



Estimation of the Level of Oxidative Factors and Some Biochemical Parameters that Affect Renal Disease

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Abstract

The kidneys are crucial organs with a bean-like form that can be found on either side of the spine and filter toxins and excess water from the blood. Similar to other organs in the body, the kidneys are susceptible to injury due to a variety of conditions. The current study included a hundred individuals on dialysis who were suffering from renal failure. Their ages ranged from 16 to 70 years old. These patients were enrolled in Al-Sadder Medical City in the governorate of Al-Najaf Al-Ashraf. The same precautions were also taken on the healthy group, which consisted of fifty samples whose ages were roughly equal to those of the dialysis patients. These healthy samples were kept in test tubes designated for this purpose and were kept at the proper temperature until the required number of samples was completed. According to the current study's outcomes, dialysis patients had significantly higher levels of oxidative factors, urea, creatinine, and glucose than did the control group. Creatinine, Urea, Uric Acid, Fe, and Ferritin levels were higher in patients than in the control group, but D3 and HGB levels were significantly lower. Due to patients' impaired kidney function, the immune response is to blame for this. Regarding GSH, there is no appreciable distinction between the sick group and the healthy group. The patients' participation in some treatments is said to be the cause.

Introduction

Renal disease, also known as kidney disease or renal failure, denotes to the impairment or damage to the kidneys' function. The filtration of wastes, excess fluids, and toxins from the blood, management of electrolyte balance, and generation of hormones that regulate blood pressure and promote red blood cell development are all crucial functions of the kidneys (Gounden et al., 2018; Temirova & Asadova, 2025; Nadagouda, 2025; Ali & Alaubydi, 2025). There are several types and causes of renal disease, (Chronic Kidney Disease CKD) this situation progresses over time and is usually caused by conditions like high blood pressure, diabetes, or other underlying health issues that affect the kidneys' function (Webster et al., 2017). Acute Kidney Injury (AKI) occurs suddenly and is often caused by severe infections, dehydration, injury, or certain medications. It is typically reversible if the underlying cause is treated promptly (Kellum et al., 2021). A genetic condition known as polycystic kidney disease (PKD) is marked by the development of multiple kidney cysts. Over time, these cysts can cause kidney damage and impede function (Bergmann et al., 2018). Glomerulonephritis is the medical term for the inflammation of the glomeruli, which are the small kidney structures in charge of filtering waste and extra fluid. It may result from autoimmune illnesses, infections, or other conditions (Test, 2022; Casuscelli et al., 2023; Jasim et al., 2024). The symptoms of renal disease might vary depending on the underlying cause and the stage of the disease. Kidney stones are hard deposits that form in the kidneys and can cause excruciating pain and perhaps restrict the flow of urine. Fatigue, difficulty concentrating, decreased appetite, swelling in the

