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IMPACT OF KIDNEY TRANSPLANTATION ON ERECTILE FUNCTION AND REPRODUCTIVE HEALTH IN MEN WITH CHRONIC KIDNEY DISEASE

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Objective: to evaluate the impact of kidney transplantation (KT) on erectile function and reproductive health in men with chronic kidney disease (CKD). **Materials and methods.** A prospective study was conducted involving 276 male patients (mean age 44.3 ± 5.8 years) with CKD who underwent KT from a living related donor. Erectile function was assessed using the International Index of Erectile Function (IIEF-5). Penile hemodynamics were evaluated by Doppler ultrasonography of the penile arteries, while hormonal status was determined by measuring serum testosterone, luteinizing hormone, and follicle-stimulating hormone (FSH) levels. Reproductive function was assessed by semen analysis and testicular volume measurements at five time points: baseline, at high azotemia, and at 3, 6, and 12 months post-transplantation. Management of post-transplant erectile dysfunction included phosphodiesterase type 5 inhibitors (IIEF-5, 5 mg daily for 3 months, followed by 20 mg on demand), pelvic floor muscle exercises, vacuum therapy, and physiotherapy. **Results.** After 12 months of follow-up, erectile function was fully restored in 65.6% of patients. The proportion of moderate-to-mild erectile dysfunction decreased to 9.4%, while mild dysfunction persisted in 25% of patients, primarily due to residual vascular, hormonal, and psychoemotional factors. The mean IIEF-5 score increased significantly from 13.2 ± 0.1 to 21.2 ± 0.2 ($p < 0.001$). The average peak systolic velocity in the right cavernous artery rose from 5.6 ± 0.1 cm/s to 7.2 ± 0.1 cm/s ($p < 0.001$). Serum testosterone levels increased from 4.6 ± 0.1 ng/ml to 5.6 ± 0.2 ng/ml ($p < 0.001$), and the proportion of patients with normospermia grew from 37.3% to 61.2% ($p < 0.001$). Erectile dysfunction persisted in 34.4% of patients despite therapy. **Conclusion.** The findings demonstrate a significant restoration of erectile function and fertility in most patients following KT and supported by comprehensive management of residual vascular, hormonal, and psychoemotional disorders.

Keywords: chronic kidney disease, erectile dysfunction, kidney transplantation, hormonal status, penile Doppler ultrasound, spermatogenesis.

INTRODUCTION

Erectile dysfunction (ED) is a significant complication of chronic kidney disease (CKD), affecting approximately 70–86% of patients, including those on hemodialysis (77–84%) and peritoneal dialysis (up to 84%) [1–3]. In recent decades, the mechanisms underlying ED and reproductive disorders in CKD have been actively investigated. These conditions are typically multifactorial, resulting from a combination of hormonal imbalances, uremic toxicity, vascular lesions, and metabolic problems. The importance of this issue extends beyond its physical consequences, as ED also profoundly affects patients' psycho-emotional state, often leading to social isolation and a reduced overall quality of life [4–6].

The effect of kidney transplantation (KT) on erectile function (EF) has drawn considerable attention, as KT not only prolongs survival but also enhances quality of life in patients with CKD. By normalizing hormonal function, KT can improve patients' sexual health (libido) [7–10]. However, the prevalence of ED after KT remains high at 46% [8, 11–13].

Data confirming the effect of KT on EF remain limited due to the small number of available studies, underscoring the need for further scientific research. The relevance of the present study lies in the comprehensive assessment of factors influencing EF recovery in patients after KT.

Unlike previous fundamental studies that focused on histological research, we presented clinically significant correlations between restoration of penile hemodynamics, hormonal balance, and spermatogenesis indicators. The novelty of this study lies in the integration of long-

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term prospective observation with a simultaneous evaluation of reproductive health and varicocele, an approach not previously implemented within a single research protocol. This methodology enabled us to identify key predictors of successful EF recovery that are relevant to clinical practice.

The aim of the study was to evaluate the effect of KT on EF and reproductive health in men with CKD.

MATERIALS AND METHODS

This study was based on a prospective analysis of treatment outcomes in 276 men with CKD who underwent KT from a living related donor at the Republican Specialized Scientific and Practical Medical Center for Surgery (Tashkent, Uzbekistan) and subsequently received treatment for ED at the Republican Specialized Scientific and Practical Medical Center for Urology (Tashkent, Uzbekistan).

