Bilateral cilio-retinal artery occlusion in classic migraine

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Summary: A case of bilateral cilio-retinal artery occlusion in a patient with classic migraine is described. It is unlikely that this hitherto unreported occurrence represents altered tone in the dense adrenergic innervation to the posterior ciliary circulation. We advise the continued use of beta-adrenergic blockers as prophylaxis in ocular migraine.

Introduction

Permanent infarctions in ocular migraine usually involve the retinal circulation.^{1,2} Posterior ciliary circulation infarcts are less well reported³ but include one case of unilateral cilio-retinal artery occlusion.⁴ The posterior ciliary vessels and the central retinal artery posterior to the lamina cribrosa are adrenergically innervated.⁵ These factors were believed important in three cases of ocular infarction that occurred shortly after propranolol, a mainstay of migraine prophylaxis, was commenced,⁶ leading to a less frequent use of the drug in ocular migraine. Our patient suffered two separate ocular circulation infarcts, but was treated with propranolol with no adverse effects.

Case report

A 29 year old female suddenly noticed a small scotoma nasal to fixation in her left eye, 3 days after the start of an attack of classic migraine characterized by fortification spectra, headache and nausea. She had developed migraine 7 years earlier and had experienced one attack in the mid-cycle phase of each month ever since. She was taking an oral contraceptive pill. Right and left visual acuities were 6/4. Right and left near vision was N5 and she saw all 17 Ishihara colour plates with each eye. Pupil responses were normal, and there was no relative afferent pupillary defect. Anterior segments were normal and intraocular pressures were 12 mmHg. An absolute wedge shaped field defect was present nasal to fixation in her left eye. Fundoscopy revealed occlusion of the left cilio-retinal artery (Figure 1). The right fundus was normal. Cardiac evaluation was unremarkable, no carotid bruits were heard and there was no evidence

of a collagen vascular disease. Blood count and film, clotting factors including anti-thrombin 3 level, serum fibrinogen and biochemistry were normal. Lupus anti-coagulant was not found. The erythrocyte sedimentation rate was 5, antibodies to rheumatoid factor, antinuclear factor, double-stranded DNA and syphilis serology were negative. Chest X-ray, electrocardiogram and echocardiogram were unremarkable. The oral contraceptive pill was stopped.

Six months later she presented with a scotoma in the right eye, 2 days after the onset of a further severe attack of classical migraine. Right and left visual acuities were 6/4. Near and colour vision remained normal. Both anterior segments were quiet, intraocular pressures were right 16 mmHg and left 14 mmHg. Perimetry showed an absolute field defect infero-nasal to fixation in the right eye, the left field was unchanged.

Examination of the right fundus revealed retinal cloudy swelling in the distribution of the superior

Figure 1 Retinal cloudy swelling due to left cilio-retinal artery occlusion.

branch of a large cilio-retinal artery (Figure 2). Fluorescein angiography showed occlusion of this vessel but dye transit time was normal in the remainder of the retinal vessels (Figure 3). Systemic examination and investigation was again unremarkable. She was commenced on propranolol 20 mg, three times a day and has had no further attacks.

Figure 2 Retinal cloudy swelling temporal to a fixation pointer due to occlusion of the superior branch of a right cilio-retinal artery.

Figure 3 Mid-venous phase fluorescein angiogram, showing complete occlusion of the superior branch of the cilio-retinal artery. There is no evidence of focal arteritis.

Discussion

This patient experienced bilateral cilio-retinal artery occlusion during attacks of classical migraine. Permanent retinal infarcts are well reported in classic migraine and include central and branch retinal artery occlusion, central retinal vein occlusion, central serous retinopathy, vitreous haemorrhage, retinal haemorrhage and transient monocular visual loss.^{1-4,7} Posterior ciliary circulation occlusions are less frequent but include anterior ischaemic optic neuropathy,⁸ retinal pigment epithelial change⁹ and one instance of cilio-retinal artery occlusion.⁴

The posterior ciliary circulation and the retinal circulation proximal to the lamina cribrosa are adrenergically innervated,⁵ and migraine-related infarction in the ocular circulation has been thought to represent reduced adrenergic vasodilator tone with resultant increased vasoconstriction.¹⁰ However, infarctions are well reported in the retinal circulation distal to the lamina cribrosa which is devoid of adrenergic innervation. Furthermore, although regional cerebral blood flow studies have shown reduced blood flow during attacks of classic migraine, evidence that this is due to autonomically mediated vasoconstriction is lacking. In fact, it is more likely that reduced cerebral blood flow is an epiphenomenon occurring secondarily to spreading cortical depression,^{11,12} a wave of altered cortical function and metabolic activity. Local release of vasoactive substances, such as 5-hydroxytryptamine from endothelial cells, platelet release reactions and possibly cerebral arterio-venous shunting may all be contributory factors.¹³

Propranolol is a well established prophylactic in classic migraine although its use in ocular migraine has been questioned recently^{7,14} because it was thought to have precipitated infarction shortly after commencement of therapy, due to its effect on the adrenergic innervation to the ocular circulation. Since there is minimal evidence that this innervation is important in the pathogenesis of ocular migraine, we considered it justified to use low dose propranolol as prophylaxis in this patient, with prevention of further attacks and no adverse effects. At present therefore, we advocate the continued use of low dose propranolol in cases of ocular migraine.

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