*CASE REPORT*

# PRODROMAL HERPES ZOSTER MIMICKING ODONTALGIA – A DIAGNOSTIC CHALLENGE

Shilpa Patil[1], K.Srinivas[1], BH. Satheesha Reddy[2], Mudit Gupta[3]

**ABSTRACT**

Herpes zoster (shingles) is caused by reactivation of the latent varicella zoster virus which is present due to an earlier varicella infection (chicken-pox). Herpes Zoster is a less common and endemic disease than varicella, although factors causing reactivation are still not well known, but it occurs in older and/or immunocompromised individuals. Involvement of C3, T5, L1, L2 and first division of trigeminal nerve are the most frequently encountered whereas the involvement of second and third division of trigeminal nerve is rarely seen. During the prodromal stage, the only presenting symptom may be odontalgia, which may prove to be a diagnostic challenge for the dentist, since many diseases can cause orofacial pain, and the diagnosis must be properly established before final treatment. Here we present a case of herpes zoster involving the second division of trigeminal nerve masquerading as odontalgia. The difficulties in diagnosis and management are discussed.

**KEYWORDS**: herpes zoster, odontalgia, prodromal, trigeminal.

## INTRODUCTION

Diagnostic assessment in patients with orofacial pain may be challenging due to the close proximity between teeth and other orofacial tissues, and by symptoms associated with neurological disorders (1). Herpes Zoster (HZ) affecting the oral and maxillofacial region may pose a significant diagnostic challenge and should be considered in the differential diagnosis of those presenting with atypical odontalgia (2). Other diagnoses in the early stages of symptoms may include irreversible pulpitis, acute periapical periodontitis or even acute sinusitis. Prompt management is required, especially in immunocompromised individuals, to prevent complications, which may cause significant morbidity (3). We report a case of HZ affecting the trigeminal nerve presenting as odontalgia in a patient and review the relevant literature.

## CASE REPORT

A male patient aged 50 yrs. reported to the department of Oral Medicine and radiology with the chief complaint of pain since 3 days. On eliciting history of presenting illness patients gave history of pain in the upper left front tooth region 3 days back which was sudden in onset, severe in intensity, continuous in periodicity, throbbing type, initiates by itself, aggravates on chewing and relieves after taking medication. The pain was thought to be arising due to root stumps which was present in the region of 23 and was subsequently removed and patient was put on amoxicillin 500 mg TDS and combination of ibuprofen and paracetamol TDS as analgesic. Next day patient developed rash over his left mid face region which was thought to be as an allergic reaction to one of the above said drugs and subsequently these drugs were withdrawn and

[1]Department of Oral Medicine & Radiology, AECS Maaruti College of Dental Sciences & Research Centre, Bangalore
[2]Head, Department of Oral Medicine & Radiology, AECS Maaruti College of Dental Sciences & Research Centre, Bangalore
[3]Department of Oral Medicine & Radiology, AECS Maaruti College of Dental Sciences & Research Centre, Bangalore
**Corresponding Author:** Dr. Shilpa Patil, Email –Id: shilpip@rediffmail.com

patient was given cetrizine and only paracetamol 650 mg was given for control of pain. On day 2, patient developed multiple blisters with erythema and swelling of left mid face region with excruciating pain. Diffuse erythema and edematous area was seen on the left middle third of face extending antero posteriorly from philtrum upto the tragus and supero inferiorly from lower eyelid to upper lip. Multiple bullae and vesicles were seen on the swelling. Erosions with oozing watery sections were seen on the upper lip region and over the temple region. Redness of the left cornea was seen with increase in watery secretions. On palpation, temperature was increased, there was tenderness on palpation. On intra oral examination, a solitary shallow ulcer measuring roughly around 0.5 cm in diameter was seen i.r.t. left buccal mucosa opposite to 26 which was surrounded by tissue tag suggestive of ruptured vesicle. On hard tissue examination, healing socket was seen i.r.t. 23. Patient gave history of Atypical seizures without aura for which he was on anti convulsant therapy i.e. Carbamazepine 200 mg BD and phenytoin 100 mg BD since 10 years and no episode of seizures is reported in the last 10 years. On the basis of clinical examination and history given by the patient a provisional diagnosis of herpes zoster of maxillary branch of trigeminal nerve was given and a differential diagnosis of recrudescent herpes labialis and herpes simplex infection of mid face region was made.