Patient mean age was 34.9 ± 1.9 years. The majority (83.7%) were young adults (18–44 years), while 12.3% were middle-aged (45–59 years) and 4.0% were elderly (60–74 years).

The primary cause of stage 5 CKD was chronic glomerulonephritis (88.8%). Other causes included polycystic kidney disease (2.9%), CKD of unknown etiology (2.5%), urolithiasis (2.2%), chronic pyelonephritis (1.4%), type II diabetes mellitus, and congenital anomalies of the urinary tract (1.1% each).

The study complied with the Helsinki Declaration. All participants gave informed consent, and the study protocol was approved by the local ethics committee.

The inclusion criteria were as follows: men with preserved EF, a permanent sexual partner, stable graft function, and no concomitant diseases in the acute or decompensated stage (such as diabetes or stage II–III arterial hypertension).

The study design included the following stages of observation and EF assessment in patients with CKD:

– Baseline stage – assessment of EF status prior to development of severe renal failure.
– High azotemia stage – stage 5 CKD, characterized by accumulation of uremic toxins and systemic dysfunction, including impaired EF.
– Three months after KT – the first follow-up period, reflecting the early adaptation phase, initial stabilization of hormonal levels, and improvement in hemodynamic parameters.
– Six months after KT – mid-term assessment, during which further recovery of endothelial function and hormonal balance is expected.
– Twelve months after KT – long-term assessment to record the final therapeutic outcomes, including complete or partial restoration of EF.

To ensure data representativeness at the baseline stage (before the onset of severe chronic renal failure), the study included patients who had been registered at the transplant center as candidates for living-related KT and had undergone standard pre-transplant evaluation, including assessment of EF and reproductive health. This approach provided reliable baseline data before the onset of end-stage renal disease.

EF was evaluated using the International Index of Erectile Function (IIEF-5) questionnaire, with the following classification: severe ED (≤7), moderate (8–11), mild to moderate (12–16), mild (17–21), and no ED (22–25). Penile hemodynamics were assessed by ultrasound (US) with Doppler imaging of the penile arteries to determine the peak systolic velocity (PSV) in the cavernous and dorsal arteries. The hormonal profile – including serum testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) – was analyzed using the enzyme-linked immunosorbent assay (ELISA) method. Testicular volume was measured using ultrasound and verified using an orchidometer. Reproductive function was evaluated by semen analysis, assessing the frequency of normospermia, asthenospermia, oligospermia, oligoasthenoteratozoospermia (OAT syndrome), and azoospermia among the study participants. Varicocele was diagnosed by scrotal ultrasound with Doppler imaging and classified according to severity (grades 1–3).

In the management of persistent ED after KT, patients received first-line therapy with phosphodiesterase type 5 (PDE-5) inhibitors – either sildenafil (5 mg daily) or tadalafil (2.5–5 mg daily) for an initial 3-month course. Thereafter, treatment was continued with standard therapeutic doses (sildenafil 20–50 mg or tadalafil 10–20 mg) administered on demand approximately 30 minutes before sexual intercourse. The pharmacological therapy was supplemented with a pelvic floor muscle exercise program to enhance penile blood flow, vacuum therapy (10 daily sessions followed by 12 sessions every other day), and physiotherapy sessions (15 minutes daily for 10 days).

Descriptive and comparative statistical methods were used to analyze the collected data. Data accumulation, correction, systematization, and visualization were performed using Microsoft Office Excel 2016. Statistical analysis was conducted with IBM SPSS Statistics v.26 (IBM Corporation, USA). Descriptive statistics were used to characterize the clinical and demographic parameters of patients, including the calculation of mean values (M), standard deviations (m), and percentage distributions. To evaluate statistically significant differences across the various stages of observation, a one-way analysis of variance (ANOVA) was applied. The chi-square test (χ^2) was used to analyze categorical variables.

RESULTS

In the initial period (before KT), according to the IIEF-5 scale, most patients (52.5%) had no signs of ED, while 47.5% had mild ED (Table 1).

However, at the high azotemia stage, all patients lost normal EF: 87.0% presented with mild to moderate ED, and 12.3% had moderate ED. Three months after KT, EF began to recover – the proportion of patients with mild to moderate ED decreased to 68.8%, while 21.7% showed mild ED.

