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Original Article

Efficacy and safety of febuxostat in peritoneal dialysis patients with hyperuricemia: a pilot study with one year follow-up

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Abstract: To investigate the efficacy and safety of febuxostat on lowering serum uric acid (SUA) and slowing decline of residual kidney function in peritoneal dialysis (PD) patients with hyperuricemia (HUA). Eighteen PD patients with HUA were recruited in this study, and received febuxostat treatment from 10 mg/d to 40 mg/d to maintain SUA in the target range: 240-420 μmol/L (4-7 mg/dL) in men and 210-360 μmol/L (3.5-6 mg/dL) in women. Clinical and biological data was recorded at month 0 (M0) (baseline), M6 and M12, while data at 6 months before the baseline (M-6) was also collected. Among the 18 enrolled participants, 1 case lost follow-up, and 17 cases were included in the final analysis. After febuxostat initiation, SUA was rapidly reduced at M1 compared to M0 (p<0.001). This declining trend was sustained to the end of the study (M12), and the SUA value was decreased at M12 compared with M0 (P<0.001). Residual renal Kt/V (RKt/V) was reduced at M12 compared to M0 (P=0.001) and residual renal creatinine clearance rate (RCCr) declined along the observational time, which was decreased from M0 to M12 (P=0.017). All doses of febuxostat were well tolerated and no adverse effects were observed during treatment. In addition, we found high RKt/V, RCCr, short duration of PD, and low serum creatinine (SCr) were likely to predict achievement of SUA target by univariate logistic regression analysis, but without significance (all 0.05<P<0.1). Our study indicates that febuxostat treatment reduced SUA remarkably and safely in PD patients with HUA.

Keywords: Peritoneal dialysis, hyperuricemia, febuxostat, serum uric acid

Introduction

Hyperuricemia (HUA) is an easily detected and common chemical abnormality that has attracted tremendous attention in recent years due to its increasing prevalence and correlation with risk of hypertension, gout, cardiovascular disease, diabetes, and chronic kidney disease (CKD) [1-7]. The prevalence of HUA varies among different populations and areas, ranging from 2.6% to 36% [6-9]. In the United States, the prevalence increased from 18.2% between 1988 and 1994 to 21.4% between 2007 and 2008 [7]. In China, the prevalence also raised from 8.4% between 2008 and 2009 to 10.9% between 2012 and 2013 [6, 10].

Previous studies indicate that HUA induces renal injury by multiple mechanisms, which include renal vasoconstriction, afferent arteriolopathy, activating the renin-angiotensin system, and epithelial-to-mesenchymal transition in renal tubular cells [11, 12]. HUA is also considered a critical risk factor for CKD and is associated with a higher disease severity [2, 13].

While in peritoneal dialysis (PD) patients, serum uric acid (SUA) is observed to be correlated with residual renal function and HUA could predict both higher all-cause mortality and cardiovascular mortality [14, 15]. The urate lowering therapy (ULT), which has been revealed to slow the kidney function declining in CKD patients with HUA as well as decrease the cardiovascular events, may improve the prognosis of PD patients with HUA [16, 17]. However, the efficacy and safety of ULT in PD patients with HUA is still obscure and needs to be further explored.

Febuxostat, a xanthine oxidase inhibitor, is an emerging drug for ULT which has been demonstrated to be more effective and safer compared with allopurinol and recommended as

Table 1. Demographic, clinical and biological characteristics of PD patients with HUA at baseline

