Biochemical evaluation of chronic renal failure

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Abstract

Failure of the kidneys to eliminate waste materials and maintain an internal balance of water and salts in the body is referred to as renal failure. In this research, individuals with renal failure were studied during September 2023 at Ibn Sina center for hemodialysis in Baqubah teaching hospital in Diyala province. This study included 25 samples of patients (males) with kidney failure, and the different parameters compared with the same variables in 25 females. During this study, some hematological variables examined, which included the concentration of hemoglobin, and biochemical variables in the blood such as the concentration of

albumin to determine the most significant physiological changes associated with renal failure and to compare the results. In patients with renal failure, the hemoglobin concentration in males was found to be not significantly changed than in female individuals. In addition, albumin level was shown to be not statistically altered and individuals with renal failure had no significant changes in urea, creatinine, and uric acid, as well as levels of calcium ions when compare the blood samples of male and female. Chronic renal illness has been shown to have a detrimental influence on the majority of physiological and biochemical variables examined in this research.

Introduction

Renal failure is a disorder where the kidneys fail to eliminate metabolic waste products from the blood and maintain the proper concentration, pH, and volume of extracellular fluids. Urologic problems of nonrenal origin, systemic disease, or renal disease could be the underlying cause. Both acute and chronic kidney failure are possible conditions (1). Chronic renal failure, in contrast to acute renal failure, is characterized by the progressive and permanent degradation of kidney structures. The high death rate was linked to insufficient treatment for renal disease as well as the extremely high cost of continuing medical treatment (2).

The kidneys have a variety of roles in preserving glucose homeostasis, including gluconeogenesis and glucose reabsorption. Normally, glucose filtered by glomeruli is fully reabsorbed; however, glucosuria can happen when there is hyperglycemia or a decrease in resorptive ability. By reabsorbing extra glucose, the kidneys can exacerbate hyperglycemia (3). This can lead to chronic hyperglycemia, which in turn can cause insulin resistance, reduced glucose absorption, and pancreatic β-cell failure. In turn, hyperglycemia damages glomeruli in the kidneys, which leads to microalbuminuria and nephropathy (4). An early indication of renal failure is the buildup of nitrogenous wastes, which typically happens before other symptoms show up. One of the first nitrogenous wastes to build up in the blood is urea, and as renal failure worsens, the blood pressure level rises more and more. Urea in plasma typically has a normal concentration of less than 20 mg/dL. This level can reach 800 mg/dL in cases of renal failure. A high creatinine level is indicative of renal disease or poor kidney function. The low renal clearance (removal) of creatinine will cause the blood's creatinine level to rise when the kidneys deteriorate for whatever reason. Thus, abnormally elevated creatinine levels indicate a potential renal malfunction. This is the reason why the level of creatinine in the blood is regularly measured by conventional blood tests. As a byproduct of muscle metabolism, creatinine is freely filtered in the glomerulus and does not return to the renal tubules after being reabsorbed. The production of creatinine is comparatively constant, and any creatinine that gets filtered out of the bloodstream in the glomerulus is lost in the urine (5). As a result, serum creatinine can be used as a proxy for measuring GFR and the degree of renal damage sustained during renal failure. Age and muscle mass have an impact on serum creatinine levels since it is a byproduct of muscle metabolism. Ninety percent of renal function is thought to have been lost when serum creatinine levels reach 10 mg/dL or above. A threefold increase in creatinine means that seventy-five percent of renal function has been lost. The level of blood

urea nitrogen (BUN) serves as an additional measure of renal function. Another metabolic waste that can accumulate in cases of compromised renal function is urea. When compared to the creatinine level alone, (6) the BUN-to-creatinine ratio typically yields more accurate information on renal function and any potential underlying causes. Uncertain causes may underlie the observed decrease in blood levels of liver enzymes in patients with chronic kidney disease (CKD) on HD compared to those with normal renal function. The diagnosis, clinical management, and course of treatment of liver disease in these patients may be negatively impacted by this profile (7). Multivalent cations including calcium, phosphate, and magnesium are necessary for a variety of biological and cellular processes. An essential part of maintaining these ions' equilibrium is the kidneys (8). Renal control of these ions is a key factor in determining plasma ion concentration since it happens via glomerular filtration, tubular reabsorption, and/or secretion. Under physiological conditions, urinary excretion is finely adjusted to equal net intake in order to maintain the body's calcium, phosphate, and magnesium balance (9). Early on in chronic renal failure, abnormalities of calcium, phosphate, and vitamin D are observed. They include parathyroid hormone (PTH) regulation, vitamin D activation, and renal control of serum calcium and phosphate levels. An amount equal to what is absorbed from the diet must be excreted in the urine each day in order to maintain serum phosphate levels. Phosphate excretion is hampered by declining renal function, which raises serum phosphate levels. Because serum calcium and serum phosphate levels are inversely controlled, serum calcium levels decrease at the same time (10). The decrease in serum calcium then prompts the release of PTH, which increases the absorption of calcium from bone. Chronic stimulation of the parathyroid glands results in secondary hyperparathyroidism, which affects most patients with end-stage renal disease (ESRD). Even though

elevated PTH function keeps serum calcium levels stable, the skeletal system and other organs suffer as a result of this adjustment.

