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ORIGINAL ARTICLE / ОРИГИНАЛНИ РАД

Urinary stasis in a transplanted kidney – 20 years of experience of one transplant center

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SUMMARY

Introduction/Objective Urinary stasis in a transplanted kidney occurs due to ureteral obstruction caused by intrinsic or extrinsic etiological factors. The aim of this study was to determine the prevalence, time of occurrence, and etiopathogenetic factors of urinary stasis and their distribution according to the type of kidney donor. And to analyze the success of different types of surgical and conservative treatment.

Methods The retrospective-prospective randomized study included 580 patients transplanted in the Transplant Center, Clinic of Urology, University Clinical Center of Serbia, for a period of 20 years. After diagnosing urinary stasis, minimally invasive or open surgical interventions were performed, while for one group of patients the definitive treatment was non-surgical with observation and active monitoring. The main control parameters during non-surgical treatment were the diameter of pyelon, serum creatinine values, and urine culture findings.

Results Urinary stasis was found in 15% of transplanted patients. The largest number of transplanted patients had early urinary stasis, within three months of transplantation (68%). The most common etiological factors of urinary stasis were intrinsic factors (66%), which were significantly more frequent in transplant patients from a living donor. Non-surgical treatment with observation and active monitoring was successfully performed in 22% of the patients.

Conclusion The largest number of transplanted patients with urinary stasis has been successfully treated surgically, most often with open surgery. Surgical correction is advised in cases of pronounced dilatation of the canalicular system with a tendency to increase, in progressive decrease in renal function, and recurrent complicated urinary infections refractory to antibiotic therapy.

Keywords: kidney transplantation; urinary stasis; surgical treatment; conservative treatment

INTRODUCTION

Urinary stasis in a transplanted kidney occurs due to obstruction of the ureter caused by intrinsic or extrinsic etiological factors. The cause of intrinsic etiological factors where the pathological process involves the wall of the ureter is most often ischemia, and less often edema and technical factors in reimplantation of the ureter into the bladder. Ischemia occurs as a result of extensive dissection around the ureter with a lesion of the blood vessels that vascularize the ureter and denudation of the adventitia of the ureter, which contains small blood vessels, during kidney explantation or kidney preparation for transplantation [1]. Therefore, the vascularization of the ureter should be preserved by a minimal peri-urethral dissection and especially by the preservation of the so-called "golden triangle," the space between the ureter, the lower half of the kidney, and the renal vascular pedicle. Stenting of the ureter protects against ureteral ischemia shortly after transplantation [2]. Extrinsic etiological factors can be extraureteral, which compress the ureter (hematoma, lymphocele), and intraureteral, which are in the lumen of the ureter (blood clot, calculus, tumor).

Early obstruction occurs within three months of transplantation and is most often caused by ischemia of the ureter, and less often by technical errors when performing ureterocystoneostomy, and compression from the outside [3]. Late obstruction, three months after transplantation or later, occurs due to ischemic fibrosis caused by deficient vascularization of the ureter, vasculitis in the context of episodes of acute rejection, vasoconstriction caused by immunosuppressive therapy, and chronic infection [4]. Viral and bacterial infections, acute rejection, and toxicity of immunosuppressive therapy can cause occasional transient obstruction due to ureterocystoneostomy edema and a loss of ureteral tone, due to denervation and impaired ureteral peristalsis. Ureteral tumors and urolithiasis are rare causes of late ureteral obstruction in transplanted patients [5].

Urinary stasis in a transplanted kidney most often requires surgical treatment in order to preserve renal function, prevent graft loss and death of the recipient. Any surgical intervention on a transplanted kidney is extremely precarious due to the possibility of further damage and loss of graft function and endangering the life of the patient [6, 7]. Therefore, it is justified to consider the possibility of a non-surgical expectant approach in the treatment of this complication [8].

The aim of this study was to determine the occurrence, time of development, and

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etiopathogenetic factors of urinary stasis in the transplanted kidney and their distribution according to the type of kidney donor; to assess the success of different types of surgical treatment: open and minimally invasive endoscopic and percutaneous interventions, and non-surgical approaches to the treatment of urinary stasis; and to analyze the most important parameters in the observation and active monitoring of transplanted patients with urinary stasis who are treated non-surgically, i.e., the parameters based on which the indication for surgical treatment will be set.

