



## Research Article

# Examination of amino acid profile in patients with chronic renal failure

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### Abstract

**Objectives:** This study aims to analyze the plasma free amino acid profiles pre and post dialysis in patients with chronic kidney failure (CRF), and to evaluate their potential utility in diagnosis and treatment by comparing them with profiles from a healthy control group.

**Methods:** Plasma samples were collected from 46 healthy control and 46 patients diagnosed with CRF who applied to Şanlıurfa Harran University Medical Faculty Dialysis Department. Plasma free amino acid profiles were analyzed with LC-MS/MS.

**Results:** Mean values of alanine, arginine, aspartic acid, citrulline, histidine, methionine, tyrosine, hydroxyproline, glycine, leucine, isoleucine, lysine, ornithine, phenylalanine, proline, serine glutamic acid, glutamine, valine, taurine, alioisoleucine, alphaaminoadipic acid, anserine, gammaaminobutyric acid, 1- methylhistidine, 3-methylhistidine, 5-hydroxytryptophan levels in CRF patients exhibited higher levels compared to the control group. Phosphoethanolamine, cystine, alphaaminobutyric acid, betaaminoisobutyric acid and tryptophan were found to be lower in CRF patients than control group. When post-dialysis compared to pre-dialysis; there was an increase in citrulline, histidine, alanine, arginine, aspartic acid, glutamic acid, glycine, cystine, isoleucine, proline, phosphoethanolamine, taurine, alioisoleucin, alphaaminoadipic acid, anserine, alphaaminobutyric acid, betaaminoisobutyric acid, beta alanin, 1-methylhistidine , 5-hydroxytryptophan levels; there was a decrease was observed in glutamine, leucine, lysine, ornithine, phenylalanine, serine, valine, asparagine, methionine, tryptophan, tyrosine, hydroxyproline, gammaaminobutyric acid, 3-methylhistidine levels. Citrulline, glycine, anserine, alphaaminobutyric acid, gammaaminobutyric acid, phosphoethanolamine and taurine levels were found to be significant in the Paired samples test, which was used to test the significance of the difference between the arithmetic means of the groups ( $p < 0.05$ ).

**Conclusion:** More studies were needed to understand the role of amino acids in CRF.

**Keywords:** Amino acid, chronic kidney disease, LC/MS, metabolic

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Chronic kidney disease (CKD) refers to a group of diverse conditions that affect the anatomy and function of the kidneys, is particularly common in individuals with diabetes and hypertension and is a progressive condition associated with significant illness and death rates [1–4].

CKD is defined by persistent urinary abnormalities, structural changes in the kidneys, or a decline in excretory function, all of which indicate a progressive loss of functional nephrons. It is a worldwide health concern linked to considerable illness and death rates, primarily due to its strong link with cardiovascular

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disease. A large group of CKD patients are more susceptible to cardiovascular complications and early fatality. As chronic kidney disease advances to end-stage renal disease, initiating renal replacement therapy becomes necessary. However, in many parts of the world, access to renal replacement therapy remains limited. Various risk factors play a role in both the initiation and progression of CKD. This includes having fewer nephrons at birth and the loss of nephrons due to aging, and exposure to nephrotoxic agents. Additionally, chronic conditions significantly lead to ongoing kidney damage. Effective management of CKD requires early detection, identification and treatment of the underlying cause, and careful attention to secondary mechanisms that perpetuate nephron loss. Primary treatment approaches involve tight regulation of blood pressure, suppression of the renin-angiotensin system, and targeted therapies designed to delay the progression of kidney dysfunction [5, 6].

Chronic renal failure (CRF) is characterized by a gradual and ongoing decline in kidney function, impairing fluid and solute regulation as well as metabolic and endocrine processes, due to reduced glomerular filtration rate (GFR). This condition usually occurs when GFR falls below 25 mL/min. When GFR is reduced by 75% of normal, the deterioration in kidney function continues even when the damage that caused it is removed [7].

Chronic kidney failure and dialysis treatment induce metabolic alterations that are not fully captured by routine biochemical parameters, but also involve changes in metabolic biomarkers and amino acid-related pathways [8].

