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ON THE QUESTION OF DIASTOLIC DYSFUNCTION OF THE LEFT VENTRICLE IN METABOLIC SYNDROME

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Abstract:

In this article, an epidemic of metabolic diseases is currently approaching - obesity, diabetes mellitus (DM), arterial hypertension (AH). Analyzes have been reported that the proportion of the population with metabolic syndrome (MS) leading to the development of type 2 diabetes and cardiovascular disease (CVD) is growing faster.

Predicting the progression of Metabolic Syndrome is determined not only by blood pressure (BP), glycemic control level, and metabolic disorders, but also by heart remodeling.

Keywords: Metabolic Syndrome, direct correlation, systolic parameters, diastolic filling, hypertrophy, diastolic myocardial dysfunction.

INTRODUCTION

Currently, an epidemic of metabolic diseases is approaching - obesity, diabetes mellitus (DM), arterial hypertension (AH). The proportion of the population with metabolic syndrome (MS), a precursor to the development of type 2 diabetes mellitus and cardiovascular disease (CVD), is increasing even more rapidly [1]. Predicting the course of MS is determined not only by blood pressure (BP), the degree of glycemic control, and metabolic disorders, but also by cardiac remodeling [9]. One of the main early manifestations of the so-called "metabolic heart" is the development of diastolic dysfunction (DD) of the left ventricle (LV), which is observed in most patients with MS [5,10]. Successful prevention of the development of heart failure in people with MS consists in the earliest possible detection of DD and its timely correction [2,4,5].

Most recent studies indicate that the violation of LV relaxation is the earliest manifestation of the "metabolic heart" [3,4,9]. There is evidence that impaired LV diastolic dysfunction is invariably associated with the development of its visible structural changes [10]. Thus, left ventricular hypertrophy (LVH) with an increase in its mass leads to LV diastolic dysfunction: an increase in the time of LV isometric relaxation, a decrease in blood flow to the early filling phase, an increase in LV end-diastolic pressure, etc. However, the relationship between the level of blood pressure in MS, the degree of LVH and the severity of its diastolic dysfunction is estimated inconsistently [1,2,5]. There are also indications that DD develops at the initial stages of MS and precedes LVH [13]. The available data on the prevalence of DD among patients with MS are also contradictory, which is due to the dependence of this condition on many factors, including the age of the examined individuals [7,9]. The development of DD in MS is often observed in patients without LVH, while in some patients with LVH, DD indicators remain within the conventional norm [7,8,9].

THE AIM of the study was to study the relationship between the level of blood pressure, the mass of the left ventricular myocardium and indicators of its diastolic function in patients with metabolic syndrome.

MATERIAL AND METHODS:

We examined 60 patients diagnosed with MS (27 men and 33 women aged 35-60 years) (average 47.3±5.1 years). The control group consisted of 20 people matched by sex and age.

Metabolic syndrome was diagnosed according to the criteria proposed by the US National Cholesterol Education Program Experts (2005). The criteria for MS were waist circumference over 94 cm in men and over 80 cm in women; blood pressure 130/85 mmHg and above, the level of glucose in the blood plasma on an empty stomach is 5.6 mmol / l or more. Body mass index (BMI, Quetelet index) was calculated using the formula BMI = body weight (kg) / height (m2).

Echocardiographic examination was performed in 1- and 2-dimensional mode using the Sono-Scape ultrasound machine (China). The anterior-posterior size of the left atrium (LA), the end systolic size (ESD) of the LV and the end diastolic size (EDD) of the LV were determined. The end diastolic volume (EDV) of the LV and the end systolic volume (ESV) of the LV were calculated using the formula of L. Teichheltz et al. LVM was calculated based on the echocardiographic

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determination of its length and thickness according to the formula of R. Devereux [8]. The LVMI index (LVMI) was defined as the ratio of LVMI to body surface area. The LVH criterion was taken as the LVMI value exceeding 134.0 g/m2 in men and 110.0 g/m2 in women.

LV diastolic function was assessed using Doppler echocardiography [5,6]. At the same time, the maximum blood flow velocity through the left atrioventricular orifice in the phase of rapid filling of the left ventricle (E), the maximum velocity of blood flow through the left atrioventricular orifice in the phase of atrial systole (A), their ratio (E/A), the integral of the rate of early filling LV (Ei), late LV filling rate integral (Ai), Ei/Ai ratio, deceleration time of maximum early LV filling rate (B3), isovolumetric relaxation time (IVVR). IVVR>100ms and E/A<1.0 were considered signs of diastolic dysfunction.

Statistical processing of the results was carried out using the methods of variation statistics, including correlation analysis, as well as the calculation of an unpaired Student's t-test to assess the significance of differences between groups.

