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THE ROLE OF INTRAVITREAL TRIAMCINOLONE IN REFRACTORY DIABETIC MACULAR EDEMA: AN OCT STUDY

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ABSTRACT

Diabetic macular edema (DME) includes focal and diffuse types. Diffuse type responds poorly to laser and anti-VEGF treatments. Triamcinolone acetonide has been effective in treating various macular conditions. IVTA has shown promising results in managing diffuse DME unresponsive therapies. To investigate the effectiveness of IVTA for the treatment of DME unresponsive to previous laser photocoagulation and 2 doses of anti-VEGF. 21 patients from western Maharastra completed 3-8 months follow up. Sex distribution was 8 females and 13 males. At end of 1 month mean BCVA improved from 0.83 to 0.38. Mean OCT macular thickness improved from 448.24µm to 322.24µm. At the 3 month follow up mean visual acuity was 0.42. Mean OCT thickness was 331.38µm. One sample T-test value for all variables have significantly higher means than 0 ($p<0.001$) for all. The p value for BCVA correlation for pre and post injection is 0.002. The p value for central retinal thickness correlation for pre and post injection is 0.001. Refractory DME Patients showed anatomical and functional benefits from IVTA. Effect peaked at 1 month, but benefits partially sustained at 3 months. Reinjection timing and close monitoring might be needed beyond 3 months due to rebound.

KEYWORDS: Triamcinolone, Diabetic Macular Edema, Optical Coherence Tomography

Diabetic retinopathy is the leading cause of blindness among individuals aged 20 to 74 years in India. Among diabetic patients who have had the condition for 20 years or more, nearly 29% develop macular edema, which is the primary contributor to vision loss in this group (Klein *et al.*, 1984). The Early Treatment Diabetic Retinopathy Study (ETDRS) reported that individuals with clinically significant macular edema (CSME) had a 30% risk of experiencing moderate vision loss within three years. This was defined as a doubling of the visual angle or a reduction of three lines on a logMAR visual acuity chart. The study showed that laser photocoagulation could lower the risk of moderate vision loss by about 50% in eyes affected by diabetic macular edema (DME) along with mild to moderate non-proliferative diabetic retinopathy. Despite treatment, 12% of eyes still suffered significant vision decline losing 15 or more ETDRS letters over three years while fewer than 3% saw notable visual improvement.

DME is categorized into two main types. The first, focal macular edema, is characterized by localized fluid leakage from microaneurysms, often accompanied by intraretinal lipid deposits in a circular, or circinate, pattern. The second type, diffuse macular edema, involves more extensive leakage from the retinal capillaries and is typically associated with cystoid space formation. Due to the widespread nature of fluid accumulation and thickening of the entire macula, targeted laser therapy is generally ineffective for diffuse macular edema. In the ETDRS, grid laser photocoagulation was applied to areas affected by diffuse edema. However, previous studies have shown that diffuse macular edema often carries a poor visual prognosis, even when treated with laser therapy and 2 doses anti-VEGF injections. (Bresnick, 1983; Lee and Olk, 1991)

Optical coherence tomography (OCT) enables high-resolution cross-sectional imaging of the retina by using light to identify variations in reflection at optical boundaries. This technology has demonstrated its effectiveness in measuring macular thickness in patients with diabetic macular edema. (Hee *et al.*, 1995 & 1998)

Triamcinolone acetonide (TA) is a corticosteroid suspension administered through periocular injections to treat cystoid macular edema caused by uveitis or resulting from intraocular surgery (Riordan-Eva and Lightman, 1994; Stern *et al.*, 1981). Intravitreal triamcinolone acetonide (IVTA) has been utilized to address various conditions, including uveitic cystoid macular edema (CME) (Antcliff *et al.*, 2001; Martidis *et al.*, 2001; Young *et al.*, 2001) exudative age-related macular degeneration (Danis *et al.*, 2000; Challa *et al.*, 1988; Jonas *et al.*, 2002; Spaide *et al.*, 2003), neovascular glaucoma (Jonas *et al.*, 2001; Jonas *et al.*, 2003), proliferative diabetic retinopathy (Jonas *et al.*, 2001), hypotony (Jonas *et al.*, 2001), proliferative vitreoretinopathy (Machemer *et al.*,

