• Clinical Research •

Nomogram-based prediction of postoperative proliferative vitreoretinopathy following scleral buckling surgery for rhegmatogenous retinal detachment

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Abstract

● **AIM:** To identify independent risk factors for postoperative proliferative vitreoretinopathy (PVR) in patients with primary rhegmatogenous retinal detachment (RRD) treated with scleral buckling surgery and to develop a nomogram for predicting postoperative PVR.

● **METHODS:** Patients who underwent scleral buckling surgery for primary RRD were retrospectively enrolled. Patients were randomly assigned to a training cohort (*n*=515) and a validation cohort (*n*=55). Candidate variables included demographic characteristics, systemic comorbidities, preoperative ocular status, and retinal break features. Independent predictors of postoperative PVR were identified using univariate and multivariate logistic regression analyses. A nomogram was constructed to predict the risk of PVR at 1, 3, and 6mo after surgery. Model performance was evaluated using the concordance index (C-index), receiver operating characteristic (ROC) curves, calibration plots, and decision curve analysis (DCA).

● **RESULTS:** A total of 570 eyes were included, with mean age of 52.65±15.70y, and 51.4% of patients were male. Postoperative PVR occurred in 28.8% of eyes overall, including 28.5% in the training cohort and 30.9% in the validation cohort. Postoperative PVR developed in a subset of patients during follow-up. Preoperative PVR, a history of diabetes mellitus, and more than four retinal breaks were identified as independent risk factors for postoperative PVR. In the training cohort, the C-indices of the nomogram at 1, 3, and 6mo were 0.888, 0.931, and 0.948, respectively, and 0.885, 0.885, and 0.909 in the validation cohort. ROC and calibration analyses demonstrated good discrimination and agreement, while DCA showed favorable net clinical benefit across a wide range of threshold probabilities.

● **CONCLUSION:** Preoperative PVR, diabetes mellitus, and more than four retinal breaks are independent predictors of postoperative PVR after scleral buckling for RRD. The proposed nomogram provides accurate individualized risk prediction at 1, 3, and 6mo postoperatively, and may assist clinicians in postoperative surveillance and decision-making.

● **KEYWORDS:** nomogram; proliferative vitreoretinopathy; rhegmatogenous retinal detachment; retinal breaks; risk factors

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INTRODUCTION

Rhegmatogenous retinal detachment (RRD) is a critical eye disorder that may result in blindness without prompt treatment[1]. Various treatments, such as pars plana vitrectomy (PPV), pneumatic retinopexy, and scleral buckling (SB), have demonstrated favorable outcomes[2]. Historically, SB was the preferred method for repairing retinal detachment (RD). The rates of retinal reattachment are between 93.2% and 99.4% in cases of uncomplicated RRD[3-5].

Even after successful RRD repair, medium- and long-term proliferative vitreoretinopathy (PVR) complications

can arise, hindering functional recovery and necessitating additional medical or surgical intervention. PVR is a frequent complication following successful repair of RRD[6-8].

Postoperative PVR incidence following RRD repair varies widely from 3% to 43%[9-12], frequently delaying visual recovery, with a peak occurrence between 4 and 12wk[13-15].

Prior investigations using regression analyses have indicated that a variety of clinical factors influence the likelihood of PVR. These include conditions of the vitreous, such as hemorrhage and preoperative opacity, the degree of detachment, and the size and number of retinal breaks[16-18]. However, the results of these studies are often inconsistent.

Despite substantial research efforts, data on postoperative PVR following SB remain limited. Because the vitreous body is preserved and the surgical techniques differ markedly between SB and PPV, the mechanisms underlying postoperative PVR in SB-treated patients may be distinct. To identify potential risk factors more effectively, this study aimed to develop a reliable and practical approach for predicting the likelihood of PVR development. Nomograms are commonly used clinical tools for predicting tumor survival and can be used to assess patients' mortality and recurrence rates[19]. As an easy-to-master graphical interface, the nomogram provides an intuitive visual presentation for clinical decision-making, and simultaneously displays complex mathematical formulas graphically[20]. Here, risk factors linked to the development of PVR following surgical correction of RRD were assessed. In addition, a nomogram for predicting the likelihood of PVR at various time points after surgery was constructed.

PARTICIPANTS AND METHODS

Ethical Approval In compliance with the Declaration of Helsinki and authorized by the hospital's Medical Ethics Committee, a retrospective study was carried out at the Second Affiliated Hospital of Harbin Medical University from February 1, 2022, to August 31, 2025 (YJSKY2023-194). Every participant provided their informed consent.

