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Left Ventricular hypertrophy: prevalence in chronic haemodialysis patients and its predictive factors of occurrence

L.ALTIT (1);S.ANIBAR(1);I.OUGHAZZOU(2) ;A.HADI (2);S.KARIMI (2);M.EL HATTAOUI (2);M.CHETTATI(1); W.FADILI (1) ;I.LAOUAD (1)

(1) Nephrology, haemodialysis and kidney transplant department, Mohamed 6th University Hospital Centre, Marrakech, Morocco.

(2) Cardiology Department, Mohamed 6th University Hospital Centre, Marrakech, Morocco.

ABSTRACT

Background: *Cardiovascular complications, particularly left ventricular hypertrophy (LVH), are the leading cause of morbidity and mortality in chronic haemodialysis patients.* ***Study objective:*** *describing the LVH prevalence in our population and determining its risk factors.* ***Patients and Methods:*** *We adopted a multicentric analytical prospective study carried out in both the nephrology, haemodialysis and kidney transplant department, and in the cardiology department of the Marrakech University Hospital Centre. We selected chronic haemodialysis patients that underwent thrice-weekly sessions of 4 hours and with a long-standing haemodialysis exceeding 9 months. Patients received a trans-thoracic echocardiogram 24 hours before and after the haemodialysis session by the same operator.* ***Results:*** *Our study concerned 40 patients. The age average is of 41.05 ± 18.8 years old. Seniority in haemodialysis is of 6.9 ± 6.17 years. Initial nephropathy was diabetic in 12.5% of cases. 72.5% cases had anaemia. 70% of patients had secondary hyperparathyroidism. 67.5% cases exhibited LVH. The interventricular septum thickness in diastole was averaging 12.92 ± 2.66 mm and 10.8 ± 2.5 mm before and after the haemodialysis session respectively. The posterior wall thickness in diastole was of 12.07 ± 2.2 mm before the haemodialysis session, and 10.2 ± 2.4 mm after it.* ***Conclusion:*** *Our study shows a high LVH prevalence. The LVH risk factors identified are anemia, high blood pressure, and hyperparathyroidism.*

Keywords : chronic haemodialysis, LVH, predictive factors

INTRODUCTION

Haemodialysis treatment in patients with renal insufficiency may affect cardiac function due to acute changes in blood volume, blood pressure, electrolytes and sympathetic vagal balance. Cardiovascular complications, particularly left ventricular hypertrophy (LVH), are the leading cause of morbidity and mortality in chronic haemodialysis patients. Being frequent and multifactorial,

LVH has a high prognostic value, most often associated with extensive myocardial fibrosis, and can have deleterious clinical consequences.

Cardiovascular diseases are the leading cause of mortality and morbidity in chronic kidney disease (CKD) patients .[1] Despite advances in CKD's therapeutic management, the increase in cardiovascular risk (CVR), with respect to the general population after adjustment for age, sex, geographical origin and diabetes, is significant at all stages of this disease and for any type of replacement therapy.[2]

The Scribner group's initial publication in 1974 drew attention to the unusually high rate of CV deaths in haemodialysis patients. [3]

In the Framingham cohort, the observed myocardial infarction (MI) incidence in dialysis patients was ten times higher than in hypertensive patients at equal age, even though these authors proposed the concept of "accelerated" atherosclerosis that is prompted by the uremic state prolonged by dialysis.

The aim of our work is to describe the LVH prevalence in our population and to determine its risk factors.

MATERIALS AND METHOD

We adopted a multicentric analytical prospective study which was carried out in both the nephrology, haemodialysis and kidney transplant department, and in the cardiology department of the Marrakech University Hospital Centre.

We selected chronic haemodialysis patients that underwent thrice-weekly sessions of 4 hours and with a long-standing haemodialysis exceeding 9 months. Patients received a trans-thoracic echocardiogram 24 hours before and after the haemodialysis session by the same operator. In order to elicit the LVH's occurrence factors, we compared demographic, clinico-biological, and echocardiographic parameters between patients with and without LVH.

Statistical analysis was performed using SPSS Version 20.0. The result is significant if $p < 0.05$.

