CASE REPORT

Management of Constant Proptosis after Traumatic Orbital Roof Fracture Caused by Firearm Projectile

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Abstract
Orbital injuries causing severe proptosis are most often caused by high-energy, blunt force trauma, but can also be associated with penetrating traumas, such as firearm projectiles. Head trauma resulting in orbital roof fracture and significant communication between the anterior cranial fossa and posterior orbit are rare events, with herniation of brain tissue and cerebrospinal fluid into the orbit even more uncommon and found in only a small percentage of cases. When present, the herniating tissue and/or fluid can cause significant proptosis and potentially orbital compartment syndrome that increases the risk of damage to the optic nerve and its surrounding structures. Often this condition is managed surgically in an urgent manner to preserve visual acuity and the ocular tissues. In cases of constant proptosis in the setting of orbitocranial injuries, practitioners should balance the risks and benefits of intervention ensuring the best possible clinical outcomes. It is imperative to remember that the proptosis is a clinical finding that is itself not pathognomonic for any particular disease entity.
The following case describes a 20-year old male that presented with multiple gunshot wounds, one of which caused a penetrating injury to the orbit and subsequent brain tissue herniation and cerebrospinal fluid leak with associated constant proptosis and orbital compartment syndrome. This case report examines the treatment options in severe cases of traumatic, constant proptosis, with or without orbital compartment syndrome, and the methods used to preserve vision, anatomy, and prevent severe complications.

Keywords: Proptosis; Orbital compartment syndrome; Orbital roof fracture; Projectile; Canthal cutdown

1. Introduction

Orbital injuries causing severe proptosis are most often caused by high-energy, blunt trauma[1]. Trauma resulting in orbital roof fracture and significant communication between the anterior cranial fossa and posterior orbit are rare events that overwhelmingly result from high-energy, blunt trauma, such as head trauma from a motor vehicle accident. Subsequent herniation of cranial contents including brain tissue and cerebrospinal fluid (CSF) into the orbit causing significant proptosis and/or orbital compartment syndrome (OCS) is even more rare[2]. Cases documented in the literature are often managed surgically with coordination between members of a multi-disciplinary team[3,4]. The typical approach to surgical intervention is resection and/or replacement of herniated brain tissue and reconstruction of the orbital roof, either with autologous grafts or with synthetic mesh[1,3]. This both reduces the mass effect caused by the tissue as well as stems the flow of CSF into the orbit, which alone can cause OCS[5].

Penetrating orbital trauma in the form of a firearm projectile introduces unique management challenges and increased risks for complications when compared to blunt trauma[6]. Complications such as risk of infection, due to connection between external and internal structures, CSF leak, and damage to vasculature are all risks that must be taken into consideration. It may also be difficult to distinguish hemorrhage and tissue edema from tissue herniation and/or CSF on imaging in the setting of severe damage and fragmentation along the bullet tract, often requiring magnetic resonance imaging (MRI) in order to properly identify the contents in the orbit before deciding on a treatment plan[7,8]. Reports indicate rapid resolution of proptosis and recovery of vision in patients that maintained significant visual potential throughout the course of their care[1-3]. However, penetrating injury carries an overall poorer prognosis than blunt trauma injury[9].

In the following case, a 20-year old male was brought to the trauma center with multiple gunshot wounds, some of which struck his head causing bilateral orbital roof fractures with cerebral tissue herniation similar to previously reported cases. In the case of this patient, orbital roof reconstruction on the left was not performed resulting in increasing proptosis and optic nerve stretch. Additionally, the large bony defect resulted in a latent accumulation of CSF, which combined with the mass effect of the tissue, resulted in OCS and ultimately optic neuropathy. A combination of surgical intervention and medical management was performed based upon visual potential, ocular function, and systemic condition. The rationale and methods of ophthalmic management described are applicable to cases with similar presentation.

