World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

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Pathophysiology analysis of patients with chronic kidney disease after dialyzes

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World Journal of Advanced Research and Reviews, 2025, 27(02), 501-507

Publication history: Received on 25 June 2025; revised on 02 August 2025; accepted on 05 August 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.27.2.2763

Abstract

This study aimed to assess various blood components and electrolyte levels in patients with chronic kidney disease (CKD). Hematopathological parameters, including renal and liver function tests, electrolytes, calcium, phosphate, and vitamin D3, were measured and compared with those in healthy controls. A hospital-based comparative study was conducted with 40 patients diagnosed with (CKD), who exhibited anemia, liver abnormalities, and abnormal electrolyte and vitamin D3 levels. These patients were compared to 20 healthy control subjects without (CKD). Key parameters such as hemoglobin, serum iron, renal and liver function, calcium, phosphate, and vitamin D3 were analyzed across both groups. Hemoglobin levels, Total Iron Binding Capacity (TIBC), and serum iron were significantly lower in (CKD) patients compared to healthy controls ($p < 0.05$). Patients also had elevated levels of liver function markers (GOT, GPT, and ALP) and renal function markers (urea and creatinine) relative to healthy controls ($p < 0.05$). Additionally, calcium and vitamin D3 levels were lower, while phosphate levels were higher in patients than in controls ($p < 0.05$).Conclusions Patients exhibited significant anemia, notably lower hemoglobin levels, serum iron, calcium, and vitamin D3 levels. In contrast, levels of urea, creatinine, and phosphate (ALP, GOT, and GPT) were significantly elevated. This indicates that (CKD) is associated with distinct hematological and biochemical imbalances.

Keywords: CKD; Anemia; Hemoglobin; Urea; Creatinine

1. Introduction

According to the World Health Organization (WHO), anemia is defined as a hemoglobin concentration below 13 g/dL for adult males and below 12 g/dL for adult females (1). Chronic kidney disease (CKD) frequently presents with iron-deficiency anemia (2). Several factors contribute to anemia in this population, including relative erythropoietin deficiency, both absolute and functional iron deficiency, impaired hepcidin clearance, shortened erythrocyte lifespan, and nutritional deficiencies (such as folic acid and vitamin B12). Patients with CKD stage G5 on hemodialysis (HD) experience additional iron loss, up to 3 grams per year, due to chronic bleeding, recurrent phlebotomy, and blood loss during dialysis and through access lines (3). as well as accelerated development of end-stage renal disease (ESRD) caused by anemia (4). When the kidneys fail to function, the production of erythropoietin (EPO) is significantly reduced. This, in turn, reduces the number of red blood cells produced by the bone marrow, leading to anemia. Consequently, the blood, with its reduced red blood cell count, deprives the body of oxygen (5).

In healthy individuals, the senescent erythrocytes are recycled iron from them and carried into the bone marrow via the reticuloendothelial system, which is integrated into erythroblasts (6). Recompensed daily loss is maintained. Normal iron homeostasis occurs through dietary iron absorption by the duodenum (7). Anemia is due to increased blood losses, mainly in dialysis-dependent patients (8).

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Markers of liver cell damage, such as serum levels of the liver enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are indicative of hepatocyte injury (9). These enzymes are elevated in various conditions, such as chronic viral hepatitis (10), non-alcoholic fatty liver disease (11), autoimmune hepatitis (12), hemochromatosis 13, and alcoholic liver disease (13). They reflect inflammatory activity in the liver parenchyma, serving as a basis for diagnosis, patient monitoring, and assessing response to treatment by these enzymes [(14),(15),(16)]. Interestingly, some research has shown that patients with chronic kidney disease (CKD) on hemodialysis (HD) may have lower serum levels of liver enzymes compared to those with normal renal function, though the underlying causes remain unclear (17),(18).

As well liver dysfunction and kidney failure associated with plasma ALP levels can start in the liver, bone, intestine, and placenta. Most of the circulating enzyme levels are commonly contributed by the isoenzymes from the liver and bone. However, a significant increase in the bone isoenzyme of ALP produced from renal osteodystrophy in a chronic kidney disease patient results in a high serum ALP level. In truth, increased mortality in pediatric chronic kidney disease patients, as well as patients on maintenance hemodialysis, is associated with higher ALP(19).

