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ISSN: 2684-1622
Journal of Eye Diseases and Disorders

Commentary

Treatment of Uveitic Macular Edema with Subconjunctival Injections of Triamcinolone Acetonide

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ABOUT THE STUDY

Macular Edema (ME) is a common complication of uveitis and is responsible for a substantial amount of visual impairment among patients with uveitis. ME is believed to result from fluid leakage across the blood-retinal barrier and fluid accumulation in the macular region, sometimes with a characteristic distribution in the outer plexiform layer and subretinal area. Corticosteroids are the first choice for treating Uveitic Macular Edema (UME), while long-acting and sustained release implants represent the newest treatment method. However, immune suppressants such as cyclosporine, methotrexate, azathioprine and mycophenolate mofetil can only be used specifically for chronic and intractable UME. Moreover, various newly developed biological agents, such as anti-VEGF, interferon-α and anti-TNF, have provided options for UME pharmacotherapy.

Triamcinolone Acetonide (TA), a long-acting glucocorticoid, is still widely used because of its efficacy and affordable cost, but the use of local applications is controversial. Periocular or intraocular injections of TA have been previously reported in detail. However, few studies have been conducted on subconjunctival injections of TA for treating UME.

UME is frequently encountered in patients with uveitis (20.5% in the clinic) and can cause permanent vision loss. The management strategies vary significantly as no optimal strategy exists. Periocular injections or intraocular injections of TA and intraocular sustained-release glucocorticoid implants have been previously reported in detail. Recently, the POINT trial compared the effectiveness of 3 methods of administering regional corticosteroids for UME, including periocular injections of 40 mg TA (periorbital floor or posterior sub-Tenon's approach), intraocular injections of 4 mg TA and a 0.7 mg dexamethasone intravitreal implant. The results showed that all treatment groups had clinically meaningful reductions in central subretinal thickness compared with baseline.

Efficacy and tolerability of subconjunctival injection of TA, subtenon TA and intravitreal dexamethasone implants and showed improvements in CMT with no significant. Bae et al. reported that the eyes treated with peribulbar injections of 40 mg TA showed reductions in CMT. However, the curative effect declined after 3 months. Similarly, Henry A. Leder et al. also reported that the eyes treated with a single posterior-subtenon TA injection had clinically resolved 1 month after the injection, and the eyes had clinically resolved 3 months after the injection. However, another recent study administered periocular injections of 40 mg TA using a periorbital floor or posterior subtenon approach, and the percentage of CMT reduction was only 23% after 2 months.

Subconjunctival hemorrhage is also a well-known but trivial side effect. One case of conjunctival ulceration caused by a subconjunctival injection of 40 mg triamcinolone has been reported; other reported side effects of subconjunctival triamcinolone acetonide include infectious scleritis, blepharoptosis, mydriasis, conjunctival ischemia and conjunctival necrosis. These side effects were not observed in our patients, which may be due to the halved dose.

Subconjunctival injection, which could be performed in the outpatient department, is much easier to administer than posterior subtenon injection and intravitreal injection, which must be performed in the operating room. On the other hand, subconjunctival injections are more likely to cause IOP elevation, although elevated IOP could be well controlled by application of 1 or 2 types of topical IOP-lowering agents. Furthermore, subconjunctival TA deposit removal may cause less damage than intravitreal injection or posterior subtenon injection in patients suffering IOP elevation who require pars plana vitrectomy or trabeculectomy.

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Received: 02-May-2022, Manuscript No. JEDD-22-17145; **Editor assigned:** 04-May-2022, PreQC No. JEDD-22-17145(PQ); **Reviewed:** 18-May-2022, QC No JEDD-22-17145; **Revised:** 25-May-2022, Manuscript No. JEDD-22-17145(R); **Published:** 02-Jun-2022, DOI: 10.35248/2684-1622.22.7.171

Citation: Huang B (2022) Treatment of Uveitic Macular Edema with Subconjunctival Injections of Triamcinolone Acetonide. J Eye Dis Disord. 7:171.