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DISTURBANCES IN HEMATOLOGICAL PROFILES AND ELECTROLYTE HOMEOSTASIS IN MAINTENANCE HEMODIALYSIS PATIENTS: A COMPARATIVE CASE-CONTROL STUDY

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ABSTRACT

Background: There are several different types of kidney replacement therapy, but the most frequent one is hemodialysis (HD).

Aim of the study: The present study aimed to evaluated level of hematological, and electrolyte in HD patients and control.

Materials and methods: This study involved 60 individuals of Iraqi renal failure patients with hemodialysis from Tikrit Teaching. The healthy group in volved 30 individuals, who were free from renal failure symptoms and other diseases. These samples were collected during the period from September 2025 to March 2026. Exclusion criteria Renal failure patients' pre-hemodialysis.

Result: White blood cell (WBC) and platelet (PLT) counts in HD patients were found to be non-significantly different from those in the control group ($P > 0.05$). On the other hand, HD patients had considerably lower levels of packed cell volume (PCV) and haemoglobin (Hb) than controls ($P < 0.001$). HD patients had considerably higher serum phosphate levels (5.8 ± 0.9 mg/dL) than the control group ($P < 0.001$). On the other hand, HD patients had considerably lower serum levels of calcium, chloride, potassium, and sodium than controls ($P < 0.001$).

Conclusion: The present study concluded that hemodialysis patients had significantly lower hemoglobin, packed cell volume, calcium, chloride, potassium, and sodium levels, while serum phosphate levels were significantly elevated.

Keywords: Maintenance Hemodialysis, Hematological Profiles, Electrolyte Homeostasis, End-Stage Renal Disease (ESRD)

1 INTRODUCTION

Haemodialysis (HD) is the predominant modality of kidney replacement therapy globally, comprising roughly 69% of all kidney replacement therapies and 89% of all dialysis procedures[1]. In the United States, more than 450,000 patients rely on maintenance HD to regulate serum electrolyte levels and acid-base balance [2]. Historically administered as a triweekly treatment, hemodialysis employs diffusion and convection to effectively and safely eliminate certain molecules while preserving or replenishing others[3, 4]. During hemodialysis, blood is extracted from the body and circulated via several minuscule porous tubes, which are perpetually immersed in a solution called dialysate. The dialysate possesses a specific electrolyte and chemical composition, allowing hazardous chemicals in the blood to permeate across the dialysis membrane and be eliminated in the dialysate. The formulation of the dialysate establishes the minimum threshold for the diffusion of electrolytes and glucose from the blood, so preventing their excessive depletion; surplus bodily fluid can be eliminated by adjusting the pressure across the dialysis membrane to facilitate ultrafiltration. The purified and concentrated blood is subsequently reinfused into the patient. A standard hemodialysis session endures for 4 hours and occurs thrice weekly (Kusiak et al., 2002).

The fast elimination of solutes and fluid can lead to clinical hypotension, the most prevalent acute consequence of hemodialysis treatment[5]. Electrolytes, or ions, are vital constituents that execute numerous critical processes within the body[6]. [6]. The main electrolytes that control metabolic and homeostatic processes such enzymatic reactions, bone mineralisation, nerve impulse conduction, muscle contraction, and osmotic equilibrium include sodium, potassium, calcium, magnesium, chloride, and phosphorus[6]. Their typical concentrations are essential for optimal bodily function[7]. Patients with HD experience significant variations in blood electrolytes due to compromised renal clearance, buildup during interdialytic periods, inconsistent nutritional intake, and rapid shifts during dialysis sessions [8]. Hypomagnesaemia and hyponatraemia are prevalent in hemodialysis[9]

Extreme hyperkalemia is marked by blood potassium levels above 6 or 5.5 mEq/l and clinical signs include arrhythmias, ECG abnormalities, muscular weakness, and ascending paralysis.1.2 Reduced potassium excretion and altered intracellular-extracellular distribution induce hyperkalemia in acute and chronic renal failure[10].

Kidney diseases are associated with changes in many biochemical and haematological indicators. Anemia is associated with the degree of renal impairment, primarily due to insufficient erythropoietin secretion by the kidneys. Additional factors include extended blood loss, hemolysis, and bone marrow suppression resulting from accumulated uremic chemicals[11]. The predominant synthesis of erythropoietin (EPO) occurs in the juxtaglomerular apparatus, with 10% produced in the liver and other organs. Alongside decreased erythropoietin levels, modifications in red blood cell (RBC) indices may arise from deficiencies in vitamin B12, iron, and folic acid, either from dietary insufficiencies or haemorrhage, or from a shortened lifespan of erythrocytes. Leukocyte count also affected in HD patients in addition platelet count, bleeding time, and prothrombin time[12].

2 MATERIALS AND METHODS

2.1 Sample collection

This study encompassed 60 Iraqi patients with renal insufficiency undergoing hemodialysis at Tikrit Teaching Hospital. The control groups comprised 30 healthy individuals, all of whom exhibited no indications of renal failure or other ailments. The samples were gathered between September 2025 and March 2026. Exclusion criteria: Patients with renal failure prior to hemodialysis.

2.2 Blood sample collection

A venipuncture was used to draw five millilitres of venous blood. Following sampling, a number of blood specimens were promptly put into EDTA-containing tubes for a thorough blood count examination. A different aliquot of venous blood was collected in anticoagulant-free, sterile polypropylene tubes. The serum was moved to sterile, dry, and clean tubes after centrifugation. the tests for biochemistry.

2.3 Assessment of biochemical parameters

Serum calcium and phosphate levels were measured using spectrophotometry using the serum analysis kit (Standard Kits) purchased from the French company Biolabo. FUJIFILM was used to measure the levels of [Na], [Cl], and [K].

