ANISOCORIA RELATED TO BUPROPION IN MIGRAINE
ANISOCORIA PRODUCIDA POR BUPROPIÓN EN PACIENTE MIGRAÑOSA

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ABSTRACT

Introduction: Bupropion is used to help people stop smoking. This drug can cause visual alterations but, up to now, its use was not associated with anisocoria.

Case report: A 40 year-old woman with a personal history of migraines, presented with monocular mydriasis during treatment with bupropion. Both pupils dilated equally after a 5% cocaine test, and constricted equally after a 0.025% Pilocarpine test. The anisocoria disappeared after the treatment with bupropion was withdrawn.

Discussion: Bupropion inhibits neural uptake of norepinephrine and dopamine. Patients with migraine could have a minor unilateral sympathetic pupillary deficit. This deficit could cause hypersensitivity in the oculosympathetic pathway that could be stimulated with bupropion treatment. This feature could explain the mydriasis in our patient. Another explanation could be that bupropion could unmask a physiological anisocoria (Arch Soc Esp Oftalmol 2007; 82: 521-522).

Key words: Bupropion, anisocoria, migraine, Horner syndrome, physiological anisocoria.

INTRODUCTION

In recent times, the administration of Bupropion has been widely prescribed for tobacco cessation.

Bupropion is an antidepressant and helps patients to stop smoking. It is a selective inhibitor of the neuronal reuptake of catecholamines (noradrenaline and dopamine) with minimal impact on
indolamine reuptake (serotonin) and in addition it does not inhibit the action of monoaminooxidase. The mechanism by which Bupropion boosts the ability of patients to abstain from smoking remains unknown. However, it is assumed that its action is aided by noradrenergic and/or dopaminergic mechanisms.

Although this medication is known to cause certain types of visual alterations during treatment, the emergence of anisocoria has not been confirmed so far. The case described herein is that of an adult female suffering from anisocoria associated with this treatment.

CASE REPORT

A 40-year-old woman reported mydriasis in the right eye after a 4-week-long treatment with 300 mg/d of Bupropion, prescribed for tobacco cessation. The only significant element in her personal history was migraine. Ophthalmologic exploration revealed the following: normal visual acuity, anterior and posterior pole. Intraocular pressure was 17 mm Hg.

The right eye pupil measured 4.5 mm and the left eye’s was 3.5 mm. After instilling 5 percent cocaine, both pupils dilated in similar ways. After instilling .025 percent pilocarpine, both pupils constricted in the same fashion.

Anisocoria subsided one week after suspending treatment with bupropion.

DISCUSSION

Some patients with a history of migraine suffer during and between headaches a compromise of the sympathetic ocular fluid and the subsequent unilateral alteration of the sympathetic pupillary pathway. In darkness, the symptomatic pupil in these patients dilates less and more slowly (1) than the contralateral pupil. The average pupillary diameter of patients suffering from migraine has also been studied during and after these episodes. The average diameter is smaller in patients suffering from migraines during their onset than in normal individuals. However, after a migraine episode, both pupils have the same size. These findings back the assumption that migraines are associated with a sympathetic deficit which is larger on the side where migraines occur (2). In other words, there could be a subclinical «Horner Syndrome» with the subsequent slight hypersensitivity due to denervation. In these patients, inhibiting the reuptake of dopamine, an immediate metabolic precursor of norepinephrine and epinephrine, by means of drugs such as bupropion, may cause pupils suffering from this potential hypersensitivity to dilate unilaterally due to this increase in vasoactive substances.

Furthermore, bupropion may lead to mydriasis when administered in high doses in animals used for experimental purposes (3).

In the present case, the Addie Syndrome was discarded due to the symmetry of response to .125 percent pilocarpine. A slight alteration in the sympathetic pupillary pathway could possibly result in a slight hypersensitivity due to denervation and unilateral dilation caused by the decreased reuptake of vasoactive amines induced by bupropion. In any case, the response to the cocaine test was negative, which raised doubts regarding the likelihood of a Horner Syndrome caused by bupropion.

On the other hand, the most frequent causes of anisocoria in human populations are physiological (4), arising in some cases arises intermittently. The response to the eyedrop test in physiological anisocoria is similar for both eyes, since there are only differences in the sympathetic tone. In patients suffering from physiological anisocoria, the latter is more evident in darkness. Bupropion triggers a mydriatic stimulus that could have unmasked a physiological anisocoria.

REFERENCES