CASE REPORT

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Orbital Apex Syndrome Without Facial Fractures: A Case Report and Review of Literature

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Abstract

Background: Orbital Apex Syndrome (OAS) is an extremely rare yet significant complication following craniomaxillofacial trauma. The syndrome's clinical features predominantly involve a combination of vision loss and ophthalmoplegia. Despite the severity of its implications, there is scant literature addressing traumatic OAS without associated facial bone fractures.

Case presentation: We present the case of a young adult male who experienced traumatic OAS without facial bone fractures following a head injury from an assault. The patient exhibited ptosis, complete ophthalmoplegia in the left eye, and visual acuity impairment. Diagnostic imaging revealed comminuted fractures of the anterior sphenoid bone and slight narrowing of the optic canal, but no involvement of the intraconal region or other craniofacial injuries. The patient was managed conservatively with close monitoring. While there was some improvement in extraocular muscle movements, the visual impairment persisted.

Conclusion: A comprehensive literature search identified two studies that met the inclusion criteria for traumatic OAS without facial bone fractures or severe traumatic brain injuries. Treatment approaches ranged from observation to high-dose corticosteroids and surgical optic canal decompression. However, visual acuity remained impaired regardless of the treatment type. The variability in outcomes underscores the need for a standardized treatment protocol. Traumatic OAS without facial bone fractures presents a complex clinical challenge with variable progression and mixed treatment outcomes. Early and accurate diagnosis, individualized treatment plans, and a multidisciplinary approach are essential for improving patient prognosis. Further research is necessary to establish a unified treatment protocol for managing this rare condition.

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Introduction

Orbital Apex Syndrome (OAS) is a complex medical condition characterized by a combination of symptoms caused by various pathological processes affecting the region around the optic nerve foramen and the superior orbital fissure (1). The key clinical features of OAS consist of optic neuropathy, which results in vision loss, and ophthalmoplegia, a condition that impairs eye movement (2). This syndrome is considered rare and necessitates thorough evaluation and management due to its intricate nature and potential adverse effects on vision and eye movement. OAS is characterized by a variety of potential causes, including inflammation, infection, neoplasia, vascular issues, and traumatic injuries (3). The orbital apex, a confined space housing complex neurovascular structures, is susceptible to even minor damage. Traumatic OAS, although uncommon, can result from factors such as bone injuries, foreign objects, or bleeding at the

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orbital apex. In traumatic cases, fractured bones and hematoma can directly compress nerves, leading to inflammation and edema, which can disrupt blood circulation in the orbital apex. Symptoms may manifest immediately or several days after the injury, with a significant risk of vision loss (4).

The literature is particularly scant on traumatic OAS in the absence of severe traumatic brain injuries or craniofacial fractures. As a result, there is a lack of consensus on the underlying etiology, pathophysiology, and optimal therapeutic approach for this subgroup of OAS. This paper seeks to bridge this gap by detailing the clinical course and management of a case of isolated traumatic OAS. Furthermore, a review of literature sheds light on the nature of isolated traumatic OAS and evaluates the implications of current treatment strategies. Aiming to enhance the clinical understanding of OAS, this study sets out to: 1) characterize the clinical presentation and progression of traumatic OAS without facial bone fractures; 2) evaluate the treatment options employed and their outcomes; and 3) propose recommendations for a more unified treatment protocol to improve patient prognosis.

Case presentation

A young adult male, otherwise healthy, presented to the emergency department at the University of Florida Health in Jacksonville, Florida, after experiencing a head injury secondary to assault. The patient was reported to have a blow to the head with a metal hanger. The patient was found to have altered mental status, complaining of headache and visual disturbance. On physical examination, the patient had a small scalp laceration anterior to the left preauricular area, and a small left earlobe laceration, and dried blood at both nostrils.

There were no signs or symptoms of facial bone fractures. Right eye examination was within the normal limit. However, the left eye was found to have ptosis with complete ophthalmoplegia (cranial nerves III, IV, VI palsy) (Figure 1).

