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# Post-Traumatic Visual Loss Treated with Endoscopic Orbital Decompression: Case Report

## Endoskopik Orbital Dekompresyon ile Tedavi Edilen Post-Travmatik Görme Kaybı: Olgu Sunumu

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## ABSTRACT

A case of visual loss after maxillofacial trauma is presented. Patient had midfacial fractures and delayed loss of visual acuity following the reduction of these fractures, four days after the initial trauma. Endoscopic orbital decompression was performed without optic nerve decompression. The patient had total return of visual acuity. We discussed the role of endoscopic orbital decompression in visual loss, the etiology of visual loss and treatment of it in maxillofacial trauma. We believe endoscopic orbital decompression should be considered for management of optic nerve involvement in medial orbital fractures. Patient selection and immediate intervention are considered the essentials of the treatment.

Keywords

*Maxillofacial injuries; visual acuity; endoscopy; orbital diseases; decompression*

## ÖZET

Maksillofasiyal travma sonrası gelişen görme kayıplı bir olgu sunulacaktır. Hastada midfasyal kırıklar ve bu kırıkların redüksiyonu ardından travma sonrası dördüncü günde gelişen geç görme kaybı vardır. Hastaya optik sinir dekompresyonu yapılmadan endoskopik orbital dekompresyon yapılmıştır. Hastanın görmesi tam olarak düzelmiştir. Bu olgu sunumunda görme kaybında endoskopik orbital dekompresyonun yeri, görme kaybının etyolojisi ve maksillofasiyal travmadaki tedavi yaklaşımı tartışılacaktır. Medial orbital kırık sonrası optik sinirin etkilendiği durumlarda tedavide endoskopik orbital dekompresyonun kullanılabileceğine inanıyoruz. Hasta seçimi ve erken müdahale tedavinin temelini oluşturmaktadır.

Anahtar S zc kler

*Maksillofasiyal yaralanmalar; görme keskinliği; endoskopi; orbita hastalıkları; dekompresyon*

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## INTRODUCTION

Endoscopic sinus surgery is widely used in management of numerous nasal and paranasal pathologies, including chronic sinusitis, nasal polyposis, tumors and dacryostenosis. Optic nerve decompression and orbital decompression for Graves' disease are also well established techniques using nasal endoscopy.[1] Even though 44% of facial fractures involve one or both orbits, blindness is relatively uncommon, accounting for only 2-5%.[2] We present a case with midfacial fractures and delayed loss of visual acuity after the reduction of these fractures. We will discuss the use of endoscopic orbital decompression in the management of this case.

## CASE REPORT

A 17 year old boy had maxillofacial trauma after a motor vehicle accident. Physical examination revealed periorbital edema and ecchymosis on the left side, laceration and crepitation on the nasal dorsum, left infraorbital rim fracture and maxillary bone displacement. Initial opthalmologic examination revealed no loss of visual acuity. Relative afferent pupillary defect was absent. Computed tomography (CT) scanning of the head and neck showed a Le Fort II fracture along with the left infraorbital rim fracture and medial orbital wall fracture. A bone fragment was displaced close to the medial rectus muscle impinged on the muscle (Figure 1). There were no signs of an optic involvement and eye movements were free in every direction so immidiate intervention was not considered at the time.

Four days after the accident the patient was taken to the operating room and a maxillomandibular fixation was performed through a maxillary vestibular incision. The left infraorbital rim fracture was reducted by screw and plate via subciliary incision. Endoscopic excision of the bone fragments inside the nasal cavity was performed continuously. On the second post operative day patient complaint about visual loss and diplopia in his left eye and examination revealed a 5/10 visual acuity. Relative afferent pupillary defect was present which also implicates optic nerve involvement. Patient was taken to the operating room and an endoscopic medial orbital decompression was performed. Lamina papyracea and the etmoid fragments displaced towards the orbita were removed. Orbital periosteum was not incised during this operation. Postoperatively, visual acuity had improved to 7/10 and there was no diplopia or any sign of medial rectus entrapment. CT scanning confirmed the decompression of the orbital cavity (Figure 2). A course of steroid (50 mg prednisolone once a day) and oral antibiotics (Amoxicillin and clavulanic acid 1 g oral every 12 hours) was started before surgery and continued for

