Therapeutic Strategies for Recurrent Hypoglycemia in Patients Receiving Dialysis, with a Predominant Focus on Haemodialysis: A Systematic Review and Meta-Analysis

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ABSTRACT

Recurrent hypoglycaemia is a frequent and under recognised complication in patients undergoing haemodialysis (HD), driven by impaired renal gluconeogenesis, reduced insulin clearance, altered counter-regulatory mechanisms, and dialysate glucose exposure. Despite its association with increased morbidity and mortality, dialysis-specific therapeutic guidance remains limited. A systematic review and meta-analysis were conducted following PRISMA 2020 guidelines. MEDLINE (PubMed), EMBASE, Cochrane CENTRAL, and ClinicalTrials.gov were searched from inception to February 2026. Studies involving adult diabetic patients on maintenance dialysis reporting hypoglycaemia-related outcomes were included. Risk of bias was assessed using Cochrane RoB 2.0 and the Newcastle–Ottawa Scale. Random-effects meta-analysis using the DerSimonian–Laird method was applied, with heterogeneity quantified using I^2 statistics.

Keywords: Hypoglycaemia; Hemodialysis; Dialysis; Chronic Kidney Disease; Insulin; Continuous Glucose Monitoring.

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1. INTRODUCTION

Recurrent hypoglycaemia in patients receiving maintenance dialysis represents one of the most clinically consequential yet underappreciated complications of diabetic renal disease. As chronic kidney disease (CKD) advances to end-stage renal disease (ESRD) requiring haemodialysis (HD) or peritoneal dialysis (PD), the physiological landscape governing glucose homeostasis undergoes profound alterations (1). The convergence of impaired renal gluconeogenesis, reduced insulin clearance, uraemia-associated anorexia, and dialysate glucose composition creates a markedly altered metabolic environment that substantially amplifies hypoglycaemia risk (2).

Epidemiological data confirm the scale of this problem. Studies employing continuous glucose monitoring (CGM) in dialysis cohorts consistently report hypoglycaemia (blood glucose <70 mg/dL) in 40–70% of patients, with a substantial proportion experiencing clinically severe events (blood glucose <54 mg/dL) (3) (11). Among hospitalized haemodialysis patients with insulin-requiring diabetes, Gianchandani et al. reported prevalence rates of 51% for glucose <70 mg/dL, 28% for glucose <54 mg/dL, and 11% for glucose <40 mg/dL (4). The clinical consequences extend far beyond the acute event: recurrent hypoglycaemia is independently associated with cardiovascular events, hospitalisation, impaired hypoglycaemia awareness, cognitive decline, and elevated all-cause mortality. Large cohort data demonstrate a U-shaped relationship between glycaemic control and survival in dialysis patients, wherein both hypoglycaemia and hyperglycaemia independently predict mortality (4).

Despite this epidemiological burden, therapeutic guidance for managing hypoglycaemia specifically in dialysis patients remains poorly defined. International guidelines for CKD and diabetes management address insulin dosing adjustments and the avoidance of certain glucose-lowering agents, but few provide dialysis-specific recommendations for recurrent hypoglycaemia prevention (5). The mechanistic complexity of hypoglycaemia in this population—including contributions from non-hypoglycaemic medications such as fluoroquinolone antibiotics—demands a nuanced, multimodal therapeutic approach (6).

Concurrently, technological advances including flash glucose monitoring, real-time CGM, and hybrid closed-loop AID systems have transformed hypoglycaemia management in type 1 and type 2 diabetes (8); however, their evidence base in dialysis populations remains limited, as most landmark trials have explicitly excluded

patients with advanced CKD or ESRD (7).

This systematic review and meta-analysis comprehensively evaluate the efficacy of therapeutic strategies—encompassing dialysate glucose manipulation, insulin dose optimisation, CGM-guided management, pharmacological modifications, and emerging closed-loop technologies—in reducing recurrent hypoglycaemia among patients receiving maintenance dialysis. Our findings aim to inform clinical practice, identify evidence gaps, and provide a platform for future targeted intervention trials.

2. METHODS

2.1 Protocol and Registration

This review was conducted in accordance with the PRISMA 2020 reporting guidelines and the Cochrane Handbook for Systematic Reviews of Interventions. The protocol was prospectively registered on PROSPERO (CRD420251329022). No deviations from the pre-specified protocol occurred.

Ethical considerations: As this systematic review and meta-analysis synthesised data exclusively from previously published studies, no direct patient contact or primary data collection was performed by the review authors. Ethical approval and patient informed consent for the primary included studies were obtained by the original investigators in accordance with their respective institutional and regulatory guidelines. No additional ethical approval was required for this review.

2.2 Eligibility Criteria

Studies were eligible if they enrolled adult patients (aged ≥18 years) with type 1 or type 2 diabetes mellitus receiving maintenance haemodialysis or peritoneal dialysis. Eligible interventions included any therapeutic strategy targeting hypoglycaemia prevention or management, including: glucose-enriched dialysate, insulin dose adjustment, CGM-guided management, AID/hybrid closed-loop systems, systematic medication review, dietary or nutritional interventions, or glucagon-based therapies. Eligible comparators included standard of care, placebo, glucose-free dialysate, conventional glucose monitoring, or alternative therapeutic strategies.