Dialysis or transplantation can be used as a form of renal replacement treatment in addition to conservative management of renal insufficiency to treat chronic renal failure. Conservative treatment consists of actions to help the body adjust to the impairment already present and to stop or slow the decline of residual renal function. Interventions such as dietary protein restriction and blood pressure stabilization have been demonstrated to dramatically slow the progression of chronic renal insufficiency. To make up for decreased renal function and treat the anemia, hypocalcemia, and acidosis that occur, a variety of therapies are employed (11). Patients with end-stage renal disease (ESRD) frequently receive these therapies in addition to dialysis therapy. The aim of this research is to observe some variables (albumin, blood sugar, calcium, blood nitrogen, phosphate, total protein, Alk, Tsb) in a group of dialysis patients.

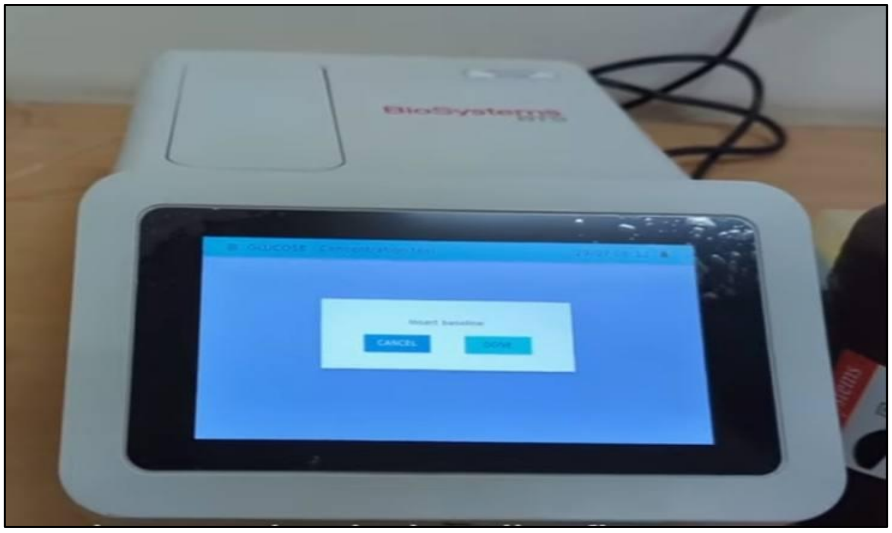
Materials and Methods:

The study was conducted in the Ibn Sina center for hemodialysis in Baqubah teaching hospital in Diyala province, during September 2023.The study included 50 patients (25 females and 25 males).

We reviewed all diagnosed patients and their medical records after taking the permission of Ibn Sina doctors and special questionnaire formula used to address the vital issues of the study. Fifty patients diagnosed as renal failure and started on hemodialysis in Ibn Sina center for HD, whom were diagnosed by specialist

physicians in this center and or referred from private clinics. Patients were categorized according to sex (males and females).

Statistical analysis was performed using SPSS program, where a t-analysis was performed for two independent samples.



Device used in biochemical analyses

Results:

The results (Table 1) show that there is a minor difference in the average levels of blood sugar between males and females with a probability value of (0.9) that is higher than the significance level (0.05). Accordingly, there is no statistically significant differences between the average blood sugar levels for females and the average male with kidney failure. It is clear from the table that the average blood nitrogen (BN) level for males is higher than the average for females with a probability value of (0.17) that is slightly higher than the significance level (0.05).

