CASE REPORT

A 60-year-old man who had worked manufacturing photographic film for 39 years was referred to the Glaucoma Service following an earlier diagnosis of and treatment for glaucoma.

His medical history was significant for hypertension and hyperlipemia. He denied a family history of glaucoma. He initially had been seen by an ophthalmologist 8 months before. At that time, visual acuity was 20/20 in both eyes and there was a relative afferent pupillary defect in the left eye. Intraocular pressures (IOPs) by applanation tonometry were 20 mm Hg in the right eye and 36 mm Hg in the left. Slit-lamp examination showed a punctate gray appearance on the posterior aspect of both corneas. Gonioscopy revealed grade-4 angles in both eyes. A dilated fundus examination was noteworthy for glaucomatous optic atrophy in the left eye. Automated static visual field testing showed a suprropsychic defect in the left eye.

Following this initial examination, the patient had been started on levobunolol hydrochloride 0.5% in the left eye, with an initially favorable response. He was then started on pilocarpine 1%, but he discontinued it after experiencing blurred vision and headaches. Argon laser trabeculoplasty was performed in the left eye, following which the IOP dropped from 22 mm Hg to 18 mm Hg.

The initial examination at the Glaucoma Service showed a visual acuity of 20/20 in both eyes, with IOPs of 23 mm Hg in the right eye and 22 mm Hg in the left eye. The slit-lamp examination was noteworthy for gray staining of the palpebral and bulbar conjunctiva (Fig 1) and dirty-gray-appearing posterior corneas in both eyes. Gonioscopy revealed deep, open angles, with a prominent appearance to Schwalbe's line in both eyes (Fig 2), most likely due to surrounding silver pigment. Fundus examination and visual field testing were consistent with a diagnosis of glaucoma in the left eye.

Guarded filtration surgery was recommended, but the patient elected to defer surgery and is currently taking both levobunolol hydrochloride 0.5% and dipivefrin hydrochloride 0.01% in the left eye two times a day.

DISCUSSION

Ocular argyrosis has been reported as a common side effect of prolonged use of silver-protein-salt eye solutions (Argyrol). Most reports describe conjunctival, corneal, and lacrimal sac involvement, with silver deposits. Systemic argyria reportedly leaves deposits in the basement membranes, including the lens capsule, Bowman's membrane, and Bruch's membrane. Ocular argyrosis is not known to affect vision, but 10 of 30 workers in this study reported experiencing nightblindness. Seven of these 10 underwent detailed testing, which revealed no physiologic abnormalities. However, nyctalopia was directly correlated with the level of argyrosis.

Ocular argyrosis has mainly been associated with the use of Argyrol, but cases also have been described among silver polishers, factory workers manufacturing silver nitrate and silver acetate, a pipe-fitter using silver soldering, a photographer using developing solutions who frequently rubbed her eyes, and individuals manufacturing photographic film.

Clinical studies have reported typical findings of ocular argyrosis patients as follows: Silver acetate and silver nitrate factory workers usually have conjunctival deposits, most frequently in the caruncle and semilunar...
folds. Most cases of Argyrol-induced argyrosis involve the inferior cul-de-sac, although the entire bulbar conjunctiva can be affected. Corneal deposition is located primarily in the anterior portion of Descemet’s membrane, although a case of an Argyrol-treated corneal ulcer revealed more superficial involvement. Lacrimal sac involvement has been shown to stain the overlying skin a dark gray. To our knowledge, no study has looked at the gonioscopic appearance of ocular argyrosis. Only one patient in the above studies had open-angle glaucoma, but the argyrosis was considered unrelated.

Several histologic and ultrastructural studies have further illustrated the location of silver in the eye. Transmission electron microscopy has shown intracellular conjunctival silver deposits and extracellular corneal deposits in the anterior two thirds of Descemet’s membrane. One histologic study revealed chronic inflammation surrounding silver granules in the lacrimal sac. Finally, Bruch’s membrane has been shown to be diffusely involved with silver deposits in cases of systemic argyrosis. In one fluorescein study, this deposition produced a “silent choroid” indistinguishable from that seen in Stargardt’s disease. The trabecular meshwork was free of silver deposition in two enucleated eyes of patients with ocular argyrosis.

Our patient has classic findings of ocular argyrosis. The conjunctiva is diffusely involved, and the cornea is involved at the layer of Descemet’s membrane. In addition, Schwalbe’s line is abnormally prominent. There is no known association between argyrosis and glaucoma. Glaucoma has been caused by siderosis bulbis, although the mechanism is unclear. There is no pathologic evidence to support a causal effect of silver deposition on our patient’s glaucoma. However, the apparent involvement of Descemet’s membrane by silver deposition suggests that silver may affect the adjacent trabecular meshwork. Further evaluation is needed to clarify this point.

REFERENCES