A urinoma results when urine extravasates from the kidney, ureter, bladder, or urethra. Urinomas may be unilateral, bilateral, symptomatic, or asymptomatic, and can manifest as a confined or encapsulated collection, or as free fluid mimicking ascites. The causes of urinomas may be divided into either obstructive or non-obstructive. Obstructive causes include pregnancy, ureteral calculi, pelvic masses, posterior urethral valves, congenital anomalies, post-radiation scarring, enlarged lymphatic glands, retroperitoneal fibrosis, and prostate enlargement. Non-obstructive causes include external trauma to the kidneys, the urinary collecting system, or injury during pelvic, gynecological, retroperitoneal, or genitourinary surgery. Although all causes are rare, in adults, trauma to the urinary system is the most common cause, while obstructive causes, specifically ureteral calculi, are less likely.

Encapsulated collections of extravasated urine in the subcapsular or perirenal space, are called subcapsular and perirenal urinomas respectively. The most accepted mechanism of this type of lesion is pyelosinus backflow of urine, that can occur with intrapelvic pressures rises greater than 35 cm H₂O, with subsequent rupture of caliceal fornices. Subcapsular urinomas consist of urine in between the kidney’s parenchyma and capsule. In contrast, perirenal urinomas consist of a urine collection in between Gerota’s fascia and the capsule.

Previously, urinomas were thought to have a protective effect on renal function but most recent studies have questioned this concept because some patients show impaired renal function in the kidney ipsilateral to the urinoma. Patil et al. showed that there was no difference in renal function in patients with urinoma, ascites and controls. In this report we discuss a case of an urinoma due to ureteral calculi and present a brief review of the literature.

CASE PRESENTATION
Our case involved a 55 year old female with type 2 diabetes, hypertension, and stage 4 chronic kidney disease. She presented to the hospital with complaints of fatigue, fever, chills, mild right-sided flank pain, nausea, vomiting and shortness of breath for over one week. She had recently been hospitalized in Ciudad Juarez, Mexico where she was treated with antibiotics and analgesics for two days. Upon presentation to our facility, she denied having any abdominal pain, dysuria, diarrhea, or leg edema.

She was previously on losartan 50 mg/day, spironolactone 25 md/day, furosemide 40 mg/day, nifedipine 30 mg/day, ferrous sulfate 325 mg/day, erythropoietin 4000 units twice a week, and insulin NPH 15 units/day. The patient did not use tobacco products, alcohol or illegal drugs.

On physical examination the temperature was 36.5, pulse 55, blood pressure 120/56 mmHg, respiratory rate 11. The skin was pale. There were decreased breath sounds at both bases. There was mild costovertebral angle tenderness on the right side. The remainder of the physical examination was normal.

Laboratory studies upon admission: Hemoglobin 7.8 g/dL, hematocrit 23.5%, leukocytes 15500/uL with 88% polymorphonuclears, platelets 242,000/uL; sodium 132 mEq/L; potassium 7.2 mEq/L; chloride 107 mEq/L; bicarbonate 11 mEq/L; blood urea nitrogen (BUN) 82 mg/dL; serum creatinine 7.8 mg/dL; glucose 212 mg/dL; corrected calcium 9.24 mg/dL; albumin 2.2 g/dL. The urine analysis showed: small blood, protein >300 mg/dL, glucose 100 mg/dL, WBC 5-10, RBC 0-3, bacteria 2+, leukocyte esterase moderate, nitrite negative. Extended spectrum beta-lactamase producing Escherichia coli grew in the urine and in the blood cultures.

Treatment for pyelonephritis was started with parenteral meropenem and hemodialysis was initiated. On the fourth hospital day, the patient developed severe right-sided flank pain and gross hematuria. On examination she then had tenderness to palpation in her right lower and upper quadrant as well as increased tenderness over the right costovertebral angle. She was afebrile, but her leukocytosis increased to 22,900/uL despite antimicrobial therapy. Urine analysis was then remarkable for too numerous to count red and white blood cells; there were no crystals. Renal ultrasonography (see Image 1), performed on suspicion of possible nephrolithiasis, revealed mild to moderate right hydronephrosis, and right subcapsular & perinephric fluid collections. Computed tomography (see Image 2) demonstrated a right renal subcapsular fluid collection with continuity with a fluid collection in the psoas muscle; mild to moderate right hydroureteronephrosis and a 7.2 x 8.2 mm calculus in the right ureterovesical junction. The findings were interpreted as consistent with an urinoma secondary to the obstructive right ureterovesical nephrolith.

The patient then underwent percutaneous drainage of the fluid collections as well as cystoscopy with stone removal and ureteral stent placement. The perirenal and subcapsular collections, the flank pain, and the gross hematuria all subsequently resolved.

DISCUSSION
Urinomas forming due to obstruction from ureteral calculi are very rare. Calculi lead to a rise in intrapelvic pressures, pyelosinus backflow, and subsequent rupture of caliceal fornices, which result in extravasation of urine. Most commonly, urine leaks into the subcapsular space or into the perirenal space within Gerota’s fascia.

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With extensive extravasation, urine may cross the midline, travel inferiorly, superiorly, or through lymphatic vessels. If it extends inferiorly, it travels along the iliopsoas compartment below the inguinal ligament to the soft tissues of the thighs, pelvis, buttocks, or scrotum or into the peritoneum. There are few reports in the literature with stone-related urinomas.6

The typical presentation of a patient with a stone-associated urinoma resembles that of a ureteral stone itself. Most patients complain of nausea, vomiting, and flank pain, similar to our patient. Other symptoms include urgency, fever, ileus, and pain in the abdomen and/or genitals. Fluid analysis shows a significantly higher creatinine level and a lower glucose concentration relative to the serum. Urinalysis can show hematuria and pyuria.4

The initial evaluation of a patient suspected of having a urinoma includes renal ultrasonography, followed by an abdomen and pelvis CT without contrast. The CT is better able to demonstrate the relationship between the urinoma and the kidney, the ureter, and the fascial planes.7

The initial management of a urinoma is conservative. If the size of the urinoma does not decrease with conservative management after several days, a patient may need an intervention. Initially, a percutaneous catheter under CT or ultrasound can be placed in the most gravity-dependent portion of the urinoma, and the output can be monitored. A sample of the fluid should be cultured and the patient should begin empiric antimicrobial therapy until culture results are available. If there is decreased output, the catheter may be removed and follow-up can be done with ultrasound to ensure complete resolution. If the drainage volume does not decrease, or if it increases, further intervention will be necessary, because persistent fluid drainage indicates a continuous leak from the collecting system. Placement of an anterograde nephrostomy with or without a ureteral stent or a nephroureterostomy catheter will be necessary.8 In the case of our patient, she had percutaneous catheter drainage and ureteroscopy, stone removal, and stent placement with complete resolution of her urinoma four days later.

Urinomas can cause serious complications, requiring immediate management until their complete resolution. Potential complications include hydronephrosis, paralytic ileus, electrolyte imbalances, abscess formation, and sepsis. The practicing clinician should have a high index of suspicion for this rare but serious entity to be able to promptly diagnose and manage this condition.

**REFERENCES**


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