Ferritin as a predictor of decline in residual renal function in peritoneal dialysis patients

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Background/Aims: Inflammation is an important factor in renal injury. Ferritin, an inflammatory marker, is considered an independent predictor of rapid renal progression in patients with chronic kidney disease. However, the relationship between ferritin and residual renal function (RRF) in patients undergoing peritoneal dialysis (PD) remains unclear.

Methods: We reviewed the medical records of patients who started PD between June 2001 and March 2012 at Soonchunhyang University Bucheon Hospital, Korea. A total of 123 patients were enrolled in the study. At 1 month after the initiation of PD, RRF was determined by a 24-hour urine collection and measured every 6 months thereafter. Clinical and biochemical data at the time of the initial 24-hour urine collection were considered as baseline.

Results: The RRF reduction rate was significantly greater in patients with high ferritin (ferritin \( \geq 250 \text{ ng/mL} \)) compared with those with low ferritin (ferritin \(< 250 \text{ ng/mL} \); \(-1.71 \pm 1.36 \text{ mL/min/yr/1.73 m}^2 \) vs. \(-0.84 \pm 1.63 \text{ mL/min/yr/1.73 m}^2 \), respectively; \(p = 0.007\)). Pearson correlation analysis revealed a significant negative correlation between the baseline serum ferritin level and the RRF reduction rate \((r = -0.219, p = 0.015)\). Using multiple linear regression analysis and adjusting for other risk factors, baseline serum ferritin was an independent factor for the RRF reduction rate \((\beta = -0.002, p = 0.002)\).

Conclusions: In this study we showed that a higher ferritin level was significantly associated with a more rapid RRF decline in patients undergoing PD.

Keywords: Ferritins; Peritoneal dialysis; Residual renal function

INTRODUCTION

Peritoneal dialysis (PD) is a well-accepted modality for the treatment of end-stage renal disease (ESRD). Preservation of residual renal function (RRF) is a major factor influencing the quality of life and mortality in PD patients [1,2]. Consequently, determining the predictors of RRF decline has become important in the treatment of PD patients. In previous studies, the RRF decline has been associated with several factors, including diabetes mellitus (DM), congestive heart failure (CHF), high body mass index (BMI), episodes of peritonitis, higher baseline RRF, and higher baseline proteinuria [3-5].

Inflammation is common in chronic kidney disease (CKD) and maintenance dialysis patients. Inflammation is a pathogenic factor of poor renal outcome in patients with glomerulosclerosis or tubulointerstitial fibrosis [6] and a predictor of poor clinical outcome, including high mortality in dialysis patients [7,8]. Ferritin, a clinical marker of iron storage, is also an acute-phase reactant and can be increased in chronic inflammatory states. Ferritin induces macrophage accumulation and increas-
es reactive oxygen species formation [9]. Several studies have shown that ferritin is a significant risk factor associated with rapid renal progression, morbidity, and mortality in CKD and hemodialysis (HD) patients [10-13]. A trend of poor outcomes was observed in advanced CKD patients with serum ferritin levels > 250 ng/mL [12,14]. However, few reports exist on the relationship between ferritin and renal outcome in PD patients. Thus, we examined whether a high ferritin level was associated with a more rapid RRF decline in PD patients.

METHODS

Patients

We reviewed the medical records of patients who started PD between June 2001 and March 2012 at Soonchunhyang University Bucheon Hospital, Korea. We enrolled patients for whom complete data sets describing changes in RRF over at least a 1-year period were available. Of the 282 patients screened, 159 were excluded for anuria (24-hour urine volume < 100 mL) at the start of PD (n = 66), duration of PD maintenance < 1 year (n = 65), and inadequate data (n = 28), resulting in 123 patients included in the study. The patients were divided into two groups according to the baseline serum ferritin level: low (1st to 75th percentile, n = 89, < 250 ng/mL) and high (75th to 100th percentile, n = 34, ≥ 250 ng/mL) groups.

This study was approved by the Institutional Review Board of Soonchunhyang University Bucheon Hospital.

