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A CLINICAL CASE: RAMSEY HUNT SYNDROME IN A CHILD

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Summary

Ramsay Hunt syndrome, a manifestation of Varicella Zoster Virus (VZV) reactivation, represents a rare complication in pediatric patients and is characterized by facial nerve involvement and a vesicular rash localized to the auricular region. This paper presents a clinical case involving a 7-year-old boy with varicella zoster infection complicated by Ramsay Hunt syndrome in the absence of any signs of immunodeficiency. The diagnosis was confirmed based on clinical findings (facial nerve paresis and auricular vesicles) and supported by laboratory data. Appropriate therapeutic measures were undertaken. The article discusses the etiopathogenesis of VZV, current approaches to diagnosis and treatment, as well as the critical role of preventive vaccination. Particular emphasis is placed on the importance of timely initiation of therapy and interdisciplinary collaboration in order to minimize the risk of neurological complications in pediatric clinical practice.

Key words: *Ramsey-Hunt syndrome; Chickenpox; Herpes zoster; Varicella Zoster Virus; Facial nerve paresis; Clinical case*

Introduction

Ramsay Hunt syndrome is regarded as a complication of herpesvirus infection, characterized by lesions of the geniculate ganglion [1]. In most documented cases, it presents clinically with viral involvement of the skin of the external ear, vesicular eruptions, and peripheral facial nerve paresis [2].

Herpes zoster (HZ) is an infectious disease caused by the varicella-zoster virus (VZV), also known as human herpesvirus type 3. The pathophysiology of herpes zoster includes several stages – from primary infection to viral latency and subsequent reactivation. Herpes zoster is a reactivation of the latent virus following a prior episode of varicella (chickenpox). The source of infection is an individual with active varicella (or, less commonly, varicella-zoster) from the onset of vesicle formation until five days after the appearance of the last skin lesion. The virus is transmitted via the airborne route and demonstrates universal susceptibility; children may contract the infection even during the neonatal period. Vaccination remains the cornerstone of prevention and is included in routine immunization schedules in many countries. . In Ukraine, varicella vaccination is administered in accordance with epidemiological indications – particularly for healthy children aged 12 months or older who have not had chickenpox, as well as for healthcare and education professionals at high risk of exposure [3,4].

The pathogenetic mechanisms of VZV infection are well established. The virus enters the body through the mucosal surfaces of the upper respiratory tract. During the initial phase of the incubation period, it replicates rapidly within regional lymph nodes and subsequently disseminates to the reticuloendothelial system, including the spleen and liver, via the bloodstream. Toward the end of the incubation period, secondary viremia occurs, allowing the virus to spread to epithelial cells of the skin and mucous membranes, clinically presenting as vesicular eruptions [5,6]. The formation of the varicella vesicle (blister) starts with the protoplasm in the spinous layer of epithelial cells that swell. At the subsequent stage, reticular dystrophy is observed, characterized by fluid accumulation and the persistence of the cell's protoplasm solely in the form of septa, resulting in the formation of a radial network. The next ballooning dystrophy stage is characterised by fluid accumulation, which leads to swelling of the membrane and nucleus until they are completely dissolved; the cell becomes round, loses its spines and connections with other cells, and transforms into a vesicle turning into a vesicle (balloon). At the early stages of the process, round eosinophilic inclusions, as described by Tietzer, are formed within the nuclei of the affected cells. The virus enters primarily through the mucous membranes of the upper respiratory tract and conjunctiva. It subsequently targets T lymphocytes of the lymphopharyngeal ring (Pirogov–Waldeyer's ring), predominantly activated CD4+ memory T cells. Dendritic cells may also serve as early target cells, which then migrate to peripheral lymph nodes, where they transmit the virus to T lymphocytes. Notably, VZV demonstrates tropism for cells of the nervous system [7, 8, 9, 10].

When vesicular contents are stained with silver, numerous minute coccoid structures – referred to as Aragão bodies – can be observed. These formations may appear singly, in pairs, or in chains. Electron microscopy studies of VZV have revealed its presence in the nuclei of infected cells, consisting of a central body enclosed by a membrane [11]. Extracellular viral particles have a double shell and a brick-like appearance. Herpes zoster may lead to several serious complications, including postherpetic neuralgia, meningitis, encephalitis, and herpetic keratitis. One notable manifestation of VZV reactivation is Ramsay Hunt syndrome, which occurs when the ganglion of the seventh paired cranial nerve (facial nerve) becomes involved [12-18].

