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ORIGINAL PAPER

THE ROLE OF OXIDATIVE STRESS IN ARTERIAL HYPERTENSION IN OBESE CHILDREN WITH A POSITIVE FAMILY HISTORY

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Abstract

Introduction. Arterial hypertension (AH) in children has emerged as a growing public health concern, frequently associated with excess body weight. Oxidative stress (OS), a key contributor to endothelial dysfunction, is further amplified in the presence of a positive family history (PFH) of cardiovascular diseases. The interaction between these factors facilitates the early onset of hypertension in the pediatric population.
The objective of the study was to assess OS parameters in hypertensive overweight children according to the presence of a PFH of cardiovascular pathology.
Material and methods. A total of 75 children aged 10-18 years were included and stratified into two subgroups, according to the presence of a PFH of cardiovascular pathology. The OS parameters assessed included superoxide dismutase (SOD), glutathione peroxidase (GPx), glutathione reductase (GRx), and glutathione S-transferase (GST), determined by immunoenzymatic methods; catalase (CAT) and malondialdehyde (MDA), measured via spectrophotometry; total antioxidant

Résumé

Le rôle du stress oxydatif dans l'hypertension artérielle chez les enfants obèses ayant des antécédents familiaux positifs

Introduction. L'hypertension artérielle (HTA) chez l'enfant est devenue une préoccupation croissante de santé publique, fréquemment associée à un excès pondéral. Le stress oxydatif (SO), contributeur clé de la dysfonction endothéliale, est davantage amplifié en présence d'antécédents familiaux (AF) positifs de maladies cardiovasculaires. L'interaction entre ces facteurs favorise l'apparition précoce de l'HTA dans la population pédiatrique.
L'objectif de cette étude était d'évaluer certains paramètres du SO chez des enfants hypertendus en surpoids, en fonction de la présence ou non d'AF de pathologie cardiovasculaire.
Matériel et méthodes. Au total, 75 enfants âgés de 10 à 18 ans ont été inclus et répartis en deux sous-groupes selon la présence ou non d'AF de

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activity (TAA) and advanced oxidation protein products (AOPP), assessed by colorimetric assay. Serum levels of advanced glycation end-products (AGEs) were measured.
Results. A PFH of hypertension in first-degree relatives was observed in both maternal and paternal lines, with rates of 70% and 25%, respectively. Hypertensive children with PFH exhibited significantly higher levels of AGEs (389.1 μM/L vs 249.6 μM/L; $p<0.05$) and lower TAA (0.410 vs 0.506 ABTS; $p<0.05$); AOPP were also higher (87.9 μM/L vs 78.0 μM/L), but did not reach statistical significance ($p>0.05$). In the PFH group, body mass index (BMI) showed a positive correlation with CAT activity ($r=+0.433$; $p<0.01$) and negative correlations with TAA ($r=-0.340$; $p<0.05$) and GST ($r=-0.324$; $p<0.05$).
Conclusions. The findings underscore the importance of OS assessment in the management of AH among overweight and obese children with a PFH of cardiovascular disease. Measuring oxidative markers such as TAA, AGEs, and glutathione-dependent enzymes may support early cardiovascular risk identification and prevention of long-term complications.

Keywords: children, arterial hypertension, overweight, oxidative stress, family history.

List of abbreviations:
AH – arterial hypertension
OS – oxidative stress
SOD – superoxide dismutase
GPx – glutathione peroxidase
GRx – glutathione reductase
GST – glutathione S-transferase
MDA – malondialdehyde
CAT – catalase
TAA – total antioxidant activity
AOPP – advanced oxidation protein products
AGEs – advanced glycation end-products
BMI – body mass index
ROS – reactive oxygen species
ESC – European Society of Cardiology
MI – myocardial infarction
DM – diabetes mellitus
CVA – cerebrovascular accident

