RESEARCH ARTICLE

C-reactive protein variability is associated with vascular access outcome in hemodialysis patients

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Background: Hemodialysis (HD) vascular access failure is one of the most important causes of morbidity and contributes to the cost of dialysis care. There is paucity of data evaluating long-term monitoring of C-reactive protein (CRP) on outcome of HD vascular access.

Methods: We conducted a retrospective study to investigate whether variability of serum CRP level was associated with vascular access failure rate over a 7-year period. A total of 318 HD patients were included. Their demographic data, co-morbidities and biochemical data were reviewed and collected. Serum high-sensitivity CRP (hs-CRP) level was measured every 6 months. Patients were divided into three groups according to their serial hs-CRP levels. Patients with their hs-CRP below 2 mg/L were defined as low group (n=65, 20.4%) and those with higher than 4 mg/L were defined as high (n=39, 12.3%). The rest were classified as fluctuated hs-CRP group (n=214, 67.3%). Treatment of vascular access failure includes angioplasty and access re-creation.

Results: Their body mass index, indicators of dialysis adequacy and serum albumin and hs-CRP levels differed significantly among three groups. The annual vascular access failure rate was significantly higher in fluctuated hs-CRP group than in high hs-CRP group (0.41 vs 0.36, $P=.037$). Serum albumin was a significant associate of vascular access failure. Kaplan-Meier survival analysis indicated patients with high or fluctuated hs-CRP had shorter free interval of vascular access failure than low hs-CRP group.

Conclusions: HD patients with fluctuated hs-CRP levels were associated with increased vascular access failure.

KEYWORDS

C-reactive protein, hemodialysis, vascular access

1 | INTRODUCTION

The increasing number of patients with chronic kidney disease (CKD) and end-stage renal disease (ESRD) has become a major public health issue with large financial implications. Hemodialysis (HD), one of the modalities of renal replacement therapy, depends on long-term patency of vascular access to allow adequate dialysis. In clinical practice, the choice of hemodialysis vascular access includes arteriovenous

fistula (AVF), arteriovenous graft (AVG) or central venous catheter (CVC). HD vascular access failure is one of the most important causes of morbidity and mortality and contributes to the cost of ESRD care.¹

The pathologic mechanisms of HD vascular access failure are multiple. Many risk factors such as diabetes, hypotension, insufficient surgical experience, higher level of uremic toxin, and decrease in heparin administration all had been documented to be related to poor outcome of vascular access.^{2,3} Besides, stenosis often develops

in the venous segment of the AVF due to neointimal hyperplasia (NIH) and at the vein-graft anastomosis in AVG, which obstructs blood flow and thus leads to vascular access failure finally.⁴ Increased expression of matrix metalloproteinase-9 secondary to extracellular matrix dysregulation may also cause structural instability of the neointimal layer and disorganize the overlying endothelial layer subsequently.⁵ Inflammatory process has been proposed to play a pathologic role in NIH. Available data investigating the association between systemic inflammatory markers including high-sensitivity C-reactive protein (hs-CRP), tumor necrosis factor alpha and interleukin-6 (IL-6) and AVF dysfunction and demonstrated that all these systemic inflammatory markers were elevated in patients with AVF dysfunction even though AVF is a local tissue.⁶ Indeed, inflammatory mediators, such as tumor necrosis factor alpha, fibroblast growth factor, and vasoactive and adhesion molecules can also integrate the proliferation of myofibroblasts and migration of smooth muscle cells, facilitate a process like atherosclerosis and give rise to vascular access stenosis or thrombosis.⁷ Tintut et al.⁸ have convincingly indicated that inflammatory mediators can induce mineralization of calcifying vascular cells in vitro and calcium phosphate crystals could activate human monocyte-derived macrophages resulting in a pro-inflammatory state via protein kinase C and MAP kinase pathways. Given this background, inflammation and arterial calcification could explain partially the association between inflammation and HD vascular access failure in ESRD patients.

However, most of the studies focusing on inflammation and HD vascular access failure were cross-sectional or short-term. There is paucity of data evaluating long-term impact of inflammatory mediators such as hs-CRP fluctuation on HD vascular access failure rate. Thus, the current study specifically aimed to investigate whether longitudinal change of CRP levels was associated with vascular access failure in HD patients.

