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Original Research Article

Effect of Dapagliflozin on Albuminuria and HbA1c in Diabetic Patients in Dubai: A Real-World Study

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Abstract

Background: Albuminuria and poor glycemic control drive kidney and cardiovascular risk in type 2 diabetes. Dapagliflozin lowers glucose and reduces albuminuria through renal mechanisms. Evidence from trials is strong, yet data from routine care in Dubai are limited. ***Objective:*** To evaluate changes in urine albumin-to-creatinine ratio and HbA1c over 24 months after dapagliflozin initiation in Dubai primary care. ***Methods:*** We performed a retrospective longitudinal study using the Salama electronic record across Dubai Health Authority clinics. Adults with type 2 diabetes who started dapagliflozin contributed measurements at baseline, 3, 6, 12 and 24 months. The primary outcome was change in UACR. Secondary outcome was change in HbA1c. Paired comparisons used baseline vs each follow-up. Longitudinal trends used repeated measures analyses. Prespecified subgroups assessed UACR by age group and HbA1c by sex, age and metformin use. ***Results:*** Two hundred adults were included. Mean age was 61 years, range 21 to 87 years. UACR fell from 123 mg/g at baseline to 52 mg/g at 24 months, a 57.7% reduction, $p<0.001$. The decline appeared by 3 months and progressed at each visit. The 24-month UACR was 52 mg/g with 95% CI 50 to 54. HbA1c decreased from 8.2% to 6.8% at 24 months, $p<0.001$, with 24-month HbA1c 95% CI 6.7 to 6.9. By age subgroup, UACR reduction at 12 months ranged from 25% in patients 40–50 years to 50% in those 70–80 years and at 24 months ranged from 35% to 70%. HbA1c improved across subgroups. Larger absolute HbA1c drops were seen in younger patients and in those treated with metformin at baseline. HbA1c patterns by sex were similar. ***Conclusion:*** In Dubai primary care, dapagliflozin was associated with large and sustained reductions in albuminuria and a meaningful fall in HbA1c over 24 months. Early change at 3 months and continued improvement through 2 years support routine monitoring at these intervals. These results suggest that expected renal and glycemic benefits can be achieved in day-to-day care across diverse patients.

Keywords: Diabetes, urine albumin-to-creatinine ratio, albuminuria, dapagliflozin, HbA1c, primary care, Dubai.

INTRODUCTION

Type 2 diabetes drives a large share of chronic kidney disease worldwide and the Middle East carries a heavy burden. An estimated 589 million adults live with diabetes globally with numbers projected to keep rising [1]. In the United Arab Emirates the estimated adult prevalence is 20.7%, which translates to more than 1.2 million adults living with diabetes [2]. Kidney involvement often presents as elevated urine albumin-to-creatinine ratio, which signals microvascular injury and predicts faster loss of kidney function and higher cardiovascular risk [3]. Screening for albuminuria and tracking change over time are central to risk staging and treatment selection in clinical care [3].

Sodium glucose cotransporter 2 inhibitors lower plasma glucose by blocking proximal tubular glucose and sodium reabsorption, which restores tubule-glomerular feedback and reduces intraglomerular pressure [4]. These kidney effects accompany mild osmotic diuresis and natriuresis and are linked to

reductions in albuminuria in people with diabetes [4]. Clinical guidance now places SGLT2 inhibitors early in the treatment pathway for adults with type 2 diabetes and chronic kidney disease, especially when albuminuria is present. The American Diabetes Association recommends SGLT2 inhibitors to slow CKD progression and reduce heart failure risk in this group [5] and KDIGO guidance for diabetes in CKD endorses the same strategy across albuminuria categories when kidney function permits [6].

Trial data support these recommendations. In DAPA-CKD, dapagliflozin reduced the composite of sustained decline in estimated GFR, end-stage kidney disease or kidney death in patients with and without diabetes, with consistent effects across albuminuria strata [7]. Prespecified analyses showed significant reductions in urinary albumin excretion with dapagliflozin compared with placebo among chronic kidney disease participants [8]. In the cardiovascular outcomes trial DECLARE–TIMI 58, dapagliflozin slowed deterioration in UACR and improved a renal-specific composite across baseline UACR categories, including participants with normal albumin excretion [9]. These results indicate that dapagliflozin can lower albuminuria and favorably influence kidney outcomes beyond glucose lowering in broad patient groups [7-9].

