



Department of Pediatric Surgery,
Lady Hardinge Medical College &
Kalawati Saran Children's Hospital,
New Delhi, 110001, India

* Correspondence to: Amit Gupta,
Department of Pediatric Surgery,
Post Graduate Institute of Child
Health, Sector-30, Gautam Buddh
nagar, Noida, 201303,
India. Tel.: +91-9911295661
ashitoshpd106@gmail.com
(A.D. Pokharkar)
rohitmesh2905@gmail.com
(R.B. Meshram)
charuarrow@gmail.com (C. Yadav)
amitpedsurgeon@gmail.com
(A. Gupta)
rajiv_chadha_01@yahoo.com
(R. Chadha)

Keywords

Pelviureteric junction obstruction;
Ureteropelvic junction obstruction;
Postobstructive diuresis; Pyelo-
plasty; Hydronephrosis

Abbreviations

POD, Postobstructive diuresis; UUO,
unilateral ureteric obstruction;
PUJO, pelviureteric junction
obstruction; AQP, aquaporin; ANP,
atrial natriuretic peptide; OK, oper-
ated kidney; NCK, normal contralat-
eral kidney; APD, antero-posterior
diameter; HN, hydronephrosis; DJ,
Double J; NT, Nephrostomy tube;
PUC, Per urethral catheter; USG, Ul-
trasonography; SFU, Society for Fetal
Urology; PT, Renal Parenchymal
thickness on USG; DRS, Dynamic
Renal Scan; EC, Tc-99 m L,L-
Ethylene dicysteine; GFR, Glomer-
ular Filtration Rate; SG, Specific
Gravity; FeNa⁺, fractional excretion
of sodium; FeK⁺, fractional excre-
tion of potassium; FePO₄⁻, fractional
excretion of phosphate; FeMg⁺,
fractional excretion of magnesium;
ORS, Oral Rehydration Solution;
iNOS, Intravenous Nitrous oxide syn-
thetase; PGE2, Prostaglandin E2;
CrCl, Creatinine clearance

Received 2 October 2025

Revised 4 March 2026

Accepted 7 March 2026

Available online 13 March 2026

Original Research

Postobstructive diuresis and its biochemical characteristics in children undergoing pyeloplasty for unilateral pelviureteric junction obstruction - a prospective observational study



Ashitosh D. Pokharkar, Rohit Bhashkar Meshram, Charu Yadav,
Amit Gupta*, Rajiv Chadha

Summary

Background

The characteristics of postobstructive diuresis (POD) following relief of unilateral ureteric obstruction (UUO) has been studied in animals, but there is scarcity of literature on translation of these findings in human pediatric patients with unilateral pelviureteric junction obstruction (PUJO) following pyeloplasty.

Objective

The primary objective was to assess the biochemical characteristics of the POD from the operated kidney. Our secondary objective was its clinical implications and identifying predisposing factors.

Study design

A prospective observational study was conducted on pediatric patients who underwent pyeloplasty for unilateral PUJO. Post-operative differential urine output (UO) from the operated kidney (OK) was compared with the normal contralateral kidney (NCK). Patients were divided into two groups: Group-1 with POD [n = 32; 60.4%] and Group-2 without POD [n = 21; 39.6%]. Data was collected for the occurrence of POD, urine biochemistry and postoperative electrolyte derangement requiring nephrology consultation.

Results

POD occurred in Group-1 patients within 48-h after surgery. They had statistically significant lower median renal parenchymal thickness and higher median renal pelvis antero-posterior diameter

(APD), differential ratio of kidney size and proportion of grade-4 hydronephrosis (HN) than Group-2 patients. None of the kidneys with PUJO were small in size. The abnormal biochemistry of POD from OK matched with experimental animal studies except the FeK⁺ excretion which was significantly higher [13,14,15]. Four patients in Group-1 required nephrology consultation for prolonged POD with electrolyte derangement: one concomitant pyelonephritis in NCK and other three with past history of renal injury. Predisposing factors for POD were parenchymal thinning in enlarged kidneys with SFU grade-4 hydronephrosis and higher renal pelvis APD.

Discussion

Urine biochemistry in human pediatric subjects matches animal studies to a large extent except FeK⁺ excretion and suggests compensation by NCK to maintain homeostasis during POD. Besides the predisposing factors in OK, concomitant or pre-existing (subclinical) functional injury to NCK despite normal appearance on preoperative imaging may limit the compensation resulting in electrolyte derangement.

