Effect of Obesity on Pulmonary Vascular Hemodynamics

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Abstract

Context: Obesity-related pulmonary arterial hypertension (PAH) is associated with hypoxia and metabolic abnormalities. Although right heart catheterization is the gold standard method for the diagnosis of PAH, Doppler echocardiography is more common. On the other hand, there is no definite echocardiographic parameter for PAH diagnosis. Novel echocardiographic parameter, pulmonary pulse transit time (pPTT), is assumed to be a surrogate marker for the assessment of PAH. Aims: The aim was to evaluate whether pPTT might be valuable for evaluating pulmonary vascular hemodynamics in obese patients. Settings and Design: A cross-sectional observational study. Methods: A total of 130 consecutive obese patients and 50 controls were included. Obese patients were divided into three groups according to body mass index (BMI): <30 < BMI <35 kg/m² formed Group 1, 30 < BMI <35 kg/m² formed Group 2, and 35 < BMI kg/m² formed Group 3. All patients underwent a standard echocardiography including pPTT measurement. pPTT was defined as the interval between R-wave in the electrocardiography and the corresponding peak late systolic pulmonary vein flow velocity measured by pulse wave-Doppler in the pulmonary vein. Statistical Analysis Used: Intergroup differences were analyzed with analysis of variance or Kruskal–Wallis test. Pearson’s or Spearman’s correlation analysis was used for correlation, multivariate logistic regression analysis, and regression. Results: Statistically significant reduction in pPTT was detected as early as in the first group (361.24 ± 25.54 vs. 391.26 ± 15.07; P = 0.015) and continued throughout Groups 2 and 3 (299.92 ± 35.10 vs. 391.26 ± 15.07; P < 0.0001, and 245.46 ± 11.25 vs. 391.26 ± 15.07; P < 0.0001, respectively). There was a strong negative correlation between pPTT and BMI (r = −0.848, P = 0.001). On linear regression analysis, BMI was found to be an independent risk factor for pPTT (confidence interval: −9.164–6.379, β = −0.525, P = 0.0001). Conclusion: The results of this study suggest that obesity leads to an increase in PAH, and pPTT allows noninvasive determination of the pulmonary hemodynamics in obese patients. pPTT might be a useful parameter in terms of predicting pulmonary hemodynamics and vascular alterations in obese patients. Further studies are warranted to evaluate the association between obesity and PAH.

Keywords: Obesity, pulmonary pulse transit time, pulmonary vascular hemodynamics

Introduction

Obesity is one of the uppermost medical conditions, which threatens especially developed countries as a result of Western style diet and sedentary lifestyle. The rapidly increasing prevalence of obesity and associated complications makes it a global pandemic. There is mounting evidence that physical changes and metabolic abnormalities may contribute to the development of serious conditions in these patient population. Obesity-related pulmonary arterial hypertension (PAH) is one of the substantial conditions associated with lung hypoxia due to hypoventilation and inflammatory disturbances. PAH in obese patients has been linked to several comorbidities, including obstructive sleep apnea, obesity hypoventilation syndrome, anorexigenic use, cardiomyopathy of obesity, and pulmonary thromboembolic disease.[1,3] Sustained vasoconstriction and progressive remodeling of pulmonary arteries and arterioles are well known to cause PAH. These changes yield elevated pulmonary arterial pressure (PAP) and pulmonary vascular resistance, decreased pulmonary arterial compliance, and right ventricular (RV) failure.[4,5] Although right heart catheterization is the gold-standard method for the diagnosis of PAH, it has serious complications due to its invasive nature.[6] Because of its noninvasive nature and easy availability, echocardiographic evaluation is more

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common than right heart catheterization for the diagnosis of PAH. On the other hand, poor image quality and operator dependence make it cumbersome. In addition, there is no certain echocardiographic parameter in PAH diagnosis. Therefore, novel techniques are mandatory to assess pulmonary hemodynamics. Recently, Wibmer et al. showed a new echocardiographic indicator for the assessment of pulmonary hypertension, “pulmonary pulse transit time” (pPTT), defined as the delay between the onset of ventricular electric activity (on electrocardiogram) and the arrival of the pulse wave in the pulmonary vein (as determined by Doppler echocardiography of the pulmonary vein). According to their study, the time needed for the pressure pulse wave to travel from the pulmonary valve to the left atrium was shorter in PAH patients compared to controls, and hence postulated that there is a strong association between pPTT and decreased pulmonary artery compliance and increased pulse wave velocity.