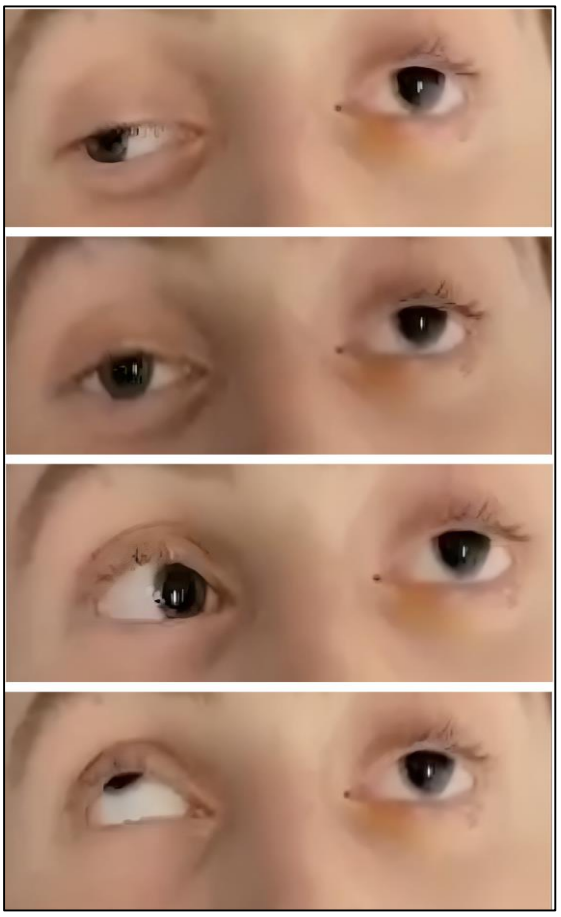

Figure 1: Initial clinical presentation of left eye ophthalmoplegia and ptosis in the patient.

The left pupil was dilated (8mm), round, and not reactive to light. Right eye vision was intact, though the patient denied light perception in the left eye, with assessed visual acuity of 20/400. Additionally, a relative afferent pupillary defect (RAPD) was observed in the left eye. On dilated fundoscopy, vitreous was clear bilaterally, Maculae were flat and optic nerves were sharp, and pink with no elevation. The remainder of the head and neck examination was not remarkable. A head computed tomography (CT) revealed comminuted fractures of the anterior sphenoid bone, along with small air and bone fragments in the superior orbital fissure. There was also a slight narrowing of the optic canal without involvement in the intraconal region. A small

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patchy subarachnoid hemorrhage (SAH) and pneumocephalus were identified. There was no evidence of swelling in the optic nerve or ocular muscles (Figure 2). No other craniofacial injury was noted. The neck CT was normal, and no aneurysm was detected in the brain CT angiography.