Participants The inclusion criteria comprised eyes with noncomplex primary RRD, either macula-on or macula-off, as defined by Adelman *et al*[21]. This definition encompassed cases with single or multiple retinal breaks, PVR grades A, B, and C1, unclear retinal break status (defined as an invisible break on preoperative examination), and minimal vitreous hemorrhage that did not interfere with retinal visualization.

Exclusion criteria included PVR grades C2, C3, and D, giant retinal tears, retinal dialysis, traumatic RRD, tractional or combined tractional RRD, exudative RD, full-thickness macular holes (FTMH), and coexisting ocular conditions that could affect best-corrected visual acuity (BCVA), including lens opacification. Additional exclusions were prior intraocular surgery other than uncomplicated cataract extraction, high myopia with posterior staphyloma, and/or myopic traction maculopathy. The diagnostic criteria for PVR adhered to the Retina Society's 1991 classification[22].

Data Extraction Patient data were recorded over a six-month follow-up, including demographics (age, gender, affected eye), medical history (hypertension, diabetes), refractive status, symptom onset time, prior retinal laser photocoagulation, lens condition, presence of preoperative PVR, vitreous hemorrhage, retinal breaks (number and type), pre- and post-operative visual acuity and intraocular pressure, extent of RD, SB procedure details, and intraocular filling materials used. According to the results of fundus examination after SB, especially optical coherence tomography (OCT; Heidelberg, Eye 1.10.2.0, GPVRany), the occurrence of PVR in patients was evaluated.

Surgical Techniques All patients with RRD underwent primary SB surgery performed by the same experienced surgeon under an operating microscope. A 240-model circumferential silicone band (Beijing Jingcheng Chuangye Medical Device Co., Ltd.) was used in all cases. Retrobulbar anesthesia was administered routinely. Following conjunctival peritomy and exposure of the sclera, the silicone band was placed circumferentially, and subretinal fluid drainage was performed when indicated. Cryotherapy was routinely and uniformly applied to all identified retinal breaks and adjacent degenerative areas using carbon dioxide until a visible whitening response was achieved. The buckle was secured with appropriate tension to produce mild scleral indentation, and intravitreal air injection was performed when necessary. Conjunctival closure was completed in a standard fashion.

Statistical Analysis Independent samples *t*-test was used to compare continuous variables with normal distributions, which are displayed as mean±standard deviation. Rank-sum tests were used to compare variables having non-normal distributions, which were presented as median (Q1, Q3). Chi-square tests or, if inappropriate, Fisher's exact tests are used to analyze count data, which are displayed as frequency (%). Logistic and stepwise regression analyses were used to identify risk factors for postoperative PVR. Factors identified as significant in the multivariate analysis were used to construct a nomogram to predict the likelihood of no PVR at 1, 3, and 6mo after surgery. The model was established in the training cohort before validation in the validation cohort. Data were analyzed using SPSS 24.0 and R version 4.4.1. Receiver operating characteristic (ROC) curves and Harrell's concordance index (C-index) were used to assess the nomogram's predictive accuracy, and decision curve analysis (DCA) was used to determine its clinical effectiveness. *P*-values less than 0.05 were considered statistically significant.

RESULTS

Participant Characteristics Overall, 663 cases of RRD

were initially recruited. After screening and excluding cases that did not meet the criteria, 570 were ultimately enrolled, of whom 515 and 55 were randomly allocated to the training and validation cohorts, respectively. In the training cohort, 86.6% of the patients were under 70 years old, male (49.3%), 11.7% had type 2 diabetes, and 15.5% had hypertension. The cohorts were similar in terms of participant characteristics (Table 1). After surgery, 147 patients (28.5%) ultimately developed PVR; among them, 116 (78.9%) had preoperative PVR, while 31 (21.1%) did not (Table 2).

Univariate and Multivariate Logistic Regression Univariate analysis of the training cohort indicated significant associations between six characteristics and postoperative PVR. These were diabetes, hypertension, high myopia, preoperative retinal photocoagulation, preoperative PVR, and the number of retinal breaks (P<0.001; Table 3). However, as shown in Figure 1, the multivariate analysis revealed that only diabetes, preoperative PVR, and the number of retinal fractures were substantially correlated with postoperative PVR.

