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Investigation of the predictive roles of erythropoietin, ghrelin and biochemical parameters in patients with renal failure

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Abstract

Introduction: kidney dysfunction is the loss of elimination of wastes of the kidneys in an adequate manner, which causes the accumulation of nitrogenous substances in the circulation. This condition is present in acute and chronic forms. The last stage renal disease (ESRD) is the terminus stage which requires renal replacement measures.

The purpose of the research was to examine how the erythropoietin and ghrelin hormones are expected to play and how the biochemical parameters play in clinical assessment of patients with renal failure .

Materials and Methods: The present study was undertaken using the sample of patients with renal failure undergoing dialysis at Baquba Teaching Hospital during the months of January to April 2022. The 62 and 35 were the patients who reported kidney failure and non-renal healthy controls, respectively. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) on serum samples were measured using the Cobas e411 analyzer. Nephric functional parameters (urea, uric acid and creatinine) were also measured on serum samples through the Cobas e411 analyzer. Ghrelin and erythropoietin hormone assays were done through the enzyme linked immunosorbent assay. The SPSS comprehension software was used to conduct the statistical analysis and data processing.

Results: Metabolic, hepatic and renal biomarkers, and hormonal values were also important ($P < 0.05$) between the patient group and the control group. The patients had high glucose, triglycerides, cholesterol, liver enzymes (ALT, AST and ALP) and kidney (urea, urate acid, creatinine and potassium) indicators. The patients experienced the reduction of albumin and erythropoietin. There was no significant difference between the total protein, sodium and calcium levels according to the group ($p > 0.05$).

Conclusions: We found that physiological parameters are connected with the presence of renal dysfunction to a large extent. The chronic renal failure affects to a considerable extent the biochemical indicators and blood regulators, that is, the level of erythropoietin and ghrelin. The results indicate a lack of electrolyte, metabolism, and hormone homeostasis, which implies that ghrelin and erythropoietin are the possible diagnostic and treatment etiological variables in the treatment of renal diseases.

Keywords: renal failure, erythropoietin, ghrelin, biochemical parameters.

1. Introduction

With the epidemiology of chronic kidney disease increasing across the world and prevalence of about 14 percent in the US alone, chronic kidney disease is becoming prominent. Past research has found out that an inverse relation exists between survival and kidney failure and greater possibilities of impaired cardiovascular results up to the end-stage renal disease (ESRD) [1]. It has made renal replacement treatment even more prevalent all around the globe that is bound to continue to grow even in the coming decade [2]. Hyperkalemia is one of the frequently occurring electrolytes disorders in chronic kidney disease. This is one of the extant effects between the two states profibrotic [3]. A number of cardiometabolic risk factors, excessive amounts of proinflammation, and inadequate magnesium and salt levels [4]. The risk factors that predispose development of cardiovascular disease entail elevated cholesterol levels of triglycerides, decreased levels of HDL cholesterol and total cholesterol (TC) and low-density lipoprotein (LDL) cholesterol [5]. Nevertheless, the dialysis or transplantation leading into specialization dyslipidemia may trigger the specialization process that is associated with massive quantities of qualitative and quantitative lipid disorders in CKD [6]. The most active effect of chronic renal disease is dyslipidemia which is connected with the lowered rate of glomerular filtration (GFR) [7].Among the adverse effects of lipoprotein metabolism imbalance, dyslipidemia is one of the most common effects of chronic renal disease. Pleiotropic biological activities of erythropoietin (EPO) have since been established since the discovery of EPO receptors on neurons, muscle cells, kidney cells, endotheliocytes and other cells [8]. The researchers showed that EPO was able to restore the functional activity and qualitative structuring of the peripheral blood leukocytes by decreasing mortality of lymphocytes by necrosis and apoptosis [9] in a model of chronic renal failure (CRF). Ghrelin is a peptide hormone that consists of 28 amino acids; the stomach, the hypothalamus, pituitary gland, as well as other peripheral organs, produces a minimal amount of this hormone [10]. In one of the previous studies, the renal failure subjects were observed to have elevated ghrelin levels compared to the controls [11]. Rashad et al. were able to notice that the malnourished patients with end-stage renal diseases and mild/moderate conditions showed significantly higher plasma ghrelin levels and expression of the gene compared to the severely malnourished ones [12].

