

Treatment of Pseudophakic Cystoid Macular Oedema

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ABSTRACT

Cystoid oedema of macula is the most frequent problem following non-complicated cataract surgery, which leads to weakening of visual acuity. We have studied the treatment outcomes of the topical therapy with corticosteroids and nonsteroid antirheumatics during prospective randomized study in comparison with orally applied acetazolamide. However, 8 weeks later in treatment was observed a positive effect in both groups, on the eyes of patients, treated with acetazolamide, resorption of the oedema was faster, more intensive and ultimate visual acuity was statistically significantly better. Results demonstrated a benefit of combined therapy with antiinflammatory drugs and orally prescribed acetazolamide in pseudophakic macular oedema.

Key words: *pseudophakic cystoid macular oedema, treatment*

Introduction

Cystoid macular oedema (CME) is one of the most frequent causes of unexpected poor visual acuity following cataract surgery. It usually occurs 4 to 6 weeks after the surgery. It's typical side-effect is visual acuity, which is a consequence of fluid accumulation in cystoid areas of the predominantly external plexiform foveae layer¹. Cystoid oedema of the macula is detected by fluorescein angiography, through which one can best observe cystoid areas distributed in a radius form. It may occur at non-complicated cataract surgery in patients with eye diseases on healthy eyes, then as a consequence of intraoperative complications or in patients with the eye diseases; such are uveitis or diabetic retinopathy². At non-complicated extracapsular extraction and at CME cataract phacoemulsification it usually occurs in approx. 1% of the operated eyes¹. Although there have been several theories on this issue, an overwhelming opinion is that likely cause of CME after non-complicated cataract surgery is a release of inflammatory mediators (prostaglandin and similar substances), leading to increased permeability of the retinal capillaries due to which fluid accumulation in cystoid areas is to take place³. Ultimate objective of the CME treatment is to reduce macular oedema along with an improvement of visual acuity and to prevent development of foveal cyst

and lamellar macular rupture. There have been several therapeutic options for the CME treatment. Medicament therapy, with corticosteroids and non-steroid antirheumatics, which interrupts a course of prostaglandin synthesis has proven as successful. These drugs are applied topically, in a form of subtenonial injections – intravitro and systematically^{4–8}.

It is also possible to reduce macular oedema and to improve visual acuity through improvement of the pigment epithelium pump with inhibitors of carbonic anhydrase (acetazolamide-Diamox)^{9,10,11}. For eyes failing to react adequately to this therapy we tried grid laser photocoagulation or vitrectomy that would have an effect at incarceration or vitreous traction onto macular region^{12,13}. The favorable fact is that a numerous CME several months later tend to disappear spontaneously. However, in the cases when treatment is required, choice of an adequate medicament and adequate application course is disputable. The fact there is insufficient number of randomized controlled studies is making this situation even more difficult. We were aiming with this study to find an optimal medicament approach in treating cystoid oedema at non-complicated extracapsular and PHACO cataract surgery.

Patients and Methods

Prospective randomized study was carried out at Department for Ophthalmology from 2001 to 2004. The study covered patients after non-complicated extracapsular and PHACO cataract surgery. There were 14 eyes (14 patients) suffering from cystoid macular oedema. The study included the eyes with CME detected with fluorescein angiography and visual acuity less than 0.5 according to Snellen optotype.

Criteria for exclusion from the study were other eye diseases, which would jeopardize visual function as: glaucoma, uveitis, macular diseases, optic nerve disease and diabetes mellitus. Patients were divided into two groups.

The first group, 7 patients, were treated topically with 0.1% dexamethason solution 3x and flurbiprofen liquifilm also three times a day. The second group, also with 7 patients had identical topical therapy and acetazolamide prescribed orally in dosage of 250 mg 3 times a day along with 500 mg potassium chloride. Duration of treatment with acetazolamide was for a two weeks in the full dosage and for another two weeks with 250 mg daily and by the follow-up examination 125 mg a day.

The follow up examinations were carried out after two months by determining visual acuity and fluorescein angiography. Statistic processing was carried out by t-test for dependent samples in pairs and with Student t-test.

Results

Total number of eyes with cystoid macular oedema, meeting the study requirements were 14. Of which 5 were demonstrating minimal indications of inflammation in a form of cells in the anterior optic chamber and mild mixed injections.

