INTRAVITREAL INJECTION OF BEVACIZUMAB FOR PSEUDOPHAKIC CYSTOID MACULAR EDEMA RESISTANT TO STEROIDS

INYECCIÓN INTRAVÍTREA DE BEVACIZUMAB PARA EDEMA MACULAR QUÍSTICO PSEUDOFÁQUICO RESISTENTE A ESTEROIDES

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ABSTRACT

Clinical case: A 71-year-old man presented with pseudophakic cystoid macular edema (PCME) and a visual acuity (VA) of 0.1. He had been treated with 2 intra-vitreal injections of triamcinolone acetonide (4 mg) 16 months previously. One week after the intra-vitreal injection of bevacizumab (1.25 mg), VA improved to 0.33, and the OCT demonstrated decreased macular thickness. Two months after the injection, no ocular complications were observed, VA was 0.5 and the OCT showed a significant reduction in the retinal thickness.


Key words: Bevacizumab, intra-vitreal injection, pseudophakic cystoid macular edema, cataract surgery, corticoid resistant.

RESUMEN

Caso clínico: Paciente varón de 71 años con edema macular quístico pseudofáquico (EMQP) y agudeza visual (AV) de 20/200 que fue previamente tratado con 2 inyecciones intravítreas de acetónido de triamcinolona (4 mg) 16 meses atrás. Una semana tras inyección intravítrea de bevacizumab (1,25 mg), la AV mejoró a 20/60, y la OCT demostró reducción del espesor retiniano. Dos meses tras la inyección los autores no observaron ninguna complicación ocular, una AV de 20/40 y la OCT reveló un importante adelgazamiento retiniano.

Conclusión: La inyección intravítrea de bevacizumab puede aportar una nueva opción para los pacientes con EMQP resistente a tratamiento.

Palabras clave: Bevacizumab, inyección intravítrea, edema macular quístico pseudofáquico, cirugía de la catarata, resistente a corticoides.
INTRODUCTION

Pseudophakic cystoid macular oedema (PCME) that is resistant to periocular and intraocular steroids usually does not evolve well on its own (1). Cyst-causing accumulation of fluid/oedema in the macula after eye surgery (typically cataract surgery) is also known as Irving-Gass syndrome. The fluid may be intercellular, located in the external plexiform layer and the internal nuclear layer, or intracellular, thus causing degeneration of the Müller cells with intracellular vacuolization.

There are multiple theories for PCME pathogenesis, such as intraocular inflammation and vitreomacular traction, leading to a tear in the blood-retinal barrier. Inflammatory mediators, such as prostaglandins and leukotrienes, have been linked to its pathogenesis. Prostaglandins are vasoactive substances that increase vasodilation and permeability of the retina’s vessels. These effects lead to a weakening of the intercellular bonds of the retinal capillary endothelium as well as a decrease in retinal fluid pumping by the retina’s pigment epithelium, leading to a tear in the blood-retinal barrier. On another hand, leukotrienes are agents with chemotactic properties over other inflammatory cells.

Steroids have a major role in treating PCME secondary to uveitis by inhibiting the phospholipase enzyme, which is a mediator in the release of arachidonic acid from the cell wall, and necessary for prostaglandin and leukotriene synthesis. However, some patients do not tolerated steroids well because of the secondary ocular and systemic side-effects. There is a sub-population of patients with intraocular inflammation who simply do not respond to steroids (1).

Bevacizumab (Avastin, Genentech) is a recombinant humanized anti-VEGF (vascular endothelial growth factor) monoclonal antibody that binds to all VEGF-A isoforms. In this report we describe the effect that a one-time intravitreal injection of bevacizumab (1.25 mg) had on a steroid-resistant PCME patient.

CASE STUDY

A 71-year-old male came to our service showing a best-corrected visual acuity (BCVA) of 20/200 in his right eye and cystic macular oedema (CME) of the macula in the absence of vitreomacular traction associated with a serous detachment of the neuroepithelium identified through fluorescein angiography (FAG) (Fig. 1) and optical coherence tomography (OCT) (Fig. 2). The patient had a history of progressive loss of visual acuity in the right eye after cataract surgery done by phacoemulsification and an intraocular lens implant placed inside the capsular sac, with no complications, in both eyes four years ago, and with no complications in the placement of the soft intraocular lens in capsular sac. After ruling out other causes of uveitis, a PCME diagnosis was reached and the patient was
given two injections of triamcinolone acetonide (4.0 mg), 24 and 16 months ago, respectively, with poor results, reaching a 20/100 BCVA after the first shot and 20/160 after the second. Aside from the alterations found through the OCT and angiograph, the rest of the ophthalmological exam was normal and the aetiological exams for uveitis were negative.

A week after the intravitreal bevacizumab injection (1.25 mg), his BCVA improved to 20/60 and the OCT showed a decrease in the macular thickness (from 677 µm initially to 406 µm) along with a decrease in the intraretinal oedema, height and extension of the serous detachment of the neuroepithelium (Fig. 3).

Eight weeks later, the patient presented a 20/40 BCVA and a noticeable thinning, which was seen through OCT (from 677 µm to 356 µm) (Fig. 4).

At the end of the follow-up period (three months after the injection), the authors did not see any eye complications, a 20/40 BCVA with a significant decrease in retinal thickness (from 677 µm to 267 µm) (Fig. 5).

### DISCUSSION

Intravitreal injections of triamcinolone acetonide and pars plana vitrectomy have been seen to be effective in most cases of pseudophakic cystoid macular oedema. However, some cases are refractory and lead to a severe loss of visual acuity (1).

Bevacizumab (Avastin, Genentech) is a recombinant humanized anti-VEGF monoclonal antibody that was initially approved for treating metastatic colorectal cancer. In studies using off-label injections of intravenous bevacizumab for neovascular ARMD (age-related macular degeneration) initial results have been promising (2,3). The intravitreous use of bevacizumab has recently been reported as improving these results and showing a good safety profile (4,5).

Four weeks after the 1.25 mg bevacizumab injection, the patient showed an improvement in vision and the OCT showed a decrease in macular thickness, both in the intraretinal cystic oedema as well as the neuroepithelial detachment. The anatomical and functional results improved during the entire three-month follow-up period, until a retinal thickness of 267 µ was reached at the fovea.

The intravitreal bevacizumab injection has been an effective and safe alternative in the treatment of this patient suffering from treatment-resistant pseudophakic cystoid macular oedema.

### REFERENCES