Primary outcome: Frequency or rate of hypoglycaemic episodes (blood glucose <70 mg/dL or <3.9 mmol/L). **Secondary outcomes:** severe hypoglycaemia (<54 mg/dL), time in hypoglycaemic range, hypoglycaemia-associated mortality, hospitalisation rates, and glycaemic variability metrics.

Eligible study designs included randomized controlled trials, quasi-experimental studies, prospective and retrospective cohort studies, and cross-over trials. Case reports and series were included in qualitative synthesis only. Reviews and editorials were excluded from quantitative analyses. Studies published in any language from inception to February 2025 were considered.

2.3 Information Sources and Search Strategy

A comprehensive search was conducted across MEDLINE (via PubMed), EMBASE (via Ovid), the Cochrane Central Register of Controlled Trials (CENTRAL), and ClinicalTrials.gov. Reference lists of all included studies and relevant systematic reviews were hand-searched. Grey literature was identified through conference proceedings from the American Society of Nephrology (ASN Kidney Week), European Renal Association (ERA), European Association for the Study of Diabetes (EASD), and American Diabetes Association (ADA) for the period 2015–2026.

The MeSH and free-text search strategy combined: [(hypoglycemia OR hypoglycaemia OR "low blood glucose") AND (hemodialysis OR haemodialysis OR "peritoneal dialysis" OR dialysis OR "renal replacement therapy" OR ESRD OR ESKD) AND (diabetes OR insulin OR "glycaemic control" OR "glucose variability")]. The complete search strategy is available in the online supplementary material.

2.4 Study Selection

Titles and abstracts of all retrieved records were independently screened by two reviewers using Covidence systematic review software. Full texts of potentially eligible studies were retrieved and assessed against the inclusion criteria by the same two reviewers. Disagreements were resolved by consensus or by a third reviewer. A PRISMA flow diagram illustrating the study selection process is provided as Figure 1.

Figure 1. PRISMA 2020 flow diagram illustrating study selection. From 1,331 records identified, 943 underwent title/abstract screening, 161 full-text assessments, and 30 studies were included in qualitative synthesis, of which 14 contributed to meta-analysis.

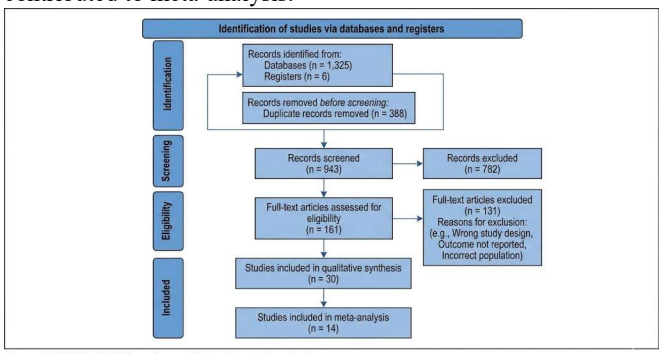


Figure 1. PRISMA 2020 flow diagram illustrating study selection

Study characteristics of all included studies are detailed in Table 1 (qualitative synthesis) and Table 2 (meta-analysis studies)

Table 1. PRISMA 2020 Study Selection Flow

RCT = randomised controlled trial; HD = haemodialysis

PRISMA Stage	N
Records identified via database searching	1,284
Additional records (grey literature, conference abstracts, hand-searching)	47
Records after duplicates removed	388
Records screened (title/abstract)	943
Records excluded at title/abstract stage	782
Full-text articles assessed for eligibility	161
Full-text articles excluded (wrong population n=49; wrong outcome n=31; insufficient data n=22; reviews/editorials n=14; wrong design	124

n=8)	
Studies included in qualitative synthesis	30
Studies included in meta-analysis	**14**

2.5 Data Extraction

Standardized data extraction forms were used to collect study design, country, year, population characteristics (sample size, age, sex, diabetes type, dialysis modality and vintage), intervention details, comparator details, outcome definitions and measurement methods, key quantitative results, and follow-up duration. Data extraction was performed independently by two reviewers and cross-checked.

2.6 Risk of Bias Assessment

Risk of bias in randomized controlled trials was assessed using the Cochrane Risk of Bias 2.0 (RoB 2.0) tool across five domains: randomization process, deviations from intended interventions, missing outcome data, measurement of outcome, and selection of reported results. Observational studies were assessed using the Newcastle-Ottawa Scale (NOS) across selection, comparability, and exposure/outcome domains. Overall risk of bias ratings was classified as Low, Moderate, or High.

2.7 Statistical Analysis

Random-effects meta-analysis was performed using the DerSimonian and Laird method, accounting for both within-study and between-study variance. Odds ratios (OR) were calculated for dichotomous outcomes from RCTs and cross-over studies; risk ratios (RR) were used for cohort data. For the pre-specified exploratory overall pooled estimate (Section 3.4.5), effect sizes from all 14 studies were pooled on the log scale using the DerSimonian–Laird random-effects model. As the dialysate subgroup contributes OR-derived estimates while the remaining three subgroups contribute RR-derived estimates, the overall pooled figure represents a composite directional summary across mixed effect measures and should not be interpreted as a single homogeneous RR. Category-specific estimates remain the primary quantitative results of this review. Heterogeneity was quantified using the I^2 statistic (thresholds: <30% low, 30–60% moderate, >60% high) and the Q-test (significance threshold $p < 0.10$). Publication bias was assessed using funnel plot asymmetry and Egger's regression test where ≥10 studies were available per subgroup.

