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Cardiorenal Syndrome Type II with Heart Failure with Preserved Ejection Fraction

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Abstract

Cardiorenal syndrome type 2 (CRS2) is characterized by chronic abnormalities in cardiac function leading to kidney injury or dysfunction. The incidence of heart failure with preserved ejection fraction (HFPEF) is reported to include about 50% of the general heart failure population, while the prevalence of HFPEF is still increasing over the last years when compared to the prevalence of heart failure with reduced ejection fraction. Its prevalence is higher in the elderly especially in females; in a recent study of HFPEF, all patients were aged > 80 years, with a mean age of 87. This is Mr A.P, aged 84, referred to the emergency room by his attending physician for a flare-up of acute renal failure on a chronic basis and cardiopulmonary decompensation. His background: in 2020, chronic kidney failure at stage IV on probable nephroangiosclerosis with a baseline serum creatinine at 183 µmol/L or a glomerular filtration rate (GFR) according to Chronic Kidney Disease-epidemiology (CKD-EPI) at 27 ml/min in 2020, proteinuria at 0.36 g/24H, no hematuria. Clinical examination found a weight gain of 4 kg with a usual weight of 68 kg, BMI: 21.46 kg/m^2. A BP: 89/52mmHg, Pulse: 65 b/min, saturation at 90% AA, it has three FRIED criteria of fragility (walking speed, reduction in muscular strength and involuntary weight loss over one year); edema of the lower limbs, respiratory distress with bilateral pleurisy. His electrocardiogram was unchanged, Troponin: 120 pg/ml, BNP: 919 ng/l; serum creatinine: 316 µmol/L, serum potassium: 5.8 mmol/L, proteinuria at 0.38 g/24hours, hematuria at 12 hties/mm^3. Transthoracic ultrasound shows a congestive heart, IVC: 26 mm, LV dysfunction, no hypokinesia, ejection fraction (EF) at 50%, dilated OG and CD. His usual treatment is Seretide 250, 2 doses/day, PREVISCAN 2 mg/day, ENTRESTO 24/26mg, 2 times/day, FUROSEMIDE 125 mg/day, Allopurinol 100 mg/day, SERESTA 5 mg/day at bedtime and INEXIUM 40 mg/day. The emergency action was the cessation of ENTRESTO, oxygeno-

therapy, increase of FUROSEMIDE to 1 g IVSE/24H. The evolution is marked by the increase in acute renal failure with a Creatinine level of 400 μmol/L, an oliguria of 300 ml, a uremia of 60 mmol/L leading to extra-renal purification with the prescription of isolated daily ultrafiltrations for a week. Then dialytic frequencies were reduced to two per week in the face of clinical improvement marked by his oxygen withdrawal, stabilization of his renal function, regression of excess weight from 68 kg to 58 kg with reduction in BNP to 400 ng/l compared to 2000 ng/l. However, the patient remains dependent on dialysis and his loss of autonomy has increased in connection with a fracture of the neck of the femur on his right hip prosthesis and pulmonary embolism reinforcing his fragility. The coexistence of renal impairment in heart failure with preserved ejection fraction (CRS type 2 and 4) is common especially in older females with hypertension and/or diabetes. The management of this syndrome requires cardio-nephrological collaboration and characterization of patients and their prognosis.

Subject Areas

Nephrology

Keywords

Cardiorenal Syndrome Type 2, Chronic Kidney Disease, Preserved Ejection Fraction

1. Introduction

Cardiorenal syndrome (CRS) is defined as a complex pathophysiological disorder of the heart and kidneys in which acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ. All subtypes are associated with increased mortality and morbidity, with a significant impact on health resource utilization. Cardiorenal syndrome type 2 (CRS2) is characterized by chronic abnormalities in cardiac function leading to kidney injury or dysfunction [1] [2].

Heart failure is a major public health problem, approximately 2.3% of French people suffer from heart failure and up to 10% of people aged 70 and over. It is 5.8 million people in the USA and more than 23 million worldwide [3]. The incidence of HFPEF is reported to include about 50% of the general heart failure population [4], while the prevalence of HFPEF is still increasing over the last years when compared to the prevalence of heart failure with reduced ejection fraction (HFREF) [5]. Its prevalence is higher in the elderly [6] [7] especially in females) in a recent study of HFPEF, all patients were aged > 80 yrs, with a mean age of 87 [8]. There is a strong association between HF and worsening renal function. A recent meta-analysis showed that among all subgroups of patients with HF, the presence of impaired renal function was significantly associated with mortality and had even greater prognostic significance among those with