RESULTS AND DISCUSSION

Our study concerns 40 patients that met the inclusion criteria (22 females and 18 males). The age average is of 41.05 ± 18.8 years old. 55% of the patients are females with a 0.82 sex ratio. Seniority in haemodialysis is of 6.9 ± 6.17 years. 12.5% of the subjects are diabetics, 5% are smokers, and 45% are dyslipidemic.

Initial nephropathy was diabetic in 12.5% of cases, hypertensive in 12.5% of cases, segmental and focal hyalinosis in 2.5% of cases, lithiasic in 5% of cases, and undetermined in 60% of cases (figure 1).

The symptomatology exhibited 20% cases of dyspnea (8 patients), 12.5% cases of atypical chest pain (5 patients), 12.5% cases of palpitations, and 5% cases of oedema.

72.5% of cases had anaemia with an average hemoglobin level of 10 ± 1.7g/dl before dialysis, and of 11.37 ± 1.76 g/dl after dialysis. 70% of patients had secondary hyperparathyroidism with a 519 ± 577 UI average.

52.5% of our patients were hypertensive. The systolic blood pressure average was of 110.1 ± 18.9 mmhg pre-dialysis, and 100.4 ± 17.9 mmhg post-dialysis. The diastolic blood pressure average was of 71.7 ± 14.5 mmhg pre-dialysis, and 60.1 ± 14.2 mmhg post-dialysis. Vascular access was through arteriovenous fistula for all patients.

32.5% cases exhibited LVH while 65% of cases showed concentric LVH. For LVH patients, the interventricular septum thickness in diastole was averaging 12.92 ± 2.66 mm and 10.8 ± 2.5 mm before and after the haemodialysis session respectively. The posterior wall thickness in diastole was of 12.07 ± 2.2 mm before the haemodialysis session, and 10.2 ± 2.4 mm after it.

Other observed echocardiographic abnormalities were 22.5% cases of pulmonary hypertension with a 34.3 ± 9.37 mmhg average, 15% cases of dilated cardiomyopathy, 10% cases of valvular heart disease, and 10% cases (4 patients) of left ventricular systolic dysfunction with a left ventricular ejection fraction (LVEF)<50%. Aortic root calcifications with no repercussions were observed on 2 patients and 77.5% of cases exhibited elevated left ventricular filling pressures.

After statistical analysis, the LVH's predictive factors of occurrence (figure 2) were anemia (p=0.03), high blood pressure (p=0.03), hyperparathyroidism (p=0.05), dyslipidemia (p=0.007), and a long-standing haemodialysis exceeding 10 years (p=0.02) (table 1).

Discussion:

Chronic kidney disease (CKD) is a major public health issue due to its steadily increasing incidence. This disease affects more than one tenth of the general population, of whom 4 out of 100,000 reach the dialysis stage. [1]. It is estimated that more than 2 million people in the United States will be needing dialysis by 2030. Cardiovascular disease (CVD) is of great concern to nephrologists since it is a major cause of morbidity and mortality for CKD patients, affecting all stages of the disease even the earliest ones. Uremic patients are more at risk of dying from CVD than reaching the dialysis stage [4]. CV deaths are three to twenty times higher in dialysis patients than in the general population at the same age [5, 6]. A high prevalence of CV co-morbidity is observed at the onset of the replacement therapy and is predictive of the subsequent mortality in dialysis [5, 6]. Indeed, CVD develops well before dialysis since its risk factors are present at the initial stage of CKD.

The introduction to the ultrasonic echocardiogram and pulsed Doppler in CV exploration enabled a better understanding and analysis of uremic cardiomyopathies' pathophysiology. The main morphological abnormality in patients with kidney failure is left ventricular hypertrophy (LVH), which is present in 60% to 80% of patients starting dialysis [11]. LVH is of high prognostic value, it is an independent predictor of cardiac mortality [12]. For patients with renal insufficiency, it is most often associated with extensive myocardial fibrosis and can have deleterious clinical consequences : alteration of both diastolic and systolic functions (responsible for cardiac insufficiency), ventricular arrhythmia, and a particular susceptibility to myocardial ischemia.

