

Renal and Metabolic Biomarker Profiles in Dialysis vs. Non-Dialysis CKD Patients: A Cross-Sectional Study

Assia Ramdan^{1*} , Adel Mahfoud² 

¹Department of Basic and General Sciences, Faculty of Dentistry, University of Zawia, Zawia, Libya

²Department of Medical Laboratory, Faculty of Medical Technology, University of Zawia, Zawia, Libya

adelmahfoud@zu.edu.ly

Keywords:

Chronic Kidney Disease,
Electrolytes
Urea, Creatinine
Uric Acid,
Glomerular Filtration Rate,
Dialysis.

ABSTRACT

This study investigated key renal and biochemical parameters in Chronic Kidney Disease (CKD) patients by comparing a cohort of 147 non-dialysis patients with 147 patients undergoing dialysis. Key findings indicate that while both groups exhibited elevated urea and creatinine levels, urea was lower in dialysis patients, whereas creatinine was significantly higher. Blood pressure was minimally increased in both groups, but was higher in the non-dialysis cohort. Uric acid levels were notably lower in patients on dialysis. Regarding electrolytes, potassium levels were higher in the dialysis group, while sodium levels remained normal and stable across both cohorts. Serum phosphorus was elevated in both groups, with slightly higher levels in non-dialysis patients. A significant difference was observed in serum calcium, which was normal in non-dialysis patients but below the normal range in the dialysis group. Both groups also showed elevated fasting blood sugar. As expected, Glomerular Filtration Rate (GFR) was sharply decreased in all patients, most severely in the dialysis group, consistent with end-stage renal disease. The results underscore the profound metabolic and physiological changes in CKD patients. The study concludes that enhancing dialysis efficiency and providing comprehensive patient education on medication, diet, and lifestyle are critical for improving quality of life, reducing complications, and lowering healthcare costs associated with CKD management.

Introduction

Chronic Kidney Disease (CKD) is becoming a significant public health concern. It is characterized by irreversible deterioration of renal function that eventually leads to end-stage renal disease (ESRD). At that point, patients need renal replacement treatment like dialysis or kidney transplantation [1]. Soon, the prevalence of CKD is predicted to grow, with the anticipation that more than two million individuals will be in demand for renal replacement therapy by 2030 [2]. The primary role of the kidneys is the removal of toxic byproducts and excess fluid from the body; in addition, they are essential for maintaining acid-base homeostasis and electrolyte balance [3]. Renal function can be determined through blood tests, with commonly used biomarkers such as creatinine, uric acid, electrolytes, and urea [4]. Glomerular filtration rate (GFR) is widely known as the best parameter for determining kidney function [5].

GFR gradually falls in CKD, which leads to the accumulation of urea, creatinine, and other wastes in the blood [6]. Based on the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, a GFR below 60 mL/minute/1.73 m² is a sign of CKD [6]. Beyond this point, serious complication begins to appear, such as electrolyte disturbance, mineral bone disease, anemia, metabolic acidosis, and atherosclerosis [7]. A reduction in GFR can also lead to high levels of uric acid in the blood, a condition known as hyperuricemia (HUA). This condition is frequently seen in people with CKD [8,9]. Additionally, abnormalities in calcium and phosphorus are commonly seen in CKD patients.

Renal osteodystrophy, a form of metabolic bone disorder often associated with CKD [10]. Estimates suggest that 25%-40% of patients with diabetes and hypertension are expected to develop chronic kidney disease (CKD) [11]. Hypertension is one of the most significant risk factors for developing kidney failure. High blood pressure can cause vascular damage within the kidneys, thereby reducing their ability to excrete waste products. Accumulation of wastes in the body fluids contributes to elevated blood pressure, which in turn leads to the development of end-stage renal disease (ESRD) [12,13]. Moreover, kidney disease is the most frequent complication observed in patients with diabetes, with approximately 50% of diabetic patients suffering from kidney disease throughout their life [14]. When kidneys are dysfunctional, wastes such as urea, creatinine, and free water are eliminated from the blood through one replacement treatment called haemodialysis [15].

