Acute red eye with vesicular upper hemifacial rash in an older man

NATHAN WALKER BAppScOptom(Hons), MB BS(Hons) **ANDREW FIELD** MB BS, FRANZCO, FRACS

Early systemic treatment will be critical in reducing this patient's risk of blinding complications.

Case presentation

Mr L, a 68-year-old man, presented to his GP with a two-day history of a blistering right-sided upper facial rash and a right eye that was irritable and red. The rash had developed in the same distribution as tingling 'electric shock' pains he had first noticed five days earlier.

Apart from mild hypertension treated with lifestyle modifications, he had no general medical or ocular history of note, and he took no medications. When asked specifically, he recalled having chickenpox as a young child. He did not mention any recent opportunistic infections to suggest an immunocompromised state, but he reported the recent collapse of his family business as a source of considerable stress.

Examination

A vesicular rash was observed on the right upper eyelid and right side of his forehead and scalp (Figure 1). Vesicles were also noted extending down the right side of his nose to its tip.

Unaided vision was 6/18 (right) and 6/12 (left); this improved with spectacles to 6/12 and 6/6, respectively, but there was no further improvement with a pinhole. The right conjunctiva was diffusely injected. Examination with a magnifying loupe and cobalt blue light after instilling fluorescein eyedrops revealed numerous discrete areas of corneal and conjunctival epithelial staining that resembled small dendritic ulcers.

Diagnosis and treatment

The GP made a diagnosis of herpes zoster ophthalmicus, based on the characteristic distribution of the vesicular rash. No investigations were necessary.

Oral valaciclovir was prescribed, along with a paracetamol–codeine combination for progressively worsening pain. In consideration of the red eye and suboptimal visual acuity, the patient was referred to an ophthalmologist for a comprehensive ocular examination within three days.

Discussion

Herpes zoster is caused by the varicella-zoster virus. The primary infection usually occurs in childhood, after which the virus remains dormant in sensory root ganglia. During a period of waning immunity, and under the influence of largely unknown trigger factors, the virus may reactivate and migrate down sensory nerves, causing the characteristic skin and ocular lesions.

About 15% of cases involve the ophthalmic division of the trigeminal nerve – this is called herpes zoster ophthalmicus, regardless of whether the eye itself is involved. Involvement of the external nasal branch of the nasociliary nerve (Hutchinson's sign) correlates significantly with subsequent ocular complications because the nerve supplies the eye as well as the side and tip of the nose. The incidence of herpes zoster ophthalmicus increases with advancing age, particularly after 50 years; an immunocompromised state needs to be excluded in any patient under 40 years. Only 1% of cases occur in children under 12 years – such patients should be referred to a paediatrician.

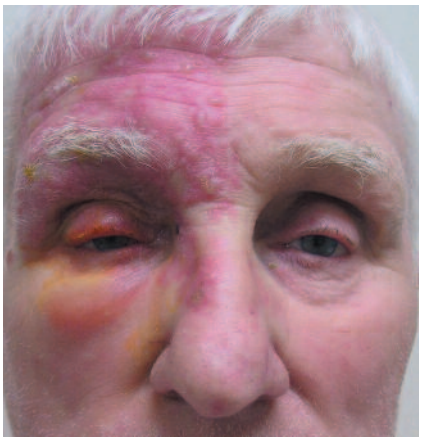

Figure 1. The vesicular rash. The lower lid is oedematous from contiguous spread of fluid but is not infected.

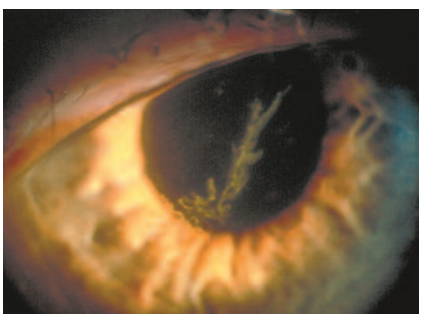

Figure 2. 'Pseudodendrites' in acute epithelial keratitis.

The disease

The acute phase of herpes zoster is generally an influenza-like illness lasting up to a

Dr Walker is Ophthalmology Senior House Officer, Royal Brisbane Hospital, Brisbane, Qld.
Dr Field is Visiting Medical Officer, Cairns Base Hospital, and an Ophthalmologist in private practice in Cairns, Qld. He has fellowship training in corneal and external eye disease from Johns Hopkins Hospital, USA.

week, followed by preherpetic neuralgia in the distribution of the affected nerve. The skin rash characteristically fits a unilateral dermatomal distribution, respecting the body's midline – it begins with macules that rapidly progress to vesicles and pustules and then to crusts and scars after a few days. Rarely, the rash may become generalised and the patient systemically unwell (such individuals are generally immunocompromised).

Herpes zoster ophthalmicus may affect almost any part of the eye, so prompt referral to an ophthalmologist is needed if the eye is red. Both direct viral invasion and the secondary immunological reaction are thought to contribute to damage because ocular involvement can develop weeks to months after the cutaneous eruption. Acute epithelial keratitis affects about 50% of patients and is characterised by 'pseudodendrites' – small, fine, dendritic or stellate lesions that stain with fluorescein (Figure 2). These lesions develop within two days of the rash onset and generally resolve spontaneously a few days later; they are subtly different from the dendritic ulcers seen in herpes simplex keratitis (zoster pseudodendrites have tapered ends whereas simplex dendrites have terminal bulbs).

