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CORRESPONDENCE

Comments to: Relation between interleukin-13 and annexin-V levels and carotid intima-media thickness in nephrotic syndrome

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Elsehmawy et al. wrote, "in children with idiopathic nephrotic syndrome (INS), atherosclerotic changes may accelerate progression to chronic kidney disease and are mainly related to dyslipidemia" (1). The authors aimed with their study to "assess the relation between carotid intima-media thickness (CIMT) measurements, renal Doppler resistive index (RI) and serum levels of interleukin-13 (IL-13) and annexin-V (An-V) in children with INS…" (1). As surrogate marker for preclinical atherosclerosis the authors used the carotid intima-media thickness (CIMT), measured by ultrasonography (1). The study was based on 60 children with INS and 60 healthy controls matched for age and gender (1). The patients had significantly higher CIMT as compared to controls (0.49 ± 0.06 vs. 0.35 ± 0.03, p<0.001) (1). The authors concluded that "the present study suggests an association between early atherosclerosis expressed as elevated CIMT measurements in children with INS and elevated serum levels of An-V and IL-13… In children with INS, atherosclerotic changes may accelerate progression to chronic kidney disease and are mainly related to dyslipidemia" (1). Some comments to the CIMT findings/conclusions of this study are needed. Elsehmawy et al. wrote, "Measurement of CIMT was done using the ultrasound machine …with high-frequency linear probe (7.5 MHz) …The arterial wall of the common carotid artery was assessed bilaterally in a longitudinal view…. Measurements were taken from the far wall of each common carotid artery 1 cm proximal to bifurcation. The measurements were made three times on each side and the average of the measurements was taken. The mean of measurements of the left and right common carotid arteries was calculated" (1). The authors (1) measured CIMT only at one predetermined carotid artery (CA) segment, the far wall of the common carotid artery (CCA) for higher spatial resolution (2). The major disadvantage of a one-site CIMT measurement method is to miss potential atherosclerotic altered vessel segments, given that atherosclerosis is a systemic disease yet, importantly, presents asymmetrically (3). A composite CIMT measure including all CA sections, e.g. both walls (far/near) of the CCA, bifurcation and internal CA, provides a more precise estimate of the CIMT (4); the authors (1) missed to mention and to discuss in the analysis of their results this important methodological aspect and limitation in their CIMT evaluation. Furthermore, Elsehmawy et al. (1) did not specify if the CIMT measurement was synchronized with the cardiac cycle and made, as recommended, at the end-diastole (2). CIMT values are subject to obvious vessel diameter changes that occur during the cardiac phases, with thinner values in systole (lumen expansion) and greater values in diastole (lumen reduction) (5). The differences between the patients and controls can be due to measurements that occurred during different cardiac phases and therefore cannot be compared. Sub-millimetric differences in CIMT values will classify subjects into normal or abnormal CIMT groups (2). A meticulous and detailed CIMT measurement protocol is fundamental when CIMT is used as surrogate marker (2,4). In conclusion, CIMT values less than 0.6 mm are considered as normal (2). The mean CIMT of the group of patients (0.49 ± 0.06 mm) (1) falls within the normal CIMT range and is void of diagnostic and prognostic value. The CIMT values of this study (1), given the methodological flaws, should be considered with caution.

Disclosures

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