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# A prospective study of living kidney donors: 6 years follow-up from a cardiovascular disease risk perspective

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## SUMMARY

**OBJECTIVE:** The purpose of this prospective study was to evaluate the clinical, laboratory, and donation-specific outcomes of living kidney donors 6 years after donation.

**METHODS:** We included a total of 93 kidney donors and 54 age- and sex-matched individuals as control group through a type 2 cohort consecutive recruitment. We detected kidney function abnormalities and the presence of hypertension, diabetes, and cardiovascular events during the 6 years follow-up period.

**RESULTS:** The mean serum creatinine levels were higher (p<0.001), and the estimated glomerular filtration rate levels were lower (p<0.001) in living kidney donors 6 years after donation when compared with controls. The protein/creatinine ratio of the study population was also higher (p=0.014). There was no difference in outcomes between the groups for end-stage kidney disease and cardiovascular mortality. A higher rate of new-onset hypertension (6.4 vs. 32.9%), diabetes mellitus (0.0 vs. 4.3%), chronic kidney disease (0.0 vs. 2.1%), and cardiovascular disease (0.0 vs. 2.1%) was demonstrated among donors 6 years after donation (p<0.001, respectively).

**CONCLUSION:** Our data have demonstrated that the reduction in Glomerular filtration rate induced by kidney donation might cause an increase in adverse renal and cardiovascular events.

**KEYWORDS:** Living donors. Hypertension. Cardiovascular Abnormalities. Diabetes Mellitus.

## INTRODUCTION

Living donor kidney transplantation is the preferred treatment for end-stage kidney disease (ESKD), mainly because it improves graft and patient survival and quality of life when compared with the transplantation from a deceased donor and waiting list patients who remain on dialysis[1]. Turkey is among the countries with the most living donor transplants per million population. According to the 2020 Turkey Registry System Report, 2250 (90%) of 2500 kidney transplantations performed in 2020 are living donor transplantations[2]. Each year, over 27,000 people around the world become kidney donors, and this number is increasing in response to a shortage of kidneys for transplantation from deceased donors[3]. However, the mid- and long-term cardiovascular and metabolic risks among donors remain uncertain.

A number of studies suggest that the risk of developing ESKD in donors is similar to that of the general population[4] Some studies have suggested that there are small but measurable increases in the risk of HT, proteinuria, preeclampsia, gout, acute dialysis, and ESKD after donor nephrectomy, in addition to the risks of surgery[5,6]. These factors are associated with an increased risk for cardiovascular and all-cause mortality in the general population. Multiple studies have shown no evidence of reduced survival among living kidney donors as compared with the general population. In contrast, Mjoen et al. evaluated the long-term kidney function and cardiovascular and all-cause mortality over a 15-year follow-up period and found that all-cause death, cardiovascular death, and ESKD were significantly increased in donors after about 10 years[7].

In this study, we aimed to demonstrate the renal consequences of donation and the evidence of the effects of donation on the cardiovascular system in 93 living kidney donors after 6 years from donation and in 54 age- and sex-matched controls.

## METHODS

### Patients

We performed a type 2 cohort study to collect the data on the health status of kidney donors who had the transplantation operation in Kartal Training Hospital. Between January 2011

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and August 2014, a total of 157 living donor nephrectomy was performed in our transplantation center. We made phone calls to invite kidney donors to participate in the study. We could not make contact with 30 donors, and 93 of the remaining 127 donors accepted to participate in the study. The demographic characteristics of the study population at the time of donation were extracted from the medical records, which also included a detailed medical history. After 6 years of follow-up, demographic, clinical, and laboratory characteristics of kidney donors were updated. In addition, a control group was formed with 54 age- and sex-matched individuals who were selected based on self-reported medical history to fit the donor demographic and baseline characteristics. Clinical and laboratory characteristics of controls were also updated after 6 years from their baseline.

## Definitions

### *Hypertension*

Kidney donors and controls were defined as hypertensive if they had a previously known diagnosis of HT (treated or not) or if the office BP was measured >140/90 mmHg[8].

