Scientific Research Publishing

International Journal of Otolaryngology and Head & Neck Surgery, 2026, 15(3), 209-216
https://www.scirp.org/journal/ijohns
ISSN Online: 2168-5460
ISSN Print: 2168-5452

Ramsay Hunt Syndrome Revealed by a Severe Vertigo

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How to cite this paper: Ndiaye, C.B., Diallo, T.B., Diouf, K.A., Diop, M., Touré, A., Ndiaye, M.R., Diagne, C.L., Danfa, C., Lame, C.A. and Loum, B. (2026) Ramsay Hunt Syndrome Revealed by a Severe Vertigo. *International Journal of Otolaryngology and Head & Neck Surgery*, **15**, 209-216.
https://doi.org/10.4236/ijohns.2026.153019

Received: February 3, 2026
Accepted: May 8, 2026
Published: May 11, 2026

Abstract

Ramsay hunt syndrome (RHS) also called herpes zoster oticus, is a clinical presentation of varicella-zoster virus (VZV) reactivation that has remained dormant within geniculate ganglion, after primary varicella infection. Sicard syndrome is the most complete presentation of RHS with 7th and 8th cranial nerve involvement. Diagnosis of RHS is clinical, but further investigations may be necessary for atypical symptoms. We report a 43-year-old woman, referred from the emergency department for a disabling acute vertigo, ongoing for the last 24 hours. The history revealed right otalgia, hearing loss and facial asymmetry that appeared 3 days ealier. Physical examination found a right-sided peripheral facial palsy, right-sided peripheral vestibular syndrome and painful vesicular lesions around the ipsilateral ear in Ramsay Hunt area. Tonal audiometry noted severe right-sided sensorineural hearing loss, and endocochlear impairment on auditory evoked potentials. Temporal bone MRI showed contrast enhancement of the right acoustic facial bundle, the internal auditory canal, and slight contrast of the ipsilateral cochlea and vestibule. Diagnosis of complete RHS also known as Sicard syndrome was confirmed. Treatment, including combined antiviral agents and intravenous corticosteroids was initiated with good outcome. However, mild sensorineural hearing loss was noted after an 8-month follow-up. RHS is a relatively rare condition in our practice. Early and appropriate management could prevent potentially serious complications and limit functional sequelae.

Keywords

Ramsay Hunt Syndrome, Sicard Syndrome, Peripheral Facial Palsy, Vertigo, Hearing Loss

1. Introduction

Ramsay Hunt syndrome (RHS) also called herpes zoster oticus, is a clinical manifestation of reactivation of the latent varicella-zoster virus (VZV) in the geniculate ganglion, after a primary varicella infection. It is frequently associated with acute peripheral facial palsy, a vesicular eruption of the ear, and sensorineural hearing loss, thus affecting the seventh and eighth cranial nerves [1] [2].

Sicard syndrome, which is less common, is the most typical and complete presentation of RHS. It is an inflammation of all elements of the acoustic facial bundle [3]. However, involvement of other cranial nerves, particularly the glossopharyngeal and vagus nerves has been described [4].

Diagnosis of RHS is essentially clinical, based on a triad of otalgia, ipsilateral peripheral facial palsy, and a painful vesicular skin rash on the auricle, palate or tongue [5].

Cerebrospinal fluid (CSF) changes, multiple cranial neuropathies and aseptic meningitis are the main neurological complications of this condition [6]**.** We report a case of complete Ramsay Hunt syndrome or Sicard syndrome, revealed by a severe vertigo.

2. Case Report

A 43-year-old woman, hypertensive, referred from the emergency department for a disabling acute vertigo, ongoing for the last 24 hours. The history revealed a right otalgia, right hearing loss and facial asymmetry that appeared 3 days ealier. She underwent unspecified outpatient medical treatment without improvement.

ENT clinical examination found a right-sided peripheral facial palsy, grade III of House-Brackmann scale (**Figure 1**), a right-sided peripheral vestibular syndrome with severe vertigo and left beating horizonto-rotatory nystagmus, painful vesicular lesions around the ipsilateral ear, in Ramsay Hunt area (**Figure 2**). No other cranial nerve impairment was noted.

Figure 1. Patient presenting with right peripheral facial palsy.

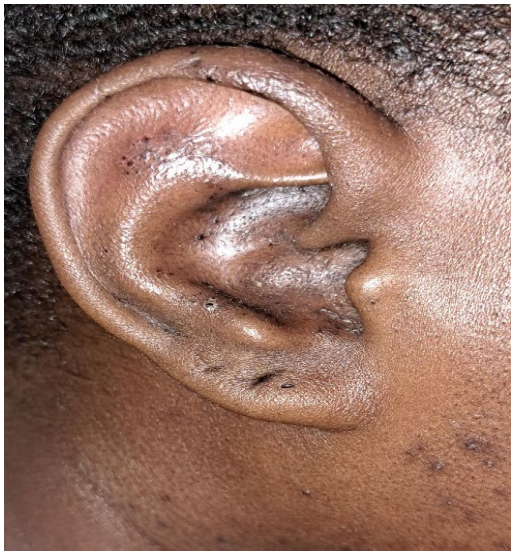


Figure 2. Vesicular rash on the right auricle in Ramsay Hunt area.

