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A case of subperiosteal abscess of the orbit with central retinal artery occlusion

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PURPOSE. *To report a rare case of orbital subperiosteal abscess with central retinal artery occlusion that resulted in blindness.*

METHODS. *A 60-year-old previously healthy woman was referred to the hospital with 4-day history of orbital cellulitis, progressive left eye pain, eyelid swelling, and 1-day history of visual impairment. The patient was diagnosed with subperiosteal abscess of the orbit on diagnostic imaging.*

RESULTS. *An emergent surgical intervention was performed due to prominent visual impairment. One day after surgery, ophthalmoscopic examination revealed cherry-red spot of the left fundus and central retinal artery occlusion. The final vision of the left eye was no light perception.*

CONCLUSIONS. *Central retinal artery occlusion, which is a therapeutic emergency, can be associated with acute progression of space-occupying lesion in the orbit such as subperiosteal abscess. (Eur J Ophthalmol 2009; 19: 288-91)*

KEY WORDS. *Blindness, Central retinal artery occlusion, Orbital cellulites, Subperiosteal abscess*

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INTRODUCTION

Orbital cellulitis is not uncommon in ophthalmic daily practice, but reports of severe cases have been decreasing recently because of widespread use of effective antibiotic therapy (1). Subperiosteal abscess (SPA) of the orbit, a specific entity of the orbital cellulitis, however, often resists antibiotic therapy and requires surgical intervention (2). The complicated, intractable orbital SPA can lead to serious visual loss and impairment of ocular motility. Such complications of orbital SPA can be caused by 1) disturbance of the retinal circulation due to acute elevation of the intraocular pressure (IOP); 2) direct compressive optic neuropathy; 3) ischemic neuropathy from compression of small nutrient vessels supplying the optic nerve; 4) traction on the optic nerve and tenting of the posterior globe (3). Although acute progression of space-occupying lesion in the orbit can theoretically disturb ophthalmic artery circulation, the association of central retinal artery with SPA has never been reported. Central retinal artery occlusion (CRAO) represents one of the true therapeutic emergencies in ophthalmology, as time is of the essence if vision is to be successfully restored. Herein, we describe a rare case of orbital SPA with CRAO that resulted in blindness.

METHODS

A 60-year-old Japanese woman experienced mild headache and swelling of the left eyelid on awakening. She had no history of systemic disease. The next day, she consulted with her local ophthalmologist because the symptoms and signs had gradually worsened. With a diagnosis of orbital cellulitis, eyedrops of 4% levofloxacin (6 times a day) and intravenous administration of tobramycin (120 mg/day) were started. These medications for 2 days were found to be ineffective, and the systemic treatment regimen was replaced by intravenous administration of imipenem/systharacine (2 g/day). These treatments continued over a couple of days but proved ineffective for resolution of the symptoms and signs. The patient was referred to our institution 4 days after initiation of the symp-

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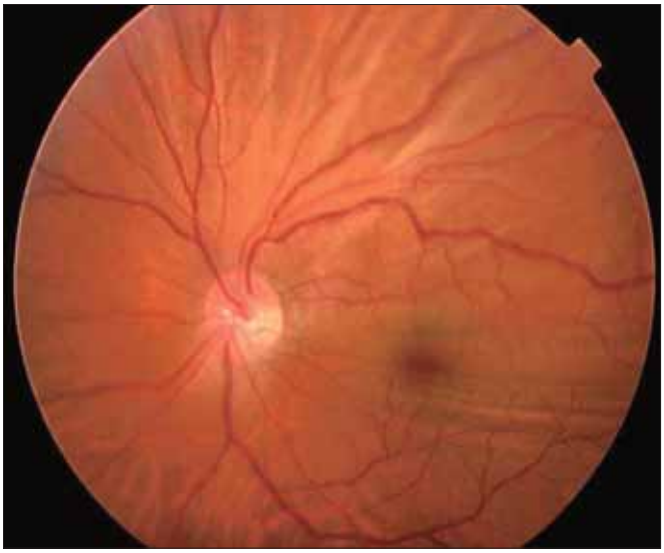

