BMC Ophthalmology

RESEARCH

Open Access

High BMI, silicone oil tamponade, and recurrent vitreous hemorrhage predict poor visual outcomes after pars plana vitrectomy in proliferative diabetic retinopathy

Zhi-gang Wang[1], Ping Li[1], Xiu-min Yang[1], Zi-qi Wang[1,3*] and Pei-ran Zhang[2]

Abstract

Background Proliferative diabetic retinopathy (PDR) is a serious microvascular complication of diabetes and a leading cause of global vision loss. Pars plana vitrectomy (PPV) is the primary surgical treatment for PDR, but visual outcomes vary due to multiple influencing factors. This study aims to evaluate the factors predicting visual prognosis in patients with PDR after PPV.

Methods A retrospective analysis was performed on 112 eyes from 87 patients with PDR who underwent PPV between May 2020 and May 2024. Data collected included patient demographics, preoperative and postoperative best-corrected visual acuity (BCVA), and other baseline clinical data. Data analysis was performed with IBM SPSS Statistics Version 24.0. Univariate and multivariate linear regression models were applied to assess the relationship between the final BCVA and various clinical parameters.

Results The mean BCVA improved significantly, from 1.94 ± 0.89 logMAR preoperative to 0.76 ± 0.70 logMAR postoperatively ($P < 0.001$). Multivariate linear regression identified body mass index (BMI) (B = 0.035; 95% CI 0.003–0.066; $P = 0.033$), silicone oil (SO) tamponade (B = 0.354; 95% CI 0.005–0.643; $P = 0.029$), and recurrent vitreous hemorrhage (VH) (B = 0.585; 95% CI 0.304–0.867; $P < 0.001$) as significant negative predictors of final BCVA.

Conclusions While PPV improves visual outcomes in PDR patients, factors such as high BMI, SO tamponade, and recurrent VH negatively affect prognosis and could serve as predictors of poor visual outcomes following the procedure. This study emphasizes the importance of tailored management strategy for PDR, including early intervention, optimal BMI control, and minimizing SO tamponade duration.

Keywords Proliferative diabetic retinopathy (PDR), Pars plana vitrectomy (PPV), Visual outcomes, Predictive factors, Body mass index (BMI), Silicone oil (SO), Vitreous hemorrhage (VH)

*Correspondence:
Zi-qi Wang
wangziqi199@gmail.com
[1]Department of Fundus Diseases, Hefei Aier Eye Hospital, Hefei 230000, China
[2]Department of Fundus Diseases, Anhui Aier Eye Hospital, Hefei 230000, China
[3]Department of Fundus Diseases, Anhui Medical University Aier Eye Hospital Medical Center, Hefei 230000, China

Background

Proliferative diabetic retinopathy (PDR) is a severe ocular complication affecting 0.99-2.3% of individuals with diabetes mellitus [1–3]. Its pathogenesis involves chronic hyperglycemia induced ischemia, oxidative stress, and inflammation, driving vascular endothelial growth factor (VEGF) upregulation and pathological neovascularization, which can lead to vitreous hemorrhage (VH) and traction retinal detachment (TRD), often resulting in irreversible vision loss [4]. Key risk factors, including increased haemoglobin A1c (HbA1c), prolonged diabetes duration, hypertension, obesity, smoking, and high body mass index (BMI), contribute to retinal ischemia and hypoxia, thereby accelerating the progression of PDR [5–7]. With an aging population and changing lifestyles, the prevalence of PDR is increasing, posting significant health and socio-economic challenges.

Pars plana vitrectomy (PPV) is the primary surgical intervention for PDR, aiming to restore vision by clearing VH and relieving vitreoretinal traction. Indications for PPV outcomes include non-clearing VH, TRD, progressive fibrovascular membrane proliferation, macular TRD, macular hole, and neovascular glaucoma [8].

Although many studies focus on the pathogenesis and anatomical changes in PDR, fewer have examined visual recovery following PPV [9–11]. This study investigates visual recovery and identifies key factors that influence postoperative best-corrected visual acuity (BCVA), providing critical insights for the clinical management of PDR patients following PPV.

Methods

This retrospective study included 112 eyes from 87 PDR patients who underwent PPV at Hefei Aier Eye Hospital between May 2020 and May 2024. Inclusion criteria included diagnosis with PDR and treatment with PPV, comprehensive preoperative and postoperative data, and a follow-up duration of at least three months. Exclusion criteria included patients with neovascular glaucoma, trauma, uveitis, retinal vein occlusion, endophthalmitis, or macular degeneration.

