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Impact of Preoperative Metabolic Comorbidities on Renal Function and Survival in Living Kidney Donors

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Abbreviations:
BMI: Body Mass Index;
CI: Confidence Interval;
CKD: Chronic Kidney Disease;
GFR: Estimated Glomerular Filtration Rate;
ESKD: End-Stage Kidney Disease;
HTN: Hypertension;
KDIGO: Kidney Disease: Improving Global Outcomes;
OR: Odds Ratio;
RRT: Renal Replacement Therapy;
sCR: Serum Creatinine;
SD: Standard Deviation.

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Rezumat

Impactul comorbidităților metabolice preoperatorii asupra funcției renale și supraviețuirii în cazul donatorilor vii de grefă renală

Context: Transplantul renal de la donator viu reprezintă tratamentul optim pentru pacienții cu boală renală cronică în stadiu final, însă impactul pe termen lung al comorbidităților metabolice rămâne insuficient studiat. Ghidurile contemporane recomandă stratificarea individualizată a riscului, dar semnificația clinică a unor afecțiuni precum obezitatea, hipertensiunea arterială şi dislipidemia necesită investigații suplimentare. *Obiectiv:* Evaluarea impactului comorbidităților metabolice preoperatorii asupra funcției renale pe termen lung şi a supraviețuirii pacienților într-o cohortă de donatori vii de grefă renală.
Metode: Am realizat un studiu observațional retrospectiv pe 142 de donatori vii de grefă renală, consecutivi, carora li s-a efectuat nefrectomia în perioada ianuarie 2015 – decembrie 2019, într-un centru terțiar de transplant. Perioada medie de urmărire a fost de 100,88 ± 17,75 luni. Obiectivul primar a fost un endpoint combinat definit ca scăderea ratei de filtrare glomerulară estimată (RFGe) sub 45 mL/min/1,73 m² sau decesul de orice cauză. Au fost analizate datele demografice, clinice şi biochimice de bază. Regresia logistică binară a fost utilizată pentru identificarea predictorilor independenți ai endpointului combinat.
Rezultate: 24 de donatori (16,9%) au atins endpointul combinat, dintre care 14,7% au prezentat RFGe < 45 mL/min, iar 2,2% au decedat. În analiza univariată, vârsta la momentul donării (OR 1,12; p < 0,001), dislipidemia (OR 3,32; p = 0,009) şi RFGe bazală (OR 0,94; p < 0,001) au fost semnificativ asociate cu endpointul combinat. În analiza multivariată, vârsta la momentul donării s-a menținut ca factor de risc independent (OR 1,11; IC 95% 1,02–1,21; p = 0,01). Declinul mediu al RFGe în perioada de urmărire a fost de −27,37 ± 17,80 mL/min, corespunzând unei scăderi anuale medii de 3,3 mL/min pe an. Niciunul dintre donatori nu a prezentat RFGe < 15 mL/min şi nu a necesitat terapie de substituție renală.
Concluzii: În concluzie, vârsta la momentul donării a fost singurul predictor independent al endpointului compozit reprezentat de deteriorarea funcției renale sau mortalitatea post-donare în rândul donatorilor vii de rinichi. Comorbiditățile metabolice preoperatorii nu au fost asociate în mod independent cu rezultate adverse în această cohortă. Aceste constatări susțin o abordare individualizată a selecției

donatorilor, integrând vârsta cu povara şi gradul de control al comorbidităților metabolice,atât pentru optimizarea siguranței donatorului cât şi a rezultatelor transplantului.

Cuvinte cheie: donatori vii de grefă renală, nefrectomie de la donator, comorbidități metabolice, rată de filtrare glomerulară estimată, siguranța donatorului, obezitate, hipertensiune arterială, stratificarea riscului

Abstract

Background: Living donor kidney transplantation is the optimal treatment for patients with end-stage kidney disease, but the long-term impact of preoperative metabolic comorbidities on donor outcomes remains insufficiently characterized. Contemporary guidelines advocate individualized risk stratification, but the clinical significance of conditions such as obesity, hypertension, and dyslipidemia in the context of donor nephrectomy requires further elucidation. *Objective:* To evaluate the impact of preoperative metabolic comorbidities on long-term renal function and survival in a cohort of living kidney donors.

