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STUDY THE LEVELS OF ERYTHROFERRONE, UREA AND CREATININE IN CHRONIC KIDNEY DISEASE PATIENT IN KIRKUK CITY

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Abstract

The dysfunction of kidney functions is one of the most common diseases among people on a global scale. This disease can be classified as low levels of renal infiltration rate less than 60 ml/min. and the renal failure disease were divided into five stages that are evaluated through some important parameters, which include glomerular filtration rate, urea and creatinine levels in serum. the current study included 75 Patients of both gender, ages (40-70). They suffer from renal failure at different stages which has been classified into five stages and each stage include15 sample as well as, 15 healthy people as a control group. The current study aimed to estimate the concentration of erythroferon, urea and creatinine in CKD patients. The results showed a significant decrease ($p \leq 0.01$) in the concentration of erythroferon in CKD patients compared with a control group. the results showed significant increase in urea and creatinine levels in CKD patients compared with a control group. With decrease the glomerular filtration rate (GFR), its concluded from the current study that the concentration of erythroferon decreased and the levels of creatinine and urea increased during the stages of chronic renal failure.

Keywords: Erythroferon, urea, creatinine, chronic renal failure.

Introduction

Renal failure (RF) has become more prevalent worldwide than it used to be. As it affects 10-15% of the adult population in all countries, many of them need renal replacement therapy. RF is defined as structural or functional abnormalities or a decreased glomerular filtration rate of less than 60 mL/min for more than 3 months, with or without kidney damage. Pathological events include pathological renal structure or functional abnormalities, signs of kidney damage, or abnormalities on imaging tests. Kidney failure can occur as an acute or chronic disorder (1). Erythroferrone (ERFE) is the master regulator of erythropoiesis in association with hepcidin, a hormone that controls levels of plasma iron and total iron in the body. When the release of erythropoietin from the kidney stimulates the production of new

red blood cells, it also increases the synthesis of ERFE in erythrocytes in the marrow. bone. Increased ERFE inhibits hepcidin synthesis, thus mobilizing cellular iron stores for use in heme and hemoglobin synthesis. Recent studies have shown that ERFE inhibits hepcidin transcription by inhibiting protein pathways in hepatocytes. When erythropoiesis is ineffective, pathological overproduction of ERFE leads to via Increasing the number of erythrocytes, hepcidin inhibition, and causes iron overload. ERFE may be a useful biomarker of ineffective erythropoiesis and an attractive target for treatment of its systemic effects (2). Urea is one of the non-protein nitrogenous wastes in the blood. It is characterized by its toxicity to the body, so it must be eliminated from the body through the kidneys, as urea constitutes about 50% of the nitrogenous waste (3). The concentration of urea in the blood reflects the balance between the production of urea in the liver and the excretion of urea by the kidneys in the urine, so the increase in urea in the plasma can be due to increased production of urea, or decreased elimination of urea or a combination of the two, and its concentration may reach the highest levels In the case of decreased excretion of urea in the urine due to advanced kidney disease (4). creatininea bout by-product of creatinine phosphate in muscle, it is released into plasma at a constant rate, freely filtered by glomeruli, and is not absorbed or absorbed through the kidneys, so it is excreted naturally in the urine(5)And the rate of its spread in the blood reflects the functions of the kidneys so it depends on creatinine more than urea, because its concentration in the blood is not affected, just as the concentration of urea is affected by the amount of protein in the food (6). Mostly, an increase in creatinine in the blood is caused by a decrease in renal filtration(7). Creatinine is one of the compounds that does not have reabsorption in the kidneys, so an increase in its concentration in the blood plasma is equivalent to an increase in its concentration in the urine if the other factors are normal (8).

Materials & Methods

Study Design

This study was conducted in the city of Kirkuk from the period between October 2022 to March 2023 in specialized clinics, and (90) blood samples were obtained, which included (75) samples from patients suffering from kidney diseases and (15) samples from the healthy group, whose ages ranged between (40-70) years.

Blood Samples

5 ml of venous blood was withdrawn from the patients and the control group, the blood was placed in a gel tube test tube, and then the blood serum was separated from it by centrifugation at a speed of 3000 rpm, and then the blood serum was separated and withdrawn in Eppendorf tubes and preserved In (-20) pm until the required tests are conducted.

