



## นิพนธ์ต้นฉบับ

# การศึกษาความชุกและปัจจัยที่มีความสัมพันธ์กับการเกิดภาวะ post-obstructive diuresis หลังการทำการใส่สายสวนผ่านทางผิวหนังเข้าไปในรายได้ ในผู้ป่วยที่มีการอุดตันของระบบปัสสาวะและส่งผลเสียต่อการทำงานของไต

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### คำสำคัญ:

post-obstructive  
diuresis, การอุดตัน  
ของระบบปัสสาวะ,  
ปัจจัยเสี่ยง

### บทคัดย่อ

**วัตถุประสงค์:** เพื่อศึกษาความชุกและปัจจัยเสี่ยงในการเกิดภาวะ post-obstructive diuresis ภายหลังการทำการใส่สายสวนผ่านทางผิวหนังเข้าไปในรายได้ ในผู้ป่วยที่มีการอุดตันของระบบปัสสาวะและส่งผลต่อการทำงานของไต

**ผู้ป่วยและวิธีการศึกษา:** ศึกษาระหว่างเดือนตุลาคม พ.ศ. 2554 ถึงเดือนพฤษจิกายน พ.ศ. 2557 ในผู้ป่วยที่ได้รับการวินิจฉัยว่ามีการอุดตันของระบบปัสสาวะ จากการตรวจด้วยคลีน เสียงความถี่สูง หรือเอกซเรย์คอมพิวเตอร์ในช่องท้อง แล้วพบว่ามีการภาวะไตบวมน้ำ ร่วมกับ มีการทำงานของไตที่ผิดปกติจากการเจาะเลือด และผู้ป่วยได้รับการรักษาด้วยการใส่สายสวนผ่านทางผิวหนังเข้าไปในรายได้ที่โรงพยาบาลราชวิถี โดยมีการเก็บข้อมูล ได้แก่ ลักษณะ ทั่วไปของผู้ป่วย ลักษณะรูป่างของไต อาการและอาการแสดงของผู้ป่วย รวมถึงผลทางห้องปฏิบัติการก่อนเริ่มการรักษา จากนั้นนำปัจจัยดังกล่าวมาวิเคราะห์เพื่อหาปัจจัยเสี่ยงของการเกิดภาวะ post-obstructive diuresis

**ผลการศึกษา:** ผู้ป่วยที่มีการอุดตันของระบบทางเดินปัสสาวะ 309 คน มีผู้ป่วยที่เข้าเกณฑ์การคัดเลือกจำนวน 209 คน และมีผู้ป่วยที่มีข้อมูลครบถ้วนที่ใช้ในการศึกษา 142 คน จากการศึกษาพบความชุกในการเกิดภาวะ post-obstructive diuresis 61 คน (ร้อยละ 43) และปัจจัยเสี่ยงในการเกิดภาวะ post-obstructive diuresis ได้แก่ ผู้ป่วยที่มีไตช้ำงเดียว (OR 6.13, 95% CI 1.48-25.32, p=0.012) และ ผู้ป่วยที่มีค่า glomerular filtration rate (GFR) ต่ำ (OR 0.94, 95% CI 0.90-0.98, p=0.005)

**สรุป:** ความชุกในการเกิดภาวะ post-obstructive diuresis คิดเป็นร้อยละ 43 และปัจจัยเสี่ยงในการเกิดภาวะ post-obstructive diuresis ได้แก่ ผู้ป่วยที่มีไตช้ำงเดียว และผู้ป่วยที่มีค่า GFR ต่ำ อย่างไรก็ตาม ควรมีการศึกษาเพิ่มเติมในกลุ่มประชากรที่มีขนาดใหญ่ต่อไป



## Original article

## Prevalence and risk factors of post obstructive diuresis after percutaneous nephrostomy in obstructive nephropathy patients

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**Keyword:**

post obstructive diuresis, obstructive uropathy, risk factor

**Abstract**

**Objective:** To identify the prevalence and risk factors of post-obstructive diuresis after percutaneous nephrostomy in obstructive nephropathy patients who have functional or anatomic renal damage from urinary tract obstruction.

