lINTRAOPERATIVE INTRAVITREAL TRIAMCINOLONE ACETONIDE DURING CATARACT SURGERY FOR THE PROPHYLAXIS AND MANAGEMENT OF POSTOPERATIVE DIABETIC MACULAR EDEMA

Dr Wajida Saad Bunian, M.B.CH.B D.O M.Sc Ophthalmology*

ABSRACT

Cataract extraction in diabetic patients is commonly indicated, both for visual rehabilitation and for improved visualization of the fundus. Unfortunately the visual prognosis for diabetic patients undergoing cataract surgery is guarded, mainly because of the risk for worsening retinopathy levels and exacerbation of macular edema.

The Aim of this study is to evaluate the efficacy of intravitreal Triamcinolone acetonide injected during cataract surgery in the prophylaxis and management of postoperative macular edema following uneventful cataract surgery in diabetics.

The study included 2 groups, Group A included 15 patients divided into 2 subgroups 1)-Diabetic patients without any excising macular oedema., 2)-Diabetic patients with pre-existing macular edema. Patients in Group A were subjected to phacoemulsification with Posterior chamber intraocular lens implantation.

Subjects and methods: Group B included 15 diabetic patients with or without preoperatively existing macular edema including patients with previous macular laser treatment with visually significant cataract. Patients were subjected to Phacoemulsification with PCIOL implantation and Intravitreal triamcinolone injection (dose of 8 mg in 0.2 ml will be injected slowly through the inferior pars plana).

Results: The results of this study showed that phacoemulsification with intravitreal TA in patients with CSME appears to be a safe intervention to avoid the postoperative exacerbation of the edema in patients with dense cataract precluding macular laser treatment. TA may serve as mean to control postoperative inflammation and prevent exacerbation of the macular edema. Postoperative laser treatment may be needed in some cases to augment the effect of intravitreal TA.

KEY WORDS

Phacoemulsification, diabetic macular edema, Intravitreal Triamcinolone injection

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*** Ophthalmological Dep. , Thi-Qar University , Medical College.**

INTRODUCTION

Macular edema is the most frequent cause of visual loss among patients with diabetic retinopathy. It may be present at any level of retinopathy ***(Klein A ,et al, 1984),*** The edema is caused primarily by breakdown of the inner blood retinal barrier and increase vascular permeability. Recent Studies suggest that inflammatory mediators such as Prostaglandin's (PG's) and the Vascular Endothelium Growth Factors (VEGF's), are at least partially responsible for the increased vascular permeability. Diabetic maculopathy is classified as focal maculopathy, diffuse macular edema (which can be cystoid), ischemic maculopathy, or mixed. Cystoid macular edema is a result of accumulation of fluid in the outer plexiform and the inner nuclear layers of the retina, centered about the fovea with secondary formation of cystic spaces that can be identified either ophthalmoscopically, angiographically, or by the use of Optical coherence tomography. ***(Bresnick GH, 1983).*** Cataract extraction in diabetic patients is commonly indicated, both for visual rehabilitation and for improved visualization of the fundus. Unfortunately the visual prognosis for diabetic patients undergoing cataract surgery is guarded, mainly because of the risk for worsening retinopathy levels and exacerbation of macular edema ***(Pollack et al, 1991).*** An important determinant of the visual outcome after cataract surgery is the severity of the preoperative maculopathy. For patients demonstrating pre-existing maculopathy, the post operative vision is often worsened by the development of a diffuse, exudative form of macular edema, particularly in women and older individuals. Cataract extraction has been shown to exacerbate or cause diabetic macular oedema (DME). Cystoid macular oedema (CME) is another common cause of poor vision in the postoperative period, and approximately 50% of patients with pre-existing maculopathy show evidence of persistent CME especially during the first few postoperative weeks (up to 6 months) ***(Pollack A et al, 1992)*** Patients undergoing extracapsular cataract extraction experience a higher rate of maculopathy progression when compared with groups undergoing phacoemulsification. The incidence of angiographic CME following extracapsular cataract extraction (ECCE) is 20 to 30% compared to 10 to 20% with phacoemulsification. CME following extracapsular cataract extraction was typically detected between 2 and 3 months after surgery**.. *(Charters L, 1997).*** Treatment of cystoid macular edema is often unsatisfactory. The available treatment options include steroids given posterior subtenon or intravitreally, laser photocoagulation (grid or focal), systemic carbonic anhydrase inhibitors, and Vitrectomy. Unfortunately laser photocoagulation of the macular region failed in improving the visual outcome in a substantial group of patients, which promoted interest in other treatment methods. Corticosteroids are known for their ability to inhibit the arachidonic pathway of which PG's are products and to down regulate VEGF's. Stabilization of the blood retinal barrier introduces a rationale for the treatment of diabetic macular oedema. ***(Abelson MB, 1992).*** Triamcinolone acetonide is a corticosteroid that has been used locally as periocular and recently intravitreal injections for the treatment of refractory diabetic macular edema. Intravitreal injection of triamcinolone achieves highest concentration of the drug at its site of

