

Rapid Systematic Narrative Review: Patiromer in Adults with Non-Dialysis-Dependent Chronic Kidney Disease

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Executive Summary (Patiromer in Adults with NDD-CKD; English; last 3 years)

- **Evidence base identified:** A sensitive PubMed/MEDLINE search (English; 2023-03-09 to 2026-03-09) retrieved 22 records; after conservative title/abstract screening, 4 primary clinical studies were eligible for extraction: 1 CKD feasibility trial, 2 real-world CKD retrospective cohorts, and 1 heart-failure randomized trial subgroup analysis reporting CKD/eGFR strata (PMIDs: 39156169; 38319545; 41791977; 39159624).
- **Potassium lowering in NDD-CKD cohorts:** In a single-center stage 3b–4 CKD cohort (n=40), mean serum potassium decreased from 5.77 ± 0.41 mmol/L at initiation to 5.13 ± 0.48 mmol/L at 2 months ($p < 0.001$), with 82.5% (33/40) achieving normokalaemia at 2 months; reductions were reported as sustained through 6 months (PMID: 38319545).
- **Durability over 12 months in routine care:** In a multicentre real-world cohort (n=59), baseline potassium 5.72 mmol/L decreased to 5.02 (1 month) and 5.01 mmol/L (12 months), with all timepoints reported as statistically significant vs baseline (all $P < .001$) (PMID: 41791977).
- **RAASi/MRA enablement signals:** RAASi continuation was reported as 93% (27/29) in the single-center CKD cohort and 94.9% in the multicentre cohort; MRA continuation was 98.3% in the multicentre cohort (PMIDs: 38319545; 41791977).
- **Randomized evidence in reduced eGFR strata (HF setting):** In the DIAMOND trial subgroup analysis, patiromer enabled achievement of RAASi/MRA targets during run-in in ~79–81% of participants with eGFR < 60 , < 45 , and < 30 mL/min/1.73 m², and the abstract reports greater potassium-control efficacy vs placebo at lower eGFR (interaction $p \leq 0.027$), though absolute effect sizes were not provided in the abstract (PMID: 39159624).
- **Safety/tolerability (abstract-reported):** Adverse events were described as mild–moderate in 27.5% of patients in the single-center CKD cohort; in the multicentre cohort, gastrointestinal events were most frequent and 13.5% discontinued patiromer, with adverse effects accounting for about half of discontinuations; DIAMOND subgroup analysis reported adverse effects similar to placebo across eGFR strata (PMIDs: 38319545; 41791977; 39159624).
- **Key limitations and strength of inference:** The recent, English-language evidence retrieved here is small and dominated by uncontrolled retrospective cohorts; abstracts often lacked essential details (CKD stage distribution including stage 5 not on dialysis, dosing, standardized outcome definitions, magnesium effects, and time-to-event outcomes). Consequently, estimates for clinically important outcomes (hospitalizations, arrhythmias, CKD progression, mortality) and for advanced CKD (stage 5 not on dialysis) remain uncertain without full-text verification and broader searching.

This review provides a rapid systematic narrative overview based primarily on title/abstract screening and available retrieved source material; findings should be interpreted as provisional.

Structured Abstract

Background: Hyperkalaemia is common in chronic kidney disease (CKD) and can constrain use or intensification of renin–angiotensin–aldosterone system inhibitors (RAASi) and mineralocorticoid receptor antagonists (MRAs). Patiromer is a non-absorbed potassium binder intended for chronic potassium control and may support ongoing RAASi/MRA therapy in non–dialysis-dependent CKD (NDD-CKD).

Methods: We conducted a rapid systematic narrative review with a transparent, reproducible workflow (scoping; sensitive search; conservative selection; structured extraction; narrative synthesis). PubMed/MEDLINE was searched via NCBI E-utilities on 2026-03-09 using a sensitive strategy combining patiromer terms with CKD, hyperkalaemia/potassium, and RAAS/RASi concepts. Limits were English language and publication date 2023-03-09 to 2026-03-09. Title/abstract screening applied conservative eligibility: adults with NDD-CKD (stages 1–5, including stage 5 not on dialysis) or an explicitly analyzable CKD subgroup; patiromer exposure; and extractable potassium, RAASi/MRA, or safety outcomes. Reviews/consensus statements were excluded from primary evidence. Data were extracted from available retrieved source material (predominantly PubMed abstracts) and synthesized narratively.

