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Concurrent Involvement of Trigeminal and Facial Nerves in Herpes Zoster

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ABSTRACT

A 51-year-old female reported pain and an erythematous rash on the right side of her face, along with mouth deviation on the left side and inability to close her right eye for 23 days. The blisters ruptured and healed. Later, the patient reported severe pain and stiffness on the right side of her face. The patient was treated conservatively with medication and physiotherapy, which yielded satisfactory results. Varicella-zoster virus reactivation in ganglion in the 6th decade leads to the recurrence of Herpes. Due to their anatomical proximity, the lesion involved both the trigeminal and facial nerves, as we see in this case. It can be associated with many other symptoms and likewise got recognition as the Ramsay James Hunt Syndrome. At present, only 126 cases have been reported, making it quite a rare entity. Apart from management using drugs, physiotherapy also plays a primary role in the recovery of such cases. In this case report, it is aimed to present a herpes zoster case with the involvement of the trigeminal and facial nerves.

Keywords: Facial nerve, facial paralysis, herpes zoster, trigeminal nerve



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INTRODUCTION

Trigeminal, a mixed nerve and facial, predominantly a motor nerve, are two of the essential cranial nerves of the head and neck.^[1] Connoisseurs and wine experts believed that somatosensory fibers (cranial nerve V) on the tongue interact with “taste” (cranial nerve VII) due to their anatomical proximity and relations. Electrophysiological studies have revealed that the taste (gustatory) neurons that arise from the facial nerve at the level of the medulla and lower pons (solitary nucleus) are modulated by the trigeminal nerve (V), innervating somatosensation of the tongue.^[2]

Shingles (herpes zoster) is a cutaneous viral infection caused by varicella-zoster virus (VZV) reactivation, typically presenting with a characteristic vesicular rash following a dermatomal distribution, along with neuralgiform pain along the division of nerve involved, not crossing the midline.^[3,4] Complete involvement of all trigeminal nerve branches is rare, with facial nerve involvement still rarer, with only 126 such cases found in recorded history.^[5] In this case report, it is aimed to present a herpes zoster case with the involvement of the trigeminal and facial nerves.

CASE REPORT

A rare case report of a 51-year-old female with shingles involving all divisions of the trigeminal nerve, along with facial nerve involvement. This case was reported to the Department of Oral and Maxillofacial Surgery with a chief complaint of erythematous rashes and pain on the left side of the face (Fig. 1), along with deviation of mouth to the left side and inability to close her eyes for the past 10 days (Fig. 2). The patient is a known diabetic, under regular medication for the past 10 years. The patient consulted a dermatologist who prescribed topical steroids but to no avail. The patient reported severe pain and stiffness on the right side of her face, inability to close her eye, and ophthalmic involvement. There was no change in hearing or taste sensations. The symptoms were suggestive of herpes zoster infection, involving all three branches of the trigeminal and the facial nerve and affecting the left side of the face. Laboratory tests of the patient are summarized in Table 1. The final diagnosis made was herpes zoster infection with associated facial nerve palsy. The patient was prescribed acyclovir (800 mg qid), pregabalin (75 mg tds), amitriptyline (50 mg bd), and duloxetine (60 mg od). The patient was also prescribed corticosteroid and lubricating eye drops to be used in the affected eye until symptoms disappeared. In addition to medicinal therapy, physiotherapy treatment was started to restore the strength of facial muscles, including neuromuscular retraining (NMR). The symptoms of both herpes zoster and facial palsy almost disappeared in 10 weeks.

DISCUSSION

Herpes zoster occurs from latent VZV reactivation from the dorsal root or neurosensory ganglion, mostly in immunocompromised patients.^[6] In such patients, the frequency of infection is 53% in thoracic dermatomes, 20% in other cranial nerves, 15% in the trigeminal nerve, 4–20% in cervical dermatomes, and 11% in lumbosacral dermatomes.^[4] Patients typically complain of characteristic, unilateral eruptions along the distribution of a nerve, progressing in stages of papules, vesicles, bullae, pustules, and crusting over a few days to 3 weeks.^[7] The rash is frequently preceded by a prodromal period comprising malaise, headache, fever, and eye pain. The prevalence of the disease rises with age.^[3,8]

Ophthalmic involvement is present in 30–40% of patients involving the ophthalmic branch.^[8] Involvement of the nasociliary branch is indicated by Hutchinson's sign (vesicles on the tip or side of the nose), a strong predictor of corneal denervation and ocular inflammation.^[6] When the mandibular branch is involved, lesions are localized to the V3 dermatome.^[9]



Figure 1. Erythematous rashes on the left side of the face.



Figure 2. The inability to close the eye and deviation of the mouth to the left side.

Table 1. Laboratory tests of the patient

	Patient's values	Normal range
Hemoglobin	11.2 g/dL	11–15 g/dL
Total leucocyte count	5,200/ μ L	4,000–11,000/ μ L
Neutrophils	66%	55–70%
Lymphocytes	23%	20–45%
Monocytes	7%	2–10%
Eosinophils	4%	1–6%
Basophils	0%	0–1%
Bleeding time	4 min 20 s	2–7 min
Clotting time	9 min 10 s	8–15 min
Random blood glucose	106 mg/dL	<126 mg/dL
HBsAg	Negative	Negative
HCV	Negative	Negative
HIV I and II	Negative	Negative

Bell's palsy, where branches of facial nerve controlling the muscles of the face become weak or paralyzed, was previously thought to be an idiopathic condition but is now closely associated with the viral infection.^[8] Many studies point out that the reactivation of latent VZV infection is a crucial factor contributing to Bell's palsy.^[10] Furthermore, it is now believed that Bell's palsy is a type of herpetic neuritis of the facial nerve caused by reactivated VZV in the geniculate ganglion.^[11] According to Edgerton, "the association of facial paralysis with ophthalmic zoster is of relatively infrequent occurrence." Laboratory analysis has revealed that, out of all patients with Bell's palsy, 56% have Zoster sine herpete.^[4]

In cases of herpes zoster infection, therapy aims to shorten the duration, provide pain relief, and prevent any complications.^[12] Antivirals are administered for 7–10 days.^[7] Corticosteroids, found to be anti-inflammatory, are primarily used to treat acute pain.^[2,4] Drugs such as corticosteroids and analgesics can relieve palsy, and if it is secondary to diseases such as Ramsay James Hunt syndrome and Lyme disease, acyclovir, and antibiotics like amoxicillin can cause relief to the patient.^[12] Surgical options such as facial nerve decompression, nerve grafting, subcutaneous electrical nerve stimulation and myringotomy, ventilation tube, and or cortical mastoidectomy have also been advised.^[13]

Vaccines with 99% effectiveness against varicella infection provide active immunity.^[14] Depending on the amount of lagophthalmos (inability to completely close the eye) and the presence of paralytic ectropion and Bell's phenomenon, the patient management should focus on limiting corneal exposure. Corneal protection is critical, and this

necessitates ocular lubrication with ophthalmic ointments, artificial tears, or moist chambers.^[15] In addition, bedtime eye taping is advised. Other temporary options include upper lid botulinum toxin A injection, temporary tarsorrhaphy, and/or punctual occlusion.^[16]

Physical therapy, specifically NMR, is an effective treatment for Bell's palsy and can be used in conjunction with other interventions, such as botulinum toxin injections.^[17] Physical therapy and medical and/or surgical interventions are best delivered in a closely integrated multidisciplinary setting.

CONCLUSION

The prodromal indications of herpes zoster of the trigeminal and facial nerve must be recognized by the practicing dentist, who should be conversant with the presenting signs and symptoms.

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