Original Article

# Short-term efficacy of intravitreal triamcinolone acetonide for macular edema secondary to retinal vein occlusion that is refractory to intravitreal bevacizumab

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**Aims:** To evaluate the 1-month efficacy of intravitreal triamcinolone acetonide (TA) in treating macular edema secondary to retinal vein occlusion (RVO) that was refractory to intravitreal bevacizumab. **Materials and Methods:** This retrospective, observational study included 23 eyes from 23 patients with macular edema secondary to RVO. Macular edema that did not respond to two or more consecutive intravitreal bevacizumab injections was treated with intravitreal TA. Central foveal thickness (CFT) and best-corrected visual acuity (BCVA) were compared before and one month after TA injection. **Results:** Fifteen eyes were diagnosed with central RVO, and eight eyes were diagnosed with branch RVO. All patients were previously treated with 2.4 ± 0.6 intravitreal bevacizumab injections. The TA injection was performed, on average, 5.8 ± 1.4 weeks after the last bevacizumab injection. The CFT before TA injection was 516.6 ± 112.4 μm and significantly decreased to 402.3 ± 159.7 μm after TA therapy ($P < 0.001$). The logarithm of the minimal angle of resolution BCVA was 0.72 ± 0.34 before TA therapy and was not significantly improved by the treatment (0.67 ± 0.35, $P = 0.119$), despite a decrease in CFT. However, seven eyes (30.4%) had a BCVA gain of one or more lines. **Conclusions:** Intravitreal TA therapy was beneficial in some patients with macular edema secondary to RVO that was refractory to intravitreal bevacizumab therapy. This study suggests that intravitreal TA should be considered as a treatment option for refractory macular edema.

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Intravitreal injection of triamcinolone acetonide (TA) is an effective treatment for macular edema secondary to retinal vein occlusion (RVO).[1-4] Unfortunately, common complication for this therapy include increased intraocular pressure, cataract progression, and noninfectious intraocular inflammation.[5,6] Intravitreal injection of anti-vascular endothelial growth factor (VEGF) has more recently been developed as a treatment for macular edema secondary to RVO.[7-9] Recent comparative studies have shown that anti-VEGF therapy has equal,[10-13] or even superior[4,14,15] efficacy as intravitreal TA with a lower complication rate. Many clinicians use anti-VEGF therapy as a first-line treatment for macular edema secondary to RVO. However, the efficacy of anti-VEGF is limited in some cases and treating the macular edema refractory to anti-VEGF has been deemed an important issue in RVO.

Efficacy of various treatment modalities, including vitrectomy,[16] anti-VEGF/TA combination therapy,[17] intravitreal dexamethasone implant,[18] and intravitreal pegaptanib[19] have been evaluated as alternative treatments for macular edema refractory to anti-VEGF therapy. Unfortunately, limited knowledge is available regarding the efficacy of intravitreal TA in treating the condition. Although one previous study showed that intravitreal TA may lead to an improvement in visual acuity and a decrease in refractory macular edema, the conclusion was drawn based on results from only two patients.[20]

The purpose of the present study was to evaluate the short-term efficacy of a single intravitreal TA in treating macular edema secondary to RVO that was refractory to anti-VEGF therapy.

## Materials and Methods

This retrospective, observational case series was performed at a single center. All study conduct adhered to the tenets of the declaration of Helsinki, and the study was approved by the institutional review board at Kim's Eye Hospital.

### Patients

We conducted a retrospective review of the medical records of patients who were diagnosed with macular edema secondary to RVO between January 2010 and December 2012. The inclusion criteria were as follows: (1) Initially treated with two or more consecutive intravitreal bevacizumab injections (1.25 mg/0.05 ml), (2) refractory to bevacizumab therapy (<150 μm reduction in central foveal thickness [CFT] or CFT >300 μm), (3) underwent intravitreal TA injection (4 mg/0.1 ml) within 8 weeks of last bevacizumab injection, (4) followed-up for at least one month after TA injection. Exclusion criteria included severe media opacity, previous vitreoretinal surgery, intraocular inflammation, and other disorders that may have influenced macular function (e.g. exudative age-related macular degeneration, proliferative diabetic retinopathy, epiretinal membrane). Patients with a visual acuity worse than 20/400 were also excluded.

