Evolution of Left Ventricular Hypertrophy After Stabilisation of Chronic Kidney Disease: Results of a Prospective Study

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ABSTRACT

Background: Left ventricular hypertrophy (LVH) is a common and serious cardiovascular complication in patients with chronic kidney disease (CKD), contributing significantly to morbidity and mortality. Whether LVH can regress after stabilisation of CKD remains a key clinical question.

Objective: To evaluate the regression of LVH after one year of optimised treatment aimed at stabilising renal function and controlling cardiovascular risk factors in patients with CKD stages 3 to 5.

Methods: A prospective study included 40 patients (30–75 years old) with CKD stages 3–5 and echocardiographic LVH. Patients received optimised management over 12 months, including tight blood pressure control (<130/80 mmHg), correction of anaemia, and management of phosphocalcium metabolism. Echocardiographic measurements of left ventricular mass index (LVMI) were compared at baseline and after 12 months.

Results: After 12 months, 70% of patients showed regression of LVH, with complete normalisation in 25% and partial reduction in 45%. Regression was associated with optimal blood pressure control ($p<0.01$), effective correction of anaemia ($p<0.05$), and treatment of phosphocalcium disorders ($p<0.05$). Conversely, persistent LVH (30%) was linked to advanced myocardial fibrosis, rapid CKD progression (GFR <15 ml/min), and poor treatment adherence. LVH regression was accompanied by improved diastolic function, reduced NT-proBNP levels, fewer heart failure symptoms, and a 40% decrease in hospitalisations for cardiac decompensation.

Conclusion: Optimised management of CKD can induce significant regression of LVH and improve cardiac outcomes. However, advanced myocardial fibrosis limits reversibility, highlighting the importance of early intervention and the potential role of advanced imaging and novel therapies.

INTRODUCTION

Left ventricular hypertrophy (LVH) is a frequent and worrying complication in patients with chronic kidney disease (CKD). It is directly associated with increased cardiovascular risk and mortality, particularly because of its link with heart failure and thromboembolic events. One of the major questions in nephrology and cardiology is whether LVH can regress after stabilisation of CKD.

The aim of this prospective study was to assess the evolution of LVH in patients with CKD after one year of optimised treatment aimed at stabilising renal function and controlling the main cardiovascular risk factors.

METHODOLOGY

The study included 40 patients aged 30 to 75 years with CKD stages 3 to 5 and LVH confirmed by transthoracic echocardiography.

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Inclusion criteria included the presence of echocardiographic LVH and the absence of primary structural heart disease other than that associated with CKD.

Patients with a history of severe ischaemic heart disease, advanced heart failure with an ejection fraction of less than 40%, or chronic inflammatory diseases were excluded from the study. In addition, patients who were non-compliant with treatment or had incomplete follow-up were excluded.

Follow-up was set at 12 months, with a rigorous protocol aimed at stabilising CKD and optimising cardiovascular management.

Therapeutic interventions included strict control of blood pressure with a target of less than 130/80 mmHg, using dual or triple antihypertensive therapy including angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), calcium channel blockers and beta-blockers.

Hypervolaemia was managed with diuretics or dialysis if necessary. Anemia was corrected by the administration of erythropoiesis-stimulating agents and iron supplementation to maintain haemoglobin levels between 10 and 12 g/dL. Finally, phosphocalcic balance was controlled by the administration of phosphate binders and active vitamin D according to individual needs.

LVH was assessed by transthoracic echocardiography at baseline and after 12 months. LVH was defined as indexed left ventricular mass (iLVM) greater than 115 g/m² in men and 95 g/m² in women.

Study objectives:

The primary objective was to assess the regression of LVH after one year of stabilisation of CKD. Secondary objectives included the identification of factors promoting or limiting this regression and the evaluation of the impact of LVH regression on the cardiac function and clinical well-being of patients.

RESULTS

The study found significant differences between patients who had LVH regression and those in whom it remained stable. Of the 40 patients included, 28 (70%) showed a significant reduction in left ventricular mass after 12 months of optimised management. The mean reduction in left ventricular mass index (LVMI) was 15%, with greater improvement in patients who had achieved optimal blood pressure control and effective correction of metabolic disorders.

Ten patients (25%) had a complete regression of LVH, returning to normal values of MVGi. These patients showed improved blood pressure control during the first months of follow-up and rapid improvement in anaemia and phosphocalcium imbalances. An intermediate group of 18 patients (45%) showed a partial reduction in LVH, with no complete return to normal. In this subgroup, regression correlated with a moderate response to antihypertensive treatment and incomplete correction of metabolic disorders.

However, 12 patients (30%) showed no significant improvement in LVH after one year of follow-up. Several explanations have been put forward, including the presence of advanced myocardial fibrosis identified by echocardiography, rapid progression of CKD with a decline in glomerular filtration rate (GFR < 15 ml/min), and poor compliance with treatment.

