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Original Article

Prognostic Value of Aortic Pulse Wave Velocity as Index of Arterial Stiffness in the General Population

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ABSTRACT

Background: *During a person's lifetime, as part of the aging process or as a consequence of hypertension, atherosclerosis, or other pathological processes, the aorta stiffens. Accordingly, the forward pulse wave travels faster, and the arterial waves reflected from the periphery reach the heart early during systole, which leads to higher systolic but lower diastolic blood pressure, an augmentation of the cardiac workload, and a decrease of the coronary perfusion pressure. The aortic pulse wave velocity (APWV) reflects central arterial stiffness. Some studies addressed the prognostic significance of APWV above and beyond other cardiovascular risk factors. Furthermore, pulse pressure, an indirect measure of increased arterial stiffness, predicts a poor prognosis in treated and untreated hypertensive subjects.*

Methods: *The study was conducted on a sex- and age-stratified random sample of 167 rural and suburban population of Haryana state aged 40 to 70 years.* ***Results:*** *Cox regression analysis was used to investigate the prognostic value of APWV, pulse pressure (PP) and other covariates. We adjusted for sex, age, body mass index. MAP measured in the office (conventional PP) and APWV by Periscope TM. With these adjustments, APWV maintained its prognostic significance in relation to each end point (P<0.05), whereas office PP lost their predictive value (P>0.19). In sensitivity analyses, APWV still predicted all cardiovascular events after standardization to a heart rate of 60 beats per minute, after adjustment for MAP.* ***Conclusions:*** *In a general population of Haryana, APWV predicted a composite of cardiovascular outcomes above and beyond traditional cardiovascular risk factors.*

Keywords: *Arterial stiffness, cardiovascular diseases, Pulse pressure, Risk factors*

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INTRODUCTION

During a person's lifetime, as part of the aging process or as a consequence of hypertension, atherosclerosis, or other pathological processes, the aorta stiffens. Accordingly, the forward pulse wave travels faster, and the arterial waves reflected from the periphery reach the heart early during systole, which leads to higher systolic but lower diastolic blood pressure, an augmentation of the cardiac workload, and a decrease of the coronary perfusion pressure. The aortic pulse wave velocity (APWV) reflects central arterial stiffness. Some studies addressed the prognostic significance of APWV above and beyond other cardiovascular risk factors. Furthermore, pulse pressure, an indirect measure of increased arterial stiffness, predicts a poor prognosis in treated and untreated hypertensive subjects.

METHODS

Study Population and data collection

Prior informed written consent was obtained from the participants provided. The study was conducted on suburban and rural areas of Haryana with the goal to recruit an equal

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number of women and men aged 40, 50, 60 &70 years. At baseline, the participation rate was 82.6%. PeriScopeTM was applied for assessment of Aortic Pulse Wave Velocity. Body mass index was weight in kilograms divided by height in meters squared. After the subjects had rested for 5 minutes in the supine position, 2 consecutive blood pressure readings were obtained with a random zero mercury sphygmomanometer fitted with an appropriate cuff size. The 2 readings were averaged for analysis. Hypertension was defined as an office blood pressure of e"140 mm Hg systolic or 90 mm Hg diastolic. Heart rate was counted at the radial artery over 15 seconds. Immediately thereafter, the same trained individuals used PeriScopeTM to record in all subjects the arterial wave simultaneously at the left common carotid and femoral arteries. APWV was the travel distance between the 2 transducers, measured on the body surface, divided by the transit time. For analysis, we averaged from 2 to 15 heart cycles. Pulse pressure (the difference between systolic and diastolic blood pressure) and mean arterial pressure (diastolic blood pressure plus one third of pulse pressure) were computed from the office. Venous blood samples collected after overnight fasting were analyzed by standard automated methods for lipids and blood glucose. According to published criteria, diabetes mellitus was defined as a fasting blood glucose level of e"7.0 mmol/L or as the use of oral antidiabetic drugs or insulin.

