

Comparison of Intraocular Pressure Response Between Intravitreal Dexamethasone Implant and Triamcinolone Acetonide Suspension in Patients with Retinal Diseases

Scroggins Casey¹, Sciulli Harrison², Miller Alexander³, Wuller Dashiell⁴, Yarosh Sofia², Natarajan Varun², Heath Ronain², Wuller Allison⁵, James Michelle² and Miller David G²

¹University of Toledo College of Medicine and Life Sciences, Toledo, OH, USA

²Retina Associates of Cleveland Inc, Beachwood, OH, USA

³University of Missouri, Columbia, MO, USA

⁴Saint Louis University School of Medicine, St. Louis, MO, USA

⁵Rush Medical School, Chicago, IL, USA

Submission: July 14, 2025; **Published:** July 25, 2025

***Corresponding author:** David G Miller, Retina Associates of Cleveland, Inc, 24075 Commerce Park Beachwood, Ohio 44122, USA

Abstract

Purpose: To compare the incidence and severity of intraocular pressure (IOP) elevation following intravitreal dexamethasone implant versus preservative-free triamcinolone acetonide suspension in patients with retinal diseases.

Methods: A retrospective observational cohort study was conducted at a large retina practice. Patients who received at least two injections of either dexamethasone implant or triamcinolone acetonide between 2011 and 2023 were included. IOP was measured at baseline and follow-up using Goldmann or Tonopen tonometry. The primary outcome was a change in IOP from baseline. Secondary outcomes included IOP increases ≥ 5 or ≥ 10 mmHg, IOP > 21 mmHg, and need for IOP-lowering therapy.

Results: A total of 165 patients received dexamethasone implants, and 146 patients received triamcinolone acetonide injections for analysis. In the dexamethasone group, the mean IOP rose from 14.8 to 19.4 mmHg ($p = 0.0001$), with 31.2% of eyes showing ≥ 5 mmHg increase, 7.9% of eyes showing ≥ 10 mmHg increases and 23.6% exceeding 21 mmHg. IOP-lowering medications averaged 0.48 in treated eyes and 0.26 in fellow eyes ($p = 0.001$). In the triamcinolone group, mean IOP changed from 15.3 to 14.5 mmHg ($p = 0.71$), with 19.1% of eyes showing ≥ 5 mmHg increase, 2.8% of eyes showing ≥ 10 mmHg increases and 11.8% exceeding 21 mmHg. IOP-lowering medications averaged 0.21 in treated eyes and 0.12 in fellow eyes ($p = 0.03$).

Conclusion: Intravitreal dexamethasone implants demonstrated a significantly higher risk of intraocular pressure elevation compared to those receiving preservative-free triamcinolone acetonide. These findings highlight the need for individualized treatment selection and careful post-injection monitoring

Keywords: Intravitreal Corticosteroids; Intraocular Pressure; Dexamethasone Implant; Triamcinolone Acetonide; Steroid Response

Abbreviations: IOP: Intraocular Pressure; MYOC: Myocilin Gene; OCT: Optical Coherence Tomography

Introduction

Intravitreal corticosteroids are frequently used to treat a wide range of posterior segment diseases, including diabetic macular edema, uveitis, and retinal vein occlusions. Among these, dexamethasone implants and preservative-free triamcinolone acetonide are two commonly employed intravitreal steroids. While these

agents provide potent anti-inflammatory effects and help stabilize the blood-retina barrier, one of their well-documented adverse effects is the elevation of intraocular pressure (IOP). The elevation in IOP following intravitreal steroid administration is believed to occur through multiple mechanisms.

Corticosteroids are known to induce structural and functional changes in the trabecular meshwork, the primary outflow pathway for aqueous humor. These changes include accumulation of extracellular matrix materials, such as glycosaminoglycans, and suppression of the phagocytic activity of trabecular meshwork cells, which together increase resistance to aqueous humor outflow [1]. Additionally, upregulation of glucocorticoid receptors and alterations in cytoskeletal organization within trabecular meshwork cells have been implicated in impaired outflow facility [2].

Steroid responsiveness also varies between individuals. Some have hypothesized that genetic predispositions, such as expression of the myocilin (MYOC) gene, may contribute to differential susceptibility to steroid-induced ocular hypertension. However, current literature does not demonstrate a consistent association between MYOC mutations and steroid responsiveness [3,4]. In clinical studies evaluating the ocular hypertensive effects of intravitreal dexamethasone implants in retinal vascular disease, most patients experience only modest elevations in IOP. One investigation reported that 79% of treated eyes demonstrated an IOP increase of 5mmHg or less, while approximately 20% required initiation of IOP-lowering therapy following injection [5].

