ORO-FACIAL HERPES ZOSTER: A CASE REPORT WITH A DETAILED REVIEW OF LITERATURE

D.A.Vineet[1] R.Mithra[2] Pavitra Baskaran[2] Satyaranjan Mishra[3]

Department of Oral Medicine & Radiology, [1]PMS College of Dental Sciences and Research, Trivandrum,, Kerala. [2]SRM Kattankulanthur dental college, SRM Nagar, Kattankulathur , Kancheepuram District, Tamil Nadu. [3]SCB Dental College, Cuttack, Odisha.

Corresponding Author: D.A.Vineet, Department of Oral Medicine & Radiology,PMS Dental College of Dental Sciences and Research, Thiruvananthapuram, Kerala.India.

Abstract

Herpes zoster or shingles is a reactivation of the Varicella zoster virus that entered the cutaneous nerve endings during an earlier episode of chicken pox, travelled to the dorsal root ganglia, and remained in a latent form. Nerves most commonly involved are C3, T5, L1, L2 and first division of trigeminal nerve. The condition is characterized by occurrence of multiple, painful, unilateral vesicles and ulceration which shows a typical single dermatome involvement. The infection usually affects elderly individuals, and if present in the younger age group, immune-compromised status such as HIV/AIDS may be suspected. In this case report we present a patient with herpes zoster involving the maxillary and mandibular divisions of the trigeminal nerve, with unilateral vesicles over the left side of lower and middle 1/3rd of face along the trigeminal nerve tract, with intraoral involvement of buccal mucosa, labial mucosa and the palate of the same side.

Key Words: Herpes zoster, Shingles, Unilateral vesicular lesions, Trigeminal nerve

Introduction

Varicella zoster virus is a ubiquitous; DNA virus which belongs to the subfamily of human alpha herpes virus.[1]The association between varicella and herpes zoster was first made in 1892. It was later recognized that the pathologic changes of herpes zoster 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. It is now well established that a herpes zoster infection (shingles) requires pre-exposure to the varicella zoster virus. The primary varicella virus infection causes an acute, generally mild infection (Chicken pox) and the virus subsequently establishes latency elsewhere within the sensory ganglia. The virus is then later reactivated to cause a herpes zoster (HZ) infection. [2] Zoster probably results most often from a failure of the immune system to contain latent virus replication. Whether other factors such as radiation, physical trauma, medications, other infections, or stress can also trigger zoster has not been determined with certainty. Nor is it entirely clear why circulating varicella antibodies and cell-mediated immune mechanisms does not prevent recurrent overt disease, as is common with most other viral illness.[3] As Herpes zoster virus outbreak is commonly characterized by easily observed vesicular skin eruptions that follow the anatomic distribution of affected nerve or nerve branch. A few cases have even been reported without vesicular eruption, making diagnosis difficult.[2] Herpes Zoster infection of the maxillary branch of the trigeminal nerve produces vesicles on the palate, uvula and the tonsils, while in case of the involvement of the mandibular division; the vesicles appear on the anterior part of tongue, the floor of the mouth and buccal mucosa. In oro-facial herpes zoster, toothache may be the presenting symptom.[4]

Oral manifestations of herpes zoster appear when the mandibular and maxillary divisions of the trigeminal nerve are affected.[5] Osseous and dental manifestations such as devitalized teeth, internal resorption, abnormal development of permanent teeth, spontaneous exfoliation of teeth and necrosis of maxilla and mandible have been reported.[6] Herpes zoster affecting the oral and maxillo-facial region may pose a significant diagnostic challenge and should be considered in the differential diagnosis of those presenting with atypical odontalgia.[7] Prompt management is required, especially in immune compromised individuals, to prevent complications, which may cause significant morbidity.[8]

Case Report

A 53 year old male patient came to the dental hospital with the complaint of ulcerations over the left side of the face and mouth since 3 days. History revealed that the patient had fever and severe throat infection a week ago. Then he had burning sensation in the left side of the face as well as in the oral cavity. Gradually vesicles appeared 4 days back and then those vesicles ruptured to form ulcers which were very painful. All the ulcers were limited to the face and oral cavity of the left side only.