Baseline clinical data were systematically collected. Demographics, duration of symptoms, systemic diseases, diabetes duration and treatment methods were obtained from medical records. Measurement of BMI on the day of surgery. Blood glucose (GLU) and HbA1c levels were measured via standard laboratory tests. All patients underwent preoperative and postoperative follow-up ophthalmic examinations by highly skilled and experienced ophthalmologists. Preoperative (baseline) and postoperative BCVA was assessed using the Logarithm of the Minimum Angle of Resolution Chart (GB11533-2011), and intraocular pressure (IOP) was measured with Computerized Non-Contact Tonometry (Topcon Corporation, Tokyo, Japan). Intraoperative tamponade agents, such as silicone oil (SO) or liquid were documented from surgical records. Slit-lamp biomicroscopy was used to examine preoperative and postoperative lens status. Postoperative complications, including recurrent VH and other complications, were evaluated through slit-lamp biomicroscopy, indirect ophthalmoscopy, B-scan ultrasonography, or scanning laser ophthalmoscopy fundus imaging. PDR was staged according to the 2014 Chinese Clinical Guidelines for the Diagnosis and Treatment of DR: Stage IV involves vitreous hemorrhage, Stage V includes fibrous vascular membranes, and Stage VI involves TRD [12].

The surgeries were performed by two experienced surgeons, both employing identical surgical techniques. Local anesthesia was induced by retrobulbar nerve block. All patients underwent a 3-port 25-gauge PPV (Alcon Laboratories, Incorporated, Ft Worth, Texas, USA) following intravitreal injections of anti-VEGF 3–5 days prior to the surgery. During triamcinolone acetonide assisted vitrectomy, complete removal of the vitreous and posterior hyaloid detachment was achieved using a vitreous cutter and suction technique. Fibroproliferative membranes were meticulously removed to relieve traction if required. Subsequently, liquid-air exchange was performed, followed by pan-retinal photocoagulation. Choice of intraocular tamponade depended on the characteristics of PDR. SO tamponade was indicated for active neovascularization with persistent intraoperative bleeding, tractional retinal breaks secondary to fibroproliferative membranes, or the necessity for retinotomies to achieve retinal reattachment. In other cases, the standard irrigation solution was retained. In the absence of complications, SO was typically removed 3 to 6 months postoperatively. When necessary, severe cataract extraction (phacoemulsification) and intraocular lens implantation were performed either during the initial surgery or at the time of SO removal. Intraoperative complications were managed include hemorrhage was controlled with diathermy, increased infusion pressure, epinephrine irrigation, or SO tamponade; expulsive choroidal hemorrhage with immediate incision closure and surgical pause; and lens extraction was performed for lens injuries.

Statistical analysis was performed using SPSS Statistics 24.0. Changes in BCVA were assessed using paired t-tests. The association between final BCVA and clinical parameters was analyzed using univariate and multivariate linear regression. Univariate linear regression was performed to evaluate the independent effect of each clinical parameter on final BCVA, with statistically significant variables subsequently included in the multivariate linear regression analysis. Multivariate linear regression was then used to account for confounding factors and

identify independent predictors of final BCVA. The statistical significance set at P value < 0.05.

Results

Baseline characteristics

The baseline clinical characteristics for 112 eyes from 87 PDR patients are shown in Table 1. The study cohort consisted of 41 males and 46 females, with a mean age of 53.73 ± 11.82 years. Both eyes were equally represented (52 right, 60 left). The mean duration of symptoms was 7.08 ± 7.40 months. Among the symptoms, progressive loss of vision had the longest duration (15.13 ± 7.21 months), followed by progressive loss with distorted vision (6.13 ± 4.69 months) and progressive loss with occlusion (5.99 ± 6.49 months). Sudden vision loss had the shortest duration, averaging 0.67 ± 0.48 months. Additionally, the mean duration of diabetes was 10.92 ± 6.80 years.

Table 1 Baseline characteristics of 112 eyes from 87 proliferative diabetic retinopathy patients

Factor	
Male/female, *n*	41/46
Age, mean ± SD (range), years	53.73 ± 11.82 (26–76)
Eye, right/left, *n*	52/60
Duration of symptoms, mean ± SD (range), months	7.08 ± 7.40 (0.07-36.00)
progressive loss of vision	15.13 ± 7.21(4.00–36.00)
progressive loss of vision with distorted vision	6.13 ± 4.69(1.00–12.00)
progressive loss of vision with occlusion of vision	5.99 ± 6.49(0.50–36.00)
sudden loss of vision	0.67 ± 0.48 (0.07-2.00)
Course of diabetes, mean ± SD (range), years	10.92 ± 6.80 (0.08-25.00)
Diabetes treatment, *n* (%)	
Oral medications	28 (25.0%)
Insulin	24 (21.4%)
Insulin and oral medications	60 (53.6%)
BMI, mean ± SD (range), kg/m^2	25.80 ± 3.75 (19.38–37.18)
Hypertension, *n* (%)	66 (58.9%)
Cerebral and myocardial infarction, *n* (%)	23 (20.54%)
Staging of DR, *n* (%)	
†IV	33 (29.5%)
‡V	35 (31.1%)
*VI	44 (39.3%)
Preoperative BCVA, mean ± SD (range), logMAR	1.94 ± 0.89 (0.50-3.00)
Preoperative IOP, mean ± SD (range), mmHg	18.00 ± 3.61 (11.00–43.00)
Tamponade, *n* (%)	
SO	43 (38.4%)
Liquid	69 (61.6%)
Recurrent VH, *n* (%)	
Medication treatment	10 (8.90%)
Surgical treatment	3 (2.70%)
Final BCVA, mean ± SD (range), logMAR	0.76 ± 0.70 (0–5.00)
Final IOP, mean ± SD (range), mmHg	17.79 ± 3.62 (10-31.4)
Follow-up, mean ± SD (range), months	5.54 ± 4.80 (3.00–28.00)
Complications, *n* (%)	
Cataract	2 (1.79%)
Glaucoma	18 (16.07%)
Lens status, *n* (%)	
Phakic	30 (26.8%)
Pseudophakic	82 (73.2%)

