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Epiretinal Membrane Formation in Terson Syndrome

Masahiko Yokoi, Manabu Kase, Toshiki Hyodo, Midori Horimoto, Fumihiko Kitagawa and Renpei Nagata

Department of Ophthalmology, Teine Keijinkai Hospital, Sapporo, Japan

Abstract: Clinical features of epiretinal membranes were examined in 22 eyes of 13 patients with Terson syndrome who were treated with pars plana vitrectomy. The shape and localization of the epiretinal membranes were intraoperatively evaluated and correlated with the presence or absence of posterior vitreous detachment (PVD). Patients with complete PVD, but with no membrane found during surgery, were followed postoperatively. Membrane formation ultimately developed in 13 of the 22 eyes. In eight eyes, PVD was incomplete and the epiretinal membrane was found at the optic disc or along the temporal vascular arcades, displaying retinal folds and vascular tortuosity. Three eyes had massive tractional retinal detachment; five of those with complete PVD developed a thin epiretinal membrane around the posterior pole that became more apparent during long-term follow-up. From these observations, we can classify epiretinal membrane formation in Terson syndrome into two groups: with complete, or with incomplete, PVD. It also appears that multiple pathological processes involving the vitreoretinal interface were responsible for the formation of epiretinal membranes. **Jpn J Ophthalmol 1997;41:168–173** © 1997 Japanese Ophthalmological Society

Key Words: Epiretinal membrane, posterior vitreous detachment, Terson syndrome, vitreous hemorrhage.

Introduction

Patients with Terson syndrome have subarachnoid and vitreous hemorrhages, as well as subretinal or preretinal hemorrhages.[1,2] The visual outcome, with or without vitrectomy, is generally good.[3] The outcome is poor, however, if optic atrophy, macular hole, or epiretinal membrane formation occur. Epiretinal membranes in Terson syndrome are seen, ophthalmoscopically, as a cellophane-like appearance of the retina,[4] premacular fibrosis, or as a preretinal membrane[5,6] persisting even after resolution of the vitreous hemorrhage. Schultz et al[3] reported the presence of epiretinal membranes in Terson Syndrome in 19 or 30 eyes (63%), stating that there was no difference in incidence in eyes treated with or without vitrectomy. Velikay et al[7] recently described proliferative membrane formation with severe retinal detachment leading to postoperative deterioration of visual acuity in Terson syndrome.

Although epiretinal membranes occur frequently in this syndrome, the etiology remains unclear. We, therefore, studied fundus changes in 22 eyes from 13 patients with Terson syndrome treated by vitrectomy. The patients were followed for more than 1 year postoperatively. We found epiretinal membranes in 13 eyes and analyzed the structures and sites in order to understand more about their etiology.

Materials and Methods

Twenty-two eyes of 13 patients with Terson syndrome were treated by pars plana vitrectomy after vitreous hemorrhages had caused massive opacities persisting for more than 3 months. Patients ranged in age from 25 to 61 years; the cause of the hemorrhage was idiopathic (13 eyes of eight patients) or traumatic (nine eyes of five patients) (Table 1). Results of visual acuity, electroretinogram, visual evoked response and echogram evaluations were recorded preoperatively. During surgery, any posterior vitreous detachment (PVD) and vitreoretinal adhesions were noted. Most importantly, we confirmed the presence or absence of an epiretinal membrane. If present, it was removed by membrane peeling or

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Address correspondence and reprint requests to: Manabu KASE, MD, Teine Keijinkai Hospital, 1-12 Maeda, Teine-ku, Sapporo 006, Japan

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Table 1. Clinical Data: Onset of Subarachnoid Hemorrhage to Vitrectomy