Nomogram Construction This nomogram included three independent risk factors for postoperative posterior vitreous detachment identified by multivariate logistic regression. Figure 2 presents the performance of this nomogram in the training set for predicting postoperative PVR probability at 1, 3, and 6mo after surgery. Summation of the scores for each variable enabled convenient calculation of the individual probability of postoperative PVR detachment at different time points.

Nomogram Validation The nomogram's accuracy in predicting postoperative PVR was good in both external and internal validation. While in the training cohort, the C-indices for PVR prediction at 1, 3, and 6mo after surgery were 0.888, 0.931, and 0.948, respectively, whereas those in the validation group were 0.885, 0.885, and 0.909, respectively. The model's strong predictive accuracy was validated by the high consistency of the calibration curves across time points, demonstrating a strong correlation between the predicted and actual probabilities (Figure 3).

Decision Curve Analysis As shown in Figure 4, at 1, 3, and 6mo after surgery, the DCA curves for both sets indicated significant clinical decision-making value across most threshold probabilities, indicating a significant net benefit.

The model's discriminative performance was assessed using the ROC area under the curve (AUC).

The threshold probability is shown on the X-axis, while the net benefit rate is shown on the Y-axis. The horizontal line represents all negative and untreated samples with a net benefit of zero. The slanted curve displays positive values in every sample, and the net benefit forms a backslash with a downward slope.

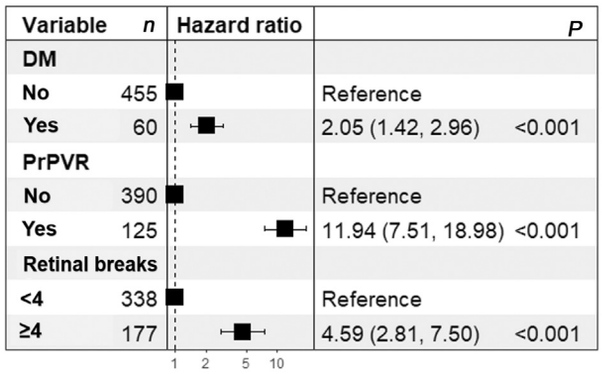

Figure 1 Forest plot of the multivariate regression results in the training cohort DM: Diabetes mellitus; PrPVR: Preoperative proliferative vitreoretinopathy.

DISCUSSION

PVR secondary to retinal repair surgery not only causes problems such as blurred vision, visual distortion, and decreased visual acuity in patients after surgery, but also imposes economic and psychological burdens[23-25]. This study comprehensively considered various clinical characteristics and patients' demographic features. Multivariate analysis identified diabetes, preoperative PVR, and multiple retinal breaks as significant risk factors, leading to the construction of a model for predicting the risk of PVR following SB surgery. This model is expected to assist with clinical diagnosis, formulate treatment plans, and evaluate patients' prognosis.

To the best of our knowledge, the proposed nomogram represents the first predictive model specifically designed to estimate the risk of PVR following SB in patients with primary RRD. In 2005, Pastor *et al*[26] developed a statistical model to assess PVR risk in patients undergoing retinal repair surgery. Although this model demonstrated improved sensitivity and specificity compared with earlier studies[27-28], the optimal values reached only 78.0% and 75.6%, respectively. Subsequently, Wickham *et al*[17] proposed a simplified formula based on preoperative clinical parameters to predict PVR after vitrectomy in primary RRD, achieving an area under the ROC curve of 0.86. However, the sensitivity and specificity of these models remained insufficient for clinical application.

In the present study, the nomogram achieved a C-index of 0.9 or higher in both internal and external validation, indicating excellent discriminatory performance. Furthermore, the calibration curves confirmed the model's accuracy and robustness. These findings suggest that the proposed nomogram reliably predicts the probability of PVR at 1, 3, and 6mo following SB in patients with RD. Importantly, the predictive performance and reliability of our model are comparable to those reported by Gao *et al*[29], who developed a nomogram for predicting PVR after PPV in patients with

Table 1 Baseline demographic and clinical features of the training and validation cohorts *n* (%) or mean±SD

