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Effect of a Single Hemodialysis Session on Hemostatic Function in Patients With Chronic Kidney Disease

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Abstract

Chronic kidney disease (CKD) is associated with complex disturbances in hemostasis that may increase susceptibility to both bleeding and thrombotic complications. Hemodialysis, an essential treatment for advanced CKD, may further influence hematological and coagulation measurements through extracorporeal blood circulation, fluid removal, and anticoagulation. This observational study assessed changes in selected hematological and coagulation parameters immediately before and after a single hemodialysis session. Thirty-two patients receiving maintenance hemodialysis at Al-Fallujah Teaching Hospital were enrolled, and paired blood specimens were analyzed using complete blood count measurements and routine coagulation assays, including prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen, and D-dimer. Most hematological comparisons were based on 31 paired measurements, whereas coagulation parameters were available for 32 participants.

WBC and neutrophil counts, RBC count, hemoglobin, and hematocrit showed statistically significant post-dialysis increases in the reported analyses. Platelet count and routine platelet indices showed no significant differences. PT and APTT decreased slightly, while fibrinogen and D-dimer increased modestly; none of these coagulation differences reached statistical significance. These results suggest that one hemodialysis session is associated with selective short-term changes in circulating blood-cell measurements, whereas conventional coagulation indices remained relatively stable in this small cohort. Larger studies that document anticoagulation, fluid removal, and additional measures of platelet and coagulation function are warranted to clarify the mechanisms and clinical relevance of these findings.

Keywords: chronic kidney disease; hemodialysis; hemostasis; coagulation; platelets; D-dimer

1. INTRODUCTION

Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function that persist for at least three months and have implications for health. CKD represents a substantial global burden because it is associated with progressive impairment of renal function, cardiovascular complications, morbidity, and mortality [1, 2]. Advanced CKD may progress to kidney failure, requiring kidney replacement therapy, including hemodialysis or transplantation. The progressive loss of functional nephron mass affects fluid and electrolyte homeostasis and has consequences that extend well beyond the kidneys.

Beyond impaired renal function, CKD affects hematological and hemostatic systems. Hemostasis is a tightly regulated physiological process that keeps circulating blood fluid while permitting the prompt formation of a localized clot following vascular injury. Platelets, coagulation factors, the vascular endothelium, and fibrinolytic mechanisms work together to maintain this balance. In CKD, uremic toxins, chronic inflammation, and endothelial dysfunction can disrupt several components of this system, thereby increasing susceptibility to both hemorrhagic and thrombotic complications [3].

Hemostatic abnormalities in CKD arise through interactions among procoagulant, anticoagulant, and fibrinolytic pathways. Accumulation of uremic toxins may impair platelet adhesion and aggregation, whereas inflammation and endothelial dysfunction can promote platelet activation and coagulation. Changes in fibrin formation and dissolution further complicate this balance. Consequently, bleeding and thrombosis can coexist in affected individuals despite their apparently opposing mechanisms. These disturbances may become more pronounced as renal function deteriorates and uremic toxins accumulate [4].

Hemodialysis is a frequently used kidney replacement therapy for patients with advanced CKD. Although the procedure removes uremic solutes and helps regulate fluid and metabolic homeostasis, it can also influence hematological and coagulation indices. During treatment, blood encounters the artificial surfaces of the extracorporeal circuit, including the dialysis membrane, which may activate inflammatory, complement, platelet, and coagulation pathways. Anticoagulants are also routinely administered to prevent clotting within the circuit. These physiological and pharmacological influences may alter hemostatic measurements during or immediately after dialysis [5].

Repeated exposure to extracorporeal circulation may contribute to platelet activation, endothelial stimulation, and changes in fibrinolytic activity. These mechanisms are particularly relevant to individuals receiving regular hemodialysis, because they undergo repeated extracorporeal procedures and anticoagulant administration. The interaction between underlying CKD-related hemostatic abnormalities and changes related to an individual dialysis session may influence the risks of both bleeding and thrombosis [6].

Despite these recognized mechanisms, findings concerning the immediate effects of hemodialysis on hematological and coagulation indices remain inconsistent. Some investigations have reported measurable changes after dialysis, whereas others have observed small or statistically nonsignificant differences. Variation in patient characteristics, dialysis prescriptions, anticoagulation regimens, membrane types, sampling time, and laboratory measurements may contribute to these discrepancies. Comparing paired pre- and post-dialysis samples may therefore help characterize acute changes, although the findings do not establish their mechanisms or clinical consequences.

Accordingly, this study evaluated selected hematological and coagulation parameters immediately before and after a single hemodialysis session in patients with CKD. By comparing measurements from the same individuals at both time points, it aimed to describe the short-term changes associated with the procedure and identify which routine parameters were most responsive. The analysis also considered differences among age groups, while recognizing the limitations of a small observational sample.

2. LITERATURE REVIEW

Chronic kidney disease is characterized by persistent abnormalities of kidney structure or function, with clinical consequences that may increase as the disease advances. In patients undergoing kidney replacement therapy, impaired renal function can contribute to the retention of uremic toxins, inflammation, and disturbances in physiological regulation. These processes affect hematological functions, including erythropoiesis, platelet function, and coagulation, and complicate the clinical management of advanced CKD [1, 4].

Anemia is among the major hematological complications of advanced CKD. Reduced erythropoietin production contributes to impaired red-cell formation, while inflammation, iron restriction, nutritional factors, and blood loss related to repeated procedures may also be involved [7, 8]. In addition to anemia, platelet and coagulation abnormalities are clinically important manifestations of the hematological disturbances observed in people with CKD.

Hemostatic disturbances in CKD are multifactorial and involve interactions among platelets, coagulation factors, fibrinolytic proteins, and vascular integrity. Uremic toxins, chronic inflammation, oxidative stress, and endothelial dysfunction may impair platelet adhesion or aggregation while simultaneously promoting activation of coagulation pathways [9, 10]. Accordingly, patients may experience an increased tendency toward either bleeding or thrombosis, depending on their

physiological and treatment-related circumstances.

