Published in final edited form as:

Retina. 2018 September ; 38(Suppl 1): S41–S48. doi:10.1097/IAE.0000000000001942.

Outcomes of Pars Plana Vitrectomy for Macular Hole in Uveitis Patients

Natalia F. Callaway, MD, MS¹, Marco A Gonzalez, MD¹, Yoshihiro Yonekawa, MD^{2,3}, Lisa J. Faia, MD², Efreem Mandelcorn, MD⁴, Rahul N Khurana, MD⁵, Mohamed G. A. Saleh, MD^{6,7}, Phoebe Lin, MD, PhD⁶, Lucia Sobrin, MD, MPH³, and Thomas A Albin, MD¹

¹Bascom Palmer Eye Institute at University of Miami School of Medicine, Miami, FL

²Associated Retinal Consultants, P.C., Royal Oak, MI

³Massachusetts Eye and Ear Infirmary at Harvard Medical School, Boston, MA

⁴University of Toronto University Eye Clinic, Toronto, ON, Canada

⁵Northern California Retina Vitreous Associates, Daly, CA

⁶Casey Eye Institute at Oregon Health and Science University, Portland, OR

⁷Assiut University, Assiut, Egypt

Abstract

Purpose—Inflammatory macular hole (IMH) is a rare complication of uveitis and data on surgical outcomes of closure is scarce. The purpose of this study is to evaluate the anatomic and visual outcomes of conventional pars plana vitrectomy (PPV) for patients with uveitis.

Methods—Non-comparative, interventional, consecutive case series from six vitreoretinal surgical centers from 2007 to 2015. 20 eyes of 19 patients were included with four patients separated as viral retinitis. The primary outcome was change in best corrected visual acuity (BCVA) at month 3. Secondary outcomes were closure of the macular hole and postoperative OCT characteristics.

Results—All eyes underwent conventional 3-port PPV with indocyanine green-assisted internal limiting membrane peeling. Mean Snellen BCVA improved from 20/200 to 20/63 ($p=0.01$ for a difference in LogMAR) at month 3. Twelve (75%) of patients achieved two or more lines of visual acuity improvement by POM#3. Surgery resulted in decreased epiretinal membrane ($p=0.002$), intraretinal fluid ($p<0.001$), subretinal fluid ($p=0.029$), central subfield thickness ($p<0.001$) and central cube volume ($p=0.041$). Surgical intervention achieved anatomic success, as measured by macular hole closure, in 13 (81%) of patients at POM#3.

Corresponding Author: Thomas Albin, MD, Department of Ophthalmology, Bascom Palmer Eye Institute, 900 NW 12th Avenue, Tel: 305 326 6000, Talbini@med.miami.edu.

Meeting Presentations:

American Society of Retina Specialists, August 2016, San Francisco, CA

International Uveitis Study Group, August 2016, Dublin, Ireland

Sonoma Eye, February 2017, Sonoma, CA

Duke Advanced Vitreous Surgery Course, April 2017, Durham, NC

Proprietary Interest: None of the authors have any proprietary interest in the material presented to disclose.

Conclusion—Patients with IMH respond well to conventional surgery, with good anatomic and visual acuity outcomes.

Keywords

Full-thickness Macular Hole; Inflammatory Macular Hole; Pars Plana Vitrectomy; Uveitic Macular Hole; Uveitis

INTRODUCTION

Uveitis is a sight-threatening ocular inflammatory condition that accounts for 10–15% of blindness among young, working-age adults in the United States.^{1–3} Vision loss from macular abnormalities is most often secondary to cystoid macular edema (CME), however, rarely patients can develop macular holes.^{4–6} Inflammatory macular holes (IMH) are full thickness macular holes that form in the setting of inflammatory eye conditions.