Variables	Total (*n*=570)	Training cohort (*n*=515)	Validation cohort (*n*=55)	*P*
Age (y)	52.65±15.70	53.11±15.64	51.38±14.71	0.268
Gender				0.290
Female	277 (48.6%)	254 (49.3%)	23 (41.8%)	
Male	293 (51.4%)	261 (50.7%)	32 (58.2%)	
Eye side				0.298
Right eye	348 (61.1%)	318 (61.7%)	30 (54.5%)	
Left eye	222 (38.9%)	197 (38.3%)	25 (45.5%)	
Diabetes	75 (13.2%)	60 (11.7%)	15 (27.3%)	0.001
Hypertension	94 (16.5%)	80 (15.5%)	14 (25.5%)	0.060
High myopia	108 (18.9%)	87 (16.9%)	21 (38.2%)	0.001
Retinal photocoagulation	61 (10.7%)	56 (10.9%)	5 (9.1%)	0.684
Lens condition				0.435
Cataract	161 (28.2%)	131 (25.4%)	30 (54.5%)	
Intraocular lens	38 (6.7%)	35 (6.8%)	3 (5.5%)	
Preop. PVR	138 (24.2%)	125 (24.3%)	13 (23.6%)	0.917
PVR (A)	0	0	0	
PVR (B)	108 (18.9%)	97 (18.8%)	11 (20.0%)	
PVR (C1)	30 (5.3%)	28 (5.4%)	2 (3.6%)	
Preop. vitreous hemorrhage	49 (8.6%)	40 (7.8%)	9 (16.4%)	0.143
Number of retinal breaks				0.002
<4	424 (74.4%)	382 (74.2%)	42 (76.4%)	
≥4	146 (25.6%)	133 (25.8%)	13 (23.6%)	
Morphology of retinal breaks				0.305
Horseshoe-shaped tear	340 (59.6%)	311 (60.4%)	29 (52.7%)	
Atrophic hole	136 (23.9%)	123 (23.9%)	13 (23.6%)	
Mixed hole	94 (16.5%)	81 (15.7%)	13 (23.6%)	
Suprachoroidal fluid drainage	546 (95.8%)	495 (96.1%)	51 (92.7%)	0.234
RD extension				0.246
1 quadrant	294 (51.6%)	268 (52.0%)	26 (47.3%)	
2 quadrants	246 (43.2%)	221 (42.9%)	25 (45.5%)	
3 quadrants	30 (5.2%)	26 (5.1%)	4 (7.2%)	
Preop. CD				0.611
Yes	38 (6.7%)	35 (6.8%)	3 (5.5%)	
No	532 (93.3%)	480 (93.2%)	52 (94.5%)	
RTS				0.416
<1 PD	491 (86.1%)	443 (86.0%)	48 (87.3%)	
≥1 PD, <2 PD	79 (13.9%)	72 (14.0%)	7 (12.7%)	
TOS (d)				0.358
I (1-7)	462 (81.1%)	417 (81.0%)	45 (81.8%)	
II (8-30)	85 (14.9%)	77 (15.0%)	8 (14.5%)	
III (31-90)	21 (3.7%)	19 (3.6%)	2 (3.6%)	
IV (>90)	2 (0.3%)	2 (0.4%)	0	
Intraoperative air filling	434 (76.1%)	389 (75.5%)	45 (81.8%)	0.299

PVR: Proliferative vitreoretinopathy; CD: Choroidal detachment; RD: Retinal detachment; RTS: Retinal tear size; PD: The diameter of the optic papilla; TOS: Time from onset to surgery; SD: Standard deviation.

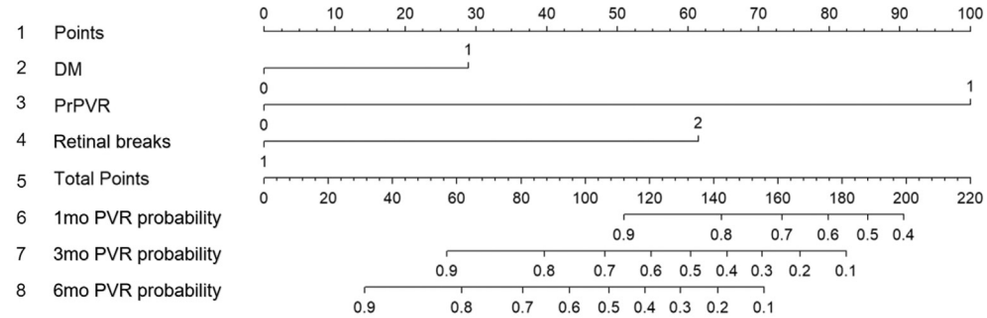

Figure 2 Nomogram for predicting the probability of absence of proliferative vitreoretinopathy (PVR) in patients with primary rhegmatogenous retinal detachment (RRD) at 1, 3, and 6mo postoperatively Lines 2-4 present the three predictors in the nomogram, while the first line outlines the scoring criteria for each predictor. Each patient has an integral value assigned to each predictor based on their individual characteristics, with the total integral value displayed in the "Total Integral" row. Lines 6 to 8 show the probabilities of no PVR in RRD patients at 1, 3, and 6mo following surgery, respectively. DM: Diabetes mellitus.

