International Journal of Advances in Medicine
Kakkadapram HV et al. Int J Adv Med. 2025 Jul;12(4):434-446
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20251943

Systematic Review

Long-term cardiovascular outcomes in peritoneal dialysis patients: a systematic review

Hajira V. Kakkadapram[1]*, M. S. Huq[2]

[1]Department of Critical Care, Iqraa International Hospital and Research Center, Calicut, Kerala, India
[2]Department Public Health, Deep Institute of Community Ophthalmology, Rangpur, Bangladesh

Received: 02 May 2025
Accepted: 06 June 2025

***Correspondence:**
Dr. Hajira V. Kakkadapram,
E-mail: haju.207@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Peritoneal dialysis (PD) is a therapy for end-stage kidney disease that is increasingly used worldwide, especially in developing countries. Despite its benefits, PD patients remain at high risk for cardiovascular disease and related mortality due to unique metabolic and inflammatory risk factors. We performed a systematic review of cohort studies reporting cardiovascular and all-cause events in adult patients receiving PD. A search of PubMed, Embase, Scopus, and the Cochrane Library identified 716 studies; following after the screening and full-text review, 20 studies (7 prospective, 13 retrospective) were included. Data were extracted on study and participant characteristics, PD modality, outcomes, risk factors and methodological quality. All-cause mortality varied between 19.4 and 42.4% with CVD-related mortality representing 40–55% of all deaths. Vascular calcification (HR 8.01 for AAC >39%), hypoalbuminemia (HR 2.84), hypomagnesemia (HR 1.58), and inflammation (elevated neutrophil-to-lymphocyte ratio, HR 2.60; platelet-to-albumin ratio, HR 1.50) were significant modifiable risk factors for events. The incidence of peritonitis predicted cardiovascular death in a dose-response relationship. Cardiopathy (LV diastolic dysfunction, HR 2.25) and metabolism (remission of low triiodothyronine, HR 0.86 for each 10 ng/dl) were also independent predictors. Confounding and missing data yielded risk of bias as moderate or serious in most studies. Long-term cardiovascular outcomes in PD patients are driven by a complex interplay of vascular, inflammatory, metabolic, and cardiac factors. Addressing these modifiable risks should be prioritized in clinical management and research to improve survival in this high-risk population.

Keywords: Peritoneal dialysis, Cardiovascular mortality, Long-term outcomes, Risk factors

INTRODUCTION

A peritoneal dialysis (PD) is a renal replacement therapy that utilizes the peritoneum as a natural semipermeable membrane to remove waste products, electrolytes, and excess fluids from the body through the infusion of dialysis fluid into the peritoneal cavity.[1] This approach is particularly beneficial for patients with end-stage kidney disease (ESKD), offering the convenience of home-based treatment and improved quality of life compared to conventional hemodialysis.[2–5] Globally, PD accounts for 11% of dialysis cases and 9% of kidney replacement treatment procedures.[6] The global distribution of dialysis patients using peritoneal dialysis systems stands markedly different across developed and developing regions as developing nations maintain higher numbers.[7]

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among dialysis patients, contributing to approximately 45% of deaths in this population.[8] People receiving dialysis treatment experience cardiovascular death risks which exceed those of the general population by 20 times.[9,10] Patients undergoing PD treatment experience specific

cardiovascular risk elements connected to volume load from renal function loss and elevated glucose concentrations that rule PD solution chemistry as well as glucose degradation products that produce advanced glycation end-products.[11,12] Inflammation together with atherosclerosis and these additional factors substantially increase the risk of cardiovascular mortality. Medical experts have identified heart failure and sudden cardiac death as significant causes of cardiovascular death in dialysis patients beyond traditional atherosclerotic disease processes.[13]

Although PD represents a significant clinical issue for healthcare providers, we still need to understand specific long-term heart problems that occur in PD patients. Studies comparing cardiovascular risks between PD and additional dialysis treatments produce different results. some research suggests that home hemodialysis confers better protection against stroke and heart attacks and mortality from all causes than PD, while other reviews highlight PD as a cost-effective alternative for ESKD patients with cardiovascular risks.[14,15] Presence of peritonitis infection, a common PD-related complication that typically intensifies cardiovascular risks in this treatment.[16] The basis of conducting this systematic review includes several key factors such as the global increase of PD treatment alongside its escalating prevalence rates in developing nations, along with varying research methods and outcome reports, and the necessity for consolidated insights to enable better clinical choices.[17,18]

This review aims to comprehensively evaluate long-term cardiovascular outcomes in PD patients, identify modality-specific cardiovascular risk factors, compare outcomes between PD and other dialysis modalities where data permits, and assess the quality of existing evidence to inform future research directions. By addressing these objectives, this review seeks to provide an evidence-based foundation for optimizing cardiovascular care in the growing population of PD patients worldwide.

METHODS

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review aimed to synthesize the available evidence on long-term cardiovascular outcomes in patients undergoing peritoneal dialysis (PD), identify PD-specific cardiovascular risk factors, and compare outcomes between PD and other dialysis modalities.

Search strategy

A comprehensive search of electronic databases was performed to identify relevant studies. Databases included PubMed, Embase, Scopus and Cochrane Library. The search covered all publications from January 2015 to December 2024. The search strategy utilized a combination of Medical Subject Headings (MeSH) terms and keywords related to peritoneal dialysis (e.g., "peritoneal dialysis," "PD," "continuous ambulatory peritoneal dialysis") and cardiovascular outcomes (e.g., "cardiovascular mortality," "myocardial infarction," "stroke," "heart failure"). Boolean operators ("AND," "OR") were applied to refine the search results.

In addition to database searches, manual screening of reference lists from relevant systematic reviews and primary studies was conducted to identify additional eligible articles. Grey literature sources, including conference proceedings and reports, were also reviewed to capture unpublished data that met inclusion criteria.

Study selection

Studies were selected based on predefined inclusion and exclusion criteria. The inclusion criteria were as follows.

Population

Adults aged 18 years or older undergoing peritoneal dialysis for end-stage kidney disease (ESKD).

Outcomes

Studies reporting long-term cardiovascular outcomes, including cardiovascular mortality, myocardial infarction, stroke, or hospitalization due to heart failure.

Study design

Randomized controlled trials (RCTs), cohort studies, case-control studies, and cross-sectional studies with a minimum follow-up duration of five years.

Exclusion criteria included non-English language studies, pediatric populations, animal studies, case series or reports with fewer than 50 participants, and studies with insufficient follow-up data.