The process of haemodialysis can be an effective method for the removal of harmful wastes from the blood, which helps patients live longer [16]. Proper dialysis can reduce the occurrence of complications and care expenses [17]. However, when hemodialysis is insufficient, the morbidity and mortality will be higher among dialysis patients [18]. Mineral metabolism, for example, is a critical indicator of morbidity and mortality among patients undergoing dialysis [19]. There are factors affecting dialysis efficiency, which include appropriate scheduling of dialysis, patient education, suitable dialyzer, and the meal habits of

patients [20]. High-protein diet, particularly from meat and certain vegetables, can increase the renal load and lead to the accumulation of urea and creatinine in the blood [21].

Methods

This cross-sectional study was conducted at Zawia Kidney Hospital in Zawia, Libya, from March 1st to October 31st, 2024. A total of 294 Libyan patients participated in the study, divided into two distinct groups. Group A comprised 147 renal dialysis patients (60 males, 87 females), who were recruited from the hospital's dialysis unit randomly. Group B consisted of 147 chronic kidney disease (CKD) non-dialysis patients (75 males, 72 females), who were randomly selected for follow-up at the Outpatient Department (OPD) clinic of Zawia Kidney Hospital. The age range for CKD patients in Group B was 34 to 80 years, while renal dialysis patients in Group A ranged from 32 to 75 years.

Data Collection and Questionnaire

Data were collected using a standardized interview questionnaire. This questionnaire encompassed demographic information such as name, gender, age, and weight. Clinical history details included the presence of heart disease, bone disorders, and other comorbidities, as well as the type of treatment received, duration of diabetes mellitus, duration of dialysis, and current medications. Additionally, the study involved the analysis of various biochemical parameters, including urea, creatinine, calcium (Ca), sodium (Na), potassium (K), phosphorus (P), uric acid, and fasting blood sugar analyses (FBS).

Blood Sample Collection and Processing

Blood samples were collected from both groups. For each participant, samples were drawn after dialysis (for Group A) and randomly for Group B. These samples were collected into clean, dry tubes without anticoagulant for serum separation. Subsequently, the samples were centrifuged at 2000 rpm for 15 minutes. The clear, non-hemolyzed supernatant serum was then utilized for comprehensive biochemical analysis, which includes kidney function tests, blood glucose levels, and electrolyte measurements. All analyses were performed using an automated Hitachi chemistry analyzer.

Ethical Approval

Ethical approval for this study, including the collection and processing of data from the medical laboratory unit, was obtained from Zawia Kidney Hospital.

Data Management and Analysis

All collected data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) software, version 21 (IBM SPSS, NY, USA). Continuous variables were presented as Means $\pm$ Standard Error of the Mean (SEM). Statistical significance was defined as a p-value less than 0.05 ($P < 0.05$) or less than 0.01 ($P < 0.01$).

Results

This cross-sectional investigation was undertaken at the Zawia Kidney Hospital in Zawia city, Libya. The study period extended from February 1st, 2024, to October 31st, 2024, where 294 patients participated. The cohort was divided into two distinct groups for analysis. The first group included 147 renal dialysis patients (60 males, 87 females), where 40.82% were male and 59.18% were female. The second group consisted of 147 patients with chronic kidney disease (CKD) not undergoing dialysis (75 males, 72 females) 51% of them were male and 49% were female, as shown below in (Table 1). The non-dialysis CKD Patients group had a mean age of 59.2857 ± 13.07032 years $SD \pm$ and a mean weight of 82.0204 ± 11.67528 Kg, which was higher than the age mean and the weight mean of the dialysis patients group, 49.9796 ± 13.94407 years and 69.3061 ± 15.17047 kg, respectively (Table 2). The clinical and biochemical parameters for both the non-dialysis patient group and dialysis patient groups are summarized in (Table 3). The mean levels of blood pressure were higher in the non-dialysis group at 135/88 mmHg, compared to 130/75 mmHg in the dialysis group.