2 | MATERIALS AND METHODS

2.1 | Study population and design

We conducted a retrospective study and enrolled patients with ESRD on maintenance hemodialysis during a period of 7 years from 2006 to 2012 in Kaohsiung Chang Gung Memorial Hospital, Kaohsiung, Taiwan. The study was reviewed and approved by the Institutional Review Board of Chang Gung Memorial Hospital (201600039B0). To be included in the study, participants had to be at least 18 years old, receiving outpatient hemodialysis treatment thrice weekly in 3- to 4-hour sessions for at least 6 months and surviving at least longer than 4 years after the enrollment. The blood flow rate was 200-300 mL/min and dialysate flow rate was 500 mL/min. They used bicarbonate dialysate and reverse-osmosis-purified water. Patients were excluded if they were <18 years old, had a life expectancy of <6 months or had incomplete laboratory data. Their demographic data, co-morbidities, and biochemical data were reviewed and collected according to medical record. Blood samples were obtained immediately prior to the HD procedure of the mid-week session. All laboratory tests were performed by the hospital's central laboratory. Inflammation was

assessed by measuring serum hs-CRP every 6 months over a 7-year period and using the immunoturbidimetric method (Spectra East Laboratories, Rockleigh, NJ, USA). A total of 318 patients were included in the final analysis. Patients were divided into three groups according to serial hs-CRP levels. Patients with more than 80% measurements below 2 mg/L were defined as low hs-CRP group (n=65) and those with more than 80% measurements higher than 4 mg/L were defined as high hs-CRP group (n=39). The rest of these patients were classified as fluctuated hs-CRP group (n=214).

Diabetes mellitus (DM) was defined as patients who were receiving oral anti-diabetic or insulin treatment; with fasting blood sugar ≥ 126 mg/dL or random blood sugar ≥ 200 mg/dL with associated symptoms. Hypertension was defined as systolic blood pressure (SBP) over than 140 mm Hg, diastolic blood pressure (DBP) over than 90 mm Hg, or history of the use of antihypertensive medications for lowering blood pressure. Body mass index (BMI) was calculated as weight (kg)/height (m²). Single pooled Kt/v was used to represent the weekly dialysis dose.

2.2 | Definition of HD vascular access failure and combined endpoint

The definition of HD vascular access failure was made clinically, based on the following criteria: the decrease or absence of palpable and audible bruit in patients with AVG, or clinical signs of AVF dysfunction (diminished thrill, limb swelling, decreased flow rate less than 150 mL/min or increased venous pressure more than 200 mm Hg during HD) or the need for placement of another new CVC. Treatment of HD vascular access failure includes angioplasty and access re-creation. Angioplasty represents percutaneous transluminal angioplasty and thrombectomy, while access re-creation means surgical operations for either AVF, AVG or CVC. The annual vascular access failure rate, as the combined endpoint, was defined as the rate of the need for either angioplasty or access re-creation every year.

2.3 | Statistical analysis

Statistical analysis was computerized using SPSS package for windows version 18.0 (SPSS Inc., Chicago, IL, USA). All continuous variables were provided for mean values $\pm$ standard deviation (SD) or median (inter-quartile ranges), according to whether normal distribution as the Kolmogorov-Smirnov Z test. Meanwhile, all categorical variables were presented as absolute numbers (percentages). For the assessment of clinical features, biochemical factors and access failure rate among three groups depending on serial hs-CRP levels, appropriate one-way analysis of variance (ANOVA), Kruskal-Wallis ANOVA and chi-square test were applied between continuous and categorical variables, respectively. In addition, unpaired test and Fisher's test were used to compare the differences between patients with diabetes or not. Cox regression analysis was utilized to identify factors relevant to vascular access failure. Time interval from enrollment to the first episode of vascular dysfunction was presented using Kaplan-Meier survival curve. A P value of <.05 was considered to be statistically significant.

3 | RESULTS

3.1 | Subject characteristics

A total of 318 patients were enrolled in our study. Table 1 shows the demographics and baseline characteristics of all participants and the comparison among low hs-CRP group, high hs-CRP group and fluctuated hs-CRP group. The mean age of all participants was 64.2±10.9 years old and male patients account for 47% of total subjects. Among the patients, 72% had hypertension and 21% had diabetes mellitus. The mean dialysis vintage was 15.5±4.7 years. At enrollment, most of our subjects had AVF (83%), 14% had AVG and 3% had CVC.

