CASE REPORT

Trigeminal herpes zoster: early recognition and treatment are crucial

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SUMMARY

Reactivation of varicella zoster virus (VZV) is not uncommon in older patients, particularly in cases of chronic autoimmune disorders and in patients taking immunosuppressant drugs. We present a case of a 57-year-old woman presenting with severe herpes zoster infection, involving the maxillary and ophthalmic branches of the trigeminal nerve. Despite an initial delay in instigating crucial antiviral treatment, the patient achieved an excellent recovery, with only some mild scarring at 2 months postinfection. Trigeminal herpes zoster is a potentially devastating clinical occurrence, and is associated with severe long-term neurological sequelae, including encephalitis, vision loss and postherpetic neuralgia. Physicians must be aware of risk factors and treatment modalities.

BACKGROUND

This patient unfortunately succumbed to a particularly severe clinical case of herpes zoster. The absence of prompt diagnosis and management necessitated attendance and admittance in the emergency department (ED), which was possibly an avoidable event. This case highlights not only the cutaneous appearance of the herpes zoster ([figure 1](#)), but also serves as an important clinical lesson for physicians in the recognition and management of this relatively common disease.

CASE PRESENTATION

A 57-year-old woman presented to the ED with a 4-day history of a severe facial skin eruption, associated with swelling and pain. Her general practitioner had prescribed first oral flucloxacillin for presumed facial cellulitis, and when her condition failed to improve, oral co-amoxiclav. She suffered from rheumatoid arthritis (RA), for which she took methotrexate, hydroxychloroquine and adalimumab. Her RA was well controlled on this triple therapy. She was otherwise fit and well, and worked at a primary school. She did not smoke or excessively drink alcohol.

Examination of the patient revealed severe swelling of the left face and neck, with involvement of the left eyelid ([figure 1](#)). There were crusted, weeping vesicles over the left maxilla, which extended to the nose tip. There were also two lesions above the left eyebrow.

The underlying skin was erythematous, warm and clinically cellullitic. The left eye was partially closed due to peri-orbital swelling. The left conjunctiva was injected, although there were no lesions on the cornea. Pupillary responses and eye

movements were normal bilaterally, and there was no proptosis. Visual field testing was normal, and although the patient reported some subjective clouding of vision in the left eye, visual acuity was normal. Apart from hyper-aesthesia of the maxillary branch of the trigeminal nerve, all other cranial nerve testing was normal.

Neck examination revealed palpable lymphadenopathy of the anterior cervical chain. The patient was afebrile, although she reported documented fevers of up to 38°C at home prior to admission. There was no evidence of meningism or an encephalitic process. Cardiovascular, respiratory and abdominal examination was normal, and full exposure of the patient did not reveal any further skin lesions.

INVESTIGATIONS

Blood testing revealed a white cell count of $5.8 \times 10^9/L$, neutrophils $4.1 \times 10^9/L$ and lymphocytes $0.9 \times 10^9/L$. C reactive protein was mildly elevated at 19 mg/L. Electrolytes and renal function were normal. PCR testing of the patient's serum confirmed the presence of varicella zoster virus (VZV) DNA.

DIFFERENTIAL DIAGNOSIS

A clinical and microbiological diagnosis of herpes zoster involving the maxillary and ophthalmic

Figure 1 Hemifacial vesicular eruption affecting the left maxillary, nasal and periorbital regions.

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branches of the trigeminal nerve was made. The severity of the hemifacial subcutaneous swelling, cellitic appearance of the underlying skin and the extensive breach of the superficial dermis by the lesions, raised the clinical concern of potential superadded bacterial infection—particularly as the patient was taking immunosuppressant medication.

TREATMENT

Initial treatment was intravenous aciclovir. The maximum dose of 10 mg/kg was used due to concerns over the patient's immune status, and concerns regarding potential ophthalmic involvement (see the Discussion section). Concerns about the possibility of bacterial superinfection led to a decision to instigate treatment with oral clindamycin. Although this is a third-line agent for skin and soft tissue infection, the patient had failed to respond to flucloxacillin and co-amoxiclav therapy.