Results: The search retrieved 22 records; four primary clinical studies were eligible for extraction: one CKD feasibility trial, two retrospective real-world CKD cohorts, and one heart-failure randomized trial subgroup analysis reporting results by baseline estimated glomerular filtration rate (eGFR) strata. In a single-center stage 3b–4 CKD cohort (n=40), mean serum potassium decreased from 5.77 ± 0.41 mmol/L at initiation to 5.13 ± 0.48 mmol/L at 2 months ($p<0.001$) with 82.5% achieving normokalaemia; reductions were reported as sustained at 6 months. In a multicentre cohort (n=59), potassium decreased from 5.72 mmol/L at baseline to 5.02 at 1 month and 5.01 mmol/L at 12 months (all $P<.001$). RAASi continuation was reported as 93% (27/29) in the single-center cohort and 94.9% in the multicentre cohort; MRA continuation was 98.3% in the multicentre cohort. In the DIAMOND trial subgroup analysis (heart failure population), 78.9–81.3% of participants with reduced eGFR strata achieved prespecified RAASi/MRA targets during run-in, and the abstract reported greater potassium-control efficacy of patiromer versus placebo at lower eGFR (interaction $p\leq 0.027$), without providing full effect estimates. Safety reporting in abstracts suggested predominantly gastrointestinal adverse events and discontinuations in a minority (13.5% in the multicentre cohort), while the DIAMOND subgroup reported adverse effects similar to placebo across eGFR strata. The feasibility trial encountered substantial recruitment/randomization constraints, limiting inference on its albuminuria endpoint.

Conclusions: In the recent PubMed-indexed evidence captured by this review, patiromer use in CKD settings was consistently associated with lower serum potassium and high rates of RAASi/MRA continuation in observational cohorts, with tolerability signals mainly gastrointestinal. However, the evidence base retrieved within the last 3 years is small, largely observational, and frequently under-reported in abstracts; effects in advanced CKD (including stage 5 not on dialysis) and on clinical outcomes (hospitalization, mortality, CKD progression) remain uncertain without full-text verification and broader searching.

This review provides a rapid systematic narrative overview based primarily on title/abstract screening and available retrieved source material; findings should be interpreted as provisional.

Disclaimer (required): This systematic narrative review used structured search, title/abstract-based study selection, and extraction from available retrieved source material to provide a rapid overview of the topic. Accordingly, eligibility assessments, extracted details, and evidence appraisals should be interpreted as provisional.

Introduction

Hyperkalaemia is a clinically important electrolyte abnormality that becomes increasingly common as kidney function declines. Among people with chronic kidney disease (CKD), reduced urinary potassium excretion, intercurrent illness, and co-prescription of medications that reduce potassium excretion can all contribute to elevated serum potassium. In parallel, renin–angiotensin–aldosterone system inhibitors (RAASi) and mineralocorticoid receptor antagonists (MRAs) are widely used because they provide cardio-renal benefits in many patients with CKD and/or heart failure. However, these therapies can increase serum potassium and are frequently down-titrated or discontinued when hyperkalaemia occurs. This creates a recurring clinical tension: the therapies that improve long-term outcomes may be constrained by a short-term safety risk.

Patiromer is an oral, non-absorbed potassium binder designed for chronic management of hyperkalaemia. In routine practice, patiromer is commonly used in non-dialysis-dependent CKD (NDD-CKD) to lower serum potassium and potentially allow continuation of RAASi/MRA therapy. The present report focuses on adults with NDD-CKD (stages 1–5, explicitly including stage 5 not on dialysis) and asks: what does the most recent English-language evidence (past three years) show regarding patiromer’s potassium-lowering effect, its relationship to RAASi/MRA continuation or optimization, and its tolerability?

This review is intended to help clinicians and researchers rapidly understand what recent PubMed-indexed literature contributes to the patiromer-in-NDD-CKD evidence landscape. It is not intended to replace full systematic reviews, guideline methods, or full-text critical appraisal.

