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Is the Treatment of Attention Deficit Hyperactivity Disorder a New Cause of Cataract?

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Abstract

Attention deficit hyperactivity disorder (ADHD) is the most common behavioral disorder of childhood and it has 5% prevalence among children worldwide [1]. In the treatment of ADHD stimulant medications are recommended as the first choice of pharmacotherapy [2]. Methylphenidate-HCl is a sympathomimetic amine derivative pharmacological agent; and it is widely used in the treatment of ADHD. Although there are some articles showing that oral methylphenidate increases the risk of glaucoma [3], to the best of our knowledge, there is only one case report that indicates a possible relation of methylphenidate treatment and cataract formation [4].

Keywords

Attention Deficit Hyperactivity Disorder, Methylphenidate, Cataract

1. Introduction

17-year-old boy admitted to Adnan Menderes University Medical School, Department of Ophthalmology with the complaint of progressive vision loss, light sensitivity, glare and halos in his right eye. Best corrected visual acuity was found 0.3 in the right and 0.8 in the left eye. On slit lamp examination scattered-punctuate, anterior-posterior subcapsular cataract in the right eye and minimal posterior subcapsular star shaped punctuate opacities in the left eye was found (**Figure 1**: Slit lamp photographs [(a), (b), (c), (d)]) show the cataract formation. The morphology of posterior subcapsular cataract is demonstrated on retroillumination in the right eye (**Figure 1(a)**). Slit lamp appearance of anterior subcapsular cataract in the right eye (**Figure 1(b)**), **Figure 1(c)**). Minimal posterior subcapsular cataract is

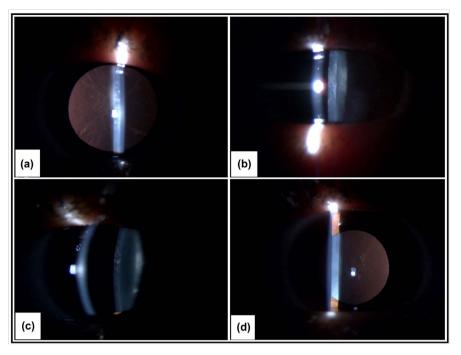


Figure 1. Slit lamp photographs (a), (b), (c), (d) show the cataract formation. The morphology of posterior subcapsular cataract is demonstrated on retroillumination in the right eye (a). Slit lamp appearance of anterior subcapsular cataract in the right eye (b), (c). Minimal posterior subcapsular cataract is demonstrated on retroillumination in the left eye (d).

demonstrated on retroillumination in the left eye (Figure 1(d)). His eye examination was otherwise normal. The patient's medical history revealed that he was under follow up with the diagnosis of ADHD since three years old. He had a history of methylphenidate HCL 10 mg tab PO 2 × 1/2 per day (Ritalin[®] 10 mg tab, Novartis, Barbera del Valles, Spain) uptake for four years, followed by methylphenidate HCL 54 mg tab PO 1 × 1 per day (Concerta® 54 mg tab, Johnson & Johnson, Vacaville, CA, USA) for an additional four years. It was learned that the patient also received steroid treatment (methylprednisolone succinate) 3 - 4 times per year (1 mg/kg IM, only during attack periods in the emergency service), for about 9 years due to bronchial asthma. The patient's mother was not found to have any ocular pathology on a detailed ophthalmological examination. It could not be possible to examine the father, but he had no known ocular pathology in his previous medical reports. The patient's mother and father did not have consanguinity. There was not any history of ocular trauma. For etiological investigation other possible systemic factors (diabetes, hyperthyroidism, rheumatic pathology, etc.) were ruled out by medical history and laboratory tests (including possible metabolic and non-metabolic causes such as galactosemia and TORCHS). Phacoemulsification and posterior chamber intraocular lens (IOL) implantation were performed in standard fashion without posterior capsulectomy in the right eye. Intraoperatively, excessively fragile structure of the anterior capsule was observed during capsulorhexis. Pars plana vitrectomy or anterior vitrectomy was not performed. At the end of the surgery no suture was used for the incisions. One month after the phacoemulsification, his vision improved from 0.3 to 1.0. It is considered that anterior-posterior subcapsular cataracts and capsular fragility could have been developed secondary to methylphenidate HCL.

