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Peripheral facial nerve palsy in a child with reactivated Epstein–Barr virus infection and human herpesvirus 6 infections: a case report

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Neurological complications of Epstein–Barr virus (EBV) infection include a wide range of diseases with the involvement of both central and peripheral nervous systems. Peripheral facial nerve palsy is the most common cranial nerve damage and herpesviruses play an important role in its etiology. We present a clinical case of peripheral facial nerve palsy associated with reactivation of EBV infection and human herpesvirus type 6 (HHV-6) infection in a 16-year-old boy.

The aim of the study is to describe a clinical case of peripheral facial nerve palsy in a child with associated herpesvirus infection.

Clinical case. A 16-year-old boy was hospitalized in the neurology department with complaints of numbness of the tongue, lips, enlargement of the submandibular lymph nodes and parotid salivary glands, facial asymmetry, dry eyes, and mild redness of the sclera. The submandibular lymph nodes were enlarged to 2.5 cm in diameter, tender to palpation, mobile, and not adherent to the surrounding tissues. The parotid and sublingual salivary glands are enlarged, dense, and painful on palpation. Pathological neurological symptoms were detected: sagging eyebrows, lagophthalmos on the left side, nasolabial folds S<D, and drooping of the left corner of the mouth. It was performed: DNA HHV-6 – $1.0^3 \times 10^4$ copies and DNA EBV – 7.65×10^5 copies were detected. Immune enzyme analysis of serum revealed positive anti-VCA IgG, anti-EA IgG, and anti-EBNA IgG. Antiviral treatment was prescribed – valganciclovir and recombinant human interferon alpha-2b. On the background of antiviral therapy from week 3, a noticeable regression of neurological symptoms was observed, and by week 5, the size of the salivary glands had normalized.

Conclusions. In the case of neuropathy, EBV and its association with other herpesviruses, particularly HHV-6, should be considered as a possible etiologic factor. Tests to detect these pathogens should be included in the list of workups for such patients. In addition to the characteristic lymphadenopathy, sialoadenitis is one of the important clinical markers of replicative forms of infections caused by EBV and HHV-6.

The study was conducted in accordance with the principles of the Declaration of Helsinki. The informed consent was obtained from the patient and parents. The authors declare no conflict of interest.

Keywords: sialoadenitis, facial nerve palsy, peripheral neuropathy, Epstein–Barr virus, human herpesvirus type 6, children, case report.

Периферичний параліч лицевого нерва в дитини з реактивацією Епштейна–Барр вірусної інфекції та інфекції, спричиненої вірусом герпесу людини 6 типу: клінічний випадок

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До неврологічних ускладнень Епштейна–Барр вірусної інфекції (ЕБВ) належить широкий спектр захворювань з ураженням як центральної, так і периферичної нервової системи. Периферичний параліч лицевого нерва є найпоширенішим пошкодженням черепних нервів і віруси герпесу відіграють важливу роль в його етіології. Представлено клінічний випадок периферичного паралічу лицевого нерва, пов'язаного з реактивацією ЕБВ та інфекції, спричиненої вірусом герпесу людини 6 типу (ГВЛ-6) у 16-річного хлопця.

Мета: описати клінічний випадок периферичного паралічу лицевого нерва в дитини з асоційованою герпесвірусною інфекцією.

Клінічний випадок. Хлопець, 16 років, госпіталізований до неврологічного відділення зі скаргами на оніміння язика, губ, збільшення підщелепних лімфатичних вузлів та привушних слинних залоз, асиметрію обличчя, сухість очей, незначне почервоніння склер. Підщелепні лімфатичні вузли збільшені до 2,5 см у діаметрі, чутливі при пальпації, рухливі, не спаяні з оточуючими тканинами. Привушні та під'язикові слинні залози збільшені, щільні, болючі при пальпації. Виявлено патологічні симптоми: опущення брів, лагофтальм зліва, носогубні складки S<D, опущення лівого кута рота. Підтверджено присутність ДНК ГВЛ-6 – $1{,}0^3 \times 10^4$ копій та ДНК ЕБВ – $7{,}65 \times 10^5$ копій та позитивні анти-VCA IgG, анти-EA IgG та анти-EBNA IgG. Призначено противірусне лікування – валганцикловір і рекомбінантний людський інтерферон альфа-2b. На фоні противірусної терапії з 3-го тижня спостерігався помітний регрес неврологічної симптоматики, а до 5-го тижня нормалізувалися розміри слинних залоз.

