Ghost cell glaucoma after intravitreous injection of ranibizumab in proliferative diabetic retinopathy

CURRENT STATUS: UNDER REVIEW

Jun XU
Beijing Tongren Hospital

Meng ZHAO zhaomeng_jasmine@sina.com
Beijing Tongren eye center
Corresponding Author
ORCiD: 0000-0002-3705-3056

Jipeng LI
Beijing Tongren Hospital

Ningpu LIU
Beijing Tongren Hospital

DOI:
10.21203/rs.2.11864/v1

SUBJECT AREAS
Ophthalmology

KEYWORDS
intravitreous injection, vitrectomy, proliferative diabetic retinopathy, ghost cell glaucoma
Abstract

BACKGROUND: Intravitreous injection of anti-vascular endothelial growth factor agents has been widely used as an adjunctive method to vitrectomy in eyes with vitreous hemorrhage due to proliferative diabetic retinopathy (PDR). Here we reported a series of patients with PDR who developed ghost cell glaucoma after intravitreous injection and analyzed the potential factors that might be related to the development of ghost cell glaucoma.

METHODS: Retrospective case series study. A total of a consecutive 71 eyes of PDR patients who received vitrectomy after intravitreous injection of ranibizumab (IVR) from January 2015 to January 2017 were enrolled in the study. Intraocular pressure (IOP) was recorded before and after intravitreous injection. Medical records of patients were recorded and investigated. The onset and treatment of ghost-cell glaucoma were recorded. RESULTS: There were 8 out of 71 eyes of the PDR patients developed ghost cell glaucoma after they received IVR. The interval between detection of elevation of IOP and intravitreous injection ranged from 0 to 2 days. There were 2 eyes had IOP greater than 30mmHg at the first IOP measurement at 30 minutes after IVR and remained elevated thereafter. The mean maximum IOP was 46.5±8.0 mmHg. There were 5 patients required medicine and 3 patients required additional paracentesis to control IOP. All patients gained normal IOP after vitrectomy and did not require medicine for lowering IOP ever since. The binary backward stepwise logistic regression model showed that the presence of ghost cell glaucoma was associated with tractional retinal detachment (RR= 4.60 2.02~8.48, p= 0.004) and fibromembrane involving disk (RR=-3.57 -7.59~-0.92, p=0.03) (AIC= 39.23 AUC=0.88). CONCLUSION: The ghost cell glaucoma can occur after IVR among PDR patients who are required vitrectomy. Attention on postoperative IOP should be paid to patients with PDR undergoing vitrectomy who receive a preoperative intravitreous injection of anti-VEGF agents, especially in patients with severe PDR.
Background

Severe vision impairment with proliferative diabetic retinopathy (PDR) frequently results from complications due to neovascularization and fibrovascular proliferation[1]. Non-clearing vitreous hemorrhage, tractional retinal detachment (TRD), extensive fibrovascular proliferation are common indications for vitrectomy in patients with PDR[2, 3]. Intravitreous injection of anti-vascular endothelial growth factor (VEGF) agents has been widely used as an adjunctive method to vitrectomy in eyes with vitreous hemorrhage due to PDR[4-8]. It has been proved to be a safe method and to be effective to decrease the overall surgery time, lower the rate of intraoperative complications and reduce the occurrence of postoperative hemorrhage[9, 10].

The development of ghost cell glaucoma in patients with PDR after intravitreous injection was rarely as reported[7, 11]. Here we reported a series of patients with PDR who developed ghost cell glaucoma after intravitreous injection and analyzed the potential factors that might be related to the development of ghost cell glaucoma.

Methods

This study was a retrospective case series of consecutive patients with a diagnosis of PDR who were injected with intravitreous ranibizumab before the surgery of a planned vitrectomy. Records of 78 patients with PDR between January 2015 to January 2017 in the Southern Section of the Beijing Tongren Eye center were retrospectively reviewed. This study was adhered to the tenets of the Declaration of Helsinki and was approved by the Ethics Committee of Beijing Tongren Hospital.

Inclusion criteria: 1)Patients diagnosed with PDR 2)patients took an intravitreous injection of ranibizumab before vitrectomy. 3)Records with intraocular pressure (IOP) values measured before and after intravitreous injection of ranibizumab (IVR).

