development of a provocation test to indicate whether an iridotomy is necessary in eyes that have pigment dispersion syndrome or pigmentary glaucoma without observed iris concavity.

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Visual Acuity as a Prognostic Indicator in Stage I Macular Holes

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PURPOSE/METHODS: To evaluate the role of initial visual acuity as a factor for progressive loss of vision or progression to a full-thickness macular hole in eyes with stage I macular holes (tractional foveal detachment without dehiscence). The study population included 35 patients with stage I macular holes with best-corrected visual acuity of 20/25 to 20/80 in one eye, and a full-thickness macular hole in the fellow eye.

RESULTS/CONCLUSIONS: Eyes with stage I macular holes with best corrected visual acuity between 20/50 and 20/80 had a 66% (ten of 15 eyes) rate of progression to full-thickness macular hole, whereas eyes with best-corrected visual acuity of between 20/25 and 20/40 had a 30% (six of 20 eyes) risk of progression to full-thickness macular hole. The risk of progression to macular hole is significantly higher in eyes with stage I macular holes with best-corrected visual acuity of 20/50 or worse (P = .03).

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STAGE I MACULAR HOLES (TRACTIONAL FOVEAL DETACHMENT without dehiscence) represent the earliest clinical stage of macular hole development in the classification system of Gass,[1] and manifest as a yellow spot (stage Ia) or yellow ring (stage Ib) in the fovea without clinical evidence of a full-thickness retinal defect. In previous reported clinical series, the reported rate of progression of stage I macular holes to full-thickness macular holes varied from 56% to 67%,[1,2] and was 40% overall in the observation group of the vitrectomy for prevention of macular hole study.[3] This study evaluates the observation group of the national randomized trial on vitrectomy for prevention of macular holes,[3] in order to assess the role of initial visual acuity on progression to a full-thickness macular hole.

The inclusion criteria for the prevention of macular hole study have been previously published.[3] Visual acuity was measured with careful refraction by an ophthalmic technician in a standardized fashion, using modified Bailey-Lovie charts (Early Treatment of Diabetic Retinopathy Study). The study required a symptomatic stage I macular hole in one eye with best-corrected visual acuity of 20/25 to 20/80, and a full-thickness macular hole in the fellow eye with best-corrected visual acuity of 20/80 or worse. The group randomized to observation included 35 patients of the potential 62 patients included in the study. Best-corrected visual acuity at entrance into the study ranged from 20/25 to 20/40 in 20 eyes, and from 20/50 to 20/80 in 15 eyes.

Stage I macular holes with best-corrected visual acuity of 20/25 to 20/40 (group 1) had only a 30% (six of 20 eyes) rate of progression to full-thickness macular holes, compared to 66% (ten of 15 eyes) in stage I macular holes with best-corrected visual acuity of 20/50 to 20/80 (group 2)(P = .03). The average length of follow-up in group 1 was 19 months and in group 2 was 15 months. In the 16 eyes that progressed to a full-thickness macular hole, the average time to progression was 4.1 months. Additionally, the final visual acuity outcome at one year in group 1 was 20/40 or better in 67% (13 of 20 eyes) and 20/50 or worse in 33% (seven of 20 eyes), compared to a final visual acuity outcome of 20/40 or better in 36% (six of 15 eyes) and 20/50 or worse in 64% (nine of 15 eyes) in group 2, although the difference between the two groups was not statistically different (P = .09). Of

the 16 stage I eyes, which progressed to a full-thickness macular hole, the final visual acuity was 20/40 or better in 6% (one eye) and 20/50 or worse in 94% (15 eyes).

The risk of progression to a full-thickness macular hole varies markedly with the level of initial visual acuity in stage I macular holes, which should thus greatly influence treatment decisions in these eyes. Since stage I macular holes in eyes with best-corrected visual acuity of 20/40 or better have a 70% chance of spontaneous improvement, observation and a conservative approach should be considered. Once eyes with stage I macular holes progress to a best corrected visual acuity of 20/50 or worse unrelated to other causes for vision loss, stage I macular holes now have a 66% chance of progression to a full-thickness macular hole and loss of vision. This markedly increased risk of progression with visual acuity worse than 20/50 may be related to progressive foveal thinning, as well as the difficulty in differentiating early stage IIb from advanced stage Ib macular holes with very thin central foveal tissue. Although the vitrectomy for prevention of macular hole study was unable to show a benefit to pars plana vitrectomy and peeling of the posterior cortical vitreous in stage I eyes,[3] further investigation into therapeutic intervention may be indicated for this particular group of stage I macular holes with a high risk of progression.[4] Possible reasons for this lack of benefit for pars plana vitrectomy alone include the following: (1) surgically induced vitreofoveal traction during peeling of the posterior hyaloid; (2) inadequate release of cortical vitreous traction during surgery; (3) structural weakness of foveal tissue because of the previous traction; (4) intraoperative, or unrecognized preoperative foveal tears; (5) residual intraretinal tractional forces caused by inherent retinal elasticity; and (6) epiretinal membrane formation with secondary foveal traction. Pars plana vitrectomy, combined with gas-fluid exchange for postoperative tamponade, may be a reasonable therapeutic alternative, in order to allow support of the weakened foveal tissue after vitrectomy surgery, as well as to treat any intraoperative dehiscence in the fovea. A similar surgical approach has become increasingly successful in the treatment of full-thickness macular holes.[5] Further investigation into alternative therapies may be warranted in stage I macular holes that are at high risk for progression.

