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Original Research Article.

Calcium and Phosphorus Metabolism in Patients with Congestive Heart Failure: A Cross-Sectional Study

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ABSTRACT

Background: To study the role of calcium and phosphorus metabolism including the hormones (Vitamin D and PTH) controlling them in patients with congestive heart failure. While extensive studies are available regarding bone mineral metabolism in CKD patient with regard to CVD / HF similar studies in non-CKD patients are sparce, hence the present study.

Methods: A cross sectional study was done on a total of 50 patients with signs, symptoms and Echocardiographic findings suggestive of heart failure admitted in medicine wards of Government Medical College Patiala. Patients with type 2 diabetes mellitus, chronic renal disease, thyroid disease and pregnant/lactating mothers were excluded from the study. Serum calcium, phosphorus, Vitamin D and parathyroid hormone were estimated in all patients.

Results: The study population consisted of 54% males, 46% females. Mean age was 61.78 +/- 9.18 years. Majority of the patients had cardiac ejection fraction in the range of 22-28%. CAD and HT were present in 58% and 66% patients respectively. Mean values for Vitamin D levels were 19.5 ng/ml. While in patients with normal ejection fraction Vitamin D levels was 29.34ng/ml, it progressively decreased with decreasing ejection fraction, patients with ejection fraction of <20% had Vitamin D levels of 8.98 ng/ml. Similarly mean value of PTH was 89.07 pg/ml and progressively increased with decreasing ejection fraction. Maximum valve of 138.27 pg/ml was found in patients with ejection fraction of <20%. Serum phosphorus value progressively increased with decreasing ejection fraction. Similar trends were found for serum calcium levels also.

Conclusion: There is an association between congestive heart failure and calcium and phosphorus metabolism (along with their controlling hormones Vitamin D and parathyroid hormones). Rising levels of phosphorus and PTH and decreasing levels of Vitamin D are directly related with progressive heart failure.

Keywords: Calcium, Phosphorus, Metabolism, Congestive Heart Failure.

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INTRODUCTION

The current American Heart Association (AHA) guidelines define heart failure (HF) as a complex clinical syndrome that result from structural or functional impairment of ventricular filling or ejection of blood which in turn leads to the cardinal clinical symptoms of dyspnea and fatigue and signs of HF namely edema and rales[1]. HF is now classified as heart failure with a preserved Ejection Fraction (HFpEF) i.e. EF >50% and HF with reduced EF (HFrEF) i.e. EF<40%. Patients with a LV Ejection Fraction (EF) between 40-50% are considered as having a borderline or mid-range EF[1].

HF is accompanied by a series of adaptive/maladaptive neurohormonal, adrenergic and cytokine system changes resulting in excessive salt and water retention. It is important to understand the metabolic milieu of HF. It is now established that neurohormonal changes (hyperaldosteronism and excessive catecholamines) leads to a salt and water retaining status.[2,3] This hyperaldosteronism leads to calcium loss and hence secondary hyperparathyroidism. Vitamin D deficiency has been pointed out in CHF, it may be in part due to reduced exposure to sun (majority of patients being homebound) this further adds to secondary hyperparathyroidism. Excess Parathyroid hormone (PTH) leads to excessive intracellular calcium leading on to oxidative stress.[4,5] The use of diuretics in CHF is universal for symptomatic relief. This further adds on to calcium loss and secondary hyperparathyroidism. The resulting systemic inflammation affects the myocardium and vessels.[6] Role of phosphate and FGF-23 is coming up in many recent clinical and experimental studies. Deranged calcium phosphate metabolism is being considered as a major non-conventional cardiovascular risk factor.[11,12,13]

Studies done in Chronic renal failure (CRF) patients have clearly established the role of bone mineral metabolism in cardiovascular disease (CVD). However, contribution of these factors in producing CVD in non-Chronic kidney disease (CKD) patients is not established.[30] It is being studied in experimental and clinical studies. The traditional risk factors like Diabetes Mellitus (DM), Hypertension (HTN) and dyslipidaemia are known to produce CVD but despite our knowledge of modifying these risk factors by maximal pharmacological intervention, CV outcome is not improving.

