Bilateral hydronephrosis and acute kidney injury secondary to pelvis lipomatosis

Wesley Baas, MD, Blake O'Connor, Ahmed El-Zawahry MD
Division of Urology, Southern Illinois University School of Medicine, Springfield, Illinois, USA

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A 57-year-old African American male presented with vague abdominal pain and bilateral flank pain. The patient was found to have bilateral hydronephrosis and significant renal function impairment secondary to pelvic lipomatosis. Pelvic lipomatosis represents a clinically-diagnosed unique cause of ureteral obstruction and subsequent renal impairment. We present a case report of newly diagnosed pelvic lipomatosis, the clinical and imaging characteristics for diagnosis, and its conservative management with serial ureteral stent exchanges.

Key Words: lipomatosis, hydronephrosis, acute kidney injury, obesity, case study

Case report

A 57-year-old morbidly obese (body mass index = 57) African American male with a past medical history of rectal cancer status post abdominoperineal resection, obstructive sleep apnea, and atrial fibrillation presented to the emergency room with complaints of vague epigastric pain going to his umbilicus as well as bilateral flank pain. A basic metabolic panel showed a blood urea nitrogen (BUN) of 36 and a creatinine of 4.9 mg/dL.

The patient underwent renal ultrasound that showed new bilateral hydronephrosis. Ureters were unable to be evaluated because of body habitus. A foley catheter was placed and there was no clinical improvement. A subsequent computer tomography (CT) abdomen/pelvis redemonstrated bilateral hydronephrosis with dilation of the left ureter down to a fullness of presacral area with increased fat density in the perivesical and perirectal regions. The patient then underwent cystoscopy with bilateral retrograde pyelograms and bilateral ureteral stent placement.

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Address correspondence to Dr. Wesley Baas, SIU Medicine – Division of Urology, PO Box 19665, Springfield IL 62794-9665 USA

Cystoscopy examination demonstrated an elongated dilated prostatic urethra, wide open bladder neck, and the bladder was small in capacity (only holding 100 mL). Retrograde pyelograms at that time demonstrated hydroureter along the entire course of the ureters bilaterally. Ureteral stent placement was particularly difficult because of significant J-hooking of the ureters.

Intraoperative cystogram was notable for an "inverted pear" shaped bladder seen with pelvic lipomatosis (PL), Figures 1 and 2. The patient's acute kidney injury subsequently resolved after stent placement. Ensuing CT scans have continued to demonstrate increased perivesical and perirectal fat, Figure 3. Given the clinical and radiographic findings, the patient was diagnosed with PL causing acute kidney injury. The patient has since been managed with serial stent changes every 3 to 4 months and returned to his baseline renal function (Cr = 1.6mg/dL) with significant improvement of bilateral hydronephrosis.

Discussion

First described by Engels in 1959, and later refined into a clinical-radiologic entity by Fogg and Smyth in 1968, PL is a rare disease characterized by abnormal deposition of mature, non-encapsulated fat tissue in the pelvic cavity.[1] PL is most often found in the perivesical and perirectal spaces and subsequently spreading upward to the abdominal cavity.[2]

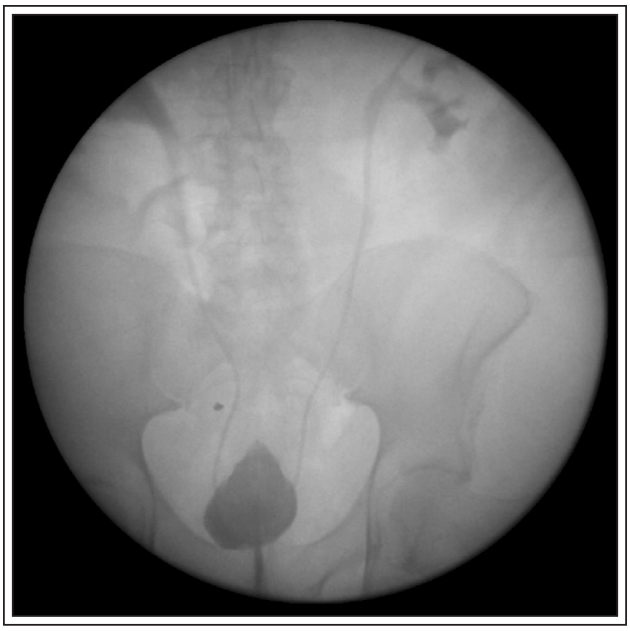

Figure 1. Cystography demonstrating "inverted pear" shaped bladder indicative of pelvic lipomatosis.

