

CLINICAL AND LABORATORY ASPECTS OF NEUROVASCULAR PATHOLOGY IN ADOLESCENTS

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Abstract: Cerebrovascular pathology in adolescence represents a pressing medical and social problem that requires special attention due to the age-related characteristics of brain vascular system formation. The pubertal period is characterized by intensive neuroendocrine restructuring, changes in hemodynamics, and metabolic processes, which creates prerequisites for the development of various forms of cerebral circulatory disorders.

Key words: Cerebrovascular Pathology, Adolescents, Laboratory Diagnostics, Biomarkers, Endothelial Dysfunction, Cerebral Circulatory Disorders, Pubertal Period

Introduction. The diagnosis of cerebrovascular disorders in adolescents is complicated by the polymorphism of clinical manifestations, often the nonspecific nature of symptoms, and the need for differential diagnosis with functional disorders characteristic of this age period. Traditional diagnostic approaches used in adult patients are not always adequate to the characteristics of the adolescent organism, necessitating the development of specialized assessment criteria.

Laboratory research methods are of particular importance in diagnosing cerebrovascular pathology in adolescents, allowing for the objectification of pathological processes and the identification of markers for endothelial dysfunction, hemostasis disorders, inflammatory reactions, and metabolic disorders. The determination of specific biochemical indicators characterizing the state of the neurovascular system contributes to the early detection of pathology and the monitoring of therapeutic efficacy[1].

Neurovascular pathology in adolescence is considered a rapidly growing clinical and social problem today, as global epidemiological studies record an increase in stroke incidence in children and adolescents over recent decades while simultaneously reducing mortality due to early diagnosis and the accessibility of neuroimaging. Simultaneously, "non-manifest" (subclinical) neurovascular forms persist, manifesting as cephalgic and astheno-vegetative syndromes, impaired adaptation to workloads, decreased school productivity, and quality of life, which increases the risk of chronicity and late vascular vulnerability[2]. At the international level, it is emphasized that the key limitation remains delays in recognition and insufficient standardization of routing for adolescents with vascular symptoms, especially outside of specialized centers[3]. A fundamental feature of adolescent neurovascular pathology in contrast to the "adult" cerebrovascular model. While atherosclerosis, long-term metabolic comorbidity, and age-related remodeling of the vascular wall predominate in adults, in adolescence, congenital vascular anomalies, arteriopathies, infectious-inflammatory triggers, as well as functional disorders of hemodynamic regulation against the background of arterial hypertension and stress during the pubertal period, are leading [4,5,6]. The aspect of cerebral blood circulation "maturation" is also important: cerebral blood flow parameters and cerebrovascular reactivity change with age, requiring age-specific interpretation of examination results in adolescents[7]. Against the background of primary hypertension in children and adolescents, a deterioration in a number of cognitive domains (attention,

information processing speed, memory) is shown, confirming the clinical-cerebrovascular connection at a young age[8]. Despite the progress in instrumental diagnostics (ultrasound methods, TCDG, MR angiography), a clinical and laboratory "bridge" that allows for early detection of vascular vulnerability and risk stratification in adolescents has not been sufficiently developed[9]. In recent years, the role of endothelial dysfunction as an early, potentially modifiable link in adolescent hypertension and vascular damage has been actively discussed; the prospects for combining functional tests and biomarkers of inflammation/oxidative stress to clarify the risk are emphasized[10,11]. In pediatric vascular pathology, data are also accumulating on the significance of inflammatory blood proteins as markers of severity and variation of a vascular event, including hemorrhagic forms in vascular malformations [12]. However, in the clinical practice of adolescents, there are still no unified algorithms where laboratory markers are interpreted in conjunction with clinical syndromes and objective indicators of cerebral hemodynamics and reactivity. Thus, the scientific and practical significance of studying the clinical and laboratory aspects of neurovascular pathology in adolescents is determined by the need for a comprehensive assessment of the clinical manifestations of the disease in combination with laboratory markers of vascular dysfunction, endothelial activation, and inflammatory changes. Combining clinical analysis with objective laboratory indicators allows for a deeper understanding of the pathogenesis of neurovascular disorders in the pubertal period, differentiates the functional and structural mechanisms of vascular vulnerability, and enhances the accuracy of early diagnosis and prognostic assessment of the disease course[13,14]. The development and verification of clinical and laboratory criteria creates a basis for more justified risk stratification and optimization of therapeutic and preventive measures in adolescents with neurovascular pathology.

