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Arrhythmia and Chronic Kidney Disease

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ABSTRACT

Cardiac involvement is the leading cause of death in patients with chronic kidney disease (CKD), including cardiac arrhythmias that increase the risk of sudden death. The purpose of our work is to study the prevalence of heart complications in this population and to propose better cardiac and nephrological management. A total of 92 patients with CKD participated in the study, in the Department of Renal Dialysis and Renal Transplantation the Military Training Hospital Mohammed V in Rabat, the mean age of the patients was 50 years ± 16.55 with extremes ranging from 34 to 76 years, sex ratio was 1.1(H / F). Causal nephropathy is indeterminate in 48% of cases, 58% of our patients are hypertensive; 64% had cardiac arrhythmias; the electrical abnormalities were respectively: left ventricular hypertrophy in 41.4% of cases, left atrial hypertrophy in 9.2% of cases, atrial fibrillation in 5.5% of cases and repolarization disorders respectively in 12 % of cases.

Introduction

People with CKD have an increased burden from heart rhythm disorders, including atrial fibrillation (AF)/atrial flutter, supraventricular tachycardia, ventricular arrhythmias, and sudden cardiac death (SCD) relative to those without CKD (1). While treatment options, including drug and procedural therapies are available, their use in the setting of CKD is complex and limited. The risk for SCD is increased in patients with CKD, and for those with ESKD on dialysis, several factors that increase risk have been identified.

Evidence from older randomized trials indicates that pharmacological rhythm and rate control strategies are equivalent in terms of their efficacy on risks of heart failure, stroke, and survival for patients undergoing hemodialysis, both the potassium concentration in the dialysate and the schedule of hemodialysis treatments affect the risk of sudden death.

Patients and methods

We recruited 92 patients with CKD who participated in the study, 52 patients were on hemodialysis in the Department of Renal Dialysis and Renal Transplantation the Mohammed V Military Training Hospital in Rabat, clinical parameters (age, sex, medical history, smoking and alcohol consumption, blood pressure, temperature, physical signs..), biochemical (creatinine, urea, hemoglobin, fasting glucose, albumin, C-reactive protein, calcemia, phosphatemia, and PTHi 1-84) and dialytics for hemodialysis patients (frequency and duration of the dialysis session, dry weight, interdialytic weight gain, mean Kt / v, serum potassium before dialysis) were collected from the medical records of patients.

The diagnosis of these cardiac disorders were done by the electrocardiogram, the cardiac ultrasound, the stress test was carried out in 18 patients and the holter ECG was realized in 6 patients and we also realized a front chest x-ray for all patients.

The data were analyzed using SPSS software version 16.0, 2007. Quantitative variables were expressed as means ± standard deviation and qualitative variables as numbers and percentages.

The Student's t-test was used to compare the quantitative variables and the exact Chi 2 or Fisher test for the comparison of the qualitative variables. Multivariate analysis used multiple logistic regression. A value p <0.05 was considered significant.

Results

A total of 92 patients with CKD participated in the study, 52 patients were on hemodialysis in the department of Renal Dialysis and Renal Transplantation the Military Training Hospital in Rabat, the average age was 50 years ± 16.55 with extremes ranging from 34 to 76 years and the sex ratio H/F of 1.1. The physical signs are shown in table 1 respectively. Causal nephropathy was indeterminate in 48% of cases, diabetic nephropathy in 20% of patients respectively **(Table 1)**, we also identify demographic, anthropometric, laboratory and dialysis characteristics of the population study (**Table 2).**

Concerning radiological findings, the mean cardiothoracic index (ICT) was 0.58 ± 0.06 (range: 0.42-0.76). 65 patients had cardiomegaly at the left ventricular, while 18 patients had an ICT less than 0.5, and only 2 patients had a convex average left arc suggestive of pulmonary arterial hypertension.

