Impact of Hemodiafiltration and Hemodialysis on FGF-23 Levels and Cardiovascular Calcification in End-Stage Renal Disease

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Abstract

Background: End-stage renal disease (ESRD) often leads to systemic complications, including vascular calcification, which significantly increases cardiovascular (CV) risk. Fibroblast growth factor 23 (FGF-23), an endocrine regulator of phosphate metabolism, has been implicated in the development of vascular calcification, though its precise role remains unclear. Hemodiafiltration (HDF) is an advanced dialysis modality that may influence FGF-23 levels and, consequently, CV risk. This study aims to explore the relationship between FGF-23, dialysis modality, and vascular calcification in ESRD patients. Methods: A cross-sectional study was conducted on 50 ESRD patients (25 on hemodialysis and 25 on hemodiafiltration) at Ain Shams University Specialized Hospitals, Egypt. FGF-23 levels were measured pre- and post-dialysis, and cardiovascular calcification was assessed using echocardiography and carotid duplex ultrasound. Laboratory parameters and inflammatory markers were also recorded. Statistical analysis was performed using SPSS version 26. Results: Pre-dialysis FGF-23 levels were significantly lower in the HDF group compared to the HD group ($p = 0.015$). The reduction ratio of FGF-23 was significantly higher in HDF patients ($p < 0.001$). Both groups exhibited similar levels of arterial plaque, but the HD group had significantly greater carotid intima-media thickness (IMT) ($p = 0.035$), suggesting an increased risk of cardiovascular complications. Post-treatment FGF-23 levels correlated with serum calcium, phosphorus, parathyroid hormone (PTH), and C-reactive protein (CRP), with stronger correlations observed in the HDF group. Conclusion: Hemodiafiltration is more effective than hemodialysis in reducing FGF-23 levels, potentially offering an advantage in controlling vascular calcification and reducing cardiovascular risk in ESRD patients. Despite similar plaque burdens, HDF patients demonstrated less carotid intima-media thickening, suggesting a better overall cardiovascular profile.

Keywords: FGF-23, hemodialysis, hemodiafiltration, vascular calcification, cardiovascular risk

Significance | This study determines the effects of dialysis modalities on FGF-23 levels and cardiovascular calcification, offering insights into improving ESRD patient outcomes.

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Introduction

End-stage renal disease (ESRD) represents the final, irreversible stage of chronic kidney disease (CKD), characterized by a profound loss of kidney function that necessitates either dialysis or kidney transplantation for survival (Afshar et al., 2024). Patients with ESRD frequently experience a cascade of systemic complications, including disturbances in mineral and bone metabolism, chronic inflammation, and a significantly heightened risk of cardiovascular (CV) events. These complications are often driven by progressive vascular calcification, a pathological process strongly associated

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with increased CV morbidity and mortality. Arterial calcification and its sequelae, such as ventricular hypertrophy and micro-embolic disease, play a central role in the exacerbation of CV risk in ESRD patients (Tashiro et al., 2019).

Fibroblast growth factor 23 (FGF-23), a 251-amino acid protein primarily secreted by osteocytes, is a key endocrine regulator of phosphate homeostasis. Its biological activity depends on its interaction with α-klotho, a co-receptor predominantly expressed in the kidney, choroid plexus, and parathyroid glands. FGF-23 exerts its phosphate-lowering effects by inhibiting renal phosphate reabsorption and suppressing calcitriol (1,25-dihydroxy vitamin D) synthesis, thereby maintaining phosphate balance. However, dysregulated FGF-23 activity can contribute to phosphate-wasting disorders such as autosomal recessive hypophosphatemic rickets, X-linked hypophosphatemic rickets, and fibrous dysplasia (Hao et al., 2019). Despite significant advances, the precise mechanisms governing FGF-23 regulation remain incompletely understood. High dietary phosphate intake, elevated 1,25-dihydroxy vitamin D levels, and parathyroid hormone (PTH) are known modulators of FGF-23, but emerging evidence suggests that inflammation may also influence its production, adding complexity to its regulatory network (Zununi Vahed et al., 2020).

