performed a comprehensive immunohistochemical comparison of the vascular growth factors and hormonal markers in angiomatous and atypical meningiomas as well as hemangiopericytomas. They demonstrated evidence of hormonal receptors to act only in angiomatous meningiomas, and to muscle actin only in hemangiopericytomas. In addition, they showed a complete absence of VEGF-1 in the extravasates obtained from hemangiopericytomas. This case further validates their findings by demonstrating the presence of VEGF-1 and VEGF-2 as well as vimentin in the specimen. Therefore, in the absence of clear morphological differences, in certain cases the clinical distinction between vascular tumors and highly vascularized meningiomas can be difficult; one may need to rely on histochemical analysis.

This case is unique because primary orbital meningiomas not arising from the optic nerve are exceedingly rare. Although somewhat controversial, it is believed that a subpopulation of orbital meningiomas arises from within the orbital tissues, unrelated to the walls or the optic nerve. In a review of several cases of free-floating orbital meningiomas, McMichael and Cullen proposed various possibilities as the origin of these tumors: (1) that the tumor is originally attached to the optic sheath but later migrates away from it; (2) that the tumor cells arise from the orbital interstitial tissues; (3) that the tumor cells arise from sutures between orbital bones that contain herniated tumor cells; or (4) that the cells originate from the dura mater lining the orbit. Because of their peculiar behavior and location, these orbital meningiomas are often misdiagnosed as hemangiomas or dermoid cysts.

In an attempt to find other diagnostic differentiating clues, Utley at al. reviewed several cases of intracranial atypical meningiomas as compared with hemangiopericytomas. They discovered that on angiography, hemangiopericytomas in particular demonstrated a major vascular supply from the branches of the internal carotid or vertebral artery. However, they were unable to show clear differences on magnetic resonance imaging or computed tomography. Therefore, it is doubtful that radiologic clues alone can truly aid in the diagnosis of this condition.

This case is especially unique in that the histologic examination showed areas completely devoid of tumor and an uninvolved optic nerve sheath. On further review, the histologic findings are consistent with a primary angiomatous meningioma of the orbit, a previously unreported entity. We conclude that in cases of atypical behavior of an otherwise classic vascular tumor of the orbit, one should consider the possibility of an angiomatous meningioma.

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Nonarteritic Anterior Ischemic Optic Neuropathy Associated With Acute Glaucoma Secondary to Posner-Schlossman Syndrome

The occurrence of nonarteritic anterior ischemic optic neuropathy (NAION) has been well described following cataract surgery. It has been postulated that a periprogressive increase in intraocular pressure leads to a decrease in optic nerve head perfusion pressure, which results in ischemia of the optic disc. Acute rises in pressure secondary to other causes could also result in NAION. The lack of cases in the literature reporting NAION following episodes of acute glaucoma is thus surprising. Although vision loss in acute glaucoma has always been thought to be secondary to optic nerve head ischemia, to our knowledge, NAION following acute glaucoma has only been described in one report. We report a case of NAION occurring during an episode of acute glaucoma secondary to Posner-Schlossman syndrome.

Report of a Case. A 71-year-old white woman with a history of hypertension and hypercholesterolemia sought treatment for a 1-week history of intermittent left eye pain and decreased vision. Results of a right eye examination were normal. Initial visual acuity was 20/200 OS, and a relative afferent pupillary defect was present in the left eye. Corneal edema and trace anterior chamber cell and flare were noted. Intraocular pressure was 69 mm Hg. Gonioscopy revealed grade IV angles with light trabecular meshwork pigmentation and no peripheral anterior synchiae.

A dilated fundus examination revealed a mildly edematous left optic nerve and a crowded right optic nerve with a cup-disc ratio of 0.2. Humphrey visual field tests detected an inferior altitudinal defect in the left eye. The erythrocyte sedimentation rate was 16 mm/h. Once the pressure was controlled, the patient had no further complaints of eye pain. The patient had no family history of stroke at a young age, blood clots, unexplained loss of vision, or glaucoma. The patient denied having other neurologic symptoms, including symptoms suggestive of giant cell arteritis. Results of tests for homocysteine, anticardiolipin antibody, lupus anticoagulant, rapid plasma reagin, fluorescent treponemal antibody absorption, and antinuclear antibody were all within reference ranges. Left NAION was diagnosed. The patient was advised to take an aspirin daily.

