Transpl Int (1999) 12: 334–340

ORIGINAL ARTICLE

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Ureteral stenosis after kidney transplantation

A study on 869 consecutive transplants

Received: 15 October 1998
Received after revision: 30 March 1999
Accepted: 13 July 1999

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Abstract Ureteral obstruction with impaired urine flow is the most common urological complication following renal transplantation. From December 1976 to December 1997, 869 kidney grafts were performed by our kidney transplantation group, 96 from living related donors and 773 from cadaver donors (736 first grafts and 37 regrafts). A stricture of the ureter (SU) was observed in 27 cases with a follow-up ranging from 18 months to 18 years after the graft and 11 months to 11 years after the treatment of the SU. In six patients, SU was immediately apparent and limited to the anastomosis: they were obviously technical flaws. In all the other patients, there was a free interval ranging from 2 months to 11 years after surgery; the SU usually involved the entire ureter, suggesting multiple etiologies. Repeated urinary infections could be a cause but immunological problems might be more determinant. In our series, acute rejection was more common than chronic so that the correction of SU was followed in many cases by a good and long lasting result (up to 11 years). In our experience, SU was not a dangerous complication even in patients in whom for different reasons (mainly refusal of treatment) the therapy was delayed – even if anuria occurred, no case of graft loss or serious damage were observed. At the beginning of our experience, the diagnosis of SU was based on urography, and therapy has always been re-operation. For 15 years, the diagnosis of SU has been based on routine echographic surveillance, which was intensified after each rejection, and the first treatment of SU in the last 8 years was re-operation in early technical SU and interventional radiology (balloon dilatation with or without temporary stent) in other cases. When it failed or in case of recurrence, surgical correction was performed utilizing the native ipsilateral or contralateral ureter for a ureteroureterostomy.

Key words Kidney transplantation · Ureteral stenosis

Introduction

Ureteral obstruction with impaired urine flow is the most common urological complication following renal transplantation. Some authors report a certain amount of graft loss and operative mortality. Although ureteral obstruction may be due to intraluminal obstacles (stones, blood clots) or compression from collections (blood, lymph), this paper will deal only with its most frequent cause – the stricture of the ureter (SU). SU may appear days or years after transplantation, with an incidence ranging from 2% to 7%. There is a tendency also to increase with time [10], suggesting that there is no single cause of the obstruction, as we have already stated in a previous paper [4].

Since the renal graft is denervated, the evolution of a stricture is usually asymptomatic until graft failure sets in. Aware of this fact and of its frequency, today all re-

cipients are submitted to ultrasonographic and clinical monitoring. Nevertheless, the finding of a dilated ureter at routine echography doesn't necessary mean that there is a stricture, since it is well known that dilatations without obstruction may be present [16]. Moreover, in the presence of a SU and renal impairment, therapeutic strategies can be problematic; in fact, kidney malfunction may be caused not only by SU but also by chronic rejection or cyclosporin toxicity. Finally, a SU deserves some attention since it can be frequently corrected with a long-lasting success rate and because the therapy of this condition has changed in the last few years – the recent advances in percutaneous invasive radiological maneuvers have significantly replaced surgical revision of the implant.

The aim of this paper was to review our experience on 27 cases of SU with a follow-up after transplantation ranging from 18 months to 18 years and a follow-up after treatment ranging from 11 months to 11 years.

Patients and methods

Between December 1976 and December 1997, our kidney transplant group performed 869 renal allografts – 96 from living and 773 from cadaver donors. The charts of donors and recipients were reviewed and all data collected along with the surgical procedures, the interventional radiology maneuvers and early and late patient outcome. All the information was assessed for this retrospective analysis. Efforts were made to understand the possible etiology of the SU in each case, focusing attention to extension and morphology of the SU, and the number and timing of acute rejections and urinary infections.