Conclusions

High UO from OK mainly contributes to POD. Larger hydronephrotic kidneys with parenchymal thinning are more predisposed to POD. Our observations reveal that concomitant or prior subclinical injury to NCK may not be apparent on preoperative imaging and it could be a warning signal to the clinician for occurrence of electrolyte derangement with prolongation of POD and such patients should be monitored closely during post-operative period.

<https://doi.org/10.1016/j.jpuro.2026.105878>

1477-5131/© 2026 Journal of Pediatric Urology Company. Published by Elsevier Ltd. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

Background

Postobstructive diuresis (POD), although well-recognized in bilateral ureteral obstruction, is considered rare in unilateral pelviureteric junction obstruction (PUJO) due to compensation by unaffected kidney [1–3]. *Physiologic* POD, usually self-limiting (24 h or less), occurs due to excretion of accumulated solutes and free water from volume expansion during obstruction [1,4]. *Pathologic* POD lasts longer than 48 h [3,4] characterised by prolonged and inappropriate handling of water and/or solutes by down regulation of sodium transport and aquaporin (AQP) channels, nephrogenic diabetes insipidus (NDI) and/or altered regulation of atrial natriuretic peptide (ANP) [2,5]. There is very limited literature on biochemistry of POD following pyeloplasty for unilateral PUJO in human pediatric patients, with a normal contralateral kidney (NCK) on imaging [6–11]. Two studies observed electrolyte derangement during POD mandating close clinical observation [10,11].

Resource limitation restricts availability of double-J stent at our centre. It is our usual practice to place an external trans-anastomotic stent along with a nephrostomy tube (NT) draining the operated kidney (OK) and a per urethral catheter (PUC) in bladder mainly draining the NCK, allowing separate collection of urine from the two kidneys. We observed disproportionately high differential urine output (UO) from NT which kindled the idea for this study. Literature search revealed two prospective studies in children on the differential UO and comparison of urinary biomarkers from OK and NCK, respectively [8,9]. Two more retrospective studies concentrated on the incidence and risk factors for POD but not on differential UO [10,11]. The pathophysiology of urinary biochemical derangements has been described on animal studies following release of artificially created unilateral ureteric obstruction (UUO) [12–15]. But no study has been conducted to observe *translation of these findings in human pediatric patients* with unilateral PUJO. We have addressed this lacuna in knowledge through this unique in-vivo study conducted in a true clinical setting.

Objective

The primary objective was to study the incidence and biochemical characteristics of the POD from the OK. Our secondary objective was the clinical implications and identification of predisposing factors.

Methods/study design

This is a prospective observational study conducted at a tertiary-care children's hospital over a period of two years after obtaining Institutional Ethical Committee clearance number LHMC/IEC/2019/37. A convenient sample size of 60 patients was decided based preceding 10 years data, but due to covid restrictions only 53 patients were studied. We included all children undergoing pyeloplasty for unilateral PUJO with contralateral kidney appearing normal on pre-operative evaluation with Ultrasonography (USG) and Diuretic Renal Scanning (DRS). Patients having a solitary kidney with PUJO or recurrent PUJO operated elsewhere

were excluded. Pre-operative demographic and imaging data was recorded. The USG parameters recorded were: (i) antero-posterior diameter of the renal pelvis (APD), (ii) Society for Fetal Urology (SFU) grading of the hydronephrosis, (iii) renal parenchymal thickness in mm (PT) at mid-polar region, (iv) length and breadth of both kidneys. Two parameters calculated from the USG findings were: (i) Differential size (cm), calculated as the difference of length between affected and unaffected kidneys, and (ii) Differential ratio, calculated by dividing the length of affected and unaffected kidneys. DRS using Tc-99 m L,L-Ethylene dicycysteine (EC) was done both pre- and post-operatively to assess the differential renal function (DRF) in percentage (%), the Glomerular Filtration Rate (GFR) in ml/min/1.73 m², and the drainage.

Open Anderson-Hynes dismembered pyeloplasty was performed. As a standard practice, we placed a trans-anastomotic stent in upper ureter well away from ureterovesical junction (3–4.5 Fr umbilical catheter without side holes [Vygon, India]), a 8–10 Fr Foleys catheter as nephrostomy tube [NT], and a perirenal drain, all secured separately on the skin to facilitate removal at different time periods postoperatively. A per-urethral Foley's catheter (PUC) of appropriate size was placed for bladder drainage. Intravenous fluid (IVF) supplementation was administered at maintenance rate with Ringer lactate during intra-operative and 5% dextrose + half-normal saline on the day of surgery; IVF was stopped on POD-1 after resumption of full feeds. The drain was removed 24 h after minimal or no drainage and the trans-anastomotic stent removed on postoperative day-6 followed by graded clamping of the NT over next 3–5 days. The NT was removed if patient remained asymptomatic after >24-h of clamping. PUC was removed on postoperative day-7 and later in patients with prolonged POD.

