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Comparative Study of Efficacy and Safety of Sevelamer versus Calcium Acetate in CKD-Related Hyperphosphatemia

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ABSTRACT

Background: Hyperphosphatemia is a critical complication in chronic kidney disease (CKD), contributing to cardiovascular morbidity. While calcium-based binders are effective, they carry a risk of hypercalcemia and vascular calcification. This study compares the efficacy and safety of sevelamer versus calcium acetate in managing CKD-related hyperphosphatemia.
Methods: This prospective, randomized, open-label trial enrolled 100 patients with CKD stages 4-5 and persistent hyperphosphatemia (>5.5 mg/dL). Patients were randomized to receive sevelamer (800 mg thrice daily, n=50) or calcium acetate (667 mg thrice daily, n=50) for 12 weeks. The primary outcome was the change in serum phosphate levels. Secondary outcomes included changes in serum calcium, calcium-phosphate (Ca x P) product, lipid profile, and incidence of adverse events.
Results: Baseline characteristics were similar between groups. At week 12, sevelamer resulted in a significantly greater mean reduction in serum phosphate from baseline compared to calcium acetate (2.04 ± 0.87 vs. 1.53 ± 0.91 mg/dL; p=0.0051). The incidence of hypercalcemia was significantly lower with sevelamer (4% vs. 20%; p=0.0277). Furthermore, sevelamer demonstrated a significant reduction in LDL cholesterol (-18.07±12.86 mg/dL) compared to minimal change with calcium acetate (-2.11±10.26 mg/dL; p<0.0001). A strong trend toward reduced progression of vascular calcification was observed with sevelamer (8% vs. 24%; p=0.0538).
Conclusion: Sevelamer is more effective than calcium acetate in lowering serum phosphate and offers significant advantages in reducing the risk of hypercalcemia and improving the lipid profile. These benefits support its preferential use in CKD patients at high cardiovascular risk.

Keywords: Sevelamer, Calcium Acetate, Hyperphosphatemia, Chronic Kidney Disease, Vascular Calcification

INTRODUCTION

Chronic kidney disease (CKD) is a progressive condition characterized by a gradual decline in renal function, ultimately leading to end-stage renal disease (ESRD). [1] Among the numerous metabolic derangements associated with CKD, disturbances in mineral metabolism—particularly hyperphosphatemia—are of paramount clinical importance. [2] Elevated serum phosphate levels are not only a consequence of reduced renal excretion but also a major contributor to secondary hyperparathyroidism, vascular calcification, and increased cardiovascular morbidity and

mortality in this patient population. [3] The management of hyperphosphatemia, therefore, represents a cornerstone of CKD care, with the dual goals of improving patient outcomes and mitigating long-term complications. [2-4]

Phosphate binders remain the mainstay of therapy for controlling serum phosphate levels in CKD patients, especially those on dialysis. [5] Traditionally, calcium-based binders such as calcium acetate and calcium carbonate have been widely prescribed due to their efficacy, availability, and relatively low cost. Calcium acetate, in particular, has demonstrated superior phosphate-binding capacity compared to calcium carbonate, making it a preferred choice among calcium-based agents. However, despite their effectiveness, calcium-based binders are associated with significant drawbacks. [6, 7] Chief among these is the risk of hypercalcemia, which can exacerbate vascular and soft tissue calcification, thereby increasing cardiovascular risk. This concern has prompted the exploration of non-calcium-based alternatives.

Sevelamer, a non-absorbable polymeric phosphate binder, represents one such alternative. Unlike calcium-based binders, sevelamer does not contribute to hypercalcemia and has been shown to attenuate the progression of vascular calcification. [8] Additionally, sevelamer exerts favorable effects on lipid metabolism, reducing low-density lipoprotein cholesterol (LDL-C) levels, which may confer added cardiovascular protection. [9] These pleiotropic benefits make sevelamer an attractive therapeutic option, particularly in patients at high risk of vascular calcification or those with concomitant hypercalcemia. Nonetheless, sevelamer is not without limitations. Gastrointestinal side effects, pill burden, and higher cost compared to calcium acetate remain practical challenges that influence its widespread use, especially in resource-limited settings. [10]

Hyperphosphatemia remains a critical challenge in CKD care, with significant implications for patient morbidity and mortality. While calcium acetate continues to be widely used due to its effectiveness and affordability, sevelamer offers distinct advantages in terms of safety and cardiovascular protection. A direct comparative evaluation of these agents is essential to inform clinical practice, optimize patient outcomes, and balance efficacy with safety in the management of CKD-related mineral metabolism disorders. This study endeavours to contribute to this ongoing discourse by providing robust data on the comparative efficacy and safety of sevelamer versus calcium acetate, thereby enhancing the evidence base for therapeutic decision-making in CKD patients.

