Kidney Transplantation into Bladder Augmentation or Urinary Diversion: Long-Term Results

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Background. We report on a single-institutional experience with renal transplantation in patients with severe lower urinary tract dysfunction (LUTD) who underwent bladder augmentation or urinary diversion, and assess the long-term results.

Methods. From September 1987 to January 2005, 255 patients (161 male and 94 female), 7 months to 39 years old of age (median age at time of transplantation 14 years), received 271 kidney transplants. Etiology of end-stage renal disease was LUTD in 83 cases. Among these patients, 24 had undergone bladder augmentation or urinary diversion.

Results. We identified two groups of patients surgically treated due to LUTD: group 1 included 16 patients (eight male, eight female) aged 4 to 39 years (median 19 years) with bladder augmentation, whereas in group 2, seven patients (five male, two female) 7 months to 31 years old (median 17 years) with incontinent urinary diversion were reported. In the first group, surgical complications after kidney transplantation included one urinary fistula, one ureteral stenosis. Three patients of second group developed recurrent urinary tract infection. Cumulative graft survival rates of all patients transplanted was 69.4% after 15 years, whereas in the two investigated groups, group 1 and group 2, was 80.7% and 55.5% respectively (P=NS.).

Conclusions. Drainage of transplanted kidneys into an augmented bladder or urinary diversion is an appropriate management strategy when the native bladder is unsuitable. Kidney transplantation in patients with bladder augmentation or urinary diversion for LUTD let achieve similar results to those obtained in the general population with normal lower urinary tracts.

Keywords: Kidney transplantation, Urinary diversion, Bladder augmentation, Pediatric.

(*Transplantation* 2005;80: 1435–1440)

Approximately 15% of patients with end-stage renal disease (ESRD) have associated structural urological abnormalities that may lead to dysfunction of the lower urinary tract (*1*). This percentage increased to 20–30% in the pediatric population with ESRD (*2*).

Nowadays the better understanding of lower urinary tract evaluation and management using medical therapy, clean intermittent self-catheterization (CISC), and the development of more complex urinary reconstructive procedures had permitted greater improvement of natural history of patients who lost bladder function, and kidney transplant opportunity was offered to individuals without a functional bladder (*3*). There is some controversy about the safety of renal transplantation in patients with an augmentation cystoplasty due to the possibility of urinary tract infections development in immunosuppressed patients that can lead to pyelonephritis and graft loss. Thus, not more than 20 years ago patients with untreatable lower urinary tract disease had been excluded from renal transplant programs, because it seemed reasonable that the bladder that contributed to the destruction of the native kidneys would threaten a subsequently placed renal allograft (*4*). Nevertheless it is now well known that, in patients with a small volume, poorly compliant bladder, reconstructive bladder surgery in the form of bladder augmentation, or a urinary diversion creates a low pressure and compliant mechanism, which protects the upper urinary tract or the renal allograft (*5*). Actually there is no reason why such patients are not accepted for renal transplantation. Recently several series of kidney transplants drained to augmented bladders or urinary diversions have been published (*6–12*). However, there are few references in the literature specifically examining the long-term course of children with lower urinary tract dysfunction (LUTD); moreover no homogeneous large series of such kidney transplants in the pediatric population with long-term follow up (>10 years) have been reported. We analyzed our experience with renal transplantation in children with end-stage renal disease and bladder augmentation or urinary diversion and assessed long-term results.

MATERIALS AND METHODS

In our renal pediatric transplant center, we retrospectively reviewed a total of 271 kidney transplants performed on 255 patients (161 men and 94 women) 7 months to 31 years old (median age at time of transplantation 14 years) between September 1987 and January 2005. Causes of end-stage renal disease (ESRD) was congenital lower urinary tract dysfunction (LUTD) in 83 cases. The etiology of LUTD was posterior urethral valves in 18 cases, neurogenic bladder in 31, Prune-Belly syndrome in 5, malformation of lower urinary tract (including vesico-ureteral reflux) in 20, urethral hypoplasia in 6,

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Received 11 May 2005. Accepted 16 May 2005.