Regarding kidney function, serum urea levels were elevated in both cohorts, though higher in non-dialysis patients (11.8823 ± 0.6516 mmol/L) than in dialysis patients (11.1725 ± 0.4521 mmol/L). A more pronounced difference was observed in serum creatinine, which was significantly higher in the dialysis cohort (1161.65 ± 210.2 μ mol/L) versus the non-dialysis cohort (243.188 ± 26.3087 μ mol/L). Analysis of serum electrolytes and minerals indicated that mean calcium, sodium, and phosphorus levels were marginally higher in the non-dialysis group. In contrast, mean potassium levels were substantially more elevated in the dialysis group (1.5672 ± 0.2409 mmol/L) compared to the non-dialysis group (1.3876 ± 0.0611 mmol/L). The correlation of Creatinine and Calcium was statistically significant in both study

groups ($p = 0.000$, $p = 0.018$) respectively, while the correlation of blood pressure, Urea, K, Na, and P in both groups was statistically not significant ($p = 0.214$, $p = 0.373$, $p = 0.472$, $p = 0.711$, $p = 0.711$, $p = 0.078$) respectively, as shown in (Table 4).

Table 5 summarizes the mean serum biochemical concentrations and estimated glomerular filtration rate (e-GFR) for the non-dialysis CKD patients' group and dialysis patients' group. The mean uric acid level was observed to be $496.524 \pm 199.611 \mu\text{mol/L}$ in non-dialysis patients and $418.222 \pm 77.1617 \mu\text{mol/L}$ in the dialysis cohort. Fasting blood sugar (FBS) levels averaged $8.9446 \pm 4.5501 \text{ mmol/L}$ in the non-dialysis group and $7.9648 \pm 4.4004 \text{ mmol/L}$ in the dialysis group. Finally, the mean e-GFR values were $37.94 \pm 17.23 \text{ mL/min/1.73m}^2$ for non-dialysis patients and $14.93 \pm 41.59 \text{ mL/min/1.73m}^2$ for dialysis patients. Analysis of the data presented in (Table 6) reveals statistically significant differences between the non-dialysis patients group and dialysis patients group for several key biomarkers. Specifically, significant correlations were observed for serum uric acid ($p = 0.012$). Furthermore, a highly significant difference was found in e-GFR between the two groups ($p = 0.001$). In contrast, the correlations for serum biochemical markers of fasting blood sugar (BS), did not reach statistical significance. (Table 7) presents the distribution of non-dialysis patients according to their disease stage. The largest proportion of patients (63.26%) was classified as having Stage 3 CKD, which is characterized by an estimated glomerular filtration rate (e-GFR) between 30 and 60 mL/min/1.73m². A smaller percentage of patients (26.48%) were in Stage 4. This distribution is likely attributable to the inclusion criteria, which specified the group of pre-dialysis CKD patients

Table 1. The frequency distribution of gender (Non-dialysis and Dialysis patients) (n=294)

Gender	Non-dialysis patients (N)	Non-dialysis patients %	Dialysis patients (N)	Dialysis patients %
Male	75	51	60	40.82
Female	72	49	87	59.18

Table 2: The frequency distribution of Age and weight (Non-dialysis and Dialysis patients)

Participant groups		Age	Weight
Non-dialysis patients	Mean	59.2857	82.0204
	N	147	147
	SD±	13.07032	11.67528
Dialysis patients	Mean	49.9796	69.3061
	N	147	147
	SD±	13.94407	15.69263
Total	Mean	54.6327	75.6633
	N	294	294
	SD±	14.23470	15.17047

Table 3. Comparison of the mean of blood pressure, kidney function, and electrolytes among non-dialysis and dialysis patients