Висновки. При нейропатії можливим етіологічним фактором слід розглядати ЕБВ та його асоціації з іншими герпесвірусами, зокрема ГВЛ-6. Дослідження для виявлення цих збудників повинні бути включені у список обстежень для таких пацієнтів. Окрім характерної лімфаденопатії, сіалоаденіт є одним із важливих клінічних маркерів реплікативних форм інфекцій, спричинених ЕБВ та ГВЛ-6.

Дослідження виконано відповідно до принципів Гельсінської декларації. На проведення досліджень отримано інформовану згоду пацієнта та батьків. Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: сіалоаденіт, параліч лицевого нерва, периферійна нейропатія, вірус Епштейна–Барр, герпесвірус людини 6 типу, діти, клінічний випадок.

Introduction

Around 90-95% of the population is infected with Epstein-Barr virus (EBV) worldwide. EBV infection in the human body can be latent or replicative. B lymphocytes and oropharyngeal epithelial cells are the target cells for EBV. Primary EBV infection is asymptomatic or manifests as infectious mononucleosis [10]. EBV can be detected in the saliva of patients during the acute stage of the disease, as well as in healthy seropositive

individuals [2]. Reactivation of latent EBV infection in immunosuppressed individuals is associated with a wide range of diseases, including neurological ones [5.11]. The frequency of neurological complications of EBV infection is estimated to be between 0.37–7.3% [6]. EBV can directly or indirectly infect neurons via infected B lymphocytes, induce inflammation and demyelination, and promote glial cell proliferation, degeneration, and necrosis. The central nervous system (CNS) lesions associated with EBV include meningitis, encephalitis, and acute disseminated encephalomyelitis [19]. The role of EBV in the development of diseases of the CNS – Alzheimer's disease, Parkinson's disease, multiple sclerosis (MS), acute cerebellar ataxia, acute disseminated encephalomyelitis, and brain tumors – is being actively studied [19,4]. The following manifestations of peripheral nervous system (PNS) damage in EBV infection are possible: myeloradiculitis, encephalomyeloradiculitis, Guillain-Barré syndrome, and cranial nerve paralysis [11,5,8].

Current research is focused on the neurotropic properties of human herpesvirus type 6 (HHV-6), its association with encephalitis, seizure disorders, Alzheimer's disease, and MS is being studied [4]. Both primary infection with HHV-6 and its reactivation result in acute seizures associated with encephalitis and status epilepticus [3]. A positive correlation was found between the severity of nerve fiber damage and HHV-6 infection in patients with fibromyalgia [14]. Active or latent HHV-6 infection in immune and glial cells can alter the balance between demyelination and remyelination, which determines MS progression [4].

Peripheral facial nerve palsy (PFNP) is the most common cranial nerve damage in children and adults. In most cases, it is not possible to identify the cause, so the diagnosis of idiopathic Bell's palsy is established [9,17]. Neuroborreliosis, as well as herpesvirus infections – varicella-zoster virus (VZV), human herpesvirus type 1 (HSV-1), EBV, cytomegalovirus (CMV), and HHV-6 are the most common infectious causes of PFNP. Information on the etiologic role of most of the above-mentioned pathogens is based on clinical case reports. There is a lack of systematic studies in children that demonstrate results on infectious factors of PFNP, and little is known about the frequency and significance of other pathogens that may be potentially involved in the development of PFNP in children [17]. Cases associated with diseases such as influenza, measles, and mumps are described [13]. We present a clinical case of peripheral facial nerve palsy associated with reactivation of EBV infection and HHV-6 infection in a Ukrainian 16-year-old boy.

The aim of the study is to describe a clinical case of peripheral facial nerve palsy in a child with associated herpesvirus infection – EBV and HHV-6.

The results of clinical examination, laboratory, and instrumental tests of a 16-year-old patient hospitalized in the neurological department were analyzed. The study was performed in accordance with the principles of the Declaration of Helsinki. The informed consent of the the patient and his parents was obtained for the study. The study was approved by the Ethics Committee of the Ivano-Frankivsk National Medical University, protocol No. 147/24, November 6, 2024.