ISSN 0041-1337/05/8010-1435
DOI: 10.1097/01.tp.0000174342.19265.f4

urogenital sinus anomalies in 2, bladder exstrophy in 1. The medical records diagnostic imaging and laboratory evaluations of all children with ESRD were reviewed. Examination of clinical records showed as in patients with known or suspected LUTD an urodynamic evaluation was performed. Conservative or pharmacological approach was not successful for bladder preservation in 23 patients, 10 girls and 13 boys, with small bladder volumes and poor compliance. In these patients, the etiologies of bladder dysfunction were neurogenic bladder in 10 cases, posterior urethral valves in 5, urethral hypoplasia in 4, noncompliant bladder in 2, and malformation of lower urinary tract (including vesico-ureteral reflux), and bladder exstrophy in 1 each. Prophylactic antibiotic chemotherapy was administered in all cases to prevent urinary tract infection.

The immunosuppression regimen after transplantation consisted of cyclosporine or tacrolimus, azathioprine, and prednisolone. Cyclosporine levels were maintained at 150–250 ng/ml and tacrolimus at 5–10 ng/ml; this regimen does not differ from other patients in our schedule for the same period. Pretransplant and posttransplant clinical management and follow-up were uniform and were performed by the same medical and surgical team according to a protocolized schedule for examination and treatment. Postoperative urinary tract sonography was performed in all patients. Urodynamic assessment was performed in selected patients who were at risk for graft function from lower urinary tract dysfunction. We defined urine infection not by a positive urine culture, but by a symptomatic event (fever, dysuria, abdominal pain, etc.) associated with a positive urine culture. A patient was considered to have pyelonephritis if the infection was manifested by fever and/or kidney tenderness (*13*). The transplanted patients surgically treated for LUTD (bladder augmentation and or urinary diversion) were compared to those without severe voiding dysfunction undergone transplant during the same period. More specifically, patients were analyzed by two groups into those with bladder augmentation and those with urinary diversion.

Graft survival rate was determined from the time of transplantation to the last follow-up visit; therefore, we defined graft survival by a functioning kidney unit that did not require dialysis or explantation during the follow-up.

Statistical Analysis

Data are given as median values. Graft survival was assessed using Kaplan-Meier cumulative nonparametric survival plots for months. Groups were compared using the log-rank and wilcoxon test. A P value <0.05 was considered to show a statistical significant difference between two groups.

RESULTS

A total of 14 males and 10 females were identified. Median patient age at time of transplantation was 18 years (range, 7 months to 39 years). Follow-up ranged from 1 to 207 months (average 67.2). One patient was lost at follow-up.

We categorized the remaining 23 evaluable patients into two separate groups according to their surgical treatment received. The first (group 1) included 16 patients with bladder augmentation, whereas in the second group (group 2), seven patients with incontinent urinary diversion were reported. Patient characteristics are detailed in Table 1. The majority of transplants from Group 1 were performed within the last decade, whereas those in Group 2 generally were performed earlier.

The 16 patients in Group 1—16 patients (eight males, eight females) aged 4 to 39 years (median 18 years), median age at bladder reconstruction 15 years (range 4 to 37 years)—received 17 grafts. The cause of LUTD included seven patients with neurogenic bladder, three with posterior urethral valve, three with urethral hypoplasia, two with noncompliant bladder, and one with bladder exstrophy. Detubularized bowel segments were used to augment the bladder in 13/16 patients. Eleven patients were submitted to ileocystoplasty, three to ureterocystoplasty; in one case a gastric segment (gastrocystoplasty) was performed in another department before admission to our transplant center and in two patients we used a combination of useful segments, (including native ureter) according to the patient surgical history (Table 1). The interval from bladder reconstruction to transplantation ranged from 6 to 120 months (median 24). The bladder augmentation was created and functioning for drainage of the native kidneys before kidney transplantation in all except one case. Nevertheless a second bladder augmentation was performed after kidney transplant in one of these patients previously augmented by ureter; this was a child who was developing mild progressive deterioration of graft function. An urodynamic investigation documented a noncompliant, low-capacity reservoir (depending on poor result of ureterocystoplasty). After a second augmentation cystoplasty (with ileum), graft function did well.