After 6 months, 72.5% of recipients still had mild to moderate ED, 23.6% had moderate impairment, and 4.0% demonstrated mild ED. By 12 months post-transplant, 65.6% of patients had fully restored normal EF, while the proportion with moderate to mild ED declined to 9.4%, and mild ED persisted in 25.0% of cases.

An analysis of the mean IIEF-5 scores in the cohort of 276 KT recipients confirmed that patients with a transplanted kidney show EF recovery (Fig. 1).

The mean IIEF-5 score before KT was 21.6 ± 0.1, but it declined to 13.2 ± 0.1 at the high azotemia stage, indicating a marked deterioration in erection. In the first 3 months after KT, a partial improvement was observed (15.0 ± 0.1), followed by a further increase to 18.4 ± 0.2 after 6 months. By 12 months post-transplant, the index had almost returned to its preoperative level, reaching 21.2 ± 0.2. According to ANOVA, these changes were statistically significant ($F(4.1375) = 702.33$; $p < 0.001$), confirming a consistent trend of EF recovery (Fig. 1).

In parallel with EF improvement, penile blood flow also showed positive changes, as reflected by the increase in PSV in both the cavernous and dorsal arteries (Table 2).

The initial PSV values were 6.5 ± 0.1 cm/s in the right cavernous artery, 6.3 ± 0.1 cm/s in the left cavernous artery, and 12.4 ± 0.2 cm/s in the dorsal artery. At the high azotemia stage, these values decreased to 5.6 ± 0.1, 5.4 ± 0.1, and 10.7 ± 0.2 cm/s, respectively, indicating a deterioration in penile arterial blood flow.

During the first year after KT, gradual recovery of penile hemodynamics was observed. After 3 months, PSV increased to 5.8 ± 0.1, 5.7 ± 0.1, and 11.2 ± 0.2 cm/s; after 6 months, to 6.3 ± 0.1, 6.1 ± 0.1, and 12.1 ± 0.2 cm/s; and by 12 months, reached 7.2 ± 0.1, 7.1 ± 0.1, and 13.9 ± 0.2 cm/s, surpassing baseline levels. These differences were statistically significant according to ANOVA ($F(4.1375) = 194.69$; $p < 0.001$ for the right cavernous artery; $F(4.1375) = 68.40$; $p < 0.001$ for the left cavernous artery; $F(4.1375) = 43.09$; $p < 0.001$ for the dorsal artery).

Testicular volume showed no significant changes (Fig. 2).

Initially, the right testicular volume was 16.9 ± 0.2 cm^3, and the left was 12.8 ± 0.2 cm^3. At the high azotemia

Table 1

Erectile dysfunction (ED) severity according to the IIEF-5 scale at different stages of the study

ED severity (based on IIEF-5 score)		Baseline	High azotemia stage	3 months post-KT	6 months post-KT	12 months post-KT
Severe ED	n	0	0	0	0	0
	%	0.0%	0.0%	0.0%	0.0%	0.0%
Moderate ED	n	0	34	26	65	0
	%	0.0%	12.3%	9.4%	23.6%	0.0%
Moderate–mild ED	n	0	240	190	200	26
	%	0.0%	87.0%	68.8%	72.5%	9.4%
Mild ED	n	131	2	60	11	69
	%	47.5%	0.7%	21.7%	4.0%	25.0%
No ED	n	145	0	0	0	181
	%	52.5%	0.0%	0.0%	0.0%	65.6%
Total	n	276	276	276	276	276
	%	100.0%	100.0%	100.0%	100.0%	100.0%

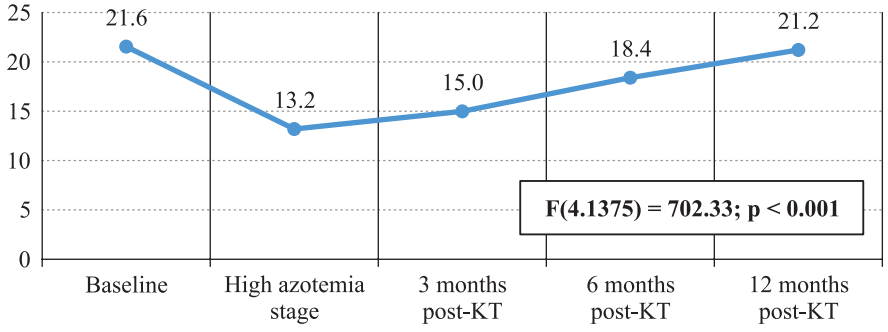

Fig. 1. Mean IIEF-5 scores in the kidney transplant group

stage, these values decreased slightly to 16.6 ± 0.2 cm^3 and 12.3 ± 0.2 cm^3, respectively. During the year following KT, only minor fluctuations were observed: after 3 months, the volumes remained nearly unchanged (16.5 ± 0.2 and 12.3 ± 0.2 cm^3); after 6 months, there was a modest increase to 16.8 ± 0.2 and 12.5 ± 0.2 cm^3; and by 12 months, the values slightly decreased again to 16.3 ± 0.2 and 12.1 ± 0.2 cm^3.