Clinical case

A 16-year-old Ukrainian boy presented to the neurology department on the second week of illness and was hospitalized with complaints of numbness of the tongue, lips, enlargement of the submandibular lymph nodes and parotid salivary glands, facial asymmetry, dry eyes, and mild redness of the sclera. These symptoms appeared 1.5 weeks before admission and increased gradually, the patient was examined and treated on an outpatient basis by a family doctor, ibuprofen and amoxicillin with clavulanic acid were prescribed. He had not been ill for 3 months before the onset of symptoms. From his medical history, it is known that the boy was born from a full-term pregnancy, complicated by intracranial hemorrhage. He grew and developed according to his age. There is no history of hereditary or allergic diseases. The boy was vaccinated according to the National Immunization Schedule of Ukraine. At the age of 10, he was diagnosed with EBV infection with the persistence of the virus in the oropharynx (EBV DNA was detected in a polymerase chain reaction (PCR) test of tonsil scrapings and saliva). Over the next 4 years, exacerbations of EBV infection were observed 2 times a year, manifested by febrile fever, and pharyngotonsillitis, without generalized lymphadenopathy syndrome. He had acute respiratory viral infections no more than 4 times a year without complications.

On objective examination, the patient's general condition is of moderate severity. No rash on the skin and visible mucous membranes. The tonsils are first-degree hypertrophied, moderately hyperemic,

and without exudates. The posterior pharyngeal wall is clean, and normal in color. The submandibular lymph nodes are enlarged to 2.5 cm in diameter, tender to palpation, mobile, and not adherent to the surrounding tissues. The parotid and sublingual salivary glands are enlarged, dense, and painful on palpation. During auscultation, vesicular breathing in the lung is heard. Heart sounds are rhythmic. Blood pressure is 125/80 mm Hg, heart rate – 85/min. The abdomen is soft, and not painful. The liver and spleen are not enlarged. The stool is 1 time per day with normal color and consistency. Diuresis is normal.

Neurological status of the patient: consciousness is clear, and his intellect is preserved. Cranial nerves – eye slits D=S, pupils D=S, no nystagmus. Pathologic symptoms were detected: sagging eyebrows, lagophthalmos on the left side, nasolabial folds S<D, and drooping of the left corner of the mouth. According to the House–Brackmann facial nerve grading system scale – 3–4 points. Active and passive movements in the limbs are full, muscle normotone, tendon reflexes D=S, active. Statics and coordination are not impaired. There were no seizures. Meningeal symptoms were negative.

Laboratory test results:

Complete blood count (May 31, 2024): Hemoglobin 154 g/L, erythrocytes 5.53×10^{12}/L, leukocytes 6.5×10^{9}/L, eosinophils 4%, band neutrophils 0%, segmented neutrophils 66%, lymphocytes 26%, monocytes 2%, ESR 4 mm/hour.

Complete blood count (June 6, 2024): Hemoglobin 154 g/L, erythrocytes 5.10×10^{12}/L, leukocytes 8.04×10^{9}/L, eosinophils 4%, basophils 1%, band neutrophils 5%, segmented neutrophils 60%, lymphocytes 25%, monocytes 5%, ESR 5 mm/hour.

Biochemical blood tests (June 3, 2024): total protein 70 g/L, urea 6.7 mmol/L, creatinine 83.6 μmol/L, C-reactive protein (CRP) 7.79 mg/L, total bilirubin 12.8 μmol/L, direct bilirubin 5.0 μmol/L, indirect bilirubin 7.8 μmol/L, alanine transaminase (ALT) 17 IU, aspartate transaminase (AST) 13 IU, glucose 4.4 mmol/L, lactate dehydrogenase 148 IU.

Coagulogram (June 4, 2024): prothrombin time 12.2 sec, prothrombin index 102.2%, International Normalized Ratio (INR) 1.02, fibrinogen 2.55 g/L.

Instrumental tests were conducted:

Magnetic resonance imaging (MRI) of the brain on May 31, 2024: no pathological structural changes in the brain were visualized at the time of the examination.

Chest X-ray (June 10, 2024): the lung fields are evenly pneumatized. Pulmonary pattern is without clear focal changes and diffuse changes. The sinuses are intact. The heart shadow is within normal limits. There is relaxation of the diaphragm.

Ultrasound examination of the abdominal cavity (May 29, 2024): The liver is located typically, does not protrude from under the rib arch, the right lobe is 142 mm, and the left lobe – 67 mm. The liver parenchyma is of normal echogenicity. The bile ducts are not compacted, the gallbladder is 11 cm^3, its wall is not thickened, not compacted, and the contents are homogeneous. The pancreas is visualized throughout, the contours are clear, and even, the structure is somewhat heterogeneous, due to hyperechoic linear inclusions. The spleen is not enlarged, size is 123×48 mm. The kidneys are typically located. The left kidney is 110×43 mm, parenchyma is 13 mm. The right kidney is 121×42 mm, parenchyma is 13 mm. The contours of the kidneys are even, and clear. Differentiation of the cortical and medullary layers is preserved. Small echo+ inclusions in both kidneys. Bladder – volume 20 cm^3. The wall is 1.5 mm. The contents are anechogenic.

