



Original article

Cardiac abnormalities among chronic hemodialysis patients

Lallafatimaezzahra el hassani¹, Salwacheraou^{2*}, Latifaoukerraj³, Nadia fellat⁴, Rokya fellat⁵,
Mohamed cherti⁶

¹⁻⁶Department of cardiology, IbnSina University Hospital Center, Rabat, Morocco.

ABSTRACT

Objective: The annual mortality of cardiovascular diseases of dialysis patients is higher than the general population. The transthoracic echocardiography allows the evaluation of the heart structure and function within the treated patients by hemodialysis in order to identify patients with cardiovascular high risk. **Methods:** This work is a descriptive retrospective study. The objective is to determine the major cardiac abnormalities diagnosed with echocardiography in patients with chronic hemodialysis and to list their epidemiological, clinical and biological characteristics at the time of the study. **Results:** The average age of our patients is of 50.2 ± 7 years with an average hemodialysis endurance of 12.1 ± 2.4 years. The main etiologies of the chronic renal failure were essentially the diabetes type 2, the arterial hypertension and chronic nephritis tubule-interstitial. The most cardiac abnormalities was the left ventricular hypertrophy. The presence of a systolic or diastolic arterial hypertension has been noted as a significant factor fostering the LVH within the chronic hemodialysis ($p=0.002$). The anemia is not said to be associated to the development of the left ventricular hypertrophy ($p=0.09$). **Conclusions:** This study revealed the etiology leading to chronic renal failure insufficiency. Echocardiography accurately diagnosed cardiac abnormalities such as left ventricular hypertrophy. The study allowed to detect the factors involved in the development of this HVG especially systolic arterial hypertension. This result permit us to act on these factors in order to prevent the cardiovascular events to which hemodialysis patients will be exposed

KEYWORDS: Cardiovascular disease, Chronic kidney, Doppler, Echocardiography, Hemodialysis, Left ventricular hypertrophy.

INTRODUCTION

The annual mortality of cardiovascular diseases of dialysis patients is higher than the general population. The main etiologies of the chronic renal failure are essentially the diabetes type 2, the vascular pathology such the arterial hypertension or anomaly of kidney arteries, the glomerulopathies and the kidney polykystose.

This high incidence reflects in part the frequent heart anomalies structural and functional within that group of patients [1] [2]. The echocardiography; an non-invasive examination; takes a great importance in the screening and the diagnosis of heart attack. It allows the evaluation of the heart structure and function within the treated patients by hemodialysis in order to identify patients with cardiovascular high risk [3].

The heart attack of chronic hemodialysis is multifactorial to know the arterial hypertension, the anemia, the chronic inflammation, the hyperparathyroidism, the

homocysteinemia and so on [4]. The left ventricular hypertrophy (LVH) is the morphological anomaly the most frequent. The incidence of the LVH increases with the deterioration of the kidney function.

The LVH is found in 60 to 75% of patients who reach the terminal or final phase of the chronic renal insufficiency and in 60 to 90% of those regularly dialysed [5] [6]. It is about an adaptive remodeling to an overcharge of a volume (hypervolemia) and/ or of a pression (arterial hypertension) permitting also to the left ventricular to maintain a stable parietal tension [7] [8].

There are also non-hemodynamic factors that stimulate directly the cell growth, causing the LVH, such as the chronic kidney disease, the bony and mineral troubles, the renin-angiotensin system, and the endotheline [9] [10].

In addition the reduction of the ventricular mass consists in a treatment of risk factors of the LVH permitting also to decrease the causes of cardiovascular mortality

[11].Through this work performed on 29 chronic hemodialysis patients, focused on the different heart anomalies objective to the transthoracic-electrocardiography and enumerate the clinical and biological characteristics of patients at the time of the study.

MATERIALS AND METHODS

This work is a descriptive retrospective study, lasting or spreading on a period of 12 months. Our study involves 29 cases of chronic renal failure at final or terminal phase treated by hemodialysis since more than 8 months ago and having been the object of a transthoracic echocardiography. We collected all the patients who came to our department and who met the criteria of the study.

We have resorted all medical files of patients who are followed up in the department of nephrology and sent by their nephrologists in our department to benefit from an echocardiography. Their medical files contain the clinical observation of patients, the paraclinical examinations, the transthoracic echocardiographic reports and the patient follow-up. All clinical and paraclinical parameters were reported in the SPSS software to establish the statistics.