action. Animal studies have shown that the intravitreal –injected suspension maintained a depot lasting 4-6 weeks. In addition, triamcinolone has a vitreous half-life of 1.6 days compared to 2.5 hours for dexamethasone. ***(Jonas JB, et al, 2001).*** Careful clinical examination of the macula with slit lamp biomicroscopy allows diagnosis of macular oedema in most cases. Still, in some cases there is difficulty in identifying macular oedema ophthalomoscopically. In these cases Fluorescein angiography and Optical Coherence Tomography (OCT) are helpful in establishing or confirming the diagnosis in addition to evaluating the degree of vascular leakage and the macular thickness ***(Nussenblatt RB, et al, 1994).*** OCT is a powerful tool for detecting and monitoring a variety of macular diseases including macular edema. It is non-contact and non-invasive imaging technique that uses infrared optical illumination. The use of optical rather than acoustic waves enables higher resolution, cross sectional retinal imaging with a measurement approaching 10 µm which enables OCT of quantifying retinal thickness in eyes with macular edema. ***(Hee MR, et al, 1995).*** Fundus Fluorescine angiography (FFA) is a powerful imaging tool that has been widely used to detect integrity of blood retinal barrier, detecting signs of vessel leakage and signs of macular ischemia ***(Yannuzzi ,et al, 1988).***

AIM OF WORK:

To evaluate the efficacy of intravitreal triamcinolone acetonide injected during cataract surgery in the prophylaxis and management of postoperative macular oedema following uneventful phacoemulsification in diabetics.

SUBJECTS & METHODS

This study included 30 patients divided into 2 groups

Group A: Control Group

Inclusion criteria: included 2 subgroups:

1)-Diabetic patients without any excising macular edema (7 patients).

2)-Diabetic patients with pre-existing macular edema (8 patients).

Procedure:

Phacoemulsification with Posterior chamber intraocular lens implantation (PCIOL).

Group B:

Inclusion criteria: diabetic patients with or without preoperatively existing macular edema including patients with previous macular laser treatment with:

1) Visually significant cataract.
2) Visual acuity (VA) showing deterioration with best corrected visual acuity (BCVA) ranging from 3/36 to CF at 3 meters.
3) If any previous laser treatment was done (focal or grid), at least 3 month should elapse since the last treatment.
4) Fundus Fluorescein angiography showing signs of macular edema and vessel leakage.
5) OCT showing retinal thickness of 300 µm or more in the foveal center
6) Anterior segment examination showing clear cornea and no signs of active or previous uveitic attacks.

Exclusion criteria:

1-Glaucoma and ocular hypertension and steroid responders.

2-Uveitic patients.

3-Cases complicated with opening of the posterior capsule and vitreous loss during cataract surgery.

4-Cases with Optic nerve diseases.

5-Decreased ocular barrier function such as blepharitis, dry eye, chronic nasolacrimal duct obstruction and presence of filtering bleb.

6-Additional causes of systemic immunosuppression apart from diabetes mellitus, that would compound the risk of infectious endophthalmitis.

Procedure:

a)Phacoemulsification with PCIOL implantation

b)Intravitreal triamcinolone injection.

A dose of 8mg in 0.2 ml was be injected slowly through the inferior pars plana at the end of the surgery.

Surgical technique:

-Povidine iodine 5% application to the ocular surface for full asepsis.

-Retrobulbar or peribulbar anesthesia.

-Corneal incision, capsulorechxis and phacoemulsification of the nucleus.

-Irrigation aspiration of the lens matter and implantation of PCIOL under viscoelastic material.

-Under indirect ophthalmoscopy , using a 27-guag needle ,a doze of 8 mg of TA in 0.2 ml, is slowly injected through the inferior pars plana (3-5 mm from the 6 o'clock limbus) into the mid vitreous. The 27-gauge needle is used to prevent clogging of the TA particles. The injecting needle is obliquely inserted through the sclera in a tunnel fashion which helps to prevent escape of liquefied vitreous from the injection site and may decrease the remote risk of postoperative endophthalmitis.

-Closure of the corneal wound.

Triamcinolone acetonide being a crystalline milky liquid, remains in the vitreous for a few days after injection as a discrete white cloud with little or no surrounding reaction. Patients are informed that prominent floaters might be commonly encountered after treatment which subsides within a few days as the material drops out of the visual axis.

Both groups were subjected to:

Baseline evaluation:

1- Visual acuity:

The patient's visual acuity was measured preoperatively using Snellen's charts. Best-corrected visual acuity was assessed after the patient's refraction was determined.

2- Slit lamp examination

Thorough anterior segment examination was performed preoperatively to assess the density of cataract and to exclude the presence of any anterior segment pathology.

3- Indirect ophthalmoscopy

Fundus examination was done preoperatively to record the fundus state and asses the macular condition.

4- FFA

FFA was performed using image net, if the density of cataract permitted, to detect signs of vascular leakage as well as ischemia in patients who demonstrate clinically visible maculopathy.

5- Intraocular pressure (IOP)

IOP chart was done.

6- OCT:

Retinal map with 6 linear cuts 5mm length centered on fixation at 0, 30, 60, 90,120 and 150 degrees using OCT machine.

7- Color photographs

Color photographs of the fundus and anterior segment were taken at the first visit.

Follow up:

Follow up at 24 hours, 7 days,14 days,1 month,2 month, 3 month, 6 month and at any time the patient develops new symptoms.