Two mechanisms for the development of IMH have been proposed: 1) an inflammatory focus in the macula may cause localized tissue necrosis and result in macular hole formation, or 2) persistent hyaloidal traction, chronic cystoid macular edema, and epiretinal membrane traction may combine to exert sufficient force to cause hole formation.⁷ These mechanisms differ from idiopathic macular hole development and given the rare occurrence of this condition little is known about the optimal management strategy for IMH. To date, case reports and small series have reported inconsistent visual acuity outcomes with surgical intervention for uveitic macular holes.

The purpose of the current study is to report the anatomic and visual results of conventional pars plana vitrectomy (PPV) for the treatment of IMH. We hypothesize that patients undergoing conventional surgical treatment for IMH will have improved visual acuity and anatomic outcomes.

METHODS

The institutional review board at the University of Miami (IRB #20140693) approved the study protocol and the research adhered to the tenets of the Declaration of Helsinki and complied with the Health Insurance Portability and Accountability Act of 1996. None of the authors have a proprietary interest in any product discussed in the manuscript.

Study Design

The present study is a multicenter, retrospective, non-comparative, interventional, consecutive case series. The study population consisted of patients who had undergone PPV for full-thickness IMH attributed to uveitis at one of six vitreoretinal surgical centers from 2007 to 2015: Bascom Palmer Eye Institute, Associated Retinal Consultants, University of Toronto, Northern California Retina Vitreous Associates, Casey Eye Institute and Massachusetts Eye and Ear Infirmary. Patients were excluded if there was a confounding visually significant comorbidity, specifically advanced age-related macular degeneration, glaucoma, or proliferative diabetic retinopathy, which was felt to be the primary cause of

vision loss by the investigator. Patients were also excluded if post-operative follow-up data was not available at the primary study end-point, post-operative month three (POM3).

Baseline Characteristics

All eyes underwent a standard clinical examination, including best corrected Snellen visual acuity (BCVA), fundus examination, and spectral domain optical coherence tomography (OCT) imaging of the macula. Baseline characteristics were collected for each patient including patient age, sex, laterality of surgery, uveitic diagnosis, duration of diagnosis, co-morbid ocular and systemic conditions, uveitis treatment, pre-operative treatment, presence of posterior vitreous detachment (PVD) and lens status.

OCT Characteristics

The presence of IMH was determined with pre-operative spectral domain OCT using Cirrus OCT (Carl Zeiss Meditec, Dublin, CA, US) or Spectralis OCT (Heidelberg Engineering Inc, Franklin, MA). IMH was defined as OCT confirmed full-thickness retinal defect within 3 mm of the fovea causing symptomatic visual loss in the setting of past or current intraocular inflammatory disease. The presence of the inflammatory macular hole had to be judged to be the prominent cause of decreased visual acuity. Only OCT images with a signal strength greater than 5 were included. Pre-operative and post-operative OCT images were used to collect the following anatomic parameters: epiretinal membrane (yes/no), intraretinal fluid (yes/no), subretinal fluid (yes/no), central subfield thickness (μm), central cube volume (mm^3). OCT images before and after surgery were compared for each subject to make a determination of anatomic hole closure (yes/no).

Surgical Intervention

The operating surgeon selected the surgical approach for the individual patient as there was no predefined protocol in this retrospective study. All surgeons in this series have completed uveitis fellowship training in addition to vitreoretinal surgical training. All patients underwent conventional small gauge 3-port PPV. The following surgical parameters were collected: gauge (23, 25), membrane peel (yes/no), internal limiting membrane (ILM) peel (yes/no), intravitreal triamcinolone acetonide (yes/no), indocyanine green (ICG) dye (yes/no), fluid-air exchange (yes/no). If gas or oil were used during the procedure the type (C3F8, SF6, or silicone oil) and percent of gas air mixture were recorded. Surgeons' documentation of whether the uveitis was active at the time of surgery was recorded when available.

Outcome Measures

The primary outcome measure was change in visual acuity before and after surgery at post-operative month (POM#3) three. Secondary outcomes were anatomic change in OCT characteristics as well as hole closure, complications within three months, and the rate of reoperation.

