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Outcomes of living related kidney donors of recipients with familial mediterranean fever-associated Amyloid A Amyloidosis

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Summary

Background Living kidney donation among carefully selected individuals is generally considered safe but post-donation health risks vary by factors including family history. Data on long-term outcomes of related living kidney donors of recipients with familial Mediterranean fever (FMFLRD) are limited.

Methods This retrospective multicenter study reviewed the medical records of living donors who donated between 2010 and 2021 across six transplantation centers. Cardiovascular and kidney outcomes were compared between 37 FMFLRD and a propensity-matched control group of 74 donors to recipients without FMF (non-FMFLRD). Propensity score matching was based on donor age, sex, and follow-up time. Outcome measures included major cardiac events (MACE), new-onset hypertension, new-onset diabetes mellitus, proteinuria and estimated glomerular filtration rate (eGFR).

Results The median follow-up duration of all donors was 4.0 years (interquartile range, IQR 2.0–11.0 years). The mean age of the FMFLRD group was 48.9 ± 9.3 years, The FMFLRD group showed a significantly lower proportion of MACE (2.7% vs. 17.6%, $p = 0.026$) and new-onset hypertension (13.5% vs. 39.2%, $p = 0.004$) and a trend towards a lower proportion of new-onset diabetes mellitus (2.7% vs. 14.9%, $p = 0.052$) compared to the non-FMFLRD group. There were no significant differences in long-term kidney function, as evidenced by similar eGFR and proteinuria levels between the groups.

Conclusion The FMFLRD group showed similar kidney outcomes and lower rates of cardiovascular and metabolic complications during follow-up compared with matched non-FMFLRD; however, given the retrospective design and relatively short follow-up duration, long-term donor safety remains uncertain and the results should be interpreted cautiously.

Previous presentation Not previously presented.

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Introduction

Familial Mediterranean fever (FMF) is a hereditary periodic fever syndrome characterized by recurrent episodes of polyserositis. The most severe consequence of FMF is Amyloid A (AA) amyloidosis, where amyloid deposits can accumulate in various organs, such as the heart, blood vessels, spleen, liver, gut and kidneys [1, 2]. Kidney involvement is particularly prevalent and often leads to kidney failure [3, 4]. Among affected populations, FMF is most frequently observed in the Ashkenazi Jewish, Turkish, Arabic and Armenian communities, significantly contributing to end-stage kidney disease (ESKD) in these populations [5–7]. Despite the elevated mortality risk associated with AA amyloidosis, kidney transplantation continues to provide a substantial survival benefit compared to dialysis [8, 9].

In regions where FMF is endemic, heterozygous carriage of Mediterranean Fever gene variant (*MEFV)* variants is common. Population-based and regional studies from Türkiye and neighboring Mediterranean/Middle Eastern cohorts report carrier frequencies around 20 %, underscoring the likelihood that potential living related donors of FMF recipients can themselves be *MEFV* variant carriers [10]. Importantly, several investigations suggest that even asymptomatic heterozygous carriers can display low-grade systemic inflammation or metabolic alterations, raising a biologically plausible concern that shared genetic background could influence long-term health trajectories after donation [11–13].

Living donor kidney transplantation (LDKT) is particularly favored as a treatment option for patients with ESKD, offering a lower risk of rejection, improved allograft function and a longer life expectancy compared to deceased donor kidney transplantation [14]. Nonetheless, in LDKT, the risk of renal complications developing in donors due to shared genetic predispositions requires careful predonation evaluation and long-term monitoring to ensure donor safety. [15–17].

It is crucial to assess prospective related donors for the potential presence of the same hereditary disease, due to their inherent genetic predisposition, before granting donor candidacy approval [15–17]. Nevertheless, technical and logistical limitations may sometimes impede these assessments. Additionally, shortages in the organ pool may necessitate the utilization of donations from related individuals, further complicating the decision-making process.

