



Article

Early Post-Transplant Glycemic Variability and Its Association with Post-Transplant Dysglycemia After Kidney Transplantation

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Abstract

Background: Glycemic variability (GV) may play an important role in metabolic outcomes after kidney transplantation (KT). This study aimed to evaluate the association between early post-transplant GV, assessed by continuous glucose monitoring (CGM), and the subsequent development of post-transplant dysglycemia. **Methods:** This prospective exploratory pilot study included adult recipients of primary deceased-donor KT at Martin University Hospital, Slovakia. The Dexcom G6 CGM system was applied immediately before KT and continuously measured interstitial glucose for seven days after KT. Demographic, clinical, and biochemical data were collected on day 0, and 7, and at months 3, 12. **Results:** Twenty patients completed the 12-month follow-up. PTDM/prediabetes developed in 10 (50%) and had higher HbA1c on day 7 ($p = 0.038$) and lower postprandial insulin ($p = 0.042$). At 3 and 12 months, HbA1c ($p = 0.006$), fasting insulin ($p = 0.0219$), and HOMA-IR ($p = 0.006$) were significantly elevated in this group. CGM demonstrated higher mean glucose ($p = 0.021$), SD ($p = 0.001$), and estimated HbA1c ($p = 0.017$), with longer time above range ($p = 0.042$) and shorter time in range ($p = 0.037$). In exploratory regression analyses, $CV \geq 36\%$ (aOR 7.31, $p = 0.049$) and TAR (aOR 1.35, $p = 0.045$) were associated with subsequent PTDM/prediabetes. **Conclusions:** In this prospective exploratory pilot study, higher early postoperative GV was associated with subsequent post-transplant dysglycemia. These findings suggest that CGM-derived metrics may help identify patients at higher metabolic risk after KT; however, given the small sample size and exploratory design, the results should be interpreted as hypothesis-generating.



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Keywords: glycemic variability; continuous glucose monitoring; deceased-donor kidney transplantation; post-transplant diabetes mellitus

1. Introduction

Over the past decade, improving long-term patient survival and quality of life has become one of the major challenges in KT [1]. Despite substantial advances in immuno-suppressive therapy and post-transplant care, metabolic complications, cardiovascular diseases, and infections remain common and continue to adversely affect long-term outcomes [2]. Post-transplant diabetes mellitus (PTDM) and prediabetes develop in more than

one-third of KT recipients, both representing significant risk factors for graft and patient survival [3,4]. In addition, transient perioperative hyperglycemia occurs in up to 80% of recipients, irrespective of pre-transplant glycemic status, and has been associated with an increased risk of subsequent PTDM [5,6]. Consequently, PTDM is now recognized as one of the most important metabolic complications after KT and a major contributor to cardiovascular morbidity and mortality [7,8].

Glycemic variability reflects dynamic fluctuations in blood glucose concentrations, including both hypoglycemic episodes and postprandial hyperglycemic excursions, thereby providing a more comprehensive assessment of glucose homeostasis than average glycemia alone. In clinical practice, GV can be assessed over the short term (inter- and intraday variability) or over longer periods extending from weeks to months [9]. Increasing evidence suggests that GV contributes to the development of both microvascular and macrovascular complications in diabetes mellitus (DM) and may be associated with adverse cardiovascular outcomes in individuals without DM [10]. Traditionally, glycemic control has been assessed using glycated hemoglobin (HbA1c) and self-monitoring of blood glucose. However, HbA1c reflects average glycemia over several weeks and does not capture short-term glucose excursions or GV, particularly during the dynamic early post-transplant period [11].

Perioperative hyperglycemia after KT is largely driven by the initiation of diabetogenic immunosuppressive therapy combined with surgical stress. Given the high efficacy of immunosuppression in preventing graft rejection, strategies aimed at reducing long-term metabolic complications should focus on the early identification of patients at increased metabolic risk. Recent studies using CGM in KT recipients have suggested not only more precise monitoring of GV and the potential for timely intervention but also non-invasiveness and a possible predictive role for PTDM development [12]. However, most available studies have included recipients of living-donor KT, and data in deceased-donor recipients remain limited. Therefore, the aim of this prospective exploratory pilot study was to investigate the association between perioperative GV assessed by CGM and the subsequent development of PTDM and prediabetes in deceased-donor KT recipients.

2. Materials and Methods

This prospective exploratory pilot study was conducted as a hypothesis-generating analysis and included adult recipients of primary deceased-donor KT at the Transplant-Nephrology Department, University Hospital Martin, Slovakia. Eligible participants were adults (>18 years) undergoing their first deceased-donor KT, with low immunological risk, and written informed consent. Exclusion criteria included previous KT, pediatric age, known DM or prediabetes at the time of KT, treatment with immunosuppressive therapy for at least six months before KT, refusal to participate, or a positive crossmatch test.

2.1. Investigation Algorithm

1. Waiting-list assessment

All patients listed for KT underwent metabolic screening to exclude DM or prediabetes according to the 2024 International Consensus Recommendations on PTDM. Screening included an oral glucose tolerance test (oGTT), fasting plasma glucose, and HbA1c. Subsequently, oGTT and HbA1c were repeated annually, whereas fasting glucose was monitored monthly. Patients diagnosed with DM or prediabetes at any stage before KT were excluded from the study.

2. Kidney transplantation

Upon admission for transplantation, approximately 4–5 h before surgery, body weight, waist circumference, and body mass index (BMI) were recorded. Blood samples were col-

lected for routine biochemical analyses, including plasma glucose, lipid profile, complete blood count, and a crossmatch testing. After confirmation of a negative crossmatch result and final eligibility for transplantation, CGM was initiated using the Dexcom G6 system, Dexcom, Inc. (San Diego, CA, United States). The glucose sensor was placed on the posterior aspect of the upper arm, and glucose data were continuously transmitted to a paired smartphone running the Dexcom Clarity application. Interstitial glucose concentrations were monitored for seven consecutive days.