Case	Age	Sex	Cause	Time Interval	Visual Acuity		Type of PVD	Dome	Formation of ERM	
					Preoperative	Postoperative			Preoperative	Postoperative
1. R	25	M	trauma	3 mo	hm	20/20	○	−		[a]
L					hm	15/20	▲	−	+	
2. R	29	M	trauma	6 mo	20/200	20/20	○	−		
L					2/200	20/30	○	−		[a]
3. R	34	M	trauma	7 mo	hm	20/100	▲	−	+	[a]
L					hm	20/20	▲	−	+	
4. R	36	F	idiopathic	3 mo	hm	20/40	○	−		
L					npl	npl	○	−		
5. R	37	M	trauma	7 y	20/60	20/60	▲	−	+	
L					20/200	20/20	▲	−	+	
6. R	47	M	idiopathic	4 mo	2/200	20/30	▲	−	+	
L					10/200	20/25	○	−		[b]
7. R	48	M	idiopathic	3 mo	hm	20/20	▲	+	+	
L					hm	20/20	○	−		
8. L	48	M	trauma	3 mo	fc	20/25	▲	−	+	
9. R	51	F	idiopathic	20 mo	2/200	20/25	○	−		
L					10/200	20/25	○	−		
10. L	53	M	idiopathic	7 mo	fc	25/20	○	−		
11. R	53	F	idiopathic	4 mo	hm	20/20	○	−		
12. L	56	M	idiopathic	3 mo	fc	20/30	○	−		[a]
13. R	61	M	idiopathic	4 mo	fc	20/30	×	−		
L					fc	hm	×	−		

PVD type: ○ = complete, ▲ = incomplete, × = posterior vitreous membrane not detached. Dome: dome-shaped membrane. ERM: epiretinal membrane.

[a]The first subgroup of epiretinal membrane with complete PVD.

[b]The second subgroup of epiretinal membrane with complete PVD.

delamination. Visual functions, ophthalmoscopic fundus appearance (even in eyes with complete PVD), and fluorescein angiograms (FAG) were monitored for more than 1 year.

Results

With Incomplete PVD

Epiretinal membranes were found preoperatively at the vitreoretinal interface in eight eyes with incomplete PVD. The membrane in Figure 1 (top) originated in the inferior vascular arcade and attached to a vitreous gel mass. This pulled the retina and retinal vessels into the vitreous cavity, covering the macular region (black arrow, Figure 1, top). The patient had lost visual acuity in both eyes from subarachnoid hemorrhage 7 years earlier. Following pars plana vitrectomy to remove the epiretinal membrane (black arrow, Figure 1, bottom), visual acuity of the left eye improved to 20/20; visual acuity of the right eye improved only to 20/60 because of chorioretinal atrophy in the papillomacular region.

In patient 1, preoperative echography of the left eye showed retinal detachment with dense vitreous insertion to the optic nerve head (Figure 2, top). During vitrectomy, a thin, broad epiretinal membrane was found expanding along the temporal vascular arcades with its trunk attached to the optic nerve head, causing a severe tractional detachment. Vitrectomy with complete removal of the membrane and gas tamponade resulted in reattachment of the retina, although several retinal folds remained around the posterior pole (Figure 2, bottom). Final visual acuity was 15/20.

In patient 8, a thin epiretinal membrane was discovered in the left eye just after the vitrectomy, while separating and removing the posterior vitreous membrane from the retina. Fundus examination of this eye, 1 week postoperatively, found that the superior temporal vessels were tortuous and dilated. Retinal folds were connected to the epiretinal membrane (open arrow, Figure 3, top) at the crossing of the superior temporal artery and vein (Figure 3, top). There was a 5-disc-diameter (DD) pigmented circle at the posterior pole (black arrow). Ophthalmoscopic examination 6 months later found that the

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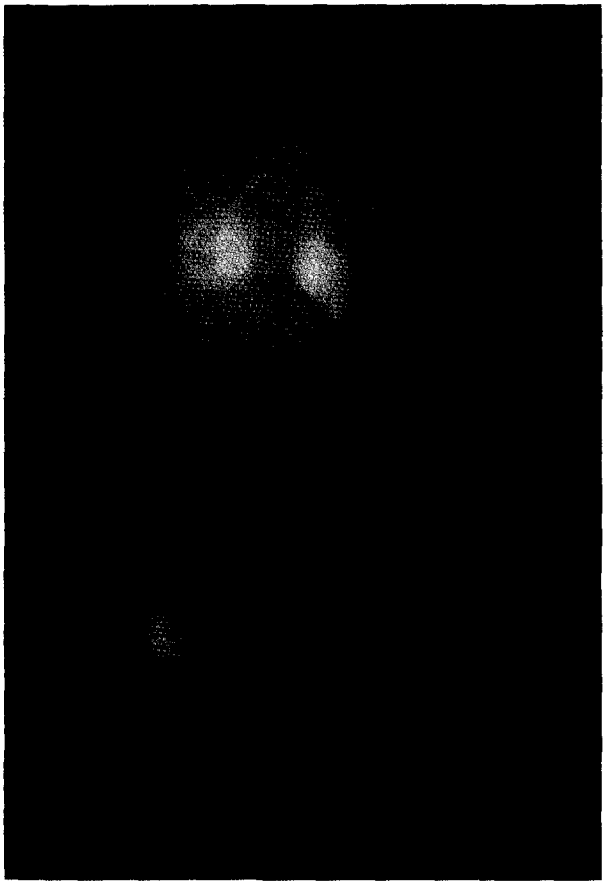