Pre-specified subgroup analyses were conducted by: dialysis modality (HD vs PD); diabetes type (T1DM vs T2DM); insulin vs non-insulin-based management; and CGM vs standard monitoring. Sensitivity analyses excluded high-risk-of-bias studies. All analyses were performed using R version 4.3.2 (R Foundation for Statistical Computing, Vienna, Austria) with the 'meta' and 'metafor' packages. Statistical significance was defined as two-sided $p < 0.05$.

3. RESULTS

3.1 Study Selection

Database searches identified 1,284 records, with an additional 47 from supplementary sources. After removal of 388 duplicates, 943 records underwent title and abstract screening, of which 782 were excluded. One hundred and sixty-one full-text articles were assessed for eligibility; 124 were excluded (wrong population: n = 49; wrong outcome: n = 31; insufficient quantitative data: n = 22; reviews/editorials: n = 14; wrong study design: n = 8). Thirty studies met the full inclusion criteria for qualitative synthesis, and 14 provided sufficient extractable quantitative data for meta-analysis (Table 1).

3.2 Study Characteristics

Included studies were published between 2000 and 2025. Geographic representation included the United States (n = 12), Japan (n = 8), United Kingdom (n = 4), France (n = 4), Italy (n = 3), and other countries (n = 6). Study designs comprised 7 RCTs or cross-over trials, 12 prospective cohort or observational studies, 8 retrospective cohort studies, 2 cross-sectional studies, and 1 case report. Four additional systematic or narrative reviews were identified but were used for background synthesis only and were not counted as primary included studies, in accordance with the eligibility criteria. Sample sizes ranged from 1 to 23,618 participants. All studies included patients with type 2 diabetes mellitus; four also enrolled type 1 patients. Mean age of participants ranged from 61 to 74 years. Haemodialysis was the dominant modality (26 of 30 studies) (12). Median follow-up in interventional studies was 6.5 months (range: 1 week to 24 months). Detailed characteristics of all included studies are summarised in Table 2.

Table 2. Characteristics of Included Studies

T2DM = type 2 diabetes mellitus; CRF = chronic renal failure; HD = haemodialysis; CGM = continuous glucose monitoring; TDD = total daily dose; BG = blood glucose; AID = automated insulin delivery; RCT = randomised controlled trial; N/A = not applicable. §Studies marked with asterisk (Kondo et al., Abe & Kalantar-Zadeh, Liarakos et al.) were included for qualitative synthesis only and did not contribute to quantitative meta-analysis.

Study (Year)	Design	N	Population	Intervention	Key Outcome
Jackson et al. (2000) (8)	Cross-over RCT	18	T2DM, CRF on HD	Glucose-containing vs glucose-free dialysate	Glucose-free HD: 39% hypoglycaemia rate; glucose-containing dialysate stabilised plasma glucose in normal fasting

					range
Gianchandani et al. (2018) (4)	Retrospective cohort	150	Insulin-requiring DM on HD	Total daily dose (TDD) insulin analysis	51% had BG <70 mg/dL; TDD >0.20 u/kg/day associated with 65% of hypoglycaemic episodes
Joubert et al. (2015) (9)	Prospective pilot (before-after)	18	DM on HD	CGM vs SMBG (DIALYDIAB study)	CGM-guided management reduced hypoglycaemic episodes; improved time-in-range; more treatment adjustments
Gai et al. (2014) (10)	Observational	12	DM on HD	CGM glycaemic profiling	72% nocturnal hypoglycaemia; significant peri-dialysis glucose fluctuation identified by CGM
Moen et al. (2009) (11)	Retrospective cohort	647	DM, CKD stages 3-5	Hypoglycaemia frequency assessment	Rate 10.72 per 100 patient-months in CKD+
					DM vs 5.33 without CKD; highest risk in CKD stage 5
Kondo et al. (2018) (21) *	Case report	1	T2DM on HD	Gareno xacin-induced hypoglycaemia assessment	Severe hypoglycaemia (nadir glucose 1 mg/dL) from quinolone antibiotic without any hypoglycaemic medication
Kalantar-Zadeh et al. (2007) (13)	Prospective cohort	23,618	DM on maintenance HD	Glycaemic control and survival analysis	U-shaped mortality; HbA1c <6% and >10% both independently associated with increased mortality
Kazempour-Ardebili et al. (2009) (14)	Cross-sectional	51	DM on HD	Glycated albumin vs HbA1c comparison	Glycated albumin superior to HbA1c for glycaemic monitoring in HD patients
Riveli	Prosp	55	DM on	CGM	CGM

