CENTRAL AND BRACHIAL PULSE PRESSURE PREDICTS CARDIOVASCULAR AND RENAL EVENTS IN TREATED HYPERTENSIVE PATIENTS

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Objective: Central blood pressure is a stronger predictor of cardiovascular prognosis rather than brachial blood pressure. Reflection wave reaches abdominal aorta sooner than ascending aorta. Thus, the contribution of central pulse pressure (cPP) to renal events may differ from that to cardiovascular events.

Design and method: The subanalysis of ABC-J II study (Hypertens Res. 2018;41:947–956) was performed. Subjects were 3434 treatedhypertensive patients with mean follow-up of 4.7 years. The contribution of brachial pulse pressure (bPP) and cPP to cardiovascular and renal events were analyzed.

Results: Cox proportional-hazard analysis revealed that sex (p < 0.001), height (p < 0.05), history of cardiovascular diseases (p < 0.001), numbers of antihypertensive drugs (p < 0.05) and cPP (p < 0.05) contributed to cardiovascular events. However, baseline serum creatinine (p < 0.001) and bPP (p < 0.05) were selected as significant predictors of renal events.

Summary of Cox proportional-hazards analyses

Analysis on cardiovascular events

| Variables | β | Wald | Р |
|------------------------------|-------|-------|--------|
| Sex | 1.28 | 12.4 | <0.001 |
| Height | -0.04 | 4.2 | 0.04 |
| History of MI/stroke | 1.53 | 29.2 | <0.001 |
| cPP | 0.02 | 4.2 | 0.04 |
| numbers of antihypertensives | 0.22 | 4.5 | 0.04 |
| Analysis on renal events | | | |
| Variables | β | Wald | Р |
| Baseline serum creatinine | 2.62 | 132.9 | <0.001 |
| bPP | 0.02 | 4.4 | 0.04 |

MI: myocardial infarction, cPP: central pulse pressure, bPP: brachial pulse

pressure.

Conclusions: The present findings indicate that cPP and bPP independently contribute to cardiovascular and renal events, and suggest that the diverse results may be attributable to the anatomy that abdominal aorta feeds renal arteries, but thoracic aorta does cerebral and coronary arteries.

DIRECT COMPARISON OF INTERSECTING-TANGENTS VERSUS SECOND DERIVATIVE FOR DETERMINATION OF PULSE TRANSIT TIME AND PULSE WAVE VELOCITY

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Objective: Arterial stiffness is a non-traditional risk factor for cardiovascular disease. Aortic stiffness is assessed by determination of pulse wave velocity

using pulse transit time and the distance between carotid and femoral arteries. Transit time is obtained by using the foot-to-foot method to define the transit time through intersecting tangents algorithm or the point of maximal upstroke during systole (2nd derivative). Millasseau et al have proposed a formula for converting transit time between methods using SphygmoCor (intersecting tangents) and the Complior Analyse (2nd derivative). Based on a mathematical modeling of the proposed formula, there is discrepancy between values of pulse wave velocities, especially in subjects with higher aortic stiffness. The objective of this study is to directly compare the two methods using the same pressure waveforms obtained by the newer generation of Complior Analyse and using Millasseau's formula.

Design and method: In a cross-sectional study of heterogeneous subjects, aortic stiffness was assessed by the Complior Analyse device which uses 2nd derivative. The pulse waveforms were extracted and used for the analysis by custom MAT-LAB algorithm for intersecting tangents, and the results were compared to the formula proposed by Millasseau.

Results: The preliminary results of the first 24 patients (men: 71%; mean age: 61 \pm 18 years) show that Millasseau's formula underestimates the transit times values by about 19% in comparison with the transit times obtained by the intersecting tangents method using MATLAB software (49,8 \pm 18,8 ms vs 61,6 \pm 18,6 ms; P < 0,001). This results in an overestimation of the pulse wave velocities values by about 30% (13,8 \pm 3,8 m/s vs 10,6 \pm 2,7 m/s; P < 0,001).

Conclusions: Our preliminary results allow us to conclude that the values of pulse wave velocities obtained with Millasseau's formula are overestimated values when comparing with values obtained by using the intersecting tangents method. Increasing the number of subjects will allow us to examine the possibility of a more reliable formula for converting transit times from one method to the other.

ALDOSTERONE SYNTHASE CYP11B2 (-344C/T) GENE POLYMORPHISM INFLUENCES RISK OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH ARTERIAL HYPERTENSION

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Objective: Renin-angiotensin aldosterone system plays a major role in blood pressure regulation. Aldosterone, synthesized in the adrenal cortex by aldosterone synthase is encoded by the cytochrome 11B2 aldosterone synthase gene (CYP11B2). The aim of the study was to analyze the association of aldosterone synthase gene (CYP11B2) biallelic polymorphism (-344C/T) with Chronic Kidney Disease (CKD) in patients with essential arterial hypertension (EAH) in West-Ukrainian population.

Design and method: One-hundred subjects with EAH and target-organ damaging (2nd stage), moderate, high or very high cardiovascular risk were involved in the case-control study. Among them 79.0% (79) females and 21.0% (21) males, mean age 59.87 ± 8.02 yo; disease duration from 6 to 25 years. CKD was determined by the National Kidney Foundation recommendations (Kidney Disease: Improving Global Outcomes (KDIGO), 2012) after glomerular filtration rate (GFR) decline < 60 ml/min/1.73sq.m for over 3 months (by Cockroft-Gault formula and CKD-EPI for Cystatin-C and Creatinine serum levels depending on gender). CKD was diagnosed in 29 persons. All enrolled / screened patients signed the Informed Consent to participate in the research. Control group included 48 practically healthy persons of relevant age. Gene polymorphism of aldosterone synthase gene CYP11B2 (-344C/T) was examined by polymerase chain reaction (PCR).

Results: The probability of EAH in observed population increased 1.49 times in T-allele carriers of CYP11B2 gene, but only in females [OR = 1.90; 95%CI:1.02–3.54; p = 0.029], with contrary decreasing in C-allele women (p = 0.041). No relevant dependences were observed in hypertensive males. Also T-allele increased probability of CKD (GFR< 60 ml/min/1,73m2) in hypertensive population 1.48 times [OR = 1.86; 95%CI:1.01–3.58; p = 0.049], especially in T-allele females 1.53 times [OR = 6.51; 95%CI:1.39–30.60; p = 0.007] with low CKD risk in T-allele males [OR = 0.15; 95%CI:0.3–0.72; p = 0.009], respectively. Some predictors like DM2, the 2nd and 3rd grades of Obesity, and the 3rd grade level of Blood Pressure elevation escalated the risk of CKD 2.4, 2.08–2.32 and 2.91 times, accordingly (p< 0.05).

Conclusions: Aldosterone synthase gene CYP11B2 (-344C/T) is associated with EAH. T-allele increased risk of CKD in hypertensive population, especially in females.