Research article

Pulse Wave Analysis in Obese Children with and without Metabolic Syndrome

BAŞARAN C et al. Pulse Wave Analysis in Children with Metabolic Syndrome

Cemaliye Başaran¹, Gökçen Erfidan¹, Özgür Özdemir-Şimşek¹, Seçil Arslansoyu-Çamlar², Demet Alaygut², Fatma Mutlubaş³, Cem Karadeniz³, Bumin Nuri Dündar⁴, Belde Kasap-Demir¹,⁵¹
¹University Health Sciences, İzmir Tepecik Training and Research Hospital, Department of Pediatrics, Division of Nephrology, İzmir, TURKEY
²University Health Sciences, İzmir Faculty of Medicine, Department of Pediatrics, Division of Nephrology, İzmir, TURKEY
³Izmir Katip Celebi University, Faculty of Medicine, Department of Pediatrics, Division of Cardiology, İzmir, TURKEY
⁴Izmir Katip Celebi University, Faculty of Medicine, Department of Pediatrics, Division of Endocrinology, İzmir, TURKEY
⁵Izmir Katip Celebi University, Faculty of Medicine, Department of Pediatrics, Division of Nephrology & Rheumatology, İzmir, TURKEY

What is already known on this topic?
In the latest arterial HT guidelines published for adult patients, PWA is recommended because of its high predictability, easy applicability, and reproducibility in determining cardiovascular risk. Study on pulse wave velocity in children and adolescents with metabolic syndrome is limited.

What this study adds?
This study showed that additional risk factors other than obesity, which are required for the diagnosis of MS, further increase 24-hour and daytime cSBP and cDBP. Therefore, PWA analysis is needed to better determine cardiovascular risk and target organ damage in children with MS.

ABSTRACT
Objective: We aimed to compare the pulse wave analysis (PWA) of obese children with and without metabolic syndrome (MS) with healthy non-obese children and to evaluate the reflections of additional risk factors that children with MS have in addition to obesity on PWA.

Methods: Among all obese patients examined between June 2019 and June 2021, 41 patients with MS, 36 obese patients without MS, and 34 healthy non-obese children of similar age and gender were evaluated retrospectively. Anthropometric measurements, biochemical evaluations, 24-hour ambulatory blood pressure measurement (ABPM), left ventricular mass index (LVMI) and PWA measurements were compared.

Results: When the three groups were compared, weight SDS, height SDS and BMI SDS; it was found to be significantly higher in the MS group (p<0.05). In ABPM measurements, the systolic and MAP blood pressure (BP) SDSs load; in PWA, night central systolic BP, 24-hour, day and night pulse pressure values and 24-hour, day and night pulse wave velocity (PWV) rates; in cardiac evaluations, LVMI and relative wall thickness measurements were significantly higher in MS and non-MS obese patients compared to the control group (p<0.05). 24-hour and daytime central systolic and diastolic BP values were significantly different in 3 groups, the highest in MS patients (p<0.05).

Conclusion: Obesity causes higher office, ambulatory and central BP, PWV and LVMI, however additional risk factors leading to MS do not contribute to these parameters except for 24-hour and daytime cSBP and cDBP values.

Keywords: Children, pulse wave analysis, metabolic syndrome, obesity
INTRODUCTION
Metabolic syndrome (MS) is a cluster of medical problems that put patients at risk for cardiovascular diseases. There are different definitions proposed by different research groups for the definition of MS in children (1). All of these definitions include high body mass index (BMI) and waist circumference measurement, high triglyceride and low HDL cholesterol levels, high blood pressure (BP) and high fasting blood glucose or high fasting insulin level. The International Diabetes Federation (IDF) recommends that definitions be evaluated separately according to age groups due to age-related variability in children (2). The most important reason for the increase in the frequency of metabolic syndrome (MS) in children is the increase in the prevalence of obesity (3). It is known that obesity carries a risk for cardiovascular diseases, and the risk increases even more in the presence of MS due to additional components.