METHODS

The research was conducted as a retrospective-prospective randomized cohort study. The study included 580 patients transplanted over a period of 20 years, from 1999 to 2018, at the Transplantation Center, Clinic of Urology, University Clinical Center of Serbia. In all living donor kidney transplant patients, the donors were close relatives of the recipient. All cadaveric kidney donors were heart-beating (brain-dead) donors. In most cases, cadaveric uniorgan explants of only kidneys, were performed. Multi-organ cadaveric explants with kidney and liver explants were extremely rare. In the selection of a living donor, while determining the suitability of a cadaveric donor, as well as determining suitable recipient for a kidney transplant, all immunological and clinical criteria were met.

In all the patients, a standard operative technique was used during kidney explantation, from both living and cadaveric donors, and kidney transplantation. In all cases of transplantation from a living donor, termino-terminal arterial anastomosis of the renal artery and hypogastric artery was performed, while termino-lateral anastomosis of the renal artery and external iliac artery was performed in cadaveric transplantation. Urethrocystoneostomy was performed extravesically according to the Lich–Gregoir technique with mandatory placement of a "double J" stent within three weeks. In all cases of kidney transplantation, whether from a living or cadaveric donor, the left kidney was transplanted into the right iliac fossa, and the right kidney into the left. In most transplants, graft perfusion was performed using the Collins solution, and extremely rarely, only in multiorgan explantation, using histidine-tryptophan-ketoglutarate. All the patients were on a standard protocol of triple conventional immunosuppressive therapy. Induction immunosuppression was performed in all the patients. In kidney rejection, with or without previous biopsy, standard immunosuppressive treatment was applied.

The following diagnostic procedures were used in the diagnosis of urinary stasis: ultrasonography, antegrade pyelography, multislice computed tomography (MSCT), cystography, intravenous urography, nuclear magnetic resonance (NMR), and dynamic scintigraphy. As part of the diagnosis of urinary stasis in the transplanted kidney, the existence of an associated urinary fistula was ruled out in all the subjects.

In all the patients undergoing surgical treatment, percutaneous nephrostomy was initially performed. The initial surgical therapeutic procedures were minimally invasive endoscopic or percutaneous, and some of the open surgical interventions were performed in case of their failure. The group of patients whose definitive treatment was non-surgical was under observation and active monitoring with frequent ultrasonography controls, as well as repeated laboratory and microbiological analyses. The main observed control parameters in accordance with the data from the literature were as follows: the diameter of the pyelon of the transplanted kidney (measured ultrasonographically), serum creatinine values and urine culture (positive result was a bacterial count > 10^5) [8]. The values are presented as initial (value at the beginning of the observation), maximum (highest value during the observation) and final (value at the end of the observation).

Written consent was obtained from all the patients, the study has been approved by the relevant ethics committee, and conforms to the legal standards.

RESULTS

Out of 580 transplanted patients, a slightly higher number were from a living donor (306; 53%) (Figure 1). The largest number of transplantations from a living donor was performed in the first half of the study, from 1999 to 2008 (259), and from a cadaveric donor in the second half, from 2009 to 2018 (216). The difference in the number of transplants performed in the first and second half of the study in relation to the type of kidney donor is statistically significant ($p < 0.05$).

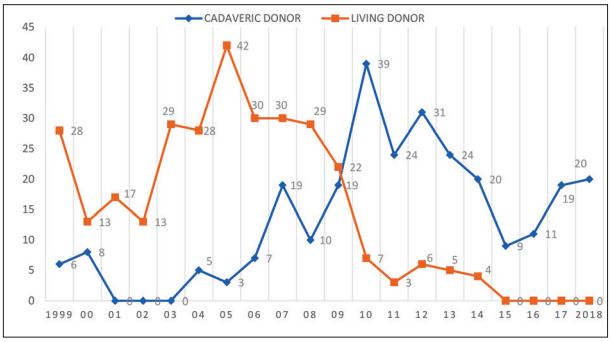

Figure 1. Annual distribution of kidney transplants according to donor type (n = 580)

The average age of the transplanted patients was 39.6 ± 10.6 years (range 18–62 years). The largest number of transplanted patients, 412 of them (71%) were between the ages of 30 and 50 years, 110 (19%) were under 30 years old, and 58 (10%) were older than 50 years. Among the transplanted patients, there were slightly more female patients (348; 60%).

In 87 (15%) transplanted patients, urinary stasis was found on the transplanted kidney. Approximately the same incidence of urinary stasis was registered in transplants from living (51; 17%) and cadaveric donors (36; 13%). The

difference in the number of patients with urinary stasis among transplanted patients from living and cadaveric donors is not statistically significant ($p > 0.05$). There was also no statistically significantly higher frequency of urinary stasis in older recipients. The largest number of transplanted patients had early urinary stasis, within three months of transplantation (59; 68%). No statistically significant difference was found in the prevalence of early and late urinary stasis in transplanted patients from different types of kidney donors ($p > 0.05$).

