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Case Report

Challenging the norm- unmasking ramsay hunt syndrome: A case without classic otologic features

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Abstract

Ramsay Hunt syndrome is a peripheral facial nerve palsy which is accompanied by erythematous vesicular rash on the ear (Zoster Oticus) or in the mouth. Another common symptom of this syndrome is persistent and excruciating ear ache that usually precedes the facial nerve palsy. Other symptoms include tinnitus, vertigo and sensorineural hearing loss. Hereby, reporting a case of 44year old non-diabetic, non-hypertensive female who presented with pain and swelling in the left parotid region followed by left facial nerve palsy, skin changes over the concha. There was improvement of facial nerve palsy, parotid swelling after 2 weeks of conservative management.

Keywords: Ramsay Hunt syndrome, Facial nerve paralysis, Vertigo, Parotid swelling

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1. Introduction

Ramsay Hunt Syndrome (RHS) is a rare manifestation of reactivated varicella-zoster virus (VZV), leading to inflammation of the geniculate ganglion of the facial nerve (cranial nerve VII).[1] Also termed herpes zoster oticus, the condition was first described by James Ramsay Hunt in 1907.[2] VZV initially causes chickenpox, commonly seen in children presenting with fever and diffuse vesicular rash, and later remains dormant in cranial or spinal nerve ganglia. Reactivation, often triggered by immune compromise results in herpes zoster, a painful vesicular eruption along a specific dermatome. When the facial nerve is involved, the result is Ramsay Hunt syndrome, accounting for less than 1% of all zoster cases.[3]

RHS classically presents with a triad: unilateral facial paralysis, otalgia, and a vesicular eruption. However, presentations can vary. In some cases, paralysis may precede the rash or the rash may be entirely absent—termed zoster sine herpete.[4] This variant poses diagnostic challenges, as it mimics idiopathic facial nerve palsy (Bell's palsy). Up to 30% of RHS cases are believed to fall under this atypical category.[5]

Additional symptoms may include taste disturbances, dry eye, lacrimation, hyperacusis, nasal congestion, dysarthria, vertigo, tinnitus, and hearing loss, hoarseness of voice, aspiration depending on the extent of cranial nerve involvement, especially if cranial nerves V, VIII, IX, and X, XI, XII are affected- cranial polyneuropathy.

2. Case Report

A 44-year-old woman with no known comorbidities presented with pain in the left parotid region lasting three days, followed by diffuse swelling in the same area for two days. Subsequently, she developed left-sided facial weakness, difficulty in left eye closure, deviation of the mouth to the right, and drooling. She also reported low-grade fever one day prior to facial symptoms. No prior treatment was initiated.

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On clinical examination, she was alert, febrile, and hemodynamically stable. A 4 x 3 cm diffuse, tender, firm swelling was palpable in the left parotid region. It was non-fluctuant and non-translucent, with overlying skin remaining unaffected. The ear lobule appeared displaced anteriorly. There was facial nerve paralysis of grade 3 (House-Brackmann classification), as the patient developed deviation of angle of mouth (**Figure 1** a), incomplete eye closure, absence of wrinkling in the forehead (**Figure 2** b), weakness on left cheek muscle (**Figure 1** c) after a day or two following facial swelling. She also developed skin changes (rugosities with discoloration) over the conchal cartilage of the left ear. After 2 days of admission she developed giddiness, on and off, self-spinning type, lasting for few minutes (2-3 mins), not associated with nausea, vomiting, photophobia, postural variation, no specific aggravating factors, relieved with medications. Examination of oral cavity was normal. Complete blood count revealed leukocytosis (7000uL.)

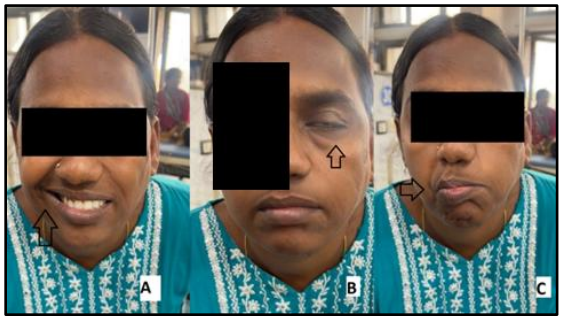

Figure 1: **A)** Patient with deviation angle of mouth, **B)** incomplete eye closure, absence of wrinkling in the forehead, **C)** weakness on left cheek muscle after 2 days

