Submacular hemorrhage from polypoidal choroidal vasculopathy after cataract surgery

Dear Editor,

We read with interest the article by Kim *et al*.,[1] describing a patient with submacular hemorrhage arising from polypoidal choroidal vasculopathy (PCV) which had been diagnosed three years earlier but treated conservatively. This case highlights an important consideration when managing patients with PCV: Some patients may develop sudden onset of submacular hemorrhage, with resultant reduction of visual acuity, after a period of relative quiescence of the disease.

PCV manifests with a variety of clinical presentations.[2] Of the patients with relatively quiescent disease, some are asymptomatic, with subretinal nodules that are detected incidentally, while others, as in this case, experience only mild, non-specific decrease in visual acuity. The natural course of PCV is highly variable, with some responding well to treatment, while others have a chronic course with remissions and relapses.[2,3] There exists controversy whether patients who are asymptomatic or have good visual acuity should be treated, with some authors recommending a conservative approach.[3] Patients such as the lady described by Kim *et al*.,[1] provide a timely reminder that although the clinical outcome of PCV is generally better than age-related macular degeneration,[2] a subset of patients may suddenly develop large areas of submacular hemorrhage,[4] which may be difficult to treat and eventually result in permanent visual loss.

It is interesting to note that the preoperative OCT scan in Figure 1a of the article by Kim *et al*.,[1] shows a small amount of subretinal fluid beneath the fovea, indicating that the disease is not totally quiescent at this time. The factors which may trigger a worsening of PCV remain unclear. The authors postulate that the changes in intraocular pressure during the cataract surgery may have caused a rupture of the abnormal blood vessels. Another patient with macular PCV presented suddenly with two episodes of suprachoroidal hemorrhage, resulting in secondary angle closure, even though there was no known precipitating factor.[5] It has been suggested that systemic factors such as age and hypertension may increase the fragility of the choroidal vessels, making them more susceptible to shearing forces.[5] A similar mechanism may be responsible in the current case,[1] and it would be interesting to know if the patient had hypertension, atherosclerosis or other vascular risk factors.

In conclusion, the authors describe a very interesting case, which highlights that the optimal management of relatively quiescent PCV remains uncertain, and merits further research.

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