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Distended Bladder Presenting with Altered Mental Status and Venous Obstruction

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ABSTRACT

Background: New onset or acute worsening of bilateral lower extremity swelling is commonly caused by venous congestion from decompensated heart failure, pulmonary disease, liver dysfunction, or kidney insufficiency. A thromboembolic event, lymphatic obstruction, or even external compression of venous flow can also be the culprit.

Case Report: We report the case of an 83-year-old male with a history of myelodysplastic syndrome that progressed to acute myeloid leukemia, bipolar disorder, and benign prostatic hypertrophy. He presented with altered mental status and new onset lower extremity edema caused by acute bladder outflow obstruction. Computed tomography of the abdomen and pelvis showed the patient's distended bladder compressing bilateral external iliac veins.

Conclusion: Insertion of a Foley catheter resulted in several liters of urine output and marked improvement in his lower extremity edema and mental status a few hours later. Our extensive workup failed to reveal a cause of the patient's acute change in mental status, and we attributed it to a concept known as cystocerebral syndrome.

INTRODUCTION

Among the many causes of bilateral lower extremity swelling, common etiologies are liver disease, kidney disease, heart failure, pulmonary hypertension, chronic venous stasis, and side effects of medication such as dihydropyridine calcium channel blockers. A distended urinary bladder compressing the inferior vena cava or external iliac vein can also produce bilateral pedal edema and is easily misdiagnosed. We report the case of a patient who presented with altered mental status and new onset lower extremity edema resulting from compression of the external iliac veins by a distended bladder.

CASE REPORT

An 83-year-old male with a history of myelodysplastic syndrome with progression to acute myeloid leukemia (AML), idiopathic thrombocytopenic purpura, adrenal insufficiency, a questionable history of dementia, bipolar disorder, and benign prostatic hypertrophy (BPH) presented to the emergency department with altered mental status. His daughter found him walking around his apartment complex in an agitated and confused state. The patient lived alone and was known to be fully functional and independent, although he likely had an underlying history of dementia with worsening intermittent confusion in the several months prior to admission. He did not have a history of drug or alcohol abuse. His home medications included tamsulosin, prednisone, and metoprolol tartrate. The patient was not on any medications for bipolar disorder, and it was not known if he had ever been hospitalized for psychiatric disease. He also had a history of chronic urinary retention caused by BPH with previous postvoid residuals as high as 600 mL of urine. Recently, his BPH had been medically managed with postvoid residuals of $<$200 mL of urine.

On admission, the patient's vitals were within normal range, and his body mass index was 22. His jugular venous pressure was 6 cm H_2O, and his lungs were clear to auscultation. His S_1 and S_2 sounds were normal, and no murmurs were heard. He had new onset moderate lower extremity edema that extended from his feet to his sacrum. He had a mildly distended and nontender abdomen with no suprapubic fullness. His prostate was also mildly enlarged without nodularity. The patient was alert but confused and agitated and could not answer questions in full sentences.

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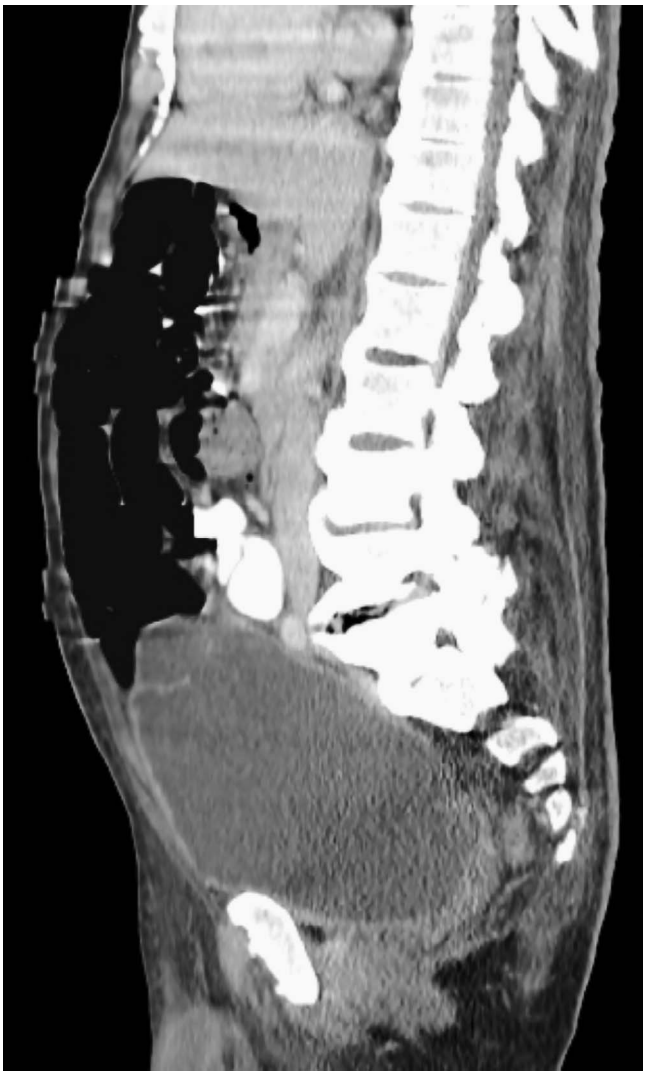