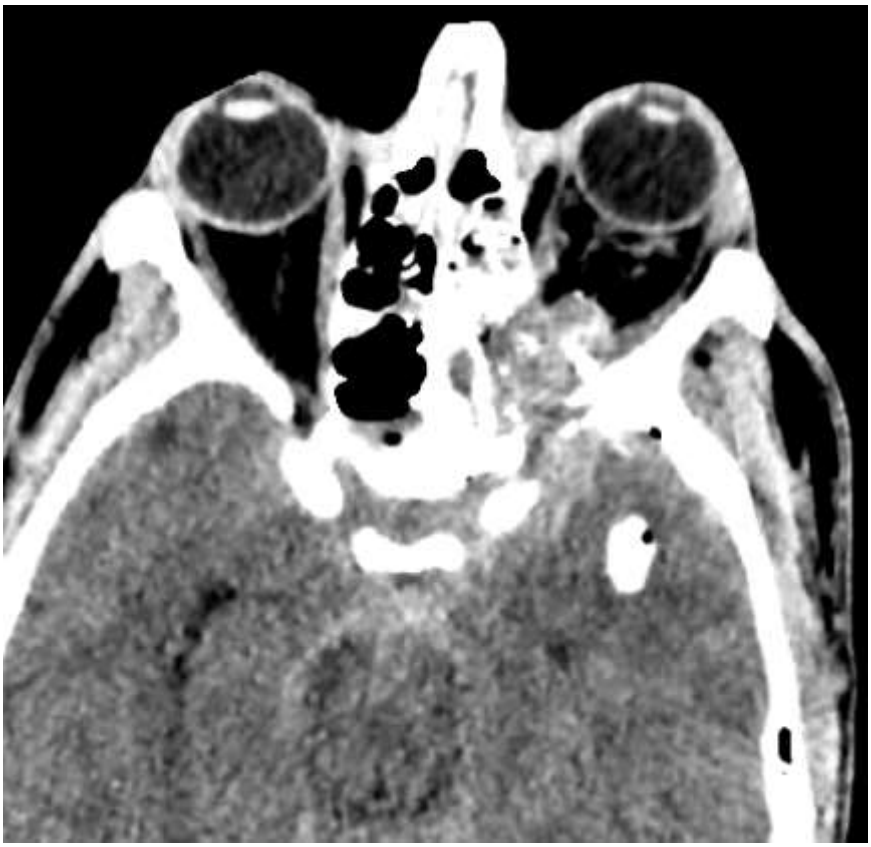
2. Case Report

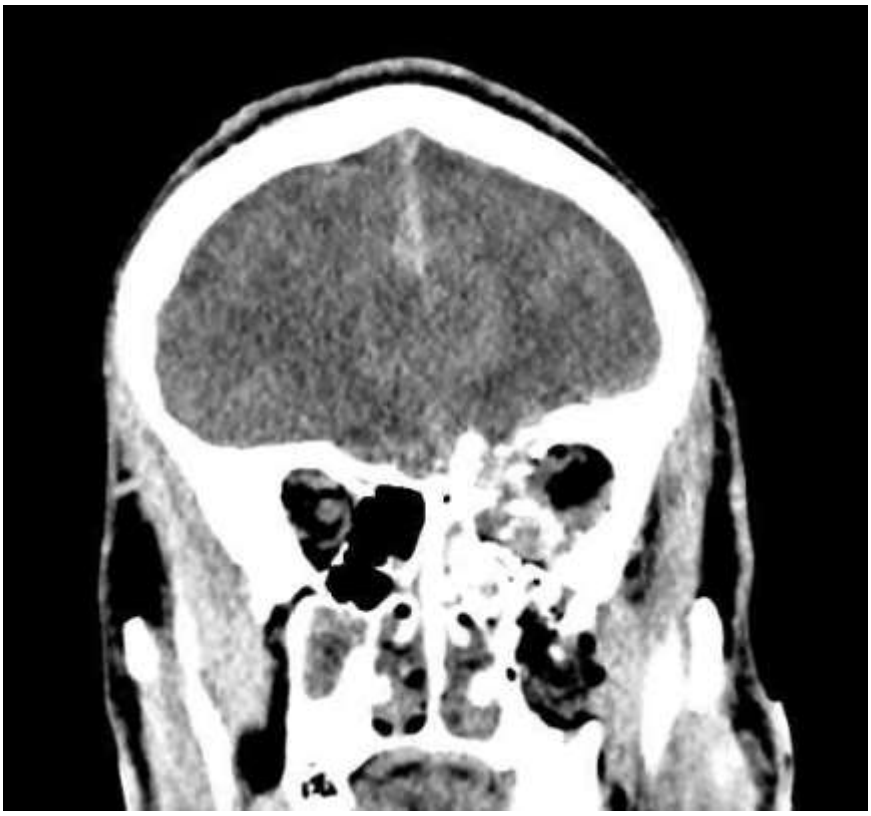
A 20-year old black male was brought to the Level 1 trauma center with multiple gunshot wounds. Head and neck computed tomography (CT) scans were obtained which showed multiple bony defects of the head including displaced fractures of middle cranial fossa, sphenoid of the left greater wing, and bilateral frontal sinuses with intracranial and orbital extension of the bony fragments and brain tissue on the left. Imaging also revealed a small subarachnoid hemorrhage, subdural hemorrhage along the anterior interhemispheric falx, small volume pneumocephalus, and mild hydrocephalus. The patient had no significant past medical or ophthalmic history. Ophthalmology was consulted for evaluation of left sided proptosis. During this initial evaluation by ophthalmology, the patient was intubated and sedated and therefore could not participate in the examination. The patient had significant edema and ecchymosis to the upper and lower eyelids on the left. There was 2-3mm of

