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ORIGINAL ARTICLE

Individuals with high-normal blood pressure have different metabolic and haemodynamic characteristics to those with optimal blood pressure

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Abstract

Background: The ESH classification of blood pressure includes the high-normal blood pressure (HNBP) category, which is within normal limits but associated with increased cardiovascular (CV) risk.

Aim: To identify additional CV risk factors and early signs of target organ damage in healthy individuals with HNBP.

Methods: Healthy volunteers (n = 74) with optimal blood pressure or HNBP were compared with respect to metabolic and haemodynamic parameters.

Results: The HNBP was associated with higher serum uric acid (333.1 ± 65.4 *vs* 267.7 ± 65.4 μmol/L, $p < 0.05$) and glucose (4.7 ± 0.3 *vs* 4.5 ± 0.3 mmol/L, $p < 0.01$) concentrations, intima-media thickness (0.39 ± 0.06 *vs* 0.36 ± 0.04 mm, $p < 0.05$), systemic vascular resistance index (2,678.2 ± 955.9 *vs* 1,930.2 ± 625.5 dyn × s × m^2/cm^5, $p < 0.001$), lower total arterial compliance index (1.04 ± 0.42 *vs* 1.44 ± 0.48 mL/[mm Hg × m^2], $p < 0.01$) and baroreflex sensitivity (14.2 ± 3.8 *vs* 18.0 ± 8.8 mm Hg^2/Hz, $p = 0.05$).

Conclusions: The observed differences in metabolic and haemodynamic profile in HNBP may adversely affect CV risk in these individuals.

Key words: high-normal blood pressure, cardiovascular risk factors, target organ damage, baroreflex sensitivity, impedance cardiography, intima-media thickness, uric acid

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INTRODUCTION

Although within normal limits, high-normal blood pressure (HNBP, i.e. systolic blood pressure of 130–139 and/or diastolic blood pressure of 85–89 mm Hg) is associated with increased cardiovascular (CV) risk. It has been demonstrated that CV mortality increases from blood pressure values of 115/75 mm Hg, doubling with each increment of 20/10 mm Hg [1]. Observational studies have shown that individuals with HNBP have a five-fold increased risk for the development of hypertension and CV events than those with optimal blood pressure (OBP < 120/80 mm Hg) [2, 3]. Furthermore, studies of HNBP treatment, though not free from limitations, seem to indicate that lowering blood pressure in this group is beneficial [4–7]. The increased CV risk associated with HNBP makes it necessary to identify other factors contributing to the elevation of blood pressure and the development of athe-

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rosclerosis. The haemodynamic profile of the CV system can be evaluated by impedance cardiography. Impedance cardiography can be helpful in estimating CV risk and the choice of treatment in hypertensive patients [8]. Increased activity of the sympathetic nervous system is thought to contribute to the elevation of blood pressure. This effect is especially pronounced in the early stages of hypertension development. Measurement of baroreflex sensitivity (BRS) is an established method for estimating sympathetic activity. Intima-media thickness (IMT) is used as an index of target organ damage in hypertension. IMT is a strong prognostic factor for CV events. Our study compared the CV profiles of subjects with OBP and HNBP. We hypothesised that, given the increase in CV risk associated with HNBP, there may be concurrent differences in blood pressure regulation, as well as haemodynamic and metabolic parameters.

METHODS

The experimental protocol was approved by the institution's Ethics Committee, and all participants expressed their written consent to take part in the study. Healthy volunteers (n = 74; 28 female, 46 male) aged 20–39 (median 23 years) were included in the study. A detailed medical history, physical examination, and office blood pressure measurements were obtained from all participants. Subjects were allocated to the OBP or the HNBP group in accordance with European Society of Hypertension (ESH) guidelines, based on the mean value of three separate blood pressure measurements following established protocols [9]. In order to exclude subjects with masked hypertension, 24-hour ambulatory blood pressure measurements (ABPM, SpaceLabs 90207) were conducted in all subjects. Participants were instructed to follow their regular daily activities during ABPM recording. A fasting blood sample was obtained for measurement of lipid profile and glucose, uric acid, and high-sensitivity C-reactive protein (hsCRP) concentrations. The IMT was assessed by B-mode ultrasound (GE Vivid V). Measurements were obtained in the longitudinal plane, bilaterally from the far wall of the common carotid artery, within 1 cm proximally to its bifurcation. The mean of ten measurements (five each from the left and right sides) was used to calculate IMT. Impedance cardiography was performed using a CAVASCREEN cardiograph (ITAM, Zabrze, Poland). All measurements were obtained in the supine position, after a 15-min rest.