α-Klotho is an essential cofactor that enables FGF-23 to regulate phosphate (Pi) and calcium (Ca) homeostasis. In the absence of α-klotho, FGF-23 signaling is impaired, leading to unchecked calcitriol production, increased intestinal and renal phosphate absorption, and subsequent hyperphosphatemia. This disruption exacerbates vascular calcification, a hallmark of CKD-associated mineral and bone disorder (Baradaran, 2023). Additionally, studies indicate that chronic inflammation, a common feature of CKD, may act as a potent stimulus for FGF-23 secretion, further linking inflammation to disordered mineral metabolism (Czaya & Faul, 2019).

Despite strong associations between elevated FGF-23 levels and adverse cardiovascular outcomes in CKD, its direct role in vascular calcification remains unclear. Studies investigating this relationship have yielded conflicting results, underscoring the need for further research to elucidate the precise mechanisms underlying FGF-23-mediated vascular pathology (González-Casaus et al., 2021).

Hemodiafiltration (HDF) is an advanced renal replacement therapy that combines diffusive and convective transport mechanisms to enhance the clearance of small- and middle-molecular-weight solutes. Online HDF, which utilizes ultrapure dialysate to replace fluid losses, offers potential advantages over conventional hemodialysis, particularly in mitigating inflammation and improving biocompatibility. Modern dialysis machines have been adapted to perform online HDF, incorporating stringent filtration mechanisms to ensure dialysate sterility and safety (Molina et al., 2024).

This study aims to explore the interplay between membrane permeability, hemodialysis modality, and FGF-23 levels to elucidate their combined impact on cardiovascular calcification in ESRD patients.

Materials and Methods

This cross-sectional study was conducted on 50 adult patients (>18 years) diagnosed with end-stage renal disease (ESRD), undergoing maintenance hemodialysis for at least six months on the same dialysis modality prior to enrollment. The research was carried out between June 2021 and September 2021, following approval from the Ethical Committee of Ain Shams University Specialized Hospitals, Egypt (approval code: 335/2018). Participants were recruited from Ain Shams University Specialized Hospitals in Cairo and Qutor Central Hospital in Gharbia Governorate.

Ethical Considerations

The investigation was approved by the Ethics Committee of Internal Medicine, Ain Shams University number MS 335/2018. The patients agreed to participate in the research, after receiving detailed information about the research. Written informed consent for participation in the study was obtained from subjects.

Inclusion and Exclusion Criteria

Inclusion criteria encompassed adult ESRD patients on stable hemodialysis for over six months. Exclusion criteria were applied to patients with severe infections within the past three months, active malignancies, acute or chronic inflammatory diseases, advanced or decompensated liver disease, and advanced or severe heart failure.

Study Groups

Participants were divided into two equal groups. Group 1 consisted of 25 patients receiving conventional hemodialysis using high-flux dialyzers (Platinum H, 1.8 m^2 surface area) on Fresenius 4008S or Gambro AK96 machines. Dialysis sessions for this group were conducted for four hours, three times per week, with a dialysate flow rate of 500 mL/min and a blood flow rate maintained between 300 and 350 mL/min.

Group 2 included 25 patients undergoing online hemodiafiltration (HDF) with post-dilution substitution fluid volumes exceeding 23 liters per session. These patients were treated using high-flux dialyzers (Platinum H, 2.0 m^2 surface area) on Fresenius 5008S machines. Similar to Group 1, the dialysate flow rate was maintained at 500 mL/min, and the blood flow rate ranged from 300 to 350 mL/min.

All dialyzers used in both groups were composed of polysulfone membranes and sterilized using autoclaving procedures.

Physical Examination

Vital signs, including blood pressure, were recorded, and body weight was measured pre- and post-dialysis.

Blood Sampling and Laboratory Analysis

Pre-dialysis blood samples (5 mL) were collected during the mid-week dialysis session using aseptic techniques from the dialysis bloodline. These samples were drawn into tubes containing EDTA anticoagulant, clot activator, and serum separation gel. The collected samples were then centrifuged at 2000-3000 rpm for five minutes to separate the plasma, which was subsequently stored at -80°C until analysis.

