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OPTIC NEUROPATHY IN ORAL AND MAXILLOFACIAL TRAUMA

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ABSTRACT

Mid-facial injuries directly or indirectly could cause visual consequences. The diagnosis and timing of intervention is a crucial aspect of the treatment of these maxillofacial injuries, and the prognosis of these injuries directly depends on the approach of the oral and maxillofacial surgeon to manage these patients. This article presents the rationale and treatment modalities for traumatic optic neuropathy.

Keywords: *Maxillofacial Trauma; optic canal; Traumatic optic neuropathy*

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ORAL VE MAKSILLOFASIYAL TRAVMADA OPTIK NÖROPATI

ÖZ

Orta yüz yaralanmalarında, direkt veya indirekt görme bozukluğuna sebep olabilir. Böyle yaralanmalarda ve ona bağlı gelişen nöropatide ayrıntı tanı ve medikal veya cerrahi müdahale zamanlanması hastanın tedavi başarısı için en önemli faktördür. Bu derleme, travmatik optik nöropati, tedavi gerekçe ve yaklaşımlarına göre açıklamaktadır.

Anahtar Kelimeler: *Maksillofasiyal Travma; optik kanal; Travmatik optik nöropati*

Introduction

Around half a million people globally are becoming blind annually, which is a huge burden on society. Even a slight vision loss is considered great morbidity for the patient. The trauma may not be the leading cause of blindness in the population, but it is the most common preventable cause. Most non-congenital external monocular blindness is due to facial trauma. At present, there are around 18 million blind cases globally due to traumatic accidents. The incidence of blindness post-trauma is between 2–14%.[1–6] The 7.4% of vision loss in Turkey is associated with optical nerve atrophy, which could have initiated due to maxillofacial trauma.[7] The globe injury is consulted and treated by the ophthalmologist. However, non-globe injuries and indirect trauma concomitant with a midfacial fracture are attended by the maxillofacial surgeons. One of the pathologies behind the visual impairment post-trauma is traumatic optic neuropathy (TON). About 10% of the craniofacial fractures are associated with visual complications, and 2.5% have been diagnosed with TON.[8–10] Intracranial direct forces to optic ganglion or optical nerve sheath and indirect ischemic pressure or peripheral inflammation cause neural cell degeneration and visual impairment. In this article, we presented a review of treatment modality for post-maxillofacial TON.

Diagnosis

The general ophthalmologic examination includes visual acuity, visual fields, central visual function, binocular and peripheral visual field, fundoscopy and structural examination, motility, forced duction test, globe position, and pupil reflexes. The viable blurriness or visual impairment should be differentiated from an anatomic standpoint. The post-traumatic relative afferent pupillary defects need further computerized tomographic (CT) examination, and the extension of fracture lines to the optical canal is investigated (Fig 1.). However, the optic canal findings on the tomographic image do not correspond to the severity of TON. The zygomaticomaxillary complex fracture which is the most concomitant fracture, Le fort II and cranial bone fracture are other common probable findings respectively. The complexity of trauma such as intracranial bleeding, coexisting unconsciousness, extraocular foreign body or ruptured globe, and late development of symptoms make the diagnosis difficult.[11–13] In the case of bilateral injuries or the unreliability of other findings, the flash visual evoked potentials test could recognize the severity and prognosis of optic

neuropathy. This method records the electric activity of the brain to light stimulation like electroencephalography but amplifies the perception of the brain, especially to light. Less than 50% of the amplitude of the normal side would be considered as poor prognosis.[14]

Anatomy of pathology

The course of the optic nerve to proximal ganglion has distincts anatomic sections, including intraocular, intraorbital, intracanalicular, and intracranial. Most of the length of this nerve path is surrounded by soft tissue like a cushion that could absorb the trauma forces and prevent delivering it to the optic nerve. The intracanalicular portion of this pathway is the narrowest and never comes in tight contact with periosteum and bone, and any fracture segment could impinge the nerve or put some strain to neural cells. About 50% of TON in facial trauma cases show optical canal fracture in their CT findings.[15] Secondary to blunt trauma, any bleeding or inflammation in a non-expandable bony canal leads to jeopardizing the vascular supply and irreversible loss of neural cells. The treatment methods mostly focus on the pathophysiology to reduce the pressure on the sensitive optic nerve in intracanalicular segment.[16] The other segment's neuropathy treatments are beyond the focus of this article and may concern other specialties (intracranial segment for neurosurgeons and intraocular for ophthalmologists).

Medical management

The numerous animal trial studies introduced medications for neuroprotection or neuro-regeneration. The available drugs were also used in clinical or animal trials for the treatment of TON namely, the tissue necrosis factor-α (TNF-α), nitric oxide synthase inhibitors, oncomodulin, brain-derived neurotrophic factor, non-encephalitogenic myelin peptides, erythropoietin, and β/γ-crystallin.[17–22] The mechanism behind these medications is restricting the apoptosis of axons of retinal ganglion cells. The clinical trial phase is going on for most of these medicines, following which it can be used in practice.

