

## Vague and Versatile - Herpes Zoster, 2 Case Reports and Review

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### Abstract

Varicella is a DNA virus, causes small pox and on reactivation causes herpes zoster involves older and immunocompromised patients. It causes severe painful, vesicles and ulcerations with unilateral involvement of dermatomes. Many patients come with complaint to plain burning without vesicles or erythema, so initial diagnosis is important to reduce fiery lesions to erupt and reduce chances of complications like post herpetic neuralgia, Ramsay hunt syndrome, post herpetic neuralgia, osteonecrosis to multiple other complications can arise due to herpes zoster. Here we reported 2 cases of herpes zoster of one ophthalmic and maxillary division and another with maxillary division with follow up and treatment.

**Keywords:** Herpes Zoster; Sensory Ganglia; Varicella; Trigeminal

### Abbreviations

HZ: Herpes Zoster; VZV: Varicella Zoster Virus

### Introduction

Herpes derived from Greek word "herperin" means creep. Varicella zoster virus is a Double stranded DNA virus, causing two lesions chicken pox firstly, then remain latent and later reactivated causing herpes zoster also known as shingles, incidence increases with increasing age and reduced immunosuppression, Herpes zoster may affect any sensory ganglia and its cutaneous nerve and involves any of the three branches of trigeminal nerve sometime with few prodromal symptoms so early the diagnosis better the compliance, as it has severe complications. This is a case report of 2 cases of Herpes zoster infection involving older patients.

### Case Report 1

A 70-year old female patient reported to the department of oral medicine and radiology with chief complaint of pain in upper right back tooth region since 10 days. History of malaise, weakness, burning sensation and tingling sensation over face 10 days back and later on painful multiple fluid filled vesicular lesions formed on the same region. Pain was sudden, continuous and pricking in nature, it radiates to lip, nose and lower eyelid. Blister were initially smaller and few in numbers but later on increased in size and numbers, ruptured and formed ulcers. Patient visited to dermatologist for skin lesions 7 days back and now skin lesions were almost healed.

On extraoral examination (Figure 1) showed healed brownish crusted lesions seen on right side of face involving from midline ala of nose, upper lip, angle of mouth and cheek surrounding skin is healed. On palpation surface is rough and slightly tender. On intraoral examination (Figure 2) showed multiple ruptured vesicles coalesce to form ulcer of size 2 to 3cms in size in right hard palate, with irregular border covered with pseudomembranous slough areas with 3 vesicles in center and adjacent, erythematous area extending from incisive papillae till soft palate without crossing midline, on palpation soft and tender. Hard tissue examination partially edentulous maxillary arch with generalized mobility present and root pieces seen.



*Figure 1a and 1b: Showing extra-oral lesions over right nose, upper lip and cheek.*

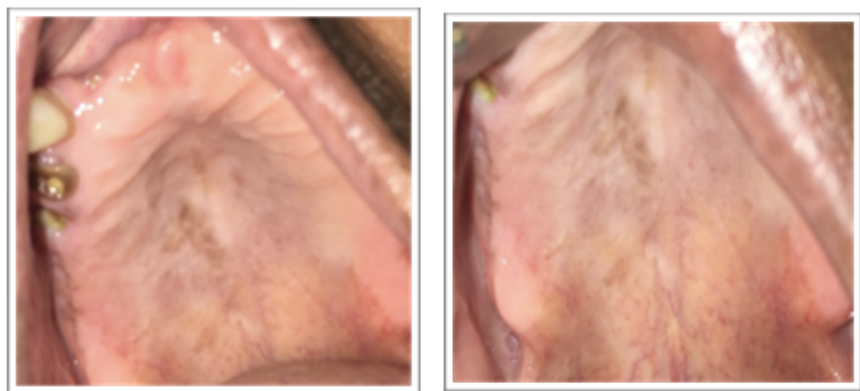


*Figure 2a and 2b: Showing unilateral erythematous areas with multiple ulcers.*

Exfoliative cytology was done which was sent for cytopathological examination revealed non conclusive report. All the hematological investigations were normal. Based upon history, clinical examination and investigations, a final diagnosis of Herpes Zoster virus infection of the right side of face involving the maxillary branch of the trigeminal nerve was made. After first follow up extractions of root pieces was done and second follow up recorded (Figure 3 and 4).



**Figure 3a and 3b:** Showing follow up extra oral lesions were healed.



**Figure 4:** 4a showing first follow up and later on extraction of root pieces were done and 4b show second follow up.

### Case Report 2

An 73 year old male patient reported to department of oral medicine and radiology with chief complaint of pain in upper right back tooth region since 3 days. History of fever, weakness, mild burning sensation over lip, temple, and cheek since 2 days. History of diabetes since 6 years and is under medication. Pain was sudden, mild, and sharp, intermittent type, pain radiates to right temple, eye, cheek and nose.

Extra-oral examination (Figure 5) showed multiple crops of blisters over right temple region, and on palpation surface is soft and tender. On intraoral examination there were no intraoral lesion.