The kidney's role in amino acid and protein metabolism, including the metabolic processes regulated by the kidney for dietary protein and the breakdown and secretion of protein metabolites, is important [9]. Amino acids are essential components that support life, growth, reproduction, development, and well-being in all organisms [10]. Measuring the levels of free amino acids present in bodily fluids and tissues provides nutritional insights that are valuable for diagnosing certain diseases, particularly those related to metabolic disorders. Particular irregularities in concentrations of amino acids have been linked to various diseases and conditions such as liver and kidney failure, cancer, diabetes, fatty liver, muscle dysfunction and protein malnutrition. The role of plasma free amino acids in disease risk assessment prediction has been seen as potential applications for monitoring nutrition [11].

The primary objective of this study was to assess and compare serum amino acid levels in patients diagnosed with chronic kidney failure before and after dialysis treatment. Additionally, the study aimed to investigate the potential impact of these amino acid levels on treatment strategies and clinical outcomes, thereby providing valuable insights into the role of amino acid metabolism in kidney disease management.

## Materials and Methods

### Experimental design

Ethical approval for this observational study was obtained from the Harran University Clinical Research Ethics Commit-

tee (No: 20, Date: 29/11/2021). Informed consent was obtained from all of the patients included in this study and the research was conducted according to the ethical principles the Declaration of Helsinki.

Plasma samples were collected from 46 patients diagnosed with chronic renal failure who presented to the Department of Nephrology and Dialysis Unit of Sanliurfa Harran University Faculty of Medicine Hospital, both before and after dialysis, between the years 2021 and 2022. We collected blood samples from patients with chronic kidney disease undergoing hemodialysis and those pre and post-dialysis. The type of dialysis we performed was hemodialysis (HD). The most common type of dialysis involves a dialyzer that filters waste, salt, and excess fluid from the blood. This was performed three times a week at the dialysis center of Harran University Hospital, and each visit lasted approximately four hours. A catheter was also used to create a vein in the patients. Blood is withdrawn from the body, filtered through the dialyzer, and returned. All patients underwent three four-hour hemodialysis sessions per week at the dialysis department of Harran University Hospital over a period of min 3 max 4 years. 21 of these patients were male and 25 were female. The total sample size was 46, and the mean age for both sexes was  $35.4 \pm 15.6$  years (Table 1). However, glomerulonephritis and polycystic kidney disease were found to be significant etiological factors for the disease. None of the patients receiving hemodialysis had diabetic nephropathy. Patients who had undergone kidney transplantation or peritoneal dialysis were excluded from the study. Baseline information, including age, sex, age at hemodialysis initiation, and cause of renal failure, was obtained from medical records at the study center. The urea reduction rate was used as an index of hemodialysis adequacy, and baseline information and other parameters were compared between the two groups to determine the effect of hemodialysis duration on these factors. Plasma samples from 46 healthy individuals were used as the control group. The healthy control group had a normal kidney function (serum creatinine and eGFR within reference ranges), no history of chronic or renal diseases, and not using medications affecting renal function. Amino acids were measured once for control group. Of the individuals in the control group, 26 were female and 20 were male, and the mean age was  $33.4 \pm 12.6$  years (Table 1). In the control group, creatinine and eGFR values were 0.72 (0.30) mg/dL and 105.4 (46.82) mL/min/1.73 m<sup>2</sup>, respectively, reflecting normal kidney function (Table 1). In the patient group, creatinine and eGFR values were 7.15 (3.02) mg/dL and 7.05 (5.35) mL/min/1.73 m<sup>2</sup>, respectively, representing end-stage chronic kidney disease patients undergoing hemodialysis (Table 1). There was no significant difference in age between the groups ( $p=0.50$ ). However, the differences in creatinine and eGFR values were statistically significant ( $p<0.001$ ). These findings indicate that the patient and control groups reflect the clinically expected profiles. Blood samples taken from the individuals were taken into EDTA tubes, centrifuged to obtain plasma, and stored at -80°C until analysis.

