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RE CARTPEN	EVA CHF 50 E	LUATION OF 2-D ECHO FINDINGS IN RONIC KIDNEY DISEASE :A CASE STUDY OF ND STAGE RENAL DISEASE PATIENTS	KEY WORDS:		
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INTRODUCTION		BESILT			

Chronic kidney disease (CKD) is a major public health problem worldwide with increase in incidence and prevalence. Diabetes and hypertension are the leading cause of CKD worldwide, whereas hypertension is a cause as well as effect of CKD. CKD is a risk factor for cardiovascular events and complications which increase as CKD progress to ESRD [3]. Cardiovascular mortality is 10-20 times more common in ESRD patients on renal replacement therapy as compared to general population. One of the major structural cardiac abnormalities in CKD patients is left ventricular hypertrophy (LVH) and is associated with increased risk for cardiac ischemia, congestive heart failure, as well as a very strong independent predictor for cardiovascular mortality [4]. Majority patients with CKD die due to cardiovascular events before reaching ESRD due to risk factors [5]. Anemia and hypertension are most consistent with heart failure that causes 2/3rd death of all dialysis patients. ESRD patients do have myriads of structural and functional cardiac abnormalities which include LVH, depressed LV function, regional wall motion abnormality, pericardial effusion and valvular calcification.

METHODS AND MATERIALS

50 ESRD patients irrespective of underlying etiology who were admitted in G.K.GENERAL HOSPITAL ADANI BHUJ and were on maintenance hemodialysis for at least 3 months were included in this study. A person was labelled ESRD is his or her GFR was less than 15ml/1.7m2 as per modified diet in renal disease (MDRD) formula and who were on MHD. Patient with obvious clinical evidence of coronary artery disease, valvular heart disease, pericardial effusion, rheumatic heart disease, congenital heart disease and primary cardiomyopathy were excluded from the study. All patients were clinically evaluated thoroughly and subjected for complete blood count, renal function test, serum calcium, phosphorous and 2-D echo. M mode recording perpendicular to the long axis of and through the centre of the left ventricle at the papillary muscle level was taken as standard measure of systolic and diastolic wall thickness and chamber dimensions. Left ventricular ejection fraction (LVEF) and fractional shortening (FS) were taken as a measure of left ventricle systolic dysfunction and ejection fraction <55% was considered as systolic dysfunction.

Diastolic dysfunction was determined by measuring E/A ratio by special Doppler inflow velocity (E is peak early diastole velocity and A is peak atrial filling velocity of left ventricle across mitral valve). E/A ratio less than 0.75 and more than more than 1.8 was considered as diastolic dysfunction. LVH was diagnosed when inter ventricular septum thickness or left ventricular posterior wall thickness was > 12 mm. Hypertension was defined as BP >= 140/90 mm hg in right arm supine position and anaemia was diagnoses with Hb <13 g/dl in male and <12 g/dl in female.

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Out of 50 ESRD	patients	.echocardio	ograph	v revealed
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Findings	No. of Patients	Percentage
Left Ventricular Dilatation and	26	52%
Diastolic Dysfunction		
Left Ventricular Hypertrophy	24	48%
Systolic Dysfunction	14	28%
Pericardial Effusion	9	18%
RWMA Regional Wall Motion	4	8
Abnormality		

In a sub group of 35 patients with Hb <10g%, LVH was present in 24 patients (70%) vs 3 out of 15 patients (20%) in patients group with Hb $> 10 \, \text{g\%}$.

Hypertensive patients were 38 of 50 ESRD patients, 16 out of 38 had higher prevalence of LVH (50%).

Systolic dysfunction and RWMA was absent in normotensive group.

DISCUSSION

Cardiovascular disease is the major cause of death in patients with ESRD. The detection of echocardiographic abnormalities with sub clinical cardiac disease is considered to be an important step for characterization of individual risk for heart failure in the general population as well as in patients of ESRD [5].

The common cardiac complications in CKD patients are LVH, systolic dysfunction and diastolic dysfunction due to myocardial fibrosis, myocardial calcification and changes in the vasculature structure, leading to adverse cardiovascular events.

In our study in G. K. GENERAL Hospital, out of 50 ESRD patients, LVH was present in (48%) patients, systolic dysfunction in 10 (28%) patients and diastolic dysfunction in 26 (52%) patients. Echocardiographic findings in other studies also confirmed presence of systolic dysfunction in 20% and diastolic dysfunction in 50% patients [8,9].

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Agarwal S, et al. Had observed diastolic dysfunction in 53.2% and systolic dysfunction in 30% patients with severe CKD (S. Cr >6 mg%) [10]. In a study conducted by Laddha M, et al. in 2014, reported LVH in 74%, systolic dysfunction in 24.3%, diastolic dysfunction in 61.4% and pericardial effusion in 14.34% ESRD patients on MHD [11]. Zoccali C, et al. had reported incidence of LVH and systolic dysfunction in 77% and 22% patients respectively in ESRD patients on MHD [12]. Valvular calcifications are 4 times more common in dialysis patients compared to general group [13]. None of our patients had valvular calcifications probably because of small study population. Levin, et al. also reported association between elevated systolic blood pressure and low Hb levels with LVH in pre dialysis patients [15, 16]. Anemia is a strong predictor of development of LVH causing mortality and morbidity in ESRD [6]. Data et al had observed severity of anemia correlated to LVH in patients with CKD [17]. In ESRD patients on hemodialysis it has been observed that decrease in Hb levels of 1g% increased LVH by 50% and mortality by 18-25% [18].

