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Original Article

Cases of Acute Hemiparesis in Childhood

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Abstract

Introduction: Acute hemiparesis is an emergency of various etiologies and possible fatal outcome.

Aim: The aim of this study was to determine the etiology, clinical manifestations, and prognosis of acute hemiparesis in childhood.

Materials and methods: This is a retrospective study of 52 children (28 boys and 24 girls) aged 4 months to 16 yrs 11 months with acute hemiparesis, admitted to the Clinic of Pediatrics at St George University Hospital between 2013 and 2020. After clinical examination, a modern diagnostic and therapeutic approach was used.

Results: The identified causes of acute hemiparesis were ischemic stroke (21), postictal paresis after seizure (9), hemorrhagic stroke (6), venous sinus thrombosis (5), encephalitis (3), transient ischemic attacks (TIAs) (2), posterior reversible encephalopathy syndrome (PRES) (2), hemiplegic migraine (1), demyelinating CNS disease (1), ventriculoperitoneal shunt complication (1), glutaric aciduria type 1 (1), brain tumor (1), and CNS metastasis (1).

The most common manifestations associated with acute hemiparesis were impaired consciousness (27), involvement of cranial nerves – central (25) and peripheral (1) facial nerve palsy, abducens (1), ocolomotorius (1), hypoglossus (1) nerve palsy, seizures (25), raised intracranial pressure (18), speech disorders (15), signs of infection (14), headache (12), and hemibody sensory disturbances (9).

In 8 children (14%), the hemiparesis was permanent, in 2 recurrent, and in 7 (12%) fatal.

Conclusions: Unlike in adult patients, acute hemiparesis in children has many causes, which requires a large number of etiological studies. With a quick diagnosis and adequate treatment, recovery of function in children is more common than in adults – it is achieved in two thirds of children.

Keywords

cerebrovascular disease, neuroimaging, stroke-like conditions

INTRODUCTION

Acute hemiparesis is a medical emergency of various etiologies with a possible fatal outcome. It requires a timely diagnosis and adequate therapy, sometimes including emergency neurosurgery. It is defined as paralysis of one side of the body due to involvement of the corticospinal tract on the contralateral side at the level of the cortex, corona radiata, internal capsule, brainstem, or spinal cord.

Knowledge of the causes and treatment of acute childhood hemiparesis has increased in the last decade of the last century as a result of an initiative of the International Pedi-

atric Stroke Study Group.[1] Structural and functional MRI, conventional MR angiography, and the advances in genetics also contribute to this. Acute hemiparesis is the most common presentation of a vascular stroke, but about 20%-30% of the children with acute hemiparesis have no cerebrovascular involvement but a stroke-like condition – the so-called 'stroke mimics'.[2] In 2015, Bhate and Ganesan described the clinical features that differentiate vascular stroke syndrome from stroke mimics and developed an algorithm for diagnostic and therapeutic approach to acute hemiparesis in children.[3]

Since 2000, there have been few publications examining the incidence, etiology, the clinical presentation, and outcome of acute childhood hemiparesis. The present study analyzes acute hemiparesis on the basis of modern etiological classification, including cerebrovascular and stroke-like diseases. It describes the clinical and radiological characteristics of children with acute hemiparesis, their treatment, and outcome.

AIM

To determine the etiology, clinical manifestations, and prognosis of acute hemiparesis in childhood using a modern diagnostic and therapeutic approach.

MATERIALS AND METHODS

This is a retrospective study of 54 children (28 boys and 26 girls) aged 4 months to 16 yrs 11 months with acute hemiparesis, admitted to the Clinic of Pediatrics at St George University Hospital, Plovdiv between 2013 and 2020. Imaging of the CNS (transfontanelle ultrasound, contrast-enhanced CT, MRI angiography, digital subtraction angiography), EEG, echocardiography, ECG, cerebrospinal fluid examination, biochemical, virologic, serological, immunological, metabolic and genetic tests, and assessment of prothrombotic factors were performed. Conservative etiologic, pathogenetic, and symptomatic treatment was used depending on the cause of the acute hemiparesis.

The data were analyzed using the specialized statistical software SPSS 21. Descriptive statistics were presented as frequency. Chi-square test was used to evaluate the significance of difference. A p value of <0.05 was accepted as statistically significant.

