

CASE REPORT

Herpes Zoster with Postherpetic Neuralgia involving the Left Maxillary and Mandibular Branch of Trigeminal Nerve

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ABSTRACT

Herpes zoster (HZ) is an acute, neurocutaneous, self-limiting viral infection caused by the reactivation of the Varicella zoster virus (VZV) that remains latent in the dorsal root ganglion. About 50% of occurrence is seen in older age groups and immunocompromised patients. Less than 5% occur in children. Herpes zoster is characterized by unilateral pain, burning, and tingling sensation followed by vesicular eruptions limited to the single dermatome that are innervated by the single cranial ganglion. Sometimes it leads to postherpetic neuralgia (PHN). We report a case of HZ in a 49-year-old young female involving the left maxillary and mandibular branch of the trigeminal nerve along with PHN.

Keywords: Fifth cranial nerve, Varicella zoster virus, Viral infection.

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CASE REPORT

A 49-year-old female reported to the department with the complaint of intense burning pain on the left side of the face along with the pain in the left upper and lower back teeth region since 4 days. History revealed that the patient had chicken pox when she was 2.5 years old, which was transmitted from her mother. History of fever since 4 days

was also present. On examination, cluster of vesicles were present in the left middle and lower third of the face along with cervical lymphadenopathy and redness of skin, thus following the dermatomal rule (Fig. 1).

Intraoral examination revealed clusters of shallow ulcers on the left side of the upper buccal mucosa, which were about 3.5 mm in size with erythematous and irregular borders and tissue tags were seen. Shallow ulcers unilaterally on left palatal mucosa were also present (Fig. 2). The left mandibular posterior teeth appeared normal without any dental or periodontal pathology, but tenderness was elicited on percussion with teeth #45, 46.

Based on the history of fever, unilateral intense burning pain, and redness on the left side of the face along with the dental pain in the left mandibular posterior teeth, and also the presence of vesicles along the course of the maxillary and mandibular division of trigeminal nerve, it was provisionally diagnosed as HZ of left maxillary and mandibular division of trigeminal nerve. The differential diagnoses included were angioneurotic edema, acute drug eruption and erythema multiforme, pemphigus, and pemphigoid.

Investigation included Tzanck smear, in which there were numerous lymphocytes, multinucleated giant cells, with intranuclear inclusion bodies. Differentiation of HZ and herpes simplex infection was confirmed by polymerase chain reaction (PCR). There was elevation in the levels of immunoglobulin (Ig)G and IgM antibodies against VZV. Varicella zoster virus IgM level was 23.9 μ /mL and VZV IgG level was greater than 155 μ /mL, which was suggestive of acute infection of HZ.

Patient was treated with tablet valacyclovir thrice daily for 15 days and also tablet gabapentin once daily for 15 days, and 0.5% acyclovir cream topically for five times a day. The patient was reviewed after 15 days, showed healing of all the vesicles along with scarring, but persistence pain in the dermatome supplied by the affected nerve was suggestive of PHN (Figs 3 and 4). Patient was then advised to continue tablet gabapentin for another 15 days following which the pain subsided in a month.

DISCUSSION

Herpes zoster also named as zona or shingles is a common viral disease caused by the reactivation of VZV. Varicella zoster virus first enters the host and infects the cells of the

