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(CASE REPORT)

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Case report: Atypical Ramsey Hunt syndrome (zoster sine herpete): Two case reports

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Abstract

Introduction: Ramsay Hunt Syndrome is an uncommon disease caused by reactivation of latent varicella zoster virus infection in the geniculate ganglion. The classic triad consists ofotalgia, vesicles in the auditory canal and ipsilateral facial paralysis. Without treatment, full recovery of the facial paralysis occurs in 20% of cases; this is much improved if treatment is started within 72 hours. Zoster sine herpete is a subtype of RH syndrome and consists of facial paralysis without vesicular rash in the skin and mucosa.

Methodology: We present the clinical cases of two patients who presented to our clinic with peripheral facial paralysis but without auricular skin rash. Serological laboratory tests were positive for HZV. Both were placed on corticosteroid and antiviral therapy.

According to House Beckerman classification the grade of paralysis, improved for the first case from grade 4 to grade 1 and for the second case, from grade 5 to grade 4. The time of initiation of corticosteroid and antiviral therapy was different. In the first case, therapy was started immediately and in the second case after 5 days.

Conclusion: RHS is a rare syndrome that should not be underestimated. It is presented by a typical tirade: facial paralysis, auricular skin rash and otalgia. A special manifestation is the Zoster sine herpete form, which is diagnosed by serology and PCR for viral DNA. Immediate initiation of treatment (within 72h) is an important factor in prognosis.

Keywords: Ramsey Hunt Syndrome; Herpes Zoster Virus; Zoster Sine Herpete; Facial Paralysis; Serological Tests

1. Introduction

Ramsay Hunt syndrome, also known as herpes zoster oticus or geniculate ganglion herpes zoster, is a late complication of varicella-zoster virus (VZV) infection, resulting in inflammation of the geniculate ganglion of cranial nerve VII.[1] Early stages of VZV infection cause fever and diffuse vesicular rash, a condition that is commonly referred to as chickenpox. After the initial infection, the virus will often remain dormant in the body. Subsequent reactivation of the virus causes a "zoster" or "herpes zoster" phenomenon. This syndrome consists of pain and a vesicular rash along the involved nerve's distribution, typically corresponding to a single dermatome. The distribution and associated symptoms depend on the nerve involved. Less than 1% of zoster cases involve the facial nerve and result in Ramsay Hunt syndrome.[3]

Although the classic triad of Ramsay Hunt syndrome is ipsilateral facial paralysis, otalgia, and a vesicular rash, there is significant variability in clinical presentation, with some patients demonstrating facial paralysis before the rash or, sometimes, no rash at all. In the latter, the patient's chief complaints are severe ear pain and facial weakness; this variant is known as zoster sine herpete and can be very difficult to clinically distinguish from Bell's palsy. Zoster sine herpete has been reported to comprise up to 30% of Ramsay Hunt cases. If a rash is present, it may be frankly vesicular or

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maculopapular, and can involve the affected side of the face, scalp, palate, and tongue. Additional symptoms that may be reported include a change in taste sensation, dry eye, tearing, hyperacusis, nasal obstruction, and dysarthria. Hearing loss, tinnitus, and vertigo can be seen with involvement of the vestibulocochlear nerve, and hoarseness or aspiration may indicate involvement of the vagus nerve.

We present two clinical cases of two patients with zoster sine herpete, who followed the same treatment regimen but had different outcomes.

2. Clinical case 1

We present the case of a 15-year-old boy who comes to our clinic with paralysis of the left half of the face. In the anamnesis, he refers to a 1-week history of ear pain, accompanied by high temperature up to 39.5*C and ear discharge on the 3rd day of symptoms. He was diagnosed with acute otitis media and oral therapy with antibiotics was started. After 2 days of oral therapy, the condition did not improve, fever persisted and hospitalization and intravenous therapy with antibiotics were recommended. WBC 17.3 K/Ul, Neutrophil 83%, PCR 6.3%, fibrinogen 592. On the 7th day of therapy, the patient showed inability to move the muscles of the left side of the face. On neurological examination, the diagnosis of facial paralysis sinister House Brackmann grade 4 was made. No exacerbation of otitis media was observed in otoscopy, it was supplemented with a head CT with IVF which resulted in: Free tympanic cavity, bony chain with normal aspect, no image of lesions or secretions along the labyrinthine extension of the facial nerve. Mastoid cells with secretions inside, antrum with secretions inside.