In the majority of our 869 transplant recipients, the reestablishment of the urinary tract was performed through a ureteroneocystostomy (UNC) according to Lich-Gregoir (L-G). Whenever the LG technique wasn't feasible because of fragility of the bladder mucosa we turned to the Politano (P) technique. No stents were ever used. An end-to-end uretero–ureteric (U–U) anastomosis was adopted in the patients who required the removal of the native kidney with ascertained absence of vesicoureteral reflux: a continuous suture with 7/0 absorbable synthetic monofilament was fashioned. After transplantation to exclude reflux, a retrograde cystography was performed. Urinary infections were monitored with urine cultures.

The time of onset of SU was established as exactly as possible and was utilized to subdivide the patients into four groups: patients in whom the SU appeared within 1 month (group 1), between 2 months and 12 months (group 2), between 1 year and 2 years (group 3) and over 2 years (group 4). Echography was the fulcrum of the follow-up confirmed by a urography when the dilatation persisted after micturition. A diuresis nephrography was carried out in a few cases. We never performed a Wittaker test.

In the cases of SU submitted to operative radiology, a percutaneous nephrostomy catheter was inserted under local anesthesia and echographic control. The nephrostomy catheter was aimed at the superior calix, since the insertion of the guide into the ureter was easier through this approach. The guide allowed the positioning of a straight tipped 5-Fr nephrostomy catheter beyond the stricture and its replacement was easy by a rigid superstiff catheter. A high-pressure balloon, selected according to ureter and stricture size, was then inserted and advanced to the stricture and inflated for 1–4 min. When the pressure dents on the balloon disappeared, the stricture had been dominated, and a 6–8 Fr double-pigtail stent was passed into the ureter. The nephrostomy catheter was left in situ only for the time necessary to do washouts with antibiotics and radiological controls. At the end, the stent was removed by cystoscopy.

In the cases submitted to surgery, a U–U anastomosis was carried out – a continuous suture with 7/0 absorbable synthetic monofilament was fashioned. An extraperitoneal approach or a median laparotomy was managed.

The ipsilateral or the contralateral ureter was employed. When using the native ureter for the anastomosis, the proximal portion was always tied off, performing a nephrectomy if necessary. In a few cases, a new UNC or a pyelo-ureterostomy were performed. The immunosuppressive therapy was never discontinued and prophylactic antibiotics were administered. A histological study of the ureter was possible only in a few cases.

Results

Among the cadaveric transplant recipients, 736 were first grafts while 37 were regrafts. No SU was ever observed in the 96 patients who had received a graft from living donors (91 L-G, 2 Politano and 3 U–U). Cadaver donor kidneys were used in 736 first grafts: none of the 95 U–U strictured, although, among the 641 UNC (628 L-G and 13 P), 25 strictures were reported. Regrafts were necessary in 37 cases and the ureter was implanted four times with U–U and 33 times with UNC (31 L-G and 2 P): two late strictures developed in the last group.

Altogether, 27 cases of SU appeared from a few days to 12 years after transplantation among 869 kidney grafts, with an overall incidence of 3.1 %. The incidence increased with time from 0.8 % at 1 month to 2 % at 1 year, 3.2 % at 2 years up to 9 % at 10 years. It was remarkable that the incidence was nil in the 95 cases of U–U and 4.0 % in all the UNC.

