EXTENDED REPORT

Cystoid puncture for chronic cystoid macular oedema

Rishi P Singh, Ronald Margolis, Peter K Kaiser

Br J Ophthalmol 2007;**91**:1062–1064. doi: 10.1136/bjo.2006.101790

See end of article for
authors' affiliations

Correspondence to:
Mr P K Kaiser, Digital
Optical Coherence
Tomography Reading Center
(DOCTR), 9500 Euclid
Avenue, Desk i2, Cleveland,
OH 44195, USA; pkkaiser@
aol.com

Accepted 1 January 2007
Published Online First
17 January 2007

Objective: To evaluate the new surgical technique of cystoid macular oedema puncture (CMOP) in patients with longstanding cystoid macular oedema refractory to standard treatments.

Design: Interventional, retrospective case series

Methods: Retrospective review of patients with chronic cystoid macular oedema from vascular retinopathy for whom maximal medical or surgical treatment failed and who underwent pars plana vitrectomy and CMOP. Clinical findings, best-corrected Snellen visual acuity, stereo colour fundus photography, intravenous fluorescein angiograms, and optical coherence tomography were obtained before and after treatment to evaluate the efficacy and safety of the treatment.

Results: Seven patients were included in the study. Cystoid macular oedema was due to diabetic retinopathy in five patients, central retinal vein occlusion in one patient and branch retinal vein occlusion in one patient. Preoperative intravitreal steroids failed for all patients, and three patients also had focal grid photocoagulation. Previous pars plana vitrectomy, with elevation of the posterior hyaloid, internal limiting membrane peeling, and intravitreal steroid injection, had failed in three patients. The median time to CMOP was 488 days. Resolution or improvement of cystoid oedema occurred in all patients as determined by fluorescein angiography or optical coherence tomography, or both. However, visual acuity was unchanged in five patients, declined in one patient and stable in one patient.

Conclusions: Although cystoid macular oedema does improve quantitatively after CMOP, the technique fails to improve visual acuity in patients.

Cystoid macular oedema represents an important cause of visual disability among patients with diabetic retinopathy and venous occlusion. Although the exact pathological mechanism is still uncertain, hypotheses have been developed to explain its formation. In diabetic patients, the pathogenesis appears to be due to disruption of the blood–retinal barrier at the level of the retinal capillaries, with exudation and pooling of fluid into the retina.¹ Diabetic patients may also have abnormalities in the structure of the vitreo–retinal interface, with posterior hyaloidal traction contributing to the formation of macular oedema.^{2–5} Conversely, obstructions of a retinal vein result in retinal capillary engorgement and a rise in the hydrostatic pressure proximal to the obstruction site. This leads to fluid exudation and stress on the retinal capillaries, causing further disruption of the blood–retinal barrier.⁶ The release of vascular endothelial growth factor and subsequent increase in vascular permeability in response to the ischaemia exhibited in both conditions further adds to the leakage.⁷

When medical management, such as focal laser photocoagulation, and periocular and intraocular steroid injections have failed, surgical interventions are attempted. Removal of the vitreous and elevation of the posterior hyaloid with pars plana vitrectomy has been shown to be successful in the management of persistent diabetic macular oedema. Surgical success (defined as macular oedema resolution) has been reported to be 70–100% in several case series.^{8–11} Positive predictive factors for success of vitrectomy include the presence of diffuse oedema and a taut posterior hyaloid on optical coherence tomography (OCT) examination.¹² However, while these manoeuvres can have a significant impact on diffuse retinal oedema, cystoid macular oedema improves less frequently and can be a chronic cause of visual loss.¹³

The method of cyst puncture and cyst removal was initially proposed by Dr Yuichiro Ogura and evaluated in a pilot study of six patients.¹⁰ Since this initial report, we are unaware of any other reports investigating the technique of cystoid macular

oedema puncture (CMOP) or any other surgical techniques in the management of chronic cystoid macular oedema. The purpose of this report was to investigate the efficacy and safety of CMOP in patients with longstanding cystoid macular oedema due to vascular retinopathies.