Ultrasound examination of lymph nodes (May 29, 2024) – near the right angle of the lower jaw lymph nodes 23×8.5 mm, on the left side lymph nodes 12 mm. In the submandibular area on the right side lymph node 8 mm, on the left side – lymph node 8 mm. Along the vascular bundle of the neck on the right side lymph nodes are 6–9 mm, and on the left side – 8–9 mm. Under the sternocleidomastoid muscle on the right lymph node 6 mm, on the left – 6 mm. Posterior cervical lymph nodes on the right side – 5 mm, on the left – 6 mm. Supraclavicular lymph nodes on the right side – 2–3 mm, on the left side – 2–3 mm. In the sublingual area, there is a conglomerate of lymph nodes measuring 10×4.3 mm, 14×6.5 mm, 8×4.5 mm, 7.5×4 mm, 5 mm, of medium echogenicity, with preserved cortical-cerebral differentiation, with central blood supply, without signs of liquefaction. The subclavian lymph nodes are not enlarged. Axial lymph nodes on the right side – 5–8 mm, on the left – 6–7–8 mm. Paraumbilical lymph nodes – 8–10–12 mm. Inguinal lymph nodes on the right side – 1–12 mm, on the left side – 8–10 mm. No enlarged lymph nodes or additional masses were detected in the anterior-upper mediastinum.

Ultrasound examination of parotid salivary glands (May 29, 2024). The parotid gland on the right side is

46×17×21 mm, structurally heterogeneous, solitary lymph nodes are 6–10 mm. The parotid gland on the left side is 45×15×23 mm, structurally heterogeneous, lymph nodes lobules are 5–7 mm. The sublingual salivary gland is 29×12×21 mm in size, structurally heterogeneous, solitary lymph nodes are 2–3–4 mm.

Ultrasound examination of parotid salivary glands (June 3, 2024). The right parotid gland is 45×16×20 mm, structurally heterogeneous, solitary lymph nodes are 4–8 mm. The left parotid gland is 44×15×22 mm, structurally heterogeneous, solitary lymph nodes are 4–6 mm. The size of sublingual salivary gland is 27×10×20 mm, structurally heterogeneous, solitary lymph nodes are 2–3–4 mm.

Based on the complaints, medical history, objective examination data and test results, the diagnosis was established: acute neuropathy of the left facial nerve.

For detection of the neuropathy cause the patient underwent further tests.

Cerebrospinal fluid (CSF) test (June 5, 2024): colorless, transparent, protein – 0.33 g/L, Pandy test +, cytosis – 3 cells/μL, lymphocytes – 50%, granulocytes 50%, no cells with signs of atypia. Glucose – 3.22 mmol/L, chlorides – 117.6 mmol/L.

CSF culture (June 5, 2024): negative.

Immune enzyme analysis of serum (June 4, 2024): *Borrelia burgdorferi* IgM – negative, *Borrelia burgdorferi* IgG – negative; antiVCA IgM – negative, antiVCA IgG – 11.9 (positive), anti EA IgG – 30.0 (positive), anti EBNA IgG – 9.48 (positive), anti HSV1/2 Ig M – negative, anti HSV1/2 Ig G 9.2 – positive, anti CMV Ig M – negative, anti CMV IgG – 13.6 – positive. Antibodies to human immunodeficiency virus (HIV) – negative.

PCR of blood (June 4, 2024) – DNA HSV-1 – not detected, DNA HSV-1 – not detected, DNA HHV-6 – $1.0^3 \times 10^4$, DNA EBV – 7.65×10^5, DNA CMV – not detected.

Neuron specific enolase (June 4, 2024): 5.45 ng/mL.

Procalcitonin (June 4, 2024): 0.05 ng/mL

The patient was consulted by specialists who provided the following conclusions.

Ophthalmologist: two eyes – normal, optic media clear. Fundus: optic discs are pink, borders clear, narrowed excavation, retinal angiopathy. The periphery is intact.

Dentist: diagnosis: sialoadenitis

Otolaryngologist diagnosed edema of the nasal mucosa and no evidence of acute otitis media. Consultation with an audiologist: diagnosis: hearing is within normal limits.

