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Clinical and Therapeutic Features of Herpes Zoster Oticus: A Case Report

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Abstract **Case Report**

Ear shingles, also called herpes zoster oticus or Sicard syndrome, are a viral infection of the outer, middle and/or inner ear due to varicella zoster virus (VZV). Patients with ear involvement associated with facial palsy are clinically diagnosed with Ramsay-Hunt´s syndrome. We here report the case of a 22-year-old patient with herpes zoster oticus associated with peripheral facial palsy, Associated with other signs such as tinnitus, peripheral rotational vertigo, and hypoacusis. The objective of this study is to report our experience in the treatment of sicard syndrome and investigate the various clinical, para-clinical and evolutionary features of herpes zoster oticus and the therapeutic approaches.

Keywords: Herpes zoster oticus, peripheral facial paralysis, Ramsay-Hunt, sicard syndrome, case report.

INTRODUCTION

Auricular herpes zoster, also known as herpes zoster oticus, is a viral infection affecting the external, middle, and/or inner ear. When ear involvement is associated with facial paralysis, it is referred to as Ramsay Hunt syndrome or Sicard syndrome.

Lettule described this condition in 1882, followed by Koener in 1884. However, it was James Ramsay Hunt in 1907 who conducted a detailed study of the syndrome. He was the first to attribute the collection of clinical signs to an involvement of the geniculate ganglion by the varicella-zoster virus (VZV).

It was only in the early 1950s that Weller and Stoddart [1-2], using modern virological techniques, confirmed the relationship between herpes zoster and chickenpox, which had been suspected since the early 20th century.

Clinically, auricular herpes zoster begins with deep, intense, paroxysmal otalgia. Then, a vesicular skin rash appears in the concha and external auditory canal. This rash generally precedes the onset of facial paralysis by 24 to 48 hours. However, in some cases, the rash may be absent, making the diagnosis of auricular herpes zoster more difficult, a condition referred to as zoster sine herpete.

It is like our case and about 25% of cases, cochlear or vestibular involvement is associated (Ramsay Hunt syndrome or sicard syndrome), resulting from the extension of inflammation from the geniculate ganglion to the inner ear, affecting the cochlear and vestibular ganglia [3-4].

Magnetic resonance imaging (MRI) [5], although not necessary for diagnosis, may show contrast enhancement of the facial nerve in the intrapetrous segment, primarily at its first portion and the geniculate ganglion. Some studies have shown a significant benefit of corticosteroid treatment if started early. In contrast, antiviral treatment and/or surgical decompression of the nerve have not proven effective.

Facial paralysis in auricular herpes zoster has a worse prognosis than Bell's palsy, which is associated with herpes simplex virus (HSV). Full recovery occurs in only 16 to 22% of cases, according to some authors, meaning that a mild paresis often persists or synkinesis may develop [6].

CASE REPORT

Mr. F. M, 22 years old, no significant medical history presents to the emergency department in August 2024 at the Hôpital Haute-Saône, vesoul France, for the management of right peripheral facial paralysis associated with vertigo and right hypoacusis. The

symptoms have been evolving for 2 days, preceded by otalgia and right otorrhea.

Clinical Results: On physical examination, the findings included right peripheral facial paralysis, stage IV on the house -Brackmann scale. The Charles-Bell sign was positive, and there was no dissociation between automatic and voluntary movements.

There was inflammatory swelling of the auricle, with vesicles in the concha and tragus (Figure 1). The otoscopic examination revealed an inflammatory external auditory canal and a congested tympanic membrane.