Donor nephrectomy introduces potential risks both in the short-term complications, such as surgical and infectious issues, and the long-term complications, such as hypertension and cardiovascular and metabolic diseases [18]. Kidney donors can face a small but statistically significantly increased relative risk of ESKD, estimated to be 8–11 times higher compared to healthy nondonors [19, 20]. Additionally, many living donors, often first or second-degree relatives of the recipients, are at an elevated risk of developing ESKD, even in the absence of known genetic diseases within the family [19, 21]. This underscores the complexity of donor-related risks and emphasizes the necessity of a thorough assessment to protect the health of potential donors.

Despite being clinically healthy at the time of donation, related donors can still share genetic susceptibility and potential long-term risk for renal or cardiovascular diseases. Data on the long-term follow-up kidney and cardiovascular outcomes of living related donors for kidney transplant recipients with FMF (FMFLRD) is scarce. Therefore, the aim of this retrospective cohort study was to evaluate the long-term renal and cardiovascular outcomes of FMFLRD and to compare these outcomes with those of a matched control group of other kidney disease donors (non-FMFLRD).

Material and methods

Study cohort and design

We reviewed the medical records in this retrospective multicenter study, including LDKT for FMF and non-FMF kidney diseases. Records from six transplantation centers between 2010 and 2021 identified living donors (LD) and recipients. The study group (FMFLRD) included related LDs whose recipients had a diagnosis of FMF complicated by AA amyloidosis, confirmed by native kidney biopsy. An FMF was diagnosed based on family history and/or clinical findings, with or without genetic confirmation. Genetic testing was performed when available, and all available genetic results were included in the analysis.

It is important to emphasize that the donors included in this study did not have clinical FMF or systemic AA amyloidosis. All donors underwent standard living kidney donor evaluation according to institutional and national transplantation guidelines, including medical history, physical examination, blood pressure measurement, laboratory testing, urine analysis, assessment of kidney function, and cardiovascular evaluation. Donors with clinical FMF, proteinuria, reduced kidney function, uncontrolled hypertension, diabetes mellitus, cardiovascular disease, or any evidence of systemic amyloidosis or organ involvement were not accepted as donors. Accordingly, all donors included in this study were considered clinically healthy individuals without FMF or systemic inflammatory disease and had no indications for anti-inflammatory therapy at the time of donation. The control group (non-FMFLRD) consisted of living kidney donors of recipients without FMF who donated during the same study period.

These donors also underwent standard living kidney donor evaluation and were considered clinically suitable for donation according to institutional donor selection criteria. Postdonation outcomes, such as hypertension, diabetes mellitus and cardiovascular events were recorded during follow-up. We examined donor profiles including demographic characteristics, kidney function tests, lipid parameters, body mass index, history of hypertension, diabetes mellitus, cardiovascular disease, and hospitalizations. Because follow-up duration varied among participants, outcomes were reported as cumulative proportions rather than incidence rates based on person-time. Baseline demographic and clinical data, as well as follow-up outcomes, were collected from medical records, including both hard-copy charts and electronic health records. The ethical standards described in the Declaration of Helsinki were adhered to in the review. Approval was received from the ethical committee at the center of the first author (approval number: 727087). All LDs who could be reached during routine follow-up or via telephone were included in the study after written or verbal informed consent was obtained.

Propensity score matching

The LD databases of the participating transplantation centers were used to construct a cohort of control donors without a family history of FMF. Propensity scores were estimated using a logistic regression model including donor sex, age at donation, and duration of follow-up as covariates. Propensity score matching was performed in R (R Foundation for Statistical Computing, Vienna, Austria) using the MatchIt package, applying nearest-neighbor matching in a 1:2 ratio without replacement. No caliper was specified; therefore, each donor in the FMF group was matched to up to two control donors with the closest estimated propensity score. The propensity score was treated as a continuous variable, and post-matching balance was evaluated using standardized mean differences.