The clinical aspects : age, sex, the hemodialysis duration, the initial kidney disease, the systolic (SBP) and diastolic = (DBP) arterial pressure ,the inter dialytic weight gain.

The biological aspects: the serum calcium, the phosphoremia, the B natriuretic peptide (BNP) rate, the cholesterol rates, of high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, hemoglobin rate, the ferritin, the c-reactive protein, the albuminemia and the parathormone

The echocardiographic aspects: the study has involved: the heart structures (myocardium, pericardium, endocardial), the different types of morphological anomalies (calcification, dilation and hypertrophy), the systolic and/ or diastolic functional repercussion, the heart parameters studied: the inter-ventricular septum, the posterior wall, the ejection fraction, the telediastolic diameter of the left ventricle so as to calculate the left ventricle mass and the diameter of the inferior cave vein. We used the SPSS software to do the statistics.

RESULTS

We collect in our study 29 patients including 17 women and 12 men with a sex ratio at 0.9. The average age of our population is of 50.2 ± 7 years with an average hemodialysis endurance of 12.1 ± 2.4 years.

The demographic, clinical, and biological characteristics of our population are represented in table 1 and 2. These values are attributed by our laboratories (laboratories of IbnSina Hospital Center).

Table 1: Initial kidney disease among study subjects

Diabetic nephropathy	9 (31%)
Arterial hypertension	5 (17%)
Chronic nephritis tubulo-interstitial	4 (14%)
Chronic Glomerulo-nephritis	1 (3.5%)
Undetermined	10 (34.5%)

Table 2: Biological Characteristics and blood pressure of participants at the time of the study

Biological analysis	Mean \pm SD	Normal value
Calcemia	90 mg/l \pm 5	(80-105)
Phosphoremia	32 mg/l \pm 12	(25-50)
B-Natriuretic Peptide	170 pg/ml \pm 75	(<50)
Albuminemia	42 g/l \pm 3	(35-50)
LDL‡	1.05 g/l \pm 0.3	(<1.55)
HDL†	0.35 g/l \pm 0.5	(0.40-0.60)
Triglyceride	1.5 g/l \pm 0.6	(0.6-1.5)
Cholestérole mia	2 g/l \pm 0.5	(1.50-2.00)
Hemoglobin	9.5 g/dl \pm 1	(12-16)

Ferritinemia	328 ng/ml ± 120	(30-300)
C-reactive protéine	4 mg/l ± 2.8	(<5.0)
Parathormone	52 ng/l ± 8	(11-54)
Average systolic blood pressure (mmHg)	145mmHg ± 0.5	
Average diastolic pressure (mmHg)	90mmHg ± 1.5	

† High density lipoprotein, ‡ Lowdensity lipoprotein

As part of the follow-up, hemodialysis patients were referred by their nephrologist; in our department; to benefit from trans-thoracic-echocardiography. The echocardiography has been realised between 2 hemodialysis

sessions to have a volemia close to the normal in order to avoid the sodium and water overcharge factor. The echocardiographic anomalies found shown in table 3.

Table 3: The different heart anomalies shown in trans-thoracic echocardiography

Left ventricular hypertrophy	13 (44.5%)
Valvular calcification	7 (26.5%)
Left ventricule dilation	3 (10%)
Pulmonary arterial hypertension	3 (10%)
Pericarditis	2 (6%)
Dysfunction of left ventricular	1 (3%)

DISCUSSION

Patients with chronic renal failure treated by dialysis show and present high morbidity and mortality rates. Heart attack represents nearly half of this mortality. In case of terminal renal failure, the mortality is about ten times superior to that observed within the general population [12].

In 2010, the incidence of all-causes mortality was of 236 deceased/ 1000- year patients at risk. [13]. The most common cause is sudden death. This high incidence reflects in part the frequent structural and functional heart anomalies within this population.

The precocious detection of these heart anomalies could be important in order to permit opportune and appropriate heart interventions integrating several ultrasonographic techniques in a single examination. The Doppler echography is a non-invasive examination which is largely available and reproducible that permits to afford precious information on the heart morphology and function. It is recommended to realise a reference examination at the beginning of dialysis then each 3 years. In case of heart affections the check-up will be done annually considering the frequency and the seriousness of the associated cardio-vascular pathologies.

