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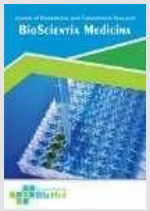

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Beyond the Floor: Traumatic Medial Rectus Entrapment in a Medial Orbital Wall Fracture Presenting with Diplopia and Retinal Hemorrhage

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A B S T R A C T

Background: Medial orbital wall fractures with extraocular muscle entrapment represent a significant but less common variant of orbital trauma compared to floor fractures. These injuries pose a diagnostic and management challenge, with the potential for severe, long-term functional deficits and life-threatening systemic complications if not addressed promptly. This report details a case of medial rectus muscle entrapment complicated by a concurrent posterior segment injury. **Case presentation**: A 21-year-old male presented to the emergency department following a motorcycle accident, sustaining blunt trauma to his left eye. He reported an acute onset of blurred vision and binocular diplopia. Ophthalmic examination revealed a visual acuity of 20/80 in the left eye. There was a manifest esotropia and a profound abduction deficit, with marked restriction of movement on attempted lateral, superolateral, and inferolateral gaze. The forced duction test was positive, confirming mechanical restriction. Funduscopy identified significant retinal hemorrhages. A maxillofacial computed tomography scan confirmed a comminuted fracture of the left medial orbital wall (lamina papyracea) with clear evidence of medial rectus muscle entrapment within the fracture fragments. The patient underwent urgent surgical intervention involving exploration of the medial orbit, careful release of the incarcerated medial rectus muscle, and anatomical reconstruction of the wall with a titanium mini-plate. Intraoperative forced duction testing confirmed complete resolution of the mechanical restriction. Postoperatively, the patient showed free passive ocular motility, although active movement was recovering, and the retinal injuries required continued observation. **Conclusion**: This case underscores the critical importance of maintaining a high index of suspicion for medial rectus entrapment in patients presenting with post-traumatic diplopia and an abduction deficit. A thorough clinical examination, particularly the forced duction test, is paramount and often more definitive than imaging alone. Urgent surgical decompression and reconstruction are imperative to prevent permanent strabismus from muscle ischemia and to mitigate the risk of the oculocardiac reflex. Furthermore, the presence of concomitant intraocular injuries, such as traumatic retinopathy, must be diligently assessed as they significantly impact the final visual prognosis.

1. Introduction

Facial trauma is a prevalent and significant cause of morbidity worldwide, frequently resulting from interpersonal violence, motor vehicle accidents, falls, and sports-related injuries.[1] Among the spectrum of facial injuries, fractures of the orbital skeleton are particularly common and carry the potential for devastating functional and aesthetic consequences. The orbit, a complex pyramidal bony cavity that houses and protects the globe and its adnexa, is composed of seven different bones.[2] Its unique anatomical structure, featuring both robust rims and paper-thin walls, makes it susceptible to characteristic fracture patterns following blunt

periorbital trauma. The mechanism of injury typically involves a direct impact from an object larger than the orbital aperture, causing a sudden increase in intraorbital pressure. This hydraulic force is transmitted to the weakest points of the orbit, leading to a "blowout" fracture of the orbital walls, most commonly the floor (maxillary sinus roof) followed by the medial wall (ethmoidal lamina papyracea).[3] These two locations are particularly vulnerable; the floor is a thin bone overlying the maxillary sinus, while the medial wall is the thinnest bone in the orbit, providing little resistance to pressure changes.

Orbital fractures present with a constellation of clinical signs and symptoms, including periorbital ecchymosis and edema, enophthalmos (posterior displacement of the globe), and hypoesthesia in the distribution of the infraorbital nerve.[4] However, one of the most functionally debilitating complications is diplopia, or double vision. Post-traumatic diplopia can arise from several etiologies, including direct cranial nerve palsy, restrictive strabismus due to soft-tissue edema or hemorrhage, or, most critically, the mechanical entrapment of one or more extraocular muscles within a fracture site.[5] This entrapment, also referred to as incarceration, represents a true ophthalmological emergency. If not relieved in a timely manner, the compromised blood supply to the entrapped muscle can lead to ischemia, subsequent fibrosis, and permanent motility restriction, resulting in intractable, lifelong diplopia. Furthermore, the entrapment of an extraocular muscle can precipitate the oculocardiac reflex (OCR), a potent trigemino-vagal reflex arc. Stimulation of the ophthalmic branch of the trigeminal nerve (CN V1) through traction on the extraocular muscles generates an afferent signal to the trigeminal sensory nucleus, which then connects to the visceral motor nucleus of the vagus nerve (CN X).[6] The resulting efferent vagal stimulation can cause profound systemic effects, including sinus bradycardia, arrhythmia, nausea, syncope, and, in rare but reported cases, asystole and death.[7] The presence of the OCR transforms an urgent clinical situation into a life-threatening emergency necessitating immediate surgical intervention.

While fractures of the orbital floor with entrapment of the inferior rectus and inferior oblique muscles are well-described and represent the classic presentation, fractures of the medial orbital wall with entrapment of the medial rectus muscle are also a significant clinical entity.[8] Entrapment of the medial rectus muscle characteristically produces a profound abduction deficit, leading to esotropia (inward deviation of the eye) in primary gaze and severe diplopia on side gaze. The diagnosis relies heavily on a meticulous clinical examination, with the forced duction test (FDT) serving as the definitive maneuver to differentiate a mechanical restriction from a neurogenic palsy.[9] In a mechanical restriction, the examiner will feel a firm resistance when attempting to passively move the globe away from the site of entrapment. The diagnostic workup is completed with high-resolution computed tomography (CT) of the orbits, which remains the gold standard for imaging. Thin-slice (1-1.5 mm) imaging in axial, coronal, and sagittal planes is essential to accurately delineate the fracture anatomy, assess orbital volume, and visualize the relationship between the extraocular muscles and the bony fragments. However, it is a well-established clinical principle that the diagnosis of muscle entrapment is ultimately clinical. So-called "trapdoor" fractures, where a linear fracture segment opens and snaps shut on orbital tissue, can be notoriously subtle or even invisible on CT scans, yet present with profound clinical signs of entrapment. Therefore, clinical findings must always take precedence over imaging when determining the need for surgical exploration.

Complicating the clinical picture is the frequent association of orbital fractures with direct globe injuries.[10] The same traumatic force that fractures the bone can cause a spectrum of ocular damage, ranging from corneal abrasions and hyphema in the anterior segment to more severe, vision-threatening injuries in the posterior segment, such as retinal hemorrhages, commotio retinae (retinal bruising), retinal tears or detachments, choroidal rupture, and traumatic optic neuropathy. The presence of these injuries

significantly influences the overall management strategy and the ultimate visual prognosis for the patient. A comprehensive ophthalmological assessment is therefore mandatory in every case of orbital fracture. The aim of this report is to present a comprehensive account of a case of traumatic medial rectus muscle entrapment secondary to a medial orbital wall fracture, complicated by concurrent retinal hemorrhage. We seek to detail the diagnostic process, surgical management, and postoperative course, thereby reinforcing key clinical principles for managing this specific and challenging injury pattern. The novelty of this case lies in its classic yet less-frequently reported presentation of isolated medial rectus entrapment, distinct from the more common inferior rectus involvement. It serves as a critical reminder for clinicians that post-traumatic diplopia with an abduction deficit warrants a high index of suspicion for a medial wall fracture. Furthermore, this report uniquely documents the co-existence of both a significant mechanical motility restriction and a direct posterior segment injury (retinal hemorrhage), highlighting the dual nature of the visual threat posed by such trauma and underscoring the necessity of a holistic ophthalmological evaluation that extends beyond the orbital frame to the globe itself.

