The varicella virus is a member of the Herpesviridae family that includes herpes simplex, Epstein-Barr, and cytomegalovirus. This article discusses manifestations of the virus, how glaucoma occurs in infected patients, and the options for treatment.

ABOUT THE VIRUS

Ninety-five percent of the population has serological evidence of prior varicella-zoster virus (VZV) infection, which, when first symptomatic, manifests as chickenpox. After primary viremia, VZV lies dormant in one or more neurosensory ganglia throughout the body. Reactivation of latent virus produces the characteristic manifestation of herpes zoster, commonly known as shingles. When the ophthalmic division of the fifth cranial nerve in the trigeminal ganglia is involved in a secondary latent infection, it is called herpes zoster ophthalmicus (HZO). Reactivation of the virus is associated with a decline in cell-mediated immunity, but poor nutrition, excessive fatigue, advanced age, and physical or emotional stress may play a role. The condition may also occur in otherwise healthy adults.1

The ophthalmic nerve further divides into nasociliary, lacrimal, and frontal branches, of which the last is most commonly involved by HZO. The virus spreads by secondary perineural and intraneural invasion of the sensory nerves. Most cases of HZO follow 24 to 72 hours of malaise, headache, and dysesthesia (tingling or pain in the scalp, forehead, or face on one side). In its most typical presentation, erythematous macules appear along a single dermatomal distribution that does not cross the midline in contradistinction to the total body rash of the primary chickenpox infection (Figure 1). The cutaneous infection progresses over several days to papules and vesicles and, later, to pustules that eventually rupture and crust over, requiring several weeks to heal. Because the virus particles can be transmitted through direct contact with secretions from vesicles, the disease is contagious until crusted pustules develop.

The nasociliary nerve innervates the tip of the nose, and lesions occurring in this area—called Hutchinson sign—are often followed by ocular inflammation and corneal denervation from the disease. A minority of patients shows no classic dermatomal rash and instead demonstrates only ophthalmic findings, usually limited to the cornea (Figure 2). In other cases, ocular manifestations can be extensive and include every ocular structure from the eyelid and conjunctiva to the sclera, cornea, retina, optic nerve, and extraocular muscles. Early or mild anterior uveitis may present with minimal symptoms, whereas more severe cases are heralded by blurred vision, intense photophobia, injection, and fibrinoid iritis. Granulomatous keratic precipitates, posterior synechiae, and hypopyon may form. Herpes zoster uveitis can occur independently of corneal disease.

Acutely elevated IOP occurs when keratitis and uveitis complicate herpes simplex and zoster. The incidence of secondary glaucoma ranges from 16% to 56%3,4; the number is difficult to determine, because it has largely been reported from uveitis clinics rather than from glaucoma practices. Glaucoma occurs by several different mechanisms, including outflow obstruction from inflammation of the trabecular meshwork, synechial scarring, and the long-term use of steroids. In a series of 34 uveitic patients with HZO, Thean et al found that 56% developed glaucoma.1 In this report, uveitis and glaucoma occurred early in the course of HZO, which in most cases was responsive to treatment with glaucoma medication and topical corticosteroids. Filtration surgery
of the trabecular meshwork. When IOP responds rapidly to the application of corticosteroids, one may conclude that trabeculitis plays a role in the glaucoma. Vigilant monitoring for corticosteroid-induced glaucoma is demanded when these patients require long-term treatment to control corneal or anterior segment inflammation. If maintenance of corticosteroid treatment is warranted, glaucoma treatment is escalated as needed to protect the optic nerve and avoid vision loss. If medical therapy fails to control the IOP, then filtration surgery or a tube shunt procedure is recommended. Ideally, inflammation should be controlled for as long as safely possible prior to glaucoma surgery to optimize a favorable outcome in these high-risk eyes. Perioperative corticosteroid therapy provided topically, orally, or as a sub-Tenon injection together with intraoperative mitomycin C and postoperative 5-flourouracil is advised.16-19

As with any form of uveitis, acute angle closure is possible from the formation of 360° posterior synechiae around the pupil or by relative pupillary block from fibrin obstructing the pupil. In more chronic scenarios, inflammatory peripheral anterior synechiae occlude trabecular outflow.

After the infection subsides, most patients suffer constant or intermittent discomfort or pain for a period of time in the distribution of their rash, called postherpetic neuralgia. Various systemic analgesics and antiinflammatory, antidepressant, and anticonvulsant medications may be used to address this complication, which may last for many months and be difficult to control.

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