



HERPES ZOSTER OF RIGHT MAXILLARY DIVISION OF TRIGEMINAL NERVE ALONG WITH ORAL MANIFESTATIONS IN A 46 YEAR OLD MALE

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ABSTRACT

Herpes zoster in the distribution of the maxillary and mandibular divisions of the trigeminal nerve is characterized by painful vesicular eruptions of the skin and oral mucosa in the distribution of the affected nerves. Oral complications may occur, including post-herpetic neuralgia, devitalisation of teeth, and abnormal development of permanent teeth, root resorption and periapical lesions. This article reports a case of 35-year-old man with herpes zoster infection of the right maxillary division of trigeminal nerve showing manifestations in middle one third of face & oral cavity.

KEYWORDS: Herpes Zoster, Varicella Zoster, Trigeminal nerve, Hard Palate.

INTRODUCTION

Herpes zoster also known as shingles is an acute infectious viral disease of extremely painful and incapacitating nature which is characterized by inflammation of dorsal root ganglia or extra medullary cranial nerve ganglia, associated with vesicular eruptions of the skin or mucous membrane in an area supplied by the affected nerve^[1]. It results from reactivation of the varicella-zoster virus. It mainly affects distribution of the maxillary and mandibular divisions of the trigeminal nerve and is characterized by painful vesicular eruptions of the skin and oral mucosa in the distribution of the affected nerves. The most commonly affected dermatomes are the thoracic (45%), cervical (23%) and trigeminal (15%)^[2]. Oral complications may occur, including post-herpetic neuralgia, devitalisation of teeth, abnormal development of permanent teeth, root resorption, periapical lesions, and necrosis of the alveolar bony process and spontaneous exfoliation of teeth in few cases. Varicella-zoster virus (VZV), also known as Human Herpes Virus III (HHVIII), is a member of the herpes virus group. As all the other viruses from this group, VZV can manifest itself as a recurrent infection. After entering the body and causing primary infection, varicella-zoster virus remains latent in the neurons of sensory ganglion, especially dorsal roots of ganglion of the spinal nerves and extra medullary ganglion of the cranial nerves. Reactivation of the VZV infection is easily triggered by immune suppression. VZV infection is common in elder persons, immunocompromised or HIV positive individuals and patients affected by malignant blood dyscrasias, malignant tumours, or undergoing immunosuppressive therapy and radiotherapy. It is an anaerotropic virus; the possible provoking factors may be

the infection of the nerves innervating the periosteum or the chronic inflammatory changes in the form of adverse periodontal disease and delayed healing of the extraction sockets associated with compromised host resistance. Reports of herpes zoster infection of the fifth cranial nerve along with oral manifestations are scarce in literature. So we hereby report a case of herpes zoster infection involving right maxillary division of trigeminal nerve along with oral manifestations^[3].

Epidemiology

Varicella (chickenpox) is the primary infection and it is very common among children of both sexes. Herpes zoster is the recurrent form of infection and occurs in the 3-5% of population, mainly among older individuals and immunocompromised. One percent of the persons who are 80 years old may have an infection during the period of one year. In 10% of HIV positive patients, HIV disease starts with herpes zoster infection in the oral cavity as an oral opportunistic infection. Reactivation of infection is infrequent in younger people and children. Post herpetic neuralgia, a significant pain or dysaesthesia present 3 or more months after herpes zoster, approximately 10–20% of zoster patients of all ages are affected, but frequency increases with age^[4].

Etiopathogenesis

Following primary infection, the virus is latent in the neurons of the sensory ganglia and reactivates itself as a consequence of immunodeficiency. The inflammation of the ganglion is followed by hemorrhagic necrosis of the nerves together with a partial necrosis of the ganglion. VZV affects neighbouring neurone ganglia and it might affect several branches of the nerve. Viruses spreading through sensory

parts of the second and third branch of the trigeminal nerve, lead to the pathological changes in the oral cavity. The viral presence further leads to the acantholysis in the prickle cell of the epithelium and formation of the vesicles. Because of the subtle overlying layer, vesicles rupture rapidly, leaving erosions. VZV damages peripheral nerves through demineralisation, leading to sclerosis and degeneration^[5].

