2

MANAGEMENT OF THE RETINAL BREAK TREATMENT IN A COACH WHO IS FORMER GOALKEEPER

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Introduction

Retinal breaks are full-thickness defects in the neural retina that occur secondary to vitreous traction. The prevalence of retinal breaks aged 10 years or more with no known antecedent ocular disease is in the range about 11% (3). Blunt traumas also increase the risk for development of retinal breaks as a result of vitreous traction on the peripheral retina (2). All ocular conditions with the extent of vitreoretinal adhesions are associated with a higher incidence of retinal detachment. The usual pathologic sequence that results in the retinal detachment is vitreous liquefaction followed by a Posterior Vitreous Detachment (PVD), which in turn causes a retinal break at the site of a significant vitreoretinal adhesion. Symptoms associated with acute retinal break include floaters secondary to vitreous debris (hemorrhage, retinal pigment epithelium cells) and flashes that result from persistent vitreous traction. Timely recognition of the symptoms and signs of retinal tears is important to maximize the chances of favorable outcomes and preserve visual acuity. Nearly all symptomatic rhegmatogenous retinal detachments progress to total blindness unless they are repaired successfully. The age, location, and size of the retinal break, the status of the fellow eye, return to play should be considered and discussed with all patients before treatment, especially in athletes. In this case study, we report the identification of risk factors of retinal breaks and discuss the appropriate management strategy.

Case report

Sixty years old healthy male patient, who is a coach and former professional goalkeeper, complained of floaters and flashes in his right eye. His all complaints began suddenly on the same day during the training session. He had many previous head and blunt ocular traumas in his past but not that day. His uncorrected visual acuity was 20/20 in both eyes. Following the comprehensive ophthalmic examination retinal tear associated with lattice degeneration at the superior temporal quadrant in fundus examination of his right eye was detected by fundus examination. There was PVD identified by slit lamp fundus biomicroscopy with a 90 diopter lens and by optical coherence tomography. Laser photocoagulation treatment was performed by the panfundoscope lens on the slit-lamp delivery system with topical anesthesia. The tear was surrounded completely by three rows of Laser burns. The settings were 200 µm spot size and 0.1 s application time. There was no need for additional topical or systemic treatment. Vigorous patient activity was discouraged initially. After 10 days of the Laser photocoagulation treatment, chorioretinal adhesion started to occur. A firm chroioretinal adhesion was present after three weeks. During the follow up period of five years, there was no new break or need an additional procedure to prevent or repair a retinal detachment or epiretinal membrane formation.

Conclusions

A big part of the brain is directly or indirectly involved in visual processing and, many central nervous system diseases and chronic traumatic encephalopathy can be detected on the retina level (1). Awareness of the effects of heading on human body is on the rise, but some related problems have been overlooked in clinical practise. Things may not be seen unless specifically looking for them. We conclude that the fundus examination may be added to athlete (players and ex-players) health assessment protocols even with 20/20 best corrected visual acuity, because retinal detachment is not just a boxer disease.

References

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