Al-Rafidain J Med Sci. 2026;10(1):113-116.
DOI: https://doi.org/10.54133/ajms.v10i2.2814

AJMS

Online ISSN (3219-2789)

Research Article

Metabolic–Renal Divergence in Diabetic Patients Three Months After COVID-19: Evidence from a Real-World Cohort

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Received: 30 January 2026; Revised: 25 March 2026; Accepted: 30 March 2026

Abstract

Background: The short-term impacts of COVID-19 in diabetes are still unknown as well. When glycemic improvement is followed by renal stabilization in a patient after treatment of COVID-19 infection. ***Objective***: To evaluate three-month glycemic and renal trajectories following COVID-19 infection and to identify predictors of persistent poor glycemic control. ***Methods***: This retrospective real-world cohort study included 301 adults with diabetes and confirmed COVID-19 infection. Baseline measurements were obtained at the first post-COVID clinical assessment, and follow-up measurements were recorded approximately three months later. Paired t-tests assessed within-subject changes in HbA1c, fasting plasma glucose (FPG), and serum creatinine. McNemar's test evaluated shifts in glycemic control category (HbA1c<8%). Multivariable linear and logistic regression analyses identified predictors of glycemic change and persistent poor control. Effect sizes were calculated using Cohen's d. ***Results***: HbA1c was significantly decreased (9.08±2.22% to 8.43±2.04%. FPG was reduced (51mg/dL; p<0.001). The creatinine level remained similarly in line but slightly rose in diabetic patients at 3 months of chronic comorbidity (+ 0.094mg/dL; p=0.001), an indication of metabolic-renal divergence. Significant improvement of the glycemic control category was observed as well (p<0.001). ***Conclusion***: Three months after COVID-19 treatment, there was a positive correlation between glycemic control and glucose decline, along with a reasonable renal outcome. This clearly illustrates an acute need for early cardiometabolic care after infection.

Keywords: COVID-19; Cohort study; Diabetes mellitus; Glycemic control; Renal function; Therapeutic inertia.

التباعد الأيضي والكلوي لدى مرضى السكري بعد ثلاثة أشهر من الأصابة بكوفيد-19: أدلة من دراسة جماعية واقعية

الخلاصة

الخلفية: لا تزال التأثيرات الأيضية والكلوية قصيرة الأمد لكوفيد-19 لدى مرضى السكري غير موصوفة بشكل كافٍ. ومدى توازي تحسن السيطرة السكرية مع استقرار الوظيفة الكلوية بعد العدوى الحادة ما يزال غير واضح. **الهدف**: تقييم المسارات السكرية والكلوية خلال ثلاثة أشهر بعد الإصابة بكوفيد-19، وتحديد العوامل المتنبئة باستمرار سوء السيطرة على سكر الدم. **الطرائق**: أُجريت دراسة أترابية استعادية قائمة على بيانات واقعية شملت 301 مريضًا بالغًا مصابًا بالسكري ومؤكد إصابتهم بكوفيد-19. تم الحصول على القياسات الأساسية عند أول تقييم سريري بعد التعافي من كوفيد-19، وسُجلت قياسات المتابعة بعد نحو ثلاثة أشهر. تم تقييم التغيرات في HbA1c وسكر الدم الصائم والكرياتينين المصلي. كما استُخدم اختبار McNemar لتقييم التحول في فئات السيطرة السكرية (HbA1c< 8%). وأُجريت تحليلات انحدار خطي ولوجستي متعدد المتغيرات لتحديد العوامل المتنبئة بالتغير في سكر الدم واستمرار سوء السيطرة. وتم حساب حجم الأثر باستخدام معامل كوهين. **النتائج**: انخفض متوسط HbA1c بشكل ملحوظ من 9.08 إلى 8.43 كما انخفض سكر الدم الصائم بمقدار −51 ملغم/دل؛ وارتفع الكرياتينين المصلي ارتفاعًا طفيفًا (+0.094 ملغم/دل)؛ مما يشير إلى وجود تباين أيضي–كلوي. ولوحظ تحسن معنوي في فئة السيطرة السكرية .وفي تحليل الانحدار اللوجستي متعدد المتغيرات، ارتبط ارتفاع ضغط الدم بشكل مستقل بانخفاض احتمال استمرار سوء السيطرة السكرية بعد ثلاثة أشهر. **الاستنتاجات**: أظهر مرضى السكري بعد ثلاثة أشهر من الإصابة بكوفيد-19 تحسنًا ملحوظًا في السيطرة السكرية، ترافق مع تدهور كلوي طفيف، مما يشير إلى وجود تباين أيضي–كلوي قصير الأمد. ويؤكد استمرار سوء السيطرة لدى نسبة ملحوظة من المرضى أهمية تبني استراتيجيات متابعة تكاملية واستباقية لإدارة المخاطر القلبية الأيضية بعد الإصابة بكوفيد-19.

