ABSTRACT

Ramsay Hunt syndrome (RHS) is defined as an acute peripheral facial neuropathy caused by the reactivated latent varicella zoster virus (VZV) in the geniculate ganglion; characterized with erythematous vesicular rash of the skin of the ear canal, auricle, facial skin, oral mucosa and facial palsy (also known as herpes zoster oticus). This article reports a case of Ramsay Hunt Syndrome (RHS) in a 37-year-old male patient depicting the classical signs.

Keywords: Varicella zoster, Geniculate ganglion, Vesicles, Facial palsy.

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INTRODUCTION

Ramsay Hunt syndrome (RHS) is defined as an acute peripheral facial neuropathy associated with erythematous vesicular rash of the skin of the ear canal, auricle (also termed herpes zoster oticus), and/or mucous membrane of the oropharynx. RHS was first described in 1907 by James Ramsay Hunt in a patient who had otalgia associated with cutaneous and mucosal rashes, which he ascribed to the infection of geniculate ganglion by human herpes virus 3 (i.e. varicella zoster virus [VZV]). This syndrome is also known as geniculate neuralgia or nervus intermedius neuralgia. Primary infection of VZV, also known as varicella or chickenpox, is a common pediatric erythematous disease.

VZV remain latent in neurons of cranial nerve and dorsal root ganglia, subsequent reactivation can result in prodromal period of severe pain followed by localized vesicular rash, known as herpes zoster (HZ).

Classic RHS usually present with paroxysmal pain deep within the ear, often radiates outward into the pinna and may be associated with a more constant, diffuse and dull background pain. The onset of pain usually precedes the rash by several hours and even days. Vesiculation and ulceration of the external ear and ipsilateral anterior two-thirds of the tongue and soft palate are common (as many as 80% of cases). The rash might precede the onset of facial paresis/palsy; other manifestations include vertigo and ipsilateral hearing loss (CN VII), tinnitus, otalgia, headaches, dysarthria, gait ataxia, fever, cervical adenopathy.

CASE REPORT

Here, we present a 37-year-old male patient who reported with clusters of vesicles in the skin over the left side of the face, following removal of carious left mandibular third molar (38). Patient also complained of fever, fatigue, myalgia, facial and ear pain, altered taste perception. The vesicles were present unilaterally in the facial skin along the left mandibular dermatome (Fig. 1). External auditory canal and tragus of left ear also showed similar lesions. Intraorally multiple ulcers of varying size distributed over the left buccal mucosa, left half of the labial mucosa, tongue and palate (Fig. 2) The case was provisionally diagnosed as herpes zoster involving mandibular and maxillary (palate) dermatome. Patient was prescribed tablet acyclovir 800 mg five times a day for 5 days, tablet paracetamol 500 mg sixth hourly for 3 days, chlorhexidine mouthwash and topical...
Ramsay Hunt Syndrome (Herpes Zoster Oticus)

Fig. 3: Facial view before and after facial palsy: Patient face showing features of complete peripheral facial nerve palsy of left side showing lack of wrinkling of forehead, inability to close left eyelid and deviation of angle of the mouth to the normal (right) side suggestive of left peripheral facial nerve paralysis and healed lesions in skin showed hyperpigmentation (Fig. 3). The diagnosis was revised as RHS. Patient was advised physiotherapy for facial palsy and showed good recovery.

DISCUSSION

RHS is estimated to account for 18% of facial palsies in adults. Early initiation of antiviral treatment and adjuvant steroid therapy can prevent the occurrence of facial paralysis. Poor prognostic factors for good functional recovery of facial nerve include patient older than 50 years, complete facial paralysis and lack of CN VII nerve excitability. Approximately 25% of patients with RHS experience vestibular or cochlear symptoms or both; apparently caused by the spread of inflammatory cells from geniculate ganglion to inner ear, spiral or vestibular ganglion. In one-fourth of patient, symptom also includes vertigo, nystagmus, tinnitus and hearing loss. Sensorineural hearing loss occurs in only approximately 6% of patients. Audiograms vary and suggest primary cochlear involvement or cochlear nerve damage. In our case, there was no history of deafness or vertigo. Examination revealed normal tuning fork test and normal pure tone audiogram (Fig. 4), thereby ruling out involvement of auditory apparatus. There was no feature suggestive of vestibular involvement which was evident by the absence of vertigo and nystagmus. This case was presented for its rarity and classical clinical presentation. Similar to our case many of the patients with HZ in the head and neck region undergo unnecessary extraction/endodontic treatment for many teeth during the prodromal period with no clinical lesions.

REFERENCES


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