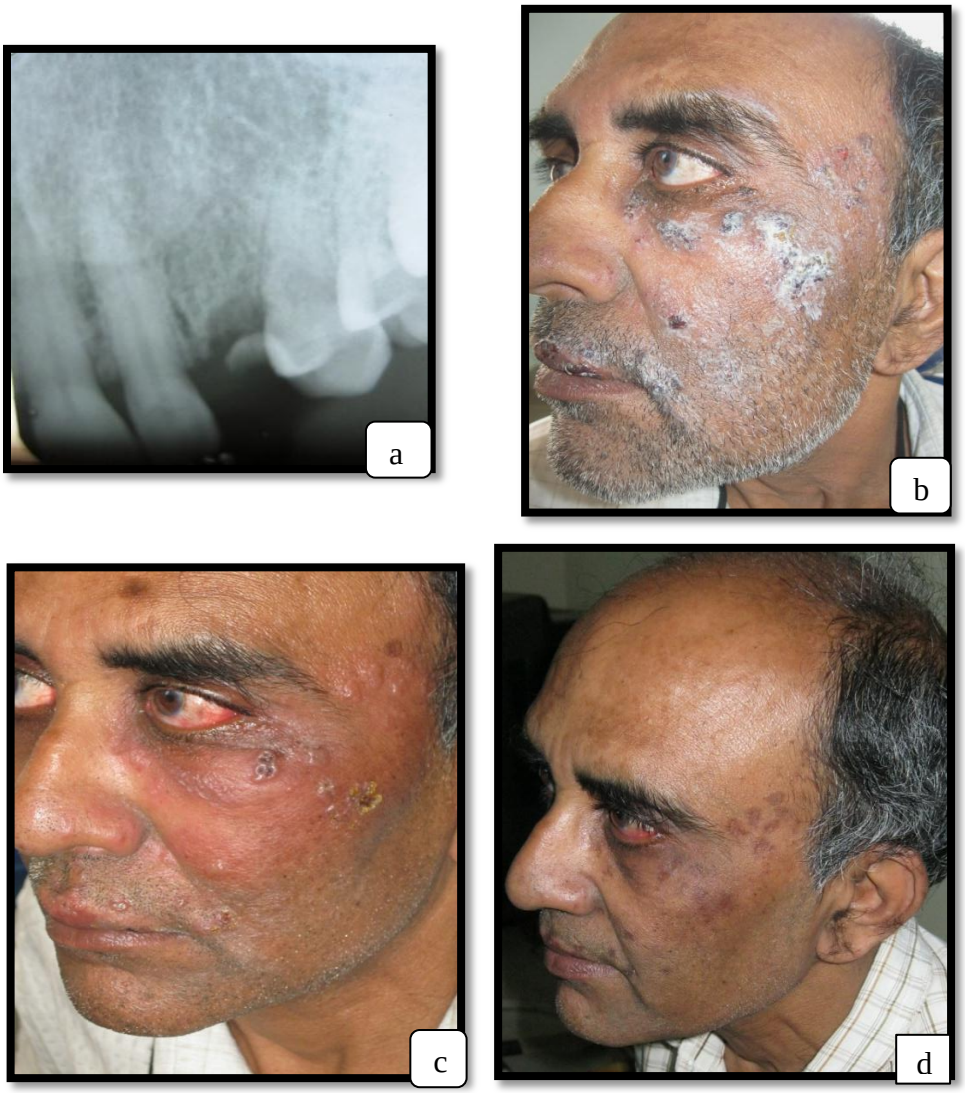


Figure: a) Intra oral periapical radiograph of 23 region showing root stumps, b) photograph on Day 2, c) photograph on Day 7 and d) photograph after 1 month

Since the clinical features were characteristic i.e. unilateral and localized dermatomal presentation of the vesicles, so we made the final diagnosis of herpes zoster of maxillary branch of trigeminal nerve and patient was educated about the viral, self-limiting and contagious nature of the disease and that the disease can spread to other family members and even school children, so necessary precautions needs to be followed, and was given symptomatic treatment and was put on acyclovir 800 mg five times a day for a period of 2 weeks and topical acyclovir to be applied over the lesion. Patient was referred for ophthalmic consultation for increased redness which was present in his left cornea, and ophthalmologist told that it was corneal inflammation due to local conditions, and patient was kept on follow up. After 15 days patient was relieved of the symptoms and at one month follow up, patient was totally free of any symptoms. Although the lesions healed with scarring, no post therapeutic complications were reported.