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Article citation: Majeed SH. Metabolic–Renal Divergence in Diabetic Patients Three Months After COVID-19: Evidence from a Real-World Cohort. *Al-Rafidain J Med Sci*. 2026;10(1):113-116. doi: https://doi.org/10.54133/ajms.v10i2.2814

INTRODUCTION

As global morbidity and mortality depend on diabetes mellitus, both microvascular and macrovascular morbidity are still associated with diabetes mellitus. The COVID-19 pandemic included systemic inflammation, corticosteroid exposure, healthcare disruption, and reduced care access [1]. There is recent evidence that COVID-19 could further cause diabetes to fail to be cured by glycemic instability, but longitudinal measurements of metabolic and renal trajectory are still lacking [2-4]. The inertia of the therapy (failure to achieve treatment response when glycemic targets are not met or failure to respond) in diabetes management is still prevalent. After the virus is no longer infectious, there might be more complications in treatment coordination because of its infection that led to worsened glycemic control, which could result in increased difficulty in managing diabetes effectively and addressing related health issues [5,6]. Importantly, renal involvement has been reported in

diabetic patients following COVID-19 infection in prior years. It is uncertain whether glycemic parameter improvements are faster than renal parameter improvements in diabetic patients. If metabolic and renal trends are divergent, how to treat diabetes with less blood sugar or, in particular, diabetes with a more accurate diagnosis will be important [7-9] in the future. In this respect, we proposed to estimate glycemic parameter improvements and renal marker changes in the short duration (3 months) following COVID-19 infection and, therefore, to search for signals of chronic dysregulation.

METHODS

Study design and setting

The retrospective real-world cohort study was performed at a major diabetes center in Baghdad, Iraq. The records of patients with COVID-19 from January 2020 to December 2021 were taken and simultaneously evaluated as patients with COVID-19 diagnosed, and 301 patient records were analyzed for inclusion criteria. Baseline laboratory measurements were obtained during routine follow-up after COVID-19 infection. No exact timing between diagnosis and baseline assessment was given, but it seems that these measurements would be a reliable indication of the recovery phase, such as post-acute recovery. However, the lack of the exact timing from COVID-19 diagnosis and baseline assessment can generate temporal variability. Figure 1 shows the patient selection and inclusion process.

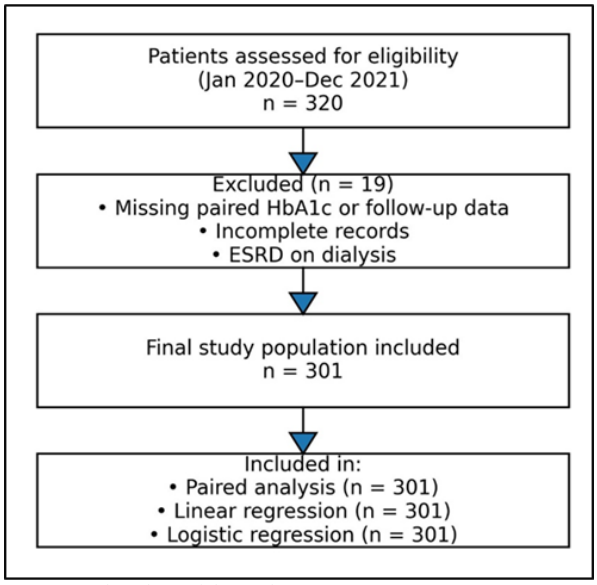

Figure 1: Flowchart of the study.

Baseline and follow-up data

We defined baseline measurements as the first data (medicinal and laboratory) that were taken after the COVID-19 diagnosis in diabetes follow-up at the local diabetes center. Follow-up measurements were made with laboratory results approximately three months (±2 weeks) after baseline. Although all of the patients were gathered over a period of one year (2020–2021), each patient contributed paired measurements spanning a three-month post-COVID interval in the literature. We considered glycemic control based on current guidelines around diabetes treatment [10]. Each patient is recorded on body mass index (BMI), hypertension, use of statins (ACEI/ARBs), and treatment group (no medicine, oral hypoglycemic agent, insulin, or combination therapy).