The choice between calcium acetate and sevelamer, therefore, is not merely a matter of efficacy in lowering serum phosphate but also involves considerations of safety, tolerability, and cost-effectiveness. Several studies have compared these agents, with varying conclusions. [11-14] Some have highlighted the superior safety profile of sevelamer in terms of reducing vascular calcification and avoiding hypercalcemia, while others emphasize the cost-effectiveness and accessibility of calcium acetate, particularly in developing countries where healthcare resources are constrained. Given these divergent findings, there remains a need for well-structured comparative studies that evaluate both efficacy and safety outcomes in a balanced manner.

This study seeks to address this gap by conducting a comparative analysis of sevelamer and calcium acetate in the management of CKD-related hyperphosphatemia. By systematically evaluating their efficacy in lowering serum phosphate levels and assessing their safety profiles—including the incidence of hypercalcemia, gastrointestinal intolerance, and vascular calcification—this research aims to provide evidence that can guide clinicians in making rational, patient-centered therapeutic choices. Furthermore, the study's objective is to consider the broader implications of these agents on cardiovascular risk factors, quality of life,

and treatment adherence, thereby offering a holistic perspective on their role in CKD management.

MATERIALS & METHODS

Study Overview: This study was designed as a prospective, randomized, open-label, comparative trial conducted at a tertiary care nephrology center. The primary objective was to evaluate the efficacy and safety of sevelamer versus calcium acetate in the management of hyperphosphatemia among patients with chronic kidney disease (CKD). The study duration was 12 weeks, during which patients were monitored for biochemical parameters, clinical outcomes, and adverse events.

Eligibility Criteria

Patients aged 18–75 years with CKD stages 4 and 5, including those on maintenance hemodialysis, were eligible if they had persistent hyperphosphatemia (serum phosphate >5.5 mg/dL) despite dietary phosphate restriction. Exclusion criteria included "history of parathyroidectomy or severe hyperparathyroidism requiring surgical intervention; baseline hypercalcemia (serum calcium >10.5 mg/dl); active gastrointestinal disorders (e.g., inflammatory bowel disease, chronic diarrhea); use of other phosphate binders within 4 weeks prior to enrolment; pregnancy or lactation and known hypersensitivity to study drugs."

Sample Size: A sample size of 100 patients was calculated based on an expected mean difference of 0.8 mg/dL in serum phosphate reduction between groups, with a standard deviation of 1.2, power of 80%, and alpha of 0.05. Patients were randomized in a 1:1 ratio into two groups: sevelamer group (n=50) and calcium acetate group (n=50).

Intervention: Patients in Group A received sevelamer hydrochloride at an initial dose of 800 mg three times daily with meals. Group B received calcium acetate at an initial dose of 667 mg three times daily with meals. Doses were titrated every 2 weeks based on serum phosphate levels, aiming to maintain phosphate between 3.5–5.5 mg/dL. Both groups were advised on dietary phosphate restriction and continued standard CKD care including dialysis, erythropoiesis-stimulating agents, and antihypertensive therapy as indicated.

Outcome Parameters

Primary outcome measure was "change in serum phosphate levels from baseline to 12 weeks" whereas secondary outcomes included **"**change in serum calcium and calcium-phosphate product; incidence of hypercalcemia (serum calcium >10.5 mg/dl); change in intact parathyroid hormone (iPTH) levels; occurrence of vascular calcification assessed by lateral abdominal radiograph; adverse events including gastrointestinal intolerance, constipation, or diarrhea; treatment adherence and pill burden assessment."