Two independent reviewers screened titles and abstracts for relevance using Rayyan software for systematic reviews. Full-text articles were assessed for eligibility based on inclusion criteria. Disagreements between reviewers were resolved through discussion or consultation with a third reviewer.

Data extraction

Data extraction was performed using a standardized template designed to capture key study characteristics. Extracted information included.

Study characteristics

Author(s), year of publication, study design, sample size, geographic location.

Patient demographics

Age, sex, dialysis vintage, comorbidities.

Cardiovascular outcomes

Definitions of outcomes (e.g., myocardial infarction), event rates, hazard ratios (HRs), and confidence intervals (CIs).

Peritoneal dialysis-specific risk factors

Residual renal function status, glucose exposure from PD solutions, presence of peritonitis infection.

Data extraction was conducted independently by two reviewers to ensure accuracy and completeness. Any discrepancies in extracted data were resolved through consensus.

Quality assessment

The methodological quality of all included studies was evaluated using the ROBINS-I (risk of bias in non-randomized studies - of interventions) tool, which is specifically designed for cohort and other non-randomized studies.[20] This tool assesses risk of bias across seven domains: confounding, selection of participants, classification of interventions (exposures), deviations from intended interventions, missing data, measurement of outcomes, and selection of the reported result. For each study, two reviewers independently assessed risk of bias, with disagreements resolved by consensus. Studies were rated as having low, moderate, or serious risk of bias in each domain and overall.

Data synthesis

A narrative synthesis was conducted for all included studies to summarize findings related to long-term cardiovascular outcomes in PD patients. Subgroup analyses explored potential sources of heterogeneity based on geographic region (developed vs developing countries), PD modality (continuous ambulatory vs automated PD), and residual renal function status.

RESULTS

A comprehensive literature search was performed across four major electronic databases: PubMed/MEDLINE, Embase, Scopus and Cochrane Library, resulting in the identification of 716 studies.

After removing duplicates, 658 studies remained for title and abstract screening. Following a detailed full-text review, 20 studies were included in the final synthesis. Of these, 8 were prospective cohorts and 12 were retrospective cohorts, with no randomized controlled trials identified. The PRISMA flow diagram (Figure 1) illustrates the study selection process. Geographically, Chinese (n=10) and Taiwanese (n=6) cohorts dominated (16/20 studies), alongside studies from Türkiye (n=1), Portugal (n=1), Brazil (n=1), and South Korea (n=1). Sample sizes varied widely, ranging from 123 to 5,707 participants, with follow-up durations spanning 2.5 to 14 years. Adult populations were exclusively studied, with mean ages between 42.3 and 59.4 years. Comorbidities were prevalent, particularly diabetes (19–56%) and hypertension (53–94%), while cardiovascular disease (CVD) affected 7–44% of participants. Table 1 presents the study and participant characteristics of the included studies.

Intervention and comparator characteristics

Continuous Ambulatory Peritoneal Dialysis (CAPD) was the predominant modality, utilized in 12 studies, while two studies included Automated Peritoneal Dialysis (APD).[25,34] Dialysis prescription details, such as Kt/V targets or exchange frequency, were inconsistently reported, with only eight studies providing granular data. For instance, Tsai et al documented a weekly Kt/V of 1.9±0.3, while Hsieh et al reported a total weekly Kt/V of 2.08±0.56.[24,36]

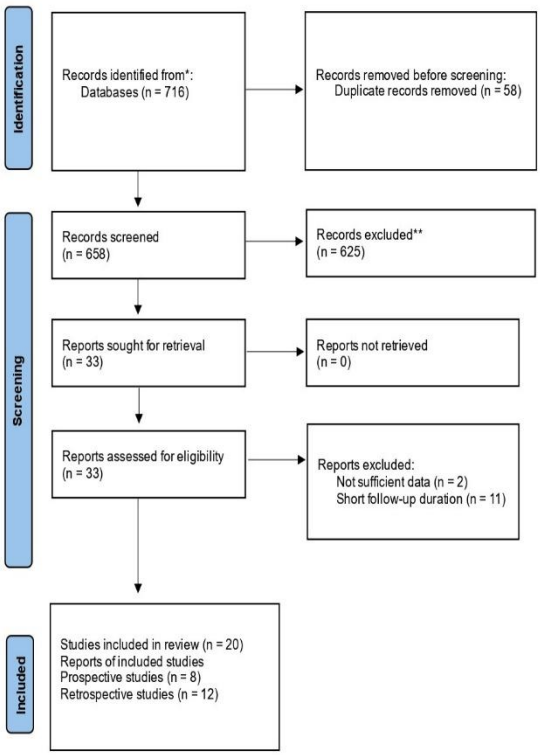

Figure 1: PRISMA-2020 flow for systematic review.

Peritonitis episodes, a critical complication in PD, were quantified in five studies, with rates ranging from 0.2 to 0.35 episodes per patient-year.[34,36] Comparators were heterogeneous: two studies contrasted PD with hemodialysis (HD) while others stratified outcomes by

biomarkers or clinical parameters such as hypervolemia or residual renal function. Table 2 presents intervention/exposure and comparator details of the included studies.[27,28]

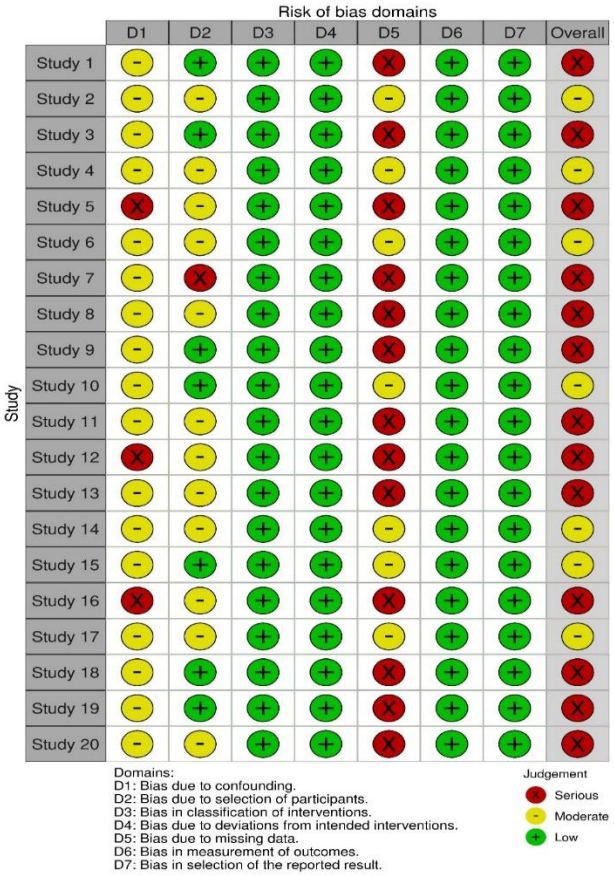

Figure 2: Risk of bias assessment using ROBINS-I tool for non-randomized studies.

