Chorioretinal neovascular membranes complicating contusional eye injuries with indirect choroidal ruptures

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Abstract

Chorioretinal neovascular membranes are a recognised but rare cause of late visual loss in eyes suffering contusional injuries. A series of eight cases is presented all with indirect choroidal ruptures involving the perifoveal region. Two main patterns of 'at risk' rupture were noted: a temporally situated rupture passing almost directly through the fovea, and a rupture which curves inferior or superior to the optic disc stopping just short of the fovea. In six of eight cases there was only a partial thickness rupture of the choroid. These neovascular membranes may present at any time after the original injury, either early (within six months of the injury), which could be related to persistence of the normal reparative neovascular response, or late (at least one year after the injury), which are more likely to have resulted from a secondary breakdown of the outer blood-retina barrier.

Late visual loss following a contusional eye injury may result from retinal detachment, angle recession glaucoma, or the growth of a chorioretinal neovascular membrane (CRNVM).¹⁻³ CRNVM formation is a rare complication which usually occurs in eyes with perifoveal indirect choroidal ruptures (ICR), the overall rate of development of CRNVM in ICR being less than 5%.¹⁴ While in some cases substantial loss of central vision occurs, many follow a remarkably benign course compared to CRNVM in senile macular disease.⁴⁻⁶ In certain cases, however, laser treatment has appeared to be beneficial.⁴⁷⁻¹⁰

This is a report on eight patients who developed a CRNVM as a complication of a perifoveal ICR and a review of the literature on the subject. The two main aims of the study were: (1) to see if we could identify the specific type or types of ICR which are likely to develop a CRNVM; (2) to find if there is a specific time interval between the injury and the onset of a CRNVM.

Materials and methods

Patients were included in this study if they had an ICR following a non-penetrating eye injury, with clinical and angiographic evidence of the CRNVM. Six out of eight patients had hospital records available from the time of the original injury. The other two patients could accurately date their eye injury. All patients had a full ophthalmic assessment, including biomicroscopy of the anterior and posterior segment, indirect ophthalmoscopy, retinal photography,

and fluorescein angiography. For each patient the time interval between the injury and either the onset of symptoms of the CRNVM or the finding of clinical or angiographic evidence of a CRNVM was noted. A total of eight patients with a CRNVM developing as a result of an ICR were recruited for the study. A composite diagram was constructed which outlined the site of each rupture which had developed a CRNVM by projecting individual fundus photographs on to a screen. For ease of comparison all slides were projected as if they were a left eye.

Results

Of the eight patients recruited for the study six were male and two female, with an age range of 18-45 years and a follow-up time of 15-72 months. The age at the time of the injury and the time interval between the injury and the onset of the CRNVM are shown in Table I. Four cases developed the CRNVM within four months of the injury. In the other four cases the CRNVM occurred between 20 months and 37 years after the injury. The time delay between injury and CRNVM is also shown in Table II, which includes a review of the literature.¹³⁻⁵⁷⁻¹⁵ Although this table divides the patients by the time delay into three groups, <6 months, 6-12 months, and >1 year, most cases fell into the <6 months (early onset) or >1 year (late onset) groups.

All eight ICR are illustrated in the composite diagram (Fig 1). Two main patterns emerge: *type*

TABLE I Summary of clinical details of patients developing a CRNVM as a complication of a contusional eye injury

Case	Age at injury (years)	Injury to CRNVM (months)	Initial VA	Final VA	Site	Other factors
1	29	2	6/5	2/60	Temporal	
2	20	4	6/6	6/6	Inferior	
3	36	15 years	6/6	6/60	Superior	Diabetic
4	39	20	6/6	3/60	Superior	Detachment
5	37	8 years	6/9	6/9	Inferior	
6	36	4	6/12	6/18	Temporal	
7	8	37 years	6/60	6/60	Temporal	
8	18	4	3/60	2/60	Temporal	Hypotony

TABLE II Time interval between initial injury and development of a CRNVM in our series (first set of figures) and in series reported elsewhere (figures in brackets)

Time from injury to CRNVM	No of patients
Less than 6 months	4 (17)
6 to 12 months	0 (5)
More than 1 year	4 (13)

The numbers in brackets refer to reported cases¹³⁻⁵⁷⁻¹⁵ combined with our series.

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Figure 1: Composite diagram illustrating sites of individual ICR developing CRNVM. Black areas are the actual site of the neovascular membrane.

1 temporally situated passing through the fovea; and type 2, inferior/superior to disc with apex near fovea.