Follow up included the following:

1-Visual acuity: Snellen chart.

2-IOP: maximum increase and its timing.

3-OCT: Retinal map to detect the maximum height and its timing.
4-Any complications of the procedure: hemorrhage, endophthalmitis and retinal detachment (RD).

RESULTS

This study was conducted to evaluate the efficacy of intravitreal triamcinolone acetonide injected during cataract surgery in the prophylaxis and management of postoperative macular edema following uneventful phacoemulsification in diabetics. All patients received a thorough explanation of the study design and aims, and were provided with written informed consent. The study included 30 eyes of 24 diabetic patients (15 males and 9 females), whose age ranged from 47 to 64 years (mean =53.5 years ±1.75) The patients had dense cataract as diagnosed by slit-lamp biomicrosopy and confirmed with fluorescein angiography (FA) to have clinically significant macular edema according to the Early Treatment Diabetic Retinopathy Study. The patients were randomly assigned to either receive Intravitreal injection of triamcinolone acetonide during phacoemulsification or not. Postoperative visual acuity, intraocular pressure, FA, and Optical Coherence Tomography were assessed, recorded, and compared in both the study and the control groups for up to 6 months postoperative. No eyes had a history of ocular hypertension, glaucoma or previous laser treatment. Cataract surgery was performed routinely using the phacoemulsification technique. Following IOL implantation, TA was injected slowly through the infero-temporal pars plana at a dose of 8mg (0.2ml). A 27-gauge needle was used to prevent clogging by suspended corticosteroids particles. Indirect ophthalmoloscopy was used to confirm proper intravitreal localization of the. In the control group, no intravitreal injection was received. Patients were followed up at 1, 2, and 4 weeks then at 2, 3 and 6 months, and at any time the patient developed new symptoms, with monitoring of the response functionally (visual acuity), anatomically (OCT measurements), together with observing steroids-related and injection-related complications i.e. elevated intraocular pressure, retinal detachment, vitreous hemorrhages and endophthalmitis. The study and control groups were compared in all aspects. All patients completed 6 months of follow-up. On the first intraoperative day, all patients who received intravitreal triamcinolone complained of veiling of vision or musca for up to 1 week postoperatively. Crystals were located preretinaly in the vitreous cortex at the 6-o'clock position, and they did not however, optically interfere with visual assessment. Three months after the injection, the triamcinolone crystals resolved, and completely disappeared out of the vitreous cavity in 80% of the cases. At six months, the mean postoperative visual acuity in the study group was 0.6 (ranging from 0.4-0.9) which was significantly better than the control group (0.3 with a range of 0.1-0.4). Four patients of the study group required additional focal laser treatment for residual macular thickening. All patients in the control group received focal laser postoperatively. IOP were elevated in 40 % of patients in the study group from a baseline mean of 16$\pm$3 mmHg to a mean of 21.5$\pm$4 mmHg one month after injection, all brought under control with anti-glaucoma medications. One myopic patient experienced an elevation of IOP to 35 mmHg but was controlled with intensive anti-glaucoma treatment. Glaucomatous

damage to the optic nerve as determined by biomicroscopic examination was not noticed in any eye. One patient experienced subhyloid deposition of Triamcinolone particles at the macula which needed a longer time to clear compared to the rest of the patients. In none of the eyes included in the study did postoperative infectious endophthalmitis or proliferative vitreoretinopathy occur. None of the eyes showed progression of diabetic retinopathy. Show figure (1). For the 20 patients for whom fluorescein angiograms were available during the preinjection and postinjection periods, mean ± SD fluorescein leakage on the angiograms decreased significantly (from 29.13 ±18.85 mm^2 (range,8.27-50.0 mm^2) at baseline, to a minimal value of 25.28 ±17.3 mm^2 (range, 0.56-50.0 mm^2) during the follow-up period. Evaluated subjectively in a masked fashion, the postinjection angiograms of all 20 patients were graded to show less fluorescein leakage than on the preinjection angiograms. In the study group, mean ± SD intraocular pressure increased significantly (*P*<.001), from 16.3 ± 2.5 mm Hg at baseline, to a maximal value of 21.5 ± 4.7 mm Hg (median, 19.0 mm Hg; range, 16-35 mm Hg) during the follow-up period, and decreased again significantly (*P* = .03) to 17.7 ± 3.6 mm Hg at 5 months after the injection **(Table 1).** The intraocular pressure measurements taken at the end of the follow-up period did not vary significantly from those measured at baseline. During the study period, intraocular pressure was higher than 21 mm Hg in 10 (34.6%) of the 15 study eyes. In all these eyes, intraocular pressure could be normalized by topical antiglaucomatous medication. Glaucomatous damage to the optic nerve, as determined by biomicroscopy of the optic nerve head, was not detected in any eye.

Table (2) shows the mean ±SD for phacoemulsification parameters used in both groups.

