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The combined effect of systemic hypertension and obesity on biventricular diastolic function

Left ventricular (LV) cardiomyopathy is a frequent complication of untreated or undertreated systemic hypertension. LV concentric hypertrophy, increased oxygen demand and impaired left atrial emptying due to diminished LV compliance and increased ventricular afterload in patients with systemic hypertension lead to inappropriate LV filling (6, 8). LV dilatation is a common consequence of increased preload in patients with obesity (3). Eccentric LV hypertrophy is an adaptive mechanism lowering increased wall stress of dilated LV cavity. While it is evidenced that systemic hypertension influences right ventricular (RV) structure, diastolic performance and pulmonary hemodynamics, little is known about RV function and the interaction between right and left ventricles in hypertensive patients with coexisting obesity.

The aim of our study was the echocardiographic assessment of biventricular structure, diastolic function and pulmonary hemodynamics in obese patients with untreated, mild to moderate systemic hypertension.

MATERIAL AND METHODS

The study group consisted of 31 patients aged 26 to 55 years (mean 44.19±7.89) with untreated mild to moderate systemic hypertension, obesity expressed as a body mass index >26 kg/m^2 and sinus rhythm in standard electrocardiography. In all patients lung function tests were performed to exclude patients with severe lung disorders. Left ventricular (LV) end-diastolic diameter (LVEDD), interventricular septum diameter (IVSD), posterior wall diastolic diameter (PWDD), LV ejection fraction (EF), right ventricular wall diastolic diameter (RVWDD), right ventricular outflow tract diameter (RVOTD) and left atrium diameter (LAD) were measured during echocardiography.

During pulsed Doppler echocardiography early filling wave velocity of the left (MVE) and right ventricle (TVE), late filling wave velocity of the left and right ventricles (MVA and TVA, respectively) were recorded. Early to late filling wave velocity ratio was calculated both for the left and right ventricles (MVE/A and TVE/A). Additionally, velocity time integral (VTI) of early and late filling wave and velocity time integrals ratio were measured (MVTIE, MVTIA, MVTIE/A and TVTIE, TVTIA, TVTIE/A, respectively). LV isovolumic relaxation time (IVRT) was measured. Pulmonary artery acceleration time corrected for heart rate (AcT), RV ejection time (RVET) and AcT/RVET ratio were measured. Mean pulmonary artery pressure (MPAP) was calculated according to the formula: $\log_{10}MPAP = -0.0068\ AcT + 2.1$ (5).

18 hypertensive subjects aged 23 do 55 years (mean 42.08±8.96) with BMI <26 kg/m^2 were enrolled to the study as a control group.

Statistical analysis. The statistical analysis was performed using STATISTICA V 4.3 for Windows. Data are presented as mean ± standard deviation. Student's t test was used to compare data, p values < 0.05 were considered statistically significant.

RESULTS

Clinical features of the hypertensive obese and hypertensive lean patients are listed in Table 1. Patients from the study group demonstrated significantly thicker LV posterior wall and enlarged left atrial cavity compared to controls (11.16±1.98 vs 9.75±2.06 mm and 39.3±4.95 vs 35.71±6.08 mm, respectively). There were no significant differences of LVEDD, IVSD and EF. Also mean RV outflow tract diameter and RV free wall thickness were similar both in the study and control group. In the hypertensive obese group we showed significantly lower LV early to late filling velocity ratio (0.99±0.27 vs 1.33±0.54, p<0.01) and early to late filling wave velocity-time integrals ratio (1.37±0,69

Table 1. General characteristics of the studied population

Parameter	Study group	Controls	p
Age (years)	44.19±7.89	42.08±8.96	NS
Height (cm)	168.42±9.15	168.62±6.93	NS
BMI (kg/m^2)	30.52±3.2	23.4±1.58	<0.001
$RR_{systolic}$	158.06±17.97	155.38±15.74	NS
$RR_{diastolic}$	93.06±11.6	86.15±11.39	NS
$R\text{-}R_{interval}$ (s)	0.8±0.12	0.87±0.14	NS

vs 2.53±1.19, p<0.01) reflecting mild impairment of LV diastolic function. The analysis of RV filling patterns revealed only decreased early to late filling wave velocity-time integral ratio in the study group (1.76 vs 2.43, p<0.05), while other measured parameters did not differentiate obese and lean subjects.