Nummular keratitis (i.e. multiple coin-like corneal lesions) or disciform keratitis (i.e. a larger discrete disc-like region of central corneal oedema) may develop in the following weeks, affecting the corneal stroma or endothelium, respectively. These (and other less common forms of zoster keratitis) may become chronic and are thought to be due to an immune response to viral DNA imprinted on corneal cells. Treatment is complex, and such patients require the care of an ophthalmologist, who may commence topical steroid therapy.

Conjunctivitis is common in herpes zoster ophthalmicus. Inflammations of deeper layers (episcleritis, scleritis, and uveitis) may also occur but are less common. Retinitis, cranial nerve palsies (including complete external ophthalmoplegia), optic neuritis, encephalitis and even contralateral hemiplegia may occur, but fortunately are rare. Glaucoma may complicate zoster uveitis. Abnormal pupil reactions (including light-near dissociation) may also occur.

The differential diagnosis includes herpes simplex virus disease, which generally occurs in younger patients and does not follow a dermatomal distribution or obey the midline. Herpes zoster can be a presenting complaint of AIDS, so some consideration of a patient's risk factors for HIV infection should be given, particularly if the zoster recurs.

In atypical cases, a herpes swab and slide can be sent for immunofluorescence, PCR, and viral culture to aid diagnosis; of these, the PCR result is the most useful. A hypodermic needle is used to unroof a vesicle or pustule and the swab rubbed on the base of the lesion to obtain the specimen.

Management

Oral antiviral agents that are effective in treating herpes zoster ophthalmicus are:

- famciclovir (Famvir), 250 mg three times daily for seven days
- valaciclovir (Valtrex), 1000 mg three times daily for seven days
- aciclovir, 800 mg five times daily for seven days.

These agents have been shown to decrease pain, keratitis, uveitis and the severity of skin lesions if commenced within 72 hours of rash onset, so early systemic treatment is critical in reducing the risk of complications causing blindness. Valaciclovir produces faster pain relief than aciclovir in adults over 50 years of age and has a simpler dosing regimen. In severe cases, particularly those with systemic involvement or if sight is threatened (e.g. retinitis), hospital admission for intravenous aciclovir is often required.

Cold saline packs may provide some relief from the pain of the blistering skin rash. Secondary bacterial infection of the skin lesions may occur and should be treated as for impetigo or cellulitis. If ocular involvement persists after completing the systemic antiviral course, aciclovir ointment (Zovirax Ophthalmic Ointment) can be used, five times a day until resolution.

Postherpetic neuralgia can be chronic, severe, debilitating, and depressing. The incidence is about 10%, with age over 55 years and ophthalmic distribution identified as risk factors. Agents that may be trialled include amitriptyline (Endep, Tryptanol), carbamazepine (Tegretol, Teril), phenytoin (Dilantin), or capsaicin cream (Zostrix), but postherpetic neuralgia is often refractory to treatment.

Varicella-zoster virus is highly contagious from four days before rash onset and until all lesions have formed scabs (about one week). Spread is by droplets, so infected patients must be advised to avoid the elderly, children, pregnant women and any immunocompromised individuals.

The varicella-zoster vaccine (Varilrix, Varivax Refrigerated) is now on the Australian Standard Vaccination Schedule. This live attenuated vaccine affords protection from primary infection in 70 to 90% of recipients, but the duration of immunity is not known. In the absence of long term studies, current evidence suggests vaccination will also reduce herpes zoster risk.

Follow up

Mr L was reviewed a week later by his ophthalmologist. The pseudodendrites had resolved and the rash had formed scabs. He was referred back to his GP for pain management and advised to return promptly for reassessment if his vision subsequently deteriorated or he suspected the onset of a recurrence. MT

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

DECLARATION OF INTEREST: None.

Medicine Today 2004; 5(8): 79-80

Acute red eye with vesicular upper hemifacial rash in an older man

NATHAN WALKER BAppScOptom(Hons), MB BS(Hons) **ANDREW FIELD** MB BS, FRANZCO, FRACS

Further Reading

1. NHMRC. The Australian immunisation handbook. 8th ed. Canberra: National Capital Printers; 2003.
2. Therapeutic guidelines: antibiotic. Version 12. Melbourne: Therapeutic Guidelines; 2003.
3. Rhee DJ, Pyfer MF, eds. The Wills eye manual: office and emergency room diagnosis and treatment of eye disease. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 1999.
4. Kanski JJ. Clinical ophthalmology: a systematic approach. 5th ed. London: Butterworth-Heinemann; 2003.
5. Opstelten W, Mauritz JW, de Wit NJ, van Wijck AJ, Stalman WA, van Essen GA. Herpes zoster and postherpetic neuralgia: incidence and risk indicators using a general practice research database. Fam Pract 2002; 19: 471-475.
6. Ormrod D, Goa K. Valaciclovir: a review of its use in the management of herpes zoster. Drugs 2000; 59: 1317-1340.
7. Zaal MJ, Volker-Dieben HJ, D'Amaro J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. Graefes Arch Clin Exp Ophthalmol 2003; 241: 187-191.