# Postobstructive diuresis

## *Pay close attention to urinary retention*

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### EDITOR'S KEY POINTS

- Acute drainage of an obstructed urinary tract can unmask deranged renal mechanisms and result in uncontrolled, unregulated urine production known as *postobstructive diuresis* (POD).
- Postobstructive diuresis can occur in up to 50% of patients with substantial urinary tract obstruction and can be life-threatening if it becomes pathologic.
- Patients with decompressed urinary obstruction might have to be admitted for a 24-hour observation period. If pathologic POD ensues, then polyuria will continue even after a euvolemic state is reached. Patients with pathologic POD require strict monitoring of vital signs, fluid status, and serum electrolyte levels, and benefit from consultation with a nephrologist.

### POINTS DE REPÈRE DU RÉDACTEUR

- Le drainage aigu des voies urinaires obstruées peut dévoiler des mécanismes rénaux déficients et provoquer une production d'urine non contrôlée et non régulée, connue sous le nom de *diurèse postocclusive* ou syndrome de levée d'obstacle (SLO).
- Le syndrome de levée d'obstacle peut se produire chez près de 50 % des patients souffrant d'une importante obstruction des voies urinaires et peut mettre leur vie en danger si elle devient pathologique.
- Les patients chez qui on dégage une obstruction urinaire devraient être admis pour une période d'observation de 24 heures. S'il s'ensuit un SLO pathologique, la polyurie continuera même après que le patient aura atteint un état euvolémique. Les patients souffrant d'un SLO pathologique exigent une surveillance rigoureuse des signes vitaux, de l'état liquidien et des niveaux d'électrolytes sériques, et auraient avantage à consulter un néphrologue.

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## Abstract

***Objective*** To educate primary health care professionals about the diagnosis and treatment of postobstructive diuresis (POD), a rare but potentially lethal complication associated with the relief of urinary obstructions.

***Sources of information*** The main concepts and clinical evidence reviewed in this article were derived from a literature search of PubMed and Google Scholar. Expert opinion was used to supplement recommendations in areas with little evidence.

***Main message*** Urinary retention is a frequently encountered presentation seen by all physicians. Most family physicians are comfortable treating these patients, initiating investigations, and organizing appropriate follow-up. This article reviews a rare but potentially lethal complication known as POD. Postobstructive diuresis is a polyuric response initiated by the kidneys after the relief of a substantial bladder outlet obstruction. In severe cases this condition can become pathologic, resulting in dehydration, electrolyte imbalances, and death if not adequately treated. Primary care physicians should be familiar with this potential clinical entity, especially as they are generally the first to encounter and treat these patients.

***Conclusion*** Physicians aware of POD will be able to identify patients at risk and arrange the appropriate monitoring after relieving a urinary obstruction. Early diagnosis and treatment of pathologic POD will prevent mortality.

# Diurèse postocclusive

## *Apporter une attention particulière dans les cas de rétention urinaire*

## Résumé

***Objectif*** Renseigner les professionnels des soins primaires concernant le diagnostic et le traitement du syndrome de levée d'obstacle (SLO), une complication rare mais potentiellement mortelle associée au soulagement des obstructions urinaires.

***Sources des données*** Les principaux concepts et données scientifiques cliniques dans le présent article sont tirés d'une recherche documentaire dans PubMed et Google Scholar. Les opinions d'experts viennent compléter les recommandations dans les domaines où les données probantes se font rares.

***Message principal*** Tous les médecins voient couramment des cas de rétention urinaire. La plupart des médecins de famille se sentent à l'aise de traiter de tels patients, amorcent des investigations et organisent le suivi qui s'impose. Cet article présente une complication rare mais possiblement mortelle connue sous le nom de SLO. Le SLO, ou diurèse postocclusive, est une réaction polyurique déclenchée par les reins après le soulagement d'une obstruction importante du col de la vessie. Dans les cas graves, ce problème peut devenir pathologique, entraînant une déshydratation, un déséquilibre hydroélectrolytique et la mort, s'il n'est pas traité adéquatement. Les médecins de soins primaires devraient être familiers avec ce problème clinique potentiel, surtout qu'ils sont généralement les premiers à voir et traiter de tels patients.

