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## CASE REPORT

### RAMSAY HUNT SYNDROME: A CASE REPORT

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Ramsay Hunt syndrome, varicella-zoster virus, facial paralysis, vestibulocochlear dysfunction, case report.

#### Abstract

**Background:** Ramsay Hunt syndrome (RHS) results from reactivation of the varicella-zoster virus (VZV) in the geniculate ganglion of the facial nerve. It typically presents as peripheral facial palsy associated with erythematous vesicular eruptions of the external ear or oral mucosa, and may involve cochleovestibular dysfunction.

**Case Presentation:** A 56-year-old male with poorly controlled type II diabetes presented with right peripheral facial palsy (House-Brackmann grade IV) associated with vesicular lesions on the auricular concha (Ramsay Hunt zone). The patient reported hearing loss and vertigo. Clinical examination showed a horizontal-rotatory nystagmus beating to the left. Videonystagmography revealed a 58% right vestibular deficit on caloric testing. The Video Head Impulse Test (vHIT) demonstrated bilateral posterior canal involvement and right lateral canal hypofunction. Vestibular Vibration Stimulation (VVS) was pathologic at 16° on the right side. Early combined therapy with acyclovir and corticosteroids was initiated. The vesicular lesions resolved, and hearing partially improved. However, vertigo persisted with covert saccades on vHIT, and no facial recovery was observed after two months of follow-up.

**Conclusion:** This case illustrates an atypical form of Ramsay Hunt syndrome with non-systematized vestibular involvement and poor facial nerve recovery despite early antiviral and steroid therapy. Rapid diagnosis and prompt management remain crucial to improving functional outcomes.

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#### Introduction:-

Ramsay Hunt syndrome, first described in 1907, is caused by the reactivation of the varicella-zoster virus in the geniculate ganglion of the facial nerve. It typically presents as a peripheral facial palsy accompanied by vesicular eruptions of the auricle or oral cavity. When associated with cochlear and vestibular dysfunction, it constitutes the so-called 'otic' or 'total' form of herpes zoster oticus (1). RHS is the second most common infectious cause of peripheral facial paralysis after Bell's palsy. The prognosis for facial nerve recovery is poorer, particularly in elderly or immunocompromised patients. Early antiviral therapy and corticosteroid administration are essential to reduce viral replication and inflammation (2).

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**Materials and Methods / Case Presentation:-**

A 56-year-old male, with a history of poorly controlled type II diabetes, presented to the ENT department with right-sided peripheral facial palsy (House–Brackmann grade IV). Examination revealed erythematous vesicular eruptions of the right auricle, corresponding to the Ramsay Hunt zone. The patient complained of hearing loss and rotatory vertigo. Neurological assessment revealed horizontal–rotatory nystagmus beating to the left. Vestibular and Audiological Findings: Videonystagmography (VNG) showed right vestibular hypofunction (58%) on caloric testing. Video Head Impulse Test (vHIT) revealed bilateral posterior canal impairment and right lateral canal hypofunction. Vestibular Vibration Stimulation (VVS) was pathologic at 16° on the right side, reproducible. Treatment and Evolution: Early combination therapy of acyclovir and corticosteroids was initiated. The patient also received symptomatic vestibular treatment and diabetic control optimization. At two-month follow-up: vesicular eruptions resolved completely, hearing improved partially, but vertigo persisted with covert saccades on vHIT and no recovery of facial paralysis.

**Results:-**

The patient experienced partial vestibulocochlear improvement but no facial recovery. This suggests extensive neural damage possibly enhanced by viral spread and underlying metabolic comorbidity. Despite early antiviral therapy, facial prognosis remained unfavorable (3).

**Discussion:-**

Ramsay Hunt syndrome arises from reactivation of latent varicella-zoster virus in the geniculate ganglion, potentially spreading to adjacent cranial nerves (VIII, IX, X). This explains the variety of symptoms, including facial paralysis, hearing loss, and vertigo (1,4). The absence of typical vestibular patterning, as in this case, may reflect diffuse vestibular involvement rather than localized neuritis. The facial nerve prognosis in RHS is significantly worse than in Bell's palsy, with less than 50% of patients achieving full recovery (4,5). Diabetes mellitus and delayed management are recognized poor prognostic factors (6). Studies demonstrate that combined acyclovir–prednisone therapy initiated within 72 hours improves outcomes in both facial and cochleovestibular function (3,7). Recent reviews confirm that early combination therapy remains the gold standard, despite limited randomized controlled data (8,9). The persistent facial paralysis in our case may be explained by underlying diabetic neuropathy, delayed consultation, or extensive viral inflammation involving multiple cranial nerve branches.

**Conclusion:-**

Ramsay Hunt syndrome should be suspected in any facial palsy associated with auricular or oral vesicles. Audiovestibular testing is crucial to assess the full extent of neural involvement. Early antiviral and corticosteroid treatment offers the best chance of recovery, but prognosis remains guarded in patients with comorbidities such as diabetes.

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**Conflict of Interest:**

The author declares no conflicts of interest related to this case report.

**Ethical Statement:**

Written informed consent was obtained from the patient for publication of this case report and accompanying images. The study complies with institutional ethical standards and the Declaration of Helsinki.

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