants?</th>
<th>Jamais</th>
<th>Plusieurs jours</th>
<th>Plus de la moitié des jours</th>
<th>Presque tous les jours</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Peu d’intérêt ou de plaisir à faire des choses</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>b. Se sentir démoralisé, déprimé ou désespéré</td>
<td>☐</td>
<td>☐</td>
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PHQ-2 Copyright © 1999 Pfizer Inc. Tous droits réservés. Reproduit avec autorisation.

Peu de données sont disponibles sur la prévalence de la dépression clinique chez les patients ayant besoin de soins oculovisuels. Dans un sondage sur les pratiques optométriques, Soroka et coll. ont déterminé que seulement 0,41 % des patients optométriques avaient reçu un diagnostic de dépression, ce qui est considérablement inférieur à ce qui est attendu compte tenu de la prévalence du problème dans la population générale et dans les milieux médicaux de soins de première ligne.24,25 Cette valeur, qui est fondée sur les antécédents de cas, ne tient pas compte des patients non diagnostiqués ou de ceux qui ne veulent pas révéler un diagnostic de dépression.

La présente étude vise à déterminer le taux de résultats positifs au PHQ-2 administré dans une grande clinique de soins oculovisuels de première ligne en milieu urbain. Bien que les résultats obtenus au moyen d’un outil de dépistage n’indiquent pas la prévalence de la dépression clinique, ils fournissent une base de comparaison avec les résultats obtenus dans d’autres contextes de soins de première ligne.

MÉTHODES

Sujets et procédure

L’un des chercheurs a demandé aux patients assis dans la salle d’attente de la clinique de soins de première ligne du University Eye Center (UEC) du College of Optometry de la SUNY, s’ils souhaitaient répondre à un court sondage. L’UEC, situé à Midtown Manhattan, offre des services de soins oculovisuels à une population urbaine diversifiée.
Si le patient avait donné son accord, on lui a remis le questionnaire et une déclaration de consentement, ainsi qu’une enveloppe dans laquelle placer le questionnaire une fois rempli. Le haut et le bas du sondage d’une page affichaient l’énoncé « NE PAS INSCRIRE VOTRE NOM SUR CE FORMULAIRE NI LE SIGNER ». Un énoncé demandant au sujet de lire le consentement joint avant de remplir le questionnaire a également été imprimé sur le formulaire de sondage, tout comme des énoncés indiquant que « les réponses aux questions du sondage sont anonymes » et « personne, y compris les chercheurs, ne saura quelles ont été vos réponses au sondage ». Les enveloppes contenant les questionnaires ont ensuite été recueillies par l’enquêteur, qui les a placées dans un sac en présence du patient. L’âge minimum pour participer à l’étude était de 18 ans. Le protocole expérimental a été approuvé par le comité d’examen de l’établissement du State College of Optometry de la SUNY.

Au total, 739 questionnaires ont été remplis sur une période d’environ deux mois. Le taux de participation au sondage était de 69 %, pour les 797 derniers sujets potentiels qui ont été abordés. Environ 16 % ont refusé de répondre au sondage et 2 % des questionnaires renvoyés étaient incomplets. Les autres sujets potentiels n’ont pas pu répondre au sondage en raison de barrières linguistiques (6 %), d’incapacité (1 %), parce qu’ils ont été convoqués pour leur examen (3 %) ou parce qu’ils avaient eu les pupilles dilatées et qu’ils étaient incapables de voir clairement les questions du sondage (3 %).

**Instrument du sondage**
Le sondage comportait 10 questions, dont les deux premières étaient celles du questionnaire PHQ-2 (tableau 1). Les autres questions portaient sur la démographie (âge, sexe et appartenance ethnique). Une échelle de Likert a été utilisée pour le PHQ-2. Les données de chaque questionnaire terminé ont été saisies dans une base de données SPSS à des fins d’analyse.

**RÉSULTATS**
Le tableau 2 résume les caractéristiques démographiques de notre échantillon, qui semblent être représentatives de la population desservie par la clinique. Les résultats au PHQ-2 sont présentés au tableau 3, qui montre que les valeurs seuils de 2, 3 et 4 ont donné des taux de dépistage positif de 0,29, 0,13 et 0,07 respectivement. Une cote de 3 ou plus est normalement considérée comme un dépistage positif.21

<table>
<thead>
<tr>
<th><strong>Tableau 2 : Données démographiques de l’échantillon</strong></th>
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<tr>
<td><strong>Données démographiques</strong></td>
</tr>
<tr>
<td><strong>Âge (a)</strong></td>
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<tr>
<td>18-30</td>
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<td>31-45</td>
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<td>46-60</td>
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<td>61-75</td>
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<td>76 ans ou plus</td>
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<td>Femme</td>
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<td><strong>Race/appartenance ethnique</strong></td>
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<td>Asiatique</td>
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<td>Noir</td>
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<td>Hispanique</td>
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<td>Amérindien</td>
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<tr>
<td>Mixed</td>
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<tr>
<th><strong>Tableau 3 : Répartition de la fréquence des cotes au PHQ-2</strong></th>
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<tbody>
<tr>
<td><strong>PHQ-2 Cote totale</strong></td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>5</td>
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<tr>
<td>4</td>
</tr>
<tr>
<td>3*</td>
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<tr>
<td>2</td>
</tr>
<tr>
<td>1</td>
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<td>0</td>
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</table>

*Seuil standard
Les résultats prévus dans le présent rapport indiquent une prévalence de dépression beaucoup plus élevée chez les patients en optométrie que ce que l’on pourrait supposer d'après un sondage sur les pratiques optométriques. Les patients qui ont un résultat de dépistage positif au test PHQ-2 ne répondent pas aux critères diagnostiques de dépression majeure. L'application concrète de ces résultats aux soins oculovisuels est complexe. Une méta-analyse des effets d'un dépistage plus complet montre que l'utilisation des outils de dépistage PHQ-2 est une option efficace et pratique pour évaluer une prévalence de dépression clinique dans le milieu médical.

Les patients qui obtiennent un résultat positif au dépistage plus complet peuvent alors être aiguillés vers une évaluation plus approfondie. Ce qui est nécessaire est de la plus haute importance lorsqu’on utilise cet outil de dépistage dans les cliniques de soins oculovisuels.
RÉFÉRENCES

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- **BEPREVE™** should not be instilled while wearing contact lenses. **BEPREVE™** contains benzalkonium chloride as a preservative, which may be absorbed by soft contact lenses. Remove contact lenses prior to instillation; lenses may be reinserted 10 minutes after the administration of **BEPREVE™**
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For more information:
Please consult the Product Monograph at http://www.bausch.ca/Portals/59/Files/Monograph/Pharma/bepreve-pm-en.pdf for important information relating to adverse reactions, drug interactions, and dosing information that has not been discussed in this piece. The Product Monograph is also available by calling 1-888-459-5000.