The following parameters were measured: cardiac output (CO, L/min), cardiac index (CI, L/min/m^2), and thoracic fluid content (TFC, Ohm^{-1}). Systemic vascular resistance index (SVRI, dyn × s × cm^5 × m^2) was calculated according to the formula SVRI = (MAP – CVP)/CI × 80, assuming a central venous pressure (CVP) of 4 mm Hg (MAP, mean arterial pressure) [10]. Following Windkessel's two-element model of vascular branching, total arterial compliance index (TACI, mL × mm Hg^{-1} × m^2) was calculated as the ratio of stroke index (SI, mL/m^2) to pulse pressure (PP, mm Hg): TACI = SI/PP. Baroreflex control of the heart rate or BRS was measured with the non-invasive sequence and spectral method (Nevrokard BRS software, version 5.1.3, Nevrokard, Ljubljana, Slovenia) after 15 min of blood pressure and heart rate recording. For spectral analysis, two discrete frequency bands were distinguished: high frequency (HF, 0.15–0.60 Hz) and low frequency (LF, 0.02–0.15 Hz), and their respective power spectral densities (PSD) were calculated. Quantitative BRS was evaluated by the square of the coherence value of blood pressure and R-R interval. The alpha indices (alpha-LF and alpha-HF) were calculated as the square root of the ratio of blood pressure PSD to R-R interval PSD in the respective frequency bands. Statistical analyses were performed using SAS® software (v. 9.2). Groups were compared using Student's t-test for quantitative variables, and the χ^2 test for qualitative variables. Correlations between quantitative parameters were evaluated by Pearson's linear correlation. General linear models were used to assess the association between independent variables (group [OBP/HNBP] and gender) and dependent variables (CI, SVRI, TACI, TFC). Potential confounders taken into account included body mass index (BMI) and age [11].

RESULTS

The baseline characteristics of the study groups are presented in Table 1; 75% of the patients in the HNBP group and 72% in the OBP group had a positive family history of hypertension. There were 29% and 18% smokers in HNBP and OBP, respectively. No significant differences were present with reference to presented values. Subjects with HNBP were found to have higher concentrations of uric acid (333.1 ± 65.4 *vs* 267.7 ± 65.4 μmol/L, $p < 0.05$), triglycerides (1.2 ± 0.5 *vs* 0.8 ± 0.4 mmol/L, $p < 0.01$) and glucose (4.7 ± 0.3 *vs* 4.5 ± 0.3 mmol/L, $p < 0.01$). No significant differences were observed with respect to concentrations of HDL and LDL cholesterol, hsCRP, and creatinine, or glomerular filtration rate. The results of biochemical assays are included in Table 2. Intima-media thickness did not exceed normal values (≤ 0.9 mm) in any subject. Mean IMT was significantly higher in the HNBP group than in the OBP group (0.39 ± 0.06 *vs* 0.36 ± 0.04 mm, $p< 0.05$). Haemodynamic examination revealed that subjects with HNBP had a higher mean SVRI than those with OBP ($p < 0.001$) (Fig. 1), as well as a lower mean CI ($p < 0.05$). In multivariate analysis including gender, age, and BMI, these differences remained significant only for SVRI. There was no significant difference in heart rate between the groups (66.2 ± 10.2 *vs* 68.2 ± 6.9/min, OBP *vs* HNBP). Moreover, the HNBP group had lower TACI than OBP group (Fig. 2, 1.04 ± 0.42 *vs* 1.44 ± 0.48 mL/mm Hg × m^2], $p < 0.01$). The HNBP was also associated with higher TFC (46.3 ± 4.1 *vs* 42.8 ± 5.4 1/Ohm, $p < 0.01$) (Table 3). The differences in TACI and TFC remained significant after adjustment for gender, age, and BMI. Baroreflex sensitivity assessed by the sequential and spectral

Table 1. Clinic and ambulatory blood pressure monitoring values (ABPM) and anthropometric indices in the group with optimal blood pressure (OBP) and the group with high-normal blood pressure (HNBP)

