Interferon alfa Therapy Against Metastatic Iris Tumor of Renal Cell Carcinoma

Ocular metastasis of renal cell carcinoma is rare, and metastasis to the iris or ciliary body is especially uncommon. Ferry and Font1 studied 227 cases of metastatic tumors of the eye and orbit and reported that 26 (11.5%) of these lesions were iris or ciliary body tumors and that 2 (0.9%) originated in the kidney. We examined a patient with an iris tumor that metastasized from a renal cell carcinoma and observed marked effects of interferon alfa administration.

Report of a Case. A 55-year-old man was seen on September 3, 1992, with a 1-week history of decreased vision in his right eye. His history included radical right nephrectomy for renal cell carcinoma in December 1990 and left partial pneumonectomy due to lung metastasis in June 1992.

The corrected visual acuity was 20/20 OU and the intraocular pressure was 11 mm Hg OU. Slitlamp biomicroscopy showed cells in the anterior chamber and a well-circumscribed solid tumor (8 × 4 mm) in the iris of the right eye (Figure 1). The mass was vividly red and contained prominent internal blood vessels. Satisfactory visualization of the entire right fundus was not possible because of insufficient mydriasis, but a retinal detachment was noted in the periphery between the 3- and 7-o’clock positions. B-scan ultrasonography revealed a ciliary body mass at the inferonasal part of the ciliary body. No abnormality was noted in the left eye.

Systemic examination revealed swelling of the cervical lymph nodes, and results of computed tomographic scans and magnetic resonance images of the head disclosed a cerebral tumor in the right occipital lobe. Biopsy of the cervical lymph nodes established lymph node metastasis of the renal cell carcinoma (Figure 2). Because neurological symptoms such as disturbance of consciousness appeared thereafter, the brain tumor was resected on October 8, and the tumor was confirmed histopathologically to be a metastasis of the renal cell carcinoma.

Systemic interferon alfa was started on October 23 for conservative treatment of the iris tumor. The dose was set initially at 21,000,000 U/wk, but it was reduced to 9,000,000 U/wk in the second week due to bone marrow suppression (decrease in white blood cells and platelets) and to 6,000,000 U/wk in and after the third week. The iris tumor began to regress about 3 weeks after the beginning of interferon alfa administration, and it appeared totally regressed and white after 16 weeks (Figure 1). The retinal detachment disappeared, and the ultrasonographically demonstrated tumor in the ciliary region was not detected. However, the tumor metastasized to the lung, liver, and bones thereafter, and the patient died on June 20, 1993.

Comment. Renal cell carcinoma accounts for 80% to 85% of the malignant tumors of the kidney.2 Metastases were reported to be present in 25% to 30% of patients with renal cell carcinoma at the initial examination.2 Metastases of renal cell carcinoma are hematogenous and lymphogenous, and the lungs are the most frequent site of the distant metastases, followed by metastases to bones, liver, and brain.2 Metastases to the eye are rare, and those to the iris have been documented in only a few cases, including those reported by Ferry and Font1 and Wyzinski et al.3 The iris tumor of our patient appeared vividly red through the biomicroscope (Figure 1). This color has been recognized by some clinicians as characteristic of many metastatic ocular tumors from re-

Figure 1. Left, A well-circumscribed solid tumor, which appeared vividly red and contained prominent internal blood vessels, is observed on the iris between the 3- and 7-o’clock positions. Right, The tumor appeared to be totally regressed and white 16 weeks after the beginning of interferon alfa administration.
nal cell carcinoma. Our patient was treated conservatively because no reduction of vision or secondary glaucoma due to the tumor was noted, and because the prognosis was poor due to multiple organ metastases.

Renal cell carcinoma resists routine chemotherapy and radiotherapy. However, since interferon alfa has recently been reported to have a direct antiproliferative effect on renal tumor cells in vitro, and to stimulate host mononuclear cells and enhance the expression of major-histocompatibility-complex molecules, it has been introduced as a new treatment for renal cell carcinoma. More recently, the effects of interferon alfa when used in combination with interleukin 2 and fluouracil, or with 13-cis-retinoic acid, have been evaluated. The response rate of patients to interferon alfa alone is considered to be about 20%, and its therapeutic effect is limited when it is used alone. However, as some patients such as ours show marked responses, interferon alfa may be useful in patients with intraocular metastases of renal cell carcinoma.

