3. Keane JR. Combined VIth and XIIth cranial nerve palsies: a clival syndrome. Neurology 2000;11:1540–1541.
4. Malone DG, O'Boynick PL, Ziegler DK, et al. Osteomyelitis of the skull base. Neurosurgery 1992;30:426–431.
5. Girkin CA, Perry JD, Miller NR, Reich SG. Pachymeningitis with multiple cranial neuropathies and unilateral optic neuropathy secondary to *Pseudomonas aeruginosa*: case report and review. J Neuroophthalmol 1998;18:196–200.

Corneal Tattooing for the Treatment of Debilitating Glare in a Child With Traumatic Iris Loss

Arif O. Khan MD, and David Meyer, MB ChB, PhD

PURPOSE: To report the cosmetic and therapeutic use of corneal tattooing for a child with sectoral traumatic corneal scarring and symptomatic glare from sectoral traumatic iris loss.

DESIGN: Interventional case report.

METHODS: A six-year-old girl underwent corneal tattooing (platinum chloride reduced by hydrazine) in the relevant scarred corneal sector.

RESULTS: Six months after the procedure, the child enjoyed a more normal corneal appearance and no longer suffered from glare. Postoperative epithelial healing, however, was slow and required vigilance.

CONCLUSIONS: Corneal tattooing can allow both cosmetic and therapeutic benefit when indicated in a child. However, postoperative healing may require management when using platinum chloride reduced by hydrazine.

FOR CENTURIES, CORNEAL TATTOOING HAS BEEN USED IN adults for unsightly corneal scars and, less commonly, symptomatic glare associated with iris loss.[1-3] We report the use of this procedure for both indications (cosmetic and therapeutic) in a child. A 6-year-old brown-eyed girl was suffering from debilitating glare in the right eye since corneal laceration repair in that eye at 5 years of age. Examination showed uncorrected visual acuity of 20/40 OD and 20/20 OS. There were significant findings nasally OD: a vertical linear corneal scar, loss of 3 clock-hours of peripheral iris, including the pupillary margin, and exposure of the crystalline lens edge (Figure 1). Cycloplegic refraction (cyclopentolate hydrochloride 1%, Alcon, Spain) was +2.50–3.00 × 060 OD and +1.50–0.50 × 180 OS. With this correction visual acuity OD was 20/30. For cosmetic and optical benefit, corneal tattooing OD was performed. The corneal epithelium in the area of iris defect was removed with a sharp blade. A piece of filter paper cut to the size of the defect that had been soaked in platinum chloride 2% (from platinum [IV] chloride anhydrous 57.5%, Merck, Germany) was applied to the de-epithelialized cornea for 2 minutes. A second piece of similarly-sized filter paper that had been soaked in hydrazine 2% (from hydrazine hydrate, Signa-Aldrich, Germany) was then applied to the de-epithelialized area for 25 seconds. The eye was observed until the treated area became dense black in color (approximately 30 seconds) and was then vigorously irrigated with balanced saline solution for 1 minute. Cyclopentolate 1%, ofloxacin 0.3% (Jamjoon Pharma, Saudi Arabia), and a pressure patch were applied. The next day the epithelial defect was unchanged. A bandage contact lens was placed (but was lost within 1 day) and ofloxacin 0.3% four times a day OD was prescribed. Because the epithelial defect still persisted 10 days postoperatively, autologous serum 20% drops were prescribed OD every 3 hours while awake, as well as tetracycline ophthalmic ointment 1% (Riyadh Pharma, Saudi Arabia) at bedtime. Three weeks postoperatively the cornea was re-epithelialized, and the medications were discontinued. At her 6-month follow-up, the patient had an uncorrected visual acuity of 20/25 OD, no symptoms of glare, and an improved cosmetic appearance (Figure 2).

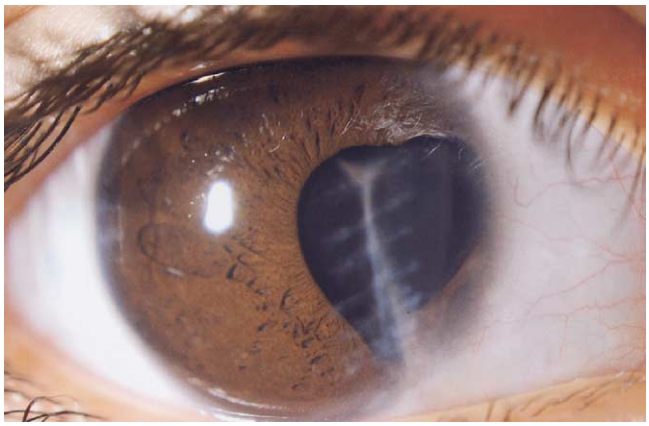

FIGURE 1. Before corneal tattooing: Nasally OD, there is a linear paracentral scar, missing iris, and an exposed edge of the crystalline lens. The patient was symptomatic from glare effect OD (pupil not pharmacologically dilated in figure).

Historically, there are two techniques for corneal tattooing: (1) imbedding insoluble pigment into the corneal stroma (India ink, iron oxide, titanium dioxide), or (2) placing metallic salts (gold chloride, silver nitrate, platinum chloride) on the de-epithelialized corneal stroma and then chemically reducing them.[1] Use of hydrazine to reduce platinum chloride results in dispersion of platinum metal ("platinum black"). This gives a darker and more permanent color[2] (but does not

Accepted for publication Oct 18, 2004.

From the Department of Pediatric Ophthalmology (A.O.K.) and Department of Oculoplastic Surgery (D.M.), King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia; Department of Ophthalmology (D.M.), Faculty of Health Sciences, University of Stellenbosch, Tygerberg, South Africa.