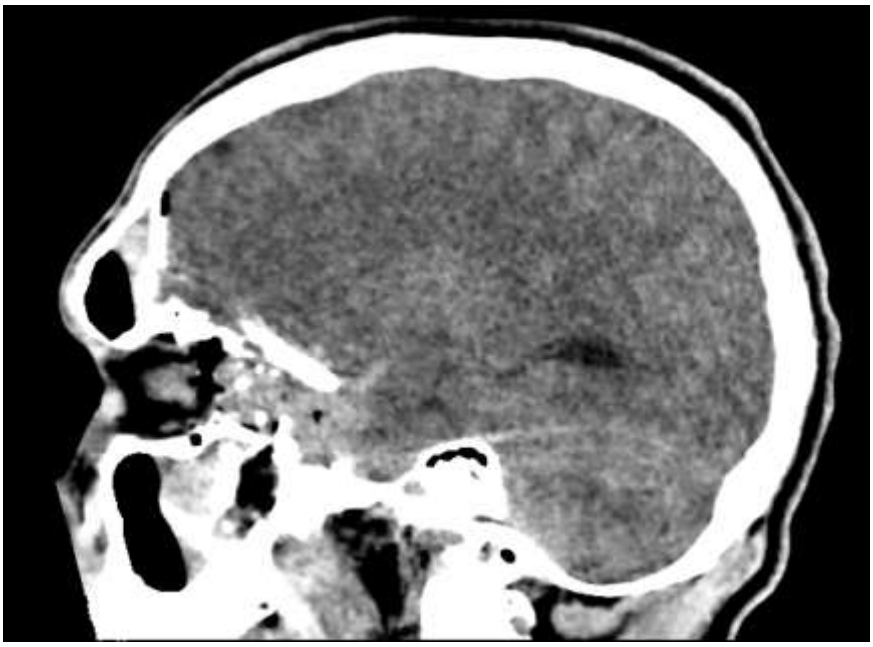
proptosis of the left globe. The patient's pupils were 2.5mm on the right (OD) and 6mm on the left (OS) which sluggishly constricted to 2.0mm OD and did not change OS under direct illumination. An afferent pupillary defect (APD) was noted OS. The intraocular pressures (IOP) were 21mmHg OD and 58mm OS via Perkins tonometer. Anterior segment evaluation was significant for 1+ conjunctival edema OS. Dilated fundus examination revealed pink and flat optic nerves with sharp margins and a cup to disc ratio (CD) of 0.3 bilaterally (OU). The macula, vessels, periphery, and vitreous were without abnormal findings OU.

A CT scan of the orbits revealed bilateral lamina papyracea fractures, bilateral orbital roof fractures OS>OD, orbital floor and posterior medial wall fractures extending to the orbital apex OS, hemorrhage/fat stranding with intra and extraconal free air OS, and mildly enlarged rectus muscles OS (Figure 1). Based on the clinical and radiographic findings there was a high suspicion for OCS and an emergent canthal cutdown was performed bedside in a manner previously described[10]. Following the procedure, the IOP was checked and found to be unchanged OD and significantly lowered OS to 22mmHg. The patient was started on Timolol 0.5% drops every 12 hours to further decrease IOP.