Exclusion criteria: 1) patients failed to finish at least 1 mo follow-up; 2) patients with
history of preexisted open-angle glaucoma; 3) patients with preexisted narrow/closed angle; 4) patients received an intravitreous or subtenon injection of corticosteroids or steroid eye drop within the latest six months; 5) uncontrolled neovascular glaucoma by at least 3 kinds of antiglaucoma medicine. 68 patients were enrolled in this study. The three cases without IOP measurements before or after IVR did not develop ghost cell glaucoma after IVR and were ruled out.

Examinations at Baseline: All patients underwent comprehensive ophthalmological examinations, including best-corrected visual acuity (BCVA) testing using a decimal visual acuity (VA) chart, slit-lamp biomicroscopy, IOP measurement with an air tonometer (Nidek Tonoref 3), dilated fundus examination with indirect ophthalmoscopy, color fundus photograph with a digital fundus camera, optical biometry (The ZEISS IOLMaster, Carl-Zeiss Meditec, Dublin, California, USA), optic coherent tomography (OCT) B scan. OCT images were obtained by spectral-domain OCT (Carl-Zeiss Meditec, Dublin, California, USA). Gonioscopy was considered when iris neovascularization was found. BCVA, the axial length, the presence of posterior vitreous detachment (posterior vitreous detachment, PVD, was defined as the presence of a Weiss ring and visible posterior vitreous cortex under the slit-lamp biocular biomicroscopy examination by the same surgical doctor[12], or B scan verified the presence of PVD, PVD was also confirmed by findings in triamcinolone acetonide-assisted vitrectomy), the history of diabetes mellitus, history of visual acuity decrease, use of insulin, history of retinal photocoagulation for diabetic retinopathy, sex, age, refraction, BCVA, presence of iris neovascularization, intraocular lens (IOL), dense vitreous hemorrhage that obscure the view of optic disc and details of fundus, tractional retinal detachment that threatened the central vision or caused repeated vitreous hemorrhage, fibrovascular membrane involving the disk, presence of macular edema were recorded as the baseline data.
Intravitreous injection of ranibizumab (IVR): An intravitreous injection of ranibizumab 0.5 mg injection was performed 1-10 days before vitrectomy. For IVR, topical anesthesia was applied, and 10% povidone-iodine was used to scrub eyelids and lashes, 5% povidone was applied for more than 90 seconds, and a sterile lib speculum was put between the eyelids. Ranibizumab (0.5mg/0.05ml, Lucentis; Novartis AG, Bülach, Switzerland) was injected into the vitreous cavity through the inferior sclera using a 30-gauge needle, 3.5 mm posterior to the corneal limbus. Sterile cotton was pressed over the injection site for more than 60 seconds to prevent leakage.

The 3-port pars plana vitrectomy: All patients underwent a 3-port pars plana 23-gauge vitrectomy under general anesthesia. Phacoemulsification surgery was performed before vitrectomy in case of necessary determined by the surgeon before surgery. The presence of PVD, a tractional retinal detachment that threatens central vision or caused repeated vitreous hemorrhage, fibrovascular membrane involving the disk were confirmed in the vitrectomy after removal of dense vitreous hemorrhage and recorded. The silicon oil tamponation and laser points during vitrectomy were recorded.

IOP measurements: All patients underwent a complete series of IOP (intraocular pressure, IOP) measurement with an air tonometer (Nidek, Tonoref 3). Baseline IOP was defined as the IOP measurement before IVR. The following IOP were measured during the follow-up. IOP was measured 30min, 2h, 1d, 2d, 3d after intravitreous injection. If elevated IOP occurred, IOP was measured twice a day until the IOP was controlled. The ghost cell glaucoma was defined as the presence of both high IOP and ghost cell in the anterior chamber proved by characters of the red cell in the anterior chamber[13, 14] or biopsy[15].

Follow-up visits were scheduled at 1,2,7,14 and 30 days after the initial vitrectomy. BCVA, IOP, dilated fundus examination and occurrence of any surgery-related complication was
recorded. The potential complications of vitrectomy were listed as following: endophthalmitis, retinal detachment, vitreal hemorrhage, hypotony, secondary glaucoma.

