Intravitreal Bevacizumab Therapy in Non-Arteritic Anterior Ischemic Optic Neuropathy

Non-Arteritik İskemik Optik Nöropatide İntravitreal Bevasizumab Tedavisi

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ABSTRACT

Up to date there is no golden standard for the treatment of non-arteritic ischemic optic neuropathy. Here we report two cases which underwent intravitreal bevacizumab injection for non-arteritic ischemic optic neuropathy. In the first case, visual acuity changed from 2/200 to 20/400 at 2 week and 4/200 at one year. In the second case, visual acuity decreased from 20/30 to 20/400 with a worsened visual field defect at 6 week and to 20/200 at 5.5 month. Bevacizumab did not seem to improve visual outcome in non-arteritic ischemic optic neuropathy.

Key Words: Anti-vascular endothelial growth factor, bevacizumab, non-arteritic ischemic optic neuropathy.

ÖZ

Günümüzde non-arteritik iskemik optik nöropati tedavisinde altın standart bulunmamaktadır. Bu yazıda non-arteritik iskemik optik nöropati nedeniyle intravitreal bevasizumab enjeksiyonu yapılan iki olgu bildirilmektedir. İlk olguda 0.01 olan görme keskinliği tedaviden sonraki ikinci haftada 0.05, 1. yılda ise 0.01 saptanmıştır. İkinci olguda ise 0.7 olan görme keskinliği tedaviden sonraki 6. haftada 0.05'e düşmüş ve görme alanı defekti artmış, 5.5 ayda ise 0.1 olarak izlenmiştir. Non-arteritik iskemik optik nöropatide bevasizumabın görsel sonuçlara olumlu bir etkisi saptanmamıştır.

Anahtar Kelimeler: Bevacizumab, non-arteritik iskemik optik nöropati, vasküler endotelyal büyüme faktörü inhibitörü.

INTRODUCTION

Non-arteritic anterior ischemic optic neuropathy (NAION) is a common cause of optic nerve related visual loss in the elderly population. To date, there is no consensus on the ideal management of NAION. Systemic steroid therapy, intravitreal triamcinolone or intravitreal bevacizumab (IVB) injections have recently been reported to show some benefit.¹⁻⁴ Here we report the failure of intravitreal bevacizumab treatment in two cases of NAION.

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SINGLE FIELD ANALYSIS

CENTRAL 24-2 THRESHOLD TES

FERRITION MONITOR'S CREE/BLIND SPE PUPEL DIAMETER FEXATEDN TRACET: CENTRAL FENRITION LOSSES: 4/13 N FALSE POS ERRORS: FALSE NEG ERRORS: 16 1 TEST DURATION: 03:0 FONER: 25 DB HAN LOW TEST RELIGED ITY HA SCREEL STRITS



STND F FTFLD AND YSTS



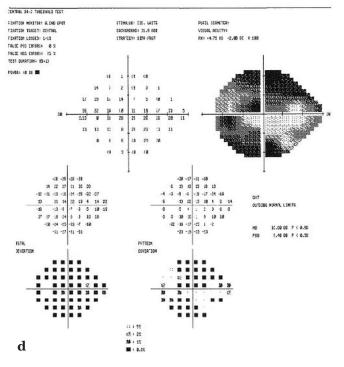


Figure 1: Colour fundus photo shows blurred disc margin with splinter haemorrhages in the left eye (a). Visual field revealed superior altitudinal defect and inferotemporal arcuate defect (b) Pale optic disc two weeks after intravitreal bevacizumab injection. (c) Visual field shows minimal changes at 2 week visit (d).

CASE REPORT

Case 1

b

Sixty seven years old male presented with blurred vision temporally on left field of the left eye for one week. Best corrected visual acuity (BCVA) was 2/200 with a relative afferent pupillary defect in the left eye. Fundus examination showed blurred disc margin with splinter haemorrhages in the left eye (Figure 1a). Humphrey visual field (VF) revealed superior altitudinal defect and inferotemporal arcuate defect in the left eye (Figure 1b). The patient was diagnosed with NAION and received a 1.25 mg IVB in his left eye.

Two weeks later, BCVA was 20/400 and optic disc was pale with resolving disc oedema (Figure 1c). VF did not improve despite resolution of oedema (Figure 1d). At 1 year visit, BCVA was 4/200 in the left eye.

Case 2

Fifty four years old male presented with visual loss in his right eye. He had a history of NAION in his left eye which occurred 3 months ago and the right eye was symptomatic for a few weeks. The previous workup including cranial computed tomography, magnetic resonance imaging and spinal fluid analysis were negative.

EYE: RIGHT

HA LOW TEST RELIABLITY HA

8.47 DB P (8.5%

DITSIDE NIGHT LENTES

PUPIL DIRMETER



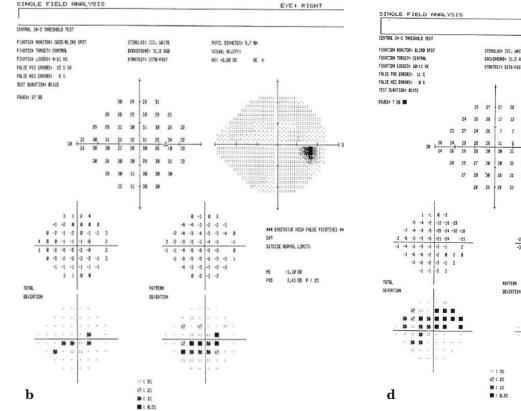


Figure 2: Colour fundus photo shows disc pallor in the left eye and disc oedema in the right eye (a) and visual field shows central scotoma (b). Mildly regressed disc oedema with a peripapillary haemorrhage at 6-week (c) and visual field shows superotemporal defect extending to the center (d).

