Association Between Left Ventricle Ejection Fraction and Vitamin D Levels in Congestive Heart Failure; A Cross Sectional Study.

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ABSTRACT

Background: Congestive heart failure (CHF) is a chronic disease that incidence is growing in the population. Vitamin D effects on directly myocardial cells. The aim of this study was to investigate the association between vitamin D levels with ejection fraction of left ventricle.

Methods: The study population consisted of 101 patients admitted with heart failure. Age, gender, and demographic characteristics (diabetes mellitus, hypertension, smoking-nonsmoking, coronary artery disease) of all patients were recorded. In all patients for blood analyses vitamin D, creatine, crp and lipid profile were studied. And all patients were studied echocardiography.

Results: CHF patients, in our population 35 (34.7%) patient is female and 66 (65.3%) patient is male. The mean age of our patients was calculated as 67.4±12.9 years. Our patients laboratory parameters mean were measured for vitamin D 24.3±17.3 ng/ml. Left Ventricular Ejection Fraction (LVEF) mean was measured 31.8±9.5. In our study group LVEF and vitamin D was correlated with each other.

Conclusion: Vitamin D and LVEF had a positive correlation in heart failure patients. It may be occurred with the protective effect of vitamin D or consequence of hipovitaminosis.


Introduction

Heart failure (HF) remains a leading cause of morbidity and mortality, affecting more than 37 million people worldwide and conferring a substantial burden on the health-care system (1).

In patients with advanced HF, despite effective medical treatment regimens that target recovery of clinical and prognostic improvement, mortality and morbidity remain substantial. Vitamin D is a lipophilic, secosteroid hormone, which majorly exists in two form; vitamin D2 and vitamin D3. Vitamin D2, commonly known as ergocalciferol, is manufactured through the ultraviolet irradiation of ergosterol from yeast. It cannot be synthesized inside the human organism but can enter blood circulation with dietary sources, such as mushroom, supplementation, and fortification (2). Vitamin D3, commonly known as cholecalciferol, is the only form produced in the human organism that is synthesized in the skin through the ultraviolet irradiation of 7-dehydrocholesterol. It can also be obtained from sources like fatty fish, egg and dairy products, and dietary supplements. In mammals, synthesis of vitamin D3 gets initiated in the epidermis, with cleavage of the B ring of 7-dehydrocholesterol, under UVB radiation of wave length 290 to 315 nm. Vitamin D is affects skeletal and extraskeletal system. It effects on calcium metabolism. And also Vitamin D effects on directly myocardial cells. Vitamin D metabolites have direct effects on cardiomyocytes including anti-hypertrophic actions, regulation of extracellular matrix turnover, and modulation of contractility (3, 4, 5). In addition, vitamin D insufficiency has effect on HF prognosis. There is evidence that low serum 25OHD levels are associated with an increased risk of cardiovascular disease (CVD) (6), including hypertension, coronary artery disease, ischemic heart disease, stroke and type 2 diabetes (7, 8, 9, 10, 11, 12). Decreased Vitamin...
D levels are common in heart failure patients. There are several mechanisms by which vitamin D may be associated with CVD including its effect on the rennin-angiotensin system, vessel compliance, blood pressure, parathyroid hormone level, and also glycemic control. Correlation of low vitamin D status with cardiovascular diseases is studied in several studies. Vitamin D status has been implicated in the pathophysiology of HF. Vitamin D deficiency is the cause and consequence of heart failure. The aim of this study was to investigate the association between vitamin D levels with ejection fraction of left ventricle.

Methods
The study population consisted of 101 patients admitted with heart failure. The patients were retrospectively collected in Adana State Hospital between 2013-2015. We enrolled symptomatic HF patients (New York Heart Association functional class ≥II). All patients’ demographic characteristics (age, gender, smoking-nonsmoking), underlying diseases (diabetes mellitus, hypertension, coronary artery disease), past medical history (coronary artery disease, a history of by-pass, coronary artery intervention and angiography), patient’s blood pressure and pulse were recorded.

Blood test was taken on the first day of hospitalization. 25 (OH) D was measured in ng/mL by enzyme-linked immunosorbent assay (EIA; Immuno Diagnostic Systems, Boldon, UK). Patients with 25 (OH) D levels ≤20 ng/mL are considered vitamin D insufficient. Examinations were performed using an iE33 ultrasound system (Philips Healthcare, Bothell, WA, USA) with an S5-1 transducer (Philips Healthcare). Patients were examined in the left lateral decubitus position. Measurements were recorded from parasternal long-axis and short-axis views, and apical four- and two-chamber views. All examinations were performed by a single skilled sonographer. All patients underwent echocardiography. LV volume was measured from the end-diastolic and end-systolic endocardial borders on apical four-chamber and two-chamber views. LVEF was calculated using the modified Simpson’s method for biplanar assessment, and expressed as a percentage.

