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Massive Urinary Retention Presenting as Dyspnea and Upper Abdominal Discomfort From Bladder-Related Diaphragmatic Compression: A Case Report

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Patient: Male, 58-year-old
Final Diagnosis: Urinary retention
Symptoms: Abdominal pain • dyspnea
Clinical Procedure: —
Specialty: Urology
Objective: Unusual clinical course

Background: Massive urinary retention is an emergency diagnosis that has been rarely reported in the literature. Complications include bladder rupture, acute renal failure, ascending urinary infection, and shock. Although typically presenting as difficulty voiding, subtle signs can include dyspnea and upper abdominal discomfort, as seen in our case, potentially delaying definitive diagnosis and treatment.

Case Report: We describe massive urinary retention in a 58-year-old man presenting to the emergency department with shortness of breath and upper abdominal discomfort. His symptoms were initially concerning for complications from metastatic pancreatic cancer. Examination revealed dyspnea and diffuse abdominal tenderness with mild distention. Computed tomography imaging revealed massive distention of the urinary bladder with the dome extending above the umbilicus. After consultation with Urology, foley catheterization was performed with return of 3.8 liters of urine. The patient was observed following decompression without hemodynamic instability or hematuria. Given his history of obstructive benign prostatic hypertrophy status after transurethral aquablation, he was discharged with an indwelling catheter. In the outpatient setting, a trial of void was unsuccessful, and he remained dependent on an indwelling catheter at the last documented follow-up.

Conclusions: Clinicians evaluating patients for dyspnea and abdominal discomfort should be alert to possible urologic causes, including massive bladder dilation from retention. Our patient's dyspnea and upper abdominal discomfort improved promptly after bladder decompression, supporting an association with massive bladder distention. A cephalad mass effect of the distended bladder on intra-abdominal structures and the diaphragm is a plausible but unconfirmed explanation, as no imaging or physiologic study directly demonstrated this mechanism. Prompt recognition and management along with appropriate consultation are necessary to mitigate risk of adverse outcomes in this urologic emergency.

Keywords: Emergency Medicine • Urinary Retention • Urinary Catheterization
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954899

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Introduction

Normal functional bladder capacity in healthy adults typically ranges from 400 to 600 mL, with the urodynamic desire to void occurring at approximately 500 mL [1]. Acute prolonged bladder overdistension is formally defined as a persistent filling volume exceeding 120% of normal functional capacity for at least 24 hours [2]. While non-neurogenic chronic urinary retention is commonly defined by the American Urological Association as a persistently elevated post-void residual volume greater than 300 mL [3], clinical thresholds for high-risk or severe retention in international guidelines often scale upwards of 1000 mL [4]. However, documented cases of extreme, massive bladder distension—specifically, volumes approaching or exceeding 3 to 4 liters—are exceptionally rare in modern clinical practice [5].

In typical cases of severe urinary retention, the clinical presentation is dominated by lower urinary tract symptoms, acute kidney injury, bilateral hydroureteronephrosis, or overflow incontinence [6]. Furthermore, extreme overdistension places patients at significant risk for severe complications, including spontaneous intraperitoneal bladder rupture if intravesical pressure spikes acutely against a thinned bladder wall [7]. When chronic distension develops insidiously, however, the detrusor muscle can slowly accommodate enormous volumes over time without immediate rupture or a marked elevation in baseline storage pressure [5]. In such instances, patients may remain paradoxically asymptomatic from a urologic standpoint, presenting instead with symptoms caused by the physical mass effect of the enlarged bladder pressing against adjacent organ systems [8].

We present a remarkable case of massive, silent urinary bladder overdistension in a 58-year-old man who presented with dyspnea on exertion, early satiety, and abdominal discomfort. This report highlights the insidious nature of painless, massive urinary retention in complex oncology patients receiving multi-agent chemotherapy and anticholinergic adjuncts and underscores the critical importance of considering genitourinary mass effect in the differential diagnosis of unexplained cardiopulmonary and gastrointestinal problems.

