Foreign Body Entrapment in Radial Keratotomy Incisions

Radial keratotomy (RK) incisions may gape in response to external trauma, allowing small or particulate foreign bodies to become embedded within.

Report of a Case. A 41-year-old man had successful bilateral 8-incision RKs done elsewhere in 1992, resulting in 20/20 OU uncorrected visual acuity. As he was trimming a bush in September 1998, a branch (Figure 1) struck him in the right eye. There was immediate pain, blurriness, and tearing. He was seen immediately at a local hospital emergency department by an ophthalmologist and was referred to our cornea clinic for further treatment.

At presentation to our clinic, his uncorrected visual acuity was 20/70 OD and 20/20 OS. Manifest refraction in the right eye improved the visual acuity to 20/50 with a +0.75+1.00×135 correction. Dark brown particles of what appeared to be wood bark were wedged in the anterior corneal stroma in the nasal, inferonasal, and inferior RK incisions (Figure 2). The anterior lips of these incisions were splayed open by a combination of the foreign bodies, reactive inflammation, and secondary corneal edema. In addition, there was a partial-thickness, paracentral corneal laceration perpendicular to and crossing the 3 injured RK incisions. Findings from the Seidel test were negative. The rest of the anterior segment, including the crystalline lens, was normal. A dilated retinal examination revealed no intraocular foreign bodies or retinal pathologic condition.

Under retrobulbar anesthesia, the foreign bodies were easily swept out of the RK incisions with a Barraquer iris sweep and were sent for microbiological cultures. The 3 RK incisions, together with the anterior stromal laceration, were copiously irrigated with balanced-salt solution. A subconjunctival injection of cephalosporin was administered and a 1-week course of topical ofloxacin, 4 times daily, was begun.

The uncorrected visual acuity improved to 20/30 OD within 1 week. After 5 weeks, the RK incisions and the paracentral laceration appeared to be healing well, with only mild anterior stromal scarring and mild residual fluorescein pooling over the injured incisions. The uncorrected visual acuity remained 20/30 OD. The cycloplegic refraction was -0.25+0.25×30 OD, giving a corrected visual acuity of 20/25 OD. The cultures were negative for organisms.

Comment. Many studies have previously shown delayed wound healing in RK wounds. The failure of RK incisions to regain full preoperative tensile strength has been responsible for complications ranging from unstable or progressive refractive changes to traumatic rupture of the globe. Radial keratotomy incisions frequently open spontaneously during penetrating keratoplasty, even as late as 9 years after the original surgery. Full-thickness corneal perforation through a preexisting RK incision by a wooden foreign body has been previously reported in the military literature. In that case, the injured eye had undergone a 3-step, 24-incision RK 1 year previously. Our patient had considerably fewer incisions and was already 6 years from the time of RK surgery. Unsutured wounds typically have a plug of epithelial cells that may persist for years, thus weakening the structural integrity of the stroma. Even in the absence of epithelial plugs, well-healed stromal scars have less than 70% of native tensile strength. Consequently, RK wounds appear to

Figure 1. Broken branch responsible for eye injury.

Figure 2. Wood foreign bodies lodged in three consecutive radial keratotomy incisions (direct slit-beam illumination).
be susceptible to entry or entrapment of particulate foreign matter.

Kaz Soong, MD
Ann Arbor, Mich

Reprints: H. Kaz Soong, MD, W. K. Kellogg Eye Center, 1000 Wall St, Ann Arbor, MI 48105.

6. Nolan BT. Perforation by a foreign body through the right eye revealed a “T sign,” suggesting posterior scleritis.

Ocular Inflammation Associated With Alendronate Therapy

Bisphosphonates are potent inhibitors of osteoclast-mediated normal and abnormal bone resorption. Increasingly, they are being used for the management of Paget disease, hypercalcemia associated with malignant neoplasms, painful bone metastases, and osteoporosis. The first approved bisphosphonates, pamidronate and resclonate, have been associated with ocular inflammation including iritis, nonspecific conjunctivitis, episcleritis, and scleritis.1-2 Reintroduction of the offending drug, in some of these cases, led to recurrence of ocular inflammation.3-5

