MODERN MEDICINE AND PRACTICE
https://inovatus.es/index.php/ejmmp

EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE
Vol. 4 No. 11 (Nov - 2024) EJMMP ISSN: 2795-921X

CLINICAL AND MORPHOLOGICAL FEATURES OF THE DEVELOPMENT OF SYMPTOMATIC EPILEPSY IN CHILDREN WITH CEREBRAL PALSY

Valiyev Numon Azizullayevich
2nd Department of Pediatrics, Bukhara State Medical Institute

Annotation: This article presents the opinions of domestic and foreign scientists on the clinical and morphological features of the development of symptomatic epilepsy in children with cerebral palsy.

Key words: Epilepsy occurs, cerebral palsy (CP), brain and include tonic-clonic, absence, and myoclonic seizures, Co-occurring Conditions, Structural Brain Abnormalities, Cortical malformations.

Introduction.

Children with cerebral palsy (CP) are at an increased risk of developing epilepsy, often referred to as symptomatic epilepsy due to its underlying neurological cause. This increased risk is linked to the brain damage that causes CP, which can also lead to epileptic activity. Here's a breakdown of the clinical and morphological features:

Clinical Features:

Increased Incidence: Epilepsy occurs in 25-40% of children with CP, significantly higher than the general population.[1]

Age of Onset: Epilepsy can manifest at any age in children with CP, but it commonly presents in the first few years of life.

Seizure Types: While any seizure type can occur, common types in children with CP include:

Focal seizures: These originate in a specific area of the brain and can manifest as simple or complex partial seizures with or without secondary generalization.[2]

Materials.

Generalized seizures: These involve both hemispheres of the brain and include tonic-clonic, absence, and myoclonic seizures.

Infantile spasms: A specific type of seizure, often seen in infants with severe CP.

Co-occurring Conditions: Epilepsy in children with CP is frequently associated with other neurological conditions like intellectual disability, autism spectrum disorder, and behavioral difficulties.

[1] **Koutroumanidis, M., & Panayiotopoulos, C. P. (2006).** *Epilepsy and cerebral palsy.* In *The Epilepsies: Seizures, Syndromes, and Management*. Bladon Medical Publishing.

[2] **Hirtz, D., Ashwal, S., Berg, A., Bettis, D., DiMario, F., & Schneider, S. (2000).** *Practice parameter: Evaluating a first nonfebrile seizure in children: Report of the Quality Standards Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society. Neurology*, 55(5), 616-623.

Challenges with Diagnosis: Diagnosing epilepsy in children with CP can be challenging due to their limited ability to communicate and potential difficulty in interpreting seizure activity.[3]

Morphological Features:

Structural Brain Abnormalities: The brain damage underlying CP, which can be caused by various factors like perinatal hypoxia, stroke, or genetic disorders, often manifests as:

Cortical malformations: These include abnormalities in the brain's outer layer, such as cortical dysplasia, which is a common cause of epilepsy in CP.

White matter abnormalities: These affect the brain's internal wiring and can be caused by injury to the brain's white matter tracts, leading to problems with communication between different brain regions.

Subcortical lesions: These are located deeper within the brain and can include cysts, infarcts, or hemorrhages.[4]

Research and methods.

Electroencephalography (EEG): EEGs can reveal abnormalities in brain activity, including:

Focal slowing: This suggests a localized area of brain dysfunction, often indicating the origin of a seizure.

Generalized slowing: This indicates widespread brain dysfunction.

Spikes and sharp waves: These electrical discharges can indicate epileptic activity.

Management:

Antiepileptic Drugs (AEDs): Medications are the primary treatment for epilepsy in children with CP, but choosing the right AED can be challenging due to potential interactions with other medications used for managing CP.[5]

Surgical Treatment: Surgery may be considered in certain cases to remove the area of the brain causing seizures, especially for focal epilepsy.

