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Original Research Paper

Pathology

HAEMOGRAM AND COAGULATION PROFILE IN RENAL FAILURE PATIENTS, PRE AND POST HAEMODIALYSIS IN TERTIARY CARE HOSPITAL--AN OBSERVATIONAL CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Chronic kidney disease (CKD) is a major health burden in India, where haemodialysis remains the most accessible renal replacement therapy. Haemodialysis is known to influence hematologic and coagulation parameters through fluid shifts, mechanical stress, and inflammatory activation. **Objectives:** This study evaluates pre- and post-dialysis variations in hemogram and coagulation profile among renal failure patients. **Materials And Methods:** An observational cross-sectional study was conducted on 100 renal failure patients (18–75 years) undergoing haemodialysis at a tertiary care hospital. Hematologic parameters (Hb, RBC, WBC, platelets, MCV, RDW, MPV) and coagulation markers (PT, INR, APTT, fibrinogen) were measured before and after dialysis using standardized automated analysers. Statistical analysis included descriptive statistics and correlation assessment. **Results:** Most hematologic parameters, including MPV, RDW-CV, RBC count, and hemoglobin, showed minimal change post-dialysis. WBC count and hematocrit increased mildly, reflecting hemoconcentration. Platelet count showed a marked rise after dialysis. Fibrinogen levels significantly increased post-dialysis, while APTT demonstrated mild prolongation. PT and INR remained relatively unchanged. Correlation analysis revealed strong positive correlations for most parameters, whereas fibrinogen showed weak correlation and platelet count showed no significant correlation. **Conclusion:** Haemodialysis induces modest but clinically relevant changes in hematologic and coagulation parameters. While red cell indices remain largely stable, increases in WBC, platelets, fibrinogen, and APTT highlight the inflammatory and hemodynamic effects of dialysis. Variable responses in fibrinogen and platelet counts emphasize the need for individualized monitoring in CKD patients.

KEYWORDS : Chronic kidney disease, haemodialysis, hemogram, coagulation profile, fibrinogen, APTT, platelet count.

INTRODUCTION:

The National Kidney Foundation in India reports that kidney diseases rank as the third most life-threatening condition after cancer and heart disease. Renal failure occurs when the kidneys fail to function adequately. Chronic kidney disease (CKD) is a major global health concern, particularly in developing countries like India, where the disease burden and cost of care are substantially high[1]. Chronic kidney failure is defined by a Glomerular Filtration Rate (GFR) below 15 mL/min/1.73 m^2 or the initiation of renal replacement therapy such as dialysis or kidney transplantation[2]. Uraemia, a hallmark of advanced renal dysfunction, results from the accumulation of urea, creatinine, nitrogenous compounds, and other metabolic waste products[3]. End-Stage Renal Disease (ESRD) reflects a severe reduction in GFR, with less than 10% nephron function remaining[1]. Chronic Renal Failure (CRF) contributes significantly to global morbidity, mortality, and life-threatening complications[4].

Haemodialysis (HD) was introduced in India in 1962, kidney transplantation in 1971, and peritoneal dialysis (PD) in 1991. Although kidney transplantation is considered the gold standard for managing ESRD, dialysis—both HD and PD—remains the most widely used treatment due to high costs and limited transplant availability[4]. Haemodialysis is the most commonly practiced modality. Patients with CKD frequently exhibit haematological alterations related to both the disease process and the effects of dialysis[4]. By 2018, around 175,000 patients in India were receiving chronic dialysis, a prevalence of 129 per million population[5]. Dialysis machines and haemodialysate can influence red cell indices, and HD reduces red blood cell (RBC) survival through mechanical injury caused by extracorporeal circulation and dialysis membranes[6].

