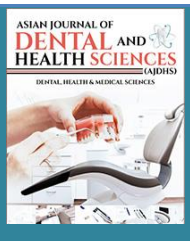

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Multidermatomal Herpes Zoster of the trigeminal nerve in an immunocompetent patient: a case report

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Article Info:

Article History:

Received 09 December 2023
Reviewed 11 January 2024
Accepted 06 February 2024
Published 15 March 2024

Cite this article as:

Sun CE, Sanusi S, Multidermatomal Herpes Zoster of the trigeminal nerve in an immunocompetent patient: a case report, Asian Journal of Dental and Health Sciences. 2024; 4(1):1-3
DOI: http://dx.doi.org/10.22270/ajdhs.v4i1.67

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Abstract

Herpes Zoster is a neurocutaneous viral infection caused by the reactivation of the Varicella Zoster Virus in the dorsal root ganglion. It is characterized as vesicular rash along a unilateral dermatome, usually associated with pain or paresthesia of the involved area. Multidermatomal involvement is rare in immunocompetent patients. We report an unusual case of Herpes Zoster involving the maxillary(V2) and mandibular(V3) branches of the trigeminal nerve in a healthy immunocompetent lady.

Keywords: herpes zoster, neurocutaneous viral infection, trigeminal nerve

Case Presentation

A 25-year-old lady presented with vesicular lesions with crusts on left side of face for 3 days associated with left sided facial pain and burning sensation. The lesions began in the left upper lip and progressed within the span of 3 days to involve the left cheek and chin area, which further spread to involve intraorally. There was no facial paralysis, hearing loss or visual symptoms. She had no constitutional symptoms. Her past medical history was unremarkable.

General examination of the patient was normal and her vitals were stable. Cutaneous examination revealed edema over the left side of the face. Multiple grouped maculopapular and vesicular lesions were seen over the left lower eyelid, cheek, external ear, upper and lower lip, and chin. [Fig 1,2] Intraoral examination revealed similar lesions involving left side of hard and soft palate and left buccal mucosa. [Fig-3] Ear examination revealed vesicles over left pinna. Otherwise, external auditory canal and anterior part of the tympanic membrane was normal with normal hearing assessment. Ophthalmic examination was unremarkable. Facial nerve was intact. The affected areas corresponded to the distribution of the left maxillary(V2) and mandibular(V3) branches of the trigeminal nerve. Complete blood count, serum electrolytes, renal function and blood sugar were within normal limits.

The patient was started on intravenous Acyclovir 400mg TDS for 5 days, followed by T. Acyclovir 400mg QID for another 5 days. Topical Acyclovir cream, mouth rinse and oral analgesics were also prescribed. The patient showed gradual recovery with healing of lesions 2 weeks after onset of symptoms.

