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. 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.

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.

In 1981, Chamot et al reported total blindness and phthisis bulbi after sclerotherapy for frontal cutaneous hemangioma in a 4-month-old baby. This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Dehghani A, Rezaei L, Ghanbari H, Nasrollahi K, Tavakoli M. Ophthalmic artery occlusion following facial sclerosing therapy. J Ophthalmic Vis Res 2018;13:351-4.
In 2009, Matsuo et al described visual loss caused by occlusion of the central retinal and posterior ciliary arteries following intraliesional 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 intraliesional 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 intraliesional 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.

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.

A fundus examination of the left eye 3 months later showed optic disc pallor with total vessel attenuation, atrophic changes at the macula, and a relatively featureless retina with diffuse disturbance of retinal pigment epithelium.

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 muscular 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.\textsuperscript{[13]}

Furthermore, orbital apex syndrome and cavernous sinus thrombosis may have similar presentations and should be kept in mind.\textsuperscript{[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.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

There are no conflicts of interest.

REFERENCES

1. Berg KT, Harrison AR, Lee MS. Perioperative visual loss in ocular and nonocular surgery. \textit{Clin Ophthalmol} 2010;4:531-546.
2. Myers MA, Hamilton SR, Bogosian AJ, Smith CH, Wagner TA. Visual loss as a complication of spine surgery: A review of 37 cases. \textit{Spine (Phila Pa 1976)} 1997;22:1325-1329.
3. Barnacle AM, Theodorou M, Maling SJ, Abou-Rayyah Y. Sclerotherapy treatment of orbital lymphatic malformations: A large single-centre experience. \textit{Br J Ophthalmol} 2016;100:204-208.
4. Chamot L, Zografos L, Micheli JL. Ocular and orbital complications after sclerosing injections in a case of a frontal cutaneous angiomata. \textit{Ophthalmologica} 1981;182:193-198.
5. Matsuo T, Fujiwara H, Gobara H, Mimura H, Kanazawa S. Central retinal and posterior ciliary artery occlusion after intralesional injection of sclerosant to glabellar subcutaneous hemangioma. \textit{Cardiovasc Intervent Radiol} 2009;32:341-346.
6. Kanter A, Thibault P. Saphenofemoral incompetence treated by ultrasound-guided sclerotherapy. \textit{Dermatol Surg} 1996;22:648-652.
7. Li X, Du L, Lu JJ. A novel hypothesis of visual loss secondary to cosmetic facial filler injection. \textit{Ann Plast Surg} 2015;75:258-260.
8. Egbert JE, Paul S, Engel WK, Summers CG. High injection pressure during intralesional injection of corticosteroids into capillary hemangiomas. \textit{Arch Ophthalmol} 2001;119:677-683.
9. Lazzari D, Agostini T, Figus M, Nardi M, Pantaloni M, Lasser S. Blindness following cosmetic injections of the face. \textit{Plast Reconstr Surg} 2012;129:995-1012.
10. Sui HJ, Yin L, Yu SB. Anatomical Altas of Interventional Therapy: Nerves and Blood Vessels. Shenyang: Liaoning Science and Technology Press; 2006.
11. Brown GC, Magargal LE, Sergott R. Acute obstruction of the retinal and choroidal circulations. \textit{Ophthalmology} 1986; 93:1373-1382.
12. Feied CF, Jackson JJ, Bren TS, Bond OB, Fernando CE, Young VC, et al. Allergic reactions to polidocanol for vein sclerotherapy. Two case reports. \textit{J Dermatol Surg Oncol} 1994;20:466-468.
13. Hollenhorst RW, Sven HJ, Benoit CF. Unilateral blindness occurring during anaesthesia for neurosurgical operations. \textit{AMA Arch Ophthalmol} 1954;52:819-830.
14. Chang-Godinich A, Lee AG, Brazis PW, Liesegang TJ, Jones DB. Complete ophthalmoplegia after zoster ophthalmicus. \textit{J Neuroophthalmol} 1997;17:262-265.
15. Kraus CL, Culican SM. Challenging presentations of cavernous sinus thrombophlebitis. \textit{J Ophthalmic Inflamm Infect} 2012;2:133-136.