nature and significance of PAS patterns and contend the patterns described are based not on microcirculation but fibrovascular tissue. Foss *et al.*[10] ascribed the patterns to three underlying factors: (1) disordered growth, (2) emergence of rapidly growing subclones and (3) section orientation. The first two factors have prognostic significance.[10]

There is evidence to suggest that the inflammatory response and spontaneous necrosis of melanoma may have an immunological basis.[6,7] Melanoma cells contain tumour-specific antigens and tumour-associated antigens, which act as a stimulus for the immune system.[6,7] The tumour-specific antigens are recognised by antigen-specific receptors on T lymphocytes and are associated with cell-mediated immunity. The cellular infiltration mediated by the immune response is most marked in the immediate vicinity of blood vessels, in contrast to that observed in ischaemic necrosis.[6]

A relatively large mass of necrotic tumour can incite a non-specific response and the chemical mediators and cytotoxic products released by the inflammatory infiltrates also cause direct cellular damage, vasculitis and thrombosis inciting scleritis.[3]

It may be postulated that the scleritis is immunologically induced from the locally produced tumour antigens (type IV delayed hypersensitivity reaction). The cellular infiltrate in granulomatous types of scleritis consists of lymphocytes, plasma cells, macrophages and giant cells but not usually polymorphs.[11] However, our case did not show a lymphocytic response. The cellular infiltration was predominantly polymorphonuclear leucocytes. This may be related to a type III reaction due to immune complexes precipitating within the sclera. Another possible explanation is that tumour antigen liberated by the necrotic melanoma permeates the sclera overlying the tumour forming antigen-antibody complexes locally, as evidenced by the scleritis and necrosis being most marked overlying the tumour. In type III reaction the polymorphonuclear leucocytes predominate and excite an inflammatory response.[11] This may explain the unusual feature of acute scleritis with predominant polymorphonuclear infiltration and scleral necrosis observed in our patient with necrotic malignant melanoma in the absence of scleral invasion by the tumour.

Intraocular haemorrhage may be an associated finding in uveal melanomas, originating either from tumour necrosis or spontaneously.[4] Spontaneous intraocular haemorrhage may precipitate an acute rise in intraocular pressure resulting in stagnation of blood flow, hypoxia and tumour necrosis. In the case we report the possible source of haemorrhage is likely to be tumour necrosis, as the intraocular pressure was less than 38 mmHg at the time acute presentation.

Eyes with scleral necrosis and raised intraocular pressure are more vulnerable to globe perforation with orbital spillage during enucleation and extra caution should be exercised to prevent this complication from occurring.

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Sir,

Choroidal ischaemia and serous retinal detachment in toxaemia of pregnancy

Deterioration of vision is not uncommon during both normal and complicated pregnancies. Serous retinal detachment is a known ocular complication of toxaemia of pregnancy.[1,2] However, serous retinal detachment with fluorescein angiographic evidence of choroidal ischaemia without retinal vascular change is very rare. We report a case of choroidal ischaemia and serous retinal detachment in the absence of retinal vascular changes.

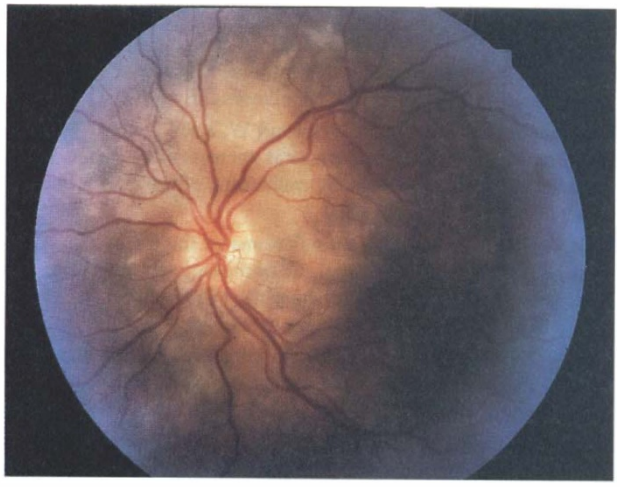

Fig. 1. *Left fundus picture showing serous retinal detachment. Absence of haemorrhages, soft exudate and arterial spasm is a notable feature.*