Because ALP is related to liver problems, it is also related to bone problems. Thus, we will discuss some minerals that are linked to bone disorders in chronic kidney disease. In aging, atherosclerosis, hypertension, and diabetes, increased vessel stiffening, and an increased danger of myocardial infarction lead to prevalent vascular calcification. Its most damaging effects happen in patients with chronic kidney disease (CKD), who are seen as a greatly raised threat of cardiovascular mortality when compared with age-matched controls. CKD patients have accelerated calcification, and this calcification in patients on dialysis more quickly (20). In people with chronic kidney disease who have bone disease, the chance of losing kidney function increases. In infamous cases, phosphorus homeostasis is contributed to by bone (21).

Beginning in the early stages of CKD, the action causes unregulated mineral metabolism, bone disease, and progressive loss of kidney function, which continues throughout this course. The numerous therapeutic approaches just now used may be influenced beneficially or adversely. So, improving CKD patients' quality of life and longevity is most important; therefore, barring disturbances in mineral and bone metabolism and their management early in chronic kidney disease (22).

In patients living in northern communities, limited daylight exposure and poor vitamin D intake lead to a high risk of vitamin D insufficiency (23). There is proof that vitamin D (vit D) insufficiency is an independent risk factor for diabetes in adults, and this disease is also related to kidney failure (24). As a result, when kidney problems occur, the level of D3 decreases.

2. Material and Methods

2.1. The research was started with permission from the Institutional Ethics Committee.

Blood and serum samples were also collected from people (male and female) with chronic kidney disease. To control the group of healthy people who attended the dialysis center. A total of 60 cases were included in the study, The control group was obtained from 20 samples, and 40 were patients. Following the iron state (hemoglobin and iron) were measured by a hematology analyzer (CBC), kidney markers (urea and creatinine) and liver markers (GOT and GPT, ALT), as well as calcium and phosphor, were measured by using a spectrophotometer. Elisa Kit measured D3. Total iron binding capacity (TIBC), serum iron, and serum ferritin were measured in the biochemistry lab using a specific kit for each one.

2.2. Statistical analysis

Statistical analyses of all results were done with the help of GraphPad Prism Version 5 software statistical package using a t-test (with a p-value at a level of significance less than 0.05) to compare the values of the results between groups. Result values were indicated as mean ± SE, number of patients, or percentage.

3. Results

Our current study includes 60 cases, 40 of whom have chronic kidney disease (CKD) and are undergoing continuous dialysis. At the same time, the remaining 20 are healthy and serve as the control group.

We observed significant differences in mean hemoglobin levels ($p<0.05$) between patients with CKD (9.278 ± 0.2132) and the control group (13.36 ± 0.1636), as shown in Figure 1.

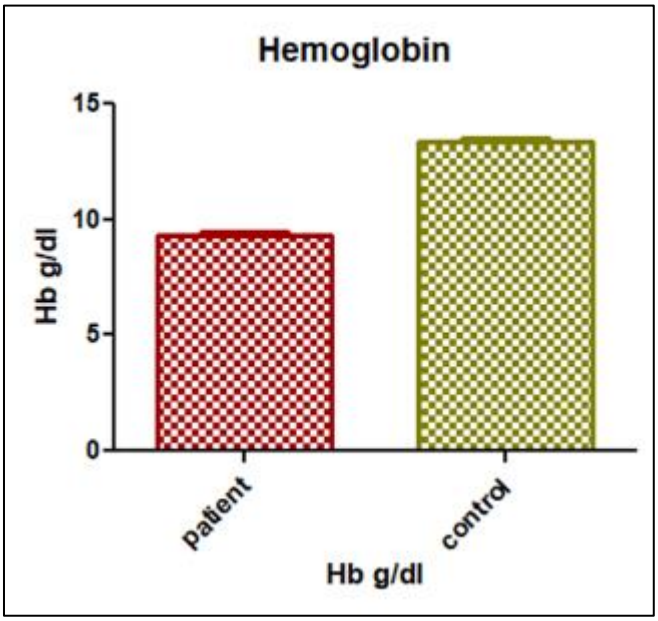

Figure 1 The hemoglobin level in CKD patients compared to healthy individuals

Additionally, the results indicated a significant decrease in iron levels ($p<0.05$) in CKD patients (71.52 ± 17.89) compared to the control group (161.4 ± 1.814), as shown in Figure 2.