Of the 16 patients with reconstructed bladder, the urethra served as primary urinary drainage in six (37.5%). In 10 patients, auxiliary urinary stomas (five Mitrofanoff, three Yang-Monti, two ureter Mitrofanoff-like) were created and all served as the primary urine drainage port. In 15 of the 16 cases, clean intermittent self-catheterization CISC was used. Only one patient voided. All patients except one were continent. The graft ureter was anastomosed to the native bladder in 10 patients, to the bowel segment used for bladder augmentation in three, and to the ureteral segment of an ureterocystoplasty in one. In 13 of the 16 cases (81.2%), the transplant kidney ureter was anastomosed to the reservoir with extravesical antirefluxing ureteroneocystostomy (Lich-Gregoire), whereas in two intravesical antirefluxing reimplant (Politano) was created and in one case a refluxing anastomosis was performed. The graft was from a cadaveric donor in 16 cases and a living related donor in one case.

For Group 2, seven patients (five males, two females) 7 months to 31 years old (median 17) with incontinent urinary diversion (two vesicostomy, three cutaneous ureterostomy, two sigmoid conduit), as outlined in Table 1, were reported. Median age at time of urinary diversion was 9.5 years (range 3 months to 23 years). The cause of LUTD included three patients with neurogenic bladder, two with posterior urethral valve, one with urethral hypoplasia, and one with vesicoureteral reflux. The interval from urinary diversion to transplantation ranged from 0 (considered time 0 in case of urinary diversion at the time of transplantation) to 120 months (median 85). In two cases, a "graft" cutaneous ureterostomy was assessed at the time of transplantation. One of these was due to the evidence of a small volume, poorly com-

TABLE 1. Patient demographics

Patient	Age	Sex	Etiology	Cystoplasty	Months from bladder augmentation to transplant	Creatinine (mg/dl)	Months of follow-up
				Group 1 (bladder augmentation group)			
1	13	M	Posterior urethral valve	Ileal	84	1	103
2	12	M	Posterior urethral valve	Colocecal+ureter	30	0.95	136
3	19	F	Neurogenic bladder	Ileal	15	0.97	120
4	17	M	Posterior urethral valve	Ileal	15	1.6	113
5	19	F	Neurogenic bladder	Ileal+Sigmoid	96	0.89	108
6	39	M	Noncompliant bladder	Ileal	84	1.6	100
7	23	F	Neurogenic bladder	Ileal	120	On dialysis	80
8	19	F	Noncompliant bladder	Ileal	10	1.5	54
9	11	M	Urethral hypoplasia	Ileal	After transplant	1.4	42
10	13	M	Bladder exstrophy	Gastric segment	84	1.5	40
11	25	F	Neurogenic bladder	Ureter	15	0.9	47
12	4	M	Urethral hypoplasia	Ureter before graft; Ileal after graft	6	2	26
13	5	M	Urethral hypoplasia	Ureter	7	0.9	24
14	23	F	Neurogenic bladder	Ileal	60	0.89	53
15	26	F	Neurogenic bladder	Ileal	62	0.86	2
16	16	F	Neurogenic bladder	Ileal	14	0.97	1
				Group 2 (urinary diversion group)			
1	14	F	Neurogenic bladder	Vesicostomy	108	On dialysis	193
2	17	M	Neurogenic bladder	Sigmoid conduit	96	1.3	184
3	20	F	Neurogenic bladder	Sigmoid conduit	84	1.6	148
4	31	M	Posterior urethral valve	Ileocystoplasty–"graft" cutaneous ureterostomy	84	1.3	115
5	1	M	Urethral hypoplasia	Vesicostomy	12	On dialysis	79
6	20	M	Vesicoureteral Reflux	Cutaneous ureterostomy	120	1.2	72
7	1	M	Posterior urethral valve	"Graft" cutaneous ureterostomy	During transplant	1.2	59

pliant bladder in a infant (7 months) with a ESRD secondary to posterior urethral valve; the second patient showed during transplant an unsuitable augmented bladder.

No details regarding the ureter to conduit anastomosis were reported in the two cases with sigmoid conduit. The transplant kidney ureter was anastomosed to the native bladder in the two vesicostomies using the Lich-Gregoire technique, to the bowel segment used for sigmoid conduit in two, and to the native ureter (ureteroureterostomy) in one.

