

Choroidal rupture and optic atrophy

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Abstract

The association between post-traumatic optic disc pallor and traumatic choroidal rupture is poorly understood. To further define this relationship, nine cases of indirect traumatic choroidal rupture and post-traumatic optic disc pallor were compared with cases of indirect choroidal rupture without disc pallor in terms of severity of ocular injury, fundus findings, and visual outcome. The type and severity of the injury did not appear to influence the risk of optic disc pallor. Optic disc pallor was associated with a slightly poorer long term visual acuity than eyes without pallor ($p=0.059$). The presence of a relative afferent pupillary defect was strongly associated with optic disc pallor ($p=0.016$). Peripapillary retinal pigment epithelial abnormalities were a common finding, suggesting peripapillary trauma as a cause for optic disc pallor.

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Choroidal rupture is a common complication of blunt ocular trauma. The presence of visual field defects in eyes with indirect choroidal ruptures,^{1,2} and the frequent concentric relationship of such ruptures to the optic disc,³ suggests that the optic nerve may play a mechanical role in the formation of such ruptures,² and may itself be subject to injury. We observed optic nerve pallor in a significant percentage of patients with choroidal rupture without other causes predisposing them to optic atrophy. In the present retrospective study, the type of injury, the number of choroidal ruptures, the proximity to the optic nerve head of the rupture, and the presence of other ocular injuries were compared between patients with and without optic nerve pallor. The study was performed to determine risk factors for optic disc pallor associated with choroidal rupture, to determine its visual significance, and to further define the relationship between these two conditions.

Materials and methods

Twenty one eyes of 20 patients with choroidal ruptures were examined in a retrospective analysis from photographic and clinical records obtained at the Medical College of Wisconsin between November 1977 and February 1989. Patients were excluded who had incomplete clinical records or inadequate or absent fundus photographs. For purposes of the study, the presence of optic disc pallor was determined by notations in the clinical records as to the presence of this finding when compared with the uninjured eye. These findings were confirmed by masked comparisons of stereo colour fundus photographs of the injured versus the uninjured eye. Nine of the eyes had optic nerve pallor when compared with the contralateral eye. One patient had bilateral choroidal ruptures after a motor vehicle accident and developed optic nerve pallor in one eye. None of the patients had a previous history of glaucoma or known history of elevated intraocular pressure, or previous history of orbital or ocular trauma.

In order to determine risk factors for the development of optic atrophy after traumatic choroidal rupture, the visual acuity, age, sex, presence of a relative afferent pupillary defect, number and location of the ruptures, and associated ocular defects were compared in patients with and without optic atrophy. Statistical analysis using Fisher's two tailed exact test, χ^2 square tests, and Wilcoxon two sample tests were employed to compare the study groups with regard to each of the above factors. Visual acuity was entered as the log of the reciprocal of the Snellen acuity and recorded when the patient was first seen following trauma and during the last follow up visit.

Details of patients with choroidal rupture with and without nerve atrophy are summarised in Tables 1 and 2.

Results

The mean age (SD) of the patients without optic

Table 1 Patients with choroidal rupture and optic atrophy

Patient no	Age/sex	Initial vision	Final vision	RAPD*	Number of choroidal ruptures	Peripapillary pigment changes	Cause of visual loss
1	15/M	20/200	20/400	Yes	2	Yes	Optic nerve damage; subfoveal choroidal rupture
2	37/M	CF ^b	8/200	Yes	1	Yes	Optic nerve damage; peripapillary fibrous scar
3	18/M	LP ^c	CF	Yes	1	No	Optic nerve damage; macular scar
4	5/F	HM ^d	HM	Yes	3	No	Optic nerve damage; retinal pigment epithelium damage in macular area
5	18/M	20/400	20/400	Yes	2	No	Optic nerve damage; macular scar
6	14/F	20/60	20/60	Yes	1	Yes	Choroidal rupture through macula
7	20/M	CF	HM	Yes	2	Yes	Choroidal rupture through macula; macular pucker
8	22/M	20/200	CF	Yes	2	No	Macular hole; choroidal neovascular membrane with epiretinal membrane
9	14/M	20/400	20/20	No	1	No	Choroidal rupture through papillomacular bundle; retinal striae

*RAPD=relative afferent pupillary defect; ^bCF=counting fingers; ^cLP=light perception; ^dHM=hand motions.