In SU cases, the mean peak PRA (panel reactive antibodies) was 22.3 ± 25.07 % (15 patients) (range 0–90). The immunosoppressive therapy was azathioprine and steroids in 16 patients, cyclosporin and steroids in 9 and triple therapy (cyclosporin, azathioprine and steroids) in 2. In eight patients, the therapy became triple. In group 1, the mean human leukocyte antigen (HLA) A mismatches was 0.8 ± 0.7, the mean HLA B mismatches 1.1 ± 0.5 and the mean HLA DR mismatches 1.2 ± 0.8. In group 2, the mean HLA A mismatches was 0.8 ± 0.8, the mean HLA B mismatches 1.1 ± 0.4, and the mean HLA DR mismatches 1.2 ± 0.5. In group 3, the mean HLA A mismatches was 1.6 ± 0.5, the mean HLA B mismatches 1.5 ± 0.5 and the HLA DR were not detected. In group 4, the mean HLA A mismatches was 1.7 ± 0.8, the mean HLA B mismatches 1.2 ± 0.4 and the HLA DR were not detected. The mean HLA first class in the SU were: group 1, 2.3 ± 0.8; group 2, 3.2 ± 0.5; group 3, 3.1 ± 0.7; and group 4, 3 ± 0.9. The

cold mean ischemia time was 11.7 ± 6.2; there were 17 local kidneys and 10 shipped ones. The type of preservation solution was Eurocollins in 20 kidneys and Belzer Solution in 7. The incidence of late acute rejection in the study group of SU was 44.4%.

Diagnosis

A diuresis nephrography carried out in three cases gave two false results: a false negative in one patient who developed an acute dilatation with a fall in function and a false positive in one case who maintained good renal function. In six patients (group 1), the SU was limited to the UNC junction and was already present at the first echography, a few days after transplantation. In all the other patients who developed a SU, one or more postoperative study had shown a normal urinary flow, followed by a free interval varying from 1 year to 11 months before a sudden pyelectasis appeared. In all these patients, the SU involved a long segment of the distal ureter. The SU developed between 2 months and 12 months in eight patients (group 2). In six of them, it occurred within a mean interval of 5 months after an episode of acute rejection. In one case, no special evolution has been detected, and, in one, a lymphatic collection remained for a long period around the ureter: its drainage did reveal an organic SU. In six cases, the stenosis appeared between the 12th and the 24th postoperative month (group 3). In three patients acute rejection occurred before SU onset with a mean interval of 4 months, and, in the other three, there was a chronic rejection. In seven patients the SU developed from 2–12 years after the graft (group 4). In three patients, an acute rejection had been reported shortly before the pyelectasis, and, in three, apparently with no immunological problems, repeated urinary infections were reported.

Operative radiology

Operative radiology was of paramount importance in the six patients who become suddenly anuric and in whom a pyelostomy was necessary to overcome the emergency: all patients had to be re-operated. In 11 of the 21 remaining cases, interventional radiology was considered first choice. One patient had a segmental stenosis of the ureter and a single sitting of dilatation sufficed in resolving the problems definitively. The positioning of a stent was attempted in ten cases and succeeded in eight. In four patients, good function returned and remained 5, 6, 7 and 10 years after destenting, even if, in two cases, a mild innocuous stricture persisted after 6 months and 4 years. Four patients did not follow the advice of destenting at the right time and they kept it in situ for 6, 18, and 19 months and 3 years, respectively. When the stents were finally removed, since the patients were symptom free, a tight stricture recurred in three cases and the other become anuric so that corrective surgery was mandatory in all.

Surgical correction

Surgical correction was performed in 20 patients, in 14 as first option, in 2 after failure of stenting the SU, and 4 were operated for recurrence of the SU after destenting. An end-to-end U–U anastomosis was adopted in 18 of the 19 revisions of the ureteral implant for SU where a UNC had been first performed (in the 19th patient, a new UNC and in the 20th patient a pyelo-ureterostomy were preferred). The ipsilateral ureter was always used except in a case of renal agenesis, in which the contralateral ureter was employed. When using the native ureter for the anastomosis, the proximal portion was always tied off, performing a nephrectomy only in one case of polycystic kidney. An extraperitoneal approach was preferred to fashion the first four U–U in implant revisions while the remaining cases were managed through a median laparotomy

Of the 20 operated patients, one had a urinary leak after a pyelo-ureterostomy with also a severe acute rejection, and the graft had to be removed. In the remaining 19 patients, the operation was successful (1 ureteral re-implantation and 18 U–U): 1 died of unrelated causes at 3 months, 3 returned to hemodyalisis within 1 year, but 15 had good renal function for a long period (from 1 year to 11 years).