3. Postoperative day 7 (in hospital)

On the final day of CGM, blood samples were collected for biochemical analyses, including fasting glucose, lipid profile, complete blood count, tacrolimus trough concentration, and HbA1c. HbA1c was measured on postoperative day 7 as part of the predefined study protocol to compare conventional laboratory markers with CGM-derived parameters during the early post-transplant period. It was not used for the diagnosis of PTDM. Fasting and postprandial insulin and C-peptide concentrations were determined, and the estimated glomerular filtration rate (eGFR) and homeostatic model assessment for insulin resistance (HOMA-IR) were calculated. CGM data were downloaded from the Dexcom Clarity platform and included mean glucose, standard deviation (SD), coefficient of variation (CV), estimated HbA1c, interquartile range (IQR), time in range (TIR), time above range (TAR), and time below range (TBR). CGM was performed under standardized inpatient conditions, with all patients continuously supervised throughout the 7-day monitoring period. The CGM receiver remained connected to a paired smartphone positioned close to the patient, ensuring uninterrupted data transmission. CGM data completeness exceeded 90% in all included participants. Patients with more than 10% missing CGM data were excluded from further analysis.

4. Three-month follow-up (short, planned hospital stay)

At three months after KT, all patients underwent a protocol graft biopsy and donor-specific antibody (DSA) assessment using the Luminex assay. Blood samples were collected to determine eGFR, fasting glucose, lipid profile, tacrolimus trough concentration, HbA1c, insulin, C-peptide, and HOMA-IR. Each patient also underwent a standard oGTT.

5. Twelve-month follow-up (as an outpatient)

The same evaluation protocol at the 3-month visit was repeated 12 months after KT.

During follow-up, patients attended the transplant outpatient clinic weekly during the first 3 months, every two weeks between months 3 and 6, and monthly thereafter until 12. Additional graft biopsy and DSA assessment were performed whenever clinically indicated, including deterioration of kidney function or unexplained proteinuria.

2.2. Immunosuppressive Treatment

All participants were classified as having low immunological risk. Induction immunosuppression consisted of either basiliximab administered in two doses (immediately before KT and on postoperative day 4) or antithymocyte globulin (ATG) administered at a cumulative dose of 3.5 mg/kg over three days, together with intravenous methylprednisolone (500 mg before KT and on postoperative day 1). Maintenance immunosuppressive therapy consisted of tacrolimus, mycophenolic acid, and prednisone. Prednisone was gradually tapered from 20 mg/day to 7.5 mg/day by month 12. Tacrolimus dosing was adjusted according to trough concentrations throughout follow-up.

2.3. Diagnosis of Glucose Metabolism Disorder

PTDM was diagnosed according to the 2025 American Diabetes Association criteria as one or more of the following: fasting plasma glucose ≥ 7.0 mmol/L, random plasma

glucose ≥ 11.1 mmol/L in the presence of classic hyperglycemic symptoms, 2 h plasma glucose ≥ 11.1 mmol/L during oGTT, or HbA1c $\geq 6.5\%$. Prediabetes was defined as fasting glucose between 5.6 and 6.9 mmol/L, 2 h plasma glucose between 7.8 and 11.0 mmol/L during oGTT, or HbA1c between 5.7 and 6.4% [13]. In accordance with the updated 2024 International Consensus Recommendations on PTDM, glucose metabolism was primarily assessed using a standard oGTT. Plasma glucose concentrations were measured at baseline and 1 and 2 h after ingestion of 75 g of glucose dissolved in 200 mL of water [14]. The HOMA-IR index, calculated by multiplying fasting plasma glucose (FPG) and fasting plasma insulin (FPI), divided by the constant 22.5, i.e., $\text{HOMA-IR} = (\text{FPG} \times \text{FPI})/22.5$, was used to determine insulin resistance (IR). No glucose-lowering therapy, including insulin, was administered during the seven-day CGM period. Patients requiring initiation of insulin therapy during this period were excluded from further prospective follow-up. This approach enabled assessment of intrinsic glucose patterns without pharmacological interference.

2.4. Statistical Methods

Descriptive statistics were used to summarize the study population. Normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Between-group comparisons were performed using Student's *t*-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the χ^2 test or Fisher's exact test, as appropriate.

To evaluate associations between CGM-derived parameters and outcomes, both univariable and adjusted logistic regression analyses were performed. Given the limited sample size ($n = 20$) and number of outcome events, a parsimonious modeling strategy was applied to minimize overfitting. Each adjusted model included one CGM-derived parameter and two clinically relevant covariates selected a priori: age and body mass index (BMI) at transplantation, both established risk factors for post-transplant diabetes mellitus. The number of covariates was intentionally limited, and highly correlated metabolic variables (e.g., HbA1c, OGTT-derived measures, insulin resistance indices) were not included simultaneously to avoid collinearity and model instability. Odds ratios (ORs) and adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported.

Receiver operating characteristic (ROC) curve analyses were performed to assess the discriminatory performance of HbA1c measured at different time points (day 7, month 3, and month 12) for prediction of post-transplant diabetes mellitus (PTDM) and the combined outcome of PTDM + prediabetes. The area under the curve (AUC) with 95% confidence intervals was calculated. Optimal cut-off values were determined using the Youden index. Comparisons between ROC curves were performed using the DeLong method.

All statistical tests were two-sided, and a *p*-value < 0.05 was considered statistically significant. Statistical analyses were performed using MedCalc Statistical Software version 19.6.4 (MedCalc Software Ltd., Ostend, Belgium).

Given the exploratory nature of the study and the limited number of events, emphasis was placed on effect size estimation and consistency of associations rather than solely on statistical significance.

3. Results

3.1. Baseline Characteristics

A total of 20 KT recipients completed the 12-month follow-up and were included in the final analysis. Thirteen patients (65%) were men, and the mean age at KT was 47.5 ± 14.6 . Induction therapy with ATG was administered to 12 patients (60%), six (30%) received kidneys from expanded-criteria donors (ECDs), and delayed graft function (DGF) occurred in two patients (10%). At transplantation, the mean BMI was 24.0 ± 3.3 kg/m², body weight

was 76.3 ± 12.5 kg, and waist circumference was 91.4 ± 11.1 cm. Mean pre-transplant plasma glucose was 4.9 ± 0.4 mmol/L, total cholesterol 4.5 ± 1.1 mmol/L, LDL cholesterol 2.8 ± 1.2 mmol/L, HDL cholesterol 1.1 ± 0.3 mmol/L, triacylglycerols 1.9 ± 0.8 mmol/L, and hemoglobin 119.5 ± 13.3 g/L.