### *Diabetes*

Diabetes was defined as fasting plasma glucose levels >126 mg/dL (7.0 mmol/L) or hemoglobin A1c (HbA1c) level >6.5% (48 mmol/mol) or in a patient with classic symptoms of hyperglycemia, a random plasma glucose ≥200 mg/dL (11.1 mmol/L)[9].

All analyses of the donors were performed at the time of donation and 6 years after donation, and all analyses of the control group were performed at baseline and after 6 years of follow-up in the biochemistry laboratory of Kartal Training Hospital. Chronic disease was defined as the presence of HT, DM, coronary artery disease (CAD) (defined as myocardial infarction, percutaneous coronary intervention, and coronary artery bypass surgery), CKD (GFR <60 mL/min/1.73 $m^2$), and ESKD.

### *Laboratory data*

The blood examinations including serum creatinine, urea, glucose, HbA1c, lipid fractions, uric acid, ferritin, and parathormone (PTH) were conducted following overnight fasting. Microalbuminuria was defined as the presence of 30–300 mg/g of creatinine, and proteinuria was defined as the presence of >300 mg/g of creatinine[10]. Urine albumin and protein excretions were determined in the first-morning urine sample.

### *Statistical analysis*

Descriptive data were presented as mean ± standard deviation (SD), median and interquartile range (IQR) for the continuous variables, and frequency and percentages (%) for the categorical variables. Continuous variables were evaluated for normality distribution using the Shapiro-Wilk test. T-test for normally distributed variables and Mann-Whitney U test for non-normally distributed variables were used in comparison with 2 independent groups. Categorical variables were compared by using the chi-square or Fisher's exact test for proportion. All significance tests were two-tailed, and p-values <0.05 were considered statistically significant. All statistical analyses were performed by the SPSS software version 21 (Chicago, IL). Reporting of the study conforms to the STROBE statement along with references to the STROBE statement and the broader EQUATOR guidelines[11].

### *Ethics*

Ethics committee approval for the study was obtained from the Ethical Review Board of Kartal Training Hospital (514/193/5-01.13.2021). All procedures were performed in accordance with the ethical standards of the Declaration of Helsinki. All participants gave written informed consent for the study.

# RESULTS

## Demographic and clinical characteristics

The study population consisted of 93 kidney donors and 54 age- and sex-matched controls. Demographic and clinical characteristics of the patient and control groups are given in Table 1.

**Table 1.** General characteristics of the study population and controls at baseline.

| | Kidney donors (n=93) | Controls (n=53) | p-value |
|---|---|---|---|
| Age, years | 53.37 [27–68] | 55.44 [28–68] | 0.403 |
| Gender (male/female), n (%) | 34 (37)/58 (63) | 26 (49)/27 (51) | 0.278 |
| Body mass index, kg/$m^2$ | 26.5 [17.9–34.4] | 27.3 [17.1–38.7] | 0.594 |
| Waist circumference, cm | 98 [73–131] | 93 [77–143] | 0.378 |
| Hypertension (yes/no), n (%) | 6 (6.4)/87 (93.5) | 4(7.6)/49(92.4) | |
| Diabetes mellitus, n (%) | NA | NA | NA |
| Coronary artery disease | NA | NA | NA |

NA: not applicable. Normally distributed data are presented as mean±standard deviation. Non-normally distributed data are presented as median and interquartile range (IQR).

Participants' mean age was 53.37 [27–68] years, with 58 female (63%) and 34 male (37%). Similar to the control group, kidney donors were generally female and middle-aged. In total, 39 (41.9%) donors were spouses, 33 (35.4%) donors were sons or daughters, 16 (17.3%) donors were parents or siblings, and the remaining 5 (5.4%) donors were other relatives. The mean duration following transplantation was 78.03±41.09 months. None of the participants developed major surgical complications after donor nephrectomy.