58% of our patients are hypertensive; 64% had cardiac arrhythmias; the electrical abnormalities were respectively: left ventricular hypertrophy (LVH), left atrial hypertrophy, atrial fibrillation and repolarization disorders respectively in 45, 10, 6 and 13 patients.

20 patients had electric signs of ventricular extrasystoles on the ECG Holter.

Echo cardiographic abnormalities are shown in table 3.

For hemodialysis patients, the mean duration of hemodialysis was 60 ± 16.5 months, 12 hours a week with an arteriovenous fistula (FAV), 3 of whom had a complication such as hyper debit. In multivariate analysis, age, sex, interdialytic weight gain, arteriovenous fistula site, hyperparathyroidism, hypoalbuminemia, weekly dialysis and Kt / V were not associated with the LVH and cardiac arrythmia

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However, hypertension, diabete and anemia were identified as major determinants of LVH occurrence in hemodialysis patients (significant difference with respectively p <0.024 and 0.012). There was no statistically significant difference between the occurrence of repolarization disorders and anemia (p = 0.3), dyslipidemia (p = 0.8), hypertension (p = 0.21), diabetes (p = 0.64) and biological inflammatory syndrome (p = 0.3).

Cardiac valvular calcifications were found in 4 patients (3 of them were diabetic), there were no factors detected in their onset including age and seniority in hemodialysis.

For all patients, in multivariate analysis, age, sex, and hypertension were not associated with cardiac arrythmia. In contrast, anemia, atherosclerotic heart disease, left ventricular hypertrophy were identified as major determinants of arrhythmia occurrence in patients with CKD. cardiac valvular calcifications were found in 16 patients.

Table 1. Causal nephropathy and physical signs.

Initial nephropathy	
Unknown	48,2%
Glomerular	21%
Diabetic	20%
Vascular	10,53%
Tubulo interstitial	6%
Chronic tubulo interstitial nephropathy	4,8%
Physical signs	
Dyspnea	80%
Anemia	24%
Tachycardia	76%
Hypertension	58%
heart failure	38%

Table 2. Demographic, anthropometric, laboratory and dialysis characteristics of the population study.

	Results	**Percentage**
Age (years)	50 ±16,55	
Gender (% male)		52%
Body mass index (Kg/m2) 18,5 - 25 25 - 30 30 – 35		 22% 60% 15%
Dyslipidemia		34%
Median ESRD vintage (years)	4,2±16,55	
C-reactive protein (mg/l)	20±8,2	
Serum albumin (g/dl)	38±23	
Serum hemoglobin (g/dl)	8,9±10,1	
Phosphatemia(mg/l)	48±25	
Calcemia (mg/l)	79±31	

Table 3. Echo cardiographic abnormalities of study population.

Left ventricular hypertrophy	48%
Atrial hypertrophy	10%
Mitral insufficiency	27%
Aortic insufficiency	12%
Tricuspid insufficiency	1 %
Pulmonary arterial hypertension	2 %
Valvular calcifications	16%
Systolic dysfunction	6 %
Hypokinesia	5%
Pericardial effusion	9%

Discussion

Multiple cardiovascular risk factors, including atherosclerotic heart disease, left ventricular hypertrophy, and accelerated cardiac fibrosis appear to contribute to arrhythmia pathogenesis in ESRD (2). Although arrhythmia and ultimately sudden cardiac death can occur in patients (in the general population) with apparently structurally nor- mal hearts, most patients (particularly those with CKD) have underlying structural heart disease, and some type of acute event interacts with the underlying substrate to pro- duce the fatal arrhythmia (3,4). In patients with advanced CKD particularly those undergoing dialysis, myocardial ischemia is likely to be a contributor, but it is also plausible that myocardial ischemia (of the type mediated by epicardial coronary artery disease) may play a less predominant role and that other factors, such as inflammation and autonomic imbalance or increased sympathetic activity (including sleep apnea), may be important contributors to sudden cardiac death (5,6).