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Table 2 Patients with choroidal rupture and no optic atrophy

Patient no	Age/sex	Initial vision	Final vision	RAPD	Number of choroidal ruptures	Peripapillary pigment changes	Cause of visual loss
10	24/M	20/50	20/75	Yes	1	No	Comotio retinae; macular scar
11	21/M	5/200	5/200	Yes	1	No	Macular scar
12	23/M	20/200	HM	Yes	2	No	Choroidal rupture through macula
13	20/M	20/20	20/70	No	2	No	Macular hole
14	22/M	20/40	20/40	No	1	No	Choroidal rupture near macula
15	47/M	LP	20/300	No	3	No	Choroidal rupture through macula
16	27/M	20/300	20/300	No	1	No	Peripapillary choroidal rupture; scar in papillomacular bundle
17	38/M	4/200	20/400	No	2	No	Choroidal rupture through macula
18	29/M	20/200	20/70	Undetermined	3	No	Macular hole
19	11/M	HM	4/200	Undetermined	2	No	Macular scar
20	52/M	20/40	20/40	No	1	Yes	Macular scar
21	5/F	20/80	20/80	Undetermined	1	Yes	Retinal striae; cataract

disc pallor was 26.6 (13.4) years and in the patients with optic disc pallor was 18.1 (8.6) years. There were 17 males and three females. The mean time interval (SD) between the injury and the initial examination was 6.3 (12.1) months in the no pallor group and 6.2 (10.1) months in the pallor group. Sixteen patients were seen within 12 months of their injury. The final follow up visit occurred 18.0 (28.2) months after the injury in the no pallor group and 27.0 (28.7) months after the injury in the pallor group. Eight patients were followed for 12 or more months after their initial visit. The difference in follow up time between the two groups was not statistically significant.

In affected individuals optic disc pallor was noted to be present as early as 1 month after the injury. In all cases the disc pallor was noted on the initial examination, except when the fundus view was obscured by media opacity (three cases). The disc pallor, once noted, appeared stable without obvious progression.

Eight of nine patients with optic nerve pallor had relative afferent pupillary defects while three patients in the no pallor group had relative afferent pupillary defects. The difference between the two groups was significant at the 0.016 level using χ^2 analysis. While the mean initial visual acuity was poorer in the group of patients with pallor (log MAR 1.66 versus 1.05) the difference between the two groups was not statistically significant. The final visual acuity in patients with nerve pallor was poorer than in patients without pallor (1.61 versus 0.98) and

this difference approached significance ($p=0.059$). In five of the nine eyes with disc pallor optic atrophy was felt to be the primary cause of visual loss.

Sex, age, the number of choroidal ruptures, the distance of the nearest choroidal rupture to the optic nerve head, the type of injury, and the severity of the ocular injury as measured by the trauma index were not statistically different in the two groups of patients. Peripapillary pigmentary abnormalities and relative afferent pupillary defect were the only associated ocular abnormalities that were increased in the group of patients with optic disc pallor. Four of nine patients had peripapillary pigmentary abnormalities in the pallor group and two patients had peripapillary abnormalities in the no pallor group ($p=0.163$). The pigmentary abnormalities consisted of hypopigmentation, hyperpigmentation, or scarring at the level of the retinal pigment epithelium contiguous with the optic disc margin and usually non-contiguous with the choroidal ruptures (Fig 1). The presence of iridodialysis, traumatic cataract, hyphaema, retinal detachment, and vitreous haemorrhage was not different in two groups of patients. Six of 12 patients developed choroidal neovascular membranes in the no pallor group while one patient developed a choroidal neovascular membrane in the pallor group. This difference was not significant ($p=0.075$, Fisher's exact test).

Discussion

Indirect rupture of the choroid is a well described phenomenon following blunt ocular trauma. Approximately 5% of blunt ocular injuries lead to indirect choroidal ruptures.⁴ Visually significant complications resulting from indirect choroidal ruptures include acute subretinal haemorrhage, macular damage, and late occurrence of choroidal-retinal anastomoses and subretinal neovascularisation.^{5,6}

Visual field defects are also common sequelae of choroidal ruptures and their aetiology is unclear. In such cases, the role of optic neuropathy has not been defined. Frequently, visual field loss is greater than expected based on clinical ophthalmoscopy.⁷ A variety of visual field defects can be seen including nasal steps,⁷ central or centrocaecal scotomas, barring of the blind spot, and generalised constriction of the visual field.⁸ Visual field loss is frequently not related to the location of the rupture or to the interruption of nerve fibre bundles from the

Figure 1 Case 21. Fundus photograph of right eye 1 month after injury demonstrates peripapillary hypopigmentation and non-contiguous choroidal ruptures. No optic disc pallor was present. Visual acuity was 20/80 and a traumatic cataract was present.



rupture.⁹ It has been noted to occur in the absence of optic atrophy.⁹

Our finding of optic nerve pallor in nine of 21 eyes with choroidal rupture suggests that optic nerve damage may occur more commonly than has previously been appreciated in association with such injuries. It appears likely that optic nerve damage could contribute to visual loss, as suggested by the following findings in our study: (1) whether pallor was detected or not, all eyes with visual acuity of counting fingers or less were associated with relative afferent pupillary defects, an indicator of optic nerve damage and a known correlate with visual field loss¹⁰; (2) the mean final visual acuities in patients with optic nerve pallor were poorer than those without pallor; (3) two patients with optic nerve pallor (patient numbers 2 and 7) had Goldmann perimetry performed following their injury and were found to have severe generalised visual field deficits (not shown), similar to those of previously described cases.⁶

Macular abnormalities were common in both the pallor and non-pallor groups, making it difficult to determine the relative effects of macular and optic nerve damage in eyes with both types of lesions. Nevertheless, the severity of visual loss observed in some of the patients (count fingers – hand motions) could not easily be explained by the macular findings alone, except by the possibility of widespread involvement by commotio retinae which might have subsequently resolved, leaving minimal retinal findings. However, in none of our patients was widespread commotio retinae documented. Nine patients were seen within 2 weeks of the injuries.