SD, standard deviation; BMI, body mass index; DR, diabetic retinopathy; BCVA, best-corrected visual acuity; log MAR, logarithm of minimum angle of resolution; IOP, intraocular pressure; SO, silicone oil; VH, vitreous hemorrhage

The staging of DR in this study was determined based on the definitions provided in the 2014 Chinese Clinical Guidelines for the Diagnosis and Treatment of DR [12]

†With retinal neovascularization or optic disc neovascularization

‡With fibrovascular membrane

*With tractional retinal detachment

Table 2 Univariate linear regression analysis of clinical parameters for final BCVA in 112 eyes from 87 PDR patients

	B (95%) CI	*P*
Age (years)	-0.008(-0.019-0.003)	0.162
Symptoms	-0.123(-0.256-0.011)	0.071
Duration of symptoms (months)	0.018(0.000-0.035)	0.049
Course of diabetes (years)	-0.016(-0.035-0.003)	0.103
Diabetes treatment	-0.007(-0.165-0.150)	0.929
BMI (kg/m^2)	0.044(0.010–0.078)	0.012
Hypertension	0.097(-0.171-0.365)	0.477
Cerebral and myocardial infarction	0.025(-0.302-0.353)	0.878
Staging of DR	0.225(0.071–0.380)	0.005
Tamponade	0.380(0.117–0.642)	0.005
Recurrent VH	0.625(0.333–0.917)	< 0.001
Final IOP (mmHg)	0.061(0.026–0.096)	0.001
GLU (mmol/L)	0.040(-0.025-0.106)	0.225
HbA1c (%)	0.005(-0.073-0.084)	0.890

PDR, proliferative diabetic retinopathy; BMI, body mass index; DR, diabetic retinopathy; BCVA, best-corrected visual acuity; log MAR, logarithm of minimum angle of resolution; VH, vitreous hemorrhage; IOP, intraocular pressure; GLU, glucose; HbA1c, hemoglobin A1c; B, regression coefficient; CI, confidence interval

The staging of DR in this study was determined based on the definitions provided in the 2014 Chinese Clinical Guidelines for the Diagnosis and Treatment of DR [12]

Regarding diabetes treatment, 28 cases (25.0%) were on oral medications, 24 cases (21.4%) on insulin, and 60 cases (53.6%) on both. The mean BMI was 25.80 ± 3.75 kg/m². Medical history-wise, 66 cases (58.9%) had hypertension, and 23 cases (20.54%) had a history of cerebral or myocardial infarction. According to the 2014 Chinese Clinical Guidelines for DR, 33 eyes (29.5%) were classified as Stage IV, 35 eyes (31.1%) as Stage V, and 44 eyes (39.3%) as Stage VI.

Intraoperatively, SO tamponade was used in 43 cases (38.4%) and liquid tamponade in 69 cases (61.6%). Postoperative recurrence of VH requiring pharmacotherapy occurred in 10 cases (8.9%), and surgical treatment was required in 3 cases (2.7%). Preoperative BCVA improved significantly from 1.94 ± 0.89 logMAR to 0.76 ± 0.70 logMAR postoperatively ($P < 0.001$). The mean preoperative IOP was 18.00 ± 3.61 mmHg, and the mean final IOP was 17.79 ± 3.62 mmHg. The mean follow-up duration was 4.84 ± 5.23 months. Regarding lens status, 30 cases (26.8%) had phakic lenses, and 82 cases (73.2%) had pseudophakic lenses.

A total of 112 eyes were included, with 51 operated on by Surgeon 1 and 61 by Surgeon 2. Preoperative BCVA was comparable between the two groups (1.93 ± 0.90 logMAR for Surgeon 1 vs. 1.95 ± 0.89 logMAR for Surgeon 2, $P = 0.917$). Postoperatively, both groups showed significant improvement in final BCVA, achieving 0.85 ± 0.65 logMAR and 0.68 ± 0.74 logMAR, respectively; however, the difference between the two groups was not statistically significant ($P = 0.202$). The mean surgical duration was slightly shorter for Surgeon 1 (76.18 ± 24.51 min) compared to Surgeon 2 (83.85 ± 31.99 min), although this difference also did not reach statistical significance ($P = 0.154$).

