Role of High Parathormone Level as a Risk Factor for Cardiac Dysfunction in Hemodialysis Patients

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Abstract: Chronic kidney disease (CKD) is a worldwide public health problem that affects 5% to 10% of the world population, with increasing prevalence and adverse outcomes. Cardiovascular disease is the leading cause of death in patients with CKD. Cardiac hypertrophy is a major complication in end stage renal disease (ESRD) patients. Disturbances in mineral metabolism are common complications of CKD and an important cause of morbidity and decreased quality of life. Importantly, increasing evidence suggests that these disturbances are associated with changes in arterial compliance, cardiovascular calcification, bone disorders and all-cause cardiovascular mortality reported that the presence of left ventricular hypertrophy in 74% of patients starting dialysis therapy higher i-PTH was significantly associated with higher LV mass in both genders. The objective of this work was to evaluate the relationship between serum levels of intact Para hormone (i.PTH) and the degree of myocardial dysfunction in non-diabetic normotensive hemodialysis patients. We studied 70 chronic renal failure on hemodialysis patients. The results of this study showed that, there was a highly significant positive correlation of serum iPTH and left ventricular mass index, in HD patients, Serum iPTH >300pg/ml was a predictor in part of cause cardiovascular dysfunction in HD patients and LVH. Serum calcium was a predictor in part of cause cardiovascular dysfunction in HD patients, Serum phosphorus >6.5mg/dl a predictor of cardiovascular dysfunction in HD patients and there was a highly significant positive correlation of serum phosphorus and left ventricular mass index. The conclusion from our results suggested the link i.PTH level and myocardial dysfunction in hemodialysis patients.

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Keywords: Role; High Parathormone Level; Risk Factor; Cardiac Dysfunction; Hemodialysis; Patient

1. Introduction

Chronic kidney disease (CKD) is a worldwide public health problem that affects 5% to 10% of the world population, with increasing prevalence and adverse outcomes **(Eknoyan *et al.*, 2004).**

Cardiovascular disease is the leading cause of death in patients with CKD **(London *et al.*, 2003).**

Disturbances in mineral metabolism are common complications of CKD and an important cause of morbidity and decreased quality of life. Importantly, increasing evidence suggests that these disturbances are associated with changes in arterial compliance, cardiovascular calcification, bone disorders and all-cause cardiovascular mortality **(Palmer *et al.*, 2005, Drueke *et al.*, 2010).**

Established risk factors insufficiently explain CVD occurrence. Therefore, it is important to elucidate possible associations of PTH with the development of premature cardiac diseases and CVD risk factors in chronic kidney disease patients. **(Wang *et al.*, 2006).**

The Kidney Disease Improving Global Outcomes (KDIGO) guidelines for diagnosis, evaluation, prevention and treatment of CKD-MBD have been published that the achievement of the target ranges set by the KDOQI or KDIGO guidelines is quite challenging (Young ***et al.*,** 2004) and failure to reach these targets has been shown to be associated with increase drisk for death compared to simultaneously achieving the targets for all three biochemica lparameters PTH, calcium and phosphorus **(Danese *et al.*, 2008).**

2. Patients and Methods

This work was done to assess the cardiac dysfunctions in relation to various surrogate markers of calcification in hemodialyzed patients.

Study design:

The present study included 70 ESRD patients on hemodialysis divided into 2 groups:

Group (A): 50 ESRD patients on maintenance hemodialysis for at least six months. They were 20 males and 30 females, with mean age 46.6 ± 13.1 years, mean duration of HD was 3.5 ± 2.2 years.

Group (B): 20 ESRD patients on hemodialysis with (i.PTH) < 300 pg/ml as controls. They were 14 male and 6 female, with mean age 52.6 ±13.7 years, mean duration 4 ± 1.8 years.

Informed consents were obtained from each participant. Dialysis was performed for 4 hours three times weakly using conventional heparin. Blood access

was through arterio-venous fistula. Blood flow rate was of 300 ml/min. ultrafiltration varied according to patients actual weight. The membrane used was polysalphone with surface area suitable for each patient. Bicarbonate was the buffer used throughout the study for all patients. Erythropoietin was given for each patient according to body weight, Hb and iron profile.

Inclusion criteria:

1-Patients who have end stage renal disease on regular hemodialysis for at least 6 months.

2-Patients age above 18 years.

3-Serum calcium> 8.4mg/dL. and Cax PO 4 ≥ 45 mg/dL.

Exclusion criteria:

1-Diabetes Mellitus and / or Hypertension

2-Evidence of rheumatic or congenital heart disease

3-Patients who underwent Para thyroidectomy.

4-Decompensated cardiac disease.

5-Severelyanemic patients (Hb< 7gm/dL).

6-Patients who had any malignancy or advanced liver disease.

7-Patients on regular hemodialysis for <6 months.

