Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies *NLM ID: 101716117*

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr Indian Citation Index (ICI) Index Copernicus value = 100

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Case Report

Osteomyelitis of Maxilla : Associated with Herpes Zoster Infection :A Rare Case Report

[1]Geetika Gupta, [2]Sangeeta Malik, [3]Swati Gupta, [4]Preeti Sharma, [5]Falguni Sharma, [6]Saba

[1]Junior Resident1, [2,3]Professor, Department of Oral Medicine and Radiology, Subharati Dental College, Meerut, India
[4]Professor, Department of Oral Pahology and Microbiology, Subharati Dental College, Meerut, India
[5,6]Intern, Subharti Dental College, Meerut, India

ABSTRACT:
Herpes Zoster (HZ) is a viral infection caused by reactivation of latent varicella zoster viruswithinthesensory nerveganglia. Herpes zoster involving the trigeminal branch leads to complications like pulpnecrosis, osteomyelitis and post herpetic neuralgia[PHN].The present report describes a rare case of maxillary osteomyelitis secondary to herpes zoster infection.
Keywords-Herpeszoster,osteomyelitis,Trigeminalnerve, sensoryganglion.

Received: 03 December, 2025 Accepted: 05 January, 2026 Published: 08 January, 2026

Corresponding Author: Geetika Gupta, Junior Resident1, Department of Oral Medicine and Radiology, Subharati Dental College, Meerut, India

This article may be cited as: Gupta G, Malik S, Gupta S, Sharma P, Sharma F, Saba Osteomyelitis Of Maxilla : Associated With Herpes Zoster Infection :A Rare Case Report. J AdvMed Dent Scie Res 2026; 14(1):103-106.

INTRODUCTION

The diagnosis of Herpes Zoster involving branches of Trigeminal nerve is of particular importance to dental practitioners as around 20% cases of herpes zoster involves this nerve. While PHN being the most common complication of Herpes Zoster, osteomyelitis is the rarestone. Avascular necrosis ,also termed as osteonecrosis is a condition where the bone tissue succumbs to inadequate blood flow [1,2,3,4].If left untreated, it can lead to severe functional disability, the collapse of bone, and joint discomfort[5,6,7].
Clinically presents with pain, swelling, suppuration and in severe cases may lead to bonynecrosis. Occurence inmandible is more than that in maxilla (2:1)due to decreased vascularity and dense cortical bones.

CASE REPORT

An 80 years old male patient reported to the Department of Oral Medicine and Radiology, Subharti Dental College and Hospital, Meerut with the chief complaint of continuous pain and bone like outgrowth in the upper right front edentulous region of jaw since 6 months. The pain was dull and intermittent in nature aggravated on chewing hard food. Patient has been edentulous for around 6-7 yearsand has had an episode of Herpes Zoster infection 1.5 yrs ago. No other relevant medical, dental and personal history was reported by the patient apart from an incident of injury sustained to the right upper alveolar mucosa while eating something hard,about a month and a half ago.
On extraoral examination a single right submandibular lymph node was palpable which was firm, tender and mobile. Intraoral examination revealed edentulous maxillary arch with breach in the continuity of mucosa and exposed bone in right maxillary canine alveolus region along with mild swelling and inflammation in the surrounding soft tissues which was tender on palpation.

FIG:1.1Exposed bone in right maxillary canine edentulous alveolus region.

Based on the history and clinical findings a provisional diagnosis of Herpes Zoster induced Osteomyelitis of the right side of maxilla was made and a
Differential diagnosis of bacterial osteomyelitis, mucormycosis and fungal osteomyelitis was given.

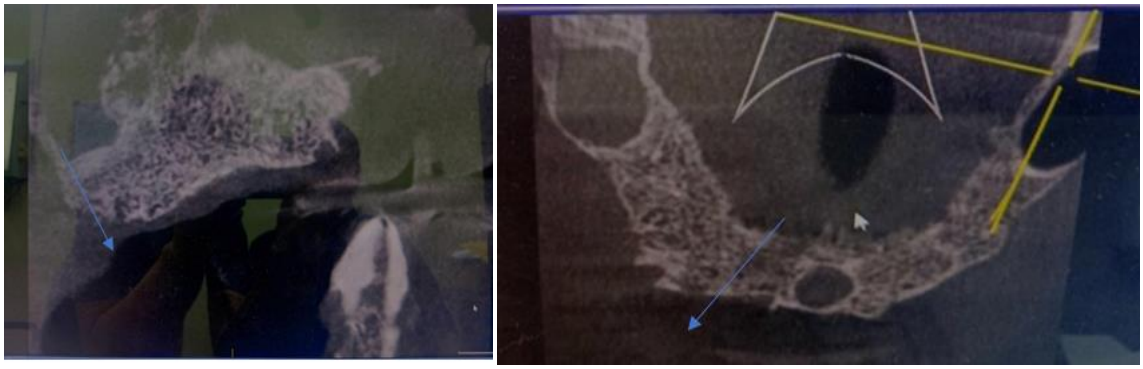

FIG:1.2 and 1.3 revealed mixed lytic sclerotic bone destruction in the right maxillary canine premolar region with cortical breach.

INVESTIGATIONS

Radiographic and histopathological examination of the patient was done.CBCT revealed mixed lytic sclerotic bone destruction in the right maxillary canine premolar region with cortical breach/ perforation and sequestrum formation, consistent with osteomyelitis. Histopathology reportrevealed multiple areas of necrotic bone devoid of osteocytes ,representing the sequestrated bony tissues and chronic inflammatory cells ,comprising predominantly of lymphocytes and plasma cells.
Based on the investigations ,a final diagnosis of OSTEOMYELITIS was made.

