

Uveitis and Maculopathy

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Uveitis represents the third leading cause of blindness in developed countries, including the United States. The visual degradation of patients with this disease occurs, usually as a result of very slow damage to the macula, as a consequence of low grade but chronic inflammation. Occasionally some cataclysmic event will occur, as can be seen in an explosive episode of Behcet's retinopathy or in subretinal neovascularization in cases of choroiditis; but the vast majority of cases are secondary to chronic macula edema or to epiretinal membrane preretinal fibrosis. This phenomenon occurs with surprising frequency in patients who have even primarily anterior uveitis.

We believe, therefore, that treating active inflammation must be the paradigm shift, going forward in our care of patients with uveitis, rather than using a particular level of visual degradation before getting more aggressive in treating the inflammation. Only in this way, we believe can one hope for additional progress in reducing the prevalence of blindness secondary to the long-term consequences of chronic, low grade inflammation. For example, most practitioners across the United States today "require" the patient with active pars planitis to suffer visual deterioration to at least the 20/40 level before intervening therapeutically. Yet experience tells us (indeed, has told many others in the past) that a significant number of patients with pars planitis who do have a drop in vision to the 20/40 level will never recover vision to 20/20, simply because permanent damage to the macula intervenes. We believe, therefore, that the mere presence of inflammation at the pars plana, regardless of whether or not there is an effect on the macula, should be the primary indicator for therapy, employing the stepladder approach in aggressiveness of treatment previously outlined in prior contributions to this web site. We hold the same belief in juvenile rheumatoid arthritis-associated uveitis, and indeed, in most uveitic entities.