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Original Article

Evaluation of inflammation with CRP, Pro-BNP, Leptin and Pentraxin-3 levels in children with obesity-related hypertension

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Abstract

Aim: The objective of this study is to explore the association between inflammation and the severity of hypertension by analyzing inflammation biomarkers, such as CRP, IL-1, proBNP, leptin, and PTX-3 levels, in children with obesity-related hypertension.
Material and Methods: The study included 30 children (20 girls, 10 boys) with obesity-related hypertension and 30 healthy children (22 girls, 8 boys) as controls. Clinical and demographic data were collected for all participants. Echocardiographic measurements of left ventricular systolic and diastolic diameters, volumes, and wall thicknesses were taken and adjusted for body surface area. After echocardiographic evaluation, blood samples (10 cc) were drawn from both groups to measure IL-1, CRP, leptin, proBNP, and PTX-3 levels.
Results: The patient group had significantly higher systolic and diastolic blood pressure, height, weight, and body mass index compared to the control group. Echocardiographic assessments revealed significantly increased interventricular diastolic diameter (IVSd), left ventricular diastolic diameter (LVIDd), left ventricular posterior wall diastolic diameter (LVPWd), interventricular systolic diameter (IVSs), and left ventricular systolic diameter (LVIDs) in the patient group. Leptin and CRP levels were notably higher in the obesity group. In healthy children, there was a moderate positive correlation between leptin and systolic blood pressure. A moderate negative correlation was observed between proBNP and systolic blood pressure, and a weak positive correlation between leptin and diastolic blood pressure was noted in the obesity group. Leptin, blood pressure, height, weight, body mass index, and cardiac dimensions were significantly elevated in obese patients requiring anti-hypertensive treatment.
Conclusion: Inflammatory markers may be crucial for early diagnosis of obesity-related hypertension, guiding the development of therapeutic approaches to reduce target organ damage. Elevated inflammation markers in obese patients may indicate the severity of hypertension, aiding in organ damage prevention through lifestyle changes.

Keywords: Obesity, hypertension, inflammation, blood pressure monitoring, echocardiography

INTRODUCTION

Obesity describes the increase in body fat beyond normal levels. The most commonly used method for obesity screening is the calculation of body mass index (BMI) (1). In children and young people up to 20 years of age, obesity is defined as a BMI at or above the 95th percentile, adjusted for age and gender. Overweight is defined as a BMI between the 85th and 95th percentiles. Severe obesity is defined as either a BMI at or above 120% of the 95th percentile (corresponding to the 99th percentile BMI) or a BMI of ≥35 kg/m² (equivalent to the cut-off point for Class II obesity in adults). After the age of 20, adult criteria for overweight and obesity apply (BMI between 25 and 30 kg/m² for overweight, and BMI≥30 kg/m² for obesity).

Studies on adults have shown that the risk of hypertension increases up to 4.8 times in those with Class III obesity (BMI≥40 kg/m²) compared to adults with a normal BMI (2). Two primary hormonal mechanisms are emphasized in obesity-related hypertension. The first is hyperinsulinemia, which results from peripheral insulin resistance; the second is hyperleptinemia, which arises due to an increase in fat tissue mass (3). Obesity is also characterized as a common vascular and systemic inflammatory condition. Obesity-induced proinflammatory cytokines and oxidative stress elevate peripheral vascular resistance by causing vascular endothelial dysfunction and disrupting local vasodilator responses (4). Insulin resistance, low adiponectin levels, increased plasma leptin levels, and elevated plasma glucose and free fatty acids are important indicators of inflammation (5). Changes in circulating cytokine levels and acute phase reactants are characteristic of obese individuals, indicating a state of chronic low-grade systemic inflammation (6,7). Numerous studies have compared left ventricular measurements between obese and healthy children, finding a significant increase in left ventricular mass in obese children (8,9). In obesity-related hypertension, changes in inflammatory markers such as interleukin-1 (IL-1), C-Reactive Protein (CRP), leptin, pro-BNP, and pentraxin-3 (PTX-3) are commonly observed. PTX-3 is an acute phase protein that is released in response to proinflammatory stimuli from various cell types, including adipocytes, neutrophils, monocytes, and macrophages, as part of the inflammatory response associated with obesity. It is produced at sites of inflammation or tissue damage in response to cytokines like IL-1 and tumor necrosis factor-α (TNF-α).

