Journal of Sports and Performance Vision

Original Article
DOI:XXXXXXXXX

OCULAR PATHOLOGICAL FINDINGS IN PROFESSIONAL BASEBALL PLAYERS

David G. Kirschen, OD, PhD[1] and Daniel M. Laby, MD[2]
[1]Southern California College of Optometry, Fullerton, CA; Jules Stein Eye Institute, UCLA School of Medicine, Los Angeles, CA; Private Practice.
[2]Department of Ophthalmology, Harvard Medical School, Boston, MA; Private Practice; Sports and Performance Vision Center, Director, SportsVisionNYC.

Corresponding Author: davidkirschen@mac.com

Submitted: October 10, 2019. Accepted: November 14, 2019. Published: December 5, 2019.

ABSTRACT

This article highlights the eye findings uncovered among 800 professionals, with no visual complaints. Methods: During their yearly sports vision screenings between 2004 and 2011 these retinal and anterior segment pathologies were detected and documented using a Kowa-Optimed (Hawthorne, CA) non-mydriatic fundus camera. Results: Twelve athletes (1.5%) were found to have ocular pathology in the posterior pole or anterior portion of the eye. The findings were categorized into five groups, nevi, hemorrhages, retinal scars and abnormalities, optic nerve changes, and vascular changes. Conclusion: This work illustrates the importance of eye health screenings as an integral part of a comprehensive sports vision evaluation.

Key Words: *Sports Vision, Ocular Pathology, Ocular Photography*

Despite the superior physical prowess of professional athletes, these individuals still require regular eye care just like the general population. This article highlights the pathological eye findings uncovered among 800 professional athletes, with no visual complaints, during their yearly sports vision evaluations between 2004 and 2011. There is insufficient time during Spring Training to do a complete comprehensive medical eye examination on each of these athletes. The purpose of the eye health screenings is to identify those players who have suspicious pathological findings and send them for complete medical eye examinations for a definitive diagnosis and treatment if indicated. These findings are observations, with the potential of clinical significance, in otherwise healthy individuals. We have limited our discussion to abnormal findings in the posterior pole and the anterior surface captured by a Kowa non-mydriatic fundus camera providing 20 degree and 45-degree views. No other ophthalmic techniques or methods were used to examine the posterior segment. We were able to classify the atypical eye findings found in 12 different athletes into one

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

of five separate categories: nevi, hemorrhages, retinal scars and abnormalities, optic nerve changes, and vascular changes.

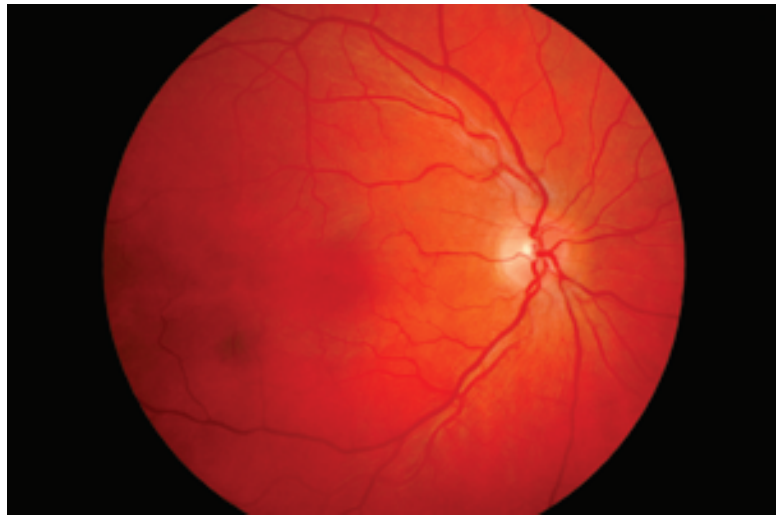
NEVI

We documented three athletes each with a single nevus in one eye. The first can be described as circular, slate-gray, with indistinct borders, approximately a half disc diameter in size, and is located 4 disc diameters away from the optic disc in the lower temporal quadrant of the right eye. (Figure 1A athlete RM) The second is round with irregular borders, that is half a disc diameter in size, and is located three 3-disc diameters away from the optic disc along the superior arcade in the upper temporal quadrant of the right eye. (Figure 1B, athlete RR) The third is vertically elongated with irregular borders, which is a single disc diameter in size, and is located within a disc diameter of the optic nerve head. (Figure 1C athlete JH) This nevus has a curved edge that follows the contour of the optic disc from 1 to 3 o'clock in the left eye. The latter two nevi have a green-gray appearance.

A choroidal nevus is defined as a benign tumor composed of atypical melanocytes.[1,2] Nevi are typical findings made during ocular examination that are usually flat or slightly elevated by 1–2 mm, are round or oval with defined but not sharp borders,[1,3] and tend to vary in size between one third and 7 disc diameters.[1] The many reports in the literature suggest the prevalence of choroidal nevi varies from 0.2 to 32%; this is due to disparities in examination methods, criteria for identifying a nevus, and the type of report (histopathologic or clinical).[1,3] Prevalence rates for non-Caucasian races is understudied, but are considered to be very low and rare.[3] Relevant to our findings and the prevalence rates reported in a study by Naumann et al [4] suggests that while 75% of nevi are located in the posterior pole and immediately surrounding area, 25% of nevi remain undetected due to limitations of the field of view in fundus photography.

The primary clinical significance of nevi is the rare potential for malignant transformation to uveal melanomas[3], which assumes uveal melanomas arise from preexistent benign nevi.[1] The Blue Mountains Eye study[3] estimates an incidence of 1 melanoma per 4300 nevi per year for both sexes, which is relatively consistent with an estimate by Ganley and Comstock[5] that for every 4800 nevi diagnosed per year, one will transform into a choroidal melanoma. There is no association between nevi and the presence of eye diseases such as glaucoma, cataracts, or diabetes.[6] As noted in our three athletes, The Blue Mountains Eye study[3] also reported no instances of decreased visual acuity due to nevi, however other studies disagree and attribute findings of visual field defects to nevi.[1,2] There appear to be no risk factors associated with the

FIG. 1A A circular, slate-gray nevus with indistinct borders, approximately a half disc diameter in size located 4 disc diameters away from the optic disc in the lower temporal quadrant of the right eye. (Athlete RM)



This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

FIG. 1B A round nevus with irregular borders, a half a disc diameter in size, and located three 3 disc diameters away from the optic disc along the superior arcade in the upper temporal quadrant of the right eye. (Athlete RR)

