Necrotizing Stomatitis in Varicella Zoster Infection

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ABSTRACT

Necrotizing periodontal disease are acute periodontal conditions which can present with simple gingivitis to more complex life threatening condition where it can perforate the skin of cheek. They are found in patients with severe illness, stress and reduced immunity. The prevalence of this whole group of disease is usually very low and is stated frequently as the first manifestation in HIV infection. Herpes zoster, a common dermatological condition is a secondary infection due to re-activation of Varicella zoster virus in a person previously acquiring a chicken pox in his/her lifetime. There are limited scientific literatures citing the occurrence of necrotizing stomatitis in varicella zoster infection. Thus, here we present a detail about a case and management of 46 years old female who had a hospital stay of over 5 days for treatment of Herpes zoster infection of mandibular nerve presented with necrotizing stomatitis lesion within oral cavity.

KEY WORDS

HIV, Necrotizing stomatitis, Varicella zoster infection

INTRODUCTION

Necrotizing stomatitis is a severe inflammatory condition of the periodontium and the oral cavity in which soft tissue necrosis extends beyond the gingiva and bone denudation may occur through the alveolar mucosa, with larger areas of osteitis and formation of bone sequestrum. It is one of the variant of the broad category of disease called as Necrotizing periodontal disease (NPD). It typically occurs in severely immune-compromised patients. In addition to immunosuppression, malnutrition, psychological stress, insufficient sleep, inadequate oral hygiene, pre-existing gingivitis or periodontitis, tobacco consumption, alcoholism, age, seasonal variations, etc. are the suggested etiology and risk factors for the development of NPDs. Prevalence of necrotizing stomatitis are not estimated and are only limited to case reports whereas its predecessor Necrotizing gingivitis prevalence in the general population is estimated to be around 0.51 to 3.3% which is also not a worldwide prevalence.

Chicken pox and Herpes zoster (HZ), both are caused by Varicella zoster virus (VZV). Chicken pox, a very common skin infection which occurs in younger age group is a primary disease caused by VZV. Herpes zoster (HZ), or shingles, is the painful eruption of a rash, usually unilateral. Post herpetic neuralgia with tingling sensation is a common feature in HZ. HZ is caused by reactivation of the varicella zoster virus (VZV) which remained latent in the dorsal ganglion cells of sensory nerves. HZ is a common disease with a lifetime risk of 10-30% which increases to 50% among individuals ≥ 85 years.

CASE REPORT

A 46 year old female patient reported to the Department of Periodontology, Kathmandu University School of Medical Sciences, Dhulikhel with a chief complaint of gingival swelling in lower left back region of mouth since 1 week. Medical history revealed that she had a hospital stay for 5 days for management of Herpes zoster infection of mandibular nerve with dissemination. Laboratory investigations suggested that she was HIV seronegative.
Her personal history revealed that she was a chronic current smoker (23.25 pack-years) and had a poor socio-economic background.

Extra-oral examination revealed the presence of severely hyperpigmented patches which were present unilaterally on left side of the face. (fig. 1) On intraoral examination, her oral hygiene was poor, severely inflamed, edematous gingiva with spontaneous bleeding on slight provocation was noted along with exposure of alveolar bone with respect to 33 and 34. (fig. 2) The periodontal diagnosis was made as necrotizing stomatitis (Hornig and Cohen Stage 6) whose prevalence is estimated to be only 1% of total NPDs.5

Patient conditions were evaluated. The gingival margins were erythematous without any superficial necrotic areas. The overt inflammation was dramatically reduced but exposing more amount of alveolar bone in the affected area. (fig. 4) The area was irrigated using 3% H2O2. Patient was asked to follow the same instructions given previously and was recalled after 5 days.

The area was anesthetized by use of topical anesthetic (2% Lignocaine gel). Small cotton pellets were moistened in 3% hydrogen peroxide solution which delivers nascent oxygen to the area where anaerobic bacteria are believed to be predominant. Cotton pellets were intended for single use and any sweeping motion which could possibly transfer infection to adjacent non affected sites was prevented.6

The area was anesthetized by use of mental nerve block technique with 2% Lignocaine hydrochloride and 1:200,000 epinephrine. Extraction of tooth 34 and 35 was performed along with removal of sequestrum and was sent for histopathological examination. (fig. 7) The area was sutured for healing with primary intention and recalled after 10 days for suture removal. (fig. 8)

Follow up visits

Suture removal was done but the wound closure was not achieved primarily. The large defect was present
on the buccal aspect of the extracted site. (fig. 9) So, for further anticipation of restorative therapy on mind to gain keratinized tissue secondary suturing was performed and bleeding was intentionally induced. (fig.10) Histopathological slides confirmed the specimen was of dead bone where there was empty bony lacuna. In a viable bone, the osteocytes are present within the bony lacuna.7

**DISCUSSION**

NPDs are more common in HIV seropositive patients and can be the 1st manifestations of HIV infection or an indication of immune deterioration. In HIV-positive patients, decreased peripheral CD4 lymphocytes are correlated with NPDs. The HIV patients with NPDs were 20.8 times more likely to have CD4+ cell counts below 200 cells/mm$^3$ than patients without NPDs which suggests severe immune deficiency in HIV-NPDs patients.8 As with other periodontal diseases, the etiology for NPDs appears to be polymicrobial. The bacterial etiology of NPD (fusobacterium) was first demonstrated by Plaut in 1894 and Vincent in 1896 using electron microscopy. Since then, many bacteria and viruses have been isolated from NPD lesions. Culture studies identified P. intermedia, Treponema, Selenomonas and Fusobacterium species and are considered as “constant flora” in NPD lesions. The microbiota associated with NPD in HIV is like that of periodontitis in non HIV patients, with some specific features, such as presence and invasion of Candida albicans, herpes viruses or superinfecting bacterial species.3 Thus, in the recent past the role of Herpes virus has been advocated for aggressive forms of periodontal disease. There are total of eight human herpes virus (HHV) of which varicella zoster is HHV-3. HHV-2 (Herpes simplex 2) and HHV-3 (VZV) are isolated from severe periodontal disease and the periodontal herpes virus total copy counts can exceed the total bacterial cell counts.9 The risk behind the presence of HHV in periodontal sites are : Studies have found that the sites where HHV are isolated has
more amount of periodontal destruction, can persist in periodontal sites for longer duration, can replicate in oral/gingival epithelial cells and herpes-bacteria co-interaction can pose an ongoing risk for disease progression.\textsuperscript{9,10}

So, necrotizing stomatitis is a rare clinical entity which is seen once in a long while. Its close association with HIV patients is sometimes breached and seen in non-HIV patients as well. The viral etiology of periodontal disease has time and again led the periodontal scientific committee towards finding the exact link of pathogenesis and formulation of management protocol for virus associated periodontal disease. The aggressive nature of this disease demands prompt action to limit the periodontal destruction and slight variation in management protocol can give us a chance to offer patient a near-natural prosthetic rehabilitation.

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**REFERENCES**


