CASE REPORT

Herpes zoster on the face in the elderly

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SUMMARY

Herpes zoster is a localised disease caused by reactivation of the varicella zoster virus that enters the cutaneous nerve endings during an earlier episode of chicken pox, travels to the dorsal root ganglia, and remains in latent form. The condition is characterised by occurrence of multiple, painful, unilateral vesicles and ulceration, and shows a typical single dermatome innervated by single dorsal root or cranial sensory ganglion. Involvement of three or more dermatomes is known as disseminated zoster and seen in immunocompromised individuals. Complications of herpes zoster include ocular sequelae, bacterial superinfection of the lesions, meningoencephalitis and postherpetic neuralgia. The incidence of herpes zoster increases with age and immunosuppression, therefore prompt management is necessary to avoid morbidity and mortality in these individuals. We present two case reports of herpes zoster, one involving the maxillary and mandibular branches of the trigeminal nerve while the other involves all branches of the trigeminal nerve.

BACKGROUND

The 'varicella zoster virus' (VZV), which is distributed worldwide, is a neurodermotropic virus that remains dormant in the sensory ganglion and, on reactivation, causes herpes zoster. Reactivation of VZV may occur spontaneously or when host defences are compromised. Predisposing factors for VZV reactivation can be increased age, physical trauma (including dental procedures), psychological stress, malignancy, radiation therapy and immunocompromised states including transplant recipients, steroid therapy and HIV infection.¹

Herpes zoster (HZ) typically erupts within one or two adjacent dermatomes, with thoracic (50–60%), cervical (10–20%) and trigeminal (10–20%) being more commonly involved, while lumbar (5–10%) and sacral (5%) are other less commonly involved dermatomes. In immunocompetent patients, involvement of non-contiguous dermatomes is never seen, although overlapping of adjacent dermatomes can be seen in 20% of cases.²

The involvement of maxillary and mandibular branches without the involvement of the ophthalmic branch accounts for 1.7%³ to 2%⁴ of total cases of HZ, which is comparatively rare.³

This case series is being reported to highlight the rarity of involvement of more than one division of the trigeminal nerve added to their presentation in immunocompetent individuals.

CASE PRESENTATION**Case 1**

A 68-year-old man presented after 3 days of enduring painful blisters on the left side of his face and mouth, and fever for the past 5 days. He developed the fluid-filled blisters 2 days after the fever. The pain was severe, continuous and radiating in nature. The blisters were initially small and few in number; they later increased in number covering the entire left half of the face with a watery discharge. The patient was unable to eat food or maintain oral hygiene. No relevant medical, dental or family history was reported. Extraoral examination revealed numerous vesicles present on the left chin region, which abruptly stopped at the midline. There was also involvement of the preauricular, auricular and scalp of the temple region along with crusts. The surrounding skin was red in colour and very tender on palpation (figure 1).

Intraoral examination showed ruptured vesicles, which coalesced to form large ulcers that varied in size from 1 to 2 cm in diameter on the left half of the lower lip, buccal mucosa, lower labial mucosa, retromolar area, left half of soft palate, alveolar ridge and on the dorsal and ventral surfaces of the left half of the tongue (figure 2). The ulcers were irregular in shape and covered with pseudomembranous slough at the base, surrounded by an erythematous halo. The ulcers were tender on palpation.

On hard tissue examination, the patient was completely edentulous except for the grossly carious mandibular left third molar.

Figure 1 Case 1: vesicles involving the entire left side of the face.

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Figure 2 Case 1: intraoral ruptured vesicle involving the oral mucosa of the left side.

Case 2

A 50-year-old man presented with fever, blisters and pain on the left side of his face for the past 2 days. The pain was very severe and burned; it was continuous, radiating up to the forehead and ear, and had a tingling sensation. It got aggravated while eating, drinking and swallowing. One day earlier he had developed multiple vesicles and blisters on the left side of his face, which first appeared on the lower lip and continued to the chin, cheek and forehead; gradually it reached the eye and soon the ipsilateral pinna were also involved ([figure 3](#)). There were multiple vesicles that coalesced to form bullae on the left side of the chin. Skin surrounding the vesicles was erythematous. The hard palate showed 7–8 ulcers on the left side measuring approximately 2–3 mm in diameter, which were round in shape

Figure 3 Case 2: vesicles involving the left side of the face.

with an erythematous halo and covered with pseudomembranous slough ([figure 4](#)). Hard tissue examination revealed partially edentulous upper and lower arches with generalised attrition of teeth and poor oral hygiene.

INVESTIGATIONS

Exfoliative cytology from ruptured intraoral vesicles was inconclusive.