The aim of this study is to determine the relationship between inflammation and the severity of hypertension, using inflammatory biomarkers such as CRP, IL-1, proBNP, leptin, and PTX-3 levels in children with obesity-related hypertension.

MATERIAL AND METHODS

This cross-sectional prospective study was conducted with children aged 2 to 17 who were followed for obesity-related hypertension at the İnönü University Turgut Özal Tıp Merkezi Department of Child Health and Diseases, specifically in the Pediatric Endocrinology, Pediatric Nephrology, and Pediatric Cardiology Clinics, between 2021 and 2022. A total of 60 pediatric patients were included, with 30 in the patient group and 30 in the control group. The control group consisted of children who came for routine pediatric examinations and were found to have no underlying diseases during their physical exams. Exclusion criteria included the presence of major cardiac problems identified during echocardiographic evaluation, age younger than 2 years or older than 17 years, and having systemic diseases other than obesity and hypertension. Prior to the study, ethical approval was obtained from the İnönü University Faculty of Medicine Health Sciences Clinical Research Ethics Committee, and consent was secured from the participants or their parents.

After resting for at least 5 minutes in the clinic, participants' blood pressure was manually measured three times from the right arm using a sphygmomanometer by trained medical personnel in a seated position. Measurements were taken with at least 3 minutes between each reading, and the mean of the three measurements was recorded. The arm was kept at heart level during the measurements. When necessary, patients underwent ambulatory blood pressure monitoring (ABPM); measurements were made using the ABPM device in 20 of the 30 patients in the patient group.

2D and color Doppler echocardiographic evaluations were performed by the same pediatric cardiologist according to the recommendations of the American Society of Echocardiography, using the Vivid Pro 7 GE (GE Healthcare, Florida, USA). After excluding structural heart diseases through 2D and color Doppler echocardiography, left ventricular diameter, wall thickness, and function were assessed using M-mode in the parasternal long axis view. Left ventricular measurements were made according to the recommendations of the American Society of Echocardiography, including interventricular septum diastolic thickness (IVSd), interventricular septum systolic thickness (IVSs), left ventricular diastolic diameter (LVIDd), left ventricular systolic diameter (LVIDs), left ventricular posterior wall diastolic thickness (LVPWd), left ventricular posterior wall systolic thickness (LVPWs), left ventri-cular diastolic mass (LVd mass), left ventricular systolic mass (LVs mass), left ventricular diastolic mass thickness (LVd mass-ASE), and left ventricular systolic mass thickness (LVs mass-ASE). Left ventricular diameter, volume parameters, and wall thickness parameters measured by echocardiography were adjusted for body surface area (m²).

After echocardiographic evaluation, a total of 10 cc of blood was drawn from both the control group and the patients into two biochemistry tubes for measuring IL-1, CRP, leptin, proBNP, and PTX-3 levels. The serum was separated by centrifugation and stored at -80°C. IL-1 was analyzed using the electrochemiluminescence method on a Roche Cobas e 601 device. Hs-CRP levels were determined by the nephelometric method on a Siemens Dade Behring Nephelometer 100 analyzer (Siemens Healthcare Diagnostik Products GmBH, Germany). IGF-1 and NT-proBNP were measured by the chemiluminescence method on a Siemens Immulite-2000 device (Siemens Healthcare Diagnostic Products Ltd., UK). IL-1B, Pentraxin-3, and leptin levels were analyzed using commercial enzyme-linked immunosorbent assay (ELISA) kits (USCN SEK411Hu Pentraxin 3 kit, DIASource ImmunoAssays S.A. Belgium KAP1211 IL-1B kit, DIASource ImmunoAssays S.A. KAPD2395) and were processed using an ELISA method in the research laboratory of the Department of Biochemistry, İnönü University Faculty of Medicine, with a Biotek brand SYNERGY H1 device.

Data analysis was conducted using SPSS version 22. The Kolmogorov-Smirnov test was utilized for assessing normality

distribution. Parametric tests were applied for normally distributed data, while non-parametric tests were used for non-normally distributed data. Mann-Whitney U test, Student's t-test, and Spearman and Pearson correlation analyses were employed. A p-value of <0.05 was considered statistically significant for all tests. Anth-ropometric measurements, blood pressure readings, and echocardiographic measurements of the patients were recorded. Inflammatory parameters and echocardiography results were compared between the two groups.

