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Review Article

Managing Advanced Diabetic Kidney Disease in the UAE: National Consensus Recommendations for Dialysis, Transplantation, and Emergencies

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Abstract

Background Diabetic kidney disease (DKD) is the leading cause of end-stage kidney disease (ESKD) in the UAE, contributing substantially to cardiovascular morbidity and mortality.

Purpose To provide UAE-specific expert recommendations for managing advanced DKD, including stage 5 disease, dialysis, transplantation, and post-transplant metabolic complications.

Methods A multidisciplinary panel of 14 UAE nephrology and endocrinology experts used a Modified Mini-Delphi process to adapt international guidelines (ADA, KDIGO, NICE, UAE Cardiometabolic) to regional clinical practice. Consensus was defined as $\geq$80% agreement.

Results Recommendations emphasize individualized glycemic and blood pressure control during dialysis, early recognition and treatment of post-transplant diabetes, and targeted management of DKA and hyperosmolar states in dialysis patients. Integration of SGLT2 inhibitors, GLP-1 receptor agonists, and multidisciplinary digital care models was endorsed.

Conclusion These UAE consensus recommendations provide unified, evidence-based guidance for advanced DKD management, forming a nationwide framework to prioritize lives through standardized care and education.

Keywords
- diabetes mellitus
- reno-metabolic
- diabetic kidney disease
- end-stage renal disease
- dialysis

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Introduction

Diabetic kidney disease (DKD) is a serious chronic vascular complication of diabetes and a leading cause of end-stage renal disease (ESRD), contributing substantially to cardiovascular (CV) morbidity and mortality.[1] Chronic kidney disease (CKD) is estimated to affect over 10% of the general population worldwide, representing more than 800 million people; DKD accounts for a substantial proportion of this burden.[2] The number of people developing kidney failure and requiring kidney replacement therapy (KRT) is estimated to increase to 5.4 million by 2030.[3]

The prevalence of DKD mirrors that of diabetes in the United Arab Emirates (UAE); almost one in two UAE nationals with type 2 diabetes (T2D) has moderately increased albuminuria (formerly microalbuminuria), a key biomarker of early DKD.[4] In a single-center analysis at Dubai Hospital HD unit (January 2012–October 2016), DKD was identified as the primary cause of ESRD, accounting for 57% of the dialysis population; however, this single-center finding may not be fully representative of the national picture.[4] Within the Gulf Cooperation Council (GCC) countries, the diabetes prevalence among hemodialysis (HD) patients varies regionally, with Kuwait showing 74%, Bahrain 72%, Qatar and the UAE 68%, Oman 59%, and Saudi Arabia 45%.[5]

Advances in pharmacotherapy have delayed progression through earlier stages of DKD, yet the population living with ESRD continues to expand.[6] Management of these patients demands an integrated reno-metabolic approach, a term adopted by the Emirates Diabetes and Endocrine Society to describe the clinical framework addressing the bidirectional relationship between kidney disease and metabolic disorders, principally diabetes, that balances glycemic safety, hemodynamic stability, and cardio-protection.[7] This article revisits key aspects of late-stage DKD management, including HD and peritoneal dialysis (PD), blood pressure optimization, and the complex landscape of post-transplant diabetes mellitus (PTDM). It also highlights the potential role of multidisciplinary coordination in this high-risk group.

The management of advanced DKD in the UAE is shaped by distinctive regional factors. A diverse population with many transient expatriates can disrupt long-term care continuity and necessitate digital health tracking to maintain follow-up.[8] Fasting during Ramadan may increase risks such as dehydration and glycemic fluctuations, requiring tailored protocols for patients with advanced DKD, especially those on dialysis or with kidney transplants.[9] Differences in healthcare resources and infrastructure across the seven emirates further underscore the need to adapt international guidelines to local realities, ensuring equitable access to best-practice care across the country.

Materials and Methods

This article complements a previously published UAE Reno-Metabolic Consensus, which addresses stages 1 to 4 DKD.[10] The current article focuses on the management of stage 5 DKD, including glycemic control during dialysis, blood pressure management, kidney transplantation, and post-transplant care.

The expert panel was convened under the leadership of the Emirates Diabetes and Endocrine Society and the Emirates Medical Association–Nephrology and Transplant Society. Panelists were identified through a formal nomination process led by both societies. Eligibility criteria included a minimum of 5 years of specialist clinical experience in nephrology or endocrinology, active practice in the UAE, and affiliation with a major healthcare institution. Representatives were sought from across all seven emirates to ensure national coverage. A total of 18 experts were invited, of whom 14 participated in the full consensus process, ensuring comprehensive representation across major healthcare institutions and disciplines.

Draft statements covering the management of stage 5 DKD, dialysis, and post-transplant care were derived from international guidelines (ADA, KDIGO, NICE) and regional UAE cardiometabolic guidelines. Voting was conducted using a 5-point Likert scale (1 = strongly disagree to 5 = strongly agree), with consensus predefined as $\geq$80% of panelists rating a statement 4 or 5. All survey voting rounds were anonymous and administered via a secure online platform. The consensus process involved two structured rounds of anonymous survey voting. Statements achieving borderline agreement (approximately 75%) during the initial survey were brought to a virtual consensus meeting facilitated by an independent moderator with no voting rights. During these meetings, the underlying evidence was discussed, and statements were subsequently revised and subjected to a re-vote until the $\geq$80% threshold was achieved. To mitigate the influence of dominant views, all survey votes were blinded to individual identities. The final approved statements are presented as recommendations within this article.

All panel members completed a formal conflict of interest (COI) declaration prior to participation. Declared COIs were reviewed by the steering committee; any panelist with a direct financial relationship to a topic under discussion was required to abstain from voting on the relevant statement. Individual COI declarations are included as supplementary material.

To support readers in calibrating the strength of recommendations, each recommendation is labeled as: (a) Guideline-derived (adapted from a named international guideline), (b) guideline-informed expert consensus (synthesizing international evidence with regional expert judgment), or (c) expert consensus (primarily based on panel agreement where direct guideline data are limited or unavailable). A limitation of this consensus is the absence of a formal evidence-grading framework (such as GRADE) for the final UAE-specific statements. While foundational guidelines such as KDIGO and ADA are heavily graded, the regional adaptations presented here rely on expert consensus ($\geq$80% agreement) to bridge gaps between international trial data and local clinical realities.