The bleeding tendency associated with advanced CKD is partly attributed to platelet dysfunction. Impaired platelet adhesion, aggregation, or interactions with von Willebrand factor may compromise primary hemostasis [4, 11]. Conversely, elevations in procoagulant factors such as fibrinogen and factor VIII, together with endothelial activation, can contribute to a prothrombotic state [8, 10]. Thus, hemostatic disorders in CKD cannot be described adequately as exclusively hypocoagulable or hypercoagulable.

Hemodialysis can further influence hematological and hemostatic measurements. During treatment, blood circulates through an extracorporeal circuit and comes into contact with artificial membrane surfaces. Anticoagulation is often necessary to maintain circuit patency and may itself alter the measured coagulation profile. Interactions among these factors can affect platelet activity, plasma composition, and routine test results during and shortly after dialysis [5].

Published studies have reported variable effects of hemodialysis on platelet and coagulation parameters. Although the removal of uremic toxins may improve some aspects of platelet function, dysfunction may persist, and extracorporeal circulation can produce additional platelet activation [3, 11, 12]. Similarly, studies assessing conventional coagulation measures, including prothrombin time (PT) and activated partial thromboplastin time (APTT), have not demonstrated consistent directions or magnitudes of post-dialysis change.

Fibrinogen may also be altered during hemodialysis. Elevated fibrinogen in CKD can reflect chronic inflammation and contribute to a prothrombotic tendency, while changes within a single session may be influenced by hemoconcentration and inflammatory activation [10]. D-dimer, a marker reflecting fibrin formation and degradation, may provide complementary information about coagulation and fibrinolytic activity. However, changes in either marker require interpretation in the context of the overall clinical condition and dialysis protocol.

Differences in reported findings may reflect variation in patient demographics, dialysis duration, the amount and timing of anticoagulation, dialyzer membrane characteristics, fluid removal, and laboratory methodology. Assessing samples collected immediately before and after a treatment therefore provides a practical approach to studying short-term physiological responses. Interpretation nevertheless requires careful attention to preanalytical factors and to the absence of more specialized platelet-function and coagulation measurements.

Although hemodialysis is essential for many patients with advanced CKD, its immediate influence on conventional hematological and coagulation parameters remains incompletely characterized. This uncertainty supports a focused, paired comparison of measurements obtained around a single treatment session.

3. MATERIALS AND METHODS

The laboratory instruments, reagents, and diagnostic kits used for the assessment of hematological and coagulation parameters are summarized in Tables 1 and 2.

Table 1. *Laboratory instruments used in the study*

Instrument / Material	Company	Country
Centrifuge	–	–
EDTA Tube	–	–
GENEX Thrombo Coagulation Analyzer	GENEX	China
Getein 1180 GP Immunofluorescence Analyzer	Getein Biotech	China
Micropipette (100–1000 μ L)	–	–
Micropipette (10–100 μ L)	–	–
Plain Tube	–	–
Sodium Citrate Tube	–	–
Syringe (5 mL)	–	–
Zybio Z50 Hematology Analyzer	Zybio Inc.	China

Table 2. *Reagents and diagnostic kits used in the study*

Material / Reagent	Company	Country
BIOLAB PT Reagent	BIOLABO	France
BIOLAB APTT Reagent	BIOLABO	France
BIOLAB Fibrinogen Reagent	BIOLABO	France
D-dimer Fast Test Kit	Getein	China

This observational, paired pre- and post-dialysis study evaluated changes in selected hematological and coagulation measurements associated with one hemodialysis session in individuals with CKD. The study was conducted in a hospital laboratory setting, and the same participants contributed measurements at both collection times.

A total of 32 patients undergoing maintenance hemodialysis were selected from the Hemodialysis Unit at Al-Fallujah

Teaching Hospital. Blood specimens were collected immediately before and after one dialysis session. Matched observations were intended for comparison of hematological and coagulation parameters. The reported number of available observations varied by laboratory parameter and should be reconciled with the study's missing-data records.

The study was conducted from 10 October 2025 to 20 October 2025.

The study procedure, including patient selection, blood collection at both time points, sample processing, laboratory measurements, and statistical analysis, is summarized in Figure 1. Blood specimens were obtained immediately before and after hemodialysis. Each specimen was labeled at the time of collection to support patient identification, sample traceability, and the prevention of preanalytical errors [13].

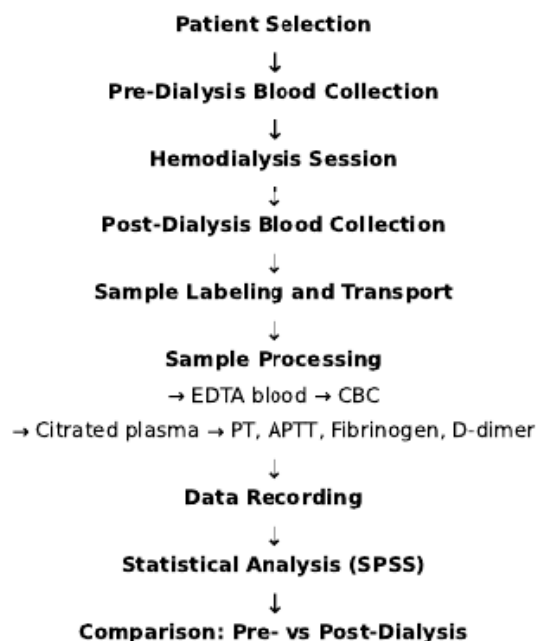


Figure 1. Schematic workflow of the study, including patient selection, pre- and post-hemodialysis blood collection, sample processing, laboratory analysis, and statistical evaluation

Venous blood was obtained using aseptic venipuncture procedures and sterile disposable collection devices. Tourniquet application was limited to the period necessary to establish adequate blood flow, thereby reducing the likelihood of venous stasis and associated preanalytical variation.

The recommended order of draw was followed to reduce additive carryover between collection tubes. When specimens were obtained during the same venipuncture, coagulation tubes containing sodium citrate were collected before EDTA tubes. Sample collection and handling procedures were based on established phlebotomy and laboratory recommendations [13–15].