3.2. Evolution of Clinical and Metabolic Parameters After KT

During the 12-month follow-up, BMI, body weight, and waist circumference increased progressively. Mean BMI increased from 24.9 ± 3.3 kg/m² on postoperative day 7 to 25.4 ± 4.1 kg/m² at month 3 and 26.5 ± 4.8 kg/m² at month 12. Similarly, body weight increased from 76.3 ± 12.5 kg to 77.2 ± 13.9 kg and 81.1 ± 16.3 kg, while waist circumference increased from 91.4 ± 11.1 cm to 92.8 ± 12.1 cm and 95.6 ± 12.7 cm, respectively. Renal function and several laboratory parameters improved over time. Mean eGFR (36 ± 22.3 , 54.2 ± 17 , 52.7 ± 17 on day 7, month 3 and month 12, respectively), hemoglobin (96 ± 7.2 , 130 ± 18.8 , 139.7 ± 20.3 on day 7, month 3 and month 12, respectively), LDL (2.7 ± 1.3 , 2.8 ± 1 , 2.4 ± 0.7 on day 7, month 3 and month 12, respectively), HDL (1.2 ± 0.4 , 1.3 ± 0.5 , 1.4 ± 0.7 on day 7, month 3 and month 12, respectively), and TAG (2.3 ± 0.9 , 2.1 ± 1.1 , 1.7 ± 0.6 on day 7, month 3 and month 12, respectively) improved. There was a significant rise in mean fasting insulin increased from 4.8 ± 1.5 mIU/L on postoperative day 7 to 7.6 ± 3.4 mIU/L at month 3 and 7.7 ± 3.8 mIU/L at month 12. Postprandial insulin increased from 11.8 ± 5.4 mIU/L to 35.9 ± 40.0 mIU/L and 33.5 ± 34.6 mIU/L, respectively. HbA1c increased from $3.6 \pm 0.4\%$ on postoperative day 7 to $3.9 \pm 1.0\%$ at month 3 and $4.1 \pm 1.0\%$ at month 12. Similarly, HOMA-IR increased from 1.1 ± 0.4 to 1.8 ± 1.0 at both month 3 and month 12. Two episodes of biopsy-proven antibody-mediated rejection (ABMR) were recorded during follow-up. One episode was detected at protocol biopsy (month 3), whereas the second was diagnosed later by indication biopsy (Table 1, Figure S1).

Table 1. Basic characteristics detected during the study.

<i>n</i> = 20	Baseline	Day 7	Month 3	Month 12
DSA positive (%)	0 (<i>n</i> = 0)	-	5 (<i>n</i> = 1)	20 (<i>n</i> = 4)
BPAR (ABMR) (%)	-	-	5 (<i>n</i> = 1)	5 (<i>n</i> = 1)
BMI (kg/m ²) $\pm$ SD, median	24.9 $\pm$ 3.3, 26.4	-	25.4 $\pm$ 4.1, 26.1	26.5 $\pm$ 4.8, 26.5
weight (kg) $\pm$ SD, median	76.3 $\pm$ 12.5, 77.5	-	77.2 $\pm$ 13.9, 76.6	81.1 $\pm$ 16.3, 80
waist circumference (cm) $\pm$ SD, median	91.4 $\pm$ 11.1, 92.5	-	92.8 $\pm$ 12.1, 94.5	95.6 $\pm$ 12.7, 99
fasting glucose (mmol/L) $\pm$ SD, median	4.9 $\pm$ 0.4, 5	5.2 $\pm$ 1.1, 4.9	5.4 $\pm$ 1.6, 5.1	5.3 $\pm$ 1.2, 5
1 h post load plasma glucose (mmol/L) $\pm$ SD, median	-	-	8.7 $\pm$ 3.6, 8.6	8.5 $\pm$ 2.3, 8
2 h post load plasma glucose (mmol/L) $\pm$ SD, median	-	-	8 $\pm$ 2.5, 7.5	8 $\pm$ 2.2, 7.4
cholesterol (mmol/L) $\pm$ SD, median	4.5 $\pm$ 1.1, 4.4	4.4 $\pm$ 1.5, 4	5 $\pm$ 1, 5.3	4.4 $\pm$ 1, 4.3
LDL (mmol/L) $\pm$ SD, median	2.8 $\pm$ 1.2, 3	2.7 $\pm$ 1.3, 2.4	2.8 $\pm$ 1, 3	2.4 $\pm$ 0.7, 2.4
HDL (mmol/L) $\pm$ SD, median	1.1 $\pm$ 0.3, 1	1.2 $\pm$ 0.4, 1.1	1.3 $\pm$ 0.5, 1.2	1.4 $\pm$ 0.7, 1.2
TAG (mmol/L) $\pm$ SD, median	1.9 $\pm$ 0.8, 1.8	2.3 $\pm$ 0.9, 2.3	2.1 $\pm$ 1.1, 1.6	1.7 $\pm$ 0.6, 1.7
hemoglobin (g/L) $\pm$ SD, median	119.5 $\pm$ 13.3, 120.5	96 $\pm$ 7.2, 95.5	130 $\pm$ 18.8, 128.5	139.7 $\pm$ 20.3, 141
creatinine (μ mol/L) $\pm$ SD, median	-	238.9 $\pm$ 147.9, 184.5	131.9 $\pm$ 35, 129	135.5 $\pm$ 39.7, 130
eGFR (ml/min) $\pm$ SD, median	-	36 $\pm$ 22.3, 31.5	54.2 $\pm$ 17, 55	52.7 $\pm$ 17, 51
TAC level (ng/mL) $\pm$ SD, median	-	12.6 $\pm$ 4.8, 11.5	8.5 $\pm$ 1.8, 8.8	6.5 $\pm$ 1.5, 6.6
IRI fasting (mU/L) $\pm$ SD, median	-	4.8 $\pm$ 1.5, 4.7	7.6 $\pm$ 3.4, 7.3	7.7 $\pm$ 3.8, 6.3
IRI 2 h post load (mU/L) $\pm$ SD, median	-	11.8 $\pm$ 5.4, 11.1	35.9 $\pm$ 40, 28.9	33.5 $\pm$ 34.6, 24.4

Table 1. *Cont.*

<i>n</i> = 20	Baseline	Day 7	Month 3	Month 12
C-peptide fasting (µg/L) ± SD, median	-	4 ± 1.4, 3.7	3.8 ± 1.4, 3.7	3.5 ± 1.3, 3.2
C-peptide 2 h post load (µg/L) ± SD, median	-	7.2 ± 2.8, 6.6	10.1 ± 5.9, 8.9	9.9 ± 6.9, 8.3
HOMA-IR ± SD, median	-	1.1 ± 0.4, 1.1	1.8 ± 1, 1.7	1.8 ± 1, 1.5
HbA1c (%) ± SD, median	-	3.6 ± 0.4, 3.8	3.9 ± 1, 3.8	4.1 ± 1, 3.8

DSA—donor specific antibodies, BPAR—biopsy proven acute rejection, BMI—body mass index, LDL—low-density lipoprotein, HDL—high-density lipoprotein, TAG—triglycerides, eGFR—estimated glomerular filtration rate, TAC—tacrolimus, IRI—immunoreactive insulin, HbA1c—glycated hemoglobin, HOMA-IR—homeostatic model assessment for insulin resistance, SD—standard deviation, PTDM—post-transplant diabetes mellitus.