Statistical Analysis. Statistical analysis was performed using R version 3.20 (http://www.R-project.org). Patient characteristics were retrieved from their medical charts and recorded in Epidata EntryClientversion2.0.3.15 (http://epidata.dk). BCVA results were converted to a logMAR value for statistical analysis. Mean and standard deviation (SD) were calculated for continuous variables with normal distribution. Median with quartiles was calculated for continuous variables with a non-normal distribution. The t-test or Mann-Whitney U test was carried out for continuous variables. The Chi-square test or Fisher’s exact test was carried out for discrete data. To explore the potential factors that may influence the occurrence of ghost cell glaucoma, we divided the patients into two groups, patients with ghost cell glaucoma and patients without ghost cell glaucoma after IVR, several factors including duration of diabetes mellitus, onset of decreased vision, use of insulin, pre-existence of retinal photocoagulation for DR, refraction error, axial length, sex, age, presence of PVD, presence of iris neovascularization (NVI), tractional retinal detachment that threatened the central vision or caused repeated vitreous hemorrhage, IOL, fibrovascular membrane involving the disk, presence of clinical significant macular edema, laser points during vitrectomy, tamponation of silicon oil, interval between IVR and vitrectomy were compared between eyes with development of ghost cell glaucoma and eyes without the development of ghost cell glaucoma (Table 1). Variables were further enrolled in a binary backward stepwise logistic regression model. One variable was included or excluded from the model each time by comparing the Akaike information criterion (AIC) value, and the model that had the lowest AIC was chosen. The model was accessed by receiver operating characteristic curve (ROC curve).

Results
A total of 71 eyes of 68 patients were included. Among them, 3 patients received an intravitreous injection in both eyes. Patients baseline characteristics are presented in Table 1.

There were 8 out of 71 eyes of the PDR patients developed ghost cell glaucoma after they received IVR. The basic characteristics of the patients developed ghost cell glaucoma were listed in Table 1. The interval between the onset of visual symptom and the intravitreous injection was ranged from 0.2 m to 24 m, with a median of 4 [1st quality was 2, 3rd quality was 7]m. There were 7 out of 8 patients who failed to complete pan-retinal photocoagulation due to dense vitreous hemorrhage. There were 3 patients who were presented with tiny iris neovascularization on the pupil margin and normal IOP before intravitreous injection. There were 3 patients with dense vitreous hemorrhage and sight threatened tractional retinal detachment. The interval between detection of elevation of IOP and intravitreous injection ranged from 0 to 2 days. Among them, there were 6 eyes developed ghost cell glaucoma within 1 day after intravitreous injection there were 2 eyes developed ghost cell glaucoma 1 day later after IVR. Among the 6 eyes who had elevated IOP within the first day after IVR, there were 4 eyes showed normal IOP at 30 min after IVR and developed ghost cell glaucoma later after IVR. The other 2 eyes had IOP greater than 30mmHg at 30 min as the first IOP measurement after IVR and remained elevated. The mean maximum IOP was 46.5±8.0 mmHg. There were 5 patients required methazolamide, brimonidine tartrate, and carteolol hydrochloride to control IOP and 3 patients required additional paracentesis to control IOP. All patients gained normal IOP after vitrectomy and did not have medicine for lowering IOP. There were 3 patients had persistent ghost cell in the anterior chamber 1 week after vitrectomy. At 1-month follow-up, all patients’ IOP remained normal.

To confirm the potential factors that may be related to the development of ghost cell
glaucoma, the data was divided into two groups by the presence of post-IVR ghost cell glaucoma. Variables in Table 1 with a p-value < 0.4 in the two independent sample comparison were selected in the initial logistic regression model, including age, onset of vision decrease, history of retinal photocoagulation, presence of iris neovascular, presence of tractional retinal detachment, pseudophakic eye, laser burns of photocoagulation during vitrectomy, interval between IVR and vitrectomy. Variables were further enrolled in a binary backward stepwise logistic regression model. One variable was included or excluded from the model each time by comparing the Akaike information criterion (AIC) value, and the model that had the lowest AIC was chosen. The presence of ghost cell glaucoma was associated with tractional retinal detachment (RR = 4.60 [2.02~8.48], p = 0.004) and fibromembrane involving disk (RR = -3.57 [-7.59~0.92], p = 0.03) (AIC = 39.23 AUC = 0.88). The logistic regression ROC curve was shown in figure 1.

Discussion

Here we report a group of patients with PDR who developed ghost cell glaucoma after intravitreous injection of ranibizumab and show some potential factors that may be related to the development of ghost cell glaucoma.