### *Primary outcomes*

The biochemical parameters of the study population are shown in Table 2. In the donor population, the median eGFR 6 years after donation was 81.24 [67.36–88.62] mL/min/1.73 $m^2$ and was significantly lower in the donor group than that in the control group after 6 years of follow-up period (p<0.001). Serum creatinine was 0.93 [0.80–1.80] mg/dL 6 years after donation and significantly higher when compared with non-donors (p<0.001). While there was no significant difference between the two groups in the albumin–creatinine ratio in the spot urine (7.40 [3.70–15.50] vs. 7.22 [5.04–14.40] mg/g creatinine, p=0.431), the protein-creatinine ratio in the spot urine was found to be significantly higher in the donor group when compared with controls (85.52 [65.26–116.04] vs. 67.94 [56.12–87.80] mg/g creatinine, p=0.014).

Mean levels of ferritin were lower in the donor group 6 years after donation, when compared with controls (p<0.001) (Table 2). Mean levels of uric acid and parathyroid hormone (PTH) were significantly higher in the donor group, 6 years after donation, when compared with controls (p<0.001, and p=0.049, respectively) (Table 2). There was no significant difference between the two groups in terms of fasting blood glucose, HbA1c, low-density lipoprotein (LDL), albumin, and hemoglobin values.

### Secondary outcomes

As shown in Table 3, 32.9% of the donors (n=31) have HT, 2.1% (n=2) of the donors have CAD, 2.1% (n=2) of the donors have CKD, and 4.3% (n=4) of the donors have diabetes 6 years after donation. Of the kidney donors, 6.4% (n=6) of the

**Table 2.** Laboratory parameters of the kidney donors and controls at baseline and after 6 years.

| | Kidney donors at donation(n=93) | Kidney donors 6 years after donation(n=93) | Controls at baseline(n=54) | Controls after 6 years(n=54) | p-value |
|---|---|---|---|---|---|
| Urea (mg/dL) | 27.50 [23.00–33.00] | 35.00 [29.00–39.00] | 28.00 [24.00–33.25] | 27.00 [22.00–32.30] | **<0.001*** |
| Creatinine (mg/dL) | 0.75 [0.65–0.84] | 0.93 [0.80–1.80] | 0.67 [0.59–0.74] | 0.70 [0.62–0.80] | **<0.001*** |
| GFR (mL/min/1.73 $m^2$) | 101.0 [92.58–109.36] | 81.24 [67.36–88.62] | 109.0 [101.75–117.0] | 102.0 [94.9–109.1] | **<0.001*** |
| Albumin/creatinine (mg/g creatinine) | 5.50 [3.00–10.00] | 7.40 [3.70–15.50] | 7.05 [4.95–14.43] | 7.22 [5.04–14.40] | 0.431 |
| Protein/creatinine (mg/g creatinine) | 85.20 [66.85–109.90] | 85.52 [65.26–116.04] | 67.93 [56.29–87.79] | 67.94 [56.12–87.80] | **0.014*** |
| Hemoglobin (gr/dL) | 13.45±1.70 | 13.61±1.57 | 13.60±1.48 | 13.87±1.51 | 0.325 |
| Ferritin (ng/mL) | 33.00 [14.25–59.79] | 36.40 [19.90-64.90] | 33.50 [14.65–81.95] | 75.25 [31.40–112.70] | **<0.001*** |
| Total cholesterol (mg/dL) | 198.10±45.68 | 186.38±38.45 | 209.69±46.86 | 165.66±34.67 | 0.094 |
| LDL cholesterol (mg/dL) | 120.00±31.76 | 122.28±47.90 | 134.53±45.74 | 123.88±34.12 | 0.868 |
| Albumin (mg/dL) | 5.69±9.59 | 4.26±0.27 | 4.34±0.23 | 4.47±0.25 | 0.165 |
| Glucose (mg/dL) | 95.00 [89.6–101.0] | 92.00 [85.00–106.00] | 98.00 [89.50–105.25] | 95.00 [86.00–108.00] | 0.508 |
| HbA1c (%) | 5.50 [5.20–5.70] | 5.72 [5.48–6.96] | 5.40 [5.30–5.70] | 5.70 [5.60–5.90] | 0.717 |
| Uric acid (mg/dL) | 5.04±1.21 | 5.62±1.18 | 4.53±0.81 | 4.43±0.99 | **<0.001*** |
| PTH (pg/mL) | 52.35 [40.7–67.72] | 57.52 [40.70–76.55] | 48.25 [35.58–68.63] | 51.00 [36.80–65.30] | **0.049*** |

GFR: glomerular filtration rate; LDL: low-density lipoprotein; HbA1c: hemoglobin A1c; PTH: parathyroid hormone. Normally distributed data are presented as mean±standard deviation and non-normally distributed data are presented as median (IQR). Bold values indicate statistical significance at the p<0.05 level. *Kidney donors 6 years after donation vs. controls after 6 years.