A member of this group, alendronate sodium (Fosamax; Merck & Co Inc, Whitehouse Station, NJ) is 100 to 500 times more potent than initial amino-bisphosphonates6 and is being used successfully to prevent and treat osteoporosis in postmenopausal women. It induces progressive increases in bone mineral density, thereby reducing the incidence of osteoporosis-related pathologic fractures.4,7 Adverse effects include upper gastrointestinal tract complaints such as dyspepsia, heartburn, vomiting, dysphagia, esophageal reflux, and esophageal ulceration and strictures.7 To our knowledge, alendronate has not been previously associated with any kind of ocular inflammation. We report 3 cases of alendronate-associated posterior scleritis and anterior nodular scleritis with possible contiguous orbital inflammation and myositis that resolved after antiinflammatory therapy and discontinuation of alendronate.

Report of Cases. Case 1. A 77-year-old woman reported a 1-month history of right eye pain exacerbated by extraocular movements. Progression to conjunctival hyperemia, horizontal diplopia, and upper eyelid swelling prompted her internist to prescribe, sequentially, a combination product consisting of topical neomycin sulfate and polymyxin B sulfate and dexamethasone and then prednisolone, to which she had a modest response. A subsequent ophthalmologic examination revealed iritis in the right eye. Treatment with topical prednisolone and cyclopentolate for intraocular inflammation and oral prednisone for probable orbital myositis significantly relieved the ocular pain and diplopia, but not the painful lid swelling. Results of a computed tomographic scan of the orbits and erythrocyte sedimentation rate test were reportedly normal. Intercurrent problems of osteoporosis, hypertension, and mitral valve prolapse were being treated with alendronate, atenolol, and aspirin, respectively. Alendronate therapy had been commenced 10 days prior to the ocular symptoms.

Our examination, 1 month after the initial onset of her ocular symptoms, revealed a best-corrected visual acuity of 20/25 OU. Extraocular movements were full and painless with a 12–prism dioptr esotropia on right gaze. The right upper lid was modestly erythematous, swollen, and tender on palpation. Biomicroscopy of the anterior segments revealed a hyperemic conjunctiva in the right eye with no anterior chamber reaction. The intraocular pressures were normal and dilated funduscopy results were unremarkable. A B-scan ultrasound of the right eye revealed a “T sign,” suggesting posterior scleritis.

Topical and oral steroids were continued and tapered gradually. One month after stopping steroid therapy, however, the symptoms and signs recurred. Funduscop examination at this time revealed choroidal striae in the right eye, consistent with posterior scleritis. Ancillary hematologic and serologic tests (complete blood cell count, erythrocyte sedimentation rate, VDRL, antineutrophil cytoplasmic antibodies, and antinuclear antibodies) revealed an abnormal antinuclear antibody titer of 1:160 (speckled pattern). Results of subsequent anti-Ro and anti-La antibody assays were normal and antihistone antibody levels were modestly elevated to 8.3 (reference range, 0-5). Oral and topical steroid therapy was recommenced and tapered accordingly, with good effect. Alendronate was discontinued at this stage. Subsequent evaluations have been stable with no evidence to suggest recurrence of ocular or orbital inflammation.

Case 2. A 57-year-old woman, shortly after starting to take alendronate, noted diplopia, left ocular pressure sensation and pain, and left-sided headaches. The diplopia resolved spontaneously, but she subsequently had recurrent episodes of left upper lid swelling and then developed ocular hypertension. Five months after the initial onset of ocular symptoms she developed redness and pain in the left eye and was found to have left anterior nodular scleritis, which resolved after treatment with topical dicyclofenac. Alendronate was discontinued, without subsequent recurrence of episodes of ocular inflammation.