Results

Management of DKD Stage 5

Definition of CKD and ESRD

According to KDIGO 2024, CKD is defined as abnormalities of kidney structure or function, present for more than 3 months, with implications for health.[11] These abnormalities may include a glomerular filtration rate (GFR) <60 mL/min/1.73 m^2, albuminuria (urine albumin ≥30 mg per 24 hours or urine albumin-to-creatinine ratio [ACR] ≥30 mg/g [>3 mg/mmol]), abnormalities in urine sediment, histology, or imaging suggestive of kidney damage, renal tubular disorders, or a history of kidney transplantation.[11] ESRD is defined as a GFR of <15 mL/min. As kidney function declines, timely nephrology referral ideally at CKD stage 4 (eGFR <30 mL/min/1.73 m^2) or earlier in cases of rapid progression or significant albuminuria is essential to allow shared decision-making, transplant evaluation, and preparation for kidney replacement therapy before stage 5 is reached.[12]

Physiological Changes in Insulin Degradation

Under physiological conditions, filtered insulin is primarily reabsorbed in the proximal tubules of the kidney and, to a lesser extent, by peritubular cells, where it undergoes degradation into peptide fragments.[13] In patients with CKD, a reduced GFR leads to decreased insulin degradation, resulting in lower insulin requirements.[14] Paradoxically, CKD is also recognized as a state of insulin resistance, a condition that often improves with the initiation of dialysis therapy.[14]

Managing diabetes in patients with CKD and those undergoing dialysis presents unique challenges.[11] According to the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines, the use of insulin is strongly recommended for dialysis patients, particularly formulations that closely mimic human physiology.[11] Conversely, certain oral hypoglycemic agents should be used with caution or avoided altogether in this population.[11]

At the initiation of dialysis, glycemic control should be reassessed using HbA1c and/or self-monitoring of blood glucose (SMBG).[15] Patients with persistent hyperglycemia despite oral therapy should be considered for insulin therapy, with dose adjustments guided by frequent glucose monitoring to reduce hypoglycemia risk.[16]

For dialysis patients, insulin therapy may involve using either basal insulin alone or as part of a multiple daily injection (MDI) regimen.[16] Notably, the initial insulin dose for dialysis patients should be reduced to approximately 50% of the dose used in pre-dialysis patients, with subsequent adjustments based on SMBG readings and HbA1c levels.[17] For patients initiating PD, oral hypoglycemic agents may be continued if glycemic control is maintained; however, insulin may be required to achieve and sustain glycemic targets.[18]

For patients undergoing HD or PD and receiving insulin therapy, the route of administration requires careful consideration.[19] Insulin can be administered either subcutaneously or intraperitoneally for PD patients, although the intraperitoneal route often requires higher doses due to adsorption of insulin onto the plastic surfaces of dialysate solutions; intraperitoneal doses may need to be approximately twice the doses required for subcutaneous administration.[19] Despite its benefits, intraperitoneal insulin administration has several drawbacks, including an increased risk of bacterial infections, fibroblastic proliferation, and hepatic subcapsular steatonecrosis. Another significant limitation is the variability in insulin dose delivery via the intraperitoneal route, which complicates achieving stable glycemic control.[20]

Patients undergoing PD or HD while receiving insulin therapy require meticulous adjustment of their regimen to mitigate the high risk of hypoglycemia associated with dialysis.[21] Individuals treated with premixed insulin are advised to reduce their morning dose by approximately 50% prior to dialysis.[22] In contrast, those on MDI require a more stratified approach: basal insulin administered in the morning should be reduced by 40 to 50%, whereas basal doses given at night should be tapered by 30 to 40% before the subsequent dialysis session.[23] Similarly, rapid-acting bolus insulin doses are commonly decreased by 50% in the morning.[23] Across all regimens, continuous monitoring of blood glucose levels during and after dialysis is essential to ensure glycemic stability and prevent adverse metabolic events[11] (►**Fig. 1**).

Newer treatment options for CKD have resulted in updated global recommendations emphasizing a balanced approach that combines these therapies with existing effective treatments (►**Fig. 2**).

Specific Considerations for Peritoneal Dialysis

While PD offers benefits such as continuous fluid removal and preservation of residual renal function, it poses metabolic challenges for patients with DKD.[24] Continuous absorption of glucose from dextrose-based dialysate can worsen hyperglycemia, increase insulin needs, and promote weight gain and hyperlipidemia.[25] Careful glycemic monitoring is therefore required, and non-glucose osmotic agents such as icodextrin may be considered, particularly for long dwell exchanges.[25] Intraperitoneal insulin is an option but often requires about twice the subcutaneous dose due to adsorption to dialysate bags and carries risks such as peritonitis and hepatic complications.[26] Maintaining euvolemia is also essential, as persistent fluid overload can reduce the effectiveness of antihypertensive therapies such as ACE inhibitors and angiotensin II receptor blockers.[27]

Blood Pressure Management in Patients with DKD Receiving HD or PD

Hypertension is a major risk factor for CV disease (CVD) and a main cause of morbidity and mortality in the dialysis population. The management of hypertension in dialysis patients is complex, involving multiple contributing factors and therapeutic approaches.[28] Extracellular fluid (ECF) volume expansion, reflected in part by interdialytic weight gain, is a major contributor to hypertension in dialysis patients, alongside increased sympathetic activity, stimulation of the renin angiotensin aldosterone system (RAAS), and arterial stiffness contributing to systolic hypertension.[29] In patients with underlying obesity, elevated BMI may contribute to hypertension through additional mechanisms including increased cardiac output and neurohumoral activation, independent of