Review question and PICO

This review evaluates whether patiromer, compared with any or no comparator, improves potassium control and supports RAASi/MRA continuation in adults with non-dialysis-dependent CKD (stages 1–5, including stage 5 not on dialysis), while also assessing safety/tolerability and any reported clinical outcomes.

Table 1. PICO Matrix

Item	Specification for this review
Population (P)	Adults (≥ 18 years) with non-dialysis-dependent CKD (NDD-CKD), stages 1–5, explicitly including CKD stage 5 not on dialysis. Studies may include other diseases/populations (e.g., HF, diabetes) only if NDD-CKD is separately analyzed/extractable. Kidney transplant recipients (KTx) excluded.
Intervention (I)	Patiromer (any dose, regimen, duration; any setting).
Comparator (C)	Any comparator (placebo, usual care, dietary/medication adjustments, alternative potassium binders, active drug comparators, or no comparator in single-arm observational cohorts).
Outcomes (O)	Potassium control: serum potassium change, normalization, recurrence/incidence of hyperkalaemia, time to recurrence. RAASi enablement: continuation, dose maintenance/up-titration, discontinuation avoidance. Safety/tolerability: GI AEs, hypomagnesemia, discontinuation due to AEs, serious AEs. Clinical/utilization outcomes (if reported): hospitalizations/ED, CV events, mortality, eGFR trajectory/CKD progression. Patient-reported outcomes (if available).
Limits (applied in search/selection)	English language only. Publication date within the past 3 years: 2023-03-09 to 2026-03-09 (relative to today). Humans only.

Methods

Design and reporting approach

We conducted a rapid systematic narrative review using a staged workflow: Phase 1 scoping, Phase 2 sensitive search, Phase 3 conservative selection, Phase 4 structured extraction, and Phase 5 narrative synthesis. The emphasis was on transparency and reproducibility rather than exhaustive evidence capture. All quantitative values were taken exactly as reported in the retrieved PubMed source material (predominantly abstracts). When key details were not available in the retrieved material, fields were recorded as *Not Available* rather than inferred.

Information source

PubMed/MEDLINE was searched via the NCBI E-utilities API on 2026-03-09.

Search strategy and limits

Phase 2 (Search Strategy & Sources) was executed on 2026-03-09 (Europe/Rome) in PubMed/MEDLINE via NCBI E-utilities. The strategy was intentionally sensitive, combining broad patiromer terms with CKD/kidney disease terms, hyperkalaemia/potassium terms, and RAAS/RAS context terms without study-design filters to reduce the risk of missing eligible evidence. Execution limits were English language and publication date 2023-03-09 to 2026-03-09. PubMed API output reported a MeSH mapping warning for “Patiromer” [Mesh] (quoted phrase not found), but title/abstract terms remained active and retrieval completed successfully.

Total retrieval was 22 records (esearch IDs + esummary metadata), with downstream conservative selection planned at Phase 3 (adult NDD-CKD separability, excluding kidney transplant and dialysis-dependent populations).

Table 2. Study Selection matrix

PMID	Year	Journal	Title	Type
40152479	2025	Pharmacotherapy	CKD management update	SR
40434618	2025	Adv Ther	Cardiorenal therapies narrative review	NR
39577885	2024	Adv Kidney Dis Health	US nephrologists position statement	CS
39159624	2024	Am J Nephrol	DIAMOND subgroup (HF \pm CKD)	RCT-SA
40841869	2025	Heart Fail Rev	Hyperkalemia in HFrEF	NR
37545002	2023	Expert Opin Pharmacother	Hyperkalemia pharmacotherapy in CKD	NR
37648583	2024	Eur J Intern Med	Potassium binders to optimize RAASi in HF	SR/MA
37583425	2023	Front Med	Hyperkalemia in CKD overview	NR
39883259	2025	Heart Fail Rev	SZC & MRA optimization (REALIZE-K)	NR
37125304	2023	Eur Heart J Suppl	RAASi & hyperkalaemia treatment	JA
41233949	2025	Cardiol Rev	Hyperkalemia in HF systematic review	JA
38173862	2023	J Pharm Pharm Sci	Oral potassium resins in CKD	SR
39156169	2024	Kidney Int Rep	Patiromer/RAASi feasibility trial in CKD	JA
37775712	2024	Intern Emerg Med	Hyperkalemia management on RAASi	NR
36890464	2023	BMC Nephrol	UK cost-effectiveness of patiromer	JA
38319545	2024	J Nephrol	Patiromer in CKD real-world (single-center)	JA
38403867	2024	Nephrology (Carlton)	Asia-Pacific consensus hyperkalaemia	CS
40788620	2025	Eur J Heart Fail	CARE-HK in HF registry	MCS
41487544	2025/2026	Proc (Bayl Univ Med Cent)	Novel binders meta-analysis of RCTs	NR

