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The impact of diabetes and hypertension on renal allograft survival- A single centre study

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Abstract

Background: To determine the impact of pre-transplant diabetes mellitus (DM) and post-transplant hypertension (HT) at 1 year on renal allograft survival in all adult first kidney-only (FKO) transplant recipients at a single transplant centre in Johannesburg, South Africa.

Materials and methods: A retrospective review was conducted of all adult FKO transplant procedures at the Charlotte Maxeke Johannesburg Academic Hospital transplant unit between 1966 and 2013.

Results: During the stipulated timeframe, 1685 adult FKO transplant procedures were performed. Of these, 84.1% were from deceased donors ($n=1413/1685$). The prevalence of pre-transplant DM transplant recipients with no missing or incomplete records was 6.5% ($n=107/1625$). Of the total cohort of 1685 adult FKO transplant recipients, 63.6% of those with no missing data survived to 1 year ($n=1072/1685$). The prevalence of HT at 1-year post-transplant was 53.6% ($n=503/1072$). HT at 1-year post-transplant, even after adjusted survival analysis, proved a significant risk factor for renal allograft loss (HR = 1.63; 95%CI = 1.37–1.94) ($p<0.0001$). Similarly, after adjusted survival analysis, the risk of renal allograft loss within the pre-transplant DM group was significantly higher ($p=0.043$; HR = 1.26; 95% CI = 1.01–1.58).

Conclusions: This study identified pre-transplantation diabetes mellitus and post-transplantation HT as significant risk factors for graft loss within the population assessed in this region of the world. These factors could potentially be used as independent predictors of renal graft survival.

Keywords: Diabetes; Hypertension; Johannesburg; Renal transplant; South Africa

1. Introduction

In South Africa (SA), the first kidney transplant was performed at the old Johannesburg Hospital on the August 25, 1966.[1,2] Kidney transplantation (KT) has been shown to significantly reduce the risk of death by over 60% when compared to chronic dialysis (both peritoneal and haemodialysis modalities), thus doubling the expected survival time and greatly improving quality of life.[3]

According to the South African Renal Registry (SARR) Annual Report 2018, the most commonly reported definitive causes of end-stage kidney disease (ESKD) within patients presenting to both the public and private sector chronic dialysis units were hypertension (HT) (35.1%) and diabetes mellitus (DM) (15.0%).[4] KT is of vital importance in SA, especially because dialysis is often rationed due to limited resources.[5] However, KT rates remain lower than countries with comparable economic capacity.[5,6]

Certain donor and recipient characteristics and risk factors may negatively impact graft survival, including immunological (allo-immune mediated injury) and non-immunological causes (advanced donor age, cerebrovascular accident, DM, HT, presence of infection, hemorrhage, and malignancy).[7–10] HT is a common and serious post-KT complication, with a prevalence as high as 80% in KT recipients.[11] Despite the common description of a negative causal effect between post-KT, HT and graft survival, previous studies have left unanswered whether HT is the cause or result of progressive renal dysfunction and graft failure.[12] Numerous international studies suggest that the aetiology of post-KT HT is multifactorial and most commonly occurs secondarily to loss of native kidney function, immunosuppressive regimens, renal artery stenosis, and chronic allograft nephropathy.[13]

Historically, it has been demonstrated that an increase of greater than 10 mm Hg in systolic blood pressure (SBP) was associated with a 5%–12% increased risk of graft failure.[13,14] Opelz et al., in a retrospective analysis of the Collaborative Transplant Study database, further illustrated that KT recipients who achieved HT control post-KT (defined as a SBP < 140 mm Hg) had better graft survival rates at 1 year post-KT compared to those with sustained HT (RR = 0.79).[15]