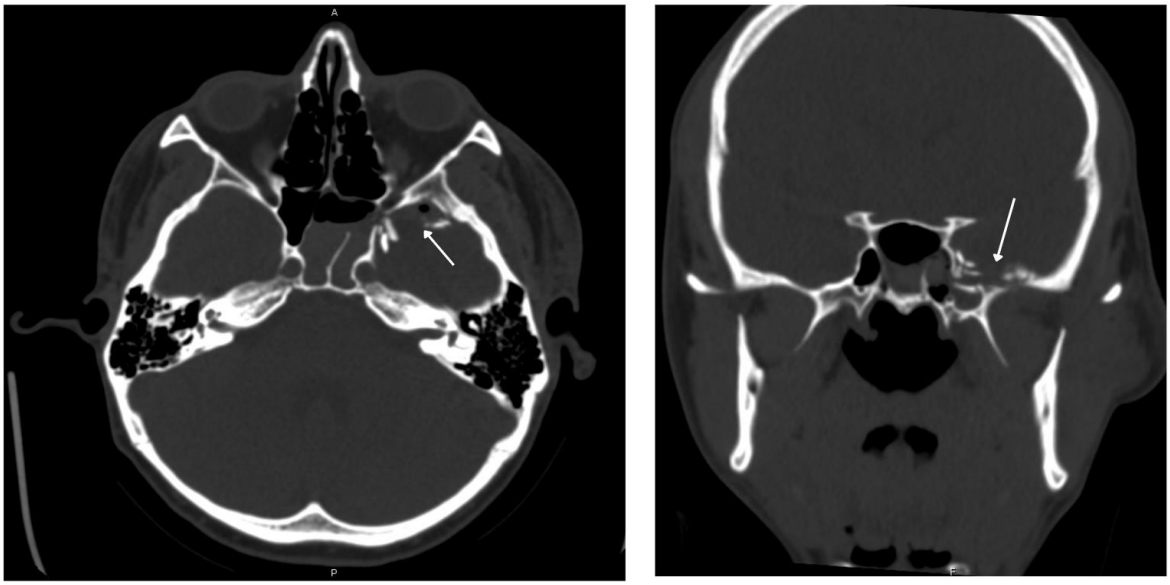

Figure 2: Axial and coronal CT images of the patient's head showing small air and comminuted fractures of the anterior sphenoid bone. The white arrows show bone fragments in the superior orbital fissure.

A diagnosis of traumatic OAS without facial bone fractures was made. No immediate medical or surgical intervention was deemed necessary for the patient, who was closely monitored for 48 hours. The patient regained full mental capacity during the admission. Repeat head CT showed the SAH and pneumocephalus to be stable in size. The second ophthalmologic examination at 48 hours remained unchanged except a mild temporal pallor in the left optic nerve. The patient was eventually discharged. At one-month follow-up, the patient displayed a slight improvement in upward gaze, with no changes in the pupillary condition and visual acuity. Although there was no red desaturation observed, the optic nerve exhibited temporal pallor. Optical coherence tomography (OCT) showed the normal structure of Retinal Nerve Fiber Layer (RNFL). At 6 months, the patient continued to have no light perception out of the left eye, even though extraocular muscle movements showed mild progression, mainly in upward and downward gazes. The patient was also able to blink, with a good corneal sensation. OCT finding was significant for the global thinning of RNFL.

Review of literature

A systematic review of English literature published from 1960 to 2023 was conducted by searching the ISI Web of Knowledge database, PubMed, Scopus, Google Scholar, Embase, and the Cochrane library using the keywords “orbital apex syndrome”, "superior orbital fissure syndrome", “traumatic optic neuropathy”, “TON”, and “ophthalmoplegia”. Our goal was to identify articles presenting patients with traumatic OAS without any associated facial bone fracture and moderate to severe traumatic brain injuries.

The PRISMA Flow Diagram provides a visual representation of our systematic selection process (Figure 3). Initially, a total of 81 records from the databases were identified. Four duplicate records were removed, and an additional set of records was marked as ineligible by automation tools, leaving 32 records included at the screening stage. These

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32 studies were carefully reviewed. Reports of OAS associated with facial fractures (n=29) and those related to severe brain-head injuries (n=20) were excluded. Ultimately, two studies met the inclusion criteria and were included in the comprehensive review. Table 2 outlines the general characteristics of these two articles.

