

Estimation of Urea, Creatinine, Uric Acid and Total Protein in Blood of Acute Renal Failure Patients in Basra Governorate

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Abstract

When your kidneys stop functioning on their own, you may have renal failure in one or both of them. Acute renal injury, diabetes, and high blood pressure are among the causes. The purpose of this research is to measure the levels of serum urea, creatinine, uric acid, and total protein in patients with renal failure in order to demonstrate the correlation between these parameters and the illness. Thirty of the sixty individuals were kidney failure patients, and the remaining thirty were normal patients. The patients were selected and admitted to several hospitals in Basra City between January and February of 2023. Enzymatic reaction techniques were used to determine the serum levels of urea, uric acid, creatinine, and total protein in all 60 patients. The results of this investigation showed that the sick group's serum levels of urea, creatinine, and uric acid were considerably ($p < 0.05$) higher than those of the normal group. In comparison to the control group, which had serum concentrations of 25.27 ± 5.12 , 1.63 ± 0.36 , 4.93 ± 0.33 , and 3.5 ± 0.81 mg/dL, the investigation revealed a rise in creatinine, urea, uric acid, and total protein in the serum of the suffering group (7.63 ± 4.28 , 102.12 ± 67.23 , 7.5 ± 1.93 , and 5.83 ± 2.7 mg/dL, respectively). The findings also indicated that as people aged, all parameter concentrations increased.

Keywords: Renal failure, kidney proteins, Urea, Uric acid, Creatinine

1. INTRODUCTION:

The kidneys play a crucial role in regulating blood pressure, maintaining the body's fluid balance, electrolyte levels, and acid-base balance, and filtering waste and toxins from the blood, which are then excreted into the urine. A potentially fatal condition or bodily injury could result from waste products building up in the blood and dangerously high fluid levels if the kidneys are not working correctly. Kidney injury can arise from a variety of ailments and diseases. Diabetes and hypertension are the two main risk factors and causes of renal disease (1).

Nephrons, which are approximately a million microscopic blood-filtering units, are located inside the kidneys. Blood is continuously filtered in each nephron by a glomerulus, a group of looping blood arteries that lets water and tiny molecules flow through while holding on to larger molecules, proteins like albumin, and blood cells (2).

Acute renal failure is a sudden decrease in kidney function that lasts for a few days to weeks, causing creatinine and nitrogenous wastes to be excreted in insufficient amounts. It's possible to experience electrolyte imbalances and loss of fluid homeostasis (3). A collection of examinations known as renal function tests (RFT) can be used in tandem to assess renal (or kidney) function. To evaluate the

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<https://doi.org/10.2478/9788368412543-024>

current state of the kidneys, the tests examine levels of various chemicals in the blood, including glucose (sugar), proteins, electrolytes, and other minerals. The four types of renal function tests include tubular function tests, glomerular function tests, blood examinations, and urine analyses (4).

As a byproduct of creatine phosphate breakdown in muscle, serum creatinine is typically produced by the body at a fairly consistent rate, based on muscle mass. Normal ranges for men and women are 0.8-1.3 mg/dL and 0.6-1.0 mg/dL, respectively (5). The primary nitrogenous byproduct of protein and amino acid breakdown is urea, which is produced by the liver and found in both intracellular and extracellular fluids. The glomeruli freely filter urea. Several renal disorders that cause injury to the glomeruli, tubules, interstitial spaces, or vessels can increase the concentration of plasma urea. In healthy adults, the standard range for serum urea is 10–40 mg/dl. Males also typically have slightly higher plasma concentrations than females do. Urinary excretion and plasma urea concentrations are significantly increased by high-protein diets (6).

