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ORIGINAL ARTICLE

Efficacy of posterior subtenon triamcinolone in the treatment of diabetic macular edema in patients with full panretinal photocoagulation

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Abstract

Purpose: To investigate the efficacy of posterior subtenon triamcinolone (PSTA) injection for diabetic macular edema (DME) in eyes with full panretinal photocoagulation (PRP).

Methods: Study included 44 eyes of 37 patients with center involving DME with full PRP. Best corrected visual acuity (BCVA), central retinal thickness (CRT) on spectral domain OCT were measured at 1, 3 and 6 months.

Results: Mean follow-up was 15,1 months. Significant improvement in visual acuity from 0.86 ± 0.36 to 0.73 ± 0.31 LogMAR (-0.13) was observed 1 month after the PSTA injection ($p < 0.001$). Vision improved in 29 (65.9%), not changed in 13 (29.6%) and decreased in 2 eyes (4.5%) at month 1. Statistically significant CRT decrease from $509.1 \pm 177 \mu\text{m}$ to $337.8 \pm 143 \mu\text{m}$ was observed at month 1 ($p = 0.000$). A minor reincrease in CRT was observed at month 3. However, there was still a statistically significant improvement in BCVA and CRT at month 3 compare to baseline ($p = 0.000$). Mean letter gain was 9.8 (5-30) at month 3. Reinjection was performed when edema recurred. Reinjection interval was 4.6 ± 1.2 months. Mean reinjection number was 2.8 ± 1.8 . Only one eye did not respond response to treatment. Intraocular pressure elevated in 15.9% of the eyes. No serious complications were observed.

Conclusion: Posterior subtenon triamcinolone is a safe and effective treatment method for center involving DME in eyes with full PRP.

Keywords: Diabetic macula edema; panretinal photocoagulation; posterior subtenon triamcinolone.

Diabetic macular edema (DME) is still a leading cause of blindness worldwide.^[1] Microangiopathy, neuroretinopathy, and chronic low-grade inflammation play a role in its pathogenesis.^[1,2] Vascular endothelial growth factor (VEGF) release from ocular tissues causes vascular endothelial proliferation and retinal capillary obliteration. Inflammation and VEGF release cause retinal vasodilatation, vessel tortuosity, and permeability increase

through breakdown of blood–retina barrier and eventual fluid accumulation in retinal layers.^[3,4] Today, intravitreal anti-VEGF injections are considered as the first-line treatment in DME. Intravitreal steroid implants are generally used as second line of therapy or adjuvant therapy.^[5] They are preferred as initial therapy in certain indications. However, they carry the inherent risk of endophthalmitis, and high cost is another concern. Corticosteroids reduce



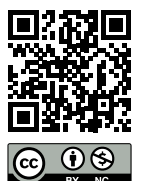
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VEGF expression, decreasing leukocyte recruitment and production of inflammatory cytokines.^[1] They restore blood–retina barrier and decrease vasodilatation.^[4]

Triamcinolone acetonide (TA) is a minimally soluble repository steroid that can slowly release its content. Edelman et al.^[3] showed that a single intravitreal TA completely blocked VEGF-induced retinal leakage and inner retinal edema for 45 days in the rabbit model. Thomas reported TA concentrations at therapeutic level in the vitreous after 1–29 days after posterior subtenon TA injection (PSTA) comparable to intravitreal injection.^[6] Clinical studies have shown PSTA has comparable effectiveness to intravitreal triamcinolone acetonide (IVTA) in diffuse DME with a lower risk of intraocular pressure (IOP) elevation.^[7,8] In addition, its simultaneous use increases the effectiveness of anti-VEGF.^[5,9] In proliferative diabetic retinopathy; panretinal laser treatment is generally completed in three sessions. Ischemic retinal areas are destroyed, and VEGF release from these areas is significantly reduced.^[10] Despite low VEGF levels, DME can persist in these eyes due to ongoing low-grade inflammation and disruption of the blood-retina barrier. PSTA targets that inflammation and leakage. This study was conducted to investigate the effect of PSTA treatment on DME in eyes with full panretinal photocoagulation (PRP).

