Clinical Report

Journal of INTERNATIONAL MEDICAL RESEARCH

Journal of International Medical Research
2017, Vol. 45(6) 2065–2071

DOI: 10.1177/0300060517708942
journals.sagepub.com/home/imr
SAGE

Early vitrectomy for dense vitreous hemorrhage in adults with non-traumatic and non-diabetic retinopathy

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Abstract

Objective: To evaluate the etiologies for dense vitreous hemorrhage in adults with non-traumatic and reveal management of early vitrectomy for the disease.

Methods: Study included 105 eyes from 105 patients. Outcome measures were etiologies of vitreous hemorrhage, formation of retinal and/or disk neovascular membrane (NVM), incidence of retinal tear and detachment, visual acuity (VA) and postoperative complications.

Results: Mean time between presentation and surgery was 7.1 days. The most common etiologies were retinal vein occlusion (RVO) (58.1%), retinal tear (22.9%) and retinal vasculitis (10.4%). Most RVO (77.0%) and retinal vasculitis (72.7%) eyes were associated with retinal and/or disk NVM. Retinal tear and retinal detachment was found in 24 and 48 eyes, respectively. VA improved significantly from 1/70 to 0.6 following vitrectomy. The most common postoperative complication was cataract (28.6%).

Conclusion: RVO, retinal tear and retinal vasculitis were the most common causes of dense vitreous hemorrhage. Early vitrectomy has a good outcome with acceptable complication rates in this setting.

Keywords

Early vitrectomy, dense vitreous hemorrhage, etiology, complication

Date received: 28 August 2016; accepted: 18 April 2017

Introduction

Vitreous hemorrhage is one of the most common ocular disorders, causing a sudden, painless loss of vision. Patients with a fundus-obscuring vitreous hemorrhage, which is generally caused by a retinal tear or retinal vasculopathy, face a classic clinical dilemma.[1] Many clinicians favor a conservative treatment, which involves keeping the head in an upright position

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combined with immobilization, to promote settling and gradual absorption of the blood.[2,3] However, the conservative approach for dense vitreous hemorrhage has potential risks such as retinal detachment, glaucoma and pigmentary retinopathy.[4,5] To avoid these complications, many clinicians prefer more aggressive treatment with a vitrectomy or early vitrectomy.

As there have been few reports focused on postoperative visual acuity (VA) and complications arising from vitreous hemorrhage treatment, it is difficult to weigh the risks and benefits of surgery. A previous study showed the most common etiology of vitreous hemorrhage in children was blunt or penetrating trauma,[6] while the main causes of vitreous hemorrhage in adults were posterior vitreous detachment and diabetic retinopathy.[2] While multiple studies have investigated the prognosis for vitreous hemorrhage caused by proliferative diabetic retinopathy or trauma, less is known about the outcomes for patients with dense vitreous hemorrhage, even though it frequently causes more devastating complications. Therefore, we undertook this study to better understand the etiologies and visual prognosis following early vitrectomy for dense vitreous hemorrhage in adults with non-traumatic and non-diabetic retinopathy.

Methods

Ethics approval and patient recruitment

The study was approved by the institutional review board of Shandong Eye Institute, Jinan, China and conformed to the guidelines of the Declaration of Helsinki. Medical records of patients who underwent early vitrectomy for dense vitreous hemorrhage between March 1, 2012 and March 1, 2014 were reviewed. All patients underwent a comprehensive ophthalmic examination including VA, applanation tonometry, slit-lamp examination, gonioscopy, indirect ophthalmoscopy and B-scan ultrasound. The severity of vitreous hemorrhage was scored on a 5-point scale, which was modified from a previous study: Grade 0 (no vitreous hemorrhage); Grade 1 (minimal vitreous hemorrhage, optic disk and retinal vessels were clearly visible); Grade 2 (mild vitreous hemorrhage, most of the optic disk and retinal vessels were visible); Grade 3 (moderate vitreous hemorrhage, optic disk, or retinal vessels were barely visible); Grade 4 (severe vitreous hemorrhage was too dense to allow visualization of the optic disk).[7,8] We included cases of adults who underwent early vitrectomy for vitreous hemorrhage of Grade 3 or 4 that had a follow-up of at least 6 months. We excluded cases of vitreous hemorrhage caused by trauma or proliferative diabetic retinopathy.