HFPEF [9]. Various studies have shown that HF, including HFPEF, is associated with worsening renal function [10] [11] with right ventricular (RV) dysfunction and elevated right atrial (RA) pressures being implicated in the pathophysiology of progressive cardiorenal syndrome (CRS) [12] [13]. Identifying the onset or progression of SCR is essential for good management and can slow progression and prolong survival in these patients suffering from SCR with HF FEP [14]. So far there are no specific data on the diagnostic interventions in patients with cardiorenal syndrome and HFPEF. The diagnosis is limited to traditional markers, serum creatinine to assess GFR and albuminuria. Though current research has been focusing on identifying markers that would permit an earlier or more accurate diagnosis of cardiorenal syndrome, no factor is specific for patients with HFPEF and CRS. B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-pro-BNP) levels [15], which are general markers of HF, can be evaluated when HF diagnosis is not certain. Plasma levels of BNP or Ntpro-BNP increase with left ventricular mass, wall stress, and filling pressures. To date, baseline levels of NT-pro-BNP of 339 and 409 pg/mL have been reported in patients with HFPEF, higher than in normal subjects but less elevated than usually observed in decompensated HF with low EF. However the relationship between BNP, renal function, and the severity of heart failure is less clear [9], not only for diagnostic purposes, but also for the management of therapy [16]. The most recent HF guidelines propose a revised algorithm for the treatment of HFREF, with the "quadruple therapy" approach with the use of Sodium-glucose cotransporter 2 inhibitors (SGLT-2), angiotensin receptor blocker neprilysin inhibitors (ARNI) as a replacement of angiotensin-converting enzyme inhibitors (ACE-I) and angiotensin receptor blockers (ARBs) or in de novo HFREF patients with class of recommendation IIb), on top on B-blockers, and mineralocorticoid receptor antagonists (MRAs), with a substantial improvement in clinical outcomes in terms of hospitalization and mortality [17]. A better understanding of this syndrome is therefore necessary before considering targeted therapies to improve the prognosis and especially the quality of life of patients with HFPEF. The objective of this work is to show our therapeutic approach in treating SCR type 2 with HFPEF.

2. Case Reported

This is Mr A.P, aged 84, referred to the emergency room by his attending physician for a flare-up of acute renal failure on a chronic basis and cardiopulmonary decompensation. His background: in 2020, chronic kidney failure at stage IV on probable nephroangiosclerosis with a baseline serum creatinine at 183 µmol/L or a GFR according to CKD-EPI at 27 ml/min in 2020, proteinuria at 0.36 g/24H, no hematuria, the immunological assessment shows dysimmunity with antinuclear Ab positive at 640, Anti desoxyribonucleic acid antibody (Anti DNA) and anti-extractable nuclear antigen (Anti ENA) negative, anti Neutrophil Cytoplasmic Antibody (ANCA) positive at 80 with myeloperoxidase (MPO) and serine protease 3 negatives; non-Embologenic atrial fibrillation under PREVISCAN;

Renal ultrasound shows that the Right Kidney measures 96 mm and the Left Kidney measures 101 mm, well differentiated, with regular contours; post-hypertensive heart disease with left ventricular hypertrophy, ejection fraction of 55%; wearer of a pace maker for rhythmic atrial disease; chronic interstitial lung disease with fibrosis including functional assessment showing a restrictive ventilatory syndrome, the CO_2 transfer test slightly decreased to 17%; former smoker at 15 PA quit in 1987. Clinical examination found a weight gain of 4 kg with a usual weight of 68 kg, BMI: 21.46 kg/m^2. A BP: 89/52mmHg, Pulse: 65 b/min, saturation at 90% AA, it has three FRIED criteria of fragility (walking speed, reduction in muscular strength and involuntary weight loss over one year); edema of the lower limbs, respiratory distress with bilateral pleurisy. His electrocardiogram was unchanged, Troponin: 120 pg/ml, BNP: 919 ng/l; serum creatinine: 316 µmol/L, serum potassium: 5.8 mmol/L, proteinuria at 0.38 g/24 hours, hematuria at 12 hties/mm^3. Transthoracic ultrasound shows a congestive heart, IVC: 26 mm, LV dysfunction, no hypokinesia, ejection fraction (EF) at 50%, dilated OG and CD. His usual treatment is Seretide 250, 2 doses/day, PREVISCAN 2 mg/day, ENTRESTO 24/26mg, 2 times/day, FUROSEMIDE 125 mg/day, Allopurinol 100 mg/day, SERESTA 5 mg/day at bedtime and INEXIUM 40 mg/day. The emergency action was the cessation of ENTRESTO, oxygenotherapy, increase of FUROSEMIDE to 1 g IVSE/24H. The evolution is marked by the increase in acute renal failure with a Creatinine level of 400 µmol/L, an oliguria of 300 ml, a uremia of 60 mmol/L leading to extra-renal purification with the prescription of isolated daily ultrafiltrations for a week. Then reduction in dialytic frequencies to two per week in the face of clinical improvement marked by his oxygen withdrawal, stabilization of his renal function, regression of excess weight from 68 kg to 58 kg with reduction in BNP to 400 ng/l compared to 2000 ng/l. However, the patient remains dependent on dialysis and his loss of autonomy has increased in connection with a fracture of the neck of the femur on his right hip prosthesis and pulmonary embolism reinforcing his fragility.