Obese patients demonstrated significantly shortened pulmonary artery acceleration time (0.110 s vs 0.129 s, p<0.001). AcT/RVET was significantly lower in the study group 0.39±0.06 vs 0.45±0.07, (p<0.01). Noninvasively estimated mean pulmonary artery pressure was significantly increased in the study group 18.2±5.4 mmHg vs 13.35±3.9 mmHg, p<0.01.

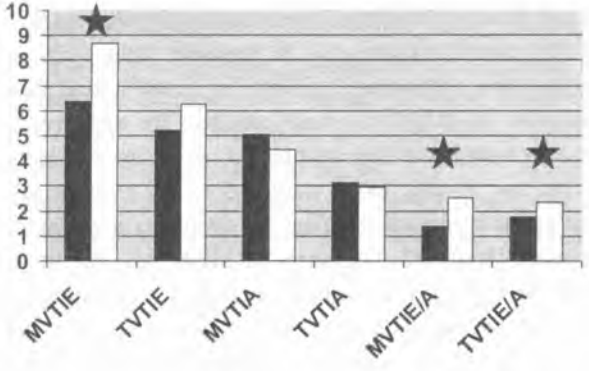

Fig. 1. Left and right ventricular diastolic filling parameters; MVTIE (cm) – early LV filling wave velocity-time integral; TVTIE (cm) – early RV filling wave velocity-time integral; MVTIA (cm) – late LV filling wave velocity-time integral; TVTIA (cm) – late RV filling wave velocity-time integral; MVTIE/A – early to late LV filling wave velocity-time integral ratio; TVTIE/A – early to late RV filling wave velocity-time integral ratio; Significant differences are labelled with an asterisk

DISCUSSION

The coexistence of essential systemic hypertension and obesity is a frequent clinical finding. The degree of LV dysfunction in hypertensive obese patients is a result of the combined effect of increased preload and afterload on ventricular structure and function. Concentric LV hypertrophy is the classic adaptation of the heart to the increased LV afterload in hypertensive patients (4). In contrast, increased total blood volume in obese leads to eccentric hypertrophy due to sustained elevation of LV preload (3). This double hemodynamic burden is also responsible for the premature manifestation of LV diastolic dysfunction.

In our study we demonstrated the impairment of LV diastolic function more pronounced in obese subjects. Our findings are in accordance with some previous studies reporting the premature development of LV diastolic filling abnormalities in obese pa-

tients compared to lean subjects with the same levels of systolic and diastolic blood pressure (3, 1). Recently, it has been demonstrated that also RV function is influenced by increased systemic hypertension and RV filling parameters correlate with LV indexes of diastolic performance. This ventricular interaction in systemic hypertension is probably mediated by common interventricular septum (Bernheim syndrome), increased pulmonary artery pressure due to impaired left atrial emptying and last but not least by systemic substances like angiotensine II, aldosterone and catecholamines (2).

The degree of RV involvement in hypertensive obese subjects remains still unexplained. The statistical analysis of RV echocardiographic measurements showed no significant differences between lean and obese patients. Only RV filling wave velocity-time integrals ratio was significantly diminished in patients with overweight, which might indicate inappropriate RV diastolic filling due to impaired right atrial emptying and RV compliance. The noninvasive estimation of pulmonary artery systolic flows revealed statistically significant increase of MPAP in obese subjects. The right ventricle, as a central element of low-pressure system is better adapted to the changes of blood volume passing through. Thus, the increase in total blood volume does not produce substantial elevation of pulmonary artery pressure that might affect RV emptying and stimulate RV hypertrophy (7). In our opinion, significant differences of MPAP are due to the changes in LV function rather than the primary disturbed pulmonary circulation. The elevation of MPAP may reflect the impairment of left atrial inflow as a result of decreased LV compliance and diastolic filling. Thus, we suggest that RV abnormalities in patients with systemic hypertension with concurrent obesity depend mainly on increased RV afterload, secondary to LV dysfunction, while increased preload affects directly LV but not RV performance.

