# EFFICACY OF INTRACAMERAL BEVACIZUMAB INJECTION WITH AND WITHOUT PANRETINAL PHOTOCOAGULATION IN MANAGEMENT OF NEOVASCULAR GLAUCOMA

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## ABSTRACT

**Objective:** To evaluate the efficacy and safety of intracameral bevacizumab with and without panretinal photocoagulation in treating neovascular glaucoma.

**Method:** This prospective interventional case series included fifty eyes of fifty patients were included in this study; twenty five eyes with neovascular glaucoma were treated with one dose of 2.5 mg intracameral bevacizumab only (group I) while other twenty five eyes were treated with one dose of 2.5 mg intracameral bevacizumab and laser panretinal photocoagulation (group II).

**Results:** The study showed that there was no statistical difference regarding visual acuity in group I (P>0.05), while there is a highly significant difference in group II (P <0.001). Also, there was no statistical difference regarding IOP in group I (P>0.05), while there is a highly significant difference in group II (P <0.001).

**Conclusions:** Intracameral bevacizumab and laser panretinal photocoagulation is more effective in treatment of NVG in terms of angle neovascularization regression and IOP control.

## INTRODUCTION

Neovascular glaucoma (NVG) is a serious complication of retinal ischemic disorders, such as vascular occlusions, and proliferative diabetic retinopathy[1]. The hallmark of NVG is new vessels formation of in the iris (NVI), which progress to form a fibrovascular membrane on the surface of the iris. This membrane contracts slowly and closes the anterior chamber angle, thus impeding aqueous outflow and resulting in an elevation in intraocular pressure (IOP), which is difficult to control and often leaves the patient with a blind painful eye[2].

The etiology of NVG is related to the production of vascular endothelial growth factor (VEGF) by the underlying ischemic retina, which in turn stimulates neovascularization[3]. In primates, injection of recombinant VEGF produces NVI and NVG; inhibition of endogenous VEGF prevents retinal ischemia and NVG formation[4,5].

To date, the gold standard in treatment of NVG is laser panretinal photocoagulation (PRP)[1]. Retinal ischemia is reduced after PRP, which in turn decreases the level of VEGF and control of NVG. Nevertheless, sometimes PRP may be difficult, for example in eyes with media opacities like cataract or vitreous hemorrhage. PRP is also less effective in rapidly progressing NVG.

Case series employing anti-VEGF agents in the treatment of NVG have been described[6-11]. They mainly entailed the use of bevacizumab (Avastin®; Genentech, Inc., South San Francisco, CA), which is a full-length humanized monoclonal antibody that binds all isoforms of VEGF. Results so far have been promising in terms of NVI regression and IOP control. To date, however, the US Food and Drug Administration have not yet approved bevacizumab for intraocular use.

## PATIENT AND METHODS

Fifty eyes of 50 patients with rubeosis iridis were included in this study. Ocular examination included visual acuity assessment, slit-lamp biomicroscopy of the anterior segment, gonioscopy, applanation tonometry, indirect ophthalmoscopy and finally fluorescein angiography of iris neovascularization. The iris neovascularization was graded according to the iris angiography grading system. This grading included grade 0: the vessels that fill briefly with fluorescein are radial and do not leak; grade 1: the vessels appear more prominent and tortuous than normal, appear discontinuous, but do not leak fluorescein; grade 2: the vessels are more prominent, nonradial and leak fluorescein; grade 3: the vessels are more prominent, nonradial and leak early in the angiogram (by 20-30 seconds); and grade 4: individual vessels cannot be delineated in the early angiogram (by 20-30 seconds) and the iris appears as a diffuse opaque fluorescent sheet.

All patients received intracameral injection of bevacizumab (avastin), 2.5 mg in 0.1 mL. The following steps were used for injection: Eye speculum application, disinfection of conjunctival sac with povidone iodine 5%, anterior chamber paracentesis was done and then the intracameral injection of bevacizumab (ICB). Pan retinal photocoagulation was applied for twenty five cases after intracameral injection. The patients were followed daily for 1 week and then weekly, for 2 months. The study was approved by the local ethics committee, and all patients signed informed consent

before entering the study. All applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research.

## RESULTS

Table (1): Age and sex distribution of the studied groups

| Item | Males | Females |
|---|---|---|
| No. (%) | 25 (50%) | 25 (50%) |
| Mean ±SD | 53.38 ± 1.99 | |

This table showed the distribution of the 50 studied patients and divided into two equal groups (50%) of both sexes (25 males and 25 females) and showed no statistically significant difference as regard the pathological findings (P >0.05).

The study compared the visual acuity (VA), intraocular pressure (IOP) as well as angular neovascular-ization (ANV) pre- and post-treatment and then statistically analyzed and recorded in the following tables.