Variables	Patients groups		N	MEAN	SD±
Blood Pressure	Non-dialysis patients	Systolic	147	135.0000	2.79699
		Diastolic	147	88.000	2.89323
	Dialysis patients	Systolic	147	130.0000	2.85714
		Diastolic	147	75.000	2.93201
Urea mmol/L	Non-dialysis patients		147	11.8823	0.6516
	Dialysis patients		147	11.1725	0.4521
Creatinine $\mu\text{mol/L}$	Non-dialysis Patients		147	243.188	26.3087
	Dialysis patients		147	1161.65	210.2
Ca mmol/l	Non-dialysis Patients		147	2.1956	0.0501
	Dialysis patients		147	2.0427	0.0381
K mmol/l	Non-dialysis Patients		147	1.3876	0.0611
	Dialysis patients		147	1.5672	0.2409

Na mmol/L	Non-dialysis Patients	147	139.4694	0.7262
	Dialysis patients	147	139.1224	0.588
P mmol/L	Non-dialysis Patients	147	1.8794	0.3914
	Dialysis patients	147	1.71	0.5394

Table 4: Correlation of blood pressure, urea, creatinine, and electrolytes of serum biochemical test values in non-dialysis and dialysis patients.

The variable	t value	d.f	p-value
Blood Pressure	1.251	294	0.214
Urea	0.895	294	0.373
Creatinine	4.336	294	0.000
Ca	2.413	294	0.018
K	0.723	294	0.472
Na	0.371	294	0.711
P	1.799	294	0.078

Table :5 Comparison of mean values of Uric acid, and BS serum biochemical concentration, and e-GFR in Non-dialysis and dialysis patients.

Variable	Stages	N	Mean	SD±
Uric acid μmol/L	Non-dialysis patients	147	496.524	199.611
	Dialysis patients	147	418.222	77.1617
BS mmol/L	Non-dialysis patients	147	8.9446	4.5501
	Dialysis patients	147	7.9648	4.4004
e-GFR mL/min/1.73m ²	Non-dialysis patients	147	37.9398	17.2362
	Dialysis patients	147	14.9339	6.5914

Table 6: correlations of Uric acid and BS of serum biochemical tests values and e-GFR stages among non-dialysis and dialysis patients.

Variable	t-value	d.f	p-value
Uric Acid	2.561	294	0.012
BS	1.083	294	0.281
e-GFR	3.577	294	0.001

Table 7: Stages of kidney disease according to "e-GFR" in the Non-dialysis patient group.

e-GFR STAGES	Frequency	%	Mean
Stage 1	3	2.04	107.3
Stage 2	12	8.16	66.2
Stage 3	93	63.26	86.8
Stage 4	39	26.48	35.9

Discussion

The present study involved 294 participants, divided into two groups: the renal dialysis patients group included 147 patients, and the non-dialysis patients group included 147 patients. The non-dialysis Patients group had a mean age of 59.2857, while the dialysis patients group had a mean age of 49.9796. These findings align with previous studies showing that most people affected by CKD are aged 40 to 60 years [16,22]. The present study showed a slight elevation in blood pressure levels in both non-dialysis and dialysis patients. The blood pressure in non-dialysis patients was higher than in dialysis patients; however, the difference between the two study groups was not significant. In fact, Hypertension often targets the kidneys, and prolonged exposure to high blood pressure can contribute to early kidney damage [23]. It has been confirmed that 85% of hemodialysis patients suffer from hypertension [24]. In addition, a previous study showed that systolic blood pressure is strongly linked with cardiovascular mortality in dialysis patients [25].

