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Discrepancy of hemodialysis patients in some electrolyte in Babylon province

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Abstract

Background: The current study deals chronic kidney disease patients suffering hemodialysis (HD) exhibit significant alterations in hematological and biochemical. checking these alterations is a proven necessity for tracking disease headway and improving patient persistence.

Methods: We linked 40 patients with 40 strong controls at Imam Al-Sadiiq hospital in Babylon. Blood samples were analyzed for Complete Blood Count, renal signs

Results: hemodialysis patients showed a dramatic rise in urea and creatinine, equaled to controls. Moreover, potassium and phosphorus surged, while hemoglobin and platelet counts dropped significantly.

Conclusions: Hemodialysis primes to systemic metabolic and hematological variability that wants constant medical surveillance. Regular lab monitoring is essential to prevent fatal complications and guide handling.

Keywords: HD; CKD; Hematological Changes; Liver Enzymes; Biochemical Markers

1. Introduction

Chronic kidney illness denotes a multi-systemic extreme condition that leads to reformist structural and handy renal damage, substantially elevating the risk of cardiovascular morbidity, mortality, consensus expresses CKD as persistent abnormalities in renal architecture or function that endure for a duration above three months [1].

The expansion of CKD-associated with anemia contains several physiological pathways, primarily characterized by a profound deficiency, disrupted iron homeostasis, determined systemic tenderness, and broader metabolic derangements [2].

Hemodialysis involved metabolic alterations, such as hyperkalemia, hyperphosphatemia, hypocalcemia, and altered liver enzyme activity, and potential viral hepatitis exposure. Accordingly, vascular disease relics the primary peril to life within these tolerant residents, office for nearly half of all documented deaths mid those with end-stage renal disease [3].

In clinical watching, pre-dialysis serum stages of urea and creatinine serve as the foundational biochemical bounds employed to gathering of nitrogenous, and phosphorus guideline persist typical features of CKD-mineral and bone disorder (CKD-MBD), actively driving secondary hyperparathyroidism and systemic vascular calcification [4].

Hematologic ally, patients undergoing long-term hemodialysis are also highly susceptible to thrombocytopenia and platelet dysfunction, this is mediated both by circulating uremic toxins that compromise platelet adhesion and

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aggregation, and by the physical trauma of the extracorporeal circuit, which induces platelet activation and subsequent consumption [5].

Inflammatory state is characterized by elevated circulating cytokines, particularly interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which worsen endothelial dysfunction and impair effective erythropoiesis [6]. Such inflammatory mediators simultaneously disrupt iron metabolism by up regulating hepcidin production, a mechanism that induces functional iron deficiency by restricting both intestinal iron absorption and the mobilization of iron from body storage sites. Additionally, the physical nature of the dialysis procedure often leads to micronutrient depletion, as water-soluble vitamins like folate and vitamin B12 are routinely cleared during sessions [7].

Impaired renal phosphate excretion led to Hypophosphatemia, while hypocalcemia arises for the reason that kidneys cannot adequately activate vitamin D into calcitriol [8]. Secondary hyperparathyroidism progresses as a compensatory response to chronic hypocalcemia and phosphate retention, leading to bone demineralization and renal osteodystrophy in advanced CKD patients [9].

Oxidative stress contributes significantly to cardiovascular complications and progression of renal injury [10]. Infections remain a major cause of morbidity in HD patients because of impaired immune function, repeated vascular access manipulation, and prolonged hospitalization. Dialysis patients are particularly susceptible to blood-borne viral infections such as hepatitis B virus (HBV) and hepatitis C virus (HCV) [11].

As a result, continuous monitoring of hematological and biochemical parameters is important in hemo dialysis patients to estimate disease progression, inflammatory activity, treatment effectiveness, dialysis adequacy, nutritional status [12].

2. Materials and Methods

2.1. Study Design and Setting

This study was a prospective case-control study conducted at the Hemodialysis Center and Clinical Laboratory of Imam Al-Sadiq Hospital, Babil, Iraq, over a period of six months (October 2025 – March 2026).

2.2. Study Population

The study comprised two groups: (1) a patient group consisting of 40 patients undergoing maintenance hemodialysis, and (2) a control group consisting of 40 healthy group with no history of renal, hepatic, hematological, or chronic disease. All participants were adults aged 18–75 years.

2.3. Inclusion and Exclusion Criteria

2.3.1. Inclusion Criteria – Patient Group

- Confirmed diagnosis of ESRD receiving regular hemodialysis for at least 6 months
- Dialysis performed 3 times per week via arteriovenous fistula or graft
- Age 18–75 years
- Willing to provide written informed consent

2.4. Exclusion Criteria

- Active malignancy or immunosuppressive therapy
- Acute infection or hospitalization within the past month
- Patients with known primary liver disease (cirrhosis, hepatocellular carcinoma)
- Pregnancy or lactation
- History of blood transfusion within the past 3 months

2.5. Sample Collection and Laboratory Methods

For HD patients, blood samples (10 mL) were collected immediately before the dialysis session after an overnight fast of 8–12 hours. Samples were divided into: (a) EDTA tube for complete blood count (CBC) analyzed by automated hematology analyzer (Sysmex XN-1000) and (b) gel tube for biochemical tests. Serum was separated by centrifugation

at 3000 rpm for 10 minutes. Biochemical analyses were performed on automated chemistry analyzer. Assays included: urea, creatinine, liver enzymes (ALT and AST), electrolytes (Na, K,Ca, PO4).