Vitamin D deficiency seems to be linked to various mechanisms that play a significant role in the pathogenesis of CHF. These mechanisms include Presence of oxidative stress in different tissues including the skin, skeletal muscles, heart, and peripheral blood mononuclear cells**.** Activation of pro-inflammatory cytokines such as interleukin (IL)-8 and tumor necrosis factor (TNF)-alpha. Vitamin D suppresses the pro-inflammatory state by downregulating nuclear factor-*κ*B activity and decreasing IL-6, IL-12, IL-10, interferon-*γ* and TNF-*α* production. At the same time, it increases anti-inflammatory cytokines.[7] The levels of Vitamin D have been linked to many of the clinical and laboratory parameters of CHF including the New York Heart Association (NYHA) functional classifications, NT pro-BNP (N-terminal of the prohormone brain natriuretic peptide), NT-proANP (N-terminal of the prohormone atrial natriuretic peptide) and even LVEF (left ventricle ejection fraction).[8,9]

In heart failure, secondary hyperparathyroidism is a consequence of renin-angiotensin-aldosterone activation, chronic hyperaldosteronism, and loop diuretic use which leads to calcium excretion. The result is an inflammatory state with adverse effects on myocardial remodeling and systemic complications. Excess PTH is correlated with a higher incidence of hypertension, left ventricular hypertrophy, heart failure, cardiac arrhythmias and valvular calcific disease which contributes to higher cardiac morbidity and mortality.[10]

With this background the present study was attempted to look into the effects of calcium and phosphorous metabolism in HF along with the hormones (Vitamin D and PTH) controlling their metabolism.

MATERIAL AND METHODS

A Hospital based Cross Sectional study was conducted on fifty patients with signs, symptoms and echocardiographic findings suggestive of congestive heart failure admitted in medicine wards of Rajindra Hospital/Government Medical College, Patiala, a tertiary care teaching hospital. The study proposal and ethical procedure was approved by the ethics Committee of Government Medical College/Rajindra Hospital, Patiala. The study included patients of either sex, age more than 18 years, presenting with clinical features of heart failure and/or echocardiography criteria suggestive of heart failure. We excluded patients with type 2 diabetes mellitus, chronic renal disease with secondary hyperparathyroidism, pregnant and lactating females and patients with thyroid disease.

Serum Vitamin D and Parathyroid Hormone levels were measured using ELISA method. To define the deficiency status, serum levels of 25-OHD were further classified into three groups: <10 ng/ml, deficient; 10-30 ng/mL, insufficient; >30 ng/mL, sufficient. Serum iPTH levels were also measured using ELISA method with normal range of 10-65 pg/ml. Levels >65 pg/ml were considered as hyperparathyroidism. Serum Calcium and Serum Phosphorus were measured using Arsenazo III and UV Molybdate method respectively. Normal values of Calcium in adults were taken as 8.6-10.2 mg/dl, similarly normal values for phosphorus were taken as 2.5-4.5 mg/dl.

Statistical Analysis: The collected data was analysed using SPSS statistics software 21.0 Version. To describe the data descriptive statistics, frequency analysis and percentage analysis was used for categorical variables and the mean & S.D were used for continuous variables. To find the significance in categorical data, Chi Square/Fisher Exact test was used; for quantitative data one-way ANOVA test and Pearson's coefficient was applied for correlation. p-value of <0.05 was considered as statistically significant and <0.001 as statistically highly significant.

OBSERVATIONS AND RESULTS

Data was collected and analysed from fifty patients admitted in our hospital with diagnosis of congestive heart failure (Table 1). In the study the mean age of population was 61.78 ± 9.18 years. The majority of the patients were in the age group 61-70 years. Our sample size consisted of both males (54%) and females (46%). One of the classical risk factors in the pathogenesis of CHF includes hypertension. In the present study, 33 (66%) patients were found to be hypertensive and the remaining 17 (34%) patients did not have hypertension. In the study 58% of patients had coexisting coronary artery disease.

Study population having heart failure in the range of EF 30-40% was 28%, only 2% had EF less than 20% while 20% patients had normal EF (50-60%). Majority of them had functional class NYHA II /III. In this study the patients who had EF between 50-60%, the mean levels of serum vitamin D were 29.34 ±2.42 ng/ml and the patients who had EF between 40-50%, the mean levels decreased to 25.49±3.56 ng/ml. While the patients who had EF between 30-40%, the mean vitamin D levels further reduced to 17.02 ±2.89 ng/ml. The mean levels of Vitamin D in patient groups with EF of 20-30% and those with EF of less than 20 % were 11.24 ±1.78 ng/ml and 8.98 ±3.19 ng/ml respectively. On correlation analysis EF was observed to show a highly significant correlation with serum Vitamin D levels in CHF patients (r=-0.447; p=0.001). Hence, our results suggest that mean Vitamin D levels decreased with decreasing ejection fraction.

The patients who had EF between 50-60%, the mean levels of serum PTH were 61.34 pg/ml and the patients who had EF between 40-50%, the mean levels increased to 70.95 pg/ml. While the patients who had EF between 30-40%, the mean PTH levels further increased to 94.68 pg/ml. The mean levels of PTH in patient groups with EF of 20-30% and those with EF of less than 20 % were 112.13 pg/ml and 138.27 pg/ml respectively. This corroborates with Vitamin D levels whose deficiency was directly related to LV systolic function i.e. lower the levels of Vitamin D, lower the cardiac Ejection Fraction.

