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EARLY DETECTION AND TREATMENT OF ACUTE VIRAL ENCEPHALITIS IN CHILDREN

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Abstract: An analysis of 41 cases of acute viral encephalitis in children who underwent inpatient treatment in Andijan from 2019 to 2023 was conducted. The diagnosis of encephalitis caused by HSV type ½ was established in 17.1% of children, VZV etiology - in 41.5%, EBV - in 9.8%, CMV - 4.9%, enterovirus - in 2.4%, combined EBV and CMV etiology - in 2.4%. In 21.95% of children, the etiology of the disease was not established. In most children with viral encephalitis (90.2%), the disease is severe and requires intensive care. In children with viral encephalitis, disturbances of central and peripheral hemodynamics are mainly associated with hypovolemia, complications of the cardiovascular system, and functional changes in cerebral circulation.

Keywords: varicella-zoster virus, viral encephalitis, herpes viruses, diagnostics.

INTRODUCTION

Viral encephalitis is an inflammatory process in the brain associated with clinical manifestations of neurological dysfunction [1]. The incidence among children is 10.5–13.8 per 100 thousand children [2]. Viruses have an overwhelming advantage in etiology, although bacteria, prions, parasites and fungi can also cause the disease. The most common pathogens include herpes simplex viruses types 1 and 2 (HSV type 1/2), varicella-zoster virus (VZV), human herpesvirus type 6 (HHV 6), influenza, measles, mumps viruses, arboviruses, Epstein-Barr virus (EBV) [1]. According to a survey of more than 3,000 patients with encephalitis in Finland, VZV was detected in the cerebrospinal fluid of 29%, HSV in 11%, enteroviruses in 11%, influenza virus in 7%, HHV6 in 5%, adenoviruses in 4%, tick-borne encephalitis virus in 3%, mycoplasma pneumoniae in 3%, EBV in 2% [3]. The prognosis for encephalitis is often unfavorable. Thus, with the most common HSV and VZV encephalitis, the mortality rate is 15-20%, and in survivors, the probability of developing persistent neurological deficits is 12-40% [4, 5]. Publications in recent years on the spread of viral encephalitis among children in Ukraine, their etiological structure and clinical features are isolated and do not reflect general population trends.

MATERIALS AND METHODS

An analysis was conducted of 41 cases of acute viral encephalitis in children who were treated in Andijan in 2019–2023. The children's ages ranged from 3 months to 15 years. Boys accounted for 51.2%, girls – 48.8%. The weight of patients was within 5.3–70.0 kg (M – 24.8 kg (SD \pm 18.32), body area – M – 0.85 m2 (SD \pm 0.46). The diagnosis of acute viral encephalitis was established on the basis of clinical criteria, laboratory and instrumental studies. The diagnosis of encephalitis caused by HSV type ½ was established in 17.1% of children, VZV etiology – in 41.5%, EBV – in 9.8%, CMV – 4.9%, enterovirus – in 2.4%, combined EBV and CMV etiology – in 2.4%. In 21.95% of children, the etiology of the disease was not established.

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RESULTS AND DISCUSSION

In all patients, the onset of the disease was sudden. Neurological symptoms were varied and included lesions of the cerebral hemispheres and brainstem, cerebellum, and peripheral nerves. The most common were impaired consciousness (61.0%), seizures (56.1%), cranial nerve lesions (58.5%), ataxia (41.5%), meningeal syndrome (31.7%), paresis and paralysis of the limbs (29.3%). Among the general symptoms, the most common were fever (90.2%), headache (43.9%), and vomiting (24.4%). Systolic blood pressure was within 81-140 mm $Hg (M - 112.0 \pm 15.63)$, and diastolic pressure was 44–95 mm $Hg (M - 66.5 \pm 12.81)$. Pulse rate was from 56 to 166 /min. (M - 103.9±29.25). Capillary filling time was within normal limits in all patients (< 2 s). In the general blood test, slight leukocytosis was noted in most patients (75.0%), and normal leukocyte count was observed in 25%. A band shift was noted in the leukocyte formula in 87.5% of patients. In all patients with viral encephalitis, inflammatory markers (C-reactive protein, procalcitonin) were within reference values. Changes were detected in cerebrospinal fluid analysis only in 37.5% of patients. They included an increase in protein and leukocyte content mainly due to lymphocytes. Moreover, the increase in protein did not exceed 1 g/l, and leukocytes – up to 100/µl. MRI studies were performed on all patients. Repeated examinations were performed dynamically if indicated. Focal changes in the parenchyma were detected in 51.2% of patients. In half of the patients, structural changes were not observed. The state of the central and peripheral hemodynamics was carried out on all patients during the first three days from the date of hospitalization. The obtained results were compared with the data of the comparison group, which consisted of 20 patients comparable in age, gender and body surface area with the main group. The comparison group included children with normal cardiac function (according to clinical data, ECG, EchoCG results) and without signs of acute inflammatory process. No pathological structural disorders of the heart were detected in the children of the comparison group.

One patient with herpes encephalitis had mitral valve prolapse with mild regurgitation (grade 1). Cardiac pumping function was assessed by stroke index (SI) and systolic index (SI), as well as by left ventricular (LV) outflow tract flow determined by Doppler sonography. Among patients with viral encephalitis, the majority (75.6%) had normal cardiac output, in 6 children (14.6%) it was decreased, and in 4 it was increased.

CONCLUSION

- 1. Herpes viruses predominate in the etiology of viral encephalitis in children: VZV in 41.5% of cases, HSV type 1/2 in 17.1%, EBV in 9.8%, CMV in 4.9%, and a combination of EBV and CMV in 2.4%.
- 2. Most children with viral encephalitis (90.2%) have a severe course of the disease and require intensive care.
- 3. A large percentage of undiagnosed diagnoses of viral encephalitis in patients with persistent neurological disorders (21.95%) requires expanded diagnostic procedures.

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