ISSN 2524 – 0684 e-ISSN 2524 – 0692

UDC 617.735–007.23
IRSTI 76.29.56
DOI: 10.53065/kaznmu.2026.76.1.002

Received: 13.01.2026
Accepted for publication: 11.03.2026

EFFECTIVENESS OF INTRAVITREAL FARICIMAB IN PATIENTS WITH AGE–RELATED MACULAR DEGENERATION, DIABETIC MACULAR EDEMA, AND CENTRAL RETINAL VEIN OCCLUSION

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Abstract

Introduction. Neovascular age–related macular degeneration, diabetic macular edema, and macular edema secondary to retinal vein occlusion are leading causes of persistent central vision loss. Faricimab is the first bispecific intravitreal agent targeting both VEGF–A and Ang–2, potentially providing meaningful early anatomical and functional benefits in real–world practice.

Aim. To assess the real–world effectiveness and safety of faricimab (6 mg/0.05 mL) in patients with neovascular age–related macular degeneration (nAMD), diabetic macular edema (DME), and macular edema secondary to central retinal vein occlusion (CRVO).

Materials and Methods. Twelve treatment–naïve patients who had not previously received intravitreal therapy with vascular endothelial growth factor (VEGF) inhibitors were included: 4 patients in each cohort (nAMD, DME, and CRVO). All patients received four monthly intravitreal injections of faricimab (6 mg), with assessment of best–corrected visual acuity (BCVA) and central retinal thickness (CRT).

Results. OCT demonstrated a reduction in the severity of macular edema in all patients across all cohorts after the loading phase. In the overall group, BCVA improved from 0.23 ± 0.22 to 0.56 ± 0.34 ($p = 0.016$). CRT decreased from 503 ± 129 μm to 328 ± 76 μm ($p = 0.003$). No serious injection–related complications were observed.

Conclusion. After the loading phase, faricimab showed early signs of functional and anatomical improvement with a favorable safety profile.

Key words: Wet Macular Degeneration, Macular Edema, Diabetic Retinopathy, Retinal Vein Occlusion, Intraocular Injections.

Introduction. Neovascular age–related macular degeneration (nAMD), diabetic macular edema (DME), and macular edema secondary to central retinal vein occlusion (CRVO) are among the leading causes of persistent central vision loss and disability [1–4]. Their pathogenesis is driven by pathological vascular permeability, inflammation, and angiogenesis. The current standard of care relies on intravitreal therapy with inhibitors of vascular endothelial growth factor (VEGF). However, clinical outcomes are strongly influenced by the regularity of injections and frequent follow–up visits; in routine practice this often results in reduced adherence and undertreatment. Therefore, there remains a need for agents that can maintain comparable efficacy while potentially reducing the injection burden.

Faricimab is the first bispecific monoclonal antibody for intravitreal use that simultaneously inhibits VEGF–A and angiopoietin–2 (Ang–2), targeting not only VEGF–

ISSN 2524 – 0684 e-ISSN 2524 – 0692

driven leakage and neovascularization but also Ang–2–mediated vascular destabilization [1]. In pivotal trials in nAMD (TENAYA, LUCERNE), faricimab demonstrated non–inferior efficacy compared with aflibercept and enabled treat–and–extend intervals up to 16 weeks in a substantial proportion of patients while maintaining visual and anatomical outcomes [2]. Similarly, the YOSEMITE and RHINE trials in DME reported sustained functional and morphological improvements over 2 years with treat–and–extend dosing and extension of treatment intervals in many patients [3]. For RVO–related macular edema, 24– to 72–week results from BALATON and COMINO confirmed faricimab efficacy and an acceptable safety profile [4, 5].

Despite robust evidence from randomized trials, real–world data on faricimab use in the Republic of Kazakhstan are currently lacking. Within the clinical protocol of the Kazakh «Badge of Honour» Research Institute of Eye Diseases, faricimab has been introduced as a novel therapeutic option. The present study aimed to evaluate the effectiveness and safety of faricimab in treatment–naive patients with nAMD, DME, and CRVO, focusing on changes in visual acuity and OCT parameters. Given the limited sample size, this study should be considered a real–world observational case series aimed at generating preliminary clinical insights.

