2 was relatively solid but appeared hollow ultrasonographically. We advise that UBM be used for assessment of caruncular tumors, as it provides high-resolution imaging to submillimeter levels and can delineate tumor extent and configuration. However, we caution that both cystic and densely packed solid tumors can appear echolucent.

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Choroidal Neovascularization After Epiretinal Membrane Removal

Surgical removal of an epiretinal membrane (ERM) is a relatively common procedure, often resulting in significant visual improvement. Several complications, however, are well recognized, and include cataract formation, retinal breaks and detachments, cystoid macular edema, retinal phototoxicity, and endophthalmitis.1 Choroidal neovascularization (CNV) is a rare complication of ERM surgery, and, to our knowledge, only 3 cases have been reported in the literature.2,4 We report 3 additional cases of CNV after surgical removal of idiopathic macular pucters, and comment on the literature regarding this entity.

Report of Cases. Case 1. A 47-year-old man complained of increasing metamorphopsia in his left eye for 3 years. His ophthalmic history was notable for myopia of 7.75 diopters, cataract surgery in his left eye at the age of 40 years, and a small retinal tear in the nasal periphery in the left eye treated with a laser. His visual acuity (VA) was 20/60 OS, and the result of a fundus examination revealed a macular pucker (Figure 1A). Examination results were unremarkable in the right eye. The patient underwent pars plana vitrectomy and an uncomplicated ERM peel using fine forceps.

Four weeks postoperatively, the patient reported a central scotoma and blurred vision. Eight weeks postoperatively, his VA decreased to 20/800 OS and juxtafoveal CNV was noted on fundus examination and fluorescein angiography (FA) (Figure 1B and Figure 2A). The patient underwent pars plana vitrectomy, and the subretinal membrane was removed. Two weeks later, his VA was 20/125 OS and the results of a fundus examination revealed significant improvement (Figure 2B). However, 8 months postoperatively, the patient developed a rhegmatogenous retinal detachment requiring vitrectomy and silicone oil placement; his VA 2 years later was 20/500 OS.

Case 2. A 77-year-old man complained of decreased VA in his right eye for 2 years. His VA was 20/80 OD. The results of an ophthalmic examination revealed cataracts in both eyes, a mild ERM in his left eye, and an ERM associated with cystic retinal changes in his right eye. The patient underwent uncomplicated pars plana vitrectomy and an ERM peel in his right eye using Tano forceps (Synergetics Inc, Wilsonville, Ore). A trace intraretinal hemorrhage was noted inferotemporal to the fovea postoperatively.

Four weeks postoperatively, the patient’s VA improved to 20/50 OD. Because of a residual intraretinal hemorrhage, FA was performed, revealing no evidence of CNV.

Five months postoperatively, the patient developed metamorphopsia and his VA declined to 20/125 OD. The results of a fundus examination revealed an elevated grayish lesion temporal to the fovea. The FA showed classic extrafoveal CNV that was treated with laser photocoagulation. Three weeks following treatment, his VA was 20/100 OD. The FA showed no evidence of CNV. Seven weeks later, his VA remained stable at 20/100 OD.

Case 3. An 80-year-old man complained of deteriorating vision in his left eye 1 month after uneventful cataract extraction and intraocular lens implantation. His ocular history was significant for cataract extraction in his left eye and medically managed glaucoma in both eyes. The result of a fundus examination was unremarkable in his right eye and revealed a macular ERM and cystoid macular edema in his left eye, which was confirmed by FA. Eighteen months after cataract surgery, the cystoid macular edema had resolved; however, the patient’s VA remained poor, at 20/50+1 OS, secondary to the ERM. A pars plana vitrectomy and membrane peel using Tano forceps were performed. Minimal surface bleeding was noted during the procedure; 1 week later, a local intraretinal hemorrhage was seen nasal to the fovea and a preretinal hemorrhage was seen inferior to the fovea. One month postoperatively, the patient’s VA was 20/200 OS. The FA showed classic extrafoveal CNV that was treated with laser photocoagulation.
Three weeks following laser photocoagulation, the patient’s VA deteriorated further to 20/400 OS. The FA showed recurrence of subfoveal CNV, which was then treated with photodynamic therapy. The patient required 4 sessions of photodynamic therapy, 2, 5, 10, and 13½ months after ERM removal. The FA 17 months following surgery showed subretinal fibrosis with no evidence of leakage. At the 2-year follow-up, the patient’s VA remained poor, at 20/800 OS.

Comment. In this series, we report 3 cases of CNV that developed after idiopathic ERM removal. Three additional cases have been reported in the literature. The mean duration of the ERM before surgery in the 6 cases was 22.6 months (range, 5 months to 4 years). Choroidal neovascularization was diagnosed between 1 month and 2 years postoperatively. The mean preoperative VA was 20/65 (range, 20/50-20/200). The CNV complexes were predominantly classic and were initially extrafoveal or juxtafoveal. Management included surgical removal, photocoagulation, and photodynamic therapy. The mean follow-up after CNV treatment was 1 year (range, 4-24 months). The mean final VA was 20/270 (range, 20/40-20/800). The visual outcome was largely poor when the follow-up interval was longer, and variable at shorter follow-ups.