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The prevalence of DM is increasing worldwide and according to the SARR 2018, DM is the third most common cause of ESKD in SA, with a reported prevalence of 15.0% among patients with ESKD.[4] KT recipients with pre-existing DM have been shown to have a significantly higher rate of graft loss and mortality after transplantation.[16] Pre-transplant hyperglycaemia is thought to independently contribute to decreased graft survival, possibly due to ischaemic reperfusion injury and the inflammatory state it provokes.[17] Non-immunological factors (HT and DM) are increasingly identified as modifiable risk factors which adversely affect renal allografts.[7,9,18] Early assessment of graft function in relation to underlying comorbidities, namely HT and DM, and appropriate management of these comorbidities remains vital for long term recipient and graft survival.[7,10,16]

In this study, we aimed to analyse the impact of pre-transplant DM and post-transplant HT (at 1-year post-KT) on renal allograft survival in all adult first kidney-only (FKO) transplant recipients (both deceased and living donor) from a large kidney transplant centre in Johannesburg, SA.

2. Materials and methods

A retrospective analysis of all adult FKO transplant recipients was performed at Charlotte Maxeke Johannesburg Academic Hospital in Johannesburg, SA. The period under review was from January 1, 1966 to December 31, 2013. To achieve the required study outcomes, the required sample size was calculated assuming a 10-year graft survival of 36% and an estimated prevalence of the risk factors (HT or DM) of 10%. Detection of a hazard ratio (HR) of at least 1.65 with 80% power at the 5% significance level required a minimum sample size of 1532 patients.[19] The study was approved by the University of the Witwatersrand Human Research Ethics Committee (HREC Medical); R14/49, Certificate number: M200566.

2.1. Data collection

The following recipient variables were included in the analysis: age at transplant, sex, self-reported population group, cause of ESKD, indication for KT, presence or absence of pre-transplant DM, presence or absence of HT at 1-year post-transplant graft type (deceased or living donor), graft survival at 1, 5, and 10 years post-KT and immunosuppressive regimens used. KT recipients were categorized into 3 eras. Within each of the eras, corticosteroids (prednisone) were included as one component of a dual or triple immunosuppression regimen:

During Era 1, from 1966 to 1983, a dual regimen with prednisone and azathioprine was used. During Era 2, from 1984 to 2000, a triple regimen was used, with addition of calcineurin-inhibitors (cyclosporine) to azathioprine and prednisone. During Era 3, from 2000 to 2013, a triple regimen comprised of next generation calcineurin inhibitors (tacrolimus) that replaced cyclosporine, mycophenolic acid that replaced azathioprine, and introduction of mammalian target of rapamycin inhibitors in selected cases.

2.2. Statistical analysis

The effect of pre-transplant diabetes on graft survival, unadjusted and adjusted for age at transplant, sex, population group, donor type and transplant era, was assessed by Cox Proportional Hazards regression. The effect of HT at 1-year post transplant in the group which survived to 1 year was determined similarly. Data analysis was carried out using SAS software v9.4 for Windows.[20] The level of statistical significance was set to 0.05 or 5%.

3. Results

3.1. Study demographics

Data from a total of 1685 adult FKO transplant recipients were included over the 48-year study period. The mean age at transplant was 37.9 years (SD = 10.6 years) and 61.4% of all first adult transplant recipients were male (n = 1034/1685). Of the total cohort of 1685 FKO KT recipients, 63.6% of post-KT patients had HT data with no missing data or records who survived to 1 year (n = 1072/1685). Data for 96.4% of pre-transplant DM recipients were found to be adequate for analysis with no missing data or records (n = 1625/1685). The prevalence of pre-transplant DM and HT 1-year post-KT were 6.5% (n = 107/1625) and 53.6% (n = 503/1072), respectively. The largest proportion of transplants was performed in Era 2 (1983–2000), accounting for 54.2% of the entire cohort (n = 914/1685). The distribution relating to population groups included in this study was heterogeneous, with an initial preponderance for those patients of white descent, accounting for 56.6% of the total sample (n = 951/1685). However, when factoring in the change in socio-political climate that occurred in SA during the post-apartheid era (after 1994), our data indicates a shift in racial distribution toward a black predominance at our centre, where previously only white patients were treated. Of all donors, 84.1% were deceased (n = 1413/1685). The most common cause identified for ESKD at the time of transplant was primary renal disease accounting for 52% (n = 780/1685), followed by HT at 32.6% (n = 489/1685) (Table 1).