Materials and methods. This single–center prospective pilot study assessed the effectiveness and safety of intravitreal faricimab therapy in patients with macular edema of different etiologies. The study was conducted at the Kazakh «Badge of Honour» Research Institute of Eye Diseases LLP (Almaty, Kazakhstan). The follow–up duration for each patient was 7 months and included an active treatment phase of 4 months followed by 3 months of post–loading observation.

The study protocol was reviewed and approved by the Local Ethics Committee of the Kazakh Research Institute of Eye Diseases (№ 6 – 09.06.2025) and was conducted in accordance with the Declaration of Helsinki. All participants received full information regarding study objectives, procedures, potential risks and benefits, and alternative treatment options, and then provided written informed consent. Confidentiality principles were observed; personally identifiable data were not used in analysis or publication.

Three clinical cohorts were formed: nAMD, DME, and CRVO–related macular edema. Patients were recruited among those presenting to the institute with clinical indications for intravitreal anti–VEGF therapy.

Inclusion criteria were: (1) confirmed diagnosis of nAMD, DME, or CRVO with macular edema documented by clinical examination and multimodal imaging of the macula; (2) no prior intravitreal anti–VEGF therapy; (3) age criteria: ≥50 years for the nAMD cohort; and ≥18 years for the DME and CRVO cohorts.

Exclusion criteria included: (1) conditions likely to substantially affect functional prognosis or the safety of intravitreal procedures; (2) active ocular inflammatory disease (including uveitis or infection); (3) glaucoma; (4) advanced scar/atrophic macular changes; (5) pregnancy.

At baseline and follow–up visits, all patients underwent a standard ophthalmic examination including BCVA assessment, intraocular pressure measurement, slit–lamp biomicroscopy, dilated fundus examination, and macular evaluation using spectral–domain OCT and OCT angiography (OCTA) (SOLIX, Optovue Inc., Fremont, CA, USA).

OCT was used for quantitative assessment of central retinal thickness (CRT) and qualitative assessment of activity signs (presence/persistence of intraretinal and/or subretinal fluid and foveal contour). In patients with nAMD, OCTA was additionally performed to characterize neovascular activity.

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Effectiveness endpoints included changes in BCVA and CRT compared with baseline. The presence/persistence of fluid on OCT and response characteristics by cohort (nAMD, DME, CRVO) were also analyzed. Safety was assessed by the frequency and type of adverse events, including intraocular inflammation, intraocular pressure elevation, and other potential complications.

All participants received intravitreal faricimab at a dose of 6 mg/0.05 mL. The treatment regimen consisted of a loading phase of four monthly injections (4–week intervals). After the loading phase, patients were observed, and subsequent management was determined by the treating physician based on clinical and OCT signs of disease activity according to institutional routine practice.

All injections were performed in an operating room under strict aseptic conditions. Topical anesthesia and antiseptic preparation of the periocular skin and conjunctival sac were applied; the drug was administered via the pars plana, 3.5–4.0 mm posterior to the limbus.

Statistical analyses were performed using GraphPad Prism 10 (GraphPad Software Inc., San Diego, CA, USA). Quantitative data are presented as mean ± standard deviation (mean ± SD). Distribution normality was assessed using the Shapiro–Wilk test. Given the small sample size, statistical analysis was primarily descriptive. Changes in BCVA were analyzed for the overall cohort using the Wilcoxon matched–pairs signed rank test. Subgroup analyses were descriptive and no inferential statistics were applied due to limited statistical power. A p–value < 0.05 was considered statistically significant. Due to the exploratory nature of the study, patients with different etiologies of macular edema were included and analyzed both separately and descriptively as a combined cohort.

Results. Twelve patients were included and allocated into three cohorts of 4 patients each: nAMD, DME, and CRVO–related macular edema. In the nAMD cohort, mean symptom duration was 1 year ± 4 months (n = 4). In the DME cohort, mean symptom duration was 3 ± 2 months
(n = 4), and in the CRVO cohort, 7 days ± 4 days (n = 4).