The daily differential UO (ml/kg/hour) from the OK and PUC was recorded through NT and PUC respectively, for first five postoperative days. Both the values were summed together to get the total daily UO. The number of days required for normalization of UO was recorded. The urine and blood biochemistry were recorded on postoperative day-1, 3 and 5. The urinary analysis was done separately for NT & PUC and the parameters were – urinary specific gravity (SG), pH, sodium, potassium, magnesium, phosphates, and creatinine levels. The urinary SG, albumin, and pH were measured by UroColor™10 strips for Urinalysis. The results were interpreted by a test results coding chart. Blood biochemistry parameters were blood urea, serum creatinine, serum electrolytes, serum calcium, magnesium, and phosphate.

The following variables were calculated for urine draining from NT and PUC separately: CrCl (CrCl) and the fractional excretion of sodium (FeNa⁺), potassium (FeK⁺), phosphate (FePO₄⁻), and magnesium (FeMg⁺) levels.

Polyuria definition varies broadly in literature as >2.5–6 ml/kg/hr, but recent clinical practice and literature provide a more specific definition as >4 ml/kg/hr in infants and children and >6 ml/kg/hr in newborn [16,17]. Therefore, we defined POD as total UO (NT and PUC) of >4 ml/kg/hour because all of our patients were infants and children. Accordingly, the patients were divided into two groups – Group-1 with POD, and Group-2 without POD.

Patients were monitored for any electrolyte derangement during POD and nephrology consultation was sought if present. The volume loss due to POD was replaced 4-hourly with Oral Rehydration Solution (ORS) administered as half the volume of UO for 48-h. The postoperative day for removal of the peri-renal drain, trans-anastomotic stent, NT and PUC were also recorded.

Statistical analysis

SPSS statistical software version 17.0 was used. Qualitative variables were analyzed using Chi square Test/Fischer Exact Test while quantitative variables were subjected to unpaired T-test & Mann Whitney Tests. Data was expressed as Median (IQR) and range. P-value of less than 0.05 was taken as statistically significant.

Results

Supplementary Table-1 summarises the demographic data and findings on preoperative imaging (USG and DRS). 34 children (64.2 %) were infants, there was a male predominance (n = 41; 77.3%) and the left kidney was more commonly affected (n = 42; 79.2%). In Group-1 patients, three had a history of prior renal injury and one developed pyelonephritis in NCK during postoperative period; *these four patients developed electrolyte derangement and required nephrology consultation.*

Biochemistry of urine samples from the NT (OK) and PUC (NCK) in all the 53 patients has been summarised in Table-1. Statistically significant observations comparing the affected kidney with the normal contralateral kidney were: *higher* mean UO, pH, FeNa+, FeK+, FeMg+ and *lower* SG, CrCl, FePO4- (p < 0.05). These were more pronounced in the four patients with electrolyte derangement. The UO decreased gradually over 5-days but the difference in values of all these parameters remained significant. On comparing these parameters between Group-1 & 2, the difference in NT & PUC were significant on day-1 & 3 but became insignificant on day-5 except CrCl (Supplementary Table-2).

Table-2 represents the comparison between the two groups of patients: Group-1 with POD (60.4%; n = 32), and Group-2 without POD (39.6%; n = 21). Age was comparable in both the groups. In Group-1 patients, the onset of POD occurred within 48 h after surgery in all patients: 62.5% (n = 20) on day-1 & 37.5% (n = 12) on day-2. Diuresis lasted for more than 48-h in 53.1% (n = 17) of patients and resolved by postoperative day-5 in all but four patients. Group-1 patients had a statistically significant *lower* median PT (p < 0.05) and *higher* median APD, differential ratio of the kidney size and proportion of SFU grade-4 HN (p < 0.05). Regression analysis showed PT as the most significant amongst all. None of the OK were small in size on preoperative imaging. However, there was no significant difference between the two groups with respect to preoperative SRF and GFR on DRS. Only two patients in Group-1 had SRF<10% and none had small kidneys.

The mean total UO was significantly higher in Group-1 than Group-2 on postoperative days-1 and 2 (p < 0.05) and while higher levels persisted in Group-1 patients on days 3–5, the difference was not statistically significant (Table 3). Interestingly, the difference in UO from NT and PUC was statistically significant in all patients (both Group-1 and 2) on all the five post-operative days (Table 4).