Cardiovascular outcomes and risk factors

All-cause mortality, universally reported, ranged from 19.4% to 42.4%, with higher rates observed in studies with prolonged follow-up.[22,38] Cardiovascular mortality, analyzed in 15 studies, was significantly associated with modifiable risk factors bdominal aorta calcification (AAC) ratio was an independent predictor of cardiovascular mortality (HR 8.01 for AAC >39%).[21] Hypervolemia, indicated by icodextrin use, increased cardiovascular event risk (HR 3.8), and hypoalbuminemia (<3.35 g/dl) was an independent predictor of cardiovascular events (HR 2.84).[25]

Hypomagnesemia (<0.7 mmol/l) was a potent predictor of all-cause mortality (HR 1.58).[26] Elevated lipid ratios, such as TG/HDL-C ≥1.94 (HR 3.57) and non-HDL-C/HDL-C≥2.86 (HR 2.58), also independently predicted mortality.[23] Left ventricular diastolic dysfunction (LVDD) doubled the risk of mortality (HR 2.25), and low triiodothyronine (T3) levels were associated with a 14% reduction in mortality risk per 10 ng/dl increase (HR 0.86).[29,30]

Secondary cardiovascular outcomes

Secondary outcomes highlighted the importance of cardiac pathology progression. Incidence of atrial fibrillation was 1.32-fold higher in PD patients compared to non-ESRD controls.[28] Cardiorenal syndrome (CRS), especially type IV, was independently associated with higher cardiovascular mortality (HR 2.10).22 Left ventricular fraction shortening (LVFS) was a robust predictor of cardiovascular events, with lower values linked to higher risk (HR 0.51 for highest vs. lowest tertile).[39]

Inflammatory and hematologic biomarkers

Inflammatory markers were consistently associated with adverse outcomes. Neutrophil-to-lymphocyte ratio (NLR ≥2.87) predicted all-cause (HR 2.60) and cardiovascular mortality (HR 2.89).[33] Platelet-to-albumin ratio (PAR >7.27) was also a strong predictor of all-cause (HR 1.50) and cardiovascular mortality (HR 1.79).[35] Mean platelet volume (MPV ≥10.2 fl) was independently associated with mortality (HR 0.73 for all-cause; HR 0.69 for CV mortality).[31]

Technique failure and peritonitis

Technique failure, including transfer to HD or transplantation, was reported in 16–24% of patients. Peritonitis frequency was a strong, dose-dependent predictor of cardiovascular mortality (HR 1.22 for one episode, up to HR 3.84 for four or more episodes).[34] Hyperuricemia and higher peritonitis rates independently predicted technique failure.[36]

Methodological quality and risk of bias

Figure 2 presents the risk of bias evaluation for the 20 included non-randomized studies using the ROBINS-I tool. Across domains, the majority of studies (13/20, 65%) exhibited a serious overall risk of bias, primarily due to unresolved confounding (e.g., unadjusted comorbidities), substantial missing data (15/20 studies lacked handling methods), or selection bias by Hu et al with registry-based sampling.

The remaining seven studies (35%) were rated moderate risk, reflecting partial confounding control but robust adjustment for key variables.[22,25,27,35] Notably, intervention classification, deviations from intended interventions, and outcome measurement were consistently rated low risk across all studies, suggesting reliable exposure and outcome definitions. However, the frequent serious ratings for confounding, missing data, and selection bias underscore inherent limitations in observational PD research, particularly from retrospective designs and registry data.

Table 1: Study and participant characteristics of included studies.

Author and year	Study design	Country	Sample size	Follow-up (type)	Age (Mean±SD or range)	Sex (% male)	Primary renal disease	Key comorbidities (DM/HTN/CVD)	PD vintage	Residual renal function
Tsai et al[21], 2020	Prospective cohort	Taiwan	123	Median 6.8 yrs (IQR 3.6–9.2)	53–59 yrs (mean)	33–48%	Not reported	HTN 88–94%, DM 19–28%	Mean 42–43 mo	Not reported
Xue et al[19], 2022	Retrospective cohort	China	748	Median 6.23 yrs (IQR 2.42-11.65)	57–68 yrs (mean)	37–51%	DN 27–52%, HTN 12–25%	DM 31–56%, CHD 13–44%	Incident	Not reported
Xia et al[23], 2022	Retrospective cohort	China	214	Median 59 mo (Range 3–60)	50±14 yrs	59%	Not specified	DM 19–23%, HTN 53–61%, CVD 7–10%	Incident (1–3 mo)	Not reported
Tsai et al[24], 2020	Prospective cohort	Taiwan	133	Median 6.37 yrs (IQR 2.96–9.17)	59±8.3 yrs (CV mortality group)	38–48%	Not specified	HTN 86%, DM 29%, CVD 21%	>3 months	Not reported
Querido et al[25], 2017	Retrospective cohort	Portugal	112	Mean 29.3 mo (Range up to 5 yrs)	53.7±16.1 yrs	65.2%	DN 31%, CGN 15%	DM 37.8%, IHD 17.9%	Incident	Baseline eGFR 6.76 ml/min/1.73 m²
Zhang et al (2021)[26]	Retrospective cohort	China	1,004	Median 39.4 mo (Range 3–117, Max ~9.8 yrs)	52.3±14.4 yrs	56.6%	GN 33.3%, DN 22.9%	DM 23.5%, CVD 20.5%	≥3 mo	Median 3.30 ml/min/1.73 m²
Hu et al[27], 2020	Retrospective cohort	Taiwan	109,256	Maximum 10 yrs (2006–2015); Median NR	54.5–57.3 yrs (mean)	52%	Not specified	DM 35.7–46%, HTN 83–88%	Not reported	Not reported
Shen et al[28], 2016	Retrospective cohort	Taiwan	15,947	Mean 8–10 yrs	53.7–61.3 yrs (mean)	39.2–48.5%	Not specified	DM 32.9–50.4%, HTN 66.1–73.7%, CAD 19.9–26.4%	Incident ESRD	Not reported
Wu et al[29], 2015	Prospective cohort	Taiwan	149	Mean 3.8 yrs (Range up to ~5.5)	55.2±12.7 yrs	44%	Not specified	HTN, DM, prior MI	>6 mo	24h urine output included
Chang et al[30], 2015	Prospective cohort	South Korea	447	Median 46 mo (Range 7–142, Max ~12 yrs)	58 yrs (mean)	54.3%	Not specified	DM 50.2%, prior CV events 30.7%	>6 mo	Mean RRF higher in high T3
Wang et al[31], 2023	Retrospective cohort	China	1,322	Median 50 mo (IQR 30–80, Max 15 yrs)	49.3±14.5 yrs	57.6%	Not specified	DM 18.8%, HTN 75.3%	≥3 mo	Not reported
Demir et al (2024)[32]	Retrospective cohort	Türkiye	250	Median 39.5 mo (IQR 17–71), 5/10/15-yr survival reported	Not specified	Not specified	CGN most common	DM 13.6%, CVD 16%	>3 mo	Not reported