Case Report

A 58-year-old man presented to the emergency department (ED) with a chief concern of shortness of breath and dyspnea on exertion over the previous 6 days. He additionally reported upper abdominal discomfort, early satiety, and pain with eating. He did not endorse difficulty urinating on initial evaluation and reported ability to urinate prior to his arrival to the ED. He denied any fevers, chills, vomiting, diarrhea, or recent history of urinary tract infection, and did not report any history of bowel obstruction or pulmonary embolism. His medical history was significant, however, for metastatic pancreatic cancer, and he was on an active chemotherapy regimen of paclitaxel, cisplatin, and gemcitabine. Oral medications pertinent to this emergency presentation included prochlorperazine 10 mg as needed, ondansetron 4 mg as needed, dexamethasone 4 mg (given as an adjunct with the chemotherapy regimen), dicyclomine 10 mg as needed, and omeprazole 40 mg twice daily. He denied any anticoagulant use. He reported an episode of urinary retention 7 years prior, later deemed secondary to obstructive benign prostatic hypertrophy (BPH), and was treated with transurethral aquablation therapy. His surgical history was otherwise unremarkable other than remote bilateral inguinal hernia repair.

Upon arrival to the ED, the patient's vital signs revealed mild tachycardia with a heart rate of 112, blood pressure of 113/80 mm Hg, temperature of 36.7 °C, respiratory rate of 18, and oxygen saturation of 98% on room air. Physical examination was concerning for dyspnea noted with minimal exertion and upper abdominal tenderness without rebound or guarding. The patient's skin was warm, dry, and well-perfused. The genitourinary exam was unremarkable. Initial laboratory evaluation was significant for notable absence of leukocytosis with white blood cell count of 4.1×10^9/L, hyponatremia of 132 mmol/L, hypokalemia of 3.5 mmol/L, normal creatinine of 0.97 mg/dL, normal albumin of 3.5 g/dL, unremarkable N-terminal pro B-type natriuretic peptide of 174 pg/mL, and mild elevation of lactate to 2.4 mmol/L. The patient's liver function tests were near his baseline as well. Interestingly, he was able to provide a urine specimen for urinalysis, which revealed no bacteria, negative leukocyte esterase, negative nitrites, 3 WBCs/hpf, and 5 RBCs/hpf. Given his history and findings on physical examination, computed tomography (CT) of the chest, abdomen, and pelvis with contrast was performed.

CT imaging revealed massive urinary bladder distention with the dome extending above the umbilicus (**Figures 1-3**). Findings were suggestive of massive bladder overdistension of uncertain chronicity. There was no evidence on imaging of bladder rupture, obstructive genitourinary masses, hydronephrosis, or hydroureter. Furthermore, there was no finding of acute pulmonary embolism or other intrathoracic pathology to explain the shortness of breath. Given this constellation of findings, Urology was consulted. Recommendations from Urology included insertion of an indwelling foley catheter for bladder decompression and a period of observation in the ED to ensure symptomatic improvement. Foley catheterization was performed successfully, and the bladder was drained of 3.8 liters of urine, with significant improvement in subjective dyspnea and abdominal discomfort. The bladder was decompressed rapidly without catheter clamping, with the full volume returning over

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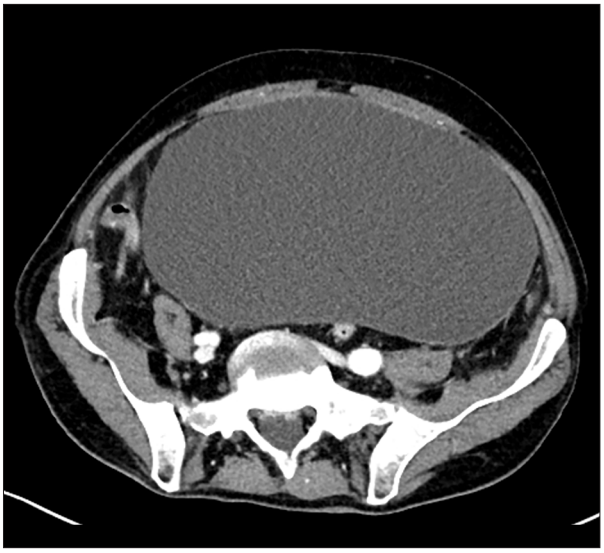


