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Predictive Roles of Some Electrolytes and Biochemical Markers in The Pathophysiology of Renal Failure Diseases; Case-Control Study

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Abstract. Objective; detection of the predictive role of some electrolytes and biochemical indicators in the pathophysiology of renal failure diseases. Methods; The present investigation was done in Diyala province within time (July-November 2024). We have 120 blood samples from hemodialysis patients that sleeping in the Ibn-Sina Center for Kidney Dialysis / Ba'aqubah Teaching Hospital. In addition, we collected 80 blood samples from people without diseases (healthy) and depended on them as a control group. Serum levels of all biochemical indicators in all samples of participants were quantified by Cobas e411 machine. SPSS v. 22.0 with Prism v.10 programs were based for the analysis our data. Results; Present outcomes showed that most renal failure patients were males (51.7%) within age groups 41-60 years (48.3%) and living in rural areas (51.7%). Additionally, most patients were no smokers (90.0%) with underweight BMI (33.33%) . Levels of RBS, creatinine, sodium, potassium and urea were highest in patients compared to controls, while the levels of calcium and iron were lowest in patients versus controls with significant difference ($p<0.05$). ROC curve results showed the urea scored highest sensitivity and specificity (100% and 94%) at cut off (40.50), followed by creatinine (100% and 89%) and albumin (88% and 86%) at cut off values (1.50 and 3.90) respectively, with significant differences ($p<0.05$), in diagnosis renal failure patients. pearson correlation coefficient showed there is negative significant correlation between RBS and creatinine (r=-0.189* and p= 0.039), albumin and RBS (r=-0.310* and p= 0.001), and a positive significant correlation between albumin and sodium (r=-0.227* and p= 0.013) and iron with sodium (r=-0.182* and p= 0.046). Conclusions; We concluded that renal failure disease has more effect on patients >40 years with underweight BMI. No effect of gender, living and smoking on disease. The increase and decreased levels of all indicators are related to kidney damage and loss of homeostasis. Urea, creatinine and albumin indicators are more preferred in screening disease due to have these indicators high sensitivity and specificity than another indicators

Highlights:

1. Assessed electrolytes' role in renal failure diagnosis using biochemical indicators.
2. Analyzed 120 hemodialysis patients vs. 80 healthy controls using Cobas e411.
3. Urea, creatinine, albumin showed highest sensitivity/specificity; significant correlations found.

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Keywords: Renal failure, kidney diseases, renal diseases, electrolytes, biochemical indicators

Introduction

Damage to the kidneys resulting in poor blood filtration is the hallmark of chronic kidney disease (CKD). The accumulation of excess waste products and fluids in the body might result in further health issues such as strokes and heart disease [1]. According to Abo-Ghneim et al. (2024), [2] it affects 8–16% of the world's population and is frequently disregarded by both patients and medical professionals. Research has indicated that low- and middle-income countries are more likely to experience it than nations with high incomes [3]. Globally, chronic kidney disease (CKD) has historically been linked primarily with high blood pressure and diabetes. Genetic, medical, way of life, metabolic, ecological, and socioeconomic variables, as well as psychological aspects, all influence the ways through which CKD establishes and evolves. Genetic characteristics are also important in predicting CKD, especially in individuals whose condition increases to end-stage renal disease (ESRD) [4] (Additionally, genetic susceptibility may also increase the chances of developing kidney failure [5]