All patients were subjected to the following:

1- Full history taking.

2- Complete clinical examination including measurement of mean arterial blood pressure.

3-Laboratoryinvestigations, included:

1- Hemoglobin level, Serum creatinine, blood urea, fasting and postprandial blood sugar and HbA1c.

2- Intact PTH (i PTH) assay was done by using sandwitch principle test (Electrochemiluminescence Immune Assay) the immune assay for the in vitro quantitative determination of intact parathyroid hormone.

- 1st incubation: 50 ul of sample, a biotinylated monoclonal PTH-specific antibody, and monoclonal PTH-specific antibody labeled with a ruthenium complex form a sandwitch complex.

- 2nd incubation: After addition of streptavidin-coated microparticles, the complex becomes bound to the solid phase via interaction of biotin and streptavidin.

- The reaction mixture is aspirated into the measuring cell where the microparticles are magnetically captured on to the surface of the electrode. Unbound substances are then removed with ProCell. Application of a voltage to the electrode then induces chemiluminescent emission which is measured by aphotomultiplier.

- Results are determined via a calibration curve which is instrument –specifically generated by 2-point calibration and a master curve provided via the reagent barcode.

3- Assessment of the surrogate markers of calcification (serum calcium, serum phosphorous and Ca X PO4 PRODUCT) was done.

4) Echocardiographic imaging:

- The echocardiogram was performed with the patient breathing quietly and lying in the left lateral position.

- Four acoustic views (parasternal long axis, parasternal short axis, apical four chamber, and apical two chamber) were obtained to assess the systolic LV function by measuring the end diastolic and end systolic diameters of left ventricle using M-mode recording under guidance of 2D Echocardiography according to the recommendation of the American Society of Echocardiography. The end diastole at the onset of QRS complex and the end systole at the point of maximum upward motion of LV posterior wall endocardium. These measurements should be made from leasing edge to leading edge.

The M-mode LV dimensions used to estimate ventricular volume and ejection fraction if desired most simply by merely cubing the value (D)3 or using Teicholz Technique and calculating the ejection fraction (EF).

Statistical Analysis:

Data were collected and submitted to statistical analysis. The following statistical tests and parameters were used:

$$EF\ \% = \frac{LV\ end\ diastolic\ volume\ (D^3) - LV\ end\ systolic\ volume\ (D^3)}{LV\ end\ diastolic\ volume\ (D^3)}$$

mean ± standard deviation (SD), the student t-test, categorical variable were compared by mean of chi-square.

3. Results

This study included 36 patient divided into two groups:

- **Study group:**

It consisted of 21 patients divided in to two groups.

There was no significantdifference between two groups as regard Hb, FBS, serum uric acid, serum albumin and serum creatnine.

There was highly significant difference between two groups as regard s. Ca, i. PTH, and there was significant difference between two groups as regard S. PO4.

Table (1): shows clinical parameters of group A.

			Minimum	Maximum	Mean±S.D
Age (years)			18	64	46.6±13.06
Duration of hemodialysis (years)			.5	10	3.47±2.2
Blood pressure	Range(mmHg)		Mean ± SD		
	Systolic	Diastolic	Systolic	Diastolic	
	110-150	70-90	123.5±2.43	80±1.62	

Table (2): shows laboratory data of group A.

	Minimum	Maximum	Mean±S.D
Hb % (g/dl)	8.3	12.9	10.5±1.5
Serum Ca (mg/dl)	7.7	10.9	8.7±1.14
Serum Po4 (mg/dl)	3.2	6.1	5.5±1.5
Serum creatinine (mg/dl)	4.5	15	8.536±2.4
I P.T.H (pg/dl)	302.0	2439	570.862±471.9
Fasting blood sugar (mg/dl)	69	124	101.5±13.1
Serum albumin (g/dl)	3.3	4.5	3.7±0.55
Serum uric acid (mg/dl)	3.9	8	5.1±0.95

Table (3):shows echocardiographic parameters of group A.

	Minimum	Maximum	Mean±S.D
LVEDD (cm)	4.3	7.2	5.290±.5986
PWT (mm)	10.8	12.5	11.676±.4667
IVST (mm)	9.5	11.5	10.428±.4941
LVMI (g/m2)	154	196	170.20±12.685
EF%	48	69	59.54±5.369

Table (4):clinical parameters of group B.

			Minimum	Maximum	Mean±S.D
Age (years)			19	66	52.6±13.7
Duration of hemodialysis (years)			1.0	7.0	4.0±1.8
Blood pressure	Range(mmHg)		Mean ± SD		
	Systolic	Diastolic	Systolic	Diastolic	
	110-145	70-90	121.5±2.43	80±1.62	

Table (5): shows laboratory parameters of group A.

