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ORIGINAL RESEARCH

Evaluation of relation of serum parathyroid hormone levels with severity and duration of heart failure

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ABSTRACT

Background: Heart failure (HF) is a clinical illness that affects people with structural and/or functional abnormalities of the heart that are either acquired or hereditary. The present study was conducted to evaluate relation of serum parathyroid hormone levels with severity and duration of heart failure.

Materials & Methods: 70 patients with CCF of both genders were selected. Parameters such as ejection fraction, and NYHA grading was recorded. Serum parathyroid hormone levels were measured by chemiluminescence immunoassay technique.

Results: Out of 70 patients, 40 were males and 30 were females. Out of 25 patients with duration >2 years, 16 had serum PTH <72pg/ml and 9 had <72pg/ml. Out of 30 patients with EJ <35%, 12 had serum PTH >72pg/ml and 18 had <72pg/ml. Out of 22 patients with EF 35-40, 7 had serum PTH >72pg/ml and 15 had serum PTH <72pg/ml. Out of 18 with EJ >40, 2 had serum PTH >72pg/ml and 16 had <72pg/ml Out of 43 grade II patients, 14 had serum PTH >72pg/ml and 29 had serum PTH <72pg/ml. Out of 27 grade II patients, 7 had serum PTH >72pg/ml. Out of 25 patients with duration 1-2 years, 24 had serum PTH >72pg/ml and 20 had serum PTH <72pg/ml. The difference was significant (P< 0.05).

Conclusion: Both inside and outside of hospitals, elevated rates of cardiac morbidity and death have been associated with blood PTH and other serum-derived indicators of cardiomyocyte necrosis.

Keywords: congestive cardiac failure, chemiluminescence immunoassay, parathyroid hormone

INTRODUCTION

Heart failure (HF) is a clinical illness that affects people with structural and/or functional abnormalities of the heart that are either acquired or hereditary. A variety of clinical symptoms, including fatigue and dyspnea, along with indicators like edema and rales, are experienced by patients with heart failure (HF), which lowers their quality of life, increases their risk of hospitalization, and shortens their life expectancy.[1] Worldwide, 38% of heart failure cases were caused by ischemic heart disease, with the remaining 34% being caused by hypertensive heart disease, rheumatic heart disease, and cardiopulmonary illness. A broader perspective on CHF recognizes its systemic causes, which have been described as an interference with the neuroendocrine-immune system.[2]

An abnormal increase in serum PTH leads to cardiomyocytes accumulating excessive quantities of calcium intracellularly, which ultimately results in replacement fibrosis and myocyte death.[3] The nonphysiological opening of the mPTP in mitochondria is brought on by oxidative stress and Ca2+ excess. This results in the structural and functional degradation of these organelles based on osmotic forces, which in turn triggers the last common cell death pathway that leads to necrosis and replacement fibrosis in cardiomyocytes.[4] Due to the progressive loss of contractile elements, the deposition of fibrous tissue, and stiff in-series and in-parallel elastic elements made primarily of type I fibrillar collagen with the tensile strength of steel, this once-effective muscular pump gradually fails during the systolic and/or diastolic phases of the cardiac cycle.[5]The present study was conducted to evaluate relation of serum parathyroid hormone levels with severity and duration of heart failure.

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MATERIALS & METHODS

The present study was conducted on70 patients with congestive cardiac failure (CCF)of both genders.All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. Every patient had a 2-D echocardiogram. Every patient had measurements such ejection fraction, NYHA grading, CBC, urine microscopy, RFT, LFT, RBS, serum electrolytes, lipid profile, and thyroid function tests where necessary. The method used to test serum parathyroid hormone levels was chemiluminescence immunoassay. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table: I Distribution of patients

Total- 70		
Gender	**Male**	**Female**
Number	40	30

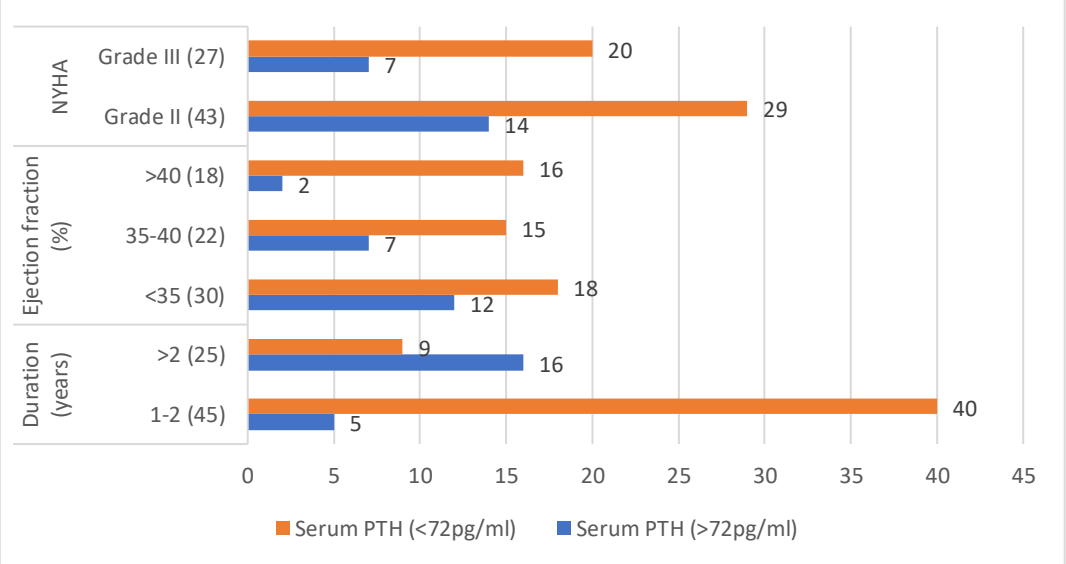
Table I shows that out of 70 patients, 40 were males and 30 were females.

