Fugacious foveal hard exudate following full thickness macular hole

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DESCRIPTION

A 66-year-old man, diabetic, hypertensive and dyslipidaemia, presented with gradual onset of blurring of vision in both eyes (OU) since past 3 months. The best corrected visual acuity (BCVA) at presentation was 20/200 in right eye (OD) and 20/160 in left eye (OS). While, the anterior segment examination was unremarkable OU, the dilated fundus examination of OD revealed presence of hard exudate plaque at macula with overlying shiny crystal-like deposits along with multiple cotton wool spots, dot-blot and flame shaped haemorrhages in the posterior pole suggestive of mixed retinopathy (severe non-proliferative diabetic retinopathy along with hypertensive retinopathy) (figure 1A). Similar fundoscopic picture was noted in OS also except for the hard exudate plaque at the fovea. Swept source optical coherence tomography (OCT) line scan passing through fovea in OD showed foveal thinning along with hyper-reflective foci subfoveally with absent photoreceptor layer, juxtafoveal intraretinal hypo-reflective spaces in outer plexiform and outer nuclear layer (figure 1B). OCT line scan passing through fovea in OS revealed presence of juxtafoveal hyper-reflective foci with retinal thickening and intraretinal fluid. In view of deranged glycaemic status, patient was referred to the treating endocrinologist. Approximately 5 months later, patient presented with further drop in vision in OU (BCVA 20/400 OD and 20/200 OS). At this point the diabetic retinopathy had worsened to the proliferative stage in either eye (as confirmed on fluorescein angiography). OD revealed presence of fibrous thick taut posterior hyaloid along the inferotemporal arcade, marked resolution of subfoveal hard exudates with a full thickness macular hole (as demonstrated in OCT) (figure 1C,D). In addition, there was worsening of macular oedema with subfoveal hard exudate deposition in OS.

Secondary macular hole formation in pre-existing diabetic retinopathy is a documented occurrence.[1–3] The proposed mechanisms include tangential traction, intra-retinal exudation along with increased vitreo-macular attachment and tractions[3]; although a multifactorial pathogenesis is more likely to be valid. Extrapolating the concept of sink-hole effect,[4] the existence of the subfoveal hard exudates equates to the presence of long standing intra-retinal oedema. The presence of inferotemporal tangential traction secondary to the thick taut posterior hyaloid could have contributed to the formation of macular hole with subsequent presumed migration of hard exudates into the vitreous cavity in the incumbent case. The persistence of intra-retinal hard exudates and disappearance of subretinal hard exudates subsequent to the macular hole formation also supports the hypothesis of migration into

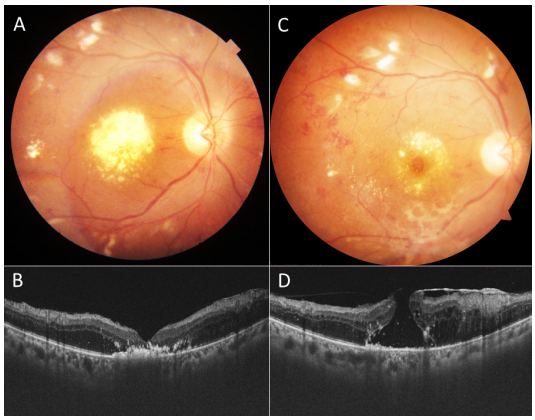

Figure 1 (A) Colour fundus photograph of right eye shows submacular accumulation of hard exudates in a background of mixed retinopathy (severe non-proliferative diabetic retinopathy with hypertensive retinopathy); (B) swept source optical coherence tomography line scan passing through fovea shows foveal thinning along with hyper-reflective foci subfoveally with absent photoreceptor layer, juxtafoveal intraretinal hypo-reflective spaces in outer plexiform and outer nuclear layer; at 5 months follow-up (C) colour fundus photograph right eye reveals presence of fibrous thick taut posterior hyaloid along the inferotemporal arcade, marked resolution of subfoveal hard exudates with a full thickness macular hole; (D) swept source optical coherence tomography line scan passing through fovea shows vitreo-foveal adhesion and vitreo-macular separation with a full thickness macular hole with large detachment area and disappearance of subfoveal hard exudates.

Patient's perspective

I am thankful to the treating physician who made me aware of the progression of diabetic retinopathy in both my eyes along with macular hole formation in my right eye. I was clearly intimated about the immediate requirement for strict diabetes control and pan retinal photocoagulation in both my eyes to prevent worsening of diabetic retinopathy. I was also clearly intimated about the need for vitreoretinal surgery in my right eye for closure of macular hole.

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Learning points

- A typically flat hole, minimal cystic change with large detachment area and thickened pre-macular membrane points towards a macular hole secondary to fibrovascular proliferation.
- Disappearance of foveal hard exudates can take place subsequent to the formation of full thickness macular hole.
- Peculiar feature of macular holes secondary to fibrovascular proliferations is the rapidity of progression to full thickness holes without going through the series of foveal changes as seen in idiopathic ones.

vitreous cavity. To our knowledge, only one similar report has been described in the literature.[2] One peculiar feature of macular holes secondary to fibrovascular proliferations is the rapidity of progression to full thickness holes without going through the series of foveal changes as seen in idiopathic ones.[3] Lai and Yang have reported the presence of a typically flat hole with large detachment area and thickened pre-macular membrane points towards a macular hole secondary to fibrovascular proliferation.[3 5] Post-pars plana vitrectomy with internal limiting membrane peeling for macular hole in patients with proliferative diabetic retinopathy have exhibited contradictory results[2 6]

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