INTRAOCULAR PRESSURE ELEVATION IN A CHILD DUE TO THE USE OF INHALATION STEROIDS - A CASE REPORT

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ABSTRACT:

Inhalation steroid therapy can cause ocular hypertension or open angle glaucoma. The authors describe the case of a young girl who presented with raised intraocular pressure and headaches due to the prolonged administration of nasal and inhalation steroids. The ophthalmologist should monitor the intraocular pressure in patients who use inhalation or nasal steroid therapy on a regular base. The physician or paediatrician should be aware of this complication in children with headaches or diminished visual acuity.

RÉSUMÉ:

Un traitement à la cortisone par aérosol ou spray nasal peut causer une augmentation de la tension intraoculaire ou un glaucome à angle ouvert. Les auteurs décrivent le cas d' une jeune fille qui présente une augmentation de la tension intra-oculaire due au traitement à la cortisone par aérosol ou spray nasal prolongé. Il est important que l'ophtalmologue fasse des contrôles réguliers de la tension oculaire chez des patients sous ce traitement. Le médecin généraliste et le pédiatre doivent prendre cette complication en considération chez les enfants présentant des maux de tête ou une baisse de l' acuité visuelle.

SAMENVATTING:

Inhalatietherapie met corticosteroïden of gebruik van nasale sprays met steroïden kunnen intraoculaire drukstijging en open hoek glaucoom veroorzaken. De auteurs beschrijven een meisje dat verhoogde oogdruk vertoonde als gevolg van het langdurig gebruik van nasale en inhalatie corticotherapie. Het is belangrijk dat de oogarts regelmatig de oogdruk controleert bij patiënten die dit soort therapie gebruiken. De huisarts en pediater moeten deze complicatie overwegen bij kinderen die klagen van hoofdpijn of verminderde visus.

KEY-WORDS

Intraocular pressure, inhalation steroids, nasal steroids, case report

MOTS-CLÉS

Tension intra-oculaire, cortisone par aérosol, cortisone par spray nasal

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INTRODUCTION:

Nasal and inhalation steroids have been described to cause systemic side effects. Only more recently, since 1993, several authors reported an association between inhalation and nasal steroids and glaucoma or raised intraocular pressure.

CASE REPORT:

An 8 year old girl was referred by the paediatrician with complaints of headaches in the frontal area, especially at the right side, since two years. There was no other systemic cause for the headaches. Her past medical history revealed bronchial asthma, which was treated with inhalation therapy of budesonide (Pulmicort®) 200µg a day. Since two years, she had been using intranasal budesonide spray (Rhinocort®) 100µg a day. The family history was normal. The mother mentioned that the headaches started shortly after the use of the intranasal spray. On ophthalmologic examination, the visual acuity was 10/10 at both sides. She could read Snellen 1 bilaterally. Her eyes were straight and eye motility was normal. Slit lamp examination showed no abnormalities. Cycloplegic refraction showed emmetropia at both eyes. Her intraocular pressures by Goldmann applanation tonometry were 23 mm Hg on the right side and 19 mm Hg on the left side. Ophthalmoscopy showed bilaterally normal optic discs. Visual field examination was performed with the Goldmann perimeter and showed normal sectoral limits (I/4 index). Since her intraocular pressures were raised according to her age, she was asked to protect her eyes while she used the inhalation therapy of the steroids. The physician and paediatrician were notified that she did respond with high intraocular pressures to the inhalation of the steroids and intranasal steroid spray. We saw her back 4 weeks later. The physician had decided to discontinue the intranasal steroid treatment and changed the inhalation of steroids into the inhalation of sodiumchromoglycate (Lomudal®). Since then, the headaches had disappeared. She only mentioned one period of frontal headache during that interval. Her visual acuity was 10/10 at both sides. Slit lamp examination was normal. The intraocular pressures with Goldmann applanation tonometry were 13 mm Hg on both sides. Ophthalmoscopy showed bilateral healthy optic discs. Her peripheral and central visual fields, plotted with the Goldmann perimeter and Friedmann perimeter were within normal limits. Another applanation tonometry one month later also revealed normal intraocular pressures. Further follow-up of the intraocular pressures, optic discs and visual fields are planned.

DISCUSSION:

Advances in the medical therapy of systemic disorders can cause ocular disease.

Since 1954, ocular hypertension induced by *topical* (ophthalmologic and dermatological), *periorbital* or *systemic* use of steroids is well described (2,6). Significant glaucomatous optic neuropathy with visual field loss has been observed based on steroid response. Persistent elevation of the intraocular pressure, even after discontinuing the steroids, has been reported (4).