The patient’s intraocular pressure was acutely controlled with eye
drops to treat glaucoma and oral acetazolamide. After resolution of the corneal edema, the patient’s vision improved to 20/20. During the next year our patient experienced recurrent episodes of elevated intraocular pressure and was diagnosed as having Posner-Schlossman syndrome. There was no further visual field loss associated with her subsequent attacks of elevated pressures and no evidence of recurrence of NAION. Her visual field deficit showed mild improvement at the 1-month visit but remained stable thereafter. Two months later, her left optic nerve showed mild temporal pallor and resolved edema.

Comment. Perfusion of the optic disc is directly proportional to mean arterial pressure and inversely proportional to intraocular pressure. In our case, the presumed mechanism causing NAION was decreased perfusion to the optic disc secondary to the rise in intraocular pressure. In addition, our patient had a small cup-disc ratio, which implies crowding of the nerve fibers as they pass through a smaller scleral canal. This renders the disc more susceptible to fluctuations in perfusion pressure. Recurrence of NAION in the same eye is rare, probably because after the initial episode, a reduction in the number of nerve fibers results in decompression of the crowded disc. Interestingly, this may explain why our patient had no further detectable visual field loss with recurrent rises in intraocular pressure. This case further suggests that the mechanism of NAION is related to decreased perfusion of the optic nerve. It also illustrates the importance of pressure control in all cases to prevent NAION.

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Traumatic Retinal Break From Viscoelastic Cannula During Cataract Surgery

The overall incidence of retinal detachment after extracapsular cataract extraction is between 1% and 2%. Retinal detachment after cataract surgery usually occurs after a posterior vitreous detachment creates one or more retinal tears in the region of the vitreous base. In rare cases, direct surgical trauma to the posterior segment can cause a retinal break and subsequent retinal detachment. To our knowledge, this is the first report of a retinal tear secondary to direct trauma from a cannula used to inject viscoelastic material.

Report of a Case. A 59-year-old woman was referred to the Retina Service at the Scheie Eye Institute, Philadelphia, Pa, for evaluation after cataract surgery. The patient had undergone extracapsular cataract extraction by phacoemulsification the previous day. After the cataract was extracted, a syringe containing the viscoelastic hyaluronate sodium with an appropriate-sized cannula tip was inserted into the eye. While the viscoelastic was being injected into the capsular bag, the cannula was forcefully ejected from the syringe. The cannula pierced the posterior capsule of the lens centrally (Figure) and drove inferiorly into the posterior segment, where it directly struck the retina. The cannula was removed, an anterior vitrectomy was performed, and a posterior chamber intraocular lens was placed. The clear corneal incision was left unsutured.

On postoperative day 1, the patient noted floaters in the right eye. Her visual acuity was 20/40 and the intraocular pressure was 23 mm Hg. Slitlamp examination was notable for a Seidel-negative clear corneal incision, minimal anterior segment inflammation, and a well-centered posterior chamber intraocular lens. A few pigmented cells were present in the vitreous.

The patient was referred to our service because the posterior segment examination showed a blood clot that emanated through a peripheral retinal break inferonasally and an operculated hole at the 9-o’clock meridian. The posterior pole was normal. The retinal breaks were treated with laser retinopexy.

Six weeks later the patient had a visual acuity of 20/20. The preretinal hemorrhage was almost completely resorbed, and no new retinal breaks or tears were seen. The retinopexy surrounded both retinal tears with moderate pigmentary response; no subretinal fluid was present. The patient retained 20/20 visual acuity and was without complication at 12 months of follow-up.

Comment. Iatrogenic retinal breaks occur rarely during cataract sur-

Red reflex photograph demonstrating laceration of the posterior capsule.