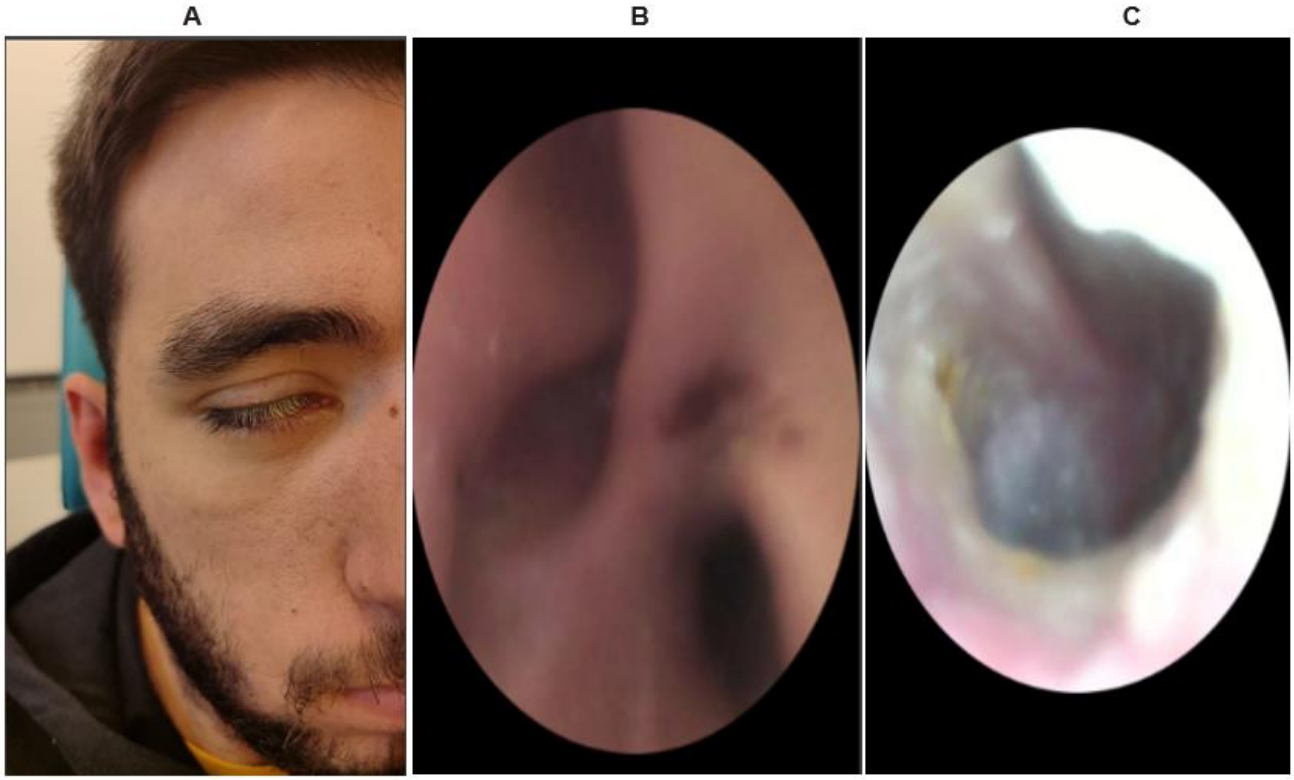


Figure 1: A- Right Peripheral Facial Paralysis B- Herpes Zoster of the Right Ramsay Hunt Region C- congested tympanic membrane

Vestibular Examination: The findings were consistent with acute right peripheral vestibular syndrome, with a harmonious presentation. The postural deviation was to the right (as demonstrated by the index test, Romberg test, and Fukuda stepping test). A pure horizontal nystagmus was present, directed to the left. The Alexandre grade 3 was noted, indicating a significant degree of nystagmus. The nystagmus was inhibited by fixation of the gaze and accentuated during the shaking test.

Ophthalmological Examination: No corneal lesions (keratitis) were observed. The rest of the neurological and somatic examination was unremarkable, with no other abnormalities detected.

Diagnostic Approach: The tonal audiometry revealed right conductive hearing loss at 50 dB (Figure 3-A).

Brain MRI Findings: The MRI showed multifocal contrast enhancement of the right acoustic-facial bundle, which is compatible with right-sided VII (facial) nerve neuritis.

VZV Serology: The VZV (varicella-zoster virus) serology was positive, With associated biological inflammatory syndrome (Figure 2).

