Sir,

Acute nonarteritic ischaemic optic neuropathy after phentermine

Amphetamine derivatives used in weight reduction regimens are considered safe for use over a several week period. Ischaemic and/or haemorrhagic infarcts and necrotizing angiitis are common neurologic complications of amphetamine abuse. Ocular side effects of amphetamine abuse include mydriasis, decreased accommodation and convergence, visual hallucinations (blue tinge to objects), retinal vasoconstriction and vasculitis, retinal microaneurysms and haemorrhage, and anterior ischaemic optic neuropathy.[1] In this report a 35-year-old woman developed acute nonarteritic anterior ischaemic optic neuropathy (NAION) after two prescribed dosages of phentermine hydrochloride as part of her weight loss treatment.

Case report

This obese 35-year-old Caucasian woman developed an acute superior nasal visual field defect in her left eye 1 day after taking Adipex-P (phentermine hydrochloride) 37.5 mg p.o.q. day for 2 days. Her body mass index was 34 kg/m^2. She had no history of heart disease, diabetes, hypertension, hypercholesterolaemia, or strokes.

On neuro-ophthalmologic examination her visual acuity with correction was 20/20 OD and 20/100 OS. Humphrey visual fields revealed a left superior nasal focal defect respecting the horizontal meridian. She had a left relative afferent pupillary defect and a protan defect. Extraocular motility was normal. Slit-lamp examination revealed no cells or flare in the anterior chambers. On dilated funduscopic examination, her left optic disc appeared swollen with small slit-like peripapillary haemorrhages in the superior and temporal aspect of the disc. A small petechial haemorrhage was seen at the 5 o'clock position in the peripapillary region. No cotton wool spots were observed. Her right cup-to-disc ratio was 0.3. Maculae and peripheral retina were normal.

MRI of the brain and orbits with gadolinium, MRA of the cerebral blood vessels, carotid ultrasound, and transoesophageal echocardiogram were all normal. Her cerebrospinal fluid (CSF) opening pressure was 148 mm H_2O; CSF cell count, protein, glucose, VDRL, gram stain, bacterial, viral, and fungal cultures were all unremarkable. Leber's hereditary optic neuropathy mitochondrial DNA mutations for 3460, 11778, 14484, and 15257 alleles were all negative. Autoimmune profile including antinuclear antibody, double stranded DNA, anti-SSA, anti-SSB, anti-Smith, anti-RNP, scleroderma antibody, and Jo-1 antibody were all negative. Hypercoagulable laboratories including cardiolipin antibody screen with reflex testing, lupus anticoagulant, beta-2 glycoprotein I antibodies, phosphatidylserine antibodies, VDRL, and dilute Russell viper venom were all negative. Serum Chlamydia pneumoniae PCR, Lyme antibodies, and HIV 1 antibody were negative. Serum angiotensin-converting enzyme, serum methylmalonate, and urine for heavy metals were negative. Fasting plasma homocysteine test was normal and PCR for methylene tetrahydrofolate reductase was negative. Her haemoglobin A1C and HDL/LDL ratio were normal.

Three weeks later, after discontinuing the medication, her visual acuity was 20/20 OD and 20/60 OS. Disc haemorrhages resolved and the left optic disc had mild temporal pallor.

Comment

Phentermine hydrochloride is a sympathomimetic amine with pharmacologic activity similar to the prototype class of drugs, the amphetamines. It is an FDA-approved prescription medication indicated for short-term (a few weeks) adjunct to a regimen of weight reduction based on exercise, behavioural modification, and caloric restriction in the management of exogenous obesity for patients with an initial body mass index greater than or equal to 30 kg/m^2 (PDR drug).

