Predictors of Visual Outcome and Choroidal Neovascular Membrane Formation After Traumatic Choroidal Rupture

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Objective: To determine predictors of choroidal neovascularization (CNV) and visual outcome after traumatic choroidal rupture.

Methods: A retrospective review of patients with traumatic choroidal rupture diagnosed in the Retina Service, Massachusetts Eye and Ear Infirmary, Boston, between January 1993 and August 2001 was performed. Parametric and nonparametric statistical methods were used to evaluate visual prognosis, CNV, and retinal detachment after traumatic choroidal rupture.

Results: One hundred eleven cases were identified and reviewed. Visual acuity (VA) changes were recorded in all of the cases. Thirty-eight (34%) of the 111 patients recovered driving vision (VA ≥20/40). Rupture location was recorded in 107 cases. Recovery of driving vision was seen in 20 (59%) of 34 eyes with peripheral choroidal ruptures, 17 (22%) of 73 eyes with macular choroidal ruptures, 38 (38%) of 99 eyes without CNV, 1 (8%) of 12 eyes with CNV, 38 (40%) of 96 eyes without retinal detachment, and 1 (7%) of 15 eyes with retinal detachment. Older age and location of rupture within the arcades were positively associated with CNV formation (P = .04 and .03, respectively). Foveal location of rupture, multiple ruptures, and poor baseline VA were associated with failure to recover driving vision in univariate regression analyses. In multivariate analysis, rupture location and baseline VA were independently associated with visual outcome. Of 12 patients diagnosed with CNV, 5 were not treated, 4 were treated with argon laser photocoagulation, 1 was treated with surgery, 1 was treated with argon laser photocoagulation followed by surgery, and 1 was treated with verteporfin photodynamic therapy.

Conclusions: Most patients with traumatic choroidal rupture do not achieve final VA of 20/40 or better. Poor visual outcome was most highly associated with a macular rupture and baseline VA of less than 20/40. The formation of CNV was most strongly associated with older age and macular choroidal rupture.

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Choroidal neovascularization (CNV) is a common abnormality associated with many disease states, including age-related macular degeneration, presumed ocular histoplasmosis syndrome (POHS), angiod streaks, pathologic myopia, photocoagulation scars, posterior uveitis, and choroidal rupture. The new vessels lack endothelial tight junctions and may cause exudation, hemorrhage, and serous retinal detachment. The scarring and atrophy that follow may cause irreversible visual morbidity.

Choroidal rupture may be direct, occurring at the site of impact, or indirect, occurring remotely from the site of impact (countercoup). Eighty percent of choroidal ruptures are indirect.\(^1,2\) Indirect rupture is generally associated with nonpenetrating closed-globe blunt trauma. When the globe is mechanically compressed in the anteroposterior plane, it expands in the horizontal plane. The shear force transmitted is centered at and radiates concentrically outward from the peripapillary uveoscleral tether. Bruch’s membrane is less elastic than the retina and has less tensile strength than the sclera, making it most susceptible to traumatic rupture. Patients with underlying compromise of Bruch’s membrane, as can be seen in angiod streaks, are more susceptible to indirect rupture than the normal population.

Previous studies\(^3,6\) have shown that the majority of indirect ruptures occur temporal to the disc and involve the fovea. Many are isolated, but there can be multiple ruptures. Initial visual acuity (VA) after injury that results in choroidal rupture is usually poor. In a prospective study.
by Bressler and Bressler\(^7\) of 10 patients, initial VA in all of the patients was less than 20/200. However, 6 of the patients regained VA of 20/30 after 3 months. Initial VA and visual recovery are heavily influenced by associated injuries, such as intraocular hemorrhage, commotion retinae, and retinal detachment. Five percent to 10% of eyes develop CNV that may cause delayed visual loss after the rupture heals.\(^3\)\(^-\)\(^5\)

