Journal JBB
ISSN:2958-0528

Journal of Biomedicine and Biochemistry
Volume 2 Issue 1 (2023), pp. 28–35

ORIGINAL ARTICLE

Role of Routine Markers in the Diagnosis of Patients with Chronic Kidney Disease

Haider Ali Laibi[1,*], Abdulkareem M. Jewad[1], and Mohammed Mahdi Salih[2]

[1]Medical and Health Technical College, Southern Technical University, Iraq
[2]Basrah University Teaching Hospital, Iraq.

Corresponding author:

fx0guakhd6@gmail.com

Medical and Health Technical College,
Southern Technical University,
Basrah, Iraq

Received:February 17, 2023,
Revised:February 27, 2023,
Accepted:March 15, 2023,

DOI: 10.57238/jbb.2023.6532.1027

OPEN ACCESS

Access this article online

Abstract

Background Acute kidney injury (AKI) and chronic kidney disease CKD cause different GFR declines (AKI). A gradual, irreversible decline in GFR is a CKD symptom. Diseases impair the kidneys' ability to concentrate or dilute tubular filtrate, eliminate nitrogenous waste, and maintain acid-base balance. Acute kidney failure (AKF) is a sudden, reversible renal function loss requiring RRT. Chronic kidney disease has a 13.4% global prevalence and 1.2 million annual deaths.

The Aim Serum creatinine, urea, potassium, chloride, calcium, sodium, and serum uric acid in CKD patients and healthy controls, and how these indicators vary with CKD development.

Determination of some trace elements such as copper, zinc, and iron in the sera of patients.

Materials and Methods The study included (30 healthy participants) and (60) patients with CKD (control). The participants in this study were people who traveled to Basrah Teaching Hospital aged 25 to 65. From October 2020 to February 2021, specialized physicians examined each participant in this study at Basrah Teaching Hospital. Serum potassium, sodium, chloride, calcium, phosphorus, urea, uric acid, and serum creatinine were measured using kits.

Results The current study results have shown a significant increase ($P < 0.01$) in the following biomarkers in CKD patients: serum creatinine, urea, phosphorus, chloride, uric acid, and Cystatin-C. The results also showed a significant decrease in levels following biomarkers in CKD patients: serum calcium and sodium.

Conclusion Chronic renal disease raises serum creatinine, urea, potassium, chloride, and uric acid. Chronic renal disease patients had lower calcium and sodium levels.

Keywords: CKD, Creatinine; Urea; Potassium; Chloride; Calcium; Sodium; Uric Acid

1 Introduction

The kidney is a bean-shaped organ with a central evagination known as the hilum, through which blood arteries, lymphatic vessels, and nerves escape and enter the kidney. The two kidneys are on opposite sides of the human spinal column [1]. By raising the risk of at least five other significant causes of death—cardiovascular disease (CVD), diabetes mellitus (DM), hypertension, HIV, and malaria—chronic kidney disease indirectly affects global disorder and mortality. For instance, the Global Burden of Disease (GBD) is anticipated to result in 1.2 million annual deaths, 19 million disability-adjusted life years (DALYs), and almost the same million years of life lost due to reduced glomerular filtration rates. Furthermore, 1.2 million people died from renal failure in 2015, a 32% rise from 2005 [2].

According to estimates, 2.3 and 7.1 million people with end-stage renal illness passed away in 2010 without access to chronic dialysis. Additionally, 1.7 million people are thought to pass away annually due to acute renal impairment. As a result, it is estimated that 5 to 10 million people every year pass away from renal disease. According to some epidemiological data, these numbers may underestimate the actual number of fatalities brought on by renal sickness because of a general lack of knowledge and frequently insufficient laboratory expertise [3].

Patients with chronic kidney disease (CKD) may have cardiovascular or cerebrovascular problems, and their deaths might be attributed to either. Clients with hypertensive and ischemic heart disease, linked to an increased risk of cardiovascular illness and mortality, often have altered kidney function. Diabetic nephropathy affects around 30% of diabetic patients, with certain ethnic groups having higher trouble [4]. The rate of GFR decline varies between acute kidney injury (AKI) and chronic kidney disease (CKD) (AKI). GFR declines slowly and irreversibly over many months, years, or even decades due to CKD [5].