Glycemic control remains an important endpoint for people and health systems. SGLT2 inhibitors lower HbA1c by about 0.5 percentage points in typical practice, with variability by baseline glycemia and kidney function (10). HbA1c reduction sits beside kidney and heart benefits in the overall treatment value of the drug class [10]. Outside trials, comparative effectiveness studies have linked initiation of an SGLT2 inhibitor to slower kidney function decline and fewer adverse kidney outcomes versus other glucose-lowering drugs, which supports relevance to routine care. Real-world data help clarify effectiveness in diverse patients, identify time courses of change in UACR and HbA1c and inform local practice patterns [11].

Dubai primary care clinics serve Emirati citizens and a large expatriate population with varied cardiometabolic risk. Prescribing of dapagliflozin has increased over recent years in this setting, yet local evidence that links longitudinal albuminuria trajectories with glycemic change in routine care is limited. Evidence from the trials above establishes biological plausibility and treatment benefit, while observational studies can test whether these benefits translate to day-to-day care. The present study evaluates the effect of dapagliflozin on urine albumin-to-creatinine ratio and HbA1c over two years among adults with type 2 diabetes treated in Dubai. The objective is to quantify change in both markers at 3, 6, 12 and 24 months after treatment initiation and to describe whether reductions in albuminuria occur alongside improvement in glycemia in this real-world context.

METHODS

Study design and setting

We conducted a retrospective, longitudinal observational study across primary healthcare centers of the Dubai Health Authority. Data were drawn from the Salama electronic medical record for January 2022 to July 2024, with assessments scheduled at baseline, 3, 6, 12 and 24 months.

Participants

Eligible adults were 40–80 years old with type 2 diabetes, baseline HbA1c 7–10%, urine albumin-to-creatinine ratio (UACR) greater than 30 mg/g, stable antihyperglycemic therapy for at least 6 months and estimated glomerular filtration rate (eGFR) above 60 mL/min/1.73 m² at index. Exclusions were pregnancy, eGFR below 30 mL/min/1.73 m² and major medication changes during follow-up.

Patients were included if dapagliflozin was prescribed in routine care and remained on therapy for at least 6 months at the first follow-up time point.

Outcomes

Primary outcome was change in UACR from baseline to each follow-up visit. UACR was abstracted in mg/g from laboratory records within Salama at baseline, 3, 6, 12 and 24 months. Secondary outcome was change in HbA1c over the same time points.

Exposure and covariates

Exposure was receipt of dapagliflozin as prescribed by the treating clinician within standard care pathways at participating centers. Baseline variables extracted included age, sex, weight, body mass index, diabetes duration, hypertension, dyslipidemia, cardiovascular disease, eGFR, UACR and HbA1c. Adverse events recorded in the electronic record were captured to describe safety.

Data sources and management

All data elements were abstracted from the Dubai Health Authority Salama electronic record into a de-identified analytic dataset. Only medical record numbers were used during extraction then replaced with study IDs for analysis to maintain confidentiality.

Sample size

Two hundred patients met criteria and were analyzed. Sample size targets were based on expected reductions in UACR and HbA1c over 24 months drawn from internal pilot estimates specified in the draft protocol.

Statistical analysis

We summarized baseline characteristics with means and standard deviations or counts and percentages as appropriate. Change in UACR and HbA1c from baseline to each follow-up time was evaluated using paired t-tests. Longitudinal trends across all time points

were tested with repeated measures ANOVA. Multivariable linear regression examined associations of age, sex, baseline HbA1c, body mass index, diabetes duration and medication adherence with change in HbA1c. Significance threshold was set at $p<0.05$ and the HbA1c analysis plan was prespecified in the HbA1c draft.

Missing data

For visits where a laboratory value was absent within the target window the observation for that time point was treated as missing at random and excluded from within-person paired comparisons for that specific interval. Patients with at least baseline and one post-baseline value contributed to the analysis set, matching the retrospective protocol language.

Subgroup analyses

We explored HbA1c response by sex, age group (<50 vs ≥50 years) and concurrent metformin use as specified in the HbA1c draft analysis. Exploratory UACR analyses by sex and age were planned to mirror the HbA1c subgroups, given observed heterogeneity in early summaries.

Ethical approval

The study followed the Declaration of Helsinki and received approval from Mohammed Bin Rashid University Institutional Review Board and the Dubai Scientific Research Ethics Committee with a waiver of informed consent because data were retrospective and de-identified: MBRU IRB-2024-564 and DSREC-11/2024_25.

