Case Reports

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Pediatric Traumatic Optic Neuropathy: A Case of Emergency Multidisciplinary Surgical Decompression

Déborah Laurindo Pereira Santos, Renato Torres Augusto Neto, Mário Francisco Real Gabrielli, Eduardo Hochuli Vieira

Department of Diagnostic and Surgery, Araraquara School of Dentistry, São Paulo State University (UNESP), São Paulo, Brazil.

✉ Déborah Laurindo Pereira Santos, DDS, PhD

e-mail: deborahlpsantos@gmail.com

Available at:
http://www.archpedneurosurg.com.br/

Background: Traumatic optic neuropathy following orbital roof fractures is rare in pediatric populations but represents a surgical emergency due to risk of permanent blindness. Approximately 1.6% of traumatic brain injury cases involve direct optic nerve injury. Orbital roof fractures in children are uncommon and may be associated with frontal lobe injury, dural tears, and proptosis. Early recognition and multidisciplinary intervention are critical for visual preservation.

Case Presentation: A 2-year-old girl sustained high-energy motor vehicle trauma resulting in left orbital roof fracture with optic nerve compression. Clinical examination revealed proptosis, anisocoric pupil without photoreaction, and restricted ocular movement. Computed tomography confirmed displaced orbital roof fracture compressing the optic nerve. Emergency multidisciplinary surgery was performed via medial superior palpebral approach with bone fragment repositioning and optic nerve decompression. Intraoperative cerebrospinal fluid leak resolved spontaneously with conservative management. At 28 months postoperatively, the patient demonstrates complete visual recovery without diplopia, exophthalmos, or dystopia.

Conclusion: Pediatric orbital roof fractures with optic nerve compression require immediate surgical decompression to prevent permanent visual loss. Multidisciplinary collaboration between neurosurgery, oral-maxillofacial surgery, and ophthalmology is essential. Despite potential complications including CSF fistula, timely intervention can achieve excellent functional outcomes.

Keywords: traumatic optic neuropathy; orbital roof fracture; pediatric trauma; optic nerve decompression; emergency surgery

INTRODUCTION

Facial fractures in infants require special attention in diagnosis and treatment. Although they are relatively uncommon compared with adults, they may be associated with cranial trauma and therefore demand careful evaluation [1]. Treatment selection should consider age-specific anatomical, physiological, and psychological characteristics.

Orbital fractures are particularly important because of their close relationship to the visual apparatus, especially when associated with optic nerve compression or orbital compartment syndrome. These injuries are commonly related to high-energy trauma and may occur in the setting of polytrauma [2,3]. Optimal management often requires a multidisciplinary team including neurosurgeons, oral and maxillofacial surgeons, and ophthalmologists.

Orbital compression is an emergency because delayed treatment may result in permanent visual loss. Patients may present with proptosis, restricted ocular motility, pupillary abnormalities, loss of photoreaction, and decreased visual acuity; imaging may demonstrate orbital roof displacement, retrobulbar hemorrhage, or optic nerve compromise [2,3]. This report describes the successful emergency management of a rare pediatric orbital roof fracture causing optic nerve compression.

CASE REPORT

A 2-year-old female patient was referred to the Oral and Maxillofacial Surgery and Traumatology Service of the Araraquara School of Dentistry, UNESP, after a high-energy motor vehicle accident. At initial

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presentation, the patient was in good general condition, with no imminent spinal or intracranial instability, and the complete Advanced Trauma Life Support (ATLS) protocol was followed.

Physical examination revealed facial edema and periorbital ecchymosis (Figure 1A). The left orbit showed proptosis, anisocoria with no pupillary photoreaction to stimulation, and limitation of ocular movement in retroversion. Computed tomography demonstrated a left orbital roof fracture with displacement and compression of the optic nerve (Figure 1B). Because of the risk of permanent blindness, immediate surgical treatment was indicated.

