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Risk of Post-transplant Erectile Dysfunction in Dialysis-dependent Patients With Advanced-stage Chronic Kidney Disease

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Abstract

Purpose:

Erectile Dysfunction (ED) is common in chronic kidney disease (CKD). While certain aspects of sexual function often recover after transplant, rates of ED are still high in transplant recipients. We compared rates of new-onset ED in dialysis-dependent CKD patients treated with kidney transplant (KT) vs those managed on dialysis.

Methods:

This retrospective cohort study used the TriNetX Global Collaborative network, a research database containing de-identified clinical data from 172 health care organizations for over 200 million patients. We evaluated patients with CKD on dialysis who undergo KT vs those without KT. We used a 1:1 greedy nearest-neighbor propensity score matched on age, race/ethnicity, and comorbidities. Rates of new onset ED were compared among cohorts before and after matching. Cox regression determined the percentage disease free survival at the end of the study period. Outcomes of interest were the overall rates of ED after transplant in patients with KT or initiation of dialysis in patients without KT.

Results:

298,915 patients were included: 33,063 with KT and 265,852 without. After propensity matching, 31,625 KT patients and 31,625 non-transplant patients were compared; KT was associated with a lower ED risk (RR 0.51 [0.48–0.55]; HR 0.32 [0.30–0.34]). More KT patients were ED-free at follow-up (85.2% vs 70.9%) and average follow-up was longer (2,020 vs 1,141 days).

Conclusions

Lower rates of ED after KT support a protective effect of KT on new ED diagnosis. Non-zero rates suggest that these patients may focus on quality-of-life post-transplant and have more frequent interactions with the healthcare system, leading to greater attention to ED. New ED rates were lower after KT than in dialysis-dependent patients with CKD who do not undergo KT. For CKD patients on dialysis, addressing ED is an opportunity to enhance quality of life for those without access to transplantation.

Introduction

The pathogenesis of erectile dysfunction (ED) is multifactorial, encompassing both physical and psychological domains, but generally includes cardiovascular, neurologic, hormonal, medication related, and psychogenic causes.[1,2] An additional risk factor for the development of ED is chronic kidney disease (CKD); with a rising incidence of CKD in the United States, many urologists may encounter a growing number of CKD patients in their practice with ED.[3–7]

CKD likely contributes to the development of ED through its downstream effects on blood pressure, endothelial impairment, and systemic perfusion.[8,9] Pizzol et al. found an ED prevalence of 76% in CKD patients, with similar rates seen between dialysis and advanced CKD patients.[10–14] Interestingly, observational studies suggest that individuals have subjective improvement in erectile function after kidney transplant (KT), with both serum testosterone and subjective reports of sexual dysfunction and ED improving after transplant surgery.[15] Despite ample evidence that a functional renal graft can alleviate sexual dysfunction for some patients, a significant portion of recipients still experience ED (20–50%), and little is known about new onset ED following transplantation in this population.[15,16]

We seek to clarify the relationship between new-onset ED in dialysis-dependent adult men with CKD compared to those who receive renal transplantation. To clarify this relationship, we queried the TriNetX Research Network, a federated database that pools patient data from electronic medical records (EMR's) from a multinational network of health care organizations (HCO's).

Materials and Methods

Our study population consisted of men ≥ 18 years of age in the TriNetX Research Network, a global federated database containing the cumulative data from a multinational collaboration between participating HCO's. We specifically used the Global Collaborative Network, which contains data from 172 participating HCO's from around the world, and approximately 205,687,916 patients, including diagnoses, procedures, and medications. Participating HCO's periodically update patient information and are not consistently online simultaneously. We based our analysis on results and analytics performed on the access date of 01/13/2026.

TriNetX complies with the Health Insurance Portability and Accountability Act (HIPAA) and is certified to the ISO 27001:2022 standard and maintains an Information Security Management System (ISMS) to ensure the protection of the healthcare data it has access to and to meet the requirements of the HIPAA Security Rule. Since our study contains no patient or institution identifiers, no IRB or IRB waiver was required in the publication of this research.

Information collected included demographics, comorbid medical conditions, and outcomes based on International Classification of Disease (ICD) codes, Current Procedural Terminology (CPT) codes, Systematized Nomenclature of Medicine (SNOMED), and RxNorm codes. TriNetX curated terms for certain diagnoses, medications, and laboratory results were also recorded for analysis. The presence of the corresponding ICD code linked to a patient's data within the TriNetX database was considered to be equivalent to a diagnosis.