3.2 | Comparison between low hs-CRP, high hs-CRP and fluctuated hs-CRP group

As shown in Table 1, 65 patients (20.4%) were categorized as low hs-CRP group and their mean hs-CRP level was 1.0 mg/L. Thirty nine patients (12.3%) were categorized as high hs-CRP group and mean hs-CRP level was 12.2 mg/L. Two hundred and fourteen patients (67.3%) were categorized as fluctuated hs-CRP group with mean hs-CRP level 4.6 mg/L. The mean hs-CRP level of all subjects was 4.3 mg/L. The prevalence of diabetes mellitus and hypertension was similar among three groups ($P=.199$ and $P=.702$, respectively). Compared with high hs-CRP group and fluctuated hs-CRP group, patients in low hs-CRP group were younger (60.5±10.0 vs 66.5±11.0 vs

64.9±10.9 years old, $P=.006$) and less likely to be male (29% vs 58% vs 50%, $P=.005$). In addition, patients in low hs-CRP group also had lower BMI (20.1±3.5 vs 23.0±4.0 vs 22.2±3.1 kg/m², $P<.0001$) and higher albumin level (3.9±0.2 vs 3.8±0.3 vs 3.8±0.3 g/dL, $P=.002$). Compared with low hs-CRP group and high hs-CRP group, patients in fluctuated hs-CRP group had lower Kt/V (1.42±0.22 vs 1.56±0.24 vs 1.43±0.21, $P<.0001$) and lower urea reduction ratio (URR) (0.74±0.06 vs 0.78±0.05 vs 0.75±0.05, $P<.0001$). The rates of angioplasty and access re-creation did not differ among three different groups (34% vs 50% vs 45%, $P=.191$ and 23% vs 33% vs 35%, $P=.195$, respectively). But the annual vascular access failure rate, as the combined endpoint, was significantly higher in fluctuated hs-CRP group than low hs-CRP group and high hs-CRP group (0.41 vs 0.19 vs 0.36, $P=.037$). In low hs-CRP group, distribution of vascular access at enrollment was: AVF: 86%, AVG: 9% and CVC: 5%. In high hs-CRP group, AVF: 93%, AVG: 7%. In fluctuated hs-CRP group, AVF: 80%, AVG: 17%, CVC: 3%. There was no significant difference regarding the distribution of various vascular access ($P=.315$).

Table 2 displays the comparison between diabetic group and non-diabetic group. Diabetic patients were more likely to be male (59% vs 43%, $P=.024$) and had higher percentage of hypertension (82% vs 69%, $P=.025$). Diabetic patients also had lower URR (0.73±0.06 vs 0.76±0.06, $P=.003$), lower Kt/V (1.38±0.21 vs 1.47±0.23, $P=.003$) and higher hs-CRP level (5.8 vs 3.6 mg/L, $P<.0001$). The rate of angioplasty was higher in diabetic group (54% vs 40%, $P=.036$). The annual vascular access failure rate, as the combined endpoint, was not different between two groups (0.43 vs 0.31, $P=.137$).

TABLE 1 The demographic characteristics and laboratory data of all participants and comparison among three groups

	Total (N=318)	Low hs-CRP hs-CRP <2 (N=65)	High hs-CRP hsCRP >4 (N=39)	Fluctuated hs-CRP 2 ≤hs-CRP≤ 4 (N=214)	P value
Age, y	64.2±10.9	60.5±10.0	66.5±11.0	64.9±10.9	.006
Male, %	149 (47)	19 (29)	23 (58)	107 (50)	.005
BMI, kg/m ²	21.9±3.4	20.1±3.5	23.0±4.0	22.2±3.1	<.0001
Dialysis vintage, y	15.5±4.7	15.4±5.2	15.4±3.8	15.6±4.8	.950
Diabetes, %	68 (21)	9 (14)	11 (28)	48 (22)	.199
Hypertension, %	228 (72)	49 (75)	29 (73)	150 (70)	.702
URR	0.76±0.06	0.78±0.05	0.75±0.05	0.74±0.06	<.0001
Kt/V urea (Gotch formula)	1.45±0.23	1.56±0.24	1.43±0.21	1.42±0.22	<.0001
Calcium, mg/dL	9.2±0.6	9.1±0.5	9.3±0.5	9.3±0.6	.282
Phosphorus, mg/dL	5.0±1.0	4.9±0.9	5.2±1.0	5.0±1.0	.540
Intact PTH, pg/mL ^a	284.7 (129.3-555.9)	228.9 (111.0-424.8)	371.0 (144.5-650.3)	282.0 (129.3-571.9)	.091
Albumin, g/dL	3.9±0.2	3.9±0.2	3.8±0.3	3.8±0.3	.002
ALK-P, U/L ^a	81.5 (67.1-108.8)	78.3 (62.0-96.8)	93.9 (80.2-122.5)	80.7 (66.7-109.8)	.005
hs-CRP, mg/L ^a	4.3 (1.9-8.3)	1.0 (0.7-1.3)	12.2 (9.5-15.3)	4.6 (2.8-7.2)	<.0001
Angioplasty, %	138 (43)	22 (34)	20 (50)	96 (45)	.191
Access re-creation, %	103 (32)	15 (23)	13 (33)	75 (35)	.195
Access failure per year	0.34	0.19	0.36	0.41	.037