2. Case Presentation

A 21-year-old male student with no significant past medical or ocular history was referred for an urgent ophthalmology consultation on May 5th, 2024. The patient's profile, as summarized in Figure 1, establishes a crucial baseline of a young, healthy individual, making the subsequent traumatic injuries all the more significant as they occurred in the absence of pre-existing systemic or ocular vulnerabilities. His status as a student underscores the potential for profound, long-term functional and quality-of-life implications should his injuries result in permanent deficits. The inciting event was a high-energy traumatic incident—a motorcycle accident—resulting in direct blunt force trauma to the left periorbital region. This mechanism is critical, as it is well-established to generate the kind of hydraulic forces necessary to fracture the thin, internal walls of the orbit. The patient presented to the clinic one day following this injury, a timeline that is of paramount importance in the context of potential orbital soft tissue or muscle entrapment, where the risk of irreversible ischemic damage escalates rapidly after the initial 24 to 48 hours. Clinically, the patient presented with a constellation of highly specific and alarming symptoms. His chief complaint of "blurred vision" in the left eye immediately signaled a potential organic injury to the globe itself, suggesting that the concussive forces had disrupted the visual axis or damaged the delicate neural tissues within the eye. This was compounded by his second, equally debilitating symptom: "binocular horizontal diplopia." The binocular nature of the double vision indicated a misalignment of the two eyes, while its horizontal characteristic strongly pointed towards a dysfunction of the medial or lateral rectus muscles, the primary drivers of horizontal eye movement. The combination of these two distinct symptoms—one affecting visual acuity and the other affecting ocular alignment—immediately framed this case as a complex injury involving both the globe and the surrounding orbital structures, setting the stage for a comprehensive diagnostic workup.

The initial ophthalmological examination revealed a dramatic and telling asymmetry between the patient's two eyes, as graphically detailed in Figure 2. The right eye (Oculus Dexter, OD) served as a perfect internal control, presenting as entirely normal across all tested domains. Its visual acuity was an optimal 20/20, and all anterior segment structures were clear and quiet, with normal pupillary responses and no external signs of trauma. In stark contrast, the left eye (Oculus Sinister, OS) exhibited a constellation of pathological findings that painted a clear picture of severe, multi-faceted trauma. The most striking finding was the profound reduction in visual acuity to 20/80. This significant impairment immediately indicated that the injury was not confined to the orbit but involved a direct, organic insult to the globe itself,

compromising its ability to process visual information. Crucially, however, the neurological responses of the left eye were intact; the pupil was normally reactive and, importantly, there was no relative afferent pupillary defect (RAPD). The absence of an RAPD was a key diagnostic finding, making a severe traumatic optic neuropathy less likely and pointing towards the retina as the probable source of the vision loss. While the anterior segment structures of the left eye were surprisingly spared, showing no signs of hyphema or corneal injury, the external examination corroborated the history of significant trauma. Marked periorbital edema and ecchymosis were present, alongside hypoesthesia in the distribution of the infraorbital nerve—a classic sign of a fracture involving the orbital floor, where the nerve resides. This combination of a grossly damaged "container" (the orbit, evidenced by swelling and nerve damage) and a functionally compromised "content" (the globe, evidenced by poor vision) confirmed the complex nature of the injury and set the stage for the more detailed motility and funduscopic assessments that would follow.

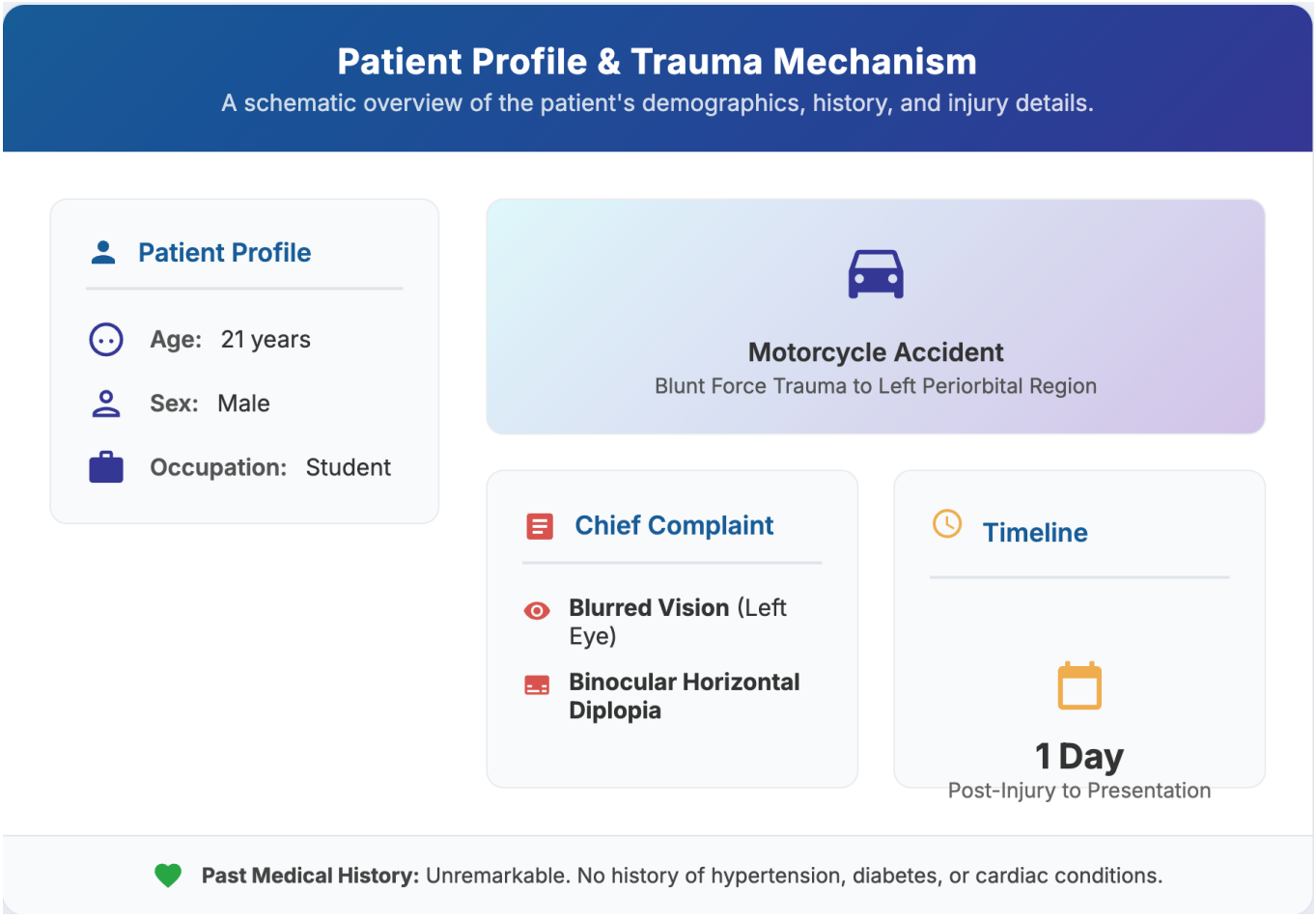

Figure 1. Patient profile & trauma mechanism.

