**Case Report**

**Late Occurrence of Pelvi-ureteric Junction Obstruction in Renal Allograft and Live-Related Kidney Donor**

Kurian George¹, Ganesh Gopalakrishnan², Said Abdallah Al-Mamari¹, S. B. Viswaroop²

¹Department of Urology, The Royal Hospital, Muscat, Oman, ²Department of Urology, Vedanayagam Hospital, Coimbatore, Tamil Nadu, India

**ABSTRACT.** Pelvi-ureteric junction (PUJ) obstruction is an enigmatic condition. While in a reasonable majority it is clear cut, the diagnosis and the need for intervention in the remainder is still a challenge. We would like to share the details of two cases, one in a transplant recipient and the other in a living kidney donor, and propose an explanation as to why PUJ obstruction becomes manifest after such a long period of time. In this presentation, we would like to propose that forced drinking of fluids by patients who have an equivocal PUJ and a single kidney could tilt the balance resulting in overt PUJ obstruction.

**Introduction**

Pelvi-ureteric junction (PUJ) obstruction is the most common congenital cause of upper tract obstruction. In the vast majority, the diagnosis is cast iron; however, in the remainder, it is equivocal and poses a dilemma regarding the appropriate line of treatment.

It is a common clinical situation when patients in the later life get a health check and hydronephrosis is detected. More commonly, for nonspecific abdominal symptoms, an ultrasound is also done, and again, hydronephrosis might be detected. The dilemma worsens when it is realized that the patient has a single kidney and has been asymptomatic. The diethylenetriaminepentaacetic acid (DTPA) renal scan is invariably equivocal. The long-term outcome is not clear, and for want of data, most patients would be subjected to surgical treatment.¹

It is conceivable that if these patients had undergone antenatal ultrasound (US), while *in utero*, then they would have been subjected to intervention in the postnatal period, especially if they had a single kidney.

A single-kidney situation exists in a renal allograft. In a deceased donor transplant, one does not usually have any presurgical details of the kidney and it is reasonable to assume that kidneys with obvious PUJ obstruction would be rejected. In a live kidney donor, we have at our command a battery of tests to evaluate the kidney before transplantation. While this is not a common occurrence, there
are reports in literature of PUJ obstruction occurring in a renal allograft,2–4 considerable time after the transplantation. This should not be confused with the term “marginal kidney” having PUJ obstruction where the diagnosis is made pretransplant and the kidney nevertheless transplanted due to lack of suitable donors, after ex vivo pyeloplasty.

We would like to share the details of two cases, one in a transplant recipient and the other in a living kidney donor, and propose an explanation as to why PUJ obstruction becomes manifest after such a long period of time.

**Case Reports**

Written informed consent was obtained from both patients before reporting the cases.

**Case 1**

A 54-year-old woman underwent live-related donor kidney transplantation in 2011. The donor was her son, and the left kidney was used. The donor kidney was normal with no suggestion of even equivocal PUJ obstruction (Figure 1). The patient was not a diabetic but was on medication for hypertension. Standard triple immunosuppression was used, and the postoperative period was uneventful. She maintained stable graft function until 2017. The only significant feature in the history was that she used to consume 6 L of fluid every day on the advice of her physician who asked her to take “plenty of fluids.” She was on regular checkup and as she continued to show normal graft function, her physicians did not feel the need to carry out any imaging of the allograft.

In January 2017, she observed a sudden decrease in urine output at which time the creatinine was found to be elevated (238.68 µmol/L). US of the graft showed significant hydronephrosis (Figure 2). DTPA renogram suggested PUJ obstruction to the renal allograft. DJ stenting was done and creatinine returned to normal (88.4 µmol/L). The stent was removed in April 2017, and the very next day, she experienced pain in the graft area and anuria. US-guided percutaneous nephrostomy was performed. Three days later, she underwent YV pyeloplasty. Following this procedure, increased drain output was noted and re-exploration was carried out. Native nephrectomy was considered with a view to using the ureter if needed for anastomosis. However, this was abandoned and anastomotic leak was repaired. Three months later, the DJ stent placed in the allograft was removed. Three weeks after stent removal, decreased urine output and rising creatinine were noted which settled spontaneously in two days.

At this point, she was referred to our center...
for a second opinion. While under our care, an US and Lasix renogram were performed (Figures 3 and 4), and this confirmed an obstructed trace. While waiting in the ward, she developed pain in the graft and decreased urine output with rapid elevation of creatinine to 442 µmol/L. An emergency DJ stenting was carried out. The renal function returned to normal, and three weeks later, she underwent a Boari flap procedure. At surgery, there were dense adhesions between the allograft ureter and the lower pole of the kidney. It was obvious that the previous anastomosis was kinked and obstructed. The renal pelvis was dissected in the Gil-Vernet plane and was completely freed. The native right ureter was identified to carry out a pyeloureterostomy, but this idea was abandoned as the vascularity of the ureter was doubtful, and a Boari flap was considered a better option. The flap was raised from the anterior wall of the bladder and joined to the pelvis. No attempt was made to provide an anti-reflux anastomosis. The anastomosis was stented.