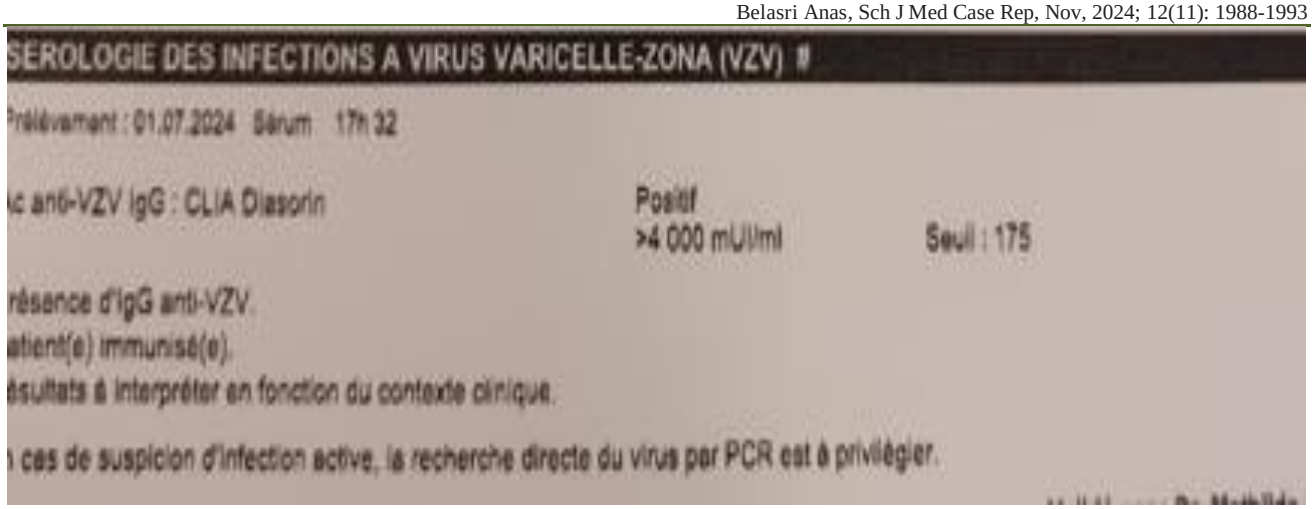


Figure 2: The VZV (varicella-zoster virus) serology

Therapeutic intervention and follow-up: The patient was treated with oral valacyclovir at a dosage of 500 mg tablets, 6 tablets per day divided into 3 doses, for 15 days. This was combined with:

Corticosteroid therapy at 1 mg/kg/day in a short 10-day regimen.
Level 2 analgesics for pain management.
Local auricular and ocular care to prevent exposure keratitis.
Early facial paralysis rehabilitation, with 10 sessions of functional rehabilitation targeting hypertonia and synkinesis.
And vestibular rehabilitation.

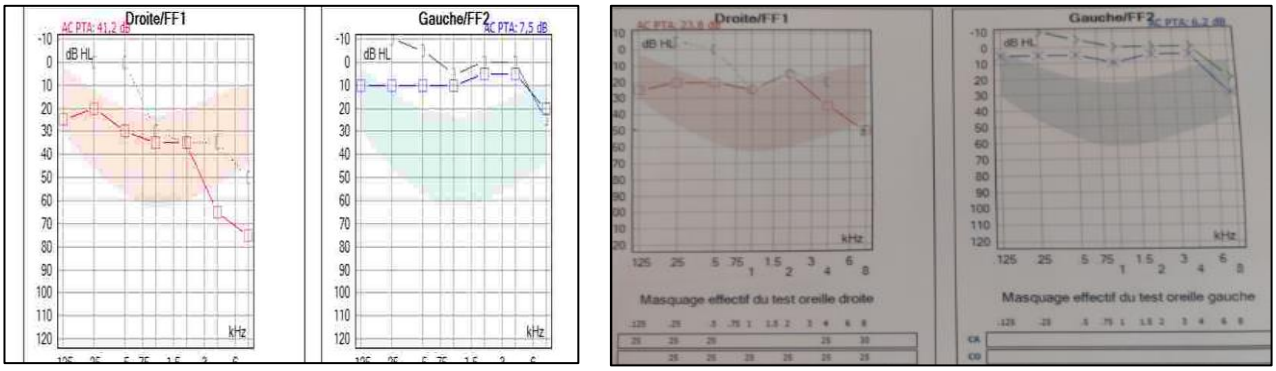
Symptomatic treatment for vertigo: The patient was treated with Tanganil (acetylleucine) to alleviate vertigo.

Outcome at 3 months: The patient demonstrated:

A Grade III Peripheral Facial Paralysis (PFP) according to the House-Brackmann scale, with:

Complete eye closure with effort, A positive Souques sign (indicating residual orbicularis oculi weakness).

Improvement in right-sided hearing loss, as confirmed by follow-up audiometry (Figure 3-B).



A **B**

Right **Left** **Right** **Left**

Figure 3: A- The tonal audiometry revealed right conductive hearing loss

B- outcome at 3 months Improvement in right-sided hearing loss, as confirmed by follow-up audiometry

DISCUSSION

It was James Ramsay Hunt in 1907[1] who studied the condition in detail. He was the first to associate the full range of clinical signs with involvement of the geniculate ganglion by the varicella-zoster virus (VZV).