Table (2): Pre- and post-treatment VA in the two studied groups

| Visual acuity | Pre-treatment | Post-treatment | r | P value |
|---|---|---|---|---|
| Group I (Mean ±SD) | 1.52 ± 0.38 | 1.16 ± 0.55 | 0.13765 | >0.05 |
| Group II (Mean ±SD) | 1.71 ± 0.34 | 0.87 ± 0.36 | 0.67314 | <0.001 |
| Total (Mean ±SD) | 1.61 ± 0.37 | 1.03 ± 0.48 | 0.23393 | <0.05 |

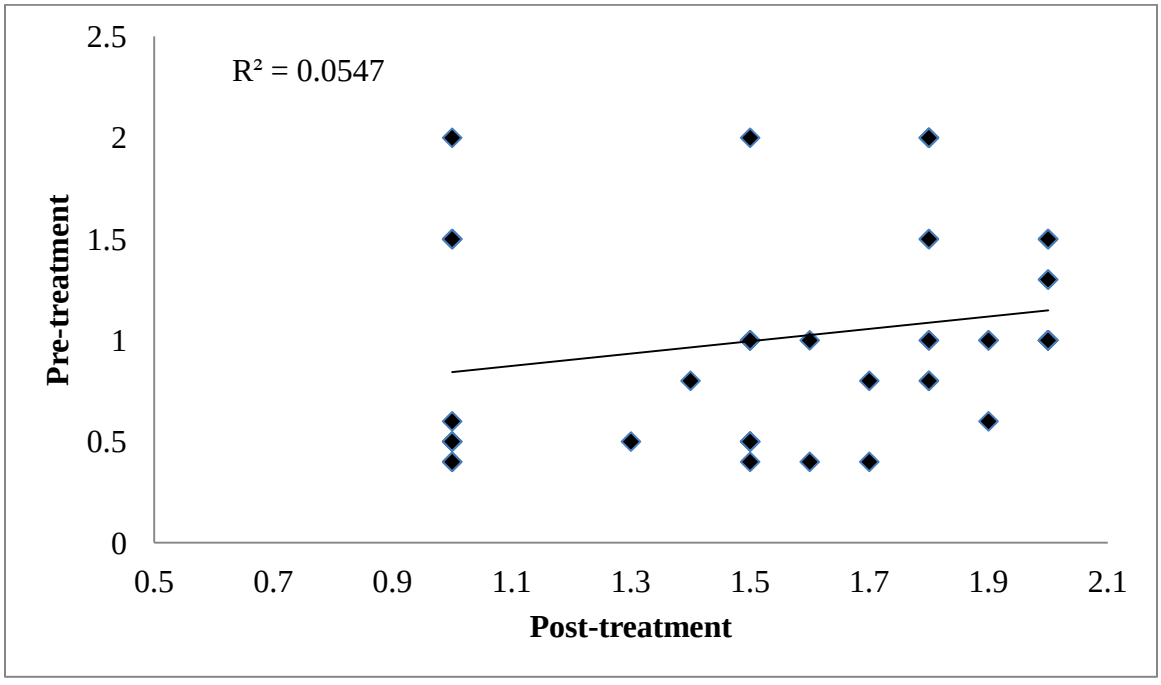


Fig. (1): Correlation coefficient (r) between pre- and post-treatment VA

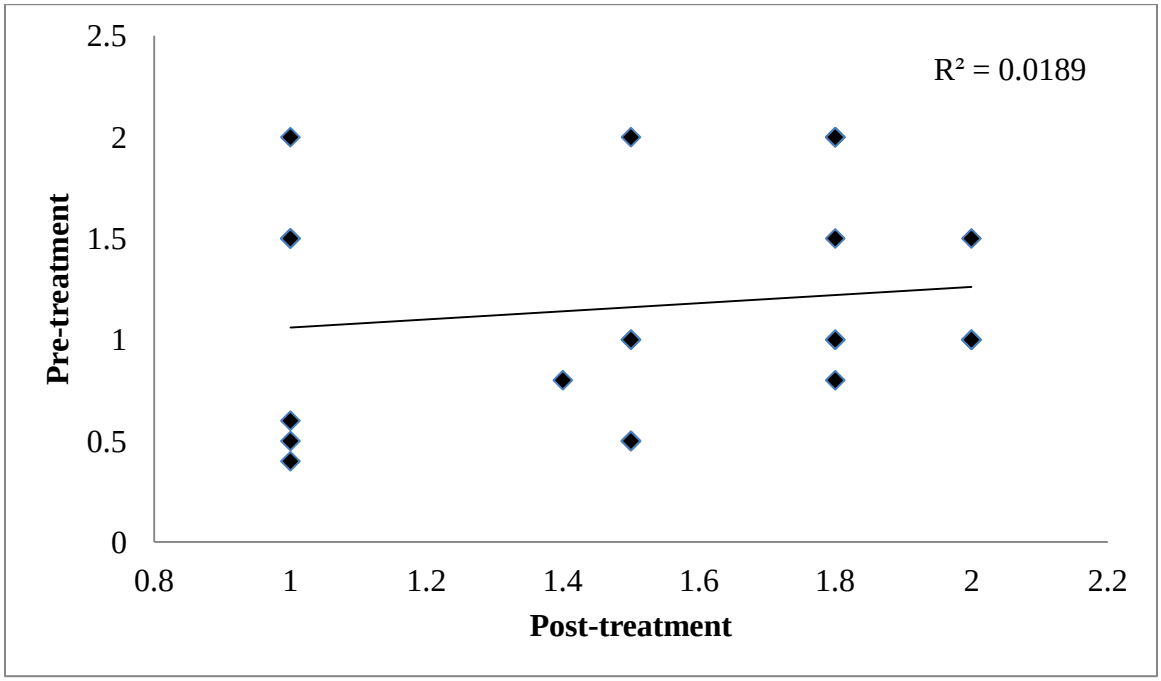


Fig. (2): Correlation coefficient between pre- and post-treatment VA in group I

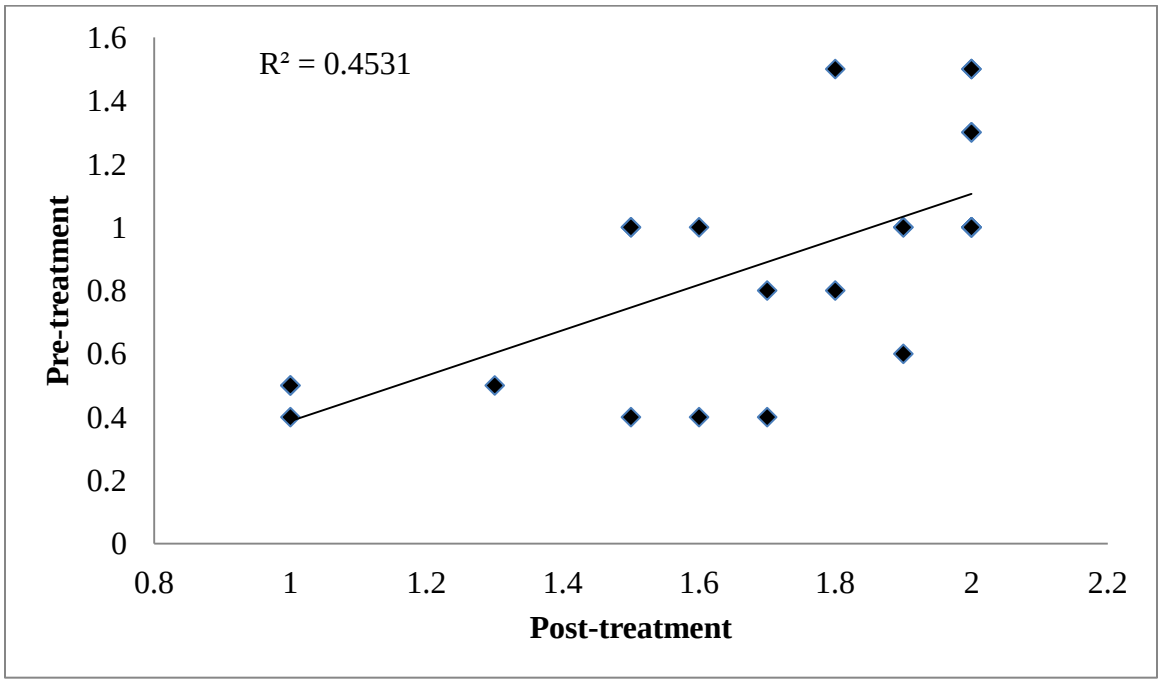


Fig. (3): Correlation coefficient between pre- and post-treatment VA in group II

Table (2) showed that there was no statistical difference regarding visual acuity in group I (P>0.05) as shown in fig. (2), while there is a highly significant difference in group II (P <0.001) as shown in fig. (3) and also the visual acuity in all studied cases showed statistically significant value (P <0.05), illustrated in fig. (1).