Statistical Analysis

For statistical analysis, we used SPSS software, version 19. To compare means, we used the standard normal Z test for large samples or ANOVA with Tukey test for multiple comparisons. For proportions, we used the 2 statistics with Bonferroni correction of the probability values, if appropriate. In the analysis of outcome, for participants who experienced multiple events, we considered only the first event. We implemented Cox proportional hazard regression to calculate relative hazard ratios in relation to APWV and pulse pressure. First, in exploratory analyses, we calculated relative hazard ratios for the composite cardiovascular end point by quintiles of the distribution of APWV and the pulse pressures, unadjusted or with adjustment for sex and age. We used the deviation from mean coding to compute hazard ratios in quintiles relative to the overall risk in the study population. This approach avoids any assumption about the shape of the association between outcome and APWV or pulse pressure. The baseline measurements considered as predictors were sex, age, body mass index, waist-to-hip ratio, mean arterial pressure, use of antihypertensive drugs, current smoking, alcohol intake, physical activity, ratio of total to HDL serum cholesterol, and diabetes mellitus. To test for heterogeneity between women and men in the associations between outcome and APWV, we forced the appropriate interaction term into the regression models. In a sensitivity analysis, we standardized each participants APWV to a heart rate of 60 beats per minute by means of regression analysis in women and men, separately. Statistical significance was a probability value of d"0.05 on 2-sided tests.

RESULTS

BP indicates blood pressure. Values are mean ± SD or number of subjects (%). Gender differences were significant (*P*<0.05) except for age class, office and total cholesterol (0.07<*P*<0.48).

Table 1. Baseline Characteristics of Participants

Variables	Women (n=70)	Men (n=97)
Anthropometrics		
Age class, 40/50/60/70 y, %	17/19/16/18	24/28/25/20
Body mass index, kg/m^2	25.3±4.4	26.4±3.6
Hemodynamic measurements		
Office BP systolic, mm Hg	126.9±19.1	132.5±17.2
Office BP diastolic, mm Hg	79.2±10.1	82.9±10.1
Office heart rate, bpm	64.0±8.8	61.0±9.7
APWV, m/s	10.8±3.2	11.8±3.6
Office pulse pressure, mm Hg	46.9±13.2	47.9±13.2
Biochemical measurements		
Fasting glucose, mmol/L	4.7±0.9	5.0±1.1
Total cholesterol, mmol/L	6.23±1.12	6.1±1.10
HDL cholesterol, mmol/L	1.60±0.44	1.29±0.33

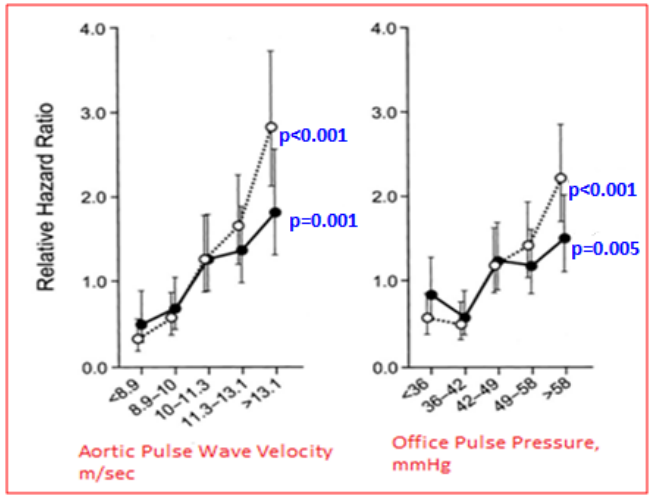

Figure I. Relative hazard ratios for the composite cardiovascular end point by quintiles of the distribution of APWV and office pulse pressures unadjusted (open symbols) or with adjustment for sex and age (closed symbols). The hazard ratios express the risk in each quintile vs the average risk in the whole population. Vertical lines denote 95% CIs. *P* values are for trend. The explanatory analysis unadjusted or adjusted for sex and age revealed strong associations of the risk of the composite cardiovascular end point with APWV and office pulse pressures.

