ORBITAL DISEASE IN HERPES ZOSTER OPHTHALMICUS

STEPHEN J. VARDY and GEOFFREY E. ROSE
London

SUMMARY

Three cases of orbital inflammatory disease caused by herpes zoster are described. This extremely rare complication occurred between 5 days and 14 days following the skin eruption and slowly resolved with or without treatment. Biopsy of a chronic inflammatory lesion on the cheek of one patient demonstrated a sterile vasculitis and granulomatous liponecrosis, a process which may underlie the orbital disease in these patients.

The ophthalmic division of the fifth cranial nerve is affected in 7–17.5% of herpes zoster patients[1–5] and ophthalmic zoster may vary from severe disease, threatening life and sight, to disease so mild that it may pass unnoticed. Ocular involvement complicates approximately 50% of these cases and occasionally maxillary zoster can affect the globe. Ophthalmic involvement may result from inflammatory changes, nerve damage or secondary to tissue scarring; the inflammatory changes may be in the form of keratitis (dendritic, nummular or disciform) or a vasculitis, as in episcleritis, scleritis, iritis, ischaemic papillitis and optic neuritis. Orbital vasculitis is presumed to occur but is apparently unrecorded. Neural damage (namely neuroparalytic keratitis, ocular motor nerve palsies or neuralgia) may be secondary to a vasculitis affecting the vasa nervorum. Changes subsequent to tissue scarring may include eyelid deformity, neuralgia or lipid keratopathy.

Orbital involvement in ophthalmic zoster is extremely rare but might be expected to include extensive vasculitis, haemorrhage, perineuritis and inflammatory cell infiltrate affecting all orbital contents including the extraocular muscles and the optic nerve.[6] We report three cases of orbital inflammatory disease due to herpes zoster and propose that inflammation may result from ischaemic liponecrosis secondary to a vasculitis.

CASE REPORTS

Case 1

A 70-year-old male developed herpes zoster affecting the ophthalmic and maxillary divisions of the right trigeminal nerve and later became comatose due to central nervous system disease, requiring ventilation for 7 days. Whilst being ventilated he developed orbital cellulitis and septicaemia, the septicaemia being associated with acute renal failure. The orbital abscess was drained via a superomedial orbitotomy and *Streptococcus pyogenes* isolated from the fluid contents. His condition improved slowly on appropriate antivirals (acyclovir, intravenously and topically), antibiotics and anti-inflammatory agents.

Three months after the onset of zoster he was referred to Moorfields Eye Hospital because of persistent orbital inflammation and a palpable tender mass in the right cheek with swelling extending up to the medial canthus (Fig. 1). Visual function was normal and there were no signs of intraocular disease. The right globe was displaced medially by 3–4 mm and adduction was slightly restricted. The right upper eyelid was slightly red, tender and a mild ptosis was present with normal levator function; there was a tightly bowed dermal and subdermal scar at the site of previous orbitotomy (Fig. 1). The lower eyelid was drawn into an ectropion by a firm mass 2 cm in diameter in the dermal and subdermal tissues of the right cheek (Fig. 2). Light touch sensation in the maxillary division of the right trigeminal nerve was slightly reduced but there was no evidence of palatal or gingival disease.

Computed tomography showed a mild diffuse swelling overlying the right zygoma and in the region of the right upper and lower eyelids; within the prezygomatic tissues there was an area of radiolucency with an enhancing rim. The radiolucency was suggestive of a fat loculus or air in the subdermal tissues (Fig. 3).

Biopsy of the cheek nodule revealed a fibrous capsule surrounding a collection of clear, straw-coloured oily fluid which failed to cultivate any organisms. Histological examination of the capsule demonstrated areas of fat necrosis and foreign body granulomatous reaction (Fig. 4) with a conspicuous vasculitis but no obvious perineuritis (Figs. 4, 5).

The patient made a complete systemic recovery, but the right lower lid ectropion and the bowing at the medial can-

Correspondence to: Mr G. E. Rose, MS, FRCS, FRCOphth, Orbital and Adnexal Service, Moorfields Eye Hospital, City Road, London EC1V 2PD, UK.

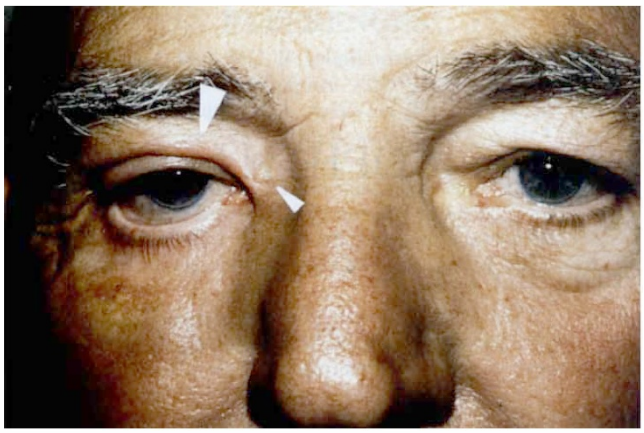

Fig. 1. *Case 1. The upper right eyelid is slightly red (large arrowhead) and there is mild ptosis with a tightly bowed dermal and subdermal scar (small arrowhead) at the medial canthal angle. The right lower eyelid is drawn into an ectropion.*

