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Association Between Body Mass Index and Serum Urea Levels Among Women of Reproductive Age in Waihaong Village, Maluku, Indonesia

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ABSTRACT

Chronic kidney disease (CKD) is a growing global health concern, highlighting the importance of identifying modifiable risk factors that affect renal function. Nutritional status, commonly reflected by Body Mass Index (BMI), is one such factor, while serum urea serves as a key biomarker of kidney function. This study examined the relationship between BMI and serum urea levels among women of reproductive age in Waihaong Village. Using a cross-sectional analytical design, 75 participants were selected through purposive sampling. Data collection included informed consent, 10–12 hours fasting, BMI measurement, venous blood sampling, and serum urea analysis. Data were processed with SPSS using the Spearman correlation and multiple regression tests. Results revealed a significant positive correlation between BMI and serum urea levels ($r = 0.259$, $p = 0.025$). The median serum urea concentration was 18.3 mg/dL (range: 12.8–37.4 mg/dL), remaining within normal limits. The Kruskal–Wallis test showed no significant difference among BMI categories ($\chi^2 = 7.792$, $p = 0.100$), although mean rank values indicated a trend of increasing urea with higher BMI. Multiple linear regression indicated that BMI significantly influenced serum urea ($\beta = 1.045$, $p = 0.031$) after adjusting for diet and physical activity, while other variables showed no significant associations ($p > 0.05$). The model's stability was confirmed (VIF < 2). In conclusion, higher BMI is associated with increased serum urea levels, suggesting the need for routine BMI screening to help detect early kidney dysfunction in women of reproductive age.

Keywords
Body Mass Index
Kidney Function
Ureum Levels
Women of Reproductive Age

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Introduction

The kidneys are vital organs that play a central role in maintaining fluid balance and excreting metabolic by-products; however, their function is highly susceptible to various risk factors that may lead to chronic kidney disease (CKD). Currently, CKD is recognized as a major global health problem, affecting more than 850 million people worldwide, and is among the fastest-growing causes of mortality [1]. In Indonesia, the prevalence of CKD in 2023 reached 0.18% (approximately 638,718 cases), with Maluku Province reporting a slightly higher prevalence (0.21%) compared to the national average [2].

Nutritional status, commonly assessed through Body Mass Index (BMI), is one of the key factors influencing renal function [3]. Renal function can be assessed using various parameters, such as glomerular filtration rate (GFR), serum creatinine, and urea, as well as other biomarkers including cystatin C and β2-microglobulin. Among these, urea is one of the most commonly used parameters due to its simplicity and wide availability [4]. Urea is a nitrogen-containing compound that represents the final product of protein metabolism in the liver and the urea cycle. It is excreted through the kidneys, and its concentration depends on the efficiency of glomerular filtration. Within the glomerulus, the filtration barrier—composed of the capillary endothelium, basement membrane, and podocytes—selectively filters blood based on molecular size and charge. Because urea molecules are small, uncharged, and freely filtered almost completely with plasma flow, plasma urea concentration serves as a reliable indicator of the glomerular filtration rate (GFR). A reduction in GFR, as seen in chronic kidney disease, impairs urea elimination, resulting in elevated serum urea levels [5]. In healthy populations, urea measurement is also valuable for detecting subclinical renal impairment [6].

Elevated BMI increases renal workload through mechanisms such as glomerular hyperfiltration, insulin resistance, activation of the renin–angiotensin–aldosterone system (RAAS), and chronic low-grade inflammation, all of which contribute to structural damage and progressive decline of kidney function. Conversely, low BMI is associated with malnutrition and muscle loss, both of which impair renal function. Previous studies have shown a positive correlation between BMI and serum urea, suggesting that higher BMI tends to elevate urea levels [7][8].

In underweight individuals, however, serum urea may decrease in chronic protein deficiency or increase under catabolic stress, dehydration, or renal impairment.

Epidemiological evidence also indicates that underweight is associated with a higher risk of end-stage renal disease, meaning it cannot be considered protective [9].

Women of reproductive age present unique physiological and hormonal characteristics that may influence kidney function. Nevertheless, research specifically investigating the relationship between BMI and serum urea among women of reproductive age remains limited. Local health data from Waihaong Village, Ambon, revealed a significant increase in obesity cases among women, indicating a rising trend in BMI. This condition may contribute to impaired renal function, as reflected in elevated serum urea levels. Therefore, investigating the association between BMI and serum urea in women of reproductive age is essential for early detection of kidney dysfunction and for developing preventive strategies and community health education.

