Original Research Article

A cross sectional follow-up study of kidney donors in a tertiary care centre from south India

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Abstract

Background: Living renal donors form the major pool of kidney transplantation in India. Even though many studies have demonstrated the safety of living renal donation there is always a concern about safety of renal donors. **Materials and method:** This was a cross sectional study conducted at Govt. Kilpauk Medical college hospital and Government Royapettah Hospital attached to Govt. Kilpauk Medical College, Chennai. Thirty living related renal donors were included in the study. Their case records were examined for immediate and long term complications. They were examined for HT, IFG, proteinuria, microscopic hematuria. Remnant kidney size was measured using ultra sonogram. Their GFR values were calculated using Cockcroft- Gault, a MDRD and CKD- EPI creatinine formulae. GFR was measured by Tc 99 DTPA isotope scan. Their kidney sizes, calculated and measured GFR were compared with pre-nephrectomy values. Correlation between calculated and measured GFR was assessed. **Results:** Two donors had hypertension. Five donors had IFG. Two had sub nephrotic proteinuria. Eleven donors had anemia. One developed CKD due to glomerular disease. One died because of malignancy. Donors had expected increase in measured GFR. There were no correlations between calculated eGFR and measured GFR. **Conclusion:** Even though living renal donation is a safe procedure regular follow up of donors and insistence on life style modifications during each visit is important.
Key Word: Renal donors, measured GFR, e GFR, CKD, proteinuria HT ,IFG.

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INTRODUCTION

Kidney transplantation is the best renal replacement modality for ESRD patients. The source of transplant kidney can be either from living or cadaver donors. In India where 3000 – 4000 renal transplantations take place annually, the donor pool is mainly from living donors who are mostly related to the recipient. In a few states viz. Tamilnadu, Gujarat and Maharashtra, deceased donors form a significant percentage of renal transplantation. Even then cadaver donors contribute 2% of the total renal transplantation only. After donating a kidney, the remaining kidney increases its function to compensate for its lost pair. In a short time after donation, the total GFR of the single kidney reaches 70 - 80% of the total GFR due to hyperfiltration and increased renal parenchymal volume(RPV). There have been several studies that have been reassuring and some studies revealing the risks acquired due to donation. This is yet another study on South Indian kidney donor population.

MATERIALS AND METHOD

This was a cross- sectional study conducted at Govt. Kilpauk Medical college hospital and Govt. Royapettah Hospital, attached to Govt. Kilpauk Medical College, Chennai. At present about 40 living related renal recipients are attending renal transplant follow up clinic at our centres. The renal donors who have completed 3 months of post-operative period were included in this study. Of these one donor had died of uterine malignancy. One developed CKD due to glomerular disease. Six did not give consent. Two could not be traced. Thirty donors were included in our study. Their case records were analysed for immediate and long term complications. Their BP was measured at least on 3 occasions to detect HT.FBS was measured. They underwent urine

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examination to detect proteinuria and hematuria. Serum creatinine was done by modified Jaffe's method. Ultrasonogram was done to measure transplant kidney size. Their eGFR was calculated by Cockcroft-Gault, aMDRD, CKD-EPI equations. Their GFR was measured by Tc 99- DTPA isotope scan. Kidney sizes, eGFR and measured GFR were compared with corresponding pre transplant values. Correlation between GFR and measured GFR was done for both pre transplant and post-transplant values. Statistical Analysis was done using SPSS version 19.0 and $p < 0.05$ was taken as statistical significance.

RESULTS

The earliest period of evaluation was 4 months post-transplant and the longest period was 156 months and median period of follow-up was 29 months (IQR 22.75). Of the 30 donors, 22 (73.3%) were females and 8 (26.7%) were males.