The etiology of PL is currently unknown, and this may be in part to its rarity. It has been historically reported to be of an incidence between 0.6-1.7 per 100,000 hospital admissions in the USA.[3] However, Zhang et al report

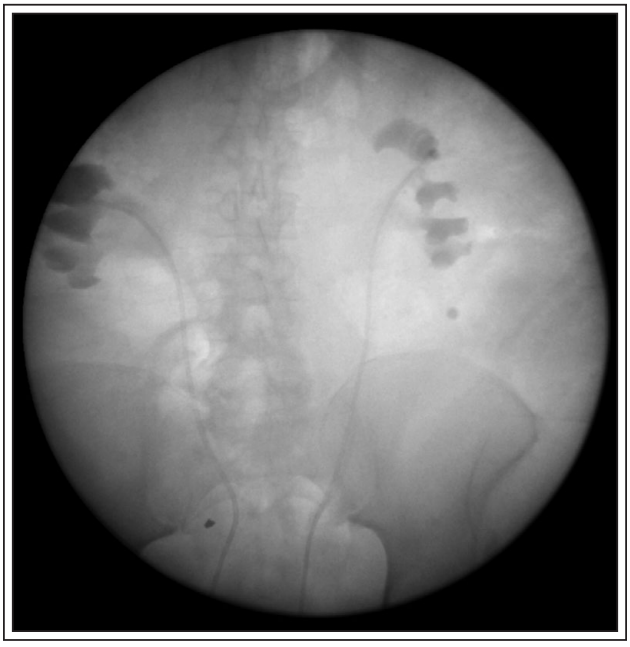

Figure 2. Retrograde pyelogram at time of stent placement again showing medially displaced ureters and bilateral hydronephrosis.

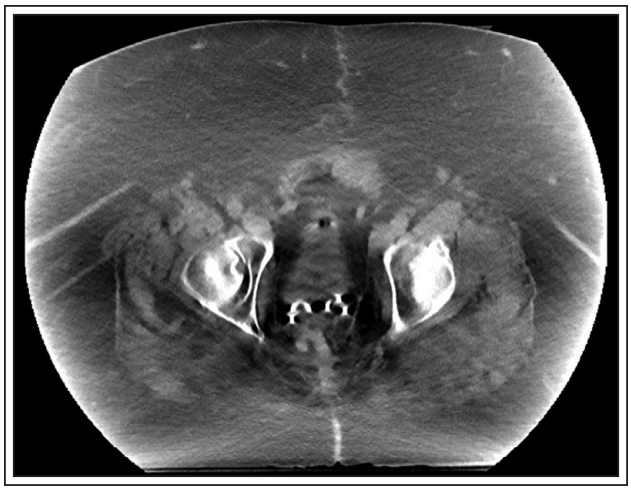

Figure 3. CT abdomen/pelvis after stent placement continues to demonstrate a small pear-shaped bladder surrounded with increased perivesical and perirectal fat consistent with pelvic lipomatosis.

that since PL is often discovered incidentally in elderly men undergoing routine or unrelated evaluation, the true disease prevalence is likely underestimated.[2] It has been suggested that this disease occurs primarily in men (as high as an 18:1 ratio), with a predominance in African-American individuals, and is associated with obesity.[4,5] While Heyns demonstrated that 65% of PL patients were clinically obese and it has been shown that weight gain or loss can contribute to disease development or improvement, respectively, obesity is not the only factor responsible for PL pathogenesis.[4,6] In fact, in the Japanese literature, there were no patients with a body mass index (BMI) of greater than 30 and in Zhang's study of 32 PL cases only two patients presented with obesity (BMI > 30). Additionally, the narrower male bony pelvis may increase the propensity for fat deposition. Chronic inflammatory states due to recurrent lower urinary tract infections may also lead to fatty tissue development. Endocrine dysfunction and genetic predisposition have been implicated as well.[6,7] Two distinct patient populations with PL have been defined in the literature. One group consisting of young, obese patients with nonspecific pelvic symptoms shows an increased risk of hydroureteronephrosis and renal failure. The second group consists of elderly men whose PL is discovered incidentally and have little risk of significant disease progression.[2,7]

