Hypertension and Cerebral Vascular Reactivity in Childhood: Challenge and Opportunity

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The study of childhood origins of adult cardiovascular disease has grown dramatically in the past decade. Childhood-onset obesity, hypertension, and hyperlipidemia confer increased risk of cardiovascular morbidity and mortality in adulthood. The mechanisms for these relationships are incompletely understood. Even less is known about the extent to which end-organ injury referable to these risk factors actually begins during childhood. The study by Wong et al provides new insights concerning the early-life effects of hypertension on cerebral vascular physiology. The rapid increase in prevalence of cardiovascular risk factors such as obesity and hypertension among US children underscores the public health implications of their observations.

Wong et al evaluated cerebrovascular reactivity to hypercapnia among children with essential hypertension compared with normotensive control subjects. They tested the hypothesis that hypertension is associated with impaired hypercapnic reactivity measured by increased cerebral blood flow velocity on transcranial Doppler. Hypertension was carefully measured and quantified using well-defined and standardized 24-hour ambulatory blood pressure monitoring methods. Cerebrovascular reactivity measures were likewise well-defined and carried out in a standardized manner. These transcranial Doppler data on cerebral vascular reactivity are interesting and novel in their own right, because there are few published studies reporting transcranial Doppler data, much less hypercapnic reactivity, in awake, neurologically well children. They found hypercapnic reactivity is decreased in hypertensive children compared with normotensive control subjects with a negative linear correlation between reactivity and both mean 24-hour diastolic blood pressure and diastolic load. The relatively small sample size precluded additional analyses of potential modifying factors such as sex, race, age, and the effect of treatment with antihypertensive drugs.

Having confirmed that hypertension is associated with decreased cerebrovascular reactivity, as is seen in adults, a number of compelling questions beg to be further evaluated. The authors have raised the question, for example, as to whether impaired cerebrovascular reactivity with hypertension is associated with impaired neurocognitive function in children as is seen in adults. A few tantalizing clues already exist in a small number of published studies that support an association between hypertension and diminished neurocognitive function and academic attainment in children.

Much more study is needed, however, to reproduce these observations in larger populations to determine the longitudinal trends through different stages of childhood and beyond to adult life and, importantly, to establish what the causal relationships and mechanisms may be. For example, there are no published studies evaluating neuroimaging correlates of neurocognitive impairments in hypertensive children. The existence of potentially quantifiable and anatomically specific structural neuroimaging markers of cerebral injury from childhood hypertension may provide additional insights concerning mechanism as well as objective end points for interventions. Juonala et al reported age-dependent associations between cardiovascular risk factors and the findings of early atherosclerosis on vessel imaging in children and adolescents. It would be of great interest to evaluate the relative contribution of childhood hypertension compared with other cardiovascular risk factors such as obesity and hyperlipidemia on vessel wall markers of early atherosclerosis and to understand how this is related, if at all, to the development of disturbed cerebrovascular reactivity.

Ultimately, a clearer understanding of the mechanisms and epidemiology of childhood precursors of cerebrovascular disease will be essential for designing interventions and subjecting these to clinical trial. The magnitude of the public health implications of these problems can hardly be overstated. Studies such as that of Dr Wong and colleagues are groundbreaking by demonstrating that understanding and effectively preventing chronic adult-onset cardiovascular and cerebrovascular disease may best begin in childhood. Moreover, the dividends of treating childhood precursors of adult disease may become evident in childhood by halting or reversing clinically significant end-organ injury even early in life.

Disclosures

None.

References


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