Parameters	PD patients with HUA (n=17)
Age (years)	55.7 ± 11.4
Gender-Female (%)	11 (65%)
BMI (kg/m^2)	25.0 ± 3.2
Duration of PD (months)	16.7 (13.6-33.3)
SUA (μmol/L)	8.48 ± 1.24
Kt/V	1.86 ± 0.22
RKt/V	0.65 ± 0.46
CCr (L/1.73 m^2)	62.0 ± 14.6
RCCr (L/1.73 m^2)	30.5 ± 25.5
WBC (10^9/L)	7.20 ± 2.51
NEUT (10^9/L)	4.65 ± 1.70
Hb (g/L)	98.47 ± 14.44
Plt (×10^9/L)	223.5 ± 100.8
BUN (mmol/L)	24.25 ± 6.055
SCr (μmol/L)	909.7 ± 236.4
Alb (g/L)	33.53 ± 4.900
Ca (mmol/L)	2.187 ± 0.238
P (mmol/L)	2.012 ± 0.346
PTH (ng/L)	357.8 (51.8, 512.9)
TC (mmol/L)	4.809 (3.780, 5.105)
TG (mmol/L)	2.023 (1.100, 2.715)
HDL (mmol/L)	0.974 ± 0.291
LDL (mmol/L)	2.781 ± 1.223
ALT (U/L)	9.04 (7.05, 11.35)
AST (U/L)	12.69 ± 3.366
TNT (ng/mL)	0.052 (0.022, 0.067)
MY (ng/mL)	213.1 (142.1, 270.2)
CK-MB (ng/mL)	1.866 (0.930, 2.295)
Complications	
Hypertension	17 (100%)
Diabetes mellitus	6 (35%)
Cardiac disease	11 (65%)
Cerebrovascular disease	3 (18%)
Drug combinations	
Losartan	1 (6%)
Aspirin	5 (29%)
Diuretic	14 (82%)

Data was presented as Mean value ± SD, median and 25th-75th or count (percentages). PD, peritoneal dialysis; HUA, hyperuricemia; BMI, body mass index; SUA, serum uric acid, CCr, creatinine clearance rate; RCCr, residual renal creatinine clearance rate, WBC, white blood cell; NEUT, neutrophil; Hb, hemoglobin, Plt, platelet, BUN, blood urea nitrogen, SCr, serum creatinine; Alb, albumin; Ca, calcium; P, phosphorus; PTH, parathyroid hormone; TC, total cholesterol; TG, total triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TNT, high-sensitivity troponin-t; MY, myoglobin; CK-MB, creatine kinase-MB.

first-line ULT drug according to 2012 American College of Rheumatology Guideline [18]. Recent studies suggest febuxostat decreases SUA and delays the decline of eGFR in CKD patients and is effective and tolerable in hemodialysis patients [16, 19]. However, no studies on the application of febuxostat in PD patients with HUA have been reported. Thus, this pilot study aimed to investigate the efficacy and safety of febuxostat on lowering SUA and slowing the decline of residual kidney function in PD patients with HUA.

Materials and methods

Participants

Eighteen PD patients with HUA were recruited in this cohort study between Jul. 2014 and Aug. 2015 in the PD unit affiliated with the Department of Nephrology in Shanghai Tenth People's Hospital. The inclusion criteria were: (1) Age between 18 and 80 years; (2) Duration of PD was no less than 6 months; (3) SUA≥420 μmol/l (7 mg/dL) in men, while SUA≥360 μmol/l (6 mg/dL) in women. Exclusion criteria were: (1) Serious infection; (2) Malignant hypertension (3) History of malignant tumor; (4) Previous kidney transplantation; (5) History of heart failure, unstable angina or acute stroke; (6) Pregnancy or lactation. (7) Cognitive impairment, or poor adherence and could not understand the study protocol.

Each participant provided written informed consent and this study was approved by the Ethics Committee of Shanghai Tenth People's Hospital in accordance with the 1964 Helsinki declaration and it's later amendments.

Treatment and follow-ups

No interventions were implemented in the therapy of all participants and the treatments were determined by clinical needs and the patients' willingness to participate. All the patients received febuxostat treatment (Jiangsu Hengrui Medicine Co., Ltd) from 10 mg/d to 40 mg/d to maintain SUA in the target range: 240-420 μmol/L (4-7 mg/dL) in men and 210-360 μmol/L (3.5-6 mg/dL) in women.