2. Discussion

Juvenile or developmental cataract which is also named as presenile cataract is a kind of slowly progressive cataract and does not cause too much visual impairment and there can be many causes in its etiology. In the differential diagnosis of juvenile cataract, many factors should be excluded, such as congenital cataract, trauma, systemic diseases (diabetes mellitus, rheumatic diseases, thyroid dysfunction, etc.) and drug use.

In our case, in both eyes, especially in the right eye, punctuate cataracts were found and the anterior capsule was observed highly fragile during the intraoperative period. The symptoms were not since birth and in ocular examination the morphology of the cataract was unlike to any other forms of congenital cataract. The congenital cataract was excluded due to ocular examination findings, history, and the absence of any ocular pathology in patient's first degree relatives. Systemic diseases were excluded through the patient's history and laboratory workup. There were no history of ocular trauma and continuous use of any additional medication.

It is known that the use of steroids can cause cataracts, especially in the form of posterior subcapsular cataracts (PSCC). However, doses and duration are thought to be effective in the formation of cataracts. Mino et al. [5] and Fine et al. [6] proposed that the dose of steroid use is associated with the formation of PSCC. On the other hand Kobayashi et al. [7] and Bihari et al. [8] thought that both dose and duration of steroid use affect the development of PSCC. Fine et al. [6] found that PSCC formation is associated with high dose of steroid use (54.9 mg/kg/month). Joan et al. [9] also found an association between PSCC formation and high dose (17322 \pm 736 mg/kg) steroid use, but not with duration. In our case, the patient did not receive any steroid treatment for a period; he received steroid injection only during attacks with the dose of 1 mg/kg, which is much less than the dose that is required for cataract formation according to the reported dose by Joan et al. Also the duration of steroid use was not considered to be at a level that could lead to cataract formation. The morphology of the cataract was found different than that of the typical posterior subcapsular morphology of the steroid cataract.

Methylphenidate is a kind of sympathomimetic amine derivatives and it can induce the acute angle-closure glaucoma or may worsen chronic angle-closure glaucoma [10]. In our case, no evidence was found in the retinal nerve fiber layer analysis, ocular examination and visual field examination suggestive as glaucoma.

In the literature, methylphenidate associated cataract has been reported in the form of a single case report. Chao Li-Kung *et al.* [4] reported PSCC and primary open-angle glaucoma in ocular examination of a 10-year-old patient who was diagnosed as ADHD and had a history of 60 mg/day use of methylphenidate over two years. They reported that cataract was developed secondary to use of methylphenidate after exclusion of other factors that may cause cataracts in that patient.

Criado-Álvarez JJ et al. [11] reported ADHD prevalence of 13.22/1000 and showed an increase in methylphenidate and similar drugs such as atomoxetine, and lisdexamfetamine. More than 89% of them were treated with methylphenidate. Methylphenidate has been used since about 1960, with increasing use through the 1990's and it is still being used. Criado-Álvarez JJ et al. [11] also reported differences in drug consumption between different geographic localization, which reflect differences in ADHD management in clinical practice. Children have taken billions of doses of this medicine.

In our case's history, there have been approximately eight years of methylphenidate use. Cataract formation and abnormally fragile capsule morphology is thought to be induced by methylphenidate use. Due to limited reports in the literature in terms of possible relationship between methylphenidate and cataract formation, prospective long-term case studies are required to clarify other factors involved.

3. Conclusion

We suggest an ophthalmological examination prior to the treatment and during follow up in children who are prescribed methylphenidate treatment. The possible relationship between the drug use and cataract formation should be kept in mind in the treatment ADHD.

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Disclosure

The authors report no conflicts of interest in this work.

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