3.2. Graft survival among pre-transplant DM vs. non-DM KT recipients

For the purposes of this sub-group analysis, 96.4% of cases (n = 1625/1685) were found to have no missing data for variables required for survival analysis. For those FKO recipients with pre-transplant DM, graft survival at 1 year for the entire cohort was 67.5% (95% CI = 65.2%–69.8%) (Table 2). The unadjusted survival analysis illustrated no difference in graft failure between those with and without pre-transplant diabetes (HR = 1.13; 95% CI = 0.91–1.41). In contrast, the adjusted analysis demonstrated that the risk of graft loss was significantly higher in those with pre-transplant DM (HR = 1.26; 95% CI = 1.01–1.58) (Fig. 1). A significantly higher risk of graft loss ($p <$ 0.0001), was also noted for those patients aged 50 and above (HR = 1.68; 95% CI = 1.38–2.05), and recipients of black ($p <$ 0.001; HR = 1.43; 95% CI = 1.22–1.69) and Asian (p = 0.0048; HR = 1.48; 95% CI = 1.12–1.89) origin when compared to the white population group. A lower risk ($p < 0.0001$) of allograft loss was observed for FKO living donor transplant recipients (HR = 0.61; 95% CI = 0.51–0.75) when compared to those of deceased donor origin. When analysing according to predefined immunosuppressive regimen Eras 1 to 3, the lowest risk ($p <$ 0.0001) of allograft loss was noted in Era 3 (HR = 0.42; 95% CI = 0.33–0.51) (Table 3).

3.3. Graft survival in HT vs. non-HT at 1-year post-KT

For all adult FKO transplant recipients with HT present at 1-year post-KT, the overall graft survival analysis at 5 and 10 years was 75.4% (95% CI = 72.4%–78.1%) and 52.6% (95% CI = 49%–56%), respectively (Table 2). The unadjusted survival analysis of the data revealed that those FKO transplant recipients with HT present at 1-year post-KT had a significantly higher risk of allograft failure, compared with those who did not (HR = 1.63; 95% CI = 1.37–1.93). In the adjusted analysis, the risk of

Table 1

Demographic data of the cohort overall and by era.

	Overall		Alive at 1 year post-transplant	
Variables	**n**	**%**	**n**	**%**
Total	1685	100	1072	100
Transplant Era				
Era 1 (1966–1983)	458	27.2	271	25.3
Era 2 (1984–2000)	914	54.2	580	54.1
Era 3 (2001–2013)	313	18.6	221	20.6
Recipient age at transplant, yr				
18–29	422	25.0	283	26.4
30–39	499	29.6	315	29.4
40–49	526	31.2	336	31.3
50+	238	14.1	138	12.9
Recipient sex				
Female	651	38.6	413	38.5
Male	1034	61.4	659	61.5
Recipient population group				
White	951	56.6	620	57.9
Black	516	30.7	326	30.4
Mixed	117	7.0	70	6.5
Asian	95	5.7	55	5.1
Unknown	6		1	
Cause of ESKD				
Renal disease	780	52.0	481	51.1
Hypertension	489	32.6	304	32.3
Urological	127	8.5	80	8.5
Inherited	126	8.4	87	9.2
Anatomical	14	0.9	8	0.8
Other	29	1.9	21	2.2
Unknown	184		130	
Donor type				
Deceased	1413	84.1	870	81.4
Living	268	15.9	199	18.6
Unknown	4		3	
Diabetes at time of transplant				
Yes	107	6.5	72	6.9
No/unknown	1528	93.5	976	93.1
Not recorded	50		24	
*SBP at 1 year, mmHg				
<140			503	53.6
≥140			435	46.4
Not recorded			134	

