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End-Stage Chronic Kidney Disease and Heart Failure with Reduced Ejection Fraction: A Bad Association?

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Abstract

Background: The prevalence of congestive heart failure increases greatly as the patient's renal function deteriorates, and, at end-stage chronic kidney disease, can reach 65-70%. There is evidence that chronic kidney disease is a major contributor to severe cardiac damage. The aim of the study is to determine the impact of end-stage chronic kidney disease in patients with heart failure and reduced ejection fraction in a Moroccan population.

Results: We collected 3412 patients, 439 (12%) in group A (with end-stage chronic kidney disease) and 2973 (82%) in group B (without end-stage chronic kidney disease). Main etiology of heart failure was ischemic heart disease in both groups. Hypertension and dyslipidemia were more prevalent in group A. Diabetes was as prevalent in 2 groups. Comorbidities were more prevalent in group A as we observe more strokes (20% vs 8%, p=0,0001) and myocardial infarctions (31% vs 28%, p=0,0001). Group A patients were more symptomatic: stage III and IV New York Heart Association in 30% vs 19%, p=0,0001. Atrial fibrillation was more prevalent in group A (14% vs 9%, p=0,0001). Elevated left ventricle filling pressure was more prevalent in group A (40% vs 34%, p=0,003). Mean LVEF was 35,69 +/- 12,56 % in group A vs 33,46 +/- 10,42 % in group B (p=0,63). Patients of group A were more at risk of heart failure hospitalization (21% vs 9%, p=0,005).

Conclusions: Cooperation between nephrologists and cardiologists may improve quality of care and subsequent prognosis for heart failure and chronic kidney disease.

Keywords: Heart failure with reduced ejection fraction; End-stage chronic kidney disease; Heart failure hospitalization; Stroke; Myocardial infarction

Abreviations: HFrEF: Heart failure with reduced ejection fraction; HF: Heart failure; CKD: Chronic kidney disease; eGFR: estimated glomerular filtration rate; NYHA: New York Heart Association; LVEF: Left ventricular ejection fraction; KDIGO: Kidney Disease Improving Global Outcomes; CVD : Cardiovascular disease

Background

The prevalence of congestive heart failure increases greatly as the patient's renal function deteriorates, and, at end-stage chronic kidney disease, can reach 65-70%. Heart failure with reduced ejection fraction (HFrEF) is a leading cause of morbidity and mortality in patients with chronic kidney disease (CKD), and the population of CKD patients with concurrent heart failure HF continues to grow. HFrEF is found in about one-quarter of cases of chronic kidney disease [1]. The prevalence of HFrEF increases

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greatly as the patient's renal function deteriorates, and, at end-stage renal disease, can reach 65-70% [2]. The pathophysiology between the heart and the kidneys is complex and bidirectional. Patients with CKD have greater prevalence of traditional HFrEF risk factors as well as unique kidney-specific risk factors. Control of the anemia and aggressive use of the recommended medication for HFrFE may improve the cardiac function, patient function and exercise capacity, stabilize the renal function, reduce hospitalization and improve quality of life [3]. There is mounting evidence that CKD itself is a major contributor to severe cardiac damage [1-3]. The aim of the study is to determine the impact of end-stage chronic kidney disease in patients with heart failure and reduced ejection fraction in a Moroccan population.

Methods

It is a transversal retrospective study conducted between May 2006 and June 2019 including all Moroccan patients beyond the age of 14 with HFrEF followed-up in the therapeutic unit of heart failure of our department. Data were collected on Excel and statistical analysis was made using IBM SPSS statistics 2.0 software. We define 2 groups of patients. Group A with end-stage CKD (defined as a reduction of estimated glomerular filtration rate (eGFR) to an absolute value of less than 15ml/m²/1.73m² body surface area) and group B without end-stage CKD. We studied demographic, clinical and paraclinical characteristics among these 2 groups of patients.

Results

We collected 3412 patients, 439 (12%) in group A and 2973 (82%) in group B.

We observed male predominance in both groups (63%). Mean age was 65,58 +/- 13,46 in group A and 64,82 +/- 12,74 in group B with no statistical difference (p=0,24).

The etiology of HFrEF was dilated cardiomyopathy in 12% in group A vs 13% in group B, ischemic heart disease in 74% in group A vs 77% in group B, valvular heart disease in 9% in group A vs 4% in group B, toxic heart disease in 2% in group A vs 2% in group B (p=0,018).

Cardiovascular risk factors: Hypertension (47% vs 37%, p=0,0001) and dyslipidemia (13% vs 9%, p=0,015) were more prevalent in group A while smoking (30% vs 32%, p=0,0001) was more prevalent in group B. Diabetes (30% in both groups, p=0,445) was as prevalent in both groups.

Comorbidities were more prevalent in group A as we observe more strokes (20% vs 8%, p=0,0001) and myocardial infarctions (31% vs 28%, p=0,0001).

Group A patients were more symptomatic: stage III and IV New York Heart Association (NYHA) in 30% vs 19%, p=0,0001, stage I and II NYHA in 70% vs 81%, p=0,0001).

Mean heart rate was 78,74 +/- 17,64 bpm in group A vs 76,89+/- 16,32 bpm in group B (p=0,030). There was no statistical difference concerning blood pressure control in both groups.

Electrocardiogram: Atrial fibrillation was more prevalent in group A (14% vs 9%, p=0,0001).

