busy eye clinic. In this case, considerable delays in establishing diagnosis could have been avoided.

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Eye (2006) **20,** 946–948. doi:10.1038/sj.eye.6702057;
published online 19 August 2005

Sir,

Recurrent branch retinal vein occlusion with factor V leiden mutation

The normal homeostasis between procoagulant and anticoagulant activities in the body is a delicate balance of a large number of plasma factors. Although symptomatic retinal venous occlusion (RVO) is a common disorder among population 65 years or older, recurrent RVO is less frequently encountered. Recurrent central RVO due to factor V mutation has been reported in the literature.[1,2] Here we report a case of recurrent branch retinal vein occlusion in a patient with mutation of clotting Factor V gene.

Case report

A 60-year-old white male was urgently referred from the optician with complaint of sudden impairment of vision in the left eye. The presenting visual acuity was 6/5 in the right eye and 6/18 in the left eye. Ocular examination revealed an infero-temporal branch retinal vein occlusion with macular oedema in the left eye. Assessment of risk factors revealed him to be a hypertensive on regular antihypertensive medication. As a part of initial work-up for venous occlusion, we asked for full blood count, glucose estimation, and lipid profile evaluation—all of which came as normal. He was advised regular blood pressure check-up and we started him on Aspirin 75 mg, once daily. Fundus fluorescein angiography carried out 4 months later showed small areas of capillary dropout along the blocked vessel with resolving macular oedema. No neovascularisation was noted on the disc or elsewhere. Subsequent follow-up showed spontaneous resolution of macular oedema, well-developed collateral circulation, and visual acuity in the left eye improved to 6/5. He was discharged from specialist care. The patient was referred back to us about 18 months later with similar complaints in the left eye. The presenting visual acuity was 6/5 in the right eye and 6/12 in the left eye. This time he was found to have supero-temporal branch vein occlusion in the left eye with macular oedema. His blood pressure was well under control with felodopine and he was taking aspirin regularly. We were unable to elicit any evidence of thromboembolic disease on past history or systemic examination. We ordered repeat full blood count, glucose, and lipid estimation as well as tests for anticardiolipin antibody, ANCA, Clotting profile, plasma homocysteine level and plasma electrophoresis. All the above investigations came as normal except slightly prolonged activated partial thromboplastin time (APTT). A carotid doppler scan did not reveal any stenosis on either side. He was referred to the haematologist and a thrombophilic screen was performed. The result showed modified activated protein C (APC) resistance consistent with Factor V Leiden mutation. Considering the increased risk of future thrombo-embolic events, he was started on warfarin prophylaxis. The patient is asymptomatic presently with vision of 6/5 in both eyes. Repeat fluorescein angiogram showed scattered areas of capillary dropout, well-developed collaterals, no macular oedema and no neovascularisation. There have been no further thrombo-embolic episodes during the last 18 months of follow-up.

Comments

Retinal venous occlusion (RVO) is the second most common cause of reduced vision due to retinal disease with branch vein occlusion being about 2–3 times more common than central venous occlusion. More than half of the patients of RVO are over 65 years. Numerous acquired and hereditary risk factors have been identified for venous thrombosis. The main acquired risk factor for RVO is hypertension and it is more commonly associated with branch vein occlusion. Younger patients with central RVO need to be investigated regarding use of oral

contraceptives, optic disc vasculitis and thrombophilic factors. A number of coagulation disorders increase the risk of systemic venous thrombosis,[3–9] Factor V Leiden being the most frequently identifiable cause.[7] The Factor V gene is located in the short arm of chromosome 1. It acts as a cofactor for activation of prothrombin by factor Xa. Factor V Leiden is a thrombophilic genotype characterised by homozygosity or heterozygosity for a point mutation in the factor V gene, where glutamine is substituted for arginine at position 506. The resultant mutation makes factor V resistant to degradation by APC, thereby increasing thrombotic tendency. Factor V Leiden is responsible for about 95% cases of APC resistance.[6] It is found in 1–7% of Caucasian alleles.[8] It is already known to be a risk factor in systemic thromboembolism including deep venous thrombosis. Although this patient had hypertension as an important risk factor for developing RVO, the recurrence of RVO in spite of adequate blood pressure control prompted us to investigate for other uncommon identifiable causes. This case demonstrates the need of thrombophilic screening in patients with recurrent venous occlusion.
Haematological opinion should be sought in cases of young patients, patients with history of systemic thrombo-embolic disease, family history of thromboembolism, recurrent foetal loss. Timely institution of antithrombotic therapy may help prevent further potentially fatal thrombo-embolic accidents in these cases.

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Eye (2006) **20,** 948–949. doi:10.1038/sj.eye.6702060; published online 19 August 2005

Sir,

Diagnosis of pre-existing posterior capsule defect in traumatic white mature cataract with intact anterior capsule

In cases of blunt ocular injury, cataract may result from the impact of trauma with or without anterior and/or posterior capsular defect. We report an intralenticular sign to anticipate a pre-existing posterior capsule defect (PPCD) in white mature cataract with an intact anterior capsule following blunt trauma.

Case report

A 17-year-old male presented with the history of trauma to the left eye with a plastic ball 10 days back and progressive dimness of vision since then. He had an accurate projection with normal pupillary reactions. Slit-lamp examination revealed a white mature cataract with intact anterior capsule and a 'sinking cortex' sign. Intraocular pressure was normal. Fundus details were not visible. A-Scan examination was un-confirmatory. B-Scan ultrasonography revealed floating vitreous echoes of moderate density just behind the posterior capsule (Figure 1). Right eye examination revealed no abnormality on detailed slit-lamp examination after dilatation of pupils.

At the time of surgery, on the following day, slit-lamp examination on the operating table also revealed 'sinking cortex' sign (Figure 2a). There was no postural difference in the appearance on slit-lamp examination in sitting