**Risk factors for the development of CNV after rupture are debated.** To date, the largest case series of traumatic choroidal rupture was compiled by Secretan et al,\(^6\) who retrospectively reviewed 79 eyes of 79 patients diagnosed with indirect choroidal rupture at 1 tertiary referral center. Sixteen (20%) of the eyes developed CNV. Secretan and colleagues found that ruptures located juxtafoveally (<200 µm from the center of the foveal avascular zone [FAZ]) and extrafoveally (200-1500 µm from the FAZ) were significantly more likely to develop neovascularization than ruptures located peripherally (>1500 µm from the FAZ). Longer ruptures (>4000 µm), independent of location, also predisposed to the development of neovascularization. Width of the rupture was not found to be significant. Initial VA was not found to be predictive of final visual outcome. Development of neovascular lesions occurred during the first year in 82% of the patients. Based on the findings, it was recommended that extrafoveal membranes be treated with photocoagulation and that subfoveal membranes be observed. In support of the recommendation not to treat, Secretan and colleagues stated that patients with subfoveal neovascular membranes had an average final VA of 0.18 (20/111) and cited 2 published series\(^4\)\(^-\)\(^6\) in which lesions were described to have spontaneously regressed.

The literature\(^4\)\(^-\)\(^6\)\(^,\)\(^9\)\(^-\)\(^13\) addressing the natural history of CNV associated with choroidal rupture consists primarily of uncontrolled case series. Pooled analysis of the data in these articles reveals that visual outcome in untreated eyes is poor. Caution must always be exercised when comparing results collected by noncomparable methods; however, a summary of reported results is instructive. Of 17 untreated cases reported in 7 case series,\(^3\)\(^-\)\(^6\)\(^,\)\(^9\)\(^-\)\(^13\) only 5 patients (29%) recovered VA to better than 20/50. Despite this evidence, it is commonly taught that traumatic CNV should be observed because it is likely to regress spontaneously.

In this study, we examine potential risk factors for the development of CNV after traumatic choroidal rupture as well as treatment outcomes. In addition, we describe potential predictors of final visual outcome independent of CNV formation.

### METHODS

All of the patients diagnosed with choroidal rupture at the Massachusetts Eye and Ear Infirmary, Boston, between January 1993 and August 2001 were identified with a computerized diagnosis-code search. Medical records, funduscopic photographs, and fluorescein angiograms of 169 patients were reviewed. Fifty-eight patients were excluded; 40 of them were excluded because they had fewer than 3 months of posttrauma follow-up, and 18 were excluded because evidence of choroidal rupture was not clearly demonstrated in the record.

The following information was extracted for each patient: age, type and mechanism of injury, concomitant injuries, late complications, preexisting retinal disease, initial and subsequent VA, and number and location of choroidal ruptures. Blunt ocular injuries were classified as external, anterior segment, or posterior segment according to the classification devised by Pieramici et al\(^14\) (Table 1). Intraocular foreign body cases were excluded from the blunt trauma criteria and were dropped from our data set (2 cases).

For the morphometric analysis, funduscopic photographs were digitized and analyzed using Adobe Photoshop 6.0 software (Adobe Systems, Inc, San Jose, Calif). Measurement of the length of the rupture and the minimum distance from the center of the FAZ was carried out by a single investigator (D.N.Z.). Patients were excluded from morphometric analysis if the rupture was not visible, was entirely peripheral, or had margins obscured by scar, hemorrhage, or artifact. Measurements were made in pixels and were normalized based on the vertical optic disc diameter using a standard vertical disc diameter of 1300 µm.

Multivariate Cox proportional hazards regression\(^15\) was performed to determine statistically significant prognostic factors related to posttrauma retention or improvement of VA (VA ≥20/40). Since CNV and retinal detachment were rare outcomes (n = 12 and 15, respectively), multivariate analysis was judged to be inappropriate. Instead, \(\chi^2\) tests and the Fisher exact test were used to identify factors that may be associated with these outcomes.