The steroids are another option that have a long-term application and are successful in many cases. However, the method of administration and dosage is debatable. A range of continuous low doses to mega-doses has been administered. The steroids could inhibit oxygen free radical-induced lipid peroxidation and reduce the swelling, thus acting as a neuroprotective agent. However, the effect of steroid administration on recovery and ganglion stability of injured neurons is controversial.[23,24] Back in 1980 and 1990, there was a guideline presented by the National Acute Spinal Cord Injury Study (NASCIS), which was based on clinical trials of 1292 patients with acute spinal injuries. The result of these studies favored starting the use of methylprednisolone 3–8 h post-injury and continuing. There are controversies on the degree of improvement, reproducibility of study, and application to other neural injuries like TON.[25] A poll of 12303 patients who received corticosteroids post-head injury, showed an increase of risk of death by 18% which made its administration to post-traumatic patients more controversial. The current trend of steroid treatment is optional for spinal injuries and is not indicated in head injury.[26]

Since the benefits of steroids questioned the treatment protocols have changed from time to time, and there is no standard regimen. The mega doses were introduced by NASCIS. The initial dose was a single shot of 30 mg/kg of methylprednisolone and was continued with 5.4 mg/kg/h for 23 h. The dosage from 5399 to 2000 mg is considered very high, and less than 500 mg is a high dose.[27] Levin et al. found that the dose, timing, and length of the regime were independent of the degree of visual recovery. Considering other possible side effects, the more tapered doses may offer the same results. The treatment trend changed toward the high dosage and long-acting drugs.[28,29]

Surgical Management

In 1922, Pringle investigated autopsies of 122 patients with head injuries who were comatose post-trauma. He found hemorrhage in optic nerve sheath and hypothesized that the bleeding promoted nerve dysfunction and degeneration. Crompton investigated a similar group of patients and concluded that the neural hemorrhage was seen in 86% of cases.[28] In another study, ischemic necrosis associated with hemorrhage was found in intracanalicular segment in about 35.7% patients.[29] To prevent the ischemic degeneration, the intracanalicular segment is surgically opened, and the pressure over the optic nerve is removed. The primary-historical transcranial approaches were not very promising, and most clinicians shifted to medical strategies at that time.[30] With the broadening of our knowledge on anatomy and physiology of optic nerve along with the advancement of instruments, surgery is becoming a more predictable option. Since nerve decompression is the primary aim of the intervention, any optical nerve disruption, intracranial accidents, or complete atrophy of nerve would be a contraindication of the procedure. For better prognosis, the surgery should be decided within three days of injury, as subsequently, the prognosis would decrease dramatically.[31]

There are three major surgical approaches to the intracanalicular segment of the optic nerve. The transcranial approach was historically the first approach to TON. The craniotomy is performed on the semi-coronal incision. The extradural dissection continues caudally to anterior clinoid. The roof of the optic canal is opened through the removal of clinoid and optic strut (Fig 2.). In some cases, these structures are very thick, and small instruments like micro curette or rongeurs are ineffective. Thus, the bone should be removed by round bur under bolus irrigation to prevent any thermal damage. The extension of clinoidectomy to the medial side would expose the cavernous sinus, and bleeding should be controlled. The release of the falciform ligament is debatable, and if this ligament needs to be released through a dural incision and arachnoid dissection, the ligament could cut from the medial aspect of the optic nerve(Fig 3.). This maneuver completely decompresses the nerve from the dura ring, but the dura should be carefully repaired and sutured.[27–32] The transethmoid-orbital approach is another method that is very fast and does not need any sophisticated equipment. The manipulation of orbital content may result in other complications, which makes it unfavorable for the inexperienced surgeons in orbital surgery. The location of the incision depends on the surgeon's experience or preference, which could be transcaruncular or

superior-medial eyelid crease. The dissection is toward the posterior lacrimal crest. Most of the articles explained the procedures directly cut all components to the periosteum; however, the authors believe that most of these components are unexpendable and should be preserved. The medial canthus ligament, jones', and horn's muscles are essential functional structures and retracted medico-inferior to preserve them. Globe is also retracted by a malleable retractor and trochlea is identified superiorly and avoided. Posterior to lacrimal crest, periosteum incises, and elevation continues till identification of the fronto-ethmoidal suture and followed posteriorly. The anterior and posterior ethmoidal arteries coagulate. The optical canal is 4 to 6 mm posterior to posterior ethmoidal artery. The medial wall of the optical canal is removed by micro bur and irrigation including the lateral wall of ethmoidal bone (Fig 4.). The entrance of the canal is usually thick and osteotomy needs more meticulous management. The ethmoidal part and posterior section of the canal could be manipulated by a micro curette (Fig 5.). The suturing of transcaruncular closure is optional but the trans eyelid approach needs to be sutured cutaneously.[31,33] The lateral orbital approach is not suggested and the only indication for canthotomy is the incidence of retrobulbar hemorrhage which is beyond the scope of this article.