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All subjects underwent a comprehensive ophthalmologic examination, including best-corrected visual acuity (BCVA) measurement, 90-diopter lens slit-lamp biomicroscopy, fundus photography, fluorescein angiography, and spectral domain optical coherence tomography (SD-OCT; Spectral OCT/SLO®; OTI Ophthalmic Technologies Inc., Miami, FL, USA). Because the evaluation of macular volume was not routinely performed as part of SD-OCT testing, CFT measurements were used in analyses. The vertical distance between the internal limiting membrane and the retinal pigment epithelium at the foveal center was measured based on horizontal and vertical foveal-centered SD-OCT images. The mean of OCT parameters measured on the horizontal and vertical scans were used in analyses. Visual acuity measurements were converted to the logarithm of the minimal angle of resolution for analyses.

### Outcome measures

The BCVA and CFT, one month after the last bevacizumab injection, was compared with those measured one month after TA injection. Eyes exhibiting >150 μm of a decrease in CFT or a CFT ≤250 μm after TA injection were classified into the responsive group. The remaining eyes were classified as nonresponsive group. In each group, BCVA and CFT after bevacizumab injection were compared to measurements made one month and three months after TA injection. Patient age, diagnosis, BCVA, and CFT before TA injection were compared between groups, as was the time between symptom onset and TA injection.

### Statistics

Data are presented as mean ± standard deviation. Comparisons of values between different time points within the same group were performed using a paired *t*-test, repeated measures analysis of variances, or Friedman test. Comparisons between the responsive and nonresponsive groups were performed using a Mann–Whitney U-test or a Fisher's exact test. Statistical analyses were performed with a commercially available software package (SPSS version 12.0 for Windows, SPSS Sciences, Chicago, IL, USA). A $P < 0.05$ was considered as significant.

## Results

Twenty-seven eyes from 27 patients satisfied eligibility criteria. Among these, 4 eyes were excluded because SD-OCT had not been performed after intravitreal TA injection. Ultimately, 23 eyes from 23 patients (12 male [52.2%], 11 female [47.8%]) were included in study analyses [Table 1]. Mean patient age was 59.8 ± 10.4 years (range: 41–80 years). Central RVO (CRVO) and branch RVO (BRVO) were diagnosed 15 (65.2%) and eight eyes (34.8%), respectively. Eighteen eyes (78.3%) were phakic, and five eyes (21.7%) were pseudophakic. At the time of RVO diagnosis, mean BCVA was 0.67 ± 0.34 (Snellen equivalent = 20/93, range: 20/400–20/30) and mean CFT was 523.5 ± 120.9 μm (range: 357–804 μm).

### Comparison of central foveal thickness and best-corrected visual acuity before and after intravitreal triamcinolone acetonide injection

Patients were initially treated with an average of 2.4 ± 0.6 (range: 2–4) monthly injections of intravitreal bevacizumab. Mean BCVA and CFT measured at 1 month after the last bevacizumab injection were 0.72 ± 0.34 and 516.6 ± 112.4 μm, respectively. The TA injection was performed an average of 5.8 ± 1.4 weeks after the last bevacizumab injection and the mean duration between symptom onset, and TA injection was 19.8 ± 4.6 weeks. Fig. 1 shows a representative case of change in macular thickness after TA treatment. One month after TA injection, mean BCVA and CFT had changed to 0.67 ± 0.35 and 402.3 ± 159.7 μm, respectively [Fig. 2]. Visual acuity had improved by one to two lines in 3 eyes (13.0%) and by two lines or greater in four eyes (17.4%). When compared with values measured before the injection, CFT had significantly decreased ($P < 0.001$), but the BCVA improvement was not significant ($P = 0.119$).

Seventeen eyes completed 3 months follow-up. In these eyes, the BCVA before TA injection and at 1 month and 3 months after the injection was 0.74 ± 0.29, 0.67 ± 0.30, and 0.73 ± 0.27, respectively [Fig. 2a]. The CFT was 521.1 ± 105.3 μm,

**Table 1: Baseline characteristics of patients with macular edema secondary to retinal vein occlusion that was refractory to intravitreal bevacizumab therapy (*n*=23 eyes)**

| Characteristic | |
|---|---|
| Age, years | 59.8±10.4 (range 41-80) |
| Sex, number (%) | |
| Male | 12 (52.2) |
| Female | 11 (47.8) |
| Diagnosis, number (%) | |
| Central retinal vein occlusion | 15 (65.2) |
| Branch retinal vein occlusion | 8 (34.8) |
| Lens status, number (%) | |
| Phakia | 18 (78.3) |
| Pseudophakia | 5 (21.7) |
| Number of bevacizumab injections | 2.4±0.6 (range 2-4) |
| logMAR BCVA | 0.67±0.34 (range 20/400-20/30) |
| Central foveal thickness, μm | 523.5±120.9 (range 357-804) |

