



Figure 2. Axial computed tomographic scan with contrast showing left orbital lesion extending posteriorly beyond septal plane with central hypodensity and rim enhancement but no adjacent bone erosion, edema, or sinus disease. R indicates right.

cillin allergy. Microbiology cultures identified *Staphylococcus aureus* that was sensitive to clindamycin. The drain was removed 3 days after insertion, and the antibiotics were administered for 2 weeks with clinical resolution of the abscess and no recurrence 8 months after treatment.

Comment. Subtenon corticosteroid injections are commonly used in the treatment of macular edema or posterior segment intraocular inflammation. Complications include inadvertent intravascular injection, globe perforation, cataract formation, ocular hypertension, blepharoptosis, orbital fat atrophy, strabismus, or allergic reactions.^{3,4} To our knowledge, this is the first report of an abscess resulting from such an injection. The lack of inflammation was atypical for an infection and was likely related to the local immunosuppressive effects of triamcinolone.

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1. Lesser A, Stern GA. Preseptal and orbital cellulitis. *Infect Dis Clin North Am.* 1992;6:933-952.
2. Hornblass A, Herschorn BJ, Stern K, Grimes C. Orbital abscess. *Surv Ophthalmol.* 1984;29:169-178.
3. Helm CJ, Holland GN. The effects of posterior subtenon injection of triamcinolone acetonide in patients with intermediate uveitis. *Am J Ophthalmol.* 1995;120:55-64.
4. Dafflon ML, Tran VT, Guex-Crosier Y, Herbort CP. Posterior sub-tenon's steroid injections for the treatment of posterior ocular inflammation: indications, efficacy and side effects. *Graefes Arch Clin Exp Ophthalmol.* 1999;237:289-295.

Acute Angle-Closure Glaucoma Associated With Intranasal Phenylephrine to Treat Epistaxis

Phenylephrine hydrochloride is a direct-acting α_1 -adrenergic agonist used for its mydriatic and vasoconstrictive properties. In addition to its ophthalmic uses, phenylephrine is used in the management of epistaxis for which it is instilled intranasally to induce vasoconstriction prior to cautery or packing. We describe a patient who developed sequential ipsilateral acute angle-

closure attacks after intranasal phenylephrine use.

Report of a Case. At initial examination, a 67-year-old woman had right eye pain, redness, blurry vision, and nausea. Prior to the onset of symptoms, the patient had experienced right-sided epistaxis that was treated in an emergency department with intranasal 0.25% phenylephrine hydrochloride, topical tetracaine, silver nitrate cautery, and nasal packing.

Visual acuity was 20/200 OD and 20/40 OS. The patient's refractive error was +1.75 diopters (D) OD and +2.00 D OS. The right pupil measured 7 mm and was nonreactive. The left pupil measured 4 mm and constricted to direct and consensual stimulation. Intraocular pressure was 62 mm Hg OD and 18 mm Hg OS. The right eye had conjunctival hyperemia, corneal edema, a quiet anterior chamber, and iris bombe. The lens had moderate nuclear sclerosis. Findings from the left eye were unremarkable, except for similar lens changes. There was a hazy view of the right fundus. The left fundus was normal, with a cup-disc ratio of 0.2.

The patient was treated with 1 drop of 0.5% timolol maleate and 2% dorzolamide hydrochloride to the right eye followed by 100 mL of oral glycerin. This was repeated 15 minutes later. An attempt to create a Nd:YAG laser peripheral iridotomy was unsuccessful. The patient had multiple episodes of emesis during treatment, and she was transferred to the emergency department, where she received 12.5 g of intravenous mannitol and 1 drop of 4% pilocarpine hydrochloride. Intraocular pressure improved to 30 mm Hg. The patient was given 12.5 mg of promethazine hydrochloride for emesis and subsequently developed mental status changes, and she was admitted for observation.

The evening prior to hospital discharge, the patient had an episode of left-sided epistaxis and was emergently treated, without ophthalmic consultation, with intranasal 0.25% phenylephrine hydrochloride and nasal packing by the hospital staff (not affiliated with the hospital of initial examination) who were unaware of the inciting event of her

angle-closure attack. The following day the patient was seen for left eye pain, redness, and blurry vision. Visual acuity was 20/100 OS, and there was corneal edema and iris bombe. Intraocular pressure was 42 mm Hg. The patient was treated with 2% pilocarpine hydrochloride and 0.2% brimonidine tartrate to the left eye and underwent Nd:YAG laser peripheral iridotomy in the left eye as well as revision of iridotomy in the right eye. Intraocular pressure normalized in both eyes.

The patient was referred to the Kresge Eye Institute/Detroit Medical Center. Visual acuity was 20/40 OD and 20/30 OS. Intraocular pressure was 17 mm Hg OD and 16 mm Hg OS. There were 2 patent peripheral iridotomies in the right iris and 1 patent iridotomy in the left iris. Fundus examination findings were normal in both eyes.

Comment. To our knowledge, there are no previously reported cases of acute angle-closure glaucoma secondary to the intranasal application of phenylephrine. Phenyleph-

rine is frequently used in the acute management of epistaxis because of its vasoconstrictive properties. Our patient experienced sequential angle-closure attacks after the administration of intranasal phenylephrine that likely were precipitated by phenylephrine-induced pupillary mydriasis in the ipsilateral eye. Previous reports of angle-closure glaucoma induced by intranasally administered substances have been reported.^{1,2} Hari et al¹ reported a case of acute angle-closure glaucoma after ipsilateral intraoperative painting of the nasal mucosa with 25% cocaine paste. Mitchell and Schwartz² described a patient with a history of cocaine abuse who developed angle-closure glaucoma ipsilateral to the intranasal use (the patient had a traumatically deviated septum and could only use 1 naris). In these cases, like ours, the angle closure occurred in the ipsilateral eye. There is some evidence that intranasally administered aerosolized medications may reflux through the ipsilateral nasolacrimal duct and be detected in tears.³ Systemic absorption through

the nasal mucosa is another possible mechanism of action. Physicians should be aware of this uncommon cause of acute angle-closure glaucoma.

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1. Hari CK, Roblin DG, Clayton MI, Nair RG. Acute angle closure glaucoma precipitated by intranasal application of cocaine. *J Laryngol Otol.* 1999; 113:250-251.
2. Mitchell JD, Schwartz AL. Acute angle-closure associated with intranasal cocaine abuse. *Am J Ophthalmol.* 1996;122:425-426.
3. Orlick ME, Kastl AR, Donzis PB, Howard R, Rice J, Tauber S. Ocular effects and detection in tears of aerosolized intranasal cocaine and fluorescein. *Ann Ophthalmol.* 1990;22:249-254.