A histological study was possible only in two cases in which the pathologist reported the presence of aspecific tissue with no cell population indicative of a precise pathogenesis. The characteristics of SU and results of management in our kidney transplant recipients are summarized in Table 1.

Discussion

Etiology

In six patients, group 1, the SU was limited to the UNC junction. It was easy to deduce that a technical error was the cause (faulty surgical technique in four cases and exuberant scar tissue growth in the others, where a small urinary fistula had been treated conservatively in the first patient and corrected with a single stitch in the second).

In six patients of group 2, the SU occurred within a short period of time after an episode of acute rejection: it could be the cause. In one case, no special evolution has been detected and in one a lymphatic collection re-

Table 1 Characteristics of ureteral stenosis (US) and results of management in kidney transplant recipients. *U–U* uretero-ureterostomy; *CR* chronic rejection; *AR* acute rejection; *HD* hemodyalisis

Time of onset N°	Possible etiology	Management	Outcome
Group 1 (1–30 d) 6 pts.	6 Technical	1 Reimplantation	HD at 4 mo for CR
		4 U-U	3 Well at 17 mo, 2 and 4 y 1 HD at 1 y
		1 Stent	Well at 8 y
Group 2 (2–12 mo) 8 pts.	1 Unknown	U-U	Well at 8 y
	1 Chronic lymphocele	1 Stent	Well at 3,5 y
	6 AR	4 U-U	2 Well at 1,3 and 8 y 1 HD at 6 mo for CR 1 Transplantectomy
		1 Percutaneous pyelostomy	Well at 2 mo (Died medullary aplasia)
		1 Percutaneous dilatation	Well at 8 mo (Died hepatic failure)
Group 3 (1–2 y) 6 pts.	3 AR	3 U-U	Well at 3 mo (Died unrelated) 1 Well at 18 mo, 1 HD at 8 y
	3 CR	1 Medical Therapy	HD at 1 y for CR
		2 U-U	2 HD at 10 and 12 mo.
Group 4 (> 2 y) 7 pts.	1 Unknown	1 Stent	Well at 4,5 y
	3 AR	1 Stent	Well at 5,5 y
		2 U-U	1 Well at 3,5 y, 1 HD at 11 y.
	3 Urinary Infections	3U-U	3 Well at 4,4 and 4,5 y.

mained for a long period around the ureter and may have caused the stricture – its drainage did reveal an organic SU.

In group 3, acute rejection seemed to trigger SU in three patients while chronic rejection was the likely reason in the other three, two of whom returned to dialysis within 1 year after a successful re-operation and the third was excluded from any treatment for the clinical diagnosis of chronic rejection.

In group 4, in one patient no reasonable cause of SU was detected while in three patients an acute rejection had been reported shortly before the pyelectasis and in three, apparently with no immunological problems, repeated urinary infections may have played an important role.

The hypothesis that most urological complications of kidney transplantation are due to faulty technique [8, 12] may be true only for early leaks but not for SU, at least when they appear late. When problems are seen early, errors in technique or an imperfect healing of the anastomosis may be imputed and these strictures usually occur at the site of the anastomosis. It is hard to believe that an incorrect harvest with damage to the blood supply to the ureter may have caused the strictures, since in these cases, a leak would more probably occur.

If one considers that only early and segmental strictures at the UNC junction are probably technical, there are series in which this kind of stenosis is frequent. In Mundy's series [14], in 49 of 52 cases, the SU is of the distal ureter only. The same was observed for 35 of the 40 cases reported by Keller [9]. In Nicholson's experience [15], 12 of the 15 strictures appeared within 2 months. In Rigg's study [19], 47% of the SU were observed within the first postoperative month, 73% within the second postoperative month and in 89 of the 104 patients it was at the vesicoureteral junction.