Biochemical Analyzes:

The basic principle for estimating the concentration of erythroferon, urea, and creatinine through the use of a set of analysis tools from the French company Biolab, and it relies on the events of the enzyme-linked immunosorbent assay (ELESA) techniques.

Statistical Analysis:

The results of the current study were analyzed statistically using a program(SPSS-version 23) By using method T-test arithmetic means were chosen using Duncan's multiple range test at the level of probability ($p \leq 0.01$) to determine the differences significantly different between groups (9).

Results & Discussion

Erythroferon

The results of the current study in Figure (1) indicate a significant decrease in the concentration of erythroferon in all stages of chronic renal failure ($p \leq 0.01$) compared with the control group, no significant difference was observed in the concentration of the hormone between the third and fourth stages, which reached the stage first (0.64±27.23 ng / ml) and in the second stage (0.48±26.07 ng / ml) and in the third stage (0.30±25.33 ng / ml) and in the fourth stage (0.16±25.07 ng/ml (up to the fifth stage)0.81±23.03 ng/ml (compared to the control group, which reached (0.25±29.34 ng/ml)

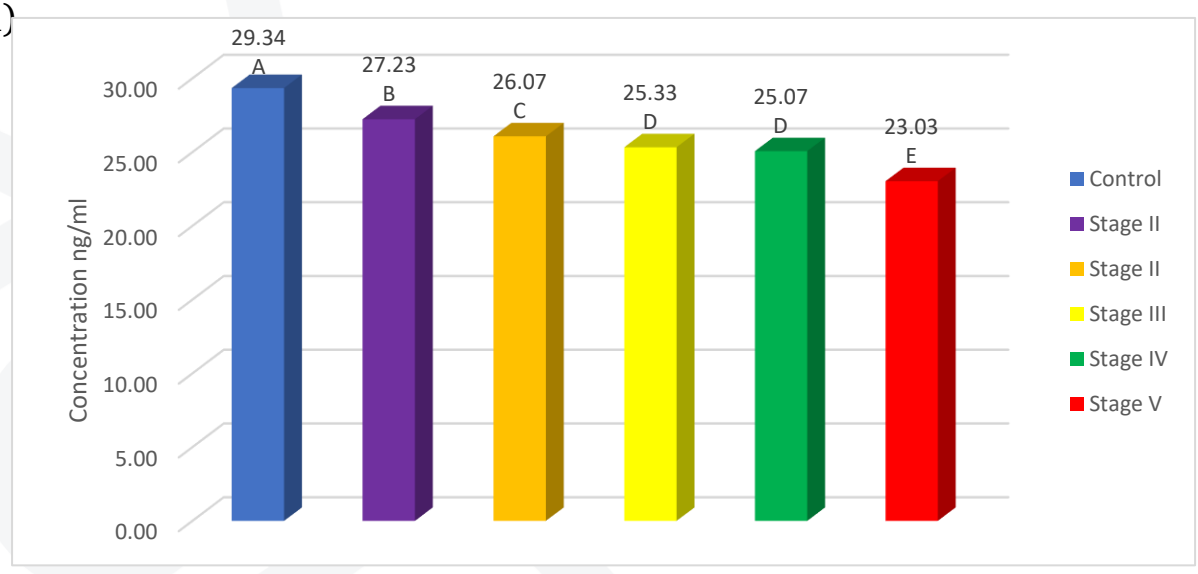

Figure (1): Concentrations of erythroferon in the serum of healthy and sick groups

The main reason for the decrease in erythroferon concentration is the decline in renal function in patients with chronic renal failure (10). It has been shown that there is a decrease in the levels of this hormone in people who suffer from poor kidney function (11). Another study indicated a decrease in the concentration of this hormone in patients with chronic kidney disease and its direct association with erythropoietin concentrations (12). The reason behind the decrease in the concentration of the hormone erythroferon may be due to the close association of this hormone with the hormone erythropoietin, which in turn increases the process of producing blood cells in the bone marrow, and that the high concentration of the hormone erythropheron leads to a decrease in the concentration of hepcidin, which in turn works to raise the concentrations of iron available in the plasma in normal cases for healthy people (13). Which causes a decline in the total production of erythrocytes in the blood and contributes to the process of anemia associated with the stages of decreased kidney efficiency, leading to cases of hemodialysis in patients with kidney failure, in which patients suffer from severe anemia that requires giving the patient doses of the hormone erythropoietin (14). For the purpose of correcting anemia and returning to acceptable limits of hemoglobin in these patients (15).