***Conclusion*** Les médecins qui connaissent le SLO seront en mesure de dépister les patients à risque et d'assurer une surveillance appropriée après avoir soulagé l'obstruction urinaire. Un diagnostic et un traitement sans délai d'un SLO pathologique préviendront la mortalité

## Case description

An 87-year-old man was urgently recalled to his family medicine clinic after his physician received an ultrasound report revealing a postvoid residual volume estimated at 3790 mL, bilateral severe hydronephrosis, a marked distended bladder, and 2 suspicious 2.5-cm solid growths arising from the bladder wall (**Figure 1**). The investigation had been ordered 6 days previously after identifying a progressive rise in the patient's serum creatinine level. The patient's serum creatinine levels during the previous 3 months were as follows: 141 µmol/L, 165 µmol/L, 180 µmol/L, and 203 µmol/L. On average, his baseline creatinine levels were between 130 and 150 µmol/L and his serum glucose and electrolyte levels were consistently within normal limits. His past medical history included chronic kidney disease, type 2 diabetes, hypertension, congestive heart failure, osteoarthritis, and benign prostatic hyperplasia. His relevant past surgical history included a transurethral resection of the prostate 2½ years previously. He denies any symptoms other than passing only 250 mL of urine during the previous 48 hours. On examination, his abdomen was distended, with the top of the bladder palpable midway between the umbilicus and the xiphoid process. Further, an examination of his external genitalia revealed a substantial phimosis to the distal 1 cm of the foreskin.

## Sources of information

The literature review was conducted using PubMed and Google Scholar and the key words *urinary retention*, *urinary obstruction*, *bladder outlet obstruction*, and *post obstructive diuresis*. All relevant articles providing information and evidence on urinary retention and postobstructive diuresis (POD) epidemiology, risk factors, diagnosis, and management were considered for inclusion. Recommendations were graded based on their level of evidence in accordance with the Canadian Task Force on Preventive Health Care.[1] Level I evidence originated from properly conducted randomized controlled trials, systematic reviews, or meta-analyses. Level II evidence originated from other non-randomized comparison trials, and level III evidence is based on expert opinion or consensus statements.

## Main message

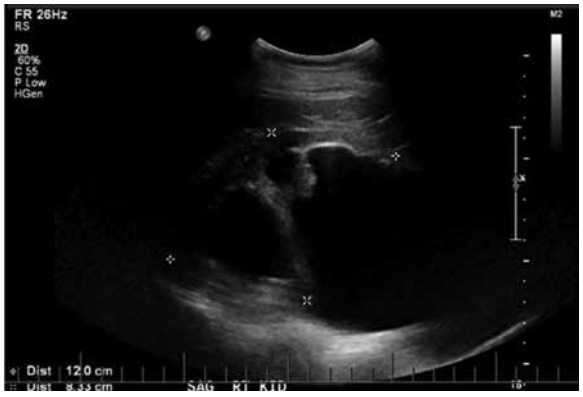
***Urinary retention.*** Urinary retention is a common clinical condition addressed in both primary health care clinics and hospitals. It is traditionally classified as acute or chronic. Acute urinary retention (AUR) is a rapid-onset condition associated with suprapubic pain and the inability to urinate. Conversely, chronic urinary retention (CUR) has a gradual onset, with no associated pain and the ability to pass only small amounts of urine. These patients might also present with overflow urinary incontinence.[2] Chronic urinary retention is further classified into high- and low-pressure CUR. The presence or absence of intrinsic detrusor pressure is what differentiates them. High-pressure CUR results from an obstruction with the presence of detrusor muscle activity, while low-pressure CUR involves detrusor failure with low intravesical pressure.[3] This distinction is important, as high detrusor pressure can lead to upper urinary tract damage and present with new-onset hypertension, peripheral edema, or renal dysfunction.[5]