All the participants were followed up at month 0 (M0) (baseline), M1, M2, M3, M6, M9, and M12. The data was recorded at each visit, while data

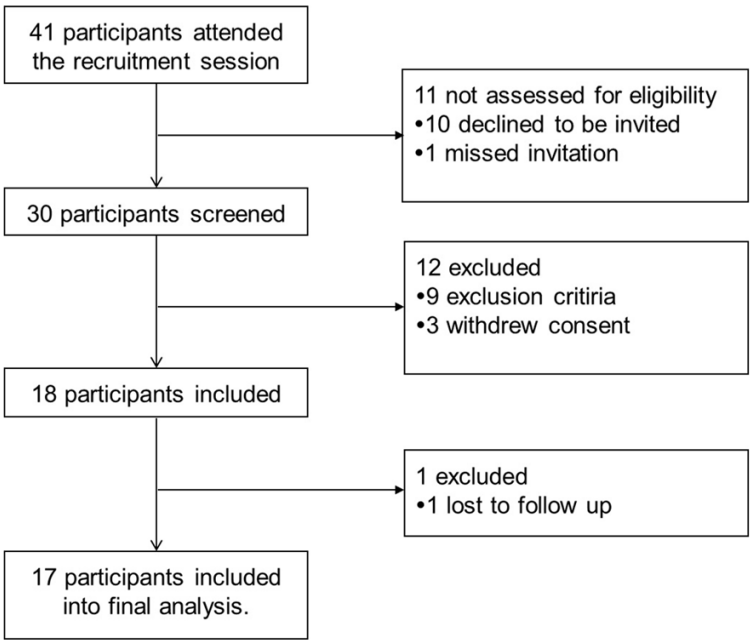

Figure 1. Study profile.

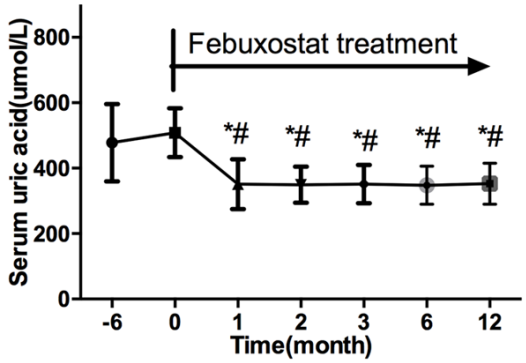

Figure 2. Serum uric acid (SUA) at each visit during febuxostat treatment in peritoneal dialysis (PD) patients with hyperuricemia (HUA). *P<0.05 vs. baseline (M0), #P<0.05 vs. 6 months before baseline (M-6).

at 6 months before the baseline (M-6) was also screened.

Laboratory examination

Blood samples were collected from all subjects at M0, M6 and M12 for laboratory examinations such as routine parameters of blood, liver, and renal function, serum lipid, serum electrolytes and myocardial enzymes. Parts of the parameters were shown in **Table 1**. Additionally, PD adequacy indices of weekly urea clearance index (Kt/V) and creatinine clearance rate (CCr) were analyzed using PD Adequest 2.0 (Baxter Healthcare Ltd, Newbury, UK) at M0, M6, and M12. The retrospective results of blood examinations, Kt/V and CCr at M-6 were also collected for statistical analysis.

Endpoints

The primary endpoint was the change of SUA from M0 to M12, and the secondary endpoints were the changes of residual renal Kt/V (RKt/V) and residual renal CCr (RCCr) from M0 to M12.

Statistics

Data was mainly presented as mean ± standard deviation (SD), median (25^{th}-75^{th}) or counts (percentage). Differences between each visit and baseline or M-6 was compared by paired t-test or Wilcoxon signed rank sum test. The influence of factors at baseline on achieving SUA target at M12 was measured by univariate logistic regression analysis, while all factors with a *P* value<0.1 were further analyzed by multivariate logistic regression. A *P* value<0.05 was considered significant. Statistical analysis was performed using the SPSS 18.0 program (SPSS Inc., Chicago, IL, USA) and Graphics were constructed by GraphPad Prism 6.0 software (GraphPad Software, San Diego, CA, USA).

Results

Baseline characteristics

We invited 41 participants to attend the recruitment session, while 30 cases were screened for eligibility, and 18 participants met the criteria were enrolled, among which 1 case lost follow-up, finally 17 cases were included into the analysis (presented in **Figure 1**).

