Original Article

Impact of Vitamin E Supplementation on Inflammatory and Nutritional Markers in Patients Undergoing Hemodialysis With Chronic Kidney Disease: A Randomized Controlled Trial

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ABSTRACT

Background: Chronic kidney disease (CKD) is a worldwide concern, and individuals undergoing hemodialysis (HD) are at high risk for cardiovascular disease because of malnutrition and chronic inflammation. This study evaluated the efficacy of vitamin E (VitE) supplementation in modulating nutritional and inflammatory markers in participants undergoing HD.

Methods: This double-blind, randomized, controlled trial lasted 12 weeks and included 40 participants undergoing HD who received 400 IU of VitE daily or placebo. Inflammatory markers (ferritin, C-reactive protein [CRP], and erythrocyte sedimentation rate [ESR]) and nutritional markers (body mass index [BMI] and serum albumin) were assessed at baseline and after the intervention. Lipid profile and HD adequacy were also evaluated.

Results: Participants receiving VitE exhibited significant improvements in nutritional markers (serum albumin and BMI) and inflammatory markers (ferritin, CRP, and ESR) compared with the placebo group. Nonetheless, lipid profile and HD adequacy did not significantly improve in either group.

Conclusions: VitE supplementation improved nutritional status and reduced inflammation in participants with CKD undergoing HD. Still, additional studies are required to determine long-term efficacy and generalizability. ***(Iranian Heart Journal 2026; 27(3): 27-36)***

KEYWORDS: chronic kidney disease; hemodialysis; vitamin E; inflammation; nutritional status

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Chronic kidney disease (CKD) is a significant public health problem. In dialysis-dependent individuals, including those undergoing hemodialysis (HD), CKD is associated with a particularly high risk of complications, with cardiovascular disease accounting for a high proportion of deaths among individuals with end-stage renal disease.[1] The mortality rate among individuals undergoing HD is approximately 20 to 30 times higher than that of the general population, and traditional risk factors alone do not fully explain this increased risk.[2] Further, lipoprotein levels are strongly correlated with atherosclerotic cardiovascular disease in participants undergoing HD, emphasizing the importance of monitoring lipid profile in this vulnerable group.[3]

Increased oxidative stress and inflammation are important predictors of prognosis in individuals undergoing HD.[4] Strong evidence supports a positive association between elevated concentrations of reactive oxygen species and progression of atherosclerotic disease in this population.[5] Furthermore, ongoing inflammation in CKD is accompanied by malnutrition, both of which contribute to increased cardiovascular morbidity and mortality.[6] Hence, oxidative stress and inflammation must be addressed to improve prognosis in individuals undergoing HD.[7]

Vitamin E (VitE) (α-tocopherol) is a fat-soluble antioxidant with an important role in counteracting oxidative stress and exerting anti-inflammatory effects.[8] The antioxidant and anti-inflammatory effects of VitE have been demonstrated in many studies, including reductions in C-reactive protein (CRP) and other inflammatory markers. Be that as it may, its effects on lipid profile and HD adequacy remain controversial, with studies reporting variable results.[9]

Accordingly, the present study aimed to investigate the effects of VitE supplementation on inflammatory markers, nutritional markers, lipid profile, and HD adequacy in participants undergoing HD. By clarifying the therapeutic role of VitE in this population, this study may contribute to the development of more effective nutritional and pharmacologic interventions for management of CKD, with the goal of improving patient care and outcomes.

METHODS

The present study used a structured design to assess VitE intervention for anti-inflammatory and nutritional improvement in participants undergoing HD with CKD.

Trial Design

This single-blind, parallel-group, placebo-controlled trial was conducted at Bu Ali Hospital in Tehran, Iran. The Faculty of Pharmacy and Pharmaceutical Sciences at Islamic Azad University approved the study (ethical approval No. IR.IAU.PS.REC.1398.290).

Inclusion and Exclusion Criteria

Participants were recruited according to the following inclusion and exclusion criteria.

Inclusion criteria: Participants aged 40 to 90 years with a confirmed medical diagnosis of CKD who had been undergoing HD for at least 3 months and had no change in medication during the previous 3 months.