The incidence of urinary retention is higher in men than in women and increases with age. It is estimated that 1 in 10 men between the ages of 70 and 79 and 3 in 10 men between the ages of 80 and 89 will have an episode of AUR within their lifespans.[2] Apart from age and sex, other risk factors associated with an increased risk of urinary retention include lower urinary tract symptoms, prostate disease, long-standing diabetes, recurrent catheterization, fecal impaction, and the use of anticholinergic medications.[5,6]

After a thorough history taking and physical examination, the patient should be assessed for a palpable bladder and neurologic integrity, and a rectal examination should be performed to assess the prostate and rectal tone. Urinary retention can be confirmed with a postvoid bladder ultrasound or the placement of a urinary catheter demonstrating a large residual urine volume. There are currently no universally accepted diagnostic values for a normal postvoid residual volume, but it has been reported that AUR is associated

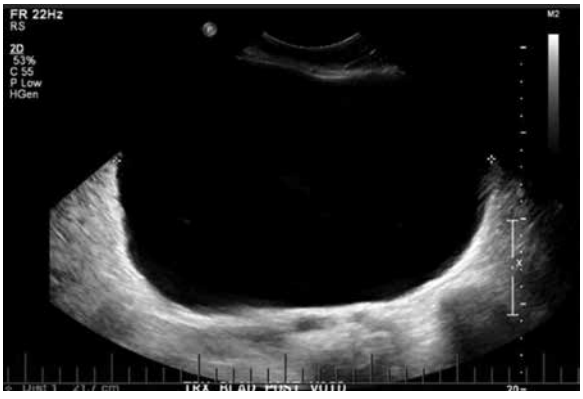
**Figure 1.** Renal ultrasound of bladder outlet obstruction


Left hydronephrosis




Right hydronephrosis


Bladder masses




Distended postvoid bladder

with a residual urine volume of 500 to 600 mL, whereas CUR is associated with a volume of greater than 800 mL.[6,7] Commonly, patients with CUR present with 1.0 L to 1.5 L of retained urine, with some case reports noting volumes greater than 4 L.[7]

***Bladder outlet obstruction (BOO).*** Bladder outlet obstruction is the most common cause of urinary retention and a list of causes is outlined in **Box 1**.[2,8] In many cases this obstructive process is gradual and can evolve into CUR. As the BOO progresses, the detrusor pressure will also increase, resulting in high-pressure CUR. In severe cases, BOO can lead to vesicoureteral reflux and subsequent hydronephrosis. Prolonged high-pressure hydronephrosis can injure nephrons and result in renal dysfunction.[9]

***Postobstructive diuresis***. Postobstructive diuresis is a polyuric state in which copious amounts of salt and water are eliminated after the relief of a urinary tract obstruction. The incidence of POD is unclear but estimates suggest 0.5% to 52% of patients will experience POD after relief of obstruction.[10] It generally occurs after relieving BOO, bilateral ureteric obstruction, or unilateral ureteric obstruction in a solitary kidney.[11] Diuresis is a normal physiologic response to help eliminate excess volume and solutes accumulated during the prolonged obstruction. In most patients, the diuresis will resolve once the kidneys normalize the volume and solute status and homeostasis is achieved. Some patients will continue to eliminate salt and water even after homeostasis has been reached, referred to as pathologic POD. These patients are at risk of severe dehydration, electrolyte imbalances, hypovolemic shock, and even death if fluid and electrolyte replacement is not initiated.[9]

Numerous mechanisms have been proposed to describe the pathophysiology of POD, which include a progressive reduction in the medullary concentration gradient secondary to vascular washout and downregulation of sodium transporters in the thick ascending loop of Henle; reduction in glomerular filtration rate, which leads to ischemia and loss of juxtamedullary nephrons; and reduced response of the collecting duct to circulating antidiuretic hormone, leading to nephrogenic diabetes insipidus.[12-15] The most likely cause is a combination of all these mechanisms.