According to Jakubowska and Malyszko (2024)[6] , kidney illness impacts the kidneys' glucose sensitivity. A low estimated GFR (eGFR) lowers the filtered glucose load, and the loss of working nephrons lowers reabsorption capability and consumes around 10% of plasma glucose. A kidney abnormality is indicated by an eGFR <60 ml/min/1.73 m2 and/or a urine albumin-creatinine ratio (uACR) >30 for ≥3 months. In a similar vein, renal dysfunction is also indicated by hematuria or structural abnormalities, or by an albuminuria concentration that is at least 30 mg per 24 hours[7] A major side effect of chronic kidney disease (CKD) is iron deficiency anemia, which is which is defined by both functional and quantitative iron deficiency and is brought on by elevated hepcidin levels and decreased iron reserves [8] .Due to iron loss from the hemodialysis machine, hemodialysis patients produce less erythropoietin. They also have problems absorbing iron and moving it from their stomach into their bloodstream. Last but not least, the form of anemia that is characterized by normally sized red blood cells and normal hemoglobin (Hb) levels with reduced red blood cell production may be linked to the negative health effects and elevated death rates caused by CKD[9] . Abnormalities in electrolytes are commonly seen in individuals with chronic kidney disease (CKD), and the

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kidneys are essential in maintaining electrolyte balance. The disturbance of several electrolyte regulators brought on by a loss or deterioration in renal function leads to considerable pathophysiological changes and implications. A number of problems, including problems with the heart, bones, muscles, and metabolism, are associated with electrolyte imbalances in chronic kidney disease (CKD), which have important pathophysiological ramifications [10] Due to inadequate renal fluid regulation, electrolyte abnormalities such as hyperkalemia and dysnatremia are frequently caused by renal failure; however, it is unclear how frequently dysmagnesemia occurs [11].CKD-mineral bone disease (CKD-MBD) is a complicated condition caused by a substantial disturbance in bone and mineral metabolism in CKD patients. According to Izzo et al. (2024)[12], perturbations start in the early stages of CKD and get worse as the condition progresses. Elevated blood phosphate and reduced serum calcium are two of the biochemical changes associated with CKD-MBD. Furthermore, reduced calcium absorption and decreased urine calcium excretion are noted, as well as varied bone disease and significant vascular and soft tissue calcification[13] .

Serum measurement of renal function indicators such as urea, creatinine, uric acid and electrolyte is frequently utilized in place of testing for urine, which can be somewhat uncomfortable for patients [14] . Blood tests for creatinine, the broken-down product of creatine phosphate in muscle, and blood urea nitrogen (BUN), a key nitrogenous end product of protein and amino acid catabolism, are eliminated by the kidneys [15] . BUN is a crude and indirect indicator of renal function that gauges blood urea nitrogen levels and is closely linked to kidney elimination function. Creatinine assays quantify blood levels of creatinine phosphate and identify decreased renal function. Increases in the serum are signs of renal disease, while urea and creatinine are favorable markers of a healthy kidney. The most extensively used and highly recognized metrics for evaluating renal function are serum creatinine and BUN [16]

Aim of study

The current investigation aim to studying predictive role of some electrolytes and biochemical indicators in pathophysiology of renal failure diseases in serum patients within Diyala province.

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Methods

Specimens collection

The present investigation was done in Diyala province within time (July-November 2024). We have 120 blood samples from renal failure patients that sleeping in Ibn-sina center for kidney dialysis / Baquba Teaching Hospital. In additionally, we collected 80 blood samples from peoples without diseases (healthy) and depended them as control group. Demographic features were filled in questioner document.

Methods

We harvested 5 ml of blood participants (patients and controls) in gel tube and then separated it by centrifuge machine (6000 rpm for 3 minutes) to has serum. Serum levels of all biochemical indicators in all samples of participants were quantified by Cobas e411 machine.

Statistical analysis

Biochemical indicators were showed as average and SD. Independent t test was based to detect differences of serum levels of biochemical indicators between study groups. Demographic features were showed as frequencies and percentages, and differences among these features calculated by X2 test. Receiver operating characteristic (ROC) curve was depended to measure cut-off, specificity and sensitivity of biochemical markers. Pearson coefficient was utilized to discover type and strength relation between biochemical indicators. Significance level $P \leq 0.05$ was based to have statistical variations. SPSS v. 22.0 with Prism v.10 programs were based for analysis our data

Result and Discussion

1- Demographic elements of renal failure patients

Outcomes of our study showed that most renal failure patients were males (51.7%) within age groups 41-60 years (48.3%) and living in rural areas (51.7%). Additionally, most patients were no smokers (90.0%) with underweight BMI (33.33%). The differences among percentages of demographic features were significant ($p<0.05$) for age groups, smoking and BMI, while no significant ($p>0.05$) for gender and living (table 1).