Inclusion criteria

Adults (≥18 years of age) with known diabetes mellitus and documented COVID-19 infection were allowed, provided the baseline and three-month interval measurements for HbA1c, fasting plasma glucose (FPG), and serum creatinine were available.

Exclusion criteria

As a consequence, those patients were eliminated if they had end-stage kidney disease requiring dialysis at baseline, were missing paired outcome data or pregnancy during the trial period, or were missing key clinical records for regression adjustment.

Outcome measurements

HbA1c (%) at baseline (HbA1c1) and 3 months follow-up (HbA1c2). Fasting plasma glucose (FPG, mg/dL) at baseline (FPG1) and follow up (FPG2). Serum creatinine (mg/dL) at baseline (Creatinine1) and follow-up (Creatinine2). ΔHbA1c = HbA1c2 − HbA1c1. ΔFPG = FPG2 - FPG1. $\Delta\Delta$creatinine = Creatinine2 - Creatinine1. Glycemic control in both cases was determined as controlled (HbA1c < 8%) or uncontrolled (HbA1c ≥ 8%). The regression model used baseline HbA1c as an important covariate because it is widely used to predict glycemic change. The creatinine was initially recorded in samples as µmol/L but then converted to mg/dL to monitor the renal function of the skin of the man. Renal function was further evaluated using estimated glomerular filtration rate (eGFR), calculated using the CKD-EPI equation.

Ethical considerations

This retrospective study was conducted on anonymized clinical record data. Ethical approval was issued from the Institutional Research Ethics Committee of Al-Nahrain University–College of Pharmacy, Baghdad, Iraq. We were able to design and use the de-identified data without written informed consent from the ethics committee.

Statistical analysis

Continuous variables were indicated as mean ± standard deviation (SD) and categorical factors as frequency and percentage. On the condition of complete case data (n=301). We measured paired differences using Shapiro-Wilk tests. To test the relative magnitude and reliability

of these differences, we used paired t-tests to examine the differences between HbA1c, FPG, and serum creatinine within subjects. The effect sizes were calculated for paired samples using Cohen's d. Changes in glycemic control category were evaluated using McNemar's test. One-way ANOVA was conducted to compare follow-up HbA1c of the groups. We used Levene's test to assess its homogeneity. The size of the effect that follows is η^2. Different data were analyzed from linear regression by using ΔHbA1c as the dependent. The covariates were baseline HbA1c, BMI, hypertension, statin use, and ACEI. VIF (variables are inflation factors) was used for multi-reliance regression to measure multicollinearity. The fit for the model was obtained with adjusted R^2. We applied binary regression for predictors of continued poor glycemic control for 3 months, at which high HbA1c was predicted. Adjusted odds ratios (aOR) were presented for all three parameters and 95% confidence intervals (CI) of the two-thirds and 90% estimated levels of treatment (CI-P&C). We also undertook simulation of the model using the Hosmer-Lemeshow test on different instruments and found good agreement. All statistical tests were two-sided, with $p < 0.05$ being considered statistically significant. SPSS version 26 (IBM Corp., Armonk, NY, USA).

RESULTS

The clinical characteristics were examined in a three-month follow-up study in which 301 patients fulfilled our eligibility criteria and were included in the study. At both baseline and follow-up, all participants had paired laboratory observations. Baseline clinical characteristics are summarized in Table 1. HbA1c was 9.08 ± 2.22% and at follow-up in this case 8.43 ± 2.04%, a mean value of Δ 95% CI= 0.49 to -0.82; t= 7.81 and Cohen's d= 0.45.

