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One-year Outcomes and Early Prognostic Factors in Young-onset Proliferative Diabetic Retinopathy

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Abstract

Objectives

To evaluate 1-year visual and structural outcomes after initial treatment of young-onset proliferative diabetic retinopathy (PDR) and to identify early predictors of poor visual outcome and progression to pars plana vitrectomy (PPV).

Methods

This retrospective cohort study included patients aged ≤ 40 years with newly diagnosed PDR and at least 12 months of follow-up. Longitudinal changes in best-corrected visual acuity (BCVA), metabolic parameters, PDR severity, and optical coherence tomography-based structural features were assessed. Poor visual outcome was defined as BCVA logMAR ≥ 1.0 at 1 year. Multivariable analyses accounting for inter-eye correlation were performed. In eyes without tractional retinal detachment (TRD) at baseline, predictors of progression to PPV were evaluated using Kaplan–Meier and Cox proportional hazards models.

Results

Fifty-two patients (101 eyes; mean age, 31.6 ± 5.2 years) were included. Glycaemic control improved significantly over 1 year (haemoglobin A1c, 9.1 ± 2.2% to 7.5 ± 1.5%, P < 0.001), whereas mean BCVA showed no significant change. PDR severity and arcade-involving fibrovascular proliferation (FVP) decreased significantly, whereas retinal and choroidal thickness remained unchanged. Macula-involving TRD independently predicted poor visual outcome at 1 year. Among TRD-free eyes, arcade-involving FVP not only independently predicted progression to PPV (adjusted OR, 9.71; 95% CI, 2.96–31.80) but was also associated with a significantly earlier time to vitrectomy (adjusted HR, 5.95; 95% CI, 2.03–17.48).

Conclusions

In young-onset PDR, visual prognosis is primarily determined by macula-involving TRD, whereas arcade-involving FVP identifies eyes at high risk of early surgical intervention prior to TRD development.

INTRODUCTION

Diabetic retinopathy is widely recognised as the most prevalent microvascular manifestation of diabetes mellitus (DM) and remains a leading cause of vision impairment worldwide [1–5]. Among the stages of diabetic retinopathy, proliferative diabetic retinopathy (PDR) represents the most advanced and vision-threatening form. It is characterised by abnormal retinal neovascularisation and fibrovascular tissue formation driven by retinal ischaemia [6–8]. These changes frequently lead to serious complications,

including vitreous haemorrhage and tractional retinal detachment (TRD), which may result in permanent visual loss despite appropriate treatment [6,9].

As the age at diabetes onset continues to decrease, an increasing number of patients receive a diagnosis of PDR at a younger age [2,4,10]. PDR in younger individuals, often presenting in the third or fourth decade of life, tends to progress more rapidly and aggressively, with more extensive fibrovascular proliferation (FVP) and a higher risk of tractional complications [11–13]. These patients may reach advanced stages of diabetic retinopathy after a relatively short duration of diabetes and often require surgical treatment earlier in the disease course, raising concern regarding long-term visual outcomes [14–16].

Despite these distinctive features, most previous PDR studies have focused on older populations or mixed-age cohorts, with limited emphasis on disease characteristics and outcomes in young-onset PDR. Consequently, data on post-treatment clinical outcomes and prognostic factors in young patients with PDR remain scarce. Few studies have systematically distinguished factors that determine visual prognosis from those that drive progression to surgical intervention.

In this study, we aimed to evaluate 1-year clinical outcomes in young patients with PDR following initial treatment and to identify prognostic factors associated with visual outcomes and disease progression. Specifically, the study sought to distinguish predictors of progression to pars plana vitrectomy (PPV) among the eyes without TRD from determinants of poor visual prognosis in the overall cohort, thereby providing a stage-specific understanding of disease progression in young-onset PDR during the early post-diagnostic period.

METHODS

Study design and population

This retrospective cohort study included consecutive patients aged ≤ 40 years who were newly diagnosed with PDR and received initial treatment between January 2020 and December 2024 at a single tertiary referral centre in Korea. Eligibility required at least 12 months of clinical follow-up after the initial diagnosis of PDR.

Eyes were excluded if they had a history of prior retinal surgery, coexisting retinal diseases unrelated to DM (e.g., retinal vein occlusion, uveitis, or hereditary retinal disorders), or insufficient clinical or imaging data for reliable assessment. When both eyes of a patient met the inclusion criteria, data from both were included in the analysis, and inter-eye correlation was accounted for using appropriate statistical methods.

The study received approval from the Institutional Review Board of Yonsei University Severance Hospital (IRB No. 4-2025-1376) and followed the tenets of the Declaration of Helsinki. The requirement for informed consent was waived.