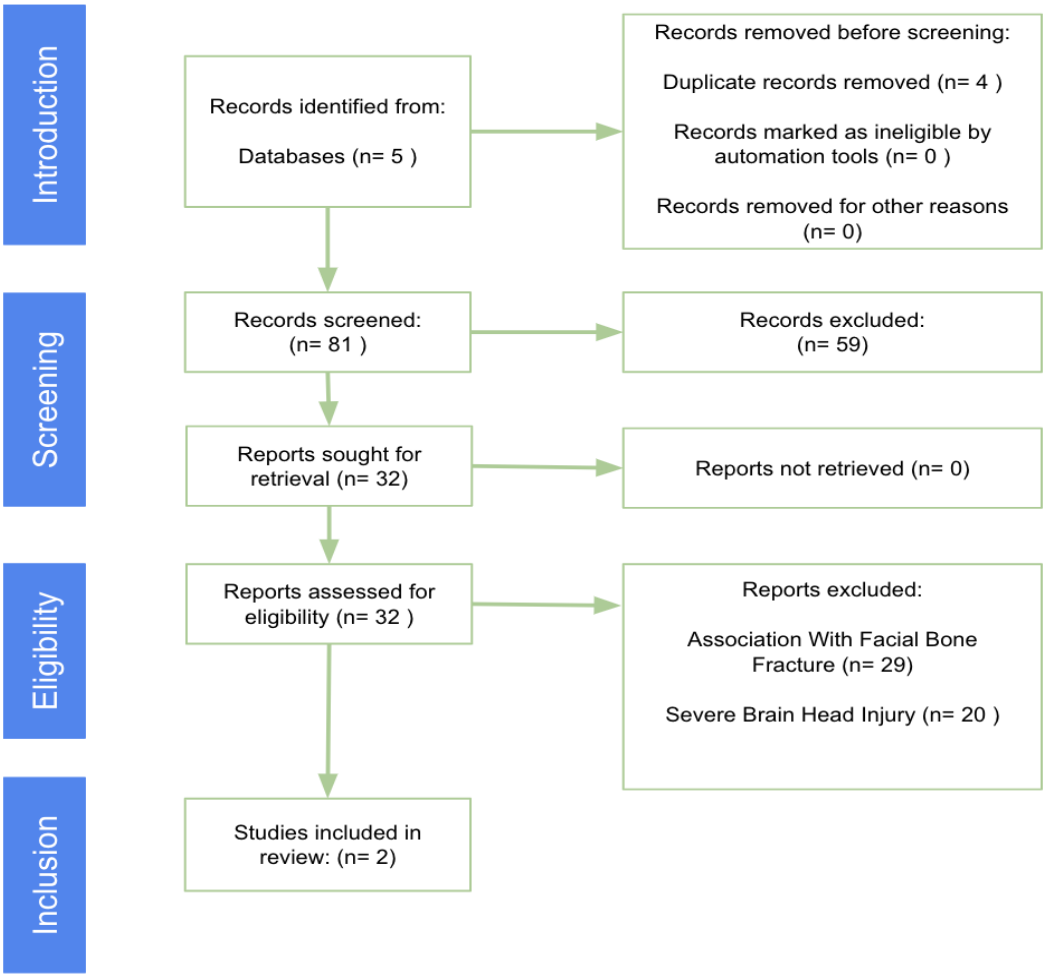

Figure 3: PRISMA Flow Diagram for Study Selection on Isolated Traumatic Orbital Apex Syndrome

In our systematic review, we only found 2 case reports, each Peter and Pearson presented a case of non-penetrating orbital trauma to the left eye secondary to assault with a delayed presentation to the emergency department in 2010. The patient had complete left ptosis associated with a large pupil, blurred vision, and diplopia. Fundoscopy findings were normal. The CT of the brain and orbits was normal and no evidence of bony or penetrating injury was detectable. Oral steroid, tapering over 6 weeks was started. Visual acuity and sensation returned to normal in 3 weeks, though ptosis and ocular motility continued to improve more gradually over 6 months. Also, the pupil remained dilated with an impaired reaction to light and accommodation (5).

In 2015, Gupta and Khan reported a case of OAS from blunt ocular trauma to the left eye without bony involvement after a fall accident. The patient had no light perception, complete ptosis, or restricted ocular motions except the lateral gaze. Dilated fundus examination revealed central retinal artery occlusion with edematous optic nerve. CT imaging showed no bony involvement, with proptosis of the globe and moderate inflammation of orbital contents. The authors did not discuss their treatment plan

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for the patient nor the visual acuity in short-term follow-up (6). reporting one patient sustaining OAS without associated facial fracture (5, 6). Both patients, as well as the patient in our case report, experienced blunt head trauma around the orbital area. A summary of these 2 patients is shown in Table 1.