At baseline, the patients were 55.7 ± 11.4 years old with 11 females (65%), and had duration of PD for 16.7 (13.6-33.3) months. All the 17 patients (100%) were complicated with hypertension, 6 cases out of them (35%) with

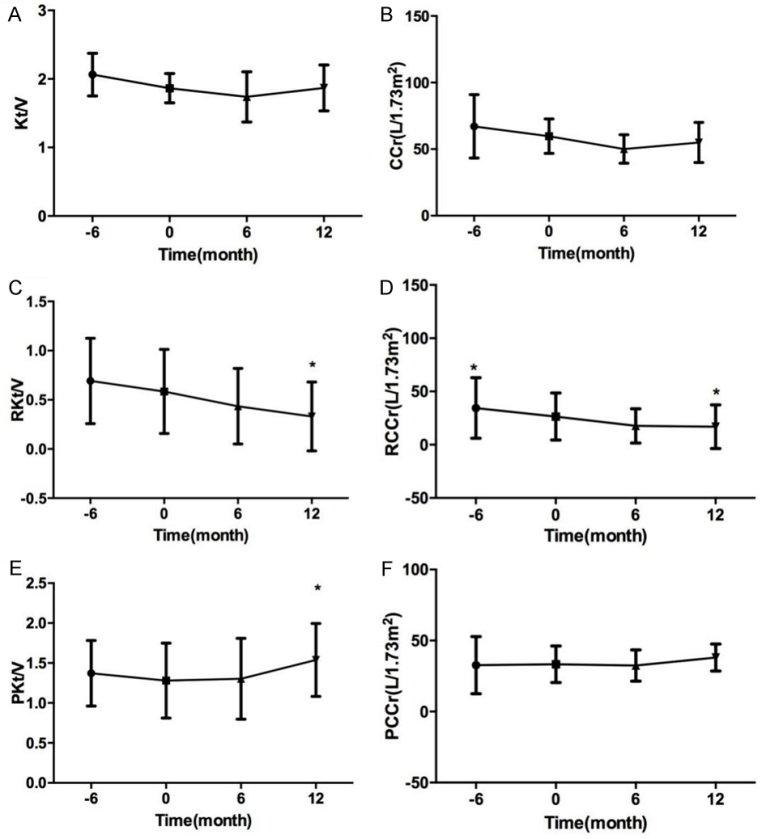

Figure 3. Parameters of residual renal function at each visit in peritoneal dialysis (PD) patients with hyperuricemia (HUA). A. Kt/v, total Kt/V; B. CCr, total CCr (L/1.73 m^2); C. RKt/V, residual renal Kt/V; D. RCCr, residual renal CCr (L/1.73 m^2); E. PKt/V, peritoneal Kt/V; F. PCCr, peritoneal CCr (L/1.73 m^2). *P<0.05 vs. baseline.

diabetes mellitus (DM), 11 cases (65%) with cardiac disease, and 3 cases (18%) with cerebrovascular disease. Other detailed demographic, clinical and biological characteristics as well as drug combinations were presented in **Table 1**.

Primary endpoint-effect on lowering SUA level

As presented in **Figure 2**, before febuxostat treatment, SUA was numerically elevated from M-6 to M0 (baseline), while no significant difference (P=0.452) was observed. After febuxostat initiation, SUA rapidly was reduced at M1 compared to M0 (P<0.001). This declining trend was sustained to the end of the study (M12), and the SUA value was decreased at M12 compared to M0 (P<0.001) as well as M-6 (P=0.007). Twelve out of 17 patients (71%) achieved the treatment target of SUA at M12, while SUA in other 5 patients (29%) were higher than the target value. These indicated the convincing efficacy of febuxostat in lowering SUA in PD patients with HUA.