The eye exam revealed right lagophtalmos and no exposure keratitis complications.

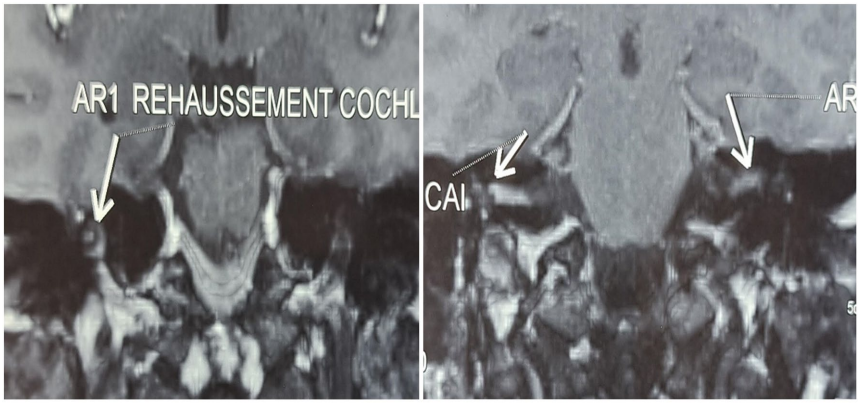
The tympanogram was normal, and the stapedial reflex was absent on the right side.

Tonal audiometry noted severe right-sided sensorineural hearing loss, predominantly in the high frequencies.

Auditory evoked potentials were suggestive of an endocochlear lesion.

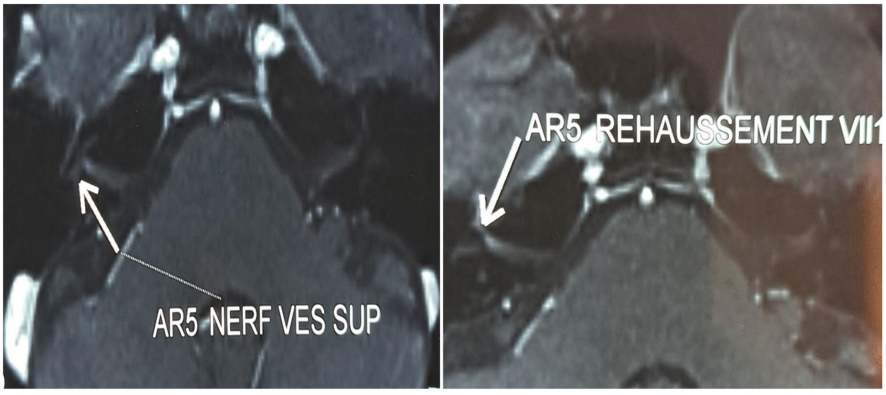
No abnormality was found on the brain CT scan.

Temporal bone MRI showed a right-sided labyrinthitis, associated with right vestibular and facial neuritis, which manifested as a contrast enhancement of the 3 facial nerve segments, the right internal auditory canal, and slight contrast of the ipsilateral cochlea and vestibule (**Figure 3**, **Figure 4**).



(a) (b)

Figure 3. Coronal view MRI of the temporal bone showing enhancement of the right cochlea (a) and internal auditory canal (IAC) (b).



(a) (b)

Figure 4. Axial view MRI of the temporal bone revealing contrast enhancement of the right superior vestibular nerve (a) and the first intrapetrous segment of the right facial nerve (b).

Medical treatment including 5 days of parenteral corticosteroids, followed by 10 days oral switch with tapering doses, combined with oral antiviral (acyclovir) drugs for 3 weeks, was initiated. Analgesics and ocular protection measures were also started. The patient's condition improved with complete resolution of vertigo, within 3 days of hospitalization, and significant regression of peripheral facial palsy from grade III to grade II, observed on day 8 of admission. However, mild sensorineural hearing loss was noted after an 8-month follow-up. Hearing aid rehabilitation is planned to our patient.

3. Discussion

Ramsay Hunt syndrome (RHS) also known as herpes zoster oticus, is first described by the neurologist Ramsay Hunt in 1907. It is caused by reactivation of latent varicella-zoster virus (VZV) that has remained dormant within the geniculate ganglion of the facial nerve, after primary varicella infection [7] [8].

This condition represents less than 1% of herpes zoster cases [7]. Its incidence, which is 5 per 100000 people, and its severity would increase with age and immune status [4] [9]. RHS is more frequently reported in adults over 50 years of age and immunocompromised patients [10].

