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ORIGINAL ARTICLE

Comparison of myocardial performance index and right ventricular myocardial acceleration during isovolumic contraction in detection of right ventricular dysfunction in obese patients

Obez hastalarda sağ ventriküler disfonksiyonunun saptanmasında miyokardiyal performans indeksi ile isovolumetrik kasılma fazındaki sağ ventriküler akselerasyonun karşılaştırılması

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ABSTRACT

Objective: Although obesity is a risk factor for heart failure, studies analyzing the effect of obesity on heart functions have primarily examined the left side of the heart. This study is an analysis of the effect of the severity of obesity on right heart functions and a comparison of the sensitivity and specificity of different echocardiographic modalities in the detection of right heart dysfunction.

Methods: A total of 116 subjects were included and divided into 4 age- and sex-matched groups according to body mass index (BMI) values. Right heart functions were evaluated with transthoracic echocardiography.

Results: The right atrium (RA) diameter was significantly larger in the obese group (OBG) and the morbidly obese group (MOG); the right ventricle (RV) diameter was significantly larger only in the MOG. In the overweight group, the OBG, and the MOG, the RV isovolumic acceleration (R-IVA) was significantly lower (p=0.020; p<0.001; p<0.001, respectively) and the myocardial performance index (MPI) value was significantly higher (p=0.015; p<0.001; p<0.001, respectively). There was a strong positive correlation between the MPI and the BMI (r=0.833, p<0.001), and a moderate negative correlation between the R-IVA and the BMI (r=-0.547, p<0.001). A cut-off value of 30.45 kg/m^2 was associated with 93.3% sensitivity and 94.3% specificity in the prediction of RV systolic dysfunction defined by the MPI. A cut-off value of 30.50 kg/m^2 was associated with 76.7% sensitivity and 72.3% specificity in the prediction of RV systolic dysfunction defined by the R-IVA.

Conclusion: Obesity significantly affected right heart function and there was a significant correlation between the degree of obesity and right heart functional deterioration. The BMI could be used to predict RV systolic dysfunction.

ÖZET

Amaç: Obezitenin kalp yetersizliği için bir risk faktörü olduğu bilinmesine rağmen, bu konuda yapılmış çalışmaların çoğunda obezitenin sol kalp fonksiyonları üzerine etkisi incelenmiştir. Bu çalışmada, obezite derecesinin sağ kalp fonksiyonları üzerine etkisini incelemeyi, farklı ekokardiyografik modalitelerin sağ ventriküler disfonksiyonu belirlemedeki duyarlılık ve özgüllüğünü karşılaştırmayı amaçladık.

Yöntemler: Toplam 116 hasta çalışmaya dahil edildi. Dahil edilen hastalar beden kitle indeksine (BKİ) göre, yaş ve cinsiyet açısından eşleştirilmiş 4 gruba ayrıldı. Transtorasik ekokardiyografi ile sağ kalp fonksiyonları değerlendirildi.

Bulgular: Sağ atriyum (RA) çapı; obez ve morbid obez grupta, sağ ventriül (RV) çapı ise sadece morbid obez grupta anlamlı derecede daha büyüktü. Sağ ventriküler isovolumetrik akselerasyon (R-IVA); fazla kilolu gruptan itibaren anlamlı derecede daha düşüktü (sırasıyla, p=0.020; p<0.001; p<0.001). Miyokart performans indeksi (MPİ) ise fazla kilolu gruptan itibaren anlamlı derecede daha yüksekti (sırasıyla, p=0.015; p<0.001; p<0.001) MPİ ile BKİ arasında güçlü pozitif korelasyon r=0.833, p<0.001), R-IVA ile BKİ arasında ise orta derecede negatif korelasyon = (-0.547, p<0.001) mevcuttu. Reciever operator curve analizi BKİ 30.50 kg/m^2 kestirim değerinin R-IVA için %76.7 duyarlılık ve %72.3 özgüllük ile, BKİ 30.45 kg/m^2 kestirim değerinin ise MPİ için %93.3 özgüllük ve %94.3 duyarlılıkla RV sistolik disfonksiyonunu öngördürdüğünü göstermiştir.

Sonuç: Bu çalışmada, bilinen hastalığı olmayan sağlıklı bireylerde obezitenin sağ kalp fonksiyonlarını anlamlı derecede etkilediği ve obezite derecesi ile sağ kalp fonksiyonlarındaki bozulma arasında anlamlı korelasyon olduğu gösterilmiştir. Ayrıca BKİ'nin RV sistolik disfonksiyonunu öngörmede tek başına kullanılabileceği gösterilmiştir.

