HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

CLINICAL AND ELECTROENCEPHALOGRAPHIC FEATURES OF COMORBID MIGRAINE AND EPILEPSY IN CHILDREN

Safarov Saydullo Ubaydullo o'g'li
Pediatric Neurologist at the Kimyo Universitety Hospital in Tashkent

Isroilkhonov Azizkhon Inomjon ugli
Tashkent State Medical University Termez Branch

Israilxanova Mahliyoxon Inomxon qizi
Tashkent State Medical University

Doi: https://doi.org/10.5281/zenodo.22171104

Relevance. Migraine and epilepsy are among the most common chronic paroxysmal neurological disorders in childhood, characterized by episodic disturbances of brain function. In pediatric practice, comorbidity between these conditions is significantly more common than epidemiological data for individual disorders would suggest, representing a complex clinical and pathophysiological problem. Numerous studies in recent years support the hypothesis of common mechanisms of cerebral hyperexcitability, neurotransmitter imbalances, and genetic predisposition underlying the development of both disorders. However, despite growing interest in this issue, clinical and electroencephalographic markers of comorbidity in childhood remain poorly understood, and diagnostic criteria require further development and refinement.

Purpose of the study. To present a comprehensive review of current data on the clinical and electroencephalographic features of the comorbidity of migraine and epilepsy in children, and to systematize existing knowledge on the epidemiology, pathophysiological mechanisms, and diagnostic approaches to the management of this category of patients.

Materials and methods.A critical analysis of the literature, presented in peer-reviewed sources and databases such as Scopus, PubMed, Web of Science, and others, was conducted, covering the period primarily from 2000 to 2024. The focus was on original clinical studies, systematic reviews, meta-analyses, and clinical guidelines related to the diagnosis and management of children with comorbid migraine and epilepsy. The search was conducted using the following keywords: pediatric migraine, childhood epilepsy, comorbidity, electroencephalogram, ictal epileptic headache, and interictal epileptiform discharges.

Epidemiological aspects of comorbidity. The incidence of migraine and epilepsy comorbidity in children varies significantly depending on the population studied and the research methodology. The overall prevalence of headache among children with epilepsy, according to various authors, reaches 8-15%. However, in specific patient groups, these figures are significantly higher: in children with benign epilepsy with centrotemporal spikes (BETS), the incidence of migraine reaches 63%, while in absence epilepsy, it is 33%. Of particular note is the fact that migraine with aura has the strongest association with epileptic seizures compared to other forms of primary headache. It was found that in the group of children with migraine and aura, the frequency of occurrence of epileptiform discharges on the EEG reaches 43.5%, which is a reliably significant difference compared to children with migraine without aura and the control group ($p \leq 0.01$).

Clinical features of comorbid course. The comorbidity of migraine and epilepsy in children is characterized by a "clinical continuum" phenomenon, characterized by a complex interaction of

HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

paroxysmal events. Epileptic seizures can trigger the development of migraine headaches in the postictal period, while migraine with aura can present with symptoms clinically resembling focal epileptic seizures, particularly when the epileptic focus is localized in the occipital regions of the cortex. A key clinical phenomenon illustrating the close relationship between these two conditions is the rare but clinically significant form of "ictal epileptic headache," in which headache is the sole or dominant symptom of an epileptic seizure. This condition presents significant diagnostic challenges, as it requires differential diagnosis using long-term video-EEG monitoring to confirm the epileptic nature of the paroxysm and initiate appropriate antiepileptic therapy. It has been shown that occipital epileptic seizures can be misdiagnosed as migraine with aura, leading to unnecessary numerous radiological examinations and delays in pathogenetically based therapy. A high degree of disability in children is also an important clinical marker of comorbidity. According to the results of a multicenter study, children with migraine and concomitant epileptiform changes on the EEG had significantly higher PedMIDAS (Pediatric Migraine Disability Assessment) scores compared to patients with a normal electroencephalographic pattern ($p = 0.001$).

Electroencephalographic features. Interictal epileptiform discharges in childhood migraine are a key diagnostic marker of comorbidity, reflecting the presence of common pathophysiological mechanisms. The frequency of detection of specific EEG changes in the general population of children with migraine ranges, according to various studies, from 11.7% to 19.2%. Moreover, pathological patterns are most frequently recorded in the occipital, temporo-occipital, and temporo-parieto-occipital leads as spike-wave complexes with a characteristic feature – suppression of pathological activity upon opening the eyes. This electroencephalographic phenomenon was described in 14 children with classic migraine, 13 of whom had epileptic seizures, confirming the close association of occipital epileptiform activity with migraine with aura.

Of particular importance in the diagnosis of comorbidity are the results of functional tests, in particular photostimulation and hyperventilation. A study that included 52 children aged 4-14 years with migraine showed that photostimulation in children aged 4-9 years resulted in a significant increase in the number of paroxysmal changes on the EEG ($p=0.005$). Significant gender differences were also revealed: theta-delta bursts in response to functional tests were more typical for boys ($p=0.03$), while interictal epileptiform discharges predominated in girls ($p=0.046$). In addition to epileptiform discharges, diffuse slowing of the main activity (13.5%) and theta-delta wave bursts (34.6%) were recorded on the EEG in children with migraine. The pattern of “14 and 6/second positive spikes” deserves special attention; according to earlier studies, it was found in 40.7% of pathological recordings in children with headaches and was considered as a marker of autonomic hyperreactivity; however, this pattern has not been sufficiently studied in modern studies.