pathologies cardiovasculaires. Les paramètres de SO évalués comprenaient la superoxyde dismutase (SOD), la glutathion peroxydase (GPx), la glutathion réductase (GRx) et la glutathion S-transférase (GST), déterminées par des méthodes immunoenzymatiques; la catalase (CAT) et le malondialdéhyde (MDA), mesurés par spectrophotométrie; l'activité antioxydante totale (AAT) et les produits d'oxydation avancée des protéines (AOPP), évalués par dosage colorimétrique. Les niveaux sériques des produits avancés de glycation (AGEs) ont été mesurés.
Résultats. Des AF positifs d'HTA chez des parents au premier degré ont été observés sur les lignées maternelle et paternelle, dans 70% et 25% des cas, respectivement. Les enfants hypertendus avec AF présentaient des niveaux significativement plus élevés d'AGEs (389,1 μM/L contre 249,6 μM/L) et AAT plus faible (0,410 contre 0,506 ABTS) comparativement aux enfants sans AF ($p < 0,05$); les AOPP étaient également plus élevés (87,9 μM/L contre 78,0 μM/L), sans atteindre la signification statistique ($p>0,05$). Dans le groupe avec AF, l'indice de masse corporelle (IMC) montrait une corrélation positive avec l'activité de la CAT ($r=+0,433$; $p<0,01$), et des corrélations négatives avec l'AAT ($r = -0,340$; $p < 0,05$) et l'activité de la GST ($r = -0,324$; $p < 0,05$).
Conclusions. Les résultats soulignent l'importance de l'évaluation du SO dans la prise en charge de l'HTA chez les enfants en surpoids ou obèses ayant des AF de maladies cardiovasculaires. Le dosage de marqueurs oxydatifs tels que l'AAT, les AGEs et les enzymes dépendantes du glutathion pourrait favoriser l'identification précoce du risque cardiovasculaire et contribuer à la prévention des complications à long terme.

Mots-clés: enfants, hypertension artérielle, surpoids, stress oxydatif, antécédents familiaux.

Introduction

Arterial hypertension (AH) in overweight children with a positive family history (PFH) of cardiovascular disease is driven by multiple pathogenetic mechanisms that influence disease progression[1-5]. Oxidative stress (OS) has garnered significant attention due to its involvement in endothelial dysfunction and the initiation of chronic inflammation[6-9]. The imbalance between excessive production of reactive oxygen species (ROS) and the efficiency of endogenous antioxidant systems leads to measurable alterations in the oxidative profile, even in children with AH and excess weight, manifested by elevated levels of lipid peroxidation and advanced glycation end-products, alongside reduced enzymatic antioxidant activity[10-13].

Among the relevant biomarkers, the glutathione system (glutathione peroxidase (GPx), glutathione

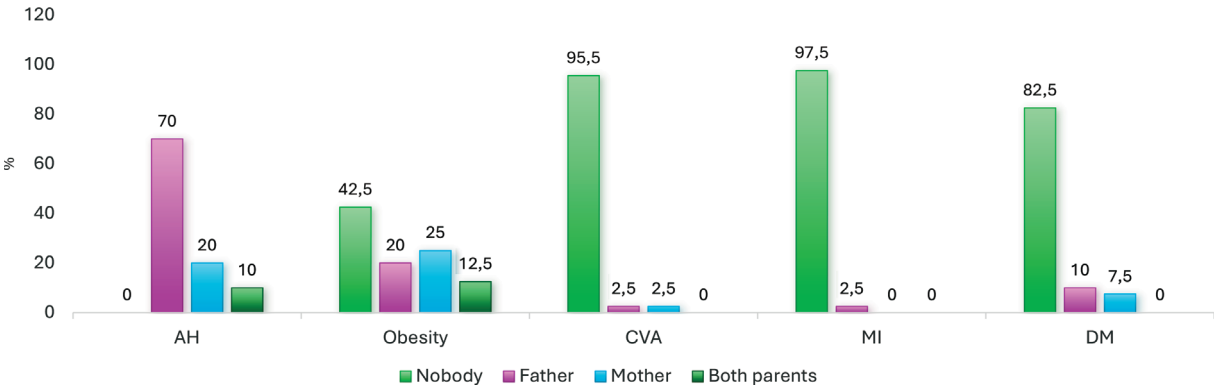


Figure 1. Characteristics of the positive family history in first-degree relatives (%).
Note: AH – arterial hypertension; CVA – cerebrovascular accident; MI – myocardial infarction; DM – diabetes mellitus.