Case report

A 30-year-old primigravid developed blurring of vision, 4 days after Caesarean delivery of triplets at 28 weeks. She had been treated for moderate pre-eclampsia. Visual acuity was 6/24 in the right eye and 6/36 in the left eye. Fundal examination revealed bilateral choroidal effusion affecting the posterior poles with overlying shallow serous retinal detachment (Fig. 1). There were no retinal haemorrhages, cotton wool spots or arterial constriction in either eye. Fluorescein angiography showed areas of poor choroidal filling, with late staining, but the retinal vasculature appeared normal (Fig. 2). The vision improved to 6/12 in both eyes with complete ophthalmoscopic resolution of retinal detachment within a week. Two months later the vision improved to 6/6 in both eyes leaving residual retinal pigmentary changes.

Comment

Toxaemia of pregnancy is characterised by hypertension, generalised oedema and proteinuria commonly occurring during the third trimester of pregnancy. Serous retinal detachment in pre-eclampsia is well documented but it is usually associated with retinal arterial spasm, retinal haemorrhages and cotton wool spots.[1–3] Absence of retinal circulation involvement as in the case we report is very rare.

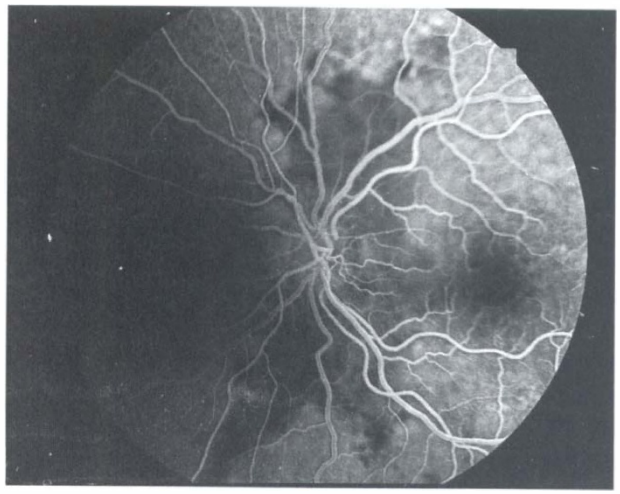

Fig. 2. *Fluorescein angiogram of the same eye in the venous phase showing areas of choroidal non-filling. The retinal vessels appear normal.*

The exact mechanism of serous retinal detachment in toxaemia of pregnancy has not been fully explained. Both the choroid and the retinal circulation have been implicated as a source of the subretinal fluid.[1,2] In addition to toxaemia of pregnancy, choroidal ischaemic infarcts (Elschnig's pearls) may be associated with malignant hypertension and terminal renal failure.[4,5] These choroidal ischaemia infarcts are known as acute triangular syndromes because of their three-sided appearance, which arises from acute occlusion of choroidal capillaries and small arterioles.[4,5]

Choroidal ischaemia involves the retinal pigment epithelium, causing breakdown of the blood-retinal barrier, resulting in leakage of proteins and fluid through the retinal pigment epithelium.[1,2,4] The fluid accumulates in the subretinal space producing serous retinal detachment. Summarising the mechanism of 'hypertensive choroidopathy', Hayreh[6] postulated free leakage of endogenous vasoconstrictor agents such as angiotensin II, adrenaline and vasopressin from the choriocapillaries into the choroidal interstitial fluid. These vasoconstrictors act on the walls of the choroidal vessels, resulting in choroidal vasoconstriction and subsequent ischaemia. Three detailed fluorescein angiographic reports in the literature relating toxaemia of pregnancy and serous retinal detachment suggest that serous retinal detachment is secondary to choroidal involvement.[1–3] The present report provides further evidence that the retinal detachment can be secondary to choroidal damage. Why the mechanisms affecting the choroidal circulation spared the retinal circulation cannot be explained and need further observations.

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