Continued.

Author and year	Study design	Country	Sample size	Follow-up (type)	Age (Mean±SD or range)	Sex (% male)	Primary renal disease	Key comorbidities (DM/HTN/CVD)	PD vintage	Residual renal function
Zhang et al (2021)[33]	Prospective cohort	China	140	Median 66 mo (Max 5.5 yrs)	54.7±15.7 yrs	57.1%	GN 44.3%, DN 13.6%	DM 22%, CVD 36.4%	Incident	Mean rGFR 5.61±1.94 ml/min/1.73 m²
Pecoits-Filho et al[34], (2018)[34]	Prospective cohort	Brazil	5,707	Maximum 63 mo (Mean 18±13 mo); Median NR	59.4±16.0 yrs	47.5%	DM 37.2%, HTN 16.8%	DM 43.5%, HTN 72.7%, CAD 21.3%	Incident (≥90 days)	Not reported
Ma et al[35], 2024	Retrospective cohort	China	2,825	Mean 47.5±28.3 mo (Range up to ~10 yrs)	52.6±14.7 yrs	44.7%	Not specified	DM 34.2%, CVD 27.8%	≥3 mo	Median eGFR 6.1 (4.5–8.1) ml/min/1.73 m²
Hsieh et al[3], 2017	Retrospective cohort	Taiwan	371	Mean 36.7±27.5 mo (Median 30, Range up to 9 yrs)	55.7±16.0 yrs	43.9%	CGN 30.7%, DM 27.5%	DM 31.5%, HTN 82.2%, CAD 15.6%	Incident	Mean eGFR 2.53±2.68 ml/min/1.73 m²
Li et al[37] 2021	Retrospective cohort	China	1,589	Median 47.2 mo (IQR 21.2–82.2, Max 13 yrs)	46.9±15.3 yrs	60%	GN 60.5%, DN 22.7%	DM 25.7%, CVD 21.2%	≥3 mo	Median 3.2 (1.9–5.0) ml/min/1.73 m²
Xia et al[38] 2019	Prospective cohort	China	533	>10 yrs (All patients)	48±16 yrs	57%	GN 61%, DN 24%	DM 24%, HTN NR	Incident	Median eGFR 4.9 (3.8–6.3) ml/min/1.73 m²
Dai et al[39] 2023	Observational cohort	China	784	Median 42.3 mo (IQR 24–79, Max ~10 yrs)	42.3±14.5 yrs	56.6%	GN 65.8%, DN 12.2%	HTN 54.2%, DM 20%	Incident	Not reported
Xu et al[40], 2021	Retrospective cohort	China	376	Up to 14 yrs (1-, 3-, 5-yr survival reported)	57.8±15.1 yrs	57.7%	Not specified	DM 30.9%, HTN 88.3%	≥3 mo	Not reported

Table 2: Intervention/Exposure and comparator details of included studies.

Study	PD modality	Dialysis prescription	Membrane transport status	Peritonitis episodes	Comparator	Comparator characteristics
Tsai et al[21]	Not specified	PD Kt/V: 1.9±0.3–0.4	Not reported	Sepsis (primarily peritonitis) reported	None (single group)	N/A
Xue et al[22]	CAPD or DAPD (majority)	Not specified	Not reported	12% of deaths in non-CRS group	Non-CRS, AHF, type II/IV CRS	Group comparisons by CRS type
Xia et al[23]	CAPD (all patients)	Total Kt/V: median ~2.1	Not reported	Not reported	None	Stratified by TG/HDL-C and non-HDL-C/HDL-C ratios

Continued.

Study	PD modality	Dialysis prescription	Membrane transport status	Peritonitis episodes	Comparator	Comparator characteristics
Tsai et al[24]	Not specified	PD Kt/V: 1.9 ± 0.3	Not reported	Not reported	None	Stratified by heart rhythm complexity (DFAα1 ≤0.98 vs. >0.98)
Querido et al[25]	CAPD (67.9%), APD (32.1%)	Bicarbonate-buffered; 42.9% used icodextrin	PET performed (details NR)	Not reported	None	Stratified by mitral calcification and albumin
Zhang et al[26]	Not specified	Kt/V median ~2.01	D/P creatinine ratio reported	Not reported	None	Stratified by serum magnesium group (<0.7, 0.7–1.2, >1.2 mmol/l)
Hu et al[27]	PD vs. HD	Not reported	Not reported	Not reported	HD	Large national HD cohort, PS-matched and time-varying analyses
Shen et al[28]	PD vs. HD	Not reported	Not reported	Not reported	HD and non-ESRD controls	14,854 HD, 1,093 PD, 47,841 controls; matched by age, sex, index year
Wu et al[29]	Conventional PD (likely CAPD)	Peritoneal Kt/V, residual Kt/V measured	Not reported	Not reported	None	Stratified by LVDD status
Chang et al[30]	CAPD	Target Kt/V ≥1.7/week	Not reported	Not reported	None	Stratified by T3 tertiles
Wang et al[31]	Not specified	Not reported	Not reported	Not reported	None	Stratified by MPV (<10.2 fl vs. ≥10.2 fl)
Demir et al[32]	Not specified	Weekly total Kt/V urea reported	High permeability	Peritonitis rate: 0.2/pt-year	None	Stratified by survival status, comorbidities
Zhang et al[33]	CAPD	3–5 daily exchanges, 1.5–4.25% dextrose	Not reported	42 episodes	None	Stratified by NLR (median cutoff 2.87)
Pecoits-Filho et al[34]	CAPD/APD	Not specified	Not reported	2,405 episodes (time-dependent)	None	Stratified by peritonitis frequency (0, 1, 2, 3, ≥4)
Ma et al[35]	CAPD	Kt/V median 2.0 (1.8–2.4)	Not reported	Not reported	None	Stratified by PAR quartiles (Q1–Q4)
Hsieh et al[36]	CAPD	Weekly total Kt/V: 2.08±0.56	D/P creatinine at 4h: 0.67	Peritonitis rate: 0.18 ± 0.35/yr	None	Stratified by uric acid (>8 mg/dL vs. ≤8 mg/dl)
Li et al[37]	CAPD	Weekly total Kt/V: 2.45±0.73	Not reported	Not reported	None	Stratified by CVD readmission status (within first year)
Xia et al[38]	CAPD	3–5 daily exchanges	Not reported	Peritonitis rate: 0.19/pt-year	None	Stratified by age, Charlson index, education, etc.
Dai et al[39]	CAPD	Not reported	Not reported	Not reported	None	Stratified by LVFS tertiles (≤31%, 31–35%, >35%)
Xu et al[40]	Not specified	Not reported	Not reported	Not reported	None	Stratified by T3 status (low vs. normal)