Figure 1. Top, Case 5: Preoperative fundus OS. Epiretinal membrane with insertion to dense vitreous gel at inferior temporal vascular arcade, pulling retinal vessels into vitreous cavity (black arrow). Bottom: Postoperative fundus with membrane removed (black arrow); atrophic chorioretinal region in papillomacular area.

Figure 2. Top, Case 1: Preoperative echographic findings. Traction retinal detachment and dense vitreous insertion to optic nerve head. Bottom: Fundus 1 week postoperatively. Retinal folds cross macula; retinal hemorrhages; tortuousity of retinal vessels.

epiretinal membrane had disappeared, as had the vascular abnormalities, pigmented circle, and retinal folds (Figure 3, bottom).

With Complete PVD

Five eyes with complete PVD had preretinal membranes around the posterior pole identified during postoperative examinations. The left eye of patient 6 had retinal folds extending radially to the macula, found 1 week following vitrectomy (black arrow, Figure 4, top). Fluorescein angiography (FAG) revealed a salt-and-pepper-like leakage of dye at the superotemporal side of the vascular arcade (Figure 5, top).

Ophthalmoscopic examination 2 years later found venous dilatation and severe distortion of the vessels; massive retinal folds covered the entire posterior pole (Figure 4, bottom). An epiretinal membrane with indistinct demarcation was also observed (black arrow, Figure 4, bottom). At this time, leakage of fluorescein on the superotemporal side of the vascular arcades was greater (large arrow, Figure 5, bottom) and new leakage was observed temporal to the macula (small arrow, Figure 5, bottom).

In patient 2, ophthalmoscopic examination of the right eye 1 week postoperatively revealed a wrinkled retinal surface around the macula. Six weeks later, the thin epiretinal membrane covered the papillomacular region (black arrow, Figure 6, top). Three years later, the membrane was smaller but more easily visible than earlier (Figure 6, bottom). FAG was normal. In spite of the preretinal membrane, the postoperative visual acuity was 20/20.

The epiretinal membrane observed in the right eye of patient 1 was clearly visible 1 week following surgery (Figure 7, top). The membrane, with a 0.5 DD hole lay over the posterior pole and gave the retinal surface an irregular reflex. Ophthalmoscopic

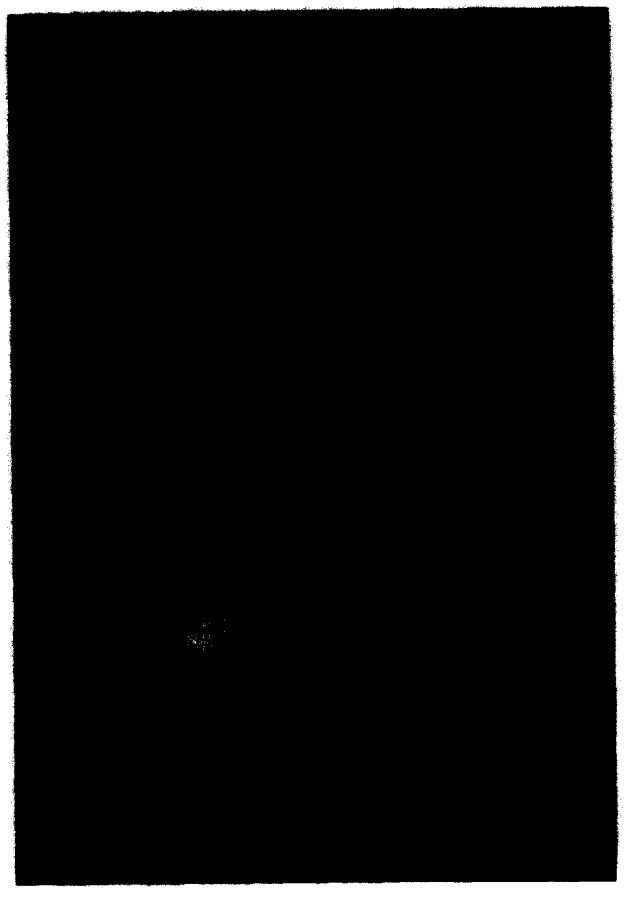

Figure 3. Top, Case 8: Fundus OS 1 week postoperatively. Open arrow: Crossing point of tortuous, dilated superior temporal artery and vein, which also shows that the retinal folds were formed directly to the preretinal membrane. Black arrow: 5DD pigmented at posterior pole. Bottom: Fundus examination 6 months later: retinal vascular changes, pigmented circle and retinal folds have disappeared; no preretinal membrane.