3.3. Post-Transplant Diabetes Mellitus and Prediabetes

At the 12-month follow-up, PTDM was diagnosed in seven patients (35%) based on the oGTT, including four patients who had already met the diagnostic criteria at the 3-month visit. Prediabetes was diagnosed in three patients (15%) at month 12, resulting in an overall incidence of post-transplant dysglycemia of 50%. Baseline and 12-month characteristics of patients who developed PTDM/prediabetes and those who remained free of post-transplant dysglycemia are summarized in Table 2. Corresponding comparisons at postoperative day 7 and month 3 are presented in Table S1.

Table 2. Comparison of basic characteristics between control and PTDM/prediabetes group.

	Control Group (<i>n</i> = 10)	PTDM/Prediabetes (<i>n</i> = 10)	<i>p</i> Value
		baseline	
male (%)	70 (<i>n</i> = 7)	60 (<i>n</i> = 6)	0.7166
age at KT (years) ± SD, median	42.8 ± 15.6, 41.4	52.1 ± 12.5, 53	0.1585
ECD donor (%)	30 (<i>n</i> = 3)	30 (<i>n</i> = 3)	1.0000
induction—ATG (%)	60 (<i>n</i> = 6)	60 (<i>n</i> = 6)	1.0000
DGF (%)	10 (<i>n</i> = 1)	10 (<i>n</i> = 1)	1.0000
BMI (kg/m ²) ± SD, median	24.9 ± 3.5, 26.3	25 ± 3.3, 26.4	0.9483
weight (kg) ± SD, median	78.2 ± 11.3, 81.3	74.4 ± 13.9, 77	0.5109
waist circumference (cm) ± SD, median	91 ± 11, 92	91.8 ± 11.8, 94	0.8771
fasting glucose (mmol/L) ± SD, median	4.8 ± 0.4, 4.7	5.1 ± 0.3, 5.1	0.0739
cholesterol (mmol/L) ± SD, median	4.7 ± 1.3, 4.6	4.2 ± 1, 4.4	0.3478
LDL (mmol/L) ± SD, median	3.1 ± 1.3, 3.3	2.6 ± 1.2, 2.9	0.3833
HDL (mmol/L) ± SD, median	1.1 ± 0.3, 1.1	1.1 ± 0.3, 1	1.0000
TAG (mmol/L) ± SD, median	1.6 ± 0.7, 1.4	2.3 ± 0.8, 1	0.0518
hemoglobin (g/L) ± SD, median	117.8 ± 11.4, 119	121.1 ± 15.5, 122.5	0.5942
		month 12	
TAC level (ng/mL) ± SD, median	6.6 ± 1.4, 6.6	6.4 ± 1.6, 6.9	0.7695
cholesterol (mmol/L) ± SD, median	4.4 ± 0.7, 4.2	4.5 ± 1.2, 4.5	0.8225
LDL (mmol/L) ± SD, median	2.4 ± 0.7, 2.3	2.5 ± 0.7, 2.6	0.7531
HDL (mmol/L) ± SD, median	1.4 ± 0.9, 1.2	1.3 ± 0.4, 1.2	0.7518
TAG (mmol/L) ± SD, median	1.7 ± 0.6, 1.8	1.7 ± 0.6, 1.7	1.0000
IRI fasting (mU/IL ± SD, median)	5.8 ± 2.3, 5	9.6 ± 4.2, 9.5	0.0219
IRI 2 h post load (mU/L) ± SD, median	24.4 ± 16.3, 18.4	42.6 ± 45.6, 26.8	0.2501
C-peptide fasting (µg/L) ± SD, median	3.4 ± 1.1, 3	3.6 ± 1.6, 3.4	0.7484
C-peptide 2 h post load (µg/L) ± SD, median	10.6 ± 6.1, 8.9	9.2 ± 8, 6.7	0.6651

Table 2. Cont.

	Control Group (<i>n</i> = 10)	PTDM/Prediabetes (<i>n</i> = 10)	<i>p</i> Value
fasting glucose (mmol/L) $\pm$ SD, median	4.9 $\pm$ 0.3, 4.9	5.7 $\pm$ 1.6, 5.1	0.1376
1 h post load plasma glucose (mmol/L) $\pm$ SD, median	7.1 $\pm$ 1.3, 7	10.8 $\pm$ 1.7, 10.9	<0.0001
2 h post load plasma glucose (mmol/L) $\pm$ SD, median	6.6 $\pm$ 0.8, 6.7	10.2 $\pm$ 1.9, 10.4	<0.0001
HOMA-IR $\pm$ SD, median	1.3 $\pm$ 0.5, 1.2	2.4 $\pm$ 1, 2.4	0.0060
HbA1c (%) $\pm$ SD, median	3.5 $\pm$ 0.2, 3.6	4.7 $\pm$ 1.2, 4.4	0.0059
creatinine (μ mol/L) $\pm$ SD, median	144.2 $\pm$ 36.1, 134.5	126.7 $\pm$ 43.1, 115	0.3380
eGFR (ml/min) $\pm$ SD, median	50 $\pm$ 14.5, 48.5	55.3 $\pm$ 19.6, 54	0.5006
hemoglobin (g/L) $\pm$ SD, median	140.4 $\pm$ 16.2, 140.5	139 $\pm$ 24.7, 143	0.8825
BMI (kg/m ²) $\pm$ SD, median	27.6 $\pm$ 5.8, 28.5	25.4 $\pm$ 3.5, 26.2	0.3180
weight (kg) $\pm$ SD, median	87.6 $\pm$ 16.8, 89	74.7 $\pm$ 13.6, 76.4	0.0754
waist circumference (cm) $\pm$ SD, median	96.8 $\pm$ 13.5, 101	94.4 $\pm$ 12.4, 96	0.6837

KT—kidney transplantation, ECD—expanded criteria donor, DGF—delayed graft function BMI—body mass index, LDL—low-density lipoprotein, HDL—high-density lipoprotein, TAG—triglycerides, eGFR—estimated glomerular filtration rate, TAC—tacrolimus, IRI—immunoreactive insulin, HbA1c—glycated hemoglobin, HOMA-IR—homeostatic model assessment for insulin resistance, SD—standard deviation, PTDM—post-transplant diabetes mellitus.