Table (3): Pre- and post-treatment IOP in different studied groups

| IOP (mmHg) | Pre-treatment | Post-treatment | r | P value |
|---|---|---|---|---|
| Group I (Mean ±SD) | 28.64 ± 4.91 | 23.88 ± 9.03 | 0.20203 | >0.05 |
| Group II (Mean ±SD) | 32.16 ± 6.95 | 20.32 ± 5.65 | 0.85106 | <0.001 |
| Total (Mean ±SD) | 30.4 ± 6.21 | 22.1 ± 7.67 | 0.36826 | <0.05 |

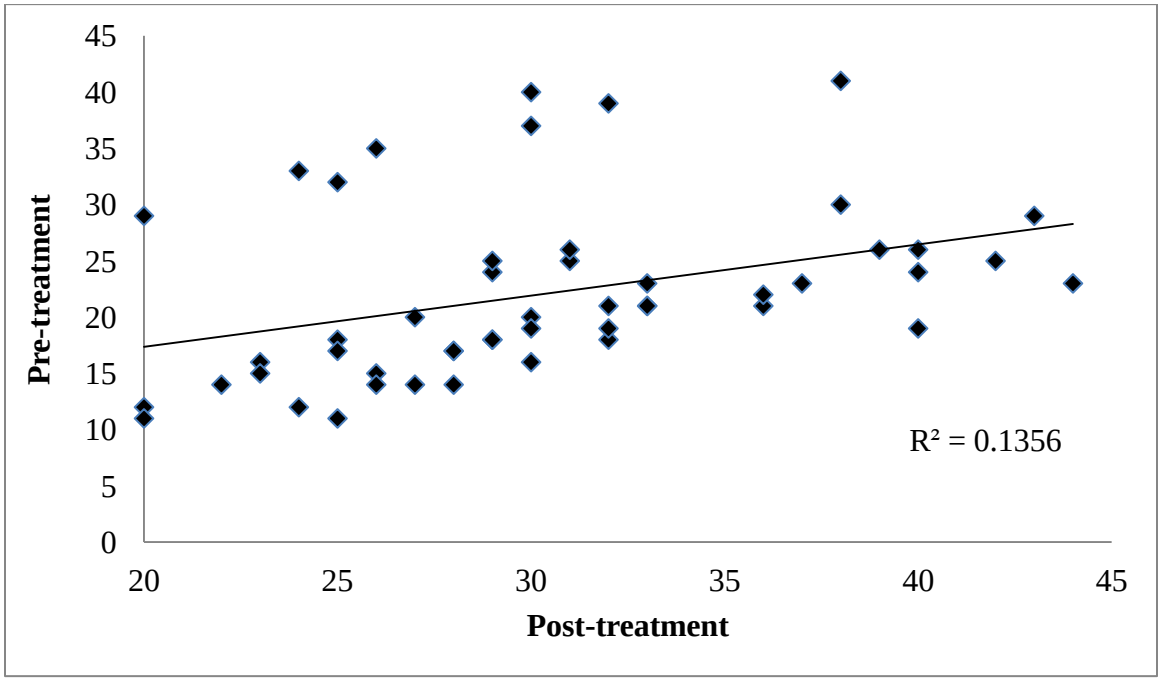


Fig. (4): Correlation coefficient of IOP (mmHg) between pre- and post-treatment

Table (3) showed that there was no statistical difference regarding IOP in group I (P>0.05) as shown in fig. (5), while there is a highly significant difference in group II (P <0.001), illustrated in fig. (6), and also the IOP showed statistically significant value (P <0.05) in all studied cases (Fig. 4).