Only two other reports of amphetamine-related acute visual loss have shown a temporal relationship of acute NAION following oxymetazoline[2] used in nasal decongestants[3] and intranasal methamphetamine abuse. Unlike these patients who had underlying insulin-dependent diabetes, hypertension, or crowded optic nerve heads, the patient presented here was healthy. Investigation for autoimmune disorders, hypercoagulable disorders, hypercholesterolaemia, hypertension, diabetes, strokes, valvular and cardiac wall abnormalities, LHON mutation, infectious disorders, sarcoidosis, and B12 deficiency was negative. This patient also did not show any central nervous system findings or fluoroscein angiographic evidence of necrotizing angiitis related to vascular occlusion.[4] Her acute unilateral visual loss is a result of ischaemia to the posterior ciliary arteries of the left eye from vasoconstriction secondary to phentermine.

This report emphasizes the possible ocular adverse effects of prescribed diet pills, such as phentermine hydrochloride. It is important to be aware that acute NAION may occur not only in patients who abuse amphetamines, but also in patients who take the usual

prescribed doses of phentermine hydrochloride, a Federal Drug Administration (FDA)-approved medication for the treatment of obesity.

References

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Sir,

Hemiretinal arterial supply by the cilioretinal artery

Cilioretinal arteries are reported to be present in up to 50% of eyes, and are considered to be the commonest retinal vascular anomaly.[1] When present, cilioretinal arteries vary in size, number, distribution, and point of origin from the optic disc. Large cilioretinal arteries can supply more than a quarter of the retinal circulation in 0.6% of cases of which 15% are bilateral.[2]

We present a rare case of a patient with symmetrical, bilateral cilioretinal arteries that arise temporally and supply the entire superior hemiretina.

Case report

A 75-year-old lady was referred for cataract surgery with a vision of 6/18 and 6/36 in the right and left eyes, respectively. Examination revealed possible retinal oedema at the left macula with pigmentary changes and an incidental finding of large cilioretinal arteries supplying the superior hemiretina in both eyes (Figure 1). A fluorescein angiogram showed no neovascular membrane but highlighted the cilioretinal artery as it arose from the disc margin (Figure 2). She was otherwise healthy.

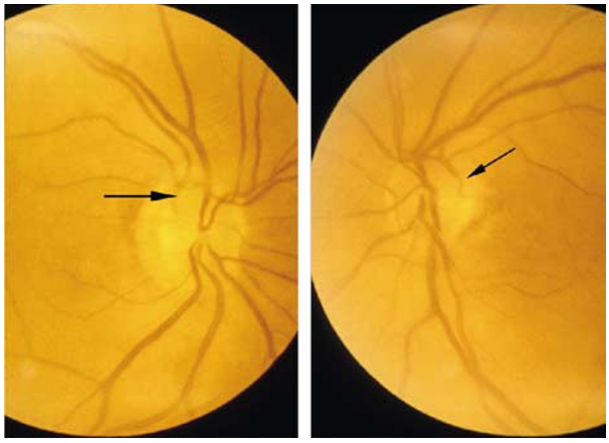

Figure 1 Right and left posterior pole fundus photographs show that there is no superior branch of the central retinal artery visible in either fundus, but a cilioretinal artery can be seen exiting the disc (arrow) with a superior hemiretinal distribution.

Discussion

Cilioretinal arteries arise from the short posterior ciliary arteries, as does the choroidal circulation. They make a characteristic bend as they leave the disc margin and are recognisable on fundoscopy. If rapid sequence, early-phase images are taken during fluorescein angiography, the dye fills the choroidal vessels and cilioretinal artery simultaneously, approximately 1–2 s before the central retinal artery. However, this is not diagnostic and the reverse can occur.[1]

In the healthy eye, the presence or absence of a cilioretinal artery is clinically insignificant. If retinal vascular occlusion occurs, the presence of a cilioretinal artery can be a significant factor influencing visual morbidity.

In central retinal artery occlusion (CRAO), a large temporal cilioretinal artery maintains the circulation to the papillomacular and macular regions of the retina, therefore sparing central vision.

However, the cilioretinal artery itself can become obstructed. This occurs as three clinical variants, Brown *et al*.[3] (1) Isolated cilioretinal artery obstruction (40%). This has a good prognosis with 90% of eyes returning to 6/12 vision and 60% to 6/6. (2) Cilioretinal artery obstruction with central retinal vein obstruction (40%).