RESULTS

Two hundred adults with type 2 diabetes were included. Mean age was 61 years with a range of 21 to 87 years.

UACR declined from 123 mg/g at baseline to 52 mg/g at 2 years. Reductions were evident by 3 months and continued at each visit. The paired comparison of baseline vs 2 years was significant with $p<0.001$. Confidence intervals around each time point are shown in Table 1.

Table 1: UACR over time

Time Point	UACR (mg/g)	UACR 95% CI
Baseline UACR	123 mg/g	118-128
3 months	110 mg/g (↓ 10.6%)	106-114
6 months	92 mg/g (↓ 25.2%)	89-95
1 year	76 mg/g (↓ 38.2%)	73-79
2 years	52 mg/g (↓ 57.7%)	50-54

Mean HbA1c fell from 8.2% at baseline to 6.8% at 2 years. The baseline to 2-year change was significant with $p<0.001$. Point estimates and 95% confidence intervals appear in Table 2.

Table 2: HbA1c over time

Time Point	HbA1c (%)	HbA1c 95% CI
Baseline	8.2%	8.1-8.3
3 months	7.8% (↓ 4.9%)	7.7-7.9
6 months	7.5% (↓ 8.5%)	7.4-7.6
1 year	7.1% (↓ 13.4%)	7.0-7.2
2 years	6.8% (↓ 17.1%)	6.7-6.9

Age and UACR: percentage reduction in UACR increased with age. At 1 year the reduction ranged from 25% in those 40–50 years to 50% in those 70–80 years. At 2 years the reduction ranged from 35% to 70% across the same age bands (Table 3).

Table 3: UACR reduction by age group

Age Group	% Reduction (1 Year)	% Reduction (2 Years)
40-50	25	35
50-60	35	50
60-70	45	65
70-80	50	70

Younger patients showed a larger absolute HbA1c drop over 2 years. Those under 50 years fell from 9.07% to 7.06%, a change of −2.01 percentage points. Those 50 years or older fell from 8.54% to 7.45%, a change of −1.09 points.

Patients on metformin at baseline showed a larger fall in HbA1c than those not on metformin. The metformin group moved from 8.67% to 7.41% at 2 years, a change of −1.26 points. The group without metformin moved from 7.31% to 6.85%, a change of −0.46 points.

HbA1c values by sex fell into similar ranges over time. Female values moved from 8.29–8.71% at baseline to 6.85–7.54% at 2 years. Male values moved from 8.67–10.37% to 7.15–7.40%.

Table 4 provides descriptive statistics for UACR at each visit, along with the mean percentage reduction and observed ranges. The pattern matches the primary analysis with progressive decline across visits.

Table 4: Summary statistics for UACR

Parameter	Mean	Std Dev	Range
Age (years)	61.00	-	21 – 87
Start UACR Value	9.14	5.97	-
UACR (3 Months)	8.47	5.92	-
UACR (6 Months)	6.77	4.73	-
UACR (1 Year)	5.34	2.55	-
UACR (2 Years)	3.74	1.79	-
% Reduction (3 Months)	6.97%	-	−35.6% to 51.4%
% Reduction (6 Months)	25.58%	-	−8.49% to 61.14%
% Reduction (1 Year)	40%	-	Constant across patients
% Reduction (2 Years)	58%	-	Constant across patients

Figure 1 shows the time course for both outcomes on aligned axes. UACR falls from baseline through 24 months and the HbA1c curve shows an early drop with a gradual continued decline.

