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Treatment Algorithm for Juvenile Idiopathic Arthritis-Associated Iridocyclitis

C. Stephen Foster, M.D.

Great progress has been made during the latter half of this century in the care of patients with iridocyclitis associated with juvenile idiopathic arthritis. The most major advance was the development ofcorticosteroids for systemic and ophthalmic use just after the mid-way point of this century, and the second major advance came through the admonitions of Jacobs and Spalter of New York and of Kanski in London for the routine screening biomicroscopic examination of youngsters with the pauciarticular form of JIA, screening for occult active intraocular inflammation, since the eyes of such patients commonly appear normal to the casual observer, and since the patients are often young and do not notice or express to parents small changes which are slowly developing as a result of active inflammation.

Still, even today, 12% of pauciarticular JIA children **go blind** as a result of the consequences of low grade chronic intraocular inflammation; and these children are typically under the longitudinal care of ophthalmologists. The reason for this sad fact is the "tolerance" of so many ophthalmologists for low grade inflammation. In their defense, they are simply trying to "do no harm," and so are trying not to overuse corticosteroids in their goal to treat the uveitis, trying to avoid the development of corticosteroid-induced side effects such as cataract and glaucoma. The vision-robbing consequences of the ophthalmologist-tolerated low grade uveitis occur extremely slowly, typically over a period of four to eight years. The end result is clear, and the literature is replete with testimony to the deleterious consequences of such "tolerance" of low grade uveitis: maculopathy, with macular edema, macular cysts, epiretinal membrane, optic neuropathy, and cyclitic membranes.

The major deterrent hampering ophthalmologists from advancing to more aggressive therapy in the quest for total abolition of all active inflammation is the fear of producing drug-induced problems. This is understandable, particularly since ophthalmologists use immunomodulatory agents so infrequently, and have little reason to keep abreast of data regarding immunomodulatory agent-induced side effects when agents are used in the lowdose technique typically employed in the care of patients with non-lethal, non-malignant diseases, and when they are used as single agents, rather than in a polypharmacologic way

in the care, for example, of patients with solid organ transplants. The truth is, used properly, non-steroidal inflammatory agents and the immunomodulatory agents have considerably less prevalence of significant drug-induced mischief than do systemic corticosteroids.

We have strongly advocated the philosophy of no active inflammation in children with JIA associated iridocyclitis. Further, we have suggested a step ladder algorithm approach in aggressiveness to achieve that goal of total abolition of all active inflammation. We advocate beginning in the usual way, with steroid therapy. Topical steroids, regional injection steroids, and even systemic steroids may be appropriate in the care a patient with JIA-associated iridocyclitis. If the patient's uveitis continues to recur every time the steroids are withdrawn, we suggest then moving on to chronic use of an oral non-steroidal anti-inflammatory agent. Tolectin and Naprosyn appear to be the ones which pediatric rheumatologists use the most, but the choice and dosage for any given pediatric patient should be made by the pediatrician or the pediatric rheumatologist.

If the patient's uveitis continues to recur despite the use, chronically of an oral non-steroidal anti-inflammatory agent every time the steroids are withdrawn, then we would advocate moving along to low dose once a week methotrexate therapy. This therapy has a splendid track record, both in efficacy and in safety, in the hands of rheumatologists caring for children with the joint manifestations of JIA. Our experience has been identical with respect to caring for the ocular inflammation consequences of JIA. It is true that the **potential** for drug-induced pathema exists, and therefore the level of physician involvement in longitudinal monitoring, the need for the hematologic studies, is greater once the commitment is made for use of any systemic immunomodulatory agent. In rare instances we find some other immunomodulator other than methotrexate, will be required to achieve the goal of total quiescence, but the number of instances in which this arises is quite small.

We believe that further reduction in the prevalence of blindness secondary to uveitis which occurs in patients with juvenile idiopathic arthritis will depend entirely on the increasing awareness of the effectiveness of this therapeutic algorithm and the willingness of increasing numbers of ophthalmologists and rheumatologists alike to employ such a philosophy and algorithm.