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DIASTOLIC DYSFUNCTION AND REMODELING LEFT VENTRICLE DEPENDING ON THE CONTROL GLYCEMIA IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT
It is proved that type 2 diabetes mellitus (DM), the incidence of which has been continuously increasing in recent years, is an independent risk factor for the development of coronary heart disease (CHD). The presence of DM in patients with coronary heart disease significantly increases the likelihood of developing chronic heart failure (CHF), and is also a predictor of death in patients with CHF. At the same time, it was shown that 30-50% of DM patients with a transient decrease in local myocardial contractility do not have hemodynamically significant lesions of the coronary arteries, which indicates the presence of a specific myocardial condition – diabetic cardiomyopathy. Its existence was confirmed by the Framingham study, in which revealed a large thickness and mass of the myocardium of the left ventricle (LV) in patients with DM. It is believed that an increase in the diastolic stiffness of the myocardium is one of the characteristic features of diabetic cardiomyopathy, since disorders of LV diastolic function are its earliest manifestations. To date, numerous studies have proven the effectiveness of strict glycemic control in reducing the risk of development and progression of various complications of diabetes.

Keywords: type 2 diabetes mellitus, diabetic cardiopathy, arterial hypertension.

Introduction

The aim of the study was to study the influence of control glycemia on the development of diastolic dysfunction and LV remodeling in DM patients with the absence or presence of concomitant arterial hypertension.

Material and Methods

56 patients with DM aged from 26 to 66 years (mean age 46.3 ± 8.4 years) were examined. In 32 (56.4%) patients arterial hypertension (AH) was detected with DM, among which there were 13 (42.7%) with grade 1 and 18 (57.3%) patients with grade 2 of the disease. The average duration of DM was less than 5 years in 28 (50.4%) patients, from 5 to 10 years – in 12 (21.1%), more than 10 years – in 1 (0.7%). In 3 (6%) patients, the experience of DM could not be established, in 12 (21.8%) patients, DM was detected for the first time.

As a criterion for compensation of carbohydrate metabolism in patients with diabetes, according to the recommendations of the European Bureau the glycosylated hemoglobin (HbAlc) index was used by the International Diabetes Federation and the WHO European Bureau. It was revealed that 61 (45.9%) patients with DM had a low risk of vascular

complications (HbAlc ≤ 6,5%), 12 (9%) – risk of developing macroangiopathy (HbAlc > 6,5%), 60 (45,1%) patients are at risk of developing microangiopathy (HbAlc >7.5%). There were no significant differences in the state of compensation depending on the presence of hypertension in patients with DM.

The criterion for exclusion from the study was the presence of coronary artery disease, for the diagnosis of which electrocardiography (ECG), bicycle ergometry, Holter ECG monitoring, echocardiography were performed.

To assess diastolic properties, the time of isovolumic relaxation (IVRT, ms), the time of slowing down of early filling (DT, ms), the ratio of peaks of the rates of early and late LV diastolic filling (E/A), the ratio of the maximum rates of systolic and diastolic antegrade blood flow in the pulmonary veins (S/D). Diastolic dysfunction was diagnosed according to the criteria of the working group of the European Society of Cardiology, depending on the age of patients: IVRT (less than 30 years) > 92 ms; IVRT (from 30 to 50 years) > 100 ms; IVRT (more than 50 years) > 105 ms; DT (less than 50 years) > 220 ms; DT (more than 50 years) > 280 ms; E/A (less than 50 years) < 1,0; E/A (more than 50 years) < 0.5; S/D (less than 50 years) > 1.5; S/D (more than 50 years) > 2,5. Three types of LV filling disorders were distinguished: with delayed relaxation, pseudonormal and restrictive, which correspond to a minor, moderate and severe degree of diastolic dysfunction.

In order to diagnose LV remodeling , the following were determined myocardial mass (MM) according to the formula R.B. Devereux: 0.8 × 1.04 × (final diastolic size + thickness of the interventricular septum + thickness of the posterior LV wall)3 – final diastolic dimension3) + 0.6; myocardial mass index (iMM) – by dividing MM by body surface area; index of relative wall thickness (IOTs) of the left ventricle – the ratio of the sum of the thickness of the interventricular septum and posterior wall to the final diastolic LV size. To evaluate the geometric model of LV, the classification of G. Ganau et al. was used: normal geometry – iMM ≤ 125 g/m2 for men and ≤ 110 g/m2 for women, and ABSENCE < 0.45; eccentric hypertrophy – an increase in iMM with normal IOTs; concentric remodeling – normal iMM, IOTs ≥ 0.45; concentric hypertrophy – an increase in iMM, and OTS = 0.4

The diagnosis of diabetic neuropathy was carried out on the basis of collecting anamnesis and identifying patient complaints, studying vibration, tactile and temperature sensitivity, determining the rate of propagation of excitation in various areas of the motor fibers of the nerves of the lower extremities on the Neuro-EMG-Micro apparatus. Three types of neuropathy were distinguished: mainly axonal, mainly demyelinating, mixed lesion of the nerve conductor. A total of 70 (52.6%) DM patients with nephropathy were identified, of which 57 (81.4%) with axonal, 8 (11.4%) with demyelinating and 5 (7.2%) patients with mixed type.