Statistical Analysis

All variables were evaluated graphically for normality. Visual acuity was converted to LogMAR units for statistical analysis. Baseline characteristics were described with

frequency distributions appropriate to the variable distribution. Categorical variables were reported as frequencies and compared as two-sample proportions using Fisher's exact test while LogMAR visual acuities before and after surgery were compared with the Wilcoxon signed-rank test. Viral retinitis etiologies of macular hole were hypothesized *a priori* to demonstrate more aggressive clinical course with poorer visual outcomes, therefore these patients were excluded from statistical analysis. All statistical tests employed a two-tailed alpha with statistical significance set at <0.05 .

RESULTS

Baseline Characteristics

The study reviewed cases of uveitic macular hole undergoing surgical closure in 24 eyes of 23 patients. Three subjects were excluded because they did not have post-operative follow-up data. One subject was excluded because of advanced age-related macular degeneration (AMD) that was felt to be the primary reason for vision loss by the investigator. Among the remaining 20 eyes of 19 patients, four patients were separated as viral retinitis. These cases were hypothesized *a priori* to demonstrate a more aggressive clinical course with poorer visual outcomes and thus were excluded from statistical analysis, but included as a separate descriptive result section.

The final study analysis included 16 eyes of 15 patients followed for a mean of 18.1 months after surgery (range 3–106 months). One patient had both eyes included in the study. Subjects were 43.75% (7/16) male and the average age at the time of surgery was 56 years old (SD 14 years, range 28–75).

The etiology of uveitis and case information for cases included in the analysis is demonstrated in Table 1. The median duration of symptoms prior to surgery was 4 months (SD 23 months, range 1 to 96 months). Active uveitis was noted intraoperatively in 37.5% of eyes. The right eye was the operative eye in 40% of patients with one patient having bilateral macular hole repair at different times. Prior to surgery, one eye received topical prednisolone, two received subtenons triamcinolone, two received oral prednisone, and one received intravenous methylprednisone. At the time of surgery, 12 (75%) eyes were phakic and four eyes (25%) were pseudophakic. A PVD was present in 7 (43.8%) of eyes at the time of surgery. The average time between pre-operative OCT and initial surgical intervention was 33 days (SD 33, range 5–141 days). The most common uveitic diagnosis (excluding viral retinitis) among patients undergoing surgical macular hole repair were idiopathic panuveitis (25%) and chronic anterior uveitis (16.7%).

Surgical Intervention

All eyes underwent conventional 3-port PPV with ICG assisted ILM peeling. 12 (75%) of vitrectomies were performed with a 23-gauge instrumentation and 4 (25%) with a 25-gauge instrumentation. Nine eyes (56%) underwent intravitreal triamcinolone acetate staining to ensure complete vitreous removal from the posterior pole. The extent of ILM peel around the fovea was not quantifiable based on the available operative note. A fluid-air exchange

was performed in all eyes followed by gas tamponade with either SF₆ (n=11, 69%) or C3F8 (n=5, 31%).

Anatomic and OCT Outcomes

The mean time between surgery and first postoperative OCT was 85 days (SD 54 days, range: 17 to 230 days). Table 2 compares the baseline and post-operative month three OCT characteristics and visual outcomes. Surgical intervention resulted in resolution of all epiretinal membranes since ILM was peeled in all cases. Surgical repair of the macular hole was associated with a statistically significant decrease in the proportion of patients with intraretinal fluid ($p=0.0001$) and subretinal fluid (0.029). There was also a statistically significant decrease in all three central retinal thickness measures: 1) central subfield thickness ($p=0.0007$), 2) cube volume ($p=0.04$), and 3) central cube volume ($p=0.00068$). Surgical intervention achieved anatomic success, as measured by macular hole closure, in 13 (81%) of patients at POM#3.

