• Clinical Research •

# Clinical characteristics of delayed reversible loss of light perception following vitreoretinal surgery and risk factors influencing time to recovery

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## Abstract

● **AIM:** To evaluate the incidence and clinical features of delayed reversible loss of light perception (D-RLLP) following vitreoretinal surgery, including vitrectomy or silicone oil (SO) removal, and to identify risk factors influencing its recovery time.

● **METHODS:** This study retrospectively reviewed 19 029 cases (19 029 eyes) of vitreoretinal surgery performed between October 2016 and March 2024. Based on strict temporal criteria, postoperative loss of light perception was classified as immediate postoperative loss of light perception (IP-LLP), D-RLLP, and delayed permanent loss of light perception (D-PLLP). The incidence of reversible loss of light perception (RLLP) and its clinical features were evaluated. A multiple linear regression model was constructed to identify the independent predictors of the recovery time of D-RLLP.

● **RESULTS:** Among 19 029 vitreoretinal surgeries, 113 eyes (0.59%) experienced postoperative loss of light perception (LLP), including 32 eyes (28.32%) with IP-LLP, 78 eyes (69.03%) with D-RLLP, and 3 eyes (2.65%) with D-PLLP. The overall incidence of D-RLLP was 0.41% (78/19 029). D-RLLP incidence was significantly higher following SO removal (0.89%, 33/3703) than general vitrectomy (0.29%, 45/15 326; *P*<0.001). In 78 D-RLLP cases, the median recovery time was 47min (interquartile range: 28–71min), with 25.64% recovering in ≤30min, 64.10% in 30–60min, and 10.26% requiring >60min. Multivariate regression identified a history of hypertension (*B*=18.63, *P*=0.046) and intraocular pressure difference (*B*=1.77/min per mm Hg, *P*=0.011) as independent predictors of prolonged recovery time.

● **CONCLUSION:** D-RLLP is a rare clinical event following vitreoretinal surgery, with a median recovery time of 47min, and the vast majority of affected eyes recovered within 1h. SO removal displayed a significantly higher RLLP incidence than vitrectomy. Importantly, a history of hypertension and greater intraocular pressure difference correlated significantly with an extended recovery time.

● **KEYWORDS:** vitreoretinal surgery; delayed reversible loss of light perception; recovery time; hypertension; intraocular pressure difference

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## INTRODUCTION

Loss of light perception (LLP) following vitreoretinal surgery is a serious complication that causes profound visual loss and substantial distress to both patients and surgeons[1]. Pathogenesis is considered multifactorial, involving a combination of anesthetic, ischemic, phototoxic, and mechanical factors[2-3]. Particularly when irreversible, LLP severely impairs quality of life and imposes a significant burden on healthcare systems[4]. The management of postoperative LLP is contentious, with treatment approaches ranging from observation for presumed anesthetic effects to urgent intervention for potential ischemic injury[1-2,5-7]. Despite the empirical use of therapies such as oxygen, vasodilators, and neuroprotective agents, clinical outcomes are inconsistent

and recovery remains unpredictable[3,8]. Of particular note, previous literature has focused predominantly on intraoperative LLP or permanent postoperative no light perception, whereas insufficient attention has been paid to the specific phenotype in which light perception is initially preserved at the end of surgery, subsequently deteriorates to no light perception within a short period, and ultimately recovers. This delayed reversible loss of light perception (D-RLLP) is clinically important for two reasons: first, it can easily be confused with a transient anesthesia-related response; second, its recovery time course and influencing factors remain unclear, thereby limiting the optimization of perioperative monitoring and intervention strategies[2,9-10]. Therefore, based on a large single-center retrospective cohort, this study established a temporal classification framework for postoperative LLP. The aims were to quantify the incidence of postoperative delayed LLP across different subtypes and, with a particular focus on D-RLLP, to characterize its clinical features and identify factors associated with recovery time, thereby providing evidence to support early postoperative recognition, risk stratification, and clinical management.

## PARTICIPANTS AND METHODS

**Ethical Approval** This retrospective study was approved by the Ethics Committee of the Eye Hospital of Wenzhou Medical University (2025-053-K-038) and adhered to the Declaration of Helsinki.

**Study Design and Participants** We reviewed the records of all patients who underwent vitreoretinal surgery [vitrectomy or silicone oil (SO) removal] at our institution between October 2016 and March 2024 and subsequently developed postoperative LLP. Exclusion criteria: Patients were excluded if they had preoperative no light perception, LLP attributed to the immediate effects of retrobulbar anesthesia, or substantial missing data. All surgeries were performed using a three-port 23-gauge vitrectomy system for either vitrectomy or SO removal. Retrobulbar block anesthesia was uniformly administered using a 1:1 mixture of lidocaine hydrochloride 2% 100 mg (Hunan Kelun Pharmaceutical Company, China, National Drug Approval Number: R25041403) and bupivacaine 0.75% 37.5 mg (Humanwell Healthcare Group Company, China, National Drug Approval Number: AB50300311) for all procedures. All procedures were completed by one of four experienced vitreoretinal surgeons (Zhang ZD and three other surgeons). When intraoperative retinal detachment was present, the surgeon determined the tamponade type based on the pathology: SO (RT SIL-OL 5000 cs; Carl Zeiss Meditec AG, Jena, Germany) or perfluoropropane. Repeat vitreoretinal surgeries were performed by the original surgeon.