Table 3 Multivariate linear regression analysis of clinical variables for final BCVA in 112 eyes from 87 PDR patients

	B (95%) CI	*P*
Duration of symptoms (months)	0.016 (-0.001-0.032)	0.058
BMI (kg/m^2)	0.035 (0.003–0.066)	0.033
Staging of DR	0.000(-0.195-0.196)	0.996
Tamponade	0.354 (0.005–0.643)	0.029
Recurrent VH	0.585 (0.304–0.867)	< 0.001
Final IOP (mmHg)	0.027 (-0.007-0.061)	0.119

PDR, proliferative diabetic retinopathy; BMI, body mass index; DR, diabetic retinopathy; VH, vitreous hemorrhage; IOP, intraocular pressure; B, regression coefficient; CI, confidence interval

The staging of DR in this study was determined based on the definitions provided in the 2014 Chinese Clinical Guidelines for the Diagnosis and Treatment of DR [12]

Univariate linear regression analysis of final BCVA and clinical parameters

Univariate linear regression analysis revealed that the duration of symptoms ($P = 0.049$), BMI ($P = 0.012$), PDR stage ($P = 0.005$), tamponade type ($P = 0.005$), recurrent VH ($P < 0.001$), and final IOP ($P = 0.001$) were independently associated with final BCVA. In contrast, age, symptoms, diabetes duration, diabetes treatment, history of hypertension, history of cerebral and myocardial infarction, GLU, and HbA1c were not significantly associated with final BCVA (Table 2).

Multivariate linear regression analysis of final BCVA and clinical parameters

Multivariate linear regression analysis identified BMI, tamponade type, and recurrent VH as independent negative predictors of final BCVA. Specifically, each unit increase in BMI was associated with a 0.185 increase in logMAR BCVA ($P = 0.033$). Compared to fluid tamponade, SO tamponade was associated with a 0.225 increase in logMAR BCVA ($P = 0.029$). Recurrent VH had the most significant impact, with a 0.351 increase in logMAR BCVA ($P < 0.001$). In contrast, the duration of symptoms, PDR stage, and final IOP were not significantly associated with final BCVA (Table 3).

Discussion

Although PDR has traditionally been considered an incurable blinding disease, significant anatomical and visual improvements can be achieved with PPV. The PPV procedure involves removing cloudy refractive media, relieving vitreoretinal traction, eliminating the retinal membrane, applying laser photocoagulation, and achieving retinal reattachment using gas or SO [13]. PPV

enhances oxygen diffusion and disrupts proliferative scaffolds, reducing fibrovascular membrane growth and slowing the progression of DR. However, patients with advanced PDR often face challenges due to low preoperative BCVA and uncertain surgical outcomes. Ricca et al. reported that preoperative BCVA ≥ 0.5 was seen in 6% of patients, which increased to 28% postoperatively [14]. Nisic et al. found a mean preoperative BCVA of 0.03, which significantly improved to 0.18 and 0.2 at 6 and 12 months postoperatively [15]. In our study, the preoperative BCVA was 1.94 ± 0.89 logMAR, which improved significantly to 0.76 ± 0.70 logMAR at the final follow-up, consistent with other studies. While PPV effectively enhances visual acuity, outcomes vary among patient groups.

Recent research has extensively documented the relationship between BMI and DR. A cross-sectional study by Yi et al. in Shanghai found a positive association between BMI and the incidence and severity of DR [14]. This finding aligns with recent Mendelian randomization studies which demonstrated a causal effect of BMI on DR development [16, 17]. Furthermore, Singh et al. showed that bariatric surgery significantly reduces the risk of diabetes-related microvascular complications, further supporting the association between obesity and DR [18]. Our study also found a significant association between BMI and visual recovery after PPV. Specifically, multivariate regression analysis showed that for each unit increase in BMI, the final BCVA increased by 0.185 logMAR ($P = 0.033$), suggesting that higher BMI negatively impacts visual recovery. This may be due to factors such as chronic low-grade inflammation, insulin resistance, and dyslipidemia, which are common in obese patients [19, 20]. These factors can disrupt retinal microcirculation and induce immune responses, ultimately affecting retinal repair and surgical outcomes. Our findings underscore the importance of recognizing high BMI as a significant risk factor in managing PDR. Patients with elevated BMI often present with worse postoperative visual acuity. The mechanisms underlying poor postoperative visual outcomes in high BMI patients may involve multiple pathophysiological alterations. Obesity-associated chronic low-grade inflammation can lead to insulin resistance and endothelial dysfunction, exacerbating oxidative stress and disrupting retinal microcirculatory stability [21]. Additionally, studies have shown that obese and diabetic patients exhibit significantly lower adiponectin levels compared to healthy individuals [22, 23]. As a key adipocytokine, adiponectin regulates glucose homeostasis and inhibits vascular smooth muscle cell migration and proliferation [24]. Reduced adiponectin levels may promote the development and progression of DR [23, 25]. These metabolic disturbances and vascular dysfunctions may impair retinal tissue repair capacity, affecting postoperative functional recovery. To address this, comprehensive management strategies — such as dietary control, regular physical activity, pharmacologic interventions, individualized surgical decision-making, and postoperative care — may help reduce BMI and slow PDR progression.