Twelve patients (22.6%) developed pain and/or flank fullness on clamping the NT following trans-anastomotic stent removal on day-6; nine (28.1%) Group-1 patients belonged to the subset where POD lasted for >48 hrs (n = 17) and three (14.3%) Group-2 had SFU grade-4 hydronephrosis. In these patients, the NT was reopened and clamping restarted after a gap of 2-days; it was removed between postoperative days 10–12. Although total UO in all patients decreased to <4 ml/kg/hr, NT output remained high in both the groups, Group-1 mean 1.7 ± 0.24 ml/kg/hr & Group-2 patients 1.4 ± 0.17 ml/kg/hr compared to overall mean 1.35 ± 0.49 & 1.06 ± 0.39 respectively which along with postoperative edema may result in inefficient (obstructed) drainage across the pelviureteric anastomosis.

Hydration was maintained with oral replacement using ORS and no intravenous fluid replacement was required clinically for POD except in *four patients in Group-1 with*

Table 1 Post-operative urine biochemistry in all patients (n = 53).

Variable	Day-1			Day-3			Day-5		
	NT (OK)	PUC (NCK)	p-value	NT (OK)	PUC (NCK)	p-value	NT (OK)	PUC (NCK)	p-value
Urine output (ml/kg/hr)	3 ± 1.07	1.96 ± 1.03	< 0.001*	2.2 ± 0.83	1.01 ± 0.54	< 0.001*	1.24 ± 0.47	0.6 ± 0.17	< 0.001*
Specific gravity	1.01 ± 0.003	1.06 ± 0.11	< 0.001*	1.01 ± 0.004	1.05 ± 0.008	< 0.001*	1.02 ± 0.035	1.05 ± 0.007	< 0.001*
Creatinine clearance (mg/ml/min)	22.81 ± 8.9	64.9 ± 5.1	< 0.001*	27.1 ± 9.8	47.48 ± 6.29	0.005*	29.1 ± 9.9	45.6 ± 12.8	< 0.001*
pH	6.9 ± 0.28	6.0 ± 0.46	< 0.001*	6.93 ± 0.25	5.78 ± 0.41	< 0.001*	6.62 ± 0.57	5.68 ± 0.53	< 0.001*
FeNa+	3.26 ± 0.94	1.57 ± 0.41	< 0.001*	1.89 ± 0.63	0.93 ± 0.26	< 0.001*	1.7 ± 0.24	0.79 ± 0.15	< 0.001*
FeK+	17.51 ± 6.43	11.5 ± 4.6	< 0.001*	16.3 ± 5.7	9.96 ± 4.6	< 0.001*	13.2 ± 4.8	8.4 ± 3.2	< 0.001*
FePO4-	11.5 ± 4.58	19.9 ± 6.4	< 0.001*	9.2 ± 2.99	16.25 ± 4.16	< 0.001*	9.96 ± 3.8	19.99 ± 5.23	< 0.001*
FeMg+	8.63 ± 2.39	3.48 ± 0.89	< 0.001*	5.9 ± 1.5	2.72 ± 0.95	< 0.001*	6.26 ± 1.42	2.52 ± 0.69	< 0.001*

Table note: Abbreviations used in the table – OK= Operated kidney, NCK= Normal contralateral Kidney, NT= Nephrostomy tube, PUC = per urethral catheter, Fe= Fractional excretion, Na+ = Sodium, K+ = Potassium, PO4- = Phosphates, Mg+ = Magnesium; *significant p-value <0.05.

Table 2 Comparison of preoperative parameters between groups 1 and 2.

Preoperative parameters		Group 1 (n = 32)	Group 2 (n = 21)	p- value
Age in months	Median (IQR)	10 (17.5)	11 (12)	0.948
Gender (n)	Male	25	16	1.0
	Female	7	5	
Weight (kg)	Median (IQR)	9 (3.5)	8 (4.5)	0.488
Operated kidney (n)	Right	6	5	0.37
	Left	26	16	
Parenchymal thickness (mm)	Median (IQR)	2.6 (1)	4.7 (3)	0.008*
Differential size (cm)	Median (IQR)	2.24 (2.05)	2 (2.75)	0.084
Differential ratio	Median (IQR)	1.7 (0.55)	1.1 (0.3)	0.007*
Renal pelvis APD (mm)	Median (IQR)	35 (17)	21 (6.5)	< 0.001*
SRF of affected side (%)	Median (IQR)	34 (15.5)	31 (19)	0.749
SFU grade	Grade 3	5	18	< 0.001*
	Grade 4	27	3	
GFR of the affected side (ml/min/1.73 m ²)	Median (IQR)	24 (21)	28 (19.5)	0.914

Table note: Abbreviations used in the table - IQR = Interquartile range, APD = anteroposterior diameter, SRF = Split renal function (on Dynamic renal scintigraphy), SFU = Society of fetal urology, GFR = Glomerular filtration rate; *significant p-value <0.05.