The primary byproduct of adenosine and guanosine, two purine nucleosides, is uric acid. Purines are produced by the breakdown of endogenous nucleic acids and the catabolism of ingested nucleic acids. Increased synthesis of purine precursors may lead to an excess of uric acid production. Around 75% of the uric acid excreted by humans is lost in the urine; the majority is released into the GIT (7). Furthermore, the total protein content in a patient's serum or plasma should be ascertained, and its composition should then be examined by electrophoresis. Relative hyperproteinemia is caused by the drop in plasma water volume (haemoconcentration), which happens frequently when there is dehydration (acute vomiting, diarrhea, Addison's illness, or diabetic acidosis). Hypoproteinemia relative is the outcome of hemodialysis (increased plasma water volume), which can happen during intravenous infusion, in cases of water intoxication or salt retention syndromes, or physiologically in cases of extended bed rest. Frequently and for many causes, a drop in albumin concentration can also be the cause of hypoproteinemia. Infection or mild hyperproteinemia can also result from an increase in specific proteins. A substantial rise in monoclonal immunoglobulins produced during multiple myeloma or other malignant hyperparaproteinemia may be the cause of marked hyperproteinemia (8).

2. MATERIALS AND METHODS

2.1 Study area and study population

This study involved dialysis patients with kidney failure from Al-Sadr Teaching Hospital and Basra Teaching Hospital. In addition to a group of 30 healthy individuals (18 males and 12 females) without any other illnesses, the study also included 30 cases (10 males and 20 females) with kidney failure.

Sample gathering: Ten milliliters of blood were extracted from the venous blood of both groups using sterile medical syringes that were placed in anticoagulant-free special tubes and allowed to sit at room temperature for ten to thirty minutes. After the samples were separated into blood and serum, they were centrifuged for 10 minutes at 3000 cycles per minute. The serum was then stored at -20 °C until the analysis was completed, which included measurements of total protein, urea, creatinine, and uric acid for each of the following: urea, creatinine, uric acid, and total protein, as well as blocks from the French-based Biolabo company.

Sample collection and biochemical tests:

Serum used for biochemical measurements of kidney function, including urea (9) uric acid, and creatinine (10), total protein (11).

2.2 Statistical analysis:

The statistical analysis of the current study results was conducted using the 2008 statistical package (Social Science, version 20(SPSS)) program to investigate the impact of the materials used in the experiment and to identify any significant differences between the average probability level ($p \leq 0.05$) (12).

3. RESULTS AND DISCUSSION

In the current study, 60 patients, aged 16 to 70 years, were enrolled. Thirty subjects were classified as having kidney failure, and the remaining thirty comprised the control group. The overall characteristics of all participants in the study are presented in Table 1. Each participant was categorized based on their age, sex, and family background.

Table 1: Major clinical characteristics of all studied groups

Variables		Control	Patients
Total (No.)		30	30
Age (Years)	16-30	18	15
	31-50	7	8
	51- 70	5	7
	Male	18	12
Sex	Female	20	10
	Urinary failure	-	3
Family History	Non-Urinary failure	-	27

The statistical analysis revealed that the group of patients with chronic renal failure had higher concentrations of urea, creatinine, uric acid, and total protein when compared to the healthy group, as shown in Table 1 and Figures 1 and 2.

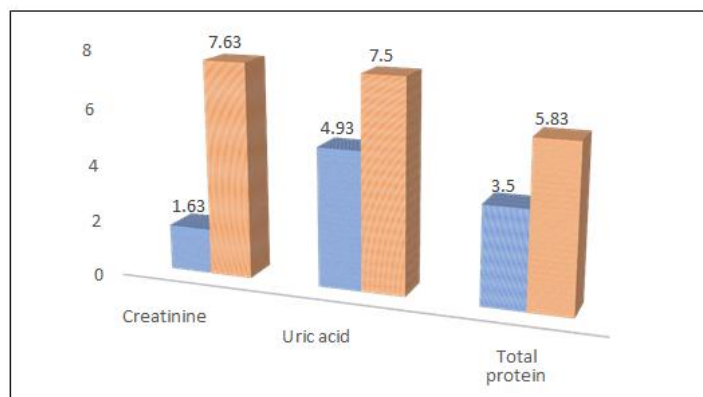


Figure 1. Creatinine, uric acid and total protein levels in serum of patients and control groups