36264349	2023	Nephrol Dial Transplant	MRAs & enabling RAASi via binders	JA
41791977	2026	Nefrologia (Engl Ed)	Real-world patiromer (Valencian Community)	JA
37695628	2023	J Cardiovasc Med	Practical appraisals new binders	JA

Legend: This table summarizes the PubMed Phase 2 output (n=22), listing publication year, journal, abbreviated title, and indexed study-type classification for downstream screening.

Note: SR = systematic review; SR/MA = systematic review and meta-analysis; NR = narrative review; CS = consensus statement; RCT-SA = randomized controlled trial subgroup analysis; JA = journal article; MCS = multicenter study.

Study selection (conservative screening)

Phase 3 study selection was conducted at title/abstract level on the 22 records retrieved in Phase 2. Screening basis was title plus abstract text (PubMed efetch abstract when available), with a conservative posture for off-scope populations and for records without extractable NDD-CKD signal. Eligibility retained primary studies with patiromer exposure in adult NDD-CKD (stages 1–5, including stage 5 not on dialysis), or mixed populations with explicitly separable CKD subgroup findings. Reviews, systematic reviews/meta-analyses, consensus/position statements, and non-primary evidence were excluded from extraction.

This process yielded 5 provisionally included records and 17 exclusions. One included record is an economic model (PMID 36890464), retained here as CKD-specific patiromer evidence and to be handled as economic/model evidence in extraction.

Table 3. Studies included after Phase 3 title/abstract screening

PMID	Study type (from abstract)	Rationale for inclusion vs PICO
39156169	Feasibility trial in CKD	Patiromer used in CKD context to enable RAASi intensification; adult CKD population (NDD implied; eGFR 25–60; trial setting).
38319545	Retrospective cohort (single-center)	Explicit stage 3b–4 CKD patients treated with patiromer; potassium outcomes + RAASi continuation reported.
41791977	Retrospective multicenter real-world study	CKD + hyperkalaemia treated with patiromer 3 months; potassium trajectory + RAASi/MRA continuation + AEs.
39159624	RCT subgroup analysis (DIAMOND)	HF trial with explicit CKD subgroup analyses by eGFR; evaluates patiromer vs placebo on potassium control and RAASi/MRA targets; CKD subgroup is extractable.

36890464	Cost-effectiveness model	CKD \pm HF decision model evaluating patiromer strategy for HK/RAASi maintenance; not primary clinical outcomes, but directly addresses patiromer use in CKD context.
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Table 4. Studies excluded at Phase 3 title/abstract screening

PMID	Citation short title	Primary reason for exclusion at Phase 3
40152479	CKD management update (systematic review)	Secondary synthesis (systematic review); not primary patiromer study for extraction.
40434618	Cardiorenal GDMT narrative review	Narrative review; no primary extractable patiromer NDD-CKD dataset.
39577885	US nephrologists position statement	Consensus/position statement; not primary study.
40841869	Hyperkalemia in HFrEF review	Review article; not primary study.
37545002	Hyperkalemia in CKD pharmacotherapy review	Review article; not primary study.
37648583	NPB to optimize RAASi in HF (SR/MA)	Systematic review/meta-analysis; not primary study for extraction.
37583425	Hyperkalemia in CKD overview	Narrative review/overview; not primary study.
39883259	SZC for MRA optimization (REALIZE-K lessons)	Review focused on SZC; not primary patiromer study.
37125304	RAASi & hyperkalaemia treatment overview	Overview article; no primary patiromer dataset.
41233949	Hyperkalemia in HF systematic review	Systematic review; not primary study.
38173862	Oral potassium resin use in CKD (SR)	Systematic review; not primary patiromer study.
37775712	Hyperkalemia management recommendations	Narrative review/recommendations; not primary study.
38403867	Asia-Pacific hyperkalaemia consensus	Consensus statement; not primary study.

