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CASE REPORT

Preserved Residual Kidney Function after Twelve Years' Hemodialysis

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Residual kidney function (RKF) contributes significantly to solute clearance and fluid removal for dialysis patients, and the presence of RKF is associated with less morbidity and better long-term outcome. Most studies demonstrate that peritoneal dialysis preserves RKF better than hemodialysis (HD). Herein, we report a 55-year-old man with end stage renal failure who had been on chronic HD for 12 years. His RKF is preserved with very slow decline during the past years. Without specific intervention, delicate fluid management, minimal ultrafiltration, and stable hemodynamics during HD may help maintain his RKF. He is currently normotensive with good nutritional status. Although unexpected, we report this HD patient can preserve his RKF for at least 12 years.

Keywords residual kidney function, hemodialysis, proteinuria, ultrafiltration

INTRODUCTION

Accumulating evidence has demonstrated that residual kidney function (RKF) plays an important role in maintaining the overall quality of life and improving the survival rate of dialysis patients, despite it being less well studied in hemodialysis (HD) patients.[1,2] The presence of RKF is frequently ignored, as HD can remove fluid and solute effectively and rapidly. Although hemodialysis is utilized more frequently than peritoneal dialysis as a renal replacement therapy, RKF is generally better preserved in peritoneal dialysis (PD) than HD patients.[3] The ultrafiltration during HD, occurring in a rather short time period, often causes a lowering of blood pressure and frequently leads to symptomatic hypotension. The repeated ischemic insult is considered to be one of the most important factors related to decline of RKF in HD patients.[4] Therefore, achieving dry weight without excessive fluid remains a clinical challenge for nephrologists. We have encountered a 55-year-old man who commenced HD 12 years ago and still had considerable RKF with daily urine output of about 2.0 liters. This case report indicates RKF can be preserved for a long period of time in patients receiving chronic HD. The current condition of this patient regarding adequacy of dialysis, blood pressure control, as well as nutritional status is discussed.

CASE PRESENTATION

A 55-year-old man presented with progressive nausea, dyspepsia, lethargy, and general weakness for several weeks and was transferred from a local medical clinic to our hospital with a provisional diagnosis of uremia in September 1995. During that period of time, the patient also complained of insomnia, anorexia, and generalized skin itching. No specific past medical history was found except bilateral renal stones, which were identified more than 10 years prior to admission. Since then, there was no urolithiasis-related illness. The family history was noncontributory. He denied any systemic disease and did not take herbal medicines.

Upon admission, physical examination revealed an ill-looking general appearance, with normal skin turgor and normotension. No evidence of excessive fluid was noted. The laboratory data were as follows:

- *complete blood cell count:* leukocytes, 5700/μL; hemoglobulin, 6.0 g/dL; hematocrit, 19.4%; mean corpuscular

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volume, 86.3 fL; mean corpuscular hemoglobin, 27.2 pg; mean corpuscular hemoglobin concentration, 31.6 g/dL; and platelet count, 259,000/μL.

- *biochemistry panel:* blood albumin, 4.1 g/dL; glucose, 101 mg/dL; sodium, 122 meq/L; potassium, 5.1 meq/L; blood urea nitrogen, 121 mg/dL; creatinine, 12.5 mg/dL; calcium 8.8, mg/dL; phosphorus, 7.9 mg/dL.

Serology study did not identify any abnormalities. The daily urine amount was 2300 mL, and calculated creatinine clearance was 3.0 mL/minute. The chest x-ray revealed a normal heart size without evidence of lung edema. Renal sonography found bilateral staghorn stones with borderline sized kidneys and reduced parenchymal thickness, but no evidence of obstruction. Because failing renal function and uremic manifestations were evident, HD was initiated three times per week, for four hours per session during hospitalization. No evidence of renal recovery was noted in his follow-up, and his blood urea nitrogen and creatinine level did not show any improvement compared to pre-dialysis levels. In the past twelve years, he had only been hospitalized for inguinal hernia surgery. The operation course had been smooth without complication. In May 2007, he received a parathyroidectomy because of uremic hyperparathyroidism, which was refractory to medical therapy. During the past 12 years, there was no history of emergent dialysis for any urgent condition. His serial change in daily urine quantity and creatinine clearance is shown in Table 1.

At the time of presentation, the dialysis course was HD once and hemodiafiltration twice per week. The ultrafiltration amount was less than 0.5 kg per session. He is healthy and appears well. The urea reduction rate and Kt/V of hemodialysis are 0.77 and 1.46, respectively. The blood pressure is normal at 130/80 mmHg pre-dialysis and 110/70 mmHg post-dialysis. Neither hypotensive agents nor diuretics were administered. The cardio-to-chest ratio is 0.45. The hemoglobin level is 10.1 g/dL under erythropoietin treatment with a dose of 4000 unit per week subcutaneously, and his albumin level is above 4.2 g/dL without dyslipidemia. The plasma level of high sensitivity C-reactive protein was 2.71 mg/L.

