

Post Obstructive Diuresis After Ureterscopy Lithotripsy Procedure in Obstructive Uropathy due to Unilateral Ureteral Stone: A Case Report

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Abstract

Background: Post Obstructive Diuresis (POD) is defined as urine output 200 ml/hour for two consecutive hours immediately subsequent to the relief of urinary tract obstruction. POD occurs in 0,5% to 52% of patients after the relief obstruction. It is usually self-limited, but If the POD becomes pathologic, it can cause serious consequences. We report a case of woman who had POD after underwent ureteroscopic lithotripsy procedure due to unilateral ureteral stone.

Case Presentation: A 35-year-old woman came to ER with episodic left flank pain and LUTS. On examination, left costovertebral angle (CVA) tenderness was positive. An Abdominal X-Ray confirmed left distal ureteral stone. The patient was scheduled to have ureteroscopic lithotripsy procedure. After underwent the procedure, the patient drained 3500 ml of urine over 24 hours and showed clinical deterioration. The patients was diagnosed with acute kidney injury secondary to obstructive uropathy with serum creatinine 2.5 mg/dl. The patient's urine output is 3150-4100 cc per day. Resuscitation with normal saline and moderate hypokalemia correction with potassium chloride infusion is initiated. Close monitoring of fluid balance, urine output, and electrolytes was done on our patients. A negative fluid balance with normal saline infusion should be limited to no more than 75% of 2-hour urine production to avoid diuresis stimulation.

Conclusion: The prolonged course of polyuria in our patient may be a reflection of the chronicity of her obstruction. Patients at high risk for POD should be detected and monitored properly. Early diagnosis and appropriate management of POD will reduce mortality

Keywords: Post-Obstructive Diuresis, Obstructive Uropathy, Fluid and Electrolyte Correction

1. Introduction

Post Obstructive Diuresis (POD) is defined as urine output 200 ml/hour at least two consecutive hours immediately subsequent to the relief of urinary retention or obstructive uropathy. POD may also defined as urine output more than 3000 ml over 24 hours.^(1,2) POD occurs in 0,5% to 52% of patients after the relief obstruction.⁽³⁾ POD may occurs after the release of the obstruction in various ways such as insertion of foley catheter for obstructed bladder, percutaneous nephrostomy (PCN), and Double-J stent (DJ Stent) in patients who have unilateral or bilateral ureteric obstruction. It is usually self-limited and is not typically an issue unless the residual urine is 1500 cc or more. If the POD condition becomes pathologic, it can cause serious consequences such as dehydration, electrolyte abnormalities, hypovolemic shock, and even result to death.⁽⁴⁾ Primary care physicians should be familiar with this potential clinical entity, as they are generally the first to encounter and treat these patients. Early detection and treatment of POD will prevent adverse outcomes. We report a case of women who had POD after underwent ureteroscopic lithotripsy procedure due to unilateral ureteral stone.

2. Case Presentation

A 35 year old woman came to the emergency room (ER) with history of episodic left flank pain for the past 8 months. The pain has gotten worse within a week associated with lower urinary tract symptoms (LUTS) such as difficulty and frequent urination, and sometimes sudden urge to urinate. Initial examination, vital signs were as follows: temperature 36.5 degrees celsius, blood pressure (BP) 118/84 mmHg , heart rate (HR) 80 bpm, respiratory rate (RR) 18/min. On physical examination left costovertebral angle (CVA) tenderness was positive, further examination within normal limits. Laboratory findings, her glucose, serum creatinine, and urea were normal, with mild decrease serum electrolytes sodium 134,64 mmol/L, potassium 3,16 mmol/L, chloride 96,79 mmol/L, Calcium 0,83 mmol/L (Table 1). An Abdominal x-ray confirmed left kidney hydronephrosis due to ureteral stone (Figure 1). Subsequently, the patient was scheduled to have URS (Ureteroscopic Lithotripsy) procedure.

