Case Report

Ophthalmic Artery Occlusion Following Facial Sclerosing Therapy

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Abstract

Purpose: To describe a case of ophthalmic artery occlusion and complete ophthalmoplegia after intralesional injection of a sclerosing agent into a subcutaneous hemangioma on the forehead.
Case Report: A 16-year-old girl underwent direct injection of 3 mL of sodium tetradecyl sulfate (Fibrovein) emulsion 1% (10 mg/mL) with a 23-gauge needle into a subcutaneous hemangioma on the forehead. Immediately after the injection, she developed sudden loss of vision and lid swelling of the left eye. Her visual acuity in the left eye became no light perception. Her left eye also developed a dilated pupil, ptosis, and complete external ophthalmoplegia. Funduscopy of the left eye revealed signs of central retinal artery occlusion. Magnetic resonance imaging of the orbit showed thickening of the medial and lateral rectus muscles of the left eye. Magnetic resonance venography of the brain was normal with no evidence of cavernous venous thrombosis. After 3 months, her ptosis and ophthalmoplegia resolved but her visual acuity remained no light perception.
Conclusion: Persistent total visual loss should be kept in mind as a disastrous complication of sclerosing therapy in a patient with facial hemangioma.

Keywords: Central Retinal Artery Occlusion; Hemangioma; Ophthalmoplegia; Sclerosing Agent; Sclerotherapy

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INTRODUCTION

Ocular complications following non-ophthalmic procedures are rare but may be devastating. The incidence of perioperative visual loss after non-ophthalmic procedures is 0.013% for all surgeries.[1] Sudden visual loss may be caused by an embolic event that occludes the arterial lumens around the visual system and gives rise to ischemic optic neuropathy, central retinal artery occlusion, or cortical ischemia.[2]

Sclerotherapy involves injection of a sclerosant into vascular lesions to induce localized thrombosis, fibrosis, and obliteration of the vessels. Occlusion of the ophthalmic artery and ophthalmoplegia following sclerotherapy is very rare.[3]

In 1981, Chamot et al reported total blindness and phthisis bulbi after sclerotherapy for frontal cutaneous hemangioma in a 4-month-old baby.[4]

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In 2009, *Matsuo* et al described visual loss caused by occlusion of the central retinal and posterior ciliary arteries following intralesional injection of a sclerosing agent into a glabellar hemangioma in an 18-year-old male patient.[5]

CASE REPORT

A 16-year-old girl developed sudden diminution of vision in the left eye immediately after intralesional injection of a sclerosing agent into an extensive hemangioma of the forehead. The diagnosis of subcutaneous hemangioma was made by a vascular surgeon. She had undergone direct intralesional injection of 3 mL of sodium tetradecyl sulfate (Fibrovein; STD Pharmaceutical Products Ltd, UK) emulsion 1% (10 mg/mL) with a 23-gauge needle. Obscuration of vision progressed in the first hour after injection with total drooping of the left superior eyelid [Figure 1].

The patient denied any history of floaters, tinnitus, headache, nausea/vomiting, weakness of either side of the body, dysphagia, ataxia, loss of consciousness, arthralgia, fever, or photosensitivity.

On examination in the recovery room, the patient was conscious and oriented but agitated. Visual acuity in the right eye was normal but there was no light perception (NLP) in the left eye. There was a relative afferent pupillary defect on the left eye with a pupil that was dilated and poorly reactive to light. In addition, severe ptosis and eyelid swelling was evident on the left side. Extraocular movements were restricted in all directions in the left eye (indicating palsies of cranial nerves III, IV, and VI; Figure 2) and were normal in the right eye. Cranial nerves V and VII were intact. Intraocular pressure was within normal limits in both eyes.

Fundus examination revealed marked retinal pallor with a cherry-red spot on the left side, suggesting central retinal artery occlusion [Figure 3b]. The retina on the right side was normal [Figure 3a].

In view of these findings, the patient was diagnosed to have central retinal artery occlusion with total ophthalmoplegia in the left eye. Anterior chamber paracentesis was performed urgently in the left eye with a 27-gauge needle and oral acetazolamide was prescribed. She also received intravenous methylprednisolone 1000 mg/day for 3 days. A complete blood count, kidney and liver function tests, erythrocyte sedimentation rate, C-reactive protein, and serum electrolytes were unremarkable. Computed tomography (CT) and magnetic resonance imaging of the brain were normal. CT and magnetic resonance imaging of the left orbit showed thickening of the medial and lateral rectus muscles [Figure 4]. Magnetic resonance venography of the brain was also normal with no evidence of central venous thrombosis.