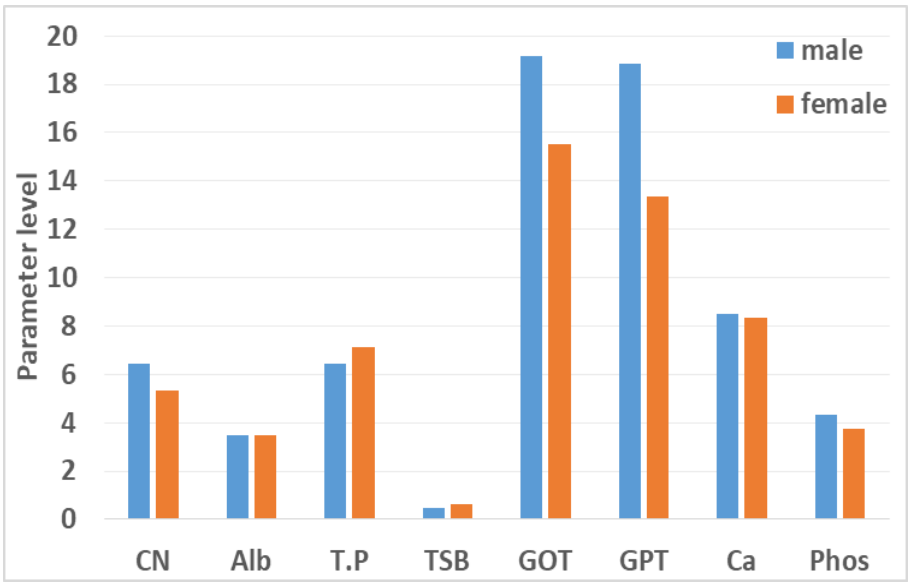
Therefore, there is no significant differences between the average percentage blood nitrogen for females and the average for males with kidney failure.

It can be seen from the data in Table 1 that there is only slight differences in the values of serum creatinine, albumin, total proteins, alkaline phosphatase, TSB, GOT, GPT, iron, unsaturated iron binding capacity (UIBC), calcium, and phosphorus between women and men. The p value of these results did not show any significant differences between males and females. Overall, this disease did not affect males and females differently in these measures and no statistically significant difference was observed between women and men groups. The levels of the measured parameters in males and females with CKD are shown in Figure 1.

Table 1. Biochemical changes associated with kidney failure in males and females

Parameters	Male (No. 25) Mean ± SD	Female (No. 25) Mean ± SD	P - value
Blood sugar (BS) mg/dl	137.77 ± 81.43	136.81 ± 63.53	0.9
Blood nitrogen (BN)mg/dl	126.25 ± 48.55	111.03 ± 26.57	0.17
Creatinine (CN) mg/dl	6.43 ± 2.49	5.32 ±1.95	0.08
Albumin (Alb)g/dl	3.49 ± 0.462	3.48 ± 0.326	0.88

Total Protein g/dl	**6.46 ± 1.34**	**7.12 ± 1.05**	**0.06**
Alkaline phosphatase (ALP) UI/L	**185.76 ± 84.79**	**187.73 ± 92.26**	**0.9**
TSB mg/dl	**0.49 ± 0.33**	**0.65 ± 0.41**	**0.13**
GOT UI/L	**19.18 ± 14.85**	**15.5 ± 6.72**	**0.26**
GPT UI/L	**18.87 ± 15.02**	**13.36 ± 5.57**	**0.95**
Iron µg/dl	**94.89 ± 54.84**	**81.98 ± 46.202**	**0.37**
Unsaturated Iron Binding Capacity (UIBC) µg/dl	**169.58 ± 97.49**	**165.04 ± 77.36**	**0.85**
Calcium mg/dl	**8.51 ± 1.79**	**8.34 ± 1.26**	**0.7**
Phosphorus mg/dl	**4.31 ± 1.33**	**3.76 ± 1.16**	**0.12**



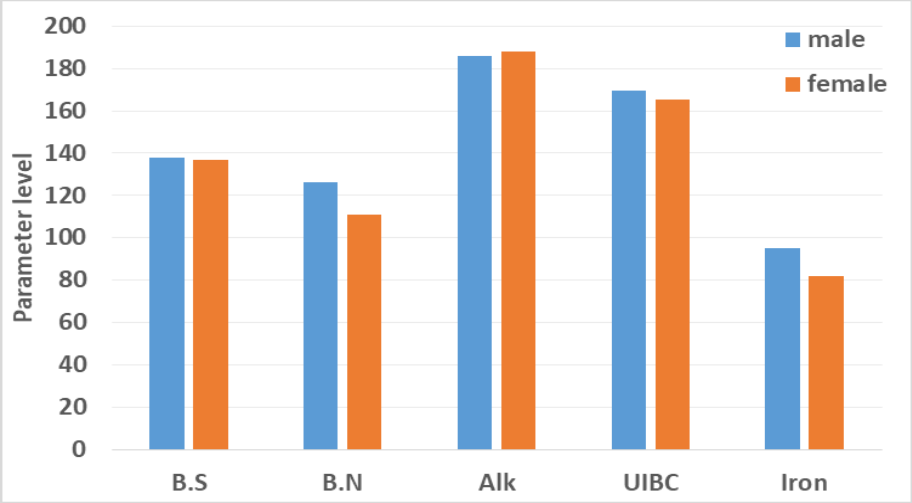


Figure 1. Biochemical changes associated with kidney failure in males and females

