

The Impact of Renal Replacement Therapy and Kidney Transplantation on Erectile Function and Reproductive Health in Men with Chronic Kidney Disease

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Abstract

Introduction: Chronic kidney disease (CKD) is a pressing medical and social problem accompanied by a wide range of complications, including erectile dysfunction (ED) and reproductive health disorders. **Objective:** To study the effect of renal replacement therapy (RRT) in the form of hemodialysis (HD) and kidney transplantation (KT) on erectile function (EF) and reproductive health of men with CKD. **Material and Methods:** Prospective observation of 477 male patients (mean age 35.1 ± 2.0 years) with CKD. 201 patients (42.1%) were on program HD, 276 patients (57.9%) underwent KT from a living related donor. The EF was assessed using the IIEF-5 score, doppler sonography of the penile arteries was used to diagnose hemodynamics in the vessels of the penis, the hormonal profile of patients was determined using testosterone, luteinizing hormone, and follicle-stimulating hormone levels, and reproductive function was determined using a spermogram at three stages of the study: initially, high azotemia and 12 months after RRT or KT. **Results:** In patients after KT, 65.6% of cases of EF were completely restored after one year, the proportion of moderate-mild erectile dysfunction decreased to 9.4%, and the mild form persisted in 25%. The average IIEF-5 score increased from 13.2 ± 0.1 to 21.2 ± 0.2 ($p < 0.001$), the average peak systolic velocity in the right cavernous arteries increased from 5.6 ± 0.1 to 7.2 ± 0.1 cm/s ($p < 0.001$), testosterone level—from 4.6 ± 0.1 to 5.6 ± 0.2 ng/ml ($p < 0.001$), the frequency

of normospermia—from 37.3% to 61.2% ($p < 0.001$). In patients on HD, ED progression was noted with a decrease IIEF-5 score from 21.9 to 9.7 points ($p < 0.001$). The average peak systolic velocity in the right cavernous arteries decreased from 6.5 ± 0.1 to 4.8 ± 0.1 cm/s ($p < 0.001$), testosterone level—from 5.1 ± 0.2 to 4.0 ± 0.2 ng/ml ($p < 0.001$), luteinizing hormone—from 8.9 ± 0.1 to 6.9 ± 0.1 ($p < 0.001$), follicle-stimulating hormone—from 6.3 ± 0.1 to 5.0 ± 0.1 ($p < 0.001$), the frequency of normospermia—from 59.2% to 50.7% ($p < 0.001$), with an increase in cases of pathological disorders of spermatogenesis in total up to 14.0% ($p < 0.001$). **Conclusion:** The obtained data indicate effective restoration of EF and fertility in most patients after KT. In patients on HD, there is persistence of multifactorial ED caused by vascular, hormonal and structural changes.

Keywords

Chronic Kidney Disease, Erectile Dysfunction, Renal Replacement Therapy, Kidney Transplantation

1. Introduction

Erectile dysfunction (ED) is a prevalent and debilitating complication of chronic kidney disease (CKD), affecting 70% - 86% of male patients, including those undergoing hemodialysis (77% - 84%) and peritoneal dialysis (up to 84%) [1]-[5]. The pathogenesis of ED and reproductive disorders (RD) in CKD is multifactorial, involving hormonal imbalances (e.g., hypogonadism, hyperprolactinemia), uremic toxicity, vascular dysfunction, and metabolic disturbances. Beyond its physical effects, ED profoundly impacts patients' psychological well-being, contributing to social withdrawal and reduced quality of life [2] [4] [5].

While renal replacement therapy (RRT) and kidney transplantation (KT) are life-sustaining interventions, their effects on erectile function (EF) remain incompletely understood. Existing data suggest that ED is pervasive among RRT recipients, with prevalence rates ranging from 41% to 98%, and worsens with prolonged dialysis duration. Notably, 50% of patients exhibit ED even in the pre-dialysis stage, escalating to 80% after initiating RRT [3]-[5].

Data confirming the impact of KT on EF remain limited due to the small number of studies, highlighting the need for further scientific research. Several studies indicate that KT not only prolongs life but also improves the quality of life in CKD patients. By normalizing hormonal disturbances, KT contributes to improved sexual health (libido) [6]-[9]. KT has been associated with improved hormonal profiles and libido, yet post-transplant ED persists in approximately 46% of patients. However, the mechanisms underlying these outcomes—particularly the role of vascular recovery, hormonal normalization, and graft function—are poorly characterized, and longitudinal data on EF post-KT are scarce [7] [10]-[12].