Figure 1. Axial computed tomography imaging of the abdomen and pelvis showing massive distention of the patient's bladder.



Figure 2. Coronal computed tomography imaging of the abdomen and pelvis showing bladder dome well above the umbilical region (red star).

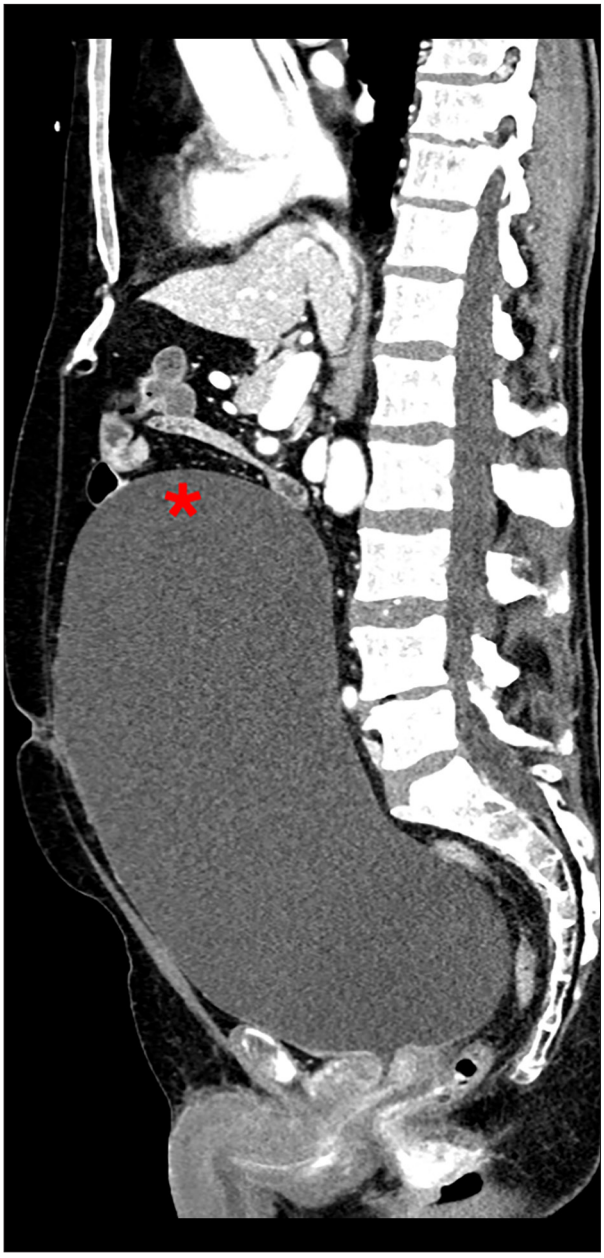


Figure 3. Sagittal computed tomography imaging of the abdomen and pelvis showing bladder dome well above the umbilical region (red star).

approximately 10 minutes. The patient was then observed in the ED for approximately 2.5 hours, during which vital signs remained stable and the heart rate improved from 112 to 96 beats per minute, with a blood pressure of 112/84 mm Hg. Urine output during this observation period was not separately recorded, and no repeat renal function or electrolyte measurements were obtained prior to discharge.