DISCUSSION

Herpes zoster infection (HZI), commonly known as Shingles or Zona, is a neurotropic viral disorder characterized by painful skin rashes and blisters with unilateral distribution.
Primary infection with Varicella zoster virus (VZV), an α-herpesvirus, leads to chicken pox. Transmission is usually by the respiratory route, with an incubation period of 2 to 3 weeks. As with HSV, this virus is cytopathic to the epithelial cells of the skin and mucosa, causing blisters and ulcers. Usually, oral vesicles appear after skin lesions. Oral vesicles rupture and coalesce as large mucosal erosions(8)
After the initial infection with VZV, the virus becomes latent in the nerve cell bodies,less frequently in non-neuronal satellite cells of the dorsal root, cranial nerve or autonomic ganglion, without causing any symptom(9,10). Reactivation in association with immunosuppression and mechanical or psychological stress causes the virus to travel along sensory nerves and infect the epithelial cells leading to secondary infection which manifests as vesicular rash, radicular pain in the affected dermatomal area. It affects 20 - 30 % of the population and produces severe prodromal symptoms such as localized pain, burning, tingling or hyperesthesia in specific dermatomes, usually unilaterally. It is then followed by clusters of fluid filled blisters which crust over and heal within 2-4 weeks, potentially leaving scars or discolouration. The incidence of HZI increases with age and the degree of immunosuppression(11). There are 1.5 to 3 cases of HZI per 1,000 subjects; this increases to 10 per 1,000

in those over age 75 years. Therefore, it is not uncommon to see HZI in the elderly, in patients undergoing cancer chemotherapy, in patients on chronic immunosuppressive drug therapy (such as those who have received organ transplants), and in patients with AIDS.

Although the thoracic dermatomes are most frequently involved, cranial nerve involvement occurs in approximately 20%of cases,with the trigeminal nerve being the most commonly affected cranial nerve. It can affect any of the three branches of trigeminal nerve with ophthalmic branch being most commonly involved. The involvement of the mandibular and the maxillary branches (that produces oral manifestations), without the involvement of the ophthalmic branch is relatively rare and accounts for only 1.7% of HZ cases(12)

The most common post zoster related complications include ocular complications, facial palsy, post herpetic neuralgia (PHN), bacterial superinfections, osteonecrosis, periodontitis, exfoliation of teeth, calcified and devitalized pulps, periapical lesions and root resorption(12,13)

Postherpetic neuralgia (PHN), a complication of HZI, is a neuropathic disorder caused by peripheral and central nervous system injury and altered central nervous system processing.

Maxillary osteomyelitis secondary to Herpes Zoster Virus (VZV) is an exceedingly rare and an infrequently reported entity but a debilitating complication, occuring in roughly 13% to 22% of cases involving the trigeminal nerve. Although herpes zoster incidence increases with advancing age, progression to osteomyelitis of the maxillofacial skeleton is rarely observed, even in elderly male patients. While the mandible is more frequently affected, involvement of maxilla is significant due to its aesthetic,functional implications and typically rich blood supply, which usually protects it from such extensive necrosis.

The exact pathogenesis remains unclear and is still debated.However three main hypothesis have been suggested which are as follows –

1) Viral induced vasculitis: Most commonly supported hypothesis which proposes that reactivation of VZV may cause localized vasculitis of superior alveolar artery, resulting in ischemia and bone necrosis.
2) Neural inflammatory response -suggests that inflammation originating in the maxillary division (V2) of the trigeminal nerve may extend directly to the periosteum and may impair the vitality of bone .
3) Secondary bacterial invasion- Mucosal ulceration and denervation during the acute phase may facilitate bacterial invasion, leading to osteomyelitis in the affected bone.

In edentulous cases like this one, the diagnosis can be particularly challenging as typical dental warning signs like tooth mobility or "spontaneous exfoliation" are absent.So, the absence of teeth in the affected regioneliminates the common odontogenic sources of infection, thereby underscoring the atypical pathogenesis and strengthening the causal association with herpes zoster- induced vasculopathy / viral - induced ischemia.

Underlying comorbidities, especially poorly controlled diabetes, are reported in over 68% to 85% of maxillary osteomyelitis cases, as they significantly impair immune surveillance and wound healing.

Management and Prognosis

A combined medical and surgical approach is typically required for resolution:

Antiviral Therapy: Antiviral Therapy: Immediate administration of agents like Acyclovir (800 mg 5x daily) or Valacyclovir (1000 mg 3x daily) is critical to limit viral replication.

Surgical Intervention: Sequestrectomy or thorough debridement of necrotic bone is necessary once the area is demarcated to prevent further spread into the maxillary sinus or skull base.

Antibiotic Coverage: Extended courses of broad-spectrum antibiotics (e.g., Clindamycin or Amoxicillin/Clavulanate) address secondary bacterial osteomyelitisand stringent management of blood sugar levels are vital for patients with systemic risk factors.

CONCLUSION

Osteomyelitis as a complication of Herpes Zoster is an uncommon but serious entity that can result inconsiderablefunctionalandstructuralimpairmentifnotidentifiedproperly.

The present case emphasizes that Herpes Zoster should be considered a potential etiological factor in patients presenting with unexplained osteonecrosis or osteomyelitis of the jaws, particularly when preceded by a history of dermatomal vesicular eruptions and neuralgic pain.

The convergence of maxillary involvement, edentulous status and herpes zoster etiology, render this case an exceptional presentation and awareness of this rare association is crucial for dental and medical practitioners, as early intervention with antiviral therapy, appropriate antimicrobial coverage, and surgical management whenindicatedcansignificantlyimprovepatientoutcomes.

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