Methods: We conducted a retrospective observational study of 142 consecutive living kidney donors who underwent nephrectomy between January 2015 and December 2019 at a single tertiary transplant center. The mean follow-up time was 100.88 ± 17.75 months. The primary endpoint was a composite outcome defined as decline in estimated glomerular filtration rate (eGFR) <45 mL/min/1.73 m² or all-cause death. Baseline demographic, clinical, and biochemical data were analyzed. Binary logistic regression was used to identify independent predictors of the composite endpoint.

Results: Twenty-four donors (16.9%) reached the composite endpoint, comprising 14.7% with eGFR <45 mL/min and 2.2% who died. On univariate analysis, age at donation (OR 1.12, $p < 0.001$), dyslipidemia (OR 3.32, $p = 0.009$), and baseline eGFR (OR 0.94, $p < 0.001$) were significantly associated with the composite endpoint. On multivariate analysis, age at donation retained as an independent risk factor (OR 1.11, 95% CI 1.02–1.21, $p = 0.01$). The mean decline in eGFR during follow-up was −27.37 ± 17.80 mL/min, corresponding to an average annual decrease of 3.3 mL/min per year. None of the donors had eGFR < 15 mL/min or required renal replacement therapy.

Conclusions: In conclusion, age at donation was the only independent predictor of the composite outcome of renal function decline or mortality after living kidney donation. Preoperative metabolic comorbidities were not independently associated with adverse outcomes in this cohort. These findings support an individualized approach to donor selection, integrating age with the burden and control of metabolic comorbidities to optimize donor safety and transplant outcomes.

Keywords: living kidney donor, donor nephrectomy, metabolic comorbidities, estimated glomerular filtration rate, donor safety, obesity, hypertension, risk stratification

Introduction

Living donor kidney transplantation represents the optimal therapeutic modality for patients with end-stage kidney disease, offering superior outcomes compared with deceased donor transplantation (1). Despite these advantages, ensuring donor safety remains an ethical priority. Living donors undergo major surgery and permanent reduction of nephron mass without direct medical benefit, making accurate assessment of their long-term health outcomes critically important (2).

Over time, the living donor pool has expanded to include individuals with metabolic risk factors such as obesity, hypertension, and dyslipidemia, reflecting both organ scarcity and evolving evidence suggesting acceptable outcomes in carefully selected candidates (3). This shift has been driven by the widening gap between organ supply and demand, but also by accumulating evidence suggesting that carefully selected donors with these comorbidities can donate safely. However, practice patterns remain heterogeneous across transplant centers (2).

The Kidney Disease Improving Global Outcomes (KDIGO) guidelines emphasize individualized risk assessment in the evaluation of living kidney donors, recognizing that the long-term impact of metabolic comorbidities in the context of unilateral nephrectomy remains incompletely defined. Accordingly, they recommend comprehensive cardiometabolic evaluation and risk stratification, while acknowledging that conditions such as obesity, impaired fasting glucose, hypertension, and dyslipidemia may influence long-term renal and cardiovascular outcomes. At the same time, the guidelines support individualized decision-making rather than categorical exclusion, reflecting the persistent

uncertainty regarding the absolute risk conferred by these factors in the donor setting (4,5).

The physiological response to unilateral nephrectomy is characterized by compensatory hypertrophy of the remaining kidney and an expected reduction in glomerular filtration rate of approximately 25-30%, followed by partial functional adaptation (6,7). This process is well described and generally considered clinically benign in healthy individuals. However, it remains unclear whether the presence of metabolic comorbidities modifies this adaptive response and increases the risk of clinically significant long-term renal impairment (8).

The present study evaluates the impact of preoperative metabolic comorbidities, including obesity, hypertension, and dyslipidemia, on long-term renal function and survival in a single-center cohort of living kidney donors. We hypothesize that, although these conditions may be associated with differences in renal parameters, they are not independent predictors of clinically significant adverse outcomes in carefully selected donors.

Materials and Methods

Study Design and Population

We conducted a retrospective observational study including 142 consecutive living kidney donors who underwent donor nephrectomy at the Center of Surgical Urology and Kidney Transplantation, Fundeni Clinical Institute, Bucharest, Romania, between January 2015 and December 2019. All donors underwent a comprehensive preoperative evaluation in accordance with institutional protocols aligned with the KDIGO guidelines for living kidney donor evaluation. Donors were followed longitudinally to assess renal function and survival outcomes. The mean follow-up period was 100.88 ± 17.75 months. Data were extracted from medical records and institutional databases.