Another study found that only 12% of eyes exhibited post-treatment IOP values exceeding 25mmHg [6]. Notably, current evidence does not support a cumulative IOP-elevating effect with repeated dexamethasone implant administrations. The incidence of IOP elevation following intravitreal triamcinolone acetonide administration has been consistently reported across multiple studies. For example, Vasconcelos-Santos et al. [7] observed that 32% of patients treated with 4mg intravitreal triamcinolone acetonide for various posterior segment disorders developed an IOP of ≥ 21 mmHg over an average follow-up of 7.7 months [7].

Similarly, Smithen and colleagues reported a 40.4% incidence of IOP ≥ 24 mmHg with a mean follow-up of 9.3 months in a retrospective observational case series [8]. In contrast, Roth et al. [9] found a slightly lower incidence of 28.2% for IOP > 25 mmHg over a 24-month period in a larger retrospective cohort [9]. This study seeks to characterize and compare the incidence and severity of intraocular pressure elevation following the use of intravitreal dexamethasone implants and preservative-free triamcinolone acetonide injections in a large retina practice.

Materials and Methods

This study employed a retrospective, observational cohort design to evaluate IOP changes following intravitreal corticosteroid administration. The two corticosteroid agents compared were dexamethasone intravitreal implant and preservative-free triamcinolone acetonide. The study was conducted within a singular large retina practice of 15 retinal specialists and was approved by the Institutional Review Board.

Electronic health records were reviewed to identify patients who had received dexamethasone implant or triamcinolone ace-

tonide injections between 2011 and 2023.

Inclusion criteria consisted of patients who received a series of at least two intravitreal corticosteroids for the treatment of posterior segment pathology, including diabetic macular edema, central or branch retinal vein occlusion with macular edema, pseudophakic cystoid macular edema, macular or retinal edema, uveitis, diabetic retinopathy, and neovascular macular degeneration. Exclusion criteria included concurrent use of multiple intravitreal steroid formulations, incomplete IOP data, or follow-up consisting of less than 2 injections.

Patients were categorized into two primary cohorts based on the intravitreal steroid administered:

- Dexamethasone implant group
- Triamcinolone acetonide injection group

IOP was measured using Goldmann applanation and Ton open tonometry at baseline (pre-injection) and at follow-up visits post-injection. The primary outcome measure was the change in IOP from baseline to the final visit in the treatment period. Secondary outcome measures included:

- Incidence of IOP elevation ≥ 5 mmHg from baseline
- Incidence of IOP elevation ≥ 10 mmHg from baseline
- IOP > 21 mmHg at any follow-up point
- Need for initiation of IOP-lowering therapy (topical medications)

To investigate differences between steroid types, the magnitude and incidence of IOP elevation were compared between the dexamethasone and triamcinolone cohorts. Statistical significance was assessed using Student's T-tests for continuous variables.

Results

A total of 165 patients who received intravitreal dexamethasone implants and 146 patients who received preservative-free triamcinolone acetonide were included in the study. The mean age of patients receiving dexamethasone implant was 72 years, while the mean age in the triamcinolone group was 69.8 years. Each cohort was further stratified by diagnosis, number of injections, and IOP outcomes. Among the dexamethasone implant group, the most common indications were diabetic macular edema (41.2%), central retinal vein occlusion with macular edema (24.8%), and branch retinal vein occlusion with macular edema (17.0%).

Less frequent indications included pseudophakic cystoid macular edema (8.5%), uveitis (3.0%), and others comprising less than 2% of the population. See Figure 1 for a detailed representation. In the triamcinolone cohort, diabetic macular edema also represented the leading indication (30.1%), followed by pseudophakic cystoid macular edema (27.4%) and macular or retinal edema (15.8%). Other diagnoses included retinal vein occlusions, uveitis, epiretinal membrane with edema, serous choroidal de-

tachment, and neovascular glaucoma, each comprising a smaller portion of the cohort. See Figure 2 for a detailed representation.

