Pathomechanism of traumatic indirect choroidal rupture

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ABSTRACT

The probable chain of events responsible for choroidal rupture is as follows. During high-speed orbital injuries, the protective ocular reflexes position the eye in an elevated, and abducted position. At this point in time, the anteroposterior compressive forces on to the globe create an eccentrically positioned circle of damaging currents along the posterior ocular coats against a relatively static optic nerve. Because of this eccentricity, a longer radius of curvature is expected to lie along the temporal half of the globe leading to an elastic recoil of the retinal and scleral layers and a fracture along the RPE-Bruch’s-Choriocapillaris complex manifesting clinically as choroidal rupture.

Introduction

Post-traumatic choroidal rupture is a common posterior segment complication following a blunt trauma. It can be of two types: direct and indirect [1–3]. Direct choroidal ruptures are usually located along the site of direct trauma, that is, along the ora serrata and peripheral retina. In contrast to this, the indirect choroidal ruptures are usually seen along the posterior pole or at the site of counter coupé injury. The exact mechanism of indirect choroidal rupture is not known. Therefore, in this report, we systematically elaborate the probable chain of events responsible for indirect choroidal rupture with a simple experiment.

Mechanism

In cases of blunt ocular trauma, there is a rapid transmission of anteroposterior compressive forces along the ocular coats. During the process of trauma, impact and the consequences, there are few important physiological functions which are always there to protect the eyes. Just moments before the target hits the eyes/orbit, protective Bell’s phenomenon will come into action to elevate, and abduct the eye so as to minimize the injury. However, even though the globe has entered a safe zone the impact related to anteroposterior compressive force continues to act. Under such circumstances the globe absorbs the compressive forces, leading to the posterior displacement of the globe and extraocular muscle. During this rapid change in the globe position with respect to the horizontal, vertical and anteroposterior meridians, the optic nerve appears to remains less disturbed in its anatomical position with minimal/absent torsional changes and infero-nasal displacement. Thus during the peak effect of compressive forces, the globe will be sandwiched between the compressive force anteriorly and optic nerve posteriorly. (Fig. 1A).

Now, this practical situation is explained with a balloon filled with carboxymethyl cellulose as the globe, which is being sandwiched between the punching force anteriorly and a relatively static optic nerve posteriorly. In normal anatomical position the optic nerve enters the globe on the nasal aspect it (Fig. 1A). With Bell’s reflex in action, the eye will become elevated, and abducted (Fig. 1B). Now in this position when the anteroposterior compressive force is transmitted onto the globe, the more eccentrically or nasally shifted optic nerve impinges to the globe. Thus creating a shearing force in a circular manner from the back of the globe in an asymmetric manner (Fig. 1C). This asymmetrically created circle will have a larger radius of curvature along the temporal aspect as compared to the nasal aspect of the globe. In some cases, the asymmetric circle of forces may occupy a more nasal area, which is in very close proximity to the optic nerve or over the edges of it (Fig. 1C).

Along these curvatures, the temporal retina receives the maximum pressure brunt due to the relative position of the globe with respect to reflexes as mentioned above. Thus the elastic structures such as retina and the sclera dissipate the tension by a rapid posterior passive stretch followed by an immediate recoil, but the brittle structures like Bruch’s-Choriocapillary complex fractures instead of an active recoil due to inherent stiff nature of the tissues. Thus the manifestation as choroidal rupture along the longer curvature (temporal retina) (Figs. 2 and 3).

Discussion

Choroidal rupture is an important vision-threatening complication
following blunt trauma. Previously in the literature some authors have explained the probable mechanism involved in this unfortunate complication, according to them, following trauma there is an anteroposterior compression of the globe with subsequent equatorial expansion leads to choroidal rupture, however, this is a generalized view, as the choroidal ruptures are anterior or posterior which are situated away from the equator [1,2]. Thus, this generalized concept does not explain the exact mechanism involved in this. In others view during the time of trauma due to anteroposterior compression there is a concentric transmission of the shearing force around the optic disc [1]. This seems logical but firstly the optic nerve is not situated exactly in the centre of the globe to produce concentric forces and secondly, it does not explain why the majority of choroidal ruptures are being situated temporally.

Thus to better explain the mechanism involved in the choroidal rupture, a small understanding regarding the physical properties of ocular tissues is necessary. As classically known, during the anteroposterior compression of the globe, the retinal tissue with its inherent elastic property and sclera with inherent rigidity tends to recoil back to their original positions following the removal of external deforming forces. But, the Bruch’s-Choriocapillary complex being inelastic in nature tends to fracture during the impaction of force.

As per our model, the damaging forces exerted onto the globe were in an asymmetric circle, that is the temporal ocular coats received the maximum stress in contrast to nasal coats leading to more tissue damage along this area, that is why around 82% of the choroidal rupture lesions are seen along the temporal half of the retina [1]. Similarly, if the stretching forces are acting more along the inferior or nasal or any other aspect of the retino-choroidal layers then that particular area is at risk of stress-driven damage.

The above hypothesis explains the majority of the findings observed among the noted cases in the literature and clinical practice. It also supports the previous findings of optic disc pallor associated with choroidal rupture [3]. As the optic nerve is the critical centre of force dissipation, it may be damaged to a varying extent depending upon the underlying position, force and other factors. However, the inverse and poor Bell’s phenomenon may explain the other unusual location of
indirect choroidal rupture.

What was known before

Antero-posterior compressive forces along the globe with an equatorial expansion and concentric forces along the optic disc are likely to lead to choroidal rupture.

What this study adds

In cases of blunt trauma to the eye, the protective Bell’s reflex positions the eye in an up and outwardly rolled position. In this position, the exerted anteroposterior compressive forces create an eccentrically positioned circle of forces with respect to the optic nerve. This induces a rapid posterior bowing of the ocular coats followed by a sudden recoiling, leading to a discontinuity along the brittle choroid. In addition, as the temporal retino-choroidal layers receive the maximum brunt of the stretch, this corresponds with clinically observed temporal choroidal rupture in a majority of the cases.

Conflict of interest

There are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.mehy.2019.02.010.

References