In this study, we aimed to evaluate the association between echocardiographic findings, including pPTT and PAH parameters, in obese patients.

**Methods**

**Study population and design**

In this cross-sectional study, participants were selected from patients who were admitted to the obesity outpatient clinic between August 1, 2019, and November 31, 2019. All patients’ past medical records and medications were scanned. Patients having left-sided valve pathologies, ejection fraction <55%, New York Heart Association functional status ≥2, chronic obstructive pulmonary disease, pulmonary thromboembolism history, atrial fibrillation, malignancy, acute or chronic renal insufficiency, pregnancy, and inadequate image quality were excluded from the study. After exclusion, a total of 130 obese patients were included in the study. The included patients were divided into three groups according to their calculated body mass index (BMI). A total of fifty patients (25 < BMI <30 kg/m²) formed Group 1, a total of fifty patients (30 < BMI <35 kg/m²) formed Group 2, and thirty patients (35 < BMI kg/m²) formed Group 3. A total of fifty age- and sex-matched healthy volunteers (20 < BMI <25 kg/m²) formed the control group.

**Study protocol**

Detailed medical histories and clinical and demographic features of the included patients were recorded. All patients’ weights and heights were measured to calculate BMI. 12-lead electrocardiography (ECG) recording (Cardiofax S 2250; Nihon Kohden; Shinjuku-Tokyo/Japan), which was obtained at a paper speed of 25 mm/s and gain of 10 mm/mV in the supine position breathing freely but not allowed to speak during the ECG, and standard transthoracic echocardiography (TTE) were performed in all groups. Informed consent of all participants was taken, and approval was obtained from the local ethics committee (TUEK 05.09.2019/48929119/774).

**Echocardiographic evaluation of the pulmonary vascular hemodynamics**

Pulmonary vascular hemodynamics were evaluated with TTE using General Electric Vivid S5 (Boston, USA) by an experienced cardiologist who was blinded to patient data. Complete 2-dimensional echocardiograms, including Doppler examination, were obtained in all standard views (parasternal long-axis, parasternal short-axis, apical four-chamber, and apical two-chamber views). The following measures were obtained and evaluated. (1) Right atrial (RA) and RV diameters, expressed in millimeters; (2) tricuspid annular plane systolic excursion (TAPSE) evaluated in M mode and expressed in millimeters. A TAPSE of <17 mm indicates RV dysfunction; (3) myocardial Performance Index (MPI), also known as Tei index, was calculated with the use of pulsed-wave Doppler as (isovolumetric contraction time [IVCT] + isovolumetric relaxation time [IVRT])/ejection time (ET). IVCT was measured from the end of the A’ wave to the onset of the S’ wave, and IVRT was measured from the end of the S’ wave to the onset of the E’ wave, and ET is the time from the onset to the end of the S’ wave. MPI > 0.55 is considered abnormal. S’ value of RV lateral wall (RV TDI S’) <0.95 m/s indicates RV dysfunction; (4) pulmonary vein flow was studied by pulse wave Doppler of the right superior pulmonary vein from the apical four-chamber view according to guidelines of the American Society of Echocardiography. pPTT was defined as the time interval between the R-wave peak in ECG and the corresponding peak late-systolic pulmonary vein flow velocity (R-PVs2 interval); and (5) main pulmonary artery diameter and estimated pulmonary artery systolic pressure (ePASP) were calculated by the sum of the Doppler-derived from the tricuspiduspid gradient and the estimated RA pressure, as assessed by the inspiratory collapse of the inferior vena cava. Estimated pulmonary artery systolic pressure (ePASP) >36 mmHg was considered as PAH. All the Doppler recordings were made at a sweep speed of 25 mm/s with a simultaneous superimposed electrocardiogram and measurements of three cardiac cycles averaged for all echocardiographic parameters.

