Progression of glaucomatous optic neuropathy associated with chorioretinal microvascular embolism after intranasal injection of a corticosteroid suspension

Daisuke Nagasato, Naohiro Ikeda, Akiko Masuda, Ryosuke Kashimoto, Tomohiro Ikeda

A patient with glaucoma developed sudden blurred vision immediately after the nasal mucosal injection of a betamethasone acetate solution into the inferior turbinate. The fundus examination revealed several white emboli in the choroidal vessels of the temporal region of the optic disc. After vigorous massage, her visual acuity recovered from counting fingers to 20/20. Six days after the initial examination, Goldmann perimetry showed expansion of the superior and inferior arcuate scotomas. In this case, temporary ischemia of the central retinal and short posterior ciliary arteries involving the arterial circle of Zinn–Haller led to the deterioration of the preexisting glaucomatous optic neuropathy.

Key words: Glaucomatous optic neuropathy, ischemia, steroid suspension, visual acuity

Olfactory disturbances stemming from chronic rhinitis or sinusitis are generally treated with nasal steroid drippings. Recently, an occasional injection of a steroid suspension (SS) into the inferior turbinate was suggested for a longer-lasting effect. SS intranasal injection is a safe treatment when performed correctly. However, in rare cases, inadequate injection techniques or significant deviations from the correct SS injection protocol may cause a temporary or permanent visual reduction. We report the case of a patient who developed a sudden reduction of vision and a subsequent rapid progression of pre-existing glaucomatous optic neuropathy associated with chorioretinal circulatory disturbances after SS intranasal injection.

Case Report

A 63-year-old woman with an olfactory disturbance was referred to our clinic with blurred vision and ophthalmoplegia in the right eye that developed immediately after the nasal mucosal injection of a betamethasone acetate solution into the ipsilateral inferior turbinate by an otolaryngologist. The best-corrected visual acuity of her right eye was reduced to counting fingers at approximately 30 min after the injection. She took 20 min to the first exam. We performed digital massage over her closed eyelids for about 30 min. After this ocular massage, her best-corrected visual acuity was recovered to 20/20. The fundus examination revealed the presence of several white emboli in the choroidal vessels of the temporal region of the optic disc. The vertical cup/disc ratio was 0.9 in both eyes, with a loss of the superior temporal and inferior neural rim and nasal displacement of the major retinal vessels [Fig. 1a]. The intraocular pressure was 17 mmHg OD and 18 mmHg OS. A hypofluorescent area was noted around the optic disc on indocyanine green angiography [ICGA; Fig. 1b]. Goldmann perimetry detected a nasal visual field defect and superior and inferior arcuate scotomas in the right eye [Fig. 1c].

A diagnosis of chorioretinal microvascular embolism accompanied by normal-tension glaucoma was established. Drip infusion of prostaglandin E1 and topical administration of latanoprost 0.005% and timolol 0.5% were initiated. She was given 50 µg of prostaglandin E1 intravenously over 120 min. 6 days after treatment, an expansion of the superior and inferior arcuate scotomas was observed on Goldmann perimetry [Fig. 1d]. Her best-corrected visual acuity recovered to 20/20.

Discussion

The ethmoidal arteries are branches of the ophthalmic artery that are distributed in the nasal mucosa. During the intranasal injection of SS, the tip of the needle can be accidentally inserted into these arteries at an infusion pressure that is higher than the arterial blood pressure. In these rare cases, the SS can then travel retrogradely, reach the ophthalmic artery, and enter the chorioretinal circulation through the central retinal artery (CRA) and short posterior ciliary arteries (SPCAs). Subsequently, the SS can form emboli that enter the chorioretinal vessels.

Among the various SSs, 6 mg/mL solutions of betamethasone contain particles measuring 90–117 µm, which can be detected by fundus examination. Conversely, the diameter of the terminal retinal and choroidal arteries is 8–15 µm and the size of the arterial circle of Zinn–Haller is 20–230 µm. Triamcinolone adopts a size similar to that of betamethasone suspension.
when aggregated. Li et al. reported the occurrence of embolic retinal and choroidal vascular occlusion after peribulbar triamcinolone injection. Therefore, the SS particles may develop into emboli at the choroidal and retinal artery level, including the CRA, SPCA, and the arterial circle of Zinn–Haller.

