



CLINICAL AND ANAMNESTIC RISK FACTORS FOR THE DEVELOPMENT OF SYMPTOMATIC EPILEPSY IN INFANTILE CEREBRAL PALSY

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Abstract

The high prevalence of infantile cerebral palsy (ICP) in industrialized countries has become an important problem of pediatric neurology. The study of cerebral palsy, its clinical and neurological aspects, the search for new methods of diagnosis and improvement of treatment results is carried out purposefully, with a priority focus on the wound detection of the disease, the frequency of risk and pathogenetic causes of symptomatic development. epilepsy (SE), which aggravates the disease; determination of the role of the epileptic process in the mental development and intellectual functions of patients with cerebral palsy.

Keywords: Cerebral palsy, childhood disability, pathological process, haptoglobin.

Relevance

It is appropriate to cite the recommendation of M.D. Hensleih "The prevention program will be unsuccessful as long as the cause of cerebral palsy is unknown." The most optimal results of preventive work can be obtained only with early identification of risk factors in order to prevent the birth of a sick child. But risk factors and morphofunctional disorders, which are the basis for the formation of epilepsy, have not been studied enough. According to publications, cerebral palsy is the result of adverse effects of a number of hereditary and perinatal factors [5, p. 1646-1650; 24, p. 35-36; 34, p. 18-19]. The question of the genesis of epilepsy in cerebral palsy has not yet been clarified, in particular, the risk factors for the development of SE have not been clearly defined. Namely, they make it possible to accurately predict the likelihood of developing epilepsy and, accordingly, to carry out early preventive treatment.





Material and research method

The study included 308 children aged 1-16 years. The diagnosis was made on the basis of the clinical classification of cerebral palsy by K.A. Semenova, which corresponds to the International Classification of Diseases ICD-10 / ICD-10 No. 7. We carried out a retrospective study: analysis of the medical history of all patients with cerebral palsy, questioning their mothers using special questionnaires, analysis of the influence of various prenatal and natal factors on the formation of SE in children in Uzbekistan. They took into account - the data of the anamnesis of the disease: the course of pregnancy, concomitant diseases, bad habits, history of childbirth, natal pathologies and full-term baby. Further, we identified prognostically significant factors for predicting the risk of developing epilepsy in patients with cerebral palsy.

Research results

Our studies have shown (Table 1) that a certain role in the onset of cerebral palsy is assigned to a violation of the normal course of pregnancy at various times.

Table 1. Violation of the timing of gestation and the risk of developing SE in cerebral palsy ($M \pm m$),%

Gestational age	Total	with solar	cell without	t	P
Full-term	161	19,9±3,1	80,1±3,1	13,73	<0,001
Premature	147	55,8±4,1	44,2±4,1	2,00	<0,05

There were more premature babies in the group with SE than in the group without SE. At the same time, prematurity as a whole in the group of patients with cerebral palsy dominates, consistent with the data of publications [51, C13-18; 84.21-25; 93.31-33]. Some epidemiological studies have shown the dependence of the development of cerebral palsy on the age of the mother: the largest number of women in labor were over 30 years old [108, pp. 18-22].

In our studies (Table 2), it turned out that early motherhood (under 18 years of age) plays a significantly greater role. Older women in labor, an independent risk factor for cerebral palsy, are not a significant risk factor for complications of its SE.



Table 2 Maternal age and risk of developing SE in cerebral palsy (M ± m),%

Mother's age, years	Total	with solar	cell without	t	P
before 18	22	63,6±10,3	36,4±10,3	1,87	>0,05
19-30	178	23,6±3,2	76,4±3,2	11,67	<0,001
30-39	95	52,6±5,1	47,4±5,1	0,72	>0,05
over 40	13	61,5±13,5	38,5±13,5	1,20	>0,05

We compared the significance of perinatal hereditary factors and found that chronic extragenital diseases of the mother (obesity, diabetes mellitus), maternal perinatal risk factors (medication during pregnancy, stress and psychological discomfort), as well as hereditary burden of epilepsy very often affect the development of epilepsy in children with cerebral palsy (Table 3).