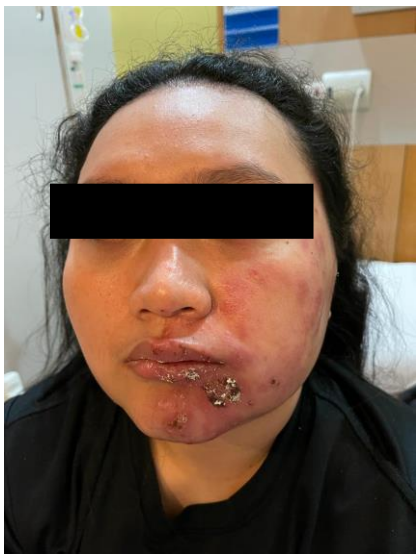

Figure 1

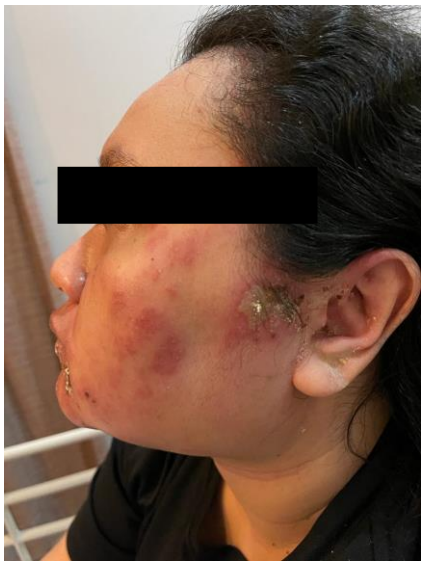

Figure 2

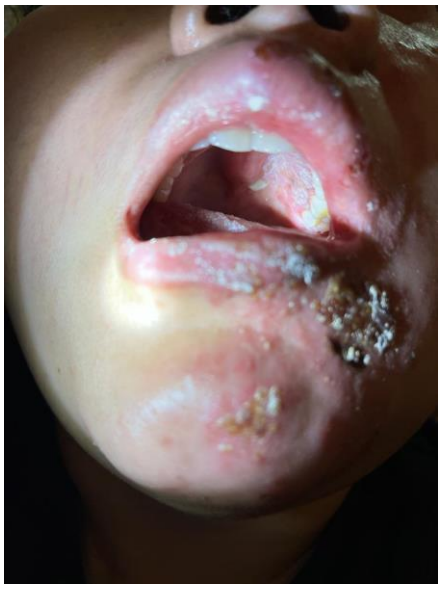

Figure 3

Discussion

Herpes Zoster (HZ), also known as Shingles, is a common viral disease caused by reactivation of a latent infection of Varicella Zoster Virus(VZV), and alpha herpes virus. After an initial infection, the virus remains latent in the perineural satellite cells of the dorsal nerve root ganglion.[1,2] The frequency of zoster in thoracic dermatomes is 53%, cranial nerves is 20%, cervical dermatomes is 4-20%, followed by lumbosacral 11%.[3] In HZ, a few lesions can normally appear adjacent to the affected dermatome. More extensive skin involvement of several adjacent dermatomes is known as multidermatomal zoster.[4] The ophthalmic branch(V1) of the trigeminal nerve is affected about 20 times more often than maxillary(V2) and mandibular(V3) branches of the nerve. The maxillary nerve(V2) is the least frequently affected branch. It is unusual for zoster to involve maxillary(V2) or mandibular(V3) divisions without ophthalmic(V1) involvement. Only anecdotal reports exist about HZ affecting two or three branches of trigeminal nerve.[2,5] In our patient, the lesions appeared in the area of maxillary(V2) and mandibular(V3) nerve distribution, sparing the ophthalmic(V1) nerve. In contrast to immunodeficient patients, multidermatomal involvement in HZ is rarely observed in immunocompetent patients.[2] Our case represents a rare example of involvement of multidermatomal distribution of HZ in a healthy, immunocompetent young lady.

It has been stipulated that HZ is triggered in part by a decrease in immunity. Stress and depressive symptoms are also identified as possible trigger of HZ. In conjunction with other factors such as age, nutritional status and underlying comorbids, stress and psychological symptoms may contribute to a lowering of immunity.[6] HZ occurs in three successive stages, namely prodromal, acute and chronic neuropathic stage.[1] The prodromal (pre-eruptive stage) presents as pain or dysesthesia over the involved nerve distribution associated with mild fever. The acute stage is characterized by appearance of an initial erythematous macular lesion involving unilateral dermatome, that progress into vesiculopapular phase that appears within 1-2 days and continues to erupt over another 3-4 days.[1,3] Intraoral lesions, when maxillary or mandibular division of trigeminal nerve is involved, typically develop after cutaneous rash. HZ involving the maxillary nerve (V2) typically present with lesions over middle third of the face, lower eyelid, side of the nose and upper lip, buccal mucosa and palate; while mandibular nerve (V3) involvement is evidenced by lesions over the lower third of face, lower lip, temporal region and intraoral mucosa. The distribution of vesicles in our patient correlated with involvement of maxillary(V2) and mandibular(V3) nerves. Chronic neuropathic pain, also known as post-herpetic neuralgia, involves around 30% of patients with HZ. It is defined as a sharp, intense, radiating pain lasting after eruptive stage for about one to three months.[7]

In around 16% of patients with HZ, the rash disseminates beyond one dermatome, especially in elderly and immunocompromised individuals. Only a few cases of multidermatomal HZ have been reported in the literature, with only a handful number among them occurred in healthy immunocompetent individuals.[3] Among immunocompetent patients, HZ is considered a self-limiting, localized infection. Life-threatening and debilitating complications occur almost exclusively in immunocompromised patients. These complications include encephalitis, herpes zoster ophthalmicus, retinitis, delayed contralateral hemiparesis, stroke, and myelitis.[4]

Early diagnosis and prompt treatment of HZ is the mainstay of treatment. Antiviral drugs are best administered within the viral replication period, which is ≤72 hours after onset of rash. Topical antiviral agents such as Acyclovir cream and docosanol cream are also effective. Recommended duration of antiviral is 7-10 days. Antiviral therapy was shown to be effective in shortening the viral shedding period, reducing zoster-associated pain severity and duration, preventing new lesion formation, hastening the healing of skin lesions, and reduction in incidence of postherpetic neuralgia.[1,5] When vesicles postulate, patients are at risk of secondary bacterial infection, common organisms being Staphylococcus aureus or Streptococcus pyogenes. In secondary infection, oral antibiotic is appropriate.[8]

A step-wise approach should be taken to treating acute neuralgia and post-herpetic neuralgia, according to the severity of the patient's symptoms. Topical analgesics such as capsaicin cream and topical lidocaine patch can be used for mild acute neuralgia. Tricyclic antidepressants, opioid analgesics and gabapentin are commonly used in managing post herpetic neuralgia. Gabapentin has been shown to significantly reduce the duration of post-herpetic neuralgia.[9] In the recent years, the use of vitamin C appears to be an emerging treatment for attenuating acute neuralgia and post herpetic neuralgia by modulating serum levels of cytokine IL-6 and IL-8.[10]

Overall incidence rate of HZ is 5.1 per 1000 person years. HZ generally is considered to occur only once in a lifetime with recurrence limited to immunocompromised individuals. However, recurrence rate of 12.0 per 1000 person years among immunocompetent persons has been reported.[11] Mean time

between initial and recurrent HZ episodes was 1063 days. Risk factors for recurrence are elderly age group, women, longer duration of post-herpetic immunocompromised status, and comorbid conditions.[11] Studies have demonstrated that immunization of immunocompetent older individuals with live attenuated Varicella-Zoster-Virus vaccine reduced the incidence and severity of HZ and postherpetic neuralgia. In the USA and Canada, the vaccine is recommended for people aged ≥60 years, whereas in Europe and Australia the vaccine is approved for adults aged ≥50 years; inclusive of those who have a previous HZ episode and those with underlying chronic medical conditions.[12]

Our patient was treated with intravenous acyclovir, which was converted to oral for a total of 10 days. Topical acyclovir cream was also prescribed, along with analgesics and mouth rinse for symptomatic relief. The vesicles crusted and healed within 5 days of initiation of treatment.

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