Intrapapillary, Peripapillary, and Vitreous Hemorrhage

Dear Editor:

I read with interest the article by Drs. Katz and Hoyt entitled, "Intrapapillary and Peripapillary Hemorrhage in Young Patients with Incomplete Posterior Vitreous Detachment" (*Ophthalmology 1995;102:349–54*). I have evaluated four otherwise healthy Asian patients (range, 18–48 years; average, 40 years) with moderate myopia (−1.50 to −4.25 sphere) and a similar appearance of intrapapillary and peripapillary subretinal hemorrhage. Two of the patients have the exact appearance of the patients described in this article. However, their chief symptoms were floaters, and both had microscopic vitreous hemorrhage on contact lens biomicroscopy. Being previously aware of the theory of vitreopapillary traction postulated by the authors, these patients were evaluated carefully for clinical and ultrasonographic evidence of vitreous traction on the disc. Clinically and ultrasonographically, there was no evidence of prior posterior vitreous detachment, nor was there evidence of vitreopapillary traction. Ultrasonography is a sensitive means of evaluating the vitreoretinal relations and assessing for posterior vitreous detachment.[1] Echography also is useful in demonstrating cortical vitreous traction before posterior vitreous detachment, as in foveal traction in early stages of macular holes.[2] I was unable to demonstrate evidence of cortical vitreous traction on the optic disc in these eyes, which also did not have clinical or ultrasonographic evidence of partial or complete posterior vitreous detachment. In addition, there was no ultrasonographic evidence of disc drusen, which is another possible cause of peripapillary subretinal hemorrhage. Fluorescein angiography at the time of presentation in both patients showed localized disc staining adjacent to the areas of hemorrhage and blocking defects due to intrapapillary and peripapillary subretinal hemorrhage (Fig 1). After resolution of the hemorrhage 4 months later, a repeat angiogram in one patient showed mild residual nasal disc staining with no evidence of vascular abnormalities or other disc abnormalities.

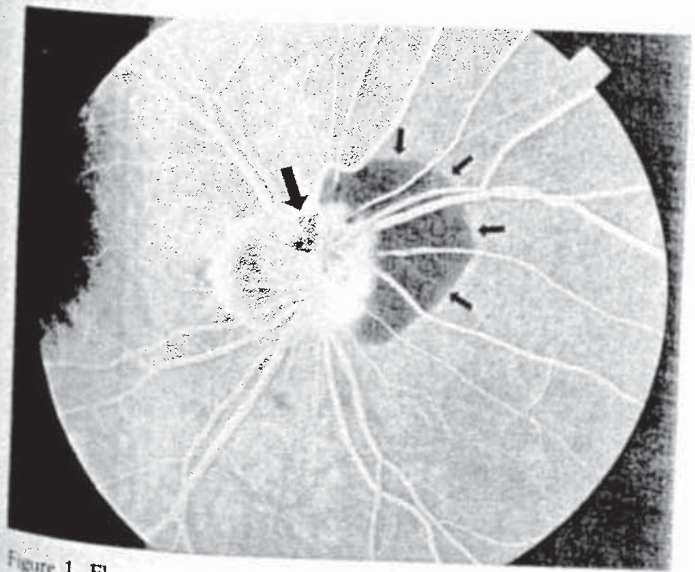

Figure 1. Fluorescein angiogram demonstrates an intrapapillary hemorrhage in the superior aspect of the disc (large arrow) and a nasal peripapillary subretinal hemorrhage (small arrows). Notice the nasal disc staining in this late stage of the angiogram.

