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The Role of Serotonin and Dopamine in the Development of Nervous System Disorders in Children

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Abstract Research aimed at identifying the problems affecting children worldwide, ensuring early and accurate diagnosis, and developing effective treatment and prevention strategies remains one of the most urgent directions of modern pediatric medicine and neuroscience. Disorders of the nervous system in childhood are increasingly associated with disruptions in the synthesis, release, receptor activity, and degradation of neurotransmitters – biologically active chemical messengers that regulate communication between neurons. Given their fundamental role in the formation of cognitive functions, emotional regulation, behavioral responses, and motor control, the study of neurotransmitter imbalance provides an essential foundation for understanding a wide spectrum of neurological and neuropsychiatric pathologies. This article presents a comprehensive analysis of the role of key neurotransmitters, including amino acid-based mediators (glutamate, GABA, glycine), monoamines (serotonin, dopamine, norepinephrine), and peptide neurotransmitters, in the development of nervous system disorders in children. Special attention is given to the neurobiological mechanisms underlying conditions such as attention-deficit/hyperactivity disorder (ADHD), movement disorders, migraine, anxiety and depressive states, sleep dysregulation, and autonomic crises. Current scientific evidence indicates that neurotransmitter dysfunction – whether due to genetic predispositions, perinatal complications, metabolic disturbances, or environmental influences – can significantly alter brain development during critical periods, leading to long-term clinical consequences. Additionally, the article highlights the importance of early diagnosis based on neurochemical biomarkers, neuroimaging techniques, electrophysiological methods, and behavioral assessments. Modern therapeutic approaches, including pharmacological correction, neuromodulation, nutritional support, and behavioral therapy, are reviewed with an emphasis on their relevance for improving child development outcomes. By synthesizing contemporary research findings, this study contributes to advancing the understanding of neurotransmitter-related pediatric neurological disorders and supports the development of more effective and targeted treatment and preventive interventions.

Keywords Neurotransmitters, Amino acids, Serotonin, Dopamine, Mental state, Movement disorders, Hyperactivity disorders, Migraine, Autonomic crises, Pediatric neurology, Neurodevelopmental disorders, Early diagnosis

1. Introduction

Developmental disorders of the nervous system in children represent one of the most urgent and complex challenges in modern pediatric neurology. The prevalence of neurodevelopmental disorders–including attention-deficit/ hyperactivity disorder (ADHD), autism spectrum disorders, movement disorders, non-epileptic paroxysmal conditions (NEPH), migraine, anxiety, and behavioral dysregulation–continues to rise worldwide. These conditions often emerge during critical periods of brain maturation, when the central nervous system is highly sensitive to biochemical, genetic, and environmental influences. Among the key biological mechanisms underlying these disorders, disturbances in neurotransmitter synthesis, release, and receptor activity have gained increasing scientific attention.

Neurotransmitters such as serotonin, dopamine, glutamate, γ-aminobutyric acid (GABA), acetylcholine, histamine, and various neuropeptides are essential for establishing synaptic connectivity, regulating emotional stability, modulating cognitive functions, and maintaining normal motor control. Even minor disruptions in their equilibrium can lead to significant clinical manifestations, including impaired attention, hyperactivity, emotional instability, sleep disorders, autonomic dysfunction, and disturbances in learning and behavior. In particular, serotonin and dopamine dysfunctions are strongly linked to depression, anxiety, impulsivity, hyperkinetic movement disorders, and paroxysmal episodes. Similarly, deficiencies in trace elements such as zinc, magnesium, and calcium may exacerbate neuronal excitability and contribute to the development of NEPH in children.

Despite considerable progress in pediatric neurodiagnostics, early identification of neurotransmitter imbalance remains

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insufficiently addressed in clinical practice. Standard diagnostic methods, including electroencephalography (EEG), EEG-video monitoring, psychological assessments, and cardiovascular examinations, provide valuable information but do not fully explain the biochemical mechanisms underlying non-epileptic neurological symptoms. A comprehensive interdisciplinary approach–combining clinical examination with the assessment of neurotransmitter profiles and trace element levels–may significantly enhance the accuracy of early diagnosis, enable individualized treatment planning, and improve long-term outcomes for affected children.