Secondary endpoints-effect on RKt/V and RCCr

Total Kt/V was divided into RKt/V and peritoneal Kt/V (PKt/V), while total CCr consists of RCCr and peritoneal CCr (PCCr). As presented in **Figure 3A** and **3B**, total Kt/V and CCr remained stable at M-6, M6 and M12 compared to M0 (baseline). While RKt/V was numerically decreased from M-6 to M0 (P=0.066), and further reduced at M12 compared to M0 (P=0.001) (**Figure 3C**). RCCr declined along with the observational time (**Figure 3D**), which was decreased from M-6 to M0 (P=0.009), and from M0 to M12 (P=0.017). PKt/V was elevated from M0 to M12 (P=0.006), while PCCr exhibited the same during M-6 to M12 (**Figure 3E** and **3F**). Furthermore, we compared the change of Kt/V and CCr from M-6 to M0 and from M0 to M12, as shown in **Figure 3**. ΔRKt/V from M0 to M12 was decreased compared with M-6 to M0 (P=0.049) (**Figure 4C**), meanwhile, ΔPKt/V from M0 to M12 was elevated than M-6 to M0 (P=0.015) (**Figure 4E**). However, no differences were found in total ΔKt/V, total ΔCCr, ΔRKt/V and ΔRCCr (**Figure 4A**, **4B**, **4D** and **4F**). These results suggested febuxostat treatment lacked efficacy in slowing the decline of residual renal function in PD patients with HUA.

Safety analysis

All doses of febuxostat were well tolerated and no adverse effects were observed during treatment. After the application of febuxostat therapy, Alb and HDL level were increased at M12 compared to M0 (P=0.023 and P=0.002), but within the normal range as presented in **Table 2**. PTH was also elevated from M0 to M12

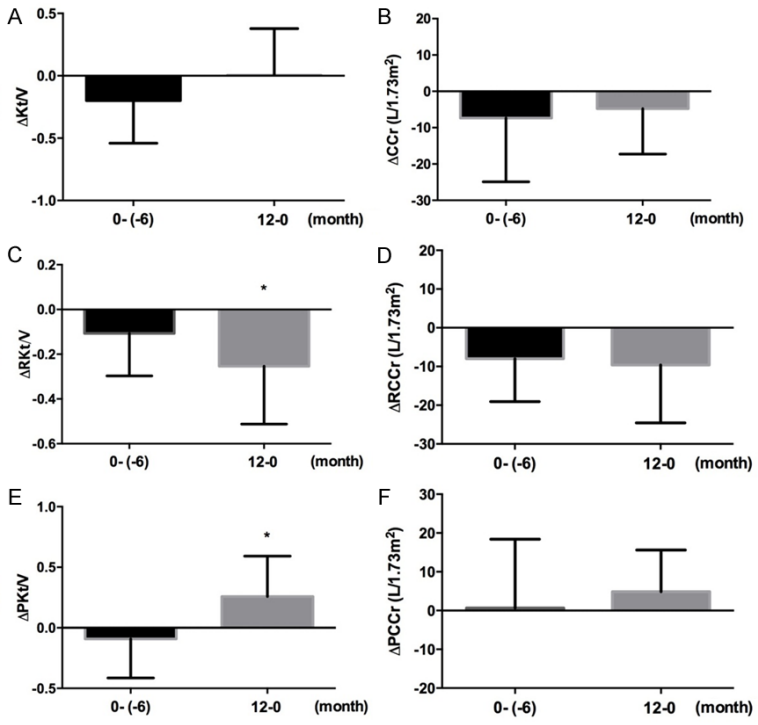

Figure 4. Change of parameters for residual renal function in peritoneal dialysis (PD) patients with hyperuricemia (HUA). A. Kt/v, total Kt/V; B. CCr, total CCr (L/1.73 m^2); C. RKt/V, residual renal Kt/V; D. RCCr, residual renal CCr (L/1.73 m^2); E. PKt/V, peritoneal Kt/V; F. PCCr, peritoneal CCr (L/1.73 m^2). *P<0.05 between 2 groups.

(P=0.006), which might due to the decline of renal function. Other laboratory measurements were not changed after febuxostat treatment as presented in **Table 2**, which indicated febuxostat did not affect the blood system, serum lipid, electrolytes, liver function, and heart function in PD patients with HUA.