Fluorescein angiograms of typical type 1 (Figs 2, 3, 4) and type 2 (Figs 5, 6) ICR with CRNVM are illustrated. As can be seen from the angiograms, these ICR are mainly very thin, partial thickness ruptures. Figure 6 is an exception, being a broader, mainly full thickness rupture; however, it is at the thin apex, which extends into the perifoveal region, that the CRNVM developed. The other broad full thickness rupture in the series was an unusual case in that the CRNVM developed 37 years after the injury and was very similar in appearance to another late onset CRNVM (26 years after injury) described by Yanuzzi *et al.*¹⁵

In three eyes complicating factors were present, which may have influenced the development of a CRNVM. Patient 3 was a diabetic with an ICR above the disc and had inferotemporal laser for peripheral new vessels. Two months later a CRNVM occurred related to the ICR. There was no damage to the ICR from the laser, nor was there any significant diabetic

Figure 2: Late phase fluorescein angiogram of patient 1 six weeks after injury. CRNVM present but is asymptomatic (? persistence of normal reparative choroidal neovascularisation in a dormant stage). Visual acuity 6/5.

maculopathy. Patient 4 had a retinal detachment due to a dialysis which occurred before the CRNVM. Patient 8 had prolonged hypotony, which produced disc swelling (Fig 7) and macula oedema and later developed glaucoma.

The visual outcome is displayed in Table I and Figure 8. Three eyes with good vision maintained this without treatment (patients 2, 5, 6). Three patients with initially good vision had their vision reduced to 6/60 or less (patients 1, 3, 4), one having unsuccessful laser therapy for the CRNVM and another losing central vision owing to a retinal detachment. Two patients with initially poor vision were unchanged. The time of onset or type of rupture did not affect the visual outcome.

Discussion

Following contusional eye injuries the retinal pigment epithelium (RPE) and Bruch's membrane (BM) are particularly at risk of damage, and the outer blood-retina barrier breaks down in response to this insult.^{14 16} This primary breakdown is usually restored to normal within

Figure 3: Patient 1 six months after injury. CRNVM is now symptomatic and is a growth phase. (a) Early phase. (b) Late phase.

Figure 3A

Figure 3B

Figure 4: Fluorescein angiogram of temporally situated ICR with CRNVM occurring four months after injury. (a) Early phase. (b) Late phase.

Figure 4A

Figure 4B

7–14 days of the injury.¹⁶ Histopathological studies in eyes with choroidal ruptures have shown that choroidal neovascularisation occurs as part of the repair process, generally regressing completely in the early postinjury stage.¹⁷ Whether an exuberant overgrowth of this normal reparative choroidal neovascularisation or a persistence of this membrane in a dormant asymptomatic^{18,19} phase under the RPE is responsible for the early type of CRNVM is open to speculation. Late onset CRNVM (more than one year after the injury) could also arise from a dormant choroidal neovascular membrane. However it would seem more likely, especially in cases occurring many years after the injury, that these represent a new CRNVM developing in an area of weakness – perhaps constituting a form of involutional macular degeneration due to localised, excessive ‘wear and tear’ following the trauma.

We noted two distinct patterns of rupture which gave rise to a CRNVM both in our series and in the majority of the reported cases^{1,3-5,7-15,20}: (1) thin partial thickness ICR situated temporally and curving through the fovea; (2) ICR superior or inferior to the disc stopping just short of the fovea.

With thin, partial thickness ICR the CRNVM can develop at almost any point on the rupture, though it is usually near the fovea. If the ICR is

broad and full thickness, a CRNVM is most likely to develop if the apex of the rupture extends into the perifoveal region, and the CRNVM would then occur at the foveal end.

The predilection of CRNVM for the macular region is well established in other diseases^{6,21} and presumably accounts for the perifoveal situation of most CRNVMs associated with ICRs. The neovascular inhibitory ability of the RPE is least in the perifoveal region and appears to decline with age.²¹ Consequently ruptures of BM and damage to the RPE are much more likely to result in a CRNVM in this site than at other areas in the retina.²²

The final visual outcome in these eyes was relatively good, if we remember that most had no treatment. The benign nature of these CRNVMs is attributable to the fact the majority of contusional eye injuries occur in young males²³ with healthy RPE. Furthermore the type of rupture which gives rise to a CRNVM often produces a very focal area of damage; therefore the ICR is surrounded by a zone of healthy RPE functioning as normal to inhibit angiogenesis.²⁴ Presumably CRNVMs arise at sites where a balance has been achieved, such that the damage to the RPE and Bruch’s membrane is sufficient to prevent them from performing their normal task of inhibiting subretinal neovascularisation, while the damage incurred by the choriocapillaris is insufficient to prevent it from proliferating through any gaps created.

It is worth noting that ‘other factors’ (laser for diabetic retinopathy, retinal detachment, hypotony) may have played a part in the development of the CRNVM in three out of eight

Figure 5: Patient presenting with visual distortion. Fluorescein angiogram shows ICR inferior to disc with CRNVM at foveal end. History of contusional injury eight years previously (punch) given only in retrospect. Symptoms settled without treatment, maintaining 6/9 vision.

Figure 6: Broad ICR inferior to disc with CRNVM at foveal end. Visual acuity maintained at 6/6.