SEMINARS IN
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# Temporary Dialysis Treatments for Heart Failure in Chronic Kidney Disease

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**Patients with cardiac disease and chronic kidney disease are admitted to our emergency unit with signs and symptoms of severe heart failure more and more frequently. Resistance to high-dose loop diuretics imposes the use of renal replacement therapy. We treated a group of these patients with personalized bicarbonate dialysis, deciding the number and frequency of treatment sessions according to the patient's clinical conditions. Heart failure can be classified as mainly diastolic or systolic. Results show that bicarbonate dialysis is effective and well tolerated, primarily in the treatment of patients with prevalently diastolic heart failure. Patients with prevalently systolic heart failure have a worse prognosis.**


**KEYWORDS** CKD, heart failure, bicarbonate dialysis, systolic heart failure, diastolic heart failure

The presence of both heart and kidney disease in the same patient is an ever more frequent occurrence in nephrology, internal medicine, and cardiology divisions. Surely, one of the most important causes is represented by the increase in the average age of the population, and therefore the increase in the incidence of vascular kidney pathologies. These pathologies still are underestimated in their frequency because of scarce availability of markers and low sensitivity of instrumental methods, as instead occurs in the cardiologic field.

Already in 1995, Fooley et al,[1] studying 433 patients who started intermittent hemodialytic treatment 2 years before, verified that 31% of them had heart failure. The physiopathology and clinical picture of heart failure involves principally the heart, the kidney, and the lung axis. Many complicated neurohormonal systems influence the reciprocal bonds between the 3 organs, but probably the most important effect is hydrosaline retention.[2] For a long period of time, hydrosaline retention from heart failure can be well controlled by using loop diuretics, alone or combined with other diuretics. With time, the same compensatory mechanisms generate a suicide effect,[3] favored sometimes by so-called provoking events. Often, in these cases, and especially if renal failure already was present, the kidney is unable to respond to higher doses of diuretics. Therefore, to reduce fluid accumulation and the electrolytic and acid-base imbalances, it is necessary to prescribe renal replacement hemodialysis treatment.

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## Patients and Methods

Twenty-four patients were studied (18 men and 6 women) who arrived in our ward in the 2 years after January 2001. All patients were affected by heart failure, with various degrees of chronic kidney disease (CKD) antecedent to the cardiac disease. All patients were oligoanuric and showed resistance to high doses of loop diuretics. After having diagnosed refractory heart failure, a standard bicarbonate dialysis treatment was started. For all patients, vascular access was a monolumen cannula placed in the inferior vena cava, inserted via the femoral vein. If it was not possible to find an adequate vascular access for blood re-entry, a double-lumen cannula was used. The filter used was low-flux polyamide 1.4 $m^2$ or 1.7 $m^2$ (GAMBRO Dialysatoren GmbH & Co, Hechingen, Germany), according to the patient's weight. Dialysate was personalized according to electrolytic and acid base imbalances ($Na^+$, $K^+$, $Ca^{2+}$, $Mg^{2+}$, $HCO_3$). Blood flow was 200 mL/m, dialysate flow was 500 mL/min. The ultra-filtration was programmed for a maximal gradual decrease of 500 mL/h. Treatment duration was decided according to the patient's gradual weight loss, which was not supposed to be more than 1,500 mL per session. Treatment frequency was based on patient clinical conditions, considering primarily the presence of orthopnea and dyspnea. Treatments were interrupted when a diuresis en-

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**Table 1** The European Society of Cardiology Criteria for Isolated Diastolic Heart Failure

| | |
|---|---|
| Signs and symptoms of heart failure | Dyspnea from effort, Orthopnea, III–IV tone, lung rattle |
| Normal systolic or slightly reduced function | FE ≥ 45% and DTDI > 3.2 cm. M-2 |
| Evidence of anomalies of relaxation/refilling/distensibility | IVRT <30 years old > 92 ms, 30 to 50 years old > 100 ms, >50 years old 1 > 105 ms |
| | E/A < 1 + DT > 220 ms + S/D < 1.5 <50 years old |
| | E/A < 0.5 + DT > 280 ms + S/D > 2.5 >50 years old |