Bladder Reconstruction-Related Complication

We have observed in a Mitrofanoff stoma stenosis in three patients that required a surgical treatment, whereas in the first case of enterocystoplasty a retraction of the reservoir, due to defunctionalization in an anuric patient, has taken place. This latter patient required a cutaneous "graft" ureterostomy management during transplantation (as detailed in Table 1). In one case, the ureterocystoplasty did not achieve results in terms of storage function and after transplant underwent to another augmentation cystoplasty (with ileum), We focused on the patient with a gastrocystoplasty performed in the other department, in which his quality of life was deeply threatened because of a severe chronic hematuria-Dysuria syndrome.

Complications after Transplant

Surgical complications included one urinary fistula and one distal ureteral obstruction. In the first patient fistula was caused by an ureteral stenosis. The ureter was stented on the seventh postoperative day by an ureteral endoprothesis (double-J); contemporary patient developed a thrombosis of the graft and 2 months later a ureter-iliac (artery) fistula, most probably caused by stent decubitus, lead to emergency removal of the graft. Seven years later, the patient received another graft (the unique living donor one) and its function is regular. In the second one, the patient—a girl with distal ureteral obstruction following transplantation—was managed endourologically with a double-J stent. Unfortunately, she lost her graft 10 months later due to chronic rejection and actually is on hemodialysis regimen. There was no renal artery stenosis or other surgical complication.

Two of the 16 patients with augmented bladder developed urinary tract infections or pyelonephritis after transplantation. On the other hand, in three patients of Group 2, uncomplicated recurrent urinary tract infections occurred despite prophylaxis; nevertheless, these events did not lead to loss of graft function. No graft was lost due to infection. All of these patients are maintained on chronic antimicrobial prophylaxis. Bladder (one case) or upper tract stones (two cases) occurred in three patients of Group 1, whereas we observed one bladder stone in a patient with vesicostomy (Group 2). We treated with extracorporeal shock waves lithotripsy the upper tract stones and percutaneously the bladder stones; all patients are now stone free. No metabolic acidosis developed.

Survival

Graft survival is illustrated in Figure 1. Table 2 summarizes and outlines the survival rates of the patient's population according to the group division.

Probability of graft survival at 1, 5, 10, and 15 years in the overall population (relatively to all the transplanted patients in the same period) was 87.4%, 77.1%, 73.8% and 69.4% respectively, with an average follow up of 127.7±4.3 months (95% CI 119.2–136.3).

Figure 2 shows probability of graft survival at 1, 5, 10, and 15 years in the LUTD population (Group 1 + Group 2), which was 96.1%, 82.8%, 66.1%, and 66.1% respectively without any significant differences (P=NS) if compared with graft survival of overall population during the same time.

Probability of graft survival in the Group 1 was 94.1%, 80.7%, 80.7%, and 80.7% at 1, 5, 10, and 15 years respectively with a mean of 56.5±4.7 months (95% CI 47.1–65.8). Probability of graft survival at 1, 5, 10, and 15 years in the Group 2 was 100%, 83.3%, 55.5%, and 55.5% respectively with a mean of 110±12.9 months (95% CI 84.6–135.3). The statistical analysis of the above described survival curves with standard tests (log-rank and Wilcoxon) did not show significant results (P=0.643, P=0.399). This may be due to the small sample size of the investigated sample. Function of the allografts, as measured by serum creatinine, is illustrated in Table 1. Of the kidneys, four were lost, including two (11.7%) in Group 1 and two (28.5%) in Group 2. Etiology of graft loss included chronic rejection in two cases, a viral chronic nephropathy in one case, and one graft thrombosis. No graft losses were related to urological disease; there were not any deaths.

