Ruptured globe from badminton racquet injury while wearing spectacles

Dear Editor:

We commend the work performed by Luong et al.[1] regarding their description of traumatic hyphemas sustained during badminton play and the need to review the current state of protective eyewear while playing sports. We agree with their recommendation to require eye protection in badminton. To further support the need for protective eyewear, we report a ruptured globe injury in a child sustained from blunt trauma when a badminton racquet shattered his spectacles.

A 9-year-old male was referred to our department for surgical evaluation of a left traumatic globe injury sustained while participating in recreational doubles badminton. He wore spectacles to correct a refractive error, but no protective eyewear was worn. The patient presented with 20/20 vision OD and hand motion vision OS. Examination of the right eye was unremarkable. Examination of the left eye demonstrated temporal chemosis and a zone II full-thickness laceration extending from the central cornea temporally into the sclera with iris prolapse. There was no violation of the lens capsule and no intraocular foreign body. The patient underwent ruptured globe repair and was discharged home on postoperative day 3 with 20/800 vision. The remainder of his follow-up care was performed out of state.

Our case illustrates the need for protective eyewear even when wearing spectacles. Regular prescription eyeglasses do not conform to the American Society for Testing and Materials F803 (ASTM F803) specifications for protective eyewear in sports,[2] and corneoscleral lacerations have been reported in badminton players with glass spectacles.[3] Although the material of the lens or frame was not known in our patient, plastic or polycarbonate lenses are frequently prescribed. Direct racquet impact on spectacles may cause a different spectrum of injury with an increased potential for shattering lenses, penetrating injury, and blunt or transferred impact from the spectacles to the bridge of the nose and surrounding orbital bones.[4]

A joint statement by the American Academy of Pediatrics and the American Academy of Ophthalmology recommended that all youth participating in organized sports wear eye protection.[2] In the specific case of individuals who wear spectacles, they recommend several options, including polycarbonate lenses in a sports frame that passes the ASTM F803 standards, contact lenses with appropriate sport-specific protective eyewear, or an over-the-glasses eyeguard that confirms to the ASTM F803 standards.[2] To the best of our knowledge, in the United States, there are no mandatory requirements for eye protection in badminton. Further research is required to understand the hazards encountered in specific racquet sports and to determine the appropriate level of protective eyewear. Until there are further official recommendations regarding protective eyewear while playing badminton, we agree with the proposal by Luong et al. that protective eyewear be worn in badminton,[1] even in the case of participants who wear spectacles.

Disclosure: The authors have no proprietary or commercial interest in any materials discussed in this article.

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Can J Ophthalmol 2017;52:625
0008-4182/17/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2017.04.015

Exudative complications following photodynamic therapy

Dear Editor:

We read the interesting, previously unreported case report by Al-Awadi et al. titled "Atypical transient subretinal exudation following photodynamic therapy for chronic central serous retinopathy: a case report" published in the February issue.[1] Although many series have reported photodynamic therapy (PDT) induced significant subretinal exudation resulting in bullous subretinal fluid and fibrin in the immediate post-treatment period in cases of classic or occult choroidal neovascular membrane