BMI, body mass index; URR, urea reduction ratio; Intact PTH, intact parathyroid hormone; ALK-P, alkaline phosphatase; hs-CRP, high-sensitivity C-reactive protein.

^aMedian and interquartile range.

TABLE 2 Comparison between diabetic and non-diabetic groups

	Total (N=318)	Diabetes (N=68)	Non-diabetes (N=250)	P value
Age, y	64.2±10.9	66.4±9.4	63.6±11.2	.055
Male, %	149 (47)	40 (59)	109 (43)	.024
BMI, kg/m ²	21.9±3.4	20.1±3.5	23.0±4.0	.076
Dialysis vintage, y	15.5±4.7	13.8±3.6	16.0±4.9	.001
Hypertension, %	228 (72)	56 (82)	172 (69)	.025
URR	0.76±0.06	0.73±0.06	0.76±0.06	.003
Kt/V urea (Gotch formula)	1.45±0.23	1.38±0.21	1.47±0.23	.003
Calcium, mg/dL	9.2±0.6	9.2±0.5	9.3±0.6	.332
Phosphorus, mg/dL	5.0±1.0	4.9±1.0	5.1±1.0	.147
Intact PTH, pg/mL ^a	284.7 (129.3-555.9)	249.9 (109.7-465.7)	300.4 (133.6-609.3)	.194
Albumin, g/dL	3.9±0.2	3.8±0.2	3.9±0.2	.099
ALK-P, U/L ^a	81.5 (67.1-108.8)	81.3 (69.4-113.9)	81.7 (66.8-106.9)	.733
hs-CRP, mg/L ^a	4.3 (1.9-8.3)	5.8 (3.6-10.6)	3.6 (1.7-7.3)	<.0001
Low hs-CRP	65 (20%)	9 (13%)	56 (22%)	.199
High hs-CRP	39 (12%)	11 (16%)	29 (12%)	
Fluctuated hs-CRP	214 (67%)	48 (71%)	166 (66%)	
Angioplasty, %	138 (43)	37 (54)	101 (40)	.036
Access re-creation, %	103 (32)	22 (32)	81 (32)	.990
Access failure per year	0.34	0.43	0.31	.137

BMI, body mass index; URR, urea reduction ratio; Intact PTH, intact parathyroid hormone; ALK-P, alkaline phosphatase; hs-CRP, high-sensitivity C-reactive protein.

^aMedian and interquartile range.

As shown in Table 3, Cox regression analysis revealed that albumin ($\beta = -1.414$, 95% CI 0.113-0.526, $P = .000$) was an independent predictor for vascular access failure. Grouping of hs-CRP was not related to outcome of vascular access. However, association analysis found an inverse relationship between hs-CRP and albumin ($r = -.339$, $P < .0001$).

The duration from enrollment to the first vascular access failure episode among three CRP groups was compared and we found that vascular access failure rate was not different among the three groups ($P = .091$, Figure 1). Both fluctuated hs-CRP group and high hs-CRP

group had shorter period than low hs-CRP group ($P = .03$ and $.026$). No difference was noted between high and fluctuated hs-CRP groups.