Study outcomes

Living donors (LDs) were monitored postdonation for kidney function, including estimated glomerular filtration rate (eGFR), proteinuria, new-onset hypertension, and major adverse cardiac events (MACE), during follow-up. Hypertension, diabetes mellitus, and cardiovascular events reported in this study represent postdonation outcomes observed during follow-up and were not present at the time of donor evaluation. The primary endpoint was MACE defined as a composite of myocardial infarction, stroke, hospitalization due to heart failure and revascularization. Secondary outcomes included new-onset hypertension, new-onset diabetes mellitus and assessments of eGFR and proteinuria at the last follow-up visit. Hypertension was defined as a sustained blood pressure $\geq 140/90$ mm Hg or the initiation of antihypertensive therapy. The ascertainment of hypertension and diabetes mellitus in this study was based on physician documentation, medication records, or persistently abnormal measurements on at least two occasions. The eGFR was calculated using the CKD-EPI 2021 equation [22], and proteinuria was measured using the urine protein-to-creatinine ratio (UPCR) in the first morning specimens.

Statistical analysis

Statistical analyses were performed using SPSS for Windows (version 25.0; IBM Corp., Armonk, NY, USA). Data are presented as mean ± standard deviation (SD) for normally distributed variables, and as median with interquartile range (IQR) for non-normally dis-

Table 1 Various characteristics of living donors for recipients with versus without familial Mediterranean fever

Characteristics	FMFLRD ($n=37$)	Non-FMFLRD ($n=74$)	*p*-value
Age at donation (years), mean ± SD	48.9 ± 9.3	49.1 ± 10.8	0.923
Sex (male/female), *n* (%)	18 (48.6)/19 (51.4)	31 (41.9)/43 (58.1)	0.499
Total cholesterol, mg/dl, mean ± SD	184.1 ± 49.4	189.4 ± 39.9	0.322
HDL, mg/dl, mean ± SD	47.8 ± 16.1	47.2 ± 13.4	0.883
LDL, mg/dl, mean ± SD	96.5 ± 35.7	118.7 ± 33.4	0.015
Triglyceride, mg/dl, mean ± SD	150.0 ± 58.2	148.8 ± 44.2	0.080
Body mass index, kg/m^2, mean ± SD	20.7 ± 3.9	22.0 ± 1.8	0.638
Donor's relationship to recipient, *n* (%)			
Father	12 (32.4)	13 (17.6)	0.115
Mother	12 (32.4)	25 (33.8)	
Sibling	9 (24.3)	12 (16.2)	
Cousin	3 (8.1)	7 (9.5)	
Others	1 (2.7)	11 (14.8)	
LURD (spouse)	0 (0.0)	6 (16.2)	
Etiology of ESKD in recipients, *n* (%)			
HT	–	21 (28.4)	–
DM	–	12 (16.2)	–
CGN	–	9 (12.2)	–
ADPKD	–	1 (1.4)	–
CAKUT	–	7 (9.5)	
Others	–	5 (6.8)	–
Unknown	–	19 (17.1)	–
Donors' causal variants			
NM_000243.3(MEFV): c.2080A > G (p.Met694Val)	8 (21.6)	–	–
Genetic test not performed	29 (88.4)	–	–

FMFLRD living related donors for recipients with familial Mediterranean fever, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein, *LURD* living unrelated donor, *NA* not available, *HT* hypertension, *DM* diabetes mellitus, *ADPKD* autosomal dominant polycystic kidney disease, *CGN* chronic glomerulonephritis, *CAKUT* congenital anomalies of the kidney and urinary tract, *SD* standard deviation, *IQR* interquartile range

Table 2 Genetic test results of the recipients and the related living donors and outcomes of the donors