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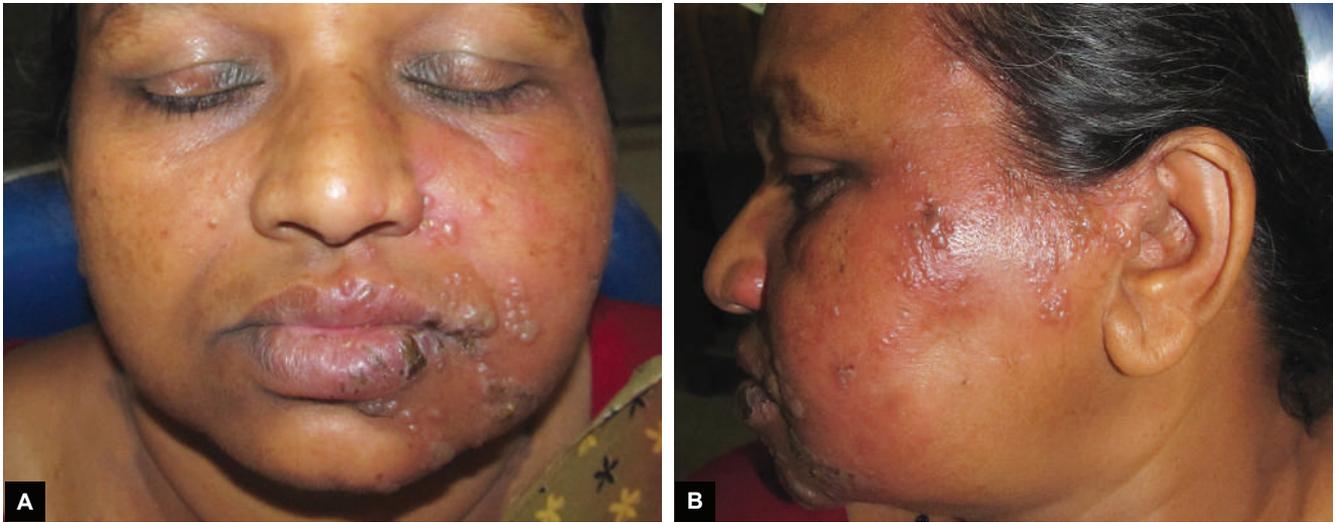
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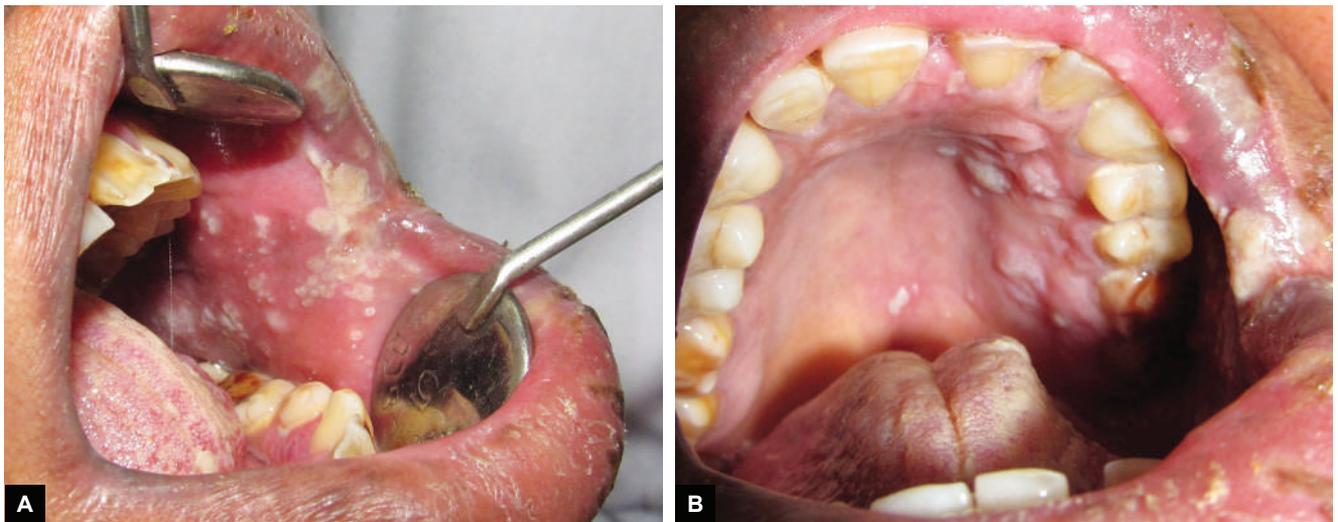
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Figs 1A and B: Extraoral picture showing intact vesicles and redness on the left side of face



Figs 2A and B: Intraoral lesion showing cluster of vesicles on the left side of the buccal and palatal mucosa



Figs 3A and B: Three months postoperative extraoral pictures showing healing with scarring

respiratory tract and epithelium of conjunctiva where it replicates.¹ It spreads throughout the body via reticulo-endothelial system, then reaches the epidermis through

the mononuclear cells via capillary epithelium.¹ It destroys the basal cell layer leading to generalized rash of chicken pox.¹ After healing, the virus passes through the