40788620	CARE-HK in HF registry	Registry in HF high-risk HK; patiromer use reported but not a patiromer-focused CKD analysis and CKD/NDD subgroup not clearly extractable for patiromer outcomes from abstract.
41487544	NPB in chronic HK (SR/MA of RCTs)	Systematic review/meta-analysis; not primary study.
36264349	MRAs and enabling RAASi via binders	Commentary/review style article; not primary study.
37695628	Practical appraisals new binders	Practice appraisal/review; not primary study.

Data extraction

A structured template captured: identification; study design and setting; population criteria; intervention/comparator; follow-up; outcomes and reported effect estimates; and limited, provisional bias considerations based on abstract information. Economic models were excluded from extraction per user instruction.

Synthesis

Because included evidence was heterogeneous and often incompletely reported in abstracts, synthesis was narrative rather than meta-analytic. Evidence statements are separated from interpretation where helpful, and uncertainty is explicitly described.

Evidence overview and results

Search yield and included studies

The PubMed search retrieved 22 records. After conservative title/abstract screening, four primary clinical studies were included for extraction: one CKD feasibility trial, two real-world retrospective CKD cohorts, and one subgroup analysis from a randomized trial conducted in a heart failure population with eGFR-defined strata (used here as the CKD-relevant subgroup evidence).

Table 5. General Information

Author (Year)	Journal	PMID	Country	Setting
Mårup (2024) [1]	Kidney Int Rep	39156169	Denmark	Nephrology clinics; trial screening using laboratory databases; outpatient CKD context (abstract)
Riccio (2024) [2]	J Nephrol	38319545	Italy	Single-center retrospective CKD clinic experience (abstract)

Moncho-Francés (2026) [3]	Nefrologia (Engl Ed)	41791977	Spain	Multicentre retrospective routine-care study (abstract)
Weir (2024) [4]	Am J Nephrol	39159624	Multinational	Randomized trial subgroup analysis; HF population with CKD subgroups by eGFR (abstract)

Table 6a. Methods: Study Design, Sample Size, and Eligibility Criteria

PMID	Design	Sample_Size	Key_Eligibility_Criteria
39156169	Open-label randomized trial with run-in; feasibility-focused	Planned 140; 75 included; 23 randomized; 20 completed	Age 18–80; CKD with eGFR 25–60 ml/min/1.73m ² ; albuminuria; history of hyperkalaemia; randomized only if P-K \geq 5.5 mmol/L during intensified RAASi run-in
38319545	Retrospective observational cohort; plus cross-sectional postprandial substudy	40	Stage 3b–4 CKD; treated with patiomer; routine-care patients
41791977	Retrospective, observational, multicentre, non-interventional real-world study	59	Received patiomer \geq 3 months due to hyperkalaemia; CKD noted (stage not stated in abstract)
39159624	DIAMOND RCT subgroup analysis by baseline eGFR; randomized withdrawal	Not stated in abstract (subgroup proportions reported)	HFrEF with/without CKD; run-in required normokalaemia on patiomer while achieving \geq 50% recommended RAASi doses and MRA 50 mg/day

Table 6b. Methods: Interventions, Comparators, and Statistical Strategy

PMID	Intervention_or_Exposure	Comparator_or_groups	Statistical_Strategy
39156169	Patiomer introduced in those developing HK during RAASi intensification	Randomized to patiomer + maximally tolerated RAASi vs no patiomer + RAASi (randomization conditional on HK during run-in)	Not described in abstract
38319545	Patiomer initiation (T0)	Single group (within-person over time); cross-sectional fasting vs postprandial at T12	Not described in abstract (reported p-values for within-person comparisons)
41791977	Patiomer in routine care	Single group (within-person over time); RAASi/MRA continuation assessed	Not described in abstract (reported significance testing)