Materials and Methods

The research was carried out in the dialysis unit of Baquba Teaching Hospital, and the research time is the period of the month of January until April 2022 on the target population of people with kidney failure. The research subjects consisted of 62 patients of renal failure (34 males and 28 females) with age (25-70 years) and 35 healthy controls of the same age group (17 males and 18 female).

Ten ml of the dialyzed venous blood plasma were transferred in test tubes and left to stay at 37 o C after half an hour and then centrifuged to get blood serum. The COPAS analyzer determined the biochemical parameters of serums including hepatic (ALT, AST, and ALP) and renal types (urea, uric acid and creatinine). The concentration of ghrelin and erythropoietin were detected by ELISA method using a commercial kit (DRE11406, Glory Science Co., Ltd., USA) and at 450nm of wavelength using Statfax reader (Awareness, USA). The SPSS software was used in carrying out the statistical analysis with mean and standard deviation as the descriptive statistics of the data. T-test was used to compare the means of the two groups of the study with statistical hypotheses of level of significance (= 5) (value-P < 0.05).

3. Results

The results showed that there was a significant difference between the groups of glucose, triglyceride, cholesterol and albumin ($p < 0.05$). The glucose, triglycerides and cholesterol levels in patients (149 ±9.7, 166.7 ±8.1 and 178.1 ±7.9) were higher than the level of healthy people (99.7 ±7.4, 6.3+136.2 and 143.1 ±8.4) respectively and the level of albumin was lower in patients (3.1 ±0.28) than in healthy people (4.57 ±0.39). Lastly, the study groups did not show significant differences ($p > 0.05$) in the content of total protein [Table 1].

Table 1. Comparison of metabolic parameters between renal failure patients and healthy controls

Parameters	Healthy	Patients

	Mean SD	Mean SD
glucose (mg/dl)	7.4±99.7	9.7±149*
Triglycerides (mg/dl)	6.3±136.2	8.1±166.7*
cholesterol (mg/dl)	8.4±143.1	7.9±178.1*
Albumin (g/dl)	0.39±4.57	0.28±3.1
Total Protein (G/Dl)	0.29±7.24	0.31±6.2

* signifies a statistically significant difference at the $p < 0.05$ level.

Statistical test showed that there were significant differences ($p < 0.05$) in the hepatic enzyme (ALT, AST, and ALP) levels in the experimental groups. The concentration of ALT, AST and ALP was elevated in patients (40.1 ± 4.8, 33.9 ± 2.9 26 and 368PLUS 41.3, respectively) when compared to healthy controls (20.1 ± 9.1, 22.9 PLUS 2.4 and 86.3PLUS 3.45, respectively).

Table 2. Comparison of liver function parameters between renal failure patients and healthy controls

Parameters	Healthy Mean SD	Patients Mean SD
ALT	20.1±9.1	40.1±4.8*
AST	22.9±2.4	33.9±2.9*
ALP	3.45±86.3	368±41.3*

*Indicates that there is a significant difference at the 0.05 level.

The findings of the study were that the levels of urea, uric acid, creatinine and potassium significantly differed ($p < 0.05$) between the study groups. Urea, uric acid, creatinine, and potassium levels of the patients (67.5 ± 8.9, 11.8 ± 3.1, 12.7 ± 3.9, and 7.9 ± 1.02, respectively) were higher than those of healthy people (19.1 ± 2.1, 3.9 ± 1.25,1.01 ± 0.59 and 3.01±139.8, respectively). Lastly, there were no significant differences ($p > 0.05$) in sodium and calcium content in the study groups [Table 3].

Table 3. Comparison of renal function and electrolyte parameters between renal failure patients and healthy controls

Parameters	Healthy	Patients
Urea (mg/dl)	2.1±19.1	8.9±67.5*
uric acid (mg/dl)	1.25±3.9	3.1±11.8*
creatinine (mg/dl)	0.59±1.01	3.9 ±12.7*
Sodium (mmol/l)	3.01±139.8	3.2±143.7*
Potassium (mmol/l)	0.36±4.2	1.02±7.9
Calcium (mmol/l)	9.7±0.4	8.1±0.6

* Indicates that there is a significant difference at the 0.05 level.

The findings indicated that there were significant disparities ($p < 0.05$) in the erythropoietin and ghrelin parameters between the study groups. The erythropoietin level was less in patients (1.78±0.56) as compared with healthy controls (6.45±2.18) and ghrelin was more in patients (1.23±0.59) compared with healthy controls (0.80±0.34).[(Table 4].