Continued.

Table 3: Outcomes, risk factors, and statistical methods in included studies.

Study	Outcome measures and effect sizes	Key risk factors analyzed	Adjustments for confounders	Handling of missing data	Subgroup analyses/stratification
Tsai et al[21]	CV mortality: HR 1.057 (95% CI 1.030–1.085); All-cause: HR 1.035 (1.018–1.052); AAC cutoff >39%: HR 8.01 (3.14–20.44)	AAC ratio, age	Age, sex, DM, HTN, LVEF	Not reported	None
Xue et al[22]	All-cause mortality: 42.4%; CV mortality: HR 2.10 (1.03–4.28) for type IV CRS	CRS type, age, DM, albumin	Age, DM, albumin, calcium, eGFR	Multiple imputation	By CRS type
Xia et al[23]	All-cause mortality: 25.2%; TG/HDL-C ≥1.94: HR 3.57 (1.99–6.39); Non-HDL-C/HDL-C ≥2.86: HR 2.58 (1.39–4.81)	TG/HDL-C, non-HDL-C/HDL-C, age, iPTH	Age, DM, CVD, HTN, iPTH, lipids	Not reported	By lipid ratio cutoffs
Tsai et al[24]	CV mortality: 22% (21/133); DFAα1 HR 0.076 (95% CI 0.016–0.366, p=0.001); MSE area 1–5 HR 0.645 (0.447–0.930, p=0.019); MACE: 26%	DFAα1, MSE area 1–5, age, HRV	Multivariate Cox (age, sex, DM, CVD, HRV vars)	Not reported	By DFAα1 cutoff (≤0.98 vs. >0.98)
Querido et al[25]	CV mortality: 9.8%; Major CV events: HR 3.25 (1.3–8.3) for mitral calcification; Hypervolemia: HR 3.8 (1.13–12.8); Hypoalbuminemia: HR 2.84 (0.15–7.0)	Mitral calcification, hypervolemia (icodextrin), albumin	Age, DM	Not reported	By mitral calcification, icodextrin use, albumin
Zhang et al[26]	All-cause mortality: HR 1.58 (1.20–2.08) for hypomagnesemia; CVD mortality: HR 1.63 (1.11–2.38); Infection-related: HR 1.92 (1.13–3.26)	Serum magnesium, age, albumin, CRP, DM, CVD	Age, albumin, CRP, DM, CVD	Multiple imputation	By magnesium group
Hu et al[27]	All-cause mortality: HR 1.13 (1.09–1.17) PD vs. HD; MACE: HR 1.06 (1.01–1.12) PD vs. HD	Modality, age, sex, DM, comorbidities	Age, sex, DM, comorbidities, dialysis vintage	Not reported	By modality, age, sex, DM
Shen et al[28]	Atrial fibrillation: HR 1.32 (1.00–1.83) PD vs. controls	Age, HTN, DM, heart failure, valvular disease, COPD	Age, HTN, DM, heart failure, valvular disease	Not reported	By age, sex, comorbidities
Wu et al[29]	All-cause mortality: HR 2.25 (1.45–2.91) for LVDD; MACE: HR 1.71 (1.43–3.51) for LVDD	LVDD, hsCRP, age, DM, HTN, prior MI	Age, DM, HTN, RRF, haemoglobin, prior MI, LDL, hsCRP, LVDD	Not reported	By LVDD status
Chang et al[30]	All-cause mortality: HR 0.86 (0.78–0.94) per 10 ng/dL T3; Sudden death: HR 0.69 (0.56–0.86)	Low T3, age, DM, albumin, RRF	Age, s Continued. prior C v events, albumin, nPCR, LBM, CRP, RRF	Exclusion of missing data	By T3 tertile
Wang et al[31]	All-cause mortality: HR 0.73 (0.59–0.91) for MPV ≥10.2 fL; CV mortality: HR 0.69 (0.50–0.95)	MPV, age, sex, DM, HTN, CVD, BMI, CCI, albumin, lipids	Age, sex, CCI, DM, HTN, CVD, BMI, antiplatelet meds, haemoglobin, albumin, TG, HDL, LDL	Not reported	By MPV group, age, sex, DM, HTN, CVD

Continued.