Infectious diseases specialist: diagnosis: herpesvirus infection caused by EBV and HHV type 6, replication phase (PCR of blood: EBV DNA positive, HHV-6 DNA positive) with damage of the peripheral nervous system (mononeuritis of the left facial nerve), parotid and sublingual salivary glands.

During inpatient treatment, the patient received intravenous saline, mannitol, furosemide, dexamethasone, magnesium sulfate, oral nimesulide, acetazolamide, magnesium asparaginate and potassium asparaginate, phonophoresis with hydrocortisone on the left side of the face.

Since the blood PCR test revealed HHV-6 DNA ($1.0^3 \times 10^4$ copies) and EBV DNA (7.65×10^5 copies), valganciclovir at a dose of 450 mg twice daily orally for 21 days, rectal suppositories of recombinant human interferon alpha-2b, 1000,000 twice daily, morpholine salt of thiazolic acid at a dose of 200 mg three times daily orally, a complex of amino acids for oral administration (arginine, betaine and L-carnitine) were prescribed. The patient was discharged with improvement in condition. Follow-up by a neurologist, massage, and gymnastics are recommended. On the background of antiviral therapy from week 3, a noticeable regression of neurological symptoms was observed, and by week 5, the size of the salivary glands normalized.

Discussion

Nervous system lesions associated with EBV infection and HHV-6 may be underdiagnosed. Facial nerve paralysis associated with EBV infection is quite rare, but this complication has been described in the literature. The incidence of facial nerve palsy associated with EBV infection is unknown, it can be unilateral or bilateral [6].

The cases of peripheral nerve injury in young children with acute EBV infection are described, including bilateral facial nerve paralysis associated with EBV infection in a 14-month-old girl [18]. Peripheral facial nerve paralysis in a 10-year-old boy with a confirmed reactivated EBV infection was described, he recovered spontaneously within 2 weeks [8].

A case of grade III left-sided peripheral facial paralysis in a male infant of 23 months is described. Tests for various viral infections were performed, EBV in the serum sample was detected. Serological test results revealed positive anti-VCA IgM, negative anti-VCA IgG and anti-EBNA IgG. IgM antibodies against CMV, Herpes simplex, and Lyme disease were near the cutoff levels and were regarded as

non-specific results due to the polyclonal activation of lymphocytes. [1].

A clinical analogue of the case described by us is described in the literature. Parotid mass and facial nerve paralysis were diagnosed in a 10-year-old child, without fever, lymphadenopathy, and pharyngitis. A neoplastic process was suspected, a biopsy was prescribed, and the decision was made to perform a parotidectomy. However based on the results of serological examination, acute EBV infection was confirmed, so the patient received conservative treatment. After 6 weeks of follow-up, the right parotid gland mass disappeared, and the function of the right facial nerve improved, but mild residual paresis persisted [15].

A case of facial nerve inflammation after influenza was described in a 15-year-old adolescent, and reactivation of herpes simplex virus type I was confirmed [12].

The role of HHV-6 in the onset of facial nerve palsy is being studied. The virus was detected in the saliva of children during the acute period of the disease, and its amount decreased with the regression of symptoms [7]. In patients with Bell's palsy, this virus is detected in saliva by PCR more often than in healthy patients, and the amount of virus in patients is higher than in healthy persons [16]. Our clinical experience confirms this opinion.

The peculiarity of the case described by us is that mononeuropathy arose on the background of the reactivation of two herpesviruses with neurotropic properties – EBV and HHV-6. The following manifestations of EBV infection are noteworthy: lymphadenopathy and, more rarely, sialoadentitis in the form of parotitis and affecting the sublingual gland. Although the differential diagnosis was primarily focused on neuroborreliosis, HIV, tumor, lymphoma, it was possible to confirm the herpesvirus etiology of the disease based on serological and virological markers. Sialoadenitis is an additional clinical marker of herpesvirus reactivation. These herpesviruses can persist asymptomatically in the salivary glands, and when the infection reactivates, they can induce an inflammatory process.

Conclusion

In the presence of neuropathy, EBV and its association with other herpesviruses, in particular HHV-6, should be considered as a possible etiologic factor. Tests to detect these pathogens should be included in the list of workups for such patients. In addition to the characteristic lymphadenopathy, sialoadenitis is one of the important clinical markers of replicative forms of EBV and HHV-6 infections, in particular their associations.

The prospects for further research are to study the role of reactivated forms of herpesvirus infections, in particular EBV infection and infection caused by HHV-6, in the occurrence of neurological complications as well as factors that contribute to this.

The authors declare no conflict of interests.

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