reductase (GRx), and glutathione S-transferase (GST)) ensures the detoxification of peroxides and regeneration of reduced glutathione. Total antioxidant activity (TAA) reflects the organism's integrated capacity to neutralize free radicals; and advanced glycation end-products (AGEs) contribute to enhanced OS and inflammation via receptor activation, promoting vascular stiffening and pathological remodeling[14]. In this context, a PFH of cardiovascular disease may act not only as a hereditary risk factor but also as an epigenetic modulator of oxidative response[14,15].

Although numerous studies have explored the relationship between OS and pediatric AH, data on the direct influence of hereditary predisposition on redox parameters remain fragmented, particularly in the setting of obesity[10-12,16].

The objective of the study was to assess OS parameters in hypertensive overweight children according to the presence of a PFH of cardiovascular pathology.

Material and methods

The study was conducted in the Pediatric Cardiology Research Laboratory of the Public Healthcare Institution Institute of Cardiology, Chisinau, Republic of Moldova. It included a sample of 75 inpatients, aged 10-18 years, with elevated blood pressure and classified as overweight or obese. Based on the presence of a PFH of cardiovascular disease in first-degree relatives under the age of 45 years, the participants were divided into two main groups: group I (35 hypertensive overweight/obese children without PFH) and group II (40 hypertensive overweight/obese children with PFH of cardiovascular pathology in first-degree relatives under 45 years of age). The diagnosis and grading of AH were established according to the 2022 European Society of Cardiology Guidelines for the management of pediatric hypertension[17].

To evaluate the influence of PFH on OS status, the hypertensive overweight children were further subdivided into 8 children with AH, overweight status, without PFH; 13 children with AH, overweight status, and PFH. Similarly, 54 obese, hypertensive children were stratified into two groups according to PFH (27 with PFH and 27 without). A control group was also formed, comprising 35 healthy volunteers (both sexes, aged 10-18 years), non-smokers, with no chronic or acute illnesses, and no personal or family history of cardiovascular disease or obesity.

OS parameters included superoxide dismutase (SOD), GPx, GRx, and GST, measured by ELISA; catalase (CAT) and malondialdehyde (MDA), assessed by spectrophotometry; TAA and advanced oxidation protein products (AOPP), evaluated by colorimetric methods; AGEs, determined according to the methodology described by Makita et al.[1]

The study protocol was approved by the Medical Ethics Committee of Public Healthcare Institution Institute of Cardiology, approval no. 64-AC from 14.10.2010 and informed consent was obtained from all participants and their legal representatives.

Results

A PFH in first-degree relatives was identified in 11 patients (27.5%), while 29 children (72.5%) were found to have a PFH across two generations of relatives. An aggravated family history of AH in first-degree relatives on the paternal side was identified in 28 children (70%), on the maternal side in 8 children (20%), and on both parental lines in 4 children (10%).

In terms of obesity associated with familial history, 10 children (25%) had obese mothers, 8 children (20%) had obese fathers, and 5 children (12.5%) had obesity reported on both parental lines. Diabetes mellitus (DM) was reported in 4 fathers (10%) and 3 mothers (7.5%) of the children included in the study (Figure 1).