* SBP = systolic blood pressure; ESKD = end-stage kidney disease.

allograft loss remained significantly higher ($p < 0.0001$) in those with HT present at 1-year post-transplant (HR = 1.63; 95% CI = 1.37–1.94) (Fig. 2). A higher risk of allograft loss ($p = 0.0001$) was noted in recipients older than 50 years of age (HR = 1.75; 95% CI = 1.31–2.34) and those from the black ($p < 0.0001$; HR = 1.66; 95% CI = 1.31–2.09) and Asian ($p = 0.0071$; HR = 1.67; 95% CI = 1.15–2.42) population groups. A lower risk ($p = 0.037$) of allograft loss was also demonstrated in recipients of living donor FKO transplants (HR = 0.76; 95% CI = 0.59–0.98). The comparison of the effect of the use of different immunosuppressive regimens over the three predefined Eras 1 to 3 on allograft survival, showed that the lowest risk ($p = 0.0002$) for allograft loss occurred during Era 3 (HR = 0.49; 95% CI = 0.33–0.71) (Table 3).

Table 2

Overall graft survival in the overall cohort (n = 1625) and those who survived to 1-year post-KT (n = 925).

	Overall cohort		Alive at 1-year post-KT	
Time (yr)	**Graft survival (%)**	**95% CI (%)**	**Graft survival (%)**	**95% CI (%)**
1	67.5	65.2–69.8	100.0	–
5	50.7	48.1–53.2	75.4	72.4–78.1
10	36.1	33.6–38.7	52.6	49.0–56.0

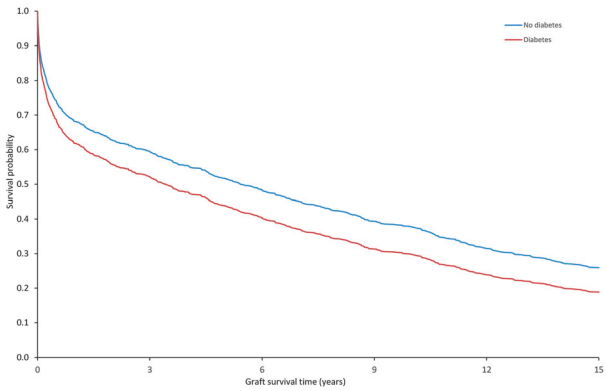

Figure 1. Adjusted graft survival analysis curve for pre-transplant DM KT recipients. Non-DM (blue line) vs. DM (red line).

4. Discussion

Since the inception of KT, renal allograft survival has improved due to presence of newer immunosuppressive therapies, better immunological understanding and advances in surgical techniques.[1,5] In SA, access to dialysis is limited and hence preservation and optimization of renal allograft survival is paramount.[1,5]

Local data on the impact of DM and HT on renal allograft survival in adult FKO KT remains scarce.[2,4,6] On review of currently available literature, no such studies have been performed in a Southern African context.[2,4,5] This study serves, as the first comprehensive report from this region of the world, to assess the impact of pre-transplant DM and post-transplant HT on renal allograft survival.

Overall, graft survival outcomes, at 1 year, among HT FKO transplant recipients within our study compared unfavourably to international standards, which have demonstrated survival rates of up to 95%.[18,21] This may be attributed to the lower percentage of living donor KT (LDKT) within our cohort, which appeared to demonstrate a favourable outcome in other studies.[21,22] This finding bodes well for SA, as recent studies project that LDKT may exceed deceased donor KT in years to come.[2]

An analysis performed by Cosio et al. of post-KT HT in deceased donor KT recipients demonstrated a significant negative causational relationship between mean arterial blood pressure and renal allograft survival in African-American recipients ($p < 0.001$).[23,24] Similarly, within our cohort, the racial distribution of KT recipients with HT, indicated a predominance of the black population group (post-1994), possibly as a result of a predisposing genetic component within this group.[23,24] This finding may be a valuable consideration for clinicians to guide earlier risk stratification, identification and optimization of HT control in these patients.