Table 2 Postoperative proliferative vitreoretinopathy characteristics in the training and validation cohorts *n* (%)

Category	Training cohort		*P*	Validation cohort		*P*
	Postop. PVR (*n*=147)	Without postop. PVR (*n*=368)		Postop. PVR (*n*=17)	Without postop. PVR (*n*=38)	
Preoperative PVR			0.001			0.001
Yes	116 (78.9%)	9 (2.4%)		10 (58.8%)	3 (7.9%)	
No	31 (21.1%)	359 (97.6%)		7 (41.2%)	35 (92.1%)	
PVR (A)	0	0		0	0	
PVR (B)	99 (85.3%)	7 (77.8%)		8 (80.0%)	2 (66.7%)	
PVR (C1)	17 (14.7%)	2 (22.2%)		2 (20.0%)	1 (33.3%)	

PVR: Proliferative vitreoretinopathy.

Table 3 Results of the univariate analysis in the training cohorts

Category	HR	95%CI	*P*
Age	1.00	0.99-1.01	0.96
Gender	1.55	1.12-2.15	0.01
Eye side	0.95	0.81-1.13	0.58
Diabetes	4.23	2.95-6.08	<0.001
Hypertension	5.69	2.13-15.22	<0.001
High myopia	4.70	2.20-10.04	<0.001
Retinal photocoagulation	2.24	1.36-3.68	<0.001
Cataract	0.96	0.65-1.42	0.32
Intraocular lens	1.51	0.86-2.64	0.85
Preoperative PVR	26.88	17.80-40.60	<0.001
Preoperative vitreous hemorrhage	4.60	0.63-33.37	0.131
Number of retinal breaks			
≥4	14.75	9.57-22.76	<0.001
Morphology of retinal breaks	0.81	0.53-1.23	0.31
Suprachoroidal fluid drainage	1.16	0.48-2.84	0.73
RD extension	1.23	1.02-1.31	0.79
Preoperative CD	0.86	0.58-0.93	0.03
RTS	1.86	1.43-2.47	0.12
TOS	0.73	0.61-0.87	0.45
Intraoperative air filling	0.97	0.67-1.42	0.89

PVR: Proliferative vitreoretinopathy; CD: Choroidal detachment; RD: Retinal detachment; RTS: Retinal tear size; TOS: Time from onset to surgery; HR: Hazard ratio; CI: Confidence interval.

RD. The differences between our findings and those reported by Gao *et al*[29] may be primarily attributed to differences in surgical technique, study population, and outcome definition. Their nomogram was developed in patients undergoing PPV and incorporated procedure-specific variables such as silicone oil tamponade time and photocoagulation energy, which are not applicable to SB surgery. In contrast, our model was specifically designed for primary RRD treated with SB, and therefore focuses on preoperative and disease-related predictors relevant to this surgical approach. Although diabetes mellitus accounted for a relatively small proportion of patients in our cohort, it remained an independent predictor of postoperative PVR after multivariate adjustment. This finding suggests that the clinical impact of diabetes on PVR risk is not solely determined by prevalence but rather by its biological effects, including chronic inflammation, microvascular dysfunction, and hypoxia-related pathways, which may promote cellular proliferation and membrane formation after RD surgery.

The nomogram proposed in this study includes three independent prognostic factors: preoperative PVR, history of diabetes, and the number of retinal breaks. Through stepwise backward- and forward-selection of the regression model, this study identified several predictors, including preoperative PVR, which was confirmed as an important predictor and risk factor for postoperative PVR[30]. Surgical trauma, cryotherapy-induced aseptic inflammation, and alterations in retinal morphology can trigger the release of inflammatory mediators, cell migration, and proliferation, which can also lead to