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Obesity, which is defined as a body mass index (BMI) value of 30 kg/m^2 or more, is a growing public health problem all around the world.[1] It is now becoming a global epidemic.[2] Due to a close relationship between obesity and several clinical conditions, including hypertension (HT),[3] diabetes mellitus (DM),[4] and atherosclerosis,[5] it is now accepted as one of the most common causes of mortality and morbidity. Over the past decades, our understanding of the pathophysiological outcomes of obesity for the cardiovascular system have advanced significantly.[6] Recent studies have shown that there is a strong association between obesity and hemodynamic changes that contribute to the impairment of ventricular function.[7] Obesity may lead to deterioration in cardiac functions, such as an increase in left ventricular (LV) mass, LV hypertrophy, and LV and left atrial (LA) dilatation.[8] In addition, it has been established that obesity is a risk factor for left-sided heart failure in both men and women.[9]

Most studies analyzing the effect of obesity on heart functions have focused on the left side of the heart. Although there has been some research investigating the relationship between obesity and right heart functions, the underlying mechanism is still not understood. This study was an analysis of the effect of obesity on right heart functions and a comparison of the sensitivity and specificity of different echocardiographic modalities in the detection of right ventricular (RV) dysfunction.

Abbreviations:

AcT	*Peak systolic acceleration time*
AUC	*Area under the curve*
BMI	*Body mass index*
BUN	*Blood urea nitrogen*
CAD	*Coronary artery disease*
CI	*Confidence interval*
DM	*Diabetes mellitus*
ECG	*Electrocardiography*
ePASP	*Estimated pulmonary artery systolic pressure*
ET	*Ejection time*
HDL	*High-density lipoprotein*
LA	*Left atrium*
LDL	*Low-density lipoprotein*
LV	*Left ventricle*
MOG	*Morbidly obese group*
MPI	*Myocardial performance index*
NWG	*Normal weight group*
OBG	*Obese group*
OR	*Odds ratio*
OWG	*Overweight group*
RA	*Right atrium*
R-IVA	*Right ventricular isovolumic acceleration*
R-IVV	*Right ventricular myocardial velocity during isovolumic contraction*
ROC	*Receiver operating curve*
RV	*Right ventricle*
TG	*Triglyceride*
TAPSE	*Tricuspid annular plane systolic excursion*
TTE	*Transthoracic echocardiography*

METHODS

Study population and design

In this observational cross-sectional study, subjects were selected from consecutive patients who were admitted to the hospital obesity outpatient clinic between March 7, 2019 and July 31, 2019. All of the patient medical data and medications were reviewed. Patients were excluded if there was a record of HT; DM; coronary artery disease (CAD); LV systolic dysfunction (LV ejection fraction <50%); moderate-to-severe valve disease; myocardial ischemia observed in any myocardial stress test, electrocardiography (ECG), or transthoracic echocardiography (TTE) examination; any cardiac rhythm problem; chronic obstructive pulmonary disease/asthma; congenital right heart disease; or pulmonary arterial HT (estimated pulmonary artery systolic pressure [ePASP] was calculated by the sum of the Doppler-derived transtricuspid gradient and the estimated right atrial [RA] pressure, as assessed by the inspiratory collapse of the inferior vena cava. An ePASP >40 mmHg was considered pulmonary arterial HT). A total of 116 patients were included after applying the exclusion criteria. The patients enrolled in the study were divided into 4 groups according to a BMI calculation. Participants with a 19.99<BMI<24.99 kg/m^2 were assigned to the normal weight group (NWG), those with a 25≤BMI<29.99 kg/m^2 were classified in the overweight group (OWG), patients with a 30≤BMI<34.99 kg/m^2 made up the obese group (OBG), and those with a 35≤BMI kg/m^2 were categorized in the morbidly obese group (MOG).

Study protocol

Clinical and demographic features of the study patients were recorded. The weight and height of each patient was measured to calculate the BMI: the weight in kilograms was divided by the square of the height in meters. Venous blood samples for biochemical analyses were drawn from all of the subjects after overnight fasting. A complete blood count and levels of triglyceride (TG), total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), blood urea nitrogen (BUN), creatinine, sodium, potassium, and plasma glucose were analyzed. Standard TTE was performed. Informed consent of all of the study participants was obtained and approval was granted by the local ethics committee (TUEK 03.03.2019/01-06).

Evaluation of right heart functions: Right heart functions were assessed by 2 experienced cardiologists using TTE and a Vivid S5 device (GE Healthcare, Inc. Chicago, IL, USA). Complete 2-dimensional echocardiograms, including a Doppler examination, were obtained in all of the standard views (parasternal long-axis, parasternal short-axis, apical 4-chamber, apical 2-chamber). The following measures of the right heart were obtained and evaluated: 1) the RA and RV diameters, expressed in millimeters; 2) the tricuspid annular plane systolic excursion (TAPSE) evaluated in M-mode and expressed in millimeters; 3) the RV myocardial acceleration during isovolumic contraction (R-IVA), which was calculated by dividing the RV myocardial velocity during isovolumic contraction (R-IVV) by the time interval from the onset of this wave to the time at peak velocity (AcT), and an R-IVA of <3.5 cm/s^2 was considered abnormal;[10] 4) the myocardial performance index (MPI), also known as the Tei index, which was calculated with pulse-wave Doppler as (isovolumic contraction time [IVCT] + isovolumetric relaxation time [IVRT]) / ejection time (ET). The IVCT was measured from the end of the A′ wave to the onset of the S′ wave, and the IVRT was measured from the end of the S′ wave to the onset of the E′ wave, and the ET is the time from the onset to the end of the S′ wave. An MPI of >0.55 was considered abnormal.[11] Measurements of 3 cardiac cycles were averaged for all of the right heart function parameters.