**Definition and Classification of Postoperative LLP** At the end of surgery and before ocular dressing, all patients underwent an immediate light perception assessment performed by the operating surgeon, in which the fellow eye was occluded, the room lights were turned off, and the operating microscope illumination was directed toward the surgical eye to confirm the presence or absence of light perception. After return to the ward, patients who were fully conscious and able to cooperate reliably underwent repeat assessment in a dark room using the maximum light intensity of an indirect ophthalmoscope, with the fellow eye occluded[7]. Inability to perceive the light stimulus was recorded as LLP. All LLP events were confirmed by one or more physicians.

Postoperative LLP was categorized into three types: 1) immediate postoperative loss of light perception (IP-LLP), defined as LLP identified by the operating surgeon at the end of surgery before ocular dressing; 2) D-RLLP, defined as preserved light perception at the end-of-surgery assessment but LLP detected after return to the ward, with recovery of light perception within 24h following immediate emergency intervention; 3) D-PLLP, defined as preserved light perception at the end-of-surgery assessment but LLP detected after return to the ward and failure to regain light perception despite immediate emergency intervention. The D-RLLP recovery time was defined as the interval between confirmation of LLP and recovery of light perception.

**Emergency Intervention and Monitoring Protocol** 1) Oxygen therapy: Administered hourly for three cycles (10min per cycle, 2–3 L/min flow rate); 2) Vasodilation: Sublingual nitroglycerin application, 0.6 mg (Viatris, National Drug Approval Number: H20171217); 3) Microcirculation improvement: Subcutaneous injection of 2 mL of the compound anisodine (containing anisodine hydrobromide 0.2 mg+ procaine hydrochloride 20 mg; China Resources Zizhu Pharmaceutical Company, National Drug Approval Number: H20000495) at the ipsilateral superficial temporal artery; 4) Neuroprotection: Subcutaneous methylcobalamin injection, 0.5 mg (Weicai Pharmaceutical Company, National Drug Approval Number: H20174048). Visual acuity monitoring was continuous. Intraocular pressure (IOP) was measured at 2h postoperatively and subsequently at 6-hour intervals. Interventions were discontinued if light perception recovered and persisted for >1h. IOP was first measured after the patient's initial report of symptoms and was subsequently reassessed every 6h until discharge.

**Intraoperative IOP Control** All procedures were performed using a three-port 23-gauge pars plana vitrectomy system on the CONSTELLATION Vision System (Alcon Laboratories, USA). Active infusion was applied, with the machine-set infusion pressure routinely maintained at 30 mm Hg. Before SO removal or major intraocular manipulation, the correct position and patency of the infusion cannula were confirmed.

During SO removal, SO was aspirated gradually under continuous infusion to avoid abrupt ocular decompression. During air–fluid exchange, fluid aspiration and air infusion were carefully balanced. Globe tension, retinal artery pulsation, and optic disc perfusion were assessed under the operating microscope, and infusion or air pressure was adjusted when necessary to reduce the risk of marked hypotony or excessive intraoperative IOP elevation.

**Clinical Data Collection** Patient medical records were reviewed, and the following clinical data were obtained: 1) general patient characteristics, including age, sex, history of hypertension, history of diabetes, postoperative systolic blood pressure (SBP) and diastolic blood pressure (DBP), and follow-up time; 2) ocular characteristics, including the involved eye, original diagnosis and/or previous ocular surgery, axial length (AL) measured using the IOL Master 500 (Carl Zeiss), presence of pathologic myopia, type and status of intraocular tamponade, including SO tamponade and the presence or absence of SO emulsification when applicable, preoperative and postoperative best-corrected visual acuity (BCVA) assessed with a phoropter (IS-600, Topcon, Tokyo, Japan), preoperative IOP, B-scan ultrasound findings (UD-7800, Tomey, Tokyo, Japan), widefield fundus photography findings (Optos, Marlborough, MA, USA), and spectral-domain optical coherence tomography findings (Heidelberg Spectralis OCT, Heidelberg, Germany); 3) data related to the present vitrectomy, including the surgical procedure, intraoperative and postoperative complications, LLP recovery time, and the IOP measured immediately at the time LLP was confirmed, which was defined as the onset IOP and determined using a non-contact tonometer (CT-78/78A, Topcon).

Pathologic myopia was defined as an AL>26 mm with characteristic pathologic myopia-related changes in the fundus. The intraocular pressure difference (IOP-d) was defined as onset IOP minus the preoperative baseline IOP. Postoperative follow-ups occurred at 1wk and 1, 3, and 6mo with standardized acuity and IOP assessments.