Medical history was non contributory except for the fact that the patient suffered from chicken pox in the childhood.

On examination multiple irregular shallow ulcerations and crusts are seen on the lips and the peri-oral skin on the left side of the face not crossing the midline. [Fig 1& 2]

Intra-orally multiple shallow ulcerations, with erythematous irregular borders with tissue tags are seen on the buccal mucosa, palate and the labial mucosa unilaterally on the left side.[Fig 3& 4] No dysphagia or odynophagia was reported. There were no other skin lesions accompanying the oro-facial lesions.

Investigations included Tzanck smear, which revealed multi-nucleated giant cells. Serum immunoglobulin levels, herpes simplex virus (HSV) antigen detection and viral culture were not done due to lack of facilities. The patient was prescribed oral acyclovir (800 mg five times a day for 10 days), with antihistaminic-anaesthetic mouth rinses and healing of lesions can be seen after 5 days of the treatment. [Fig 5-8]

The patient showed remarkable improvement in the lesions and had shown no signs of recurrence in 3 months follow up period.

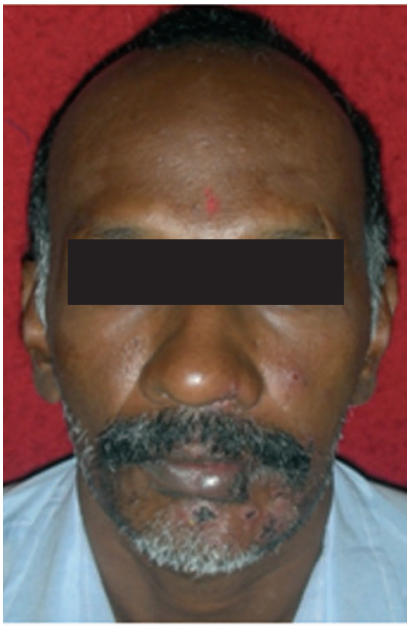

Fig 1 Clinical photograph

Fig 2 Clinical photograph

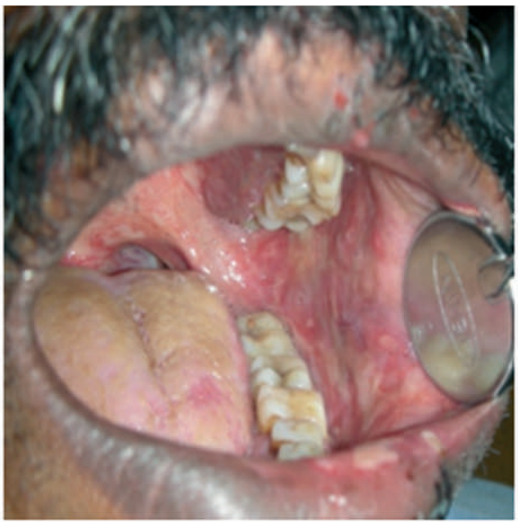

Fig 3 Photograph showing ulcers

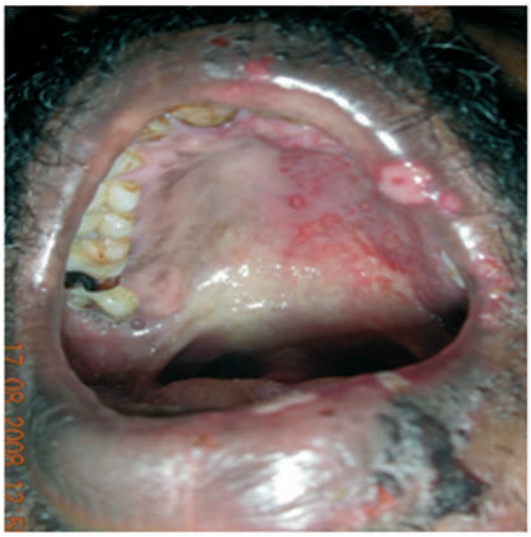

Fig 4 Photograph showing ulcers

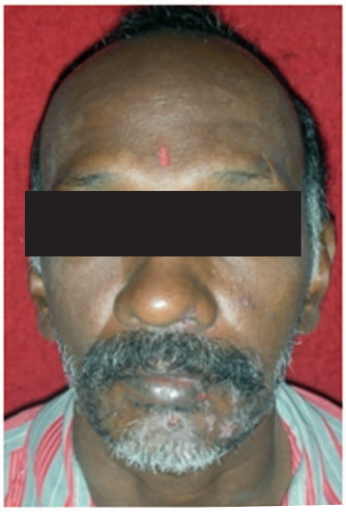

Fig 5 Post treatment Clinical photograph

Fig 6 Post treatment Clinical photograph

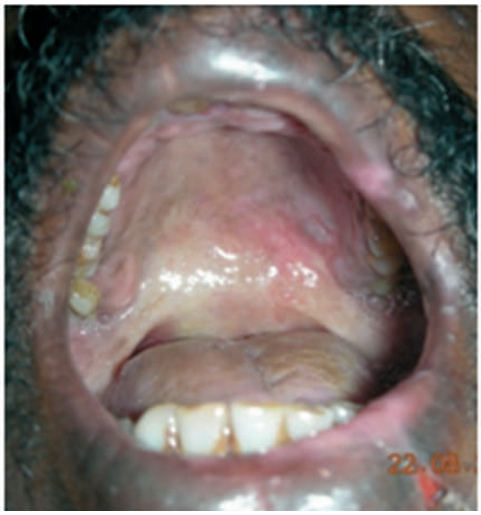

Fig 7 Intra oral photograph after treatment

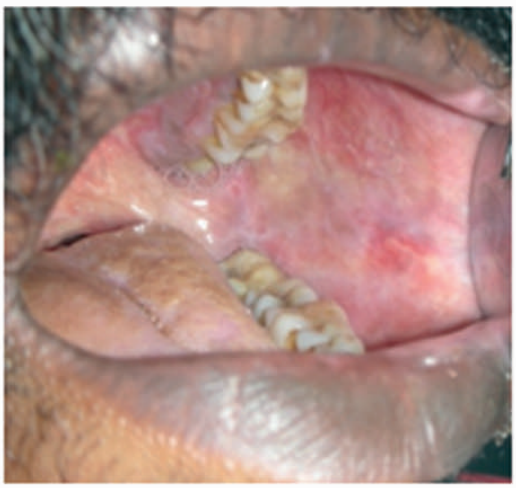

Fig 8 Intra oral photograph after treatment

Discussion