Hypertension diagnosis was established via ABPM and blood pressure readings at or above the 95th percentile, adjusted for gender, age, and height, based on measurements taken at three different times. In the obesity group, 17 of the 20 obese and hypertensive patients were initiated on medical treatment, while the remaining 3 patients were advised on diet and exercise.

RESULTS

This cross sectional prospective study was conducted with children between the ages of 2 and 17 who were followed for obesity-related hypertension at İnönü University Turgut Özal Medical Center Department of Child Health and Diseases Pediatric Endocrinology, Pediatric Nephrology and Pediatric Cardiology Clinics between 2021 and 2022.

Mean age of the 30 obese and hypertensive patients included in the study was found as 13.86±3.38 years, while the mean age of the 30 individuals in the control group was found as 12.40±2.47 years. Demographic characteristics and anthropometric measurements of the patient and the control group are shown in Table 1.

On the echocardiographic evualation, IVSd, IVSs, LVIDd, LVIDs, LVPWd, LVPWs were found to be statistically higher in the patient group (Table 2).

Table 1. Demographic characteristics and anthropometric measurements of the patient and the control group

	Group		
	Patient (n=30)	**Healthy (n=30)**	**p**
Age (years)	13.86±3.38	12.40±2.47	0.016
Gender			
Male	10 (33.3)	8 (26.7)	0.778
Female	20 (66.7)	22 (73.3)	
Height (cm)	160.90±14.53	155.64±12.76	0.042
Weight (kg)	83.38±25.39	48.47±13.03	<0.001
Body mass index (kg/m²)	31.24±6.05	19.61±2.85	<0.001
Body mass index (percentile)	98.89±1.10	42.81±28.38	<0.001
Systolic blood pressure (mmHg)	141.73±12.15	110.43±4.50	<0.001
Diastolic blood pressure (mmHg)	81.50±11.95	62.93±4.87	<0.001

Table 2. Comparison of echocardiography findings of the children in the patient and control group

	Group				
	Patient (n=30)		**Control (n=30)**		
Variable	**X±SD**	**Median**	**X±SD**	**Median**	**p**
IVSd	0.60±0.13	0.56	0.49±0.12	0.47	**0.004**
LVIDd	2.98±0.42	2.89	2.35±0.37	2.35	**<0.001**
LVPWd	0.56±0.14	0.53	0.48±0.11	0.49	**0.046**
IVSs	0.76±0.14	0.74	0.64±0.11	0.62	**<0.001**
LVIDs	1.83±0.27	1.80	1.35±0.28	1.32	**<0.001**
LVPWs	0.92±0.25	0.85	0.75±0.14	0.74	**0.002**
LVd mass	86.74±33.32	79.41	80.12±16.31	78.42	0.871
LVs mass	66.23±22.86	60.86	59.84±15.68	60.17	0.712
LVd mass AS	75.96±26.39	69.33	72.26±12.92	71.31	0.965
LVs mass AS	61.23±13.98	57.34	57.74±17.83	53.37	0.169

When the inflammation parameters were compared, CRP and leptin levels were found to be statistically significant in the patient group. No statistically significant difference was found between groups in terms of Pentraxin-3, IL-1 and Pro-BNP values (Table 3).

Table 3. Comparison of inflammation parameters in terms of groups

	Group				
	Patient (n=30)		Control (n=30)		
Variable	X±SD	Median	X±SD	Median	p
CRP	1.26±3.64	0.33	0.34±0.07	0.33	**0.041**
Pro-BNP	50.29±58.08	28.15	47.17±32.54	38.50	0.288
Leptin	14.28±11.09	11.16	2.71±3.65	1.35	**<0.001**
IL-1	10210.50±55925.21	0.00	324.90±1779.55	0.00	0.981
Pentraxin-3	68.93±64.47	51.28	66.59±57.15	51.23	0.917

When the correlations between Pentraxin-3 and CRP and echocardiography results were examined in the patient and control group, no significant correlation was found (Table 4). Statisticaly correlation was found between Pro-BNP and echocardiography results in the patient and control group. Negative and moderate correlation was found between LVd mass and LVs mass and Pro-BNP (Table 4) (p=0.02 r=-0.392 and p=0.006 r=-0.492).