Data collection and definitions

Baseline demographic and systemic variables were collected from medical records, including sex, ages at PDR and DM diagnoses, duration of DM, DM treatment status, regular ophthalmic follow-up for diabetic retinopathy, and systemic comorbidities, such as hypertension and chronic kidney disease. Laboratory data, including haemoglobin A1c (HbA1c) and serum creatinine levels, estimated glomerular filtration rate, lipid profile, and body mass index, were also recorded when available.

Ocular variables included baseline best-corrected visual acuity (BCVA), presence of preretinal haemorrhage, vitreous haemorrhage, and macular oedema, as well as detailed features of PDR severity. PDR severity was subclassified into early, high-risk, and advanced stages using an operational definition informed by established clinical severity frameworks and treatment guidelines for diabetic retinopathy [6,8,9]. Early PDR was defined as the presence of retinal neovascularisation without high-risk characteristics. High-risk PDR was defined according to the Early Treatment Diabetic Retinopathy Study criteria [6]. Advanced PDR was defined as PDR complicated by extensive FVP, recurrent vitreous or preretinal haemorrhage, or TRD. Eyes initially classified as high-risk or advanced PDR that demonstrated resolution of active neovascularisation and fibrovascular activity after treatment were categorised as having regressed PDR rather than being reclassified as early PDR, reflecting changes in disease activity rather than stage regression.

The extent of FVP was graded according to the number of quadrants involved (0–4 quadrants), and arcade-involving FVP was recorded separately. The classification of FVP extent and arcade involvement was performed during chart review according to predefined operational criteria based on fundus examination and multimodal imaging findings and was determined by retinal specialists (THK and JJK). TRD was defined as any fibrovascular traction associated with retinal elevation on clinical examination or optical coherence tomography (OCT). Macula-involving TRD was defined as TRD involving or threatening the fovea, as confirmed by fundus examination and/or optical coherence tomography (OCT). TRD-free eyes were defined as the eyes without TRD at baseline.

Ophthalmic examinations and treatment

All patients underwent comprehensive ophthalmic examinations at baseline and during follow-up visits, including BCVA measurement, intraocular pressure measurement, slit-lamp biomicroscopy, and dilated fundus examination. Multimodal imaging included spectral-domain OCT, wide-field fundus photography, and wide-field fluorescein angiography.

Treatment modalities during the follow-up period were recorded, including panretinal photocoagulation, intravitreal anti-vascular endothelial growth factor (VEGF) injections, and PPV. The number of anti-VEGF injections during the first year was documented for descriptive purposes. Indications for PPV typically include non-clearing vitreous haemorrhage, TRD threatening or involving the macula, or progressive FVP despite adequate laser or medical therapy, at the discretion of the treating retinal specialist.

Outcome measures

The primary endpoint was poor visual outcome at 1 year, defined as BCVA of logMAR ≥ 1.0 (Snellen equivalent ≤ 20/200). The cutoff of logMAR ≥ 1.0 was selected to denote severe visual impairment. This level of visual acuity corresponds to the commonly accepted threshold for legal blindness in clinical and regulatory frameworks and represents a degree of vision loss associated with substantial functional disability [17,18].

Secondary endpoints included longitudinal changes in BCVA, alterations in retinal structural parameters (central retinal thickness and subfoveal choroidal thickness), temporal changes in PDR severity and the extent of FVP, and progression to PPV within 1 year. In a prespecified subgroup analysis, data from the TRD-free eyes were analysed separately to identify baseline factors associated with progression to PPV during the first year of follow-up.

Statistical analysis

Continuous variables were presented as mean ± standard deviation and were compared using paired t-tests or Wilcoxon signed-rank tests, as appropriate. Categorical variables were compared using the chi-square test, Fisher's exact test, or McNemar test, as appropriate.

Univariate analyses were first performed to screen factors associated with poor visual outcome at 1 year, and variables with $P < 0.05$ were considered for entry into multivariable models. To account for inter-eye correlation, multivariable logistic regression was performed using generalised estimating equations with an exchangeable working correlation structure. To minimise multicollinearity and overadjustment, parsimonious models were constructed using clinically representative variables from distinct domains. Variables reflecting overlapping aspects of disease severity (e.g., advanced PDR type and overall extent of FVP) were not simultaneously included in the multivariable models to minimise collinearity with traction-related structural features. The final models included baseline BCVA, vitreous haemorrhage, macula-involving TRD, and arcade-involving FVP for visual outcome, and baseline BCVA, vitreous haemorrhage, and arcade-involving FVP for PPV in the TRD-free eyes.