Discussions

In this study the results show that there is no statistically significant differences between the average blood sugar levels for females and the average male with kidney failure. Urea, creatinine, albumin, and uric acid in blood serum are utilized as diagnostic tests for kidney function. Female and males patients with renal failure had no statistically significant differences between the average of females and the average for males with kidney failure. They had no statistically significant differences between the average of renal function (B.N, C.N) for females and the average for males with kidney failure. Patients with renal failure have elevated levels

of urea and creatinine in their blood serum, which might be explained by the fact that deficiency in kidney function results in a reduction in the elimination of wastes, which leads to an increase in their concentration in the blood serum. As a consequence of the kidneys' inability to eliminate as much uric acid when the blood level is low, patients' males and females blood serum contains excessive amounts of uric acid. The GRF procedure, which leads in very high levels of uric acid in the blood. Non-enzymatic dehydration helps creatine be converted into creatinine. Our bodies continuously produce creatinine, which is then eliminated through the kidney's glomerular filtration unit. The rate at which creatinine is filtered by the kidneys can be influenced by reduced renal function, which can also be used to gauge kidney function. Renal function declines as a result of the kidneys' incapacity to excrete creatinine through urine, which raises the concentration of creatinine in the serum. In many mammals, urea is the main byproduct of protein catabolism and is created in the liver through a series of processes known as the urea cycle. Ammonia is converted to urea in the urea cycle, and the kidneys use the blood to remove the urea from the body. Increased urea levels in the blood may also be a sign of renal failure. When urea and creatinine concentrations in the blood rise in patients with chronic kidney disease (CKD), the body becomes extremely ill and is eliminated through the kidneys (12). Decreases in several calcium detecting receptors and fat-soluble vitamin D receptors inside the parathyroid glands create hypocalcemias due to hyperphosphatemia linked to chronic kidney disease. In addition, insufficiency of 1, 25 dihydroxycholecalciferol, also known as calcitriol, an active form of vitamin D that facilitates the intestinal absorption of calcium from diet (13). Significantly lower levels of calcium in CKD patients were found in a recent study by Salih H15 and AL Hisnawi RAAA than in controls (14). Similar findings were noted by Freethi R et al. (2016), who discovered that CKD patients had significantly higher levels of hypocalcaemia (9.8±0.45 mg/dl) than controls (10.17±0.37 mg/dl) (15). Patients

with chronic renal failure have hemoglobin Hb concentrations indicating that they are anemic. Previous research has shown that anemia is a prominent consequence of chronic renal failure [16]. A deficiency in the hormone erythropoietin, which promotes bone marrow erythropoiesis, causes chronic kidney disease. The iron deficit is one of the most significant causes of low hemoglobin levels [17]. Malnutrition in individuals with renal failure and immune cytokine suppression of intestinal iron absorption are two major factors contributing to low hemoglobin levels. So that people with chronic renal failure, who are accumulating waste nitrogen in their blood, develop anemia because the bone marrow is unable to produce red blood cells.

By appropriately excreting phosphorus through the urine in reaction to elevated levels, the kidneys serve a crucial role in maintaining phosphorous homeostasis and ensuring that serum phosphorus concentrations are sufficient for performing a variety of tasks. Serum phosphorus levels will rise in response to decreased GFR because less of it will be excreted (18). Since the kidneys are primarily in charge of maintaining fluid and electrolyte balance, acute or long-term alterations in renal function can lead to a variety of abnormalities. Nursing assessment and action are vital to preventing complications and potentially fatal consequences due to the severe renal impairment that can occur quickly. Patients with acute or chronic renal failure are more likely to experience metabolic acidosis, hyperphosphatemia, hyperkalemia, hypovolemia, and hypocalcemia. Although sodium is usually retained, dilution from fluid retention might lead it to seem normal, or hyponatremic.

Conclusions

The hematological variables examined, which included the concentration of hemoglobin, and biochemical variables in the blood such as the concentration of

albumin to determine the most significant physiological changes associated with renal failure and to compare the results. In patients with renal failure, the hemoglobin concentration in males was found to be not significantly changed than in female individuals. In addition, albumin level was shown to be not statistically altered and individuals with renal failure had no significant changes in urea, creatinine, and uric acid, as well as levels of calcium ions when compare the blood samples of male and female.