



Central retinal artery occlusion in the case of traumatic orbital cellulitis

Travmatik orbital selülitli olguda santral retinal arter oklüzyonu

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ABSTRACT

Vision can be lost after orbital infection as a result of optic nerve atrophy, central retinal artery occlusion (CRAO). We report such a case. We present a case report CRAO secondary to periorbital cellulitis. A 52-years-old male patient attended our clinic with left upper eyelid edema, hyperaemia, pain and visual loss. History revealed that he had an ocular surface trauma with cement to his left eye 7 days before. Fundus examination showed patchy filling of the branches of the central retinal artery, retinal opacification at the posterior pole and cherry red spot at the macula. His left visual acuity was declined to no perception of light. There was no clinical improvement after 7 days treatment. Orbital cellulitis did not respond to all treatment and magnetik resonance imaging demonstrated subperiosteal abscess at the posterior-superior orbital wall. We report a case of orbital cellulitis following an ocular surface trauma resulting in the rare complication of central retinal artery occlusion.

Keywords: Orbital cellulitis, central retinal artery occlusion, ocular surface trauma

ÖZ

Orbital travma ve selülit sonrası görme, optik atrofi ya da santral retinal arter tıkanıklığı (SRAT) gibi nedenlerle kaybedilebilir. Böyle bir olguyu sunmayı amaçladık. Travmatik orbital selülit sonrası SRAT nedeniyle görmesini kaybeden bir olgu sunuldu. Sol göz üst kapakta şişlik, ödem, ağrı ve görme kaybı şikayetleri olan 52 yaşındaki erkek hasta kliniğe başvurdu. Hastanın hikayesinde yedi gün önce sol gözüne çimento kaçma hikayesi mevcuttu. Fundus muayenesinde arterlerde dolmama, retinada beyazlaşma ve makulada kiraz kırmızısı görünüm bulguları vardı. Sol gözde görme ışık yokluğu düzeyinde tespit edildi. Tedaviden yedi gün sonra klinikte düzelme olmadı. Bütün tedavilere rağmen bulgularda düzelme olmadı ve üst arka orbital duvarda görüntüleme periorbital apse tespit edildi. Oküler yüzey travması sonrası orbital selülit gelişmesi ve nadir bir komplikasyonu olan SRAT nedeniyle görme yetisini kaybeden olgu sunuldu.

Anahtar Kelimeler: Orbital selülit, santral retinal arter tıkanıklığı, oküler yüzey travması

INTRODUCTION

Orbital cellulitis (OC) is a relatively uncommon infective process involving the tissues posterior to the orbital septum. In literature, the most common predisposing factor for orbital cellulitis has been found as sinus disease (1). In the pediatric group, 91% of patients may have radiologically confirmed sinus disease, the most common being ethmoid and maxillary sinuses⁽¹⁾. Ethmoidal sinusitis has been demonstrated

as the source of infection ranging from 43% to 75% of patients in various series (2). Additionally, orbital trauma, introduction of foreign bodies, and fractures leading to acute orbital infection have been described (3).

OC is a condition that rarely causes complete loss of vision if treated in a timely fashion (4). Vision can be lost after orbital infection as a result of optic nerve atrophy, central retinal artery occlusion (CRAO), exposure keratopathy, retinal detachment, retinal haemorrhages, and

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exudates. We report a case of orbital cellulitis developed after ocular surface trauma and resulting in a CRAO.