Facial nerve impairment is associated with cochlear and vestibular involvement in approximately 25% of cases, due to the extension of the inflammation from the geniculate ganglion to the inner ear, affecting the spiral and Scarpa ganglia. The concomitant involvement of these three ganglia, corresponds to Sicard syndrome [11]. Our patient presented with signs related to the involvement of these three nerve structures.

Transmission of VZV infection via the oval or round window from a dehiscent facial canal, is being considered as possible route of spread to the inner ear. For interneuronal transmission, extension of VZV infection to perineural tissues in the internal auditory canal is probable [7].

Immunosuppression resulting from chronic diseases, radiotherapy, chemotherapy, human immunodeficiency virus (HIV) infection, advanced age and evi-

dence of psychological or physical trauma, are the main risk factors for reactivation of latent varicella zoster virus in the geniculate ganglion [3] [12]. A chronic stress condition was observed in our patient.

RHS can develop without a vesicular skin rash, and it can also be complicated by cranial polyneuropathy [6] [8]. Glossopharyngeal and vagus nerves are the most frequently affected at this stage [2].

In the case of RHS with fever and multiple associated cranial neuropathies, central nervous system involvement such as meningitis or encephalitis could be suspected [13].

Hearing and vestibular impairments can occur without facial palsy and eruptive syndrome, suggestive of sudden deafness as a differential diagnosis [14].

The differential diagnosis of a subacute history of multiple cranial nerve involvement is broad, ranging from infectious and inflammatory etiologies to malignant ones, requiring imaging investigation and probably cerebrospinal fluid tests [5].

RHS diagnosis is clinical, but can be facilitated by laboratory examination [8] [12].

The typical clinical presentation includes unilateral peripheral facial palsy, otalgia, ipsilateral vesicular lesions of the external auditory canal and auricle, and vestibulocochlear dysfunction [1] [15].

Severe forms of herpes zoster oticus are common in immunocompromised patients and elderly people; however, a few severe cases in immunocompetent adults has been reported [3] [12].

Varicella zoster virus can be detected by polymerase chain reaction (PCR) on vesicular eruptions, tears, saliva, or in the cerebrospinal fluid. A fourfold increase in anti-VZV IgG antibody titers in the blood, indicates persistent viral infection, and positive anti-VZV IgM confirms recent infection or reactivation [6] [16].

For hearing assessment, pure tone audiometry rarely finds conductive hearing loss; most often, it is sensorineural deafness, predominantly affecting high frequencies. Cophosis is exceptional. Auditory evoked potentials reveal endocochlear lesions, more rarely retrocochlear impairments [14] [17]. That is in line with our patient findings.

Hearing dysfunction is more severe (in both high and low frequencies) in those with vertigo, compared to patients without vertigo [7] [17].

MRI is not necessary for diagnosis when the clinical presentation is typical, but it finds its full interest in case of unusal clinical manifestations. Enhancement of the intrapetrous facial nerve can be found, particularly in the first segment of the nerve and the geniculate ganglion [11] [18]. Unlike Bell's palsy, in RHS, temporal bone MRI may show not only contrast enhancement of the facial nerve, but also in the vestibulocochlear nerve and dural enhancement along the internal auditory canal, as shown in our study [9] [18].

CT scan is useful to explore the facial bone canal and the mastoid process [14].

The standard treatment of RHS is based on the combination of antivirals (acy-

clovir) for 3 weeks to prevent VZV replication, and systemic steroids (prednisone) due to its anti-inflammatory and anti-edema effects, for 2 weeks. It is also, the first line treatment for herpes zoster associated with multiple cranial nerve involvement [4] [5] [10]. Ocular protection measures (occlusion and humidification) are recommended in case of facial palsy [11].

Early and appropriate administration of antiviral agents and corticosteroids is more effective than using corticosteroids alone [2] [9].

Oral brivudine is as effective as intravenous acyclovir in the treatment of herpes zoster, thus providing more outpatient treatment options [19].

Prevention of herpes zoster virus through vaccination, using a recombinant vaccine with an over all efficiency of around 97%, could significantly reduce the risk of serious complications, thus improving the quality of life in high-risk populations. According to some authors, this vaccine is recommended for adults over 50 years and for people over 19 years of age with at increased risk due to immunodeficiency or immunosuppression [12] [13].

Facial and vestibulocochlear nerve impairment is typical in RHS; however, the occurrence of multiple cranial neuropathy with involvement of trigeminal, glossopharyngeal and vagus nerves is rare, associated with a poor prognosis and systemic complications [13] [15].

Several studies indicate that early diagnosis followed by rapid initiation of treatment within 72 hours, improve the prognosis by limiting functional sequelae. That was noted in our patient [7] [8] [10].

4. Conclusion

Ramsay Hunt syndrome although relatively rare in our practice, occurring most often in patients with specific health conditions, can be observed in immunocompetent people. Early and appropriate management could prevent severe complications and improve facial, hearing and vestibular functional prognosis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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