Collection tubes were selected according to the anticoagulant and specimen requirements of the laboratory assays. Coagulation testing was performed on citrated plasma, whereas hematological testing was performed on EDTA-anticoagulated whole blood.

For coagulation studies, venous blood was collected into tubes containing 3.2% sodium citrate (0.109 M), maintaining a nominal blood-to-anticoagulant ratio of 9:1. Citrated plasma was used for PT, APTT, fibrinogen, and D-dimer measurements. Appropriate filling of citrate tubes is important for obtaining valid coagulation results [15].

For hematological testing, EDTA-anticoagulated whole blood was used for a complete blood count (CBC), including white blood cell (WBC) and red blood cell (RBC) counts, hemoglobin (Hb), hematocrit (HCT), platelet count, and associated cellular indices [14].

After collection, blood specimens were transported promptly to the laboratory under routine temperature conditions. Excessive agitation and exposure to extreme temperatures were avoided to minimize deterioration and preanalytical variation [13].

Citrated blood specimens underwent centrifugation at $1,500\text{--}2,000 \times g$ for 10–15 minutes at room temperature to obtain platelet-poor plasma. The plasma fraction was then transferred carefully into clean tubes without disturbing the cellular layer [16].

Routine specimen-rejection criteria were applied to visibly clotted or hemolyzed samples and tubes with an unsuitable blood-to-anticoagulant ratio resulting from inadequate filling. These controls were intended to reduce errors arising from specimen collection and processing [16].

The main laboratory analyzers used in this study are described below and illustrated in Figures 2–4:

- The GENEX Thrombo semi-automated coagulation analyzer (GENEX, China) detects clot formation using an optico-mechanical principle and was used for PT, APTT, and fibrinogen assays (Figure 2).



Figure 2. GENEX Thrombo semi-automated coagulation analyzer used for determination of PT, APTT, and fibrinogen

- The Getein 1180 GP immunofluorescence analyzer (Getein Biotech, China) employs a fluorescence-based immunoassay for quantitative determination of D-dimer (Figure 3).

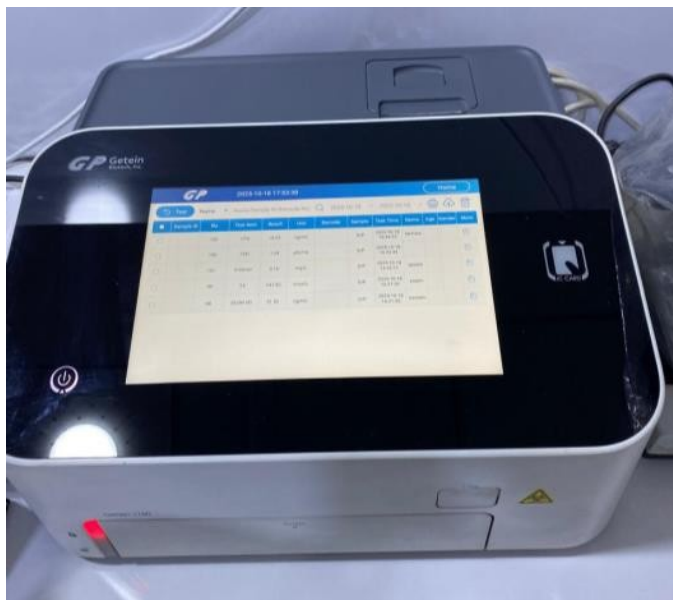


Figure 3. Getein 1180 GP immunofluorescence analyzer used for quantitative determination of D-dimer

- The Zybio Z50 hematology analyzer (Zybio Inc., China) was used for CBC testing, employing electrical impedance for cell counting and a colorimetric method for hemoglobin measurement (Figure 4).

Laboratory tests were performed according to the relevant reagent and analyzer instructions. Analytical conditions, including sample and reagent volumes, incubation times, and reaction temperatures, were monitored to maintain consistency between the pre- and post-dialysis measurements.

Citrated platelet-poor plasma was used for the prothrombin time (PT) test with BIOLABO PT reagent (REF 13880) and GENEX Thrombo semi-automated coagulation analyzer. This assay tests the extrinsic pathway and common pathway of coagulation (Figure 5).

PT reagent included a freeze-dried thromboplastin from rabbit brain tissue (R1), as well as a reconstitution buffer based on HEPES containing a stabilizer (R2). The working reagent was obtained by dispensing 4 mL of R2 into the R1 vial, followed by gentle manual mixing until dissolution occurred.

For the assay, the working reagent and plasma samples were maintained at 37°C. A 50-μL aliquot of plasma was introduced into the analyzer, followed by 50 μL of pre-warmed thromboplastin reagent. Fibrin clot formation was detected

using the analyzer's optico-mechanical clot detection system, and the clotting time was automatically recorded in seconds as the PT value, according to the manufacturer's instructions [17].