**Statistical Analysis** All data were analyzed using SPSS Statistics (Version 27.0). BCVA was converted to logarithm of the minimum angle of resolution (logMAR) values prior to analysis. The normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Normally distributed data are presented as mean±standard deviation, while non-normally distributed data are presented as median (interquartile range, IQR). Categorical variables are shown as frequencies (%). The Mann-Whitney *U* test was used for intergroup comparisons of categorical variables against recovery time. To identify predictors for the recovery time (the dependent variable) of D-RLLP, we first performed univariate linear regression. This model was deemed appropriate, despite the non-normal distribution of the dependent variable, because an analysis of the model's residuals confirmed their normality. To build the final multivariate linear regression model, we included variables that were either statistically significant in the univariate analysis ($P$<0.05) or considered clinically relevant. The final model covariates were sex, age, AL, diabetes, pathologic myopia, hypertension, SBP and DBP, 2-hour postoperative IOP, and IOP-d. Multicollinearity was assessed using the variance inflation factor (VIF<5). For all analyses, statistical significance was set at $P$<0.05.

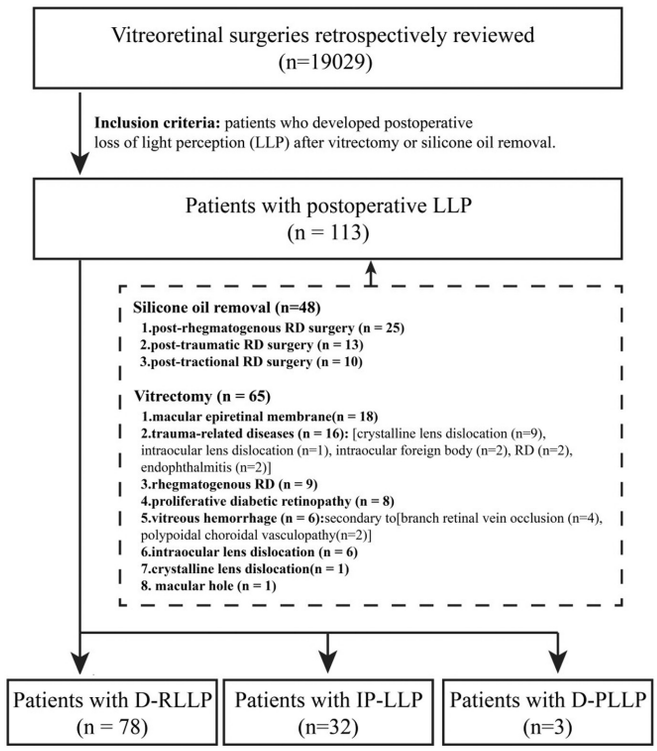


**Figure 1 Flow chart of patient inclusion** LLP: Loss of light perception; RD: Retinal detachment; D-RLLP: Delayed reversible loss of light perception; IP-LLP: Immediate postoperative loss of light perception; D-PLLP: Delayed permanent loss of light perception.

## RESULTS

This study retrospectively reviewed 19 029 vitreoretinal surgeries performed between October 2016 and March 2024. Postoperative LLP cases were identified and classified according to the predefined temporal criteria, as shown in Figure 1. The primary pathologies among these cases are summarized in Table 1. D-RLLP was the predominant delayed LLP phenotype, with an overall incidence of 0.41% in the entire surgical cohort. Representative clinical courses and postoperative imaging findings of D-RLLP and D-PLLP are shown in Figures 2 and 3, respectively.

**D-RLLP** Table 2 summarizes the clinical and perioperative characteristics of the 78 patients with D-RLLP. All procedures were performed under retrobulbar block anesthesia. D-RLLP occurred after vitrectomy in 45 eyes and after SO removal

**Table 1 Primary pathologies among patients with postoperative LLP (*n*=113)**

| Primary pathology | Eyes |
|---|---|
| Eyes with silicone oil tamponade | 48 |
| After surgery for rhegmatogenous retinal detachment | 25 |
| After surgery for traumatic retinal detachment | 13 |
| After surgery for TRD | 10 |
| Trauma-related diseases | 16 |
| Crystalline lens dislocation | 9 |
| Intraocular lens dislocation | 1 |
| Intraocular foreign body | 2 |
| Retinal detachment | 2 |
| Endophthalmitis | 2 |
| Proliferative diabetic retinopathy | 8 |
| Vitreous hemorrhage | 6 |
| Secondary to branch retinal vein occlusion | 4 |
| Secondary to polypoidal choroidal vasculopathy | 2 |
| Macular epiretinal membrane | 18 |
| Rhegmatogenous retinal detachment | 9 |
| Intraocular lens dislocation | 6 |
| Crystalline lens dislocation | 1 |
| Macular hole | 1 |

LLP: Loss of light perception; TRD: Tractional retinal detachment.

in 33 eyes. The incidence was significantly higher after SO removal than after vitrectomy [0.89% (33/3703) *vs* 0.29% (45/15 326), *P*<0.001]. Most patients recovered within 60min, with a median recovery time of 47.00min. The median follow-up duration was 3.27mo.