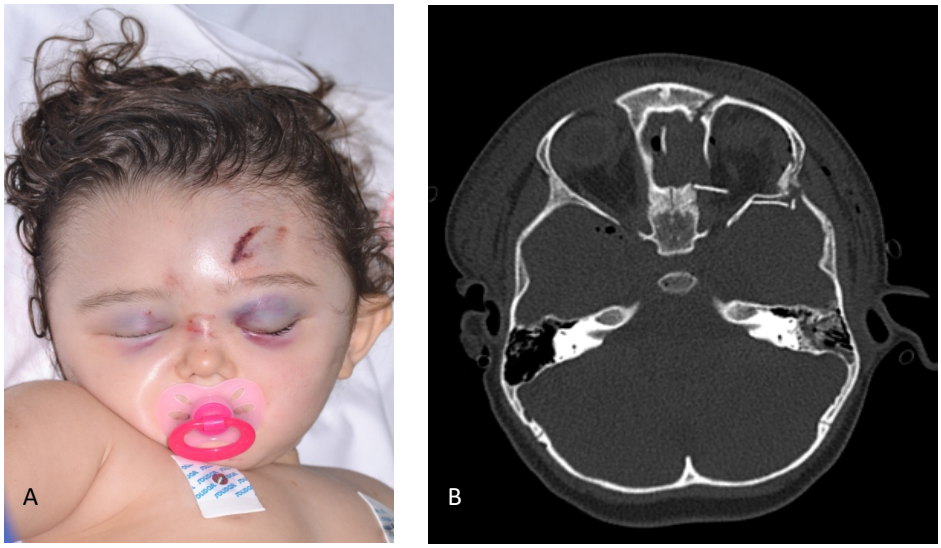

Figure 1. Preoperative findings. (A) Clinical photograph showing bilateral periorbital ecchymosis and the site of frontal impact. (B) Axial computed tomography (CT) scan demonstrating a displaced left orbital roof fracture with compression of the optic nerve.

The patient underwent multidisciplinary surgical treatment by the oral and maxillofacial surgery and neurosurgery teams for orbital and optic nerve decompression through repositioning of the superior orbital wall. A left medial superior palpebral approach was used, and the displaced bone fragment was elevated and repositioned (Figure 2). An intraoperative cerebrospinal fluid (CSF) fistula occurred but ceased spontaneously and was managed conservatively. A Penrose drain was placed to prevent local hematoma formation.

Figure 2. Medial superior palpebral approach and repositioning of the displaced bone fragment

Postoperative imaging confirmed satisfactory repositioning of the orbital roof and decompression of the optic nerve (Figure 3). The postoperative course was favorable, with recovery of ocular motility and preservation of visual acuity. At 28 months of follow-up, the patient showed no visual deficit, diplopia, exophthalmos, or dystopia.

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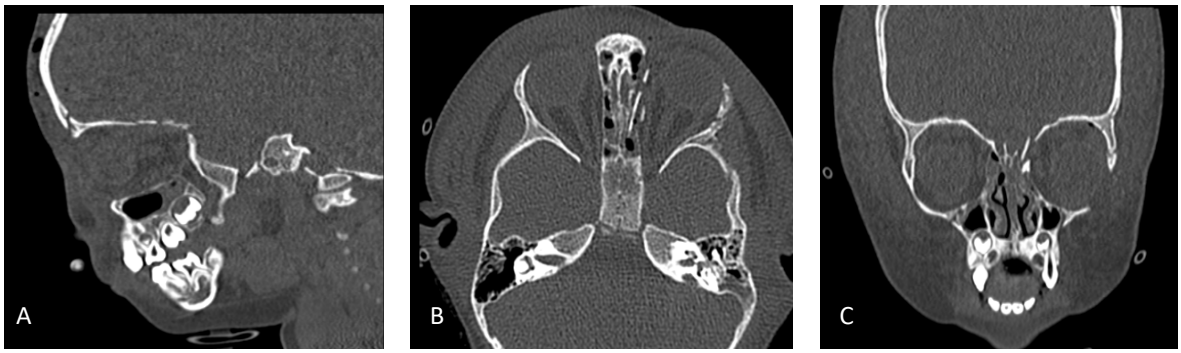

Figure 3. Postoperative CT scans showing sagittal (A), axial (B), and coronal (C) views, respectively

DISCUSSION

Epidemiology and Mechanisms of Traumatic Optic Neuropathy

Traumatic optic neuropathy (TON) in children is uncommon but may occur after high-energy craniofacial trauma and orbital fractures [2,3]. CT-based studies have shown that specific orbital and facial findings, such as intraconal hematoma, optic canal fracture, and related post-traumatic changes, are associated with increased risk of optic nerve injury and visual compromise [3].