Preoperative Assessment and Variables

Baseline demographic, clinical, and biochemical data were collected at the time of donation, including age, sex, body mass index (BMI), fasting plasma glucose, serum uric acid, serum creatinine (sCr), and estimated glomerular filtration rate (eGFR). Metabolic comorbidities were defined as follows: obesity was defined as BMI ≥30 kg/m²; hypertension (HTN) was defined as a documented diagnosis of arterial hypertension or the use of antihypertensive medications; dyslipidemia was defined as an established diagnosis or the use of lipid-lowering therapy. Active or former smoking status was recorded. The presence of any comorbidity and the number of coexisting conditions (one versus two or more) were additionally analyzed.

Outcome Measures

The primary endpoint was a composite outcome defined as either decline in renal function to an eGFR < 45 mL/min/1.73 m² or all-cause death during follow-up. This composite endpoint was chosen to capture both fatal and clinically significant non-fatal adverse outcomes, reflecting the most meaningful long-term risks for living donors. The eGFR threshold of 45 mL/min/1.73 m² corresponds to CKD stage 3b, a clinically relevant cutoff associated with increased cardiovascular risk and progression to end-stage kidney disease.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation or median (interquartile range), as appropriate. Categorical variables were expressed as counts and percentages. Comparisons between groups were performed using the chi-square test or Fisher's exact test for categorical variables, Student's t-test for continuous parametric variables, and the Mann-Whitney U test for continuous non-parametric variables. Binary logistic regression analysis was used to identify risk factors associated with the composite endpoint, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). Variables with a p-value <0.15 on univariate analysis were entered into a multivariate logistic regression model. Paired samples t-test was used to assess the mean eGFR change from baseline to last follow-up. A two-tailed p-value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 26 (IBM Corporation, Armonk, NY, USA).

Results

General Characteristics of the Cohort

The study included 142 living kidney donors with a mean age of 52.44 ± 11.27 years, of whom 39 (27.5%) were male. At the moment of kidney donation, donors had a mean BMI of 26.96 ± 4.99 kg/m², a mean fasting plasma glucose of 89.66± 18.09 mg/dl and a median plasma uric acid level of 4.82 (IQR: 3.88-5.40) mg/dl. Metabolic comorbidities at donation included hypertension (28.2%), dyslipidemia (34.5%), and obesity (12.7%), while 21.8% were active or former smokers. Overall, 57 donors (40.1%) had at least one comorbidity, and 17

(12.0%) had two or more comorbidities. Baseline mean serum creatinine was 0.86 ± 0.18 mg/dL and mean eGFR was 88.66 ± 18.44 mL/min/1.73 m². General characteristics of the cohort are presented in *Table 1.*

During a mean follow-up time of 100.88 ± 17.75 months, 24 donors (16.9%) reached the composite endpoint. Of these, 14.7% (n = 21) had an eGFR <45 mL/min/1.73 m² at last follow-up and 2.2% (n = 3) died (*Fig. 1*).

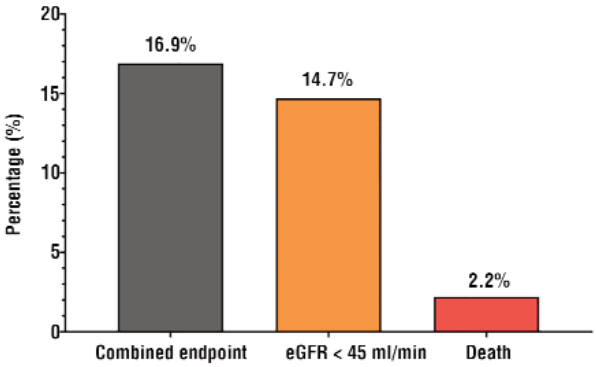

Figure 1. Distribution of composite endpoint components among 142 living kidney donors.