Figure 1A-C. CT of the head and orbits at initial presentation. All images show a large defect of the orbital roof on the left with bony fragments and brain tissue present in the orbit. Bullet fragments can be seen in the orbital apex. A. Axial view. B. Coronal view. C. Sagittal view.

A 

B 

C 

The following day, the patient remained intubated and sedated following a bifrontal craniotomy with exoneration of the frontal sinuses. Due to the extent of the injury, orbital roof repair was not performed on either side. On ophthalmic exam, the external findings were unchanged other than increased proptosis OS to 5-6mm that was now pulsatile. Pupillary exam was unchanged and IOP was 17mmHg OD and 18mmHg OS. Anterior segment

evaluation was similarly unchanged other than an increase in conjunctival edema to 2+ OS. At this point the clinical indicator available for OCS were normalized, so management was directed towards protecting against ocular exposure with frequent application of topical ophthalmic ointment (q4 hr) and constant ocular surface and adnexal covering with a Tegaderm (3M, St. Paul, MN) dressing (Figure 2).

Figure 2. To prevent corneal decompensation from exposure and conjunctival desiccation, ophthalmic ointment is placed on the eye every 4-6 hours and the eye and adnexa are covered with a Tegaderm dressing. The 4x4 taped adjacent to the lateral canthus is to collect seepage from the canthal cutdown site.

Over the following six days, the patient was noted to have a significant increase in the amount of proptosis OS (Figure 3A&B). Repeat CT and CTA imaging of the head and orbits did not reveal any evidence of new hemorrhage, encephalocele, or vascular malformation. However, herniation of frontal lobe tissue through the orbital roof defect along with findings consistent with CSF accumulation in the orbit were noted on the left with redemonstration of previous hemorrhage/ fat stranding (Figure 4 A-C).

Figure 3 A&B. A. Over the course of one week, the patient developed increasing proptosis as shown in this image. The Tegaderm dressing remains in place and frequent ocular lubrication with ointment is continued. B. lateral view with Tegaderm dressing removed. A central area of corneal epithelial irregularity can be seen resulting from insufficient surface lubrication and contact with the dressing.