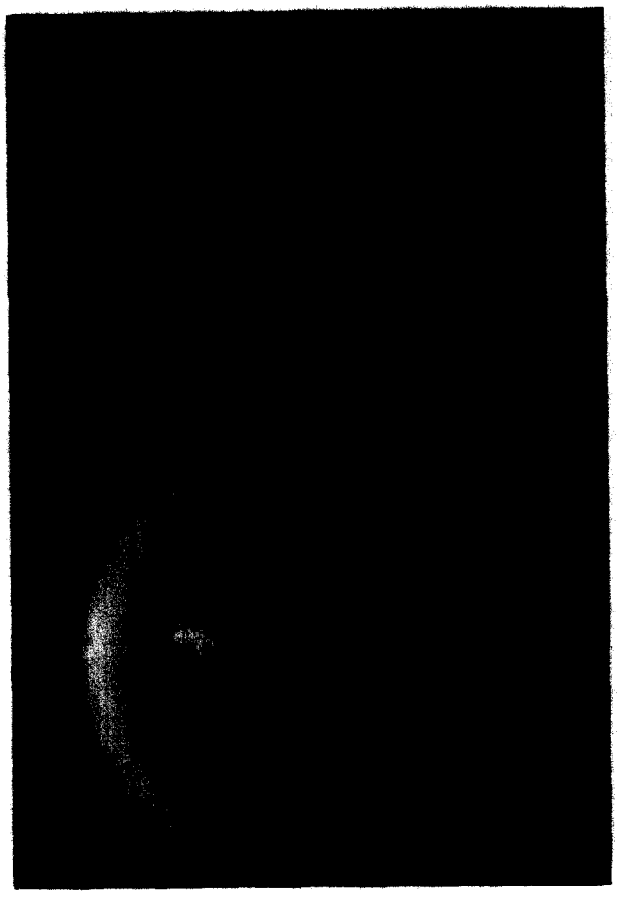

Figure 4. Case 6: Postoperative ophthalmoscopic findings OS. Top: 1 week postoperatively: Within superior and inferior vascular arcades, retinal folds radial to the macula (open arrow) and distortion of retinal vessels (black arrow). Bottom: 2 years later: Severe distortion of vessels (open arrow); massive retinal folds cover entire papillomacular region, indicating formation of new preretinal membrane.

examination 14 months later revealed that the membrane had contracted into radiating corrugations; the perimeter was clearly detached from the retina (Figure 7, bottom); FAG was normal. Final visual acuity was 20/20.

The epiretinal membranes with complete PVD could be further classified into two subgroups. The first subgroup expanded from the site of a previous preretinal hemorrhage, causing tortuous retinal folds with masking of underlying retinal vessels, similar to that reported by Schultz et al.[3] Fluorescein angiography (Figure 5) also revealed that the underlying retina was hyperpermeable to dye, suggesting that additional retinal abnormalities, other than preretinal or vitreous hemorrhage, may also have had significant influence on the epiretinal membrane formation. The second subgroup, observed in four eyes with complete PVD, had a clear margin; however, unlike the first subgroup, did not involve the retina and optic nerve head. These epiretinal membranes varied little in size, over time, shrinking very little. The underlying retina and retinal vessels appeared normal by ophthalmoscope and FAG. The membranes may be remnants of the posterior wall of the precortical vitreous pocket,[8] remaining after spontaneous PVD,[9] although we included them in epiretinal membranes in this study, based on ophthalmoscopic appearance.