Of the etiological factors of urinary stasis, intrinsic factors were more frequent (57; 66%). Urinary stasis in patients transplanted from living donors was more often caused by intrinsic factors (42; 48%), and in cadaveric donors by extrinsic factors (21; 25%) (Figure 2). The difference in the prevalence of intrinsic and extrinsic etiological factors of urinary stasis, in transplantation from living and cadaveric donors is statistically significant ($p > 0.05$). Of the extrinsic etiological factors that led to urinary stasis in the transplanted kidney, the extraureteral factors were the following: lymphocele (14; 47%), hematoma (10; 33%), and compression of the funiculus on the ureter (5; 17%); the only intraureteral factor was calculosis (1; 3%).

The largest number of patients with urinary stasis required surgical treatment (68; 78%). In all cases of surgical treatment, percutaneous nephrostomy was initially performed. The majority of transplanted patients with urinary retention required treatment with open surgery (43; 63%). The following types of open surgical treatment were performed: replacement of the ureteral graft with a native ureter and ureteropyeloanastomosis (22; 32%), revision of ureterocystoneostomy (8; 12%), deliberation of the ureter with probe placement (4; 6%), hematoma drainage (4; 6%), marsupialization of lymphocele (3; 4%), and nephrectomy of the transplanted kidney (2; 3%). Minimally invasive endoscopic and percutaneous procedures were performed in 25 (37%) patients: percutaneous drainage with sclerotherapy (11; 16%), retrograde endoscopic placement of the probe (7; 10%), antegrade percutaneous placement of the probe (4; 6%), percutaneous hematoma drainage (2; 3%), and extracorporeal lithotripsy (1; 2%) were performed (Figure 3). Due to an unsuccessful previous intervention, 42 (62%) patients underwent two or more interventions until successful surgical treatment was achieved. The treatment was unsuccessful and ended with nephrectomy of the transplanted kidney in only two (3%) patients.

Non-surgical treatment involving observation with active monitoring was successfully performed in 19 (22%) transplanted patients with urinary retention. The average duration of observation was 8.3 ± 4.1 months (range 3–18 months). Non-surgical treatment led to the complete

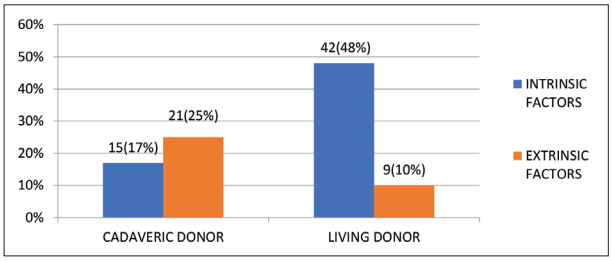

Figure 2. Etiological factors of urinary stasis in a transplanted kidney according to the type of kidney donor

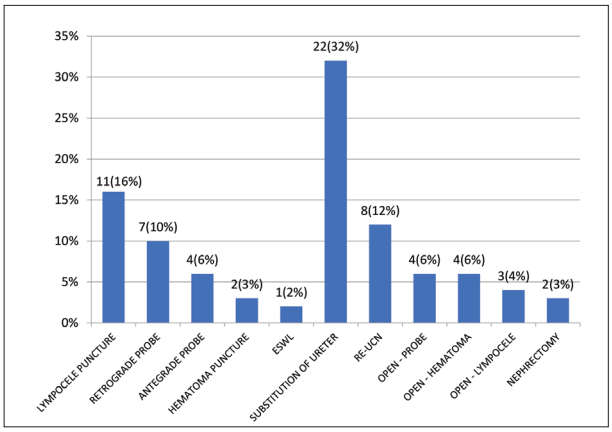

Figure 3. Surgical treatment of urinary stasis (n = 68) with a representation of minimally invasive endoscopic and percutaneous (n = 25) and open surgical procedures (n = 43)

resolution of urinary stasis in five patients (26%). The parameters observed during the conservative treatment of urinary stasis in the transplanted kidney are presented in Table 1. Statistically significant difference was found in the initial and maximum values of serum creatinine in relation to the end creatinine values ($p < 0.05$). No statistically significant difference was found between the initial, maximum, and final values of the pyelon diameter ($p < 0.05$). The frequency of positive urine culture initially and during observation is statistically insignificant ($p > 0.05$), while all patients had a negative urine culture at the end of observation.