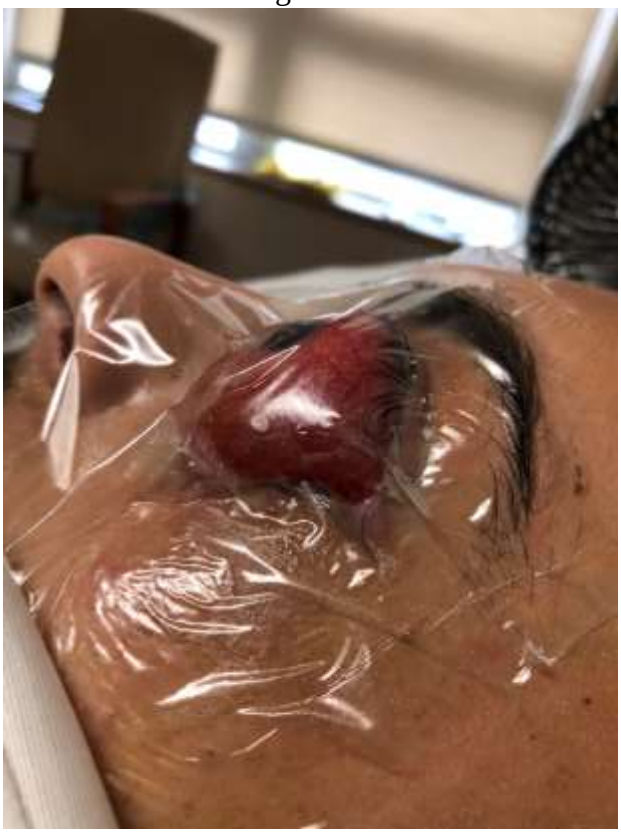
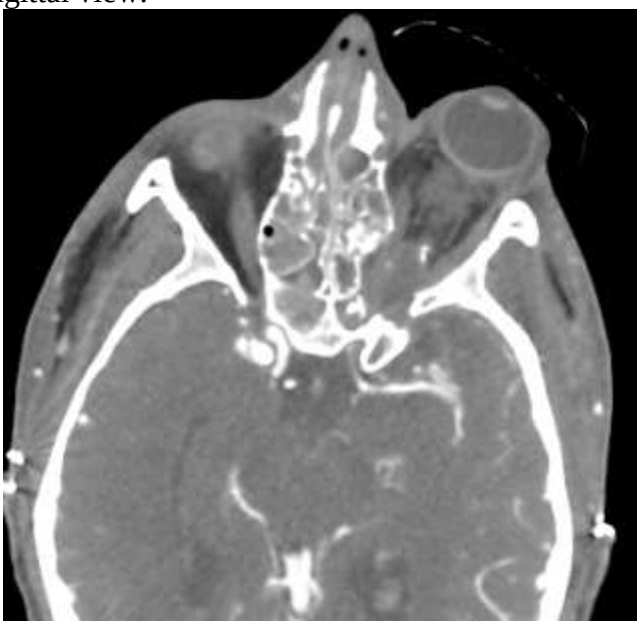
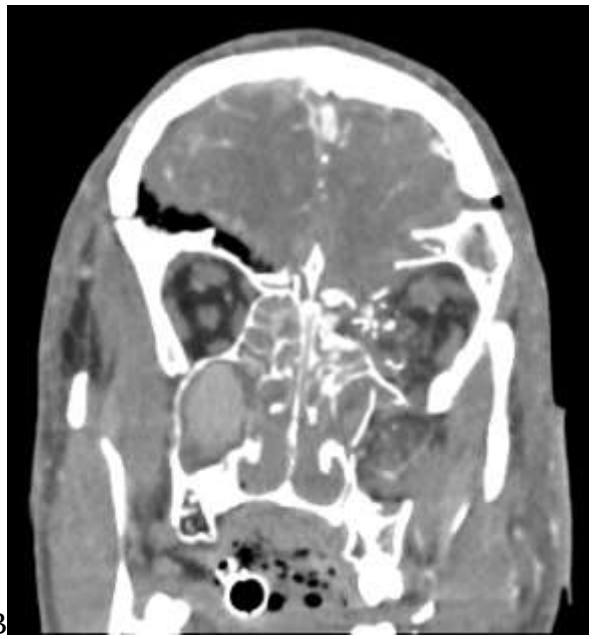
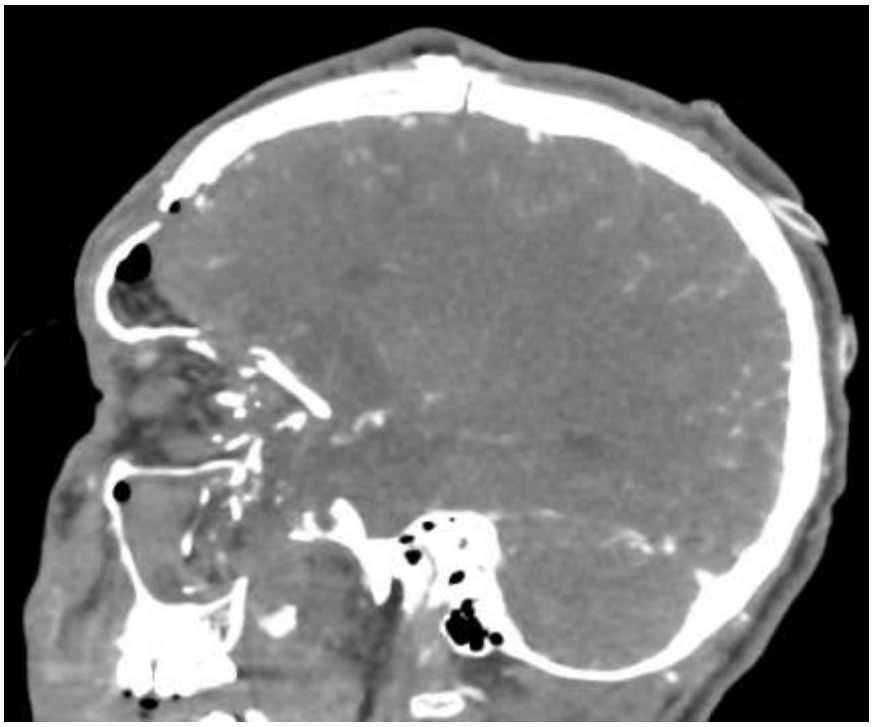
A  B 