METHODOLOGY

Baseline demographic and clinical data were recorded, including age, sex, CKD stage, dialysis duration, and comorbidities. Laboratory investigations (serum phosphate, calcium, iPTH, alkaline phosphatase, lipid profile) were performed at baseline, 6 weeks, and 12 weeks. Radiographic evaluation for vascular calcification was performed at baseline and at study completion. Adverse events were documented at each follow-up visit. Compliance was assessed by pill counts and patient diaries.

Randomization was performed using a computer-generated sequence, and allocation was concealed using sealed opaque envelopes. Although the study was open-label, laboratory personnel analyzing samples were blinded to treatment allocation to minimize bias.

Statistical Analysis

Data were analysed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages.

Between-group comparisons were performed using unpaired t-tests for continuous variables and chi-square tests for categorical variables. Repeated measure ANOVA was used to assess changes of continuous variable within group. A p-value <0.05 was considered statistically significant.

RESULTS

Figure 1 illustrates the CONSORT flow diagram depicting the recruitment, allocation, follow-up, and analysis of participants in the study. A total of 123 individuals were assessed for eligibility. Of these, 23 participants were excluded, including 19 who did not meet the inclusion criteria and 4 who declined to participate, while no participants were excluded for other reasons. The remaining 100 participants were randomized into two equal groups. During the allocation phase, 50 participants were assigned to the Sevelamer group and 50 participants to the Calcium Acetate group. All participants in both groups received the allocated intervention, and no participants failed to receive the assigned treatment. In the follow-up phase, no participants discontinued the intervention and no participants were lost to follow-up in either group. Finally, in the analysis phase, all randomized participants were included in the primary outcome analysis, with 50 participants analyzed in the Sevelamer group and 50 in the Calcium Acetate group, and no participants were excluded from the analysis. This flow diagram demonstrates complete follow-up and analysis of all randomized participants in both treatment arms.