All 78 eyes with D-RLLP regained light perception within 6h after intervention. The overall median recovery time was 47min (IQR, 28–71min). When stratified by recovery time, 20 eyes (25.64%) recovered within ≤30min, 50 eyes (64.10%) within 30–60min, and 8 eyes (10.26%) after >60min. The vast majority of cases (89.74%) regained light perception within 1h postoperatively. There was no statistically significant difference in recovery time between the two surgical procedures (Table 3).

**Univariate Analysis** At the univariate level, Mann-Whitney *U* tests were performed to assess differences in D-RLLP duration across baseline categorical variables (Table 3). No statistically significant differences in the D-RLLP recovery time were observed for glaucoma (*P*=0.413), sex (*P*=0.238), pathologic myopia (*P*=0.381), SO tamponade (*P*=0.623), surgical method (*P*=0.678), hypertension (*P*=0.103), or diabetes mellitus (*P*=0.972).

Univariate linear regression models quantified potential associations and identified the following significant predictors: hypertension (reference: no hypertension; $\beta$=21.81, *P*=0.012); 2-hour postoperative IOP (per 1 mm Hg increase; $\beta$=1.52, *P*=0.019); IOP-d (per 1 mm Hg increase; $\beta$=1.42, *P*=0.003); and postoperative DBP (per 1 mm Hg increase; $\beta$=0.82, *P*=0.043; Table 4). Male sex (reference: female; $\beta$=-17.01, *P*=0.087) displayed a potential trend toward a longer recovery

**Table 2 Basic characteristics of patients (*n*=78) who experienced reversible loss of light perception following vitreoretinal surgery**

mean±SD, M ($Q_1$, $Q_3$), or *n* (%)

| Characteristic | Value |
|---|---|
| Age (y) | 56.35±12.81 |
| Postoperative DBP (mm Hg) | 75.79±10.35 |
| Postoperative SBP (mm Hg) | 126.81±15.47 |
| Axial length (mm) | 23.75 (22.97, 25.00) |
| Recovery time (min) | 47.00 (28.00, 71.00) |
| Preoperative IOP (mm Hg) | 14.20 (11.18, 16.40) |
| Postoperative 2h IOP (mm Hg) | 9.10 (5.93, 11.88) |
| IOP difference (mm Hg) | -3.80 (-8.40, 0.07) |
| Discharge IOP (mm Hg) | 9.45 (7.15, 12.23) |
| Final follow-up IOP (mm Hg) | 13.95 (11.43, 17.50) |
| Preoperative BCVA (logMAR) | 1.30 (0.62, 2.98) |
| Discharge BCVA (logMAR) | 2.00 (1.10, 3.00) |
| Final follow-up BCVA (logMAR) | 0.95 (0.32, 1.65) |
| Follow-up duration (mo) | 3.27 (0.32, 18.70) |
| Sex | |
| Female | 18 (23.08) |
| Male | 60 (76.92) |
| Hypertension | |
| No | 50 (64.10) |
| Yes | 28 (35.90) |
| Diabetes | |
| No | 60 (76.92) |
| Yes | 18 (23.08) |
| Pathological myopia | |
| No | 67 (85.90) |
| Yes | 11 (14.10) |
| Surgical techniques | |
| Vitrectomy | 45 (57.69) |
| SO removal | 33 (42.31) |
| SO tamponade | |
| No | 66 (84.62) |
| Yes | 12 (15.38) |
| Glaucoma | |
| No | 75 (96.15) |
| Yes | 3 (3.85) |
| Recovery time distribution | |
| ≤30min | 20 (25.64) |
| >30 and ≤60min | 50 (64.10) |
| >60min | 8 (10.26) |

SD: Standard deviation; DBP: Diastolic blood pressure; SBP: Systolic blood pressure; M: Median; $Q_1$: The first quartile; $Q_3$: The third quartile; IOP: Intraocular pressure; BCVA: Best-corrected visual acuity; logMAR: Logarithm of the minimum angle of resolution; SO: Silicone oil.