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Table 1; frequency and percentages of demographic features of renal failure patients

Number total= 120		Count	Percent	P value
Age groups (years)	1-20	29	24.2%	p<0.05*
	21-40	33	27.5%	
	41-60	58	48.3%	
Gender	Males	62	51.7%	p>0.05
	Females	58	48.3%	
Living	Rural	62	51.7%	p>0.05
	Urban	58	48.3%	
Smoking	Smoker	12	10.0%	p<0.001***
	No smoker	108	90.0%	
BMI	Under weight	40	33.33%	p<0.05*
	Normal weight	32	26.66%	
	Overweight	31	25.8%	
	Obesity	17	14.2%	
No significant different (P>0.05)				significant different
(P<0.05*)			High significant different	
((P<0.01**)			very high significant different (P<0.01***)	

2. Levels of biochemical indicators within study groups

Present outcomes showed significant differences (p<0.05) between biochemical indicators (rbs, creatinine, sodium, potassium, calcium , urea and iron) and study groups. The levels of RBS, creatinine, sodium, potassium and urea were high in patients (149.09±69.35, 4.02±1.16, 170.43±36.26, 6.02±1.69, and 91.91±20.02) compared to controls (94.28±9.21, 1.01±0.45, 140.70±45.36, 3.82±0.65, and 32.37±7.68). In opposite, The levels of calcium and iron were low in patients (7.14±3.23 and 100.78±39.81) versus controls (9.35±1.12 and 138.93±24.66). Finally, our study showed no differences (p>0.05) between albumin and study groups (table 2 and figure 3).

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Table 2; comparative concentrations of electrolytes and biochemical indicators between patients versus controls

groups		N	Mean	Std. Deviation	P value
RBS	Patients	120	149.09	69.35	*P<0.001***
	Controls	80	94.28	9.21	
Creatinine	Patients	120	4.02	1.16	*P<0.001***
	Controls	80	1.01	0.45	
Albumin	Patients	120	3.65	0.59	*P>0.05*
	Controls	80	4.42	0.79	
Sodium	Patients	120	170.43	36.26	*P<0.05**
	Controls	80	140.70	45.36	
Potassium	Patients	120	6.02	1.69	*P<0.05**
	Controls	80	3.82	0.65	
Calcium	Patients	120	7.14	3.23	*P<0.05**
	Controls	80	9.35	1.12	
Urea	Patients	120	91.91	20.02	*P<0.001****
	Controls	80	32.37	7.68	
Iron	Patients	120	100.78	39.81	*P<0.05**
	Controls	80	138.93	24.66	

No significant different (P>0.05) significant different (P<0.05*)
High significant different ((P<0.01**)
very high significant different (P<0.01***)