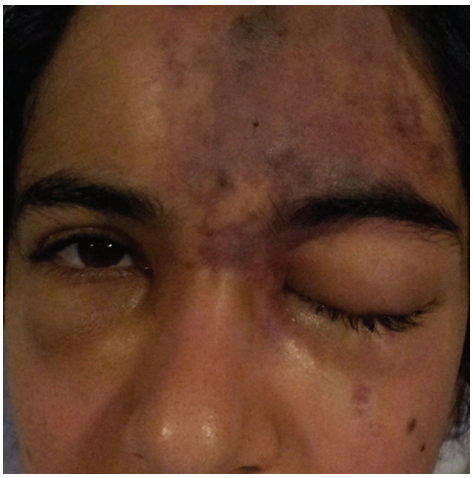

Figure 1. External photograph of the face showing ptosis and lid swelling in the left eye.

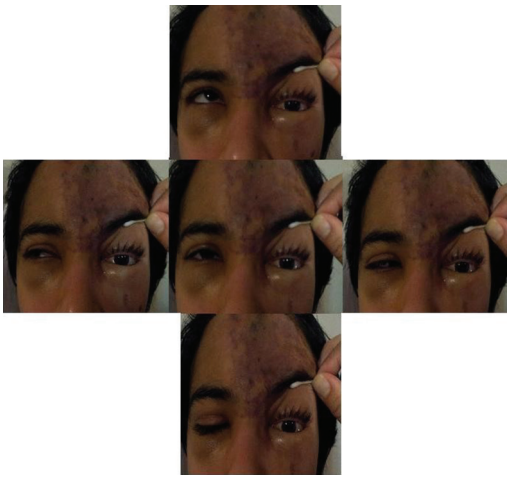

Figure 2. Extraocular movements in five cardinal positions of gaze showing complete ophthalmoplegia of the left globe.

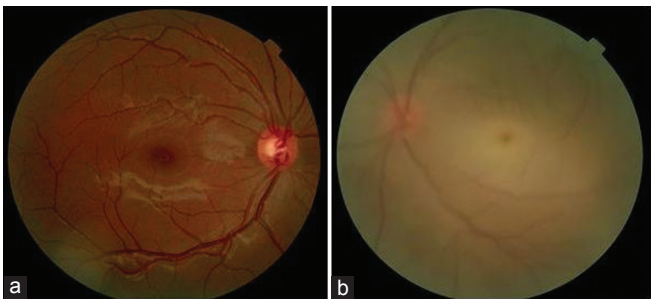

Figure 3. (a) Fundus photograph of the right eye is normal. (b) Fundus photograph of the left eye shows optic disc pallor and marked retinal pallor with a cherry-red spot suggestive of central retinal artery occlusion.

The patient's blepharoptosis and ophthalmoplegia resolved within 3 months, but her visual acuity remained NLP [Figure 5]. Fundus evaluation of the left eye revealed optic disc pallor with attenuation and sclerosis of the retinal vessels, presence of a fibrovascular tuft,

diffuse preretinal gliosis, chorioretinal atrophy, and diffuse disturbance of the retinal pigment epithelium [Figure 6a and b].

DISCUSSION

Thromboembolism, blurring of vision, allergic reaction, thrombophlebitis, skin necrosis, and hyperpigmentation have been reported as rare complications of sclerotherapy for periocular lesions.[6]

The main cause of perioperative visual loss following periocular injections is obstruction of the ophthalmic artery or retinal artery. Injected material, including the sclerosing agent, might travel from the vascular connections of the hemangioma to the orbit and to the central retinal artery and posterior ciliary arteries.

There are many reports of acute loss of vision after injection of cosmetic fillers for cosmetic purposes that explain the anatomy and mechanisms of embolization of the injected material.[7] When surgeons inject facial fillers into the glabella or forehead region, the injecting needle may accidentally damage the wall of a distal artery, such as the supraorbital or supratrochlear artery.