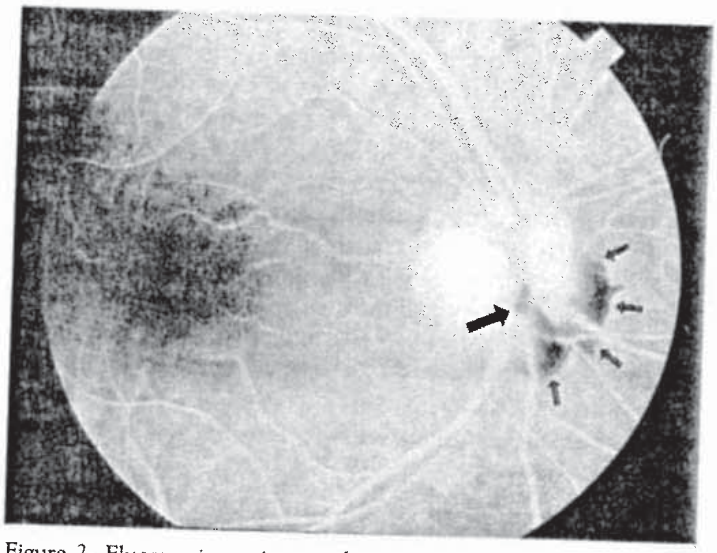

Figure 2. Fluorescein angiogram demonstrates an intrapapillary hemorrhage in the inferior aspect of the disc (large arrow) and a nasal peripapillary subretinal hemorrhage (small arrows). Notice the diffuse and severe disc staining in this late stage of the angiogram. Stellate macular exudates later developed in this eye.

In the two other atypical patients, there was also evidence of intrapapillary and peripapillary subretinal hemorrhage, but there was significant diffuse disc edema, associated with peripapillary serous retinal detachment. Fluorescein angiography showed severe disc leakage in both eyes, as well as blocking defects from the intrapapillary and peripapillary subretinal hemorrhage (Fig 2). Stellate macular exudates, typical for Leber idiopathic stellate neuroretinitis,[3] later developed in both of these patients. These two patients also had no biomicroscopic evidence of vitreopapillary traction or posterior vitreous detachment.

Although these four patients had intrapapillary and peripapillary subretinal hemorrhage, none had clinical or ultrasonographic evidence of vitreopapillary traction. In addition, all four patients had evidence of disc leakage on fluorescein angiography adjacent to areas of hemorrhage. In the series of Katz and Hoyt, only four of the eight patients had fluorescein angiography, but one of these four patients did have evidence of late disc leakage. In addition, two of their patients also did not have any funduscopic evidence of partial posterior vitreous detachment.

My series of four patients, and especially the two patients with findings of intrapapillary and peripapillary subretinal hemorrhage associated with Leber idiopathic stellate neuroretinitis, suggest that inflammatory disc swelling also may be a possible cause of intrapapillary and peripapillary subretinal hemorrhage, unassociated with vitreous traction. Although I agree that vitreous traction may be a possible cause of this syndrome in some patients, other possible causes must be considered, including the

possibility of inflammatory disc swelling. It is possible that these Asian eyes with moderate myopia may have optic discs, which are more susceptible to bleeding by any process which distorts the anatomy of the disc, be it tractional or inflammatory. Alternatively, the inflammatory process causing the disc swelling may be related to a vasculitis involving the prelaminar arterioles,[3] which also may predispose to intrapapillary and subretinal bleeding. I am also impressed by the lack of subretinal or intrapapillary hemorrhage, which occurs during diabetic vitrectomy, in which fibrotic proliferans are peeled from the surface of the disc. This peeling often results in significant surgically induced traction on the disc and peripapillary retina, and evaluation of these membranes often shows the presence of axons on pathologic evaluation.[4] Despite this significant surgical traction on the disc performed in numerous diabetic vitrectomies, many of which were in young Asian patients, I have yet to see evidence of intrapapillary or peripapillary subretinal hemorrhage intraoperatively or postoperatively.

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References

1. Fisher YL, Slakter JS, Freidman RA, Yanuzzi LA. Kinetic ultrasound evaluation of the posterior vitreoretinal interface. Ophthalmology 1991;98:1135–38.
2. Kokame GT. Clinical correlation of ultrasonographic findings in macular holes. Am J Ophthalmol 1995 (in press).
3. Dreyer RF, Hopen G, Gass JDM, Smith JL. Leber's idiopathic stellate neuroretinitis. Arch Ophthalmol 1984;102: 1140–45.
4. Pendergast SD, Martin DF, Proia AD, et al. Removal of optic disc stalks during diabetic vitrectomy. Retina 1995;15: 25–8.

