Bilateral stones as a cause of acute renal failure in the emergency department

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INTRODUCTION

The incidence of a unilateral uterovesicular junction obstruction secondary to a stone is reported at 20% in the literature.[1] Patients may present with the classic symptoms of renal colic and hematuria. Others may be asymptomatic or have atypical symptoms. Possible confirmatory radiologic tests include abdominal plain film, intravenous pyelography (IVP), ultrasonography, and most commonly, non-contrasted helical CT scan, which is the test of choice. Urgent urologic consultation is warranted in patients with urosepsis, acute renal failure, anuria, and/or unyielding pain, nausea, or vomiting. There are few case reports regarding the incidence of bilateral ureteral calculi.[2] Cases of bilateral ureteral calculi are uncommon, and cases resulting in such obstruction are rare.

CASE REPORT

A 47-year-old man with lumbar pain presented to the emergency department. Two days before admission to the emergency department, malaise developed with diffuse lower lumbar pain. His history included smoking 1 pack of cigarettes for 20 years (but not in the past 2 years), diabetes mellitus, hypertension, and hypercholesterolemia. His medications included metformin, losartan, simvastatin and aspirin. He had no recent changes in his medication regimen. He had no family history of vascular disease, hypertension and coronary artery disease and/or stroke. He reported no recent use of non-steroidal anti-inflammatory drugs or antibiotics. Two months before the admission to the hospital, he had had a sudden, dull pain in the left flank that radiated to the left groin and was associated with mild hematuria. He was evaluated in an emergency department and diagnosed as having renal colic; the pain was relieved after the administration of dextroketoprofen, and he was discharged home asymptomatic, no additional analgesic was required. On arrival to the emergency department (ED) he complained of intermittent, mild, bilateral flank pain that was unrelated to physical activity. He reported...
drinking 1 L to 2 L of fluid daily the previous day. His
urinary output was diminished and concentrated and in
the previous 24 hours the total absence of urine output
was recorded. Gross or microscopic hematuria was not
present on admission. Physical examination showed
acceptable general condition, no fever, and normal
blood pressure (125/80 mmHg), heart rate 87 beats per
minute, temperature 36.9 °C, costovertebral angles not
tender on palpation, etc. Laboratory tests at admission
showed glucose 146 mg/dL, GOT 78 IU/L, GPT 45 IU/
L, bilirubin 0.6 mg/dL, LDH 450 U/L, C-reactive protein
46, CK 347 U/L, creatinine 4.5 mg/dL, urea 85 mg/dL,
Na 132 mmol/L, K 5.8 mmol/L, pH 7.31, and bicarbonate
21 mmol/L. Urinalysis showed a pH of 6.5, urine tested
positive (+++) for occult blood; 200 red blood cells;
proteins negative; glucose negative; and leukocytes
0. Prothrombin and partial-thromboplastin times were
all within normal ranges. The patient was admitted to
the observation unit, an indwelling urethral catheter
was inserted yielding no urine. Given these findings,
the emergency physicians performed a bedside renal
ecography showing bilateral moderate hydronephrosis
(Figure 1).

The causes of acute renal failure in this patient were
considered according to the diagnostic paradigm based
on prerenal, intrinsic renal and postrenal factors. The
absence of prerenal factors (those that reduce blood flow
to the kidneys) and intrinsic renal disease with a history
of physical examination and laboratory studies that
rule out shock, rapidly progressive glomerulonephritis,
cortical necrosis, and acute tubular necrosis, as the
patient did not have hypotension or inadequate oxygen
delivery, systemic symptoms, edema, or any other signs
of vasculitis. The patient was afebrile, and microscopy
showed no schistocytes, thus ruling out hemolytic-
uremic syndrome and thrombotic thrombocytopenic
purpura. Complete urinary tract outlet obstruction is the
most common cause of anuria and should be investigated
first as in this patient. According to the hospital protocol,
a helical CT of the abdomen and pelvis was performed
to show the presence of several stones, three in the right
kidney (lower, middle and upper pole), all below 1 cm,
one in the right medial ureter of 1.5 cm and other in the
left distal ureter, 0.5 cm, the latter producing bilateral
moderate hydronephrosis. Furthermore, several simple
bilateral renal cysts were presented, empty bladder and
prostate of normal size (Figures 2–4)