In the present series, only 6 of the 27 cases of SU appeared within 2 months from grafting and were at UNC level; the others involved the ureter extensively and showed up from 2 months to 12 years postoperatively. We are therefore led to believe that a faulty technique is limited to these six patients and that, in most cases, the causes of obstruction may have been different. Several hypotheses have been advanced: motility disorders, infections, exuberant scarring, kinking of the ureter but none of these appear to be likely in our patients.

Immunological phenomena often seem to play an important role, as observed by us and others [1, 2, 4, 6, 19]. Probably, acute rejection is often the cause as stated recently by Mayer [13] and this hypothesis is supported by the fact that, once the stricture is corrected, long-term normal graft function can be expected. In some rare cases, the ureteral stricture seems to be a consequence of a not fully fledged chronic rejection that in three of our cases, hadn't been ascertained until corrective surgery gave unsatisfactory functional results and the patients progressed to an early return to dialysis.

The actuarial rise in the incidence of SU with time observed by Kinnaert [10] and confirmed by the present report gives credit to the hypothesis that immunological phenomena are the main culprits. It is interesting to

note that in patients in whom the SU appears during the first 12 months and is not due to technical errors, an episode of acute rejection is, usually, promptly followed by the onset of the stenosis which evolves rapidly to anuria. In those cases where a stricture occurs after 12 months, the evolution is slower and the causes may be many; however, an episode of acute rejection appears to be the likely cause even in some of the more tardy. The relationship between SU and acute rejections is so common in our series to advise an increase in control ecographies after each episode.

Diagnosis and management

Since the evolution of SU is almost always symptom free, close echographic surveillance of graft morphology is ideal, considering that a simple dilatation of the renal pelvis doesn't necessarily mean that there is an obstruction. Straiton detected a dilatation of the urinary tract in 80 patients, but the impediment to urine flow was confirmed in only 34. He noticed that if the calices were also dilated and the dilatation persisted after micturition, a stricture was present in 50 % of the cases. Furthermore, in the patients in whom a dilated renal pelvis appeared after 30 days, there was no obstruction, while if it appeared earlier a stenosis was present in 6 of 18 patients [22]. If diagnosis is still dubious, a pressure-perfusion test according to Wittaker through a pyelostomy or a diuretic –nephroscintigraphy has been advised. This last technique was not very helpful in our series as in others' [5, 7] and we never did a Wittaker test.

In no case was the dilatation of the urinary tract considered an emergency by our team, even if transplant function decreased. Several of these patients remained with an untreated SU for a long period of time (from a few months to three years) for their refusal of treatment so that some of them become suddenly anuric. As a matter of fact, the assessment of the data gathered in those individuals who refused all repair surgery shows that in the presence of a SU, there is no particular risk for patient and graft function in contrast with Shoskes [20] option that a rapid diagnosis is mandatory or with Jaskowski's statement that obstructive uropathy must be dealt with immediately and aggressively lest function be lost [8]. If renal function is maintained, a tight stricture left untreated for a long period of time did not jeopardize either patient or graft survival even in the three cases who became anuric. This suggests that costly sophisticated diagnostics are not really that necessary. If there is a functional deficit of the kidney, a nephrostomy should be carried out and maintained for several days before the dilatation of the SU and the creatinine level are monitored to quantify the importance of the obstruction on renal function [24]. Our experience confirms that the presence and severity of chronic rejection is not always easy to evaluate, but, in our opinion, immediate stenting and withdrawal of the pielostomy is preferable for the patient and reduces the risk of infection. These observations allowed us to accurately assess the two modalities of treatment: surgery and operative radiology.