	Minimum	**Maximum**	**Mean±S.D**
Hb % (g/dl)	9.5	13.0	10.75±1.4
Serum Ca (mg/dl)	8.8	11.1	10.0±0.75
Serum Po4 (mg/dl)	3.0	5.6	4.25±0.65
Serum creatinine (mg/dl)	5.8	12.0	8.2±1.6

I P.T.H (pg/ml)	13.5	250.0	103.87±65.25
Fasting blood sugar (mg/dl)	89	119	103.4±13.2
Serum albumin (g/dl)	3.0	4.3	3.8±0.3
Serum uric acid (mg/dl)	4.8	6.7	5.61±0.62

Table (6): shows echocardiographic parameters of group B.

	Minimum	Maximum	Mean± S.D
LVEDD (cm)	4.8	6.0	5.460±.3872
PWT (mm)	10.3	12.1	11.530±.4953
IVST (mm)	9.9	11.8	10.805±.6755
LVMI (g/m2)	145	162	152.75±5.379
EF%	53	65	59.10±3.432

Table (7): shows comparison between the two groups as regard clinical parameters.

		Mean±S.D	t	*p*-value	Significance
Age (years)	Group A	46.6 ±13.0	-1.7	0.09	N.S
	Group B	52.6±13.7			
Duration of hemodialysis (years)	Group A	3.4 ± 2.2	-0.95	0.34	N.S
	Group B	4.0 ± 1.8			
Blood pressure (mmHg)					

There was no significant difference between two groups as regard age and duration of dialysis.

Table (8):shows comparison between the two groups as regard blood pressure.

Group	***Range (mmHg)***		***Mean ± SD***		***p-value***		***significance***	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
A	*110-150*	*70-90*	*123.5±2.43*	*80±1.62*	0.1	0.186	***NS***	***NS***
B	*120-145*	*70-90*	*121±1.86*	*83±1.63*				

There was no significant difference between two groups as regard blood pressure

Table (9): shows comparison between the two groups as regard laboratory parameters.

		Mean±S.D	t	*p*-value	Significance
Hb % (g/dl)	Group A	10.50 ±1	-1.16	0.1	NS
	Group B	11.00±0.8			
Serum Ca (mg/dl)	Group A	8.7 ±1.14	-4.64	<0.01	H.S
	Group B	10.0 ± 0.79			
Serum Po4 (mg/dl)	Group A	5.58 ±1.53	3.79	<0.05	S
	Group B	4.22±0.67			
Serum creatinine (mg/dl)	Group A	8.53±2.46	0.55	0.58	N.S
	Group B	8.2±1.6			
I P.T.H (pg/ml)	Group A	570.8±471.9	4.39	<0.01	H.S

	Group B	103.8±65.27			
Fasting blood sugar (mg/dl)	Group A	102.5±17.12	-1.23	0. 1	NS
	Group B	113.4±11.6			
Serum albumin (g/dl)	Group A	3.34±0.59	-0.94	0. 1	N.S
	Group B	3.8±0.3			
Serum uric acid (mg/dl)	Group A	5.53±0.98	-0.31	0.75	N.S
	Group B	5.6 ± 0.62			

Table (10): shows comparison between the two groups as regard echocardiographic parameters.

		Mean±S.D	t	*p*-value	Significance
LVEDD (cm)	Group A	5.29±0.59	-1.17	0.24	
					N.S
	Group B	5.46 ±0.38			
PWT (mm)	Group A	11.67 ±0.46	1.16	0.24	N.S
	Group B	11.53±0.49			
IVST (mm)	Group A	10.4 ±0.49	-2.58	0.01	S
	Group B	10.8 ±0.67			
LVMI (g/m2)	Group A	170.2 ±12.68	5.92	<0.01	H.S
	Group B	152.75 ±5.37			
EF%	Group A	59.54±5.36	0.33	0.73	N.S
	Group B	59.1 ±3.43			

There was no significant difference between two groups as regard LVEDD, PWT and EF.

There was significant difference between two groups as regard IVST.

There was highly significant difference between two groups as regard LVMI.

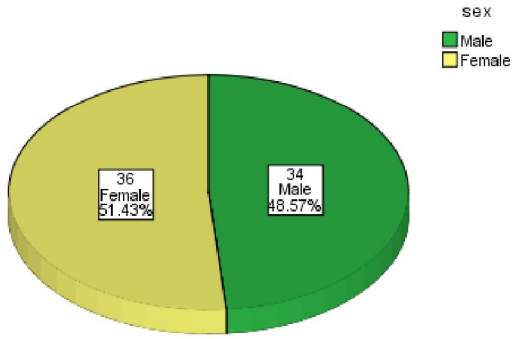

Figure (1): shows distribution of studied patients as regard sex.