Family		Age at KT (years)/sex	Gene variant	Zygosity	ACMG classification	Donor HTN	Donor DM	Donor MACE	Donor Death	Pre-KT eGFR (ml/min/1.73 m^2)	Post-KT eGFR (mL/min/1.73 m^2)	Pre-KT proteinuria (g/g)	Post-KT proteinuria (g/g)
Family 1	Donor (father)	40/M	M694V	Het	P/LP	No	No	No	No	95	68	0.1	0.1
Family 2	Donor (mother)	40/M	M694V	Het	P/LP	No	No	No	No	90	77	0.1	0.1
Family 3	Donor (sibling)	36/F	M694V	Het	P/LP	No	No	No	No	111	58	0.1	0.1
Family 4	Donor (sibling)	45/F	M694V	Het	P/LP	No	No	No	No	95	88	0.1	0.1
Family 5	Donor (father)	58/M	M694V	Het	P/LP	Yes	No	No	No	108	64	0.1	0.2
Family 6	Donor (father)	63/M	M694V	Het	P/LP	No	No	No	No	108	51	0.1	0.2
Family 7	Donor (mother)	59/F	M694V	Het	P/LP	Yes	No	No	No	120	81	0.1	0.1
Family 8	Donor (father)	48/M	M694V R202Q	Het Het	P/LP Benign	No	No	No	No	100	82	0.1	0.1

ACMG American College of Medical Genetics and Genomics, *DM* diabetes mellitus, *eGFR* estimated glomerular filtration rate, *F* female, *Het* heterozygous, *HTN* hypertension, *KT* kidney transplantation, *LP* likely pathogenic, *M* male, *MACE* major cardiac events, *P* pathogenic

tributed variables. Parametric or nonparametric tests were applied based on data distribution. Continuous variables were compared using the independent samples t-test or the Mann-Whitney U test, as appropriate. Categorical variables were analyzed using the χ^2-test or Fisher's exact test. All statistical tests were two-sided, and a p-value of ≤ 0.05 was considered statistically significant.

Results

Donor characteristics and outcomes

The median follow-up duration of all donors was 4.0 years (IQR 2.0–11.0 years). The FMFLRD cohort comprised 37 related living donors, with a mean age of 48.9 ± 9.3 years at the time of donation. A control group of 74 non-FMFLRD donors was included for comparison. Baseline demographic and genetic characteristics of both groups are summarized in Table 1. At the time of donation, all donors had 24-h creatinine clearance values exceeding 80 ml/min/1.73 m^2, and none exhibited microscopic hematuria, proteinuria, or any documented history of hypertension, valvular heart disease, coronary revascularization, heart failure exceeding New York Heart Association (NYHA) class II, arrhythmia, stroke, transient ischemic attack, active infection, or overt noninfectious inflammation, as indicated by elevated acute phase reactants.

The FMFLRD group included 37 related living donors. The donor-recipient relationship was mother (12 donors, 32.4 %), father (12, 32.4 %), sibling (9, 24.3 %), cousin (3, 8.1 %), and other relatives (1, 2.7 %). All donors were European. There were no significant differences between the FMFLRD and non-FMFLRD groups in terms of lipid profiles or body mass index. In the non-FMFLRD group, the most common primary causes of ESKD in recipients were hypertension (21 recipients, 28.4 %) and diabetes mellitus (12 re-

Table 3 Long-term outcomes of the study groups

Outcomes	FMFLRD (n = 37)	Non-FMFLRD (n = 74)	p
Follow-up time, years, median, (IQR 25–75)	3.0 (1.0–11.5)	5.0 (2.0–10.3)	0.109
Serum creatinine at last follow-up (mg/dl), mean ± SD	1.1 ± 0.2	1.0 ± 0.2	0.183
eGFR at last follow-up (ml/min/1.73 m^2), mean ± SD	72 ± 13.9	77.8 ± 16.2	0.119
Proteinuria at last follow-up (g/g), median (IQR)	0.1 (0.1–0.1)	0.9 (0.1–0.6)	0.792
Hypertension after donation, *n* (%)	5 (13.5)	29 (39.2)	0.004
Diabetes mellitus after donation, *n* (%)	1 (2.7)	11 (14.9)	0.052
Major cardiac events, *n* (%)	1 (2.7)	13 (17.6)	0.026
Acute coronary ischemia	–	7	
Congestive heart failure	–	2	
Arrhythmia	1	1	
Stroke	–	3	

eGFR estimated glomerular filtration rate, *FMFLRD* living related donors for recipients with familial Mediterranean fever, *IQR* interquartile range, *SD* standard deviation

cipients, 16.2%). Genetic testing was performed in 8 related donors after donation, all of whom were found to carry heterozygous pathogenic or likely pathogenic variants in the *MEFV* gene (Table 2).

