



Sawa Medical
Journal

Impact of Arginine, Taurine, and Tryptophan on Neutrophil Function in Renal Failure Patients in Al-Muthanna City

Seher abdulkhadim seher¹; Ahmed Waleed Dheyab Alnassar²; Hayder Raad Abbas³ and Mohammed Jasim Mohammed Shallal³

¹Lecturer Assistant, Department of Medical Laboratories, College of health and Medical Technology, Sawa University, Al-muthanna, Iraq.

²Bacteriologist at microbiology laboratory, Gynecology and Pediatrics Teaching Hospital in Al-Muthanna Governorate.

³Department of Microbiology, College of Medicine, University of Thi-Qar, Thi-Qar 64001, Iraq.

Abstract

Background: Amino acids are the basic building blocks of proteins. Amino acids are protein-building ingredients that may be derived from dietary sources or via the protein synthesis process. Protein synthesis employs a set of twenty-two isotropic amino acids. Although twenty of these 22 amino acids are required for protein synthesis, the other eighteen are mostly used as building blocks for other proteins.

Aim: The present study aimed to investigate the effects of the amino acids arginine, taurine, and tryptophan on human neutrophil functions in patients with renal failure.

keywords: complete blood count(CBC), White Blood Cells (WBC)

Introduction

Amino acids are fundamental components of proteins and are essential for human growth and development. They are the building blocks of the body, containing nitrogen, which is absent in fatty acids or sugars. Proteins are vital for all living organisms and play key roles in numerous biochemical processes essential for life. The human body requires over 1,600 different proteins, each of which has 22 amino acids (1,2). After digestion, protein is reduced to its component amino acids; eight of these acids are deemed essential and cannot be made by the body; the other 21 amino acids are less crucial and may be made by the body. Diseases and conditions may worsen when even a single amino acid is missing from the body. Amino acid shortage is common and may be caused by a lack of protein in the diet. Furthermore, the body's chemical imbalances, ageing, trauma, illness, and therapy all have a role (3,4). The decline in renal function correlates with a reduced ability to resist infections, though the exact mechanisms remain unclear. Patients with renal failure, especially those on dialysis, often have neutrophil function abnormalities, making them more vulnerable to

infections. These immune deficiencies complicate the treatment of Gram-negative septicemia, leading to significant morbidity and mortality despite available antimicrobial therapies. Malnutrition may contribute to these impairments, with neutrophil dysfunction frequently being an early indicator. Intravenous nutritional support may help restore neutrophil function and reduce infection-related issues (5,6). Arginine, taurine, and tryptophan are investigated for their potential to modulate neutrophil function in renal failure patients. Arginine is crucial for nitric oxide production, promoting immune cell proliferation, and providing antimicrobial defences. Taurine preserves membrane integrity and cellular calcium levels while acting as an antioxidant. Tryptophan's catabolism affects immune responses by influencing T-cell activity (7). Arginine is crucial for immune cell function, influencing neutrophil chemotaxis, phagocytosis, and oxidative burst. Administering it in animal models prevents thymic involution and boosts lymphocyte counts. Clinical trials indicate advantages for high-risk surgical and trauma patients, though effects on critically ill or septic patients remain unclear. Taurine enhances neutrophil activity and reduces inflammation, with its supplementation linked to better renal failure outcomes. Tryptophan metabolism affects neutrophil behaviour and immune modulation, with its availability possibly influencing patient outcomes. The combined roles of arginine, taurine, and tryptophan on neutrophils suggest potential therapeutic strategies for immune deficits in renal failure (7,8,9).

Methods

Participants and study design

The samples were obtained from laboratories at Al-Hussein Teaching Hospital in Al-Muthanna Governorate, Iraq and categorized into three groups: two groups comprised individuals with kidney failure, and the third group served as a control. The initial two groups of samples were categorized based on the type of kidney failure, while the third group comprised 30 samples from individuals without kidney failure, along with 27 and 42. The participants were categorized into two groups based on their type of failure, which encompassed blood pressure issues and 42 individuals with chronic kidney disease.