Study	Outcome measures and effect sizes	Key risk factors analyzed	Adjustments for confounders	Handling of missing data	Subgroup analyses/stratification
Demir et al[32]	5-yr survival: 86.8%; CV mortality: 54.2% of deaths; Age: HR 1.06 (1.04–1.09); Male: HR 2.07 (1.10–3.90)	Age, male sex, DM, transfer for vascular access, peritoneal permeability	Age, sex, transfer reason, DM, CVD, permeability	Not reported	By survival status, comorbidities
Zhang et al[33]	All-cause mortality: HR 2.60 (1.04–6.54) for NLR ≥2.87; CV mortality: HR 2.89 (1.01–8.28)	NLR, age, Ca x P, CVD history	Age, CVD, DM, Ca x P, albumin, hsCRP, NLR	Not reported	By NLR group
Pecoits-Filho et al[34]	CV mortality: HR 1.22–3.84 per peritonitis episode	Peritonitis frequency, age, sex, DM, CAD, LVH, pre-dialysis care, haemoglobin	Age, sex, DM, CAD, LVH, pre-dialysis care, haemoglobin	Not described	By peritonitis frequency
Ma et al[35]	All-cause mortality: HR 1.50 (1.21–1.86) for PAR Q4 vs. Q1; CV mortality: HR 1.79 (1.32–2.43)	PAR, age, sex, DM, CVD, BMI, labs	Age, sex, BMI, BP, DM, CVD, labs, eGFR, Kt/V	Exclusion of missing data	By PAR quartile, age, sex, BMI, DM, CVD, HTN
Hsieh et al[36]	Technique failure: HR 1.24 (1.09–1.42) for hyperuricemia; Peritonitis-related: HR 1.29 (1.07–1.57)	Hyperuricemia, DM, peritonitis rate, RRF, icodextrin use	Age, sex, BMI, DM, peritonitis rate, RRF, icodextrin use	Not reported	By UA level, sensitivity analyses
Li et al[37]	All-cause mortality: HR 2.66 (1.91–3.70) for CVD readmission; CV mortality: HR 3.42 (2.20–5.31)	CVD readmission, age, albumin, DM, CVD history	Age, sex, DM, CVD, HTN, BMI, haemoglobin, albumin, labs, RRF	Pairwise deletion	By CVD readmission status
Xia et al[38]	10-yr survival: 36%; CV death: 47% of deaths; Technique failure: 16%	Age, Charlson index, education, RRF, diabetic nephropathy	Age, sex, BMI, cause of ESRD, CCI, urine output, eGFR, education, income	Not reported	By age, CCI, education
Dai et al[39]	CV events: HR 0.51 (0.37–0.71) for highest LVFS tertile	LVFS, age, sex, BMI, HTN, dyslipidemia, albumin, diabetes	Age, sex, BMI, HTN, DM, cholesterol, LDL, albumin, CRP, Ca, Phos, meds	Not reported	By LVFS tertile, age, sex, HTN, BMI, albumin, DM, dyslipidemia
Xu et al[40]	All-cause mortality: HR 0.633 (0.431–0.930) for low FT3; CVD mortality: NS	Low FT3, heart disease, TT4, albumin, haemoglobin, age, DM, eGFR, CRP	Age, DM, CVD, albumin, haemoglobin, eGFR, CRP	Not reported	By T3 group

DISCUSSION

This systematic review combined evidence across 20 cohorts totaling more than 140,000 adults for PD and provides a complete synthesis of long-term outcomes and cardiovascular risk in this emergent population of patients. While the global uptake of PD has been increasing, especially in less developed regions, our findings reaffirm that cardiovascular disease (CVD) continues to remain the primary cause of death in PD patients, contributing to 40–55% of all-cause deaths across varied populations. This is consistent with previous large-scald meta-analyses and registry studies, where CVD was consistently presented as the leading COD in ESKD patients, with several fold higher of the risk compared to the general population.[33,41]

Our analysis demonstrates that CV risk in PD patients is determined by a complex interplay of traditional and non-traditional risk factors. Age, diabetes, and preexisting cardiovascular disease are consistently identified as the main determinants of all-cause as well as cardiovascular death.[26] But, PD-specific factors, including chronic inflammation, metabolic derangements, and exposure to glucose-containing dialysate increase the risk in PD population. Vascular calcification, in particular abdominal aorta calcification (AAC) was also found being most powerful independent predictors of cardiovascular mortality in PD an AAC-ratio > 39% being associated with 8.2-fold relative risk.[21] This goes along with previous work in dialysis (and non-dialysis) CKD patients that have shown that the presence of vascular calcification in a marker of advanced atherosclerosis and a harbinger of bad outcomes.[42]

Malnutrition and inflammation, with hypoalbuminemia and increased inflammatory markers, also constituted important predictors of adverse results. Hypoalbuminemia (<3.35 g/dl) independently predicted cardiovascular events and all-cause mortality consistently in our study, which was in accordance with the previously documented malnutrition-inflammation complex syndrome among patients on dialysis.[25,30,43] Inflammatory biomarkers, including the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-albumin ratio (PAR), were robustly associated with mortality risk.[33,35] These findings are supported by broader evidence implicating chronic inflammation as a driver of atherosclerosis and cardiac dysfunction in CKD.[44]

Electrolytes, especially hypomagnesemia, were independently associated with the all-cause and cardiovascular mortalities.[26] This would be biologically plausible as magnesium participates in the regulation of vascular tone, prevention of arrhythmias and endothelial function.[45] Metabolic risk factors, such as higher TG/HDL cholesterol ratios and higher non-HDL/HDL ratios, were equally closely associated with heightened mortality and reflected the atherogenic lipid profiles typically seen in PD patients.[31,41]

Apart from these biochemical and clinical markers the statement points new prognostic markers e.g., heart rhythm complexity expressed by nonlinear HRV parameters such as DFA α1 and multiscale entropy (MSE). Tsai et al found that decreased heart rhythm complexity was independently associated with cardiovascular death and major adverse cardiovascular events and was superior to conventional linear HRV parameters.[24] This discovery implies that autonomic disturbances and impaired cardiac adaptability are neglected cardiac risk factors in PD and provides novel opportunities for risk stratification and intervention.[46]

Our synthesis also highlights the importance of PD-related complications, particularly peritonitis, in determining both technique failure and CV outcomes. Peritonitis rate had a clear dose–response trend for cardiovascular mortality, with the risk increasing for each extra episode.[34] The systematic review by Sahlawi et al draws attention to the substantial heterogeneity of peritonitis definitions and presentation in different studies that precludes direct comparisons but emphasizes the importance of common outcome measures. Peritonitis is still in fact, a leading cause of technique failure and switch to hemodialysis, especially in the elderly/fail patients which are also exposed to a greater risk of both death and infection.[47,48] Review showed the comparison between PD and hemodialysis (HD), and meta-analyses by Loziers et al report that all-cause and cardiovascular mortality are generally comparable among modalities, with pooled risk ratios near unity and no significant differences in adverse cardiovascular events when comparing across modalities.[41] However, a few subgroups, like the elderly and diabetes patients have marginally elevated risk on PD, which would represent greater comorbidity burden and more rapid loss of residual renal function.[27,48] Conversely, atrial fibrillation was less common among PD patients than among HD patients, probably because of more stability in hemodynamics and avoidance of rapid fluid shifts.[28,49]

The strengths of this review include its strict inclusion of long-term follow-up studies, comprehensive adjustment for confounders in key analyses, and the inclusion of diverse international cohorts. However, several limitations must be acknowledged. The majority of studies were observational, single-center, or registry-based, which introduces some risk of residual confounding and of selection bias. A lot of studies did not mention their handling of missing data and definition of outcomes-most notably cardiovascular events and peritonitis-differed for which comparability was limited. Most of the included studies were in Asian populations, so this might limit the extrapolation of findings to Western countries with other patients' characteristics and health systems. Besides, few studies provided detailed data on residual renal function or dialysis prescription, both of which are known to influence cardiovascular outcomes in PD.