Table 4. Comparisons of the mean levels of erythropoietin and ghrelin between the study groups.

Parameters	Patients	Healthy	P value
Erythropoietin (mIU/mL)	1.78±0.56	6.45±2.18	P<0.001***
Ghrelin (ng/mL)	1.23±0.59	0.80±0.34	P<0.01**

* $p < 0.05$ considered statistically significant.

4. Discussion

The present paper has discussed the predictive and comparative impact of erythropoietin, ghrelin and various biochemical parameters among patients with renal failure compared to healthy people. It was established that significant biochemical and hormonal alterations occurred, which proved an overall physiological disturbance provided by kidney failure. The results of that provide a clue regarding the dysregulation of the metabolic, hepatic, and endocrine systems that are typical of chronic kidney disease (CKD) and end-stage renal disease (ESRD).

The high levels of glucose, triglycerides as well as total cholesterol that are recorded in patients indicate the metabolic effects of kidney dysfunction. Lipid metabolism is affected by impaired renal function reducing the activity of lipoprotein lipase, the clearance of triglyceride-rich lipoproteins, and atherogenic particles. The resultant abnormalities lead to the dyslipidemic profile that is normally linked to CKD as earlier explained by Vaziri [13] and Bulbul et al. [14]. The ongoing hypertriglyceridemia and hypercholesterolemia in ESRD enhances endothelial dysfunction, oxidative stress, and cardiovascular disease, which is still one of the primary causes of death in dialysis patients. Moreover, insulin insensitivity and reduced glucose metabolism in muscle tissue can be the reason behind the presented hyperglycemia in renal failure [15,16]. Collectively, these metabolic alterations indicate that renal failure alters carbohydrate and lipid homeostasis, making the patients susceptible to cardiovascular events and inadequate metabolic performance.

The significantly decreased serum albumin levels in the renal failure group is an indicator of protein-energy malnutrition and systemic inflammation which is an essential characteristic of advanced CKD. Hypoalbuminemia is also identified to predict either morbidity or mortality in dialysis patients, indicating the reduced production and also the loss of albumin in proteinuria or dialysis membranes [17,18]. According to Gatta et al., low serum albumin is also associated with enhanced oxidative stress, endothelial damage and reduced antioxidant capacity. The preservation of an optimal state of nutrition and monitoring of the albumin level are thus two important elements in the management of a renal patient [19,20].

The current findings also revealed significant variations in the activities of liver enzymes- ALT, AST and ALP in renal failure patients. The increase may be attributed to the fact that uremic toxins accumulate and the membranes of the hepatocyte are directly influenced by metabolic acidosis. Similarly, Sette and de Almeida Lopes [21] reported the altered liver enzyme activity on CKD patients and concluded that there was hepatic stress or secondary hepatocellular injury present. Raising of ALT and AST may also be the manifestation of insulin resistance, liver lipid deposition, whereas raising of ALP is typically associated with mineral bone disorder (CKD-MBD) and vascular calcification [22,23]. The general conclusion of these studies is that liver dysfunction is a prevalent comorbidity of renal diseases, and hepatic monitoring is to be performed regularly.

The elevated rate of urea and creatinine in the group of patients is an important sign of the impaired glomerular filtration and retention of nitrogen related to CKD. These are still simple parameters that indicate renal performance and disease severity [24,25]. These wastes of nitrogen caused systemic toxicity, inflammation and altered metabolic states as a result of accumulation. High potassium levels, as observed

in the given case, are an expression of the failure of electrolytes by the kidneys to be excreted normally that can lead to cardiac arrhythmias and neuromuscular disturbances in case of non-treatment. The relatively constant amounts of sodium and calcium, however, are indications of partial homeostasis of the two ions, but they are prone to alterations as the disease progresses [26,27]. The deviations of CKD are not only an indicator of the dysfunction of the kidneys but an influence on the cardiovascular, skeletal, and muscular systems, but that is why the disease is systemic as well.