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Clear Corneal Cataract Wound Dehiscence During Pneumatic Retinopexy

Pneumatic retinopexy is generally accepted as a safe and effective treatment for certain types of retinal detachment. This procedure recently has been demonstrated to produce equivalent final visual outcomes and reattachment rates compared with scleral buckling for primary rhegmatogenous retinal detachment. The growing popularity of this technique was verified in a recent survey of members of the Retina and Vitreous Societies that revealed that pneumatic retinopexy is the treatment of choice for “uncomplicated” retinal detachments. Factors influencing the selection of this modality by vitreoretinal specialists included number of years since completion of clinical training and complexity of the detachment. Complications of pneumatic retinopexy have been well described and involve the anterior and posterior segments. These include: cataract, choroidal detachment, delayed resorption or shifting of subretinal fluid, endophthalmitis, pars plana/subconjunctival/subretinal gas, malignant glaucoma, peripheral subretinal hemorrhage, vitreous hemorrhage, iris incarceration, macular hole/pucker, neck pain, subretinal/vitreous pigment, proliferative vitreoretinopathy, refractive changes, untreated retinal breaks, uveitis, vitreous incarceration/loss, and central retinal artery occlusion. Self-sealing, clear corneal incisions for cataract extraction have become increasingly popular recently owing to the combined advantages of phacoemulsification, foldable intraocular lenses, and topical anesthesia. Wound strength was one of the early criticisms of this technique, although studies suggest that the integrity of small incision, clear corneal incisions is comparable with traditional limbal or scleral based incisions. Typically, self-sealing clear corneal incisions are reinforced via stromal hydration at the end of the procedure, and sutures can be used to enhance wound integrity.

The 2 cases in which clear corneal incisions from recent cataract surgery dehisced during pneumatic retinopexy. To our knowledge, this potentially dangerous complication of pneumatic retinopexy has not been reported previously. However, it may become increasingly common given the growing popularity of clear corneal cataract extraction and the increased risk of retinal detachment imparted by intraocular surgery and pseudophakia.

Report of Cases. Case 1. A 49-year-old white man underwent clear corneal cataract extraction and posterior chamber intraocular lens implantation in the left eye on June 11, 1999. The procedure was complicated by a small tear in the posterior lens capsule at the 4-o’clock position, which did not require an-
terior vitrectomy. The corneal wound was closed with a single 10-0 polyglactin (Vicryl) suture, and the wound was watertight at the conclusion of the operation. The postoperative course was uneventful until June 23, 1999, when the patient was diagnosed with a single retinal break at the 12:30-0’clock position and a macula-on, rhegmatogenous retinal detachment in the left eye supertemporally. Intraocular pressure (IOP) was noted to be 15 mm Hg. Topical and subconjunctival anesthesia were administered, and cryopexy was applied to the breaks. The 10-0 polyglactin suture placed at the conclusion of the cataract extraction was absent from the clear corneal wound. Pneumatic retinopexy then was performed using 0.3 cc of 100% sulfahexafluoride injected through the pars plana. During the gas injection, the clear corneal wound dehisced and aqueous fluid was ejected forcibly enough to impact the surgeon’s face at a distance of approximately 2 ft (60.9 cm). No prolapse of ocular structures occurred, and the dehisced wound was allowed to self-seal without sutures. The postoperative IOP was 16 mm Hg. On September 4, 1998, the persistent retinal detachment was reattached successfully via pars plana vitrectomy, cryopexy, scleral buckling, fluid gas exchange, and endolaser treatment.

**Comment.** We reported 2 cases of a previously unreported complication of pneumatic retinopexy causing dehiscence of clear corneal incisions from recent cataract extractions. Despite the potential seriousness of this complication, these cases resolved without incident. Among the previously described complications of pneumatic retinopexy, central retinal artery occlusion also results from high IOPs created during this procedure. As suggested by Abe et al, such complications could be minimized via routine IOP monitoring and pars plana vitrectomy.