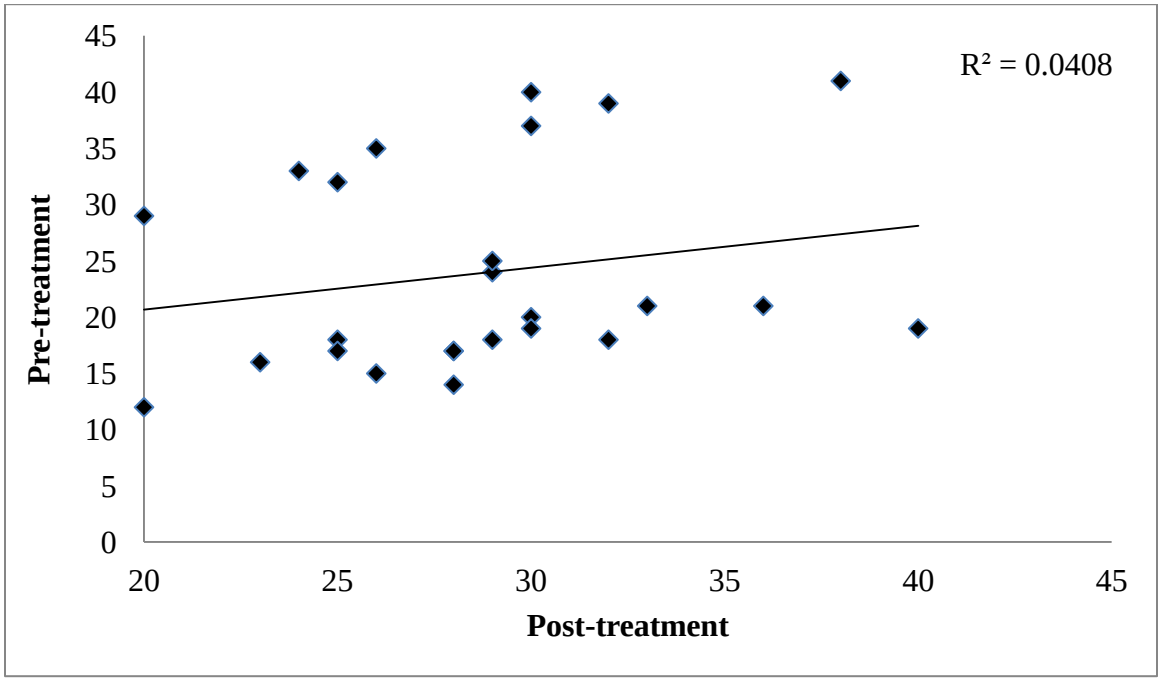


Fig. (5): Correlation coefficient of IOP between pre- and post-treatment in group I

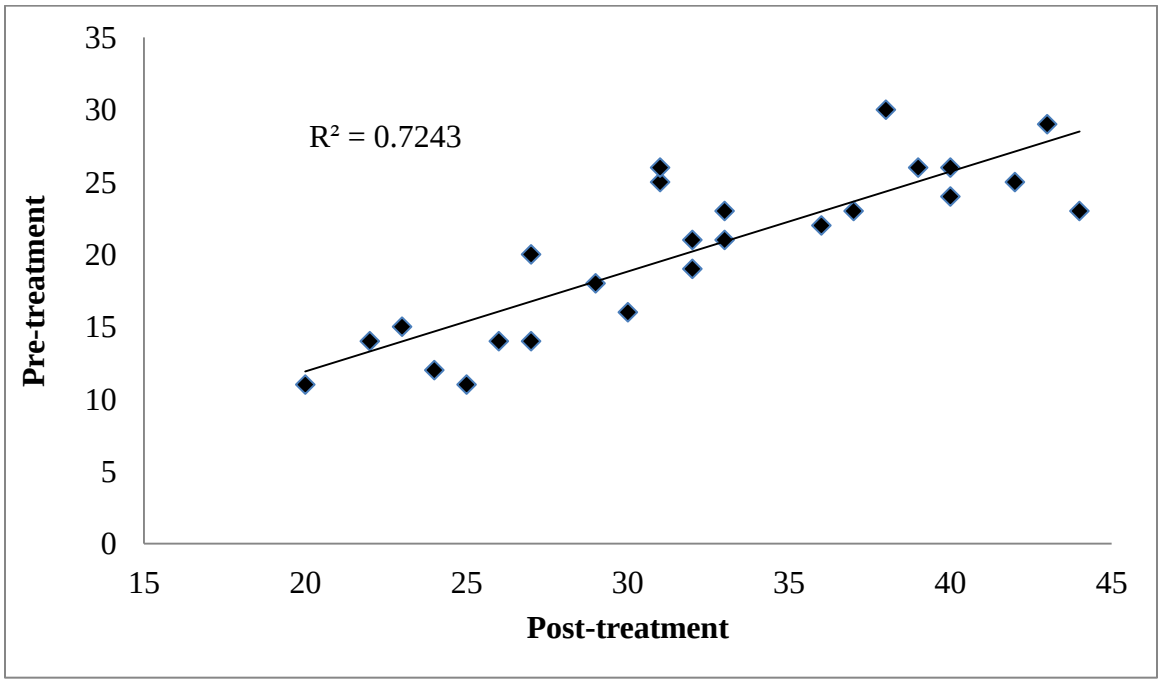


Fig. (6): Correlation coefficient of IOP between pre- and post-treatment in group II