39159624	Patiromer (trial strategy)	Patiromer vs placebo; subgroups by eGFR ≥ 60 , ≥ 45 (prespecified), ≥ 30 (post hoc)	Interaction testing reported (p-interaction)
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Table 6c. Methods: Follow-Up Duration, Outcomes, and Source Basis

PMID	Follow_Up_Duration	Outcomes	Source_Basis
39156169	Not clearly stated in abstract	Primary endpoint: change in UACR (urine albumin-creatinine ratio); feasibility metrics (screening/yield), HK occurrence	Abstract
38319545	Follow-up at 2, 6, 12 months; postprandial sampling at T12	Serum K change over time; RAASi continuation; AEs; postprandial K vs fasting	Abstract
41791977	Potassium assessed at 1, 3, 6, 12 months	Primary: serum K evolution over time; Secondary: baseline characteristics; RAASi/MRA changes; AEs; discontinuation	Abstract
39159624	Not stated in abstract	Serum K control; achieving/maintaining RAASi/MRA targets; AEs across eGFR strata	Abstract

Potassium control

Evidence: Two real-world cohorts provided the most direct recent evidence for potassium lowering in CKD settings. In stage 3b–4 CKD (n=40), mean potassium decreased by 0.64 mmol/L from initiation to two months, with a high proportion achieving normokalaemia. In a multicentre cohort (n=59), potassium decreased from 5.72 mmol/L to near 5.0 mmol/L at 12 months, with statistically significant reductions at each measured timepoint. The DIAMOND subgroup analysis (heart failure setting) reported greater efficacy of patiromer compared with placebo at lower eGFR, but did not provide absolute potassium differences in the abstract.

Interpretation: Taken together, these findings are consistent with patiromer functioning as a chronic potassium-lowering therapy in CKD care. However, the observational cohorts have no control group and therefore cannot attribute potassium changes solely to patiromer; concomitant changes (dietary counseling, diuretic adjustments, intercurrent illness resolution, regression to the mean) could contribute. The randomized heart failure evidence supports a causal effect on potassium control, but the CKD-only magnitude and applicability to NDD-CKD without heart failure cannot be determined from the abstract.

RAASi/MRA continuation and optimization

Evidence: Both real-world cohorts reported high continuation rates of RAASi, and one reported high continuation of MRAs. The DIAMOND subgroup analysis provides a mechanistically aligned signal: during a run-in designed to achieve guideline-recommended RAASi/MRA targets while maintaining normokalaemia on patiromer, roughly four in five participants with low eGFR strata achieved those targets. The abstract also notes larger effects on secondary endpoints in the lower-eGFR subgroups, but without defining the endpoints or presenting effect sizes.

Interpretation: The observed continuation rates are consistent with the clinical rationale for patiromer: maintain potassium within a safer range to avoid RAASi/MRA interruption. Clinicians should interpret these findings as supportive rather than definitive. “Continuation” is not the same as “optimization”: the cohorts did not report dose titration, persistence over time, or standardized

criteria for “optimized” RAASi. Additionally, the DIAMOND evidence comes from a heart failure population, which may differ from CKD-only populations in comorbidity profile, medication regimen, and monitoring intensity.

Safety and tolerability

Evidence: Abstract-level safety reporting suggested adverse events were largely gastrointestinal and that discontinuation occurred in a minority (13.5% in one cohort). One cohort reported 27.5% experiencing adverse events, characterized as mild-to-moderate. In the DIAMOND subgroup, adverse effects were similar between patiromer and placebo across eGFR strata.

Interpretation: While the pattern of gastrointestinal tolerability aligns with the pharmacology of non-absorbed binders, abstracts did not provide key safety details relevant to CKD practice, such as hypomagnesemia frequency, constipation burden, drug–drug separation adherence, and serious adverse event definitions. For patient counseling and safety monitoring, full-text evaluation and product labeling should be used.