**Table 1. Age, sex and clinical characteristics of the patient and control groups**

Groups	Patient group (n=46)	Control group (n=46)	p
Age (year)	35.4±15.6	33.4±12.6	0.50
Creatinine (mg/dL)	7.15 (3.02)	0.72 (0.30)	<0.001
eGFR (mL/min/1.73 m <sup>2</sup> )	7.05 (5.35)	105.4 (46.82)	<0.001
Sex, n (%)			
Female	25 (54.3)	26 (56.5)	0.84
Male	21 (45.7)	20 (43.5)	

An independent t-test was used for age, and the data were presented as mean±standard deviation (mean±SD). Mann–Whitney U test was used for creatinine and eGFR. Creatinine and eGFR values are presented as median (IQR, Interquartile Range). p-value for sex was determined by Chi-square ( $\chi^2$ ) test.

### LC-MS/MS analysis

Analyses were conducted using a LC-MS/MS (Shimadzu 8045, Japan) device. For the patient group, two plasma samples (pre- and immediately after post-dialysis) were collected from each individual, resulting in a total of 92 samples. In the control group, a single sample was obtained from each subject, yielding a total of 46 samples. For amino acid studies, samples taken from -80°C were kept until they reached room temperature. JASEM amino acid kit was used for analysis. The kit's working principle was followed for plasma samples from each patient. First, 50µl of patients' plasma was taken into numbered sterile Eppendorf tubes. 50µl of the Internal Standard solution in the amino acid kit was transferred to these tubes. Each tube was vortexed for 5 seconds. 700µl of the Reagent-1 solution in the kit was added to the vortexed tubes. The tubes were vortexed again for 15 seconds. The vortexed tubes were centrifuged at 3000 rpm for 5 minutes. The supernatant portion of the samples carefully taken from the centrifuge was transferred to HPLC vial tubes with the help of a sterile pipette. For analysis, vial tubes were placed in the tray compartment in the HPLC section of the LC-MS/MS device and read. Mobile Phase-A and Mobile Phase-B in the amino acid kit were used as mobile phase. Restek LC Columns were used as columns.

### Statistical analysis

Kolmogorov-Smirnov and Shapiro-Wilk tests were applied to evaluate whether the data followed a normal distribution. Since the variables showed normal distribution, Independent Samples test was used to compare the average amino acid values of healthy individuals and patients before dialysis. Paired samples test was used to compare the amino acid values of patients before and after dialysis. Descriptive statistics for numerical variables were presented as mean ± standard deviation. Statistical analyses were performed using the SPSS software package (Windows version 24.0), with a p<0.05 considered statistically significant.

### Results

The amino acid levels of the healthy group and the pre-dialysis and post-dialysis amino acid levels of patients diagnosed with chronic renal failure are given collectively in Table 2.

In patients with chronic renal failure, alanine, arginine, aspartic acid, citrulline, histidine, methionine, hydroxyproline, glycine, leucine, isoleucine, lysine, ornithine, phenylalanine, proline, serine, glutamic acid, glutamine, valine, taurine, alloisoleucine, gammaaminobutyric acid, 3-methylhistidine amino acid levels were found higher than in the control group (p<0.05). Tyrosine, alphaaminoadipic acid, anserine, 1-methylhistidine, 5-hydroxytryptophan amino acid levels were also found higher in patients with chronic renal failure compared to the control group, but this was not statistically significant. Phosphoethanolamine, cystine, alphaaminobutyric acid, betaaminoisobutyric acid and tryptophan amino acids were decreased in CRF patients relative to the control group (p<0.05).

After dialysis, compared to pre-dialysis, there were increases in citrulline, glycine, phosphoethanolamine, taurine, anserine and alphaaminobutyric acid amino acid levels (p<0.05). After dialysis, there were increases in alanine, arginine, aspartic acid, glutamic acid, cystine, isoleucine, proline, histidine, alloisoleucine, alphaaminoadipic acid, betaaminoisobutyric acid, beta alanine, 1-methylhistidine and 5-hydroxytryptophan amino acid levels compared to pre-dialysis, but this was not statistically significant. After dialysis, there were decreases in glutamine, leucine, lysine, ornithine, phenylalanine, serine, valine, asparagine, methionine, tryptophan, tyrosine, hydroxyproline, gammaaminobutyric acid and 3-methylhistidine amino acid levels compared to pre-dialysis.