In our clinic, BCVA was 20/30 in the right eye and 8/200 with temporal fixation in the left eye. Fundus examination disc pallor in the left eye and disc oedema in the right eye (Figure 2a). VF revealed central scotoma in the right eye (Figure 2b).

The patient received 1.25 mg IVB injection in his right eye. BCVA was 20/200 at one week and 20/400 at 6 week follow-up. Disc oedema in the right eye mildly regressed with a peripapillary haemorrhage nasally at 6 week (Figure 2c).

VF demonstrated superotemporal defect extending to the center despite decreasing optic disc swelling (Figure 2d). BCVA was 20/200 in the right eye and 2/200 in the left eye at 5.5 month.

DISCUSSION

Currently there is no proven treatment altering the course of NAION. IVB is being widely used in several ocular disorders in which vascular leakage and oedema are involved. In NAION, inhibition of VEGF is hypothesized to reduce vasogenic oedema by decreasing the vascular permeability and therefore provide some relief to the optic nerve tissue.⁴ Bajin et al.,⁵ reported 4 cases of NAION with a mean duration of symptoms of 7.8 days, with increase in VA after intravitreal ranibizumab injection. Bennett et al.⁴, reported a case of NAION with 3 weeks duration and documented resolution of disc swelling, decrease in dye leakage and improvement of VA, which remained stable for 6 months after treatment with IVB.

Prescott et al.,⁶ described 5 NAION patients following IVB injections with a minimum 2 months followup. VA improved from 20/150 to 20/40 in one but decreased in 4 patients. Visual field improved slightly in 1, remained stable in 1 and progressed in 3 patients. The case with an increase in VA had the injection on the next day of presentation and the duration of symptoms was changing between 5 days to 5 weeks in the remaining cases. In our cases, VA remained the same after injection in one case for 1 year and worsened after injection and did not improve for 5.5 months in the second one. The duration of symptoms was one week in one patient and few weeks in the other. The time interval between the beginning of the symptoms and the treatment might be important since the hypothesis of IVB treatment is based on resolution of the oedema and thus preventing or limiting the further damage on axons which is caused by the mechanical effect of the oedema. However, the number of cases in literature is limited to clearly determine the effect of timing of treatment on the visual outcome. It has been suggested that bevacizumab might induce NAION. Four cases have been reported to develop NAION following IVB injections; of these cases, one had diabetic macular oedema, one had CNV secondary to angioid streaks and two had wet AMD.⁷⁻¹⁰ The suggested mechanisms are the impairment of autoregulation of optic nerve circulation with the blockage of VEGF and transient IOP increase which causes the decrease of perfusion pressure of the optic nerve head.⁸⁻¹⁰

In conclusion, unlike proven VEGF driven diseases such as age-related macular degeneration, diabetic retinopathy or retinal vein occlusion in which IVB has very impressive effect, in NAION, the effect could be questionable, even undesirable. IVB should not be part of clinical practice unless beneficial outcome is proven with randomized controlled studies.

REFERENCES/KAYNAKLAR

- 1. Hayreh SS, Zimmerman MB. Non-arteritic anterior ischemic optic neuropathy:Role of systemic corticosteroid therapy. Graefes Arch Clin Exp Ophthalmol 2008;246:1029-46.
- Kaderli B, Avci R, Yucel A, et al. Intravitreal triamcinolone improves recovery of visual acuity in nonarteritic anterior ischemic optic neuropathy. J Neuroophthalmol 2007;27:164-8.
- 3. Yaman A, Selver OB, Saatci AO, et al. Intravitreal triamcinolone acetonide injection for acute non-arteritic anterior ischaemic optic neuropathy. Clin Exp Optom 2008;91:561-4.
- 4. Bennett JL, Thomas S, Olson JL, et al. Treatment of nonarteritic anterior ischemic optic neuropathy with intravitreal bevacizumab. J Neuroophthalmol 2007;27:238-40.
- Bajin MS, Selver OB, Taskin O, et al. Single intravitreal ranibizumab injection in eyes with acute non-arteritic anterior ischaemic optic neuropathy. Clin Exp Optom 2011;94:367-70.
- 6. Prescott CR, Sklar CA, Lesser RL, et al. Is intravitreal bevacizumab an effective treatment option for nonarteritic anterior ischemic optic neuropathy? J Neuroophthalmol 2012;32;51-3.
- 7. Huang JY, Ozaki H, Hayashi H, et al. Anterior ischemic optic neuropathy following intravitreal bevacizumab. Jpn J Ophthalmol 2010;54:252-4.
- Hosseini H, Razeghinejad MR. Anterior ischemic optic neuropathy after intravitreal injection of bevacizumab. J Neuroophthalmol 2009;29:160-1.
- 9. Bodla AA, Rao P. Non-arteritic ischemic optic neuropathy followed by intravitreal bevacizumab injection: is there an association? Indian J Ophthalmol 2010;58:349-50.
- Ganssauge M, Wilhelm H, Bartz-Schmidt KU, et al. Nonarteritic anterior ischemic optic neuropathy (NA-AION) after intravitreal injection of bevacizumab (Avastin) for treatment of angoid streaks in pseudoxanthoma elasticum. Graefes Arch Clin Exp Ophthalmol 2009;247:1707-10.