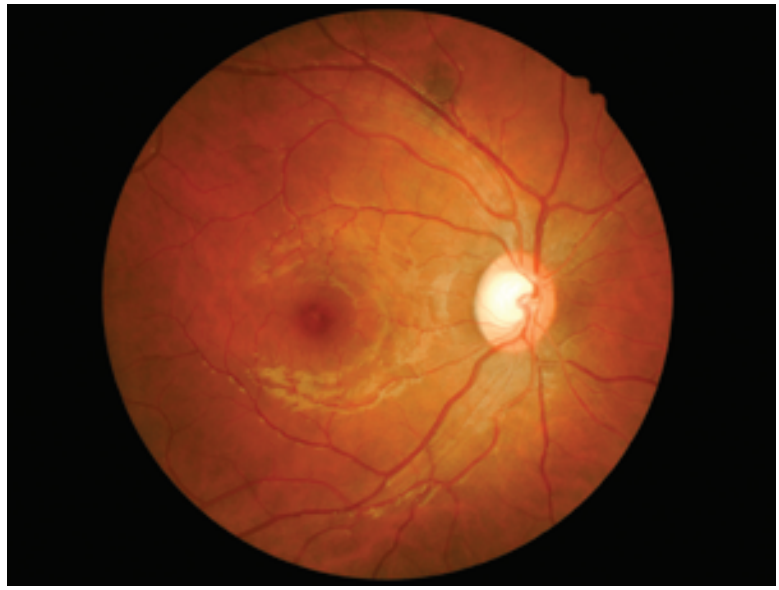
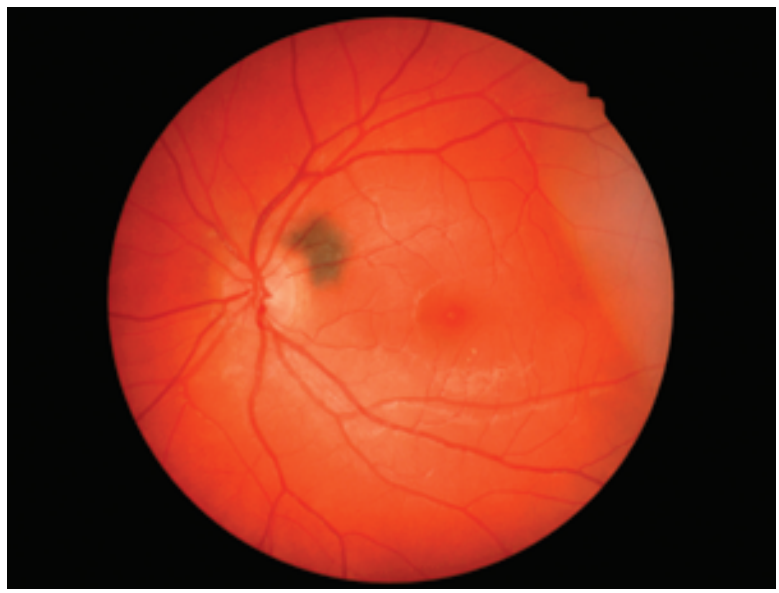


FIG. 1C A vertically elongated nevus with irregular borders, a single disc diameter in size, located within a disc diameter of the optic nerve head in the left eye. (Athlete JH)



presence of choroidal nevi in regards to iris color, skin color [1,3], hair, or sex.[1] However, The Blue Mountains Eye Study did find that nevi were significantly less frequent in individuals with blond hair in comparison to all other hair colors.[3]

After observing a choroidal nevus, the primary course of action should be to differentiate suspicious from non-suspicious lesions through careful observation for enlargement, which is an indicator of potential malignant transformation. Conversely, a stable appearance

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

over a prolonged period suggests a benign nevus. There is consensus among several studies on a constellation of reliable factors associated with nevus growth; these consist of orange pigmentation (lipofuscin), thickness greater than 1.5 mm, visual symptoms[1,2], exudative/serous retinal detachment[1], and largest diameter exceeding 3 disc diameters.[2] Generally, larger elevated lesions are more likely to grow and become malignant rather than smaller flatter lesions. More specifically Gass showed that nevi with diameters greater than 6 mm and thickness greater than 2 mm had a greater propensity to enlarge.[7] In comparison, Tamler and Maumenee[8] observed that nevi less than 2 disc diameters (3mm) showed no growth after 9 years. Another notable indicator is the presence of overlying drusen which demonstrates chronicity of the lesion and is a sign of low growth potential.[3] There is a discrepancy as to the importance of the presence of subretinal fluid affecting the potential growth of a nevus.[1,3] While The Blue Mountains Eye Study[3] found that nevi within 2 disc diameters of the optic disc are a relatively common feature, other studies have found an association between nevus growth and proximity of the most posterior edge of nevus to the optic nerve.[9] Therefore we are more apt to watch for stronger indicators of growth about recommending care for the one athlete(Fig 1C athlete JH) in our screening with the nevus next to their optic disc.

The average optic disc size is 1.76 mm horizontally and 1.92 mm vertically. So, the first 2 athletes discussed above, RM and RR, each have a nevus measuring approximately half a disc diameter, which is less than 1 mm wide. In consideration of the criteria discussed above, both lesions show a low indication of potential growth and malignant transformation due to the relatively small size and flat appearance. The third athlete, JH, poses a greater risk for growth due to the proximity of the nevus to the optic disc.

At the very least, for benign nevi, individuals should be monitored annually and have baseline documentation with photographs, preferably red-free because a red-free filter helps differentiate the depth of the lesion in the retina (RPE vs. choroid). For suspicious nevi, periodic ophthalmoscopy and fundus photos are recommended. Adjunct testing with visual field testing, fluorescein angiography, and ultrasounds are useful in more suspicious cases.[1,2] More advanced imaging technology, such as OCT, is now available to document changes in choroidal nevi. Shields et. al [10] concluded that "OCT is a useful diagnostic modality for imaging the retina overlying a choroidal nevus. Numerous overlying changes such as subretinal fluid, retinal edema, retinal thinning, and photoreceptor attenuation are visible by OCT."[10]