Fig. 1 - *Fundus photograph at presentation, illustrating retina folds.*

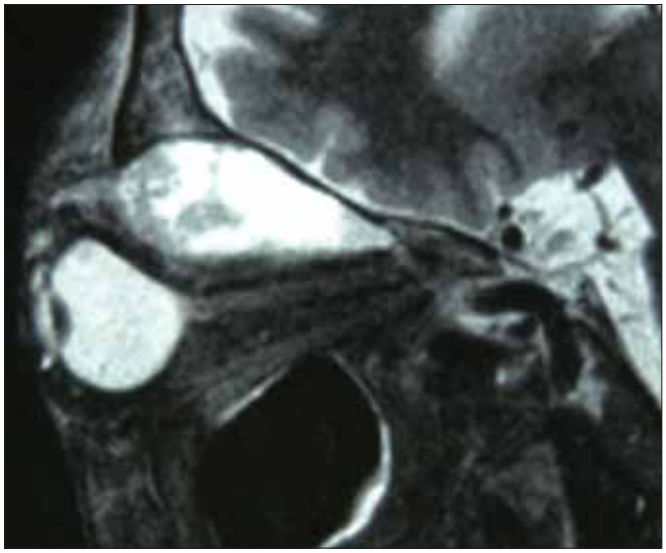

Fig. 2 - *Sagittal scan MRI illustrating a well-defined mass lesion compressing the eyeball superiorly and stretching of the optic nerve.*

toms, and she was hospitalized because of a prominent visual impairment and severe orbital inflammation.

Best-corrected visual acuity of the left eye was light perception. The left pupil was nonreactive. The right pupil reacted briskly and showed a reverse afferent papillary defect. The left eyelid was extremely swollen. The left eyeball was remarkably displaced inferotemporally and presented exophthalmia of about 6 mm over the right eyeball as measured by Hertel exophthalmometer. Ocular motilities were strongly limited in all directions. Massive subconjunctival hemorrhages was also noted. Ocular pressure could not be measured due to the marked eyelid swelling. No abnormality was found in the cornea, anterior segment, or crystalline lens. Ophthalmoscopy disclosed retinal folds in the upper region from the optic disc (Fig. 1). The optic disc had normal color. B-scan ultrasonography revealed a space-occupying mass lesion in the retrobulbar area that appeared to strongly compress the posterior pole of the eyeball. The right eye was normal in all aspects. Physical examinations revealed no abnormalities. Laboratory studies were unremarkable for peripheral blood leukocyte, C-reactive protein, and erythrocyte sedimentation rate.

Orbital diagnostic imaging modalities with computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated an extension of inflammatory mass in the left orbit, frontal and ethmoidal sinusitis accompanying destruction of the orbital bones, capsular formation of the tumor, and inflammation in the subperiosteal region. In addition, the left eyeball was deformed and considerably displaced inferotemporally with elongation of the optic nerve, due to compression by the orbital mass lesion (Fig. 2).

RESULTS

The clinical and diagnostic imaging examination findings led to a diagnosis of paranasal sinusitis-induced subperiosteal abscess. Due to the presence of severe visual impairment, an emergent surgical intervention was performed for endoscopic paranasal sinus incision and orbital abscess drainage. It was remarkable intraoperatively that dark-red pus-like fluids including clot were drained on penetration of the periosteum of the involved orbital bone. We completed the surgery after confirming the release of the eyeball from compression along with resultant shrinkage of the orbital mass lesion using B-scan ultrasonography. Drainage cultures were negative for bacteria probably because of the usage of antibiotics before surgery. After the patient came out from under the general anesthesia, several hours after the surgical intervention, the left eyelid swelling decreased considerably, but the left eye had no visual perception. Ophthalmoscopic examination disclosed typical features of acute CRAO, including cherry-red spot of the macular area. Fluorescein angiographic examination showed normal arm-retina circulation time and reperfusion of the central retinal artery, which indicated that the retinal