Interventional radiology

The antegrade catheterization of the pelvis and ureter under radiological guidance in our earlier experience was limited to verifying the diagnosis of compromised renal graft function and the management of acute anuria as a palliative solution in preparation for implant revision. Today, these methods are employed not only in reestablishing urine flow and in assessing the true importance of the strictured ureter on renal function but also in correcting the obstruction. We agree with Rosenthal [18] who states that " probably the operative intervention continues to be the mainstay for the majority of the patients " but interventional radiology offers excellent options to temporize surgery, select the right patients to operate and sometimes reduce the number of patients who need an intervention more or less by 50 % [17].

In the last 8 years, stricture dilatation and stent positioning were always attempted except in early "technical" SU where surgery is the best option in our opinion because a dilatation of the ureterovesical junction could leave a reflux. Stent positioning, when successful, resulted in good urine outflow for long periods of time. In patients with advanced chronic graft rejection, this can be a good definitive solution which prolongs graft function. The withdrawal of the stent was followed by a permanent satisfactory dilatation of the SU in five patients and by a recurrence in four; these were all patients who had refused any therapy for too long a period of time (the stents remained in situ for 3 months to 3 years, a period much longer than the 3 months usually considered safe, and probably contributed to the recurrence). Even in these cases, the stents proved to be harmless, for whenever an infection set in the stent was simply removed and in no case did the kidney suffer a permanent damage. We have never attempted to position stents through cystoscopy. In our opinion, a percutaneous pyelostomy under local anesthesia with dilatation of the stricture is less traumatic and causes fewer infections. Our policy was never to maintain long term pyelostomies; if the stent passes beyond the stenosis, we remove the pyelostomy leaving the stent in situ to be pulled out by cystoscopy at the right moment; if the stricture is too tight and renal function remains normal, we prefer to operate.

Surgery

In the past, revision of a strictured ureter of a transplant often caused graft loss and even a certain mortality [10, 14, 21]. Kinnaert recognizes the difficulty of redo surgery, especially when using the extraperitoneal approach where tissue damage forms blood and lymphatic collections that may easily be infected. We met the same difficulties but without problems in our first four cases, then we turned to the easier and safer transperitoneal approach.

The U–U or the uretero-pyelostomy with a vital native ureter is the best solution. We prefer the U–U because it is easier and it permits us to perform a pyeloureterostomy if it fails. The preoperative assessment of the native ureter is not essential since we saw many small ureters with tight entry to the bladder return to excellent function once urine flow started. It is sufficient to assess the absence of a reflux from the history and to verify the ureter at surgery. If the ipsilateral ureter is not available, it is possible to use the contralateral one as we did in a patient who had an excellent functional results for over 10 years. The proximal stump of the ureter in the U–U patients, always tied off and abandoned, did not give early or late complications.

Conclusions

Our experience confirms that late ureteral strictures after renal grafts may be due to multiple causes, but the most frequent in our series appears to be acute rejection which may occur at any time. If this is the case, once the implant has been revised, the graft will function for many years. The diagnosis of a ureteral stricture implies only close clinical and echographic monitoring: since this condition doesn't jeopardize graft function, aggressive diagnostics are unnecessary.

Today, in our opinion, surgery is preferable in early cases with distal stenosis of technical origin and interventional radiology must be the first choice in late SU because the modern armamentarium frequently allows the return to good graft function and urine flow by correcting the stricture without surgery.

Needless to say, when facing adamantine strictures, which don't give way, it is useless to insist on attacking them with very complicated non-surgical techniques combining percutaneous and cystoscopy maneuvers under general anesthesia as suggested by some authors [3, 11, 23]. It is better to perform a surgical revision which can give, as in many of our patients, good and long-lasting results at a low risk. Since the non-technical stenosis usually involves large segments of the distal ureter during the last 200 kidneys, we diminished the number of UNCs adopting the U–U whenever the patient had a normal ureter and less of 300 cc of residual diuresis, with the hope of diminishing the incidence of late US with a shorter transplant ureter. We used to perform a contemporary nephrectomy only in cases of septic kidneys.

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