The International Index of Erectile Function (IIEF-5) questionnaire is widely

used in the diagnosis of ED and is considered the gold standard, as it covers the key aspects of sexual function. However, its results may be influenced by the patient's psycho-emotional state. In CKD patients, important risk factors for ED include hormonal imbalances (low testosterone, hyperprolactinemia), as well as metabolic disorders, including dyslipidemia and impaired glucose metabolism. To assess vascular disorders, instrumental methods such as pharmacodopplerography and shear wave elastography are used; these allow for the identification of fibrotic changes and differentiation between vasculogenic and non-vasculogenic forms of ED [13]-[16]. Critically, no consensus exists on optimal strategies for ED management in CKD, and the impact of RRT modality or KT timing on EF remains unresolved.

Despite the high prevalence of ED in CKD patients, issues related to the diagnosis, pathogenesis, and correction of EF remain insufficiently addressed in clinical practice, emphasizing the relevance of further research. Special attention is given to analyzing the vascular, hormonal, and structural changes occurring at various stages of the disease, during RRT, or after KT, to develop effective approaches for their correction.

Objective: to study the effect of RRT in the form of hemodialysis (HD) and KT on EF and reproductive health of men with CKD.

2. Material and Methods

The study was based on a prospective analysis of treatment outcomes in 477 men with CKD, who underwent KT from living related donors ($n = 276$) at the Republican Specialized Scientific Practical Medical Center of Surgery named after Academician V. Vakhidov (Tashkent, Uzbekistan), and those who were receiving RRT and treatment for ED ($n = 201$) at the Republican Specialized Scientific Practical Medical Center of Urology (Tashkent, Uzbekistan). The mean age of the patients was 35.1 ± 2.0 years. The majority of patients were young adults (aged 18 - 44 years), accounting for 82.4%. Middle-aged patients (45 - 59 years) comprised 15.1%, and elderly patients (60 - 74 years) accounted for only 2.5%.

The primary cause of stage 5 CKD in most cases (88.3%) was chronic glomerulonephritis. Less frequent causes included polycystic kidney disease (2.9%), CKD of unknown etiology (2.7%), urolithiasis (2.3%), chronic pyelonephritis (1.7%), type 2 diabetes mellitus, and congenital anomalies of the urinary system (1.0% each).

The study was conducted in accordance with the Declaration of Helsinki. All patients provided informed consent, and the protocol was approved by the local ethics committee of the Republican Specialized Scientific Practical Medical Center of Urology (Tashkent, Uzbekistan).

Inclusion criteria for the study were: male patients with CKD, preserved EF, a regular sexual partner, treatment in the form of RRT ($n = 201$), stable graft function after KT ($n = 276$), and absence of other comorbidities in the acute or decompensated stage.

In general, the study included the following stages of patient follow-up with EF assessment:

- Initial stage—evaluation of EF before the development of advanced renal failure;
- Stage of severe azotemia—during stage 5 CKD, characterized by accumulation of uremic toxins and deterioration of systemic functions, including EF;
- 12 months after initiation of RRT or KT—long-term evaluation reflecting the final outcome of therapy on EF, including complete or partial restoration of EF.

EF was assessed using the International Index of Erectile Function (IIEF-5) with classification as follows: severe ED (≤ 7 points), moderate (8 - 11 points), mild to moderate (12 - 16 points), mild (17 - 21 points), and no ED (22 - 25 points).

To assess vasculogenic ED we used the high-resolution penile Doppler ultrasound (HR-PDU) with intracavernosal prostaglandin E1 (PGE1) injection. Parameters such as peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistive index (RI) were measured for subclassifying ED.

Shear-wave elastography further quantifies penile fibrosis by measuring tissue stiffness (kPa).

Hormonal profile (testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH)) was analyzed using enzyme-linked immunosorbent assay (ELISA). Reproductive function was evaluated by semen analysis, assessing the prevalence of normozoospermia, asthenozoospermia, oligozoospermia, OAT syndrome (oligoasthenoteratozoospermia), and azoospermia in the overall patient sample.

Both RRT and KT groups received the following protocol for 12 weeks, unless contraindicated:

Phosphodiesterase-5 inhibitors (PDE5i): Sildenafil 50 mg (or tadalafil 10 mg) on demand, adjusted to 100 mg (or 20 mg) if ineffective after 4 weeks.

Vacuum Erection Device (VED): Daily use (10 minutes at 100 - 150 mmHg) with a tension ring applied for ≤ 30 minutes.

A special set of pelvic floor muscle exercises aimed at improving penile blood flow was also implemented, in addition to vacuum therapy: the first 10 sessions were conducted daily, and the remaining 12 sessions with 2-day intervals. Physiotherapy using a specialized device was applied for 15 minutes over 10 consecutive days.