**Material and methods:** Between October 2011 and November 2014, obstructive nephropathy patients who had hydronephrosis, diagnosed by ultrasound or CT scan, acute kidney injury from blood test and underwent percutaneous nephrostomy at Rajavithi Hospital were enrolled in this retrospective study. Patient demographics, kidney characteristics, clinical and laboratory values were collected at the time of admission and analyzed for risk factors of post obstructive diuresis using univariate and multivariate analysis.

**Results:** Of 305 patients with obstructive uropathy, 229 patients were eligible for inclusion based on our eligibility criteria. Of 229 patients included, 142 had complete data available. Post obstructive diuresis occurred in 61 of the 142 (43%) patients. Single kidney (OR 6.13, 95% CI 1.48-25.32,  $p=0.012$ ) and low glomerular filtration rate (GFR) (OR 0.94, 95% CI 0.90-0.98,  $p=0.005$ ) independently predicted post obstructive diuresis after multivariate analysis.

**Conclusion:** Prevalence of post obstructive diuresis was about 43%. Our study demonstrated that single kidney and low GFR are risk factors for post obstructive diuresis. However, a large prospective study is required to further investigate these findings.

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## Introduction

Acute kidney injury (AKI) is a major problem in Thailand and around the world. However, the worldwide incidence of acute kidney injury is unclear because of under reporting, regional disparities, and differences in definition and case mix. Recent studies in the United States and Spain have shown incidences varying between an average of 23.8 cases per 1000 with an 11% yearly increase between 1992 and 2001<sup>1</sup>. In all, 5-10% of AKI episodes are classified as post-renal (PR-AKI), i.e., caused by urinary tract obstruction<sup>2,3</sup>.

The pathophysiology of obstructive nephropathy has been extensively studied in animal models for ureteric obstruction<sup>4-7</sup>, but clinical studies on PR-AKI are scarce. The few existing studies are outdated, and/or have focused on the mechanisms of 'post-obstructive diuresis' (POD), a potentially life-threatening polyuria that can develop after the release of the obstruction in various ways, such as percutaneous nephrostomy (PCN), double-J stent or Foley catheter insertion. POD can occur especially in patients who have bilateral ureteric obstruction or unilateral ureteric obstruction in a solitary functioning kidney<sup>5,11,12</sup>.

The definition of POD varies. Vaughan et al<sup>8</sup> defined it as urine output greater than 200 ml per hour for at least 2 hours. Bishop<sup>9</sup> defined it as urine output of between 125 and 200 ml per hour. Oster et al<sup>10</sup>, defined it as urine output of more than 4 liters per day.

The clinical presentation does not predict which patients will have POD. No correlation has been found between the severity or duration of POD and the plasma urea value before decompression, electrolyte value, creatinine clearance, bladder pressure, or blood pressure<sup>13</sup>. Vaughan and Gillenwater<sup>8</sup> studied 22 patients who had POD and found that the patients with the greatest risk of significant POD had fluid overload, severe renal impairment, or central nervous system manifestations. Loo and Vaughan found that patients who are susceptible to POD typically have signs of fluid overload, including edema, congestive

heart failure, or hypertension.

Some clinically relevant questions remain unanswered. Clinically relevant questions are still unanswered. For example, can we predict POD occurrence, and thus select those patients who should undergo careful monitoring of volume and electrolytic status. Therefore, we conducted this retrospective study in order to identify the prevalence and predictors of POD from clinical tests taken at the time of patient admission.

## Material and methods

This study was conducted from October 2011 to November 2014 with a retrospective design. Obstructive nephropathy patients who were admitted and underwent percutaneous nephrostomy at Rajavithi Hospital during that time were enrolled in this study. We excluded patients with the following conditions: no AKI, coagulopathy or bleeding diathesis, incomplete information, and those whose urinary tract obstruction were relieved in other ways (e.g., double-J stent or Foley catheter). The study was approved by the Ethics and Research Committee of Rajavithi Hospital.