Data presented as mean±SD, where applicable. BCVA: Best-corrected visual acuity, logMAR: Logarithm of the minimal angle of resolution, SD: Standard deviation

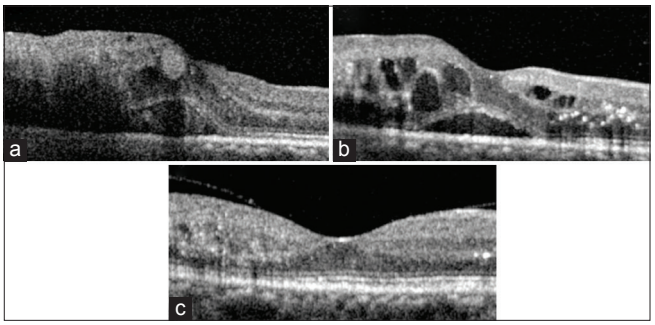


**Figure 1:** Optical coherence tomography images of an eye diagnosed with refractory macular edema secondary to branch retinal vein occlusion. Compared to baseline (a), macular edema remained relatively unchanged after three consecutive intravitreal bevacizumab injections (b). A marked decrease in macular edema was noted 1 month after an intravitreal injection of triamcinolone acetonide (c). Best-corrected visual acuity improved from 20/50 to 20/30 after triamcinolone acetonide injection

388.8 ± 166.8 μm, and 436.1 ± 149.4 μm, respectively [Fig. 2b]. The CFT measured at 1 and 3 months after TA injection was significantly decreased when compared with the value before the injection ($P = 0.004$ and $P = 0.018$, respectively). However, BCVA at 1 and 3 months after TA injection was not different when compared with the value before the injection ($P = 0.452$ and $P = 1.000$, respectively). At 3 months, visual acuity had improved by one to two lines in two eyes (11.8%) and by two lines or greater in two eyes (11.8%).

### Comparison between the responsive and nonresponsive groups

Nine eyes (39.1%) and 14 eyes (60.9%) were included in the responsive and nonresponsive group, respectively. In the responsive group, mean age was 60.1 ± 9.5 years. A CRVO and BRVO diagnosis were given to 4 (44.4%) and 5 (55.6%) eyes, respectively, and the time between symptom onset and TA injection was 21.2 ± 6.0 weeks. Mean BCVA at diagnosis, after bevacizumab injection, and after TA injection was 0.73 ± 0.32, 0.71 ± 0.32, and 0.52 ± 0.3 and CFT was 509.2 ± 78.8, 480.0 ± 102.3, and 237.8 ± 34.6 μm, respectively. Compared with measurements before intravitreal TA therapy, after TA injection BCVA had significantly improved ($P = 0.018$, Fig. 3a), and CFT had significantly decreased ($P = 0.008$, Fig. 3b). In the nonresponsive group, mean patient age was 59.6 ± 11.2 years. A CRVO and BRVO diagnosis were given to 11 (78.6%) and 3 (21.4%) eyes, respectively, and the time between symptom onset and TA injection was 18.9 ± 3.2 weeks. Mean BCVA at diagnosis, after anti-VEGF therapy, and after TA injection was 0.63 ± 0.36, 0.73 ± 0.36, and 0.76 ± 0.36 and CFT was 532.6 ± 143.9, 540.1 ± 115.9, and 508.0 ± 107.9 μm, respectively. Compared with measurements before intravitreal TA therapy, neither BCVA ($P = 0.102$) nor CFT ($P = 0.074$) had significantly changed [Fig. 3].

Patient age ($P = 0.926$), diagnosis distribution ($P = 0.179$), BCVA ($P = 0.898$), and CFT ($P = 0.219$) before TA injection were not significantly different between the two groups [Table 2]. In addition, the time between symptom onset and TA injection was not significantly different between the two groups ($P = 0.477$).