The advancement of endoscopic technology is instrumental in the surgeon's hands. Nowadays, the endoscopic non-invasive approach is a routine surgical practice. The advantages of this technique are reducing surgical time and hospitalization. The common disadvantages are expensive instruments and learning curve of surgeons to adapt. The endoscopic optic nerve decompression technique is similar to transnasal functional endoscopic sinus surgery. The 0 and 30-degree rigid endoscope is used. The middle turbinate could be removed or lateralized (or medialized if trans ethmoidal approach is required), and endoscope advances the posterior superior to the ostium of the sphenoid sinus. The sphenoidotomy starts at this point, and the inferior wall of the sinus is completely removed (Fig 6-7.). The identification of sphenoid anatomy is crucial before any advancement. The lateral and medial optico-carotid recess, optic strut, carotid artery, and optic nerve protuberance (as a bony bulge to recess) are significant compartments (Fig 8.). The bony optic strut is removed by a micro bur, and intracanalicular segment optic nerve is visualized (Fig 9.). The nerve decompression is performed by removing the bone from the distal to proximal side. Bony structure in this segment is weak, and micro curette is required for bone removal (Fig 10). The carotid bulge should be avoided. The incision over optic nerve sheath to release internal pressure is controversial due to cerebrospinal fluid leak, and it should not damage any vascular component.[34]

The approach to selecting the correct technique is a balance between surgeon's experience, equipments, and point of intervention (Fig 11.). Endoscopic transnasal is the method of choice for the non-fractured cases. However, if there is fracture depending on the location of fracture, the approach may change (e.g., transcranial for associated frontal fractures or transorbital for associated orbital fractures).

Conclusion

Various modalities and approaches have been used for the treatment of TON. However, clinical trials comparing modalities are scarce. Chou et al. compared untreated TON to interventional treatment and found that there was a significant improvement in the interventional treatment group, but the difference of improvement among intervention modalities (medicinal/ surgical) was not significant. The untreated group had no visual improvement, and the authors concluded that the patients with TON should accordingly receive surgical or medical treatment.[35] The study of stepwise combining medicinal and surgical treatment modalities showed that 76% of cases were not improved by sole high-dose steroid therapy and surgical intervention improved or at least stabilized their visual acuity.[36] About 30–70% of the patients treated surgically (by any modalities) had visual improvement; however, the transnasal endoscopic approaches had the more consistent result for 51-58%.[37-40] The prognosis mostly depends on the severity of injury and timing of intervention than the modalities. The current trend is initial steroid therapy, and depending on advancement, the choice of surgery should be considered. The diagnosis still has the most important role in the management of these cases.[41] Delay in surgical intervention could lead to severe visual impairment or blindness, and this is a life-time burden for the patients.

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Legend of figure:

Figure 1: CT view of optical canal fracture (arrow: fracture segment)

Figure 2: Transcranial exposure of optical nerve canal and extradural view (the curette removing the roof of optic canal and arrow indicate the bone margin of anterior clinoid, tip of suction toward the optic never)

Figure 3: Inradural view of 250 degree decompressed optic nerve with falciform ligament release (tip of suction toward the optic never)

Figure 4: Optical nerve exposure via the extended upper-eyelid incision (optical nerve: blue arrow, extended eyelid incision line: yellow arrow).

Figure 5: External trans-orbitoethmodial approach

Figure 6: Anatomic view of sphenoid sinus and it's relation to carotid artery (C.A.) and optic nerve.

Figure 7: Transnasal sphenoidotomy to access the sphenoid sinus.

Figure 8: After sphenoidotomy opticocarotid recess identified, posterior to this recess is optic nerve bulge and anterior to it the carotid bulge which should avoided.

Figure 9: The optic bulge is identified after strut remova.

Figure 10: Unroofing the optical nerve with a delicate instrument (boney edges indicate with arrows and optic nerve in middle)

Figure 11: The circumferential access of each approach to decompress optical nerve (transcranial: blue, trans-orbitoethmodal: red, endoscopic trans-nasoethmoidal: green)