Materials and Methods

This interventional clinical study included 44 eyes of 37 patients, with complete PRP and center-involved DME, who had PSTA injection. Seven patients had PSTA injection in both eyes. The study was carried out between March 2022 and February 2023 in our Retina facility at the University Hospital, Ophthalmology Department.

The study was conducted under the tenets of the Helsinki Declaration. The ethical approval was obtained from the institutional Ethical Board (Date: April, 04th 2022. Reference Number: 2022/63). Informed consent was obtained from all participants included in the study.

All the eyes had at least 1,500–2,000 shots with a 532 nm argon laser. Inclusion criteria were as follows; eyes with center-involved, diffuse DME with central retinal thickness (CRT) ≥ 250 μm , minimum follow-up of 6 months after injection, regressed proliferative diabetic retinopathy with no signs of neovascularization, PRP completed at least 6 months before the study entrance, no previous treatment for DME within 3 months; including focal laser treatment, intravitreal steroid or anti-VEGF injection. Exclusion criteria were; patients under 18 years of age, visual acuity of

counting fingers <1 m, any media opacity, recent ocular surgery, eyes with significant optic atrophy, exudate plug in fovea with atrophy, severe disorganisation of outer retinal layers, macular scar, epiretinal membrane or vitreomacular traction, uveitis, uncontrolled glaucoma, and patients lost to follow-up. In eyes with significant cataract, PSTA injection was not performed. However, injection was performed in eyes with few cortical opacities away from the optical axis. Best-corrected visual acuity (BCVA) was measured by a resident with a geometric chart at 4 m at study entry and each control after the injection. Values were converted into logarithm of the minimum angle of resolution (LogMAR) for statistical analysis. IOP measurements were measured with a Goldmann applanation tonometer. Dilated biomicroscopic fundus examination was performed with a 90D lens. CRT was measured with spectral domain-optical coherence tomography (SD-OCT), Heidelberg Engineering, Heidelberg, Germany). Eyes with macular ischemia or any leakage from neovascularization on fluorescein angiography were excluded from the study. The main outcome measures were visual improvement and CRT.

PSTA injections were performed in the minor procedure room, under topical proparacaine anesthesia. Conjunctiva and Tenon capsule were incised in the superotemporal quadrant 6–8 mm posterior to the limbus. A 23-gauge curved blunt subtenon cannula was introduced and 40 mg/mL TA (Kenacort-A, 40 mg/mL; Bristol-Myers Squibb, Princeton, NJ) was injected avoiding drug reflux. Postoperative visits were performed at 1, 3, and 6 months. BCVA, IOP and CRT measurements were repeated on each visit. If there is an increase of more than 5 mmHg in IOP compared to baseline or IOP > 21.0 mmHg; a topical antiglaucomatous was started. If DME recurs, reinjection was performed. Reinjection criteria were a vision loss ≥ 5 letters and an increase of 50–100 μm in CRT. The main outcome measures were BCVA and CRT changes at month 1 and 3.

Statistical Analysis

Statistical analysis was performed with the statistical program Statistical Packages for the Social Sciences 21.0 (SPSS Inc., Chicago, IL). The data obtained by taking the average of all measurements were recorded as mean \pm standard deviation. One-way analysis of variance was used for multiple comparisons, and paired t-test was used for pairwise comparisons. Relationships between dependent variables were analyzed by Pearson correlation analysis. A value of $p < 0.05$ was considered statistically significant.