**Table 3.** Target organ damage status of kidney donors 6 years after donation.

| | Kidney donors at donation (n=93) | Kidney donors 6 years after donation (n=93) | Controls at baseline (n=54) | Controls after 6 years(n=54) | p-value |
|---|---|---|---|---|---|
| Hypertension (n, %) | 6 (6.4) | 31 (32.9) | 4 (7.6) | 6 (11.1) | **<0.001*** |
| Diabetes mellitus (n, %) | NA | 4 (4.3) | NA | 1 (1.8) | **<0.001*** |
| Cardiovascular morbidity (n, %) | NA | 2 (2.1) | NA | 1 (1.8) | **<0.001*** |
| Cardiovascular mortality (n, %) | NA | NA | NA | NA | NA |
| Chronic kidney disease (n %) | NA | 2 (2.1) | NA | NA | **<0.001*** |
| End-stage kidney disease (n %) | NA | NA | NA | NA | NA |

NA: not applicable. *Kidney donors at donation vs. 6 years after donation. Bold values indicate statistical significance at the p<0.05 level.

donors have HT at the time of donation. The rate of hypertensive kidney donor increased after 6 years when compared with the baseline (6.4 vs. 32.9%, p<0.001) (Table 3).

Similarly, 7.6% (n=4) of the control group have HT at baseline. As reported in the methods section, the control group was formed with age- and sex-matched individuals who were selected based on self-reported medical history to fit the donor demographic and baseline clinical characteristics. There were no other chronic diseases such as diabetes, CAD, CKD, and ESKD in the control subjects at baseline. When clinical characteristics of controls were updated after 6 years from their baseline, we found that 11.1% (n=6) of the control group have HT, 1.8% (n=1) have diabetes, and 1.8% (n=1) have CAD.

## DISCUSSION

In this type 2 cohort study, we showed that not only the kidney function abnormalities were higher in the study population when compared with controls but also the incidence of new-onset HT was higher among kidney donors when compared with age- and sex-matched individuals. Furthermore, incidences of diabetes, CAD, and CKD were higher in the kidney donor group compared with their baseline.

According to our results, the mean eGFR was significantly lower in the donor group 6 years after donation compared with the control group (p<0.001) (Table 2). Also, the mean serum creatinine was significantly higher compared with non-donors (p<0.001). Our findings are in accordance with the current literature. Ibrahim et al reported that in an average of 12 years following donation, 15% of kidney donors had a GFR<60 mL/min/1.73 $m^2$ [12]. Similarly, Liboria et al reported that 29% of donors had an eGFR<60 mL/min/1.73 $m^2$ 5 years after donation[13]. Karahan et al recently reported that 3 years after donation, 11% of the kidney donors had GFR<60 mL/min/1.73 $m^2$ [14]. We found that the mean eGFR was 81.2 mL/min/1.73 $m^2$ 6 years after donation, and 2.1% (n=2) of donors had an eGFR<60 mL/min/1.73 $m^2$ in our study population. We interpreted that reduced renal function of kidney donors could be due to the reduction of kidney mass.

We also found that the mean levels of ferritin (36.40 [19.90–64.90] vs. 75.25 [31.40–112.70], p<0.001) were lower; the mean levels of uric acid (5.62±1.18 vs. 4.43±0.99, p<0.001) and PTH (57.52 [40.70–76.55] vs. 51.00 [36.80–65.30], p=0.049) were higher among kidney donors compared with controls. Kasiske et al reported that the GFR decreased 1.47±5.02 mL/min/1.73 $m^2$ per year in kidney donors between 6 and 36 months. The authors also reported that serum PTH, uric acid, homocysteine, and potassium levels were higher in kidney donors. The mean levels of PTH and uric acid in our study population were in line with Kasiske et al[15]. We found that kidney donors manifest several consequences of mild CKD in the long term. Yildirim et al reported that living kidney donors exhibit slightly reduced kidney function, increased oxidative stress, and decreased antioxidant activity[16]. It could be speculated that oxidant/antioxidant system imbalance may facilitate the development of kidney function abnormalities.

