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Ramsay Hunt Syndrome in an Immunocompetent 17-Years-Old Boy: A Case Report

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ABSTRACT

Background: Ramsay-Hunt syndrome (RHS) is an uncommon complication of herpes zoster caused by the reactivation of the latent varicella-zoster virus (VZV) in the geniculate ganglion. It typically presents as a triad of ipsilateral facial paralysis, ear pain, and vesicular eruptions in the auditory canal or on the auricle. RHS can affect both immunocompetent and immunocompromised individuals, with an incidence of approximately 5 cases per 100,000 people annually. The incidence increases with age and is rarely observed in children. It is frequently reported in immunocompromised patients or individuals aged 50 years and older. Moreover, RHS is believed to account for up to 20% of clinically diagnosed Bell's palsy cases. Major risk factors for VZV reactivation include immunosuppression, advanced age, pregnancy, lack of varicella vaccination, and physical or psychological stress, which may lead to central nervous system involvement.

Case presentation: A 17-year-old immunocompetent boy presented with multiple erythematous vesicles on the right side of his chin, lower lip, cheek, and slightly on the auricle, which appeared one day prior to examination. The lesions began with pain, burning sensations, and mild fever. Initially, the patient was diagnosed with herpes zoster involving the mandibular division (V3) of the trigeminal nerve. However, nine days after symptom onset, he developed right peripheral facial palsy, leading to a diagnosis of Ramsay Hunt syndrome with House. Treatment included a combination of antiviral medication, corticosteroids, and supportive care. The patient showed significant recovery within six weeks.

Conclusion: Ramsay-Hunt syndrome is a rare complication of herpes zoster, commonly occurring in elderly and immunocompromised patients. Nonetheless, it can also manifest in younger individuals. Therefore, RHS should be also considered in the differential diagnosis when typical symptoms are present in young age population. Early diagnosis and initiation of treatment within 72 hours of symptom onset using antivirals and corticosteroids are essential for achieving optimal outcomes and minimizing long-term complications.

Keywords: Ramsay-Hunt syndrome, Varicella zoster virus, young age, facial paralysis

INTRODUCTION

Ramsay-Hunt syndrome (RHS) is a rare condition resulting from the reactivation of latent varicella zoster virus (VZV) in the geniculate ganglion.[1] Varicella zoster virus, a member of the human herpes virus family, is a double-stranded DNA virus, also known as human alphaherpesvirus 3 (HHV-3).[2] Primary VZV infection causes chickenpox, characterized by fever and

widespread vesicular rash. After this initial infection, the virus remains dormant in cranial and dorsal root ganglia, but may reactive later in life, leading to herpes zoster, commonly known as "shingles". A decline in VZV-specific cell mediated immunity can facilitate viral reactivation.[3] This syndrome consists of pain and a vesicular rash along the involved nerves distribution, typically corresponding to a single dermatome. RHS occurs in less than 1% of herpes zoster cases, involving the facial nerve (CN VII).[4]

Ramsay Hunt syndrome affects both immunocompetent and immunocompromised individuals, with an incidence of approximately 5 cases per 100,000 people annually. The incidence increases with age and is uncommon in children, thought it can account for 16% of unilateral facial palsy cases in children and 18% in adults. It is also implicated in up to 20% of cases clinically diagnosed as Bell's Palsy.[5] Risk factors for reactivation include immunosuppression, advanced age, pregnancy, lack of varicella vaccination, and physical or psychological stress, all of which may lead to viral invasion of the central nervous system.[2]

While RHS mainly affects the facial nerve, other cranial nerves may also be involved due to complex anastomoses between cranial nerve such as the facial, trigeminal, glossopharyngeal, vagal, and cervical nerves. This can lead to a variety of symptoms including ear pain, increased sensitivity to sound (hyperacusis), taste disturbances, reduced tear secretion, hoarseness, weakness of mastication muscles, asymmetric elevation of the soft palate, facial numbness, tongue deviation, difficulty swallowing, ocular deviations, and pupil dilation, listed in decreasing order of frequency.[6]

Clinically, Ramsay Hunt syndrome presents with a triad of ipsilateral facial paralysis, ear pain, and vesicles eruptions in the auditory canal or on the auricle.[7] The timing of vesicle appearance relative to facial palsy varies: vesicles may appear before or simultaneously with paralysis; after paralysis onset (usually preceded by pain); or not at all in a variant called zoster sine herpete, which can mimic Bell's palsy and complicate diagnosis.[8] Severity of facial palsy is often assessed using several grading systems, with House-Brackmann being the most widely used. However, no universally accepted standard grading system exists.[7]