Included criteria

The study categorized patients according to specific criteria. All patients participating in this study are using specific data pertaining to their needs. The following patients were enrolled in the study and had their information gathered, as detailed in the questionnaire: The study also collected a medical history from the patient's family. Date of birth

Excluded criteria

The study established specific inclusion criteria for patients based on age groups, and it excluded patients 65 years old or older. The study also excluded patients with autoimmune diseases or chronic diseases.

Samples Collection

This study includes 4 ml of control and patient whole blood samples. The samples are centrifuged for 5 minutes at 4000 rpm.

Urea and Creatinine Investigation

Urea and Creatinine were measured and detected according to spectrophotometer kit instructions (Erba).

Statistical Analysis:

Data were gathered, summarized, analyzed, and presented using the Statistical Package for the Social Sciences (SPSS) version 23 and Microsoft Office Excel 2010. Qualitative (categorical) variables were represented as counts and percentages, while quantitative (numeric) variables were initially assessed for normality using the Kolmogorov-Smirnov test. Normally distributed numeric variables were reported as mean (a measure of central tendency) and standard deviation (a measure of dispersion), whereas non-normally distributed numeric variables were reported as median (a measure of central tendency) and interquartile range (a measure of dispersion).

Ethical considerations:

Participation in the study was subject to informed consent. All participants had renal failure. This protocol was approved by the Board of the Research Ethics Committee of the Health Office in Al-Muthana Governorate. Informed consent was obtained from all patients and members of the control group. All samples taken from those patients are under the direct supervision of specialist physicians in hospitals.

Results

Table 1: General Characteristics of the Patients.

	Group 1 ($n = 27$)	Group 2 ($n = 42$)	Group 3 ($n = 30$)	P
Age (years)	54.5 ± 13.9	46.9 ± 13.1	43.6 ± 8.9	0.002
Height (cm)	162.8 ± 8.4	163.6 ± 10.1	165.9 ± 7.1	0.566
Weight (kg)	73.7 ± 9.5	78.2 ± 18.9	75.4 ± 14.7	0.675
BMI (kg/m^2)	27.6 ± 3.2	28.9 ± 7.5	27.3 ± 4.8	0.330
Gender (M/F)	10/17	18/24	9/21	0.539
SBP (mmHg)	128.8 ± 14.1	123.9 ± 14.5	115.1 ± 13.2	0.001
DBP (mmHg)	77.5 ± 8.7	79.6 ± 7.4	76 ± 9	0.089
ACE inhibitors	7	4	0	0.000
CaCB	5	12	0	0.000

Table 2: Limit Characteristics between Patients and Control Groups.

Parameters	Group 1 (n = 27)	Group 2 (n = 42)	Group 3 (n = 30) control	P
Urea (mg/dl)	63.8 ± 33.4	72.6 ± 31.4	25.3 ± 6.3	<0.001
Creatinine (mg/dl)	1.7 ± 0.47	2.4 ± 1.25	0.73 ± 0.1	<0.001
Uric acid (mg/dl)	6.6 ± 1.3	6.8 ± 1.6	4.5 ± 1.3	<0.001
Albumin (g/dl)	4.01 ± 0.3	3.9 ± 0.36	4.03 ± 0.21	0.397
e GFR (ml/min/1.73 m ²)	41.77 ± 10.72	36.45 ± 14.41	96.3 ± 12.21	<0.001

Table 3: Blood Count Results.

Parameters	Group 1 (n = 27)	Group 2 (n = 42)	Group 3 (n = 30) control	P
Hemoglobin (g/dl)	12.8 ± 2.3	12.6 ± 2.4	11.9 ± 1.4	0.02
Hematocrit (%)	35.8 ± 4.91	36.8 ± 4.88	39.88 ± 4.35	0.008

Platelet (mm ³)	230.77 ± 47.95	271.14 ± 71.49 ^a	247.2 ± 55.99	0.029
Mean platelet volume (fl)	8.23 ± 1.06	8.4 ± 1.30	9.99 ± 13.66	0.408
White blood count (μl)	7330.03 ± 2012.12	7700.38 ± 2,362.05	6883.33 ± 1,674.36	0.262
Neutrophil(μl)*10 ³	68.88 ± 3.4a	67.88 ± 3.3a	61.82 ± 1.59	0.031
Platelet/lymphocyte	0.11 ± 0.02	0.14 ± 0.045	0.11 ± 0.03	0.004
Neutrophil/lymphocyte	7.34 ± 5.93	7.6 ± 5.80 ^b	2.9 ± 1.5	0.016

Table 4: Effect arginine on neutrophil elevation.