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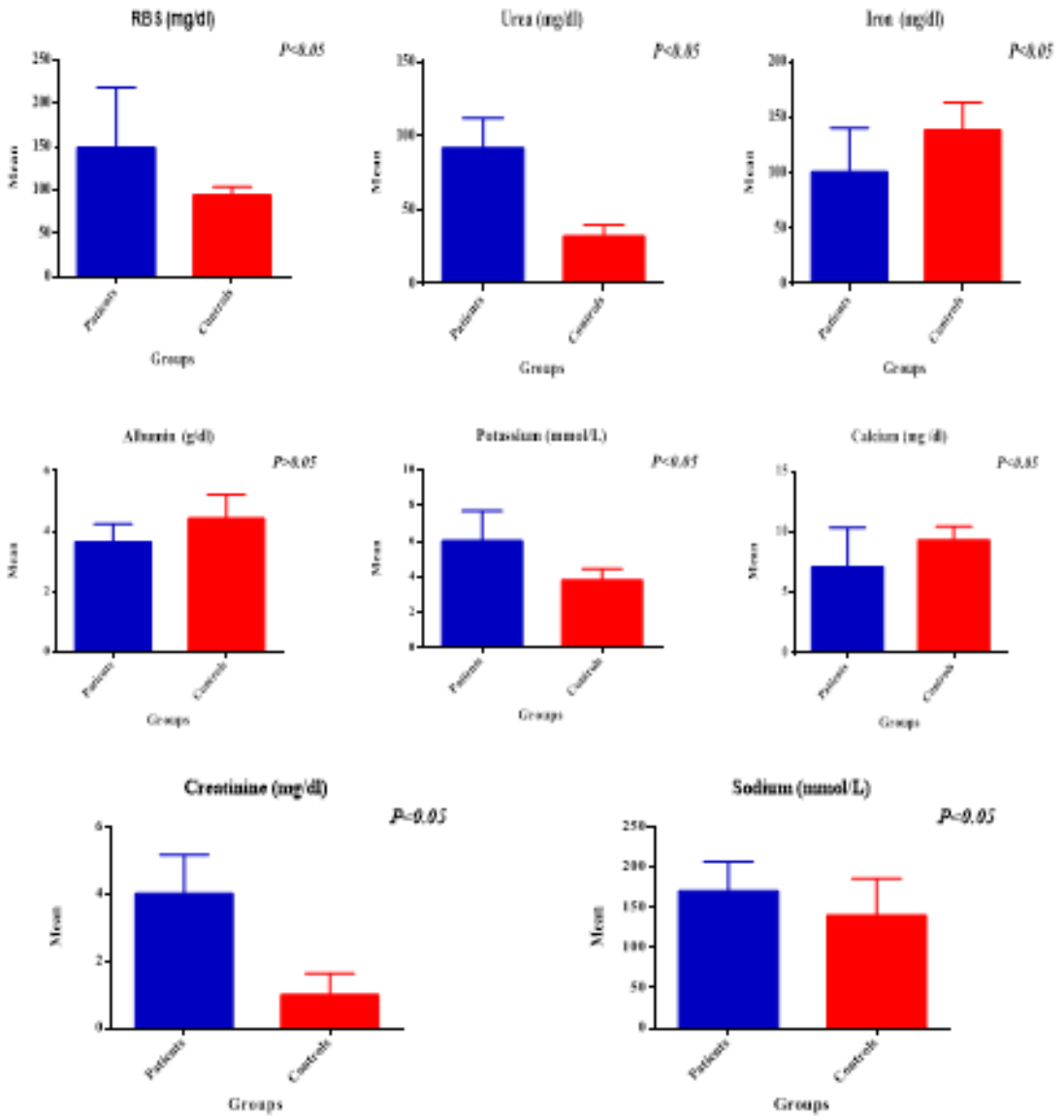

Figure 1; comparative concentrations of electrolytes and biochemical indicators between study groups

3. Receiver operating characteristic (ROC) curve of electrolytes and biochemical indicators

ROC curve results showed the urea scored highest sensitivity and specificity (100% and 94%) at cut off (40.50), followed by creatinine (100% and 89%) and albumin (88% and 86%) at cut off values (1.50 and 3.90) respectively, with significant differences ($p<0.05$), in diagnosis renal failure patients. Additionally, the sensitivity of RBS, sodium, potassium, iron, and calcium (78%, 74%, 73%, 73%, and 80%), while

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specificity (94%, 80%, 87%, 73%, and 70%) at cut off (121.50, 139.50, 4.10, 131.00, and 7.50) respectively, with significant differences ($p<0.05$), in diagnosis those patients (table 3 and figure 2).