In these conditions, Ramsey Hunt syndrome was suspected, although no vesicles were observed on otoscopy.

He was immediately placed on prednisolone therapy 25mgx2/day.

A serological test for VZV was performed, which resulted in: IgM 12.3 IU/ml (norm >11 IU/ml) and IgG 24.7 IU/ml (norm >11 Iu/ml).

Acyclovir 500mg x3 was also added to the therapy.

In audiometry (figure 1), a decrease in hearing in the left ear was noted, which improved in the following days (figure 2).

In the patient's follow-up during 3 weeks of treatment, the degree of paralysis improved from grade 4 to grade 1.

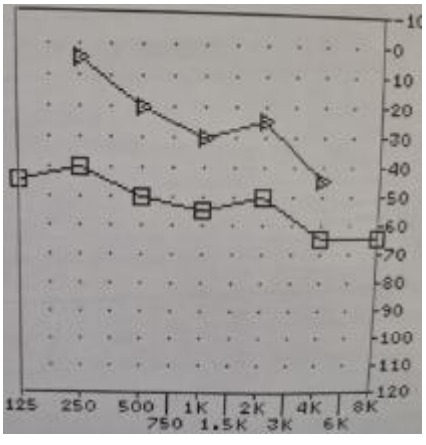


Figure 1 Audiology before treatment

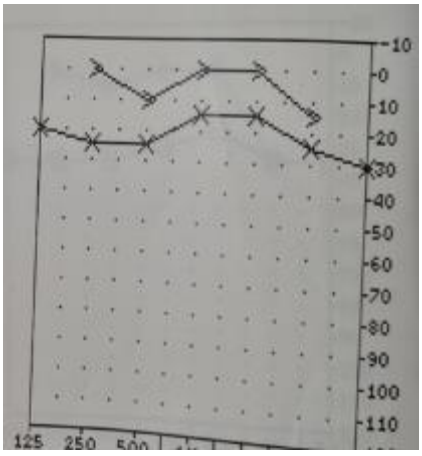


Figure 2 Audiology after treatment

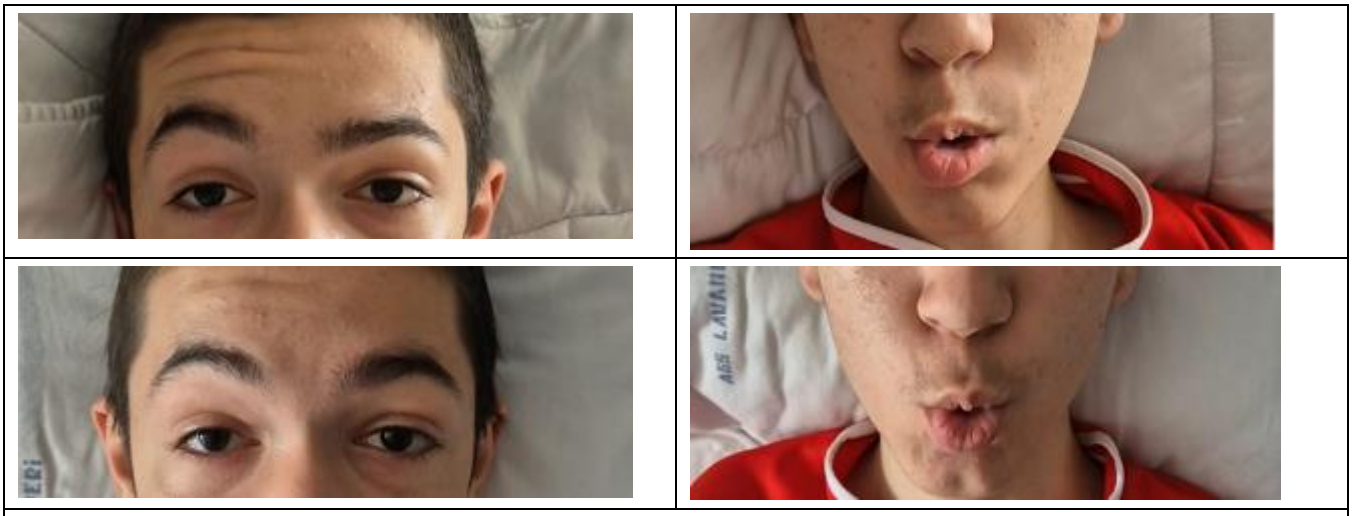


Figure 3 Progression of facial paralysis during 3 weeks of treatment**.**

3. Clinical Case 2

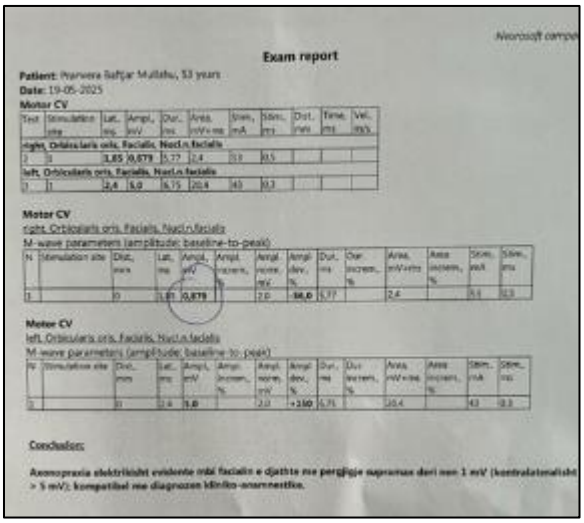
We present the case of a 53-year-old woman, without other comorbidities, who comes to the emergency department with severe headaches that begin at the level of the right MAE and spread to the half of the head on that side. The pain is in the form of crises, stabbing and burning that are not relieved by AIJS. An edema of the parotid gland is noted in the EO, normal otoscopy, neurological examination not significant. Outpatient therapy with AIJS is initiated. The patient returns after 5 days with paralysis of the right side of the face, which had started 5 days earlier, was assessed as House Brackman grade 5. Otoscopy: slightly hyperemic MAE dex, intact MT, normal audiometry and she was hospitalized.