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1979; Tano *et al.*, 1980; Jonas *et al.*, 2000), macular edema due to retinal vascular occlusive disease, (Greenberg *et al.*, 2002; Jonas *et al.*, 2002; Degenring *et al.*, 2003; Park *et al.*, 2003) pseudophakic CME (Jonas *et al.*, 2003), and cystoid macular edema associated with retinitis pigmentosa (Saraiva *et al.*, 2003). Additionally, IVTA has shown promising outcomes in managing diffuse diabetic macular edema (Jonas and Sofker, 2001; Jonas *et al.*, 2003; Martidis *et al.*, 2002).

Due to the challenges in managing patients with diffuse macular edema that does not respond to laser therapy and two doses of anti-VEGF, we chose to investigate the potential effectiveness of IVTA in reducing macular edema and enhancing visual acuity. OCT imaging was utilized to quantitatively evaluate changes in retinal thickness before and after IVTA treatment.

MATERIALS AND METHODS

We conducted a retrospective chart review of medical records from a series of consecutive patients diagnosed with clinically significant macular edema (CSME) in Western Maharashtra. A total of 21 eyes from 21 diabetic patients met the ETDRS criteria for CSME and were treated with intravitreal triamcinolone acetonide (IVTA) between January 2019 and December 2023. All patients underwent thorough evaluation and received appropriate management. Each patient was fully informed about the nature of the treatment and provided written informed consent.

In this series, every eye had previously undergone at least two sessions of laser photocoagulation and received two doses of anti-VEGF therapy, in accordance with ETDRS guidelines. The most recent laser treatment was performed at least three months prior to IVTA administration. All eyes selected for IVTA treatment exhibited persistent macular thickening on clinical examination, accompanied by a decline in visual acuity compared to baseline. Optical coherence tomography (OCT) confirmed central foveal thickness greater than 300μm. None of the eyes had a prior history of ocular hypertension or glaucoma.

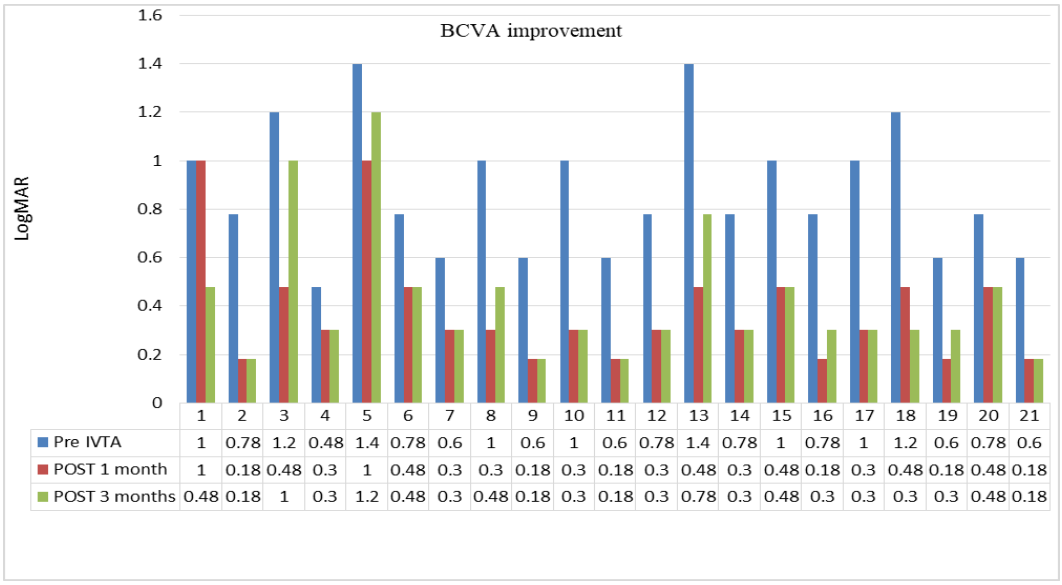
IVTA was offered to treat residual macular edema. At baseline all patients received a complete eye examination inclusive of best corrected visual acuity, applanation tonometry, slit lamp examination, lens status evaluation, dilated fundus examination, and biomicroscopy of the posterior pole with a contact lens.