Transthoracic echocardiography: Elevated left ventricle filling pressure was more prevalent in group A (40% vs 34%, p=0,003). Mean left ventricular ejection fraction (LVEF) was 35,69 +/- 12,56 % in group A vs 33,46 +/- 10,42 % in group B (p=0,63). Left ventricle was more dilated in group A (left ventricle end-diastolic diameter of 59,24 +/- 8,2 mm vs 57,91 +/- 9,31 mm, p=0,020).

Outcomes: Patients of group A were more at risk of HF hospitalization (21% vs 9%, p=0,005).

Discussion

The Kidney Disease Improving Global Outcomes (KDIGO) defines CKD as abnormalities of kidney structure or function present for more than 3 months with health implications [4]. CKD globally affects 15-20% of adults [5], but the real prevalence of CKD is way more important, as it is estimated that it affects nearly 40% of adults who have severe kidney disease [6].

Kidney disease and Cardiovascular Disease (CVD) are intimately connected and the pathophysiology between the heart and the kidneys is complex, bidirectional and involves many different pathways [7]. The cardio-renal syndrome is a disorder that implicates both of the heart and kidneys, in which the acute or chronic dysfunction of one organ can lead to the acute or chronic dysfunction of the other [8].

CKD adds a certain number of additional cardiovascular risk factors including albuminuria, systemic inflammation, anemia, hypervolemia, increased oxidative stress, left ventricular hypertrophy, and toxic metabolites [9]. All of these cardiovascular risk factors provide a fertile ground for the development of CVD and HF [10]. On the other hand, HF may worsen CKD by lowering renal perfusion, leading to renal venous congestion and the activation of both sympathetic nervous and renin–angiotensin–aldosterone systems, increase of the inflammation and oxidative stress, resulting in the aggravation of CVD and HF [11]. In our study, hypertension and smoking were more prevalent in the group A (with CKD) than in the group B (without CKD), while diabetes and dyslipidemia were as prevalent in both of the groups.

An observational echocardiographic study reported that increased left ventricular hypertrophy leads to a decrease in eGFR ranging from around 32% in patients with eGFR ≥60 ml/min/1.73 m² to 75% in patients with eGFR <30 ml/min/1.73 m²) [11]. On the other hand, in large HF registries,

20-68% of patients who have HF were found to have concomitant moderate to severe renal disease [12].

Diagnosis of HF and CKD is based on clinical functional signs and symptoms, as well as structural and/or functional cardiac and renal abnormalities. Blood biomarkers also contribute to diagnosis by providing additional information on possible cardiac and renal lesions, and also have a prognostic and predictive role [13].

In the results of our study, we have highlighted the fact that strokes, myocardial infarction, atrial fibrillation and elevated filling pressures were more prevalent in the group with end-stage CKD than in the group without end-stage CKD with a statistically significant difference ($p<0.05$). Moreover, patients in the group A (with end-stage CKD) were more symptomatic and at a higher risk of acute decompensated HF with a more dilated left ventricle, once again with a statistically significant difference.

The main aims of HF treatment in all patients, whether with or without CKD, are to decrease both preload and afterload, reduce left ventricular hypertrophy, treat myocardial ischemia and inhibit neurohumoral hyperactivity, especially the sympathetic nervous and renin-angiotensin-aldosterone systems. These objectives, particularly in HF with reduced ejection fraction, can be achieved mainly with renin-angiotensin system inhibitors, anti-aldosterone drugs, beta-blockers and SGLT2 (sodium – glucose cotransporter 2) inhibitors, in line with ESC guidelines [14].

The management of HF in patients with CKD can be particularly challenging and complex, given that these patients tend to be older, more vulnerable and often have numerous associated comorbidities, and the optimal medical treatment used in HF can potentially harm the kidneys and degrade renal function acutely or chronically [15].

The close association between HF and CKD worsens patients' prognosis. According to our study, the presence of end-stage CKD increases the risque of HF hospitalization. The 2016 United States Renal Data System (USRDS) Annual Report describes that the prevalence of congestive hypertension in CKD patients aged 66 years and more was 28%, compared with barely around 6% in non-CKD patients [16]. Hakopian et al. [16] demonstrated in their study that CKD stages 4 to 5 were associated with a significant risk of all-cause hospitalization, HF hospitalization, rehospitalization and mortality in HF patients.

Therefore, and in the light of these results, we can assume that CKD, and especially end-stage, is indeed a factor worsening heart failure symptoms, increasing the risk of myocardial infarction and stroke and increasing the rate of HF hospitalization. The results and conclusions of our study are in line with those of larger studies in the literature.

Conclusions

Cooperation between nephrologists and other physicians in the treatment of patients with HFrEF may improve the quality of care and the subsequent prognosis for both heart failure and chronic kidney disease. More targeted HF therapies may improve outcomes in patients with kidney disease as current HF therapies are underutilized in this population. Further work is also needed to develop novel HF therapies for the CKD population.

Declarations:

Ethics approval and consent to participate:

All participants were included after written consent signed by the participants. It was conducted ethically in accordance with the World Medical Association Declaration of Helsinki, 1975. The Ethical Committee of Faculty of Medicine, Hassan II University approved the study.

Consent for publication:

Not applicable

Availability of data and material:

The manuscript data is available on request to the corresponding author.

Competing interests:

The author declares that there are no competing interests.

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