Sir,
Antiphospholipid antibody-associated choroidopathy

We here report a case of bilateral choroidal infarctions due to catastrophic antiphospholipid antibody syndrome.

Case Report

A 47-year-old male suffering a catastrophic antiphospholipid antibody syndrome (CAPS) flare with hepatic and splenic infarctions developed bilateral, painless vision loss. The patient's best-corrected visual acuity was 20/60 in the right eye and 20/200 in the left eye. Intraocular pressures were 16 mm Hg in both eyes. Anterior segment examination revealed 3+ periorbital edema, 4+ bilateral chemosis and bilateral scleral icterus. Bilateral fovea involving serous retinal detachments were noted on posterior segment evaluation. The patient was treated with intravenous Eculizumab (900–1200 mg × 8), intravenous Rituximab (1000 mg × 2), oral prednisone (30 mg daily), plasmapheresis (seven rounds of 1.25–1.50 × plasma volume), and hemodialysis (3 × /week) for his CAPS with eventual resolution of his retinal detachments. He was eventually discharged on warfarin (goal international normalized ratio (INR) of 2.0 to 3.0) and oral plaquenil (200 mg twice daily).

Three months after initial evaluation, his visual acuity was 20/20 − 1 in both eyes. His intraocular pressure and anterior segment examination were unremarkable. Though his retinal detachments remained resolved, numerous wedge-shaped chorioretinal pigmentary