The patients who had EF between 50-60%, the mean levels of serum Calcium were 9.5 mg/dl and the patients who had EF between 40-50%, the mean levels decreased to 9.3 mg/dl. While the patients who had EF between 30-40%, the mean Calcium levels were 9.7 mg/dl. The mean levels of Calcium in patient groups with EF of 20-30% and those with EF of less than 20 % were 8.9 mg/dl and 9.3 mg/dl respectively. (Figure 1). These

findings probably can be explained on the basis of Calcium paradox i.e. myocardial injury produced by excessive intracellular accumulation of Calcium due to secondary hyperparathyroidism.

The patients who had EF between 50-60%, the mean levels of serum phosphorous were 4.23 mg/dl and the patients who had EF between 40-50%, the mean levels increased to 4.41 mg/dl. While the patients who had EF between 30-40%, the mean phosphorus levels further increased to 4.93 mg/dl. The mean levels of phosphorous in-patient groups with EF of 20-30% and those with EF of less than 20 % were 5.08 mg/dl and 4.75 mg/dl respectively (Figure 2). The results also show that in patients who were using diuretics, vitamin D levels were lower (15.7 versus 26.3), PTH levels were higher (99.3 versus 70.8), Calcium levels were lower (9.3 versus 9.4) and phosphorus levels were higher (4.9 versus 4.3) compared to patients who were not using diuretics. (Table 2)

Decompensation of heart failure is associated with worsening of symptoms and signs of HF. It is associated with increased cardiac morbidity and mortality. In our study, thirty-eight (76%) patients were in a compensated state and only twelve (24%) patients had decompensated heart failure**.** It was found that decompensation of CHF is negatively correlated with Vitamin D levels while it had a positive correlation with PTH levels (r=-0.588 and 0.620 respectively). The mean difference in values between both groups was found to be statistically significant (p<0.0001*). This shows that decompensated CHF is associated with decreased Vitamin D levels while it is associated with increased PTH levels.

Table 1: Epidemiological and clinical profile of patients

Age	**Mean Age**	**16%**	**20%**	**40%**	**18%**
	61.78±9.18 Yr	(40-50 Yr)	(51-60 Yr)	(61-70 Yr)	(71-80 Yr)
Sex			Males-54%		
			Females-46%		
NYHA Class	20%	32%	28%	20%	
	(NYHA-I)	(NYHA-II)	(NYHA-III)	(NYHA-IV)	
Ejection Fraction	20%	22%	28%	26%	2%
	(EF50-60%)	(EF40-50%)	(EF30-40%)	(EF20-30%)	(EF<20%)
Compensated HF			76%		
Decompensated HF			24%		
CAD	Present: 58%			Absent: 42%	
Hypertension	Present: 66%			Absent: 34%	
Diuretic			64% used diuretics.		
			36% did not use diuretics		

Table 2: Laboratory parameters and their correlation with Cardiac Ejection Fraction

	Mean	**With diuretics**	**Without diuretics**	**EF < 20%**	**EF 20-30%**	**EF 30-40%**	**EF 40-50%**	**EF 50-60%**
Vit-D (ng/ml)	19.52	15.7	26.3	8.98	11.24	17.02	25.49	29.34
PTH (pg/ml)	89.07	99.3	70.8	138.27	112.13	94.68	70.95	61.34
Ca (mg %)	9.36	9.3	9.4	9.3	8.9	9.7	9.3	9.5
P (mg %)	4.7	4.9	4.3	4.75	5.08	4.93	4.41	4.23