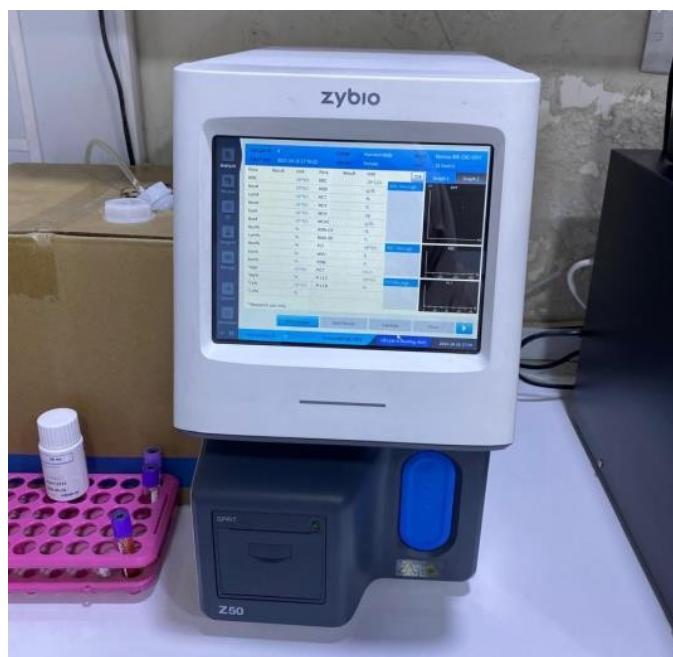


Figure 4. Zybio Z50 automated hematology analyzer used for CBC analysis

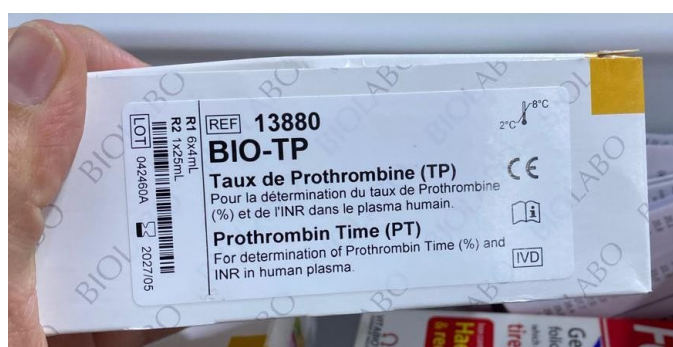


Figure 5. BIOLABO prothrombin time reagent used for determination of PT

The manufacturer-reported reference range for PT was 11–13.5 s.

Activated partial thromboplastin time (APTT) was determined using BIOLABO APTT reagent (REF 13560) with the GENEX Thrombo semi-automated coagulation analyzer. The assay evaluates the intrinsic and common coagulation pathways (Figure 6).

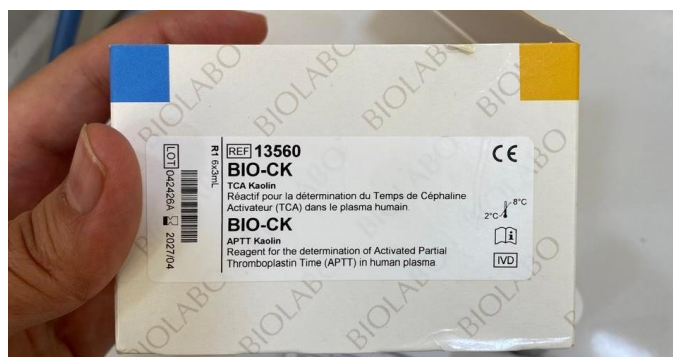


Figure 6. BIOLABO APTT reagent used for assessment of the intrinsic coagulation pathway

The BIO-CK reagent (R1) contained cephalin as a platelet substitute and kaolin as an activator. The freeze-dried reagent was reconstituted with 30 mL of distilled water and gently mixed until complete dissolution to obtain the working reagent.

For the analysis, 50 μL of plasma was combined with 50 μL of the working reagent and incubated at 37°C for the designated duration. Subsequently, 50 μL of calcium chloride solution was added rapidly to initiate coagulation. Clot formation was detected by the analyzer, and the clotting time was automatically recorded in seconds according to the manufacturer's instructions [17].

The manufacturer-reported reference range for APTT was 25–40 s. Reagents and plasma samples were brought to the required temperature before analysis, and new pipette tips were used between samples to minimize cross-contamination.

Plasma fibrinogen concentration was determined using the BIOLABO fibrinogen reagent (REF 13450) with the GENEX Thrombo semi-automated coagulation analyzer according to the Clauss method (Figure 7). The assay is based on measuring the clotting time of diluted plasma following exposure to a high concentration of thrombin.



Figure 7. BIOLABO fibrinogen reagent used for quantitative determination of plasma fibrinogen concentration

The BIO-FIBRI reagent consisted of freeze-dried thrombin derived from animal sources with a small quantity of kaolin (R1), while R2 served as the plasma-diluting buffer. Reagent R1 was reconstituted with 4 mL of demineralized water and gently mixed until complete dissolution. The reconstituted reagent was allowed to equilibrate at room temperature (20–25°C) before use.

Plasma was diluted at a ratio of 1:10 by combining 20 μL of plasma with 180 μL of R2. A 50- μL sample of the diluted plasma was maintained at 37°C for 2 minutes, after which 50 μL of the thrombin reagent was swiftly introduced to initiate fibrin clot formation. The clotting time was measured by the analyzer and converted to fibrinogen concentration using the manufacturer's calibration curve [18].

The manufacturer-reported reference range for plasma fibrinogen was 200–400 mg/dL.

D-dimer concentration was quantitatively determined using the Getein 1180 GP immunofluorescence analyzer and D-dimer Fast Test Kit (REF IF3006) (Figure 8). The test is based on D-dimer antigen fluorescent immunodetection.



Figure 8. Getein D-dimer Fast Test Kit used for quantitative determination of plasma D-dimer concentration

Test cards, plasma samples, and reagents were brought to room temperature before testing. The analyzer was prepared according to the manufacturer's instructions, including installation of the assay-specific SD card and entry of sample information. A 100- μL aliquot of plasma was added to the specified diluent and mixed thoroughly. Subsequently, 100 μL of the prepared sample mixture was dispensed into the sample well of the test card. The card was inserted into the analyzer, and the reaction was allowed to proceed for approximately 10 minutes. The instrument measured the fluorescence signal resulting from the antigen–antibody reaction and reported the quantitative D-dimer concentration.

The analysis was conducted in accordance with the guidelines provided by the manufacturer [19]. The reference range for D-dimer, as provided by the manufacturer, was <0.5 mg/L. Test cards were used within 1 h of opening the foil package, as recommended by the manufacturer.

A complete blood count (CBC) was performed on EDTA-anticoagulated whole blood using the Zybco Z50 automated hematology analyzer. Before analysis, blood samples were gently mixed by inversion to ensure uniform distribution of blood cells within the anticoagulant.

The analyzer uses electrical impedance for cellular counting and colorimetric measurement for hemoglobin determination. The measured parameters included white blood cell (WBC) count, red blood cell (RBC) count, haemoglobin

(Hb), haematocrit (HCT), platelet count, and related red blood cell and platelet indices. All evaluations were conducted in accordance with the manufacturer's guidelines [20].

