As the patient had new VH and persistent leakage from NVD after aggressive PRP, he was offered intravitreal bevacizumab OD, after a full discussion of its off-label nature and potential risks. Using a sterile protocol,[4] 0.05 cc (1.25 mg) of bevacizumab was injected intravitreally OD.

One week later, vision measured 20/25 + 2 OD, the VH had cleared, and the NVD appeared fibrotic OD. Fluorescein angiography (Figure 2) showed dramatic regression and cessation of leakage from NVD.

Comment

This case illustrates rapid cessation of leakage from NVD 1 week following adjunctive intravitreal bevacizumab. Although it may be argued that PRP caused regression of NVD, examination and fluorescein angiography 5 weeks after PRP showed new VH and persistent leaking NVD, which stopped leaking 1 week after intravitreal bevacizumab. As PRP is the standard of care for high-risk PDR,[5] it was only after maximal PRP that we offered the patient off-label adjunctive treatment. Further studies are needed to determine the role of bevacizumab in the management of PDR.

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Sir,

Epipapillary isolated astrocytoma associated with branch retinal artery occlusion in a single eye

Astrocytomas are congenital glial tumours of the sensory retina that are usually seen in patients with tuberous sclerosis with well-described characteristic features.[1–4] Various ocular complications, including vitreous haemorrhage and seeding,[1–4] subretinal

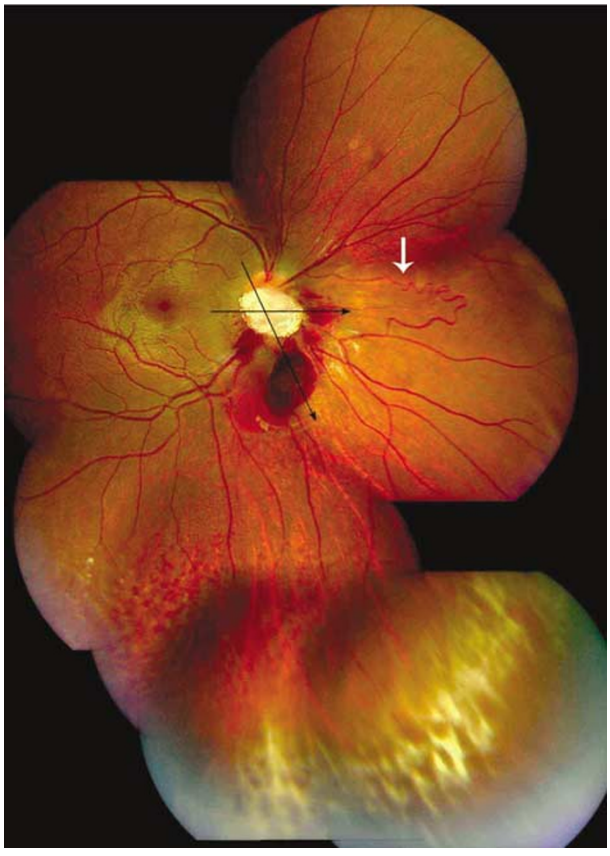

Figure 1 Colour fundus photograph (montage) of the right eye of the patient showing the epipapillary astrocytoma, with a blotch of sub-ILM haemorrhage inferiorly. Also seen are the surrounding superficial retinal haemorrhages and vitreous haemorrhage inferiorly. The collateral between the superonasal and inferonasal branch retinal arteries (white arrow) is also seen. The horizontal and oblique black arrows correspond to the direction of the line scans of Figures 3a and b, respectively.

haemorrhage,[1] and retinal vascular abnormalities (including telangiectasia, neovascularization, and exudation)[2] have been reported in association with such tumours. However, vascular occlusions have not been reported as an association, to the best of our knowledge (MEDLINE search). We report a case of branch retinal artery occlusion (BRAO) in an eye with astrocytoma.