Figure 1. Computed tomography sagittal image of the abdomen and pelvis (with contrast) reveals marked distention of the bladder.

The altered mental status workup revealed normal results for complete blood count, complete metabolic panel, B_{12} levels, and thyroid-stimulating hormone. Urinalysis was normal. Urine and serum toxicology screens were negative. His electrocardiogram demonstrated normal sinus rhythm, and his troponin level was normal. Chest radiographs did not reveal an acute cardiopulmonary process. Computed tomography (CT) of the head did not show an acute process. Magnetic resonance imaging of the patient's brain was also negative for an acute event and showed no signs of metastatic disease. Abdominal radiograph showed prominent gas-filled loops of the small and large bowel, concerning for an ileus. Bilateral lower extremity ultrasound was negative for deep vein thrombosis.

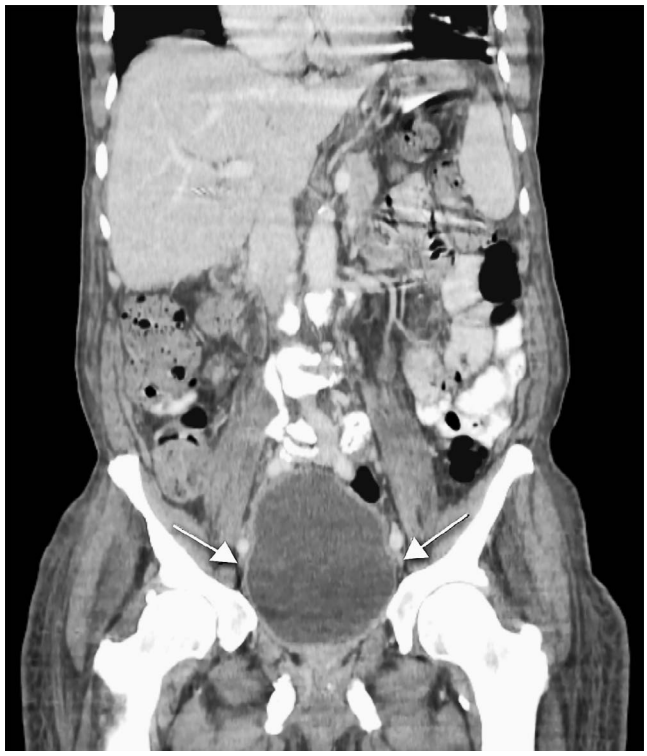

Figure 2. Coronal view shows compression of the bilateral external iliac veins (arrows).

A cardiac or pulmonary cause of the patient's new onset lower extremity swelling was quickly ruled out based on normal results of a B-type natriuretic peptide blood test and a recent transthoracic echocardiogram from a previous admission that showed normal left ventricular function, an ejection fraction of 60%, and an estimated systolic pulmonary artery pressure of 21 mmHg.

CT performed after intravenous administration of iodinated contrast demonstrated marked distension of the bladder with the dome extending to the level of the umbilicus (Figure 1). Sagittal imaging to the left of midline demonstrated a few urothelial trabeculations and septations near the dome, sequelae of chronic bladder outlet obstruction. Axial and coronal imaging demonstrated symmetric compression of the external iliac veins as they course past the bladder (Figures 2 and 3).

A Foley catheter was placed that drained 3 liters of urine. Within hours of placement of the catheter, the patient's lower extremity edema and mental status significantly improved. Since admission, the patient had been incontinent and wearing a diaper; thus urine output was not accurately measured, and his acute urinary retention (AUR) was only revealed by CT. The workup conclusion was that his AUR was caused by BPH. After resolution of his symptoms, the patient was discharged home with a urethral Foley catheter