The diagnosis of obstructive nephropathy (functional or anatomic renal damage from urinary tract obstruction) was based on the following criteria: (i) dilatation of the renal calyces on ultrasonographic examination or computed tomography (CT) scan and (ii) blood test before treatment showed AKI, defined as an increase in serum creatinine by 1.5 times above baseline/above normal value if unknown and an estimated glomerular filtration rate (eGFR) that was less than 60 mL/min/1.73 m<sup>2</sup> [calculated by Cockcroft-Gault formula: (140-age) x (weight in kg) x (0.85 if female)/ (72 x creatinine)]

Percutaneous nephrostomy was conducted by 2<sup>nd</sup> to 4<sup>th</sup> year urologic residents at the Division of Urology, Department of Surgery, Rajavithi Hospital. The patient was placed in the prone position. After the patient was adequately cleaned and draped using sterile methods, the puncture site was infiltrated



with an acceptable local anesthesia, such as 1% xylocaine. A needle was used to puncture the skin by ultrasonic guidance. Spontaneous urine drainage was particularly seen in an obstructed system. If urine was not spontaneously draining, it was aspirated from the needle instead. Renal entry can be further confirmed by administration of contrast medium into the collecting system via the needle. A guide wire was passed through the needle to enter the renal pelvis. Over the guide wire, the tract was dilated with a fascial dilator No.8-14 Fr. The nephrostomy catheter No.12 Fr was then inserted over the guide wire. Its position was further confirmed by administration of contrast to opacify the collecting system via the tube. The catheter was secured externally with suture.

After percutaneous nephrostomy, urine output was monitored hourly to check for the occurrence of POD. In this study, POD was defined as urine output of more than 200 ml/hr. If POD occurred, urinary losses were only partially compensated. No fixed rule for compensation was applied.

The following data were collected from medical charts and OPD cards at the time of admission.

- General demographic characteristics: age, gender, weight, height, body mass index (BMI) [weight (kg)/height (m<sup>2</sup>)], underlying disease, and cause of urinary tract obstruction.
- Kidney characteristics: number of kidneys (single or both), number of hydronephrosis (unilateral or bilateral), degree of hydronephrosis (mild, moderate, severe), and cortical thickness.

A single kidney was defined as one kidney or one working kidney for one of three main reasons: the patient was born with only one, the patient lost one to disease or injury, or the patient donated a kidney to a family member or friend who had lost one of their own.

Hydronephrosis was defined as mild, moderate, or severe according to standard definitions. Mild hydronephrosis was defined as enlargement of the calices with preservation of the renal papillae. Moderate hydronephrosis was defined as rounding of the calices with obliteration of the renal papillae. Severe hydronephrosis was defined as caliceal ballooning with cortical thinning<sup>14</sup>.

The renal cortical thickness was measured in the sagittal plane at the level of the mid kidney following the process described by Moghazi et al<sup>15</sup>. The measurement was taken over a medullary pyramid, perpendicular to the capsule at the shortest distance from the base of the medullary pyramid to the renal capsule.

- Clinical values: oliguria, anuria, congestive heart failure (CHF), edema, uremia, systolic and diastolic blood pressure.
- Laboratory values: hemoglobin (Hb), blood urea nitrogen (BUN), creatinine (Cr), estimated glomerular filtration rate (eGFR), sodium (Na), potassium (K), chloride (Cl), bicarbonate (HCO<sub>3</sub>), calcium (Ca), magnesium (Mg), and phosphate (PO<sub>4</sub>).

Data are shown as percentage or mean/median (range) values. Predictors of POD occurrence were identified using univariate analysis, followed by multivariate analysis.