Table 4. Correlation between inflammation parameters and left ventricular diameter, wall thicknesses and mass measurements

		Patient				Control			
		CRP	ProBNP	Leptin	PTX-3	CRP	ProBNP	Leptin	PTX-3
IVSd	r	-0.326	0.023	-0.014	0.085	0.054	0.087	0.018	-0.133
	p	0.079	0.903	0.942	0.654	0.778	0.649	0.923	0.483
LVIDd	r	-0.136	-0.290	-0.441	-0.180	-0.032	0.032	-0.189	-0.197
	p	0.472	0.121	0.015	0.340	0.866	0.867	0.317	0.297
LVPWd	r	-0.243	0.169	-0.139	-0.273	-0.011	0.171	-0.012	-0.074
	p	0.196	0.373	0.464	0.145	0.955	0.367	0.948	0.696
IVSs	r	-0.151	-0.311	-0.319	-0.333	0.225	0.285	-0.079	-0.217
	p	0.427	0.094	0.086	0.072	0.231	0.127	0.678	0.249
LVIDs	r	-0.220	-0.005	-0.410	-0.182	-0.064	-0.094	-0.002	-0.223
	p	0.242	0.979	0.025	0.336	0.735	0.622	0.993	0.236
LVPWs	r	0.099	0.239	-0.176	0.127	-0.032	0.186	-0.103	-0.021
	p	0.601	0.204	0.351	0.504	0.866	0.325	0.587	0.913
LVd mass	r	-0.236	-0.392	0.082	-0.183	0.118	-0.321	-0.160	0.231
	p	0.208	0.032	0.668	0.334	0.535	0.083	0.397	0.220
LVs mass	r	0.215	-0.492	-0.094	-0.128	0.290	0.005	-0.013	-0.004
	p	0.253	0.006	0.621	0.501	0.121	0.979	0.945	0.981

When the systolic and diastolic blood pressure and left ventricular measurements were compared, negative weak correlation was found between systolic blood pressure and LVIDs, while positive moderate correlation was found between systolic blood pressure and LVd mass and LVs mass (Table 5).

Table 5. Correlations between blood pressure and left ventricular echocardiography findings

		Sistolic blood pressure	Diastolic blood pressure
LVIDd	**r**	-0.112	-0.234
	p	0.555	0.214
LVPWd	**r**	-0.233	-0.057
	p	0.214	0.764
IVSs	**r**	-0.054	-0.185
	p	0.779	0.329
LVIDs	**r**	-0.373	-0.057
	p	**0.042**	0.765
LVPWs	**r**	-0.173	-0.259
	p	0.359	0.167
LVd mass	**r**	0.467	0.226
	p	**0.009**	0.230
LVs mass	**r**	0.543	0.222
	p	0.002	0.239
LVd mass	**r**	0.434	0.189
	p	**0.017**	0.318
LVs mass	**r**	0.424	0.037
	p	**0.020**	0.847

When the correlations between inflammation parameters and blood pressure findings were examined, a positive moderate correlation was found between leptin and systolic blood pressure in healthy children, while a negative moderate correlation was found between Pro-BNP and systolic blood pressure and a positive weak correlation was found between leptin and diastolic blood pressure in patient group (Table 6).

20 of 30 patients evaluated with ABPM. Medical treatment was started for 17 of 20 obese and hypertensive patients in the obesity group, while diet and exercise were recommended to the remaining 3 patients.

Table 6. Correlations between blood pressure and inflammation parameters in healthy and obese children

		Patient		Healty	
		Sistolic blood pressure	**Diastolic blood pressure**	**Sistolic blood pressure**	**Diastolic blood pressure**
CRP	**r**	0.057	0.275	0.098	-0.011
	p	0.763	0.141	0.607	0.955
Pro-BNP	**r**	-0.566	-0.114	-0.108	0.066
	p	**0.001**	0.547	0.571	0.728
Leptin	**r**	0.064	0.384	0.425	-0.019
	p	0.736	**0.036**	**0.019**	0.921
IL-1	**r**	-0.161	-0.301	-0.272	-0.011
	p	0.395	0.106	0.147	0.955
Pentraxin-3	**r**	**0.037**	0.054	0.187	0.136
	p	0.846	0.777	0.323	0.475

Table 7 shows the comparison of the data of patients in the obesity group who were started hypertension treatment and the patients who were not receiving treatment. Leptin, systolic blood pressure, diastolic blood pressure, height, weight and body mass index, IVSd, LVIDd, LVPWd, IVSs, LVIDs, LVPWs and LVs mass values were found to be statistically significantly high in obese patients who were started anti-hypertensive treatment (Table 7).