3. Discussion

HFPEF is a phenotypically heterogeneous syndrome, [18] [19] a constellation of symptoms of exercise intolerance, exertional dyspnea, volume overload, and renal dysfunction that are a consequence of abnormal cardiac structure and mechanics resulting in elevated filling pressures. HFPEF comprises about 50% [20] of the cases of acute and chronic heart failure. Multiple comorbidities predispose to HFPEF, and renal dysfunction and HFPEF frequently coexist.

The Diagnostic and Therapeutic Interventions for Cardiorenal Syndrome in HFPEF. So far there are no specific data on the diagnostic interventions in patients with cardiorenal syndrome and HFPEF. Identifying the onset or progression of cardiorenal syndrome is paramount to proper management and can result in disease attenuation and prolonged survival both in patients with preserved EF and in those with reduced EF [21]. Though current research has been focusing on identifying markers that would permit an earlier or more accurate

diagnosis of cardiorenal syndrome, no factor is specific for patients with HFPEF and CRS.

All drugs used in HF patients have potentially detrimental effects on the renal function, and they expose HF patients with renal dysfunction to a greater risk of adverse renal complications, such as hyperkalemia and dialysis. Historically, data from randomizedcontrolled trials on the effect of HF medications in HF patients and CKD were limited, due to the exclusion of patients with CKD. The studies of left ventricular dysfunction (SOLVD) trial enrolled 36% of patients with CKD and eGFR < 60 mL/min/1.73m^2; 33% of all patients presented a >0.5 mg/dL increase in serum creatinine; in the final analyses, the benefits on all-cause mortality were maintained across the entire CKD spectrum [22]. Several clinical trials in the management of cardiorenal syndrome including the study use irbesartan in patients with heart failure and preserved ejection fraction. They rolled up 4128 patients who were at least 60 years of age and had New York Heart Association class II, III, or IV heart failure and an ejection fraction of at least 45% and randomly assigned them to receive 300 mg of irbesartan or placebo per day. Renal failure was found in 6.4%. [23] In the clinical TOPCAT (Spirolactone or placebo) with 3445 patients the Treatment with spironolactone was associated with increased serum creatinine levels and a doubling of the rate of hyperkalemia (18.7%, vs. 9.1% in the placebo group [24]. However in the PARADIGM study in 2014 (Sacubitril/Valsartan or ENALAPRIL only) they found a significant reduction in the number of hospitalizations or cardiovascular mortality [25].

These results were similar in the Paramount study using the same treatment in 2015 with reduction in renal function decline [26].

In the case of preserved HF-LVEF, the study shows a NON-SIGNIFICANT reduction of 13% in the primary composite endpoint (hospitalization for heart failure and cardiovascular death): RR 0.87, 95% CI 0.753 - 1.005, p = 0.0585 The Sacubitril/Valsartan combination appears to be beneficial in patients with moderately impaired LVEF (45% - 57%) and in women Safety and efficacy data are similar to those observed in PARADIGM-HF. Nevertheless, there was a significant improvement in the NYHA functional class and the lesser appearance of renal insufficiency at 8 months of follow-up in the Sacubitril/Valsartan group [27].

In clinical practice, as demonstrated in several trials, the initiation of SGLT-2 inhibitors was associated with an initially mild drop of eGFR over the first weeks. This decrease in eGFR was reversible, and the renal function gradually returned to its baseline levels, with a stabilization of the renal function during the follow-up [28] [29].

Renal dysfunction and resistance to diuretics are often associated with salt and water overload, making congestion difficult to control, thus worsening the prognosis [30] [31]. At this level, the therapeutic options remain rare and limited.

Among them, peritoneal dialysis (PD) was first used in 1949 and gained in its

interest gradually over the last twenty years. Different clinical cases, small series, often retrospective studies have reported favorable outcomes with peritoneal dialysis in patients with different types of cardiomyopathy responsible for congestive heart failure with preserved or reduced ejection fraction [32]. In our clinical case, hemodialysis was the chosen technique used to treat not only cardiac congestion but also the correction of uremic syndrome with a rather favorable result and this is proven in the literature [33].

4. Conclusion

In CRS2, the HF temporally precedes the occurrence or progression of CKD, and the manifestation and degree of kidney disease is plausibly explained by the underlying heart condition. Clinical trials recruiting these patients are still lacking, unlike the SCR in HFREF. The therapeutic strategy, although difficult, is far from being completely elucidated. Hemodialysis constitutes an accessible and effective treatment among the therapeutic options in patients with heart failure refractory to optimal drug treatment. The most impressive results are observed in the improvement of morbidity with a very significant reduction in hospitalizations, improvement in the patient's functional state, quality of life and possible improvement in prognosis.

Conflicts of Interest

The authors declare no conflicts of interest.

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