Adverse effects of subconjunctival injections of mydriatic agents

Sir,—The adverse effects of mydriatic agents are well documented. When using these drugs one must carefully consider the relevant medical history. The uveitic patient with early posterior synechiae and concomitant systemic disease is illustrative of this problem. We report a serious complication in a patient treated with mydriatic agents.

A 26-year-old male had a 10-day history of photophobia, fatigue, and fevers for several months. On examination his vision was 20/40 and the pupils were constricted secondarily to 27° posterior synechiae. He had hyperaemic conjunctiva, diffuse ‘mutton-fat’ keratic precipitates, and 3+ cells. Dilatation of topical and pleged synechiae 1% and phenylephrine 2.5% was unsuccessfully attempted. The patient returned 24 hours later, and a mixture of two drops (100 μl) each of tropicamide 1%, atropine 1%, and phenylephrine 2.5% was injected subconjunctivally at the inferior limbus of the right eye. This mixture comprised 7 mg of phenylephrine, 1 mg of atropine, and 1 mg of tropicamide. The patient immediately developed severe headache, dyspnoea, and diaphoresis. His pulse was 110 per minute and blood pressure of 160/110 mmHg.

He was taken to the emergency room, where he was found to be in respiratory failure. Chest x-ray revealed diffuse interstitial infiltrates not present on the previous day’s films. The patient was intubated, and Swan-Ganz catheterisation revealed a pulmonary wedge pressure of 8 mm Hg. He was transferred to the intensive care unit for management of non-cardiogenic pulmonary oedema. Subsequent diagnostic testing was consistent with a diagnosis of sarcoidosis. The pulmonary oedema resolved and the patient was discharged on topical steroids with subsequent resolution of the iritis.

This is the first reported case of non-cardiogenic pulmonary oedema associated with the use of mydriatic drugs. Both atropine and tropicamide are anticholinergic drugs whose actions include relaxation of the circular muscle of the iris and paralysis of the ciliary muscle. The principal adverse cardipulmonary effect of these drugs is tachycardia. Phenylephrine, a potent, direct-acting, and selective α, agonist exerts its main mechanism of action by stimulation of α receptors of the dilator pupillae.1 Stimulation of these receptors can cause constriction of the systemic, pulmonary, and coronary arteries, leading to severe hypertension, headache, ventricular arrhythmias, myocardial infarction, and cardiac arrest.1 It is conceivable that in our patient phenylephrine may have produced severe pulmonary vasoconstriction leading to alveolocapillary damage and consequent non-cardiogenic pulmonary oedema. In addition our patient had pre-existing pulmonary sarcoidosis, which may have increased his susceptibility to lung injury from other insults.

Subconjunctival injections of mydriatic agents to forcibly dilate pupils with synechiae is recommended2 and is commonly used in several eye centres. These injections, especially in hyperaemic conjunctiva, can lead to enhanced systemic absorption and serious complications. We advise extreme caution when using this method of administering dilating agents.

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Branch retinal artery occlusion in toxoplasma retinochoroiditis

Sir,—Although toxoplasma retinochoroiditis may cause retinal vein occlusion, vascular remodelling, and even retinochoroidal anastomosis,3 retinal artery obstruction is rare.4 We have recently studied a patient suffering from such a complication.

An 18-year-old student attended in March 1989, 24 hours after the sudden loss of inferotemporal field in the right eye, which was confirmed by automated perimeter. A branch retinal artery occlusion was found in a corresponding area of focal retinochoroidal inflammation. Some areas of peripheral retinitis were also seen peripherally. Fluorescein angiography confirmed occlusion of the artery within the area of inflammation (Fig 1). Antibodies to toxoplasma were detected by latex agglutination at serum dilutions of more than 1:1000.

The patient was treated with clindamycin, 150 mg four times a day, and prednisolone 60 mg daily, reducing over eight weeks. At the end of this period the inflammatory focus had become inactive. Repeat fluorescein angiography demonstrated reperfusion of the artery (Fig 2). However, the field defect persisted.

This case confirms that field loss in toxoplasma retinochoroiditis can result from arterial occlusion. Arterial thrombosis is presumably caused by the intense adjacent inflammation. Corticosteroid and antimicrobial therapy5 may assist resolution of the inflammatory focus and should be considered when retinal vessels are involved and therefore at risk of occlusion. If, as in this case, occlusion has already occurred, retinal function may not recover despite resolution of the inflammation and reperfusion of the blood vessel.

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