Inquiries to Arif O. Khan, MD, Consultant Pediatric Ophthalmologist, King Khaled Eye Specialist Hospital, P.O. Box 7191, Riyadh, 11462, Saudi Arabia; fax: 966 01 482 9311; e-mail: arif.khan@mssm.edu

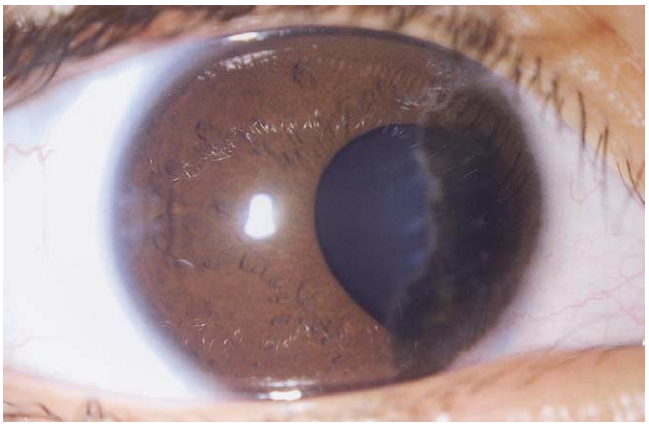

FIGURE 2. After corneal tattooing: Six months after the procedure in the affected area OD, the patient was asymptomatic and had an improved cosmetic appearance (pupil not pharmacologically dilated in figure).

allow for color variation). Our patient's delayed postoperative epithelialization, a reason for caution before recommending this procedure, was likely because of the duration of hydrazine exposure. Hydrazine is a reducing agent with a known potential for epithelial surface toxicity.[4,5] Its intraoperative application should perhaps be less than the 55 seconds we allowed. Follow-up is required to assess whether significant fading of pigment will occur over the long-term.[3]

REFERENCES

1. Leigh AG. Tattooing of the cornea. In: Duke-Elder S. System of ophthalmology. Volume 8. Part 2. London: Henry Kimpton, 1965:645–648.
2. Roper-Hall MJ. The conjunctiva, cornea, and sclera. In: Stallard's eye surgery. 7th ed. Kent: Butterworths 1989;252–254.
3. Reed JW. Corneal tattooing to reduce glare in cases of traumatic iris loss. Cornea 1994;13(5):401–405.
4. Budavari S. In: Budavari S, editor. The Merck index–an encyclopedia of chemicals, drugs, and biologicals. Whitehouse Station, New Jersey: Merck and Co. Inc., 1996:816.
5. Hathaway GJ, Proctor NH, Hughes JP. In: Hathaway GJ, Proctor NH, Hughes JP editors. Chemical hazards of the workplace. 3rd ed. New York: Van Nostrand Reinhold. 1991:329–331.

Corneal Pseudomembrane From Acute Inflammatory Response and Fibrin Formation to Acute Myeloid Leukemic Infiltrate

Shree K. Kurup MD, Hanna Coleman MD, and Chi-Chao Chan, MD

PURPOSE: To describe an unusual ocular manifestation of a patient with acute myeloid leukemia (AML).

DESIGN: Observational case report.

METHODS: A 59-year-old woman with a history of preleukemic myelodysplastic syndrome (MDS) and status post bone marrow transplant (BMT) complained of a sudden onset of poor vision associated with a corneal pseudomembrane. Ocular graft vs host disease was suspected, and the pseudomembrane was excised for histopathologic examination.

RESULTS: The pseudomembrane showed myeloblasts admixed with an acute inflammatory response suggestive of the development of AML, a complication of MDS. Bone marrow examination confirmed the diagnosis of relapsing AML.

CONCLUSIONS: Acute myeloid leukemia could present as a pseudomembrane; thus, examination of relevant ocular tissue is recommended. (Am J Ophthalmol 2005;139: 921–923. © 2005 by Elsevier Inc. All rights reserved.)

Accepted for publication Oct 20, 2004.

CORNEAL PSEUDOMEMBRANES ARE MEMBRANOUS INflammatory coagulums lacking true basement membrane and abundant collagen tissue that are usually associated with infections of the anterior segment.[1] We herein present the first known case of a hematologic malignancy presenting as a corneal pseudomembrane.

A 59-year-old, diabetic female presented to the eye clinic with progressively worsening vision in the right eye for the past 2 days. The patient has a history of myelodysplastic syndrome (MDS) diagnosed 22 years ago. She was treated with equine anti-thymocyte serum, maintained on blood transfusions and received a successful nonmyeloablative 6/6 HLA matched sibling bone marrow transplantation 2 years ago. She suffered from two consequent skin and gastrointestinal graft vs host disease that was controlled by immunosuppressives. On clinical examination on June 2, 2004, at the National Eye Institute, the vital signs were normal, and the patient's medications were cyclosporine 75 mg twice daily for the graft and glyburide 5 mg daily for type II diabetes mellitus. She presented numerous peticheal lesions on the skin consistent with a history of low platelets. The vision was 20/40 and 20/20 in the right and left eye, respectively. There was no pupillary afferent defect, and the external ocular movements were full. The intraocular pressures were recorded as 6 mm and 8 mm in the right and left eye, respectively. There was a thin pinkish membrane from the inner surface of the upper eyelid covering two thirds of the superior cornea, which obscured the right pupil. The membrane was removed

From the National Eye Institute, National Institutes of Health, Bethesda, Maryland.

Inquiries to Shree K. Kurup, MD, Building 10, Room 10N112, National Eye Institute, National Institutes of Health, Bethesda, MD 20892; fax: (301) 496-7295; e-mail: kurups@nei.nih.gov