Table-1 A, B: Clinical and Paraclinical Findings Per Case

A

Case	soft tissue trauma	Visual acuity	Ophthalmologic examination findings	Sensory nerve involvement	MRI	CT
Gupta et al.	Laceration over the left superior orbital rim	NLP OS	edematous optic nerve, cherry red spot	No	Was not ordered	Normal
Peter et al	mild ecchymosis and abrasion of the eyelids	6/12 OS	Normal	No	very mild high signal at the orbital apex	Normal

B

Case	Loss of consciousness	ptosis	proptosis	Pupil diameter and RAPD	Extra ocular movement
Gupta et al.	One minute	Yes	Yes	Not mentioned	Limited except for lateral gaze
Peter et al	No	Yes	No	Large pupil and mild RAPD	complete left ophthalmoplegia, with some relative sparing of abduction only

Table-2: Quality Assessment of Reviewed Case Studies on Isolated Traumatic Orbital Apex Syndrome

Criteria	Gupta & Khan (2015)	Peter & Pearson (2010)
Clear Objective Stated	Yes	Yes
Detailed Patient Demographics	No	Yes
Clinical Presentation Described	Yes	Yes
Diagnostic Methods Described	No	Yes
Treatment Methods Described	No	Yes
Outcome Measures Stated	No	Yes
Follow-up Period Specified	No	Yes
Potential Conflict of Interest Declared	No	Yes
Limitations Discussed	No	Yes

Discussion

The orbital apex is a complex region where neural, vascular, and bony structures converge. This confined space houses the optic, oculomotor, abducens, and nasociliary nerves. The oculomotor, abducens, and nasociliary nerves access the orbit through the superior orbital fissure. On the other hand, the optic

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nerve enters the orbit via the optic canal, alongside the ophthalmic artery (6). Importantly, the optic nerve is enveloped by meningeal coverings, including the pia, arachnoid, and dura mater, which firmly adhere to the surrounding periosteum (7). OAS is an uncommon complication of craniomaxillofacial trauma that endangers vision. This syndrome's clinical features result from the involvement of cranial nerves II, III, IV, and VI, and the ophthalmic division of cranial nerve V (8). Two other syndromes, superior orbital fissure syndrome and cavernous sinus syndrome, present similar symptoms to OAS (9). In addition to the cranial nerves affected in OAS, cavernous sinus syndrome (CSS) involves sympathetic nerves and the maxillary division of the trigeminal nerve. Optic nerve neuropathy distinguishes true OAS from superior orbital fissure syndrome (SOFS) (10).

Craniomaxillofacial trauma stands out as a primary contributor to OAS, although its overall occurrence rate remains quite low. Given the reported incidence of traumatic superior orbital fissure syndrome at only 0.3-0.8% (11) and traumatic optic neuropathy at 0.7–2.5 % (12), it is logical to infer that the prevalence of traumatic OAS (where both conditions coexist) would be notably scarcer than either condition in isolation. The scientific literature contains only a limited number of documented cases of OAS without facial fractures. Multiple mechanisms have been proposed for this type of optic nerve damage, including deformation of the orbital roof due to frontal region trauma, direct shearing injury to axons, disruption of blood supply, and pressure from micro hematomas and edema (13). These mechanisms can deform the bony orbit around the optic foramen areas without causing other orbital bone fractures. Stress concentration areas may occasionally experience fractures, with the thickness of bone in these areas determining whether it undergoes elastic deformation or fractures. Importantly, even in cases of fractured orbital canals, nerve injury due to fracture fragment displacement is rare (14).

