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Research Article

HERPES ZOSTER OF LEFT MAXILLARYDIVISION OF TRIGEMINAL NERVE- A CASE REPORT

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A R T I C L E I N F O

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A B S T R A C T

Herpes zoster or 'shingles' is a painful vesicular rash resulting from reactivation of the varicella-zoster virus that also causes chickenpox as primary infection. The incidence of HZ infection (HZI) increases with age and the degree of immunosuppression. Herpes zoster of the trigeminal nerve is a clinical entity consisting of erythematous macules, papules, vesicles, bullae, small ulcers and erythematous plaques in the dermatome supplied by the involved nerve, with characteristic short acute/pre-eruptive phases and long herpetic periods with pain. During the prodromal stage of infection of V2 or V3, the only presenting symptom may be odontalgia, which may prove to be a diagnostic challenge for the dentist.In the present article we report a case of herpes zoster infection in a 46 years old female involving left maxillary division of the trigeminal nerve and the article also focuses on the pathogenesis, clinical picture, diagnosis, complications and management of such cases.

INTRODUCTION

Herpes zoster (HZ) is an acute infectious viral disease of extremely painful and incapacitating nature which is characterised by inflammation of dorsal root ganglia or extramedullary cranial nerve ganglia, associated with vesicular eruptions of the skin or mucous membrane and burning type of pain in an area supplied by the affected nerve.When herpes zoster affects the oral and maxillofacial region it should be considered in the differential diagnosis of those presenting with atypical odontalgia.[1]

Approximately 13% of the patients present with infections involving any of the three divisions of the trigeminal nerve. In the present article we report a caseof Herpes zoster infection of the maxillary nerve, which is quite rare, approximately seen in 1.7% of the cases. Immediate management is required to reduce the risk and the complications of the infection, especially in immunocompromised individuals, which may be responsible for significant morbidity; within 72 hours the antiviral therapy should be started to prevent complication such as post-herpetic neuralgia (PHN).[2]

Case Report

A 46 year old female patient presented with the multiple progressive vesicular eruptions on the left side of the face from past 3 days. Patient also complaints of continuous watering from the left eye. She had burning sensation and pain over the lesion, with no history of similar lesions in the past.

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On extraoral examination, a diffuse swelling of left half of the face (Fig.1A) with presence of small clusters of pinheaded vesicles around the slopes of nasal bridge, lateral canthus of eye, upper lip and philtrum region on the left side with some focal areas of ruptured vesicular conglomerate on upper lip near philtrum region on left side were seen. All the lesions were limited to the left side of the face, not crossing the midline (Fig.1B).

Intraoral examination revealed, clusters of shallow irregular ulcers on the left side of upper labial mucosa with erythematous surrounding mucosa and with tissue tags (Fig.2A). Multiple small vesicles were evident on the left half of the hard palate with surface nodularity and erythematous mucosa(Fig.2B). The teeth 21,22,23,24,26,77 were mobile and non tender.

Based on the history and clinical presentation, a provisional diagnosis of Herpes Zoster of left maxillary division of trigeminal nerve was given. Differential diagnosis of Recurrent Intraoral Herpes Simplex Infection, Acute Drug Eruption and Cytomegalovirus Infections were considered.

The lesion was subjected for Tzanck smear, which showed numerous lymphocytes, multi nucleated giant cells and intra nuclear inclusion bodies.

The patient was also referred to the ophthalmic consultation, ocular examination revealed no evidence of lesions on eyelid or conjunctival and corneal involvement.

A Final diagnosis of Herpes Zoster Viral Infection involving dermatome supplied by left maxillary nerve was given.

The treatment was immediately started with antiviral medication; tablet acyclovir 400mg 5 times a day for 7 days and 0.5% Acyclovir cream to be applied topically for five times a day for 1 week. Since the vesical were also seen near the outer canthus of left eye, patient was advised to use cool compresses and mechanical cleansing of the eye region. The patient was reviewed after 4 days, responded well to the treatment and showed healing of all the vesicles along with scaring and scab formation (Fig.3A), and on intra oral examination, partial remission of the lesions was evident(Fig.3B). Patient was further followed up till the complete remission of the lesions.