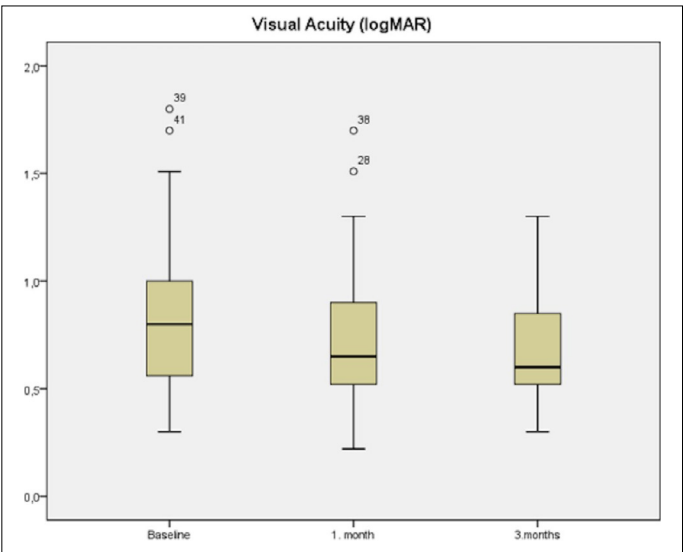


Fig. 1. Best corrected visual acuity improvement, following posterior subtenon triamcinolone injection, from baseline to 1st and 3rd months.

Results

Twenty patients, 20/37 (54%), were male and 17/37 (46%) were female. Mean age was 62.4±6.8 (50–81). All the patients had type II diabetes. Mean HbA1c was 8.3±1.9 (5.8–12.3) mg/dL. Twenty-four 24/37 (64.9%) of the patients had hypertension. Thirteen (29.5%) of the eyes were pseudophakic. Mean follow-up was 15.1±14.6 (6–53) months. The mean reinjection interval was 4.6±1.2 months. Mean reinjection number per eye was 2.8±1.8 in all study period. Mean BCVA was 0.86±0.36 LogMAR (counting fingers at 1 m and 45 letters) at baseline. A statistically significant improvement in BCVA from 0.86±0.36 to 0.73±0.31 (–0.13) was observed 1 month after PSTA injection (p=0.000) (Fig. 1). BCVA improved further to 0.68±0.24 (–0.18) LogMAR at month 3. When the 3rd month values were compared with baseline, there was a statistically significant improvement in BCVA (p=0.000), decrease in CRT (p=0.000), and increase in IOP (p=0.000) (Table 1). Of the 44 eyes, vision improved in 29/44 (65.9%), not changed in 13/44 (29.6%), and decreased in 2/44 (4.5%)

at month 1. BCVA improvement at 1 month was 5 letters in 15/44 eyes (34.1%), 10 letters in 8/44 eyes (18.2%), 15, 20, and 25 letters in 1 eye (2.3%) each. Mean letter gain was 8.3 (5–25) letters at month 1. In patients who responded well after injection treatment, serous macular detachment regressed, tense Muller cell bridges and cystic spaces disappeared. There was a decrease in the number of hyperreflective spots. A more significant increase in vision was achieved in cases with regular outer retina layers and regular ellipsoid zone. When the 3rd month values were compared with month 1; BCVA showed a statistically significant increase (p=0.027) and CRT showed a significant decrease (p=0.021) (Fig. 2). Thirteen eyes did not gain vision at month 1. However, 4 of them gained vision (late responders) at month 3. Seven eyes remained the same, and 2 eyes lost 5 letters at month 3. Overall; when compared with baseline, BCVA improved in 29 eyes (65.9%), remained the same in 13 (29.6%), and

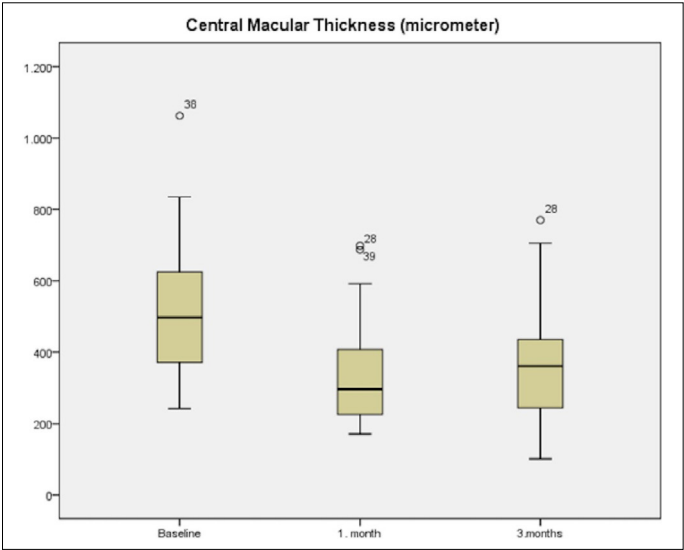


Fig. 2. Resolution of diabetic macular edema 3 months after posterior subtenon triamcinolone injection in a 67-year-old female diabetic macular edema patient with complete panretinal photocoagulation. Best corrected visual acuity increased from 5 letters to 20 letters, and central retinal thickness decreased from 828 µm to 173 µm at month 3.