Clinical case. A 7-year-old male patient was admitted to the infectious diseases department of a pediatric hospital with complaints of a maculopapular rash and signs of facial nerve paresis. According to the medical history, the illness

began five days prior with the onset of a rash. . For the first three days, the child continued attending school, as the mother assumed the cutaneous manifestations were allergic in nature and would resolve spontaneously. By the end of the third day, peripheral facial nerve paresis developed Medical attention was sought on the fifth day after the rash appeared, and no treatment had been initiated prior to hospital admission. Vaccination history: According to the mother, all scheduled vaccinations were administered. However, the child had not received immunization against varicella. Epidemiological history: The mother reported that the child had not previously contracted any common childhood infectious diseases.

On physical examination, the patient's general condition was assessed as moderately severe. Vital signs were as follows: body temperature – 36.8 °C; respiratory rate – 20 breaths per minute; heart rate – 112 beats per minute; peripheral oxygen saturation (SpO_2) – 98%. The skin and visible mucous membranes appeared pale pink. The oropharyngeal mucosa was pink and mildly granular. Nasal breathing was unobstructed. Vesicular breath sounds were present bilaterally. Cardiac auscultation revealed rhythmic activity. The abdomen was soft and non-distended; the liver was palpable 1 cm below the costal margin. Muscle tone and reflexes were preserved and symmetrical (right = left). Isolated vesicular lesions were observed in the region of the external auditory canal and retroauricular area on the left side, accompanied by localized erythema and tenderness upon palpation. Visual inspection of the face revealed marked asymmetry attributable to peripheral left-sided facial nerve paresis:: the angle of the mouth was depressed, the nasolabial fold on the left was flattened, the left eyelid could not be fully closed, and tongue protrusion revealed deviation to the right (Fig. 1, Fig. 2). Gait was unaffected, meningeal signs were negative.

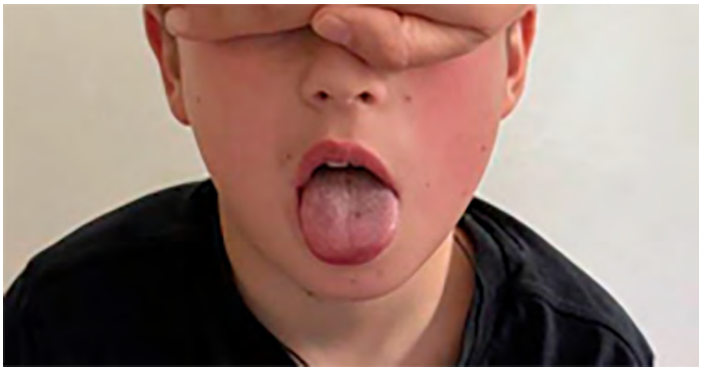

Fig. 1. Paresis of the facial nerve. Deviation of the tongue

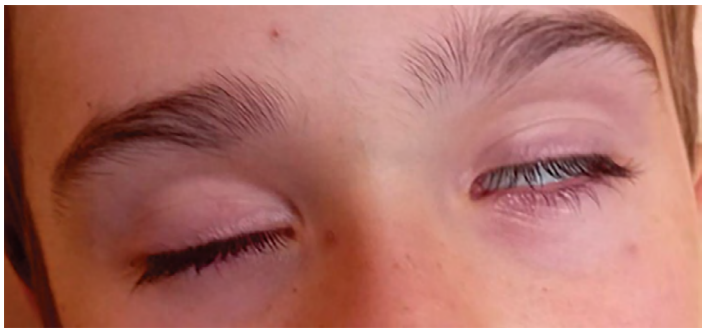

Fig. 2. Paresis of the oculomotor nerve.

To confirm the diagnosis, exclude concomitant pathology, and evaluate the presence of a systemic inflammatory response, a series of clinical and laboratory investigations were conducted (Table 1, Table 2). The results of the complete blood count and biochemical blood analysis revealed no significant deviations from the reference ranges, indicating the absence of systemic inflammation or associated comorbidities. Both the general urinalysis and stool examination (coprogram) were within normal limits.