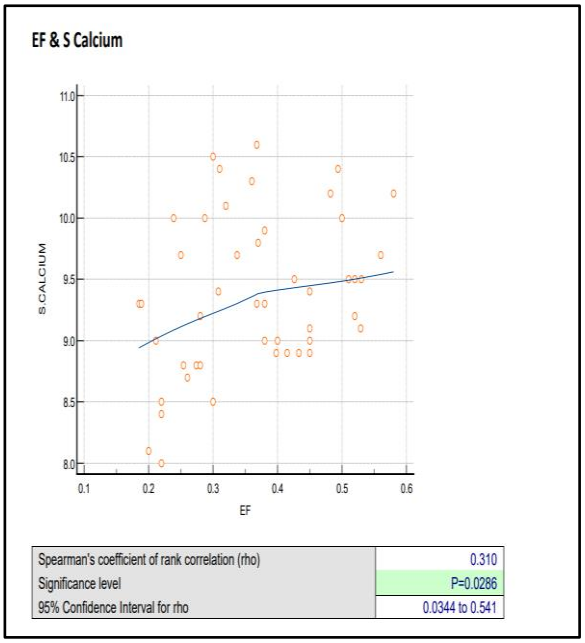

Figure 1

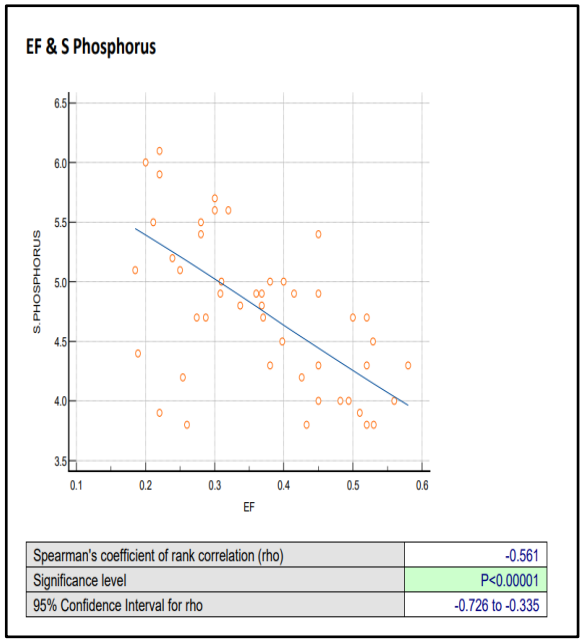

Figure 2

DISCUSSION

The cardiovascular diseases and the resulting heart failure remain one of the topmost causes of death all over the globe. While we understand the conventional risk factors like DM, HTN and dyslipidemia in a much better way, aggressive control of these conventional risk factors has not provided the desired benefits in terms of mortality reduction.[14-16]

Chronic Kidney disease (CKD) represents a special patient subgroup with enhanced CVD risk. It is increasingly proven in studies, both experimental and epidemiological that Calcium and Phosphate metabolism is a major non-conventional risk factor for CVD.[11] By analogy one questions whether calcium phosphorus metabolism and their regulatory hormones like vitamin D, PTH and FGF 23 are new villains for CVD in patients who do not have CKD.[12,13]

The results of our present study points towards the same in many ways. As noted in various studies done earlier, supplementation of calcium in CKD patients resulted in progression of vascular calcification.[17-19] In non-CKD patients also high dose calcium supplementation leads to calcium deposition in tissues. This further allows phosphate to accumulate intracellularly.[20] In our study when calcium levels were correlated with EF it was noted that rising levels of calcium were associated with falling EF.[21-23]

Phosphorus is tightly regulated by normal kidney function, PTH and FGF 23. Recent evidence points out that even with normal range of phosphorus adverse CV outcomes occur as suggested by Cholesterol and Recurrent Events (CARE) study. Numerous other studies have also confirmed this association. Phosphorus is now being touted as the new cholesterol recently.[24] An elevated phosphorus level contributes to vascular calcification, atherosclerosis and myocardial hypertrophy.[25] On similar lines, in present study it is clearly seen that when phosphorus is correlated to EF /NYHA class, rising levels of phosphorus positively correlate to worsening EF/NYHA class.

Moreover, in our study phosphorus levels seem to be independent of both hyperparathyroidism and vitamin D deficiency. In non-CKD patients' hyperparathyroidism is associated with hypophosphatemia so is vitamin D deficiency. But in the present study falling EF is associated with rising phosphorus despite rising PTH levels. Similarly falling vitamin D levels are directly related to falling EF. Vitamin D supplementation increases phosphate levels. This hypothesis may further suggest that calcium and vitamin D supplementation may be harmful in CVD/HF. This will negate any positive benefit that vitamin D supplements may provide.[26] Further, results of recently completed Finnish Trial of vitamin D supplementation[27] failed to lower CVD events. With this rising evidence in favour of phosphate /FGF 23 levels as the novel risk factors for CVD, further studies are needed to look at this association. This will surely have implications of therapeutic interventions namely phosphate binders or FGF 23 antagonists benefitting CVD/HF.[28,29]

CONCLUSION

Emerging clinical and experimental evidence is growing in favour of calcium, phosphorus and their regulating hormones like Vitamin D, PTH and FGF23 as novel risk factors for CVD. For conventional risk factors like HTN, DM and dyslipidemia maximal control has not given the desired result. The present study suggests that therapeutic intervention aimed at phosphorus burden reduction and FGF 23 / PTH antagonist might help. This may mean that sevelamer will be the next in the armamentarium of CVD / HF in non-CKD patients as well. Further studies are needed to substantiate this hypothesis.

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