Table 1. Study results

	Baseline	Month 1	Month 3	P*	P**
BCVA (LogMAR)	0.86±0.36	0.73±0.31	0.68±0.24	0.000	0.000
Letter gain		8.3 (5–25)	9.8 (5–30)		
CRT (µm)	509.1±177	337.8±143	366.9±145	0.000	0.000
IOP (mmHg)	14.5±3.8	18.6±5.6	17.6±3.3	0.000	0.000

P*: Month 1 × baseline; P**: Month 3 × baseline; CRT: Central retinal thickness; IOP: Intraocular pressure; BCVA: Best-corrected visual acuity; LogMAR: Logarithm of the minimum angle of resolution.

decreased in 2 (4.5%) eyes at month 3. Mean letter gain was 9.8 (5–30) letters at month 3.

There was no change in vision at 1 and 3 months after injection in 11 eyes. These eyes were analyzed further. Interestingly, all of these eyes had a decrease in CRT compared to baseline, except one real “unresponder.” That means anatomical success was 98% but functional success was 75%. An improvement in vision over time can be expected in these eyes. In those 11 eyes, the mean baseline CRT was $534.6\ \mu\text{m}$ (283–1062), and it decreased to mean $333.6\ \mu\text{m}$ (212–510) at 3 months.

A statistically significant decrease in mean CRT was observed 1 month after PSTA injection. Mean CRT decreased from $509.1\pm177\ \mu\text{m}$ to $337.8\pm143\ \mu\text{m}$ at month 1 ($p=0.000$). CRT decreased in 40 eyes (90.9%) and increased in 4 eyes (9.1%) at 1 month. The increase was $5\ \mu\text{m}$, $6\ \mu\text{m}$, and $10\ \mu\text{m}$ in three eyes and $48\ \mu\text{m}$ in 1 eye. A minor reincrease was observed at month 3 and mean CRT rose to $366.9\pm145\ \mu\text{m}$ ($+29.1\ \mu\text{m}$). However, mean change was still $-142.2\ \mu\text{m}$ compared to baseline (Fig. 3).

Complete resolution of DME, with a CRT $\leq 250\ \mu\text{m}$, was achieved in 19 eyes (43.2%) at month 1 and 15 eyes (34.1%) at month 3. The majority of the patients responded to therapy. CRT decreased in 41 (93.2%) eyes, increased in 3 eyes (6.8%) at month 3 compared to baseline. Two of these 3 eyes had shown an initial CRT decrease at month 1, but

exceeded preinjection level at month 3. Thus, only 1 eye of 44 eyes (2.3%) never responded to therapy at any visit (unresponder).

Safety: IOP showed a statistically significant increase 1 month after the injection ($p=0.000$). Seven eyes 7/44 (15.9%) showed an IOP over 21 mmHg or an increase of more than 5 mmHg. Mean IOP increased from $14.5\pm3.8\ \text{mmHg}$ to $18.6\pm5.6\ \text{mmHg}$ at 1 month. It showed a gradual decrease to $17.6\pm3.3\ \text{mmHg}$ at month 3, that change was not statistically significant ($p=0.152$). IOP increase occurred at month 1 in four of 7 eyes and at month 3 in three eyes. IOP was controlled successfully in all 7 eyes with temporary topical antiglaucomatous medication. No injection-related complications occurred, such as infection or globe perforation. Cataract surgery was performed in 5 eyes (11.4%) all through the follow-up.