Long-term outcomes were assessed in 37 FMFLRDs and 74 non-FMFLRDs. During follow-up, MACE occurred in 1 donor (2.7%) in the FMFLRD group (arrhythmia), compared to 13 donors (17.6%) in the non-FMFLRD group, including acute coronary ischemia ($n=7$), congestive heart failure ($n=2$), arrhythmia ($n=1$), and stroke ($n=3$) ($p=0.026$) (Table 3).

Postdonation hypertension developed significantly more frequently in non-FMFLRD group than in FMFLRD group (29 , 39.2% vs. 5, 13.5%, $p=0.004$). New-onset diabetes mellitus was observed in 1 FMFLRD (2.7%) and 11 non-FMFLRD donors (14.9%), with a trend toward statistical significance ($p=0.052$) (Table 3).

None of the donors in either group progressed to ESKD (eGFR < 15 ml/min/1.73 m^2 or need for dialysis) during follow-up. Kidney function at last follow-up was comparable between the groups. In the FMFLRD group, mean serum creatinine was 1.1 ± 0.2 mg/dL, mean eGFR was 72 ± 13.9 ml/min/1.73 m^2, and median proteinuria was 0.1 g/g (IQR, 0.1–0.1g/g), showing no significant differences from the non-FMFLRD group ($p=0.183$, $p=0.119$, and $p=0.792$, respectively) (Table 3).

Discussion

Our study investigating the long-term outcomes of living related kidney donors in the context of FMF revealed interesting and unexpected findings. The FMFLRD group exhibited a significantly lower proportion of major adverse cardiac events (2.7% vs. 17.6%, $p=0.026$) and new-onset hypertension (13.5% vs. 39.2%, $p=0.004$) compared to the non-FMFLRD group. The overall cardiovascular risk profile appeared markedly more favorable in the FMFLRD group; however, this observation should be interpreted cautiously. This difference might reflect selection bias, as potential FMFLRDs are often younger, more health-conscious, and undergo extensive medical evaluation due to their familial relation to patients with a known genetic disorder.

Additionally, new-onset diabetes mellitus occurred more frequently in the non-FMFLRD group (14.9% vs. 2.7%), with the difference approaching statistical significance ($p=0.052$), further suggesting a divergence in cardiometabolic risk burden between the two groups.

Despite the benefits provided by the recipient and the meticulous evaluations performed in kidney donor evaluation, some degree of risk exists for kidney donors. There is a relationship between living kidney donation and an increased risk of hypertension. In a large longitudinal cohort of over 3700 donors, postdonation hypertension developed in approximately 27% of individuals over a mean follow-up of 16.6 years, without a concomitant increase in cardiovascular or mortality risk [23, 24].

In our cohort, the elevated rates of hypertension, diabetes mellitus and MACE in the non-FMFLRD group can be attributed to an increased baseline susceptibility to cardiovascular and metabolic disease within these families. Notably, hypertension and diabetes mellitus were the most common causes of ESKD among recipients in the non-FMFLRD group, accounting for 28.4% and 16.2% of cases, respectively. Additionally, FMF families may receive more frequent medical surveillance due to chronic disease management of affected relatives, which could lead to earlier detection and intervention of comorbid conditions, thereby conferring an apparent protective effect. A survivorship bias may also exist, as only relatives who remained healthy and eligible for donation were included, potentially representing a resilient subset of FMF families.