The main heart anomaly; described in literature; noticed in dialysis patients is the LVH [8]. Its incidence increases with the deterioration of kidney function [3] [4]. Eventually, 70% of patients attaining renal failure will have an LVH at the beginning of dialysis [1] [14]. This LVH is a process of adaptive remodeling on iterative overloads of volume and pressure. The object is to maintain a stable parietal tension.

These phenomena of remodeling cause in long term an alteration of the systolic or diastolic function resulting in heart failure, rythm trouble, sudden death.

There exist other non-hemodynamic factors that stimulate directly the cell growth, causing LVH, which are: mineral trouble, the activation of the renin angiotensin system [9] [10].

The hypervolemia, anemia and the arteriovenous fistula also contribute to a volumic overload [15] [16] and consequently to a risk of hypertrophy development. The anemia causes an increase of the cardiac flow by elevating the cardiac frequency and the volume of systolic ejection. This will lead to a chronic volemia overload responsible for a left ventricular dilation and a septal thickening in echocardiography [17]. The implication of the anemia has been highlighted in a number of series, notably london and coll [18] which finds so an inverse link between the hemoglobin concentration, on one hand, the dilation and the left ventricular mass, on the other hand, in our series, the anemia is not said to be associated to the development of the LVH (p=0.09).

In literature, the LVH is found in 75% in the london series [19] and in 73.9% in the series of foley [20]. In our series the LVH has been found in 44.5% the cases which concords perfectly with the literature (p=0.001). The increase of the post charge within the frame of an arterial hypertension may also result in an LVH constitution. The concentric hypertrophy is more frequent than the excentric hypertrophy within hemodialysis patients, reflecting high prevalence of

chronic hypertension within this population [21]. In our series, like in several studies [22], the presence of a systolic or diastolic arterial hypertension has been noted as a significant factor fostering the LVH within the chronic hemodilysis (p=0.002).

The LVH represented a prognostic factor. It permits to predict the mortality and the cardiovascular occurrence events within dialysis patients [23] [24]. However, it can become poorly adapted and deleterious because of the cell death (apoptosis) secondary to a continued ventricular overcharge, to a reduction of the capillar thickness and to the myocardial fibrosis [11] [25] [26]. This influence has been highlighted by several authors, notably London and coll [18] that finds thereby an inverse relation between the concentration of the hemoglobin, on one hand, the dilation and the left ventricular mass, on the other hand. In our series, the anemia has not been revealed associated with the LVH development.

CONCLUSION

This study revealed the etiology leading to chronic renal failure. Echocardiography accurately diagnosed cardiac abnormalities such as left ventricular hypertrophy. The study allowed to detect the factors involved in the development of this HVG especially systolic arterial hypertension. These results permit us to act on these factors in order to prevent the cardiovascular events to which hemodialysis patients will be exposed. The electrocardiography allowed the prognostic and also permits to direct and evaluate the different therapeutic strategies for our patients.

Competing interest: The authors declare that they have no competing interests.

