Uveitis Treatment Options

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Thomas Albini, MD, moderator of the Uveitis Resource Center, spoke with Sunil Srivastava, MD of the Cleveland Clinic, Cole Eye Institute, about treatment options for infectious and non-infectious uveitis. Their conversation follows:

Thomas Albini, MD: *Syphilis is a relatively rare infectious ideology for uveitis, but one that is still important to diagnose. What are your treatment protocols?*

Sunil Srivastava, MD: Intravenous doses of penicillin for two weeks are sufficient for most cases of posterior uveitis associated with syphilis. In the presence of an inflammatory response, it's reasonable to put patients on systemic steroids and watch them carefully. Local steroids should be avoided because their use is associated with necrotizing-retinitis in these patients. When uveitis patients are diagnosed with syphilis, we bring in an infectious disease specialist and sometimes a neurologist because of the potential for brain infection.

TA: *What are your treatment strategies and guidelines regarding steroid use in toxoplasmosis, the most common infectious form of uveitis?*

SS: In most cases, I rely on monotherapy with Bactrim double strength twice a day. In immune-suppressed patients, I typically use triple therapy: pyrimethamine, sulfadoxine and leucovorin. I've begun to use intravitreal clindamycin in some patients, and in other cases where I want to be more aggressive I combine intravitreal therapy and systemic therapy.

TA: *Acute retinal necrosis is a less common, but also devastating cause of infectious uveitis. What are your therapeutic considerations for treatment?*

SS: I treat aggressively because it occurs fast and spreads quickly. I typically dose patients with 1 or 2 grams of valacyclovir, three times a day, and I also supplement with intravitreal foscarnet. I think acyclovir is a good choice, as well, and I inject patients every few days until the retinitis is under control. I also use a systemic steroid on these patients until the eye calms down because the inflammatory reaction is quite brisk. It's important to watch these patients carefully because there is a 50% to 70%. risk of retinal detachment.

TA: *What is oral prednisolone's role in posterior segment panuveitis and other sight threatening diseases?*

SS: Oral prednisone is fast and effective; the problem with it is its known side effects. In the case of sight threatening diseases, however, using oral steroids makes sense -- at least initially -- to get the eye quiet before moving on to the next form of therapy. In sight-threatening cases, I usually start with 1 mg per kg; and I stick with the initial dose for at least two weeks and then I slowly taper in increments of 10 mg over three months with a goal of being at or under 10 mg at three months. As far as steroid use, I weight base the dose going with 0.5 mg to .75 mg per kilo, and then I watch for a response before doing anything. I usually stick to that does for a week or two prior to tapering.

TA: *If patients do not have a full response to prednisone or are intolerant of systemic prednisone, or if they need doses higher than 10 mg a day for longer than three months, which steroid sparing agents do you commonly use?*

SS: CellCept mycophenolate is my first choice and methotrexate and immuryal are second line agents. I use a lot less cyclosporine now than I did when I trained as a retina fellow. Unfortunately, most people regress with uveitis and sometimes you have to use toxic agents, but thankfully that's rare.

TA: *What are the common clinical scenarios where you would use biologics, such as adalimumab and inflixamab?*

SS: I see this as an option for patients who fail on CellCept mycophenolate, methotrexate or immuryal who want to try something else and are not interested in having a steroid implant placed in their eye.

TA: *Under what circumstances do you think it is appropriate to use local approaches, such as sub-tenons injections vs intravitreal injections vs injection of Allergan's six month sustained release Ozurdex (dexamethasone intravitreal implant) vs Bausch & Lomb's Retisert (fluocinolone acetonide intravitreal implant).*

SS: It depends on the duration of therapy required. I think of sub-tenons injections as bridge therapy for someone who needs a little bit of extra therapy for a few weeks. Injections of triamcinolone or dexamethasone work well for patients who have aggressive posterior segment disease or cystoid macular edema that is not responding to other treatment. The fluocinolone implant works well at controlling inflammation and improving vision in patients who have recurrent cystoid macular edema (CME) or recurrent retinal vascular leakage. I've been using the Ozurdex implant a lot more because I it offers more stable pharmakinetics than triamcinolone. I use it in patients who have had uveitis and have been vitrectomized and need sustained therapy. We are also using it in pediatric patients who have recurrent inflammation, as a bridge therapy while they switch amino suppressant medications. Then I also use it in uveitis patients who have macular edema and vitreous haze and perhaps have not responded well to triamcinolone.

TA: *How long does the Ozurdex insert typically last?*

SS: In most patients, I get the full effect for three months and then partial effect for another three months, as it begins to wear off and the eye slowly becomes inflamed again.

TA: *If you have a patient who requires chronic treatment, and you're trying to decide between a steroid sparing agent such as mycophenalate vs a Retisert implant, what are the clinical characteristics that you take into consideration?*

SS: When patients have a significant amount of cystoid macular edema (CME) and retinal vascular leakage, I think they have a tendency to respond better to steroid therapy. I'm not convinced that using a single agent, like mycophenalate or methatrexate or inflixamab, will necessarily eradicate the macular edema consistently enough -- so those patients may do better with the Retisert implant, whereas, for patients who have multiple lesions or less vascular leakage or less macular edema, I tend to lean toward systemic therapy. ■