**Box 1. Causes of bladder outlet obstruction**

The following are causes of bladder outlet obstruction:

- Genitalia: Meatal stenosis, phimosis, paraphimosis, atrophic vaginitis
- Urethra: Stones, strictures, diverticulum, posterior urethral valves, carcinoma, surgery
- Prostate: Benign prostatic hyperplasia, calculi, abscess, prostate carcinoma
- Gynecologic: Prolapse, cystocele, pregnancy, ovarian mass, uterine tumour, cervical tumour
- Bladder: Calculi, blood clot, tumour, bladder neck dysfunction
- Bowel: Fecal impaction
- Neurogenic: Multiple sclerosis, diabetes, spinal cord trauma, Parkinson disease, cerebrovascular accident

Data from Klahr.[8]

It is difficult to predict which patients will develop POD after the release of urinary tract obstruction. A recent article by Hamdi et al identified the initial presence of a high serum creatinine level, a high sodium bicarbonate level, and urinary retention as independent risk factors for developing POD after decompressing an obstructed urinary tract.[16] Further, there are very few clinical markers that help predict which individuals with physiologic POD will progress to pathologic POD. There is no correlation between initial creatinine values, urea values, electrolyte values, creatinine clearance, or presence of hypertension with the severity of diuresis.[17,18] However, some studies have found that the presence of renal insufficiency, heart failure or evidence of volume overload, dizziness, and central nervous system depression are risk factors for developing substantial POD.[14,19]

Postobstructive diuresis is a clinical diagnosis based on urine output after decompressing an obstructed bladder or ureter. Urine production exceeding 200 mL per hour for 2 consecutive hours or producing greater than 3 L of urine in 24 hours is diagnostic of POD (level III evidence).[14,19] Physiologic POD is self-limiting and generally lasts 24 hours. Pathologic POD generally lasts longer than 48 hours and can be exacerbated with excessive intravenous fluid replacement.[14]

The treatment of urinary retention begins with immediate catheter placement to decompress the bladder. It was once thought that decompression of the bladder should be performed gradually with periodic clamping to prevent hematuria, hypotension, and POD. However, a recent review by Nyman et al revealed that there is no evidence linking these complications with quick, complete bladder emptying, and the authors conclude that this is a safe and effective method of bladder decompression (level I evidence).[10,20] A list of general urinary catheter complications is outlined in **Box 2**.[21]

Patients with decompressed urinary obstruction (BOO, bilateral ureteric obstruction, or unilateral ureteric obstruction of a solitary kidney) need to be monitored closely for POD and might have to be admitted for a 24-hour observation period. These individuals should have their urine output recorded every 2 hours and vital signs checked every 6 to 8 hours. Further, their serum electrolyte (especially potassium), magnesium, phosphate, urea, and creatinine levels should be checked every 12 to 24 hours and corrected if necessary.[14] These patients should be allowed free access to oral hydration and can be discharged after 24 hours if POD is not confirmed.[12] A follow-up visit with a urologist should be arranged for a later date for further management of their obstructive uropathy.

If the patient's urine output exceeds 200 mL per hour for 2 consecutive hours, or is greater than 3 L over 24 hours, then this is diagnostic of physiologic POD and requires closer monitoring for conversion to pathologic POD. These individuals should continue to have their urinary outputs recorded, should be weighed daily, and should have their serum electrolyte, magnesium, phosphate, urea, and creatinine levels monitored every 12 hours or more frequently as necessary (level III evidence).[9,12,14] A urine sample should be collected for urinary sodium and potassium levels and urine osmolality to determine if it is a salt or urea type of diuresis.[9] Urea diuresis is generally self-limiting, whereas salt diuresis can convert to pathologic POD and requires careful monitoring of serum electrolyte levels and hydration status. Spot urine sodium levels greater than 40 mEq/L suggest renal tubular injury and if prolonged can lead to pathologic POD.[14]

A simple method to estimate urine osmolality, if an automated method is not available, is to assess the urine 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. This is consistent with physiologic POD and is generally self-limiting. A specific gravity of 1.020 demonstrates that the kidneys are concentrating the urine and POD has resolved or has nearly resolved. However, a specific

**Box 2. Complications of urinary catheters**

The following are complications associated with urinary catheters:

