

Case Report

Improvement of polyuria, bladder sensation, and bladder capacity by renal transplantation: A case report

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Abstract

Here, we report a patient with renal failure and bladder dysfunction whose symptoms improved following renal transplantation. A 23-year-old woman underwent renal transplantation for renal failure due to dysplastic kidneys. Urodynamic evaluation prior to transplantation showed decreased bladder sensation and increased bladder capacity, probably because of congenital polyuria. One week after surgery, dry mouth disappeared, and urine volume normalized gradually. Urodynamic studies performed three and ten months after transplantation showed improvement of bladder function, and the frequency/volume charts of urination also showed normalization of urine volume. Forty-five months after transplantation, renal and bladder functions were almost normal. Bladder dysfunction involves numerous factors, but the primary cause was probably congenital polyuria in the present case. This case suggested that blood purification and correction of urine volume by renal transplantation may lead to restoration of normal bladder function.

Introduction

Renal transplantation has been established as a treatment for chronic renal failure. However, its effects on urological organs have not been fully elucidated. Particularly, there have been only limited reports regarding the involvement of the lower urinary tract, including the bladder and urethra. Here, we report a case in which polyuria, bladder sensation, and bladder capacity were improved following renal transplantation, and the results of urodynamic studies are presented with a review of the relevant literature.

Case report

The patient was a 23-year-old woman whose chief complaint was polyuria. Her past history was non-contributory. She was born after 42 weeks of gestation and weighed 2,040 g at birth. Postnatal hospitalization lasted for 14 months due to low birth weight and developmental retardation of her weight. Polyuria and low specific gravity of urine were found. Renal function was also impaired, and diabetes insipidus was suspected, but the cause was unclear. At the age of 5, she was diagnosed with chronic renal failure due to dysplastic kidney. Renal function progressively worsened, with pain in the lower extremities, due to ectopic calcification associated with polyuria. Blood purification treatment was required. Her family requested renal transplantation, with her father as a donor. The 75 g OGTT test showed mild diabetes mellitus pattern, and serum HbA1c was 7.0% (normal range 4.3 - 5.8%).

Video urodynamic study was performed before transplantation (Fig. 1, 2). The patient was placed in the supine position at the filling phase, and in the sitting position at the voiding phase. Contrast medium was instilled at a speed of 30 ml/min through a 12 Fr. urethral catheter. Bladder pressure was measured with a 4.7 Fr. urethral catheter. Several parameters, including first desire to void (FDV), maximum desire to void (MDV), bladder compliance, uninhibited contraction, vesicoureteral reflux, bladder deformity, maximal detrusor contraction pressure, and residual volume, were evaluated before the operation. The results and daily frequency-volume charts indicated left vesicoureteral reflux (grade 2), increased bladder capacity, and decreased bladder sensation. No uninhibited contraction or bladder deformities were found. There were no abnormalities during the voiding phase. On February 24, 1998, renal transplantation was performed with the left antireflux procedure. The kidney was placed in the right iliac fossa, and the ureter was implanted by the

Politerno-Leadbetter method. Cyclosporin, azathioprine, and prednisolone were used as immunosuppressant drugs. The postoperative course was uneventful. Serum creatinine level decreased to 0.8 mg/dl one week after transplantation. Approximately one week after surgery, dry mouth had improved somewhat, and urine volume was normalized. Urodynamic evaluation performed 3 and 10 months postoperatively demonstrated progressive improvement in urinary desire to void (Table 1). Following correction of the desire to void, voided volume also showed gradual normalization. No rejection has occurred. At 51 months postoperatively, the serum creatinine level is 1.1 mg/dl. Urine volume for 24 h is 1.5-2.0 l, and voided volume is 100-300 ml (no residual urine). Vesicoureteral reflux has disappeared (Fig. 3).

Discussion

Evaluation of bladder function prior to renal transplantation is of great importance as patients with chronic renal failure can develop various bladder dysfunctions. In particular, it is strongly recommended that patients with some clinical symptoms involving the lower urinary tract, long-term anuria, or past urological history should undergo urodynamic study, including cystometry, before renal transplantation [1]. A typical form of bladder dysfunction in patients with chronic renal failure under blood purification therapy is the so-called "defunctionalized bladder," which is characterized by low capacity and compliance of the bladder on urodynamic study. The physiological and pathophysiological characteristics of this disorder are not fully understood. However, it has been suggested to involve atrophic changes of the bladder smooth muscle that accompany long-term oliguria [2]. It has been reported that, even in cases with long-term bladder defunctionalization, urinary dysfunction can be improved with bladder training after renal transplantation [1, 3]. The present case showed polyuria, decreased bladder sensation, and increased bladder capacity. These findings resemble those of bladder dysfunction in diabetes patients (diabetic cystopathy). "Diabetic cystopathy" is characterized by a decrease in bladder sensation, increase in bladder capacity, and decrease in bladder contraction [4]. There are various patterns of bladder dysfunction in diabetes patients, and the clinical entity can be explained mainly by impairment of the peripheral nerves [4, 5]. In the present case, the patient demonstrated mildly impaired glucose tolerance. However, as treatment was not required for this glucose intolerance, the condition may not be considered DM cystopathy. Long-term polyuria may induce a decrease in bladder sensation and an increase in bladder capacity. Cystometric study indicated an increase in bladder capacity in diuretic rats as compared with controls [6]. By recording micturition pattern

and cystometry, Malmgren *et al.* demonstrated that bladder capacity and micturition volume were increased in rats with heredity diabetes insipidus [7]. In the present case, the urodynamic parameters of the emptying phase, such as maximal detrusor contraction pressure, were not impaired. This was different from the findings in DM cystopathy, suggesting that the primary cause of bladder dysfunction in this case was polyuria. It is rare that renal failure patients who are candidates for transplantation maintain polyuria. In such cases, it is of interest to understand the changes in bladder sensation and bladder capacity before and after renal transplantation. However, to our knowledge, there have been no previous reports of such studies. In a series of animal experiments, Kang and colleagues reported that acute bladder distention did not change bladder capacity [8]. According to their report, bladder function of the overdistended bladder normalized within a few days. We observed that indwelling catheter management restores normal bladder capacity in acute urinary retention of patients with prostate hypertrophy. However, it is unclear how long-term bladder distention improves. In the present case, thirst disappeared one week after transplantation, and bladder sensation showed marked recovery three months postoperatively. Daily water intake control may contribute to this recovery of bladder function.

Cyclosporin used as an immunosuppressive agent may have an effect on the smooth muscle of the bladder. Kitani *et al.* reported that cyclosporin relaxed bladder smooth muscle tone and increased urethral resistance in rabbits [9]. There has also been a clinical report that cyclosporin induced urinary retention [10]. Although cyclosporin may have an effect on the storage phase of bladder function, its effect and the recovery of bladder function in the present case remain unclear.

In the present case, it was difficult to evaluate the changes in bladder

function precisely because of the antireflux surgery and placement of the kidney in the iliac fossa. However, the blood purification and corrected urine volume following renal transplantation may have led to the restoration of bladder function.

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Legends to Table and Figures

Table 1

Changes in the parameters of video urodynamic study before and after transplantation.

Fig. 1

Cystometography before transplantation. Bladder capacity was 990 ml.

Fig. 2

Cystometography before transplantation.

Left vesicoureteral reflux grade 2 was observed.

Fig. 3

Cystometography 45 months after transplantation. Bladder capacity was 354 ml.