### RESULTS

One hundred eleven cases were identified and reviewed. Descriptive demographic data are compiled in Table 2. Visual acuity progression was recorded for all of the cases, with a median follow-up of 20 months (follow-up range, 3 months to 50 years). Thirty-eight (34%) of the 111 patients recovered driving vision (VA ≥20/40). Median VA at the last evaluation was 20/100 (VA range, 20/16 to no light perception). The location of the rupture was recorded in 107 of the 111 cases. Recovery of driving vision
was seen in 20 (59%) of 34 eyes with peripheral choroidal ruptures, 16 (22%) of 73 eyes with macular choroidal ruptures, 38 (38%) of 99 eyes without CNV, 1 (8%) of 12 eyes with CNV, 37 (39%) of 96 eyes without retinal detachment, and 1 (7%) of 15 eyes with retinal detachment.

The mechanism of injury was recorded in 100 of 111 cases (Table 2). Because so many types of injuries occurred, there were few results in any single group and results are reported descriptively. Thirty-five (35%) of 100 patients recovered VA of greater than 20/40. These consisted of 14 (67%) of 21 patients hit with a ball, 3 (60%) of 5 patients hit with a BB, 1 (50%) of 2 patients hit with an airbag, 7 (37%) of 19 patients who were assaulted, 3 (27%) of 11 patients involved in nonexplosive industrial accidents, 3 (23%) of 13 patients hit with a large blunt object, 2 (20%) of 10 patients who fell, 1 (10%) of 10 patients injured in motor vehicle accidents, and 0 (0%) of 3 patients involved in explosions.

Predictors of recovery of driving vision evaluated in univariate regression are shown in Table 3, and Kaplan-Meier estimates of cumulative rates of eyes with driving vision according to the time after trauma are shown in Figure 1. Peripheral location of rupture (relative risk [RR], 5.8; 95% confidence interval [CI], 2.5-13.6;
The final patient was treated with 3 sessions of PDT. He with surgery only achieved a VA of 20/50 at follow-up. The patient treated with laser photocoagulation and surgery achieved a final VA of counting fingers. The patient treated with surgery only achieved a VA of 20/30 at follow-up. The final patient was treated with 3 sessions of PDT. He achieved resolution of leakage after all of the treatments, and his VA was 20/320 at 13 months.

**CASE 1: NATURAL HISTORY OF UNTREATED CNV**

A 33-year-old male firefighter was hit in the line of duty by falling ceiling material, sustaining a blunt injury to the right eye. Initial evaluation at 2 weeks after the trauma showed VA of 20/100. He was diagnosed with a traumatic hyphema, vitreous hemorrhage, and a crescentic choroidal rupture involving the fovea (Figure 2A). On serial examinations 3 and 9 months after the trauma, VA was 20/80. A subfoveal CNV was diagnosed 10 months after the trauma (Figure 2B). At the 12-, 14-, and 18-month examinations, VAs were 20/125, 20/125, and 20/160, respectively. The CNV became a fibrotic scar and had associated pigmentation changes. At 20 months after the trauma, new subretinal hemorrhage was noted but VA was unchanged. Visual acuity remained 20/160 at 82 months after the trauma (Figure 2C).

**CASE 2: CNV TREATED WITH PHOTOCOAGULATION**

A 31-year-old man was hit in the left eye with a plastic disk. Initial evaluation was performed 2 days after the trauma. His VA was counting fingers at 1 ft, and he was diagnosed with a hyphema, iridodialysis, vitreous hemorrhage, and 2 choroidal ruptures (1 foveal and 1 peripheral). Two weeks after the trauma, VA was 20/200. Four weeks after the trauma, he was diagnosed with a retinal detachment and underwent repair with a combined pars plana vitrectomy and scleral buckle placement. At 4, 6, and 8 weeks after the trauma, VA was stable at 20/80. A juxtafoveal CNV was diagnosed 7 months after the trauma, with VA fluctuating between 20/160 and 20/320. The patient was treated with laser photocoagulation. Fluorescein angiography (FA) 2 weeks after treatment showed residual CNV inferior to the treated area, and no further treatment was given. During 9 years of follow-up examinations after the CNV was diagnosed, VA fluctuated between 20/160 and 20/320. The retinal appearance showed a flat scar and pigmentary change in the area of the rupture and the CNV.
CASE 3: CNV TREATED WITH SUBMACULAR SURGERY