Immunosuppressants in KT Group: Target trough levels of Tacrolimus—5 - 10 ng/mL (early post-KT) or 3 - 7 ng/mL (maintenance). Target trough levels of Cyclosporine—100 - 200 ng/mL; mTOR inhibitors (e.g., sirolimus): Levels 4 - 12 ng/mL. Prednisone ≤ 5 mg/day.

Descriptive and comparative statistical methods were used to analyze the obtained data. Data collection, adjustment, systematization, and result visualization were performed using Microsoft Office Excel 2016. Statistical analysis was conducted using IBM SPSS Statistics v.26 (developed by IBM Corporation, USA). Descriptive statistics were used to characterize clinical and demographic indicators,

including calculations of means (M), standard errors of the mean (m), and percentage distributions. One-way analysis of variance (ANOVA) was used to assess statistically significant differences across various follow-up stages. The chi-square (χ^2) test was used for categorical variables.

3. Results

In the initial period, in the KT group, the majority of patients (52.5%) showed no signs of ED according to the IIEF-5 scale, while 47.5% had a mild form of ED (Table 1). However, at the stage of severe azotemia, all patients lost normal erectile function: 87.0% had mild to moderate ED, and 12.3% had moderate ED. Twelve months after transplantation, normal erectile function had fully recovered in 65.6% of patients, the proportion of mild to moderate ED decreased to 9.4%, and mild ED persisted in 25% of patients.

Table 1. Dynamics of ED severity according to the IIEF-5 scale at different stages of the study.

ED severity according to the IIEF-5		Initial	High azotemia stage	12 months after KT/start of RRT
KT group (n = 276)				
Severe ED	n	0	0	0
	%	0.0%	0.0%	0.0%
Moderate	n	0	34	0
	%	0.0%	12.3%	0.0%
Moderate-mild	n	0	240	26
	%	0.0%	87.0%	9.4%
Mild	n	131	2	69
	%	47.5%	0.7%	25.0%
No ED	n	145	0	181
	%	52.5%	0.0%	65.6%
Total	n	276	276	276
	%	100.0%	100.0%	100.0%
HD group (n = 201)				
Severe ED	n	0	0	28
	%	0.0%	0.0%	13.9%
Moderate	n	0	8	132
	%	0.0%	4.0%	65.7%
Moderate-mild	n	0	186	41
	%	0.0%	92.50%	20.4%
Mild	n	75	7	0

Continued

	%	37.3%	3.50%	0.0%
No ED	n	126	0	0
	%	62.7%	0.00%	0.0%
Total	n	201	201	201
	%	100%	100%	100%

Note: n—number of cases, IIEF-5—international index of erectile function, ED—erectile dysfunction, KT—kidney transplantation, RRT—renal replacement therapy, HD—hemodialysis.

In the HD group, at the initial stage of the study, 75 patients (37.3%) had mild ED, while 126 patients (62.7%) showed no signs of ED. However, as the underlying disease progressed to end-stage renal disease (stage 5 CKD) and during severe azotemia, all patients developed ED of varying severity—from mild to moderate—according to the IIEF-5 scale. After 12 months of RRT, no improvement in erectile function was observed. On the contrary, in a significant proportion of patients, dysfunction worsened, and 13.9% of patients developed severe ED (Table 1).

Before KT, the mean IIEF-5 score was 21.6 ± 0.1 ; however, during the stage of severe azotemia, it decreased to 13.2 ± 0.1 , indicating a significant deterioration in erectile function. In the first three months postoperatively, partial improvement was observed with a score of 15.0 ± 0.1 , which increased to 18.4 ± 0.2 after six months, and nearly returned to the baseline level one year after KT, reaching 21.2 ± 0.2 ($F(4, 1375) = 702.33$; $p < 0.001$).

In the HD group, at the initial stage (before azotemia progression), the mean IIEF-5 index was 21.9 ± 0.1 , reflecting a pronounced reduction in EF, yet with preserved sexual activity. During the stage of severe azotemia, further deterioration was noted—the IIEF-5 dropped to 13.7 ± 0.1 , indicating a significant decline in EF. One year after the initiation of RRT, an even more pronounced decrease was observed, with the IIEF-5 dropping to 9.7 ± 0.1 ($F(2, 600) = 2418.29$; $p < 0.001$), signifying a critical impairment of EF despite ongoing therapy.

