Herpes Zoster Involving Maxillary and Mandibular Branch of Trigeminal Nerve in HIV Patient: A Case Report

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Abstract:
Herpes zoster (HZ) or shingles consist of a reactivation of the Varicella Zoster virus that had entered cutaneous nerve endings through an earlier occurrence of chicken pox, passes to the dorsal root ganglia and resides there in a latent form. Nerves usually involved are C3, T5, L1, L2, and the first division of trigeminal nerve. The characteristic appearance of this condition is occurrence of multiple, unilateral, painful vesicles, and ulceration involving a single dermatome. The infection commonly affects elderly individuals, however if present in the younger age group, raises the suspicion of immunocompromised status such as acquired immunodeficiency syndrome (AIDS)/human immunodeficiency virus. In this case report, we present a patient with AIDS who developed HZ of the left side involving the maxillary and mandibular divisions of the trigeminal nerve. The patient presented with unilateral crustations involving the left side of lower and middle one-third of the face along the tract of the trigeminal nerve, intra-orally involving buccal and labial mucosa ipsilaterally.

Key Words: Acquired immunodeficiency syndrome, herpes zoster, shingles, trigeminal nerve, unilateral vesicular lesions

Introduction
HZ is an acute viral infection characterized by vesicular skin lesions which are usually distributed over several unilateral adjacent sensory dermatomes.1 This infection occurs because of reactivation of Varicella zoster virus (VZV) which belongs to a subfamily of human alpha herpes virus2 which causes varicella (chicken pox) in childhood and then remains latent for decades in cranial nerve, dorsal root, and autonomic nervous system ganglia along the entire neural axis. Reactivation of VZV may occur spontaneously or when host defenses are compromised. Increased age, physical trauma, (including dental procedures), psychological stress, malignancy, radiation therapy, and immunocompromised states including transplant recipients, steroid therapy, and HIV infection are predisposing factors for VZV reactivation.3 The predisposing factor in the present case was immunocompromised state of the patient as the medical history of the patient revealed HIV infection which was diagnosed about 6 years back. Onunu and Uhunmwangho4 evaluated the clinical spectrum of HZ in HIV-infected patients and found that the age distribution of the patients in the HIV-positive group was 36.1 ± 16.14 years and infection was generally more severe in the presence of HIV infection.

Case Report
A 35-year-old male patient reported to the out-patient department with pain and swelling over the left mandibular region radiating to the left ear since a week. The patient also complained of multiple ulcerations on the left side of the face extending from corner of the mouth to the temporal region. The patient initially developed an extraoral swelling on the left side of the face associated with pain in the lower left posterior teeth for which he visited a local doctor and was prescribed antibiotics and analgesics. The patient gave a history of fever about 10 days back, after which he developed ulcerations on the face since 5 days associated with burning sensation intra-orally on the affected side. His medical history revealed that he was diagnosed with human immunodeficiency virus (HIV) infection about 6 years back. The CD4 count was 446 cells/mm3 for which he was advised antiretroviral therapy (ART) which included tablets lamivudine, zidovudine, and nevirapine. The patient took the above-mentioned medicines regularly for a month after which he discontinued abruptly.

Figure 1: Clinical picture of case. (a) Extraoral view showing crustations (dark arrow), lesions typically not crossing the midline (light arrow), (b) extraoral view showing ulcerations on labial mucosa (dark arrow).
On extraoral examination, the skin over the left side of face showed diffuse crustations extending from the chin region along the left side of the face up to the temporal region characteristic not crossing the midline (Figure 1). Gross asymmetry of the face was seen with a diffuse oval swelling approximately 2 cm × 2 cm was seen on the left side of the face extending over the left body of the mandible with normal overlying skin, soft in consistency, tender on palpation with no local rise in temperature. Multiple, irregular ulcerations along with intact vesicles were present over the vermilion border and labial mucosa of the lower lip on the left side not crossing the midline (Figure 1). The crustations and ulcers appeared large, irregular, and seemed to coalesce with each other and were tender on palpation. Intraoral examination revealed deep occlusal caries with a lower left first molar. Limited mouth opening led to incomplete intraoral examination. Based on the history and clinical examination, a provisional diagnosis of chronic periapical abscess secondary to advanced occlusal caries with 36 and herpes zoster (HZ) infection involving the maxillary and mandibular division of the trigeminal nerve in HIV-infected individual was made.

Radiographic investigations included a mandibular left lateral oblique radiograph which revealed a grossly carious lower left first molar associated with a well-defined, smooth, round, continuous, corticated radiolucency approximately 2 cm in diameter (Figure 3). Blood investigations were carried out. Complete hemogram showed elevated white blood cells (17000/μL), decreased hemoglobin (8.4 g/dl), and raised erythrocyte sedimentation rate of 32 mm at the end of 1 h. Distinct fall in CD4 count (280 cells/mm³) was noted. Exfoliative cytology (Tzanck smear) was made by scraping an intact vesicle located on the left labial mucosa of the lower lip and stained with H and E stain. Histopathological examination of the Tzanck preparation showed acantholysis along with numerous free-floating Tzanck cells and ballooning degeneration (Figure 2) confirming the clinical diagnosis of HZ infection.

The patient was referred to his physician for overall assessment and reconsideration to restart the ART. The ART was restarted which consisted of tablets lamivudine (150 mg) twice daily, zidovudine (300 mg) twice daily, and nevirapine (200 mg) once daily for 2 weeks.

Furthermore, in consultation with the treating physician, the patient was prescribed tablets acyclovir (800 mg) five times a day for 10 days and benzydamine mouth rinse (tantum oral rinse) 4-5 times per day for 10 days for symptomatic relief.

