Original Research Article

Evaluation of 2-D echo findings in chronic kidney disease: Case study of 35 end stage renal disease patients

Jas Pal Dhamija[1], Nishant Saxena[2*], Shubhi Saxena[3]

[1]Associate Professor, Department of General Medicine, Mahatma Gandhi Hospital and Medical College, Sitapura, Jaipur, Rajasthan, India
[2]Resident, M.D. General Medicine, Mahatma Gandhi Hospital and Medical College, Sitapura, Jaipur, Rajasthan, India
[3]Resident, M.D. Pathology, Mahatma Gandhi Hospital and Medical College, Sitapura, Jaipur, Rajasthan, India
[*]Corresponding author email: **nsaxena014@gmail.com**

International Archives of Integrated Medicine, Vol. 3, Issue 9, September, 2016.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 12-08-2016 **Accepted on:** 23-08-2016
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: Dhamija JP, Saxena N, Saxena S. Evaluation of 2-D echo findings in chronic kidney disease: Case study of 35 end stage renal disease patients. IAIM, 2016; 3(9): 61-65.

Abstract

Background: 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.

Objectives: To evaluate and analyze the echocardiographic changes in the end stage renal disease patients on maintenance hemodialysis.

Materials and methods: End stage renal disease (ESRD) patients who were on maintenance Haemodialysis for at least 3 months, in MG hospital were included in the study. We performed 2D echocardiography in 35 ESRD patients during inter-dialytic period. Patients with clinical evidence of coronary artery disease, valvular heart disease, congenital heart disease and pericardial effusion were excluded from the study.

Results: Out of 35 ESRD patients, echocardiography revealed LV dilatation and diastolic dysfunction in 18 patients (51.2%) , LV hypertrophy in 17 patients (48%), systolic dysfunction and pericardial effusion in 10 patients (28.57%) and 6 patients (17.14%) respectively. RWMA was present in 3 patients (8.5%) and no valvular calcification was seen in any patient. In a sub group of 21 patients with Hb <10g%, LVH was present in 15 patients (71.42%) vs 2 out of 14 patients (14.28%) in patients group with Hb >10 g%. Hypertensive patients were 27 of 35 ESRD patients, 13 out of 27 had

higher prevalence of LVH (51.85%). Systolic dysfunction and RWMA was absent in normotensive group.

Conclusion: LV diastolic dysfunction and hypertrophy were most common echocardiographic findings. There was statistically significant correlation between anaemia and presence of LVH and positive correlation between presence of hypertension and LVH.

Key words

ESRD (end stage renal disease), MHD (maintenance haemodialysis), LVH (left ventricular hypertrophy), Diastolic dysfunction.

Introduction

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. Recent genetic background of hypertension is gaining importance in pathophysiology of hypertension. G protein coupled and calcium dependent kinase is responsible for control of blood pressure [1]. Even lots of mutation can cause changes in the receptors, which in turn raise blood pressure [2]. 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 both traditional and non traditional risk factors [5]. Whether CV events differ in patients with and without CKD is poorly defined and also whether differences in cardiovascular diseases in CKD patients suggest preventive or therapeutic strategies unique to this population is unclear.

Anaemia 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.

Hemodialysis is one form of renal replacement therapy, during which metabolic waste products including creatinine, urea, excess water and salts are removed. It also maintains the nutritional status, mental and physical well being if done on a regular basis. Noor ul amin, et al. had shown that hemodialysis is an effective means of removing metabolic waste product [7]. In this study we evaluated the cardiovascular abnormalities by performing 2D-echocardiography in patients of CKD on maintenance hemodialysis (MHD).

Materials and methods

35 ESRD patients irrespective of underlying etiology who were admitted in MGMCH, Jaipur 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/1.7m^2 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 cholesterol, 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.

Statistical analysis was done by using chi square test by SSPS software version 15. A 'p value' less than 0.05 was considered significant.

Results

This study included 35 patients of ESRD on MHD. Clinical examination suggested laboratory test and echocardiography was performed in every patient.