It was not until the early 1950s [2] that Weller and Stoddart confirmed, through modern virological techniques, the connection between herpes zoster (shingles) and varicella (chickenpox), a link that had been suspected since the beginning of the century.

The global incidence is 5 per 100,000 inhabitants per year [7]. The varicella-zoster virus (VZV) has a strictly human reservoir.

Isolated shingles occurs in individuals without specific underlying conditions, and no definitive cause can usually be identified. However, certain contributing factors, such as cold exposure, trauma, or mild immunosuppressive treatments, may sometimes be noted.

On the other hand, symptomatic shingles often accompanies severe generalized conditions, such as malignant hematological disorders or AIDS [8].

Most autopsy studies after facial paralysis or on biopsy of the petrosal nerves or chorda tympani have found demyelination of the facial nerve in its intrapetrous course, mainly at the level of the labyrinthine portion and the meatal foramen, as well as an inflammatory infiltrate and vascular congestion. There would be a clear demarcation at the meatal foramen with nerve degeneration downstream while the nerve is normal upstream, which would indicate that the nerve is mainly injured at this level [9].

The complete auricular shingles, or SICARD syndrome, without being the most frequent, represents the most typical and comprehensive presentation of the disease. It affects all the elements of the acoustic-facial nerve bundle [8].

Clinical presentation of auricular shingles Classically, auricular shingles begins with deep, intense, paroxysmal otalgia radiating toward the auricle. The onset of pain generally precedes the eruption by a few hours or days (up to 5). This skin eruption is vesicular.

Measuring between 3 and 5 mm in diameter and located in the concha and external auditory canal (EAC).

An eruption may also appear in the oral cavity [10]. This eruption generally precedes facial paralysis by 24 to 48 hours. However, in some cases, the eruption occurs after the facial paralysis or may be absent, making the diagnosis of auricular shingles more difficult, known as "zoster sine herpete".

Facial paralysis reaches its peak severity by the 3rd day. Complete paralysis is twice as common as partial paralysis and occurs more frequently in individuals over the age of 50 [6]. It is associated with sensory disturbances in the Ramsay Hunt region, taste disorders, intolerance to loud sounds or hyperacusis, and dry eyes.

In approximately 25% of cases, patients have cochlear or vestibular involvement associated with facial paralysis (Sicard syndrome), related to the extension of inflammation of the geniculate ganglion to the inner ear affecting the spiral and vestibular ganglia [3-4]. According to a retrospective study of 325 patients.

Murukami *et al.,* showed subjective cochleovestibular involvement in 20% of cases, but objective in 48% [11]. Cochlear signs are tinnitus (24.7%) and generally mild to moderate sensorineural hearing loss (52.7%). Partial recovery is possible. Vestibular signs are acute imbalance, or even a true attack of rotatory vertigo, indicating vestibular neuritis (31.8%). They fade away in a few days [12].

Auricular zoster can be associated with meningitis, encephalitis or a more diffuse neuropathy that can affect the trigeminal nerves (trigeminofacial zoster) [13], the glossopharyngeal, vagus, spinal and hypoglossal nerves. Involvement of these mixed nerves is described as acute jugular foramen syndrome [14]. In these cases, analysis of the cerebrospinal fluid (CSF) can detect VZV by PCR. Magnetic resonance imaging (MRI) can also show inflammation of the jugular foramen. Very rare cases of hemiparesis and hemihypoesthesia have been reported in conjunction with Ramsay-Hunt syndrome. A case of contralateral cerebral infarction has also been described in association with Ramsay-Hunt syndrome.

Clinical diagnosis is often easy when faced with a classic clinical picture, sometimes difficult when faced with a polymorphic and dissociated presentation, virological and serological diagnosis takes on its full importance when faced with severe and atypical forms [15], virological and serological diagnosis generally highlights the virus or viral antigens by complement deviation techniques, immunofluorescence and ELISA [7].

Imaging is necessary in incomplete forms, if recovery of facial paralysis is incomplete at three months, if it is recurrent or if it is associated with damage to other cranial pairs. MRI with gadolinium injection is recommended. It must involve the brain, the base of the skull, the petrous bone and the parotid region.

Auditory Assessment Pure-tone audiometry rarely reveals conductive hearing loss; instead, sensorineural hearing loss, predominantly affecting high frequencies, is more common. Complete hearing loss (cophosis) is exceptional. Brainstem Evoked Potentials (BEP) often indicate that cochlear (endocochlear) hearing loss is more frequent compared to retrocochlear damage. Vestibular Assessment Vestibular testing, such as caloric and rotational tests, should only be performed under specific conditions: Spontaneous vestibular symptoms must have resolved.