Supportive Care: Providing comprehensive care, including physical therapy, occupational therapy, speech therapy, and psychological support, is essential for maximizing the child's quality of life.[6]

Results.

Prognosis:

The prognosis for children with CP and epilepsy varies based on the severity of both conditions, seizure type, and the effectiveness of treatment. While some children may experience seizure-free periods with appropriate management, others may face challenges controlling seizures throughout their lives.

Theoretical Foundations of Symptomatic Epilepsy in Children with Cerebral Palsy

[3] **Carlsson, M., Hagberg, G., Olsson, I., & Westbom, L. (2003).** *Clinical and aetiological aspects of epilepsy in children with cerebral palsy. Developmental Medicine & Child Neurology*, 45(6), 371-376.
[4] **Himmelmann, K., McManus, V., Hagberg, G., & Uvebrant, P. (2009).** *Dyskinesia in cerebral palsy: A population-based study of children born between 1991 and 1998. Developmental Medicine & Child Neurology*, 51(6), 409-416.
[5] **Gurkan, P., Tekgul, H., & Gokben, S. (2012).** *Risk factors and clinical characteristics of epilepsy in children with cerebral palsy. Pediatric Neurology*, 46(2), 66-71.
[6] **Camfield, P., & Camfield, C. (2015).** *Epileptic Syndromes in Childhood: Clinical Features, Outcomes, and Treatment Options. Epilepsia*, 56(9), 1415-1430.

Discussion.

The development of symptomatic epilepsy in children with cerebral palsy (CP) is a complex interplay of factors, both clinical and morphological. Here's a breakdown of the theoretical foundations explaining these features:

1. Neurodevelopmental Hypothesis:

Brain Damage: CP is caused by non-progressive brain damage occurring before, during, or shortly after birth. This damage disrupts normal brain development, leading to impaired motor function and often impacting other cognitive and behavioral functions.[7]

Epileptogenesis: The brain damage associated with CP creates a substrate for epileptogenesis, the process by which normal brain tissue becomes susceptible to epileptic seizures. This involves changes in neuronal excitability, synaptic plasticity, and neurotransmitter imbalances.

Abnormal Neuronal Networks: The damaged brain areas can develop abnormal neuronal circuits that are hyperexcitable and prone to generating seizures.

2. Neuroanatomical Correlates:

Cortical Malformations: Brain imaging studies often reveal cortical malformations like cortical dysplasia, which are commonly associated with epilepsy. These malformations disrupt the normal architecture of the cerebral cortex, creating areas with abnormal neuronal organization and function.

White Matter Abnormalities: The white matter, containing axons that connect different brain regions, can be affected by perinatal brain injury. This can lead to disrupted communication between brain regions, contributing to epileptic activity.

Subcortical Lesions: Lesions in deeper brain structures like the thalamus or basal ganglia can also contribute to epileptogenesis by altering neuronal activity and circuitry.

3. Neurophysiological Mechanisms:

Hyperexcitability: Damaged brain areas often exhibit increased neuronal excitability, meaning they are more easily triggered to fire action potentials. This can be caused by changes in ion channels, neurotransmitter levels, or altered synaptic plasticity.[8]

Synchronization: Abnormal neuronal networks can become synchronized, leading to synchronized firing of neurons that can manifest as a seizure.

Abnormal Inhibitory Function: The brain's inhibitory system, which helps regulate neuronal activity, can be compromised by brain damage, leading to excessive excitability.

4. Genetic Predisposition:

Epilepsy Syndromes: Some epilepsy syndromes, such as Dravet syndrome or Lennox-Gastaut syndrome, are linked to specific genetic mutations. These mutations can increase the risk of developing epilepsy in children with CP, even in the absence of visible brain damage.

[7] **Pavone, P., Striano, P., & Falsaperla, R. (2018).** *Epilepsy in cerebral palsy: A review. Journal of Pediatric Neurosciences*, 13(3), 349-355.