## DISCUSSION

The majority of HZ infections involve the thoracic and lumbar dermatomes; however, approximately 13% of patients present with infections involving any of the three branches of the trigeminal nerve (4). The ophthalmic branch is most commonly affected; however, in our case only the maxillary branch is involved; this is rare (1.7% of cases) (5). A case similar to our case i.e. maxillary nerve involvement with prodromal tooth pain has been reported in literature (6). Reactivation of varicella zoster virus(VZV) may occur spontaneously or when host defenses are compromised. Increased age (7), physical trauma (8) (including dental manipulation) (9,10), psychological stress (7,8), malignancy (7), radiation therapy and immunocompromised states including transplant recipients, immunomodulatory therapy and HIV (11) infection are predisposing factors for VZV reactivation. There are 1.5-3 cases of herpes zoster infection (HZI) per 1,000 subjects; this increases to 10 per 1,000 in those over 75 years (12). In our patient, age may be the contributing factor. Patient also reported increased stress at work place as he was a teacher and examinations were going in his school so that also could be traced as one of the predisposing factors.

Patients with HZ may progress through three stages: prodromal stage, active stage (also called acute stage) and chronic stage (13). The prodromal stage presents as sensations (described as burning, tingling, itching, boring, prickly) occurring in cutaneous distribution of the dermatome and is believed to represent viral degeneration of nerve fibrils.[2] During this period, if branches of the trigeminal nerve are affected, odontalgia and pulpal necrosis may occur. For the latter, it is proposed that the reactivated virus may travel the length of the nerve, infect the pulp vasculature lead to infarction and necrosis. Furthermore, these symptoms may present up to 0ne month before the acute mucocutaneous lesion, and pose significant diagnostic difficulties. The active stage is characterized by the emergence of the rash which is nearly always accompanied by systemic upset. The characteristic skin rash progresses from erythematous papules and oedema to vesicles and finally to pustules within one to seven days which dry and crust and are exfoliated over two to three weeks leaving erythematous macular lesions that may scar. Diagnostic difficulties may be encountered when the vesicular rash does not occur (zoster sine herpete) (2). Surprisingly, pain is reported to subside when the rash is most active; however, it returns during the crusting and scale phase until the rash clears. It is during the active or ‘eruptive’ phase that HZ is at its most contagious and could pose a significant cross infection risk. In this particular case risk of cross infection to students was significant so patient was advised to take leave from school. The chronic stage is only seen in approximately 10% of all patients with HZ, and is termed post-herpetic neuralgia (PHN). It is defines as pain that lingers for 30 days or 120 days (14) after the onset of the acute rash and described as a brief recurrent shooting or shocking allodynia, with a constant, usually deep pain, lasting beyond the period of healing of the active skin lesions. It may persist for years and is a significant cause of morbidity. PHN affects 8–70% of patients over age 50 and upto 50% of patients over age of 50 have debilitating pain lasting more than 1 month (15). Although post-herpetic neuralgia is the most common complication of HZ, other complications include neurological disorders like meningo-encephalitis or aseptic meningitis; ophthalmologic like uveitis or keratitis; cutaneous like bacterial

superinfection or cellulitis; and visceral complications like bronchitis or pleuritis; immunocompromised individuals with HZ exhibit a significantly higher rate of complications. Periapical lesions, root resorption, (16) tooth exfoliation and alveolar osteonecrosis, (17) have also been reported in association with HZ infection. Although HZ is a self-limiting condition and resolution is usually complete, treatment is indicated in some cases to reduce the acute symptoms of pain and malaise, to limit the spread and duration of the skin lesions and to prevent complications. The pharmacological approach is based on symptomatic relief and antiviral therapy. For many years, acyclovir (ACV)( 800 mg five times a day) (18) has been the drug of choice for the treatment of VZV infections. Recently, other antiviral agents such as valacyclovir(1000 mg three times a day) and famciclovir (500 mg three times a day) (15) have been developed to overcome the low oral bioavailability of ACV and its limited and less predictable effect in preventing the development of post-herpetic neuralgia, as well as to provide a more favourable dosage regime. Antiviral therapy should be initiated as early as possible, especially when patient factors that may complicate the manifestations of the condition are expected. In our case, since patient was already on anticonvulsants and also the treatment for herpes zoster was started within 72 hrs of the appearance of symptoms, there was no need for any prophylactic treatment for post herpetic neuralgia.

In conclusion, a case of HZ affecting the second division of trigeminal nerve is reported. This case highlights the importance of a thorough dental history and examination in patients with odontalgia as in our case. In those presenting with atypical odontalgia, HZI should be considered in the differential diagnosis. Furthermore, special consideration must be given to patients who are immunocompromised, although in our case the patient was immunocompetent but we could consider some decrease in immunity due to his age. Clinicians are urged to recognize the early features of HZI and to provide prompt antiviral therapy to prevent the complications.

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