Meier-Kreische et al., in an analysis of 40,289 KT recipients, demonstrated that patients aged 55 years and older had a 110% risk of chronic allograft failure (RR = 2.1; $p < 0.01$).[25] Similarly, we also demonstrated that KT recipients with HT at 1 year post-transplant aged 50 years and older had a significantly higher risk

Table 3

Adjusted survival analysis of the overall cohort ($n = 1625$) and those who survived to 1-year post-KT ($n = 925$).

	Overall cohort				Alive at 1 year post-KT			
	HR	95% CI for HR		p	HR	95% CI for HR		p
SBP at 1 year, mm Hg								
<140				1		Reference		
≥140					1.63	1.37	1.94	<0.0001
Diabetes at time of transplant								
No/unknown	1	Reference						
Yes	1.26	1.01	1.58	0.043				
Transplant Era								
Era 1 (1966–1983)	1	Reference		1		Reference		
Era 2 (1984–2000)	0.82	0.71	0.95	0.0075	0.88	0.72	1.08	0.21
Era 3 (2001–2013)	0.42	0.33	0.54	<0.0001	0.49	0.33	0.71	0.0002
Recipient age at transplant, yr								
18–29	1	Reference		1		Reference		
30–39	1.12	0.95	1.33	0.17	1.09	0.86	1.37	0.47
40–49	1.16	0.99	1.37	0.072	1.11	0.88	1.40	0.37
50+	1.68	1.38	2.05	<0.0001	1.75	1.31	2.34	0.0001
Recipient sex								
Male	1	Reference		1		Reference		
Female	1.01	0.89	1.14	0.88	0.99	0.83	1.17	0.86
Recipient population group								
White	1	Reference			1	Reference		
Black	1.43	1.22	1.69	<0.0001	1.66	1.31	2.09	<0.0001
Mixed	1.16	0.92	1.48	0.21	0.99	0.70	1.40	0.94
Asian	1.46	1.12	1.89	0.0048	1.67	1.15	2.42	0.0071
Donor type								
Deceased	1	Reference		1		Reference		
Living	0.61	0.51	0.75	<0.0001	0.76	0.59	0.98	0.037

HR = hazard ratio; SBP = systolic blood pressure.

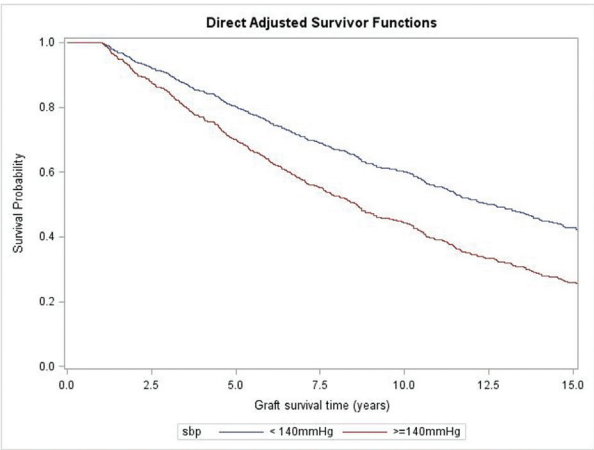

Figure 2. Adjusted survival anaylsis curve for Post-transplant KT recipients who survived to 1-year. Non-HT (blue line) vs. HT (red line).

of allograft failure when compared to those in the 18–29 year old group.

Despite initial poor outcomes, we noted a significant time-dependent improvement in allograft outcomes in Era 3 as compared to earlier Eras. This is likely attributable to the evolution of immunosuppressive regimens, improved early diagnosis and management of post-transplant HT.[5,26] When compared to larger international studies of a similar nature, our study paralleled the conclusion that elevated SBP negatively impacts renal allograft survival, demonstrating its significant impact on overall graft survival, as evidenced by the decline in graft survival at both 5 and 10 years graft survival predictions post-KT when compared to non-hypertensive KT recipients.[12,27] Yet again, this finding highlights the importance of early diagnosis and control of post-transplant HT among FKO transplant recipients.