DISCUSSION

The topic of bladder surgery related to kidney transplant is certainly not a new issue; nevertheless, it is still a source of dilemma and nowadays only the knowledge based on few, not standardized, institutional center experiences offer a reasonable know-how about management of lower urinary tract restoring function and kidney transplant. Previously, patients with severe lower urinary tract malformations had been excluded from renal transplant programs, as it appeared reasonable that a bladder that contributed to the destruction of the native kidneys would threaten a renal allograft (*14*, *15*). Enterocystoplasty or complete bladder replacement combined with CISC and suppressive antibiotic therapy as been shown to be an effective means to manage patients with refractory lower urinary tract dysfunction (*5*). Our approach is highly related with the statement that it is absolutely mandatory avoiding placing a graft into a dysfunctional bladder, particularly where the thick-walled, poorly compliant bladder was a factor in causing the subsequent renal damage. Where there is a history of such condition, bladder augmentation or urinary diversion is likely to be a safer option before transplantation.

Recently Hatch et al. from the Urologic Society for Transplantation and Vascular Surgery retrospectively reviewed the experience of 16 transplant centers showing an allograft survival similar with 90% at 1 year, 78% at 5 years, and 60% at 10 years in a group of children with urinary diversion or bladder augmentation; actually, this study did not difference the graft survival of patients with augmented bladder from that with incontinent urinary conduits (*13*). These results are similar to that reported by the North American Pediatric Renal Transplant Cooperative Study with 1- and 5-year actuarial allograft survivals of 91% and 77% for living donor kidneys and 81% and 61% for cadaver donor kidneys in pediatric renal transplant recipients (*16*).

Other recent series have shown that kidney transplantation is generally successful in patients with urinary diversion or bladder augmentation but there is a higher rate of complications among these transplant recipients (*11*, *12*). Furthermore, some series include only patients with bladder augmentation, whereas others, like the Hatch report, present graft survival rates in patients with augmented and nonaugmented bladders as well as urinary conduits. We intentionally analyzed patient by two groups into those with bladder augmentation and those with urinary diversion.

Our study confirms that bladder reconstruction before transplantation enables even the most severely compromised patient to receive a renal allograft successfully. Of our 16 bladder augmented patients, 14 (87, 5%) have a functioning allograft at 15 years follow-up with a probability of graft survival of 94.1%, 80.7%, 80.7%, and 80.7% at 1, 5, 10, and 15 years respectively (with a mean of 56.5±4.7 months). Transplant outcome in our patients with augmented bladder, as shown in Figure 1, was comparable to that in our cumulative ESRD pediatric renal transplant population.

Although supravesical diversion and urinary conduit has been historically associated with inferior graft survival and high complication rates (*3*, *11*), long-term renal function and acceptable complication rates were achieved in the patients with incontinent urinary diversions. In fact, five (71.4%) of our seven patients with urinary incontinent diversion (Group 2) showed probability of graft survival of 83.3%, 55.5%, and 55% at 5, 10, and 15 years respectively with a mean of 110±12.9 months. Data from 55 renal allografts into enteric conduits show that 73% were functioning at a mean (range) of 7.8 (0.2–20) years later (*14*, *25*). Several other sin-

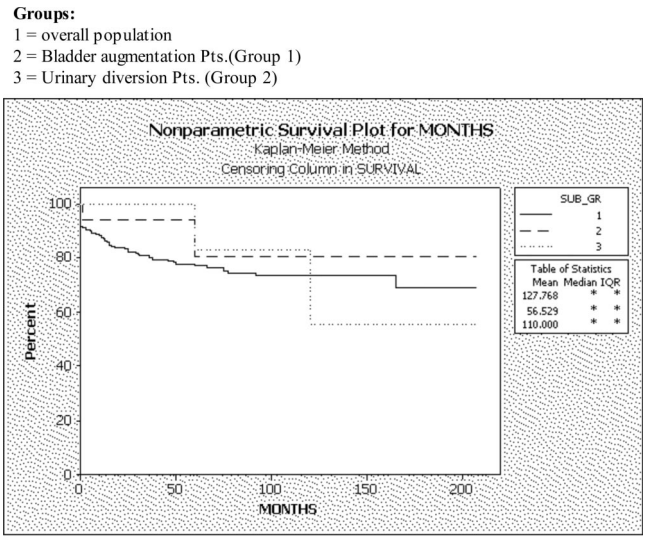

FIGURE 1. Graft survival rates. 1, overall population; 2, bladder augmentation (Group 1); 3, urinary diversion (Group 2).