In the TRD-free eyes, time-to-event analyses for PPV within 1 year were performed using Kaplan–Meier curves with log-rank tests and Cox proportional hazards models adjusted for baseline BCVA and vitreous haemorrhage.

All analyses were two-sided, with a significance level of $P < 0.05$, and were conducted using SPSS Statistics (version 29; IBM Corp., Armonk, NY, USA). Missing data were handled using available-case analysis.

RESULTS

Study population and baseline characteristics

The study included 52 patients (101 eyes) with young-onset PDR. The mean age at PDR diagnosis was 31.6 ± 5.2 years, and 42 patients (80.8%) had type 2 DM. Notably, 30.8% of patients were not receiving any diabetes-related treatment at the time of PDR diagnosis, and 17.3% received a diagnosis of DM only after presenting with PDR. Baseline glycaemic control was poor, with a mean HbA1c level of 9.1 ± 2.2%. The mean duration of DM at presentation was 89.8 ± 95.0 months, and 32.7% of patients underwent regular retinal screening at the time of PDR diagnosis. Systemic comorbidities included hypertension (30.8%) and chronic kidney disease (9.6%).

At presentation, a substantial proportion of eyes already demonstrated advanced structural disease activity, including vitreous haemorrhage (42.6%), preretinal haemorrhage (46.5%), arcade-involving FVP (41.6%), and macula-involving TRD (14.0%). During the first year of follow-up, 66 of 101 eyes (65.3%) received panretinal photocoagulation, 87 of 101 eyes (86.1%) received intravitreal anti-VEGF therapy, and 53 of 101 eyes (52.5%) received combined panretinal photocoagulation and anti-VEGF treatment. The mean number of intravitreal anti-VEGF injections during the first year was 3.6 ± 2.5 (median, 3 [IQR 1–5]). Most eyes received standard-of-care treatment for PDR during follow-up. Baseline demographic, systemic, and ocular characteristics appear in Table 1.

Changes in visual acuity and systemic metabolic control

At the eye level, mean BCVA (logMAR) did not change significantly from baseline to 1 year (0.6 ± 0.8 vs. 0.5 ± 0.7, P = 0.171). In contrast, at the patient level, systemic glycaemic control improved significantly during the same period, with the mean HbA1c levels decreasing from 9.1 ± 2.2% to 7.5 ± 1.5% (P < 0.001). These longitudinal changes in visual acuity and systemic metabolic parameters are shown in Table 2.

Longitudinal changes in PDR severity and structural features

Over the 1-year follow-up period, significant longitudinal changes were observed in PDR severity and fibrovascular characteristics. The proportion of eyes classified as advanced PDR decreased markedly (from 39.6% at baseline to 14.9% at 1 year, P < 0.001). The proportion of eyes with regressed PDR increased substantially from 0.0% to 33.7% at 1 year (P < 0.001). In contrast, the proportion of high-risk PDR did not change significantly (26.7% vs. 25.7%, P = 1.000).

The extent of FVP also showed significant temporal changes. The proportion of eyes without quadrant involvement increased at 1 year (46.5% to 58.4%, P = 0.043), whereas the eyes with involvement of all four quadrants showed a decreasing trend (15.8% to 7.9%, P = 0.057). In addition, the prevalence of FVP involving the vascular arcades decreased significantly over time (41.6% to 27.7%, P = 0.009). Macular oedema was present in 58.7% of cases at baseline, decreasing non-significantly to 48.9% (P = 0.108), while no significant longitudinal changes were observed in central retinal thickness (P = 0.839) or subfoveal choroidal thickness (P = 0.235) (Table 2). Despite the observed regression in proliferative activity, mean visual acuity remained largely unchanged during the same period, indicating a discordance between structural improvement and functional visual outcomes.

Determinants of 1-year visual prognosis

Visual prognosis was categorised using a threshold of logMAR ≥ 1.0 at 1 year. In univariate analyses, several baseline factors were associated with poor visual outcome, including worse baseline BCVA (odds ratio [OR], 3.67; 95% confidence interval [CI], 1.74–7.73; P < 0.001), presence of preretinal haemorrhage (OR, 11.91; 95% CI, 2.53–56.09; P = 0.002) or vitreous haemorrhage (OR, 14.03; 95% CI, 2.87–68.71; P = 0.001), macula-involving TRD (OR, 15.13; 95% CI, 3.93–58.23; P < 0.001), more advanced PDR type (OR, 4.23; 95% CI, 1.62–11.04; P = 0.003), greater extent of FVP (OR per quadrant, 1.86; 95% CI, 1.27–2.72; P = 0.001), and arcade-involving FVP (OR, 8.19; 95% CI, 1.92–34.88; P = 0.004).