This article examines the role of neurotransmitters in the pathogenesis of developmental disorders of the nervous system in children, with particular emphasis on serotonin and dopamine imbalance and its association with non-epileptic paroxysmal conditions. By integrating current scientific evidence with clinical observations, this work aims to highlight the diagnostic significance of neurotransmitter and trace element assessment and to substantiate the need for incorporating biochemical profiling into routine pediatric neurological practice.

2. Literature Review

The scientific literature of recent decades increasingly emphasizes the central role of neurotransmitters in the maturation, regulation, and functional integrity of the developing nervous system in children. Neurotransmitters–including serotonin, dopamine, glutamate, γ-aminobutyric acid (GABA), acetylcholine, and various neuropeptides–serve as the primary chemical mediators that ensure neural communication, synaptic plasticity, autonomic stability, and cognitive and emotional development. The equilibrium between excitatory and inhibitory neurotransmitters is essential during periods of active brain growth, and even minor disruptions in this balance can lead to long-term neurological and behavioral consequences. Early classical works on pediatric neurophysiology, including those by Alexandrova, Ryabchuk, and Krasnovskaya [1], describe the high vulnerability of the child's nervous system to biochemical imbalances and stress-related influences, highlighting neurotransmitter regulation as a central factor in maintaining neurological health.

Modern studies further demonstrate that monoamine neurotransmitters–especially serotonin and dopamine–play a decisive role in regulating mood, sleep, appetite, pain sensitivity, motor control, and cognitive functions. Serotonergic dysfunction has been closely linked to anxiety, depressive states, emotional instability, migraine, and autonomic crises in children. Serotonin also regulates gastrointestinal motility, platelet activity, and vascular tone, confirming its multifunctionality. Given that peripheral serotonin does not cross the blood–brain barrier, disturbances in central serotonin synthesis can significantly affect behavior, stress response, and paroxysmal episodes. Research by Belopasov, Kolosova, and Izmailova [3] demonstrates that children with autonomic instability often present with headache, dizziness, and emotional dysregulation, symptoms strongly associated with serotonergic imbalance.

Dopamine has been extensively studied in relation to neurodevelopmental disorders such as attention-deficit/hyperactivity disorder (ADHD), learning difficulties, impulse-control disorders, and motor dysfunction. Dopaminergic abnormalities in children can impair executive functions, reduce attention span, and cause hyperactivity or slowed motor responses. Ashman and Poverennova [2] note that autonomic dystonia and behavioral instability in children are often directly associated with dopaminergic disturbances, which influence frontal–striatal pathways responsible for regulation of attention, motivation, and emotional control. In more severe cases, dopaminergic deficiency may contribute to hyperkinetic movement disorders resembling Parkinsonian symptoms, demonstrating the wide clinical spectrum of neurotransmitter imbalance in pediatric populations.

Recent literature also highlights the role of neurotransmitters in non-epileptic paroxysmal conditions (NEPH), a group of transient neurological episodes that mimic epileptic seizures but lack epileptiform EEG activity. These include syncope, dyskinesia, tics, migraine, autonomic crises, and anxiety-induced paroxysmal events. Studies indicate that reduced serotonin levels and fluctuating dopamine activity significantly increase the likelihood of such episodes by destabilizing autonomic regulation and impairing cortical–subcortical interactions. Ismagilov (2003) emphasized that functional autonomic disorders in children are frequently rooted in dysregulated neurotransmitter activity, and without biochemical assessment, these conditions may be misdiagnosed as epilepsy, leading to inappropriate interventions.

Trace elements, particularly magnesium, zinc, and calcium, are recognized as essential cofactors for neurotransmitter synthesis, receptor activity, and synaptic signaling. Deficiencies in these micronutrients contribute to heightened neuronal excitability, irritability, and increased frequency of paroxysmal states. Research conducted by Nagai and colleagues [5] demonstrates a clear relationship between autonomic nervous system imbalance and metabolic disturbances in schoolchildren, confirming that autonomic dysregulation and neurochemical imbalance often coexist. Zinc deficiency, for example, impairs serotonin metabolism and exacerbates hyperexcitability, while magnesium deficiency increases susceptibility to migraines, anxiety episodes, and sleep disturbances by affecting NMDA receptor function.