Statistical analysis

Statistical analyses were conducted with a commercially available software package (SPSS for Windows, Version 16.0; SPSS, Inc., Chicago, IL, USA). Parametric test data are expressed as mean±SD for continuous variables and as counts and percentages for categorical variables. Continuous variables of nonparametric test data are expressed as median (first quartile-third quartile). Differences were considered statistically significant at p<0.05. Fit with the normal distribution was analyzed using the Kolmogorov-Smirnov test. Intergroup differences were compared with one-way analysis of variance for continuous variables and multiple chi-square tests for categorical variables. Pairwise post hoc tests were performed using either the Tukey Honest Significant Difference test or the Games-Howell test for further analysis of significant results between groups. The Kruskal-Wallis test was conducted to compare groups for parameters that were not distributed normally and for ordinal variables. Post hoc analyses were performed with the Bonferroni test. Variables with a linear correlation were evaluated using Pearson's correlation analysis, and nonlinear variables were evaluated using Spearman's correlation test. In order to analyze independent risk factors associated with RV dysfunction first, all right heart function variables were analyzed for goodness of fit for logistic regression with the Hosmer-Lemeshow test. Multivariate binary logistic regression analyses were performed for variables with a p value of <0.25. An MPI >0.55 and an R-IVA <3.5 cm/s^2 were defined as RV dysfunction. Receiver operating curve (ROC) analysis was used to determine the diagnostic accuracy of the BMI value for the prediction of right heart dysfunction.

RESULTS

A total of 116 patients were enrolled: There were 28 patients (15 female, 13 male) in the NWG, 26 patients (14 female, 12 male) in the OWG, 30 patients (16 female, 14 male) in the OBG, and 32 patients (16 female, 16 male) in the MOG. Demographic and biochemical characteristics of all 4 groups are presented in Table 1. There were no significant intergroup differences in age, sex, smoking history, or systolic and diastolic blood pressure measurements.

Analysis of the lipid profile revealed that the total cholesterol and LDL levels did not significantly increase with the BMI. However, the HDL level was significantly lower in the MOG compared with the NWG (p=0.046). The TG level was significantly higher in the OWG, the OBG, and the MOG compared with the NWG (p=0.017; p=0.007; p<0.001, respectively). Furthermore, there was a moderate positive correlation between the TG level and the BMI (r=0.364, p<0.001). The correlation between the HDL level and the BMI was not statistically significant.

Right heart function analysis results

A comparison of the right heart function parameters of all 4 groups is shown in Table 2. The RA diameter was significantly larger in the OBG and the MOG compared with the NWG (p=0.041; p=0.007, respectively). There was no statistically significant difference in the RA diameter between the NWG and the OWG (p=1.000). The RV diameter was significantly larger

Table 1. Demographic and biochemical characteristics of the NWG, OWG, OBG, and MOG

Variables	NWG	OWG	OBG	MOG	*p*
Age (years)	37.3±11.35	40.3±16.66	40.3±9.22	38.3±11.80	0.067
Male/female (n)	13/15	12/14	14/16	16/16	0.234
Systolic blood pressure (mmHg)	122.5±8.07	125.0±9.49	131.0±6.28	126.4±8.49	0.731
Diastolic blood pressure (mmHg)	59.6±6.23	65.0±7.51	61.5±3.45	64.6±7.21	0.234
Smoking (n)	8	6	8	7	0.896
Fasting blood glucose (mg/dL)	90.5±10.04	99.5±17.23	102.4±22.6	101.9±17.68	0.256
Low-density lipoprotein (mg/dL)	119.5 (107.0–144.7)	118.5 (98.5–142.5)	124 (110.2–158.7)	126.5 (115.2–154.0)	0.079
High-density lipoprotein (mg/dL)	54.3±6.93	52.6±11.04	50.2±10.39	45.0±6.61	0.046[1]
Triglyceride (mg/dL)	137 (98.2–195.7)	166 (119.2–270.0)	162.5 (116.7–219.7)	152.5 (121.5–223.2)	0.002[2]
Total cholesterol (mg/dL)	200 (173.5–230.2)	199.5 (175.2–219.2)	207 (185.7–235.5)	226 (183.7–246.5)	0.088
Blood urea nitrogen (mg/dL)	22.2±6.61	23.5±7.55	25.9±5.87	24.5±8.36	0.493
Creatinine (mg/dL)	0.7±0.08	0.7±0.11	0.8±0.08	0.7±0.09	0.805
Sodium (mEq/L)	140.0±2.08	139.1±1.80	138.0±1.67	139.0±2.21	0.081
Potassium (mEq/L)	4.3±0.32	4.2±0.24	4.2±0.32	4.4±0.38	0.494
Aspartate amino transferase (mg/dL)	19.7±3.56	19.4±6.35	21.3±8.15	28.7±19.42	0.072
Alanine amino transferase (mg/dL)	15.7±5.76	19.6±8.63	20.2±13.53	30.9±26.53	0.053
Hemoglobin (g/dL)	13.1±1.61	13.9±1.64	13.5±1.49	13.4±1.48	0.635
White blood cell (10^9/L)	7.5±1.63	6.4±2.27	7.3±1.84	7.3±1.78	0.248
Platelet (10^9/L)	295.0±83.81	290.7±86.93	260.3±56.82	295.3±78.05	0.437

[1]NWG vs OBG ($p<0.05$); [2]NWG vs OWG ($p<0.05$), NWG vs OBG ($p<0.01$), and NWG vs MOG ($p<0.01$).
MOG: Morbidly obese group; NWG: Normal weight group; OBG: Obese group; OWG: Overweight group.