References:
1. BEPREVE™ (bepotastine besilate ophthalmic solution 1.5%) Product Monograph. Bausch & Lomb Canada Inc.; July 22, 2016.

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Scores des démangeaisons oculaires 3 minutes après PCA (moyenne pour les deux yeux)

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- Il faut éviter tout contact de l’embout compte-gouttes du flacon avec les paupières et les surfaces environnantes et il faut garder le flacon hermétiquement fermé entre les utilisations.
- BEPREVEMC ne doit pas être utilisé pour traiter une irritation liée aux lentilles cornéennes.
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Références :
Early Primary Open-Angle Glaucoma

Austin R. Lifferth, OD, FAAO
Veterans Affairs,
Outpatient Clinic,
The Villages, FL, USA

Introduction

Glaucoma is the leading cause of irreversible blindness worldwide and is projected to affect more than 79.6 million people by 2020, over 10% of whom will be bilaterally blind.1

This multifactorial progressive optic neuropathy causes characteristic retinal nerve fiber layer damage that will eventually lead to associated glaucomatous visual field defects if left untreated. Unfortunately, these visual field defects are difficult for the patient to detect until more advanced stages and, as a result, early glaucoma is usually asymptomatic.2

This paper presents a case that is consistent with population studies that suggest that as many as half of people with glaucoma are unaware that they have the disease.3

CASE REVIEW

A healthy 71-year-old Caucasian male reported to our office as a new patient with complaints of mild blurry vision, OD=OS, at both distance and near.

The patient reported that he had had an eye exam “6-7 years” prior to our exam and was told that he had “symptoms of glaucoma”; however, he was not diagnosed with glaucoma and did not receive additional treatment. He denied any family history of glaucoma, was in good general health, and reported no other difficulties involving either eye.

The patient was self-medicating with 81mg aspirin, multi-vitamins, fish oil capsules, vitamin B complex, and red yeast rice capsules, all taken once daily.

His most recent blood pressure was 150/67 at 14:05 in the seated position, while his A1C was 5.8% and his blood glucose level was 116 mg/dL at 14:37. His body mass index was 28. No allergies to any medications were reported.

The patient’s unaided entering distance visual acuities and pinhole acuities (PH) were:
OD: 20/60-1 PH: 20/25
OS: 20/40+2 PH: 20/25+2

Subjective refraction and best-corrected visual acuities were:
OD: -0.75 -1.50 x070 20/20
OS: -0.25 -1.50 x062 20/25+1

Preliminary testing showed that the pupils were equally round and reactive to light with no relative afferent pupillary defect. Confrontation visual fields were full to finger count OD, OS. Motility testing was full without restriction or pain OD, OS.
Slit lamp examination showed mild bilateral blepharitis, clear conjunctiva, and clear cornea without endothelial pigment or keratic precipitates. The anterior chamber was deep and quiet by Van Herick angle estimation and the iris was normal without signs of atrophy, obvious posterior synechiae, or trans-illumination defects. Intraocular pressures (IOP) were 17 OD, 15 OS at 08:01 by Goldmann applanation tonometry.

Dilated examination showed trace nuclear sclerosis and cortical opacities without evidence of pseudoxefoliation or pigment. The macula, vessels, and periphery were all normal OD, OS. There was a posterior vitreous detachment OD, OS with no evidence of peripheral retinal abnormality.

The optic nerve head was average to large size OD>OS based on the vertical disc height using the adjusted slit lamp graticule and a Volk 78 D lens with a correction factor of 1.2x. The optic cup was of moderate depth, with early visible laminar dots OU. There was mild alpha zone parapapillary atrophy, but no signs of pallor or disc hemorrhages OU. There was a subtle inferior retinal nerve fiber layer wedge defect with associated inferior rim thinning, inferior vessel baring, and inferior arteriole narrowing OU. Additionally, the superior rim was suspicious for glaucomatous optic neuropathy OU with evidence of early vessel baring OD>OS and relative thinning compared to other optic nerve sectors. Cup-to-disc ratios were estimated to be 0.7 v/0.7 h OD and 0.75 v/0.7 h OS.

Baseline photos and optical coherence tomography (OCT) Optic Nerve Head (ONH) and Retinal Nerve Fiber Layer (RNFL) Analysis were acquired. Both subjective and objective imaging confirmed the findings in the clinical exam, as shown in Figures 1 and 2.

**Figure 1:** Retinal images showing inferior-temporal localized retinal nerve fiber layer defects with associated inferior-temporal neuroretinal rim thinning. Note the early relative superior neuroretinal rim thinning OU.

The patient was given a provisional diagnosis of early glaucoma, OS>OD, and asked to return within 1 month for repeat IOP measurements with baseline gonioscopy, pachymetry, and threshold visual field testing.

At the one-month follow-up appointment, the patient was found to have stable acuities without any additional ocular complaints. His intraocular pressures were slightly higher than baseline, at 21 OD, 19 OS at 10:42 am by Goldmann applanation tonometry. Pachymetry yielded central corneal thickness measurements that were slightly thinner than average, at 524u OD, 525u OS. Gonioscopy showed that the ciliary body was visible in all four quadrants with flat iris insertion, light trabecular meshwork pigmentation, and no evidence of peripheral anterior synechiae or angle recession OD, OS.
Figure 2: Optical coherence tomography (OCT) Optic Nerve Head (ONH) and Retinal Nerve Fiber Layer (RNFL) Analysis OU. Note the bilateral nasal posterior vitreous detachments with associated artifacts observed on the RNFL Thickness and Deviation Maps. Furthermore, the RNFL thinning is localized and, as such, is only noted on the RNFL Clock Hours Map while appearing as “normal” on the RNFL Quadrant Map.
Baseline threshold visual field testing (Humphrey 24-2 SITA Fast) showed normal-sensitivity OD with good reliability (0/10 fixation losses, 5% false positives, and 0% false negatives) and several focially depressed superior nasal defects OS with lower reliability (5/11 fixation losses, 3% false positives, and 8% false negatives).

Figure 3: Baseline Humphrey 24-2 SITA Fast visual field plots show focially depressed defects OS only.

Due to the normal gonioscopic appearance of the angle, the patient was given a more specific diagnosis of early primary open angle glaucoma OS-OD with questionable early perimetric defects OS based on the SITA Fast testing algorithm. Bengtsson and Heijl\(^1\) found that SITA Fast test times (avg 5.0 min) were significantly shorter than Full Threshold (avg 14.6 min) and Fastpac (avg 9.4 min) test times, but were essentially equal in terms of reproducibility. However, as expected, the sensitivity for detecting shallow (subtle) defects was greater with Full Threshold testing. In other words, shallow defects noted in Full Threshold testing were progressively less visible (perhaps even absent) with Fastpac and SITA Fast, while the detection of deeper focal defects was essentially identical with all three strategies.