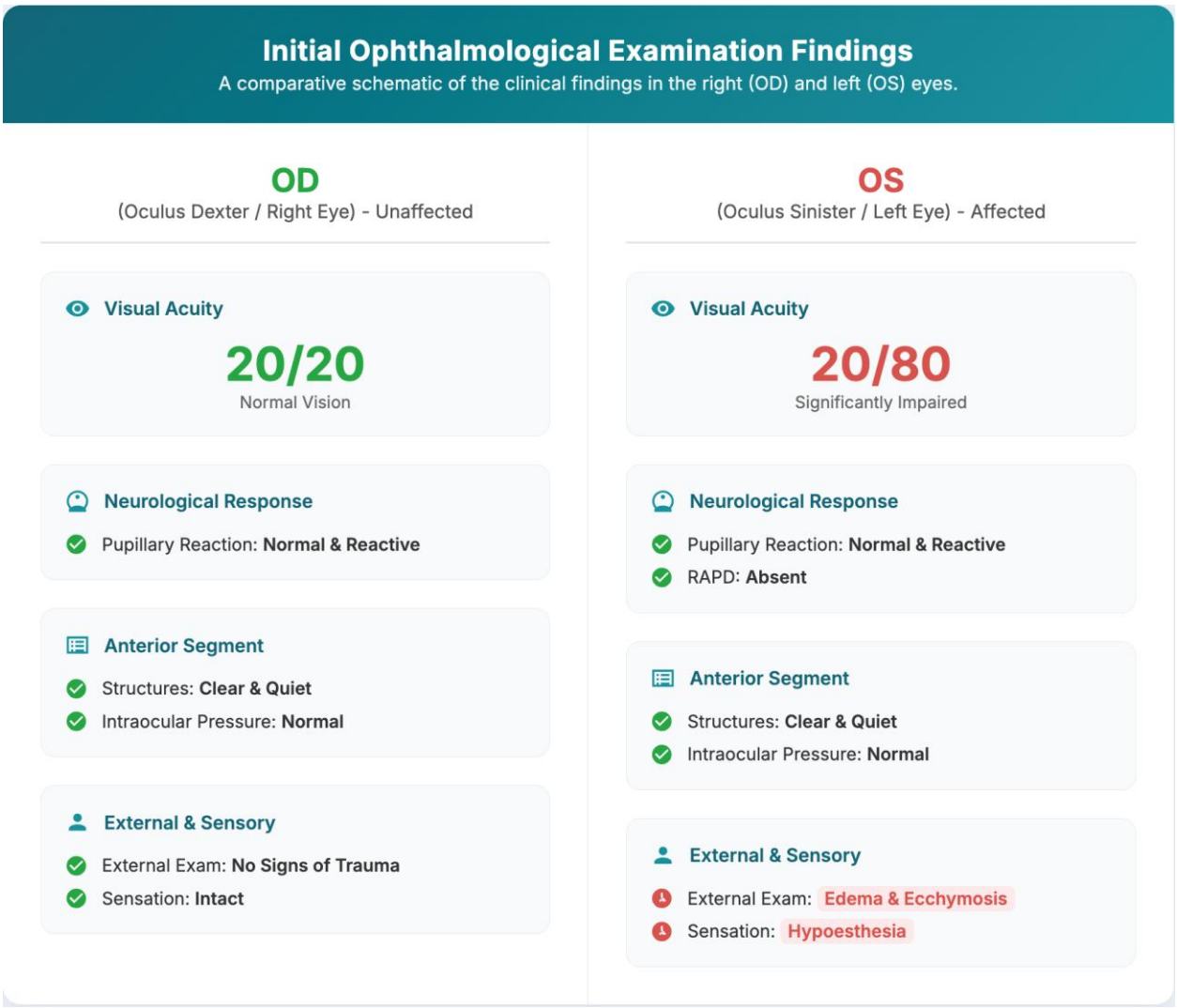

Figure 2. Initial ophthalmological examination findings.

The assessment of ocular motility and alignment, presented schematically and clinically in Figure 3, provides the definitive evidence for the diagnosis of a mechanical restriction in the left eye. The figure masterfully contrasts the physiological normality of the right eye with the profound pathology of the left, serving as the central pillar of the clinical diagnosis. The right eye (OD), depicted in the left column, demonstrates a full and unrestricted range of motion. The schematic shows the globe moving smoothly and completely into all nine cardinal positions of gaze, confirming the intact function of all six extraocular muscles. This is complemented by the key findings of orthotropia, indicating perfect alignment in primary position and full active motility. This side of the figure serves as a crucial baseline, representing the physiological state that surgical intervention aims to restore in the affected eye. In stark contrast, the left eye (OS) exhibits a dramatic and unequivocal pattern of restrictive strabismus. The schematic vividly illustrates a profound abduction deficit; the eye is unable to move into the lateral, superolateral, and inferolateral fields. This failure to abduct is the hallmark sign of medial rectus muscle entrapment. In the primary position, the eye is shown to be esotropic, or deviated inward, a direct consequence of the unopposed tone from the tethered medial rectus muscle. The key findings for the left eye crystallize the

diagnosis. The esotropia and abduction deficit are noted, but the most critical piece of evidence is the positive forced duction test (FDT). The description of a "hard, unyielding stop" on attempted passive abduction is the pathognomonic sign that differentiates this mechanical entrapment from a neurological sixth nerve palsy, where the globe would move freely. The accompanying clinical photograph provides compelling real-world validation of the schematic, clearly showing the left eye's inability to move past the midline on right gaze, thereby confirming the diagnosis and justifying the need for emergent surgical intervention.