Table 1: The age distribution of donors was as follows

Age group (years)	Number	Percentage
18 – 29	1	3.3
30 – 39	7	23.3
40 – 49	14	46.7
> 50	8	26.7

The eldest donor was 58 years old at the time of donor nephrectomy and the youngest donor was aged 24. Immediate post-operative complications: Five donors (16%) had immediate post–operative complications. Two had atelectasis which improved with spirometry and chest physiotherapy. Two donors developed pneumonia which required escalation of antibiotics and prolonged hospital stay. One donor developed wound dehiscence requiring secondary suturing. No deaths occurred in the post-surgical period. Long term complications: One died because of uterine malignancy 2 years post donation. One developed CKD due to IgA nephropathy with crescents. Comparison of pre and post donation investigations The mean systolic BP was 119.53 and 119.50 mm of Hg pre and post-donation respectively and mean diastolic BP was 78.13 and 78.80 mm of Hg pre-donation and post-donation respectively. Two donors (6.7%) developed hypertension as defined by JNC 7after two years, one at stage 1 and another at stage 2. The mean Hb pre-donation was 11.40% and it was 11.13% post-donation. Eleven had anemia. There was a marginal rise in proteinuria from 0.12 to 0.17 post-donation. Two developed sub nephrotic proteinuria (PCR 0.4, 1). None developed microscopic hematuria post-donation. Mean FBS pre and post donation were 84.5 and 91.9 mg/dl respectively (p **0.001).**Five of the donors developed IFG. Mean serum creatinine pre and post donation were 0.84 and 1.01respectively (p **0.001).** Kidney length and width were measured using ultrasound and kidney size was calculated and compared with pre donation values. The depth was not measured and hence, renal parenchymal volume could not be calculated.

Mean kidney length(mm)			Mean kidney width(mm)			Mean kidney size(mm^2)		
Pre-donation	Post-donation	Increment	Pre-donation	Post-donation	Increment	Pre-donation	Post-donation	Increment
98.77	108.5	9.73	41.17	46.53	5.37	4082.8	5082	999.20
	p **0.001**			p **0.001**			p **0.001**	

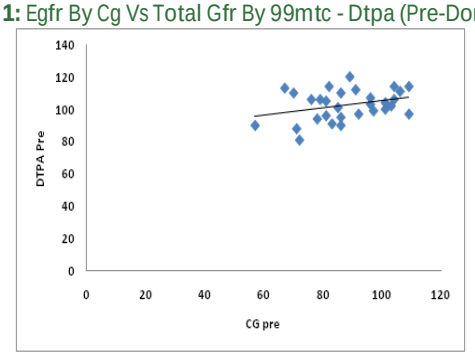
eGFR(ml/min/1.73 m^2) was calculated by CG formula, aMDRD and CKD EPI creatinine equation, pre and post donation.

eGFR by C-G formula (ml/ min/ 1.73 m2)			eGFR by aMDRD formula (ml/ min/ 1.73 m2)			eGFR by CKD-EPI formula (ml/ min/ 1.73 m2)		
Pre-donation	Post-donation	Decrement	Pre-donation	Post-donation	Decrement	Pre-donation	Post-donation	Decrement
97.93	72.47	15.46	84.34	66.97	17.37	93.17	75.17	18
	p **0.001**			p **0.001**			p **0.001**	

GFR measured by ^{99m}Tc DTPA pre and post-donation					
Total GFR by DTPA	Mean	SEM	Mean difference	T value	P value
Pre	102.53	1.70	21.76	11.715	**0.001**
Post	80.77	1.14			

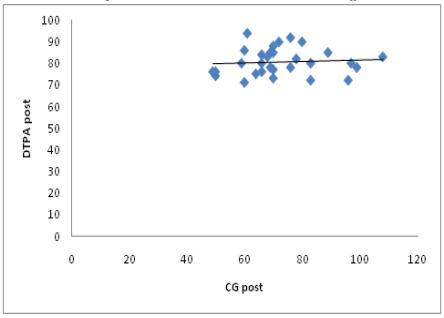
Correlation of eGFR vs GFR measured by ^{99m}Tc DTPA Pearsons correlation was used to analyze the correlation between eGFR calculated by different equations and that measured by ^{99m}Tc DTPA, pre and post donation. They are shown by the images below, along with the correlation coefficient r and its p value.