In clinical practice, these findings underscore the importance of comprehensive risk assessment in PD

patients, including regular evaluation of vascular calcification, nutritional and inflammatory status, and cardiac function. Interventions targeting modifiable risk factors-such as optimizing nutrition, using biocompatible PD solutions, and preventing peritonitis-should be prioritized. The emerging evidence for heart rhythm complexity and inflammatory biomarkers as prognostic tools may refine risk stratification and guide individualized care. Future research should focus on multicentre, prospective studies with standardized outcome definitions, greater representation of non-Asian cohorts, and interventional trials targeting inflammation, vascular calcification, and metabolic disturbances.

CONCLUSION

Despite providing crucial benefits in patient autonomy and hemodynamic stability, peritoneal dialysis is counterbalanced by continuous metabolic and infectious burdens. Several comparative analyses have shown that overall cardiovascular outcomes are comparable for PD compared with haemodialysis, but that specific high-risk subgroups on PD (in particular the elderly and those with diabetes or pre-existing CVD) may need monitoring and individualization of treatment. The review points to large heterogeneity in outcome definitions and reporting, indicating that standardized research methods and multicentre studies are urgently needed. However, addressing these modifiable risks through the implementation of comprehensive cardiovascular risk assessment and management, prevention of infective complications, and targeted interventions will be vital for improving survival and quality of life in patients with PD. Further studies should focus on the need for global representation, harmonized reporting of clinical end points and trials of interventions that will optimize cardiovascular management of this growing population.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Hashimoto K, Kamijo Y. Current progress in peritoneal dialysis: a narrative review of progress in peritoneal dialysis fluid. Life. 2025;15(2):279.
2. Elzorkany K, Alhamad MA, Albaqshi BM. Knowledge about peritoneal dialysis among patients with end-stage kidney disease on hemodialysis: a cross-sectional study. Ann Med. 2024;56(1):2411019.
3. François K, Bargman JM. Evaluating the benefits of home-based peritoneal dialysis. Int J Nephrol Renovasc Dis. 2014;7:447-55.
4. Villa JD, Escobar KB, Escobar GL, Rodriguez CFE. WCN24-1639 automated peritoneal dialysis in patients with low peritoneal transport, experience in a single Mexican center. Kidney Int Reports. 2024;9(4):595.
5. Malik S. Peritoneal Dialysis. In: Malik S, ed. Best of Five MCQs for the European Specialty Examination in Nephrology. 1st ed. Oxford University Press Oxford; 2023:167-80.
6. Bello AK, Okpechi IG, Osman MA. Epidemiology of peritoneal dialysis outcomes. Nat Rev Nephrol. 2022;18(12):779-93.
7. Li PKT, Chow KM, Van De Luijtgaarden MWM. Changes in the worldwide epidemiology of peritoneal dialysis. Nat Rev Nephrol. 2017;13(2):90-103.
8. Herzog CA. Cardiac arrest in dialysis patients: Approaches to alter an abysmal outcome. Kidney Int. 2003;63:197-200.
9. Echefu G, Stowe I, Burka S, Basu-Ray I, Kumbala D. Pathophysiological concepts and screening of cardiovascular disease in dialysis patients. Front Nephrol. 2023;3:1198560.
10. Jegatheesan D, Cho Y, Johnson DW. Clinical Studies of Interventions to Mitigate Cardiovascular Risk in Peritoneal Dialysis Patients. Sem Nephrol. 2018;38(3):277-90.
11. Peng J, Li A, Yin L, Yang Q, Pan J, Yi B. Estimated Glucose Disposal Rate Predicts Renal Progression in Type 2 Diabetes Mellitus: A Retrospective Cohort Study. J Endocrine Soc. 2023;7(7):69.
12. Roumeliotis S, Dounousi E, Salmas M, Eleftheriadis T, Liakopoulos V. Unfavourable effects of peritoneal dialysis solutions on the peritoneal membrane: the role of oxidative stress. Biomolecules. 2020;10(5):768.
13. McIntyre CW. Hemodynamic Effects of Peritoneal Dialysis. Perit Dial Int. 2011;31(2):73-6.
14. Shah S, Weinhandl E, Gupta N, Leonard AC, Christianson AL, Thakar CV. Cardiovascular outcomes in patients on home hemodialysis and peritoneal dialysis. Kidney360. 2024;5(2):205-15.
15. Lozier MR, Sanchez AM, Lee JJ, Tamariz LJ, Valle GA. Comparison of Cardiovascular Outcomes by Dialysis Modality: A Systematic Review and Meta-Analysis. Perit Dial Int. 2019;39(4):306-14.
16. Hepburn KS, Lambert K, Mullan J, McAlister B, Lonergan M, Cheikh Hassan HI. Peritoneal dialysis-related peritonitis as a risk factor for cardiovascular events. Internal Med J. 2021;51(3):404-10.
17. Jain AK, Blake P, Cordy P, Garg AX. Global trends in rates of peritoneal dialysis. J American Soc Nephrol. 2012;23(3):533-44.
18. Yang B, Wang M, Tong X, Ankawi G, Sun L, Yang H. Experimental models in peritoneal dialysis (Review). Exp Ther Med. 2021;21(3);9671.
19. Page MJ, McKenzie JE, Bossuyt PM. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;3:71.
20. Higgins JPT, Morgan RL, Rooney AA. A tool to assess risk of bias in non-randomized follow-up studies of exposure effects (ROBINS-E). Env Int. 2024;186:108602.
21. Tsai CH, Lin LY, Lin YH, Tsai IJ, Huang JW. Abdominal aorta calcification predicts