The ghost cell glaucoma is rare[7, 11] after IVR in PDR patients. It has been reported that sustained IOP elevation after IVR caused by ghost cell glaucoma only occurs in 3% PDR eyes with vitreous hemorrhage [16], but do not occur in eyes with diabetic macular edema or PDR without vitreous hemorrhage [17, 18]. In our series of patients with PDR, the incidence of ghost cell glaucoma after IVR (8/71) is higher than previously reported[7, 11] but much lower than eyes with PDR treated after vitrectomy [19]. In L. Liu et al’s study[19], the incidence of ghost cell glaucoma after IVR in eyes with PDR treated after vitrectomy is 3 out of 8 eyes. It has been reported that ghost cell glaucoma generally occurs where there are vitreous hemorrhage and disruption of the anterior hyaloid surface
following surgery or trauma\textsuperscript{15, 20–22}. Although the ghost cell glaucoma can occur after intravitreous injection while the anterior hyaloid surface of the patients was relatively intact such as the eyes in our series, higher incidence of ghost cell glaucoma after vitrectomy in L. Liu et al’s study may indicate the ghost cell could gain entrance to the anterior chamber more easily in the condition of the removal of vitreous by vitrectomy. The onset of ghost cell glaucoma after IVR in our series is different from eyes without IVR. It has been reported the ghost cell can occur in the vitreous and enter the anterior chamber in phakic eyes spontaneously between 18 months and 4 years after vitreous hemorrhage\textsuperscript{13, 14}. The development of ghost cell glaucoma within 1 week after intravitreous injection of bevacizumab in eyes with postoperative vitreous hemorrhage after vitrectomy for PDR has also been reported\textsuperscript{19}. In our study, the development of ghost cell glaucoma may be found right after IVR or 1–2 days delay. The development of ghost cell glaucoma was more rapidly in eyes with IVR compared with eyes without intravitreous injection\textsuperscript{13}. It may be suggested that IVR may be a risk factor for the development of ghost cell glaucoma in eyes with vitreous hemorrhage.

The development of ghost cell glaucoma after one single intravitreous injection in our series is 11.27%, which is higher than previously reported as 0.7–2.2\%\textsuperscript{7, 11, 23}. The late complications of PDR result from the development of posterior vitreous detachment and contraction of the fibrovascular membranes. Tractional retinal detachment is a common indication for vitrectomy in eyes with PDR\textsuperscript{3, 24}. Our result suggested the presence with tractional retinal detachment and fibrovascular membrane on the optic disc are a potential factor that may be related to the development of ghost cell glaucoma in our study. Both tractional retinal detachment and fibrovascular membrane on optic disk may result from adherent of vitreous to the retina, which provides a scaffold for retinal
neovascularization to grow into vitreous and can cause repeated vitreous hemorrhage[25]. Most cases in our study have a long-standing vitreous hemorrhage. Long-standing vitreous hemorrhage or repeated vitreous hemorrhage may be the source of a large amount of ghost cell in vitreous[13, 19] in our series. The higher incidence of ghost cell glaucoma in our series of PDR patients may due to the high amount of ghost cell in the vitreous body. It should be proved by further large sample study.

Vitrectomy is effective as a treatment for ghost cell glaucoma by cleaning both the vitreous hemorrhage and reservoir of ghost cell in the vitreous as previously reported[15, 26]. In our study, All patients gained normal IOP after vitrectomy without medicine for lowering IOP.

Vitreous hemorrhage is an important factor to develop ghost cell glaucoma[15, 20-22], but we failed to show the presence of vitreous hemorrhage is associated with the development of ghost cell glaucoma. Most of PDR patients with vitreous hemorrhage in our series had either tractional retinal detachment or fibrovascular membrane. Only a few of PDR patients in our series had vitreous hemorrhage without tractional retinal detachment or fibrovascular membrane. Further study with a larger sample is required to confirm whether vitreous hemorrhage alone in PDR patients was related to the development of ghost cell glaucoma after intravitreous injection.

The limitation of this study was due to it was a retrospective case series. We failed to include enough sample with pseudophakic eye or vitreous hemorrhage without tractional retinal detachment. The selected bias was also presented due to the patients enrolled in this study was in a tertiary hospital. More complicated cases with longer duration may be enrolled in this study, which may contribute to the high occurrence of ghost cell glaucoma after intravitreous injection. The use of air tonometer to record IOP may influence the accuracy of the IOP, but the tendency of IOP changes measured by one air tonometer may
reflect the real changes of IOP. A Goldmann tonometer may be included in further research dealing with ghost cell glaucoma after intravitreous injection. We failed to obtain each biopsy result of patients who developed ghost cell glaucoma in our series. There were only 2 cases with the biopsy proved ghost cell. There were 6 cases with clinically proved ghost cell in the anterior chamber by slit lamp examination.