A statistically significant difference was noted only for the right testicle ($F(4.1375) = 3.66$; $p = 0.006$), whereas the left testicle showed no significant changes ($F(4.1375) = 2.09$; $p = 0.08$).

An analysis of hormonal status revealed a consistent trend in the dynamics of testosterone, LH, and FSH levels (Table 3). The testosterone level, initially 5.2 ± 0.2 ng/ml, decreased to 4.6 ± 0.1 ng/ml during high azotemia. Three months after KT, a slight increase was noted (4.7 ± 0.1 ng/ml), followed by further rises after 6 months (5.1 ± 0.1 ng/ml) and 12 months (5.6 ± 0.2 ng/ml) – ($F(4.1375) = 16.1$; $p < 0.001$).

LH level decreased from 8.9 ± 0.1 mIU/mL at baseline to 7.8 ± 0.1 mIU/mL with high azotemia, followed by a gradual increase to 8.0 ± 0.1 mIU/mL after 3 months, 8.6 ± 0.1 mIU/mL after 6 months, and 9.5 ± 0.1 mIU/mL after 1 year ($F(4.1375) = 81.2$; $p < 0.001$). A similar trend was observed for FSH, which decreased from 6.3 ± 0.1 to 5.5 ± 0.1 mIU/mL during high azotemia, followed by a recovery to 6.8 ± 0.1 mIU/mL after 12 months ($F(4.1375) = 22.5$; $p < 0.001$).

No cases of right-sided varicocele were identified at any stage of the study among all 276 patients. Analysis of left-sided varicocele (Fig. 3) showed that, initially, 22.1% of patients had grade 2 varicocele, with no cases of grade 3. However, at the stage of high azotemia,

Table 2

Mean peak systolic velocity (PSV) in the cavernous arteries during Doppler ultrasound examination

Study stage	Right cavernous artery (n = 276)	Left cavernous artery (n = 276)	Dorsal artery (n = 276)
	M ± m	M ± m	M ± m
Baseline	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
3 months post-KT	5.8 ± 0.1	5.7 ± 0.1	11.2 ± 0.2
6 months post-KT	6.3 ± 0.1	6.1 ± 0.1	12.1 ± 0.2
1 year post-KT	7.2 ± 0.1	7.1 ± 0.1	13.9 ± 0.2
ANOVA	$F(4.1375) = 194.7$; $p < 0.001$	$F(4.1375) = 68.4$; $p < 0.001$	$F(4.1375) = 43.1$; $p < 0.001$

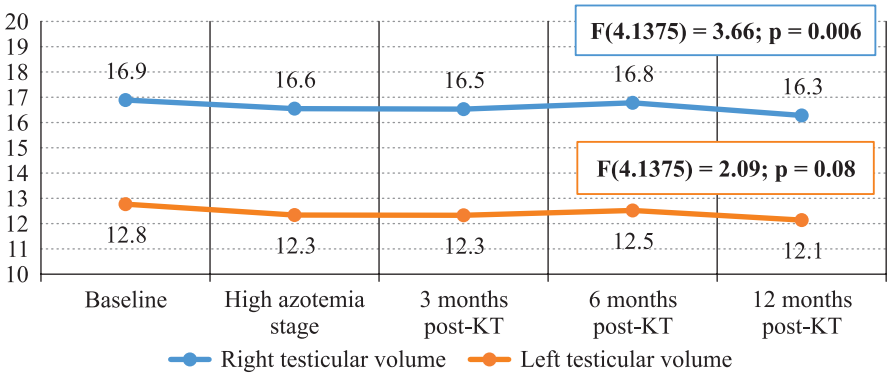

Fig. 2. Mean right and left testicular volumes in the kidney transplant group

Table 3

Mean serum levels of key hormones regulating reproductive function in the KT group