cardiovascular but not non-cardiovascular outcome in patients receiving peritoneal dialysis: A prospective cohort study. Medicine. 2020;99(37):21730.
22. Xue Y, Xu B, Su C, Han Q, Wang T, Tang W. Cardiorenal syndrome in incident peritoneal dialysis patients: What is its effect on patients' outcomes. PLoS ONE. 2019;14(6):8082.
23. Xia W, Yao X, Chen Y, Lin J, Vielhauer V, Hu H. Elevated TG/HDL-C and non-HDL-C/HDL-C ratios predict mortality in peritoneal dialysis patients. BMC Nephrology. 2020;21(1):93.
24. Tsai C, Huang J, Lin C. Heart rhythm complexity predicts long-term cardiovascular outcomes in peritoneal dialysis patients: a prospective cohort study. JAHA. 2020;9(2):3036.
25. Querido S, Quadros Branco P, Silva Sousa H. Hypervolemia, hypoalbuminemia and mitral calcification as markers of cardiovascular risk in peritoneal dialysis patients. Revista Portuguesa de Cardiologia. 2017;36(9):599-604.
26. Zhang F, Wu X, Wen Y. Hypomagnesemia Is a Risk Factor for Cardiovascular Disease- A nd Noncardiovascular Disease-Related Mortality in Peritoneal Dialysis Patients. Blood Purif. 2022;51(1):23-30.
27. Hu PJ, Chen YW, Chen TT, Sung LC, Wu MY, Wu MS. Impact of dialysis modality on major adverse cardiovascular events and all-cause mortality: a national population-based study. Nephrology Dialysis Transplantation. 2021;36(5):901-8.
28. Shen CH, Zheng CM, Kiu KT. Increased risk of atrial fibrillation in end-stage renal disease patients on dialysis: A nationwide, population-based study in Taiwan. Medicine. 2016;95(25):3933.
29. Wu CK, Lee JK, Wu YF. Left ventricular diastolic dysfunction in peritoneal dialysis: a forgotten risk factor. Medicine. 2015;94(20):819.
30. Chang TI, Nam JY, Shin SK, Kang EW. Low Triiodothyronine Syndrome and Long-Term Cardiovascular Outcome in Incident Peritoneal Dialysis Patients. Clin J Am Soc Nephrol. 2015;10(6):975-82.
31. Wang J, Ma X, Si X, Wu M, Han W. Mean platelet volume and the association with all-cause mortality and cardiovascular mortality among incident peritoneal dialysis patients. BMC Cardiovasc Disord. 2023;23(1):3551.
32. Demir E, Çalışkan Y. Transplant immunology research centre of excellence, KOÇ university hospital, KOÇ university, Istanbul, Türkiye, outcome and risk factors for mortality in peritoneal dialysis patients: 22 Years of Experience in a Turkish Center. Turkish J Nephrol. 2024;33(2):223-8.
33. Zhang L, Nie Y, Guo M. Neutrophil to Lymphocyte Ratio as a Predictor of Long-Term Outcome in Peritoneal Dialysis Patients: A 5-Year Cohort Study. Blood Purif. 2021;50(6):772-8.
34. Pecoits-Filho R, Yabumoto FM, Campos LG. Peritonitis as a risk factor for long-term cardiovascular mortality in peritoneal dialysis patients: The case of a friendly fire. Nephrol. 2018;23(3):253-8.
35. Ma H, Chen J, Zhan X. Platelet-to-albumin ratio: a potential biomarker for predicting all-cause and cardiovascular mortality in patients undergoing peritoneal dialysis. BMC Nephrol. 2024;25(1):8.
36. Hsieh YP, Chang CC, Kor CT. Relationship between uric acid and technique failure in patients on continuous ambulatory peritoneal dialysis: A long-term observational cohort study. BMJ Open. 2017;7(4):1138.
37. Li J, Huang N, Zhong Z. Risk factors and outcomes of cardiovascular disease readmission within the first year after dialysis in peritoneal dialysis patients. Renal Failure. 2021;43(1):159-67.
38. Xia X, Qiu Y, Yu J. Ten-year survival of patients treated with peritoneal dialysis: A prospective observational cohort study. Perit Dial Int. 2020;40(6):573-80.
39. Dai L, Yang Y, Liu L. The association of left ventricular fraction shortening with cardiovascular events in peritoneal dialysis patients. Renal Failure. 2023;45(2):1786.
40. Xu LC, Zhou FF, Li M, et al. The correlation between low serum T3 levels and all-cause and cardiovascular mortality in peritoneal dialysis patients. Therap Clin Risk Man. 2021;17:851-61.
41. Lozier MR, Sanchez AM, Lee JJ, Tamariz LJ, Valle GA. Comparison of Cardiovascular Outcomes by Dialysis Modality: A Systematic Review and Meta-Analysis. Perit Dial Int. 2019;39(4):306-14.
42. London GM. Arterial media calcification in end-stage renal disease: impact on all-cause and cardiovascular mortality. Nephrol Dial Transpl. 2003;18(9):1731-40.
43. Cooper BA, Penne EL, Bartlett LH, Pollock CA. Protein malnutrition and hypoalbuminemia as predictors of vascular events and mortality in ESRD. American J Kid Dis. 2004;43(1):61-6.
44. Stenvinkel P, Heimbürger O, Paultre F. Strong association between malnutrition, inflammation, and atherosclerosis in chronic renal failure. Kidney International. 1999;55(5):1899-911.
45. Sakaguchi Y, Fujii N, Shoji T, Hayashi T, Rakugi H, Isaka Y. Hypomagnesemia is a significant predictor of cardiovascular and non-cardiovascular mortality in patients undergoing hemodialysis. Kidney Int. 2014;85(1):174-81.
46. Faitatzidou D, Karagiannidis AG, Theodorakopoulou MP, Xanthopoulos A, Triposkiadis F, Sarafidis PA. Autonomic Nervous System Dysfunction in Peritoneal Dialysis Patients: An Underrecognized Cardiovascular Risk Factor. Am J Nephrol. 2024;55(1):37-55.
47. Sahlawi MA, Wilson G, Stallard B, et al. Peritoneal dialysis-associated peritonitis outcomes reported in trials and observational studies: A systematic review. Perit Dial Int. 2020;40(2):132-40.

48. Jiang C, Zheng Q. Outcomes of peritoneal dialysis in elderly vs non-elderly patients: A systemic review and meta-analysis. PLoS ONE. 2022;17(2):263534.
49. Assimon MM, Wenger JB, Wang L, Flythe JE. Ultrafiltration Rate and Mortality in Maintenance Hemodialysis Patients. American J Kidney Dis. 2016;68(6):911-22.

Cite this article as: Kakkadapram HV, Huq MS. Long-term cardiovascular outcomes in peritoneal dialysis patients: a systematic review. Int J Adv Med 2025;12:434-46.