In the majority of patients, one or more associated pathological conditions were treated during conservative treatment: cytomegalovirus infection (4; 22%), BK virus infection (8; 44%), acute graft rejection (7; 39%), toxicity of immunosuppressive therapy (10; 55%), and urinary infection (11; 66%).

Finally, successful treatment of urinary stasis in the transplanted kidney was achieved in 85 (98%) patients. In two (2%) patients, the graft was lost, explanted, and the patient was returned to dialysis. There were no fatalities.

Table 1. Parameters of conservative treatment of urinary stasis in a transplanted kidney (n = 19)

Parameters	Initial value	Maximal value	End value	p
Pyelon (mm)	22 ± 10 (10–30)	28 ± 9 (15–35)	20 ± 12 (0–30)	$p > 0.05$
Serum creatinine (μmol/L)	197 ± 89 (120–287)	236 ± 75 (130–325)	118 ± 18 (70–138)	$p < 0.05$
Positive urine culture (%)	50	61	0	$p > 0.05$

The values are presented as initial (at the beginning of the observation), maximum (highest during the observation), and final (at the end of the observation period).

DISCUSSION

Etiopathogenetic factors that lead to urological complications in a transplanted kidney are of donor or recipient origin and of medical or technical nature. Damage to the transplanted kidney is the result of ischemia, inflammation, infection, toxicity of immunosuppressive drugs, clinical condition of the donor and recipient, or technical errors during kidney explantation and transplantation [9]. The incidence of urinary stasis in a transplanted kidney is highly variable according to different studies, and ranges 3–20% [4, 10, 11, 12]. In older studies, it is registered more often than in today's researches. This may be related to advances in the technical factors of explantation and transplantation and new, improved perfusion solutions and the use of continuous mechanical hypothermic perfusion. In addition, new, more effective and less toxic immunosuppressive drugs and revised immunosuppressive protocols with lower doses of immunosuppressive drugs lead to a reduction in complications. In our study, urinary stasis in the transplanted kidney was detected in 15% of transplanted patients and occurred twice as often in the first 10 years of the study.

The most common etiological factors of urinary stasis are intrinsic factors due to ischemia, technical errors, or edema localized in the distal part of the ureter, most often on ureterocystoneostomy. It has been stated that ischemia is the most common cause of obstruction [7]. In our study, intrinsic factors as a cause of urinary stasis were registered in 66% of patients and were statistically significantly more common in patients transplanted from a living donor. Extrinsic etiological factors were significantly more frequent in cadaveric donors, and lymphocele was the most common among them.

Increasingly high-quality dialysis and improved drugs for chronic renal failure lead to an older age of the recipients. The data on the association of the age of the recipient with the onset of complications are controversial. In our study, 71% of the transplanted patients were between the ages of 30 and 50 years, and only 10% were older than 50 years. Irdam et al. [12] showed that older donor and recipient age are risk factors in developing urinary stasis after kidney transplantation. In our study no statistically significantly higher frequency of urinary stasis was found in older recipients.

The technical factor is important in causing urinary stasis in the transplanted kidney, because interruption of the ureter vascularization leads to ischemia and finally stricture of the ureter. Preservation of normal ureter vascularization by minimal periureteral dissection reduces the possibility of ureteral complications. Even with all the measures to preserve ureteral vascularization employed, the distal ureter is prone to ischemia due to its location and distance from the renal blood vessels (Figure 4). Furthermore, technical factors such as the type of ureterocystoneostomy and prophylactic stent use may also influence the higher incidence of urinary stasis in the transplanted kidney [3]. Today, ureterocystoneostomy is most often performed according to the modified Lich–Gregoir extravesical technique. It is performed easily and quickly on the front-lateral wall of the bladder. It includes a short muscular tunnel across the end of the ureter to prevent vesicoureteral reflux and development of compressive ischemia and subsequent stricture [13]. There are controversies about the necessity of stenting the transplanted ureter. In most studies, the prophylactic use of stents significantly reduces the incidence of urethral strictures [14]. In our study, all transplanted patients underwent ureterocystoneostomy according to the Lich–Gregoir technique using the extravesical route, and the ureter was routinely stented by placing a double J probe within three weeks.