All three players have been followed on an annual basis and no changes in their nevi have been noted. The question often arises about an athlete's ability to decrease their risk of developing new or changes in existing nevi. Eye care professionals can agree on recommending UV protecting sunglass wear to patients for protection from damaging UV rays, however, the potential for specifically decreasing the risk of uveal melanomas is not yet supported by literature.[11–13] Overall, studies examining sunlight exposure as a risk factor for uveal melanoma are not conclusive. [11-13] The suggestion that acute or intense UV exposure may increase the risk for uveal melanoma is still being explored. [11–13]

HEMORRHAGES

Flame Hemorrhages

Two athletes each presented with a single and small unilateral flame-shaped hemorrhage. The first is located within one disc diameter below the superior arcade and above the macula in the superior-temporal quadrant of the individual's right eye. (Figure 2A athlete CC) The second is located within one disc diameter of the optic disc just below the superior arcade in the superior-temporal quadrant of the individual's left eye.(Figure 2B athlete BC) Flame-shaped hemorrhages result from intra-retinal blood vessel rupture located in the superficial nerve fiber layer of the retina. The fundus photos show that both hemorrhages are confined by, and run parallel along, the arching nerve fiber layers. Also, flame hemorrhages are typically found in the posterior pole, as opposed to the peripheral retina. Such intra-retinal hemorrhages can be caused by a multitude of disorders, including several systemic diseases and retinal vascular occlusions. In particular, the presence of unilateral intra-retinal hemorrhages is most commonly due to venous occlusive disease.[14] Other conditions that are often associated with flame hemorrhages include systemic hypertension, leukemia, severe anemia, thrombocytopenia, and trauma.

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

FIG. 2A Flame shaped hemorrhage located within one disc diameter below the superior arcade and above the macula in the superior-temporal quadrant of the right eye. (Athlete CC)

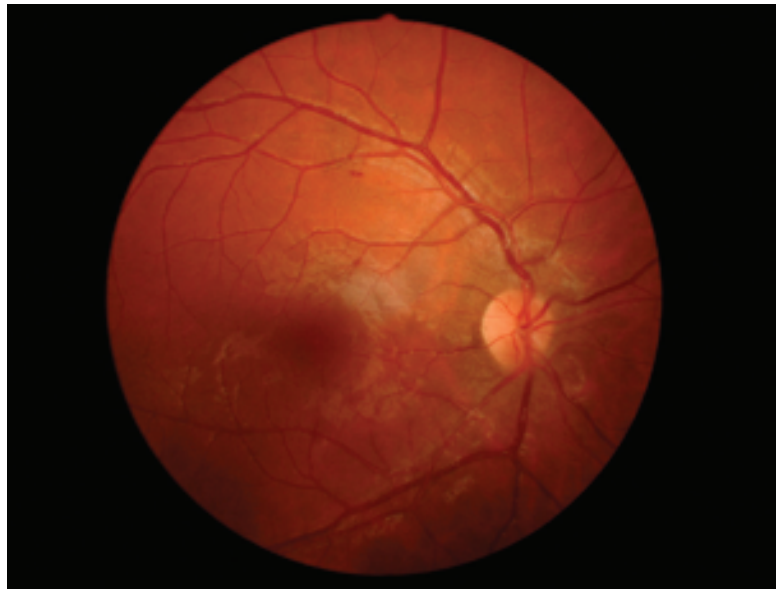
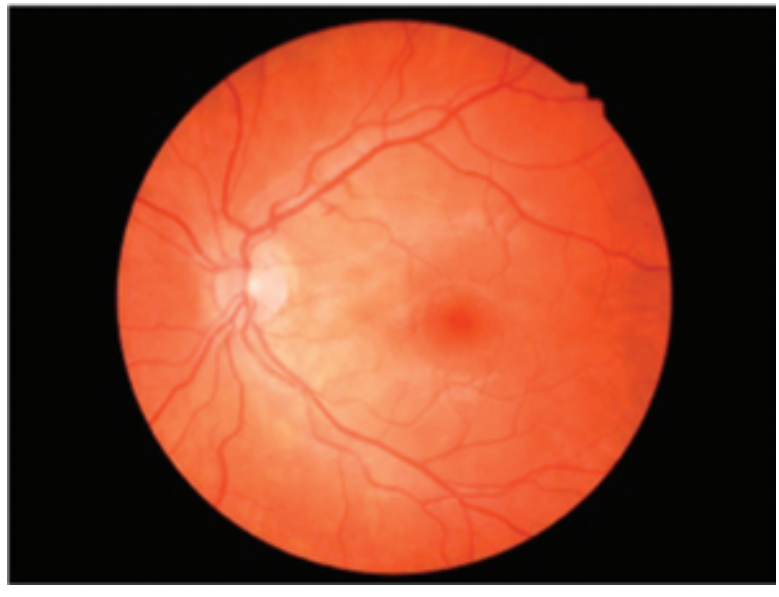


FIG. 2B Flame shaped hemorrhage within 1 disc diameter of the optic disc just below the superior arcade in the superior-temporal quadrant of the left eye. (Athlete BC)



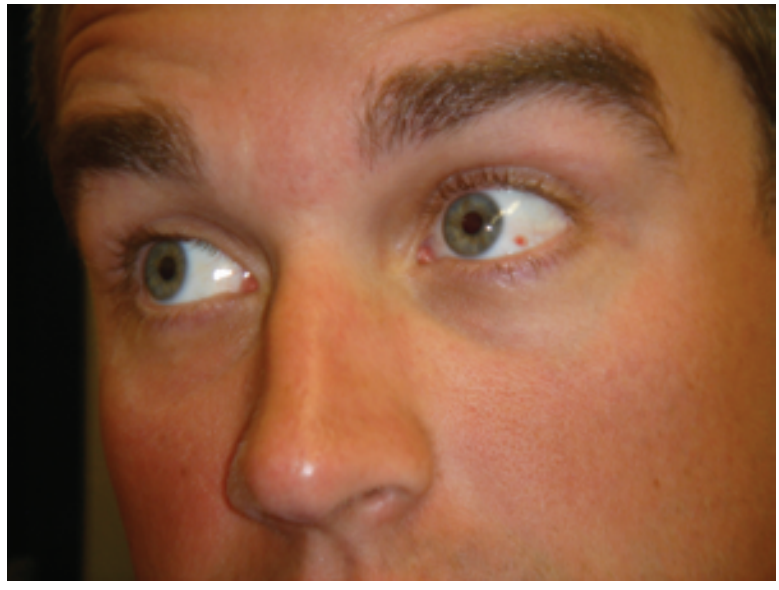
In the absence of any systemic disease, these players should be examined on an annual basis with photo documentation of any retinal abnormalities. These two players were lost to follow-up but these results remain in their medical file that usually follows the players when they are traded to a different team.

