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## Study on left ventricular hypertrophy in chronic kidney disease patients attending tertiary care hospital in Central India

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### ABSTRACT

**BACKGROUND:** Chronic kidney disease (CKD) is associated with increased cardiovascular risk. LVH is highly prevalent in CKD even in early stages, as compared to general non-selected population. This is mainly due to the multifactorial pathogenesis of left ventricular hypertrophy (LVH) in renal patients where both haemodynamic and non-haemodynamic stimuli synergically act inducing either an increase in left ventricular mass or an LV dilation. The presence of LVH in patients with CKD is associated with worsening cardiovascular outcomes.

**OBJECTIVES:** To find the factors which correlate with left ventricular hypertrophy in chronic kidney disease patients.

**MATERIALS AND METHODS:** This study was done in the Department of Medicine of GMC & Hamidia hospital Bhopal. It was a hospital based prospective observational study in which 50 diagnosed CKD patients (stage 3 and 4) were studied. All the patients fulfilling the inclusion criteria were subjected to detailed clinical history, systemic examination, routine investigation, SUA measurement and echocardiography for determination of LVH. The results thus obtained from clinical/laboratory examinations were analysed with the ECHO findings to determine the correlation with progression in CKD. the categorical data was compared by using chi-square test. P value of < 0.05 is considered as significant.

**RESULTS:** The study had an mean age of  $54 \pm 13.49$  years. Mean Ejection fraction of the patients was 54.13%. The mean baseline Systolic Blood pressure recorded was  $150.92 \pm 24.82$  mmHg. with a range from 140 to 165 mmhg and Diastolic blood pressures recorded was  $103.92 \pm 19.97$  mmHg. There were 27% of the patients who had left ventricular hypertrophy at baseline. Around 6 patients had RWMA and 16 patients had dilated cardiomyopathy. ECG revealed that, around 27% of the sample had LVH. At baseline, the patients LVH was determined to be  $111.09 \pm 29.41$ . Majority (58.0%) of the sample have reduced ejection fraction

**CONCLUSION:** The results of this study conclude that more clinical trials are needed to assess guidelines for treating CKD related hypertrophic cardiomyopathy. Study also showed a higher percentage of hypertension patients were found in patients with a high LVH and uric acid, which was accompanied by a high LVH. Patients with hypertension, raised serum creatinine, high LVH, and high uric acid exhibited a statistically significant higher incidence of decline in renal function.

**Keywords:** Left ventricular hypertrophy, CKD, Tertiary care hospital, LVH

### INTRODUCTION

Burden of chronic disease throughout the world is steadily increasing. Cardiovascular disease (CVD) and CKD frequently coexist and represent a major challenge in

today's medicine. The prevalence of chronic kidney disease, is showing an upward trend in the elderly.<sup>[1,2]</sup> CKD is associated with end-stage renal disease (ESRD) and increases morbidity, mortality and cost of the health care system.<sup>[3-5]</sup> CKD is defined as either kidney damage,

estimated by using such markers as albuminuria, or estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m<sup>2</sup>.<sup>[3]</sup> CKD is emerging to be an important chronic disease globally<sup>[6]</sup> the reason behind it is rapidly increasing incidence of diabetes<sup>[7]</sup> and hypertension globally<sup>[8]</sup> In India, whose population is over 1 billion, the rising CKD incidence may lead to major problems in context with both healthcare and the economy. It has been estimated that the age-adjusted incidence rate of ESRD in India to be 229 per million population (pmp)<sup>[9]</sup>, and >100,000 new patients registered in renal replacement programs yearly in India.<sup>[10]</sup> In CKD patients, hypertension is closely associated with left ventricular hypertrophy. LVH is caused by chronically increased workload on the heart<sup>[11]</sup>, by hypertension and is a strong predictor of mortality in CKD patients<sup>[12]</sup>. Heart disease is a major risk factor of kidney damage, the prognosis of which depends on not only blood pressure level but also on associated factors such as LVH. The latter frequently develops among hypertensive patients or CVDs. In Several previous echocardiography studies have indicated that CKD is independently associated with the presence of LVH, suggesting that renal function is related to LVH. Furthermore, the results of experimental and animal studies also support the association between CKD and LVH<sup>[13-15]</sup> CKD considerably increases the risk of CVD. both CKD and CVD patients share same risk factors, abnormal heart function and structure are the principal predictors of adverse outcomes<sup>[16]</sup> LVH is more common in CKD patients and is linked to increased mortality and a poor prognosis.<sup>[17-19]</sup> CKD is a leading cause of morbidity and mortality, particularly in the middle-aged and geriatric populations, has been the subject of extensive research for several decades. The factors affecting the initiation as well as progression of CKD to ESRD has been researched by many independent clinicians around the globe. SUA and LVD are two of the many probable components that have been studied in recent years. Left ventricular hypertrophy (LVH) is a cardiovascular complication highly prevalent in patients with chronic kidney disease (CKD) and end-stage renal disease. LVH in CKD patients has generally a negative prognostic value, because it represents an independent risk factor for the development of arrhythmias, sudden death, heart failure and ischemic heart disease. Researches targeting the role of SUA as well as LVH is sparse in this part of the country and hence the study was planned to