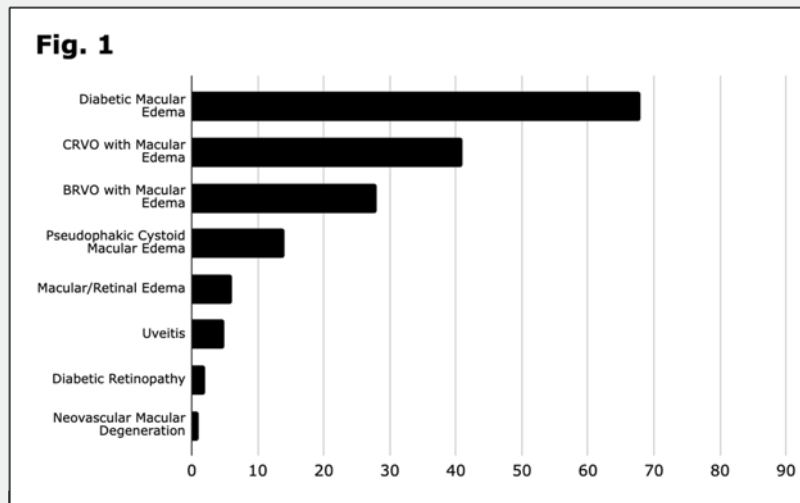


Figure 1: Number per diagnosis for patients in the dexamethasone study group. Diabetic macular edema (68), central retinal vein occlusion with macular edema (41), branch retinal vein occlusion with macular edema (28), pseudophakic cystoid macular edema (14), macular/retinal edema (6), uveitis (5), diabetic retinopathy (2), neovascular macular degeneration (1).

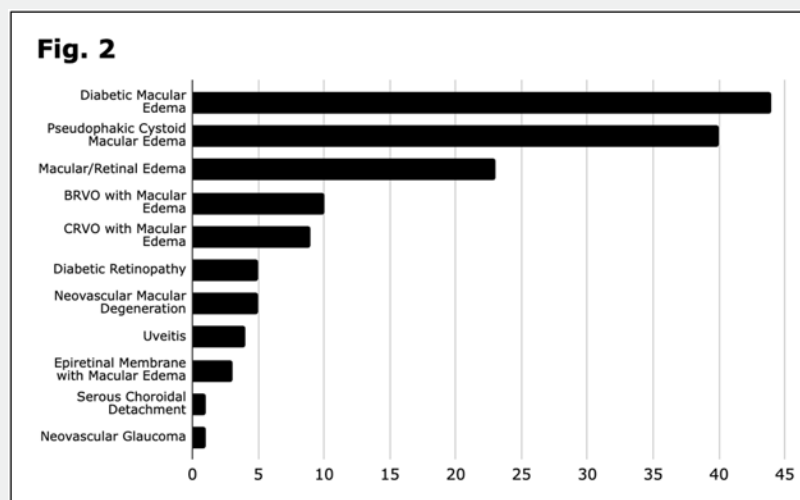


Figure 2: Number per diagnosis for patients in the triamcinolone acetonide study group. Diabetic macular edema (44), pseudophakic cystoid macular edema (40), macular/retinal edema (23), branch retinal vein occlusion with macular edema (10), central retinal vein occlusion with macular edema (9), diabetic retinopathy (5), neovascular macular degeneration (5), uveitis (4), epiretinal membrane with macular edema (3), serous choroidal detachment (1), neovascular glaucoma (1).

Dexamethasone Implant

In the first arm of analysis, 165 patients receiving dexamethasone were evaluated for pressure elevation over time. Baseline IOP averaged 14.8 mmHg in treated eyes, increasing to 19.4 mmHg at final follow-up ($p = 0.0001$). Figure 3 illustrates this increase. Notably, 31.2% of patients experienced an IOP increase in the treated eye ≥ 5 mmHg, and 7.9% had increases ≥ 10 mmHg. Of those with baseline IOP ≤ 21 mmHg, 23.6% exceeded the 21 mmHg threshold during follow-up. The average number of IOP-lowering medications at the baseline visit was 0.25 in treated eyes and 0.21 in fellow eyes ($p = 0.49$). At the final follow-up, IOP-lowering med-

ications averaged 0.48 in treated eyes and 0.26 in fellow eyes ($p = 0.001$). See Figure 4 for graphical comparison.