In the multivariable generalised estimating equation logistic regression model accounting for inter-eye correlation, macula-involving TRD was the only factor independently associated with poor visual outcome at 1 year (adjusted OR, 5.40; 95% CI, 1.06–27.58; P = 0.043; Table 3). Other baseline variables did not retain statistical significance after adjustment.

Determinants of early vitrectomy in the TRD-free eyes

To evaluate disease progression prior to the development of TRD, a prespecified subgroup analysis was performed in eyes without TRD at baseline, using PPV within 1 year as the outcome of interest. In univariate analyses, several baseline ocular factors were associated with progression to vitrectomy among the TRD-free eyes, including worse baseline BCVA (OR, 3.67; 95% CI, 1.35–9.94; P = 0.011), presence of preretinal haemorrhage (OR, 3.67; 95% CI, 1.26–10.68; P = 0.017) or vitreous haemorrhage (OR, 4.62; 95% CI, 1.68–12.72; P = 0.003), more advanced PDR type (OR, 5.51; 95% CI, 2.28–13.32; P < 0.001), greater extent of FVP (OR per quadrant, 2.71; 95% CI, 1.61–4.54; P < 0.001), and arcade-involving FVP (OR, 13.78; 95% CI, 4.42–42.98; P < 0.001). In the multivariable generalised estimating equation model, arcade-involving FVP emerged as the only factor independently associated with PPV within 1 year among the TRD-free eyes (adjusted OR, 9.71; 95% CI, 2.96–31.80; P < 0.001; Table 4).

Kaplan–Meier survival analysis further demonstrated a marked difference in PPV-free survival according to the presence of arcade-involving FVP. Eyes with arcade-involving FVP showed significantly earlier progression to vitrectomy, compared with those without arcade involvement (log-rank test, P < 0.0001; Figure 1).

Consistent with these findings, Cox proportional hazards analysis adjusted for baseline BCVA and vitreous haemorrhage confirmed that arcade-involving FVP was independently associated with earlier progression to PPV in the TRD-free eyes (adjusted hazard ratio, 5.95; 95% CI, 2.03–17.48). Neither baseline BCVA nor vitreous haemorrhage was independently associated with time to vitrectomy after adjustment.

DISCUSSION

This study provides a comprehensive evaluation of early clinical outcomes and prognostic determinants in young patients with PDR following initial treatment. By analysing visual outcomes in the overall cohort together with progression to surgery in the eyes without TRD, the findings indicate that baseline fibrovascular features primarily correlate with the need for early surgery, whereas the presence of TRD serves as the main determinant of 1-year visual outcomes. Unlike most previous studies that focused mainly on post-surgical outcomes, this study integrates early structural and functional outcomes after initial treatment with the risk of progression to surgery prior to the development of TRD in young-onset PDR. In young-onset PDR, irreversible retinal damage may begin at an early stage of disease, even before TRD becomes clinically apparent. Accordingly, the 1-year follow-up period should be interpreted as an early assessment of prognosis rather than as an indicator of long-term visual outcomes.

Young-onset PDR often follows a rapid and aggressive course and may remain clinically silent until sudden visual decline occurs [14,15,19]. Compared with older individuals, younger individuals tend to exhibit more extensive FVP, which increases the risk of TRD [11–13]. Experimental and clinical studies suggest greater activity of fibrotic pathways in young-onset PDR alongside angiogenic signalling [20–22]. Previous studies have reported age-related differences in intraocular cytokine profiles in PDR, with relatively lower VEGF levels and higher levels of connective tissue growth factor in younger patients [20]. Although the present study did not directly assess these molecular findings, they provide a plausible biological context for the pronounced fibrotic phenotype observed in young-onset PDR. In addition, the relative absence of posterior vitreous detachment in younger individuals may further augment tractional forces, allowing fibrovascular tissue to use the vitreous as a scaffold and predisposing these eyes to early traction-related complications.

Over the first year after diagnosis, multiple structural indicators of disease activity showed significant regression. The proportion of eyes classified as having advanced PDR declined, whereas early-stage PDR and eyes without active neovascularisation became more prevalent. In parallel, the extent of FVP decreased, including a significant reduction in arcade-involving FVP. Despite these anatomical improvements, central retinal thickness, subfoveal choroidal thickness, and visual acuity remained largely unchanged. This suggests that reduction in neovascular or fibrovascular activity does not necessarily lead to visual improvement once substantial structural damage has occurred. In young-onset PDR, foveal damage associated with the mechanical behaviour of fibrovascular tissue—such as contraction, adhesion to the vitreous, and the generation of TRD—may influence visual function more than the degree of neovascular regression alone. Therefore, improvement in proliferative findings should not be interpreted as evidence that traction-related structural changes have been reversed.