No significant baseline differences were observed between patients who subsequently developed PTDM/prediabetes and those who remained free of post-transplant dysglycemia with respect to age, sex, donor type, induction therapy, DGF, or baseline anthropometric and laboratory parameters. Tacrolimus trough concentrations and cumulative corticosteroid exposure were comparable between groups. No between-group differences were observed in the incidence of acute rejection episodes, infectious complications known to influence glucose metabolism (hepatitis C virus, cytomegalovirus), duration of dialysis prior to KT or family history of DM.

On postoperative day 7, the PTDM/prediabetes group had higher HbA1c levels (3.8 ± 0.4 vs. 3.4 ± 0.4 , $p = 0.0382$) and lower postprandial immunoreactive insulin (IRI) concentrations (9.3 ± 3.2 vs. 14.2 ± 6.3 , $p = 0.0417$).

At the 3-month follow-up, the PTDM/prediabetes group exhibited significantly higher 1 h and 2 h plasma glucose concentrations during the oGTT (11.2 ± 3.3 vs. 6.3 ± 1.5 , $p = 0.0005$; 10 ± 1.7 vs. 5.9 ± 0.8 , $p < 0.0001$; respectively), together with higher HbA1c levels (4.4 ± 1.2 vs. 3.5 ± 0.5 , $p = 0.0420$), and HOMA-IR (2.3 ± 1.2 vs. 1.4 ± 0.5 , $p = 0.0420$).

At 12 months, patients who developed PTDM/prediabetes continued to demonstrate higher fasting IRI concentrations (9.6 ± 4.2 vs. 5.8 ± 2.3 , $p = 0.0219$), higher 1 h and 2 h plasma glucose concentrations during the oGTT (10.8 ± 1.7 vs. 7.1 ± 1.3 , $p < 0.0001$; 10.2 ± 1.9 vs. 6.6 ± 0.8 , $p < 0.0001$; respectively), higher HbA1c levels (4.7 ± 1.2 vs. 3.5 ± 0.2 , $p = 0.0059$), and higher HOMA-IR values (2.4 ± 1 vs. 1.3 ± 0.5 , $p = 0.0060$).

Longitudinal analysis demonstrated a significant increase in both fasting and postprandial IRI concentrations in the PTDM/prediabetes group (fasting IRI, $p = 0.0047$; postprandial IRI, $p = 0.0344$), whereas no significant changes were observed in controls. Likewise, HOMA-IR increased significantly only in the PTDM/prediabetes group ($p = 0.0032$) (Figure 1).

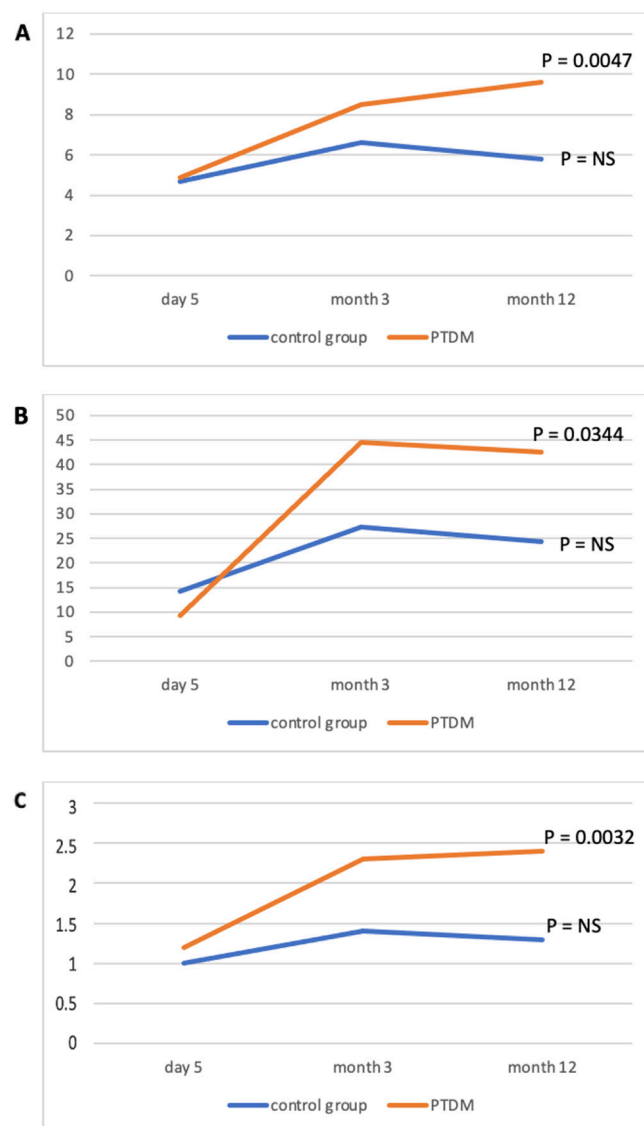


Figure 1. Development of specific characteristics in both groups: (A) fasting insulin, (B) postprandial insulin, (C) HOMA-IR. Abbreviations: PTDM—post-transplant diabetes mellitus, NS—nonsignificant.

3.4. Glycemic Variability

Seven-day CGM demonstrated a mean glucose concentration of 7.5 ± 1.8 mmol/L. On average, patients spent $80.6 \pm 17\%$ of the monitoring time within the target range, $12.5 \pm 15\%$ above range (TAR-high), $5.1 \pm 7.4\%$ above the very high range (TAR-very high), $3.0 \pm 5.4\%$ below range (TBR-low), and $0.5 \pm 0.8\%$ below the very low range (TBR-very low). The mean SD was 2.4 ± 1.0 mmol/L; estimated HbA1c was $6.5 \pm 0.9\%$; and the mean CV was $31.2 \pm 8.0\%$. Five patients (25%) had $CV \geq 36\%$.

Compared with patients who remained free of post-transplant dysglycemia, the PTDM/prediabetes group exhibited significantly higher mean glucose concentrations (8.4 ± 1.9 vs. 6.6 ± 1.2 , $p = 0.0208$), SD (2.9 ± 1.1 vs. 1.2 ± 0.7 , $p = 0.0006$), and estimated HbA1c (7 ± 0.9 vs. 6.1 ± 0.6 , $p = 0.0169$), shorter TIR (72.8 ± 20.2 vs. 88.4 ± 8.3 , $p = 0.0365$), and longer TAR—high (19.2 ± 18.5 vs. 5.8 ± 5.8 , $p = 0.0423$) and TAR—very high (8.8 ± 9 vs. 1.3 ± 2.1 , $p = 0.0194$) (Table 3).