METHODS

A retrospective chart review was conducted on all patients who underwent CMOP at the Cole Eye Institute, Cleveland, OH. Inclusion criteria included patients with persistent cystoid macular oedema secondary to either diabetic retinopathy or retinal vein occlusion, and for whom previous medical and/or surgical management had failed, including retrocecal or intravitreal steroids, focal laser or surgery, if applicable, without any resolution of the cystoid macular oedema. Patients were excluded from the analysis if their cystoid macular oedema was due to other causes, if a thickened posterior hyaloid was observed on clinical or OCT examination, or if it was less than 4 months since their last medical or surgical intervention. Data from the clinical and operative notes were collected, including best-corrected Snellen visual acuity, stereo colour fundus photographs, intravenous fluorescein angiograms, and OCT examinations.

The CMOP procedures were all performed by one surgeon (PKK) between November 2004 and September 2005. The surgical procedure consisted of a standard 20-gauge pars plana vitrectomy followed by production of four small incisions in a cross pattern parallel to the nerve fibre layer around the fovea with a 20-gauge microvitrectoretinal blade (fig 1). A 39-gauge cannula was then placed through these small incisions into the cysts, and the cysts were carefully drained. In most cases, the cysts visibly collapse when this manoeuvre is performed. Finally, a gas–fluid exchange using filtered air or 10–14%

Abbreviations: CMOP, cystoid macular oedema puncture; OCT, optical coherence tomography

Figure 1 Schematic illustration of the cystoid macular oedema puncture technique.

C₃F₈ gas was performed, and the patient was instructed to remain in the prone position for a period of 24 hours.

RESULTS

Seven patients were included in the study. Table 1 outlines the patient demographics. Five men and two women with a median age of 67.2 years (range 55–81) were included in the study. The persistent cystoid macular oedema was due to diabetic retinopathy in five patients, central retinal vein occlusion in one patient, and branch retinal vein occlusion in one patient. All patients had received at least one previous preoperative 0.4 mg triamcinolone acetonide (Kenalog-40; Bristol Meyers Squibb, Peapack, New Jersey, USA) intravitreal injection and two patients had focal grid photocoagulation. In addition, three patients had previous pars plana vitrectomy with elevation of the posterior hyaloid and peeling of the internal limiting membrane with injection of 0.4 mg triamcinolone acetonide at the end of the case. No significant anatomical or visual benefits were seen with these interventions. All patients had persistent cystoid macular oedema despite these medical and surgical interventions, with a median (SD) time to CMOP of 488 (239) days (range 47–764). The median follow-up after CMOP surgery was 234 days.

Biomicroscopic examination, fluorescein angiography and OCT showed improvement in the cystoid macular oedema in all eyes (fig 2). Complete resolution of the intraretinal cystic spaces was seen, verified by OCT in three eyes. The median baseline central subfield retinal thickness was 534 µm. The median baseline central subfield retinal thickness decreased by 267.9 µm (range 248–631). Despite this anatomical improvement, the visual acuity did not improve. Median baseline best-corrected Snellen acuity was 20/400 at last follow-up (p = 0.52). Six patients had stable vision within one line of baseline, and one patient had a decrease in vision of more than one line. Preoperative and postoperative fluorescein angiography data were available for three patients. A qualitative decrease in the

amount of leakage within late frames was seen in two patients. A third patient demonstrated capillary dropout with enlargement of the foveal avascular zone after the procedure.

Only two complications were identified after the procedure. Two patients developed a vitreous haemorrhage months after the surgery. The first patient, with a central retinal vein occlusion, subsequently developed neovascular glaucoma necessitating a glaucoma shunt procedure. The second patient developed diffuse vitreous haemorrhage 1 year after CMOP and it reabsorbed without intervention over the ensuing 2 months. No neovascularisation or other causes of vitreous haemorrhage were identified after clearance of the haemorrhage.