Figure 4. Repeat CT obtained 6 days after initial imaging is shown in Figure 1A-C. The images did not reveal any evidence of new hemorrhage, encephalocele, or vascular malformation. Increased herniation of frontal lobe tissue through the orbital roof defect along with findings consistent with CSF accumulation in the orbit were noted on the left with redemonstration of previous hemorrhage/fat stranding and bone/bullet fragments. A. Axial view. B. Coronal view. C. Sagittal view.

A 

B 

C 

On examination, the patient had significant reduction of edema and ecchymosis to the upper and lower eyelids OS. The proptosis OS was now 10-12mm and remained pulsatile. The patient's pupils were 3mm constricting to 2mm with direct light OD and 6mm and non-reactive OS with an APD. The IOP remained stable at 16mmHg OD and 18mm OS. The patient was now able to participate in the exam which revealed a best self-corrected near visual acuity (NVAsc) of 20/40 OD and hand motion (HM) OS. Confrontation visual fields (CFVF) were full OD and unable to be performed OS due to visual acuity (VA). Ocular motility was full OD and globally restricted 70-100% OS. Anterior segment evaluation was significant for 4+ conjunctival edema with superficial punctate keratopathy (SPK) OS. Dilated fundus examination was unchanged from the initial evaluation. Due to the patient's visual potential, stabilized IOP, patent canthal cutdown, and risk of infection with further reconstructive intervention, medical management for ocular surface protection was continued. Over the course of 10 days the orbital congestion decreased to the point where a temporary tarsorrhaphy could be placed for definitive treatment of the exposure keratopathy OS (Figure 5).

Figure 5. Patient 18 days after initial presentation. Proptosis and conjunctival edema have improved to the point where a temporary tarsorrhaphy can be placed to prevent corneal exposure.

The patient presented for follow up in the ophthalmology clinic three months following his initial injury. All previous abnormal external findings had resolved other than trace periorbital ecchymosis OS, including the globe proptosis (Figure 6 A&B). Intraocular pressures were normal at 15mmHg OU. Confrontation visual fields remained full OD and unable to be performed due to VA OS. Motility was full OD and 10-100% globally restricted OS, with a severe hypertropia of over 50 prism diopters on primary gaze. Pupillary evaluation showed a 3mm pupil OD that constricted to 2mm with direct light and a 5mm, non-reactive pupil OS. There was a 4+ APD present OS. Best corrected visual acuity was 20/20 OD and no light perception (NLP) OS. Anterior segment evaluation revealed normal findings OU. Dilated fundus exam was normal OD with a CD ratio of 0.3 and a normal appearing nerve. The CD ratio was also 0.3 OS, however the nerve showed 2+ pallor.

Figure 6 A&B. Patient three months following initial presentation. Temporary tarsorrhaphy was removed approximately one month prior. Proptosis has fully resolved and area of canthal cutdown has healed iatrogenically. Patient has significant strabismus and motility deficits on the left.

A

B

3. Discussion

Medical and surgical decision-making in the setting of severe orbitocranial injuries have to be balanced with systemic condition and visual potential. In the acute treatment phase time is critical and intervention, if required, must be urgent to prevent visual loss. Once stabilized, coordination and cooperation between multiple medical specialties provides the best clinical outcomes. Major factors to consider in these clinical scenarios include, but are not limited to, visual acuity potential, preservation of orbital and ocular tissues, prevention of serious and/or life-threatening

complications, and risks associated with surgical intervention versus medical management.

In evaluating any patient with traumatic proptosis, the primary goal is to determine if there is compromise of the optic nerve. The clinical presence of proptosis in and of itself does not equate to active optic nerve harm. Traumatic optic neuropathy (TON) can occur at the time of injury due to shear/strain forces, transection, and/or bony compression or subsequently as a result of nerve stretch or mechanical compression (i.e. hemorrhage, edema, CSF, emphysema, or displaced tissue causing OCS)[11]. In cases where significant proptosis is not present, OCS may still be present, which in turn can quickly lead to optic neuropathy. The prognosis of TON depends on the pathophysiology and time between occurrence and treatment.