Case report

A 46 year old man reported in outpatient department of Oral Medicine, Diagnosis and Radiology in Institution of Dental Education & Advance Studies with chief complaint of swelling and pain with itching in right ala of nose & burning sensation of oral cavity since 5 days. Gradually 2-3 vesicles appeared 2 days back and then those vesicles ruptured to form ulcers which were very painful. These ulcers and vesicles were limited to the right ala of nose only (Fig 1). Medical history was non contributory except for the fact that patient suffered from chickenpox in the childhood. On

examination multiple grouped vesicles and little shallow ulceration on right ala of the nose not crossing the midline. Intraoral examination revealed multiple vesicles with erythematous patch on right half of dorsum of hard palate not crossing midline (Fig 2). No dysphagia and odynophagia was reported. There were no skin lesions accompanying the oro-facial lesions. Based on the clinical presentation a provisional diagnosis of herpes zoster infection of right maxillary division was made. Patient was immediately started on antiviral medications. He was given acyclovir 800 mg 5 times daily, tantum (.15% Benzyldiamine Hcl) & topical application of calamine lotion for skin lesions. He was instructed to report after week for follow up and after one and a half week patient reported with the improvement in lesion but not complete remission & was asked to continue medications for another week but patient did not turn up again.



FIGURE 1: Clinical picture of the patient showing few vesicles on right ala of the nose

Courtesy: Outpatient Department of Oral Medicine, Diagnosis & Radiology, Institute of Dental Education & Advance Studies



FIGURE 2: Clinical picture depicting vesicles on right half of dorsum of hard palate not crossing midline

Courtesy: Outpatient Department of Oral Medicine, Diagnosis & Radiology, Institute of Dental Education & Advance Studies

DISCUSSION

Varicella zoster is a ubiquitous DNA virus which belongs to subfamily of human alpha herpes virus. Herpes zoster is an acute viral infection characterized by vesicular skin lesions which are usually distributed over several unilateral adjacent sensory dermatomes. It causes chicken pox and then remains latent for decades in cranial nerve, dorsal root and autonomic nervous system ganglia along the entire neural axis. Herpes zoster infection can be of various types. Herpes zoster ophthalmicus involves the orbit of the eye and occurs in approximately 10-25% of cases. It is caused by the virus reactivating in the ophthalmic division of the trigeminal nerve. In a few patients, symptoms may include conjunctivitis, keratitis, uveitis, and optic nerve palsies that can sometimes cause chronic ocular inflammation, loss of vision and debilitating pain^[6]. Herpes zoster oticus, also

known as Ramsay Hunt syndrome type II, involves the ear. It results from the virus spreading from the facial nerve to the vestibulocochlear nerve. Symptoms include hearing loss and vertigo. Zoster is a common predominantly dermal and neurologic disorder caused by the VZV, a virus morphologically and antigenically identical to the virus causing varicella (chickenpox). The most frequent and debilitating complication of Herpes Zoster is post herpetic neuralgia (PHN), which is a form of neuropathic pain that appears in the dermatomes affected by the VZV infection. Difference in clinical manifestations between varicella and zoster apparently depends on the immune status of individual patients; those with no prior immunologic exposure to varicella virus, most commonly children, develop the clinical syndrome of varicella, while those with

circulating varicella antibodies develop a localized zoster [7]. Herpes zoster in the oral cavity results from the involvement of second and third branch of the trigeminal nerve. It develops 2-4 days after prodromal period, manifesting itself with general symptoms, such as fever, weakness, fatigue and neck stiffness. Paresthesia and burning sensation in the region of the affected nerve are also frequent consequences of the VZV infection. Characteristic sign of oral Herpes Zoster is the presence of unilateral vesicles that break rapidly, leaving small ulcers. On skin and lips, vesicle rupture can result in erosions covered by pseudo membranes and haemorrhagic crusts. Oral lesions without facial skin involvement are rather infrequent. Crusts and pseudo membranes, developing during the first week of vesicle formation, usually disappear in the second or third week. Zoster most commonly manifests in one or more posterior spinal ganglia or cranial sensory ganglia, presumably because viral particles have been preserved within these ganglia in a dormant state since the original episode of varicella. This results in pain and characteristic cutaneous findings along the corresponding sensory dermatomes of the involved ganglia. Less often, involvement of anterior and posterior horn cells, leptomeninges, and peripheral nerves is observed, with consequent muscle weakness or palsy, pleocytosis of spinal fluid, and/or sensory loss^[8]. Rarely myelitis, meningitis, encephalitis, or visceral involvement may occur. The diagnosis of HZ virus infection generally depends on the appearance of a characteristic cutaneous or mucocutaneous vesicular eruption. Lesions begin as erythematous macules and papules that quickly develop into vesicles. New lesions tend to form over a period of 3-5 days, sometimes coalescing to form bullae. After they form vesicles, lesions progress through stages in which they rupture, release their contents, ulcerate, and finally crust over and become dry. Almost all adult patients experience pain (ie, acute neuritis) during the eruptive phase.