Stage	Testosterone (ng/mL, n = 276)	LH (mIU/mL, n = 276)	FSH (mIU/mL, n = 276)
	M ± m	M ± m	M ± m
Baseline	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
3 months post-KT	4.7 ± 0.1	8 ± 0.1	5.7 ± 0.1
6 months post-KT	5.1 ± 0.1	8.6 ± 0.1	6.1 ± 0.1
1 year post-KT	5.6 ± 0.2	9.5 ± 0.1	6.8 ± 0.1
ANOVA	$F(4.1375) = 16.1$; $p < 0.001$	$F(4.1375) = 81.2$; $p < 0.001$	$F(4.1375) = 22.5$; $p < 0.001$

22.1% of patients developed grade 3 varicocele, while grade 2 was not recorded.

After 3 months, the proportion of grade 3 left-sided varicocele decreased to 18.8%, and remained unchanged after 6 months ($\chi^2 = 76.5$; $p < 0.01$).

Significant changes were also observed in semen analysis (Fig. 4).

At the initial stage, normospermia was detected in 59.1% of patients, while asthenozoospermia – characterized by reduced sperm motility – was noted in 40.9%. Other spermatogenesis disorders (oligospermia, OAT syndrome, and azoospermia) were absent. At the high azotemia stage (stage 2), the proportion of normospermia decreased to 37.3%, while asthenozoospermia increased to 54.0%. Additionally, new cases of oligospermia (5.8%), OAT syndrome (1.8%), and azoospermia (1.1%) appeared, indicating suppression of spermatogenesis due to uremic intoxication and hormonal imbalance.

Three months after KT (stage 3), a slight improvement was noted: normospermia increased to 38.8%, asthenozoospermia decreased to 47.5%, though the incidence of OAT syndrome rose to 6.5%. After 6 months (stage 4), spermatogenesis showed marked recovery: normospermia increased to 52.9%, asthenozoospermia decreased to 35.1%, and the rates of oligozoospermia and OAT syndrome stabilized. By 12 months (stage 5), the recovery became most pronounced: normospermia reached 61.2%, exceeding baseline levels, while asthenozoospermia declined further to 27.5%. Although the proportion of oligospermia slightly increased to 7.2%, the rates of OAT syndrome (2.2%) and azoospermia (1.8%) continued to decline.

Thus, at the high azotemia stage (before KT), there was a marked deterioration in spermatogenesis, manifested by a decrease in normospermia and an increase in the frequency of pathological conditions, including asthenozoospermia, oligozoospermia, OAT syndrome, and azoospermia. Already 3 months post-transplant, improvement in the main indicators was evident, particularly an increase in the proportion of normospermia. After 6 and 12 months, there was a further restoration of fertility, with normospermia rising to 61.2% and asthenozoospermia decreasing to 27.5%.

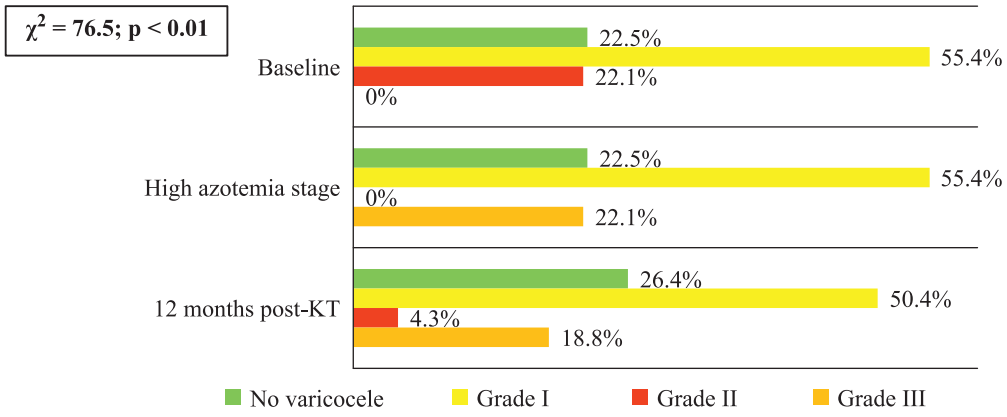

Fig. 3. Distribution of cases with or without left-sided varicocele in the kidney transplant group