- Urethral trauma
- Urinary tract infection
- Retained balloon fragments
- Bladder stone formation
- Bladder fistula
- Bladder perforation

Data from Schaeffer.[21]

gravity of 1.000 is hypo-osmotic with serum osmolality, indicating the kidneys' inability to concentrate the urine. This is consistent with pathologic salt-wasting POD and should alert the health care team to monitor the patient closely (level III evidence).[14]

Fluid balance should be closely monitored and a negative balance should be targeted in these patients. It is recommended to replace 75% of the previous 1-hour urinary output (level III evidence).[9] Excessive fluid should be avoided as it can prolong or exacerbate the diuresis. Individuals without cognitive impairment should continue to take hydration orally. However, cognitively impaired patients should receive 0.45% normal saline intravenously. In physiologic POD, when the patient reaches a euvolemic state, the diuresis should resolve and this will be evident by a 24-hour urine production of less than 3 L.[19]

If pathologic POD ensues, then polyuria will continue even after a euvolemic state has been reached. These patients are at risk of hypovolemia and can become hemodynamically unstable. Prolonged polyuria also places the patient at risk of electrolyte and acid-base disturbances (**Box 3**).[9,14,18] Patients with pathologic POD require strict monitoring of vital signs, fluid status, and serum electrolyte levels, and benefit from the involvement of a nephrologist. Regardless of cognitive function, all patients with pathologic POD require intravenous fluid replacement run at a negative balance. The type and amount of fluid should be tailored to the patient's needs based on his or her serum and urinary electrolyte levels and clinical hydration status.

## Case resolution

An abdominal examination and ultrasound imaging confirmed BOO, complicated by a phimosis. With the aid of an assistant and 2.5% lidocaine cream, the foreskin was successfully retracted and a no. 16 French gauge urinary catheter was inserted. The patient drained 5500 mL of urine over 90 minutes. Arrangements were made to admit the patient to the local hospital under the care of family medicine to further monitor urine output, hydration status, and serum electrolyte levels. On admission, his serum electrolyte and glucose levels were normal, with serum creatinine and urea levels elevated to 190 µmol/L and 14.7 mmol/L, respectively. The patient's course in hospital was unremarkable, his urine output remained below 200 mL per hour, and his serum electrolyte levels remained within normal limits. Although this patient was at risk of developing POD, he fortunately did not. The patient was discharged 24 hours later with a catheter in situ. A cystoscopy appointment was arranged to investigate his bladder masses and revealed regrowth of residual prostatic tissue at the base of his bladder.

**Box 3. Complications of postobstructive diuresis**

The following are complications of pathologic postobstructive diuresis:

- Volume depletion
- Hyponatremia or hypernatremia
- Hypokalemia
- Hypomagenesemia
- Metabolic acidosis
- Shock
- Death

Data from Baum et al[9] and Gonzales.[14]

## Conclusion

Urinary retention is a common clinical condition encountered by all physicians at some point in their careers. As described above, the initial medical approach involves decompressing the bladder with the placement of a urethral catheter, followed by a workup to identify the underlying cause. However, how often do primary care physicians consider the potential effects of rapidly draining a patient's bladder on the rest of the body? One might argue there is little effect, as doing so mimics urination, the normal physiologic process that has been perfected over thousands of years of natural selection. However, in some situations, acute drainage of an obstructed urinary tract can unmask deranged renal mechanisms and result in uncontrolled, unregulated urine production known as *postobstructive diuresis*. Postobstructive diuresis is important because it can occur in up to 50% of patients with substantial urinary tract obstruction and can be life-threatening if it becomes pathologic and is not adequately treated. The goal of this article is to make primary care physicians aware of this clinical entity and help them confidently identify individuals at risk, initiate appropriate monitoring to allow for early diagnosis, and provide adequate treatment to avoid any adverse outcomes.

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**Contributors**

**Drs Halbgewachs** and **Domes** contributed to the literature review and interpretation, and to preparing the manuscript for submission.