Factors affected achievement of SUA target

In order to further investigate the factors affecting achievement of SUA target at M12 to febuxostat treatment, univariate logistic regression analysis was performed and high RKt/V, RCCr, short duration of PD, and low SCr were likely to predict achievement of SUA target as presented in **Table 3**, but without reaching statistical significance (all P between 0.05 and 0.1). These results implied higher renal function might predict better outcome for SUA controlling. All factors with a P≤0.1 in univariate mo-del were subsequently analyzed by multivariate logistic regression model, in which no factors were observed to be associated with achievement of SUA target.

Discussion

In this pilot study, we observed febuxostat reduced SUA remarkably and safely in PD patients with HUA, but was less effective in slowing the decline of residual renal function. Higher residual renal function at baseline might be associated with achievement of SUA target.

HUA could induce hypoxia, glomerulosclerosis, tubulointerstitial, fibrosis and inflammation of kidney by multiple mechanisms, and further facilitate or accelerate the renal injury in many diseases such as CKD, DM, and hypertension [11, 20]. Previous studies have illustrated that HUA increases the risk of CKD, and raising SUA indicates a worse disease severity [13]. In PD patients, HUA is also reported to be associated with decline of residual renal function as well as deterioration of endothelial function, and increased cardiovascular or all-cause mortality [21, 22]. Thus, ULT may contribute to improve the outcomes in PD patients through controlling HUA.

Febuxostat, as a novel ULT drug developed in recent decade, has been demonstrated to be more effective and safer in HUA treatment compared to conventional allopurinol therapy [23]. However, the efficacy and safety of its application in PD patients has been obscure. Sircar D *et al*. revealed in III-IV stage CKD patients with HUA, febuxostat 40 mg/d reduced SUA dramatically and slowed the eGFR decline compared to placebo (PBO) [16]. Tsuruta Y, *et al.* enrolled CKD patients with HUA whose eGFR was below 45 mL/min/1.73 m^2 and patients received allopurinol therapy and switched the allopurinol to febuxostat treatment in some patients while others continued with the primary drug. The results display that SUA is decreased in the febuxostat group compared to allopurinol group at 12 months and eGFR was evaluated in febuxostat group as well [24]. While in hemodialysis patients with HUA, SUA

Table 2. Clinical and biological parameters at M-6, M0 (baseline), M6 and M12 in PD patients with HUA

Parameters	Time point (months)			
	M-6 (n=17)	M0 (n=17)	M6 (n=17)	M12 (n=17)
WBC (10^9/L)	7.63 ± 2.09	7.20 ± 2.51	7.64 ± 4.76	8.07 ± 3.14
NEUT (10^9/L)	5.10 ± 1.67	4.65 ± 1.70	5.54 ± 4.64	5.61 ± 2.40
Hb (g/L)	107 ± 20.59	98.47 ± 14.44	102.8 ± 16.80	99.37 ± 28.97
Plt (×10^9/L)	244.0 ± 83.86	223.5 ± 100.8	247.5 (186.5, 277.5)	265.1 ± 86.80
SUA (μmol/L)	477.8 ± 188.4	508.5 ± 74.55	369.5 ± 72.43*,#	352.7 ± 62.57*,#
BUN (mmol/L)	21.99 ± 8.248	24.25 ± 6.055	23.65 ± 6.553	21.89 ± 4.023
SCr (μmol/L)	808.4 ± 266.7	909.7 ± 236.4	958.7 ±2 57.6#	930.4 ± 243.4#
Alb (g/L)	33.82 ± 5.758	33.53 ± 4.900	33.00 ± 6.042	35.76 ± 4.657*
Ca (mmol/L)	2.252 ± 0.290	2.187 ± 0.238	2.191 ± 0.191	2.229 ± 0.267
P (mmol/L)	1.822 ± 0.442	2.012 ± 0.346	1.887 ± 0.424	1.997 ± 0.500
PTH (ng/L)	185.8 (76.4, 260.9)	357.8 (51.8, 512.9)	353.4 (170.4, 420.3)	497.8 (217.7, 644.9)*,#
TC (mmol/L)	4.807 ± 1.343	4.809 (3.780, 5.105)	5.155 (3.685, 6.165)	4.513 ± 1.537
TG (mmol/L)	2.359 (0.750, 2.505)	2.023 (1.100, 2.715)	2.496 (1.060, 2.025)	1.933 (1.050, 1.925)
HDL (mmol/L)	1.123 ± 0.351	0.974 ± 0.291	1.060 ± 0.235	1.130 ± 0.302*
LDL (mmol/L)	2.584 ± 1.051	2.781 ± 1.223	2.736 ± 0 .856	2.648 ± 1.167
ALT (U/L)	11.48 (7.10, 13.05)	9.04 (7.05, 11.35)	9.34 (5.75, 12.50)	10.47 (7.00, 13.40)
AST (U/L)	13.52 (9.95, 14.60)	12.69 ± 3.366	13.66 (10.70, 14.15)	14.86 ± 3.82
TNT (ng/mL)	0.047 (0.022, 0.057)	0.052 (0.022, 0.067)	0.069 (0.028, 0.085)	0.078 (0.030, 0.087)#
MY (ng/mL)	207.3 (124.8, 276.8)	213.1 (142.1, 270.2)	236.4 (145.9, 290.5)	225.3 (152.1, 255.2)
CK-MB (ng/mL)	1.881 (0.970, 2.300)	1.866 (0.930, 2.295)	1.969 (1.120, 2.175)	2.125 ± 1.219

