Sir,

Bilateral serous retinal detachment as a complication of HELLP syndrome

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Bilateral, serous, non-rhegmatogenous retinal detachment is a rare complication of toxaemia of pregnancy. In the vast majority of the cases the detachment occurs concomitantly with hypertensive retinopathy.¹ We report a primigravida with preeclampsia and HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome who in the absence of any hypertensive retinopathy, developed bilateral, bullous retinal detachments in the puerperium.

Case report

A 34-year-old primigravid patient experienced bilateral blurring of vision following Caesarean delivery at 26 weeks. She had the Caesarian section for preeclampsia, which she had been treated for the previous 3 months. On the first day post partum, she developed features consistent with HELLP syndrome.

Her past ophthalmic history included left amblyopia due to anisometropia with best-corrected visual acuity of 6/5 and 6/9 in the right and the left eye respectively, as recorded by her optometrist 11 months before the delivery.

On examination the first day post partum her visual acuity was 6/24 and 6/60 in the right and left eye respectively. Anterior segment examination was unremarkable, however fundoscopy revealed bilateral serous retinal detachments affecting predominantly the posterior pole and the peripapillary area. (Figure 1). There were no clinical signs of hypertensive retinopathy. Spontaneous resorption of the subretinal fluid started promptly 2 days after the delivery leaving mild residual mottling of the retina secondary to pigment epithelial changes. Complete resolution of the retinal detachments (Figure 2) occurred within 2 weeks and 1 month post partum in the right and the left eye respectively, resulting in recovery of the visual function to pre-detachment levels.

Comment

Retinal detachment is an unusual but well documented complication of hypertensive disorder in pregnancy affecting 0.2–2% of patients with severe preeclampsia and 0.9% of patients with HELLP syndrome.² However absence of retinal circulation involvement as in our case is rare with only a few anecdotal cases.^{3,4}

The precise pathogenesis of serous retinal detachments in preeclampsia remains elusive. Fluorescein angiographic and indocyanine green observations support the hypothesis that retinal detachments are secondary to choroidal ischaemia.⁵ Hayreh suggested that in hypertensive choroidopathy endogenous vasoconstrictor agents leak freely from the choriocapillaries and act on the walls of the choroidal vessels resulting in choroidal vasoconstriction and ischaemia. Subsequently ischaemia of the RPE causes degradation of the outer blood-retinal barrier and formation of a serous proteinaceous exudate from the choroid, through the RPE, into the subretinal space, producing serous retinal detachment.⁶

It has also been postulated that placental thromboplastin may release into maternal circulation and activate the extrinsic coagulation system with



Figure 2 Spontaneous resolution of serous retinal detachment leaving mild residual mottling of the retina attributable to pigment epithelial changes.

resultant disseminated intravascular coagulation. This may be responsible for choroidal ischaemia and consequent serous retinal detachment.⁷

Spontaneous serous retinal detachment in toxaemia of pregnancy can occur both ante partum and post partum causing marked reduction in visual acuity. In most cases the detachment resolves with a return to normal visual function within the first few weeks post partum. However, some patients may develop residual macular retinal pigment epithelial change, which may represent areas of infarction of the choriocapillaries (Elschnig's spots). These changes can mimic a macular dystrophy or tapetoretinal degeneration and infrequently may result in permanent visual impairment.⁸

Our case points out that retinal detachment should always be considered within the differential diagnosis of sudden loss of vision in cases complicated with HELLP syndrome and more generally toxaemia of pregnancy.

It is useful for the ophthalmologists to be aware that retinal detachment may present in the absence of other hypertensive retinal changes.

Finally the favourable prognosis and natural course of this clinical condition should always be emphasised to the patient relieving them from unnecessary distress.

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Sir,

Vitreous amyloidosis and secondary glaucoma—a case report

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We present a case of familial ocular amyloidosis currently without systemic involvement. Ocular features included vitreous opacities and secondary glaucoma. The patient had a transthyretin mutation (TTR-glycine 54) previously reported in his family only.

Case report

A 38-year-old man presented to Eye Casualty with a 7week history of floaters in both eyes. The patient's father and paternal uncle both died from systemic complications of amyloidosis. His paternal grandmother was blind 20 years prior to her death. The family history is plotted in Figure 1.

The visual acuities were 6/5 and 6/9 in the right



Figure 1 Gene sketch of the family.