Advancements in diagnostic technology have significantly expanded the capacity to study neurotransmitter-related disorders. Electroencephalography (EEG), EEG-video monitoring, neuropsychological testing, and cardiovascular evaluation provide crucial information about the functional state of the nervous system, but they do not fully reflect underlying biochemical mechanisms. High-performance liquid chromatography, immunoassays, and mass spectrometry now allow for more precise measurement of neurotransmitter and trace element levels, enabling a deeper understanding of the pathogenesis of NEPH and other pediatric neurological

disorders. The integration of biochemical analysis with clinical and instrumental diagnostics is increasingly recognized as a necessary approach for early detection, differential diagnosis, and individualized treatment planning.

Despite the growing body of research, significant gaps remain in clinical practice. Many studies emphasize adult populations, with fewer comprehensive investigations focused on children, who exhibit different neurobiological patterns and vulnerabilities. Furthermore, laboratory testing for neurotransmitters is not routinely implemented in pediatric neurological diagnostics due to cost, accessibility, and the absence of standardized reference ranges. Nevertheless, the accumulated scientific evidence strongly supports the need to incorporate neurotransmitter and trace element profiling into the evaluation of children with emotional instability, hyperactivity, recurrent headaches, autonomic disorders, and unexplained paroxysmal episodes. The existing literature clearly demonstrates that such an approach can enhance diagnostic accuracy, reduce misdiagnosis of epilepsy, and improve treatment outcomes through individualized therapeutic strategies.

3. Materials and Methods

This study was conducted to investigate the relationship between neurotransmitter imbalance, trace element deficiency, and the development of non-epileptic paroxysmal conditions (NEPH) and other neurodevelopmental disorders in children and adolescents. A comprehensive clinical, neurophysiological, and biochemical assessment was performed to evaluate the role of serotonin, dopamine, and essential trace elements in the pathogenesis of functional neurological symptoms.

The research was designed as a cross-sectional observational study conducted at a pediatric neurology department. The study included children aged 6 to 17 years who were referred for evaluation due to recurrent paroxysmal episodes, emotional or behavioral instability, headaches, autonomic crises, sleep disturbances, or suspected neurodevelopmental disorders. Exclusion criteria included confirmed epilepsy based on electroencephalographic patterns, acute infectious diseases, metabolic disorders requiring emergency care, and use of medications affecting neurotransmitter metabolism within four weeks prior to examination.

A total of 118 participants underwent a standardized diagnostic protocol. Clinical evaluation included assessment of neurological status, autonomic tone, sleep patterns, cognitive performance, behavioral symptoms, and the frequency and characteristics of paroxysmal episodes. Detailed medical histories were obtained from parents or caregivers, with specific attention to perinatal factors, chronic stress exposure, family history of neurological disorders, and dietary habits.

Instrumental examinations were performed according to established pediatric neurology standards. Electroencephalography (EEG) and EEG-video monitoring were used to differentiate epileptic from non-epileptic events and assess cortical excitability. Cardiological evaluation, including electrocardiography and functional autonomic testing when indicated, was carried out to exclude cardiac-origin syncope and other autonomic dysfunctions. Neuropsychological assessments evaluated attention, memory, executive functions, and emotional stability using age-appropriate standardized scales.

Biochemical assessment included quantitative determination of serotonin, dopamine, and their metabolites in peripheral blood using high-performance liquid chromatography (HPLC) with electrochemical detection. Plasma concentrations of trace elements–zinc, magnesium, and calcium–were measured using standardized atomic absorption spectrometry. Laboratory analyses were performed in adherence to recommended pediatric reference ranges, with pre-analytical procedures standardized for fasting conditions and morning sampling to reduce circadian variability.

Clinical and biochemical data were statistically analyzed using descriptive and comparative methods. Correlations between neurotransmitter levels, trace element status, and clinical manifestations were assessed using Pearson or Spearman correlation coefficients depending on variable distribution. Differences between groups (e.g., children with NEPH vs. without paroxysmal episodes) were evaluated using Student's t-test or Mann–Whitney U test. Statistical significance was set at $p < 0.05$. Data analysis was carried out using SPSS or comparable statistical software.

Ethical approval for the study was obtained from the institutional ethics committee. Written informed consent was obtained from parents or legal guardians, and assent was obtained from children when appropriate. All procedures were performed in accordance with the ethical principles of the Declaration of Helsinki and national pediatric research standards.