A head CT with IVF was made, which results: no visible intracranial lesions, free right and left MAE, right laterocervical lymph nodes located from the mandibular angle and distally, slightly enlarged parotid gland.

It is placed on therapy with prednisolone 25mg X 3/day.

Serological testing for HZV was performed, which resulted in: IgM neg, IgG: 36.3 IU/mL (norm >11 IU/mL). The diagnosis of Zoster sine herpete was established, since IgG was about 4 times the norm [3]. Acyclovir 250mg X3 /day was added to the therapy. In the 2nd week of hospitalization, ENoG was performed, which resulted in: Axonopraxia in the n. facial dex in response below 1mV. (fig.4)

In the patient's follow-up during 3 weeks, the degree of paralysis improved from grade 5 to grade 4 HB.


Exam report

Patient: Hanvera Bajtar Mullahu, 53 years

Date: 19-05-2025

Motor CV

Test	Stimulation site	Lat. ms	Ampl. mV	Dur. ms	Area mV×ms	Stim. mA	Stim. ms	Dist. mm	Time ms	Vel. m/s
right, Orbicularis oris, Facialis, Nucl.n.facialis										
1	1	3,85	0,879	5,77	2,4	0,9	0,5			
left, Orbicularis oris, Facialis, Nucl.n.facialis										
1	1	2,4	5,0	6,75	20,4	40	0,3			

Motor CV

right, Orbicularis oris, Facialis, Nucl.n.facialis

M-wave parameters (amplitude: baseline-to-peak)

N	Stimulation site	Dist. mm	Lat. ms	Ampl. mV	Ampl. increm. %	Ampl. norm. mV	Ampl. dev. %	Dur. ms	Dur increm. %	Area mV×ms	Area increm. %	Stim. mA	Stim. ms
1		0	3,85	0,879		2,0	-56,0	5,77		2,4		0,9	0,5

Motor CV

left, Orbicularis oris, Facialis, Nucl.n.facialis

M-wave parameters (amplitude: baseline-to-peak)