All patients and their parents gave informed consent to be included in scientific studies.

RESULTS

Acute hemiparesis was found in 0.11% (54 of 45820) of the pediatric patients treated in our hospital during the study period. Of the 54 patients included for further analysis, 4 presented at ≤1 year of age, 39 presented at 1-10 years of age, and 11 presented at >10 years of age.

Cerebrovascular diseases (ischemic stroke, transient ischemic attack, hemorrhagic stroke, venous sinus thrombosis, hemiplegic migraine) were the leading causes of hemiparesis among the studied children (65%), ischemic stroke being the most common (39%, n=21) **(Table 1)**.

Table 1. Etiology of hemiparesis

Causes of hemiparesis	n	%
Cerebrovascular diseases:	35	65
Ischemic stroke	21	39
Transient ischemic attack	2	4
Hemorrhagic stroke	6	11
Venous sinus thrombosis	5	9
Hemiplegic migraine	1	2
Stroke mimics:	**19**	**35**
Encephalitis	3	5
Demyelinating CNS disease	1	2
Complications of VPS (ventriculoperitoneal shunt)	1	2
CNS tumors and metastases	2	4
Glutaric aciduria type 1	1	2
Posterior reversible encephalopathy (PRES)	2	4
Postictal paresis	9	16
Total	**54**	**100**

In 11 children (21%), we found accompanying chronic diseases – acute leukemia in 5 patients and 6 other cases with one of the following conditions: osteosarcoma, Down syndrome with atrial septal defect, congenital cardiopathy, Ehlers Danlos syndrome, Marfan syndrome with mitral valve prolapse, and congenital brain malformation with implanted ventriculoperitoneal shunt.

Accompanying acute infection [respiratory (9), gastrointestinal (4), urinary combined with respiratory (1)] was found in 14 children (26%), mostly with stroke mimics (in 11 out of the 19 with stroke mimics) compared to children with cerebrovascular disease in which infection was found in only 3 out of 35 cases (p=0.001, χ^2=15.60). Acute infection was more common in the first decade of life being observed in this study only in children up to 10 years of age (in 14 out of 43 cases) and in none of the 11 cases over the age of 10 years (p=0.028, χ^2=4.83).

Eleven children (20%) had received cytostatics and/or corticosteroids at the onset of hemiparesis. These were cases with PRES (n=2), CNS tumors (n=2), venous sinus thrombosis (n=3) and hemorrhagic stroke in the course of leukemia (n=4) (p=0.001, χ^2=15.60). Thrombophilia was found in 19 (35%) of all cases, all of them with a cerebrovascular cause of hemiparesis. Vascular anomalies were found in 10 (18%) cases, all of them also with a cerebrovascular cause. There were 8 patients with thrombophilia in whom it was com-

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bined with medial cerebral artery pathology, and one with thrombophilia and cardiopathy. The association of cerebrovascular diseases with thrombophilia (p=0.001, χ^2=21.64) or vascular abnormalities (p=0.01, χ^2=6.66) was significant. Of the 21 ischemic strokes identified, 17 (80%) were in the region of medial cerebral artery, one – of the anterior cerebral artery, and one – of the left vertebral artery. Two children presented with acute hemiparesis after minimal head trauma as a result of ischemic stroke in the lenticulostriate vessels region. Ischemic strokes in the basin of *a. cerebri media* were the most common in our study. They were all diagnosed by MR angiography and diffusion sequences.

Hemorrhagic strokes (n=6, 11%) were the next most common vascular cause of acute hemiparesis in the study group. Four such strokes occurred in children with malignancies undergoing chemotherapy. The other 2 hemorrhages were in children with cerebrovascular abnormalities – dural arteriovenous fistulas (DAVFs) and cerebral proliferative angiopathy. All were diagnosed by an emergency computer tomography (CT) of the CNS.

Another vascular cause of acute hemiparesis was venous sinus thrombosis (VST), diagnosed in 5 children (9%). Three of them had leukemia and were treated with corticosteroids and L-asparginase. Thrombophilia was found in 3 children with VST. In 2 children, VST occurred during infection with fever and dehydration – one of them had also fibromuscular dysplasia, and the other – thrombophilia. VST diagnosis was made by emergency CT of the CNS with subsequent magnetic resonance angiography (MRA) or just with MRA.

