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Demographic Variations and Their Impacts on Biochemical and Hematological Profiles in Patients with Chronic Renal Failure

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ABSTRACT

Background: *Chronic renal failure permanently harms the kidney and results in significant metabolic derangements. It is one of the key global health challenges of our times.* ***Aim:*** *The study evaluated the biochemical and hematological changes in the patients affected by chronic renal failure as compared to healthy ones.* ***Methods:*** *A total of 80 subjects participated in the study, out of which 40 were diagnosed as CRF patients. 40 age-matched healthy subjects were done as well. Serum urea, creatinine, uric acid, albumin, calcium, and hemoglobin level measurement will be done using standard laboratory methods. Patients and controls exhibited clear differences.* ***Results:*** *In the group of individuals with chronic renal failure the serum urea reached a mean value of 179.41 ± 58.27 mg/dL and that of creatinine 8.80 ± 1.79 mg/dL which were statistically significantly elevated ($p < 0.001$). 25% of cases were seen in individuals aged 51–60 years, which was the highest proportion. The calcium level observed was lower at 8.06 ± 1.37 mg/dL and the albumin level was reduced to 3.85 ± 0.75 g/dL. There was also evidence of renal anemia as noted by the decline of hemoglobin to a mean value of 9.45 ± 1.96 g/dL ($P < 0.001$).* ***Conclusion:*** *The chronic renal failure disorder causes an impairment of the filtration and endocrine kidney functions. The first stages of biochemical marker monitoring and management of metabolic risk factors, may slow down the disease. According to the laboratory changes observed, the clinician can assess the severity of the disease and anticipate developing complications like cardiovascular disorders and renal bone disease.*

Keywords: Chronic Renal Failure, Azotemia, Renal Anemia, Hypoalbuminemia, Glomerular Filtration Rate, Metabolic Homeostasis.

Article Information

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INTRODUCTION

He kidney is considered one of the most important organs in the human body because it plays a central role in maintaining physiological balance. It not only filters metabolic waste products but also helps preserve the internal environment by regulating fluid volume, maintaining acid–base balance, and controlling electrolyte concentrations (Johnson & Feehally, 2021; Skorecki et al., 2023). The renal system maintains the level of vital ions such as sodium, potassium, calcium and phosphorus which helps in maintaining cardiovascular and neuromuscular function (Webster et al., 2017). The kidneys also act as an endocrine organ because they secrete erythropoietin for stimulation of blood cell formation and activates vitamin d for bone health and regulating systemic blood pressure through the renin-angiotensin-aldosterone system (Zeisberg & Neilson, 2020; Stevens & Levin, 2013).

When these complex regulatory systems fail the result is a clinical syndrome known as renal

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failure. Renal failure is no longer considered to be only a dysfunction of the kidney, but rather a multi-system crisis that triggers a cascade of metabolic derangements involving the hematopoietic, nervous and cardiovascular systems (Ronco et al., 2019). The global burden of kidney damage has increased massively. There is a need to understand the two main types of kidney damage - CKD and AKI.

Chronic Kidney Disease: Progressive Loss of Renal Function

Chronic Kidney Disease (CKD) is described as a long-term decrease in kidney function. This is usually defined for the purposes of classification as a glomerular filtration rate (GFR) below 60 mL/min/1.73 m^2 or alternatively evidence of kidney damage which has lasted over three months (Levey & Coresh, 2012; KDIGO, 2024). The condition arises gradually owing to the loss of nephrons over time. There occurs compensatory hyperfiltration and increasing fibrosis (Romagnani et al., 2017). The leading causes of kidney damages are hypertension and diabetes mellitus. Though glomerulonephritis and polycystic kidney disease also contribute significantly towards the progression of the disease (Hill et al., 2016; Bello et al., 2017). As CKD worsens these changes result in a decreased filtration capacity that causes continual systemic complications.CKD affects the clinical situation beyond just the kidneys. When nitrogenous waste products accumulate, it can lead to what are known as uremic symptoms, systemic inflammation and higher cardiovascular disease risk (Massy & Drueke, 2017). Disorders of mineral metabolism also occur and they can cause Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD) which is characterised by hyperphosphataemia, secondary hyperparathyroidism, vascular calcification, and increased cardiovascular mortality (Moe et al., 2006; Isakova et al., 2011; Block et al., 2013).