The aim of the study was to determine the clinical and laboratory features of neurovascular pathology in adolescents and to establish the diagnostic significance of laboratory markers of vascular dysfunction.

Materials and research methods. The study was conducted at the Multidisciplinary Clinic of Samarkand State Medical University in the departments of pediatric neurology, neurology, and therapeutic departments during 2024–2025. The study included adolescents aged 16 to 19. The main group consisted of 41 patients with clinically verified neurovascular pathology in accordance with the ICD-10 classification. In the structure of the main group, the following subgroups were identified: adolescents with congenital cerebral vascular anomalies (Q28.0–Q28.3) – 24.4% (n=10), patients with other cerebrovascular diseases (I67) – 21.9% (n=9), and adolescents with secondary neurovascular conditions against the background of arterial hypertension (I10–I15) – 53.7% (n=22). The average age of patients in the main group was 17.8 ± 1.1 years. In terms of gender distribution in the main group, girls accounted for 51.2% (n=21), while boys accounted for 48.8% (n=20). The control group consisted of 37 practically healthy adolescents of comparable age (17.6 ± 1.0 years) who underwent preventive medical examination at the SamSMU Polyclinic Department. Girls in the control group accounted for 54.1% (n=20), while boys accounted for 45.9% (n=17). In adolescents of the control group, there were no clinical or laboratory signs of neurovascular pathology. The inclusion criteria were age from 16 to 19 years, the presence of clinical signs of neurovascular pathology, and a verified diagnosis according to ICD-10.

Exclusion criteria included acute infectious diseases, severe somatic pathology in the decompensation stage, congenital malformations of the central nervous system not associated with vascular pathology, and refusal to participate in the study. All adolescents underwent a comprehensive clinical examination with an assessment of complaints and disease history, clarification of risk factors (arterial hypertension, excess body weight, hypothyroidism, stress, sleep disorders, family history of vascular diseases), as well as a standard neurological examination documenting focal and general cerebral symptoms.

Particular attention was paid to the characteristics of cephalic syndrome (frequency, duration, triggers), the presence of dizziness, syncopal/presyncopal episodes, decreased tolerance to physical and educational loads, sleep disturbances, and asthenic manifestations. The assessment of autonomic status included recording autonomic complaints (heart palpitations, labile blood pressure, cold limbs, sweating, thermoregulatory disorders, weather sensitivity), determining the Kerdo index, and analyzing heart rate variability indicators based on a short ECG/cardiointervalography record, calculating temporal parameters (SDNN, RMSSD) and, if possible, frequency indicators (LF, HF, LF/HF). To objectify the

neuropsychological profile, standardized tests and questionnaires were used: MoCA-adolescent for the screening assessment of cognitive functions, Trail Making Test-A (TMT-A) for assessing information processing speed and attention, the MFI-20 scale for the quantitative characterization of asthenovegetative syndrome, and the PedsQL quality of life questionnaire. Laboratory examination was conducted to identify markers of vascular dysfunction, inflammatory activity, and hemostasis disorders as possible pathogenetic components of neurovascular pathology in adolescents. All patients underwent basic clinical and biochemical studies, including general blood analysis, lipid profile (total cholesterol, LDL, HDL, triglycerides), fasting glucose, as well as liver and kidney function indicators according to clinical indications. Given the direction of the study, special attention was paid to the following laboratory markers: Endothelial dysfunction marker (endothelin-1), which reflects the degree of vasoconstrictive activity and vascular regulation. Subclinical inflammation marker, highly sensitive C-reactive protein (hs-CRP) and interleukin-6 (IL-6), as indicators of systemic inflammatory activation and vascular vulnerability. Indicators of the coagulation link, fibrinogen, and D-dimer characterizing prothrombotic readiness and hemostasis status. A marker of metabolic and vascular vulnerability is homocysteine, which is associated with endothelial dysfunction and microcirculation disorders. Indicators were determined in the clinical-diagnostic laboratory of SamSMU MS using standardized enzyme-linked immunosorbent and biochemical methods.