Data collection

We collected demographic and clinical data, including age, gender, BMI, underlying cause of ESRD, episodes of peritonitis and comorbid conditions such as hypertension, DM and CHF. We recorded medications such as renin-angiotensin system (RAS) blockers, calcium channel blockers, β-blockers, diuretics, intravenous (IV)/oral iron, and erythropoiesis-stimulating agents at baseline. The PD modality was classified as continuous ambulatory PD or automated PD. For the subjective global assessment (SGA), we used a four-item, 7-point Likert-type scale [15], which included weight loss, anorexia, subcutaneous fat, and muscle mass. We performed laboratory tests, 24-hour urine and dialysate effluent collection at 8:00 AM after at least 8-hour nil per os within 1 month of the start of PD (as the baseline) and every 6 months thereafter. Normalized protein nitrogen appearance rate (nPNA), renal Kt/V, and peritoneal Kt/V were calculated using urea clearances from a 24-hour urine and dialysate effluent collection. Total Kt/V was calculated as the sum of renal and peritoneal Kt/V. RRF was calculated as the average of the 24-hour urine urea and creatinine clearances to avoid overestimation errors by creatinine clearance and underestimation by urea clearance [16]. The change in RRF over a 1-year period was determined by calculating the change in RRF from the initiation of PD to 1 year after start of PD. All patients were followed until death, follow-up loss, transfer to HD, transplant, transfer to other hospitals, or the end of the study in March 2013. Patients who had anuria during the study period were eliminated from the study and the mean of the RRF decline was calculated in patients who developed anuria. The mean RRF decline was determined by calculating the change in RRF from the initiation of PD to the time of anuria.

Statistical analyses

Statistical analyses were performed using the SPSS version 18.0 (IBM Co., Armonk, NY, USA). Continuous variables were expressed as means ± SD or medians (25th, 75th percentiles) and categorical variables were expressed as percentages. To evaluate the differences between the two groups, Student t test, chi-square test, Mann-Whitney U test, and analysis of covariance were used. When comparing time periods, the paired-sample t test was used for continuous variables. Pearson correlation analysis was performed to estimate the correlations between the rate of reduction in RRF and other variables. Additionally, univariate and multivariate Cox-regression models were used to identify significant predictors of RRF decline. A Kaplan-Meier survival curve was generated for the descriptive analysis of patient survival. Statistical significance was set at a p value < 0.05.

RESULTS

Baseline characteristics of the study population

We screened 282 patients for eligibility who started PD between June 2001 and March 2012; 123 patients with
complete data sets followed up for at least 1 year were enrolled. Table 1 shows the baseline demographic, clinical, and laboratory characteristics of the study patients. The mean patient age was 52.7 ± 12.2 years and 57 (46.3%) were males. Diabetes was the most common cause of ESRD in this study (47.1%). The median serum ferritin level was 167 ng/mL (range, 101 to 268).

Comparison of the low- and high-ferritin groups at baseline and 12 months
At baseline, 27.6% of the patients had serum ferritin ≥ 250 ng/dL. Based on serum ferritin concentrations, there was no significant difference between the low- and high-ferritin groups in age, proportion of males, BMI, proportion of patients with a history of CHF, use of RAS blockers, diuretic use, statin, vitamin C, vitamin D analogues, IV/oral iron supplement, dose of erythropoiesis-stimulating agents, serum iron, serum hemoglobin, serum albumin, or serum creatinine. Additionally, serum high-sensitivity C-reactive protein (hs-CRP), total Kt/V, nPNA, SGA, baseline RRF, and 24-hour urine protein were similar between the low- and high-ferritin groups.

However, the proportion of patients with a history of DM was significantly higher in the high-ferritin compared with the low-ferritin group (50.6% vs. 70.6%, respectively, \( p = 0.045 \); data not shown). No correlation was found between baseline RRF and the serum ferritin level \( (r = –0.009, p = 0.926) \). Additionally, at 12 months after PD initiation, there was no significant difference between the low- and high-ferritin groups in serum hemoglobin, serum albumin, serum creatinine, total Kt/V, nPNA, SGA, or peritonitis rates. Total Kt/V showed a significantly greater decline in the high-ferritin group compared with the low-ferritin group (Table 2).

Consistency of serum ferritin levels
The serum ferritin level did not change significantly at the 12-month follow-up \( (p = 0.352; \text{data not shown}) \). A positive and statistically significant correlation was found between the baseline and mean serum ferritin levels \( (r = 0.700, p < 0.001; \text{data not shown}) \).