Pathophysiological mechanisms of comorbidity. The comorbidity of migraine and epilepsy is based on impaired cortical hyperexcitability, caused by an imbalance between excitatory and inhibitory processes in neural networks. Genetic studies indicate the important role of mutations in genes encoding ion channels, particularly CACNA1A, ATP1A2, and SCN1A, which are associated with various forms of epilepsy and familial hemiplegic migraine. Similar pathophysiological mechanisms explain the effectiveness of certain antiepileptic drugs (valproate, topiramate) and non-pharmacological methods (ketogenic diet, vagal stimulation) for both the treatment of epilepsy and the prevention of migraine in children. It is believed that both conditions are based on a genetically determined decrease in the excitability threshold of neurons, which leads to the occurrence of both epileptic discharges and waves of cortical depression characteristic of migraine aura.

HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

Diagnostic approaches and differential diagnostics. Diagnosing comorbid conditions in pediatric practice is a complex task requiring a multidisciplinary approach and the use of a range of instrumental methods. The basis for diagnosis is a thorough analysis of the clinical picture, with an emphasis on the characteristics of paroxysmal events, their temporal relationships, and the presence of trigger factors. An EEG examination, including functional tests (photostimulation, hyperventilation) and, if necessary, long-term video-EEG monitoring, plays a key role in identifying electroencephalographic markers of comorbidity. According to current guidelines, EEG is indicated for children with migraine with aura, as well as in cases of suspected ictal epileptic headache or in the presence of atypical clinical manifestations. Considering the high frequency of detection of epileptiform discharges in this group of patients, early EEG allows for the timely identification of electro-clinical correlations, avoiding overdiagnosis and unjustified radiological studies, and prescribing pathogenetically justified therapy.

Conclusion. The comorbidity of migraine and epilepsy in children is a clinically significant problem characterized by a high incidence, diagnostic complexity, and a significant impact on patients' quality of life. The clinical presentation is diverse, ranging from the classic combination of migraine with aura and epileptic seizures to rare forms of ictal epileptic headache requiring video-EEG monitoring. EEG is a key tool for identifying comorbidity markers, with the most specific signs being interictal epileptiform discharges in the occipital leads, suppressed upon eye opening, and activation of paroxysmal activity during functional testing, especially in children aged 4-9 years. Early detection of electroclinical correlations enables timely, pathogenetically based therapy (antiepileptic drugs with an anti-migraine effect), improving the prognosis, and reducing the incidence of disability. Promising areas for further research include the study of molecular genetic markers of predisposition to comorbidity, the development of unified diagnostic criteria for the early identification of patients at risk, and the conduct of prospective randomized clinical trials to optimize therapeutic approaches in this category of patients.

Bibliography

1. Momen, A. A., Jelodar, G., & Azizimalamiri, R. (2024). Migraine and Epilepsy in Children: A Narrative Review of Comorbidity and Similar Treatment Options. Iranian Journal of Child Neurology, *18*(3), 9-20.
2. Sowell, M. K., & Youssef, P. E. (2016). The Comorbidity of Migraine and Epilepsy in Children and Adolescents. Seminars in Pediatric Neurology, *23*(1), 83-91.
3. Jancic, J., Djuric, V., Hencic, B., van den Anker, J., & Samardzic, J. (2018). Comorbidity of Migraine and Epilepsy in Pediatrics: A Review. Journal of Child Neurology, *33*(12), 801-808.
4. Parisi, P., et al. (2012). Benign epilepsy of childhood with centro-temporal spikes (BECTS) versus migraine: a neuropsychological assessment. Child's Nervous System, *28*(12), 2129-2135. [PubMed]
5. Cianchetti, C., et al. (2013). Pure epileptic headache and related manifestations: a video-EEG report and discussion of terminology. Epileptic Disorders, *15*(1), 84-92. [PubMed]

HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

6. Vollono, C., Mariotti, P., Losurdo, A., et al. (2015). Headache Following Occipital Brain Lesion: A Case of Migraine Triggered by Occipital Spikes? Clinical EEG and Neuroscience, *46*(4), 364-369.

7. Barca, S., Belcastro, V., Striano, P., & Parisi, P. La comorbidità cefalea-epilessia nel bambino: revisione critica dei dati della letteratura [Headache and epilepsy comorbidity in children: a critical review of the literature]. Medico e Bambino. [Italian Journal]

8. Kelley, S. A., & Hartman, A. L. (2012). Comorbidity of migraine in children presenting with epilepsy to a tertiary care center. Neurology, *79*(5), 468-473.

9. Wirrell, E. C., et al. (2006). Do children with benign rolandic epilepsy have a higher prevalence of migraine than those with other partial epilepsies or nonepilepsy controls? Epilepsia, *47*(10), 1674-1681.

10. Bellini, B., et al. (2013). Headache and comorbidity in children and adolescents. The Journal of Headache and Pain, *14*(1), 79.

11. Winawer, M. R., & Ottman, R. (2013). Evidence for a shared genetic susceptibility to migraine and epilepsy. Epilepsia, *54*(2), 288-295.

12. Clarke, T., et al. (2009). Evidence of shared genetic risk factors for migraine and rolandic epilepsy. Epilepsia, *50*(11), 2428-2433.

13. Addis, L., et al. (2014). Evidence for linkage of migraine in Rolandic epilepsy to known 1q23 FHM2 and novel 17q22 genetic loci. Genes, Brain and Behavior, *13*(3), 333-340.

14. Costa, C., et al. (2014). A novel ATP1A2 gene mutation in familial hemiplegic migraine and epilepsy. Cephalalgia, *34*(1), 68-72.