Parameter	value after admission G1	value after admission G2
Neutrophil (μl) 10 ³	55.1 ± 2.82b	47.61± 1.52c

Platelet/lymphocyte	280.6 ± 84.2	289.3 ± 83.8
Neutrophil/ly (ul)	2.8 ± 1.7	2.9 ± 1.7
Hemoglobin (g/dl)	11.8 ± 1.4	11.8 ± 1.4
Urea (mg/dl)	27.34 ± 1.87	22.58 ± 2.11
Creatinine (mg/dl)	$0.732 \pm 0.02a$	$0.71 \pm 0.07a$

Discussion

The results demonstrated that elevated neutrophil counts significantly affect the current mortality rate. A total of 69 patients were studied, and all relevant analyses were performed on their urea and creatinine levels, neutrophil ratios, and the values of these components. It was discovered that there is an imbalance between the two, and urea levels can rise. When creatinine levels rise to dangerously high levels—70 or even 200—it usually means that there are major health issues going on in the body, such as kidney failure (10,11).

Conditions like high blood pressure slowly damage the kidneys, leading to a medical emergency known as kidney failure. Treating this problem may be possible with amino acid therapy. Increases in neutrophil concentrations and numbers are the result of these acids' direct and indirect effects. This results in improved kidney function compared to pre-damage levels when a particular kidney disease prevents the kidneys from performing their function for long periods of time, months or even years. As damage accumulates, kidney function declines and chronic injury sets in (12,13,14,15).

The decline in neutrophil function was a significant factor, and urea plasma was also outside of the voluntary range. A rise in the neutrophil rate, normalisation of function, and a decrease in neutrophil elevation were all seen after adding tryptophan. Furthermore, statistical analysis revealed an inverse association between neutrophil levels before and after amino acid addition, suggesting that amino acids significantly and

effectively raise neutrophil levels and improve renal failure severity. Assessing the muscular function of individuals with renal failure revealed a significant rise, reaching 7480.4 ± 2812.2 ; in the second group, it reached $68.88 \pm 3.4a$ G1 and $67.88 \pm 3.3a$ G2. Arginine had a ratio of $55.1 \pm 2.82b$ and $47.61 \pm 1.52c$, whereas tryptophan had a ratio of $56.2 \pm 2.9b$ and $48.8 \pm 1.62c$ in the second group, with the natural neutrophil ratio acting as the antagonist (16,17,18,19).

Conclusion

Neutrophil function changes in renal failure patients play a crucial role in disease progression.