All patients underwent baseline OCT imaging to measure central macular thickness. For each eye, the OCT software's topographic mapping protocol was used. This method, previously described, involves acquiring six consecutive linear scans each 3.01 mm long at each visit. These scans are taken at evenly spaced angles in a radial spoke pattern centered on the point of fixation. Each of the six tomographic images (B-scans) is aligned along a line passing through the central fovea and includes 100 evenly spaced depth profiles (A-scans) that capture optical reflectivity. An automated computer algorithm then identifies the inner and outer retinal boundaries on each B-scan, from which retinal thickness is calculated.

Color fundus photographs were taken at baseline and during each follow-up visit. These images were used to assess the presence of hard exudates in the macular region at both the initial and subsequent visits

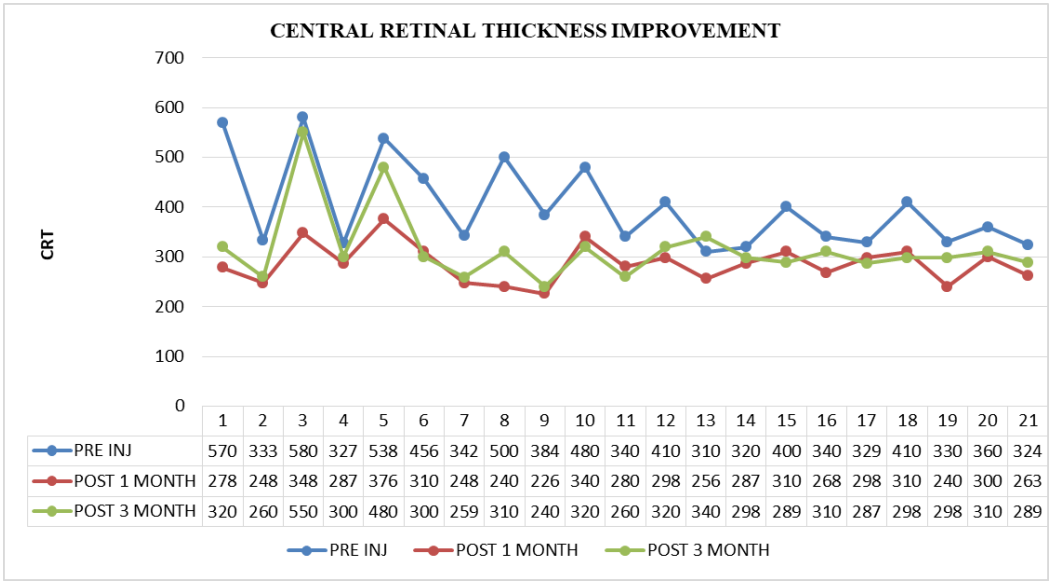
All patients received an IVTA injection following the same procedure. Topical anesthesia was achieved using proparacaine eye drops, followed by the application of two drops of moxifloxacin and a 5% povidone-iodine solution. A dose of 4 mg triamcinolone acetonide in 0.1 ml was then injected into the vitreous cavity in the inferotemporal quadrant using a 30-gauge needle. The correct placement of the injection and the optic nerve head perfusion were verified using indirect ophthalmoscopy.

Patients were scheduled for follow-up visits at 1 day, 1 month, 3 months, and 8 months after treatment. Those who experienced a decline in vision were evaluated sooner and, if needed, received additional treatment before their next scheduled visit. Subsequent follow-up appointments were arranged based on the recurrence of macular edema and the need for repeat IVTA injections. OCT scans were conducted at baseline and during every follow-up visit for all patients. Any complications related to the corticosteroid or the injection procedure were also monitored.



