A woman with an aching, red eye

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With a careful history and simple examination, a family doctor can diagnose this painful blinding condition and initiate early treatment and referral.

Case presentation

A 38-year-old receptionist presented to her GP during spring with a three-day history of an aching right eye with mild redness. She said the ache radiated to her brow and that at times she felt nauseated. The eye felt bruised and the pain was worse with reading. She wore contact lenses on weekends only, and she thought her glasses were causing glare when she was working on a computer or driving at night.

On questioning, the patient reported that the eye had been intermittently red over the previous winter since a flu infection but had not been painful previously. She had a history of oral cold sores each summer, and the previous spring she had been excluded from contact lens wear by her optometrist because of a herpes dendrite on her right cornea.

Comment

Latent herpes simplex infection (HSV) is almost ubiquitous in adults. By the age of 5 years, 60% of children have been infected with HSV1, but only 6% will have had clinically apparent primary disease and only 1% will present with a primary ocular infection by this age. The vast majority of cases are oral, which gives the virus access to the trigeminal ganglion and leads to ocular reactivation by retrograde axonal transmission. Triggers for reactivation are thought to include fever, UV light, systemic illness, minor local trauma, immunosuppression, emotional stress and menstruation.[1]

A history of contact lens wear and red eye would trigger suspicion of a corneal infection. This patient's history of herpes dendritic keratitis is of greater concern because affected corneas remain hypoaesthetic and may not alert the patient to retained foreign bodies or infections until the inflammation involves the uveal tract – this is uveitis.

In uveitis involving the anterior chamber, trabecular meshwork and iris (anterior uveitis), patients typically describe a bruised sensation to the globe, and immediate pain with bright light miosis (photophobia). Pain is also induced by activities that require near accommodation with its neurologically linked miosis, such as reading on a computer.

Glare, however, is not a prominent complaint in anterior uveitis. The glare described by this patient would direct the examiner to the ocular media, such as the cornea, lens and vitreous. The ache radiating to her brow is also not consistent with this diagnosis, and should lead to consideration of more distant structures in the orbit or sinuses or the possibility of high intraocular pressure, in which globe distension can induce nausea.

Examination

On examination, the patient's eye was mildly red, mainly at the junction of the cornea and sclera (limbus). Vision with her glasses was 6/18 (right) and 6/5 (left).

The ophthalmoscope induced some photophobia, but not markedly. Distance illumination and viewing through the ophthalmoscope for the red reflex showed a mildly dimmer reflex on the right but also anisocoria, with the right pupil being larger than the left in the lighted room (Figure 1). When the room lights were switched off, the anisocoria reversed (i.e. the left pupil became larger than the right) and the right iris showed transillumination (Figure 2).

Closer inspection with the ophthalmoscope used as a torch showed surface derangement of the right iris (iris stromal atrophy) in the area of transillumination. Pigmented clumps were observed on the back surface of the cornea (keratic precipitates) and the thickness of the cornea had patches of hazy infiltration (Figure 3). The corneal surface had lost its lustre and

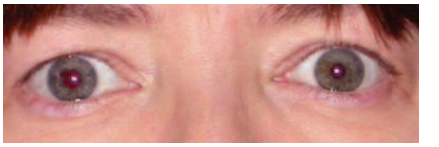

Figure 1. Red reflex and anisocoria.

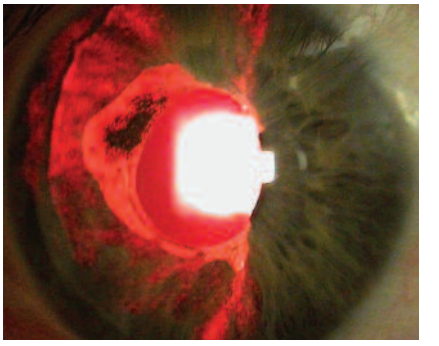

Figure 2. Transillumination of the right iris.

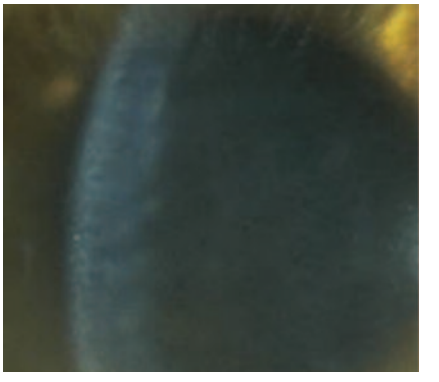

Figure 3. Interstitial corneal inflammation. The slitlamp beam is highlighted by haze and cellular infiltrate as it passes through the corneal stroma. Its concave edge is irregular, showing folding of the corneal endothelium caused by stromal swelling.