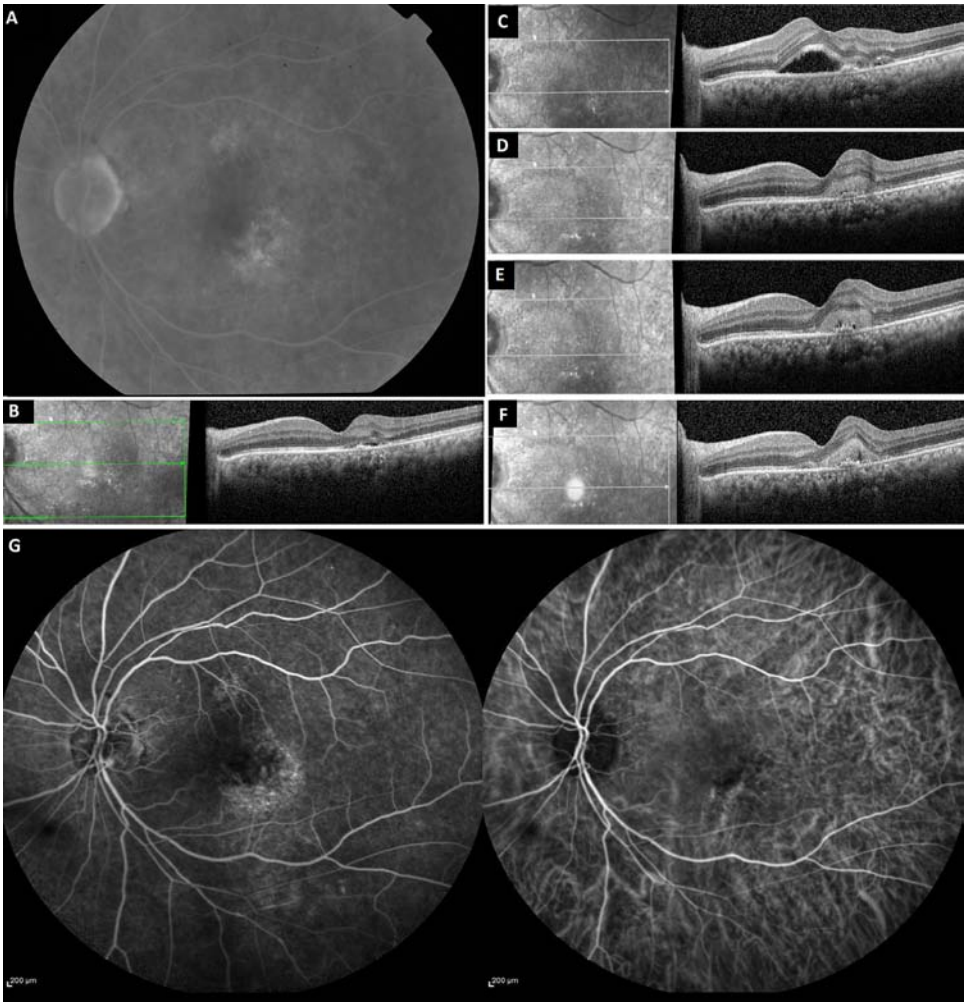

Fig. 1—(A) Fluorescein angiography of a 67-year-old male who presented with a history of persisting central serous chorioretinopathy post–focal laser and best corrected visual acuity (BCVA) 6/12, showing diffuse parafoveal leakage around focal laser scar in the left eye (LE). (B) Optical coherence tomography (OCT) LE showing shallow subretinal fluid and small juxtafoveal Pigment epithelial detachment (PED) with central macular thickness 193 μm. (C) OCT at 1 week post–photodynamic therapy (post-PDT) showing serofibrinous exudation with central macular thickness 375 μm, with maximum increase in subretinal fluid (289 μm) noted at infrafoveal region, BCVA 6/36. (D) At 6 weeks post-PDT, OCT picture returned to baseline status (central macular thickness 193 μm) with persisting subretinal fibrin clump, BCVA 6/12. (E, F) OCT at 14 weeks (E) and 24 weeks (F) post-PDT showing persisting fibrin with new subretinal fluid pockets, BCVA 6/18. (G) Fluorescein angiography and Indocyanine green angiography at 24 weeks post-PDT showing diffuse oozing of dye, and polyp with branched vascular network, respectively, for which repeat photodynamic therapy was advised.

(CNVM) and intraocular tumours,[2,3] it is still unreported in chronic central serous chorioretinopathy (CSC). We have noted this PDT-induced complication in several cases with very low incidence rate. All of them had the diagnosis of either CNVM or polypoidal choroidal vasculopathy (PCV). None of the cases with chronic CSC showed this exaggerated response after PDT, except 1 case, which later turned out to be a case of PCV on Indocyanine green angiography (Fig. 1).

As the authors have discussed, the exact mechanism of PDT-induced exaggerated exudation resulting from the breakdown of blood–retinal barrier or retinal pigment epithelium (RPE) pump dysfunction is unknown.[1] Histopathological study in human eyes has shown PDT-induced damage to choroidal vasculature characterized by swelling and detachment from basement membrane of vascular endothelial cells leading to endothelial degeneration, resulting in filling of capillary lumina with cellular debris, fibrin, and thrombocytes.[4] Sub-RPE neovascularization can also predispose the RPE cells to unusual damage by causing upregulation in low-density lipoprotein receptors and/or by making the cells less resistant to oxidative stress.[2] Another possible mechanism is post-PDT angiogenic response due to inflammatory surge of vascular endothelial growth factor.[3] All previously reported cases of post-PDT excessive exudation have been reported

in CNVM, PCV, or intraocular tumours, and all of these pathologies are characterized by presence of abnormal vascular channels or network, unlike in CSC cases. We believe that PDT-induced tissue damages and VEGF surge are accentuated in conditions with abnormal vascular tissue (pre-existing angiogenic or neovascular conditions) resulting in excessive vascular permeability and consequently excessive exudation. This could be the factor responsible for differential incidence of post-PDT exudation with a higher incidence in CNVM, PCV, and intraocular tumours.

Although Al-Awadi et al. have ruled out the presence of CNVM or polyp, a close association of chronic CSC with type 1 CNVM as well as polyp is well described as part of the pachychoroid spectrum.[5] Hence, the case reported by them could possibly be chronic CSC with evolving angiogenic tissue in the form of polyp or CNVM, and should be closely followed up for the development of the same, especially if the patient shows signs of recurrent activity.

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Can J Ophthalmol 2017;52:625–627
0008-4182/17/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2017.05.013

Response to "Exudative complications following photodynamic therapy"

Dear Editor:

I would like to thank Ranjan et al. for submitting a stimulating response to what is indeed a unique case report.

We agree that the previously unreported case report titled "Atypical transient subretinal exudation following photodynamic therapy for chronic central serous retinopathy"[1] represents a unique challenge in describing underlying mechanisms.

The postulated mechanisms submitted by Ranjan et al. to explain an exaggerated exudative response after photodynamic therapy (PDT), as well as the caution to follow such patients closely for a potential evolution to a choroidal neovascular membrane (CNVM) or polyp, are indeed valuable and appreciated. However, this case does highlight that a post-PDT response can occur in the absence of any CNVM or polyp, albeit rarely. Interestingly, in addition to the index case being stable with no evidence of recurrence, CNVM, or polyp for more than 2 years after PDT, the author has observed a second case with a similar transient exudation after PDT for chronic CSR with complete resolution (Fig. 1).

These observations suggest that other mechanisms also need to be explored and critically appraised. Perhaps the large choroidal vessels situated in Haller's layer, which are part of the pachychoroid spectrum,[2] are abnormal enough to drive this response, independent of the presence of a CNVM or polyp. Other factors that may also play a role in the differential incidence include either a dose-dependent response or an idiosyncratic-type reaction similar to that reported for verteporfin infusion–associated pain.[3] Interestingly, both the index case and the case illustrated in Figure 1 involve half-dose verteporfin, which perhaps may be less proinflammatory than the standard verteporfin doses used in cases involving polyps or CNVM.

Nonetheless, this post-PDT response, regardless of mechanism, is uncommon and warrants further study into the pachychoroid spectrum of diseases.[4]

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