Exclusion criteria: Participants unable to provide consent; those taking VitE; those with contraindications to VitE or a history of allergic reaction to VitE; pregnant or lactating females; those with severe medical disease; those with active inflammatory or rheumatologic disease; and those participating in an ongoing clinical trial.

Intervention and Comparator

Participants were randomly assigned to 1 of 2 groups.

Study group: Participants received 400 IU of VitE daily (E-Zavit; Zahravi Pharmaceutical Company, Iran) administered as soft gelatin capsules.

Control group: Participants received soft gelatin capsules containing similar excipients but no VitE.

Randomization and Allocation Concealment

Randomization was performed using a web-based system (Amaracademi for Biostatistics) with a minimization algorithm and a small degree of randomness to balance key baseline factors, including study site, age, sex, and duration of HD.

Primary and Secondary Outcomes

Primary outcome: The change in CRP levels between groups after 12 weeks of intervention, adjusted for baseline values.

Secondary outcomes:

- **Inflammatory markers:** Erythrocyte sedimentation rate (ESR) and ferritin.
- **Nutritional markers:** Serum albumin level and body mass index (BMI).
- **Lipid profile:** Total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride levels.
- **HD adequacy:** Quantified by Kt/V, dialysis duration, and urea distribution.

Sample Size Calculation

The sample size was calculated based on the primary outcome, the change in CRP levels. With the aid of G*Power software (version 3.1.9.7), a 2-tailed independent *t* test was performed with an effect size of 0.8, an alpha level of .05, and power (1 − β) of 80%. The calculation indicated a required sample size of 18 participants per group. To account for a potential 10% dropout rate, 20 participants were recruited per group, resulting in a total sample size of 40.

Data Collection

Blood samples were collected from participants after a 12- to 14-hour overnight fast at baseline and 12 weeks after the intervention for analysis of the aforementioned biomarkers and variables.

Statistical Analysis

Statistical analyses were performed using GraphPad Prism (version 10.0) and SPSS (version 26). For longitudinal data (baseline and 12-week measurements), a 2-way repeated-measures analysis of variance (ANOVA) was employed to assess the effects of time, group, and time × group interaction, followed by Bonferroni post hoc tests for pairwise comparisons. For non-normally distributed data, the Friedman test was applied. Between-group comparisons of baseline characteristics were performed using independent *t* tests, Mann–Whitney *U* tests, chi-square tests, or Fisher exact tests, as appropriate. A *P* value of less than .05 was considered statistically significant.

RESULTS

Participant Flow and Baseline Features

A total of 57 participants were assessed for eligibility; 13 were excluded and 4 declined to participate. Therefore, 40 participants were randomized into 2 groups: 20 received VitE supplementation and 20 received placebo (Figure 1).

There were no significant differences in baseline characteristics between groups, including age, sex, dialysis duration, and clinical factors ($P > .05$) (Table 1).