Given these results we requested an urgent review
by the Department of Urology because of acute renal
failure of obstructive etiology (bilateral renal lithiasis),
proceeding to transfer the patient to the operating room,
where two ureteral double-J stents were inserted for
urinary tract decompression. A radiograph after the
procedure showed the stent in good position (Figure
5). Subsequent urine output was 1 500–2 000 mL on
both sides and serum creatinine decreased to a normal
range of 0.6 mg/dL. The patient subsequently underwent
successful shock wave lithotripsy (SWL) for dissolution
of calculi. Stone analysis revealed a predominance of
calcium. Serum levels of calcium, phosphorus, calcitriol
and parathyroid hormones were all normal, ruling
primary hyperparathyroidism as a cause of lithiasis out.
The patient has been reported asymptomatic in a follow-
up.

**DISCUSSION**

Postrenal causes of acute renal failure occur when
both urinary outflow tracts are obstructed or when one tract
is obstructed in a patient with a single functional kidney.
Obstruction is most common due to prostatic hypertrophy,
cancer of the prostate or cervix, or retroperitoneal
 disorders and often presents in the outpatient setting.[1,3,4]
A neurogenic bladder can result in functional obstruction.
Other less frequent postrenal causes of acute failure can
be intraluminal, such as bilateral renal calculi, papillary
necrosis, coagulated blood, bladder carcinoma, and
fungus, or extraluminal, such as retroperitoneal fibrosis,
colorectal tumor, and other malignant conditions.
Because postrenal causes are readily reversible, it is
imperative to exclude them.[5] Rapidly evaluation of
the patient in the emergency department demonstrated
bilateral renal stones as the cause of acute renal failure.

Urinary stone disease is commonly seen in 1 of 10
people over a lifetime. Symptoms include severe colicky
flank pain. Gross hematuria is present in 15 percent.[6]
Others may be asymptomatic or have atypical symptoms such as vague abdominal pain, acute abdominal or flank pain, nausea, urinary urgency or frequency, difficulty in urinating, penile pain, or testicular pain. The renal pelvis and ureter contain highly vascular epithelium that, when irritated, can cause bleeding. Blood clots may form and obstruct the flow of urine, which results in pain. Stones may contain either calcium or uric acid. A study by Wang et al. \[7\] conducted between February 2002 and December 2009 showed a prevalence of ARF of 0.72% in a series of 2,073 cases of ureteral stones and only five cases (33.3%, 5/16) were caused by bilateral ureteral stones. The study suggests that risk factors for developing ARF in ureteral stone patients are bigger stones, ureteral stones in patients with only one functioning kidney or pre-existing kidney disease, and bilateral ureteral stones.

Eighty percent of patients with nephrolithiasis form calcium stones, most of which are composed primarily of calcium oxalate or, less often, calcium phosphate. \[8,9\] The other main types include uric acid, struvite (magnesium ammonium phosphate), and cystine stones. The same patient may have more than one type of stone concurrently (e.g., calcium oxalate and uric acid). \[9\] The risk of nephrolithiasis is influenced by urine composition, which can be affected by certain diseases and patient habits. For calcium oxalate stones, urinary risk factors include hypercalciuria, hyperoxaluria, hypocitraturia, and dietary risk factors such as a low calcium intake, high oxalate intake, high animal protein intake, high sodium intake, or low fluid intake. The diagnosis of nephrolithiasis is initially suspected by the clinical presentation. The best imaging study to confirm the diagnosis of a urinary stone in a patient with acute flank pain is unenhanced, helical CT of the abdomen and pelvis. \[10\] That can detect both stones and urinary tract obstruction, and has become the gold standard for the radiologic diagnosis of stone disease. \[11-13\] CT cuts that are 3 to 5 mm in thickness are optimal for the detection of stones. The specificity of helical CT is nearly 100%, \[14,15\] thus, a positive study confirms the diagnosis of nephrolithiasis and patients should be treated appropriately. Furthermore, a CT scan in patients with a suspected kidney stone can

Figure 2. Left ureteral stone.