Studies reported that 4–45.2% of PDR patients experience recurrent VH after PPV [26, 27]. Leading causes include residual blood clots on the retinal surface, persistent bleeding from fibrovascular membranes, blood retention in the peripheral vitreous, and iatrogenic injury to retinal vessels during surgery [28]. Early and mild VH can often be managed with oral medications designed to enhance absorption. For instance, medications that improve circulation and alleviate blood stasis are commonly prescribed to facilitate blood resorption. In patients with artificial lenses, a posterior capsulotomy using an Nd: YAG laser can sometimes redirect blood into the anterior chamber, where it is cleared through the trabecular meshwork or anterior chamber drainage [29]. However, persistent VH requires more aggressive treatments, such as vitrectomy or intravitreal anti-VEGF injections. Vitrectomy can directly remove the VH, while anti-VEGF agents, such as bevacizumab or ranibizumab, reduce neovascularization and bleeding by inhibiting VEGF [30]. In our study, 11.6% of patients experienced recurrent VH, with 10 cases treated with oral medication and 3 requiring vitrectomy lavage to clear the hemorrhage. Recurrent VH indicates persistent neovascularization or ongoing release of angiogenic factors, which can lead to active bleeding or contraction of fibrovascular tissue, resulting in proliferative vitreoretinopathy and irreversible retinal damage. Our results show that recurrent VH significantly negatively impacts postoperative visual recovery in PDR patients. Therefore, promptly addressing unresolved VH is crucial to prevent further retinal damage and promote visual recovery.

Indications for intraoperative SO tamponade in PDR patients include TRD, multiple significant retinal breaks, VH with severe fibrovascular proliferation, and eyes undergoing retinectomy or retinotomy. Wang et al. identified intraoperative SO tamponade as a significant risk factor for poor postoperative visual acuity [31]. SO tamponade or removal may lead to vision loss [32]. Our study confirms this finding, showing that intraoperative SO tamponade is associated with poorer visual outcomes. Patients requiring SO tamponade often present with severe, advanced proliferative changes, including fibrovascular membrane traction, which significantly affects retinal anatomy and function. As a result, postoperative visual recovery is typically poor. SO is widely used to preserve retinal anatomy by providing internal tamponade and promoting retinal reattachment. However, SO tamponade can cause complications such as glaucoma,

SO emulsification, migration into the anterior chamber, cataracts, corneal damage, and optic nerve toxicity, all of which can result in postoperative vision loss [33–35]. Scheerlinck et al. advocate for early SO removal when possible, as the duration of SO tamponade may be a risk factor for vision loss [36]. SO exerts mechanical pressure on the retinal surface and blocks metabolic exchange, particularly by inhibiting the diffusion of oxygen into the vitreous cavity, thereby increasing the risk of retinal ischemia [37]. Furthermore, the sub-SO fluid enhances these biological responses, including VEGF, reactive Muller cells, fibronectin and inflammatory cytokine [38]. Additionally, SO impedes the diffusion of anti-VEGF agents to the retinal surface, reducing their therapeutic efficacy [39]. These findings suggest that minimizing SO tamponade duration could reduce complications and improve visual outcomes. Mitigating the adverse effects of SO tamponade duration includes careful patient selection, prone or lateral positioning, close monitoring of intraocular pressure, and timely surgical intervention to manage complications.

TRD requires individualized management, as detachment configuration and surgical timing significantly influence outcomes. Urgent intervention is critical for macula-on TRD or progressive cases threatening the macula, whereas macula-off or stable detachments may allow elective surgery. Silicone oil is indispensable in complex PDR, particularly for extensive detachment, retinal breaks, persistent traction, or severe hemorrhage, providing durable tamponade and retinal stability. When retinal breaks have closed, the retina is reattached, and no fresh hemorrhage is present, silicone oil removal is typically performed within 3–6 months. However, factors such as poor glycemic control, recurrent vitreous hemorrhage, or newly formed fibroproliferative membranes inducing traction may necessitate prolonged retention or substitution to maintain retinal stability. While anatomical outcomes post-removal was generally favorable, visual recovery is often constrained by pre-existing pathology. Although this study observed worse final BCVA in eyes with silicone oil compared to those without, the difference was not statistically significant. Nevertheless, silicone oil remains invaluable in refractory or poor-prognosis cases, ensuring structural preservation and long-term stability.