Acute Kidney Injury: Rapid Decline in Renal Function

Acute Kidney Injury (AKI) is a rapid reduction in kidney function, occurring over hours to days, distinct from CKD and potentially reversible (Bellomo et al., 2012) . The underlying cause of AKI is often classified into prerenal, intrinsic and postrenal. The kidney perfusion is reduced in Prerenal AKI due to sepsis, bleeding and heart failure (Hoste et al., 2015). Intrinsic AKI refers to the impairment of the kidney parenchyma such as acute tubular necrosis which can occur due to ischemia or use of nephrotoxic drugs (Bouchard & Mehta, 2011; Chertow et al., 2005). When the obstruction of urinary flow occurs due to the presence of kidney stones or an enlargement of the prostate, postrenal AKI takes place (Mehta et al., 2007). Recent studies indicate that AKI and CKD may not be distinct but interconnected forms of kidney disease. According to Heung & Chawla in 2014 and Coca et al. in 2012, recurring injuries may exacerbate chronic kidney harm and trigger chronic kidney injury.

Biochemical Correlates and Diagnostic Biomarkers

Relying on biochemical markers that reflect damage and loss of function, renal failu diagnosis and monitoring is definitely stronger. Serum creatinine remains the one of the frequently used estimators of glomerular filtration rate (GFR). It inference is affected by muscle mass and nutritional status (Perrone et al., 1992; Inker et al., 2012). Blood urea nitrogen (BUN) is a helpful test. An increase in BUN generally indicates accumulation of waste in blood and change in the way body breaks down protein (Arnett et al, 2014). According to doctors, BUN is used with various other tests to measure the efficiency of the kidneys and follow the changes in kidney disease. Severe kidney disease also often causes problems with minerals and salts. As an example, phosphate often become high and sometimes calcium often become low which can weaken bones and cause

calcium deposits in soft tissues (Goodman et al, 2000; Slatopolsky et al, 1999). Chronic kidney disease may also give rise to anemia early on. The kidneys are producing less of the hormone erythropoietin, which encourages the body to make red blood cells. Uremia can also cause red blood cells to break down more quickly than normal, exacerbating the condition of anemia (Babitt and Lin, 2012; Astor et al., 2002). Monitoring of serum albumin, in the long run, is among prognostic markers. Its low level usually reflects malnutrition along with chronic inflammation (Kaysen et al, 2004; Don & Kaysen, 2004). In addition, elevated uric acid has been known to damage renal vasculature and acts as an indicator of impaired purine metabolism (Kim et al., 2000; Johnson et al., 2013). These biochemical abnormalities show that renal failure is much more than a simple filtration effect. Effective control of renal failure can be possible with early diagnosis and treatment. According to Meyer & Hostetter (2007), Alghythan & Alsaeed (2012), and Levin et al. (2013), clinical management typically involves pharmacological nephroprotection, diet modification, and renal replacement therapy, including dialysis and transplantation.

MATERIALS AND METHODS

Study Design and Participant Selection:

The purpose of this cross-sectional case-control study was to assess biochemical changes in patients with CRF. The participant made up of 40 male patients suffering from CRF who were undergoing maintenance hemodialysis at the Hemodialysis Unit of Al-Hakeem Teaching Hospital, Najaf, Iraq, from March to July 2021. The diagnosis and inclusion criteria were based on Kidney Disease: Improving Global Outcomes (KDIGO) recommendations and clinical history, persistent reduction of estimated glomerular filtration rate (eGFR), and supporting laboratory tests (KDIGO, 2024; Levey et al., 2020).

To make a comparison, another 40 age-matched healthy male's control group (20–70 years) was recruited.The control group was carefully screened for metabolic disorders, cardiovascular diseases and renal conditions (Stevens & Levin, 2013). These diseases could potentially affect biochemical findings. The institutional review board granted ethical approval for this study and written informed consent was obtained from all participants before the study.

Blood Sampling and Serum Preparation:

The sampling was done for the fasting blood samples (5-7 ml) was drawn after an overnight fast of at least 8 hrs from each participant to restrict post prandial variation of biochemical parameters. Samples were obtained from the patients before the beginning of the hemodialysis session, so that metabolic waste products measured during the peak phase. Blood was collected into sterile vacuum tubes without anticoagulant to separate the serum. Before performing the centrifugation of the samples at 3000 rpm for 10 minutes (Tietz, 2006) , they were allowed to clot at room temperature for 15 minutes. The separated serum was aliquoted and stored at −20 °C in the deep freezer to maintain the stability of proteins and prevent degradation of sensitive metabolites until biochemical analysis (Burtis et al., 2012).