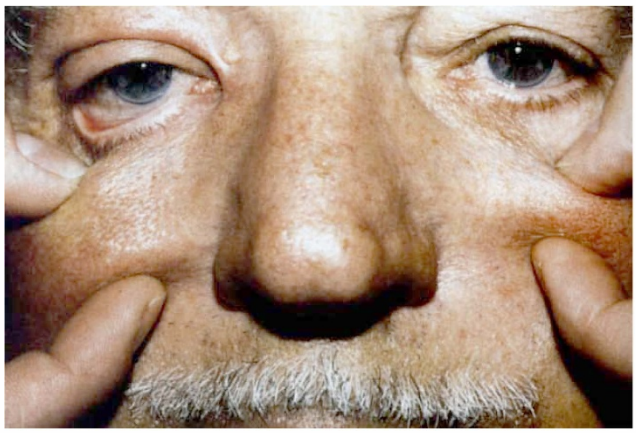

Fig. 2. *Case 1. There is a firm mass in the dermal and subdermal tissues of the right cheek.*

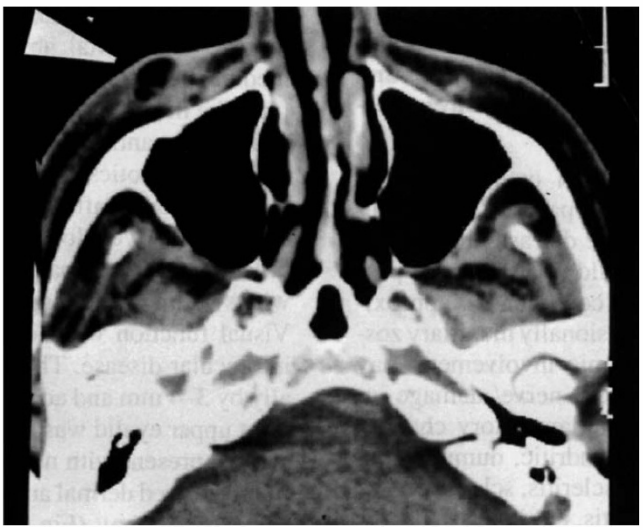

Fig. 3. *Case 1. CT scan of the diffuse swelling over the right zygoma in the region of the lower eyelid (large arrow). The radiolucency is suggestive of a fat loculus or air.*

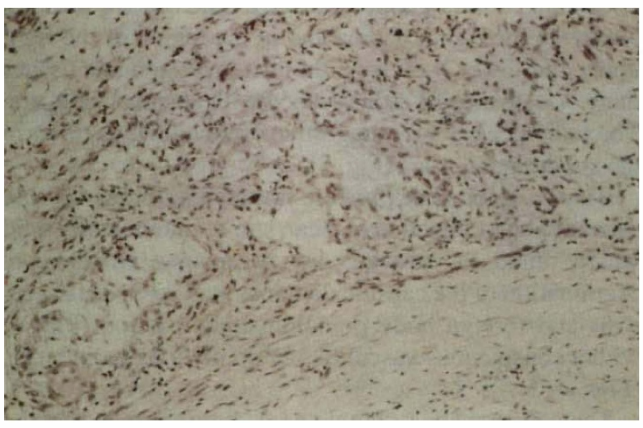

Fig. 4. *Case 1. Histopathology of the cheek nodule wall, with foreign body granulomatous reaction and areas of fat necrosis.*

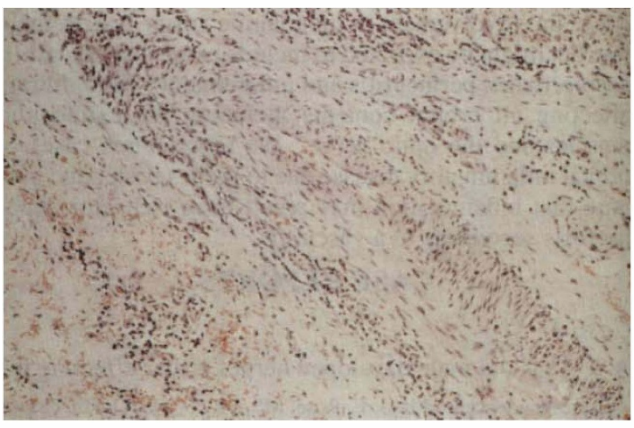

Fig. 5. *Case 1. Histopathology of the cheek nodule wall with conspicuous vasculitis but no peri-neuritis.*

thus persisted, as did a mild asymptomatic medial displacement of the globe.

Other investigations, such as full blood count, erythrocyte sedimentation rate, biochemistry, serum autoantibody screen, plasma protein electrophoresis and serum ANCA, were normal.