Table 3 Intrauterine hazards, hereditary factors and the risk of developing SE in cerebral palsy (M ± m),%

Risk factors	Total	with solar	cell without	t	P
Hypertensive Disease	42	76,2±6,6	23,8±6,6	5,61	>0,001
Heart defects	158	67,7±3,7	32,3± 3,7	6,77	<0,001
Anemia	31	61,3±8,7	38,7±8,7	1,84	<0,05
Obesity	7	71,4±17,1	28,6±17,1	1,77	<0,05
Diabetes	84	64,3±5,2	35,7±5,2	3,89	<0,001
Taking medication	42	47,6±7,7	52,4±7,7	0,44	<0,05

Parental alcoholism	18	88,9±7,4	11,1±7,4	7,43	<0,001
Stress, psychological discomfort	109	63,3±4,6	36,7±4,6	4,09	<0,001
Physical injury during pregnancy	28	53,6±9,4	46,4±9,4	0,54	<0,05
Infectious lesions of the fetus with a virus in the blood plasma of antibodies (Toxoplasma gondii), etc.	75	56,0±5,7	44,0±5,7	1,49	<0,05
Maternal or paternal epilepsy	22	86,4±7,3	13,6±7,3	7,05	<0,001
Stillbirth in the family	8	62,5±17,1	37,5±17,1	1,03	<0,05



An important factor is the nature of the course of pregnancy (Table 4).

Table 4 The course of pregnancy and the risk of developing SE in cerebral palsy ($M \pm m$),%

Risk factor	Total	with solar	cell without	t	P
Indomitable vomiting	75	73,3±5,1	26,7±5,1	6,46	<0,001
Nephropathy	32	62,5±8,6	37,5±8,6	2,06	>0,05
Interruption threat	164	63,4±3,8	36,6±3,8	4,99	<0,001
Uterine bleeding, violation of the planntal to / o, present. placenta, its detachment	48	81,3±5,6	18,8±5,6	7,89	<0,001
Immunological incompatibility between mother and fetus (ABO- and Rhesus incompatibility)	28	67,9±8,8	32,1±8,8	2,88	>0,01

It was shown that in all cases of pregnancy complicated by preeclampsia, threats of termination of pregnancy and immunological incompatibility of the mother and fetus, children with cerebral palsy (SE) significantly prevailed (Table 5).

Table 5 Fetal damage in the natal and perinatal periods and the risk of developing SE in cerebral palsy ($M \pm m$),%

Risk factor	Total	with solar	cell without	t	P
Weakness of contractile activity	85	74,1±4,8	25,9±4,8	7,11	<0,001
Rapid labor	18	71,3±10,3	28,7±10,3	2,88	<0,05
Cesarean section	17	72,4±7,8	28,6±7,8	3,28	<0,001
Prolonged labor	81	85,3±3,6	14,7±3,6	14,09	<0,001
Long dry period	78	88,2±3,6	11,8±3,6	14,12	<0,001
Breech presentation	15	60,0±12,6	40,0±12,6	1,12	>0,05
Long period of standing of the head in the birth canal	77	88,3±3,7	11,7±3,7	14,64	<0,001
Instrumental obstetrics	48	87,5±4,8	12,5±4,8	11,05	<0,001
A history of febrile seizures in the child	125	83,2±3,3	16,8±3,3	14,23	<0,001
Concomitant diseases internally. organs of the child	72	81,9±4,5	18,1±4,5	10,03	<0,001



The overwhelming majority of factors complicating labor, cause the development of perinatal pathology, and in the future - the development of epilepsy. Among the identified intranatal pathologies, we most often noted the weakness of contractile activity during childbirth. Almost all women in labor (90%, 10% found it difficult to remember) underwent rhodostimulation with drugs that enhance contractions, synthesized artificial hormone oxytocin or its synthetic analogs - prostaglandins. According to V.E. Rodzinsky, at the present stage of obstetrics there has been a substitution of assistance in childbirth for aggressive intervention in the process of childbirth (Obstetric aggression as a reason for the decline in the quality of obstetrics, Moscow, 2004). From the action of oxytocin, prostaglandins and antiprogesterones, hypoxia of the child occurs in the intranatal period. The obstetrician is obliged to inform the woman in labor about possible complications in the child, since these drugs, causing spasm of the vessels of the uterus, reduce blood flow to the placenta and the fetus, which causes hypoxic-ischemic encephalopathy in the womb, which in the future may be complicated by cerebral palsy and epilepsy.