Paired samples test was performed to evaluate the significance of the difference between the arithmetic means of the groups given in Table 2, and citrulline, glycine, anserine, alphaaminobutyric acid, gammaaminobutyric acid, phosphoethanolamine and taurine levels were found to be significant (p<0.05).

### Discussion

The kidneys are fundamentally involved in protein metabolism, taking part in the synthesis, breakdown, filtration, reabsorption, and excretion of amino acids and peptides. They also contribute to several key metabolic pathways, including the conversion of phenylalanine to tyrosine, arginine metabolism, and transmethylation. In patients with chronic kidney disease, disruptions in these processes can occur due to metabolic acidosis, chronic inflammation, dietary restrictions and amino

**Table 2. Amino acid levels of control, pre-dialysis and post-dialysis patients groups ( $\mu\text{mol/L}$ ) (mean $\pm$ SD)**

Aminoacids	Control	Pre-dialysis	Post-dialysis	p (C-pre)	p (pre-post)
1-methylhistidine	1.30 $\pm$ 0.37	1.35 $\pm$ 2.06	5.81 $\pm$ 12.10	0.927	0.128
3-methylhistidine	0.66 $\pm$ 1.15	5.42 $\pm$ 7.74	2.90 $\pm$ 2.81	0.013*	0.107
5-hydroxytryptophan	0.04 $\pm$ 0.09	0.40 $\pm$ 0.89	0.82 $\pm$ 1.25	0.087	0.146
Alanine	274.73 $\pm$ 83.43	348.79 $\pm$ 101.11	355.59 $\pm$ 98.91	0.007*	0.802
Alloisoleucine	0.36 $\pm$ 0.20	1.35 $\pm$ 0.85	1.75 $\pm$ 0.94	0.000*	0.197
Alphaaminoadipic acid	0.94 $\pm$ 0.57	1.10 $\pm$ 1.90	2.35 $\pm$ 5.35	0.723	0.277
Alphaaminobutyric acid	13.32 $\pm$ 6.12	0.38 $\pm$ 0.16	0.82 $\pm$ 0.87	0.000*	0.029**
Anserine	2.11 $\pm$ 2.31	2.96 $\pm$ 1.59	4.50 $\pm$ 2.69	0.158	0.045**
Arginine	68.49 $\pm$ 22.42	262.88 $\pm$ 94.63	269.19 $\pm$ 85.75	0.000*	0.817
Asparagine	43.93 $\pm$ 11.42	61.09 $\pm$ 43.31	40.03 $\pm$ 25.48	0.098	0.073
Aspartic acid	10.49 $\pm$ 10.83	156.45 $\pm$ 38.16	172.78 $\pm$ 75.13	0.000*	0.464
Beta alanine	3.04 $\pm$ 1.05	2.22 $\pm$ 1.34	3.63 $\pm$ 5.59	0.019*	0.275
Betaaminoisobutyric acid	2.66 $\pm$ 0.90	1.12 $\pm$ 0.99	2.12 $\pm$ 2.52	0.000*	0.137
Citrulline	19.04 $\pm$ 7.21	31.74 $\pm$ 10.99	40.26 $\pm$ 13.67	0.000*	0.024**
Cystathionine	0.12 $\pm$ 0.12	0.20 $\pm$ 0.12	0.34 $\pm$ 0.47	0.021*	0.189
Cystine	46.55 $\pm$ 24.44	0.56 $\pm$ 0.58	0.70 $\pm$ 0.48	0.000*	0.416
Gammaaminobutyric acid	4.89 $\pm$ 1.70	15.87 $\pm$ 13.79	8.71 $\pm$ 2.61	0.002*	0.042**
Glutamine	139.17 $\pm$ 65.15	277.72 $\pm$ 115.66	276.19 $\pm$ 197.17	0.000*	0.975
Glutamic acid	78.31 $\pm$ 47.00	400.64 $\pm$ 126.12	409.59 $\pm$ 157.74	0.000*	0.792
Glycine	198.01 $\pm$ 50.47	337.48 $\pm$ 50.64	404.20 $\pm$ 75.75	0.000*	0.009**
Histidine	57.52 $\pm$ 12.48	89.14 $\pm$ 22.65	90.76 $\pm$ 21.33	0.000*	0.828
Hydroxyproline	25.67 $\pm$ 10.39	95.33 $\pm$ 44.36	81.25 $\pm$ 37.95	0.000*	0.289
Isoleucine	59.96 $\pm$ 15.95	107.90 $\pm$ 26.12	117.64 $\pm$ 74.83	0.000*	0.590
Leucine	95.27 $\pm$ 27.06	381.40 $\pm$ 124.91	341.20 $\pm$ 106.80	0.000*	0.251
Lysine	132.56 $\pm$ 36.88	267.12 $\pm$ 148.09	198.06 $\pm$ 72.99	0.001*	0.054
Methionine	24.25 $\pm$ 6.27	44.20 $\pm$ 31.17	34.77 $\pm$ 13.49	0.010*	0.246
Ornithine	67.51 $\pm$ 22.88	148.12 $\pm$ 107.29	141.74 $\pm$ 89.76	0.003*	0.865
Phenylalanine	51.10 $\pm$ 10.20	146.47 $\pm$ 36.48	143.42 $\pm$ 23.46	0.000*	0.783
Phosphoetanolamine	28.38 $\pm$ 24.45	3.90 $\pm$ 1.56	7.47 $\pm$ 2.91	0.000*	0.000**
Proline	155.49 $\pm$ 40.28	338.91 $\pm$ 91.73	384.03 $\pm$ 118.90	0.000*	0.146
Serine	130.52 $\pm$ 31.19	246.53 $\pm$ 62.05	233.32 $\pm$ 55.63	0.000*	0.526
Taurine	78.29 $\pm$ 37.28	193.30 $\pm$ 18.46	227.04 $\pm$ 23.25	0.000*	0.000**
Threonine	132.52 $\pm$ 37.08	144.03 $\pm$ 45.99	135.44 $\pm$ 33.15	0.334	0.518
Tryptophan	56.44 $\pm$ 14.93	42.11 $\pm$ 16.15	38.26 $\pm$ 13.82	0.002*	0.433
Tyrosine	68.62 $\pm$ 16.83	76.36 $\pm$ 31.81	72.44 $\pm$ 20.71	0.268	0.638
Valine	174.70 $\pm$ 47.57	236.04 $\pm$ 63.30	227.09 $\pm$ 58.11	0.000*	0.647