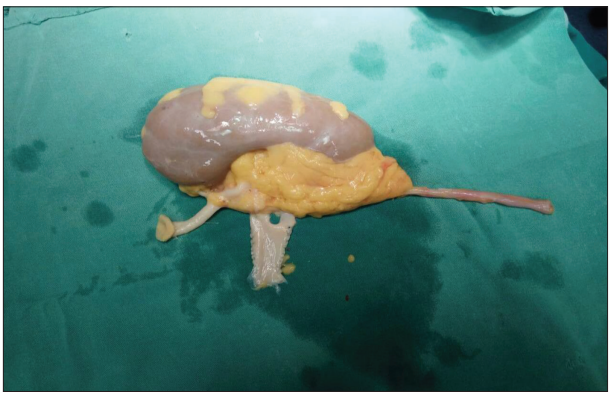

Figure 4. Right kidney explanted from cadaver and ready for transplantation; the renal artery has an aortic "Carrel patch," and the vein is lengthened by the reconstruction of part of the vena cava; fatty tissue is preserved in the hilus of the kidney and around the ureter ("golden triangle") to preserve ureteral circulation

Urinary stasis is most often described as a complication of kidney transplantation during the first year after transplantation [10]. In our study, we found a more frequent occurrence of early obstruction, during the first three months after transplantation (68%), which is consistent with the results of similar studies [4, 15].

The connection between episodes of acute rejection and the toxicity of immunosuppressive therapy in the occurrence of urinary stasis in a transplanted kidney is well known [16]. Vasoconstriction during kidney rejection and vasculitis caused by toxic immunosuppression therapy lead to ischemia and further complications in ureter. Immunological complications in the occurrence of urinary stasis are less common in transplants from living relatives and in transplants from cadaveric donors with standard immunological risk. In our study, urinary stasis occurred statistically insignificantly more often in transplantations from living kidney donors.

Today, the main goal of surgical treatment of urinary stasis in a transplanted kidney is the use of minimally invasive surgical techniques due to lower accompanying morbidity. Most patients with urinary stasis in a transplanted kidney can be treated with a percutaneous and endoscopic approach [17]. Short ureteral strictures on the distal ureter or on ureterocystoneostomy can be treated by antegrade percutaneous dilatation and placement of the probe into the ureter, or less often by a retrograde approach. In our study, surgical treatment was performed in 78% of patients, and among them, open surgery was

performed more often in 63% of patients, that is in accordance with similar studies [18]. In all cases of surgical treatment, percutaneous nephrostomy was initially performed as a temporary measure to establish urinary drainage, improve renal function, and treat infection [19]. Of the minimally invasive procedures, percutaneous drainage and lymphocele sclerotherapy were performed most often. Open surgical intervention is indicated after previously unsuccessful endoscopic or percutaneous treatment [20]. In our patients, ureter graft replacement with native ureter and ureteropyeloanastomosis of native ureter with pyelon of graft was most frequently performed open surgical treatment.

The non-surgical approach in treatment of urinary stasis in transplanted patients involves observation with active monitoring [8]. Successful non-surgical treatment in our study was performed in 22% of the patients. Repeated and periodic ultrasonographic examination, monitoring of renal function through serum creatinine levels, and monitoring of presence of urinary infection are crucial before making the decision to perform surgical intervention on the transplanted kidney [14]. Surgical correction is advised in cases of pronounced dilatation of the canalicular system with a tendency to increase, in cases of progressive decrease in renal function, and recurrent urinary infection refractory to antibiotic therapy [8]. Progressive elevation of serum creatinine is a strong indicator of graft dysfunction, while there is no standard threshold value of pyelon diameter in predicting the need for surgical intervention. It must be in conjunction with other parameters, especially with serum creatinine values, but also with the presence of a urinary infection refractory to antibiotic therapy [8, 21]. In our study, in successfully conservatively treated patients, a significant reduction in serum creatinine was achieved with improvement or normalization of graft function, followed by the absence of urinary infection with sterile urine culture. In one-quarter of patients, the dilatation of the canalicular system of the graft disappeared spontaneously, while in the others, functionally insignificant residual stasis remained without the need for surgical correction (Figure 5). Possible causes for residual stasis are a transient disturbance of peristalsis with blockage of the transmission of peristaltic waves through the wall of the ureter due to edema and ischemia, as well as transient vesicoureteral reflux [22].

