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Oxidative Stress Biomarkers in Cerebral Palsy: Ischemia-Modified Albumin, Protein Carbonyl Compounds, AOPP, and Functional Status

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ABSTRACT

Objective: This study aimed to investigate the relationship between oxidative stress markers, including ischemia-modified albumin (IMA), protein carbonyl compounds (PCC), and advanced oxidation protein products (AOPP), and functional status in children with cerebral palsy (CP).

Materials and Methods: A total of 53 children aged 5–12 years diagnosed with CP and 28 age-matched healthy controls were included. Demographic and clinical characteristics, comorbidities, and the use of assistive devices were recorded after physical examination. Serum levels of IMA, PCC, and AOPP were measured in peripheral blood samples. Functional status was assessed using the Gross Motor Function Classification System (GMFCS). Correlations between GMFCS levels and oxidative stress markers were evaluated using Spearman's correlation analysis.

Results: PCC and IMA levels were significantly higher in the CP group than in the control group ($p<0.001$ for both), whereas no significant difference was observed in AOPP levels ($p=0.450$). A statistically significant weak positive correlation was found between GMFCS levels and AOPP levels (rho=0.321, $p=0.022$). No significant correlations were observed between GMFCS levels and PCC, PCC protein, or IMA levels.

Conclusion: IMA and PCC levels were elevated in children with CP compared with healthy controls. In addition, AOPP levels showed a weak but significant association with functional status. These findings indicate that certain oxidative stress-related biomarkers are elevated in children with CP, whereas AOPP levels show a weak association with functional severity. Further longitudinal studies are warranted to clarify the role of oxidative stress in disease progression.

Keywords: Advanced oxidation protein products, cerebral palsy, ischemia-modified albumin, oxidative stress, protein carbonyl compounds.

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INTRODUCTION

Cerebral palsy (CP) is a group of permanent disorders characterized by disturbances in movement, posture, and balance resulting from nonprogressive lesions in the developing fetal or infant brain.[1,2] Children with CP frequently experience a wide range of comorbid conditions, including pain, epilepsy, cognitive and perceptual impairments, communication difficulties, behavioral problems, mental health disorders, and sleep disturbances.[3]

Oxidative stress occurs when the production of reactive oxygen species exceeds the capacity of antioxidant defense mechanisms, leading to cellular and molecular damage.[4] This imbalance contributes to lipid peroxidation, protein oxidation, and DNA damage. Previous studies have demonstrated increased oxidative stress markers and reduced antioxidant capacity in both central and peripheral tissues in neurodegenerative diseases.[5] Excessive oxidative product formation has been observed in various pathological conditions, including cancer, neurodegenerative disorders, and immune dysfunction,[6–8] indicating that oxidative stress plays a critical role in the pathogenesis of brain-related disorders, such as mitochondrial dysfunction, cerebral ischemia, and epilepsy.[9]

In children with CP, several studies have reported elevated oxidative stress markers and diminished antioxidant levels, resulting in a shift toward a pro-oxidative state.[10] Nutritional deficiencies, recurrent epileptic seizures, limited physical activity, and environmental factors have been suggested as potential contributors to oxidative imbalance in this population. Similar interactions between oxidative stress and disease progression have also been reported in other pediatric disorders.[11]

With advances in analytical techniques, the assessment of specific biomarkers of protein oxidation has gained increasing attention.[12] Ischemia-modified albumin (IMA) is one such biomarker that can be measured in peripheral blood. Under conditions such as hypoxia, ischemia, and acidosis, reactive oxygen species modify the N-terminal region of human serum albumin, leading to the formation of IMA.[13] Elevated IMA levels have been reported not only in ischemic conditions but also in metabolic syndrome and chronic liver diseases.[14,15]

Protein carbonyl compounds (PCC) are formed as a result of protein oxidation and represent stable, widely used markers of oxidative stress. Increased PCC levels have been documented in various pathological states, including malnutrition, epilepsy, and sarcopenia.[16,17] Similarly, advanced oxidation protein products (AOPP) are generated during oxidative stress and have gained clinical relevance because of their ease of measurement and strong association with disease severity.