Biochemical Analysis Protocols:

All biochemical measurements were carried out using standardized commercial kits and automated spectrophotometric analyzers in accordance with International Federation of Clinical Chemistry (IFCC) quality-control standards.

Nitrogenous Metabolites (Urea and Creatinine):

Serum urea was measured using the enzymatic urease-GLDH colorimetric method (Tietz, 1999). Serum creatinine was determined by the kinetic Jaffe reaction, in which creatinine reacts with alkaline picrate to form a colored complex that is measured spectrophotometrically (Jaffe, 1986; Peake & Whiting, 2006). These markers were used as

primary indicators of glomerular filtration efficiency.

Uric Acid Determination:

Serum uric acid was measured using the uricase-PAP enzymatic technique (Fossati et a!., 1980). During this test, first, the uric acid is broken down to allantoin and hydrogen peroxide. Subsequently, a color reaction occurs and the intensity of the color developed in the test solution is measured at a wavelength of 520 nm.

Mineral Profile (Calcium):

The o-cresolphthalein complexone (OCPC) method (Tietz, 1999; Endres & Rude, 1999) measured total serum calcium. When calcium reacts with a chemical reagent in alkaline solution, visually violet colour is formed in the test. This colour's intensity indicates the level of calcium in blood, and this gives a clear approximation of the balance of minerals.

Hematological Assessment (Hemoglobin):

For the measurement of hemoglobin in whole blood, the cyanmethemoglobin method was used. Cyanmethemoglobin method which is color-based is the standard method that is used for the detection of anemia due to kidney disease (Patel, 2010; Beutler & Waalen, 2006).

STATISTICAL ANALYSIS

Data were arranged and their analysis were conducted using the software Microsoft Excel and SPSS version 26.0. The Shapiro-Wilk test was used to check normality, and values are presented as mean ± standard deviation (SD).This study employed a variety of statistical tests. We applied the independent Student's t-test for comparison of biochemical values between patients and healthy controls. The extent of relationship between glomerular filtration rate and selected biochemical markers viz. urea, creatinine, calcium was examined by using Pearson's correlation coefficient (r) Demographic variables were evaluated using a chi-square test. Also, regression analysis was done to find out if some markers could predict the progression of renal failure.P-value since .05 was taken as statistically significant ($P < 0.05$) (Altman, 1991; Rosner, 2015).

RESULTS

Demographic Analysis of the Study Population

The frequency distribution of chronic renal failure patients showed variation between age groups. According to **(Table 1)**, the maximum number of cases (10 patients) were found in the age group of 51-60 years (25 percent) of the study population. The second largest group, accounting for 22.5%, or 9 patients, was 31 - 40 years. The findings indicate that the population had a high frequency of kidney disease occurring in age groups.

Table 1: The frequency distribution of chronic renal failure patients showed variation between age groups.

Age Interval (Years)	Patient Count (n=40)	Relative Percentage (%)	Cumulative Percentage (%)
20 – 30	5	12.5%	12.5%
31 – 40	9	22.5%	35.0%
41 – 50	8	20.0%	55.0%
51 – 60	10	25.0%	80.0%
61 – 70	8	20.0%	100.0%
Total	40	100%	—

Comparative Assessment of Biochemical and Hematological Markers

Compared to healthy individuals, CKD patients showed a clear difference in metabolic and hematological indicators. According to **Table 2**, CKD patients had elevated nitrogenous waste products and decreased levels of various physiological indicators.

Research indicated that accumulation of nitrogenous waste products was one of the key indicators of kidney malfunctioning. Comparison of Study Groups. In the sampled population, serum urea, creatinine levelsand uric acid were significantly higher in patients than in healthy controls as shown in **(Table 2)**, The average serum urea level was found to be (179.41 ± 58.27, 8.80 ± 1.79 and 10.95 ± 3.28 mg/dL respectively) in patients while it was (20.70 ± 2.17,0.81 ± 0.16 and 5.38 ± 1.62 mg/dL respectively) in controls. Such a large increase indicated that the kidneys were not filtering waste and were unable to filter protein breakdown. It was observed that metabolic balance was profoundly affected in patients suffering from chronic renal failure.

