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CORRESPONDENCE

Sir,

Cotton-wool spots and migraine: a case series of three patients

We have observed three cases of isolated cotton-wool spot (CWS) accompanied by a history of migraine, and would suggest a pathophysiological association between these two clinical entities.

CWS are believed to result from acute retinal arteriolar occlusion leading to hypoperfusion and ischaemia of the retinal nerve fibre layer (RNFL). Subsequent impairment of axoplasmic flow causes accumulation of axoplasmic debris in the retinal ganglion cell axons, impairing signal conduction and producing a characteristic CWS lesion. Isolated CWS are usually asymptomatic although may produce a localised non-arcuate or arcuate scotoma.[1,2]

All three patients presented with a new visual disturbance associated with recent migraine. In each case an isolated CWS was identified, corresponding to an area of scotoma. Optical coherence tomography (OCT), fundus fluorescein angiography (FFA), and Goldmann visual field assessment were performed in each case. Blood pressure examination and serum biochemistry/haematology analysis were unremarkable. In all three patients the symptoms and the CWS resolved within 6 months. We describe one example case in further detail.

Case report

A 38-year-old man presented ten days after a severe migraine associated with slightly blurry central vision. Although the migraine and blurriness had quickly resolved, the patient reported a residual right temporal–paracentral field defect. His medical history included recurrent migraines, often involving visual symptoms. He had no other ophthalmic history and took no regular medications.

An isolated CWS between the right macula and optic disc was detected and further investigated (Figure 1a and b). The patient reported a recent increase in coffee consumption and was advised to reduce this. At 6 months the patient's symptoms and the CWS had resolved.

Comment

The clinical manifestations of migraine are believed to result from transient vasospasm of the cerebral vasculature, inducing cerebral hypoperfusion. Our case series suggests vasospasm may also occur within the retinal microvasculature, although the exact mechanism of this may differ. Retinal vasospasm has been observed directly during a migrainous episode, and has also been implicated as a possible cause of branch retinal arteriolar occlusion in the presence of migraine.[3,4] Furthermore, studies have shown patients with migraine to have reduced RNFL thickness, suggesting that, although vasospasm is in itself a transient occurrence, the chronic natural history of migraine may lead to permanent structural changes.[5]

This small case series highlights an association between migraine and CWS, and emphasises the importance of taking a full medical history when assessing patients with sudden onset of visual field defect secondary to CWS.

Conflict of interest

The authors declare no conflict of interest.

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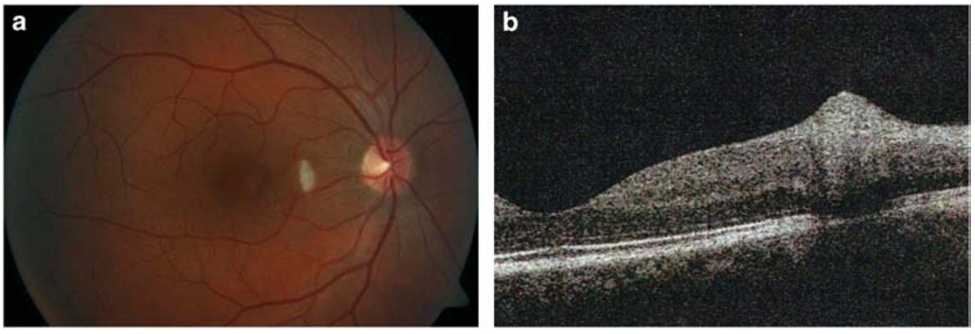

Figure 1 Results of various investigations. (a) Right retinal colour photography of an isolated CWS which is lying between the macula and optic disc and is equidistant to both. Corrected visual acuity was 6/5 and Goldmann visual field assessment highlighted a region of reduced sensitivity immediately inferior to the physiological blind spot. (b) OCT reveals localised swelling of the RNFL at the location of the CWS. FFA showed no signs of retinal vasculitis or ischaemia. Blood pressure measurements, serum biochemistry, and haematology analysis were normal.

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Sir,
Electroretinography can provide objective assessment of inner retinal function prior to atrophic change on OCT

The paper by Yusuf *et al*[1] describing cases of transient artery occlusion following phacoemulsification surgery provides an important addition to the differential for visual loss following cataract extraction, and in their subsequent letter[2] they suggest prospective case finding to establish what risk factors might be associated with this phenomenon. They state that OCT 'may provide the only objective evidence of TRAO, particularly in patients not presenting in the immediate post-operative period'. Unless patients are seen acutely when the characteristic inner retinal thickening may be evident, OCT changes may be quite subtle until inner retinal atrophy develops some time later. A modality that may be helpful in this intermediate period is electroretinography, which provides objective assessment of function, with some localisation of dysfunction. The full-field flash electroretinogram (ERG) can discern inner retinal dysfunction (by selective impairment of the b-wave in comparison with a relatively preserved a-wave, giving an electronegative ERG,[3] and also, more recently described, by reduction of the photopic negative response[4]). Electrodiagnostic testing is not as readily available as OCT, so this may not be always feasible. The development of handheld devices may allow more widespread use,[5] although recordings using these devices may need greater validation. Also, more localised arteriolar insufficiency may not be detected so sensitively by full-field techniques, in which case multifocal electroretinography can be helpful. This highlights the likely added value of using objective tests of retinal function in conjunction with high-resolution imaging of retinal structure; the latter is not always abnormal when function can be markedly impaired.

Conflict of interest

The author declares no conflict of interest.

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Sir,
Transient retinal artery occlusion: the potential utility and limitations of electroretinography

We agree with Mahroo's[1] helpful suggestions on the utility of electroretinography (ERG) in suspected transient retinal artery occlusion (TRAO) cases.

TRAO is a recently proposed clinical entity supported by OCT findings.[2,3] The ERG features of TRAO are yet to be described, and may be sought as part of a prospective case-finding study. Two limitations of Ganzfeld ERG in TRAO are: (1) branch pattern TRAO may not be detected; and (2) b-wave attenuation on ERG reverses fully after 30 min in experimental models of transient retinal ischaemia.[4] ERG evidence of widespread ischaemia may vanish before testing takes place.

However, if there is perimetric or OCT evidence of ongoing retinal ischaemia when ERG is performed, it is likely that ERG abnormalities would be detectable. If the ischaemic changes extend beyond the obviously affected area of the retina, the Ganzfeld ERG might provide evidence of retinal ischaemia in the form of b-wave amplitude reduction and increased 30-Hz photopic flicker implicit time.

The multifocal ERG (mfERG) may provide evidence of localised ischaemic changes and might have contributed to the diagnosis in cases 2 and 3.[2] Branch retinal artery occlusion attenuates the N1, P1, and N2 components in the distribution of ischaemic retina on mfERG.[5] mfERG is capable of identifying wider retinal dysfunction than that suggested clinically.[6] It has been used to demonstrate functional recovery following retinal artery occlusion[7] and to detect subclinical retinal dysfunction in Susac's syndrome.[8] Pattern ERG may also demonstrate reduced amplitude or delayed P50 in cases