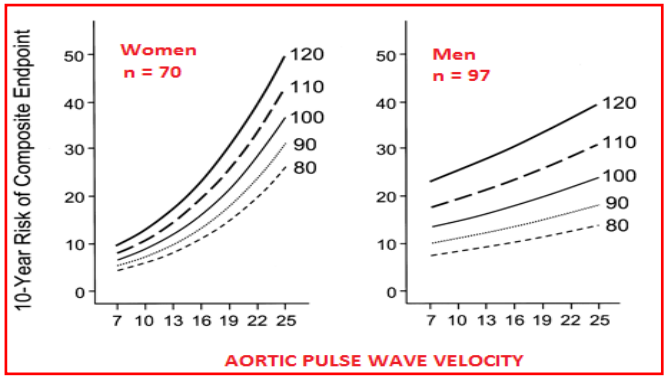

Figure II. Absolute risk associated with APWV in women and men at different levels of mean arterial pressure in the office controlling for age, body mass index, current smoking, and alcohol intake.

In Cox models unadjusted or only adjusted for sex and age, APWV and the office pulse pressures consistently predicted each of the 3 outcomes under study. In the fully adjusted models, APWV maintained its prognostic significance in relation to each end point, whereas the office pulse pressures no longer predicted outcome. Figure 2 shows the absolute risk in women and men associated with APWV at different levels of mean arterial pressure in the office, while controlling for age, body mass index, current smoking, and alcohol intake.

DISCUSSION

The key finding of our study was that in middle-aged and elderly individuals andomly recruited from the population of rural Haryana, APWV measured over in the office was a significant predictor of cardiovascular complications, above and beyond mean arterial pressure and other risk factors, including sex, age, body mass index, current smoking, and alcohol intake. With similar adjustments applied, the office pulse pressures lost their prognostic value with the exception of office pulse pressure in relation to coronary heart disease. For each 1-SD increment in APWV, the risk of an event increased by 16% to 20%. In sensitivity analyses, APWV still predicted all cardiovascular events after standardization to a heart rate of 60 beats per minute, after adjustment for mean arterial pressure, and/or after additional adjustment for the ratio of total to HDL serum cholesterol and diabetes mellitus at baseline.

The present study must be interpreted within the context of its potential limitations and the choices that we made in our epidemiological and statistical approach. At baseline, we did not determine the reproducibility of the APWV measurements. However, only 1 trained observer acquired and read all APWV recordings. The exclusion from analysis of participants with a previous history of cardiovascular disease lends support to the concept that stiffening of the central arteries is already prognostically relevant in relatively healthy subjects. Fourth, whether or not APWV should be standardized for heart rate remains a matter of debate. In the present study, heart rate did not behave as a significant forerunner of a worse cardiovascular outcome. Even when adjusted for mean arterial pressure determined blood pressure recordings, APWV kept its prognostic value in relation to the composite of all cardiovascular events. The present findings highlight the need to develop more sensitive techniques to measure the stiffness of various compartments of the arterial tree, which can be readily applied in routine clinical practice for risk stratification. Moreover, further molecular, clinical, and epidemiological research should clarify the genetic mechanisms, environmental factors, and their interaction that lead to premature stiffening of the arterial wall.

CLINICAL PERSPECTIVE: Aortic pulse wave velocity is easily acquired at the bedside and reflects central arterial stiffness.

Accordingly, we investigated this as a predictor of outcome in study population comprised of 167, aged 40 to 70 years, randomly recruited from the population of rural Haryana, over a period of 6-10 months. We adjusted for sex, age, body mass index, mean arterial pressure measured in the office by PeriscopeTM, smoking, and alcohol intake. With these adjustments, aortic pulse wave velocity maintained its prognostic significance in relation to each end point, whereas office pulse pressure lost their predictive value with the exception of office pulse pressure in relation to coronary heart disease.

CONCLUSION

Aortic pulse wave velocity predicted cardiovascular outcomes over and beyond blood pressure and traditional risk factors. These findings highlight the potential of indexes of arterial stiffness in risk stratification and the need to introduce such measurements into clinical practice.

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