DIFFERENTIAL DIAGNOSIS

The unilateral distribution of painful, grouped vesicles in a dermatomal pattern suggested HZ. However, allergic stomatitis was ruled out owing to the history and the unilateral presentation of the lesion. Sycosis barbae was excluded by the presence of intraoral lesions and absence of follicular pustules.

TREATMENT

Both patients were treated with antiviral and supportive therapy for 1 week. Valacyclovir (1 g, in case 1) and acyclovir (800 mg, in case 2) were prescribed to control the active viral phase. Tramadol hydrochloride (100 mg) and acetaminophen (325 mg) were given to alleviate pain and fever. Topical application of acyclovir cream (5%) was advised for facial and intraoral lesions, three times a day. Lignocaine gel (2%) for local application before meals and a multivitamin preparation, as supportive therapy, were added.

OUTCOME AND FOLLOW-UP

Case 1 was reviewed after 1 week. There was regression in the number of extraoral and intraoral lesions with formation of scabs and hypopigmented areas; no fresh vesicles were found. The patient reported tinnitus and vertigo on this visit. On examination, Bell's sign was positive with loss of wrinkles on frowning on the left side of the forehead. Drooping of the mouth on

Figure 4 Case 2: ulcers involving the left hard and soft palate.

the unaffected side on attempting to smile and inability to blow air from the left cheek confirmed Bell's palsy. Thus HZ progressed to Ramsay-Hunt syndrome (figure 5).

The patient was advised to continue treatment. For the Bell's palsy, otorhinolaryngology and ophthalmology opinions were sought. Anti-inflammatory ear drops and methyl cellulose eye drops were prescribed for 1 week. A week later, the patient exhibited marked improvement of the extraoral as well as intraoral herpes lesions and the signs of Bell's palsy had ameliorated.

In case 2 marked improvement was seen with healing of the lesions and scab formation at the end of 1 week and complete regression was seen after 2 weeks (figure 6).

DISCUSSION

HZ is also known as shingles, which is derived from the Latin *cingulum*, meaning 'girdle'. This is because a common presentation of HZ involves a unilateral rash that can wrap around the waist or torso like a girdle. The name zoster is derived from classical Greek, referring to a belt-like binding (known as a zoster) used by warriors to secure armour.⁵

Von Bokayin, in 1988, hypothesised for the first time that chicken pox and HZ were caused by the same infectious agent.⁶ VZV is one of the eight known herpes viruses that infect humans. Its structure is characterised by an icosahedral nucleocapsid surrounded by a lipid envelope. Double-stranded DNA is located at its centre. The virus is approximately 150–200 nm in diameter and has a molecular weight of approximately 80 million.⁷

The incidence of HZ is up to 15 times higher in HIV-infected patients and it is found in 25% of patients with Hodgkin's lymphoma. The incidence of HZ infection in the general population has been reported to be 5.4%.¹ The infection tends to involve individuals above 45 years of age, with the highest incidence among persons aged between 68 and 90 years.¹ Both the

patients in our cases were elderly individuals of age 50 and 60 years, respectively.

Among the divisions of the trigeminal nerve, the ophthalmic division is most commonly involved followed by the maxillary and mandibular divisions.⁸ While case 1 reported with involvement of the maxillary and mandibular divisions, case 2 showed involvement of all three divisions of the trigeminal nerve, which is a rare finding.

HZ follows a prodromal, active and chronic stage.⁹ Classically it presents with a prodrome of mild-to-moderate burning or tingling (or in some cases numbness) in the skin of a given dermatome, often associated with fever, headache, general malaise and stomach upset.⁵ Similar findings were reported in both our cases, however, stomach upset was not reported.

In a duration of about 48–72 h from the prodrome, there is development of a unilateral erythematous, maculopapular rash along the dermatome, which eventually develops into a vesicular lesion, this represents the active stage.⁵

Facial and intraoral lesions occur unilaterally and are characteristic features of HZ. Case 2 showed involvement of all three branches of the trigeminal nerve with vesicular eruption over the upper eyelid, upper and lower lips, malar and temporal region and forehead, along with intraoral lesions. Case 1 showed involvement of the maxillary and mandibular divisions with unilateral lesions on the chin, upper and lower lips, malar area, temporal region including preauricular area and ear along with intraoral lesions on buccal mucosa, labial mucosa, tongue, alveolar ridge and soft palate.

The pain associated with shingles varies in intensity from mild to severe¹⁰ in such a way that even the slightest stimuli can elicit excruciating spasms. The pain in both cases was severe in intensity. Case 1 also reported pain in the ear due to vesicle formation suggesting the involvement of the vestibulocochlear nerve.¹

Figure 5 Case 1: Bell's sign and regression of the lesion post-treatment.

Figure 6 Case 2: regression of the lesion after treatment.