The patient was deemed stable for discharge from the ED with the urinary catheter in place, and Urology follow-up in

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the outpatient setting was scheduled. No additional medications were prescribed upon discharge from the ED. Urine cultures were negative for any bacterial growth. In follow-up in the outpatient setting 4 days after discharge, the indwelling foley catheter was removed for a trial of void, but the patient failed the trial and returned to the urology clinic the same afternoon for replacement of the indwelling catheter. A basic metabolic panel obtained at that visit showed a serum creatinine of 0.77 mg/dL, improved from 0.97 mg/dL at presentation, with a potassium of 3.3 mmol/L and a blood urea nitrogen of 7 mg/dL; no post-obstructive renal injury was evident. Therefore, Urology recommended continued indwelling catheterization rather than clean intermittent catheterization, a decision based on his comorbid burden and ongoing treatment for metastatic pancreatic cancer, until further definitive measures could be pursued. The catheter has since been exchanged monthly in the home setting. No further urologic follow-up occurred, as the patient's subsequent course was dominated by multiple hospitalizations related to his pancreatic cancer. At the last documented follow-up available for this report, an oncology visit approximately 11 weeks after the index ED presentation, the patient remained dependent on an indwelling catheter and had not undergone a further trial of voiding. The patient did not require additional urologic studies or other inpatient hospital treatment directed at his voiding dysfunction during this interval.

Discussion

Acute urinary retention (AUR) is defined as the inability of the bladder to voluntarily pass urine [9,10]. AUR is the most common urologic emergency and is seen predominantly in males [9,10]. Causes of AUR include obstruction, as well as neurologic, infectious, and inflammatory causes [10]. The most common cause is obstructive with up to 53% of cases due to BPH [10]. The patient in this case report had a history of BPH status after transurethral aquablation. Aquablation has a 98% success rate of spontaneous voiding at 5 months postoperatively, so this patient may have been in the procedure failure population [11].

Diagnosis of AUR is made by physical exam and measuring post-void residual (PVR). PVR can be measured through urinary catheterization, bladder scan, or ultrasound [12]. Bedside point-of-care ultrasound (POCUS) permits rapid estimation of bladder volume and can identify marked distention within minutes of clinical suspicion arising, well before cross-sectional imaging can be obtained; handheld devices can over- or underestimate volume, particularly at large bladder volumes, so the catheterized volume remains the definitive measurement [13]. In our patient, CT was performed because the differential diagnosis also encompassed thoracic and oncologic pathology that bedside ultrasound would not address, and POCUS would not have substituted for that broader evaluation. There is no specific PVR volume threshold used to diagnose AUR, whereas CUR is defined by a PVR greater than 300 mL documented on 2 or more occasions and persisting for greater than 6 months [10,14]. Our patient did not meet these criteria: serial post-void residual measurements were not available, and the duration of his bladder overdistension could not be established from the available record. The episode is therefore best characterized as massive urinary retention of uncertain chronicity, and any reference to chronic retention in this report should be understood as a possible rather than an established interpretation.

While normal functional bladder capacity is approximately 400 to 600 mL [1], volumes up to 5000 mL have been rarely documented [5,15]. The patient in our case study had 3.8 L removed via urinary catheterization, well exceeding the bladder's normal volume. In a study by Purohit et al examining voiding dysfunction and large bladder volumes, the primary cause was bladder outlet obstruction (48% of cases), impaired detrusor contractility (11%), absent detrusor contractility (24%), and normal detrusor pressure/uroflow study (17%) [5]. Other diagnoses associated with voiding dysfunction and large bladder capacity included BPH and neurologic disorders in men and pelvic organ prolapse, stress incontinence, and neurologic disorders in women [5].

