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CASE REPORT

Ramsay Hunt Syndrome in a Pediatric Patient with Atypical Presentation: Case Report

Síndrome de Ramsay Hunt en paciente pediátrico con presentación atípica: Reporte de Caso

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ABSTRACT

The case of a preschooler with Ramsay Hunt Syndrome (RHS) is reported, an infrequent pediatric neurological condition caused by the reactivation of the varicella-zoster virus, whose atypical manifestation represented a diagnostic challenge. The patient, a 3-year and 4-month-old male, presented with vesicular lesions on the forehead and periorbital region, without the classic auricular rash, which led to a delayed diagnosis. Once RHS with bacterial superinfection was confirmed, acyclovir, prednisone, and antibiotics were administered, achieving a favorable evolution. The atypical presentation highlights the diagnostic challenges in children and the risk of delayed management. Therefore, it is crucial to maintain a high index of suspicion for RHS in pediatrics, even with unusual presentations, to ensure early treatment and prevent severe long-term complications.

Keywords: Ramsay Hunt Syndrome; Pediatrics; Facial Palsy; Varicella-Zoster Virus; RHS; PFP; Ramsay-Hunt.

RESUMEN

Se reporta el caso de un preescolar con Síndrome de Ramsay Hunt (SRH), una infrecuente condición neurológica pediátrica causada por la reactivación del virus varicela-zóster, cuya manifestación atípica representó un desafío diagnóstico. El paciente, un masculino de 3 años y 4 meses, presentó lesiones vesiculares en la frente y la región periorbitaria, sin la clásica erupción auricular, lo que condujo a un diagnóstico tardío. Una vez confirmado el SRH con sobreinfección bacteriana, se administró aciclovir, prednisona y antibióticos, logrando una evolución favorable. La presentación atípica resalta los desafíos diagnósticos en niños y el riesgo de un manejo tardío. Por ello, es crucial mantener un alto índice de sospecha de SRH en pediatría, incluso con cuadros inusuales, para asegurar un tratamiento temprano y prevenir complicaciones severas a largo plazo

Palabras clave: Síndrome de Ramsay Hunt; Pediatría; Parálisis Facial; Virus Varicela-Zoster; SRH; PFP; Ramsay-Hunt.

INTRODUCTION

Ramsay Hunt syndrome (RHS), also known as otic herpes zoster, is a rare neurological condition caused by the reactivation of the varicella-zoster virus (VZV) in the geniculate ganglion of the facial nerve.[1,2,3] It manifests clinically with the classic triad of ipsilateral peripheral facial paralysis (PFP), ear pain (otalgia), and vesicular rash in the auricle or external auditory canal.[4,5] After the primary infection, the virus (VZV) remains dormant for life in the ganglia of the spinal and cranial nerves. Following a stressful event or condition (such as chemotherapy, immunosuppression, malnutrition, infections, or non-immunization with the varicella vaccine), it can reactivate and migrate along the sensory branches of the facial nerve around the external auditory meatus, initiating virus replication and developing a rash with typical clinical features. However, the clinical presentation of HRS can be variable; vesicles can also appear in the mouth, tongue, cheek, or scalp due to the anastomosis between the cranial and cervical nerves.[4,5] Although HRS is a known cause of acute FOP, accounting for 7 % of cases, it is rare in the pediatric population,[5,6] accounting for 10 % of FOP cases in children under 6 years of age and 16,7 % of FOP cases in children overall.[3,5] The reported incidence in the pediatric population is at least 2,7 per 100 000. Studies have shown that the incidence of SRH is significantly higher in children aged 6 years (24,3 %) compared to those under 6 years (10,5 %).[4,7]

The diagnosis of SRH is clinical, based on medical history and physical examination findings.[2,8,9] However, atypical presentations, rarity, and the patient's age, in this case report, pediatric age, can lead to misdiagnosis or delayed diagnosis.[3] The condition is self-limiting. The importance of adequate and early treatment with antiviral agents and corticosteroids is crucial for improving prognosis and reducing the incidence of complications, such as irreversible corneal damage caused by lagophthalmos, postherpetic neuralgia, and facial scarring.[1,2]

The case report describes a case of HRS in a preschool patient, highlighting an atypical clinical presentation with severe periorbital involvement and complications that arose due to a delayed initial diagnosis.