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Pre IVTA	1	0.78	1.2	0.48	1.4	0.78	0.6	1	0.6	1	0.6	0.78	1.4	0.78	1	0.78	1	1.2	0.6	0.78	0.6
POST 1 month	1	0.18	0.48	0.3	1	0.48	0.3	0.3	0.18	0.3	0.18	0.3	0.48	0.3	0.48	0.18	0.3	0.48	0.18	0.48	0.18
POST 3 months	0.48	0.18	1	0.3	1.2	0.48	0.3	0.48	0.18	0.3	0.18	0.3	0.78	0.3	0.48	0.3	0.3	0.3	0.3	0.48	0.18

Figure 1: BCVA (Best corrected visual acuity)



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
PRE INJ	570	333	580	327	538	456	342	500	384	480	340	410	310	320	400	340	329	410	330	360	324
POST 1 MONTH	278	248	348	287	376	310	248	240	226	340	280	298	256	287	310	268	298	310	240	300	263
POST 3 MONTH	320	260	550	300	480	300	259	310	240	320	260	320	340	298	289	310	287	298	298	310	289

Figure 2: Central Retinal Thickness

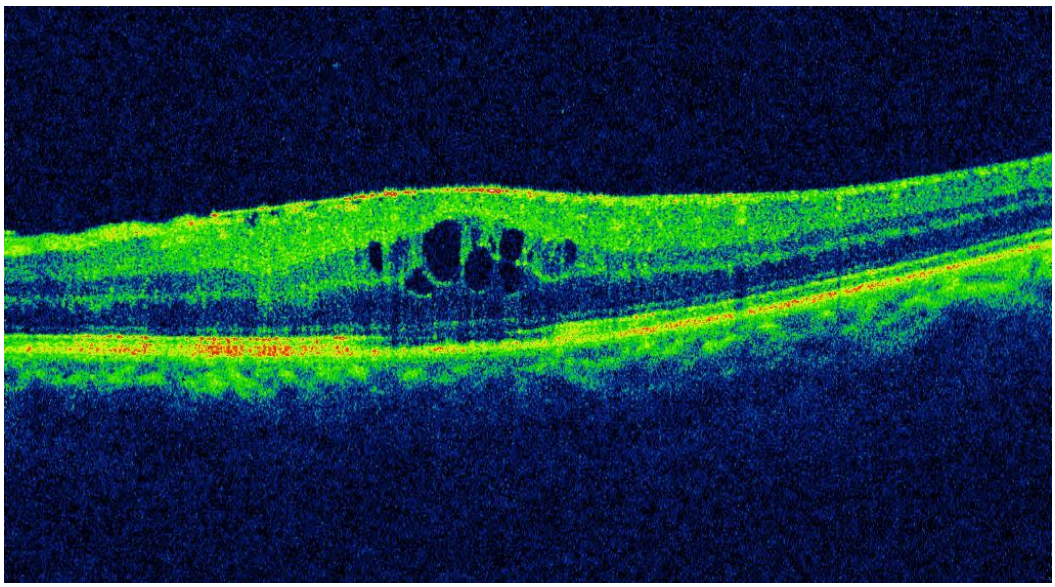


Figure 3: OCT showing Refractory Diabetic macular Edema (Pre-injection)