Table: II Assessment of parameters

Parameters	Variables	Serum PTH (>72pg/ml)	Serum PTH (<72pg/ml)	P value
Duration (years)	1-2 (45)	5	40	0.05
	>2 (25)	16	9	
Ejection fraction (%)	<35 (30)	12	18	0.05
	35-40 (22)	7	15	
	>40 (18)	2	16	
NYHA	Grade II (43)	14	29	0.02
	Grade III (27)	7	20	

Table II, graph I shows that out of 25 patients with duration >2 years, 16 had serum PTH <72pg/ml and 9 had <72pg/ml. Out of 30 patients with EJ <35%, 12 had serum PTH >72pg/ml and 18 had <72pg/ml. Out of 22 patients with EF 35-40, 7 had serum PTH >72pg/ml and 15 had serum PTH <72pg/ml.Out of 18 with EJ >40, 2 had serum PTH >72pg/ml and 16 had <72pg/mlOut of 43 grade II patients, 14 had serum PTH >72pg/ml and 29 had serum PTH <72pg/ml. Out of 27 grade II patients, 7 had serum PTH >72pg/ml. Out of 25 patients with duration 1-2 years, 24 had serum PTH >72pg/ml and 20 had serum PTH <72pg/ml. The difference was significant (P< 0.05).

Graph I: Assessment of parameters



DISCUSSION

Although parathyroid hormone (PTH) is a major regulator of bone and mineral metabolism, there is a growing body of evidence suggesting that hyperparathyroidism is associated with adverse outcomes in patients with cardiovascular diseases. In patients with advanced heart failure (HF), urinary and fecal excretion of calcium induced by hyperaldosteronism, together with calcium excretion caused by high dosages of loop diuretics, results in secondary hyperparathyroidism.[6] In experimental studies, PTH has been shown to have direct actions of

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hypertrophy and arrhythmogenicity on myocardial cells and tissues partly through cytoplasmic calcium overloading and triggered oxidative stress.[7,8]The existence of oxidative stress in a variety of tissues, including skin, skeletal muscle, the heart, peripheral blood mononuclear cells (lymphocytes and monocytes), and blood, which overwhelms endogenous antioxidant defense.[9] A proinflammatory phenotype characterized by elevated levels of circulating chemokines and cytokines, including interleukin-6 and tumor necrosis factor (TNF)-α3.9, as well as activated peripheral blood mononuclear cells a catabolic state known as cardiac cachexia, which results in the loss of soft tissues and bone and is partly caused by an unfavourable nitrogen and calorie balance.[10]The present study was conducted to evaluate relation of serum parathyroid hormone levels with severity and duration of heart failure.

We found that out of 70 patients, 40 were males and 30 were females. Hagstrom et al[11] in their study 864 elderly men without HF or valvular disease at baseline (mean age 71 years, the ULSAM study) the association between plasma (P)-PTH and HF hospitalization was investigated adjusted for established HF risk factors (myocardial infarction, hypertension, diabetes, electrocardiographic left ventricular hypertrophy, smoking, and hypercholesterolaemia) and variables reflecting mineral metabolism (S-calcium, S-phosphate, P-vitamin D, S-albumin, dietary calcium and vitamin D intake, physical activity, glomerular filtration rate, and blood draw season). During follow-up (median 8 years), 75 individuals were hospitalized due to HF. In multivariable Cox-regression analyses, higher P-PTH was associated with increased HF hospitalization (hazard ratio for 1-SD increase of PTH, 1.41, 95% CI 1.12-1.77, P = 0.003). Parathyroid hormone also predicted hospitalization in participants without apparent ischaemic HF and in participants with normal P-PTH.

We found that out of 25 patients with duration >2 years, 16 had serum PTH <72pg/ml and 9 had <72pg/ml. Out of 30 patients with EJ <35%, 12 had serum PTH >72pg/ml and 18 had <72pg/ml. Out of 22 patients with EF 35-40, 7 had serum PTH >72pg/ml and 15 had serum PTH <72pg/ml. Out of 18 with EJ >40, 2 had serum PTH >72pg/ml and 16 had <72pg/ml Out of 43 grade II patients, 14 had serum PTH >72pg/ml and 29 had serum PTH <72pg/ml. Out of 27 grade II patients, 7 had serum PTH >72pg/ml. Out of 25 patients with duration 1-2 years, 24 had serum PTH >72pg/ml and 20 had serum PTH <72pg/ml. In a study by Wannamethee SG[12], 3731 men aged 60 to 79 years with no prevalent HF followed up for a mean period of 13 years, in whom there were 287 incident HF cases. Elevated PTH (≥55.6 pg/mL; top quarter) was associated with significantly higher risk of incident HF after adjustment for lifestyle characteristics, diabetes mellitus, blood lipids, blood pressure, lung function, heart rate, renal dysfunction, atrial fibrillation, forced expiratory volume in 1 second, and C-reactive protein. The increased risk was seen in both men with and without previous myocardial infarction or stroke. Elevated PTH was significantly associated with N-terminal probrain natriuretic peptide, a marker of left ventricular wall stress. By contrast, 25-hydroxyvitamin D and other markers of mineral metabolism including serum calcium and phosphate showed no significant association with incident HF after adjustment for age.

The shortcoming of the study is small sample size.

CONCLUSION

Authors found that both inside and outside of hospitals, elevated rates of cardiac morbidity and death have been associated with blood PTH and other serum-derived indicators of cardiomyocyte necrosis.

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