KEY MESSAGES

- IMA and PCC levels were significantly higher in children with cerebral palsy than in healthy controls, indicating increased oxidative stress.
- AOPP levels showed a weak but significant positive association with GMFCS scores.
- These findings suggest that protein oxidation biomarkers may provide new insights into oxidative stress mechanisms in pediatric cerebral palsy.

Elevated AOPP levels have been observed in children with severe obesity and chronic renal failure, where they correlate with oxidative damage and disease progression.[18,19]

To the best of our knowledge, no previous study has evaluated IMA, PCC, and AOPP levels in children with CP. Therefore, the present study aimed to investigate the relationship between these oxidative stress markers and functional status in children with CP. Identifying potential associations between oxidative damage and functional impairment may contribute to improved prognostic assessment and the development of targeted therapeutic strategies.

METHODS

Study Design and Participants

This cross-sectional study was conducted between September 2024 and September 2025 and included 53 children aged 5–12 years who were diagnosed with cerebral palsy by a pediatric neurologist and followed up in the outpatient clinic. A control group consisting of 28 age-matched healthy children who attended the physical medicine and rehabilitation outpatient clinic for a routine physical examination was also recruited.

The exclusion criteria were the presence of genetic or metabolic disorders, autism spectrum disorder, chronic inflammatory diseases, malignancy, psychiatric disorders or intellectual disability in the child or parents, acute infection, recent trauma, epileptic seizures within the previous 3 months, and the use of immunosuppressive or nonsteroidal anti-inflammatory drugs.

The study was approved by Hitit University Faculty of Medicine Research Ethics Committee (Approval Number: 2024-45, Date: 14.08.2024), and written informed consent was obtained from the parents of all participants. All procedures were conducted in accordance with the principles of the Declaration of Helsinki.

Clinical Assessment

Demographic and clinical characteristics, comorbidities, and assistive device use were recorded for all participants.

Motor function in children with CP was classified using the Gross Motor Function Classification System (GMFCS), which categorizes functional status into five levels ranging from Level I, indicating independent ambulation, to Level V, indicating severe functional limitation.[20]

Biochemical Measurements

Sample Collection and Preparation

After 12 hours of overnight fasting, 8 mL of venous blood was collected between 08:00 and 10:00 a.m. into clot activator tubes. Samples were allowed to clot at room temperature for 30 minutes and then centrifuged at 4,000×g for 10 minutes. Serum samples were separated and stored at −70°C until analysis.

Reagents and Instruments

All chemicals and reagents used in the assays included dithiothreitol, cobalt chloride ($CoCl_2 \cdot 6H_2O$), sodium chloride, Tris-barbital buffer, human serum albumin, phosphate-buffered saline, glacial acetic acid, potassium iodide, chloramine-T, 2,4-dinitrophenylhydrazine, trichloroacetic acid (20% TCA), an ethanol/ethyl acetate mixture (1:1, v/v), and guanidine hydrochloride.

Spectrophotometric measurements were performed using a UV-visible spectrophotometer (Thermo Scientific Evolution 201, USA).

Ischemia-Modified Albumin Assay

Serum IMA levels were determined using the albumin cobalt-binding (ACB) assay. This method is based on the reduced capacity of modified albumin to bind cobalt ions. Briefly, serum samples were incubated with Co^{2+} ions, followed by the addition of dithiothreitol. Unbound cobalt formed a colored complex with DTT, and absorbance was measured at 470 nm. Higher absorbance values indicated increased IMA levels.

Determination of Protein Carbonyl Compounds

Serum PCC levels were measured based on the reaction between carbonyl groups in oxidized proteins and 2,4-dinitrophenylhydrazine (DNPH), resulting in the formation of hydrazone derivatives. The absorbance of these derivatives was measured at 360 nm using a spectrophotometer.