Triamcinolone Acetonide

Among 146 patients receiving preservative-free triamcinolone, baseline IOP in treated eyes averaged 15.3 mmHg and decreased slightly to 14.5 mmHg at final visit ($p = 0.71$). Figure 5 illustrates this. A total of 19.1% of patients experienced an IOP increase in the treated eye ≥ 5 mmHg, and 2.8% had increases ≥ 10 mmHg. Of those with baseline pressures ≤ 21 mmHg, 11.8% exceeded this threshold post-treatment. The average number of

IOP-lowering medications at the baseline visit was 0.26 in treated eyes and 0.21 in fellow eyes ($p = 0.3$). At the final follow-up,

IOP-lowering medications averaged 0.21 in treated eyes and 0.12 in fellow eyes ($p = 0.03$). See Figure 6 for graphical comparison.

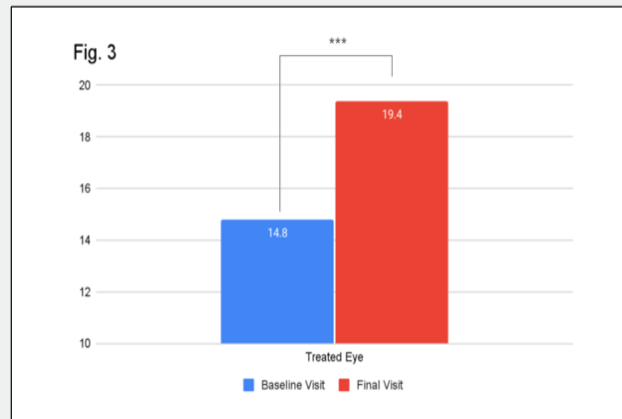


Figure 3: Mean IOP at baseline and final visit in the dexamethasone study group. Treated eye baseline IOP, 14.8 mmHg, compared to 19.4 mmHg at the final visit ($p=0.0001$).

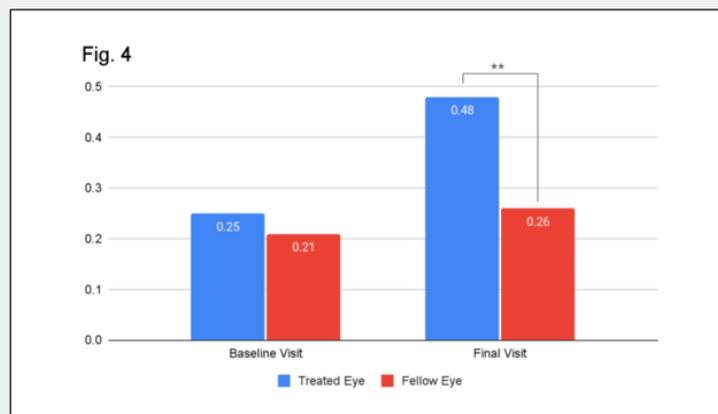


Figure 4: Mean number of glaucoma medications at baseline and final visit in the dexamethasone study group. Mean baseline number of medications in treated eyes, 0.25, compared to 0.21 in fellow eyes ($p=0.49$). Mean number of medications at the final visit in treated eyes, 0.48, compared to 0.26 in fellow eyes ($p=0.001$).

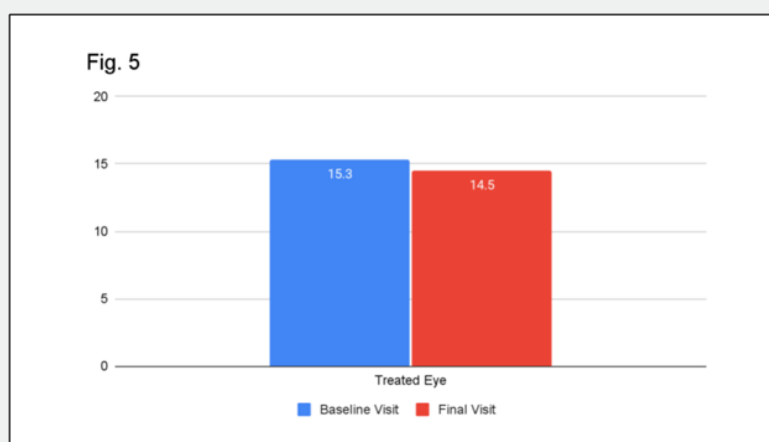


Figure 5: Mean IOP at baseline and final visit in the triamcinolone acetonide study group. Treated eye baseline IOP, 15.3 mmHg, compared to 14.5 mmHg at the final visit ($p=0.71$).