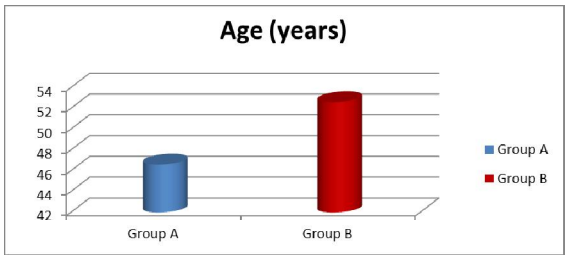

Figure (2): shows comparison between group A and group B a regard age. there was no significant difference between the two groups.(t=-1.7, p=0.09).

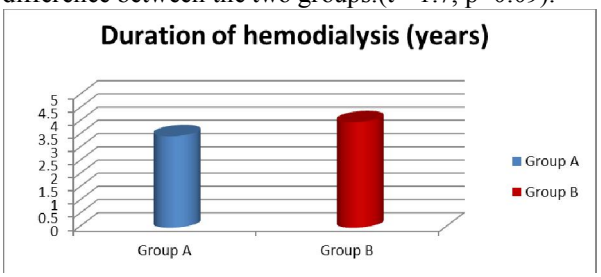

Figure (3): shows comparison between group A and group B a regard Duration of hemodialysis, there was **no significant** difference between the two groups.(t=-0.95, p=0.34).

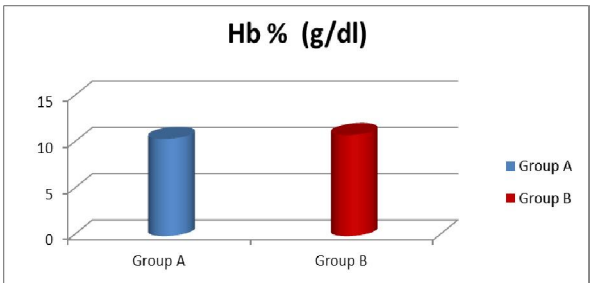

Figure (4): shows comparison between group A and group B a regard Hb%, there was no significant difference between the two groups.(t=-1.16, p=0.2).

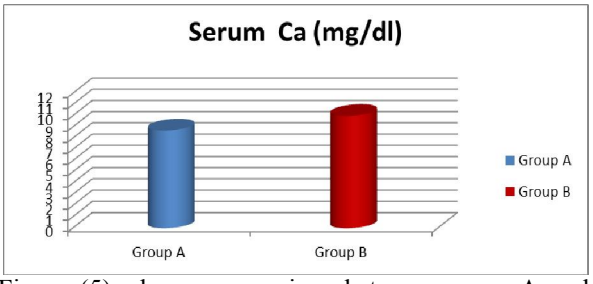

Figure (5): shows comparison between group A and group B a regard serum calcium, there was a highly significant difference between the two groups.(t=-4.64, p=<0.01).

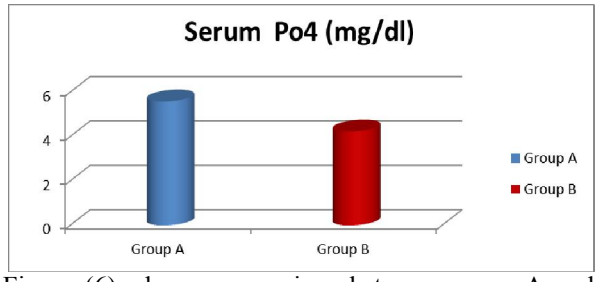

Figure (6): shows comparison between group A and group B a regard serum phosphorus. there was a significant difference between the two groups.(t=-3.79, p=<0.01).

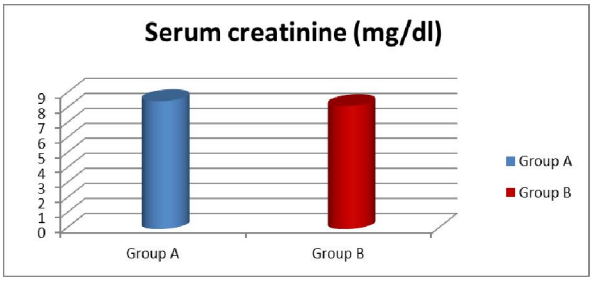

Figure (7): shows comparison between group A and group B a regard serum creatinine, there was no significant difference between the two groups.(t=-0.55, p=0.58).

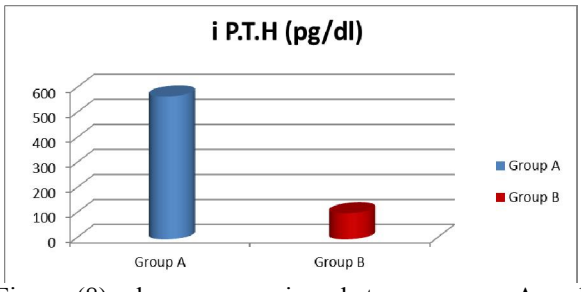

Figure (8): shows comparison between group A and group B a regard IPTH. **there was a highly significant difference** between the two groups. (t=4.39, p=<0.01).