A 27-year-old man sustained a closed-globe injury to the right eye during a motor vehicle accident. His first examination was performed approximately 1 month later. At that time, VA was 20/100 and the patient had a 35-prism diopter right exotropia. He had a right afferent pupillary defect secondary to a presumed traumatic optic neuropathy and a macular choroidal rupture. Nine months after trauma, gray subretinal tissue was noted in the central macula with associated hemorrhage. Eighty-two months after trauma, a dense, fibrotic, pigmented macula is seen.
after the trauma, the patient noted a decrease in VA. Visual acuity was 20/80, and an epiretinal membrane with subretinal hemorrhage and fibrosis were noted on examination (Figure 3A). Fluorescein angiography revealed CNV. Repeat examination 3 weeks later showed VA of 20/50. Repeat FA showed decreased leakage compared with the first FA. It was felt that the lesion was regressing, and no treatment was given. Approximately 22 months after the original trauma, the patient again noticed decreased vision. Visual acuity measured 20/80, and CNV with associated subretinal hemorrhage was noted. The FA at that time showed that the CNV had increased in size compared with the previous examination (Figure 3B-E). The patient underwent surgical removal of the CNV (Figure 3F). Thirty-nine months postoperatively (62 months after the original injury), VA was stable at 20/50, the retina was flat, and no recurrent CNV was apparent.

Figure 3. Case 3. A, Nine months after trauma, choroidal rupture through the macula with a dumbbell-shaped gray fibrotic subretinal lesion and a superior crescentic rupture with an additional area of subretinal tissue can be seen. B, Twenty-two months after trauma, macular choroidal ruptures with increased subretinal fluid and hemorrhage are seen. An angiogram shows hyperfluorescence and staining of the choroidal rupture (C) with focal areas of fibrosis centrally (D). E, There is a large area of hypofluorescence and leakage with some areas of blocked flow. F, After surgical removal of the neovascular membrane, the macula appears flat with pigmentary changes and no evidence of neovascularization.
CASE 4: SUBFOVEAL CNV TREATED WITH PDT

A 47-year-old man was shopping when a store display fell on his head. He sustained blunt trauma to the left eye. Initial ophthalmic evaluation was performed 2 weeks after the trauma, although the patient recalled immediate loss of vision in his left eye after the accident. At the time of the examination, the patient complained of metamorphopsia. Visual acuity was 20/200, and dilated fundus examination revealed a subfoveal CNV. Four weeks after the trauma, the CNV was shown to be predominantly classic on FA, and VA had improved to 20/40 (Figure 4A-C). The patient was treated with verteporfin PDT according to standard protocols. Visual acuity 3 months after treatment was 20/40, and FA did not show any leakage (Figure 4D-F). Six months after the initial treatment, FA showed leakage and the patient underwent repeat PDT. Ten months after the trauma, VA had decreased to 20/320 and recurrent CNV was noted on FA. He underwent another session of PDT that resolved the leakage. He was left with a fibrotic scar in the area of CNV. Thirteen months after the trauma, VA remained at 20/320 (Figure 4G-I).

COMMENT

Traumatic choroidal rupture can be a devastating ocular injury. Visual loss associated with CNV formation is particularly devastating, not only because it causes profound visual disability but also because the onset of visual loss may be delayed and may follow a period of relative visual stability.

Our study was designed both to elucidate the natural history of choroidal rupture and to identify attributes of rupture that predispose to the development of CNV. To our knowledge, this is the largest series of traumatic choroidal ruptures published to date. Our data suggest that visual outcome after choroidal rupture is worse than previously thought, even in the absence of CNV. We found that foveal location of rupture and poor VA at the initial visit were significant prognostic factors for poor visual outcome, that early and persistent poor VA is most frequently a consequence of foveal or macular involvement in the rupture, and that the mechanism of injury is associated with visual outcome, with the poorest outcome being after injury from an explosion and the best outcome after injury with a sports ball.

