Letter to the Editor

Essential Hypertension, Hypertension in Pregnancy, and Subsequent Blood Pressure: Another Old Dilemma Revisited

To the Editor:

We read with interest the recent article by Lazdam et al concerning the potential mechanisms linking prematurity, maternal blood pressure, and the subsequent blood pressure of offspring. However, we believe that two issues arising from this otherwise carefully conducted observational study deserve further attention and debate.

The first is the interpretation of the aortic stiffness data. The authors used carotid-femoral pulse-wave velocity as a well-accepted, gold standard, noninvasive methodology. However, with the exception of β-index, all measures of stiffness depend on the distending pressure at which the measurement is made. Therefore, differences in mean arterial pressure will confound stiffness measurements and should be corrected for. If carotid-femoral pulse-wave velocity remains significantly higher after adjustment, then this suggests that there is a primary elevation of stiffness, which may explain, in part, an elevation of blood pressure, particularly systolic/pulse pressure.

In the study by Lazdam et al, carotid-femoral pulse-wave velocity was ~0.14 m/sec lower in the preterm hypertensive pregnancy offspring and ~0.36 m/sec higher in the normotensive preterm offspring. However, mean arterial pressure was ~6 mm Hg higher in each of the preterm groups compared with controls. Unfortunately, like many other authors, Lazdam does not adjust for this. However, based on 2500 individuals in the ENIGMA study, we have estimated that carotid-femoral pulse-wave velocity increases by 0.5 m/sec for each 10-mm Hg elevation in mean arterial pressure. This suggests that isobaric carotid-femoral pulse-wave velocity was probably not different between the normotensive preterm offspring and controls, but it was actually lower in the hypertensive preterm offspring. Clearly, had this been the case, then structural or functional differences in large vessels such as the aorta could not be responsible for the observed differences in adult blood pressure between the groups, contrary to the authors’ interpretation.

The second issue is the blood pressure of the mothers before they became pregnant. Preconception blood pressure and hypertension per se increase the likelihood of hypertension during pregnancy and premature delivery. Because hypertension is a hereditary factor, the offspring of hypertensive patients have a higher blood pressure than those with normotensive parents. Therefore, the higher blood pressure in the offspring of women who had hypertension in pregnancy may simply relate to the fact that these women had higher blood pressures before conception or had hypertension. Unfortunately, Lazdam et al do not provide these data, and such factors are often ignored in other cohort studies. We welcome further discussion of this issue, but new prospective studies may be required to obtain such data.

In summary, Lazdam et al demonstrate that individuals born prematurely have higher blood pressure as young adults, confirming the observations of several other groups. However, we do not believe that this is attributable to alterations in the structure or function of the aorta. More data are required to determine whether preconception factors such as maternal blood pressure may confound such observations.

Disclosures

None.


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