

Percutaneous Urine Sampling from Renal Pelvis: A Minimally-Invasive Method to Determine the Origin of Post-Transplant Proteinuria

Ali Babaei Jandaghi¹,
Hamidreza Badeli¹,
Afagh Hassanzadeh Rad¹,
Ramin Pourghorban²

1 Pediatric Diseases Research Center, Guilan University of Medical Sciences, Rasht, Iran.

2 Department of Radiology, Shohada-e-Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

***Corresponding Author**

Hamidreza Badeli, MD.

Pediatric Diseases Research Center, Guilan University of Medical Sciences, Rasht, Iran

E-mail: badeli@gums.ac.ir

Mobile: 098-9112397157

Fax: 0981333369061

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Introduction

Focal segmental glomerulosclerosis (FSGS) caused by glomerular disease in childhood and adulthood will progress to end stage renal disease and it accounts for a high rate of recurrence in post transplanted patients.

One of the early findings of recurrent post-transplantation FSGS is detecting heavy proteinuria during a short time after a normal urine protein (1,2). In few cases, the recurrent FSGS may be detected by abrupt proteinuria during first days after transplantation which may

Abstract

A 14-year-old boy with end-stage renal disease secondary to focal segmental glomerulosclerosis complicated with heavy proteinuria received a non-related living kidney transplantation. Postoperatively he continued to excrete higher level of proteinuria. Allograft biopsy showed mild mesangial expansion and hypercellularity. Urine sample was collected from allograft renal pelvis under local anesthesia and ultrasound guidance.

Based on the importance of heavy proteinuria and lack of definite method of differentiating its source during the early weeks after kidney transplantation, it seems that percutaneous renal pelvis urine sampling may be noted as a preferred method of detecting the source of proteinuria.

Keywords: Focal Segmental Glomerulosclerosis; Kidney Transplantation; Ultrasonography; Proteinuria; Allografts.

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be difficult to discriminate its source especially if the native kidney has residual urine output with proteinuria.

Since proteinuria is an independent risk factor for the occurrence of cardiovascular diseases and can predict the allograft kidney damage and loss, the early detection of the source of proteinuria is of utmost importance (3,4). Determining the source of proteinuria remained a complex issue, especially in the early phases. It is postulated that the native kidney proteinuria usually resolves within 8-10 weeks after transplantation, and continuous and increased level of proteinuria

during first months after kidney transplantation may suggest the recurrence of disease in the allograft kidney (5,6). In addition, the histopathologic findings of FSGS by biopsy may not be evident in the early post-transplantation phase (7).

Therapeutic approach to recurrent FSGS in allograft kidney may require several manipulations such as plasmapheresis, intravenous immunoglobulin, Rituximab, increasing calcineurin inhibitor (CNI) dose, and etc. To shed the light on the transitional phase before disappearance of native kidney proteinuria and positive biopsy findings in the allograft kidney, clinicians usually require a definite diagnostic method for understanding the source of proteinuria during the first months after transplantation. Therefore, we aimed to introduce percutaneous renal pelvis urine sampling as a minimally-invasive technique to differentiate native from allograft proteinuria.

Technical report

A 14-year-old boy with end-stage renal disease (ESRD) secondary to FSGS with heavy proteinuria (10-20 g/d) underwent a non-related living kidney transplantation. Renal function returned to normal level during the first week after kidney transplantation. Thereafter, he continued to excrete a high level of proteinuria. At the end of first month after kidney transplantation, the patient developed generalized edema with a low serum albumin (<2.5 g/dL) and a mild increase in creatinine level (1.8 mg/dL).

As the high level of proteinuria was detected since the first weeks and continued up to the first month (15 g/d) and regarding the difficulty of distinguishing the origin of proteinuria (native vs. transplanted kidney), the investigators consulted with the transplant urologist for urine sampling from native and allograft ureters at the time of removing ureteral stent. However, the procedure was unsuccessful.

To differentiate the various etiologies of allograft dysfunction, percutaneous renal biopsy was done at the first month after transplantation. Allograft biopsy showed mild mesangial expansion and hypercellularity. No glomerulosclerosis, significant interstitial fibrosis, or tubular atrophy was identified.

Immunofluorescent study was unremarkable. No histopathological finding, favoring antibody mediated rejection, T-cell mediated rejection, CNI toxicity, or polyomavirus was identified. Electron microscopy results showed extensive effacement of visceral foot processes and mesangial matrix expansion.

Assuming the recurrence of FSGS as the cause of nephrotic range proteinuria, daily plasmapheresis for 7 days, IVIG and 4 doses of Rituximab were administered. Also, an adjunct therapy with oral galactose (8) was tried to reduce the level of proteinuria.

The nephrotic range proteinuria and edema continued to present, and the patient had to receive a required dose of intravenous albumin. Regarding the lack of response to above mentioned treatments and in an attempt to make a decision for a possible native nephrectomy, the investigators consulted an interventional radiologist to take urine sample from allograft renal pelvis.

Urine sampling was done from the allograft renal pelvis under guidance of real-time ultrasonography with local anesthesia. Color Doppler study was used during the advancement of the sampling needle (26-gauge spinal needle) into the mildly dilated renal pelvicalyceal system to avoid the nearby intra-renal vascular structures. Simultaneous voided urine sample was also obtained. Both urine samples were analyzed for the protein level and creatinine ratio. Allograft urine had higher level of protein and albumin comparing with the voided urine.

Discussion

When clinicians cannot differentiate the source of heavy proteinuria in the first few weeks after transplantation, percutaneous renal pelvis urine sampling might be helpful as a minimally invasive method, especially when the clinical and laboratory findings and the results from renal biopsy are not quite indicative of the source of proteinuria.

There are several studies mentioning the transplant kidney as an origin of proteinuria during the first 2 months after transplantation (9-11). In this case report, clinicians had to find out the exact source of proteinuria because of following two main issues:

First, the patient had residual urine output and a heavy proteinuria before transplantation, and the pre transplantation heavy proteinuria may be assumed as a native kidney proteinuria. In patients with native kidney proteinuria, nephrectomy may decrease the level of proteinuria and consequently eliminate the administering of the albumin and repeated hospital admission. But there are some previous investigations which noted that pretransplantation nephrectomy might increase the risk of recurrence since native kidneys may have an absorbent effect and decrease the concentration of plasma permeability factors (12-13).

Second, nephrotic range proteinuria in the first weeks after transplantation may suggest the FSGS recurrence of the allograft kidney and indicate a different therapeutic approach.

Regarding the native kidney proteinuria, the reflux of urine from the native kidney to the allograft kidney should be taken into consideration. However, a lower level of protein and albumin in the voided urine compared with that from the allograft kidney could have rule out the probability of reflux in our patient.

Based on the clinical importance of heavy proteinuria after kidney transplantation of patients with FSGS, and lack of a definite method to differentiate its source during the early weeks after kidney transplantation, it seems that percutaneous renal pelvis urine sampling can be considered as a helpful method to determine the source of proteinuria.

Conflict of interest

The authors declared no conflict of interest.

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