Conclusions

In summary, this study reports a series of patients with PDR who developed ghost cell glaucoma after intravitreous injection of ranibizumab. The development of ghost cell glaucoma varies from 0–2 days and is mostly anticipated within 1 day after intravitreous injection. Presence of tractional retinal detachment and fibrovascular membrane on the optic disc are factors may be related to the development of ghost cell glaucoma after intravitreous injection. Attention on postoperative IOP should be paid to patients with PDR undergoing vitrectomy who receive a preoperative intravitreous injection of anti-VEGF agents, especially in patients with severe PDR.

Abbreviations

Akaike information criterion AIC
best-corrected visual acuity BCVA
fluorescence angiography FA
intraocular pressure IOP
intravitreous injection of ranibizumab IVR
intraocular lens IOL
iris neovascularization NVI
optical coherence tomography OCT
posterior vitreous detachment PVD
receiver operating characteristic curve ROC curve
standard deviation SD

Declarations

Ethics approval and consent to participate:
Ethical approval was given by the medical ethics committee of Beijing Tongren Hospital. It does not have reference number as a retrospective case series study.

Consent for publication: Not applicable

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

Competing interests
The authors declare that they have no competing interests.

Funding
Not applicable.

Authors’ contributions
All authors read and approved the final manuscript. MZ collected and analyzed the data, she was the one major contributor in writing the manuscript. XJ interpreted the data and performed the vitrectomy surgeries, he was one major contributor in writing and reviewing the manuscript. JPL performed the vitrectomy surgeries and reviewed the manuscript. NPL reviewed the manuscript and contribute in writing the manuscript.

Acknowledgments
We acknowledge the residents who worked as assistants.

References
1. Klein BE: Overview of epidemiologic studies of diabetic retinopathy. Ophthalmic
2. Castillo J, Aleman I, Rush SW, Rush RB: Preoperative Bevacizumab Administration in Proliferative Diabetic Retinopathy Patients Undergoing Vitrectomy: A Randomized and Controlled Trial Comparing Interval Variation. American journal of ophthalmology 2017, 183:1-10.

3. Newman DK: Surgical management of the late complications of proliferative diabetic retinopathy. Eye (London, England) 2010, 24(3):441-449.

4. Spaide RF, Fisher YL: Intravitreal bevacizumab (Avastin) treatment of proliferative diabetic retinopathy complicated by vitreous hemorrhage. Retina (Philadelphia, Pa) 2006, 26(3):275-278.

5. Rizzo S, Genovesi-Ebert F, Di Bartolo E, Vento A, Miniaci S, Williams G: Injection of intravitreal bevacizumab (Avastin) as a preoperative adjunct before vitrectomy surgery in the treatment of severe proliferative diabetic retinopathy (PDR). Graefe’s archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie 2008, 246(6):837-842.

6. di Lauro R, De Ruggiero P, di Lauro R, di Lauro MT, Romano MR: Intravitreal bevacizumab for surgical treatment of severe proliferative diabetic retinopathy. Graefe’s archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie 2010, 248(6):785-791.

7. Randomized clinical trial evaluating intravitreal ranibizumab or saline for vitreous hemorrhage from proliferative diabetic retinopathy. JAMA ophthalmology 2013, 131(3):283-293.

8. Martinez-Zapata MJ, Marti-Carvajal AJ, Sola I, Pijoan JI, Buil-Calvo JA, Cordero JA, Evans JR: Anti-vascular endothelial growth factor for proliferative diabetic retinopathy. Cochrane Database Syst Rev 2014(11):CD008721.
9. Sousa DC, Leal I, Costa J, Vaz-Carneiro A: Analysis of the Cochrane Review: Anti-vascular Endothelial Growth Factor for Prevention of Postoperative Vitreous Cavity Hemorrhage after Vitrectomy for Proliferative Diabetic Retinopathy. Cochrane Database Syst Rev. 2015;8:CD008214.]. Acta Med Port 2017, 30(7-8):513-516.

10. Zhao LQ, Zhu H, Zhao PQ, Hu YQ: A systematic review and meta-analysis of clinical outcomes of vitrectomy with or without intravitreal bevacizumab pretreatment for severe diabetic retinopathy. Br J Ophthalmol 2011, 95(9):1216-1222.

11. Arevalo JF, Wu L, Sanchez JG, Maia M, Saravia MJ, Fernandez CF, Evans T: Intravitreal bevacizumab (Avastin) for proliferative diabetic retinopathy: 6-months follow-up. Eye (London, England) 2009, 23(1):117-123.