Through collaboration with the patient, we decided to monitor the condition closely at that time without treatment until repeat structural and functional testing confirmed the suspected defects (and/or suggested progression) and to further establish baseline intraocular pressures at various diurnal time points. However, based on the case findings thus far, and based on the Early Manifest Glaucoma Trial (EMGT) and the Collaborative Normal Tension Glaucoma Study (CNTGS), treatment will likely be initiated to help reduce the risk of disease progression. Furthermore, and perhaps more specific to our patient, Kim et al. found that sufficient IOP reduction slowed disease progression even in patients with suspected preperimetric glaucoma.\(^6\)

The patient was asked to return to our office in 3–4 months for repeat tonometry, pachymetry, and 24-2 threshold visual field testing, as well as baseline optical coherence tomography Ganglion Cell Analysis and 10-2 visual field testing. As a brief review, the importance (and benefits) of central 10-degree visual field analysis cannot be overstated: approximately 50% of eyes with mild-moderate glaucoma were found to have defects within the central 3 degrees,\(^6\) 11 eyes with normal 24-2 visual fields outside the central 10 degrees showed arcuate defects within the central 10 degrees with 10-2 testing,\(^7\) nine percent of normal 30-2 threshold visual fields in glaucoma...
suspect or early glaucoma patients were actually classified as abnormal with 10-2 testing and approximately 50% of eyes will show macular glaucomatous damage on 10-2 testing while being classified as normal with just 24-2 testing."

**DISCUSSION**

Weinreb et al. proposed a glaucoma continuum - a spectrum of structural and functional stages in glaucoma in which the patient generally progresses from “normal” and asymptomatic disease to functional blindness – with structural glaucomatous changes usually preceding functional symptoms. The World Glaucoma Association also described this temporal relationship between structural and functional changes throughout the course of the disease and both representations suggest that structural changes are usually detected prior to functional changes. However, and as an important reminder from Alasil et al.’s retrospective study and their findings of a structural and functional “tipping point”, both structural and functional tests are still necessary to assess early glaucomatous damage.

The American Academy of Ophthalmology Preferred Practice Pattern (AAO PPP) for Primary Open-Angle Glaucoma states that mild (early) glaucoma is characterized by “optic nerve abnormalities consistent with glaucoma [such as] ... diffuse thinning, focal narrowing, or notching of the optic disc rim, especially at the inferior or superior poles; progressive narrowing of the neuroretinal rim with an associated increase in cupping of the optic disc; diffuse or localized abnormalities of the parapapillary RNFL [retinal nerve fiber layer], especially at the inferior or superior poles; disc rim, parapapillary RNFL, or lamina cribrosa hemorrhages; [and/or] optic disc neural rim asymmetry of the two eyes consistent with loss of neural tissue” in the presence of “a normal visual field as tested with standard automated perimetry.”

In the American Optometric Association Optometric Clinical Practice Guidelines (AOA CPG), mild glaucoma is defined as an optic nerve with “mild concentric narrowing or partial localized narrowing of the neuroretinal rim; disc hemorrhage; [and/or] cup/disc asymmetry”. Furthermore, the nerve fiber layer shows a “less bright reflex; fine striations to texture; [and/or] large retinal blood vessels [that appear relatively] clear [whereas] medium retinal blood vessels are less blurred [and] small retinal blood vessels are blurred”. However, unlike the AAO PPP, the AOA CPG says that early glaucoma may show “isolated paracentral scotomas, partial arcuate or nasal step [defects]; [and that the] damage is limited to one hemifield with fewer than 25% of points involved, [with a] mean deviation (MD) less than -6dB.” A more succinct definition that seems to help bridge the two above definitions is given by Song and Caprioli: “a progressive optic neuropathy that is defined by characteristic structural changes of the optic nerve with corresponding functional changes of the visual field.”

Nonetheless, once functional loss is detectable, the severity of the glaucomatous optic neuropathy increases with the severity of the visual field loss, as shown by Ng et al. The present patient presented with several risk factors for open-angle glaucoma, including his slightly elevated intraocular pressure with mild fluctuation (albeit based on only two isolated readings) and his mid-advanced age. However, additional risk factors that were not applicable to this case, but which should also be considered, include presence of lenticular exfoliation, glaucomatous disc hemorrhages, African-American ancestry, a first-degree history of glaucoma, and a general history of diabetes or hypertension.

The primary clinical sign that was most convincing that this patient did indeed have glaucoma was the appearance of the optic nerve and retinal nerve fiber layer. Specifically, the inferior retinal nerve fiber layer defects with associated inferior rim thinning, inferior vessel baring, and inferior arteriole narrowing (and potential superior rim thinning) are all characteristic of early glaucoma. Because of this preferential pattern of neuroretinal rim loss, the ISNT Rule mnemonic has been proven to be very effective in differentiating normal optic nerves from those with early glaucomatous damage. Furthermore, the absence of rim pallor helps rule out other optic neuropathies (ischemic, infiltrative, traumatic, toxic, metabolic, and compressive) that could also result in retinal nerve fiber layer defects and arteriole narrowing, and which would necessitate a more through systemic workup, possibly including blood work and neuroimaging. Baseline photos were taken to assist in monitoring for future structural progression that would manifest as widening of the nerve fiber layer defects (locations of future progression and correlating visual field defects), increased rim thinning/vascular baring/arteriole narrowing, increased parapapillary atrophy, and/or further nasalization of the central retinal vessel trunk.
Subjective evaluation of serial photos and objective imaging (OCT) are complementary structural evaluations used in concert with regular functional (visual field) assessment to monitor for progression. All are necessary: the World Glaucoma Association notes that “(c)urrently, no specific test can be regarded as the perfect reference standard for detection of glaucomatous structural and/or functional progression”. Supporting this position, Banegas et al. reported that, in their observational study of 246 eyes, glaucomatous progression was detected in 6.9% of eyes by stereo photos, 15% of eyes by visual field testing, and 25.6% of eyes by OCT-guided progression analysis (GPA) software. Interestingly, of those cases that showed progression, most were only discovered by either stereophotos, perimetry, or OCT testing alone, suggesting a lower percentage of positive agreement among evaluation methods, and emphasizing the importance of using all three to monitor for change. In this situation, the clinical appearance of the ONH correlated very well with the baseline RNFL OCT testing, establishing supporting subjective and objective structural baselines.

In support of making a diagnosis of glaucoma based solely on the appearance of the optic nerve and not waiting for the development of correlating glaucomatous visual field defects, Sommer et al. suggested in 1977 that glaucomatous nerve fiber layer defects (such as those observed in this patient) may develop several years prior to reliable glaucomatous visual field defects. Furthermore, and more recently, Kuang et al. found that RNFL defects observed on OCT testing were noted up to 8 years prior to associated glaucomatous visual field defects. Consistent with these findings, histological studies have found that as much as 50% of retinal ganglion cells are lost prior to *clinically detectable* visual field defects - resulting in a “broken-stick” correlation model between retinal nerve fiber thickness and glaucomatous visual fields, as described previously by Alasil et al.