Discussion

We found epiretinal membranes in 59.1% (13/22) of vitrectomized eyes from two groups of patients with Terson syndrome: those with complete, or with

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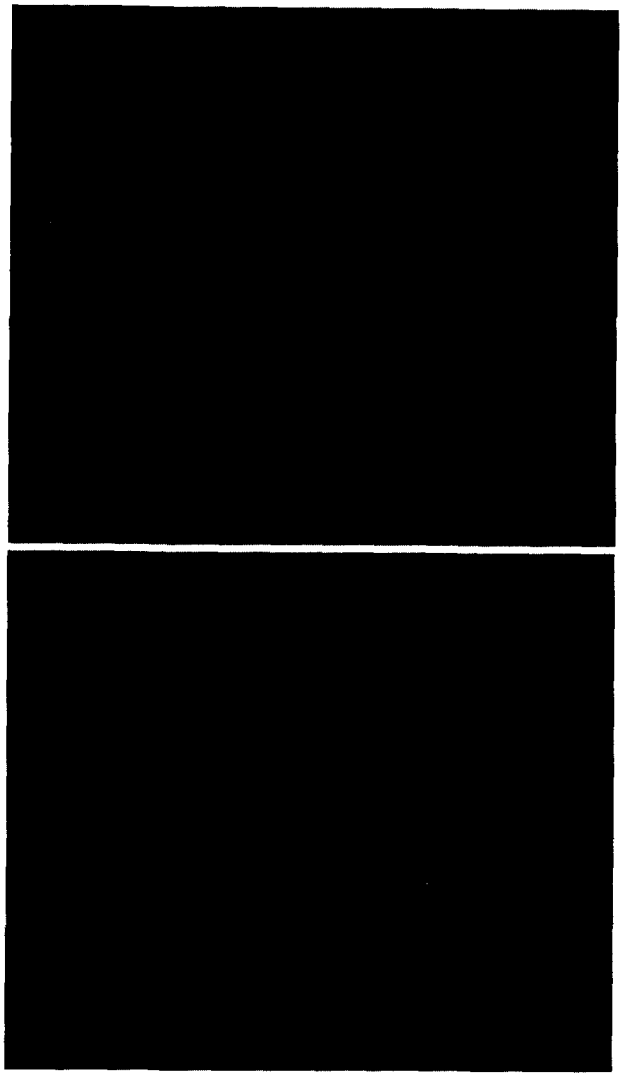

Figure 5. Top, Case 6: Fluorescein angiography OS: Salt-and-pepper-like leakage of dye at superior-temporal side of vascular arcade 1 week postoperatively. Bottom: 2 years later: Increased dye leakage in same area (large arrow); new dye leakage temporal to macula (small arrow).

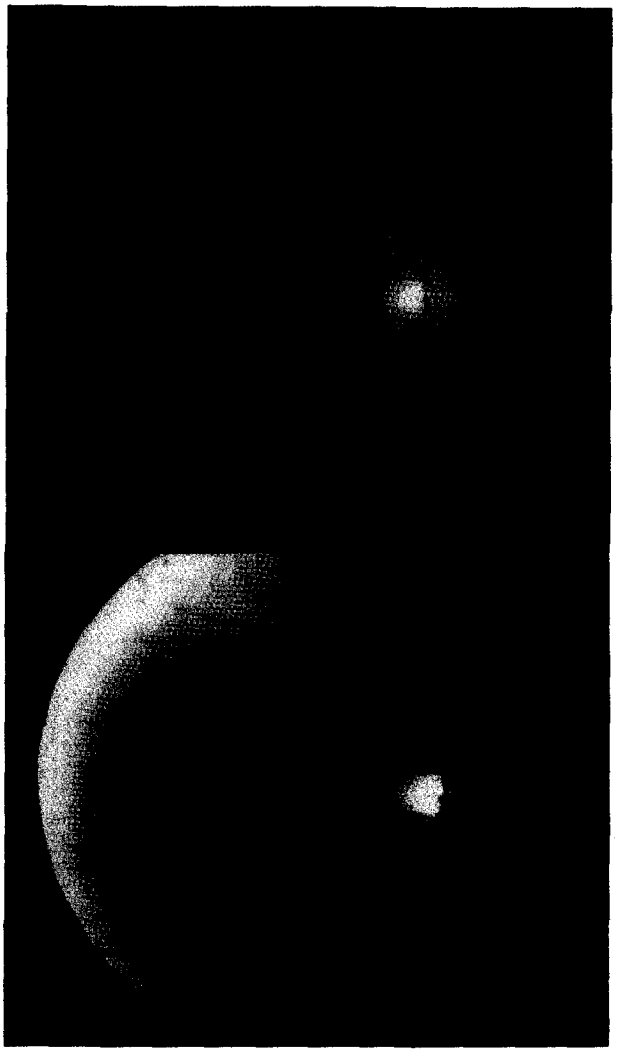

Figure 6. Top, Case 2: Ophthalmoscopic findings OD 1.5 months postoperatively: Thin, irregular preretinal membrane covers papillomacular region with retinal folds; black arrow: clear margin of preretinal membrane. Bottom: 3 years later, epiretinal membrane is more evident; lower margin has shifted upwards because of membrane shrinkage (black arrow).