Laboratory tests were conducted as follows: Pre-dialysis tests included complete blood count (CBC), C-reactive protein (CRP), serum creatinine, blood urea nitrogen (BUN), sodium (Na), potassium (K), calcium, phosphate, parathyroid hormone (PTH), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and albumin. Post-dialysis, BUN was measured to assess dialysis adequacy.

FGF-23 Measurement

Pre- and post-dialysis FGF-23 levels were quantified using commercial ELISA kits (Cat. No. E0059HU). Separate blood samples (5 mL) were collected from the dialysis line into tubes containing clot activator and serum separation gel, followed by centrifugation at 2000-3000 rpm for 20 minutes. Both kits and samples were brought to room temperature prior to analysis to ensure accuracy.

Dialysis Adequacy

The Kt/V ratio, an established measure of dialysis adequacy, was calculated for all patients before the study.

Cardiovascular Assessment

Echocardiography: A semi-quantitative echocardiographic approach was used to assess coronary artery calcification scores (CACS). Two-dimensional (2D) echocardiographic images evaluated calcification at the aortic valve (AVC), mitral annulus (MAC), aortic root, and papillary muscles. Each site was graded on a 0-3 scale based on echogenicity and structural changes. The cumulative score ranged from 0 to 8, providing an overall calcification assessment (Gaibazzi et al., 2014).

Carotid Duplex Ultrasound: Carotid intima-media thickness (IMT) was measured via B-mode ultrasound, capturing the double-line pattern that distinguishes the lumen-intima and media-adventitia interfaces. Increased IMT indicated vascular calcification and atherosclerosis (Øygarden, 2017). Additionally, color Doppler ultrasound evaluated carotid artery stenosis, with narrowing quantified using the NASCET formula. Stenosis severity was categorized as mild (<30%), moderate (30-69%), severe (70-99%), or complete occlusion (100%) (Chu et al., 2019).

Data Collection

Participants underwent structured interviews to gather data on ESRD etiology, comorbidities, dialysis duration, and type of vascular access (e.g., arteriovenous fistula, graft, or catheter).

Statistical Analysis

Data analysis was performed using SPSS version 26 (IBM Corp., Chicago, IL, USA). Quantitative variables were presented as means ± standard deviations (SD) and compared between groups using the unpaired Student's t-test. Categorical variables were expressed as frequencies and percentages and analyzed using the Chi-square test or Fisher's exact test where appropriate. Pearson's correlation coefficient was employed to examine relationships between continuous variables. A p-value of <0.05 was considered statistically significant.

Results

Patient demographics and characteristics were comparable between the two groups. Hemodiafiltration (HDF) patients demonstrated significantly higher dialysis efficiency, as evidenced by higher Kt/V values compared to hemodialysis (HD) patients ($p < 0.012$), indicating more effective waste removal. Both groups underwent dialysis for similar durations, showing no difference in treatment time required. Most laboratory parameters, including hemoglobin, white blood cell count, platelets, blood urea nitrogen (BUN), calcium, phosphorus, parathyroid hormone (PTH), creatinine, and albumin, did not exhibit significant differences between the groups. However, C-reactive protein (CRP), an inflammatory marker, was significantly lower in the HDF group ($p < 0.032$). Pre-dialysis FGF-23 levels were significantly lower in the HDF group compared to the HD group, and the FGF-23 reduction ratio was significantly higher in the HDF group ($p = 0.015$ and $p < 0.001$, respectively) (Table 1, 2).

While there were no significant differences in cardiac calcification as assessed by echocardiography (ECHO score) or overall plaque burden in the carotid arteries (carotid duplex score), HD patients exhibited significantly greater carotid intima-media thickness (IMT) compared to HDF patients ($p = 0.035$). This suggests a potential increased risk of cardiovascular complications in HD patients despite similar levels of overall arterial plaque (Table 3).