Table 1. Comparative profile of oxidative stress parameters in overweight children

	AH (N=21)						*P*
	Overweight without PFH			*Overweight with PFH*			
	N	M	*m*	*N*	M	*m*	
MDA (µM/L)	8	19.095	10.110	13	18.433	6.170	>0.05
AOPP (µM/L)	8	78.044	14.826	13	87.890	10.396	>0.05
TAA (ABTS)	8	0.506	0.027	13	0.410	0.037	**<0.05**
SOD (u/g. Prot)	8	1340.088	20.077	13	1363.048	64.796	>0.05
GPx (nmol/s/L)	8	9.840	0.977	13	7.182	1.560	>0.05
GRx (ng/mL)	8	1.785	0.138	13	1.422	0.057	**<0.05**
CAT (mmol/s.L)	8	15.873	1.651	13	14.059	0.838	>0.05
AGEs(µM/l)	8	249.550	26.592	13	389.075	49.066	**<0.05**
G-S-T (nMol/s. L)	8	28.969	3.584	13	37.257	4.846	>0.05
BMI	8	25.073	0.296	13	25.786	0.436	>0.05

Note: MDA – malondialdehyde; AOPP – advanced oxidation protein products; TAA – total antioxidant activity; SOD – superoxide dismutase; GPx – glutathione peroxidase; GRx – glutathione reductase; CAT – catalase; AGEs – advanced glycation end products; GST – glutathione S-transferase; BMI – body mass index.

Table 2. Comparative profile of oxidative stress parameters in obese children

	AH (N=54)						*p*
	Obese, without PFH			*Obese, with PFH*			
	N	M	*m*	*N*	M	*m*	
MDA (µM/L)	27	24.087	3.416	27	32.675	4.039	>0.05
AOPP (µM/L)	27	86.520	8.330	27	90.198	7.171	>0.05
TAA (ABTS)	27	0.434	0.022	27	0.401	0.020	>0.05
SOD (u/g. Prot)	27	1264.485	26.119	27	1264.709	25.444	>0.05
GPx (nmol/s/L)	27	7.103	0.621	27	6.572	0.420	>0.05
GRx (ng/mL)	27	1.786	0.116	27	1.519	0.089	>0.05
CAT (mmol/s.L)	27	15.815	0.598	27	16.483	0.800	>0.05
AGEs (µM/L)	27	312.044	15.712	27	310.371	24.262	>0.05
G-S-T (nMol/s.L)	27	30.641	2.540	27	29.050	3.085	>0.05
BMI	27	31.505	1.012	27	31.622	0.845	>0.05

Note: MDA – malondialdehyde; AOPP – advanced oxidation protein products; TAA – total antioxidant activity; SOD – superoxide dismutase; GPx – glutathione peroxidase; GRx – glutathione reductase; CAT – catalase; AGEs – advanced glycation end-products; GST – glutathione S-transferase; BMI – body mass index.

The comparative analysis of relevant biochemical parameters revealed significant differences between the group of overweight hypertensive children with a PFH and those without (Table 1).

TAA was significantly lower in the PFH group (0.410±0.037) compared to children without cardiovascular PFH (0.506±0.027; $p < 0.05$).

GRx levels were also significantly reduced in the PFH subgroup (1.422±0.057 ng/mL) compared to the subgroup without PFH (1.785±0.138 ng/mL; $p < 0.05$).

AGEs showed a significant increase in children with PFH (389.075±49.066 µM/L) versus those without (249.550±26.592 µM/L; $p < 0.05$).

Although not statistically significant, clinically relevant trends were observed: AOPP were higher in the PFH group (87.890±10.396 µM/L) compared to the group without PFH (78.044±14.826 µM/L).

The activity of antioxidant enzymes (SOD, GPx, GST, and CAT) was slightly lower in the PFH subgroup, but the differences did not reach statistical significance. Body mass index (BMI) values were comparable between groups (without PFH 25.073±0.296 vs with PFH 25.786±0.436; $p > 0.05$), confirming the homogeneity of the cohorts in terms of weight status.

In the subgroup of hypertensive children with obesity, the comparative analysis of redox parameters revealed the following trends: TAA was lower in the PFH group (0.401±0.020 ABTS) compared to the group without PFH (0.434±0.022 ABTS), although the difference did not reach statistical significance ($p > 0.05$) (Table 2). A similar pattern was observed

for the glutathione system enzymes (GPx, GRx, and GST), which showed slightly lower values in the PFH group, without statistical significance.