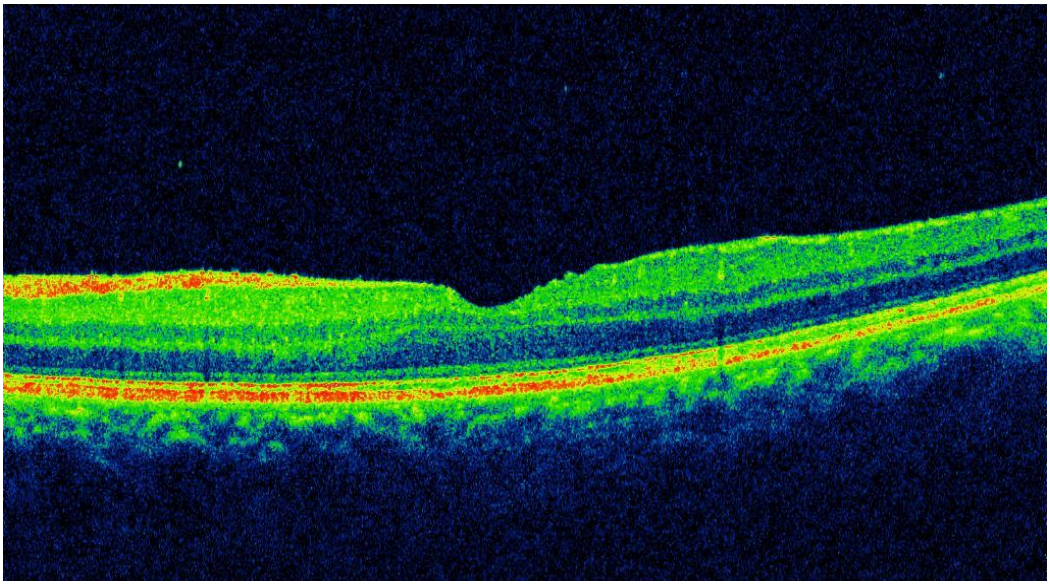


Figure 4: OCT showing foveal contour maintained after Intravitral IVTA Injection

RESULTS

There were 8 females and 13 males, for a total of 21 eyes. Mean (SD) age of the patients was 62.80 years (median, 62 years; range, 46–78 years). Pre-injection Mean (SD) visual acuity was 0.83 (median, 0.78; range 0.48–1.4), Mean (SD) OCT central retinal thickness was 448.24µm (median, 360µm; range, 310–580µm). All 21 eyes completed 3 months of follow up, 7 eyes completed 8 months of follow up. The p value for BCVA correlation for pre-injection and 1month post injection is 0.002 (which is less than 0.5) and BCVA between pre-injection and 3 month post injection is 0.01 (which is less than 0.5). The p value for central retinal thickness correlation for pre-injection and 1month post injection is 0.001 (which is less than 0.5) and central retinal thickness between pre-injection and 3 month post injection is 0.001 (which is less than 0.5). One sample T-test value for all variables have significantly higher means than 0 ($p<0.001$ for all), indicating strong statistical deviation from zero.

At the 1 month follow up mean (SD) visual acuity improved from 0.83 to 0.38 (median, 0.3; range 0.18-1) (fig 1). Mean (SD) OCT macular thickness improved from 448.24µm at baseline of the study to 322.24µm (median, 287µm; range 226–376µm) (Fig 2). At the 3 month follow up mean (SD) visual acuity was 0.42 (median 0.3; range 0.18–1.2). Mean (SD) OCT macular thickness was 331.38µm (median, 300µm; range 240–550µm). In the first month of follow up 20 (95%) of 21 eyes experienced improvement in vision and all 21(100%) patients showed decrease in OCT central retinal thickness. In the 3 months of follow up 21 (100%) of 21 eyes experienced improvement in vision and all 21(100%) patients showed decrease in OCT central retinal thickness compared to pre-injection status. There was a reduction in macular thickness at 1 month post-IVTA, with partial rebound by 3 months. This is consistent with the known pharmacodynamics of IVTA (peal effect at 4-6 weeks, tapering thereafter). There was gain in visual acuity by 1 month that remained largely stable by 3 months. Although there was slight decline between 1 and 3 months, visual function remained much better than baseline.

Patients unresponsive to laser and anti-VEGF showed clear anatomical and functional benefits from IVTA. Effect peaked at 1 month, but benefits partially sustained at 3 months. Reinjection timing and close monitoring might be needed beyond 3 months due to rebound. (All result show in figure 1, 2, 3 & 4)

DISCUSSION

The use of intravitreal corticosteroid was first advocated by Machemer *et al* for the treatment of proliferative vitreoretinopathy (Machemer *et al.*, 1979). McCuen *et al.*(1981) demonstrated in an experimental rabbit model the lack of ocular toxicity of intravitreal triamcinolone acetonide. Their results were in agreement with clinical observation of cases in which corticosteroids were accidentally injected into the eye, and no major toxic reactions were detected (Giles, 1974; McLean, 1975). Since then for decades intravitreal steroids have been used in experimental trials in patients without noticing a significant toxic effect.