Risk Factors Associated with the Composite Endpoint

Donors who reached the composite endpoint were significantly older at donation (61.48 ± 6.72 vs. 50.68 ± 11.15 years, p < 0.001) and had lower baseline renal function, reflected by higher serum creatinine (0.98 ± 0.16 vs. 0.83 ± 0.17 mg/dL, p < 0.001) and lower eGFR (74.66 ± 16.13 vs. 91.51 ± 17.51 mL/min/1.73 m², p < 0.001). Also, dyslipidemia was significantly more prevalent among donors who experienced the composite endpoint (58.3% vs. 29.7%, p = 0.009). No significant differences were observed between donors who reached the endpoint and those who did not in terms of BMI (28.86 ± 4.10 vs. 26.47 ± 5.12 kg/m², p = 0.12), obesity (25% vs. 10.2%, p = 0.07), and hypertension (41.7% vs. 25.4%, p = 0.11). Likewise, fasting plasma glucose, uric acid levels, smoking status, and male sex were comparable between the two groups (*Table 1*).

Predictors of the Composite Endpoint

Variables with a p-value < 0.15 in the comparative analysis were included in the univariate logistic regression analysis model. On univariate logistic regression analysis, age at donation (OR 1.12, 95% CI: 1.06–1.20, p < 0.001), dyslipidemia at donation (OR 3.32, 95% CI: 1.34–8.18, p = 0.009), and eGFR at donation (OR 0.94, 95%: CI 0.91–0.97, p < 0.001) were identified as significant risk factors associated with the composite endpoint. Obesity at donation (OR 2.94, 95% CI: 0.98–8.84, p = 0.05) presented a trend of association with the composite endpoint, while hypertension at donation (OR 2.09, 95% CI: 0.84–5.21, p = 0.11) and BMI at donation were not found as risk factors for the endpoint (OR 1.09, 95% CI: 0.97–1.24, p = 0.13) (*Table 2*).

In the multivariate logistic regression model, which included age, dyslipidemia, obesity, hypertension, and eGFR at the moment of donation, only age at donation

Table 1. Baseline characteristics of the study cohort and comparison between donors who reached the composite endpoint and those who did not.

Variable	Entire cohort (n = 142)	Composite endpoint (n = 24)	Without composite endpoint (n = 118)	P value
Age at donation (years)	52.44 ± 11.27	61.48 ± 6.72	50.68 ± 11.15	< 0.001
Male sex, n (%)	39 (27.5)	7 (29.2)	32 (27.1)	0.80
Fasting glucose (mg/dL)	89.66 ± 18.09	89.05 ± 14.99	89.79 ± 19.76	0.87
Uric acid (mg/dL)*	4.82 (3.88–5.40)	5.00 (3.75–5.88)	4.80 (3.90–5.40)	0.62
BMI (kg/m²)	26.96 ± 4.99	28.86 ± 4.10	26.47 ± 5.12	0.12
Active/former smoking, n (%)	31 (21.8)	5 (20.8)	26 (22.0)	1.00
Obesity, n (%)	18 (12.7)	6 (25.0)	12 (10.2)	0.07
Hypertension, n (%)	40 (28.2)	10 (41.7)	30 (25.4)	0.11
Dyslipidemia, n (%)	49 (34.5)	14 (58.3)	35 (29.7)	0.009
sCr at donation (mg/dL)	0.86 ± 0.18	0.98 ± 0.16	0.83 ± 0.17	< 0.001
eGFR at donation (mL/min/1.73 m²)	88.66 ± 18.44	74.66 ± 16.13	91.51 ± 17.51	< 0.001
Any comorbidity, n (%)	57 (40.1)	12 (50.0)	45 (38.1)	0.28
One comorbidity	40 (28.1)	7 (29.2)	33 (28.0)	0.34
Two or more comorbidities	17 (12.0)	5 (20.8)	12 (10.2)	

Values are mean ± SD unless otherwise indicated. *Median (interquartile range). BMI = body mass index; sCr = serum creatinine; eGFR = estimated glomerular filtration rate.

Table 2. Univariate and multivariate logistic regression analysis of predictors for the composite endpoint.