According to the SARR 2018 annual report findings, DM accounts for the third most common cause of ESKD in SA.[2,4] When reviewing the impact of pre-transplant DM on renal allograft survival, our findings appear consistent with international consensus.[16,23] Kuo et al., while conducting a retrospective analysis of 45,989 adult FKO KT from the Scientific Registry of Transplant Recipients, noted that KT recipients with pre-existing DM had a 30%–40% increased risk of mortality, but found no significant impact on death-censored graft loss.[16,28] Our overall graft survival analysis of KT recipients with pre-transplant DM yielded a similar finding, however after performing a multivariable analysis, we demonstrated a significantly higher overall risk of graft loss in those KT recipients with pre-transplant DM, among which those patients who were LDKT recipients were shown to have a lower risk of graft failure.

Kuo et al. further indicated that 52% of pre-transplant DM KT recipients were in the 40–60 years old age category.[28] Within our cohort, only 45.3% of pre-transplant DM KT recipients fell into this age category, a discrepancy which may be attributed to the fact that within public sector in SA, where resources are constrained, transplant criteria may favour younger and healthier patients.[5] Our analysis suggested that pre-transplant DM KT recipients 50 years and older had the highest risk of graft failure.

In terms of assessment of the association between racial population group and pre-transplant DM KT recipient outcomes within our sample, in agreement with international studies of similar cohort sizes, we observed a significantly higher risk of graft failure in Black African and Asian KT recipients as compared to other population groups.[13,29] The observed change in population group composition of KT recipients in the post-apartheid era in this study correlated with 2011 South African census findings, which determined black South Africans to be the largest racial demographic group (79.2%). In addition, evidence suggests that the Asian population has a genetic predisposition to diseases such as DM and therefore an overall increased risk of progression to ESKD irrespective of aetiology.[30,31]

Our findings further suggest that early detection, monitoring and optimizing control of pre-existing DM in FKO transplant recipients should play an important role in improving renal allograft graft outcomes within this patient sub-group.

5. Conclusion

Our findings add valuable information to the paucity of data that exist when assessing the impact of DM and HT on FKO KT outcomes in Sub-Saharan Africa. Our study identified pre-transplant DM and post-transplant HT (at 1-year post-transplantation) as significant risk factors for graft loss at our centre. These results may aid in identifying those at high risk for graft loss, emphasizing the importance of optimizing management of these common conditions in the peri-transplant period.

Limitations of this study include the retrospective study design over a long time period with unavoidable missing data. Additionally, the study was from a single centre, which may not be nationally or regionally representative. Pertaining specifically to HT, we were unable to ascertain the pre-transplant HT status of recipients due to the paucity of this data recorded over the stipulated time frame. Finally, since the time frame under review spanned almost 50 years, certain population groups were also under-represented as the apartheid policy precluded their access to equal medical care.

Acknowledgments

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Statement of ethics

The study was approved by the University of the Witwatersrand Human Research Ethics Committee (HREC Medical); R14/49, Certificate number: M200566. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No patient consent was required for any procedures as the database was created with files and patient records de-identified and approved via the same HREC as mentioned.

Conflict of interest statement

The authors declare no conflicts of interest.

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Author contributions

Sumesh Padayachee: Guarantor of the manuscript and responsible for the integrity of the data and interpretation of the data; and drafting, writing, incorporation of co-author feedback, revision, and final submission composition;
Ahmed Adam: Contributed to the study design, analysis and interpretation of the data, revision and approval of the manuscript;
June Fabian: Contributed to the study design, analysis and interpretation of the data, revision and approval of the manuscript.

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