Short communication

Pharmacological pseudo-Fuchs

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**A R T I C L E  I N F O**

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**A B S T R A C T**

Case report: A case of unilateral iridis hyperpigmentation and uveitis due to travoprost is presented.

Discussion: Anterior uveitis is a rare side-effect of travoprost. In this case, heterochromic iritis was also presented, which led us to the wrong diagnosis of a Fuchs heterochromic iridocyclitis. The differential diagnosis along with the associated literature is discussed.

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**Pseudo-Fuchs farmacológico**

**R E S U M E N**

Caso clínico: Se presenta un caso de uveitis e hiperpigmentación iridiana unilateral por travoprost.

Discusión: Se trata de un efecto secundario poco frecuente del travoprost que en este caso, por sus características, nos hizo pensar en una iridociclitis heterocromática de Fuchs. Se realiza un diagnóstico diferencial de las heterocromías iridianas.

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**Introduction**

Travoprost is a prostaglandin F<sub>2α</sub> analog that acts as an agonist of the prostanoid F receptor, increasing the output of aqueous humor through the uveoscleral pathway. It exhibits several well-known side effects such as hyperemia (35–50%), eyelash increase (55%), periorbital and iris hyperpigmentation as well as uveitis and arbitrary fatty atrophy.

Fuchs heterochromic iridocyclitis is characterized by unilateral and recurring anterior uveitis, sometimes hypertensive, which does not generate synechiae. The Amsler sign is typical of this condition (bleeding during corneal...
paracentesis). Recently an association has been found with the rubella virus.\textsuperscript{5,9} Iris heterochromia is characteristic (40%\textsuperscript{5}) generally in the form of hypochromia due to iridial atrophy, although in 10% of cases, usually in light eyes, hyperchromia occurs because the atrophy reveals the iris pigment epithelium.\textsuperscript{2,9} It generally produces cataracts (69%) and secondary glaucoma (12%) but without requiring aggressive treatment because it is frequently controlled with non-steroidal anti-inflammatory drugs.\textsuperscript{5,9}

**Case report**

A male, 84 years of age, without systemic or family history of interest visited the practice due to discomfort in the right eye (RE). He was using daily eye drops but was unable to remember the name.

The ophthalmological examination revealed a visual acuity of 0.6 in both eyes (BE) which did not improve with correction, intraocular pressure (IOP) of 18 mmHg in BE and normal funduscopy. Extrinsic and intrinsic motility were normal. The anterior segment of the right eye (RE) was normal with the exception of lens nuclear sclerosis. Marked iris heterochromia was observed, and the patient referred he had noticed it about 4–5 years ago (Figs. 1 and 2). He did not refer previous trauma.

The biomicroscopic examination revealed slight Tyndall in the right eye (RE) of about 5 cells in each field without granulomatous precipitats or posterior synechiae and also without iris anomalies such as atrophy. However, traces of pigments could be observed in the anterior capsule (Fig. 3). The patient also exhibited moderate phakosclerosis.

In the presence of slight, unilateral, non-hypertensive anterior uveitis without posterior synechiae and clear iris heterochromia, Fuchs heterochromic iridocyclitis was suspected and topical diclofenac treatment was established at 4-h intervals and cycloplegic at 12-h intervals.

When reviewing the patient’s clinical record the next day it was seen that the daily treatment was travoprost (Travatán\textsuperscript{6}, Alcon Cusí S.A.) every 24 h in RE, having started 5 years ago. This was established as the cause of heterochromia and the withdrawal of the hypotensor drug caused the disappearance of Tyndall and patient discomfort.

The visual field, with the cataract artifacts, did not exhibit campimetric defects. Corneal pachymetry was of 564 and 538 μm. Gonioscopy revealed a grade 4 Schaffer open angle without neovessels or synechiae.

The appearance of the papilla, the excavation thereof and the retina ganglion fiber layer OCT were within normal limits and therefore the patient was deemed to be ocular hypertensive. After withdrawing the topical medication, the IOP values reached 22–23 mmHg in RE and 20 mmHg in LE.

In addition, slight increase in the depth of the superior orbital sulcus was observed in the right side (Fig. 1).

**Discussion**

Iris heterochromia is a disorder caused by several etiologies in which it is necessary to differentiate whether it is due to hyperchromia in one eye or hyperchromia in the opposite eye.\textsuperscript{2} The differential diagnosis of a region heterochromiae is shown in **Table 1**.

The hyperpigmentation caused by topical prostaglandins, in this case travoprost, varies depending on the color of the iris, being more noticeable in green-brown, yellow-brown and blue-brown heterochromic patients.\textsuperscript{3,5} Hyperchromia increases together with the exposure time.\textsuperscript{7,5} This Iris

**Table 1 – Differential diagnosis of iris heterochromiae.**

<table>
<thead>
<tr>
<th>Hypochromia</th>
<th>Hyperchromia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>Pigmented iris tumors and Ota nevus</td>
</tr>
<tr>
<td>Isolated</td>
<td></td>
</tr>
<tr>
<td>Associated to Waardenburg syndrome</td>
<td></td>
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<tr>
<td>Associated to Parry-Romberg syndrome</td>
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<tr>
<td>Associated to Hirschprung disease</td>
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<tr>
<td>Fuchs uveitis</td>
<td>Fuchs uveitis</td>
</tr>
<tr>
<td>Diffuse atrophy (ICE, albinism, postherpetic...)</td>
<td>Pharmacological (prostaglandins)</td>
</tr>
<tr>
<td>Horner syndrome</td>
<td>Ocular siderosis</td>
</tr>
</tbody>
</table>

ICE, irido-corneo-endothelial syndrome.
hyperchromia is usually irreversible\textsuperscript{2} and is due to increased tyrosinase activity in melanocytes.\textsuperscript{5}

In what concerns the frequency of hyperchromia, Huang et al.\textsuperscript{1} studied brown eyes in treatment with travoprost and obtained a hyperpigmentation percentage of 35%; Stjernschantz et al.\textsuperscript{3} and Wistrand et al.\textsuperscript{5} obtained 45% and 20% respectively after at least 2 years of latanoprost in blue-brown iris such as those of our patient. It must be noted that said percentages were obtained studying the patient under slit lamp because only one third of patients were aware of their hyperchromia.\textsuperscript{1,3} The study by Netland et al.\textsuperscript{2} obtained 5% of hyperchromia with travoprost in one year, while in our environment Teus reported an incidence of up to 69% of hyperchromia with latanoprost.\textsuperscript{10}

In what concerns anterior uveitis as a side effect of travoprost, only 5 cases have been published\textsuperscript{4,6–8} of which involved corneal edema\textsuperscript{4,8} (even though the case of Faulkner previously exhibited predisposing cornea guttata) and another case was relatively doubtful because the patient exhibited recent phacotrabeculectomy surgery which could have influenced another type of uveitis.\textsuperscript{5} The only difference with the patient of this paper is that his uveitis was diagnosed after 5 years of treatment while in the 4 described cases appeared after the application of a few dosages.

According to the above data, the probability of one eye comprising pharmacological anterior uveitis due to travoprost (only four reported cases\textsuperscript{4,6–8} and hyperchromia iridis (5–30% in a different studies) is very small. In addition the fact of not knowing that the patient was in treatment with Travatán\textsuperscript{8} initially lead the authors to erroneously diagnose Fuchs uveitis.

The authors are not aware of any publication of a similar case reporting the joint existence of the 2 side effects described above, as well as other side effects which are also described such as orbital fatty atrophy causing increased depth of the superior sulcus.

**Conflict of interests**

No conflict of interest has been declared by the authors.

**References**