Material and Methods

A. Research design

This study employed an observational analytic design with a cross-sectional approach, conducted in Waihaong Village, Nusaniwe District, Ambon City, from July 16 to 25, 2025. The target population consisted of women aged 20–35 years, with purposive sampling applied based on inclusion and exclusion criteria. Using a correlation sample size formula, the minimum required sample was 48; however, 75 participants were enrolled. Inclusion criteria were women aged 20–35 years, normotensive, and willing to fast 10–12 hours prior to venous blood collection, while exclusion criteria included pregnancy, smoking, alcohol consumption, use of nephrotoxic medications or hormonal contraception, diabetes mellitus, or pre-existing kidney disease. These inclusion and exclusion criteria were designed to minimize potential confounding factors.

B. Measurement of Body Mass Index (BMI)

Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2) and classified according to the Indonesian Ministry of Health guidelines [10] as follows: severely underweight (<17.0 kg/m^2), underweight (17.0–18.5 kg/m^2), normal (18.5–25.0 kg/m^2), overweight (25.0–27.0 kg/m^2), and obese (>27.0 kg/m^2).

C. Collection of blood and sample processing

All study subjects were required to fast for 10–12 hours. The blood collection procedure was conducted in the morning at 8:00 a.m. Venous blood collection began with the application of a tourniquet approximately 10 cm above the antecubital fossa, followed by palpation of the venous area to locate the vein. The puncture site was disinfected using 70% alcohol swabs and allowed to air dry. The cleaned area was not touched afterward. The vein was punctured with the needle bevel facing upward at a 30-degree angle. Once blood appeared in the indicator, the syringe plunger was pulled until the required volume was obtained. The tourniquet was then

released, and an adhesive bandage was applied to the puncture site. The collected blood in the syringe was transferred into a vacutainer tube and subsequently centrifuged at 3,000 rpm for 15 minutes. Serum urea levels were analyzed using the Biosystem BA200 Biochemistry Analyzer following standard laboratory protocols.

D. Statistical analysis

Data analysis was performed using IBM SPSS Statistics version 25. Univariate analysis was conducted to describe participants' characteristics. In this study, the Body Mass Index (BMI) variable was categorized as ordinal data, whereas the serum urea level variable was classified as ratio data; therefore, a normality test was initially performed. The Kolmogorov–Smirnov method was applied since the sample size exceeded 50. The normality test results for serum urea levels showed a value of 0.047 ($p < 0.05$), indicating that the data were not normally distributed. Accordingly, the Spearman's rank correlation test was employed to assess the association between BMI and serum urea levels. The Kruskal–Wallis test to compare median serum urea levels among different BMI categories. Furthermore, multiple linear regression analysis was conducted to control for potential confounding variables such as dietary patterns and physical activity, to identify whether BMI independently predicted serum urea levels. A p-value < 0.05 was considered statistically significant.

E. Research ethics

Ethical approval was obtained from the Health Research Ethics Committee, Faculty of Medicine, with approval number No.133/FK-KOM-ETIK/VII/2025. Informed consent, confidentiality, and anonymity of all participants were ensured throughout the study.

Results

As presented in Table 1, the majority of participants were aged 20–24 years (n = 41; 54.7%), followed by those aged 25–29 years (n = 18; 24.0%) and 30–35 years (n = 16; 21.3%). In total, 75 women of reproductive age were included in the study.

Table 1. Distribution by Age Group (n=75)

Age	Frequency	%
20 – 24 years	41	54.7
25 – 29 years	18	24.0
30 – 35 years	16	21.3
Total	**75**	**100**

Table 2 presents the frequency distribution of participants based on dietary and beverage consumption habits. The majority of participants reported consuming sweet foods 1–2 times per week (n = 24, 32.0%), followed by once daily (n = 15, 20.0%) and 3–6 times per week (n = 13, 17.3%). In addition, 9 participants (12.0%) consumed sweet foods more than once per day, 11 participants (14.7%) consumed them less than three times per month, and 3 participants (4.0%) reported never consuming sweet foods. Regarding sweetened beverages,

23 participants (30.7%) consumed them 1–2 times per week, 16 participants (21.3%) consumed them once daily, and 13 participants (17.3%) reported consuming them either 3–6 times per week or less than three times per month. Meanwhile, 6 participants (8.0%) reported never consuming sweetened beverages, and 4 participants (5.3%) consumed them more than once per day. In terms of fast food consumption, most participants reported consuming it 1–2 times per week (n = 23, 30.7%). This was followed by consumption 3–6 times per week and less than three times per month (n = 19, 25.3% for each category). A smaller proportion consumed fast food more than once per day (n = 2, 2.7%), once per day (n = 7, 9.3%), or never (n = 5, 6.7%).