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 27. Paired-samples t-tests were used to compare hematological and coagulation measurements obtained immediately before and after dialysis in the same individuals. One-way analysis of variance (ANOVA) was used to compare continuous variables among the reported age groups. All tests were interpreted using a two-sided significance threshold of $p < 0.05$. The original paired records and group sizes are required to independently reproduce the reported tests and assess missing observations.

4. RESULTS

Thirty-two patients with CKD receiving maintenance hemodialysis were enrolled at the Hemodialysis Unit of Al-Fallujah Teaching Hospital. Laboratory measurements were performed before and after a single session. Although the enrolled sample comprised 32 individuals, the available sample sizes for several hematological indices were reported as 31, with 30 observations for basophil counts. These differences should be accounted for in the final analysis.

The hematological parameters measured before and after hemodialysis are summarized in Tables 3 and 4 and illustrated in Figure 9.

Table 3. Comparison of leukocyte parameters before and after hemodialysis in patients with CKD

Hematological marker		Dialysis status		Significance (p -value)
		Before	After	
WBC	Mean $\pm$ SD	5.45 $\pm$ 2.003	6.21 $\pm$ 3.247	0.044*
	No.	31	31	
Neutrophil	Mean $\pm$ SD	3.33 $\pm$ 1.734	4.11 $\pm$ 2.635	0.006**
	No.	31	31	
Lymphocytes	Mean $\pm$ SD	1.44 $\pm$ 0.740	1.86 $\pm$ 2.543	0.377 ^{NS}
	No.	31	31	
Monocytes	Mean $\pm$ SD	0.47 $\pm$ 0.259	0.67 $\pm$ 1.062	0.313 ^{NS}
	No.	31	31	
Eosinophils	Mean $\pm$ SD	0.19 $\pm$ 0.121	0.19 $\pm$ 0.124	0.914 ^{NS}
	No.	31	31	
Basophil	Mean $\pm$ SD	0.02 $\pm$ 0.011	2.07 $\pm$ 11.243	0.324 ^{NS}
	No.	30	30	

NS = not statistically significant; * $p < 0.05$; ** $p < 0.01$.

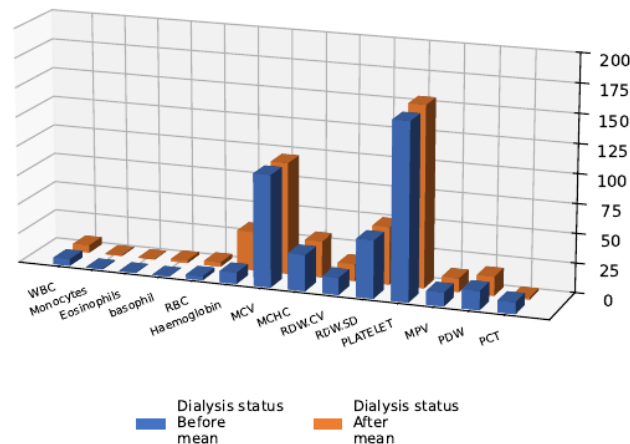


Figure 9. Comparison of hematological parameters before and after hemodialysis in patients with CKD

The mean WBC count increased from $5.45 \pm 2.00 \times 10^9/L$ before dialysis to $6.21 \pm 3.25 \times 10^9/L$ afterward ($p = 0.044$). The mean neutrophil count also increased, from 3.33 ± 1.73 to $4.11 \pm 2.64 \times 10^9/L$ ($p = 0.006$). These changes were statistically significant. By contrast, lymphocyte, monocyte, eosinophil, and basophil counts showed no statistically significant pre- to post-dialysis differences in the reported analyses.

Among erythrocyte parameters, the mean RBC count increased from 3.19 ± 0.53 to $3.46 \pm 0.66 \times 10^{12}/L$ after dialysis (reported $p < 0.001$). Mean hemoglobin increased from 9.27 to 9.98 g/dL, and mean hematocrit increased from 30.20% to 32.74% (reported $p < 0.001$ for both). The corresponding standard deviations should be checked against the original data because the values in the narrative and table are not entirely consistent. No statistically significant differences were

Table 4. Comparison of erythrocyte and platelet parameters before and after hemodialysis in patients with CKD

Hematological marker		Dialysis status		Significance (p-value)
		Before	After	
RBC	Mean±SD	3.19 ±0.533	3.46± 0.660	< 0.001**
	No.	31	31	
Hemoglobin	Mean±SD	9.27 ±1.277	9.98± 5.240	< 0.001**
	No.	31	31	
Hematocrit	Mean±SD	30.20 ± 4.265	32.74 ±5.240	< 0.001**
	No.	31	31	
MCV	Mean±SD	95.05 ±5.059	94.64±5.696	0.501 ^{NS}
	No.	31	31	
MCHC	Mean±SD	30.69 ±0.810	30.56±0.969	0.198 ^{NS}
	No.	31	31	
RDW.CV	Mean±SD	14.12 ±0.988	14.09±0.954	0.495 ^{NS}
	No.	31	31	
RDW.SD	Mean±SD	48.91 ±4.555	48.81± 4.438	0.457 ^{NS}
	No.	31	31	
Platelet	Mean±SD	150.52 ±55.970	154.13 ± 60.389	0.334 ^{NS}
	No.	31	31	
MPV	Mean±SD	11.42 ±1.231	11.41± 1.129	0.921 ^{NS}
	No.	31	31	
PDW	Mean±SD	15.89 ± 0.461	15.98 ± 0.475	0.169 ^{NS}
	No.	31	31	
PCT	Mean±SD	9.77 ± 45.146	1.75± 0.712	0.329 ^{NS}
	No.	31	31	

NS = not statistically significant; * $p < 0.05$; ** $p < 0.01$.

reported for MCV, MCHC, RDW-CV, or RDW-SD.

Platelet count increased slightly from 150.52 ± 55.97 to $154.13 \pm 60.39 \times 10^9/L$, but the difference was not statistically significant ($p = 0.334$). Likewise, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) showed no statistically significant changes after dialysis in the reported analysis.

The coagulation parameters measured before and after hemodialysis are summarized in Table 5 and Figure 10.