Subconjunctival Hemorrhage

One athlete presented with a small subconjunctival hemorrhage (SCH) located infero-temporally; the lesion is approximately 3-mm wide. (Figure 2C athlete JO). The anterior segment image was also obtained using the Kowa fundus camera. SCH is a condition

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

FIG. 2C Small subconjunctival hemorrhage located infero-temporally in the left eye approximately 3-mm wide. (Athlete JO)



commonly encountered by eye care practitioners, especially since its striking appearance quickly brings distressed patients to the office.[15,16] SCH is characterized by the acute manifestation of a defined area of blood between the bulbar conjunctiva and sclera that results from the rupture of small capillaries.[17] The condition is generally benign[15,16] and is self-limiting with resolution within five to ten days in most cases.[17] A study by Fukuyama et al found that from a total of 8,726 patients, there was a 2.9% SCH presentation in an outpatient eye clinic.[18] SCH appears to have no risk factors regarding age, sex, or race.[16,17]

The most common causes are due to physical forces with the leading etiology being acute ocular trauma including seemingly insignificant events like vigorous eye rubbing.[15,17] Also SCH frequently results from numerous mechanical actions inducing a Valsalva maneuver, sneezing, coughing, lifting heavy objects, vomiting, constipation, sexual activity, and playing wind instruments.[16,17] When these mechanical causes can be ruled out, one must then consider systemic diseases and medications particularly in the presence of diffuse SCH. Although not true in the case of our athletes, in subjects over age 50 especially, hypertension becomes a major risk factor for SCH.[15] Other systemic conditions associated with SCH include hyperthyroidism, diabetes, hyperlipidemia, anemia, blood clotting disorders like von Willebrand's disease, and chronic anticoagulation therapy[19]. Conjunctival conditions such as primary conjunctival amyloidosis (aka Kawasaki disease) and conjunctival tumors may result in SCH.[16,19] Also, both bacterial and viral conjunctival infections can cause small patches of SCH called petechial hemorrhages.[19]

The small extent and inferior-temporal location of the SCH found in our athlete is consistent with the conclusions of Mimura's study.[15] The study found that traumatic SCH tended to occur in younger patients and involve less conjunctival areas than systemic etiologies; also such lesions more frequently localize in temporal rather than nasal areas.[15] Also, the study determined that overall SCH is found more often in the inferior aspect in comparison to superior.[15] In a healthy individual with a SCH, patient education, palliative treatment like cold and warm compresses, avoidance of blood-thinning agents, and restriction of strenuous activity are indicated.[17] Recurrent episodes of SCH, especially when there is a negative history of ocular trauma, warrant further investigation into a patient's systemic health.[16,17] This athlete (JO) was

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

examined the following year and the SCH had completely resolved.

RETINAL SCARS AND ABNORMALITIES

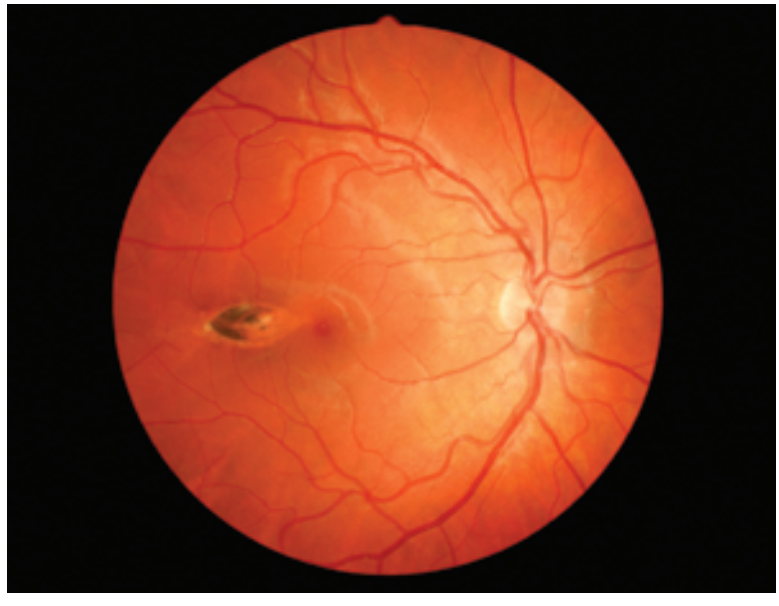
Athlete TW presented with a football-shaped, horizontally elongated chorioretinal scar about one and a half disc diameters long and adjacent to the macular region temporally in the right eye.[20] (Figure 3A) Chorioretinal scars are a common, but nonspecific, clinical finding that can be the result of many conditions.[21] The list of potential causes include, but are not limited to, the following: age-related macular degeneration, geographic atrophy of the retinal pigment epithelium, old idiopathic central serous chorioretinopathy, presumed ocular histoplasmosis syndrome, ocular toxoplasmosis, acute posterior multifocal placoid pigment epitheliopathy, serpiginous choroidopathy, multifocal choroiditis, birdshot retinochoroidopathy, pattern dystrophies of the retinal pigment epithelium, acute retinal pigment epitheliopathy, and punctate inner choroidopathy.[21] A recent paper by Shields et al suggests that this lesion is "torpedo maculopathy."[20] This congenital defect closely resembles solitary CHRPE but differs in its non-random macular location and pointed torpedo shape.[22–25]

Athlete WR presented with an area of superficial retinal scarring adjacent to the macular area temporally in the right eye. (Figure 3B) The lesions are comprised of multiple circular spots of hypopigmentation with each spot encircled by mild pigmentation. Several photos were taken to demonstrate that observation was not a photographic artifact. A careful search of the literature revealed no specific name for the appearance of this lesion other than an old choreo-retinal scar of unknown etiology.