Study Population

Men ≥ 18 years old with a diagnosis of CKD and receiving hemodialysis in the 20 years prior to the access date were included in the study. CKD was defined by chronic kidney disease (stage IV [ICD-10 N18.4] and stage V [N18.5]) or End Stage Renal Disease (ESRD) (N18.6) and hemodialysis (ICD-10 Z99.2, ICD-9 39.95, CPT 1012752, 90935, 90937, or SNOMED 302497006). Patients are automatically excluded from analysis if they met the index event greater than 20 years prior to the database access date.

We excluded patients with a prior history of ED as defined by ICD-10 N52 (Male Erectile Dysfunction), or by a new prescription of any dose of avanafil, sildenafil, vardenafil, or tadalafil 20 mg or 40 mg (RxNorm 1291301, 136411, 306674, and 358263, respectively). Tadalafil 5 was not included due to potential confounding with prescriptions for benign prostatic hyperplasia (BPH). In addition, patients with prior history of alternative treatments for erectile dysfunction such as vacuum erection devices, intracavernosal injections, or inflatable penile prostheses were excluded. KT was defined by ICD 10 BT49, 0TY00Z1, 0TY10Z1, 0TY10Z2, 0TY00Z0, 0TY10Z0, and Z94.0; as well as CPT 1008098, and SNOMED 175901007, 113127005, and 175902000.

From this study population, we made two cohorts; one with a KT (as previously defined) and one with no history of transplant.

Statistical Analysis

The percentage of dialysis-dependent patients with new ED after transplant or after CKD in the absence of transplant was calculated, along with risk ratios for the diagnosis of ED in the KT and no-KT groups. Follow-up duration and hazard ratios for the duration of ED-free survival for both groups were also determined. Incidence rates per 1000 person years were calculated at 6 month intervals up to 3 years and then 1 year intervals from years 3 to 5 to account for differences in follow-up duration between the two groups.

The logic built into TriNetX analytics identifies an index event and assesses for outcomes after all criteria for the index event have been met. For both groups, the index event was the first occurrence of hemodialysis and CKD. Patients with ED in the KT group that were diagnosed between the index event and their transplant date were excluded from this cohort to ensure the integrity of the analysis.

To account for confounding factors and ensure homogeneity between the two groups, these datapoints were collected before and after propensity score matching as described previously. The TriNetX platform conducts a 1:1 greedy nearest-neighbor propensity score powered by Python and R software. Criteria are set such that differences between two cohorts are considered sufficiently minimized if standardized differences between propensity scores are ≤ 0.1. Results were assessed before and after matching with a 1:1 greedy nearest-neighbor propensity score matched on age, race/ethnicity, and comorbid conditions. Propensity matching factors included ICD-10 E11 (Type 2 Diabetes Mellitus), TNX Curated BMI > 30 (9083), Primary Hypertension (I10), Secondary hypertension (I15), Atherosclerosis (I70), Chronic Ischemic Heart Disease (I25), Alcohol use (F10), Tobacco use (Z72.0), Obstructive Sleep Apnea (G47.33), Hyperlipidemia (TNX curated 9004), and kidney disease severity (Serum Creatinine). A summary of diagnostic and billing codes used for the various definitions in this study are summarized in Table 3.

A cox regression model was used to determine hazard ratios for ED development and duration of ED-free survival with the same index event as previously described. Software used for this research was built into the TriNetX platform via TriNetX Live *(TriNetX LLC, Cambridge, MA*). Statistical significance was defined by p-value less than 0.05.

Results

298,915 patients were included: 33,063 with KT and 265,852 without. Before matching, KT was associated with black race and Hispanic/Latino ethnicity, and all comorbidities were more common in the no KT group

except for secondary hypertension (Table 1). After propensity score matching, all cohorts were sufficiently matched based on standard mean difference < 0.1 for age, race/ethnicity, and all comorbidities.