assess the correlation of LV hypertrophy in CKD patients to provide insight to factors responsible for CKD progression and the possible interferences to hold the progression and for the better clinical management, hence reducing the death rate and disease burden in CKD patients.

**OBJECTIVES:** To find the factors which correlate with left ventricular hypertrophy in chronic kidney disease patients

## MATERIAL AND METHODS

Study was conducted in the Department of Medicine, Gandhi Medical College and associated Hamidia Hospital Bhopal during June 2020 to May 2021. It was an Hospital based Prospective observational study conducted on 50 consecutive CKD patients grade 3 and grade 4 coming to the OPD, irrespective of gender and age. After approval of the study protocol by the Institutional Ethics Committee, written consent was taken. The study was conducted in Department of Medicine, Gandhi medical college & Hamidia hospital Bhopal on already diagnosed patients of CKD. Patients demographic details along with phone number and address was be noted down on admission. Patient was called up on the phone for follow up. At follow up patients would be assessed clinically and radiologically for functional activity. All the patients fulfilling the inclusion criteria were subjected to detailed clinical history, systemic examination, routine investigation, SUA measurement and echocardiography for determination of LV hypertrophy. The results thus obtained from clinical/laboratory examinations were analysed with the ECHO findings to determine the correlation with progression in CKD.

**ETHICAL APPROVAL:** The study was approved by the Institutional Ethics Committee, Gandhi Medical College, Bhopal, Madhya Pradesh.

## RESULTS

The patients in the study had a mean age of 54±13.49 years. Table No. 1 shows the distribution. Males outnumbered females. In our study, 44% of the patients had more than one year of duration of CKD. There were only 20% of the participants who were suffering from the illness < 6 months. Only 20% of the patients had less than 6months of duration of illness. Sixty six percent of the patients belonged to stage 4 of illness according to KDIGO staging.

**Table 1: Distribution of patients according to baseline characteristics (N=50)**

Variables	Frequency	Percentage
<b>Age (years)</b>		
<40	8	16
41-60	29	58
>60	13	26
Mean (±SD)	54.0 (13.49)	
<b>Gender</b>		
Male	29	58
Female	21	42
<b>Duration of CKD From diagnosis (months)</b>		

1-6	10	20
7-12	18	36
>12	22	44
Mean (±SD)	12.2 (12.7)	
<b>CKD Stage</b>		
3	17	34.0
4	33	66.0

**Table 2: Distribution of patients according to Cardiac parameters**

Cardiac Parameters	Mean(S.D)	Median (IQR)
LVM(gm)	175.02±50.11	165.5(139.5-165.5)
LVMI(gm/m <sup>2</sup> )	111.09±29.41	108.5(88.2-131.0)
EF %	54.13±11.25	58.0(49.7-60.0)
Systolic BP (mmHg)	150.92±24.82	158(140-165)
Diastolic BP (mmHg)	103.92±19.97	107(99-120)

RWMA (%)	6
DCMP (%)	16
Left Ventricular Hypertrophy (%)	27

The table above shows the descriptives of the patient’s cardiac parameters. Mean LVM of the patient was 175.02±50.11 ranging from 82.0 to 276.0 grams. The LVMI calculated at baseline was 111.09±29.41. Mean Ejection fraction of the patients was 54.13%. The mean baseline Systolic Blood pressure recorded was 150.92+24.82mmHg.

with a range from 140 to 165 mmHg and Diastolic blood pressures recorded was 103.92 + 19.97mmHg. there were 27% of the patients who had left ventricular hypertrophy at baseline. Around 6 patients had RWMA and 16 patients had dilated cardiomyopathy.