Fig. 4: Complete healing of intraoral lesions

myelinated nerve fibers and reaches the perineural satellite cells of the dorsal nerve root ganglion; reactivation of virus leads to HZ.¹ Opstelten et al,² Chen et al,⁴ and few other authors found female predilection with 3.9/1,000 patients/year in females and 2.5/1,000 patients/year in males. Herpes zoster occurs most commonly in the older age groups. Chen et al⁴ stated that there is lower incidence of HZ among patients aged ≤ 65 years.¹⁻⁴ The various triggering factors include malnutrition, patients on steroids, cytotoxic drugs, chemotherapeutic agents, diabetes, chronic obstructive pulmonary disease, malignancies, such as leukemia and lymphoma, and immune disorders.^{1,3,5-7} Trauma and stress can lead to reactivation of VZV due to reflex irritation and hyperemia of ganglion. Our case was a 49-year-old female who had history of chicken pox during childhood.

The frequency of the involved nerves, according to Costache and Costache,⁸ is 48% cervical branch (C1–C4),

22% intercostal branch (T2–T12), 15% branchial branch (C5–T1), 9% lumbar (L1–L4), 5% sacral (L5–S4). The dermatome affected in the head and neck region along with the area affected along the distribution of the affected nerve is given in Table 1.^{9,10}

Herpes zoster occurs in three successive stages: prodromal, acute, and chronic neuropathic stages, but few patients do not develop symptoms of all the stages.^{1,6} The prodromal (pre-eruptive stage) presents as pain associated with mild fever, headache, and dysesthesia. Patil et al¹¹ reported a case of HZ affecting the trigeminal nerve presenting with odontalgia. This was in accordance to our case reported with history of odontalgia for past 4 days with neuralgia, burning, and tingling sensation along the course of the affected nerve.

The acute stage is characterized by appearance of rash in a unilateral dermatome that progresses into pustule within 1 to 7 days; then dry and form a painful crust in 14 to 21 days. Intraoral lesions develop after cutaneous rash.^{1,6} Oral manifestation occurs when the HZ involves the maxillary or mandibular division of trigeminal nerve.¹ Patil et al¹¹ and Patro et al¹² reported cases of HZ involving the maxillary division of trigeminal nerve extraorally showing unilateral diffuse erythema, edematous area along with multiple vesicles involving the upper lip, zygomatic region, ala of the nose and lower eye lid, and intraorally involving the upper labial mucosa and hard palate. The distribution of vesicles along the left maxillary nerve was similar to our case, and she also had involvement of the left side of palate. Ophthalmic nerve involvement was ruled out by consulting an ophthalmologist lead to the diagnosis of HZ involving right maxillary branch of trigeminal.

Table 1: Dermatomes affected by HZ

<i>Dermatome affected</i>	<i>Area affected along the nerve distribution</i>
Ophthalmic branch of CN V	Lesions involving the frontal region, upper eye lid, conjunctiva, lacrimal gland, anterior part of the scalp, involvement of ciliary ganglion causes Argyll-Robertson pupil. The involvement of nasociliary branch causes lesions in the tip and side of the nose called "Hutchinson's sign"
Maxillary branch of CN V	Lesions involving middle third of the face, lower eyelid, upper eyelid, side of the nose, upper lip, nasopharynx, antrum, ethmoids, and intraorally affecting maxillary arch, buccal mucosa, uvula, and tonsillar area
Mandibular branch of CN V	Lesions in the lower third of the face, lower lip, temporal region and intraorally involving anterior part of the tongue, floor of the mouth, and buccal mucus membrane
Facial nerve (VII)	Ramsay Hunt syndrome type I causes herpetic lesions in the oral cavity or external ear, combined with facial palsy. It is often accompanied by neurological disturbances
Facial nerve with vestibulocochlear nerve (VIII)	Ramsay Hunt syndrome type II causing facial palsy, hearing loss, vertigo, and tinnitus with characteristic vesicle on external ear meatus
VII with VIII, IX, X, and V cranial nerve	Ramsay Hunt syndrome III associated with Frankl-Hochwart syndrome (polyneuritis cranialis menieriformis)
Glossopharyngeal (IX) and vagal nerve (X)	Rash on the palate, posterior tongue, epiglottis, tonsillar pillars, and occasionally external ear vesicles
Arnold nerve which is the cervical branch of the vagus nerve	Zoster meningoencephalitis

CN: Cranial nerve

The chronic neuropathic pain syndrome is also known as PHN. It is defined as a sharp, intense, radiating pain lasting after eruptive stage for about 1 to 3 months but may also last for years and decades.^{1,6} It occurs due to demyelination of the affected nerve leading to pain in the affected dermatome. Lapolla et al¹³ stated that the prevalence of PHN ranges from 9 to 73% in which pain persists even after healing of the vesicle for more than a week, month, or year. Lapolla et al¹³ also stated that when gabapentin was combined with valacyclovir in treating the acute herpetic zoster, it reduced the rate of PHN. This was in accordance with our case who had persisting pain even after the healing of the vesicles, but there was decrease in the intensity of the pain after 15 days and total remission in a month's duration.