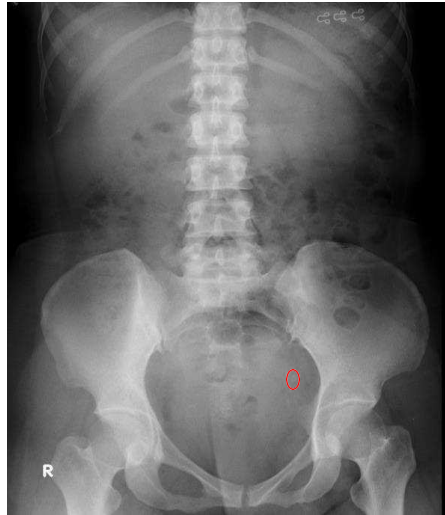


Figure 1. Abdominal X-Ray showing left distal ureterolithiasis

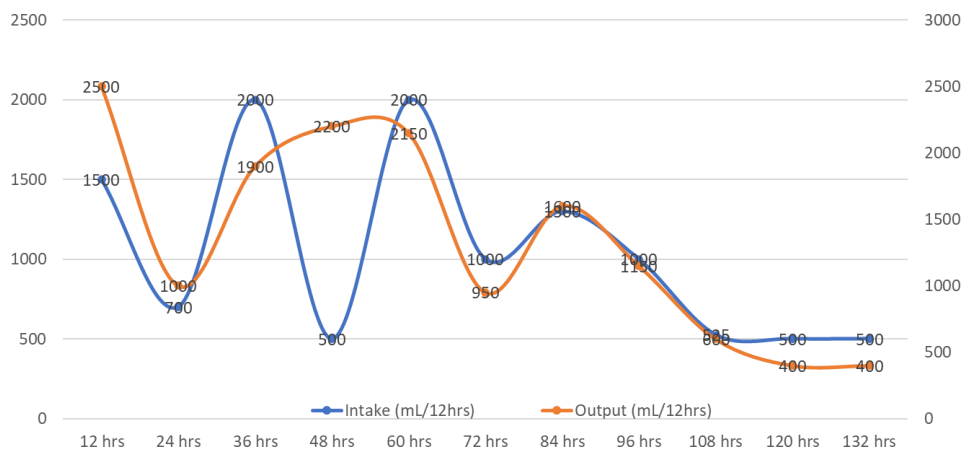


Figure 2. urine output monitor

After underwent the URS procedure, the patient drained 3500 ml of urine over 24 hours and showed clinical deterioration with changes in vital signs, i.e; BP 60/30 mm/Hg, HR 96 bpm, and cold extremity. Laboratory findings showed an increment in serum creatinine and urea to 2.5 mg/dl and 64.42 mg/dl. Thus, the patient was diagnosed with acute kidney injury secondary to obstructive uropathy. Resuscitation with normal saline was initiated.

Despite gradual improvement in her clinical status and renal function, she continued to have polyuria (3150 ml - 4100 ml per day). Laboratory data showed serum osmolarity 335 mOsm/kg, urine SG of 1.010, pH 5.5. Urine analysis was negative for glucose and protein. Partial nephrogenic diabetes insipidus (NDI) was suspected. However, we cannot performed the desmopressin (DDAVP) stimulation test due to limited resources. Thus, the diagnosis of partial nephrogenic diabetes insipidus can not be ruled out in our patient. The clinical course of the patient showed in figure 1.

We evaluated the patient's clinical status day by day. After 5th day of the treatment, the patient was stable hemodynamically and clinically, hence she was moved to the general ward. After 1 day of monitoring without any intravenous fluid support, the patient's condition was still stable with urine output remained less than 200 ml per hour, and then she was discharged home. Vital sign in last hospitalization was BP 125/75, heart rate 88x/m, respiratory rate 16x/min with oxygen saturation 98% in room air condition.

Table 1. Serial Laboratory Finding

| Examination | Day-1 | Day-2 | Day-3 | Day-4 |
|---------------------|--------|--------|--------|--------|
| Sodium (mmol/l) | 134,64 | 133,41 | 137,64 | 133,88 |
| Potassium (mmol/l) | 3,16 | 3,71 | 2,74 | 3,3 |
| Chloride (mmol/l) | 96,79 | 91,36 | 101,98 | 97,1 |
| Calcium (mmol/l) | 0,83 | 0,8 | 0,81 | 0,89 |
| sCreatinine (mg/dl) | 1,01 | 2,5 | 1,76 | 1,32 |

3. Discussion

Urinary retention is a common clinical condition addressed in both primary and secondary healthcare facilities. The incidence of urinary retention is higher in men than in women and increases with age.⁽⁵⁾ It is estimated 3 to 7 cases per 100,000 women yearly, with the female-to-male ratio is 1 : 13.⁽⁶⁾ POD is a clinical diagnosis based on urine output after decompression of urinary tract obstruction.⁽⁴⁾ Our patient met the definition for post-obstructive diuresis with 3500 ml of urine output over 24 hours after the release of obstruction.

It is difficult to predict which and when the patient will become POD after the release of obstruction. Study from Hamdi, et al stated that initial presence of elevated serum creatinine, high level of sodium bicarbonate and urinary retention as independent risk factors for developing POD.⁽⁷⁾ Another study also found that the presence of renal insufficiency, heart failure, dizziness, and central nervous system deterioration are risk factors for POD.⁽⁸⁾ Apart from age, sex, other risk factors associated with an increased of urinary retention include LUTS, prostate disease, long-standing diabetes, recurrent catheterization, fecal impaction, and the use of anticholinergic medication.⁽⁵⁾

Physiological POD is self-limiting and generally lasts 24 hours. Pathological POD generally lasts longer than 48 hours and can be exacerbated with excessive intravenous fluid replacement.⁽⁸⁾ In this patient the POD occurred for about 72 hours and decreased gradually into normal status. The pathophysiology of POD is complex to understood, which includes reduction in the medullary concentration gradient secondary to vascular washout and down-regulation of sodium transporters in the thick ascending loop of Henlé, reduction in glomerular filtration rate, leading to reduced blood flow and loss of juxtamedullary nephrons, reduced response of the collecting duct to antidiuretic hormone, leading to nephrogenic diabetes insipidus, down-regulation of aquaporin channels and renal cell stretching causes increased levels of angiotensin II and transforming growth factor-beta, leading to cell apoptosis and fibrosis.⁽⁹⁾ A combination of these mechanisms is likely to contribute. Due to this pathophysiology, partial nephrogenic diabetes insipidus was suspected in our patient. However, we cannot do

the desmopressin (DDAVP) stimulation test due to limited resources. Thus, the diagnosis of partial nephrogenic diabetes insipidus can not be ruled out in our patients.