Chronic refers to something that lasts for a very long time, and CKD is characterized by developing kidney damage, structural changes to the kidney, a decline in renal function, or renal damage and decreased function that last for longer than three months. Chronic kidney disease, which has a global prevalence of 13.4% and a mortality rate of 1.2 million per year, is a significant public health issue (about). Numerous study groups have looked into CKD's physical and chemical causes, including soil, water, food, heat stress, radiation, pesticides, and environmental samples. They have yet to identify the specific factors that are to blame [6].

A tiny chemical compound called urea has a molecular weight of 60 and is made up of two bonded amino (NH2) and carbonyl (C=O) groups. It is a nitrogenous byproduct of the degradation of proteins and amino acids (AA). Proteins are initially broken down into the component AA, which is then deaminated to produce the deadly ammonia (NH3). The "urea cycle" is a series of five enzymatically controlled processes that transform hazardous ammonia from protein degradation into non-toxic urea [7, 8]. Plasma and/or serum creatinine are two of the chemicals most frequently analyzed in clinical chemistry labs worldwide. The molecular weight of creatinine, an amino acid derivative, is 113 Da. Almost exclusively found in striated muscle tissues, it is a waste metabolic byproduct of creatine and phosphocreatine (90 percent). Typically, muscle contains 125 mmol/kg dry mass of total creatine. A daily conversion of 2% of the body's creatine to creatinine results in consistent creatinine output. It is filtered freely by the renal glomeruli and released by the proximal tubules, accounting for 5 to 10% of the creatinine removed [9].

Numerous animals, including insects, birds, and reptiles, have the heterocyclic compound uric. Uric acid is a nitrogenous metabolic waste product that is almost insoluble. Some mammals' blood and urine contain uric acid, the end product of purine metabolism. The last stage of the breakdown of nucleic acids and proteins leads to the release of uric acid in urine. Uric acid is synthesized by plants as well. When xanthine oxidase genes contain mutations, uric acid levels may rise in specific organs. Algae symbiotically coupled with some types of sea anemones were said to collect significant levels of potassium oxalate. However, this prediction was shown to be wrong when it was proven [10]. The body needs sodium in very high doses since it is an electrolyte. When electrolytes are dissolved in biological fluids like blood, they acquire an electric charge. Most of the body's salt is in the blood and the surrounding fluid cells. The body needs sodium to maintain a healthy fluid balance. Sodium is required for normal neuron and muscle function [11]. As a nutrient, calcium is frequently associated with bone metabolism and formation. Calcium hydroxyapatite, which makes up more than 99 percent of the body's total calcium and strengthens hard tissue, is found in the bones and teeth. Calcium is required for vascular contraction, vasodilation, myofunction, neuronal transmission, intracellular communication, and hormone production in the circulatory blood, extracellular fluid, muscle, and other tissues. Through bone remodeling, bone tissue acts as a calcium supply and reserve for these vital metabolic needs[12].

When potassium (K^+) intake and elimination are balanced, and the distribution of K^+ between extracellular and intracellular fluid compartments is kept regular, plasma K^+ content is kept within a narrow range. The latter is crucial since cells contain 98% of exchangeable K^+ and just 2% of the body's total K^+,

which is included in extracellular fluid. This kind establishes the resting cellular charges, with the interior oriented negatively with respect to the outside, and it explains how problems with plasma K^+ homeostasis lead to symptoms in active tissues. The typical total body K^+ level of a 70 kg individual, which varies from 3,000 to 4,000 mEq, is maintained mainly by the renal tissues. Only 60 to 80 mEq of the body's total reserves are present in extracellular fluid, where they are typically held at a concentration of between 3.5 and 5.3 mEq [13].

The primary anion in the body, chloride, has a molecular weight of 35.5 and accounts for 70% of the hazardous ions. An adult's body typically contains 115 grams of chloride or roughly 0.15 percent of their entire body weight. The vital extracellular anion chloride is responsible for many bodily processes, such as maintaining osmotic pressure, acid-base balance, muscle activation, and water transport between fluid chambers [14]. Phosphorus is found in every living cell and is second only to calcium in overall body weight, representing 1% of the entire body weight. Phosphorus works as a structural component of bones and teeth, DNA and RNA, and as a component of membrane lipid bilayers and lipoproteins [15]. When present in phosphoric acid buffers, phosphorous takes part in the generation of ATP (energy) by phosphorylating a phosphate on the ATP molecule, buffers the plasma, modulates gene transcription, aids enzyme catalysis, and allows signal transduction pathways affecting a variety of organ activities, including renal elimination, to take place. An inorganic phosphate concentration between 2.5 and 4.5 mg/dL is firmly managed in adult humans' plasma and/or serum concentrations [16].

