Oral propranolol and intravitreal ranibizumab for refractory serous macular detachment secondary to retinal capillary hemangioblastoma

Propranolol e inyecciones intravítreas de ranibizumab en el desprendimiento seroso macular secundario a hemangioblastoma retiniano capilar

Dear Sir,

Several publications have reported the efficacy of oral propranolol in the treatment of infantile hemangioma and the reduction of subretinal exudation secondary to diffuse and circumscribed chorioidal hemangiomas. We have successfully treated macular detachment secondary to exudation of a capillary retinal hemangioma (CRH) with 80 mg a day in the case of a relapse to intravitreal treatment with 0.5 mg of ranibizumab (Fig. 1).

The patient was a male, 30, who visited due to diminished vision in the left eye. Exploration revealed a CRH which was superior and nasal to the papilla, with associated macular serous detachment and visual acuity diminished to 20/200.

Initially, the CRH periphery was treated with laser photocoagulation and intravitreal injection of 0.5 mg of ranibizumab (Lucentis, Genentech, San Francisco, USA). Fifteen days later subretinal fluid volume diminished and best corrected visual acuity (BCVA) increased to 20/25. However, one month after the initial combined therapy the subretinal fluid increased and BCVA diminished 20/200. A second ranibizumab 0.5 mg intravitreal injection was administered and we prescribed 80 mg/day oral propranolol (the usually applied therapeutic

Fig. 1 – Left ocular fundus photograph (A) showing the baseline appearance of the retinal capillary hemangioblastoma in the superior and nasal retinal periphery. Spectral domain optic coherence tomography-yellow line-(B) shows cystic changes in the external nuclear layer (triangles) and serous macular detachment (arrows). Two weeks after the first treatment (C), subretinal liquid diminished and retinal edema resolved.

dose for treating simple cardiac insufficiency and systemic arterial hypertension).

One month later, left eye BCVA improved up to 20/50 with complete resolution of the macular serous detachment (Fig. 2). Three months after beginning oral propranolol treatment, BCVA had improved up to 20/20 and the macular area remained without subretinal fluid, at which point the patient decided to discontinue oral treatment.

After 3 months without propranolol treatment, the patient exhibited another relapse of the macular serous detachment with BCVA diminishing to 20/60. The therapeutic approach consisted in the administration of another 0.5 mg ranibizumab intravitreal injection and the reinstatement of 80 mg per day of oral propranolol. In subsequent checkups, BCVA was 20/20 and spectral domain optic coherence tomography revealed complete absence of subretinal fluid.

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Fig. 2 – Monthly follow-up with thickness maps of the macular serous detachment secondary to retinal capillary hemangioblastoma. Initial treatment with ranibizumab intravitreal injection and laser photoacoagulation around the lesion (A), with significant improvement after 15 days (B). However, two weeks later the lesion reappeared (C) and was treated with 0.5 mg intravitreal ranibizumab and 80 mg propranolol every 24 h (D, E), observing subretinal fluid regression. During propranolol treatment (D–F) no exudative recurrence was observed. Upon discontinuation of oral propranolol treatment (G), 2 months later the macular central thickness increased from the superior and nasal area (H). The administration of another intravitreal ranibizumab injection and the reinstatement of oral propranolol (L–N) produced successful exudation control. After 15 months follow-up, central macular thickness was of 206 mm (O).
Oral propranolol is a nonselective beta-blocker that could play a relevant role in exudative control of retinal hemangioblastomas, as during oral propranolol treatment the size of the lesion remained stable and exudative activity was entirely inhibited. The relapse exhibited by the patient during the months in which he discontinued propranolol treatment gave rise to its reintroduction. Since then, 18 months have passed without relapse, which led us to take the decision to prescribe the treatment indefinitely.

Propranolol inhibits the vasodilating effect of adrenergic molecules by inhibiting Beta receptors of endothelial cells, promoting vasoconstriction and diminished blood flow in the hemangioma. In addition, it has an angiogenesis-inhibiting capacity because adrenaline and noradrenaline can induce the expression of VEGF and blocking the action of these molecules by means of propranolol could diminish the concentration thereof. There is a range of possible action mechanisms that reduce exudative activity and therefore treatment with oral propranolol could be considered as a therapeutic option in ocular hemangioblastomas that do not respond to conventional therapies.

To the best of our knowledge, this is the first described case of a serous macular detachment secondary to CRH successfully treated with oral propranolol. Subsequent studies should be proposed to confirm the potential efficacy of propranolol in the treatment of CRH.

REFERENCES


P. Hernández-Martínez a,*, R. Gallego-Pinazo a, R. Dolz-Marco b, J.F. Arevalo c,d, M. Díaz-Llopis b, A. Cisneros Lanuza a
a Departamento de Oftalmología, Hospital Universitario y Politécnico La Fe, Valencia, Spain
b Departamento de Cirugía, Facultad de Medicina, Universidad de Valencia, Valencia, Spain
c Retina Division, Wilmer Eye Institute, Johns Hopkins School of Medicine, Baltimore, MD United States
d Vitreoretinal Division, King Khaled Eye Specialist Hospital, Riad, Saudi Arabia
* Corresponding author.
E-mail address: pablooftalmologia@gmail.com (P. Hernández-Martínez).

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Comparative study of modification of the osmolarity in graft versus host disease

Estudio comparativo de la modificación de la osmolaridad en enfermedad injerto contra huésped

Dear Editor,

Patients with the Graft Versus Host Disease (GVHD) develop severe ocular dryness that produces significant modifications in the ocular surface and is difficult to treat. Our study endeavors to estimate ocular surface osmolarity in patients with GVHD and to demonstrate the possible differences in osmolarity evolution by the use of a hypo-osmolar tear (Lubristil®) (Angelini, Barcelona, Spain) versus a non-hypo-osmolar tear (Artific®) (Angelini, Barcelona, Spain).