In the nAMD cohort, OCT/OCTA identified type 1 macular neovascularization in 2/4 (50%), type 2 in 1/4 (25%), and type 3 in 1/4 (25%) patients. In the CRVO cohort, 1 patient (25%) had the ischemic type and 3 patients (75%) had the non–ischemic type.

In the overall cohort, BCVA increased significantly from 0.23 ± 0.22 to 0.56 ± 0.34 ($p = 0.016$) compared with baseline, with a mean gain of +0.33 ± 0.27 (Table 1). Improvements were observed across all cohorts: BCVA improved by $\Delta = 0.13 \pm 0.04$ in nAMD, by $\Delta = 0.41 \pm 0.27$ in DME; and $\Delta = 0.38 \pm 0.35$ in CRVO.

Table 1. Changes in best–corrected visual acuity (decimal) from baseline to after four loading injections (Wilcoxon matched–pairs signed rank test for overall cohort only)

Parameter (mean ± SD)	Baseline	After 4 loading injections	Difference	P–value
nAMD	0.37 ± 0.47	0.50 ± 0.42	0.13 ± 0.04	n/a
DME	0.19 ± 0.18	0.60 ± 0.40	0.41 ± 0.27	n/a
CRVO	0.17 ± 0.03	0.55 ± 0.37	0.38 ± 0.35	n/a
Overall	0.23 ± 0.22	0.56 ± 0.34	0.33 ± 0.27	0.016*

* Statistical significance

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OCT demonstrated a reduction in macular edema severity in all patients across all cohorts after completion of the loading phase (12/12; 100%). In the overall cohort, CRT decreased after completion of the loading phase compared with baseline (from 503 ± 129 μm to 328 ± 76 μm, p = 0.003, with mean decrease by 175 ± 98 μm; Table 2). CRT decrease in the DME, CRVO, and nAMD cohorts were Δ = 156 ± 74, 255 ± 109, and 114 ± 52, respectively. Given the limited sample size in each subgroup, these findings should be interpreted as descriptive. Representative OCT images by indication are described in the figure legends (Figures 1–3).

Table 2. Changes in central retinal thickness (μm) from baseline to after four loading injections (Wilcoxon matched–pairs signed rank test for overall cohort only)

Parameter (mean ± SD)	**Baseline**	**After 4 loading injections**	**Difference**	**P–value**
nAMD	412 ± 86	298 ± 54	114 ± 52	n/a
DME	487 ± 102	331 ± 71	156 ± 74	n/a
CRVO	611 ± 118	356 ± 95	255 ± 109	n/a
Overall	503 ± 129	328 ± 76	175 ± 98	0.003*