Table 7. Comparison of measurements in terms of groups

	Group									
	Obese treatment started			Obese not receiving treatment			Control			
	X	SD	Median	X	SD	Median	X	SD	Median	p
Height	164.00	13.78	165.00	156.85	15.02	161.00	155.64	12.76	157.50	**0.041**
Weight	90.66	24.26	89.00	73.87	24.50	81.00	48.47	13.04	50.50	**0.000**
BMI	33.02	6.44	30.86	28.92	4.79	29.02	19.62	2.85	19.56	**0.000**
BMI percentile	99.02	1.07	99.22	98.73	1.17	98.98	42.82	28.38	29.23	**0.000**
SKB	145.76	10.35	147.00	136.46	12.69	135.00	110.43	4.51	110.00	**0.000**
DKB	85.82	13.61	87.00	75.85	6.11	76.00	62.93	4.88	62.00	**0.000**
CRP	1.82	4.79	0.33	0.52	0.62	0.33	0.34	0.07	0.33	0.091
Pro-BNP	33.60	21.25	24.30	72.12	81.47	41.60	47.17	32.54	38.50	0.137
Leptin	15.02	12.76	7.08	13.31	8.86	14.76	2.71	3.65	1.35	**0.000**
IL-1	18018.53	74292.30	0.00	0.00	0.00	0.00	324.90	1779.55	0.00	0.669
Pentraxin3	75.41	84.24	49.56	60.45	21.14	51.68	66.59	57.15	51.23	0.870
IVSd	0.60	0.13	0.56	0.52	0.12	0.54	0.46	0.12	0.45	**0.006**
LVIDd	2.98	0.42	2.89	2.39	0.35	2.37	2.30	0.40	2.31	**0.000**
LVPWd	0.56	0.14	0.53	0.51	0.12	0.49	0.45	0.09	0.47	0.051
IVSs	0.76	0.14	0.74	0.65	0.14	0.63	0.62	0.08	0.61	**0.001**
LVIDs	1.83	0.27	1.80	1.36	0.30	1.37	1.33	0.28	1.31	**0.000**
LVPWs	0.92	0.25	0.85	0.76	0.16	0.77	0.74	0.14	0.73	**0.009**
LVd mass	93.87	38.57	80.22	80.12	16.31	78.42	77.43	23.10	75.15	0.575
LVs mass	74.27	24.08	65.42	59.84	15.68	60.17	55.71	16.67	49.59	**0.039**
LVd mass	81.60	30.80	70.16	72.26	12.92	71.31	68.58	17.74	65.92	0.482
LVs mass	62.91	19.53	56.81	61.23	13.98	57.34	50.97	13.12	48.95	0.077

DISCUSSION

Metabolic syndrome (MS) was first described by Reaven in 1988 under the name Syndrome X to highlight the relationship between insulin resistance and the increased risk of lipid disorders, hypertension, type 2 diabetes, and atherosclerotic heart diseases in adults. While metabolic syndrome is primarily recognized as a health problem in adults, it has recently emerged as a significant issue in children and adolescents. The rise in obesity prevalence among these age groups has contributed to an increased frequency of metabolic syndrome. Similar to adults, obesity and insulin resistance are largely responsible for the pathogenesis of metabolic syndrome in childhood.

Hypertension (HT) and obesity represent important health challenges with rising prevalence and incidence worldwide. Although studies documenting the increase in HT prevalence among children are limited, regional research indicates that hypertension rates may be rising in this population. The foundation of systemic arterial hypertension, a significant health issue in adults, begins in childhood and can be identified through blood pressure measurements at that age (10). Routine blood pressure assessments in healthy children during hospital visits facilitate the early detection of essential hypertension and the timely diagnosis of asymptomatic hypertensive patients who may develop hypertension secondary to other underlying

conditions. Studies across various countries report that hypertension prevalence ranges from 1% to 22% (11-13). In our country, reported prevalence figures have varied between 3.63% and 17.8% (14).