Another important observation is that in both groups eGFR and proteinuria levels were similar during postdonation follow-up. There are limited data on long-term kidney outcomes of FMFLRDs in the literature. In a retrospective study comparing the mid-term and long-term outcomes of 12 donors carrying the *MEFV* gene variants with 24 age-matched and sex-matched healthy donors of non-FMF kidney donors as control subjects, there was no difference in the GFR values of both groups [25]. This study reported an increasing proteinuria trend in donors carrying *MEFV* gene variants associated with FMF but it did not provide data on hypertension or cardiovascular events.

The finding that postdonation eGFR levels were comparable to those of non-FMFLRDs suggests no increased risk of kidney function decline in FMFLRDs during the follow-up period and may alleviate concerns regarding genetic predisposition in this group.

In our study, FMFLRDs were clinically healthy related donors, some of whom were *MEFV* gene variant carriers, but none had clinical FMF or systemic amyloidosis at the time of donation; however, given the limited number of donors who underwent genetic analysis, these results should be interpreted with caution. We believe that genetic testing should be considered in all FMFLRD candidates and donor candidates who carry high-risk mutations or share the same pathogenic variants with the recipient should be evaluated carefully during donor selection.

Subclinical amyloid deposition could not be assessed in this retrospective study. In our donor evaluation protocol, routine kidney biopsy at the time of donor nephrectomy and cardiac imaging specifically for amyloid deposition were not performed in clinically healthy donors. Therefore, the presence of subclinical renal or cardiac amyloid deposition could not be excluded. Given that amyloidosis may develop insidiously in FMF patients and possibly in heterozy-

gous carriers, its potential influence on long-term donor outcomes warrants future prospective studies.

The major strengths of our study are that it is multicentric and includes changes in kidney function (eGFR and proteinuria) and cardiovascular outcomes such as hypertension, and MACE. Additionally, these results were compared to a propensity-matched control group; however, several limitations should be acknowledged. The retrospective nature of the study, limited sample size, and incomplete genetic screening among FMFLRDs could restrict the generalizability of the findings. Serial measurements of serum amyloid A were not systematically available, and amyloid burden was not assessed using whole-body serum amyloid P component scintigraphy, which may have limited precise evaluation of subclinical inflammation and amyloid deposition. The median follow-up of 4 years is insufficient to determine lifetime donor safety, particularly in relatively young individuals. Late renal or cardiovascular complications can emerge decades after donation [26] and therefore long-term risk cannot be excluded. Our conclusions are limited to early-term and mid-term outcomes.

The genotype-phenotype relationship among related donors is also of scientific interest; however, comprehensive longitudinal genetic and inflammatory data were not uniformly available in this retrospective cohort, limiting detailed subgroup analyses.

These findings should also be interpreted in light of potential selection, surveillance and survivorship biases as well as unmeasured confounders such as subclinical amyloidosis. Further large-scale prospective studies incorporating genetic and inflammatory profiling are warranted for better elucidation of the biological and environmental mechanisms underlying these observations.

In conclusion, carefully selected FMFLRDs who were clinically healthy at the time of donation showed similar kidney outcomes and no excess early-term and mid-term cardiovascular risk compared with matched donors; however, due to the retrospective design and relatively short follow-up duration, long-term donor safety remains uncertain and these findings should be interpreted cautiously rather than as a general recommendation for donation in this population.

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Declarations

Conflict of interest Outside the submitted work, S. Mirioglu received support for meeting registration and travel from Amgen and Sanofi Genzyme. O.A. Oto, T.E. Ozler, A. Velioglu, D.B. Atas, G. Kumru, A. Yildiz, S. Ozkurt, R.H. Hocaoglu, Y. Tahillioglu, M. Doksan, V. Suleymanova, Z. Celebi, A.S. Artan, A.B. Dirim, H. Yazici, K.L. Lentine and Y. Caliskan declare that they have no competing interests.

Informed consent All participants agreed to participate by signing the corresponding informed consent form.

Ethical standards The study was authorized by Istanbul University Ethical Committee (approval number: 727087) and was conducted in accordance with the ethical principles of the Declaration of Helsinki and data protection regulations.

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