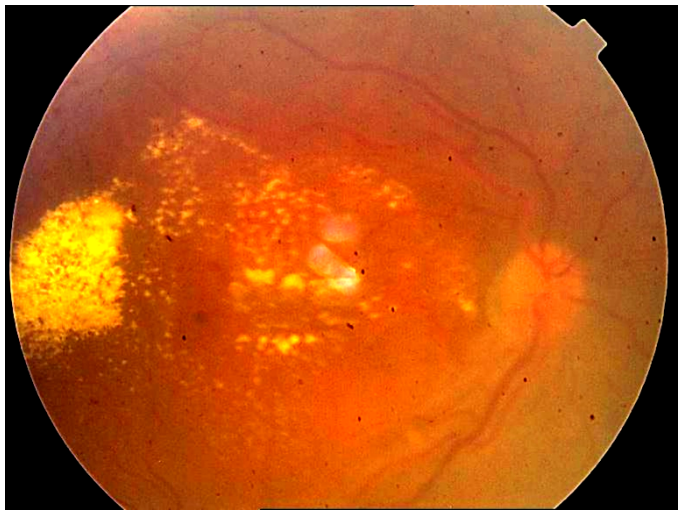

Figure (1) Fundus photography showing TA in the vitreous cavity

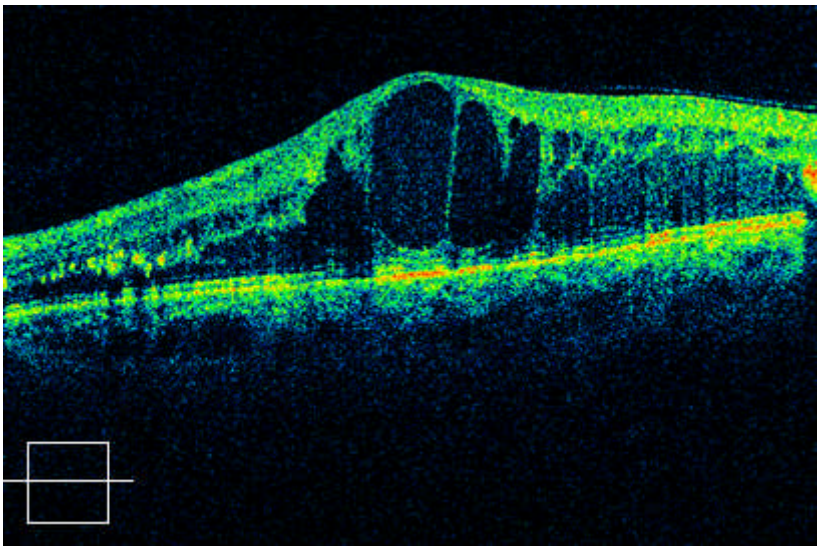

Figure (2) A patient with CME 1 day following phacoemulsification and IVT injection. Note the presence of increased macular thickness (664 µm).

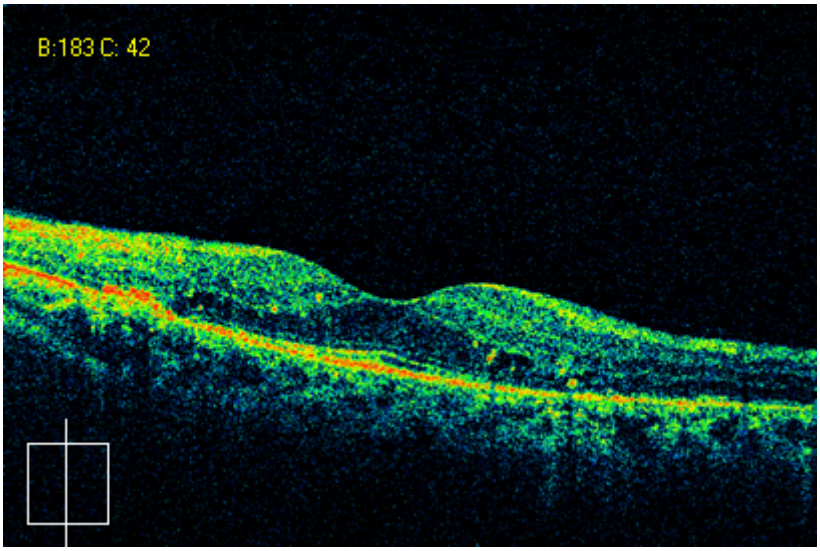

Figure (3) The same patient one month later after injection shows dramatic resolution of the macular edema to (306 µm).

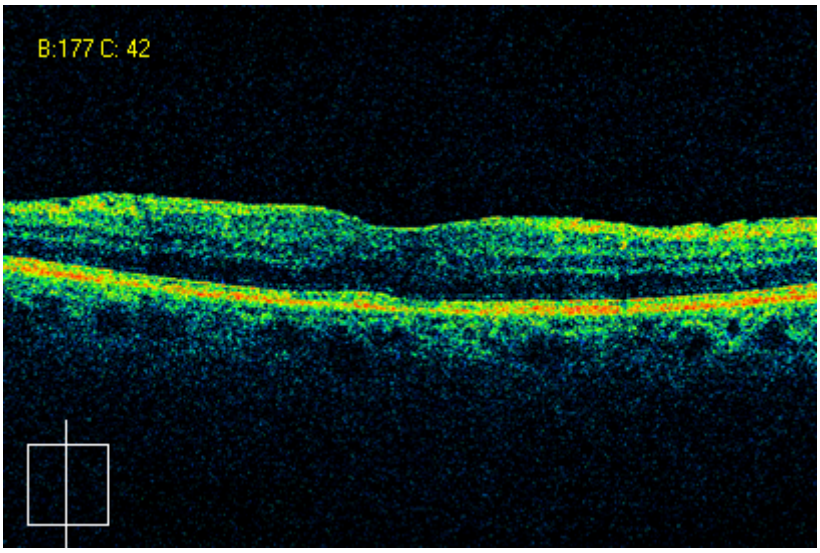

Figure (4) The same patient 3 months post operatively still showing resolution of the macular edema & within normal thickness (288µm)