Likewise, AOPP and MDA levels were higher in the PFH group; however, these differences were also not statistically significant (AOPP: 90.198±7.171 µM/L vs 86.520±8.330 µM/L; MDA: 32.675±4.039 µM/L vs 24.087±3.416 µM/L; $p > 0.05$).

The concentration of AGEs was similar between the two groups (with PFH: 310.371±24.262 µM/L vs without PFH: 312.044±15.712 µM/L; $p > 0.05$), in contrast to the findings in overweight children, where AGEs were significantly higher in those with a PFH.

In the subgroup without a PFH, a moderate positive correlation was observed between BMI and CAT activity ($r = +0.279$; $p < 0.05$). In contrast, the subgroup with PFH exhibited a stronger positive correlation between BMI and CAT activity ($r = +0.433$; $p < 0.01$), along with two significant negative correlations: between BMI and TAA ($r = -0.340$; $p < 0.05$) and between BMI and GST ($r = -0.324$; $p < 0.05$).

Discussion

AH associated with excess body weight represents a major health concern, with numerous studies highlighting the impact of a PFH on the prevalence, onset, progression, and severity of hypertension[18-20]. It has been demonstrated that children with a PFH of AH, especially among first-degree relatives, have a 2-4 times higher risk of developing the condition[21,22]. In the present study, a PFH among first-degree relatives was identified in 27.5% of participants, while 72.5% had an aggravated familial history involving two generations. Hypertension and obesity were most reported among both maternal and paternal relatives.

The complex pathogenesis of AH involves multiple mechanisms, with OS playing a central role[7,23]. Redox imbalance, defined by excessive production of ROS and impaired antioxidant defense, contributes to endothelial dysfunction, vascular stiffness, and chronic inflammation[7-9]. Excess weight, as a marker of altered metabolic status, exacerbates OS, increasing the risk of early-onset and progressive hypertension starting in childhood.

This study supports this complex relationship, showing that hypertensive children with overweight or obesity exhibit significant redox alterations, including reduced antioxidant enzyme activity (GPx, GRx, CAT, GST), decreased TAA, and elevated prooxidant markers such as AGEs and AOPP. These changes were more pronounced in children with both excess weight and a PFH of cardiovascular diseases.

Correlation analysis further deepens the understanding of these interactions. While only a positive correlation between BMI and CAT activity was found in the subgroup without PFH, the PFH subgroup showed both a positive correlation with CAT and significant negative correlations with TAA and GST. This dual pattern, suggestive of both compensatory activation and functional depletion, reflects a deeper redox dysfunction that may be modulated by genetic or epigenetic factors, clearly distinguishing children with hereditary predisposition. Furthermore, a significant increase in AGEs was observed only in overweight children with PFH, supporting the hypothesis that a PFH of cardiovascular disease acts not merely as a passive risk factor, but as an active determinant of redox vulnerability and early vascular impairment.

Study limitations

The main limitations of this study are the relatively small sample size and the restricted range of OS parameters evaluated. Additionally, the limited number of relevant studies published in the past five years, identified through a literature review of major medical science databases, emphasizes the need for further research in this field. Expanded and up-to-date investigations are required to deepen current understanding and open new directions for research with translational potential.

Conclusions

The present study highlights the importance of integrating OS assessment into the management strategy of hypertensive children with excess body weight, particularly in the context of a PFH of cardiovascular disease. OS markers such as TAA, AGEs, and glutathione-dependent enzymes may serve as valuable tools for cardiovascular risk stratification and for guiding personalized therapeutic approaches. Early identification of redox imbalances may contribute to the prevention of cardiovascular complications later in adulthood.

Author Contributions:

N.M., L.B.-T., S.C., N.R., D.T. were responsible for clinical diagnosis, paraclinical investigations. N.M., L.B.-T., S.C., N.R., D.T. wrote the manuscript. All authors have read and agreed to the published version of the manuscript.

Compliance with Ethics Requirements:

"The authors declare no conflict of interest regarding this article"

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as

well as the national law. Informed consent was obtained from the patients included in the study"

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