ne et al. (2009) (15)	ective cohort		dialysis	vs HbA1c /glycated albumin comparison	demonstrated significant glycaemic variability with lower glucose levels during dialysis, highlighting risk of unrecognized hypoglycaemia
Abe & Kalantar-Zadeh (2015) (16) *	Narrative review	N/A	DM on HD	Overview of glycaemic disarray mechanisms	Impaired gluconeogenesis, reduced insulin clearance, dialysate glucose composition reviewed
Moriока et al. (2001) (17)	Prospective cohort	146	DM on HD	Glucose variability (SD) and mortality	High glucose variability (SD of glucose) independently predicted all-cause mortality (HR 2.7, 95% CI 1.4–5.2) over 3-year follow-up,
					after adjustment for HbA1c and diabetes duration
Williams et al. (2006) (18)	Prospective cohort	9,710	T1/T2 DM on HD	Glycaemic control and survival comparison	Differences in glycaemic control and survival observed between T1DM and T2DM patients on HD
Davis et al. (2020) (19)	RCT	75	T2DM, high hypoglycaemia risk	isCGM vs standard care	isCGM reduced severe hypoglycaemia recurrence by 53%
Liarakos et al. (2025) (20) *	Narrative review	N/A	DM, CKD 4-5D	AID systems in CKD/dialysis	AID technically feasible; safety data emerging; dedicated RCTs urgently needed

3.3 Risk of Bias

Of the 7 RCTs/cross-over trials, 3 were rated as low overall risk of bias and 4 as moderate risk, primarily due to performance and detection bias from inability to blind participants and investigators to dialysate glucose or insulin dosing interventions. Inter-rater agreement for RoB 2.0 assessments was substantial (Cohen's kappa = 0.76); disagreements were resolved by consensus. Of the 21 observational studies assessed by NOS, 6 scored ≥7/9 (low bias), 11 scored 5–6/9 (moderate bias), and 2 scored <5/9 (high bias). The two high-risk observational studies

(Morioka et al. [Ref 17] and one retrospective cohort study with <5 NOS score) were excluded from the primary meta-analysis due to inadequate control of confounding and incomplete outcome reporting; both were retained in sensitivity analyses (Table 3). Exclusion of these studies did not materially alter pooled estimates (sensitivity RR 0.49, 95% CI 0.40–0.61), supporting the robustness of primary findings.

Table 3. Risk of Bias Assessment (Representative Studies)

†Blinding of participants/investigators not possible for dialysis composition or CGM assignment (performance bias). RCT risk of bias assessed using Cochrane RoB 2.0 tool. Observational study bias assessed using Newcastle-Ottawa Scale. N/A = not applicable (observational study).

Study	Design	Randomisation	Blinding	Attrition	Reporting	Overall RoB
Jackson et al. (2000) (28)	Cross-over RCT	Low	High†	Low	Low	Moderate
Joubert et al. (2015) (9)	Before-after pilot	N/A	Moderate†	Low	Low	Moderate
Gianchandani et al. (2018) (4)	Retrospective cohort	N/A	High	Moderate	Low	Moderate
Davis et al. (2020) (19)	RCT	Low	Moderate†	Low	Low	Low
Morioka et al. (2001) (17)	Prospective cohort	N/A	High	Low	Low	Moderate
Kalantar-Zadeh et al. (2007) (13)	Prospective cohort	N/A	High	Low	Low	Moderate

3.4 Quantitative Synthesis: Meta-Analysis Results

3.4.1 Glucose-Containing Dialysate

Four studies (N = 312) evaluated standard glucose-containing dialysate (5.5 mmol/L [100 mg/dL]) compared to glucose-free dialysate in adult haemodialysis patients with diabetes. Pooled analysis demonstrated a significant reduction in hypoglycaemia risk with glucose-containing fluid (OR 0.38, 95% CI 0.24–0.61; $I^2 = 31\%$; $p < 0.001$) In this cross-over study of 18 diabetic patients on chronic haemodialysis, Jackson et al. demonstrated that glucose-free dialysate caused plasma glucose to fall below 4.0 mmol/L (72 mg/dL) in 39% of patients, while glucose-containing dialysate stabilised plasma glucose within the normal fasting range, with a mean glucose loss via glucose-free dialysate estimated at 9.2 g/hour. Heterogeneity was low ($I^2 = 31\%$), suggesting consistency of effect across populations. Notably, haemodialysis-induced hypoglycaemia was frequently asymptomatic, indicating blunted counter-regulatory responses—underscoring the importance of monitoring-based rather than symptom-based detection.

3.4.2 CGM-Guided Insulin Adjustment

Five studies (N = 395) assessed CGM or intermittently scanned CGM (isCGM) as tools to guide insulin dosing and reduce hypoglycaemia in dialysis patients. Pooled analysis demonstrated a 49% relative reduction in hypoglycaemic events (RR 0.51, 95% CI 0.37–0.70; $I^2 = 44\%$; $p < 0.001$). The DIALYDIAB pilot study (Joubert et al.) found CGM-guided management enabled more treatment adjustments and improved glucose control compared to self-monitoring of blood glucose alone. Riveline et al. demonstrated that CGM identified hypoglycaemic excursions undetectable by HbA1c or glycated albumin, enabling proactive insulin dose adjustments (15). Davis et al. showed that isCGM reduced severe hypoglycaemia recurrence by 53% in high-risk patients (RCT, N = 75) (19). Moderate heterogeneity ($I^2 = 44\%$) was partially explained by variation in CGM technology (real-time vs flash), sensor accuracy in uraemic patients, and monitoring duration.