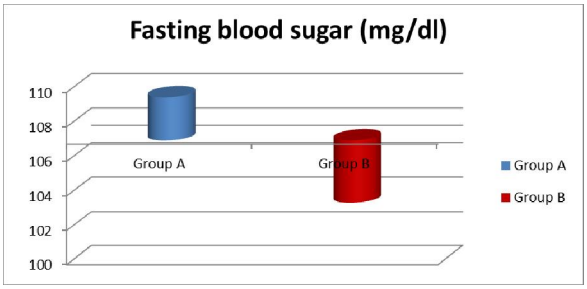

Figure (9): shows comparison between group A and group B a regard fasting blood sugar, there **was no significant difference** between the two groups.(t=-1.23, p 0.2).

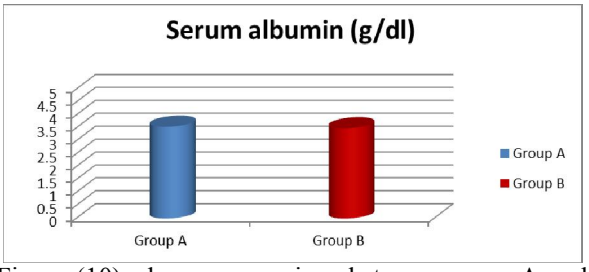

Figure (10): shows comparison between group A and group B as regard serum albumin. **there was no significant difference** between the two groups.(t=0.24, p=0.1).

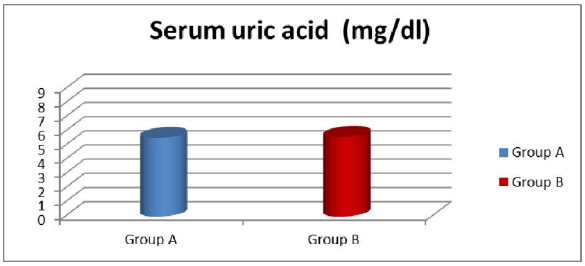

Figure (11): shows comparison between group A and group B a regard serum uric acid. there was **no significant difference** between the two groups.(t=-0.31, p=0.75).

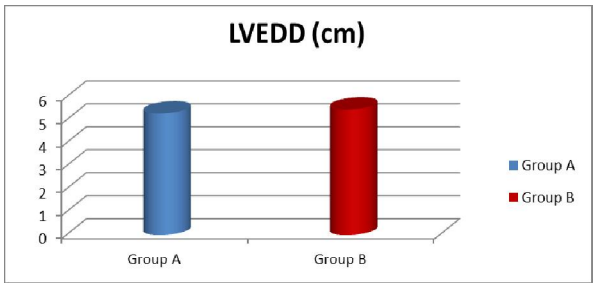

Figure (12): shows comparison between group A and group B a regard LVEDD. There **was no significant difference** between the two groups.(t=-1.17, p=0.24).

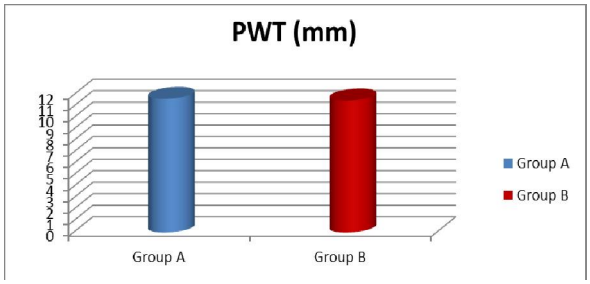

Figure (13): shows comparison between group A and group B a regard PWT. There was **no significant difference** between the two groups.(t=-1.16, p=0.04).

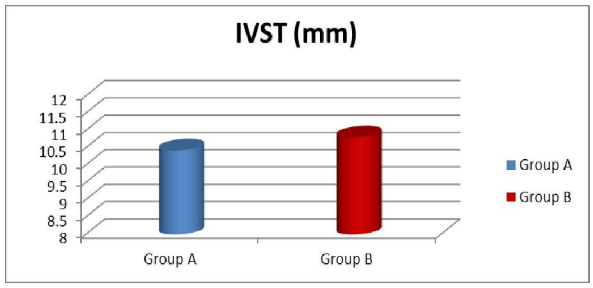

Figure (14): shows comparison between group A and group B a regard IVST. There was a significant difference between the two groups.(t=-2.58, p=0.01).