N	Stimulation site	Dist. mm	Lat. ms	Ampl. mV	Ampl. increm. %	Ampl. norm. mV	Ampl. dev. %	Dur. ms	Dur increm. %	Area mV×ms	Area increm. %	Stim. mA	Stim. ms
1		0	2,4	5,0		2,0	+150	6,75		20,4		40	0,3

Conclusion:

Axonopraxia elektrikisht evidente mbi facialin e djathte me perfijje supranue deri ne 1 mV (kontralateralisht > 5 mV); kompatibel me diagnozen kliniko-anamnestike.

Figure 4 ENoG of the right and left orbicularis oris, facialis and Nucl.n.facialis

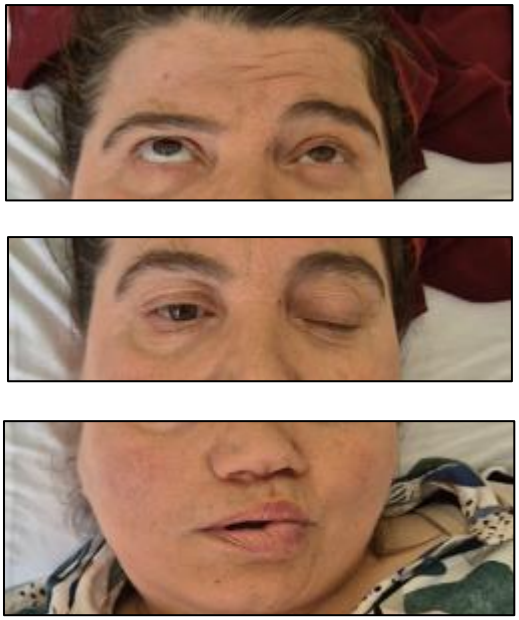


Figure 5 Grade of facial paralysis

4. Discussion

Ramsay Hunt syndrome (RHS) represents the second most frequent cause of peripheral facial palsy, accounting for approximately 12% of cases, and is associated with a significantly poorer prognosis compared to Bell's palsy[5]. The condition is classically defined by a triad comprising otalgia with vestibulocochlear involvement, ipsilateral peripheral facial paralysis, and a vesicular rash affecting the auricle.

According to Coulson et al., ipsilateral otalgia is the most common initial manifestation, reported in 55% of patients, with facial paralysis and vesicular eruptions typically developing within 2–3 days. Facial paralysis was identified as the presenting symptom in 23% of cases, whereas vesicles appeared first in only 2%. The prognosis of facial nerve paralysis in RHS is notably worse than that observed in Bell's palsy. Approximately 70% of patients with RHS achieve normal or near-normal recovery of facial function, compared to recovery rates exceeding 90% in Bell's palsy. Furthermore, the incidence of synkinesis following RHS is substantially higher, approaching 40%, whereas rates of approximately 16% have been reported following Bell's palsy[12].

Ramsay Hunt syndrome is primarily a clinical diagnosis, as laboratory confirmation of varicella-zoster virus (VZV) infection is often limited by practicality and sensitivity. Although VZV can be isolated from vesicular fluid, blood, saliva, or tears, polymerase chain reaction (PCR) testing demonstrates a sensitivity of approximately 58%. Enzyme-linked immunosorbent assay (ELISA) techniques may reach sensitivities of 82–99%, but their diagnostic utility in the acute setting is limited. In certain patients, VZV reactivation presents with facial paralysis and severe otalgia in the absence of vesicular eruption. In these cases, diagnostic confirmation relies on PCR detection of viral DNA or serological evaluation of VZV-specific IgM and IgG antibodies, with antibody titers exceeding four times the normal reference range considered confirmatory[2]. Reactivation without cutaneous manifestations, termed *zoster sine herpete* (ZSH), may present with diverse neurological manifestations, including vasculopathy, radiculopathy, ophthalmic neuralgia, vagus nerve paralysis, and facial nerve palsy.