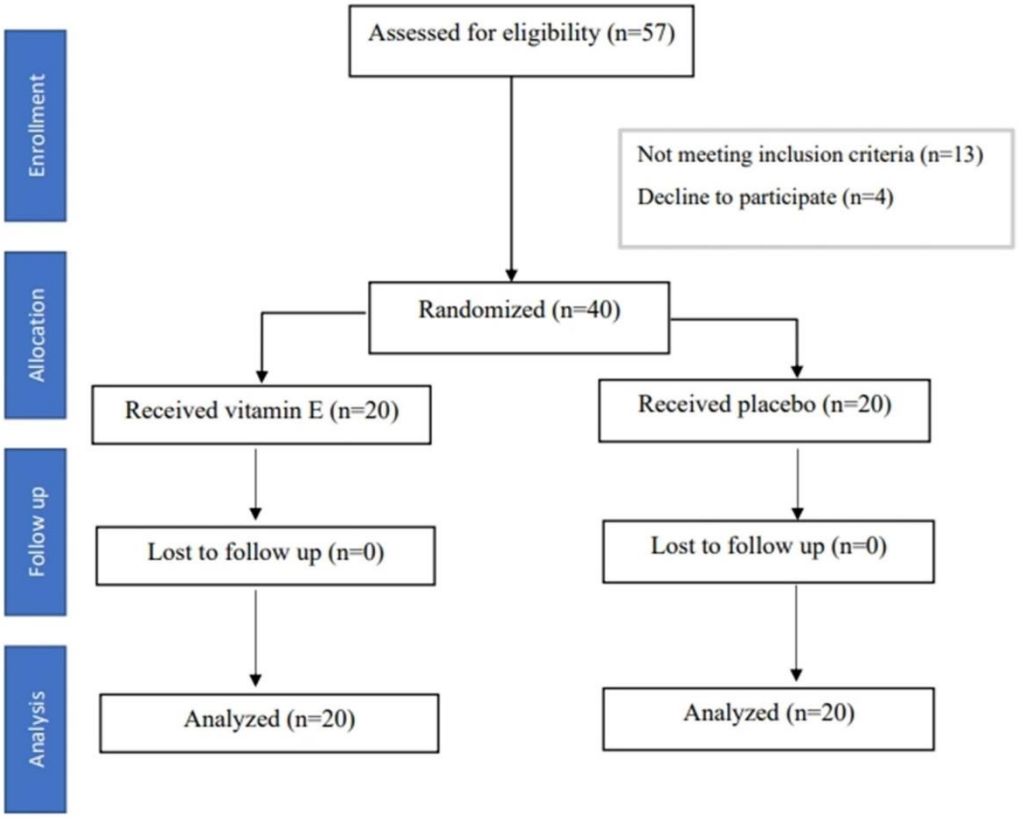

Figure 1. Enrollment and randomization flow diagram

Table 1. Baseline Characteristics of Participants in the Vitamin E and Control Groups

Characteristic		Control Group	Vitamin E Group	*P*
Age, y, mean (SD)		73.85 (13.91)	66.7 (7.29)	.058
Hemodialysis history, y, mean (SD)		3.6 (2.41)	4.2 (2.76)	.466
Sex, No. (%)				
Male		12 (60%)	8 (40%)	.343
Female		8 (40%)	12 (60%)	
Past medical history Patient, No. (%)				
Losartan (mg/d)	0	11 (55%)	12 (60%)	.171
	25	4 (20%)	7 (35%)	
	50	5 (25%)	1 (5%)	
Valsartan (mg/d)	0	17 (85%)	15 (75%)	.429
	80	3 (15%)	5 (25%)	
Atorvastatin (mg/d)	0	15 (75%)	12 (60%)	.343
	10	0 (0%)	3 (15%)	
	20	3 (15%)	3 (15%)	
	40	2 (10%)	2 (10%)	
Metformin (mg/d)	0	15 (75%)	12 (60%)	.501
	500	5 (25%)	8 (40%)	
Captopril (mg/d)	0	18 (90%)	18 (90%)	1.000
	25	2 (10%)	2 (10%)	
Glibenclamide (mg/d)	0	20 (100%)	18 (90%)	.487
	5	0 (0%)	2 (10%)	

Primary Outcomes

The primary outcome was the change in CRP levels between groups after 12 weeks of intervention. CRP levels were significantly lower in participants receiving VitE, with values of 6.52 (0.66) mg/L compared with 7.31 (0.89) mg/L in the control group ($P < .05$) (Table 2).

Inflammatory Markers

In addition to CRP, significant reductions in other inflammatory markers were observed. Ferritin levels in participants receiving VitE were 35.85 (11.13) ng/mL compared with 47.05 (15.63) ng/mL in the control group ($P < .05$). Similarly, ESR was 37.25 (12.41) mm/h in the VitE group compared with 46.45 (12.69) mm/h in the control group ($P < .05$) (Figure 2).

These findings indicate that VitE supplementation reduced inflammatory markers in participants undergoing HD.

Nutritional Markers

Significant improvements in nutritional markers were observed after 12 weeks. Serum albumin levels in participants receiving VitE were 3.79 (0.38) g/dL compared with 3.41 (0.44) g/dL in the control group ($P < .05$).

BMI was 21.47 (1.79) kg/m^2 in the VitE group compared with 19.88 (2.37) kg/m^2 in the control group ($P < .05$) (Figure 2).

HD Adequacy

HD adequacy, assessed using Kt/V, was higher in the VitE group (1.355 [0.29]) than in the control group (1.209 [0.28]); nonetheless, the difference was not statistically significant ($P > .05$) (Figure 2).