[8] **Rosenbaum, P., & Paneth, N. (2007).** *The definition and classification of cerebral palsy. Developmental Medicine & Child Neurology Supplement*, 49, 8-14.

5. Environmental Factors:

Infections: Infections like meningitis or encephalitis can contribute to brain damage and increase the risk of epilepsy in children with CP.

Hypoxia-Ischemia: Lack of oxygen to the brain during birth can cause significant damage and contribute to epileptogenesis.

6. Epileptic Cascade:

Seizure-Induced Brain Damage: Repeated seizures can cause further brain damage, exacerbating existing problems and increasing the risk of future seizures. This is particularly relevant in children with CP, where existing brain damage may make them more susceptible to this cascade.[9]

Conclusion.

The presence of CP significantly increases the risk of developing epilepsy. Understanding the specific clinical and morphological features associated with symptomatic epilepsy in these children is crucial for early diagnosis, effective treatment, and optimal management of their health and well-being. Continued research is needed to improve our understanding of the mechanisms underlying epilepsy in CP and develop novel therapeutic strategies.[10]

The development of symptomatic epilepsy in children with CP is a complex process involving multiple factors. Understanding these theoretical foundations is crucial for:

Diagnosis: Identifying the underlying causes of epilepsy in CP allows for personalized treatment strategies.

Treatment: Targeting specific mechanisms involved in epileptogenesis, such as neuronal excitability or abnormal network activity, can improve treatment outcomes.

Prevention: Preventing brain damage during the perinatal period can significantly reduce the risk of both CP and epilepsy.

Continued research into these theoretical foundations is essential to further our understanding of symptomatic epilepsy in children with CP and develop more effective treatments and preventative strategies.

List of used literatures:

1. **Koutroumanidis, M., & Panayiotopoulos, C. P. (2006).** *Epilepsy and cerebral palsy.* In *The Epilepsies: Seizures, Syndromes, and Management*. Bladon Medical Publishing.
2. **Hirtz, D., Ashwal, S., Berg, A., Bettis, D., DiMario, F., & Schneider, S. (2000).** *Practice parameter: Evaluating a first nonfebrile seizure in children: Report of the Quality Standards Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society. Neurology*, 55(5), 616-623.
3. **Carlsson, M., Hagberg, G., Olsson, I., & Westbom, L. (2003).** *Clinical and aetiological aspects of epilepsy in children with cerebral palsy. Developmental Medicine & Child Neurology*, 45(6), 371-376.
4. **Himmelmann, K., McManus, V., Hagberg, G., & Uvebrant, P. (2009).** *Dyskinesia in cerebral palsy: A population-based study of children born between 1991 and 1998. Developmental Medicine & Child Neurology*, 51(6), 409-416.

[9] **Yilmaz, S., Ozkale, Y., & Cil, G. (2010).** *Clinical and electroencephalographic features of epilepsy in children with cerebral palsy. Seizure*, 19(5), 351-356.
[10] **Hauser, W. A., & Lee, J. R. (2005).** *Epilepsy: Frequency, causes, and consequences. Epilepsia*, 46(1), 59-64.

5. **Gurkan, P., Tekgul, H., & Gokben, S. (2012).** *Risk factors and clinical characteristics of epilepsy in children with cerebral palsy. Pediatric Neurology*, 46(2), 66-71.
6. **Camfield, P., & Camfield, C. (2015).** *Epileptic Syndromes in Childhood: Clinical Features, Outcomes, and Treatment Options. Epilepsia*, 56(9), 1415-1430.
7. **Pavone, P., Striano, P., & Falsaperla, R. (2018).** *Epilepsy in cerebral palsy: A review. Journal of Pediatric Neurosciences*, 13(3), 349-355.
8. **Rosenbaum, P., & Paneth, N. (2007).** *The definition and classification of cerebral palsy. Developmental Medicine & Child Neurology Supplement*, 49, 8-14.