Both cases demonstrated wound dehiscence less than 3 weeks after cataract surgery. More cases of this complication will be required before a reliable estimate can be made of the length of time sufficient for such wounds to become fully stable for pneumatic retinopexy. Given the increasing popularity of clear corneal cataract extraction and the elevated risk of retinal detachment imparted by cataract extraction and intraocular lens implantation, this complication may be observed with greater frequency in the future. To reduce the potential for clear corneal wound dehiscence during pneumatic retinopexy, paracentesis may be performed prior to gas injection to minimize a rapid increase in IOP. Alternatively, smaller volumes of injected gas may be used for pneumatic retinopexy performed in the setting of recent clear corneal cataract extraction.

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**Bilateral Scleral Thermal Injury: Complication After Skin Laser Resurfacing**

In 1980, Beckmann and Fuller were the first to use the carbon dioxide (CO2) laser in blepharoplasty surgery. Reported advantages of CO2 laser blepharoplasty include decreased postoperative edema, less pain, and a shorter convalescence in comparison with conventional surgery. We are aware of only 1 report on the use of a combined CO2/Nd:YAG laser: in 1996, Katalinich reported that 50 patients treated for cutaneous neurofibromas had shorter surgery times, less hemmorhages, and sufficient removal with the Nd:YAG laser vs with the CO2 laser alone. To the best of our knowledge, complications of this treatment have not yet been described.

Report of a Case. A 45-year-old man with Recklinghausen neurofibromatosis had undergone laser resurfacing because of multiple facial neurofibromas, especially in the periorbital region. He was treated in a plastic surgeon’s office using, as far as we know, a combined CO2/Nd:YAG laser (Combolaser; Madtec GmbH, Ulm, Germany) (power settings: CO2 laser, 20–25 watts [W]; Nd:YAG laser, 7-8 W). The technique has been described by Katallinich: the skin over the tumor is dissected, then the tumor is evaporated by a defocussed laser beam. The use of 2 laser types is supposed to remove the neurofibromas radically enough. Five days after treatment, the patient was referred to our hospital suffering from deteriorated vision in the left eye. Best-corrected visual acuity was 1.0 OD and 0.16 OS. In both eyes, left worse than right, slitlamp microscopy revealed broad-based defects of the lower eyelid margins (small arrows) with corresponding conjunctiva-covered scleral lesions (large arrows), resembling a thermal injury (Figure). The left eye showed slight intraocular inflammation. No injury of other anterior segment structures was observed in either eye. Intraocular pressure was 12 mm Hg OU. The swinging flashlight test showed no afferent papillary defect. Funduscopy was normal in the right eye; in the left eye, it revealed discrete optic nerve head swelling, with peripapillary bleeding and angiographically proven peripapillary exudation, which explained the transient visual acuity loss. In kinetic perimetry, a relative defect to the right side was observed. Systemic and neurological inflammatory diseases were excluded by clinical and laboratory investigations and magnetic resonance imaging. Visual acuity recovered with high-dose systemic corticosteroids, beginning with 100 mg of fludroco- lone daily and tapering off over 6 weeks. Seven months after the laser injury, visual acuity was 1.0 OU. Slitlamp microscopy revealed scarred scleral lesions in correspondence with scarred lower eyelid margin defects in both eyes. Macula and optic nerve head were normal in both eyes. A second fluorescence angiogram was refused by the patient.

Comment. In a safety study, plastic and metal corneal eye protectors were tested with CO2 laser beams of different energy levels to assess flammability and heat production: only the metal protectors showed no damage. Therefore, metal scleral eye protectors are recommended for laser treatment in the periorbital region. Although the plastic surgeon insists that he took adequate measures to protect the globe, some doubts remain about whether these recommendations were considered properly in the case of this patient. To find out which laser source had caused which type of damage, we used a Monte-Carlo calculation (S. William, PhD, A. Terenji, PhD, unpublished data, December 1998) to estimate the laser intensity (watts per square centimeter) in different tissues of the human eye when
either a CO₂ or a Nd:YAG laser is employed (Table). This calculation shows that nearly all the energy of the CO₂ laser is absorbed in the sclera, whereas the energy of the Nd:YAG laser easily penetrates the sclera and is able to damage the posterior pole. We therefore conclude that the scleral lesions in our patient were most probably caused by a CO₂ laser, whereas the posterior pole damage in the left eye must have been caused by a Nd:YAG laser. Penetrations of the globe after CO₂ laser treatment have been reported anecdotally but have not been published so far (J. J. Woog, written communication, June 29, 1998).