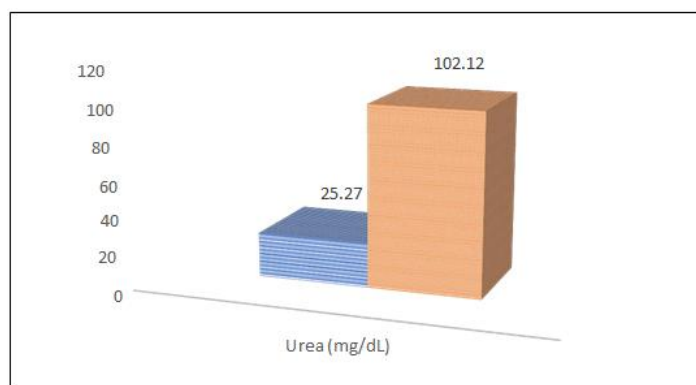


Figure 2. Urea levels in serum of patients and control groups

Table 2. Urea, creatinine, uric acid and total protein levels in serum of patients and controls

Parameters	Control No. = 30	Patients No.=30
Urea (mg/dl)	25.27 $\pm$ 5.12	102.12 $\pm$ 67.23***
Creatinine (mg/dl)	1.63 $\pm$ 0.36	7.63 $\pm$ 4.28***
Uric acid (mg/dl)	4.93 $\pm$ 0.33	7.50 $\pm$ 1.93**
Total protein(mg/dl)	3.5 $\pm$ 0.81	5.83 $\pm$ 2.7*

Values were expressed as mean $\pm$ SD

* The level of significance between patients and control

* P < 0.05, ** P < 0.01, *** P < 0.0001

Tables 3, 4, and Figures 3, 4, 5, and 6 illustrate how the analysis results for both groups were categorized based on age groupings.

Table 3. Relationship between age and level of urea, creatinine, uric acid and total proteins in serum of control

Age	Urea Mean $\pm$ SD mg/dl	Creatinine Mean $\pm$ SD mg/100ml	Uric acid mg/dl Mean $\pm$ SD	Total protein Mean $\pm$ SD g/dl
16- 30(No.=18)	22.32 $\pm$ 2.31	0.85 $\pm$ 0.01	3.41 $\pm$ 0.01	5.1 $\pm$ 0.31
31-50 (No.=7)	35.53 $\pm$ 3.7	1.3 $\pm$ 0.04	4.43 $\pm$ 0.06	3.7 $\pm$ 0.21
51-70(No.=5)	39.00 $\pm$ 5.79	0.9 $\pm$ 0.02	7.50 $\pm$ 0.05	2.2 $\pm$ 0. 5

Table 4. Relationship between age and level of urea, creatinine, uric acid and total proteins in serum of patients

Age	Urea Mean $\pm$ SD mg/dl	CreatinineMean $\pm$ SD mg/dl	Uric acid mg/dl Mean $\pm$ SD	Total protein Mean $\pm$ SD g/dl
16-30 NO.(15)	105.6 $\pm$ 4.78 ***	4.3 $\pm$ 0.78***	7.51 $\pm$ 1.9*	6.0 $\pm$ 0.01**
31-50 NO.(8)	183.07 $\pm$ 23.96 ***	7.6 $\pm$ 4.2***	12.9 $\pm$ 2.4*	6.6 $\pm$ 0.3**
51-70 NO.(7)	253.6 $\pm$ 87.8 ***	14.5 $\pm$ 0.09***	10 $\pm$ 0.96*	7.4 $\pm$ 0. 4**

Values were expressed as mean $\pm$ SD.