Eight eyes of the responsive group completed 3 months follow-up. In these eyes, the BCVA before TA injection and at 1 month and 3 months after the injection was 0.75 ± 0.32, 0.55 ± 0.30, and 0.65 ± 0.28, respectively. The CFT was 491.9 ± 102.5 μm, 238.4 ± 36.9 μm, and 351.6 ± 159.8 μm, respectively. The BCVA and CFT measured after TA injection was significantly decreased when compared with the value before the injection ($P = 0.017$ and $P = 0.011$, respectively). When compared to the values before the injection, BCVA had improved by one to two lines in two eyes (25.0%) and by two lines or greater in two eyes (25.0%). No eye exhibited deterioration in BCVA at 3 months. Nine eyes of the nonresponsive group completed 3 months follow-up. In these eyes, the BCVA before

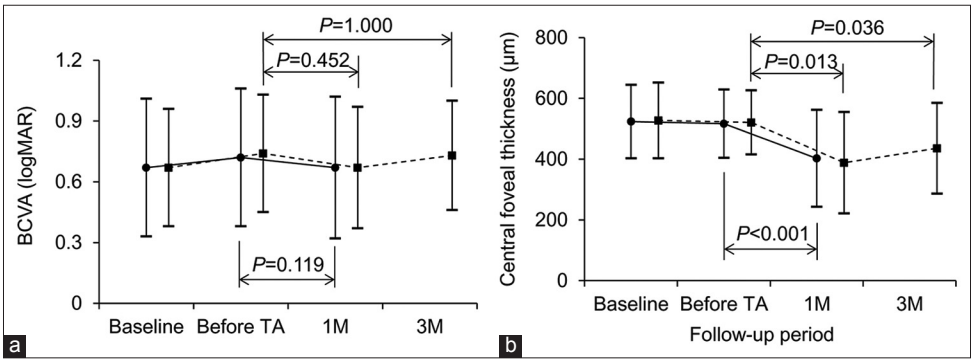


**Figure 2:** Changes in logarithm of the minimal angle of resolution best-corrected visual acuity (a) and central foveal thickness (b) in eyes with macular edema secondary to retinal vein occlusion. Measurements were made at diagnosis (baseline), after intravitreal bevacizumab, and after intravitreal triamcinolone acetonide. Statistical analyses were performed using paired *t*-tests. Solid line (closed circle) indicates all the included eyes ($n = 23$), whereas dashed line (closed square) indicates eyes completed 3 months follow-up ($n = 17$)

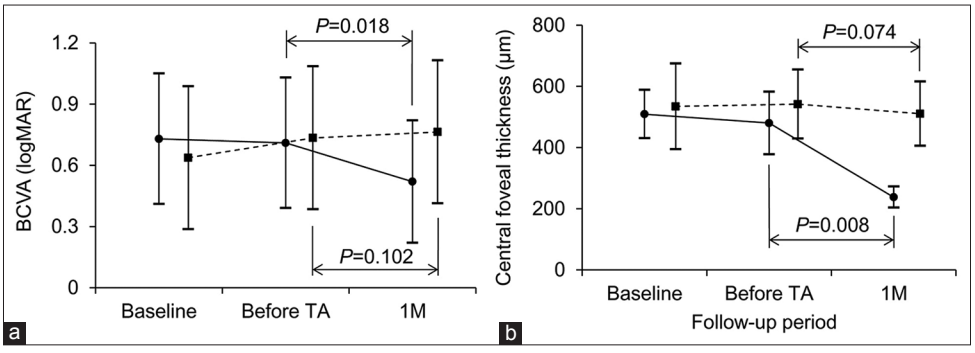


**Figure 3:** Changes in logarithm of the minimal angle of resolution best-corrected visual acuity (a) and central foveal thickness (CFT) (b) in eyes with macular edema secondary to retinal vein occlusion. Measurements were made at diagnosis (baseline), after intravitreal bevacizumab, and after intravitreal triamcinolone acetonide. The solid line (closed circle) indicates eyes that had a >150 μm decrease in CFT or a CFT ≤250 μm after TA injection (responsive group, $n = 9$). The remaining eyes were included in the nonresponsive group (dashed line, closed square, $n = 14$)

**Table 2: Comparison of characteristics of patients between the responsive group (*n*=9) and the nonresponsive group (*n*=14)**

| Characteristic | Responsive group (*n*=9) | Nonresponsive group (*n*=14) | *P* |
|---|---|---|---|
| Age, years | 60.1±9.5 | 59.6±11.2 | 0.926† |
| Diagnosis, number (%)* | | | |
| Central retinal vein occlusion | 4 (44.4) | 11 (78.6) | 0.179†† |
| Branch retinal vein occlusion | 5 (55.6) | 3 (21.4) | |
| Time between symptom onset and TA injection, weeks | 21.2±6.0 | 18.9±3.2 | 0.477† |
| logMAR BCVA* | 0.71±0.32 | 0.73±0.36 | 0.898† |
| Central foveal thickness*, μm | 480.0±102.3 | 540.0±115.9 | 0.219† |