In conclusion, severe ocular complications caused by CO₂ or Nd: YAG laser treatment can occur if eye protection is inappropriate. Therefore, safety considerations should be observed carefully.

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Sutureless Pars Plana Anterior Vitrectomy Through Self-sealing Sclerotomies in Children

Intraocular lens implantation in children has become increasingly common after cataract surgery with encouraging visual results. However, thickening of the posterior capsule along the visual axis remains a common and major problem that can lead to irreversible amblyopia. Laser capsulotomy is not always possible for pediatric patients and surgical removal is often necessary. The thickened membrane can be approached anteriorly through the limbus or posteriorly through the pars plana. A limbal approach can render the intraocular lens unstable and the pars plana approach is usually more difficult owing to the lower scleral rigidity in children. It is more common for the eyeball to collapse with leakage through the wounds intraoperatively. Sutureless pars plana vitrectomy through self-sealing sclerotomies has been successfully performed in adults. This study aimed at evaluating prospectively the safety and efficacy of sutureless pars plana anterior vitrectomy through self-sealing sclerotomies in children with thick posterior pseudohypoplastic membrane.

Report of a Case. A total of 8 eyes in 5 children were recruited from the Prince of Wales Hospital, Hong Kong, China, from March 1, 1998, to December 31, 1998. The children’s mean age was 22 months (age range, 8-48 months) and the follow-up time ranged from 3 to 12 months (mean follow-up, 9 months). All patients had congenital cataract with cataract extraction performed by phaco-assisted aspiration. Primary posterior chamber intraocular lens was inserted with subsequent thickening of the posterior capsule along the visual axis deemed necessary for surgical removal. A 2-port pars plana approach was adopted as illumination was adequate from the operating microscope. Self-sealing sclerotomies were constructed in the same fashion as those described by Chen. Only the 2 superior sclerotomies were necessary, one for irrigation and the other for the vitreous cutter, avoiding the inferior sclerotomy that was often the main source of leakage as reported by Milibak and Suveges. Scleral tunnels of 2 mm long were created 3.5 to 4.5 mm posterior to the limbus and the actual sclerotomy sites were 1.5 to 2.5 mm from the limbus depending on the age of the patient. The integrity of the wound closure was tested by inspection and none of the sclerotomy sites required suturing. No intraoperative or postoperative complication was encountered.

Comment. Although the scleral rigidity in children is lower, the self-sealing effect of this technique was good with the integrity of the eyeballs well maintained. Sudden changes in intraocular pressure during operation are reduced, minimizing complications like intraoperative hemorrhage, vitreous herniations, and others. Suture-related problems, such as loosening, exposure, and infections, are also avoided. This would have been more difficult to manage in children where extra sessions under general anesthesia may become necessary. The exposure for surgery in younger children could be suboptimal; scleral tunnels were created without difficulty 4.5 mm posterior to the limbus in our cases. This can be further optimized by the modified technique described by Chen and Kwok et al for eyes with small palpebral fissures. The sutureless sclerotomy approach appears to be safe and effective and with its additional values in pediatric patients, it can be considered in selected cases.