Table 2. Left ventricular ejection fraction, heart rate, and right heart function parameters of the NWG, OWG, OBG, and MOG

Variables	NWG	OWG	OBG	MOG	*p*
LVEF (%)	62.2±2.92	63.3±2.46	61.9±3.17	62.1±2.43	0.440
Heart rate (bpm)	78.8±5.59	77.6±7.56	77.8±7.98	79.8±9.64	0.813
RA diameter (mm)	28.6±3.92	28.6±2.70	30,9±4.56	32.9±5.08	<0.001[1]
RV diameter (mm)	23.5±2.36	25.0±3.73	27.9±4.51	29.3±3.77	<0.001[2]
TAPSE (mm)	33.6±3.19	35.9±3.64	30.6±5.71	30.3±4.94	0.065
S' lateral annulus (cm/s)	14.7±1.89	13.5±2.37	14.0±2.56	13.9±2.60	0.454
E' lateral annulus (cm/s)	13.9±3.38	14.1±1.74	11.7±2.62	12.7±3.22	0.340
A' lateral annulus (cm/s)	12.9±2.58	14.3±4.76	15.6±4.00	16.7±4.06	0.021[3]
IVCT (ms)	52.7±9.44	64.7±12.47	63.7±11.93	69.5±17.74	0.002[4]
IVRT (ms)	59.1±10.77	65.4±15.16	64.1±16.00	72.7±16.56	0.004[5]
ET (ms)	279.9±47.49	263.6±41.76	226.3±56.66	206.2±42.38	<0.001[6]
MPI	0.3±0.03	0.4±0.08	0.6±0.14	0.7±0.11	<0.001[7]
R-IVV (cm/s)	17.8±4.06	14.9±3.36	12.5±2.30	12.0±2.30	<0.001[8]
AcT (s)	37.7±6.58	40.9±12.10	41.1±11.89	41.5±11.29	0.170
R-IVA (cm/s^2)	4.5±0.65	3,6±0.93	3,2±0.79	2.9±0.73	<0.001[9]
Systolic PAP (mmHg)	17.7±3.73	18.8±4.10	21.5±4.71	24.7±4.75	<0.001[0]

[1]NWG vs OBG (p<0.05), NWG vs MOG (p<0.01); [2]NWG vs MOG (p<0.001); [3]NWG vs MOG (p<0.01); [4]NWG vs OWG (p<0.05), NWG vs OBG (p<0.05), NWG vs MOG (p<0.01); [5]NWG vs MOG (p<0.01); [6]NWG vs OBG (p<0.01), NWG vs MOG (p<0.001); [7]NWG vs OWG (p<0.05), NWG vs OBG (p<0.001), NWG vs MOG (p<0.001); [8]NWG vs OBG (p<0.001), NWG vs MOG (p<0.001); [9]NWG vs OWG (p<0.01), NWG vs OBG (p<0.001), NWG vs MOG (p<0.001); [0]NWG vs MOG (p<0.001). AcT: Peak systolic acceleration time; ET: Ejection time; IVCT: Isovolumic contraction time; IVRT: Isovolumic relaxation time; LVEF: Left ventricular ejection fraction; MOG: Morbidly obese group; NWG: Normal weight group; OBG: Obese group; OWG: Overweight group; PAP: Pulmonary arterial pressure; RA: Right atrium; R-IVA: Right ventricular isovolumic acceleration; R-IVV: Right ventricular myocardial velocity during isovolumic contraction; RV: Right ventricle; TAPSE: Tricuspid annular plane systolic excursion.

only in the MOG compared with the NWG (p=0.004). There was no statistically significant difference in the RV diameter in the NWG compared with the OWG and the OBG (p=0.620; p=0.090, respectively). There was no significant difference in the TAPSE of all 4 groups. The results of the Doppler examination revealed that the R-IVA was significantly lower in the OWG, the OBG, and the MOG compared with the NWG (p=0.020; p<0.001; p<0.001, respectively). Investigation also showed that the MPI was significantly higher in the OWG, the OBG, and the MOG compared with the NWG (p=0.015; p<0.001; p<0.001, respectively). Detailed analysis of the R-IVA revealed that, although the IVV value was significantly lower in the OBG and the MOG compared with the NWG (p<0.001; p<0.001, respectively), the AcT did not change significantly in the 4 groups. However, all of the Doppler parameters (IVCT, IVRT, ET) of the MPI were significantly different (Fig. 1). Moreover, the systolic pulmonary arterial pressure was significantly higher only in the MOG compared with the NWG (17.7±3.73 vs. 24.7±4.75; p<0.001). The differences between other groups were statistically nonsignificant.