A few experience severe pain without any evidence of a vesicular eruption (*i.e.*, zoster sine herpette), and a small number of patients have a characteristic eruption but do not experience pain. Symptoms and lesions in the eruptive phase tend to resolve over 10-15 days. However, lesions may require up to a month to completely heal, and the associated pain may become chronic. Patients are infectious until the lesions have dried. Anyone who has not previously had varicella is at risk of acquiring this readily transmitted virus. Pregnant women and immunosuppressed patients have the highest risk of serious sequelae. Therefore, 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. Antiviral therapy has been shown to decrease the duration of HZ rash and the severity of pain associated with it^[9].

DIAGNOSIS

It is made on the basis of clinical manifestations and subjective symptoms, presence of the viral antigens as well as presence of antibodies against VZV. Differential diagnosis of other viral infections is also possible so this

infection must be well documented. The best laboratory diagnostics are Polymerase Chain Reaction and direct Varicella Zoster virus identification in the cell culture of human fibroblasts. The sample should be taken from vesicle or serum. Sometimes herpes zoster may be present in an atypical form, especially in immune compromised patients, requiring further laboratory examination like direct immunofluorescence with fluorescein-tagged antibody (DFA) or polymerase chain reaction (PCR). The Tzanck smear in vesicular lesions is a classical method, but there is no differentiation between herpes zoster and herpes simplex. Biopsy is applied in difficult cases and VZV can be cultured successfully. The presence of Varicella Zoster virus is evidenced by direct immunofluorescence of antibodies against Varicella Zoster virus from the vesicle and up to 80% of the VZV infections could be detected using this method. Serological findings are helpful in recurrent VZV infections and show increased IgM, ten days after eruptions and increased IgG and IgA four days after the eruptions. Serological tests which reveal antibody titers might be useful in immunocompromised patients^[10].

TREATMENT

Therapeutic regimens have become more efficient nowadays, especially when they are applied 48-72 hours after the appearance of the oral lesions. Systemic intake of antiviral agents is urgent in the patients who are older than 50 years of age, in immunocompromised, and in all patients with infection of the head and neck region, especially in those with Herpes Zoster of the ophthalmic branch. In adult immunocompetent subject of less than 50 years of age, symptomatic treatment is generally sufficient. Acyclovir, valacyclovir, famciclovir or brivudin must be administered systemically. Valacyclovir is proven to be more efficient when compared to the acyclovir. Brivudin showed higher antiviral potential than acyclovir, valacyclovir and famciclovir. Brivudin is also more easily administered (*i.e.* once a day during 7 days) and has no nephrotoxic properties. Systemic use of the antiviral drugs shortens the healing period and lessens the pain symptoms together with prevention of other acute and/or chronic complications. Treatment of PHN usually comprises of analgesics together with neuroactive agents as well as with antiviral drugs. Corticosteroids administered systemically during the first two weeks of the disease is helpful in the PHN prevention, but they should not be given when PHN is already present. Some authors suggested combination of the perilesional anesthetic and corticosteroid injections. ¹¹ Reports upon shortening of the period of healing, but not PHN prevention have been documented. While treating neuralgia, analgesics, neuroactive agents and B vitamin complex should be administered. Some trials suggested that tricyclic antidepressants can be effective in alleviating neuropathic pain. The therapy in herpes zoster infection aims to shorten the clinical course, provide analgesia and to prevent complications. Antiviral agents (acyclovir, famciclovir, penciclovir, valacyclovir) are nucleotide-like substances with per os administration for 7-10 days.

Corticosteroids (prednisone) have anti-inflammatory action used mainly in acute but not in long-term pain. Analgesics (acetaminophen, non steroidal anti-inflammatory drugs) and tricyclic antidepressants (amitriptylin) can be used. Active immunity is provided by vaccines with 99% effectiveness against varicella infection^[12].

PROGNOSIS & COMPLICATIONS

Complications can occur in 10-46% patients with herpes zoster infection. Severe immunodeficiency which precedes HZ infection might predispose viral dissemination in the visceral organs, microbial super infection and staphylococcal sepsis. Disseminated HZ infection might manifest as pneumonia, meningitis, encephalitis and hepatitis, as well as dermatological diseases. Paresis of facial nerve might develop as a complication when ganglion oticum is affected. When HZ affects the first branch of the trigeminal nerve, serious damage of the eye might occur (zoster opthalmicus). Oral consequences of HZ might include heavy scarring, pulpal necrosis and internal root resorption. Also, cases of bone necrosis with teeth loss in immunocompromised patients with long term HZ have been described. Finally, patients suffering from recurrent HZ may have increased incidence of malignant diseases^[13].

CONCLUSION

Oral physicians should have a thorough knowledge about the presentation of this condition, its treatment and the possible complications. Differential diagnosis is very important to ensure that the correct treatment is performed. Because the manifestations of a trigeminal herpes zoster resemble to other oral entities the oral practitioners must be aware about the differential diagnosis and definitive treatment modalities before any dental therapy is applied and intraoral examination is necessary when skin facial lesions are observed by professionals.

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