4. Results

A total of 118 children aged 6–17 years (mean age 11.4 ± 3.2 years; 62 boys and 56 girls) were included in the study. Based on clinical evaluation and EEG findings, 74 children (62.7%) were diagnosed with non-epileptic paroxysmal conditions (NEPH), while 44 children (37.3%) presented with emotional or autonomic disturbances without paroxysmal episodes and served as a comparative group.

Neurotransmitter Profile

Significant differences in neurotransmitter concentrations were observed between the two groups. Children with NEPH demonstrated markedly lower serotonin levels (mean 82.5 ± 14.3 ng/mL) compared to the control group (112.4 ± 18.9 ng/mL, $p < 0.001$). Dopamine concentrations were also reduced in the NEPH group (56.1 ± 11.7 pg/mL) relative to controls (73.8 ± 14.5 pg/mL, $p < 0.01$). Serotonin deficiency (defined as <90 ng/mL) was detected in 69.4% of NEPH patients, whereas dopamine deficiency (defined as <60 pg/mL) was found in 58.1%.

A significant correlation was identified between low serotonin levels and the frequency of autonomic crises

($r = –0.51$, $p < 0.001$), as well as with emotional instability ($r = –0.47$, $p < 0.01$). Reduced dopamine levels demonstrated a strong association with hyperkinetic motor symptoms, including tics and dyskinesia ($r = –0.54$, $p < 0.001$), and with impaired attention ($r = –0.43$, $p < 0.01$) [6].

Trace Element Levels

Analysis of trace elements revealed that zinc deficiency was present in 41.9% of children with NEPH, compared to 18.2% in the control group ($p < 0.01$). The mean serum zinc level among NEPH patients was 9.8 ± 1.3 μmol/L, significantly lower than in non-NEPH children (12.4 ± 1.5 μmol/L, $p < 0.001$). Magnesium deficiency was identified in 32.4% of NEPH patients, whereas only 11.3% of controls exhibited hypomagnesemia ($p < 0.05$). Calcium levels remained within the normal range in both groups.

A strong positive correlation was found between zinc and serum serotonin levels ($r = 0.58$, $p < 0.001$), suggesting that zinc insufficiency may exacerbate serotonergic dysfunction. Magnesium levels correlated inversely with the frequency of headaches and migraine-like episodes ($r = –0.41$, $p < 0.01$) [7].

EEG and Neuropsychological Findings

EEG recordings in the NEPH group showed non-epileptiform abnormalities in 37.8% of cases, mostly represented by diffuse cortical irritability and mild regional slowing. No epileptiform discharges were detected in any child included in the NEPH group, consistent with their clinical diagnosis.

Neuropsychological assessment revealed that 54.0% of NEPH patients exhibited attention deficits, 46.0% demonstrated emotional dysregulation, and 28.3% had sleep disturbances. These symptoms were significantly more common in children with documented serotonin and dopamine imbalance.

Clinical Correlations

Children with combined serotonin and dopamine deficiency were more likely to present with multiple symptom clusters, including headache, tics, autonomic crises, and behavioral instability. Among NEPH patients with dual neurotransmitter deficiency ($n = 39$), the mean frequency of paroxysmal episodes was significantly higher (3.8 ± 1.1 episodes/month) compared to patients with isolated deficiency or normal neurotransmitter levels (1.6 ± 0.9 episodes/month, $p < 0.01$).

1. Serotonin and dopamine levels were significantly reduced in children with NEPH.
2. Zinc and magnesium deficiencies were more frequent among NEPH patients and correlated with neurotransmitter imbalance.
3. Lower serotonin was associated with autonomic instability and emotional disorders.
4. Lower dopamine correlated with hyperkinetic motor symptoms and attention deficits.
5. EEG abnormalities did not indicate epilepsy but supported functional dysregulation.
6. Combined neurochemical deficiencies were associated with more severe clinical presentation.

These findings collectively support the hypothesis that neurotransmitter imbalance and trace element deficiency play a central role in the development and severity of non-epileptic paroxysmal conditions and related neurodevelopmental disturbances in children [7].

5. Discussion

The findings of this study confirm that neurotransmitter imbalance–particularly serotonin and dopamine deficiency –plays a central role in the development and clinical manifestation of non-epileptic paroxysmal conditions (NEPH) and related neurodevelopmental disturbances in children. The significant reduction in serotonin and dopamine levels observed in the NEPH group aligns with existing literature suggesting that monoaminergic dysfunction is a key mechanism in the pathogenesis of autonomic instability, emotional dysregulation, and hyperkinetic motor symptoms. Earlier research by Alexandrova et al. and Ashman & Poverennova has similarly emphasized the vulnerability of the pediatric nervous system to biochemical disturbances, particularly during critical developmental periods when neurotransmitter systems mature rapidly [8].

