

CASE REPORT

Methylphenidate (Ritalin)-associated Cataract and Glaucoma

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Methylphenidate hydrochloride (Ritalin) is the drug of choice for attention deficit hyperactivity disorder (ADHD). However, an association of Ritalin with glaucoma has been reported. We report a case of Ritalin-associated cataract and glaucoma. A 10-year-old boy was diagnosed with ADHD and had received methylphenidate hydrochloride, 60 mg/day for 2 years. He presented with blurred vision. Best-corrected visual acuity was 6/60 in both eyes. Ocular examinations revealed intraocular pressure (IOP) of 30 mmHg under medication, dense posterior subcapsular opacity of lens, pale disc with advanced cupping, and marked constriction of visual field. Despite maximal anti-glaucomatous medication, IOP still could not be controlled. The patient then received combined cataract and glaucoma surgery. Visual acuity improved and IOP was within normal limits in both eyes postoperatively. Large dose of methylphenidate may cause cataract and glaucoma. The mechanism remains unclear. Doctors should be aware of the possible ocular side effects of methylphenidate. [*J Chin Med Assoc* 2006;69(12):589–590]

Key Words: cataract, glaucoma, methylphenidate, Ritalin

Introduction

Methylphenidate hydrochloride (Ritalin), a short-acting central nervous system stimulant, is common and efficacious in the treatment of attention deficit hyperactivity disorder (ADHD) in psychiatric clinic, especially for children.¹ It is a sympathomimetic amine and is contraindicated in patients with glaucoma, as mentioned in the Physicians' Desk Reference and package insert. Glaucoma exacerbated by oral methylphenidate has been reported in the literature.² However, there has been no report of methylphenidate-associated complicated cataract. We present a case of bilateral complicated cataract and glaucoma following large-dose methylphenidate use for 2 years.

Case Report

A 10-year-old boy presented with progressive blurred vision in both eyes over the past 1 year. He had no

history of systemic disease, ocular trauma or ocular disorder except myopia before this episode. His corrected visual acuity was 6/7.5 in both eyes 1 year previously. Decreased visual acuity was noted during routine school physical check-up 3 months before this visit. Glaucoma and cataract were diagnosed at the ophthalmic clinic. The patient was referred to our department due to poorly controlled intraocular pressure (IOP) under 2% carteolol eye drops twice a day. His best corrected visual acuity (BCVA) was 6/60 with sph -5.25 cyl $-1.5 \times 160^\circ$ in the right eye and 6/60 with sph -5.25 cyl $-0.75 \times 40^\circ$ in the left eye on presentation.

Slit-lamp biomicroscopy revealed clear cornea, deep anterior chamber, and dense posterior subcapsular opacity of lens in both eyes (Figure 1). Gonioscopy showed Grade 4 open (Shaffer system) and well developed angle. Indirect ophthalmoscopy revealed pale disc with advanced glaucoma cupping (0.9×0.9) in both eyes. IOP was 26 mmHg in both eyes. Automated perimetry revealed generalized visual field constriction in the right eye and upper arcuate scotoma in the left

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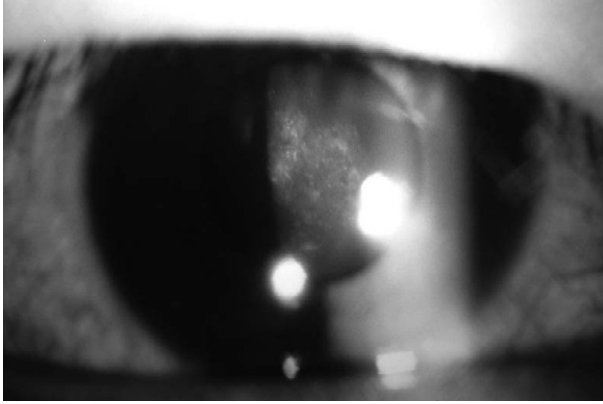


Figure 1. Slit-lamp biomicroscopy of the patient's right eye shows dense posterior subcapsular lens opacity.

eye. The patient had ADHD and had been taking methylphenidate hydrochloride (Ritalin) 40 mg/day in 2 divided doses as prescribed by his psychiatrist. However, his mother had increased the dose to 60 mg/day for the past 2 years. Otherwise, his mother denied any other medication including steroid and herbs or any ocular trauma history.

Under the impression of methylphenidate-associated cataract and glaucoma, latanoprost every night, 2% carteolol twice a day for both eyes and oral acetazolamide 125 mg twice a day were given. Methylphenidate was discontinued. Despite maximal antiglaucomatous medications for 1 month, IOP still fluctuated up to 30 mmHg in both eyes. The patient underwent trabeculectomy and phacoemulsification with intraocular lens implantation for both eyes. IOP was maintained around 16 mmHg in both eyes, and BCVA was 6/6 in the right eye and 6/12 in the left eye at 1 year of follow-up.

Discussion

When glaucoma is encountered in children, the usual first thought is congenital glaucoma. In the congenital glaucomatous eye, we can see Haab's striae on the cornea and underdeveloped mesodermal tissue over the chamber angle by gonioscopy. In our patient, the cornea was clear without any striation. Gonioscopy showed a well developed wide open angle without any residue mesodermal tissue. Congenital glaucoma was therefore ruled out.

In congenital cataract, one can usually see snowflake or sclerosis of the lens nucleus. In our patient, only posterior subcapsular opacity with clear nucleus could be seen. If the lens displays posterior subcapsular opacity, our first consideration would be complicated cataract

related to topical steroid use, diabetes, hyperthyroidism, systemic medications such as phenothiazines, amiodarone, steroid and herbs, etc.³ However, none of the above were found in our patient.

A child with ADHD is prone to head trauma. This could induce traumatic cataract. In such situations, lens subluxation or nucleus milky change would be found. None of that was found in our case, so traumatic cataract was also ruled out.

Methylphenidate is a sympathomimetic amine. It has the potential to induce acute angle closure glaucoma or worsen chronic angle closure glaucoma.^{2,4,5} The Physicians' Desk Reference indicates that the maximum daily dosage is 60 mg, and a dosage >60 mg/day is not recommended. Our patient took a high dose of the medication for 2 years. A characteristic glaucomatous optic neuropathy with associated visual field defect and elevated IOP, and complicated cataract in both eyes were noted. Since the congenital factor was not likely, and there were no other medication- or trauma-related factors, the possibility of prolonged high-dose Ritalin use-related complicated cataract and glaucoma was our first consideration.

Despite discontinuation of methylphenidate, dense cataract and persistent high IOP caused severe vision loss in both eyes. One possible mechanism is that methylphenidate may cause cytoskeletal change in the chamber angle trabecular meshwork or obstruction of trabecular meshwork. Even after the discontinuation of methylphenidate, the function of the trabecular meshwork did not recover. Therefore, the glaucoma remained out of control. Filtering surgery for glaucoma management was necessary.

From the literature, no report of Ritalin-related cataract has been found. This is the first report of methylphenidate-associated cataract and glaucoma. Although the underlying mechanism is unknown, the possible ocular side effects should be considered in patients on methylphenidate treatment.

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