Discussion

Our study results showed that PSTA is effective in the treatment of DME in eyes with full PRP. It provided visual improvement in 2/3 of the eyes. Anatomical success was much higher, exceeding 90%. However, no visual improvement was achieved in 1/4 of the eyes despite a significant decrease in CRT. Similarly, Luis et al.^[11] reported no statistically significant visual improvement despite mean $-117\ \mu\text{m}$ decrease in CRT with neither intravitreal dexamethasone nor TA in refractory DME. Authors stressed that the disorganization of retinal inner layers in 62.5% of the eyes was the main reason for that. Concurrent cataract progression may also limit the visual improvement after steroid injection. PRP can cause optic atrophy and macula ganglion cell loss, limiting vision. Furthermore, the proliferative phase is the last phase of the diabetic retinopathy and usually associated with a long-standing DME. Chronic edema can cause permanent damage to photoreceptors and Müller cells. Eventual foveal atrophy also leads to visual impairment despite a “thin” CRT. Browning et al.^[1] reported that the outer retinal layer thickness correlated better with visual acuity than CRT.

In our study, a significant improvement in vision was achieved, mean change was $-0.13\ \text{LogMAR}$ at month 1 and $-0.18\ \text{LogMAR}$ at month 3. These numbers were reported in a review study as -0.14 and $-0.07\ \text{LogMAR}$, respectively, for IVTA.^[12] In our study, BCVA improvement ≥ 5 letters was achieved in 59.2%. That rate is higher than the reported rate of 56% with IVTA.^[13] Mean letter gain at month 3 was 9.8 letters in our study, whereas only 5.7 letters with IVTA.^[13] In our opinion, patient selection is the key factor for that success.

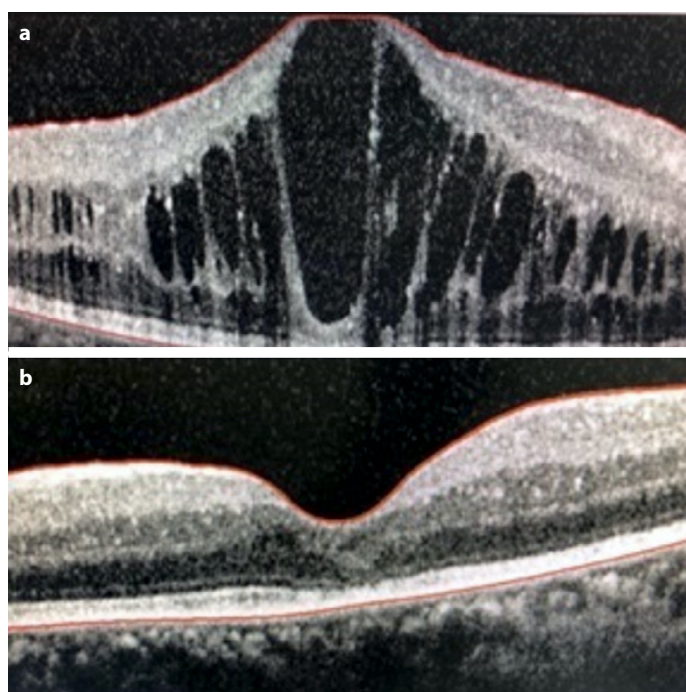


Fig. 3. Changes in central macular thickness from baseline through the 1st and 3rd months after posterior subtenon triamcinolone acetonide injection.

Only patients with full PRP were included in our study. In these eyes, VEGF secretion is diminished since ischemic areas are destroyed by photocoagulation. Our results suggest that DME in these eyes is caused by interleukin and similar inflammatory mediators rather than VEGF. That renders the effect of PSTA more prominent in eyes with panretinal laser. PSTA is also known to be effective in inflammatory macular edema such as uveitis and pseudophakic cystoid macular edema.^[14,15] Koga et al.^[16] reported the mean laser flare, which is an indicator of intraocular inflammation, decreased from baseline value of 15.5 photon/ms to 9.6 at 1 month after PSTA. This result shows the anti-inflammatory effect of PSTA. They also reported re-elevated laser flare values before recurrences of DME.