Table 1: Baseline clinical characteristics and laboratory parameters of the study population (n=301)

Variable	Value
Demographic characteristics	
Age (year)	57.60±10.96
Male sex	157(52.2)
BMI (kg/m²)	30.19±5.44
Comorbidities and medications	
Hypertension	59(19.6)
Statin use	81(26.9)
ACE inhibitor use	17(5.6)
ARB use	8(2.7)
Treatment Distribution	
No medication	5(1.7)
OHA only	212(70.4)
Insulin only	24(8.0)
OHA + Insulin	59(19.6)
Laboratory parameters	
HbA1c at baseline	9.08±2.22
HbA1c at 3 months	8.43±2.04
FPG at baseline (mg/dL)	211.98±84.84
FPG at 3 months (mg/dL)	160.96±56.86
Serum creatinine at baseline (mg/dL)	0.62±0.43
Serum creatinine at 3 months (mg/dL)	0.72±0.45

Data are presented as mean±SD or number (percentage). OHA: oral hypoglycemic agents; ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; FPG: fasting plasma glucose.

In contrast, serum creatinine increased slightly from 0.62 ± 0.43 mg to 0.72 ± 0.45 mg/dL (Δ +0.094 ± 0.176 mg/dL; t= -9.23; $p<0.001$; Cohen's d= 0.53). In Table 2, there were significant decreases in HbA1c and FPG and hence a relatively large effect size (Cohen's d= 0.45 to 0.73), whereas in serum creatinine it was the other extreme. In eGFR, eGFR decreased slightly, from 92.4 ± 18.6 to 87.1 ± 17.9 mL/min/1.73 m² ($p< 0.001$), suggesting a statistically significant though clinically low impact.

Table 2: Paired changes in metabolic and renal parameters over three months (n=301)

Parameter	Baseline	After 3 months	Mean Difference	95% CI	*p*-value	Cohen's d
HbA1c (%)	9.08±2.22	8.43±2.04	−0.65±1.45	−0.82 to −0.49	<0.001	0.45
FPG (mg/dL)	211.98±84.84	160.96±56.86	−51.02±70.11	−58.96 to −43.08	<0.001	0.73
Creatinine (mg/dL)	0.7±0.49	0.81±0.51	0.11±0.2	0.09 to 0.13	<0.001	0.53
eGFR (mL/min/1.73m²)	92.4±18.6	87.1±17.9	−5.3±9.2	−6.4 to −4.2	<0.001	0.57

Values represent mean±SD. Differences were assessed using paired t-tests. CI: confidence interval; SD: standard deviation. Values are presented as mean ± SD. Differences were assessed using paired t-tests. Serum creatinine values were originally recorded in µmol/L and converted to mg/dL for analysis. eGFR was estimated according to the CKD-EPI equation. CI: confidence interval; SD: standard deviation.

Table 3 shows significant change in glycemic control category (McNemar χ^2= 18.97, $p<0.001$) where 79 patients are now in control, whereas others have changed their status from uncontrolled to controlled or vice versa.

Table 3: Shift in glycemic control category (HbA1c < 8%)

	Follow-up Controlled	Follow-up Uncontrolled	Total
Baseline Controlled	72	18	90
Baseline Uncontrolled	79	132	211
Total	151	150	301

McNemar χ^2 = 18.97, $p< 0.001$. Controlled defined as HbA1c < 8%. McNemar's test was used to evaluate within-subject category shifts.

A large proportion of previously uncontrolled patients are achieving HbA1c of less than 8 percentage points post-interventions. As shown in Table 4, the effect size with subsequent treatment was very small (η^2 = 0.040), indicating no meaningful variation in HbA1c between groups after interventions. HbA1c from baseline was a clear independent predictor of glycemic control with a larger decrease from baseline (β= −0.58, $p< 0.001$). Hypertension was independently associated with a lower likelihood of persistent poor glycemic control for the linear regression case (adjusted OR: 0.214-95%; CI: 0.079–0.579; $p= 0.002$).

Table 4: One-Way ANOVA – Follow-up HbA1c according to treatment group

Source	df	Mean Square	F	*p*-value
Treatment group	3	32.92	4.12	0.007
Residual	297	7.97	—	—

Effect size: $\eta^2 = 0.040$. One-way ANOVA was used. η^2 represents eta squared effect size. Treatment groups were coded as: 0 = no medication; 1 = OHA; 2 = insulin; 3 = OHA + insulin.

In the linear regression model (Table 5), none of the covariates predicted the value of ΔHbA1c independently. The model was moderately explanatory ($R^2 = 0.194$), and the logistic model was modestly explanatory (Nagelkerke $R^2 = 0.044$), as shown in Table 6.