12. Kicova N, Bertelmann T, Irle S, Sekundo W, Mennel S: Evaluation of a posterior vitreous detachment: a comparison of biomicroscopy, B-scan ultrasonography and optical coherence tomography to surgical findings with chromodissection. Acta ophthalmologica 2012, 90(4):e264-268.

13. Frazer DG, Kidd MN, Johnston PB: Ghost cell glaucoma in phakic eyes. Int Ophthalmol 1987, 11(1):51-54.

14. Brooks AM, Gillies WE: Haemolytic glaucoma occurring in phakic eyes. Br J Ophthalmol 1986, 70(8):603-606.

15. Montenegro MH, Simmons RJ: Ghost cell glaucoma. Int Ophthalmol Clin 1995, 35(1):111-115.

16. Diabetic Retinopathy Clinical Research N: Randomized clinical trial evaluating intravitreal ranibizumab or saline for vitreous hemorrhage from proliferative diabetic retinopathy. JAMA ophthalmology 2013, 131(3):283-293.

17. Bressler SB, Beaulieu WT, Glassman AR, Gross JG, Jampol LM, Melia M, Peters MA, Rauser ME: Factors Associated with Worsening Proliferative Diabetic Retinopathy in Eyes
Treated with Panretinal Photocoagulation or Ranibizumab. *Ophthalmology* 2017, 124(4):431–439.

18. Writing Committee for the Diabetic Retinopathy Clinical Research N, Gross JG, Glassman AR, Jampol LM, Inusah S, Aiello LP, Antoszyk AN, Baker CW, Berger BB, Bressler NM et al: *Panretinal Photocoagulation vs Intravitreous Ranibizumab for Proliferative Diabetic Retinopathy: A Randomized Clinical Trial. JAMA* 2015, 314(20):2137–2146.

19. Liu L, Wu WC, Yeung L, Wang NK, Kuo YH, Chao AN, Chen KJ, Chen TL, Lai CC, Hwang YS et al: *Ghost cell glaucoma after intravitreal bevacizumab for postoperative vitreous hemorrhage following vitrectomy for proliferative diabetic retinopathy. Ophthalmic surgery, lasers & imaging: the official journal of the International Society for Imaging in the Eye* 2010, 41(1):72–77.

20. Campbell DG, Simmons RJ, Grant WM: *Ghost cells as a cause of glaucoma. American journal of ophthalmology* 1976, 81(4):441–450.

21. Campbell DG, Simmons RJ, Tolentino FI, McMeel JW: *Glaucoma occurring after closed vitrectomy. American journal of ophthalmology* 1977, 83(1):63–69.

22. Campbell DG: *Ghost cell glaucoma following trauma. Ophthalmology* 1981, 88(11):1151–1158.

23. Arevalo JF, Lasave AF, Wu L, Maia M, Diaz-Llopis M, Alezzandrini AA, Brito M, Pan-American Collaborative Retina Study G: *INTRAVITREAL BEVACIZUMAB FOR PROLIFERATIVE DIABETIC RETINOPATHY: Results From the Pan-American Collaborative Retina Study Group (PACORES) at 24 Months of Follow-up. Retina (Philadelphia, Pa)* 2017, 37(2):334–343.

24. Farahvash MS, Majidi AR, Roohipoor R, Ghassemi F: *Preoperative injection of intravitreal bevacizumab in dense diabetic vitreous hemorrhage. Retina (Philadelphia, Pa)* 2011, 31(7):1254–1260.

25. Bressler SB, Beaulieu WT, Glassman AR, Gross JG, Jampol LM, Melia M, Peters MA,
Rauser ME, Diabetic Retinopathy Clinical Research N: Factors Associated with Worsening Proliferative Diabetic Retinopathy in Eyes Treated with Panretinal Photocoagulation or Ranibizumab. Ophthalmology 2017, 124(4):431-439.

26. Abu el-Asrar AM, al-Obeidan SA: Pars plana vitrectomy in the management of ghost cell glaucoma. Int Ophthalmol 1995, 19(2):121-124.

Figures

![Image of a receiver operating characteristic curve](image.png)

Figure 1

The receiver operating characteristic curve for the model of logistic regression analysis for potential factors associated with development of ghost cell glaucoma.

Supplementary Files

This is a list of supplementary files associated with the primary manuscript. Click to download.

table1supp.docx