In the KT group, alongside improvement in EF, changes were also observed in penile blood flow, reflected in PeakSV values of the cavernosal and dorsal arteries (Table 2). Baseline PeakSV values were 6.5 ± 0.1 cm/s in the right cavernosal artery, 6.3 ± 0.1 cm/s in the left cavernosal artery, and 12.4 ± 0.2 cm/s in the dorsal artery. During the stage of severe azotemia, these values decreased to 5.6 ± 0.1 , 5.4 ± 0.1 , and 10.7 ± 0.2 cm/s, respectively, indicating reduced arterial inflow. Over the first year following KT, blood flow progressively improved and exceeded baseline values, reaching 7.2 ± 0.1 , 7.1 ± 0.1 , and 13.9 ± 0.2 cm/s, respectively ($F(4, 1375) = 194.69$; $p < 0.001$; $F(4, 1375) = 68.40$; $p < 0.001$; $F(4, 1375) = 43.09$; $p < 0.001$).

Analysis of PeakSV dynamics during the observation period in CKD patients on RRT (Table 2) showed that in the right cavernosal artery, baseline PeakSV was 6.5 ± 0.1 cm/s, decreasing to 5.6 ± 0.1 cm/s during severe azotemia (a 13.8% re-

duction), and further declining to 4.8 ± 0.1 cm/s after one year on HD ($p < 0.001$). Similar trends were observed in the left cavernosal artery: the initial value was 6.3 ± 0.1 cm/s, 5.4 ± 0.1 cm/s during azotemia, and 4.7 ± 0.1 cm/s after one year on HD ($p < 0.001$). The dorsal artery, initially showing higher values (12.2 ± 0.2 cm/s), also demonstrated progressive deterioration: 10.5 ± 0.2 cm/s during azotemia and 9.0 ± 0.2 cm/s one year after HD initiation ($p < 0.001$).

Table 2. Mean PeakSV in cavernosal arteries according to doppler ultrasound.

Research stage	PeakSV on the right cavernous artery M \pm m	PeakSV on the left cavernous artery M \pm m	PeakSV on the dorsal artery M \pm m
KT group (n = 276)			
Initially	6.5 ± 0.1	6.3 ± 0.1	12.4 ± 0.2
High azotemia stage	5.6 ± 0.1	5.4 ± 0.1	10.7 ± 0.2
12 months after KT	7.2 ± 0.1	7.1 ± 0.1	13.9 ± 0.2
ANOVA	F (4, 1375) = 194.7; p < 0.001	F (4, 1375) = 68.4; p < 0.001	F (4, 1375) = 43.1; p < 0.001
HD group (n = 201)			
Initially	6.5 ± 0.1	6.3 ± 0.1	12.2 ± 0.2
High azotemia stage	5.6 ± 0.1	5.4 ± 0.1	10.5 ± 0.2
12 months after the start of RRT	4.8 ± 0.1	4.7 ± 0.1	9 ± 0.2
ANOVA	F (2, 600) = 287.65; p < 0.001	F (2, 600) = 77.81; p < 0.001	F (2, 600) = 81.40; p < 0.001

Note: n—number of cases, M—mean value, m—standard error of the arithmetic mean, KT—kidney transplantation, RRT—renal replacement therapy, HD—hemodialysis, PeakSV—peak systolic velocity

In the analysis of hormonal status in the KT group, a consistent dynamic was observed in the levels of testosterone, LH, and FSH (**Table 3**). The testosterone level, initially 5.2 ± 0.2 ng/mL, decreased to 4.6 ± 0.1 ng/mL during the stage of advanced azotemia. After one year, it increased to 5.6 ± 0.2 ng/mL (F (4, 1375) = 16.1; $p < 0.001$). LH decreased from 8.9 ± 0.1 mIU/mL at baseline to 7.8 ± 0.1 mIU/mL in the stage of high azotemia, and then gradually increased, reaching 9.5 ± 0.1 mIU/mL one year after KT (F (4, 1375) = 81.2; $p < 0.001$). A similar trend was noted for FSH: a decrease from 6.3 ± 0.1 to 5.5 ± 0.1 mIU/mL during high azotemia, followed by recovery to 6.8 ± 0.1 mIU/mL after one year (F (4, 1375) = 22.5; $p < 0.001$).

In the HD group, the analysis of hormonal status (**Table 3**) revealed a progressive decrease in testosterone, LH, and FSH levels as the patients' condition worsened and RRT continued. At the baseline stage, the testosterone level was 5.1 ± 0.2 ng/mL, which decreased to 4.5 ± 0.2 ng/mL during the stage of high azotemia,

and further declined to 4.0 ± 0.1 ng/mL after one year of RRT ($F(2, 600) = 23.49$; $p < 0.001$). A progressive decrease in LH levels ($F(2, 600) = 127.81$; $p < 0.001$) and FSH levels ($F(2, 600) = 47.88$; $p < 0.001$) was also observed against the background of deteriorating kidney function and ongoing RRT. This may be associated with suppression of the hypothalamic-pituitary-gonadal axis, metabolic disorders, and possible dysregulation of gonadotropin secretion, leading to the development of hypogonadotropic hypogonadism.