REFERENCES

1. Alpert M. A. et al.: Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. Am. J. Cardiol., 55, 783, 1985.
2. Chakko S. et al.: Right ventricular diastolic function in systemic hypertension. Am. J. Cardiol., 65, 1117, 1990.
3. Grossman E. et al.: Left ventricular filling in the systemic hypertension of obesity. Am. J. Cardiol., 68, 57, 1991.
4. Inoyue I. et al.: Abnormal left ventricular filling: an early finding in mild to moderate systemic hypertension. Am. J. Cardiol., 53, 120, 1984.
5. Kitabatake A. et al.: Noninvasive evaluation of pulmonary hypertension by a pulsed Doppler technique. Circulation, 68, 302, 1983.
6. Oh J. K. et al.: The noninvasive assessment of left ventricular diastolic function with two-dimensional and Doppler echocardiography. J. Am. Soc. Echocardiogr., 10, 246, 1997.

7. Olivari M. T. et al.: Pulmonary hemodynamics and right ventricular function in hypertension. Circulation, 57, 1185, 1978.
8. Thomas J. D. et al.: Echocardiographic Doppler evaluation of left ventricular diastolic function. Physics and physiology. Circulation, 84, 977, 1991.

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SUMMARY

Left ventricular (LV) cardiomyopathy in hypertensive obese patients is a consequence of increased LV afterload due to systemic hypertension and increased LV preoload subsequent to the augmentation of total blood volume in obese. However, little is known about right ventricular (RV) functional status and biventricular interaction in patients with systemic hypertension and concurrent obesity. The aim of our study was the echocardiographic assessment of left and right ventricular function and diastolic performance in hypertensive obese patients. The study group consisted of 31 subjects with BMI> 26 kg/m^2 and mild to moderate systemic hypertension, patients with systemic hypertension and normal BMI served as a control group. M-mode echocardiographic measurements of left and right ventricles were performed. By pulsed Doppler echocardiography LV and RV recordings of diastolic filling and systolic flows in pulmonary artery were obtained. Hypertensive obese patients demonstrated significantly thicker posterior wall and enlarged left atrium, while no differences in RV structure were found between both groups. In obese subjects we showed significantly lower LV early to late filling wave velocity ratio and LV early to late filling wave velocity-time-integral ratio ($p<0.01$). Only RV early to late filling wave velocity-time-integral ratio differentiated obese and lean subjects ($p<0.05$). In obese subjects we demonstrated significantly increased mean pulmonary artery pressure. The results of our study indicate that RV is protected from the direct influence of hemodynamic changes observed in overweight/obesity better than the left ventricle and RV abnormalities in patients with systemic hypertension and obesity depend mainly on increased RV afterload, secondary to LV dysfunction.

Złożony wpływ nadciśnienia tętniczego i otyłości na czynność rozkurczową obu komór serca

Rozwój kardiomiopatii u chorych z nadciśnieniem tętniczym i otyłością jest następstwem zwiększonego obciążenia wstępnego i następczego (*preload* i *afterload*) lewej komory serca. W literaturze istnieje stosunkowo niewiele danych dotyczących skojarzonego wpływu nadciśnienia i otyłości na budowę i czynność prawej komory serca. Celem pracy

była ocena wpływu nadciśnienia tętniczego współistniejącego z otyłością na czynność rozkurczową obu komór. Badana grupa składała się z 31 pacjentów z nadciśnieniem tętniczym i otyłością wyrażoną jako BMI>26 kg/m^2, grupę kontrolną stanowili chorzy z nadciśnieniem i prawidłową masą ciała. Badaniem dopplerowskim oceniano przepływy rozkurczowe przez lewe i prawe ujście przedsionkowo-komorowe oraz przepływ skurczowy w pniu płucnym. W grupie chorych z nadciśnieniem i otyłością stwierdzono przerost tylnej ściany lewej komory serca oraz powiększenie lewego przedsionka. Nie wykazano różnic morfologicznych w zakresie komory prawej. W grupie badanej stwierdzono istotnie statystycznie niższy stosunek wczesnego do późnego napływu mitralnego oraz stosunek całki wczesnego do późnego napływu. W zakresie prawej komory serca obie grupy różnicował jedynie stosunek całki wczesnego do późnego napełniania prawej komory, stwierdzono ponadto w grupie badanej istotne statystycznie podwyższenie ocenianego nieinwazyjnie średniego ciśnienia w tętnicy płucnej. Wyniki badań wskazują na to, że prawa komora podlega niekorzystnym wpływom nadciśnienia tętniczego skojarzonego z otyłością w stopniu mniejszym niż komora lewa.