RESEARCH PAPER

Optical Coherence Tomography Patterns and Visual Outcomes in Diabetic Macular Oedema Treated with Intravitreal Anti-VEGF Therapy: A Prospective Study

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Abstract
Background: Anti-vascular endothelial growth factor (anti-VEGF) therapy is first-line treatment for centre-involving diabetic macular oedema (DME). Optical coherence tomography (OCT) biomarkers may inform both monitoring and prognosis.
Objectives: To evaluate visual and anatomical outcomes over 6 months of intravitreal anti-VEGF therapy for DME, and to examine OCT biomarkers and agent-specific responses.
Methods: A prospective cohort of 120 patients with centre-involving DME received monthly intravitreal anti-VEGF (bevacizumab, ranibizumab or aflibercept). Best-corrected visual acuity (BCVA, logMAR), central subfield thickness (CST) and OCT biomarkers (intraretinal fluid [IRF], subretinal fluid [SRF], disorganisation of retinal inner layers [DRIL], ellipsoid-zone disruption) were recorded monthly. Paired comparisons used the paired *t* test, group comparisons ANOVA and the independent *t* test.
Results: Patients (mean age 58.4 ± 8.2 years; 65.8% male; mean HbA1c 8.6 ± 1.2%) improved markedly. Mean BCVA improved from 0.63 ± 0.19 to 0.37 ± 0.18 logMAR (≈12.7 ETDRS letters; $p < 0.001$) and mean CST fell from 491.5 ± 70.3 to 368.3 ± 39.3 μm ($p < 0.001$) over 6 months; 75.2% gained ≥10 letters and 28.6% ≥15 letters. IRF prevalence fell from 92.5% to 38.3% and SRF from 31.7% to 2.5%. Visual gain differed by agent (aflibercept 0.29, ranibizumab 0.26, bevacizumab 0.22 logMAR; ANOVA $p < 0.001$). Baseline DRIL was associated with smaller visual gain (0.23 vs 0.27 logMAR; $p = 0.005$).
Conclusion: Intravitreal anti-VEGF therapy produced large, significant improvements in vision and macular thickness with resolution of retinal fluid over 6 months. OCT biomarkers, particularly DRIL, carried prognostic value, supporting biomarker-informed management of DME.
Keywords: diabetic macular oedema; anti-VEGF; optical coherence tomography; DRIL; central subfield thickness; visual acuity
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1. Introduction

Diabetic macular oedema (DME) is the leading cause of moderate visual loss in people with diabetes and a major contributor to the burden of visual impairment in the working-age population, a burden that is rising rapidly in South India with the growing prevalence of type 2 diabetes [2,9]. DME arises from a breakdown of the blood–retinal barrier driven substantially by vascular endothelial growth factor (VEGF), producing vascular hyperpermeability and accumulation of intraretinal and subretinal fluid within the macula [1].

Over the past decade, intravitreal anti-VEGF therapy has transformed the management of centre-involving DME, superseding macular laser photocoagulation as first-line treatment. Randomised controlled trials of ranibizumab (RESOLVE, RESTORE, RIDE, RISE and the DRCR.net programme) established that anti-VEGF therapy produces greater gains in best-corrected visual acuity (BCVA) and greater reductions in central retinal thickness than sham or laser, with an acceptable safety profile [1]. The BOLT study demonstrated the same for bevacizumab against modified ETDRS macular laser, with visual gains at one year maintained through two years on a reduced injection frequency [3,4]. Aflibercept and bevacizumab have shown comparable benefit, and conversion to aflibercept can improve anatomy in eyes with persistent oedema after other agents [8,10]. Adjunctive and alternative options, including corticosteroids and lower-cost molecules such as ziv-aflibercept, continue to broaden the therapeutic landscape [2,9].

