

Arterial health abnormalities in women with preeclampsia: contributing role of central obesity

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Objective

Women with previous pre-eclampsia (PE) are at higher risk of cardiovascular disease (CVD). This risk can be explained in part by the observation of worse arterial health in these women. Central obesity (CO) is an established risk factor for PE, while also being known to adversely affect cardiovascular risk. Whether CO contributes to further worsening of arterial hemodynamics in PE women remains unclear. We evaluated the effect of CO when combined with previous PE on women's arterial hemodynamics.

Methods

We studied 40 women with previous PE in the last 6 years and 40 age-matched women with previous normotensive pregnancy (controls) in the same timeframe. We estimated aortic stiffness, central blood pressure (BP), and measures of steady and pulsatile arterial load with validated techniques combining applanation tonometry and echocardiography. CO was defined as a waist-to-hip ratio (WHR) ≥ 0.85 according to WHO criteria for women. Differences in arterial hemodynamics across the 3 groups [PE with CO (PE-CO), PE without CO (PE-noCO), controls] were assessed with One-Way ANOVA, and in multivariable linear regression models adjusted for age, hypertension, diabetes, serum creatinine, and parity.

Results

Twenty-six (65%) of the PE participants had CO, compared to 18 (45%) of controls ($p=0.07$). Mean age in PE-CO, PE-noCO and controls was respectively 36.6 ± 4.6 , 34.0 ± 4.1 and 35.8 ± 3.1 years ($p=0.46$). Mean WHR was respectively 0.94 ± 0.05 , 0.80 ± 0.04 and 0.83 ± 0.07 ($p < 0.01$). In unadjusted analyses, PE-CO women had higher central systolic BP as compared to PE-noCO and controls respectively (117 ± 21 vs 100 ± 12 and 95 ± 9 mmHg, $p < 0.01$), as well as higher aortic stiffness expressed by carotid-femoral pulse wave velocity (6.58 ± 0.97 vs 5.95 ± 1.01 and 5.68 ± 0.75 m/s, $p < 0.01$). They also had higher forward pressure wave amplitude, with lower proximal aortic compliance and total arterial compliance. In multivariable linear regression, PE-CO women had statistically significantly higher central systolic BP, aortic stiffness and lower proximal aortic compliance as compared to controls, while hemodynamics of PE-noCO did not significantly differ from controls.

Conclusion

We demonstrate for the first time the contribution of central obesity in adversely affecting arterial health and central hemodynamics in women with previous PE. Understanding that worse arterial stiffness and hemodynamics are linked to future CVD, our results suggest that women with preeclampsia and central obesity may represent a higher risk subgroup who should be candidates for early, targeted efforts at primary prevention of CVD.