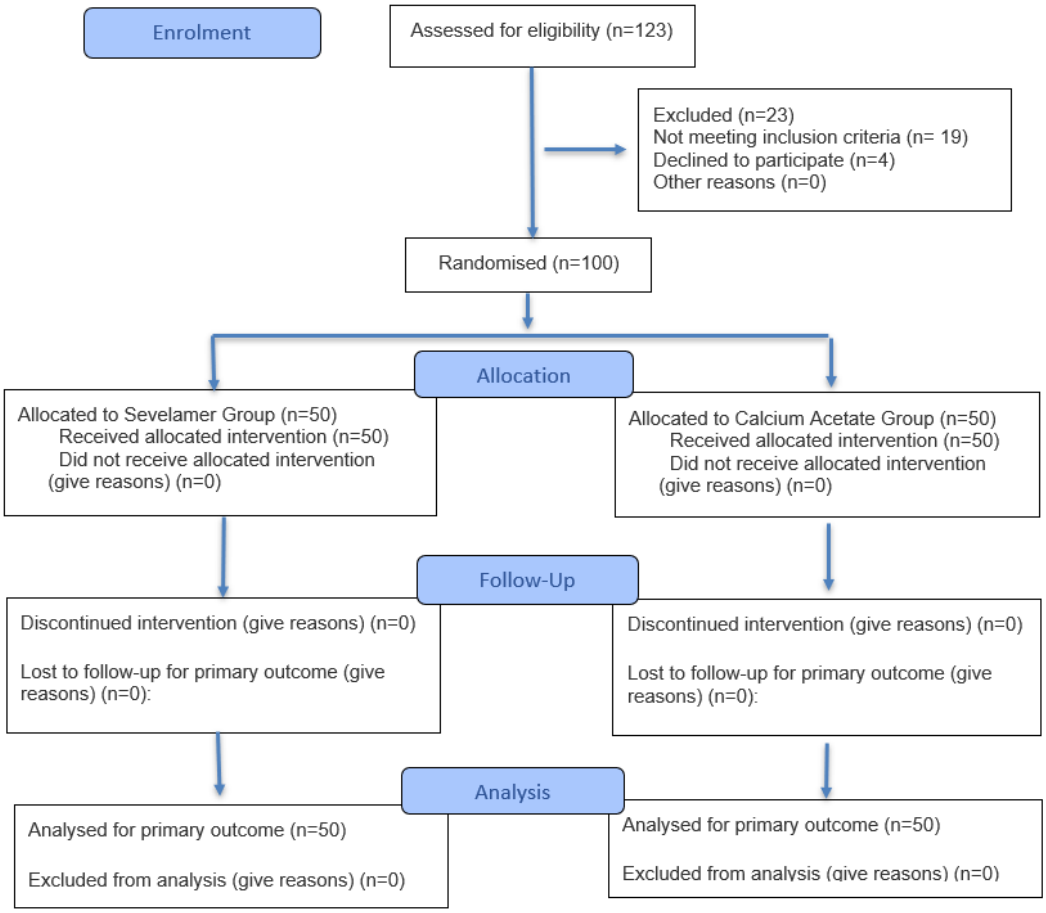

Figure 1: CONSORT flow diagram

There were no statistically significant differences between the sevelamer and calcium acetate groups in terms of age, sex distribution, the proportion of patients with CKD stage 5, duration of dialysis, or the

prevalence of common comorbidities like diabetes and hypertension [Table 1].

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Variable	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	p-value
Age in years, mean ± SD	52.41 ± 11.27	53.12 ± 10.85	0.7490*
Male sex, n (%)	28 (56%)	30 (60%)	0.8396**
CKD stage 5, n (%)	35 (70%)	34 (68%)	>0.9999**
Duration on dialysis in months, mean ± SD	18.62 ± 7.43	17.96 ± 8.19	0.6739*
Diabetes mellitus, n (%)	22 (44%)	20 (40%)	0.8396**
Hypertension, n (%)	40 (80%)	42 (84%)	0.7953**

***Unpaired t test; **Fisher's Exact Test**

Mean serum phosphate, calcium, calcium-phosphate (Ca x P) product, intact parathyroid hormone (iPTH), and LDL cholesterol levels were all statistically equivalent between the sevelamer and calcium acetate groups [Table 2].

While the phosphate reduction was similar between the two groups at the 6-week mark, a significant difference emerged by week 12. At the study's conclusion, the sevelamer group achieved a significantly lower mean serum phosphate level (4.86 ± 0.63 mg/dL) compared to the calcium acetate group (5.27 ± 0.76 mg/dL, p=0.0041). Furthermore, the mean reduction from baseline was significantly greater in the sevelamer group (2.04 ± 0.87 mg/dL) than in the calcium acetate group (1.53 ± 0.91 mg/dL, p=0.0051), suggesting superior phosphate-lowering efficacy for sevelamer over the 12-week treatment period [Table 3].

Table 2. Baseline Laboratory Parameters

Parameter	Value in Mean ± SD		
	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	p-value (Unpaired t test)
Serum phosphate (mg/dL)	6.84 ± 0.91	6.71 ± 1.08	0.5166
Serum calcium (mg/dL)	9.24 ± 0.63	9.15 ± 0.72	0.5075
Ca × P product (mg^2/dL^2)	62.68 ± 8.49	61.93 ± 9.16	0.6720
iPTH (pg/mL)	320.23 ± 110.76	315.41 ± 120.58	0.1471
LDL cholesterol (mg/dL)	118.35 ± 25.05	120.30 ± 28.94	0.7126

Table 3. Change in Serum Phosphate Over 12 Weeks

Time Point	Serum Phosphate in mg/dl, Mean ± SD		p-value (Unpaired t test)
	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	
Baseline	6.81 ± 0.94	6.72 ± 1.05	0.6526
Week 6	5.49 ± 0.76	5.68 ± 0.85	0.2415
Week 12	4.86 ± 0.63	5.27 ± 0.76	0.0041
P-Value (Repeated Measure ANOVA)	<0.0001	0.0003	-
Mean reduction	2.04 ± 0.87	1.53 ± 0.91	0.0051

The incidence of hypercalcemia was significantly lower in the sevelamer group (4%) compared to the calcium acetate group (20%, p=0.0277), confirming the key safety advantage of the non-calcium-based binder. However, this benefit came with a trend towards a different side effect profile. Although not statistically significant, gastrointestinal intolerance was numerically higher in the sevelamer group (16% vs.

12%), reflecting its known tolerability issues [Table 4].