Data was presented as Mean value ± SD, median and 25^{th}-75^{th}. *P<0.05 vs. 0 month (baseline); #P<0.05 vs. -6 months. PD, peritoneal dialysis; HUA, hyperuricemia; WBC, white blood cell; NEUT, neutrophil; Hb, hemoglobin; Plt, platelet; SUA, serum uric acid; BUN, blood urea nitrogen; SCr, serum creatinine; Alb, albumin, Ca, calcium; P, phosphorus; PTH, parathyroid hormone; TC, total cholesterol, TG, total triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TNT, high-sensitivity troponin-t; MY, myoglobin; CK-MB, creatine kinase-MB.

was found to be diminished after switching from allopurinol to febuxostat, with no changes in other clinical laboratory measurements [19]. In our present study, we show that febuxostat reduced SUA strikingly in PD patients with HUA in accordance with the results in CKD or hemodialysis patients [16, 19, 24], which demonstrates for the first time the efficacy of febuxostat in lowering SUA in PD patients. We also found febuxostat was less effective in suspending the decline of residual renal function which disagreed with the outcomes in CKD patients [16, 24]. This might due to: (1) Small sample of this study (17 participants) compared with previous studies in CKD [16, 24]; (2) No control group was recruited, which the comparative decline of residual renal function without febuxostat could not be discovered; (3) The original disease condition might be more aggressive in the observational duration of this study. Nevertheless, we found higher residual renal function at baseline might have a slight value in predicting achievement of SUA target in PD patients with HUA by univariate logistic regression analysis, which might resulted from the better excretory function of kidney.

As compared to allopurinol, febuxostat presented a non-inferior safety with numerically less adverse effects in HUA patients, therefore febuxostat was recommended to treat gout patients with CKD in 2016 Annual European Congress of Rheumatology [25-28]. Acute neutropenia and liver dysfunction are reported to be comparative common side effects in febuxostat treated CKD patients with HUA [24, 29], while in our study, we found neutrophil count and parameters of liver function remained stable before and after febuxostat treatment, and other laboratory measurements were not changed as well. These implied the febuxostat

Table 3. Factors affecting achievement of SUA target at M12 to febuxostat treatment