DISCUSSION

Chronic cystoid macular oedema in patients with diabetic retinopathy and vascular occlusion remains a vexing problem. Use of pericocular and intravitreal steroids has had some success. In this report, we evaluated the safety and efficacy of CMOP and drainage. Cystoid macular oedema improved in all patients in this study, noted on biomicroscopic, OCT and fluorescein angiographic examination. Impressive changes were also noted on the serial OCT examinations performed after the procedure and only a few complications were seen, probably unrelated to the procedure itself. However, visual acuity failed to improve in any patient during the study despite a relatively long follow-up period.

The initial report by Tachi and colleagues using the technique of CMOP was published in 1999 and included a total of six patients. Visual acuity remained stable in four patients, improved in one patient and declined in one patient. The study used biomicroscopic examination only to determine whether or not improvement had occurred. Because fluorescein angiography and OCT were not used to confirm the resolution, it is difficult to determine if these patients had true improvement after the procedure. All patients in our study had anatomical improvement confirmed by OCT or fluorescein angiography

Table 1 Patient demographics

Patient No	Age (years)	Risk for CMO	Previous laser	Previous steroid	Previous vitrectomy	Time to puncture (days)	Resolution	Preoperative acuity	Postoperative best acuity	Follow-up (days)
1	69.6	Diabetes	Focal×3	IVK×2	Yes	500	Complete	20/200	20/200	172
2	80.8	CRVO	None	IVK	No	47	Partial	HM	CF	210
3	65.2	Diabetes	None	IVK×1	Yes	488	Partial	20/400	20/400	51
4	81.1	BRVO	None	IVK×1	No	191	Complete	4/200	CF	290
5	55.3	Diabetes	Focal × 2	IVK×2	Yes	764	Complete	20/200	20/200	312
6	64.3	Diabetes	Focal	PST + IVK	No	494	Partial	20/400	20/400	369
7	67.2	Diabetes	None	PST + IVK	No	265	Partial	20/200	20/200	756

CMO, cystoid macular oedema; CRVO, central retinal vein occlusion; BRVO, branch retinal vein occlusion; IVK, intravitreal triamcinolone 4 mg; PST, posterior subtenon triamcinolone 40 mg; Focal, focal grid photocoagulation.

Figure 2 (A) Preoperative and (B) 2 months postoperative linear optical coherence tomography scans of patient after cystoid macular oedema puncture was performed. The baseline vision was 20/400 and 20/400 after the procedure.

1–2 months after the CMOP was performed. Significant quantitative changes were noted in the centre retinal thickness after the procedure by OCT and there was a qualitative decrease in leakage on fluorescein angiography.

Although the technique of CMOP leads to significant anatomical improvement, we did not observe a corresponding improvement in visual acuity. Certainly, the small sample size of this study and the pilot study report could confound the true outcome after this procedure. However, there are a variety of reasons why visual improvement may not be achieved in our patients despite the anatomical success of the procedure. First, histopathological studies on specimens with cystoid macular oedema showed Müller cell intracytoplasmic swelling leading to liquefaction necrosis, which was interpreted as representing the cystic changes noted clinically. Thus, the retinal damage caused by longstanding cystoid macular oedema may be irreversible, and visual improvement may be difficult. Second, in cases where there are both diffuse and cystic components to the macula oedema, angiographic confirmation of macular ischaemia may not be obvious until after the oedema has resolved. This ischaemia is not alleviated by the procedure and would result in persistent visual loss. Finally, even when the normal retinal ultrastructure is restored after the procedure, it is possible that key cell bodies and axons between retinal layers had been irreversibly damaged owing to structural stress.