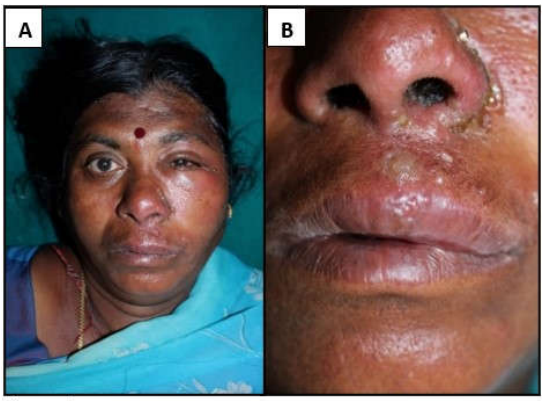

Figure 1

Fig 1 (A)Facial oedema on left side, (B)clusters of vesicles on left side of face not crossing the midline.

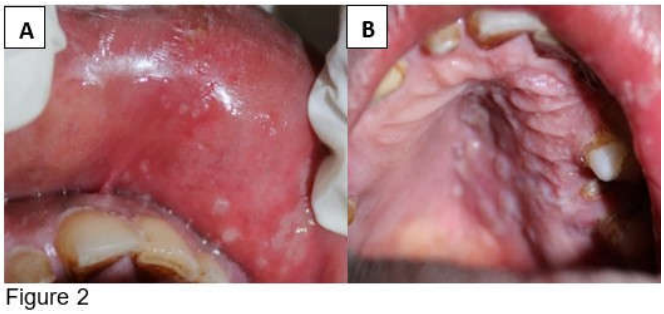

Figure 2

Fig 2 (A) Multiple small irregular ulcers on left side of upper labial mucosa, (B) clusters of vesicles on left side of palate.

Figure 3

Fig 3 (A) Healing of all the vesicles along with scaring and scab formation, (B) partial remission of the lesions on upper labial mucosa and palate.

DISCUSSION

Herpes zoster (HZ), also known as shingles, is the result of reactivation of endogenous latent varicella zoster virus (VZV), which is in dormant stage within sensory dorsal root, cranial nerve and autonomic ganglia. HZ can manifest any time after a primary infection with VZV (i.e., varicella or chickenpox). The activated virus travels back down the corresponding cutaneous nerve to the adjacent skin, causing typically a painful, unilateral vesicular eruption in a restricted dermatomal distribution.[1]

Varicella zoster virus (VZV) belongs to genus varicello – virus, family herpes viridae, subfamily alpha – herpes virnae also known as HHV – 3 (human herpes virus – 3). The virus is widely present in most populations.[3] Herpes Zoster is a painful, blistering disease caused by HHV 3, having unilateral dermatomal distribution of lesions.Word Herpes is derived from a Greek word herpein, which means "To Creep" and Zoster, a Greek word, refers to "belt or girdle", HZ is more commonly known as shingles,which is derived from the Latin word *cingulus*, refers to 'girdle or belt.' This is because a common presentation of HZ involves a unilateral rash that can wrap around the waist or torso like a girdle.[1]

In 1892, Von Bokay first suggested the relationship between the etiologies of varicella and HZ. The first suggestion was made by Garland and Hope-Simpson that HZ is caused by reactivation of latent virus acquired during varicella.[2]

Varicella-zoster virus (VZV)leads to primary infection varicella (chicken pox) and then becomes latent, usually in the dorsal root ganglia or ganglia of the cranial nerves. Unless the immune system is compromised the VZV virus is usually suppressed.[3] However, for reasons that are not fully understood, the virus reactivates from its dormant state in the sensory ganglion, replicates in the nerve cells and sheds virions from the cells that are carried down the axons to the skin served by that ganglion. The local immune response results in skin blisters or ocular inflammation depending on which tissues are affected. Perineuritis causes intense pain along with the nerve distribution. Paraesthesia and segmental pain at the area supplied by involved sensory nerve may be noted before the onset of rash. Aging, immunosuppression therapy and psychological stress all could be factors resulting in reactivation of the virus.[4]