Visual Acuity Outcome

The visual acuity outcomes are listed for each case in Table 1 and converted to LogMAR for statistical analysis in Table 2. Among the entire cohort, surgical intervention resulted in an improvement in mean Snellen BCVA from 20/200 (LogMAR 1.0 ± 0.46) to 20/63 (LogMAR 0.50 ± 0.44), slightly more than two Snellen visual acuity lines, that was statistically significant with a p-value of 0.0012. Twelve (75%) of patients achieved two or more lines of visual acuity improvement by POM#3, 3 (19%) maintained stable visual acuity and one patient lost one line of vision. Figure 1 displays the fundus photography, pre-operative and post-operative OCT of example subjects who A) did not demonstrate visual acuity improvement, or B) who had improved visual acuity at POM#3. In both cases successful anatomic closure of the hole is seen on the OCT.

Initially, four patients were pseudophakic, however, by the end of the study period three phakic patients underwent combined or sequential cataract extraction. Thus, 7 (44%) of patients were pseudophakic and 9 (56%) were phakic at POM#3. There was no statistically significant difference in the BCVA at POM#3 by lens status ($p=0.73$).

Viral Retinitis

Four patients had viral retinitis: two cases of acute retinal necrosis (ARN) and two with cytomegalovirus (CMV) retinitis. Viral retinitis was hypothesized *a priori* to follow a more aggressive clinical course and a second analysis without these subjects was planned. The baseline characteristics, surgical treatment and outcomes for patients with viral retinitis are shown in Table 3. There was an insufficient sample size in the viral retinitis group to run statistical comparisons. There was an equal distribution of men and women, all patients were treated with antiviral therapy. Three of the four viral retinitis patients had complications within three months and the final patient eventually when on to have late reopening of the hole. All viral retinitis patients required reoperation, two of whom had silicone oil placed during the second surgery. The pre-operative visual acuities in the viral retinitis group ranged from 20/150 to 20/400. Two of these patients experienced visual acuity decline by

POM#3, one remained stable and one improved from 20/150 to 20/60, but later in the year had late re-opening of the hole and had 20/200 vision by the last recorded visit.

Reoperation

Excluding cases of viral retinitis, three (19%) patients with inflammatory macular holes who underwent surgical repair required reoperation within three months. One patient had CME prior to surgery and reopened by post-operative month one. They underwent reoperation with successful closure of the hole by POM#3 and BCVA of 20/25 from 20/60 preoperatively. The second case also had reopening of the whole within one month of the first surgery and underwent secondary repair with 12% C3F8 after initial repair with SF6 20%. The patient ultimately achieve anatomic hole closure at the last clinic visit, however, there was no change in the visual acuity during the study period. Finally, the last patient, who had the longest duration of symptoms to surgical intervention time in the study, never achieve anatomic closure of the macular hole despite two surgical interventions during the study period.

Complications

As mentioned above, three study subjects and all four viral retinitis patients required reoperation. At POM#3 one patient experienced episcleritis that resolved with topical treatment. Review of the POM#3 OCTs demonstrated that two eyes (13%) had residual intraretinal fluid (IRF) and one had persistent exudates without fluid. Interestingly, both of the patients with post-operative CME had active uveitis noted intraoperatively. One subject with CME maintained stable visual acuity despite closure of the macular hole and was thought to have limited visual potential due to a large toxocariasis cyst in the macula.

DISCUSSION

IMH is a rare cause of decreased vision in patients with uveitis. Nussenblatt initially described macular hole as a very rare complication of posterior segment inflammation.⁵ A large cross sectional study of 582 patients with uveitis did not demonstrate a case of IMH.⁸ One possibility for this is that biomicroscopic evaluation of the macula is often limited in cases of dense media opacification or other sequellae of uveitis. The advent of spectral domain OCT has improved the diagnostic sensitivity of IMH detection. A recent review listed 55 reported cases of IMH in the literature, mostly secondary to Behcet's disease (33 cases).⁷ Since then a few additional cases have been added to the literature.^{9–14}