Table 1

Comparison of demographic and comorbidities between men receiving dialysis for treatment of CKD vs those with kidney transplant after prior treatment with dialysis

	Before Propensity Matching				After Propensity Matching		
	KT	**No KT**	**P value**	**Standard Mean Difference**	**KT**	**No KT**	**Standard Mean Difference**
Total Patients (n)	33,063	265,852	n/a	n/a	31,625	31,625	n/a
Current Age (years)	59.4 ± 14.8	68.5 ± 14.6	0.0001	0.62	59.5 ± 14.6	59.6 ± 15.6	0.0011
Age at Index (years)	51.4 ± 14.7	62.2 ± 14.5	0.0001	0.74	51.6 ± 14.5	51.6 ± 15.2	0.0002
Race/Ethnicity							
White % (n)	46.6 (14,819)	46.6 (118,079)	0.88	0.0009	46.5 (14,702)	45.5 (14,398)	0.019
Black/AA % (n)	26.3 (8,369)	25.7 (64,936)	0.011	0.015	26.4 (8,348)	27.5 (8,697)	0.025
Hispanic/Latino % (n)	12.7 (4,041)	11.3 (28,546)	0.0001	0.044	12.7 (4,015)	12.1 (3,819)	0.019
Medical Comorbidities							
Primary HTN % (n)	43.8 (13,912)	48.4 (122,413)	0.0001	0.093	43.7 (13,814)	42.4 (13,399)	0.027
Secondary HTN % (n)	5.8 (1,847)	3.8 (9,480)	0.0001	0.097	5.6 (1,781)	5.2 (1,647)	0.019
DM II % (n)	26.9 (8,534)	39.0 (98,762)	0.0001	0.26	27.0 (8,532)	26.3 (8,313)	0.016
CIHD % (n)	14.6 (4,632)	25.3 (63,978)	0.0001	0.27	14.6 (4,630)	13.7 (4,360)	0.025
Atherosclerosis % (n)	5.4 (1,704)	8.5 (21,617)	0.0001	0.13	5.4 (1,701)	4.7 (1,493)	0.030
OSA % (n)	6.1 (1,954)	9.1 (23,130)	0.0001	0.11	6.2 (1,949)	6.0 (1,881)	0.0090
BPH % (n)	5.5 (1,757)	13.0 (32,788)	0.0001	0.26	5.6 (1,757)	4.9 (1,559)	0.028
Obesity (BMI > 30) % (n)	10.8 (3,427)	15.4 (38,884)	0.0001	0.14	10.8 (3,419)	10.0 (3,175)	0.025

Abbreviations: AA, African-American; HTN, Hypertension; DM II, Type II Diabetes Mellitus; CIHD, Chronic Ischemic Heart Disease; OSA, Obstructive Sleep Apnea; BPH, Benign Prostatic Hyperplasia; BMI, Body Mass Index; HLD, Hyperlipidemia (serum triglycerides > 150); KT, Kidney Transplant. Categorical variable shown as % (n). Continuous variables shown as mean ± standard deviation.

After propensity matching, 31,625 KT patients and 31,625 non-transplant patients were compared. KT was associated with a lower ED risk (RR 0.51 [0.48–0.55]; HR 0.32 [0.30–0.34]) (Table 2). More KT patients were ED-free at follow-up (85.2% vs 70.9%) and average follow-up was longer (2,020 vs 1,141 days). Figure 1 illustrates the ED-free rate over a 5 year period as well as over the entire follow-up period. The incidence rate per 1000 person years over the general 5 year period was 6.48 (95% CI 6.08–6.88) for the KT group, and for the no KT group it was 14.46 (95% CI 13.87–15.05).

Table 2

Rates of erectile dysfunction in dialysis-dependent men with advanced CKD treated with transplant vs continued on dialysis, before and after propensity score matching for demographics and comorbidities

Before Propensity Matching					
	Mean Follow-Up Duration (days) (interquartile range)	**Number of Patients with ED (% of Cohort)**	**Risk Ratio**	**Hazard Ratio**	**Disease-Free Survival (%)**
Transplant (33,063)	2,031 (2,032)	1,394 (4.2)	0.60 (0.57–0.63)	0.33 (0.31–0.35)	85.5
No Transplant (265,852)	893 (1,247)	18,758 (7.1)			72.8
After Propensity Matching					
	Mean Follow-Up Duration (days), (interquartile range)	**Number of Patients with ED (% of Cohort)**	**Risk Ratio**	**Hazard Ratio**	**Disease-Free Survival (%)**
Transplant (31,625)	2,020 (2,030)	1,364 (4.3)	0.51 (0.48–0.55)	0.32 (0.30–0.34)	85.2
No Transplant (31,625)	1,141 (1,649)	2,656 (8.4)			70.9
P < 0.001					