Systemic metabolic control improved substantially during the first year of follow-up, as reflected by a marked decline in HbA1c levels. However, this improvement did not accompany meaningful gains in visual acuity. Many young patients experience prolonged periods of poor or fluctuating glycaemic control before diagnosis, during which retinal ischaemia and fibrovascular remodelling may progress silently. Once proliferative changes advance to extensive FVP or TRD, macular architecture may already undergo alterations that limit the potential for visual recovery. In this context, post-diagnostic improvement in

glycaemic control may not translate into short-term visual improvement, not because metabolic control lacks importance, but because structural damage has already accumulated.

When visual outcomes were categorised using a clinically meaningful cutoff, macula-involving TRD was the only factor independently associated with poor visual outcome at 1 year. Several baseline indicators of disease activity, including baseline visual acuity, were associated with worse vision in univariate analyses; however, these associations weakened after adjustment for TRD. This pattern suggests that many conventional severity markers influence visual outcomes largely through their relationship with TRD. Once TRD involves the macula, retinal structure may undergo permanent distortion, with disruption of photoreceptor alignment that limits the potential for visual recovery. In this context, the apparent role of baseline visual acuity likely reflects a close association with traction-related structural damage rather than an independent influence on short-term visual prognosis.

Among the eyes without TRD at baseline, FVP involving the vascular arcades independently predicted progression to vitrectomy within the first year. This finding highlights the importance of lesion location, rather than overall disease stage alone, in anticipating the need for early surgical intervention before overt TRD becomes clinically apparent. Arcade involvement likely represents a location where fibrovascular tissue more readily exerts both anteroposterior and tangential traction, promotes recurrent vitreous haemorrhage, and facilitates firm vitreoretinal adhesion, thereby increasing the likelihood of surgical intervention. The time-to-event analyses supported this association, showing earlier and more frequent progression to vitrectomy in eyes with arcade-involving FVP.

Overall, these findings indicate that the location and extent of FVP play an important role in determining the need for surgery before TRD develops, whereas macula-involving TRD largely determines early visual prognosis. From a clinical perspective, young patients with arcade-involving FVP may benefit from closer follow-up and earlier consideration of intervention, even in the absence of clinically apparent TRD. The 1-year outcomes in this study should be interpreted as an early window of prognostic assessment, during which traction-related irreversible damage may already be developing.

This study has some limitations. The retrospective design and modest sample size, particularly in subgroup analyses, may limit statistical power and result in wide CIs. Treatment strategies were not standardised and may reflect confounding by indication. In addition, direct measurements of intraocular cytokines and detailed characterisation of the vitreoretinal interface were not available. Although time-to-event analyses were performed at the eye level, residual within-patient correlation cannot be completely excluded. Finally, the study was conducted at a tertiary referral centre within a single country, which may limit generalisability. Extended follow-up is necessary to determine whether early structural markers identified in this study predict visual and surgical outcomes over longer periods.

In young-onset PDR, macula-involving TRD was the main factor associated with poor visual outcome at 1 year, whereas FVP involving the vascular arcades independently predicted progression to vitrectomy in the eyes without baseline TRD. The time-to-event analyses showed that arcade-involving FVP was associated with earlier surgical intervention, supporting its role as an early risk marker for progression to

surgery before the development of TRD. The absence of visual improvement despite better metabolic control highlights the dominant impact of traction-related structural damage in advanced disease and suggests that glycaemic improvement alone is unlikely to restore vision once tractional changes have developed. Together, these findings emphasise the importance of early identification of high-risk structural features and timely strategies to prevent TRD during the early post-diagnostic period in young-onset PDR. These findings also suggest that structural features such as arcade-involving FVP may serve as candidate markers for future risk stratification models in young-onset PDR. Prospective studies will be necessary to determine whether early identification of these features can guide surveillance strategies or earlier intervention aimed at preventing tractional complications.

Declarations

CONFLICT OF INTEREST

The authors declare no competing interests.

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AUTHOR CONTRIBUTIONS

THK, YJK, JL, CSL, SHB, SSK and JJK made substantial contributions to the conception and design of the work. THK and JJK collected and analysed the data. THK and JJK interpreted the data. THK drafted and all the authors substantively revised the paper.