TABLE 2. Graft survival rates

	n	1-year graft survival (%)	5-year graft survival (%)	10-year graft survival (%)	15-year graft survival (%)	*P* value
Overall population	255	87.4	77.1	73.8	69.4	
Groups 1 and 2	23	96.1	82.8	66.1	66.1	NS
Group 1	16	94.1	80.7	80.7	80.7	NS
Group 2	7	100	83.3	55.5	55.5	NS

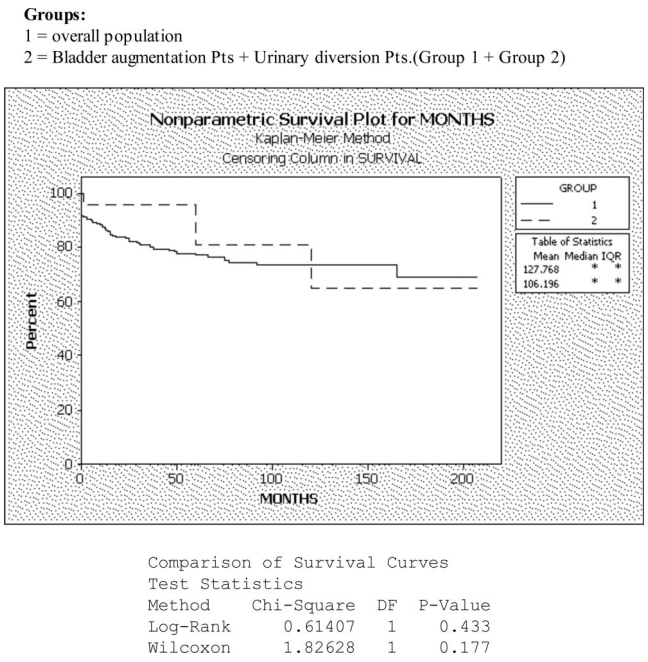

FIGURE 2. Graft survival rates. 1, overall population; 2, bladder augmentation + urinary diversion (Group 1 + Group 2).

gle-center and multicenter studies have reported similar graft survival values (*14*, *24*, *25*).

The timing of the bladder reconstructive surgery relative to transplantation is contentious; some groups advise cystoplasty before transplantation, so that immunosuppression does not influence the healing of the augmented bladder (*9*, *17*). Others consider that bladder augmentation and/or urinary diversion should be delayed until renal function is stable after transplantation, and when the immunosuppressive regimen has been reduced (*14*, *17*).

Thomalla et al. (*7*) and Fontaine et al. (*9*) suggested undertaking the reconstructive surgery as soon as abnormal bladder function is recognized before transplantation, so that immunosuppressive regimen does not influence the healing process. However, when abnormal bladder function is not diagnosed, successful reconstructive bladder surgery may be performed subsequently with no significant morbidity (*7*, *9*, *17*, *18*). We used to perform bladder reconstruction before kidney transplantation, to avoid graft loss due to lower urinary tract dysfunction, when small noncompliant bladder is recognized. Nevertheless, our experience led us to perform two augmentation cystoplasty after transplantation. In both cases, urodynamic surveillance demonstrated that bladder functional characteristics had deteriorated, so bladder function restoration was required (one of these was previously augmented by ureter). Therefore, in two cases, a cutaneous ureterostomy was assessed at the time of transplant. In all patients, no loss of graft function or any complications were observed. However, no studies have formally randomized patients to bladder surgery (either bladder augmentation either urinary diversion) before transplant or at the time as transplantation, and most series do not precisely specify the interval between conduit bladder surgery and transplantation.

Another issue is also problems associated with a "dry" cystoplasty (*9*, *14*, *19*). Problems may arise if the augmented bladder is small and contracted because of its lack of use. McInerny et al. (*18*) reported experience with eight cystoplasty; two cases with a dry cystoplasty required long-term suprapubic catheterization and were very difficult to manage, as persistent mucus production caused recurrent pyocystis requiring lavage.

Various methods of "bladder recycling" in the interval after bladder augmentation and before transplantation, particularly in the anuric population, to prevent the decrease of capacity and compliance secondary to the defunctionalization of the reservoir have been described (*19*).