Varicella zoster (VZV) is a herpes virus, and, like other herpes viruses, it causes both primary and recurrent infection and remains latent in neurons present in sensory ganglia. Varicella zoster virus is responsible for two major clinical infections of humans: chicken pox (varicella) and shingles (herpes zoster). Chicken pox is a generalized primary infection that occurs the first time an individual contacts the virus. After the primary disease is heals, varicella zoster virus remains latent in the dorsal root ganglia of spinal nerves or extra medullary ganglia of cranial nerves. A child without prior contact with varicella zoster virus can develop chicken pox after contact with an individual with Herpes zoster. In 3-5 of every 1000 individuals, varicella zoster virus becomes reactivated, causing lesions of localized herpes zoster. The incidence of herpes zoster increases with age or immunosuppression.[9] Herpes zoster is more commonly known as shingles, from the Latin cingulum, for "girdle". This is because a common presentation of herpes zoster involves a unilateral rash that can wrap around the waist or torso like a girdle. Similarly, the name zoster is derived from classical Greek, referring to a belt-like binding (known as a zoster) used by warriors to secure armour.[10]Reactivation of VZV may occur spontaneously or when host defences are compromised. Increased age,[2,11-13] physical trauma,[11,12,14] (including dental procedures), psychological stress,[2,11,12,14] malignancy,[11] radiation therapy[14] and immune-compromised states including transplant recipients, steroid therapy and HIV infection[15,16] are predisposing factors for VZV reactivation. Herpes zoster can affect any sensory ganglia and its cutaneous nerve.[17] Most of the infections affect dermatomes of T-3 to L-3 but about 13% of the patients present with infections involving any of the three branches of the trigeminal nerve.[18]