Table 3. Comparison of CGM characteristics between the groups.

<i>n</i> = 20	Control Group (<i>n</i> = 10)	PTDM/Prediabetes (<i>n</i> = 10)	<i>p</i> Value
average glucose (mmol/L)	6.6 ± 1.2, median 6.5	8.4 ± 1.9, median 8.4	0.0208
standard deviation ± SD, median	1.2 ± 0.7, median 1.8	2.9 ± 1.1 median 2.8	0.0006
coefficient of variation ± SD, median	28.8 ± 6.8, median 27.3	33.6 ± 8.8, median 31.2	0.1819
coefficient of variation ≥ 36 (%)	10 (<i>n</i> = 1)	40 (<i>n</i> = 4)	0.6124
estimated A1c (%)	6.1 ± 0.6, median 6	7 ± 0.9, median 6.9	0.0169
IQR 25/75 ± SD, median	2.4 ± 1, median 2.2	3.3 ± 1.7, median 2.7	0.1662
TIR (%) ± SD, median	88.4 ± 8.3, median 91.5	72.8 ± 20.2, median 74	0.0365
TAR—high (%) ± SD, median	5.8 ± 5.8, median 4.5	19.2 ± 18.5, median 13.5	0.0423
TAR—very high (%) ± SD, median	1.3 ± 2.1, median 0	8.8 ± 9, median 10.5	0.0194
TBR—low (%) ± SD, median	4.3 ± 7.4, median 1	1.8 ± 1.8, median 1.3	0.3130
TBR—very low (%) ± SD, median	0.5 ± 1, median 0	0.5 ± 0.7, median 0	1.0000

PTDM—post-transplant diabetes mellitus, SD—standard deviation, IQR—interquartile range, TIR—timer in range, TAR—time above the range, TBR—time below the range.

In exploratory logistic regression analyses, selected CGM-derived parameters were associated with subsequent PTDM/prediabetes. A CV ≥ 36% showed an association with the composite outcome in both the univariable analysis (OR 9.00, 95% CI 1.38–58.44, *p* = 0.021) and the limited age- and BMI-adjusted sensitivity analysis (aOR 7.31, 95% CI 1.19–43.36, *p* = 0.049). Similarly, higher TAR was associated with the outcome in both the univariable (OR 2.78, 95% CI 1.16–6.64, *p* = 0.021) and adjusted sensitivity analyses (aOR 1.35, 95% CI 1.01–2.10, *p* = 0.045) (Table 4). Other CGM-derived parameters, including SD and CV analyzed as continuous variables, showed similar directional trends but did not reach statistical significance in adjusted models. Likewise, TIR demonstrated a consistent inverse association with the outcome. When PTDM alone was analyzed as the outcome, the direction and magnitude of the observed associations remained broadly consistent; however, none reached statistical significance, most likely reflecting the smaller number of events and the resulting limited statistical power (Table S2). Notably, both CV ≥ 36% and TAR showed comparable effect sizes, although the confidence intervals were wide.

Table 4. Univariable and limited age- and BMI-adjusted exploratory logistic regression analyses for PTDM/prediabetes.

<i>n</i> = 20, Outcome: PTDM + Prediabetes Development	OR (95% CI)	<i>p</i> Value	aOR (95% CI)	<i>p</i> Value
age at the time of KT (years)	1.05 (0.98–1.12)	0.1597	1.05 (0.97–1.14)	0.1519
BMI at the time of KT (kg/m ²)	1.01 (0.77–1.33)	0.9091	0.92 (0.67–1.27)	0.6455
average glucose	2.17 (1.00–4.68)	0.0478	1.71 (0.10–2.84)	0.4011
standard deviation	2.89 (0.93–8.98)	0.0649	1.35 (1.01–2.10)	0.0734
coefficient of variation	0.70 (0.46–1.07)	0.1040	0.70 (0.46–1.07)	0.0556
coefficient of variation ≥ 36	9.00 (1.38–58.44)	0.0213	7.31 (1.19–43.36)	0.0491
estimated A1c	1.10 (0.62–1.95)	0.7482	1.01 (0.55–1.84)	0.9831
IQR 25/75	1.79 (0.79–4.04)	0.1576	1.03 (0.72–1.46)	0.8999
TIR	0.92 (0.85–4.00)	0.0661	1.54 (1.03–3.45)	0.0828
TAR—high + very high	2.78 (1.16–6.64)	0.0209	1.35 (1.01–2.10)	0.0454
TBR—low + very low	1.00 (0.38–2.60)	1.0000	1.10 (0.99–1.23)	0.1032

Values are presented as odds ratios (OR) and adjusted odds ratios (aOR) with 95% confidence intervals (CI). Adjusted models were performed as exploratory sensitivity analyses and included one CGM-derived parameter together with age and BMI at transplantation, selected a priori as clinically relevant confounders to minimize overfitting. Analyses were performed using logistic regression. Given limited sample size and number of outcome events, these models were not intended to provide definitive evidence of independent prediction and should be interpreted with caution. PTDM—post-transplant diabetes mellitus, KT—kidney transplantation, BMI—body mass index, IQR—interquartile range, TIR—timer in range, TAR—time above the range, TBR—time below the range.

Exploratory ROC analysis suggested that $SD > 2.1$ mmol/L (AUC 0.735, $p = 0.049$) may discriminate patients who subsequently developed PTDM/prediabetes with a specificity of 80% and sensitivity of 70% (Figure 2).