Risk factors for the development of CNV after choroidal rupture are debated. The largest series published to date is by Secretan et al5 and included 79 eyes; other published reports3,9,12,13 are smaller case series. Secretan and colleagues found that a rupture located less than 250 µm from the fovea was as likely as a rupture located 250 to 1500 µm from the fovea to develop CNV (occurring in 44% of eyes) whereas a rupture located 1500 to 2100 µm from the fovea was less likely to do so (occurring in 12% of eyes). In contrast, we found that within the macula, decreased incremental distance between the rupture terminus and the center of the FAZ did not increase the risk of CNV formation. Our results support the finding by Secretan and colleagues that ruptures that spare the macula are less likely to develop CNV. Secretan and colleagues also found a shorter median rupture length in eyes that did not develop CNV (median rupture length, 3054 µm) as compared with those that did (median rupture length, 4504 µm) (P = .03). We found formation of CNV to be most strongly associated with older age, macular rupture, and in morphometric analysis, longer length of the rupture.

The physiologic basis of the increased susceptibility of the macula compared with the peripheral retina to CNV after compromise of Bruch’s membrane is a subject of intense research. A report of laser photocoagulation in 8 eyes of 4 monkeys showed that 89% of macular laser scars develop CNV compared with 22% of extramacular lesions.10 There are many factors that might explain the physiologic basis of this observation. New vessel formation is thought to result from an imbalance of proangiogenic and antiangiogenic cytokines that are secreted in response to cellular injury. There are significant differences in hydrostatic and oncotic pressure gradients, blood flow, and retinal pigment epithelial and neural architecture between the central and peripheral retina. Differences in the local environment may contribute to the proangiogenic characteristics of the macula.16-23

The observation that peripheral ruptures are more often associated with retinal detachment than are macular ruptures may explain why overall visual outcome was very poor in our study. Patients with macular ruptures lost vision because of direct macular injury whereas patients with peripheral ruptures lost vision because of retinal detachment.

Choroidal neovascular membranes may be treated with observation, surgery, photocoagulation, or PDT. According to our pooled analysis of published case series,3,9,12,13 the natural history of untreated CNV is poor. In our study, 0 of 5 untreated eyes regained VA of better than 20/100. We suggest that since visual outcomes with observation alone are poor, therapy may be appropriate.

Guidelines for treatment with photocoagulation are extrapolated from the Macular Photocoagulation Study Group data. In our study, 1 of 4 patients treated with laser photocoagulation recovered VA to better than 20/50. Extrafoveal lesions that fit the Macular Photocoagulation Study Group criteria should be treated with photocoagulation. However, the majority of posttraumatic membranes were subfoveal, making laser photocoagulation less attractive as a treatment.

Guidelines for treatment with submacular surgery may be extrapolated from experience with CNV secondary to POHS. Bressler24 reported a series of 63 cases of subfoveal CNV secondary to POHS treated with subfoveal surgery. Twelve months after surgery, 29% of eyes improved by 2 lines of VA whereas 13% worsened by 2 or more lines. Follow-up was variable, but within the period of observation, at least 38% of eyes had recurrent CNV. Three of 8 eyes lost VA after repeated surgery, suggesting that surgery for CNV secondary to POHS is of limited benefit. Gross et al25 described results in 3 cases of submacular surgery for posttraumatic CNV. Patients recovered VAs of 20/20, 20/25, and 20/30 after follow-up of at least 7 months. The results of the Submacular Surgery Trials by Hawkins et al26 for patients with CNV...
Figure 4. Case 4. A, Two weeks after trauma, foveal choroidal rupture with associated hemorrhage and subretinal fluid are seen. An angiogram shows hyperfluorescence and staining of the choroidal rupture and areas of blocked fluorescence (B), as well as early hyperfluorescence and leakage consistent with classic choroidal neovascularization (C). D, Three months after trauma, foveal choroidal rupture with a fibrotic subretinal scar is seen. The early (E) and late (F) phases of an angiogram show hyperfluorescence and staining of the choroidal rupture, areas of blocked fluorescence, and no evidence of leakage. G, Thirteen months after trauma, a large fibrotic scar is seen in the central macula. The early (H) and late (I) phases of an angiogram show hyperfluorescence and staining of the choroidal rupture and fibrotic scar, with no evidence of leakage.
secondary to POHS or idiopathic causes were disappointing. In 112 patients randomized to surgery and 113 randomized to observation, median VAs at 24 months were 20/160 and 20/250, respectively. Subgroup analysis showed that eyes with initial VA of less than 20/100 benefited more from surgery. Choroidal neovascularization recurred in 58% of surgically treated eyes at 24 months. Hawkins and colleagues concluded that although the risks of submacular surgery do not outweigh the benefits in most cases, there may be individual cases in which surgery is indicated. Visual outcome in 1 patient treated with submacular surgery in our study was excellent. Visual acuity improved from baseline and remained stable 3 years after surgery and 5 years after injury. Visual acuity in the second patient treated with surgery may have been limited owing to the laser photocoagulation performed prior to the initial visit.