\*: p&lt;0.05, control group compared to pre-dialysis groups (C-pre); \*\*: p&lt;0.05, pre-dialysis group compared to post-dialysis groups (pre-post).

acid losses during dialysis. As a result, both the quantity and quality of protein intake become particularly important. While protein restriction is generally recommended for individuals not yet on dialysis, those undergoing dialysis typically require more protein to compensate for increased catabolism and losses through the treatment [12].

Nutritional status is closely tied to clinical outcomes in CKD. Although serum albumin is widely used as an indicator of nutritional health, plasma amino acid levels may offer additional insights. Previous studies have shown that amino acids such as glutamine, homocysteine, and glutamate are associated with nutritional status. For example, a study in-

volving children with stage 4–5 CKD found significantly higher glutamine levels compared to healthy peers [13]. Similarly, our own findings revealed elevated plasma glutamine levels in patients with renal failure. Since the kidneys play a central role in glutamine metabolism, particularly in ammonia production for acid-base balance, these elevations may reflect impaired utilization in CKD. In support of our findings, Yardım et al. [8] demonstrated in their study that metabolic biomarker levels are altered in hemodialysis patients; this suggests that amino acid metabolism associated with diabetic and inflammatory processes may be affected in chronic kidney failure and during hemodialysis [8].

Branched-chain amino acids (BCAAs), including leucine and valine, are also considered markers of nutritional status. One study reported reduced levels of these amino acids in early-stage CKD patients compared to healthy controls [14]. In contrast, our findings showed higher concentrations of leucine and valine in CRF patients, which may be explained by decreased renal clearance, metabolic adaptations, or effects related to dialysis.