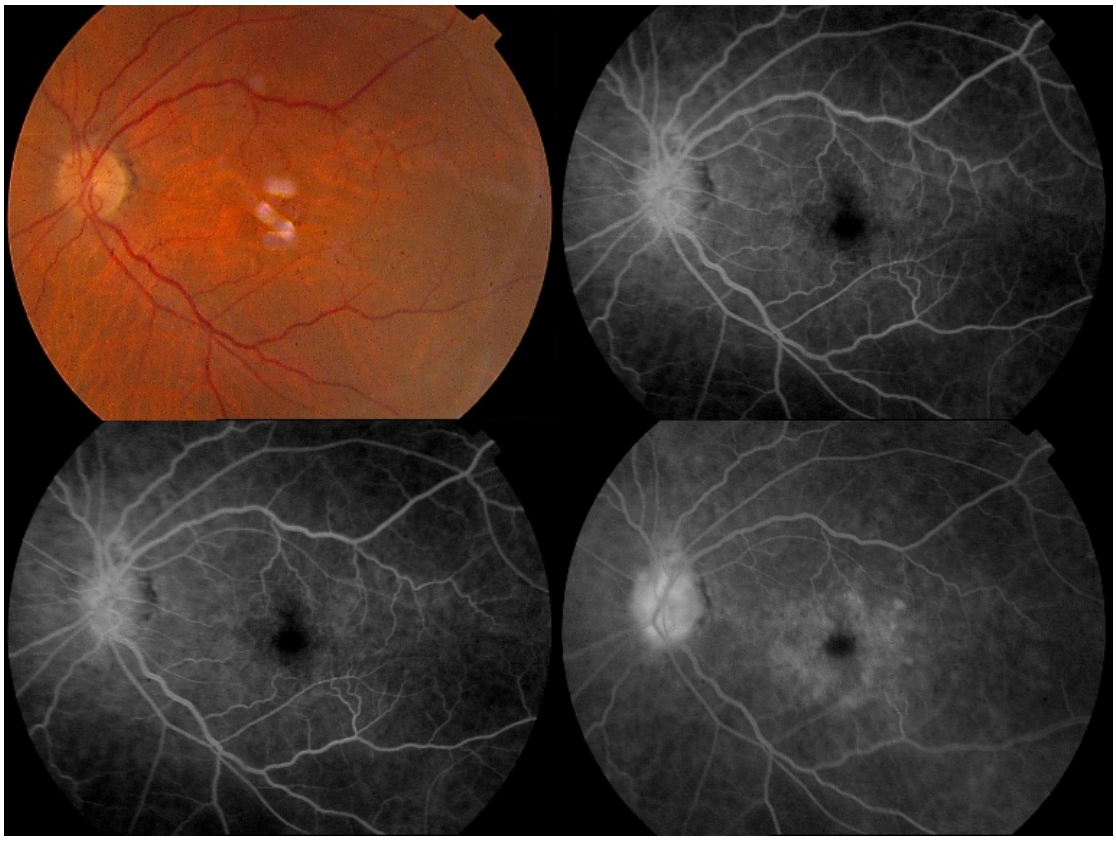

Figure (5) FFA of the same patient first week postoperatively.

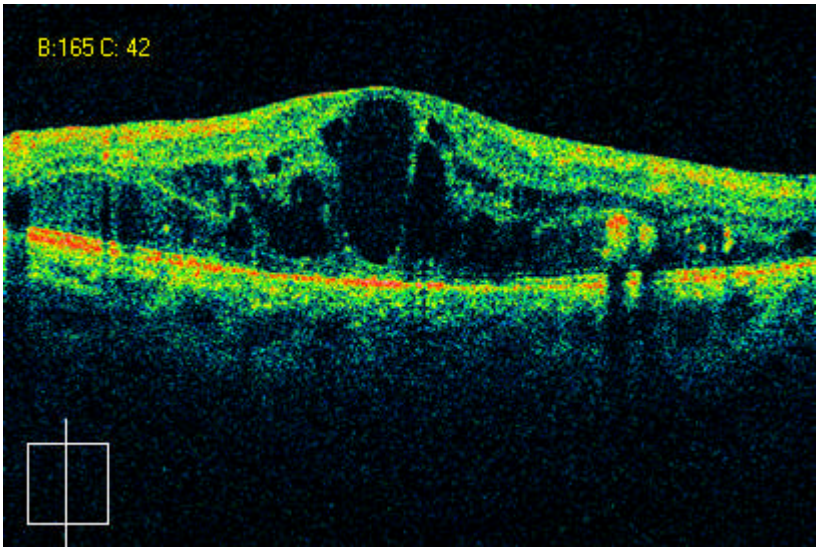

Figure (6) A patient with CME 1 week preoperatively. Note the presence of increased macular thickness (638 µm).