4 | DISCUSSION

In the present study, we demonstrated that patients with different CRP groups in longitudinal observation were characterized by various demographic as well as laboratory data. The rate of annual vascular

	B	P	Exp(B)	95% CI	
				Lower	Upper
hs-CRP groups	0.208	.168	1.231	0.916	1.654
Male, %	-0.377	.081	0.686	0.449	1.048
Diabetes	0.253	.202	1.288	0.874	1.898
Albumin, g/dL	-1.414	.000	0.243	0.113	0.526
Kt/V urea (Gotch formula)	-0.319	.490	0.727	0.294	1.798
Calcium, mg/dL	-0.034	.823	0.966	0.714	1.306
Phosphorous, mg/dL	0.158	.117	1.172	0.961	1.428
PTH, pg/mL	0.000	.667	1.000	1.000	1.001
ALK-P, U/L	-0.003	.179	0.997	0.994	1.001
The types of vascular access	0.222	.138	1.249	0.931	1.675

hs-CRP, high-sensitivity C-reactive protein; Intact PTH, intact parathyroid hormone; ALK-P, alkaline phosphatase.

TABLE 3 Cox regression analysis of vascular access failure

FIGURE 1 Kaplan-Meier curve for the free interval to the first vascular access failure episode in three groups showed vascular access patency rate was not different among the three groups ($P=.091$) but fluctuated hs-CRP group had shorter period than patients with low hs-CRP group ($P=.030$). Comparing with low hs-CRP group, patients with high hs-CRP developed the first failure episode earlier ($P=.026$). Group 1: low hs-CRP group; Group 2: high hs-CRP group; Group 3: fluctuated hs-CRP group

access failure was significantly higher in fluctuated hs-CRP group. Compared with non-diabetic group, the rates of angioplasty was higher in diabetics but the annual vascular access failure rate was similar between diabetics and non-diabetics. Patients with fluctuated hs-CRP levels had shorter period than patients with low hs-CRP group for occurrence of vascular access dysfunction in longitudinal observation.

The adipocytes and other cells within adipose tissue can secrete IL-6 which stimulates liver to produce many kinds of acute phase reactants including CRP.⁹ CRP can also take part in the complement-mediated removal of injured vascular cells and might participate in the pathogenesis of NIH.¹⁰ CRP is a common, reliable and available inflammatory parameter. Recent study has used newly developed high-sensitivity tests to measure CRP level and these enzyme-linked immunosorbent assays can detect CRP level at a concentration of 0.007 mg/L.¹¹ But CRP level is very sensitive and fluctuated easily by many situations such as occult infections or comorbidities. In order to provide more accurate information on the real inflammation state, continuous monitoring of CRP levels had been recommended.¹²

Low grade inflammation state is often present in patients with ESRD and serum markers of chronic inflammation are strongly related to survival in long-term HD patients.¹³ Over a 7-year period, our study disclosed that most HD patients (67.3%) had fluctuated hs-CRP levels and less than one quarter patients (12.3%) were with persistent high hs-CRP levels. Our finding supported the emphasis on long-term monitoring of hs-CRP levels. Both diminished renal clearance and increased production, as a result of uremic factors, can lead to higher systemic concentration of pro-inflammatory cytokines. The causes of inflammatory reaction among dialysis patients are not fully understood. It had been postulated that incompatibility of the dialyzer

membrane, infection, comorbidities, genetic factors and diet all can be the potential sources.¹⁴

Available evidence suggested CRP level was an independent risk factor for AVF thrombosis and forecasted vascular access thrombosis.¹⁵ Additionally, Chang et al.⁵ reported that expression of vascular cell adhesion molecule-1, tumor necrosis factor alpha and IL-6 were increased in thrombosed AVF compared with non-thrombosed AVF. The authors concluded that marked inflammation was associated with thrombosed AVF. Furthermore, vascular calcification and endothelial dysfunction may also play some roles in the pathogenesis of dysfunction of AVF. It has been speculated that chronic inflammation and/or increased oxidative stress promote accelerated vascular calcification directly in dialysis patients.¹⁶ One recent study indicated that low grade chronic inflammation transforms endothelium to a pro-inflammatory and pro-coagulant surface making the vasculature more susceptible to injury and endothelial dysfunction in patients with chronic kidney disease.¹⁷ To the best of our knowledge, this is the first report describing association of long-term monitoring of inflammatory mediators such as hs-CRP fluctuation and outcome of HD vascular access. We found patients with fluctuated hs-CRP had the highest annual failure rate. Several mechanisms may explain this finding. First, gene polymorphism has been identified and was associated with CRP levels.¹⁸ The genetic background may lead to variation in response to inflammatory stimuli. One recent study indicated the influence of IL-10 gene polymorphism.¹⁹ A 'low producer' genotype for the anti-inflammatory cytokine had high levels and more fluctuating patterns of inflammatory reaction. Other genetic influences from IL-1 or IL-6 might also contribute to the variation of CRP fluctuation. Moreover, status of oxidative stress secondary to uremia, dialysis or other factors can also explain this phenomenon.²⁰ In real world, clinicians often focus on patients with persistent high hs-CRP level and neglect the clinical significance of fluctuated hs-CRP because of occasional low level. Similar to our study, longitudinal observational studies have indicated the association between CRP level and all-cause and cardiovascular mortality.^{21,22} Kaplan-Meier curve indicated that patients with either high or fluctuated hs-CRP had shorter period than low hs-CRP group for the free interval of first vascular access dysfunction. This finding also highlights the impact of fluctuated hs-CRP on vascular access failure.