The results of **Table 2** showed decreased calcium and albumin levels in a patient with chronic kidney disease. The mean calcium level was (8.06 ± 1.37 mg/dL) in the patient, while the mean in healthy individuals was higher, at (9.56 ± 0.58 mg/dL). This decrease suggests that kidney disease causes an imbalance in minerals. The decreased serum albumin level.

The results of **Table 2 showed** Blood tests much lower hemoglobin levels in patients of chronic renal failure. The average hemoglobin level in the patient group was (10.95 ± 3.28g/dL); whereas, in the healthy individuals, it was (5.38 ± 1.62g/dL), confirming renal anemia. Moreover, the level of uric acid in patients was high which indicates the lesser ability of the kidney to remove uric acid and changes of purine metabolism due to chronic damage to the kidney.

Table 2. Comparison of Biochemical and Hematological Parameters between Patients with Chronic Renal Failure (CRF) and Healthy Controls.

Parameter (Unit)	CRF Patients (n = 40)	Control Group (n = 40)	P-value
Serum Urea (mg/dL)	**179.41 ± 58.27**	**20.70 ± 2.17**	**< 0.001 ****
Serum Creatinine (mg/dL)	**8.80 ± 1.79**	**0.81 ± 0.16**	**< 0.001 ****
Serum Uric Acid (mg/dL)	**10.95 ± 3.28**	**5.38 ± 1.62**	**< 0.001 ****
Serum Calcium (mg/dL)	**8.06 ± 1.37**	**9.56 ± 0.58**	**< 0.05 ***
Serum Albumin (g/dL)	**3.85 ± 0.75**	**4.41 ± 0.41**	**< 0.05 ***
Hemoglobin (Hb) (g/dL)	**9.45 ± 1.96**	**15.18 ± 1.14**	**< 0.001 ****

Data are expressed as mean ± standard deviation.

**Statistically significant at p < 0.05, ** Highly significant at p < 0.01.*

DISCUSSION

The biochemical changes in patients with chronic kidney disease indicate that, at this stage, the kidneys can no longer maintain homeostasis in the body When kidney function declines, waste products build up (uremia) because of the impairment of the excretory, hormonal and metabolic functions of the kidney (KDIGO, 2024; Webster et al., 2017). The findings indicate that the disease affects many kidney functions simultaneously.

Epidemiological Shifts and Age-Related Vulnerability:

In this investigation, middle age may be a risk factor for chronic kidney disease (CKD). The maximum number of cases observed was in

age group 51-60 years which is 25% of the study group. This implies the kidney decline may commence earlier in this population than previously reported in the Western world (Hill et al., 2016; Al-Rubeaan et al., 2017). This may be due to genetic factors and poor control of disease (eg, type 2 diabetes and hypertension), although kidney function usually declines slowly with age because of structural changes (Denic et al., 2017; Glassock & Rule, 2012). The findings endorse the preventive measures for early screening programs of the local community (Kadhum, 2008; Stevens & Levin, 2013).

Azotemia and the Uremic Environment:

This group has developed severe kidney dysfunction, as demonstrated by the significant rise in serum urea and creatinine levels. The average level of serum urea was 179.41 ± 58.27 mg/dL and that of creatinine turned out to be 8.80 ± 1.79 mg/dL which means that the kidneys are not filtering the waste efficiently. The accumulation of creatinine is widely used as an indicator of diminished kidney filtration (Inker et al., 2012; Peake & Whiting, 2006). Elevated levels of these toxic substances may induce oxidative stress and injure endothelial cells, thereby inducing a uremic milieu which contributes to the complications of hemodialysis patients (Vaziri, 2004; Massy and Drueke, 2017; Liakopoulos et al., 2017; Ronco et al., 2019).

Protein-Energy Wasting (PEW) and Hypoalbuminemia:

This study found lower serum albumin levels, which shows that the patients with CKD have nutritional problems and have continuing inflammation. The average albumin level was reduced to 3.85 ± 0.75 g/dL, indicating poor protein balance. Reduced albumin levels may occur due to loss of albumin through damaged filters or decreased albumin production during chronic inflammation (Don & Kaysen, 2004; Moshage et al., 1987). Medically, this is known as Protein-Energy Wasting (PEW), which is associated with cardiovascular risk and poorer outcome in badly off renal patients (Kaysen et al., 2004; Fouque et al., 2008; Ronco et al., 2017). The importance of nutritional support and early intervention are highlighted.