RESULT AND DISCUSSION:

The anteroposterior size of the LA was increased ($p<0.05$) already at the initial stage of MS, which indicates the early involvement of the LA in the pathological process even before the development of LVH (Table 1) [5]. LA dilatation increases significantly ($p<0.01$) with an extended clinical picture of MS. The dimensions of the left ventricle changed in a similar way: in the initial stage of MS, a relatively small but statistically significant increase in LV EDR was noted, which in a patient with a clinically advanced stage of MS reached significantly larger values (Table 1). The LV volume in systole and diastole in patients with the initial stage of MS does not differ from the norm; in the presence of an advanced stage of MS, it was significantly increased (Table 1).

When using EchoCG criteria R. Devereux LVH was diagnosed in 32 patients, in 30 patients LVMI and LVMI did not exceed the norm. The average values of LVML (205.7±6.4 g) and LVMI (112.6±3.8 g/m2) in patients with the initial stage of MS were 1.7 times ($p<0.001$) and 1.3 times ($p<0.01$) corresponding indicators of the normal group (126.1±3.7 g and 71.6±1.41 g/m2), however, this increase did not reach values sufficient for the diagnosis of LVH according to EchoCG criteria R. Devereux .

In patients with a clinically advanced stage of MS, LVMM and LVMI corresponded to LVH, and the average values of LVMM (312.0±8.3 g) and LVMI (164.2±4.3 g/m2) more than 2 times ($p<0.001$) exceeded the control group.

When studying the LV diastolic function, it was found that in patients with the initial stage of MS, IVVR significantly exceeded the control, while there were no significant differences in E and B3 between patients with the initial stage of MS and the control. In patients with MS of the clinically advanced stage, the LV relaxation function continued to deteriorate, with a decrease in E and an increase in the contribution of atrial systole to the structure of diastolic filling of this part of the heart (Table 2). The E/A ratio did not reach the same values as compared with the MS group of the initial stage and the control group (Table 2). Similar changes, but with a greater degree of severity, were characteristic of VIVR and B3 (Table 2).

LV EDR in patients with MS in the initial stage and in the control group did not differ significantly ($p>0.1$). With MS at the clinically advanced stage, this indicator was significantly higher ($p<0.001$). This allows us to say that with severe MS, the preload increases sharply as a result of an increase in venous blood flow. Such a change indirectly confirms a gradual decrease in elasticity and worsening of diastolic relaxation with an increase in the severity of MS [1,7].

The described changes in the transmitral blood flow indicate an increase in LV stiffness, an increase in the hemodynamic load of the LA, and impaired LV diastolic function already in the early stages of MS and an increase in this process as the disease progresses [11].

The pathogenesis of the development of DD is possible as follows. So Sabbar H.N. [14] connects worsening of LV diastolic stretch with an increase in myocardial wall tension, due to increased sympathetic influences. Other authors, in particular Barbieri A. et al. [7] believe that in patients with MS in the presence of AH, chronic pressure overload can increase the content of myocardial collagen even before the development of a hypertrophic process, which affects the function of myocardial relaxation.

We established a direct correlation of LVMI with systolic BP (SBP) ($r=0.38$, $p<0.01$) and diastolic BP (DBP) ($r=0.35$, $p<0.01$). There was also a direct correlation between LVMI and LV dimensions in diastole - LV EDD ($r=0.43$, $p<0.01$) and LV EDV ($r=0.41$, $p<0.01$). In systole, these coefficients were significantly lower for both LV ESV ($r=0.32$, $p<0.01$) and LV ESV ($r=0.30$, $p<0.01$). Apparently, LVH affected diastolic rather than systolic LV parameters to a greater extent.

We also established a clear dependence of the nature of LV diastolic filling on the degree of its hypertrophy. There was a direct correlation between

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LVMI and IVVR (r=0.52, p<0.01). In addition, LVMI was directly correlated with A (r=0.37, p<0.01) and B3 (r=0.31, p<0.01) and inversely correlated with E (r=-0.31, p< 0.01), E/A (r= -0.44, p<0.01). This confirms the results of other authors who have determined a close relationship between the degree of LVH and the severity of diastolic myocardial dysfunction [2,5].

CONCLUSION

In patients with clinically pronounced MS, an increase in LV end diastolic size, LV end diastolic volume, LV end systolic size and LV end systolic volume was revealed, which indicates the presence of LV hypertrophy and its dilatation. In early-stage MS patients, similar changes are much less pronounced and there are no signs of LVH. However, LV diastolic dysfunction is observed already in the initial stage of MS, which increases with the progression of the disease. LV diastolic dysfunction precedes cardiac remodeling in patients with MS.

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