Adequate treatment of associated pathological conditions on the transplanted kidney with urinary stasis can lead to an improvement in graft function and at the same time to a reduction or complete disappearance of stasis on the transplanted kidney. Therefore, a detailed exploration of all possible factors of deterioration of graft function is required. There is a causal relationship between cytomegalovirus, BK virus, and bacterial infection and stasis in a transplanted kidney [23, 24, 25]. Infections can cause edema, spasm, and ischemic damage to the ureter

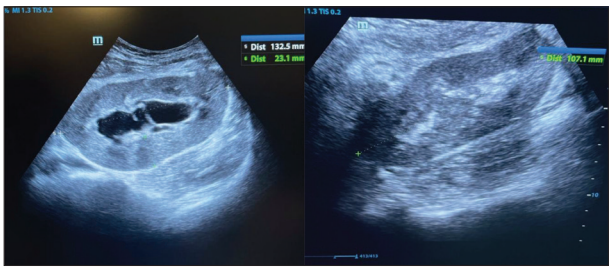

Figure 5. Complete spontaneous resolution of urinary stasis in the transplanted kidney during observation and active follow-up over a period of six weeks

leading to urinary stasis. The toxicity of immunosuppressive therapy is accompanied by ischemic damage to the ureter based on vasoconstriction and edema formation. Therefore, minimizing the negative effects of immunosuppressive therapy is very important. Renal rejection can be accompanied by obstruction due to local inflammation and vasculitis-induced ischemia of the ureter, which results in decreased tonus of the ureter due to denervation, and leads to edema and later fibrosis [26]. It is important to focus on the etiopathogenesis of urinary retention and to recognize and treat associated pathological conditions that may lead to graft dysfunction. Because of the unpredictable clinical course, individual evaluation of each transplant patient with urinary retention is crucial. In our study, the majority of patients under observation with active monitoring were treated for associated pathological conditions.

CONCLUSION

Urinary stasis in the transplanted kidney was found in 15% of transplanted patients without significant differences in representation according to the type of kidney donor. Most often, urinary stasis in the transplanted kidney occurred early, during the first three months after transplantation, and the most common etiological factors were intrinsic factors, significantly more common in transplantation from a living donor. The largest number of patients with urinary stasis were treated surgically with open surgery, most often by replacing the graft ureter with a native ureter with ureteropyeloanastomosis. Nonsurgical treatment was successful in 22% of transplant patients with urinary retention. Non-surgical treatment with active monitoring requires repeated ultrasound examinations with monitoring of renal function and the presence of urinary infection. Surgical correction is advised in cases of pronounced dilatation of the canalicular system of the transplanted kidney with a tendency to increase, in cases of progressive decrease in renal function and accompanying recurrent complicated urinary infection refractory to antibiotic therapy.

Conflict of interest: None declared.

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Уринарни застој на трансплантираном бубрегу – двадесетогодишње искуство једног трансплантационог центра

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САЖЕТАК

Увод/Циљ Уринарни застој трансплантираног бубрега настаје због опструкције уретера изазване интринзичним или екстринзичним етиолошким факторима.

Циљ рада је био утврдити заступљеност, време настанка и етиопатогенетске факторе уринарног застоја на трансплантираном бубрегу и њихову дистрибуцију према врсти донора бубрега, као и анализирати учесталост и успешност различитих врста хируршког и конзервативног лечења.

Методе У ретроспективно-проспективној рандомизованој студији обухваћено је 580 болесника са трансплантираним бубрегом у Центру за трансплантацију Клинике за урологију Клиничког центра Србије у периоду од 20 година. По дијагностиковању уринарног застоја на трансплантираном бубрегу, урађене су минимално инвазивне или отворене хируршке интервенције, док је за једну групу болесника дефинитивно лечење било нехируршко са опсервацијом и активним праћењем. Главни контролни параметри у току нехируршког лечења били су дијаметар пијелона трансплантираног бубрега, вредности серумског креатинина и налаз уринокултуре.

Резултати Код 15% болесника са трансплантираним бубрегом нађен је уринарни застој на трансплантираном бубрегу. Највећи број болесника имао је рани уринарни застој, у току прва три месеца од трансплантације (68%). Најчешћи етиолошки фактори уринарног застоја су били интринзични фактори (66%), који су се значајно учесталије јављали код болесника са трансплантираним бубрегом од живог донора. Нехируршко лечење са опсервацијом и активним праћењем је успешно спроведено код 22% болесника.

Закључак Највећи број болесника са уринарним застојем лечен је хируршки. Хируршка корекција се саветује у случајевима изражене дилатације каналикуларног система са тенденцијом повећања, код прогресивног снижења реналне функције и рецидивне компликоване уринарне инфекције отпорне на антибиотску терапију.

Кључне речи: трансплантација бубрега; уринарни застој; хируршко лечење; нехируршко лечење