Orbital roof fractures are rare in the pediatric population but may be associated with frontal lobe injury, dural tears, proptosis, traumatic optic neuropathy, and other ophthalmic complications [4-6]. Most pediatric orbital roof fractures can be managed conservatively; however, displaced fractures associated with intracranial involvement, ocular displacement, CSF leak, or visual compromise may require surgical intervention [4-6]. The present case illustrates such a scenario, in which a high-energy mechanism produced a displaced orbital roof fracture with direct optic nerve compression requiring emergency decompression.

The optic nerve is approximately 50 mm long and is divided into intraocular, intraorbital, intracanalicular, and intracranial segments, with the intraorbital segment being the longest. The intracanalicular portion is especially vulnerable to trauma, and prognosis tends to be worse when this segment is involved [7]. High-resolution CT is essential for evaluation of bony anatomy and fracture-related compression, whereas MRI may be useful in selected stable patients for assessment of soft tissues and the optic nerve itself [10,13]

Classification: Direct versus Indirect Injury

Traumatic optic nerve injury is commonly classified as direct or indirect. Direct traumatic optic neuropathy is associated with structural injury to the optic nerve caused by orbital trauma, penetrating injury, fracture fragments, contusion, or avulsion. Indirect traumatic optic neuropathy refers to force transmission to the nerve through adjacent soft tissues or bony structures, resulting in edema, hematoma, ischemia, or shearing injury without obvious transection [9-13].

This distinction remains clinically relevant for prognostic assessment and treatment planning [11,12]. Direct injuries caused by penetrating trauma or displaced fracture fragments generally have a poorer prognosis, particularly when there is gross structural disruption of the nerve [12]. Indirect injuries may be reversible in selected patients, although outcomes remain variable and treatment is still controversial [9,11,13].

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Role of Corticosteroids: Current Evidence

High-dose corticosteroids have historically been proposed for indirect optic nerve injury in an attempt to reduce edema within the rigid optic canal [14]. However, contemporary evidence does not support the routine use of high-dose corticosteroids for traumatic optic neuropathy and suggests potential harm, especially with megadose intravenous methylprednisolone [12,14-16].

A recent systematic review found no evidence of benefit for medical therapies and highlighted increased mortality associated with megadose intravenous methylprednisolone in major traumatic brain injury studies [15]. Randomized evidence has not demonstrated clear benefit for intravenous methylprednisolone or other medical therapies in TON [14,15]. The International Optic Nerve Trauma Study likewise found no clear superiority of corticosteroid therapy or optic canal decompression over observation alone in the overall TON population [16]. Nevertheless, corticosteroids continue to be used in some surgical series as adjunctive treatment, making it difficult to isolate the contribution of steroids from that of surgery itself [17-20].

Overall, current evidence does not support routine high-dose corticosteroid therapy as standard treatment for TON [14-16].

Surgical Decompression: Techniques and Outcomes

Surgical treatment is generally considered in cases of direct optic nerve compression by bone fragments or hematoma and in selected patients with progressive visual deterioration [10,13]. In cases of clear orbital or optic nerve compression, early decompression may be justified to reduce ischemia and secondary injury [2,10].

Several surgical approaches have been described, including endoscopic transnasal decompression, transorbital approaches, and transcranial/open techniques, each with specific indications [10,13,17-20]. Endoscopic transnasal decompression is most commonly used for optic canal and orbital apex decompression, particularly when bony compression or apical hematoma is identified [8,18,20]. Published series have reported postoperative visual improvement in selected patients, including pediatric cohorts [8,17-20].