Ferritin and change in RRF at the 1-year follow-up
All patients underwent a minimum of two timed urine collections to measure RRF. The RRF decreased signifi-

### Table 1. Baseline characteristics of study subjects (n = 223)

| Characteristic                  | Value                  |
|---------------------------------|------------------------|
| Age, yr                         | 52.7 ± 12.2            |
| Male sex                        | 57 (46.3)              |
| Body mass index, kg/m²          | 23.4 ± 0.3             |
| Causes of ESRD                  |                        |
| DM                              | 58 (47.1)              |
| Hypertension                    | 30 (44.4)              |
| Hypertension combined with DM   | 6 (4.9)                |
| Chronic glomerulonephritis      | 13 (10.6)              |
| Others                          | 16 (13.0)              |
| Comorbid conditions             |                        |
| Hypertension                    | 112 (91.1)             |
| DM                              | 69 (56.3)              |
| Congestive heart failure        | 11 (8.9)               |
| Medication                      |                        |
| RAS blockers                    | 81 (65.9)              |
| Calcium channel blockers        | 100 (81.3)             |
| ß-Blockers                      | 72 (58.3)              |
| Diuretics                       | 66 (53.7)              |
| Statin                          | 48 (39.0)              |
| Vitamin C                       | 101 (82.1)             |
| Vitamin D analogues             | 22 (17.9)              |
| IV/oral iron                    | 71 (57.7)              |
| Erythropoietin, IU/kg/wk        | 115.1 ± 65.0           |
| PD schedule                     |                        |
| CAPD                            | 64 (52.0)              |
| APD                             | 59 (48.0)              |
| Solute transport and PD adequacy|                        |
| Total Kt/V urea                 | 2.75 ± 1.71            |
| nPNA, g/kg/day                  | 1.12 ± 0.88            |
| SGA                             | 26.1 ± 2.2             |
| Laboratory data                 |                        |
| Hemoglobin, g/dL                | 9.5 ± 1.5              |
| Albumin, g/dL                   | 3.9 ± 1.5              |
| BUN, mg/dL                      | 52 (40.65)             |
| Creatinine, mg/dL               | 9.2 ± 6.6              |
| Potassium, mg/dL                | 4 ± 3.75               |
| Phosphorus, mg/dL               | 4.12 ± 0.7             |
| Cholesterol, mg/dL              | 161.4 ± 47.3           |
| Triglyceride, mg/dL             | 102 (65.146)           |
| hs-CRP, mg/dL                   | 0.1 (0.1, 0.6)         |
| Ferritin, ng/mL                 | 167 (101, 268)         |
| Fe (iron), μg/dL                | 52 (31.8, 86.0)        |
| Residual renal function, mL/min/1.73 m² | 2.45 (1.34, 4.01) |
| 24-Hour urine protein, g/day    | 1.27 (0.68, 2.38)      |

Values are presented as mean ± SD, number (%), or median (25th, 75th percentile).

ESRD, end-stage renal disease; DM, diabetes mellitus; RAS, renin-angiotensin system; IV, intravenous; PD, peritoneal dialysis; CAPD, ambulatory peritoneal dialysis; APD, automated peritoneal dialysis; nPNA, normalized protein nitrogen appearance rate; SGA, subjective global assessment; BUN, blood urea nitrogen; hs-CRP, high sensitivity C-reactive protein.
A significant correlation was found between the baseline serum ferritin level and a negative change in RRF at the 1-year follow-up ($r = -0.219$, $p = 0.015$; data not shown). When comparing the groups at the 1-year follow-up, the negative change in RRF was significantly greater in the high-ferritin than in the low-ferritin group ($-1.67 \pm 1.36$ mL/min/1.73 m² vs. $-0.84 \pm 1.63$ mL/min/1.73 m²).