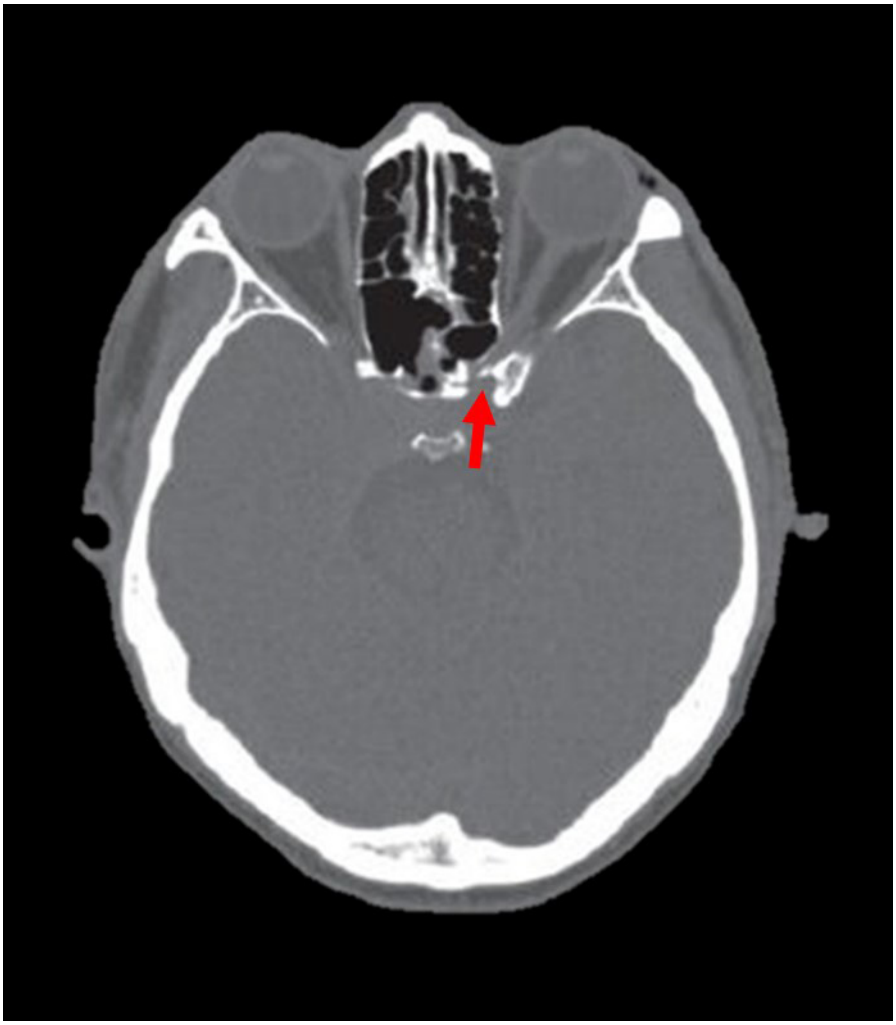
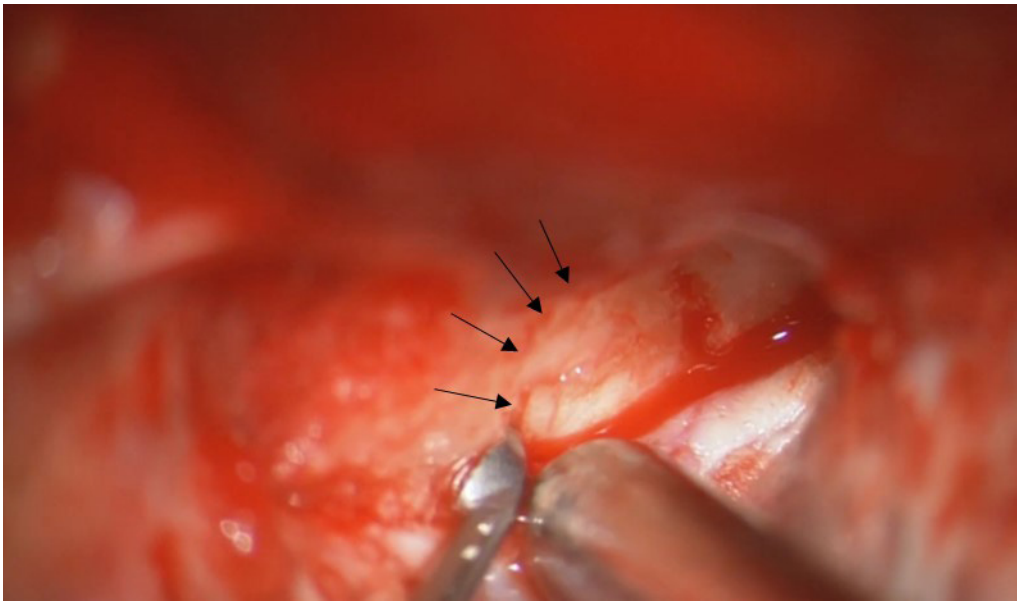


Figure 1.



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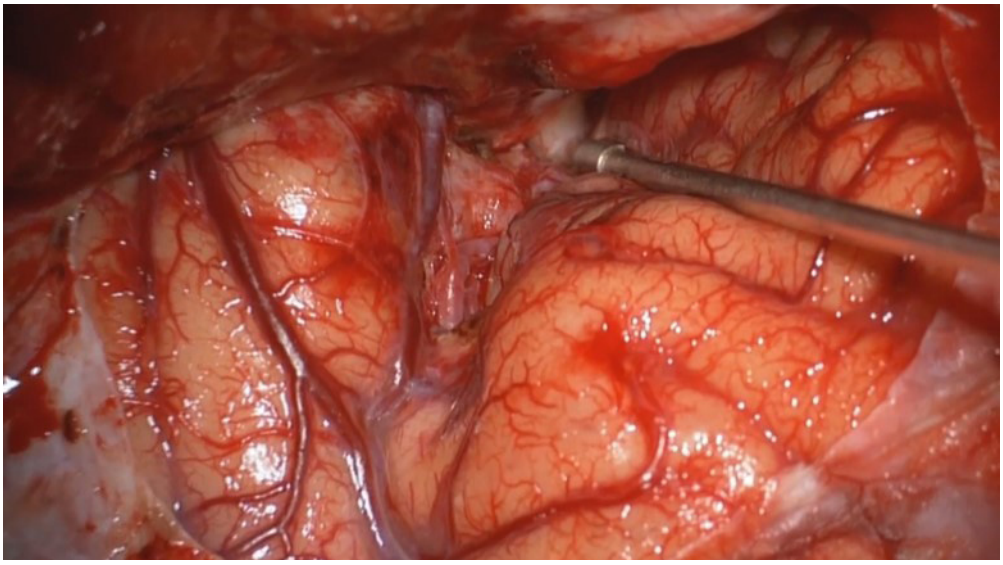