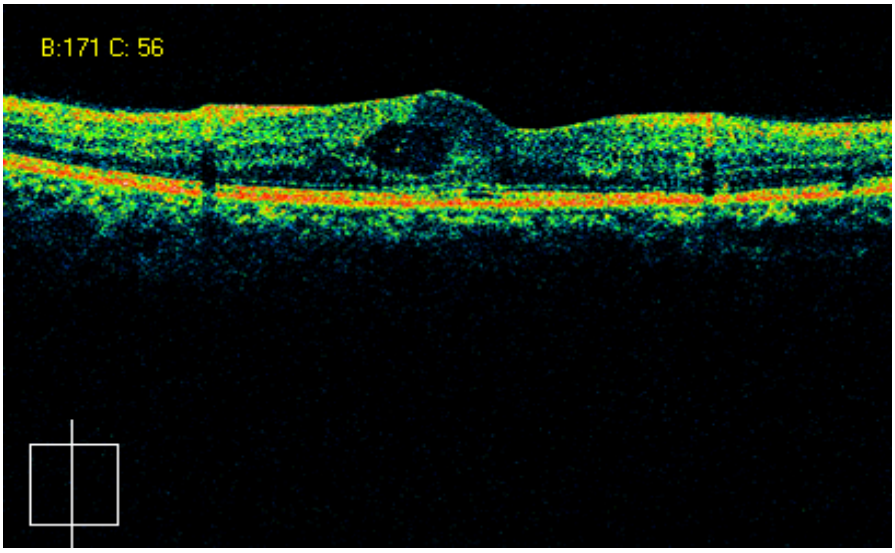

Figure (7) The same patient one month later after IVTA injection shows dramatic resolution of the macular edema to (319 µm).

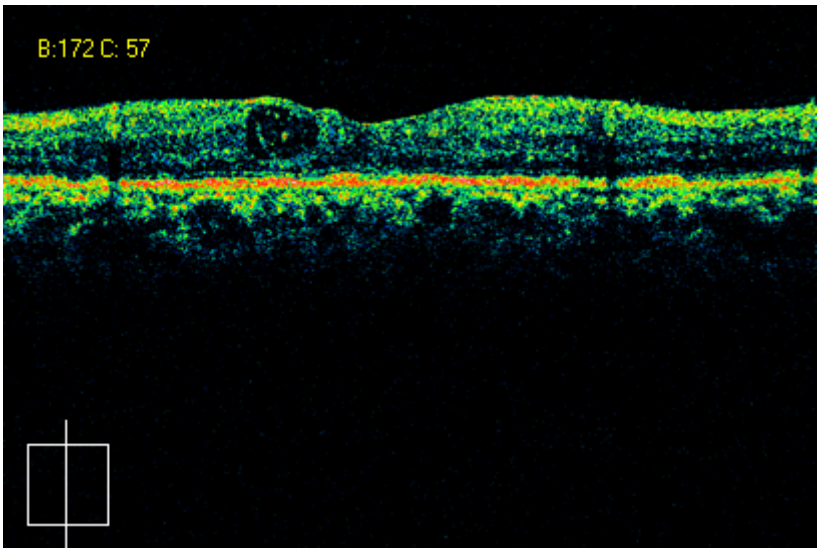

Figure (8) The same patient 3 months post operatively still showing resolution of the macular edema & within normal thickness (256µm)

The figures (9, 10) show correlation between the mean central foveal thickness in microns of both the study and control groups postoperatively as well as correlation between mean visual acuity in decimals of study and control groups postoperatively.

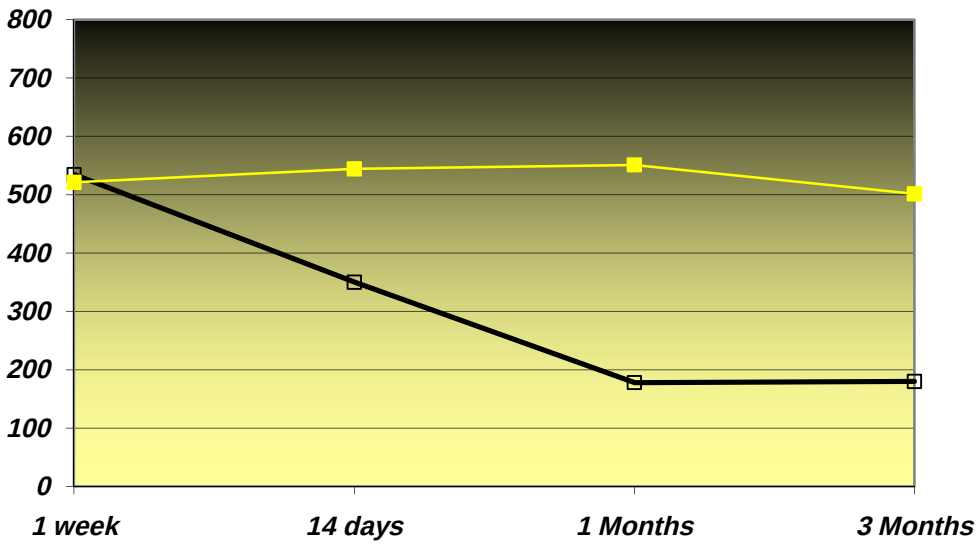

Figure (9): Mean Central Foveal Thickness in microns of study and control groups postoperatively.