Dr Dunlop runs a uveitis clinic at: The John Hunter Hospital, Newcastle; The Eye Associates, Sydney; and The Eye Institute, Chatswood, Sydney, NSW.

appeared steamy. There was no focal or dendritic staining, but the right cornea had a diffuse uptake of fluorescein compared with the left. On double-digital (two index finger) palpation of the globes, the right felt harder (like a golf ball), whereas the left had more give (like a squash ball).

Comment

The final finding would alert the physician to a raised intraocular pressure, which explains the patient's brow ache and nausea. The diffuse fluorescein uptake is due to corneal epithelial oedema induced by the high intraocular pressure, where the imbibing pressure overcomes the capacity of the corneal endothelial pump to maintain deturgescence. The lack of focal staining shows that there is no epithelial infection or foreign body.

Corneal herpetic disease is manifold, and recurrences often involve only the stroma without any epithelial changes (so called disciform keratitis or interstitial keratitis). The hazy infiltration within the thickness of the cornea is the cause of this patient's diminished visual acuity and glare, as well as the reduced brightness of the right red reflex.

This patient's disciform keratitis has a concurrent anterior uveitis characterised by limbal erythema (ciliary injection), which highlights that the underlying ciliary body is inflamed. Just anterior to the ciliary body is the trabecular meshwork, the structure by which 90% of aqueous leaves the eye. Viral infiltration and inflammation of this meshwork (trabeculitis) is thought to be a significant factor in herpes uveitis presenting with a high intraocular pressure.

Herpetic eye disease is also a vasculitis that can cause iris ischaemia. This causes iris stromal atrophy, and loss of sphincter and dilator function – hence the patient's reversing anisocoria. More particularly, it causes the iris pigment epithelium to shed, leaving transillumination defects. The liberated pigment circulates in the aqueous and is deposited on the corneal endothelium and within the trabecular meshwork. This pigment debris combined with the cells, protein and fibrin of the uveitic exudate obstruct aqueous outflow at the trabecular meshwork and further elevate intraocular pressure.

Diagnosis

The patient was diagnosed with herpetic uveitic glaucoma.

Comment

The diagnosis in this case was clinical, with possibly only herpes zoster ophthalmicus having similar features (although in this case without preceding or current dermatomal cutaneous eruptions herpes zoster ophthalmicus was less likely). Aqueous PCR could resolve this differential, but treatment would remain the same. Other vasculitides, such as systemic lupus erythematosus and Behçet's disease, can cause

transillumination defects but not corneal disease. Past bacterial or chemical trauma may lead to corneal stromal scars, but not iris changes. Past mechanical trauma can cause corneal and iris changes but is unlikely to give recurrent inflammation.

Ocular herpes simplex infections will be recurrent in 63% of cases within 20 years' follow up;[2] of those that recur, 54% will present with high intraocular pressure.[3] The combination of high intraocular pressure and uveitis is nearly always associated with stromal disease reactivation (in 96% of cases), and never with dendritic keratitis or primary infection.[4] Iris atrophy is present in 41% of cases.[4]

Management

In this case, a phone call to the local ophthalmologist with an explanation of the history and examination findings enabled treatment to be commenced immediately and specialist review planned for the following day. Therapy was entirely topical: aciclovir ointment (five times daily), timolol 0.5% drops (twice daily), dexamethasone 0.1% drops (four times daily) and homatropine 2% drops (twice daily).

The patient was instructed not to wear contact lenses until topical therapy had been ceased, and a work certificate was issued to assist recovery without stress.

Comment

The aims of treatment for herpetic uveitic glaucoma are to prevent further virus replication (aciclovir) and to reduce inflammation (dexamethasone). Topical steroids should not be given without antiviral cover in a patient with a history of herpetic keratitis for fear of accelerated infection, and they may also induce raised intraocular pressure in susceptible individuals. It is important that ophthalmic review always be arranged to manage these two risks.

In herpetic uveitic glaucoma, raised intraocular pressure is managed with aqueous suppressants, such as topical beta blockers, alpha agonists and carbonic anhydrase inhibitors. Prostaglandin analogues are avoided because of the possible induction of inflammation.

Despite herpetic and uveitis disease clinical quiescence, high intraocular pressure may last up to eight weeks, and 10% of patients will develop glaucomatous field loss if adequate control of the pressure is not achieved.[4] As with all inflammatory disorders, the earlier the diagnosis and instigation of therapy, the more readily control of disease will be achieved and the better the prognosis. If recurrences are frequent, studies have shown that oral aciclovir, 400 mg twice daily, will reduce this, but the frequency returns on ceasing therapy.[5] MT

A list of references is available on request to the editorial office.

DECLARATION OF INTEREST: None.

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