Conclusion

To reduce the risk of epilepsy and cerebral palsy, it is necessary to revise and take into account the preparations of oxytocin and its synthetic analogs - prostaglandins. In perinatal centers and maternity hospitals, immediately after childbirth, it is necessary to carry out screening - neuroimaging using neurosonography and increase the responsibility of obstetricians for the outcome of childbirth and the condition of the child. Perhaps this will serve as one of the methods of preventing cerebral palsy and the development of epilepsy.

Our results allow us to make an unambiguous conclusion that the main reasons for the development of SE in cerebral palsy are concomitant extragenital diseases and maternal intoxication during pregnancy, early motherhood plays an important role - the age of up to 18 years, as well as the totality of all prenatal and intranatal factors. hereditary predisposition and febrile seizures with their further transformation into SE. Therefore, patients with cerebral palsy, in whose history there are a lot of unfavorable hereditary and perinatal factors, should be prescribed GABA-ergic drugs as a prophylaxis, and early anticonvulsant therapy at the onset of epilepsy.





REFERENCES

1. Artykova M. A. Morphological changes in children with cerebral palsy with symptomatic epilepsy //European science review. – 2016. – №. 7-8. – С. 49-51. 2.
2. Abdurahmanovna A. M. Morphological and morphometric features of the brain in children with cerebral palsy complicated by epilepsy //European science review. – 2016. – №. 7-8. 3.
3. Артыкова, Мавлюда Абдурахмановна, And Нозима Абдурахимовна "Нейровизуализационные Характеристики Структурных Изменений Головного Мозга При Детском Церебральном Пара-Личе И Эпилепсии." Журнал неврологии и нейрохирургических исследований 1.1 (2020).
4. Артыкова, М. А. "Клинико-anamнестические факторы риска развития симптоматической эпилепсии при детском церебральном параличе." Медицинские новости 10 (265) (2016).
5. Артыкова, М. А. "Оценка провоспалительных цитокинов у детей с эпилепсией на фоне церебрального паралича." Медицинская иммунология 19.S (2017).
6. Артыкова, М. А., & Набиева, Н. А. (2017). Биохимический дисбаланс в сыворотке крови и ликворе при детском церебральном параличе. Журнал теоретической и клинической медицины , (1), 99-102.
7. Glass HC, Grinspan ZM, Shellhaas RA. Outcomes after acute symptomatic seizures in neonates.Semin Fetal Neonatal Med. 2018 Jun;23(3):218-222. doi: 10.1016/j.siny.2018.02.001.
8. Gulati S, Sondhi V. Cerebral Palsy: An Overview. Indian J Pediatr.2017 Nov 20.doi: 10.1007/s12098-017- 2475-1.
9. Hafström M, Källén K, Serenius F, Maršál K, Rehn E, Drake H, Ådén U, Farooqi A, Thorngren-Jerneck K, Strömberg B. Cerebral Palsy in Extremely Preterm Infants.Pediatrics. 2018 Jan;141(1). pii: e20171433. doi: 10.1542/peds.2017-1433.
10. Hirschberger RG, Kuban KCK, O'Shea TM, Joseph RM, Heeren T, Douglass LM, Stafstrom CE, Jara H, Frazier JA, Hirtz D, Rollins JV, Paneth N; ELGAN Study Investigators. Co-occurrence and Severity of Neurodevelopmental Burden (Cognitive Impairment, Cerebral Palsy, Autism Spectrum Disorder, and Epilepsy) at Age Ten Years in Children Born