In the correlation analysis, it was found that there were moderate nonlinear positive correlations between the RA and RV diameters and the BMI (r=0.417, p<0.001; r=0.430, p<0.001, respectively), a strong linear positive correlation between the MPI and the BMI (r=0.833, p<0.001), and a moderate linear negative correlation between the R-IVA and the BMI (r=-0.547, p<0.001) (Fig. 2). In addition, there were weak positive nonlinear correlations between the IVCT and the IVRT and the BMI (r=0.301, p=0.007; r=0.384, p<0.001) and a moderate nonlinear negative correlation between the ET and the BMI (r=-0.536, p<0.001). On the other hand, the correlation between the R-IVV and the BMI was statistically nonsignificant (r=-0.194, p=0.087). Multivariate binary logistic regression analyses were performed to investigate independent risk factors for RV systolic dysfunction

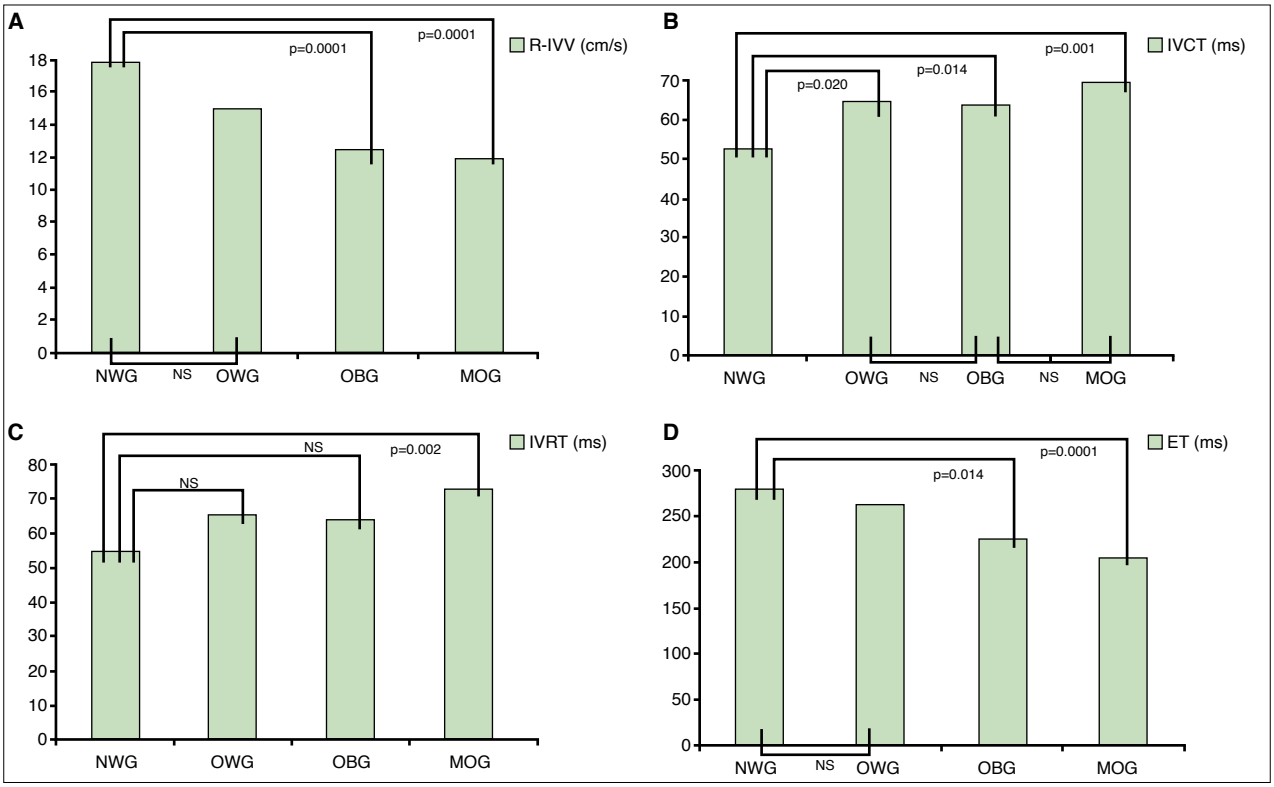

Figure 1. Graphs illustrating **(A)** the significant difference in R-IVV between the OBG, the MOG, and the NWG, **(B)** the significant difference in the IVCT between the OWG, the OBG, the MOG, and the NWG, **(C)** the significant difference in the IVRT between the MOG and the NWG, and **(D)** the significant difference in the ET between the OBG, the MOG, and the NWG.

ET: Ejection time; IVCT: Isovolumic contraction time, IVRT: Isovolumic relaxation time; R-IVV: Right ventricular myocardial velocity during isovolumic contraction; MOG: Morbidly obese group; NS: Nonsignificant; NWG: Normal weight group; OBG: Obese group; OWG: Overweight group.

defined separately by the MPI and the R-IVA (Table 3, 4). Only the BMI was found to be an independent risk factor for RV dysfunction. (R-IVA: B value: 0.191, odds ratio [OR]: 1.211, confidence interval [CI]: 1.074–1.366, p=0.002; MPI: B value: 0.662, OR: 1.939, CI : 1.320–2.850, p<0.001).