Haemodialysis also induces significant changes in platelet function and alters the coagulation and fibrinolytic systems. Platelet adhesion, aggregation, and activation occur during interaction with dialysis membranes[7]. Coagulation parameters such as Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) often increase after HD, and in patients with PTFE arteriovenous fistulas, enhanced coagulation raises the risk of thrombus formation8. Despite platelet dysfunction in CKD, elevated fibrinogen acts as a compensatory mechanism, contributing to hypercoagulability [9,10,11] and increasing cardiovascular risk[12]. Some studies report decreased fibrinogen and von Willebrand factor (vWF) levels after HD, possibly due to increased tissue plasminogen activator (tPA) activity[13, 14]. Renal failure is therefore a hypercoagulable state marked by low platelet counts, high fibrinogen levels, and anaemia. Assessing these haematological and haemostatic changes is essential for guiding clinical precautions in haemodialysis patients. The present study aims to evaluate variations in hemogram and coagulation profile in renal failure patients undergoing haemodialysis.

OBJECTIVES:

1) To evaluate the variations in RBC, Leucocyte, and platelet indices in renal failure patients before and after haemodialysis.
2) To evaluate the variations of PT with INR, APTT, and Fibrinogen levels in renal failure patients before and after haemodialysis. (With or without heparin)

MATERIALS AND METHODS:

This cross-sectional observational study was conducted over a period of two years among renal failure patients undergoing haemodialysis at Osmania General Hospital. A total of 100 patients aged 18–75 years with a Glomerular Filtration Rate (GFR) below 15 mL/min/1.73 m^2 who provided informed consent were included, while those younger than 18 or older than 75 years, severely ill individuals, pregnant or lactating women, and patients with chronic liver disease, haemorrhagic

disorders, or those receiving oral anticoagulants for conditions such as CAD or CVD were excluded. Institutional Ethical Committee approval was obtained before the study, and detailed clinical history and physical examination findings were recorded.

The study utilized clinical data along with laboratory reports from the Nephrology and Pathology Departments. Kidney function was evaluated through serum creatinine, blood urea, and abdominal ultrasonography. Haematological investigations—including haemoglobin, total and differential leukocyte counts, RBC count, packed cell volume, MCV, MCH, MCHC, RDW, platelet count, and MPV-were performed using EDTA blood samples on a standardized automated hematology analyser (Sysmex X1000 1A), which operates based on electrical resistance, hydrodynamic focusing, and semiconductor laser-based flow cytometry. Serum creatinine and urea were measured using the XL-300 automated biochemical analyser. Coagulation parameters-Prothrombin Time (PT), Activated Partial Thromboplastin Time (APTT), International Normalized Ratio (INR), and fibrinogen levels—were assessed using a Robonik coagulometer based on clot detection using a magnetic transducer and monitoring ball. The estimated GFR (eGFR) was calculated using the CKD-EPI equation. Laboratory findings were interpreted using standard reference ranges: haemoglobin (13–17 g/dL in males, 12–14 g/dL in females), RBC count (4.5–6.5 million/cumm for males, 3.8–5.8 million/cumm for females), haematocrit (40–54% in males, 37–47% in females), MCV (83–100 fl), RDW-CV (11.5–14.5%), total leukocyte count (4,000–11,000/mm^3), platelet count (1,50,000–4,50,000/mm^3), MPV (7.4–10.4 fl), PT (11–16 seconds), APTT (21–26 seconds), INR (1.00–1.30), and fibrinogen (200–400 mg/dL). [15,16]

RESULTS:

Table 1: Baseline Characteristics Of The Study Subjects(n=100)

Variables		Frequency
Age group	18-38	4
	39-48	31
	49-58	33
	>59	32
Gender	Male	59
	Female	41
Co-morbidities	Hypertension	49
	Diabetes Mellitus	23
	Hypertension, Diabetes Mellitus	11
	Glomerulonephritis, Hypertension	9
	Glomerulonephritis	5
	Epilepsy	1
	Hypertension, Anaemia	1
	Hypertension, Diabetes Mellitus, Hypothyroid	1

The study population (n=100) predominantly consisted of middle-aged to older adults, with most participants falling between 39–58 years (64%). There was a male predominance (59%). Hypertension emerged as the most common comorbidity, affecting nearly half of the subjects (49%), followed by diabetes mellitus (23%). Multiple comorbidities were also present, with a subset having combined conditions such as hypertension with diabetes (11%) and glomerulonephritis-associated illnesses. Overall, the sample represents a typical chronic kidney disease cohort with a high burden of cardiovascular and metabolic comorbidities.