The postoperative period was uneventful. The bladder catheter was removed after two weeks and the stent two weeks later. At discharge, she was voiding normally; creatinine was normal at 70.72 µmol/L, and urine was sterile on culture. She was reviewed at eight weeks, and a repeat DTPA scan showed patent drainage.

Case 2

The second case is that of a living kidney donor, an Omani national, who underwent left donor nephrectomy in October 2016. At that time, there was no suspicion of PUJ obstruction in the right kidney (Figure 5). Being from a country with a hot-dry climate and a propensity for stone disease, this patient was advised to drink about 4–5 L of fluids per day. One year after surgery, he presented with right loin pain and elevated creatinine level (192 µmol/L). Non contrast computerized tomography showed features of PUJ obstruction (Figure 6).
and DTPA scan showed an obstructed trace (Figure 7). Cystoscopy and right retrograde pyelogram suggested a lower polar crossing vessel (Figure 8). This was noted on the preoperative angiogram (Figure 9) but was considered an insignificant finding as there was no secondary effect or complaint. The patient was unhappy to undergo any further surgical intervention; hence, a DJ stent was placed after which the serum creatinine level was normalized. The stent was removed three months later and he remains stable with no pain or changes in serum creatinine over six months, and he continues to be on close follow-up.

**Discussion**

PUJ obstruction occurs in one out of 500 live births. The incidence in a solitary kidney is not very clear. Albani et al in a study of 319 patients with PUJ obstruction reported that 13 had a solitary kidney. The incidence in a renal allograft is probably low, considering that there are only sporadic cases reported in literature. The reported cases have been in living donor
transplantation. It is low because kidneys are screened thoroughly in live donor transplantation, and in a deceased donor program possibly, many of these kidneys may be rejected. Thus, while kidneys are adequately screened, it is interesting to speculate as to how apparently normal kidneys develop PUJ obstruction.

One mechanism is probably the use of a box-shaped pelvis in a donor. At a cursory glance, one could pass off the donor’s intravenous urogram (in case 2) as normal; however, a closer look might indicate a somewhat box-shaped configuration. The angiogram also showed an accessory lower polar vessel. Whether a combination of box-shaped pelvis, lower polar vessel, and increased fluid intake tipped the balance to result in overt obstruction is a tenable hypothesis. It is conceivable that in a two-kidney situation, borderline PUJ does not manifest because the index kidney is protected. It is also well known that PUJ obstruction is usually bilateral in 10% of people; if one kidney is nonfunctional or poorly functioning and if the contralateral PUJ is suspected, then nephrectomy on the affected side could pose a strain on the so-called normal side and result over time in overt PUJ obstruction. Hence, long-term follow-up of such kidneys is recommended, but how long is not clear.

In the second patient, the preoperative intravenous urogram and imaging showed no suspicion of a crossing vessel. It is important that a computed tomography angiogram which is the standard investigation in donor evaluation today needs to be looked at carefully with respect to vascular anomalies. Transplant surgeons need to be constantly aware of keeping in mind that living donors should come to no harm.

Increased urine output is a well-accepted phenomenon in a live donor transplantation. It is conceivable that continuing to drink copious amount of fluid approximately 5–6 L/day as in both our patients could pose a strain on the PUJ and over time result in obstruction. This is amplified by the fact that following a Lasix renogram, the patient went into anuria with elevated creatinine. Increased diuretic load was also proposed by Venkatramani et al as a possible mechanism. Shabtai et al proposed denervation as a possible mechanism for the occurrence of PUJ obstruction. Our patient did have pain in the graft on both occasions when the kidney was obstructed probably negating the denervation concept.

**Conclusion**

These cases and others published in the
urologic literature emphasize the fact that we need to be more vigilant while accepting kidneys for transplantation from living donors. *Primum non nocere* should be the watchword. Patients with single kidneys and suspicious pelvi-ureteric anatomy should not be forced to drink large volumes of fluids for fear of possible PUJ obstruction occurring later.

**Conflict of interest:** None declared.

**References**

1. Kinn AC. Ureteropelvic junction obstruction: Long-term follow up of adults with and without surgical treatment. J Urol 2000; 164:652-6.

2. Shabtai M, Nativ O, Dreznik Z, Jacob ET. Decompensated ureteropelvic junction obstruction in renal allograft. J Urol 1988;139: 578-9.

3. Venkatramani V, Mukha RP, Kekre NS. Equivocal pelvi-ureteric junction obstruction manifesting in a renal transplant recipient. Indian J Urol 2012;28:350-2.

4. Waltzer WC, Gonda A, Lehr H, et al. Management of transplant ureteropelvic junction obstruction by dismembered pyeloplasty. Transplant Proc 1985;17:2149-51.

5. Albani JM, Desai MM, Gill IS, Streem SB. Repair of adult ureteropelvic junction obstruction in the solitary kidney: Effect on renal function. Urology 2006;68:718-22.

6. Khosroshahi HT, Oskui R, Shoja MM, Tubbs RS, Ardalan MR. Time-dependent variations in urine output after renal transplantation. Transplant Proc 2007;39:932-3.

Date of manuscript receipt: 22 November 2018.
Date of final acceptance: 19 December 2018.