Table (4): Pre- and post-treatment ANV in different studied groups

| ANV | Pre-treatment | Post-treatment | r | P value |
|---|---|---|---|---|
| Group I (Mean ±SD) | 2.80 ± 0.91 | 1.92 ± 1.32 | 0.12445 | >0.05 |
| Group II (Mean ±SD) | 2.92 ± 0.104 | 1.12 ± 0.93 | 0.78981 | <0.001 |
| Total (Mean ±SD) | 2.86 ± 0.97 | 1.52 ± 1.19 | 0.36241 | <0.05 |

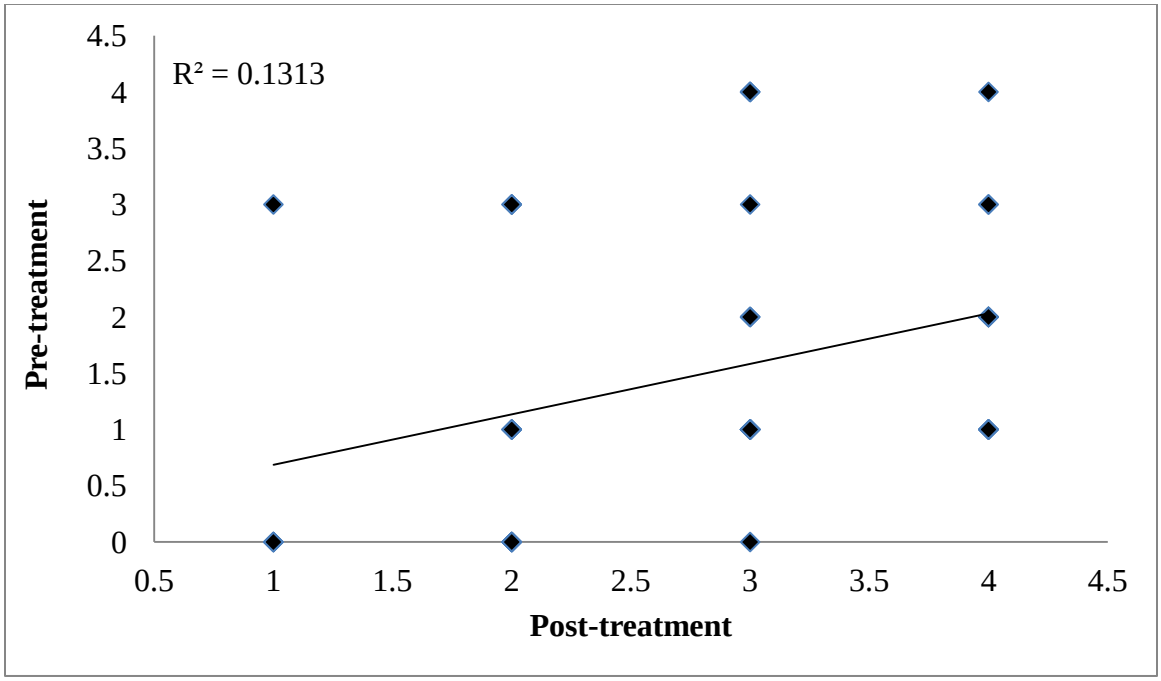


Fig. (7): Correlation coefficient between pre- and post-treatment as regard NVA

Table (4) showed that there was no statistical difference regarding angular neovascularization in group I (P>0.05) as shown in fig. (8), while there is a highly significant difference in group II regarding angular neovascularization (P <0.001), illustrated in fig. (9), and also the angular neovascularization showed statistically significant value (P <0.05) in all studied cases (Fig. 7).