Cherry Red Spot

One athlete (BP) presented with bilateral cherry-red spots which is a typically concerning retinal sign, but after evaluation of the fundus photos, and clinical correlation, we can describe this finding as a "pseudo cherry-red spot" (Figure 3C). The view we have of the fundus in both eyes shows the healthy retinas of a young person with no evidence of retinal pathology that is commonly associated with this sign such as central retinal artery occlusion or neurometabolic disorders.[26–28] These accompanying disease processes affect the ganglion cell layer represented by signs of retinal opacification or whitening in the posterior pole and all of the retina surrounding a cherry red fovea.[28] The classic neurometabolic disorder, Tay-Sachs disease,

FIG. 3A A football-shaped horizontally elongated chorioretinal scar about one and a half disc diameters long and adjacent to the macular region temporally in the right eye. (Athlete TW)



This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

FIG. 3B An area of superficial retinal scarring adjacent to the macular area temporally in the right eye. The lesions present with multiple circular spots of hypopigmentation with each spot encircled by mild pigmentation. (Athlete WR)

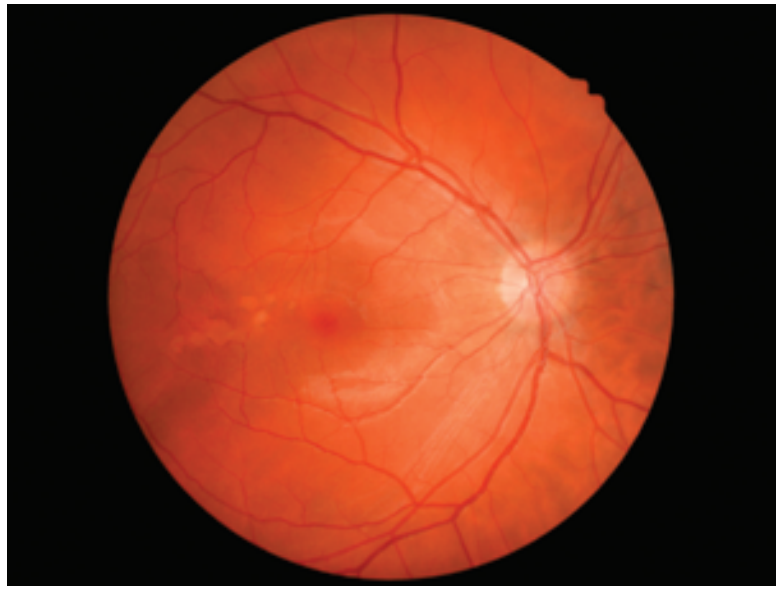
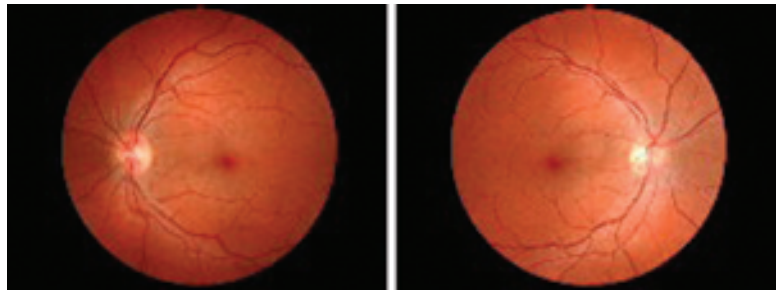


FIG. 3C Bilateral cherry-red spots at the macula of each eye. (Athlete BP)



results in an accumulation of metabolic products in the ganglion cell layer that gives the retina a pale, milky appearance.[26,27] Other commonly encountered metabolic disorders with the same presentation include Sandhoff disease, gangliosidosis, Niemann-Pick disease, sialidosis, and Farber disease.[26,27] Similar in appearance, the opaque and swollen retinal manifestations of central retinal artery occlusions are caused by edema and cellular necrosis.[26]

The reason for the difference in appearance between the fovea and the surrounding retina is the fact that the central fovea is devoid of the nerve fiber and ganglion cell layers.[29] Therefore, the color of the fovea is a result of the underlying retinal pigment epithelium and the vascular choroidal layer.[26,27] This being said, Ospina et al. suggest that a red fovea occurs only in individuals with a Caucasian background because pigment epithelium varies with race and skin color.[27] In this case, the finding of bilateral pseudo cherry-red spot in an otherwise healthy Caucasian athlete is explained by the accompanying blonde fundus that is common to individuals with light hair and fair skin. The athlete's less pigmented blonde fundus contrasts the prominent choroidal vasculature underlying the foveal area, which contributes to the bold red coloration of the fovea. No treatment was required for this athlete and he has been examined in subsequent years and there was no change to his retinal appearance.

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

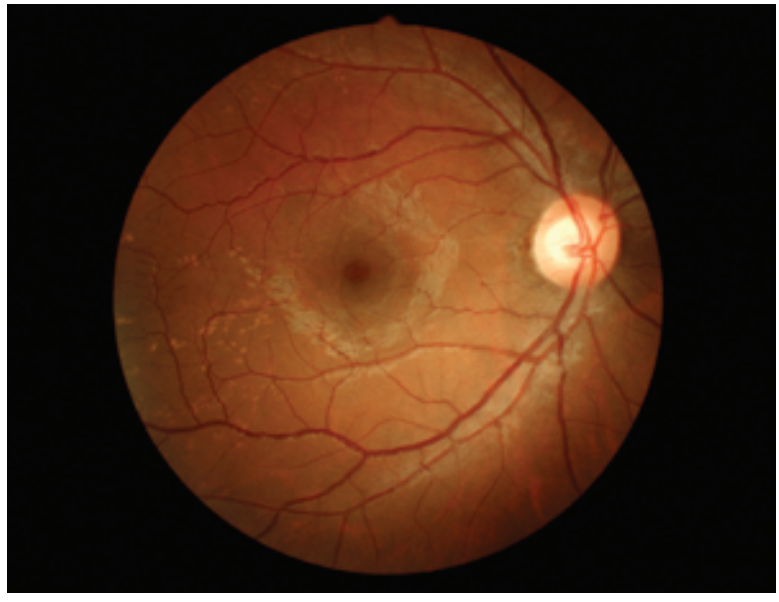
Drusen

We found that one athlete (DB) had numerous small "hard" drusen scattered temporal to the macula in the right eye only (Figure 3D). Several photos were taken to demonstrate that this retinal finding was not a photographic artifact. The concern regarding the presence of this type of drusen in a young individual is the possibility that it is a precursor for the development of age-related macular degeneration (AMD) and/or age-related maculopathy (ARM). As of a 1999 study[30], the significance of small hard drusen in younger subjects with normal visual function remains ambiguous. Studies do confirm that small hard drusen are prevalent in healthy young and middle-aged subjects,[31,32] and that numerous hard drusen can predict the incidence of soft drusen and retinal pigmentation abnormalities.[33] Munch et al. examined twins between the ages of 24 and 46 and determined that number of drusen had no statistically significant association with sex, smoking, serum lipids, fasting blood glucose, or blood pressure.[34] The most important conclusion from this study was that young and middle-aged adults having more than 20 drusen per eye is a highly heritable feature.[34] The highest reported heritability for an ophthalmic trait is 0.99 for the phenotype greater than or equal to 20 small hard drusen.[35] This finding was the result of an examination of existing literature regarding heritability of a variety of ocular traits as it relates to different conditions such as glaucoma, refractive error, AMD, diabetic retinopathy and cataracts.[35] The strong genetic component of the prevalence of drusen is not exclusive to younger populations. Hammond et al.[36] report a similarly high heritability of 0.81 of having more than 20 hard drusen per eye in female twin pairs between the ages of 49-79. Since our athlete has considerably more than 20 drusen it is presumed that the etiology of these drusen is heredity. This conclusion, of course, is an assumption because we have no data on this athlete's parents. It is also a bit unusual for a hereditary condition to be present only in one eye.