DATA AVAILABILITY STATEMENT

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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Tables

Table 1. Baseline characteristics of the study cohort (patient- and eye-level)

Characteristic	Value
Patients, n	52
Eyes, n	101
Sex (M:F)	27 (51.9%):25 (48.1%)
Age at PDR diagnosis (y)	31.6 ± 5.2
Age at DM diagnosis (y)	24.1 ± 9.8
First diagnosis of DM after PDR diagnosis	9 (17.3%)
No DM treatment	16 (30.8%)
DM duration (mo)	89.8 ± 95.0
Regular diabetic retinopathy screening	17 (32.7%)
Type 2 DM	42 (80.8%)
Hypertension	16 (30.8%)
Chronic kidney disease	5 (9.6%)
HbA1c (%)	9.1 ± 2.2
Creatinine (mg/dL)	1.1 ± 1.0
Triglyceride (mg/dL)	199.8 ± 243.1
Total cholesterol (mg/dL)	195.2 ± 56.3
BMI (kg/m^2)	26.3 ± 4.2
Baseline BCVA (logMAR)	0.6 ± 0.8
Vitreous haemorrhage (eye-level)	43 (42.6%)
Preretinal haemorrhage (eye-level)	47 (46.5%)
Macula-involving TRD (eye-level)	14 (14.0%)
Macular oedema (OCT; eye-level)	57 (60.0%)
FVP involving vascular arcades (eye-level)	42 (41.6%)

Values are presented as mean ± standard deviation or n (%). Patient-level variables are summarised per patient (N = 52), and eye-level variables are summarised per eye (N = 101).

M, male; F, female; PDR, proliferative diabetic retinopathy; DM, diabetes mellitus; HbA1c, haemoglobin A1c; BMI, body mass index; BCVA, best-corrected visual acuity; TRD, tractional retinal detachment; OCT, optical coherence tomography; FVP, fibrovascular proliferation

Table 2. Longitudinal changes from baseline to 1 year after initial treatment

Characteristic	Baseline	1 y	P
BCVA (logMAR)	0.6 ± 0.8	0.5 ± 0.7	0.171
HbA1c (%)	9.1 ± 2.2	7.5 ± 1.5	< 0.001
Macular oedema	54 (58.7%)	45 (48.9%)	0.108
CRT (μm)	271.4 ± 107.9	268.2 ± 117.5	0.839
SFCT (μm)	315.1 ± 80.9	311.0 ± 84.3	0.235
Arcade-involving FVP	42 (41.6%)	28 (27.7%)	0.009
PDR type			
Early	34 (33.7%)	26 (25.7%)	0.002
High-risk	27 (26.7%)	26 (25.7%)	1.000
Advanced	40 (39.6%)	15 (14.9%)	< 0.001
Regressed	0 (0.0%)	34 (33.7%)	< 0.001
FVP quadrants involved			
0	47 (46.5%)	59 (58.4%)	0.043
1	16 (15.8%)	17 (16.8%)	1.000
2	12 (11.9%)	10 (9.9%)	0.815
3	10 (9.9%)	7 (6.9%)	0.581
4	16 (15.8%)	8 (7.9%)	0.057

Paired t-tests or Wilcoxon signed-rank tests were used for continuous variables, and the McNemar test was used for paired categorical variables. McNemar tests compared each category with all other categories combined.

BCVA, best-corrected visual acuity; HbA1c, haemoglobin A1c; CRT, central retinal thickness; SFCT, subfoveal choroidal thickness; FVP, fibrovascular proliferation; PDR, proliferative diabetic retinopathy

Table 3. Prognostic factors for poor visual outcome (BCVA logMAR ≥ 1.0) at 1 year