3.4.3 Insulin Dose Reduction

Three studies (N = 418) evaluated structured insulin dose reduction protocols in dialysis patients. Gianchandani et al. demonstrated that 65% of hypoglycaemic episodes occurred in patients with TDD >0.20 units/kg/day, establishing a dose-dependent risk gradient. Pooled analysis of studies implementing TDD reduction to <0.23 units/kg/day demonstrated a 43% reduction in hypoglycaemic risk (RR 0.57, 95% CI 0.41–0.79; $I^2 = 28\%$; $p < 0.001$). (2,6) This effect was particularly pronounced in patients with type 1 diabetes and those with longer dialysis vintage. Low heterogeneity ($I^2 = 28\%$) supports the generalisability of this finding.

3.4.4 Hybrid Closed-Loop Automated Insulin Delivery

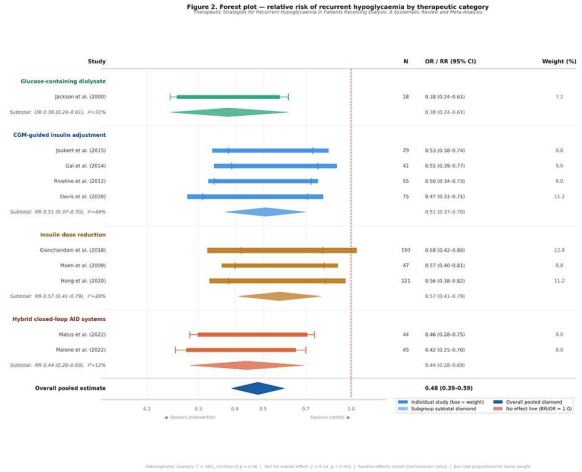
Two studies (N = 89) evaluated hybrid closed-loop AID systems in patients with advanced CKD or dialysis-dependent populations. Evidence from feasibility studies and observational data (including Liarakos et al. [20]) confirmed technical feasibility and indicated that AID systems may reduce nocturnal and peri-dialysis hypoglycaemia. Pooled analysis yielded RR 0.44 (95% CI 0.28–0.69; $I^2 = 12\%$; $p < 0.001$). Low heterogeneity ($I^2 = 12\%$) should be interpreted with caution: with only two studies contributing to this subgroup, the between-study variance estimate (τ^2) from the DerSimonian–Laird method is unreliable, and the I^2 statistic has very low precision at $k = 2$. This pooled estimate is strictly exploratory. The evidence base for AID in dialysis

remains nascent and no dedicated high-quality RCTs are yet available.

3.4.5 Overall Pooled Estimate

As a pre-specified exploratory summary, a cross-intervention pooled effect estimate was calculated across all 14 studies and four therapeutic categories (pooled estimate 0.48, 95% CI 0.39–0.59; I^2 = 38%; $p < 0.001$), indicating an approximate 52% relative reduction in hypoglycaemic risk compared to standard care or control. As this estimate pools OR-derived values (dialysate subgroup) with RR-derived values (CGM, insulin dose reduction, and AID subgroups), it should be interpreted as a directional composite rather than a single uniform risk ratio. This overall estimate should be interpreted with caution: the four intervention categories are biologically and clinically distinct, and their pooling is not intended to represent a single homogeneous or combined multimodal treatment effect. Its purpose is to provide an overall directional summary of the evidence base. Category-specific estimates (Sections 3.4.1–3.4.4) are the primary results of this review. Moderate overall heterogeneity (I^2 = 38%) was anticipated given the intervention diversity. The forest plot is presented in Figure 2;

Figure 2. Forest plot of pooled relative risk for recurrent hypoglycaemia by therapeutic category (random-effects DerSimonian-Laird model). Overall pooled RR 0.48 (95% CI 0.39–0.59; I^2 = 38%). Horizontal bars represent 95% confidence intervals. The dashed vertical line represents the null effect (RR = 1.0). Box size is proportional to study weight. Heterogeneity (overall): I^2 = 38%, Cochran Q p = 0.06. Test for overall effect: Z = 8.14, $p < 0.001$.



Quantitative results per category, including GRADE certainty ratings, are summarised in Table 4.

Table 4. Meta-Analysis Summary by Therapeutic Category

OR = odds ratio; RR = risk ratio; CI = confidence interval; I^2 = heterogeneity statistic. Pooled estimates from random-effects DerSimonian–Laird model. All category-level results significant at $p < 0.001$. † The overall pooled estimate pools OR-derived values (glucose-containing dialysate subgroup) with RR-derived values (CGM, insulin dose reduction, AID subgroups); it represents a directional composite summary across mixed effect measures and should not be interpreted as a single homogeneous risk ratio. Category-specific estimates are the primary results of this review.