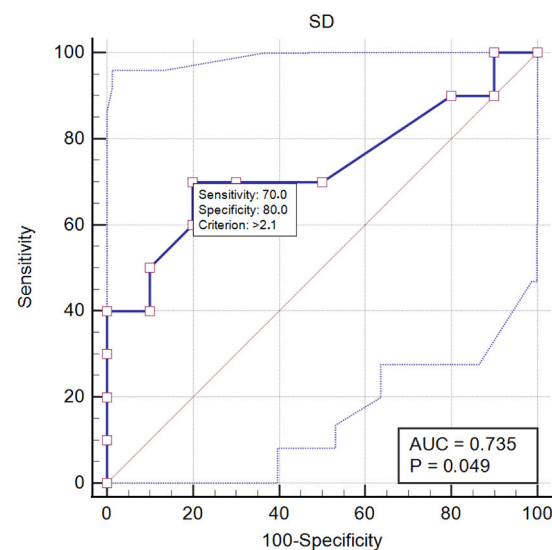


Figure 2. Exploratory discriminatory performance of standard deviation for the combined outcome of PTDM and prediabetes. Receiver operating characteristic (ROC) curves demonstrate the ability of SD measured by 7-day CGM after transplantation to discriminate patients who developed the respective outcome. $SD > 2.1$: AUC 0.735 ($p = 0.049$), sensitivity 70%, specificity 80%. Cut-off values were determined using the Youden index. Given the small pilot cohort, ROC-derived cut-off values were considered exploratory and were not interpreted as clinically validated thresholds. Abbreviations: SD—standard deviation, AUC—area under the curve.

4. Discussion

In this prospective exploratory pilot study of primary deceased-donor KT recipients, patients who subsequently developed PTDM or prediabetes had higher mean glucose, SD, and estimated HbA1c, shorter TIR, and longer TAR during the 7-day CGM period. Exploratory regression analyses suggested associations between selected CGM-derived parameters, particularly $CV \geq 36\%$ and TAR and subsequent PTDM/prediabetes at 12 months after KT. In addition, $SD > 2.1$ mmol/L during the early post-transplant period showed potential discriminatory ability for subsequent post-transplant dysglycemia. To our knowledge, previous studies evaluating CGM for the prediction of PTDM have been conducted exclusively in living donor KT recipients.

These findings are consistent with previous reports demonstrating the feasibility of CGM for assessing GV after transplantation [5,12,15,16]. Unlike conventional capillary glucose monitoring, CGM provides continuous 24 h glucose profiles, allowing a more comprehensive assessment of glycemic fluctuations. Randomized studies have shown that CGM improves post-transplant glycemic control and may facilitate the early identification of patients at increased risk of PTDM compared with intermittent glucose monitoring [12,16].

Our cohort consisted of urgent deceased-donor KTs, in whom preoperative CGM assessment is generally not feasible. Therefore, all participants underwent comprehensive metabolic screening before KT in accordance with the 2024 International Consensus Recommendations to exclude pre-existing DM or prediabetes [14]. Previous studies have demonstrated that pre-transplant impaired glucose tolerance and fasting hyperglycemia are established risk factors for PTDM [12,17]. Our exploratory findings suggest that CGM-derived parameters may contribute to early metabolic phenotyping after KT. Nevertheless, subtle abnormalities of glucose metabolism cannot be completely excluded in patients with end-stage kidney

disease despite standardized pre-transplant screening. Because oGTT was repeated annually while patients remained on the waiting list, metabolic abnormalities may still have developed between the last scheduled assessment and KT. Although PTDM and prediabetes are distinct diagnostic entities, post-transplant abnormalities of glucose metabolism are often considered part of a continuum ranging from impaired glucose tolerance to overt PTDM. Owing to the limited sample size and number of outcome events, these conditions were analyzed as a composite endpoint representing post-transplant dysglycemia.

Conventional blood glucose monitoring, whether venous or capillary, is invasive and does not adequately capture GV, which is increasingly recognized as an important contributor to diabetes-related complications [10]. Although oGTT performed at 3 and 12 months enables the diagnosis of PTDM and prediabetes, it does not provide information on early post-KT prediction. Early identification of patients with adverse glycemic profiles may facilitate future studies evaluating individualized preventive strategies, such as optimizing immunosuppression, adjusting corticosteroids, and targeting lifestyle interventions. Whether CGM-guided interventions can improve long-term metabolic outcomes after KT remains to be established.

Our exploratory findings are consistent with those of recent studies. Shin et al. (2024) reported that a postoperative TAR ≥ 180 mg/dL was associated with an increased risk of PTDM in a cohort of 60 living-donor KT patients [12]. A major strength of their study was the use of preoperative CGM, which enabled clear identification and exclusion of patients with pre-existing glucose metabolism disorders [12]. Similarly, Eleftheriadis et al. (2024) reported that TAR thresholds of 140 and 180 mg/dL were associated with subsequent PTDM from postoperative day 8 and with PTDM/impaired glucose tolerance from day 30 after KT [18]. However, a limitation of that study was the inability to completely exclude pre-existing dysglycemia, as baseline metabolic assessment relied solely on HbA1c values and a history of antidiabetic treatment at the time of KT [18].

GV metrics, including SD and CV, describe the magnitude and frequency of glucose fluctuations around the mean glucose concentration. SD is a well-established measure of GV and is highly correlated with mean glucose levels and A1c (19). In our exploratory ROC analysis, SD > 2.1 demonstrated potential discriminatory ability for subsequent PTDM/prediabetes. However, SD may be less reliable when glucose values are not normally distributed around the mean, a common characteristic of CGM data. A stable glucose profile has been proposed to correspond to an SD lower than one-third of the mean glucose concentration (19). This relationship was also evident in our cohort, where this calculation indicated an unstable glucose profile in the PTDM/prediabetes group, whereas patients who remained free of post-transplant dysglycemia generally exhibited a stable glucose profile. Among currently available GV metrics, CV is considered the most robust indicator because it is less dependent on mean glucose levels and A1c than SD. Current data show that CV $\geq 36\%$ indicates an unstable glucose profile, while CV $< 36\%$ indicates low GV and this threshold was adopted in the International Consensus on the use of CGM [19,20]. In our cohort, a CV $\geq 36\%$ was associated with subsequent post-transplant dysglycemia in exploratory analyses. However, continuous CV values did not reach statistical significance, most likely because of the limited sample size and statistical power. Therefore, these findings should be interpreted as hypothesis-generating. Consistent with previous transplant studies, higher TAR was also associated with subsequent disturbances of glucose metabolism [12,18].

Conventional laboratory parameters remain essential for the assessment of glucose metabolism and IR after KT. However, fasting glucose, insulin, C-peptide, and HOMA-IR represent single time-point measurements, while HbA1c reflects average glycemia over several weeks and may be difficult to interpret in the early post-transplant period.