This study has several strengths and limitations. Its large sample size enhances statistical power, while the use of multivariate analysis allows for a comprehensive evaluation of factors influencing visual outcomes after PPV for PDR, providing valuable insights. However, its retrospective nature and potential for selection bias due to surgeries performed by two different surgeons are limitations. Additionally, the varied follow-up length among patients, ranging from just a few months to several years, may affect outcome reliability. Our study sample size fell short of the ideal target, highlighting the need for future multicenter, prospective clinical trials with longer follow-up periods are needed to obtain more comprehensive results, reduce bias, and increase the reliability of findings. Involving multiple centers would enhance the generalizability of the results and provide more robust data to guide clinical treatment.

Conclusions

While PPV improves visual outcomes in PDR patients, high BMI, SO tamponade, and recurrent VH are risk factors for poor prognosis and could serve as predictors of poor visual outcomes following the procedure. This study emphasizes the importance of tailored management strategy for PDR, including early intervention, optimal BMI control, and minimizing SO tamponade duration. Future multicenter, prospective studies are needed to validate these findings and to develop a more robust and reliable algorithm for predicting visual prognosis in PDR patients following PPV.

Acknowledgements

I would like to extend my heartfelt gratitude to Professor Cheng Lin for her invaluable assistance in revising and polishing the manuscript.

Author contributions

ZG W analyzed the patient data and wrote the manuscript. PL and XM Y participated in the conceptualization of the study. ZQ W was responsible for the study and review the manuscript. PR Z collected the patient data. All authors read and approved the final manuscript.

Funding

This study was supported by research grants from the Anhui Medical University School Fund Project (No. 2022xkj135).

Data availability

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

Declarations

Ethics approval and consent to participate

The study adhered to the principles of the Declaration of Helsinki and was approved by the Institutional Review Board of Hefei Aier Eye Hospital (Approval No. 202212). Written informed consent was obtained from all participants prior to their inclusion in the study. This study was conducted as a retrospective analysis using anonymized data. The requirement for informed consent was waived by the Hefei Aier Eye Hospital because the research involved no more than minimal risk to participants, and the use of identifiable data was not necessary for the study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 17 January 2025 / Accepted: 3 March 2025
Published online: 19 March 2025