The most common target organ damage seen in both adults and children with hypertension (HT) is an increase in left ventricular mass and increased carotid intima-media thickness (4,5). The concept of arterial stiffness (AS) has also emerged in recent years as a strong independent predictor of cardiovascular events. Arterial stiffness (AS) is used for the viscoelastic property of the vessel wall, is also a strong independent indicator of cardiovascular events (6). Pulse wave analysis (PWA), which evaluates the compliance and wave reflections created by the pressure created on the vessel wall during the progression of the pulse wave originating from the aortic arch to the periphery, is the most commonly used method to evaluate arterial stiffness (7,8). In case of increased arterial stiffness, the higher the central systolic BP (sBP) central diastolic BP (cDBP) increases, central pulse pressure (sPP) rises, and left ventricular mass and load increase (9).

In PWA, an idea about vascular structures can be obtained by looking at parameters such as pulse wave velocity (PWV) and augmentation index (AIx) (10). PWV is the rate of passage of the pulse wave between two points in the arterial system (11). The most important factors affecting PWV are age and BP. It has been shown to be high in chronic diseases such as kidney failure, obesity and diabetes mellitus (12,13). In cases with increased AS the back reflection wave reaches the aortic root earlier than diastole since the PWV is high. Adding to the forward wave causes an increase in the amplitude of the wave and the systolic pressure. This percentage of increase is expressed as the AIx (14). High AIx values are associated with increased PWV. In other words, arterial stiffness. AIx is a normalized index for a rate of 75 beats per minute due to heart rate differences (AIx@75). In the latest arterial HT guidelines published for adult patients, PWA is recommended because of its high predictivity, easy applicability and reproducibility in determining cardiovascular risk (15).

Our aim in this study was to compare the PWA of obese children with and without MS with healthy non-obese children and to evaluate the effects of additional risk factors that children with MS have in addition to obesity.

METHODS
Study population
The protocol of this single-center cross-sectional study was approved by the ethics committee of our hospital (2021/10-09). In our study, the results of ABPM and PWA performed in obese (BMI ≥ 95 percentile) children with and without MS and non-obese (BMI < 85 percentile) children who applied to the pediatric outpatient clinic between June 2019 and June 2021 were analyzed retrospectively. Patients aged between 10 and 18 years (because it is difficult to diagnose MS at <10 years of age) and taller than 120 cm (as ABPM data was created for children with a height of 120 cm and above) were included in the study. Patients with missing data, those who dropped out, or had an additional chronic disease such as cardiac disease, chronic kidney disease, hyperthyroidism or hypothyroidism, etc., were not included in the study. The data of healthy non-obese children were evaluated as the control group.

Anthropometric measurement
Weight measurement was carried out by removing all the thick clothes and shoes on the child, on digital scales used for adults, preferably sensitive to 100 g weight change. Height was measured while the child was standing. The measurement was performed on a hard surface, barefoot and without a hat on, with the child's back facing the measuring instrument, with the most protruding part of the head, shoulders, hips and heels in full contact with the measuring instrument, with the arms hanging down, the heels together, and the head straight. The hard flat and moving head of the measuring instrument was partially touched to the upper part of the head, and this point was read from the scale and the height was determined. Weight and height percentiles were calculated using our national reference data (16). BMI was calculated as the ratio of body weight (kg) to height squared (m²). The standard deviation score (SDS) of BMI was calculated with the help of the Child Metrics program (17). In the BMI reference curve, which was prepared for Turkish children and adjusted for age and gender, those with BMI values between the 85th percentile and the 95th percentile were defined as "overweight", and those above the 95th percentile were defined as "obese" (16).

Waist circumference was measured at the end of expiration from the midpoint between the lower edge of the last rib and the apex of the iliac crest while standing comfortably with the feet approximately 25-30 cm apart. Waist circumference percentiles were evaluated according to percentiles calculated for Turkish children (18). The presence of puberty was evaluated as having physical examination findings compatible with at least Tanner 2 (19,20).
The criteria recommended by The International Diabetes Federation (IDF) were used for the diagnosis of MS (2).