Subsequent to the relief of obstructive uropathy, usually there is an increment in sodium and water excretion even with temporary decrease in glomerular filtration rate. Volume expansion that happens during obstructive uropathy may suppress antidiuretic hormone (ADH) and aldosterone, this leads to minimal reabsorption at the distal and collecting duct level until volume depletion sets in to reestablish reabsorption. Physiologically, this is usually a self-limiting phenomenon.⁽¹⁰⁾ In most patients, the diuresis will resolve once the kidneys normalize the volume and solute status reach homeostasis by a 24-hour urine production of less than 3 L.⁽⁵⁾ But in this case the urine production is more than 3 L in 24-hour. It happens because it eliminates salt and water even after homeostasis has been achieved, referred as pathologic POD.⁽¹⁰⁾ These patients are at risk of severe dehydration, electrolyte imbalance, hypovolemic shock, and even death if fluid and electrolyte replacement is not initiated.⁽⁵⁾

It was once thought that large bladder volumes decompression should be performed gradually by intermittent clamping of the catheter to prevent hematuria, post-obstructive diuresis, and hypotension.⁽⁵⁾ However, a recent study by Etafy, et al stated that no significant difference was found between gradual and rapid decompression of the bladder in patients with acute urinary retention.⁽¹¹⁾ Hence, we performed rapid decompression of the bladder in our patients, however our patients develop into POD after underwent the URS procedure. To ensure early detection of POD, it is important to monitor hourly urine specimens for volume, osmolality, sodium, and urea.⁽⁴⁾ It is also important to monitor the patient's fluid status, electrolytes serum, creatinine, urea, magnesium, and phosphate levels.⁽⁵⁾

Urine osmolality can be estimated from specific gravity. A specific gravity of 1.010 is iso-osmotic with serum osmolality, indicating that the kidneys do not need to concentrate the urine. If the urine SG is 1.020 or more, means that the urine is being concentrated by the kidneys and diuresis is resolving. If the urine's SG is lower than 1.010, the kidneys are not or may not be able to concentrate the urine, this would indicate pathological POD.^(5,9,12) The urine specific gravity in our patients was 1.010 which makes the urine osmolality normal. Thus, fluid balance should be closely monitored in our patients.

Treatment of POD should be focused on complete relief of urinary tract obstruction, fluid and electrolytes replacement, and close monitoring of the patients.⁽⁴⁾ Fluid balance should be closely monitored and a negative balance should be targeted in these patients. Intravenous (IV) fluid support should be normal saline and limited to no more than 75% of the prior 1 to 2 hour urine production to avoid stimulation further diuresis.⁽¹²⁾ Individuals without cognitive impairment should continue to take hydration orally. However, cognitively impaired patients should receive 0.45% normal saline intravenously.⁽⁵⁾ If pathological post-obstructive diuresis develops, diuresis will continue despite euvolemic state is reach – fluid replacement will be required, but the amount and type of intravenous fluid should be determined.⁽⁹⁾ Close monitoring of fluid balance status, urine output every 2 hours, and electrolytes every 24 hours was done on our patients. Following the relief of the obstruction, our patients were hemodynamically unstable with urine output 3500 mL over 24 hours. Therefore, treatment with IV fluid replacement then initiated. Our patients get 2750 mL of normal saline in the first 24 hours, followed by 75% of fluid replacement based on urine output. After several days, the fluid replacement should be tapered off.⁽⁴⁾ Gradual improvement was shown in our patient, therefore we tapered off her fluid replacement. We stopped intravenous fluid replacement on her 5th day of the treatment due to her hemodynamically stable condition, urine output remained below 200 mL per hour, and her serum electrolyte levels remained within normal limits. The patient was discharged 24 hours later.

There are limited clues to help identify which patients will progress to pathological POD. Besides the diagnosis criteria, we should closely monitor the patient's clinical condition, fluid status, urine output, serum electrolytes, and kidney function level. Hourly monitoring of vital signs, urine output, and serial laboratory studies at least every 24 hours.

4. Conclusion

Urinary tract obstruction can cause several damage or diminished renal function resulting in POD. The prolonged course of polyuria in our patient may be a reflection of the chronicity of her obstruction. Patients at

high risk for POD should be detected and monitored properly. Serial urine and laboratory parameters should be used to guide appropriate fluid and electrolyte management with the intention to maintain intravascular volume, electrolyte serum, and restore renal function's baseline. Early diagnosis and appropriate management of POD will reduce mortality.

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6. Conflict of Interests

The author(s) declared no potential conflict of interest with respect to the case report, authorship, and/or publication of this article

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