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RESEARCH ARTICLE

ACETAZOLAMIDE - INDUCED VISUAL DETERIORATION IN A CASE OF IDIOPATHIC INTRACRANIAL HYPERTENSION

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ABSTRACT

We report a case of idiopathic intracranial hypertension, in whom visual deterioration was noticed, after starting treatment with acetazolamide. Initially a possibility of optic nerve decompensation due to papilledema was considered. On subsequent opthalmological evaluation the patient was found to have bilateral myopia, which resolved completely after stopping acetazolamide.

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INTRODUCTION

Idiopathic intracranial hypertension (IIH) is not uncommon in neurological practice. Most often it is managed medically with drugs like acetazolamide and other anti-edema measures. Visual worsening despite therapy is an ominous sign and optic nerve decompression surgery is sometimes required. Medication related side effects are uncommon, but they do occur. We report a case of IIH, in whom treatment with acetazolamide resulted in visual deterioration.

Case History

A 27-year old male presented to us with headache of seven months duration, which had worsened for the past seven days. Headache was diffuse and was not associated with any vomiting, diplopia, transient visual obscurations or seizures. He was a moderately built and nourished adult with a body mass index of 18. Neurological examination was normal except for bilateral papilledema. His visual acuity was 6/6 with normal color vision in both eyes.

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Visual field examination by Humphrey perimeter was normal. Magnetic resonance imaging of brain and magnetic resonance venogram did not reveal any abnormality. CSF analysis with manometry showed opening pressure of 210 mm of H_2O, no cells, normal protein and sugar. Serum calcium, phosphorus, thyroid function tests were normal and vasculitic work up was unremarkable. He was not consuming any medications that can cause raised intra cranial pressure. He was started on oral acetazolamide (diamox) 250 mg bid and oral glycerol 30 ml TID for the raised intra cranial pressure. Forty-eight hours after starting acetazolamide patient developed blurring of vision in both eyes. An optic nerve decompensation due to papilledema was considered and an urgent ophthalmological evaluation was sought. Ophthalmological evaluation did not show any fresh changes in the retina. There was deterioration of visual acuity to 6/60, which improved to 6/9 with pin-hole correction, in both eyes. Color vision was normal and visual field analysis did not show any abnormality. A refraction of –1.75D was detected on retinoscopy with visual acuity improving to 6/6 on correction. Anterior chamber was normal with an anterior chamber depth of 2.6mm, on ultrasonography. Intra ocular pressure was 12 mm of Hg in both eyes. A possibility of acetazolamide-induced myopia was considered and acetazolamide was stopped. Patient was continued on oral glycerol. Twenty-four hours after stopping acetazolamide his

visual acuity returned to normal and no refractive error was detected on retinoscopy. Anterior chamber depth was 2.8 mm and intraocular pressure was 14 mm of Hg in both eyes.

DISCUSSION

Drug-induced transient myopia has been reported in literature as a rare side effect of certain group of drugs. They occur as an idiosyncratic reaction to drugs like sulfonamides (including anti-bacterials and diuretics) and carbonic-anhydrase inhibitors like acetazolamide (Murihead, 1960; Beasely, 1962) and topiramate (Boentert, 2003 and Bhattacharyya, 2005). The exact mechanism of this phenomenon is not known. As a result of the idiosyncratic reaction to the drugs cilliary body swelling occurs as the primary event. Cilliary body edema has been postulated to be caused by increased prostaglandin levels (Kreig, 1996). There is an associated supracilliary choroidal effusion, which has been demonstrated on ultrasound biomicroscopy (Postel, 1996). These events lead to relaxation of zonules, increased anterior-posterior diameter of the lens and anterior displacement of the iris-lens diaphragm, all of which contribute to the myopic shift in refraction. These effects when more severe can also cause significant shallowing of the anterior chamber and angle closure glaucoma, as reported in some cases (Postel, 1996 and Fan, 1993). Fan et al reported a case of transient myopia and acute angle closure glaucoma secondary to choroidal detachment. Their patient had received only a single dose of 500mg acetazolamide as routine preoperative medication (Fan, 1993). Our patient had normal anterior chamber depth and intra ocular pressures, with a moderate myopic shift in refraction. No angle closure glaucoma was observed in our patient. The occurrence of shallow anterior chamber and angle closure glaucoma was associated with a higher myopic shift, which was most likely due to more severe cilliary body swelling as seen in case reports of Fan etal and Postel etal (Postel, 1996 and Fan, 1993). It can be inferred from the above discussion that the severity of myopia, occurrence of shallow anterior chamber and angle closure glaucoma is due to increasing severity of cilliary body swelling and supracilliary choroidal effusion.

Visual loss is the most serious complication of idiopathic intracranial hypertension, warranting regular monitoring of visual function. Visual deterioration is usually secondary to severe intracranial hypertension poorly responsive to medical therapy. But in patients treated with acetazolamide, the possibility of drug-induced transient myopia should also be considered.

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