Postvitrectomy full-thickness macular hole and retinal detachment in a case of polypoidal choroidal vasculopathy

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DESCRIPTION

A 44-year-old man presented to us with sudden-onset painless diminution of vision in the right eye since the past 2 weeks. The best-corrected visual acuity (BCVA) was hand motions in the right eye and 20/20 in the left eye. Anterior segment examination under slit lamp and intraocular pressures were within normal limits. He was otherwise healthy without any systemic illness, and there was no history of prior ocular or head trauma. Dilated fundus of the right eye revealed presence of vitreous haemorrhage, whereas that of the left eye was unremarkable. Subsequently, pars plana vitrectomy, along with intravitreal antivascular endothelial growth factor (VEGF, bevacizumab 1.25 mg/0.05 mL) was performed; intraoperatively, subretinal and subretinal pigment epithelium (RPE) altered blood with orangish polyp (figure 1A) was noted, which was confirmed to be polypoidal choroidal vasculopathy (PCV) on postoperative indocyanine green angiography, which revealed the presence of a branching vascular network with increasing fluorescence with time (though unable to detect a polyp, figure 2) The BCVA improved to 20/400 with optical coherence tomography revealing the presence of sub-RPE hyper-reflective material with back shadowing and retinal undulations at the second week postoperatively (figure 1B). At the second month of follow-up, the BCVA improved to 20/200 but with a development of full-thickness macular hole with coexistent surrounding posterior pole retinal detachment. The subretinal and sub-RPE altered haemorrhage was noted to be evacuated into the vitreous cavity through the macular hole to leave only a peripheral rim of subretinal haemorrhage at the border of attached and detached retina (figure 1C), which was confirmed on OCT (figure 1D). Internal limiting membrane peeling and fluid drainage, along with gas tamponade, were suggested to the patient; however, he denied any further surgical intervention.

PCV is a pachychoroid spectrum disorder characterised by polypoidal aneurysmal dilations of choroidal vessels presenting as multiple recurrent serosanguinous detachment of the neurosensory retina, pigment epithelial detachment (PED) and spontaneous submacular haemorrhage from rupture of thin-walled fragile choroidal vessels, sometimes even leading to breakthrough vitreous haemorrhage, retinal detachment and hyphema. Anti-VEGF therapy has now become the preferred treatment modality for cases of PCV. Development of macular hole following anti-VEGF injection is an exceedingly rare but reported complication in these patients. In that case, the development of macular hole following anti-VEGF injection was attributed to the anomalous perifoveal vitreous adhesion by Cho et al, with postinjection exacerbation of
traction. Another mechanism postulated was change in tangential shearing force on the retina due to changes in or contraction in the underlying polyvesseloids and neovascular membrane, and reduction in the critical cell–cell adhesive forces between the RPE cells. The same mechanism has also been implicated in pigment epithelium rips after anti-VEGF therapy, especially in cases with tall, tense PED. Baskaran and Pan reported a case of PCV presenting as massive submacular haemorrhage and macular hole presenting simultaneously.

PCV has been known to cause sudden massive subretinal haemorrhage or exudation; the development of macular hole can be attributed to the sudden rise in pressure underneath the retina and the fovea, being the thinnest part, dehiscence of the neural tissue and hole formation. Spontaneous closure of the full-thickness macular hole with anti-VEGF therapy alone has been reported by Lindeke-Myers et al, wherein they postulated the decrease in exudation, settling down of PED, as one of the causes to decrease surface traction and closure of the hole. Chino et al reported delayed-onset macular hole in a case of submacular haemorrhage from PCV, which over a period of observation of 38 months showed spontaneous closure. They ascribed the macular hole formation to the secondary retinal degeneration due to the underlying fibrin and subretinal blood.

The spontaneous closure could be explained by the smaller hole size, no intraretinal cysts and serous macular detachment allowing edges to approximate.

Our case is unique not only in the presentation at a younger age group with massive subretinal haemorrhage along with vitreous haemorrhage but also in the development of macular hole 2 months postvitrectomy with localised retinal detachment involving the posterior pole. Though relatively uncommon, most young patients with PCV exhibit submacular haemorrhage at their initial presentation with poor response to anti-VEGF. The altered subretinal haemorrhage and sub-RPE was noted to have evacuated spontaneously into the vitreous cavity. In the setting of vitrectomised eye, probably the shearing force from the underlying contracting choroidal vascular network and decrease in PED height after anti-VEGF might have led to the macular hole formation. Small macular holes without serious retinal detachment or intraretinal cysts may be observed for spontaneous closure or can be medically managed with anti-VEGF injections alone; however, larger holes with coexistent retinal detachment mandate immediate surgical intervention.

**REFERENCES**