The strong association between serotonin deficiency and increased frequency of autonomic crises in our study reinforces current neurobiological models that link serotonergic pathways with regulation of vascular tone, stress response, and autonomic stability. Low serotonin levels likely compromise descending inhibitory pathways that modulate sympathetic activation, increasing susceptibility to panic-like episodes, hyperventilation crises, and migraine-like headaches. This is consistent with international studies demonstrating that serotonin plays an integral role in autonomic balance and pain modulation in pediatric populations.

Dopaminergic dysfunction observed in NEPH patients provides further insight into the mechanisms underlying motor instability, tics, dyskinesia, and attention deficits. Dopamine is crucial for the regulation of frontal–striatal circuits involved in executive function, movement control, and behavioral regulation. The significant correlation between low dopamine levels and hyperkinetic motor symptoms in this study supports findings from neuroimaging and neuropathological research linking dopaminergic hypoactivity with movement disorders and attention dysfunction. These results also correspond with current ADHD pathophysiology models, which implicate insufficient dopamine transmission in impaired attention, impulsivity, and hyperactivity [9].

An important contribution of this study is the demonstration of trace element deficiency–particularly zinc and magnesium–as a contributing factor to neurotransmitter imbalance and clinical symptom severity. The high prevalence of zinc deficiency among children with NEPH suggests a mechanistic link between micronutrient status and serotonergic function, as zinc is known to influence

serotonin synthesis and receptor activity. The significant correlation between zinc levels and serotonin concentrations supports the hypothesis that micronutrient insufficiency may exacerbate serotonergic dysfunction. Similarly, the association between magnesium deficiency and increased frequency of headaches and paroxysmal episodes aligns with existing evidence that magnesium is a natural NMDA receptor modulator and plays a protective role against cortical hyperexcitability.

Interestingly, the study found no significant deviations in calcium levels, suggesting that zinc and magnesium status may be more directly implicated in the neurochemical imbalances observed in NEPH. This aligns with several pediatric studies demonstrating that zinc and magnesium deficiencies are more prevalent in children with autonomic dysfunction, learning disabilities, and paroxysmal disorders [9].

EEG findings in the NEPH group were consistent with functional rather than epileptic abnormalities. While EEG abnormalities–such as diffuse slowing or cortical irritability–were present in a subset of patients, no epileptiform activity was recorded. This supports the growing consensus that NEPH represents a distinct diagnostic category characterized by episodes resembling epilepsy but arising from autonomic, psychological, or metabolic dysregulation rather than cortical epileptic discharges. Consequently, unnecessary antiepileptic treatment may be avoided by integrating biochemical analysis with EEG findings.

The combined deficit of serotonin and dopamine was associated with a more severe clinical presentation, including higher frequency of paroxysmal episodes and greater emotional and behavioral instability. This finding is clinically important, as it suggests that children with dual neurotransmitter deficiency may require more comprehensive diagnostic evaluation and individualized treatment strategies, addressing both neurochemical and psychological factors.

Overall, the results highlight the importance of a multimodal diagnostic approach that incorporates clinical examination, neurophysiological studies, and biochemical profiling. Traditional diagnostic tools such as EEG and neuropsychological testing are essential but may not adequately capture the underlying neurochemical disturbances contributing to NEPH and related disorders. The integration of neurotransmitter and trace element assessments significantly enhances the diagnostic accuracy and supports more targeted therapeutic interventions [10].

Despite the strengths of this study, several limitations should be noted. The cross-sectional design prevents assessment of causal relationships between neurotransmitter levels and clinical manifestations. Circadian variations and environmental factors may also influence biochemical markers despite standardization of sampling conditions. Additionally, reference ranges for pediatric neurotransmitter levels vary across laboratories, which may limit the comparability of results with other studies. Future research should focus on longitudinal designs, larger sample sizes, and standardized biochemical reference values to better elucidate causal pathways.

Nevertheless, the results provide compelling evidence that neurotransmitter imbalance and micronutrient deficiencies contribute significantly to the pathogenesis of NEPH and neurodevelopmental disorders in children. Early identification of these biochemical markers may improve diagnostic precision, reduce misdiagnosis of epilepsy, and guide personalized treatment strategies that address both neurochemical and functional aspects of pediatric neurological disorders.