Table 2. Distribution of Sweet Food, Sweet Beverage, and Fast Food Consumption Patterns (n=75)

Description	Sweet Food Consumption		Sweetened Beverages Consumption		Fast Food Consumption	
	Frequency	%	Frequency	%	Frequency	%
> Once per day	9	12.0	4	5.3	2	2.7
Once per day	15	20.0	16	21.4	7	9.3
3 – 6 times per week	13	17.3	13	17.3	19	25.3
1 – 2 times per week	24	32.0	23	30.7	23	30.7
< 3 times per week	11	14.7	13	17.3	19	25.3
Never	3	4.0	6	8.0	5	6.7
Total	**75**	**100**	**75**	**100**	**75**	**100**

Table 3 shows that the majority of participants did not engage in weight-lifting activities (n = 49; 65.3%), while 26 participants (34.7%) reported performing such activities. In contrast, most participants engaged in walking activities (n = 59; 78.7%), whereas 16 participants (21.3%) reported not engaging in walking.

Table 3. Frequency Distribution of Physical Activity Patterns (n=75)

Description	Weight Lifting*		Walking**	
	Frequency	%	Frequency	%
Yes	26	34.7	59	78.7
No	49	65.3	16	21.3
Total	**75**	**100**	**75**	**100**

n = number of study participants.
*Weight lifting/carrying heavy loads (≥ 20 kg) repeatedly for at least 10 minutes continuously
** Walking for a minimum of 10 minutes continuously

Table 4 shows that the majority of participants had a normal BMI (n = 46; 61.3%), followed by obesity (n = 13; 17.3%), severe underweight (n = 8; 10.7%), overweight (n = 5; 6.7%), and underweight (n = 3; 4.0%).

Table 4. Frequency Distribution of Body Mass Index (n=75)

BMI Category	Frequency	%
Severely Underweight	8	10.7
Underweight	3	4.0
Normal	46	61.3
Overweight	5	6.7
Obesity	13	17.3
Total	**75**	**100**

Table 5 presents the results of the univariate analysis, indicating that all participants (100%) had serum urea levels with a median value of 18.3 mg/dL and a range of 12.8–37.4 mg/dL.

Table 5. Frequency Distribution of Median Serum Urea Levels According to Body Mass Index Category (n = 75)

Body Mass Index Category	Urea Levels median (mg/dL)	Frequency	%
Severely Underweight	14.45	8	10.7
Underweight	15.00	3	4.0
Normal	18.35	46	61.3
Overweight	19.20	8	6.7
Obesity	20.10	13	17.3
Total	**18.30**	**75**	**100**

Table 6 presents the results of the Spearman correlation test, demonstrating a statistically significant association between BMI and serum urea levels, with a p-value of 0.025 ($p < 0.05$). The correlation coefficient ($r = 0.259$) indicates a weak positive correlation, suggesting that higher BMI is associated with slightly elevated serum urea levels.

Table 6. Bivariate Analysis of the Association Between Body Mass Index and Serum Urea Levels (n=75)

Serum Urea (mg/dL)	Body Mass Index					*p-value*	R
	Severely Underweight	Underweight	Normal	Overweight	Obesity		
Median	14.45	15.00	18.35	19.20	20.10	0.025	0.259
Min-Max	12.8 – 20.1	14.9 – 21.0	12.8 – 29.5	13.6 – 20.6	13.1 – 37.4		

Table 7 presents the comparison of urea levels across different BMI categories using the Kruskal–Wallis test. The analysis compares the mean ranks of urea levels among BMI groups to determine whether significant differences exist.

Table 7. Comparison of urea levels among BMI categories (Kruskal-Wallis test)

Category	BMI	n	Mean Rank	p-value
Urea Levels	Severly Underweight	8	18.69	0.100
	Underweight	3	32.33	
	Normal	46	39.85	
	Overweight	5	40.20	
	Obesity	13	43.81	
Total		**75**		

Based on the Kruskal–Wallis test, there was no significant difference in urea levels among the different BMI groups (p = 0.100). The mean rank values of urea levels in the severely underweight, underweight, normal, overweight, and obese groups were 18.69, 32.33, 39.85, 40.20, and 43.81, respectively.