*The level of significance between the patients and the control groups

* P < 0.05, ** P < 0.01, *** P < 0.0001

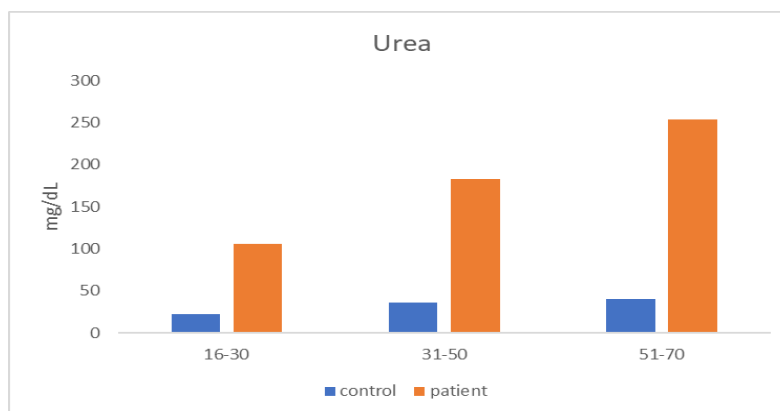


Figure 3. Relationship between age and urea levels in the serum of patients and control groups

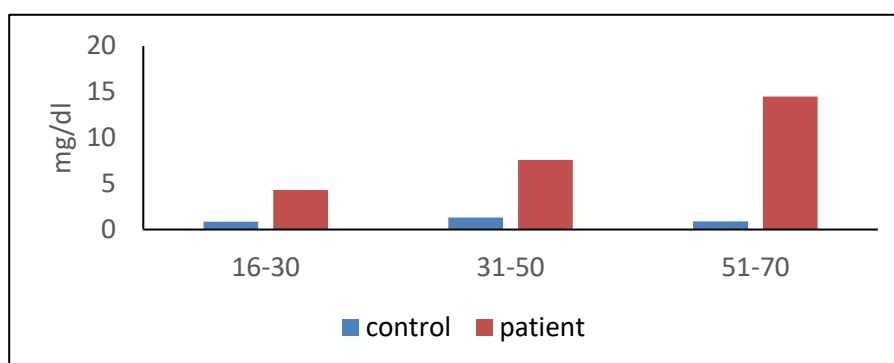


Figure 4. Relationship between age and Creatinine levels in the serum of patients and control groups

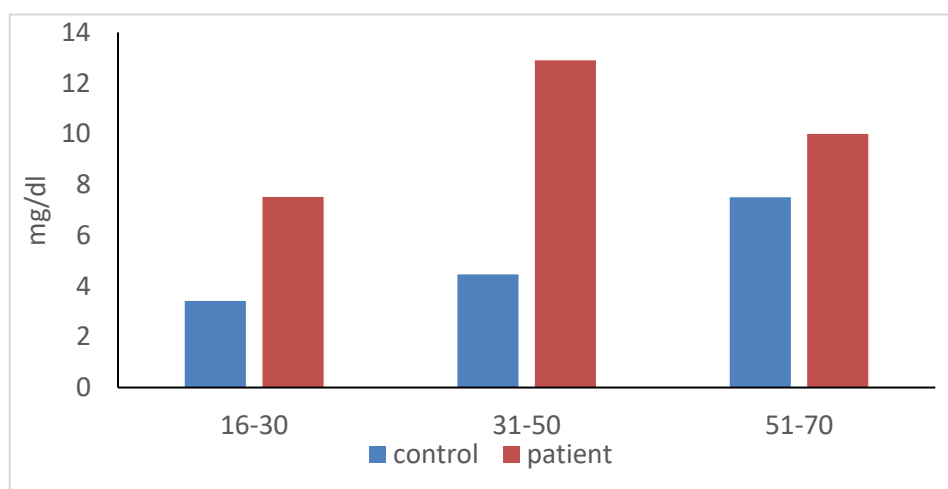


Figure 5. Relationship between age and uric acid levels in serum of patients and control groups