	OBP	HNBP	P
Male/female	27/23	19/5	
Age	25 ± 4	25 ± 4	NS
Weight [kg]	68.2 ± 10.8	78.8 ± 12.7	< 0.001
Height [cm]	174.5 ± 8.3	177 ± 7.6	NS
BMI [kg/m^2]	22.3 ± 2.4	25.1 ± 3.0	< 0.001
BSA	1.8 ± 0.2	2.0 ± 0.2	< 0.01
WC [cm]	(M) 85.7 ± 5.7/(F) 72.6 ± 6.8	(M) 91.6 ± 7.4/(F) 80.5 ± 7.0	< 0.01/< 0.05
SBP [mm Hg]	115 ± 6	128 ± 6	< 0.001
DBP [mm Hg]	74 ± 5	83 ± 6	< 0.001
SBP day ABPM [mm Hg]	118 ± 9	132 ± 9	< 0.001
DBP day ABPM [mm Hg]	71 ± 5	75 ± 7	< 0.01

BMI — body mass index; BSA — body surface area; WC — waist circumference; SBP — systolic blood pressure; DBP — diastolic blood pressure; M — male; F — female

Table 2. Biochemical indices in patients with optimal (OBP) and high-normal blood pressure (HNBP)

	OBP	HNBP	P
Uric acid [μmol/L]	267.7 ± 65.4	333.1 ± 65.4	< 0.05
Creatinine [μmol/L]	70.7 ± 8.8	76.0 ± 8.8	NS
GFR [mL/min]	143.2 ± 30.6	151.3 ± 26.8	NS
Triglycerides [mmol/L]	0.8 ± 0.4	1.2 ± 0.5	< 0.01
LDL [mmol/L]	2.1 ± 0.7	2.5 ± 0.8	NS
HDL [mmol/L]	(M) 1.5 ± 0.3/(F) 1.7 ± 0.4	(M) 1.3 ± 0.3/(F) 1.8 ± 0.4	NS/NS
Glucose [mmol/L]	4.5 ± 0.3	4.7 ± 0.3	< 0.01
CRP [nmol/L]	14.9 ± 27.6	9.14 ± 7.9	NS

GFR — glomerular filtration rate; CRP-C — reactive protein; M — male; F — female

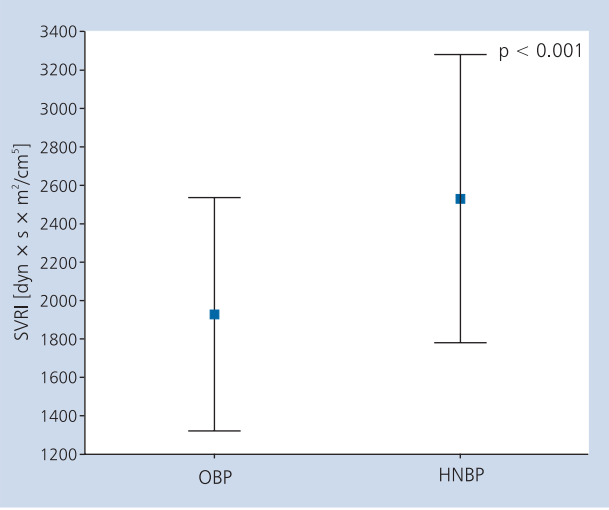

Figure 1. Systemic vascular resistance index (SVRI) values in high-normal blood pressure (HNBP) and in optimal blood pressure (OBP) patients (mean value, 95% CI)

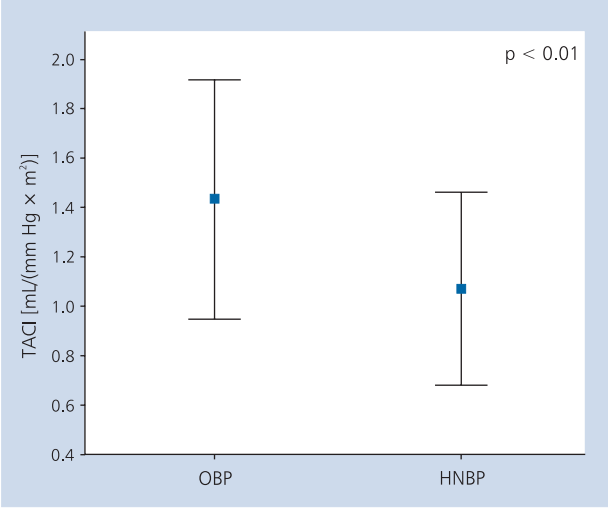

Figure 2. Total arterial compliance index (TACI) values in high-normal blood pressure (HNBP) and in optimal blood pressure (OBP) patients (mean value, 95% CI)

Table 3. Haemodynamic indices found during impedance cardiography in patients with optimal (OBP) and high-normal blood pressure (HNBP)