Table 3. Dynamics of meanlevels of key hormones regulating reproductive function.

Research stage	Testosterone (ng/mL) M ± m	LH (mIU/mL) M ± m	FSH (mIU/mL) M ± m
KT group (n = 276)			
Initially	5.2 ± 0.2	8.9 ± 0.1	6.3 ± 0.1
High azotemia stage	4.6 ± 0.1	7.8 ± 0.1	5.5 ± 0.1
12 months after KT	5.6 ± 0.2	9.5 ± 0.1	6.8 ± 0.1
ANOVA	$F(4, 1375) = 16.1$; $p < 0.001$	$F(4, 1375) = 81.2$; $p < 0.001$	$F(4, 1375) = 22.5$; $p < 0.001$
HD group (n = 201)			
Initially	5.1 ± 0.2	8.9 ± 0.1	6.3 ± 0.1
High azotemia stage	4.5 ± 0.2	7.8 ± 0.1	5.6 ± 0.1
12 months after the start of RRT	4 ± 0.1	6.9 ± 0.1	5 ± 0.1
ANOVA	$F(2, 600) = 23.49$; $p < 0.001$	$F(2, 600) = 127.81$; $p < 0.001$	$F(2, 600) = 47.88$; $p < 0.001$

Note: n—number of cases, M—mean value, m—standard error of the arithmetic mean, KT—kidney transplantation, RRT—renal replacement therapy, HD—hemodialysis, LH—luteinizing hormone, FSH—follicle-stimulating hormone.

In the KT group, during the stage of severe azotemia (before transplantation), there was a marked deterioration of spermatogenesis, including a decrease in normospermia and an increase in the frequency of pathological conditions (asthenozoospermia, oligozoospermia, oligoasthenoteratozoospermia (OAT-syndrome), and azoospermia). After 12 months, fertility recovery was observed, with an increase in the rate of normozoospermia to 61.2% and a decrease in the rate of asthenozoospermia to 27.5% ($\chi^2 = 165.5$, $p < 0.001$). These results confirm the positive effect of KT on the restoration of fertility in CKD patients (Figure 1).

Analysis of the KT outcomes showed that 65.6% (181 out of 276) of patients experienced complete restoration of EF within one year after KT, primarily associated with the normalization of vascular function, improved blood flow in the cavernosal arteries, and restoration of hormonal balance. However, in 95 out of 276 recipients (34.4%), moderate and mild degrees of ED persisted one year after KT, despite the normalization of renal function and improvement of systemic cir-

ulation. These patients were categorized into five main groups of factors that could contribute to the persistence of ED (**Table 4**).

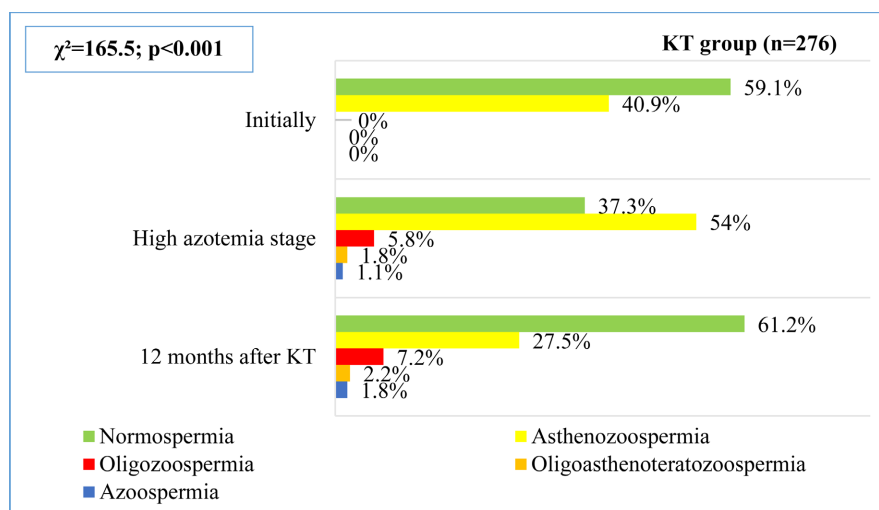


Figure 1. Distribution of patients in the KT group based on the dynamics of changes in the spermogram.

Table 4. Summary of data on possible reasons for the persistence of ED in kidney transplant recipients.