Table 1

Indicators of the patient's complete blood count

Indicator	Results
Erythrocytes, T/l	4.57
Haemoglobin, G/l	119
Thrombocytes, G/l	272
CRP, mg/l	1.63
Leukocytes, G/L	7.62
Granulocytes, %	54.9
Stab neutrophils, %	3
Segmented neutrophils, %	49
Eosinophils, %	4
Lymphocytes, %	38
Monocytes, %	6

Table 2

Indicators of biochemical blood test of the patient

Indicator	Results
Glucose, mmol/l	5,93
Total protein, g/l	78,4
Total bilirubin, μmol/l	4,8
Indirect bilirubin, μmol/l	-
AlAT, IU/l	19,7
AsAT, IU/l	21,3
Creatinine, μmol/l	51,7
Urea, mmol/l	5,79
Amylase, U/l	41,5
Ca2+ (ionised), mmol/l	1,17
K+ (ionised), mmol/l	4,7
Na+ (ionised), mmol/l	145,3

Based on the patient's history, clinical presentation, physical examination findings, and the results of laboratory investigations, a clinical diagnosis of Ramsay Hunt syndrome was established. The patient received antiviral therapy with acyclovir at a dose of 20 mg/kg, in conjunction with age-appropriate corticosteroid therapy (dexamethasone). Topical antiseptic solutions were used to manage the vesicular rash and to prevent secondary bacterial infection. Following appropriate pharmacological management, the patient's condition demonstrated clinical improvement.

Discharge recommendations included follow-up observation by a local pediatrician and neurologist at the patient's place of residence. A reassessment of neurological status was advised after two weeks to monitor the regression of facial nerve paresis. In addition, a consultation with an otolaryngologist was recommended to rule out complications involving the auditory system. The parents were informed about the necessity of varicella vaccination for other family members who had not previously contracted the disease.

Discussion

The diagnosis of Ramsay Hunt syndrome is typically made on clinical grounds, based on the characteristic combination of a vesicular rash localized to the external ear and peripheral facial nerve paresis. In the present report, we describe a rare clinical case of herpes zoster in a child without evident clinical signs of immunodeficiency, which was complicated by unilateral facial muscle palsy. Treatment involved a combination of acyclovir and high-dose methylprednisolone, resulting in a favorable clinical outcome. This case is notable for the first-time detection of immune status abnormalities, which may have contributed to the development of herpes zoster in a child.

An analysis of the potential causes of complications associated with herpes zoster and varicella reveals multiple contributing factors, including viral characteristics, host immune response, and other systemic vulnerabilities [19]. A diminished immune response is observed in individuals with immunosuppression – such as newborns, pregnant women, patients with primary or secondary immunodeficiencies, and those receiving immunosuppressive therapy – which permits uncontrolled viral replication and increases the risk of severe complications. The virus poses a particular threat to immunocompromised individuals. In adults, the immune response may be less robust compared to that of children, predisposing them to more severe sequelae such as pneumonia or encephalitis. Additionally, patients with underlying chronic conditions (e.g., cardiovascular or respiratory diseases) are at greater risk of complications from varicella due to their already compromised systemic health. Secondary bacterial infections may also arise, as the varicella rash can serve as an entry point for pathogens, potentially leading to conditions such as pyoderma or cellulitis.

Inadequate or inappropriate treatment, including the administration of insufficient therapeutic dosages, further elevates the risk of complications, particularly in vulnerable populations. This underscores the importance of targeted prevention strategies, especially routine immunization against varicella within the general population.

For the purposes of differential diagnosis, Table 3 presents an overview of rare complications associated with varicella. It includes a summary of their clinical manifestations, diagnostic approaches, and key therapeutic strategies.

Ramsay Hunt syndrome presents a complex interdisciplinary challenge in terms of both diagnosis and treatment. Accurate diagnosis requires coordinated efforts among specialists from various fields, including otorhinolaryngology, neurology, and infectious diseases. The classical clinical presentation is typically well-defined, comprising a vesicular rash on the external ear and tympanic membrane, accompanied by otalgia and facial muscle dysfunction due to peripheral facial nerve palsy [20, 21, 22]. However, as originally described by Hunt, clinical manifestations may vary and include pathological changes not only in the external ear but also in the pharynx, often in combination with lesions of the trigeminal, parietal, lingual, abducens, and vagus nerves. In rare cases, the optic nerve may be affected, and serious central nervous system complications such as viral encephalitis and cerebral vasculitis may develop. The literature outlines conventional therapeutic strategies based on the use of antiviral agents in combination with glucocorticosteroids. Nevertheless, the optimal duration of therapy and appropriate dosages of specific drugs remain subjects of ongoing clinical debate. Delayed initiation of antiviral and corticosteroid therapy, or the use of subtherapeutic doses, may contribute to disease progression and elevate the risk of complications and adverse outcomes. The prognosis is significantly worsened in the presence of comorbid conditions such as diabetes mellitus, oncological diseases, or HIV infection [23, 24, 25, 26, 27]. Furthermore, severe cases and complications of varicella are frequently associated with underlying immunodeficiencies [28, 29, 30].