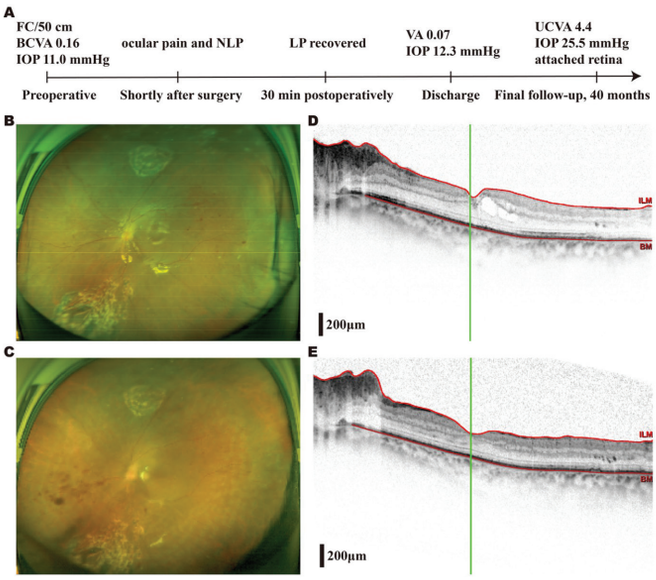


**Figure 2 Representative case of delayed-onset reversible loss of light perception after vitreoretinal surgery** A 63-year-old woman with long-standing type 2 diabetes and hypertension underwent silicone oil removal, internal limiting membrane peeling, retinal photocoagulation, secondary intraocular lens implantation, and intravitreal injection in the left eye under retrobulbar anesthesia. Preoperative visual acuity was finger counting (CF) at 50 cm, best-corrected visual acuity (BCVA) was 0.16, and intraocular pressure (IOP) was 11.0 mm Hg. Shortly after returning to the ward, the patient reported no light perception (NLP) in the operated eye, which was confirmed on examination. After immediate emergency treatment, light perception (LP) recovered 30min postoperatively, and visual acuity (VA) gradually improved to 0.07. At discharge, VA was 0.07 and IOP was 12.3 mm Hg. A: Clinical timeline showing changes in VA and IOP; B: Ultra-widefield fundus photograph before silicone oil removal; C: Ultra-widefield fundus photograph after silicone oil removal; D: OCT image before silicone oil removal; E: OCT image after silicone oil removal. At the final follow-up 40mo postoperatively, uncorrected visual acuity (UCVA) was 4.4 on the 5-point chart, IOP was 25.5 mm Hg, the intraocular lens was well positioned, and the NLP episode had completely resolved. OCT: Optical coherence tomography.

time but did not reach statistical significance. Diabetes mellitus, pathologic myopia, SO tamponade, AL, and SBP demonstrated no statistically significant associations with the D-RLLP recovery time.

**Multivariate Analysis** To identify independent predictors of the D-RLLP recovery time, a multiple linear regression model was constructed. Collinearity diagnostics confirmed no significant multicollinearity (variance inflation factors: 1.148–3.513, all<5). After adjustment *via* multivariate regression, the results indicated that the history of hypertension ($B$=18.63, $P$=0.046) and IOP-d ($B$=1.77, $P$=0.011) were correlated with prolonged RLLP recovery. Postoperative 2h IOP, pathologic myopia, sex, diabetes mellitus, postoperative SBP, and AL