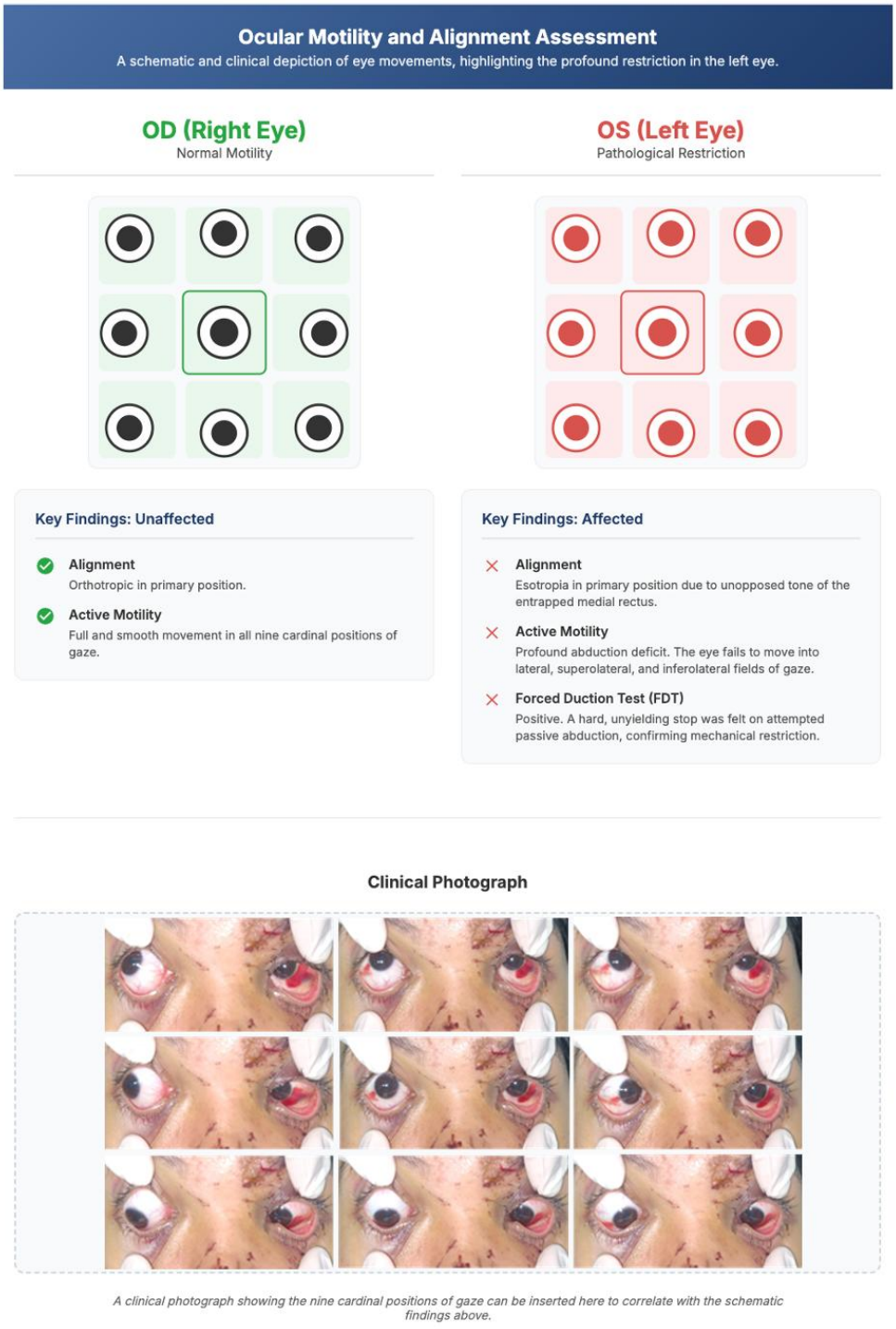

Figure 3. Ocular motility and alignment assessment.

A dilated fundus examination, schematically and clinically detailed in Figure 4, was a critical step in the diagnostic workup, as it revealed the organic cause of the patient's primary complaint of blurred vision. This examination highlights the "dual threat" nature of the injury, demonstrating that even as the orbital fracture compromised ocular function through muscle entrapment, the concussive force of the trauma simultaneously inflicted a direct, sight-threatening injury to the retina itself. The schematic for the right eye (OD) serves as a healthy control, illustrating a pristine posterior segment. The optic disc is well-defined with a normal cup-to-disc ratio, the retinal vasculature follows a normal course, and the macula exhibits a crisp foveal reflex. This depiction of normal anatomy provides a stark and effective contrast to the pathology observed in the affected eye. The left eye (OS) presents a dramatic and troubling picture of traumatic retinopathy. The most prominent findings, clearly illustrated in the schematic and validated by the clinical photograph, are the multiple intraretinal hemorrhages scattered across the posterior pole. These hemorrhages represent the rupture of small retinal vessels from the shearing forces of the impact. Furthermore, the schematic indicates a generalized whitening of the retina, characteristic of commotio retinae or "Berlin's edema." This represents a contusion of the outer retinal layers and photoreceptors, which is the primary cause of the significant reduction in visual acuity to 20/80. Critically, the key findings note that the optic disc itself appears normal, a crucial observation that makes a traumatic optic neuropathy less likely at this stage. The combination of these findings confirms a significant concussive injury to the globe. This part of the examination definitively separated the causes of the patient's symptoms: the diplopia was from the muscle entrapment, but the vision loss was entirely due to the retinal damage depicted here.

Following the definitive clinical diagnosis of mechanical muscle entrapment, high-resolution computed tomography (CT) was performed. The radiological findings, presented schematically and clinically in Figure 5, served two critical functions: first, to provide unequivocal anatomical confirmation of the clinical suspicion, and second, to create a detailed surgical roadmap for the necessary reconstructive procedure. The imaging powerfully visualizes the extent of the craniofacial trauma and the precise mechanism of the patient's debilitating diplopia. The 3D volumetric reconstruction provides a global and intuitive overview of the injury's scale. As shown in the clinical CT images, the reconstruction reveals a complex, multi-bone fracture pattern involving not just the orbit but the associated zygomaticomaxillary complex. This three-dimensional view is invaluable for surgical planning as it allows the surgeon to appreciate the full scope of the skeletal disruption and plan the necessary approaches and fixation points for a stable anatomical reduction. However, the most critical information for this specific pathology is derived from the multiplanar slices, particularly the coronal view, which is presented schematically. The coronal schematic in Figure 5 is the anatomical key to this case, clearly delineating the triad of injuries. It confirms a comminuted fracture of the lamina papyracea, the paper-thin medial orbital wall. Most importantly, it demonstrates the direct cause of the patient's profound abduction deficit: the belly of the medial rectus muscle is shown herniating through this bony defect, becoming physically trapped or "entrapped" within the ethmoid sinus. This image is the radiological correlate of the "hard stop" felt on the forced duction test. Furthermore, the scan reveals a concurrent fracture of the orbital floor, which corresponds to the patient's clinical finding of infraorbital nerve hypoesthesia. Figure 5 transitions the diagnosis from a clinical certainty to an anatomical reality. It provided the surgical team with the precise location of the entrapped muscle and the exact architecture of the bony defects, information that was indispensable for performing an efficient and successful release and reconstruction, ultimately restoring the potential.