Urea

The results of the current study are shown in the figure(2)There was a significant increase in the concentrations of urea in the serum of patients with chronic renal failure compared with the control group. And that the gradient in moral elevation started from the first stage, which reached (0.90±67.00 mg / ml) and the second stage, which reached (2.89±88.07 mg / ml) and the third stage, which reached (2.90±101.88 mg / ml) and the fourth stage, which reached (5.02±153.7 mg / ml (up to the fifth stage, which reached (6.85±161.99 mg/ml (compared to the control group, which reached (3.18±43.59 mg/ml).

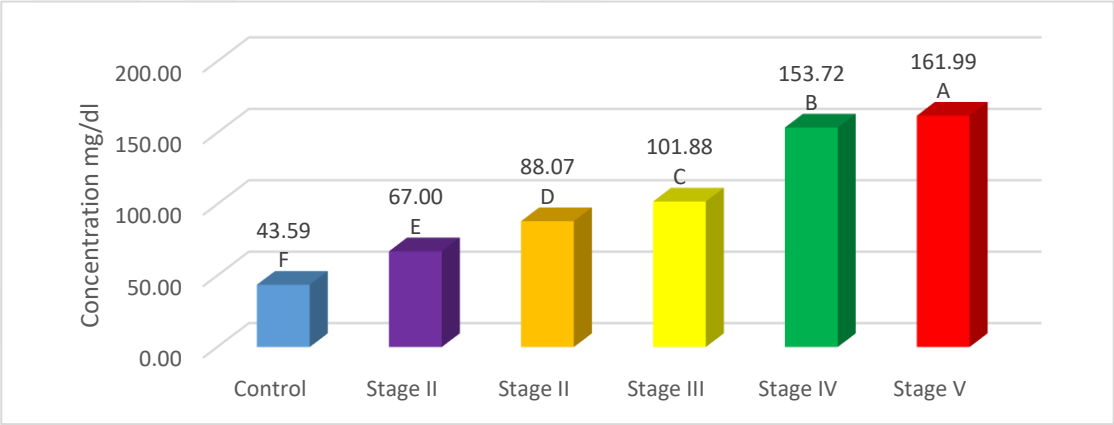

Figure (2): Serum urea concentrations in healthy and sick groups

The results of the current study were consistent with a study conducted on patients with chronic renal impairment and proved that blood urea concentrations rise with the progression of renal function impairment stages.(16). The results of our study were similar to a study that showed that blood urea concentrations rise significantly in patients with kidney failure, and that urea concentrations are proportional to the severity of the deficiency in kidney function.((17. The reason behind the high rate of blood urea in the category of patients who suffer from poor kidney function may be attributed to the fact that the source of filtration of urea is mainly through the renal glomeruli and then the nephrons, and that any imbalance in the function of the kidneys in a chronic manner may lead to a significant increase in urea concentrations. In addition, blood urea is directly affected by the amounts of protein consumed by the individual and in healthy people, and that frequent intake of proteins leads to an increase in the process of filtering urea through the kidneys, which leads to an increase in the concentration of this substance resulting from the metabolism of proteins and amino acids.(18). In addition to the foregoing, the concentration of urea filtration is directly affected by the concentration of dietary protein that enters the blood circulation, and whenever the pressure on the renal nephrons increases, the filtration rate increases, which leads to pressure on the filtration process, especially in patients with chronic renal insufficiency, in addition to that, urea concentrations are affected directly by the amount of glomerular filtration, the amount of protein in the blood, and the filtration rate in the renal tubules((19.

Serum Creatinine

The results of the current study in Figure (3) show that there are significant differences($P \leq 0.01$)Among the groups of patients with chronic renal failure in all its stages, compared to the healthy group, as the average creatinine in the first category was (0.13±1.318 mg / 100 ml), and in the second category (0.27±2.034 mg / 100 ml), and in the third category (0.39±3.770 mg/100 ml), and (0.55±8.802 mg / ml (in the fourth category, and)0.88±9.418 mg/ml) in the fifth category compared to the adult control group (0.836±0.06mg/mL) and as shown in the figure(4-8).