Table 2. Comparison of the baseline and 12-month values of factors related to ferritin

| Variable                        | Ferritin < 250 ng/mL (128.2 ± 57.5) (n = 89) | Ferritin ≥ 250 ng/mL (386.9 ± 140.6) (n = 34) | p value |
|---------------------------------|-----------------------------------------------|-----------------------------------------------|---------|
| Age, yr                         | 52.3 ± 12.4                                   | 53.9 ± 12.0                                   | 0.520   |
| Male sex                        | 30 (43.8)                                     | 18 (52.9)                                     | 0.392   |
| Body mass index, kg/m²          | 23.3 ± 3.1                                    | 25.5 ± 2.8                                    | 0.861   |
| History of DM                   | 45 (30.6)                                     | 24 (70.6)                                     | 0.045   |
| History of CHF                  | 5 (5.6)                                       | 6 (17.6)                                      | 0.070   |
| RAS blockers                    | 57 (64)                                       | 24 (70.6)                                     | 0.494   |
| Diuretics                       | 46 (51.7)                                     | 20 (58.8)                                     | 0.478   |
| Statin                          | 35 (39.3)                                     | 13 (38.2)                                     | 0.912   |
| Vitamin C                       | 73 (82.0)                                     | 28 (82.4)                                     | 0.966   |
| Vitamin D analogues             | 17 (19.1)                                     | 5 (14.7)                                      | 0.569   |
| IV/oral iron                    | 53 (59.6)                                     | 18 (52.9)                                     | 0.507   |
| Erythropoietin, IU/kg/wk        | 110.0 ± 65.2                                   | 128.3 ± 63.6                                  | 0.165   |
| Fe (iron), μg/dL                | 62.3 ± 37.6                                    | 57.2 ± 32.7                                   | 0.614   |
| Hemoglobin, g/dL                | 9.6 ± 1.5                                     | 9.3 ± 1.5                                     | 0.144   |
| Albumin, g/dL                   | 3.8 ± 0.5                                     | 3.7 ± 0.6                                     | 0.531   |
| Creatinine, mg/dL               | 8.5 ± 2.8                                     | 8.6 ± 2.7                                     | 0.800   |
| hs-CRP, mg/dL                   | 0.1 (0.1, 0.6)                                | 0.1 (0.1, 0.7)                                | 0.935   |
| Total Kt/V urea                 | 2.8 ± 1.9                                     | 2.4 ± 0.8                                     | 0.668   |
| nPNA, g/kg/day                  | 1.2 ± 1.0                                     | 1.0 ± 0.3                                     | 0.350   |
| SGA                             | 26.1 ± 2.3                                    | 26.0 ± 2.0                                    | 0.784   |
| Baseline RRF, mL/min/1.73 m²    | 2.4 (1.2, 4.2)                                | 2.8 (1.8, 3.7)                                | 0.705   |
| 24-Hour urine protein, g/day    | 1.2 (0.6, 2.3)                                | 1.6 (0.9, 2.7)                                | 0.101   |

Values are presented as mean ± SD, number (%), or median (25th, 75th percentile).

DM, diabetes mellitus; CHF, congestive heart failure; RAS, renin-angiotensin system; IV, intravenous; hs-CRP, high sensitivity C-reactive protein; nPNA, normalized protein nitrogen appearance rate; SGA, subjective global assessment; RRF, residual renal function.
respectively, \( p = 0.007 \) (Fig. 1). A significant correlation existed between the groups in the model after adjusting for DM, CHF, and total Kt/V \( (p = 0.003) \).

**Ferritin and mean RRF reduction rate**

When further subanalyses were performed in the 72 patients who developed anuria during the study period, a significant correlation between the baseline serum ferritin level and the rate of RRF decline was observed \( (r = 0.586, p < 0.001; \text{data not shown}) \). The mean follow-up period was 58.6 ± 28.7 months. The mean RRF reduction rate was significantly greater in patients with a high ferritin level than with a low ferritin level \( (–0.19 ± 0.14 \text{ mL/min/mon/1.73 m}^2 \text{ vs. } –0.09 ± 0.07 \text{ mL/min/mon/1.73 m}^2, \text{respectively, } p = 0.003) \) (Fig. 2). A significant correlation was observed between the two groups in the model after adjusting for DM, CHF, and total Kt/V \( (p < 0.001) \). The length of time to anuria onset was 28.6 ± 2.2 months in the low-ferritin group and 19.4 ± 3.0 months in the high-ferritin group \( (p = 0.023) \) (Fig. 3).

