Association between vitamin D deficiency and heart failure in the elderly
Ángela Ruiz de Temiño a, Judith Gil a, Teresa Pérez b, Marta González a, Mónica Pineda a, Antonio Dueñas-Laita c, José Luis Pérez-Castrillón a,⁎

a Internal Medicine Service, Hospital Universitario Río Hortega, Valladolid, Spain
b Heart Failure Unit, Cardiology Service, Hospital Universitario Río Hortega, Valladolid, Spain
c Clinical Toxicology Unit, Hospital Universitario Río Hortega, Valladolid, Spain

A R T I C L E   I N F O
Article info
Article history:
Received 3 August 2011
Accepted 17 August 2011
Available online 13 September 2011

Keywords:
Vitamin D
Heart failure
Elderly

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 institutionalization, 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.

⁎ Corresponding author at: Servicio de Medicina Interna, Hospital Río Hortega, C/ Dulzaina Nº 2, 47012-Valladolid, Spain. Tel.: +34 983 420400; fax: +34 983 331566.
E-mail address: castrv@terra.es (J.L. Pérez-Castrillón).

Available online 13 September 2011
Received 3 August 2011
Clinical Toxicology Unit, Hospital Universitario Río Hortega, Valladolid, Spain
Internal Medicine Service, Hospital Universitario Río Hortega, Valladolid, Spain
Elderly
Heart failure
Vitamin D
Keywords:
Elderly
Heart failure
Vitamin D

* Corresponding author at: Servicio de Medicina Interna, Hospital Río Hortega, C/ Dulzaina Nº 2, 47012-Valladolid, Spain. Tel.: +34 983 420400; fax: +34 983 331566.
E-mail address: castrv@terra.es (J.L. Pérez-Castrillón).
Piagment epithelium-derived factor (PEDF) blocks advanced glycation end products (AGEs)-RAGE-induced suppression of adiponectin mRNA level in adipocytes by inhibiting NADPH oxidase-mediated oxidative stress generation

Sayaka Maeda a, Takanori Matsui a, Masayoshi Takeuchi b, Sho-ichi Yamagishi a, *

a Department of Pathophysiology and Therapeutics of Diabetic Vascular Complications, Kurume University School of Medicine, Kurume, Japan
b Department of Advanced Medicine, Medical Research Institute, Kanazawa Medical University, Kanazawa, Japan

ARTICLE INFO

Article history:
Received 21 July 2011
Accepted 17 August 2011
Available online 10 September 2011

Keywords:
AGEs
Adiponectin
Insulin resistance
PEDF

Reactive derivatives from non-enzymatic glucose-protein condensation reactions, as well as lipids and nucleic acids exposed to reducing sugars, form a heterogeneous group of irreversible adducts called "advanced glycation end products (AGEs)" [1]. The formation and accumulation of AGEs have been known to progress at an accelerated rate under diabetes [1]. There is accumulating evidence that AGEs and their receptor RAGE interaction elicits oxidative stress generation and inflammatory reactions, thereby being involved in the pathogenesis of vascular complications in diabetes [2,3]. Recently, AGEs-RAGE axis has also been shown to play an important role in insulin resistance [4-7]. Indeed, AGEs attenuate cellular insulin sensitivity in cultured adipocytes by increasing reactive oxygen species (ROS) generation through the interaction with RAGE [5]. Further, an inhibitor of AGEs formation ameliorates insulin sensitivity in obese, type 2 diabetic mice [6]. Moreover, serum level of AGEs was reported to be one of the