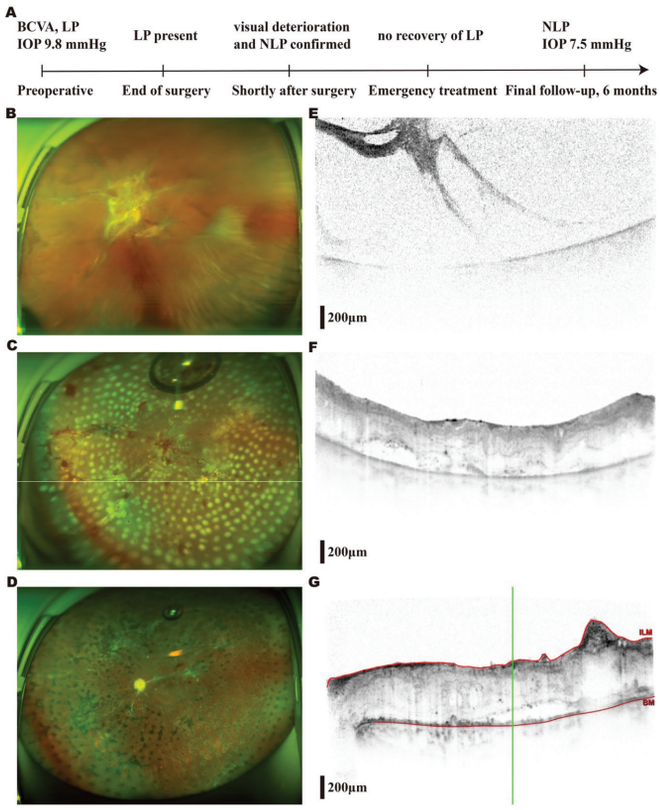


**Figure 3 Representative case of delayed-onset permanent loss of light perception after vitreoretinal surgery** A 57-year-old man with type 2 diabetes presented with blurred vision in the left eye for 1y and was diagnosed with proliferative diabetic retinopathy, tractional retinal detachment, and diabetic cataract in the left eye. He underwent combined pars plana vitrectomy, retinal detachment repair, phacoemulsification, silicone oil tamponade, and panretinal photocoagulation under retrobulbar anesthesia. Preoperative best-corrected visual acuity (BCVA) was light perception (LP), and intraocular pressure (IOP) was 9.8 mm Hg. LP was present at the end of surgery; however, visual deterioration was reported shortly after surgery, and no light perception (NLP) was confirmed in the operated eye. Emergency treatment, including high-concentration oxygen therapy, vasodilator therapy, microcirculation-improving agents, and neuroprotective treatment, failed to restore LP. IOP was 13.6 mm Hg at 2h postoperatively and 16.9 mm Hg on postoperative day 1, while NLP persisted. At the 1-week follow-up, the operated eye remained NLP, with an IOP of 8.4 mm Hg. At 6mo postoperatively, IOP was 7.5 mm Hg, and NLP persisted. The patient subsequently underwent silicone oil removal in the left eye, but LP did not recover. A: Clinical timeline showing changes in visual acuity and IOP; B: Ultra-widefield fundus photograph before pars plana vitrectomy; C: Ultra-widefield fundus photograph after pars plana vitrectomy; D: Ultra-widefield fundus photograph after silicone oil removal; E: OCT image before pars plana vitrectomy; F: OCT image after pars plana vitrectomy; G: OCT image after silicone oil removal. The persistent absence of LP despite emergency intervention and subsequent silicone oil removal was consistent with delayed-onset permanent loss of light perception (D-PLLP). OCT: Optical coherence tomography.

**Table 3 Comparison of patient recovery time by baseline characteristics**

| Characteristic | *n* | Recovery duration (min), M ($Q_1$, $Q_3$) | Statistic | *P* |
|---|---|---|---|---|
| Total | 78 | 47.00 (28.00, 71.00) | | |
| Glaucoma | | | *Z*=-0.82 | 0.413 |
| No | 75 | 47.00 (28.00, 73.50) | | |
| Yes | 3 | 35.00 (27.50, 47.00) | | |
| Sex | | | *Z*=-1.18 | 0.238 |
| Female | 18 | 59.00 (31.25, 85.00) | | |
| Male | 60 | 44.50 (27.25, 61.75) | | |
| Pathological myopia | | | *Z*=-0.88 | 0.381 |
| No | 67 | 47.00 (25.00, 64.50) | | |
| Yes | 11 | 60.00 (36.00, 75.50) | | |
| Surgical method | | | *Z*=-0.41 | 0.678 |
| Vitrectomy | 45 | 38.00 (28.00, 73.00) | | |
| SO removal | 33 | 59.00 (23.00, 64.00) | | |
| SO tamponade | | | *Z*=-0.49 | 0.623 |
| No | 66 | 53.50 (28.25, 71.00) | | |
| Yes | 12 | 36.50 (24.50, 58.75) | | |
| Hypertension | | | *Z*=-1.63 | 0.103 |
| No | 50 | 39.00 (23.50, 62.00) | | |
| Yes | 28 | 59.00 (23.00, 64.00) | | |
| Diabetes | | | *Z*=-0.04 | 0.972 |
| No | 60 | 47.50 (28.75, 64.25) | | |
| Yes | 18 | 40.50 (25.75, 82.75) | | |

M: Median; $Q_1$: The first quartile; $Q_3$: The third quartile; *Z*: Mann-Whitney test; SO: Silicone oil.

**Table 4 Univariable linear regression analysis of factors associated with recovery time**

| Variable | Coefficient ($\beta$) | 95%CI for $\beta$ | *P* |
|---|---|---|---|
| Axial length (mm) | 1.61 | -1.43–4.65 | 0.302 |
| Postoperative 2h IOP (mm Hg) | 1.52 | 0.28–2.76 | 0.019[a] |
| Postoperative DBP (mm Hg) | 0.82 | 0.04–1.61 | 0.043[a] |
| Postoperative SBP (mm Hg) | 0.21 | -0.33–0.74 | 0.453 |
| IOP difference (mm Hg) | 1.42 | 0.50–2.35 | 0.003[b] |
| Sex | | | |
| Female | Ref. | | |
| Male | -17.01 | -36.25–2.24 | 0.087 |
| Diabetes | | | |
| No | Ref. | | |
| Yes | 4.80 | -14.79–24.39 | 0.632 |
| Hypertension | | | |
| No | Ref. | | |
| Yes | 21.81 | 5.29–38.32 | 0.012[a] |
| Pathological myopia | | | |
| No | Ref. | | |
| Yes | 1.76 | -21.99–25.50 | 0.885 |
| Surgical method | | | |
| Vitrectomy | Ref. | | |
| SO removal | 3.88 | -12.83–20.59 | 0.650 |
| SO tamponade | | | |
| No | Ref. | | |
| Yes | 4.95 | -17.93–27.84 | 0.672 |

[a]*P*<0.05; [b]*P*<0.01. CI: Confidence interval; Ref.: Reference category; IOP: Intraocular pressure; DBP: Diastolic blood pressure; SBP: Systolic blood pressure; SO: Silicone oil.

displayed no independent statistical effects on the D-RLLP recovery time (Table 5).

