

could be attributed to the small sample size and the high level of cardiovascular disease risk at baseline.

**Disclosure of interest** The authors declare that they have no competing of interest

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## Analysis of the associations between anthropometric parameters, arterial hypertension and dyslipidemia among type 2 diabetic patients



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**Aim** To assess the relationships between anthropometric parameters, arterial hypertension and dyslipidemia in patients with type 2 diabetes mellitus.

**Methods** Our cross-sectional study included 457 patients with type 2 diabetes mellitus. We measured the systolic and diastolic blood pressures, weight, height and waist circumference (WC). Body mass index (BMI) and waist to height ratio (WHtR) were calculated. We performed ROC curves for each anthropometric parameter to assess its discriminant power to distinguish between hypertensive and normotensive diabetic patients and between patients with and without dyslipidemia.

**Results** Obesity, arterial hypertension and dyslipidemia were observed in 49.2%, 61.1% and 67.1% of diabetics. In diabetic women, blood pressure, total cholesterol and triglyceride levels were significantly correlated with studied anthropometric parameters. In contrast, in diabetic men, a weak correlation between diastolic blood pressure and BMI was observed. Weight, BMI, WC and WHtR had areas under the curve between 0.543 and 0.664. Cut-off values of anthropometric parameters for predicting the presence of high blood pressure or dyslipidemia were higher in women than in men.

**Conclusion** In our study, anthropometric parameters had a poor discriminant power in the distinction between hypertensive and normotensive patients and between patients with and without dyslipidemia. However, the parameters evaluating visceral fat seemed to have relatively better discriminative power.

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## Correlations between B type natriuretic peptide and blood pressure in patients with type 2 diabetes mellitus



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**Aims** Brain natriuretic peptide (BNP) plays key roles in regulating blood pressure, fluid and electrolyte balance. The aim of our study was to assess correlations between serum BNP levels and blood pressure among patients with type 2 diabetes mellitus (T2DM).

**Methods** We conducted a cross-sectional study among patients with T2DM below the age of 65 years regardless of duration of diabetes and type of antidiabetic drugs. Detailed medical history, measurements of systolic (SBP), diastolic blood pressure (DBP) and serum BNP were recorded. Patients with BNP > 300 pg/mL were excluded. The included patients were divided into four groups according to BNP quartiles: Q1 < 9; Q2 between 9 and 11.9; Q3 between 11.9 and 32.2; Q4 ≥ 32.2 pg/mL. Analysis of group differences was performed using the non-parametric Kruskal-Wallis test for continuous variables. and chi<sup>2</sup> test for trend for categorical variables. The comparison of percentages on independent series was carried out by the Pearson's chi<sup>2</sup> test, and in the case of invalidity, the bilateral Fisher's exact test was used.

**Results** We included 69 diabetics, mean age: 56.7 ± 6.9 years old, sex ratio (M/F): 1.5, average duration of diabetes of 10.6 ± 7.7 years and average HbA<sub>1c</sub>: 9.2 ± 1.9%. The mean level of serum BNP was 27.8 ± 32 pg/mL. More than half (61%) of the patients were hypertensive. Serum BNP was significantly higher among hypertensive patients than non-hypertensive patients (35.6 ± 5.7 vs. 15.7 ± 17.6 μg/mL, P=0.001). We did not observe statistically significant correlation between the BNP level on the one hand and the SBP and the DBP on the other hand. Fourth quartile patients were 6.9 times more likely to have high blood pressure (95% CI [1.4, 33.5]) than patients in the first three quartiles (P=0.008). Patients in the 2nd quartile had significantly higher SBP than patients of the other quartiles (P=0.049). The DBP was comparable between the four groups (P=NS).

**Conclusion** The relationship between high blood pressure and BNP was not significant among diabetic patients of our study. But it appears that a BNP level ≥ 32.2 pg/mL was a significant predictor of high blood pressure.

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## Biomarkers of prehypertension and hypertension in women



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**Background** The percentage of undiagnosed prehypertensive and hypertensive women are high in the population of Kazakhstan.

**Aims** Our study assessed biomarkers in prehypertension and hypertension in women.

**Methods** 203 women were divided into 3 groups: "prehypertensive" group (n=30, 46.6 ± 6.2 y.o.) with a systolic blood pressure from 120 to 139 mmHg or/and a diastolic blood pressure from 80 to 89 mm Hg. In the "hypertensive" group the blood pressure was >= 140/90 mmHg (n=73, 46.6 ± 5.6 y.o.). In the normotensive group the blood pressure was <= 120/80 mm Hg (n=100, 44.2 ± 5.9 y.o.). The MILLIPEX MAP Human CVD 1 Magnetic Bead kit was used for the quantification of cardiovascular biomarkers in venous samples. The quantification of the level of cardiac markers was carried out on the Bio-Plex<sup>®</sup> 3D multiplex analyzer Suspension Array System (Bio-Rad Laboratories, Inc., USA).