As mentioned previously, despite the clinically correlating information suggesting early open-angle glaucoma, collaboration with the patient determined that we did not initiate treatment for the following 3 reasons:

1. To establish a baseline IOP range in light of potential fluctuation in initial IOP measurements,

2. To establish baseline visual field reproducibility and reliability,

3. To establish rate of progression, in recognition of the fact that not all patients with glaucoma will progress to the point of visual symptoms affecting their activities of daily living.

**CONCLUSION**

Elevated intraocular pressure is the primary (and currently the only readily modifiable) risk factor for the development of glaucoma and glaucoma progression. Accordingly, if treatment is required in the future, we will work with the patient to establish a customized, unique target IOP range - the “upper limit of a range of IOP at which it is judged likely to retard further optic nerve damage” and to minimize associated visual field loss. It is very important to balance this dynamic IOP range with quality-of-life factors including the estimated lifetime risk of visual disability for the patient, the potential side-effects of treatment options (topical vs. laser vs. minimally-invasive glaucoma surgery), the financial burden of treatment, and the instillation technique/capability.

Primary open angle glaucoma can be missed in its early stages due to its asymptomatic nature, subtle optic nerve morphological changes, and often pre-perimetric presentation. For these reasons, vigilance is required, as we have the best success of preserving a lifetime of functional vision for the patient if we can diagnose glaucoma earlier and, if needed, treat glaucoma sooner.
REFERENCES

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Amiodarone-Associated Optic Neuropathy

Abstract

A 72-year old male presented symptomatic for unilateral inferior visual field loss, but was found to have bilateral optic neuropathy. Clinical features, an extended minimally symptomatic course and a temporal relationship to amiodarone use implicated amiodarone-associated optic neuropathy. Serial ancillary testing analyses provided insight into this entity’s natural course. This patient developed the greatest retinal nerve fiber layer thinning in the inferior quadrant; this may correlate with anatomically larger-diameter axons, supporting a previous publication which suggested that larger-diameter optic nerve axons are more susceptible to amiodarone-induced lipidosis. While rare, amiodarone-associated optic neuropathy may develop and cause permanent loss of visual function.

KEYWORDS

amiodarone, optic neuropathy, optic disc edema, toxic optic neuropathy, anterior ischemic optic neuropathy

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INTRODUCTION
Amiodarone-associated optic neuropathy has been reported in 1.79% of amiodarone users. However, the most recent critical review of this entity, in 2012, identified fewer than 300 cases reported, with only 59 published, and only a small number of case reports have been published since. This case adds to the small body of literature which details the course of amiodarone-associated optic neuropathy, supports its diagnosis as a distinct clinical entity, and provides comprehensive serial photographs, optical coherence tomography and the results of visual field analyses. These findings provide insight into the natural course of amiodarone-associated optic neuropathy.

CASE REPORT
A 72 year-old white male presented as a new patient reporting a dark arc-shaped scotoma in the bottom portion of the right eye's vision. He had initially reported this to his clinical pharmacist about 2 months previously. He denied headaches, jaw claudication, temporal tenderness, transient visual obscurations, nausea, vomiting, fever, malaise and diplopia. His last eye exam had been 5 years previously and was reported as unremarkable.

His medical history included diverticulitis, esophageal reflux, colonic polyps, herpes simplex 1, degenerative joint disease, congestive heart failure and atrial fibrillation. Six months previously, the patient had been hospitalized for progressive shortness of breath with left leg edema due to atrial fibrillation with rapid ventricular response. He was given an intravenous loading dose of amiodarone for 24 hours, then 400 mg twice per day for 10 days, and was discharged on 200 mg daily. He was also taking bumetanidine 3 mg daily, metoprolol 37.5 mg daily, omeprazole 20 mg daily, polyethylene glycol powder daily and warfarin (3 mg on Mondays, Wednesdays and Fridays and 2 mg on Tuesdays, Thursdays, Saturdays and Sundays). His social and family history were non-contributory.

His best corrected acuity was 20/40 in the right eye and 20/25 in the left eye. He had a mild right afferent papillary defect. Other initial findings and anterior segment exam were non-contributory. On dilated fundus exam, the right eye had a cup-to-disc ratio of 0.15/0.15 with a sector of neuroretinal rim pallor superotemporally, and the inferior half of the disc was edematous. There were disc hemorrhages inferonasally and superonasally (Figure 1A). The left eye had no appreciable cupping with a diffusely edematous disc and disc hemorrhages nasally and temporally (Figure 1B). His blood pressure in clinic was 116/75 with a regular pulse rate of 71 beats per minute. Visual field showed a moderate inferior arcuate defect with a few depressed superior edge points in the right eye, and superior and inferior arcuate defects in the left eye (Figures 2 and 3). Optical coherence tomography retinal nerve fiber layer analysis using a Cirrus device (Carl Zeiss, Oberkochen, Germany) showed superior thinning in the right eye, with an overall average thickness of 70 μm. The left optic disc’s neuroretinal rim and retinal nerve fiber layer were grossly elevated, with an average thickness of 154 μm.

The main differential diagnoses at that time included papilledema; bilateral versus sequential anterior ischemic optic neuropathy, either arteritic or non-arteritic; and amiodarone-associated optic neuropathy.

He was initially evaluated emergently by computed tomography without contrast, and then later evaluated by magnetic resonance imaging of the brain and orbits, both with and without contrast, and by magnetic resonance venography. The results were normal with no evidence of a space-occupying lesion, or secondary radiologic signs of increased intracranial pressure or dural venous sinus thrombosis. The patient’s complete blood count was normal, platelets were normal (256 x10^3/μL, reference range 130-400 x10^3/μL), C-reactive protein was elevated (1.305 mg/dL, reference range 0-0.748 mg/dL) and the Westergren erythrocyte sedimentation rate was mildly elevated (23 mm/hour, reference range 0-15 mm/hour), though the clinical picture was not suggestive of giant cell arteritis. At that time, the most likely differential diagnoses became bilateral versus sequential non-arteritic anterior ischemic optic neuropathy and amiodarone-induced optic neuropathy.

The patient was followed-up at 4 weeks, with no changes in his health or medication history in the interim; his visual symptomatology was stable in the right eye, and he remained asymptomatic in the left eye. He again denied headaches, temporal tenderness and jaw claudication. His vision was pinholed to 20/50 right eye, 20/30- left eye. Dilated fundus exam of the right eye still showed an edematous inferior disc margin and superotemporal pallor, but the disc hemorrhage had resolved (Figure 1C). The left optic nerve was still diffusely edematous with a disc hemorrhage temporally (Figure 1D). His visual field indicated a deeper inferior arcuate defect, with worsened
sensitivity of several points and worsened mean and pattern standard deviation, with a few scattered superonasal rim defects in the right eye (Figure 2); the left eye showed an incomplete inferior arcuate defect with a cluster of defects superonasally, which was slightly improved in both hemifields (Figure 3). The retinal nerve fiber layer by optical coherence tomography was stable in the right eye, and still grossly elevated, but with an average thickness of -31 μm (123 μm) in the left eye compared to previously.

