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Familial history of hypertension as a predictor of increased arterial stiffness in normotensive offspring

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KEYWORDS

Arterial stiffness;
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Abstract *Background:* Increased arterial stiffness can be used as a prognostic marker of arterial hypertension. The relationship between arterial stiffness and arterial hypertension seems to be reciprocal.

Objective: Evaluation of changes of the arterial elastic prosperities in normotensive subjects, with and without parental history of hypertension.

Subjects and Methods: One hundred and ten normotensive individuals, aged 20–30 years, were divided into two groups: group-A ($n = 57$) and group-B ($n = 53$) subjects with positive and negative parental history of hypertension, respectively. Systolic, diastolic and pulse pressures were measured using mercury sphygmomanometer. The elastic properties of the ascending aorta and the common carotid arteries were assessed using M-mode echo and B-mode imaging, respectively. Stiffness index of the digital volume pulse (SIDVP) was measured in the right index finger using photoplethysmography.

Results: Group A subjects showed higher aortic stiffness index ($p = 0.002$), carotid stiffness index ($p = 0.001$), carotid pulse wave velocity ($p \leqslant 0.001$) and stiffness index of digital volume pulse ($p = 0.001$). Group A subjects showed lower aortic distensibility ($p = 0.001$), aortic strain ($p = 0.004$), changes in aortic diameter ($p = 0.022$), carotid distension ($p = 0.026$), carotid distensibility coefficient ($p \leqslant 0.001$) and carotid compliance coefficient ($p = 0.002$).

Conclusion: The aortic and carotid stiffness parameters and SIDVP were higher in normotensive offspring of hypertensive parents. This finding could direct the attention towards the increased cardiovascular risk in this group and thus prompt earlier and tighter prevention of cardiovascular risk factors.

1. Introduction

An abnormal large arterial function plays an important role in the pathogenesis of cardiovascular disease.[1] The aortic elastic

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properties are important determinants of blood pressure and left ventricular function.

Diastolic blood pressure (DBP) is determined by peripheral arterial resistance, while Systolic blood pressure (SBP) and Pulse pressure (PP) are influenced more by the stiffness of large arteries, as well as, peripheral pulse wave reflection and the pattern of left ventricular ejection.[2,3]

Increased stiffness of the aorta is an important determinant of future increases in Blood pressure (BP) and progression of hypertension.[4–7] The association between vascular stiffness and blood pressure is particularly interesting because the functional relationship is likely bidirectional.[8] Elevated blood pressure may cause vascular damage and accelerated conduit artery stiffness.[8] Conversely, aortic stiffening increases pressure pulsatility and therefore affects systolic blood pressure: not only the aorta, as it also found that higher carotid artery stiffness was associated with incident hypertension.[9]

Offspring of hypertensive have higher blood pressures, weight, height, and body surface area compared to the offspring of normotensive parents.[10]

In addition, family history of hypertension considered a predictor of hypertension that may be a stronger risk factor for hypertension than other factors.[11,12]

Measuring arterial stiffness helps to identify patients at high risk for hypertension and serve to re-emphasize the importance of preserve arterial function in the primary prevention of hypertension.[13] This study hypothesized that offspring of parents with hypertension would have greater arterial stiffness compared with offspring of parents without hypertension.

2. Methodology

2.1. Study population

One hundred and ten drug-naïve normotensive individuals, including volunteering doctors and nurses, aged 20–30 years, were recruited by a non-randomized way. They were divided into 2 groups, group A, 57 subjects and group B, 53 subjects with positive and negative parental history of hypertension, respectively. Exclusion criteria were diabetes mellitus, chronic renal failure (serum creatinine >1.4 mg/L) and hypertension. Subjects were asked to refrain from caffeine, smoking and strenuous exercise for 12 h before the study. After a light breakfast following overnight fasting, the subject is asked to rest in bed in the supine position for 15 min. Each subject was evaluated clinically and his/her height and weight were measured and BMI was calculated.

2.2. Blood pressure measurements

Blood pressure was measured in the right arm using mercury sphygmomanometer, taking into consideration the appropriate cuff size. Korotkoff phases I and V were used to define the systolic and diastolic blood pressures, respectively. Measurements were taken 3 times per visit, 2 min apart, and were averaged.

2.3. Echocardiographic measurements

Transthoracic echocardiography was performed to confirm absence of organic heart diseases and to evaluate the aortic elastic properties, based on the standards of the American Society of Echocardiography[14] by an experienced echocardiographer, using a commercially available machine (Esaote MyLab 60) with a 2.5 MHz phased-array transducer.

2.4. Calculation of the elastic parameters of the aorta (Fig. 1)

The diameter of the ascending aorta was measured in the parasternal long axis view by 2D-guided M-mode tracing. Measurements were taken 3 cm distal to the aortic valve. The systolic diameter was measured at the maximal anterior excursion of the aortic valve, whereas the diastolic diameter was measured at the peak of the QRS complex on the simultaneously recorded electrocardiogram. Three consecutive measurements were taken and averaged. The aortic stiffness index, aortic distensibility and aortic strain were calculated by the following formula:

$$\text{Aortic } \beta \text{ stiffness index} = \ln(\text{SBP}/\text{DBP})/[(\text{SD}-\text{DD})/\text{DD}]^{15}$$

where In, natural logarithm; SBP and DBP, systolic and diastolic blood pressures, respectively; SD = systolic diameter, and DD = diastolic diameter.

$$\text{Aortic strain } (\%) = (\text{SD}-\text{DD})/\text{DD}^{13-15}$$

$$\text{Aortic distensibility } (\text{cm}^2/\text{dyne}) = 2\times(\text{SD}-\text{DD})/[(\text{SBP}-\text{DBP})\times\text{DD}]^{15}$$

The value obtained from the equation of aortic distensibility should be multiplied to 1333 to convert mmHg to cm^2/dyne.

2.5. Ultrasound imaging of Common Carotid Artery (CCA) (Figs. 2 and 3)

Measurements of carotid distension were performed by tracking arterial wall movement with adequate spatial and temporal resolution.[16] Longitudinal images of the both CCA, were obtained with linear 10 MHz probe. A rectangular region of interest (ROI) was placed at CCA segment starting approximately 1–2 cm before the carotid bifurcation. Arterial distension was measured in 32 scanning lines positioned within the ROI (sampling rate of 550 Hz on 32 lines). During the acquisition, orange lines indicated the automatic positioning of tracking cursors on the media-adventitia interface, whereas continuous green lines displayed dynamically the amplified vessel wall movement. From the real-time distension curves, displayed in blue at the bottom of the screen, maximum and minimum carotid diameters were measured, carotid distension and minimum diameter in the last six cardiac cycles were presented beat-to-beat on the screen and the mean value and standard deviation were continuously calculated.[16] The local carotid pressure is automatically estimated by converting the distension curve to pressure curve by a linear conversion factor.[16] The peripheral BP was needed for calibration of the distension curves.[16]

The following parameters were automatically calculated: carotid distension, compliance coefficient, distensibility coefficient, pulse wave velocity (PWV), carotid beta-stiffness index and local blood pressure (systolic and diastolic).

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