FE, ejection fraction; DTDi, telediastolic diameter indicator; IVRT, isovolumetric relaxation time; E/A, ratio between the range of E and A waves; DT, deceleration time; S/D, ratio between systolic and diastolic range at lung venous level.

sued, exceeding 800 mL within 24 hours, with plasma electrolyte values and acid-base balance almost normalized. For each patient, we tried from the start to identify the renal pathology that caused the renal failure, the level of pre-existent renal failure, and creatinine values before hemodialytic treatments. At the same time, a scrupulous investigation was performed. Most importantly, an anamnestic study was performed that aimed to identify old ischemic pathologies and episodes of arrhythmia. The cardiologic study then was completed with a daily electrocardiogram, and echo Dopplers to estimate the ejection fraction, Doppler waves E and A, and eventual pericardiac effusions. The European Society of Cardiology criteria were used (Table 1)[4] to estimate diastolic failure and to classify patients as affected mainly by diastolic or systolic failure.

For each patient we looked for eventual factors that could have provoked the acute crisis to understand if these factors differed in nephropathic patients with respect to the rest of the population.

## Results

Most of the treated patients were over 65 years old, and mainly were men. Renal vascular diseases and diabetic nephropathy represented the most frequent pre-existent renal pathologies, and the pre-existent renal failure was usually of an average degree (Table 2). Pre-existing heart failure, supported by different pathologies, in most cases was New York Heart Association classification grade III and was in hemodynamic balance through therapy with cardiocynetics and loop diuretics. Provoking factors were tachyarrhythmia, electrolyte disorders, and pericardiac effusions. Evaluation, through cardiac echography of the ejection fraction, was performed within 72 hours from the acute event and the evaluation, where possible, through a cardiologic Doppler of the E and A waves, allowed us to classify heart failure patients with mainly systolic or diastolic failure. Table 3 shows the type and number of dialysis treatments and the results.

Based on the recovery results, it was possible to divide treated patients into 3 groups (Table 4). Group A (8 patients) comprised patients who recovered a sufficient renal function, and thus were able to avoid further hemodialytic treatments. Group B (7 patients) comprised those who recovered from the acute phase of heart failure; however, because renal function was not sufficient to maintain an adequate hemodynamic balance, they had to continue with chronic dialysis treatment. Group C (9 patients) comprised patients who died after a few hemodialysis treatments. Statistical analysis does not show significant differences between the 3 groups regarding age and initial creatinine values. The significant data are represented by the fact that all patients who recovered an adequate renal function, at least similar to pre-existing levels before the acute event, had been evaluated as having prevalently diastolic failure. Among the 8 patients who continued hemodialysis treatment, 5 patients were classified as affected by diastolic failure: 1 of these patients continued to present a conspicuous pericardiac effusion even after gradual weight loss obtained through dialysis. In group C, 6 patients presented with systolic heart failure, and 2 of the other 3 patients with diastolic failure presented with a discreet pericardiac effusion.

## Discussion

The nephrologist is involved increasingly in treating heart failure, particularly New York Heart Association classification grade III or IV failure. Many hemodialysis techniques are able to treat the fluid overload in patients with heart failure.[5-10] The increase of the average lifetime of the population in general has determined an increase in the incidence of heart failure, often associated with chronic renal failure, underlining how the kidney prematurely uses up its ability to maintain water and electrolyte balance. In our previous work, we treated refractory heart failure with daily hemofiltration.[11-15] This daily technique promptly achieves dray weight and electrolytes balance. In addition, with this technique, some patients recovered their diuresis and responded to diuretics again; others were placed on chronic renal replacement treatment, although many patients died despite treatment. Bicarbonate dialysis technique, using biocompatible filters, allowed personalization of treatment type even better by intervening on patient hydroelectrolyte and acid base imbalances, thus avoiding further derangements. Treatment frequency was determined according to patient clinical conditions, paying particular attention to respiratory function, arterial pressure, blood test results, and acid base balance. Major attention to the patient clinical picture, not aiming to reach dry weight with rapid fluid loss (without edemas) but with the aim of obtaining improvement of the cardiorespiratory picture, allowed us to reduce the number of treatments and to obtain a better treatment tolerance. More careful cardiologic evaluation of patients treated with bicarbonate dialysis allowed us to classify patients as prevalently systolic or diastolic failures, and to better personalize pharmacologic treatment. Because a limited group of patients are presented, it is impossible to compare the 2 studies. However, bicarbonate dialysis, personalized for every patient and started early, is effective in patients affected by heart failure and various degrees of renal failure. Our results show that diastolic heart failure responds better to bicarbonate hemodialysis

