Ocular Hypertension Requiring Suspension of Inhaled Corticosteroids

Hipertensión ocular que obliga a suspender el tratamiento con corticoides inhalados

We report the case of a 68-year-old woman who was referred to the respiratory medicine department due to a year-long history of recurrent episodes of dyspnea and wheezing. Spirometry showed a normal ventilatory pattern, with a positive bronchodilator test. A diagnosis of mild persistent asthma was made and inhaled mometasone administered using a Twishaler® device was prescribed at a dose of 400 μg/day; under this treatment, the patient’s exacerbations resolved and control was maintained.

The patient had also been in ophthalmologic follow-up for 7 years, due to ocular hypertension. Her intraocular pressure (IOP) by tonometry was 21.19 mmHg, she was receiving latanoprost and had maintained a stable IOP for the last 4 years. After starting treatment with inhaled mometasone, a progressive increase in IOP was observed during her regular ophthalmologic check-ups, so her treatment was switched to bimatoprost. The patient did not respond, and her IOP rose to 28 mmHg (right eye) and 32 mmHg (left eye). Withdrawal of the inhaled corticosteroid was proposed, and 4 weeks after suspending mometasone, IOP was 17 mmHg in both eyes, so the recommendation was made to definitively discontinue the treatment.

This led to a worsening of the asthma, and given the impossibility of continuing continuous inhaled corticosteroids (ICS), maintenance therapy with oral montelukast was initiated, and an inhaled with low-dose budesonide-formoterol was prescribed for use as needed. Under this treatment, IOP remained unchanged and the asthma was controlled.

ICS are the first line of treatment for asthma, although they are not free of adverse effects, particularly when treatment is prolonged and doses are high. A possible association between the use of ICS and the development of intraocular hypertension has been described. The effect of ICS on the eye is probably due not only to systemic absorption but also to the formation of topical ocular deposits as a result of nebulized administration. Ocular hypertension may be related with histological changes caused by ICS in the trabecular cells, consisting of the formation of deposits in the extracellular matrix and increased resistance to the outflow of aqueous humor. A genetic susceptibility to developing corticosteroid-induced glaucoma has been suggested.

Studies analyzing the relationship of ICS with glaucoma are inconclusive. Garbe et al. in a case-control study (9793 cases and 38,325 controls) found a greater risk of glaucoma among patients who used high-dose ICS (budesonide >1600 μg/day) for longer than 3 months (OR: 1.44; 95% CI: 1.01–2.06). However, this study is limited by its design and the non-standardized diagnosis of glaucoma. Mitchell et al. performed a cross-sectional study in 3654 patients with ICS aged 49–97 years: 108 had open-angle glaucoma and 160 had intraocular hypertension. A strong association between the use of ICS and ocular hypertension was observed in individuals with a family history of glaucoma (OR: 6.3; 95% CI: 1.0–38.6), but no association was found in subjects without a family history. González et al., in a study of 2291 cases and 13,445 controls with a mean age of 75 years, found an RR for glaucoma after high-dose ICS at 3, 6 and 12 months of 1.01 (95% CI: 0.70–1.44), 0.92 (95% CI: 0.61–1.38) and 0.84 (95% CI: 0.70–1.44), respectively. In 2 prospective, randomized, double-blind, placebo-controlled clinical trials, one in 1250 patients treated with high-dose budesonide for 20 weeks, and the other in 22 patients treated with inhaled fluticasone for 6 weeks, no differences were found in IOP compared to the placebo group.

We can conclude that the relationship between the use of ICS and increased IOP or the development of glaucoma has not been completely clarified. However, these drugs should be prescribed with caution, particularly in patients with risk factors or previous glaucoma in whom IOP monitoring may be advisable during treatment. In our patient, ICS treatment worsened her glaucoma to the extent that it had to be withdrawn.

References


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