	OBP	HNBP	P
HR [1/min]	66.2 ± 10.2	68.2 ± 6.9	NS
CI [L/(min × m^2)]	3.6 ± 1.2	2.9 ± 0.8	< 0.05
SVRI [dyn × s × m^2/cm^5]	1,930.2 ± 625.5	2,678.2 ± 955.9	< 0.001
TACI [mL/(mm Hg × m^2)]	1.44 ± 0.48	1.04 ± 0.42	< 0.01
TFC [1/Ohm]	42.8 ± 5.4	46.3 ± 4.1	< 0.01

HR — heart rate; CI — cardiac index; SVRI — systemic vascular resistance index; TACI — total arterial compliance index; TFC — total fluid content

Table 4. Results of baroreflex sensitivity in patients with optimal (OBP) and high-normal blood pressure (HNBP)

	OBP	HNBP	P
BRS UP SBP [ms/mm Hg]	26.1 ± 10.6	23.3 ± 8.8	NS
Alpha LF [mm Hg^2/Hz]	18 ± 8.8	14.2 ± 3.8	0.05
Alpha HF [mm Hg^2/Hz]	32 ± 20.4	26.4 ± 10.2	NS
RRI LF/HF	1.3 ± 1.0	1.5 ± 1.5	NS
SBP LF/HF	3.6 ± 2.4	4.5 ± 3.2	NS

BRS — baroreflex sensitivity; SBP — systolic blood pressure; LF — low frequency; HF — high frequency; RRI — RR interval

methods was similar in both groups (Table 4), with the exception of the alpha-LF index, which was lower in HNBP than OBP (14.2 ± 3.8 *vs* 18.0 ± 8.8 mm Hg^2/Hz, p = 0.05).

DISCUSSION

The study demonstrates that individuals with OBP and HNBP differ with respect to certain biochemical and haemodynamic indices. Subjects with HNBP had higher concentrations of uric acid, triglycerides, and glucose. Retrospective analyses of data from the Framingham study have found that a high uric acid concentration is a significant predictor of CV events [12, 13]. There is also a strong correlation between hypertension and uric acid, though the underlying mechanism has not been unequivocally established [14]. Furthermore, it has been demonstrated that uric acid concentration correlates with other risk factors including microalbuminuria and increased IMT, both in healthy individuals and in those with prehypertension [15].

Significantly, higher uric acid concentrations have also been associated with the prevalence of metabolic syndrome, including in adolescent subjects [16]. The cross-sectional design of the current study prohibits the assessment of a longitudinal effect of increased uric acid concentration in HNBP on the development of hypertension. However, the significant difference between groups suggests that uric acid concentration could rise concomitantly with blood pressure, beginning at a young age, and the link between uric acid and the metabolic syndrome provides further evidence that these processes may play a common role in the pathogenesis of hypertension. In fact, HNBP was also found to be associated with increased glucose and triglyceride concentrations, but it should be noted that these associations were strongly affected by the differences in gender and BMI distribution between groups, and were not statistically significant after excluding the effects of these factors.

Impairment of the baroreceptor reflex is considered to play a role in the pathogenesis of hypertension and has been demonstrated in the early stages of hypertension [17, 18]. In our study, BRS was compared in two groups of healthy subjects in different blood pressure categories according to the ESH/ESC classification. Previous studies have found decreased BRS in subjects with higher blood pressure (mean SBP, 103 mm Hg *vs* 130 mm Hg), but did not take into account the current ESH/ESC classification [19]. Other studies have demonstrated differences in BRS between normotensive subjects and those with borderline hypertension (130–159/85–99 mm Hg) [20]. The current study also evaluated IMT, which is considered an indicator of early, preatherosclerotic changes in the arterial wall. The ESH/ESC criteria include an IMT of > 0.9 mm as a marker of subclinical organ damage.

Multiple studies have confirmed the correlation between pathologically increased IMT and other risk factors, as well as overall risk for development of coronary artery disease and stroke [9]. The current study demonstrates a significantly increased IMT in subjects with HNBP compared to those with OBP, although it must be noted that all values fell within normal limits. Other results support this finding, including a study with almost 900 participants which found increased IMT

in prehypertensives, compared to normotensives [21]. Men with HNBP were also found to have greater IMT than those with OBP [22]. The finding that carotid IMT is increased (though still normal) in a group of young, healthy individuals with HNBP indicates that they may be at increased risk for early development of vascular lesions and atherosclerosis. Furthermore, interesting differences in haemodynamic profiles were noted in subjects with HNBP. Increased SVRI could partially explain the higher CV risk in this group, as the prognostic value of blood pressure for CV risk has been associated with indices of vascular resistance [8]. Since multivariate analysis evaluating the confounding effect of gender revealed that the observed difference in CI is not statistically significant, this may simply reflect an uneven gender distribution. The correlative of arterial compliance, arterial stiffness, is an independent predictor of CV morbidity and mortality [8, 23].