Table 8 presents the results of the multiple linear regression analysis examining the associations of BMI, dietary patterns, and physical activity with serum urea levels. The regression coefficients, confidence intervals, and significance values indicate the independent contribution of each predictor to variations in serum urea levels after controlling for the other variables.

Table 8. Multiple Linear Regression Analysis between BMI, Dietary Patterns, and Physical Activity with Serum Urea Levels

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	B		
1	(Constant)	17.455	3.513		4.968	.000
	BMI	1.045	.474	.255	2.202	.031
	Sweet Food Consumption	.497	.420	.151	1.185	.240
	Sweetened Beverages Consumption	-.328	.420	-.099	-.780	.438
	Fast Food Consumption	-.109	.451	-.028	-.241	.810
	Weight Lifting	-.201	1.087	-.021	-.185	.854
	Walking	-1.619	1.280	-.148	-1.265	.210

a. Dependent Variable: Urea Levels

Table 9 presents the summary of the multiple linear regression model. The values of R, R Square, and Adjusted R Square indicate the model's goodness of fit and the proportion of variance in serum urea levels explained by the predictors. The standard error of the estimate reflects the accuracy of the model's predictions.

Table 9. Model Summary

Model	R	R Square	Adjusted R-Square	Std. Error of the Estimate
1	.352[a]	.124	.046	4.4068

a. Predictors: (Constant), Walking, Body Mass Index (BMI), Weight Lifting, Sweet Drinks, Fast Food, Sweet Foods

b. Dependent Variable: Urea Levels

According to the multiple linear regression analysis, among the predictor variables (walking, body mass index [BMI], weight lifting, sweet food consumption, sweetened beverage consumption, and fast food consumption), only BMI demonstrated a statistically significant association with serum urea levels (p = 0.031). The coefficient of determination (R^2) was 0.124, indicating that approximately 12.4% of the variability in urea levels could be explained by the predictors included in the model, whereas the remaining variation is attributable to other

unaccounted factors. Other variables—such as sweet food consumption (p = 0.240), sweetened beverage consumption (p = 0.438), fast food consumption (p = 0.810), weight lifting (p = 0.854), and walking (p = 0.210)—did not exhibit statistically significant relationships with urea levels.

Discussion

In this study, the results of the Spearman correlation test showed a statistically significant association between Body Mass Index (BMI) and serum urea levels, with a p-value of 0.025 ($p < 0.05$) and a correlation coefficient (r) of 0.259. This indicates a weak positive but non-linear correlation, suggesting that an increase in BMI tends to be accompanied by a modest rise in serum urea levels. Although the strength of the correlation is low, this finding implies a physiological link between nutritional status and nitrogen metabolism [11],[12].

The Kruskal–Wallis test further supported this observation, showing no statistically significant difference in serum urea levels across BMI categories ($\chi^2 = 7.792$; $p = 0.100$). However, the mean rank values demonstrated a gradual upward trend from the underweight to obese categories, suggesting a tendency for higher urea levels with increasing BMI, even though the difference did not reach statistical significance. This pattern may be influenced by the limited sample size within each BMI category and interindividual biological variability [13].

Physiologically, an increase in BMI is often associated with enhanced protein metabolism and greater renal workload, reflected by slightly higher urea concentrations in overweight and obese individuals. This finding is consistent with Ude et al. [8], who reported that obesity is associated with elevated urea and creatinine levels due to chronic glomerular hyperfiltration and structural kidney changes. Similarly, Ara et al. [14] observed that women of reproductive age with BMI ≥ 18.5 kg/m^2 were more likely to have elevated urea levels compared to underweight women, even when the values remained within normal limits. Similar findings have been reported in epidemiological studies demonstrating that excess body weight is independently associated with impaired renal function and increased risk of chronic kidney disease [15],[16].