Although the precise way corticosteroids work in treating macular edema is not fully understood, their use is supported by their ability to inhibit the arachidonic acid pathway, which leads to the production of prostaglandins. Additionally, corticosteroids may reduce the levels of vascular endothelial growth factor (VEGF).

Experimental studies have also shown that triamcinolone acetonide can help maintain the integrity of the blood-retinal barrier (Wilson *et al.*, 1992). Since diabetic macular edema involves disruption of this barrier and both prostaglandins and VEGF are believed to contribute to the condition, there is a sound theoretical basis for using corticosteroids as a treatment option.

Martidis *et al.* (2002) recently reported on the use of IVTA at the dose of 4 mg in 0.1 ml for the treatment of refractory diabetic macular edema. Of the 16 eyes included in the study, 14 were assessed at the 1-month follow-up, while 8 eyes (50%) were monitored through the 6-month mark. On average, visual acuity improved by 2.4 Snellen lines at both 1 and 3 months, and by 1.3 lines at 6 months. Central macular thickness, as measured by OCT, showed reductions of 55%, 57.5%, and 38% at 1, 3, and 6 months, respectively, from a baseline mean (±SD) of 540.3 (96.3) micrometers.

Elevated intraocular pressure above 21 mm Hg was observed in five eyes at 1 month, three eyes at 3 months, and one eye at 6 months. Cataract progression was noted in one eye at the 6-month follow-up. No additional complications were reported during the average follow-up period of 6.2 months. Due to recurrence of diabetic macular edema, three out of eight eyes (37%) required a repeat injection after 6 months.

Jonas *et al.* (2003) reported similar results with the use of IVTA at the dose of 25 mg in 0.2 ml in diabetic patients with diffuse macular edema. The study included 26 eyes from 20 patients, with a mean follow-up duration of 6.64 months (±6.10). Average visual acuity improved from 0.12 (±0.08) at baseline to 0.19 (±0.14) during follow-up. One patient received a second intravitreal injection of 25 mg triamcinolone acetonide. Among the 22 patients with both pre- and post-injection fluorescein angiograms, a marked reduction in fluorescein leakage was observed following treatment with IVTA. Elevated intraocular pressure above 21 mm Hg was recorded in nine of the 26 eyes (34.6%) during the study period.

Both authors concluded that IVTA might be a promising therapeutic method for diabetic macular edema. The findings indicate that IVTA is effective in enhancing visual acuity and decreasing macular thickness, as measured by OCT. The greatest benefits are typically observed within 1 to 3 months after treatment. However, these improvements tend to diminish over time, with vision deteriorating and macular thickness increasing in the subsequent months. Administering a repeat IVTA injection was successful in restoring improvements in visual acuity. These data are consistent with the work of Beer *et al.*(2003) who showed that in human eyes after intravitreal injection, measurable concentrations of triamcinolone would be expected to last for approximately 3 months in a non-vitrectomised eye.

We also observed a gradual reabsorption of hard exudates in the macula throughout the follow-up period. While such reabsorption is generally anticipated with reduced intraretinal vascular leakage and improvement in macular edema, this effect has not previously been documented following IVTA treatment. Notably, all patients in our study had chronic macular edema and had previously undergone at least two ETDRS-guided laser treatments without any significant improvement in edema or resolution of lipid exudation.

This study does have several limitations. It is retrospective, non-randomised, and lacks a control group. Additionally, visual acuity was assessed using a Snellen chart rather than the more widely accepted and standardised ETDRS chart. Nevertheless, to the best of our knowledge, this study features a longer follow-up period than any previously published research on IVTA for diabetic macular edema (DME). Anatomical outcomes were thoroughly documented, and macular thickness was quantitatively assessed with OCT at each follow-up visit.

IVTA shows potential as a treatment option for patients with diabetic macular edema that does not respond to laser therapy. It has been found to enhance visual acuity, decrease macular thickness, and promote the reabsorption of hard exudates. Although endophthalmitis is the most serious potential complication associated with IVTA, no cases were reported in this study (Sutter and Gillies, 2003). Further research is needed to more thoroughly evaluate the effectiveness and safety of IVTA in managing refractory diabetic macular edema.

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