Authors' reply

Dear Editor:

We acknowledge and accept Dr. Kokame's thoughtful comments and observations. We are encouraged by the fact that, being in Hawaii, he has seen this pattern of intrapapillary and peripapillary hemorrhage. When we present our cases where practitioners see many Asians (e.g., in Hawaii, Taiwan, Hong Kong, and Japan), clinicians recognize these disc changes; when we present our cases where fewer Asians seek care (e.g., South America), the audience reports our observations as new to them. As well, we recently learned of case reports similar to ours published in Japan[1,4]; in the most recent study, the causal role of the vitreous was discussed.[3]

Dr. Kokame raises several issues to which we would like to respond. Vitreous surgeons (including Dr. Kokame) have taught us that one cannot tell about the extent of attachment of the posterior vitreous until a vitrectomy is performed. Our clinical skills and ophthalmologic instrumentation do not allow us to make a definitive statement about the nature and extent of posterior attachments. What this means is we are limited by happenstance—when we see a posterior vitreous separation, we know it has occurred; if we do not see it, we do not know that it has not occurred. Our focused examinations of these patients' vitreous bodies have taught us that there is such a thing as a localized partial detachment of vitreous; this may occur over an area of one or several disc diameters; it may be marked by a prepapillary glial tag, though this easily is overlooked. Also, vitreous traction (which is the real culprit here) cannot be seen or imaged. We stand by our conclusions that the hemorrhages and disc elevation in our young Asian patients with myopia were caused by vitreous traction.

Dr. Kokame's patients showed disc staining. As our experience with such patients has increased, we, too, see disc staining on fluorescein angiography. Dr. Kokame's patients showed cells within the vitreous, presumably red cells. Again, we have seen dissection of pre- and peripapillary hemorrhages into the vitreous and the display of mild cells (presumed erythrocytes) there. Dr. Kokame has seen macular star figures in such patients. We have seen patients whose discs we might have accepted as demonstrating vitreous traction, and yet when we see a frank star figure, we have diagnosed them as Leber idiopathic stellate maculopathy. Dr. Kokame may be on to something here.

What do patients with Leber stellate maculopathy have in common with the patients we recorded? We answer that for both groups, discs are small and crowded; discs are elevated; patients have floaters (suggestive of vitreal change); and patients do remarkably well when followed over time. Perhaps there is some subset of patients whose clinical findings we have in the past called Leber stellate maculopathy yet who are really showing us the result of forces of vitreous traction. We, along with Dr. Kokame, will keep an open mind and expect to broaden the spectrum of such vitreopapillary traction syndromes.

If our speculations are correct, vitreous surgeons should be seeing changes at the optic disc before, during, and after vitrectomy. Although Dr. Kokame has not seen these changes, we know other vitreous surgeons have. We have now seen several patients with vitreomacular traction who have undergone surgery for foveal concern; the discs in these patients showed elevations similar to those illustrated in our report, and, after vitrectomy, resolved. We say to Dr. Kokame, "keep looking."

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References

1. Masuda Y, Nakagawa M. Three cases of unilateral disc hemorrhage in teenagers with myopia. Jpn Rev Clin Ophthal 1984;78:368–72.
2. Miki M, Fujioka T. Two cases of spontaneous disc hemorrhage. Jpn Rev Clin Ophthal 1982;76:1542–44.
3. Okubo K, Kawakami J. Optic disc hemorrhage in myopes with posterior vitreous detachment. Ophthalmology (Japan) 1986;28:1515–19.
4. Watanabe C, Shiraki J, Yoshinaga K. Unilateral disc hemorrhage in young people. Ophthalmology (Japan) 1981;23: 241–46.