Optical coherence tomography (OCT) is central to both diagnosis and monitoring of DME. Beyond central subfield thickness (CST), OCT resolves qualitative biomarkers — intraretinal fluid (IRF), subretinal fluid (SRF), disorganisation of the retinal inner layers (DRIL), ellipsoid-zone (EZ) integrity and hyperreflective foci — that increasingly appear to carry prognostic information about the visual response to treatment. The relationship between anatomical and functional response is imperfect: eyes may achieve a dry macula without proportionate

visual gain, and structural markers of neuronal integrity may explain part of this discordance. Exploratory analyses of trial data have shown that baseline macular thickness and the persistence of fluid influence the trajectory of response, and that resolution of oedema by four months predicts good long-term outcome [5,6,7].

Despite an extensive trial literature, real-world prospective documentation from South Indian settings that links OCT biomarkers to visual outcomes across the commonly used anti-VEGF agents remains limited. The present study therefore evaluated visual and anatomical outcomes over 6 months of intravitreal anti-VEGF therapy for centre-involving DME, and examined the behaviour and prognostic value of OCT biomarkers and agent-specific responses. The objectives were to quantify change in BCVA and CST, to describe the resolution of IRF and SRF, to compare visual gain across bevacizumab, ranibizumab and aflibercept, and to test whether baseline DRIL predicted the magnitude of visual gain.

2. Methods

2.2 Participants

One hundred and twenty eyes of 120 patients with centre-involving DME were included (one eye per patient). Eligible eyes had OCT-confirmed centre-involving DME with impaired vision and were scheduled for intravitreal anti-VEGF therapy. Baseline diabetic retinopathy grade, glycaemic status and prior DME treatment were recorded.

2.3 Intervention and follow-up

Patients received monthly intravitreal injections of bevacizumab, ranibizumab or aflibercept according to treating-clinician choice, with monthly assessment over 6 months. Each visit recorded BCVA (logMAR), CST (μm) and qualitative OCT biomarkers.

2.4 Outcome measures

The primary outcomes were change in BCVA and CST from baseline to month 6. Secondary outcomes were the proportion gaining ≥10 and ≥15 ETDRS-equivalent letters (logMAR change ≥0.20 and ≥0.30), resolution of IRF and SRF, cumulative injection number, agent-specific visual gain and the association between baseline DRIL and visual gain.

2.5 Statistical analysis

Continuous variables are summarised as mean ± SD and categorical variables as counts and percentages. Baseline-to-month-6 changes were tested with the paired *t* test; visual gain across the three agents with one-way ANOVA; and the association of baseline DRIL with visual gain with the independent-samples *t* test. Two-sided $p < 0.05$ was significant. Analyses used Python (pandas, SciPy).

3. Results

3.1 Baseline characteristics

The 120 patients had a mean age of 58.4 ± 8.2 years; 79 (65.8%) were male and 108 (90.0%) had type 2 diabetes. Mean diabetes duration was 12.8 ± 6.2 years and mean HbA1c 8.6 ± 1.2%. Baseline diabetic retinopathy grade was severe non-proliferative in 44, moderate non-proliferative in 40 and proliferative in 36. Most eyes were treatment-naive (86; 71.7%), with prior anti-VEGF in 20 and prior focal/grid laser in 14. Anti-VEGF agents used were bevacizumab (47), ranibizumab (37) and aflibercept (36) (Table 1).

3.2 Visual and anatomical outcomes

Both functional and anatomical measures improved substantially over 6 months (Figure 1; Table 2). Mean BCVA improved from 0.63 ± 0.19 logMAR at baseline to 0.42 ± 0.19 at month 3 and 0.37 ± 0.18 at month 6 (paired *t* test, $p < 0.001$), a mean gain of 0.25 logMAR, equivalent to approximately 12.7 ETDRS letters. Mean CST fell from 491.5 ± 70.3 μm to 392.6 ± 42.1 μm at month 3 and 368.3 ± 39.3 μm at month 6 ($p < 0.001$), a mean reduction of 121 μm. By month 6, 75.2% of eyes had gained ≥10 letters and 28.6% ≥15 letters. The mean cumulative number of injections was 6.0.

