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The Effect of Intravitreal Injection of Avastin for Neovascular Age-related Macular Degeneration

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Abstract

Objective: To determine the effect of intravitreal injection of Avastin for Neovascular age related macular degeneration.

Methods: 76 Patients were selected by sampling. Visual acuity was evaluated by Snellen chart at a distance of 6 meters with good light and single eye. Accordingly, logarithm of Minimum Angle of Resolution (MAR) was recorded. Avastin (1.25 mg) in 0.05 ml of liquid was injected with a sterile syringe and needle. Second and third injections were repeated one month after the first injection.

Results: The results of Visual acuity (VA) in the patients (In log MAR): Mean ± SD of visual acuity (VA) in patients before the study was 0.906 ± 0.080, One month after injection was 0.818 ± 0.097, 3 months after injection was 0.6 ± 0.126 and 6 months after injection was 0.488 ± 0.164.

Conclusion: Intravitreal avastin (1.25 mg) is well tolerated and associated with improvement in VA and decreased retinal thickness by OCT and also reducing angiographic leakage in most of the patients. Decreasing BFVs in all retrobulbar arteries, suggests that avastin exerts a short-term regional effect.

Keywords: Visual acuity; Neovascularization of the retina; Avastin; ARMD.

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Introduction

Age – related macular degeneration (AMD) is the most common cause of vision loss in people over 60 years old in developed countries and more than 30% of adults 75 years old and older and 8-6% of these people develop severe vision loss. Sometimes AMD can occur in people under 40 years old [1]. Some studies have shown that antioxidants and compounds of carotenoid family may have a protective role against AMD, Thus the role of factors such as diet and lifestyle associated with this disease should be considered [2].

The greatest risk factor is age and those over 60 years old are at greater risk than other age groups. Although the risk of AMD is only 1% at age 55-64, this prevalence rises to 4% for those over 65 years old. After increasing age, smoking is the strongest predictor for AMD. Smoking can have a risk of the disease twice. Scientists are trying to find the genes responsible for the hereditary form of the disease.

Patients who present with acute vision loss, are those who have suffered bleeding of Choroidal vessels neovascularization. Other patients with choroidal neovascularization, pigment epithelium detachment, or accumulation of fluid under the retina or with central scotoma, Metamorphopsia occurs that straight lines appear crooked or some cases complain about cluttered vision. They fail to read, and color understanding changes or decreases. Central scotoma in patients whose visual acuity is below the level of reading and the legal driving, remains constant, while peripheral vision usually remains [3,4].

Although much progress has been made in the treatment of AMD, visual loss cannot be reversed. For patients who have lost their vision, there are means that with magnifying lenses and bright light help to improve vision. Some of this mean transfer the image to the more peripheral parts of the retina and out of range of macula. Experiments show that treatment with topical antibiotics, with or without medical anti-VEGF treatment, is able to suppress the development of new blood vessels in AMD. In another study of patients with AMD new vessels resistant to monotherapy anti-VEGF, anti-VEGF therapy combined with triamcinolone acetonide has been reported to be effective [5-7].

Single Nucleotide Polymorphisms (SNP) in VEGF - A gene was studied. Patients with SNP visually showed a significantly better outcome. Although Anti-VEGF therapy is with clear improvement

in vision of wet AMD patients, this response in different patients is various and identifying factors that affect the response, underlies researches for better treatments in the future [5-7].

Research Methodology

A total of 76 Patients were selected by sampling and enrolled with the consent and permission of the Ethics Committee and Registration in clinical applications. Inclusion criteria included: Age over 65 years, no previous treatment, Angiographic evidence of leakage or increased macular thickness by OCT (Optical Coherence Tomography), new subretinal hemorrhage with loss of visual acuity. Before the first injection, fluorescein angiography was performed to confirm the macular leakage. Visual acuity was determined before intervention. Visual acuity was evaluated by Snellen chart at a distance of 6 meters with good light and single eye (first right eye, and then left one). Patients whose visual acuity was detectable at distance of 6 meters or 20 feet with measures of visual acuity on the Snellen chart, were recorded as logarithm of Minimum Angle of Resolution (MAR) (minimum spatial frequency of Optotype chart of YOUNG chart was 1.5 log MAR) and those whose visual acuity was less, closed to the chart and we determined Optotype in a shorter distance. Patients who distinguished Optotype 20/600 Equivalent 1.5 log MAR at a distance of 2 feet, their vision was considered equal to 2.5 log MAR, and patients who were not able to detect the Optotype, was considered as 3 log MAR and those who were not able to detect light, was considered as 4 log MAR. The intravitreal drug injection was performed by an ophthalmologist. The drug was prepared in the injection day sterile. Injection was performed under local anesthesia, and after washing the eyelids and eyebrows with 10% povidone iodine solution and eye by using the eye drops of 5% povidone iodine in operation room. Face and body were covered with sterile sheets. Avastin (1.25 mg) in 0.05 ml of liquid was injected with a sterile syringe and needle (30). Second and third injections were repeated one month after the first injection. Data were entered into SPSS 15 software.