The ROC curves for the accuracy of BMI for predicting right heart dysfunction are shown in Figure 3. Right heart dysfunction was defined as an MPI >0.55 or an R-IVA <3.5 cm/s^2. The area under the curve (AUC) for the BMI was 0.977 (95% CI: 0.947–1.007). A cut-off value of 30.45 kg/m^2 for BMI was associated with 93.3% sensitivity and 94.3% specificity in the prediction of RV systolic dysfunction defined by the MPI. Moreover, the AUC for the BMI was 0.784 (95% CI: 0.676–0.892). A cut-off value of 30.50 kg/m^2 for the BMI was associated with 76.7% sensitivity and 72.3% specificity in the prediction of RV systolic dysfunction defined by R-IVA.

Table 3. Multivariate binary logistic regression analysis for right ventricular dysfunction defined by R-IVA

Variables	B value	SE	OR	95% CI	*p*
Right atrium diameter	0.001	0.097	1.001	0.827–1.211	0.994
Right ventricle diameter	-0.086	0.116	0.917	0.731–1.151	0.456
Systolic pulmonary arterial pressure	0.114	0.050	1.121	1.016–1.236	0.241
Body mass index	0.191	0.061	1.211	1.074–1.366	0.002

CI: Confidence interval; OR: Odds ratio; SE: Standard error.

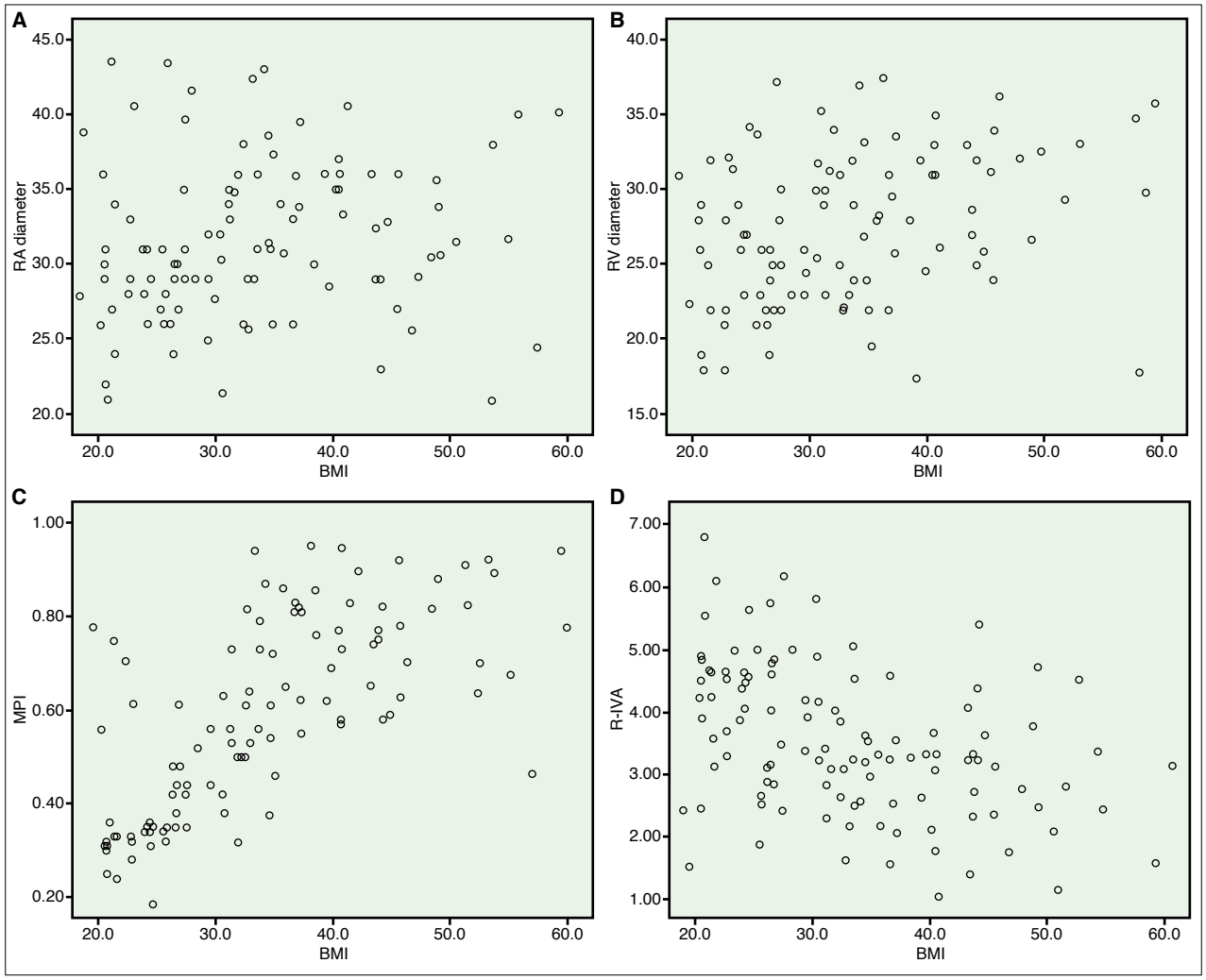

Figure 2. Graphs showing the correlation between **(A)** the RA diameter and the BMI, **(B)** the RV diameter and the BMI, **(C)** the MPI and the BMI, and **(D)** the R-IVA and the BMI. BMI: Body mass index; MPI: Myocardial performance index; RA: Right atrium; R-IVA: Right ventricular isovolumic acceleration; RV: Right ventricle.