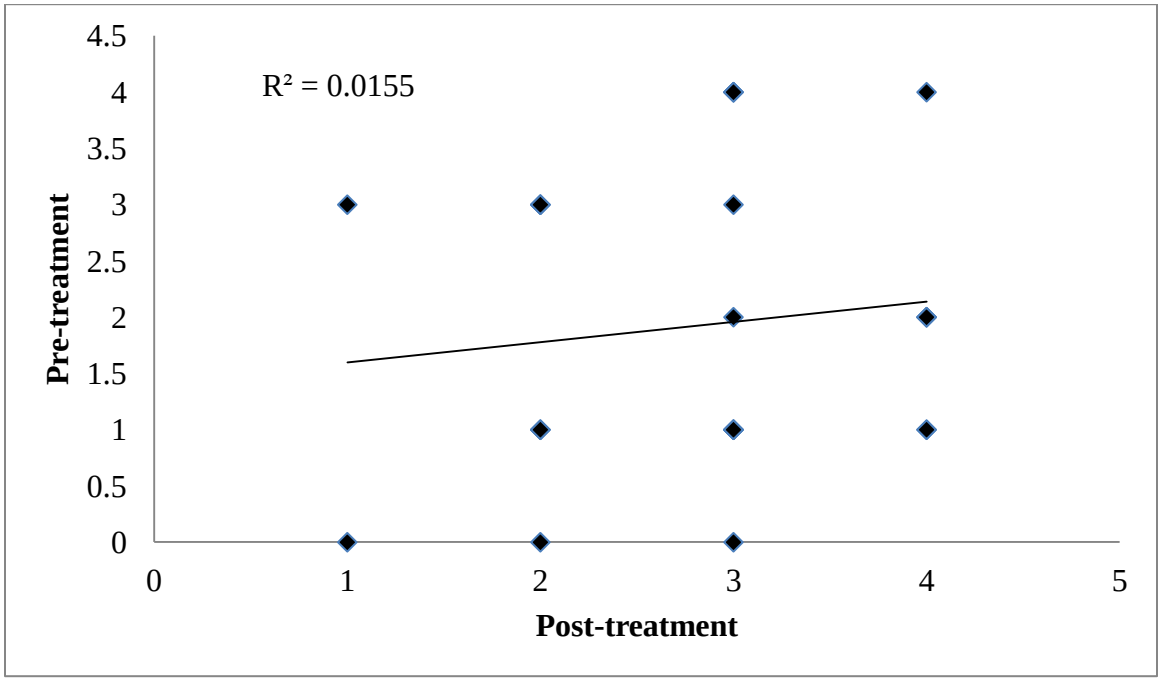


Fig. (8): Correlation coefficient of ANV in pre- and post-treatment of group I

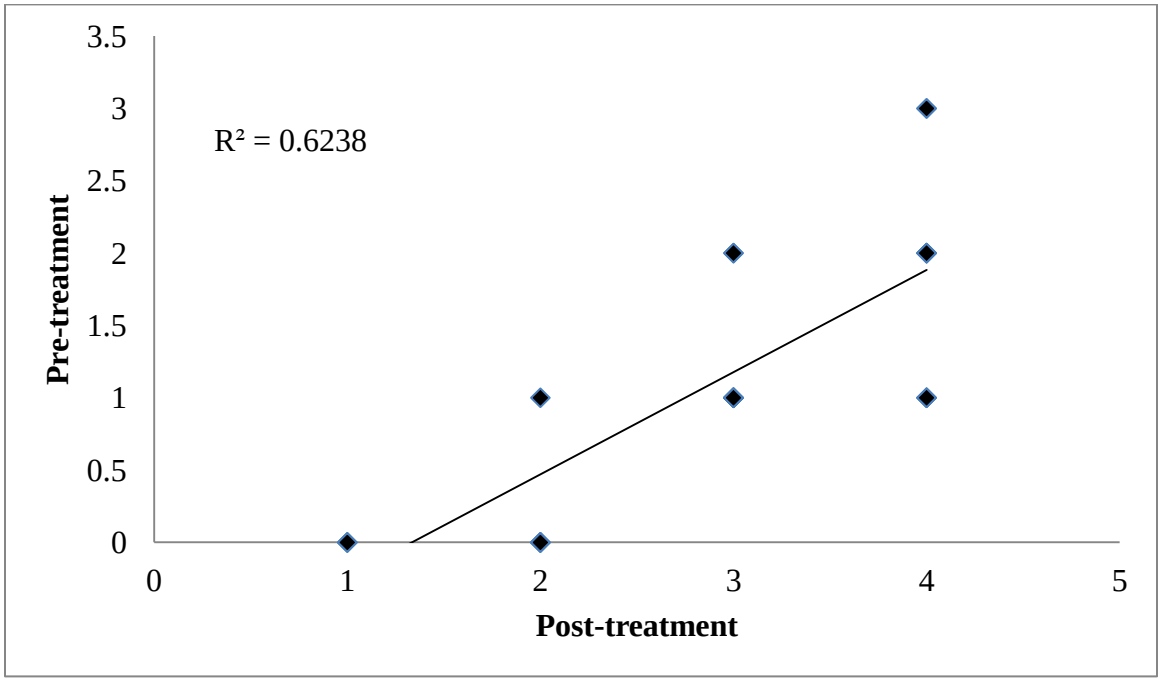


Fig. (9): Correlation coefficient of ANV in pre- and post-treatment of group II

## DISCUSSION

Neovascular glaucoma (NVG) is a serious sequelae of many ocular ischemic conditions, 97% of which are associated with retinal ischemia[12]. Retinal ischemia is the most common and important mechanism in most cases that result in the anterior segment changes causing neovascular glaucoma[13].

Ramesh et al. found that patients with NVG had significantly increased levels of VEGF in the aqueous humor, 40- and 113-fold higher than in patients with POAG or cataract, respectively, and implicated VEGF as an important factor in the pathogenesis of intraocular neovascularization[14].

Currently, there is no satisfactory treatment of neovascular glaucoma, thus the first goal should be to prevent its development by appropriate management of the causative disease. If neovascular glaucoma develops, early diagnosis together with aggressive control of IOP is crucial to minimize visual loss[15].

***Kahook and colleagues (2006)*** were the first who described the success of intravitreal bevacizumab in a patient with NVG and IOP uncontrolled, despite maximal medical therapy and cyclophotocoagulation[16].

The traditional treatment of neovascular glaucoma includes ocular antihypertensive medications, panretinal laser photocoagulation, glaucoma drainage surgeries, and cyclodestructive procedures[17].

However, neovascular glaucoma can be a refractory glaucoma that may not be controlled by any of these means[18].