Case 3. A 71-year-old woman began having right-sided headaches, tearing, and right ocular pain exacerbated by eye movement 3 weeks after starting alendronate therapy. She had horizontal diplopia, limited horizontal versions in the right eye, and modest right lid edema. A B-scan ultrasound examination revealed right choroidal and scleral thickening and orbital magnetic resonance imaging showed orbital inflammatory fat infiltration. The patient was treated for posterior scleritis with a tapering dose of oral prednisone with good effect. Alendronate was discontinued and

ARCH OPHTHALMOL/VOL 117, JUNE 1999
837
there were no recurrences of episodes of ocular inflammation.

Comment. As a pharmaceutical class, the bisphosphonates have previously been associated with various ocular inflammatory entities, namely, scleritis, episcleritis, non-specific conjunctivitis, and anterior uveitis. Most of the reported cases have followed treatment for Paget disease and hypercalcemia with intravenous pamidronate and oral risdonate. Rechallenge with pamidronate has been reported to cause recurrences of anterior uveitis, nonspecific conjunctivitis, and episcleritis. Patients previously tolerant of etidronate, a nonaminod derivative member of this class, later developed anterior uveitis shortly after exposure to bisphosphonates. This has led some to speculate on the importance of nitrogen content in bisphosphonates for stimulating inflammation.

Alendronate, an amino-bisphosphonate commonly used to prevent and treat osteoporosis in postmenopausal women, has not been documented previously to be causally related to ocular inflammations. The 3 patients described here had no history of ocular complaints or connective tissue disorders. They all developed scleritis with and without anterior uveitis and had experienced episodes of painful eye movements and diplopia. All patients were not rechallenged with the presumed offending agent, the temporal relation between alendronate exposure and occurrence of ocular inflammation strongly suggests a causal relationship. Furthermore, other bisphosphonates have caused similar ocular inflammations.

Patient 1 had an elevated antinuclear antibody titer. Although this finding is non-specific, it might represent a propensity to develop connective tissue disorders and alendronate may have exacerbated an innate proclivity for developing inflammation. Alternatively, alendronate may have positively affected the antinuclear antibody level as commonly occurs with other drugs such as hyaluradane and procainamide.

Pamidronate and risdonrate are known to cause idiosyncratic transient pyrexia, elevated C-reactive protein levels, and increases in interleukin 1, interleukin 6, and lymphocyte-stimulating cytokine levels. Alendronate may similarly affect these inflammatory mediators, thereby indirectly stimulating ocular inflammation. Although a firm relationship has not been established between alendronate and ocular inflammation, it is probable that alendronate and the inflammatory episodes we observed are causally related. We therefore recommend considering discontinuation of this drug should signs or symptoms of ocular inflammation develop.

Joyce N. Mbekeani, FRCS, FRCOphth
Thomas L. Slamovits, MD
Barbara H. Schwartz, MD
Hugo L. Sauer, MD
Bronx, NY

This study was supported in part by an unrestricted grant from Research to Prevent Blindness Inc, New York, NY, to the Department of Ophthalmology and Visual Sciences, Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, NY.

Corresponding author: Thomas L. Slamovits, MD, Department of Ophthalmology and Visual Sciences, Montefiore Medical Center, 111 E 210th St, Bronx, NY 10467.

Optic Disc Neovascularization Following Severe Retinoschisis Due to Shaken Baby Syndrome

Shaken baby syndrome has several acute ophthalmologic features that can be helpful in confirming the diagnosis of nonaccidental injury in infants with intracranial hemorrhage. Long-term visual sequelae of shaken baby syndrome include retinal folds, pigment disruption, atrophy, and fibrosis; optic nerve atrophy; and occipital infarction. Herein we report the novel complication of neovascularization of the disc following severe retinal injury due to shaking.

Report of a Case. A 4-month-old infant was admitted to the hospital for lethargy and new-onset seizures. A computed tomographic scan showed intracranial hemorrhages, diffuse cerebral edema, and almost total infarction of the occipital lobes. An ophthalmologist was consulted to examine for retinal hemorrhages.

Visual behavior could not be evaluated. Both retinas showed massive hemorrhagic retinoschisis cavities elevated into the central
vitreous, which encompassed and extended outside of the posterior poles. Of the remaining retina, no vascular details were visible owing to complete saturation with blood. The optic discs could not be identified. The right eye had a small streak of intravitreal hemorrhage over the dome of the schisis cavity.