	Univariate logistic				Multivariate logistic			
	P value	OR	95% CI		*P* value	OR	95% CI	
			Lower	Higher			Lower	Higher
Age (years)	0.108	1.144	0.971	1.347	-	-	-	-
Gender-Female (%)	0.794	1.333	0.155	11.498	-	-	-	-
BMI (kg/m^2)	0.551	0.904	0.648	1.261	-	-	-	-
Duration of PD (months)	0.074	0.907	0.814	1.009	0.791	1.025	0.852	1.235
SUA (μmol/L)	0.848	0.999	0.984	1.013	-	-	-	-
Kt/V	0.795	1.940	0.013	286.4	-	-	-	-
RKt/V	0.074	110.0	0.635	19045	0.203	1.315E^{10}	0.000	4.889E^{25}
CCr (L/1.73 m^2)	0.261	1.056	0.960	1.161	-	-	-	-
RCCr (L/1.73 m^2)	0.097	1.085	0.985	1.196	0.237	0.714	0.409	1.248
WBC (10^9/L)	0.109	2.064	0.851	5.003	-	-	-	-
NEUT (10^9/L)	0.081	4.406	0.833	23.292	-	-	-	-
Hb (g/L)	0.140	2.305	0.760	6.985	-	-	-	-
Plt (×10^9/L)	0.767	1.002	0.991	1.012	-	-	-	-
BUN (mmol/L)	0.966	1.004	0.841	1.199	-	-	-	-
SCr (μmol/L)	0.069	0.992	0.983	1.001	0.240	0.992	0.979	1.005
Alb (g/L)	0.114	0.743	0.513	1.074	-	-	-	-
Ca (mmol/L)	0.918	0.789	0.009	71.957	-	-	-	-
P (mmol/L)	0.108	0.047	0.001	1.950	-	-	-	-
PTH (ng/L)	0.192	0.998	0.994	1.001	-	-	-	-
TC (mmol/L)	0.384	0.728	0.356	1.488	-	-	-	-
TG (mmol/L)	0.230	0.620	0.284	1.354	-	-	-	-
HDL (mmol/L)	0.880	1.337	0.031	57.165	-	-	-	-
LDL (mmol/L)	0.665	0.828	0.352	1.946	-	-	-	-
ALT (U/L)	0.929	1.011	0.787	1.301	-	-	-	-
AST (U/L)	0.225	1.272	0.862	1.876	-	-	-	-
TNT (ng/mL)	0.969	0.629	0.000	7.829E^9	-	-	-	-
MY (ng/mL)	0.414	0.996	0.985	1.006	-	-	-	-
CK-MB (ng/mL)	0.765	0.889	0.413	1.915	-	-	-	-
Hypertension	-	-	-	-	-	-	-	-
Diabetes mellitus	0.794	0.750	0.087	6.468	-	-	-	-
Cardiac disease	0.406	0.350	0.029	4.153	-	-	-	-
Cerebrovascular disease	0.999	8.975e^8	0.000	-	-	-	-	-
Losartan	1.000	7.343e^8	0.000	-	-	-	-	-
Aspirin	0.540	0.500	0.055	4.583	-	-	-	-
Diuretic	0.870	1.250	0.087	17.975	-	-	-	-

Data was presented as *p* value, odds ratio (OR) and 95% CI. A univariate logistical regression model was used to analyze the factors at baseline in predicting SUA target achievement at M12 treated by febuxostat and all factors with a P≤0.1 in the univariate model were subsequently analyzed by a multivariate Logistic regression model. A P<0.05 was considered significant. The logistic analysis is not available for determining the influence of hypertension on SUA target achievement due to all patients being complicated with hypertension. BMI, body mass index; SUA, serum uric acid; CCr, creatinine clearance rate; RCCr, residual renal creatinine clearance rate; WBC, white blood cell; NEUT, neutrophil; Hb, hemoglobin; Plt, platelet; BUN, blood urea nitrogen; SCr, serum creatinine; Alb, albumin; Ca, calcium; P, phosphorus; PTH, parathyroid hormone; TC, total cholesterol; TG, total triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TNT, high-sensitivity troponin-t; MY, myoglobin; CK-MB, creatine kinase-MB.

treatment in PD patients with HUA was well tolerated and safe.

There were some limitations in this study. Firstly, the study was a single armed cohort

study, which might neglect the natural progress of disease and other confounders or potential sources of bias. Secondly, the sample was small, so some factors influencing the efficacy of febuxostat could not be observed. In conclusion, our study indicated febuxostat treatment reduced SUA remarkably and safely in PD patients with HUA.

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Disclosure of conflict of interest

None.

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