Figure 1: Serial optic nerve photographs of the right (top panel) and left (bottom panel) eyes. Initial presentation: (A) right eye showing superotemporal pallor, inferior disc edema, and inferonasal and superonasal hemorrhages; (B) left eye showing diffuse disc edema and nasal and temporal hemorrhages. At 1 month: (C) right eye showing superotemporal pallor, inferior disc edema with the resolution of disc hemorrhages but an intraretinal hemorrhage superior to the disc; (D) left eye showing diffuse edema and a disc hemorrhage nasally. At 2 months: (E) right eye showing superotemporal pallor, inferior edema; (F) left eye showing diffuse disc edema and a disc hemorrhage nasally. At 2.5 months: (G) right eye showing stable superotemporal pallor and inferior edema; (H) left eye showing diffuse disc edema without hemorrhaging. At 6 months: (I) right eye showing superotemporal pallor and resolution of the inferior disc edema; (J) left eye with disc edema clinically resolved. At 8 months: (K) right and (L) left eyes are clinically stable. At 18 months: (M) right and (N) left eyes remain clinically stable.

After consultation with the patient’s cardiologist and clinical pharmacist, given that the patient’s electrocardiogram readings since amiodarone initiation had shown normal sinus rhythm and the patient was anticoagulated with warfarin, the choice was made to discontinue amiodarone and follow-up closely with cardiology.

Over the subsequent weeks and months, the patient was routinely followed-up with serial dilated fundus exams, photographs, visual field exams and optical coherence tomography. Over that time, laboratory testing of the erythrocyte sedimentation rate and C-reactive protein down-trended, complete blood counts and platelets remained normal, hemoglobin A1c was normal at 5.9%, and rapid plasma reagin syphilis screening was non-reactive. His acuity ultimately stabilized at 20/30 in the right eye, which represented an improvement of about 1 line of Snellen acuity, and 20/25 in the left eye, which was roughly stable compared to the initial presentation.

The optic disc edema in both eyes resolved clinically at 6 months, leaving associated atrophy that corresponded to retinal nerve fiber layer thinning on optical coherence tomography. Progression analysis of the retinal nerve fiber layer clearly indicated stability in the area where pallor had been evident on presentation (superotemporally) in the right eye, but showed progressive thinning inferiorly as the edema resolved. Similarly, diffuse progressive retinal nerve fiber layer thinning over the clinical course was visualized in the left eye. Though the disc edema had resolved clinically at 6 months in both eyes, additional retinal nerve fiber layer thinning in the left eye was evident at 8 months on optical coherence tomography (Figures 4 and 5, Table 1). Visual fields ultimately stayed relatively stable in both eyes (Figures 2 and 3, Table 2). His clinical evaluation at 18 months was stable relative to the 8-month evaluation (Figure 1). Amiodarone-associated optic neuropathy was strongly implicated in this case.
Figure 2: Serial visual fields of the right eye. Visual field of the right eye at (from top down) the initial presentation, and after 1, 2, 4 and 8 months showing an inferior arcuate defect and a small cluster of supercentral edge defects, which remained relatively stable and permanent throughout the clinical course.
Figure 3: Serial visual fields of the left eye. Visual field of the left eye at (from top down) the initial presentation, and after 1, 2, 4, and 8 months showing superior and inferior arcuate defects, which remained relatively stable and permanent throughout the clinical course.
**DISCUSSION**

Amiodarone is a widely used and efficacious cardiac anti-arrhythmic benzofuran derivative that can result in toxicity to several organs including the eyes, liver, lungs, nervous system, thyroid and skin. Many amiodarone users experience side effects, around 15% by 1 year and 50% by 5 years, nearly 20% of which are serious or significant and may necessitate discontinuation.

Ocular involvement is typically isolated to corneal micro-deposits (verticillata), which are a well-established, dose-dependent amiodarone-induced keratopathy seen in 70-100% of amiodarone users. These are benign, but in some cases can cause halo vision, glare or photosensitivity. Opacities in the anterior subcapsular lens may also form. These both generally occur 6.5 weeks or later after the initiation of amiodarone, are non-threatening and generally not bothersome to patients. Corneal verticillata are due to amiodarone-induced lipodosis. Amiodarone is very lipophilic and can similarly deposit into tissues throughout the body, slow nerve conduction velocity, and lead to other documented secondary problems systemically. Amiodarone-associated optic neuropathy was first identified in 1987 and can cause visual devastation, including permanent vision loss. The pathophysiology of its development is not yet fully established; however, ischemia secondary to mechanical or biochemical hindering of axoplasmic flow due to the accumulation of intracytoplasmic lamellar inclusion bodies, a drug-induced lipidosis, particularly in large optic nerve axons, has been implicated.
Figure 5: Retinal nerve fiber layer (RNFL) thickness progression analysis of the left eye using Zeiss Cirrus optical coherence tomography. Retinal nerve fiber layer thickness progression analysis from (left to right) the initial visit and after 1, 2, 4, 6 and 8 months showing retinal nerve fiber layer thinning in all quadrants over the clinical course.

Even though corneal verticillata is the most common ocular sequelae of amiodarone use, and this patient did not exhibit verticillata, its absence does not preclude the diagnosis of amiodarone-associated optic neuropathy. In support of this statement, Johnson et al presented a case series and review and found that amiodarone-associated optic neuropathy developed within six months of initiating amiodarone therapy for 19 of 35 (54%) patients who had amiodarone keratopathy and 13 of 18 (72%) of patients without amiodarone keratopathy. The authors further stated, “Consequently, patients with amiodarone-associated optic neuropathy may not exhibit corneal verticillata, particularly since the median duration of amiodarone use in patients with vision loss is four months. Hence, absence of amiodarone keratopathy should not dissuade clinicians from establishing the diagnosis of amiodarone-associated optic neuropathy.”

Reported cases and cumulative reviews of amiodarone-associated optic neuropathy indicate that it generally occurs simultaneously bilaterally (around 66%) and most often shows an insidious onset with slowly progressive visual symptoms over months, associated with optic disc edema and subsequent atrophy. Nearly 90% of those affected develop clinical manifestations of optic neuropathy within 12 months, with symptoms of vision loss occurring at a mean of 9 months and a median of about 3-4 months after drug initiation. However, optic neuropathy has been reported as early as 3-4 weeks after intravenous or oral amiodarone administration. Acuity at presentation may range from 20/15 to light perception, with nearly 20% having acuity of 20/200 or worse. While it may present with acute vision loss, 13% to nearly 30% of patients may be asymptomatic, even with clinically evident optic neuropathy. Visual field deficits vary, but tend to...
be relatively mild and may include centrocecal scotomas, mild peripheral defects, generalized constriction or altitudinal defects.\textsuperscript{8,18,24} This characterization of insidious onset with slow, bilateral progression and prolonged optic disc edema progressing to atrophy was first described by Macaluso et al., and later echoed by subsequent reports, case series and reviews.\textsuperscript{8,17,22,23} However, cases may deviate from this generalization and can present with acute vision loss or unilateral optic neuropathy.\textsuperscript{19} Stabilization of the optic disc with resolution of edema tends not to occur for several months, with a median 3 months,\textsuperscript{2,19,23} even after the recovation of amiodarone. Interestingly, the development of optic disc edema has been reported even weeks after the discontinuation of amiodarone.\textsuperscript{18} Amiodarone in the blood may remain at therapeutic or near-therapeutic levels for extended periods due to its variable and long half-life,\textsuperscript{16,25} averaging 58 but up to 142 days,\textsuperscript{7} and thus is thought to continue to exert a toxic effect on the disc even after drug discontinuation.\textsuperscript{14,24}