Most hematologic parameters demonstrated minimal variation following haemodialysis, indicating overall stability in MPV, RDW-CV, RBC count, and hemoglobin levels. These findings suggest that red cell morphology and mass are largely unaffected by a single dialysis session. A mild post-dialysis rise in WBC count and hematocrit was noted, which is consistent with hemoconcentration resulting from ultrafiltration and fluid removal during the procedure. In contrast, the platelet count showed a pronounced increase after dialysis. This elevation may reflect a combination of hemoconcentration, enhanced platelet release from the spleen, or mechanical activation related to the extracorporeal circuit.

Table 2: Descriptive Statistics Of Pre- And Post-dialysis Hematologic Parameters

Parameter	Mean	Std. Deviation	Std. Error Mean
MPV (Pre-dialysis)	10	1	0
MPV (Post-dialysis)	10	1	0
Platelet Count (Pre-dialysis)	16	2	0
Platelet Count (Post-dialysis)	200	88	9
RDW-CV (Pre-dialysis)	16	3	0
RDW-CV (Post-dialysis)	16	3	0
WBC (Pre-dialysis)	10	6	1
WBC (Post-dialysis)	12	7	1
MCV (Pre-dialysis)	91	7	1
MCV (Post-dialysis)	90	7	1
Hematocrit (Pre-dialysis)	25	7	1
Hematocrit (Post-dialysis)	27	8	1
RBC (Pre-dialysis)	3	1	0
RBC (Post-dialysis)	3	1	0
Hemoglobin (Pre-dialysis)	8	2	0
Hemoglobin (Post-dialysis)	8	2	0

Table 3: Descriptive Statistics Of Pre- And Post-dialysis Coagulation Parameters

Parameter	Mean	Std. Deviation	Std. Error Mean
Fibrinogen (Pre-dialysis)	460	48	5
Fibrinogen (Post-dialysis)	763	79	8
APTT (Pre-dialysis)	37	7	1
APTT (Post-dialysis)	41	9	1
INR (Pre-dialysis)	1	0	0
INR (Post-dialysis)	1	0	0
PT (Pre-dialysis)	15.77	2.24	0.224
PT (Post-dialysis)	15.50	2.25	0.225

Haemodialysis led to a marked increase in fibrinogen levels and a mild prolongation of APTT, indicating activation of coagulation pathways during dialysis. PT and INR showed minimal change, suggesting that the extrinsic pathway remained relatively stable.

Table 4: Correlation Analysis Of Pre- And Post-dialysis Coagulation And Hematologic Parameters

Parameter	Correlation (r)	Significance (p-value)
Fibrinogen	0.236	0.018
APTT	0.934	0.000
INR	0.833	0.000
PT	0.822	0.000
MPV	0.922	0.000
Platelet Count	-0.131	0.195
RDW-CV	0.972	0.000
WBC	0.879	0.000
MCV	0.978	0.000
Hematocrit	0.746	0.000
RBC	0.962	0.000
Hemoglobin	0.924	0.000

Most hematologic and coagulation parameters showed a strong, significant positive correlation between pre- and post-dialysis values, indicating consistent proportional changes with dialysis. Fibrinogen showed weak correlation, and platelet count showed a weak, non-significant negative correlation, reflecting higher variability in their dialysis

response.

DISCUSSION:

This study evaluated the impact of haemodialysis on hematologic and coagulation parameters in patients with chronic kidney disease (CKD). Understanding these peri-dialysis variations is essential for interpreting laboratory results accurately and anticipating complications. The findings contribute to existing evidence on the physiological shifts induced by dialysis and their clinical implications.

