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The Relationship Between Elevated Pulse Pressure and Natriuretic Peptide

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Abstract: Pulse pressure (PP) measured as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP) increases markedly after the age of fifty because of the increase of arterial stiffness with age which leads to progressive increase of SBP and decrease of DBP. Natriuretic hormones are used as biological markers in the early diagnosis of chronic heart failure (CHF) and can also be used in routine practice simply by determining pulse pressure, i.e. increased BP indicates arterial stiffness, ventricular relaxation is impaired and diastolic heart dysfunction (DHD) develops. The diagnosis of diastolic dysfunction is difficult to determine as it is almost asymptomatic and patients come to the doctor at a later stage when systolic circulatory failure develops. The aim of the study was to determine the association between elevated PP and the level of brain natriuretic peptide (BNP) in plasma.

Keywords: pulse pressure, heart failure, natriuretic peptide, systolic blood pressure, diastolic blood pressure.

Introduction. Pulse pressure (PP), measured as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP), increases markedly after the age of fifty, due to increased arterial stiffness with age, resulting in a progressive increase in SBP and decrease in DBP [2,3,12]. With decreased aortic elasticity, arterial vascular stiffness increases and this increases postload and myocardial oxygen demand and impairs ventricular relaxation [2,12]. It can be speculated that pulse pressure, a measure of arterial stiffness, can predict congestive heart failure (CHF) in the elderly [7,12,16]. And very often in patients with elevated pulse pressure, when plasma B-group natriuretic peptide was detected, this marker was elevated [5,6,14].

Natriuretic hormones are used as biological markers in the early diagnosis of chronic heart failure (CHF) and can also be used in routine practice simply by determining pulse pressure, i.e. increased AP indicates arterial stiffness, impaired ventricular relaxation and development of diastolic heart dysfunction (DHD). Diastolic dysfunction is difficult to diagnose as it is virtually asymptomatic and patients consult a physician at a later stage when systolic circulatory failure has developed [6,7]. The significance and role of DD in CHF syndrome have become established only within the last three decades.

Brain natriuretic peptide and biologically active peptide BNP and N-terminal fragment of peptide precursor-NTproBNP are commonly used for diagnosis of CHF. The role of these peptides as prognostic markers in relation to mortality from cardiovascular complications has also been determined [1,2,8].

The main stimulus for the release of BNP (brain natriuretic peptide) is an increase in myocardial tension as a result of increased end-diastolic pressure (EDP) in the left ventricle (LV) [10,16]. In asymptomatic LV dysfunction, increased concentration of INP and NTproBNP inhibits the effects of renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system (SNS). Blocking the effects of BNP and NTproBNP leads to the development of clinically evident CHF. The effects of natriuretic hormones on the kidneys diminish with the progression of CHF, leading to sodium and water retention and a further deterioration of cardiac function [7,13].

Purpose: The aim of the present study was to evaluate BNP concentrations in blood in patients with elevated pulse pressure.

Materials and methods of the study. The study was conducted in the 1st Clinic of Samarkand State Medical University, in Cardiology Department. 106 patients aged 59 to 88 years old, 62 men and 44 women without clinical signs of CHF were studied. The patients were divided into 2 groups: 1 group of patients with elevated pulse pressure, whose PP was higher than borderline value (≥ 56 mmHg) comprised 58 patients and 2 group of patients with normal pulse pressure without PP increase (less than 56 mmHg) comprised 48 subjects (78.9%). Patients who had had a myocardial infarction within 30 days before the study, patients with low CVF, patients with prosthetic valves, implanted cardioverter-defibrillators, pacemakers; patients without severe renal, hepatic or respiratory failure, as well as patients with oncological diseases were not included in the study. All patients underwent a complex of examinations: laboratory - general blood count, urine, biochemical blood count, coagulogram, cholesterol, all types of lipoproteins, level of BNP in blood plasma; instrumental: ECG, EchoCG, chest X-ray, Holter ECG, SMAD, etc. Blood pressure was measured in all patients and we measured pulse pressure according to systolic blood pressure (SBP) and diastolic blood pressure (DBP). Pulse pressure was determined as SAP - DAP. At baseline exercise 12 patients (21.1%) in Group 1A and 1B had a PP greater than borderline (≥ 56 mmHg),

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Normal resting pulse pressure in healthy adults sitting upright is approximately 30-40 mmHg. Pulse pressure increases with exercise due to an increase in stroke volume, with a healthy person having a pulse pressure of about 100 mmHg. In healthy people, pulse pressure usually returns to normal within about 11 minutes. Using a standard mercury sphygmomanometer, trained interviewers took 3 BP measurements at 30-second intervals after the patient had spent at least 5 minutes in a sitting position

BP should be equal in both arms or the difference should not exceed 5 mm. Due to asymmetric musculature development, the right arm tends to have a higher pressure. A difference of 10 mm indicates a probable atherosclerosis, whereas 15-20 mm indicates stenosis or anomalies of the great vessels. The normal pulse pressure is 35+-10 mmHg (up to 35 years of age 25-40 mmHg, older people up to 50 mmHg). Lowering it may be caused by the heart failure (heart attack, tamponade, paroxysmal tachycardia, atrial fibrillation) or by a sudden jump of vascular resistance. High (over 60) pulse pressure reflects atherosclerotic changes of arteries, heart failure. Can occur in endocarditis, pregnant women, anaemia, intracardiac blockages.