* Statistical significance

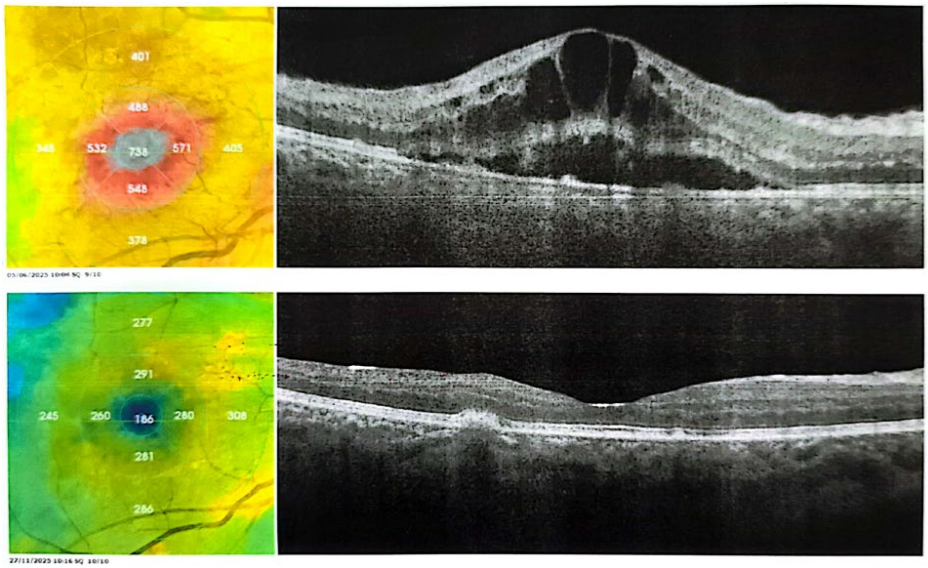

Figure 1. A 19–year–old woman with diabetic macular edema. At baseline (top), OCT shows pronounced cystoid macular edema with increased central retinal thickness (thickness map on the left). After the loading phase of faricimab (bottom), a marked anatomical response is observed with regression of cystoid spaces and normalization of the foveal contour, along with reduced central retinal thickness. Functionally, BCVA improved from 0.4 to 1.0

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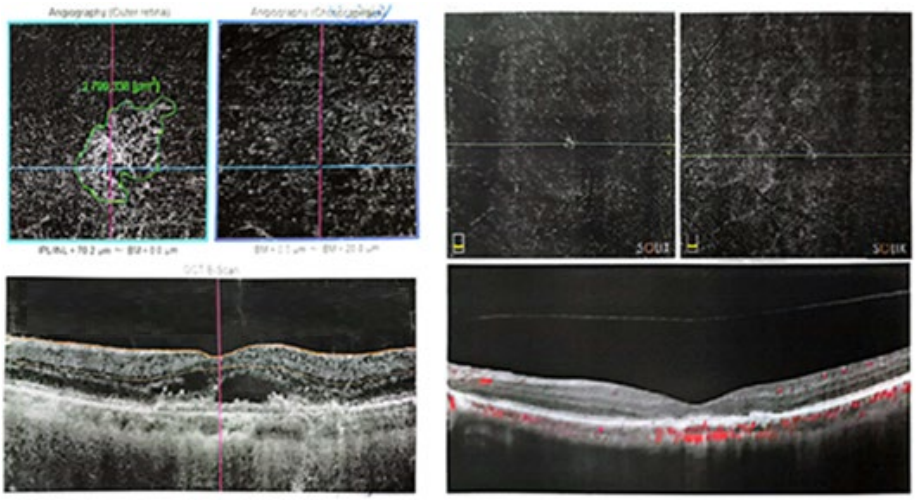

Figure 2. A 57–year–old patient with nAMD with active type 2 choroidal neovascularization. Left: OCTA demonstrates a neovascular complex in the macular area with activity on structural OCT. Right: after the loading phase of faricimab, OCT shows favorable anatomical dynamics with reduced exudation/fluid and a more stable foveal contour, accompanied by a marked regression of choroidal neovascularization. Functionally, BCVA improved from 0.02 to 0.2