Table 5: Multiple linear regression – Predictors of ΔHbA1c. Outcome: ΔHbA1c (HbA1c2 − HbA1c1)

Predictor	β	95% CI	*p*-value
Intercept	−1.422	−2.416 to −0.428	0.005
Baseline HbA1c (%)	−0.58	−0.72 to −0.44	<0.001
BMI	0.025	−0.008 to 0.058	0.139
Hypertension	−0.086	−0.734 to 0.562	0.794
Statin use	0.028	−0.386 to 0.442	0.894
ACEI use	0.040	−0.053 to 0.132	0.397
ARB use	−0.260	−1.521 to 1.001	0.685

β: unstandardized regression coefficient. Model adjusted $R^2 = 0.194$, indicating that 19.4% of the variance in ΔHbA1c was explained by the included covariates. β represents unstandardized regression coefficients. Model assumptions were verified and multicollinearity was assessed using variance inflation factors (VIF). VIF values were < 2, indicating no multicollinearity.

Table 6: Logistic regression analysis for predictors of poor persistent glycemic control at three months (HbA1c ≥ 8%)

Predictor	Adjusted OR	95% CI	*p*-value
Intercept	0.894	0.226 – 3.538	0.873
BMI	1.017	0.971 – 1.064	0.484
Hypertension	0.214	0.079 – 0.579	0.002
Statin use	1.069	0.600 – 1.905	0.821
ACEI use	1.089	0.957 – 1.240	0.198
ARB use	2.291	0.387 – 13.551	0.361

OR: odds ratio; CI: confidence interval. Model calibration was evaluated using the Hosmer–Lemeshow test. Nagelkerke $R^2 = 0.044$. Model calibration was assessed using the Hosmer–Lemeshow test.

DISCUSSION

This real-world cohort study of 301 diabetic patients who became infected with COVID-19 demonstrated significant improvements in glycemic parameters over three months following diagnosis of the disease. In contrast, renal indices showed a modest but statistically significant increase, indicating an imbalance when they come to terms with metabolism and renal function as a mechanism of impairment. During the study period of 3 months, mean creatinine was statistically significant in both creatinine per cell and eGFR, but it has not deteriorated from there, and the renal function changes are small and within healthy ranges [11]. These results align with recent evidence showing that post-COVID renal change is mild and reversible in non-critical populations. The lack of precise timing between COVID-19 diagnosis and baseline assessment may introduce temporal variability, which should be considered when interpreting the findings. The low HbA1c (−0.65%) and fasting plasma glucose (−51 mg/dL) results indicate that our body has stabilized. Moderate to large effect sizes are indicative of the clinical relevance of what it has been effective for. Despite that high mean BMI (30.19 kg/m²), a significant part of the patients were poorly controlled by the drugs. There is still significant chronic obesity and disease resistance to treatment at the diabetes management level [5,12]. In a multivariable linear regression, no independent predictors significantly contributed to the ΔHbA1c magnitude of data in a very smooth distribution. This was expected to take about 19.4% of glycemic change out of the total difference due to those covariates (adjusted $R^2 = 0.194$). This would suggest that in general, long-term metabolic effects post-COVID-19 are controlled by unspecific metabolic aspects beyond typical clinical and behavioral factors as described earlier [13,14]. We found that based on binary logistic regression (0.044), and in reality, in real life for epidemiology, it is not true. The nominal pseudo-R^2 in our models is quite low and does not suggest poor predictive power but indicates the relevance and legitimacy. Despite limited variance in the model, hypertension is found to be an independent factor of the persistently poor glycemic control rate. This could be linked to an issue relating to the engagement in healthcare, cardiovascular risk monitoring, and drug severity in hypertensive patients over time. A few other reasons for this include: First, hypertensive patients frequently receive more extensive healthcare and more care for cardiovascular risk factors. By better attaining compliance with medicines, glycemic outcomes may be enhanced. Additionally, many hypertensive patients take ACE inhibitors or ARBs, which have been believed to have positive metabolic or renal hemodynamic effects under consideration. Third, increased physician vigilance in patients with multiple comorbidities may help reduce inertia to therapeutic treatment and enhance efficiency [11]. This was in agreement with the perspective that high-health-care intensity and organized follow-up can prevent poor glycemic findings in subgroups of cardiometabolic patients. It reinforces the need for integrated, overall cardiometabolic therapy rather than different types of glycemic care by targeting distinct individuals. Multiple linear regression that took into account different groups' ΔHbA1c was able to find no significant difference in the rate of change (e.g., overall gain of glucose, with a more uniform profile at the end of the acute post-COVID study instead of focusing on some individuals' responses). Due to data limitations, treatment categories were classified into broad groups and may take into account some heterogeneity of modern antidiabetic drugs, which could influence the understanding of the treatment effects [13–14]. All of this led to a greater metabolic-renal gradient with the increase in serum creatinine but also increased renal vulnerability in glucose patients as well as in high-grade blood pressure patients with COVID-19, and the renal responsiveness was not adequately assessed or documented when glucose was not raised. COVID-19-