The patient should no longer be sedated or receiving antivertiginous medication.

The external auditory canal must be normal.

Vestibular impairment is characterized by hypovalence (reduced response), hyporeflexia, or even

areflexia. These deficits may gradually compensate and regress over time [7].

Treatment Goals [16] The management of herpes zoster oticus aims to address the inflammatory syndrome, pain, facial paralysis, cochleovestibular disorders, and to prevent the spread of the infection and the development of long-term sequelae.Antiviral Therapy, All antivirals effective against the varicella-zoster virus (VZV) are nucleoside analogs [16]. These agents inhibit viral DNA polymerase, preventing viral replication. They are virostatic, acting only on viruses in the replicative phase. The three available drugs are: Oral acyclovir: 800 mg, Valacyclovir: a prodrug of acyclovir with similar efficacy, Famciclovir. In severe cases, IV acyclovir is recommended at a dose of 10 mg/kg in adults and 500 mg/m² in children every 8 hours for 7–10 days, followed by oral therapy for another 7 days.

The use of corticosteroids remains controversial [16]. Typically, a dose of 1 mg/kg/day is prescribed for a short course of 10 days. They provide potent anti-inflammatory effects and are generally well-tolerated. However, active viral disease may limit their use. Analgesics [16] Second-line (opioids) or third-line analgesics are often required for pain management. Postherpetic neuralgia may necessitate additional treatments, such as: Amitriptyline: up to 75 mg/day, Carbamazepine: 400–1,200 mg/day for hyperalgesic paroxysms. Local Care During the acute phase, maintaining proper hygiene of the eruptive zone through daily or twice-daily washing is essential [16]. Antibiotic-corticosteroid ear drops are recommended in cases of external otitis.

Corneal protection measures (occlusion, hydration, and monitoring) are crucial, particularly if corneal irritation occurs, warranting an ophthalmological evaluation [16]. Facial Paralysis Rehabilitation Early rehabilitation is critical and ideally should begin within 15 days of onset. The goal is to reduce hypertonia and prevent synkinesis [7].

Psychological Impact The psychological aspect of facial paralysis is significant, as it can be a source of considerable distress for patients. Emotional support and addressing the psychological burden are vital components of care.

Surgical Decompression of the Facial Nerve [7], There are differing views on surgical decompression of the facial nerve. It should be performed at the site of neural damage identified through imaging, in order to reduce the compression of oedematous neurons caused by viral damage. Neural damage becomes irreversible after approximately one month, and the surgery is more effective when performed before this timeframe. Early intervention is critical, and local treatment measures should always be included.

Recovery and Prognosis [7], Skin lesions typically heal within 15 days, but facial paralysis and sensory impairments may persist. Zoster-related facial paralysis tends to have a worse prognosis than facial paralysis from cold, often leaving lasting sequelae such as contractures, hemispasms, synkinesis, and crocodile tear syndrome.

Hearing Prognosis [7], Hearing loss usually has a good prognosis, with only 5% of cases resulting in permanent deafness. There is no direct correlation between the severity of facial paralysis and hearing loss.

Vestibular Impairment [7], The presence of vestibular involvement, along with other symptoms, is a poor prognostic factor for both facial paralysis and hearing loss.

The chances of recovery are better if treatment is initiated early, ideally within 72 hours of symptom onset, with up to 70% achieving full recovery. After 72 hours, only about 50% of patients experience recovery. Younger individuals generally show a more favorable outcome [7].

CONCLUSION

Herpes Zoster Oticus (Ramsay Hunt Syndrome) is an ear condition caused by the varicella-zoster virus. It occurs due to the reactivation of the latent virus within the geniculate ganglion, often triggered by a decline in cellular immunity. This condition accounts for about 4.5% of peripheral facial paralysis cases.

Diagnosis is primarily clinical. In its typical form, it presents as a vesicular skin rash in the Ramsay Hunt region, accompanied by peripheral facial paralysis, which constitutes Ramsay Hunt Syndrome.

Treatment combines corticosteroids and antiviral medications, along with early functional rehabilitation. In severe cases with poor prognostic factors, surgical decompression of the facial nerve may be considered. Without treatment, complete recovery occurs in only about 20% of cases.

Complications associated with facial paralysis primarily include synkinesis, which refers to abnormal, involuntary facial movements during voluntary facial activities.

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