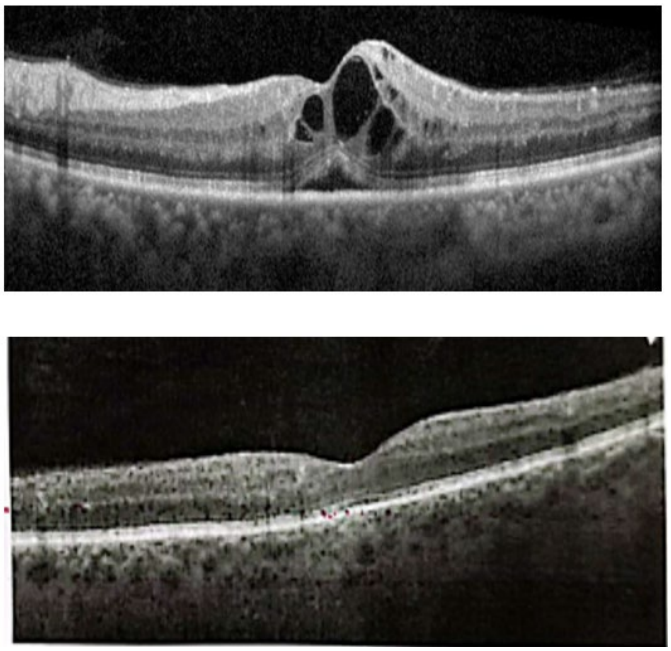

Figure 3. A 78–year–old patient with macular edema secondary to CRVO. Baseline OCT (top) demonstrates pronounced cystoid macular edema with deformation of the foveal contour. After the loading phase of faricimab (bottom), a marked positive anatomical response is observed with substantial regression of cystoid spaces, restoration of a more physiological foveal contour, and reduced exudation. Functionally, BCVA improved from 0.2 to 0.9

No complications associated with intravitreal injections and/or faricimab were observed in study participants.

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Discussion. This single–center prospective study demonstrated early functional and anatomical improvement after four monthly intravitreal faricimab injections (6 mg/0.05 mL) in treatment–naive patients with nAMD, DME, and CRVO–related macular edema. In the overall cohort, BCVA improved significantly (0.23 ± 0.22 to 0.56 ± 0.34; p = 0.016), and OCT showed a reduction in macular edema in all cohorts. The response pattern differed by disease entity, which is clinically expected.

In our study, mean BCVA in the CRVO cohort increased from 0.17 ± 0.03 to 0.55 ± 0.37 (Δ = 0.38 ± 0.35) after the loading phase. In routine clinical practice, the non–ischemic (perfused) variant of CRVO is more common (approximately 70%) than the ischemic variant (approximately 30%), which was comparable to our cohort structure (75% vs 25%) [6]. This distinction is critical for interpreting functional response. In non–ischemic CRVO, preserved foveal perfusion provides greater functional potential, and BCVA gains after intravitreal therapy are often driven by rapid resolution of edema and partial restoration of foveal architecture [7, 8]. In ischemic CRVO, functional outcomes may remain limited despite a good anatomical response due to irreversible foveal damage. Notably, the single patient with ischemic CRVO in our series had baseline BCVA of 0.3 but decreased to 0.03 after the loading phase despite the absence of macular edema on OCT.

Timely treatment of DME can yield favorable anatomical and functional outcomes, as reflected in our series, where mean BCVA in the DME cohort improved from 0.19 ± 0.18 to 0.60 ± 0.40 (Δ = 0.41 ± 0.27) after the loading phase [9]. A likely contributor to the pronounced early response was the relatively short mean symptom duration in the DME group (3 ± 2 months).

The advantage of early initiation of DME therapy is linked to the fact that, at earlier stages, visual impairment is largely driven by a reversible exudative component [10, 11]. Post hoc analyses of Protocol I suggest that persistent macular edema of at least 6 months' duration is associated with a higher likelihood of sustained microstructural changes [12, 13]. With prolonged edema, breakdown of the blood–retinal barrier and Muller cell dysfunction persist, local inflammation and tissue hypoxia are maintained, and chronic fluid accumulation may mechanically disrupt the neurosensory retina and damage photoreceptors [14].

At the same time, in the nAMD cohort, the improvement in BCVA was more modest (from 0.37 ± 0.47 to 0.50 ± 0.42; Δ = 0.13 ± 0.04). One of the key factors limiting the functional response in nAMD is the development of subretinal fibrosis (fibrovascular scarring), which is considered a typical end–stage outcome of macular neovascular activity and is associated with persistent central vision loss [2, 15–17]. Reviews emphasize that the rate of subretinal fibrosis formation is highest during the first 12 months, after which the progression typically slows [18–20]. According to meta–analytic estimates, signs of fibrosis are present at the time of first symptom onset in approximately 13% of patients, and its prevalence increases to 32% by month 12 and to 36% by month 24 of follow–up, which helps explain why, at later stages of disease, the potential for BCVA improvement may be limited even with effective suppression of exudation. Patients often underestimate early symptoms such as metamorphopsia, «blurred vision», and reduced contrast sensitivity, compensate with the better–seeing eye, and seek medical attention only after a substantial decline in vision, which likely contributes to delayed presentation (1 year ± 4 months from the onset of symptoms) and the functional outcomes observed in our study.