**Figure 5a and 5b:** Showing multiple tiny crops of vesicles during first visit with no oral lesions.



**Figure 6a and 6b:** Showing extra oral lesions on second visit severe eruptions seen over right side of face.

After 5 days fiery lesions appeared and patient revisited with severe pain, burning and swelling involving whole of right side of face, involving eye, cheek, nose and upper lip, which was severe, pricking, continuous, radiating in nature. It aggravates on touching, eating, relieves on taking medication. On extra oral examination (Figure 6)- unilateral diffuse swelling and multiple crops of vesicular eruptions seen over right side of temple, eye, canthus, cheek, nose and upper lip with erythematous base covered with crusts, with irregular shape and swollen eye and watery discharge from vesicles and eye. On intraoral examination (Figure 7) multiple ruptured vesicles coalesce to form large ulcers of varying size from 1 - 2 cms lesion followed a serpiginous pattern of size extending from incisive papillae to soft palate from mid palatine raphae to attached gingiva involving buccal vestibule, buccal mucosa they are irregular in shape covered by pseudomembranous slough and surrounded by erythematous halo. Hard tissue examination- generalized attrition, occlusal caries with 17, restoration with 24, 26, metal capping with 25, 27.



**Figure 7:** Showing intraoral lesions on right side of palate without crossing midline with multiple ulcerations covered with slough.



**Figure 8a and 8b:** Showing healed extraoral lesions on right side of face.

Based on history, clinical, and cytopathological examination, provisional diagnosis is given as Herpes zoster of ophthalmic and maxillary division and both case 1 and case 2, patient was advised with proper diet with antiviral drugs (acyclovir (800 mg) and valacyclovir (1 gm), paracetamol (500 mg), prednisolone (5 mg and 10 mg), topical application of acyclovir cream for extra oral and intraoral lesions (herpex) 4 - 5 times/day and lignocaine gel before food was advised. Patient is recalled after 2 weeks for follow up lesions were almost healed no fresh vesicles seen and pain is almost relieved, advised for regular follow up (Figure 8 and 9).



**Figure 9:** Showing healed intraoral lesions.

## Discussion

The name zoster is derived from classical Greek word, referring to belt-like binding used by warriors to secure armour [1]. Varicella (chickenpox) and herpes zoster (shingles) are produced by the same virus- VZV. Varicella results from contact of a non-immune person with this virus, whereas herpes zoster occurs in persons who have had previous chicken pox. Herpes zoster is caused by reactivation of a latent infection in either a spinal or a cranial sensory ganglion. On reactivation, the virus spreads from the ganglion along the corresponding sensory nerve or nerves to the skin [2].

The ganglia most often involved are those of the lumbar and thoracic nerves. When the virus is reactivated, newly synthesized viruses are transported along the sensory nerve and released into the skin. Trigger mechanisms include trauma, stress, older age, malignancy, and other causes of immunosuppression including therapeutic agents. The incidence of herpes zoster increases with age. Although herpes zoster occurs largely in adults, particularly in those of advanced age, about 5% of patients with herpes zoster are children younger than 15 years of age [3].

The ophthalmic division of trigeminal nerve is most commonly affected, i.e. HZ ophthalmicus. Involvement of the trigeminal nerve leads to lesions on the upper eyelid, forehead, and scalp with V1; mid-face and upper lip with V2; and lower face and lower lips with V3. In our case, maxillary division of trigeminal nerve and ophthalmic and maxillary division was involved [4,10].

## Clinical features

Patient with HZ infections usually progress through three stages: (a) prodromal stage (b) active stage (also called acute stage) and (c) chronic stage [5,6]. The prodromal syndrome stage presents as sensation of burning, tingling, itching, boring, prickly or knife-like occurring in the skin over the nerve distribution as seen in our case. This usually precedes the rash of the active stage by a few hours to several days [5,6]. Infections occur unilaterally [1]. This infection characterized by the appearance of vesicles that occur on the skin along the pathway of an involved sensory nerve usually are associated with severe pain [4-6]. When branches of the trigeminal nerve are involved, lesions may appear on the face, in the mouth, in the eye, as seen in our case. Prodromal pain that occurs in the distribution of the trigeminal nerve several days before the vesicular eruptions, later papules develop, which rapidly become vesicles [7].

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 pigmented skin to return normal [5]. Sometimes the vesicular rash does not appear (zoster sine herpette), which makes it difficult to diagnose [8,9,11]. Surprisingly, pain is reported to subside when the rash is most active; however, it reverts back until the rash clears [8,12]. 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% [11].

## Complications

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. 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 [8].

Gonnet was the first in 1922 to describe alveolar bone necrosis and tooth loss in association with Herpes Zoster (HZ) infection [10]. Osteonecrosis following HZ infection often presents as painless exfoliation of teeth in the involved area. This occurs after the acute phase of the infection has subsided. The pathogenesis of the osteonecrosis is unclear although an alteration of the vascular supply to the affected bone has been postulated [10,11]. The most debilitating complication of HZ is pain associated with acute neuritis and post-herpetic neuralgia [6]. Rarely reported complications following oral involvement with herpes zoster are devitalised teeth, internal resorption and spontaneous exfoliation of the teeth with osteomyelitis of the alveolar bone [4,7].

