Editorial

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Cardiovascular Disorders and Hypertension in Normotensive Children and Adolescents with a Positive Family History of Cardiovascular Diseases

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Editorial

The relationship between cardiovascular diseases including atherosclerosis, and family history of them, starts asymptomatically from the first years of life [1] and are predictors of cardiovascular risk in adulthood [2,3]. Among them, high blood pressure (HBP) is considered one of the major risk factors for cardiovascular disease. Normotensive children of hypertensive parents have shown early cardiovascular changes that expose them to greater risk of developing hypertension in adulthood [2,3].

Calentano et al. in a study in normotensive children of hypertensive parents showed that they exhibit an increase in left ventricular mass [4]. In turn, Meaney et al., in a comparative study with 100 young people from 10 to 21 years of age, with and without hypertensive parents, in order to determine if they had differences in arterial stiffness, they observed an early reduction in arterial elasticity, with increased stiffness and lower carotid diameter in the children of hypertensive parents regarding to the children of normotensive parents, suggesting that early blood pressure screening can help recognize individuals at risk [5].

Longitudinal studies have shown that normotensive adolescents of hypertensive parents followed for more than ten years, had persistently elevated the blood pressure (BP) values and showed an early metabolic syndrome disorders such as higher plasma insulin levels, increased triglycerides and values of Systolic BP (SBP), Diastolic BP (DBP) and heart rate [6-8].

Interestingly, adolescents with a history of a family history of early myocardial infarction and diabetes have shown a decrease brachial artery flow-mediated dilation and increased arterial stiffness and thickness of the carotid intima-media, as observed Gaeta et al., in 40 healthy young people studied with high-resolution B-mode ultrasound the vasodilatadory brachial-artery response and intima-media thickness [9].

In a study of our group performed in 87 adolescents we showed that normotensive adolescents from hypertensive parents have systolic BP and body mass index (BMI), significantly higher than their counterparts with normotensive parents [10] which agrees with previous work which showed that children of hypertensive parents have BP levels higher than those who have their normotensive parents [11] and the BP in childhood is a predictor of the development of hypertension in adulthood [12].

Given that it has been demonstrated the inverse relationship between SBP in adolescence and brachial artery flow-mediated dilation in adulthood, [13] the observed increase in SBP in children of hypertensive parents might suggest early alterations in the structure and a function of the arterial wall demonstrated in these youth groups [14] as Juonala et al. showed for example in a work done in 4320 subjects aged 3 to 18 years, where the SBP obtained at these ages, independent of other risk factors, had a strong correlation with the endothelial dysfunction obtained 21 years later. The authors concluded that the value of the SBP recorded in adolescence, can influence the processes that regulate endothelial-dependent vasodilation flow [13]. The increase in leukocyte adherence and endothelial permeability, the proliferation of smooth muscle cells and increased expression of cytokines and intimal growth factors, are some of the potential mechanisms that explain the relationship between the SBP in adolescence and endothelial dysfunction years later [15].

Moreover, increased BMI in adolescents of hypertensive parents is an independent risk factor for developing cardiovascular disease later in life, including hypertension, especially in those with a family history of it [16,17].

The increased activity of the renin-angiotensin system and plasma aldosterone levels, which would produce an increase in renal sodium reabsorption, would be linked to the increase in BP levels in obese adolescents with hypertensive parents [18] In turn, the weight gain in children has been linked to decreased arterial elasticity in adulthood [19] which may be related to some degree of inflammation of the artery wall [20] and with elevated plasma levels of leptin [21].

Finally, both SBP and BMI obtained in adolescence have proven to be predictors of increased carotid intima-media thickness and the aforementioned decrease in arterial elasticity in middle age [22]. On the other hand, the existence of alterations in ventricular geometry in normotensive adolescents with a family history of hypertension, suggest an

early heart compromise that could precede in years the increase pressure values [23].

The increaseed pulse wave velocity (PWV) in adults is accepted as an indicator of arterial stiffness and a strong cardiovascular risk [24]. The same, but not in all [25], has been shown in works carried in children and adolescents. Schieken et al. measured the aortic stiffness in nearly 400 children, eleven years old, using the Doppler acceleration time, checking that it was better predictor than SBP and independent of other variables such as height, weight or left ventricular mass [26]. In turn, the Minnesota Children's Blood Pressure Study showed an inverse relationship between the BP and arterial elasticity [27].

Atherosclerosis is a progressive asymptomatic disease that begins early in life which linked to traditional cardiovascular risk factors in childhood and adolescence, could be a predictor of cardiovascular disease in adulthood, especially in those with a family history of it. Normotensive children of hypertensive parents show a series of structural and functional arterial alterations early in life, probably interrelated, and would expose them to greater cardiovascular risk, among other things, to develop high blood pressure later in life.

Further studies are necessary to clearly the benefit of early use of techniques such as intima-media ultrasound, brachial artery flow-mediated dilation and pulse wave velocity, to identify these alterations and individuals at risk and the possibility of some early intervention that can influence the natural history of these diseases.

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