2 Material and method

2.1 Chemicals

Diagnostic kits used in the present study and their manufacturers are shown in Table 1.

Table 1: Kits and their manufacturers.

Name	Company	Country
Serum Potassium kit	Roche Diagnostic	Switzerland
Serum Sodium kit	Roche Diagnostic	Switzerland
Serum Chloride kit	Roche Diagnostic	Switzerland
Serum Calcium kit	Roche Diagnostic	Switzerland
Serum Phosphorus kit	Roche Diagnostic	Switzerland
Serum Urea kit	Roche Diagnostic	Switzerland
Serum Uric acid kit	Roche Diagnostic	Switzerland
Serum Creatinine kit	Roche Diagnostic	Switzerland

2.2 Sample collection

There were (30) healthy individuals and (60) people with chronic renal disease included in this research. Participants in the study at the Al-Basrah Teaching Hospital in the Basrah governorate varied in age from (25 to 65). From October 2020 to February 2021, specialized physicians assessed each participant in this trial at the hospital. The department of Medical Laboratory Technology at Southern Technical University in Basrah is where the practical study was conducted.

2.3 Serum samples treatment

Each participant (patients and controls) was provided five milliliters of blood, which was then transferred to sterilized test tubes and allowed to coagulate for 30 minutes at room temperature. The serum was extracted from the blood sample and stored at -20 °C until use after being centrifuged at 3000 rpm for 15 minutes.

2.4 Exclusion criteria

Many individuals were excluded from the trial because they had acute and chronic illnesses other than renal diseases, hypertension, and diabetes mellitus. We didn't include any participants in the study who had hormonal problems.

2.5 Statistical analysis

Data are stated as means ± standard deviation (SD). Both the t-test and the chi-square test were used to examine differences in group means. Additionally, correlations between variables were found. SPSS for Windows was used to execute every statistical analysis (version 23, USA).The Mann-Whitney and non-parametric Kruskal-Wallis tests were utilized when the prerequisites for a normal distribution were not met. P 0.05 was considered statistically significant in the normal distribution, whereas $P > 0.05$ was considered non-significant.

3 Results and Discussion

3.1 Demographic data of the study groups

The statistical distribution (frequency and percentage) of the study groups (patients and controls) by age, weight, and gender is shown in Table 1, Figure 1, Figure 2, and Figure 3. According to this data, men and women were evenly distributed (50 percent each), and the highest rates of the control subgroup were among those with ages between (37 and 44) and weights between (71 and 83) and (84 and 96) kg.

Table 2: Statistical distribution of the study groups by their age, weight, and gender .

Items	Sub-groups	Control Group (N= 30)		CKD Patients (N= 60)		Chi-Square (P value) Sig.
		Freq.	%	Freq.	%	
Age / Years	25-36	3	10.0	4	6.7	2.3 (0.68) NS
	37-44	15	50.0	23	38.3	
	45-55	9	30.0	22	36.7	
	56-65	3	10.0	11	18.3	
Mean ± SD		51.18 ± 10.76		51.20 ± 10.72		
Weight / Kg	45-57	1	3.3	6	10.0	9.4 (0.06) NS
	58-70	5	16.7	19	31.7	
	71-83	11	36.7	18	30.0	
	84-96	11	36.7	8	13.3	
	97-109	2	6.7	9	15.0	
Mean ± SD		77.92 ± 20.92		76.08 ± 15.11		
Gender	Male	15	50.0	30	50.0	0.09 (0.76) NS
	Female	15	50.0	30	50.0	

NS: Non-significant; SD: Standard Deviation

The same table reveals that males and females were equally distributed (50%) within the CKD Patients subgroup, with the most significant percentages being among those ages between (37.44) years old (38.3%) and those with weights between (58-70) kg (31.7%). Age, gender, and weight between the study's patient and control groups are not significantly different (P > 0.05), according to the same table; this finding is required to rule out confounding variables that could interfere with comparing patients with CKD and the control group.