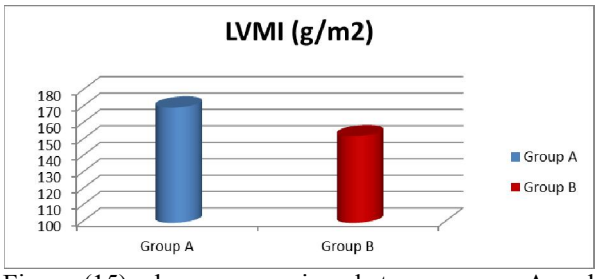

Figure (15): shows comparison between group A and group B a regard LVMI. There was a **highly significant difference** between the two groups.(t=5.92, p=<0.01).

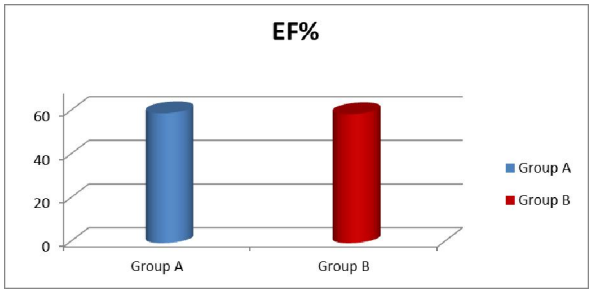

Figure (16): shows comparison between group A and group B a regard EF. There was **no significant difference** between the two groups.(t=0.33, p=0.73).

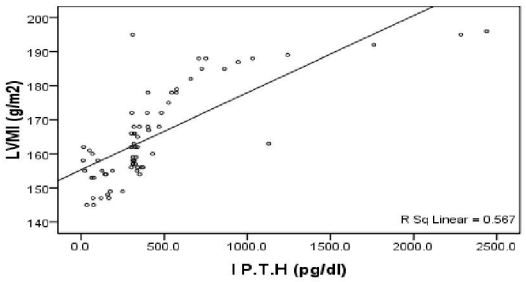

Figure (17): show correlation between IPTH and LVMI. There was a highly significant positive correlation between the two parameters (r = 0.75, p = <0.01).

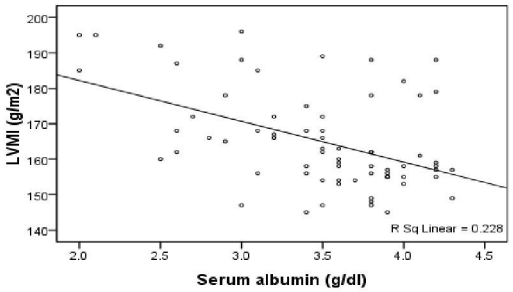

Figure (18): show correlation between serum albumin and LVMI there was non significant correlation between the two parameters (r = -0.3, p = 0.06).

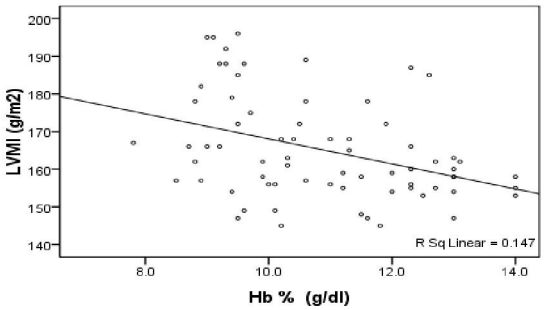

Figure (19): show correlation between Hb and LVMI there was non significant correlation between the two parameters (r = -0.01, p = 0.06).

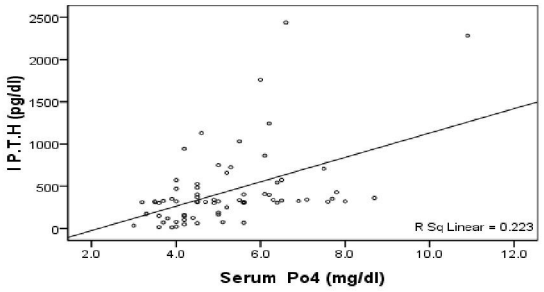

Figure (20): show correlation between serum PO4 and IP.T.H there was a highly significant positive correlation between the two parameters (r = 0.47, p = <0.01).

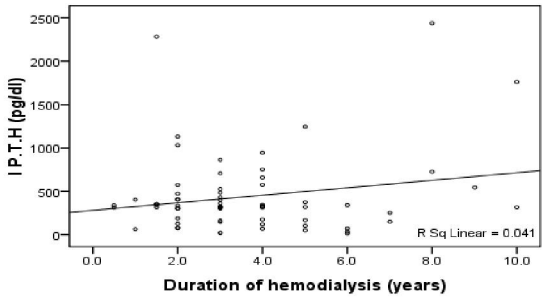

Figure (21): show correlation between duration of hemodialysis and IP.T.H there was a no significant difference between the two parameters (r = 0.20, p = 0.09).