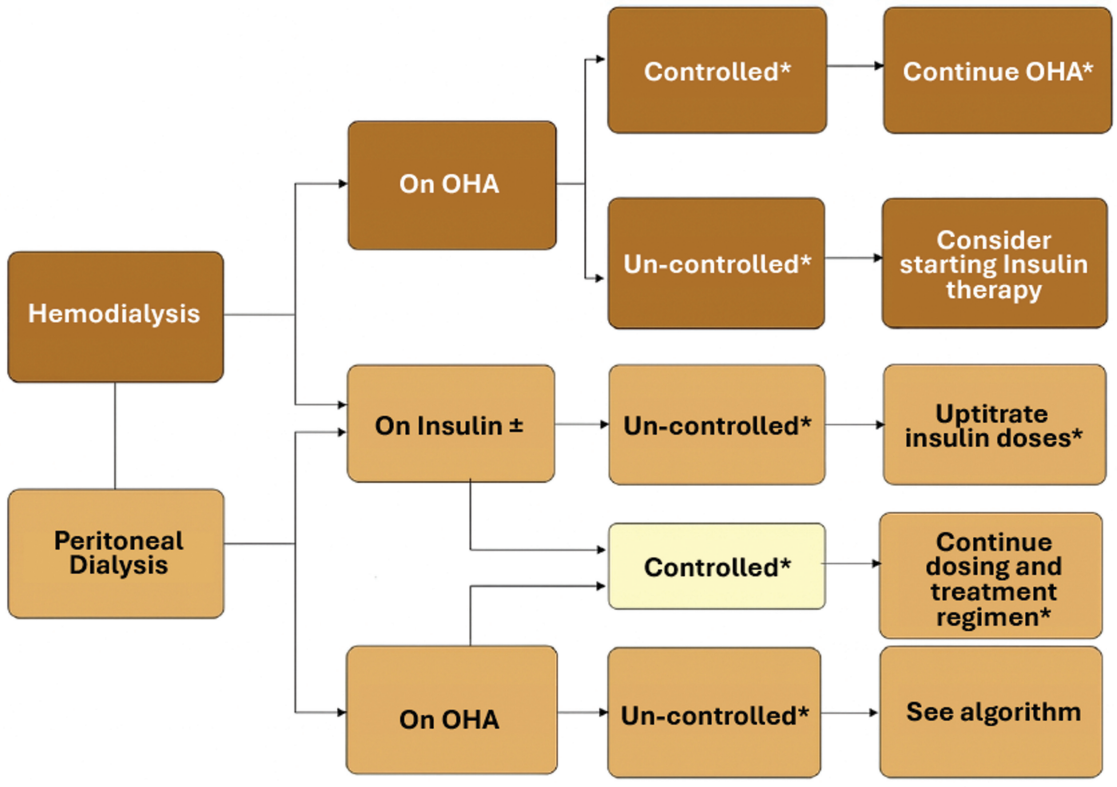

Fig. 1 Expert-recommended care pathway for proactive management of patients undergoing peritoneal dialysis (PD) or hemodialysis (HD) on insulin.

volume status.[30] The reference standard for diagnosis of hypertension in patients undergoing HD is 44-hour interdialytic ambulatory blood pressure (BP) monitoring, which demonstrates stronger associations with left ventricular hypertrophy and all-cause mortality than pre- or post-dialysis BP measurements.[31] It is best diagnosed by measuring BP intermittently every 20 to 30 minutes over the 44-hour interdialytic interval using a fully automated ambulatory BP monitor.[31] In a meta-analysis of RCTs in patients undergoing HD, BP reduction with antihypertensive treatment was associated with a 29% reduction in risk of CV events and a 20% reduction in all-cause mortality, highlighting the importance of routine BP control in individuals undergoing dialysis.[32]

Recommendation 1 (Guideline-informed expert consensus): Maintain BP below 130/80 mm Hg in both HD and PD patients. In patients aged ≥70 years, consider a more individualized approach with a systolic BP (SBP) target of 140 to 150 mm Hg, recognizing that lower targets may not be well-tolerated in this population due to increased risk of orthostatic hypotension, falls, and adverse cardiovascular events associated with aggressive BP lowering in frail elderly dialysis patients.

Intradialytic hypertension (IDH) remains variably defined. KDIGO defines it as an SBP rise of >10 mm Hg from pre- to post-dialysis in the hypertensive range.[32] Rising intradialytic BP that is unresponsive to volume removal in at least four of six consecutive HD treatments is another defining criterion.[33]

Optimal BP control requires normalizing extracellular fluid volume and maintaining a "dry weight" through ultrafiltration (UF) on dialysis. Gradual reduction of weight over several dialysis sessions is recommended, as there may be a delay between fluid control and BP reduction.[34] Rapid UF should be avoided to prevent intradialytic hypotension, necessitating saline use and hampering the achievement of dry weight. Weight gains between dialysis sessions should be limited to <2 kg by restricting salt and fluid intake.[35]

Antihypertensive therapy in dialysis patients should be carefully managed to avoid hypotension.[36] β-blockers can mitigate sympathetic overactivity but may cause hypotension by blocking baroreceptor responses; short-acting agents should be avoided on dialysis days for patients prone to hypotensive episodes.[37] Calcium channel blockers are useful but can prevent the reflex arteriolar vasoconstriction necessary to prevent IDH.[38] α-blockers are considered safe, as they do not increase hypotension risk.[39] Angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin II receptor blockers (ARBs) are preferred in patients with left ventricular

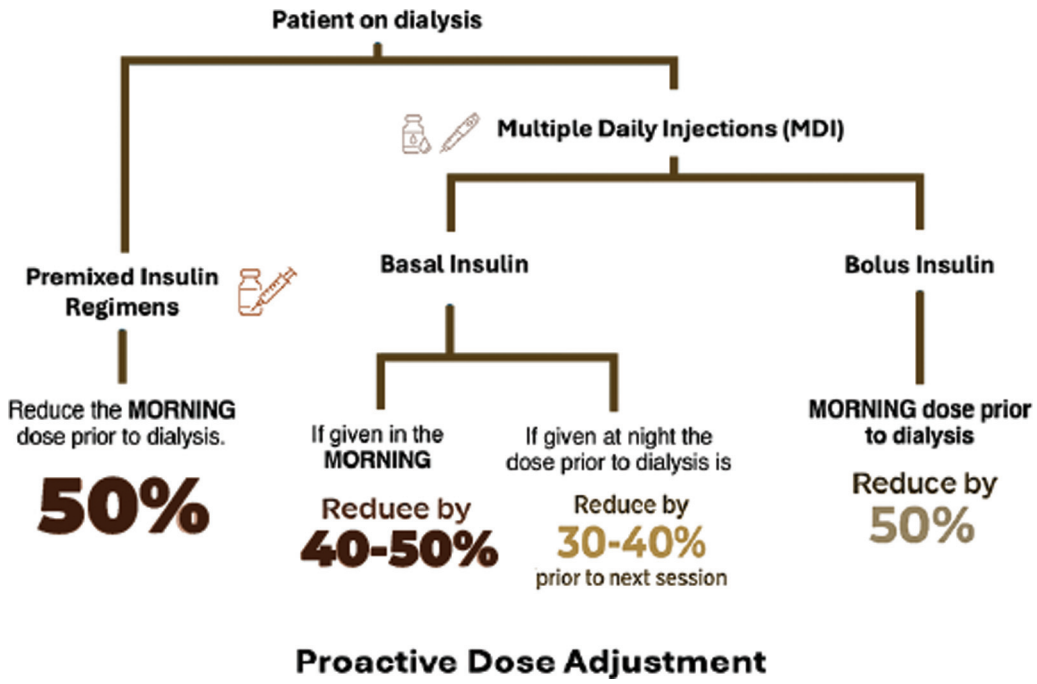

Fig. 2 Management of diabetes in patients receiving dialysis.

dysfunction, as they help preserve residual renal function, but may cause hypotension with volume depletion or provoke anaphylactic reactions with certain dialysis membranes.[40] However, they may be less effective in patients undergoing PD with persistent fluid overload.[41] Most antihypertensive agents can be used safely in patients with DKD on PD.[42] A reduction in extracellular water has been reported to be associated with regression of left ventricular mass index (LVMI).[43]

Recommendation 2 (Guideline-informed expert consensus): For patients experiencing pre-dialysis hypotension, it is crucial to determine whether they are volume-depleted; if so, fluids should be administered. Antihypertensive medications should be stopped or reduced, and patients should be allowed to remain at a new dry weight. If hypotension persists without volume depletion or drug use, poor cardiac function may be the cause, warranting echocardiography.