CLINICAL CASE

A 3-year-old male patient was admitted to the emergency department, referred from a primary care center, with a 7-day history characterized by headache, vesicles in the frontal region, erythematous lesions on the face, and fever. The patient's history revealed a chronic condition that had been developing for approximately 90 days, which began with an ulcerative lesion on the scalp accompanied by alopecia. Subsequently, confluent bullous-vesicular lesions appeared in the periorbital region of the left eye, as well as eyelid ptosis and unilateral facial edema, leading to the application of ampicillin ointment as initial treatment. During this period, the patient was misdiagnosed with measles at a pharmacy and received an intramuscular antibiotic.

Upon admission, the patient was in a fair to poor general condition, irritable, tachycardic (heart rate 145 bpm), and tachypneic (respiratory rate 41 bpm), with a temperature of 37,1°C and an oxygen saturation of 96 %. The skin and mucous membranes were moist and pale. On the head, there was an ulcerative lesion on the scalp measuring approximately 5x7 cm with alopecia. On the left side of the face and the periorbital region of the left eye, the lesions were confluent blistering-vesicular, with halos of necrotic furfurious scaling and adherent clots, giving the wound a shiny and moist appearance. Eyelid ptosis and unilateral facial edema were confirmed. The left eye had a superficial ulcerative plaque. The external auditory canals were patent, with no mention of vesicular eruption in the auricle (figure 1).

Figure 1. Wound observed on the left side of the face, periorbital region of the left eye, confluent bullous-vesicular lesions, with halos of necrotic furfurious desquamation and adherent clots

The rest of the physical examination, including the respiratory, cardiovascular, abdominal, and neurological systems, revealed no other significant abnormalities.

Laboratory tests on admission revealed leukocytosis with 22,000 white blood cells/mm^3, comprising 81 % segmented and 19 % lymphocytes; a hemoglobin (Hb) level of 9,6 g/dL and a hematocrit (Hct) of 29 %. C-reactive protein (CRP) was positive. Tests for HIV, syphilis, and Chagas disease were negative. A follow-up examination after treatment showed a decrease in leukocytes to 6,010/mm^3, with 43 % segmented and 57 % lymphocytes, and a hematocrit of 30,3 %. CRP remained elevated (+++). A general urine test showed a density of 1015, protein (+), no nitrites or erythrocytes, and 2-4 leukocytes per field with few bacteria.

Following a teleconsultation with the Infectious Diseases Department, the patient was diagnosed with Ramsay Hunt syndrome secondary to herpes zoster, with a bacterial coinfection in the integumentary tissue. Therefore, the patient was initially treated with intravenous (IV) cefotaxime, IV clindamycin, oral (PO) acyclovir, cetirizine every 12 hours, and PO prednisone, along with wound care using a cream containing clindamycin, gentamicin, and metronidazole. A teleconsultation with Infectious Diseases confirmed the diagnosis and adjusted the treatment, prescribing oral acyclovir at 10 mg/kg/dose for 10 days, oral prednisone at 1 mg/kg/day for 5 days, intravenous cloxacillin and clindamycin, and mupirocin cream to be applied three times a day. Subsequently, clindamycin IV was started, and cloxacillin was discontinued. The patient's progress was favorable, with the lesions progressing to a crusting phase. Although he had pruritus that caused excoriations, more pronounced on the scalp, the patient showed good oral tolerance to the medication (figure 2 y 3).