Table 5. Comparison of coagulation parameters before and after hemodialysis in patients with CKD

Haemostatic marker	Dialysis status		Significance (p value)
	Before	After	
Prothrombin time	17.69 ±17.081	13.38± 3.074	0.144 ^{NS}
	32	32	
Activated Partial Thromboplastin time	39.96 ±11.152	35.82± 10.394	0.061 ^{NS}
	32	32	
INR	1.29± 0.825	1.13± 0.343	0.143 ^{NS}
	32	32	
Fibrinogen	224.18±50.912	238.86± 61.983	0.084 ^{NS}
	32	32	
D-dimer	1.72± 2.359	1.88± 2.532	0.268 ^{NS}
	32	32	

NS = not statistically significant.

Prothrombin time decreased from 17.69 ± 17.08 seconds before dialysis to 13.38 ± 3.07 seconds afterward, a difference that did not reach statistical significance ($p = 0.144$). APTT declined from 39.96 ± 11.15 to 35.82 ± 10.39 seconds ($p = 0.061$), while the international normalized ratio (INR) decreased from 1.29 ± 0.83 to 1.13 ± 0.34 ($p = 0.143$). Neither comparison was statistically significant.

Fibrinogen increased from 224.18 ± 50.91 to 238.86 ± 61.98 mg/dL ($p = 0.084$), while D-dimer increased from 1.72 ± 2.36 to 1.88 ± 2.53 mg/L ($p = 0.268$). The changes were small and did not reach the prespecified threshold for statistical significance.

Age-stratified hematological findings are presented in Table 6 (leukocyte parameters), Table 7 (erythrocyte parameters), and Table 8 (platelet parameters). The separate presentation of these indices permits examination of between-group variation before and after dialysis; however, some age groups contain very small numbers of participants.

One-way ANOVA indicated that most hematological measurements did not differ significantly among the reported age groups before or after hemodialysis. Exceptions included basophil count before dialysis, MCHC before and after dialysis,

and plateletcrit before dialysis, according to the tabulated *p*-values. These findings require cautious interpretation because the subgroup sizes were small and certain values were unusually dispersed.

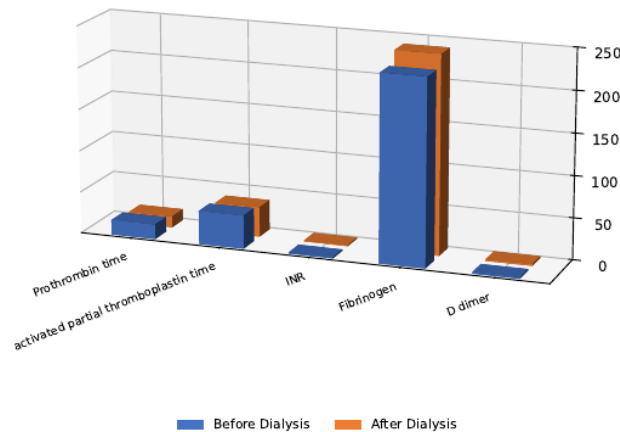


Figure 10. Comparison of coagulation parameters before and after hemodialysis in patients with CKD

Table 6. Distribution of leukocyte parameters by age group before and after hemodialysis

Hematological Markers	Dialysis Status	Age Groups	<40	41-55	56-70	>71	<i>p</i> -value
		No.	4	8	16	3	
WBC	Before	Mean ± SD	5.86± 1.4	4.82± 2.4	5.2± 1.635	7.97 ±2.06	0.108 ^{NS}
	After	Mean ± SD	6.10± 1.885	5.37± 2.7	5.80± 2.766	10.83 ±5.6	0.067 ^{NS}
NEU.	Before	Mean ± SD	3.28± 1.588	2.93± 1.5	3.16± 1.347	5.39± 3.47	0.185 ^{NS}
	After	Mean ± SD	3.79± 1.803	3.57± 2.1	3.78± 1.795	7.69± 6.14	0.096 ^{NS}
LYM.	Before	Mean ± SD	1.81± 0.836	1.31± 0.7	1.37± 0.574	1.75± 1.39	0.607 ^{NS}
	After	Mean ± SD	1.65± 0.473	1.26± 0.6	2.22± 3.497	1.82± 1.00	0.864 ^{NS}
MON.	Before	Mean ± SD	0.53± 0.256	0.40± 0.1	0.47± 0.314	0.59± 0.17	0.726 ^{NS}
	After	Mean ± SD	0.45± 0.188	0.40± 0.1	0.79± 1.452	1.01± 0.52	0.770 ^{NS}
EOS.	Before	Mean ± SD	0.23± 0.107	0.17± 0.1	0.18± 0.117	0.22± 0.20	0.810 ^{NS}
	After	Mean ± SD	0.19± 0.082	0.13± 0.07	0.20± 0.138	0.28± 0.18	0.332 ^{NS}
BAS.	Before	Mean ± SD	0.03± 0.006	0.01± 0.01	0.02± 0.010	0.02± 0.01	0.037 [*]
	After	Mean ± SD	0.02± 0.013	0.03± 0.04	3.87± 15.39	0.03± 0.01	0.833 ^{NS}

NS = not statistically significant; * *p* < 0.05; ** *p* < 0.01.

Table 7. Distribution of erythrocyte parameters by age group before and after hemodialysis