Management of Post-transplant Diabetes

Post-transplant diabetes mellitus (PTD) is a frequent complication of kidney transplantation (KT), occurring in up to 50% of high-risk KT recipients (KTRs).[44] Several pre-transplant risk factors, such as older age, obesity, male gender, genetic background, or hypertriglyceridemia, increase the risk of PTD.[45] Managing people with DKD post-KT involves a tailored approach that balances glycemic control, immunosuppressive therapy adjustments, lifestyle interventions, and careful monitoring to maximize patient and graft survival.[46] Continuous monitoring and multidisciplinary collaboration are vital for improving patient outcomes in this high-risk population.[47]

Recommendation 3 (Guideline-derived [ADA; KDIGO 2022]): Conduct consistent monitoring of post-transplant blood glucose levels, as PTD can develop due to immunosuppressive drugs. Frequent HbA1c is strongly recommended for continuous glucose control and improved patient outcomes.

Recommendation 4 (Guideline-derived [KDIGO 2022]): An oral glucose tolerance test (OGTT) should be performed pre-transplant to detect those at risk of developing diabetes. It should also be performed at 12 weeks post-transplant, as HbA1c early post-transplant is not reliable.

Lifestyle Modifications

Diet and Exercise

Dietary adjustments focusing on low-glycemic index foods and a balanced intake of carbohydrates, proteins, and healthy fats can help maintain stable glucose levels.[48] Regular physical activity is recommended, as multiple randomized clinical trials (RCTs) have highlighted that higher physical activity lowers the risk of PTD. Consuming the Mediterranean diet may also help reduce the risk of PTD after kidney transplant.[49]

Weight Management

Obesity is a risk factor for PTD; evidence supports maintaining a healthy BMI through diet and exercise to reduce post-transplant complications.[50] Education on risk factors for PTD, glucose monitoring best practices, and adherence to lifestyle recommendations can enhance patient outcomes.[50] Regular follow-ups with a multidisciplinary team (MDT), including nephrologists, endocrinologists, and dietitians, improve care continuity.[51]

A retrospective observational study suggested that intensive lifestyle modifications (dietitian referral, graded exercise program, and weight loss advice) for 6 to 12 months may reduce and, in some cases, reverse the progression of glyco-metabolic derangements in patients with altered post-prandial glucose metabolism; however, these findings should be interpreted with caution given the observational design, and further prospective data are needed to confirm this association.

Monitoring kidney function is critical as hyperglycemia can contribute to transplant kidney injury. Regular creatinine and eGFR assessments help to detect any dysfunction early.[12] Diabetes and immunosuppressive drugs elevate CV risk; thus, regular BP control, lipid management, and lifestyle interventions are necessary for CV health.[52]

Modification of Immunosuppression

Modification of immunosuppression in transplant recipients should be approached with caution, especially due to the risk of PTD.[53] However, in select patients where minimizing PTD risk is a priority, individualized adjustments may be considered.[53] Anti-thymocyte globulin (ATG) can be used as an induction agent to potentially reduce PTD risk by allowing for lower calcineurin inhibitor (CNI) exposure.[54] In patients at low risk of rejection, early tapering of corticosteroids is advisable to decrease PTD risk without compromising graft survival.[54] Adjustments to CNI therapy, such as lowering tacrolimus doses or switching to cyclosporine or newer agents like belatacept, have also been shown to help mitigate hyperglycemia in patients at high risk for PTD.[55] Mycophenolate mofetil (MMF) and azathioprine are not linked with increased PTD risk and may be chosen based on the patient's immunological risk profile or institutional protocols.[56] In women planning pregnancy, switching from MMF to azathioprine is considered safe without added PTD risk.[55]

Insulin in Early Postoperative Hyperglycemia

In the immediate postoperative period, insulin is generally the preferred agent for managing hyperglycemia, given its flexibility and titrability.[57] However, the use of other agents, such as glinides, may be considered in specific clinical contexts (e.g., during methylprednisolone-based regimens) under close monitoring.[58] Some studies have suggested that early administration of basal insulin may be effective in preventing the development of PTD in patients with hyperglycemia in the early postoperative period and contribute to improving pancreatic β-cell function.[59] In the case of glucocorticoid-induced hyperglycemia in transplant recipients, the optimal approach may be a combination of long-acting and rapid-acting insulin.[60]

Oral Hypoglycemic Agents

Metformin and DPP-4 Inhibitors

Metformin may be used in stable KTRs, particularly those with adequate renal function, as it is associated with fewer hypoglycemic events.[61] A retrospective observational study suggested that metformin use in PTD may be associated with improved patient and graft survival regardless of donor type (living or deceased), with metformin-based regimens showing a lower incidence of acute rejection, death-censored graft failure, and all-cause mortality compared with insulin-based and sulfonylurea-based regimens; however, these findings should be interpreted with caution given the observational design, and further prospective data are needed to confirm this association.[62]

DPP-4 inhibitors (e.g., sitagliptin) are also a safe option with minimal impact on immunosuppressive therapy.[63] DPP-4 inhibitors are well tolerated, safe, and effective in improving glucose metabolism in transplant recipients.[63]

Sodium–Glucose Cotransporter-2 Inhibitors (SGLT2i) in PTD

SGLT2i are oral hypoglycemic agents that may be preferred for overweight or obese KT patients with PTD, assuming stable graft function, no history of recurrent urinary tract infections (UTIs), and no recent graft rejection episodes.[64]

> **Recommendation 5 (Guideline-informed expert consensus):** SGLT2i may be considered in patients with stable graft function who have not experienced an acute rejection episode in the past 6 months and have no history of recurrent UTIs.

> **Recommendation 6 (Guideline-informed expert consensus):** Consider SGLT2i for kidney transplant patients with heart failure, provided there is stable graft function and there is no recent rejection episode, or history of recurrent UTIs.