Surgery and postoperative assessment

For these dense vitreous hemorrhage eyes, surgery was carried out once a retinal tear or detachment was detected. If no retinal tear or detachment was found, we discussed options with the patient and suggested a vitrectomy if predisposing factors were identified. If no tear or retinal detachment was found and the patient had a history of vascular disease in the eye, we advised a vitrectomy if VA remained low and showed no improvement for 1–3 weeks.

All patients were operated on at the Shandong Eye Hospital, a tertiary academic referral center. Data were retrieved from the original medical record, including basic patient information, clinical manifestation, structured operation notes and standardized fundus drawings. All operations were performed by the same surgeon (G.Y.) with the Alcon Accurus 800 (Alcon, USA). Briefly, a standard three-port vitrectomy was performed under retrobulbar or general anesthesia, depending on the individual preference of the surgeon. In the combined procedures, we started with

phacoemulsification of the lens and implantation of an intraocular lens. During vitrectomy, meticulous shaving of the vitreous base was done under a wide-angle viewing system with assisted scleral indentation. Fibrovascular tissue and tractional membrane were removed using a combination of segmentation and delamination, primarily with an intraocular scissor or a microvitrector tip. Hemostasis was maintained by raising intraocular pressure or by using endodiathermy. For the retinal vein occlusion eyes, laser photocoagulation was performed. All tears were treated with laser photocoagulation or external cryotherapy. At the end of the surgery, balanced salt solution, air, C2F6/C3F8 gas or silicone oil was adopted as an intraocular tamponade agent.

Postoperatively, patients were observed every day for the first week, weekly for 4 weeks and monthly thereafter. VA and complications were recorded during the follow-up. Statistical analysis was performed using SPSS software (version 16; SPSS, Inc., Chicago, IL, USA). VA before and after surgery was compared using the Wilcoxon signed-rank test. $P < 0.05$ was considered statistically significant.

Results

A total of 105 patients (105 eyes) were identified. The mean age of the patients was 57.14 years (range, 28 years to 78 years). There was no sex bias (58 female vs. 47 male) or eye bias (57 left eyes vs. 48 right eyes). The most common etiologies of dense vitreous hemorrhage with non-traumatic and non-diabetic retinopathy were retinal vein occlusion (RVO)(61 eyes), retinal tear (24 eyes) and retinal vasculitis (11 eyes). Other etiologies included polypoidal choroidal vasculopathy (6 eyes) and retinal macroaneurysm (3 eyes). The mean age of the patients with RVO, retinal tear and retinal vasculitis was 59.4, 54.9, and 35.8 years, respectively, while the mean age of the patients with polypoidal choroidal vasculopathy and retinal macroaneurysm was 67.5 years for both. Among the eyes with RVO, branch retinal vein occlusion (BRVO) ranked first, with superior temporal BRVO the predominant type (Table 1).

Table 1. Etiology of dense vitreous hemorrhage with non-traumatic and non-diabetic retinopathy.

Diagnosis	N (%)	Mean age	Min. age	Max. age
RVO	61 (58.1)	59.4	41	78
CRVO	4			
Hemi CRVO	6			
BRVO	51			
Superotemporal	34			
Inferotemporal	10			
Superonasal	5			
Inferonasal	2			
Retinal tear	24 (22.9)	54.9	28	72
Retinal vasculitis	11 (10.4)	35.8	28	52
PCV	6 (5.7)	67.5	61	78
RM	3 (2.9)	67.5	65	70

BRVO, branch retinal vein occlusion; CRVO, central retinal vein occlusion; N, number of eyes; PCV, polypoidal choroidal vasculopathy; RM, retinal macroaneurysm; RVO, retinal vein occlusion.