Although the urea and creatinine mean values were higher than the normal range in both population samples, the urea mean value in the current research was lower in dialysis patients than in non-dialysis patients. This finding is consistent with previously reported studies, which have concluded that hemodialysis significantly reduced the urea and creatinine levels in patients with kidney failure, but these

constituents after dialysis are still elevated relative to those of the healthy group [16,26,27,28]. On the other hand, the mean value of creatinine was significantly higher in dialysis patients than in non-dialysis patients in the current study. The elevated creatinine levels in dialysis patients in this study can be attributed to several factors: First, creatinine production primarily reflects lean muscle mass. Second, consuming cooked meat can raise serum creatinine levels, as cooking converts the creatine in meat to creatinine. Third, Certain medications, particularly the psychoactive phenacemide, can increase the rate of creatinine production [29].

Regarding uric acid, it has been confirmed that glomerular filtration rate decline leads to hyperuricemia [30,31]. The present study indicated a significant reduction in uric acid levels in dialysis patients compared to non-dialysis patients. This finding agrees with a previous study, which established that the level of uric acid in dialysis patients decreases after dialysis [32]. On the other hand, some studies have revealed that the level of serum uric acid increases to above normal values after dialysis. This is attributed to ischemic episodes during dialysis [33]. Additionally, a prior study showed an increased prevalence of hyperuricemia as the duration of dialysis treatment increases [34]. Many earlier studies confirmed that serum potassium levels after dialysis were lower than before dialysis [35,36]. However, these findings are inconsistent with ours, which revealed that the mean potassium value was higher in dialysis patients compared to non-dialysis patients. The high potassium levels in dialysis patients may be a result of their dietary habits. In the same context, the present study showed no significant difference in serum sodium concentration between dialysis patients and non-dialysis patients, with levels remaining within the normal range for both study groups. Previous studies, on the other hand, reported a significant difference in serum sodium concentration before and after dialysis [16,37]. Additionally, many studies observed that higher serum sodium levels in post-dialysis patients compared to pre-dialysis patients [35,38]. The discrepancy between the prior studies and our results likely stems from the influence of intradialytic dietary salt intake and intradialytic sodium removal on serum sodium level [39].

In terms of mineral parameters, a strong connection has been confirmed between serum phosphorus levels and an increased risk of CKD progression [40]. A previous study has shown that standard dialysis treatments cannot eliminate all ingested phosphorus, especially from a protein-rich diet designed to avoid malnutrition [41]. Furthermore, a prior study has indicated that higher phosphorus levels were observed in patients with a longer history of dialysis treatment [42].

The current study, on the other hand, has established that serum phosphorus concentrations are elevated in both dialysis and non-dialysis patient groups, and the level of phosphorus was slightly higher in non-dialysis patients relative to dialysis patients. An Interesting observation was recorded in the present study regarding the serum calcium level; we found a significant difference in serum calcium concentration between the two study groups. The serum calcium level was within the normal range in non-dialysis patients, but was lower than the normal level in CKD patients undergoing dialysis. These results align with previous research, which has reported that serum calcium levels remain within the normal range in patients with CKD until very late stages of CKD, whereupon a slight decline often occurs [43]. Also, our findings are supported by earlier research, which has shown that low calcium levels are highly probable to occur after starting dialysis treatment [44]. In contrast, another study indicated that patients with chronic kidney disease initially present with low serum calcium levels. However, after starting dialysis, their serum calcium levels often rise.

This elevation is typically attributed to many factors, such as vitamin D therapy, the use of calcium-based phosphate binders, and the calcium content within the dialysate fluid [45]. The present study reported an increase in fasting blood sugar levels above the normal range in both study groups. This finding is supported by numerous studies that have demonstrated a significant reduction in insulin sensitivity in individuals with CKD [46]. However, a slight decrease in fasting blood sugar was observed in the current study in dialysis patients compared to non-dialysis patients. This finding can be explained by the complete normalization of insulin sensitivity after dialysis treatment [47]. In contrast, another opposing study suggested that dialysis treatment led to a significant impairment of insulin sensitivity [48]. In terms of GFR, the current study demonstrated a sharp decrease in GFR in both study groups, particularly among dialysis patients. This is expected, as patients on dialysis are typically in stage 4 of CKD. Although a rapid decline in GFR is a key indicator of underlying kidney disease and propensity to progress to end-stage kidney disease [49], there is no single estimated glomerular filtration rate (e-GFR) that is recommended for starting dialysis; the decision on when to start dialysis should be made collaboratively by the patient and their clinician [50].