In the group of children with acute hemiparesis and 'stroke mimics', there were 3 cases of encephalitis – one child with HSV-2 encephalitis, one with varicella encephalitis, and one with autoimmune (NMDR) encephalitis. The diagnosis was made by MRI of the CNS, with abnormalities found in both diffusion-weighted sequences and FLAIR.

The clinical manifestations associated with acute hemiparesis are presented in **Table 2**.

Table 2. Clinical manifestations associated with acute hemiparesis

Clinical signs	N	%
Impaired consciousness	27	50%
Raised intracranial pressure	18	33%
Headache (without raised intracranial pressure)	12	22%
Seizures		
Focal seizures with/without impaired awareness	25 16	47%
Focal seizures with impaired awareness and generalized tonic-clonic seizures Generalized tonic-clonic only	5 4	
Impaired speech (motor aphasia, sensory aphasia and/or dysarthria)	15	28%
Hemicorporal sensory disturbances	9	17%
Cranial nerve involvement	27	50%
Clinical signs of infection, incl. fever	14	26%

Twenty-five children (47%) with acute hemiparesis had also seizures. They were more common in the 'stroke mimics' group (in 15 out of 19 cases, 79%) compared to the cerebrovascular diseases group (seizures in 10 out of 35 cases) (p=0.001, χ^2=12.57**).**

Focal seizures without impaired awareness and focal seizures with impaired awareness occurred in 16 patients, focal seizures with impaired consciousness and generalized tonic-clonic seizures – in 5 patients, and only generalized tonic-clonic seizures – in 4 patients. In 6 children, the seizures presented as status epilepticus.

Slow-wave activity was the most common finding in EEG (17 children). Focal epileptiform changes were found in 7 patients. In one child, there was a combination of slow-wave and focal epileptiform activity. Of these, one had focal motor epileptic status (ES)– epilepsia partialis continua and one presented with focal motor epileptic status with progression to bilateral motor epileptic status.

Involvement of cranial nerves was found in 27 children (50%), the most common being central facial nerve palsy. In one patient, it was combined with abducens nerve palsy, and in another – with hypoglossal nerve palsy. One child had isolated peripheral facial nerve palsy and another – oculomotor nerve palsy. Paralysis of the cranial nerves more commonly occurred in children with acute hemiparesis caused by cerebrovascular diseases (p=0.046, χ^2=3.98).

Headache (without raised intracranial pressure) (p=0.027, χ^2=4.88), impaired speech (p=0.006, χ^2=7.41), and hemicorporal sensory disturbances (p=0.04, χ^2=5.10) are more common in patients with cerebrovascular causes of acute hemiparesis. Hemicorporal disturbances are also more commonly found in the group over 10 years of age (p=0.001, χ^2=17.27).

The outcome of acute hemiparesis in the study group is presented in **Fig. 1**. Persistent hemiparesis is significantly more common in patients with ischemic stroke, while complete recovery is most often observed in patients with postictal paresis, lethal outcome – in hemorrhagic stroke and encephalitis. We have observed recurrent hemiparesis in patients with hemiplegic migraine and transient ischemic attack.

Cerebrovascular diseases were a more common cause in children with persistent or recurrent hemiparesis (p=0.022, χ^2=7.64). It is evident when dividing the cases into two groups: recovered and not recovered + dead (p=0.009, χ^2=16.86).

Thrombotic factors more commonly occurred in persistent or recurrent hemiparesis (p=0.002, χ^2=12.85). It is evident when classifying hemiparesis into two groups (p=0.012, χ^2=6.52).

Cerebrovascular abnormalities have been established more commonly in not recovered or dead patients with acute hemiparesis (p=0.001, χ^2=17.73).

Accompanying infections are more commonly found in patients with recovered hemiparesis (p=0.014, χ^2=5.83).