Table 3
Summary of Diagnostic and Billing Codes used in this Study

Inclusion Criteria	
Chronic Kidney Disease	Chronic Kidney Disease (stage IV [ICD-10 N18.4], and stage V [N18.5]), or End Stage Renal Disease (ESRD) (N18.6)
Erectile Dysfunction	ICD-10 N52 (Male Erectile Dysfunction), or new prescription of avanafil, sildenafil, vardenafil, or tadalafil 20 mg or 40 mg (RxNorm 1291301, 136411, 306674, and 358263, respectively) HCPS L7902 (Tension Ring, for Vacuum Erection Device), HCPS L7900 (Male Vacuum Erection System), RxNorm 598 (Alprostadil), HCPCS J0270 (Alprostadil Injection), CPT 54235 (Injection of Corpora Cavernosa with Pharmacologic Agents), RxNorm 7895 (Papaverine), HCPCS J2440 (Papaverine Injection), HCPCS J2760 (Phentolamine Injection), RxNorm 8153 (Phentolamine), SNOMED 26573008 (Implantation of Inflatable Penile Prosthesis), CPT 54401 (Insertion of Inflatable Penile Prosthesis), CPT 5405 (Insertion of Multi-Component, Inflatable Penile Prosthesis), HCPCS C1813 (Inflatable Penile Prosthesis)
Dialysis	Hemodialysis (ICD-10 Z99.2, ICD-9 39.95, CPT 1012752, 90935, 90937, or SNOMED 302497006)
Kidney Transplant	ICD 10 BT49, 0TY00Z1, 0TY10Z1, 0TY10Z2, 0TY00Z0, 0TY10Z0, and Z94.0; CPT 1008098, and SNOMED 175901007, 113127005, and 175902000
Propensity Matching Factors	
Demographics	Current age, age at index event, ethnicity (Hispanic or Latino, Not Hispanic or Latino, White, Black or African American, Asian, Native Hawaiian or other Pacific Islander, American Indian or Alaska Native)
Diagnoses	Type 2 Diabetes Mellitus (ICD-10 E11), BMI > 30 (TNX curated 9083), Essential (primary) Hypertension (ICD-10 I10), Secondary Hypertension (I15), Atherosclerosis (ICD-10 I70), Chronic Ischemic Heart Disease (ICD-10 I25), Alcohol related disorders (F10), Tobacco Use (Z72.0), Obstructive Sleep Apnea (ICD-10 G47.33), Triglycerides (Serum) > 150 (TNX curated 9004), Creatinine (Serum) (TNX curated 9024)

Incidence rates of new ED declined over the measured time intervals for both groups. In the KT group, there were 20.2 cases per 1000 person years in the first 6 months which decreased to 5.1 cases per 1000 person years in the year 4–5 time interval (Table 4a). The non-KT group had a higher incidence rate of 72.7 cases per 1000 person years in the first 6 months which decreased to 9.0 per 1000 person years by the year 4–5 time interval.

Table 4a
New ED diagnosis incidence rate over time

Period: P	Time period after index event	Number of patients newly diagnosed with ED (N)		Number of patients newly diagnosed with ED (%)		Incidence rate per 1,000 person years (95% CI)	
		KT	No KT	KT	No KT	KT	No KT
1	0–6 months	320	1,150	1.0	3.6	20.2 (18.0–22.5)	72.7 (68.6–76.9)
2	6 months – 1 year	158	467	0.50	1.5	10.0 (8.5–11.6)	29.5 (26.9–32.3)
3	1 year – 18 months	154	358	0.49	1.13	9.7 (8.3–11.4)	22.6 (20.3–25.0)
4	18 months – 2 years	129	318	0.41	1.01	8.2 (6.8–9.7)	20.1 (18.0–22.5)
5	2 years – 30 months	116	277	0.37	0.88	7.3 (6.0–8.8)	17.5 (15.5–19.8)
6	30 months – 3 years	138	270	0.44	0.85	8.7 (7.3–10.3)	17.1 (15.1–19.3)
7	3 years – 4 years	204	369	0.65	1.17	6.5 (5.7–7.5)	11.7 (10.6–13.0)
8	4 years – 5 years	161	286	0.30	0.37	5.1 (4.4–6.0)	9.0 (8.0–10.1)

Time variant analysis used to determine incidence rate per 1000 person years. P-value for group comparisons at each time point was < 0.001.