KTRs are predisposed to UTIs due to surgical factors and immunosuppression. Furthermore, they are at risk of hypovolemia from diuresis due to improved graft function and acute kidney injury.[65] As a precaution, SGLT2i should be avoided during the first 6 months post-kidney transplant, until the graft stabilizes and reaches a steady state. This minimizes risks of acute rejection, acute kidney injury (AKI), and UTI.[66]

Evidence indicates that SGLT2i use is associated with a significantly lower risk of AKI compared with non-use.[67] Use of empagliflozin in patients with PTD was not associated with any deterioration in kidney function.[68] Moreover,

SGLT2i treatment was associated with a reduced risk of the composite outcome of all-cause mortality, death-censored graft failure (DCGF), and serum creatinine doubling.[69] SGLT2i may also be beneficial in KT patients with proteinuria attributed to PTD or transplant glomerulopathy.[70] In KTRs, initiation is recommended at an eGFR $\geq$30 mL/min/1.73 m^2, as evidence for safety and efficacy at eGFR 20 to 30 in transplant recipients remains limited; initiation at eGFR <30 should be approached with particular caution.[71] In KTRs, clinicians must emphasize the importance of maintaining blood volume; patients must be informed that in volume-depletion situations (e.g., vomiting, diarrhea), SGLT2i should be discontinued.[72] In cases of AKI, ongoing UTI, rejection episodes, or diarrhea, SGLT2i should be withheld until the acute condition resolves.[73]

Injectable Non-insulin Glucose-lowering Agents (NIGLAs)

Glucagon-like Peptide-1 Receptor Agonists (GLP-1 RAs) in PTD

A GLP-1 RA can be considered in obese patients with PTD. A systematic review and meta-analysis of GLP-1 RA use in kidney transplant recipients has suggested that these agents can reduce the need for exogenous insulin and do not increase the risk of allograft failure.[74] GLP-1 RAs are classified as non-insulin glucose-lowering agents (NIGLAs) as most are administered by injection.[75] They normalize abnormalities of the pathophysiological defects found in patients with PTD, namely, reduced glucose-induced insulin secretion and impaired glucagon suppression.[76,77] However, as GLP-1 RAs may be associated with side effects, especially nausea and vomiting, they should be avoided in the early post-transplant phase until graft function is stabilized.[77] They should be slowly uptitrated to avoid these side effects and to prevent volume-depleted AKI and disturbance in absorption of immunosuppressive medicines.[78] Appropriate patient education is also advisable. Advances in pharmacogenomics and biomarker research are leading to more individualized treatment plans, aiming to optimize immunosuppressive therapy with minimal side effects[79] (►**Fig. 3**). The suggested algorithm for diabetes management beyond 6 months post-transplant in patients with stable graft function is outlined in ►**Fig. 4**.

In-hospital Management of Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State (HHS)

DKA is a serious complication of diabetes, and in patients with kidney disease, the clinical presentation of DKA may vary significantly due to the degree of renal impairment and dialysis modality.[80] Decisions on dialysis initiation or adjustments should be made in collaboration with a nephrologist, evaluating the patient's overall condition and response to conservative treatment.[81] In patients with ESRD, diagnostic criteria may differ due to altered renal function's effects on

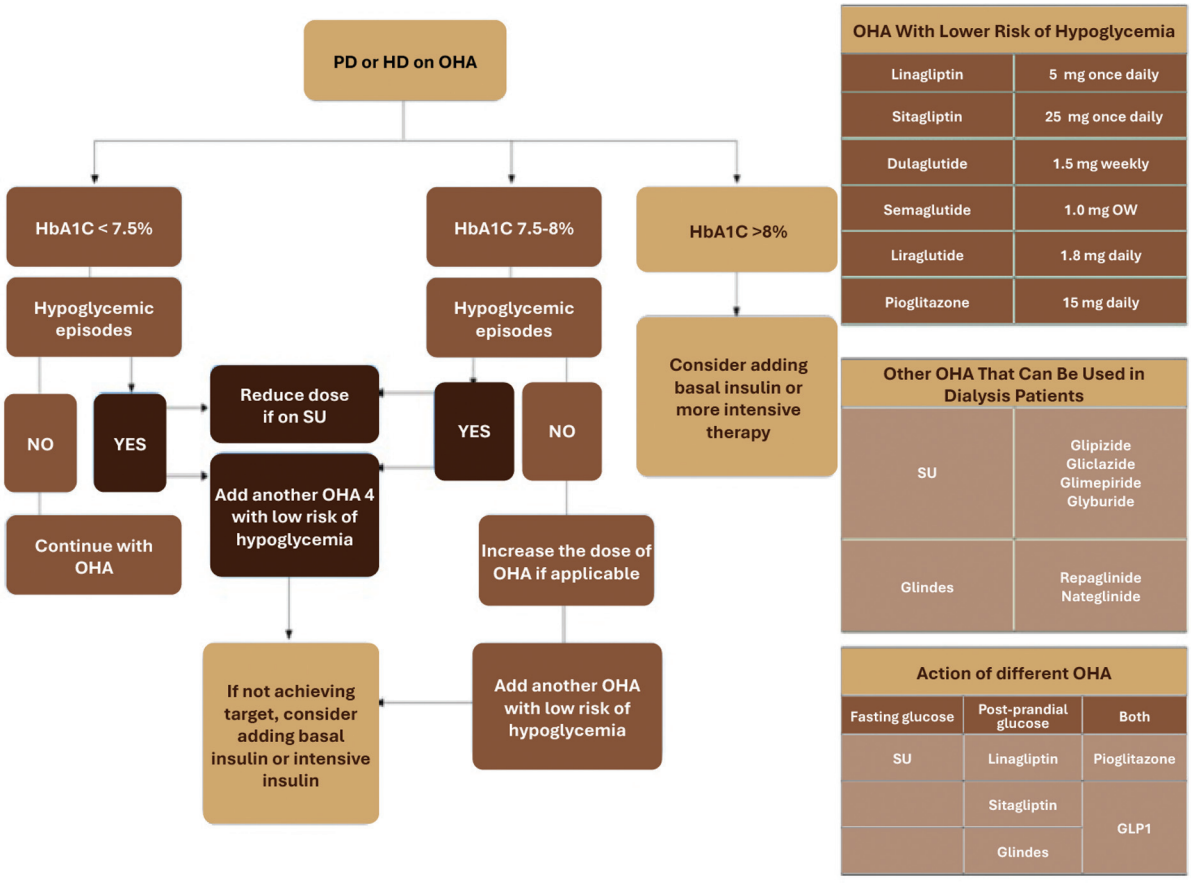

Fig. 3 Suggested algorithm for the management of diabetes post 6 months of transplant in patients with stable graft function.