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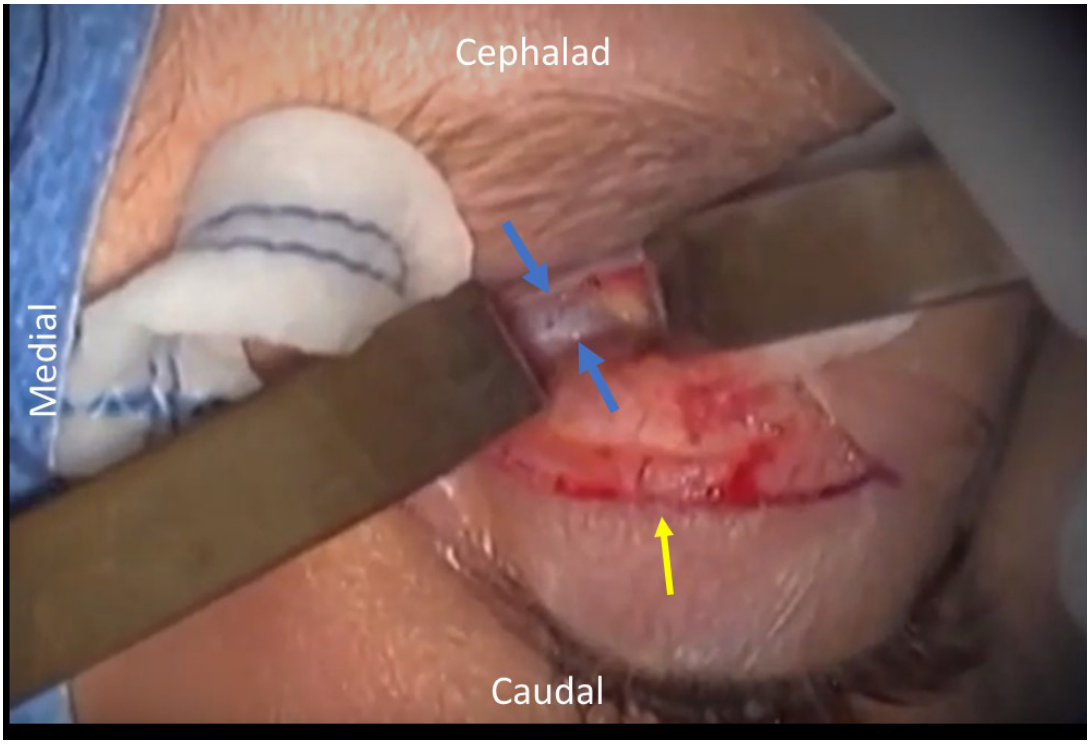

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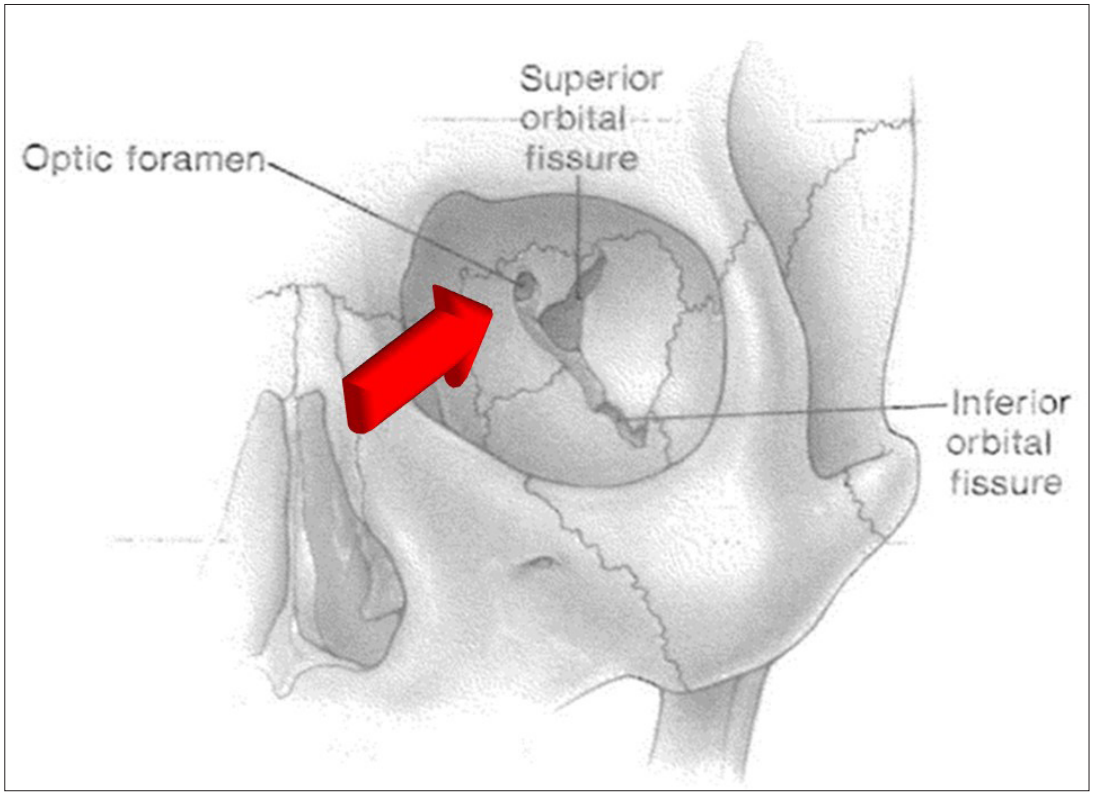