Table 3 Comparison of daily total urine output between the 2 groups.

Total urine output	Group 1 (n = 32)	Group 2 (n = 21)	p- value
Day 1	5.53 ± 1.02	3.3 ± 0.75	0.002*
Day 2	4.1 ± 0.86	3.29 ± 0.53	0.025*
Day 3	3.52 ± 0.81	2.48 ± 0.72	0.088
Day 4	2.52 ± 0.68	2.27 ± 0.56	0.475
Day 5	1.89 ± 0.52	1.65 ± 0.38	0.142

Table note: *significant p-value <0.05.

electrolyte derangement and prolonged POD during post-operative period (onset on postoperative day-1 in three and postoperative day-2 in one). These four patients, despite normal appearing NCK on preoperative imaging, required treatment by nephrologist and POD resolved by day-7 postoperatively; one patient developed postoperative pyelonephritis in NCK confirmed on USG and the other three had past history of treatment for glomerulonephritis, nephrotic syndrome and non-dilating contralateral vesicoureteric reflux with no cortical scar on renal scan, respectively. Three of these four patients had severe parenchymal thinning in OK (PT < 2 mm).

Discussion

POD following unilateral obstruction may be due to pressure atrophy of tissue nearby to the collecting duct system resulting in more tubular damage than glomerular damage at the level of proximal (PCT) and distal convoluted tubules (DCT) resulting in UCA, natriuresis, defect in urinary acidification, and deranged transport of other cations [18]. Predisposing factors in OK noted by us were parenchymal thinning, SFU grade-4 hydronephrosis, higher differential ratio and higher renal APD, similar to observations by Roth et al. [11]. PT was the most significant associated factor on regression analysis reflecting the high pressure related functional compromise of nephrons in OK. This is usually compensated by the unaffected nephrons in NCK. Interestingly, we observed electrolyte derangement and prolonged POD in four patients with either concomitant or prior subclinical injury to NCK which otherwise appeared normal on preoperative imaging. The clinical implication of these observations will be a *warning to the clinician for close monitoring of such patients whose current/prior history suggests injury to the nephrons of NCK in addition to the OK.*

UO from OK mainly contributes to the volume of POD. In our study, UO from the OK was significantly higher than from the NCK on all 5 days of postoperative assessment with

Table 4 Comparison between urine output of nephrostomy & PUC in group 1 and group 2.

Urine output (mL/kg/hour)	Group 1		p- value	Group 2		p- value
	NT	PUC		NT	PUC	
Day 1	3.41 ± 1.72	2.23 ± 1.18	0.003*	2.29 ± 0.36	1.48 ± 0.40	0.001*
Day 2	2.73 ± 1.04	1.37 ± 0.49	< 0.001*	2.02 ± 0.56	1.28 ± 0.33	0.002*
Day 3	2.52 ± 1.12	1.2 ± 0.63	< 0.001*	1.64 ± 0.50	0.83 ± 0.30	< 0.001*
Day 4	1.70 ± 0.63	0.80 ± 0.28	< 0.001*	1.40 ± 0.48	0.80 ± 0.19	0.001*
Day 5	1.35 ± 0.49	0.60 ± 0.21	< 0.001*	1.06 ± 0.39	0.60 ± 0.10	< 0.001*

Table note: Abbreviations used in the table - PUC = per urethral catheter; *significant p-value <0.05.

a declining trend. In prospective studies, Murer et al. [8] and Li et al. [9] measured the differential UO from OK and NCK by NT drainage and PUC respectively while two other studies recorded the NCK output by measuring voided volume [7,18]. Murer et al. [8] and Li et al. [9] reported similar findings over a postoperative follow-up of 5-days and 2-days respectively. Two other retrospective studies do not mention clearly, the data regarding the differential UO from OK & NCK [10,11]. The UO from OK was >4 or >5 times that of the NCK in a case report of 3 newborns [6].

Biochemical analysis was mostly comparable with available literature. Urine from OK had significantly lower SG as compared to that from NCK matching other studies [6,8,9,18,19]. Better et al. [19] reported urinary osmolality of 265 mOsm/kg water from the NT against 766 mOsm/kg water from the NCK following relief of complete urinary obstruction. The difference in UO & SG was more significant between the two groups compared to other biochemical parameters (Supplementary Table-2). Thus, *impaired UCA in the OK after relief of obstruction contributes more to the POD than natriuresis*. Hypotonic urine with reduced urinary exosomal AQP1 excretion results from selective down regulation of AQP1 and AQP2 [8,9,20]. Higher pH of the urine from OK in our study matches another study [18]; this occurs in UUO due to defect in urinary acidification caused by H^+ -ATPase down-regulation mediated by an increase in inducible Nitrous oxide synthetase (iNOS) regulated by Angiotensin 2 [21].