The diagnosis of RHS is primarily based on clinical presentation, but in uncertain cases, laboratory tests such as polymerase chain reaction (PCR) testing, direct fluorescent antibody (DFA) testing, and viral culture PCR can confirm the diagnosis using cerebrospinal fluid, blood, and other non-cutaneous specimens.[7] Brain MRI is mandatory for the exclusion of tumors or demyelinated lesions or when there is a suspicion of infective brain tissue complications of the VZV.[1]

Treatment goals of RHS include immediate pain relief, prevention of complications such as keratopathy from incomplete eyelid closure, and reduction of late sequelae like spastic facial paralysis and post-herpetic neuralgia.[9] Early antiviral therapy with agents such as acyclovir, valacyclovir, or famciclovir within 72 hours of lesion onset is advised, along with corticosteroids to reduce inflammation and edema. Combination therapy of antivirals and steroids improves recovery rates compared to steroids alone.[6] Surgical intervention is generally not beneficial. Vaccination against varicella and shingles is effective in preventing VZV reactivation and RHS.[2]

In general, the prognosis for RHS is worse than Bell's palsy. Spontaneous remission in RHS is rare, only 20% patients recover completely without treatment. The prognosis varies based on the severity of facial paralysis, age, extent of nerve damage, presence of other cranial neuropathies, and comorbidities like diabetes. Post-herpetic neuralgia is a common complication, especially in patients over 50, characterized by persistent pain beyond three months after rash onset. In

general, the prognosis for RHS is worse than Bell's palsy.[2]

CASE REPORT

A 17-years-old boy came to dermato-venerology policlinic at Wangaya Regional Hospital on October 5th 2024, presenting with multiple vesicular eruptions on his right chin, lower lip, cheek, and slightly on the auricle. It started with the appearance of one red spot on the chin 1 day before the examination, which then increased in number and spread to the ear after several hours, but was restricted only to the right side. The symptom was started by pain, burning sensation and a slight fever. The Patient did not complain of any visual or hearing problems, dizziness, nausea or facial weakness. Other complaints were denied. The patient had chickenpox when he was 3 years old and had received full chickenpox vaccinations. He is a high school student and member of the student council. In recent weeks, the patient reported having taken care of many school activities requiring him to stay up late about 4 hours of sleep only.

On physical examination, the patient was fully conscious, with a height of 170 cm, weight of 115 kg, BMI 39.8 kg/m^2 (obese), and normal vital signs. Dermatologically, on the right cheek, chin, lower lip, and crura of antihelix, there were clustered vesicles on a erythematous and edematous skin base, exhibiting herpetiform configuration with unilateral distribution, corresponding to the mandibular branch of the trigeminal nerve (V3) dermatome area (figure 1). A diagnosis of herpes zoster involving the mandibular division trigeminal nerve was made. Antiviral therapy with acyclovir 800 mg five times a day for 10 days and paracetamol 500 mg 3 times a day were started. Patient was referred to the ENT department to assess ear and the eighth cranial nerve involvement. The examination showed no abnormalities aside from lesions on right antihelix. Gentamycin 0.1% ointment 3 times a day was prescribed for the lesions that had ruptured.

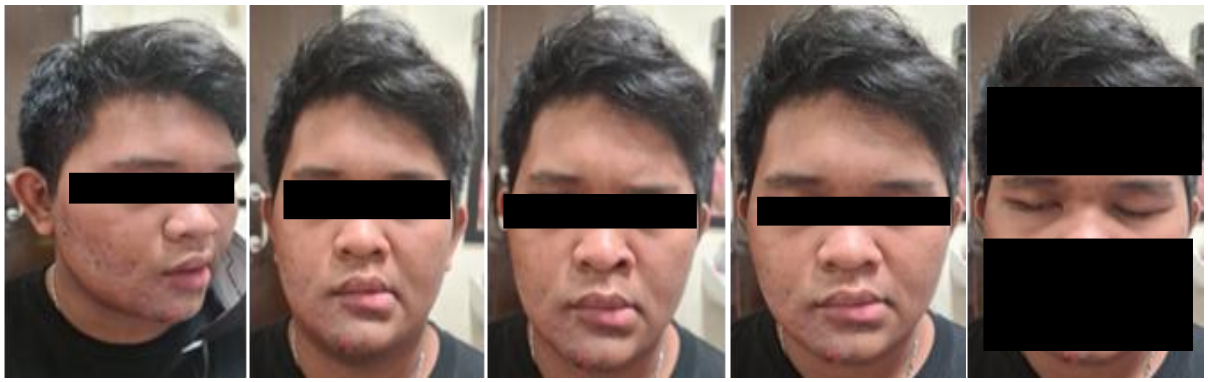
However, on October 15th 2024, the patient developed facial asymmetry. He noticed it since 2 days ago (9 days from first symptoms appeared), but only sought care then because symptoms worsened, including right eye was watering and ear pain increased. The skin lesions had mostly dried except on the chin. No new lesions were observed (figure 2). Vital signs were still within normal limits. Thus, diagnosis of Ramsay Hunt syndrome is considered. Patient was referred to the neurology department. Neurological examination revealed face asymmetry at rest; no forehead creases on the right side when frowning, drooping of the right lip corner when smiling, and incomplete closure of the right eye (lagophthalmos OD), which indicating peripheral facial nerve palsy. No other neurological deficit was found.