Evaluating optic nerve function is done by assessing visual acuity, pupillary function, visual fields, color vision (desaturation), and optic disc appearance[12]. Other clinical variables that can indicate potential nerve compression from OCS are IOP and motility. Assessing these variables can be difficult in the trauma patient who may not be able to participate in the exam, thus limiting the clinical information available. In the absence of sufficient clinical information, it is possible to directly measure the orbital compartment pressure to rule out OCS[13]. However, this quantitative assessment does not rule out TON, which requires additional clinical variables for diagnosis. It would however provide valuable data to determine if compressive optic neuropathy (CON) was occurring and appropriate treatment could then be initiated.

When present, the cause of proptosis should be identified. This is accomplished via various radiographic modalities of the brain and orbits, such as CT, magnetic resonance imaging (MRI), computed tomography angiography (CTA), and magnetic resonance angiography (MRA). The imaging findings can be placed into two categories – dynamic or static. Dynamic causes would include an active (source specific) orbital hemorrhage, CSF leak, or arterial or venous malformation[6]. Static causes include displaced fractures, orbital hemorrhage, fluid collection, tissue edema, emphysema, encephalocele, and herniation of exogenous tissue into the orbit.

In each case, the clinical and radiographic findings direct the management options taking into consideration the patient's systemic status and visual potential. In cases were clinical findings indicate optic nerve compromise from a compressive orbital process, initial management centers on reducing orbital pressure and intraocular pressure. This is typically accomplished by a canthotomy/cantholysis and/or canthal cutdown in combination with topical IOP lowering drops[10]. The canthal cutdown procedure has been shown to provide a greater and more sustained reduction in orbital pressure over isolated canthotomy/cantholysis, especially in cases of dynamic etiologies[10]. In cases where this does not adequately reduce pressure, bony orbital decompression is performed. The cause of the increased orbital pressure is then addressed. In cases of static etiologies, medical management with monitoring of nerve function is the usual course. In case of dynamic etiologies, surgical intervention to correct the cause is usually

required for adequate, maintained pressure reduction.

In the reported case the patient was intubated and sedated so VA, CFVF, motility, and color desaturation were indeterminate at initial presentation. The IOP was elevated and an APD was present, indicating likely increased orbital pressure with optic nerve compromise. The initial CT showed a small orbital hemorrhage/fat stranding and herniation of brain tissue into the orbit, findings which could further contribute to CON. An urgent canthal cutdown was performed which reduced the IOP from 58mmHg to 22mmHg. Following neurosurgical intervention the IOP remained stable, however proptosis increased and became pulsatile. Repeat imaging was obtained to rule out any type of fistula, new or increased hemorrhage, cellulitis, or encephalocele. The scans showed no change in size of the previous hemorrhage/fat stranding with brain tissue still present in the orbit and a new accumulation of CSF. At this point the patient was able to participate more with an ophthalmic exam and his vision was found be in the non-functional range of HM. As optic nerve function cannot be restored, only prevented from worsening, and the patient did not have functional vision, the primary goal became preservation of the ocular and adnexal tissues. In this case, the brain tissue and CSF in the orbit prevented the globe from being retropulsed enough for a tarsorrhaphy to be placed (Figure 3B). Aggressive lubrication and covering of the cornea and conjunctiva was done until a tarsorrhaphy could be placed to prevent corneal ulceration and conjunctival desiccation (Figures 2 & 3A).

¬In this case, the risk of further surgical intervention outweighed the benefit. First and foremost, being that the mechanism of injury was a gunshot wound, the patient already fell into the category of increased risk factors with a diminished prognosis as previously detailed. Additionally, the patient's visual potential was exceedingly low with a measured acuity of HM, though it should be noted that obtaining a perfectly accurate assessment from someone in his systemic and cognitive condition is difficult, if not impossible. Finally, the fracture of his orbital roof was markedly comminuted, leaving very little native bone structure available to achieve a successful reconstruction. These factors differentiate this patient from others that underwent successful intervention in that those patients were fortunate enough to have their vision preserved, indicating that their visual potential was intact on presentation. Furthermore, the fractures of their orbital roof(s) was either displaced or comminuted, but not so much so that the roof was obliterated[1,3]. In light of all of this, surgical intervention for this patient would only be undertaken in order to preserve the anatomy, an outcome that could also be achieved through more conservative measures. Finally, and possibly the strongest contraindication to surgical intervention, was the patient's significantly increased risk of severe and potentially life-threatening infection. Overall, these factors suggest that further surgical intervention would have carried exceedingly high risk with very little benefit to our patient. Managing our patient conservatively allowed time for significant reduction in edema, further stabilization, and overall recovery of health.