Biochemical tests
Fasting glucose, creatinine, uric acid, sodium, potassium, alanine aminotransferase (ALT), free T4, thyroid stimulating hormone (TSH), insulin levels and lipid profiles (total cholesterol, HDL cholesterol and triglycerides) were measured in blood samples taken in the morning after overnight fasting. Blood glucose levels were measured by the glucose oxidase method and serum lipid profiles were measured using routine enzymatic methods. Insulin measurements were made by the immunofluorometric method. An oral glucose tolerance test (OGTT) was performed to detect insulin resistance (IR). 1.75 g/kg (maximum 75 g) glucose was administered orally, and blood samples were taken for glucose and insulin measurements at 0, 30, 60, 90 and 120 minutes (21). HOMA-IR was calculated via Child Metrics according to the formulas: fasting plasma glucose (mg/dL) * fasting plasma insulin (μU/mL) / 405 (17). HOMA-IR levels >2.5 for the prepubertal and >4 for the pubertal period were defined as IR (22).

Office BP measurements
Office BP was measured three times at 2-minute intervals on the right arm, after 5 minutes of rest, with aneroid devices calibrated by an experienced nurse, with cuffs appropriate for the child's age, and the last two BPs were averaged for analysis. Office systolic BP (SBP) and diastolic BP (DBP) measurements of all patients were evaluated according to the American Academy of Pediatrics 2017 (AAP-2017) HT guideline (23).

ABPM measurements and PWA
Office and ambulatory BP and central BP measurements of the patients were evaluated with an oscillometric PWA-ABPM device (Mobil-O-Graph; IEM, Stolberg, Germany) (24). Daytime measurements were made at 15-minute intervals, and nighttime measurements were performed at 30-minute intervals. Results of the measurement; skewness (L), median (M) and coefficient of variation (S) were converted to SDSs by the LMS method. SDSs were calculated automatically with the help of the Child Metrics program according to the published reference LMS tables for healthy children (17, 25). The ratio of BP values above the ambulatory 95th percentile was defined as “BP load”. Patients with <10% reduction in BP at night compared to the daytime period were defined as 'dipper'. In ABPM measurements, measurements above the 95th percentile were accepted as HT (23).

Variables measured during PWA
24-hour, daytime and nighttime SBP (mmHg), 24-hour, daytime and nighttime DBP (mmHg), 24-hour, daytime and nighttime mean arterial pressure (mmHg) (MAP) = [(SBP+2 DBP) / 3], daytime and nighttime systolic load (%), daytime and nighttime diastolic load (%), systolic and diastolic dip, 24-hour, daytime and nighttime cSBP (mmHg), 24-hour, daytime and nighttime cDBP (mmHg), 24-hour, daytime and nighttime cPP (mmHg), [cPP = cSBP – cDBP], 24-hour, daytime and nighttime PWV (m/s), 24-hour, daytime and nighttime the AIX standardized according to 75 heart rate is (AIX@75) values were obtained (10). Central BP indicates BP at the aortic root and is usually lower than the brachial artery measurement (26).

Echocardiographic assessment
All children included in the study were evaluated by the same pediatric cardiologist with the same echocardiography device. Left ventricular mass index (LVMI) was calculated according to the LVM calculated via Devereux formula and indexed to height (m².7). LVM (gram): 0.8 × 1.04 [(LVEDD + IVST + PWT) − (LVEDD)] + 0.6 [LVEDD; left ventricular end-diastolic diameter, IVST: interventricular septum thickness, PWT: posterior wall thickness] (27,28). LVH defines an LVMI that exceeds the 95th percentile for sex and age in normal children and adolescents (28). The left ventricular hypertrophy index (LVHI) was obtained by dividing the LVMI by the 95th percentile for that age and gender. The relative wall thickness (RWT) was calculated by the formula: RWT = 2 × PWtd/LVIDed (29).