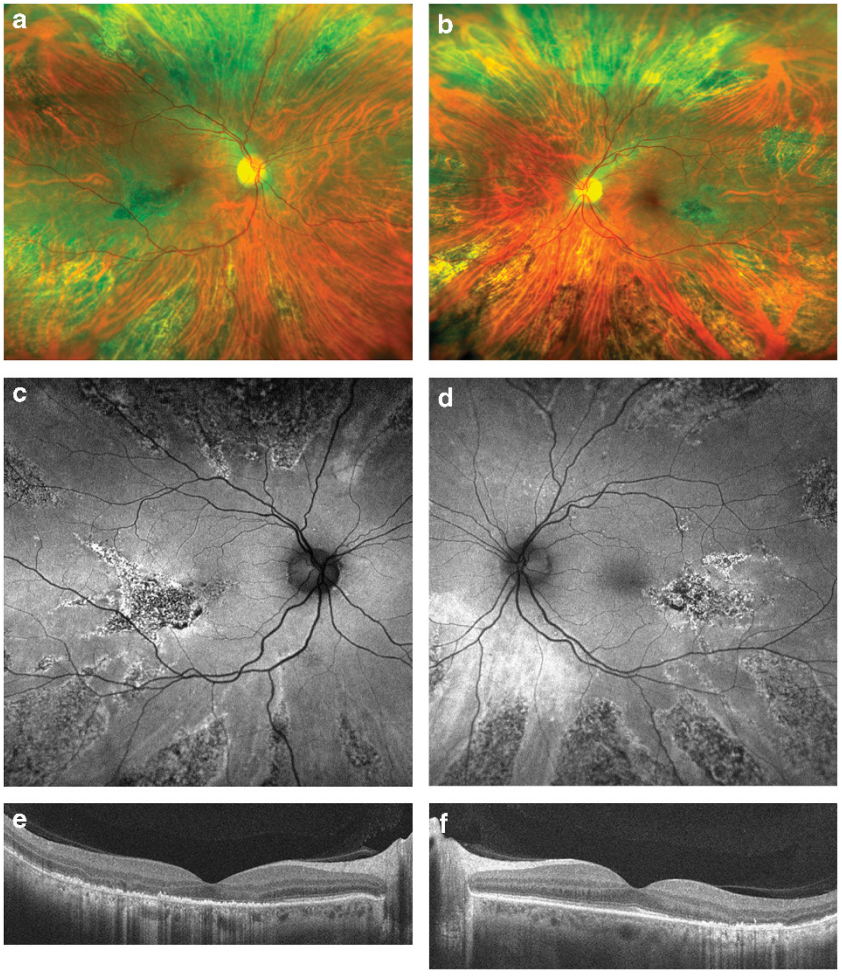

Figure 1 (a and b) Fundus photographs on 3-month follow-up. Optos wide-field posterior segment image of the right eye (a) is notable for peripheral, radial wedge-shaped regions of depigmentation with overlying retinal tessellation extending into the central macula. Posterior segment image of the left eye (b) demonstrates a similar appearance. (c and d) Autofluorescence of the right (c) and left eyes demonstrate regions of stippled hypoautofluorescence. Regions of hyperautofluorescence are also seen, most prominent inferonasal to the optic nerve (d). (e and f) Ocular coherence tomography (OCT) of the right (e) and left eyes (f) demonstrate relatively normal inner retinal architecture. The external limiting membrane and photoreceptor inner segment/outer segment lines appear sporadically disrupted: temporally in both eyes with extension subfoveally in the right eye. Both eyes also demonstrate retinal pigment epithelium (RPE)/Bruch's membrane complex thinning with hyperreflective material overlying this layer and increased choroidal reflectivity underlying it.

changes with a tessellated appearance of the overlying retina were noted (Figures 1a and b). These regions demonstrated stippled hypoautofluorescence (Figures 1c and d) and regions of inner neurosensory retinal loss, RPE disruption, and attenuated choroid layers on ocular coherence tomography (OCT, Figures 1e and f). Single-flash cone responses (light-adapted) were reduced by 20% in amplitude bilaterally and 30 Hz flicker responses showed delayed implicit times. Multifocal electroretinography of the right eye demonstrated significant noise whereas the left eye showed very low amplitudes in the inferotemporal retina. Arden ratios on electrooculography were 1.5 in both eyes. The patient's plaquenil was therefore discontinued and he was placed on Eculizumab.

Comment

The 'wedge-shaped' pigmentary changes and their distribution suggests the patient's CAPS flare yielded choroidal ischemia (precipitating serous effusions[1]) and eventual infarction through posterior ciliary artery occlusion. Interestingly, the patient's long posterior ciliary arteries seemed relatively spared with ocular hypotony (from ciliary body ischemia[1]) never noted. Retinal vasculopathy also remained notably absent, perhaps owing to the retina's autoregulatory capacity—a characteristic the choroid lacks.[2]

An important consequence of choroidal and, consequently, RPE impairment in this exceedingly rare APS manifestation[3,4] is that plaquenil, considered a standard therapy for systemic catastrophic APS,[5] should be eschewed given both the RPE's increased susceptibility to toxicity and the poor reliability of screening for such toxicity in these cases.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Anomalies in drug choice in glaucoma clinics

We investigated anomalies in drug choice when prescribing new glaucoma drops in glaucoma clinics. A total of 1436 records were assessed for 6 months. Of these, 115 patients had a change in drop. An independent glaucoma consultant ophthalmologist categorised the drug choice into three using clearly defined criteria: no anomaly/error, anomaly, error.

An anomaly was defined as the prescription of two new drugs simultaneously, prescription of an additional drug without stopping current ineffective drug,[1] prescription of a new drug without considering non-adherence,[2] and prescription change to unorthodox drug frequency.[3] An error was defined as the prescription of a contraindicated drug or a drug with a clearly documented previous adverse drug reaction. Benefit of doubt was given at all times (e.g., multiple changes in drops were considered to be reasonable practice where a pressure rise was an unacceptable risk).

We found that over three quarters of changes in medication had consultant or fellow involvement. Optometrists, registrars, and associate specialists, collectively, were responsible for less than a fifth ($n = 21$) of changes in glaucoma drops.

There was a high standard of clinical practice in 92 (80%) cases. In one-fifth, therapeutic management was considered to be anomalous or erroneous: there were 15 anomalies in management (13%, 95% CI 7–19%) and 8 errors (7%, 95% CI 2–12%). Seven of these were prescribed a drug with a clearly documented previous adverse reaction and one patient was prescribed Timolol despite advice from their cardiologist to avoid beta blockers.

The following risk factors were examined: day of week, time of clinic, patients per clinician, presence of consultant, and staff grade. There was no correlation between these factors and the numbers of errors or anomalies occurring.

Errors are inevitable, however, the magnitude reported here is unacceptably high. The majority may be accounted for by a failure to fully examine hospital records, and changes are needed to assist the clinicians in busy clinics. Electronic records accompanied by decision support reduce errors in prescribing.[4] We are currently working towards this. Another important step is to encourage shared decision making with patients. The results of the study are being introduced into the glaucoma service induction training.

Conflict of interest

The authors declare no conflict of interest.

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