6. Study Limitations

This study has several limitations that should be taken into consideration when interpreting the findings. First, the cross-sectional design does not allow for establishing causal relationships between neurotransmitter imbalance, trace element deficiency, and the clinical manifestations of non-epileptic paroxysmal conditions in children. Although significant associations were identified, it is not possible to determine whether altered serotonin and dopamine levels are the cause or the consequence of neurodevelopmental disturbances.

Second, peripheral blood measurements of neurotransmitters may not fully reflect central nervous system activity. Serotonin and dopamine have limited ability to cross the blood–brain barrier, and peripheral concentrations may be influenced by systemic physiological processes, circadian rhythms, diet, and stress. Despite standardized sampling procedures, these factors may introduce measurement variability.

Third, reference ranges for pediatric neurotransmitter levels are not fully standardized across laboratories, which may limit generalizability and direct comparison with findings from other clinical settings or populations. Similarly, the quantification of trace elements such as zinc and magnesium can be influenced by dietary intake, recent illness, and individual metabolic differences.

Another limitation relates to sample size and population characteristics. Although the study included 118 participants, larger multicenter studies would provide more robust estimates of prevalence, biochemical patterns, and symptom correlations. The study population was recruited from a single pediatric neurology center, which may limit the applicability of the findings to broader or more diverse pediatric groups.

Additionally, the study did not include advanced neuroimaging techniques—such as fMRI or PET—that could offer deeper insight into structural or functional changes associated with neurotransmitter imbalance. Incorporating these methods in future research may help clarify neural circuitry differences underlying NEPH and related disorders.

Finally, potential confounding factors such as sleep patterns, dietary habits, psychological stress, family environment, and subclinical medical conditions were based on parental reports and not controlled through objective measures. These variables may influence both neurotransmitter status and

behavioral manifestations.

Despite these limitations, the study provides important evidence supporting the role of neurotransmitter imbalance and micronutrient deficiency in the pathogenesis of pediatric neurodevelopmental disturbances. Future longitudinal and multicenter studies with standardized biochemical protocols and integrated neuroimaging may further strengthen understanding of these mechanisms.

7. Conclusions

The results of this study demonstrate that neurotransmitter imbalance–particularly decreased levels of serotonin and dopamine–plays a significant role in the development and clinical expression of non-epileptic paroxysmal conditions (NEPH) and related neurodevelopmental disorders in children. Children with NEPH showed markedly lower concentrations of serotonin and dopamine compared to peers without paroxysmal episodes, and these deficiencies were strongly associated with autonomic instability, emotional dysregulation, hyperkinetic motor symptoms, and impaired attention. The findings confirm that monoaminergic dysfunction is a central neurobiological mechanism underlying many of the functional neurological symptoms observed in pediatric practice.

Trace element deficiencies, particularly zinc and magnesium, were identified as additional factors contributing to neurotransmitter dysregulation. Their strong correlation with serotonin and symptom severity suggests that micronutrient insufficiency may exacerbate neurochemical and autonomic imbalance. This highlights the importance of including trace element profiling in the diagnostic workup for children presenting with paroxysmal events, recurrent headaches, behavioral disturbances, or autonomic symptoms.

EEG results in the NEPH group showed non-epileptiform abnormalities, reinforcing the distinction between epileptic seizures and functional paroxysmal events. The absence of epileptiform activity underscores the need for clinicians to avoid overdiagnosis of epilepsy and inappropriate prescription of antiepileptic medications. Instead, a multimodal diagnostic approach integrating EEG, neuropsychological assessment, neurotransmitter profiling, and trace element analysis offers a more accurate diagnostic pathway.

Overall, the study supports the necessity of an interdisciplinary approach that considers biochemical, neurophysiological, and clinical factors in the early diagnosis and management of pediatric neurodevelopmental disorders. Early identification of serotonin and dopamine deficiency, as well as zinc and magnesium imbalance, opens new possibilities for individualized therapeutic strategies aimed at correcting neurochemical disturbances and improving clinical outcomes. Future research should focus on longitudinal studies to better understand causal relationships, refine diagnostic standards, and develop personalized treatment protocols based on neurotransmitter and micronutrient profiles.

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