Haemodialysis treatment for end-stage renal disease (ESRD) patients can have repercussions on cardiac functions due to acute changes in blood volume, blood pressure, electrolytes, and sympathetic vagal balance.

The age average is of 41.05 ± 18.8 years old in our set and 50,1 ± 12,3 years old in J. Vigan's set [7]. Hypertensive patients represented 52.2% in our set, 67.4% in J. Vigan's, and 42.1% in Amor's [8]. 45% cases in our study were dyslipidemic against 2.6% cases in Amor's. 72.5% cases were anemic in our study, along with 60.5% in Amor's [8] and 47.7% in McClellan's, which focused on

5222 CKD patients, against 1% in NHANES' which included pre-dialysis CKD patients [10].

The conventional Doppler echocardiography is a non invasive method for the morphological and functional cardiac study of kidney disease patients. This method coupled with tissue Doppler provides additional information on systolic and diastolic function of the left ventricle. The left ventricular hypertrophy (LVH) is the most common morphological abnormality [11]. It is clinically silent in the early stage but can have long-term complications by a decrease in left ventricular compliance (diastolic dysfunction), as well as by a disturbance in myocardial contractility (systolic dysfunction), or even by a heart failure. Consequently, it is an independent predictor of cardiac mortality [12]. LVH is an adaptive response to the increasing cardiac workload generated by a volume or pressure overload [13]. For CKD cases, volume overload can be due to arteriovenous fistula, hydrosodium retention, or anaemia [14]; while pressure overload can be caused by high blood pressure [15]. This adaptive response, although reversible at first, can lead to myocyte death with fibrosis (irreversible stage) [16]. This myocardial fibrosis can be aggravated by uremic toxins, vitamin deficiencies, and hyperparathyroidism which are specific features of uremia [17].

It is important to note that a regression of echocardiographic LVH is possible at the onset, before the fibrosis stage, which will lower the probability of heart failure and mortality risk [18]. Given that patients can have well established cardiac structure abnormalities at the beginning of the dialysis treatment, a screening, identification, and early correction of risk factors is required. [18]

LVH is an independent risk factor of cardiac mortality particularly for promoting the occurrence of paroxysmal ventricular arrhythmias.[19] It increases the risk of coronary insufficiency, stroke, and congestive heart failure regardless of BP levels [20]. LVH is found in 75% of ESRD cases and is a major risk factor of cardiac mortality and morbidity for those patients, irrespective of other risk factors such as age, high blood pressure, diabetes, dyslipidemia, and smoking [11]. LVH places uremic patients at high CV risk.

LVH prevalence is of 32.5% in our study, 54.6% in J.Vigan's [7], and 47.40% in Amor's [8]. Several spot observational studies showed a high prevalence of LVH in ESRD patients. Indeed, it was diagnosed by echocardiogram in 60% to 75% of patients starting dialysis [21], and in 60% to 90% of chronic dialysis patients [22]. Several studies showed that the echographic regression of LVH lowers the probability of heart failure in uremic patients. [23]

Anemia is frequent in CKD patients. In this population, it was shown that anemia alone increases the relative risk of CV events by a 1.5 coefficient [24]. Anemia induces a left ventricular hypertrophy. The combination of a LVH and anaemia increases the relative risk of CV events by 4 [25].

The relationship between HBP and kidney disease is complex [26]. On the one hand, HBP is the potential cause of CKD, to the extent that it is responsible for approximately 30% of ESRD cases [27, 28]. On the other hand, HBP is a common consequence of CKD: about 80% of CKD patients have HBP in the dialysis stage [29]. It should be noted that only a small percentage (0.5 – 1.3%) of the hypertensive population develop an ESRD [30], suggesting that genetic and environmental factors influence individual renal susceptibility to HBP. In the general population, HBP is strongly associated to different types of CV diseases [31]. For CKD patients, HBP is frequent and has an early manifestation in the natural history of chronic nephropathy. It is one of the most important causes of LVH and cardiac dysfunctions in those patients.