Renal outcomes and feasibility of “conditional patiromer” trial designs

Evidence: The CKD feasibility trial (D4-01) tested a pragmatic-like strategy: intensify RAASi and add patiromer only if hyperkalaemia develops ($P\text{-}K > 5.5$ mmol/L). The study encountered major constraints: despite screening 800,000 individuals with laboratory data, few candidates met criteria and fewer developed hyperkalaemia during run-in; only 23 were randomized and 20 completed the study, precluding a significant effect on the primary albuminuria endpoint (UACR).

Interpretation: This feasibility experience is informative for trialists. Designs requiring both eligibility for RAASi intensification and subsequent hyperkalaemia to trigger randomization may face recruitment limitations, especially if the incidence of hyperkalaemia under intensified therapy is lower than anticipated or if real-world prescribing patterns already select against high-risk candidates. As a result, renal surrogate endpoints may be challenging to evaluate in such conditional designs without broader eligibility, multi-site recruitment, or alternative designs.

Table 7. Results

PMID	Clinical_Outcome	Comparator_or_groups	Effect_Estimate
39156169	Feasibility (screening/enrollment yield)	Single cohort	High screening attrition; low enrollment yield suggests limited feasibility for full-scale efficacy inference
39156169	Hyperkalaemia incidence during run-in	Single cohort	Substantial run-in hyperkalaemia burden observed (23/75), indicating clinically relevant event frequency in this selected CKD population
39156169	Change in UACR	Patiromer + RAASi vs no patiromer + RAASi	Direction of renal benefit is inconclusive in abstract because trial was underpowered with very low completion

38319545	Serum potassium reduction (early, T2 vs T0)	Within-person	Clear early potassium lowering signal with statistically significant improvement and high normalization proportion
38319545	Serum potassium reduction (maintenance, T6 vs T0)	Within-person	Potassium reduction appears sustained through medium-term follow-up with significant difference vs baseline
38319545	RAASi continuation	Single cohort	High continuation rate suggests supportive RAASi enablement in routine care
38319545	Adverse events	Single cohort	Safety profile appears acceptable in abstract, with events described as mostly mild to moderate
38319545	Postprandial vs fasting serum potassium (T12)	Within-person	No meaningful postprandial-fasting difference detected at T12 (non-significant comparison)
41791977	Serum potassium reduction over follow-up	Within-person	Consistent potassium lowering trend across 1–12 months with reported significant improvements vs baseline
41791977	RAASi continuation	Single cohort	Very high continuation suggests patiromer may facilitate persistence of background RAASi therapy
41791977	MRA continuation	Single cohort	Very high MRA continuation indicates potential treatment-enabling effect in real-world use
41791977	Patiromer discontinuation	Single cohort	Discontinuation is moderate; adverse events contribute to a notable minority of stoppages
39159624	Achieving RAASi/MRA targets during run-in	eGFR ≥ 60 / ≥ 45 / ≥ 30	High target-achievement proportions across CKD strata suggest effective optimization during run-in
39159624	Serum potassium control efficacy	Patiromer vs placebo; by eGFR subgroups	Qualitatively greater potassium-control efficacy vs placebo in more advanced CKD subgroups per interaction results

39159624	Secondary endpoints (composite/other)	Patiromer vs placebo; eGFR ≥ 60 and ≥ 45	Secondary outcomes appear directionally more favorable with patiromer in lower eGFR strata, though abstract lacks effect-size detail
39159624	Adverse effects	Patiromer vs placebo across eGFR subgroups	Overall adverse-event profile appears comparable between treatment arms across CKD subgroup analyses

Table 8. Risk of Bias and Limitations

PMID	Funding_or_Sponsor	COI	Key_Limitations
39156169	Not Available (abstract)	Not Available (abstract)	Major recruitment/randomization shortfall; only 23 randomized and 20 completed; feasibility design limits inference on efficacy
38319545	Not Available (abstract)	Not Available (abstract)	Retrospective single-center; no control group; confounding and selection bias; limited reporting of AE specifics
41791977	Not Available (abstract)	Reported (multiple authors received honoraria/fees; some support for congress attendance)	Retrospective; small N=59; no comparator; CKD staging not reported in abstract; potential indication bias
39159624	Not Available (abstract); COI suggests industry involvement (employees/stock noted)	Extensive COI disclosed; includes employees/stock options in sponsor-related entities	Subgroup analysis; HF trial population (CKD subgroup, not CKD-only); limited abstract reporting of effect sizes/timepoints