Herpes zoster infection of the skin (shingles) is more common in adults and starts with a prodrome of deep, aching and burning pain. There is usually little or no fever or lymphadenopathy. This is followed within 2–4 days by the appearance of crops of vesicles in a dermatomal or zosteriform pattern. The lesions usually begin to dry and scab 3–5 days after appearing. Total duration of the disease is generally between 7 and 10 days; however, complete healing may take more than 4 weeks.[5]

Herpes zoster is a less common endemic disease compared to varicella with a reported incidence of 1.5–3 cases per 1000 participants; incidence increases to 10 per 1000 in those over 75 years.[6]HZ may affect any sensory ganglia and its cutaneous nerve. Most of the infections affect dermatomes of Thoracic nerve 3 to lumbar nerve 2, however, approximately 13% of the patients present with infections involving any of the three branches of the trigeminal nerve.[7]

Involvement of the trigeminal nerve leads to lesions on the upper eyelid, forehead and scalp with V1(ophthalmic division) and less commonly with a typicalHutchinson's sign,which is defined as skin lesions at the tip, side, or root of the nose; This is a strong predictor of ocular inflammation and corneal denervation in herpes zoster ophthalmicus, especially if both

branches of the nasociliary nerve are involved.[8]Lesions on midface and upper lip with involvement of V2(maxillary division) and lower lip with involvement of V3(mandibular division) are seen. With the involvement of V2, patients experience a prodrome of pain, burning and tenderness, usually on the palate on one side. Thus, in present case left maxillary nerve (V2) is involved as the lesions were present on the midface and palate on left side.[9]

Management

The objective of conventional therapy in the treatment of HZ is to accelerate healing of the lesions, reduce the accompanying pain and prevent complications.

Antiviral drugs

Antiviral agents have been shown to decrease the duration of herpes zoster rash and the severity of pain associated with the rash. However, these benefits have only been demonstrated in patients who received antiviral agents within 72 hours after the onset of rash. Antiviral agents may be beneficial as long as new lesions are actively being formed, but they are unlikely to be helpful after lesions have crusted.[10,11]

The effectiveness of antiviral agents in preventing postherpetic neuralgia is more controversial. Numerous studies evaluating this issue have been conducted, but the results have been variable. Based on the findings of multiple studies, acylovir (Zovirax) therapy appears to produce a moderate reduction in the development of postherpetic neuralgia. Other antiviral agents, specifically valacyclovir (Valtrex) and famciclovir (Famvir), appear to be at least as effective as acyclovir.[12,13]

The recommended dosages of commonly used antiviral agents in the management of HZ infection are given in table 1.

Table 1 Antiviral Agents

Acyclovir	400 mg orally five times daily for 7–10 days, 10 mg/kg intravenously every 8 h for 7–10 days
Famciclovir	500 mg orally three times daily for 7 days
Valacyclovir	1000 mg orally three times a day for 7 days
Brivudin	125 mg once daily for 7 days

Corticosteroids

Corticosteroids, with strong anti-inflammatory effects, may effectively improve the levels of inflammatory factors such as IL-6 and IL-10, reduce nerve injury, and promote the regression of blisters as well as improve nerve pain.[14,15]Oral corticosteroids have commonly been used for pain management in HZ, although clinical trials have yielded inconsistent results for reducing development of PHN. One study using a combination of Prednisone and Acyclovir demonstrated a significant reduction in pain associated with HZ.[4]

Steroids is contraindicated in the individuals with medical conditions that could be made worse by corticosteroids. The use of epidural, intramuscular or subcutaneous corticosteroids was not be recommended. However, it was recommended that intrathecal corticosteroids be used only for people with postherpetic neuralgia who have not responded to adequate trials of other treatments. [16,17] Levin reported that corticosteroids caused immunosuppression, thus increasing or even aggravating the infection of patients with herpes zoster.[18]