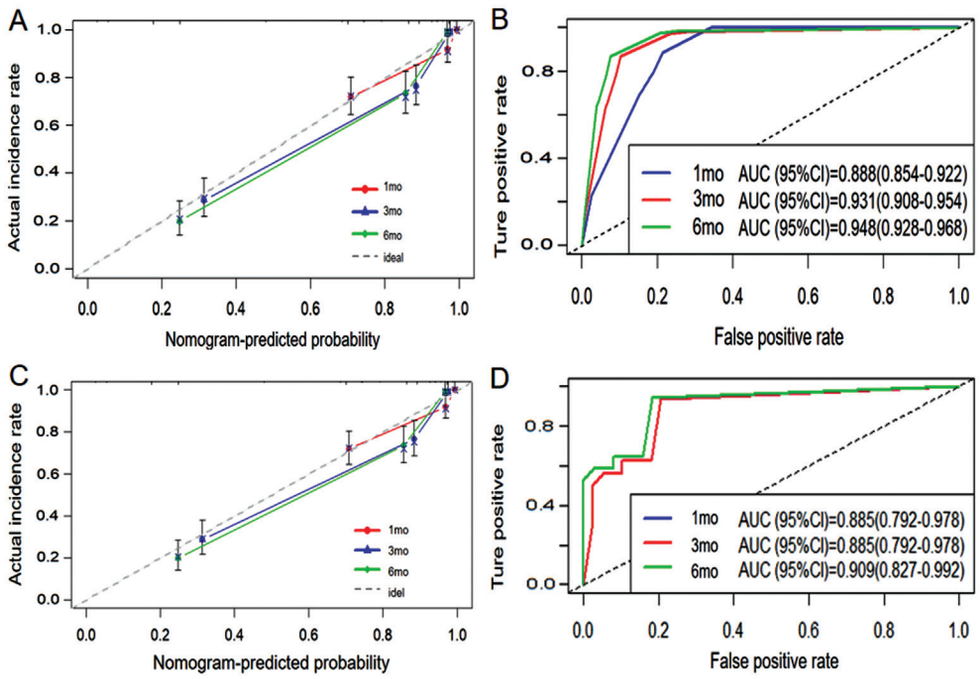

Figure 3 Calibration plots and receiver operating characteristic curves of the nomogram for predicting the absence of proliferative vitreoretinopathy at 1, 3, and 6mo postoperatively in the training (A, B) and validation (C, D) cohorts AUC: Area under the curve; CI: Confidence interval.