Epidemiology

Herpes zoster is a sporadic disease with an estimated life time incidence of 10-20%. The incidence of herpes zoster is upto 15 times higher in HIV infected patients than in uninfected patients and as many as 25% of patients with Hodgkin's lymphoma develop herpes zoster. Household transmission rates have been noted to be approximately 15%.[19] The incidence of herpes zoster infection in the general population has been reported to be 5.4%.[5,20] Herpes zoster infection typically occurs in individuals older than 45 years of age, with the highest incidence among persons 60-90 years old.[21]

Clinical Manifestations

Patients with herpes zoster infections usually progress through three stages: (1) prodromal stage, (2) active stage (also called acute stage), and (3) chronic stage.[17, 22] However, some patients do not develop symptoms of all stages. 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.[23]The prodromal syndrome stage presents as sensations described as burning, tingling, itching, boring, prickly or knife-like occurring in the skin over the affected nerve distribution. It is believed that these sensory changes are a result of degeneration of nerve fibrils from viral infection activity. This usually precedes the rash of the active stage by a few hours to several days.[17, 18, 22]The patient may present with an odontalgia that may be the only prodromal symptom.[23]

The active stage is characterized by the emergence of the rash that may be accompanied by generalized malaise, headache, low grade fever, and sometimes nausea. The rash progresses from erythematous papules and oedema to vesicles in 12-24 hours and finally progresses to pustules within 1-7 days. The pustules begin to dry with crust formations that fall off in 14-21 days, leaving erythematous macular lesions that result in hyper-pigmented or hypo-pigmented scarring. In severe cases, areas of epidermis and variable amounts of dermis

may be lost due to haemorrhagic necrosis.[17, 22] Intra-oral lesions usually appear after the cutaneous rash. Pain and dysaesthesia during the active stage are reported to be minimal when the rash is most active. However, there is a return of pain during the crusting phase of the active stage but this pain subsides as the crusts clear.[17]

The chronic pain syndrome stage is termed post herpetic neuralgia (PHN). PHN is defined as pain lasting beyond the period of healing of the active skin lesions. This has been described as pain lasting 1-3 months after the skin lesions have cleared but may in fact last for years and decades.[17, 22] PHN pain has been described as pain consisting of three distinct components: (i) a constant, usually deep pain; (ii) a brief recurrent shooting or shocking tic-like pain; and (iii) a sharp radiating dysaesthetic sensation evoked by very light touching of the skin, termed allodynia.[24]

Oral Manifestations

Herpes zoster involves one of the divisions of the trigeminal nerve in 18-20% of cases, but the ophthalmic branch is affected several times more frequently than are the second or third divisions. Herpes zoster of the first division can lead to blindness secondary to corneal scarring and should be managed by an ophthalmologist. Facial and intraoral lesions are characteristic of Herpes zoster involving the second and third divisions of the trigeminal nerve. The patient in our case showed involvement of all the three branches of trigeminal nerve. Vesicular eruptions with erythema was seen involving the nose, upper lip, lower lip, zygoma, malar area, temporal region and forehead, along with intraoral lesions of one of the trigeminal nerve branch. Each individual lesion of herpes zoster resembles lesions seen in herpes simplex infections. The diagnosis is based on a history of pain and the unilateral nature and segmental distribution of the lesions. When the clinical appearance is typical and the vesicles are present, oral herpes zoster can be distinguished clinically from other acute multiple lesions of the mouth, which are bilateral and not preceded or accompanied by pain along the course of one trigeminal nerve branch.