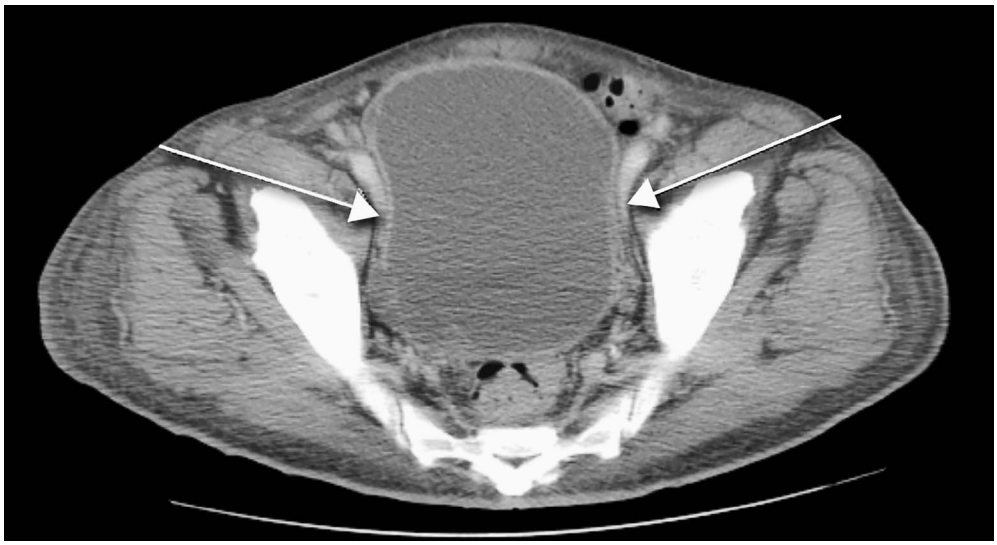

Figure 3. Axial view shows the distended bladder compressing the iliac veins (arrows).

and instructed to continue tamsulosin. He was also instructed to follow up with urology for a spontaneous voiding trial in 2 weeks. The patient did not have the opportunity to do a voiding trial without a catheter. His health quickly deteriorated because of his underlying AML, and he died.

DISCUSSION

AUR classically presents with abdominal pain and the inability to void. New onset lower extremity swelling caused by venous obstruction from a distended bladder is uncommon and was first reported in 1960.[1] Other case descriptions followed.[2-4] More common causes of new onset lower extremity swelling include heart failure, cirrhosis, proteinuria, kidney failure, bilateral lower extremity deep vein thrombosis, thrombosis of the inferior vena cava, or even vasodilating medication side effects. Extrinsic compression by a tumor, aneurysm, or retroperitoneal hematoma should also be considered. Additionally, as presented here, a distended bladder can produce critical compression.

We concluded the patient's altered mental status was caused by his distended bladder, a condition that has been called cystocerebral syndrome. Altered mental status associated with AUR was first reported in 1990.[5] Several case reports and series followed, and most patients were elderly men presenting with AUR secondary to BPH.[6-8] Interestingly, most of the patients also had an underlying psychiatric disorder or preexisting cognitive impairment. The pathophysiology of cystocerebral syndrome is not fully understood, but a hypothesis is that increased bladder wall tension induces sympathetic tone and catecholamine release that trigger an acute change in mental status.[9]

Of the several causes of AUR, BPH is the most common etiology in the elderly male. Other causes include medication side effects, neurologic disease, urethral stricture, and urolithiasis.[10] Evaluation of a patient with AUR starts with a good history focusing on previous episodes of retention, cancer, surgery, radiation therapy, trauma, neurologic disease, and medication or drug use. On abdominal examination, a distended bladder is palpated as a suprapubic fullness with dullness to percussion. A rectal examination with assessment of prostate size is also necessary. Look for abnormal laboratory findings such as worsening kidney function, hematuria, leukouria, or bacteriuria. Imaging studies—bladder scan, renal ultrasound, or CT scan—may be necessary to determine the cause of AUR.

Initial management of AUR is bladder decompression that is usually successful with a urethral or suprapubic catheter.[11] The catheter is generally left in place for several days prior to a trial without catheter. As with patients who present with BPH, an alpha adrenergic receptor antagonist should be initiated at the time of catheterization because alpha blockers have been shown to increase the success of voiding without a catheter.[11-13] Five-alpha reductase inhibitors can decrease the incidence of AUR but do not increase the success of trials without a catheter and thus are not used to treat AUR caused by BPH.[14] Multiple failed trials without a catheter while being treated with an alpha receptor blocker often lead to surgical intervention.[15] Urodynamic evaluation is generally performed prior to surgical intervention to determine whether retention is directly related to outlet obstruction with concomitant elevation in bladder pressures or to an inefficient bladder muscle.

CONCLUSION

The manifestations of urinary retention are diverse and can be confusing for the physician. Although the condition is uncommon, clinicians should maintain a high index of suspicion for bladder distention as a cause of venous obstruction, especially in elderly patients presenting with altered mental status in whom it is difficult to assess or recognize symptoms of urinary retention.

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