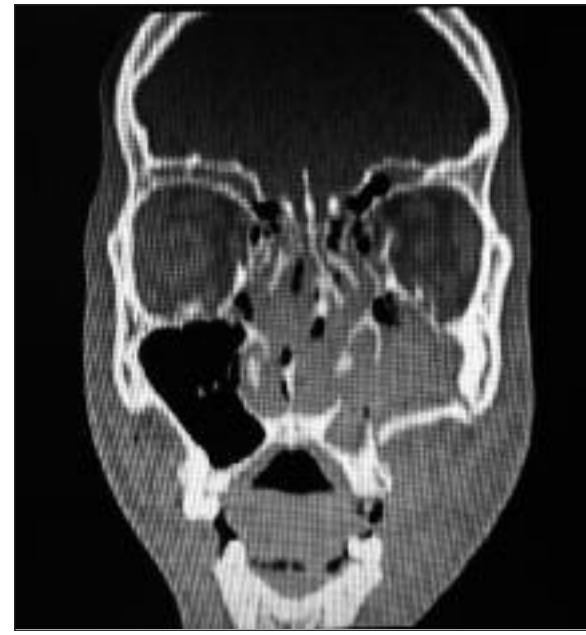

**Figure 1.** Preoperative axial computed tomography showing the impinged bone fragment to the left orbit and medial rectus muscle.

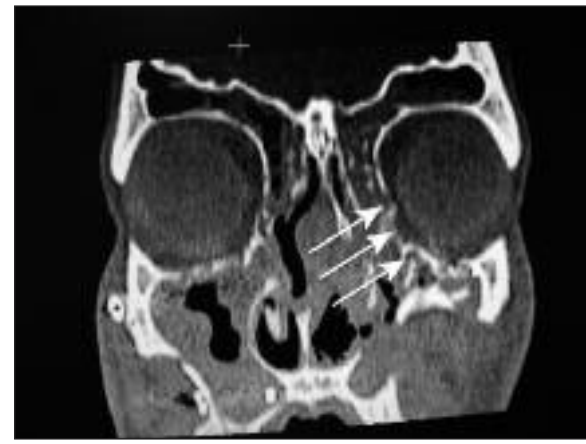

**Figure 2.** Postoperative axial computed tomography showing the decompressed orbita (Arrows point the decompression line).

two weeks. Patient was discharged uneventfully 5 days after the second operation. During the follow up period visual acuity had improved to 8/10 after the first month and 10/10 after the second month. An informed consent from the patient was taken prior to the interventions.

## DISCUSSION

Visual loss after maxillofacial trauma is relatively uncommon accounting for only 2-5% of cases. Visual loss can occur secondarily to optic nerve damage due to compression, stretching, contusion, section and ischemia or central retinal artery occlusion.[3] Majority of the optic nerve lesions are believed to be related to damage to the intracanalicular segment of the nerve, but displaced bone fragments anywhere in the orbit can damage the nerve as well.[4] In our case, an increase of the orbital pressure by a bone fragment was most likely the cause of the optic nerve damage and diplopia caused by fracture reduction. Because there were no signs of an optic involvement, immidiate intervention was not considered at the time. Orbital decompression at the initial presention was considered unnecessary and may have caused additional morbidity.

A number of treatment options are available for traumatic optic neuropathy. Regardless of the modality, immediate intervention is the most important aspect of treatment because it may be the deciding factor between reversible or irreversible visual loss.[4] Immediate vision loss after the trauma results in a poor outcome regardless of treatment.[5] In our case delayed loss of the visual acuity justified the treatment. Management includes conservative and surgical measures. Even with the presence of a medial orbital wall fracture along wtih other numerous maxillofacial fractures, a patient without diplopia, vision loss or herniation of orbital tissue into the etmoids can be managed by medical treatment and observation. However, immediate surgical intervention and optic nerve decompression is required if visual loss or the optic nerve is involved. Surgical approaches for optic nerve decompression include transfrontal craniotomy, orbitotomy, transethmoidal, transantral-ethmoidal and spheno-ethmoidal routes.[6] Endoscopic sinus surgery constitutes a direct, non- invasive tecnique that has many advantages over traditional approaches such as magnified and clearer view, less morbidity and shorter hospital stay.[7-9]

In our case we performed an endoscopic orbital decompression without decompressing the optic canal and were still able to preserve and improve visual acuity by relieving the pressure within the orbit. To our knowledge the present case is the first report of traumatic optic nerve damage managed by endoscopic orbital decompression without performing an optic nerve decompression. Some may argue that the first operation should have addressed the bone fragment. This is a reasonable argument; however, the surgeon should weigh the possible morbidity of medial orbital surgery in a patient with an unaffected optic nerve. We believe endoscopic orbital decompression should be considered for management of optic nerve involvement in medial orbital fractures. Patient selection and immediate intervention are considered the essentials of the treatment.

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