Results

In this study, 76 patients referred to the Valiasr Eye Clinic and had the inclusion criteria, were selected by convenience sampling method. Distribution of patients according to the sex: 39.5% of the patients were male and 60.5% of them were female.

The mean ± SD age of the participants in this study was 74.38 ± 10.160. Visual acuity (VA) in patients before injection and at one, three and six months after the treatment was measured. The results of Visual acuity (VA) in the patients (In logMAR): Mean ± SD of visual acuity (VA) in patients before the study was 0.906 ± 0.080, One month after injection was 0.818 ± 0.097, 3 months after injection was 0.6 ± 0.126 and 6 months after injection was 0.488 ± 0.164. As regards $P<0.001$, there is significant difference between the results of measurements of visual acuity. To determine whether the difference between what the test is, Bonferroni test was used. The mean visual acuity difference compared to measurements in before, one, three, and six months after the treatment was significant ($P<0.001$). The only complication observed during this study, was eye inflammation (Uveitis) that was created in 2 patients (2.631%).

Discussion

Now-a-days Intravitreal anti-VEGF therapy is the usual treatment for exudative AMD. But its effect on choroidal and retinal haemodynamics has not been tested yet, by persistent reduction in blood flow, its repeated injections may be contradicted. Avastin's two mechanisms are: inducing vasoconstriction soon after injection and decreasing in capillary density. The intravitreal concentration of avastin is highest just after injection, and its half-time decay is 9.82 days [8].

In this study, we observed a mean decrease VA in 6 months after avastin injection. It is unlikely that the decrease within these measures was caused by the occlusion of subfoveal CNV solely as a result of the flow in CNV is negligible compared with the flow in the temporal choroid. Thus, though the dose of 1.25 mg avastin is spare to attenuate CNV, it should conjointly induce additional widespread hypoperfusion of the choroidal circulation [9]. The diffusion of enormous molecules from the choroid to the periocular tissues through the sclerotic tissue is a known process, and it should enable avastin to enter the capillary network of the eye's annexae.

In comparison to other studies this test was repeated in the same dosage of bevacisumab or avastin and the decrease of visual acuity (OA) and mean blood flow velocities (BFVs) in the central retinal, temporal posterior ciliary and ophthalmic arteries (CRA, TPCA) were reported as the mechanism mentioned in pervious part that intravitreal injection of bevacizumab was found to induce very early reduction of choriocapillaris endothelial cell fenestration [10]. The point of this article despite others which were mostly a one-month observation is that the period of control and observation is that the period of study on these patients raised to six months and a greater number of cases were controlled. This induced reduction in blood supply and its side effects may have more interests of study in future and also doing a comparison with other agents.

Conclusion

Given that the definitive treatment for wet AMD is unconfirmed and systemic and ocular complications of intravitreal injection of Avastin is still unknown and research in this area has been limited. So, we decided to investigate the effect of intravitreal injection of Avastin in patients with neovascular AMD in controlled conditions. In a short-term study, its results suggest that intravitreal avastin (1.25 mg) is well tolerated and associated with improvement in VA and decreased retinal thickness by OCT and also having reduction in angiographic leakage in most of the patients. Decreasing BFVs in all retrobulbar arteries, suggesting that avastin exerts a short-term regional effect. Therefore, it may induce hypoperfusion of the whole eye, that may correspond to a vascular side-effect. But Further comparative evaluation against other (VEGF) agents and dosing schedule is required.

Conflict of Interests

Authors have no conflict of interests.

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