DISCUSSION

In the present study, we investigated the effect of obesity on right heart functions. Our results revealed that the diameter of the right heart chamber and right heart functional deterioration were significantly increased with obesity. The BMI value was found to be an independent risk factor for RV dysfunction. In addition, the MPI was found to be more sensitive and specific than the R-IVA in detecting RV systolic dysfunction.

Table 4. Multivariate binary logistic regression analysis for right ventricular dysfunction defined by MPI

Variables	B value	SE	OR	95% CI	*p*
Right atrium diameter	0.227	0.352	1.255	0.629–2.504	0.520
Right ventricle diameter	-0.334	0.381	0.716	0.339–1.513	0.382
Systolic pulmonary arterial pressure	0.253	0.170	1.288	0.922–1.799	0.137
Body mass index	0.662	0.196	1.939	1.320–2.850	<0.001

CI: Confidence interval; OR: Odds ratio; SE: Standard error.

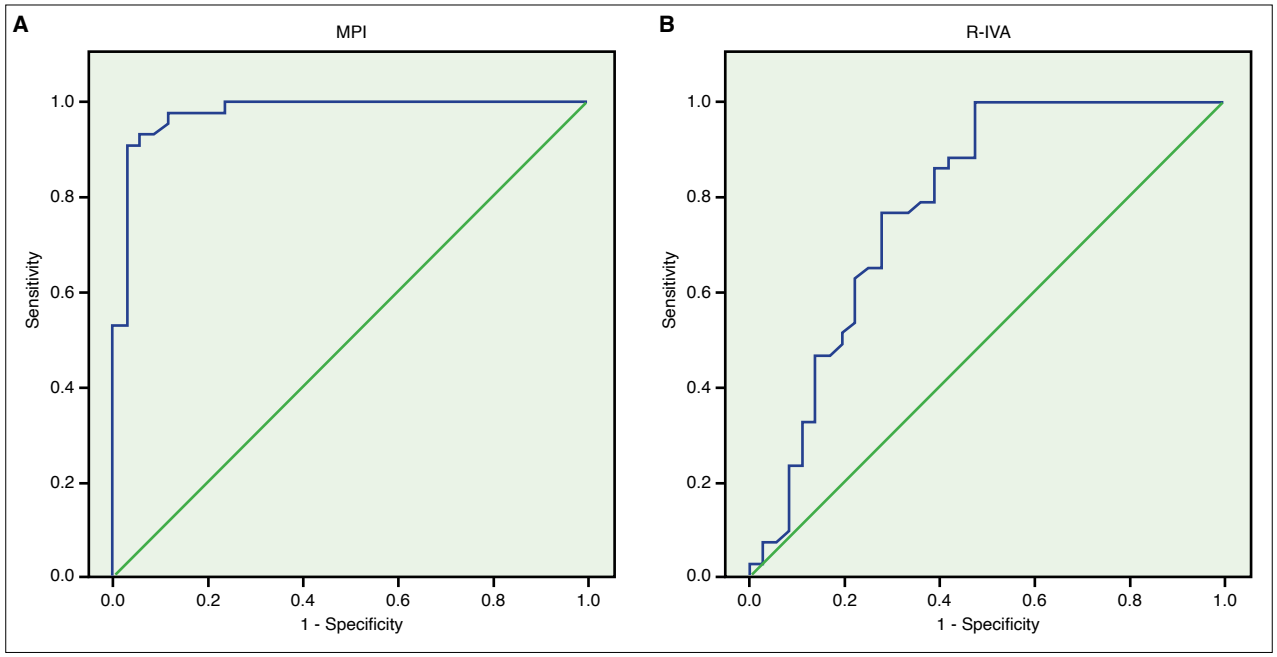

Figure 3. Receiver operating characteristic curve analysis of the performance of the BMI for diagnosing right ventricular dysfunction defined by **(A)** the MPI and **(B)** the R-IVA. BMI: Body mass index; MPI: Myocardial performance index; R-IVA: Right ventricular isovolumic acceleration.