Statistical Analysis
All statistical evaluations were performed using SPSS for Windows 24.0. Discrete variables are expressed as counts (percentage), continuous variables with normal distribution were calculated as mean ± standard deviation, and continuous variables with non-normal distribution as median (interquartile range; 25-75%). The Kruskal-Wallis test was used to evaluate the distribution of continuous variables among groups. Normal distributed variables were evaluated with the Anova Test and post-hoc analyses were performed with the Tukey Test in homogeneous groups and with Tamhane’s T2 Test in heterogeneous groups. Without normal distribution, variables were evaluated first with the Kruskal-Wallis Test between the 3 groups and then with the Mann-Whitney U Test to determine the group that caused the difference. Depending on the distribution type of the variable, Pierson or Spearman analysis was performed. P<0.05 was considered statistically significant for all statistical evaluations.

RESULTS
Thirty-six of the 111 patients included in the study were non-MS obese and 41 patients had MS. There were 34 healthy non-obese children in the control group (Figure 1).
The mean age and gender were similar between the three groups. The characteristics of the demographic and laboratory findings of the study population are given in Table 1. Height SDS was significantly higher in the MS group compared to the other two groups. Weight SDS and BMI SDS were significantly different between the three groups. Uric acid, total cholesterol, ALT, TSH levels were similar in the MS and non-MS obese groups, but significantly lower in the control group. Triglyceride levels were significantly higher in the MS group compared to the other two groups. HOMA-IR was significantly higher in the MS group than in the non-MS obese group. All children were euthyroid.

The office BP measurement and 24-hour ABPM results were compared in Table 2. Office SBP SDS values, 24-hour, daytime and nighttime SBP SDS values, 24-hour, daytime and nighttime MAP SDS values, daytime and nighttime systolic load, nighttime diastolic load were similar in the MS and the non-MS obese groups, which were higher than the control group. Office DBP SDS values and daytime DBP load were significantly higher in the MS group than the other two groups. 24-hour, daytime and nighttime DBP SDS and systolic and diastolic dip rates were similar in all groups.

When PWA data were evaluated, 24-hour and daytime cSBP values were higher in the MS group compared to the other two groups. 24-hour and daytime cDBP values were higher in the MS group than the control group, but similar between the MS and non-MS obese patients. Nighttime cSBP values, 24-hour, daytime and nighttime pulse pressure values, 24-hour, daytime and nighttime PWV values were similar in the groups with MS and in the non-MS obese group and were higher than the control group (Figure 2). 24-hour, daytime and nighttime PWV were significantly correlated with BMI SDS (r:0.356, p: 0.001; r: 0.37 p: 0.001; r: 0.345 p: 0.005, respectively). LVMi, LVMi/95P and RWT measurements were significantly higher in the MS and non-MS obese groups than the control group (Table 3). LVMi, LVMi/95P and RWT measurements were higher in the MS group than those of the non-MS obese group, although they were not statistically significant.

**DISCUSSION**

In our study, we have found that both 24-hour and daytime cSBP and cDBP were higher in the MS group than the other two groups. In addition, we have found that LVH, as an indicator of end-organ damage, was more frequent in the non-MS obese and MS groups compared to the control group, whereas it was similar between the two groups. Considering that 17% of the non-MS obese group was hypertensive and 61% of the MS group was hypertensive, we think that LVH in these patients is not affected by high BP, but by existing obesity status. High HOMA-IR and triglyceride levels are expected in MS. However, obesity caused the difference in uric acid, total cholesterol, ALT and TSH values. Free T4 and TSH values were within normal ranges in all of our patients, and none of our patients had hypothyroidism or hyperthyroidism.