Conclusion

This study showed a significant improvement in uric acid levels in CKD patients undergoing dialysis. Additionally, a marginal positive effect was observed on the other biomarkers, including hypertension,

urea, phosphorus, and glucose levels. However, the dialysis treatment failed to correct other important parameters, namely creatinine, potassium, and calcium. Although dialysis is the most effective method for removing accumulated toxins from the body, the process itself can complicate the patient's condition due to its side effects. The success of waste removal in dialysis is influenced by the patients' dietary habits, patient education, the correct timing of dialysis, and the selection of a suitable dialyzer. Currently, dialysis is sometimes used for even minor, treatable kidney issues. Therefore, the consequences of undergoing dialysis should be clearly communicated to both the physicians and the patients. There is an urgent need to educate CKD patients about the facts of their disease, medications, dietary habits, and the various measures required to manage the condition and lead a productive life. Enhancing dialysis efficiency may lead to a better quality of life for CKD patients, decrease complications, and reduce healthcare costs.

References

1. Mathur AK, Ashby VB, Sands RL, Wolfe RA. Geographic variation in end-stage renal disease incidence and access to deceased donor kidney transplantation. *Am J Transplant*. 2010;10(4 Pt 2):1069–80.
2. US Renal Data System. USRDS 2009 annual data report: atlas of chronic kidney disease and end-stage renal disease in the United States. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2009.
3. Sharma MK, Wieringa FP, Frijns AJ, Kooman JP. On-line monitoring of electrolytes in hemodialysis: on the road towards individualizing treatment. *Expert Rev Med Devices*. 2016;13(10):933–43.
4. Gounden V, Bhatt H, Jialal I. Renal function tests. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.
5. Wadei HM, Textor SC. The role of the kidney in regulating arterial blood pressure. *Nat Rev Nephrol*. 2012;8(10):602–9.
6. Levey AS, Eckardt U, Tsukamoto Y. Definition and classification of chronic kidney disease: a position statement from Kidney Disease. *Kidney Int*. 2005;67:2089–100.
7. Hu L, Napoletano A, Provenzano M, Garofalo C, Bini C, Comai G, et al. Mineral bone disorders in kidney disease patients: the ever-current topic. *Int J Mol Sci*. 2022;23(20):12223.
8. Anker SD, Doehner W, Rauchhaus M, Sharma R, Francis D, Knosalla C, et al. Uric acid and survival in chronic heart failure: validation and application in metabolic, functional, and hemodynamic staging. *Circulation*. 2003;107(15):1991–7.
9. Clive DM. Renal transplant-associated hyperuricemia and gout. *J Am Soc Nephrol*. 2000;11(5):974–9.