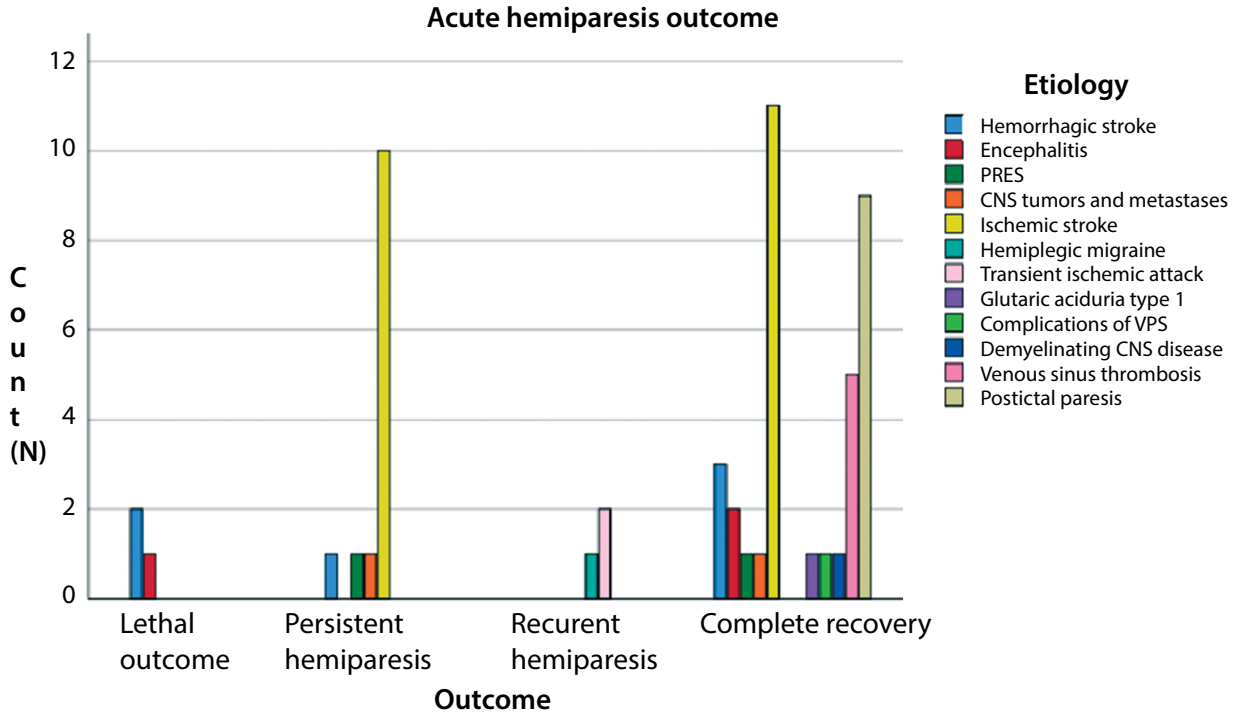

Figure 1. Outcome of acute hemiparesis.

DISCUSSION

There have been few publications in the literature since 2000 on the incidence, etiology, clinical presentation and outcome of acute hemiparesis in childhood, and the etiological causes of acute childhood hemiparesis are limited, making it difficult to compare with our results.

The incidence of hemiparesis (0.11%) among all our hospitalized patients is significantly lower than the one cited (0.44%) in a prospective observational study carried out in the department of pediatrics of a tertiary-care hospital of Western India from November 2010 to October 2012. [4] Most of the children in this study were between 1 and 5 years of age, while the majority of our patients are in the 1-10 age range.

Cerebrovascular diseases (63%) are a leading etiological factor for hemiparesis in our study. Like us, Chou et al.[5] found that cerebrovascular diseases (strokes) were the most common causes (66.7%) of acute hemiparesis, followed by tumors and trauma. Of the cerebrovascular diseases, ischemic stroke is the most prevalent etiological factor (39%), and it is most frequently localized in the basin of *a. cerebri media*, which correlates with the results of Chung and Wong[6] and Patil et al.[7].

In our study, ischemic stroke is more common than hemorrhagic stroke (39% vs. 11%), which is in line with the results of Chung and Wong[6] and Gold and Carter[8]. Intracranial hemorrhages are diagnosed in 11% of our cases, all of them in children with cerebrovascular abnormalities or undergoing chemotherapy for malignancies.