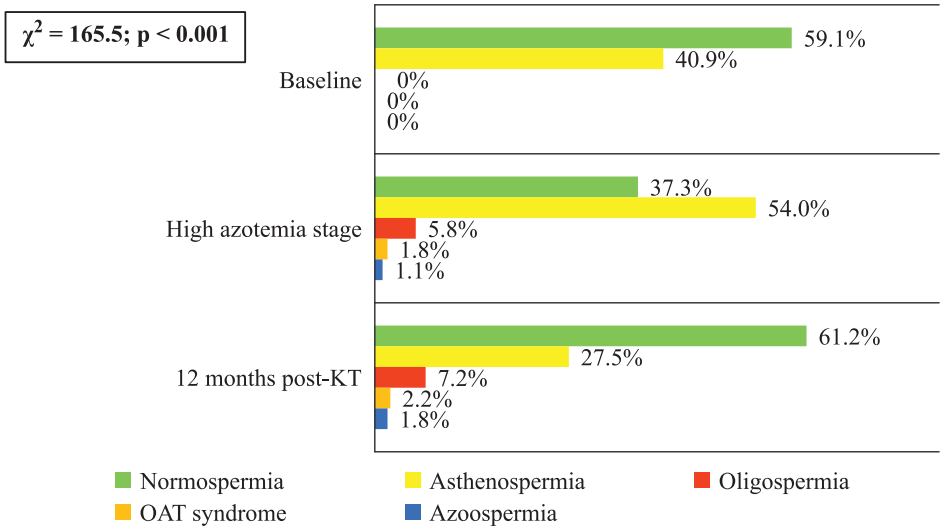

Fig. 4. Distribution of patients in the kidney transplant group according to changes in semen analysis parameters over time

The chi-square test for the distribution of spermogram types across KT stages yielded a value of $\chi^2 = 103.23$, with 16 degrees of freedom and $p < 0.001$, indicating statistically significant differences in the dynamics of spermogram parameters at various stages. These results confirm the positive effect of KT on the recovery of spermatogenesis and fertility potential in male patients.

Analysis of KT outcomes showed that 65.6% (181 of 276) of patients achieved complete restoration of EF within 1 year after KT, primarily due to normalization of vascular tone, improved cavernous arterial blood flow, and hormonal balance. However, 34.4% (95 of 276) of recipients continued to experience mild to moderate ED 1 year after KT, despite normalized renal function and improved systemic hemodynamics. These patients were categorized into five main groups according to factors that could explain why they still had ED (Table 4).

In the group of patients with vascular factors, the persistent ED was primarily attributed to long-term CKD, which led to irreversible vascular changes and progressive atherosclerosis. Clinically, these patients demonstrated a significant reduction in PSV in the cavernous arteries compared to those with restored EF. To correct ED, several therapeutic interventions were implemented. PDE-5 inhibitors (sildenafil, tadalafil) were prescribed, with a positive effect observed in 80% (24 of 30) of patients, while 20% showed no response due to severe vascular disorders. Additional management included statin and antiplatelet therapy to address systemic atherosclerosis, and the use of vasodilators (prostaglandin E1) in cases of severe arterial insufficiency, yielding a moderate effect in 36.7% (11 of 30) of patients. Physiotherapy programs, such as magnetotherapy and laser therapy, were also applied to improve regional blood flow, demonstrating 50% effectiveness (15 of 30). In two cases of advanced cavernous fibrosis, surgical treatment was performed in the form of penile implant surgery, while an additional 6 patients (20%) were indicated for this intervention.

In the group with persistent hormonal disorders, including hypogonadism and hyperprolactinemia (n = 25; 26.3%), several targeted therapeutic interventions were implemented. Testosterone replacement therapy was administered to patients with confirmed hypogonadism, resulting in clinical improvement in 72% (18 of 25) of cases. Dopamine agonists such as cabergoline and bromocriptine were prescribed for hyperprolactinemia, leading to positive dynamics in 80% (20 of 25) of patients. Additionally, metformin was used in individuals with insulin resistance, improving glucose and insulin sensitivity and indirectly enhancing EF in 40% (10 of 25) of cases.

Table 4

Summary of potential causes of persistent erectile dysfunction (ED) in kidney transplant recipients

Possible causes of persistent ED after KT	n	%
Vascular disorders (atherosclerosis, cavernous fibrosis, 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 leading to reduced penile blood flow	12	12.6%
Energy deficiency and sarcopenia (low body mass index, catabolic state)	10	10.5%

In the next group, comprising 18 of 95 patients (18.9%), the predominant factors were severe psychoemotional disturbances (depression, anxiety) and neurological disorders (polyneuropathy). Depression and anxiety contributed to psychogenic ED through reduced libido, whereas polyneuropathy disrupted nervous regulation of erections.