**D-PLLP Cases** A total of 3 eyes were classified as D-PLLP, accounting for 3.70% (3/81) of all delayed LLP cases. All 3 patients reported marked visual deterioration after surgery and were confirmed to have LLP; light perception did not recover despite emergency intervention. All three patients presented with tractional retinal detachment (TRD) secondary to proliferative diabetic retinopathy (PDR) and underwent pars plana vitrectomy with SO tamponade. These three patients also had systemic vascular risk factors and severe ocular ischemic background, such as hypertension, diabetes mellitus, or neovascular glaucoma (NVG). Notably, one case exhibited preoperative NVG, while another developed secondary NVG within 1wk postoperatively.

## DISCUSSION

This study focuses on delayed LLP following vitreoretinal surgery. As vitreoretinal surgery procedures become increasingly common, complications like delayed LLP pose substantial challenges to patient well-being and healthcare resources[1,3,7]. By conducting a large-scale retrospective analysis, we systematically characterized postoperative delayed LLP by differentiating it into reversible forms and permanent forms. Our findings provide detailed insights into the incidence rate, clinical characteristics, and risk factors influencing the D-RLLP recovery time, which collectively contribute to improved clinical management strategies.

**Table 5 Multiple linear regression analysis of factors associated with recovery time**

| Variable | Unstandardized coefficient (*B*) | Adjusted $\beta$ (95%CI) | *P* |
|---|---|---|---|
| Postoperative 2h IOP | -0.33 | -2.10–1.43 | 0.711 |
| Pathological myopia | -30.81 | -69.58–7.96 | 0.124 |
| Postoperative SBP | -0.50 | -1.22–0.22 | 0.177 |
| Postoperative DBP | 1.01 | -0.04–2.06 | 0.063 |
| Surgical method | | | |
| Vitrectomy *vs* SO removal | 9.69 | -6.35–25.74 | 0.240 |
| Sex | | | |
| Male *vs* female | -15.17 | -33.87–3.52 | 0.116 |
| Diabetes | -7.75 | -28.24–12.75 | 0.461 |
| Hypertension | 18.63 | 0.63–36.63 | 0.046[a] |
| IOP difference | 1.77 | 0.44–3.10 | 0.011[a] |
| Axial length | 4.47 | -0.63–9.56 | 0.090 |

Statistically significant variables from univariate linear regression ($P$<0.05) and variables with potential clinical relevance based on empirical evidence were included in the multivariate linear to identify independent predictors. [a]$P$<0.05. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; IOP: Intraocular pressure; SO: Silicone oil.

Our retrospective analysis of 19 029 vitreoretinal surgeries indicated an overall postoperative LLP incidence of 0.59% (113/19 029), this being markedly lower than the 56.5% LLP rate reported by Zheng *et al*[2] following pars plana vitrectomy under sub-Tenon's anesthesia for macular diseases. This discrepancy may be attributable to the differing ambient luminance for postoperative light perception assessment between the two studies. In our protocol, light perception was assessed in a dark room, whereas the evaluation by Zheng *et al*[2] was performed with the operating room lights on. The darker environment enhances ocular sensitivity to light, thus enhancing the accuracy and reliability of the findings. Within the present cohort, D-RLLP constituted 69.03% (78/113) of all LLP cases, indicating that not only is D-RLLP relatively rare (0.41%), but also it is reversible with timely intervention.

Our results demonstrated a statistically significant difference in the incidence of postoperative D-RLLP between the two surgical procedures ($P$<0.001), being significantly higher following SO removal surgery (0.89%) than following vitrectomy (0.29%). This disparity in incidence rates indicates a differing risk profile for postoperative D-RLLP between these surgical techniques. We postulate that this difference may be attributable to the greater technical challenge of maintaining stable IOP during SO removal compared with vitrectomy[11]. Importantly, no statistically significant difference was observed in the D-RLLP recovery time between the two procedures, which is clinically reassuring. It suggested that the underlying pathophysiologic mechanisms leading to light perception loss—such as transient ischemic events or optic nerve conduction block—are likely to be similar, irrespective of the initiating surgery[12-13]. The recovery time of D-RLLP was consistently brief across all cases. The overall median recovery time was 47min, with 89.74% of patients experiencing recovery within 60min. Therefore, proactively communicating the high likelihood of rapid visual restoration (nearly 90% within 1h) is paramount for managing patient expectations and alleviating unnecessary anxiety should this transient event occur. This knowledge significantly enhances the prognostic assessment of postoperative D-RLLP. Recognizing D-RLLP as a highly probable transient complication allows clinicians to confidently predict a favorable outcome and promptly implement necessary intervention measures.