This study has several important limitations. The small sample size substantially limits statistical power and generalizability. Therefore, the findings should be interpreted as preliminary and hypothesis–generating rather than confirmatory. The inclusion of three distinct disease entities (nAMD, DME, and CRVO) represents a source of clinical heterogeneity.

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Therefore, the pooled analysis should be interpreted with caution and considered descriptive rather than indicative of a uniform treatment effect across different pathologies.

Conclusion. Faricimab therapy (6 mg/0.05 mL) in patients with nAMD, DME, and macular edema due to retinal vein occlusion was associated with an early reduction of macular edema on OCT and improvement in BCVA, with a favorable safety profile. This prospective pilot study suggests that intravitreal faricimab may provide early anatomical and functional improvement in patients with nAMD, DME, and CRVO–related macular edema; however, the findings should be interpreted cautiously.

Conflict of interest. The authors declare no conflict of interest.

Author contributions. Concept and design, N.A., E.K., L.O., K.R. Clinical work and data collection, E.K., L.O., K.R., D.K., A.D. Data analysis, K. R., A.D. Scientific interpretation, N.A., L.O., K.R., Z.U. Manuscript writing, L.O., K.R., Z.U. All authors have read and agreed to the published version of the manuscript.

Funding. The study was performed within the framework of the grant application «Analysis of the effectiveness of intravitreal faricimab injections in patients with age–related macular degeneration, diabetic macular edema, and central retinal vein occlusion» and was funded by internal resources of the Kazakh «Badge of Honour» Research Institute of Eye Diseases LLP.

Data availability statement. The data supporting the findings of this study are contained within the article. Additional data are available from the corresponding author upon reasonable request.

Acknowledgments. The authors have nothing to acknowledge.

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ISSN 2524 – 0684 e-ISSN 2524 – 0692

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ISSN 2524 – 0684 e-ISSN 2524 – 0692

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НАҚТЫ КЛИНИКАЛЫҚ ТӘЖІРИБЕДЕ ФАРИСИМАБТЫҢ (6 МГ/0,05 МЛ) ТИІМДІЛІГІ МЕН ҚАУІПСІЗДІГІН НЕОВАСКУЛЯРЛЫ ЖАСҚА БАЙЛАНЫСТЫ МАКУЛЯРЛЫҚ ДЕГЕНЕРАЦИЯСЫ, ДИАБЕТТІК МАКУЛЯРЛЫҚ ІСІНУІ ЖӘНЕ ОРТАЛЫҚ ТОРҚАБЫҚ ВЕНАСЫНЫҢ

ISSN 2524 – 0684 **e-ISSN 2524 – 0692**

ТРОМБОЗЫ КЕЗІНДЕГІ МАКУЛЯРЛЫҚ ІСІНУІ БАР ПАЦИЕНТТЕРДЕ БАҒАЛАУ

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Түйіндеме

Кіріспе. Неоваскулярлы жасқа байланысты макулярлық дегенерация, диабеттік макулярлық ісіну және торқабық венасының окклюзиясына байланысты макулярлық ісіну орталық көрудің тұрақты төмендеуінің негізгі себептерінің бірі болып табылады. Фарисимаб – VEGF–A және Ang–2–ні бір мезгілде тежейтін алғашқы биспецификалық препарат, бұл нақты клиникалық тәжірибеде айқын анатомиялық және функционалдық әсер беруі мүмкін.

Мақсаты. Неоваскулярлы жасқа байланысты макулярлық дегенерациясы (нЖБМД), диабеттік макулярлық ісінуі (ДМІ) және торқабық венасының тромбозына байланысты макулярлық ісінуі (ТВТ) бар пациенттерде фарисимабтың (6 мг/0,05 мл) тиімділігі мен қауіпсіздігін бағалау.