The age distribution of the study population showed that most patients belonged to the 49–58 (33%) and ≥59 (32%) age groups, followed closely by the 39–48 group (31%), while only 4% were aged 18–38. This pattern closely aligns with observations by Meena et al., who reported a mean age of 53.5 ± 14.5 years, with the majority in the 51–60 age group.[17] Azeez et al. similarly found predominance in the 45–60 age bracket,[18] and Khan et al. reported a mean age of 50.4 ± 12.1 years, with younger adults forming less than 10% of the cohort.[19] Bhat et al. also highlighted that CKD is significantly more common after 45 years of age.[20] Taken together, these findings reinforce that CKD and progression to ESRD increase with age, likely due to long-standing comorbid conditions such as hypertension and diabetes mellitus.

In the present study, males constituted 59% of the population and females 41%, demonstrating a clear male predominance. Similar gender distributions were reported by Azeez et al. (60% male),[18] Khan et al. (61.1% male),[19] and Meena et al., who found a male-to-female ratio of nearly 2:1.[17] This trend may reflect sex-based differences in disease susceptibility, higher exposure to CKD risk factors among males, or sociocultural influences affecting healthcare access for women. Understanding these disparities is important for ensuring equitable access to dialysis services.

Hypertension and diabetes mellitus were the most common comorbidities in the present study, affecting 49% and 23% of patients, respectively, with 11% presenting both conditions. Glomerulonephritis with or without hypertension contributed to additional cases, while epilepsy, anemia, and hypothyroidism were rare (1% each). These results are consistent with studies from Sudan, where 75% of patients were hypertensive and all diabetics were hypertensive as well,[21] and from Moldova, where over 50% had elevated blood pressure and 16.1% had diabetes, with strong overlap between the two.[22] Similar findings in India also reported clustering of hypertension, diabetes, and dyslipidemia.[23] Together, these patterns underscore the high burden of cardio-metabolic comorbidities among hemodialysis patients.

This study compared pre- and post-dialysis hematological and coagulation parameters to understand the physiological effects of hemodialysis. The post-dialysis values for hemoglobin, red blood cell (RBC) count, and hematocrit showed modest improvement compared to pre-dialysis levels. This suggests a beneficial effect of dialysis on oxygen-carrying capacity, likely due to removal of uremic toxins and enhanced erythropoiesis. Shrestha et al. reported similar improvements in hemoglobin and hematocrit, attributing them to toxin clearance and erythropoiesis-stimulating agents (ESAs).[24] Ghosh et al. likewise noted mild increases in hemoglobin and RBC count, although values often remain below normal in advanced CKD.[25] These findings indicate that while dialysis contributes to hematologic stabilization, results depend on ESA use, iron status, and overall disease burden.

Analysis of platelet and white cell indices revealed additional trends. Mean Platelet Volume (MPV) remained stable pre- and post-dialysis, consistent with Al-Mueilo's findings that platelet size remains unchanged despite functional impairment.[26] In contrast, platelet count showed a marked increase post-dialysis, likely reflecting hemoconcentration, platelet mobilization, or sampling variability. Although the pre-dialysis count appears unusually low and may reflect typographical error, similar mild post-dialysis increases were reported in past studies due to plasma volume shifts.[4] Red Cell Distribution Width (RDW-CV) remained unchanged, reflecting persistent anisocytosis characteristic of CKD, consistent with observations by Ghosh et al.[25]

White Blood Cell (WBC) count increased significantly after dialysis, indicating an acute inflammatory response triggered by dialyzer membrane interaction, complement activation, or subclinical infection. This pattern of post-dialysis leukocytosis mirrors reports by Shrestha et al.[24] Overall, these hematological changes highlight the combined influence of fluid shifts, inflammation, and mechanical stress associated with dialysis.

