A Case of Cystoid Macular Edema Following Nd:YAG Capsulotomy

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CLINICAL PEARLS

1. Although very unusual, development of cystoid macular edema is a visually-threatening potential complication after Nd:YAG capsulotomy.

2. While there is likely an inflammatory component, no clear mechanism of pathophysiology has been determined.

3. Similar to after cataract extraction, acute cystoid macular edema secondary to Nd:YAG capsulotomy may be treated effectively with anti-inflammatory topical agents.

BACKGROUND

Posterior capsular opacification (PCO) is the most common cause of decreased vision in pseudophakic patients and is most commonly treated with a Nd:YAG laser capsulotomy. Although very unusual, development of cystoid macular edema (CME) is a visually-threatening potential complication after this procedure. Topical treatment with 0.5% Ketorolac and 1% Prednisolone acetate was shown to decrease macular edema within a two week period.

CASE PRESENTATION

A 71-year-old male presented with decreased visual acuity in his right eye two months after uncomplicated Nd:YAG laser capsulotomy. Ocular history includes bilateral cataract extraction, vitrectomy, ocular hypertension, and anterior chamber IOL with past stable visual acuity at 20/40 in the right eye. On presentation to clinic, the right eye’s best corrected visual acuity had decreased to 20/400, and macular edema was visible on dilated fundus exam. Macular OCT showed significant cystoid macular edema with central subfield thickness of 610 um (Fig. 1, 2).

TREATMENT

The patient was treated with topical Ketorolac 0.5% ophthalmic solution QID and PredForte 1% ophthalmic solution QOD. After two weeks of treatment, the macular edema had significantly decreased, with central subfield thickness of 555 um (Fig. 3), although visual acuity was not restored. The patient was referred to a retinal specialist for fluorescein angiography and further treatment.

CLINICAL TESTING

While several studies have published findings of cystoid macular edema following Nd:YAG capsulotomy and many hypotheses have been proposed, no clear mechanism of pathophysiology has been determined. Most theories of pathogenesis involve different modes of prostaglandin release in the posterior segment. It is thought that these inflammatory mediators may weaken fragile macular capillary walls causing leakage of fluid and resultant macular edema. The evidence of improved macular edema with use of an anti-inflammatory agent strengthens this theory. Since topical Ketorolac and Prednisolone acetate were shown to effectively treat the CME, it can be inferred that the pathogenesis is at least somewhat inflammatory in etiology. The patient in this case has a complicated ocular history involving cataract surgery and vitrectomy. Though there is no conclusive evidence in the literature, it can be postulated that previous trauma and ocular surgeries may have made this patient hypersensitive to even the smallest assault on the globe and more susceptible to complications.

CONCLUSION

Nd:YAG laser capsulotomy is the standard technique used to clear an opacified intraocular lens. This procedure is generally safe, quick, and effective; however, rarely reported side effects include increased intraocular pressure, retinal detachment, and cystoid macular edema. Similar to after cataract extraction, acute cystoid macular edema following Nd:YAG capsulotomy may be treated effectively with anti-inflammatory topical agents. Knowledge of potential complications following this procedure can better help eye care providers prepare patients for post-operative visual expectations.

REFERENCES


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