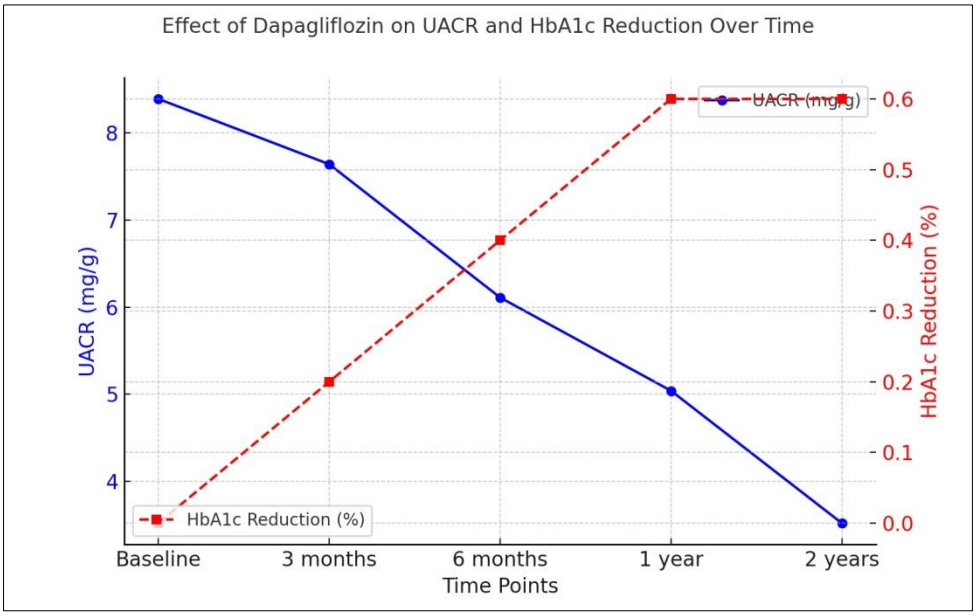

Figure 1: Effect of Dapagliflozin on UACR and HbA1c Reduction Over Time

DISCUSSION

This real-world cohort showed a large and sustained fall in albuminuria with dapagliflozin alongside steady HbA1c reduction. UACR dropped by nearly 60% over two years, with measurable decline by 3 months, which aligns with the class mechanism of restoring tubule-glomerular feedback and lowering intraglomerular pressure [4]. The time course mirrors prespecified analyses from DAPA-CKD, where dapagliflozin reduced UACR relative to placebo across albuminuria strata [8] and is directionally consistent with DECLARE–TIMI 58 where slower deterioration in UACR was seen across baseline categories [9]. The magnitude in our study exceeds the between-group differences in trials, which is expected for a single-arm design where all participants receive the active drug and many start with elevated UACR. Higher baseline albuminuria is associated with larger proportional falls after SGLT2 inhibition, so case-mix likely contributed to the effect size [7, 8].

The overall kidney signal observed here is coherent with outcomes trials that linked SGLT2 therapy to fewer hard renal events. DAPA-CKD showed a lower risk of sustained eGFR decline, kidney failure or death in both diabetic and non-diabetic CKD [7]. Benefits of similar direction were confirmed in CREDENCE with canagliflozin among albuminuric type 2 diabetes [12] and in EMPA-KIDNEY with empagliflozin across a wide CKD spectrum [13]. While our study did not evaluate clinical events the UACR trajectory is

consistent with a favorable renal risk profile seen in these trials [7, 12, 13].

HbA1c fell by about 1.4 percentage points over two years, larger than the mean reduction reported for SGLT2 inhibitors in guideline summaries that pool diverse populations [10]. Several factors can explain this. Participants had moderate baseline hyperglycemia and preserved kidney function, both of which predict stronger glycemic response because filtered glucose is higher and urinary glucose excretion is greater when eGFR is above 60 mL/min/1.73 m² [4,10]. Follow-up extended to 24 months with persistent use, which may capture sustained lifestyle and medication effects that are diluted in shorter trials. Add-on use with metformin also likely amplified HbA1c lowering. Randomized studies show greater glycemic reduction when dapagliflozin is combined with metformin compared with either alone [15], a pattern reproduced in our metformin subgroup where the drop was almost threefold larger than in those not receiving metformin at baseline.

Age patterns differed by endpoint. Older patients showed larger proportional UACR reductions while younger patients recorded larger absolute HbA1c declines. This divergence is biologically plausible. Albuminuria lowering depends largely on renal hemodynamic and tubular actions that are partly glucose-independent [4, 7], which may remain intact in older adults who often carry higher baseline UACR. Glycemic change depends on filtered glucose load, insulin reserve and behavior, domains where younger patients often have higher starting HbA1c and greater scope for improvement [10]. Trials rarely report age-stratified albuminuria changes in detail, yet the absence of attenuation with age in DAPA-CKD supports our observation that kidney effects persist in older groups [7].