References

1. Thomas RL, Halim S, Gurudas S, Sivaprasad S, Owens DR. IDF diabetes atlas: A review of studies utilising retinal photography on the global prevalence of diabetes related retinopathy between 2015 and 2018 [J]. Diabetes Res Clin Pract. 2019;157:107840. https://doi.org/10.1016/j.diabres.2019.107840
2. Song P, Yu J, Chan KY, Theodoratou E, Rudan I. Prevalence, risk factors and burden of diabetic retinopathy in China: a systematic review and meta-analysis [J]. J Glob Health. 2018;8(1):010803. https://doi.org/10.7189/jogh.08.010803
3. Yaow CY, Lin SY, Xiao J, Koh JH, Yong JN, Tay PW, Tan ST. A meta-analysis of prevalence of diabetic retinopathy in Asia [J]. Minerva Endocrinol (Torino). 2022. https://doi.org/10.23736/s2724-6507.21.03585-5
4. Schreur V, Brouwers J, Van Huet RC, Smeets S, Phan M, Hoyng CB, De Jong EK, Klevering BJ. Long-term outcomes of vitrectomy for proliferative diabetic retinopathy [J]. Acta Ophthalmol. 2021;99(1):83–9. https://doi.org/10.1111/aos.14482
5. Perais J, Agarwal R, Evans JR, Loveman E, Colquitt JL, Owens D, Hogg RE, Lawrenson JG, Takwoingi Y, Lois N. Prognostic factors for the development and progression of proliferative diabetic retinopathy in people with diabetic retinopathy [J]. Cochrane Database Syst Rev. 2023;2(2):Cd013775. https://doi.org/10.1002/14651858.CD013775.pub2
6. Kaur A, Kumar R, Sharma A. Diabetic retinopathy leading to Blindness- A review [J]. Curr Diabetes Rev. 2024;20(9):e240124225997. https://doi.org/10.2174/0115733998274599231109034741
7. Bek T, Nielsen MS, Klug SE, Eriksen JE. Increasing metabolic variability increases the risk for vitrectomy in proliferative diabetic retinopathy [J]. Int Ophthalmol. 2022;42(3):757–63. https://doi.org/10.1007/s10792-021-02041-3
8. Chen SN, Chen SJ, Wu TT, Wu WC, Yang CH, Yang CM. Refining vitrectomy for proliferative diabetic retinopathy [J]. Graefes Arch Clin Exp Ophthalmol. 2023;261(12):3659–70. https://doi.org/10.1007/s00417-023-06134-w
9. Shi Q, Wang Q, Wang Z, Lu J, Wang R. Systemic inflammatory regulators and proliferative diabetic retinopathy: A bidirectional Mendelian randomization study [J]. Front Immunol. 2023;14:1088778. https://doi.org/10.3389/fimmu.2023.1088778
10. Meng Z, Chen Y, Wu W, Yan B, Meng Y, Liang Y, Yao X, Luo J. Exploring the immune infiltration landscape and M2 Macrophage-Related biomarkers of proliferative diabetic retinopathy [J]. Front Endocrinol (Lausanne). 2022;13:841813. https://doi.org/10.3389/fendo.2022.841813
11. Fang J, Wang H, Niu T, Shi X, Xing X, Qu Y, Liu Y, Liu X, Xiao Y, Dou T, Shen Y, Liu K. Integration of vitreous lipidomics and metabolomics for comprehensive Understanding of the pathogenesis of proliferative diabetic retinopathy [J]. J Proteome Res. 2023;22(7):2293–306. https://doi.org/10.1021/acs.jproteome.3c00007
12. Fundus Disease Group OSOCMA. Guidelines for clinical diagnosis and treatment of diabetic retinopathy. (2014) [J]. Chin J Ophthalmol. 2014;50(11):851–865. https://doi.org/10.3760/cma.j.issn.0412-4081.2014.11.014
13. Smiddy WE, Flynn HW. Jr. Vitrectomy in the management of diabetic retinopathy [J]. Surv Ophthalmol. 1999;43(6):491–507. https://doi.org/10.1016/s0039-6257(99)00036-3
14. Yi QX, Zhu LN, Ma J, Yu XJ, Liu L, Shen J. Use of anthropometric measures of obesity to predict diabetic retinopathy in patients with type 2 diabetes in China [J]. Diabetes Metab Syndr Obes. 2021;14:4089–95. https://doi.org/10.2147/dmso.S321030
15. Nisic F, Gadzo AP, Fajkic A, Nisic A, Miokovic AP, Damjanovic G, Begic E, Beslic N, Lepara O. Predictors of visual outcome after Pars plana vitrectomy secondary to proliferative diabetic retinopathy [J]. Rom J Ophthalmol. 2023;67(3):283–8. https://doi.org/10.22336/rjo.2023.46
16. Shu Y, Zhou Q, Shao Y, Lin H, Qu S, Han W, Lv X, Bi Y. BMI and plasma lipid levels with risk of proliferative diabetic retinopathy: a univariable and multivariable Mendelian randomization study [J]. Front Nutr. 2023;10:1099807. https://doi.org/10.3389/fnut.2023.1099807
17. Su Z, Wu Z, Liang X, Xie M, Xie J, Li H, Wang X, Jiang F. Diabetic retinopathy risk in patients with unhealthy lifestyle: A Mendelian randomization study [J]. Front Endocrinol (Lausanne). 2022;13:1087965. https://doi.org/10.3389/fendo.2022.1087965
18. Singh P, Adderley N, Subramanian A, Gokhale K, Singhal R, Toulis KA, Bellary S, Nirantharakumar K, Tahrani AA. The impact of bariatric surgery on incident microvascular complications in patients with type 2 diabetes: A matched controlled Population-Based retrospective cohort study [J]. Diabetes Care. 2021;44(1):116–24. https://doi.org/10.2337/dc20-0571