Table 1: Retinal nerve fiber layer average thickness (\textmu m) in each quadrant in the right and left eyes at initial presentation and 1, 2, 2.5, 4, 6 and 8 months from the initial presentation, and final variance in retinal nerve fiber layer thickness (\textmu m) from the initial evaluation at 8 months.

<table>
<thead>
<tr>
<th>Interval from Presentation</th>
<th>Right Eye</th>
<th>Left Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inferior</td>
<td>Superior</td>
</tr>
<tr>
<td>0</td>
<td>105</td>
<td>63</td>
</tr>
<tr>
<td>1 month</td>
<td>107</td>
<td>65</td>
</tr>
<tr>
<td>2 months</td>
<td>98</td>
<td>59</td>
</tr>
<tr>
<td>2.5 months</td>
<td>98</td>
<td>63</td>
</tr>
<tr>
<td>4 months</td>
<td>97</td>
<td>56</td>
</tr>
<tr>
<td>6 months</td>
<td>91</td>
<td>59</td>
</tr>
<tr>
<td>8 months</td>
<td>89</td>
<td>59</td>
</tr>
<tr>
<td>Final Variance From Initial Presentation</td>
<td>-16</td>
<td>-4</td>
</tr>
</tbody>
</table>

Table 2: Visual field mean deviation (dB) and pattern standard deviation (dB) in the right and left eyes at initial presentation and 1, 2, 4 and 8 months from initial presentation, which ultimately remained relatively stable in both eyes over the clinical course.

<table>
<thead>
<tr>
<th>Interval from Presentation</th>
<th>Right Eye Mean Deviation</th>
<th>Right Eye Pattern Standard Deviation</th>
<th>Left Eye Mean Deviation</th>
<th>Left Eye Pattern Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-5.58</td>
<td>3.82</td>
<td>-7.62</td>
<td>5.61</td>
</tr>
<tr>
<td>1 month</td>
<td>-8.38</td>
<td>5.64</td>
<td>-6.36</td>
<td>2.97</td>
</tr>
<tr>
<td>2 months</td>
<td>-7.22</td>
<td>3.97</td>
<td>-7.66</td>
<td>4.74</td>
</tr>
<tr>
<td>4 months</td>
<td>-6.28</td>
<td>6.28</td>
<td>-7.33</td>
<td>6.69</td>
</tr>
<tr>
<td>8 months</td>
<td>-5.47</td>
<td>3.81</td>
<td>-8.78</td>
<td>6.75</td>
</tr>
</tbody>
</table>

The prognosis varies from some improvement in up to 40-58% of patients, a decline in acuity in 10-21%, even after amiodarone discontinuation, and permanent visual loss that remains stable compared to presentation in 21%.\textsuperscript{18} Acuity worse than 20/200 in at least one eye has been shown in 20% of those affected.\textsuperscript{3} Visual field loss is generally permanent.\textsuperscript{21} Similar to this case, previous reports have documented a transient increase in retinal nerve fiber layer thickness consistent with acute optic disc edema, followed by progressive axonal loss, as measured by optical coherence tomography, even after the discontinuation of amiodarone.\textsuperscript{21} This axonal loss is appreciated clinically as pallor of the optic disc.\textsuperscript{14,18,24}
There remains some controversy as to whether amiodarone-associated optic neuropathy is truly an entity distinct from non-arteritic anterior ischemic optic neuropathy, typically its main differential diagnosis,\(^2\,^14\) if it is simply a variant, or possibly only a risk factor for it.\(^2\,^29\) Non-arteritic anterior ischemic optic neuropathy is thought to be due to ischemia involving the posterior ciliary arteries.\(^2\) Non-arteritic anterior ischemic optic neuropathy and amiodarone-associated optic neuropathy patients share similar systemic cardiovascular risk factors,\(^2\) along with similar clinical characteristics such as the appearance of the optic discs and patterns of visual field loss,\(^2\,\,^29\) making the differentiation more difficult.\(^2\,\,^21\) However, there are features of amiodarone-associated optic neuropathy that are atypical of non-arteritic anterior ischemic optic neuropathy, and growing clinical evidence supports that it is a distinct diagnosis.\(^2\,\,^24\)

Unlike amiodarone-associated optic neuropathy, non-arteritic anterior ischemic optic neuropathy generally presents acutely (over hours to days, possibly weeks)\(^2\,\,^22\) with severe,\(^2\) unilateral optic nerve dysfunction,\(^2\) typically inferior altitudinal visual field defects,\(^2\,\,^20\,\,^21\) and acuity ranging from 20/20 to no light perception.\(^2\) The resolution of disc edema typically occurs over the course of 4-8 weeks.\(^2\,\,^42\) The incidence in those over 50 years of age has been reported to be 0.01-0.02\%,\(^2\,\,^25\,\,^29\) which is much lower than that reported for amiodarone-associated optic neuropathy. Cases may be bilateral, but are almost universally sequential rather than simultaneous.\(^2\) Bilateral cases present at a lower rate than that seen in amiodarone-associated optic neuropathy,\(^2\) as simultaneously bilateral cases are usually associated with sudden arterial hypotension or perioperative hypovolemia.\(^4\,\,^42\)

Most (90\%) cases of amiodarone-associated optic neuropathy have been reported in males,\(^2\) while non-arteritic anterior ischemic optic neuropathy exhibits an equal male-female distribution.\(^2\,\,^20\) Cheng et al. conducted a retrospective population-based cohort study to investigate whether there was an increased risk of optic neuropathy in amiodarone-treated patients, and found a 2-fold increased risk; male amiodarone users had a 3-fold greater risk of optic neuropathy.\(^2\) Amiodarone-associated optic neuropathy has no predilection for small discs or cup-to-disc ratios,\(^2\) whereas non-arteritic anterior ischemic optic neuropathy has a nearly exclusive predilection for small optic discs with small cup-to-disc ratios.\(^4\)

Performance on color vision testing may vary depending on the optic nerve's function in any optic neuropathy,\(^27\) but tends to remain normal in ischemic, but abnormal in inflammatory, optic neuropathies.\(^26\) Miller and Arnold report that loss of color vision in non-arteritic anterior ischemic optic neuropathy tends to parallel that of acuity.\(^2\) Blue color deficiency has been reported as an early indication of amiodarone-associated optic neuropathy;\(^2\) thus, color testing may be a helpful tool for assessment and monitoring. In this patient, abnormal color testing results were not used as a functional measure for disease-monitoring because he reported congenital color deficiency with unknown baseline color-discrimination ability.