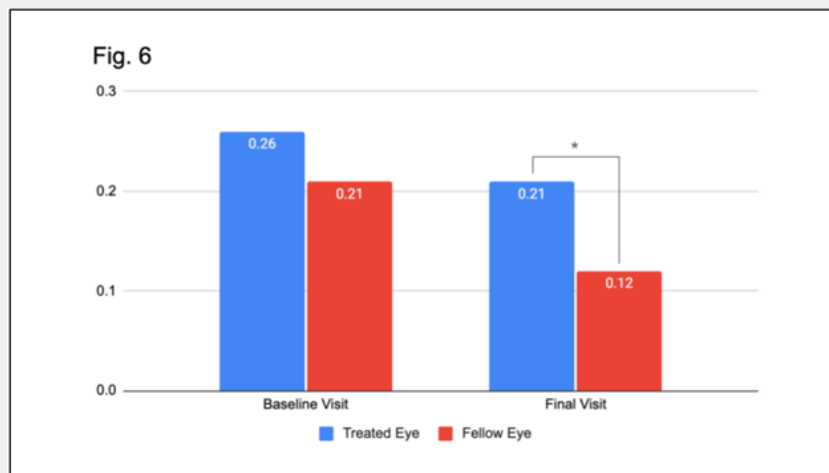


Figure 6: Mean number of glaucoma medications at baseline and final visit in the triamcinolone acetonide study group. Mean baseline number of medications in treated eyes, 0.26, compared to 0.21 in fellow eyes ($p=0.3$). Mean number of medications at the final visit in treated eyes, 0.21, compared to 0.12 in fellow eyes ($p=0.03$).

Discussion

Across several retrospective analyses, we observed a higher incidence and magnitude of IOP elevation in eyes treated with dexamethasone implants than in those treated with triamcinolone acetonide. These findings contribute to the growing body of literature examining the ocular hypertensive effects of intravitreal steroids, particularly within real-world clinical populations. Steroid-induced ocular hypertension is a well-characterized phenomenon, believed to result from impaired aqueous outflow through the trabecular meshwork. This impairment is mediated by increased deposition of extracellular matrix proteins, suppression of phagocytic activity, and cytoskeletal changes within trabecular meshwork cells, all of which increase outflow resistance.

Genetic predisposition, particularly involving the MYOC gene, may further sensitize individuals to pressure elevations following corticosteroid exposure [1]. Dexamethasone, owing to its long-acting biodegradable implant formulation and higher relative glucocorticoid potency, may exert more prolonged effects on aqueous humor dynamics compared to preservative-free triamcinolone acetonide, which is shorter acting and delivered as a solution or suspension [2]. In this retrospective analysis, dexamethasone-treated eyes exhibited clinically meaningful IOP elevations in a substantial subset of patients. Notably, up to 31.2% of patients demonstrated increases of ≥ 5 mmHg and 23.6% surpassed the 21mmHg threshold.

In contrast, triamcinolone acetonide injections resulted in smaller rates of IOP elevation: 19.1% of patients had ≥ 5 mmHg increases, and only 11.8% exceeded 21mmHg. In the subgroup of patients receiving multiple treatments, mean IOP values for dexamethasone-treated eyes rose significantly, while triamcinolone-treated eyes did not demonstrate significant mean changes, further supporting the relative pressure-sparing profile of triamcinolone acetonide. Clinical implications of these findings are

multifold. First, when considering intravitreal steroid therapy for patients at risk of steroid response-such as those with pre-existing glaucoma or ocular hypertension-triamcinolone acetonide may represent a safer alternative.

Furthermore, our results underscore the importance of close IOP monitoring following corticosteroid injection, particularly with dexamethasone implants. The increased use of IOP-lowering medications observed in dexamethasone-treated eyes at follow-up suggests that clinicians were responsive to pressure elevations, potentially mitigating more severe outcomes such as optic nerve damage or irreversible visual field loss. Additionally, differences in corticosteroid selection by diagnosis warrant consideration. Diabetic macular edema was the most common indication for both drugs, but the relative prevalence of central and branch retinal vein occlusions was higher among dexamethasone-treated patients.

These patterns may reflect clinician preference, drug efficacy profiles, or reimbursement dynamics, but they also open the door for diagnosis-specific risk-benefit analyses regarding IOP outcomes. Several limitations must be acknowledged. The retrospective nature of the analysis introduces potential biases, including selection bias and incomplete documentation of glaucoma medication use. It is also possible that some patients were prophylactically treated with IOP-lowering agents in anticipation of steroid response, particularly in the triamcinolone cohort, which could partially obscure the true hypertensive effect.