This study was conducted for evaluation of ICB alone (2.5 mg) and with PRP for treatment of NVG in a three symptomatic parameters of NVG; visual acuity, intraocular pressure and angular neovascularization. We found that in intracameral injection of bevacizumab alone did not had a significant role in improving visual acuity (r= 0.13765, P >0.05) in the 25 cases of group I as the VA had stable or mild improvement in most of the cases (76%) and only 6 cases (24%) showed deteriorated vision. The mean baseline VA was 1.52 ± 0.38 and the mean VA after ICB was 1.16 ± 0.55.

The same results by ***Beutel et al., (2010)***, at baseline mean VA was (1.43 ± 0.89). At two months after the initial bevacizumab injection, mean VA was (1.41 ± 1.01), respectively. After 6 and 12 months, mean VA was (1.28 ± 0.9) and (1.5 ± 0.98), respectively. Visual acuity remained stable with a slight reduction over the entire follow-up period. No significant changes in VA were evident during 2 months (p = 0.963), 6 months (p = 0.628) and 12 months (p = 0.737).

Also, ***Wolf et al. (2011)*** had a mean duration of the treatment effect of 23 ± 4.4 days. Compared to mean VA remained stable or improved in 75% of all cases.

In this study, we found that a combination of ICB with PRP had a marked improvement of visual acuity after the follow-up period. The initial VA was 1.71 ± 0.34 improved to 0.87 ± 0.36. It showed a highly significant difference (r= 0.67314, P <0.001). So, this combination showed a successful modality for treating VA in patients with NVG.

This study found that in ICB group there was a mild reduction of IOP in cases of NVG. The mean IOP was 28.64 ± 4.91 at initial treatment reduced to 23.88 ± 9.03, i.e. still elevated above the normal values. The study found that there was no statistically significant difference (r = 0.20203, P >0.05) in this group (ICB alone).

***Wolf et al. (2011)*** compared the mean IOP before treatment (26.3 mm Hg), which decreases to 17.5 mm Hg at 1 week after treatment (p < 0.002) and to 17.1 mm Hg (p < 0.005) at 6 months following a single injection. At 6 months, additional treatment was performed in 87.5% (n = 21) of eyes. They concluded that IOP-lowering effect of intracameral bevacizumab can be seen 1 week after the injection, but is limited to a period of approximately 3 weeks. However, the fast and effective response to intracameral bevacizumab injection opens a time window for additional treatments, which are often necessary.

***Duch et al. (2009)*** found that ICB resulted in a marked regression of anterior segment neovascularization with IOP control without filtering surgery in 2 cases. No filtering surgery was needed to control IOP<18 mm Hg. In 18 cases, iris neovascularization extension had no prognostic value in terms of IOP control. After vascular regression following the administration of ICB, filtering surgery with drainage implants or trabeculectomy were performed when needed with no added difficulties owing to the underlying NVG. No macroscopic signs of corneal toxicity were detected, even when ICB injection had to be repeated. In this case, the time elapsed for the neovascular membrane to reappear at the anterior segment was 3 months.

***Lim et al. (2009)*** injected bevacizumab in the anterior chamber of 5 NVG subjects. Concerning intraocular pressure, there was no significant IOP lowering effect two weeks after injection.

In this study, we found that a combination of ICB with PRP had a marked improvement of IOP after three weeks follow-up period. The initial IOP was 32.16 ± 6.95 mmHg improved to 20.32 ± 5.65 mmHg. It showed a highly significant difference (r= 0.85106, P <0.001). So, this combination showed a successful modality for decreasing IOP in patients with NVG.

Regarding the angular neovascularization, the present study found that there is a mild regression of angular new vessels, there was no significant difference regarding the pre- and post-treatment of ICB alone in group I as the mean pretreatment grades of ANV was 2.80 ± 0.91 compared to 1.92 ± 1.32 post-treatment where the correlation coefficient r = 0.12445 (P>0.05).

***Lim et al. (2009)*** injected bevacizumab in the anterior chamber of 5 NVG subjects. Two weeks after injection, in all the eyes, the leakage from iris neovascularization was decreased and engorged vessels were regressed on an iris fluorescein angiogram. VEGF levels were remarkably lowered to 33.2 pg/mL after intracameral bevacizumab injection, at least a 30 fold decrease, much higher than that seen after intravitreal injection. Reduced neovascularization may lead to a decrease in release of inflammatory cytokines from the iris and retinal vessels of neovascular glaucoma patients, and reduce the occurrence of peripheral anterior synechia (PAS).

In a study of intracameral bevacizumab injection, 16 eyes of 15 patients with iris neovascularization associated with or without neovascular glaucoma secondary to proliferative retinal vasculopathies received intracameral bevacizumab (1.25 mg) and all patients had complete remission of the neovascularization within three weeks after the injection. Intraocular pressure was controlled with maximum medical therapy in eight of nine eyes reducing the need for glaucoma surgery ***(Chalam et al., 2008)***.

***Grisanti et al. (2006)*** gave intracameral injection of 1.0 mg bevacizumab in 6 eyes with iris NV and claimed a decrease in leakage from the iris vessels on angiography. They concluded that intraocular injection of bevacizumab may provide an additional strategy for the treatment of iris rubeosis in neovascular glaucoma.

***Yuzbasioglu et al. (2009)*** concluded that intracameral injection of bevacizumab can cause an immediate regression of neovascularization secondary to PDR or CRVO and could be useful adjuvant to prevent dense PAS formation that lead to persistent IOP increasing.