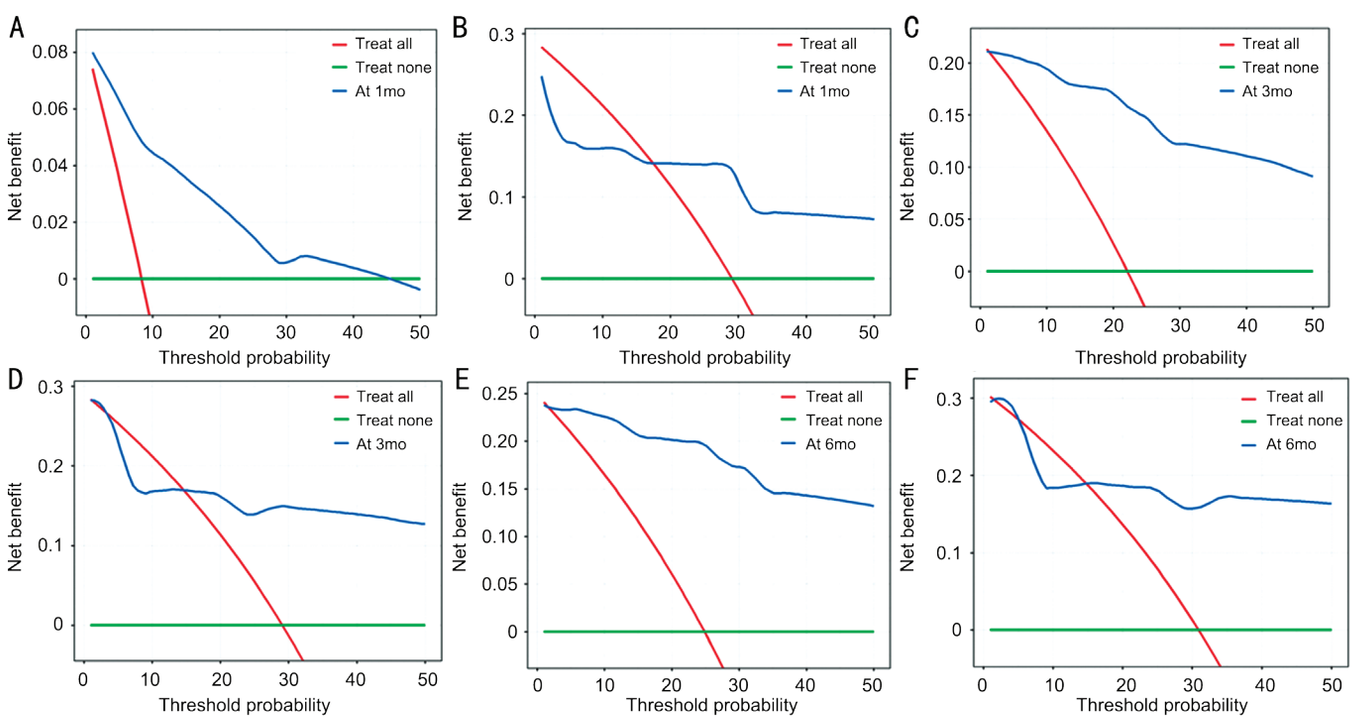

Figure 4 Decision curve analysis (DCA) of the nomogram for the prediction of the absence of proliferative vitreoretinopathy (PVR) At 1mo (A, B), 3mo (C, D), and 6mo (E, F) after surgery in patients with primary rhegmatogenous retinal detachment (RRD) in the training and validation cohorts.

postoperative PVR[31-32]. Some research findings suggest that the frequency of postoperative PVR in RRD patients ranges from 3% to 43%[9-12]. The incidence of postoperative PVR among RRD patients was 28.5% in this study, consistent with rates reported in the literature[12,33-35].

In the training cohort, we observed that a substantial proportion of patients who developed postoperative PVR had evidence of preoperative PVR (116/147, 78.9%), suggesting that postoperative disease may represent progression of early proliferative changes. In comparison, among patients without preoperative PVR, 31 of 147 (21.1%) developed PVR following surgery. We hypothesize that postoperative PVR may

result from a combination of aseptic inflammation induced by surgical trauma and cryotherapy, as well as the migration and proliferation of inflammatory mediators and cells triggered by alterations in retinal morphology[36-37]. Advanced age may also contribute to this process in some patients[14]. However, Sharma *et al*[38] reported that minimal, targeted cryotherapy, typically applied during SB procedures, may reduce the incidence of postoperative PVR, a finding that contrasts with our hypothesis and warrants further investigation.

Possibly related to the choice of surgical method and the degree of cryotherapy. Meanwhile, the proportion of preoperative PVR in members of the training set was 24.4%, thereby suggesting that the postoperative condition is an extension of the earlier proliferative disorder. The model indicated that preoperative PVR ranks first among prognostic factors, highlighting its greatest influence. In addition, most preoperative PVR cases are grade B (77.6%), which is relatively mild and does not affect the overall success of the surgery.

It was found that a history of diabetes increases the likelihood of PVR following SB surgery for RRD. Similarly, Pan *et al*[39] found differences in the formation mechanism, clinical manifestations, and effects on retinal vascular structure of PVR between diabetic and non-diabetic patients. Type 2 diabetes increases the chances of secondary PVR following PPV. In the serum of diabetic patients, insulin can enhance the production of hypoxia-inducible factor (HIF)-1 and angiogenic factors. In retinal pigment epithelial cells, insulin-like growth factor (IGF)-1 enhances HIF-1 expression and DNA-binding activity, thereby increasing vascular endothelial growth factor (VEGF) levels. HIF-1 is crucial for neovascularization and PVR development in patients with diabetes[40]. Furthermore, Zhang *et al*[41] found that changes in retinal blood vessels and self-regulatory disorders had already occurred in individuals with diabetic retinopathy before reaching the threshold for diagnosis, as evidenced by decreased blood flow density, an enlarged foveal avascular zone, and reduced retinal perfusion density. Optical coherence tomography angiography (OCTA) can detect these changes during early stages. Reduced blood supply may lead to a decline in the metabolic function of the foveal area, and decreased perfusion density may cause tissue hypoxia, further triggering inflammatory responses or other pathological changes. A history of diabetes increases the risk of PVR development after SB surgery for RRD. In the future, large-sample clinical studies will further validate these findings as diabetes classification and staging evolve, providing stronger evidence for clinical practice.