The statistical analysis software was Statistical Package for Social Sciences (SPSS) version 17.0. Descriptive statistics are shown as the number, percentage, mean, median, minimal and maximal value. In terms of inferential statistics, quantitative information comparisons used the Student t-test with normal distribution data and the Mann-Whitney U test with non-normal distribution data. Qualitative information comparisons used the Chi-square test. A p-value of less than 0.05 was considered to indicate statistical significance.

## Results

During the study period, 305 patients had obstructive uropathy, defined as the functional or anatomic obstruction of urinary flow at any level of the urinary tract. In all, 229 patients were eligible for inclusion based on our eligibility criteria; however, 87 of these patients had incomplete information. Therefore, 142 patients had complete data available. (Fig.1)

Patients' characteristics are shown in Table 1. The mean age was 56 years (range: 19-85 years). Female patients were the majority, accounting for 61%. In terms of kidney characteristics, two kidneys (90.8%) were more common than a single kidney (9.2%). These patients showed bilateral hydronephrosis (88%) with moderate degree (66.9%) and a cortical thickness of

about 10.97 mm. (range 4-18 mm.). Most of the cases presented with edema (29.6%), oliguria (25.4%), CHF (22.5%), anuria (12.7%) and uremia (9.2%).

The causes of urinary tract obstruction are shown in Table 2. Cancer was the most common cause of obstruction (90%). The most frequent type of cancer was cervical cancer (46.1%), followed by bladder cancer (19.5%), prostate cancer (9.4%), colon cancer (7.8%), ovarian cancer (3.9%), GIST (2.3%), endometrial cancer (1.6%), and lymphoma (1.6%). BPH was the cause of obstruction in about 2.8% of cases. Stones accounted for about 4.8%. Miscellaneous causes of obstruction included TB KUB (2), endometriosis (1), and ureteroenteric stricture (1).

