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Case Report Open Access

Sudden Unilateral Visual Loss and Ptosis after Autologous Fat Injection and Simultaneous Bont/A Treatment

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Abstract

Central retinal artery occlusion (CRAO) following cosmetic surgery seems to be rare and disastrous disease inducing sudden visual loss. Even if tough treatment is prescribed initially, the prognosis of visual recovery is more regretable. In this paper, we report one case of CRAO combined with ptosis resulting from simultaneous autologous fat injection and BoNT/A treatment in the glabellar area for cosmetic problems. We confirmed CRAO by fluorescein angiography. There are many possible causes of visual loss following cosmetic surgery; however, the commonest form is autologous fat injection up to now. To our knowledge, there have been little reports of CRAO combined with ptosis in autologous fat injection procedures. These facts must be kept in mind by all cosmetic plastic surgeons and ophthalmologists of the importance of careful measures and immediate treatment of iatrogenically induced ocular complications.

Introduction

Blindness following cosmetic surgery is rare. In accordance with DeMere's survey, the occurrence was 0.04% or 1 case per 2500 operations [1]. Blindness can be in two form; partial and permanent. Partial blindness has been described following injection of collagen [2] and Cymetra [3].

Permanent unilateral blindness was reported secondary to reduction of fractured zygoma, [4] the repair of orbital floor fractures, [5] retinal detachment procedures, [6] neurosurgical procedures [7], particulate substance injection (steroid injections) in different area such as nose, [8-12] Retrobulbar region, [13] scalp, [14] and eyelid [15] has been reported.

Blindness from a fat injection firstly was reported in 1988 [16]. The basic presentation was violent pain accompanied by immediate and permanent loss of vision in one eye. In the coming years, ophthalmology and neurology literature reported permanent unilateral blindness from central retinal artery occlusion by injectable fillers. Although some instances were reported with injection of fat into the nasolabial folds [17] or even the lower lip [18], most of central retinal artery occlusion and blindness resulted from injections in the nose or periorbital region [16,18,19]. In this report, we present a rare case of concurrent CRAO and ptosis.

Case Report

A 63-year-old Iranian female presented to the eye clinic in an irritated state with sudden vision loss in the right eye. His preoperative examination included a visual acuity of 20/20 in both eyes and was otherwise completely normal except for wrinkle in the forehead and canthal area. He had history of hypertension and was receiving Losartan tablet regularly. Eight hours earlier, she had undergone a fat transplantation of abdominal fat to her autologous fat injection in the glabellar area to correct a cosmetic problem. The procedure was performed by a general practitioner trained plastic surgury. Immediately after injection of autologous fat into her glabellar area under local anaesthesia via a sharp needle, she complained of unilateral blindness and became very irritable. 0.5 ml fat was injected into each lateral canthal area and 1 ml into a wrinkle in the forehead following botax injection over the periorbital area. She was transferred to an eye center (8 hours later) where the diagnosis of central retinal artery occlusion was established. Ophthalmologic exam showed best corrected visual acuity of no light perception in the right eye and 20/20 in the left eye. Pupil exam showed sluggish right pupil with relative afferent pupillary defect and a reactive pupil. Slit lamp exam showed normal anterior segments with open angles bilaterally. Fundus examination of the right eye revealed retinal opacity at posterior pole, pallor Optic disc and typical appearance of CRAO with a cherry red spot on the macula, and marked retinal ischaemia (Figure 1). Fundus examination of the left eye revealed cup to disk ratio of 0.4, otherwise normal optic

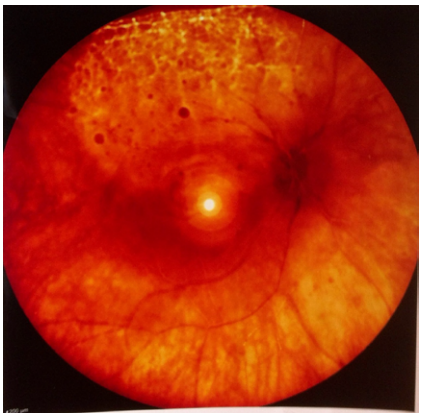

Figure 1: The Color fundus photograph of the right eye 10 hours after autologous fat injection showing segmented retinal arteries and diffuse edematous retina and cherry red spot.