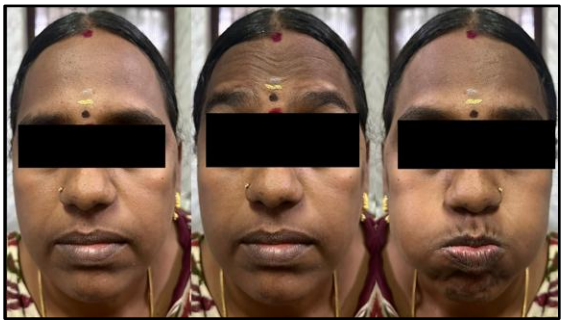

Figure 2: Reduction in the swelling in the parotid region and improvement in the facial nerve palsy after 2 days

Patient was treated conservatively with broad spectrum antibiotics (Inj. Ceftriaxone 1gm, Inj. Metronidazole 500 mg, Inj. clindamycin 600 mg) and oral steroids (T. Methylprednisolone 16mg). Ultrasonography was done which showed sub centimetric lymph nodes with maintained fatty hilum at the level of IA, bilateral level II, III, largest SAD measuring 4.7 mm in left level II. Left intra parotid lymph nodes were also noted. Patient also underwent facial physiotherapy regularly. There was reduction in the swelling in the parotid region and improvement in the facial nerve palsy (**Figure 2** a, b, c) after 2-3 days (House Brackmann grade 2). Patient was called for follow up after 1 week. There was complete recovery of facial nerve palsy, facial swelling and other associatedsymptoms after 2 weeks of conservative management.

3. Discussion

Post-primary VZV infection, the virus enters a latent phase in sensory and motor ganglia. Reactivation, commonly associated with reduced immunity can lead to RHS, typically affecting older adults with comorbidities. The syndrome's hallmark features include peripheral facial paralysis and a vesicular rash involving the auricle or ear canal, though atypical cases without rash (zoster sine herpete) are increasingly recognized.

This case is unusual in that facial nerve involvement was preceded by parotid swelling and skin discoloration, without the characteristic vesicular rash or otalgia. Neurological symptoms such as vertigo and subtle dermatological changes further complicated the clinical picture.

The mainstay of RHS treatment involves antiviral therapy (e.g., acyclovir, valacyclovir, famciclovir) alongside corticosteroids, ideally started within 72 hours of symptom onset. This approach has been shown to significantly improve facial nerve recovery and reduce complications like postherpetic neuralgia. Although acyclovir is commonly used, studies suggest that famciclovir may offer greater efficacy in some cases. The antivirals of choice are Acyclovir, Valacyclovir and Famciclovir. Acyclovir 500 mg fivetimes a day is the most preferred. Whereas Famciclovir, 500mg three times a day is more effective than Acyclovir, Valacyclovir-100 mg,[6] three times a day is more effective in Bell's palsy Corticosteroids such as prednisolone (1 mg/kg/day) are recommended to reduce inflammation and nerve damage. Proton pump inhibitors should be co-prescribed to prevent gastrointestinal side effects from steroids.

Notably, early initiation of combined acyclovir-prednisone therapy improves outcomes. One retrospective analysis of 80 patients demonstrated a 75% complete recovery rate when treatment began within three days of symptom onset, compared to only 30% when initiated after seven days.[7] Topical steroid delivery through the tympanic membrane has also been explored in patients with dehiscent facial nerve segments.[8]

Supportive care, including analgesics (NSAIDs, opioids), antidepressants for neuropathic pain, and ocular protection with lubricants and eye patching, plays a key role in management.

Despite not receiving antiviral therapy, our patient improved significantly with antibiotics, steroids, and physiotherapy, suggesting that early anti-inflammatory intervention and symptomatic treatment may be sufficient in some atypical presentations.

4. Conclusion

Ramsay Hunt Syndrome is typically characterized by peripheral facial nerve palsy and auricular rash, resulting from reactivation of latent VZV. However, this case underscores the variability in clinical presentation. The patient, a middle-aged woman without comorbidities, presented initially with parotid swelling and no vesicular rash or otalgia, hallmarks of classical RHS.

Prompt initiation of steroids and antibiotics, along with physiotherapy, led to complete recovery within two weeks, without the need for antivirals. This highlights the importance of clinical vigilance and flexibility in diagnosing and managing atypical RHS, especially in the absence of characteristic dermatological signs.

5. Patient consent

Written and informed patient consent has been taken prior to the publication.

6. Ethical Approval

This study was approved by Institute ethical committee with IHEC NUMBER: MGMCRI/2024/04/IHEC/CS/44

7. Source of Financial

None.

8. Conflict of Interest

None.

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