PSTA has been successfully used in refractory DME.^[17] In Koga's study,^[16] inferotemporal PSTA was performed on 20 eyes for refractory DME in vitrectomized eyes, and significant decrease in CRT (−174 μm) was achieved at 1 week. That decrease in edema is quite similar to our 1st month result of −171.3 μm. In Koga's study,^[16] DME resolved in 65%, improved in 20%, and unchanged in 15%. Higher success may be due to a previous vitrectomy. In our study, these percentages were 43%, 48% and 9% respectively at 1 month. Ozdek et al.^[8] compared 20 mg PSTA with 4 mg IVTA for DME refractory to grid laser. Mean CRT decreased −101 μm in PSTA and −206.1 μm in IVTA at 4 months. This reduction provided by the subtenon route is lower than our result of −171,3 μm. It may be due to their use of half dose. Although better edema resolution was achieved with IVTA, they reported PSTA was also effective and safer. In other studies, PSTA was found as effective as IVTA in diffuse DME with less complications.^[7,18] Furthermore, PSTA has been found equally effective as intravitreal dexamethasone implant in vitrectomized eyes with DME.^[19]

However, the effect of PSTA declines after 3 months, and reinjection becomes necessary.^[8,16] Treatment benefit is

no longer significant after 6 months.^[12] DME recurrence was reported in 7.1% after a single PSTA.^[8] In our study, a mild recurrence of edema was observed, manifested by a 29.1 μm increase in CRT at 3 months. Overall, 45.5% of our study eyes received at least one reinjection, within 4–25 months after the first injection. The mean reinjection interval was 4.6 months that is comparable with intravitreal steroid implants.^[20] Koga et al.^[16] reported half of the eyes needed a second injection at a mean 6.6 months after PSTA. In parallel with previous reports,^[16,21] no serious complications occurred in our patients due to PSTA injection, such as endophthalmitis or scleral perforation. IOP elevation was observed in 15.9% and controlled medically. Maeda et al.^[22] reported that rate 14.7% after PSTA. IOP increase rate was reported as 24.3% after IVTA. [8] Multiple injections during long follow-up spanning 15, 1 months might have caused cumulative IOP rise in our study. Anterior migration of TA can also increase IOP.^[23] Disruption of the host defense makes the eye more prone to infection after IVTA. Pseudomonas endophthalmitis was reported 2.4%.^[8] Cataract progression is another drawback. Cataract surgery was performed on 11.4% of the eyes in our study. Development of cataract may be due to either PSTA or natural course in an old and diabetic patient cohort during long follow-up. There are some limitations of the current study; like the lack of placebo or anti-VEGF control groups and detailed analysis of OCT biomarkers. It has been reported that, presence of hyperreflective foci in the outer retina and disorganisation of the photoreceptor layer are associated with low visual acuity.^[24] DME with high inflammatory OCT biomarkers like hyperreflective foci or subretinal fluid may respond better to intravitreal steroid implant.^[25,26] In addition, intravitreal steroid implants may be useful in cases resistant to anti-VEGFs.^[27]

For easier comparison, the results of PSTA in several types of DME in different studies are shown in Table 2.

Table 2. Comparison of different studies reporting outcomes of subtenon triamcinolone injection in various clinical settings

Study	DME status	TA injection route	Dose	Visual improvement	Decrease in CRT (μm)	Follow-up (months)
Yuksel (2025) 44 eyes	Full panretinal photocoagu-lation	Posterior subtenon	40 mg	9.8 letters (at 3 months)	201	15.1
Koga et al. (2005) 20 eyes	Pars plana vitrectomy	Posterior subtenon	40 mg	>2 lines in %45	174	13.3
Ozdek et al. (2006) 85 eyes	Refractory to grid laser	Posterior subtenon versus intravitreal	20 mg	0.03 decimal Snellen score	101	4.1
Jeon et al. (2024) 40 eyes	Bevacizumab-resistant	Posterior subtenon versus intravitreal		0.06 LogMAR	63.75	3.0

DME: Diabetic macular edema; TA: Triamcinolone acetate; CRT: Central retinal thickness; LogMAR: logarithm of the minimum angle of resolution.

Conclusion

Although there are few studies akin to the current study,^[18,28,29] it is the first research reporting the promising results of PSTA for the treatment of DME in eyes with full PRP. It provides fast resolution of the edema confirmed by improvement in OCT findings. As a result, PSTA may be considered as a reasonable alternative in the treatment of DME in eyes with complete PRP, since it is a highly effective and also safe extraocular intervention with few side effects.