The direct cause of optic neuropathy due to amiodarone use has not been well established, and thus is not universally accepted. Regarding ocular toxicology, Fraunfelder and Shults assert that there are inadequate data to support amiodarone as a cause of toxic optic neuropathy,\(^2\) and Younge aptly notes that causation cannot be confirmed without a prospective controlled study, which would be difficult to design.\(^4\) There are ethical limitations to conducting an ideal prospective, double-masked, randomized, placebo-controlled study, because this would require the withholding of treatment in the placebo control group; however, since amiodarone is used to treat life-threatening cardiac dysrhythmias, withholding treatment would be unethical.\(^5\) Similarly, positive re-challenge of the drug in cases of optic neuropathy to confirm its causation would also be unethical. Thus, causation remains only strongly speculated.

Mindel et al. undertook a randomized trial investigating amiodarone’s role in the prevention of sudden cardiac death, and bilateral vision loss was considered a secondary end-point as a way to explore this entity. Of the 837 subjects using amiodarone, none self-reported bilateral vision loss.\(^2\) However, patients did not undergo ophtalmic examinations, and this endpoint was reached if subjects reported “yes” to having “optic neuritis.”\(^2\) Therefore, mild, unilateral, peripherally affected, or asymptomatic cases may have gone undetected, and patients may have underreported their visual symptoms due to poor understanding of the investigator’s inquiry regarding “optic neuritis.”

In the present case, the patient remained visually asymptomatic in the left eye. He presented with symptomatic arcuate inferior visual field loss in the right eye, which could have represented superior optic nerve segment non-arteritic anterior ischemic optic neuropathy prior to the development of, or in conjunction with, amiodarone-asso-
cated optic neuropathy; however, it is unlikely that the subsequent optic disc swelling in the right eye represented a sequential unilateral non-arteritic anterior ischemic optic neuropathy. Subsequent non-arteritic anterior ischemic optic neuropathy in the fellow eye was found to occur in 14.7% of cases by 5 years in a ischemic optic neuropathy decompression trial; however, subsequent non-arteritic anterior ischemic optic neuropathy in the same eye is much less common, reportedly only up to 6.4%. Given that optic disc crowding is a risk factor, the low risk of sequential ipsilateral disease is presumably associated with decompression of the disc as the affected fibers atrophy, though other factors also likely contribute. This patient's baseline cup-to-disc ratio was unknown, thus the association of non-arteritic anterior ischemic optic neuropathy to small cup-to-disc ratios cannot be confidently applied or excluded; regardless, there is only a very small likelihood of a sequential non-arteritic anterior ischemic optic neuropathy in the same eye in association with sequential or simultaneous visually asymptomatic non-arteritic anterior ischemic optic neuropathy in the fellow eye in this patient. However, it remains possible that the patient had superior segmental non-arteritic anterior ischemic optic neuropathy in his right eye previous to, or co-existing with, the bilateral amiodarone-associated optic neuropathy.

Amiodarone-associated optic neuropathy remains a clinical diagnosis of exclusion, but should be a differential diagnosis with optic neuropathy surrounding use of the drug. Other differential diagnoses include papilledema and other optic neuropathies such as those associated with infectious, inflammatory, infiltrative, compressive, nutritional, metabolic or other toxic sources. This patient had a lack of pertinent exposures to various infectious causes such as tuberculosis, syphilis, Lyme disease or cat scratch disease, and, in conjunction with his clinical presentation, infectious etiologies were thought unlikely. Further laboratory investigations aside from a rapid plasma reagin to screen for syphilis were not pursued, which represents a limitation of this report.

In cases of bilateral optic disc edema, giant cell arteritis and increased intracranial pressure must be excluded. This patient initially had a mildly elevated C-reactive protein and erythrocyte sedimentation rate; both can be elevated in giant cell arteritis and numerous other conditions. The American College of Rheumatology's diagnostic criteria for giant cell arteritis include an erythrocyte sedimentation rate of 50 mm/hour or more. Hayreh et al. reported that the "clinical criteria most strongly suggestive of giant cell arteritis include jaw claudication, C-reactive protein above 2.45 mg/dl, neck pain, and an erythrocyte sedimentation rate of 47 mm/hour or more, in that order", and this patient demonstrated none of these. Also, his platelet count was normal. A platelet count greater than 400 x10^9/µL has been reported as a useful marker to predict that a temporal artery biopsy would be positive for giant cell arteritis, and thrombocytosis has been reported to be a predictor of an ultimate diagnosis of giant cell arteritis in patients referred for temporal artery biopsies; this patient's platelet count was well below that threshold. Additionally, he denied systemic symptoms typical of giant cell arteritis, and though it may be isolated without systemic symptoms despite a confirmed positive temporal artery biopsy in some cases, his central visual acuity was well-preserved in both eyes, which is not consistent with arteritic anterior ischemic optic neuropathy. Additionally, the duration of disc edema without substantial vision loss is not consistent with arteritic ischemic optic neuropathy. These several factors lead to a very low level of suspicion, so temporal artery biopsy was not warranted or pursued.

Papilledema due to increased intracranial pressure was also considered. This patient underwent neuroimaging that ruled out a mass lesion, and the radiologist detected no secondary radiologic evidence of increased intracranial pressure or dural venous sinus thrombosis, such as optic nerve sheath enlargement, flattening of the posterior sclera, or optic nerve tortuosity. Because of the patient’s anti-coagulated state and the low suspicion of increased intracranial pressure, a lumbar puncture was deferred.

CONCLUSIONS
In this patient, his retinal nerve fiber layer thickness remained roughly stable superiorly and nasally in his right eye over the duration of the clinical course, but already appeared pale at the time of presentation. The inferior and temporal retinal nerve fiber layer thickness dropped by 16 and 13 µm, respectively, as the inferior edema progressed to become somewhat atrophic. His left eye, which upon initial evaluation had gross optic disc edema, had dramatic relative retinal nerve fiber layer thinning over the clinical course, more blatantly inferiorly (~45 µm), then temporally (~105 µm), nasally (~74 µm) and lastly superiorly (~68 µm). FitzGibbon and Taylor reported that, in human retinas, retinal ganglion cell axons tend to be larger on average inferiorly and/or nasally than superiorly and/or temporally, and that foveal axons are generally smaller than extrarfoveal axons. Interestingly, this case demonstrated more retinal nerve fiber layer loss inferiorly in both eyes, suggesting that the generally larger inferior axons were more significantly affected by this optic neuropathy. In conjunction with Mansour’s findings suggesting that
amiodarone use results in the accumulation of intracytoplasmic lamellar inclusion bodies, particularly in large optic nerve axons, FitzGibbon and Taylor's finding of a variable retinal axonal size distribution may be significant for future investigations of amiodarone-associated optic neuropathy's relative regional optic nerve impact and involvement based on axonal diameter.

