

[PP.35.198] CENTRAL BLOOD PRESSURE IN TYPE 2 DIABETIC AND PREDIABETIC PATIENTS: CORRELATION WITH PERIPHERAL BLOOD PRESSURE

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Impaired glucose homeostasis is associated with arterial stiffness, but differences among relationship with central or peripheral blood pressure are unknown.

Objective: to assess the relationship between estimates of both peripheral BP (office BP, 24h, daytime, and nighttime BP) and central (c) BP in patients with impaired glucose metabolism.

Design and Methods: Cross-sectional study in subjects with type 2 DM or pre-diabetes ([≥]3 of the following: triglycerides > 150 mg/dl and/or drug treatment; HDLc < 40 mg/dl in men (M) or < 50 mg/dl in female (F) and/or drug treatment; waist circumference > 88 cm (F) or > 102 cm (M); plasma fasting glucose > 100 mg/dl and/or drug therapy; BP [≥]130/85 mmHg and/or drug treatment). Clinical data, laboratory analyses and EKG were recorded. Office BP, 24h-ABPM and central BP by radial artery applanation tonometry (Sphygmocor®) were measured.

Results: There were 506 patients (diabetics: 75%; pre-diabetics: 25%); age: 64 ± 10y; 62% M/38% F; BMI: 30.9 ± 4.3 Kg/m²; waist circumference: 106 ± 10 cm (M), 103 ± 11 cm (F). Prevalence of other risk factors was: hypertension: 91%; dyslipidemia: 72%; current smokers: 14%. Office BP (mmHg): SBP 142.3 ± 20.9; DBP 80.5 ± 12.3. ABPM (mmHg): 24h-SBP: 127.4 ± 14.5; 24h-DBP: 72.9 ± 9.1; day-SBP: 130.4 ± 15.0; day-DBP: 75.7 ± 9.6; night-SBP: 120.4 ± 16.1; night-DBP: 66.6 ± 9.3. Central BP (mmHg): cSBP: 127.4 ± 19.5; cDBP: 80.0 ± 12.6; cPP: 47.6 ± 15.2. Augmentation pressure: 15.2 ± 8.5. Augmentation index: 30.3% ± 11. Pulse wave velocity: 10.3 ± 3.3 m/s. After age- and sex- adjustment, the following correlations (Pearson's "r"; p < 0.001 for all correlations) were found: cSBP correlated with office-SBP (r = 0.818), 24h-SBP (r = 0.581), day-SBP (r = 0.594) and night-SBP (r = 0.506), whereas cDBP correlated with office-DBP (r = 0.819), 24h-DBP (r = 0.693), day-DBP (r = 0.703) and night-DBP (r = 0.535). ROC curves were performed to determine the thresholds of central BP better correlated with normal ranges of different peripheral BP measurements. The cutoff for central SBP better associated with office hypertension was 121 mmHg, with very high sensitivity [91.1% (95%CI: 88.1 – 94.2)] and high specificity (75%), whereas obtained cutoffs for ABPM estimates showed less sensitivity and specificity.

Conclusions: In type 2 DM and prediabetic patients, the best correlations of non-invasively determined central BP were with office BP, as compared to other peripheral BP estimates. Moreover, central SBP of 121 mmHg is the threshold that better discriminates the occurrence of hypertension as diagnosed by office BP assessment.

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