Hematological Markers	Dialysis Status	Age Groups	<40	41-55	56-70	>71	<i>p</i> -value
		No.	4	8	16	3	
RBC	Before	Mean ± SD	3.47± 0.741	3.01±0.50	3.17± 0.504	3.43± 0.51	0.471 ^{NS}
	After	Mean ± SD	3.81± 0.951	3.24±0.59	3.44± 0.645	3.68± 0.53	0.516 ^{NS}
HB	Before	Mean ± SD	9.85± 1.507	9.05±1.55	9.18± 1.132	9.60± 1.37	0.740 ^{NS}
	After	Mean ± SD	10.88± 1.95	9.65±1.64	9.89± 1.534	10.17± 1.4	0.648 ^{NS}
HCT	Before	Mean ± SD	31.78± 4.46	29.05±5.1	29.99± 3.89	32.30± 4.4	0.617 ^{NS}
	After	Mean ± SD	34.95± 6.25	31.13±5.2	32.62± 5.28	34.73± 4.4	0.612 ^{NS}
MCV	Before	Mean ± SD	92.60± 6.29	96.49±4.7	95.05± 5.32	94.47± 3.6	0.675 ^{NS}
	After	Mean ± SD	88.33± 8.14	96.49±4.3	95.29± 5.29	94.67± 3.6	0.107 ^{NS}
MCH	Before	Mean ± SD	28.68± 1.78	30.11±1.1	29.11± 2.09	27.97± 1.5	0.310 ^{NS}
	After	Mean ± SD	28.95± 1.88	29.90±1.0	29.55± 1.90	27.73± 1.5	0.283 ^{NS}
MCHC	Before	Mean ± SD	30.95± 0.40	31.20±0.7	30.61± 0.69	29.40± 0.5	0.005 ^{**}
	After	Mean ± SD	31.20± 0.29	31.04±0.5	30.39± 1.02	29.30± 0.7	0.018 ^{**}
RDW.CV	Before	Mean ± SD	13.15± 0.19	14.16±1.1	14.19± 0.97	14.90± 0.3	0.116 ^{NS}
	After	Mean ± SD	13.13± 0.17	14.11±1.0	14.16± 0.95	14.93± 0.2	0.080 ^{NS}

Coagulation parameters according to age group, before and after hemodialysis, are presented in Table 9. The table reports PT, APTT, INR, fibrinogen, and D-dimer measurements for each age category.

Analysis of variance did not identify statistically significant differences among the reported age categories in PT, APTT, INR, fibrinogen, or D-dimer before or after dialysis. These nonsignificant comparisons should not be taken as proof that age has no influence, particularly because the smallest categories included only three or four participants.

Table 8. Distribution of platelet parameters by age group before and after hemodialysis

Hematological Markers	Dialysis Status	Age Groups	<40	41-55	56-70	>71	p-value
		No.	4	8	16	3	
PLT.	Before	Mean $\pm$ SD	176.2 $\pm$ 34.5	129.2 $\pm$ 69.2	143.3 $\pm$ 49.9	211.3 $\pm$ 24.9	0.116 ^{NS}
	After	Mean $\pm$ SD	190.7 $\pm$ 66.4	131.6 $\pm$ 60.7	146.0 $\pm$ 57.5	208.6 $\pm$ 24.9	0.147 ^{NS}
MPV	Before	Mean $\pm$ SD	12.13 $\pm$ 1.70	11.38 $\pm$ 1.24	11.34 $\pm$ 1.23	11.03 $\pm$ 0.5	0.661 ^{NS}
	After	Mean $\pm$ SD	12.03 $\pm$ 1.50	11.04 $\pm$ 0.96	11.43 $\pm$ 1.21	11.47 $\pm$ 0.4	0.57 ^{NS}
PDW	Before	Mean $\pm$ SD	15.65 $\pm$ 0.38	16.19 $\pm$ 0.56	15.80 $\pm$ 0.40	15.90 $\pm$ 0.3	0.170 ^{NS}
	After	Mean $\pm$ SD	15.80 $\pm$ 0.62	16.18 $\pm$ 0.66	15.96 $\pm$ 0.35	15.83 $\pm$ 0.3	0.547 ^{NS}
PCT	Before	Mean $\pm$ SD	2.16 $\pm$ 0.598	1.43 $\pm$ 0.718	1.61 $\pm$ 0.551	85.6 $\pm$ 144.9	0.016 ^{**}
	After	Mean $\pm$ SD	2.31 $\pm$ 0.944	1.43 $\pm$ 0.608	1.65 $\pm$ 0.651	2.39 $\pm$ 0.2	0.070 ^{NS}
RDW.SD	Before	Mean $\pm$ SD	44.38 $\pm$ 3.00	49.76 $\pm$ 4.8	49.19 $\pm$ 4.66	51.13 $\pm$ 0.7	0.169 ^{NS}
	After	Mean $\pm$ SD	44.23 $\pm$ 3.09	49.64 $\pm$ 4.4	49.11 $\pm$ 4.56	51.17 $\pm$ 1.1	0.137 ^{NS}

NS = not statistically significant; * $p < 0.05$; ** $p < 0.01$.

Table 9. Distribution of coagulation parameters by age group before and after hemodialysis

Hematological Markers	Dialysis Status	Age Groups	<40	41-55	56-70	>71	p-value
		No.	4	8	16	3	
PT	BEFORE	Mean $\pm$ SD	11.73 $\pm$ 1.77	25.68 $\pm$ 30.9	15.86 $\pm$ 9.87	14.73 $\pm$ 4.375	0.490 ^{NS}
	AFTER	Mean $\pm$ SD	12.78 $\pm$ 1.61	13.63 $\pm$ 3.27	12.98 $\pm$ 3.35	15.80 $\pm$ 1.905	0.521 ^{NS}
APTT	BEFORE	Mean $\pm$ SD	39.20 $\pm$ 6.16	40.43 $\pm$ 5.42	38.71 $\pm$ 13.2	46.8 $\pm$ 16.4	0.731 ^{NS}
	AFTER	Mean $\pm$ SD	38.23 $\pm$ 1.58	30.30 $\pm$ 14.5	36.87 $\pm$ 9.17	41.4 $\pm$ 8.1	0.331 ^{NS}
INR	BEFORE	Mean $\pm$ SD	0.96 $\pm$ 0.169	1.21 $\pm$ 0.484	1.40 $\pm$ 1.066	1.28 $\pm$ 0.4	0.804 ^{NS}
	AFTER	Mean $\pm$ SD	1.08 $\pm$ 0.126	1.14 $\pm$ 0.360	1.09 $\pm$ 0.385	1.39 $\pm$ 0.2	0.600 ^{NS}
FIB.	BEFORE	Mean $\pm$ SD	222.5 $\pm$ 86.0	211.5 $\pm$ 46.2	228.2 $\pm$ 49.85	237.3 $\pm$ 25.7	0.863 ^{NS}
	AFTER	Mean $\pm$ SD	239.4 $\pm$ 108.9	236.8 $\pm$ 49.4	237.5 $\pm$ 61.9	251.0 $\pm$ 46.8	0.989 ^{NS}
D-DIMER	BEFORE	Mean $\pm$ SD	0.84 $\pm$ 0.200	1.41 $\pm$ 1.427	1.62 $\pm$ 2.202	4.26 $\pm$ 5.3	0.244 ^{NS}
	AFTER	Mean $\pm$ SD	0.48 $\pm$ 0.351	1.99 $\pm$ 2.177	1.65 $\pm$ 2.147	4.71 $\pm$ 5.4	0.161 ^{NS}

NS = not statistically significant; * $p < 0.05$; ** $p < 0.01$.