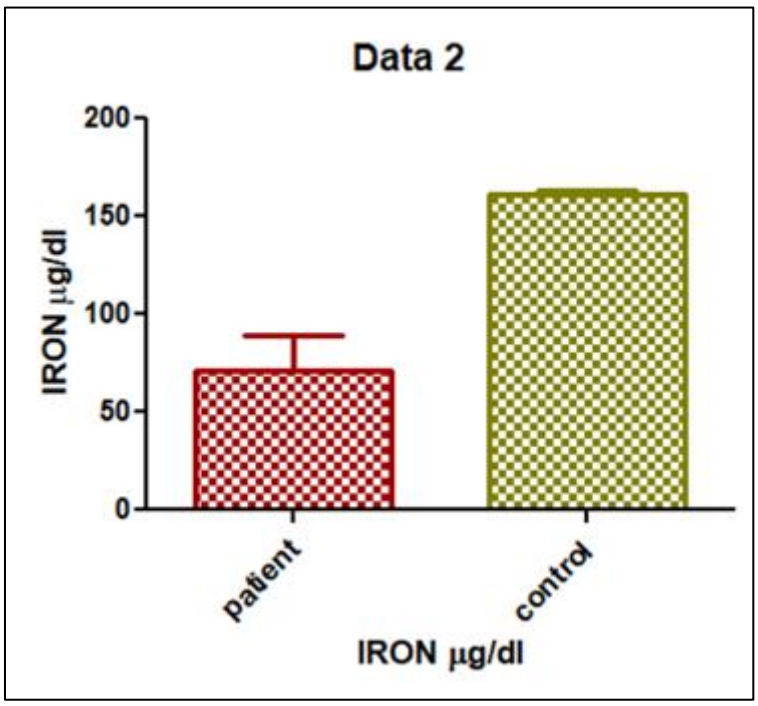

Figure 2 The iron levels in CKD patients compared to healthy individuals

Significant mean differences were showed in several hematological and biochemical markers, such as creatinine, urea, calcium, phosphorus, vitamin D3, ALP, UIBC, and TIBC. In addition, there was a slight but significant increase in GOT and GPT levels as shown in Table 1.

The result showed a significant increase ($p<0.05$) in UIBC in patients with CKD (541.6 ± 14.39) when compared with the control group (121.7 ± 10.28). At the same time showed a significant increase ($p<0.05$) in TIBC in patients with CKD (701.3 ± 53.45) when compared with the control group (302.3 ± 16.21).

In the present series Table 1 , patients with CKD showed a significant increase in ALP levels (295.4 ± 28.18) compared to the control group (161.7 ± 8.905), with ($p<0.05$).

The results showed a significant decrease in calcium levels in patients with CKD (7.93 ± 0.29) compared to the control group (11.30 ± 0.33), with ($p<0.05$). In this series, phosphorus in patients with chronic kidney disease (CKD) increased significantly ($p<0.05$) to 6.714 ± 0.3363, compared to 4.050 ± 0.1235 in the control group.

The present study demonstrated a significant decrease in vitamin D3 levels in patients with CKD (14.72 ± 1.49) compared to the control group (37.10 ± 1.07), with ($p<0.05$).

Urea level was present in 40 patients with CKD, a significant increase ($p<0.05$) (167.6 ± 5.736) compared with the control group (26.90 ± 1.709).

The creatinine levels in CKD patients were found to be significantly higher ($p<0.05$) at 9.371 ± 0.3354 than in the control group (0.7511 ± 0.05337).

The result showed a significant slight increase ($p<0.05$) in GOT level with CKD patients (42 ± 2.104) when compared with the control group (19.27 ± 1.9543). Similarly, GPT levels showed in CKD were noted to have statistical significance ($p<0.05$). a (40.00 ± 3.015) when compared with the control group (21.39 ± 2.518).

Table 1 Descriptive statistics of various hematological and biochemical parameters in CKD patients.