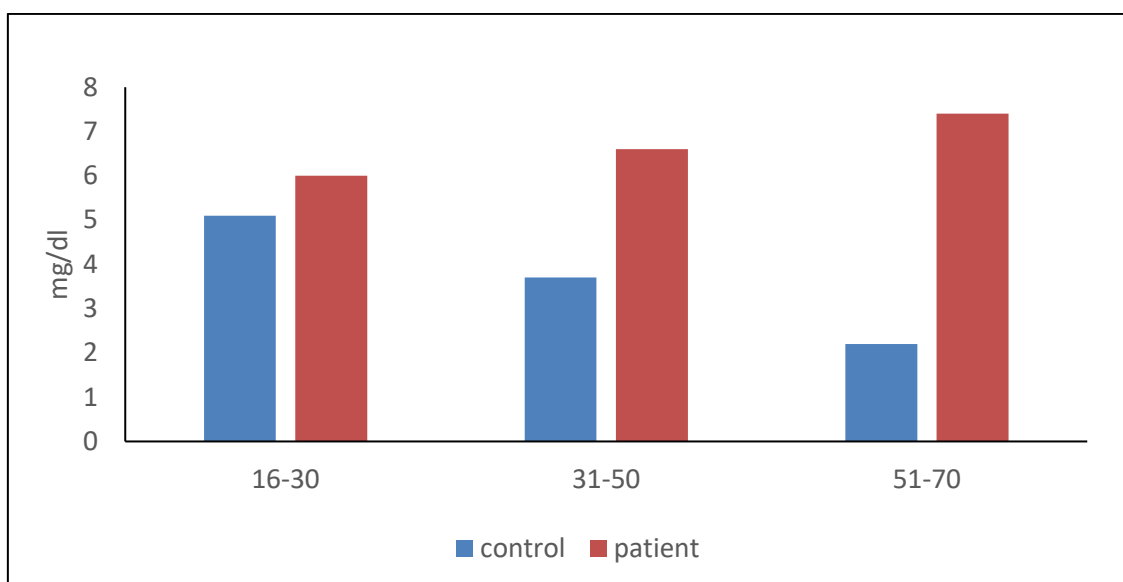


Figure 6. Relationship between age and total protein levels in the serum of patients and controls

Blood serum is known to contain urea, creatinine, total protein, and uric acid. It is used as a diagnostic test for kidney function. This study found that patients with chronic renal failure had significantly elevated blood serum levels of creatinine, urea, and total protein, and a slight elevation in uric acid, when compared to the healthy group. These findings are consistent with research (13).

The results of the control group are displayed in Table 3. It can be seen that the urea values for the age group 16–30 were 22.32 ± 2.31 , and that the urea values increased as the age groups increased from 31–50 and 51–70 to 35.53 ± 3.7 and 39.00 ± 5.79 , respectively. These results showed that the concentration of urea increased with age, and the reason for this increase is due to the weakening of kidney function with age, because glomerular filtrate decreases after the age of thirty (14). The findings in Table 4 demonstrate a significant age effect on urea concentration ($p < 0.05$) in the patient group's serum, as shown in Figure 3 and Table 4. The concentration of urea in serum for the 16–30 years age group was 105.6 ± 4.78 mg/dL, and it increased in the 40–60 and 51–70 years age groups to reach 183.07 ± 23.96 mg/dL and 253.6 ± 87.8 mg/dL, respectively. These findings align with the statement made by (15). At this point, glomerular filtrate levels begin to decline after the age of thirty, contributing to the aging-related degradation of renal function. The mean serum urea levels for each patient are displayed in Tables 3–4, along with the corresponding age groups of the control participants. When comparing the serum urea levels of patients to those of control subjects, a substantial rise was observed in all age groups ($P < 0.0001$). Additionally, there were notable variations in serum urea levels across all patient age groups ($P < 0.05$).

