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Letter to the Editor

Association between vitamin D deficiency and heart failure in the elderly

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Dear Sir

Vitamin D (25-OH-vitamin D) is a hormone which acts on the calcium-phosphorus metabolism and also has extraskeletal effects. In the cardiovascular system, it regulates the renin-angiotensin-aldosterone system (RAAS), inhibits vascular smooth muscle proliferation, and suppresses cardiac hypertrophy and hypercontractility [1].

We assessed the relationship between vitamin D deficiency and heart failure (HF) in an elderly population. We carried out a prospective case-control study in the Internal Medicine Department, Rio Hortega Hospital, Valladolid in 2010. Twenty-five patients were diagnosed with HF and 19 were institutionalized controls with no history of cardiovascular disease (CVD). The age of patients and control group was similar (83 ± 7 years vs. 85 ± 8 years, p > 0.05). The sex distribution didn’t show differences. HF was diagnosed according to clinical and laboratory criteria (B-type natriuretic peptide > 400 pg/mL). Vitamin D insufficiency was defined as levels < 20 ng/mL and deficiency as < 10 ng/mL. Two-dimensional echocardiography evaluated systolic and diastolic function, pulmonary artery systolic pressure (PASP), atrial fibrillation and valvular disease in the HF group.

Patients with HF had lower vitamin D levels than controls (8.47 ± 4.85 vs. 17.13 ± 6.44, p = 0.0001) (Fig. 1) and 78.3% had vitamin D deficiency, compared with 5.3% of controls. Vitamin D levels remained significantly higher in the HF group (p = 0.009), after stratification for institutionalization. Intact parathormone (iPTH) levels were also significantly higher in patients with HF (p = 0.0001).

Echocardiography showed a mean ejection fraction (EF) of 54% ± 15 and 45% of patients had systolic dysfunction (severe in 5%). The diastolic pattern could not be estimated in enough patients to establish a relationship between vitamin D deficiency and HF with preserved EF, because 40% of patients had atrial fibrillation. Seventy-two percent of patients had valvular disease and 80% had significant pulmonary hypertension (mean PASP 57 ± 15 mmHg).

Recent years have provided new insight into the pathophysiology of HF. Vitamin D inhibits the RAAS, which is involved in the development of heart failure and hypertension, and reduces inflammation, thereby protecting the vascular endothelium. In addition, low levels of vitamin D favour myocardial hypertrophy. Recent studies have shown an association between an increased prevalence of CVD and vitamin D deficiency [2,3].

Our findings that patients with HF had lower vitamin D levels than controls are similar to those of other studies. Kim et al. [4] and Ameri et al. [5] found hypovitaminosis D in 81% and 90%, respectively, of patients with HF. No Spanish study has previously reported this association. Ameri et al. [5] observed an increase in systolic and diastolic left ventricle diameters and volumes in patients with vitamin D deficiency. However, we found no such association, probably due to the high incidence of valvular disease and pulmonary hypertension in our sample.

Our results and those of other studies suggest a possible association between vitamin D and heart failure. However, it is unclear whether this deficit is secondary to reduced exposure to sunlight and inadequate intake in patients with HF or is a risk factor for its development. We stratified the results according to institution-alization, as these patients might be expected to have less exposure to sunlight, but patients with HF still had more-severe vitamin D deficiency, supporting the idea that this may be a risk factor for CVD [6]. Further studies should analyze the potential role of vitamin D in the pathogenesis of CVD and the possible benefits of supplementation.
References


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