The decreased TACI associated with HNBP in the present study suggests that early changes in vascular compliance may occur in subjects who are still classified as normotensive. The haemodynamic profile observed in the HNBP group is consistent with other studies in hypertensives and prehypertensives which have demonstrated increased vascular resistance and decreased arterial compliance in groups with higher blood pressure, despite similar CI values [24]. Thoracic fluid content is a measure of the amount of intra- and extracellular fluid in the anatomically defined thorax. The observed higher TFC in the HNBP group could indicate relative retention of fluids. Although few studies have assessed TFC, it has been found to be increased in patients with untreated hypertension [25]. Conversely, another study found that TFC was lower in hypertensives than in healthy subjects [26], but this was attributed to diuretic therapy and the older age of the hypertensive group. A potential limitation of the present study is that, although indexed measurement of SV/PP has been validated and compared to other methods for evaluating arterial compliance [27], the gold standard for studies of arterial compliance is currently measurement of pulse wave velocity by applanation tonometry [23]. The uneven distribution of men and women between groups is a further limitation of the study. This effect was addressed by the use of indexed parameters and multivariate analyses. It is, however, worth noting that the observed gender distribution is representative of the epidemiology of HNBP in the studied age group [28, 29]. In order to completely eliminate this difference, the sample size would have to be greatly increased.

CONCLUSIONS

Individuals with HNBP or OBP differ with respect to their biochemical, morphological, and haemodynamic profiles. Autonomic regulation of the CV system may also be different in these groups. The effect of individual factors on CV risk in HNBP should be evaluated in prospective studies.

Conflict of interest: *none declared*

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Osoby z wysokim prawidłowym ciśnieniem tętniczym posiadają odmienną charakterystykę metaboliczną i hemodynamiczną względem osób z optymalnym ciśnieniem tętniczym

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Streszczenie

Wstęp: Klasyfikacja ciśnienia tętniczego wg ESH wyróżnia ciśnienie „wysokie prawidłowe" (HNBP), które mieści się w granicach normy, lecz wiąże się z wyższym ryzykiem sercowo-naczyniowym.

Cel: Celem pracy było określenie dodatkowych czynników ryzyka sercowo-naczyniowego i wczesnych zmian narządowych u zdrowych osób z HNBP.

Metody: Porównano zdrowych ochotników (n = 74) z ciśnieniem optymalnym lub HNBP pod względem czynników metabolicznych i hemodynamicznych.

Wyniki: Osoby z HNBP wykazywały wyższe stężenia kwasu moczowego (333,1 ± 65,4 *v.* 267,7 ± 65,4 μmol/l, $p < 0,05$) i glukozy (4,7 ± 0,3 *v.* 4,5 ± 0,3 mmol/l, $p < 0,01$), wyższy wskaźnik intima-media (0,39 ± 0,06 *v.* 0,36 ± 0,04 mm, $p < 0,05$), wyższy wskaźnik obwodowego oporu naczyniowego (2678,2 ± 955,9 *v.* 1930,2 ± 625,5 dyn × s × m^2/cm^5, $p < 0,001$) oraz niższy wskaźnik całkowitej podatności naczyniowej (1,04 ± 0,42 *v.* 1,44 ± 0,48 ml/[mm Hg × m^2], $p < 0,01$) i niższą czułość odruchu z baroreceptorów (14,2 ± 3,8 *v.* 18,0 ± 8,8 mm Hg^2/Hz, $p = 0,05$).

Wnioski: Różnice w profilu metabolicznym i hemodynamicznym wykazane u osób z HNBP mogą się wiązać ze zwiększonym ryzykiem sercowo-naczyniowym w tej grupie pacjentów.

Słowa kluczowe: ciśnienie tętnicze wysokie prawidłowe, czynniki ryzyka sercowo-naczyniowego, zmiany narządowe, czułość odruchu z baroreceptorów, reokardiografia impedancyjna, wskaźnik intima-media, kwas moczowy

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