Our case report aligns with the findings of Gupta and Khan, who reported a patient with blunt ocular trauma resulting in OAS without bony involvement. In their case, the trauma resulted from a fall accident, leading to indirect injury through high-energy impact transmitting shear forces to the superior orbital fissure and the optic canal (6). Similar to our patient, their patient exhibited no light perception, complete ptosis, and restricted ocular motions. The absence of bony involvement in both cases suggests that severe ocular and neural damage can occur purely from blunt trauma, emphasizing the vulnerability of the orbital apex structures even in the absence of fractures. Additionally, the delayed presentation of symptoms, as observed in our case, aligns with Gupta and Khan's findings where significant inflammation and neural damage were present without immediate fracture evidence (6). Peter and Pearson's case further supports our findings by highlighting the impact of non-penetrating trauma on the orbital apex. Their patient experienced ptosis, a dilated pupil, and diplopia similar to our patient, resulting from an assault, which constitutes a direct injury (5). However, unlike our case and Gupta and Khan's case of indirect injury, Peter and Pearson's case involved direct trauma without subsequent fracture evidence (5, 6).

OAS can result from both direct and indirect injuries. Direct injuries typically involve penetrating trauma or impingement by displaced bony fragments in the orbital apex region. These injuries can cause compression and damage to the cranial nerves, leading to the clinical features of OAS (9, 10). Indirect injuries occur when high-energy impact to the face transmits shear forces to the superior orbital fissure (SOF) or optic canal. Such forces can disrupt the delicate structures in the orbital apex, including the cranial nerves (4, 15). The orbit functions as a closed compartment, and rapid intra-orbital pressure increase following

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trauma can potentially cause permanent vision loss due to ischemic damage to the retina and optic nerve. Additionally, associated retro-bulbar hemorrhage, edema, and orbital emphysema are often present in such cases (7, 16, 17).

Richard L. Anderson et al. used holographic interferometry to examine dried human skulls, aiming to understand how forces applied to facial features affect orbital and optic canal stress without causing fractures. [18] The research revealed that applying force to specific facial prominences, like the supraorbital ridge and malar eminence, resulted in stress concentrations in corresponding areas of the orbital roof and floor, consistent with known patterns of orbital fractures. They suggested that frontal trauma can transmit forces to the optic nerve and its vascular support, potentially causing optic nerve dysfunction through mechanisms like traction, shearing, and shock waves used holographic interferometry to examine dried human skulls, aiming to understand how forces applied to facial features affect orbital and optic canal stress without causing fractures. Furthermore, it was revealed that applying force to specific facial prominences, like the supraorbital ridge and malar eminence, resulted in stress concentrations in corresponding areas of the orbital roof and floor, consistent with known patterns of orbital fractures. Ultimately, the researchers suggested that frontal trauma can transmit forces to the optic nerve and its vascular support, potentially causing optic nerve dysfunction through mechanisms like traction, shearing, and shock waves (18).

Researchers led by Heike Huempfner-Hier used finite element analysis to simulate the impact forces on the forehead. Their findings showed that low-force impacts created stress that spread towards the optic foramen and chiasm, while higher impacts led to fracture patterns in the anterior skull base, including the optic canal. They suggested that even minor stresses in the optic foramen could cause unnoticed microscopic damage and vision loss, while more significant impacts could result in comminuted fractures, potentially affecting the optic canal's integrity (19). As shown in Table 1, clinical signs such as anesthesia, external ophthalmoplegia, ptosis, fixed dilated pupil, lacrimal hyposecretion, diplopia, and decreased visual acuity are highly indicative of OAS (10). These symptoms result from various nerve and muscle dysfunctions in the orbital apex region. Anesthesia, especially in the cornea and upper eyelid, is due to trigeminal nerve damage. External ophthalmoplegia arises from impaired oculomotor, trochlear, and abducens nerves, leading to double vision (diplopia). Ptosis occurs due to oculomotor or sympathetic nerve dysfunction.[20] Decreased visual acuity is associated with optic neuropathy. Imaging techniques such as high-resolution MRI and CT scans aid in diagnosis (20).