Results

In 24 (42.1%) of the examined DM patients, diastolic dysfunction was detected, among the types of which delayed LV relaxation prevailed – in 87.5% of cases. LV remodeling was observed in 27 (47.3%) patients with DM, of which eccentric hypertrophy prevailed – 58.7%. In 34.9% of patients with DM, concentric LV hypertrophy was detected, which has a more unfavorable prognostic value compared to other variants of LV remodeling. Diastolic

dysfunction (54.7% > 25.9%; p=0.0001) and LV remodeling (57.3% > 34.5%; p=0.01) were observed significantly more often in patients with DM and hypertension. The presence of hypertension was accompanied by a tendency to increase the development of pseudonormal type (12.2% > 6.7%), the appearance of a restrictive type of diastolic dysfunction, as well as an increase in cases of detection of concentric hypertrophy (39.5% > 25%) of LV.

The absence of DM compensation increased the frequency of diastolic dysfunction and LV remodeling. In patients with DM without hypertension and HbAlc level > 7.5%, compared with compensated patients, LV diastolic dysfunction was significantly more often detected (40% > 11.1%; p=0.03), and its more severe degree (pseudonormal type) was also observed. More frequent development of diastolic dysfunction during decompensation of DM without hypertension was accompanied by an increase in cases of concentric LV myocardial hypertrophy in patients with HbAlc levels > 6.5% (100% > 11.1%; p=0.04) and the HbAlc level > 7,5% (30% > 11,1%).

The increase in cases of diastolic dysfunction and cardiac remodeling in patients with diabetes mellitus and hypertension with decompensation was less obvious due to the contribution of hypertension to their development. However, in these patients with HbAlc levels > 6.5% and 7.5%, compared with compensated patients, pseudonormal type of diastolic dysfunction (20% and 14.3% > 6.7%) and concentric LV myocardial hypertrophy (40% and 47.4% > 31.6%) were more often detected. Thus, in patients with DM without hypertension, diastolic dysfunction and LV remodeling are often detected, which suggests the development of diabetic cardiopathy. The presence of hypertension in patients with diabetes exacerbates the course of cardiopathy, which leads to more severe remodeling of the heart. Decompensation of diabetes, both in the presence and absence of concomitant hypertension, is accompanied by an aggravation of the degree of diastolic dysfunction and LV remodeling.

The data accumulated in recent years indicate, that DM contributes to an increase in the risk of developing CHF, due, among other things, to diastolic dysfunction, which can be observed in the absence of hypertension and/or LV myocardial hypertrophy. Various mechanisms are assumed to be involved in the pathogenesis of LV diastolic dysfunction in DM – a decrease in the activity of sarcoplasmic Ca2+-ATPASE, which leads to a slowdown in the extradition of Ca2+ ions from the cytoplasm during diastole, as well as activation of kinase C, provoking the development of myocardial hypertrophy. The main role in the appearance of LV diastolic dysfunction is probably played by metabolic disorders in DM – glucose deficiency as an energy substrate and the transition of intracellular metabolism in the heart muscle to the oxidation of free fatty acids, leading to a decrease in ATP content and the accumulation of undesirable metabolites in the cardiomyocyte. It was found that an increase in HbAlc levels is associated with an increased risk of heart failure regardless of the presence of diabetes. The lack of compensation for diabetes is also associated with the risk of cardiovascular autonomic neuropathy, which is manifested by a decrease in the density of adrenergic innervation of the heart. A violation of the capture of metaiod-benzylguanidine (an analogue of norepinephrine) by postganglionic sympathetic fibers in DM was found. In turn, a violation of sympathetic innervation of the myocardium correlates with a decrease in the reserve of LV contractility in DM. Thus, cardiovascular autonomic neuropathy, the development of which depends mainly

on the compensation and duration of DM, is associated with the occurrence of LV diastolic dysfunction. In this study , the increase in the development of diastolic dysfunction and LV remodeling in patients with decompensated DM was accompanied by more frequent detection of demyelinating neuropathy. demyelinating neuropathy was significantly more often diagnosed in patients with DM with HbAlc level > 7.5%, both without hypertension (28.6% > 0%; p=0.03) and in its presence (16% > 0%; p=0.04).

It is known that hypertension often accompanies diabetes, which is accompanied by a decrease in life expectancy and an increase in the risk of premature death. There is an opinion that cardiac remodeling is a universal mechanism for the progression of all heart diseases, including hypertension, without exception. It is believed that under the influence of various stimuli (increased hemodynamic stress, effectors of hemodynamic systems), active relaxation of cardiomyocytes is disrupted and myocardial stiffness increases. This study shows that the frequency of detection and severity of diastolic dysfunction and LV remodeling in patients with DM depends not only on the presence of concomitant hypertension, but to a greater extent on the compensation of diabetes.

CONCLUSIONS

The lack of glycemic control (HbAlc level > 7.5%) in patients with type 2 diabetes mellitus, regardless of the presence of concomitant arterial hypertension, leads to aggravation of diastolic dysfunction and LV remodeling against the background of increased development of demyelinating diabetic nephropathy.

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