**Results** The values of brain natriuretic peptide (BNP), creatine kinase-MB (CK MB), and leptin were statistically increased in women depending on their blood pressure. The BNP values in the hypertensive group (Me = 157.9 pg/ml, Q<sub>1</sub> = 53.75 pg/ml and Q<sub>3</sub> = 229.6 pg/ml) were statistically greater (p=0.016) than in the normotensive group (Me = 85.14 pg/ml, Q<sub>1</sub> = 18.55 pg/ml and Q<sub>3</sub> = 113.9 pg/ml). The BNP values in the prehypertension group were greater than in the normotensive group and less

than in the hypertensive group (Me = 73.00 pg/ml,  $Q_1$  = 35.52 pg/ml and  $Q_3$  = 139.4 pg/ml). The CK MB values in the hypertensive group (Me = 3693 pg/ml,  $Q_1$  = 3105 pg/ml and  $Q_3$  = 6178 pg/ml) were statistically higher ( $p=0.022$ ) than in the normotensive group (Me = 2688 pg/ml,  $Q_1$  = 1629 pg/ml and  $Q_3$  = 4532 pg/ml). The CK MB values in the prehypertension group were greater than in the normotensive group and less than in the hypertensive group (Me = 2722 pg/ml,  $Q_1$  = 1833 pg/ml and  $Q_3$  = 5266 pg/ml). In addition, the values of leptin in the hypertensive group (Me = 23910 pg/ml,  $Q_1$  = 14510 pg/ml and  $Q_3$  = 54824 pg/ml) were statistically greater ( $p=0.001$ ) than in the normotensive group (Me = 18256 pg/ml,  $Q_1$  = 9287 pg/ml and  $Q_3$  = 34843 pg/ml). The leptin values in the prehypertension group were: Me = 26424 pg/ml,  $Q_1$  = 10514 pg/ml and  $Q_3$  = 53060 pg/ml.

**Conclusion** In hypertensive women, serum BNP, CK-MB and leptin levels were significantly different from the same parameters in control women. Pre-hypertensive patients were in-between the normotensive and the hypertensive groups. However, biomarkers were not statistically different in women with prehypertension and those from the two other groups. Thus, patients with prehypertension cannot longer be attributed to healthy individuals although, they have not yet pronounced clinical and laboratory signs of arterial hypertension, the mechanisms of arterial hypertension are already running. Our results showed that there is need to develop recommendations for the identification and monitoring of patients with pre-hypertension in order to prevent the development of irreversible cardiovascular alterations.

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## Sleep apnea syndrome and high blood pressure



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**Introduction** High blood pressure (HT) and abdominal obesity are frequently associated with sleep apnea syndrome (SAS). HT is considered as a consequence of the SAS and obesity is recognized as a contributing factor. It is often recommended to look for a SAS in hypertensive patients resistant to antihypertensive treatment.

**Patients and methods** Five patients, 2 men and 3 women, with resistant HT requiring at least triple therapy, were hospitalized in our department.

**Results** The average age was 57.26 years old, all patients were severely obese; average BMI: 44 kg/m<sup>2</sup> (39–55 kg/m<sup>2</sup>). The diagnosis of HT was made before that of SAS in all patients. All patients were diabetic and had a metabolic syndrome, 2 patients suffered of hypothyroidism. The SAS was severe in 4 cases and the equipment at home improved the blood pressure profile with a decrease in the number of antihypertensive treatments in all patients and completely stopped the treatment in 1 case.

**Conclusion** These data enhance the search for SAS by sleep recording that must be proposed in patients with obesity and a treatment-resistant HT since its management makes possible the improvement of the blood pressure profile and may prevent cardiovascular complications.

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## Correlation between Diastolic Function and left ventricular global longitudinal stress (GLS) decline in Hypertensive Patients



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**Introduction** Heart failure with preserved LVEF is a major cause of morbidity and mortality in hypertensive patients. The close correlation between the impairment of diastolic function and the longitudinal systolic dysfunction has several possible explanations. First the diastole is an energy-dependent process, especially during its first phase, it also includes active systolic components during the isovolumetric relaxation phase; in addition, the achievement of intrinsic myocyte function is part of hypertensive pathology as evidenced by recent studies.

**Methods** Echocardiography was performed in a series of 200 hypertensive patients (25 to 75 years-old). The left ventricular ejection fraction was calculated by biplane method, the indexed left ventricular mass and the relative wall thickness were measured or calculated. In order to differentiate eccentric and concentric LVH, diastolic function analysis and finally study of the LV longitudinal deformation by the speckle tracking technique (GLS calculation) were performed. Patients with secondary hypertension, valve insufficiency or stenosis, arrhythmia, history of coronary insufficiency were excluded from this study.

**Results** One hundred and seventy-eight patients (89%) of the 200 hypertensive patients had diastolic function impairment, of which 37 patients (18.5%) had high filling pressures. Mean arterial blood pressure was statistically higher in hypertensive patients with high filling pressures than in hypertensives with normal filling diastolic LV pressure. 30 of 37 patients with high filling pressure had low GLS. We reported a significant relationship between the decrease in GLS and the increase in the left ventricular filling pressure. These results suggest that the increase in filling pressures is closely associated with the atrioventricular interaction in hypertensive patients, with a significant correlation with the impairment of longitudinal systolic function and diastolic function.

**Discussion and conclusion** When myocardial fibrosis develops, collagen accumulation increases myocardial rigidity and induces diastolic dysfunction; it also deprives cardiomyocytes of the skeleton necessary for myocardial contraction. Therefore, the degree of myocardial fibrosis can be associated with the defects of systolic and diastolic properties. LVEF is a poorly sensitive indicator of myocardial contractility and subclinical alterations in LV systolic function are already evident in heart failure with preserved LVEF. The decline in the longitudinal function index, despite a preserved LVEF, is well correlated with the increase in filling pressures in hypertensive patients with LVH. The association between HT with LVH and the deterioration of asymptomatic diastolic function is well recognized. A linear relationship is thus established between the increase in left ventricular mass, diastolic dysfunction and LV systolic dysfunction. Finally, the detection of longitudinal systolic dysfunction in an hypertensive patient should impose a therapeutic prevention strategy with the objective of delaying the onset of symptomatic heart failure.

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