**Statistical analysis**

Statistical analyses were conducted with a commercially available software package (SPSS version 20.0, SPSS, Chicago, IL, USA). In this study, data were expressed as mean ± standard deviation for continuous variables and as counts and percentages for categorical variables. Differences were considered statistically significant at P < 0.05. Fitness to the normal distribution was analyzed with the Kolmogorov–Smirnov test. Homogeneity of variance was calculated with the Levene test and the Lilliefors significance correction test. Intergroup differences of continuous variables were compared with the one-way analysis of variance or its nonparametric counterpart Kruskal–Wallis test. Post hoc analysis was done with either Tukey honestly significant difference or Games–Howell tests. Chi-square and Fisher’s exact tests were used for the comparison of categorical
variables. Correlations of continuous variables were evaluated using Pearson’s correlation analysis or its nonparametric counterpart Spearman’s correlation test. Values 0–0.3 indicated weakly, 0.3–0.7 indicated intermediate, and 0.7–1.0 indicated a strong correlation. Multivariate regression analyses were performed to explore independent factors associated with vPTT.

Results

Baseline clinical and demographic characteristics of all groups are shown in Table 1. There were no statistically significant differences in terms of age, gender, and cardiovascular risk factors among each group. Only dyslipidemia was significantly higher in Groups 2 and 3 ($P = 0.001$) compared to the control group.

Echocardiographic parameters of all groups are shown in Table 2. LV systolic functions were similar in both groups ($P = 0.86$). TAPSE and main pulmonary artery diameters were significantly different only in Group 3 when compared to control group (18.23 ± 1.25 vs. 21.90 ± 1.03; $P = 0.032$, and 27.76 ± 1.30 vs. 25.96 ± 0.72; $P = 0.045$, respectively). The right ventricular myocardial performance index (RV MPI) was significantly higher in Groups 2 and 3 when compared to control group ($0.436 ± 0.023$ vs. $0.391 ± 0.013$; $P = 0.002$, and $0.453 ± 0.016$ vs. $0.391 ± 0.013$; $P < 0.0001$, respectively). In Groups 2 and 3, the mean RV TDI S’ was statistically significantly lower ($0.113 ± 0.0115$ vs. $0.121 ± 0.0909$; $P = 0.043$, and $0.121 ± 0.0909$; $P < 0.0001$, respectively) and although in normal ranges, ePASP was statistically significantly higher (31.46 ± 3.76 vs. 24.66 ± 3.46; $P = 0.032$, and 36.23 ± 4.53 vs. 24.66 ± 3.46; $P < 0.0001$, respectively) when compared to control group. Furthermore, TAPSE/mean ePASP ratio was shorter in Group 2 and 3 patients when compared to control group (0.580 ± 0.137 vs. 0.764 ± 0.977).

| Table 1: Demographic and clinical characteristics of all groups. |
|------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Variables              | Group 1 (25<BMI<30) | Group 2 30<BMI<35 | Group 3 35<BMI  | Control         |
| Number of patients ($n$) | 50               | 50               | 30              | 50              |
| Age (years)            | 38.21±10.32      | 39.31±12.26      | 40.25±6.22      | 39.30±6.85      | 0.642           |
| Gender (Male, $n$)     | 27               | 26               | 15              | 26              | 0.989           |
| Hypertension ($n$)     | 33               | 37               | 23              | 33              | 0.620           |
| Dyslipidemia ($n$)     | 30               | 39               | 24              | 26              | 0.001           |