Материалдар мен әдістер. Зерттеуге бұрын тамырлық эндотелий өсу факторының тежегіштерімен (ТЭӨФТ) интравитреалды ем алмаған 12 пациент қатысты: әр когортада 4 пациенттен (нЖБМД, ДМІ, ОТВТ). Барлық пациентке Фарисимабтың (6 мг) төрт ай қатарынан ай сайын интравитреалды инъекциясы жасалды; ең жоғары түзетілген көру өткірлігі (ЕЖТКӨ) және орталық торқабық қалыңдығы (ОТҚ) бағаланды.

Нәтижелер. ОКТ деректері бойынша жүктемелік фазадан кейін барлық топтардағы барлық пациенттерде макулярлық ісінудің айқындылығы төмендеді. Жалпы топта ЕЖТКӨ 0,23 ± 0,22–ден 0,56 ± 0,34–ке дейін артты (p = 0,016). Торқабықтың орталық қалыңдығы 503 ± 129 мкм–ден до 328 ± 76 мкм–ге дейін төмендеді (p = 0,003). Инъекцияларға байланысты асқынулар тіркелмеді.

Қорытынды. Жүктемелік фазадан кейін Фарисимаб функционалдық және анатомиялық жақсарудың ерте белгілерін көрсетті және қауіпсіздік профилі қолайлы болды.

Түйінді сөздер: ылғалды жасқа байланысты макулярлық дегенерация, макулярлық ісіну, диабеттік ретинопатия, торқабық венасының окклюзиясы, көз ішіне инъекция.

АНАЛИЗ ЭФФЕКТИВНОСТИ ИНТРАВИТРЕАЛЬНЫХ ИНЪЕКЦИЙ ФАРИСИМАБА У ПАЦИЕНТОВ С ВОЗРАСТНОЙ МАКУЛОДИСТРОФИЕЙ, ДИАБЕТИЧЕСКИМ МАКУЛЯРНЫМ ОТЕКОМ И ТРОМБОЗОМ ЦЕНТРАЛЬНОЙ ВЕНЫ СЕТЧАТКИ

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ISSN 2524 – 0684 **e-ISSN 2524 – 0692**

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Аннотация

Введение. Неоваскулярная возрастная макулярная дегенерация (нВМД), диабетический макулярный отек (ДМО) и макулярный отек при тромбозе вены сетчатки (ТВС) остаются частыми причинами стойкого снижения центрального зрения и требуют регулярной анти–VEGF терапии. Фарисимаб – первый биспецифический препарат, одновременно блокирующий VEGF–A и Ang–2, что может усилить и стабилизировать анатомо–функциональный ответ в реальной клинической практике.

Цель. Оценить эффективность и безопасность Фарисимаба (6 мг/0,05 мл) в реальной клинической практике у пациентов с нВМД, ДМО и макулярным отеком при ТВС.

Материалы и методы. В исследовании участвовали 12 пациентов, ранее не получавших интравитреальной терапии ингибиторами фактора роста сосудистого эндотелия: по 4 пациента в каждой когорте (нВМД, ДМО, ТВС). Всем выполнено четыре ежемесячные интравитреальные инъекции Фарисимаба (6 мг) с оценкой максимально корригированной остроты зрения (МКОЗ) и центральной толщины сетчатки (ЦТС).

Результаты. По данным ОКТ после загрузочной фазы у всех пациентов во всех когортах отмечено уменьшение выраженности макулярного отека. В общей когорте МКОЗ увеличилась с 0,23 ± 0,22 до 0,56 ± 0,34 (p = 0,016). Центральная толщина сетчатки уменьшилась с 503 ± 129 мкм до 328 ± 76 мкм (p = 0,003). Серьезных осложнений, связанных с инъекциями, не зарегистрировано.

Заключение. После загрузочной фазы Фарисимаб продемонстрировал ранние признаки функционального и анатомического улучшения при благоприятном профиле безопасности.

Ключевые слова: влажная возрастная макулярная дегенерация, макулярный отек, диабетическая ретинопатия, окклюзия вен сетчатки, внутриглазные инъекции.