## Treatment

A live attenuated vaccine derived from the oka strain of varicella zoster virus has been shown to be highly effective in preventing primary varicella in children and herpes zoster was introduced into the US vaccination programme in 1996. Treatment for herpes zoster infection includes Systemic antivirals- acyclovir, famciclovir, and valaciclovir, the nucleoside analogue brivudine has been shown to be as effective as famciclovir but superior to acyclovir in both healing acute lesions and reducing postherpetic neuralgia. Corticosteroids like prednisolone, tricyclic antidepressants- amitriptyline, nortriptyline, desipramine, opioids and NSAIDs- acetaminophen, aspirin or ibuprofen, opioids like oxycodone and tramadol, anticonvulsants- gabapentin, pregabalin, lidocaine patch 5%, vitamin C, and capsaicin, aloe Vera gel, calamine lotion and various other medications are under research for betterment [11-13,17,19].

## Acyclovir

Acyclovir, 9- [(2-hydroxy ethoxy) methyl] guanine is a guanosine analogue, was the first specific antiviral drug, to become widely used in the treatment of herpes simplex virus (HSV) and herpes zoster virus (HZV) infections [12-14].

## Pharmacokinetics

ACV is available as oral, intravenous and topical formulations. It is widely disseminated in the body fluids-cerebrospinal fluid, vesicle fluids, vaginal secretions and is excreted mainly through the kidneys, 85% in unmetabolized form. ACV crosses the placenta at all stages of pregnancy and is secreted into breast milk [14].

## Mechanism of action

ACV is a potent and selective inhibitor of herpes virus DNA replication. ACV monophosphate is converted into ACV triphosphate. ACV triphosphate competes with normal deoxy adenosine triphosphate and functions as a substrate for the enzyme viral DNA polymerase and produces:

- Complete and irreversible inhibition of herpes virus DNA polymerase.
- Viral DNA chain termination [15,16].

## Indications [14]

Herpes simplex infections (HSV 1 and 2) - Primary episodes- Recurrent episodes- Suppressive therapy

Varicella zoster infections- Chicken pox- Herpes zoster

Herpes simplex or varicella zoster infections in immunocompromised patients (HIV)

## Initiation of therapy

Treatment for episodic recurrent disease should begin during the prodromal period. Treatment for varicella should begin within 24 h of the onset of the rash. Treatment for herpes zoster in the immunocompetent host is of benefit only if started within three days of the onset of the rash [14].

## Indications of topical ACV include

Topical ACV ointment can shorten the period of pain and viral shedding in HSV mucocutaneous lesions in immunosuppressed patients, but not in patients with normal immunity. In contrast, ACV cream appears to reduce the duration of pain and viral shedding by one day in immunocompetent patients [17]. Herpes labialis- ACV 5% ointment is FDA approved for use in limited non- life threatening mucocutaneous HSV infection in immunocompromised patients [18,19].

## Adverse effects

- Obstructive nephropathy and interstitial nephritis
- Phlebitis and severe local inflammation
- Lethargy, tremors, confusion, hallucinations, extrapyramidal symptoms and seizures or coma.

- Skin rashes, recall dermatitis, contact dermatitis, fixed drug eruption
- Nausea, vomiting, diarrhoea
- Malaise, headache, giddiness, marked drowsiness and sleep
- Raised blood urea and/or creatinine
- Increase in liver related enzymes
- Neutropenia
- Topical: Mild pain, burning, stinging. Rash, pruritus, and vulvitis with the ointment and dry or cracked lips and desquamation with the cream have also been reported [20,21].

### Valacyclovir

It was developed to provide increased oral bioavailability of ACV [14]. VACV is better absorbed than ACV due to an active stereoselective transporter in intestinal brush border membrane. VACV is converted rapidly and virtually to ACV after oral administration in healthy adults by intestinal and hepatic first pass metabolism through hydrolysis [22].

There have been several recent advances in the nucleoside analogues used in the treatment of HSV infections. VACV is the best prophylaxis for herpes infections reducing the frequency and severity of outbreaks. When given during the prodromal stage, VACV 2 g twice daily for 1 day is beneficial [14,15]. Gilbert and Mc Burney, in an uncontrolled study, found that prophylactic VACV 500 mg twice daily started either the day before or on the day of facial resurfacing and continued for 14 days [23].

### Conclusion

Varicella zoster causes chicken pox and on reactivation herpes zoster infection, which is unilateral, painful, multiple vesicular rash involving ophthalmic, maxillary and mandibular branches of nerve. It can occur at any age, gender but more commonly affects elderly patients with immunocompromised state. Nowadays it shows many variations and seen in various forms like necrosed pulp to exfoliation of tooth, to osteonecrosis, post herpetic neuralgia and may cause syndrome like ramsay hunt syndrome. As its complications are more severe and complicating to treat, so treatment should be done aggressively. Several areas need to be researched as to prevent them. Many studies are required to treat lesions early and newer treatment modalities to reduce complications.

### Conflict of Interest

No conflict of interest exists.

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