The statistical distribution (frequency and percentage) of the study groups (patients and controls) by age, weight, and gender is shown in Table 1, Figure 1, Figure 2, and Figure 3. According to this data, men and women were evenly distributed (50 percent each), and the highest rates of the control subgroup were among those with ages between (37 and 44) and weights between (71 and 83) and (84 and 96) kg. The same table reveals that males and females were equally distributed (50%) within the CKD Patients subgroup, with the most significant percentages being among those ages between (37.44) years old (38.3%) and those with weights between (58-70) kg (31.7%). Age, gender, and weight between the study's patient and control groups are not significantly different (P > 0.05), according to the same table; this finding is required to rule out confounding variables that could interfere with comparing patients with CKD and the control group.

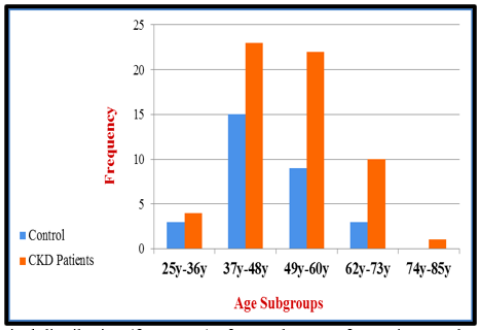

Figure 1: Statistical distribution (frequency) of age subgroups for patients and control

Table 3: Routine indicators between CKD patients and the control group were measured differently.

Routine Markers	Control Group (N= 30)		CKD Patients (N= 60)		T Test P value
	Mean	SD	Mean	SD	
Serum Creatinine (mg/dL)	0.75	0.12	5.25	3.75	6.55 0.000 HS
Serum Urea (mg/dL)	22.9	6.52	98.88	30.37	13.51 0.000 HS
Serum Calcium (mg/dL)	9.62	0.44	7.35	1.05	6.09 0.000HS
Serum Phosphorus (mg/dL)	4.29	0.72	7.62	1.59	5.42 0.000 HS
Serum Potassium (mmol/L)	4.71	0.39	6.67	2.26	3.26 0.02 S
Serum Chloride (mmol/L)	102.27	3.5	112.87	5.31	2.77 0.006 HS
Serum Sodium (mmol/L)	146.63	6.11	134.28	6.29	2.43 0.018 S
Serum Uric Acid (mg/dL)	4.9	1.09	8.03	1.43	3.74 0.000 HS

HS: High Significant at P≤0.01; **S:** Significant at P≤0.05; **SD:** Standard Deviation

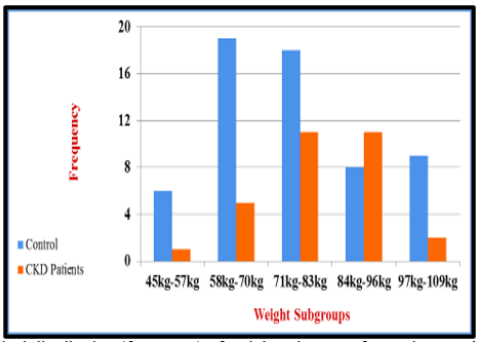

Figure 2: Statistical distribution (frequency) of weight subgroups for patients and control

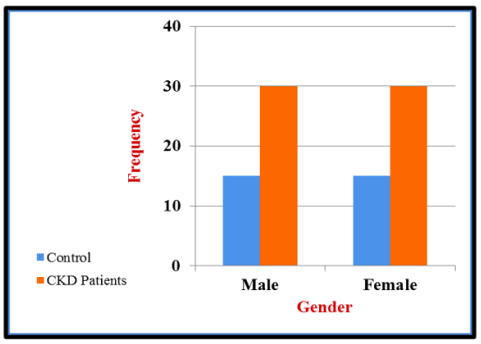

Figure 3: Statistical distribution (frequency) of gender subgroups for patients and control

Table.2 illustrates the differences in normal marker levels between the control and CKD groups. This table shows a highly significant increase ($P < 0.01$) in serum creatinine, urea, potassium, phosphorus, chloride, and uric acid levels in CKD patients compared to the control group.

The blood creatinine and urea levels in CKD patients are significantly higher ($P < 0.01$) than in the control group, as seen in this table. Wong-Vega et al. (2020) [17] stated that the rise in urea and creatinine levels in patients with CKD might be related to disrupted transport activities of the epithelial cells of the collecting tubules and diffuse impairment in the functions of proximal convoluted tubules. The kidneys' glomeruli filter creatinine, a non-protein waste product of the metabolism of creatine phosphate in muscles. On the other hand, the level of creatinine in the blood may increase if there is a deficit in the renal filtration process as a result of impaired renal function. Because creatinine is one of the metabolic waste products typically removed with urine, the content of creatinine has increased. GFR is decreased in renal failure, which raises serum urea nitrogen levels [17].