Figure 1: Egfr By Cg Vs Total Gfr By 99mtc - Dtpa (Pre-Donation)



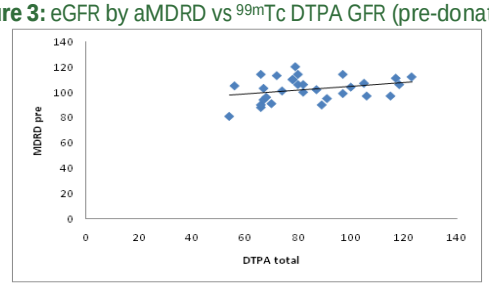
Correlation coefficient r = 0.331, p = 0.074

Figure 2: eGFR by CG vs ^{99m}Tc - DTPA GFR (post donation)



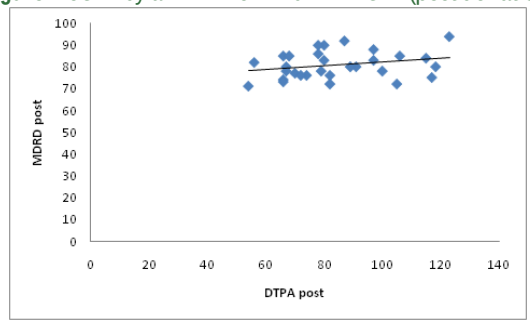
r = 0.071, p = 0.709

Figure 3: eGFR by aMDRD vs ^{99m}Tc DTPA GFR (pre-donation)



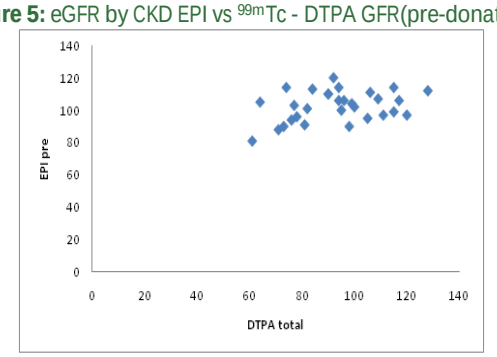
r = 0.291, p = 0.119

Figure4: eGFR by aMDRD vs ^{99m}Tc DTPA GFR (post donation



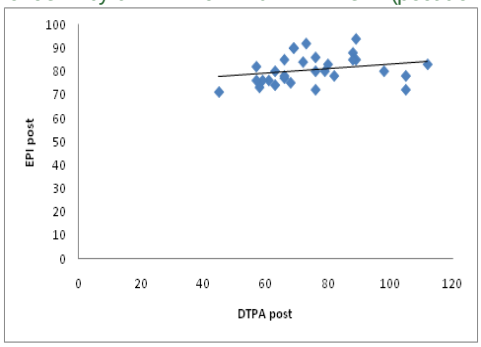
r = 0.164, p = 0.385

Figure 5: eGFR by CKD EPI vs ^{99m}Tc - DTPA GFR(pre-donation)



R = 0.340, p = 0.066

Figure 6: eGFR by CKD EPI vs ^{99m}Tc – DTPA GFR (post donation)



r = 0.254, p = 0.175

Table 1: Increase in measured GFR by ^{99m}Tc DTPA in remnant kidney

DTPA total gain	Mean	SEM	Mean difference	T value	P value
Pre single kidney GFR	50.87	1.11	-29.90	-22.182	**0.001**
Post	80.77	1.14			

The increase in GFR is more in younger age group (r = -0.362, p = 0.05). Kidney size had no correlation with measured GFR, both pre and post donation (R = 0.142, p = 0.455).

DISCUSSION

Living renal donation forms the source of more than 95 percent of renal transplantations in India. They transform the lives of numerous ESRD patients by their generosity and selfless attitude. Most of them are related to their recipients. Being aware of the morbidity and mortality of CKD patients, most of the donors take good care of themselves to avoid the risk factors for development of CKD. In our study of 30 donors, 22 were females (73.3%) and 8 were males (26.7%). There were 16 male recipients (53%) and 14 female recipients (47%). Most of the donors were females while most of the recipients were males. This is similar to the observation in studies by Biller et al[1] from Germany and Zimmermann et al[2] from Canada. It has been hypothesized that females think it is their duty to relieve the suffering of their spouse or