4. Discussion

Several studies suggested that PTH not only a biomarker of vitamin D status but also an independent cardiovascular risk factor that contributes to the progression of CVD. Excessive PTH secretion may be due to problems in the parathyroid glands themselves or may occur in response to low calcium concentrations, due to vitamin D or calcium deficiency or reduced kidney function **(Kestenbaum *et al.*, 2011).**

In addition to traditionally known target organs, PTH is of interest for its potential impact on cardiovascular disease (CVD) risk. Observational studies have demonstrated that chronic PTH elevation is linked to hypertension, cardiac hypertrophy, and myocardial dysfunction **(Walker *et al.*, 2010).** Furthermore, PTH receptors are present in the myocardium and exert hypertrophic effects on cardiomyocytes **(Potthoff *et al.*, 2011)**

Taken together, these associations suggest possible mechanisms whereby elevated PTH concentrations maybe involved in pathological processes that lead to CVD **(Adriana *et al.*, 2012).**

Established risk factors insufficiently explain CVD occurrence. Therefore, it is important to elucidate possible associations of PTH with the development of premature cardiac diseases and CVD risk factors in chronic kidney disease patients. **(Wang *et al.*, 2006).**

Cardiac hypertrophy is major complication in ESRD patients. **(Foley *et al.*, 1995)** reported that the presence of left ventricular hypertrophy in 74% of patients starting dialysis therapy. Another cross-sectional study observed an increase in left ventricular mass even at the earliest stages of renal disease, with eccentric left ventricular hypertrophy being the predominant pattern**.** With progressive decline in renal function, a progressive increase in left ventricular mass was observed, with more than 80% of patients on renal replacement therapy concentric left ventricular hypertrophy being the predominant pattern. In addition, the prevalence of diastolic dysfunction increased in parallel with changes in left ventricular mass **(Angela *et al.*, 2006).**

Our study was conducted on 70 patients with ESRD on HD to evaluate the impact of high serum levels of intact Parathormone (i PTH) on cardiac dysfunctions in hemodialysis patients. They were divided into two groups, the first group included 50 patients on regular HD, their mean duration of HD was 3.5 ± 2.2, their serum i.PTH level was 570.8 ± 471.9. The second group included 20 patients on regular HD, their mean duration of HD was 4.0 ± 1.8, and their serum i.PTH level was 103.8 ± 65.3. The present study revealed that:

- There were insignificant difference between the two groups as regard the age and sex distribution.
- There was no statistically significant differences between the two groups asregard the duration of hemodialysis as well as left ventricular end diastolic diameter and these results was in agreement with results of **Rudhani et al 2010** who found that Left ventricular diastolic dysfunction has no correlation with the duration of HD **(Rudhani et al 2010).**
- **In** our study, there was no statistically significant difference between the two groups as regard Hb, FBS, serum uric acid, serum albumin and serum creatinine as we exclude all factors that may contribute in a change in cardiac mass or function to study the effect of increment of parathormone hormone as a single factor for cardiac dysfunctions or left ventricular hypertrophy.
- In our study there was a highly statistically significant difference between two groups as regard left ventricular mass index (LVMI) that is in agreement with **Ballegooijen *et al.*,** and **Adriana *et al.* (2012).**

In the our Study, the associations between PTH and echocardiographic outcomes were studied. Among 70 included patients, higher PTH was significantly associated with higher LV mass**.** The explanation of these results could be elevated iPTH levels acts on cardiomyocytes by binding to the PTH/PTHrP

receptor, which induces a rise in the intracellular levels of calcium. Increased calcium levels activate protein kinase C and mediate hypertrophic as well as metabolic effects. (**Smogorzewski *et al.,* 2006).**

Many published experiments report that iPTH contributes to cardiac fibroblast activation and the fibrosis of intermyocardiocytes, which is a prerequisite of diastolic dysfunction. In addition, higher levels of calcium due to hyperparathyroidism have been shown to induce arrhythmia.**(McCarty *et al.,* 2009).**

Similar results were reported by **Adriana *et al.* (2012)** who concluded that higher i-PTH was significantly associated with higher LV mass in both genders**.**

Tentori ***et al.,* 2008** concluded that there was a strong association between i P.T.H and the risk of left ventricular hypertrophy and all cause cardiovascular mortality in hemodialysis patients. Our results showed that patients with i P.T.H levels (>300pg/dl) had a higher risk of cardiac dysfunctions than those who were with lower baseline i P.T.H levels (<300pg/dl) (*P* = 0.01), so, we suggest that secondary hyperparathyroidism may be a strong predictor of left ventricular hypertrophy and dysfunction in HD patients.

In the present study, there were highly statistically significant between the two groups as regard hyperphosphataemia and left ventricular hypertrophy, these results go in agreement with (**Strozecki *et al.,* 2002**) who reported that 70% of dialysis patients have LVH and those patients with higher serum phosphate concentrations associate with LVH in dialysis patients, independent of calcification. The explanation of these results may be due to one of two candidate mechanisms by which serum phosphorus could contribute to cardiovascular disease are vascular calcification and modulation of circulating serum hormones, specifically PTH, FGF-23, and calcitriol.