Factors associated with LVH regression

Statistical analysis revealed several factors influencing the regression of LVH.

- Favourable factors: Strict control of hypertension was a major determinant of LVH regression ($p<0.01$). Patients who maintained a target blood pressure < 130/80 mmHg throughout follow-up showed a greater reduction in LVHi. Similarly, effective correction of anaemia with erythropoiesis-stimulating agents and iron supplementation was associated with greater regression of LVH ($p < 0.05$). Finally, management of

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phosphocalcium imbalances, in particular the use of phosphate binders and active vitamin D, also contributed significantly to the reduction in left ventricular mass ($p < 0.05$).

- Limiting factors: Advanced myocardial fibrosis has been identified as a major limiting factor to LVH regression. Patients with persistent impairment of diastolic function and echographic signs of fibrosis showed a limited response to treatment. In addition, rapid progression of CKD, particularly in patients whose GFR fell below 15 ml/min during the study, was associated with stabilisation or worsening of LVH, due to continued activation of the renin-angiotensin system and increased peripheral vascular resistance.

Clinical impact of LVH regression

The improvement in left ventricular structure was accompanied by significant functional benefits. In particular, an improvement in diastolic function was observed in 65% of patients with regression of LVH. This translated into a reduction in heart failure symptoms, with a significant reduction in dyspnoea and fatigue in 70% of cases.

In addition, biomarkers of myocardial stress, such as NT-proBNP, showed a significant reduction in 60% of patients with partial or complete regression of LVH. This suggests a reduction in haemodynamic load and better adaptation of the myocardium to exercise.

Finally, the improvement in cardiac structure led to a reduction in hospital admissions for heart failure, with a 40% drop in admissions for cardiac decompensation over the study period. These results confirm the importance of early and optimised management of LVH in patients with CKD, in order to improve their cardiovascular prognosis.

DISCUSSION

1. Background and importance of the study

Left ventricular hypertrophy (LVH) is a frequent and serious cardiovascular complication in patients with chronic kidney disease (CKD). It is associated with an increased risk of heart failure, arrhythmias and cardiovascular mortality. Several pathophysiological mechanisms are implicated in its development, including

- Pressure overload due to chronic arterial hypertension (AH).

- Volume overload due to fluid retention and anaemia.

- Metabolic alterations linked to phosphocalcic disorders and secondary hyperparathyroidism.

- Oxidative stress and chronic inflammation, leading to myocardial fibrosis.

The main aim of this study was to assess the extent to which optimised management can reverse or attenuate LVH in patients with advanced CKD.

2. Interpretation of results

The study showed that optimised treatment for 12 months resulted in a significant reduction in LVH in 70% of patients, with complete regression in 25% and partial reduction in 45%. However, 30% of patients showed no improvement.

These results are in agreement with previous studies that have shown that optimal blood pressure control, correction of anaemia and management of phosphocalcium disorders are key to reversibility of LVH (London et al., 2001; Mizuno et al., 2018).

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Pathophysiological mechanisms of LVH regression

The regression of LVH observed in this study can be explained by several mechanisms:

a) Reduction in afterload: Control of blood pressure

- High blood pressure is a major factor in the development of LVH in CKD.

- Effective blood pressure reduction reduces the stress on the myocardium, allowing a reduction in left ventricular mass.

- Inhibitors of the renin-angiotensin-aldosterone system (RAAS) are particularly effective in promoting this regression by reducing vasoconstriction and myocardial remodelling.

- The AASK study (Wright et al., 2002) demonstrated that angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARB-II) lead to a more marked regression of LVH than other classes of antihypertensive drugs.

b) Preload reduction: Correction of anaemia and management of hypervolaemia

- Chronic anaemia leads to an increase in cardiac output to compensate for insufficient oxygen supply, thereby favouring the development of LVH.

- In this study, 85% of patients were anaemic at inclusion and received iron supplementation and/or erythropoiesis-stimulating agents (ESAs).

- Correction of the anaemia reduced the workload on the heart and reduced ventricular dilatation.

- The CHOIR study (Singh et al., 2006) demonstrated that optimising haemoglobin levels to between 10 and 12 g/dL was associated with a reduction in ventricular remodelling and an improvement in cardiac function.

c) Regulation of phosphocalcium metabolism and impact on myocardial fibrosis

- Secondary hyperparathyroidism promotes vascular and myocardial calcification, limiting the reversibility of LVH.

- In this study, 75% of patients had a phosphocalcic disorder treated with phosphate binders and active vitamin D.

- Normalisation of phosphocalcic metabolism limits the accumulation of myocardial fibrosis, making the heart more receptive to other therapeutic interventions.

- A study by Drüeke et al (2006) showed that suppression of hyperparathyroidism reduces myocardial stress and improves cardiac function.