2.4 Statistical analysis:

SPSS software (version 26, IBM Corp.) was used for all statistical analyses. The mean $\pm$ standard deviation (mean $\pm$ SD) was used to display continuous variables. The significance levels were categorised as follows: $p < 0.01$: extremely significant (**), $p < 0.05$: significant (*).

3 RESULTS

The current investigation revealed no significant differences in white blood cell (WBC) and platelet (PLT) counts in hemodialysis (HD) patients (8000 ± 145.1 cells/mm³, $141.8 \pm 23.7 \times 10^3$) compared to the control group (7600 ± 121.9 cells/mm³, $145 \pm 29.6 \times 10^3$ cells/mm³); ($P > 0.05$). Conversely, haemoglobin (Hb) and packed cell volume (PCV) levels were markedly diminished in HD patients (10.2 ± 0.5 g/dL, $32.9 \pm 2.1\%$) relative to controls (12.9 ± 0.06 g/dL, $37.4 \pm 2.7\%$) ($P < 0.001$).

Table 1: Comparison of Hematological Parameters between HD Patients and Healthy Controls

Parameters	HD patient	Control (20)	P-value
WBC cell/mm ³	8000 $\pm$ 145.1	7600 $\pm$ 121.9	0.09
Hb g/dl	10.2 $\pm$ 0.5	12.9 $\pm$ 0.06	<0.001
PCV%	32.9 $\pm$ 2.1	37.4 $\pm$ 2.7	<0.001
PLT cell/mm ³	141.8 $\pm$ 23.7	145 $\pm$ 29.6	0.2

Serum phosphate concentrations were markedly elevated in hemodialysis patients (5.8 ± 0.9 mg/dL) compared to the control group (3.5 ± 0.91 mg/dL) ($P < 0.001$). In contrast, blood levels of calcium, chloride, potassium, and sodium were markedly reduced in HD patients (7.3 ± 0.45 mg/dL, 98.05 ± 0.09 mmol/L, 2.4 ± 0.07 mmol/L, 132.01 ± 0.23 mmol/L) compared to controls (9.3 ± 1.02 mg/dL, 104 ± 0.2 mmol/L, 4.9 ± 0.13 mmol/L, 138.2 ± 0.5 mmol/L) ($P < 0.001$).

Table 2: Comparison of electrolyte between HD Patients and Healthy Controls

Parameters	HD patient	Control (20)	P-value
phosphate (mg\dl)	5.8 $\pm$ 0.9	3.5 $\pm$ 0.91	<0.001
calicum (mg\dl)	7.3 $\pm$ 0.45	9.3 $\pm$ 1.02	<0.001
chloride (Mmol/l)	98.05 $\pm$ 0.09	104 $\pm$ 0.2	<0.001
potassium(Mmol/l)	2.4 $\pm$ 0.07	4.9 $\pm$ 0.13	<0.001
sodium(Mmol/l)	132.01 $\pm$ 0.23	138.2 $\pm$ 0.5	<0.001

4 DISCUSSION

Anaemia is the predominant comorbidity linked to people undergoing chronic HD[13]. It generally arises from persistent inflammation and reduced erythropoietin synthesis by the kidneys[13]. In our study, Hb concentration exhibits a considerable reduction in HD. These findings concur with[14], which indicated that renal insufficiency is frequently linked to EPO -dependent anaemia in patients with renal failure. The principal aetiology of anaemia in dialysis patients is insufficient synthesis of EPO by the impaired kidneys. EPO circulates in plasma and stimulates erythroid progenitor cells in the bone marrow to generate RBCs[15].

This result aligns with [16], which noted that platelet counts rise during dialysis compared to healthy groups, concluding that uremic toxins accumulating in the blood of patients with renal failure may impair platelet function, induce hemostatic imbalances, and facilitate thrombotic disorders. Dialysis often enhances platelet abnormalities; nevertheless, haemodialysis may exacerbate bleeding due to persistent platelet activation caused by the interaction between blood and the artificial surfaces utilised in the dialysis process [17]

The current investigation revealed elevated phosphate levels and reduced calcium levels in HD patients compared to the control group. This study concurs with [18]. Insufficient intestinal absorption causes low calcium levels because calcium and phosphorus metabolism are linked. Intestinal calcium-to-phosphorus absorption should not exceed 1:2. As renal function declines, phosphate excretion is impaired, raising serum phosphate levels. As serum phosphate

concentrations decrease, blood calcium levels decrease. A drop in serum calcium enhances PTH secretion, which increases bone calcium resorption. Vitamin D is converted into active form by the kidneys. Low vitamin D levels limit intestinal calcium absorption, raising PTH[18].

The current study concurs with [19], which demonstrated hypochloremia and hyponatremia in hemodialysis patients; hypochloremia is frequently observed in individuals with renal failure. Baseline elevated blood chloride levels were substantially correlated with an increased risk of cardiovascular death in hemodialysis patients[20]. Electrolyte imbalances resulting from deteriorating renal function increase morbidity and mortality rates in patients with CKD [21]. Hyperkalemia is a common outcome in patients with end-stage renal failure, as shown by serum potassium concentrations. Potassium, the principal intracellular cation, modulates intracellular osmotic pressure[22]. Given that the standard potassium concentration is maintained within a narrow range, any increase may be lethal[23].

5 CONCLUSION

The present study concluded that hemodialysis patients had significantly lower hemoglobin, packed cell volume, calcium, chloride, potassium, and sodium levels, while serum phosphate levels were significantly elevated. These findings reflect the combined effects of impaired renal function and the hemodialysis process on electrolyte balance and hematological status.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

Statement of informed consent

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

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