Table 3; ROC curve, AUC, cut off , sensitivity and specificity of electrolytes and biochemical indicators in screening renal failure disease

Variables	AUC*	Std. Error	P value	cut off	Sensitivity %	Specificity %
RBS	0.713	0.037	p<0.01**	121.50	78	94
Creatinine	0.983	0.009	p<0.01**	1.50	**100**	**89**
Sodium	0.747	0.037	p<0.01**	139.50	74	80
Potassium	0.879	0.024	p<0.01**	4.10	73	87
Urea	100	0.001	p<0.01**	40.50	**100**	**94**
Albumin	0.770	0.039	p<0.01**	3.90	**88**	**86**
Iron	0.766	0.035	p<0.01**	131.00	73	73
Calcium	0.86	0.089	p<0.01**	7.50	80	70

AUC= Area under curve

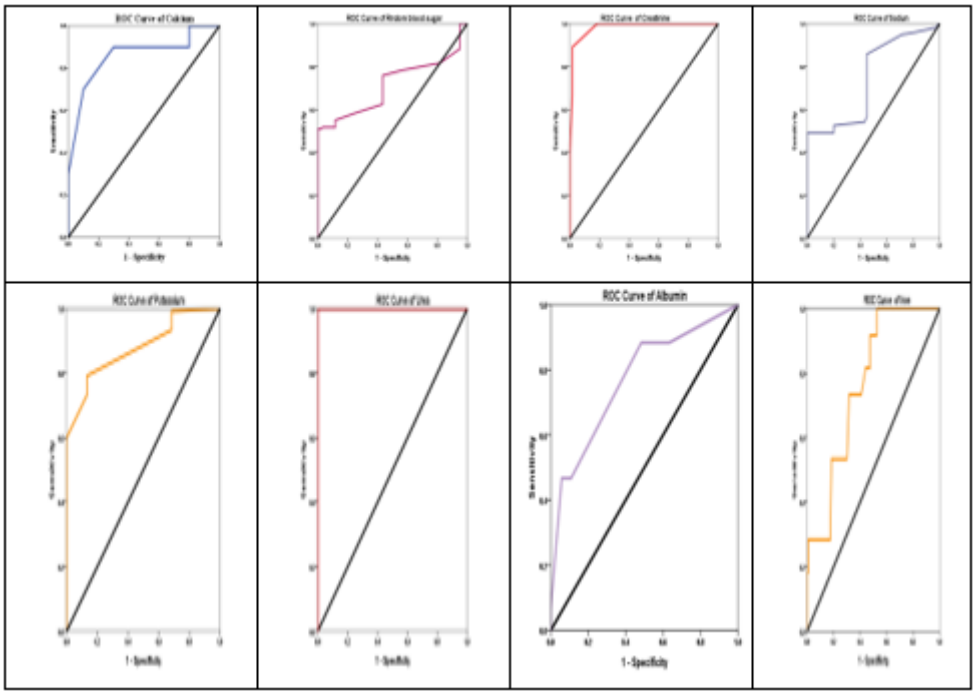

Figure 2; ROC curve of electrolytes and biochemical indicators

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4. Correlation relationship among electrolytes and biochemical indicators

Based on pearson correlation coefficient, our findings showed there is negative significant correlation between RBS and creatinine (r=-0.189* and p= 0.039), albumin and RBS (r=-0.310* and p= 0.001), and positive significant correlation between albumin and sodium (r=-0.227* and p= 0.013) and iron with sodium (r=-0.182* and p= 0.046). Another correlation were done among electrolytes and biochemical indicators but were no significant (p>0.05) (table 4).