In addition, the protein–creatinine ratio of the kidney donors is significantly higher compared with controls (p=0.014). We also found that 6.4% (n=6) of donors have controlled HT at the time of donation and 32.9% (n=31) of donors have HT 6 years after donation. A meta-analysis of 48 studies showed a clinically insignificant increased risk for the development of HT or proteinuria in a long-term follow-up among kidney donors when compared with the age-matched controls[6]. Ibrahim et al reported that 7.5% of donors developed HT and 12% of donors developed albuminuria[12]. According to our results, 25% of donors developed new-onset HT, and none of the donors developed albuminuria and/or significant proteinuria 6 years after donation. Thiel et al showed that kidney donation triples the short-term risk of developing HT and that after nephrectomy, HT becomes the main risk factor for albuminuria. Thiel et al also reported that among the initially

normotensive donors, 43% of donors developed HT diagnosed by ambulatory blood pressure monitoring within the 10-year follow-up period[17]. We reported that hypertensives comprise 30% of our donor population within 6 years of donation. We could speculate that kidney donation leads to reduced kidney function and is associated with an increase in clinically insignificant proteinuria, as well as a rise in blood pressure greater than attributable to normal aging. Increased risk of developing HT may have important implications for the long-term cardiovascular health of kidney donors. We suggest that our data are critical for improving our understanding of the consequences of nephrectomy. Further prospective, controlled studies are needed to determine the incidence of HT, target organ damage, and possible complications of HT among donors.

Multiple studies have shown no evidence of reduced survival among living kidney donors as compared with the general population. Contrarily, Mjoen et al recently evaluated long-term cardiovascular and all-cause mortality among 1900 living kidney donors compared with a control group of 32,000 individuals who would have been eligible for donation over a 15-year follow-up period and found that the hazard ratios for all-cause death and cardiovascular death were significantly increased in donors after about 10 years. They also reported that living kidney donors have a 1.4-fold increased risk for cardiovascular morbidity compared with non-donor individuals eligible for donation[7]. According to our results, 4.30% (n=4) of kidney donors have new-onset diabetes and 2.1% (n=2) of donors have new-onset CAD 6 years after donation. Although there was no cardiovascular mortality in our study population; the incidence of HT, diabetes, and CAD is higher in the donor group compared with controls. It is still not possible to understand the pathophysiological effects of kidney donation on hemodynamic and vascular system among donors. We interpreted that it is impossible to exclude that donation may lead to an increase in adverse cardiovascular events. Potential donors should be informed of increased possible cardiovascular risk, at least new-onset HT and diabetes, associated with donation in the long term.

The findings of this study have to be seen in light of some limitations. First, in our follow-up, 25% of our donors could not be reached and their follow-up is not available. Second, our study was conducted with a relatively small population. Therefore, the study results may not reflect the general kidney donor population. Third, due to the design of the study, we could collect the data of the control group observationally. It would be interesting to assess the evolution of cardiovascular morbidity not only among kidney donors but also among age- and sex-matched individuals in a long-term follow-up study.

In conclusion, we detected a high incidence of HT, diabetes, CKD, and cardiovascular morbidity among kidney donors 6 years after donation. Further studies with larger populations are needed for the estimation of long-term risks associated with donation among living kidney donors.