legs and ankles, changes in urine production or appearance, and high blood pressure are just a few of the typical symptoms (Shaker et al., 2022). Renal disease is normally diagnosed using a combination of physical examination, review of the patient's medical history, blood and urine tests, imaging procedures (such as an ultrasound or CT scan), and occasionally a kidney biopsy. The underlying cause and the illness stage affect the types of treatments available for renal disease. The course of kidney disease can sometimes be slowed down by making lifestyle adjustments, taking medications, and controlling underlying disorders like diabetes or high blood pressure. It's crucial for people with renal disease to collaborate closely with medical professionals, including nephrologists (kidney specialists), to manage their condition effectively and maintain their general health. Renal disease can progress to the point where dialysis or kidney transplantation may be required (Cheung et al., 2021; Nishio Lucar et al., 2024; Hernández & Caballero, 2023; Zitzmann, 2024). (AKI) and (CKD) are two distinct conditions that affect the kidneys, but they differ in terms of their onset, duration, and underlying causes. Here are the primary differences between AKI and CKD, AKI develops suddenly and is characterized by an express drop in kidney function over a short period, usually within hours to days. AKI can be reversible if the underlying cause is identified and treated promptly (Roberts et al., 2018). CKD develops gradually over months or years. It is a chronic circumstance that entails a gradual decline in kidney function, and may eventually lead to kidney failure. CKD is typically irreversible, but its progression can be slowed down through appropriate management (Li et al., 2019; Pethő et al., 2024; Romagnani et al., 2025; Maringhini & Zoccali, 2024). AKI is commonly caused by factors such as severe dehydration, blood loss, infections, medication toxicity (e.g., certain antibiotics or nonsteroidal anti-inflammatory drugs), blockages in the urinary tract, or underlying conditions that affect kidney function, such as heart failure or liver disease. CKD is frequently brought on by underlying medical disorders that, over time, steadily harm the kidneys. Diabetes, hypertension, glomerulonephritis, PKD, and several autoimmune illnesses are the most typical causes (Centers for Disease Control and Prevention, 2019; Ahmed et al., 2025; Casuscelli et al., 2023). AKI has the potential to be reversible if the underlying cause is promptly addressed and the kidneys are given an opportunity to heal. However, if AKI is severe or left untreated, eventually, CKD may develop. which is generally considered to be irreversible. While the progression of CKD can be slowed down and managed with appropriate treatment, the damage to the kidneys is usually permanent. At advanced stages, CKD may require dialysis or kidney transplantation (Kellum et al., 2021). there can be some overlap between AKI and CKD. Severe or recurrent episodes of AKI can contribute to the development or progression of CKD, and individuals with CKD may be at a higher risk of experiencing episodes of AKI, both AKI and CKD require medical attention and management by healthcare professionals, particularly nephrologists (kidney specialists), to optimize kidney function and prevent complications, Glutathione is a powerful antioxidant found in almost every cell of the body and plays a crucial role in maintaining cellular health and protecting against oxidative stress, in the context of renal failure, glutathione has been a subject of interest and research due to its potential implications for kidney health. However, it's important to note that the relationship between glutathione and renal failure is complex and multifaceted (Ighodaro & Akinloye, 2018; Mazari et al., 2023; Feng et al., 2023).

It's important to note that glutathione metabolism is complex, and its effects on renal failure may be influenced by various factors, including the underlying cause of renal failure, disease stage, individual variations, and other comorbidities (Ighodaro & Akinloye, 2018). important to note that the management of vitamin D deficiency in renal failure requires careful monitoring and individualized treatment plans. The dosage and type of vitamin D supplementation may vary based on the patient's specific condition and stage of renal failure. Nephrologists or

healthcare professionals familiar with the patient's medical history are best equipped to provide appropriate guidance regarding vitamin D supplementation in renal failure (MacLaughlin et al., 2022).

Urea and creatinine are two important markers used to assess kidney function and diagnose renal failure. Elevated blood urea levels, known as blood urea nitrogen (BUN), are commonly used as an indicator of renal function (Al Jameil, 2019; Zhang et al., 2022; Macedo, 2011). Higher BUN levels indicate reduced kidney function or impaired clearance of urea, which can be seen in acute or chronic renal failure. Like urea, creatinine is filtered by the kidneys and excreted in urine. Creatinine levels in the blood are also used as an important marker of kidney function. In renal failure, the kidneys are unable to effectively filter and excrete creatinine, resulting in elevated blood levels. Serum creatinine levels are closely monitored to assess the severity of renal failure and to evaluate the progression of kidney disease (Temilola et al., 2019; Spanaus et al., 2010; Baumgarten & Gehr, 2011).