Data presented as mean±SD, where applicable. *Before TA injection. †Mann-Whitney U-test. ††Fisher's exact test. BCVA: Best-corrected visual acuity, logMAR: Logarithm of the minimal angle of resolution, TA: Triamcinolone acetonide, SD: Standard deviation

TA injection and at 1 month and 3 months after the injection was 0.73 ± 0.29, 0.77 ± 0.27, and 0.79 ± 0.26, respectively. The CFT was 547.0 ± 106.7 μm, 522.4 ± 108.3 μm, and 511.2 ± 94.0 μm, respectively. The BCVA and CFT measured after TA injection was not significantly changed when compared with the value before the injection ($P = 0.137$ and $P = 0.074$, respectively). No eye exhibited improvement in BCVA at 3 months.

### Adverse events

An increase in intraocular pressure was noted in four eyes (17.4%), which were subsequently treated with topical anti-glaucoma medication. Other complications, including endophthalmitis and retinal detachment, were not noted. Cataract progression was noted in one phakic eye.

## Discussion

In the present study, a significant decrease in macular edema was noted after intravitreal TA injection in eyes with macular edema secondary to RVO that was refractory to intravitreal bevacizumab therapy. One month after TA injection, a CFT decrease >150 μm or a CFT value <250 μm was noted in approximately 40% of eyes. Although overall improvement in visual acuity was not significant, visual acuity improvements were significant in eyes with a marked decrease in macular edema following TA therapy. Visual acuity improved by at least one line in approximately 33% of eyes at 1 month and 24% at 3 months, suggesting that TA therapy is effective in some patients.

The marked decrease in macular edema in some patients likely resulted from the distinct effect of TA, which is very different from the effects of anti-VEGF agents. The excellent efficacy of anti-VEGF therapy strongly indicates that VEGF plays an important causative role in the development of macular edema. However, various cytokines, including interleukin-6 (IL-6)[21,22] and IL-8,[23] have also been associated with macular edema in eyes with RVO. It is possible that the macular edema in our patients, who were refractory to intravitreal anti-VEGF therapy, had developed with mechanisms more closely associated to pathologic changes unrelated to VEGF. The TA injection can reduce IL-6[24,25] and IL-8[24] that cannot be modulated by anti-VEGF therapy.[24]

Another possible explanation is the time lag between anti-VEGF and TA injection. In the present study, an average of 5.8 weeks separated the last anti-VEGF injection and the TA injection. It is well-known that a spontaneous decrease in macular edema[26] and an improvement in visual acuity[27,28] can occur without intervention in eyes with RVO. To minimize the effect of this spontaneous improvement, patients with an interval between anti-VEGF and TA injections longer than 8 weeks were excluded from analyses. However, it is possible that some patients did have this spontaneous improvement in visual acuity in the 5.8 week interval period.

Jonas *et al*. previously reported the efficacy of intravitreal TA injection in treating the macular edema refractory to bevacizumab injection.[20] They observed a marked decrease in macular edema and a subsequent increase in visual acuity in 2 of 2 patients, who were administered 20 mg of intravitreal TA.[20] In the present study, we used a much smaller TA (4 mg), which may explain, at least in part, our much more limited efficacy.

In this study, several analyses were performed to determine factors predictive of 1 month CFT decrease after TA injection. Although we failed to verify any significant factors, this may have been because of our small sample size. One notable finding was the marked difference in the proportion of BRVO between the responsive group (56%) and the nonresponsive group (21.4%). The proportion of BRVO in the responsive group was almost twice as great as in the nonresponsive group, suggesting that TA injection may be more beneficial for patients with BRVO. Further studies with a larger study population are needed to confirm this postulation.

This study has several limitations, mainly due to its retrospective design and small sample size. The number of anti-VEGF injections before TA injection was also not controlled. Lastly, 3 months data were analyzed with approximately 74% of patients.

## Conclusion

Intravitreal TA injection was found to be beneficial in some patients with macular edema secondary to RVO refractory to bevacizumab therapy. At 1 month after TA injection lead to a marked reduction in macular edema in approximately 40% of patients, and 33% patients had an improvement in visual acuity. Therefore, this study suggests that intravitreal TA injection may be a treatment option for macular edema secondary to RVO refractory to intravitreal anti-VEGF therapy. However, deterioration in visual acuity and increase in retinal thickness between the 1–3 months follow-up period suggests the limited long-term efficacy of this therapy. Further studies with a larger study population and a longer follow-up are needed.

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