	Univariate logistic regression analysis			Multivariate logistic regression analysis		
	OR	**95%CI**	**P value**	**OR**	**95%CI**	**P value**
Age at donation	1.12	1.06- 1.20	<0.001	1.11	1.02- 1.21	0.01
BMI at donation	1.09	0.97- 1.24	0.13	1.07	0.82- 1.40	0.57
Obesity	2.94	0.98- 8.84	0.05	1.29	0.11- 15.30	0.83
HTN	2.09	0.84- 5.21	0.11	0.86	0.16- 4.39	0.85
Dyslipidemia	3.32	1.34- 8.18	0.009	1.60	0.34- 7.44	0.54
eGFR at donation	0.94	0.91- 0.97	<0.001	0.95	0.90- 1.00	0.07

OR= odds ration; CI= confidence interval; BMI= body mass index; HTN= hypertension; eGFR= estimated glomerular filtration rate;

was found as an independent predictor associated with the composite endpoint (OR 1.11, 95% CI: 1.02–1.21, p = 0.01). eGFR at donation showed only a trend of association with the composite endpoint(OR 0.95, 95% CI: 0.90–1.00, p = 0.07) (*Table 2*).

Change in Renal Function After Donation

During a mean follow-up time of 100.88 ± 17.75 months, we observed a significant decline in renal function, with a mean decrease in eGFR of −27.37 ± 17.80 mL/min/1.73 m² (95% CI: −30.33 to −24.42, p < 0.001), corresponding to a mean annual decline of 3.3 mL/min/1.73 m² per year (*Fig. 2*). This decline is consistent with the expected physiological reduction following unilateral nephrectomy, reflecting both the immediate loss of nephron mass and the variable degree of compensatory adaptation in the remaining kidney. Notably, no donor developed an eGFR <15 mL/min/1.73 m² or required renal replacement therapy at last follow-up, and only two donors had an eGFR ≤30 mL/min/1.73 m².

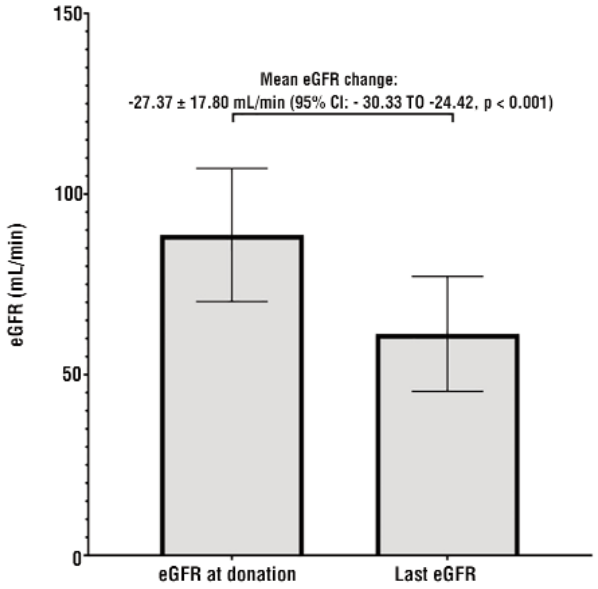

Figure 2. Mean eGFR evolution from donation to last follow-up.

Discussion

The central finding of this study is that preoperative metabolic comorbidities, including obesity, hypertension, and dyslipidemia, were not independent predictors of the composite endpoint of death or eGFR <45 mL/min/1.73 m² in living kidney donors over a mean follow-up of approximately 8.4 years. Although several of these conditions were associated with adverse outcomes in univariate analyses, none remained significant after adjustment for age and baseline renal function. These findings have important implications for the evaluation and selection of living kidney donor candidates. Older donors are inherently more likely to have accumulated metabolic comorbidities, and the effect attributed to these conditions on univariate analysis appears to be substantially mediated through the age variable. This does not imply that metabolic comorbidities are biologically irrelevant, but rather that in the context of a carefully selected donor population, their incremental contribution to risk beyond that conferred by age is not statistically demonstrable (9).

The association between dyslipidemia and the composite endpoint on univariate analysis (OR 3.32, p = 0.009) was the strongest among the metabolic variables examined. Dyslipidemia prevalence increases markedly with age, and in a cohort where the composite endpoint group was on average a decade older (61.48 vs. 50.68 years), the observed association likely reflects the overall cardiometabolic burden that accompanies aging rather than an independent nephrotoxic effect of dyslipidemia per se (9).

Obesity, defined as BMI ≥30 kg/m², showed a borderline association with the composite endpoint on univariate analysis (OR 2.94, p = 0.05). The prevalence

of obesity in our cohort was relatively modest (12.7%), which may have limited statistical power to detect a true effect. Nevertheless, the finding that obesity did not independently predict adverse outcomes aligns with several large registry-based studies that have reported acceptable long-term outcomes in obese donors, particularly when BMI falls within the class I obesity range (10,11,12). Severely obese donors (BMI >35 kg/m²) were excluded from donation in our cohort, consistent with prevailing international practice.