| Table 2: Echocardiographic parameters of all 4 groups. |
|------------------------|-----------------|-----------------|-----------------|-----------------|
| Variables              | Group 1 (25<BMI<30) | Group 2 30<BMI<35 | Group 3 35<BMI  | Control         |
| Left ventricular ejection fraction (%) | 62±3.56       | 60±5.52        | 61±4.23         | 62±4.37        | 0.71 |
| TAPSE (mm)             | 21.68±1.31       | 20.96±2.14      | 18.23±1.25      | 18.23±1.25      | 0.642 |
| RVMPI                  | 0.404±0.021      | 0.436±0.023     | 0.453±0.016     | 0.453±0.016     | 0.391±0.013 |
| RVTDIS’ (m/s)          | 0.118±0.0115     | 0.113±0.0158    | 0.0108±0.0143   | 0.0121±0.0909   | 0.121±0.0909 |
| Mean estimated PASP (mmHg) | 25.01±2.71     | 31.46±3.76     | 36.23±4.53      | 24.66±3.46      | 24.66±3.46 |
| Main pulmonary artery diameter (mm) | 26.06±0.91    | 26.66±0.95     | 27.76±1.30      | 25.96±0.72      | 25.96±0.72 |
| TAPSE/ePASP            | 0.738±0.114      | 0.580±0.137     | 0.457±0.071     | 0.764±0.977     | 0.764±0.977 |
| pPTT (ms)              | 361.24±25.54     | 299.92±35.10    | 245.46±11.25    | 391.26±15.07    | 391.26±15.07 |
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$P = 0.001$, and $0.457 \pm 0.071$ vs. $0.764 \pm 0.977$; $P < 0.0001$, respectively). One of the main purposes of this study was to evaluate the association between obesity and measurements of pPTT. Statistically significant reduction in pPTT was detected as early as in the first group (361.24 ± 25.54 vs. 391.26 ± 15.07; $P = 0.015$) and continued throughout Groups 2 and 3 (299.92 ± 35.10 vs. 391.26 ± 15.07; $P < 0.0001$, and 245.46 ± 11.25 vs. 391.26 ± 15.07; $P < 0.0001$, respectively) [Figure 1].

On correlation analysis, we analyzed the correlation between BMI and pulmonary vascular hemodynamic parameters. We found that there was a statistically significant negative correlation between pPTT and BMI ($r = -0.848, P = 0.001$); intermediate negative correlation between TAPSE, TAPSE/mean ePASP, RV TDI S', and BMI ($r = -0.616, P = 0.001$; $r = -0.620, P = 0.001$; and $r = -0.491, P = 0.001$, respectively); and intermediate positive correlation between RVMPi, mean EPASP, main pulmonary artery diameter, and BMI ($r = 0.649, P = 0.001$; $r = 0.593, P = 0.001$; and $r = 0.609, P = 0.001$, respectively) [Figure 2]. There was a weak negative correlation between left ventricular ejection fraction and BMI ($r = -0.247, P = 0.001$).

On linear regression analysis, only RVMPi and BMI were found to be independent risk factors for pPTT (RVMPi: confidence interval [CI]: $-1362.007$ to $-610.775$, $\beta = -0.414, P = 0.0001$; BMI: CI: $-9.164$ to $-6.379$, $\beta = -0.525, P = 0.0001$) [Table 3].

### Discussion

PAH is a life-threatening condition that is associated with multiple pathophysiological factors. To treat those afflicted with PAH, a systematic therapeutic approach to identify all reversible mechanisms is mandatory.

It is well known that extreme adipose accumulation in obese patients results in transient or permanent hypoventilation due to increased mechanical load.[3] Hypoxemia and lung hypoxia may cause increased upper airway resistance, decreased inspiratory and expiratory pressure, and decreased chest wall compliance. These factors lead to obesity-related obstructive sleep apnea and obesity-related hypoventilation syndrome which induces obesity-associated PAH in the long term.[11,12] Apart from the above-mentioned pathways, extreme adipose accumulation also triggers several metabolic and physiologic changes which contribute to the development of PAH. It is well known that obesity-related global insulin resistance is a common condition associated with increased levels of plasma blood insulin levels.[13] Insulin resistance not only induces hepatic glucose production but releases triglycerides into the blood, which yield increased levels of free fatty acids (FFAs).[14,15] In addition, elevated circulating inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α, and adipokines lead to peripheral insulin resistance and systemic inflammation associated with obesity.[16] Deregulated lipid and carbohydrate metabolism and obesity-associated inflammation deteriorated both cardiovascular system and pulmonary function.[17]

