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Case Study

Ramsay Hunt Syndrome in Patients with Underlying Metabolic Disorders: A Case Series of Two Clinical Presentations

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Abstract

Herpes zoster results from reactivation of latent Varicella-Zoster Virus (VZV), typically presenting with a dermatomal vesicular rash and possible post-herpetic neuralgia. Ramsay Hunt syndrome (RHS), a rare form of VZV reactivation involving the geniculate ganglion, presents with unilateral peripheral facial palsy and a vesicular ear rash. Prompt diagnosis is crucial to differentiate it from other neurological conditions. It commonly affects the elderly or immunocompromised. Herein we describe a case series of two cases with atypical RHS presentation, featuring isolated peripheral facial paralysis and a vesicular rash in the retroauricular area with metabolic disorders.

Keywords: Varicella-Zoster Virus; Ramsay Hunt Syndrome; Retroauricular Area; Vesicular Rash; Hypothyroidism; Diabetes Mellitus

Introduction

Ramsay Hunt syndrome (RHS), also known as herpes zoster oticus, occurs when the varicella-zoster virus (VZV) reactivates within the geniculate ganglion of the seventh cranial nerve. Ramsay Hunt syndrome affects both immunocompetent and immunocompromised individuals, with an annual incidence of approximately 5 per 100,000, compared to 15–30 per 100,000 for Bell's palsy. It accounts for around 7% of acute facial paralysis cases, with zoster sine herpete representing up to 30% of these. It typically presents with a combination of three key symptoms: intense, one-sided ear pain, a rash of blisters appearing in or around the ear or in the mouth, and weakness or paralysis of the facial muscles on the same side. The condition can also lead to other issues such as ringing in the ears, hearing loss, dizziness, altered taste, and dry mouth, due to the virus spreading to nearby cranial nerves (V, VIII, IX, X). RHS is more common in older individuals and those with weakened immune systems.

Initiating combined steroid and antiviral therapy within 72 hours significantly improves recovery to House-Brackmann grade I–II, with a success rate of approximately 75% compared to 48% without early treatment [1].

1st Case

A 58-year-old female, with a 17-year history of hypothyroidism, has consistently adhered to their medication regimen along with history of psoriasis managed with methotrexate for the past three years, presented to the emergency department complaining of right ear pain of three days duration. This was accompanied by a painful, erythematous, vesicular rash on the right pinna. One day prior to presentation, she developed right-sided facial nerve palsy. Associated symptoms included nausea, vomiting, giddiness and burning sensation of tongue. She also had history of fever 5 days prior to onset of symtptoms. Based on these clinical findings, a diagnosis of Ramsay Hunt Syndrome was made.

Upon examination, vesicular lesions were observed on the patient's right pinna and external auditory canal. The tympanic membrane was only partially visualized. Neurological assessment revealed a right facial nerve palsy, characterized by noticeable facial asymmetry. The patient was unable to close her right eye, exhibiting a positive Bell's phenomenon (upward and outward rotation of the eyeball when attempting to close the eye). Additionally, there was drooping of the right angle of the mouth, and forehead wrinkles were absent on the affected side - Moderately severe facial nerve palsy classified as Grade 4 on the House-Brackmann scale.

The patient was started on a combination therapy of antivirals and corticosteroids, including oral Valacyclovir 1 g thrice daily, intravenous Piperacillin-Tazobactam 4.5 g every 8 hours, and intravenous Dexamethasone 1 cc. Lid taping was done to prevent exposure keratitis. Analgesics were prescribed for neuralgic pain, and adequate hydration was advised. Physiotherapy was initiated. The patient showed symptomatic improvement.

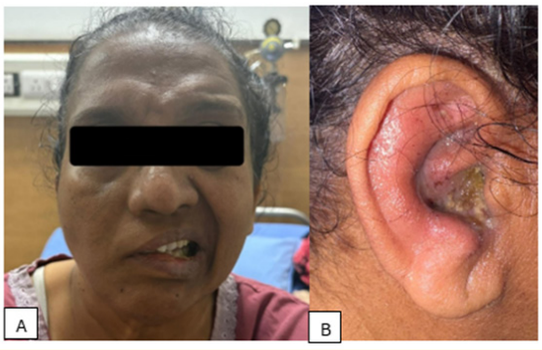

Figure 1: A - Right Facial palsy characterized by absence of forehead wrinkling, deviation of angle of mouth.
B - Grouped vesicles on patients right ear.

2nd Case

Subsequently, a 62-year-old female with a known history of hypertension and type 2 diabetes mellitus presented with a 7-day history of right ear pain associated with a painful vesicular rash over the right pinna.

On examination, multiple vesicular lesions were noted on the right pinna consistent with herpes zoster oticus. There were no signs of facial nerve palsy or other cranial nerve involvement. The patient was initiated on a combination of symptomatic and targeted therapy, including analgesics, intravenous piperacillin-tazobactam (4.5 g every 8 hours), oral valacyclovir (three times daily), and intravenous dexamethasone (1 cc). Metabolic control was optimized. The patient showed significant symptomatic improvement over the subsequent days.