During surgery, 47 eyes with RVO were found to have neovascular membrane (NVM). Of these, 33 eyes with BRVO, 2 eyes with central retinal vein occlusion (CRVO) and 4 eyes with hemi CRVO were found to have retinal NVM. Moreover, 2 eyes with CRVO had disk NVM and 6 eyes with BRVO had both retinal and disk NVM. Additionally, retinal NVM was also detected in 8 eyes with retinal vasculitis. The results demonstrated that most of the dense vitreous hemorrhage eyes with RVO (77.0%) and retinal vasculitis (72.7%) presented with retinal and/or disk NVM (Table 2).

In the vitreous hemorrhage eyes, B-scan ultrasound may discover a posterior vitreous detachment, retinal tear or retinal detachment and provide important

Table 2. Neovascular membrane detected in the dense vitreous hemorrhage eyes.

Diagnosis	N	Retinal NVM	Disk NVM	Retinal and disk NVM
RVO	61	39	2	6
CRVO	4	2	2	0
Hemi CRVO	6	4	0	0
BRVO	51	33	0	6
Retinal vasculitis	11	8	0	0
Others	0	0	0	0

BRVO, branch retinal vein occlusion; CRVO, central retinal vein occlusion; N, number of eyes; NVM, neovascular membrane; RVO, retinal vein occlusion.

information for surgical decision-making. In the present study, preoperative B-scan ultrasound revealed dense vitreous opacity in all eyes, retinal tear in 5 eyes and retinal detachment in 26 eyes. Intraoperatively, retinal tear was found in 24 eyes and retinal detachment was confirmed in 48 eyes. B-scan ultrasound identified a retinal tear or detachment in eyes with dense vitreous hemorrhage in 20.8% and 54.2% of cases, respectively.

Apart from determining the etiology of dense vitreous hemorrhage, another area of concern for the clinician is VA. In the present study, VA was recorded preoperatively and postoperatively. There was improvement in VA in 100 of the 105 cases. The median preoperative VA was 1/70 (logMAR: 1.85) and the median postoperative VA was 0.6 (logMAR: 0.22). The improvement is large and statistically significant ($P < 0.001$) (Table 3).

During the mean follow-up period of 14.5 months (range, 6 months to 2 years), 20 of the 70 phakic eyes developed cataract. Elevated intraocular pressure (over 21 mmHg) was present in 10 eyes, among which nine were controlled by medication and one eye with CRVO developed neovascular glaucoma. Among all the cases, three eyes had to be reoperated for a macular pucker after 3 months and four eyes developed an inferior rhegmatogenous retinal detachment at 2–3 months after surgery, which was repaired by repeat vitrectomy. Recurrences of vitreous hemorrhage were noted in one eye with polypoidal choroidal vasculopathy and one eye with retinal arterial macroaneurysm at one month after surgery, with both eyes achieving stability after a second vitrectomy.

Discussion

Many clinicians favor a conservative strategy for the management of vitreous hemorrhage as vitrectomy is an invasive procedure with well-known risks, including cataract, glaucoma, retinal tear or retinal detachment. However, conservative treatment for a long-standing dense vitreous hemorrhage may cause marked visual disability due to the toxic effect of the hemorrhage on the retina and the formation of epiretinal membranes. As surgery or conservative treatment has its advantages and disadvantages, treatment of dense vitreous hemorrhage poses a dilemma to the ophthalmologist. The present study was designed to investigate the etiology of dense vitreous hemorrhage with non-traumatic and non-diabetic retinopathy and determine the prognosis of early vitrectomy for these patients.

