Intravitreal Ranibizumab for the treatment of macular edema secondary to malignant hypertension

Brito RC et al. Intravitreal Ranibizumab in macular edema secondary to malignant hypertension.

ABSTRACT

Our purpose is to report a clinical case of bilateral macular edema caused by malignant hypertension in a 35-year old male. Patient presented with bilateral low visual acuity, massive macular edema, retinal hemorrhages and papilledema. Soon after he was diagnosed with other target-organ lesions, on heart and kidneys. After 6 months of controlled blood pressure, an increase in visual acuity and a small decrease of macular edema, we effectively treated the lower vision eye with two intravitreal ranibizumab injections, improving anatomy and function on both eyes. Even though we only treated one eye with ranibizumab, the contralateral eye also improved visual acuity and central macular thickness. This probably happened due to systemic absorption of ranibizumab. To the best of our knowledge, this is the first report of ranibizumab use in macular edema due to malignant hypertension.

Keywords: Malignant hypertension; macular edema; ranibizumab.

INTRODUCTION

Malignant hypertension is a rare hypertensive emergency in which systolic blood pressure is higher than 140 mmHg and target-organ lesions are present. Its ocular manifestations are due to vascular constriction, arteriolar obstruction and blood-ocular barrier disruption, and retinopathy, choroidopathy and optic neuropathy. Without treatment, the mortality rate is higher than 90% in one year.

CASE REPORT

A 35-year-old caucasian male patient, with no relevant medical history, came for urgent ophthalmologic evaluation because of bilateral progressive blurred vision and holocranial headache for the past two weeks. Best-corrected visual acuity (BCVA) on the right eye (OD) was counting fingers and on the left eye (OS) was 0,1. Pupillary reflexes were symmetric and slow, medium were clear and the average intraocular pressure was 14 mmHg on OD and 13 mmHg on OS. Funduscopy of both eyes showed flame and dot hemorrhages in all quadrants, retinal edema with massive macular edema, hard exudates in the macula forming a macular star and disc edema (Figure 1). Macular optical coherence tomography (OCT) revealed a central macular thickness (CMT) of 984 µm and 968 µm on OD and OS, respectively (Figure 2). Blood
of choroidal fluorescence by retinal hemorrhages, multiple microaneurysms in the peripapillary region, and fluorescein leakage in late phases related to the retinal edematous areas (Figure 3).

After 4 months of ophthalmologic observation only, and normal blood pressure levels, BCVA increased to 0,1 on OD and 0,6 on OS, and CMT decreased to 644 µm on OD and 600 µm on OS (Figure 4). Most of retinal hemorrhages were reabsorbed, disc edema resolved and a preretinal hemorrhage was seen on OS (Figure 5). After 6 months, BCVA remained stable but CMT lightly increased, so we decided to treat only the eye with lower visual acuity, the right eye, with intravitreal ranibizumab 0,5 mg. CMT returned to normal on both eyes after two injections with one month interval (Figure 6), and BCVA improved to 0,3 and 0,8 on OD and OS, respectively. After two years of follow-up, CMT and visual acuity are stable, as well as blood pressure levels.

DISCUSSION

In this clinical case, malignant hypertension was the first manifestation of systemic hypertension, which is rare, but must be kept on mind. The recognition of malignant hypertension has implications for the eye and general health of patients.

Intravitreal ranibizumab 0,5 mg was effective in the treatment of macular edema due to malignant hypertension, improving both visual function and anatomic retinal profile on macular OCT. Initially, in malignant hypertension, there is vascular constriction in choroidal and retinal vasculatures that cause ischemia, and these are followed by vasodilation and increased vascular permeability. Induced retinal edema, in turn, produces more ischemia and vascular endothelial growth factor release, which can be blocked by intravitreal ranibizumab.

Even though we only treated the right eye with ranibizumab, the left eye also improved BCVA and CMT. We think this happened because of systemic absorption of ranibizumab, despite the fact that ranibizumab is a monoclonal antibody fragment, having a shorter systemic half-life without the Fc domain, of about 2 hours after entering systemic circulation from the eye.5 And besides the fact that systemic absorption of ranibizumab given intravitreally seems to be minimal.

Final OCT shows IS/OS line distortion and retinal pigment epithelium atrophy on OD, signs of irreversible damage of the retina, that can explain the low visual acuity of the treated eye, even after the achievement of a normal macular thickness. ■

References