Increased $FeNa^+$ excretion in urine from the OK has been explained by various mechanisms: decreased Na^+ reabsorption due to defective sodium transport in DCT [20,21], Angiotensin II mediated decrease in aquaporin channels and Na^+ transporters in PCT of experimental rats with UUO [22–24], inhibition of $NaCl$ absorption by PGE2 in the thick ascending limb of the loop of Henle and vasopressin induced increased permeability of water in the collecting ducts [25,26]. Similar to previous reports [19], the $FeMg^+$ was significantly higher from the OK compared to NCK. It has been linked to excessive natriuresis as calcium and magnesium resorption goes hand in hand with sodium resorption [27,28]. The FeK^+ excretion was significantly higher from the OK similar to Jones et al. [29], but contrary to the observations in experimental animals [13–15] which reported decreased potassium excretion in proportion to GFR. *These contradictory translational findings merit further research*. In contrast to increased fractional excretion of cations, urinary $FePO_4^-$ was significantly lower in UO from OK. This is *peculiar to UUO* in contrast to bilateral obstruction [18,30]. After release of UUO in humans, phosphate excretion from the OK is consistently and markedly on the lower side and out of proportion to reduction in the filtered load [30]. $CrCl$ from OK improved gradually over 5-days following relief of obstruction, similar to previous studies [6,19]. A corresponding inverse change in the biochemical parameters of UO from NCK indirectly suggests existence of compensatory mechanism during POD. *Significantly low $CrCl$ and SG from OK in Group-1 even on day-5 compared to other parameters (Supplementary Table-2) is consistent with delayed recovery of glomerular function compared to the tubular function*.

In our study, POD resolved by postoperative day-5 in 28 Group-1 patients. Roth et al. reported the median

resolution time for POD to be 3-days [11]. Hydration and electrolyte balance was maintained in Group-1 patients with ORS *except in four patients (14.3%) with electrolyte derangement who needed nephrology consultation as described in the results*. A correspondingly more deranged urine biochemistry was noted in these four patients extending beyond day-5. Similar observations were made by Roth et al. in 4/7 (57%) children and Boone et al. in 3 newborns [6]. Due to very small numbers comparing these four patients with others will be more speculative. *At centres practicing early discharge, parents must be asked to monitor UO with diaper weight and if >4 ml/kg/hr, serum electrolytes should be monitored on OPD basis every 48-hrs till POD resolves usually by day-5*. Interestingly, even in patients without POD (Group 2), NT urine output was significantly higher than PUC, *suggesting a pre-diuretic state* following relief of obstruction but the severity varies. In a subset of patients with POD for >48 -hrs and SFU grade-4 hydronephrosis, NT had to be retained for a longer period.

The incidence of POD in our study was 63%. A lower incidence reported in the retrospective studies by Bermeo et al. (30%) and Roth et al. (1.8%) is due to a higher and arbitrary (without any reference) cut-off criteria for POD equating to 5 ml/kg/hr and 6 ml/kg/hr respectively [10,11]. We defined POD as total UO >4 ml/kg/hr deriving from more recent literature [16,17].

Our is the first and unique prospective study with the largest sample size observing the translational findings of animal studies for UUO in human pediatric patients under true clinical setting. Additionally, the prospective nature of our study with recent age-specific definition of polyuria provides more realistic data. But there were limitations too in our study. First, a wide range for definition of polyuria in children (>2.5 – 6 ml/kg/hr) in the literature confounds the interpretation. Our definition of 4 ml/kg/hr although in accordance with recent literature [16,17] appears broad explaining higher incidence noted by us compared to two other similar studies [10,11]. Second confounding factor also discussed in other studies [10,11] is the assumption that PUC reflects urine only from NCK. It is possible for urine to trickle alongside the trans-anastomotic ureteral stent into the bladder adding to the volume draining through PUC, but we believe this amount would be insignificant due to the occlusion of anastomotic lumen by postoperative edema around the trans-anastomotic stent during early postoperative period.