Considering the potential risks associated with delayed LLP and its severe, potentially irreversible visual consequences, our protocol mandates immediate intervention upon detecting postoperative delayed LLP. This comprised oxygen therapy, agents to improve ocular perfusion (vasodilators, microcirculation enhancers), and neuroprotective treatment (*e.g.*, methylcobalamin). Oxygen therapy enhances its delivery to ischemic retinal tissues, potentially mitigating hypoxia-induced damage to photoreceptors and neural components[14]. Vasodilators and microcirculation-improving agents aim to restore the nutrient and oxygen supplies to compromised retinal and optic nerve tissues through enhanced blood flow[15]. Concurrently, neuroprotective treatment supports neural repair and functional recovery, facilitating rapid restoration of light perception[16]. In the present cohort, almost all patients (96.30%) with postoperative delayed LLP demonstrated rapid light perception recovery following this comprehensive intervention. This outcome underscores the critical value of prompt intervention and reinforces adherence to emergency therapeutic protocols for mitigating delayed LLP complications. However, the lack of a placebo control group prevented the exclusion of spontaneous recovery as a contributing factor. Future prospective, controlled studies are essential to rigorously validate the efficacy of these interventions.

Multivariate linear regression analysis demonstrated that IOP-d was an independent predictor for the RLLP recovery time ($B$=1.77, $P$=0.011), with a greater IOP-d correlating with a longer postoperative RLLP resolution time. This implied that a greater IOP-d may delay RLLP recovery by exacerbating ocular ischemia or compromising optic nerve function[17]. Consequently, reducing postoperative IOP could facilitate a more rapid recovery by alleviating pressure-related perfusion deficits, such as through enhanced retinal blood flow[18]. Therefore, routine IOP-lowering therapy, such as anti-glaucoma medications, appears feasible as a first-line intervention for postoperative delayed LLP patients. However, in clinical practice, such treatment is not recommended for patients with an excessively low postoperative IOP (*e.g.*, ≤10 mm Hg) due to safety concerns[19].

This pressure-related mechanism may be particularly relevant in patients with a history of hypertension, which was also identified as an independent predictor of prolonged RLLP recovery time ($B$=18.63, $P$=0.046). A plausible explanation is that chronic microvascular injury induced by long-standing systemic hypertension may create a vulnerable ocular perfusion background, thereby predisposing the eye to severe visual complications[20-22]. Within this compromised haemodynamic setting, acute postoperative IOP fluctuations may represent not merely a simple fluid-dynamic change, but rather a critical trigger for local ischemic decompensation[23-24]. Specifically, chronic hypertension may impair the autoregulatory capacity of the optic nerve head and retinal microcirculation through endothelial dysfunction, vascular wall thickening, and luminal narrowing[20,25]. When postoperative IOP rises abruptly, the resultant increase in mechanical compression at the lamina cribrosa, together with a concomitant reduction in local perfusion, may precipitate focal ischemia and hypoxia, mitochondrial energy failure, ion pump dysfunction, and transient neural conduction block, ultimately manifesting as temporary LLP[23,26-27]. Importantly, because this process may remain below the threshold for irreversible apoptotic injury at an early stage, timely correction of the abnormal haemodynamic disturbance and restoration of local perfusion may still permit recovery of neural conduction function, thereby accounting, at least in part, for the reversible nature of RLLP.

Postoperative permanent LLP following vitreoretinal surgery is exceedingly rare, with only three cases identified (0.016%) in the present study cohort of 19 029 procedures. All three patients presented with TRD secondary to PDR and underwent pars plana vitrectomy with SO tamponade. Notably, one case exhibited preoperative NVG, while another developed secondary NVG within 1wk postoperatively. These findings suggested that permanent LLP may correlate with severe systemic and ocular conditions—particularly advanced PDR causing TRD—that induce extensively compromised vascularity and diminished neurological reserve[28]. Meticulous perioperative management, including stringent blood pressure and glycemic control, is recommended for patients with severe PDR[29]. For patients with established or potential NVG, perioperative adjunctive anti-vascular endothelial growth factor therapy may ameliorate disease severity and mitigate the risk of surgical-related complications[29].

This study has several limitations. As a retrospective design, it is susceptible to biases such as potential selection bias due to variations in surgical techniques across the four experienced surgeons involved. While the large sample size represents a strength, heterogeneity within the dataset may contribute to inter-batch variation. Furthermore, the absence of a placebo group for LLP treatments prevents definitive conclusions about the necessity and effectiveness of the emergency intervention, and the long-term impact of RLLP on visual function outcomes remains unclear. Another limitation is that continuous intraoperative IOP monitoring was not routinely performed or quantitatively documented in this retrospective cohort. Therefore, the potential contribution of intraoperative IOP fluctuation, particularly during SO removal or air–fluid exchange, could not be directly evaluated. Future prospective studies incorporating real-time intraoperative IOP monitoring are warranted to clarify this issue. Addressing these limitations warrants rigorously designed prospective randomized clinical trials in future research.

In summary, D-RLLP is a rare clinical event following vitreoretinal surgery, with a median recovery time of 47min. Patients undergoing SO removal exhibited a significantly higher incidence of RLLP (0.89%) compared with those receiving vitrectomy (0.29%; $P$<0.001), though recovery times did not differ between these groups. Importantly, both hypertension and the IOP-d were identified as independent predictors for the recovery time of RLLP. Specifically, hypertension and a greater IOP-d correlated significantly with an extended recovery time.

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