Though uncommon, amiodarone use can lead to optic neuropathy, which can cause a permanent loss of visual acuity or field. Symptoms are usually insidious and patients may even be asymptomatic, so detection of optic neuropathy, prior to the development of visual symptoms, would be ideal; color vision testing, a dilated exam and visual fields have great clinical utility in early detection. It is advised that patients be evaluated with a comprehensive eye examination at 4 and 12 months after initiating amiodarone therapy, and at least annually thereafter.

Aside from early detection, it can be difficult to decide upon the appropriate course of action in cases of amiodarone-associated optic neuropathy, particularly in patients who are visually asymptomatic. There is no unanimity regarding whether amiodarone should be discontinued, because there can be varying visual outcomes regardless of whether amiodarone is discontinued, and clearly these patients need to treat their high-risk underlying arrhythmia. Patient care should be individualized, and consultation with the patient’s cardiologist is needed to carefully consider the risks and benefits of amiodarone therapy.

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With a PhD in history of medicine, science and technology with over twenty years of experience in teaching and coaching, Jeanette’s research interests focus on the production and dissemination of knowledge, technology, and innovation, in both healthcare and education.

If mobile is already entrenched as the digital technology shaping our everyday lives, Artificial Intelligence (AI) is on the threshold of being the next all-pervasive and transformative development. The automation of health care by using machines that behave “intelligently” is the major application of AI in the health sector, encompassing everything from simple pattern recognition to “smart” objects like an AI-driven insulin pump to predictive data analytics (identifying, for example, patients most at risk for hospital readmission).

AI is presently garnering attention and building momentum because the technology requirements are now being met. This includes computing power and storage, cloud computing, and most importantly the availability of big data sets. Add to this the ubiquitous connectivity that enables the Internet of Things (networked devices), which have the potential to act as the mechanical “body” to AI’s mechanical “brain.”

As in the case of mobile connectivity, the addition of “intelligence” brings a new dimension to digitization. A force for long term change in healthcare, AI’s impact on both clinical practice and patient experience, whether in methods or access, is both direct--for example, in new automated clinical tools--and indirect, through its centrality to the data-driven research that makes the emerging model of precision medicine possible.

To date, much of the expansion of AI-driven health applications has been in “smart” services/products around diagnostics: for example, smart monitors, imaging analysis, and screening tools. Imaging may represent as much as 90% of all medical data. In the news recently have been a number of screening tools using automated analysis of retinal scans, whether to diagnose age-related macular degeneration or diabetic retinopathy or identify patients at risk for heart disease.

AI tools that aid in clinical decision-making support are a related area of growth. These use the study of personal health information to inform treatment decisions for individual patients, through predictive models that anticipate how a patient will respond to a particular therapy. One relevant example is Microsoft’s international collaborative eye care project (MINE), which has ongoing projects that apply machine learning to the rate of change of myopia in children or predicting outcomes of refractive surgery.

AI is also driving the expansion of telehealth and telehomecare. Ranging from simple bots for triage and patient education to intelligent assistants (think Amazon’s Alexa) that offer homecare/caregiver support to smart homes of the near-future that incorporate continuous remote monitoring of the health of the elderly, the automation of simple tasks fills gaps in care that can occur when the demand for services--as is all but inevitable in an aging population--far outstrips the supply of care providers.
Digital therapeutics, in which software acts as treatment modality, either via remote monitoring and or behavioural modification, is also rapidly gaining ground. A number of certified mobile apps that use gamified, remote monitoring of visual acuity (including those specifically for the management of macula diseases) are already available. These digital therapeutics have the additional advantage of making copious amounts of real-time data available on their implementation, creating feedback loops that offer the potential to optimize treatment regimens.

While there is considerable and legitimate excitement about the potential health applications of AI, the technology is still in its infancy. The base building block of AI, algorithms, underlie most of our existing technology. For example, the Fourier transform algorithm is the “recipe” that turns raw data into ultrasound or MRI images. The current excitement in AI are breakthroughs in machine learning and self-improving algorithms that can be “trained” with the input of large amounts of data on how to do a task better, like taking using vast quantities of medical images to improve the accuracy of an automated diagnosis of disease.

This kind of “narrow” AI that is good at one specific thing is here, but general AI that features all the facets of human intelligence such as planning, understanding language, recognizing objects and sounds, learning, and problem solving is still distant. Generally speaking, humans have no need to worry about being replaced by machines. While the automation of skilled labour will have an impact on some tasks traditionally the province of some healthcare specialties, patient-facing practitioners should regard them as labour-saving tools that extend their reach and help make their jobs easier, and more effective.

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Employee Not Meeting Expectations?

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It can be surprising and frustrating when an employee is not living up to your expectations at work. Whether that is in their performance or attitude, something is not working and as the leader of the business you have to make some changes. Before you jump too quickly to letting them go or resorting to discipline, let’s review the most common reasons someone is not meeting employer expectations:

1. **Not enough training**
   We can’t fault people for not doing what we expect if we never showed them how we wanted it done in the first place. So many workplaces leave the onboarding to one of the staff in the office with very little planning or preparation. The new employee gets hurried, ineffective training and then is expected to know it all. Set yourself up for success by developing a training program for all staff.

2. **Clear direction**
   Similar to the first point, if you want something done in a particular fashion, then you need to clearly define what you want and how you want it completed. You will get far more out of all of your employees if you take the time to document all process, policies and procedures - including individual responsibilities - and sit down with each employee to review.

3. **They are disengaged**
   If you have an employee who is actively disengaged in their role and daily tasks then how they perform them will be less than stellar. At this point you need to have a heart to heart with this employee and uncover the cause of their disengagement and whether there is the ability to turn it around. If not, actively manage them out of the business.

4. **Not the right fit**
   You may have an employee in the wrong role. You might have them at reception when they really want to be in pre-test. It’s not in their DNA to do the reception tasks and answer the phone all day but they are a great employee. Talk to the under-performing employee and see if it is a poor role fit or an understanding of the tasks at hand.

5. **Internal conflict**
   We often see performance problems when there are underlying internal conflict issues as well. Maybe you have two team members who have had a disagreement and it has not been resolved. It may be affecting one or both of them to the degree that it is also affecting their daily work and responsibilities. Helping to get the issue resolved and the ability to move forward can immediately make a difference in their overall attitude.

6. **They don’t get the big picture**
   All employees need to understand what the mission of the practice is and what you believe in, so they are inspired and encouraged to work towards that objective every day with every patient. Let them know their purpose and role in achieving the mission so they feel invested and involved.

   Last note: Most employees don’t set out to under-perform. Most want the leaders to be happy with them and their work. By setting them up for success from the beginning with great leadership, clear direction and ongoing communication you may just see them living up to your expectations.
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