Red blood cells include a protein called hemoglobin, which transports oxygen from the lungs to the tissues and removes carbon dioxide from the tissues. Renal failure frequently leads to anemia, which is indicated by a reduction in hemoglobin levels or the quantity of red blood cells (Badura et al., 2024; Santos et al., 2025). Erythropoietin, a hormone that stimulates the synthesis of red blood cells in the bone marrow, may not be produced by the kidneys in sufficient amounts in cases of renal failure. Additionally, impaired renal function can result in the buildup of uremic toxins, which can harm red blood cell survival and formation. Renal anemia or anemia of chronic kidney disease (CKD) are terms frequently used to describe anemia in renal failure. Erythropoietin, which is predominantly made by the kidneys, is essential for controlling the synthesis of red blood cells. Reduced kidney function in renal failure can cause insufficient EPO production, which in turn results in less stimulation of the bone marrow to create red blood cells. Lower Hb levels result from this, which helps anemia develop (Li et al., 2019).

Methods

One hundred dialysis patients with renal failure were enrolled in the current study. They were between the ages of 16 and 70. These patients were registered in Al-Sadder Medical City in the Al-Najaf Al-Ashraf governorate. Fifty people participated as a control group their ages asymptotic to the patients. A serum tube was filled with around 3.5 mL. After being allowed to clot for 10 minutes at room temperature, the serum was separated with a centrifuge at 3000 Xg for 10 minutes, and then the sample was put into brand-new disposable tubes and frozen at (-70 C) until it could be analyzed, At this investigation, in addition, the following kits were utilized in this investigation kit-Ichroma II Kit for Vitamin D in addition to S Creatinine, B. Urea, Uric Acid which is determined by using a spectrophotometer

Statistical analysis

Following biochemical examination, data were compiled and statistical calculations were performed on them using statistical software (Megastat). It was possible to obtain the mean, standard deviation of the mean, and F-distribution test. We utilized Microsoft Excel (2016), SPSS 23, and Minitab v. 14 to analyze the data. The critical value or test of probability less than 0.05 (p 0.05) was considered significant in addition, and if the probability less than 0.001 (p 0.001) was regarded as extremely significant as well Al-Zurfi et al. (2016) and Auda et al. (2021).

Results and Discussion

According to the results of the current study, D3 and HGB significantly decreased (P value ≤ 0.001), whereas all other parameters creatinine, urea, uric acid, iron, and ferritin significantly increased (P value ≤ 0.001). however, the same outcome in Table (1) demonstrates that there has been no discernible change in the amount of GSH in patients with renal disease as compared to control groups.

Table 1. result of limits for patients with renal disease and their control

Parameters	Patients		Control		T Test
	Mean	SD	Mean	SD	
S Creatinine mg/dl	8.9	3.21	1.35	0.42	0.001
B. Urea mg/dl	165.66	58.03	34.05	19.35	0.001
Uric Acid mg/dl	11.9	5.61	6.93	1.31	0.001
D3 ng/ml	14.67	6.05	33.41	19.74	0.001
GSH mmol/l	5.55	1.42	5.42	0.66	0.556
Fe μ g/dl	80.83	36.69	47.72	19.42	0.001
Ferritin ng/ml	475.55	169.79	260.95	95.8	0.001
HGB g/dl	10.17	2.24	13.36	0.82	0.001

Renal disease can have significant effects on various markers in the body, including urea, creatinine, uric acid, and hemoglobin (Hb).

Healthy kidneys filter urea out of the bloodstream and excrete it in the urine. In renal disease, the kidney's ability to filter urea decreases, leading to high levels of urea in the blood, a state known as uremia or azotemia. Elevated blood urea levels are commonly seen in renal dysfunction in renal disease, the kidneys' filtration function is impaired, resulting in decreased clearance of creatinine. Consequently, the blood levels of creatinine rise. Elevated creatinine levels are an important marker of impaired kidney function and are commonly used to assess renal disease severity.

the kidneys may struggle to excrete uric acid efficiently, leading to increased levels in the blood. Elevated levels of uric acid can contribute to the development of conditions like gout and kidney stones (Auda et al., 2021).