**Rate of decline predictors in RRF at the 1-year follow-up**

A negative relationship with the RRF reduction rate was found in males, baseline RRF, baseline 24-hour urine protein, and baseline serum ferritin. However, no significant correlation was observed between the RRF reduction rate and age, history of DM, history of CHF, BMI, use of RAS blockers, diuretic use, IV/oral iron supplement, or peritonitis rate. Additionally, the serum hs-CRP was not associated with the rate of RRF decline. Multivariate linear regression analysis showed that baseline RRF, baseline 24-hour urine protein, and baseline serum ferritin were associated with the RRF reduction rate after adjustment for other risk factors (Table 3). Assessment of goodness of fit indicated that the overall model was highly significant \( (p < 0.001, \text{adjusted } R^2 = 0.334) \).

**DISCUSSION**

In this study a significant correlation was found between the baseline serum ferritin level and the rate of RRF decline, and a higher ferritin level was independently associated with a more rapid RRF decline in
PD patients after adjusting for other risk factors. Preservation of RRF is a clinically important goal in CKD patients, even after starting dialysis [17]. Several studies have shown that PD patients have a lower risk of RRF compared with HD patients and that RRF is independently predictive of morbidity and mortality in PD patients [18,19].

Inflammation influences a poor renal outcome and various markers, such as white blood cell count, hs-CRP, and ferritin, have been used to assess inflammatory status [7-10,12,20].

Serum ferritin can be measured readily with a blood test using an immunoradiometric assay, which has a high specificity for detection of iron storage [21]. However, the clinical importance of serum ferritin in dialysis patients may differ from that in the general population. For example, in dialysis patients, an increased serum ferritin may not reflect increased iron stores [22] but is associated with erythropoietin resistance [23], malnutrition, or inflammatory status [10]. Additionally, in previous studies, hyperferritinemia among dialysis patients was significantly associated with high mortality [11,13,24]. Serum ferritin, which has a molecular weight of ~450 kDa, contains heavy (H) and light (L) subunits that may enter the circulation via cell secretion and leakage from damaged cells [25]. Serum ferritin is slightly different from tissue ferritin and contains little or no iron. Tissue ferritin plays a role in intracellular iron handling but the role of serum ferritin is not clearly understood [25]. Inflammatory cytokines, such as interleukin 1 and

**Figure 3.** The length of time to anuria onset was 28.6 ± 2.2 months in the low-ferritin group (n = 50) and 19.4 ± 3.0 months in the high-ferritin group (n = 22, p = 0.023).

| Variable                                | Univariate                      | Multivariate                   |
|-----------------------------------------|----------------------------------|--------------------------------|
| Age, yr                                 | `0.015 (0.012)` 0.197            | `0.181 (0.268)` 0.501          |
| Male                                    | `-0.628 (0.284)` 0.029           | `-0.331 (0.069)` `< 0.001`     |
| History of DM                           | `-0.036 (0.291)` 0.903           | `-0.311 (0.069)` `< 0.001`     |
| History of CHF                          | `-0.390 (0.505)` 0.442           | `-0.311 (0.069)` `< 0.001`     |
| Body mass index, kg/m²                  | `-0.056 (0.048)` 0.294           | `-0.311 (0.069)` `< 0.001`     |
| Baseline RRF, mL/min/1.73 m²            | `-0.360 (0.069)` `< 0.001`       | `-0.311 (0.069)` `< 0.001`     |
| Peritonitis rate, times/patient-yr      | `0.740 (2.392)` 0.757            | `-0.331 (0.069)` `< 0.001`     |
| Use of RAS blockers                     | `0.049 (0.305)` 0.874            | `-0.311 (0.069)` `< 0.001`     |
| Use of diuretics                        | `-0.167 (0.289)` 0.565           | `-0.311 (0.069)` `< 0.001`     |
| Use of IV/oral Iron                     | `0.033 (0.293)` 0.912            | `-0.311 (0.069)` `< 0.001`     |
| hs-CRP, mg/dL                           | `0.094 (0.079)` 0.239            | `-0.311 (0.069)` `< 0.001`     |
| 24-Hour urine protein, g/day            | `-0.314 (0.064)` `< 0.001`       | `-0.279 (0.059)` `< 0.001`     |
| Ferritin, ng/mL                         | `-0.002 (0.001)` 0.15            | `-0.002 (0.001)` 0.002         |