Extreme adipose accumulation in obese patients also induces pathological processes including reprogramming of oxidant genes and subsequent redox changes in different tissues.[18,19] Recent studies demonstrated that extreme conditions of hyperglycemia produce reactive oxygen species (ROS) accumulation, which induces advanced glycation end products, hyperglycemia produce reactive oxygen species (ROS) accumulation, which induces advanced glycation end products, polyol, and hexosamine pathways as well as pro-inflammatory transcriptional programs initiated by nuclear factor kappa B (NF-kB).[20] These ROS-sensitive by-products provoke endothelial dysfunction and micro and macrovascular complications.[21] In addition, FFA-related excess ROS accumulation not only activates pro-inflammatory cytokines, but also baffles the activity of key anti-atherogenic enzymes such as prostacyclin synthase (PGIS) and endothelial nitric oxide synthase (eNOS). Experimental animal models showed that activation of FFA release from the adipose tissue may cause inactivation of PGIS and eNOS.[22] The association between FFA-induced ROS expression and PGIS synthase and eNOS activities was also investigated in cultured arterial endothelial cells. Adverse changes in PGIS synthase activity were assessed in vivo after fatty acid infusion in rats. Moreover, unfavorable effects of FFA-induced ROS activation on aortic PGIS synthase activity and eNOS were demonstrated in insulin-resistant obese Zucker rat (fa/fa rat; homozygous for the mutant allele[23] and the high fat diet-induced insulin-resistant mouse).[15,24,25] Yet another underlying mechanism associated with obesity-associated PAH is downregulation of serum adiponectin concentration in obese patients. Adiponecin which is an endogenous modulator of the eNOS suppresses both endothelial inflammation and proliferation via endothelial-derived nitric oxide production.[26] Animal studies demonstrated that adiponectin-deficient mice were prone to perivascular inflammatory cell infiltration as a result of pro-inflammatory mTOR and NF-kB pathways.[27] On the other hand, overexpression of adiponectin has been shown to prevent mice from developing PAH.[28]

At present, pulmonary hypertension is defined as mean PAP 25 mmHg at rest or 30 mmHg during physical activity as measured by right heart catheterization.[29] Although right heart catheterization is the gold standard method for the diagnosis of PAH, due to its invasive nature and higher costs, it is not common in cardiovascular practice. Because of its noninvasive nature and easy access, echocardiography is

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**Table 3: Results of the linear regression analysis.**

| Variable | CI (Confidence interval) | Beta value | $p$  |
|----------|------------------------|------------|------|
| RVMPi    | $-1362.007$ to $-610.775$ | $-0.414$ | 0.0001 |
| TAPSE    | $-9.859$ to $-0.399$    | $-0.135$  | 0.070 |
| RVTDIS   | $-248.741$ to $-558.856$ | $0.032$   | 0.450 |
| ePASP    | $-1.672$ to $-2.874$    | $0.061$   | 0.602 |
| BMI      | $-9.164$ to $-6.379$    | $-0.525$  | 0.0001 |
| Main pulmonary artery diameter | $-4.387$ to $-5.312$  | $0.008$   | 0.851 |
| TAPSE/ePASP | $-39.542$ to $-232.407$ | $0.218$   | 0.163 |
Figure 1: Boxplot graphics showing results of various echocardiographic data. Upper-left = Distribution of pulmonary pulse transit time values, Upper-right = Distribution of right ventricular myocardial performance index values, Lower-left = Distribution of RVTDIS' values, Lower-right = Distribution of tricuspid annular plane systolic excursion values among all the four groups.

Figure 2: Correlation graphics of body mass index and various echocardiographic parameters. Upper-left: Body mass index and pulmonary pulse transit time, Upper-right: Body mass index and right ventricular myocardial performance index, Lower-left: Body mass index and RVTDIS', Lower-right: Body mass index and tricuspid annular plane systolic excursion.
more common in terms of diagnosing and serial assessment of pulmonary hypertension. Unfortunately, there is no reliable echocardiographic method of determining mean pulmonary artery pressure. Generally, ePASPs >40 mmHg is considered abnormal. However, a recent study conducted by Fisher et al. demonstrated that estimated pulmonary pressures by echocardiography were erroneous in nearly 48% of patients with pulmonary hypertension.\[30]\] Therefore, there is a need for a more accurate method in terms of evaluating intrinsic pulmonary hemodynamics and RV function.