10. Colledge NK, Walker BR, Ralston SH. Davidson's Principles and Practice of Medicine. 21st ed. Edinburgh: Churchill Livingstone Elsevier; 2010. p. 487–92.
11. Narula AS. Chronic kidney disease: the looming threat. *Med J Armed Forces India*. 2008;64(1):2–3.
12. Santulli G, Cipolletta E, Sorriento D, Di Giudice G, Anastasio A, Monaco S, et al. CamK4 gene deletion induces hypertension. *J Am Heart Assoc*. 2012;1(4):e001081.
13. Santulli G, Trimarco B, Iaccarino G. G-protein-coupled receptor kinase 2 and hypertension: molecular insights and pathophysiological mechanisms. *High Blood Press Cardiovasc Prev*. 2013;20(1):5–12.
14. Caramori ML, Rossing P. Diabetic kidney disease. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. *Endotext* [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000 [updated 2022 Aug 3].
15. Abram S, Anju V. Assessment of quality of life in patients on haemodialysis and the impact of counseling. *Saudi J Kidney Dis Transpl*. 2012;23:953–7.
16. Andrews L, Vegada BN, Gosai HA. Evaluating levels of urea, creatinine and electrolytes in patients with chronic kidney failure pre and post dialysis: a retrospective analysis. *Sch Int J Biochem*. 2019;2(3):79–82.
17. Sehgal AR, Dor A, Tsai AC. Morbidity and cost implications of inadequate hemodialysis. *Am J Kidney Dis*. 2001;37:1223. doi:10.1053/ajkd.2001.24526.
18. Port FK, Ashby VB, Dhingra RK, Roys EC, Wolfe RA. Dialysis dose and body mass index are strongly associated with survival in hemodialysis patients. *J Am Soc Nephrol*. 2002;13:1061–6.
19. Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie FG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol*. 2004;15:2208–18.
20. Hayrullah Y, Mehmet B, Mustafa BK, Yesim GA, Sadik B. The effects of dialyzers on some blood biochemical parameters in hemodialysis patients. *Afr J Pharm Pharmacol*. 2011;5(22):2513–6.
21. Kaysen GA, Tom G, Larive B, Ravindra L. The effect of frequent hemodialysis on nutrition and body composition: frequent hemodialysis network trial. *Kidney Int*. 2012;82(1):90–9.
22. Vadakedath S, Kandi V. Dialysis: a review of the mechanisms underlying complications in the management of chronic renal failure. *Cureus*. 2017;9(8):e1603.
23. Schillaci G, Reboldi G, Verdecchia P. High-normal serum creatinine concentration is a predictor of cardiovascular risk in essential hypertension. *Arch Intern Med*. 2001;161:886–91.
24. Mahmoud M, Salem MD. Hypertension in the hemodialysis population: a survey of 649 patients. *Am J Kidney Dis*. 1995;26(3):461–8.
25. Thomas R, Kanso A, Sedor JR. Chronic kidney disease and its complications. *Prim Care*. 2008;35(2):1–15.
26. Narayana Murthy BV, Satyanarayana V. Prescribing pattern of drugs in chronic kidney disease patients on hemodialysis at a tertiary care hospital. *Int J Basic Clin Pharmacol*. 2017;6:928–32.