Case report

A 12-year-old boy presented to our hospital with complaints of floaters in the right eye for the past 1 month, following lifting a heavy bucket of water. Ocular examination revealed a best-corrected visual acuity of 20/20 in both the eyes with normal anterior segments. The fundus examination of the right eye revealed a calcified astrocytoma overlying the optic nerve head with a huge blotch of subinternal limiting membrane (ILM) haemorrhage inferior to the disc and surrounding patches of superficial retinal haemorrhages and old vitreous haemorrhage inferiorly (Figure 1a). There was an artery-to-artery collateral in the nasal retina, between the superonasal and inferonasal branch retinal arteries that appeared very narrow as they emerged from underneath the tumour. The tumour demonstrated autofluorescence (Figure 2a). Fluorescein angiography (FA) was performed, which in the early stages revealed the collateral, with a brisk filling of the involved arteries suggesting a well-compensated BRAO. The tumour remained relatively hypofluorescent (Figures 2b and c). It was only in the late recirculation phase (>45 s) that the tumour showed hyperfluorescence owing to extensive leakage from the tortuous tumour vessels (Figure 2d). Optical coherence tomography demonstrated the shadowing corresponding to the intralesional calcification of the tumour and the sub-ILM haemorrhage (Figures 3a and b). The fundus of the left eye was normal. Detailed investigations revealed none of the primary, secondary, or tertiary features of tuberous sclerosis[5,] which was therefore ruled out in the patient. The patient remained stable with absorption of the haemorrhages over 6 months of observation.