**Table 3: Distribution of patients according to laboratory parameters (N=50)**

Laboratory parameters		Mean±SD
Blood Investigation	Hemoglobin (gm %)	9.19±2.35
	Total leucocyte count	12857.64±5190.13
Kidney Function Test	Blood Urea (mg/dl)	80.47±31.96
	Serum creatinine (mg/dl)	2.58±0.75
	Serum sodium (mmol/L)	135.61±8.43
	Serum potassium (mmol/L)	4.29±0.85
	Sr Calcium(mg/dl)	8.26±1.410
	Sr Phosphorus(mg/dl)	3.29±1.16
	eGFR	26.97±9.10

**DISCUSSION**

The goal of this study was to see correlation between left ventricular hypertrophy and renal function deterioration in chronic kidney disease patients. The patients in this study had an average age of 54 ±13.49 years. 60% of the patients belonged to the age group of 41-60years. Similar findings was observed in the study by Magdalena Madero et.al [20] (2009) where the mean (SD) age was 52 +12 years. There are many risk factors for LVH in CKD patients. Hypertension, diabetes, anemia, extra-cellular fluid expansion, arterio venous fistulas and abnormalities of calcium phosphate homeostasis are some of the common mechanisms described.[21] Zocalli et al.[22] had shown a 50% mortality risk and more than 85% cardiovascular event risk at 3 years in the patients who had significant increase in left ventricular mass while undergoing dialysis. In our study, we found LVH to increase

with the severity of CKD. In the severe CKD group, 88.63% had LVH. Similar observations were noted by Paoletti et al.,[23] who found a steady increase in the LVH as the renal dysfunction progressed during the predialysis stage of CKD. In their study they found that 70% to 80% of the severe CKD patients had LVH before the initiation of dialysis. A year had passed since the patients were diagnosed with chronic renal disease in 44% of the cases. Twenty percent of the individuals had been sick for less than six months. According to the KDIGO staging system, Sixty-six percent of the patients had advanced CKD (stage 4). Hiromichi Suzuki et al[21] (2014) included 67 patients with CKD stages 3–5 which was the similar findings with this study. The majority of the patients had hypertension, and 36% of them had diabetes. A total of 11 patients had coronary artery disease. Both diabetes mellitus and hypertension were present in 17 of the patients. Similarly in the study by Magdalena Madero et.al[20] (2009) 15% of the patients had

CVD. 417 patients (36.8%) had AMI and the remaining 715 patients (63.2%) had unstable angina pectoris in the study by Zhao Xia Yin et.al [25] (2013).

Systolic hypertension and elevated pulse pressure are strongly associated with LVH in those patients with advanced CKD, suggesting that fluid overload and increased arterial stiffness play a role in LVH even before the start of dialysis therapy<sup>[26]</sup>. The younger age, the lower pulse pressure, and the higher GFR are related to a positive response to LV mass reduction<sup>[27]</sup>. Persistent or progressive LVH is strongly associated with an increase in the risk of mortality and cardiovascular events including sudden cardiac death in ESRD patients<sup>[22]</sup>.

The reduction in the degree of LVH can be achieved by fluid balance and blood pressure control together with anemia control<sup>[28]</sup>. Foley et al. found that improvements in LV mass and systolic function 1 year after the initiation of dialysis therapy were associated with reduction rates of cardiac failure but not ischemic cardiac events and death.<sup>[29]</sup> Marchais et al.<sup>[30]</sup> found increased diastolic and mean arterial pressures, a higher cardiac index, higher heart rates, and an increased stroke index in hyperphosphatemic versus normophosphatemic patients. In fact, higher plasma phosphate is associated with signs of diastolic dysfunction and myocardial fibrosis and it is accountable for enhancing

LVH; hence, hyperphosphatemia might be an appropriate target for treatment<sup>[31]</sup>.