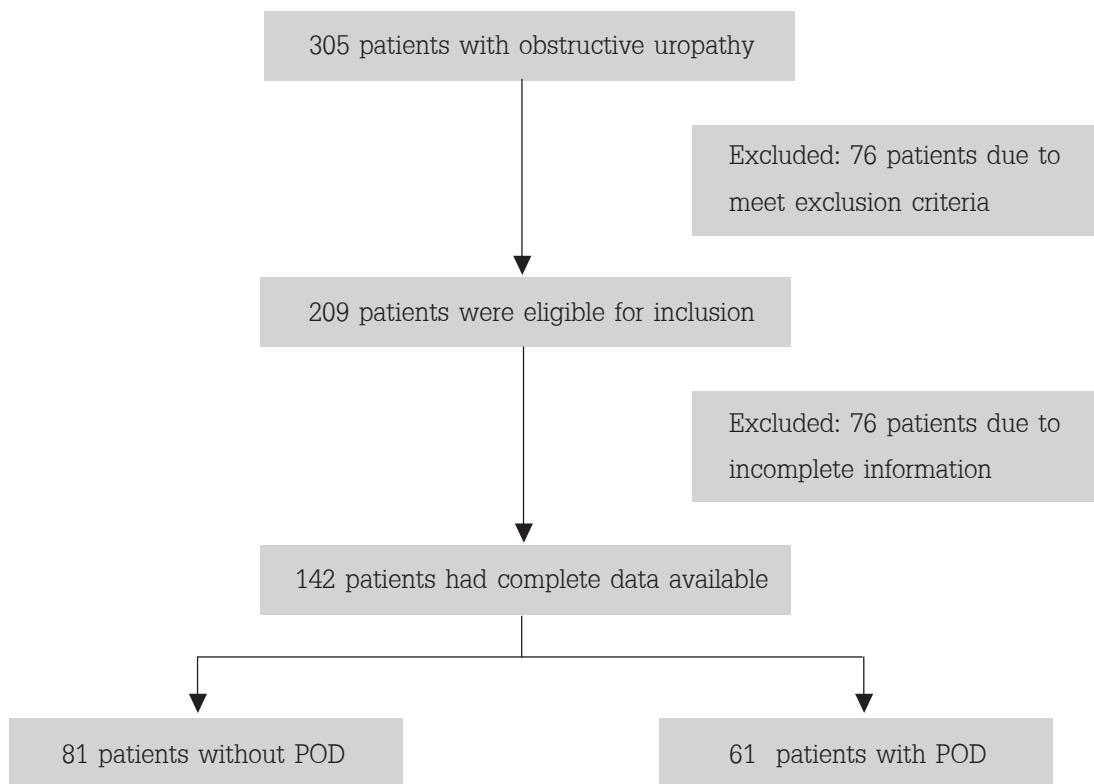


Figure 1 Flow chart of patients

**Table 1** The patients' characteristics

Characteristics	Mean/Median (range) or N (%) value
<b>Demographic data</b>	
Age, years	56.42 (19-85)
Gender male/female	55 (38.7%)/87 (61.3%)
Weight, kg	54.42 (25-100)
Height, cm	158.22 (135-180)
BMI, kg/m <sup>2</sup>	21.66 (11.57-34.96)
Underlying disease	86 (60.6%)
HT	61 (43%)
DM	19 (13.4%)
<b>Kidney characteristics</b>	
Kidney single/both	13 (9.2%)/129 (90.8%)
Hydronephrosis unilateral/bilateral	17 (12%)/125 (88%)
Degree mild/moderate/severe	33 (23.2%)/95 (66.9%)/14 (9.9%)
Cortical thickness, mm	10.97 (4-18)
<b>Signs and symptoms</b>	
Oliguria	36 (25.4%)
Anuria	18 (12.7%)
Congestive heart failure	32 (22.5%)
Edema	42 (29.6%)
Uremia	13 (9.2%)
SBP, mmHg	141.63 (90-210)
DBP, mmHg	80.38 (54-120)
<b>LAB</b>	
Hb, g/dL	8.73 (3.8-13.8)
BUN, mg/dL	57.5 (11-232)
Cr., mg/dL	6.5 (1.5-55.4)
GFR, ml/min/1.73m <sup>2</sup>	8.14 (1.24-45.38)
Na, mEq/L	131.89 (111-146)
K, mEq/L	4.75 (2.2-7.9)
Cl, mEq/L	98.33 (74-117)
HCO <sup>3</sup> , mEq/L	18.24 (5-35)
Ca, mg/dL	8.72 (5.4-14.1)
Mg, mg/dL	2.13 (1.0-4.2)
PO <sub>4</sub> , mg/dL	6.1 (2.6-16.8)



Table 2 Causes of urinary tract obstruction

Cause	Men (n=55)	female (n=87)	Total (n=142)
<b>Cancer</b>	<b>45 (81.82%)</b>	<b>83 (95.4%)</b>	<b>128 (90.14%)</b>
Cervix	0	59	59
Bladder	20	5	25
Prostate	12	0	12
Colon	6	4	10
Ovary	0	5	5
GIST	2	1	3
Endometrium	0	2	2
Lymphoma	1	1	2
Other cancer	4	6	10
<b>BPH</b>	<b>4 (7.27%)</b>	<b>0</b>	<b>4 (2.82%)</b>
<b>Stone</b>	<b>5 (9.09%)</b>	<b>1 (1.15%)</b>	<b>6 (4.22%)</b>
<b>Miscellaneous</b>	<b>1 (1.82%)</b>	<b>3 (3.45%)</b>	<b>4 (2.82%)</b>

**Other cancers:** pelvic mass (3), Ewing sarcoma (1), pancreatic cancer (1), penile cancer (1), cholangiocarcinoma (1), neuroendocrine tumor (1), urethral cancer (1), germ cell tumor (1)