Among the significant results of the present research was the significant decrease of erythropoietin (EPO) content in patients with renal failure as compared to controls. The synthesis of erythropoietin is mainly done by peritubular fibroblast-like cells located in the renal cortex and its deficiency plays a major role in the pathogenesis of anemia of chronic disease. Interstitial fibrosis, tubular atrophy and inflammatory remodeling of cells that produce erythropoietin hold back the expression of the erythropoietin gene leading to reduced erythropoiesis [28-31]. The results of the given study coincide with the findings of Panjeta et al. [28] and Fujita et al. [32], who showed that low EPO levels are associated with the rapid decrease in glomerular filtration rate and deterioration of the renal outcomes. In addition to its hematopoietic effect, erythropoietin has cytoprotective, anti-apoptotic, and anti-inflammatory effects in the renal tissue. They have demonstrated experimentally that EPO has the potential to suppress fibrosis, stimulate regeneration through a decrease in oxidative stress and expression of inflammatory cytokines [33-35]. Therefore, the EPO decrease in renal failure could be a factor in causing anemia and progressive renal injury, and early therapeutic interventions addressing EPO deficiency are required.

On the other hand, the study showed a considerable increase in the serum ghrelin levels of renal failure patients with regard to healthy controls. Ghrelin is a peptide hormone that is mostly produced in the stomach and controls the energy balance, appetite, as well as the glucose metabolism. Ghrelin metabolism during CKD is characterized by a significant change in the metabolism, as renal clearance reduces, leading to an increase in the circulating levels, especially the des-acylated form. The higher levels of ghrelin after the present study could be attributed to a compensatory mechanism due to negative energy balance, malnutrition, and systemic oxidative stress. Similarly, Canpolat et al. [36] discovered that protein-energy wasting is associated with the accumulation of ghrelin in CKD and anti-inflammatory, antioxidant properties of this hormone might serve as a protective action against the destruction of the kidneys. It has been established that experimental models have validated that ghrelin has renoprotective effects which consists of alleviating oxidative stress, mitochondrial dysfunction, and proinflammatory cytokine [37,38]. All these activities imply that high levels of ghrelin during renal failure might be one of the physiological responses to counteract catabolic stress response and maintain cellular homeostasis.

Interestingly, the antagonistic nature of the interaction between erythropoietin and ghrelin that is observed in the present study would also be indicative of antagonistic responses of the hormones to renal injury. Although EPO deficiency is one of the causes of anemia, hypoxia, and the onset of the disease, it is possible that increased ghrelin secretion is a form of adaptation reaction to antioxidative and anti-inflammatory responses. This endocrine take into consideration complicates the endocrine control in CKD and the observation confirms the hypothesis that the two hormones can be used as biomarkers to establish the severity and progression of the disease. It is possible to justify such patterns of hormones by a more personalised treatment (including erythropoiesis-stimulating agents in combination with metabolic modulators, e.g. ghrelin analogs**.**

By and large, the current research study can be used in the emerging literature on the fact that renal failure predestines a myriad of systemic biochemical and hormonal abnormalities that are not limited to renal filtration. Biochemical profile (urea, creatinine, albumin, and enzymes) and hormonal profile (erythropoietin and ghrelin) are used and assist in obtaining a more detailed picture of the pathophysiology of the disease. All these parameters can be the contributors to the enhancing of the diagnostic and assist in the observation of the therapeutic response of CKD and ESRD. The longitudinal relationship between the erythropoietin and ghrelin level and the possible prognostic and therapeutic significance of managing these hormones to enhance the process of metabolism and kidney functions should be addressed in future studies.

5. Conclusion

This discussion describes the rampant systemic manifestation of renal dysfunction, numerous systems, and metabolic pathways. This experiment shows the existence of high correlations between kidney disease and various physiological parameters particularly; hormone regulators such as erythropoietin and ghrelin. A clinical evidence suggests the presence of

considerable alteration in the activity of the key organs, in particular, of the hepatic and pancreatic system, as well as, of metabolic disturbances, including dyslipidemia. Certain tendencies of electrolyte disturbances, in the form of hyperkalemia and hypotonatremia, and the occurrence of anemia were shown in the paper. The analysis of hormonal profile showed that the changes in the homeostatic processes were great and the level of the key regulatory hormones was abnormal. The findings show that ghrelin and erythropoietin can serve as a diagnostic biomarker and even as a treatment intervention of renal disease. The interdisciplinary nature of treatment in the paper brings out the need to concentrate on a combined mode of treatment whereby the root cause of renal dysfunction and all the systemic outcomes are managed. It is expected that this knowledge should be utilized to create more specific and effective treatment plans that could lead to improved patient outcomes during the treatment of renal diseases.

Ethical Considerations

Compliance with ethical guidelines

Not applicable

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Author's contributions

All authors contributed equally to the development of this article.

Conflict of interest

The authors declare no conflict of interest.

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