Conclusions

Following pyeloplasty for unilateral PUJO in children, high UO from OK mainly contributes to POD and the deranged urine biochemistry largely matches with the animal studies. Postoperative high UO from NT and anastomotic edema may result in obstructed drainage manifesting as abdominal pain \pm lump on clamping of NT. Parenchymal thinning in larger hydronephrotic kidneys predisposes to POD. *At centres practicing early discharge, parents of patients with predisposing factors for POD must be instructed to monitor UO with diaper weight and if >4 ml/kg/hr, serum electrolytes should be monitored on OPD basis every 48-hrs till POD resolves, to detect subclinical electrolyte*

derangement early. Compensation by NCK maintains homeostasis, but any evidence of pre/co-existing functional injury to NCK while eliciting past history or during the postoperative period, respectively, should warn the clinician regarding prolonged course of POD and *more careful monitoring* for electrolyte derangement.

Future research may be focussed on: developing consensus guidelines on defining age-specific definition for POD in children, studying higher FeK⁺ excretion noted in human pediatric subjects contrary to animal studies and studying POD in kidneys with unilateral PUJO small in size and/or poorly functioning (SRF<10%).

Ethical clearance

Obtained.

Funding

The authors report no financial support/funding for this work.

Conflicts of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Acknowledgements

None.

References

- Howards SS. Post-obstructive diuresis: a misunderstood phenomenon. *J Urol* 1973;110:537–540. [https://doi.org/10.1016/S0022-5347\(17\)60273-8](https://doi.org/10.1016/S0022-5347(17)60273-8).
- Loo MH, Vaughan Jr ED. Obstructive nephropathy and post-obstructive diuresis. *AUA Update Ser* 1985;4:1–7.
- Vaughan Jr ED. Recognition and management of post-obstructive diuresis. *Weekly Urol Update Ser* 1978;1:18.
- Halbgewachs C, Domes T. Postobstructive diuresis. Pay close attention to urinary retention. *Can Fam Physician* 2015;61(2):137–142.
- Singh I, Strandhoy JW, Asimos DG. Pathophysiology of urinary tract obstruction. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. *Campbell-Walsh urology*. 10th ed. Philadelphia, PA: Elsevier Saunders; 2012. p. 1107–1108.
- Boone TB, Allen TD. Unilateral post-obstructive diuresis in the neonate. *J Urol* 1992;147:430–432. [https://doi.org/10.1016/S0022-5347\(17\)37258-0](https://doi.org/10.1016/S0022-5347(17)37258-0).
- Schlossberg SM, Vaughan Jr ED. The mechanism of unilateral post-obstructive diuresis. *J Urol* 1984;131(3):534–536. [https://doi.org/10.1016/S0022-5347\(17\)50485-1](https://doi.org/10.1016/S0022-5347(17)50485-1).
- Murer L, Addabbo F, Carosino M, Procino G, Tamma G, Montini G, et al. Selective decrease in urinary aquaporin 2 and increase in prostaglandin E2 excretion is associated with postobstructive polyuria in human congenital hydronephrosis. *J Am Soc Nephrol* 2004;15(10):2705–2712. <https://doi.org/10.1097/01.ASN.0000139689.94776.7A>.
- Li ZZ, Zhao ZZ, Wen JG, Xing L, Zhang H, Zhang Y. Early alteration of urinary exosomal aquaporin 1 and transforming growth factor β 1 after release of unilateral pelviureteral junction obstruction. *J Pediatr Surg* 2012;47(8):1581–1586. <https://doi.org/10.1016/j.jpedsurg.2011.12.024>.
- Bermeo AMP, Zableh AMO, Castillo M, Niño JFP. Risk factors for post-obstructive diuresis in pediatric patients with ureteropelvic junction obstruction, following open pyeloplasty in three high complexity institutions. *J Pediatr Urol* 2018;14(3):260.e1–260.e4. <https://doi.org/10.1016/j.jpuro.2018.01.017>.
- Roth JD, Lesier JD, Casey JT, Szymanski KM, Whittam BM, Misseri R, et al. Incidence of pathologic postobstructive diuresis after resolution of ureteropelvic junction obstruction with a normal contralateral kidney. *J Pediatr Urol* 2018;14(6):557.e1–557.e6. <https://doi.org/10.1016/j.jpuro.2018.07.012>.
- Harris RH, Yarger WE. The pathogenesis of post-obstructive diuresis: the role of circulating natriuretic and diuretic factors, including urea. *J Clin Invest* 1975;56:880–887.
- Thirakomen K, Kozlov N, Arruda JAL, Kurtzmann NA. Renal hydrogen ion secretion after release of unilateral ureteral obstruction. *Am J Physiol* 1976;231:1233–1239.
- Hanley MJ, Davidson K. Isolated nephron segments from rabbit models of obstructive nephropathy. *J Clin Invest* 1982;69:165–174.
- Bander SJ, Buerkert JE, Martin D, Klahr S. Long-term effects of 24-hr unilateral ureteral obstruction on renal function in the rat. *Kidney Int* 1985;28(4):614–620. <https://doi.org/10.1038/ki.1985.173>.
- The Royal Children's Hospital, Melbourne, Australia. Clinical practice guideline on diabetes insipidus [Internet, last updated 2022 May; cited 2024 Nov 15]. Available from: https://www.rch.org.au/clinicalguide/guideline_index/diabetes_insipidus/.
- Mishra G, Chandrashekhar SR. Management of diabetes insipidus in children. *Indian J Endocrinol Metabol* 2011;(Suppl 3):S180–S187. <https://doi.org/10.4103/2230-8210.84858>.
- Gillenwater JY, Westervelt Jr FB, Vaughan ED Jr, Howards SS. Renal function after release of chronic unilateral hydronephrosis in man. *Kidney Int* 1975;7(3):179–186. <https://doi.org/10.1038/ki.1975.26>.
- Better OS, Arieff AI, Massry SC, Kleeman CR, Maxwell MH. Studies on renal function after relief of complete unilateral ureteral obstruction of three months duration in man. *Am J Med* 1973;54:234–240. [https://doi.org/10.1016/0002-9343\(73\)90228-3](https://doi.org/10.1016/0002-9343(73)90228-3).
- Frøkiaer J, Christensen BM, Marples D, Djurhuus JC, Jensen UB, Knepper MA, et al. Downregulation of aquaporin-2 parallels changes in renal water excretion in unilateral ureteral obstruction. *Am J Physiol* 1997;273(2 Pt 2):F213–F223. <https://doi.org/10.1152/ajprenal.1997.273.2.F213>.
- Valles PG, Manucha WA. H⁺-ATPase activity on unilateral ureteral obstruction: interaction of endogenous nitric oxide and angiotensin II. *Kidney Int* 2000;58(4):1641–1651. <https://doi.org/10.1046/j.1523-1755.2000.00325.x>.
- Li C, Wang W, Knepper MA, Nielsen S, Frøkiaer J. Downregulation of renal aquaporins in response to unilateral ureteral obstruction. *Am J Physiol Ren Physiol* 2003;284(5):F1066–F1079. <https://doi.org/10.1152/ajprenal.00090.2002>.
- Jensen AM, Li C, Praetorius HA, Nørregaard N, Frische S, Knepper MA. Angiotensin II mediates downregulation of aquaporin water channels and key renal sodium transporters in response to urinary tract obstruction. *Am J Physiol Ren Physiol* 2006;291(5):F1021–F1032. <https://doi.org/10.1152/ajprenal.00387.2005>.
- Shi Y, Li C, Thomsen K, Jorgensen TM, Knepper MA, Nielsen S, et al. Neonatal ureteral obstruction alters expression of renal sodium transporters and aquaporin water channels. *Kidney Int* 2004;66(1):203–215. <https://doi.org/10.1111/j.1523-1755.2004.00721.x>.

