Answer to Ophthaproblem *continued from page 893*

3. Acute anterior granulomatous uveitis

Uveitis is a broad term that refers to inflammation of the uvea, the pigmented part of the eye comprising the iris, ciliary body, pars plana, and choroid. It is the most common inflammatory condition of the eye,[1] and anterior uveitis is the most common form of uveitis in Western nations.[2] Uveitis is divided into subtypes based on anatomy, clinical course, and pathologic findings.[3] Anatomically speaking, inflammation of the iris or ciliary body is referred to as *anterior uveitis,* inflammation of the vitreous and peripheral retina is termed *intermediate uveitis,* and inflammation of the posterior retina or choroid is called *posterior uveitis.*[3,4] Temporally speaking, an acute episode of uveitis is characterized by sudden onset and a duration of 3 months or less. Chronic episodes have a duration of longer than 3 months, with relapse occurring within 3 months of discontinuing treatment.[3] This paper focuses on the more common acute anterior uveitis (AAU).

Pathologically, uveitis is classified as granulomatous or nongranulomatous, depending on the nature of the inflammatory reaction and the typical appearance of granulomatous inflammation. Even though there are some ambiguities in the nomenclature, granulomatous inflammation is usually characterized by epithelioid and giant cells. In contrast, nongranulomatous inflammation demonstrates lymphocytes, polymorphonuclear leukocytes, and plasma cells.[4,5]

Acute anterior uveitis can be related to human leukocyte antigen–B27 positivity and its associated conditions, including ankylosing spondylitis, psoriatic arthritis, Reiter syndrome, and inflammatory bowel disease (eg, Crohn disease and ulcerative colitis).[6] Other associations include Behçet syndrome, syphilis, and respiratory disorders such as sarcoidosis and tuberculosis, as well as viral causes such as herpes simplex and herpes zoster.[7] However, the differential diagnosis of granulomatous AAU is more specific and includes sarcoidosis, syphilis, tuberculosis, and toxoplasmosis. Less common causes of granulomatous AAU include Vogt-Koyanagi syndrome, sympathetic ophthalmia, multiple sclerosis, Lyme disease, coccidioidomycosis, leprosy, and brucellosis. Granulomatous AAU can also be idiopathic or be caused by glaucoma medications such as bimatoprost and latanoprost.[8]

Diagnosis

The most common signs and symptoms of AAU include rapid onset of pain, redness, photophobia, and blurred vision. Other signs include lid swelling; apparent narrowing of palpebral fissure due to splinting of the lids from photophobia; conjunctival, episcleral, and scleral edema; vascular enlargement; and tenderness to touch.[7,9,10] The pupil might be miotic owing to iris spasm and irregular owing to posterior synechiae (ie, adhesions between the iris and lens capsule). Central to diagnosis is the notation of cell and flare within the aqueous humour of the anterior chamber, both markers of ocular inflammation, on slit lamp examination. This examination is done by focusing a short, thin beam of high-intensity light on the anterior chamber from an angle of approximately 45° with high magnification in a dark room. Using the black pupil as a background, the examiner looks for either cells (which appear as bright mobile specks) or flare (ie, the light beam appears gray against the black pupil) within the anterior chamber. Flare is caused by extravasation of proteins into the aqueous fluid as a result of the inflammatory process. Hypopyon, a collection of anterior chamber cells layered inferiorly within the anterior chamber, is seen in severe cases.[4,5] The inflammation might spread to the vitreous membrane, and cystoid macular edema is sometimes observed,[7] which can cause a substantial reduction in vision. As in the image presented (**Figure 1**), mutton-fat keratic precipitates might be seen with granulomatous inflammation. Mutton-fat keratic precipitates are large, greasy-white aggregates of cells on the endothelium of the cornea, which represent clusters of macrophages and epithelioid cells.[4,5]

Although AAU often resolves without vision loss with appropriate treatment, permanent visual deficits can result from delayed detection, delayed treatment, or inadequate control of inflammation. The macula, optic nerve, and aqueous drainage system can incur serious damage and should be thoroughly examined.[11,12] Fluorescein angiography or optical coherence tomography can be used to assess the posterior pole and aid in diagnosis.[6]

Management

A thorough history and ocular examination are the mainstays of diagnosis. The history should cover the duration and degree of symptoms, any previous episodes, and the possible presence of associated underlying systemic disorders. It is also essential to rule out mimicking conditions, such as keratitis, secondary spread from posterior uveitis, episcleritis or scleritis, acute glaucoma, problems associated with contact-lens usage, recent ocular surgery, and chronic anterior uveitis.[7] Masquerade syndromes, including primary intraocular lymphoma, leukemia, uveal melanoma,

Figure 1. Keratic precipitates on the endothelium of the cornea in a patient with acute granulomatous uveitis; mild perilimbal injection is also observed

retinoblastoma, metastatic lesions, and paraneoplastic syndromes, must also be considered. The presence of associated underlying conditions should be investigated and other health care professionals involved, depending on the diagnosis.

If AAU is suspected, prompt referral should be made to an ophthalmologist for assessment and initiation of treatment. Treatment is aimed at inflammation control, prevention of complications, and symptom relief. Topical corticosteroids should be used aggressively to quickly bring inflammation under control and to soften and break any posterior synechiae. A loading dose of prednisolone is typically provided, followed by hourly instillation for the first few days. Instillation must be continued during the night with a cortisone ointment.[7] Steroid treatment should last 6 to 8 weeks depending on clinical response,[7] ending with a slow, tapered reduction. As side effects include raised intraocular pressure, treatment should be administered and monitored by an experienced ophthalmologist. Nonsteroidal anti-inflammatory medications are not as effective as steroids in the treatment of AAU[13]; if inflammation is uncontrolled by topical corticosteroids, periocular or systemic corticosteroids should be considered.

Prevention of posterior synechiae and relief from pain and photophobia can be provided with cycloplegics and mydriatics, such as atropine or homatropine, instilled topically 2 to 4 times daily.[7,14] Some anticholinergics, such as tropicamide, might also be considered to prevent the formation of posterior synechiae, but others, such as cyclopentolate, are never used owing to possible inflammatory effects.[14] If posterior synechiae exist at presentation, aggressive dilation with 10% phenylephrine or topical cocaine eye drops should be instituted to prevent permanent synechiae formation. Initially, daily follow-up is necessary. Symptomatic improvement of pain and photophobia is expected on the first follow-up visit, and signs of inflammation should be stable. At subsequent visits there should be progressive reduction in signs and symptoms, and the intervals between visits can lengthen over time. However, the patient must remain under observation until the inflammation is completely resolved.[7,14]

Conclusion

Acute anterior uveitis is a common inflammatory condition of the eye. Careful history and physical examination should rule out mimicking conditions and distinguish AAU from other forms of uveitis. Corticosteroids and cycloplegics are the mainstay of treatment and should be administered by an experienced specialist. Frequent follow-up is necessary. The presence of associated systemic disorders should be investigated and treated.

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Competing interests
None declared

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