The obtained laboratory data were compared with clinical manifestations, autonomic regulation indicators, and neuropsychological status to identify pathogenetic relationships and determine the diagnostic significance of the markers. Statistical processing was performed using the SPSS program (on a personal computer). Quantitative data are presented as $M \pm SD$. The comparison of indicators between groups was conducted using the Student's t-test or the Mann-Whitney test. Qualitative traits were analyzed using the χ^2 -criterion. Correlation relationships were evaluated using the Pearson or Spearman coefficient. The diagnostic significance of laboratory markers was determined using the ROC analysis method. Differences were considered statistically significant at $p < 0.05$.

Result. Analysis of clinical complaints showed that in adolescents of the main group ($n=41$), cephalgic and asthenovegetative syndromes were significantly more frequently registered compared to the control group ($n=37$). Headache complaints were reported by 78.0% of patients in the main group ($n=32$), compared to 18.9% ($n=7$) in the control group ($\chi^2=24.6$; $p<0.001$), reflecting a difference of 59.1% between the groups. Dizziness symptoms were identified in 56.1% ($n=23$) of adolescents in the main group and 10.8% ($n=4$) in the control group ($p<0.001$). The next frequent sign of the disease was increased fatigue, which was recorded in 73.2% ($n=30$) of patients in the main group compared to 24.3% ($n=9$) in the control group, where the difference was $p<0.001$. Cognitive insufficiency was noted in the form of decreased attention concentration and complaints of deteriorating learning productivity, identified in 63.4% of patients in the main group ($n=26$) and only in 16.2% ($n=6$) in the control group ($p<0.001$). Gender differences as a result of the analysis revealed that headaches were more frequently registered in girls of the main group in 85.7% of cases (out of 21), and in boys of the main group in 70.0% of cases (out of 20), but no statistically significant dependence on gender was identified ($p=0.18$). At the same time, astenovegetative complaints (fatigue, sleep disturbances) were more pronounced in girls (81.0% versus 65.0%), demonstrating a trend toward gender differences. When distinguishing by disease type, it was established that in disease variants with ICD code 10 (I10–I15), headache was noted in 86.4% of patients (out of 22), while in the disease type with code Q28.0–Q28.3, it was noted in 70.0% of patients (out of 10), while in disease type I67, these symptoms were identified in 66.7% (out of 9), $p=0.04$, confirming a more pronounced clinical polymorphism in hypertensive-associated conditions.

Clinical and neurological examination showed that in this category of patients, the characteristic features are signs of moderate autonomic dysfunction (lability of pulse and blood pressure, coldness of limbs, hyperhidrosis), which were found in 68.3% of patients in the main group, while in the control group they amounted to 21.6% ($p<0.001$). Mild coordination disorders were recorded in 29.3% of the primary group and were absent in the control group ($p=0.002$). In total, signs of focal neurological symptoms (light and moderate degrees) were primarily noted in the following disease types according to ICD-10: Q28.0–Q28.3 (40.0%) compared to I10–I15 (18.2%). The expanded analysis result for vegetative status was characterized by a significant increase in the Kerdo index in the main group (18.6 ± 6.2) compared to the

control group (6.4 ± 3.1 ; $p < 0.001$), reflecting the predominance of sympathicotonia. The SDNN indicator decreased in patients of the main group (32.4 ± 7.8 ms) compared to 48.1 ± 9.2 ms in the control group ($p < 0.001$), while the RMSSD indicator decreased by 24.7 ± 6.1 ms compared to 38.6 ± 7.5 ms ($p < 0.001$). The decrease in SDNN correlated with the severity of fatigue ($r = -0.42$; $p = 0.01$) and the frequency of headaches ($r = -0.38$; $p = 0.02$).

A study in the direction of neuropsychological analysis of patients with neurovascular insufficiency showed that the average score on the MoCA-adolescent scale was 23.1 ± 2.4 in the main group and 27.4 ± 1.8 in the control group, respectively ($p < 0.001$), reflecting a decrease of 15.7%. Furthermore, the time-dependent performance level of the TMT-A test increased in the primary group (54.3 ± 9.1 sec) compared to the control group (36.8 ± 7.5 sec; $p < 0.001$). The control result on the MFI-20 scale, where the total score was 48.7 ± 6.9 in the main group, differed from the control group at 32.2 ± 5.4 , where the significance was $p < 0.001$. An important component in the study is determining the nature of the correlation analysis, as the decrease in scores on the MoCA scale significantly correlated with an increase in the Kerdo index ($r = -0.46$; $p = 0.004$) and a decrease in the SDNN indicator ($r = 0.44$; $p = 0.006$), confirming the specificity of the correlation between cognitive and autonomic disorders in the examined patients [15].