2.6. Statistical Analysis

Statistical analysis was performed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (Mean ± SD). Comparison between groups was performed using the independent samples t-test for normally distributed variables. A p-value of less than 0.05 was considered statistically significant. Significance levels were coded as: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, ns = not significant.

3. Results and Discussion

Table 1 the result show serum urea and creatinine were profoundly elevated in HD patients compared to controls (urea: 92.7 ± 20.4 vs. 27.9 ± 4.8 mg/dL, $p < 0.00$; creatinine: 9.02 ± 1.92 vs. 0.90 ± 0.13 mg/dL, $p < 0.00$). These differences reflect the complete loss of glomerular filtration capacity in ESRD, resulting in accumulation of nitrogen waste

Table 1 Comparison of Renal Function Parameters (Mean ± SD)

Parameter	Controls (n=40) Mean ± SD	Patients (n=40) Mean ± SD	p-value
Urea (mg/dL)	27.86 ± 4.82	92.73 ± 20.39	0.00*
Creatinine (mg/dL)	0.901± 0.130	9.024 ± 1.922	0.00*

Table 2 shows the Hemoglobin was significantly reduced in HD patients (9.39 ± 1.36 g/dL) compared to controls (13.77 ± 0.98 g/dL; $p < 0.001$), confirming the renal anemia characteristic of ESRD. Hematocrit and platelet count were also significantly lower in the patient group. White blood cell count was higher in patients (8.09 ± 1.68 × 10^3/μL) compared to controls (7.10 ± 1.24 × 10^3/μL, $p < 0.001$), suggesting a chronic inflammatory state (Table 2). The significantly reduced hemoglobin (mean 9.2 g/dL) and hematocrit in HD patients is consistent with extensive literature documenting renal anemia. Topical revisions have established that almost all ESRD patients' improvement anemia, mainly insufficiency, iron insufficiency, and edited red cell life cycle [13].

The compact platelet tally in patient may imitate platelet activation during extracorporeal circulation. The elevated WBC sum, while within normal range in patients, feasible echoes the low-grade enduring painfulness that describes the uremic scene and is related with fast-tracked heart difficulty ischemia [14].

Table 2 Comparison of Hematological Parameters (Mean ± SD)

Parameter	Controls (n=40) Mean ± SD	Patients (n=40) Mean ± SD	p-value
Hemoglobin (g/dL)	13.77 ± 0.98	9.39 ± 1.36	0.00*
Hematocrit (%)	42.20 ± 2.74	28.35 ± 4.50	0.00*
Platelets (×10^3/μL)	255.58 ± 45.74	193.09± 46.84	0.00*
WBC (×10^3/μL)	7.10 ± 1.24	8.09 ± 1.68	0.00*

The results in table3 shows Serum potassium and phosphorus were markedly elevated in HD patients (potassium: 5.50 ± 0.63 vs. 4.07 ± 0.32 mEq/L, $p < 0.001$; phosphorus: 6.06 ± 0.96 vs. 3.52 ± 0.44 mg/dL, $p < 0.001$), reflecting impaired renal excretion. "Simultaneously, serum Ca deliberations verified a significant drop among the patient legion (8.28 \pm 0.70 mg/dL; $p < 0.001$), a result that bring into line closely with the clinical indexes of cardiovascular problems. Serum sodium levels also demonstrated a marginal yet statistically significant decline in these entitiies.

Increase level of Ca and phosphatemia, and hypo-calcemia in the group of hemodialysis are unfailing with recognized patho-physiology. The incapacity of the diseased kidney to evacuate potassium and phosphate indications to their accumulation, whereas impaired 1,25-dihydroxy vitamin D synthesis declines intestinal calcium captivation.

Contemporary nephrology literature confirms that reduced renal potassium excretion is the primary mechanism of hyperkalemia in chronic kidney disease, especially in advanced stages [15].

New research elevations in pre-dialysis potassium (>5.5 mEq/L) are associated with increased hazard of cardiac arrhythmias and sudden cardiac death, making hyperkalemia one of the most clinically critical electrolyte disturbances in CKD patients [16].

Hypo natremia observed in patients can replicate dilutional effects due to fluid overload and inter dialytic fluid retention [17].

Table 3 Comparison of Serum Electrolytes (Mean ± SD)

Parameter	**Controls (n=40) Mean ± SD**	**HD Patients (n=40) Mean ± SD**	**p-value**
Sodium (mEq/L)	140.68± 2.34	136.97± 3.64	0.00*
Potassium (mEq/L)	4.07 ± 0.32	5.50 ± 0.63	0.00*
Calcium (mg/dL)	9.51 ± 0.51	8.28 ± 0.70	0.00*
Phosphorus (mg/dL)	3.52 ± 0.44	6.06 ± 0.96	0.00*

4. Conclusion

- Hemodialysis patients parade severely elevated pre-dialysis urea and creatinine, brilliant a profound loss of renal division ability.
- Severe anemia is commonly prevalent patients, obsessed primarily by lacking erythropoietin making and chronic iron enervation.

Recommendations

- Appliance inclusive paper laboratory panels for hemodialysis patients also complete picture of blood, liver function enzymes, setting blood profile, and lipid sheet in addition to routine renal occupation tests.
- HCV and HBV screening should be performed for all hemodialysis patients; helpful cases ought to receive antiviral therapy per current strategies.

Compliance with ethical standards

Disclosure of conflict of interest

The authors haven't conflicts of interest, and this research customary without any cash supplementary.

Statement of ethical approval

This study was formally approved by the Institutional Review Boarding at Imam Al-Sadiq Hospital in Babylon, observing with worldwide ethical advices.

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