Possible causes of ED persistence after LT	n	%
Vascular disorders (atherosclerosis, fibrosis of the cavernous bodies, diabetic angiopathy)	30	31.6%
Hormonal and metabolic disorders (hypogonadism, hyperprolactinemia)	25	26.3%
Psychoemotional and neurological factors (depression, anxiety, polyneuropathy)	18	18.9%
Vascular anastomosis with the internal iliac artery	12	12.6%
Energy deficiency and sarcopenia (low body mass index, catabolism)	10	10.5%

In the HD group, semen analysis with assessment of the degree of spermatogenesis disorders also made it possible to determine whether RRT had a positive or negative impact on the reproductive health of CKD patients (**Figure 2**). According to the results, there was a progressive deterioration in sperm quality, with an increase in the incidence of pathological spermatogenesis disorders (asthenozoospermia, oligozoospermia, and oligoasthenoteratozoospermia) reaching a combined total of 14.0% ($\chi^2 = 30.87$, $p < 0.001$).

Thus, the most significant changes in EF and reproductive health occurred during the progression of CKD to the stage of high azotemia, which is associated with increasing uremic intoxication, as well as endocrine and vascular disorders. Further treatment with RRT and specific ED therapy did not lead to substantial improvement, whereas in the KT group, the positive impact of KT on fertility restoration was confirmed.

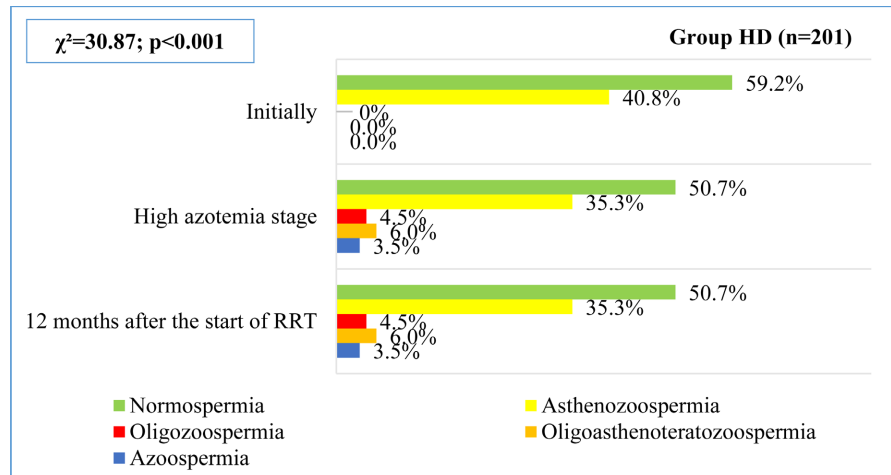


Figure 2. Distribution of patients with CKD and ED receiving RRT based on the dynamics of changes in spermogram.

4. Discussion

ED and RD in patients with CKD remain highly relevant areas of research, especially considering their impact on patients' quality of life and overall health status. In recent years, numerous studies have been published focusing on diagnostic methods that help identify and differentiate the causes of ED. Research emphasizes that hormonal and vascular disorders—including hypogonadism, hyperprolactinemia, and ischemia of the cavernous bodies—play a key role in the pathogenesis of persistent ED.

One of the most commonly used diagnostic tools for ED is the IIEF-5. Salonia A. *et al.* note that this questionnaire remains the gold standard for diagnosing ED, as it covers key aspects of sexual function, including erectile capacity, orgasmic function, sexual desire, and overall sexual satisfaction [13]. However, Neijenhuijs K.I. *et al.* point out that IIEF-5 scores may vary depending on the patient's psychological state, and patients with psychogenic ED tend to demonstrate higher scores compared to those with organic causes of ED [14].

It has been reported that despite the overall positive effects of KT, normal EF does not recover in all patients. Rahman I.A. *et al.*, in a systematic review, demonstrated that although KT indeed improves IIEF-5 scores, 20% - 50% of patients continue to experience impaired EF. The key contributing factors included the duration of dialysis, age, the type of vascular anastomosis, and the use of immunosuppressants [6].

Spirito L. *et al.* evaluated erectile and ejaculatory functions at 6 and 12 months after KT and found that men's sexual health significantly deteriorated at 6 months and remained stable over the course of a year. In the study population, a significant decrease in the mean IIEF-5 score was observed at 6 months ($p < 0.001$), which remained unchanged at 12 months post-KT ($p = 0.228$). This decrease was found to correlate with ejaculatory disorders [17].

The study by El Hennawy H.M. *et al.* showed that in patients with stage 5 CKD undergoing dialysis, EF deteriorated, whereas a positive trend was observed after KT. In a single-center cross-sectional study, the authors used the IIEF-5 to assess ED one month before and one year after KT and found significantly better results in kidney transplant recipients compared to dialysis patients [18].