Discussion. The obtained data indicate the formation of a stable clinical and functional complex in adolescents with neurovascular pathology, including cephalgic, astheno-vegetative, and cognitive syndromes, which differs significantly from the indicators of conditionally healthy peers. The most pronounced manifestations are recorded in the hypertensive-associated variant (I10–I15), indicating the leading role of regulatory-hemodynamic disorders in the pathogenesis of the disease. The identified correlations between autonomic dysfunction and cognitive decline confirm the unity of the clinical and functional mechanism of the lesion and justify the need for a comprehensive assessment of adolescents with neurovascular pathology.

The next stage of the study and analysis, in accordance with the set goal, was to determine the clinical and laboratory parameters of changes in patients with neurovascular insufficiency. Thus, the analysis of laboratory indicators showed significant differences between the main group of adolescents with neurovascular pathology ($n = 41$) and the control group ($n = 37$) regarding markers of endothelial dysfunction, inflammatory activity, and hemostasis status. The average level of endothelin-1 in the main group at the time of the study was 2.84 ± 0.63 pg/ml, and in the control group, it was 1.72 ± 0.41 pg/ml, which was 65.1% higher ($p < 0.001$). An increase in endothelin-1 was noted in 73.2% of patients in the main group, whereas in the control group, it was only 13.5% ($\chi^2 = 22.4$; $p < 0.001$). In turn, endothelin-1 levels had a positive correlation with the Kerdo index ($r = 0.48$; $p = 0.003$) and the frequency of cephalgia (headaches) ($r = 0.41$; $p = 0.01$), where a correlation between vascular regulation and clinical manifestations is observed in the examined patients. Highly sensitive CRP in the main group was 3.8 ± 1.2 mg/l, compared to 1.6 ± 0.7 mg/l in the control group, with a difference of 57.9% ($p < 0.001$). Furthermore, during the analysis, an increased value (> 3 mg/l) was noted in 58.5% of adolescents in the main group, whereas in the control group, it was 8.1% ($p < 0.001$). The IL-6 level value was higher in the main group (5.9 ± 1.8 pg/ml) compared to the control group (2.7 ± 0.9 pg/ml), where the increase parameters showed a high degree of difference, amounting to 54.2% ($p < 0.001$). Additionally, the comparison parameters between IL-6 interleukin levels and MFI-20 scores proved to be an important component, which established a positive correlation ($r = 0.44$; $p = 0.006$), indicating the inflammatory component in the formation of astheno-vegetative syndrome in the examined patients. Indicators of the coagulation link also showed significant differences. Thus, the fibrinogen level averaged 4.1 ± 0.8 g/l in the main group, unlike the control group, where the indicator was 3.2 ± 0.6 g/l ($p < 0.001$), which is 21.9% higher than the statistical difference. Fibrinogen level parameters were identical, where fibrinogen was elevated (> 4 g/l) and detected in 46.3% of patients in the main group, while in the control group, it was elevated in 10.8% of cases ($p = 0.002$). The value of laboratory data on blood D-dimer levels in patients of the main group was 0.54 ± 0.18 μ g/ml, and in the control group, it was 0.29 ± 0.11 μ g/ml ($p < 0.001$), where the difference in increase was 46.3%. Additionally, D-dimer level indicators in the statistical range correlated with the severity of cognitive decline on the MoCA scale ($r = -0.39$; $p = 0.02$), showing the interaction of microhemodynamic disorders with cognitive insufficiency in the examined patients. Laboratory

parameters for homocysteine levels were characteristic, as they were up to $12.8 \pm 3.1 \mu\text{mol/L}$ in the main group and $8.6 \pm 2.4 \mu\text{mol/L}$ in the control group, representing an average difference of 32.8% ($p < 0.001$). Furthermore, the analysis result showed that an elevated value of more than $12 \mu\text{mol/L}$ was found in 51.2% of patients in the main group and only in 13.5% in the control group ($p < 0.001$). Correlation analysis of homocysteine showed a negative correlation with the SDNN level indicator ($r = -0.43$; $p = 0.01$), and conversely, a positive correlation with the Kerdo index ($r = 0.40$; $p = 0.02$).