In this group, 61% of patients exhibited depressive disorders, 44% had sleep disorders, elevated cortisol levels, and chronic fatigue, while 28% reported reduced sensitivity in the penis and perineum. The therapeutic interventions included psychotherapeutic approaches such as cognitive-behavioral therapy and group therapy, which resulted in improvement in 50% of cases. The use of antidepressants (selective serotonin reuptake inhibitors in minimal doses) led to a positive effect in 65% of patients. Anxiolytics were administered when necessary to manage anxiety disorders, showing improvement in 45% of cases. Additionally, physiotherapy techniques aimed at stimulating peripheral nerve conduction (electrostimulation) improved nerve regulation in 35% of patients.

Patients with low BMI and sarcopenia (n = 10, 10.5%) had a general energy deficiency, which adversely affected endocrine function and erectile capacity. Mean BMI in this group was 18.9 kg/m^2, and serum albumin levels were below 3.5 g/dL, indicating a catabolic state. Testosterone level was approximately 25% lower than that of patients with restored EF. The therapeutic measures taken included: high-protein diet and nutritional support, resulting in a BMI increase of 1.5–2 kg/m^2 within 6 months; structured exercise programs (strength training), which produced an average muscle mass gain of 6% over 4 months; use of anabolic agents under endocrinological supervision, achieving improvement in 55% of cases; and correction of the catabolic state using amino acid complexes, leading to improvement in the general condition in 70% of patients.

Thus, 65.6% of patients recovered normal EF after KT, whereas 34.4% continued to experience moderate or mild ED associated with various etiological factors – vascular (31.6%), hormonal (26.3%), psychoemotional (18.9%), anatomical (12.6%), and energy deficiency (10.5%). Therapeutic correction included the use of PDE-5 inhibitors (80%), hormone therapy (72–80%),

psychotherapy (50–65%), physiotherapy (35–50%), nutritional support (70%), and surgical interventions (20%). Effective recovery required a comprehensive approach that addressed each patient's dominant risk factors.

It is also important to emphasize the influence of urological complications on EF recovery, observed in 8 recipients after KT. Among these, 4 patients (50%) had urinary tract obstruction, 2 (25%) were diagnosed with vesicoureteral reflux (VUR), and 2 (25%) had neurogenic urinary dysfunction. All patients in this subgroup presented with moderate to severe ED (IIEF-5: 12–18 points). Management of ureteral obstruction included endoscopic dilation (n = 3), neocystostomy (n = 1), PDE-5 inhibitor therapy (improvement in 3 out of 4 cases), physiotherapy (2 patients), and testosterone replacement therapy (1 patient).

Overall, for urological complications, a comprehensive treatment strategy, combining surgical correction, antibiotic therapy, physiotherapy, and vasoactive medications, proved effective in restoring EF. However, patients with severe vascular disorders may require penile implant surgery. These findings highlight that timely detection and correction of urological complications after KT increase the likelihood of EF recovery.

DISCUSSION

ED and reproductive disorders in patients with CKD and after KT remain pressing areas of clinical research due to their significant impact on patients' quality of life and overall health. In recent years, numerous studies have focused on developing diagnostic approaches to better identify and differentiate the underlying mechanisms of ED. Studies emphasize that hormonal and vascular abnormalities, including hypogonadism, hyperprolactinemia, and cavernous ischemia, play a leading role in the pathogenesis of persistent ED in this population [1, 3].

Despite the overall positive effect of KT, complete recovery of EF is not achieved in all recipients. According to Rahman et al., a systematic review demonstrated that KT leads to improved IIEF-5 scores, yet 20–50% of patients continue to experience varying degrees of ED post-transplant. The persistence of these disorders was primarily associated with long dialysis duration, advanced age, type of vascular anastomosis, and immunosuppressive therapy [7]. Similarly, Spirito et al. evaluated erectile and ejaculatory function at 6 and 12 months post-KT and observed a significant decline in sexual health quality at the 6-month follow-up, which remained stable throughout the year. In their cohort, the mean IIEF-5 score decreased significantly at 6 months ($p < 0.001$), remaining unchanged at 12 months ($p = 0.228$), correlating strongly with ejaculation disorders [14].