The Mobil-O-Graph device used to assess cSBP in children and adolescents has been shown to perform well compared to simultaneous invasive recordings (30). In recent years, it has been found that central BP measurement is superior in determining cardiovascular risk, especially in young adults (31). Studies in hypertensive adults have shown that cardiovascular mortality and hypertensive target organ damage are more correlated with cSBP and cPP than with brachial arterial BP (32,33). In children, because the brachial artery is more elastic, accumulation of pulse waves results in higher brachial BP measurements, but central BPs are within normal ranges. Therefore, central BP measurement in children and young adults may prevent the detection of more HT with peripheral ABPM measurements. A study by Tatora et al showed that adults with higher cSBP had higher cIMT, LVMi, PWV, and lower brachial artery dilatation. However, patients with higher cSBP had a significantly higher BMI (mean BMI 38.7), and 42.9% of them had type 2 diabetes mellitus (34). Few studies have been conducted in childhood. Litwin et al. found significantly higher cIMT, LVMi, and PWV values in adolescents with both younger age and lower BMI and higher cSBP. They also showed that children with primary HT with severe ambulatory HT had normal central BP values and determined that central BP measurements had the same or higher power as ABPM in predicting end-organ damage (35). In our study, 24-hour and daytime cSBP and cDBP measurements were higher in the MS group. LVMi, a marker of target organ damage, was similar in the obese group with and without MS. However, it was higher than the control group.

Since the rate of obesity, IR and cardiac disease is gradually increasing in children and adolescents, it is necessary to screen children in the risk group to prevent atherosclerosis that may develop in later ages (36). When we look at the literature, there are many conflicting reports regarding the change in PWV in children obesity alone. In some studies, it has been shown that there is an increase in PWV with obesity (37,38). In contrast, some studies have shown a “paradoxical” decrease in PWV with obesity (39). The paradoxical reduction has been attributed to precocious puberty and increased body size in obese children (40). Another hypothesis is that the decrease in PWV is a short-term adaptation, does not continue in the long term, and increases in time in longitudinal studies (41). In our study, we found that the PWV was similar in the MS and the non-MS obese groups, but it was higher than the control group. PWV increased as BMI SDS increased. Although HOMA IR was significantly higher in the MS group when compared to the non-MS obese group, PWV was similar between these groups. The effects of IR may not be reflected to the PWV yet because of the cross-sectional design of our study.
In a recently published study, it was shown that 24-hour cSBP and 24-hour PWV values in obese children and adolescents were higher in obese subjects, but 24-hour AIX@75 values were not different from non-obese subjects. These results show similar features to our study. In obese cases, the total blood volume and thus the stroke volume increase, whereas the total peripheral resistance decreases, the heart rate remains within the normal range or slightly increases keep the BP at normal levels. As a result of this situation, augmentation pressure and AIX@75 values remain low in obese cases (42). In our study, although central and peripheral values were higher in obese subjects than in non-obese subjects, AIX@75 values were similar between the groups. According to our knowledge, our study is the first study in which the PWA of children with and without MS was evaluated using the oscillometric technique, however it has limitations such as cross-sectional and retrospective design. Considering that PWV increases with age, performing longitudinal studies allows a better evaluation. The relatively small number of patients may also have caused differences in the analysis of the study results.

CONCLUSION
ABPM data were similar to the non-MS obese group, with slightly higher rates in the MS group. Both 24-hour and daytime central systolic and diastolic BP measurements were higher in the MS group. Obesity causes higher office, ambulatory, and central BP, PWV, and LVMi, but the additional risk factors in addition to obesity in MS do not contribute to these parameters, except for 24-hour and daytime cSBP and cDBP.

Authors' contributions: Surgical and Medical Practices: Cemaliye BAŞARAN, Belde KASAP-DEMİR, Gökçen ERFİDAN and Özgür ÖZDEMİR-ŞİMŞEK Concept: Cemaliye BAŞARAN, Belde KASAP-DEMİR

Data Collection or Processing: Cemaliye BAŞARAN, Gökçen ERFİDAN and Özgür ÖZDEMİR-ŞİMŞEK

Analysis or Interpretation: Belde KASAP-DEMİR, Fatma MUTLUBAŞ, Demet ALAYGUT, Seçil ARSLANSOYU-ÇAMLAR, Bumin Nuri DUNDAR, Cem KARADENIZ, Cemaliye BAŞARAN

Literature Search: Cemaliye BAŞARAN, Fatma MUTLUBAŞ, Demet ALAYGUT and Seçil ARSLANSOYU-ÇAMLAR

Writing: Cemaliye BAŞARAN, Belde KASAP-DEMİR

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hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC), J. Hypertens. 31 (7) (2013) 1281e1357.