## CONCLUSION

The results of this study showed that a higher percentage of hypertension patients were found in patients with a high LV hypertrophy and uric acid. Left ventricular hypertrophy is a very important preventive feature of CKD associated cardiomyopathy. At early stage if identification have been done for LVH & accordingly interventions should be done to avoid and/or regression of LVH should be encouraged. Echocardiography provides a simple, non-invasive technique that can identify even asymptomatic patients at a prior stage of CKD.

**LIMITATIONS:** Due to the Covid 19 epidemic, a smaller sample size was covered than intended. The Covid 19 pandemic had a significant impact on patient follow-up.

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## REFERENCES

1. Waśen E, Isoaho R, Mattila K, Vahlberg T, Kivelä S-L, Irjala K. Estimation of glomerular filtration rate in the elderly: a comparison of creatinine-based formulae with serum cystatin C. *J Intern Med.* 2004;256(1):70-8.
2. Rothenbacher D, Klenk J, Denking M, Karakas M, Nikolaus T, Peter R et al. Prevalence and determinants of chronic kidney disease in community-dwelling elderly by various estimating equations. *BMC Public Health.* 2012;12:article 343. doi: [10.1186/1471-2458-12-343](https://doi.org/10.1186/1471-2458-12-343), PMID [22574773](https://pubmed.ncbi.nlm.nih.gov/22574773/).
3. Zhang QL, Rothenbacher D. Prevalence of chronic kidney disease in population-based studies: systematic review. *BMC Public Health.* 2008;8:article 117. doi: [10.1186/1471-2458-8-117](https://doi.org/10.1186/1471-2458-8-117), PMID [18405348](https://pubmed.ncbi.nlm.nih.gov/18405348/).
4. Hemmelgarn BR, Zhang J, Manns BJ, Tonelli M, Larsen E, Ghali WA et al. Progression of kidney dysfunction in the community-dwelling elderly. *Kidney Int.* 2006;69(12):2155-61. doi: [10.1038/sj.ki.5000270](https://doi.org/10.1038/sj.ki.5000270), PMID [16531986](https://pubmed.ncbi.nlm.nih.gov/16531986/).
5. Li Z-Y, Xu G-B, Xia T-A, Wang H-Y. Prevalence of chronic kidney disease in a middle and old-age population of Beijing. *Clin Chim Acta.* 2006;366(1-2):209-15. doi: [10.1016/j.cca.2005.10.011](https://doi.org/10.1016/j.cca.2005.10.011).
6. Ruggenenti P, Schieppati A, Remuzzi G. Progression, remission, regression of chronic renal diseases. *Lancet.* 2001;357(9268):1601-8. doi: [10.1016/S0140-6736\(00\)04728-0](https://doi.org/10.1016/S0140-6736(00)04728-0), PMID [11377666](https://pubmed.ncbi.nlm.nih.gov/11377666/).
7. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care.* 2004;27(5):1047-53. doi: [10.2337/diacare.27.5.1047](https://doi.org/10.2337/diacare.27.5.1047), PMID [15111519](https://pubmed.ncbi.nlm.nih.gov/15111519/).
8. Gupta R. Trends in hypertension epidemiology in India. *J Hum Hypertens.* 2004;18(2):73-8. doi: [10.1038/sj.jhh.1001633](https://doi.org/10.1038/sj.jhh.1001633), PMID [14730320](https://pubmed.ncbi.nlm.nih.gov/14730320/).
9. Modi GK, Jha V. V: The incidence of end-stage renal disease in India: a population-based study. *Kidney Int.* 2006;70(12):2131-3. doi: [10.1038/sj.ki.5001958](https://doi.org/10.1038/sj.ki.5001958), PMID [17063176](https://pubmed.ncbi.nlm.nih.gov/17063176/).
10. Kher V. End-stage renal disease in developing countries. *Kidney Int.* 2002;62(1):350-62. doi: [10.1046/j.1523-1755.2002.00426.x](https://doi.org/10.1046/j.1523-1755.2002.00426.x), PMID [12081600](https://pubmed.ncbi.nlm.nih.gov/12081600/).
11. Lorell BH, Carabello BA. Left ventricular hypertrophy: pathogenesis, detection, and prognosis. *Circulation.* 2000;102(4):470-9. doi: [10.1161/01.cir.102.4.470](https://doi.org/10.1161/01.cir.102.4.470), PMID [10908222](https://pubmed.ncbi.nlm.nih.gov/10908222/).
12. Glasscock RJ, Pecoits-Filho R, Barberato SH. Left ventricular mass in chronic kidney disease and ESRD. *Clin J Am Soc Nephrol.* 2009;4;Suppl 1:S79-91. doi: [10.2215/CJN.04860709](https://doi.org/10.2215/CJN.04860709), PMID [19996010](https://pubmed.ncbi.nlm.nih.gov/19996010/).
13. London GM. Left ventricular alterations and end-stage renal disease. *Nephrol Dial Transplant.* 2002;17;Suppl 1:29-36. doi: [10.1093/ndt/17.suppl\\_1.29](https://doi.org/10.1093/ndt/17.suppl_1.29), PMID [11812909](https://pubmed.ncbi.nlm.nih.gov/11812909/).
14. Berl T, Henrich W. Kidney-heart interactions: epidemiology, pathogenesis, and treatment. *Clin J Am Soc Nephrol.* 2006;1(1):8-18. doi: [10.2215/CJN.00730805](https://doi.org/10.2215/CJN.00730805), PMID [17699186](https://pubmed.ncbi.nlm.nih.gov/17699186/).
15. Schiffrin EL, Lipman ML, Mann JF. Chronic kidney disease: effects on the cardiovascular system. *Circulation.* 2007;116(1):85-97. doi: [10.1161/CIRCULATIONAHA.106.678342](https://doi.org/10.1161/CIRCULATIONAHA.106.678342), PMID [17606856](https://pubmed.ncbi.nlm.nih.gov/17606856/).
16. Glasscock RJ, Pecoits-Filho R, Barberato SH. Left ventricular mass in chronic kidney disease and ESRD. *Clin J Am Soc Nephrol.* 2009;4;Suppl 1:S79-91. doi: [10.2215/CJN.04860709](https://doi.org/10.2215/CJN.04860709), PMID [19996010](https://pubmed.ncbi.nlm.nih.gov/19996010/).