**Miscellaneous:** TB KUB (2), Endometriosis (1), ureteroenteric stricture (1)

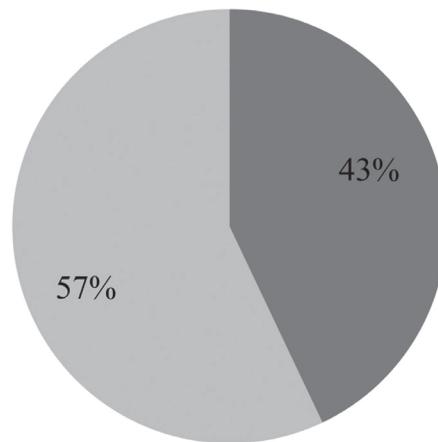
Overall, 142 patients were analyzed to access POD incidence and identify risk factors of POD occurrence. POD occurred in 61 (43%) cases (see Fig.2). It occurred for approximately 4 days (range 1-15 days). Diuresis during the first 24 hours was

about 8,300 ml (range: 4,200-23,620 ml) and fluid balance was negative.

Risk factors of POD occurrence with univariate analysis are shown in Table 3. The factors were general demographic characteristics, kidney characteristics, clinical values, and laboratory values. A single kidney was significant compared with both kidneys ( $p=0.009$ ). BUN was significantly higher (67 vs 52 mg/dL;  $p=0.011$ ), creatinine was significantly higher (8.86 vs 4.2 mg/dL;  $p<0.001$ ) and GFR was significantly lower (6.49 vs 10.46 ml/min/1.73 m<sup>2</sup>,  $p=0.001$ ) when comparing the POD group with the no POD group.

## Prevalence

■ POD ■ No POD



**Figure 2** Prevalence of post obstructive diuresis

**Table 3** Risk factors of POD: Results of the univariate analysis

Factors	Patients with no POD	Patients with POD	p-value
<b>N</b>	81 (57%)	61 (43%)	
<b>Sex, n (%)</b>			0.409
Male	29 (35.8%)	26 (42.6%)	
Female	52 (64.2%)	35 (57.4%)	
<b>Character, mean(range)</b>			
age, years	58.79 (19-86)	53.28 (21-84)	0.701
weight, kg	53.25 (25-100)	55.98 (35-85)	0.994
height, cm	157.78 (135-180)	158.8 (145-177)	0.809
BMI, kg/m <sup>2</sup>	21.28 (11.57-34.96)	22.17 (15.85-31.93)	0.367
<b>Underlying disease, n (%)</b>			
HT	47 (58%)	39 (63.9%)	0.476
DM	33 (40.7%)	28 (45.9%)	0.539
	10 (12.3%)	9 (14.8%)	0.676



Table 3 Risk factors of POD: Results of the univariate analysis (continue)

Factors	Patients with no POD	Patients with POD	p-value
<b>Kidney, n (%)</b>			0.009*
Both	78 (96.3%)	51 (83.6%)	
Single	3 (3.7%)	10 (16.4%)	
<b>Mean (range) cortical thickness, mm</b>	10.8 (4-17)	11.19 (5-18)	0.625
<b>Hydronephrosis, n (%)</b>			0.054
Bilateral	75 (92.6%)	50 (82%)	
Unilateral	6 (7.4%)	11 (18%)	
<b>Degree, n (%)</b>			0.833
Mild	19 (23.5%)	14 (23%)	
Moderate	53 (65.4%)	42 (68.9%)	
Severe	9 (11.1%)	5 (8.2%)	
<b>Oliguria, n (%)</b>	23 (28.4%)	13 (21.3%)	0.337
<b>Anuria, n (%)</b>	7 (8.6%)	11 (18%)	0.096
<b>CHF, n (%)</b>	16 (19.8%)	16 (26.2%)	0.361
<b>Edema, n (%)</b>	20 (24.7%)	22 (36.1%)	0.142
<b>Uremia, n (%)</b>	10 (12.3%)	3 (4.9%)	0.129
<b>Other factors, mean (range)</b>			
SBP, mmHg	138.40 (93-210)	145.93 (90-200)	0.735
DBP, mmHg	77.94 (54-120)	83.62 (58-100)	0.610
Hb, g/dL	9.02 (4.1-13.3)	8.34 (3.8-13.8)	0.942
Na, mEq/L	133.81 (116-146)	129.34 (111-142)	0.079
K, mEq/L	4.63 (2.2-7.2)	4.91 (2.4-7.9)	0.905
Cl, mEq/L	99.56 (74-114)	96.7 (77-117)	0.331
HCO <sub>3</sub> , mEq/L	19.38 (7-35)	16.72 (5-29)	0.699
Ca, mg/dL	8.86 (5.4-14.1)	8.55 (6-10.2)	0.332
Mg, mg/dL	2.11 (1.3-3.4)	2.15 (1-4.2)	0.465
PO <sub>4</sub> , mg/dL	5.62 (2.6-12.6)	6.73 (2.8-16.8)	0.496
<b>Other factors, median (range)</b>			
BUN, mg/dL	52 (11-203)	67 (16-232)	0.011*
Cr, mg/dL	4.2 (1.5-32.07)	8.86 (1.97-55.4)	<0.001*
GFR, ml/min/1.73 m <sup>2</sup>	10.46 (1.84-45.38)	6.49 (1.24-39.87)	0.001*