Table 3

Overview of rare complications of varicella in the context of differential diagnosis

Complications	Description	Symptoms	Diagnosis	Treatment
Pneumonia	Inflammation of the pulmonary tissue induced by the varicella-zoster virus, predominantly observed in adults.	Fever, productive or dry cough, pleuritic chest pain, and dyspnea.	Chest radiography and polymerase chain reaction (PCR) testing for varicella-zoster virus.	Antiviral therapy (e.g., acyclovir), empirical antibiotic therapy, and oxygen supplementation.
Meningoencephalitis	Inflammatory involvement of the brain and spinal cord (encephalomyelitis).	Headache, nuchal rigidity, fever, and altered mental status.	Magnetic resonance imaging (MRI) of the brain and cerebrospinal fluid (CSF) analysis.	Antiviral agents, systemic corticosteroids, and supportive symptomatic management.
Venous sinus thrombosis	Cerebral venous vessel obstruction, potentially secondary to viral infection.	Headache, epileptic seizures, and impaired consciousness.	MRI or computed tomography (CT) of the brain, supplemented by cerebral angiography.	Anticoagulant therapy in combination with antiviral treatment.
Bacterial skin infections	Secondary bacterial wound infections arising in the context of varicella-related skin lesions.	Purulent vesicles, localized pain, edema, and erythematous skin lesions.	Wound swab culture and microscopic examination.	Systemic or topical antibiotics and local antiseptic application.
Hemorrhagic syndrome	A rare complication characterized by hemorrhagic manifestations and coagulopathy.	Epistaxis, tissue edema, and cutaneous hemorrhagic manifestations.	Complete blood count (CBC), coagulation profile (coagulogram), and PCR testing for viral pathogens.	Administration of fresh frozen plasma, platelet concentrates, and antiviral medications.
Acute myelitis	Inflammation of the spinal cord (myelitis), which may result in motor dysfunction or paralysis.	Muscle weakness, flaccid or spastic paralysis, sensory deficits, and dorsalgia.	MRI of the spinal cord and PCR testing for varicella-zoster virus.	Systemic corticosteroids and antiviral agents.
Keratitis	Inflammation of the corneal tissue (keratitis).	Ocular pain, conjunctival hyperemia, visual impairment, and ocular discharge.	Ophthalmoscopic examination and fluorescein staining test.	Topical antiviral eye drops and corticosteroid preparations.
Paralysis of the facial nerve (Ramsey Hunt)	Facial nerve palsy resulting from varicella-zoster virus reactivation.	Facial asymmetry, lagophthalmos, reduced facial muscle tone.	Clinical neurological assessment and electroneuromyography (ENMG).	Systemic corticosteroids, antiviral therapy, physiotherapeutic interventions.

Conclusions

Ramsay Hunt syndrome, as a rare complication of varicella, may occur in children without signs of immune dysfunction, as demonstrated by the presented clinical case. The atypical course of varicella and herpes zoster, along with their associated complications, may present with diverse clinical manifestations, necessitating a high index of suspicion among the medical community. Primary prevention through immunisation against Varicella Zoster Virus is essential in reducing the incidence of such complications. An optimal prognosis depends on early initiation of aetiotropic therapy and coordinated multidisciplinary care.

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НЕОНАТОЛОГІЯ, ХІРУРГІЯ ТА ПЕРИНАТАЛЬНА МЕДИЦИНА
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КЛІНІЧНИЙ ВИПАДОК: СИНДРОМ РЕМСІ-ХАНТА У ДИТИНИ

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Резюме.

Синдром Ремсі-Ханта, як прояв реактивації вірусу Varicella Zoster, є рідкісним ускладненням у дітей, що супроводжується ураженням лицьового нерва та везикулярними висипами вушної локалізації. У роботі описано клінічний випадок 7-річного хлопчика з вітряною віспою, ускладненою синдромом Ремсі-Ханта без ознак імунної недостатності. Діагноз підтверджено клінічно (парез лицьового нерву, везикули за вухом) та лабораторно, було проведено лікування. У статті розглянуто етіопатогенез VZV, підходи до діагностики і терапії, а також значення профілактичної вакцинації. Підкреслюється важливість своєчасного лікування та міждисциплінарної взаємодії для мінімізації неврологічних ускладнень у педіатричній практиці.

Ключові слова: Синдром Ремсі-Ханта; вітряна віспа; оперізуючий герпес; Varicella Zoster Virus (VZV); парез лицьового нерву; клінічний випадок.

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