The child was followed up 5 weeks, 2 months, and 4 months after the original consultation. On the first clinic visit, a dispersed, white, vitreous hemorrhage was seen in the right eye that prevented a view of the retina; partial resorption of hemorrhage and settling of the schisis cavity had occurred in the left eye. At the 2-month follow-up, the right vitreous hemorrhage had not cleared but the left retinal hemorrhage had mostly resorbed, leaving a visible macular pigmentary clump and distorted retinal vasculature. There was no neovascularization. Two months later, there was still no view of the right retina, but florid neovascularization of the left optic disc had developed.

An examination under anesthesia was performed along with fluorescein angiography. The neovascularization consisted of large-caliber vessels that extended from the left optic disc directly anteriorly, crossing two thirds of the vitreous cavity; there was frondlike arborization of the anterior half of the vessels (Figure). These vessels leaked fluorescein rapidly. There was widespread posterior and midperipheral retinal nonperfusion. Panretinal argon green laser photoagulation was applied.

Comment. Vigorous shaking of infants can cause intracranial hemorrhage and intraocular hemorrhage. The intraocular hemorrhage can be subretinal, intraretinal, preretinal, or intravitreal. Retinal schisis cavities, sometimes partly or fully hemorrhagic, can also be seen acutely and are believed to be due to anteroposterior vitreous traction on the internal limiting membrane. Late retinal sequelae of these shaking-related injuries include pigmentary disturbance, retinal fibrosis, and retinal folds.

To our knowledge, this is the first report of the complication of neovascularization of the disc following retinal schisis and hemorrhage from a shaking injury. Neovascularization has been reported with juvenile retinoschisis and senile retinoschisis. These authors believed that retinal ischemia in the area of the schisis cavity was the mechanism. We believe that our patient also had widespread retinal ischemia on the basis of 2 mechanisms related to anteroposterior vitreous traction on the internal retina: ischemia of superficial retinal layers due to widespread retinoschisis, and ischemia of deeper retina due to disruption of the retinal capillary networks. The time of onset, between 2 and 4 months after injury, is similar to that of the neovascularization seen after ischemic central retinal vein occlusion. The straight anterior direction of the new vessels may be due to the vitreous scaffold having been compressed centrally by the schisis cavities, or alternatively to regrowth of vessels through the Cloquet canal. The tight vitreoretinal adhesions in an infant typically prevent the development of a partial or complete posterior vitreous detachment, which would also prevent growth of neovascularization on the back surface of the hyaloid cortex.

This patient had almost complete clearing of the retinal hemorrhage in the involved eye, and normally would have been seen at less frequent intervals (given the poor cortical prognosis for vision), except that the caregivers had a high level of concern about the vitreous hemorrhage and the status of the retina in the right eye and desired frequent reevaluation. With a longer follow-up interval, it is possible that the optic disc neovascularization could have progressed to ruberosis irides prior to detection. This case suggests that infants with massive retinoschisis and saturated hemorrhage due to shaking injury should be examined at least monthly for several additional visits after clearing of the retinal hemorrhage to monitor for the development of neovascularization.

Sandra M. Brown, MD
Michel Shami, MD
Lubbock, Tex

Corresponding author: Sandra M. Brown, MD, Department of Ophthalmology and Visual Sciences, Texas Tech University Health Sciences Center, Sixth and Flint streets, Lubbock, TX 79430 (e-mail: eyesmb@ttuhsc.edu).


Morning Glory Disc Anomaly in Neurofibromatosis Type 2

Neurofibromatosis type 2 (NF 2) is an autosomal dominant disorder characterized by a propensity for the development of vestibular schwannomas and other tumors of neural coverings (meningiomas, ependymomas) within the central nervous system. Ophthalmological findings in NF 2 include juvenile lens opacities,1 epiretinal membranes,2 combined pigment epithelial and retinal hamartomas,3 and disc gliomas.4 We describe 2 patients with NF 2 who had morning glory disc anomalies.