Variable	Univariate OR (95% CI)	P	Multivariable OR (95% CI)	P
DM-related variables				
Age at DM diagnosis (y)	1.01 (0.95–1.07)	0.765		
DM duration (mo)	1.00 (0.99–1.00)	0.753		
No DM treatment	3.12 (0.86–11.29)	0.083		
Regular DM follow-up	0.74 (0.20–2.73)	0.646		
First diagnosis of DM after PDR diagnosis	1.82 (0.56–5.89)	0.318		
DM type	1.86 (0.40–8.56)	0.425		
HbA1c	1.00 (0.74–1.35)	0.994		
Other systemic variables				
Sex	0.47 (0.10–2.15)	0.331		
HTN	1.50 (0.40–5.59)	0.544		
CKD	2.59 (0.43–15.51)	0.296		
BMI	0.89 (0.75–1.05)	0.171		
Cr	1.34 (0.72–2.47)	0.354		
TC	1.00 (0.98–1.01)	0.823		
TG	1.00 (1.00–1.00)	0.245		
HDL	1.02 (0.98–1.07)	0.353		
LDL	1.01 (0.99–1.02)	0.284		
Ocular variables				
Baseline BCVA (logMAR)	3.67 (1.74–7.73)	<0.001	1.06 (0.42–2.66)	0.900
Macula-involving TRD	15.13 (3.93–58.23)	<0.001	5.40 (1.06–27.58)	0.043
PRH	11.91 (2.53–56.09)	0.002		
VH	14.03 (2.87–68.71)	0.001	5.09 (0.55–46.87)	0.151
PDR type	4.23 (1.62–11.04)	0.003		
FVP extent	1.86 (1.27–2.72)	0.001		
Arcade-involving FVP	8.19 (1.92–34.88)	0.004	1.53 (0.25–9.51)	0.646
ME	1.21 (0.33–3.51)	0.341		

ERM	3.47 (0.82–14.73)	0.092
CRT	1.00 (1.00–1.01)	0.092
SFCT	1.01 (1.00–1.01)	0.178

Logistic regression with generalised estimating equations (exchangeable correlation structure) was used to account for inter-eye correlation. Variables reflecting overlapping disease severity were not entered simultaneously into multivariable models to minimise multicollinearity.

BCVA, best-corrected visual acuity; OR, odds ratio; CI, confidence interval; DM, diabetes mellitus; PDR, proliferative diabetic retinopathy; HbA1c, haemoglobin A1c; HTN, hypertension; CKD, chronic kidney disease; BMI, body mass index; Cr, creatinine; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TRD, tractional retinal detachment; FVP, fibrovascular proliferation; PRH, preretinal haemorrhage; VH, vitreous haemorrhage; ME, macular oedema; ERM, epiretinal membrane; CRT, central retinal thickness; SFCT, subfoveal choroidal thickness

Table 4. Predictors of pars plana vitrectomy within 1 year among the TRD-free eyes at baseline

Variable	Univariate OR (95% CI)	P	Multivariable OR (95% CI)	P
DM-related variables				
Age at DM diagnosis (y)	1.00 (0.95–1.05)	0.887		
DM duration (mo)	0.99 (0.99–1.00)	0.104		
No DM treatment	1.41 (0.52–3.79)	0.497		
Regular DM follow-up	1.11 (0.38–3.22)	0.847		
First diagnosis of DM after PDR diagnosis	0.45 (0.11–1.91)	0.280		
DM type	1.92 (0.32–7.21)	0.522		
HbA1c	1.11 (0.84–1.46)	0.470		
Other systemic variables				
Sex	0.92 (0.31–2.73)	0.882		
HTN	0.46 (0.14–1.52)	0.202		
CKD	1.99 (0.68–5.82)	0.207		
BMI	1.00 (0.87–1.15)	0.967		
Cr	0.91 (0.54–1.54)	0.734		
TC	1.00 (0.99–1.01)	0.394		
TG	1.00 (1.00–1.00)	0.342		
HDL	0.98 (0.94–1.02)	0.289		
LDL	0.99 (0.98–1.01)	0.851		
Ocular variables				
Baseline BCVA (logMAR)	3.67 (1.35–9.94)	0.011	1.57 (0.52–4.74)	0.424
PRH	3.67 (1.26–10.68)	0.017		
VH	4.62 (1.68–12.72)	0.003	1.18 (0.31–4.48)	0.807
PDR type	5.51 (2.28–13.32)	< 0.001		
FVP extent	2.71 (1.61–4.54)	< 0.001		
Arcade-involving FVP	13.78 (4.42–42.98)	< 0.001	9.71 (2.96–31.80)	< 0.001
ME	1.74 (0.61–5.00)	0.303		

ERM	4.53 (0.83–12.99)	0.096
CRT	1.00 (0.99–1.00)	0.789
SFCT	0.99 (0.99–1.00)	0.611

TRD-free eyes were defined as eyes without tractional retinal detachment at baseline.