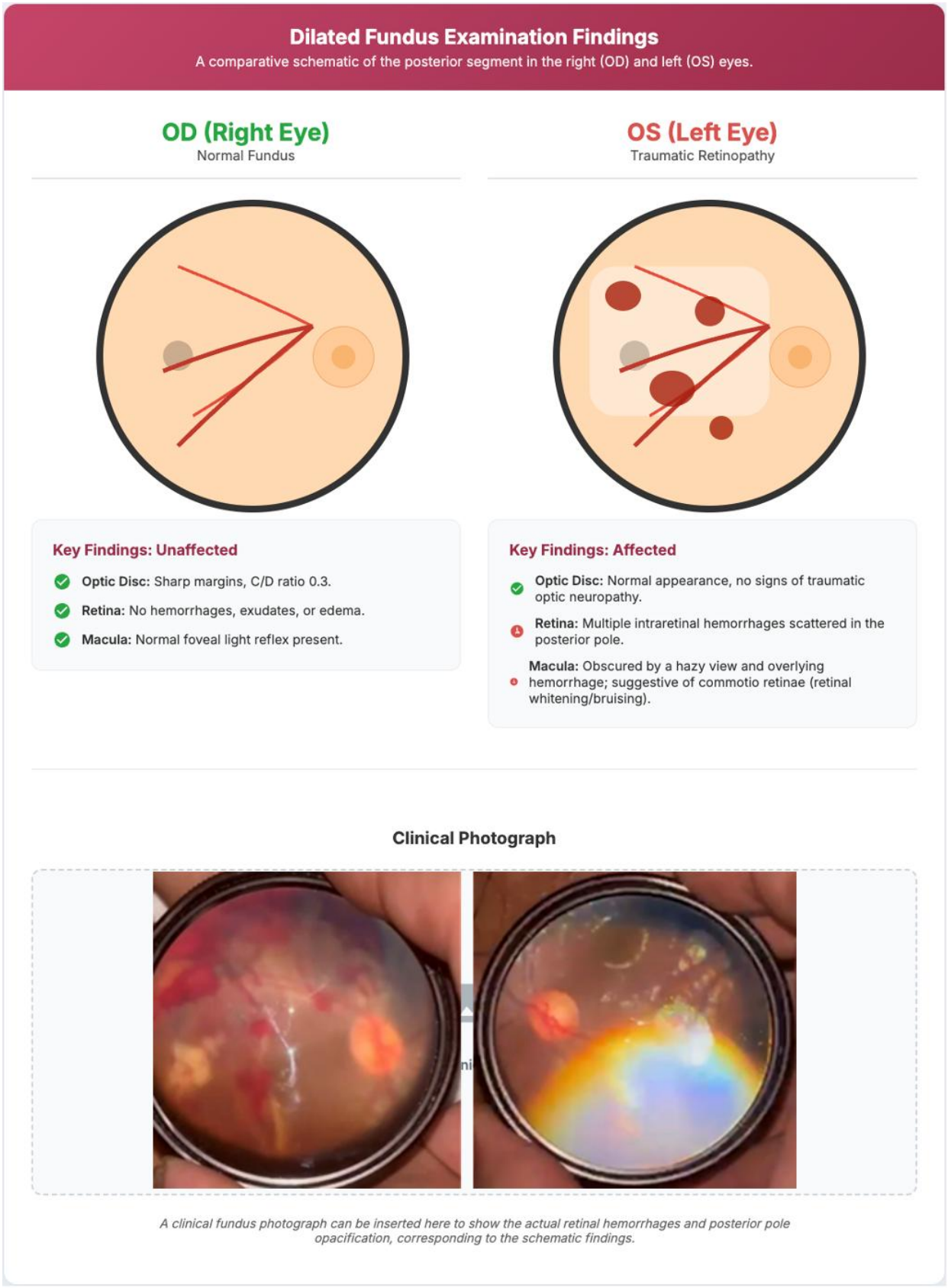

Figure 4. Dilated fundus examination findings.

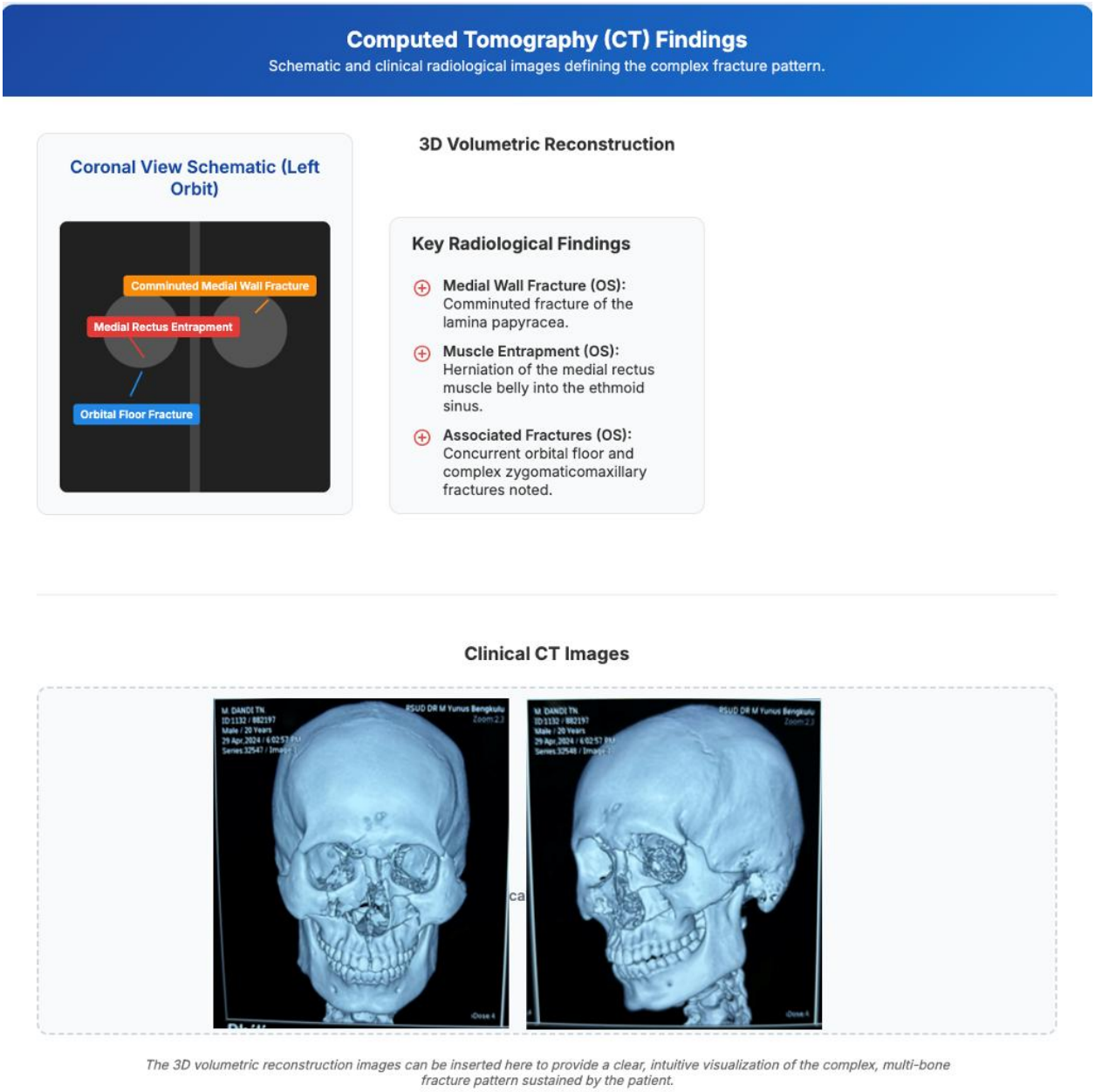

Figure 5. Computed tomography (CT) findings.