This study found that, in combination of both ICB and PRP, had a marked regression of angular new vessels, there was a very highly significant difference regarding the pre- and post-treatment of ICB alone in group II as the mean pretreatment grades of ANV was 2.92 ± 0.104 compared to 1.12 ± 0.93 post-treatment where the correlation coefficient (r) = 0.78981 (P<0.001).

Several studies propose the use of anti-VEGF agents with traditional treatments such as panretinal photocoagulation (PRP), with or without additional surgery and vary in the timing, combination, and place of injection (intracameral or intravitreal, or both simultaneously). The most frequent recommendation by various authors for treatment is the adjunct combination of intravitreal or intracameral bevacizumab with panretinal photocoagulation for the treatment of neovascular glaucoma (NVG) instead of PRP alone or as alternative treatment when visibility of the posterior segment is difficult due to opacities of the media (eg, hemorrhage). Although intravitreal, and intracameral, delivery of anti-VEGF agents is preferred for the management of NVG, several authors describe different protocols of treatment according to the stage of disease and the possible underlying cause as standardized guidelines of NVG treatment with anti-VEGFs have not yet been established ***(Ehlers et al., 2008)***.

The antiangiogenic effect of bevacizumab leads to fast reduction of the iris neovascularization with control of intraocular pressure without any surgery in grade 2 or 3 neovascular glaucoma. Panretinal photocoagulation was facilitated by improvement of corneal swelling. Diode laser cyclophotocoagulation was necessary in grade 4 ***(Douat et al., 2009)***.

ICB resulted in a rapid regression of the iris and angle neovascularization, which permitted to halt the progression of PAS process. ***Duch et al. (2009)*** in their pilot study showed that intracameral injection of bevacizumab may be a helpful adjunct for the surgical treatment of NVG.

The role of antivascular endothelial growth factor (anti-VEGF) agents in treating various ophthalmic diseases is currently being investigated. There have been many advances in the understanding of how anti-VEGF agents work and speculation on when to implement them clinically for neovascular glaucoma. Recent studies exploring the utility of anti-VEGF agents for wound modulation after trabeculectomy reveal promising results[28].

Intraocular injection of bevacizumab seems to be a safe and effective adjunctive treatment for NVG. The short half-life of bevacizumab leads to a transient resolution of neovascularization. Most patients will require pan-retinal photocoagulation or peripheral retinal cryotherapy to permanently decrease the secretion of VEGF; however, bevacizumab allows for a quick decrease neovascularization of the iris and neovascularization of the angle and quickly improves symptoms in those with elevated IOP and partially open drainage angles. This technique can potentially decrease neovascularization and improve results of more invasive surgeries such as glaucoma drainage devices and trabeculectomies with mitomycin C, and/or cyclophotocoagulation to achieve proper IOP control[29].

## CONCLUSION

Intracameral Bevacizumab seemed to have an effective against anterior segment neovascular activity, and controlled IOP in patients with early-stage NVG without angle closure, while in advanced NVG we recommended ICB to be an adjuvant therapy with traditional treatment with PRP.

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**مقدمة**

يعتبر مرض المياه الزرقاء المضاعفة للأوعية الدموية النامية بزاوية الخزانة الأمامية من أخطر الأمراض التى تصيب العين. و ينتج هذا المرض كنتيجة للأمراض المؤدية لنقص الأكسجين المتاح للشبكية مثل إنسداد الوريد الشبكى أو الإعتلال الشبكي السكري.

أثبت إستخدام عقار البيفاسيزوماب في مرض المياه الزرقاء المضاعفة للأوعية الدموية النامية بزاوية الخزانة الأمامية وتحلل الماقوله السني ومرض اعتلال الشبكية السكري نتائج أولية طيبة بالإضافة الى مضاعفات وأعراض جانبية قليلة على المدى القصير.

**الهدف من العمل**:

تقييم حقن البيفاسيزوماب فى الخزانه الأمامية للعين مع و بدون عمل كى ضوئى للشبكية لعلاج حالات المياه الزرقاء المضاعفة للأوعية الدموية النامية بزاوية الخزانة الأمامية.

**المرضى والطرق**:

بمباشرة مرضى المياه الزرقاء الثانوية المترددين على العيادة الخارجية بقسم طب وجراحة العين وفحصهم فحصا إكلينيكيا كاملا وبواسطة الموجات فوق الصوتية وتصوير قاع العين بصبغة الفلروسين سيتم اختيار المرضى كما يلى:

- مرضى المياه الزرقاء الثانوية.
- حدة الإبصار هى رؤية حركة اليد او اكثر.
- المريض موافق وقادر بدنيا على إجراء الحقن بمادة البيفاسيزوماب و عمل كى ضوئى للشبكية.

يضم البحث 50 مريضا مقسمين الى مجموعتين تضم كلا منها خمسة و عشرون مريضا تقسم كالتالى:

**المجموعة الأولى:** ستجري حقن البيفاسيزوماب فقط فى الخزانة الأمامية.

**المجموعة الثانية:** ستجري حقن البيفاسيزوماب فى الخزانة الأمامية و عمل كى ضوئى للشبكية.

و تمت متابعة المرضى فى اليوم الأول ثم بعد أسبوع ثم بعد شهر ثم بعد ثلاثة أشهر ثم ستة أشهر ثم بعد عام كامل.

**النتائج**:

حقن البيفاسيزوماب فى الخزانة الأمامية و عمل كى ضوئى للشبكية يعطى نتائج أفضل و أكثر اسقرارا من ستجري حقن البيفاسيزوماب فى الخزانة الأمامية فقط.