The current literature provides conflicting outcomes of PPV for macular pathologies in uveitis.⁶ Surgical intervention for IMH has not been extensively studied. Limited case studies exist demonstrating poor outcomes.^{15–16} Two case reports document anatomic and visual success following conventional surgery for macular hole in Behcet's disease¹⁷ and Vogt-Koyanagi-Harada disease.¹⁸ Another report documents anatomic but not visual success following PPV with inverted ILM flap technique.¹³

The current interventional case series demonstrates visual improvement in a majority of patients undergoing conventional PPV for OCT-documented IMH. Conventional PPV for macular hole resulted in statistically significant improvement in mean visual acuity. All

cases achieved anatomic closure with statistically significant reduction in epiretinal membrane, intraretinal and subretinal fluid, and central subfield thickness and central cube volume as measured on OCT.

The majority of cases required a single surgery; however, 3 cases required further surgical intervention with limited visual acuity gains. While the authors agree that as a principle intraocular inflammation should be controlled prior to surgery, due to concerns for extended delay in surgery and/or incomplete response to therapy, 38% of eyes had some degree of active uveitis during surgery as judged by the surgeon. Postoperative cystoid macular edema developed in 13% of patients in the current study and is a potentially treatable anatomic cause of limited visual acuity benefit. It is possible that more aggressive perioperative inflammatory control would have improved visual acuity outcomes.

Two confounding factors in this retrospective study are the effect of progressive cataract formation on visual outcomes and severity of underlying uveitic disease. Both of these patient characteristics are not quantifiable and impossible to adjust for. As in any surgical series, patient selection is crucial and patients with completely uncontrolled uveitis or significant visual co-morbidities were likely not offered surgery or excluded from the study. There was no specified protocol for treating inflammation perioperatively. Although surgical procedure was not pre-determined in this study, conventional surgery with small gauge instrumentation, ILM peel and gas tamponade were surprisingly similar across five centers. Prospective studies, large cases series on single uveitis entities or single surgical strategies may provide further information on success rates under particular conditions. Comparative studies of different surgical techniques may in the future establish the added benefit on nonconventional approaches to macular hole surgery, such as those involving various manipulations of the ILM other than peeling alone.

In conclusion, conventional surgical management of symptomatic IMH in patients with aggressive perioperative anti-inflammatory management achieved favorable visual and anatomic outcomes in the current series. A minority of patients required additional surgical intervention. Long-term visual acuity was limited in some patients by cataract formation, cystoid macular edema and the sequelae of viral retinitis.

Acknowledgments

Financial Support: Supported by grants from the National Institutes of Health (Center Core grant, P30EY014801), Research to Prevent Blindness, the Department of Defense (W81XWH-09-1-0675), and the Klorfine Foundation (to Dr. Albini).

References

1. Suttorp-Schulten MS, Rothova A. The possible impact of uveitis in blindness: a literature survey. *Br J Ophthalmol*. 1996 Sep; 80(9):844–8. [PubMed: 8962842]
2. Durrani OM, Meads CA, Murray PI. Uveitis: a potentially blinding disease. *Ophthalmologica*. 2004 Jul-Aug; 218(4):223–36. [PubMed: 15258410]
3. Tomkins-Netzer O, Talat L, Bar A, Lula A, Taylor SR, Joshi L, Lightman S. Long-term clinical outcome and causes of vision loss in patients with uveitis. *Ophthalmology*. 2014 Dec; 121(12): 2387–92. [PubMed: 25178807]