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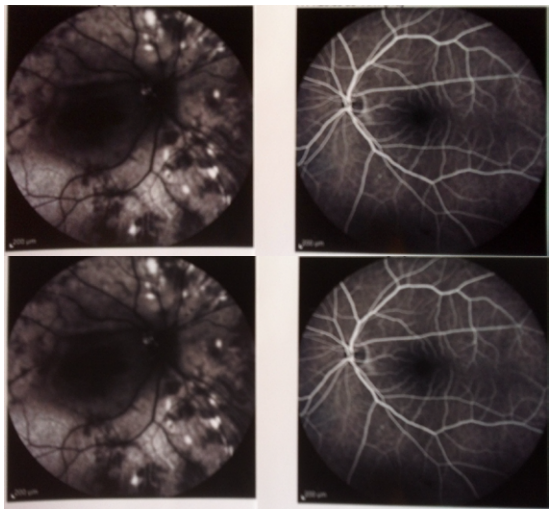

2a 2b

Figure 2: Flourescein angiogram of the right eye showing extensive areas of capillary non-perfusion (2a), versus normal angiogram of (2b).

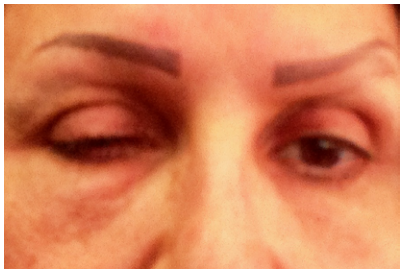

Figure 3a: Eight hours following an autologous fat injection in the glabellar area. There has been a right CRAO combined with ptosis and periorbital oedema. Vision is no light perception with the right eye and 20/20 with the left.

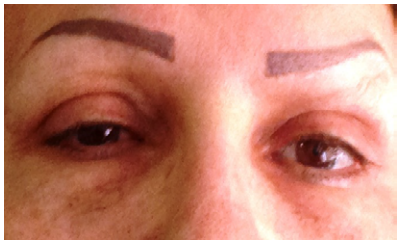

Figure 3b: Three months after the central retinal artery occlusion. A satisfactory cosmetic result has been achieved but vision is the same as before in right eye.

disc and flat macula. Flourescein Angiogram of the right eye showed extensive areas of capillary non-perfusion (Figure 2a and 2b). Patient was diagnosed with unilateral ischemic central retinal artery occlusion in the right eye. Laboratory tests including complete hypercoagulability and thrombotic work up were completed that were found to be normal. Electrocardiogram showed sinus rhythm. Immediate therapeutic measures were instituted. Initially intermittent digital massage was applied to the right eye. An intravenous 500 mg of acetazolamide was started rapidly followed by 25 g of mannitol over 30 minutes. Additionally, for the next 24 hours, 250 mg of acetazolamide was given intravenously every 6 hours. Carbogen was administered with continuous monitoring of the patient's pulse, respirations, and blood pressure for 10 minutes every hour for 12 hours. After these measures, the intraocular pressure was measured using goldmann tonometer that was within the normal range. Her right visual acuity was lost. Two days later, a superficial skin eruption of the forehead and ptosis developed respectively (Figure 3a). Her ocular condition was reexamined several times later and at 3rd month revealed no light perception in her right eye with optic atrophy and thick fibrous membrane on the posterior pole, though, ptosis gradually relieved (Figure 3b).

Discussion

Various mechanisms have been proposed as the cause of visual loss following cosmetic surgery. The usual mechanism of visual loss is increased intraorbital pressure producing a central retinal artery and/or vein occlusion, or causing direct pressure on the optic nerve with subsecutive occlusion of its vascular supply.