Dependent variable	**Group**	**Nember**	**Mean**	**Stander error**	**P.value**
hemoglobin	Patient Control	40 20	9.278 13.36	± 0.2132 ± 0.1636	0.05
Iron	Patient Control	40 20	71.52 161.4	± 17.89 ± 1.814	0.05
UIBC	Patient Control	40 20	541.6 121.7	± 14.39 ± 10.28	0.05
TIBC	Patient Control	40 20	701.3 302.3	± 53.45 ± 16.21	0.05
GOT	Patient Control	40 20	42 19.27	± 2.104 ± 1.9543	0.05
GPT	Patient Control	40 20	40.00 21.39	± 3.015 ± 2.518	0.05
ALP	Patient Control	40 20	295.4 161.7	± 28.18 ± 8.905	0.05
calcium	Patient Control	40 20	7.934 11.30	± 0.2944 ± 0.3253	0.05
phosphor	Patient Control	40 20	6.714 4.050	± 0.3363 ± 0.1235	0.05
D3	Patient Control	40 20	14.72 37.10	± 1.488 ± 1.068	0.05
urea	Patient	40	167.6	± 5.736	0.05

	Control	20	26.90	± 1.709	
creatinine	Patient Control	40 20	9.371 0.7511	± 0.3354 ± 0.05337	0.05

4. Discussion

Chronic kidney disease (CKD) is commonly complicated by anemia, which is associated with increased mortality and a reduced health-related quality of life.A decrease in hemoglobin means anemia is characterized by the body's use of iron-rich proteins for oxygen transport. Iron is wanted to produce hemoglobin, and iron deficiency Anemia happens through disruptions in iron homeostasis(25). This happens when patients lower their protein intake because of declining kidney function. Iron stores deplete when decreasing protein (e.g., meat) intake assists to reduced iron intake. Also, there may be a decrease in iron absorption from the gastrointestinal tract. so, inadequate total body iron stores result from multiple factors in patients with chronic renal failure, known as absolute iron deficiency (26).

Serum iron, transferrin (Tf), total iron-binding capacity (TIBC, calculated as Tf × 1389), transferrin saturation (TSAT, calculated as serum iron/total iron-binding capacity × 100), and serum ferritin are traditionally used to assess iron status and detect iron deficiency anemia (IDA) (27).

The mechanisms of anemia in (CKD) are multifactorial. The primary factor is the progressive reduction of endogenous erythropoietin (EPO) levels. Other contributing factors include absolute iron deficiency due to blood loss or impaired iron absorption, systemic inflammation caused by CKD and related comorbidities, a diminished bone marrow response to EPO due to uremic toxins, impaired utilization of iron stores caused by elevated hepcidin levels, reduced red blood cell lifespan, and deficiencies in vitamin B12 or folic acid. Together, these factors contribute to anemia in CKD patients (28)

In patients with CKD, we presented a decrease in iron status in our study, which was accepted by other studies.

Chronic liver disease is linked with kidney disease and has a remarkable impact on survival. Injury relies on the measurement of the concentration of serum creatinine, the evaluation of kidney function, which is affected by the level of liver disease, and the analytical method working (29)

Patients with acute liver failure (ALF) with or without chronic liver disease (CLD) are often current with linked AKI resulting from pathogenic processes different from those of HRS-1. The term HRS-2 has been used to mention hepatorenal physiology-mediated kidney dysfunction that is additional stealthy, chronic, or persistent. The ALT serum levels were higher in patients with CKD undergoing peritoneal dialysis than those on hemodialysis (30).

In patients with CKD, we showed a little increase in liver function tests; other studies accept this. Increased danger of end-stage renal disease was associated with higher levels of serum phosphorus in our great, ethnically various inhabitants of a topic who had kidney function (30).

The requirement for a stable state makes it difficult to perform formal balance studies in dialysis patients, as dialysis prevents a true steady state from being achieved for calcium. This disrupts calcium balance continually and may lead to fluctuations in soft tissue and bone mineral content. Another key principle in calcium balance studies is the need for strictly controlled dietary intake and highly accurate measurements of all inputs and outputs (31)

In our study, there was a decrease in calcium and phosphorus levels in patients with CKD because the irregularity in kidney function led to an abnormal mineral level.

5. Conclusion

This study demonstrated significant changes in hematological and biochemical markers among CKD patients on dialysis, including anemia, altered iron status, and disturbances in mineral and liver function. These findings underscore the need for regular monitoring to improve patient care and outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

All authors declare that they have no conflict of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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