27. Iqbal K, Aslam R, Iqbal SS, Munir N, Abdullah I, Rasheed S, et al. Analyzing the impact of hemodialysis on urea and creatinine analytes in renal failure patients from Lahore, Pakistan. *Natl J Med Health Sci.* 2023;5(1):27–32.
28. Fatima N, Goraya SS, Saleem S. Dyslipidemias in patients with end-stage renal disease on conventional hemodialysis: a three-month follow-up. *Natl J Med Health Sci.* 2020;2(2):61–8.
29. Walker HK, Hall WD, Hurst JW, editors. *Clinical Methods: The History, Physical, and Laboratory Examinations.* 3rd ed. Boston: Butterworths; 1990. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK201/>
30. Chonchol M, Shlipak MG, Katz R, Sarnak MJ, Newman AB, Siscovick DS, et al. Relationship of uric acid with progression of kidney disease. *Am J Kidney Dis.* 2007;50(2):239–47.
31. Madero M, Sarnak MJ, Wang X, Greene T, Beck GJ, Kusek JW, et al. Uric acid and long-term outcomes in CKD. *Am J Kidney Dis.* 2009;53(5):796–803.
32. Shabazian H, Poorvays Z. Study on the adequacy of haemodialysis in Sina Hospital. *Jundishapur Sci Med J.* 2002;33:19–25.
33. Fotbolcu H, Duman D, Ecder SA, Oduncu V, Cevik C, Tigen K, et al. Attenuated cardiovascular response to sympathetic system activation during exercise in patients with dialysis-induced hypotension. *Am J Nephrol.* 2011;33(6):491–8.
34. Silverstein DM, Srivaths PR, Mattison P, Upadhyay K, Midgley L, Moudgil A, et al. Serum uric acid is associated with high blood pressure in pediatric hemodialysis patients. *Pediatr Nephrol.* 2011;26(7):1123–8.
35. Ajam WH. Evaluating serum electrolyte changes in chronic renal failure pre and post dialysis. *Medico-Legal Update.* 2020;20(4).
36. Sreenivasulu B, Bhagyamma A, Anuradha S. Study of serum electrolyte changes in end-stage renal disease patients before and after hemodialysis sessions. *Sch Acad J Biosci.* 2016;4(3B):283–7.
37. Nisha R, Kannan SR, Mariappan KT, Jagatha P. Biochemical evaluation of creatinine and urea in patients with renal failure undergoing hemodialysis. *J Clin Pathol Lab Med.* 2017;1(2):1–5.
38. Andrews L, Vegada BN, Gosai HA. Evaluating levels of urea, creatinine and electrolytes in patients with chronic kidney failure pre and post dialysis: a retrospective analysis. *Sch Int J Biochem.* 2019;2(3):79–82.
39. Santos SFF, Peixoto AJ. Sodium balance in maintenance hemodialysis. *Semin Dial.* 2010;23:555–9.
40. Schwarz S, Trivedi BK, Kalantar-Zadeh K, Kovesdy CP. Association of disorders in mineral metabolism with progression of chronic kidney disease. *Clin J Am Soc Nephrol.* 2006;1:825–31.
41. Singh S, Upadhyay-Dhungel K, Aryal G. Value of calcium and phosphorus in chronic kidney disease patients under hemodialysis: a retrospective study. *J Pathol Nepal.* 2012;2:293–6.
42. Block GA, Klassen PS, Lazarus JM, et al. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol.* 2004;15:2208–18.
43. Craver L, Marco MP, Martinez I, et al. Mineral metabolism parameters throughout chronic kidney disease stages 1–5: achievement of K/DOQI target ranges. *Nephrol Dial Transplant.* 2007;22:1171–6.
44. Inaguma D, Koide S, Takahashi K, et al. Relationship between serum calcium level at dialysis initiation and subsequent prognosis. *Ren Replace Ther.* 2017;3:2.
45. Melamed ML, Eustace JA, Plantinga L, Jaar BG, Fink NE, Coresh J, et al. Changes in serum calcium, phosphate, and PTH and the risk of death in incident dialysis patients: a longitudinal study. *Kidney Int.* 2006;70(2):351–7.
46. DeFronzo RA, Tobin JD, Rowe JW, Andres R. Glucose intolerance in uremia: quantification of pancreatic beta cell sensitivity to glucose and tissue sensitivity to insulin. *J Clin Invest.* 1978;62:35–425.
47. Kobayashi S, Maejima S, Ikeda T, Nagase M. Impact of dialysis therapy on insulin resistance in end-stage renal disease: comparison of haemodialysis and continuous ambulatory peritoneal dialysis. *Nephrol Dial Transplant.* 2000;15:65–70.
48. Satirapoj B, Supasynndh O, Phantana-Angkul P, Ruangkanchanasetr P, Nata N, Chaiprasert A, et al. Insulin resistance in dialysis versus non-dialysis end-stage renal disease patients without diabetes. *J Med Assoc Thai.* 2011;94(Suppl 4):S87–93.
49. Kop WJ, Seliger SL, Fink JC, Katz R, Odden MC, Fried LF, et al. Longitudinal association of depressive symptoms with rapid kidney function decline and adverse clinical renal disease outcomes. *Clin J Am Soc Nephrol.* 2011;6(4):834–44.
50. Flythe JE, Watnick S. Dialysis for chronic kidney failure: a review. *JAMA.* 2024;332(18):1559–73. doi:10.1001/jama.2024.16338.