Table 4. Safety Outcomes

Adverse Event	Number of Patients (%)		p-value (Fisher's Exact Test)
	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	
Hypercalcemia (%)	2 (4%)	10 (20%)	0.0277
GI intolerance (%)	8 (16%)	6 (12%)	0.7742
Constipation (%)	5 (10%)	4 (8%)	>0.9999
Diarrhea (%)	3 (6%)	2 (4%)	>0.9999

The calcium acetate group experienced a significant mean increase in serum calcium (0.66 mg/dL) compared to a minimal change in the sevelamer group, directly correlating with the higher hypercalcemia risk noted in Table 4. Consequently, the reduction in the Ca x P product was significantly greater with sevelamer. Perhaps most notably, sevelamer demonstrated a significant lipid-lowering effect, reducing LDL cholesterol by a mean of 18.07 mg/dL, whereas calcium acetate had a minimal effect. This pleiotropic benefit of sevelamer was highly significant ($p<0.0001$) and represents an important additional cardiovascular protective mechanism [Table 5].

Table 5. Effect on Secondary Parameters

Parameter	Change (Week 12 – Baseline), Mean ± SD		p-value (Unpaired t test)
	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	
Serum calcium (mg/dL)	0.13 ± 0.38	0.66 ± 0.47	<0.0001
Ca × P product (mg²/dL²)	-10.21 ± 5.16	-6.42 ± 4.87	0.0003
iPTH (pg/mL)	-45.13 ± 30.34	-40.25 ± 35.05	0.4566
LDL cholesterol (mg/dL)	-18.07 ± 12.86	-2.11 ± 10.26	<0.0001

While the prevalence of vascular calcification was similar in both groups at baseline, a notable difference emerged after 12 weeks. Only 8% of patients in the sevelamer group showed progression of vascular calcification, compared to 24% in the calcium acetate group. Although this result fell just short of traditional statistical significance ($p=0.0538$), it demonstrates a strong trend suggesting that sevelamer may attenuate the progression of vascular calcification compared to calcium acetate.

Table 6. Radiographic Vascular Calcification Progression

Assessment	Number of Patients (%)		p-value (Fisher's Exact Test)
	Sevelamer Group (n=50)	Calcium Acetate Group (n=50)	
Baseline calcification	20 (40%)	18 (36%)	0.8369
Progression at 12 weeks	4 (8%)	12 (24%)	0.0538

DISCUSSION

Our prospective randomized study comparing sevelamer with calcium acetate in 100 patients with advanced CKD (stages 4–5) over 12 weeks yielded several clinically meaningful findings. Each of these outcomes can be contextualized against the backdrop of prior studies, highlighting both consistencies and discrepancies in the literature.

In our trial, sevelamer achieved superior phosphate reduction compared to calcium acetate, with mean decreases of 2.04 mg/dL versus 1.53 mg/dL ($p=0.0051$). This translated into lower final serum phosphate values (4.86 vs. 5.27 mg/dL). While this finding supports sevelamer's efficacy, the broader literature presents a more nuanced picture. Bleyer et al. (1999) reported

equivalent phosphate reductions with both agents in a crossover design, suggesting no superiority. [11] Similarly, Hervás et al. (2003) found comparable reductions over 34 weeks, with differences not reaching statistical significance. [12] In contrast, the CARE study by Qunibi et al. (2004) favored calcium acetate, demonstrating significantly lower phosphorus levels and higher attainment of target goals. [13] Patel et al. (2016), in a meta-analysis of 25 studies, concluded that overall phosphate control was equivalent between sevelamer and calcium-based binders. [14] Thus, while our study suggests sevelamer superiority, methodological differences—such as titration protocols, dietary compliance, and open-label design—may explain divergence from prior results. Our every-two-week titration schedule may have optimized sevelamer dosing more effectively than fixed-dose approaches used elsewhere.