Interestingly, six of the 20 patients with choroidal rupture had marked retinal pigment epithelial abnormalities consisting of hyperpigmentation or depigmentation around the disc. The localisation of pigment epithelial changes around the disc suggests that peripapillary injury may occur simultaneously with choroidal rupture, though these changes may develop without associated optic disc pallor.

Various causes of optic nerve damage following trauma include direct contusion or laceration of the optic nerve, the blunt forehead trauma syndrome,¹¹ and optic nerve avulsion.¹² In our cases, direct contusion of the optic nerve was unlikely because of the nature of the injuries which consisted solely of blunt injuries to the globe. Although we could not completely rule out some degree of blunt forehead trauma, the injuries sustained suggested blunt ocular trauma as the primary injury. In addition, the findings of peripapillary pigment epithelial disruption are not seen in the blunt forehead trauma syndrome owing to the more posterior location of optic nerve injury in the latter condition. Complete optic nerve avulsion is unlikely in our group of patients, since the clinical syndrome of interrupted retinal vascular perfusion, excavation of

the optic nerve head, and loss of light perception was not present. Cases of partial optic nerve avulsion have been reported with better than no light perception vision.¹² An injury not sufficient to cause clinical optic nerve avulsion, but sufficient to damage the optic nerve fibres might be one mechanism by which optic atrophy could be induced.

The mechanism by which optic nerve damage could occur with choroidal rupture is speculative. A pressure wave strong enough to tear the choroid could conceivably damage the anterior portion of the optic nerve or its vascular supply. Although it has been hypothesised that a 'tethering' effect induced by the optic nerve may be responsible for the concentric configuration of choroidal ruptures,⁹ damage to the optic nerve itself might occur somewhat independently of choroidal damage, and might explain why we found no relationship between the size, proximity to the optic nerve head, and number of choroidal ruptures in the development of disc pallor.

In summary, we report nine cases of traumatic choroidal rupture and associated disc pallor. Optic disc pallor was commonly accompanied by relative afferent pupillary defects and was associated with poorer long term visual acuity than that seen in eyes without pallor. The type of ocular injury and associated clinical findings did not appear to influence the risk of optic disc pallor. We postulate that an indirect and traumatic insult to the region of the optic nerve head may occur simultaneously with indirect traumatic choroidal rupture and produce optic disc pallor. Such an event might in some cases explain the previously described disparity between the nature and extent of visual field abnormalities and the ophthalmic findings in eyes with indirect choroidal rupture.

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- 1 Campos EC, Bedell HE, Enoch JM, Fitzgerald CR. Retinal receptive field-like properties and Stiles-Crawford effect in a patient with a traumatic choroidal rupture. *Doc Ophthalmol* 1978; 45: 381–95.
- 2 Aguilar J, Green WR. Choroidal rupture – a histopathologic study of 47 cases. *Retina* 1984; 4: 269–75.
- 3 Luxenberg M. Subretinal neovascularization associated with rupture of the choroid. *Arch Ophthalmol* 1986; 104: 1233.
- 4 Bellows JG. Observations on 300 cases of ocular war injuries. *Am J Ophthalmol* 1947; 30: 309–23.
- 5 Goldberg MF. Choroidoretinal vascular anastomoses after blunt trauma to the eye. *Am J Ophthalmol* 1976; 82: 892.
- 6 Smith RE, Kelley JS, Harbin TS. Late macular complications of choroidal ruptures. *Am J Ophthalmol* 1974; 77: 650–8.
- 7 Maberley AL, Carvounis EP. The visual field in indirect traumatic rupture of the choroid. *Can J Ophthalmol* 1977; 12: 147–52.
- 8 Hart JC, Natikos E, Raistrick ER, Doran RML. Indirect choroidal tears at the posterior pole: a fluorescein angiographic and perimetric study. *Br J Ophthalmol* 1980; 64: 59–67.
- 9 Wyszynski R, Grossmiklaus H, Frank KE. Indirect choroidal rupture secondary to blunt ocular trauma: a review of 8 cases. *Retina* 1988; 8: 237–43.
- 10 Thompson HS, Montague P, Cox TA, Corbett JJ. The relationship between visual acuity, pupillary defect and visual field loss. *Am J Ophthalmol* 1982; 93: 681–8.
- 11 Anderson R, Panje W, Gross C. Optic nerve blindness following blunt forehead trauma. *Ophthalmology* 1982; 89: 445–55.
- 12 Park J, Frenkel M, Dobbie JG, Choromolcos E. Evulsion of the optic nerve. *Am J Ophthalmol* 1971; 72: 969–71.