CASE REPORT

A 52-years-old male patient attended our clinic with left upper eyelid edema, hyperaemia, pain and visual loss. History revealed that he had an ocular surface trauma with cement to his left eye 7 days before. Upper eyelid edema, redness and visual loss had begun two days after and oral ciprofloxacin (1000 mg/day in two doses) and topical moxifloxacin (5 times a day) had been prescribed by an ophthalmologist and administered accordingly. In the following five days restriction of upgaze in the left eye began, inflammation of the eyelids worsened and the patient was referred to our clinic. His right visual acuity was 20/20 and left visual acuity was light perception. Examination of the left eye revealed extensive periorbital edema, redness, proptosis and ophthalmoplegia (Figure 1A,B). There was no pathological finding in right eye. Intraocular pressure (IOP) was 16 mmHg in right eye and 18 mmHg in left eye. Fundus examination showed patchy filling of the branches of the central retinal artery, retinal opacification at the posterior pole and cherry red spot at the macula. Fundus fluorescein angiography represented the findings of CRAO (Figure 1C,D). Computerized tomography demonstrated soft tissue density infiltrate extending posteriorly around the left globe, minimal ethmoidal sinus infiltrate with no subperiosteal abscess and no intracranial involvement (Figure 1E). Intravenous mannitol, ocular massage and antiglaucomatous treatment for CRAO were unsuccessful at restoring retinal perfusion. His left visual acuity was declined to no perception of light.

The patient was hospitalised and intravenous treatment with piperacillin/tazobactam 13.5 g/day in three doses and topical moxifloxacin 6 times a day was begun and administered for 7 days. There was no clinical improvement after 7 days. The treatment was shifted to intravenous ceftazidime 3 g/day in three doses and intravenous daptomycin 350 mg/day by the suggestion of the infectious disease consultant. Orbital cellulitis did not respond to treatment (Figure 2A,B) and magnetik resonance imaging demonstrated subperiosteal abscess at the posterior-superior orbital wall. Drainage of the abscess was planned but the patient did not accept the treatment.

DISCUSSION

The most common predisposing factor for OC is sinus disease, particularly in the younger age groups (1). The infection was reported to originate from sinuses, eyelids, face, dental abscess, retained foreign bodies or distant sources by hematogenous spread (1,5). There are some cases of orbital cellulitis reported which were developed after trauma. Goldfarb et al. described three cases of OC following fractures of the orbital walls (6). The time between injury and development of cellulitis varied from 5 days to 6 weeks (6). Jayamane et al. reported a case of OC 22 years after facial trauma, which created continuity between the right orbit and adjacent ethmoid cells (7). We reported here an orbital cellulitis case that developed after ocular surface trauma and resulting in a central retinal artery occlusion. Orbital cellulitis developed one week after trauma. To our knowledge, this is the first report of orbital cellulitis following ocular surface trauma.

Up to 38% of children may have multiple sinus involvement and in adult patients, up to 50% may have underlying sinus disease, while up to 11% may have multiple sinus involvement (1). In our case also had minimal ethmoidal sinus infiltrate. Computerized tomography of the patient did not show any fracture on the orbital walls.

Although uncommon the disease processes may lead to serious complications, including vision loss and a predisposition for life-threatening events such as cavernous sinus thrombophlebitis or other intracranial complications. The mechanism for loss of vision with orbital inflammation may involve; optic neuritis as a reaction to adjacent or nearby infection, ischemia resulting from thrombophlebitis along the valveless orbital veins or compressive/pressure ischemia possibly resulting in CRAO. In literature CRAO cases are reported in patients with orbital cellulitis (8,9). It is an unusual but known complication of OC (10). It has been demonstrated that following orbital inflammation, occlusion may occur at the level of the central retinal artery or occasionally at the ophthalmic artery (10). It is not so difficult to explain the mechanism of CRAO in severe traumatic cases. In our case there is orbital cellulitis following ocular surface trauma and CRAO can be explained by inflammation.

In conclusion; we report a case of orbital cellulitis following an ocular surface trauma resulting in the rare complication of central retinal artery occlusion.