4. Durrani OM, Tehrani NN, Marr JE, Moradi P, Stavrou P, Murray PI. Degree, duration, and causes of visual loss in uveitis. *Br J Ophthalmol*. 2004 Sep; 88(9):1159–62. [PubMed: 15317708]
5. Nussenblatt RB. Macular alterations secondary to intraocular inflammatory disease. *Ophthalmology*. 1986 Jul; 93(7):984–8. [PubMed: 2429245]
6. Branson SV, McClafferty BR, Kurup SK. Vitrectomy for Epiretinal Membranes and Macular Holes in Uveitis Patients. *J Ocul Pharmacol Ther*. 2017 Jan.;23. [Epub ahead of print].
7. Bonnin N, Cornut PL, Chaise F, Labeille E, Manificat HJ, Feldman A, Perard L, Bacin F, Chiambaretta F, Burillon C. Spontaneous closure of macular holes secondary to posterior uveitis: case series and a literature review. *J Ophthalmic Inflamm Infect*. 2013 Feb 11.3(1):34. [PubMed: 23514634]
8. Rothova A, Suttrop-van Schulten MS, Frits Treffers W, Kijlstra A. Causes and frequency of blindness in patients with intraocular inflammatory disease. *Br J Ophthalmol*. 1996 Apr; 80(4):332–6. [PubMed: 8703885]
9. Panos GD, Gatzoufas Z. Macular hole formation after toxoplasmic retinochoroiditis: coincidence or rare complication? *Int Ophthalmol*. 2013 Jun.33(3):219. [PubMed: 23543277]
10. Uçar D, Atalay E, Özyazgan Y, Özkök A, Yıldırım Y. An exceptional case of full-thickness macular hole closure in a patient with Behçet disease. *Ocul Immunol Inflamm*. 2014 Feb; 22(1): 79–81. [PubMed: 24063599]
11. Tanaka R, Obata R, Sawamura H, Ohtomo K, Kaburaki T. Temporal changes in a giant macular hole formed secondary to toxoplasmic retinochoroiditis. *Can J Ophthalmol*. 2014 Oct; 49(5):e115–8. [PubMed: 25284113]
12. Seth A, Raina UK, Thirumalai S, Batta S, Ghosh B. Full-thickness macular hole in Bartonella henselae neuroretinitis in an 11-year-old girl. *Oman J Ophthalmol*. 2015 Jan-Apr;8(1):44–6. [PubMed: 25709274]
13. Hirano M, Morizane Y, Kawata T, Kimura S, Hosokawa M, Shiode Y, Doi S, Hosogi M, Fujiwara A, Shiraga F. Case report: successful closure of a large macular hole secondary to uveitis using the inverted internal limiting membrane flap technique. *BMC Ophthalmol*. 2015 Jul 25.15:83. [PubMed: 26208719]
14. Mizuno M, Fujinami K, Watanabe K, Akiyama K. Macular Hole Associated with Vogt-Koyanagi-Harada Disease at the Acute Uveitic Stage. *Case Rep Ophthalmol*. 2015 Sep 15; 6(3):328–32. [PubMed: 26483677]
15. Woo SJ, Yu HG, Chung H. Surgical outcome of vitrectomy for macular hole secondary to uveitis. *Acta Ophthalmol*. 2010 Nov; 88(7):e287–8. [PubMed: 20977693]
16. Al-Dhibi H, Abouammoh M, Al-Harhi E, Al-Gaeed A, Larsson J, Abboud E, Chaudhry I. Macular hole in Behçet's disease. *Indian J Ophthalmol*. 2011 Sep-Oct;59(5):359–62. [PubMed: 21836340]
17. Wu TT, Hong MC. Pars plana vitrectomy with internal limiting membrane removal for a macular hole associated with Behçet's disease. *Eye (Lond)*. 2009 Jul; 23(7):1606–7. [PubMed: 18670457]
18. Mizuno M, Fujinami K, Watanabe K, Akiyama K. Macular Hole Associated with Vogt-Koyanagi-Harada Disease at the Acute Uveitic Stage. *Case Rep Ophthalmol*. 2015 Sep 15; 6(3):328–32. [PubMed: 26483677]

Summary Statement

Inflammatory macular hole (MH) is a rare complication of uveitis that can result in central vision loss. Little research exists on surgical outcomes of inflammatory MH. This multi-center study represents the largest case series of surgical outcomes for uveitic MH and reports improved anatomic and visual outcomes after surgical intervention.