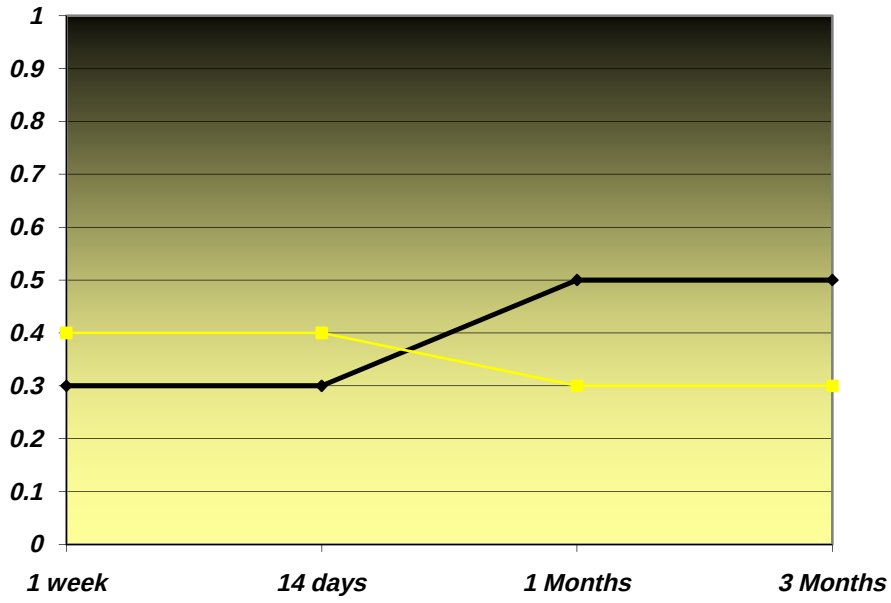

Figure (10): Mean Visual Acuity in Decimals of study and control groups postoperatively.

Discussion

Patients with clinically significant macular edema may irreversibly compromise the final visual outcome in these patients. Furthermore, it will delay the visual rehabilitation of patients seeking rapid visual improvement after cataract surgery. Triamcinolone delivered intravitreally at the time of cataract surgery may serve as a mean to buy the surgeon time to properly assess the degree of macular edema and its potential benefit from laser photocoagulation postoperatively. After intravitreal injection of triamcinolone, the drug is delivered rapidly to its site of action with maximum bioavailability. The results of the present study suggest that the intravitreal injection of triamcinolone during phacoemulsification may be beneficial as a treatment of clinically significant diabetic macular edema associated with dense cataract impeding fundus viewing needed for focal laser treatment preoperatively. The patients of the study group receiving intravitreal triamcinolone showed a significant improvement in visual acuity compared to the control group. Furthermore the results of the OCT in the study group showed marked reduction of the foveal thickness compared to the control group. All patients in the control group required focal laser therapy following cataract surgery compared to only 33% of the study group.

Potential injection-related complications include retinal detachment, vitreous hemorrhage, and endophthalmitis **(Jonas JB, 2001)**. No such complications were experienced in the current study group. All eyes experiencing IOP elevation were treated with topical antiglaucoma, with subsequent return to normal levels **(Wingate RJ. 1999).** This study does not aim to substitute laser treatment of clinically significant macular edema with intravitreal triamcinolone. Laser treatment still is the standard treatment with known

risks, and benefits to all ophthalmologists**.** However, intravitreal steroids combined with cataract surgery in patients with clinically significant macular edema and dense cataracts may serve as mean to control postoperative inflammation and exacerbation of the macular edema for a period of time until the ophthalmologist can properly evaluate whether the patient will benefit from supplementary laser treatment or not.

CONCLUSION

DME remains a significant cause of visual loss for patients with diabetes mellitus. Phacoemulsification with intravitreal TA in patients with CSME appears to be a safe intervention to avoid the postoperative exacerbation of the edema in patients with dense cataract precluding macular laser treatment. TA may serve as mean to control postoperative inflammation and prevent exacerbation of the macular edema. Postoperative laser treatment may be needed in some cases to augment the effect of intravitreal TA.