Analgesics and Non-steroidal anti-inflammatory drugs

In patients with severe pain, use of narcotics may be indicated. Use of nerve block injections is another option in the conventional medical model. Local anaesthetic may be injected around the affected nerves, providing pain relief typically lasting for 12–24 hr.[4]One of the study found that the use of NSAIDs was associated with an increased risk of severe skin and soft tissue complications in varicella, which mostly includes children and the most commonly reported complication being the varicella-associated necrotizing fasciitis.[19]

Postherpetic neuralgia

Although postherpetic neuralgia is generally a self-limiting condition, it can last indefinitely. Treatment is directed at pain control while waiting for the condition to resolve. Pain therapy may include multiple interventions, such as topical medications, over-the-counter analgesics, tricyclic antidepressants, anticonvulsants and a number of nonmedical modalities. Occasionally, narcotics may be required. Dosage recommendations are provided in table 2.[20]

The recommended treatment options for postherpetic neuralgia are given in table 2.

Table 2 Treatment Options for Postherpetic Neuralgia

MEDICATION	DOSAGE
Topical agents	
Capsaicin cream (Zostrix)	Apply to affected area three to five times daily.
Lidocaine (Xylocaine) patch	Apply to affected area every 4 to 12 hours as needed.
Tricyclic antidepressants	
Amitriptyline (Elavil)	10 to 25 mg orally at bedtime; increase dosage by 25 mg every 2 to 4 weeks until response is adequate, or to maximum dosage of 150 mg per day.
Nortriptyline (Pamelor)	10 to 25 mg orally at bedtime; increase dosage by 25 mg every 2 to 4 weeks until response is adequate, or to maximum dosage of 125 mg per day.
Imipramine (Tofranil)	25 mg orally at bedtime; increase dosage by 25 mg every 2 to 4 weeks until response is adequate, or to maximum dosage of 150 mg per day.
Desipramine (Norpramin)	25 mg orally at bedtime; increase dosage by 25 mg every 2 to 4 weeks until response is adequate, or to maximum dosage of 150 mg per day.
Anticonvulsants	
Phenytoin (Dilantin)	100 to 300 mg orally at bedtime; increase dosage until response is adequate or blood drug level is 10 to 20 µg per mL (40 to 80 µmol per L).
Carbamazepine (Tegretol)	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 to12 µg per mL (25.4 to 50.8 µmol per L).
Gabapentin (Neurontin)	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.

Botanicals with specific efficacy for HZ Capsaicin (from* Capsicum frutescens*)

Capsaicin is an alkaloid derived from cayenne pepper (*Capsicum frutescens*). A well-studied compound, Capsaicin is of particular importance in the treatment of PHN because of its effect on C-fibre sensory neurons. These neurons release inflammatory neuropeptides, such as Substance P, that mediate neurogenic inflammation and chemical-initiated pain.[4]

Traditional Chinese medicine

Acupuncture has long been regarded as an effective therapy for pain management. One study documented a case of a 52-year-old man diagnosed with PHN who was successfully treated after four sessions using a combination of Acupuncture and Moxibustion.[4,14]

Transcutaneous electrical nerve stimulation

Use of Transcutaneous Electrical Nerve Stimulation (TENS) therapy has been beneficial in the management of PHN. In one review, the use of combination therapy consisting of Amitriptyline, topical Capsaicin and TENS was recommended for the treatment of PHN over antiviral therapy.[4]

Complications

The most common complication associated with HZ is the development of Post Herpetic Neuralgia (PHN), a condition where pain persist for months and sometimes years after the rash resolves. PHN is of particular concern with increasing age because it is estimated that half the individuals over age 50 who develop shingles also develop PHN. Postherpetic neuralgia occurs in 36.6% of patients over the age of 60 and in 47.5% over the age of 70. Other potential complications of HZ include encephalitis, myelitis, peripheral nerve palsies and forms of contralateral hemiparesis. An uncommon complication of HZ involving geniculate ganglion is Ramsay Hunt syndrome. Patients develop Bell's palsy, vesicles of the external ear and loss of taste sensation in anterior two-third of the tongue.[20]

Schwartz and Kvoring reported 10 cases of HZ with postherpetic complications including osteonecrosis of jaw, exfoliation of teeth, severe periodontitis and scarring of the skin.[5]

CONCLUSION

Herpes zoster is a viral infection that is manifested in the oral mucosa as unilateral ulceration & acute pain is the symptom afflicting HZ patients the most. Early HZ diagnosis and treatment are important in the attempt to optimize pain management in the acute stage and to prevent complications, such as PHN. Therefore, dentists should have a thorough knowledge about the presentation of the condition, its treatment and probable complications. Differential diagnosis is very important to ensure that correct treatment is performed.