children. In our study, of the 22 female donors, 13 donated to their children, 6 to their husbands and 3 to their brothers. Of the 8 male donors, 4 donated to their children, 2 to their wives and 2 to their siblings. So 56.6% donors were parents, 26.6% spouses and 16.6% siblings. Peri – operative complications are relatively common[3] in renal transplantation and Blohmel demonstrated atelectasis (13.5%), prolonged ileus (5.2%), pneumonia (4.5%) and urinary tract infection (4.3%). In our centre, open nephrectomy is done and the mean period of hospital stay is 6 to 7 days. In our study 2 developed basal atelectasis (6%). Two donors developed pneumonia (6%) with necessity for escalation of antibiotics. One donor developed wound dehiscence (3%) which required secondary suturing and prolongation of hospital stay. The surgical mortality in donor nephrectomy is reported to be 0.03%by Segev[4]. In our study surgical mortality was nil. Though there was increase in spot protein creatinine ratio, from 0.13 to 0.17, it was not statistically significant. Two (6.6%) developed subnephrotic proteinuria. Similar studies show that incidence of proteinuria is about 3% to 24%[5,6,7,8,9]. Tapson observed 3% proteinuria at 10 years and 0% at 20 years (5). Natarajan observed 23% proteinuria in donors and 22% in their siblings[10]. Though late proteinuria has been reported that has not been linked to the progression of renal disease[4]. None of the donors in our study had microscopic hematuria. Though there was no statistical significance in Hemoglobin levels before and after renal donation, eleven donors had developed anemia post donation. Their peripheral smear study revealed microcytic hypochromic anemia. They improved with oral iron. Bertram l.Kasiske[11] observed 3.7% reduction of Hb in 194 donors in 8 US transplant centres 6 months post-donation. The concerning issue was the development of impaired fasting glucose in 5 donors (16.5%). Four of them were overweight. The earliest period to development was 1 year post donation and latest was 6 years. H. N. Ibrahim[12] observed154 (17.7%) T2DM from 2954 donors 9 years after donation. Imed H et al observed 8.4% T2DM in 284 Tunisian donors[13]. Two (6.6%) donors developed hypertension 2 years post donation. One was in JNC Stage 1and other in JNC Stage 2. Both were obese. The incidence of hypertension in donors has been reported to be the same as in general population. Similar studies report incidence of hypertension in renal donors between 7% to 47%[5,6,7,8,9,14]. Tapson[5] observed 13% incidence of HT at 10 years and 38% at 20 years. Williams[14] observed 47% incidence of HT in donors vs 35% in siblings, equal (42%) incidence of HT in male donors and age, race matched male controls and 50% incidence of HT in female donors comparing to 31% in female controls. One out of 30 donrs (2.7%) in our centres developed CKD due to IgA nephropathy with crescents. Hartmaan A and Holdaas H observed 7 cases of ESRD in 1800 donors and mostly due to primary kidney disease and not due to HT or hyperfiltration[15,16]. In similar studies incidences of CKD were between 2% to 12% [17,18,19]. Ellison[20] observed incidences of ESRD were similar between donors (0.04%) and general population (0.03%). One died of uterine malignancy 2 years post-transplant. Fehrman-Ekholm[21] observed 41 deaths in 430 donors between 15 months and 31 years in Sweden, majority due to cardiac disease and malignancy. The compensatory hyperfiltration by the remnant kidney leads to a maximum of 70% of pre-donation GFR in about 6 months. There was a mean 29.90(16 – 41ml/min/1.73 m^2) increase in the remnant kidney due to compensatory hyperfitration. The higher range of rise in GFR was seen with younger donors. This is similar to the observation reported in literature[22]. The other side of it, kidney donation leads to loss of total GFR by 30%. In our study, there were 0.17 increases in serum creatinine post donation (p **0.001**). The rise in creatinine resulted in statistically significant fall in estimated GFR calculated by the three equations, CG, aMDRD and CKD EPI creatinine, though all the three equations did not correlate with ^{99m}Tc - DTPA GFR in this donor population. There was decline in ^{99m}Tc - DTPA GFR, by an average of 21.76 ml/ min/1.73 m^2) (p **0.001**). This loss of GFR is similar to that reported in literature [23,24]. In our donors, remnant kidney increased its length, width and surface area (p **0.001**). This compensatory increase in size did not correlate with the increase in remnant kidney ^{99m}Tc - DTPA GFR. This is in contrast to the observation by Yasuhito Funahashi et al[25]. Small study population, short median follow up period and failure to compare eGFR and measured GFR with 24 hour creatinine clearance are limltations of our study.

CONCLUSIONS

Post-operative complications were seen in 16.5% of our donors. Two (6.6%) had sub nephrotic proteinuria. Impaired fasting glucose was seen in 16.5% of our donors and was of concern. Hypertension was seen in 6.5% of our donors, which is similar to that seen in general population. So regular follow up of donors and insistence on life style modifications are of paramount importance. There was expected increase in measured GFR. There were no correlations between calculated and measured GFR values.

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