Acute Anterior Uveitis: an Overview

Ophthalmic and systemic evaluations are key to reducing secondary complications.

BY POUYA N. DAYANI, MD

Anterior uveitis (AU) is the most common form of uveitis with an annual incidence of eight cases per 100,000 people. Although most cases of AU are effectively managed with topical medications, some patients may suffer significant vision loss from associated complications such as glaucoma, macular edema, and cataracts. Moreover, many systemic disorders can be associated with, and manifest as, anterior segment inflammation. Therefore, AU requires prompt and thorough ophthalmic and systemic evaluation.

CLINICAL DESCRIPTION

Iritis, anterior cyclitis, and iridocyclitis are types of AU. The term acute uveitis should be used to describe a course of inflammation characterized by a sudden onset and limited duration, such as human leukocyte antigen (HLA)-B27–associated uveitis. The term chronic uveitis is used to describe persistent inflammation characterized by a prompt relapse less than 3 months after the discontinuation of therapy.

Patients presenting with AU typically report pain, photophobia, tearing, and ocular injection, but some individuals may be relatively asymptomatic. The key indicators of disease on examination with slit-lamp biomicroscopy are the presence of cells and flare in the anterior chamber. A 1-mm X 1-mm slit beam is used when assessing the anterior segment, and the presence and characteristics of hypopyon (such as height, fibrin content, and mobility) should be noted. A hypopyon can be seen in a number of disease processes but is most frequently associated with HLA-B27–associated uveitis, Behçet’s disease, infectious endophthalmitis, and certain systemic medications (such as rifabutin). Fibrin is a sign of aggressive disease and is typically seen with HLA-B27–associated disease or endophthalmitis. Hyphema is rarely seen and typically resolves without permanent damage.

Inflammatory cells can also aggregate and adhere to the corneal endothelium, forming keratic precipitates. Large, greasy keratic precipitates are suggestive of granulomatous disease and may help narrow the differential diagnosis. The most common causes of granulomatous inflammation include sarcoidosis, sympathetic ophthalmia, Vogt-Koyanagi-Harada syndrome, tuberculosis, syphilis, lens-induced uveitis, and multiple sclerosis-associated uveitis. Fine stellate keratic precipitates, on the other hand, are typically seen with herpetic uveitis and Fuchs’ heterochromic iridocyclitis (FHIC).

Band keratopathy is often found in younger patients with juvenile idiopathic arthritis and in older individuals who have chronic uveitis. Additional findings upon examination may include ciliary flush, pupillary miosis, posterior or peripheral anterior synechiae, dilated iris vessels, cataracts, and macular edema. It is important to differentiate iris vessel dilation from iris neovascularization. In the former, iris vessels have a regular radial orientation and typically resolve once the inflammation is controlled. Iris atrophy or sectoral iris abnormalities may suggest a herpetic etiology, whereas iris heterochromia is suggestive of FHIC. Iris nodules are suggestive of a granulomatous process. The IOP is typically low in uveitic eyes due to decreased aqueous secretion by the ciliary epithelium. However, an elevated IOP is often seen with herpetic uveitis, ocular toxoplasmosis, lens-induced inflammation, and Posner-Schlossman syndrome. Finally, uveitis in a quiet eye is commonly observed in patients with juvenile idiopathic arthritis, FHIC, and masquerade syndromes.

A dilated fundus examination is necessary to assess vitreous and posterior segment involvement. Optical coherence tomography can be helpful in detecting macular thickening or epiretinal membrane from chronic inflammation. Fluorescein angiography may show optic nerve or macular leakage, vascular staining, or other posterior segment findings that may further guide therapy.

DIAGNOSTIC EVALUATION

Although some patients may present with ocular signs and symptoms that are characteristic of certain diseases, most disorders are differentiated by their systemic char-
acteristics. Even after a thorough medical history and physical examination, up to 50% of patients are found to have idiopathic AU. The diagnosis partially depends on the extent of the evaluation; many cases initially diagnosed as idiopathic will later be attributed to a specific disorder. Masquerade syndromes—such as malignancy (leukemia, lymphoma), intraocular foreign body, pigment dispersion syndrome, and medication-related inflammation—should be considered in patients who are either very young or old, as autoimmune diseases are less common in older patients.

There is a lack of consensus with respect to the diagnostic evaluation of a patient presenting with a first episode of AU. Some physicians suggest no evaluation at all, although others recommend an exhaustive laboratory evaluation. Most agree, however, that a complete and detailed medical history and examination is necessary to create a targeted evaluation. Age, gender, ocular symptoms, and systemic findings can all be used to tailor the evaluation. In patients in whom the aforementioned does not suggest a specific disease process, some testing may be prudent. In patients with non-granulomatous disease, HLA-B27 analysis, fluorescent treponemal antibody absorption test, complete blood count with differential, and urine analysis may be a “high-yield” initial workup. For those with granulomatous disease, a chest X-ray, purified protein derivative test with anergy panel (or QuantiFERON-TB Gold test), and serum angiotensin-converting enzyme are also considered.

The HLA-B27 haplotype is found in 6% to 14% of control white patients and 0% to 4% of black patients. Given its frequency and distinct clinical presentation, HLA-B27–associated uveitis deserves specific mention. Among those in whom a diagnosis is made, the HLA-B27 allele is the most common identifiable factor (up to 47% in one study).1 Patients with HLA-B27 uveitis are often men and develop inflammation at a younger age. The acute AU is typically unilateral, severe, recurrent, alternating, and frequently associated with posterior synechiae and cataract. A fibrinous anterior chamber reaction is common. There may be an association with systemic disease, such as ankylosing spondylitis, Reiter’s syndrome, psoriatic arthritis, and inflammatory bowel disease.2 Monnet et al found that 78% of patients with HLA-B27 uveitis have extraocular disease that is often undiagnosed (almost always a spondyloarthropathy).3

Ocular inflammation is thus frequently the first indication of a systemic process, and routine rheumatologic evaluation was recommended for all patients presenting with HLA-B27–associated uveitis in the study by Monnet et al.

**TREATMENT**

Most patients with AU can be adequately managed with topical steroids—prednisolone acetate 1% being the most commonly used agent. Cycloplegia is used in combination with anti-inflammatory agents to prevent posterior synechiae and to minimize discomfort from ciliary muscle inflammation. My colleagues and I typically recommend scopolamine 0.25% twice daily.

Initially, topical steroids are instilled frequently, as often as every hour in many cases, with a gradual taper depending on the response. Difluprednate ophthalmic emulsion 0.05% (Durezol; Sirion Therapeutics, Inc.) has shown efficacy in treating ocular inflammation with less-frequent dosing. Studies suggest that Durezol dosed four times daily may be as effective as prednisolone acetate 1% taken eight times daily. Our experience with Durezol has been that, in certain cases, it can be particularly effective in treating ocular inflammation and uveitis-associated macular edema. In refractory cases or in patients with persistent macular thickening, periocular steroid injections or systemic corticosteroids may be considered. Patients presenting with severe bilateral disease or a chronic, recurrent disease course, especially those with noninfectious systemic associations, may benefit from systemic immunosuppressive therapy.

**CONCLUSION**

AU can present with a spectrum of ocular findings and systemic associations. Although frequently idiopathic, a thorough systemic review and a guided evaluation are important in the early diagnosis of systemic disease associated with anterior segment inflammation. Prompt diagnosis and treatment are essential in minimizing the secondary complications of ocular inflammation. With appropriate therapy and follow-up, most cases can be easily treated, and patients will retain good visual function.

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