

Trauma and reconstruction

Kidney transplantation with vesicostomy in small-bladder capacity neurogenic bladder dysfunction patient: A case report

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ABSTRACT

Spinal cord injury often results in neurogenic bladder condition and eventually lead to an end-stage renal disease requiring kidney transplantation. However, transplantation in abnormal bladder carries special considerations. We report a case of an adult male with end-stage chronic kidney disease and small bladder capacity after having spinal cord injury. The evaluation of videourodynamic showed reduced compliance and detrusor overactivity during filling phase. Kidney transplantation and vesicostomy was performed. Eighteen months follow-up after surgery showed that kidney function could be maintained. The prevention of increasing bladder pressure and UTI should be monitored to prevent the damage of the graft kidney.

Introduction

In the United States, neurogenic bladder dysfunction was found in 70–80% of patients with spinal cord injury. The bladder may become overactive or underactive depends on the nature, level, and extent of nerve damage.¹

Some neurogenic bladder dysfunction patients were able to preserve adequate bladder function temporarily but gradually it decreased and lost most of bladder function. The progression of lower urinary tract disease after spinal cord injury was slowly developed in years. This process resulted in renal function decrease and chronic renal failure which ultimately need kidney transplantation.^{1,2}

We reported chronic renal failure patients with high pressure and small-bladder capacity neurogenic bladder dysfunction who was managed by kidney transplantation and vesicostomy.

Case presentation

A 31-year-old male was diagnosed with end-stage chronic renal failure with eGFR 10 mL/min/1.73 m². The patient was anuric and underwent chronic hemodialysis. The patient had history of fallen from a cliff 10 years ago and he had difficulty to urinate since the accident. MRI examination were carried out and revealed vertebral fracture at the level of L1-L2 (Fig. 1).

Videourodynamic investigation revealed reduced compliance and phasic detrusor overactivity during filling phase. There was pain related to bladder filling and the bladder capacity was reduced (53–72 mL). The voiding phase was unable to be determined due to anuria. The bladder neck was opened but the patient was unable to have spontaneous micturition with significant residual urine (Fig. 2A–D).

After discussing about several options for urine diversion, the patient underwent renal transplantation and cutaneous vesicostomy (Fig. 2E). There was gradual improvement of kidney function parameters after surgery. The improvement of the allograft kidney function was described by the increase of the haemoglobin level, the decrease of serum ureum and creatinine level, and maintenance of eGFR (Table 1). The double J stent of the allograft kidney has been removed a month after surgery. An indwelling catheter was inserted into the bladder through vesicostomy channel for maintaining the dryness and it replaced routinely every four weeks. Evaluation in 18 months after surgery, the patient has one episode of symptomatic UTI which managed with oral antibiotics; otherwise, the patient condition was good, and the value of kidney function was maintained.

Discussion

The spinal cord injury resulted in neurogenic bladder dysfunction in our case. Ischemia of the bladder walls in neurogenic bladder often

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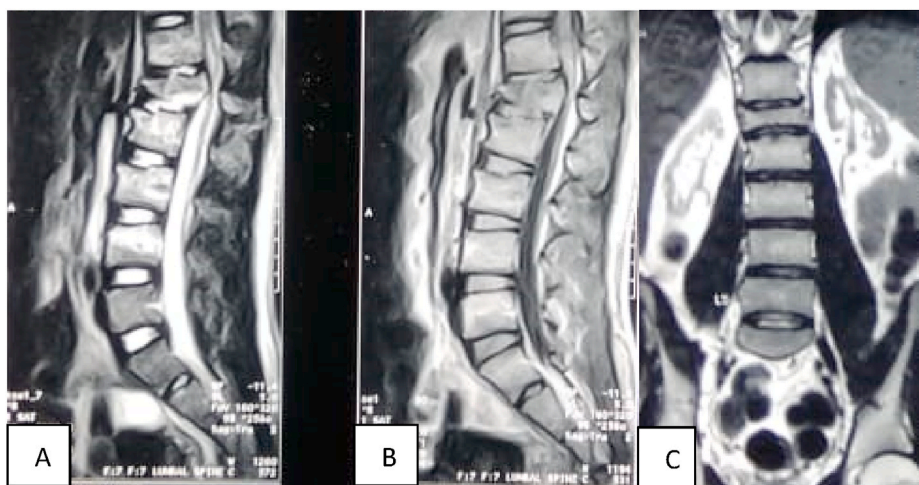


Fig. 1. MRI examination on patient revealed spinal cord injury.