Herpes zoster has been associated with dental anomalies and severe scarring of the facial skin when trigeminal herpes zoster occurs during tooth formation. Pulpal necrosis and internal root resorption have also been documented with herpes zoster. In immune-compromised patients, large chronic herpes zoster lesions have been described that have led to necrosis of underlying bone and exfoliation of teeth.9 Schwartz and Kvoring reported 10 cases of herpes zoster with post herpetic complications including osteonecrosis of jaw, exfoliation of teeth, severe periodontitis and scarring of the skin.25 Wadden reported a 70 year old woman with a history of excellent oral health, who within 3 years of an attack of Shingles affecting the maxillary division of the left trigeminal nerve had multiple devitalization of four of the five teeth in the left maxillary quadrant suggesting a central source of injury rather than a local cause.[26]

Complications

(A) Acute complications- Cutaneous varicella zoster dissemination [27], Bacterial superinfection, Zoster gangrenosum, Zoster haemorrhagicus, Septicemia,Meningo-encephalitis, Aseptic meningitis, Cranial and Peripheral nerve palsies, Conjunctivitis, Episcleritis, Uveitis, Keratitis, Secondary glaucoma, Acute renal necrosis, Loss of corneal sensations, Optic neuropathy, Ptosis, Mydriasis, Neural Bronchitis, Pleuritis, Esophagitis, Gastritis/enterocolitis, Peritonitis, Pericarditis, Pneumonia, Hepatitis, Myocarditis, Arthritis

(B) Chronic complications- Scar formation (atrophic scars, hypertrophic scars), Hypo/depigmentation, Post herpetic neuralgia (PHN), Guillain-Barre syndrome, Autonomic dysfunction, Granulomatous cerebral angiitis, Diaphragmatic paralysis, Bladder dysfunction, Sensory loss/deafness, Chorio-retinitis, Atrophy of optic nerve,

Progressive outer retinal necrosis.

Development of herpes zoster at cranial region has special importance. Involvement of cranial nerves has high complication risk. The Trigeminal has three branches; ophthalmic, maxillary, mandibular. In ophthalmic zoster the eye is affected in two-thirds of cases, especially when vesicles on the side of the nose indicate involvement of the nasociliary nerve (Hutchinson's sign).[28] Hutchinson's sign is a powerful predictor of ocular inflammation and corneal denervation. Involvement of the ciliary ganglia may give rise to Argyll-Robertson pupil.[28; 29] The facial nerve (seventh cranial nerve) is then involved. When just the external ear is affected, it is named as herpes zoster oticus.[30, 31] Pressure on the facial nerve motor fibres may evolve into facial palsy. The Vestibulocochlear nerve and the Facial nerve involvement complete the classical triad of the Ramsay Hunt syndrome; herpes zoster of external ear or tympanic membrane, ipsilateral facial paralysis and auditory symptoms.[32, 33] Compression of the vestibulocochlear nerve may cause auditory symptoms which are sensorineural hearing loss, tinnitus, dizziness and vertigo. Involvement of the nervus intermedius or its geniculate ganglion would impair taste sensation from the anterior two-thirds of the tongue and alter lacrimation. [29, 34] Herpes zoster oticus accounts for about 10% of cases of facial palsy. The paralysis is usually complete and full recovery occurs in only about 20% of untreated cases. There is permanent hearing loss in about one-third of these patients.

Diagnosis

Initial diagnosis poses a challenge during the prodromal stage of the disease, which typically lasts 1-2 days but can persist up to three weeks before the appearance of skin lesions.[19] Furthermore, some individuals may only present with prodromal symptoms, never developing the telltale rash. This phenomenon is known as "zoster sine herpete". Pain can be misdiagnosed as appendicitis, myocardial infarct, renal colic, cholelithiasis, or colitis, depending on its intensity and the location of the affected nerve.[35] Common differential diagnoses at this stage include pleurisy, cardiac disease, herniated nucleus pulposus, trigeminal neuralgia, and Bell's palsy.[19, 35] An appropriate diagnosis of HZ is aided by the appearance of a vesicular rash with characteristic distribution. When the presentation of skin lesions is not as clear, as may be the case with immunocompromised patients, laboratory confirmation is recommended. The polymerase chain reaction (PCR) technique is the most sensitive and specific diagnostic test, as it can detect VZV DNA in fluid from the vesicle. Availability of the PCR technique, however, may pose a challenge.[36] Viral culture is possible but typically has low sensitivity. VZV is labile, resulting in difficult recovery of an adequate sample from vesicular fluid. Use of direct immunofluorescence assay is a good alternative to PCR. It is preferred over viral culture, as it is more sensitive, of lower cost, and offers a more rapid turnaround time. In the present case, Tzanck smear showed the presence of multi-nucleated giant cells.