In our study, the dynamics of IIEF-5 scores demonstrated a gradual recovery of EF after KT. Prior to transplantation, during the stage of high azotemia, all patients exhibited some degree of ED. After 12 months, 65.6% of patients had regained normal EF, as confirmed by the increase in mean IIEF-5 scores from 13.2 ± 0.1 to 21.2 ± 0.2 ($p < 0.001$).

In addition to questionnaire-based assessments, laboratory markers potentially associated with ED in CKD patients are being actively investigated. Wang Q. *et al.* reported that low testosterone levels and hyperprolactinemia are among the key risk factors for ED in CKD patients [15]. The study by Zhang D.K. *et al.* confirmed that dyslipidemia and impaired glucose metabolism also play an important role in the development of vascular disorders that contribute to ED [16].

Miron A. *et al.* presented data indicating that 70% of patients continued to experience ED 12 months after KT despite having normal laboratory parameters. In these cases, pharmacological and vascular factors had a greater impact than testosterone or creatinine levels [11].

Antonucci M. *et al.* found that testosterone and prolactin levels were directly correlated with the severity of ED: 65% of patients with hypogonadism and hyperprolactinemia had persistent moderate ED despite normal graft function [19].

A meta-analysis by Kang X. *et al.*, which included nine studies, showed that after KT, the testosterone level increased by an average of 1.1 ng/mL, prolactin decreased by 6.2 ng/mL, and the frequency of ED decreased by 32% compared to patients on dialysis. These findings confirm the hormonal dependence of EF restoration after KT [20].

According to our data, patients after KT demonstrated a consistent and statistically significant improvement in testosterone, LH, and FSH levels: testosterone increased to 5.6 ± 0.2 ng/mL, exceeding baseline values; LH reached 9.5 ± 0.1 mIU/mL, and FSH— 6.8 ± 0.1 mIU/mL.

To assess vascular changes in ED, instrumental methods such as Doppler ultrasound and shear wave elastography are actively used. Zhang D.K. *et al.* studied the use of shear wave elastography to differentiate between vasculogenic and non-vasculogenic ED. Their data demonstrated that the method has high sensitivity and specificity in detecting fibrotic changes in the corpora cavernosa in CKD patients and post-transplant recipients [16].

Morphological studies of penile and testicular tissues are also of interest in the diagnosis of ED. Perri A. *et al.* analyzed biopsies of the corpora cavernosa and identified pronounced fibrotic changes that persisted even after successful KT, which may explain the continued presence of ED in this patient population [21]. Similarly, Lundy S.D. *et al.* examined histological changes in the testes before and

after KT and found that despite the resolution of uremia, most patients still exhibited signs of impaired spermatogenesis and morphological changes in Sertoli cells [22]. Moreover, their study confirmed that normalization of reproductive hormone levels after KT contributes to improved sperm quality, including concentration, motility, and morphology [22]. However, the use of immunosuppressive drugs—especially calcineurin inhibitors and mTOR inhibitors—may negatively affect spermatogenesis and reproductive outcomes.

In our study, we also evaluated vascular changes in ED by assessing penile arterial blood flow using Doppler ultrasound. The results demonstrated a gradual restoration of vascular flow after KT: by the 12th month, PeakSV exceeded baseline levels, reaching 7.2 ± 0.1 cm/s (right cavernosal artery), 7.1 ± 0.1 cm/s (left cavernosal artery), and 13.9 ± 0.2 cm/s (dorsal artery), which correlated with improved EF ($p < 0.001$).

Thus, both the literature and our findings confirm that KT promotes the restoration of EF in most CKD patients, as evidenced by improvements in IIEF-5 scores, hormonal status, and penile arterial blood flow. After 12 months, normal EF was restored in 65.6% of patients, and testosterone, LH, and FSH levels exceeded baseline values. Improved vascular flow also showed a significant correlation with increased EF. Nevertheless, some patients continue to experience ED due to vascular, hormonal, and pharmacological factors. This highlights the need for a comprehensive approach to diagnosis and treatment, including hormonal and vascular monitoring, especially under immunosuppressive therapy.

In the study by Ye H. *et al.*, the prevalence and factors associated with ED were analyzed in 176 patients undergoing RRT (mean age— 43.2 ± 9.6 years, median dialysis duration—25 [13.0 - 39.8] months, glomerular filtration rate— $1.2 [0.2 - 3.5]$ mL/min/1.73 m²). ED was diagnosed in 80.6% of patients. After adjusting for other variables, younger age ($p = 0.014$), low daily urine output ($p = 0.032$), and elevated C-reactive protein (CRP) levels ($p = 0.043$) were identified as independent predictors of ED. A positive correlation was found between daily urine volume and IIEF-5 scores ($p = 0.011$), whereas older age ($p = 0.001$) and CRP ($p = 0.017$) were negatively correlated with these scores [3].