**Table 2** Clinical Picture

| N | Age | Sex | Initial Renal Pathology | Creatinine Init Treatment (mg/dL) | Pre-existent Cardiac Pathology | Fraction of Estimated Ejection Within 72 h | Provoking Factors | Prevalent Cardiologic Pattern |
|---|---|---|---|---|---|---|---|---|
| 1 | 88 | M | Nephroangiosclerosis | 1.77 | Aortic-mitralic valvulopathy | 0.70 | Atrial-fibrillation Hyponatremia | Diastolic |
| 2 | 75 | M | Nephroangiosclerosis | 2.67 | Ischemic myocardiopathy | 0.38 | Atrial fibrillation | Systolic |
| 3 | 79 | M | ADPKD | 4.62 | Aortic mitralic-tricuspidal valvulopathy | 0.69 | Flutter fibrillation | Diastolic |
| 4 | 71 | M | Cronic interstitial nephritis | 3.7 | Postischemic dilatative cardiopathy | 0.64 | Over ventricular parossystic tachycardia Hyponatriemia | Diastolic |
| 5 | 79 | M | Nephroangiosclerosis | 1.9 | Ischemic myocardiopathy | 0.40 | Ischemia Hyponatriemia | Systolic |
| 6 | 69 | M | Diabetic nephropathy | 3.12 | Ischemic myocardiopathy | 0.50 | Sinusal tachyarrythmia Hyponatriemia | Diastolic |
| 7 | 79 | M | Nephroangiosclerosis | 2 | Diffuse postischemic hypocinesia | 0.38 | Atrial fibrillation | Systolic |
| 8 | 51 | M | Diabetic nephropathy | 6 | Ischemic myocardiopathy | 0.50 | Ischemia | Diastolic |
| 9 | 67 | M | Diabetic nephropathy | 11.99 | Hypertensive cardiopathy | 0.83 | Pericarditis with effusion Atrial fibrillation | Diastolic |
| 10 | 68 | M | Nephroangiosclerosis | 2.7 | Diffuse acinesia of interventricular septum | 0.29 | Cardiac shock Hyponatriemia | Systolic |
| 11 | 59 | M | Diabetic nephropathy | 4 | Ischemic myocardiopathy | 0.51 | Serious metabolic acidosis Hyponatriemia | Diastolic |
| 12 | 88 | M | Chronic interstitial nephritis | 2.65 | Postischemic dilatative cardiopathy | 0.40 | Atrial fibrillation Hyponatremia | Systolic |
| 13 | 78 | F | ADPKD | 3.3 | Hypertensive cardiopathy | 0.51 | Hypertensive crisis | Diastolic |
| 14 | 91 | F | Nephroangiosclerosis | 3.1 | Aortic mitralic-tricuspidal valvulopathy | 0.40 | Hyperkalemia | Systolic |
| 15 | 75 | F | Nephroangiosclerosis | 3.5 | Ischemic myocardiopathy | 0.43 | Atrial fibrillation | Systolic |
| 16 | 83 | F | Chronic interstitial nephritis | 2.8 | Tricuspidal, pulmonary, aortic valvulopathy | 0.42 | Atrial fibrillation Hyperthyroidism | Systolic |
| 17 | 74 | M | Chronic interstitial nephritis | 5.5 | Aortic mitralic-tricuspidal valvulopathy | 0.77 | Pericarditis with effusion Atrial fibrillation | Diastolic |
| 18 | 87 | M | Chronic interstitial nephritis | 4.04 | Ischemic myocardiopathy | 0.52 | Atrial fibrillation | Diastolic |
| 19 | 78 | F | Diabetic nephropathy | 1.59 | Dilatative cardiopathy | 0.85 | Hyperthyroidism Arrhythmia | Diastolic |
| 20 | 86 | F | Nephroangiosclerosis | 2.7 | Hypertensive cardiopathy | 0.50 | Atrial fibrillation | Diastolic |
| 21 | 67 | M | Diabetic nephropathy | 2.3 | Ischemic myocardiopathy | 0.55 | Ischemia | Diastolic |
| 22 | 48 | M | Chronic glomerulonephritis | 7.64 | Mitralic valvulopathy | 0.40 | Pericarditis | Diastolic |
| 23 | 87 | M | Chronic interstitial nephritis | 7.29 | Hypertensive cardiopathy | 0.60 | Atrial fibrillation | Diastolic |
| 24 | 77 | M | Nephroangiosclerosis | 5.64 | Hypertensive cardiopathy | 0.55 | Ischemia Hyponatriemia | Diastolic |

ADPKD, autosomal dominant polycystic kidney disease.