17. Park M, Hsu CY, Li Y, Mishra RK, Keane M, Rosas SE et al. Associations between kidney function and subclinical cardiac abnormalities in CKD. *J Am Soc Nephrol.* 2012;23(10):1725-34. doi: [10.1681/ASN.2012020145](https://doi.org/10.1681/ASN.2012020145), PMID [22935481](https://pubmed.ncbi.nlm.nih.gov/22935481/).
18. Paoletti E, Bellino D, Gallina AM, Amidone M, Cassottana P, Cannella G. Is left ventricular hypertrophy a powerful predictor of progression to dialysis in chronic kidney disease? *Nephrol Dial Transplant.* 2011;26(2):670-7. doi: [10.1093/ndt/gfq409](https://doi.org/10.1093/ndt/gfq409), PMID [20628183](https://pubmed.ncbi.nlm.nih.gov/20628183/).
19. Paoletti E, Bellino D, Cassottana P, Rolla D, Cannella G. Left ventricular hypertrophy in nondiabetic predialysis CKD. *Am J Kidney Dis.* 2005;46(2):320-7. doi: [10.1053/j.ajkd.2005.04.031](https://doi.org/10.1053/j.ajkd.2005.04.031), PMID [16112052](https://pubmed.ncbi.nlm.nih.gov/16112052/).
20. Madero M, Samak MJ, Wang X, Greene T, Beck GJ, Kusek JW et al. Uric acid and long-term outcomes in CKD. *Am J Kidney Dis.* 2009 May 1;53(5):796-803. doi: [10.1053/j.ajkd.2008.12.021](https://doi.org/10.1053/j.ajkd.2008.12.021), PMID [19303683](https://pubmed.ncbi.nlm.nih.gov/19303683/).
21. Glasscock RJ, Pecoits-Filho R, Barberato SH. Left ventricular mass in chronic kidney disease and ESRD. *Clin J Am Soc Nephrol.* 2009;4;Suppl 1:S79-91. doi: [10.2215/CJN.04860709](https://doi.org/10.2215/CJN.04860709), PMID [19996010](https://pubmed.ncbi.nlm.nih.gov/19996010/).
22. Zoccali C, Benedetto FA, Mallamaci F, Tripepi G, Giaccone G, Stancanelli B, et al. Left ventricular mass monitoring in the follow-up of dialysis patients: prognostic value of left ventricular hypertrophy progression. *Kidney Int.* 2004;65(4):1492-8). doi: [10.1111/j.1523-1755.2004.00530.x](https://doi.org/10.1111/j.1523-1755.2004.00530.x), PMID [15086493](https://pubmed.ncbi.nlm.nih.gov/15086493/).
23. Paoletti E, Bellino D, Cassottana P, Rolla D, Cannella G. Left ventricular hypertrophy in nondiabetic predialysis CKD. *Am J Kidney Dis.* 2005;46(2):320-7. doi: [10.1053/j.ajkd.2005.04.031](https://doi.org/10.1053/j.ajkd.2005.04.031), PMID [16112052](https://pubmed.ncbi.nlm.nih.gov/16112052/).
24. Suzuki H, Inoue T, Dogi M, Kikuta T, Takenaka T, Okada H. Decline of renal function and progression of left ventricular hypertrophy are independently determined in chronic kidney disease Stages 3-5. *Pulse (Basel).* 2014;2(1-4):29-37. doi: [10.1159/000368678](https://doi.org/10.1159/000368678), PMID [26587441](https://pubmed.ncbi.nlm.nih.gov/26587441/).