The World Health Organization defines obesity as abnormal or excessive fat accumulation that represents a risk to health. The increasing prevalence of obesity is alarming because it is a risk factor for several chronical diseases, including HT, DM, CAD, dyslipidemia, insulin resistance, metabolic syndrome, and cancer.[12–15]

Numerous metabolic steps are involved in the uptake, transport, and storage of lipids and there are a number of different factors that contribute to dyslipidemia seen in patients with obesity. These factors include greater delivery of free fatty acids to the liver from increased total and visceral adiposity, insulin resistance, and a pro-inflammatory state induced by macrophages infiltrating fat tissue.[16] Gruzdeva et al.[17] observed that in obese patients, increased free fatty acids, pro-inflammatory cytokines (interleukin 1, interleukin 6, tumor necrosis factor alpha) and leptin caused irreversible changes (i.e., dyslipidemia, insulin resistance) in the body. Clinically, dyslipidemia caused by obesity typically consists of increased TG, decreased HDL, and normal or slightly increased LDL.[18] It has been established that the greater the increase in the BMI, the greater the abnormalities in lipid levels.[19] In our study, we also found that the TG level was significantly higher in the OWG, the OBG, and the MOG, and that the HDL level was significantly lower in the MOG compared with the NWG. However, we found a significant correlation only between the TG level and the BMI. The correlation between HDL and the BMI was statistically nonsignificant, which might be due to the small size of the patient group.

As a result of hemodynamic, metabolic, and neurohormonal alterations, obesity may cause LV and RV morphological changes. Barbossa et al.[20] postulated that expanded intravascular volume and elevated vascular resistance present in obesity could cause ventricular hypertrophic remodeling and diastolic dysfunction. Controversially, Alpert et al.[21] reported that impaired systolic function in patients with obesity occurs only in the presence of coexisting heart disease or other risk factors, and that the duration of obesity may be a contributing influence. Most of the previous studies regarding heart function and obesity have been dedicated to the left heart. Hence, scientific data and knowledge of the right heart chambers regarding subjects such as function, morphology, adaptation to loading are still behind what we know for the left heart. Labombarda et al.[22] demonstrated that RV dimensions were enlarged in obese children compared with healthy controls. In another study, Jing et

al.[23] found that obese/overweight children revealed evidence of RV remodeling. Mahfouz et al.,[24] however, observed no changes in RV dimensions. In addition, Shaimaa et al.[25] showed that weight reduction surgery significantly improved right heart function. In our study, we also observed that the RA and RV diameters were significantly increased with obesity. We also demonstrated that the RA and RV diameters were positively correlated with the BMI.

Although various studies have been conducted to investigate the relationship between RV contractile function and different clinical conditions, the relationship between obesity and RV function, especially in the healthy adult population, has not been sufficiently examined. Kowalik et al.[26] showed that overweight status/obesity in adult patients with corrected congenital heart diseases was associated with RV systolic dysfunction. Alhamshari et al.[27] noted that obese patients had better RV systolic function at the time of acute myocardial infarction compared with the non-obese, and that after 2 years of follow-up, the obese patients were less likely to have developed new-onset right heart failure. Furthermore, Serrano-Ferrer et al.[28] reported that RV systolic dysfunction in metabolic syndrome patients, which could be related to obesity-induced changes, was probably not permanent and was modifiable with a healthy diet and physical activity. In our study, we analyzed the RV systolic function of otherwise healthy patients. We found that the R-IVA was significantly lower and the MPI was significantly higher in the OWG, the OBG, and the MOG compared with the NWG. Moreover, the R-IVA and the MPI were significantly correlated with the BMI. We also analyzed the individual effects of all of the parameters used in the calculation of the R-IVA and the MPI. The results showed that a decrease in the R-IVA with obesity was a result of the decrease in the R-IVV. The AcT was not significantly affected by the BMI value. When the MPI was examined, all of the parameters used to calculate the MPI were significantly changed.

There is significantly less myocardial tissue in the RV compared with the LV. Hence, the compensation capability of the RV is more limited. In our opinion, this makes the RV more susceptible to any condition affecting the RV, even at the cellular level. This is why RV systolic functional deterioration seems to affect the peak tissue velocity at the very beginning. This subclinical functional deterioration of the RV also decreases the ET and increases the time needed to counterbalance trans-chamber gradients during isovolumic contraction and relaxation periods. This makes it possible to detect RV functional deterioration even at the subclinical stage using the appropriate echocardiographic techniques. Due to relative volume independence, the IVA is considered a reliable index of global contractility to analyze the systolic function of both ventricles.[29] Furthermore, a number of studies have documented that the MPI is independent of heart rate, arterial pressure, and preload, which also makes it a reliable contractility index.[30] In our study, we also analyzed the predictive accuracy of the BMI in RV systolic dysfunction. At nearly the same BMI levels, the predictive value of the MPI was higher than the R-IVA. The accuracy of the MPI to predict RV systolic dysfunction was greater than that of the R-IVA.

Limitations

The major limitation of our study was the relatively small sample size. The study may also have been affected by the cross-sectional, single-center design. Although 3-dimensional echocardiographic examination and strain study is more sensitive and specific for right heart functional evaluation, they could not be performed for this study due to technical difficulties.

Conclusion

In this study, we concluded that obesity significantly affected right heart functions and that there was a significant correlation between the degree of obesity and right heart functional deterioration. Additionally, the BMI value was an independent risk factor for RV systolic dysfunction and could be used alone to predict RV systolic dysfunction. Furthermore, the MPI was found to be more sensitive and specific than the R-IVA in detecting RV dysfunction.

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