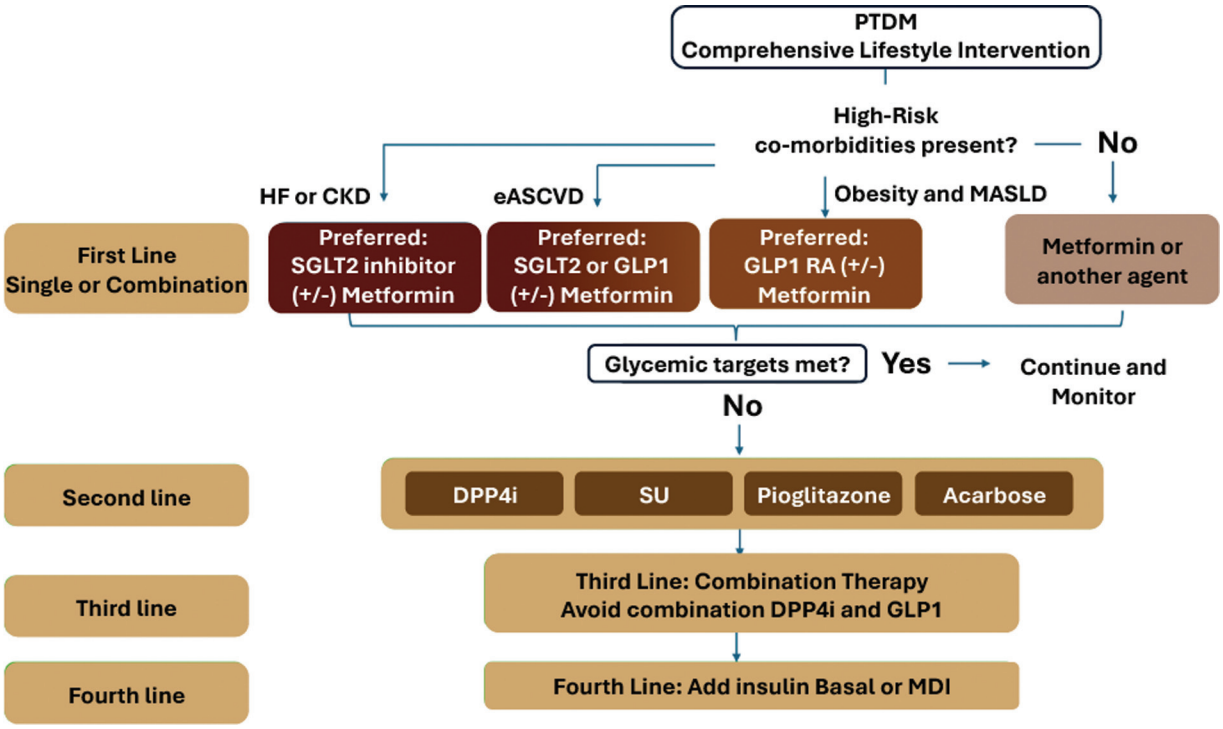

Fig. 4 Suggested algorithm for the management of post-transplant diabetes.

typical DKA markers, making blood β-hydroxybutyrate and anion gap essential for diagnosis (►**Table 1**).

Recommendation 7 (Expert consensus): DKA in patients with ESRD on HD is a rare clinical presentation and difficult to diagnose. Since ESRD is a state of persistent chronic metabolic acidosis, DKA should be suspected in patients with diabetes (especially type 1) who are unwell with an acidosis greater than would normally be expected (persistent metabolic acidosis). In such cases, measurement of serum β-hydroxybutyrate is a useful tool in establishing the diagnosis of DKA in patients with ESRD on HD.

Recommendations 8, 9, 12, and 13 are presented in tabular format within ►**Tables 2** and **3**.

Insulin Therapy and Glucose Management

Recommendation 10 (Guideline-derived [ADA]): Insulin therapy is typically the primary method of glycemic control in patients with DKA, with a target glucose range of 140 to 180 mg/dL for most inpatients. However, tighter control (below 140 mg/dL) may be considered in stable cases, though hypoglycemia should be strictly avoided due to the risks it poses for patients with impaired kidney function.

Table 1 Diagnostic criteria for DKA in dialysis patients[66]

Diagnostic marker	Criteria	Notes
Blood glucose[67]	>13.9 mmol/L	Elevated blood glucose level is a key indicator of DKA
β-Hydroxybutyrate (ketone strips)[66]	Elevated levels	Primary ketone body in DKA; useful in confirming DKA in ESRD patients
Arterial venous pH[67]	≤7.30	Indicates metabolic acidosis commonly seen in DKA
Bicarbonate[67]	≤18 mEq/L	Low bicarbonate level is consistent with metabolic acidosis in DKA
Anion gap[67]	>10–12 (adjusted for albumin), typically ≥20	A high anion gap supports DKA diagnosis; >20 is more specific in ESRD patients
Ketonuria and glycosuria[66]	May be absent in ESRD patients	Due to reduced renal clearance or anuria, ketonuria/glycosuria may not be prominent

Abbreviations: DKA, diabetic ketoacidosis; ESRD, end-stage renal disease.

Table 2 Expert consensus recommendations outlining the management of hyperosmolar states in patients with DKD and ESRD, emphasizing controlled correction of hyperglycemia, cautious use of hemodialysis, and individualized fluid and electrolyte management[68,69]

Management aspect	Expert recommendations
Gradual correction [68,69]	ESRD patients with DKA are often hypertonic with low serum sodium due to extracellular water shifts. **Recommendation 8:** Rapid correction of hyperglycemia increases the risk of cerebral edema and central pontine myelinolysis; hence, gradual correction of hyperglycemia with insulin infusion is recommended, targeting a drop of 2.8 to 4.2 mmol/L/hr (50–75 mg/dL/hr). In case of emergency hemodialysis, CRRT is preferred as it allows more controlled reduction of glycemia and maintenance of electrolyte balance.
Emergency HD[68,69]	In cases of extreme hyperglycemia, emergency HD may be necessary but must be approached with caution due to rapid changes in tonicity that could destabilize the patient. CRRT is often preferred, as it allows for more controlled correction of serum osmolality and electrolyte balance.
Fluid replacement [68,69]	Unlike typical DKA patients who require aggressive fluid resuscitation due to osmotic diuresis, HD patients are often euvolemic or hypervolemic. **Recommendation 9:** In patients with DKA and ESRD on HD, fluid resuscitation with boluses of 250 to 500 mL should be guided by continuous re-evaluation of volume status.
Rapid sodium changes [68,69]	Serum sodium should be corrected slowly, as rapid changes can result in neurological complications. For patients with normal to high serum sodium, hypotonic fluids are generally avoided. If blood glucose falls below 13.9 mmol/L, adding dextrose to the saline infusion helps prevent hypoglycemia.