Open or transcranial approaches are particularly relevant in orbital roof fractures with displaced superior wall fragments, dural injury, associated cranial pathology, or the need for direct reconstruction [4-6]. In such cases, direct visualization allows removal or repositioning of displaced fragments and management of associated CSF leak or intracranial involvement. In the present case, a transorbital approach through a medial superior palpebral incision allowed direct reduction of the displaced orbital roof fragment and decompression of the optic nerve, with neurosurgical support because of the intraoperative CSF fistula.

The choice of technique depends on the nature and location of the injury, the patient's general condition, associated craniofacial trauma, and the need for intracranial access [10,13]. Prognosis is generally worse in patients with no light perception before surgery or with severe optic canal injury [7,16,18]. Potential complications include injury to the ophthalmic artery or carotid artery, CSF fistula, meningitis, and intraorbital infection [13]. In the present case, the CSF fistula resolved spontaneously with conservative management and caused no long-term sequelae.

Timing, Prognosis, and Multidisciplinary Care

Early intervention and the presence of baseline residual vision are among the most consistently reported predictors of better visual outcomes [18,19]. Some meta-analytic and cohort data suggest superior recovery when decompression is performed within approximately 7 days of injury, particularly in selected surgically treated patients [8,18,19]. However, these findings should be interpreted with caution because the overall literature on TON remains heterogeneous, and high-level evidence has not established a universal benefit for decompression in all cases [15,16].

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Factors associated with improved postoperative recovery in published series include shorter interval between injury and treatment, residual preoperative vision, less extensive fracture-related damage, and better baseline visual acuity [8,18,19]. Contemporary series also suggest that complication rates may be acceptable when surgery is carefully selected and performed by experienced teams, although risk profiles differ by approach and case complexity [13,17-20].

Multidisciplinary management involving ophthalmology, neurosurgery, otolaryngology or skull base surgery when indicated, radiology, and oral and maxillofacial or craniofacial surgery is essential for individualized decision-making in patients with orbital roof fractures and visual compromise [4-6]. The present case illustrates the value of such coordinated care, with successful collaboration between oral and maxillofacial surgery and neurosurgery resulting in complete visual recovery.

Thus, prognosis is strongly influenced by the mechanism and severity of optic nerve injury [7,12]. Despite the severity and potential for rapid progression, timely diagnosis, careful imaging assessment, and prompt multidisciplinary planning may preserve vision and substantially improve long-term quality of life.

CONCLUSION

Pediatric orbital roof fractures with optic nerve compression are uncommon but potentially devastating injuries that require prompt recognition, imaging-based diagnosis, and multidisciplinary management. In cases of displaced orbital roof fracture with clear mechanical compression of the orbital contents or optic nerve, urgent surgical decompression may be vision-saving. Current evidence does not support routine use of high-dose corticosteroids for traumatic optic neuropathy, and their role remains controversial. As illustrated by this 2-year-old patient, timely and coordinated surgical treatment can result in excellent long-term functional recovery even in complex cases complicated by intraoperative CSF leak.

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DISCLOSURES

Ethical approval

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the local Ethics Committee

Consent to participate

The patients gave consent to use their information and images for research purposes. *Consent for publication*

Conflict of interest

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper

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CONTRIBUTIONS

Déborah Laurindo Pereira Santos: Conceptualization, Methodology, Investigation, Writing - Original Draft
Renato Torres Augusto Neto: Methodology, Investigation, Writing - Review & Editing
Mário Francisco Real Gabrielli: Investigation, Data Curation, Writing - Review & Editing
Eduardo Hochuli Vieira: Investigation, Data Curation, Writing - Review & Editing, Project administration