Discussion

Overall interpretation

The recent PubMed-indexed evidence retrieved by this review supports three broad, cautious conclusions. First, patiromer use in CKD routine care is consistently associated with reductions in serum potassium over months of follow-up. Second, RAASi continuation rates reported in observational CKD cohorts are high during patiromer treatment, and randomized evidence from a heart failure setting suggests patiromer can support RAASi/MRA target achievement in participants with reduced eGFR. Third, tolerability signals in abstracts are largely gastrointestinal, with discontinuation in a minority, and randomized subgroup evidence reports adverse effects similar to placebo.

At the same time, the evidence base in the last three years (English; PubMed; this specific search string) is small and largely observational. Many clinically critical details are absent from abstracts: CKD stage distribution (especially stage 5 not on dialysis), dialysis exclusion criteria, patiromer dosing and adherence, concurrent interventions (dietary counseling, diuretic changes), and standardized outcome definitions. Therefore, while the direction of findings is coherent with patiromer's intended role, confidence in magnitude and generalizability remains limited.

Clinical implications

For clinicians managing NDD-CKD patients with hyperkalaemia, these findings provide supportive, recent real-world signals that patiromer can reduce serum potassium and may help preserve RAASi/MRA therapy. Practical implications include:

- **Monitoring:** Potassium monitoring at initiation and during follow-up is central, especially during RAASi/MRA initiation or titration.
- **Therapy enablement:** The evidence aligns with the strategy of using potassium binders to mitigate hyperkalaemia risk rather than reflexively discontinuing RAASi/MRA.
- **Tolerability:** Gastrointestinal adverse effects and discontinuation risk should be discussed with patients; in advanced CKD, clinicians should also be attentive to laboratory monitoring needs (e.g., magnesium), although rates were not reported in the extracted abstracts.

These implications should be interpreted cautiously due to the limited evidence scope and abstract-only extraction.

Limitations of this review

This report has several limitations that materially affect interpretation:

1. **Single database:** Only PubMed/MEDLINE was searched. Relevant studies indexed elsewhere (e.g., Embase) or in registries may be missing.
2. **Time and language restriction:** English-only and last-three-years limits were user-specified and may exclude pivotal earlier NDD-CKD trials.
3. **Abstract-limited extraction:** Many key data elements are not captured in abstracts. Risk-of-bias appraisal is therefore provisional.
4. **Conservative selection:** Studies without extractable CKD subgroup analysis at the abstract level were excluded, potentially omitting some relevant mixed-population evidence.

Future directions and research priorities

To strengthen evidence for patiromer in NDD-CKD (including stage 5 not on dialysis), future work could prioritize:

- **Stage-specific evidence:** Clear reporting and stratification by CKD stage, including explicit inclusion of stage 5 not on dialysis, with dialysis exclusion criteria.
- **RAASi/MRA optimization outcomes:** Standardized definitions (dose, persistence, time-at-target dose) and reporting of medication changes after hyperkalaemia episodes.
- **Safety detail:** Systematic capture and reporting of hypomagnesemia, constipation/diarrhea, serious adverse events, and discontinuation reasons.
- **Clinically important outcomes:** Hospitalizations, arrhythmia events, cardiovascular outcomes, CKD progression/eGFR slope, and mortality, including causal inference methods for observational studies.
- **Trial design feasibility:** Alternative designs to conditional randomization triggered by hyperkalaemia, which may be recruitment-limiting in some settings.

Conclusion

Within the past three years of English-language PubMed-indexed evidence retrieved by this review, patiromer use in CKD settings was consistently associated with lower serum potassium in observational cohorts and with high reported RAASi/MRA continuation. Randomized evidence from a heart failure trial subgroup analysis supports potassium control and RAASi/MRA enablement at reduced eGFR, but CKD-only effect sizes and broader clinical outcomes are not well characterized in the extracted abstracts. Overall, recent evidence supports patiromer's role in potassium management in NDD-CKD, but definitive conclusions about magnitude of benefit, advanced-stage applicability, and clinical outcomes require full-text verification and broader searching.

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