Two types of arterial calcification:

- Intimal-within atheroscalerotic plaques. Associated with stenotic lesions and cardiovascular events.
- Medial-within the intima media; classically, Monckeberg's calcinosis. This causes vascular stiffness leading to elevated pulse pressure leading to LVH.
- In addition, cardiac valvular, and other soft tissue, calcification is a frequent finding in advanced chronic kidney disease.

Hyperphosphatemia has been identified as playing a major role in the progression of renal failure and in the generation of secondary hyperparathyroidism and mineral bone disease in renal failure **(Slatopolsky *et al.,* 2002).**

Further observational data have also shown a significant association of hyperphosphatemia with increased mortality among patients who have end-stage kidney disease onhemodialysis **(Block *et al.,* 1998; Owen & Lowrie, 1998; Ganesh *et al.,* 2001).** Moreover, elevated serum phosphorus has been associated with an increased risk for cardiovascular morbidity and hospitalization. Our results showed that patients with serum phosphorus levels (>6.5 mg/dl) had a higher risk of cardiac dysfunction and vascular calcification than those who were with lower baseline serum phosphorus levels (<6.5 mg/dl) (P <0.01)), as evidenced by echocardiographic findings suggest highly significant correlation between hyperphosphatemia, IPTH and LVMI so we suggest that Hyperphosphatemia is a strong predictor of cardiac dysfunction and vascular calcification in HD patients.

LVH is a powerful predictor of cardiovascular outcomes in hemodialysis patients and is a multifactorial process with many causes in ESRD, such as anemia, pressure loading through hypertension, and reduced vascular compliance **(London 2003). Steven *et al.,* 2006** demonstrated that even with aggressive therapy for BP control (average systolic BP of 145 mmHg) and anemia, conventional hemodialysis is associated with significant LVH (mean LVMI of 155 g/m2) **(Steven *et al.,* 2006),** whereas short daily dialysis is associated with a significant reduction in LVMI (154 to 108 g/m2) and control of serum phosphorus (mean serum phosphorus of 4.2 mg/dl) correlates with this reduction in LVMI. There findings suggest a novel mechanism for the deleterious effect of hyperphosphatemia on cardiovascular outcomes among hemodialysis patients **(Ayus *et al.,* 2005), Steven *et al.,* 2006),** showed that improvement in serum phosphorus was associated with improvement in LVH. Another studies have noted an association of altered mineral metabolism and LVH and cardiac dysfunction.(**Marchais *et al.* 1999**) noted increased diastolic and mean arterial pressures, higher cardiac index, higher heart rate, and increased stroke index in hyperphosphatemic *versus* normophosphatemic patients. Strozecki *et al.* 2001 showed that poor control of serum phosphorus and calcium-phosphorus product was associated with increased LVM report by **Galetta *et al, 2005*** using echocardiography and tissue Doppler imaging, showed that higher plasma phosphate and calcium-phosphate products were associated with signs of diastolic dysfunction in a cross-sectional study. **Hayashi *et al. 2004*** performed echocardiography before and after hemodialysis in 13 conventional hemodialysis patients and demonstrated that elevated serum phosphorus and calcium phosphorus product are associated with decreased is volumetric contraction velocity and peak systolic velocities, suggesting that poor mineral metabolism can affect systolic function. Furthermore, they showed that after a single

hemodialysis session, these indices improved. These studies suggest that poor mineral metabolism has adverse consequences on LV geometry and function and that dialysis improves LV function, particularly in those with poor control of mineral metabolism.

Also in our results there was positive correlation between increase the level of intact parathyroid hormone and hyperphosphatemia in hemodialysis patients these results go in agreement with **Drueke (2007)** who reported that patients with anuric end-stage renal disease who have increase in serum phosphate may be not only from decreased phosphate excretion but also from an increase in bone release of phosphate that occurs in response to chronically elevated PTH levels**. Drueke and Lacour (2007)**.

Conclusions

There was a highly significant positive correlation of serum iPTH and left ventricular mass index, in HD patients.

- Serum iPTH >300pg/ml was a predictor in part of cause cardiovascular dysfunction in HD patients and LVH.

-Serum calcium was a predictor in part of cause cardiovascular dysfunction in HD patients.

-Serum phosphorus>6.5mg/dl a predictor of cardiovascular dysfunction in HD patients.

-There was a highly significant positive correlation of serum phosphorus and left ventricular mass index.

Recommendations

-We recommend application of this study on large number of dialysis patients as a multicentres research to confirm our results.

-Secondary hyperparathyroidism surrogate markers are important player in development of cardiac dysfunctions and its complications.

-Hoping for cut off value of high intact Parathormone hormone as amaker of cardiovascular dysfunctions in hemodialysis patients.

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