\*Significant at p&lt;0.05

Risk factors of POD occurrence with multivariate analysis are shown in Table 4. In a multivariate model including HT, single kidney, CHF and GFR, the independent predictors of POD occurrence were single kidney (OR 6.13, 95% CI 1.48-25.32, p=0.012) and low GFR (OR 0.94, 95% CI 0.90-0.98, p=0.005).

## Discussion

As previously reported, the most common cause of urinary tract obstruction is cancer, especially prostate cancer<sup>2</sup>. This study showed that cancer is the most common cause of obstruction (90%), but cervical cancer occurs more frequently than prostate cancer. This might be due to Rajavithi Hospital being a tertiary hospital, meaning that many patients with malignant illnesses are treated there.

The true proportion of patients who experience POD is unclear; however, the range is from 0.5 to 52%, depending on how POD is defined<sup>8,9</sup>. The results of this study indicate that the prevalence of POD is about 43%. The proposed mechanisms for POD include osmotic diuresis due to urea<sup>8,9</sup>, involvement of natriuretic and diuretic factors<sup>11</sup>, disordered function of the proximal or distal nephrons, altered tubular permeability, and disturbances in sodium-regulating

hormones. The actual cause is most likely a combination of these mechanisms<sup>17</sup>.

The risks of life-threatening consequences of POD (i.e., hypovolemia, electrolyte disorders) require the close monitoring of patients with PR-AKI and compensation of urinary loss<sup>9</sup>. POD has been the subject of numerous pathophysiological studies<sup>11,13</sup>. However, in clinical practice, the risk of POD occurrence is unknown and predictors have not been determined. In this study, two independent predictors of POD were identified, all available on admission: single kidney and low GFR.

Patients with a single kidney had an increase in renal blood flow and glomerular filtration rate. The mechanisms responsible for this increase in renal blood flow are not known. It might be due to changes in renal nerve activities after contralateral nephrectomy. The elevation of the glomerular filtration rate may be due either to an increase in the population of the functioning nephrons or an increase in the single nephron glomerular filtration rate or both. Both the absolute and fractional excretions of sodium were significantly elevated by volume expansion or urea loading or both. Thus, the present findings suggest that the elevation of two factors can induce POD and natriuresis in single kidney patients<sup>5</sup>.

**Table 4** Risk factors of POD: Results of the multivariate analysis

Factor	Crude OR(95% CI)	p-value	Adjusted OR(95% CI)	p-value
HT	1.23 (0.63-2.41)	0.539	1.05 (0.51-2.14)	0.902
Single kidney	5.10 (1.34-19.42)	0.017*	6.13 (1.48-25.32)	0.012*
CHF	1.44 (0.66-3.18)	0.362	1.16 (0.50-2.69)	0.730
GFR	0.94 (0.90-0.98)	0.005*	0.94 (0.90-0.98)	0.005*

\*Significant at p<0.05

As we observed, POD incidence correlated with low GFR; the present results confirm previous observations that POD occurred 'after a relatively short period of severe obstruction without irreversible renal damage, but always in the presence of advanced renal failure<sup>16</sup>. These mechanisms are responsible for a large increase in salt and water excretion. These defects include the reduction in the reabsorptive capacity of both the surface and deep nephrons, the loss of concentrating ability, and the defective collecting tubule in salt and water reabsorption and its irresponsiveness to ADH<sup>5</sup>.