TRD, tractional retinal detachment; OR, odds ratio; CI, confidence interval; DM, diabetes mellitus; PDR, proliferative diabetic retinopathy; HbA1c, haemoglobin A1c; HTN, hypertension; CKD, chronic kidney disease; BMI, body mass index; Cr, creatinine; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BCVA, best-corrected visual acuity; PRH, preretinal haemorrhage; VH, vitreous haemorrhage; FVP, fibrovascular proliferation; ME, macular oedema; ERM, epiretinal membrane; CRT, central retinal thickness; SFCT, subfoveal choroidal thickness

Figures

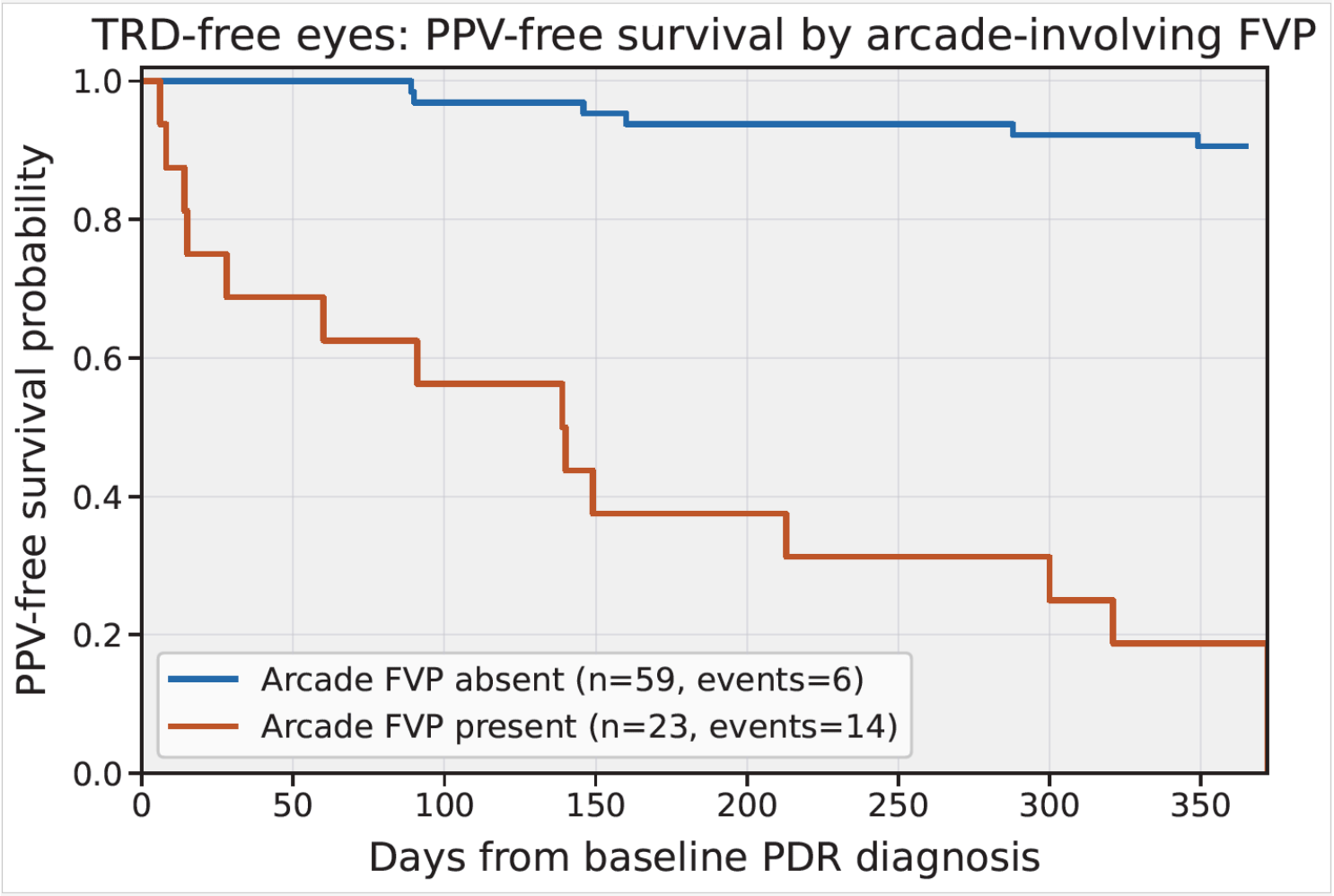


Figure 1

Kaplan–Meier curves for PPV-free survival in tractional retinal detachment-free eyes stratified by the presence of arcade-involving FVP

Eyes with arcade-involving FVP demonstrate significantly shorter time to PPV, compared with those without arcade involvement (log-rank $P < 0.0001$). The adjusted hazard ratio for arcade-involving FVP is 5.95 (95% confidence interval, 2.03–17.48) after adjustment for baseline best-corrected visual acuity and vitreous haemorrhage.

PPV, pars plana vitrectomy; PDR, proliferative diabetic retinopathy; FVP, fibrovascular proliferation