REFERENCES

1. Dourado E, Cypriano RV, Cavalcanti CDS, Domingues AA. Trauma facial em pacientes pediátricos. Rev Cir Traumatol Buco-Maxilo-Fac. 2004;4(2):101-10. Available from: https://www.revistacirurgiabmf.com/2004/v4n2/pdf/v4n2.4.pdf.
2. Ruiz S, Lim R. Orbital decompression for treatment of acute visual loss after facial trauma. J Craniofac Surg. 2019;30(7):e683-e687. doi:10.1097/SCS.0000000000005892.
3. Bodanapally UK, Van der Byl G, Shanmuganathan K, et al. Traumatic optic neuropathy prediction after blunt facial trauma: derivation of a risk score based on facial CT findings at admission. Radiology. 2014;272(3):824-31. doi:10.1148/radiol.14131873.
4. Afifi A, Jiang S, Smith D, et al. Pediatric orbital fractures: classification, management, and early follow-up. Plast Reconstr Surg. 2008;122(3):886-97. doi:10.1097/PRS.0b013e3181811e48.
5. Cossman JP, Morrison CS, Taylor HO, Salter AB, Klinge PM, Sullivan SR. Traumatic orbital roof fractures: interdisciplinary evaluation and management. Plast Reconstr Surg. 2014;133(3):335e-343e. doi:10.1097/01.PRS.0000438455.37496.9b.
6. Sandhu PS, Bucur S, Good C, Cutler M, Rajak S. The management of orbital roof fractures and defects: a review. Ophthalmic Plast Reconstr Surg. 2022;38(1):8-16. doi:10.1097/IOP.0000000000002025
7. Anderson RL, Panje WR, Gross CE. Optic nerve blindness following blunt forehead trauma. Ophthalmology. 1982;89(5):445-55. doi:10.1016/S0161-6420(82)34769-7.
8. Tu X, Xiong C, Qi H, Ou Y, Rao J, Sun Y, et al. Diagnosis and treatment of transnasal endoscopic optic canal decompression for traumatic optic neuropathy. Front Neurosci. 2023;17:1168962. doi:10.3389/fnins.2023.1168962
9. Carta A, Ferrigno L, Salvo M, Bianchi-Marzoli S, Boschi A, Carta F. Visual prognosis after indirect traumatic optic neuropathy. J Neurol Neurosurg Psychiatry. 2003;74(2):246-8. doi:10.1136/jnnp.74.2.246.
10. Oh HJ, Yeo DG, Hwang SC. Surgical treatment for traumatic optic neuropathy. Korean J Neurotrauma. 2018;14(2):55-60. doi:10.13004/kjnt.2018.14.2.55.
11. Sarkies N. Traumatic optic neuropathy. Eye (Lond). 2004;18(11):1122-5. doi:10.1038/sj.eye.6701571.
12. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy: an evolving understanding. Am J Ophthalmol. 2011;151(6):928-33. doi:10.1016/j.ajo.2011.02.007.
13. Kumaran AM, Sundar G, Chye LT. Traumatic optic neuropathy: a review. Craniomaxillofac Trauma Reconstr. 2015;8(1):31-41. doi:10.1055/s-0034-1393734.
14. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;(6):CD006032. doi:10.1002/14651858.CD006032.pub4.
15. Blanch RJ, Bishop J, Javidi H, Murray PI. Traumatic optic neuropathy management: a systematic review. Eye (Lond). 2024;38(12):2312-2318. doi:10.1038/s41433-024-03129-7. Available from: https://pubmed.ncbi.nlm.nih.gov/38862644/.
16. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. Ophthalmology. 1999;106(7):1268-77. doi:10.1016/S0161-6420(99)00707-1.
17. Li KK, Teknos TN, Lai A, Lauretano AM, Joseph MP. Traumatic optic neuropathy: result in 45 consecutive surgically treated patients. Otolaryngol Head Neck Surg. 1999;120(1):5-11. doi:10.1016/S0194-5998(99)70362-1.
18. Wu Y, Yu J, Zhang J, Chen J. Selection and prognosis of optic canal decompression for traumatic optic neuropathy. World Neurosurg. 2020;138:e564-e578. doi:10.1016/j.wneu.2020.03.007.

19. Wohlrab TM, Maas S, De Carpentier JP. Surgical decompression in traumatic optic neuropathy. Acta Ophthalmol Scand. 2002;80(3):287-93. doi:10.1034/j.1600-0420.2002.800311.x.
20. Jiang RS, Hsu CY, Shen BH. Endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy. Rhinology. 2001;39(2):71-4. Available from: https://pubmed.ncbi.nlm.nih.gov/11486441/.