Moreover, the follow-up period for some patients may have been insufficient to capture delayed-onset steroid responses, a known phenomenon with intraocular corticosteroids [10]. Future studies should aim to validate these findings in a prospective, controlled setting. Investigating the longitudinal impact of repeated steroid injections on IOP, stratified by known steroid responders, could yield important predictive insights. Likewise, correlation

with visual acuity outcomes, optical coherence tomography (OCT) changes, and quality-of-life metrics would help to better define the overall risk-benefit balance for each corticosteroid option.

Conclusion

In this retrospective study, patients treated with the dexamethasone intravitreal implant demonstrated a significantly higher risk and magnitude of intraocular pressure elevation compared to those receiving preservative-free triamcinolone acetonide. While both agents are effective corticosteroids used in the management of retinal diseases, our findings highlight the need for individualized treatment selection and careful post-injection monitoring, particularly in patients with preexisting glaucoma or ocular hypertension. These results support the growing body of evidence advocating for routine IOP surveillance following intravitreal steroid administration and may guide clinicians in balancing therapeutic efficacy with safety when choosing among steroid options.

Conflict of Interest

The authors declare there are no conflicts of interest related to this study and received no financial support for the research, authorship or publication of this article.

References

1. Patel G, Fury W, Yang H, Gomez-Caraballo M, Bai Y, et al. (2020) Molecular taxonomy of human ocular outflow tissues defined by single-cell transcriptomics. *Proceedings of the National Academy of Sciences of the United States of America* 117(23): 12856-12867.
2. AF Clark, K Wilson, MD McCartney, ST Miggans, M Kunkle, et al. (1994) Glucocorticoid-induced formation of cross-linked actin networks in cultured human trabecular meshwork cells. *Investigative Ophthalmology & Visual Science* 35(1): 281-294.
3. JH Fingert 1, E Héon, JM Liebmann, T Yamamoto, JE Craig, et al. (1999) Analysis of myocilin mutations in 1703 glaucoma patients from five different populations. *Human Molecular Genetics* 8(5): 899-905.
4. Park BC, Tibudan M, Samaraweera M, Shen X, Yue BY (2007) Interaction between two glaucoma genes, optineurin and myocilin. *Genes Cells* 12(8): 969-979.
5. Jirattanasopa P, Jiranoppasakdawong S, Ratanasukon M, Bhurayanontachai P, Dangboon W (2022) Results of intravitreal dexamethasone implant (Ozurdex®) for retinal vascular diseases with macular edema: An observational study of real-life situations. *Medicine* 101(27): e29807.
6. Singer MA, Dugel PU, Fine HF, Capone A, Maltman J (2018) Real-world assessment of dexamethasone intravitreal implant in DME: Findings of the prospective, multicenter REINFORCE study. *Ophthalmic Surgery, Lasers and Imaging Retina* 49(6): 425-435.
7. Vasconcelos-Santos DV, Nehemy PG, Schachat AP, Nehemy MB (2008) Secondary ocular hypertension after intravitreal injection of 4 mg of triamcinolone acetonide: incidence and risk factors. *Retina* 28(4): 573-580.
8. Smithen LM, Ober MD, Maranan L, Spaide RF (2004) Intravitreal triamcinolone acetonide and intraocular pressure. *Am J Ophthalmol* 138(5): 740-743.
9. Roth DB, Verma V, Realini T, Prenner JL, Feuer WJ, et al. (2009) Long-term incidence and timing of intraocular hypertension after intravitreal triamcinolone acetonide injection. *Ophthalmology* 116(3): 455-460.
10. Kocabora MS, Yilmazli C, Taskapili M, Gulkilik G, Durmaz S (2008) Development of ocular hypertension and persistent glaucoma after intravitreal injection of triamcinolone. *Clinical ophthalmology (Auckland, N.Z.)* 2(1): 167-171.



This work is licensed under Creative Commons Attribution 4.0 License
DOI: [10.19080/JOJ.2025.13.555859](https://doi.org/10.19080/JOJ.2025.13.555859)

Your next submission with Juniper Publishers will reach you the below assets

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats (**Pdf, E-pub, Full Text, Audio**)
- Unceasing customer service

Track the below URL for one-step submission

<https://juniperpublishers.com/online-submission.php>