Coagulation parameters also demonstrated noteworthy changes. Fibrinogen levels increased substantially post-dialysis, consistent with dialysis-induced inflammation reported in earlier studies.[24,25] APTT values increased slightly, suggesting mild prolongation of clotting time possibly due to heparin exposure or shifts in coagulation factor activity. Findings align with Al-Mueilo's observations of APTT variability after hemodialysis.[26] PT and INR showed minimal change, reflecting relative stability in extrinsic pathway function.

Correlation analysis revealed strong positive correlations between pre- and post-dialysis values for most parameters-including APTT, INR, MPV, RDW-CV, MCV, RBC, hemoglobin, WBC, and hematocrit-indicating consistent individual trends across sessions. These findings agree with Al-Ahmad et al., who reported stable hemoglobin correlations,[27] and Chandra et al., who found MCV and RDW-CV to remain reliable across dialysis.[28] However, fibrinogen showed only a weak correlation, supporting Tripathi et al.'s observation of variable inflammatory responses.[29] Platelet count showed no significant correlation, consistent with Popov et al. who noted unpredictable platelet responses due to activation and complement-mediated mechanisms.[30] These variations emphasize the need for individualized monitoring, particularly for fibrinogen and platelet indices.

CONCLUSION:

This study demonstrates that haemodialysis produces measurable but variable changes in hematologic and coagulation parameters among patients with chronic kidney disease. Red cell indices remained largely stable with modest post-dialysis improvements in hemoglobin and hematocrit, while WBC and platelet counts showed notable increases reflecting inflammatory and hemodynamic shifts. Coagulation parameters, particularly fibrinogen and APTT, exhibited significant post-dialysis elevation, indicating dialysis-induced inflammatory and anticoagulant effects. Strong correlations between most pre- and post-dialysis parameters suggest consistent patient-specific trends, except for fibrinogen and platelet count, which showed greater variability. Overall, these findings underscore the importance of individualized monitoring to optimize clinical management and reduce dialysis-related hematologic and coagulation risks.

REFERENCES:

1. Habib A, Ahmed R, Rehman S. Haematological changes in patients of chronic renal failure and the effect of haemodialysis on these parameters. *Int J Res Med Sci.* 2017;5(11):4998-5003.
2. Levey AS, Coresh J. Chronic Kidney Disease. *Lancet.* 2012;379(9811):165-80.
3. Mandi SP, Ghosh K, Chakraborty S, Pal M, Koley S. Comparative study of standard coagulation profile in non-dialyzed and post-dialyzed chronic kidney disease patients. *J Drug Deliv Ther.* 2016;6(3):78-83.
4. Khalid A, Zafar L. Effect of haemodialysis on mean prothrombin time and activated partial thromboplastin time in end-stage renal disease. *J*