Table 4; correlation relationship among biochemical indicators in renal failure patients

		RBS	Albumin	Potassium	Iron
RBS	Pearson Correlation (r)	1	**-.310****	.173	.076
	Probability (p)		.001	.059	.412
Creatinine	Pearson Correlation (r)	**-.189***	.074	-.027	.119
	Probability (p)	.039	.423	.768	.196
Sodium	Pearson Correlation (r)	-.118	**.227***	-.039	**.182***
	Probability (p)	.198	.013	.675	.046
Calcium	Pearson Correlation (r)	-.079	.090	-.037	-.055
	Probability (p)	.392	.327	.690	.549
Urea	Pearson Correlation (r)	-.020	.051	.163	.000
	Probability (p)	.826	.580	.076	.998
Iron	Pearson Correlation (r)	.076	.017	.129	1
	Probability (p)	.412	.855	.161	

Discussion

The present study aimed to detect some electrolytes and biochemical indicators in renal failure patients. It is found that most renal failure patients were males aged >40 years, and these outcomes were matched with findings of Togay and Akyüz, (2023)[17]. Due to variations in the amount of hormones, men could be more likely than women to develop renal failure earlier. Men who have elevated testosterone levels may have a decline in renal function. However, estrogen, which is greater in women during menopause, may not safeguard men's kidneys [18]. Researchers have demonstrated that while testosterone replacement therapy (TRT) may have major advantages for men

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with male hypogonadism who have chronic kidney disease (CKD), such as increased muscle mass, anaemia control, reduced inflammation, and possibly improved cardiovascular health in a diabetic subpopulation, it may also carry some risks for these men, particularly those who have excessive hypertension [19]. Furthermore, a number of factors, including as organ malfunction, chronic illnesses, and weakened immune systems, contribute to the increased frequency of disease in people >40 years [20] .

Based on living, our study no reveal significant differences between prevalence patients in rural and urban, and these results not compatible to results Al Mutawakil et al., (2024)[21] that mentioned high incidence of disease in rural area. These difference are related to several causes such as; sample size, lifestyle and environmental factors.

According to the current results, the majority of patients did not smoke, which is in contradiction to a research by Lang and Schiffl (2024)[22] that found the majority of patients smoked. Researchers have discovered that smoking increases the chance of developing chronic kidney disease (CKD) in patients with diabetes and/or high blood pressure. Smoking raised the risk of developing kidney illness in both groups. Additionally, smoking hastened the development of renal disease. In addition to lowering smoking-attributable morbidity and death, quitting smoking is an effective strategy to lower the risk of developing and developing chronic kidney disease (CKD) [23]. Smoking is therefore a risk trigger for renal disorders.

Our results, which were consistent with a research by Kim et al. (2023)[24], indicated that the majority of the participants were underweight according to BMI. According to Kim et al. (2023)[24], being underweight is linked to a higher risk of end-stage renal disease, and this correlation progressively gets stronger as BMI drops. The authors found that underweight people were more likely to develop end-stage renal disease (ESRD) than overweight ones. The quickest deterioration in renal function was linked to a weight reduction of more than 10% [25]. However, scientists found that a BMI of between 27.5 and 32.49 kg/m2 was linked to a decreased risk of cardiovascular and all-cause mortality in Uruguayan patients with chronic kidney disease (CKD) [26]. Studies vary in their consideration of the risk factors for renal problems, such as high or low BMI levels (table 1). Both males and females with a high BMI are at risk of developing CKD; however, the mechanisms and impact differ and are often influenced by lifestyle, hormonal factors and socioeconomic status [27]. (Table 1).

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According to the current study, patients with renal failure had lower levels of albumin, iron, and calcium and higher levels of RBS, creatinine, sodium, potassium, and urea than controls. The results reported were in line with those of Abo-Ghneim et al. (2024) [2]. In a healthy person, glucose is taken in again after being filtered at the glomerulus. Sodium-glucose co-transporters facilitate reabsorption, which can reach a maximum rate of 375 mg/min. When plasma levels rise beyond 200 mg/dl, glucose is detected in the urine [28]. Due to renal impairment, elevated blood sugar levels in individuals indicate diabetic kidney disease [29]. The current study calculated the amount of urea and creatinine, and significant boosts were found when comparing the patients to the control group. This is because the kidneys' inability to remove creatinine during the elimination of urine causes elevated blood creatinine levels. The urea cycle is a sequence of events that produces urea, the main byproduct of protein catabolism. It is commonly known that the urea cycle transforms ammonia into urea, which is then carried by the blood to the kidneys for elimination from the body [10]. The kidneys' removal of creatinine and urea from the blood is used to determine the levels of these substances in the bloodstream of CKD patients [2]. Elevated blood urea levels were linked to a greater risk of CVD events and mortality, according to a previous investigation of a sizable cohort of CKD patients [30] .