Determination of Advanced Oxidation Protein Products

Serum AOPP levels were determined by measuring the oxidation of potassium iodide by chlorinated oxidants present in serum. The formation of triiodide ions was quantified spectrophotometrically at 340 nm, and the results were expressed as chloramine-T equivalents.

Power Analysis

The sample size calculation was performed using G*Power software based on the mean±standard deviation values of IMA levels reported by Cakır et al.[21] The analysis was performed using a two-tailed independent samples t-test with an effect size of 0.79, an alpha error probability of 0.05, and a statistical power (1−β) of 0.80. The minimum required sample size was calculated as 78 participants. To compensate for potential missing biochemical data and laboratory-related sample losses, additional participants were recruited. Consequently, 81 participants (53 children with cerebral palsy and 28 healthy controls) were included in the final analysis.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 29.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as n, %, mean±standard deviation, median, minimum, and maximum values. The normality of numerical variables was assessed using the Shapiro-Wilk test, and the homogeneity of variances was evaluated using Levene's test. Numerical variables were compared between two groups using an independent samples t-test, whereas the relationship between groups based on sex was analyzed using Yates' chi-square test. Correlations between GMFCS values and blood parameters were assessed using Spearman's correlation coefficient. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 81 participants were included in the study, comprising 53 children with cerebral palsy and 28 healthy controls. There was no statistically significant difference in age between the groups. However, a significant difference was observed in sex distribution, with males accounting for 73.6% (n=39) of the CP group and 35.7% (n=10) of the control group ($p<0.05$). Maternal age at birth was significantly higher in the CP group than in the control group.

Table 1 presents the demographic and biochemical characteristics of the children. No significant difference was found in AOPP levels between the groups, whereas PCC and IMA levels were significantly higher in the CP group than in the control group.

Table 2 presents the clinical characteristics of the participants. Overall, 33 patients (62.2%) were diagnosed between 0 and 3 months of age, 10 patients (18.9%) between 3 and 6 months, and 10 patients (18.9%) between 6 and 12 months, while 14 patients (26.4%) were diagnosed at birth. Spastic CP was observed in 28 patients (52.8%), and 36 patients (67.9%) were born prematurely. Speech disorders were present in 38 patients (71.7%), intellectual disability in 32 (60.4%), hearing impairment in 26 (49.1%), visual impairment in 27 (50.9%), and

Table 1. Demographic and biochemical characteristics of children with cerebral palsy and controls

Variables	Groups		Test statistics	
	Cerebral palsy (n=53)	Controls (n=28)	Test value	p
Age, years	7.75±1.57	8.32±1.36	1.616	0.110†
Sex, n (%)				**0.002Φ**
Male	39 (73.6)	10 (35.7)	9.467	
Female	14 (26.4)	18 (64.3)		
Maternal age at birth, years	32.36±4.18	26.54±4.39	5.856	**<0.001†**
AOPP, µmol/L	3.40±1.23	3.18±1.28	0.759	0.450†
PCC, pg/mL	4.28±1.08	3.24±0.86	4.423	**<0.001†**
IMA, ABSU	0.20±0.02	0.18±0.02	4.325	**<0.001†**

n: Number; %: Percentage. Numerical variables are presented as mean±standard deviation. †: Independent samples t-test; Φ: Yates' Chi-square test; AOPP: Advanced oxidation protein products; PCC: Protein carbonyl compounds; IMA: Ischemia-modified albumin; µmol/L: Micromoles per liter; pg/mL: Picograms per milliliter; ABSU: Absorbance units.