One of the most striking findings in our study was the markedly lower incidence of hypercalcemia with sevelamer (4% vs. 20%; p=0.0277). This represents an 80% relative risk reduction and aligns closely with prior evidence. Bleyer et al. (1999) reported hypercalcemia in 22% of calcium acetate recipients versus 5% with sevelamer, nearly identical to our results. [11] Meta-analyses by Habbous et al. (2017) and Patel et al. (2016) confirmed this consistent advantage, with relative risks of 0.27 and 0.30 respectively. [14, 15] Qunibi et al. (2004) also demonstrated significantly higher hypercalcemia risk with calcium acetate (OR 6.1). [13] The clinical importance of this finding cannot be overstated: hypercalcemia contributes directly to vascular calcification progression, a complication already prevalent in CKD patients. Our data showing a trend toward reduced vascular calcification progression with sevelamer (8% vs. 24%) reinforces the mechanistic link between calcium loading and vascular injury. Thus, the safety advantage of sevelamer in avoiding hypercalcemia is one of the most robust and consistent findings across the literature.

Our study demonstrated a significantly greater reduction in the calcium–phosphate (Ca×P) product with sevelamer (−10.21 vs. −6.42 mg^2/dL^2; p=0.0003). This is clinically relevant, as K/DOQI guidelines recommend maintaining Ca×P <55 mg^2/dL^2 to reduce vascular calcification risk. [16] Prior studies have reported mixed results. Hervás et al. (2003) found similar reductions between agents, though not statistically significant. [12] Qunibi et al. (2004), paradoxically, reported lower Ca×P product with calcium acetate, consistent with their finding of superior phosphate control in that trial. [13] Bleyer et al. (1999) did not report Ca×P directly, but their phosphate and calcium data would favor sevelamer. [11] Our findings suggest that sevelamer's dual effect—better phosphate control and stable calcium levels—contributes to more favorable Ca×P reduction, which may underpin its vascular protective trend. This supports the "calcification burden" hypothesis, whereby both calcium and phosphate loads drive extra-skeletal calcification.

A unique pleiotropic benefit of sevelamer observed in our study was a substantial LDL cholesterol reduction (−18.07 mg/dL, 15.3% decrease), compared to minimal change with calcium acetate (−2.11 mg/dL). This finding is strongly corroborated by prior literature. Bleyer et al. (1999) reported a 24% mean LDL decrease with sevelamer, while Patel et al. (2016) confirmed a mean difference of −21.6 mg/dL favoring sevelamer. [11, 14] Nolan & Qunibi (2005) highlighted this lipid-lowering effect as a potential mechanism for cardiovascular benefit. [13, 16] Yilmaz et al. (2012) further demonstrated improved endothelial function with sevelamer, likely linked to lipid and anti-inflammatory effects. [17] Mechanistically, sevelamer binds bile acids in the intestine, disrupting enterohepatic circulation and forcing hepatic cholesterol utilization. Clinically, LDL reduction of ~20 mg/dL is expected to confer a 10–15% relative risk reduction in cardiovascular events, based on statin trial data. [18] Given CKD patients' extreme cardiovascular risk, this pleiotropic benefit represents a major advantage of sevelamer over calcium-based binders.

Our study showed a strong trend toward reduced vascular calcification progression with sevelamer (8% vs. 24%; p=0.0538). Although not statistically significant, the relative reduction of 67% is clinically meaningful. Prior studies support this vascular benefit. Patel et al. (2016) found reduced coronary artery calcification progression with sevelamer, [14] while Nolan & Qunibi (2005) cited the Treat-to-Goal study showing slower progression of coronary and aortic calcification. [13, 16] Yilmaz et al. (2012) demonstrated improved endothelial function and significant FGF-23 reduction with sevelamer, providing mechanistic links to vascular health. [17] Several pathways may explain these benefits: reduced calcium load prevents vascular smooth muscle osteogenic transformation, improved Ca×P product reduces precipitation risk, LDL reduction decreases pro-calcific stimuli, and FGF-23 reduction mitigates vascular toxicity. Sevelamer may also exert anti-inflammatory effects by binding endotoxins in the gut. Collectively, these mechanisms support the vascular protection trend observed in our study.[19]

Our findings suggest that sevelamer offers superior phosphate control, markedly lower hypercalcemia risk, greater Ca×P reduction, substantial LDL lowering, and a trend toward vascular protection. These benefits are particularly relevant for patients with vascular calcification, hypercalcemia risk, dyslipidemia, or low PTH. Calcium acetate remains reasonable in cost-sensitive settings or low-risk patients. Cost-effectiveness remains a critical consideration, as sevelamer is substantially more expensive. A pragmatic "treat-to-target" approach may begin with calcium acetate and switch to sevelamer when goals are unmet or hypercalcemia develops. [20] Ultimately, individualized therapy is essential, balancing efficacy, safety, pleiotropic benefits, and cost.