Figure 1. Study sample selection

- Obese children $n=87$
- Non-obese children $n=40$

Missing data
- 3 ABPM data missing
- 4 laboratory data missing
- 1 operated ASD
- 2 chronic kidney disease

Missing data
- 2 ABPM data missing
- 1 laboratory data missing
- 3 overweight children

Non-MS obese group $n=36$

MS group $n=41$

Control group $n=34$
Figure 2. Comparison of pulse wave analysis (PWA) data.

*: The results of all three groups were statistically different from each other.

**: Control group was significantly lower than the MS and non-obese MS groups.

***: MS group was higher than the control group.

Table 1: Comparison of demographic and laboratory findings.

<table>
<thead>
<tr>
<th></th>
<th>non-MS obese group</th>
<th>MS group</th>
<th>control group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>12.50 (11.00 - 15.00)</td>
<td>14.00 (12.00 - 15.00)</td>
<td>13.00 (11.00 - 15.25)</td>
<td>0.734</td>
</tr>
<tr>
<td>Male</td>
<td>14 (%38.90)</td>
<td>10 (%24.40)</td>
<td>13 (%38.20)</td>
<td>0.301</td>
</tr>
<tr>
<td>Weight SDS</td>
<td>2.76 ± 1.08</td>
<td>3.60 ± 1.06</td>
<td>-0.13 ± 1.15</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Height SDS</td>
<td>0.31 (-0.77 - 1.06)</td>
<td>0.94 (-0.07 - 2.00)**</td>
<td>-0.13 (-0.88 - 0.87)</td>
<td>0.002</td>
</tr>
<tr>
<td>BMI SDS</td>
<td>2.61 (2.10 - 3.10)</td>
<td>2.88 (2.64 - 3.41)</td>
<td>-0.15 (-1.29 - 0.80)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Glucose</td>
<td>88.20 ± 7.73</td>
<td>88.19 ± 7.90</td>
<td>89.46 ± 9.55</td>
<td>0.770</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.60 (0.60 - 0.70)</td>
<td>0.70 (0.60 - 0.70)</td>
<td>0.60 (0.59 - 0.70)</td>
<td>0.179</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>5.30 (4.50 - 5.80)</td>
<td>5.60 (5.07 - 6.82)</td>
<td>4.00 (3.50 - 4.67)**</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>103.17 ± 27.79</td>
<td>132.86 ± 61.00**</td>
<td>90.77 ± 39.93</td>
<td>0.005</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>175.30 ± 31.60</td>
<td>171.00 ± 33.80</td>
<td>150.25 ± 38.20**</td>
<td>0.019</td>
</tr>
<tr>
<td>ALT (IU/l)</td>
<td>23.00 (17.00 - 34.00)</td>
<td>22.00 (14.50 - 40.50)</td>
<td>12.00 (10.00 - 15.00)**</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Free T4 (mIU/L)</td>
<td>0.78 (0.71 - 0.89)</td>
<td>0.81 (0.72 - 0.94)</td>
<td>0.86 (0.78 - 0.91)</td>
<td>0.087</td>
</tr>
<tr>
<td>TSH (ng/dl)</td>
<td>2.40 (1.95 - 2.95)</td>
<td>2.32 (1.73 - 3.40)</td>
<td>1.80 (1.14 - 2.26)**</td>
<td>0.044</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.10 (2.40 - 4.40)</td>
<td>4.43 (3.07 - 6.18)</td>
<td>8.49 (4.62 - 21.95)</td>
<td>0.008*</td>
</tr>
<tr>
<td>Microalbumin/creatinine (mg/g)</td>
<td>5.57 (3.02 - 21.52)</td>
<td>7.00 (5.00 - 16.80)</td>
<td>8.49 (4.62 - 21.95)</td>
<td>0.423</td>
</tr>
</tbody>
</table>

*: The results of all groups were statistically different from each other.

**: The results were significantly different from the other two groups.
Table 2: Comparison of office and 24-hour blood pressure data.

