Pathomechanism of optic nerve avulsion

Amar Pujari⁎, Rohit Saxena, Swati Phuljhele, Karthika Bhaskaran, Shabeer Basheer, Pradeep Sharma

Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi 110029, India

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ABSTRACT

Optic nerve avulsion following peri-orbital trauma is an enigmatic clinical entity. Several mechanisms and ideas have been put forward to derive a logical conclusion, however, each factor independently does not appear to explain the mechanism in a logical way, therefore, here we elaborate the probable chain of events responsible for this complication. During isolated blunt trauma to the orbital framework, the globe continues to move anteriorly without any active resistance, in contrast to the globe, the optic nerve with more delicate bony and soft tissue relations, likely to remain relatively static. Thus the junction between the optic nerve and ocular coat suffers the maximum distractive injury due to anteroposterior tractional forces. In addition to this, physiological Bell’s phenomenon may induce torsional tension at this junction leading to further worsening of distractive forces and violent separation of optic nerve from the globe.

Introduction

Post-traumatic optic nerve avulsion is one of the dreaded complications of blunt ocular trauma. With growing urbanization and increasing vehicular mis-happenings, traumatic injuries are on the rise. Previously published reports and observations on the traumatic optic nerve avulsion have thought that a sudden rise in the intraocular pressures, sudden rotation of the globe, anterior luxation and other factors may be responsible for this unique complication [1–4]. However, these individual mechanisms need critical analysis with few basic anatomical considerations. Here in this report, we discuss the probable forces and ocular torsional changes which are responsible for the creation of avulsive tensions at the junction of optic nerve and posterior ocular coat.

Mechanism

In cases of high-velocity injuries to the periorbital structures, the impact of blunt trauma plays a vital role in the displacement of tissues within the orbit. During blunt trauma to the orbital margin, there is a protective Bell’s phenomenon which will come into action as a natural phenomenon. This induces certain changes in the anatomical orientation of the globe with respect to the horizontal, vertical and anteroposterior axis. Usually, the eye rolls up and out, and this happens at the level of the globe and the inserted rectus muscles, as the muscles are responsible for this neural mediated physiological change. The role of Bell’s phenomenon in the causation of traumatic indirect choroidal rupture [5], and the crucial role of orbital forces and ocular movements in the causation of extraocular muscle injuries has been highlighted by us previously [6].

The next important and significant tissue changes within the orbit are along the optic nerve. The optic nerve is a relatively fixed structure (with respect to the orbital soft tissue and bony structures) with a communication to the posterior ocular coat slightly on the nasal aspect, therefore, the rotational changes along the optic nerve are less pronounced as compared to the changes along the globe. In presence of strong deceleration forces directed along the bony framework, globe continues to move in an anterior direction as compared to the relatively static or less mobile optic nerve and orbital soft tissue complex. This anteroposterior traction onto the globe-nerve junction with added torsional tension will lead to easy separation of the optic nerve.

This can be explained by a simple experiment. A sample rubber ball with an attached band on one end is considered. To simulate the proposed forces, the ball is pulled in one direction and the optic nerve is pulled in the opposite direction so as to create an anteroposterior traction with induced extorsion (Fig. 1A, B). Following such rotation, the optic nerve and the globe will move in opposite directions, (Fig. 2) leading to new distractive tensions at the globe-nerve junction. Therefore, the junctional (tension) area formed by the reference points on the optic nerve and the sclera at the level of optic nerve entry into the posterior ocular coat receives the major brunt of torsion (Fig. 3). Thus each reference point on the optic nerve will lose its continuity with the...
corresponding scleral entry points leading to an easy separation of the two tissues. In this state of junctional discontinuity with lamina cribrosa being the weakest portion, the exerted torsion with an added anteroposterior traction easily separates the optic nerve from the globe leading to optic nerve avulsion (Fig. 4).

Discussion

Literature analysis shows that the optic nerve avulsion occurs through many undefined mechanisms. These are 1) due to a sudden rise in the intraocular pressure, leading to blowing off of the optic nerve, 2) sudden extreme rotation of the globe may be responsible for the separation but it is not clearly known how this factor plays the role. 3) Anterior globe luxation, and the 4) retro-pulsion of the optic nerve have been thought to be responsible for optic nerve avulsion [1–4]. However, by considering these individual ideas it may be difficult to logically explain the acceptable mechanism. Therefore, by considering the basic principles of these mechanisms in a combined manner, it is fairly possible to put forward the likely chain of events responsible for the optic nerve avulsion.

However, among these, the sudden rise in intraocular pressure leading to posterior blowing off the optic nerve appears to be of lesser significance. Because usually, we do not encounter optic nerve avulsion in a majority of cases with blunt trauma. Secondly the closed globe injuries with suddenly raised IOP usually leads to corneoscleral rupture, choroidal rupture, lens injury and other retinal manifestations, however, none of these features are co-existent with the reported cases of nerve avulsions. The optic nerve avulsion is mainly seen as dense hemorrhagic manifestation along the optic nerve head with or without other vascular complications. Therefore, a sudden rise in the intraocular pressure may be of lesser significance as far as the optic nerve avulsion is concerned, but rather a free anterior movement of the globe within the orbit following trauma to the orbital framework seems to be more important.

In other circumstances where the avulsion injuries are due to an
entry of pointed objects into the orbit, the separation is likely to be due to the creation of distraction forces between the more mobile (globe) and less mobile (optic nerve) tissues [3]. Globe luxation injuries as a risk factor for avulsion supports our view of the probable chain of events [1].

To conclude, optic nerve avulsion is likely to occur due to a number of factors playing knowingly or unknowingly during trauma. However, amongst these anteroposterior tractions along with a torsional tension at the junction of the optic nerve and posterior ocular coat appears to be mainly responsible for the avulsion. This hypothesis has certain limitations, which include, lack of experiments on a real globe, only consideration of post-traumatic scenario and limited known physiological responses. Therefore, future studies can be considered on the creation of an artificial environment to study these factors so as to have a better understanding regarding the ocular traumatic complications and to develop appropriate prophylactic measures.

What was known before

The exact mechanism of optic nerve avulsion is not clear till now. Certain factors like anterior globe luxation, sudden rotation, globe compression with sudden intraocular pressure rise leading to nerve blow off and others have been thought to be responsible for nerve avulsion. However, an acceptable theory for a chain of events or individual factors leading to such dreaded complication is lacking.

What this study adds

Here we elaborate the probable chain of events responsible for optic nerve avulsion using a simple concept.

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.2019.02.031.

References