Lipid Profile

Despite improvements in inflammatory and nutritional markers, no significant differences in lipid profiles between groups were observed. Total cholesterol levels in the VitE group were 172.22 (36.69) mg/dL compared with 189.23 (30.06) mg/dL in the control group ($P > .05$).

Similarly, no significant differences were observed for triglycerides (170.81 [15.96] vs 178.72 [13.16] mg/dL), LDL cholesterol (82.75 [12.65] vs 88.32 [15.22] mg/dL), or HDL cholesterol (39.65 [5.55] vs 36.65 [9.90] mg/dL) between the groups ($P > .05$) (Figure 3).

Overall, 12 weeks of VitE supplementation significantly reduced inflammatory markers (CRP, ferritin, and ESR) and improved nutritional markers (serum albumin and BMI) in participants undergoing HD; however, no significant effects were observed on HD adequacy (Kt/V) or lipid profile parameters.

Table 1. Serum Levels of Inflammatory Markers, Nutritional Markers, Lipid Profiles, and Hemodialysis Adequacy at Baseline and After 12 Weeks of Vitamin E Supplementation

Factor	Baseline, mean (SD)			After 3 Months, mean (SD)		
	Vitamin E Group	Control Group	*P*	Vitamin E Group	Control Group	*P*
Ferritin	50.41 (14.57)	46/.81 (13.87)	.42	35.85 (11.13)	47.05 (15.63)	.01
C-reactive protein	7.67 (1.16)	7.34 (0.98)	.33	6.52 (0.66)	7.31 (0.89)	≤ .01
Erythrocyte sedimentation rate	49.61 (14/42)	47.11 (14.27)	.57	37.25 (12.41)	46.45 (12.69)	.03
Albumin	3.46 (0.43)	3.45 (0.46)	.94	3.79 (0.38)	3.41 (0.44)	≤ .01
Body mass index	20.16 (2.21)	19.86 (2.59)	.69	21.47 (1.79)	19.88 (2.37)	.03
Cholesterol	194.71 (41.94)	191.75 (36.91)	.81	172.22 (36.69)	189.23 (30.06)	.11
Triglycerides	175.11 (21.03)	178.21 (18.79)	.62	170.81 (15.96)	178.72 (13.16)	.09
Low-density lipoprotein cholesterol	88.81 (17.65)	87.95 (15.43)	.87	82.75 (12.65)	88.32 (15.22)	.21
High-density lipoprotein cholesterol	35.35 (6.05)	35.05 (10.19)	.91	39.65 (5.55)	35.65 (9.90)	.12
KT/V	1.18 (0.34)	1.20 (0.31)	.92	1.36 (0.29)	1.21 (0.28)	.11