SUMMARY

Macular edema is the most frequent cause of visual loss among patients with diabetic retinopathy. It may be present at any level of retinopathy. The edema is caused primarily by breakdown of the inner blood retinal barrier and increase vascular permeability. Recent Studies suggest that inflammatory mediators such as Prostaglandin's (PG's) and the Vascular Endothelium Growth Factor (VEGF), are at least partially responsible for the increased vascular permeability. Cataract extraction in diabetic patients is commonly indicated, both for visual rehabilitation and for improved visualization of the fundus. Unfortunately the visual prognosis for diabetic patients undergoing cataract surgery is guarded, mainly because of the risk for worsening retinopathy levels and exacerbation of macular edema. An important determination of the visual outcome in cataract surgery is the severity of the preoperative maculopathy. For patients demonstration pre-existing maculopathy, the post operative vision is often worsened by the development of a diffuse, exudative form of macular edema, particularly in women and older individuals. Cataract extraction has been shown to exacerbate or cause diabetic macular oedema (DME). Cystoid macular oedema (CME) is another common cause of poor vision in the postoperative period, and approximately 50% of patients with pre-existing maculopathy show evidence of persistent CME especially during the first few postoperative weeks (up to 6 months). In addition, up to 50% of eyes with diabetic retinopathy but no prior DME, may develop DME following uncomplicated surgery. 30% or more of these eyes have a final visual acuity of less than 20/40. Treatment of cystoid macular edema is often unsatisfactory. The available treatment options include laser photocoagulation (grid or focal), systemic carbonic anhydrase inhibitors, Vitrectomy or steroids given posterior subtenon. Unfortunately laser photocoagulation of the macular region failed in improving the visual outcome in a substantial group of patients, which promoted interest in other treatment methods. Corticosteroids are known for their ability to inhibit the arachidonic pathway of which PGs are products and to down regulate VEGF's. The Aim of this study is to evaluate the efficacy of intravitreal Triamcinolone acetonide injected during cataract surgery in the prophylaxis and management of postoperative macular oedema following

uneventful cataract surgery in diabetics. The study included 2 groups, Group A included 15 patients divided into 2 subgroups 1)-Diabetic patients without any excising macular oedema., 2)-Diabetic patients with pre-existing macular edema. Patients in Group A were subjected to phacoemulsification with Posterior chamber intraocular lens implantation. Group B included 15 diabetic patients with or without preoperatively existing macular oedema including patients with previous macular laser treatment with visually significant cataract. Patients were subjected to Phacoemulsification with PCIOL implantation and Intravitreal triamcinolone injection (dose of 8 mg in 0.2 ml will be injected slowly through the inferior pars plana). The results of the study showed that phacoemulsification with intravitreal TA in patients with CSME appears to be a safe intervention to avoid the postoperative exacerbation of the edema in patients with dense cataract precluding macular laser treatment. TA may serve as mean to control postoperative inflammation and prevent exacerbation of the macular edema. Postoperative laser treatment may be needed in some cases to augment the effect of intravitreal TA.

TABLES

Table (1) shows the mean and standard deviations (SD) of different follow up criteria in both groups,

Table (1): Means and SD of follow up criteria in both groups.

	Mean ± SD (Range)	
	Group A (Control)	Group B
Age	54.5± 10.6 (47-62)	57±9.89 (50-64)
IOP Baseline 3 months 6months	 16.8± 2.3 16.5± 2.8 16.7± 2.5	 16.9± 2.5 21-3± 4.7 17.7± 3.6
Macular Volume OCT 3 months 6 months	 6.0± 4.5 3.4 mm^3	 7.8±9.8 2.4 mm^3
Visual acuity	0.3 (0.1-0.4)	0.6 (0.4-0.9)
Foveal thickness 3 months 6months	 7.0±11.7 4.6±14.7	 20.4±32.6 17.8±44.0

Table (2): phacoemulsification parameters used in both groups.

Factors	Mean ± SD		
	Group A (Control)	**Group B**	**P-Value**
Grade of the nucleus	2.4± 0.68	2.3±0.60	0.8403
Ultrasound time (seconds)	50.4± 22.2	47.8±18.0	0.8323
Ultrasound power (millijoule)	35.5± 7.0	34.2± 4.5	0.9183
Infusion volume (ml)	75.6±22.1	72.3±22.3	0.5583

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حقن مادة التريامسينولون داخل الجسم الزجاجي اثناء عملية المياه البيضاء للوقاية و العلاج من وذمة الشائبه الصفراء

د. واجده سعد بنيان*

ألخِلاصه :

وذمة البقعة الصفراء هو السبب الأكثر شيوعا لفقدان البصر بين المرضى الذين يعانون من اعتلال الشبكية السكري. قد تكون موجودة في أي مستوى من اعتلال الشبكية. ويتسبب في المقام الأول للوذمة انهيار حاجز الدم في الشبكية الداخلية وزيادة نفوذية الأوعية الدموية. الدراسات الأخيرة تشير إلى أن وسطاء مثل التهابات في بروستاجلاندين (لPG) والبطانة الوعائية نمو عامل (VEGF) ، ومسؤولة جزئيا عن زيادة نفاذية الأوعية الدموية .ويشار عادة استخراج الساد في مرضى السكري قد بسبب خطر لمستويات اعتلال الشبكية وتفاقم تدهور وذمة البقعة الصفراء.

والهدف من هذه الدراسة هو تقييم فعالية حقنة التريامسينولون داخل السائل الزجاجي خلال جراحة الساد في الوقاية ما بعد الجراحة من وذمة البقعة الصفراء في مرضى السكري.

أثبتت نتائج البحث حقن مادة التريامسينولون داخل الجسم الزجاجي اثناء عملية المياه البيضاء يعتبر من الحلول الجيدة لعلاج الارتشاح السكري للمقولة في المرضى الذين يعانون من مياه بيضاء متقدمة تحول دون اجراء ليزر. في بعض الحالات يحتاج المرضى الي عمل جلسة ليزر بعد الجراحة لتوثيق تاثير التريمسينولون.

* قسم العيون/كلية الطب /جامعة ذي قار