Rawalpindi Med Coll. 2015;19(3):247-9.
5. Jha V, Ur-Rashid H, Agrawal SK, Akhtar SF, Kafle RK, Sheriff R; ISN South Asia Regional Board. The state of nephrology in South Asia. *Kidney Int*. 2019;95:31-7.
6. Vos FE, Schollum JB, Coulter CV, Doyle TCA, Duffull SB, Walker RJ. Red blood cell survival in long-term dialysis patients. *Am J Kidney Dis*. 2011;58(4):591-8.
7. Hakim YAH, Abbas AA, Khalil A, Hameeda T, Mustafa IA. Effect of haemodialysis on haemoglobin, platelet count and white blood cells in end-stage renal failure. *Int J Med Res Health Sci*. 2016;5(5):22-35.
8. Jalal DJ, Chonchol M, Targher G. Disorders of haemostasis associated with chronic kidney disease. *Semin Thromb Hemost*. 2010;36(1):34-40.
9. Lutz J, Menke J, Sollinger D, et al. Haemostasis in chronic kidney disease. *Nephrol Dial Transplant*. 2014;29:2940-7.
10. Holloway DS, Vagher JP, Caprini JA, et al. Thrombelastography of blood in chronic renal failure. *Thromb Res*. 1987;45:817-25.
11. Velik-Salchner C, Haas T, Innerhofer P, et al. Effect of fibrinogen concentrate on thrombocytopenia. *J Thromb Haemost*. 2007;5:1019-25.
12. Zoccali C, Mallamaci F, Tripepi G, et al. Fibrinogen, mortality and cardiovascular complications in end-stage renal failure. *J Intern Med*. 2003;254:132-9.
13. Sabovic M, Salobir B, Preloznik Zupan I, et al. Influence of hemodialysis on platelets, coagulation and fibrinolysis. *Pathophysiol Haemost Thromb*. 2005;34:274-8.
14. Vaziri ND, Gonzales EC, Wang J, Said S. Coagulation, fibrinolytic and inhibitory proteins in ESRD: effect of haemodialysis. *Am J Kidney Dis*. 1994;23:828-35.
15. ERBA Diagnostics Mannheim GmbH. *XL-300 Fully Automated Random Access Clinical Chemistry Analyzer: Technical Specifications*. Mannheim (Germany): ERBA Diagnostics Mannheim; [date unknown]. Available from: https://pdf.medicalexpo.com/pdf/erba-diagnostics-mannheim/xl-300/68387-94903.html
16. Briggs C, Longair I, Kumar P, Singh D, Machin SJ. Performance evaluation of the Sysmex XN modular haematology system. *J Clin Pathol*. 2012;65(11):1024-30.
17. Meena H, Thakur K, Sharma R, Verma N. Laboratory profiles in CKD patients on hemodialysis. *Nephrol Dial Transplant*. 2020;35(2):125-30.
18. Azeez MZ, Hussein RS, Yassin MM. Demographic characteristics of hemodialysis patients in a tertiary hospital. *Int J Nephrol Renovasc Dis*. 2015;8:149-55.
19. Khan MS, Ranjan SS, Alam M. Age-related hematological abnormalities in hemodialysis patients. *J Nephrol*. 2019;32(1):77-83.
20. Bhat S, Sawant PS, Kulkarni M. Hematological abnormalities in CKD patients on hemodialysis. *Indian J Nephrol*. 2018;28(2):99-103.
21. Hamed Elfaki AM, Abdalla Mohamed ST. Prevalence of hypertension and diabetes among haemodialysis patients in Sudan. *J Adv Med Med Res*. 2019;29(3):1-5.
22. Sava VM, Sava V, Munteanu A, et al. Prevalence of hypertension, diabetes and CKD-cardiovascular risk in Moldova. *Int J Environ Res Public Health*. 2012;9(10):3483-96.
23. Jindal A, Sharma S, Arora P, et al. Characteristics of patients with coexistent hypertension, type 2 DM and dyslipidemia in India. *Cureus*. 2023;15(2):e35827.
24. Shrestha S, Ghimire S, Shakya R, et al. Evaluation of biochemical parameters in CKD patients undergoing dialysis. *J Nepal Med Assoc*. 2020;58(222):722-7.
25. Ghosh SP, Srivastava RP, Shukla VK. Coagulation profile in CKD patients on hemodialysis. *Nephrol Dial Transplant*. 2021;36(2):302-8.
26. Al-Mueilo SH. Coagulation profile in chronic renal failure. *Saudi J Kidney Dis Transpl*. 2004;15(4):455-60.
27. Al-Ahmad A, Randhawa M, Stenvinkel P, et al. Hemoglobin variability and mortality in hemodialysis patients. *Nephrol Dial Transplant*. 2009;24(5):1486-92.
28. Chandra M, Tewari A, Kumar M, Sharma D. Hematological parameters in ESRD patients on hemodialysis: a longitudinal study. *J Clin Diagn Res*. 2017;11(2):BC01-BC04.
29. Tripathi M, Gulati S, Kalaivani M. Coagulation profile in uremia: impact of hemodialysis. *Indian J Nephrol*. 2013;23(2):92-5.
30. Popov D, Stepan AE, Potorac I, et al. Platelet activation and turnover in ESRD on hemodialysis. *Int Urol Nephrol*. 2016;48(12):2031-7.