

Ramsay Hunt Syndrome

Sir,

Ramsay Hunt Syndrome (RHS), also termed as Hunt's Syndrome and herpes zoster oticus, was first described by James Ramsay Hunt in 1907.¹ It is a rare neurological syndrome caused by varicella zoster virus. RHS has unilateral facial weakness and painful blisters in the ear canal leading to auditory loss and vestibular symptoms. It occurs due to reactivation of herpes zoster virus that has previously caused the infection. It is a rare complication of primary varicella zoster infection. Unilateral facial paralysis, otalgia, and vesicular rash of external auditory canal constitute the main triad of the disease. The vestibulocochlear nerve is most commonly involved nerve.²



Figure 1: The left side of the face shows multiple blisters and ulcers.



Figure 2: The area of the left ear shows vesicular rash around auricular region.



Figure 3: The lower lips and lower part of the face show painful blisters.

A 62-year male came to the Department of Oral Medicine and Radiology with a chief complain of multiple blisters and ulcers in left side of the face, chin and ear (Figure 1). He had reduced hearing and taste sensation along with vesicular eruptions, which ruptured and became ulcerated in appearance. There was no relevant medical history and no deleterious habits. Clinical examination revealed a vesicular rash around auricular region of the left ear (Figure 2). The pain was described as burning sensation around left ear. Painful blisters were present not only on left external ear and external auditory canal, but also involved lower lips and lower part of face on left side (Figure 3). Due to involvement of vestibulocochlear nerve, patient had vertigo, sensorineural hearing loss, and tinnitus. Intraoral examination showed poor oral hygiene with periodontitis. Based on a detailed patient history, thorough clinical evaluation and identification of characteristic symptoms, it was diagnosed as RHS. Early diagnosis and treatment with combination of acyclovir and corticosteroids improved the condition. Acyclovir, 10 mg/kg/day, and prednisolone, 1 mg/kg/day, were advised for 7 days.

RHS is characterised by cutaneous lesions of external auditory canal with involvement of the ipsilateral facial and auditory nerves. RHS is caused by reactivation of the varicella zoster virus within the sensory ganglion of the facial nerve. As vestibulocochlear nerve lies near the sensory ganglion of the facial nerve within the facial canal, both the nerves are affected. This leads to facial paralysis along with hearing deficits, vertigo, and other auditory and vestibular symptoms. Patients may develop loss of taste mainly in anterior two-thirds of the tongue. Most of these neuralgias resolve within 1 year. Symptoms may also include vomiting, hearing loss, hyperacusis, tinnitus, eye pain and lacrimation.

Corticosteroids and acyclovir are main line of treatment of RMS. Supportive therapy includes non-aspirin antipyretics. Antiviral drugs reduce mucocutaneous lesions, which promotes healing after one week. Carbamazepine can be used as a vestibular suppressant to alleviate the symptoms.³ Follow-up should be

done at an interval of 2 weeks, 6 weeks and 3 months. Complications include development of post-herpetic neuralgia, unresolved facial paralysis and herpes zoster encephalitis.⁴ Patients with no hypertension and diabetes have higher chances of complete recovery.⁵ Early diagnosis and treatment are important for improvement of the involved nerves' function.

PATIENT'S CONSENT:

Informed consent was obtained from the patient to publish the data concerning this case.

CONFLICT OF INTEREST:

The author declared no conflict of interest.

AUTHOR'S CONTRIBUTION:

SS: Contributed to the conception of the work, collected the patient data and images and participated in literature review and authored the full manuscript.

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