Analysis of research results in accordance with gender comparisons noted that in girls of the main group, the level of highly sensitive C-reactive protein (hs-CRP) was higher ($4.1 \pm 1.3 \text{ mg/l}$) compared to boys ($3.5 \pm 1.0 \text{ mg/l}$), with no statistical significance ($p = 0.09$). However, boys showed a tendency toward higher blood homocysteine levels ($13.4 \pm 3.0 \mu\text{mol/L}$) compared to girls ($12.2 \pm 2.9 \mu\text{mol/L}$), which compared to the statistical control was $p = 0.12$. Thus, no significant differences in laboratory marker analysis were found between the sexes of the patients. The differentiation of laboratory studies depending on the type of disease was identified, as it was noted that the highest indicators of endothelin-1 and homocysteine were recorded in the variant with ICD 10 code (I10–I15) ($3.02 \pm 0.58 \text{ pg/ml}$ and $13.6 \pm 2.8 \mu\text{mol/l}$, respectively), which significantly exceeded the values for the corresponding type of disease according to ICD 10 code (Q28.0–Q28.3) ($2.61 \pm 0.49 \text{ pg/ml}$; $11.7 \pm 2.6 \mu\text{mol/L}$; $p = 0.03$) and disease code (I67) ($2.55 \pm 0.46 \text{ pg/ml}$; $11.9 \pm 2.4 \mu\text{mol/L}$; $p = 0.04$). Furthermore, the level of highly sensitive C-reactive protein (hs-CRP) was also maximum in the hypertensive variant of the disease ($4.2 \pm 1.3 \text{ mg/l}$; $p = 0.02$).

To evaluate the diagnostic significance of laboratory markers in differentiating adolescents with neurovascular pathology from conditionally healthy peers, ROC analysis was conducted. The model included quantitative indicators of endothelin-1, homocysteine, and hs-CRP as the most pathogenetically significant markers of vascular dysfunction and inflammatory activity. The analysis allowed for the determination of their sensitivity, specificity, and optimal threshold values to distinguish between the primary and control groups. The largest area under the curve was demonstrated by endothelin-1 (AUC=0.86; 95% CI: 0.77–0.94; $p < 0.001$), which corresponds to high diagnostic accuracy. Furthermore, the indicators for homocysteine AUC were 0.82, and for highly sensitive C-reactive protein (hs-CRP), these data varied up to 0.79. The optimal cut-off point for endothelin-1 according to the Yuden index was 2.3 pg/ml, at which the sensitivity of the method reached 80.5%, and the specificity reached 78.4%. ROC analysis demonstrates that endothelin-1 (AUC \approx 0.86) possesses the highest diagnostic value among individual laboratory indicators, corresponding to a high discriminatory ability to distinguish between adolescents with neurovascular pathology and conditionally healthy peers. Homocysteine (AUC \approx 0.82) and hs-CRP (AUC \approx 0.79) were characterized by good diagnostic accuracy. The combined model, including three markers (endothelin-1; homocysteine; hs-CRP), demonstrated higher prognostic efficacy (AUC \approx 0.91), thereby demonstrating increased diagnostic significance in the comprehensive assessment of vascular dysfunction and inflammatory components in the examined patients.

Conclusion.

1. In adolescents with neurovascular pathology, cephalgic, asthenovegetative, and cognitive syndromes are significantly more frequently identified compared to conditionally healthy peers ($p < 0.001$), forming a stable clinicofunctional complex of disorders in adolescence.
2. The most pronounced clinical manifestations are recorded in the hypertensive-associated variant of the disease according to ICD code 10 (I10–I15), indicating the leading role of regulatory-hemodynamic mechanisms in the formation of symptoms.
3. In adolescents of the main group, a significant increase in markers of endothelial dysfunction (endothelin-1), systemic inflammation (hs-CRP, IL-6), and indicators of prothrombotic activity (fibrinogen, D-dimer), homocysteine ($p < 0.001$), proved the involvement of the vascular-inflammatory component in the pathogenesis of neurovascular pathology.
4. Significant correlations were established between laboratory markers and clinical manifestations, where endothelin-1 and homocysteine levels are associated with the severity of autonomic dysfunction ($r = 0.40$ – 0.48 ; $p < 0,05$), while inflammation indicators correlate with astheno-cognitive disorders

($r \approx 0.44$; $p < 0.01$), confirming the interaction of the clinical-laboratory mechanism of the disease.

