When not to Treat Uveitis

Dale Henderly, M.D. & Ronald E. Smith, M.D.

Much has been written about various forms of treatment for uveitis, and the literature is replete with documentation of the importance of corticosteroids and other immunosuppressives in such cases. However, it is not uncommon for same forms of ocular inflammation to be over-treated with such medications. In fact, in a referral practice situation. I see numerous patients who have been unnecessarily treated with corticosteroids for months or years. Since the complications of these medications are by no means benign for the eye or for the patient's general health, some discussion of uveitis cases that do **not** warrant corticosteroid therapy seems important.

IMPORTANCE OF KNOWING THE NATURAL COURSE OF THE DISEASE

Designing therapeutic strategies depends to a large extent upon differentiating the precise diagnostic entity of uveitis; otherwise, the ophthalmologist is left with using very broad, non-specific corticosteroids. In some instances this may be the only course of action available, but in many cases it is possible to identify the syndrome and obtain data about the natural course. If the natural course of a particular form of uveitis is generally benign (as for example are most cases of pars planitis), then it is unnecessary to treat such cases intensively. On the other hand, if synechiae, secondary glaucoma and cataract are likely complications, as in cases of severe acute iridocyclitis associated with ankylosing spondylitis, then intensive corticosteroid therapy is indicated. These examples emphasize the necessity of attempting to establish the appropriate diagnosis in order to better predict the short-term and long-term course of that particular type of uveitis. When the natural history of the entity carries with it an insignificant effect on vision and very little long-term ocular damage, no therapy should be undertaken. However, when severe complications are probable, therapy is always mandatory.

Before undertaking therapy, it should be clear that the therapy will be less likely to produce ill effects than will the natural course of the disease. In Fuchs' heterochromic cyclitis, for example, cataract formation is part of the natural course in many cases. Although topical corticosteroids may reduce the inflammation in Fuchs', the medication itself carries additional risk for cataract formation, thus clearly its use will not prevent cataract formation.

In cases of extremely severe ocular inflammation in which the structural elements of the eye have already been irreparably damaged, the use of high-dose systemic corticosteroids or non-steroidal immunossupressive agents would probably not be indicated. Potential for improvement of function of the eye should be present before using such medications.

SPECIFIC PROBLEMS IN THERAPY

There are some specific situations in which the ophthalmologist must consider the role of non-treatment. In all instances, consideration must be given to the benefit of anti-inflammaory therapy versus the likelihood of deterioration of function without medication.

1. Chronic low grade iridocyclitis

The management of patients with low grade chronic iridocyclitis is problematic. This is especially true in children wth iridocyclitis associated with juvenile rheumatoid arthritis. These patients have chronic flare in the anterior chamber which may be as much as 3+ to 4+, but have very few aqueous cells. It is not uncommon, however, for such patients to be treated intensively with topical and even systemic steroids in order to suppress the aqueous flare component. This therapeutic goal is unrealistic, since chronic breakdown in the blood aqueous barrier will not be reversed by corticosteroid therapy. Such patients will probably have significant flare even a few aqueous cells indefinitely, with or without corticosteroid therapy. It is therefore important to differentiate between significant active inflammation (2+ to 3+ cells in the aqueous) and inactive disease with chronic flare resulting from damage to the blood aqueous barrier. When this distinction is not clearcut, a short therapeutic trial on corticosteroids can be helpful to determine the actual effect on any cellular reaction in the anterior chamber. Patients with chronic flare and a few cells will probably benefit from daily use of dilating drops at night to prevent synechiae

University of Southern California School of Medicine. Department of Ophthalmology and Estelle Doheny Eye Foundation — Los Angeles, Ca

formation: they generally do not benefit from anti-inflammatory therapy.

2. Toxoplasmic retinochoroiditis

Not all cases of active retinochoroiditis associated with toxoplasmosis need to be treated as the natural course of these lesions is to spontaneously resolve in a matter of weeks or months. It is the location of the lesion and the severity of the exudative reaction that determines the need for therapy. Small lesions in the periphery of the retina that do not threaten the optic nerve or macula, do not require therapy unless there is an intense exudative reaction in the vitreous cavity that causes retinal edema at the posterior pole. Topical corticosteroids can reduce significant anterior chamber "spillover" reaction if present. Otherwise, observation is the only treatment required. While patients with active toxoplasmic retinitis in the peripheral retina may complain of floaters and decreased vision (due to vitreous haze), this reaction will usually resolve spontaneously and lead to no ocular sequelae.

3. Pars planitis (chronic cyclitis, peripheral uveitis)

Patients with pars planitis are very often needlessly treated with topical subconjunctival and systemic corticosteroids. These patients may have an exuberant cellular reaction in the vitreous cavity that is very frightening to the ophthalmologist; however, this may not create any problem for the patient other than the presence of "floating spots". If the vision is 20/30 or better and cystoid macular edema is ruled out by clinical examination and fluorescein angiography, then therapy is not necessary. If there is a marked anterior chamber reaction (which is unusual in patients with pars planitis), topical steroids alone can be used. Topical corticosteroids, however, have no role in the management of the vitreous cellular reaction of pars planitis since they do not penetrate the eye. It is not uncommon, however, for a patient with pars planitis and active vitreous reaction to undergo a several month course of topical steroids which can lead to cataract and glaucoma. The main indication for steroid treatment of pars planitis is decreased vision due to cystoid macula edema. The presence of vitreous cells alone is not sufficient cause for steroid therapy as they will remain present for years, until the disease process gradually and spontaneously resolves.

4. Fuchs' heterochromic iridocyclitis

Except for pars planitis, this entity is the most overtreated of all forms of ocular inflammation. The etiology of Fuchs' heterochromic iridocyclitis is unknown, but it is probably, in actuality, a group of entities with the common denominator of small keratic precipitates and heterochromia in one eye. It is probably not a true inflammation in all cases and therefore would not be expected to respond to corticosteroids. While it is recommended that a therapeutic trial of corticosteroids be used for a few weeks in most such cases, the long-term use of topical or periocular corticosteroids is not recommended in most instances. The vast majority of instances of Fuchs' cyclitis do not respond to corticosteroids and can be expected to exhibit 1+ cells, 1+ flare and some fine keratic precipitates indefinitely, with or without treatment. The chronic use of corticosteroids promotes the early onset of cataract and glaucoma, which are intrinsic of the natural course of the disease itself. At the same time, if the patient does show a favorable response to a trial of topical corticosteroids, then the therapeutic strategy should include their judicious use.

5. Ocular toxocara

Specific antihelminthic therapy for ocular toxocara has not been shown to be effective. In addition, corticosteroids, while useful in suppressing the inflammation in the exceptional case of ocular toxocariasis, have no long term role in the management of such cases. The isolated macular granulomatous form of ocular toxocara has usually already destroyed macular function, and unless it is progressing, will not improve with therapy. Peripheral toxocara granulomas are frequently "inactive " by the time the patient is seen by an ophthalmologist, and would not be expected to improve with anti-inflammatory therapy. Cases of diffuse endophthalmitis due to ocular toxocara are better candidates for the use of corticosteroids, and possibly for antihelminthics as well. A therapeutic trial of systemic steroids in such cases may be considered.

SUMMARY

I have discussed herein some of the general principles involved in the decision **not** to treat patients with ocular inflammation. It is often more difficult to not treat a patient than it is to embark on a course corticosteroid therapy since the patient expects some sort of therapy. At the same time, there are specific situations in which the use of corticosteroid drops are ineffective in which the side effects of such treatment can be worse than the disease process itself.

SUGGESTED READINGS

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