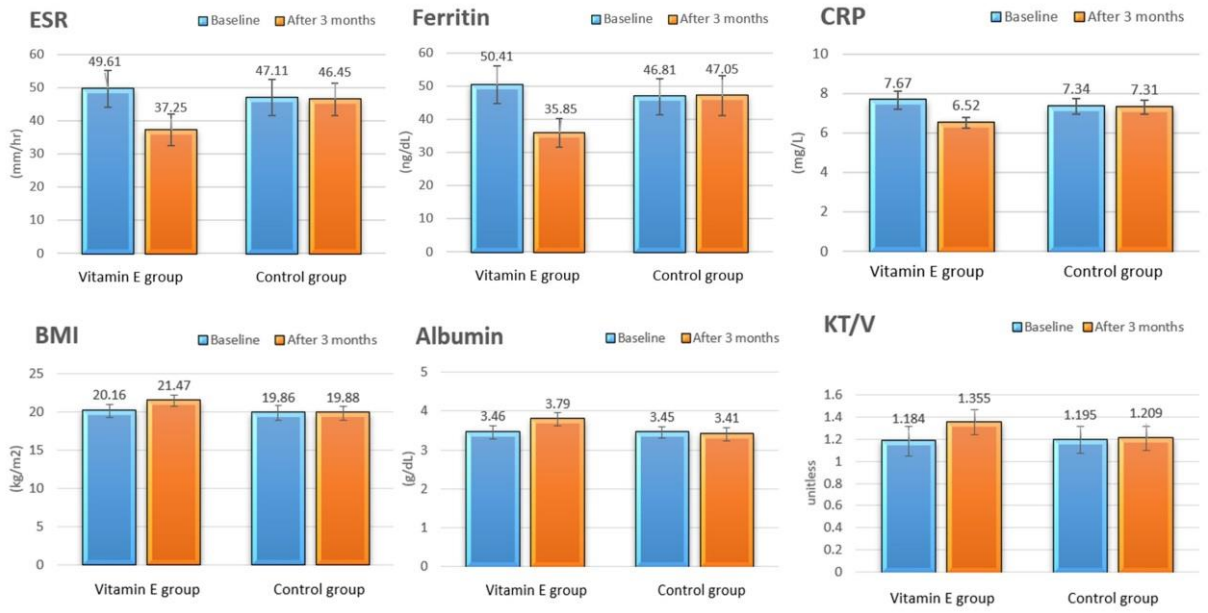

Figure 2. Changes in inflammatory markers (C-reactive protein [CRP], ferritin, and erythrocyte sedimentation rate [ESR]), nutritional markers (serum albumin and body mass index [BMI]), and hemodialysis adequacy (Kt/V) at baseline and after 12 weeks of vitamin E supplementation