Observations showed the improvement of myocardial structure after parathyroïdectomy, which suggests that high concentrations of PTH has deleterious effects on the heart [32]. The relationship

between PTH levels and LVH was reported by some researchers with contradictory results. Nasri et al. [33] studied the effects of parathyroïdectomy on LVH in 24 patients with secondary hyperparathyroidism due to CKD, and in 7 patients with primary hyperparathyroidism. Measurements of interventricular septum, posterior wall thickness, left ventricular end-diastolic diameter, shortening fraction, ejection fraction, and LV mass index were taken before parathyroïdectomy and repeated 12 months after. Basal serum levels of PTH in secondary hyperparathyroidism patients (34.4 ± 13.7ng/ml) was significantly higher than in primary hyperparathyroidism patients (3.4 ± 1ng / ml, p <0,0001). Twelve months after parathyroïdectomy, the interventricular septum, posterior wall thickness, and LV mass index significantly decreased for secondary hyperparathyroidism patients. For primary hyperparathyroidism patients, all echocardiographic parameters stayed within the normal range and showed no significant changes after parathyroïdectomy. Haras et al. [34] investigated the left ventricular function for 46 chronic haemodialysis patients, and compared the cardiac function before and after parathyroïdectomy for 10 haemodialysis patients with secondary hyperparathyroidism. Cardiac dysfunctions were observed in 80.4% of patients. The study suggests that PTH serum levels above 200 pg/ml in long-term haemodialysis patients alters myocardial function, and induces cardiac hypertrophy and HBP. After parathyroïdectomy, an improvement of cardiac function and a decrease of LV mass was observed.

Levin et al. [14] reported a predominance of eccentric LVH by 65% in their pre-dialysis CKD population. The study showed a strong correlation between LVH and anaemia, which can explain the high incidence of eccentric LVH. Paoletti et al. [35] found a predominance of concentric LVH in 244 CKD patients, probably because the study found a strong relationship between LVH and pulse pressure, and did not identify anaemia as a LVH factor. In our set, 65% cases showed concentric LVH.

Concentric LVH patients are more at risk CV complications than eccentric LVH patients [36]. As a matter of fact, in the cohort of 433 Canadian patients in ESRD at pre-dialysis, the median survival was of 48 months in concentric LVH patients, 56 months in eccentric LVH patients, and more than 66 months in patients with a normal echocardiogram.

Krumholz et al. assessed the prognostic value of LVH types in the Framingham study and showed that the poorest prognosis was that of concentric LVH, followed by eccentric LVH, then by concentric remodelling [37]. Koren et al. showed that in the hypertensive population, concentric LVH patients had the poorest prognosis [38].

The major risk factors of LVH are high blood pressure, anaemia, hydrosodium overload, and arteriovenous fistula, which are affected by the haemodynamic alterations they cause. Two Canadian study by Foley and Coll showed that anaemia and HBP are major predictive factors of LVH :

Foley and Coll [39], every 10 mmHg increase of mean arterial pressure was associated to a 48% increase of LVH risk and development of heart failure.

Foley and Coll [40], every 1 g/dL decrease hemoglobin was associated with 46% risk of acute LVH.

Another study conducted by J. Vigan et al [7] concluded that LVH predictive factors were HBP (p=0.04), obesity (p=0.01), central venous catheter (p=0.03), and anaemia (p=0.02). Amor's statistical analysis [41] showed that anaemia, high blood pressure, and secondary hyperparathyroidism were significant risk factors of LVH occurrence, which is consistent with our results (table 2).

All authors declare the absence of a conflict of interest

CONCLUSION:

Our study shows a high LVH prevalence. The identified LVH risk factors are anaemia, high blood pressure, and hyperparathyroidism. A regular monitoring in chronic haemodialysis with a control of CV risk factors can prevent the occurrence of those complications. A cooperation of cardiologists and nephrologists is necessary in order to protect patients from all complications.