Table 2. Clinical characteristics of children with cerebral palsy

Variables	n	%
Age at diagnosis		
≤3 months	33	62.2
3–6 months	10	18.9
>6 months	10	18.9
Type of cerebral palsy		
Ataxic	5	9.4
Dyskinetic	6	11.3
Hypotonic	14	26.4
Spastic	28	52.8
Risk factors and comorbidities		
Hypothyroidism	1	1.9
Premature birth	36	67.9
Prolonged labor	16	30.2
Speech disorder	38	71.7
Intellectual disability	32	60.4
Hearing impairment	26	49.1
Visual impairment	27	50.9
Epilepsy	34	64.2
Use of solid AFO	25	47.2
Botulinum toxin injections	29	54.7
GMFCS	3 (1–5)	

n: Number; %: Percentage; GMFCS values are presented as median (minimum–maximum). AFO: Ankle-foot orthosis; GMFCS: Gross Motor Function Classification System.

Table 3. Correlation between GMFCS levels and oxidative stress markers

	GMFCS	
	rho	p
AOPP	**0.321***	0.022
PCC	0.025	0.858
IMA	-0.066	0.668

*: P<0.05; rho: Spearman correlation coefficient; GMFCS: Gross Motor Function Classification System; AOPP: Advanced oxidation protein products; PCC: Protein carbonyl compounds; IMA: Ischemia-modified albumin.

epilepsy in 34 (64.2%). Twenty-five patients (47.2%) used solid ankle-foot orthoses, and 29 (54.7%) had received botulinum toxin injections. GMFCS levels ranged from I to V, with a median value of III.

Table 3 presents the results of the correlation analysis. A statistically significant weak positive association was found between GMFCS levels and AOPP levels (rho=0.321, p=0.022). Figure 1 shows the scatter plot demonstrating the relationship between GMFCS levels and AOPP concentrations. No significant correlations were observed between GMFCS levels and PCC, PCC protein, or IMA levels. Additional subgroup analyses showed no statistically significant associations between AOPP levels and CP subtype or risk factors (p>0.05 for all comparisons), although mean AOPP levels varied among the groups.

DISCUSSION

The present study demonstrated that serum IMA and PCC levels were significantly higher in children with cerebral palsy than in healthy controls, whereas these biomarkers were not associated with functional status. These findings suggest that oxidative stress is increased in children with CP regardless of

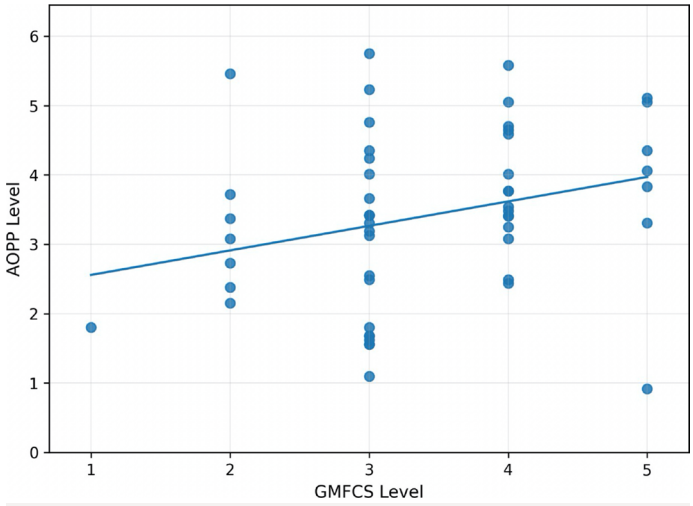

Figure 1. Scatter plot showing the relationship between GMFCS level and AOPP concentration in children with cerebral palsy.

functional severity, emphasizing the importance of evaluating oxidative imbalance in this population.

The global prevalence of CP is estimated to be approximately 0.2–0.3%.[22] Although advances in neonatal and obstetric care have reduced infant mortality, the overall burden of CP remains substantial, and the number of affected individuals continues to rise.[23] Moreover, children with CP are at increased risk of developing chronic conditions in adulthood, including anxiety, depression, cardiovascular disease, osteoarthritis, and cerebrovascular disorders, contributing to increased morbidity and mortality.[24] Oxidative stress has been implicated in the pathogenesis of various pediatric and neurodevelopmental disorders, including autism spectrum disorder and chronic renal failure.[8,11,17] It has been proposed that oxidative stress plays a central role in the gene-environment interactions underlying CP.[25] However, limited data are available on the long-term effects of oxidative imbalance and the most appropriate biomarkers for clinical assessment.