For the dental infection, the patient was prescribed tablets augmentin (500 mg/125 mg, amoxicillin/clavalunic acid) twice a day for 5 days and combilam thrice a day for 5 days. The offending tooth, 36 was extracted after the swelling had subsided about a week after initial presentation. The lesions associated with HZ infection had reduced by almost 80%, and there were drastic improvements in the symptoms as well. The patient was then lost for further follow-up.

**Discussion**

HZ is commonly known as shingles, derived from the Latin word “cingulum” meaning girdle. This is because a common presentation of HZ involves unilateral rash that can wrap around the waist or chest like a girdle. Similarly, the name “zoster” is derived from Greek word referred to a belt-like binding structure used by warriors to secure armor.5

The incidence of HZ increases by 15 times in HIV-infected patients and also about 25% cases of Hodgkin’s lymphoma suffers from HZ infection.6 Transmission of HZ infection occurs by the spread of infectious particles from oral secretion, skin lesions, or via direct contact with the patient.7 Clinical
appearance of HZ can be categorized into three phases—prodromal, acute, and chronic. In the prodromal phase, inflammation of ganglion occurs due to replication of virus which causes necrosis of the affected sensory nerve resulting in occurrence of intense pain along with burning, itching, tingling, boring, and prickly sensation in the skin and/or oral mucosa supplied by that nerve. Prodromal phase is followed by patchy erythema, occasionally accompanied by indurations that appear in the dermatome area of involvement. Rash is usually accompanied by malaise, headache, nausea, low grade fever, and further advances to blisters with erythematous base on the affected area of skin which then postulates and further results in ulceration. Subsequently, lesion dries and form crustations along the tract of affected sensory nerve and ends at the midline. These crustations reduce in 14-21 days leaving behind an erythematous macular lesion that further result in hyper or hypopigmentation of the affected area of the skin. The present case also presents a history of fever about 10 days back, after which the patient developed ulcerations on the face associated with burning sensation intra- orally on the affected side.

Involvement of the maxillary or mandibular divisions of the trigeminal nerve may result in lesions either on the oral mucosa or skin or both. In the present case, HZ infection involving the maxillary and mandibular division of the trigeminal nerve was diagnosed. The lesion was presented with multiple, irregular ulcerations along with intact vesicles over the vermillion border and labial mucosa of the lower lip on the left side not crossing the midline. The crustations and ulcers appeared large, irregular, and seemed to coalesce with each other and were tender on palpation.

HZ infection can also involve all the three branches of trigeminal nerve. Skin lesions usually herald the intraoral lesions as oral lesions generally appear after the cutaneous rash. Erythematous zone surrounds the intraoral vesicles which further result in hyper or hypopigmentation of the affected area of the skin. Rash is also evident in the present case. Histopathological examination of the Tzanck preparation demonstrated acantholysis along with the formation of many free-floating Tzanck cells and ballooning degeneration which confirmed the clinical diagnosis of HZ infection. Other approaches to confirm the clinical diagnosis consists of virological test which includes viral culture and is considered as a most accurate method with higher sensitivity and specificity. Other advanced investigation technique such as direct and indirect immunofluorescence, complement fixation test can also be used.

Therapeutic intervention for management of HZ infection should be intended at pain control, treatment of skin, and oral lesions, use of antiviral therapy and should be aimed at prevention of complications. Skin lesion can be treated by applying of open wet dressings soaked in cool water and pain can be managed by prescribing mild to moderately strong analgesics along with inflammatory agents.

Antiviral drug therapy reduces the duration and severity in the acute phase of the HZ infection. Acyclovir is the drug of choice with recommended dosage of 800 mg four to five times a day for 10 days. Other newer agents such as Famciclovir 500 mg and Valacyclovir 1 g can be prescribed three times a day for 7 days. In the present case, as the patient was earlier diagnosed as HIV, ART was restarted in consultation with the treating physician which consisted of tablets lamivudine (150 mg), zidovudine (300 mg) both tablets twice daily, and nevirapine (200 mg) once daily for 2 weeks along with acyclovir (800 mg) five times a day for 10 days and benzydamine mouth rinse (tantalum oral rinse) 4-5 times per day for 10 days for symptomatic relief.

Antiviral therapy has been found to halt progression and dissemination of acute phase of HZ in patients with immunocompromised status, even if commenced more than 72 h after rash onset. Such therapy is recommended for all HZ patients with immunocompromised status who present before the complete crusting of all lesions. IV acyclovir is preferred drug for the cases with advanced HIV/acquired immuno deficiency syndrome (AIDS) who harbor active opportunistic infections or exhibit prominent wasting. Antiviral therapy should not be stopped until all lesions have resolved. Thus, early detection and timely treatment of the HZ in the prodromal phase by the use of antiviral agents may serve in decreasing the severity and duration of this infection along with a reduction in complications.
Conclusion
Earlier, patients with AIDS had a very poor survival rate. With newer drug regimens available for the treatment of patients with AIDS, there is a better prognosis and such patients are now having better survival rates. Therefore, dental practitioners will be facing more such cases of HZ (and other oral manifestations) in patients with AIDS. Furthermore, because of compromised defense mechanism in patients with AIDS, the lesions are likely to be more extensive and more difficult to treat. Moreover, HZ infection can be transmitted from the patient to the dentist by the diffusion of infectious particles in aerosol that could be released from oral secretions, direct contact or via skin or oral lesions. Hence, it is imperative that the clinician is aware of the pathognomonic features as well as the management aspect of such infections.

References