CONCLUSION

- Cardiac structural as well as functional abnormalities are common in patients with ESRD, more so in those with hypertension and anaemia.
- LVH is the commonest cardiac abnormality in ESRD patients, followed by diastolic dysfunction.
- Both conditions are more marked in hypertensive patients and anaemic patients.
- LVH has got prognostic implications, because this group of ESRD patients have propensity of diastolic dysfunction or sudden cardiac death.
- Echocardiography is cost effective non invasive diagnostic test which can detect early changes in cardiac parameters. This is important for risk stratification and early preventive measures. Thus echocardiographic screening of asymptomatic ESRD patients, especially anaemic and hypertensive help us to check progress and prognosis of the disease.

REFERENCES

- Santulli G, Trimarco B, Iaccarino G. G- protein-coupled receptor kinase 2 and hypertension: molecular insights and pathophysiological mechanisms. High blood pressure cardiovasc Prev., 2013; 20:5-12.
- Santulli G, Cipolleta E, Sooriento D, Del Giudice C, Anastasio A, et al. CaMK4 Gene Deletion Induces Hypertension. J Am Heart Assoc., 2012; 1:e001081.
- Agodoaly Y, Egger PW. Renal replacement therapy in the United States: data from the United states Renal Data System. Am J Kidney Dis., 1995;25:119-133.
- Parfrey PS, Foley RN, Harnett JD, Kent GM, Murray DC, et al. Outcome and risk factors for left ventricular disorders in chronic uremia. Nephrol Dial Transplant, 1996; 11:12277-1285.
- Foley RN, Parfrey PS, Harnett JD, Kent GM, Martin CJ, et al. Clinical and echocardiographic disease in patients. starting end stage renal disease therapy.Kidney Int., 1995;47:1277-1285.
- Foley RN, Parfey PS, Kent GM, Murray DC, et al. The impact of anaemia on cardiomyopathy, morbidity and mortality in ESRD. Am J Kidney Dis., 1996;28: 53-61.
- Noor ul amin, Raja Thir Mahmood, M Javid Asad, Mudassar Zafar, Asad Mehmood Raja. Evaluating urea and creatinine levels in chronic renal failure pre and post dialysis: A prospective study. JCvD, 2014 in press.
- McMurray JV, Mc Donagh TA, Davie AP, Cleland JG, Francis CM, et al. Should we screen for asymptomatic left ventricular dysfunction to prevent heart failure? Eur Heart J., 1998; 19:842-846.
- Kunz K, Dimitrov Y, Muller S, Chantrel F, Hannedouche T. Uremic cardiomyopathy.NephrolDialTransplant., 1998;13 Suppl 4:39-43.
- S Agarwal, P Dandri, OP Karla, S Rajpal. Echocardiographic assessment of cardiac dysfunction in patients of chronic renal failure. JIACM, 2003; 4: 296-303
- Laddha M, V Sachdeva, PM Diggikar, PK sapathy, AL Kakrani. Echocardiographic assessment of cardiac dysfunction in patients of ESRD on hemodialysis. JAPI, 2014; 62: 28-32. 12. Zoccali C, Benedetto FA, Mallamaci F, Tripepi G, Giacone G, et al. Prognostic value of echocardiographic indications of left ventricular systolic function in asymptomatic dialysis patients. JAm Soc Nephrol., 2004; 15: 1029-1037.
- Leskinen Y, Paana T, Saha H, Groundstroem K, Lehtimaki, et al. Valvular calcification and its relationship to atherosclerosis in chronic kidney disease. JHeartValve Dis., 2009;18:429-438.
- 14. Parfey PS, Foley RN. The clinical epidemiology of cardiac disease in chronic renal failure. J Am Soc Nephrol., 1999; 10:1606-1615.
- Levin A, Thomson CR, Ethier J, Carlisle EJ, Tobe S, et al. Left ventricular mass index increase in early renal disease: impact of decline in haemoglobin. Am J Kidney Dis., 1999; 34: 125-134.
- Levin A, Singer J, Thomson CR, Ross H, Lewis M. Prevalent left ventricular hypertrophy in predialysis polulation: identifying opportunities for intervention.Am JKidney Dis., 1996;27:347-354.
- 17. Datta S, Abraham G, Mathew M, Somasundaram H, Murlidhar TR, et al.

Correlation of anemia, secondary hyperparathyroidism with left ventricular hypertrophy in chronic kidney disease patients. J Assoc Physicians India, 2006;54:699-703.

 Harnett JD, Kent GM, Foley RN, Parfrey PS. Cardiac function and hematocrit level. Am JKidney Dis., 1995;25:S3-7.19. Pecoits Filho R,

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