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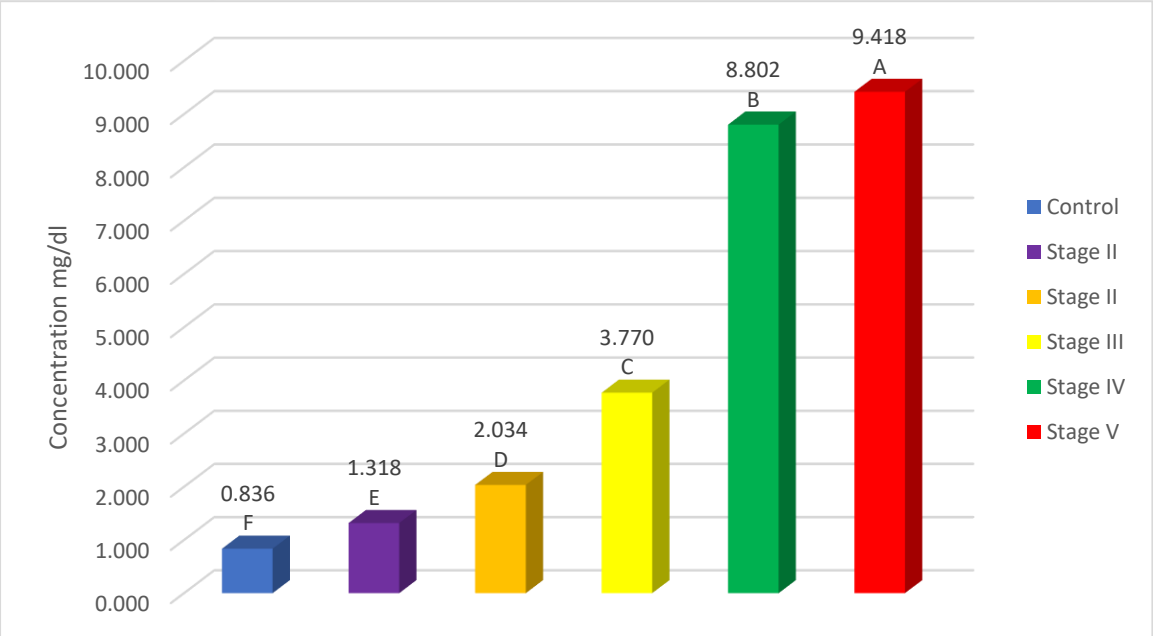

Figure (3) Serum creatinine concentrations of patients with renal impairment

The results of our study show that the rate of creatinine in the blood is directly related to kidney function, and that its concentration is closely related to the extent of deterioration of the renal filtration process in general, especially in patients with renal failure. The results of our study were consistent with the results of another study, which indicated an increase in serum creatinine concentration in patients with chronic kidney disease. A similar study also showed that creatinine concentrations increase exponentially with a decline in the work of renal nephrons and a weakness in the filtration process (20). It was found that the rate of creatinine was high in patients with renal failure, and this rise was proportional to the atrophy of the filtration function of the renal glomeruli(21). The agreement that we find in the results of the current study with the aforementioned of other researches may be due to the fact that the concentration of creatinine in the blood indicates more accurately and reliably in evaluating the performance of the kidneys, especially the renal tubules and glomeruli, being responsible for the secretion of unwanted substances from the blood (22). In addition to the fact that creatinine is not affected by the level of food consumed by people and is not affected by the amount of protein metabolism in the body. Which gives great importance to this vital compound in determining the efficiency of kidney function more accurately and more reliably than the blood urea test. It is known that creatinine is found in high concentrations in the muscles and that the metabolism process that takes place on it is not affected by the levels of protein metabolism, but the concentrations of this compound may be proportional to the age stage in addition to the person's muscle mass and the amount of secretion from the renal tubules.(23).

Conclusion

The results of the study showed a decrease in the concentration of the hormone erythroferon and an increase in levels of creatinine and urea in CKD patients.

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