Figure 3. Right ureteral stone.

Figure 4. Bilateral renal stones.

Figure 5. Ureteral double-J stents.
define an alternate significant diagnosis. Low dose non-contrast CT imaging allows identification of renal and ureteral calculi with similar sensitivity and specificity as standard CT scans but with significantly reduced radiation dosage. However, low dose non-contrast CT may be less reliable for detecting small stones (less than 2 mm in diameter). When CT confirms the presence of a stone, a plain abdominal radiograph should be obtained to assess whether the stone is radiopaque. If CT is unavailable, plain abdominal radiography should be performed. Since 75% to 90% of urinary calculi are radiopaque such as calcium, struvite, and cystine stones, but radiolucent uric acid stones will be missed, small stones or stones overlying bony structures may be missed, and obstruction will not be detected. It is not the imaging modality of choice for initial diagnosis, although it may add to the sensitivity and specificity of an intravenous pyelogram. There is little role for an abdominal plain film when helical CT is available. Although ultrasonography has a high specificity (greater than 90%), its sensitivity is much lower than that of CT, typically in the range of 11%–24%. Thus, ultrasonography is not used routinely but is appropriate as the initial imaging test when colic occurs during pregnancy. It is sensitive for the diagnosis of urinary tract obstruction and can detect radiolucent stones missed on kidney, ureter and bladder X-ray (KUB); however, ultrasonography may miss small stones and ureteral stones. In the case of bedside renal ultrasound performed by emergency physicians, a recent study demonstrated that there was only a limited impact on the physicians’ clinical impression of patients with possible ureterolithiasis and the sensitivity of sonographic hydronephrosis was modest for detecting any ureteral stone, but much better for detecting a large stone. Urgent intervention is indicated in a patient with an obstructed, infected upper urinary tract, impending renal deterioration, intractable pain or vomiting, anuria, or high-grade obstruction of a solitary or transplanted kidney. Upper tract obstruction increases renal pelvic pressure, which reduces glomerular filtration and renal blood flow. Relief of upper tract obstruction may require either antegrade (percutaneous nephrostomy) or retrograde (cystoscopy and retrograde ureteral catheterisation) approaches. Urethral catheterisation can be performed immediately, but other techniques require planning. Close collaboration between nephrological, urological, and radiological services is required, and in many cases renal replacement therapy may be necessary before relief of obstruction can be achieved. A significant diuresis can complicate relief of complete urinary tract obstruction, through both appropriate (excretion of retained solute and water) and inappropriate mechanisms (tubular concentrating dysfunction). Severe polyuria is rare and requires careful management to prevent volume depletion and possible pre-renal impairment, or overzealous fluid resuscitation and a further drive to diuresis. Obstruction may result in an impaired distal tubular response to aldosterone, resulting in paradoxical hyperkalaemic acidosis when relieved. This usually resolves spontaneously. A small number will have permanent tubular damage and persistent salt wasting nephropathy. Approximately 5% of patients with renal stones have concurrent hyperparathyroidism. In addition, most stones in patients with hyperparathyroidism are composed of calcium oxalate. The levels of serum calcium, phosphate, calcitriol, and parathyroid hormone are helpful to diagnose this condition. Primary hyperparathyroidism should be considered in patients with recurrent renal stones especially calcium stones and a high-normal or elevated serum calcium level.

Funding: None.

Ethical approval: The study was approved by the Medical Ethics Committee of Hospital Valle de los Pedroches, Pozoblanco, Córdoba, Spain.

Conflicts of interest: The authors declare that there is no conflict of interest.

Contributors: Alonso JV proposed the study, analyzed the data and wrote the first draft. All authors contributed to the design and interpretation of the study and to further drafts.

REFERENCES


Received March 28, 2013
Accepted after revision September 25, 2013