Abbreviations: CRRT, continuous renal replacement therapy; DKA, diabetic ketoacidosis; DKD, diabetic kidney disease; ESRD, end-stage renal disease; HD, hemodialysis.

Table 3 Expert consensus recommendations for managing electrolyte disturbances, acidosis, and monitoring in DKD and ESRD patients with DKA

Aspect	Expert recommendations
Potassium management	**Hyperkalemia:** Many HD patients present with hyperkalemia due to impaired renal excretion and insulin deficiency. Routine potassium replacement is generally unnecessary, and excessive levels warrant cardiac monitoring. **Recommendation 12:** Patients with DKA and ESRD on HD do not require routine potassium replacement, as these patients usually have hyperkalemia at presentation. In the presence of biochemical hypokalemia, total body potassium stores may still be elevated due to poor excretion. Cardiac monitoring is important in patients with hyperkalemia. **Hypokalemia correction:** If initial potassium levels are low (<3.5 mmol/L), cautious replacement is required due to the risk of fatal cardiac arrhythmias. Dialysis may be needed for severe hyperkalemia with ECG changes. In patients with serum potassium <3.3 mEq/L, insulin therapy should be delayed until potassium levels are corrected above 3.3 mEq/L. Potassium replacement should be initiated at 10 to 20 mEq/h.
Phosphate management	**Hyperphosphatemia:** Common in ESRD patients; insulin therapy can cause a rapid decline in serum phosphate. Replacement is indicated only in cases of severe hypophosphatemia, as overcorrection can induce hypocalcemia.
Magnesium management	ESRD patients are prone to magnesium abnormalities. Although chronic deficiency may contribute to insulin resistance, there is no evidence supporting routine magnesium supplementation in DKA.
Acidosis management	**Bicarbonate therapy:** Routine bicarbonate infusion is not recommended in DKA management, as it may exacerbate acidosis and cause hypokalemia. Dialysis using a bicarbonate dialysate can correct severe metabolic acidosis gradually.[76–81] **Recommendation 13:** In anuric patients, severe and persistent metabolic acidosis may occur at presentation. The preferred treatment is emergency hemodialysis; bicarbonate administration is not recommended for acidosis correction. **Emergency HD and CRRT:** If profound metabolic acidosis persists, emergency HD with bicarbonate may be required. CRRT is preferred for hemodynamically unstable patients, allowing gradual correction of acid–base imbalance.
Monitoring and adjustments	**Neurological monitoring:** Monitor for signs of cerebral edema, particularly during the first 8 to 12 hours of therapy when rapid osmotic shifts increase risk. **Continuous assessment:** Frequent monitoring of blood glucose, electrolytes, and acid–base balance is essential for real-time adjustment of insulin, fluid, and electrolyte therapy to prevent hypoglycemia, fluid overload, and electrolyte disturbances.

Abbreviations: CRRT, continuous renal replacement therapy; DKA, diabetic ketoacidosis; DKD, diabetic kidney disease; ESRD, end-stage renal disease; HD, hemodialysis.

Several studies have demonstrated that administering subcutaneous doses of rapid-acting insulin analogs such as insulin lispro and insulin aspart every 1 to 2 hours is an effective alternative to IV infusion of regular insulin for timely resolution of DKA.[40,82]

Recommendation 11 (Expert consensus): Insulin therapy is the mainstay of treatment of DKA in patients with ESRD on HD. It suppresses ketone body synthesis, reduces blood glucose, and corrects electrolyte imbalances, particularly hyperkalemia. Insulin dosing should be more conservative (0.05–0.07 units per hour) compared with those with normal kidney function. Dose adjustment is recommended to achieve a desirable drop in glucose and prevent hypoglycemia.

The treatment goals in DKA are to reduce plasma ketones by ≥0.5 mmol/L/hour, achieve a rise in bicarbonate of ≥3 mmol/L/hour, and reduce blood glucose by 50 to 75 mg/dL/hour.[83] Regular blood glucose and ketone monitoring allow for real-time adjustment of insulin infusion rates.[84] When blood glucose levels fall below 250 mg/dL, dextrose should be added to the saline infusion to prevent hypoglycemia, especially since ESRD patients have altered glucose metabolism.[84]

SMBG is crucial for effective diabetes management, especially in insulin-dependent individuals who must tailor insulin doses and prevent glycemic imbalances.[85] Non-insulin users may monitor blood glucose less frequently based on treatment goals but may still find SMBG useful.[85] In hospitals, SMBG should ideally integrate into electronic health records (EHRs) to track trends. Continuous glucose monitoring (CGM) is increasingly recommended for inpatient care, offering real-time data and supporting timely adjustments in therapy.[86] ►**Tables 2** and **3** illustrate the consensus recommendations for managing and monitoring DKD and ESRD patients with DKA. ►**Fig. 5** explains the DKA management pathway in dialysis patients.

BP Management in DKA

Severe hypertension may develop in patients with DKA even in the presence of dehydration and is frequently linked to