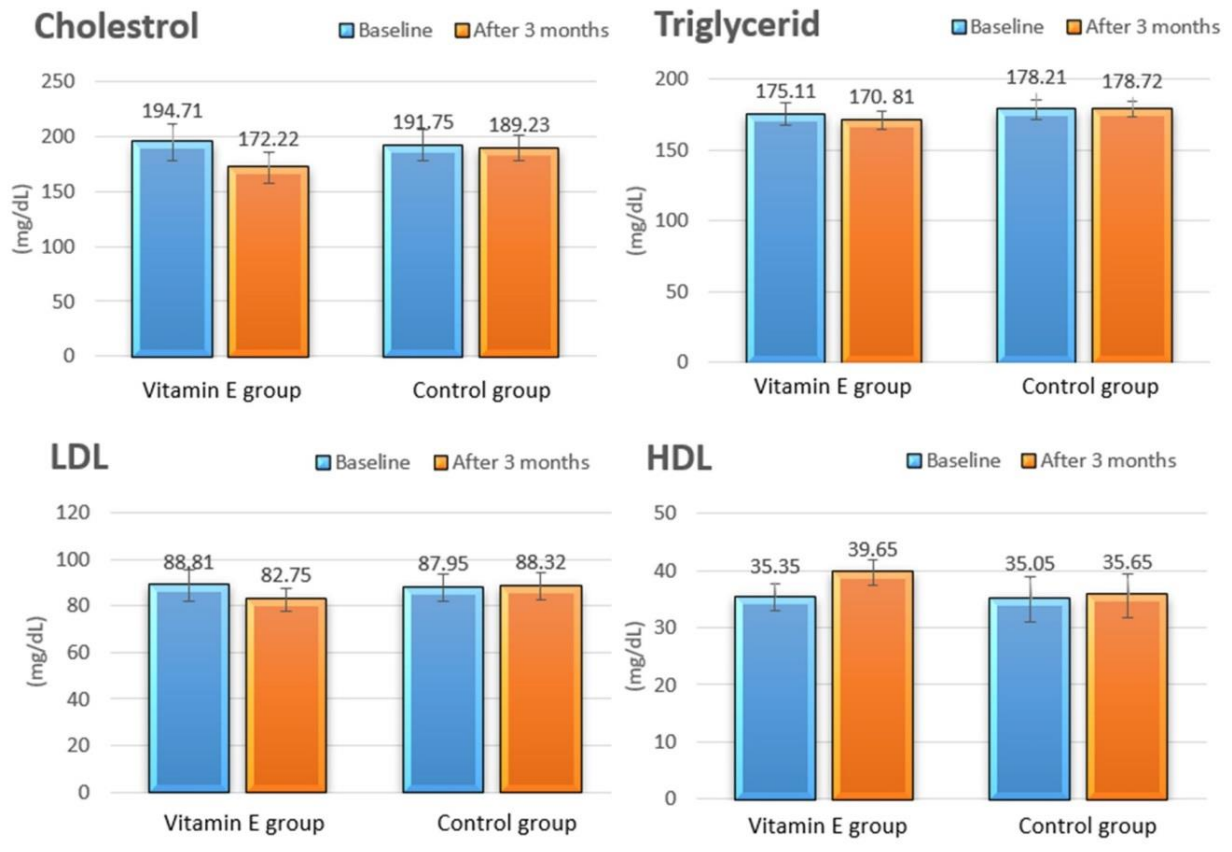

Figure 3. Comparison of lipid profiles (total cholesterol, triglycerides, low-density lipoprotein [LDL] cholesterol, and high-density lipoprotein [HDL] cholesterol) between the vitamin E and control groups

DISCUSSION

VitE supplementation reduced concentrations of inflammatory markers, including CRP, ferritin, and ESR, in participants undergoing HD. These findings are consistent with previous studies demonstrating the anti-inflammatory effects of VitE in individuals with CKD.[10, 11]

Although some studies have reported no significant effect of VitE on CRP levels, others have demonstrated significant reductions in CRP concentrations.[9, 12] Because CRP is a key marker of inflammation associated with cardiovascular disease risk, reductions in CRP levels may indicate that VitE supplementation could help mitigate inflammation-related complications in CKD.[9]

One study reported no significant reduction in cardiovascular complications after 2 years of VitE treatment in individuals with CKD; however, a meta-analysis demonstrated beneficial effects on endothelial dysfunction, inflammation, and oxidative stress biomarkers in participants undergoing HD.[9, 13]

Similarly, the observed reduction in ferritin levels may suggest a role for VitE in iron metabolism through the reduction of oxidative stress.[14] VitE functions as a lipid-soluble antioxidant that scavenges free radicals and inhibits lipid peroxidation, mechanisms that may contribute to its anti-inflammatory effects.[15]

Previous studies have also reported beneficial effects of VitE supplementation on inflammatory markers in participants undergoing HD.[9, 16] Overall, these findings suggest that VitE may have therapeutic value as an adjunct therapy for inflammation associated with CKD.

The significant improvement in nutritional markers, including serum albumin and BMI, in the VitE group suggests that VitE supplementation may contribute to improved nutritional status in participants undergoing HD. Malnutrition is common in this population and is often exacerbated by oxidative stress and chronic inflammation.

Improvement in serum albumin levels may reflect better protein nutritional status, which is clinically important because hypoalbuminemia is associated with increased morbidity and mortality in individuals undergoing HD.[17] Early serum albumin levels in patients receiving HD reflect both nutritional and inflammatory status and may predict prognosis, with higher levels associated with improved survival.[18]

The association between VitE supplementation and improved nutritional markers may indicate a beneficial role of VitE in overall health and resistance to malnutrition in this population. In addition, previous studies have reported positive associations between antioxidant supplementation, including VitE, and improved nutritional status, possibly through reductions in inflammation-related protein–energy wasting.[19]

Further research is needed to better understand the relationships among nutrition, CKD, and VitE supplementation.

Although the present study demonstrated improvements in inflammatory and nutritional markers, no significant between-group differences were observed in lipid profile parameters, including total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol. Therefore, despite its antioxidant properties, the role of VitE supplementation in modulating lipid profiles in participants undergoing HD remains inconclusive.

Some studies have suggested that VitE supplementation may reduce lipid peroxidation and increase polyunsaturated fatty acid levels, whereas others have reported no significant changes in LDL cholesterol levels after supplementation in individuals undergoing HD.[20, 21] Previous research has demonstrated variable efficacy

of VitE in modulating lipid profiles, with beneficial effects reported in some studies and no significant impact in others.[22, 23]

These findings suggest that although VitE may influence oxidative stress and lipid metabolism, individual variation and comorbid conditions should be considered in future research. Participants undergoing HD generally exhibit higher levels of malondialdehyde and lower antioxidant enzyme activity than healthy individuals.[24]

One study reported that long-term oral alpha-tocopherol administration in participants undergoing HD was associated with decreased total antioxidant status and reduced superoxide dismutase activity, suggesting a potential pro-oxidative effect of VitE in this population.[25] Combined supplementation with VitE and other agents, such as omega-3 fatty acids, may represent a potential therapeutic strategy because synergistic effects could enhance improvements in lipid profiles.