Figure 1.

Panel A) Fundus photograph of the right eye of a 48 yo Male with Toxoplasmosis complicated by a full-thickness inflammatory macular hole. Pre-operative OCT (top right) through the fovea demonstrates a full thickness macular hole that was the primary reason for visual decline in this patient. Post-operative month 3 OCT below shows anatomic closure of the hole. Despite full closure the visual acuity remained stable at 20/200 pre-operative and post-operatively. Panel B) Montage fundus photograph of the right eye of 60 yo M with presumed ocular histoplasmosis syndrome (POHS) with a full-thickness inflammatory macular hole. To the right, the top panel demonstrates pre-operative OCT with a visual acuity of 20/100 and the bottom right shows his post-operative month 3 OCT that demonstrates anatomic closure of the macular hole. The patient had visual acuity of 20/25 at post-operative month three with trace subretinal fluid and 20/20 at his last clinic visit 13 months after presentation.

Table 1

Characteristics and Outcomes of Patients Undergoing Surgical Treatment of Inflammatory Macular Hole

Sex/Age, years	Diagnosis	Uveitis Treatment	Pre-op treatment	Active at surgery	Surgery	BCVA		Anatomic Outcome at POM#3	Long-term Complications	Reoperation
						Preop	POM3			
M/60	POHS	None	None	No	PPV, MP, ILM peel, IVK, SF6 20%	20/100	20/25	Closed	None	–
M/55	TB panuveitis	Isoniazid × 6 months, topical PF	Intravenous Solumedrol	Yes	PPV, MP, ILM peel, IVK, SF6 20%	20/100	20/30	Closed	None	–
M/64	Idiopathic panuveitis	Intravitreal voriconazole	Topical PF, Voriconazole	Yes	PPV, MP, ILM peel, C3F8 14%	20/200	20/60	Closed	None	–
F/75 – Left *	Chronic AAU	MTX 12.5mg/Nevanac	SubTenon Kenalog	No	PPV, MP, ILM peel, IVK, SF6 20%	20/60	20/25	Closed	CME and Persistent Hole	ILM peel with 14% C3F8
F/68 – Right *	Chronic AAU	MTX 12.5mg/Nevanac	SubTenon Kenalog	No	PPV, MP, ILM peel, IVK, C3F8 16%	20/400	20/80	Closed	None	–
M/48	Toxoplasmosis	Bactrim, Clindamycin, Prednisone	None	Yes	PPV, MP, ILM peel, IVK, SF6 20%	20/200	20/200	Closed	Persistent hole	ILM peel with 12% C3F8
M/59	Rheumatoid Arthritis	Durezol	None	No	PPV, MP, ILM peel, IVK, SF6 20%	20/200	20/30	Closed	Episcleritis	–
F/66	Idiopathic panuveitis	Durezol	None	No	PPV, MP, ILM peel, IVK, SF6 20%	20/60	20/20	Closed	Iritis	–
M/50	Idiopathic Vasculitis	Prednisone	None	No	PPV, MP, ILM peel, IVK, SF6 20%	20/100	20/60	Closed	None	–
M/28	Toxocariasis	Bactrim, Clindamycin, Prednisone, Albendazole	None	Yes	PPV, MP, ILM peel, SF6 20%	2/200	20/200	Closed	None	
M/58	Chronic AAU	Topical PF	Prednisone	Yes	PPV, ILM peel, IVK, SF6 24%	20/100	20/150	Closed	None	–
F/56	Sarcoidosis	None	None	No	PPV, MP, ILM peel, C3F8 12%	20/100	10/200	Closed	None	–
F/64	Lupus	MTX, Plaquenil	None	No	PPV, ILM peel, SF6 22%	20/400	20/50	Closed	None	–
F/29	Behcet's	Prednisone	Prednisone	No	PPV, MP, ILM peel, C3F8 14%	20/100	20/30	Closed	None	–
F/72	Panuveitis (Sarcoid?)	N/A	None	No	PPV, ILM peel, SF6 20%	CF@ 3ft	CF@ 5ft	Open	Persistent hole	–
F/42	Idiopathic Panuveitis	Mycophenolate Mofetil, Periocular steroid	None	Yes	PPV, ILM peel, SF6 20%	8/200	20/30	Closed	None	–