## AUTHORS' CONTRIBUTIONS

**MM:** Conceptualization, Data curation, Writing – original draft, Writing – review & editing.

**EA:** Conceptualization, Formal Analysis, Writing – original draft, Writing – review & editing.

## REFERENCES


1. O'Keeffe LM, Ramond A, Oliver-Williams C, Willeit P, Paige E, Trotter P, et al. Mid- and long-term health risks in living kidney donors: a systematic review and meta-analysis. Ann Intern Med. 2018;168(4):276-84. https://doi.org/10.7326/M17-1235
2. Süleymanlar G, Ateş K, Seyahi N, Koçyiğit İ. Türkiye'de Nefroloji, Diyaliz ve Transplantasyon. Turkish J Nephrol. 2022;31:103-9. https://doi.org/10.5152/turkjnephrol.2022.22308
3. Reese PP, Boudville N, Garg AX. Living kidney donation: outcomes, ethics, and uncertainty. Lancet. 2015;385(9981):2003-13. https://doi.org/10.1016/S0140-6736(14)62484-3
4. Cherikh WS, Young CJ, Kramer BF, Taranto SE, Randall HB, Fan PY. Ethnic and gender related differences in the risk of end-stage renal disease after living kidney donation. Am J Transplant. 2011;11(8):1650-5. https://doi.org/10.1111/j.1600-6143.2011.03609.x
5. Boudville N, Prasad GV, Knoll G, Muirhead N, Thiessen-Philbrook H, Yang RC, et al. Meta-analysis: risk for hypertension in living kidney donors. Ann Intern Med. 2006;145(3):185-96. https://doi.org/10.7326/0003-4819-145-3-200608010-00006
6. Chronic Kidney Disease Prognosis Consortium, Matsushita K, van der Velde M, Astor BC, Woodward M, Levey AS, et al. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. Lancet. 2010;375(9731):2073-81. https://doi.org/10.1016/S0140-6736(10)60674-5
7. Mjøen G, Hallan S, Hartmann A, Foss A, Midtvedt K, Øyen O, et al. Long-term risks for kidney donors. Kidney Int. 2014;86(1):162-7. https://doi.org/10.1038/ki.2013.460
8. Whelton PK, Carey RM, Aronow WS, Casey Junior DE, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2018;71(6):1269-324. https://doi.org/10.1161/HYP.0000000000000066
9. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2010;33(Suppl 1):S62-9. https://doi.org/10.2337/dc10-S062

10. Lentine KL, Kasiske BL, Levey AS, Adams PL, Alberú J, Bakr MA, et al. KDIGO clinical practice guideline on the evaluation and care of living kidney donors. Transplantation. 2017;101(8S Suppl 1):S1-109. https://doi.org/10.1097/TP.0000000000001769

11. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. J Clin Epidemiol. 2008;61(4):344-9. https://doi.org/10.1016/j.jclinepi.2007.11.008

12. Ibrahim HN, Foley R, Tan L, Rogers T, Bailey RF, Guo H, et al. Long-term consequences of kidney donation. N Engl J Med. 2009;360(5):459-69. https://doi.org/10.1056/NEJMoa0804883

13. Libório AB, Barros RM, Esmeraldo RM, Oliveira ML, Silva Junior GB, Daher EF. Creatinine-based equations predicting chronic kidney disease after kidney donation. Transplant Proc. 2011;43(7):2481-6. https://doi.org/10.1016/j.transproceed.2011.04.009

14. Karahan M, Yildirim M, Kucuk HF, Turunc V, Demir H, Salturk C, et al. Oxidative DNA damage is increased in living kidney donors. Transplant Proc. 2019;51(4):1049-53. https://doi.org/10.1016/j.transproceed.2019.02.011

15. Kasiske BL, Anderson-Haag T, Israni AK, Kalil RS, Kimmel PL, Kraus ES, et al. A prospective controlled study of living kidney donors: three-year follow-up. Am J Kidney Dis. 2015;66(1):114-24. https://doi.org/10.1053/j.ajkd.2015.01.019

16. Yildirim M, Karahan M, Kucuk HF, Demir T, Demir H, Turan H, et al. Increased oxidative stress in living kidney donors: correlation of renal functions with antioxidant capacity. Transplant Proc. 2017;49(3):407-10. https://doi.org/10.1016/j.transproceed.2017.01.028

17. Thiel GT, Nolte C, Tsinalis D, Steiger J, Bachmann LM. Investigating kidney donation as a risk factor for hypertension and microalbuminuria: findings from the Swiss prospective follow-up of living kidney donors. BMJ Open. 2016;6(3):e010869. https://doi.org/10.1136/bmjopen-2015-010869