In both HD and HDF patients, higher FGF-23 levels were significantly associated with increased carotid IMT in HDF patients ($r = 0.579$, $p = 0.008$). This correlation was not observed in HD patients or for other cardiovascular measures such as ECHO scores or carotid duplex scores. Additionally, post-treatment FGF-23 levels showed positive correlations with serum calcium, serum phosphorus, PTH, and CRP in both groups. The correlation with serum calcium was stronger in HD patients ($r = 0.659$, $p = 0.002$) than in HDF patients ($r = 0.474$, $p = 0.035$). Serum phosphorus correlations were similar in both groups ($r = 0.462$, $p = 0.04$ for HD; $r = 0.478$, $p = 0.033$ for HDF). PTH correlations were stronger in HDF patients ($r = 0.547$, $p = 0.013$) compared to HD patients ($r = 0.485$, $p = 0.03$). CRP correlations were also stronger in HDF patients ($r = 0.707$, $p < 0.001$) than in HD patients ($r = 0.646$, $p = 0.002$) (Table 4).

Table 1. Patient's characteristics and demographic data and aetiology of ESRD between both groups

		Group 1 (n = 25)	Group 2 (n = 25)	P value
Age (years)	**Mean ± SD**	50.44 ± 12.24	57.68 ± 13.99	0.057
	Range	25 - 80	22 - 72	
Sex	**Male**	14 (56.0%)	18 (72.0%)	0.239
	Female	11 (44.0%)	7 (28.0%)	
Weight (kg)	**Mean ± SD**	66.92 ± 8.74	67.90 ± 13.76	0.765
	Range	54 - 83	49 - 90	
Aetiology of ESRD	**DM**	5 (20%)	4 (16%)	0.785
	HTN	7 (28%)	7 (28%)	
	Obstructive uropathy	0 (0%)	2 (8%)	
	Analgesic abuse	2 (8%)	3 (12%)	
	ADPKD	3 (12%)	4 (16%)	
	Neurogenic bladder	2 (8%)	1 (4%)	
	Unknown	6 (24%)	4 (16%)	

Data is presented as Mean ± SD, (n = 25)

Table 2. Laboratory investigations , Kt/v and duration of dialysis between both groups

		Group 1 (n = 25)		Group 2 (n = 25)		P value
Kt/v	**Mean ± SD**	1.36 ± 0.06		1.42 ± 0.10		**0.012***
	Range	1.3 - 1.5		1.3 - 1.6		
Duration of dialysis (years)	**Mean ± SD**	5.80 ±3.25		5.12 ± 3.18		0.458
	Range	2 - 14		1 - 15		
		Mean	**±SD**	**Mean**	**±SD**	
Hemoglobin (gm/dL)		10.36	1.53	11.08	1.49	0.101
Serum BUN (mg/dl)		55.048	18.05	55.05	22.12	0.870
Serum Cr (mg/dL)		9.12	1.51	9.12	1.77	1.00
Serum Ca (mg/dL)		8.25	1.93	8.84	0.78	0.074
Serum PO4 (mg/dL)		5.58	2.06	5.32	1.52	0.652
PTH (pg/mL)		459.45	322.2	377.85	246.97	0.374
CRP (mg/L)		21.58	16.98	11.75	10.05	**0.032***
Serum Albumin (g/dL)		3.65	0.33	3.67	0.29	0.856
FGF23 (pg/mL)		**Group 1**		**Group 2**		**P value**
Pre	**Mean ± SD**	944±316.33		775.2±297.7		**0.015***
	Range	650 - 1750		350 - 1500		
Post	**Mean ± SD**	373.6±79.16		176.4±91.19		**<0.001***
	Range	250 - 500		100 - 500		
Reduction ratio (%)	**Mean ± SD**	58.28% ± 15.60%		79.98% ± 8.28%		**<0.001***
	Range	20.00% - 80.00%		54.55% - 89.57%		

Cr: creatinine, Ca: calcium, Po_4 phosphorus, CRP C reactive protein, FGF 23 fibroblast growth factor 23 *: significant as p value < 0.05.

Table 3. ECHO score, carotid duplex score (%) and carotid intima-media thickness between both groups.