Chinnabhandar et al.[4] and Siddiqui et al.[9] report different etiological model of acute childhood hemiparesis, characteristic of developing countries. CNS infections (45.5%) are the leading cause in their studies and include purulent meningitis, brain abscess and TB meningitis, while our results show only encephalitis (HSV-2, varicella and autoimmune (NMDAP) encephalitis after CNS vasculitis) and represent only 5% of the cases of acute hemiparesis. The role of CNS infections is also leading in a retrospective review of 25 children with acute hemiplegia admitted to the Neurology Unit of the Department of Pediatrics of University of Calabar Teaching Hospital in Nigeria: encephalitis was the most common etiology of acute hemiparesis, followed by meningitis and sickle cell anemia.[10]

Similar to the research by Bathe and Ganesan[3] (20-30%), 37% of our patients have diagnoses of stroke-like conditions. Similar to our results are also the results of Mackay et al.[11], according to which 30-50% of children with acute neurological deficit mimic a stroke. Of the 143 children with suspected stroke in the study by Shellhaas et al.[2], stroke mimics are reported in 21% of them and include complicated migraine, conversion disorder, acute demyelinating encephalomyelitis, idiopathic intracranial hypertension, intracranial abscess, and metabolic stroke – cytochrome C oxidase deficiency. The range of stroke mimics in our patients includes postictal paresis, CNS infections, CNS tumors, and metastases, posterior reversible encephalopathy, demyelinating disease, VPS complication, and glutaric aciduria type 1.

The most common symptoms associated with hemiparesis in our study are damage of the cranial nerves and impaired consciousness, seen in 50% of patients. In chil-

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dren with cranial nerves involvement, central facial nerve palsy, ipsilateral to the side of the hemiparesis, predominates, similar to the data reported by Fritsch[12] – 63% and Chinnabhandar et al.[4] – 82%. In the study of Chou et al.[5] – paralysis of the cranial nerves is the most common manifestation associated with hemiparesis in 47% of the 57 patients under 18 years of age. Facial weakness as an element of the FAST complex (face, hand, speech, and time) is characteristic not only of cerebrovascular diseases, but also of infectious diseases of the CNS, which explains its high frequency. However, in the patients we studied, facial weakness was more common in cerebrovascular diseases. In confirmation of this, Kazutaka Uchida et al. have established facial weakness in more than 70% of strokes caused by intracranial hemorrhage.[13]

Impaired consciousness was observed in half of our patients (50%). This is similar to the reports by Chou et al.[5] – 42.1% and Fritsch[12] – 58%. In studies by Raghu Raman et al.[14], impaired consciousness, fever, vomiting, and headache are the most common symptoms associated with hemiparesis. The same is noted in the study by Shivalli et al.[15] These symptoms are probably related to raised intracranial pressure as the leading cause of acute hemiparesis among their patients are neuroinfections.

The incidence of seizures in our study group (47%) is higher than in the studies of Fritsch[12] – 32% and Chou et al. – 21%.[5] According to Abend et al., about one-fifth of children with acute AIS present with seizures. Seizures have been always accompanied by focal neurologic deficits.[16] 39.3% of patients with CVT experienced seizures, according to Ferro et al.[17] Seizures are characteristic of cerebrovascular diseases, but in our study, they are more common in children with acute hemiparesis caused by stroke mimics, which is probably due to the considerable number of patients with postictal hemiparesis.

Speech impairment was observed in 28% of our patients and in 32% in the study by Fritsch.[12] We observe this speech impairment among our patients as a combined manifestation with acute hemiparesis more commonly in cerebrovascular diseases. Our results are confirmed by the study of Sherman et al., who reported motor speech (37%) and language (31%) among 106 children (mean age 6.5 years, 73 AIS, 35 cerebral sinovenous thrombosis).[18]

Headache outside the manifestations of raised intracranial pressure was present in only 12% of the children we studied, but in the study of Taiwanese researchers Chou et al.[5], headache has a significantly higher frequency – 42.1%. The reason for this difference is the etiology of hemiparesis and the age characteristics of the patients studied. The cerebrovascular diseases causing acute hemiparesis are more commonly presented in our patients by headache outside the manifestations of raised intracranial pressure compared with stroke mimics. Hemicorporal sensory disturbances also prevail both in patients with cerebrovascular diseases and in the age group over 10 years old. Results similar to our results are provided by a retrospective review of 25 children with acute hemiplegia admitted to the Neurology Unit of the Department of Pediatrics of University of Calabar Teaching Hospital in Nigeria. It showed that prolonged seizures, speech defect, cranial nerve deficit, and loss of consciousness were the most common clinical findings among their patients but with a different etiological profile of acute hemiparesis.[10]