While some findings diverge from prior literature, the consistent safety advantage of sevelamer, its pleiotropic LDL-lowering effect, and its potential vascular protection make it an attractive option for high-risk patients. Future research should focus on long-term outcomes, cost-effectiveness, and personalized approaches to optimize binder selection. In the meantime, our results reinforce the clinical rationale for preferential use of sevelamer in patients at greatest risk of cardiovascular and calcific complications.

CONCLUSION

This prospective, randomized, open-label comparative study demonstrates that sevelamer hydrochloride is more effective than calcium acetate in controlling serum phosphate levels in patients with stage 4-5 chronic kidney disease over a 12-week period, achieving significantly greater phosphate reduction and lower final phosphate concentrations. Critically, sevelamer exhibited a markedly superior safety profile with significantly fewer episodes of hypercalcemia, greater reduction in calcium-phosphate product, and substantial LDL cholesterol reduction, a pleiotropic benefit unique to sevelamer. These findings support sevelamer as a preferred phosphate binder in CKD patients at high cardiovascular risk, particularly those with pre-existing vascular calcification, susceptibility to hypercalcemia, or concomitant dyslipidaemia.

Declaration by Authors

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REFERENCES

1. Chen TK, Knicely DH, Grams ME. Chronic Kidney Disease Diagnosis and Management: A Review. JAMA. 2019 Oct 1;322(13):1294-1304. doi: 10.1001/jama.2019.14745.
2. Cernaro V, Longhitano E, Casuscelli C, Peritore L, Santoro D. Hyperphosphatemia in Chronic Kidney Disease: The Search for New Treatment Paradigms and the Role of Tenapanor. Int J Nephrol Renovasc Dis. 2024 May 28; 17:151-161. doi: 10.2147/IJNRD.S385826.