An increase in body mass index (BMI) may influence urea levels through complex hemodynamic and metabolic pathways. Hemodynamically, obesity induces vasodilation of the afferent arteriole and enhances sodium reabsorption in the proximal tubule, which triggers glomerular hyperfiltration and increased excretion of nitrogenous waste products such as urea. Metabolically, excessive adipose tissue functions as an active endocrine organ that secretes proinflammatory cytokines (such as TNF-α, IL-6, and leptin) with nephrotoxic properties, leading to oxidative stress and renal cellular injury. In addition, insulin resistance and activation of the renin–angiotensin system in obesity contribute to elevated intraglomerular pressure and accelerate the progression of glomerulosclerosis. The combination of these

processes suggests that elevated urea levels in individuals with higher BMI are not merely a consequence of increased body mass, but also reflect systemic metabolic alterations and chronic inflammation that impair renal filtration function [17]-[19].

The multiple linear regression analysis in this study revealed that, after controlling for behavioral factors such as sweet food intake, sweetened beverage intake, fast food consumption, and physical activity (walking and weight lifting), only BMI had a significant effect on serum urea levels ($\beta = 1.045$; $p = 0.031$). This indicates that each one-unit increase in BMI contributes to an average rise of 1.045 mg/dL in serum urea concentration. Meanwhile, other variables—sweet food, sweetened beverages, fast food, and physical activity—were not significantly associated with urea levels ($p > 0.05$). The absence of multicollinearity (VIF < 2) confirmed the stability of the regression model.

These regression findings reinforce the Spearman correlation results, suggesting that BMI is the main factor associated with serum urea levels, although its contribution is relatively small. The R^2 value of 0.124 indicates that only 12.4% of the variation in serum urea levels can be explained by the predictors in the model, while the remaining 87.6% is likely influenced by unmeasured factors such as daily protein intake, hydration status, liver function, and muscle mass [20].

From a physiological standpoint, individuals with low BMI typically have reduced protein and calorie intake, leading to decreased urea production from protein metabolism. According to the MSD Manual [21], Protein–Energy Undernutrition (PEU) is often accompanied by low serum urea levels due to reduced protein catabolism and hepatic urea synthesis. This aligns with the findings in the very low BMI and underweight groups in this study, where urea levels were lower but remained within normal ranges.

Conversely, individuals with higher BMI may exhibit increased urea production due to elevated protein turnover and higher metabolic demand, as well as greater renal filtration workload. Kermani et al. [22] reported that fast food consumption contributes to renal function decline through systemic inflammation, oxidative stress, and endothelial dysfunction, particularly in overweight individuals. Although serum urea concentrations in this study remained within normal limits, the gradual increase across BMI groups suggests an early metabolic adaptation of the kidneys to excess metabolic load [23].

In addition, physical activity variables such as walking and weight lifting showed no significant association with serum urea levels. This finding is consistent with the meta-analysis by Ma et al. [24], which noted that light-to-moderate exercise does not directly affect urea levels but nonetheless supports kidney health through improved renal perfusion, energy metabolism efficiency, and reductions in oxidative stress [25].

Conclusion

The findings of this study demonstrated that body mass index (BMI) is associated with blood urea levels among women of reproductive age. The majority of participants had a normal BMI, while a smaller proportion were classified as underweight, overweight, or obese. Although all urea values remained within the normal reference range, a statistically significant relationship was observed between BMI and urea levels, with a weak positive correlation. This suggests that while BMI influences blood urea levels, other factors such as diet, hydration, physical activity, and metabolic status also contribute. Preventive strategies focusing on weight management, healthy dietary patterns, and regular physical activity therefore remain essential to reduce the risk of renal impairment. These results highlight the importance of recognizing overweight and obese individuals as a population at risk, emphasizing the need for early detection and preventive measures. Routine BMI monitoring may serve as a simple and effective approach to identify individuals at higher risk and to reduce the likelihood of future complications related to excess body weight. These findings support the integration of routine BMI assessment into community-based and maternal health screening programs to promote early identification and prevention of kidney function decline.

Limitation and Suggestions

This study assessed BMI and urea levels at a single time point, which does not capture long-term variations and may be affected by uncontrolled factors such as diet and daily activity. Moreover, because of its cross-sectional design, the analysis was limited to correlational relationships without adjustment for other potential confounders. Consequently, temporal and causal links between BMI and renal function cannot be established, leaving it unclear whether increased BMI contributes to higher urea levels or if impaired renal function influences body composition. Several potential confounders, including age, dietary protein intake, hydration status, and physical activity, could have influenced the association between BMI and serum urea. Although participants with known renal or metabolic diseases were excluded, no statistical adjustment was performed for these variables.

Conflict of Interest

The authors declare no conflict of interest.

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