The neurologist diagnosed infra nuclear facial nerve paresis (CN VII) due to RHS with a House Brackmann Grade IV. Systemic steroid, methylprednisolone tablet 4 mg was prescribed three times a day for 5 days. Other medications included omeprazole 20 mg once daily, mecobalamin 500 mg twice daily as neuroprotective agent, and lubricant eye drops six times a day for the right eye. Acyclovir therapy was continued until completion. The Patient was also referred to the medical rehabilitation department and scheduled for physiotherapy three times a week for 6 weeks.

At follow-up, after 5 days of methylprednisolone and 3 times physiotherapy sessions in 1 week, the skin lesions had completely dried, pain was reduced, the right eye was no longer sore or watery, as it could close completely, and forehead crease appeared more symmetrical. The right lip corner was still drooped at rest and when smiling. Physiotherapy was continued until 6 weeks. Mecobalamin was continued and neurology follow-up was scheduled monthly. On the further follow-up, patient showed a good recovery. After 6 weeks, patient's facial paralysis became House Brackmann Grade I which showed a full recovery. Skin lesion and pain were

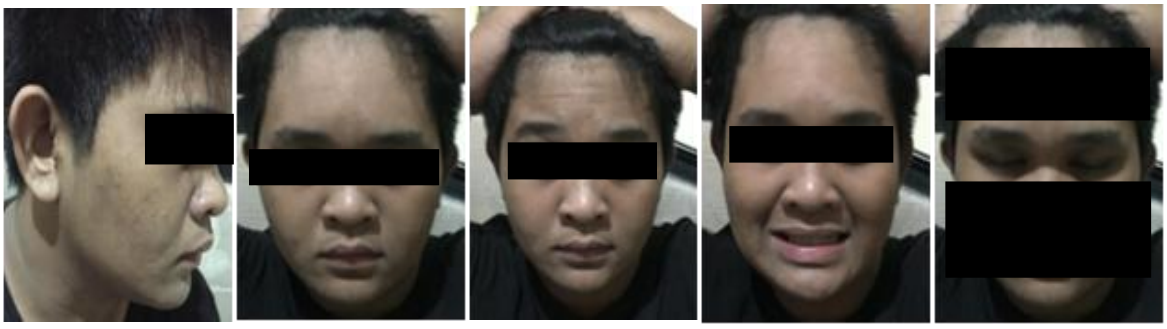
completely healed, face looked symmetrical, forehead crease were evenly distributed on both sides, lips were symmetrical at rest and smiling (figure 3). No new complaint was noted. Physiotherapy was stopped, while mecobalamin was continued for another 1 month.

Figure 1. Clinical picture of first time patient was admitted. Clustered vesicles on an erythematous and edematous skin base with herpetiform configuration and unilateral distribution were found on the right cheek, chin, lower lip, and crura of antihelix, corresponding to the mandibular branch of the trigeminal nerve (CN V3) dermatome.



A B C D E

Figure 2. 2nd visit (10 days after 1st visit). Skin lesions had mostly dried except on the chin without new lesions observed (A). Neurological findings: asymmetry face at rest (B), no forehead creases on the right side when frowning (C), dropped right lip corner when smiling (D), and incomplete right eye closure (lagophthalmos OD), consistent with House Brackmann grade IV.



A B C D E

Figure 3. 6 weeks after 2nd visit. Skin lesions had completely dried (A). Neurological findings: symmetrical face at rest (B), visible creases on both side of forehead (C), symmetrical smile (D), and complete right eye closure, consistent with House Brackmann grade I.