3. Raikou VD. Serum phosphate and chronic kidney and cardiovascular disease: Phosphorus potential implications in general population. World J Nephrol. 2021 Sep 25;10(5):76-87. doi: 10.5527/wjn. v10.i5.76.
4. Hruska KA, Mathew S, Lund R, Qiu P, Pratt R. Hyperphosphatemia of chronic kidney disease. Kidney Int. 2008 Jul;74(2):148-57. doi: 10.1038/ki.2008.130.
5. Natale P, Green SC, Ruospo M, Craig JC, Vecchio M, Elder GJ, Strippoli GF. Phosphate binders for preventing and treating chronic kidney disease-mineral and bone disorder (CKD-MBD). Cochrane Database Syst Rev. 2025 Jun 27;6(6):CD006023. doi: 10.1002/14651858.CD006023.pub4.
6. Wang Y, Xie G, Huang Y, Zhang H, Yang B, Mao Z. Calcium acetate or calcium carbonate for hyperphosphatemia of hemodialysis patients: a meta-analysis. PLoS One. 2015 Mar 23;10(3): e0121376. doi: 10.1371/journal.pone.0121376.
7. Mai ML, Emmett M, Sheikh MS, Santa Ana CA, Schiller L, Fordtran JS. Calcium acetate, an effective phosphorus binder in patients with renal failure. Kidney Int. 1989 Oct;36(4):690-5. doi: 10.1038/ki.1989.247.
8. Ferreira A, Frazão JM, Monier-Faugere MC, Gil C, Galvao J, Oliveira C, Baldaia J, Rodrigues I, Santos C, Ribeiro S, Hoenger RM, Duggal A, Malluche HH; Sevelamer Study Group. Effects of sevelamer hydrochloride and calcium carbonate on renal osteodystrophy in hemodialysis patients. J Am Soc Nephrol. 2008 Feb;19(2):405-12. doi: 10.1681/ASN.2006101089.
9. Iimori S, Mori Y, Akita W, et al. Effects of sevelamer hydrochloride on mortality, lipid abnormality and arterial stiffness in hemodialyzed patients: a propensity-matched observational study. Clin Exp Nephrol. 2012 Dec;16(6):930-7. doi: 10.1007/s10157-012-0640-4.
10. Yuste C, Mérida E, Hernández E, García-Santiago A, Rodríguez Y, Muñoz T, Gómez GJ, Sevillano Á, Praga M. Gastrointestinal complications induced by sevelamer crystals. Clin Kidney J. 2017 Aug;10(4):539-544. doi: 10.1093/ckj/sfx013.
11. Bleyer AJ, Burke SK, Dillon M, Garrett B, Kant KS, Lynch D, Rahman SN, Schoenfeld P, Teitelbaum I, Zeig S, Slatopolsky E. A comparison of the calcium-free phosphate binder sevelamer hydrochloride with calcium acetate in the treatment of hyperphosphatemia in hemodialysis patients. American journal of kidney diseases. 1999 Apr 1;33(4):694-701. DOI: 10.1016/S0272-6386(99)70221-0
12. Hervás JG, Prados D, Cerezo S. Treatment of hyperphosphatemia with sevelamer hydrochloride in hemodialysis patients: a comparison with calcium acetate. Kidney International. 2003 Jun 1;63: S69-72. DOI: 10.1046/j.1523-1755.63. s85.17.x
13. Qunibi WY, Hootkins RE, McDowell LL, Meyer MS, Simon M, Garza RO, Pelham RW, Cleveland MV, Muenz LR, He DY, Nolan CR. Treatment of hyperphosphatemia in hemodialysis patients: the Calcium Acetate Renagel Evaluation (CARE Study). Kidney international. 2004 May 1;65(5):1914-26. DOI: 10.1111/j.1523-1755.2004. 00590.x
14. Patel L, Bernard LM, Elder GJ. Sevelamer versus calcium-based binders for treatment of hyperphosphatemia in CKD: a meta-analysis of randomized controlled trials. Clinical Journal of the American Society of Nephrology. 2016 Feb 1;11(2):232-44. DOI: 10.2215/CJN.06800615
15. Habbous S, Przech S, Acedillo R, Sarma S, Garg AX, Martin J. The efficacy and safety of sevelamer and lanthanum versus calcium-containing and iron-based binders in treating hyperphosphatemia in patients with chronic kidney disease: a systematic review and meta-analysis. Nephrology Dialysis Transplantation. 2017 Jan 1;32(1):111-25. DOI: 10.1093/ndt/gfw312
16. Nolan CR, Qunibi WY. Treatment of hyperphosphatemia in patients with chronic kidney disease on maintenance hemodialysis. Kidney International. 2005 Jun 1;67: S13-20. DOI: 10.1111/j.1523-1755.2005. 09502.x
17. Yilmaz MI, Sonmez A, Saglam M, Yaman H, Kilic S, Eyileten T, Caglar K, Oguz Y, Vural A, Yenicesu M, Mallamaci F. Comparison of calcium acetate and sevelamer on vascular function and fibroblast growth factor 23 in CKD patients: a randomized clinical trial. American journal of kidney diseases. 2012 Feb 1;59(2):177-85. DOI: 10.1053/j.ajkd.2011.11.007
18. Cholesterol Treatment Trialists' (CTT) Collaborators; Mihaylova B, Emberson J, Blackwell L, Keech A, Simes J, Barnes EH,

Voysey M, Gray A, Collins R, Baigent C. The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. Lancet. 2012 Aug 11;380(9841):581-90. doi: 10.1016/S0140-6736(12)60367-5. Epub 2012 May 17. PMID: 22607822; PMCID: PMC3437972.

19. Sun PP, Perianayagam MC, Jaber BL. Endotoxin-binding affinity of sevelamer: a potential novel anti-inflammatory mechanism. Kidney Int. 2009 Dec;76(Suppl 114): S20-S25. doi:10.1038/ki.2009.403
20. Yusuf AA, Weinhandl ED, Peter WL. Comparative effectiveness of calcium acetate and sevelamer on clinical outcomes in elderly hemodialysis patients enrolled in Medicare part D. American journal of kidney diseases. 2014 Jul 1;64(1):95-103. DOI: 10.1053/j.ajkd.2013.11.015

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