incomplete, PVD. In the eight eyes with incomplete PVD, membranes associated with prominent retinal changes such as retinal folds, retinal vascular tortuosity, and venous dilatation, were found at the vitreoretinal interface around the optic nerve head and/or the temporal vascular arcades. Three eyes had tractional retinal detachment with proliferative membranes, possibly resulting from migration and proliferation of retinal glial cells caused by persistent mechanical traction from the posterior vitreous membranes. Severe retinal detachment was also reported by Velikay;[7] we, thus, assume that Terson syndrome involves proliferative vitreoretinopathy with severe traction retinal detachment more frequently than previously thought.

The epiretinal membranes in patients with complete PVD had characteristic clinical features. The membranes were not found during surgery, but noticed postoperatively due to their unusual appearance with irregular retinal surfaces between the superior and inferior temporal vascular arcades.

The intraocular hemorrhages in Terson syndrome probably result from the sudden increase of intravenous pressure secondary to elevated cerebrospinal fluid pressure[1,2] that leads to rupture of venous vessels within the retina and optic nerve head. Intrareti-

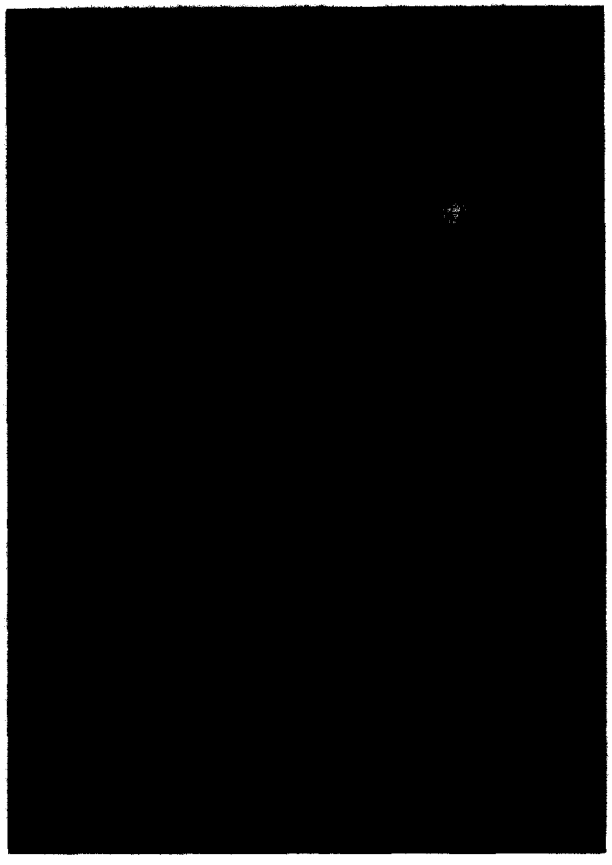

Figure 7. Top, Case 1: 1 week postoperatively: Epiretinal membrane formation with irregular reflex of the retinal surface OD 0.5 DD hole with clear demarcation at membrane. Bottom: 14 months later, shrunken membrane with radiating retinal folds temporally; round margin of membrane, with hole, clearly detached from retina.

nal hemorrhage breaks down the internal limiting membrane, resulting in preretinal and vitreous hemorrhages. This, in turn, causes glial cell migration and proliferation into the vitreous cavity. Earlier studies have shown that intravitreal injection of red blood cells can break down the internal limiting membrane and lead to the formation of epiretinal membranes.[10]

We showed epiretinal membrane formation in 59% of 22 eyes with Terson syndrome, very similar to the incidence reported by Paul et al[3] (63%), which suggests that Terson syndrome has a high incidence of epiretinal membrane formation. Nine eyes, however, had no epiretinal membrane although there were preoperative preretinal and vitreous hemorrhages. This suggests that factors other than preretinal and vitreous hemorrhages are required. The distinct morphological types of epiretinal membranes in Terson syndrome support the conclusion that several processes are responsible for the formation of these membranes. Results of the present study indicated that the posterior vitreous detachment from the retina is a significant factor modulating epiretinal membrane formation in Terson syndrome.

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