All analyses were conducted using SPSS 23.0 (SPSS for Windows 11.5, Chicago, IL). Normally distributed continuous variables were expressed as mean ± standard deviation, non-normally distributed variables were expressed as median (minimum–maximum), and categorical variables were expressed as percentages. For correlation analysis Pearson correlation used. P-value less than 0.05 considered as significant.

Results
We examined 101 HF patients. In our population 35 (34.7%) patient is female and 66 (65.3%) patient is male. The mean age of our patients was calculated as 67.4±12.9 years. In our study population 46 (45.5%) of our patients had diabetes and 63 (62.4%) had hypertension. 40 (39.6%) of our patients had smoking history and 86 (85%) of our patients had coronary atherosclerosis history. Our patients systolic blood pressure mean was calculated 123.8±24 mmhg and diastolic blood pressure mean was calculated 73.9±11.8 mmhg. Our patients laboratory parameters mean were measured for creatinine 1.2±0.6 mg/dl, low density lipoprotein (LDL) 91.7±36.3 mg/dl, triglyceride (TG) 103.3±41.8 mg/dl, hemoglobin (Hb) 12.2±1.8 g/dl, C-reactive protein (CRP) 3.7±6.3 mg/dl and vitamin D 24.3±17.3 ng/ml given Table 1. LVEF mean was measured 31.8±9.5.

In our study group LVEF and vitamin D was correlated with each other shown in Table 2. (P<0.005)

Table 1. Laboratory parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean</th>
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<tbody>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.2±0.6</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>91.7±36.3</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>103.3±41.8</td>
</tr>
<tr>
<td>WBC (cell/mm³)</td>
<td>10±5</td>
</tr>
<tr>
<td>HB (g/dl)</td>
<td>12.2±1.8</td>
</tr>
<tr>
<td>CRP (mg/dl)</td>
<td>3.7±6.3</td>
</tr>
<tr>
<td>Vitamin D (ng/dl)</td>
<td>24.3±17.3</td>
</tr>
</tbody>
</table>

CRP: C reactive protein, HB: hemoglobin, LDL: low density lipoprotein, TG: triglyceride, WBC: white blood cell

Table 2. Correlations

<table>
<thead>
<tr>
<th>LVEF</th>
<th>Pearson Correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D</td>
<td>.500**</td>
<td>.000</td>
<td>101</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed). LVEF: left ventricular ejection fraction

Discussion
Vitamin D extra skeletal effects have attracted much scientific attention over the last few decades and convincing data suggest a possible role of vitamin D in cardiovascular, autoimmune and cancer diseases. The present study shows high prevalence of vitamin D deficiency in patients with heart failure. Our findings are consistent with other studies, showing reduced circulating levels of vitamin D in heart failure patients (13). In this setting, vitamin D deficiency may be a consequent, or contributing factor to heart failure. On the other hand, several animal and human studies suggest that hypovitaminosis D may be a contributing factor to heart failure (14). In a recent study by Kim et al., hypovitaminosis D was highly prevalent in US adults with cardiovascular disorders, particularly those with both coronary heart disease and heart failure (14). Epidemiological data indicate that deficiency of vitamin D is common among CVDs patients with circulating 25(OH)D levels less than 20 ng/mL (15). Similarly, a reduced plasma level of 25(OH)D of around 25 ng/mL has been associated with an increased risk of hypertension (16, 17). A population-based study and meta-analyses associated very low levels of 3–4.8 ng/mL of plasma 25(OH)D levels with an increase
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In multivariable-adjusted risk by 40% for ischemic heart disease, by 64% for myocardial infarction (MI), and by 57% for early death when compared with the individuals having plasma 25(OH)D levels of 18.83–28.44 ng/mL (18). In our study, there was direct correlation between serum vitamin D and its deficiency with LVEF. In conclusion, vitamin D and LVEF had a positive correlation in heart failure patients. It may be occurred with the protective effect of vitamin D or consequence of hipovitaminosis. Taking vitamin D in can reduce mortality in people with CVDs. The low levels of vitamin D in HF patients are associated with poor physical function

References