Hypertension was present in 28.2% of donors and was more prevalent among those who reached the composite endpoint (41.7% vs. 25.4%). The acceptance of donors with well-controlled hypertension has been one of the most debated aspects of expanded donor criteria (13). Our data suggests that, within the context of a structured selection and follow-up practices, mild hypertension at the time of donation does not independently predispose to clinically significant renal deterioration. This finding is consistent with the existing literature, which has generally shown that well-controlled essential hypertension, particularly when managed with a single antihypertensive agent, is not an absolute contraindication to living kidney donation (14,15).

The mean eGFR decline of 27.37 ± 17.80 mL/min/1.73 m² observed in our cohort is consistent with the well-characterized physiological response to unilateral nephrectomy (7). The annual rate of eGFR decline (3.3 mL/min per year) is within the expected range when the initial surgical loss of nephron mass is averaged over the follow-up period. Importantly, no donor required renal replacement therapy, and only two donors had an eGFR ≤30 mL/min/1.73 m² at last follow-up. This physiological adaptation is distinct from pathological CKD progression, and the absolute eGFR values attained by most donors remained within clinically acceptable ranges. Baseline eGFR at the time of donation showed a borderline association with the composite endpoint on multivariate analysis (OR 0.95, p = 0.07). Donors with lower baseline renal function have reduced physiological reserve after nephrectomy and are more likely to reach clinically significant eGFR thresholds. This underscores the importance of baseline eGFR in donor risk assessment and highlight the need for particularly careful evaluation of individuals with borderline renal function (16).

It is essential to contextualize these findings within the broader framework of donor selection. Our cohort represents a population that has already undergone rigorous preoperative screening. Donors with severe or uncontrolled comorbidities were excluded from donation at the outset. Therefore, the absence of a significant independent effect of metabolic comorbidities should not be interpreted as evidence that these conditions are universally benign in the context of nephrectomy, but rather that they do not confer excess risk in appropriately selected and monitored donors. This distinction is critical for clinical practice: the finding supports the current approach of individualized risk assessment rather than categorical exclusion based on the mere presence of a metabolic condition (4).

Several limitations of this study should be acknowledged. First, the retrospective design introduces the possibility of selection bias. Second, the sample size of 142 donors, with only 24 reaching the composite endpoint, limits statistical power to detect modest effect sizes and precludes more granular subgroup analyses, thereby increasing the risk of type II error. Third, the study is from a single center, and results may not be generalizable to populations with different demographic characteristics or selection criteria. Fourth, lack of information regarding causes of death, which precludes a more detailed assessment of cause-specific mortality. However, all-cause mortality was used as it is less prone to misclassification bias and is recommended for composite outcomes in observational studies. Fifth, the inability to perform time-to-event analyses due to the lack of exact dates of death for all participants. Consequently, mortality was treated as a binary outcome rather than a time-dependent event. Therefore, logistic regression was used for the analysis of the composite endpoint, which does not incorporate variation in follow-up time and may be less informative than survival models in settings with precise temporal data.

Conclusions

In conclusion, age at donation was the only independent predictor of the composite endpoint of decline in renal function or mortality after donation. In this cohort of living kidney donors, preoperative metabolic comorbidities were not independently associated with the composite outcome. Careful pre-donation evaluation and individualized risk stratification remain essential in candidate selection for living kidney donation. These findings support the current paradigm of individualized donor selection, integrating age with the type and degree of control of metabolic comorbidities, in order to reduce post-donation risk and optimize graft outcomes. Prospective, multicenter studies with longer follow-up are warranted to further refine risk stratification strategies and confirm the long-term safety of donation in this evolving donor population.

Authors' Contributions

LGD, CB and BMS contributed for conceptualization. LGD, IFM and TC contributed for graphics and design. LGD, OM and IS supervised the research. LGD, DA and AP reviewed the manuscript. All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Ethical Statement

This study was conducted in accordance with the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of Fundeni Clinical Institute (date of approval April 14, 2025, protocol no. 15380). All participants provided written informed consent prior to inclusion in the study.

Data Availability Statement

The data that support the findings of this study are not publicly available due to privacy and ethical restrictions but are available from the corresponding author upon reasonable request and with appropriate institutional approval.

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