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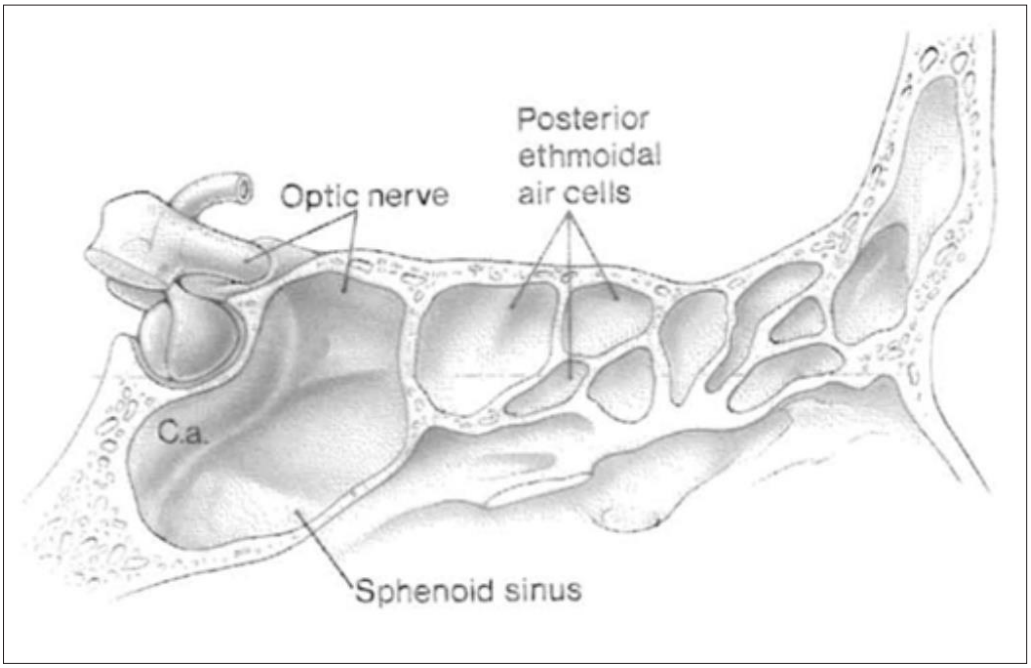

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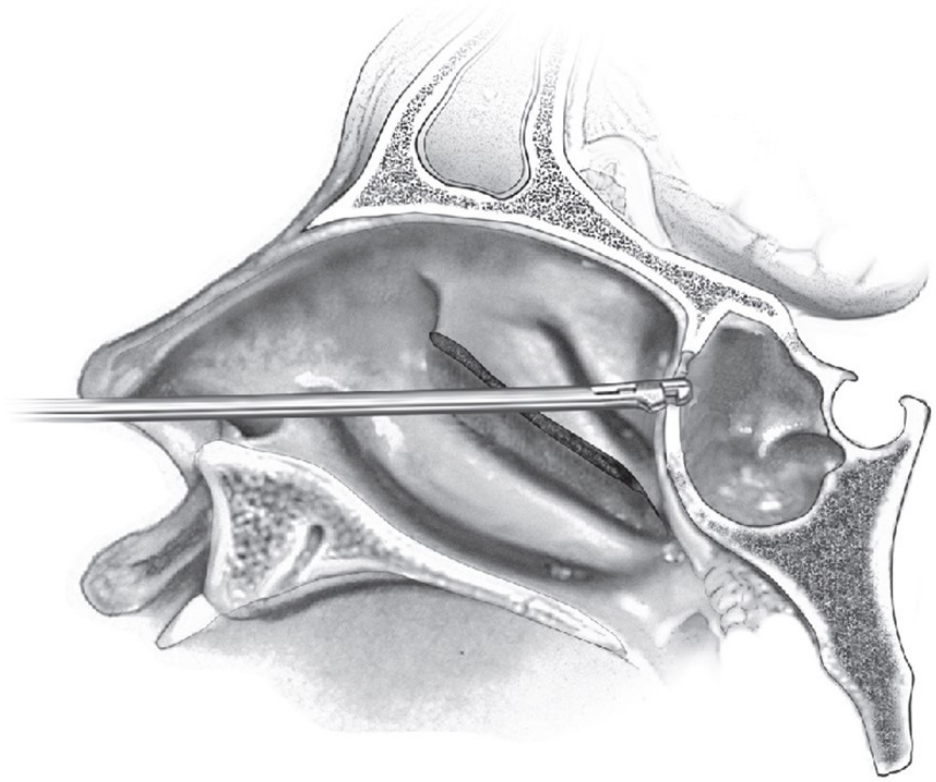