19. Kawai T, Autieri MV, Scalia R. Adipose tissue inflammation and metabolic dysfunction in obesity [J]. Am J Physiol Cell Physiol. 2021;320(3):C375–91. https://doi.org/10.1152/ajpcell.00379.2020
20. Hotamisligil GS. Inflammation and metabolic disorders [J]. Nature. 2006;444(7121):860–7. https://doi.org/10.1038/nature05485
21. Lee YJ, Kim JJ, Kim J, Cho DW, Won JY. The correlation between waist circumference and the Pro-Inflammatory adipokines in diabetic retinopathy of type 2 diabetes patients [J]. Int J Mol Sci. 2023;24(3). https://doi.org/10.3390/ijms24032036
22. Hong SB, Lee JJ, Kim SH, Suh YJ, Han JY, Kim YS, Nam M. The effects of adiponectin and inflammatory cytokines on diabetic vascular complications in obese and non-obese patients with type 2 diabetes mellitus [J]. Diabetes Res Clin Pract. 2016;111:58–65. https://doi.org/10.1016/j.diabres.2015.10.017
23. Yilmaz MI, Sonmez A, Acikel C, Celik T, Bingol N, Pinar M, Bayraktar Z, Ozata M. Adiponectin May play a part in the pathogenesis of diabetic retinopathy [J]. Eur J Endocrinol. 2004;151(1):135–40. https://doi.org/10.1530/eje.0.1510135
24. Han W, Yang S, Xiao H, Wang M, Ye J, Cao L, Sun G. Role of adiponectin in cardiovascular diseases related to glucose and lipid metabolism disorders [J]. Int J Mol Sci. 2022;23(24). https://doi.org/10.3390/ijms232415627
25. Choi HM, Doss HM, Kim KS. Multifaceted physiological roles of adiponectin in inflammation and diseases [J]. Int J Mol Sci. 2020;21(4). https://doi.org/10.3390/ijms21041219
26. Lee BJ, Yu HG. Vitreous hemorrhage after the 25-gauge transconjunctival sutureless vitrectomy for proliferative diabetic retinopathy [J]. Retina. 2010;30(10):1671–7. https://doi.org/10.1097/IAE.0b013e3181dcfb79
27. Kamura Y, Sato Y, Deguchi Y, Yagi F. Iatrogenic retinal breaks during 20-gauge vitrectomy for proliferative diabetic retinopathy [J]. Clin Ophthalmol. 2013;7:29–33. https://doi.org/10.2147/opth.S38784
28. Yang CM, Yeh PT, Yang CH, Chen MS. Bevacizumab pretreatment and long-acting gas infusion on vitreous clear-up after diabetic vitrectomy [J]. Am J Ophthalmol. 2008;146(2):211–7. https://doi.org/10.1016/j.ajo.2008.04.028
29. Sharma T, Fong A, Lai TY, Lee V, Das S, Lam D. Surgical treatment for diabetic vitreoretinal diseases: a review [J]. Clin Exp Ophthalmol. 2016;44(4):340–54. https://doi.org/10.1111/ceo.12752
30. Khuthaila MK, Hsu J, Chiang A, Decroos FC, Milder EA, Setlur V, Garg SJ, Spirn MJ. Postoperative vitreous hemorrhage after diabetic 23-gauge Pars plana vitrectomy [J]. Am J Ophthalmol. 2013;155(4):757–63. https://doi.org/10.1016/j.ajo.2012.11.004
31. Wang S, Liu Y, Du Y, Bao H, Zhu J, Liu X. Influencing factors of low vision 2 years after vitrectomy for proliferative diabetic retinopathy: an observational study [J]. BMC Ophthalmol. 2023;23(1):309. https://doi.org/10.1186/s12886-023-03071-4
32. Ghoraba HH, Zaky AG, Heikal MA, Elgemai EEM, Abd Al Fatah HM. Ophthalmologica. 2017;238(1–2):59–67. https://doi.org/10.1159/000470857. Silicone Oil-Related Visual Loss [J].
33. Federman JL, Schubert HD. Complications associated with the use of silicone oil in 150 eyes after retina-vitreous surgery [J]. Ophthalmology. 1988;95(7):870–6. https://doi.org/10.1016/s0161-6420(88)33080-0
34. Issa R, Xia T, Zarbin MA, Bhagat N. Eye (Lond). 2020;34(3):537–43. https://doi.org/10.1038/s41433-019-0551-7. Silicone oil removal: post-operative complications [J].
35. Grzybowski A, Pieczynski J, Ascaso FJ. Neuronal complications of intravitreal silicone oil: an updated review [J]. Acta Ophthalmol. 2014;92(3):201–4. https://doi.org/10.1111/aos.12212
36. Scheerlinck LM, Schellekens PA, Liem AT, Steijns D, Leeuwen R, INCIDENCE, RISK FACTORS, AND CLINICAL CHARACTERISTICS OF UNEXPLAINED VISUAL LOSS AFTER INTRAOCULAR SILICONE OIL FOR MACULA-ON RETINAL DETACHMENT [J]. Retina. 2016;36(2):342–50. https://doi.org/10.1097/iae.0000000000000711
37. Lou B, Yuan Z, He L, Lin L, Gao Q, Lin X. The Changes of Retinal Saturation after Long-Term Tamponade with Silicone Oil [J]. Biomed Res Int. 2015;2015:713828. https://doi.org/10.1155/2015/713828
38. Kaneko H, Takayama K, Asami T, Ito Y, Tsunekawa T, Iwase T, Funahashi Y, Ueno S, Nonobe N, Yasuda S, Suzumura A, Shimizu H, Kimoto R, Hwang SJ, Terasaki H. Cytokine profiling in the sub-silicone oil fluid after vitrectomy surgeries for refractory retinal diseases [J]. Sci Rep. 2017;7(1):2640. https://doi.org/10.1038/s41598-017-03124-x

39. Wohlfart S, Herth J, Böhmann MB, Pander G, Herbster L, Mier W, Auffarth GU, Uhl P, Hammer M. Exposure to silicone oil endotamponades induces VEGF antibody aggregation and loss of functionality [J]. Invest Ophthalmol Vis Sci. 2024;65(13):56. https://doi.org/10.1167/iovs.65.13.56

Publisher's note