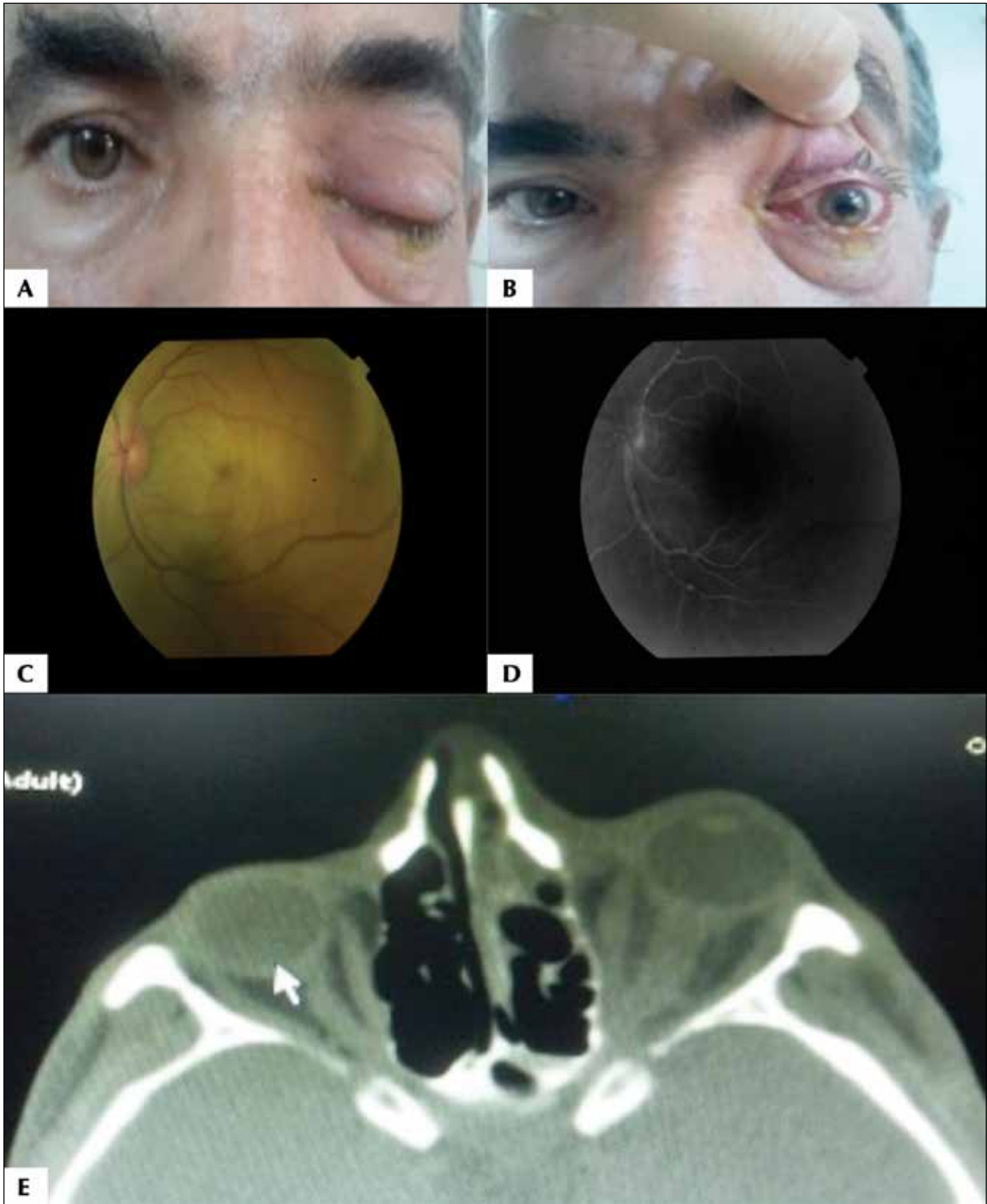


Figure 1. Upper row (left eye): (A,B) Extensive periorbital edema, redness, proptosis and ophthalmoplegia. Middle row (left eye): (C,D) Fundus fluorescein angiography represented the findings of CRAO. Lower row (left eye): (E) Computerized tomography demonstrated soft tissue density infiltrate extending posteriorly around the left globe. CRAO: Central retinal artery occlusion.



Figure 2. (A,B) Unresponsive to treatment.

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