4. Conclusion

In all cases of constant proptosis in the setting of orbitocranial injuries, practitioners should remain cognizant of the ultimate goals of care, and use those goals to guide clinical decision making, ensuring that the minimum necessary risk is taken in order to provide the patient with the best possible clinical outcomes. It is imperative to remember that the proptosis is a clinical finding that is not itself pathognomonic for any particular disease entity, specifically OCS or CON. The management of complex orbitocranial injuries vary from patient to patient and a "one size fits all" approach cannot be taken. While preservation of vision and ocular function is always a primary goal, it cannot supersede the systemic issues the patient may be experience. Therefore, a multi-disciplinary team approach to management provides the best possible clinical outcomes.

5. DECLARATIONS

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6. References

1. Mokal NJ, Desai MF. Titanium Mesh Reconstruction of Orbital Roof Fracture with Traumatic Encephalocele: A Case Report and Review of Literature. Craniomaxillofacial Trauma & Reconstruction. 2012;5(1):11-18.
2. Jaiswal M, Sundar IV, Gandhi A, Purohit D, Mittal RS. Acute traumatic orbital encephalocele: A case report with review of literature. Journal of Neurosciences in Rural Practice. 2013;4(4):467-470.
3. Ha AY, Mangham W, Frommer SA, et al. Interdisciplinary Management of Minimally Displaced Orbital Roof Fractures: Delayed Pulsatile Exophthalmos and Orbital Encephalocele. Craniomaxillofacial Trauma & Reconstruction. 2017;10(1):11-15.
4. Abou-Al-Shaar H, Krisht KM, Cohen MA, Abunimer AM, Neil JA, Karsy M, Alzhrani G, Couldwell WT. Cranio-Orbital and Orbitocranial Approaches to Orbital and Intracranial Disease: Eye-Opening Approaches for Neurosurgeons. Front Surg. 2020;7:1.
5. Habets JGV, Haeren RHL, Lie SAN, Bauer NJC, Dings JTA. Acute Monocular Blindness Due to Orbital Compartment Syndrome Following Pterional Craniotomy. World Neurosurg 2018;114:72-75.
6. Czyz CN, Piehota PG, Strand AT, Stein M. Post-traumatic ophthalmic artery pseudoaneurysm from orbital projectile. Can J Ophthalmol. 2017;52(4):e130-e132.
7. Antonelli V, Cremonini AM, Campobassi A, Pascarella R, Zofrea G, Servadei F. Traumatic encephalocele related to orbital roof fractures: report of six cases and literature review. Surg Neurol. 2002;57(2):117-25.
8. Wei LA, Kennedy TA, Paul S, Wells TS, Griepentrog GJ, Lucarelli MJ. Traumatic orbital encephalocele: Presentation and imaging. Orbit. 2016;35(2):72-7.
9. Kazim SF, Shamim MS, Tahir MZ, Enam SA, Waheed S. Management of penetrating brain injury. Journal of Emergencies, Trauma and Shock. 2011;4(3):395-402.
10. Strand AT, Czyz CN, Gibson A. Canthal cutdown for emergent treatment of orbital

compartment syndrome. Orbit. 2017;36(5):285-292.
11. Singman EL, Daphalapurkar N, White H, Nguyen TD, Panghat L, Chang J, McCulley T. Indirect traumatic optic neuropathy. Mil Med Res. 2016;11;3:2.
12. Smith AM, Czyz CN. Neuroanatomy, Cranial Nerve 2 (Optic). 2020 Nov 19. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–.
13. Czyz CN, Strand AT. Minimally invasive in vivo orbital pressure measurement. Clin Exp Ophthalmol. 2016;44(8):724-725.