References

1. Mohan RP, Verma S, Singh U, Agarwal N. Herpes zoster. BMJ Case Rep. 2013:bcr2013010246.
2. Raj SS, Verma P, Mahajan P, Puri A. Herpes zoster infection of the face: A case report with review of literature. J Indian Acad Oral Med Radiol 2017;29:159-61
3. Thakur I, Shilpa B, Satheesha Reddy HB, Koppula SK. Herpes zoster infection of maxillary nerve: A case report. J Indian Acad Oral Med Radiol 2017;29:156-8
4. Roxas M. Herpes Zoster and Postherpetic Neuralgia: Diagnosis and Therapeutic Considerations. Altern Med Rev. 2006;11(2):102-13.
5. Sampathkumar P, Drage LA, Martin DP. Herpes zoster (shingles) and post herpetic neuralgia. Mayo Clin Proc 2009;2013:274–80
6. Koshy E, Mengting L, Kumar H, Jianbo W. Epidemiology, treatment and prevention of herpes zoster: A comprehensive review. Indian J Dermatol Venereol Leprol 2018;84:251-62
7. K Kawai, BP Yawn Risk factors for herpes zoster: a systematic review and meta-analysis Mayo clinic 2017 - Elsevier
8. John B. Cason MD Herpes Zoster Ophthalmicus, American academy of ophthalmology 2020.
9. Deshmukh R *et al*. Herpes Zoster (Hz): A Fatal Viral Disease: A Comperhensive Review. IJPCBS. 2012;2(2):138-45.
10. S J Stankus , M Dlugopolski, D PackerManagement of Herpes Zoster (Shingles) and Postherpetic Neuralgia 2000 Apr 15;61(8):2437-44, 2447-8.
11. Crooks RJ, Jones DA, Fiddian AP. Zoster-associated chronic pain: an overview of clinical trials with acyclovir. *Scand J Infect Dis Suppl*. 1991;80:62–8.
12. Opstelten W, Eekhof J, Neven AK, Verheij T. Treatment of herpes zoster. Can Fam Physician. 2008;54:373-7.
13. Dworkin RH, Schmader KE. Treatment and prevention of postherpetic neuralgia. Clin Infect Dis 2003;2013:877–82
14. Peng, L., Du, B., Sun, L., Zhao, Y., Zhang, X."Short-term efficacy and safety of prednisone in herpes zoster and the effects on IL-6 and IL-10". Experimental and Therapeutic Medicine 18.4 (2019): 2893-2900.
15. Santee J A. Corticosteroids for herpes zoster: what do they accomplish? American Journal of Clinical Dermatology 2002; 3(8): 517-524.
16. Han Y, Zhang J, Chen N, He L, Zhou M, Zhu C. Corticosteroids for preventing postherpetic neuralgia. Cochrane Database Syst Rev. 2013;(3):CD005582. Published 2013 Mar 28.
17. Levin MJ: Varicella-zoster virus and virus DNA in the blood and oropharynx of people with latent or active varicella-zoster virus infections. J Clin Virol. 61:487–495. 2014.
18. Mikaeloff Y, Kezouh A, Suissa S. Nonsteroidal anti-inflammatory drug use and the risk of severe skin and soft tissue complications in patients with varicella or zoster disease [published correction appears in Br J Clin Pharmacol. 2010 Jun;69(6):722]. *Br J Clin Pharmacol*. 2008;65(2):203-209.
19. Singh S, Gupta R, Kaur S, Kaur J. Post-herpetic neuralgia: A review of current management strategies. Indian J Pain 2013;27:12-21
20. Wollina U. Variations in herpes zoster manifestation. Indian J Med Res 2017;145:294-8