Protective effect of self-settled retinal detachment resulting in asymmetric proliferative diabetic retinopathy

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DESCRIPTION
A man in his late 60s presented to the outpatient clinic with progressive loss of vision in the left eye (OS) for 4 years. His medical history was significant for panretinal photocoagulation (PRP) OS 2 years ago with a history of trauma in the right eye (OD) 15 years ago. Systems history was significant for poorly controlled diabetes mellitus and hypertension. On clinical examination, the best-corrected visual acuity was finger counting OD and 20/60 OS with normal intraocular pressures. Fundus examination revealed greater than 270° vascular attenuation with diffuse areas of the atrophic retina, pigmentary proliferation with a superior island of normal well-demarcated retina (within the dotted inset) OD (figure 1A) and 360° lasered scars with macular exudates and oedema OS (figure 1B). Fundus fluorescein angiography (FFA) OD revealed areas of hypofluorescence due to overlying pigment and areas of hyperfluorescence corresponding to window defects or late staining with a well-demarcated superior island of normal retinal fluorescence (figure 2A), while OS revealed microaneurysms at the posterior pole with leakage and laser marks (figure 2B). Ocular coherence tomography was suggestive of diffuse disruption of the outer retinal layers, degeneration of retinal pigment epithelium with hypertransmissibility of the underlying thinned choroidal vasculature and overall thinning of the fovea (figure 3A) OD, whereas OS revealed macular oedema with exudates (figure 3B). Based on the above-mentioned descriptions, a diagnosis of asymmetric proliferative diabetic retinopathy secondary to retinal atrophy OD stemming from post-traumatic self-settled retinal detachment was made. The patient was administered intravitreal bevacizumab OS 3 weeks following which focal laser of the extrafoveal leaks was done. PRP augmentation OS was performed owing to significant capillary non-perfusion areas on FFA with persistent neovascularisation elsewhere. The patient was advised strict metabolic control.

Diabetic retinopathy is the most common cause of preventable blindness in the working-age group.1 Classically, asymmetric manifestations of diabetic retinopathy have been attributed to causes of reduced vascular supply upstream from the ophthalmic artery, most commonly, stenosis of the carotid arteries.2 Therefore, an evaluation...
of the upstream vasculature in the ophthalmic and carotid circulation becomes a mandate because the vascular insult, if present, tends to be egregious resulting in significant cardiovascular morbidity and mortality. However, apart from the vascular aetiology, other causes of reduced retinal metabolism from degenerative/dystrophic changes, both acquired and congenital, provide a prelude to asymmetric manifestations of diabetic retinopathy (figure 4). Bilaterally symmetric retinal disorders vis a vis self-settled retinal detachment, affecting a large panretinal de-facto scar reduced the retinal metabolism thus mitigating the effects of diabetes mellitus in the contralateral eye resulting in the clinical manifestations of asymmetric proliferative diabetic retinopathy. An extensive fundus examination to look for an underlying cause of reduced retinal metabolism including high myopia, optic atrophy, chorioretinal scars, vitreoretinal surgery and retinitis pigmentosa amongst others should hence be performed in such cases. All cases of asymmetric diabetic retinopathy, therefore, cue for an extensive evaluation for an underlying local and systemic aetiology. The screening so performed could help reduce the morbidity and mortality from cardiovascular accidents if an underlying vascular insult exists.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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