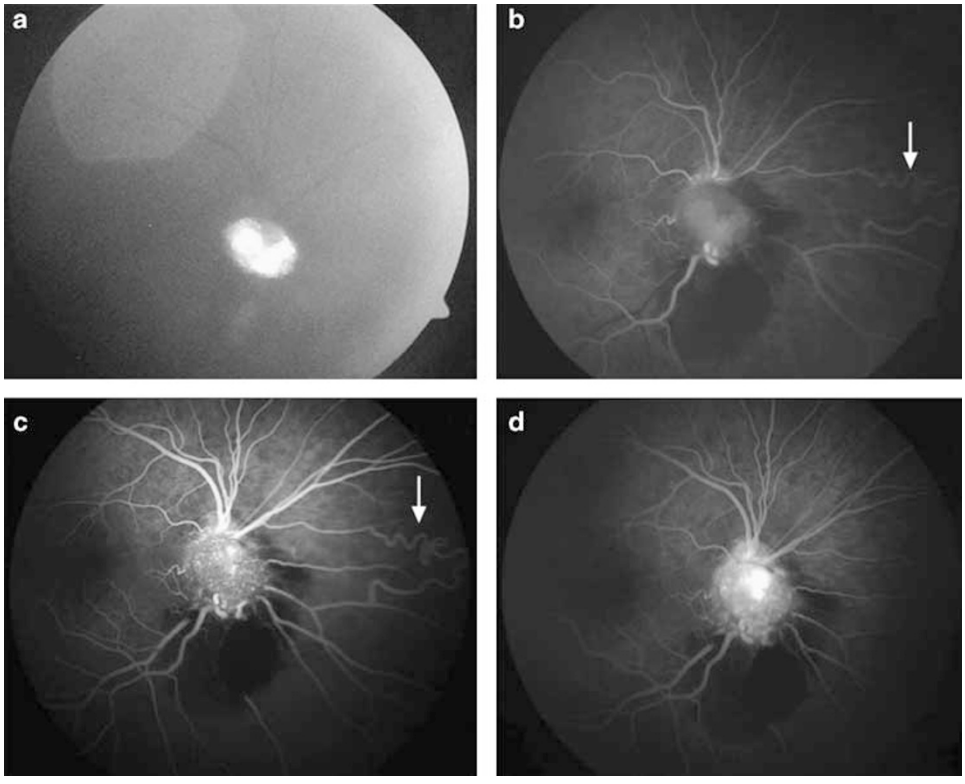

Figure 2 (a) Prefluorescein injection photograph with both the exciter and barrier filters in place, demonstrating the autofluorescence of the tumour. (b) Fluorescein angiogram (arteriovenous phase) showing the collateral (white arrow), with a brisk filling of the involved arteries suggesting a well-compensated BRAO and a relatively hypofluorescent tumour. Also seen are areas of blocked fluorescence owing to the sub-ILM and superficial retinal haemorrhages. (c) Fluorescein angiogram (transit phase) showing the collateral (white arrow) and the slowly increasing hyperfluorescence of the tumour. (d) Fluorescein angiogram (late recirculation phase; >45 s) showing marked hyperfluorescence owing to extensive leakage from the tortuous tumour vessels.

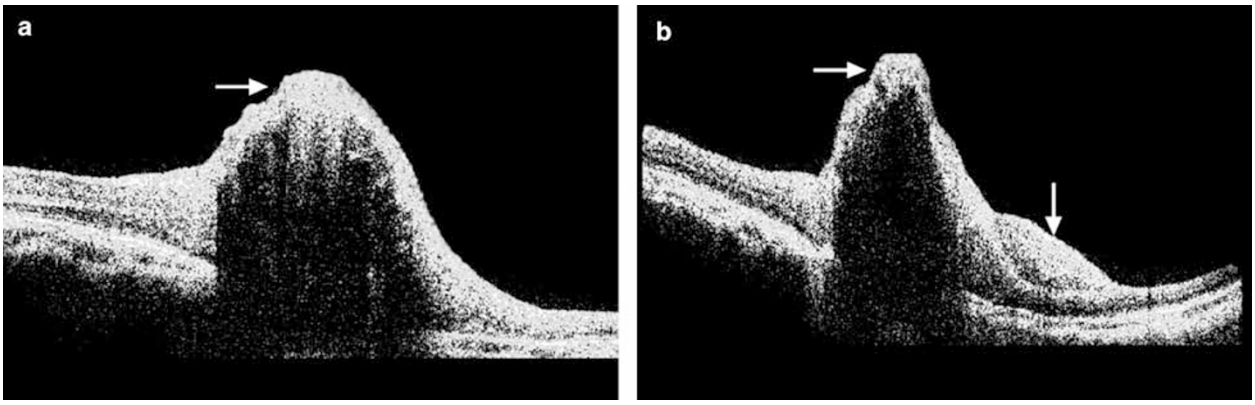

Figure 3 (a) Optical coherence tomogram (line scan corresponding to the horizontal black arrow of Figure 1 through the tumour overlying the optic disc) demonstrating the tumour with shadowing of the underlying optic nerve head owing to intralesional calcification (white arrow). (b) Optical coherence tomogram (line scan corresponding to the oblique black arrow of Figure 1 through the tumour overlying the optic disc and the sub-ILM haemorrhage) demonstrating the tumour with shadowing of the underlying optic nerve head (horizontal white arrow) and the high reflective material under the ILM (vertical white arrow) suggestive of sub-ILM haemorrhage.

Comment

Astrocytomas demonstrate a slow hyperfluorescence despite extensive vascularity.[1] This is suggestive of a sluggish circulation in the tumour vessels, in all probability owing to their corkscrew-like tortuosity with a consequently raised hydrostatic pressure. The Valsalva-like mechanism in our patient owing to lifting of a heavy weight could have further increased the vascular pressure leading to rupture of the small vessels on the tumour surface, leading to vitreous, superficial retinal, and sub-ILM haemorrhages.

The pathogenesis of the arterial occlusion in our patient could also be due to the slowed circulation in the tumour vessels that increase the chances of vascular occlusion. We were unable to find previous reports of BRAO in association with astrocytoma (Medline search). Extensive sheathing of a branch artery as it coursed through an astrocytoma alone has been noted previously.[3]

This report highlights the fact that patients with even isolated astrocytomas are at an increased risk of vascular decompensation, especially if involved in activities that are associated with a Valsalva-like mechanism and need appropriate counselling in this regard.

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