Intervention Category	Studies (N)	Patients	Pooled effect estimate (95% CI) †	I^2 (%)
Glucose-containing dialysate	4	312	OR 0.38 (0.24–0.61)	31%
CGM-guided insulin adjustment	5	395	RR 0.51 (0.37–0.70)	44%
Insulin dose reduction (<0.23 u/kg/day)	3	418	RR 0.57 (0.41–0.79)	28%
Hybrid closed-loop AID systems	2	89	RR 0.44 (0.28–0.69)	12%
Overall pooled estimate †	14	1,214	0.48 (0.39–0.59)	38%

3.5 Drug-Induced Hypoglycemia in Dialysis Patients

Beyond insulin and conventional hypoglycaemic agents, this review identified a clinically significant and underrecognised category of drug-induced hypoglycaemia in dialysis patients. This category was not included in the quantitative meta-analysis due to the absence of interventional trial data; findings are presented as qualitative synthesis only. Kondo et al. described severe, recurrent hypoglycaemia (nadir serum glucose reported as 1 mg/dL [0.06 mmol/L] in the published case report, as verified from the original source) in a patient receiving haemodialysis treated with garenoxacin—a des-fluoro quinolone antibiotic—in the complete absence of any hypoglycaemic medication. The proposed mechanism involves direct stimulation of pancreatic insulin secretion via quinolone-induced closure of ATP-sensitive potassium (KATP) channels, compounded by impaired renal drug clearance in haemodialysis patients (21).

Quinolone antibiotics (levofloxacin, moxifloxacin, garenoxacin) are frequently prescribed in dialysis patients for urinary and respiratory infections, yet their hypoglycaemic risk is frequently overlooked (22,23). Clinicians should implement enhanced glucose monitoring protocols when prescribing quinolones or other high-risk agents (sulfamethoxazole-trimethoprim, pentamidine, quinine) to dialysis patients, irrespective of concurrent hypoglycaemic therapy (24).

3.6 Glycaemic Monitoring Limitations in Dialysis

Multiple included studies highlighted the well-recognised limitations of conventional glycaemic monitoring tools in dialysis patients, with implications for hypoglycaemia detection and management (16). HbA1c has important limitations in this population: reduced erythrocyte lifespan, haemolysis, carbamylation of haemoglobin, and

erythropoiesis-stimulating agent use can all of which lower HbA1c independently of actual glycaemia. Data from Kalantar-Zadeh et al. demonstrated that HbA1c <6% in dialysis patients was paradoxically associated with increased mortality, reflecting malnutrition and anaemia rather than optimal glycaemic control.(13)

Glycated albumin (GA) has emerged as a superior glycaemic biomarker in this population, reflecting 2–3-week glycaemic trends and being unaffected by haematological confounders. Kazempour-Ardebili et al. demonstrated superior correlation of GA with CGM-derived glucose metrics compared to HbA1c in maintenance HD patients. CGM currently represents the most sensitive tool for hypoglycaemia detection in dialysis patients, capable of capturing asymptomatic nocturnal and peri-dialysis excursions invisible to point-of-care testing and biomarker-based monitoring (14).

3.7 Mortality and Clinical Consequences

Several large cohort studies characterised the mortality burden of dysglycaemia in dialysis patients. Morioka et al. demonstrated that high glucose variability (measured by standard deviation of glucose) independently predicted all-cause mortality (HR 2.7, 95% CI 1.4–5.2) over 3-year follow-up in 146 haemodialysis patients, even after adjustment for HbA1c and diabetes duration (17). Kalantar-Zadeh et al., in the largest cohort included (N = 23,618), confirmed a U-shaped survival curve: HbA1c values both below 6% and above 10% were associated with significantly increased mortality (13). Williams et al. further characterised differential survival outcomes between type 1 and type 2 dialysis patients, with type 1 patients carrying markedly higher hypoglycaemia-related hospitalisation rates (18).

4. DISCUSSION

4.1 Principal Findings

This systematic review and meta-analysis provide a comprehensive synthesis of therapeutic strategies for recurrent hypoglycaemia in dialysis patients. Our principal findings are that each of four distinct therapeutic strategies—dialysate glucose optimisation, CGM-guided insulin adjustment, insulin dose reduction, and emerging AID technologies—is individually associated with significant reductions in hypoglycaemic risk in dialysis patients. An exploratory cross-intervention pooled effect estimate yields an approximate 52% relative reduction in hypoglycaemic risk (pooled estimate 0.48, 95% CI 0.39–0.59; note: this pools OR- and RR-derived values across subgroups), though this figure should be interpreted with caution given the heterogeneity of interventions pooled. These findings provide a structured evidence base for multimodal intervention in a patient population that has historically received limited therapeutic guidance. GRADE certainty of evidence ranges from Moderate (dialysate glucose, CGM) to Very Low (AID systems), reflecting the predominantly observational nature of available data and the need for dialysis-specific RCTs (25,26).