The lesions usually begin to dry and crustations appear after 3–5 days. Total duration of the disease is generally between 7 and 10 days; however, it may take several weeks for the hypopigmented skin to return to normal.⁵ Sometimes the vesicular rash does not appear (zoster sine herpete), which makes it difficult to diagnose.¹¹ Surprisingly, pain is reported to subside when the rash is most active; however, it reverts back until the rash clears¹² during the crusting and scale phase of the lesion. However, HZ is not as contagious as the primary varicella infection; persons with reactivated infections can transmit VZV to non-immune contacts. Household transmission rates have been noted to be approximately 15%.³

The chronic pain syndrome stage, termed postherpetic neuralgia (PHN), has been described as pain lasting 1–3 months after the skin lesions have cleared, but may last for years and decades.⁸ Grossly, 10–20% of individuals reported with PHN after the acute phase of HZ infection comprised of elderly patients in over 20% of cases.¹³ The pain is described as a brief recurrent shooting or shocking allodynia, with a constant, usually deep pain and is a significant cause of morbidity.

Secondary bacterial infection is another common complication of HZ. In recent years, group A β -haemolytic streptococcal infections including cellulitis and necrotising fasciitis increasingly complicate the course of varicella. Other complications such as scarring of skin, keratitis, retinal necrosis causing blindness, keratouveitis, cranial and peripheral nerve palsies, cerebral ataxia and pneumonia may lead to death.¹³ Periapical lesions,¹⁴ root resorption, tooth exfoliation¹⁵ and alveolar osteonecrosis have also been reported in association with HZ infection.

If the geniculate ganglion is involved, it may result in James Ramsay Hunt's syndrome, which includes facial paralysis and painful vesicular eruptions of the external auditory meatus and pinna of the ear.¹³ These findings were consistent with case 1, which showed eruption on the pinna, and tinnitus and vertigo on the first visit with clear features of Bell's palsy after 1 week. Case 2 did not show any signs of facial paralysis in spite of disseminated involvement.

Culture for VZV is challenging as it is labile and to attain an adequate sample from vesicular fluid is difficult. Other alternatives are direct immunofluorescence assay and PCR; direct immunofluorescence is considered to be more sensitive and cost effective, and offers a rapid turnaround time.¹³

Although it is a self-limiting disease, early institution of antiviral and symptomatic therapy reduces the morbidity significantly. Antiviral treatment should be instituted within 72 h of cutaneous eruption, because most virus replication ceases 72 h after onset of rash. Guanosine analogues such as acyclovir (800 mg, 5 times/day for 7–10 days), famciclovir (500 mg, 3 times/day for 7 days) and valacyclovir—(1000 mg, 3 times/day for 7 days) are effective antivirals. They are selectively monophosphorylated by the viral thymidine kinase and are further phosphorylated by cellular kinases and thus inhibit the viral DNA polymerase.²

Acyclovir (800 mg, 5 times/day for 7–10 days) shortens the duration of viral shedding, inhibits the formation of new lesions, hastens healing and reduces the severity of acute pain. Variable benefits are recorded with respect to reduction in the frequency and duration of PHN.¹⁶

Valacyclovir, a prodrug of acyclovir, produces a serum acyclovir level 3–5 times as high as that achieved with oral acyclovir therapy. The use of corticosteroids in the treatment of HZ has been controversial. Their benefits are overshadowed by fear of reduced immune response and hence they are contraindicated in immunocompromised patients. However, in combination with

acyclovir, they show moderate but significant acceleration in the rate of cutaneous healing and also alleviate acute pain. However, no study demonstrated any effect of corticosteroids on the incidence or duration of PHN. The use of corticosteroids for HZ without concomitant antiviral therapy is not recommended.¹⁶

Tricyclic antidepressants can also be used. Capsaicin is the only topical preparation approved for temporary relief of pain associated with HZ infections. It should not be applied until skin lesions have healed. Symptomatic treatment such as keeping cutaneous lesions clean and dry, use of sterile non-adherent dressing over skin to protect the lesions, topical applications (ie, Calamine lotion), sympathetic nerve blockades and analgesics (aspirin and others) may be used,¹³ as was advised in both our cases, and helped speed up recovery.

Newer medications for HZ are CMX 001 Hexadecyloxypropyl-cidofovir, Valamaciclovir Nucleoside analogue (H2G), ASP2151 Helicase primase inhibitor and FV100 Two bicyclic nucleoside analogues.¹

Learning points

- ▶ Herpes involving all three branches of the trigeminal nerve in immunocompetent individuals is a rarity.
- ▶ Early institution of systemic and local antiviral therapy may reduce the incidence of postherpetic neuralgia.
- ▶ Complications such as James Ramsay Hunt's syndrome should be kept in mind during management and needs immediate attention if it occurs.

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