IJMS INTERNATIONAL JOURNAL of MEDICAL STUDENTS

Letter to the Editor

Carotid Intima-Media Thickness and Cardiovascular Risk Factors

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We read the review by Albers SS et al. emphasizing the relationship between carotid artery morphology and the evolution of atherosclerotic carotid artery disease (CAD).[1] The authors commented on the anatomic changes developing since birth and increasing with aging, which play a significant role in the development of atherosclerotic CAD. Mainly, due to the interactions with well-known risk factors of atherosclerotic plaques including hypertension, dyslipidemia, diabetes mellitus, and smoking; they also focused on intra-individual differences in the extension of carotid involvement.[1] Their findings are useful in daily practice by indicating anatomical and demographic factors related to the CAD pathogenesis, and showing that people may have unilateral carotid changes when both arteries underwent actions of the same systemic risk factors.[1] Increases in internal carotid kinking, carotid bulb diameter, internal and common carotid tortuosity, and bifurcation angle are more frequent with aging. Even in absence of disease, these alterations can contribute in disturbances of the intramural elastin, but the threshold at which the changes cause or protect from atherosclerotic CAD is not clear.[1]

In this setting, evaluations of carotid intima-media thickness (CIMT) in people with obesity, chronic kidney disease (CKD), and hyperparathyroidism seem be appropriate.[2-4] For example, the comparison of CIMT in two groups (G1 and G2) of hemodialysis people with levels of parathormone (PTH), 200 pg/ml (G1), mean age: 46 ± 5 years and above 500 pg/ml (G2), mean age: 48 ± 5 years. Patients were between the ages of 18 to 65 years and an ultrasonography study in the middle third of the right common carotids which showed no significant enhanced thickness and calcified plaques (G1: 42.86%, G2: 71.43%). CIMT was correlated with PTH levels. Worthy of note, the time on dialysis made no difference between CIMT of the groups.[2] CIMT was studied in three groups of patients aged between 30 and 60 years: G1 obese with CKD; G2 non-obese with CKD; and G3 obese without CKD.[3] All groups had measurements of CIMT higher than normal, mainly in patients of G3 (control group of study), showing that arterial changes are more related with obesity.[3]

There is a general accordance about the adoption of the earliest as possible procedures to reach and maintain a low cardiovascular risk during the entire life course. The role of vascular remodeling and risk factors for cardiovascular disorders since conception to 2 years of life also merit attention to allow for effective prevention.[4] Recent review with meta-analyses of 6,221 studies including 7,977 individuals assess for CIMT in ages from 0 to 18 years revealed risk factors in the first 1,000 days of life. Small size for gestational age was the most significant association with increased CIMT in childhood, which is related to disturbed blood flow and pressure and atherosclerosis. Although further research is needed to confirm the findings, preventive measures are justified, as eradicating gestational smoking and providing adequate maternal nutrition.[4]

Additional concern is on the relation of increased CIMT with stroke risk and vascular dementia development; with the growing population of older individuals, preventive procedures should initiate as early as possible.[5] Guidelines for control cardiovascular risks highlight the cost-effectiveness of primary and secondary prevention of stroke and high blood pressure, associated conditions with atherosclerosis that play a role in the evolution of vascular and Alzheimer's dementia. The weight control since the earliest infancy would reduce future cerebrovascular disorders and the development of dementia, increasing the quality of life of the elderly.[5]

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Editor: Mihnea-Alexandru Găman
Proofreader: Leah Komer
Layout Editor: Judie Joo
Submission: Nov 28, 2020
Revisions required: Dec 7, 2020
Received in revised form: Dec 8, 2020
Acceptance: Dec 8, 2020
Publication: Dec 14, 2020
Process: Peer-reviewed

Acknowledgments

None

Conflict of Interest Statement & Funding

The Authors have no funding, financial relationships or conflicts of interest to disclose.

Author Contributions

Conceptualization: VMS, LCM, Data Curation: VMS, LCM, Formal Analysis: VMS, LCM, Investigation: VMS, LCM, Methodology: VMS, Project Administration: VMS, Resources: VMS, LCM, Supervision: VMS, Validation: VMS, Visualization: VMS, LCM, Writing – Original Draft: VMS, LCM, Writing – Review & Editing: VMS, LCM.

Cite as:

Santos VM, Modesto LC. Carotid Intima-Media Thickness and Cardiovascular Risk Factors. Int J Med Students. 2020 Sep-Dec;8(3):322-3.

ISSN 2076-6327

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