DISCUSSION

Ramsay Hunt syndrome, also known as herpes zoster oticus or geniculate ganglion herpes zoster, is a rare complication of varicella zoster virus (VZV) reactivation in the geniculate ganglion. Ramsay Hunt

syndrome has an incidence of approximately 5 per 100,000 people annually. Cases have been reported in patients ranging from 3 months to 82 years old, although individuals in their 7th and 8th decades are most susceptible. The incidence increases with age and is rare in children. Immunocompromised conditions, advanced age, pregnancy, lack of varicella vaccination, and physical or psychological stress are major factors predisposing to viral reactivation, which may invade the central nervous system.[9]

Meanwhile RHS is known for a disease in elderly and immunocompromised patient, we reported a case that occurred in a 17-year-old male patient without other immunosuppressive conditions. He had a history of chickenpox at age 3 and had received the recommended vaccine. However, during anamnesis, the patient admitted he had been busy at school and often stayed up late (about 4 hours of sleep) during the past weeks. The history of varicella suggests reactivation of the virus. Physical and psychological stress may have triggered VZV reactivation in this patient.

Diagnosis is generally based on anamnesis, physical examination, and characteristic symptoms and signs of the syndrome. RHS typically presents with a triad of ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal or auricle. However, diagnosis can be difficult with polymorphic and dissociated presentations. The latter can be challenging to distinguish clinically from Bell's palsy. Therefore, supporting examinations are valuable in doubtful cases.[5,8]

Our patient presented with multiple reddish vesicles accompanied by pain and burning sensations since one day before examination, preceded by prodromal symptoms. The lesions were distributed unilaterally on the right chin, lower lip, cheek, and auricle, following the dermatome of the mandibular branch of the trigeminal nerve (V3). Thus, the diagnosis was initially lead to herpes zoster. However, 10 days after the first visit (8 days after blister onset), the patient showed several symptoms that indicate peripheral facial nerve palsy. A diagnosis of Ramsay Hunt syndrome was confirmed, with a House-Brackmann grade IV. The clinical presentation was sufficient for diagnosis, so further supportive tests were not performed.

The main goals of herpes zoster therapy are to provide immediate relief of acute pain and symptoms, prevent complications of keratopathy, reduce late complications like spastic facial paralysis and post-herpetic neuralgia.[9] Treatment with antivirals and corticosteroids is the current mainstay.[10] Acyclovir, valacyclovir, and famciclovir are effective antiviral options. Acyclovir 500 mg five times daily is the most affordable; valacyclovir 1000 mg three times daily is easier to take and may be more effective; famciclovir 500 mg three times daily is another effective option. Treatment usually lasts 7 to 10 days, but some recommend 21 days due to delayed nerve degeneration. Parenteral acyclovir 15mg/kg/day also may be given but has no significant difference with oral regimen.[8]

High-dose corticosteroids, oral or intravenous, should accompany antiviral treatment. Corticosteroids can accelerate the healing by reducing edema and decompression of neurogenic structures that are present in the facial nerve canal. Treatment duration varies from 4 to 37 days, usually prednisone 1 mg/kg/day (max 60 mg) with tapering to prevent adrenal insufficiency.[9] A review of 13 studies on RHS prognosis showed recovery rates varied by steroid type but were not statistically significant ($p > 0.05$).[12]

Combination therapy with steroids and acyclovir increases the recovery rate of RHS compared to steroids alone. A study has compared that statistically significant difference in recovery rate is observed when patients are treated with both steroids and acyclovir (90%) compared to steroids alone (64%).[6] Our patient was treated <72 hours from onset with acyclovir 800 mg five times daily for 10 days according to drug availability and followed with

methylprednisolone 4 mg three times daily for 5 days after RHS diagnosis.

Other medications included paracetamol 500 mg three times daily for pain, gentamycin 0.1% ointment for excoriated lessions, omeprazole 20 mg once daily for gastrointestinal protection, mecobalamin 500 mg twice daily as a neuroprotective agent, and lubricant eye drops six times daily for the right eye to prevent keratopathy. Physiotherapy three times weekly for six weeks was also given. Motor physiotherapy aimed to address motor sequelae, which tend to be more severe than in Bell's palsy.[8]

Prompt diagnosis and therapy ideally within 72 hours of symptom onset are crucial for optimal outcomes and reduce long-term complication. Poorer prognosis is associated with age over 50, metabolic disease, immunocompromise condition, and multiple cranial nerve involvement.[10] Our patient regained full recovery (facial paralysis became House Brackmann Grade I) within six weeks, likely due to early treatment, young age, absence of comorbidities, and no multiple cranial nerve involvement.

CONCLUSION

Ramsay hunt syndrome is a disease that generally known to occur in old age and with immunocompromised conditions. However, some cases are also reported in younger age population, including this patient which belongs to immunocompetent 17-years-old boy. Diagnosis is made by clinically based on history taking and physical examination. Supporting examination modalities are rarely needed, thus early diagnosis can be expected. Early diagnosis and initiation of treatment within 72 hours of symptom onset using antivirals and corticosteroids are essential for achieving optimal outcomes and minimizing long-term complications.

Declaration by Authors

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