Figure 7

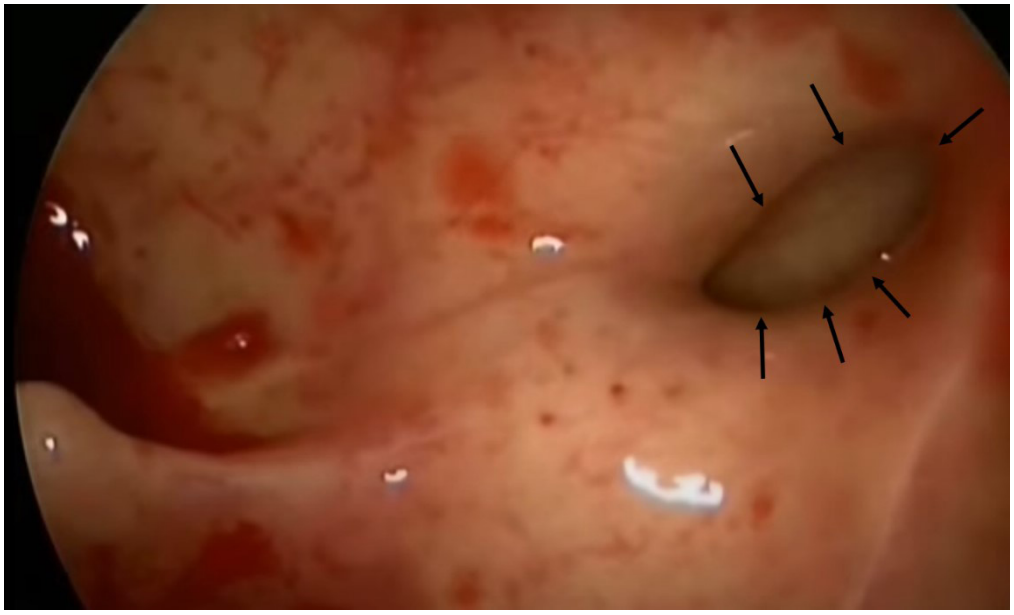
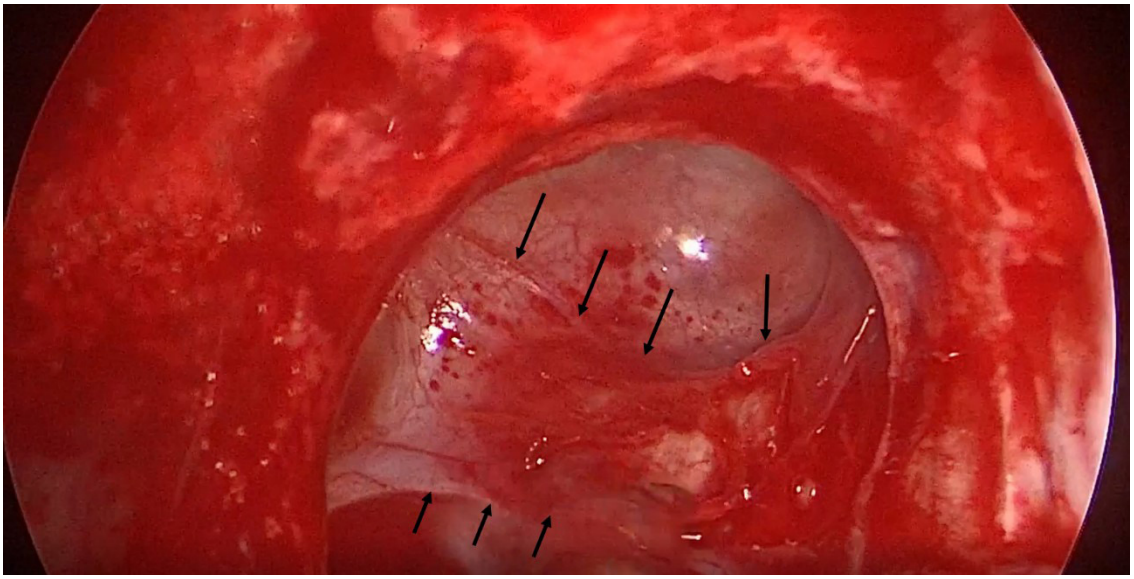