Therefore, these conventional markers may not capture short-term glycemic excursions, postprandial or nocturnal hyperglycemia, or day-to-day GV during the dynamic early post-transplant phase characterized by surgical stress, corticosteroid exposure, calcineurin inhibitor (CNI) initiation, changing renal function, and rapid metabolic shifts. In contrast, CGM provides continuous glucose profiles and enables assessment of GV, TIR, and TAR. These parameters may identify early abnormal glucose patterns before they are reflected in fasting glucose or HbA1c. However, our study was not designed or powered to determine whether CGM provides superior predictive performance compared with established early post-transplant markers. Because of the small sample size and limited number of outcome events, direct comparative predictive modeling or robust head-to-head assessment of CGM-derived metrics versus conventional laboratory parameters was not feasible. Therefore, the present findings should be interpreted as demonstrating an association between early CGM-derived abnormalities and subsequent dysglycemia, rather than proving incremental predictive superiority of CGM. Larger prospective studies with adequately powered multivariable models are needed to determine whether CGM improves risk prediction beyond routinely available metabolic markers. In the laboratory parameters we recorded during the follow-up, we found higher HbA1c by day 7 and progressive HOMA-IR increase in the PTDM/prediabetes group. This is an expected finding, as IR is common post-KT and a key PTDM predictor [21,22]. Although HbA1c differences were detectable early after KT in our cohort, interpretation of HbA1c in the immediate post-transplant period requires particular caution. HbA1c measured during the first postoperative week may be affected by procedure-related anemia, perioperative blood loss, high erythrocyte turnover, erythropoietin therapy, shortened erythrocyte lifespan, and possible blood transfusions [23,24]. These factors may lead to underestimation or distortion of true glycemic exposure and may partly explain the very low HbA1c values observed early after KT. Therefore, day-7 HbA1c should not be interpreted as a reliable diagnostic marker of PTDM in this setting. In our study, HbA1c was intentionally measured on postoperative day 7 not for diagnostic purposes, but to describe its behavior and to compare conventional laboratory markers with CGM-derived measures of GV. For this reason, current consensus recommendations highlight the limitations of HbA1c in the early post-transplant setting, while HbA1c remains more suitable for assessment of chronic glycemic control at later stages after KT [1]. In our study, CGM-derived parameters were associated with later development of post-transplant dysglycemia, suggesting that CGM may provide early metabolic phenotyping not captured by HbA1c or other conventional parameters. Although HbA1c measured at later time points showed higher discriminatory performance for established dysglycemia, its performance in the early post-transplant phase was only moderate, consistent with its known biological limitations in this setting (Figure S2). Taken together, our findings suggest that CGM-derived metrics may provide complementary metabolic information during the early post-transplant period.

Interestingly, early lower postprandial insulin and C-peptide levels on day 7 post-KT suggest transient β -cell suppression due to CNI, which inhibit the calcineurin–nuclear factor of activated T cells pathway, reducing insulin secretion [25]. This effect is partly reversible as CNI doses taper, while corticosteroids and lifestyle contribute to later IR. We did not observe a difference in the average CNI level between both groups, but the average TAC level in the entire cohort was 12.6 ± 4.8 ng/mL on day 7. Interindividual variability in TAC metabolism may also influence β -cell toxicity [26].

The main limitation of this study is the small sample size, with only 20 patients included and 10 patients reaching the combined endpoint. The small number of outcome events limits the reliability of multivariable modeling. Although limited age- and BMI-adjusted models were performed as exploratory sensitivity analyses, these models

may still be affected by overfitting and unstable estimates. Therefore, adjusted results should not be interpreted as definitive evidence of independent prediction, but rather as hypothesis-generating findings requiring external validation. Similarly, ROC-derived cut-off values should be interpreted with caution, as they were generated from a very small pilot cohort with a limited number of outcome events. These thresholds should be considered exploratory and require external validation before being used for clinical risk stratification. The use of CGM in the immediate postoperative period may be influenced by physiological factors such as fluid shifts, changes in tissue perfusion, and metabolic instability. These factors may potentially affect interstitial glucose measurements and represent a recognized limitation of CGM in acutely ill or postoperative patients. However, in our study, CGM was performed under controlled inpatient conditions with standardized clinical management. All patients were monitored in the same perioperative setting, and therefore any potential measurement variability would likely have affected all participants, similarly, minimizing its impact on between-group comparisons. In addition, previous studies have demonstrated acceptable feasibility and accuracy of CGM use in hospitalized and transplant populations, supporting its use in this clinical context [12,18]. Strengths include rigorous screening for glucose metabolism disorders and their proper exclusion before KT, subsequent standardized follow-up, and long observation period. Currently, limited data exist on CGM use in deceased-donor KT; our findings provide preliminary exploratory evidence supporting further investigation of CGM-derived GV in this setting.

5. Conclusions

In patients undergoing primary deceased-donor KT, higher early postoperative GV was associated with the development of PTDM or prediabetes at 12 months. CGM-derived parameters, including $CV \geq 36\%$, $SD > 2.1$ mmol/L, and $TAR \geq 180$ mg/dL, emerged as potential markers of subsequent post-transplant dysglycemia. However, due to the small sample size and exploratory pilot design, these findings should be interpreted with caution. Larger prospective studies are needed to validate the predictive value of CGM and to determine whether early CGM-based risk stratification can improve individualized post-transplant metabolic management.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/kidneydial6030052/s1>.

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Institutional Review Board Statement: All procedures were approved by the Jessenius Faculty of Medicine of Comenius University ethics committee (number EK 33/2018) and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki and the Declaration of Istanbul on Organ Trafficking and Transplant Tourism. The authors confirm that all organs used for transplantation were obtained through legally authorized transplantation program at the Transplant-Nephrology Department, University Hospital Martin, Slovakia. No organs were obtained from prisoners or other vulnerable populations.

Informed Consent Statement: Not applicable.

Data Availability Statement: The raw data supporting the conclusions of this manuscript will be made available by the authors, without undue reservations.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

ABMR	antibody mediated rejection
ATG	antithymocyte globulin
BMI	body mass index
CGM	continuous glucose monitoring
CNI	calcineurin inhibitor
CV	coefficient of variation
DGF	delayed graft function
DM	diabetes mellitus
DSA	donor specific antibody
ECD	expanded criteria donor
eGFR	estimated glomerular filtration rate
GV	glycemic variability
HbA1c	glycated hemoglobin A1c
HDL	high density lipoprotein
HOMA-IR	homeostatic model assessment index for insulin resistance
IQR	interquartile range
IRI	immunoreactive insulin
KT	kidney transplantation
LDL	low density lipoprotein
oGTT	oral glucose tolerance test
PTDM	post-transplant diabetes mellitus
SD	standard deviation
TAC	tacrolimus
TAG	triglycerides
TAR	time above range
TIH	time in hyperglycemia
TIR	time in range
TBR	time below range

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