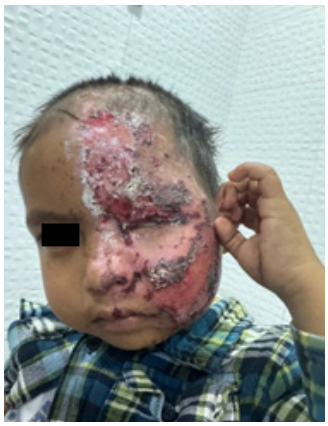

Figure 2. Lesions in the left hemiface region in the crusting phase

Figure 3. Lesions in the left hemiface region in the crusting phase. Favorable progress

DISCUSSION

HSR is caused by the reactivation of the varicella-zoster virus (VZV), which remains latent in the sensory ganglia after primary chickenpox infection.[1,2] Although this pathology can affect people of any age, data on gender incidence are inconsistent; some reports indicate an equal distribution between men and women, while others suggest a slight predilection for females. Therefore, it is considered rare in childhood, especially in young children, such as the one in this case (3 years and 4 months).[3] The incidence of HRS is generally higher in children over 6 years of age (24,3 %) compared to those under 6 years of age (10,5 %).[4,7] Certain factors, such as stress, chemotherapy, immune compromise, malnutrition, and even other infections, can increase susceptibility and severity of the disease.[3,4]

The clinical presentation of this case was notably atypical, as the classic triad of HRS includes PFP, otalgia, and a vesicular rash on the auricle.[1,2] Considered the second most common cause of non-traumatic facial

paralysis with lower motor neuron characteristics, it accounts for approximately 10 %–12 % of acute facial paralysis cases. In this patient, the eruptions were located on the forehead, left hemiface, and left periorbital region. The vesicular rash may appear in other areas innervated by the facial nerve, including the face, scalp, palate, and tongue, due to anastomoses between the cranial and cervical nerves.[3,5] The absence of the auricular rash, a key feature of this pathology, together with severe periorbital involvement, highlights the variability of the clinical picture and the inherent risk of corneal damage due to lagophthalmos.[3,4]

The diagnosis of HRS is primarily based on clinical findings.[1,5] However, initial misdiagnoses of measles and suspicion of epidermal necrosis or Stevens-Johnson syndrome underscore the diagnostic challenges in atypical presentations, especially in children.[3] Although serological tests (VZV IgG/IgM ELISA) or PCR to detect viral DNA in exudates or saliva may be useful, they lack sensitivity, and even the waiting time for results can hinder decision-making in acute cases.[8] Magnetic resonance imaging (MRI) may show inflammation of the geniculate ganglion, but it is not strictly necessary for diagnosis.[3,5,9]

The standard treatment for HRS consists of a combination of antiviral agents and high-dose corticosteroids. Antivirals such as acyclovir inhibit viral replication and limit the formation of new lesions.[1,5,6,8] Corticosteroids, such as prednisone, reduce inflammation and neural edema. Administration of antiviral agents within the first 72 hours is crucial for treatment effectiveness and to improve the recovery rate of facial paralysis.[4,5,8] Regarding complementary treatments, such as eye closure and the use of artificial tears, as long as there is no recovery of eyelid closure, they are essential to prevent ocular complications. Facial kinesiotherapy has been recommended for cases with poor response to drug treatment; in this case, the early institution of combined therapy contributed to the patient's favorable outcome. Eye protection is an essential part of management because the inability to close the eye (lagophthalmos) can lead to a complication, exposure keratopathy.[5,10] Kinesiotherapy may be useful if PFP persists even after 4 to 8 weeks.[6] The prognosis for SRH is generally less favorable than that for Bell's palsy, with a lower rate of complete recovery of facial function and a higher incidence of synkinesis.[2,10] However, recovery in children tends to be better than in adults. Factors associated with a worse prognosis include advanced age (>50 years), greater axonal damage, the presence of oropharyngeal lesions, multiple cranial neuropathies, and diabetes. Vaccination against VZV in childhood may reduce the risk of HRS and its complications.[3]

CONCLUSIONS

This case of Ramsay Hunt syndrome in a preschool patient, with an atypical presentation such as predominantly periorbital and facial vesicular lesions, underscores the importance of maintaining a high index of diagnostic suspicion, even in the absence of the classic ear rash. The severity of the initial ocular involvement emphasizes the need for thorough evaluation and immediate ocular protection measures. Early diagnosis and aggressive implementation of combination therapy with antivirals and corticosteroids are crucial to improve prognosis and prevent long-term complications, especially in vulnerable populations such as children.

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CONSENT

The patient's consent was obtained for this study.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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