In the first group were 7 eyes with the best corrected average visual acuity of 0.34 (± 0.12) against Snellen optotype. In the second group there were 7 eyes and average visual acuity was 0.32 (± 0.9),

Evident reduction of the oedema on all eyes was observed 8 weeks after the therapy with fluorescein angiography started. In the first group the leakage completely disappeared in two eyes, and in the other group in six eyes. An average best corrected visual acuity in the first group was 0.53 (± 0.14) and in the second group was 0.67 (± 0.10) (table 1). Statistically significant difference of visual acuity in both groups was evident before

and after treatment ($p < 0.05$). Clinical signs of intraocular inflammation disappeared in all eyes after 8 weeks of the therapy. In the first group, a tolerance to medicaments was good, with no significant side-effects. In the second group one patient had parasthesiae in fingers during first week, which occurred when a dosage of acetazolamide was decreased and one patient suffered from a transient nausea.

Discussion

This prospective study covered 14 eyes (14 patients), affected with cystoid macular oedema after non-complicated operation of cataract. The study included the eyes that underwent cataract surgery and extracapsular method with IOL implantation into posterior chamber and phacoemulsification since no single study has demonstrated significant differences in the CME incidence at both of these techniques³. Factors for exclusion from the study were other eye diseases that would jeopardize visual function such are: glaucoma, diseases of macula, diseases of optic nerve, uveitis and diabetes mellitus. These diseases could with their respective therapy impact the CME treatment course and in the cases of uveitis and diabetic retinopathy there has been a frequent occurrence of CME even without cataract surgery.

The eyes affected with cystoid oedema of macula in the first group which received topical therapy with 0.1% dexamethasone and flurbiprofen have demonstrated a significant improvement after 8 weeks of the therapy. In the second group which also received the therapy in combination with orally prescribed acetazolamide has occurred more evident improvement of visual acuity and the oedema disappeared on 6 to 7 eyes. Difference between these groups was statistically significant, reinforcing the argument that besides a standard anti-inflammation topical therapy on eyes affected with CME is to be introduced acetazolamide, which speeds and intensifies resorption of the macular oedema. Faster resorption of oedema is required before irreversible structural transformation of macula is to occur. Our results on favourable effects of acetazolamide were in line with former studies of other peers^{10,11}. Side-effects of the medicament were not of such an intensity that would require discontinuation of the treatment.

Disappearance of all signs of uveal inflammation in both groups along with an improvement of oedema reinforces the inflammation theory on CME occurrence. Our results reinforced a need for anti-inflammation therapy

TABLE 1
AVERAGE BEST CORRECTED VISUAL ACUTITY WITH CME PRIOR/POST TREATMENT

| | Number of eyes | Average best corrected visual acuity prior to treatment | Average post treatment best corrected visual acuity |
|----------|----------------|---|---|
| Group I | 7 | 0.34 (± 0.12) | 0.53 (± 0.14) |
| Group II | 7 | 0.32 (± 0.09) | 0.67 (± 0.10) |
| Total | 14 | 0.33 (± 0.10) | 0.60 (± 0.13) |

on eyes affected with CME¹⁴. Although some studies challenged a need for corticosteroids at CME, we had no doubts on their favourable effect for a proper anti-inflammation effect, and it was supported by other authors, too^{8,15}. Therefore we may conclude that the most favourable option for the CME treatment following up non-complicated cataract surgery is a combination of

corticosteroids therapy with NSAID topically along with orally applied acetazolamide due to intensifying and speeding up the resorption of oedema. Limitation of the study was a small number of the examinees eyes, which calls for necessity to resume further researching of this issue.

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LIJEČENJE PSEUDOFAKIČNOG CISTOIDNOG EDEMA MAKULE

SAŽETAK

Cistoidni edem makule je čest problem nakon nekomplicirane operacije katarakte i dovodi do slabljenja vidne oštine. Prospektivnom randomiziranom studijom istraživali smo rezultate liječenja topikalnom terapijom kortikosteroidima i nesteroidnim antireumaticima u usporedbi sa peroralno k toj terapiji pridodatim acetazolamidom. Iako je nakon 8 tjedana liječenja uočen pozitivni učinak u obje skupine, kod očiju pacijenata liječenih sa acetazolamidom resorpcija edema bila je brža, intenzivnija te je i konačna vidna oština bila statistički značajno bolja.