Table 4b
Incidence rate ratios between time periods for KT and No-KT groups

Time Period Comparison	KT	No KT
P2 vs P1	**0.50** (0.41–0.61)	**0.41** (0.37–0.45)
P3 vs P2	**0.97** (0.78–1.20)	**0.77** (0.68–0.87)
P4 vs P3	**0.84** (0.67–1.06)	**0.89** (0.78–1.01)
P5 vs P4	**0.89** (0.69–1.14)	**0.87** (0.76–1.00)
P6 vs P5	**1.19** (0.94–1.51)	**0.98** (0.85–1.13)
P7 vs P6	**0.75** (0.61–0.92)	**0.69** (0.60–0.80)
P8 vs P7	**0.78** (0.63–0.96)	**0.77** (0.65–0.90)

Time period definitions are from Table 4a. *Parenthesis represent 95% confidence intervals.*

Discussion

This was the first large database study to our knowledge that assessed rates of new onset ED after KT in men with dialysis-dependent CKD. We found that men with CKD who underwent KT were less likely to be diagnosed with ED following KT than those who were continued on hemodialysis without a KT. Rates of ED in the dialysis-dependent CKD population and in the post-transplant population were much lower than that commonly cited in the literature. Commonly cited numbers are as high as 79% of CKD patients on hemodialysis and 59% of post-transplant patients suffering from ED, as compared to our study, which found rates of 8.4% and 4.3%, respectively.[17–19] This is explained by the strict definition of new-onset ED that we used to ensure that we were analyzing comparable patient populations. Any new diagnoses in the KT group occurred after the KT diagnosis, which was important given that the two groups had the same index event of dialysis initiation in CKD patients. Despite variability compared to previously described ED rates, the strict definitions ensured the validity of the results and generalizability of findings.

The pathophysiology of ED in patients with CKD is multifactorial. From a cardiovascular perspective, CKD results in vascular calcification and endothelial dysfunction, which impairs penile perfusion.[20] In addition, CKD alters the ability of nitric oxide synthase - the enzyme responsible for synthesis of the potent vasodilator nitric oxide (NO) - to function, inhibiting vasodilation in the erectile bodies.[21–23] Testosterone deficiency may negatively impact erectile function, and testosterone also has been shown to decrease in CKD patients, likely secondary to de-prioritization of reproductive organs in the setting of systemic physiological decline.[24,25] As CKD progresses, chronic uremia can additionally alter the cavernosal cellular function, basement membranes, and connective tissue, further worsening erectile function. Bellinghieri et al. found that this effect was amplified by treatment duration of hemodialysis.[26] Chronic uremia also alters neuronal functioning, which can contribute to an inability to sustain an erection in these patients, as well as disrupting other facets of sexual function.[27]

Current literature has generally shown that these physiological changes are not corrected after initiating renal replacement therapy (RRT) with dialysis.[9,19,28,29] Prior studies have indicated that KT resolves many of the issues raised above, posing a possible mechanism for how studies have shown that KT often corrects sexual dysfunction.[12,15,19,30] Fundamental to these changes, renal transplant rapidly improves healthy levels of male androgens, which in addition to directly improving ED, associated research suggests may be associated with improved endothelial health.[31,32] This is supported by our study results showing lower rates of new ED after transplant. While in the minority, there are some studies that show an increase in ED rates after transplant.[33] The reason for this could be multifaceted. Of the literature cited here, most are based on surveys and questionnaires, rather than diagnostic codes mentioned in problem lists and other places in a patient's EMR.[34]