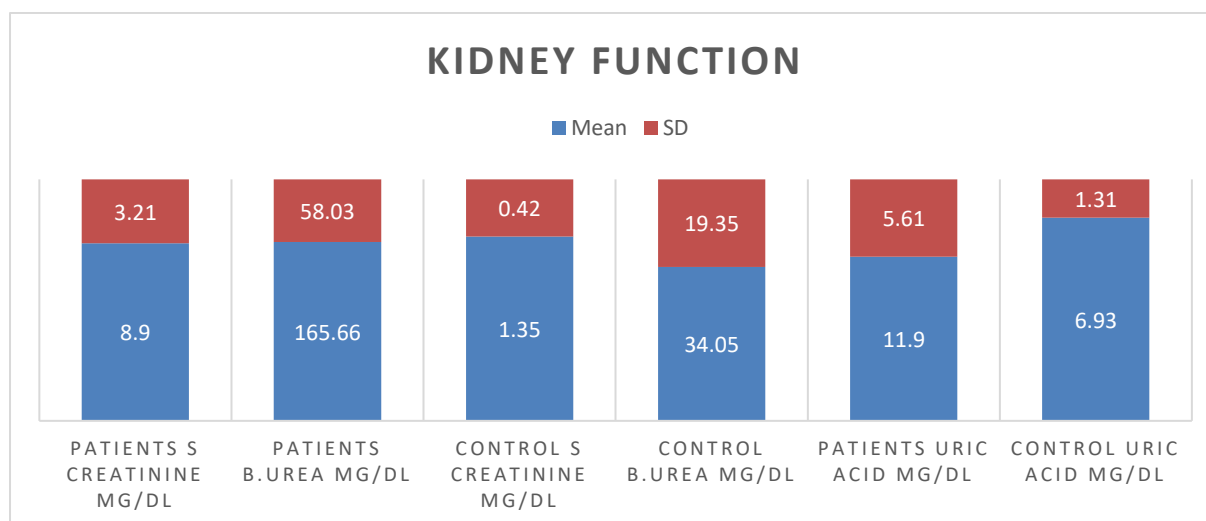


Figure 1. shows the kidney function in patients with renal failure

The protein called hemoglobin is in charge of transporting oxygen in red blood cells. A hormone required for the generation of red blood cells, erythropoietin, may be less produced by the kidneys in late renal failure. Consequently, the reduced production of red blood cells can lead to anemia, characterized by decreased hemoglobin levels.

Renal disease can have an impact on iron levels and ferritin, which is a protein that stores iron in the body the kidneys play a crucial role in regulating iron homeostasis by producing and releasing a hormone called hepcidin which helps control the absorption, release, and distribution of iron in the body. In (CKD), there is often a decrease in hepcidin production, leading to alterations in iron metabolism. Some forms of renal disease, particularly those associated with inflammation or chronic blood loss in the gastrointestinal tract or dialysis, can lead to iron deficiency. The kidneys may have a reduced capacity to reabsorb iron, leading to increased iron loss in the urine. Additionally, decreased production of erythropoietin by the kidneys can result in impaired red blood cell production, contributing to iron deficiency anemia (Auda et al., 2021).

On the other hand, individuals with advanced stages of CKD who are receiving regular blood transfusions or undergoing frequent hemodialysis may accumulate excess iron in their bodies. This occurs due to repeated transfusions or the breakdown of red blood cells during dialysis. Iron overload can lead to complications such as organ damage, particularly in the liver, heart, and endocrine system.

ferritin levels can be influenced by multiple factors. In cases of iron deficiency, ferritin levels may be low, reflecting depleted iron stores. However, in situations of iron overload, ferritin levels can be elevated (Ali et al., 2021).