The clinical pathway for this complex case, from the synthesis of diagnostic data to the immediate postoperative assessment, is systematically summarized in Figure 6. The comminuted medial orbital wall fracture with entrapment, the associated complex facial fractures, and the sight-threatening traumatic retinopathy. This triad of diagnoses codifies the "dual visual threat," acknowledging both the functional (entrapment) and organic (retinal) components of the injury. This comprehensive diagnostic summary served as the definitive justification for the urgent and targeted surgical plan that followed. The central panel of Figure 6 details the critical steps of the surgical intervention. The choice of a subciliary approach is noted, and a decision is made to provide the wide surgical field necessary to address the multiple fracture sites. The two primary goals of the operation are clearly stated: the

meticulous release of the incarcerated medial rectus muscle to restore the potential for normal movement, and the anatomical reconstruction of the bony defect with a rigid titanium mini-plate to provide a stable and durable repair. This section underscores the precision and purpose-driven nature of the surgical procedure. Finally, the third panel provides a snapshot of the patient's clinical status on the first postoperative day, offering a crucial early look at the surgical outcome. The results are a mixture of immediate success and expected sequelae. The most significant finding, highlighted in green, is the resolution of the mechanical restriction, confirmed by a negative postoperative Forced Duction Test. This is the paramount indicator of a successful surgical release. However, as expected, the patient's active motility remained limited due to significant postoperative edema and the direct contusion sustained by the muscle, resulting in a temporary esotropia, as shown in the clinical photograph. Furthermore, the visual acuity and retinal findings remained unchanged, a stark reminder that the surgical repair of the orbit does not treat the separate, concussive injury to the globe itself. This immediate postoperative assessment is therefore critical, as it confirms the success of the mechanical intervention while simultaneously setting the stage for.

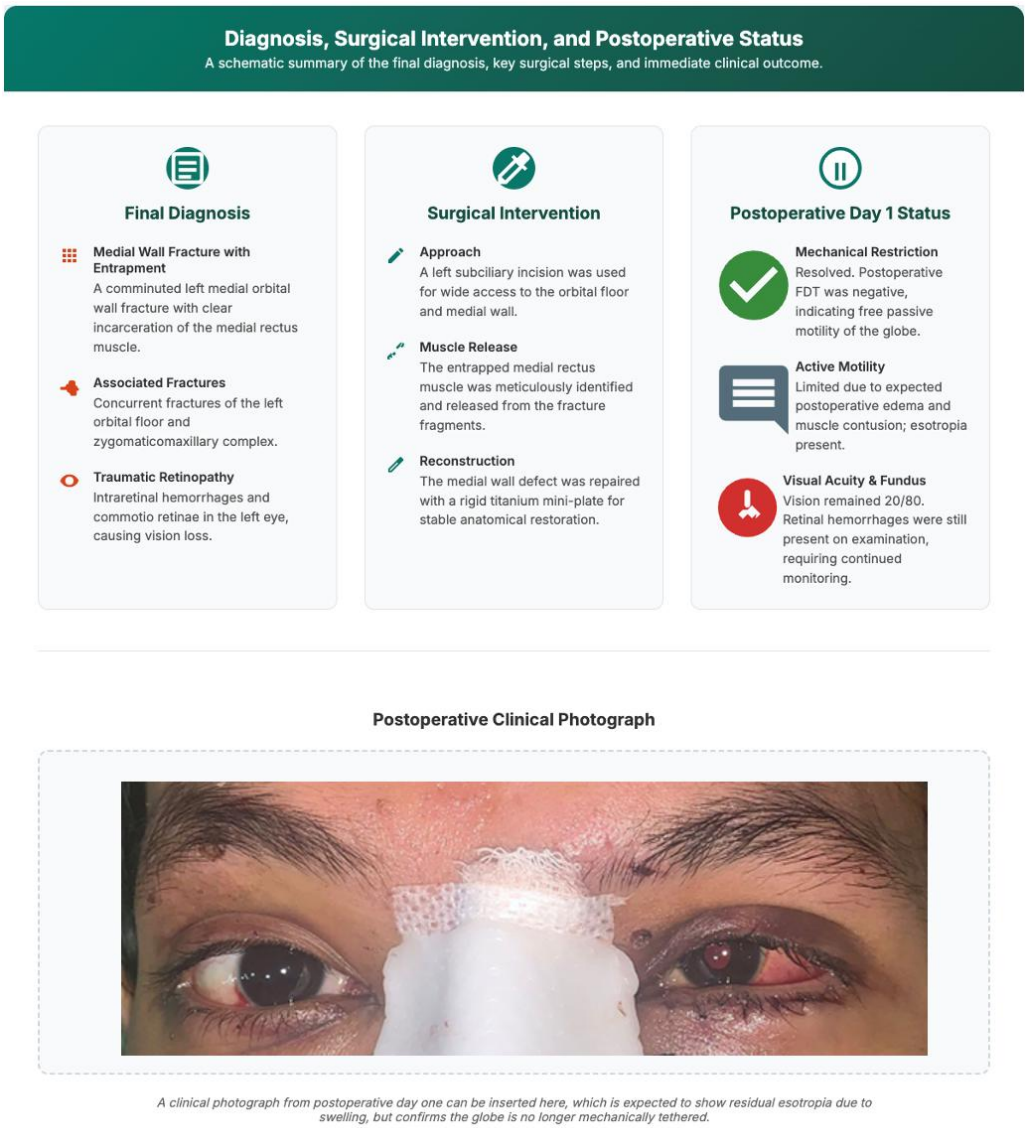

Figure 6. Diagnosis, surgical intervention, and postoperative status.

3. Discussion

The case presented provides a quintessential example of a severe orbital injury, compelling a deep and detailed exploration of the intricate interplay between orbital anatomy, trauma biomechanics, pathophysiology, and the principles of clinical diagnosis and surgical management. The discussion of this patient's journey from injury to intervention illuminates several fundamental truths in the field of ophthalmic plastic and reconstructive surgery. The findings not only confirm established theories but also serve as a powerful narrative emphasizing the urgency and precision required to manage such complex cases successfully. The foundation of understanding this injury lies in the unique and paradoxical architecture of the human orbit. It is a structure of contrasts, a bony fortress with remarkably fragile points. The four walls of the pyramidal orbit are designed to protect the globe, a delicate and vital sensory organ.[11] The orbital rims, composed of thick cortical bone from the frontal, zygomatic, and maxillary bones, are immensely strong and capable of absorbing and dissipating immense kinetic energy from direct impacts. This is the orbit's primary defense mechanism. However, this robust frame encases a space with walls of dramatically varying thickness. The lateral wall is thick and resilient, while the roof is substantial, protecting the globe from the frontal lobe of the brain.[12] The true vulnerabilities lie in the floor and the medial wall. The orbital floor, forming the roof of the maxillary sinus, is a thin plate of bone. Even more delicate is the medial wall, the lamina papyracea of the ethmoid bone, which is aptly named for its paper-thin consistency. It is this inherent structural fragility that dictates the patterns of orbital fractures following blunt trauma.[13]

The prevailing theory explaining the mechanism of this patient's injury is the "hydraulic theory." When an object with a diameter larger than the orbital aperture, such as a fist or, in this case, the surface of the asphalt, strikes the periorbital region, the force is transmitted to the orbital contents.[14] Because the orbital volume is fixed and its contents are largely incompressible fluids and soft tissues, the impact generates a sudden, massive spike in intraorbital pressure. This pressure wave radiates outwards and seeks the path of least resistance. It is inevitably directed towards the thinnest sections of the bony cavity: the floor and the medial wall. The bone buckles and fractures outwards, into the adjacent maxillary or ethmoid sinuses, creating a "blowout" fracture. This sudden decompression of the orbital cavity allows orbital fat and, critically, extraocular muscles to herniate through the bony defect. In this patient, the force vector was such that the lamina papyracea yielded, creating a trap for the adjacent medial rectus muscle. The concomitant floor fracture further illustrates the widespread transmission of this hydraulic force throughout the weaker aspects of the orbit.[15]

Once the medial rectus muscle became incarcerated within the ethmoid fracture fragments, a cascade of deleterious pathophysiological events was initiated. This process is far more complex than a simple mechanical tethering. The immediate consequence is, of course, the profound restriction of ocular motility. The medial rectus, responsible for adduction (turning the eye inward), acts as a powerful antagonist to the lateral rectus, which controls abduction (turning the eye outward). When the medial rectus is physically trapped, it cannot elongate. This creates an impassable mechanical barrier to abduction. Any attempt by the patient to look laterally with the affected eye is met with failure, and the strong, unopposed tone of the trapped medial rectus pulls the eye inward, resulting in the esotropia observed in the primary position.[16] This mechanical mismatch between the two eyes is the direct cause of the patient's binocular diplopia.