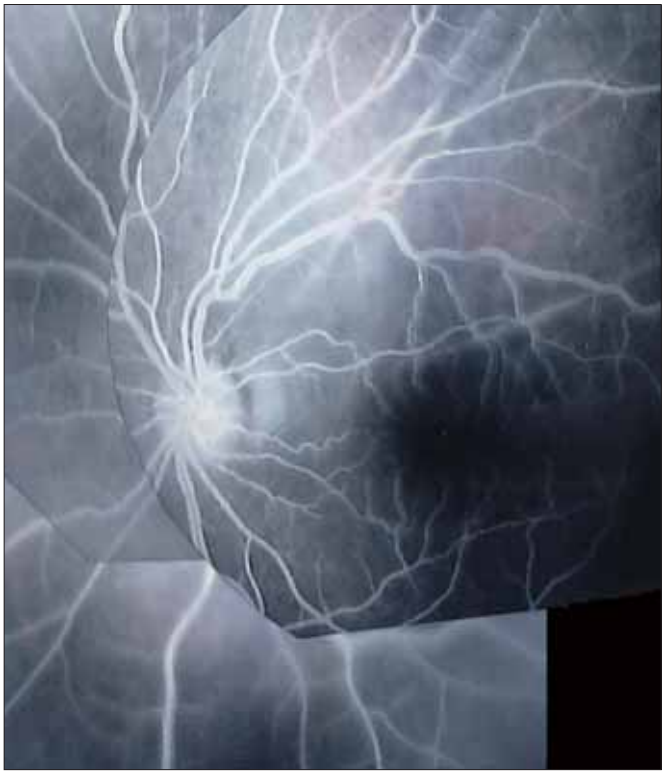

Fig. 3 - *Fluorescein angiography after surgery, which shows occurrence of central retinal artery occlusion.*

circulation had already recovered on the day after surgery (Fig. 3). Intravenous prostaglandin (PGE1) and oral carbonic anhydrase were administered immediately, but anticoagulant therapy was not performed because it was in the early stage after the surgical intervention.

DISCUSSION

Our case resulted in blindness due to acute CRAO associated with orbital SPA of paranasal sinusitis origin. It seems that CRAO was caused by the acute progression of SPA in the orbit. We do not know exactly when CRAO developed. It probably developed in a few hours between surgery and the first examination at our institute. We should have done the fundus examination at the time of the surgery end. Due to the lack of anticipation, we could not make the diagnosis of CRAO until after surgery. Although earlier diagnosis of CRAO may not have changed the visual prognosis in our case, there may be cases in which prompt diagnosis of such association would result in better therapeutic planning and outcome.

The causative diseases of orbital SPA include paranasal sinusitis, trauma, and dental diseases, but considerable cases are reported to be secondary to paranasal sinusitis (4). The bony orbit is a quadrilateral pyramidal cavity that is formed by seven separate bones. Only the thin paper plate of the ethmoidal sinus separates the contents of the bony orbit from the ethmoid cells. The superior ophthalmic vein is continuous at the inner angle of the orbit with the nasofrontal vein, communicates in turn with the angular vein of the face, and is completely devoid of valve with a free flow of blood between the ethmoidal vein and ophthalmic vein. The inferior orbital fissure is adjacent to the ethmoid, through the medial part of which the superior ophthalmic vein ends in the cavernous sinus. Thus, the anatomically intimate relationship between the orbit and the paranasal sinuses is relevant to factors leading to extension of the paranasal sinusitis to the orbital cavity (5).

Imaging of orbital SPA shows characteristic findings; contact with bony wall and capsule made of periosteum, which provides useful information for differential diagnoses from orbital inflammatory pseudotumor, orbital malignant lymphoma, lacrimal gland inflammation, posterior scleritis, carotid cavernous fistula, and dysthyroid orbitopathy.

Conservative management of orbital SPA is often ineffective. According to the studies of orbital inflammation to date, surgical intervention is indicated when antibiotic therapy is ineffective within 48–72 hours after its initiation or when ocular disorders such as impairment of visual acuity, relative afferent pupillary defect, limitation of ocular motility, and dislocation of the eyeball are confirmed (6). Severe visual loss as a complication of orbital space-occupying lesions such as SPA may be caused by 1) disturbance of the retinal circulation due to acute elevation of the IOP; 2) a direct compressive optic neuropathy; 3) ischemic neuropathy from compression of small nutrient vessels supplying the optic nerve; 4) traction on the optic nerve and tenting of the posterior globe (3). It is most probable that the blindness in the patient described herein was due to CRAO. The underlying cause is likely to be a marked compression of the eyeball by subperiosteal abscess, which resulted in rapid elevation of the IOP, hence leading to retinal circulatory disturbance. Since the drainage of the SPA was contaminated by blood, rapid elevation of IOP can be considered due to destruction of the anterior or posterior ethmoidal artery.

In this case, the accurate onset timing of orbital SPA was unknown. Despite the conservative management with antibiotics therapy for 4 days, ocular disorders such as visu-

al loss and relative afferent pupillary defect deteriorated, which eventually necessitated surgical intervention. The delay of surgical intervention for 4 days may be responsible for the poor visual outcome in our case. When initial antibiotic medications fail, earlier surgical intervention based on prompt diagnosis of orbital SPA using imaging modalities such as CT should be considered.

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