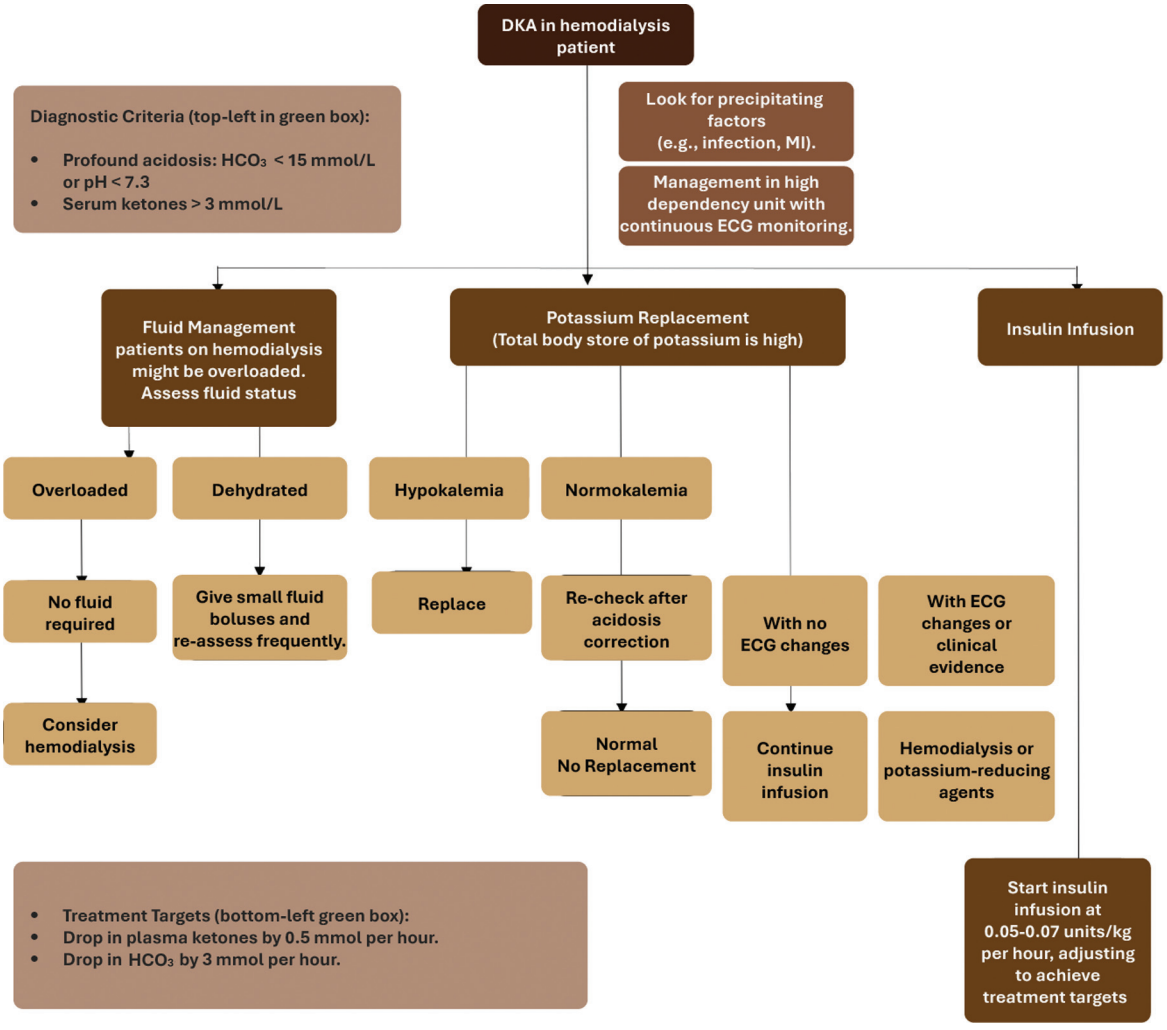

Fig. 5 Diabetic ketoacidosis (DKA) management pathway in dialysis patients.

greater degrees of metabolic acidosis and altered consciousness.[87]

Recommendation 27 (Guideline-informed expert consensus): Strict BP control, with a target of <130/80 mm Hg, is essential in patients with DKA. ACEi or ARBs are recommended as first-line antihypertensive therapy once euvolemia has been established and AKI has been excluded or resolved, given their renoprotective and antiproteinuric effects. These agents should be withheld or discontinued in the presence of active volume depletion, hyperkalemia, or acute kidney injury. If hypertension persists despite first-line treatment, diuretics (thiazides or loop diuretics) should be added. For patients whose BP remains uncontrolled, therapy should be further intensified by adding calcium channel blockers, vasodilators, or centrally acting agents, with the specific choice tailored to individual patient factors and tolerability.

In-hospital DKD Care Technology

In-hospital care of patients with advanced DKD and dialysis patients can be enhanced through the integration of various technologies.[88] Continuous glucose monitoring (CGM) offers significant advantages for dialysis patients by providing real-time glucose data, reducing the frequency of hypoglycemic events, and enabling more precise insulin adjustments during and between dialysis sessions.[89] Insulin pump therapy, where appropriate, may offer improved glycemic control compared with multiple daily injections in select dialysis patients. Electronic prescribing and medicines administration (EPMA) systems and point-of-care testing (POCT) improve the accuracy and timeliness of glucose and electrolyte management in dialysis units.[90] Integration of glucose data into EHRs allows for trend analysis and supports coordinated care between nephrology and endocrinology teams.[91]

Meaningful improvements in health outcomes for stage 5 DKD patients, including slowing disease progression and reducing mortality, require a coordinated multidisciplinary approach to service delivery that integrates nephrology and diabetes care.[51] Several observational studies have demonstrated the effectiveness of multidisciplinary clinics in preserving kidney function in patients with CKD.[92] Innovations in service delivery, including combined diabetes–nephrology clinics, primary care–based satellite clinics, and specialized nurse training, are fundamental in managing advanced CKD effectively and equitably.[93] The combined diabetes–nephrology clinic model, cited by ADA-KDIGO consensus guidelines, shows promise in reducing progression to ESRD and CVD events.[11] Separate systematic reviews found that MDTs in CKD care reduced risk of all-cause mortality and temporary catheterization for dialysis, and possibly also a reduced risk of hospitalization, versus non-MDT care.[94] MDTs also decreased risk by 36% for kidney replacement in patients with advanced CKD and showed cost benefits versus non-MDT care.[51]

Patients in these clinics are often better educated on DKD self-management, leading to higher adherence to dietary and pharmacological regimens.[95] Specialized DKD training for nurses should cover areas such as BP management, dietary advice, DKD staging, and patient education on self-care and understanding DKD progression.[96] Studies demonstrate that nurse-led interventions are effective in maintaining patient engagement, conducting routine assessments, and identifying early signs of disease exacerbation.[96]

Conclusion

The growing burden of DKD in the UAE reflects an urgent public health challenge that demands proactive, coordinated action. These UAE-specific expert recommendations aim to provide a unified, evidence-based framework to guide clinicians in managing advanced stages of DKD, including dialysis and post-transplant care. By aligning international best practices with local clinical realities, the consensus seeks to close existing care gaps, promote early intervention, and ensure equitable access to multidisciplinary management across the country, thereby mitigating disease progression, reducing complications, and improving long-term survival and quality of life for patients living with DKD. Together with the earlier publication addressing stages 1 to 4 DKD, these UAE Reno-Metabolic Guidelines reflect a shared national commitment to prioritize lives through unified, evidence-based guidance, clinical practice, and education across the country.

Authors' Contributions

A.B.: principal author for the manuscript; A.B., M.A.R., A.K. A., D.H., M.H., E.I., F.R., M.A., D.N.K., Fak.A., B.A., Y.B., S.K., and Fat.A. drafted and revised the manuscript. All authors reviewed the final version of the manuscript and take collective responsibility for all its contents.

Ethical Approval

No prior ethical approval is needed for a review-type article.

Data Availability Statement

All data, views, and recommendations generated for this initiative are included in this article and its supplementary material files. All primary literature is cited in its bibliography.

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Conflict of Interest

The authors have no conflicts of interest to declare. None of the authors received any compensation for their contributions to this work. Bayer UAE did not participate in any of the meetings or the drafting of the manuscript.

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