The present study did not detect a significant difference in dialysis adequacy, assessed using Kt/V, between groups. This finding is consistent with previous studies indicating that dialysis adequacy is not substantially affected by VitE supplementation.[26, 27] However, the higher Kt/V observed in the VitE group suggests a possible favorable trend that may warrant further investigation. Future studies with larger sample sizes and longer intervention durations are needed to clarify the potential role of VitE supplementation in improving dialysis adequacy.

This study has several limitations. The relatively small sample size may limit the generalizability of the findings, and the 12-week intervention period may not be sufficient to evaluate the long-term effects of VitE supplementation. In addition, variability in individual dietary intake and comorbid conditions may have influenced the observed outcomes.

Future research should include multicenter trials with larger sample sizes and longer follow-up periods to validate these findings. Investigating different doses of VitE, as well as combinations with other antioxidant agents, may provide a more comprehensive understanding of the optimal supplementation strategy for individuals with CKD undergoing HD.

In summary, this study suggests that VitE supplementation may reduce inflammatory markers and improve nutritional markers in individuals with CKD undergoing HD. Although no significant differences were observed in lipid profiles or HD adequacy, assessed using Kt/V, between the VitE and control groups, the observed anti-inflammatory effects may have potential clinical relevance. These findings indicate that VitE supplementation could represent a supportive therapeutic strategy for managing inflammation in this population, with possible implications for reducing cardiovascular risk.

CONCLUSIONS

This study found that VitE supplementation was associated with reductions in inflammatory markers and improvements in nutritional markers in individuals with CKD undergoing HD. Significant decreases in CRP, ferritin, and ESR, along with increases in serum albumin and BMI, were observed in the VitE group. No significant changes were observed in lipid profiles or HD adequacy, assessed using Kt/V.

These findings suggest that the anti-inflammatory effects of VitE may have potential clinical relevance for improving care and outcomes in this population, with possible implications for cardiovascular risk reduction. Larger studies with longer intervention periods are warranted to further evaluate the benefits and underlying mechanisms of VitE supplementation.