However, the more insidious and time-sensitive pathology is the vascular compromise to the entrapped muscle. The extraocular muscles have a rich blood supply, essential for their high metabolic activity.[17] When the muscle belly is compressed and strangulated by bony fragments, its arterial inflow and venous outflow are severely impeded. This leads to a state of ischemia. The muscle tissue, starved of oxygen

and nutrients, begins to suffer. Capillary permeability increases, leading to interstitial edema, which further exacerbates the compression within the confined fracture site, creating a vicious cycle analogous to a compartment syndrome.[18] If this state of ischemia is allowed to persist, the muscle cells undergo necrosis. The body's natural response to necrotic tissue is inflammation and subsequent repair through fibrosis. Over a period of days to weeks, the necrotic muscle is replaced by non-contractile scar tissue. This fibrotic transformation is irreversible. Even if the muscle is surgically released at this later stage, it will have lost its elasticity and contractile function. It becomes a shortened, stiff band, resulting in a permanent, intractable, restrictive strabismus and lifelong diplopia. This pathophysiological sequence is the central reason why acute muscle entrapment is considered a true surgical emergency. The window of opportunity to prevent irreversible ischemic damage is narrow, generally accepted to be within 24 to 48 hours of the injury.

This case powerfully illustrates the absolute primacy of the clinical examination in the diagnosis of muscle entrapment. While modern imaging technology is a remarkable adjunct, it can never replace a hands-on physical assessment. The patient's reported symptoms of diplopia and the observed esotropia and abduction deficit were highly suggestive, but these signs could also be consistent with a traumatic sixth cranial nerve palsy. The abducens nerve, due to its long and tortuous intracranial course, is susceptible to stretching and injury in the context of head trauma. The definitive diagnostic maneuver that resolved this differential was the forced duction test (FDT).[19] The sensation transmitted through the forceps during an FDT is profoundly informative to the experienced clinician. In a neurogenic palsy, the globe can be moved passively with little to no resistance; it feels "free." In stark contrast, the finding in this patient was a hard, unyielding stop when attempting to abduct the eye. This sensation is unmistakable and pathognomonic for a mechanical restriction. It provided an unequivocal diagnosis at the bedside, independent of any imaging. The decision to proceed with emergency surgery was made on the strength of this finding alone. The subsequent CT scan served to confirm the anatomical location of the entrapment and to provide a detailed roadmap for the surgical intervention, but it did not establish the diagnosis; the FDT already had.

Furthermore, the potential for the oculocardiac reflex (OCR) added another layer of urgency to this case. The OCR is a dramatic and potentially lethal physiological response.[20] The neuroanatomy of this reflex arc begins with the afferent limb: stretch receptors within the belly of the extraocular muscles detect the traction caused by the entrapment. These sensory signals travel via the ophthalmic division of the trigeminal nerve (CN V1) to the main trigeminal sensory nucleus in the brainstem. From there, short internuncial fibers connect to the visceral motor nucleus of the vagus nerve (CN X). This forms the efferent limb of the reflex. The firing of the vagus nerve leads to a powerful parasympathetic discharge, causing negative chronotropic effects on the heart's sinoatrial node. Clinically, this can manifest as sudden, profound sinus bradycardia, atrioventricular block, nausea, syncope, or even asystole. While not overtly present in this patient at rest, the reflex could have been triggered at any time by attempts to move the eye or, critically, during surgical manipulation. The only definitive way to treat and eliminate the trigger for the OCR is to surgically release the entrapped muscle. This systemic, life-threatening potential elevates muscle entrapment from a vision-threatening condition to a systemic emergency, mandating close collaboration between the surgical and anesthesia teams.

The surgical management undertaken was guided by clear and established principles. The goal was not merely to repair the broken bone but to restore normal function. A subciliary approach provided excellent access to both the orbital floor and the medial wall. The most delicate and crucial step of the operation was the identification and extrication of the medial rectus muscle from the bony fragments of the lamina

papyracea. This required microsurgical precision to avoid causing iatrogenic trauma to the muscle or its nerve supply. Once the muscle was confirmed to be free, the second goal was to reconstruct the anatomical defect. Leaving the defect open would risk late enophthalmos (posterior sinking of the eye due to increased orbital volume) and the potential for re-entrapment of soft tissues in scar. The placement of a titanium mini-plate served to restore the normal anatomical barrier between the orbit and the ethmoid sinus, providing a smooth surface for the muscle to glide over and ensuring the maintenance of normal orbital volume. The final, confirmatory step of the procedure was the intraoperative FDT. The ability to passively move the globe freely in all directions after the repair provided immediate, tangible proof of a successful surgical outcome.