In a study by Lau L.C. *et al.*, which included 164 men with CKD on maintenance HD, ED was identified in 93.3% of the participants. About 63% reported severe ED, and factors such as age and diabetes mellitus were significantly associated with ED [4].

In the study by Tekkarismaz N. *et al.*, 51 patients were included, of whom 31 were on hemodialysis and 20 on peritoneal dialysis. ED was found to be more prevalent among patients undergoing peritoneal dialysis. Factors associated with increased prevalence of ED included older age, arterial hypertension, iron therapy, hyperlipidemia, and the presence of depression [5].

Savadi H. *et al.* analyzed the dynamics of EF in men undergoing hemodialysis. The most common impairments were dissatisfaction with intercourse and overall satisfaction (100%), sexual desire (96.7%), orgasmic function (93.3%), and EF

(90%). After six months of hemodialysis, all EF parameters significantly improved ($p = 0.001$). The authors suggested that prolonged hemodialysis may positively influence various aspects of sexual health, including erection, orgasm, sexual desire, and overall satisfaction in patients with CKD [23].

Selvi I. *et al.* reported that dialysis adequacy was a significant factor in reducing the risk of ED in men ($p = 0.019$) and in women ($p = 0.041$). In women, ED was also associated with depression ($p = 0.002$). In men, ED strongly correlated with physical functioning ($r = 0.524$; $p = 0.032$), social functioning ($r = 0.565$; $p = 0.042$), and general health status ($r = 0.693$; $p = 0.037$). In contrast, in women, strong correlations were observed with anxiety ($r = 0.697$; $p = 0.002$) and depression ($r = 0.738$; $p = 0.001$). The researchers concluded that inadequate dialysis worsens ED and reduces quality of life, especially in men [24].

Ahmed A.F. *et al.* evaluated the relationship between RRT duration, EF, and gonadal hormone levels in men with end-stage CKD. The overall prevalence of ED was 78.8%, with 31.2% suffering from its severe form. The frequency of ED did not differ significantly across dialysis duration: ≤ 5 years—79.7%, 5 - 10 years—76.5%, >10 years—80.0% ($p > 0.05$). Analysis of serum gonadotropin and sex hormone levels revealed the following abnormal rates: FSH—5.1%, LH—1.6%, testosterone—18.6%, prolactin—90%, estradiol—0.0%. No statistically significant associations were found between RRT duration and IIEF-5 scores or hormone levels. Thus, the authors did not identify a reliable link between dialysis duration, testosterone levels, and EF in end-stage CKD patients, suggesting that other mechanisms such as endothelial dysfunction and vascular damage may have a greater impact [25].

According to Chou J. *et al.*, the prevalence of ED among patients receiving RRT is significantly higher than in the general population, reaching up to 83% [26]. Similar results were obtained by Antonucci M. *et al.*, who noted that ED is more common in patients on long-term RRT compared to kidney transplant recipients [19]. At the same time, the study by El H.M. Hennawy *et al.* showed that the duration of pre-transplant dialysis had no significant effect on EF improvement after KT [18].

Our study showed that the progression of CKD to end-stage renal disease and the implementation of RRT were accompanied by a deterioration in erectile and reproductive function. RRT did not lead to EF restoration and, on the contrary, exacerbated vascular, hormonal, and anatomical impairments, emphasizing the need for a comprehensive approach to ED treatment in this population. At the initial stage of the study, among 201 CKD patients, 62.7% showed no signs of ED, while 37.3% had mild ED. However, with disease progression (high azotemia stage) and the initiation of RRT, 100% of patients developed ED of varying severity. After 12 months of RRT, EF did not improve; on the contrary, 13.9% of patients developed severe ED.

5. Limitations and Difficulties

The study did not include a group of healthy individuals, which limits the inter-

pretation of causal relationships.

The assessment was conducted within 12 months after the initiation of RRT or KT, which does not allow for conclusions about the long-term dynamics of sexual and reproductive function.

6. Conclusion

The obtained data indicate effective restoration of EF and fertility in the majority of patients after KT and comprehensive correction of residual vascular, hormonal, and psycho-emotional disorders. The progression of CKD to end-stage renal failure and the implementation of RRT are accompanied by a deterioration in erectile and reproductive function due to vascular, hormonal, and structural changes.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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