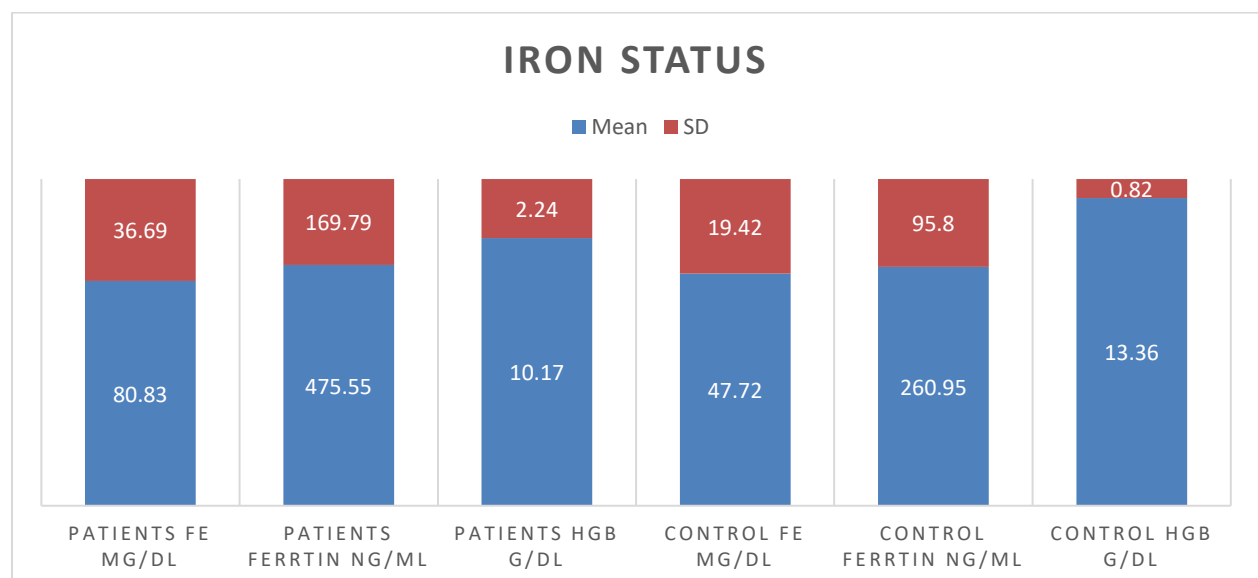


Figure 2. refers to the level of Iron Status in renal failure patients

Renal disease can have an impact on iron and ferritin levels, as well as vitamin D3 (cholecalciferol) metabolism, the kidneys converting inactive vitamin D (cholecalciferol) into its active form, known as calcitriol or vitamin D3. In renal disease, the kidneys may not be able to convert vitamin D effectively, leading to reduced levels of active vitamin D in the body.

The kidneys also play a role in controlling the body's calcium and phosphorus levels, which is crucial for preserving bone health. Active V.D. helps in the absorption of calcium from the intestines and promotes its reabsorption in the kidneys. In renal disease, impaired vitamin D

activation can disrupt calcium and phosphorus metabolism, leading to abnormalities such as hyperparathyroidism and renal Osteodystrophy. iron, ferritin, and vitamin D3 can vary depending on the stage and type of renal disease. additionally, individual variations, comorbidities, and treatments can also influence these parameters. Regular monitoring and consultation with a healthcare professional are vital for managing renal disease and interpreting these laboratory results accurately (Ali et al., 2021). Renal disease can affect the levels of glutathione (GSH) in the body. Strong antioxidant glutathione is essential for preventing oxidative stress in cells and preserving cellular health. Increased oxidative stress is related to chronic kidney disease (CKD). When there is an imbalance between the body's capacity to neutralize and eliminate damaging reactive oxygen species (ROS) and their rate of generation, oxidative stress is brought on. ROS can be produced in renal disease as a result of inflammation, ischemia-reperfusion damage, and the buildup of toxic waste products. As a result, glutathione and other antioxidants are increasingly needed to prevent oxidative damage (Ali et al., 2020). In the setting of renal disease, the increased oxidative stress can lead to the depletion of glutathione. The excessive production and accumulation of ROS can overwhelm the body's antioxidant defense systems, including glutathione. Additionally, impaired kidney function can affect the synthesis and recycling of glutathione, further contributing to its depletion.