Out of 35 patients, 27 (77%) were male and 8 (23%) female. Maximum patients were in age group between 41-50 yrs (34%). Mean age of the patients was 45.5$\pm$23.5.

Hypertension was present in 27 (77.14%) mainly in age group more than 40 years.

Most common cause of ESRD was diabetes 15 (42%), followed by hypertension 10 (28%), chronic glomerulonephritis (CGN) 5 (14%), and chronic tubule-interstitial nephritis (CTN) in 3(8.5%) patients respectively. 2 patients could not be diagnosed.

Anaemia was observed in all patients and haemoglobin of less than 10 g% was seen in 21 patients (60%). Echo-cardiographic findings were studied and analyzed in details. Echo parameters analyzed in our study were left ventricular internal diameter in diastole (LVIDd) and left ventricular internal diameter in systole (LVIIDs), inter ventricular septal diameter in systole, E/A ratio, fractional shortening, ejection fraction and size of left atrium.

On comparing the echocardiographic findings in patients with Hb <10 g%, statistically significant number of 15 out of 21 patients had LVH.

Similarly majority of the patients with LVH had hypertension (51.85%) compared to normotensives (8.58%), although it was not statistically significant.

RWMA was present in 3 (14.28%) patients with Hb <10 g% but absent in patients with Hb >10 g% and also 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 MG hospital, out of 35 ESRD patients, LVH was present in17 (48%) patients, systolic dysfunction in 10 (28%) patients and diastolic dysfunction in 18 (51.42%) patients. Echocardiographic findings in other studies also confirmed presence of systolic dysfunction in 20% and diastolic dysfunction in 50% patients [8, 9]. 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]. Out of 35 ESRD patients, we observed pericardial effusion in 6 (17.14%) patients and RWMA in 3 (8.5%) patients respectively. 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. Majority patients had hypertension 27 (77.14%). In hypertensive group LVH was present in 13 (51%) vs 1 patient (8.57%) in normotensive group of 8 patients. In sub-group of 21 patients with Hb level <10 g%, LVH was seen in 15 patients (71.42%) compared to 2 (14.28%) patients out of 14 patients with Hb >10 g%. Patric, et al. had shown that rise in mean arterial pressure was associated with increase in incidence of LVH in ESRD patients on maintenance hemodialysis [14]. 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 [19].

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.

Limitations

- Small number of patients.
- Impact of hyperlipidemia, secondary hyper parathyroidism, homocysteine levels and markers of inflammation and duration of MHD were not studied in population group.
- Lack of follow up.

References

1. 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.
2. 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.
3. 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.
4. 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.
5. 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.

6. 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.
7. 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.
8. 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.
9. Kunz K, Dimitrov Y, Muller S, Chantrel F, Hannedouche T. Uremic cardiomyopathy. Nephrol Dial Transplant., 1998; 13 Suppl 4: 39-43.
10. S Agarwal, P Dandri, OP Karla, S Rajpal. Echocardiographic assessment of cardiac dysfunction in patients of chronic renal failure. JIACM, 2003; 4: 296-303.
11. 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. J Am Soc Nephrol., 2004; 15: 1029-1037.
13. Leskinen Y, Paana T, Saha H, Groundstroem K, Lehtimaki, et al. Valvular calcification and its relationship to atherosclerosis in chronic kidney disease. J Heart Valve 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.
15. 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.
16. Levin A, Singer J, Thomson CR, Ross H, Lewis M. Prevalent left ventricular hypertrophy in predialysis polulation: identifying opportunities for intervention. Am J Kidney 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.
18. Harnett JD, Kent GM, Foley RN, Parfrey PS. Cardiac function and hematocrit level. Am J Kidney Dis., 1995; 25: S3-7.
19. Pecoits Filho R, Barberato SH. Echocardiographiy in chronic kidney disease: diagnostic and prognostic implications. Nephron Clin Pract., 2010; 114(4): c242-7.