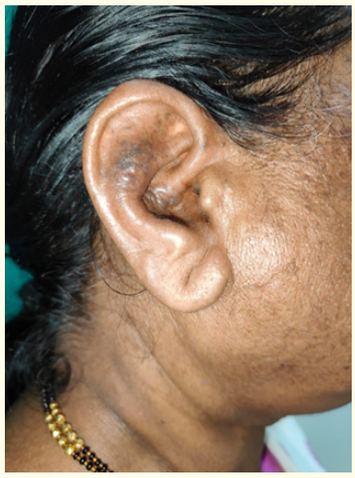

Figure 2: Vesicles on patients right ear.

Discussion

Both the cases highlights the presentation of Ramsay Hunt Syndrome (RHS) in an immunosuppressed individual. RHS is recognized as a more severe variant of facial nerve palsy compared to Bell's palsy, often leading to less complete recovery of facial function. Prompt initiation of antiviral and steroid therapy is crucial for improving outcomes, as delays can lead to poorer prognosis. It is important to note that immunocompromised individuals, including those with hypothyroidism and diabetes mellitus, are at increased risk for more frequent and severe presentations of RHS.

Ramsay Hunt Syndrome is characterized by a distinctive constellation of symptoms resulting from varicella-zoster virus reactivation within the geniculate ganglion. The classic involvement, often referred to as the "Ramsay-Hunt area," encompasses the eardrum, external auditory canal, and the cavum conchae (the central portion of the ear), where vesicular lesions are typically observed.

Furthermore, co-involvement of the acoustic (CN VIII) and vestibular nerves can lead to symptoms such as vertigo and hearing loss. Less commonly, vesicles may also appear on the tonsillar pillars, posterior pharyngeal wall, and laryngeal mucosa, indicating involvement of the glossopharyngeal (CN IX) and vagus (CN X) nerves.

Malin., *et al*. further define RHS by the presence of zoster oticus alongside peripheral facial nerve paresis. This often includes disturbances in taste sensation (due to involvement of the chorda tympani and greater petrosal nerves, affecting the anterior two-thirds of the tongue and palate), reduced tear secretion, and sensory disturbances within the innervation territory of the trigeminal nerve and cervical dermatomes.

Coulson., *et al*. [2] found that all HZO patients showed some facial function improvement, with the best outcomes in those treated with combined antivirals and steroids within 5 days. Recovery was poorer in patients with initial incomplete eye closure and dry eye.

Shingo Murakami., *et al*. [3] demonstrated that early acyclovir-prednisone therapy reduced nerve degeneration and improved hearing recovery. Facial nerve outcomes were similar between oral and intravenous acyclovir.

The extensive sensory territories involved in RHS, stemming from the geniculate ganglion's connection to various cranial nerves, underscore the multifaceted clinical picture of this syndrome.

Elsheref., *et al*. proposed that subclinical hypothyroidism may be a contributing factor in the reactivation of herpes zoster, suggesting that thyroid hormone therapy could be an effective component of treatment in such cases [4].

In a pioneering study, Ajavon et al. suggested that thyroid hormone (TH) levels may play a pivotal role in the pathogenesis of herpes zoster following varicella-zoster virus (VZV) reactivation. They recommended that TH levels be routinely evaluated as a potential biomarker in cases of recurrent VZV infection [5].

Autoimmune hypothyroidism, such as Hashimoto's thyroiditis, may indicate an underlying dysregulation of the immune system, which can predispose individuals to viral reactivations—including reactivation of varicella-zoster virus (VZV), the pathogen responsible for Ramsay Hunt Syndrome (RHS).

Conclusion

Early recognition and prompt treatment with antivirals and corticosteroids are critical for improving facial nerve recovery and reducing complications in Ramsay Hunt syndrome. Accurate clinical diagnosis, timely intervention, and close follow-up are essential for optimal outcomes. Emphasis on differential diagnosis and standardized treatment protocols can further enhance patient recovery and prevent long-term sequelae. Combined acyclovir and high-dose corticosteroids, alongside well-controlled blood sugar, significantly enhance facial nerve functional recovery in diabetic patients with Bell's palsy. Although hypothyroidism is not recognized as a direct risk factor for Ramsay Hunt Syndrome (RHS), shared factors such as immune dysregulation, advanced age, and associated metabolic comorbidities may contribute to increased vulnerability or impact recovery outcomes. While a definitive link between hypothyroidism and RHS has not been clearly established, certain indirect associations and clinical implications warrant consideration. Optimizing the management of underlying metabolic conditions is essential for enhancing the clinical prognosis and recovery in patients with Ramsay Hunt Syndrome.

Conflicts of Interest

None.

Consent

Informed consent obtained from the patients for publication of this case report and any accompanying images.

Acknowledgements

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