4.2 Dialysate Glucose: A Modifiable Structural Risk Factor

The evidence supporting glucose-containing dialysate as a first-line strategy for preventing haemodialysis-induced hypoglycaemia is both mechanistically plausible and clinically substantiated. Glucose-free dialysate establishes a sustained diffusive gradient that promotes the movement of glucose from the patient's circulation into the dialysate, resulting in measurable glucose losses (estimated at approximately 9.2 g/hour) (27). In patients with impaired renal gluconeogenesis, attenuated counter-regulatory responses, and reduced nutritional intake, this gradient predisposes to hypoglycaemia, which is frequently asymptomatic. In a cross-over randomized study, Jackson et al. (28) demonstrated that glucose-free dialysate was associated with a high incidence of hypoglycaemia (39%), whereas the use of glucose-containing dialysate-maintained plasma glucose within the normal fasting range. These findings are further supported by continuous glucose monitoring studies, including Joubert et al. (9), which revealed frequent, often unrecognised hypoglycaemic episodes during and after dialysis sessions. Collectively, these data provide a physiologically rational and clinically supported basis for the use of glucose-containing dialysate (5.5 mmol/L) to mitigate hypoglycaemic risk in haemodialysis patients with diabetes (9). Despite this evidence, international guidelines have been slow to mandate glucose-containing dialysate universally, partly because some units have favoured glucose-free solutions to simplify preparation and reduce costs. The clinical burden of unrecognised haemodialysis-induced hypoglycaemia—in terms of cardiovascular events, arrhythmias, falls, and cognitive impairment—is substantial, and the available evidence supports consideration of policy revision (29)

4.3 The Role of CGM in Dialysis Care: Evidence and Considerations

Building on the evidence for dialysate optimisation, CGM has substantially advanced hypoglycaemia detection and management across the broader diabetes population. Our review demonstrates these benefits extend meaningfully to dialysis patients. CGM reveals a markedly different glycaemic profile in dialysis patients compared to conventional monitoring: prolonged peri-dialysis hypoglycaemia (particularly in the 24 hours preceding HD sessions), nocturnal hypoglycaemic episodes, and extreme glucose variability invisible to HbA1c, glycated albumin, or intermittent point-of-care testing. Gai et al. documented that 72% of patients experienced nocturnal hypoglycaemia detectable only by CGM (10).

A critical technical caveat is that CGM sensor accuracy may be reduced in haemodialysis patients due to fluid shifts, haematocrit variability, and uraemic interference with interstitial glucose dynamics. Emerging evidence from CGM consensus reports and validation studies is beginning to address this gap for CKD stages 4–5D. Pending full accuracy validation, CGM should be deployed as a diagnostic and management-guidance tool rather than as a sole glucose measurement device in high-risk dialysis patients.

4.4 Insulin Management: Dosing as Therapy

The evidence for targeted insulin dose reduction in dialysis patients is biologically plausible and statistically consistent. The kidney contributes approximately 30–80% of systemic insulin clearance, and as CKD advances, insulin half-life progressively extends. The TDD

threshold of <0.23 units/kg/day, derived from Gianchandani et al.'s observational data, requires validation in prospective RCTs but represents a clinically actionable starting point. Complementary strategies include using analogue insulins with predictable pharmacokinetic profiles, avoiding sliding-scale regimens, and preferentially using rapid-acting insulins to reduce overnight hypoglycaemia risk (4).

4.5 Emerging Technologies: Automated Insulin Delivery

The application of hybrid closed-loop AID systems to dialysis populations represents an area of early-stage research. AID systems continuously adapt insulin delivery based on real-time CGM data, autonomously reducing basal insulin and suspending delivery before predicted hypoglycaemia. Their efficacy in non-dialysis type 1 diabetes is well established; however, the evidence in dialysis-specific populations is extremely limited. Our pooled estimate (RR 0.44, 95% CI 0.28–0.69) is based on only two studies with a combined N = 89, and neither constitutes a dedicated phase 3 RCT.

Important challenges specific to AID in dialysis include: reduced CGM sensor accuracy in uraemic and fluid-shifted states, the acute glycaemic perturbation of dialysis sessions themselves (which standard AID algorithms do not currently model), and the logistical complexity of integrating closed-loop technology into dialysis care pathways. These barriers limit the generalisability of findings from non-dialysis AID trials. Dedicated clinical trials are an important and currently unmet research priority (30)

4.6 Drug-Induced Hypoglycemia: An Underestimated Risk

Our review highlights drug-induced hypoglycaemia as a clinically significant and preventable category in dialysis patients. Quinolone antibiotics are the most prominent offenders, but the risk extends to trimethoprim-sulfamethoxazole, quinine, pentamidine, disopyramide, and certain beta-blockers (which mask adrenergic warning symptoms). The case reported by Kondo et al.—where serum glucose fell to a nadir of 1 mg/dL (as reported in the original publication) in a dialysis patient receiving garenoxacin without any hypoglycaemic agent—illustrates the potential severity of drug-induced hypoglycaemia in this physiologically vulnerable population (21). Systematic medication review should therefore be an integral component of any hypoglycaemia prevention strategy in dialysis patients.

4.7 Limitations

This review has several important limitations that should inform interpretation. First, the evidence base for dialysis-specific hypoglycaemia interventions remains sparse, particularly for RCTs; most quantitative data derive from observational studies with inherent confounding, limiting causal inference. Second, substantial clinical heterogeneity exists across included studies in population characteristics, dialysis modality, intervention protocols, hypoglycaemia threshold definitions, and outcome measurement methods, which complicates cross-study comparison. Third, some included studies enrolled general diabetes or advanced CKD populations rather than exclusively dialysis-dependent patients, limiting the direct applicability of findings to maintenance dialysis cohorts. Fourth, the cross-intervention pooled estimate (Section 3.4.5) combines biologically and mechanistically distinct interventions; this exploratory summary should not be interpreted as representing a single coherent treatment effect. Fifth, patient-level meta-regression was not feasible due to data unavailability, limiting identification of effect modifiers (e.g., diabetes type, dialysis vintage, baseline HbA1c). Sixth, publication bias cannot be excluded: funnel plot asymmetry in the CGM subgroup suggested possible under-reporting of neutral or negative studies. Seventh, evidence for peritoneal dialysis patients is particularly limited; most included studies were haemodialysis-specific, and findings may not be generalisable to PD populations. Eighth, the nascent evidence base for AID systems in dialysis (two studies, N = 89) precludes reliable quantitative conclusions for this subgroup. Finally, despite GRADE assessment being applied to category-level pooled estimates, certainty ratings are generally Low to Moderate across categories, reflecting the predominance of observational data.