Table 3 results also indicated that age had no significant effect on creatinine concentration for the control group. Ages 16 to 30 had a creatinine concentration of 0.85 ± 0.1 , but the age groups 31 to 50 had a higher creatinine concentration of 1.3 ± 0.04 , suggesting that creatinine levels increase with age. Ages 51 to 70 had a creatinine concentration of 0.9 ± 0.02 . Because the kidney cannot filter and excrete waste, creatinine levels in renal illnesses do not rise until there is significant damage to the kidney. In this scenario, high creatinine levels indicate moderate to severe kidney impairment. The creatinine results in Table 4 for the patient group stated that it was 4.3 ± 0.78 for the age group 16–30, but increased to 7.6 ± 4.2 for the age group 31–50, and 14.5 ± 0.09 for the age group 51–70. The concentration increases with age, which explains why the tiny renal tubules assist the kidney in excreting creatinine when blood creatinine levels rise above normal ranges. The mean serum creatinine values for all patients and control subjects, categorized by age, are presented in Tables 3 and 4. Serum creatinine levels in all patient age groups were significantly higher ($P < 0.0001$) than in control persons. Additionally, there were notable variations in serum creatinine levels across all patient age groups ($P < 0.05$). Because urea and creatinine are metabolic wastes that are usually excreted through urine, and because renal failure is caused by a defect and

deficiency in kidney function, which results in a lack of excretion of these wastes, they accumulate and rise in concentration in the blood serum, it may be explained by the high concentration of these wastes in the blood serum of patients with renal failure (16).

Table 3 data also showed a significant age effect on uric acid concentration: individuals aged 16 to 30 had a concentration of 3.41 ± 0.01 , those aged 31 to 50 had a concentration of 4.43 ± 0.06 , and those aged 51 to 70 had a concentration of 7.50 ± 0.05 . Observes the rise in uric acid concentration with aging and the fact that uric acid is influenced by the amount of nucleic proteins and free purine bases in food, such as those found in tea and coffee, as well as the catabolic process that occurs in the body's cells.

The patient group's uric acid results showed that it was 7.51 ± 1.9 in the 16–30 age group, but it increased to 12.9 ± 2.4 in the 31–50 age group before returning to 10 ± 0.96 in the blood due to a decrease in the rate of kidney filtration and uric acid as a result of kidney failure, as well as in the case of lysolytic. The mean serum uric acid levels for each patient are shown in Table 4 along with the age ranges of the control subjects. When comparing the serum urea levels of patients to those of control subjects, a significant rise was observed in all age groups ($P < 0.05$). Additionally, there were notable variations in serum urea levels across all patient age groups ($P < 0.05$) (17).

Regarding the protein total, we observed values of 5.1 ± 0.31 in the 16–30 age group, 3.7 ± 0.21 in the 31–50 age group, and 2.2 ± 0.5 in the 51–70 age group. This reduction is brought on by gout and/or malnutrition or malabsorption from inflammatory conditions. The protein group of patients' results in Table 3 indicated that the age group 16–30 had a mean result of 6.0 ± 0.01 , the age group 31–50 had a mean result of 6.6 ± 0.3 , and the age group 51–70 had a mean result of 7.4 ± 0.4 . This suggests that as people age and have renal failure, their concentration of total protein will rise. The mean serum levels of total protein in each patient are displayed in Tables 3 and 4, together with the age distribution of the control participants. When comparing the serum protein levels of patients across all age groups to those of control subjects, there was a less substantial difference ($P < 0.01$). Additionally, there were significant differences in blood protein levels ($P < 0.05$) across all patient age groups (18). All of the biochemical test results presented here agree with those reported in (19).

4. CONCLUSION

It is well known that blood serum levels of urea, creatinine, and uric acid can serve as diagnostic tools for renal function. In this study, patients with renal failure had slightly higher blood serum levels of uric acid and notably higher levels of both creatinine and urea compared to the healthy group. Since urea and creatinine are metabolic waste products that the body naturally excretes through urine, an imbalance caused by renal failure may explain the elevated concentrations of these substances in the blood serum of these patients. When the kidneys do not function properly, these waste products are not excreted efficiently, leading to their accumulation and increased levels in the blood serum. The study also observed a slight increase in the uric acid content in the blood serum of the patients, due to a decrease in the renal glomerular filtration rate (GFR), which reduces uric acid excretion by the kidneys and causes a buildup of uric acid in the blood. In summary, blood parameters such as urea, creatinine, uric acid, and total protein provide reliable and practical data in daily clinical practice. They offer insights into the patient's dietary habits and adherence to prescribed treatment.

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