Ethics Committee Approval: The İzmir Bozyaka Training and Research Hospital Ethics Committee granted approval for this study (date: 04.04.2022, number: 2022/63).

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References

- Browning DJ, Stewart MW, Lee C. Diabetic macular edema: Evidence-based management. *Indian J Ophthalmol* 2018;66:1736-50. [\[CrossRef\]](#)
- Noma H, Yasuda K, Shimura M. Involvement of cytokines in the pathogenesis of diabetic macular edema. *Int J Mol Sci* 2021;22:3427. [\[CrossRef\]](#)
- Edelman JL, Lutz D, Castro MR. Corticosteroids inhibit VEGF-induced vascular leakage in a rabbit model of blood-retinal and blood-aqueous barrier breakdown. *Exp Eye Res* 2005;80:249-58. [\[CrossRef\]](#)
- Kurt MM, Çekiç O, Akpolat Ç, Aslankurt M, Elçioglu M. Vessel diameter study: Intravitreal vs posterior subtenon triamcinolone acetonide injection for diabetic macular edema. *Eye (Lond)* 2017;31:1155-62. [\[CrossRef\]](#)
- Karatas G, Erden B, Cakir A, Bolukbasi S, Erdenoz S, Argon BD, et al. Intravitreal ranibizumab versus intravitreal ranibizumab combined with posterior subtenon triamcinolone acetonide in diabetic macular edema. *Beyoglu Eye J* 2021;6:229-35. [\[CrossRef\]](#)
- Thomas ER, Wang J, Ege E, Madsen R, Hainsworth DP. Intravitreal triamcinolone acetonide concentration after subtenon injection. *Am J Ophthalmol* 2006;142:860-1. [\[CrossRef\]](#)
- Choi YJ, Oh IK, Oh JR, Huh K. Intravitreal versus posterior subtenon injection of triamcinolone acetonide for diabetic macular edema. *Korean J Ophthalmol* 2006;20:205-9. [\[CrossRef\]](#)
- Ozdek S, Bahçeci UA, Gürelik G, Hasanreisoglu B. Posterior subtenon and intravitreal triamcinolone acetonide for diabetic macular edema. *J Diabetes Complications* 2006;20:246-51. [\[CrossRef\]](#)
- Chiu CY, Huang TL, Chang PY, Chen FT, Hsu YR, Chen YJ, et al. Combined intravitreal ranibizumab and posterior subtenon triamcinolone acetonide injections for patients with diabetic macular edema refractory to intravitreal ranibizumab monotherapy. *Taiwan J Ophthalmol* 2021;11:251-8. [\[CrossRef\]](#)
- Everett LA, Paulus YM. Laser therapy in the treatment of diabetic retinopathy and diabetic macular edema. *Curr Diab Rep* 2021;21:35. [\[CrossRef\]](#)
- Luís ME, Sampaio F, Costa J, Cabral D, Teixeira C, Ferreira JT. Dril influences short-term visual outcome after intravitreal corticosteroid injection for refractory diabetic macular edema. *Curr Eye Res* 2021;46:1378-86. [\[CrossRef\]](#)
- Qi HP, Bi S, Wei SQ, Cui H, Zhao JB. Intravitreal versus subtenon triamcinolone acetonide injection for diabetic macular edema: A systematic review and meta-analysis. *Curr Eye Res* 2012;37:1136-47. [\[CrossRef\]](#)
- Gillies MC, Sutter FK, Simpson JM, Larsson J, Ali H, Zhu M. Intravitreal triamcinolone for refractory diabetic macular edema: Two-year results of a double-masked, placebo-controlled, randomized clinical trial. *Ophthalmology* 2006;113:1533-8. [\[CrossRef\]](#)
- Teper SJ. Update on the management of uveitic macular edema. *J Clin Med* 2021;10:4133. [\[CrossRef\]](#)
- Erden B, Çakır A, Aslan AC, Bölükbaşı S, Elçioglu MN. The efficacy of posterior subtenon triamcinolone acetonide injection in treatment of irvine-gass syndrome. *Ocul Immunol Inflamm* 2019;27:1235-41. [\[CrossRef\]](#)
- Koga T, Mawatari Y, Inumaru J, Fukushima M, Tanihara H. Trans-Tenon's retrobulbar triamcinolone acetonide infusion for refractory diabetic macular edema after vitrectomy. *Graefes Arch Clin Exp Ophthalmol* 2005;243:1247-52. [\[CrossRef\]](#)
- Liu B, Ma G, Hou J, Cong C. Choroidal structural changes of posterior subtenon triamcinolone acetonide injection in eyes with refractory diabetic macular edema. *J Ophthalmol* 2022;2022:6882607. [\[CrossRef\]](#)
- Oshitari T, Kitamura Y, Nonomura S, Arai M, Takatsuna Y, Sato E, et al. Risk factors for refractory diabetic macular oedema after sub-tenon's capsule triamcinolone acetonide injection. *J Ophthalmol* 2015;2015:195737. [\[CrossRef\]](#)
- Gumus G, Erdogan G, Gunay BO, Karatas Durusoy G. Comparison of intravitreal dexamethasone implant and posterior subtenon triamcinolone injection in the treatment of diabetic macular edema in vitrectomized eyes. *Ophthalmologica* 2022;245:439-45. [\[CrossRef\]](#)
- Augustin AJ, Becker MD, Hatz K, Kaymak H, Shirlaw A. Assessment of reinjection numbers and intervals for diabetic macular edema patients who received dexamethasone intravitreal implants in Germany and Switzerland. *Clin Ophthalmol* 2021;15:3957-67. [\[CrossRef\]](#)