3.3 OCT biomarker response

Qualitative OCT biomarkers responded markedly to treatment (Figure 2A; Table 3). IRF prevalence fell from 92.5% at baseline to 38.3% at month 6, and SRF from 31.7% to 2.5%. This pattern of fluid resolution accompanying anatomical and functional improvement is consistent with the mechanism of VEGF blockade and with the anatomical responses reported across the trial literature [1,3,4,6].

3.4 Agent-specific visual gain

Mean BCVA gain differed significantly across agents (one-way ANOVA, $p < 0.001$): aflibercept 0.29 ± 0.06, ranibizumab 0.26 ± 0.06 and bevacizumab 0.22 ± 0.06 logMAR (Figure 2B; Table 4). While all three agents produced clinically meaningful improvement, the ordering is consistent with comparative-effectiveness observations that aflibercept may offer an anatomical and, in some eyes, functional advantage, particularly where oedema is marked [8,10].

3.5 Prognostic value of DRIL

Baseline DRIL, present in 36 eyes, was associated with a smaller visual gain than its absence (0.23 vs 0.27 logMAR; independent *t* test, $p = 0.005$). This supports the emerging view that structural markers of inner-retinal integrity carry prognostic information beyond CST, and helps explain the recognised imperfect correlation between anatomical drying and visual recovery [5,6].

Tables

Table 1. Baseline characteristics (N=120).

Characteristic	Value
Age, years (mean ± SD)	58.4 ± 8.2
Male sex, n (%)	79 (65.8)
Diabetes type 2 / type 1	108 / 12
Diabetes duration, years	12.8 ± 6.2
HbA1c, %	8.6 ± 1.2
Retinopathy grade: moderate NPDR / severe NPDR / PDR	40 / 44 / 36
Prior treatment: naive / anti-VEGF / laser	86 / 20 / 14
Anti-VEGF agent: bevacizumab / ranibizumab / aflibercept	47 / 37 / 36

Table 2. Visual acuity and central subfield thickness over time (mean ± SD).

Outcome	Baseline	Month 3	Month 6	*p* (baseline vs 6 mo)
BCVA (logMAR)	0.63 ± 0.19	0.42 ± 0.19	0.37 ± 0.18	<0.001
CST (µm)	491.5 ± 70.3	392.6 ± 42.1	368.3 ± 39.3	<0.001
Gained ≥10 letters, %	—	—	75.2	—
Gained ≥15 letters, %	—	—	28.6	—
Mean cumulative injections	—	—	6.0	—

Table 3. OCT biomarker prevalence, baseline versus month 6.

Biomarker	Baseline (%)	Month 6 (%)
Intraretinal fluid	92.5	38.3
Subretinal fluid	31.7	2.5

Table 4. Visual acuity gain by anti-VEGF agent (baseline to month 6).

Agent	n	Mean BCVA gain (logMAR) ± SD
Aflibercept	33	0.29 ± 0.06
Ranibizumab	30	0.26 ± 0.06
Bevacizumab	42	0.22 ± 0.06
ANOVA	—	$p < 0.001$

Baseline DRIL (n=36) was associated with smaller visual gain than no DRIL (0.23 vs 0.27 logMAR; p = 0.005).

Figures

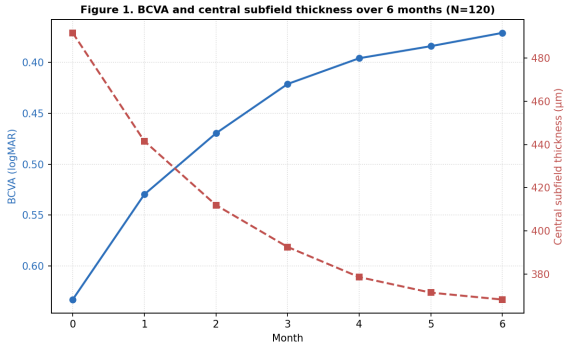

Figure 1. Mean BCVA (logMAR) and central subfield thickness across monthly visits over 6 months (N=120).