Of note, while prior studies have looked at improved ED severity after KT, our study is one of the first that looks at newly diagnosed ED after KT in patients who did not have ED prior to this. Unsurprisingly, rates of new ED diagnoses were higher in the no KT group than the KT group. However, it remains true that a large number (1,394 patients) were newly diagnosed with ED after KT, despite literature supporting that KT often resolves ED. One hypothesis is that this may be due to patients engaging with the medical system more often. This is supported by the observation that the patients receiving KT generally had longer follow-up times. Furthermore, KT commonly improves health with improved quality of life post-transplant.[35] Generally, as health improves, greater emphasis is placed on quality of life metrics such as sexual function, while chronic conditions have been associated with a neglect of these aspects of a patient's life.[36] Thus these new cases of ED post-transplant could indicate a greater concern about sexual function as health improves and activities of daily life become more routine. This suggests that ED may be underdiagnosed in CKD patients on dialysis, and interventions should be aimed at improving quality of life in these patients.

To account for the longer follow-up time in the KT group, we present incidence rates per 1000 person years in Table 4a and 4b. The incidence rates for all of the 6 month intervals from 0–3 years and the 1 year time periods from 3–5 years all demonstrated higher rates of ED diagnosis regardless of follow-up time in the no KT group as compared to the KT group. Interestingly, the highest rates of ED diagnosis occurred in the first 6 months following CKD diagnosis in both cohorts. This rate steadily declined through each successive time period as expected based on the observations in the Kaplan Meier curve (Fig. 1). An interesting increase in diagnosis was observed in the six month interval from 30 months to 3 years (time period 6 in Table 4a and 4b) in the KT group, however this slight increase was not statistically significant (Table 4b). When rate ratios (Table 4b) were compared, the decreases across several time periods in the first year and from year 3–5 were found to be significant based on calculated confidence intervals. The time variance across different follow-up schedules is an interesting nuance that we were not anticipating that is worthy of future research into factors that increase risk of ED diagnosis based on time to follow-up and diagnosis after index event.

Prior to propensity score matching, all comorbidities (except for secondary hypertension) were more common in the no-KT group than in the KT group. This suggests that the pre-matching no-KT cohort generally had a greater burden of comorbidities than the KT group (Table 1). The factors that regulate how patients are listed on a transplant registry make such a comparison complicated, but understanding these differences would be a fruitful area of future research.[37]

There were several strengths and limitations of the current study. Strengths of the study include the large sample size and wide variety and scope of HCO's from which the data was pulled. Furthermore, we were able to control for many confounding variables through a rigorous propensity matching assessment. However, given the large sample size, we inevitably cannot account for all the variables that may be impacting ED pathogenesis, particularly in the context of a multifactorial disease like ED. There was also no way to control for socioeconomic determinants of health in the propensity matching. Other limitations of the TriNetX database include that data is stratified by diagnostic and billing codes, which decreases the granularity of analyses that can be done. Furthermore, it is difficult to quantify patient-level data like the severity of ED symptoms from the database, and the complexity of the EHR means that errors such as misclassification and misdiagnosis can occur. The possibility of double counting increases if patients are seen at multiple HCO's.

Conclusion

This is one of the first assessments of new ED diagnoses after KT compared to dialysis. The rate of new diagnoses of ED in patients after KT is lower than that observed in patients with CKD who remain on dialysis. These observations support prior studies showing resolution of ED after KT in the dialysis dependent CKD population. However, rates of new ED in priorly unaffected patients was nonzero, suggesting that the improved health and focus on quality of life that comes post-transplant perhaps allows ED to now become a concern that may have been overlooked while the patient was on dialysis. Using a large collaborative network allows us to access a significant population that would be otherwise inaccessible and shows a trend towards a possible underdiagnosis of ED in this dialysis population. This reveals a potential area to improve the quality of life of CKD patients on dialysis who may not have access to immediate transplant.

Declarations

Competing Interest

The authors have no conflict of interest to disclose.

Ethics Declaration

Not applicable.

Funding Declaration

No funding was used.

Author Contribution

TS was the primary author and conducted the majority of the research and manuscript preparation. MD and GG contributed significantly to idea conceptualization, data collection, analysis, and writing. NS assisted with

conceptualization of the study and manuscript editing. BW and AT contributed to data analysis and provided editorial input. NT served as the senior author, overseeing the study design, interpretation of findings, and final manuscript revision.

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None

Data Availability

No datasets were generated or analyzed during the current study.