Accompanying diseases were found in 11 (20%) of our children. The same (20%) is the comorbidity rate in the study of Chinnabhandar et al.[4] Cardiovascular pathology (ASD, Ehlers Danlos and Marfan syndrome combined with mitral prolapse) together with leukemia were the most common comorbidities among our patients, and congenital heart disease in the aforementioned study. This confirms the role of cardiovascular pathology in the etiology of acute hemiparesis.

In a significant number of patients, we found an infection accompanying the hemiparesis (26%). They were in the age group under 10 years old and are more commonly related to stroke mimics. This is probably due to the large number of patients in our study with postictal hemiparesis after a febrile seizure in the course of an infection.

The reception of medication by 20% of the children with acute hemiparesis raises the question about its importance in its etiology (PRES, malignant diseases), requiring therapy with corticosteroids and cytostatics. The direct toxic effect of antineoplastic and immunosuppressive drugs on the cerebrovascular endothelium is well-known.[19]

Outcome after acute hemiplegia is highly variable and depends on the underlying cause. Complete resolution of acute hemiparesis was observed in 65% of the children we studied. In 23%, it remained permanent. In 6% (n=3), hemiparesis had a recurrent course. The same (6%) was the percentage of patients with a fatal outcome. All of them had malignancies. In a study by a Nigerian team of 25 children with acute hemiparesis caused by encephalitis, meningitis, and sickle cell anemia, only 16% of the patients recovered completely, 8% died, and 76% had varying degrees of weakness.[10] The probable cause for the difference in the outcome from the hemiparesis is a different etiological model of acute childhood hemiparesis, characteristic of developing countries and the organization of health care in them. Chou et al.[5] report significantly higher mortality rate – 21%, and in the study of Chung and Wong[6] it is 18%, but only among patients with hemiparesis caused by stroke. The significantly lower mortality rate in our study is probably related to the improved ability to diagnose and treat acute hemiparesis and stroke in recent decades. In the study of Chung and Wong[6], residual and recurrent paresis are presented as a percentage of surviving patients – 41% and 10%, respectively.

Statistical results show that cerebrovascular diseases as a whole and thrombophilia are more commonly related to a persistent or recurrent course of hemiparesis. Recurrent and residual hemipareses are more common in patients with ischemic stroke and proven thrombophilia. In 14 (67%) of our patients with acute hemiparesis and arterial ischemic stroke, we found thrombophilia. In 8 of them, thrombophilia was combined with a brain vessel abnor-

mality, and in one with cardiopathy. We diagnosed thrombophilia in 3 of 5 children with venous sinus thrombosis, but here too, it is combined with other risk factors – treatment with corticosteroids and L-asparginase and infection with dehydration. According to Bathe and Ganesan[3], prothrombotic factors are rarely the main cause of stroke, but increase the risk of stroke, and are more often associated with venous than arterial thrombosis. However, DeVeber et al. emphasize the role of thrombophilia in the recurrent arterial ischemic stroke in children.[20] Thrombophilia is found only in the cerebrovascular causes of acute hemiparesis in our patients, which confirms its role and importance for their genesis.

The vascular malformations we have established are more commonly associated with thrombophilia. The probable cause is a combination of risk factors in our patients leading to the occurrence of acute hemiparesis. Vascular abnormalities are more common in patients with non-recovered hemiparesis and lethal outcome. This also determines the poor prognosis of cerebrovascular malformations causing hemorrhages and accompanied by hemiparesis. Thorough evaluation for vascular anomalies which account for 40% to 90% of hemorrhagic stroke in children is critical.[21,22]

In contrast, the accompanying infection is more commonly associated with a favorable outcome in our patients, which is related to the high number of postictal pareses occurring in the course of infections and associated with favorable evolution.

CONCLUSIONS

Cerebrovascular disease with ischemic stroke is the most common cause of acute hemiparesis in childhood.

Impaired consciousness, central facial nerve palsy, and seizures accompany most frequently the acute hemiparesis (nearly 50% of cases).