		Group 1	Group 2	P value
ECHO score	**Mean ± SD**	2.6 ± 1.12	2.16 ± 1.7	0.285
	Median	3	2	
	Range	1 - 5	0 - 8	
Carotid duplex score (%)	**Mean ± SD**	6.07% ± 12.95%	3.89% ± 11.25%	0.529
	Median	0.00%	0.00%	
	Range	0.0% - 43.0%	0.0% - 44.0%	
Carotid intima-media thickness (mm)	**Mean ± SD**	0.89 ± 0.19	0.78 ± 0.18	**0.035***
	Median	0.9	0.8	
	Range	0.4 - 1.2	0.5 - 1.3	

*: significant as p value <0.05.

Table 4. Correlation between serum level of FGF23 post-dialysis and ECHO score, carotid intima-media thickness , carotid duplex score and (serum calcium, phosphorus and PTH) in the studied groups

	FGF post HD		**FGF post HDF**	
	Group 1		**Group 2**	
	r	**P value**	**r**	**P value**
ECHO score	0.505	0.158	0.043	0.856
Carotid intima-media thickness	0.201	0.395	0.579	**0.008***
Carotid duplex score	0.106	0.656	0.148	0.534
Serum Ca (mg/dL)	0.659	**0.002***	0.474	**0.035***
Serum Po4 (mg/dL)	0.462	**0.04***	0.478	**0.033***
PTH (pg/mL)	0.485	**0.03***	0.547	**0.013***
CRP (mg/L)	0.646	**0.002***	0.707	**<0.001***

Ca: calcium, po4: phosphorus, PTH: parathyroid hormone. CRP: C reatctive protein r: correlation coefficient, *: significant as P value ≤ 0.05.

Discussion

We assessed the effectiveness of different dialysis modalities in controlling fibroblast growth factor 23 (FGF-23) levels by measuring pre- and post-dialysis FGF-23 concentrations in both hemodialysis (HD) and hemodiafiltration (HDF) patients. Notably, pre-dialysis FGF-23 levels were significantly lower in the hemodiafiltration group compared to the hemodialysis group. Furthermore, the percentage reduction in FGF-23 (reduction ratio) was significantly higher in the hemodiafiltration group, highlighting the superior capability of HDF in removing this key mediator of cardiovascular complications in end-stage renal disease (ESRD) patients.

Our findings align with those of Bouma-de Krijger et al. (2021), who compared the effectiveness of online HDF and low-flux HD in controlling mortality and cardiovascular events in ESRD patients. They found that FGF-23 levels significantly decreased over time in the HDF group due to its superior clearance capacity, while remaining stable in the HD group. Their study revealed that the median FGF-23 level in the HDF group was significantly lower [3691 RU/mL (IQR 1826–12,293)] than in the HD group [4983 RU/mL (IQR 1815–12,265)]. Similarly, Choo et al. (2019) compared two dialysis regimens (8-hour hemodialysis vs. 4-hour hemodiafiltration) in stable HD patients and demonstrated that, beyond better clearance of small molecules like urea and creatinine, hemodiafiltration was more effective in removing FGF-23, a key player in cardiovascular health.

However, our results contrast with those of Kim et al. (2019), who conducted a small observational study comparing the efficacy of different dialysis modalities in six stable HD patients. Using a single midweek treatment for each of three modalities (medium cut-off HD, high-flux HD, and pre-dilution online HDF), they observed no significant differences in FGF-23 reduction ratios (55.5%, 34.6%, and 35.8% respectively). This discrepancy highlights the need for further research to elucidate factors influencing FGF-23 removal across different dialysis strategies.

In our study, while overall cardiac calcification assessed by echocardiography (ECHO score) and plaque burden in the carotid arteries (carotid duplex score) were not significantly different between HD and HDF groups, carotid intima-media thickness (IMT) was significantly greater in the HD group. This suggests that although HD and HDF patients may have similar levels of arterial calcification and plaque, HD patients might have an elevated risk of specific cardiovascular complications due to thicker carotid artery walls.