Renal disease can compromise the overall antioxidant capacity of the body. Glutathione works in conjunction with other antioxidants to neutralize ROS and protect against oxidative damage. However, in renal disease, the balance between antioxidants and pro-oxidants may be disrupted, leading to reduced antioxidant capacity. This imbalance can further perpetuate oxidative stress and contribute to the progression of renal damage (Ali et al., 2020).

It's vital to annotation that the specific effects on GSH levels in renal disease can vary depending on various factors such as disease severity, underlying causes, and individual variations. Management of renal disease often involves strategies to mitigate oxidative stress and support antioxidant defenses. This may include dietary interventions, antioxidant supplementation, and targeted therapies aimed at preserving GSH levels and reducing oxidative damage. Consulting with a healthcare professional knowledgeable in renal disease management is essential for personalized guidance and treatment (Ali et al., 2020).

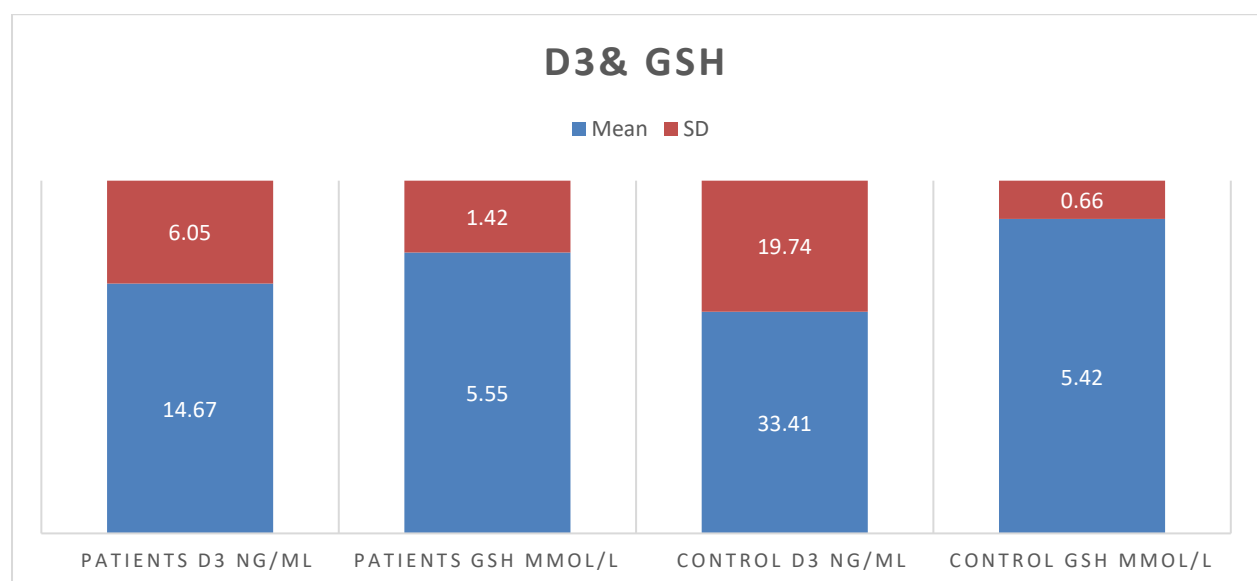


Figure 3. refers to the level of D3 & GSH in renal failure patients

Conclusion

A significant increase in the level of Creatinine, Urea, Uric Acid, Fe, and Ferritin in patients when compared with their results with the control group, with a significant decrease in D3 and HGB the reason for this is due to the immune response, in Patients due to the insufficiency of the kidneys. As for GSH, there is no significant difference in patients compared to the healthy group. The reason is attributed to the patients taking some treatments.

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