THE VITRECTOMY FOR PREVENTION OF MACULAR HOLE STUDY GROUP

The participants in the Vitrectomy for Prevention of Macular Hole Study Group follow.

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Vitreous Hemorrhage and Retinal Vein Rupture

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PURPOSE/METHODS: We examined a 23-year-old woman who had a sudden onset of floaters after self-induced emesis.

RESULTS/CONCLUSIONS: Examination showed a dense vitreous hemorrhage originating from a rupture site in the wall of the superotemporal branch vein. We postulate a preexisting weakness in the retinal vein wall as a predisposing factor to rupture. This mechanism may explain some cases of vitreous hemorrhage associated with a Valsalva maneuver.

A 23-YEAR-OLD WOMAN HAD A SUDDEN ONSET OF floaters in her left eye immediately after self-induced emesis. Her medical history was remarkable only for mild acne for which she was taking tetracycline, 250 mg orally twice a day. Ocular history and family history were unremarkable.

Examination approximately 90 minutes after the event disclosed a visual acuity without correction of R.E.: 20/20 and L.E.: 20/80. Pupils were equal and reactive to light and were without an afferent pupillary defect. Tensions by applanation tonometry were 18 mm Hg in both eyes. Confrontational visual fields and extraocular eye movements were intact in both eyes. The anterior segments were normal. Fundus examination of the left eye showed a large area of layered preretinal hemorrhage superotemporal to the fovea as well as a smaller preretinal hemorrhage inferior to the disk (Fig. 1). We observed a more diffuse area of vitreous hemorrhage over the inferior half of the macular region that was funneling into a pocket in the formed vitreous and creating a large collection of intravitreal blood inferiorly. An area of discontinuity in the wall of the superotemporal branch vein with surrounding intraretinal hemorrhage was seen above the large layered preretinal hemorrhage (Fig. 1). A small stream of preretinal blood, connected with the layered preretinal hemorrhage below, originated from a rupture site in the vessel wall, which was occluded by a small white plug, presumably fibrin. Results of fundus examination of the right eye were unremarkable. A pinpoint hyperfluorescence in the wall of the superotemporal branch vein corresponded to the rupture site on fluorescein angiography (Fig. 2).

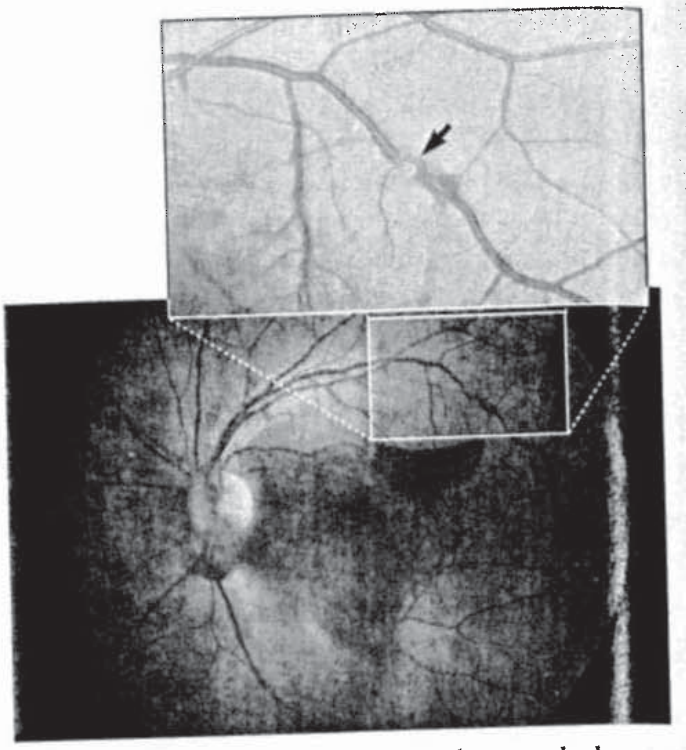

Fig. 1 (Kadrmas and Pach). Fundus photograph shows a large area of layered preretinal hemorrhage superotemporal to the fovea. Enlargement, magnified view of the superotemporal branch vein demonstrates a rupture site in the vessel wall (arrow).

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