OPTIC NERVE CHANGES

One athlete (WM) appears to be a glaucoma suspect secondary to an abnormally large cup-to-disc ratio of 0.50/0.60 (horizontal/vertical) in the right eye only (Figure 4). At the time of the screening, the athlete presented with a normal IOP measurement. The reported range of cup to disc ratios in the normal population is between 0.00 (no cupping) to 0.87,[37,38] and most fall below 0.50.[39] Therefore, because of the literature and the authors' clinical experiences, our athlete's estimated cup to disc ratio by fundus photography is considered borderline abnormal. The foremost clinical suspicion for this common finding

FIG. 3D Numerous small "hard" drusen scattered temporal to the macula in the right eye only. (Athlete DB)



This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

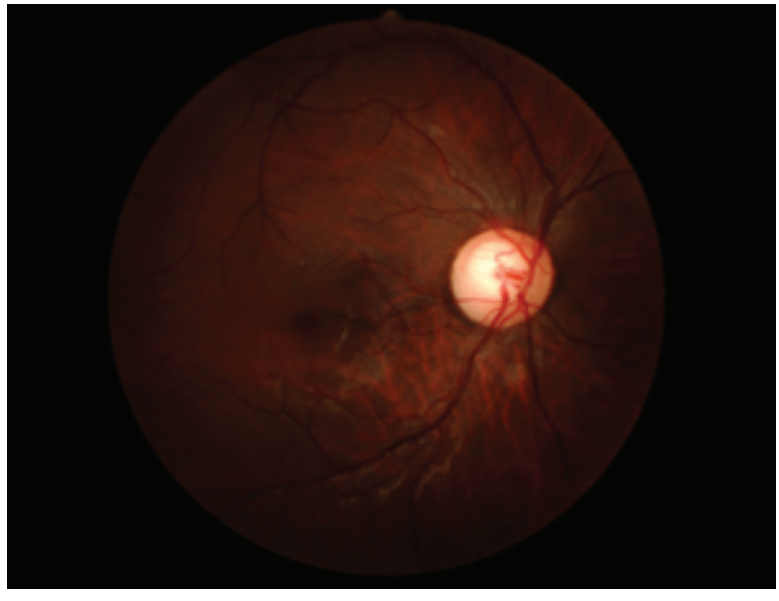
of an abnormally large cup to disc ratio, is glaucomatous cupping, especially when associated with a larger vertical measurement than the horizontal and asymmetric cupping in the fellow eye, both of which are features our athlete displays. Additional causes of large cup to disc ratios include optic neuropathies such as optic neuritis, ischemic optic neuropathy, and compressive neuropathy; alcohol consumption; a history of steroid treatment with secondary increased IOP; burned-out glaucoma; and congenital features including optic nerve pit.[40,41]

In the presence of a larger than a normal cup to disc ratio, several measurements and tests are needed to determine the precise etiology. Repeated IOP measurements and visual field testing are standard and help differentiate normal-tension from high-pressure glaucoma. According to Kesler et al. a full-day diurnal curve should be a required part of the workup which, in their study, is defined by around-the-clock IOP measurements starting at 7 A.M. and ending at 10 P.M.[37] Systemic and neurological evaluations are then indicated to rule out optic neuropathies. Integrating ocular imaging technology to examine optic nerve head parameters and the retinal nerve fiber layer (RNFL)[42] has proven to be relevant in confirming physiologic large cups. Min et al. determined that RNFL thickness obtained by optical coherence tomography (OCT) reveals significant differences between glaucomatous and physiological large cup eyes; normal and physiological large cup eyes showed no significant difference whereas glaucomatous eyes had thinner RNFL thickness. Min et al. also found that the neuroretinal rim area is an important optic nerve head parameter that is not significantly different between normal and physiological large cup eyes but is reduced in glaucomatous eyes due to loss of retinal ganglion cell axons.[38] While there are many factors to consider in a workup, a study by Jonas et al. discovered that the single most valuable optic disc variable to evaluate and monitor early glaucomatous changes in the vertical cup to disc diameter ratio corrected for optic disc size which reflects the natural pattern of neuroretinal rim loss that begins in the superior and inferior disc areas.[39] If IOP measurements, visual field testing, systemic and neurological workups are normal then physiological large cups may be considered the etiology.[37]

VASCULAR CHANGES

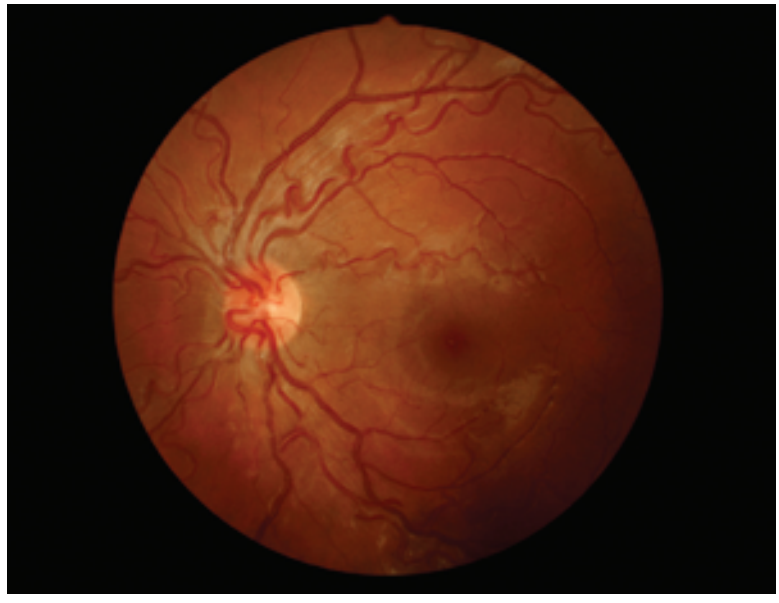
Fundus photography revealed that one athlete (BR) had bilateral tortuous retinal vessels (Figure 5). The results of a study by TaarnhØj et al. on healthy,

FIG. 4 An abnormally large cup-to-disc ratio of 0.50/0.60 (horizontal/vertical) in the right eye only (Athlete WM).