The patient also received antiseptic topical therapy and oral analgesics. She was advised not to return to her workplace (a primary school) until the lesions were crusted and no longer weeping.

OUTCOME AND FOLLOW-UP

The patient has made an excellent recovery, and aside from some scarring, the rash has cleared extremely well 2 months after the initial presentation. The rheumatology specialists decided to discontinue methotrexate and adalimumab therapy, and fortunately the patient's rheumatological symptoms have been quiescent.

Following discussion with the patient about the potential options for immunosuppressive therapy, the methotrexate was restarted. Considering the severity of the patient's neurocutaneous infection the adalimumab was not restarted. If she experienced a rheumatoid flare, this may have to be re-considered in the future. She remains under regular rheumatology review.

Fortunately, the patient reports no postherpetic neuralgia at 2 months after the event.

DISCUSSION

Primary infection with the VZV usually manifests as childhood chickenpox. After this, the virus becomes latent in cranial nerve and dorsal root ganglia, and the reactivation is suppressed by a competent immune system. Decline in VZV-specific cell-mediated immunity in elderly and immunocompromised individuals has been implicated in reactivation of VZV, manifesting as herpes zoster (shingles).

This patient had three independent risk factors for herpes zoster: age, RA and immunosuppressant therapy.¹ Reactivation of VZV has been well described in immunosuppressed patients.² Biological agents in particular have been associated with the reactivation of latent infectious processes, which may be viral or bacterial; tuberculosis reactivation remains a significant problem in immunomodulatory therapy.³ Although VZV vaccines exist, their routine use in immunosuppressed patients remains controversial—largely because they are high-potency live vaccines.⁴ There is currently no unified guidance regarding vaccination in high-risk patients.

In our case, it is clear that there is a degree of involvement of the ophthalmic division of the trigeminal nerve (herpes zoster ophthalmicus). Indeed, involvement of the ophthalmic division is fivefold more common than that of the maxillary division. The presence of vesicles on the tip of the nose was particularly worrisome. This is Hutchinson's sign, and signifies involvement

of the nasociliary branch of the ophthalmic nerve, which may herald potentially sight-threatening eye complications.⁵ For this reason, we sought ophthalmology advice in the acute stage; ophthalmologists were satisfied that there was no ocular involvement.

Chloramphenicol and topical steroid drops were prescribed as a precautionary measure.

As aforementioned, the patient was warned not to return to her work place until her lesions were healing. While Public Health England (PHE) advises that VZV is not a notifiable disease, there is a potential risk of VZV transmission to young children, causing chicken pox.

Trigeminal herpes zoster infection may lead to potentially severe conditions if unrecognised and untreated, such as encephalitis, postherpetic neuralgia, permanent vision loss and disfigurement.⁶ Some clinicians advocate the use of systemic steroids in order to reduce the incidence of long-term neurological sequelae, but there is currently no meta-analysis data to support this in the literature.⁷ Early diagnosis and treatment with high-dose intravenous antiviral agents is crucial. Clinicians should have a high index of suspicion for herpes zoster in immunosuppressed patients presenting with a skin eruption; empirical treatment with antibiotic therapy and antihistamines may cause a significant delay in antiviral medication, which in turn may lead to devastating and avoidable complications of herpes zoster.

Learning points

- ▶ Reactivation of varicella zoster virus (VZV) is a potential complication of both chronic autoimmune diseases and immunomodulatory treatment, especially with biological agents.
- ▶ Trigeminal VZV involving the ophthalmic nerve is a potentially severe condition and may lead to encephalitis, postherpetic neuralgia, scarring and disfigurement, and permanent vision loss.
- ▶ Clinicians should have a low threshold for starting antiviral treatment in immunosuppressed patients with facial swelling or rash.

Competing interests None.

Patient consent Obtained.

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