5. Among individual laboratory markers, endothelin-1 has the highest diagnostic value ($AUC \approx 0.86$), while the combined model (endothelin-1; homocysteine; hs-CRP) demonstrates higher discriminatory ability ($AUC \approx 0.91$), which justifies the expediency of a comprehensive laboratory assessment of adolescents suspected of neurovascular pathology.

References:

- [1] E. F. Ibragimova and L. M. Garifulina, "Markers of endothelial dysfunction in children with obesity," *Medical Science of Uzbekistan*, no. 3, pp. 107–111, 2025.
- [2] Zh. D. Kobalava, S. V. Nedogoda, E. V. Shlyakhto *et al.*, "Hypertension in adults: Clinical recommendations," *Russian Cardiological Journal*, vol. 29, Art. no. 6117, 2024.
- [3] N. S. Luksha, O. N. Grishkevich, N. A. Gubich *et al.*, "The state of the endothelium in hyperhomocysteinemia in children with arterial hypertension," *Journal of the Grodno State Medical University*, vol. 21, no. 4, pp. 462–468, 2023.
- [4] Z. U. Todieva, N. E. Zvartau, and S. Yu. Nikitina *et al.*, "Endothelial dysfunction in obese adolescents: clinical and laboratory markers and link with cardiometabolic risk," *Pediatrics. Medicum medicum*, no. 1, pp. 46–52, 2022.
- [5] Yu. A. Khachaturov, I. O. Shchederkina, and N. F. Plavunov *et al.*, "Stroke in children and adolescents," *Journal of Neurology and Psychiatry named after S.S. Korsakov*, vol. 122, no. 12, pp. 76–83, 2022.
- [6] P. Ambrosino, R. Lupoli, A. Di Minno *et al.*, "Endothelial dysfunction and arterial hypertension in children: pathophysiology and clinical implications," *Journal of Cardiovascular Development and Disease*, vol. 9, no. 5, p. 136, 2022.
- [7] A. Engin, "Endothelial dysfunction in hypertension," in *Advances in Experimental Medicine and Biology*, vol. 1460, pp. 489–538, 2024.
- [8] T. Hertis Petek, D. Enko, L. Pertl *et al.*, "Arterial hypertension and oxidative stress/inflammation markers in children and adolescents," *Antioxidants (Basel)*, vol. 11, no. 5, p. 1026, 2022.
- [9] E. F. Ibragimova and L. M. Garifullina, "Role of endothelial dysfunction in the development of cardiovascular complications in children with abdominal obesity," *International Journal of Scientific Pediatrics*, vol. 3, no. 8, pp. 710–718, 2024.
- [10] M. M. Islam, M. Z. Hossain, M. M. Rahman *et al.*, "Prevalence, trends, and determinants of hypertension among adolescents: a systematic review and meta-analysis," *Journal of Global Health*, vol. 15, Art. no. 04115, 2025.
- [11] Yu. A. Khachaturov, I. O. Shchederkina, and N. F. Plavunov *et al.*, "Stroke in children and adolescents," *Neuroscience and Behavioral Physiology*, vol. 53, no. 6, pp. 994–1001, 2023.
- [12] M. Angelo Mastr, C. Azzari, C. D'Aleo *et al.*, "Pediatric arterial ischemic stroke: risk factors and diagnostic challenges," *European Journal of Pediatrics*, vol. 180, no. 4, pp. 1107–1115, 2021.
- [13] V. Micieta, A. Jurko, and I. Brucknerová, "Endothelial dysfunction in adolescent hypertension: diagnostic challenges and early cardiovascular risk," *Journal of Cardiovascular Development and Disease*, vol. 12, no. 9, p. 326, 2025.
- [14] P. B. Sporns, R. Sträter, J. Minnerup *et al.*, "Treatment in pediatric stroke and outcomes: results from the German 'Save ChildS' study," *Stroke*, vol. 52, no. 2, pp. 200–208, 2021.
- [15] M. Wójcik and A. Kotara-Kozakowska, "Obesity, sodium homeostasis, and arterial hypertension in children and adolescents," *Nutrients*, vol. 13, no. 11, p. 4032, 2021.