Higher levels of inflammatory cytokines and disruptions in iron homeostasis are intimately linked to chronic kidney disease (CKD), a common worldwide health problem that exacerbates consequences such renal anemia and cardiovascular disease. The results for patients are greatly impacted by renal anemia, which is mostly caused by decreased erythropoietin (EPO) production and iron imbalances. According to Matsuoka et al. (2024) [31], uremic toxins—specifically, protein-bound substances like indoxyl sulfate (IS)—are essential in the progression of chronic kidney disease (CKD) and have an impact on iron metabolism, creating intricate regulatory issues in the treatment of renal anemia. abnormalities of iron metabolism can exacerbate the course of IgA nephropathy (IgAN), and IgAN can induce abnormalities of iron metabolism. In IgAN, iron metabolism and chronic inflammation are intimately associated. For new preventative or treatment approaches for iron metabolism abnormalities in IgAN, the hepcidin-ferroportin axis controlled by agonists/antagonists is an appealing target [32]. Yu et al. (2023) [33]showed that in male or non-anemic individuals with CKD stages 1–

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4, an iron shortage, as determined by either iron or iron saturation, was linked to poor kidney performance.

In Japanese patients with chronic kidney disease, prior research indicates a negative and non-linear relationship between albumin and the loss of kidney function and kidney prognosis. Albumin drop was strongly associated with a poor renal outlook and a reduction in renal function when albumin levels were below 4.1 g/dL. Reducing the albumin drop makes rationale from a therapeutic perspective in order to postpone the progression of CKD [34] . Our research and this one were matched.

One of the main causes of electrolyte imbalances is the emergence of chronic kidney disease (CKD), despite the paucity of research on the connection between electrolyte imbalances and kidney function tests in the population at large. When chronic kidney disease (CKD) progresses to end-stage renal disease (ESRD), electrolyte imbalances become a prevalent symptom of kidney tubular destruction [35]. Compared to propensity score-matched patients without hyperkalemia, individuals with CKD stages 3b/4 and hyperkalemia (high potassium levels in the serum) had significantly greater odds of CKD progression and overall death, according to recent research [36]. A U-shaped association between predialysis levels of potassium in the blood and both general and cardiovascular mortality is suggested by observational data from a sizable cohort of recurrent and recurrent chronic hemodialysis (HD) patients (n = 81.013) [37]. Diet, medicine, and decreased potassium excretion with decreasing kidney function are the primary causes of hyperkalemia in end-stage renal disease (ESRD); intradialytic therapy is still a clinical issue [38]. Convective and diffusive transfer promote intradialytic potassium elimination, which largely takes place within the first two hours of HD therapy. Although it is also associated with intracellular potassium capacity for preservation and intracellular-to-extracellular potassium gradients, the latter is mostly dependent on the serum-to-dialysate potassium gradient [39].

The researchers demonstrated that in individuals with a previous diagnosis of chronic kidney disease (CKD) or those receiving diuretic medication, the fractional outflow of sodium has little impact in acute kidney injury (AKI) differentiation. When oliguria is evident, it is most beneficial [40]. Patients with chronic kidney disease (CKD) are more susceptible to the detrimental effects of salt on blood pressure levels due to an excess of fluid and direct damage to the renal function, heart, and [41]. By

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strengthening the antiproteinuric action of renin-angiotensin-aldosterone system (RAAS) restriction, a low-salt diet (LSD) helps non-dialysis CKD patients regulate their hypertension regardless of blood pressure levels and reduce proteinuria [42]. It is yet unknown if these changes might enhance the cardio-renal prognosis. However, because end-stage kidney disease (ESKD) is characterized by a common mismatch between sodium intake and elimination, which raises the risk of hypertension and cardiovascular disease (CV), salt restriction becomes even more important. Thus, for hypertensive CKD patients from early stages to ESKD, lowering salt consumption is essential [43].