In patients with end-stage renal disease, urea levels can rise to pre-dialysis amounts ten times or higher than the top limit of the normal range in CKD patients. It determines the effectiveness of intradialytic solute removal and uraemic retention in CKD [18]. As the liver breaks down proteins and amino acids, urea is produced as a byproduct. Before being filtered by glomeruli, it is dispersed in the blood via extracellular and intracellular fluids. As the kidney filters urea produced by the liver, a high blood urea nitrogen level indicates impaired renal function [19].

The present study's findings also indicate that the levels of blood sodium in CKD patients are significantly lower ($P < 0.05$) than in the control group, also shows that there are highly significantly lower ($P < 0.01$) levels of serum calcium. This finding agrees with Jameson (2010)(20). He found that serum calcium concentrations are lower in renal failure patients. This decrease in serum calcium could be due to increased serum phosphorous because serum calcium and phosphorous concentrations have an inverse relationship. Any increase in one will result in a decrease in the other. Another possible cause for the reduction of serum calcium is a disturbance in vitamin D synthesis due to renal failure, which is caused by the kidney's inability

to synthesize the active form of vitamin D (1,25- dihydroxy cholecalciferol), which is very important for calcium absorption in the patient's intestine [20].

A 70-kg person's total body K^+ levels, which range from 3,000 to 4,000 mEq, are predominantly maintained by the kidney. The collecting duct and late distal tubule may sustain severe damage as a result of tubular necrosis in CKD. This direct harm to K^++ secretion cells causes K^+ to be retained in the blood. Hyperkalemia is uncommon in people with CKD until GFR falls to 15 to 20 mL/min. Even with a significant loss in kidney mass, it is still possible to maintain a somewhat normal plasma K^+ concentration since the remaining nephrons' rate of K^+ secretion has been adaptively enhanced. Similar adaptations occur in healthy individuals who consume a lot of K^+ in their diet. In animal models, long-term K^+ loading enhances the distal nephron's secretory capability, leading to enhanced renal K^+ excretion independent of plasma K^+ concentration [13]. With the development of CKD, gastrointestinal K^+ secretion becomes more essential in maintaining total-body K^+ content. Each hemodialysis treatment removes approximately 80 to 100 mEq of K^+ (up to 300 mEq/wk), while dietary K^+ consumption is typically 400 to 500 mEq/wk [21].

The current research also discovered that CKD patients had a significant rise ($P<0.01$) in blood phosphorus levels compared to the control group. This finding is consistent with Suki and Moore's (2016) [22] finding that phosphorus is required for various biological and metabolic processes essential to life, including the storage of energy for usage by all cells, including skeletal and heart muscles. Patients on dialysis may have persistent hyperphosphatemia for various reasons, including the following: Only 800 to 1,000 mg of phosphate are eliminated after a single hemodialysis session. As a result, three times per week of dialysis is insufficient to help individuals on dialysis meet their daily phosphorus need of 1,000 mg. Many retail food products contain highly accessible inorganic phosphate as a preservative to maintain freshness. These increase the patient's phosphate load, as was already mentioned. Phosphate binders increase the number of medications that dialysis patients must take and other medications. Phosphate binders are frequently large, difficult-to-swallow pills that might affect one's perception of flavor if chewed while eating, which makes compliance challenging.

Additionally, these medications frequently cause digestive issues and have a variety of phosphate-binding capacities. Finally, two more important factors related to the strategies used to treat these people have been discovered. One is using high doses of calcitriol or its equivalents, which have enhanced small intestine active phosphate absorption. The second point is that phosphate enhancement is known to be stimulated by lowering intestine-free phosphate concentrations, whether by dietary phosphate limitation or phosphate binders [22].

In numerous cross-sectional population studies, the average blood phosphorus level in people with normal renal function, also known as inorganic phosphate, or IP, has been found to be reasonably steady at 3.8 mg/dL. Until their glomerular filtration rate (GFR) falls below 30 mL/min/1.73 m2, which denotes Stage 4 chronic renal disease, it operates similarly in those with impaired kidney function (CKD). Serum phosphorus levels rise at this point and keep growing as these people develop the end-stage renal disease [22].