Recently, Wibmer et al. demonstrated a novel method for noninvasive hemodynamic assessment of pulmonary circulation known as pPTT.\[9]\] According to their study, calculated pPTT was shorter in patients with pulmonary hypertension compared to healthy controls, and there is a strong association between pPTT and pulmonary hemodynamic and vascular changes.\[9]\]

Herein, we investigated the association between obesity and pulmonary vascular hemodynamics through novel echocardiographic methods. Previous studies have shown that there is a strong association between excess body fat and pathophysiological changes in the pulmonary circulation. According to a study conducted by Taraseviciute and Voelkel, nearly 50% of patients with a history of PAH were clinically obese, while in another study, twenty ambulatory obese patients who underwent echocardiographic measurement demonstrated evidence of PAH.\[31-33\] Our results demonstrated that obese patients have a higher RVMPI, shorter TAPSE, and higher ePASP than controls, which is consistent with those of previous studies.\[34,35\] In addition, we observed statistically significant shorter pPTT values in obese patients compared to controls, which indicate that shorter pPTT values are related to premature pathological changes in pulmonary vascular circulation. We also showed that the degree of obesity was an independent risk factor for pPTT. To the best of our knowledge, this is the first study which demonstrated the relationship between obesity and pPTT. According to our findings, shorter pPTT values may provide additional information about pulmonary hemodynamics in obese patients. These findings support the hypothesis that pulmonary hemodynamics deteriorated early before clinical manifestation in obese patients.

**Study limitations**

In our study, we used conventional BMI in order to define the degree of obesity. Although this index is a simple, quick, noninvasive tool that can accurately identify increased cardiovascular risk, it evaluates “total obesity,” without assessing body fat distribution. It is well known that there is a strong association between abdominal obesity and development of cardiovascular diseases. Therefore, waist circumference (WC) is correlated with intra-abdominal total adiposity, which is an independent predictor for insulin resistance, diabetes, and cardiovascular disease.\[36-38\] Recently, Antonini-Canterin et al. developed a novel index which combines BMI (correlated with total body fat mass) and waist circumference (WC) (reflecting mainly abdominal obesity): waist-corrected BMI (wBMI). It is calculated as WC in meters × BMI in kg/m², resulting in kg/m.\[39\] By using wBMI, not only global fat mass and fat distribution evaluated concurrently, but also limitations associated with BMI and WC could be solved. According to their study, nearly 47% of the overweight and obese Grade I patients were reclassified in lower or higher risk categories after using wBMI. Given the above-mentioned implications, it is likely that using wBMI might be a better method compared to conventional BMI.

As we mentioned in our main text due to its noninvasive nature, the pulmonary hemodynamics could not be determined accurately by Doppler-derived echocardiography. However, in our text, patients whose Doppler-derived estimated systolic pulmonary artery pressure >36 mm Hg were considered as PAH. Because this diagnosis is based on invasive hemodynamic assessment of pulmonary pressures through right heart catheterization, it would be better to label them as patients with decreased pulmonary artery compliance rather than patients with PAH. We used conventional echocardiographic parameters in terms of evaluating pulmonary hemodynamics and RV functions, but there are various important echocardiographic parameters including RA area, right outflow tract acceleration time, and pulmonary regurgitation early diastolic velocity. Yet our major aim was to assess the association between PAH and obesity by using novel echocardiographic parameters of pPTT. Although it is the largest study to date of pPTT in obese patients, it is nonetheless a relatively small, single-center study. Finally, the determination of pPTT was limited by the availability and interpretability of pulmonary vein Doppler signals.

**Conclusion**

pPTT was reduced in obese patients compared to healthy controls and strongly correlated with the severity of obesity. Our data show that obesity leads to an increase in PAP, and pPTT allows noninvasive determination of pulmonary hemodynamics. Furthermore, the severity of obesity is an independent risk factor for pulmonary hemodynamic dysfunction.

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**Conflicts of interest**

There are no conflicts of interest.

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