Evenepoel et al., (2024)[44] showed decreased levels of serum calcium in CKD patients compared to controls, and these findings were agree with current research. Therefore, CKD patients must undergo calcium supplementation. Intestinal absorption of calcium, renal reabsorption, and calciotropic hormones—which initiate calcium exchanges from the bone when blood calcium levels are low—all have a role in maintaining calcium homeostasis [45]. Using active vitamin D analogues is one of the factors that contribute to changes in calcium metabolism. According to research, without the use of vitamin D analogs, people with CKD 3–4 may be able to maintain calcium homeostasis by consuming 800–1000 mg/d of calcium [44] . However, because renal function has not yet upset calcium homeostasis, calcium prescriptions for the initial phases of CKD usually follow the recommended dietary allowance (RDA) (1000–1200 mg/d) for adults, but this varies depending on the person. According to Vervloet et al. (2023)[46], vitamin D plays a crucial role in preserving calcium equilibrium. In order to improve calcium reabsorption and avoid elevated blood parathyroid hormone (PTH) and bone turnover, vitamin D (VD) supplementation treatment is recommended for patients with chronic kidney disease (CKD) who have inefficient active VD levels [47]. VD supplementation in CKD patients achieves enough active VD, which is important for calcium homeostasis, according to compelling research[48] (table 2).

ROC curve of our results showed the urea, creatinine and albumin indicators scored best markers in screening patients with renal failure due to have these indicators highest sensitivity and specificity compared to another markers. These findings were matched with results Okuyan et al., (2021)[49]. Therefore, we recommend the researchers to studying another markers in diagnosis renal failure disease in addition to urea, creatinine and albumin indicators.

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Additionally, for the diagnosis of diabetic kidney disease (DKD), a ROC curve assessment of a prior research showed that reduced albumin (rHA) and effective albumin (eHA) had a much higher sensitivity and specificity than total albumin concentration (tHA). Crucially, the eHA was found to be an independent predictor of DKD based on the multivariate logistic regression analysis (Nugnes et al., 2024).

Chang et al., (2024)[34] showed the AUC of potassium in renal failure patients was (0.845), and this study was matched with our study (AUC= 0.879). These findings refer to importance of potassium in predicting patients with CKD.

According to earlier research, blood sodium levels at the time of acute kidney injury (AKI) diagnosis may be able to predict in-hospital mortality in AKI patients (AUC = 0.621) (Marahrens et al., 2023). Our research had a higher AUC (0.747) than this one. The use of sodium in the diagnosis of renal disorders needed further research.

The predictive practicality of serum calcium isotopes (δ44/42Ca) to identify medial artery calcification in CKD is quite good (AUC=0.818, sensitivity 81.8%, and specificity 77.3%), according to the ROC curve analysis of a previous investigation. These results were almost identical to those of the current study that uses calcium for assessing CDK patients (AUC = 0.86, sensitivity 80%, and specificity 70%) (Table 3).

Finally, based on pearson correlation coeffecient , our findings showed there is positive and negative correlations among biochemical and electrolytes indicators in CKD patients due to kidney damage and subsequently lose of homeostasis (Table 4).

Conclusion

We concluded the renal failure disease is more effect on patients >40 years with underweight BMI. No effect of gender, living and smoking on disease. Increase and decrease levels of all indicators are related to kidney damage and lose of homeostasis. Urea, creatinine and albumin indicators are more preferred in screening disease due to have these indicators high sensitivity and specificity than another indicators.

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