**Table 3** Characteristics of Dialysis and Results

| N | Age | Sex | Dialysis liq. (Na mEq/L, K mEq/L, $HCO_3$ mmO/L) | Num. Treat. | Number treatment (min) | Polyamide Filter $m^2$ | Weight loss (kg) | Diuresis Recovery | Result |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 88 | M | Na, 145; K, 3; $HCO_3$-28 | 4 | 180 | 1.7 | −6 | 3 day | Diuresis recovery |
| 2 | 75 | M | Na, 142; K, 3; $HCO_3$-28 | 10 | 180 | 1.7 | −5 | Not recovery | Death |
| 3 | 79 | M | Na, 140; K, 2.5; $HCO_3$-28 | Chronic | 240 | 1.7 | −4 | Not recovery | Dialysis |
| 4 | 71 | M | Na, 145; K, 3; $HCO_3$-28 | 8 | 150 | 1.4 | −7 | Not recovery | Death |
| 5 | 79 | M | Na, 145; K, 3; $HCO_3$-28 | 3 | 120 | 1.4 | −4 | Not recovery | Death |
| 6 | 69 | M | Na, 145; K, 3; $HCO_3$-28 | 2 | 180 | 1.4 | −5 | 5 day | Diuresis recovery |
| 7 | 79 | M | Na, 142; K, 3; $HCO_3$-30; | 4 | 120 | 1.7 | −3 | Not recovery | Death |
| 8 | 51 | M | Na, 140; K, 2.5; $HCO_3$-30; | Chronic | 210 | 1.7 | −8 | Not recovery | Dialysis |
| 9 | 67 | M | Na, 140; K, 2.5; $HCO_3$-30; | 14 | 180 | 1.4 | −9 | Not recovery | Death |
| 10 | 68 | M | Na, 145; K, 3; $HCO_3$-28; | 10 | 120 | 1.4 | −9 | Not recovery | Death |
| 11 | 59 | M | Na, 145; K, 2; $HCO_3$-32; | 1 | 180 | 1.7 | −3 | 2 day | Diuresis recovery |
| 12 | 88 | M | Na, 145; K, 3; $HCO_3$-28; | 5 | 120 | 1.7 | −7,5 | Not recovery | Death |
| 13 | 78 | F | Na, 140; K, 2; $HCO_3$-30; | Chronic | 240 | 1.7 | −10 | Not recovery | Dialysis |
| 14 | 91 | F | Na, 144; K, 2; $HCO_3$-28; | 4 | 120 | 1.4 | −6 | Not recovery | Death |
| 15 | 75 | F | Na, 140; K, 2; $HCO_3$-30; | Chronic | 240 | 1.4 | −5 | Not recovery | Dialysis |
| 16 | 83 | F | Na, 145; K, 3; $HCO_3$-30; | Chronic | 240 | 1.4 | −7 | Not recovery | Dialysis |
| 17 | 74 | M | Na, 145; K, 3; $HCO_3$-28; | 14 | 120 | 1.7 | −18 | Not recovery | Death |
| 18 | 87 | M | Na, 145; K, 3; $HCO_3$-30; | Chronic | 210 | 1.7 | −10 | Not recovery | Dialysis |
| 19 | 78 | F | Na, 140; K, 3; $HCO_3$-30; | 3 | 120 | 1.4 | −4 | 4 day | Diuresis recovery |
| 20 | 86 | F | Na, 140; K, 3; $HCO_3$-30; | 2 | 120 | 1.4 | −4 | 5 day | Diuresis recovery |
| 21 | 67 | M | Na, 145; K, 3; $HCO_3$-28; | 2 | 120 | 1.4 | −4 | 3 day | Diuresis recovery |
| 22 | 48 | M | Na, 140; K, 2; $HCO_3$-30; | Chronic | 240 | 1.7 | −4 | Not recovery | Dialysis |
| 23 | 87 | M | Na, 140; K, 2.5; $HCO_3$-32; | 1 | 180 | 1.7 | −2 | 2 day | Diuresis recovery |
| 24 | 77 | M | Na, 145; K, 3.5; $HCO_3$-30; | 1 | 180 | 1.4 | −2 | 2 day | Diuresis recovery |