An intriguing aspect of the relationship between steroid use and ocular hypertension, is that people can be divided in two categories of responsiveness: steroid responders and non responders (1).

The pathogenesis of the increased aqueous outflow resistance remains unknown. In 1997, a gene coding for a trabecular meshwork induced glucocorticoid response protein (TIGR), now called myocilin, was identified (11,12). A possible mechanism for steroid responsiveness could be sequence changes in a glucocorticoid response element or other regulatory regions of the myocilin.

Other possible mechanisms include accumulation of polymerised glycosaminoglycans in the trabecular meshwork and an increase in the expression of collagen, elastin, or fibronectin in the extracellular network (3). Further study is required.

Inhaled and nasal corticosteroids are nowadays increasingly prescribed for the long-term prophylactic treatment of asthma and rhinitis. As it is well known that systemic absorption occurs after inhaled or nasal administration of corticosteroids, there has been increasing concern about the safety of these drugs. Various side effects of corticosteroids are known: e.g.

growth retardation in children, osteoporosis, infections, skin atrophy, hypothalamic-pituitary-adrenal axis suppression, cataract and glaucoma.

Given the findings in our patient and since inhaled or nasal corticosteroids have generally been considered not to have the same ocular side effects as systemic corticosteroids, we studied the published literature on the association between inhaled/nasal corticosteroids and ocular side effects.

In the literature, four case reports and one case control study on the association between inhaled corticosteroids and glaucoma are found. Dreyer reported 3 cases of glaucoma associated with inhaled corticosteroids, but gives details on only one representative case: a 57-year-old female reported diminished visual acuity six months after she was started on beclomethasone dipropionate (BDP), three puffs three times a day (5). The IOP in the left eye was 42 mmHg and fell to 7 mmHg following withdrawal of BDP. Opatowsky et al. reported on another case in 1995 (10). The case control study was performed in Canada in 1997 using a health insurance database of inhabitants over 65 years of age. Overall, there was no increased risk of glaucoma amongst people using inhaled corticosteroids. However, high dose ((1600 µg BDP/day) and long-term (3 months) inhaled corticosteroid use was associated with a small but significant increased risk of glaucoma. In the same case control study Garbe et al. also studied the association between nasal corticosteroids and glaucoma (7). They found no indication for an increased risk but the study was limited by a relatively small number of cases taking nasal corticosteroids. Opatowsky et al. described two patients, 71 and 61 years of age who developed increased IOP after treatment with nasal corticosteroids (10).

In 1999, Mitchell et al. stated that IOP rise due to inhalation of corticosteroids only occurred in patients with a first-degree family history of glaucoma (9). This may be an example of a gene-environment interaction. The result of their study is dual; since only 10% of the population has a first-degree family history of glaucoma, clinicians may be able to reassure the majority of patients that they are at low risk of steroid-induced glaucoma. Conversely, they stress that inhalation steroids should be used with even greater care in patients with a family history of glaucoma.

An increased risk of subscapular cataracts in patients receiving long-term oral corticosteroids is well recognised, but the association between inhaled corticosteroid therapy and cataract formation is more controversial. In 1999 Cave et al. reviewed the literature and found an association between the development of cataract with inhaled corticosteroid therapy but there was insufficient evidence to support an association with nasal corticosteroids (3).

Central serous retinopathy also has been described in association with inhalation of steroids (8).

The route of causing ocular hypertension or posterior subcapsular cataracts due to inhalation or nasal administration of steroids, is believed to be dual; most of the deposition is at the level of the oropharyngeal mucosa (and some in the mucosa of the gastrointestinal tract), where there is systemic absorption which can cause the adverse events in susceptible patients. Direct deposition of droplets of steroids on the corneal surface can also be responsible for the ocular side effects (10).

In this case report, we describe an 8 year old girl who developed raised intraocular pressure (IOP) after using inhalation and intranasal steroids for 2 years. There was no glaucomatous damage yet. The family history of our patient was negative for glaucoma. This is the first report in the literature in which an association between IOP and inhaled or nasal corticosteroids in a child is described. The dose of both the inhalation and the intranasal corticosteroid therapy was low but therapy had been given for 2 years.

CONCLUSION:

This case is important as inhalation or nasal steroids are nowadays often used on a long-term base. The ophthalmologist should monitor the intraocular pressure and optic nerve function of these patients on a regular base, especially if there is a family history of glaucoma. The physician or paediatrician should be aware of this complication. Complaints of headaches occur frequently in children and a com-

plete history and ophthalmologic examination can reveal the aetiology as was the case in our patient. To prevent these problems, one should try to taper the inhalation or nasal steroids to the lowest dose necessary to control the systemic condition.

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