**Competing interests**

None declared

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**References**

1. Grades of Recommendation, Assessment, Development and Evaluation (GRADE) Working Group 2011. *Grades of Recommendation, Assessment, Development and Evaluation (GRADE)*. Calgary, AB: Canadian Task Force on Preventive Health Care; 2011. Available from: **http://canadiantaskforce.ca/methods/grade/**. Accessed 2015 Jan 15.

2. Muhammed A, Abubakar A. Pathophysiology and management of urinary retention in men. *Arch Int Surg* 2012;2(2):63-9.
3. Abrams PH, Dunn M, George N. Urodynamic findings in chronic retention of urine and their relevance to results of surgery. *Br Med J* 1978;2(6147):1258-60.
4. Jones DA, Gilpin SA, Holden D, Dixon JS, O'Reilly PH, George NJ. Relationship between bladder morphology and long-term outcome of treatment in patients with high pressure chronic retention of urine. *Br J Urol* 1991;67(3):280-5.
5. Borrie MJ, Campbell K, Arcrese ZA, Bray J, Hart P, Labete T, et al. Urinary retention in patients in a geriatric rehabilitation unit: prevalence, risk factors, and validity of bladder scan evaluation. *Rehabil Nurs* 2001;26(5):187-91.
6. Kaplan SA, Wein AJ, Staskin DR, Roerhborn CG, Steers WD. Urinary retention and post-void residual urine in men: separating truth from tradition. *J Urol* 2008;180(1):47-54. Epub 2008 May 15.
7. Mitchell JP. Management of chronic urinary retention. *Br Med J* (Clin Res Ed) 1984;289(6444):515-6.
8. Klahr S. Obstructive nephropathy. *Intern Med* 2000;39(5):355-61.
9. Baum N, Anhalt M, Carlton CE Jr, Scott R Jr. Post-obstructive diuresis. *J Urol* 1975;114(1):53-6.
10. Nyman MA, Schwenk NM, Silverstein MD. Management of urinary retention: rapid versus gradual decompression and risk of complications. *Mayo Clin Proc* 1997;72(10):951-6.
11. Schlossberg SM, Vaughan ED Jr. The mechanism of unilateral post-obstructive diuresis. *J Urol* 1984;131(3):534-6.
12. Singh I, Strandhoy JW, Assimos DG. Pathophysiology of urinary tract obstruction. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peter CA, editors. *Campbell-Walsh urology*. 10th ed. Philadelphia, PA: Elsevier Saunders; 2012. p. 1107-8.
13. Sonnenberg H, Wilson DR. The role of the medullary collecting ducts in postobstructive diuresis. *J Clin Invest* 1976;57(6):1564-74.
14. Gonzalez CM. Pathophysiology, diagnosis, and treatment of the postobstructive diuresis. In: McVary KT, editor. *Management of benign prostatic hypertrophy*. New York, NY: Humana Press; 2004. p. 35-45.
15. Loo MH, Vaughan ED. Obstructive nephropathy and postobstructive diuresis. *AUA Update Series* 1985;4:1-7.
16. Hamdi A, Hajage D, Van Glabeke E, Belenfant X, Vincent F, Gonzalez F, et al. Severe post-renal acute kidney injury, post-obstructive diuresis and renal recovery. *BJU Int* 2012;110(11 Pt C):E1027-34. Epub 2012 May 15.
17. Bishop MC. Diuresis and renal functional recovery in chronic retention. *Br J Urol* 1985;57(1):1-5.
18. Maher JF, Schreiner GE, Waters TJ. Osmotic diuresis due to retained urea after release of obstructive uropathy. *N Engl J Med* 1963;268:1099-104.
19. Vaughan ED Jr, Gillenwater JY. Diagnosis, characterization and management of post-obstructive diuresis. *J Urol* 1973;109(2):286-92.
20. Ahmed M, Abubakar A, Lawal AT, Bello A, Maitama HY, Mbibu HN. Rapid and complete decompression of chronic urinary retention: a safe and effective practice. *Trop Doct* 2013;43(1):13-6. Epub 2013 Feb 26.
21. Schaeffer AJ. *Complications of urinary bladder catheters and preventive strategies*. Waltham, MA: UpToDate; 2013.