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CORRESPONDENCE

Sir,

Central serous chorioretinopathy and antiphospholipid antibodies—results of a pilot study

Central serous chorioretinopathy (CSCR) normally occurs in otherwise healthy individuals. It is characterised by the development of a serous detachment of the neurosensory retina at the macula. Bilateral disease has been reported in about 23% of cases.[1] The condition is normally self-limiting with good visual recovery. However, up to 30% of cases may develop recurrent episodes and require photocoagulation therapy.[2] Stress has been implicated, but not conclusively proven to be an aetiological factor in CSCR.[3] The aetiology of CSCR has been the subject of much speculation, but remains unknown.

There is evidence of abnormal perfusion of the choriocapillaris in CSCR.[4–6] There is also evidence of an association between serous retinal detachment and raised serum antiphospholipid antibodies.[7] Furthermore, raised antiphospholipid antibodies are seen in 42% of patients with systemic lupus erythematosus (SLE)[8] and an association between SLE and CSCR has been observed.[7,9,10] Antiphospholipid antibodies are associated with thrombophilia[11,12] and could, in theory, lead to localised thrombosis in the choriocapillaris resulting in the perfusion abnormalities found in CSCR. We performed a pilot study to begin to investigate possible evidence for a direct association between CSCR and raised levels of antiphospholipid antibodies (anticardiolipin, and anti-β2 glycoprotein-1). The prevalence of such antibodies in the normal population is reported to be approximately 6%.[13]

A total of 20 patients (13 M, 7 F) presenting with a clinical diagnosis of CSCR to one unit between September 1999 and December 2000 underwent analysis of their clotting profiles and serum testing for the presence of antiphospholipid antibodies. General health was assessed and any features specific for the primary antiphospholipid syndrome were recorded.

None of the patients who presented had clinical features of the primary antiphospholipid syndrome. Two patients showed minor abnormalities in their clotting profiles. In one patient, this was transient and the other was lost to follow-up. No patients had persistently elevated levels of antiphospholipid antibodies, although IgM anti-β2 glycoprotein-1was transiently elevated in one patient.

In conclusion, this study did not suggest an association between raised antiphospholipid antibodies and central serous chorioretinopathy. It would appear that antiphospholipid antibodies are unlikely to be directly implicated in the aetiology of CSCR.

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