* Same patient. POHS = Presumed ocular histoplasmosis syndrome; TB = tuberculosis; AAU = acute anterior uveitis; PF = Pred Forte; MTX = Methotrexate; HAAART = Highly active anti-retroviral therapy; PPV = pars plana vitrectomy; MP = membrane peel; ILM = internal limiting membrane; IVK = Intravitreal Kenalog; BCVA = Best Corrected Visual Acuity in Snellen Notation; SO = silicone oil; PVR = proliferative vitreoretinopathy; HM = hand motion;

Table 2

Optical coherence tomography (OCT) and visual function after surgical intervention for inflammatory macular hole

Variable	Pre-operative OCT N (%)	Post-operative OCT N (%)	P-value
OCT Characteristics			
Epiretinal membrane	08 (50)	0 (0)	0.002
Intraretinal fluid	15 (94)	2 (13)	<0.001
Subretinal fluid	10 (63)	3 (19)	0.029
Central Subfield Thickness - μm	470 $\pm$ 166	260 $\pm$ 52	<0.001
Cube Volume (mm^3)	9.9 $\pm$ 2.4	8.8 $\pm$ 1.4	0.041
Central Cube Volume (mm^3)	0.36 $\pm$ 0.83	0.21 $\pm$ 0.06	<0.001
Visual Acuity			
Snellen	20/200	20/63	—
LogMAR $\pm$ SD	1.00 $\pm$ 0.46	0.50 $\pm$ 0.44	0.001

For continuous variables (central thickness measures) the mean $\pm$ standard deviation are presented. Two-tailed Fishers exact test (proportions) was used for proportions, paired t-tests were used for central thickness measurements, which were normally distributed. Visual acuity was converted to LogMAR for analysis and found to exhibit a non-parametric distribution therefore statistical comparisons were made with a Wilcoxon signed-rank test for non-parametric paired data.

Characteristics and Outcomes of Patients Undergoing Surgical Treatment of Inflammatory Macular Hole with Viral Retinitis

Table 3

Sex/Age, years	Diagnosis	Uveitis Treatment	Pre-op treatment	Active at surgery	Surgery	BCVA		Anatomic Outcome at POM#3	Long-term Complications	Reoperation
						Preop	POM3			
M/53	CMV Retinitis	HAART, antiviral	None	No	PPV, MP, ILM peel, C3F8 14%	20/200	20/200	Closed	Recurrent RD	PPV with SO
F/43	Acute Retinal Necrosis	Valacyclovir	None	No	PPV, MP, ILM peel, C3F8 15%	20/400	HM	Closed	Reproliferation of PVR	RD repair with SO
F/67	CMV Retinitis	Oral antiviral	None	Yes	PPV, MP, ILM peel, C3F8 14%	20/150	20/60	Hole open	Persistent hole	PPV/ILM
M/68	Acute retinal necrosis	Valacyclovir	None	No	PPV, MP, ILM peel, SF6 24%	20/150	20/60	Closed	Late re-opening of hole	PPV/ILM peel

HAART = Highly active anti-retroviral therapy; PPV = pars plana vitrectomy; MP = membrane peel; ILM = internal limiting membrane; IVK = Intravitreal Kenalog; BCVA = Best Corrected Visual Acuity in Snellen Notation; SO = silicone oil; PVR = proliferative vitreoretinopathy; HM = hand motion; RD=retinal detachment; PVR=proliferative vitreoretinopathy.