Prevention And Treatment

The varicella zoster virus Oka strain vaccine is currently recommended by the Advisory Committee on Immunization Practices for universal childhood vaccination in USA. The vaccine increases cytotoxic lymphocyte responses specific for varicella zoster virus in seropositive elderly people.[37] Early diagnosis and prompt treatment of the disease in the prodromal phase by the use of anti-viral agents should probably be the mainstay of its management. The treatment of herpes zoster has three main objectives: (1) treatment of the acute viral infection, (2) treatment of the acute pain associated with herpes zoster and (3) prevention of post herpetic neuralgia. Anti-viral 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 demonstrated in patients who received antiviral agents within

72 hours after the onset of the rash.[19] The recommended dosages of anti-viral agents used in the management of herpes zoster infection are:-

1. Acyclovir: 800 mg orally five times daily for 7-10 days, or 10 mg per kg IV every 8 h for 7-10 days
2. Famciclovir: 500 mg orally three times daily for 7 days
3. Valacyclovir: 1 g orally three times daily for 7 days
4. Brivudin 125 mg once daily for 7 days

Some newer molecules which are under development and are found to be highly potent are still on clinical trials and yet to be approved[38,39]

Newer medications for herpes zoster.

1. CMX 001 Hexadecyloxypropyl-cidofovir
2. Valamaciclovir Nucleoside analogue (H2G)
3. ASP2151 Helicase primase inhibitor
4. FV100 Two bicyclic nucleoside analogues (BCNA)

Although PHN is generally a self limiting condition, it can last indefinitely. Treatment is directed at pain control while waiting for the condition to resolve.

Treatment options for post herpetic neuralgia.

(1) Topical agents
   (a) Capsaicin cream: Application to the affected area 3-5 times daily
   (b) Lidocaine (xylocaine) patch: Application to the affected area every 4-12 hours or as needed

(2) Tri-cyclic anti-depressants
   (a) Amitriptyline 25 mg orally at bedtime; increase dosage by 25 mg every 2-4 weeks until response is adequate, or to maximum dosage of 150 mg per day
   (b) Nortriptyline 25 mg orally at bedtime; increase dosage by 25 mg every 2-4 weeks until response is adequate, or to maximum dosage of 125 mg per day
   (c) Impramine 25 mg orally at bedtime; increase dosage by 25 mg every 2-4 weeks until response is adequate, or to maximum dosage of 150 mg per day
   (d) Desipramine 25 mg orally at bedtime; increase dosage by 25 mg every 2-4 weeks until response is adequate, or to maximum dosage of 150 mg per day

(3) Anti convulsants
   (a) Phenytoin 100-300 mg orally at bedtime; increase dosage until response is adequate, or blood drug level is 10-20 mg/ml
   (b) Carbamazepine 100 mg orally at bedtime; increase dosage by 100 mg every 3 days until dosage is 200 mg three times daily, or till response is adequate or blood drug level is 6-12 mg/ml
   (c) Gabapentin 100-300 mg orally at bedtime; increase dosage by 100-300 mg every 3 days until dosage is 300-900 mg three times daily or response is adequate.

The use of systemic corticosteroids to prevent post herpetic neuralgia in patients over 50 years of age is controversial. Recent review of the data indicated a reduction of pain and disability during the first two weeks but no effect on the incidence or severity of PNH. Some clinicians advocate the use of a combination of intra-lesional steroids and local anaesthetics to decrease healing time and prevent PNH, but a controlled study of this therapy has not been performed.[9]

Conclusion

Herpes zoster infection may be infrequently encountered in general dental practice, however many patients do report to the dental clinic with the complications of HZV infection, involving the trigeminal nerve in about 15% cases. Diagnosing these complications of herpes zoster could pose a challenge to an oral physician due to their varied presentation ranging from post herpetic neuralgia, external root resorption, osteonecrosis and tooth exfoliation. Especially the elderly and the immune compromised patients are susceptible and hence burning sensation, pain, vesiculation, ulceration along the course of the Trigeminal nerve should be treated with HZV infection in mind.

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