A condition often associated with OAS is Traumatic Optic Neuropathy (TON), which could be categorized into primary and secondary injuries (12). Primary TON involves mechanical shearing of optic nerve axons and contusion necrosis due to immediate ischemia from damage to the optic nerve microcirculation. In contrast, secondary TON results from apoptosis of both injured and initially intact adjacent neurons. Notably, many TON patients exhibit a combination of both primary and secondary mechanisms to varying degrees (12). The appearance of the optic disc in fundoscopy is influenced by the location and timing of optic nerve damage. Damage in the posterior part of the optic nerve, like our patient, often results in a normal-looking optic disc, while damage anterior to the entry site of the central retinal vessels may cause swelling and retinal hemorrhage in the optic disc (21). Regardless of the initial appearance, optic nerve atrophy and pallor typically develop about six weeks after the injury (22).

The treatment of optic nerve neuropathy in OAS remains a subject of debate, and there is

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limited specific literature on its treatment. Currently, three main treatment options exist:

1. Observation Alone (23): Some cases of OAS are managed through observation without active intervention.
2. High-Dose Corticosteroids (24): In certain instances, high-dose corticosteroids are prescribed as a treatment option. However, it's worth noting that the use of steroids has a theoretical basis in reducing inflammatory reactions.
3. Surgical optic canal decompression (24): Surgical intervention involving optic canal decompression is another approach to managing OAS.

A retrospective study by Zhenxing Li et al. on 15 patients with a history of Traumatic OSA Syndrome confirmed that CN IV experienced the lightest injury in Traumatic Orbital Apex Syndrome (TOAS) (25). They stated that it is related to unique anatomical features. It is the thinnest cranial nerve and is positioned above CN III in the superior orbital fissure (SOF). CN IV's location allows it to endure less traction during trauma compared to CNs III and VI, which pass through the common tendinous ring. Based on their study, CN II (optic nerve) tends to suffer severe injury in TOAS cases. This was attributed to its close attachment to surrounding bones in the optic canal, making it vulnerable to external impact forces. Additionally, CN II is highly sensitive to shock pressure and hypoxia, making regeneration difficult. The study suggests that while CN II may not achieve satisfactory functional outcomes, other cranial nerves involved in TOAS show significant improvement with appropriate medical or surgical intervention. These improvements can alleviate symptoms of ophthalmoplegia and facial disability.

The International Optic Nerve Trauma Study (23) compared the outcomes of traumatic optic neuropathy (TON) treated with corticosteroids, optic canal decompression surgery, or observation. Surprisingly, this study didn't find a significant difference in visual outcomes between these treatment groups, and no clear advantage was observed for either steroid therapy or optic canal decompression surgery. It's important to highlight that there are distinctions between direct and indirect TON in terms of prognosis. Direct TON typically results in severe and permanent visual impairment with limited chances of recovery (12). On the other hand, indirect TON cases managed conservatively have a better prognosis, with reported visual function retention over the long term, especially if spared three months after the injury. [12] The baseline visual acuity (VA) after trauma is a crucial predictor of the final outcome, with initially poor VA associated with limited or no visual recovery. Various factors, such as loss of consciousness after trauma and absence of visual recovery after 48 hours can influence visual recovery and final VA.[12] The treatment of OAS remains challenging, and the choice between observation, corticosteroids, or surgical intervention should be made on a case-by-case basis, considering individual factors and the current lack of clear treatment superiority.

Conclusion

Traumatic OAS without facial bone fractures reveals a complex clinical presentation, with a variable progression depending on the severity and nerves affected. Treatment options such as high-dose corticosteroids, surgical decompression, and observation showed mixed outcomes. A more unified treatment protocol is suggested, with emphasis on early and accurate diagnosis, individualized treatment plans, and a multidisciplinary approach involving various specialists. This tailored strategy aims to enhance patient prognosis by addressing the unique aspects of each case, highlighting the need for ongoing research and collaboration in the management of OAS.

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Conflicts of Interest

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Patients' Consent

Informed consent obtained for the inclusion of case, photos and CT images in the publication.

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