Although described as benign, PL can often cause a number of symptoms, most notably upper urinary tract obstruction by fatty tissue deposition with subsequent hydronephrosis and renal failure. Some

patients will present with flank and lower abdominal pain, lumbago, low-grade fever, recurrent UTI, frequent urination, dysuria, and hypertension, while others will not experience lower urinary tract obstruction or related symptoms.[6] This benign entity can also cause symptoms of constipation by rectal compression, as well as lower extremity edema from compression of pelvic vasculature.[5] In a small study, Crane and Smith suggested that 40% of patients with obstructive uropathy secondary to PL progress to renal failure within 5 years.[8]

In diagnosing PL, CT scans remain the gold-standard. Upon initial clinical suspicion, plain abdominal x-ray may reveal perivesical areas of increased radiolucency. However, contrast-enhanced CT will definitively demonstrate bladder and rectosigmoid displacement by homogenous, non-enhancing tissue with low attenuation values.[7] The adipose tissue surrounding the bladder and rectum has identical density to that of subcutaneous fat.[6] Quantitative measurement of the volume of pelvic fat by 3D imaging, evidence of hydronephrosis due to vesicoureteral obstruction, and changes in bladder morphology may reinforce the radiologic diagnosis of PL.[5] A pear- or banana-shaped bladder on CT urography are widely accepted as highly specific (100%), but poorly sensitive (40.6%) morphologies in predicting and diagnosing PL. Oval, triangular, and irregular shaped bladders are inconclusive findings. On cytology and histology, roughly 75% of PL patients are found to suffer from concomitant proliferative cystitis.[5,6] Such proliferation of the bladder mucosa may be due to lymphatic and venous stasis secondary to excess fat deposition and is highly regarded as a potential precursor to adenocarcinoma of the bladder.

Treatment options in PL vary and often rely solely on symptomatic therapies. Conservative treatment modalities have been implemented with variable, but generally poor results. These options include weight loss, long term antibiotics, steroid administration, and radiotherapy.[5-7] A more radical approach to PL treatment – complete surgical excision of pelvic fat – has mixed reviews. Some studies claim that fat removal is inadvisable due to procedural difficulty, fat adherence to important anatomical structures, indistinct surgical planes, and general ineffectiveness.[9] Conversely, some report that complete resection of pelvic mass with bladder sparing, though difficult and time-consuming, is quite possible and response to surgery can be good.[5,9] In patients with severe obstructive symptoms, worsening hydronephrosis and renal failure, ureteral reimplantation, nephrostomy, ureterostomy, or conduit are necessary considerations.[9] Klein, reports that urinary diversion was ultimately required in 39% of PL patients during a 7.5 year follow up period.[10]

The patient we have presented represents a prototypical patient that may present with urinary obstruction secondary to PL. Being an obese African-American male who presented with vague abdominal symptoms including flank pain places him into the first group discussed of young, obese males with a predilection for hydroureteronephrosis and subsequent renal failure. CT imaging was concordant with the diagnosis of PL with significant amount of adiposity surrounding the bladder and rectum. He also displayed the characteristic "inverted pear" shaped bladder on cystogram. While other authors have done very invasive measures such as urinary diversion, we have shown that these patients can be successfully managed conservatively with serial stent exchanges.

In conclusion, PL represents a rare, benign disease that can often result in significant complications including urinary obstruction and renal failure. Diagnosis is clinical in nature with CT remaining the gold-standard for evaluation of this disease. There is currently no definitive cures or therapies, and will likely be tied to case reports for the management of this disease due to its rarity. In this case report we present a male who presented in renal failure secondary to obstructive PL who has been successfully managed with recurrent ureteral stent exchanges. □

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