REFERENCES

1. Bansal N. Evolution of cardiovascular disease during the transition to end-stage renal disease. Elsevier; 2017:120-131.
2. McIntyre CW. Update on HD-Induced Multiorgan Ischemia: Brains and Beyond. Journal of the American Society of Nephrology. 2024:10.1681.
3. García de la Vega C, Blanco MA, Aguilar JC, et al. # 2141 Lipoprotein a levels and atherosclerotic cardiovascular disease in hemodialysis patients. Nephrology Dialysis Transplantation. 2024; 39(Supplement_1):gfae069-0883-2141.
4. Epifânio AP, Balbino KP, Ribeiro SM, Franceschini SC, Hermsdorff HH-M-. Clinical-nutritional, inflammatory and oxidative stress predictors in hemodialysis mortality: a review. Nutrición Hospitalaria. 2018; 35(2):461-468.
5. Burtenshaw D, Kitching M, Redmond EM, Megson IL, Cahill PA. Reactive oxygen species (ROS), intimal thickening, and subclinical atherosclerotic disease. Frontiers in cardiovascular medicine. 2019; 6:89.
6. Peev V, Nayer A, Contreras G. Dyslipidemia, malnutrition, inflammation, cardiovascular disease and mortality in chronic kidney disease. Current opinion in lipidology. 2014; 25(1):54-60.
7. Liakopoulos V, Roumeliotis S, Gorny X, Dounousi E, Mertens PR. Oxidative stress in hemodialysis patients: a review of the literature. Oxidative medicine and cellular longevity. 2017; 2017(1):3081856.
8. Garg A, Lee JC-Y. Vitamin E: Where are we now in vascular diseases? Life. 2022; 12(2):310.
9. Nguyen TTU, Yeom J-h, Kim W. Beneficial effects of vitamin E supplementation on endothelial dysfunction, inflammation, and oxidative stress biomarkers in patients receiving hemodialysis: a systematic review and meta-analysis of randomized controlled trials. International Journal of Molecular Sciences. 2021; 22(21):11923.
10. Zhang Y, Gao W, Li X. Vitamin E-coated dialyzer alleviates erythrocyte deformability dysfunction in patients with end-stage renal disease undergoing hemodialysis. Experimental and Therapeutic Medicine. 2022; 24(1):1-9.
11. Wu Q, Luo Y, Lu H, et al. The Potential Role of Vitamin E and the Mechanism in the Prevention and Treatment of Inflammatory Bowel Disease. Foods. 2024; 13(6):898.
12. Hodkova M, Dusilova-Sulkova S, Kalousova M, et al. Influence of oral vitamin E therapy on micro-inflammation and cardiovascular disease markers in chronic hemodialysis patients. Renal failure. 2006; 28(5):395-399.
13. Azouaou L, Adnane M, Chabati O, Arab M, Chahine T, Chader H. Profiling oxidative stress markers and cardiovascular complications in chronic kidney disease patients supplemented with vitamin E. Archives of Medical Sciences Atherosclerotic Diseases. 2024; 9:e183.
14. Baratz E, Protchenko O, Jadhav S, et al. Vitamin e induces liver iron depletion and alters iron regulation in mice. The Journal of Nutrition. 2023; 153(7):1866-1876.
15. Niki E. Role of vitamin E as a lipid-soluble peroxyl radical scavenger: in vitro and in vivo evidence. Free Radical Biology and Medicine. 2014; 66:3-12.
16. Asbaghi O, Sadeghian M, Nazarian B, et al. The effect of vitamin E supplementation on selected inflammatory biomarkers in adults: a systematic review and meta-analysis of randomized clinical trials. Scientific reports. 2020; 10(1):17234.
17. Boz G, Uludag K. Serum Albumin Trends in Relation With Prognosis of Individuals Receiving Hemodialysis Therapy. Cureus. 2021; 13(11)
18. Tang J, Wang L, Luo J, et al. Early albumin level and mortality in hemodialysis patients: a retrospective study. Annals of Palliative Medicine. 2021; 10(10):106970705-106910705.
19. Rojo-Trejo MH, Robles-Osorio ML, Sabath E. Liposoluble vitamins A and E in kidney

disease. World Journal of Nephrology. 2022; 11(3):96.

20. Galli F, Varga Z, Balla J, et al. Vitamin E, lipid profile, and peroxidation in hemodialysis patients. Kidney International. 2001; 59:S148-S154.

21. Montazerifar F, Hashemi M, Karajibani M, Dikshit M. Hemodialysis alters lipid profiles, total antioxidant capacity, and vitamins A, E, and C concentrations in humans. Journal of Medicinal Food. 2010; 13(6):1490-1493.

22. Mohseni S, Tabatabaei-Malazy O, Shadman Z, Khashayar P, Mohajeri-Tehrani M, Larijani B. Targeting dyslipidemia with antioxidative vitamins C, D, and E; a systematic review of meta-analysis studies: Dyslipidemia and antioxidative vitamins. Journal of Diabetes & Metabolic Disorders. 2021:1-11.

23. Cardenas E, Ghosh R. Vitamin E: a dark horse at the crossroad of cancer management. Biochemical pharmacology. 2013; 86(7):845-852.

24. Gonenc A, Atak Y, ORMAN M, Simsek B. Lipid peroxidation and antioxidant systems in hemodialyzed patients. Dialysis & transplantation. 2002; 31(2)

25. Antoniadi G, Eleftheriadis T, Liakopoulos V, et al. Effect of One-year Oral α-Tocopherol Administration on the Antioxidant Defense System in Hemodialysis Patients. Therapeutic Apheresis and Dialysis. 2008;12(3):237-242.

26. Yang S-K, Xiao L, Xu B, Xu X-X, Liu F-Y, Sun L. Effects of vitamin E-coated dialyzer on oxidative stress and inflammation status in hemodialysis patients: a systematic review and meta-analysis. Renal failure. 2014; 36(5):722-731.

27. Sanaka T, Mochizuki T, Kinugasa E, et al. Randomized controlled open-label trial of vitamin E-bonded polysulfone dialyzer and erythropoiesis-stimulating agent response. Clinical Journal of the American Society of Nephrology. 2013; 8(6):969-978.