In a study involving 2,478 school children aged 12 to 14 in Bursa, Akış et al. found a positive correlation between hypertension prevalence and weight gain. The same study identified be-ing overweight as a significant factor influencing HT prevalence (15). In our research, we found that age, anthropometric measurements, body mass indices, and blood pressure readings were statistically significantly higher in children with obesity. In their study, Tadahisa et al. performed echocardiography on 37 obese patients and examined the relationship between left ventricular functions and abdominal fat accumulation. They found that left ventricular end-diastolic volume was significantly higher in obese patients compared to the control group. This study indicated that this increase was directly proportional to body weight. The reason for this has been attributed to the increase in fat mass, which causes hypervolemia and an associated increase in cardiac output. It is thought that the increase in left ventricular diameter is a physiological adaptation (16). In a study involving 40 normal individuals and 40 obese patients without secondary damage, Gian et al. found that both left ventricular end-diastolic volume and left ventricular end-systolic volume were significantly higher in obese patients compared to healthy children (17). In the early stages of obesity, left ventricular hypertrophy develops as a consequence of increased blood pressure and left ventricular wall tension due to elevated blood volume. The long-term persistence of obesity leads to increased volume and/or pressure load, resulting in left ventricular dilation, eccentric hypertrophy, and both systolic and diastolic dysfunction. In obese patients, an increase in fatty acid oxidation and myocardial oxygen consumption can occur due to decreased glucose utilization secondary to insulin resistance. It is believed that the increase in adipose tissue may impact the heart's conduction system by causing myocardial atrophy due to pressure on the myocardium. Additionally, lipotoxicity and damage caused by the secretion of adipokines and increased triglyceride levels in myocytes may lead to deterioration in the heart's systolic function (18).

In our study, the comparison of echocardiographic findings between the patient and control groups revealed statistically significant increases in various measurements: interventricular septum diastolic diameter (IVSd), left ventricular diastolic diameter (LVIDd), left ventricular free wall diastolic diameter (LVPWd), interventricular septum systolic thickness (IVSs), left ventricular systolic diameter (LVIDs), and left ventricular free wall diastolic thickness (LVPWs) were all notably higher in the patient group. Supporting our findings, Van Putte-Katier et al. conducted a study on 49 obese children and reported that left ventricular wall sizes—particularly IVSd, LVIDd, and left ventricular posterior wall thickness—were greater in obese cases compared to controls (19). Similarly, Erdem et al. found elevated left ventricular measurements (including LVIDs, IVSd, and LV mass) in obese children, with IVSd particularly heightened in those with both hypertension and insulin resistance (20). Further analysis in our study showed a negative weak correlation between systolic blood pressure and LVIDs (p=0.042, r=-0.373), alongside positive moderate correlations between systolic blood pressure and left ventricular diastolic mass (LVd mass) (p=0.009, r=0.467) and left ventricular systolic mass (LVs mass) (p=0.002, r=0.543). Additional measurements, including IVSd, IVSs, LVIDd, LVIDs, LVPWd, and LVPWs, were also significantly higher in the patient group. The relationship between obesity and chronic inflammation is well-documented. Obesity is linked to increased levels of inflammatory cytokines, contributing to insulin resistance and hypertension as part of metabolic syndrome. Researchers have indicated that the rise in blood pressure can be influenced by bioactive substances secreted from adipose tissue, such as leptin, angioten-sinogen, interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and adiponectin (21,22). In a study by Garanty et al., examining serum markers in 50 children with obesity-related hypertension and 143 obese children with normal blood pressure, they found significantly elevated levels of inflammatory markers and endothelial activation indices in hypertensive children. This suggests that low-grade inflammation and endothelial dysfunction are closely related to the pathogenesis of obesity-associated hypertension at an early age (23). In our own study, we identified a positive moderate correlation between leptin and systolic blood pressure in healthy children (p=0.019, r=0.425). Conversely, we observed a negative moderate correlation between Pro-BNP and systolic blood pressure in obese and hypertensive children (p=0.001, r=-0.566), alongside a positive weak correlation between leptin and diastolic blood pressure (p=0.036, r=0.384). These findings highlight the significant interplay between obesity, inflammation, and hypertension in pediatric populations.