A study by El Hennawy et al. demonstrated that in patients with stage 5 CKD on dialysis, EF deteriorates; however, a positive trend is observed after KT. In their single-center crossover study, the authors assessed EF using the IIEF-5 one month before and one year after KT, revealing that KT recipients achieved significantly better results compared to dialysis patients [15].

In our study, the dynamics of IIEF-5 scores similarly showed a progressive recovery of EF after KT. Before transplantation, during the high azotemia stage, all patients exhibited varying degrees of ED. Twelve months post-KT, 65.6% of patients regained normal EF, confirmed by an 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 parameters associated with the development of ED in CKD patients have been actively explored. Wang et al. reported that decreased testosterone levels and hyperprolactinemia are key risk factors for ED in this population [16], while Zhang et al. demonstrated that dyslipidemia and impaired glucose metabolism significantly contribute to vascular disorders underlying erectile impairment [17].

Miron et al. presented data showing that 70% of patients continued to experience ED 12 months after KT, despite normal laboratory values. In these cases, medication-related and vascular factors had a greater impact than testosterone or creatinine levels [12]. Antonucci et al. found that testosterone and prolactin levels were directly correlated with the severity of ED: 65% of patients with hypogonadism and hyperprolactinemia continued to experience moderate ED despite normal graft function [18].

A meta-analysis by Kang et al., which included 9 studies, showed that after KT, testosterone levels increased by an average of 1.1 ng/mL, prolactin levels decreased by 6.2 ng/mL, and the incidence of ED declined by 32% compared to patients on dialysis. These findings confirm the hormonal dependence of EF recovery following KT [19].

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

Instrumental diagnostic methods, including pharmacodopplerography and shear wave elastography, are actively used to assess vascular alterations in patients with ED. Zhang et al. investigated the diagnostic potential of shear wave elastography in differentiating vasculogenic from non-vasculogenic ED. Their findings demonstrated that this method has high sensitivity and specificity for detecting fibrotic changes in the corpora cavernosa in patients with CKD and after KT [17].

Morphological studies of penile and testicular tissues also play an important role in understanding the pathogenesis of ED. Perri et al. analyzed cavernous body biopsies and identified pronounced fibrotic alterations that persisted even after successful KT, which may ex-

plain the persistence of ED in this patient group [20]. Similarly, Lundy et al. examined histological changes in the testes before and after KT and found that, despite the elimination of uremia, many patients continued to show signs of delayed spermatogenesis and morphological changes in Sertoli cells [21]. The same study confirmed that normalization of reproductive hormone levels after KT contributes to improvements in sperm parameters, including concentration, motility, and morphology of spermatozoa [21]. However, the use of immunosuppressive drugs – particularly calcineurin inhibitors and mTOR inhibitors – may adversely affect spermatogenesis and overall reproductive function.

In our study, we also examined vascular changes in ED by recording penile arterial blood flow dynamics using Doppler ultrasound. The results showed a gradual restoration of vascular perfusion following KT. By month 12, PSV exceeded baseline levels, reaching 7.2 ± 0.1 cm/s in the right cavernous artery, 7.1 ± 0.1 cm/s in the left cavernous artery, and 13.9 ± 0.2 cm/s in the dorsal artery, which correlated with an improvement in EF. All changes were statistically significant ($p < 0.001$).

Thus, both literature data and our findings confirm that KT promotes EF recovery in most patients with CKD, as evidenced by improvements in IIEF-5 scores, hormonal parameters, and penile arterial blood flow. After 12 months, normal EF was restored in 65.6% of patients, accompanied by increases in testosterone, LH, and FSH levels above baseline values. Enhanced vascular perfusion also correlated strongly with improved EF. However, a subset of patients continued to experience ED, primarily due to persistent vascular, hormonal, and medication-related factors. These results underscore the importance of a comprehensive diagnostic and therapeutic approach that includes hormonal and vascular monitoring, especially in the context of immunosuppressive therapy

However, our study has several limitations. The absence of a control group of dialysis patients limited the ability to perform a comparative analysis of the effects of different renal replacement therapies on erectile function. In addition, potential variations in the influence of specific immunosuppressive drugs on vascular and hormonal regulation were not considered.

CONCLUSION

The findings demonstrate that KT promotes effective restoration of EF and fertility in most patients, while targeted management of persistent vascular, hormonal, and psychoemotional disorders further enhances outcomes.

The authors declare no conflict of interest.

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