In this situation, the force of injection can overcome the systolic arterial pressure and push the tiny injected droplets proximally along the ophthalmic artery, and can even pass the origin of the central retinal artery and cause a cherry-red spot to appear initially. When the injection is completed and the force is relieved, the systolic pressure propels the injected droplets distally into the branches of the ophthalmic artery and the cherry-red spot may disappear.[7]

To reach the ophthalmic circulation, three contributing factors must occur at the same time, i.e. retrograde passage of the material, a high injection pressure, and a sufficient amount of material within the vessel lumen.[8] Once in the ophthalmic artery, the emboli may enter the posterior ciliary circulation rather than the central retinal artery and eventually occlude the vessels of the choroid.[9,10]

Obstruction of the ophthalmic artery is acute with simultaneous occlusion of both the retinal and choroidal circulations and has the following features:[11] severe visual loss with minimal or no light perception; marked ischemic retinal whitening of the macula; small-to-complete disappearance of the cherry-red spot; no recordable electroretinogram; and late disturbances of the retinal pigment epithelium. Prolonged choroidal filling in the absence of a cherry-red spot should raise suspicion of an obstruction of the ophthalmic artery.[11]

Our patient was NLP in the left eye and a cherry-red spot was apparent in fundoscopy on day 1. The patient refused to undergo electroretinography and fluorescein angiography.

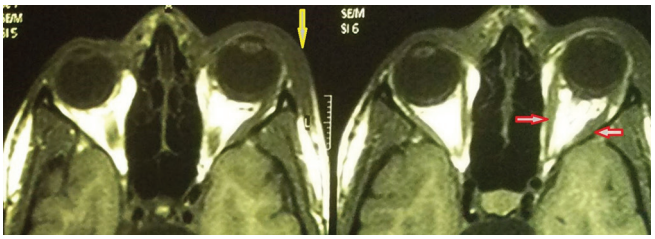

Figure 4. Magnetic resonance image of the orbit shows thickening of the medial and lateral rectus muscles in the left orbit (red arrow) and lid swelling (yellow arrow).

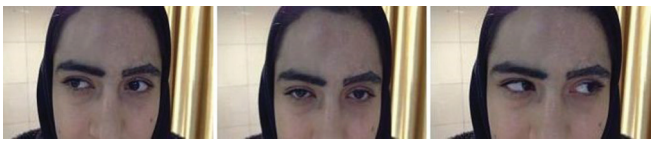

Figure 5. Eye positions in each direction of gaze after 3 months. The blepharoptosis and ophthalmoplegia had resolved.

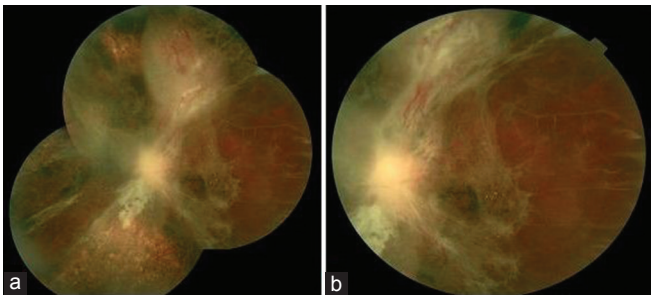

Figure 6. Montage (a) and fundus photograph (b) of the left eye showing optic disc pallor with total vessel attenuation, atrophic changes at the macula, and a relatively featureless retina with diffuse disturbance of retinal pigment epithelium.

A fundus examination of the left eye 3 months later showed optic disc pallor with totally attenuated and sclerosed retinal vessels, diffuse preretinal gliosis, chorioretinal atrophy, and disturbance of the retinal pigment epithelium, suggesting obstruction of the retinal and choroidal circulations. These observations are in accordance with previous reports of a poor visual prognosis in eyes with obstructionof both the retinal and choroidal circulations.[11]

The cause of the complete ophthalmoplegia and thickening of the rectus muscles in this patient is not known. Various mechanisms have been proposed for development of ischemic neuropathy and ophthalmoplegia following periocular injections.[12] These mechanisms include the following:

1. The sclerosing agent could flow into the arteries of the extraocular muscles and their innervations from the hemangioma and cause ischemic injury to the muscle
2. An allergic reaction to the sclerosing agent could cause orbital tissue edema (as was evident in the orbital CT scans in our patient), leading to compression of the mascular arteries[12]

3. Compressive optic neuropathy as a result of orbital inflammation and edema because of inadvertent spread of the sclerosing agent to the orbit with a tamponade effect that could lead to partial or complete collapse of the arterial and venous channels of the orbit.[13]

Furthermore, orbital apex syndrome and cavernous sinus thrombosis may have similar presentations and should be kept in mind.[14,15]

In conclusion, permanent loss of vision and total ophthalmoplegia following periocular injection of a sclerosing agent, although rare, is a devastating complication. Surgeons should be aware of this potential complication and take appropriate preventive and therapeutic measures.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of Interest

There are no conflicts of interest.

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