The impact of dialysis on BCAA levels is also noteworthy. Deb Nath et al. [15] observed a significant decrease in plasma BCAA concentrations following hemodialysis, and a negative correlation between post-dialysis BCAA levels and fatigue. In line with this, we found that leucine and valine levels declined after dialysis, while isoleucine levels remained stable. This may indicate selective removal of certain amino acids during treatment.

Aromatic amino acids such as phenylalanine, tyrosine, and tryptophan are known precursors of uremic toxins like p-cresol sulfate and indoxyl sulfate. An animal study by Barba et al. [16] showed that diets low in protein and aromatic amino acids reduced renal inflammation, fibrosis, and uremic toxin levels. These findings suggest that modifying amino acid intake may help slow CKD progression without worsening nutritional status.

It has also been reported that phenylalanine-to-tyrosine conversion is impaired in CKD, leading to elevated phenylalanine and potentially lower tyrosine levels [17]. Consistent with this, we observed increased phenylalanine levels in CRF patients. However, tyrosine concentrations remained similar between groups, which might reflect individual metabolic variability.

Tryptophan metabolism is also altered in CKD. A metabolomics-based study in the general population linked elevated levels of citrulline, kynurenine, and phenylalanine with CKD risk, and highlighted the kynurenine-to-tryptophan ratio as a relevant marker [18]. In agreement with these findings, our study showed lower tryptophan levels in CRF patients, both before and after dialysis, suggesting disrupted tryptophan metabolism.

As kidney function declines, changes in serum amino acid profiles become more evident. One study found that alanine, tyrosine, and valine levels decreased with renal dysfunction, whereas phenylalanine and citrulline increased [19]. Our data partially align with this, as we observed elevated levels of phenylalanine, citrulline, alanine, valine, and tyrosine. These discrepancies may stem from differences in patient populations, dietary habits, or treatment status.

In contrast to earlier reports that showed increased cystine levels in CKD [20], we found that cystine concentrations were lower in our patient group. This may be due to altered sulfur amino acid metabolism or increased utilization under oxidative stress, which is commonly observed in CKD.

Low-protein diets (LPDs) are often used to reduce the generation of uremic toxins. Ariyanopparut et al. [21] reported that combining LPDs with ketoanalog supplementation (LPD-KAs) delayed the progression of CKD and postponed the need for dialysis. Patients who adhered to higher doses of ketoanalogs saw more benefit, although no significant changes were seen in phosphate levels or albuminuria.

This study has several limitations that should be considered. First, the sample size was relatively small and the study was conducted at a single center, which may limit the generalizability of the findings. Dietary intake and nutritional status were not assessed, and inflammatory markers such as CRP and IL-6 were not measured, which could have provided additional context for amino acid changes. Adjustment for dialysis membrane type and dose was not performed, which may influence amino acid levels. Although dialysis adequacy was assessed using the urea reduction ratio (URR), individual URR values were not reported. This was considered acceptable because the study primarily focused on amino acid changes, and all patients met standard adequacy criteria, ensuring that dialysis efficiency was within acceptable ranges. Additionally, the study design involved single time-point sampling, which may not capture intra-individual variability. These limitations should be taken into account when interpreting the results and highlight areas for future research.

## Conclusion

Chronic kidney disease has a high prevalence in the general population and is associated with increased mortality. Therefore, more reliable biomarkers are essential for accurate diagnosis, monitoring disease progression, and guiding treatment strategies. Plasma amino acid levels reflect metabolic alterations and correlate with renal function. In this study, we evaluated the changes in amino acid profiles in patients with renal failure before and after dialysis, comparing them to a healthy control group, with these results detailed in the findings section.

Once the diagnosis and underlying cause of CKD are established, amino acid analysis can provide critical insights into the metabolic disturbances associated with the disease. Detecting specific amino acid imbalances or deficiencies enables clinicians to customize nutritional interventions or supplementation, potentially improving patient outcomes and slowing disease progression.

However, a larger sample size is required to validate these findings. Current literature on the relationship between amino acid biomarkers and chronic renal failure remains limited, highlighting the need for further research in this area.

## Disclosures

**Ethics Committee Approval:** The study was approved by the Harran University Clinical Research Ethics Committee (no: 20, date: 29/11/2021).

**Informed Consent:** Informed consent was obtained from all participants.

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