In Our study, statisticaly significant correlation was found between leptin and echocardiography results of the patient and control group. Negative moderate correlation was found between left ventricular diastolic mass (LVd mass) (p=0.032 r=-0.392) and left ventricular systolic mass (LVs mass) (p=0.006 r=-0.492) and Pro-BNP in the patient group. Negative weak correlation was found between left ventricular diastolic diameter (LVIDd) (p=0.015 r=-0.441), left ventricular systolic diameter (LVIDs) (p=0.025 r=-0.410) and Pro-BNP in the pa-tient group. When the correlation between Pentraxin-3 and echocardiographic measurements was evaluated, no statistically significant correlation was found.

In both previous studies and our own, elevated inflammatory markers have been consistently observed in obese individuals with hypertension. This underscores the critical role of obesity-induced inflammation as a significant factor contributing to hypertension. Our study revealed a moderate positive correlation between leptin and pentraxin-3 in obese children (p=0.023, r=0.413). Additionally, we found a moderate positive correlation between leptin and systolic blood pressure in healthy

children. In the patient group, a moderate negative correlation was identified between Pro-BNP and systolic blood pressure (p=0.001, r=-0.566), while a weak positive correlation was found between leptin and diastolic blood pressure (p=0.036, r=0.384). Supporting our findings, Duprez et al. demonstrated a significant relationship between CRP levels and early atherosclerosis, while Hommels et al. noted a moderate association between CRP and atherosclerosis in the abdominal aorta and renal arteries. Together, these studies suggest that the vascular effects of inflammation, indicated by elevated high-sensitivity CRP (hsCRP) levels, may lead to decreased elasticity and increased stiffness in large arteries, contributing to atherosclerosis development. The pro-inflammatory processes, initiated by mediators such as adhesion molecules, chemokines, growth factors, heat shock proteins, endothelin-1, and angiotensin, highlight the potential for inflammation to drive both the initiation and progression of hypertension. Increased levels of cytokines can hinder the production of endothelium-derived vasodilators, particularly nitric oxide (NO), which can result in endothelial dysfunction, sustained impaired vasodilation, and ultimately hypertension (24,25). Inflammation has an important role in the occurrence and prognosis of cardiovascular diseases. Hypertension is one of the most frequent systemic diseases. Inflammation has an important role in the pathogenesis of hypertension and contributes to the formation of target organ damage due to hypertension (26). In a study conducted by Wang et al., it was found that losing weight with a four-week long healthy diet and exercise caused a significant decrease in inflammation in overweight adolescents (27). Patients with high inflammation parameters may need more medical treatment in addition to lifestyle changes. In our study, leptin, systolic blood pressure, diastolic blood pressure, height, weight and body mass index, IVSd, LVIDd, LVPWd, IVSs, LVIDs, LVPWs and LVs mass values were found to be statistically significantly high in obese patients who were started anti-hypertensive treatment.

The aim of our study was to evaluate the levels of CRP, IL-1, proBNP, leptin, and PTX-3 to assess inflammation in children with obesity-related hypertension. Our findings indicated that CRP and leptin levels were significantly higher in the patient group compared to controls, while no significant differences were observed for Pentraxin-3, IL-1, and proBNP levels. These elevated levels of CRP and leptin suggest their potential roles as markers of inflammation in obese and hypertensive children. Early diagnosis and intervention are crucial to prevent complications associated with hypertension. We believe that monitoring these inflammatory markers can provide insights into the severity of hypertension and help in preventing organ damage, as well as in the reversal of hypertension through stricter lifestyle changes, diet, and exercise.

CONCLUSION

In conclusion, our study demonstrates that CRP and leptin are significantly higher in the patient group, supporting their involvement in the pathogenesis of obesity-associated hypertension. These markers could guide future strategies aimed at reversing inflammation and hypertension through lifestyle modifications. Given that hypertension is fundamentally an inflammatory disease, this understanding may pave the way for new therapeutic approaches to mitigate morbidity and mortality associated with hypertension and its related organ damage. However, further prospective studies are necessary to evaluate the long-term effects and outcomes of these interventions.

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Ethics Committee Approval: Prior to the study, ethical approval was obtained from the İnönü University Faculty of Medicine Health Sciences Clinical Research Ethics Committee, and consent was secured from the participants or their parents.

Patient Informed Consent: The study was conducted with the consent of all patients.

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