Supporting our findings, Hao et al. (2019) observed a statistically significant difference in abdominal aortic calcification (AAC) score increases in the first year, favoring hemodiafiltration (0.79 vs. 0.44 in HDF and HD groups, respectively). This suggests that while HDF might offer initial advantages in limiting AAC, long-term outcomes may be similar for both modalities. Similarly, Pradeep et al. (2020) revealed a statistically significant positive correlation between FGF-23 levels and carotid IMT, indicating that higher FGF-23 levels were associated with thicker arterial walls.

Contrastingly, Lee et al. (2021) observed no significant differences in echocardiographic measures, such as left ventricular function, over a year. However, their results diverged from ours concerning coronary artery calcification (CAC). Notably, CAC scores remained stable in the HDF group, whereas HD patients exhibited a trend towards increasing CAC scores.

Our study revealed that both HDF and regular HD exhibited positive correlations between post-treatment FGF-23 levels and various blood markers, including calcium, phosphorus, parathyroid hormone (PTH), and C-reactive protein (CRP). These findings suggest potential links between FGF-23 and various biological processes relevant to cardiovascular health in dialysis patients.

Supporting our findings, Tashiro et al. (2019) observed significant correlations between FGF-23 levels and serum calcium, phosphorus, PTH, and magnesium. However, their results did not show any significant correlations between FGF-23 and markers of cardiovascular health, such as arteriosclerosis score, heart function by echocardiography, blood pressure, carotid artery health by ultrasound, and survival prognosis. Similarly, Zeng et al. (2023) reported that serum FGF-23 showed positive correlations with blood calcium, phosphorus, and PTH levels in hemodialysis patients.

Our study also revealed a statistically significant difference in CRP levels between the hemodiafiltration and hemodialysis groups. Notably, CRP levels were significantly lower in the hemodiafiltration group, indicating a potential reduction in systemic inflammation.

Supporting our findings, Mady et al. (2021) observed a statistically significant difference in CRP levels between the two groups. Patients receiving HDF had lower mean CRP levels (63.5 ± 40.9 mg/dL) compared to those on HD (73.4 ± 33.2 mg/dL), further emphasizing the potential anti-inflammatory benefits of hemodiafiltration.

The study's limitations include the relatively small sample size, cross-sectional design, and the absence of a comparative control group. Therefore, we recommend that future studies include larger, randomized trials, incorporate control groups, and explore additional biomarkers related to cardiovascular health for a more comprehensive understanding of the impact of dialysis modalities on FGF-23 and cardiovascular calcification.

Conclusion

In conclusion, this study highlights the superior efficacy of hemodiafiltration (HDF) over conventional hemodialysis (HD) in reducing fibroblast growth factor 23 (FGF-23) levels in end-stage

renal disease (ESRD) patients. HDF demonstrated significantly higher FGF-23 reduction ratios, lower inflammatory markers (CRP), and reduced carotid intima-media thickness (IMT), suggesting potential cardiovascular protective effects. While overall arterial calcification and plaque burdens were comparable between the two groups, the increased IMT in HD patients indicates a higher risk of specific cardiovascular complications. These findings align with previous studies demonstrating the benefits of HDF in improving cardiovascular outcomes, although conflicting evidence suggests further research is needed to clarify FGF-23's role in vascular calcification. The positive correlations between FGF-23 and markers such as calcium, phosphorus, parathyroid hormone (PTH), and CRP emphasize its involvement in disordered mineral metabolism and inflammation in ESRD. Future longitudinal studies are necessary to confirm these results and assess long-term cardiovascular outcomes in HDF versus HD patients.

Author contributions

H.E. conceived the original idea and developed the necessary tools for execution. W.A.B. oversaw patient recruitment and revised the tools to enhance their effectiveness. M.A. led the data collection, analysis, and interpretation. F.A. and K.R. collaborated on designing the methodology, interpreting the results, and drafting the manuscript. All authors contributed to the final approval of the manuscript, with each playing a vital role in the research and the successful completion of the project.

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Competing financial interests

The authors have no conflict of interest.

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