The results suggest that it is possible to predict POD occurrence before the release of obstruction. Therefore, physicians should consider the predictors of POD occurrence, in combination with the criteria of AKI severity, in order to closely monitor those patients at the greatest risk<sup>16</sup>.

The limitations of this study should be highlighted. First, due to its retrospective design, some data were missing. Second, the sample size was small, and the correlation of some variables could have reached statistical significance if more patients had been included. Third, a large multicenter prospective study with adequate data collection is required to validate our results.

## Conclusion

The results of the current study indicate that the prevalence of post obstructive diuresis was about 43%. Furthermore, single kidney and low GFR were risk factors for post obstructive diuresis after percutaneous nephrostomy in obstructive nephropathy patients.

## References

1. Cerdá J, Lameire N, Eggers P, et al. Epidemiology of acute kidney injury. *Clin J Am Soc Nephrol* 2008;3:881-886.
2. Liano F, Pascual J. Epidemiology of acute renal failure: a prospective, multicenter, community-based study. *Kidney Int* 1996;50:811-818.
3. Khan IH, Catto GR, Edward N, et al. Acute renal failure: Factors influencing nephrology referral and outcome. *QJM* 199;90:781-785.
4. Yarger WE, Schocken DD, Harris RH. Obstructive nephropathy in the rat: Possible roles for the renin-angiotensin system, prostaglandins, and thromboxanes in postobstructive renal function. *J Clin Invest* 1980;65:400-412.
5. Sophasan S, Sorrasuchart S. Factors inducing post-obstructive diuresis in rats. *Nephron* 1984; 38:125-133.
6. Li C, Wang W, Kwon TH, et al. Altered expression of major renal Na<sup>+</sup> transporters in rats with bilateral ureteral obstruction and release of obstruction. *Am J Physiol Renal Physiol* 2003; 285:F889-901.
7. Chevalier RL, Forbes MS, Thornhill BA. Ureteral obstruction as a model of renal interstitial fibrosis and obstructive nephropathy. *Kidney Int* 2009;75:1145-1152.
8. Vaughan ED Jr, Gillenwater JY. Diagnosis, characterization and management of post-obstructive diuresis. *J Urol* 1973;109:286-292.
9. Bishop MC. Diuresis and renal functional recovery in chronic retention. *Br J Urol* 1985; 57:1-5.
10. Oster JR, Singer I, Thatte L, et al. The polyuria of solute diuresis. *Arch Intern Med* 1997;157: 721-729.



11. 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.
12. Schlossberg SM, Vaughan ED Jr. The mechanism of unilateral post-obstructive diuresis. *J Urol* 1984;131:534-536.
13. Maher SF, Schreiner GE, Waters TJ. Osmotic diuresis due to retained urea after release of obstructive uropathy. *N Engl J Med* 1963;268: 1099-1104.
14. Noble VE, Brown DFM. Renal ultrasound. *Emerg Med Clin N Am* 2004;22:641-59.
15. Moghazi S, Jones E, Schroeppe J, et al. Correlation of renal histopathology with sonographic findings. *Kidney Int* 2005;67:1515-1520.
16. Hamdi A, Hajage D, Van Glabeke E, et al. Severe post-renal acute kidney injury, post-obstructive diuresis and renal recovery. *BJU Int* 2012;110(11 Pt C):E1027-34.
17. Nyman MA, Schwenk NM, Silverstein MD. Management of urinary retention: rapid versus gradual decompression and risk of complications. *Mayo Clinic Proceedings*; 72:951-956.