25. Yin Z, Fang Z, Yang M, Du X, Nie B, Gao K. Predictive value of SUA levels on mortality in acute coronary syndrome patients with chronic kidney disease after drug-eluting stent implantation. *Cardiology.* 2013;125(4):204-12. doi: [10.1159/000350953](https://doi.org/10.1159/000350953), PMID [23796962](https://pubmed.ncbi.nlm.nih.gov/23796962/).
26. Aoki J, Ikari Y, Nakajima H, Mori M, Sugimoto T, Hatori M et al. Clinical and pathologic characteristics of dilated cardiomyopathy in hemodialysis patients. *Kidney Int.* 2005;67(1):333-40. doi: [10.1111/j.1523-1755.2005.00086.x](https://doi.org/10.1111/j.1523-1755.2005.00086.x), PMID [15610259](https://pubmed.ncbi.nlm.nih.gov/15610259/).
27. McMahon LP, Roger SD, Levin A, Slimheart Investigators Group. Development, prevention, and potential reversal of left ventricular hypertrophy in chronic kidney disease. *J Am Soc Nephrol.* 2004;15(6):1640-7. doi: [10.1097/01.asn.0000130566.69170.5e](https://doi.org/10.1097/01.asn.0000130566.69170.5e), PMID [15153576](https://pubmed.ncbi.nlm.nih.gov/15153576/).
28. Culleton BF, Walsh M, Klarenbach SW, Mortis G, Scott-Douglas N, Quinn RR et al. Effect of frequent nocturnal hemodialysis vs conventional hemodialysis on left ventricular mass and quality of life: a randomized controlled trial. *JAMA.* 2007;298(11):1291-9. doi: [10.1001/jama.298.11.1291](https://doi.org/10.1001/jama.298.11.1291), PMID [17878421](https://pubmed.ncbi.nlm.nih.gov/17878421/).
29. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE. Serial change in echocardiographic parameters and cardiac failure in end-stage renal disease. *J Am Soc Nephrol.* 2000;11(5):912-6. doi: [10.1681/ASN.V115912](https://doi.org/10.1681/ASN.V115912), PMID [10770969](https://pubmed.ncbi.nlm.nih.gov/10770969/).
30. Marchais SJ, Metivier F, Guerin AP, London GM. Association of hyperphosphataemia with haemodynamic disturbances in end-stage renal disease. *Nephrol Dial Transplant.* 1999;14(9):2178-83. doi: [10.1093/ndt/14.9.2178](https://doi.org/10.1093/ndt/14.9.2178), PMID [10489228](https://pubmed.ncbi.nlm.nih.gov/10489228/).
31. Galetta F, Cupisti A, Franzoni F, Femia FR, Rossi M, Barsotti G et al. Left ventricular function and calcium phosphate plasma levels in uraemic patients. *J Intern Med.* 2005;258(4):378-84. doi: [10.1111/j.1365-2796.2005.01544.x](https://doi.org/10.1111/j.1365-2796.2005.01544.x), PMID [16164578](https://pubmed.ncbi.nlm.nih.gov/16164578/).