ARCH OPHTHALMOL/VOL 117, JUNE 1999
839
Report of Cases. Case 1. An 11-year-old boy with a history of strabismus and poor vision in the right eye since birth, was examined and was found to have a dysplastic optic disc in the right eye. Computed tomographic scanning showed multiple intracranial tumors including bilateral vestibular schwannomas and a large meningioma occupying the supra- sellar cistern and extending along the sphenoid wing into the right orbit. Ophthalmological examination showed no abnormalities in the left eye. The right eye was enophthalmic and had no light perception, no direct pupillary response to light, and severely decreased range of ductions in all directions. Slitlamp biomicroscopy disclosed a posterior subcapsular lens opacity in the right eye. Retinal examination revealed a depigmented, dysplastic lesion involving the peripapillary and macular region. (Figure, left). The margins of the optic disc were difficult to identify. A white tuft of glial tissue obscured the central portion of the disc. A red mass, presumably a vascular hamartoma, was situated at the superior temporal region of the disc. The nasal retinal vessels were anomalous, increased in number, and ran an abnormally straight course over the peripapillary retina. The temporal retinal vessels had a normal configuration. Ultrasonography showed a flattened contour of the peripapillary retina and mild elevation of the disc, with no excavation or calcification.

Case 2. A 2-year-old boy with exotropia was noted to have a macular scar in the right eye and a morning glory disc anomaly in the left eye. Reexamination at 6 years of age disclosed 2 inferior retinal holes in the left eye that were successfully treated with cryotherapy. At 13 years of age his corrected visual acuity was 20/200 OD and 20/25 OS. Retinal examination in the right eye disclosed that the macular scar represented a combined pigment epithelial and retinal hamartoma. In the left eye, a partly pigmented, non-excavated ring of yellow-white subretinal tissue surrounded an elevated optic disc (Figure, right). A large, white, glial tuft covered the central portion of the disc. The superior retinal vessels appeared increased in number and ran an abnormally straight course over the peripapillary retina. The inferior retinal vessels appeared to originate from the periphery of the disc but were otherwise normal in size and configuration. Two small peripapillary arteriovenous communications at the 4-o’clock position and at the 7-o’clock position were present, along with a third that crossed the horizontal raphe temporal to the macula.

At age 17 years, a computed tomographic scan disclosed bilateral acoustic neuromas. Magnetic resonance imaging showed bilateral oculomotor schwannomas, multiple spinal tumors, and no enlargement of the retrobulbar optic nerves. At age 20 years, he developed a rhegmatogenous retinal detachment in the left eye which was surgically repaired.

Comment. The morning glory disc anomaly is a congenital, funnel-shaped excavation of the posterior fundus that surrounds and incorporates an enlarged, dysplastic optic disc. A wide annulus of chorioretinal pigmentary disturbance surrounds the disc and forms the wall of the excavation. A white central glial tuft overlies the disc. The retinal vessels arise from the periphery of the disc and run an abnormally straight course over the peripapillary retina.

The morning glory disc anomaly is rarely associated with genetic disorders. Both patients described in this case report had NF 2 with morning glory disc anomalies that shared the following atypical features: (1) no excavation of the peripapillary fundus; (2) minimal enlargement of the optic disc; (3) an unusually large depigmented ring of dysplastic tissue surrounding the disc; and (4) some preservation of the normal retinal vasculature. These atypical findings indicate that the pathogenesis of the morning glory appearance in NF 2 may be different than in other individuals with the isolated anomaly.

Case 2 had been previously reported in this ARCHIVES for the
association of multiple retinal arteriovenous communications with a morning glory disc anomaly, when significance of the contralateral fundus lesion—a combined pigment epithelial and retinal hamartoma in a patient with NF 2 —was unrecognized. The finding of a morning glory disc anomaly in a patient with hearing loss, juvenile cataracts, or an unexplained retinal lesion should prompt craniospinal neuroimaging to look for occult tumors.

Corresponding author: Klara Landau, MD, UniversitätsSpital Zürich, Augenklinik, Frauenklinikstr 24, CH-8091 Zürich, Switzerland (e-mail: Landau@opht.unizh.ch).