In the current study, we found the rate of angioplasty was higher in diabetic group but annual failure rate did not differ between diabetic and non-diabetic patients. Miller et al.²³ conducted a large prospective study of AVG thrombosis and found that diabetes was not a predisposing factor for this complication. Another study also suggested that diabetes was not a predictor for venous stenosis of AVF.²⁴ In contrast, Afsar demonstrated that the presence of diabetes was independently related to primary AVF failure, possibly because diabetic patients were more likely to have increased thrombogenicity, impaired vasodilatation resulting from endothelial dysfunction and peripheral arterial disease.²⁵ In another study, Puskar et al.² found diabetes was one of the important factors affecting AVF survival. Difference in study period may explain the contradictory results of these studies. Second, glyce-mic control was crucial in diabetic patients and may affect failure rate.

It has been well documented that diabetic HD patients with optimal glycemic control had higher patency rate than patients with suboptimal glycemic control.²⁶ In our study, glycemic control in diabetic HD subjects was relatively acceptable with mean glycosylated hemoglobin 7.1% and no significant difference was observed among three groups. Undoubtedly, because diabetic patients usually are with higher mortality rate, patients enrolled in our study might survive with lower cardiovascular risk. It is probable the selection bias affects study results.

In our study, Cox regression model found serum albumin level was the sole independent predictor for vascular access failure. Furthermore, association analysis revealed that there was an inverse relationship between hs-CRP and albumin. Another recent study had also shown the variability of CRP was also one of the powerful predictors for the levels of albumin.²⁷ Levels of albumin have long been recognized as an important prognostic factor of all-cause and cardiovascular mortality in hemodialysis patients. As we understand that albumin is not only a nutritional marker, but also an inflammatory marker, it is conceivable to accept that serum albumin was a significant outcome predictor of vascular access in hemodialysis patients. On the other hand, level of hs-CRP represents inflammatory status and the characteristics of its variability provide additional information regarding outcome of vascular access.

Current pharmacological strategy such as statin to prevent vascular access failure is mostly unsatisfactory.²⁸ A software, computer-assisted surgical planning, is another therapeutic option and can help us to optimize vascular access geometry and decrease blood velocity oscillations and flow, and shear stress instability.²⁸ Based on current knowledge of level and long-term change of hs-CRP, no specific treatment right now can confidently be suggested. In view of the clinical significance of fluctuated hs-CRP in serial monitoring, we recommend to provide more closely surveillance of vascular access such as doppler ultrasound in these high risk patients.

There remains several limitations in our study. First, as the current study was retrospective, it is not allowed to compare all factors that might affect outcome of vascular access. Second, because hs-CRP measurements were performed every 6 months for consecutive 7 years, occult inflammatory reactions such as periodontal disease²⁹ or chronic *Chlamydia pneumoniae* infections³⁰ during study period might be ignored and not detected. Third, due to small sample size, discrimination for the different locations of vascular access was not performed.

In conclusion, our study had demonstrated that HD patients with fluctuated hs-CRP level rather than high hs-CRP level were more likely to have higher vascular access failure rate. The rate of angioplasty for thrombosed vascular access was increased in diabetic patients. The free interval of vascular access dysfunction in patients with either high or fluctuated hs-CRP was shorter than those with low hs-CRP.

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