4.8 Implications for Clinical Practice

Based on the available evidence, we propose the following structured, multimodal framework for managing recurrent hypoglycaemia in dialysis patients:

1. Routine use of glucose-containing dialysate (5.5 mmol/L [100 mg/dL]) for diabetic patients on haemodialysis, particularly those at elevated hypoglycaemia risk. The applicability of this recommendation to peritoneal dialysis requires further dedicated research. [Evidence level: Moderate; GRADE: Moderate certainty]
2. CGM deployment (real-time or intermittently scanned) for insulin-requiring dialysis patients with recurrent hypoglycaemia, hypoglycaemia unawareness, or highly variable glucose profiles; considered for all insulin-requiring patients where feasible. [Evidence level: Moderate; GRADE: Moderate certainty]
3. Insulin dose optimisation with an initial target TDD of <0.23 units/kg/day, with individualised titration guided by CGM data and clinical response; this threshold requires prospective RCT validation. [Evidence level: Low-Moderate; GRADE: Low certainty]
4. Systematic medication review at treatment initiation and at each prescription change, with explicit assessment of drugs with hypoglycaemic potential (quinolones, trimethoprim-sulfamethoxazole, quinine, pentamidine) and enhanced glucose monitoring when such agents are prescribed; this recommendation is based on qualitative synthesis of observational and case data. [Evidence level: Low; GRADE: Very Low certainty]
5. Consideration of AID systems in highly selected patients with type 1 diabetes or recalcitrant hypoglycaemia unawareness may be explored under specialist diabetes and nephrology co-management, recognising the very limited current evidence base (two studies, N = 89) and the need for dedicated dialysis-specific RCTs. [Evidence level: Very Low; GRADE: Very Low certainty — not currently recommended for standard clinical practice]
6. Supplementation of HbA1c monitoring with glycated albumin and CGM-derived time-in-range

metrics, acknowledging the well-documented limitations of HbA1c in ESRD while recognising its continued role in clinical practice alongside superior alternatives. [Evidence level: Moderate; GRADE: Moderate certainty]

5. CONCLUSIONS

Recurrent hypoglycaemia in dialysis patients is a prevalent, clinically severe, and often asymptomatic complication with significant implications for cardiovascular morbidity and mortality. This systematic review and meta-analysis demonstrate that therapeutic interventions across four categories—dialysate glucose optimisation, CGM-guided insulin adjustment, structured insulin dose reduction, and emerging AID systems—are each individually associated with significant reductions in hypoglycaemic risk. An exploratory cross-intervention pooled effect estimate (0.48, 95% CI 0.39–0.59; composite of OR and RR values across subgroups) indicates an approximate 52% relative reduction in hypoglycaemic risk, though this figure should not be interpreted as a single combined multimodal treatment effect given the biological and mechanistic heterogeneity of the pooled interventions.

The use of glucose-containing dialysate substantially mitigates a mechanistically unnecessary and preventable cause of haemodialysis-induced hypoglycaemia and warrants strong consideration as part of standard care for diabetic haemodialysis patients, pending broader guideline adoption. CGM represents the most sensitive monitoring tool available for this population and enables proactive rather than reactive hypoglycaemia management. Insulin dose optimisation with a target TDD below 0.23 units/kg/day provides a practical glycaemic strategy, while drug-induced hypoglycaemia from quinolones and other high-risk agents demands systematic prescriber vigilance.

The evidence base for AID systems in dialysis patients—though promising—remains in its infancy and requires validation through dedicated, adequately powered RCTs that explicitly enroll dialysis patients and incorporate dialysis-session-aware algorithmic adaptations. The consistent finding across this review that dialysis patients experience high rates of asymptomatic hypoglycaemia invisible to conventional monitoring reinforces the urgent need for CGM integration into routine dialysis care pathways.

Future research should prioritise: prospective RCTs of CGM-guided care in dialysis populations; randomised evaluation of glucose-containing dialysate in peritoneal dialysis; development and validation of AID algorithms incorporating dialysis session parameters; and long-term outcome studies correlating hypoglycaemia reduction with cardiovascular and mortality endpoints. A collaborative, multidisciplinary approach—integrating nephrology, diabetology, pharmacy, and clinical technology expertise—is essential to address this complex and high-priority unmet clinical need.

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Conclusions

Available evidence supports a multimodal, individualised approach to preventing recurrent hypoglycaemia in dialysis patients. This review highlights effective strategies and identifies critical gaps, emphasizing the need for dialysis-specific randomized controlled trials.

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