Interestingly, despite peeling the posterior hyaloid and internal limiting membrane in three patients before the CMOP procedure, no improvement in the CME or vision was noted before the CMOP. Many recent studies have shown the critical role that the posterior hyaloid has in the development and persistence of macular oedema. Traction from the posterior hyaloid has been implicated in disruption of the blood–retinal barrier and vascular leakage.¹⁴ The tangential forces that it exerts have been implicated in subclinical macular traction syndrome.¹² Thus, the vitrectomy alone may have accounted for the visual improvement seen in patients in previous studies. The persistent cystoid macular oedema in the three patients who had previous vitrectomy and steroid injections, only improved after CMOP. Unfortunately, their vision did not change. Alternatively, it might not have been possible to improve the vision of the patients in our study owing to the longstanding cystoid macular oedema, and their vision might have improved from the procedure if early surgical intervention had been considered. Evidence for this theory comes from the visual improvement noted in many patients in previous

vitrectomy studies in which patients were treated earlier in the time course of their disease and had who still had the possibility of visual improvement.

In summary, CMOP and drainage appears effective in resolving the structural cystoid retinal changes, but fails to improve acuity despite long-term follow up. Currently, there are no data to support CMOP in cases of chronic cystoid macular oedema.

Authors' affiliations

Rishi P Singh, Ronald Margolis, Peter K Kaiser, The Cole Eye Institute, Cleveland Clinic Foundation, Ohio, USA

Competing interests: None.

REFERENCES

- 1 Cunha-Vaz J, Faria de Abreu JR, Campos AJ. Early breakdown of the blood–retinal barrier in diabetes. *Br J Ophthalmol* 1975;**59**:649–56.
- 2 Lewis H, Abrams GW, Blumenkranz MS, et al. Vitrectomy for diabetic macular traction and edema associated with posterior hyaloidal traction. *Ophthalmology* 1992;**99**:753–9.
- 3 Jalkh A, Takahashi M, Topilow HW, et al. Prognostic value of vitreous findings in diabetic retinopathy. *Arch Ophthalmol* 1982;**100**:432–4.
- 4 Tolentino FI, Lee PF, Schepens CL. Biomicroscopic study of vitreous cavity in diabetic retinopathy. *Arch Ophthalmol* 1966;**75**:238–46.
- 5 Takahashi M, Trempe CL, Maguire K, et al. Vitreoretinal relationship in diabetic retinopathy. A biomicroscopic evaluation. *Arch Ophthalmol* 1981;**99**:241–5.
- 6 Dick JSB, Jampol LM, Haller JA. Macular edema. In: Ryan SJ, Hinto DR, Schachar AP, Wilkinson P, eds. *Retina* St Louis: Mosby, 2001:973–85.
- 7 Vitoras SA, Youssri AI, Luna JD, et al. Upregulation of vascular endothelial growth factor in ischemic and non-ischemic human and experimental retinal disease. *Histol Histopathol* 1997;**12**:99–109.
- 8 Harbour JW, Smiddy WE, Flynn HW Jr, et al. Vitrectomy for diabetic macular edema associated with a taut posterior hyaloid membrane. *Am J Ophthalmol* 1996;**121**:405–13.
- 9 Pendergast SD, Hassan TS, Williams GA, et al. Vitrectomy for diffuse diabetic macular edema associated with a taut premacular posterior hyaloid. *Am J Ophthalmol* 2000;**130**:178–86.
- 10 Tachi N, Hashimoto Y, Ogino N. Cystotomy for diabetic cystoid macular edema. *Doc Ophthalmol* 1999;**97**:459–63.
- 11 Yamamoto T, Hitani K, Tsukahara I, et al. Early postoperative retinal thickness changes and complications after vitrectomy for diabetic macular edema. *Am J Ophthalmol* 2003;**135**:14–9.
- 12 Kaiser PK, Riemann CD, Sears JE, et al. Macular traction detachment and diabetic macular edema associated with posterior hyaloidal traction. *Am J Ophthalmol* 2001;**131**:44–9.
- 13 Avci R, Kaderli B, Avci B, et al. Pars plana vitrectomy and removal of the internal limiting membrane in the treatment of chronic macular oedema. *Graefes Arch Clin Exp Ophthalmol* 2004;**242**:845–52.
- 14 Hikichi T, Fujio N, Akiba J, et al. Association between the short-term natural history of diabetic macular edema and the vitreomacular relationship in type II diabetes mellitus. *Ophthalmology* 1997;**104**:473–8.