According to Soi and Yee (2017) [23], Besides being a crucial mineral for preserving the proper fluid balance inside the body, sodium has also contributed significantly to the development of the global economy. 50 - 60% of the filtered load is reabsorbed by the proximal tubule via the apical sodium-hydrogen exchanger. As GFR declines, CKD profoundly impacts normal physiology, changing the sodium balance in various ways. It has been demonstrated that each individual nephron unit experiences an adaptive increase in salt excretion. Because people with advanced CKD are more likely to experience the negative effects of high dietary sodium intake, sodium restriction becomes crucial [23].

Even though people with chronic kidney disease (CKD) frequently have elevated blood uric acid levels, this problem has generally been disregarded as inconsequential. Uric acid, however, has recently been identified as a potential risk factor that may quicken the onset of CKD. Most research has found that increased blood uric acid levels can independently predict the onset of CKD. Increased blood uric acid levels always accompany a decrease in glomerular filtration rate (GFR) because the kidneys are the organs that primarily remove uric acid. The development of intrarenal arteriolar lesions and an increased risk of cardiovascular death in people with CKD have been associated with higher blood uric acid levels, comparable to the vascular effects reported in hyperuricemic experimental animals. A higher risk of cardiovascular disease and death is associated with high and low uric acid levels, although the J-curve flips in patients with end-stage renal failure [24].

A significant increase in serum chloride was also seen in our study. It agrees with Rein Coca (2019) [25], who stated that renal vasoconstriction caused by tubuloglomerular feedback and potentially other processes, which will be thoroughly discussed later in this article, is thought to be related to hyperchloremia-associated kidney disease. Numerous studies demonstrate an increased risk of renal illness, morbidity, and death in relation to variations in blood chloride concentration, independent of serum sodium and bicarbonate [25].

4 Conclusion

There is an increase in the following biomarkers in patients with chronic kidney disease: serum creatinine, serum urea, serum potassium, serum chloride, and serum uric acid. There is a decrease in the following biomarkers in patients with chronic kidney disease: serum calcium and serum sodium.

Conflict of Interest: No conflicts of interest exist between the authors and the publication of this work.
Ethical consideration: The ethical committee approved the study at University of Al-Qadisiyah, Al Diwaniyah, Iraq.

References

[1] Premkumar K. The massage connection: anatomy and physiology. Lippincott Williams & Wilkins; 2004. [Backref page 29]

[2] Kour H, Manhas J, Sharma V. Usage and implementation of neuro-fuzzy systems for classification and prediction in the diagnosis of different types of medical disorders: a decade review. Artificial Intelligence Review. 2020;53:4651-706. doi:10.1007/s10462-020-09804-x. [Backref page 29]

[3] Luyckx VA, Tonelli M, Stanifer JW. The global burden of kidney disease and the sustainable development goals. Bulletin of the World Health Organization. 2018;96(6):414. doi:10.2471%2FBLT.17.206441. [Backref page 29]

[4] Glassock RJ, Warnock DG, Delanaye P. The global burden of chronic kidney disease: estimates, variability and pitfalls. Nature Reviews Nephrology. 2017;13(2):104-14. doi:10.1038/nrneph.2016.163. [Backref page 29]

[5] Weiner ID, Mitch WE, Sands JM. Urea and ammonia metabolism and the control of renal nitrogen excretion. Clinical Journal of the American Society of Nephrology. 2015;10(8):1444-58. doi:10.2215/CJN.10311013. [Backref page 29]

[6] Kakitapalli Y, Ampolu J, Madasu SD, Kumar MS. Detailed review of chronic kidney disease. Kidney Diseases. 2020;6(2):85-91. doi:10.1159/000504622. [Backref page 29]

[7] Rifai N. Tietz textbook of clinical chemistry and molecular diagnostics-e-book. Elsevier Health Sciences; 2017. [Backref page 29]

[8] Wu H, Li H, Fang Z. Hydrothermal amination of biomass to nitrogenous chemicals. Green Chemistry. 2021;23(18):6675-97. doi:10.1039/D1GC02505H. [Backref page 29]

[9] Pesenti S, Blondel B, Peltier E, Adetchessi T, Dufour H, Fuentes S. Percutaneous cement-augmented screws fixation in the fractures of the aging spine: is it the solution? BioMed research international. 2014;2014. doi:10.1155/2014/613890. [Backref page 29]

[10] Clode PL, Saunders M, Maker G, Ludwig M, Atkins CA. Uric acid deposits in symbiotic marine algae. Plant, cell & environment. 2009;32(2):170-7. doi:10.1111/j.1365-3040.2008.01909.x. [Backref page 29]

