Vitamin D and heart: A not so sunny pathway

In their article, Şeker et al. (1) explore the relationship between serum 25-hydroxyvitamin D (25OHD) levels and left ventricular (LV) geometry and function, evaluated through echocardiography, in 151 relatively young subjects with untreated primary hypertension without organ damage. Comorbidities and concomitant therapy were exclusion criteria. Patients were overweight (BMI: 29.7 kg/m²) and had a borderline-high lipid profile (HDL-cholesterol: 42.4 mg/dL; LDL-cholesterol: 124.0 mg/dL) but only mild hypertension (clinic systolic blood pressure (BP): 146.5 mm Hg; diastolic BP: 91.6 mm Hg). Briefly, the usual phenotype of mild hypertension in primary prevention. Of note, median 25OHD (14.3 ng/mL) was below the cut-off value for vitamin D sufficiency anyhow defined (2, 3).

In vitamin D-deficient patients (25OHD<20 ng/mL), significantly higher (+41%) left ventricular mass index (LVMI) was found, being mean LVMI above the cut-off value for left ventricular hypertrophy (LVH) (4). However, the prevalence of LVH in the two subgroups was not analyzed. Tissue Doppler (TD) examination evidenced a worse systole-diastolic profile in 25OHD deficiency, with higher mean TD- myocardial performance index (TD-MPI). An altered mean TD-MPI (>0.40) (5) was also present in 25OHD sufficiency subgroup. Multivariate stepwise regressions confirmed the inverse relationship among 25OHD and LVMI, and between 25OHD and TD-MPI. Vitamin D-deficient patients had a better lipid profile (10% lower total and LDL cholesterol), and a positive relationship between 25OHD and LDL persisted after multivariate analysis.

This is the first evidence of a relationship between TD-MPI and 25OHD in adults. Study patients were free from systolic dysfunction (mean ejection fraction: 63.7%) thus an altered TD-MPI implied diastolic dysfunction, that is a known early consequence of hypertension A large retrospective study did not confirm the association between 25OHD and TD-MPI: however, confounding factors and co-morbidities were more prevalent (6). LVMI and LVH have been previously associated with lower 25OHD levels, particularly in hypertension (7). Given the influence of 25 OHD on parathormone (PTH) and the worse cardiovascular (CV) risk profile of chronic kidney disease (CKD) patients (characterized by elevated PTH values), increased PTH may influence some associations of 25OHD deficiency, including the one with LVH in non-CKD-patients too. In the Cardiovascular Health Study, an association between LVH and PTH, but not with 25OHD, has been reported (8). Furthermore, impaired diastolic LV function evaluated through MPI has been found in primary hyperparathyroidism (9). However, in the present study, PTH was not a significant covariate in multivariate models. The relationship between lipid profile and 25OHD contrasts with most literature findings (10), and given the limited numbers in the study, it should be cautiously considered.

To date, no clear benefits on CV diseases have emerged from randomized clinical trials (RCTs) on vitamin D supplementation (11, 12). Large RCTs, such as the VITAL study (13), are now ongoing; although evidences from CKD patients dampen the enthusiasm on anti-hypertrophic effects of vitamin D (14); this potential relationship is being studied (15). PTH reduction secondary to vitamin D supplementation has to be considered as a possible mediator of supplementation effects.

We are looking forward to these results, hoping to get new insight into this field. Currently, however, vitamin D supplementation for CV prevention and/or treatment is not supported with evidenced and should be avoided.

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ERRATUM

In the article by Jonathan Lipton et al., entitled “International Research Interdisciplinary School 8-12 June 2015, Bosteri, Cholpon-Ata, Issyk-Kul, Kyrgyzstan” (Anatol J Cardiol 2015; 15: 694-700) that was published in the August 2015 issue of the Anatolian Journal of Cardiology, two of the contributing authors were erroneously omitted from the author list during the production process. Upon receipt of the written request of the contributing authors, the Editorial Board reviewed the case and approved the author list to be corrected as follows.

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