In our experience, only one case (the first performed in an anuric patient) developed a retraction of the augmented bladder; this gave us the starting point to establish a recycling regimen, with reservoir filling twice a day with 300 ml of saline solution (physiologic solution, but you can use distilled water), to maintain adequate bladder volume but also to remove enteric secretions that can accumulate, and become a source of obstruction or infection (*20*). After introduction and standardization of bladder recycling, we have observed no more this problem.

Analysis of the surgical complications in our series revealed comparable results with other published series (*4*, *7*, *9*, *11–13*, *15*). Hatch et al. (*13*) published a surgical complications rate of 19%. Luke et al. (*11*) reported 10 urologic complications in seven of 20 patients (35%), including one spontaneous bladder perforation developed after transplantation, which required four surgical procedures. DeFoor et al. (*21*) reported three patients submitted to major surgery due to complications of the reconstruction (15%) and two treated with gastrocystopasty had severe hematuria while anuric before transplantation. Like Defoor, we separated our complications into those involving the bladder reconstructive surgery and those occurring after transplantation. We further divided the complications into those requiring major surgery, such as the only one occurred in the patient with reservoir loss due to the lack of use, and those requiring minor procedures, such as stomal revision observed in three patients (18.7%).

Additionally, we have had no perforation of augmented bladder. This potentially fatal complication has been reported in up to 9% of patients undergoing augmentation cystoplasty (*22*).

Bladder and/or upper tract stones occur in 8–52% of patients with bladder augmentation (*23*). In our experience, one bladder and two upper tract stones occurred in three patients of Group 1, whereas we observed one bladder stone in a patient with vesicostomy (Group 2). No patients in our series experienced metabolic complication.

A significant number of conduits are colonized with bacteria. A more recent series reported graft pyelonephritis in half the cases (*24*). Infection led to allograft loss in two of five patients being treated for acute rejection in the series by Hatch (*25*); 10 of 15 episodes of sepsis occurring after treatment of acute rejection in this group were fatal. Two pyelonephritis have developed in our augmented series, whereas in Group 2 three patients developed uncomplicated recurrent urinary tract infections, but we did not observe a direct contribution to graft loss, although there is no doubt that recurrent symptomatic infections of the urinary tract may trigger rejection episodes and contribute to the development of chronic allograft dysfunction (*15*).

This again underscores the need for carefully monitoring urine for infection, antibiotic prophylaxis, and aggressive treatment of UTI in this renal transplant population. The application of prophylactic antibiotics on a long-term basis helped to reduce the infection rates in our patients. Our standard practice now is to give long-term antibiotic in all patients with bladder dysfunction following transplantation for at least 6 months.

Although our urologic complication rate was not poor, no grafts were lost as a result of lower tract dysfunction. This highlights the importance of correct lower urinary tract evaluation, surveillance, and management in this complex group of patients, both before and after transplantation.

Bladder reconstruction is a major operation requiring a high level of surgical expertise with bowel and reconstructive procedures. Based on our long term results, we recommend reconstructive bladder surgery, although this procedure could increase morbidity. Diligent postoperative monitoring, optimal catheter use, and use of appropriate antibiotic prophylaxis and immunosuppressive regimens are mandatory to avoid severe complications.

CONCLUSIONS

Augmentation cystoplasty and urinary diversion represent a definitive method to restore the lower urinary tract function in children with small, noncompliant bladders. Graft survival is not adversely affected when a kidney transplant is drained into a reconstructed bladder. Moreover, our long-term data confirm that kidney transplantation into patients with augmentation and/or urinary diversion can achieve outcomes comparable to transplantation into the normal urinary tract. When bowel segments are used, voiding modality with CISC does not increase the risk of urinary tract infections, even in immunosuppressed patints. We recommend antibiotic prophylaxis in all of these patients. However, because complications related to augmentation cystoplasty or presence of incontinent urinary conduit might occur, these children must be followed closely and regularly.

ACKNOWLEDGMENTS

We thank for Prof. Daniele De Martini, Eastern Piedmont State University, for statistical analysis assistance.

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