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Figures

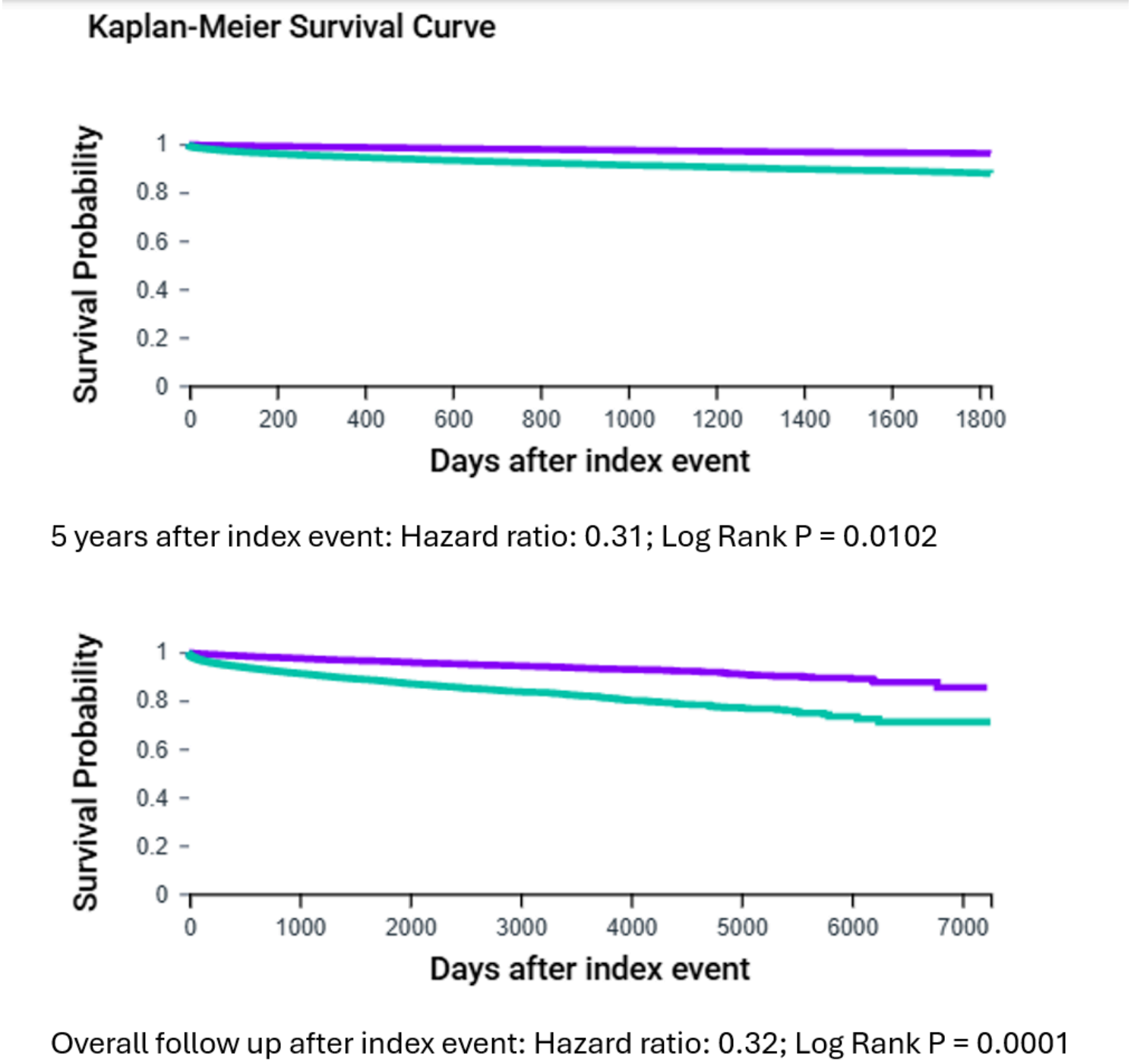


Figure 1

Kaplan-Meier Curve showing ED-free survival for both KT (purple) and no-KT (green) cohorts after propensity matching

Legend: Top graph: ED-free rates over a 5 year time period. The incidence rate per 1000 person years with 95% CI for the KT group over this time period is 6.48 (6.08–6.88), and for no KT it is 14.46 (13.87–15.05); Bottom graph: ED-free rates over entire study period; Both: purple line represents KT group, green line represents no-KT group.