Table 1
Serial changes of biochemical data, urine amount, and residual kidney function

	September 1995	June 2005	July 2006	July 2007
Blood urea nitrogen (mg/dL)	121	53	69	47
Serum creatinine (mg/dL)	12.5	11.6	11.8	10.5
Urine amount (mL/day)	2300	2700	1800	1600
Creatinine clearance (mL/min)	3.0	2.68	1.85	1.87
Urinary total protein (gm/day)	1.39	1.45	1.11	1.1

All blood samples were collected prior to midweek dialysis session.

DISCUSSION

It has been recognized that RKF exerts important beneficial effects on dialysis patients such as sodium and volume control, erythropoietin production, adjusting mineral metabolism and preventing vascular calcification, inhibiting inflammatory reaction and oxidative stress, improving nutritional status, and eventually improving the survival rate.[5,6] Therefore, preserving RKF of dialysis patients remains an important goal, although these patients are mostly dependent on dialysis therapy rather than the natural kidney for fluid and metabolite removal. These findings not only illustrate the limitation of current dialysis therapy but reinforce the importance of kidney function, though RKF might be minimal. Most patients still have RKF upon commencement of dialysis therapy. As the primary renal disease progresses, the renal function continues to deteriorate. It has been noted that the first three months after patient entry to dialysis is the most rapid period for decline of RKF.[4] On average, rate of RKF loss was 1–3% per month in PD and 6–7% in HD.[4] Calculation of glomerular filtration rate in renal failure patients using creatinine clearance might be overestimated, and an average of urea and creatinine clearance is indicated for precise estimation. In our patient, only creatinine clearance was calculated; however, the result revealed a very slow decline during a twelve-year follow-up. There was a rather marked drop in creatinine clearance from 2.68 mL/min to 1.85 mL/min in the past two years, but no remarkable event was identified to explain this loss. It is not surprising that our patient developed hyperparathyroidism. Despite the fact there is significant RKF, the uremic milieu in association with other factors may have contributed to uremic hyperparathyroidism, which can also occur as early as mild renal failure. The post-parathyroidectomy course was not followed by any recurrence.

Factors associated with continuous decline of RKF in the dialysis population have been investigated. Most studies concur that PD yields greater preservation of RKF. [4,7] The potential explanations for this may relate to the continuous nature of PD, which maximizes hemodynamic stability and thus results in reduced ischemic injury to the kidneys. Furthermore, it has been proposed that the greater

oxidative stress and inflammatory effects from the extracorporeal circulation of HD may induce renal injury.[6] A common challenge in the clinical management of HD is management of the fluid status appropriately without causing further renal hypoperfusion and symptomatic hypotension. On the other hand, the presence of RKF may be a sign of fluid overload that is deleterious to the cardiovascular system.[8] The roles of other demographic features, such as gender, race, underlying disease, and concurrent disease, however, are rather inconclusive.[4,7] For HD patients, high flux, biocompatible membranes, and ultrapure dialysis water have been shown to have optimal RRF-preserving effects.[9,10] These modifications were considered to induce less inflammation and oxidative stress due to high-flux membranes and nearly sterile and endotoxin-free water.[6] In our patient, the minimal interdialytic body weight gain and neglible ultrafiltration with stable hemodynamics during HD sessions may have played a significant role in prevention of renal ischemia and thus preserving of his RKF despite more than ten years of HD. We could not ascertain whether the usage of the biocompatible high flux artificial kidney was helpful in preserving his RKF.

Renin angiotensin system blockade is a well-known renoprotective approach for patients with proteinuric and diabetic kidney disease through anti-proteinuric effect.[11] Even in late stage, the renin angiotensin inhibitors continue to exert their renoprotective effects and slow the progression to uremia.[12] Thus, the administration of these agents in dialysis patients seems reasonable to preserve RKF. Though mild proteinuria persists, we do not apply any proteinuria-lowering agents to our patient, mostly owing to patient's reluctance. However, one major concern is that the inhibitors of the renin angiotensin system lower blood pressure, especially in normotensives like our patient, which may make dialysis therapy ineffective and result ischemic injury. The role of these agents for preserving RKF in ESRF patients with proteinuria requires further clarification.

In conclusion, we report a chronic HD patient with preservation of RKF for at least 12 years. Although no specific intervention was introduced, decline of RKF was very slow. This patient remains in good nutritional status and is without hypertension. We learned from this study that detailed assessment of volume status and meticulous ultrafiltration can help balance fluid as well as protect against renal ischemia and thus protect the RKF in hemodialysis patients.

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The authors report no conflict of interest.

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