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3. Quinn GE, Young TL. Retina and vitreous. In:
Retinal Artery Occlusion Following Coil Embolization of Carotid-Ophthalmic Aneurysms

Paraclinoid aneurysms, such as carotid-ophthalmic aneurysms, represent 5% of all intracranial aneurysms. In the past, treatment involved surgical clipping of the aneurysm, which is associated with 4% morbidity and 1% mortality even in elective cases. A nonsurgical alternative treatment for intracranial aneurysms was developed in 1990 that utilizes detachable coils placed by an endovascular route to embolize the aneurysm and induce thrombus formation within the aneurysmal sac. The procedure has the advantage of avoiding the risks of neurosurgery and enabling treatment of aneurysms that are unclippable or otherwise associated with a high morbidity. However, cerebral embolization can complicate this procedure. We report 2 cases of retinal artery occlusion following coil embolization of nonruptured carotid-ophthalmic aneurysms.

Report of Cases. Case 1. A 54-year-old man had a subarachnoid hemorrhage on November 1996 secondary to rupture of a left carotid-ophthalmic aneurysm that was successfully embolized with coils. He also had a small right carotid-ophthalmic aneurysm measuring 4 mm in diameter that was intact and not treated. The patient had full recovery and demonstrated no neurological deficits. In January 1999, he underwent coil embolization of the right paraclinoid aneurysm. While heparinized, there was perforation of the dome of the aneurysm during the procedure. The aneurysm was completely packed with coils and the heparin was reversed. A few hours after the procedure, the patient complained of blurred vision in his right eye. Visual acuity was initially 20/20 OU, and the fundi showed no signs of ischemia. Confrontation testing showed constriction of the right visual field. Eight hours later, visual acuity deteriorated to hand motions OD and a relative afferent pupillary defect was noted. Fundus examination revealed a central retinal artery occlusion in the right eye (Figure 1). Cerebral angiography showed that the coils were in satisfactory position occluding the aneurysm and that the right ophthalmic artery was patent. Because of perforation of the aneurysm and the potential risk of disturbing the coils while trying to catheterize the ophthalmic artery, thrombolytic therapy was contraindicated and intravenous heparin was given instead. Topical timolol and intravenous acetazolamide were administered, an anterior chamber tap was performed, and the eye was massaged, but there was no visual recovery. His visual acuity remained hand motions OD in the remaining temporal field 4 months later.

Case 2. A 13-year-old girl with tuberous sclerosis was found to have an intracranial aneurysm of the right carotid artery on magnetic resonance imaging. Angiography showed that the aneurysm was located in the ophthalmic branch and measured 8.6 mm in diameter with a wide neck (Figure 2). The patient had no focal neurological deficits at the time, and the aneurysm was embolized with Guglielmi detachable coils. The aneurysm was 95% occluded, but there was some coil protrusion into the parent artery. Anticoagulation with intravenous heparin was administered for 48 hours in hopes of preventing thrombus formation. Shortly after discontinuation of heparin therapy, the patient complained of 2 episodes of inferior visual field loss in the right eye. Visual acuity was 20/20 OU, but confrontation visual field testing demonstrated an inferonasal field defect in the right eye. No relative afferent pupillary defect was noted. Fundus examination showed whitening of the retina along the superotemporal arcade with sparing of the fovea consistent with a branch retinal artery occlusion (Figure 3). Angiography was performed to determine if thrombus was present in the carotid artery, but none was found. The position of the coils remained unchanged. The patient was again given anticoagulation therapy with intravenous heparin. Because of the presence of an incompletely occluded aneurysm, coil protrusion, and an embolic episode, she underwent surgical removal of the coil and clipping of the aneurysm 4 days after the initial procedure. Visual acuity remained 20/20 OD postopera-

Figure 1. Fundus photograph of the right eye 2 weeks after central retinal artery occlusion and after coil embolization of the paraclinoid aneurysm. Note the diffuse macular edema and the irregular attenuated appearance of the arterioles due to hypoperfusion of the retina. The classic cherry-red spot seen in the acute phase of central retinal artery occlusion has evolved to formation of hard exudates and intraretinal and subretinal hemorrhage in the macula at this time.

Figure 2. Fundus photograph of the right eye 11 months after coil embolization of a carotid-ophthalmic aneurysm. Note the irregular configuration of the optic disc due to chronic compression by the coils, profound chorioretinal atrophy, and the atrophic retina. The patient had no visual loss.