Unlike adult patients, acute hemiparesis in children has many causes, which requires a large number of etiological studies. With a quick diagnosis and adequate treatment, recovery of function in children is more common than in adults – it is achieved in two-thirds of children.

Supplementary material

In embolic stroke and sinus thrombosis, low molecular weight heparin enoxaparin sodium (Clexane) was administered at a dose of 1 mg/kg subcutaneously with a subsequent antiplatelet agent – aspirin 3-5 mg/kg for 6 months or more, depending on the risk of recurrent stroke. In infectious vasculitis (varicella or herpes simplex) prednisolone 1 mg/kg for 7 days + acyclovir 10-15 mg/kg every 8 hours for a minimum of 14 days was used. The treatment of encephalitis was based on the etiology, but always included acyclovir 10-15 mg/kg every 8 hours until herpes simplex virus infection was ruled out. If confirmed, treatment continued for 21 days.

Autoimmune encephalitis was treated with pulse therapy with methylprednisolone 30 mg/kg daily, for 3 to 5 days, intravenous immunoglobulins 400 mg/kg daily for 5 days and/or plasmapheresis (every other day – 5-7 sessions in total), rituximab 375 mg/m^2 weekly i.v. for 4 weeks. The treatment of demyelinating diseases was with pulse therapy with methylprednisolone 1 g/kg daily for 3 to 5 days. In case of intracranial hemorrhage, the cause of the hemorrhage was treated, and the need for surgical treatment was assessed. Posterior reversible encephalopathy (PRES) required antihypertensive, anti-edema treatment, withdrawal of the drugs that caused it, or treatment of the condition that induced it.

Postictal paresis was controlled by seizure termination with diazepam 0.3 mg/kg, phenobarbital 5 mg/kg and anti-edema treatment – 10% mannitol at a dose of 10 ml/kg/24 hrs. In metabolic and mitochondrial diseases, dietary treatment and co-factors coenzyme Q, riboflavin (B2), L-carnitine were used. Malfunction and infection of the ventriculoperitoneal shunt (VPS) required surgical and antibiotic treatment – cephalosporins at a dose of 150 mg/kg.

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Случаи острого гемипареза в детском возрасте

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Резюме

Введение: Острый гемипарез – неотложное состояние различной этиологии с возможным летальным исходом.

Цель: Целью данного исследования было определение этиологии, клинических проявлений и прогноза острого гемипареза в детском возрасте.

Материалы и методы: Ретроспективное исследование 52 детей (28 мальчиков и 24 девочек) в возрасте от 4 месяцев до 16 лет 11 месяцев с острым гемипарезом, поступивших в педиатрическую клинику Университетской больницы Святого Георгия в период с 2013 по 2020 г. После клинического обследования. был использован современный диагностический и лечебный подход.

Результаты: Выявлены причины острого гемипареза: ишемический инсульт (21), постиктальный парез после приступа (9), геморрагический инсульт (6), тромбоз венозных синусов (5), энцефалит (3), транзиторные ишемические атаки (TIA) (2), синдром задней обратимой энцефалопатии (PRES) (2), гемиплегическая мигрень (1), демиелинизирующее заболевание ЦНС (1), осложнение вентрикулоперитонеального шунтирования (1), глутаровая ацидурия 1 типа (1), опухоль головного мозга (1) и метастазы в ЦНС (1).

Наиболее частыми проявлениями, связанными с острым гемипарезом, были нарушение сознания (27), поражение черепно-мозговых нервов – центральный (25) и периферический (1) паралич лицевого нерва, паралич отводящего (1), глазодвигательного (1), подъязычного (1) нерва, судороги (25), повышение внутричерепного давления (18), нарушения речи (15), признаки инфекции (14), головная боль (12), нарушения чувствительности полутела (9).

У 8 детей (14%) гемипарез был постоянным, у 2 рецидивирующим и у 7 (12%) летальным.

Заключение: В отличие от взрослых больных острый гемипарез у детей имеет множество причин, что требует проведения большого количества этиологических исследований. При быстрой диагностике и адекватном лечении восстановление функции у детей встречается чаще, чем у взрослых – оно достигается у двух третей детей.

Ключевые слова

цереброваскулярные заболевания, нейровизуализация, инсультоподобные состояния