References

- 1-Lopez MJ, Mohiuddin SS. Biochemistry, essential amino acids. StatPearls [Internet]. 2024.
- 2-He W, Connolly ED, Cross HR, Wu G. Dietary protein and amino acid intakes for mitigating sarcopenia in humans. *Critical Reviews in Food Science and Nutrition*. 2025 May 11;65(13):2538-61.
- 3-Ling ZN, Jiang YF, Ru JN, Lu JH, Ding B, Wu J. Amino acid metabolism in health and disease. *Signal Transduction and Targeted Therapy*. 2023 Sep 13;8(1):345.
- 4- Kiani AK, Dhuli K, Donato K, Aquilanti B, Velluti V, Matera G, Iaconelli A, Connelly ST, Bellinato F, Gisondi P, Bertelli M. Main nutritional deficiencies. *Journal of preventive medicine and hygiene*. 2022 Oct 17;63(2 Suppl 3): E93.
- 5- Wang Y, Gao L. Inflammation and cardiovascular disease associated with hemodialysis for end-stage renal disease. *Frontiers in Pharmacology*. 2022.
- 6- Romejko K, Markowska M, Niemczyk S. The review of current knowledge on neutrophil gelatinase-associated lipocalin (NGAL). *International journal of molecular sciences*. 2023 Jun 21;24(13):10470.
- 7- Ma Y, Zhang Y, Li R, Deng S, Qin Q, Ran C, Hao Y, Zhang J, Zhu L. Mechanism of taurine reducing inflammation and organ injury in sepsis mice. *Cellular Immunology*. 2022 May 1;375:104503.
- 8- Qiu S, Li X, Zhang J, Shi P, Cao Y, Zhuang Y, Tong L. Neutrophil membrane-coated taurine nanoparticles protect against hepatic ischemia-reperfusion injury. *European Journal of Pharmacology*. 2023 Jun 15;949:175712.
- 9- Ma J, Yang Z, Jia S, Yang R. A systematic review of preclinical studies on the taurine role during diabetic nephropathy: focused on anti-oxidative, anti-inflammation, and anti-apoptotic effects. *Toxicology Mechanisms and Methods*. 2022.
- 10-Huang J, Ladeiras D, Yu Y, Ming XF et al. Detrimental Effects of Chronic L-Arginine Rich Food on Aging Kidney. 2021.
- 11- Lo HC, Hung CY, Huang FH, Su TC et al. The Nitric Oxide Synthase Inhibitor N(G)-Nitro-L-Arginine Methyl Ester Diminishes the Immunomodulatory Effects of Parental Arginine in Rats with Subacute Peritonitis. 2016.
- 12- Li YX, Lu YP, Tang D, Hu B, Zhang ZY, Wu HW, Fan LJ, Cai KW, Tang C, Zhang YQ, Hong L. Anthocyanin improves kidney function in diabetic kidney disease by regulating amino acid metabolism. *Journal of translational medicine*. 2022 Nov 5;20(1):510.
- 13- Landoni G, Monaco F, LK T, Baiardo Redaelli M, Bradic N, Comis M, Kotani Y, Brambillasca C, Garofalo E, Scandroglio AM, Viscido C. A randomized trial of intravenous amino acids for kidney

protection. *The New England journal of medicine*. 2024;391(8):687-98.

14- Jiang W, Shi K, Shao J, Song L, Shi Y, Wang H, Zhou L, Li L, Feng Y, Yu J, Zheng R. Protective effect of intravenous amino acid on kidney function: A systematic review and meta-analysis of randomized controlled trials. *Journal of critical care*. 2025 Feb 1;85:154937.

15- Knol MG, Wulfmeyer VC, Müller RU, Rinschen MM. Amino acid metabolism in kidney health and disease. *Nature Reviews Nephrology*. 2024 Dec;20(12):771-88.

16- Liu X, Li Q, Zhang L, He Y et al. Impacts of amino acid supplementation on renal function and nutritional parameters in patients with renal insufficiency: bibliometric analysis and meta-analysis. *Frontiers in Nutrition*. 2025.

17- Hsu HJ, Yen CH, Wu IW, Liu MH, Cheng HY, Lin YT, Lee CC, Hsu KH, Sun CY, Chen CY, Wang CH. The association between low protein diet and body composition, muscle function, inflammation, and amino acid-based metabolic profile in chronic kidney disease stage 3–5 patients. *Clinical Nutrition ESPEN*. 2021 Dec 1;46:405-15.

18- Liu L, Xu J, Zhang Z, Ren D, Wu Y, Wang D, Zhang Y, Zhao S, Chen Q, Wang T. Metabolic homeostasis of amino acids and diabetic kidney disease. *Nutrients*. 2022 Dec 30;15(1):184.

19- Hui Y, Zhao J, Yu Z, Wang Y, Qin Y, Zhang Y, Xing Y, Han M, Wang A, Guo S, Yuan J. The role of tryptophan metabolism in the occurrence and progression of acute and chronic kidney diseases. *Molecular nutrition & food research*. 2023 Nov;67(21):2300218.