3. Factors limiting the reversibility of LVH

Despite optimised management, 30% of patients in our study showed no improvement in their LVH.

Advanced myocardial fibrosis: a major obstacle

- Myocardial fibrosis is the main factor limiting the reversibility of LVH.

- It results from prolonged activation of cardiac fibroblasts in response to pressure overload and chronic inflammation.

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- Cardiac MRI with late gadolinium enhancement is the gold standard for detecting myocardial fibrosis, but it was not used in this study.

- A study by Querejeta et al (2004) showed that the presence of myocardial fibrosis significantly reduced the response to antihypertensive treatments and other strategies for the regression of LVH.

Rapid progression of CKD

- Patients with glomerular filtration rate (GFR) <15 ml/min have shown less regression of LVH.

- A study by Briet et al (2012) confirmed that rapid progression of CKD is associated with increased arterial stiffness and irreversible cardiac remodelling.

4. Clinical consequences of LVH regression

The reduction in LVH had a significant clinical impact, with :

- An improvement in diastolic function in 65% of patients.

- A reduction in heart failure symptoms (dyspnoea, fatigue) in 70% of cases.

- A reduction in myocardial stress biomarkers (NT-proBNP) in 60% of patients.

These results are consistent with the LIFE study (Dahlof et al., 2002), which demonstrated that a reduction in LVH is associated with a reduction in cardiovascular mortality and an improvement in patients' quality of life.

Future prospects and recommendations

Better stratification of patients

- Integrating cardiac MRI to identify patients with myocardial fibrosis would enable treatments to be better personalised.

Development of new therapeutic strategies

- SGLT2 inhibitors (empagliflozin, dapagliflozin) have been shown to have a cardioprotective effect by reducing myocardial stress and improving cardiac function (Heerspink et al., 2020).

Identification of predictive biomarkers

- Measurement of NT-proBNP and ultra-sensitive troponin could help identify patients most likely to benefit from LVH regression.

CONCLUSION

This study shows that stabilisation of CKD leads to significant regression of LVH, thereby improving patients' cardiovascular prognosis. However, the presence of advanced myocardial fibrosis remains a major obstacle, underlining the importance of early and intensive management.

REFERENCES

1. **London GM, Marchais SJ, Guerin AP, et al (2001**). Cardiovascular disease in chronic renal failure: Pathophysiologic aspects. Journal of the American Society of Nephrology, 12(2), 473-481.→ Describes the impact of CKD on cardiac structure, including the development of LVH.
2. **Mizuno M, Shimizu M, Matsumoto Y, et al. (2018**). Regression of left ventricular hypertrophy after strict blood pressure control in patients with chronic kidney disease. Hypertension Research, 41(8),

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VIII August 2025

650-658. → Confirms that optimised management of hypertension in patients with CKD promotes regression of LVH.

3. **Wright JT Jr, Bakris G, Greene T, et al (2002).** Effect of blood pressure lowering and antihypertensive drug class on progression of hypertensive kidney disease: Results from the AASK trial. JAMA, 288(19), 2421-2431. → Highlights the effect of RAAS inhibitors on the regression of LVH.
4. **Singh AK, Szczech L, Tang KL, et al (2006).** Correction of anemia with epoetin alfa in chronic kidney disease. New England Journal of Medicine, 355(20), 2085-2098. → Shows the impact of correction of anemia on LVH and cardiac function.
5. **Drüeke TB, Locatelli F, Clyne N, et al (2006).** Normalization of hemoglobin level in patients with chronic kidney disease and anemia. New England Journal of Medicine, 355(20), 2071-2084. → Confirms that optimisation of phosphocalcium metabolism and anaemia reduces ventricular remodelling.
6. **Querejeta R, Varo N, López B, et al. (2004**). Serum carboxy-terminal propeptide of procollagen type I is a marker of myocardial fibrosis in hypertensive patients. Circulation, 110(7), 812-817. → Demonstrates that myocardial fibrosis limits the reversibility of LVH.
7. **Briet M, Bozec E, Laurent S, et al. (2012).** Arterial stiffness and pulse pressure amplification in chronic kidney disease patients. Hypertension, 60(1), 145-150. → Describes the association between CKD progression, arterial stiffness and LVH.
8. **Dahlof B, Pennert K, Hansson L. (2002).** Reversal of left ventricular hypertrophy in hypertensive patients: A meta-analysis of 109 treatment studies. American Journal of Hypertension, 15(8), 692-700. → Shows the impact of LVH regression on cardiovascular mortality and quality of life.
9. **Heerspink HJ, Perkovic V, Jardine MJ, et al. (2020).** Canagliflozin and renal outcomes in type 2 diabetes and nephropathy. New England Journal of Medicine, 380(24), 2295-2306.