Blood was drawn from a forearm vein in the morning on an empty stomach in the procedure room and BNP content was determined in whole blood immediately after collection.

Echocardiographic examination was performed according to the recommendations of the American Society of Echocardiography.The parameters of one- and two-dimensional echocardiography and Doppler echocardiography were assessed. LVEF was estimated by Simpson method, left ventricular myocardial mass (LVMM) was calculated by cube of linear dimensions simulating LV as elongated ellipse, left atrial volume - by area-length method. Central hemodynamic indices were assessed by continuous-wave Doppler echocardiography [6], types of LV remodeling according to A. Ganau et al. (7).The main indicator of DDS is the decrease of E/A on EchoCG less than 1.0.

E/A is the ratio of maximal blood flow velocity during early diastolic filling (E) to maximal flow velocity during atrial systole (A).

Several questions had to be addressed regarding our study methods. Brachial artery BP using sphygmomanometry is less accurate in measuring central pressure than more invasive or technologically advanced methods. However, this type of BP measurement is easy to obtain in a clinical setting and in older people, such as ours, peripheral and central BP correlate better than in younger people.

Table 1

№	Parameters and their normal value	1-group (n=58)	2-group (n=48)
1	SAP	165 (145-188) mm Hg.	138 (125-146) mm Hg.
2	DAP	82(78-92) mm Hg	92 (78-96) mm Hg.
3	PP	56 (50-68)	48 (42-56)
4	BNP (пг/мл)≤ 100	183 (113-667) pg/ml	83 (75,8-96,2) pg/ml. < 0,001
5	EDV ml 110—145	153(125—188)	118 (89—147) < 0,001
6	ESV ml 45—75	98(74—115)	73 (51—82) < 0,001
7	EF % 55—65	58 (56—62)	62 (55—66) < 0,001

Results and discussion. The BNP level in patients with increased pulse pressure regardless of EF was 183 (113-667) pg/ml which was significantly ($p < 0.001$) higher than BNP level in the control group of 83 (75.8-96.2) pg/ml. The comparison of patient groups revealed significant differences in values of end-diastolic volume (EDV), end-systolic volume (ESV) and LV EF. The concentration of BNP in blood is higher in patients with increased PP than in patients with normal PP.

It is known that the main stimulus for BNP secretion is mechanical stretching of cardiomyocytes. It can be assumed that these differences are a consequence of the pathogenesis and the presence of multidirectional changes in the heart. As a rule, in such patients there is an increase of end-diastolic tension of LV wall at its insignificant dilatation [9, 10], while at decrease of EF there is a significant dilatation and overload of the left ventricle by volume.

The neurohumoral regulation of hemodynamics is realized primarily through changes in heart rate and lumen of resistance vessels, which leads to changes in the value of life-supporting blood volume. Thus, in the circulatory process the AP plays the role of the operator of this regulation, as it is the functional reflection of the pulse blood volume, and systolic and diastolic pressure are its derivatives. This position can be confirmed analytically.

Pulse pressure, an easily measured correlate of arterial stiffness and pulsatile haemodynamic load, was an independent predictor of heart failure risk in this study. Pulse pressure was the single most informative BP parameter, as it best reflects the physiological consequences of arterial stiffness, expressed as disproportionately elevated BP, decreased DAP or both. These findings support the hypothesis that the relative importance of BP components in predicting cardiovascular risk is age dependent, with BP becoming the best marker of vascular overload in older adults.

The study found that cerebral natriuretic peptide concentrations were higher in patients with elevated BP than in those with normal BP, and reflected the severity of the pathological process.

It should be emphasized that elevated AP was associated with the presence of restrictive left ventricular blood flow abnormalities.

Conclusion: Pulse pressure is thus an easy to measure factor that is an independent predictor of the risk of heart failure. Arterial stiffness increases with age and pulse pressure, can predict latent congestive heart failure.

Pulse pressure and plasma BNP may be clinically useful in identifying older people at risk of heart failure who may benefit from treatment to improve arterial stiffness. More research is needed to further investigate the relationship between arterial pliability and its cardiovascular consequences, and the possible benefits of therapy specifically designed to prevent or treat vascular pathology associated with elevated pulse pressure.

These findings have potential preventive and therapeutic implications with regard to the possibility of altering arterial stiffness and possibly the risk of heart failure.

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