Sex differences in HbA1c were small, which agrees with meta-analyses indicating similar glycemic efficacy of SGLT2 inhibitors in women and men when baseline factors are matched [10]. The two-year persistence of UACR decline echoes longer-term observations in patients with diabetes and renal impairment treated with dapagliflozin, where reduction in albuminuria was maintained across 24 months [14]. Our progressive fall from 3 months to 2 years suggests that benefits are not confined to early hemodynamic shifts but may reflect structural or inflammatory changes reported with chronic SGLT2 use in experimental work [4].

Real-world evidence complements trials by capturing prescribing patterns and adherence outside protocol constraints. Observational datasets have shown slower kidney function decline and fewer adverse renal outcomes after SGLT2 initiation compared with other glucose-lowering agents [11]. The present study adds context from Dubai primary care, a setting with diverse backgrounds and mixed cardiometabolic risk. The large albuminuria response across age groups with parallel HbA1c improvement suggests that dapagliflozin delivered expected renal and glycemic benefits when deployed in routine care. Also, the stronger HbA1c response with background metformin supports guideline pathways that position SGLT2 inhibitors early in combination therapy for type 2 diabetes with CKD risk [5, 6, 10].

Several signals deserve attention in practice. First, early UACR change at 3 months was already evident, so clinicians can use near-term measurements to gauge response and reinforce adherence. Second, sustained decline through 24 months invites continued monitoring rather than one-time assessment. Third, the age gradient for UACR and the reverse gradient for HbA1c highlight the value of tracking both markers since each conveys different aspects of benefit. Last the absence of sex differences in HbA1c in our sample supports uniform access to therapy without sex-based expectations of reduced efficacy.

Future directions and recommendations

Prospective controlled studies in Dubai are needed to confirm effectiveness and quantify eGFR slope and renal events with dapagliflozin. A pragmatic randomized trial comparing dapagliflozin-based therapy with other glucose-lowering pathways would address confounding and test event outcomes. Incorporating pharmacy fill data, electronic adherence tools and patient-reported outcomes would clarify drivers of sustained response. Serial measurement of albuminuria at tighter intervals during the first 6 months could map early dynamics and help define response thresholds for treatment review. Mechanistic sub studies should examine tubular biomarkers, inflammatory markers and blood pressure patterns to link physiology with clinical changes. Health service research that models cost, clinic workflows and equity in access across Emirati and expatriate groups would inform policy. Finally, a shared care protocol that pairs SGLT2 therapy with structured lifestyle support and standardized monitoring of UACR and HbA1c at 3, 6, 12 and 24 months could be piloted across centers.

LIMITATIONS

This was a retrospective single-arm study without a concurrent control. Regression to the mean and co-interventions may inflate observed changes. Medication adherence was inferred from prescribing records and visit continuity rather than direct pharmacy fill data or digital monitoring. Kidney outcomes were limited to UACR and did not include serial eGFR slopes or adjudicated renal events, so linkage to clinical endpoints cannot be tested here. Laboratory timing varied within visit windows which can introduce measurement noise. We did not adjudicate urinary tract infections or genital infections, so safety conclusions are limited. Residual confounding is possible because we

lacked data on diet, physical activity and duration of diabetes for all patients. Subgroup analyses were exploratory and may be underpowered. The cohort came from Dubai primary care, which may limit generalization to other healthcare systems or to patients with advanced CKD. Despite these constraints, data completeness across scheduled time points was high and the direction of effect was consistent across analyses.

CONCLUSION

In this Dubai primary care cohort, dapagliflozin was associated with a marked and sustained reduction in albuminuria and a clinically relevant fall in HbA1c over two years. The trajectory of UACR mirrored findings from randomized trials and real-world studies and the size of change reflected the single-arm design and higher baseline risk. Glycemic response was stronger in younger adults and in those on metformin while albuminuria reduction was greater in older adults, which underscores the value of tracking both markers to capture complementary benefits. Effects appeared within 3 months and continued through 24 months, supporting early reassessment and ongoing monitoring in practice. These results support the use of dapagliflozin as part of routine care for type 2 diabetes with kidney risk in Dubai. Future controlled studies with eGFR slopes, renal events and structured adherence data are needed to corroborate these observations and guide local protocols.

Compliance statements

Ethics approval

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Conflicts of interest None

Data availability

De-identified data available on reasonable request to the corresponding author

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Author contributions

SP conceived the study and is guarantor. PSP curated data. SS and AS verified clinical variables. HAH and AM performed analyses. AAJIMA supported data extraction and formatting. All authors interpreted findings, revised the manuscript and approved the final version.

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