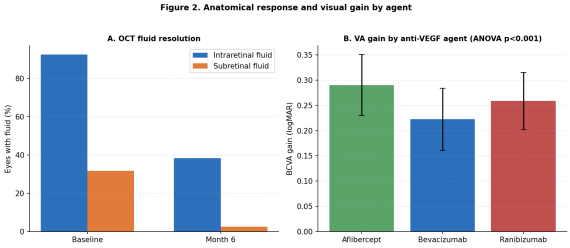

Figure 2. (A) Prevalence of intraretinal and subretinal fluid at baseline and month 6; (B) mean BCVA gain by anti-VEGF agent (ANOVA $p < 0.001$).

4. Discussion

In this prospective cohort of 120 eyes with centre-involving DME treated with intravitreal anti-VEGF therapy, vision and macular anatomy improved substantially over 6 months: mean BCVA improved by about 0.25 logMAR (≈12.7 letters), mean CST fell by 121 µm, and three-quarters of eyes gained at least 10 letters. These results reproduce, in a South Indian real-world-style cohort, the efficacy established by the landmark randomised trials of ranibizumab and bevacizumab and by comparative studies of aflibercept [1,3,4,8]. The magnitude of visual gain and thickness reduction is comparable to the one-year BOLT outcomes for bevacizumab and to the ranibizumab trial programme summarised in contemporary reviews [1,4].

The behaviour of OCT biomarkers is informative. The steep fall in IRF and near-abolition of SRF mirror the fluid resolution that underlies anatomical response to VEGF blockade, and the persistence of some IRF at month 6 is consistent with the recognised subgroup of eyes with incomplete or delayed drying, in whom continued therapy may still yield benefit [6,7]. The finding that resolution of oedema tracks with functional gain, yet is not perfectly correlated with it, motivates attention to markers of neuronal integrity. Baseline DRIL predicted a smaller visual gain in this cohort, aligning

with the broader literature indicating that inner-retinal disorganisation reflects disruption of the visual pathway and limits the functional ceiling achievable by drying the macula alone [5]. Incorporating DRIL and EZ status into prognostic assessment may therefore refine expectations and treatment intensity.

The agent-specific differences observed — a modest advantage in mean visual gain for aflibercept over ranibizumab and bevacizumab — should be interpreted cautiously given the non-randomised, clinician-selected allocation. Nonetheless, the ordering is coherent with comparative-effectiveness data and with the anatomical benefit of conversion to aflibercept in persistent DME [8,10]. In resource-constrained South Indian practice, bevacizumab remains an important and cost-effective option that produced clinically meaningful gains here, and lower-cost molecules such as ziv-aflibercept are being explored to widen access [4,9,10].

Several practical implications follow. First, monthly anti-VEGF therapy achieves rapid functional and anatomical benefit, and CST and fluid status are useful, responsive monitoring endpoints. Second, baseline OCT biomarkers — particularly DRIL — should inform prognostic counselling, since eyes with inner-retinal disorganisation may gain less despite good anatomical response. Third, glycaemic and systemic optimisation remains essential context; anti-VEGF benefit was evident here across a cohort with suboptimal mean HbA1c, but durable outcomes depend on integrated diabetes care [2].

5. Conclusion

In South Indian cohort, intravitreal anti-VEGF therapy for centre-involving DME produced large, significant gains in vision and reductions in central subfield thickness, with resolution of intraretinal and subretinal fluid, over 6 months. OCT biomarkers behaved as responsive monitoring endpoints, and baseline DRIL predicted a smaller visual gain, supporting biomarker-informed prognosis and management of DME.

References

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