REFERENCES

1. Foley RN, Parfrey PS, Harnett JD, et al. Clinical and echocardiographic disease in patients starting end stage renal disease therapy. *Kidney Int.* 1995;47 (1):186-192.
2. Stecker EC, Vickers C, Waltz J, et al. Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction: two-year findings from the Oregon Sudden Unexpected Death Study. *J Am Coll Cardiol.* 2006;47(6):1161-1166.
3. Foley R, Parfrey P, Kent G, Harnett J, Murray D, Barre P. Serial change in echocardiographic parameters and cardiac failure in end-stage renal disease. *J Am Soc Nephrol.* 2000;11(5):912-916.
4. Park M, Hsu C, Li Y, et al. Associations between kidney function and subclinical cardiac abnormalities in CKD. *J Am Soc Nephrol.* 2012;23(10):1725-1734.
5. Schiffrin E, Lipman ML, Mann FE. Chronic Kidney Disease: Effects on the Cardiovascular System. *Circulation.* 2007;116(1):85-97. [[PubMed](#)]
6. Parfrey PS, Foley RN. The clinical epidemiology of cardiac disease in chronic renal failure. *J Am Soc Nephrol.* 1999;10(7):1606-1615. [[PubMed](#)]
7. Middleton RJ, Parfrey PS, Foley RN. Left Ventricular Hypertrophy in the Renal Patient. *J Am Soc Nephrol.* 2001;12(5):1079-1084. [[PubMed](#)]
8. London GM. Left ventricular alterations and end-stage renal disease. *Nephrol Dial Transplant.* 2002;17(Suppl 1):29-36. [[PubMed](#)]
9. London GM. Left ventricular alterations and end-stage renal disease. *Nephrol Dial Transplant.* 2002;17(suppl 1):29-36.
10. Glasscock RJ, Pecoits-Filho R, Barberato SH. Left ventricular mass in chronic kidney disease and ESRD. *Clin J Am Soc Nephrol.* 2009;4 (7) (suppl 1) :S79-S91.
11. London GM, Pannier B, Guerin AP, et al. Alterations of left ventricular hypertrophy in and survival of patients receiving hemodialysis: follow-up of an interventional study. *J Am Soc Nephrol.* 2001;12(12):2759-2767. [[PubMed](#)]
12. R.N. Foley, A.J. Collins, End-stage renal disease in the United States: an update from the United States Renal Data System, *Journal of the American Society of Nephrology* 18 (2007) 2644-2648.
13. Collins AJ, Foley RN, Chavers B. United States Renal Data System 2011 annual data report: atlas of chronic kidney disease and end-stage renal disease in the United States. *Am J Kidney Dis.* 2012;59 (1) (suppl 1):e1-e420.
14. Cerasola G, Nardi E, Palermo A, Mulè G, Cottone S. Epidemiology and pathophysiology of left ventricular abnormalities in chronic kidney disease: a review. *J Nephrol.* 2011;24(1):1-10.
15. Stern A, Klemmer P. High-output heart failure secondary to arteriovenous fistula. *Haemodial Int.* 2011;15(1):104-107.
16. Okumura K, Io H, Matsumoto M, et al. Predictive factors associated with change rates of LV hypertrophy and renal dysfunction in CKD patients. *Clin Nephrol.* 2013;79(1):7-14.
17. Foley RN, Parfrey PS, et al. The impact of anemia on cardiomyopathy, morbidity and mortality in end stage renal disease. *Am J Kidney D.* 1996;28(1):53-61. [[PubMed](#)]
18. London G, Marchais SJ, Guerin AP. Cardiovascular function in hemodialysis patients. In: Grunfeld J, editor. *Advances in nephrology.* 1991. pp. 249-273. St louis. [[PubMed](#)]
19. London GM, Fabiani F. Left ventricular dysfunction in end stage renal disease Echocardiographic insights. In:

Parfery PS, editor. cardiac dysfunction in chronic uremia. 1992. pp. 117–138.

20. Foley RN, Parfrey PS, et al. Serial change in echocardiographic parameters and cardiac failure in endstage renal disease. *J Am SocNephrol.* 2000;11(5):912–916. [[PubMed](#)]

21. Agarwal R, Nissenson AR, Battle D, Coyne DW, Trout JR, Warnock DG. Prevalence, treatment, and control of hypertension in chronic hemodialysis patients in the United States. *Am J Med.* 2003;115(4):291-297.

22. Birchem JA, Fraley MA, Senkottaiyan N, Alpert MA. Influence of hypertension on cardiovascular outcomes in hemodialysis patients. *Semin Dial.* 2005 Sep-Oct;18(5):391–5. [[PubMed](#)]

23. Foley R, Parfrey P, Harnett J, Kent G, Murray D, Barre P. The prognostic importance of left ventricular geometry in

uremic cardiomyopathy. *J Am SocNephrol.* 1995;5(12):2024-2031.

24. Zoccali C, Benedetto F, Mallamaci F, et al. Prognostic impact of the indexation of left ventricular mass in patients undergoing dialysis. *J Am SocNephrol.* 2001;12(12):2768-2774.

25. Tamarappoo B, John B, Reinier K, et al. Vulnerable myocardial interstitium in patients with isolated left ventricular hypertrophy and sudden cardiac death: a postmortem histological

26. Ansari A, Kaupke C, Vaziri N, Miller R, Barbari A. Cardiac pathology in patients with end-stage renal disease maintained on hemodialysis. *Int J Artif Organs.* 1993;16(1):31-36.

*Corresponding author: SalwaCheraou
E-Mail: salwacheraou@gmail.com