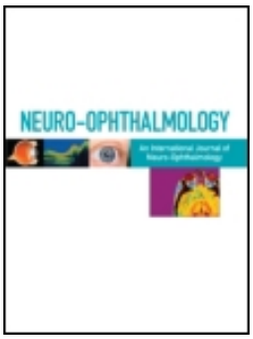

Neuro-Ophthalmology

ISSN: 0165-8107 (Print) 1744-506X (Online) Journal homepage: informahealthcare.com/journals/ioph20

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To cite this article: Celia Chen (2014) Traumatic Optic Neuropathy—The Many Presentations and Treatment Options, Neuro-Ophthalmology, 38:3, 105-106, DOI: 10.3109/01658107.2013.874457

To link to this article: https://doi.org/10.3109/01658107.2013.874457

Published online: 14 May 2014.

Neuro-Ophthalmology, 2014; 38(3): 105–106

ISSN: 0165-8107 print / 1744-506X online
DOI: 10.3109/01658107.2013.874457

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EDITORIAL

Traumatic Optic Neuropathy—The Many Presentations and Treatment Options

In this edition of Neuro-ophthalmology, Vaitheeswaran et al.[1] describe the management of a case of traumatic optic neuropathy (TON) associated with central retinal artery occlusion (CRAO). The authors performed an optic canal decompression via a trans-orbital approach and then administered thrombolytic agents directly to the ophthalmic artery. CRAO associated with TON is rare and only a handful of case reports describe the two conditions occurring together, usually in the context of severe blunt eye trauma.[2,3] Therefore, the use of thrombolytic agents is only relevant to a very few cases of TON.

Many treatments have been suggested for TON without CRAO, but at present there is no consensus on the best treatment option. Part of the controversy is because the precise pathophysiology of TON is not fully known. Broadly, three management options for TON have been described: observation alone, medical treatment, and surgical intervention.

Spontaneous vision recovery in TON has been reported to occur in up to half of the patients.[4] However, the natural history of TON has not been studied prospectively and a randomised controlled trial (RCT) is lacking. An RCT in TON would have several challenges, including the large variety of presentations and the difficulties involved in defining direct versus indirect mechanisms of visual loss. Furthermore, TON often occurs in the context of closed head injury or facial fractures, which may delay the diagnosis of TON and the timing of any treatment. The International Optic Nerve Trauma Study was a comparative nonrandomised interventional study that looked at the visual outcome of TON treated with corticosteroids, decompression surgery, or simply observed without treatment.[5] The authors found no significant difference between any of the treatment groups.

Medical treatment could actually be harmful. High-dose corticosteroids have been used in acute TON, based on the findings of the National Acute Spinal Cord Injury Study (NASCIS) 2, which found a benefit in acute spinal injury patients treated with high-dose steroids.[6] However, a subsequent post hoc analysis of the data performed by Bracken and Holford.[7] suggested that methylprednisolone treatment initiated more than 8 hours after spinal cord injury might actually be detrimental. The Corticosteroid Randomization After Significant Head Injury (CRASH) trial[8] also raised concerns regarding the use of mega-dose corticosteroids in traumatic brain injury. This study was stopped early due to the significantly increased risk of death at 6-month follow-up in patients that received mega-dose steroids when compared with the placebo group. Although the aetiology of the increased risk of death was not determined, the findings of this study must be taken into consideration when managing cases of TON with concurrent traumatic brain injury. Looking at the available evidence, a Cochrane review of corticosteroid use in TON concluded that there was no additional benefit of corticosteroids over conservative management.[4]

Surgical management includes optic canal decompression, but this is generally reserved for cases where a haematoma or a fracture with bony fragment impinging on the optic canal is seen (similar to this case report where there was a direct fracture to the orbital apex with surrounding blood). Many approaches to decompression have been described, including intracranial, transethmoidal, endonasal, and sublabial approaches. Vaitheeswaran et al.[1] now add the trans-orbital approach to this list. A Cochrane review of surgery for TON[9] noted that the wide range of surgical interventions used in TON made it very difficult to compare the studies. The review concluded that there was currently no evidence that surgical decompression of the optic nerve provided any additional benefit over observation alone. The

Received 21 November 2013; accepted 21 November 2013; published online 12 May 2014

authors cautioned that surgery in TON remains controversial and each case needs to be assessed on its own merits.

The case presented by Vaitheeswaran et al.[1] is interesting and challenges us to explore new possible avenues of treatment. At present, there is no evidence-based guideline for treatment of TON, so each case must be evaluated and managed individually, taking into consideration the concurrent injury and the possible mechanisms of injury.

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