5. DISCUSSION

This study assessed changes in hematological and coagulation parameters before and after a single hemodialysis session in patients with advanced CKD. Significant differences were reported in several cellular indices, including WBC and neutrophil counts, RBC count, hemoglobin, and hematocrit. By contrast, platelet counts and most routine coagulation measurements showed small, statistically nonsignificant changes. Together, the findings suggest that immediate post-dialysis differences were more readily detected in circulating blood-cell indices than in the conventional coagulation tests evaluated here.

The increases in WBC and neutrophil counts are broadly consistent with published observations of leukocyte abnormalities in patients undergoing hemodialysis, including findings reported by Goyoneche Linares et al. [21] and mechanistic discussions by Lutz et al. [4]. Contact with the dialysis membrane and extracorporeal circuit can activate inflammatory and complement pathways, potentially affecting circulating leukocyte populations. However, the present study did not directly measure complement activation or inflammatory mediators, so these mechanisms remain possible explanations rather than demonstrated causes of the observed increases.

The increases in RBC count, hemoglobin, and hematocrit resemble previously reported pre- and post-hemodialysis differences [22, 23]. One plausible explanation is hemoconcentration resulting from fluid removal by ultrafiltration. Such changes would reflect alterations in circulating plasma volume rather than rapid new erythrocyte production. The lack of significant differences in MCV, MCHC, RDW-CV, and RDW-SD is also compatible with little acute change in erythrocyte morphology. Without documented ultrafiltration volumes and fluid-balance measurements, however, the explanation cannot be tested directly in this study.

Platelet count showed a small, nonsignificant increase after hemodialysis, while MPV, PDW, and PCT remained statistically unchanged. Published studies have described variable platelet responses during dialysis and have highlighted

both platelet activation and altered adhesion to extracorporeal surfaces [9, 23]. The absence of a significant count change does not exclude qualitative platelet dysfunction. Differences among dialysis membranes, anticoagulation regimens, patient characteristics, and fluid removal may contribute to heterogeneous findings. Previous reviews have similarly emphasized the complexity of platelet dysfunction in kidney failure [3, 24].

The coagulation results showed modest decreases in PT and APTT and slight increases in fibrinogen and D-dimer, but none of these differences reached statistical significance. Reports from other dialysis settings have described variable coagulation-test responses [9, 25]. Variation in heparin dose, administration time, specimen-collection timing, and the effects of ultrafiltration could plausibly influence these measurements. Because those treatment details were not included in the present dataset, the mechanisms underlying the observed differences cannot be determined.

Small post-dialysis increases in fibrinogen and D-dimer could reflect a combination of hemoconcentration, inflammatory activity, coagulation, and fibrinolytic responses to extracorporeal circulation. Nevertheless, the reported differences were nonsignificant, and the study did not measure direct markers of thrombin generation or platelet activation. It would therefore be inappropriate to interpret these findings as evidence of a clinically meaningful post-dialysis hypercoagulable state [9, 10].

Overall, the findings indicate selective acute changes in circulating hematological parameters after one dialysis session, while platelet indices and conventional coagulation tests were relatively stable in this sample. The increases in leukocyte and neutrophil counts may be related to transient inflammatory responses; the rises in RBC count, hemoglobin, and hematocrit may be associated with ultrafiltration-related hemoconcentration. These explanations remain hypotheses because the study did not directly quantify the relevant mediators or dialysis-fluid balance. Larger studies with more detailed treatment data and additional measures of platelet activation, coagulation, and fibrinolysis are needed.

6. CONCLUSION

The present observational study identified immediate changes in selected hematological indices following a single hemodialysis session in patients with advanced CKD. WBC and neutrophil counts, RBC count, hemoglobin, and hematocrit increased significantly in the reported paired analyses. Potential explanations include inflammatory responses to extracorporeal circulation and hemoconcentration following fluid removal, although these mechanisms were not measured directly.

Platelet count and most measured red-cell and platelet indices did not change significantly during the observation period. Routine coagulation markers, including PT, APTT, INR, fibrinogen, and D-dimer, also showed no statistically significant pre- to post-dialysis differences. These results describe the response within this particular study population and should not be interpreted as evidence that hemodialysis has no effect on hemostatic function.

In summary, a single hemodialysis session was associated with selective changes in blood-cell measurements but limited detectable changes in conventional coagulation parameters in this sample. Further studies with larger cohorts, standardized anticoagulation and sampling protocols, detailed ultrafiltration records, and longitudinal follow-up are required to clarify the physiological mechanisms, longer-term effects, and clinical relevance of these findings.

7. RECOMMENDATIONS

1. Studies with larger sample sizes and complete paired laboratory observations are needed to assess the reproducibility and precision of these findings.
2. Longitudinal studies should assess whether acute laboratory changes persist or accumulate across repeated hemodialysis sessions.
3. Future investigations should consider thromboelastography (TEG) and rotational thromboelastometry (ROTEM) to evaluate clot dynamics alongside conventional coagulation assays.
4. Well-designed studies should examine the relationships between inflammatory markers and coagulation parameters in patients receiving hemodialysis.

Author contributions

All five authors contributed equally to the conception, design, investigation, analysis, manuscript preparation, and critical review of this work. All authors reviewed and approved the final manuscript.

Data availability

The study generated paired, participant-level laboratory data that are not publicly available because they contain sensitive health information. Requests for access to de-identified data may be directed to the corresponding author and are

subject to applicable ethical and institutional approval requirements.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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