

SHINGLES - A BUNCH OF THORNS

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ABSTRACT

Varicella zoster virus is a herpes virus, it causes both primary and recurrent infection and remains latent in neurons present in sensory ganglia. A case report of herpes zoster involving mandibular division of trigeminal nerve with differential diagnosis and treatment is discussed here. This article enlightens the knowledge of dental practitioner to diagnose and manage the disease.

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INTRODUCTION

Herpes zoster (HZ) which is also known as Shingles, is an acute infection of viral origin resulting from the reactivation of the DNA virus varicella zoster ^[1]. HZ were usually limited to one dorsal root ganglion or the sensory ganglion of a cranial nerve producing pain and skin lesions along the distribution of the involved nerve ^[2]. HZ probably results most often from a failure of the immune system to contain latent virus replication. The

triggering factors initiating the onset of an attack of herpes zoster are varied and may include trauma, development of malignancy or tumour involvement. The patient was prescribed oral amitriptyline 25mg once daily at bedtime for 4 weeks and paracetamol 500 mg Bid for 5 days. Antiviral drugs are not recommended because treatment with acyclovir remains appropriate for patients presenting upto 72 hrs after rash onset.



Fig-1: Shows unilateral distribution of lesions affecting mandibular division of Trigeminal nerve.



Fig-2: Multiple irregular ulcerations and crusts in left side of face.

Discussion

Herpes Zoster [HZ], a condition which results from the reactivation of a latent varicella zoster virus [VZV]. VZV is usually transmitted from an exogenous source to the host via direct contact with active vesicular lesions or by exposure to air droplets from nasopharyngeal secretions [3, 4]. The virus is thought to be maintained in its latent form by

VZV-specific cell mediated immunity. Therefore, people with cell mediated immune suppression are at increased risk of zoster. The incidence of shingles increases with age primarily because there is a gradual reduction in the level of T-cell immunity to VZV. The incidence of shingles is up to 15 times higher in HIV infected patients than in uninfected patients [5].

Shingles can affect any of three branches of the trigeminal nerve. Approximately 15% of all cases of herpes zoster are trigeminal. The involvement of mandibular and maxillary branches without the ophthalmic branch involvement are relatively rare and accounts for only 1.7% of the total cases of HZ. Women might be more likely to have increased prevalence of risk factors for zoster, or there may be some biological mechanism by which women are more susceptible to VZV reactivation [6]. Almost 60 % of all patients were over 45 years of age, but the age distribution in the two sexes is different in certain respects.

In this case, Patient is female of age 45 with the characteristic unilateral presentation of lesions involving only the mandibular division of trigeminal nerve.

Patients with HZ infections usually progress through three stages: i) prodromal stage ii) active stage iii) chronic stage.

The prodromal stage is characterized by sensations such as burning, tingling, itching, pricking, occurring along the cutaneous distribution of dermatome [7].

Some patients do not form vesicular eruptions of the active stage, but do develop pain restricted to a dermatome, and this has been termed zoster sine herpete which makes proper diagnosis more difficult [8].

Shingles of the first division can lead to blindness secondary to cornea scarring and should be managed by an ophthalmologist. Facial and intraoral lesions are characteristic of Shingles involving the second and third divisions of the trigeminal nerve. Early diagnosis and prompt treatment of the disease in the prodromal phase by the use of antiviral agents should probably be the mainstay of its management. For patients whose rash is recognized after 72 hr may be considered too late for initiation of effective antiviral therapy [9].

One of the most important complications of HZ is post herpetic neuralgia as pain develops after 30 days or 120 days after the onset of the acute rash. Patient was made aware about post herpetic neuralgia [10].

Valaciclovir, famciclovir and acyclovir – are anti viral drugs available for the treatment of HZ. They reduce the severity and duration of

the illness if started within 72 hours of onset of the rash. All patients with zoster ophthalmicus should receive antiviral therapy even if it is delayed beyond 72 hours. Reduction in incidence of post herpetic neuralgia at six months by about half with early commencement of low-dose amitriptyline 25 mg at night (for 90 days) . Transcutaneous electrical nerve stimulation (TENS) may also be useful [11].

Differential diagnosis

- ✓ Unilateral Herpes simplex infection
- ✓ Pemphigus foliaceus
- ✓ Bullous pemphigoid
- ✓ Hand foot and mouth disease
- ✓ Erythema Multiforme

Conclusion

Herpes zoster infection though not frequently encountered in general dental practice, many patients do report to the dental clinic with the complications of HZ infection, involving the trigeminal nerve in about 15% cases. The prodromal pain of HZ is nonspecific and even if a doctor is consulted at this stage, it will be difficult to diagnose. The rash of HZ often starts proximally within the affected dermatome. Early diagnosis and prompt treatment by antiviral drugs in the prodromal stage of the HZ may aid in reducing the

duration and the severity of pain of HZ infection and also prevent the complications.

Declaration of Conflicting Interests:

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

1. G. Sree Vijayabala .Herpes Zoster of the Trigeminal Nerve .Journal of Clinical and Diagnostic Research. 2012 ,6(7): 1365-1366.
2. A.Vineet .Oro-Facial Herpes Zoster: A Case Report With a detailed review Of Literature. Oral & Maxillofacial Pathology. 2013;4(1).
3. Manjunath Reddy Bandral. Oral Complications of Herpes Zoster Infection .Case Report. International Journal of Dental Clinics. 2010;2(4): 70-73.
4. Lorren W. Jackson. Herpes Zoster Ophthalmicus: A Case of Reactivated Varicella.Case report. Hospital Physician. 1999. 45 -49.
5. Jeffrey I. Cohen. Herpes Zoster.New Eng J Med 2013; 369: 255–263.
6. Thomas SL. What does epidemiology tell us about risk factors for herpes zoster?: Lancet Infect Dis 2004;4:26-33..
7. Prachi Chhimwal. Herpes Zoster A Case Report. Journal Of Dental & Oro-Facial Research. 2015;11(1).
8. E.Tidwell .Herpes zoster of the trigeminal nerve third branch.a case report and review of literature. International endodontic journal, 1999; (32): 61-66.
9. M. J. Wood .Treatment of Acute Herpes Zoster: Effect of Early (.48 h) versus Late (48–72h) Therapy with Acyclovir and Valaciclovir on Prolonged Pain. The Journal of Infectious Diseases 1998;178(1):S81-84.
10. Martin S. Greenberg. Ulcerative, Vesicular and Bullous lesions.Burket's oral medicine 10th Edition . BC Decker.2003.55-56.
11. MC Wehrhahn. Herpes zoster: epidemiology, clinical features, treatment and prevention. Aust Prescr 2012;35:143-147.