This study identifies having 4 or more retinal breaks as a factor associated with PVR in RRD patients post-SB treatment. This aligns with the findings of Hirakata *et al*[13], who observed that three or more retinal breaks increased PVR risk following RRD surgery, as well as those of Cacioppo *et al*[11] identified on PVR development in RRD cases post-SB surgery, also impacting postoperative visual acuity. Multiple retinal breaks can increase the risk of macular folds, possibly because of the extent and severity of retinal damage. Conversely, longer breaks extend the duration and scope of cryotherapy, exacerbating retinal epithelial cell growth and migration, particularly in horseshoe-shaped breaks[13,42]. In RRD, these cells may enter the vitreous through breaks in the retina, and cryotherapy during SB surgery, which is known to disrupt the blood-retinal barrier, can enhance this migration, potentially stimulating a proliferative response post-RRD[43].

In this study, no significant association was observed between retinal tear size (RTS) and PVR development (P=0.12). This finding may be explained by two key factors. First, the inclusion criteria for retinal tears were strictly defined. Eyes with retinal tears larger than 2 diameter of the optic papilla (PD) were not treated with SB and were therefore excluded, minimizing the confounding effect of large tears, which are well recognized as high-risk factors for PVR. Second, the study population consisted of patients with relatively simple retinal tears who developed new-onset PVR after initial SB surgery, rather than patients with recurrent RD caused by advanced PVR following SB. This distinction in study populations may largely account for the discrepancy with previous reports, such as the study by Wang *et al*[44], which included 72 cases of recurrent RD secondary to severe PVR after SB surgery and identified a single retinal tear exceeding 2 PD as a significant risk factor for severe PVR.

These findings suggest that the influence of a large retinal tear (>2 PD) on PVR is stage-dependent, with its impact being more pronounced during PVR progression, leading to recurrent RD than during the early postoperative phase of PVR development. Importantly, our study also found no significant differences in postoperative PVR incidence with respect to the extent of RD (P=0.79) or the use of suprachoroidal fluid drainage (P=0.73), which is consistent with the results reported by Sharma *et al*[38].

Moreover, the risk factors incorporated into the proposed nomogram are readily available from routine clinical records. By entering these easily accessible variables into the model, clinicians can estimate the likelihood of PVR development at 1, 3, and 6mo postoperatively, thereby supporting informed clinical decision-making during follow-up. The principal strength of this study is its large sample size, and it may represent the first investigation to apply a nomogram for predicting the risk of PVR following primary repair of RRD. Nevertheless, several limitations should be acknowledged. Initially, the nomogram was developed using retrospective data from a single center, which may introduce selection bias.

Then, owing to data limitations, several factors known to be associated with PVR, such as blood-retinal barrier breakdown, the number of surgical procedures, a history of uveitis or allergic dermatitis, the duration of intraoperative cryotherapy, and postoperative anti-inflammatory treatment, were not evaluated. In addition, cryotherapy, which has been reported as a potential risk factor for postoperative PVR, was also not analyzed in the present model. This is because cryotherapy was routinely and uniformly applied to all patients undergoing SB at our institution, precluding assessment of its independent effect. Therefore, the potential contribution of cryotherapy to PVR development could not be evaluated. Future prospective studies incorporating variations in cryotherapy technique or intensity may help clarify its role in postoperative PVR risk. Lastly, although a 6-month follow-up period was adopted in line with the protocols of Fu *et al*[34] and Blanco-Teijeiro *et al*[45], a small proportion of late-onset postoperative PVR cases may have been missed. Accordingly, future prospective studies with extended follow-up and larger sample sizes are warranted to validate our findings further.

In conclusion, this study established a regression model identifying a history of diabetes, preoperative PVR, and having four or more retinal breaks as independently predictive of PVR development following SB surgery for RRD. The nomogram illustrated the predictive value of the variables for PVR in RRD patients, providing an objective clinical prediction tool. Meanwhile, the calibration, discrimination, and clinical effectiveness of the constructed PVR clinical prediction model were evaluated. The findings indicated that the model was effective at making predictions. The prediction model developed in this study accurately assesses the risk of PVR within 6mo post-SB surgery in RRD patients, aiding clinical interventions to lower postoperative PVR incidence and enhance patient prognosis.

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Artificial Intelligence Statement Disclosure: No artificial intelligence (AI) tools were used in the preparation, analysis, or interpretation of this study.

Conflicts of Interest: Xing PY, None; **Shao H,** None; **Zhang Y,** None; **Hu XJ,** None; **Zhang L,** None; **Zhang JJ,** None; **Zhu HL,** None; **Wang SW,** None.

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