Simultaneous Occurrence of Chicken Pox and Herpes Zoster with Facial Nerve Palsy in Immunocompetent Patient

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Abstract
Patients who earlier suffered from chicken pox may develop herpes zoster (HZ) due to reactivation in dorsal sensory nerve root ganglion postprimary varicella infection (chicken pox). Association between HZ and varicella has been long established by Bokay, Ferryman, Campbell, Almeyda, Ward, Roxburgh, and Martin.¹,² They also described the simultaneous occurrence of both clinical entities in their case series within 5 days of zoster eruption. Many such reports are described in immunocompromised individuals like HIV, diabetes, corticosteroid use, cancer patients, syphilis, heavy metal poisoning, irradiation, and elderly population.³ Atypical presentation like toothache and complications like pulp necrosis and periodontitis is not uncommon in maxillary HZ. Unnecessary dental interventions should be avoided before a proper diagnosis is established. Subtle facial nerve palsy is frequently associated with HZ oticus due to anatomical predisposition. In our case, severe facial nerve dysfunction is seen with maxillary division of trigeminal HZ, which is very uncommon. Hematogenous dissemination theory has also been postulated for dissemination in an immunocompromised host. Still, the development of lesions typical of chicken pox along with HZ in the immunocompetent host is poorly understood (Figs 1 and 2).

Introduction
Herpes zoster occurs due to reactivation of latent varicella-zoster virus (VZV) in dorsal sensory nerve root ganglion postprimary varicella infection (chicken pox). Association between HZ and varicella has been long established by Bokay, Ferryman, Campbell, Almeyda, Ward, Roxburgh, and Martin.¹,² They also described the simultaneous occurrence of both clinical entities in their case series within 5 days of zoster eruption. Many such reports are described in immunocompromised individuals like HIV, diabetes, corticosteroid use, cancer patients, syphilis, heavy metal poisoning, irradiation, and elderly population.³ Atypical presentation like toothache and complications like pulp necrosis and periodontitis is not uncommon in maxillary HZ. Unnecessary dental interventions should be avoided before a proper diagnosis is established. Subtle facial nerve palsy is frequently associated with HZ oticus due to anatomical predisposition. In our case, severe facial nerve dysfunction is seen with maxillary division of trigeminal HZ, which is very uncommon. Hematogenous dissemination theory has also been postulated for dissemination in an immunocompromised host. Still, the development of lesions typical of chicken pox along with HZ in the immunocompetent host is poorly understood (Figs 1 and 2).

Case Description
A 48-year-old male patient with no history of hypertension or diabetes developed right upper molar toothache and fever. Initially, he consulted a dentist locally and was prescribed antibiotics and analgesics. But he developed rashes over his face and palate. On the 5th day of rash, he presented to us with high-grade fever, orofacial pain of burning character, papulovesicular rash distributed along with the maxillary division of trigeminal nerve, and vesicular lesions typical of chicken pox all over the body (Fig. 3). There was no eruption over the tympanic membrane, ear canal, pinna, tragus, and no earache. He also had conjunctival redness and increased lacrimation. He had no history of varicella illness. In due course, he also developed facial nerve palsy of lower motor neuron (LMN) type on the right side (Fig. 4). Other cranial nerves examinations were normal.

On clinical examination, temperature 102.6°F, pulse 90 bpm, blood pressure 120/88 mm Hg, general and systemic examinations were within normal limits.

Investigation
Random blood sugar (RBS) 116 mg/dL, hemoglobin 12.3 gm%, total count 6000/ cumm (P-63, L-25, M-10, E-02), erythrocyte sedimentation rate 15, hepatitis B and C screening test was negative, HIV-1/2 was nonreactive, liver function test was within normal limits, and swab for culture from facial lesions Staphylococcus aureus. Chest X-ray was normal, and Tzanck smear from vesicles was positive. Varicella-zoster IgM antibody was negative and IgG was positive, sent on the 10th day of fever.

Diagnosis and Treatment
The diagnosis was made based on the history of presenting illness, clinical picture, and laboratory investigations as the simultaneous occurrence of HZ of the maxillary division of trigeminal nerve and VZ with superadded secondary infection of S. aureus and peripheral type facial nerve palsy. He was managed over oral valacyclovir, gabapentin, IV ceftriaxone, and acyclovir ointment for skin lesions. He improved adequately and was then discharged. On follow-up after 2 weeks, his skin lesions healed by crust, but painful sensation and paresthesia over right maxillary nerve distribution persisted, and facial palsy became obvious (Fig. 4). On subsequent follow-up after 3 months, facial palsy improved exclusively.

Discussion
Herpes viruses like VZV and herpes simplex viruses (HSV) have the capacity for latency and later evade the host immune system and reactivate. The primary infection of VZV is chicken pox, and post-reactivation is called shingles (HZ). Immunosuppression and advanced age are risk factors for reactivation. Immune evasion of VZV is due to declining T cell-mediated immunity in the elderly population. Factors responsible for reactivation in immunocompetent hosts are largely unknown. Currently, vaccines are available, but the role in a non-elderly immunocompetent patient is debatable and not yet recommended. As healthcare facilities are improving and many patients are presenting in the pre-eruptive stage where a complete clinical picture is not yet appeared, diagnosis is often missed and may lead to unnecessary interventions; another issue is the poor response of medicine after 72 hours of development of rash; furthermore, postherpetic neuralgia also leads to comorbidity and poor quality of life in undiagnosed cases where treatment is delayed. All these factors emphasize rapid diagnosis and awareness of unusual presentations. Our patient presented with odontalgia to the dentist and later developed HZ and varicella. One should keep HZ as a differential diagnosis in patients with orofacial pain. Often herpes
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In conclusion, a case of HZ affecting the maxillary division of the trigeminal nerve is reported along with peripheral facial nerve palsy and simultaneous occurrence of chicken pox in an immunocompetent patient. This case highlights the importance of a thorough history and examination in patients with toothache and indicates the necessity for a further inquest into pathogenesis. Herpes zoster should be considered as a differential diagnosis in patients presenting with a toothache. Additionally, physicians should be aware of the unusual presentation of VZV. Physicians are encouraged to identify the early symptoms of HZ and to administer antiviral therapy timely to prevent complications.

REFERENCES


IgM indicates previous exposure. A negative result does not effectively rule out varicella infection as it may depend on when the sample was drawn. In our case, only IgG was positive as the sample was drawn on the 10th day of fever. In most HZ cases, thoracolumbar and cervical dermatomes are affected, accounting for more than 70% of cases; the trigeminal nerve is involved in 15% of cases, but mainly in the ophthalmic division, the maxillary division is affected in 1.7% of cases. The development of facial palsy in these cases is seldom reported. But the simultaneous occurrence of maxillary nerve HZ and typical chicken pox-like eruption all over the body along with LMN-type facial nerve palsy in an immunocompetent patient is unprecedented.

Fig. 1: HZ of right maxillary nerve (V2)

Fig. 2: Right side palatal mucosal involvement as part of maxillary nerve involvement. Purple color of tongue is due to gentian violet application

Fig. 3: Typical varicella (chicken pox) lesions over back

Fig. 4: Right LMN type facial nerve palsy