related endothelial failure, inflammatory activity, and hemodynamic changes could give rise to subtle renal vulnerability even if glycemic rates start to improve in diabetic patients [15] for their continued follow-ups. Glycemic surveillance should be on track and monitored as per the hypertension/diabetes patients' health status, and one should monitor both. RAAS blockades may have indirect metabolic effects that can influence glycemic progression, potentially leading to improved outcomes in diabetic patients who are also managing hypertension [7-9,16]. This finding should be interpreted with caution, as it may indicate disparities in healthcare utilization rather than a genuine protective effect of hypertension. Our findings also support evidence of residual renal vulnerability after COVID-19, with a systematic assessment showing that kidney function remained impaired even when metabolic improvement was observed. The study showed statistical significance, but changes in renal parameters were relatively small, suggesting limited clinical impact. This is indicative that we should not lose sight of such findings and that observational studies need to be interpreted more realistically [17,18]. In addition, we have also demonstrated systematic clinical pharmacy pharmacotherapy of diabetes in real-world settings to lower systemic inertia and enhance diabetes outcomes [19,20]. This fact deserves care, especially because it could simply reflect health disparities in healthcare utilization as well as a rather lower protective effect of hypertension. From a health pharmacy point of view, our findings confirm effective drug use of structured pharmacotherapy via structured drug inventory and risk stratification, and enhanced therapy is needed. The difference in fasting glucose improvement and persistent elevation in HbA1c could indicate the underlying glycemic variability or postprandial hyperglycemia not captured by single-point examinations. The improvement in glycemic control also supported findings from recent post-COVID studies suggesting that increased clinical attention, treatment intensity, and patient symptom changes after infection might all promote improved glycemic control. However, the persistence of poor control in a significant number of patients indicates long-standing therapeutic hurdles in the treatment delivery, which may include factors such as inadequate patient education, limited access to healthcare resources, and the need for more personalized treatment plans. With these important results, there may need to be integrated post-COVID follow-up measures that address glycemic control and renal monitoring. Early therapeutic optimization and continued renal screening and surveillance may be critical, even for patients who are at higher risk for an increased glycemic response in the future.

Study Limitations

This study has a few limitations: first, the retrospective single-center design is not widely applicable to other diabetic populations. Second, the follow-up period is three months, which can only provide an overall general picture of metabolic pathways and renal functions in patients with COVID-19. Third, although the renal function of the patients depends on both serum creatinine and estimated glomerular filtration rate, it comes under group-level assumptions as available data for individuals is not. Finally, clinical outcomes such as treatment initiation, duration of diabetes, compliance with medication, and other lifestyle measures are less accurately observed and hence could not be directly attributed to how effective treatment is. Finally, any residual confounding even in the multivariable model cannot be removed, particularly given the modest explanatory power that the logistic model has.

Conclusion

In this real-world setting on diabetic adults, glycemic improvement was achieved after COVID-19 infection over three months of treatment (medicine-induced changes were significant, and the eGFR change was clinically insignificant). These findings suggest that early post-COVID metabolic stabilization does not result in a successful renal recovery a few months post-infectious. This result may imply that there are remaining therapeutic gaps and/or the early maintenance of the glucose profile on their kidney that can cause delay in treatment escalation and renal monitoring in patients who recovered from COVID. Structured and integrated cardiometabolic follow-up strategies are needed while monitoring glucose levels for diabetic patients recovering from COVID-19.

ACKNOWLEDGMENTS

The author thanks the staff of the Diabetes Center for their support in helping data access and record retrieval.

Conflict of interests

The authors declared no conflict of interest.

Funding source

The authors did not receive any source of funds.

Data sharing statement

The data that support the findings of this study are available from the corresponding author upon reasonable request, subject to ethical approval and institutional regulations

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