In patients with ZSH, rising anti-VZV antibody titers serve as indicators of active viral infection. Besides VZV DNA detection, the presence of anti-VZV IgG antibody in the CSF [16] or serum can be used to diagnose ZSH. Anti-VZV IgG in the CSF, and the reduced serum/CSF ratios of VZV IgG compared with the normal level of serum/CSF ratios for albumin and total IgG, reflect the intrathecal synthesis of anti-VZV IgG [16].

When available, electrodiagnostic testing such as electroneuronography (ENoG) and electromyography (EMG) may provide useful prognostic information by quantifying the extent of nerve damage more precisely than is possible with a physical examination alone, predominantly in the case of House-Brackmann grade VI paralysis.[14]

The importance of diagnosing the Zoster virus in patients with facial paralysis helps us in prognosis, as in cases positive for the zoster virus, recovery from facial paralysis is more difficult than in those infected with HSV-1.[7]

Overall, the most consistent prognostic indicator in Ramsay Hunt syndrome is the presenting severity of facial paralysis. Patients who present with House-Brackmann grade III paralysis tend to recover to normal function; patients with House-Brackmann grade IV or V paralysis are more likely to recover to grade II function, and patients with House-Brackmann grade VI function at presentation are more likely to recover to grade III function.[11] Patients who do not recover their premorbid function will almost certainly develop some degree of synkinesis. Clinically significant flaccid paralysis is extremely rare in the long term. Most patients complete their recovery within one year. Relatively young, healthy patients with incomplete paralysis will often recover to full or near-full function within several weeks to a few months. Overall, roughly 70% of Ramsay Hunt patients will recover to House-Brackmann grade I or II function.[11] Factors that have been associated with non-recovery include age over 50 years old, a greater degree of axonal damage on electrodiagnostic testing, involvement of multiple cranial neuropathies, the presence of oropharyngeal lesions, and diabetes.[11,15]

Herpes zoster is generally self-limiting in nature. Therefore, the main goals of treatment are to decrease the incidence of late complications, including spastic facial paralysis and postherpetic neuralgia. Multiple studies have shown a significant decrease in long-term complications with the use of oral antivirals and steroids.[11,12] It is unclear, however, whether these medications decrease the length or severity of acute symptoms. Acyclovir, valacyclovir, and famciclovir have all been studied and found effective. Antiviral treatment is usually administered for 7 to 10 days; however, some studies have reported prolonged or delayed degeneration of facial nerve axons up to 21 days after paralysis onset, and therefore recommend continuing antiviral therapy for 21 days. High-dose corticosteroids, either oral or intravenous, should be co-administered with antiviral treatment. There is no overall consensus regarding the total duration of steroid treatment, which can vary from 4 to 37 days, but steroids should be prescribed at a high dose[12]. This is typically prednisone 1 mg/kg/day up to a maximum dose of 60 mg, or the equivalent of such, followed by a taper to prevent acute adrenal insufficiency.

Symptomatic management is also critical, particularly for two aspects of Ramsay Hunt syndrome: pain and corneal exposure. Analgesia is often needed with zoster; a multimodal approach with acetaminophen, non-steroidal anti-inflammatory drugs (NSAIDs), and long-acting opioids can all be used. Artificial tears throughout the day and ocular lubricant ointment at night are helpful for the prevention of exposure keratopathy.

According to Murakami S [4] in patients who started therapy within the 3rd day of symptoms, recovery was complete in 75% of cases, in patients who started therapy between days 4-7 of symptoms, recovery was complete in 48% of cases and in patients who underwent therapy after day 7, recovery was complete in 30% of cases.

In the two patients above, the first case underwent therapy immediately and the degree of recovery was from HB 4 to HB1, while the second started therapy with corticosteroids and antivirals after 5 days, with a degree of recovery from HB5 to HB4.

5. Conclusions

Ramsey Hunt syndrome is a rare syndrome that should not be underestimated, which is presented by a typical tirade: facial paralysis, auricular skin rash and otalgia. A special manifestation is the Zoster sine herpete form, which should be suspected in those cases when severe ear pain and facial paralysis persist without other apparent causes. The diagnosis is established by serology or DNA for the HZV virus. Immediate initiation of treatment (within 72h) is an important factor in prognosis. Therapy consists of a combination of corticosteroids and antivirals according to kg/weight.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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