Oxidative stress activates multiple molecular pathways, including protein oxidation, carbonylation, glycosylation, lipoxidation, and nitration, leading to cumulative cellular damage.[26] In the present study, IMA, PCC, and AOPP were evaluated as representative biomarkers of distinct oxidative pathways.

IMA is generated following the structural modification of albumin under ischemic and oxidative conditions and has been widely used as a marker of systemic oxidative stress. Elevated IMA levels have been reported in metabolic disorders, chronic kidney disease, neurodegenerative diseases, and autism, where they are associated with disease activity and clinical severity.[27] Previous studies in adolescents with obsessive-compulsive disorder and children with autism have demonstrated increased IMA levels, although associations with disease severity were inconsistent.[28,29] Consistent with these findings, our results revealed significantly higher IMA levels in children with CP than in controls, without a significant correlation with functional status. This may reflect the early and permanent nature of brain injury in CP, leading to persistent oxidative imbalance independent of current functional performance.

Protein carbonylation represents an irreversible oxidative modification that results in impaired protein function and cellular dysfunction. Increased PCC levels have been documented in metabolic, cardiovascular, and neurodegenerative disorders.[30] Elevated PCC levels have also been observed in preterm infants with respiratory distress syndrome, without clear associations with disease severity.[31] Similarly, our findings demonstrated increased PCC levels in children with CP, suggesting sustained oxidative protein damage irrespective of functional independence.

AOPP are formed through the oxidative modification of plasma proteins and are closely related to chronic inflammation and oxidative stress.[32] Previous studies in Alzheimer's disease have shown increased AOPP levels, although IMA appeared to be a more sensitive biomarker in this context.[33] In the present study, AOPP levels did not differ significantly between children with CP and controls. However, a weak but significant positive correlation was observed between AOPP levels and GMFCS scores, indicating a potential association between oxidative protein modification and functional impairment. These findings highlight the complex and heterogeneous nature of oxidative processes in CP and underscore the need for further investigation.

To date, limited studies have focused on oxidative stress biomarkers in children with CP. Aycicek et al.[10] reported increased lipid peroxidation and reduced antioxidant capacity in this population, while Dogan et al.[34] demonstrated elevated tau protein levels in children with CP and sleep disorders. Consistent with these reports, our study confirmed increased oxidative stress, as reflected by elevated IMA and PCC levels.

This study is among the first to simultaneously evaluate multiple oxidative protein stress biomarkers, including IMA, PCC, and AOPP, in children with cerebral palsy and to investigate their relationship with functional status. Assessing these biomarkers together may provide a broader understanding of oxidative imbalance in CP and its potential clinical relevance.

Limitations

Several limitations should be acknowledged. First, potential contributors to oxidative stress, including nutritional status, psychological factors, and environmental exposures, were not evaluated individually. Second, the relatively wide age range of participants may have influenced oxidative marker levels, and age-stratified analyses could provide more precise results. Third, only oxidative stress markers were assessed, whereas the simultaneous evaluation of antioxidant defense mechanisms might offer a more comprehensive understanding of oxidative balance. Fourth, sex distribution differed significantly between the groups, which may have influenced oxidative stress parameters. Finally, the cross-sectional design precludes causal inferences.

CONCLUSION

This study demonstrates that serum IMA and PCC levels are significantly elevated in children with cerebral palsy, reflecting increased systemic oxidative stress. Although these biomarkers are not strongly associated with functional status, AOPP levels show a weak correlation with motor impairment. These findings suggest that oxidative imbalance is a persistent feature of CP and may contribute to the long-term disease burden. Early identification of oxidative stress and the development of targeted antioxidant interventions may represent promising strategies to improve functional outcomes and quality of life in children with CP. Future longitudinal studies are warranted to clarify the clinical significance of oxidative biomarkers in this population.

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