Figure 8



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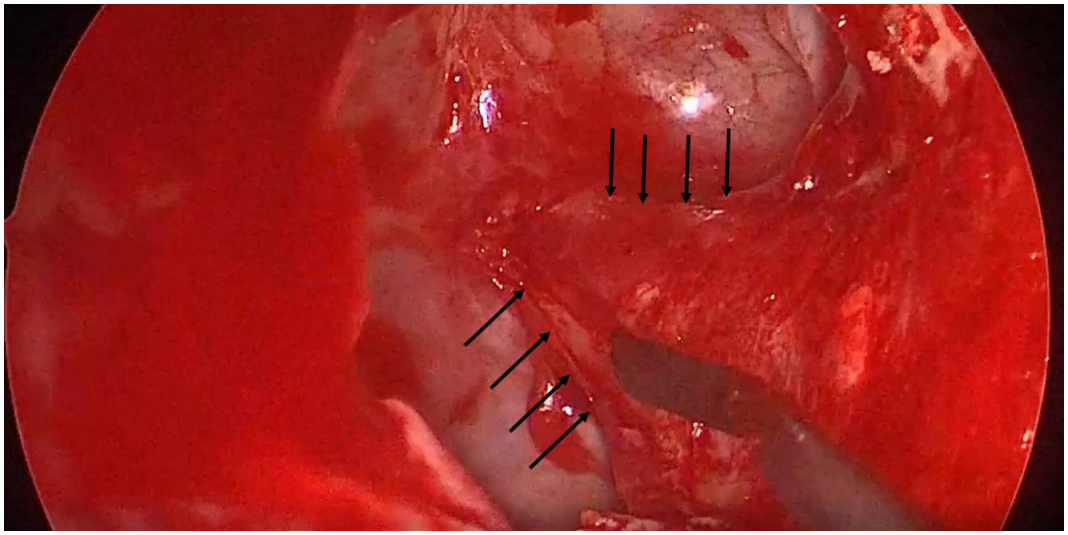

Figure 10

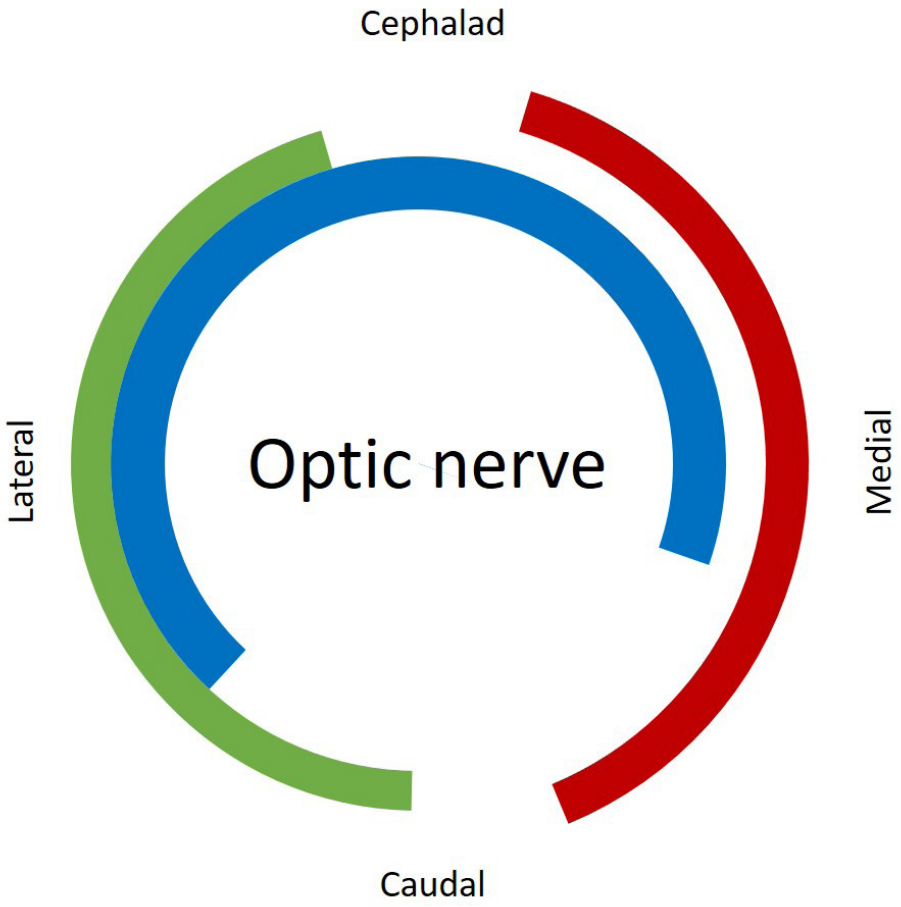

Figure 11