[11] Tinawi M. Hyponatremia and hypernatremia: a practical guide to disorders of water balance. Archives of Internal Medicine Research. 2020;3(1):74-95. doi:10.26502/aimr.0025. [Backref page 29]

[12] Fang AP, Li KJ, Shi HY, He JJ, Li H. Habitual dietary calcium intakes and calcium metabolism in healthy adults Chinese: a systematic review and meta-analysis. Asia Pacific Journal of Clinical Nutrition. 2016;25(4):776-84. doi:10.3316/informit.369937455585900. [Backref page 29]

[13] Palmer BF, Clegg DJ. Physiology and pathophysiology of potassium homeostasis: core curriculum 2019. American Journal of Kidney Diseases. 2019;74(5):682-95. doi:10.1053/j.ajkd.2019.03.427. [Backref page 30], [Backref page 33]

[14] Berend K, van Hulsteijn LH, Gans RO. Chloride: the queen of electrolytes? European journal of internal medicine. 2012;23(3):203-11. doi:10.1016/j.ejim.2011.11.013. [Backref page 30]

[15] Zoroddu M, Aaseth J, Crisponi G, Peana M, Nurchi V. The essential metals for humans: A brief overview Maria. J Inorg Biochem pagerangek https://doi org/101016/j jinorgbio. 2019;13. doi:10.1016/j.jinorgbio.2019.03.013. [Backref page 30]

[16] Calvo MS, Uribarri J. Public health impact of dietary phosphorus excess on bone and cardiovascular health in the general population. The American journal of clinical nutrition. 2013;98(1):6-15. doi:10.3945/ajcn.112.053934. [Backref page 30]

[17] Swartz S, Devaraj S, Poyyapakkam S, et al. Elevated Serum Creatinine: But Is It Renal Failure? Pediatrics. 2020;146(1). doi:10.1542/peds.2019-2828. [Backref page 32]

[18] Vanholder R, Gryp T, Glorieux G. Urea and chronic kidney disease: the comeback of

the century?(in uraemia research). Nephrology Dialysis Transplantation. 2018;33(1):4-12. doi:10.1093/ndt/gfx039. [Backref page 32]

[19] Mehan S, Dudi R, Kalra S. Renoprotective effect of corosolic acid in gentamicin-induced nephrotoxicity and renal dysfunction in experimental rats. Ann Pharmacol Pharm. 2017;2(12):1065. [Backref page 32]

[20] Natasia SMD, Evatta A. Prevalensi dan Karakteristik Pasien Retinopati Diabetik pada Pasien Prolanis di RSI Gondanglegi Tahun 2021. Syntax Literate; Jurnal Ilmiah Indonesia. 2022;7(4):3928-34. doi:10.36418/syntax-literate.v7i4.6717. [Backref page 33]

[21] Clegg DJ, Gallant KMH. Plant-based diets in CKD. Clinical journal of the American Society of Nephrology: CJASN. 2019;14(1):141. doi:10.2215%2FCJN.08960718. [Backref page 33]

[22] Suki WN, Moore LW. Phosphorus regulation in chronic kidney disease. Methodist DeBakey cardiovascular journal. 2016;12(4 Suppl):6. doi:10.14797%2Fmdcj-12-4s1-6. [Backref page 33]

[23] Soi V, Yee J. Sodium homeostasis in chronic kidney disease. Advances in chronic kidney disease. 2017;24(5):325-31. doi:10.1053/j.ackd.2017.08.001. [Backref page 33]

[24] Johnson RJ, Nakagawa T, Jalal D, Sánchez-Lozada LG, Kang DH, Ritz E. Uric acid and chronic kidney disease: which is chasing which? Nephrology Dialysis Transplantation. 2013;28(9):2221-8. doi:10.1093/ndt/gft029. [Backref page 33]

[25] Rein JL, Coca SG. "I don't get no respect": the role of chloride in acute kidney injury. American Journal of Physiology-Renal Physiology. 2019;316(3):F587-605. doi:10.1152/ajprenal.00130.2018. [Backref page 33]

How to cite this article

Laibi H.A.; Jewad A.M.; Salih M.H; Role of Routine Markers in the Diagnosis of Patients with Chronic Kidney Disease. Journal of Biomedicine and Biochemistry. 2023;2(1):28-35. doi: 10.57238/jbb.2023.6532.1027