CASE REPORT

Indapamide-induced bilateral choroidal effusion in pseudophakic patient.

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SUMMARY

We describe a case of indapamide-induced bilateral choroidal effusion, first time reported in pseudophakic patient, associated with no change in visual acuity and stable refraction.

A 70-year-old man was referred for ophthalmic assessment, with binasal visual field defect for 2 days. He had been started on treatment with indapamide 3 weeks earlier. His ophthalmic history included bilateral cataract surgery and intraocular lens implant. Fundal examination revealed bilateral choroidal effusions; B-scan ultrasonography was used to measure the extent of the choroidal detachment and the anterior chamber depth. Discontinuation of indapamide resulted in spontaneous resolution of choroidal effusion after 3 days. Our case is the first in the literature that describes bilateral choroidal effusion induced by indapamide in a pseudophakic patient. The lack of myopic shift likely resulted in a later presentation, enhancing the theory that lens thickening and/or accommodative spasm may play a crucial role in pathophysiology.

BACKGROUND

Indapamide is a sulfonamide-containing diuretic. Sulfonamide-containing drugs include antibiotics, carbonic anhydrase inhibitors, thiazide diuretics, sulfonylureas hypoglycaemics and anti-inflammatory cyclooxygenase-2 inhibitors, and are widely used for bacterial infections, hypertension, diabetes, epileptic seizures and bipolar disorder.

Choroidal effusion is a well-known side effect of sulfonamide-derived medications¹⁻⁴; the exact pathophysiology remains a challenge and three theories^{4,5} have been proposed so far, based on the anatomical and functional changes in the lens and uveal tract. According to previous studies,³⁻⁵ main signs are transient myopia, ciliary body oedema and choroidal effusion, thus the vast majority of cases presented with blurry vision due to pseudomyopia.

Three case reports⁵⁻⁷ of indapamide-induced choroidal effusion have been reported, both included phakic patients and were associated with transient myopia. We report the first case in the literature of indapamide-induced bilateral choroidal effusion in a pseudophakic patient. In comparison to the above studies, our case presented with late onset of symptoms, no myopic shift and normal intraocular pressure (IOP).

Although further research is required in order to explain the aetiology behind this condition, through

this case we provide additional clues in discovering the pathogenesis of this rare side effect.

CASE PRESENTATION

A 70-year-old Caucasian man was referred to the Accident and Emergency department at Moorfields Eye Hospital. He was suffering from bilateral nasal shadow, which started 2 days before and gradually getting worse. He had a medical history of hyperlipidaemia and hypertension for which he was on treatment with indapamide tablet 2.5 mg daily, which started 3 weeks before the onset of symptoms. His ophthalmic history included bilateral cataract surgery and intraocular lens (IOL) implant; right eye (RE) was done 2 years before and left eye (LE) 6 months before presentation. Neurological questioning was unremarkable and there was no history of ocular or head trauma reported. On ophthalmic examination, the patient's best corrected visual acuity (BCVA) was 6/9 in the RE and 6/6 in the LE. IOP was 10 mm Hg in both eyes and there was a bilateral nasal field defect to confrontation. There was no afferent pupillary defect or abnormal colour vision in either eye. Slit lamp examination showed white eyes, deep and quiet anterior chambers with no signs of intraocular inflammation and in situ IOLs. Dilated funduscopy revealed right asteroid hyalosis, and large choroidal detachments in both eyes.

INVESTIGATIONS

B-scan ultrasonography is used to assess choroidal effusion and measure the choroid elevation at 1.4 mm nasally and 7.7 mm temporally in the RE and 1.1 mm nasally and 9.0 mm temporally in the LE (figure 1). Additionally, through B-scan we assessed the anterior chamber depth (ACD) at 2.8 mm right and 2.7 mm left. Refraction revealed -1.50D sphere in the RE and -0.75D sphere in the LE, pursuant to his prescription. Further investigation was done with wide-field retinal imaging (OPTOS 200TX) and enhanced depth imaging-optical coherence tomography (EDI-OCT).

TREATMENT

After excluding other surgical and mechanical causes of bilateral choroidal effusion, we advised the patient to discontinue indapamide as the most likely precipitant of the effusions.

OUTCOME AND FOLLOW-UP

On day 3, the patient was completely asymptomatic, no visual field defect to confrontation,

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Figure 1 Wide-field retinal imaging and B-scan ultrasonography of choroidal effusion in the right (A, C) and left (B, D) eyes.

and BCVA, IOP, ACD and refraction were unchanged. Clinically, anterior chamber was deep and quiet in both eyes, and no signs of choroidal effusion detected during funduscopy. A repeat ultrasound scan confirmed the spontaneous resolution of choroidal detachment bilaterally and ACD was unchanged (figure 2). Finally, we arranged a second follow-up, 3 weeks after, where stability was established and patient discharged.

DISCUSSION

As reported in the literature, the three main theories behind this rare side effect include: (A) the lens thickening due to osmotic changes, resulting in an increase of refractive index; (B) the ciliary body oedema as a result of allergic or idiosyncratic reaction to the drug; and (C) the accommodative spasm.^{5–7} Although the mechanism remains unclear, there seems to be a consensus regarding the forward displacement of the iris-lens diaphragm resulting in shallow anterior chamber (AC), narrowing of the angle and rarely acute angle closure glaucoma.^{8–11}

Végh *et al*⁵ used ultrasound biomicroscopy (UBM) to observe the morphological changes of the lens and ciliary body in a phakic patient and concluded that both ciliary muscle contraction and

ciliary body oedema played a key role. In addition, UBM-based studies^{3 5 8 11} noted a minor increase of lens thickness but they could not correlate it with either shallowing of AC or myopic shift. Ramos-Esteban⁸ stated that the spasm of accommodation is the most unlikely mechanism in the sulfonamide-induced transient myopia, as instillation of cycloplegic drops cannot reverse the refractive change. In our case, it is of interest to note the lack of forward rotation of the IOL complex. As a consequence, there was no myopic shift that resulted in late onset of symptoms and no changes in BCVA. Moreover, IOP and ACD were stable despite the choroidal effusions.

Blain *et al*⁷ described diffuse choroidal thickening involving the posterior pole and scattered islands of delayed fluorescein filling at the early and mid-stage of fluorescein angiography, probably related with choroidal thickening. In our case, posterior pole was within normal limits, confirmed by EDI-OCT and stable visual acuity.

In conclusion, our case report is the first in the literature that describes bilateral choroidal effusion induced by indapamide in pseudophakic patient. The lack of anterior IOL rotation and stable ACD enhance the theory that lens thickening and/or accommodative spasm may have a crucial role in the pathomechanism of indapamide-induced myopia. Therefore, refraction and IOP are expected to remain stable in pseudophakic patients with a later presentation of choroidal effusion symptoms compared with phakic patients.

Learning points

- ▶ Indapamide is a sulfonamide diuretic used worldwide to treat hypertension. Rarely can cause choroidal effusion.
- ▶ Our case is the first in the literature that describes bilateral choroidal effusion induced by indapamide in a pseudophakic patient.
- ▶ We provide additional clues in discovering the pathogenesis of this rare side effect.
- ▶ We wish to draw attention to one of the potential ocular adverse effects of indapamide.

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Figure 2 Wide-field retinal imaging and B-scan ultrasonography demonstrating the spontaneous resolution of choroidal effusion in the right (A, C) and left (B, D) eyes.

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