Anemia Pathophysiology and Iron Sequestration:

One of the main findings of this study was the clear decrease in hemoglobin level realised which was 9.45 ± 1.96 g/dL shows the presence of renal anaemia. This type of anemia mainly occurs when the diseased kidney produces lesser than usual erythropoietin, which is a hormone that helps the human body to manufacture red blood cells. Uremic toxins, as well as issues with iron balance (Babitt & Lin, 2012; Erslev, 1997), can also worsen this condition. In CKD, increased hepcidin levels may limit the iron needed for red blood cell production (Ganz, 2003; Kausz, 2000). The findings suggest that anemia in kidney disease is due to multiple causes and cannot be treated with blood transfusion alone. Hormonal regulation and iron therapy are important to protect the heart and improve patients (Besarab & Levin, 2000; Costa et al., 2008; Hsu et al., 2001).

Mineral Homeostasis and Bioactive Toxins:

Patients had a mean calcium level of 8.06 ± 1.37 mg/dL, indicating that damage in the kidneys prevents activation of vitamin D (Slatopolsky et al., 1999; Shi & Wang, 2012). Such a problem may cause secondary hyperparathyroidism, which can then cause bone loss and renal osteodystrophy (Goodman et al. 2000; Block et al. 2013). On the other hand, increased uric acid of 10.95 ± 3.28 mg/dL may act as a metabolic toxin which is closely associated with impairment in the kidneys and vascular substrate (Johnson et al., 2013; Kim et al., 2000). The findings reveal the collaborative effort of mineral imbalance and metabolic toxicants in CKD progression.

Conclusion of Systemic Interactions

Chronic kidney disease affects other systems of the body that lead to biochemical and hematological changes in the body. Kidney damage may worsen due to diabetes and high blood pressure, which are illness-causing conditions (Bello et al., 2017; Levey et al., 2020). The selected laboratory indicators nitrogenous waste products and hematological indices monitored early may help postpone the onset of ESKD and assist with clinical decision-making, according to Levin et al. (2013) and Meyer and Hostetter (2007).

CONCLUSION

Chronic renal failure causes, as indicated by the biochemical and haematological results of the study, a considerable metabolic derangement due to the accumulation of nitrogenous waste products and decreased excretion from the kidney. Results of the study show that the most affected age group (51–60 years) indicates that diabetes and hypertension may induce kidney disease at an earlier age among people of this age group. A significant decrease of hemoglobin is a strong pointer of renal anemia due to slumping hormone production by kidney. The indication that the nutritional status and mineral balance in the body are disturbed is less calcium and albumin level which raises the risk of Protein-Energy Wasting (PEW) and cardiovascular (CV) complications. The rise of uric acid levels also indicates the metabolic changes in CKD. The data indicate that it is necessary to try to intervene as early as possible by laboratory monitoring, which slows the ongoing evolution of the clinical picture with subsequent lessening of complications.

Recommendations

Preliminary Evaluation Programs:

Regular testing of kidney function (eGFR and albuminuria testing) may help identify high-risk people for chronic kidney disease (CKD). It is important to detect the disease in young people. Particularly individuals over 40 years old who have either diabetes or hypertension.

Handling various fields:

Controlling blood pressure and glucose via a cardio-renal care approach may slow the disease's progression. A multi-disciplinary approach from health professionals can improve patient care and delay the onset of end-stage renal disease.

Aid for Nutrients and Minerals:

The balance of minerals can be improved by supplements of Vitamin D, Calcium along with personalized nutrition. Implementing these strategies helps to reduce the incidence of bone complications in patients with chronic kidney disease and metabolic stabilization.

Improved anemia management:

Regular monitoring of hemoglobin levels with early initiation of erythropoiesis-stimulating agents and iron therapy when needed may enhance management of renal-associated anemia and increase quality of life.

Future Molecular Research:

We need to carry out studies on larger cohorts of patients over a longer period to study the emerging biomarkers Cystatin C and NGAL. These markers may help with the detection and monitoring of kidney injury.

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