Figure 3. Fundus photograph of the right eye 5 days after surgical removal of the coil and clipping of the aneurysm. Note the markedly atrophic macula and characteristic geographic atrophy consistent with retinal arterial occlusion. The patient had no visual loss.
tively, and the inferonasal field defect persisted.

Comment. Cerebrovascular ischemia from embolism is a known complication of coil embolization of cerebral aneurysms. In one large case series involving 403 patients, Vinuela et al reported an incidence of 2.5%. The complication of retinal artery occlusion following this procedure, however, has not yet been reported in the literature to our knowledge. These 2 cases represent the only ophthalmic embolic complications of 71 paraclinoid carotid aneurysms that have been treated with detachable coils at our institution. In both cases, the ophthalmic arteries were patent at angiography and the retinal artery was too small to be visualized. The microemboli that caused the retinal artery occlusion presumably arose from the thrombus that formed around the coils in the aneurysm. Visual loss following surgical clipping of paraclinoid aneurysms has been reported, but the cause has been considered to be improper placement of clips on the ophthalmic artery, direct trauma to the nerve, or ischemia from vasospasm. Retinal artery occlusion, either branch or central, can be a devastating complication. Ophthalmologists and interventional neuroradiologists should be aware of this complication so that prompt treatment may be given. However, in the context of a recently coiled paraclinoid aneurysm associated with subarachnoid hemorrhage, we consider fibrinolysis too risky and we only administer anticoagulation therapy to the patient. In elective cases, it is prudent to perform an ophthalmic examination with perimetry prior to treatment.

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Mucosal Leishmaniasis Presenting as Sinusitis and Optic Neuropathy

Leishmaniasis, caused by the dismorphic protozoa Leishmania, is typically observed in patients as 1 of 3 clinical syndromes. Cutaneous leish-
Leishmaniasis with chronic nonhealing ulcers that involve the face and extremities is the most common manifestation. Mucosal leishmaniasis invades the nose, oral cavity, and pharynx. Visceral leishmaniasis is characterized by fever, wasting, hepatosplenomegaly, and pancytopenia. To date, the only ocular problem has been eyelid involvement secondary to adjacent facial cutaneous leishmaniasis. To date, the only ocular problem has been eyelid involvement secondary to adjacent facial cutaneous leishmaniasis. To date, the only ocular problem has been eyelid involvement secondary to adjacent facial cutaneous leishmaniasis. To date, the only ocular problem has been eyelid involvement secondary to adjacent facial cutaneous leishmaniasis.

**Report of a Case.** A 45-year-old man was hospitalized for evaluation of a right-sided headache of 4 weeks' duration and 1 week of progressive graying of vision in his right eye. His medical history was remarkable for human immunodeficiency virus infection for approximately 14 years and recent diagnosis of AIDS due to the development of pulmonary *Pneumocystis carinii* pneumonia and multidrug-resistant tuberculosis (CD4 cell count <20/µL).

Findings from neuro-ophtalmologic examination revealed a visual acuity of 20/50-2 OD and 20/20 OS. He could not see the control or the test plates of the Ishihara color plates test with his right eye, but he saw all the plates with his left eye. On tangent perimetry at 1 m he could only count fingers in all quadrants with his right eye, and with the left eye the field was normal to 2 mm white object. In the right eye, there was 2 mm of exophthalmos without ptosis, swelling, conjunctival hyperemia, or limitation of ocular motility. The pupils were round and reactive to light; the right pupil was less reactive with a relative afferent pupillary defect. Results of slit-lamp biomicroscopy and ophthalmoscopy were unremarkable.

Computed tomography demonstrated a sphenoid sinus lesion that extended into the right orbital apex through an area of bone erosion (Figure 1). Magnetic resonance imaging revealed diffuse opacification of the sphenoid sinus with areas of bright and decreased signals on T2-weighted images suggestive of fungal infection (Figure 2). Hematoxylin-eosin staining of the specimen obtained by endoscopic sphenoidectomy revealed intracellular organisms in the nasal and sphenoidal mucosa. Giemsa stain also demonstrated the intracellular organisms (Figure 3) while the Gomori methenamine-silver stain was negative for organisms. Gram stain did not show evidence of additional organisms. Fungal cultures of the biopsy specimen were also negative for organisms. A diagnosis of leishmaniasis was made and confirmed by the Centers for Disease Control and Prevention and the species identified as *Leishmania braziliensis*.