Preoperatively, all the patients underwent a B-scan ultrasound to look for the presence of a posterior vitreous detachment, retinal tear or retinal detachment. B-scan ultrasound is a well-established method for evaluating vitreo-retinal anatomy in the presence of vitreous hemorrhage. Its use in detecting large retinal tears is well documented but in patients with small, multiple tears or dense vitreous hemorrhage, its use is limited.[9] In this series, B-scan ultrasound detected a preoperative retinal tear or retinal detachment in 20.8% and 54.2% of cases, respectively. Although dense vitreous hemorrhage may interfere with the accuracy of the examination, B-scan ultrasound can still provide important information to surgeons

Table 3. Comparison of visual acuity (logMAR) before and after surgery.

VA	Sig[a]	Median	IQR	Range	Z	P value[b]	95% CI
Initial	0.000	1.85	1.08	1.0–2.8			
final	0.000	0.22	0.94	0–2.3	−7.01	0.000	0.000–0.042

[a]Kolmogorov-Smirnov test
[b]Wilcoxon signed-rank test.
CI, confidence interval; IQR, interquartile range; VA, visual acuity

to facilitate optimal surgical decision-making and preoperative management.

Previous reports have shown that proliferative diabetic retinopathy, retinal vasculitis and BRVO are the major causes of vitreous hemorrhage in adults.[2,5] The etiologies encountered among our patients were similar to those reported previously but our study mainly focused on dense vitreous hemorrhage with non-traumatic and non-diabetic retinopathy. Our findings illustrated the most common etiologies for these patients were RVO, retinal tear and retinal vasculitis.

For dense vitreous hemorrhage associated with a retinal tear or rhegmatogenous retinal detachment, early surgery is recommended as there is a higher incidence of vitreoretinopathy and more likelihood of a poorer visual outcome.[10] For eyes with dense vitreous hemorrhage, in which a retinal tear cannot be ruled out, early surgery is also advocated. A previous study demonstrated that 67% of dense vitreous hemorrhage eyes appeared to have retinal tears and even 39% had retinal detachment in a conservative and prospective follow-up, therefore delayed management can be associated with an adverse visual outcome for these patients.[11,12]

Along with retinal tears, we documented other etiologies that were associated with dense vitreous hemorrhage in the present study, including RVO, retinal vasculitis, polypoidal choroidal vasculopathy and retinal macroaneurysm. All these etiologies can be attributed to an ocular vasculopathy. For dense vitreous hemorrhage caused by a retinal vasculopathy, the hypoxic retinal tissue may produce vascular endothelial growth factors (VEGF) and release inflammatory mediators, thereby inducing the complications of macular pucker, tractional retinal detachment and neovascular glaucoma.[13–16] There are several benefits to performing early vitrectomy for vitreous hemorrhage secondary to the retinal vasculopathy, including RVO and retinal vasculitis. First, vitrectomy removes the opaque vitreous hemorrhage and clears the visual axis, which can improve vision. Second, vitrectomy reduces the expression of VEGF, which is a vital factor in neovascularization. When vitrectomy is combined with anti-VEGF agents or laser photocoagulation, it may improve the prognosis from a dense vitreous hemorrhage.[17–21] Third, thorough evaluation and treatment of eyes with NVM may reduce complications that cause a decrease in vision, such as tractional retinal detachment and macula-off retinal detachment.[22]

Based on the results of this study, we recommend an early vitrectomy for dense vitreous hemorrhage with non-traumatic and non-diabetic retinopathy. Early vitrectomy can remove the opaque vitreous, clear the visual axis and restore vision. However, taking a more aggressive approach might impose risks on individuals who could have had a good outcome with conservative treatment. Although the outcome for VA

was good and the complications acceptable, a prospective, controlled study comparing early versus late vitrectomy is needed to draw firm conclusions and guide the clinician on this significant issue.

Declaration of Conflicting Interest

The authors declare that there is no conflict of interest.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by grants from the National Natural Science Foundation of China (81530027), the Youth Foundation of Shandong Academy of Medical Sciences (2016-37) and the Innovation Project of Shandong Academy of Medical Sciences.

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