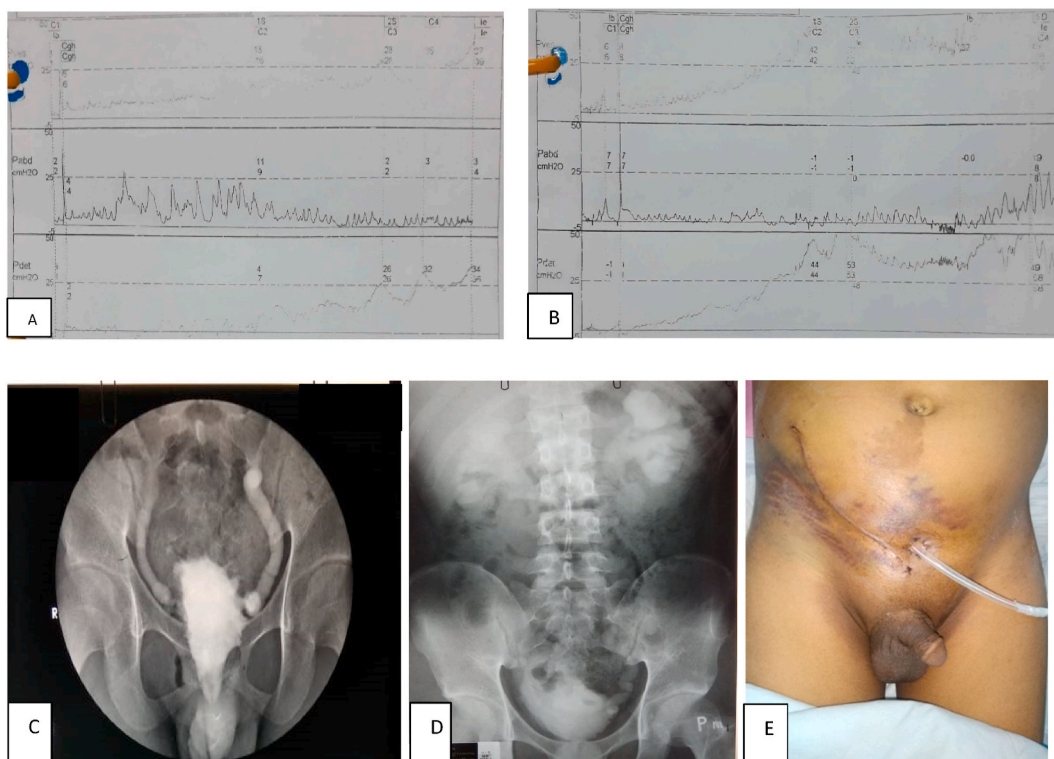


Fig. 2. Videourodynamic showed reduced compliance and detrusor overactivity (2A, 2B). Fluoroscopy revealed bilateral hydroureter and hydronephrosis (2C). Post micturition picture showed residual urine (2D). Appearance after kidney transplantation and vesicostomy (2E).

Table 1
Progression of kidney function prior and after transplantation.

	Before Surgery	After surgery	At the day of discharge	2 weeks after discharge	2 months after discharge	18 months after discharge
Hb	9.6	8.8	8.4	9.7	11.2	12
Ureum	114	62	52	52	36	42
Creatinine	7.9	6.0	1.3	1.2	1.2	1.5
eGFR	10	14	63	68	68	54

resulted in fibrosis and thickening of detrusor. Bladder high pressure and upper tract dilation caused by lower urinary tract obstruction and vesicoureteral reflux deteriorate kidney function and ended up as chronic renal failure.¹

There are many aspects should be concerned in management of neurogenic bladder patient who will undergo kidney transplantation. Bladders in transplant patients were usually atrophic because of long term anuria. The use of immunosuppressant in renal transplantation

impaired the patient's immunity while UTI, VUR, and chronic residual urine are risk factors for renal graft failure.² While augmentation cystoplasty offers transplant recipients increased urinary storage capacity and reduced bladder pressure, this procedure itself is not without any consequences. Study conducted in 2016 comparing patients with bladder augmentation with normal bladder, showed that there was 3.57 risk of graft failure in patients using bladder augmentation. Five years graft survival in reconstructed patients was 66.6%.³

There was still a debate about the perfect timing of lower urinary tract reconstruction in patients without preexisting diversion or augmentation. Patients might require bladder augmentation or complete urinary diversion before, during, or after transplantation. Proper healing of the urinary tract in the absence of immunosuppressive drugs and corticosteroids was the proponent argument of pretransplant augmentation. Having this separate procedure at least 6 weeks before transplant was recommended by most investigators. Performing augmentation first might be associated with a lower rate of complications, specifically to the ureter, and graft loss. The other investigators advocate that optimal timing of augmentation or urinary diversion was post transplantation while the renal function was stabilized, and immunosuppressive drugs are reduced. As there was no significant difference between patients who surgically treated for bladder dysfunction before transplantation and after transplantation in complications, graft survival and symptomatic UTI after transplantation, augmentation cystoplasty before or after transplantation seemed to have similar outcomes.³

Our patient had objection to undergo augmentation cystoplasty prior to kidney transplantation. Among different types of urinary diversion, he preferred to have vesicostomy due to his familiarity with indwelling urethral catheter since he got spinal cord injury. Vesicostomy in neurogenic bladder is common and accepted procedure, especially for its simplicity and effectiveness. Study by Dönmez et al. showed that vesicostomy was beneficial to prevent upper urinary tract deterioration, reduce febrile UTI, and preserve renal function in neurogenic bladder patient during long-term evaluation.⁴ In our case, the patient wished to be continent, so catheter was inserted through vesicostomy channel and changed regularly without any difficulty. For 18 months follow-up, kidney function can be maintained.

Vesicostomy as a urinary diversion in kidney transplantation was

reported in pediatric cases with small bladder by Viswanathan et al. They found that vesicostomy is a simple procedure to perform either before or during the transplantation and is easy to take care of. Furthermore, a vesicostomy does not preclude bladder augmentation.⁵ A dilemmatic issue after vesicostomy creation in this case is for how long it will be sustained, when to close it and switch to bladder augmentation. Recurrent urethral leakage and stomal stenosis upon revisions of vesicostomy can be one indication of closing a vesicostomy and to follow with another procedure. Evaluation of the urodynamics and type of continent urinary diversion must be discussed to help the decision of changing from vesicostomy into more invasive procedure.^{2,3}

Conclusion

In our case, kidney transplantation and vesicostomy have a satisfying eighteen months outcome. This could be a safe temporary option in the management of end-stage chronic renal failure caused by high pressure and small capacity neurogenic bladder. The prevention of increasing bladder pressure and UTI need to perform to prevent the damage of the graft kidney. Extensive evaluation should be done when changing to continent diversion will be performed.

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