The cause of CNV and its relation to ERM surgery is not entirely clear. It is intriguing that 3 of the 6 patients developed intraretinal or preretal hemorrhaging in the perioperative period. While peeling the membrane from the macula, tractional forces transferred to the underlying retinal pigment epithelium and Bruch membrane could have resulted in a direct break or localized trauma reflected by areas of hemorrhaging. Causes for variable times of CNV onset postoperatively are uncertain, but may include the amount of induced trauma and the patient’s predisposition to developing CNV.

Another possible explanation is that subclinical CNV coexisted with the ERM when the patient was initially seen. Gass described 2 patients with idiopathic macular pucker who developed CNV.

Figure 1. For case 1, fundus photographs of when the patient was initially seen, showing an epiretinal membrane (ERM) with macular striae (A), and 2 months after ERM removal, showing elevation of the macula by a new and extensive subretinal membrane (B).

Figure 2. For case 1, a corresponding fluorescein angiogram 2 months after epiretinal membrane removal documents intense focal hyperfluorescence consistent with fibrovascular ingrowth in the nasal macula (A) and a fundus photograph 2 weeks after subretinal membrane removal shows an improved macular appearance (B).
years later and 1 patient who had an ERM and CNV on initial examination. However, the results of a fundus examination in our patients did not reveal any clinical signs of CNV before ERM surgery. In addition, 1 patient (case 2) underwent FA 1 month after ERM removal, which showed no hyperfluorescence in the macula.

We do not believe FA is necessary before performing ERM removal to rule out preexisting CNV. However, FA is valuable in examining patients postoperatively when they develop a decreased VA and metamorphopsia. Because of the limited number of reported cases, there is no consensus about the best treatment modality. Each patient should be examined and treated based on the CNV characteristics and available treatment options.

In summary, CNV is a rare complication of ERM surgery that is likely secondary to iatrogenic trauma that is transferred to the outer retina and Bruch membrane during ERM removal. Ophthalmologists who perform ERM surgery should consider CNV a possible cause of poor visual outcome in the early and late postoperative periods.

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### Plasmapheresis for Lupus Retinal Vasculitis

Systemic lupus erythematosus (SLE) is a potentially lethal, chronic autoimmune disease that can involve the eye. It is characterized by the production of numerous autoantibodies, some of which are pathogenic. Retinal vasculitis is the most serious of ocular manifestations; it is potentially blinding and associated with a decreased survival rate.1,2 Immunosuppressive therapy is the mainstay of treatment1 but plasmapheresis can be helpful acutely by rapidly removing circulating immune complexes and immune reactants while the patient is receiving immunomodulators. We present 2 cases of severe retinal vasculitis due to SLE that were treated successfully with a combination of plasmapheresis and immunosuppression.

Report of Cases. Case 1. A 54-year-old white woman was admitted because of renal failure and changes in mental status. A month earlier the patient had complained of decreased vision in both eyes and was diagnosed as having “retinal lesions” by her ophthalmologist. On admission, findings from the medical examination revealed pericardial effusion, rash, arthritis, and pancytopenia. The visual acuity was 20/50 OD and 20/100 OS and dyspnea developed; pulmonary hemorrhage was discovered. However, the pancytopenia improved and a single intravenous infusion of cyclophosphamide (750 mg) was added to the regimen. No improvement was noted. The results of blood tests revealed high levels of antinuclear antibodies, soluble interleukin 2 receptors, circulating immune complexes, and decreased C3 complement levels. The diagnosis of SLE was proposed based on the clinical symptoms and these laboratory findings. Plasmapheresis was administered for 5 days followed by a single intravenous infusion of cyclophosphamide (750 mg). The patient’s overall medical status improved dramatically. She became capable of fluent communication, her creatinine levels decreased rapidly, and the dyspnea improved.

Systemic lupus erythematosus, polyarteritis nodosa, Wegener granulomatosis, and sepsis were included in the differential cell count. Sepsis was excluded following multiple blood cultures negative for organisms, and intravenous methylprednisolone acetate (1000 mg/d) was administered for 3 days. The patient responded with slight overall improvement, but her condition deteriorated after discontinuation of the steroid therapy. Immunosuppressive treatment was deferred because of pancytopenia with agranulocytosis seen on bone marrow biopsy specimens. Therefore, therapy with intravenous methylprednisolone acetate (80 mg/d) was instituted in combination with intravenous immunoglobulin (200 mg/kg per day) for 5 days. The patient’s mental status deteriorated further and brain vasculitis was disclosed on magnetic resonance imaging. The visual acuity dropped to 20/65 OD and 20/100 OS and dyspnea developed; pulmonary hemorrhage was discovered. However, the pancytopenia improved and a single intravenous infusion of cyclophosphamide (750 mg) was added to the regimen. No improvement was noted. The results of blood tests revealed high levels of antinuclear antibodies, soluble interleukin 2 receptors, circulating immune complexes, and decreased C3 complement levels. The diagnosis of SLE was proposed based on the clinical symptoms and these laboratory findings. Plasmapheresis was administered for 5 days followed by a single intravenous infusion of cyclophosphamide (750 mg). The patient’s overall medical status improved dramatically. She became capable of fluent communication, her creatinine levels decreased rapidly, and the dyspnea improved.

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