Symptoms of urinary distention include pelvic pain, urinary incontinence, bladder spasms, pain during urination, or lower frequency of urination [16]. However, symptoms can be silent or vague, such as generalized weakness [17]. A case report by Morrissey et al demonstrates an ST elevation myocardial infarction (STEMI) mimic in a patient with significant urinary retention [8]. Our patient presented with shortness of breath and upper abdominal symptoms due to bladder distention. Presumably, the significant distention of the bladder placed pressure on the patient's intra-abdominal organs up toward the diaphragm, thus leading to dyspnea and early satiety. Despite holding nearly 4 liters of urine—extending the bladder dome well above the umbilicus, as shown in **Figures 1-3**—or patient reported no difficulty urinating. Additionally, he successfully provided a voluntary urine specimen on arrival and exhibited completely normal serum creatinine and upper urinary tract imaging.

When bladder volumes exceed normal capacity, damage can occur throughout the urinary system, including hydronephrosis, hydroureteronephrosis, and impaired glomerular filtration rate [2,18-20]. Bladder compliance—the change in volume per unit change in detrusor pressure—determines whether rising volume translates into rising intravesical pressure; normal compliance exceeds 40 mL/cm H_2O [20-22]. Where filling is gradual, the detrusor accommodates progressively larger volumes with little rise in storage pressure, which plausibly explains how our patient retained nearly 4 liters without rupture or upper tract injury. Notably, his imaging demonstrated

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neither hydronephrosis nor hydroureter and his serum creatinine was normal; these reassuring upper tract findings should not be taken to exclude clinically significant retention.

The bladder wall has stretch receptors that signal the need to urinate when the bladder becomes distended, signaling to the brain the urge to urinate [23]. Sustained overdistension can blunt this signaling and impair detrusor contractility through reduced wall perfusion with sensory and motor nerve injury [20,24]. This may account for our patient's striking absence of voiding problems despite a bladder dome extending above the umbilicus, and for his subsequent inability to void after the catheter was removed.

Spontaneous, non-traumatic rupture accounts for less than 1% of bladder ruptures and most often occurs intraperitoneally at the dome, the weakest portion of the bladder wall [23,25]. In a systematic review by Reddy et al, pelvic radiation and alcohol intoxication were the factors most frequently associated with spontaneous rupture; alcohol increases sphincter resistance and alters central sensory processing [26]. Neither factor applied to our patient, and experimental models indicate that rupture requires substantial intravesical pressure rather than large volume alone [27].

Treatment of massive bladder distention is prompt decompression by urinary catheterization, as was performed in this case. Rapid rather than gradual decompression, such as clamping the urinary catheter, is supported by the available evidence [28], and the recognized complications of hematuria and post-obstructive diuresis are reported in 2% to 16% and 0.5% to 52% of cases, respectively, but are rarely clinically significant [29]. Post-obstructive diuresis—urine output of at least 200 mL/hour for 2 consecutive hours, or more than 3 L over 24 hours following relief of obstruction—did not develop in our patient [29]. He tolerated rapid decompression of 3.8 L without hypotension or clinically significant hematuria, but required ongoing catheterization for his retention on follow-up.

Conclusions

Massive urinary retention can present with atypical symptoms resulting from mass effect rather than classic lower urinary tract problems. This case demonstrates that severe bladder overdistension can manifest as dyspnea, upper rather than lower abdominal discomfort, and early satiety while renal function and urinary symptoms remain preserved. Emergency clinicians should maintain a high index of suspicion for urinary retention in patients with unexplained abdominal or cardiopulmonary concerns, particularly in those with prior bladder outlet obstruction or medications that can impair bladder emptying. Prompt diagnosis and bladder decompression produced rapid symptomatic improvement in our patient. Hydronephrosis, permanent bladder dysfunction, and spontaneous rupture are recognized risks of sustained overdistension that motivate timely management; none occurred in this case, although their absence here does not itself demonstrate that decompression prevented them. Our patient remained dependent on an indwelling catheter at the last documented follow-up, and the available follow-up does not establish whether his bladder dysfunction was temporary or permanent.

Institution Where Work Was Done

Mayo Clinic, Jacksonville, FL, USA.

Patient Permission

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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