21. Khalil MMAA, Mansour HO, Tawfik AMR, Elmahdy AG. Comparison between intravitreal ranibizumab injection and posterior subtenon triamcinolone acetonide injection at time of cataract surgery for prevention of progression of diabetic macular edema. *BMC Ophthalmol* 2022;22:492. [\[CrossRef\]](#)
22. Maeda Y, Ishikawa H, Nishikawa H, Shimizu M, Kinoshita T, Ogihara R, et al. Intraocular pressure elevation after subtenon triamcinolone acetonide injection; Multicentre retrospective cohort study in Japan. *PLoS One* 2019;14:e0226118. [\[CrossRef\]](#)
23. Yang YH, Hsu WC, Hsieh YT. Anterior migration of triamcinolone acetonide after posterior subtenon injection for macular edema predisposes to intraocular pressure elevation. *Curr Eye Res* 2021;46:689-93. [\[CrossRef\]](#)
24. Uji A, Murakami T, Nishijima K, Akagi T, Horii T, Arakawa N, et al. Association between hyperreflective foci in the outer retina, status of photoreceptor layer, and visual acuity in diabetic macular edema. *Am J Ophthalmol* 2012;153:710-7. [\[CrossRef\]](#)
25. Taloni A, Coco G, Rastelli D, Buffon G, Scorcia V, Giannaccare G. Safety and efficacy of dexamethasone intravitreal implant given either first-line or second-line in diabetic macular edema. *Patient Prefer Adherence* 2023;17:3307-29. [\[CrossRef\]](#)
26. Tang L, Xu GT, Zhang JF. Inflammation in diabetic retinopathy: Possible roles in pathogenesis and potential implications for therapy. *Neural Regen Res* 2023;18:976-82. [\[CrossRef\]](#)
27. Nalçacı S, Akkın C, Afrashi F. Dexamethasone implant in patients with diabetic macular edema resistant to anti-VEGF therapy. *Turk J Ophthalmol* 2019;49:73-7. [\[CrossRef\]](#)
28. Yamada Y, Takamura Y, Matsumura T, Morioka M, Gozawa M, Inatani M. Posterior subtenon infusion of triamcinolone acetonide as adjunctive treatment to panretinal photocoagulation using pattern scan laser for diabetic retinopathy. *Jpn J Ophthalmol* 2018;62:686-92. [\[CrossRef\]](#)
29. Jeon SH, Kim M, Roh YJ. Comparison of intravitreal preservative-free triamcinolone versus posterior sub-tenon triamcinolone acetonide injection for bevacizumab-resistant diabetic macular edema. *BMC Ophthalmol* 2024;24:25. [\[CrossRef\]](#)