Results of treatment of subfoveal CNV secondary to age-related macular degeneration with PDT are well described. One series of 24 eyes with CNV and angioid streaks found that treatment with PDT was not effective in preventing VA loss. Visual acuity decreased in 87.5% of patients, stabilized in 8.3%, and improved in 4.2%. Seventy-nine percent of eyes had a final VA of less than 20/400. In a series of 23 eyes with juxtafoveal CNV secondary to POHS, VA improved in 30% of eyes, stabilized in 52%, and decreased in 18%. The visual course did not differ significantly from published rates of the natural history of juxtafoveal CNV in POHS. A second series of 6 patients with juxtafoveal CNV and 5 patients with subfoveal CNV in POHS showed similar results. Visual acuity improved in 50% and 60% of patients, respectively, stabilized in 33% and 20%, respectively, and decreased in 17% and 20%, respectively. A recent review of 26 patients with traumatic choroidal rupture found that 7 patients developed CNV. Five patients were treated with an average of 2 PDT treatments. Visual acuity improved in 3 patients, did not change in 1, and decreased in 1. Intravenous FA showed resolution of leakage in the 4 patients with stable or improved VA.

Photodynamic therapy is an accepted treatment for subfoveal CNV from causes other than age-related macular degeneration. The one patient treated with PDT in our study recovered VA of 20/40 after his first laser treatment but had severe deterioration of VA despite ultimate resolution of leakage on FA. Investigations of combinations of antiangiogenic pharmacotherapy and PDT are ongoing, and we hope that they will improve outcomes in these patients.

There are several limitations of our study. It was a retrospective analysis; therefore, complete records and relevant photographs were not available for all of the patients at all of the visits. There are 2 sources of selection bias that might cause our data set to overestimate the visual morbidity of choroidal rupture. The Retina Service at the Massachusetts Eye and Ear Infirmary is a trauma center for New England and a retinal referral center, and our sample might include a disproportionate number of complicated cases. Forty (26%) of 151 patients whose medical records showed definitive evidence of choroidal rupture were excluded because they were followed up for fewer than 3 months. If patients who develop visual complications are more likely to return for care, poor visual outcome would be overrepresented in our sample. The study was underpowered to identify risk factors for CNV and retinal detachment using regression analysis. Morphometric analysis was complicated by the appearance of subretinal blood or fibrosis in several of the photographs, and the total number of cases available for morphometric analysis was limited. Variability in recording results among the 7 physicians was reduced by cross-referencing the surgical log and medical records from the other ophthalmologists who were involved in the patients’ care.

The time course of development of CNV in case 4 is unusual. The clinical history and imaging, however, are highly consistent with traumatic choroidal rupture and subsequent CNV formation. It is possible that the CNV was there prior to the accident. However, it is unlikely because the patient was not symptomatic.

Most patients with choroidal rupture do not achieve final VA of 20/40 or better. Poor visual outcome was most highly associated with macular rupture and poor baseline VA, and CNV formation was most strongly associated with older age and macular choroidal rupture. Interestingly, in morphometric analysis, CNV formation was not associated with the distance from the center of the FAZ but was associated with the length of the choroidal rupture. Treatment for CNV includes observation, argon laser photocoagulation, surgery, and PDT. Because of the small sample size, we cannot make recommendations regarding treatment.

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