This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

FIG. 5 Bilateral tortuous retinal vessels (Athlete BR).



normotensive twin pairs, aged 20 to 46 years, provides a clinical perspective for this finding.[43,44] The study concluded that there is a large variation in retinal arterial tortuosity in young, healthy subjects that can be attributed to a strong genetic influence as is evidenced by heritability of 82% for this ocular trait. In the literature, there is no standard method for measuring vessel tortuosity. The study used the same means that we used to judge vessel tortuosity, which is the visual grading of fundus images. TaarnhØj et al. showed that numerous cardiovascular risk factors like age, sex, systolic blood pressure, fasting blood glucose, total cholesterol, low-density, and high-density lipoprotein, triglycerides, smoking, and retinal artery and vein diameter have no statistically significant association with vessel tortuosity in a young, healthy study population.[44,45] While our athlete demonstrating vessel tortuosity is normotensive, as are the subjects in the TaarnhØj et al. study, observations of tortuosity in retinal arteries have typically been associated with hypertension.[46,47] Premature birth has also been found to be responsible for permanent increases in tortuosity of both retinal arteries and veins.[48] Overall there is little diagnostic value from a single, clinical assessment of retinal arterial tortuosity. Repeated fundus photographs over time for comparison provides a more effective evaluation of the potential impact of systemic health parameters on ocular morphology.[44]

CONCLUSION

Our goal in highlighting these findings is to raise awareness among eye care practitioners of the importance of eye health screening as part of a sports vision evaluation. These eye findings should not be considered "incidental" as they are being intentionally sought for as part of a complete sports vision evaluation. If it is impractical to do a dilated fundus examination as part of sports screenings, it is important to use advanced technology such as a non-myd fundus camera to screen for abnormalities in the posterior segment.

Periodic eye and vision examinations are an important part of preventive health care. Many eye and vision problems have no obvious signs or symptoms, as has been highlighted in this paper. As a result, individuals are often unaware that problems exist. Early diagnosis and treatment of eye and vision problems are important for maintaining good vision and eye health, and when possible, preventing vision loss. Healthcare practitioners all recommend periodic examinations and the frequency of those examinations increases with age.

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

DISCLOSURE

No conflicts of interest.

ACKNOWLEDGEMENT

Kowa-Optimed (Hawthorne, CA) donated the use of the non-mydriatic fundus camera.

REFERENCES

1. Gottleib JL, Sahel JA, Albert DM. Choroidal nevi. In: Ryan SJ, ed. Retina. 4th ed. Philadelphia: Elsevier/ Mosby; 2006:679-690.
2. Sahel JA, Marcus DM, Albert DM. Nevus and melanocytoma. In: Albert DM, Jakobiec FA, eds. Principles and practice of ophthalmology : clinical practice. Vol 5. Philadelphia: Saunders; 1994:3244-3251.
3. Sumich P, Mitchell P, Wang JJ. Choroidal nevi in a white population: the Blue Mountains Eye Study. Arch Ophthalmol 1998;116(5):645–50.
4. Naumann GOH, Hellner K, Naumann LR. Pigmented nevi of the choroid: clinical study of secondary changes in the overlying tissues. Trans Am Acad Ophthalmol Otolaryngol. 1971;75:110–23.
5. Ganley JP, Comstock GW. Benign nevi and malignant melanomas of the choroid. Am J Ophthalmol 1973;76(1):19–25.
6. Deutsch TA, Jampol LM. Large druse-like lesions on the surface of choroidal nevi. Ophthalmology 1985;92(1):73–76.
7. Gass JD. Problems in the differential diagnosis of choroidal nevi and malignant melanoma. XXXIII Edward Jackson Memorial lecture. Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol 1977;83(1):19–48.
8. Tamler E, Maumenee AE. A clinical study of choroidal nevi. A.M.A. Arch Ophthalmol 1959;62(2):196–202.
9. Augsburger JJ, Schroeder RP, Territo C, Gamel JW, Shields JA. Clinical parameters predictive of enlargement of melanocytic choroidal lesions. British J Ophthalmol 1989;73(11):911–17.
10. Shields CL, Mashayekhi A, Materin MA, et al. Optical coherence tomography of choroidal nevus in 120 patients. Retina (Philadelphia, Pa.). 2005;25(3):243–52.
11. Seddon JM, Moy CS. Epidemiology of uveal melanoma. In: Ryan SJ, ed. Retina. 3rd ed. St. Louis: Mosby; 2001:664–72.
12. Holly EA, Aston DA, Char DH, Kristiansen JJ, Ahn DK. Uveal melanoma in relation to ultraviolet light exposure and host factors. Cancer Res 1990;50(18):5773–77.
13. Singh AD, Rennie IG, Seregard S, Giblin M, McKenzie J. Sunlight exposure and pathogenesis of uveal melanoma. Survey Ophthalmol 2004;49(4):419–28.
14. Lowenstein JI. Retinopathy associated with blood anomalies. In: Albert DM, Jakobiec FA, eds. Principles and practice of ophthalmology : clinical practice. Vol 2. Philadelphia: Saunders; 1994:995–1000.
15. Mimura T, Yamagami S, Usui T, et al. Location and extent of subconjunctival hemorrhage. Ophthalmologica. Journal international d'ophtalmologie. International journal of ophthalmology. Zeitschrift fur Augenheilkunde. 2010;224(2):90–95.
16. Sowka JW, Gurwood AS, Kabat AG. Subconjunctival hemorrhage. Handbook of Ocular Disease Management. 12 ed: Review of Optometry; 2010:18A–19A.
17. Kabat AG, Sowka JW. Something for 'nothing' Part 2. Review of Optometry. 2006;143(11):131–32.
18. Fukuyama J, Hayasaka S, Yamada K, Setogawa T. Causes of subconjunctival hemorrhage. Ophthalmologica. Journal international d'ophtalmologie. International journal of ophthalmology. Zeitschrift fur Augenheilkunde. 1990;200(2):63–67.
19. Skorin L. Subconjunctival hemorrhages: not all of them are benign. Opto Today 2006:32–34.
20. Shields CL, Guzman JM, Shapiro MJ, Fogel LE, Shields JA. Torpedo maculopathy at the site of the fetal "bulge". Arch of Ophthalmol 2010;128(4):499–501.
21. Teitelbaum BA, Hachey DL, Messner LV. Torpedo maculopathy. Journal of the American Optometric Association. 1997;68(6):373-376.
22. Angioi-Duprez K, Maalouf T. [Torpedo maculopathy]. Journal francais d'ophtalmologie. 2000;23(2):200.
23. Rigotti M, Babighian S, Carcereri De Prati E, Marchini G. Three cases of a rare congenital abnormality of the retinal pigment epithelium: torpedo maculopathy. Ophthalmologica. Journal international d'ophtalmologie. International journal of ophthalmology. Zeitschrift fur Augenheilkunde. 2002;216(3):226-227.
24. Mahieu L, Mathis A. ["Torpedo" maculopathy]. Journal francais d'ophtalmologie. 2003;26(5):533.
25. Yang IL. Differentials of chorioretinal scarring. Clin Eye Vision Care 1994;6(4):192–202.
26. Institute MCsB. Cherry red spot. https://braininstitute.mch.com/wiki/Cherry_red_spot - Pseudo_cherry_red_spot. Accessed November 21, 2012.
27. Ospina LH, Lyons CJ, McCormick AQ. "Cherry-red spot" or "perifoveal white patch"? Can J Ophthalmol 2005;40(5):609–610.