Injectable fillers generally regarded as being safe with occasional aesthetic complications, however, skin necrosis, cerebrovascular strokes and blindness can occur second art to injection of a soft tissue filler. Iatrogenic CRAO were reported previously in several articles [10,13,16-27]. Anyway, how the iatrogenically injected materials emerged in the retinal circulation is questionable yet.

Anatomically, many superficial arteries of the face are distal branches of the ophthalmic artery; supratrochlear, supraorbital, dorsal nasal and angular artery of the nose. According to the study of Danesh-Mayer et al. [23] a needle or cannula used to inject a soft tissue filler can accidentally perforate the wall of one of the distal branches and enter the artery's lumen. Then, the soft tissue filler can embolize into any area supplied by the internal carotid artery. In addition, retrograde flow of an intra-arterial injection into the central retinal artery through directly injection of material into a branch of the ophthalmic artery and vascular disturbances and even strong enough injection force to reach into the internal carotid artery were explained previously by some authors [17-19,22]. We assume similar mechanism for CRAO development, however, unlike the some other cases, it was accompanied by ptosis due to the botox injection over periorbital area.

In this case, in one side autologous fat was transfered into glabellar area under local anaesthesia via a sharp needle. Coleman SR [28,29] believe that Sharp cannulas, small cannulas, and needles are much more likely to perforate the wall of an artery and cannulate the artery lumen and for the placement of fillers at the injection site, consideration should be given to the use of larger, blunt cannulas and epinephrine. In other word, using blunt cannulas and low pressure allow for more precise placement of fat pearls at various levels including intramuscular levels that minimizes complications in these areas [28-31].

Meanwhile, according to Matarasso et al. 2003 [32], 0.5 cm below the lateral brow on each side corresponds with the underlying lateral and superior fibers of the orbicularis oculi muscles, which act as brow depressors. Therefore, injections superior to this point inactivate the inferior fibers of the frontalis muscle, yielding brow ptosis and injections inferior to this point risk inactivation of the levator palpebrae muscle, causing eyelid ptosis.

Additionally, several reports [33-34] shown that functional occlusion of a branch of the ophthalmic artery which supplies the extraocular muscles gives rise to ischemic changes in the extraocular muscles; levator palpebrae muscle or its innervations, yielding to ptosis.

So, considering the severe ptosis in our study case; a margin reflex distance value of zero with the upper lid covering the pupillary axis (Figure 3a), to some extend might be as a result of synergestic effect of incorrect injection of concurrent BoNT-A and filler.

Subsequently, the patient was subjected to some treatment techniques including ocular massage, carbon dioxide, oxygen therapy and intravenous injection intermittently and properely during treatment period but the thrombolytic agent was not given.

According to Kwaan, [35], no consensus currently exists regarding therapy. Moreover, Hueston et al. believe that total vascular insufficiency for 60-120 minutes produces permanent loss of vision [36-38]. But a lesser duration of occlusion leads to variable visual recovery; from 20/20 to no light perception. Another theory believes that emboli resulting from lipid, cholesterol, and calcific emboli cannot be expected to respond to thrombolytic therapy [39]. Anyhow, the clinician should be familiar with these signs and symptoms so that he may act quickly when evidence of decreased retinal perfusion occurs. If the surgeon caring for the patient is not an ophthalmologist, immediate assistance from a qualified ophthalmologist should be obtained while emergency measures are being instituted.

In short, our case illustrates a presentation of unilateral ischemic central retinal artery occlusion, where autologous fat injection thought to be the main risk factor. Our management focused on controlling blood pressure as preventive measures to protect the fellow eye while many interventions for fixed visual loss associated with CRAO have largely not proven to be of benefit and this strange case should be a warning to all ophthalmologists and plastic surgeons that widely performed simple procedures can cause irreversible misfortune, and the risk of damage should be explained to the patient. If there is any evidence of a visual problem, prompt consultation with an ophthalmologist is needed.

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