SE, standard error; DM, diabetes mellitus; CHF, congestive heart failure; RRF, residual renal function; RAS, renin-angiotensin system; IV, intravenous; hs-CRP, high sensitivity C-reactive protein.
tumor necrosis factor, increase the synthesis of both the H and L subunits of ferritin by inducing ferritin gene expression independently of iron-dependent ferritin gene expression [20,26]. This inflammatory regulation of ferritin requires the presence of cellular iron. In the case of absolute iron deficiency, serum ferritin is almost always low [20,27], but once minimal iron is available, ferritin is controlled by noniron-dependent factors [20]. Thus, hyperferritinemia is considered an indicator of inflammatory status in dialysis patients [9]. Traditionally, hs-CRP has been regarded as an acute-phase protein indicating inflammatory status [9]. However, we found that baseline hs-CRP was not correlated significantly with baseline serum ferritin because of the nonacute inflammatory status of most patients (\( p = 0.723 \)) [9,28,29]. If inflammation is prolonged with a chronic acute-phase reaction, adverse outcomes such as endothelial damage and atherosclerosis [6,30], which are the principal histological features of almost all renal injuries, may occur [31]. One retrospective study documented a significant association between ferritin as a marker of inflammation and poor renal outcome in terms of the beginning of renal replacement therapy and rapid renal progression in CKD patients [12]. Similarly, we also found that RRF declined more rapidly in PD patients with high baseline serum ferritin levels. Our study is the first report regarding ferritin and renal progression in a PD population.

In this study as in previous work, male gender, baseline RRF, and baseline proteinuria were negatively associated with the rate of RRF decline [3-5]. DM is the most significant factor associated with RRF deterioration [1,5]. Similar to previous reports, in our study the mean RRF reduction rate was greater in patients without DM (–0.14 ± 0.12 mL/min/mon/1.73 m² vs. –0.09 ± 0.69 mL/min/mon/1.73 m², \( p = 0.035 \); data not shown). However, DM alone may not fully account for the rapid deterioration in RRF in the present study because many diabetic patients were excluded according to the inclusion criteria, such as preservation of RRF and a follow-up period of more than 1 year.

Albumin, nPNA and SGA, regarded as markers of nutrition, did not differ between the two ferritin groups, unlike the IV/oral iron supplement and erythropoiesis-stimulating agents, which are related to iron storage and dosage. Thus, these factors did not influence the ferritin level in our study.

An association between baseline ferritin level and overall survival rate was not confirmed. Although the duration of survival differed between the groups, no statistically significant difference was found (84.9 ± 7.6 months vs. 67.6 ± 7.9 months, \( p = 0.779 \); data not shown). We hypothesize this may be due to the small proportion of patients (21.9%) followed until death, the cause of death was not analyzed in individual patients, and baseline ferritin levels might not directly represent mean ferritin levels until death. In a previous study, predeath ferritin levels tended to increase compared to baseline levels [11]. Our study had selection bias because patients with a high risk of mortality were excluded and only patients who had RRF at the start of the study and were followed up for more than 1 year were included.

There were several other limitations to the present study. First, this was an observational study conducted at a single center with a relatively small sample size. A considerable number of patients were excluded due to unavailable baseline data and lack of follow-up. Second, single measures of the baseline serum ferritin level could have reflected acute changes in serum ferritin. However, the mean serum ferritin level did not change over a 1-year period, indicating consistency after the start of PD. Lastly, we were unable to measure the mean RRF reduction rate in all patients because the mean RRF decline was considered from the baseline RRF to anuric status during the study period. As a result, only 72 patients (58.3%) had a final date for mean RRF reduction rate. Despite these limitations, the baseline serum ferritin was independently associated with the RRF reduction rate.

In conclusion, our study demonstrated that higher ferritin levels at the start of PD were associated with an increased risk of rapid RRF decline in PD patients. Future studies investigating the pathological role of ferritin with larger study populations are warranted.

Conflict of interest
No potential conflict of interest relevant to this article was reported.

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KEY MESSAGE
1. The higher ferritin level was significantly associated with a more rapid residual renal function (RRF) decline in peritoneal dialysis (PD) patients.
2. The baseline serum ferritin at the start of PD was found to be an independent predictor of RRF reduction rate in PD patients.

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