- [25] Aarab L, Siaume-Perez S, Chabardès D. Cell-specific coupling of PGE₂ to different transduction pathways in arginine vasopressin- and glucagon-sensitive segments of the rat renal tubule. *Br J Pharmacol* 1999;126(4):1041–1049. <https://doi.org/10.1038/sj.bjpp.0702390>.
- [26] Torikai S, Kurokawa K. Effects of PGE₂ on vasopressin-dependent cell cAMP in isolated single nephron segments. *Am J Physiol* 1983;245(1):F58–F66. <https://doi.org/10.1152/ajprenal.1983.245.1.F58>.
- [27] Duarte CG, Watson JF. Calcium reabsorption in proximal tubule of the dog nephron. *Am J Physiol* 1967;212:1355–1360. <https://doi.org/10.1152/ajplegacy.1967.212.6.13>.
- [28] Falls FWJr, Stacey WK. Post obstructive diuresis. Studies in a dialysed patient with a solitary kidney. *Am J Med* 1973;54:404–412. [https://doi.org/10.1016/0002-9343\(73\)90035-1](https://doi.org/10.1016/0002-9343(73)90035-1).
- [29] Jones BF, Nanra RS. Post- obstructive diuresis. *Aust N Z J Med* 1983;13(5):519–521. <https://doi.org/10.1111/j.1445-5994.1983.tb02708.x>.
- [30] Better OS, Tuma S, Kedar S, Chamimowitz C. Enhanced tubular reabsorption of phosphate: following relief of unilateral ureteral obstruction in man. *Arch Intern Med* 1975;135(2):245–248. <https://doi.org/10.1001/archinte.1975.00330020049006.1>.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jpurol.2026.105878>.