Left ventricular hypertrophy should be considered as a poor prognosis for kidney disease patients, accepted today as an independent risk factor of sudden death, ventricular arrhythmia, myocardial ischemia, and cardiac insufficiency. Hence the necessity of certain measures : screening and treatment of LVH before the fibrosis stage, LVH ultrasound screening should be a standard procedure in all CKD check-ups (unless contraindicated), generalizing angiotensin II receptor blockers treatment for all CKD patients with LVH. Early detection and correction of risk factors specific or not to uremia that combine optimal treatment in the initial stages of chronic kidney disease, High blood pressure, anemia, diabetes, hydrosodium overload, dyslipidemia, and phosphocalcic disorders.

A full ultrasound evaluation with assessment of systolic and diastolic functions (analysis of LV filling pressures) in haemodialysis patients with an elevated left ventricular pressure: avoid sudden changes in blood volume, determine an optimal dry weight, avoid brutal ultrafiltration (as well as short dialysis). Promote peritoneal dialysis, maintain sinus rhythm to ensure LV filling.

Cardioprotective therapy should be an essential component of CKD treatment, as well nephroprotective therapy. The complexity of integrated control combining both therapies justifies early and regular nephrological care of any CKD.

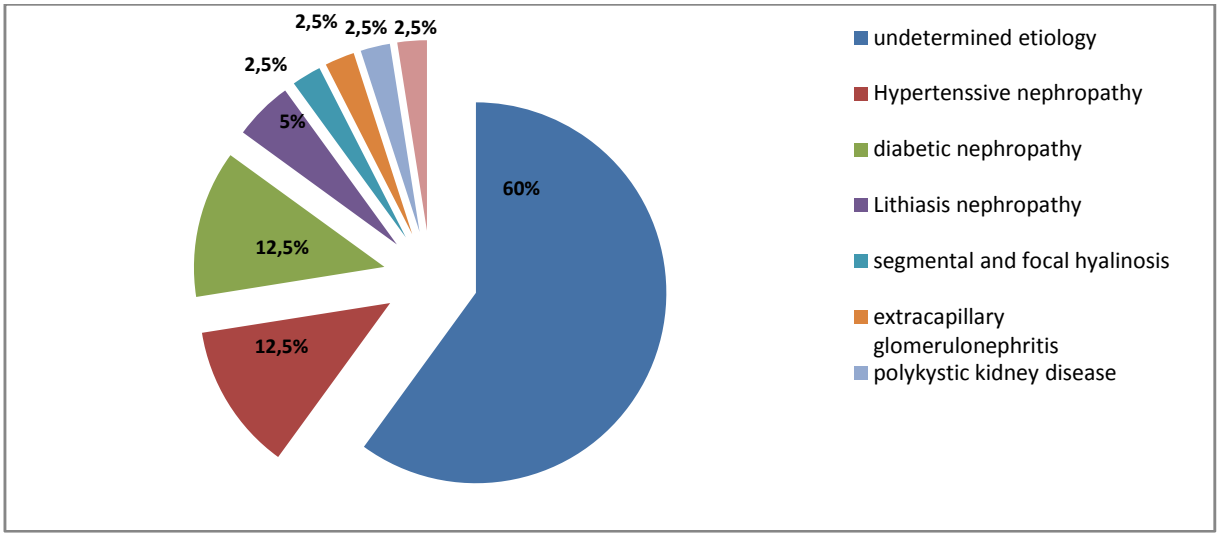

Figure 1 : etiology of renal failure

Table 1 : Predictive factors of LVH occurrence in chronic haemodialysis patients

Predictive factors	« p » value
HBP	0.03
Seniority in haemodialysis	0.02
Anaemia	0.03
Hyperparathyroidism	0.05
Dyslipidemia	0.007

Table 2 : Comparison of our study's results with other studies in the literature

	Seniority in Haemodialysis (months)	LVH	HBP	Anemia	Dyslipidemia	Hyperparathyroidism
KARIMI Oujda[42]	102,4±42	53	53	78	38,5	_
AMOR Tunisia[2]	65,8 ±47	47,4	42,1	60,5	2,6	_
BENZERDJEB Algiers[43]	_	59,8	69,3	48	24,5	50
Our set	82,8 ±74	32,5	45	72,5	45	70

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