Case 2

A 63-year-old man presented with severe herpes zoster ophthalmicus exhibiting acute vesicular skin lesions, swollen lids, chemosis and dendritiform kerato-uveitis on the left side. He was treated with topical steroids and systemic antibiotics, no antiviral agents being available. One week later the skin lesions had improved but he developed a 4 mm left relative proptosis and a total left oculomotor nerve palsy. Orbital venography revealed good flow through to an unobstructed cavernous sinus and the proptosis was presumed to be due to orbital oedema and vasculitis; no further measure to reduce intraorbital pressure or compressive effects was necessary.

Because of persistent disease (with proptosis, an unresolved third nerve palsy and reduced vision), he was commenced on systemic steroids 2 weeks later. The proptosis and oculomotor palsy resolved but the patient required long-term therapy for recurrent keratitis, uveitic glaucoma and post-herpetic neuralgia; there was no recurrence of orbital disease over 10 years of follow-up.

Case 3

A 77-year-old man presented with severe right-sided herpes zoster ophthalmicus that showed typical skin lesions, reduced vision from corneal epitheliopathy, multiple ipsilateral cranial nerve palsies (IV, V and VI) and 4 mm right relative axial proptosis.

The orbital disease recovered untreated over the following 2–3 weeks but the patient went on to suffer long-term corneal disease and secondary uveitic glaucoma. The proptosis was considered to be due to orbital inflammation and oedema.

DISCUSSION

Ocular involvement by herpes zoster affecting the ophthalmic division of the trigeminal nerve complicates approximately 50% of cases and, more rarely, affects cases of maxillary herpes zoster; all structures of the globe and orbit may be affected.[1–5]

The natural history of herpes zoster ophthalmicus typically occurs as three phrases: acute, chronic and relapsing. Recurrences are peculiarly common in zoster and evidence exists to support a reduction in the severe and late chronic complications by early treatment.

Ocular complications may be categorised according to the type of tissue damage:

Inflammatory changes are usually in the form of dendritic, nummular and disciform keratitis with uveitis but less commonly as vasculitic lesions in episcleritis or scleritis, ischaemic papillitis and orbital disease.

Nerve damage in the form of cranial nerve palsies or post-herpetic neuralgia.

Scarring with induced eyelid deformity, lipid keratopathy or neuralgia.

A recent review[6] made comparisons of the incidence of ophthalmic complications of zoster in different series;[7–9] no cases of orbital complication were cited from a total of 1550 patients.

We report three patients, all elderly males, who experienced significant proptosis during the acute phase of herpes zoster ophthalmicus and in whom signs of orbital inflammation were present. Radiological studies in one case (case 1) were compatible with orbital and pre-septal inflammatory changes and the formation of a subdermal lipid-filled cavity in the premaxillary tissues on the affected side. There was no objective evidence of optic nerve involvement in any of the three cases, the reduced vision in one case (case 2) probably being due to intraocular inflammation. Two patients experienced resolution of proptosis within 3 weeks, without surgical intervention, whereas one patient (case 2) was treated with systemic steroids and antibiotics during the acute phase.

There is a clear history of orbital involvement, with drainage of an orbital abscess and *Streptococcus pyogenes* isolation, in one patient (case 1). It is likely that the abscess arose during streptococcal septicaemia by haematogenous spread into an area of prior orbital inflammation; orbital vasculitis would predispose to this complication.

The cheek nodule biopsy resembles a 'rheumatoid vasculitic nodule' and this suggests that orbital inflammation or abscesses may be due to sterile ischaemic necrosis of fat as a result of zoster vasculitis; it is likely that such sterile abscesses are at risk of secondary infection by blood-borne bacteria during septicaemia.

Key words: Herpes zoster ophthalmicus, Lipogranuloma, Orbital inflammation.

REFERENCES

1. Burgoon CF, Burgoon JS, Baldridge GD. The natural history of herpes zoster ophthalmicus. JAMA 1957;75:193–6.
2. Edgerton AE. Herpes zoster ophthalmicus. Arch Ophthalmol 1945;93:40–114.
3. de Moragas JM, Kierland RR. The outcome of patients with herpes zoster ophthalmicus. Arch Dermatol 1957;75:193–6.
4. Worster-Drought C. Herpes zoster with localised muscular paralysis. BMJ 1923;1:970.
5. Glyn C, Crockford, Gavaghan D. Epidemiology of shingles. J R Soc Med 1990;83:617–9.
6. Marsh RJ, Cooper M. Ophthalmic herpes zoster. Eye 1993;7:350–70.
7. Womack LW, Liesegang TJ. Complications of herpes zoster ophthalmicus. Arch Ophthalmol 1983;101:42–5.
8. Harding SP, Lipton JR, Wells JCD. Natural history of herpes zoster ophthalmicus: predictors of post-herpetic neuralgia and ocular involvement. Br J Ophthalmol 1987;71:353–8.
9. Scheie HG. Herpes zoster ophthalmicus. Trans Ophthalmol Soc UK 1970;90:899–937.