Differential diagnosis is done in cases, such as multiple nonpersistent ulcer in case of herpes simplex viral infection or recurrent aphthae, and in case of multiple persistent ulcers, lichen planus, pemphigoid or pemphigus, or immune defect due to drugs. Diagnosis is frequently done by recognizing the characteristic unilateral distribution of the lesion in case of HZ as seen in our case. Cytology (Tzanck smear) plays an important role in early diagnosis in which the viral lesion can be recognized by the presence of multinucleated giant cells (Tzanck cells) and intranuclear inclusion bodies and numerous lymphocytes, which was in accordance with our case. Other advanced diagnostic techniques like fluorescent antibody staining technique and PCR can be done to identify and differentiate viral antigen. We did PCR using our patient's serum: VZV IgM of 21.9 µ/mL and VZV IgG of >150 µ/mL were found to be elevated, which are suggestive of acute infection of HZ.^{1,3,7}

Herpes zoster is treated by administering antiviral drugs within 72 hours after onset of rash.^{1,5} Topical antiviral agents, such as 0.5% acyclovir cream and docosanol 10% cream five times a day until the lesions heal, are effective in treating herpetic infection for patients who are 12 years of age or older.^{1,14} Oral administration includes acyclovir 800 mg, five times daily for 7 to 10 days; or in severe cases, 10 mg/kg IV every 8 hours for 7 to 10 days is recommended. If acyclovir is not effective, famciclovir 500 mg orally three times daily for 7 days is recommended; other drugs like valacyclovir 1,000 mg orally three times daily for 7 days have been proved to be effective. In immunocompromised patients, drugs, such as brivudine and cidofovir for 2 weeks are effective. Recent advanced targeted drug therapies are directed toward the viral deoxyribonucleic acid, such as ASP2151 amenamevir inhibits helicase primase complex, FV 100 two bicyclic nucleoside analogues, valamaciclovir nucleoside analog.⁵

Treatment of acute pain associated with PHN is done by orally administering anticonvulsants, such as: phenytoin,

100 to 300 mg orally at bedtime, increase dosage until response is adequate, or blood drug level is 10 to 20 mg/mL; carbamazepine, 100 mg orally at bedtime, increase dosage by 100 mg every 3 days until dosage is 200 mg three times daily, response is adequate, or blood drug level is 6 to 12 mg/mL; gabapentin, 100 to 300 mg orally at bedtime, increase dosage by 100 to 300 mg every 3 days until dosage is 300 to 900 mg three times daily or response is adequate.^{1,14}

Second line of treatment is done using opioids, tricyclic antidepressants, and selective serotonin norepinephrine reuptake inhibitors.¹⁴ Nagalaxmi et al¹⁴ stated that prednisolone 60 mg is considered initially to reduce the acute pain but care should be taken while tapering the drug dose, as the use of this drug is still controversial. Other advanced treatment modalities are electrical stimulation of thalamus, anterolateral cordotomy, intercostal nerve cryotherapy, pulsed radiofrequency ablation, spinal cord stimulation, botulinum toxin injection to reduce the intensity of PHN.¹⁴

We treated our patient with 1 gm tablet of valacyclovir thrice daily and also tablet gabapentin 300 mg once daily for 15 days and 0.5% acyclovir cream topically applied five times a day. Healing with scarring occurred after 15 days with persistent pain. Patient was advised to continue tablet gabapentin for another 15 days which was then followed by remission of pain. The patient was asymptomatic during continuous follow-up period of 3 months.

CONCLUSION

Herpes zoster involving the trigeminal nerve can mimic other oral lesions but complication involving HZ leads to the most painful PHN. Early diagnosis by a specialist oral physician and prompt treatment in the early stage helps in preventing the severity of PHN and prognosis of the disease.

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