therapy compared with systolic failure and compels us to obtain a more accurate cardiologic examination before treatment.

## Conclusions

Arrhythmia determined by electrolyte disorders and pericardiac effusion are the most important factors affecting mortality in acute refractory heart failure and CKD. Personalized bicarbonate dialysis is a useful technique for the treatment of patients with heart failure and pre-existing renal failure.

Bicarbonate dialysis is efficient and well tolerated in the treatment of patients with prevalently diastolic heart failure. Patients with prevalently systolic heart failure have a worse prognosis regardless of treatment. An accurate cardiologic study in these patients affected by CKD can help determine which type of dialysis treatment to use.

**Table 4** Clinical Groups

| Group | | | Age | Gender | Prevalent Cardiologic Pattern | Number of Treatments | Diuresis Recovery | Creatininemia Initial Treatment mg/dL |
|---|---|---|---|---|---|---|---|---|
| A | Recovery | 1 | 88 | M | Diastolic | 4 | 8 | 1.77 |
| | | 2 | 69 | M | Diastolic | 2 | 5 | 3.12 |
| | | 3 | 59 | M | Diastolic | 1 | 2 | 4 |
| | | 4 | 67 | M | Diastolic | 2 | 3 | 2.3 |
| | | 5 | 87 | M | Diastolic | 1 | 2 | 7.29 |
| | | 6 | 86 | F | Diastolic | 2 | 5 | 2.7 |
| | | 7 | 77 | M | Diastolic | 1 | 2 | 5.64 |
| | | 8 | 78 | F | Diastolic | 3 | 4 | 1.59 |
| Average ± SD | | | 76.4 ± 10.6 | | | 2 ± 1.1 | 3.9 ± 2.1 | 3.6 ± 2.0 |
| B | Dialysis | 1 | 79 | M | Diastolic | | | 4.62 |
| | | 2 | 83 | F | Systolic | | | 2.8 |
| | | 3 | 51 | M | Diastolic | | | 6 |
| | | 4 | 78 | F | Diastolic | Chronic | Not recovery | 3.3 |
| | | 5 | 75 | F | Systolic | | | 3.5 |
| | | 6 | 87 | M | Diastolic | | | 4.04 |
| | | 7 | 48 | M | Diastolic | | | 7.67 |
| Average ± SD | | 71.6 ± 15.6 | | | | | 4.6 ± 1.7 | |
| C | Death | 1 | 75 | M | Systolic | 10 | | 2.67 |
| | | 2 | 71 | M | Diastolic | 8 | | 3.7 |
| | | 3 | 79 | M | Systolic | 3 | | 1.9 |
| | | 4 | 88 | M | Systolic | 5 | | 2.65 |
| | | 5 | 79 | M | Systolic | 4 | Not recovery | 2 |
| | | 6 | 68 | M | Systolic | 10 | | 2.7 |
| | | 7 | 91 | F | Systolic | 4 | | 3.1 |
| | | 8 | 74 | M | Diastolic | 14 | | 5.5 |
| | | 9 | 67 | M | Diastolic | 14 | | 11.99 |
| Average ± SD | | 76.9 ± 8.3 | | | 8.0 ± 4.3 | | 4.0 ± 3.2 | |
| Student *t* test | | | | | | | | |
| Age | | | Group A versus group | | | NS | | |
| | | | Group A versus group C | | | NS | | |
| | | | Group B versus group C | | | NS | | |
| Number treatment | | | Group A versus group C | | | $P < .0001$ | | |
| Creatininemia | | | Group A versus group B NS | | | | | |
| initial treatment | | | Group A versus group C NS | | | | | |
| | | | Group B versus group C NS | | | | | |

NS, not significant, SD, standard deviation.

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