The patient was treated with intravenous Pentostam (a European orphan drug; sodium stibaglucanate) 1200 mg/d. Despite treatment, the vision in his right eye deteriorated to no light perception, and right third, fourth, and sixth cranial nerve palsies appeared. Additional computed tomography revealed progression of the process at the orbital apex. Simultaneously he developed a severe pneumonia caused by Aspergillus and 2 months following the diagnosis he was hospitalized for seizures and hemiparesis and died the next day. An autopsy was not permitted.

**Comment.** Leishmaniasis is caused by an obligate intracellular parasite that is frequently transmitted through the bite of infected sand flies from an animal reservoir. Other modes of transmission including parenteral, congenital, and sexual have been reported. Mucosal leishmaniasis develops in less than 5% of patients, typically months to years following localized cutaneous leishmaniasis. Most cases of mucosal leishmaniasis are associated with *L. braziliensis*, commonly referred to as American leishmaniasis. The process often starts in the nasal septum and results in perforation. The diagnosis is made by demonstration of parasites by Giemsa stain on biopsy specimens from affected tissue. Newer methods include antigen detection, polymerase chain reaction detection, and skin test. Mucosal leishmaniasis tends to be a chronic progressive disease that responds poorly to the treatment. Pentostam (20 mg/kg) is used as first-line therapy for leishmaniasis and amphotericin B (0.5-1 mg/kg) as second-line treatment. In patients...
with AIDS, the compromised immune status contributes to the dissemination of the infection and atypical presentation.\(^5\)

Patients with the human immunodeficiency virus commonly experience sinusitis, typically due to bacteria, but sinus infections and orbital involvement have been described with *Aspergillus* species, *Pseudallescheria boydii*, microsporidia, and *Alternaria* species. Non-Hodgkin lymphoma should also be considered in the differential diagnosis of sinus disease in a patient infected with human immunodeficiency virus.\(^6,7\) We could not find another case of sinusitis and optic neuropathy due to leishmaniasis. This case should alert ophthalmologists to the possibility of unusual infectious agents as the cause of orbital apex disorder in patients with AIDS.

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with the largest part anterior to the upper temporal arcades, and extended, with a smaller part posterior to the arcades (Figure 1). These lesions corresponded with the visual field defects.

In the left eye, we noted choroidal neovascularization at the inferior border of the sclerochoroidal calcifications, hemorrhages surrounding the membrane, and a single hemorrhage centrally in the area of the calcifications. The fluorescein angiography of the left eye showed hypofluorescence of the sclerochoroidal calcifications in the arterial phase. In the venous phase, a well-defined choroidal membrane was identified that was 1 disc area large. Moreover, progressive irregular fluorescence of the sclerochoroidal calcifications was noted, with window defects in areas with depigmentation of the retinal pigment epithelium, and with several hyperfluorescent lesions suspicious for small vascular tufts lying beneath the retina on the surface of the calcifications (Figure 2, left). In the late phases of the fluorescein angiogram, we noted intense hyperfluorescence of the sclerochoroidal calcifications in both eyes (Figure 2, right). Superior to the central hemorrhage and temporal to the neovascular membrane at the inferior border of the sclerochoroidal calcifications, we noted 2 well-demarcated zones of more profound depigmentation of the retinal pigment epithelium. These areas showed pronounced late hyperfluorescence and were suspicious for foci of subretinal neovascularization. After laser photocoagulation of the well-defined choroidal membrane, formation of a flat atrophic scar was observed. This scar enlarged during the 7-year follow-up period (Figure 3, right). The sclerochoroidal calcifications and the 2 foci suspicious for subretinal neovascularization remained unchanged and no new hemorrhages were noted.