<table>
<thead>
<tr>
<th></th>
<th>non-MS obese group</th>
<th>MS group</th>
<th>control group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Office systolic BP</strong></td>
<td>1.13 [ (0.77) - (2.00)]</td>
<td>1.88 [ (0.46) - (2.33)]</td>
<td>0.20 [ (-0.64) - (1.08)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Office diastolic BP</strong></td>
<td>0.95 ± 0.86</td>
<td>1.15 ± 0.91</td>
<td>0.57 ± 0.68</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>24-h systolic BP</strong></td>
<td>-0.02 ± 0.99</td>
<td>0.28 ± 1.17</td>
<td>-1.14 ± 0.74</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Daytime systolic BP</strong></td>
<td>-0.35 ± 1.00</td>
<td>-0.08 ± 1.08</td>
<td>-1.36 ± 0.83</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Nighttime systolic BP</strong></td>
<td>0.69 [ (0.20) - (1.53)]</td>
<td>0.76 [ (0.11) - (1.79)]</td>
<td>-0.34 [ (-0.59) - (0.35)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>24-h MAP</strong></td>
<td>-0.02 ± 0.99</td>
<td>0.28 ± 1.17</td>
<td>-1.14 ± 0.74</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Daytime MAP</strong></td>
<td>0.38 ± 1.05</td>
<td>0.71 ± 1.19</td>
<td>-0.30 ± 0.71</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Nighttime MAP</strong></td>
<td>1.55 [ (1.07) - (2.49)]</td>
<td>1.66 [ (1.06) - (2.83)]</td>
<td>0.84 [ (0.32) - (1.43)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Daytime systolic load (%)</strong></td>
<td>13.00 [ (6.00) - (21.25)]</td>
<td>19.00 [ (9.50) - (34.50)]</td>
<td>3.50 [ (0.00) - (7.25)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Nighttime systolic load (%)</strong></td>
<td>23.00 [ (9.75) - (44.25)]</td>
<td>33.00 [ (8.00) - (56.50)]</td>
<td>1.00 [ (0.00) - (12.75)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Daytime diastolic load (%)</strong></td>
<td>8.50 [ (4.00) - (16.50)]</td>
<td>14.00 [ (6.50) - (22.00)]</td>
<td>7.00 [ (3.50) - (10.50)]</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Nighttime diastolic load (%)</strong></td>
<td>19.50 [ (8.25) - (38.00)]</td>
<td>15.00 [ (3.00) - (38.00)]</td>
<td>5.50 [ (0.00) - (15.50)]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Systolic dip</strong></td>
<td>7.35 [ (0.02) - (10.42)]</td>
<td>6.40 [ (3.17) - (10.50)]</td>
<td>5.55 [ (3.07) - (10.32)]</td>
<td>0.81</td>
</tr>
<tr>
<td><strong>Diastolic dip</strong></td>
<td>12.30 [ (6.57) - (14.37)]</td>
<td>11.70 [ (5.95) - (18.85)]</td>
<td>13.95 [ (6.70) - (20.30)]</td>
<td>0.53</td>
</tr>
</tbody>
</table>

*: The results were significantly different from the other two groups.

Table 3: Comparison of echocardiographic findings between the groups.

<table>
<thead>
<tr>
<th></th>
<th>non-MS obese group</th>
<th>MS group</th>
<th>control group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LVMI (g/m²)</strong></td>
<td>35.85 (33.17 - 39.62)</td>
<td>37.85 (30.85 - 44.95)</td>
<td>31.55 (26.30 - 36.80)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>LVMI/95P</strong></td>
<td>0.95 (0.83 - 1.05)</td>
<td>0.97 (0.84 - 1.19)</td>
<td>0.82 (0.70 - 0.95)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>RWT</strong></td>
<td>0.40 ± 0.70</td>
<td>0.41 ± 0.68</td>
<td>0.34 ± 0.11</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

LVMI: Left ventricular mass index, RWT: relative wall thickness
*: The results were significantly different from the other two groups.