Hypertrophic myocardium is predisposed to both atrial and ventricular arrhythmia through the induction of prolonged action potentials and increased repolarization defects in areas of ischemia, with underling fibrosis serving as a favorable substrate for propagation of arrhythmia. While hypertension, diabetes, and other typical risk factors undoubtedly contribute (7). In our study, the average rate of LVH was lower (45%) comparing to other studies; Bah A. O. et al. in 2006 in the Republic of Guinea (72.95%) (8) and A. Aldlouni et al. in 2011 in Morocco (87%) (9). Although asystole or ventricular arrhythmias are the most likely types of arrhythmia to result in sudden death, atrial arrhythmias, particularly AF, may result in significant morbidity in patients with ESRD. AF is increasingly common in HD patients. In a study of 258,605 older participants, the AF incidence in incident dialysis patients was 14.8/100 person-years, and adjusted probabilities of developing AF during the first year of dialysis increased from 11.3% in 1995 to 14.3% in 2007 (10). Similarly, the prevalence of AF (diagnosed from administrative claims) was 10.7% in 2006—a three-fold increase from 1992 (11). Finally, the overall AF prevalence in a meta-analysis of 25 dialysis studies was 11.6% (12)

The proportion of repolarization disorders was found in 64% of our patients. In the literature, as reported by B. CHARRA (13), the high prevalence of left ventricular hypertrophy, high blood pressure and diabetes in hemodialysis is the cause of often silent coronary disease. and associated with nonspecific repolarization disorders on the resting ECG. This confirms our study in which we have established a significant correlation between LVH and the occurrence of repolarization disorders. However, there was no correlation between repolarization disorders and diabetes, as well as with hypertension. This finding goes against the results of other studies that looked for cardiovascular risk factors in hemodialysis. In particular, TAKEDA and al. (14) in Japan demonstrated that the risk of new cardiovascular events was strongly related to hypertension (p <0.0005).

In 48% of our patients, the cardiac ultrasound revealed an LVH, which could be the cause of these disorders of conduction. This theory is comforted by JUNGERS P. (15) who showed in his series that LVH and calcifications of the atrioventricular junction were the main factors responsible for conduction disorders.

In our serie, valvular calcifications were found in 16% of cases, a correlation was found between age, duration of hemodialysis and the presence of valvular calcifications in the literature (16).

No correlation was found between valvular calcifications in our patients and seniority in dialysis.

In the absence of trial data, the results from observational studies on the efficacy and safety of anticoagulation for stroke prevention in CKD patients with eCrCl < 30 mL/min not on dialysis are conflicting as they are for CKD G5D (17)

There is insufficient high quality evidence to recommend warfarin for prevention of stroke in CKD G5D patients with AF, especially when There is insufficient high quality evidence to recommend warfarin or other vita- min K antagonists (VKAs) for prevention of stroke in CKD G5D patients with AF, especially when balancing the significant risks of bleeding, accelerated vascular calcification and calcific uremic arteriopathy associated with VKAs therapy (18).

Conclusion

The kidney has numerous complex interactions with the heart, including shared risk factors (hypertension, dyslipidemia, etc.) and mutual amplification of morbidity and mortality. Both cardiovascular diseases and chronic kidney disease (CKD) may cause various alterations in cardiovascular system, metabolic homeostasis and autonomic nervous system that may facilitate the occurrence of cardiac arrhythmias. Also, pre-existent or incident cardiac arrhythmias such as atrial fibrillation (AF) may accelerate the progression of CKD.

Patients with CKD may experience various cardiac rhythm disturbances including sudden cardiac death. Contemporary management of cardiac arrhythmias includes the use of antiarrhythmic drugs (AADs), catheter ablation and cardiac implantable electronic devices (CIEDs). Moreover, CKD itself can induce profound alterations in the pharmacokinetics and pharmacodynamics of many drugs including AADs, thus facilitating the drug accumulation and increased exposure.

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