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.

28. Ehlers JP, Shah CP. Central retinal artery occlusion. In: Ehlers JP, Shah CP, eds. The Wills Eye Manual : Office And Emergency Room Diagnosis and Treatment of Eye Disease. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:282–84.
29. Ozdemir H, Karacorlu S, Karacorlu M. Optical coherence tomography findings in central retinal artery occlusion. Retina (Philadelphia, Pa.). 2006;26(1):110–112.
30. Sarks SH, Arnold JJ, Killingsworth MC, Sarks JP. Early drusen formation in the normal and aging eye and their relation to age related maculopathy: a clinicopathological study. The British J Ophthalmol 1999;83(3):35868.
31. Bressler NM, Bressler SB, West SK, Fine SL, Taylor HR. The grading and prevalence of macular degeneration in Chesapeake Bay watermen. Arch Ophthalmol 1989;107(6):847–52.
32. Klein R, Klein BE, Linton KL. Prevalence of age-related maculopathy. The Beaver Dam Eye Study. Ophthalmology 1992;99(6):933–43.
33. Klein R, Klein BE, Tomany SC, Meuer SM, Huang GH. Ten-year incidence and progression of age-related maculopathy: The Beaver Dam Eye Study. Ophthalmology 2002;109(10):1767–79.
34. Munch IC, Sander B, Kessel L, et al. Heredity of small hard drusen in twins aged 20-46 years. Investigative ophthalmology & visual science. 2007;48(2):833–38.
35. Sanfilippo PG, Hewitt AW, Hammond CJ, Mackey DA. The heritability of ocular traits. Survey Ophthalmology 2010;55(6):561–83.
36. Hammond CJ, Webster AR, Snieder H, Bird AC, Gilbert CE, Spector TD. Genetic influence on early age-related maculopathy: a twin study. Ophthalmology 2002;109(4):730–36.
37. Kesler A, Kotas-Neuman R, Kott E, Assia E. Evaluation of patients with large Cup: Disc ratio of the optic nerve head. Ann Ophthalmol 2000;32(1):46–49.
38. Min KH, Seong GJ, Hong YJ, Kim CY. Optic nerve head topographic measurements and retinal nerve fiber layer thickness in physiologic large cups. Korean J Ophthalmol: KJO 2005;19(3):189–94.
39. Leibowitz HM, Krueger DE, Maunder LR, et al. The Framingham Eye Study monograph: An ophthalmological and epidemiological study of cataract, glaucoma, diabetic retinopathy, macular degeneration, and visual acuity in a general population of 2631 adults, 1973–1975. Survey Ophthalmol 1980;24(Suppl):335–610.
40. Jonas JB, Naumann GOH. The optic nerve in glaucoma: its embryology, histology, and morphology. In: Varma R, Spaeth GL, Parker KW, eds. The Optic Nerve In Glaucoma. Philadelphia: J.B. Lippincott; 1993:3–26.
41. Airaksinen PJ, Tuulonen A, Werner EB. Clinical evaluation of the optic disc and retinal nerve fiber layer. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas. St. Louis: Mosby; 1989:3–26.
42. Quigley HA, Miller NR, George T. Clinical evaluation of nerve fiber layer atrophy as an indicator of glaucomatous optic nerve damage. Arch Ophthalmol 1980;98(9):1564–71.
43. Jonas JB, Bergua A, Schmitz-Valckenberg P, Papastathopoulos KI, Budde WM. Ranking of optic disc variables for detection of glaucomatous optic nerve damage. Investigat Ophthalmol Vis Sci 2000;41(7):1764–73.
44. Taarnhoj NC, Munch IC, Sander B, et al. Straight versus tortuous retinal arteries in relation to blood pressure and genetics. British J Ophthalmol 2008;92(8):1055–60.
45. Williams TD. Quantification of arteriolar tortuosity in two normotensive age groups. American J Optometry Physiolog Optics 1982;59(8):675–79.
46. de Margerie J, Boyd TA. A statistical investigation of the correlation of retinal arteriolar tortuosity with blood pressure and age. Trans Canadian Ophthalmologic Soc 1961;24:6-17.
47. Wolffsohn JS, Napper GA, Ho SM, Jaworski A, Pollard TL. Improving the description of the retinal vasculature and patient history taking for monitoring systemic hypertension. Journal British Coll Ophthalmic Opticians 2001;21(6):441–49.
48. Fahrni R, Thalmann J, Weber J, von Muralt G, Sidiropoulos D, Bracher D. Central retinal arteries in the full-term newborn: decrease in width and tortuosity during uneventful adaptation. Helvetica paediatrica acta 1981;36(2):107–21.

This article is distributed under the terms of the Creative Commons Attribution-Non Commercial 4.0 International License. ©Kirschen and Laby.