In the right eye, we observed a similar aspect of the sclerochoroidal calcifications, with 1 more obvious lesion at the inferior border of the mass. This lesion was clinically atrophic but was associated with intense hyperfluorescence and with recurrent hemorrhages (Figure 3, left). The atrophic aspect of the lesion remained unchanged and we never diagnosed a choroidal neovascular membrane. However, we presume that a similar lesion has preceded the neovascular growth in the left eye.

Ultrasonography showed a mass with 100% spikes and progressive at-

Figure 1. Fundus photographic views at initial examination show superotemporal sclerochoroidal calcifications in both eyes. Note a hemorrhage located centrally and a rim of hemorrhages located inferiorly of the calcifications at the border of a subretinal membrane in the left eye. No hemorrhages were observed in the right eye.

Figure 2. Left, Venous-phase fluorescein angiogram of the left eye at initial examination demonstrates a well-defined subretinal neovascular membrane. Right, Late-phase angiogram of the same eye shows diffuse leakage from the membrane and staining of the sclerochoroidal calcifications. Note also the linear and pinpoint early hyperfluorescent lesions on the surface of the calcifications and profound late hyperfluorescence of a focus superior to the central hemorrhage, as well as another focus temporal to the neovascular membrane at the inferior border of the calcifications.
scan confirmed the presence of a large calcified mass in the posterior pole of both eyes (Figure 5). The sclerochoroidal calcifications remained nearly unchanged during the long follow-up period, with only a mild increase of the associated atrophic changes. A recent indocyanine green angiography was performed, with the aim to detect occult choroidal neovascularization at an early stage. The indocyanine green angiogram revealed patchy hypofluorescence in all phases in the areas with calcifications and atrophic changes. Moreover, there was a faint late hyperfluorescence at the borders of the mass. No peculiar hyperfluorescence was noted of the lesion with recurrent hemorrhages in the right eye. During the patient’s last examination, a bilateral cataract was noted, and visual acuity was 20/30 OU.

Our patient had a multinodular goiter and mild hyperthyroidism that were treated medically. Screening of calcium metabolism showed no disturbances and no arguments for primary hyperparathyroidism, but it was confirmed that the patient had secondary hyperparathyroidism caused by vitamin A deficiency, which is a common finding in elderly persons.

**COMMENT**

Sclerochoroidal calcifications are usually observed in the midperipheral fundus of elderly patients who are asymptomatic. The lesions are frequently bilateral and localized in the superotemporal quadrant. Two types of sclerochoroidal calcifications are identified: relatively flat...
plaque-like lesions and elevated tumorlike lesions, ranging up to 6 mm in height.3

Recently, a first case of sclerochoroidal calcifications with choroidal neovascularization was reported.5 In this woman (aged 74 years), bilateral sclerochoroidal calcifications were discovered during a routine examination. One plaque of calcifications was associated with lipid exudates and serous detachment. Fluorescein angiography was indicative of ill-defined choroidal neovascularization. Argon laser photocoagulation induced regression of serous detachment and exudates, and there was no recurrence during a 2-year follow-up (S. Y. Cohen, MD, personal communication, December 14, 1998).

We report the second case in the world literature of sclerochoroidal calcifications with a well-defined subretinal membrane and several foci with hemorrhages and hyperfluorescence suspicious for choroidal neovascularization. During the 7-year follow-up, these foci did not evolve to subretinal membranes and there was no recurrence of choroidal neovascularization from the well-defined membrane after laser treatment.

Several studies of series of patients with sclerochoroidal calcifications report an absence of choroidal neovascularization. Thirty-four cases have been studied by Sivalingam et al,1 Munier et al,2 and Schachat et al3 and no choroidal neovascularization was observed in their cases. With fewer than 50 reported cases without choroidal neovascularization, the incidence of this complication is probably extremely low. Moreover, sclerochoroidal calcifications do not expand close to the macular area, and the risk of visual loss because of associated choroidal neovascularization seems to be extremely low.

On the other hand, the case of Cohen et al5 and our experience show that serous detachment and exudative lesions may occur in the context of sclerochoroidal calcifications that are complicated by choroidal neovascularization, and that laser treatment may be effective in reducing the risk of developing additional visual field loss. In 2 cases treated with lasers, no recurrence of the choroidal neovascularization was observed.

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