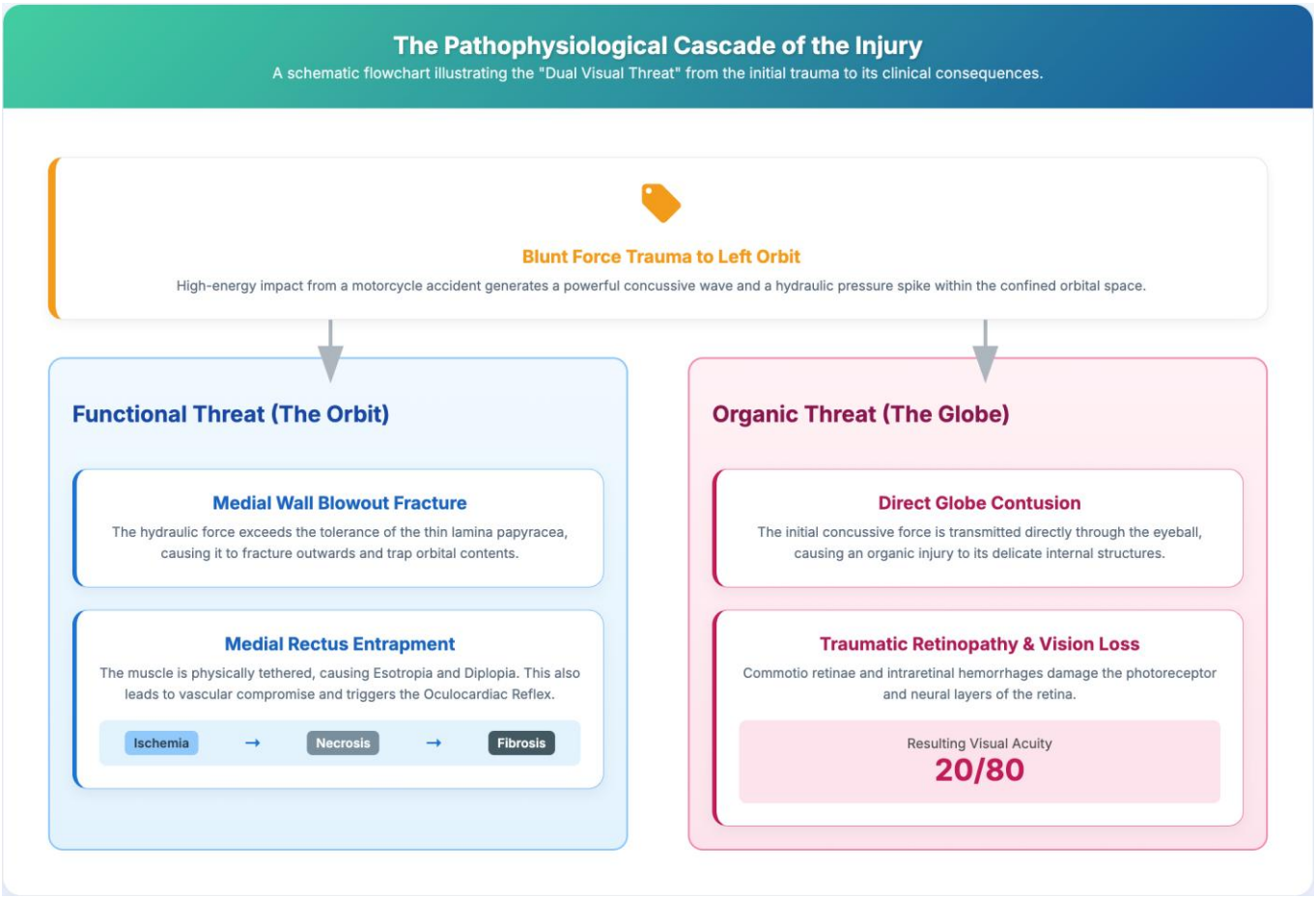

Figure 7. The pathophysiological cascade of the injury.

The entirety of this complex clinical case, from the initial traumatic event to its divergent and severe consequences, is masterfully synthesized and conceptualized in the pathophysiological flowchart presented in Figure 7. This single, catastrophic input is shown to generate two primary forms of energy transfer. The first is a hydraulic pressure spike within the confined, fluid-filled space of the orbit. The second is a direct concussive shockwave that is transmitted through the globe itself. Figure 7 then elegantly divides the consequences of these two energy transfers into two parallel but interconnected pathways, each

representing a distinct clinical emergency that demands a unique diagnostic and therapeutic focus. The immense pressure is shown to cause a blowout fracture of the weakest point, the lamina papyracea, or medial orbital wall. This structural failure leads directly to the next critical event: the herniation and entrapment of the medial rectus muscle. As the figure illustrates, this single event has profound consequences. Mechanically, it tethers the muscle, leading to the clinical signs of esotropia and debilitating diplopia. Physiologically, however, it initiates a far more insidious and time-sensitive pathology. The flowchart visually represents the dire vascular compromise that occurs, leading to a well-defined and irreversible cascade of ischemia, necrosis, and eventual fibrosis—a sequence that, if not interrupted by surgery, would result in a permanent loss of muscle function. Furthermore, this pathway correctly highlights the systemic danger of the oculocardiac reflex, a life-threatening risk triggered by the very same muscle traction. This entire blue-coded pathway represents the threat to the patient's binocular function and, in the case of the OCR, his systemic stability. This pathway is entirely separate from the bony fracture. Here, the shockwave causes a direct contusion to the delicate neural tissues of the eye, leading to a diagnosis of traumatic retinopathy. The flowchart specifies the components of this injury—commotio retinae and intraretinal hemorrhages—and correctly identifies them as the direct cause of the patient's primary complaint of blurred vision. The final, stark outcome of this pathway is the objective measurement of organic vision loss: a visual acuity of 20/80. This red-coded pathway represents the primary, and potentially permanent, threat to the patient's sight in the affected eye. By presenting these two pathways side-by-side, originating from a single traumatic event yet resulting in fundamentally different types of injury, Figure 7 provides an exceptionally clear and powerful educational tool. It masterfully demonstrates why the management of severe orbital trauma requires a dual focus, as a technically perfect repair of the functional threat on the left may not alter the final, organic visual outcome determined by the pathway on the right. It is the complete conceptual summary of this entire case.

Finally, the discussion of this case would be incomplete without addressing the significant concurrent injury to the globe itself. The patient's presenting complaint was not only diplopia but also blurred vision, and his visual acuity was reduced to 20/80. This visual loss was not a consequence of the fracture or the muscle entrapment; it was the result of the direct concussive force transmitted through the globe. This force generated shockwaves that caused traumatic retinopathy and commotio retinae. Commotio retinae, or Berlin's edema, represents a bruising of the outer retinal layers. The shockwave disrupts the delicate interface between the photoreceptor outer segments and the retinal pigment epithelium (RPE), leading to cellular damage and a transient whitening of the retina. The intraretinal hemorrhages were caused by the rupture of small retinal capillaries from the same shearing forces. This posterior segment injury is a distinct pathological process from the orbital fracture, yet it was caused by the same traumatic event. It highlights the critical need for a comprehensive ophthalmological assessment in every trauma patient. A surgeon could perform a technically perfect orbital wall reconstruction, only for the patient to have a poor visual outcome due to an undiagnosed retinal detachment or traumatic optic neuropathy. The prognosis for this patient's vision is therefore guarded and entirely dependent on the resolution of the retinal injuries, not the success of the orbital surgery. The dual pathology present in this case serves as a powerful reminder that the orbit and the globe are intimately related yet must be assessed and managed as separate, though connected, entities. The successful management of orbital trauma is a multi-faceted endeavor that requires an appreciation of complex anatomy, an understanding of time-sensitive pathophysiology, a reliance on fundamental clinical skills, and a holistic approach that addresses all aspects of the injury.

4. Conclusion

This case report provides a definitive and compelling narrative of a significant ophthalmological emergency, highlighting the critical intersection of orbital mechanics, muscle physiology, and ocular trauma. The successful outcome for this young patient hinged on the rapid and accurate clinical identification of medial rectus muscle entrapment, a diagnosis secured not by advanced imaging, but by the fundamental and indispensable forced duction test. This finding, confirming a mechanical restriction, immediately activated a protocol for urgent surgical intervention, correctly prioritizing the prevention of irreversible ischemic fibrosis in the entrapped muscle and averting the potentially catastrophic systemic consequences of the oculocardiac reflex. The surgical release and anatomical reconstruction of the medial orbital wall successfully restored the potential for normal ocular motility, representing a triumph of timely and precise surgical care. However, this case offers a broader and more profound lesson: the force that shatters bone can simultaneously inflict subtle, sight-threatening damage within the globe itself. The presence of traumatic retinopathy, the true cause of the patient's diminished vision, underscores the absolute necessity of a comprehensive ophthalmological evaluation that extends beyond the fracture lines on a CT scan. Ultimately, this report stands as a powerful testament to the principle that a successful outcome in complex orbital trauma is achieved not just by mending the socket, but by vigilantly safeguarding the jewel within it.

5. References

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