In our case, untreated glaucoma was observed during the first examination. The patient exhibited a visual acuity of counting fingers immediately after the intranasal injection, suggesting that the CRA had been occluded. The observation that her visual acuity recovered after vigorous eye massage indicates that the blood flow of the CRA was restored by this technique.

The hypofluorescence observed around the optic disc on ICGA indicates low perfusion of the short posterior ciliary artery in this region. An experiment using macaque monkeys showed that occlusion of the temporal side of the arterial circle of Zinn–Haller caused partial oxygen deficiency in the temporal arcuate fiber region, in which glaucomatous optic neuropathy develops, and induced atrophy of the arcuate bundle in the anterior lamina cribrosa over time. Moreover, in a retinal ischemia experiment involving ligation of the ophthalmic blood vessel for 90 or 120 min in Sprague Dawley rats, about half of retinal ganglion cells were lost within the first 5 days, followed by the disappearance of 40% or more of the remaining cells within several months.

In the case reported here, temporary chorioretinal microvascular embolism of the SS was observed during the first examination. The temporary ischemia of the retina caused by multiple emboli in the CRA and SPCA involving the arterial circle of Zinn–Haller led to apoptosis of the retinal ganglion cells in the temporal arcuate region. This caused further deterioration of the pre-existing glaucomatous optic neuropathy within a short period, and expansion of the superior and inferior arcuate scotomas was observed on a visual field test.

SSs can reach the ophthalmic system retrogradely through an anastomosis between the branches of the external carotid and ophthalmic arteries. Clinicians need to be aware that the accidental injection of an SS into the branches of the external carotid arteries and ophthalmic arteries in the facial area can cause visual impairment.

**Conclusion**

To prevent devastating visual complications, SS injection in the facial areas should be performed as slowly as possible and in small quantities by applying the lowest pressure possible. Careful aspiration before injection must be performed.

**Declaration of patient consent**

This study adhered to the tenets of the Declaration of Helsinki. The study protocol was approved by the ethics committee of the institution. The patients gave their written informed consent prior to this study.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Moss WJ, Kjos KB, Karnezis TT, Lebovits MJ. Intranasal steroid injections and blindness: Our personal experience and a review of the past 60 years. Laryngoscope 2015;125:796-800.
2. Byers B. Blindness secondary to steroid injections into the nasal turbinates. Arch Ophthalmal 1979;97:79-80.
3. Derby R, Lee SH, Date ES, Lee JH, Lee CH. Size and aggregation of corticosteroids used for epidural injections. Pain Med 2008;9:227-34.
4. Ko MK, Kim DS, Ahn YK. Morphological variations of the peripapillary circle of Zinn-Haller by flat section. Br J Ophthalmol 1999;83:862-6.
5. Li G, Xu D, Hu Z, Li H. Embolic retinal and choroidal vascular occlusion after peribulbar triamcinolone injection: A case report. Medicine (Baltimore) 2018;97:e0467.
6. Hiraoka M, Inoue K, Ninomiya T, Takada M. Ischaemia in the Zinn-Haller circle and glaucomatous optic neuropathy in macaque monkeys. Br J Ophthalmol 2012;96:597-603.
7. Lafuente MP, Villegas-Pérez MP, Sellés-Navarro I, Mayor-Torroglosa S, Miralles de Imperial J, Vidal-Sanz M. Retinal ganglion cell death after acute retinal ischemia is an ongoing process whose severity and duration depends on the duration of the insult. Neuroscience 2002;109:157-68.
8. McEwan G, Hofmeister E, Kubis K, Blade K. Monocular embolic retinal arteriolar occlusions after ipsilateral intrarostral triamcinolone injection. J Neuro-Ophthalmol 2010;30:94-103.